Management of Infertility in Cattle

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DR N.K. SUDEEP KUMAR; DR M. SELVARAJU; DR K. KRISHNAKUMAR; DR. S. SATHESH KUMAR; DR. T. SARATH; AND DR. S. RAJA

COMMONWEALTH OF LEARNING)COL)
BURNABY

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Introduction

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Course Description

Animal Husbandry is making a significant contribution to the national economy and socio-economic development of most developing and under developed countries. Dairying contributes significantly to the sustainable development goals proposed by the United Nations. The average milk production per crossbred and indigenous cow is 7.6 litres/day and 3.8 litres/day respectively in India. Currently the average coverage of Artificial Insemination breedable population is 28% in India. Dairy cows should calve once every year to maximize the production to have economic efficiency. Infertility among dairy animals continues to be major bottleneck in achieving this target.

Similar situation prevails in other Asian and South African countries. Practicing veterinarian can directly make a change in the livestock sectors by improving fertility in bovines. This series of lectures covers six topics of infertility problems faced by the field vets. This agMOOCs has focused all the aspects of managing infertility and its current approaches to the specific topics. Hence the vets across

Asia and Africa can get benefited and make the difference in their performance in achieving greater fertility in animals to improve the farmers' economy.

The course will cover economic importance, etiology, diagnosis and treatment of repeat breeding syndrome in cows. Anestrum – its types, diagnosis, treatment and prevention. Endometritis – types, incidence, etiology, pathogenesis, diagnosis, therapeutic approach and prevention. Cystic ovarian degeneration – predisposing factors, classification, pathogenesis, clinical signs, diagnosis, treatment and prevention. Congenital and acquired defects – defects of ovary, oviduct, uterus, cervix, vagina and external genitalia, segmental aplasia of mullerian duct and hereditary forms of infertility. Estrus synchronization – principles, benefits, criteria for controlled breeding, synchronization using prostaglandin and progesterone and synchronization of ovulation.

Course Content

- Repeat breeding syndrome in cows
- Anestrum
- Endometritis
- Cystic ovarian degeneration
- Congenital and acquired defects of reproductive tract
- Estrus synchronization

Course Audience

- Practicing Veterinarians
- Pre final, final and internee veterinary students
- Veterinarians working in State Animal Husbandry Departments, NGOs and Development Departments

- Veterinarians working in State Veterinary Universities / State Agricultural Universities / Veterinary Colleges

Outcomes of this Course

- Diagnosis and treatment of repeat breeding syndrome in cows using recent therapeutic protocols
- Types of anestrus and their remedial measures
- The significance of subclinical endometritis and tackling clinical and subclinical endometritis in bovines
- Treatable and non-treatable cases of cystic ovarian diseases in cows and buffaloes
- Sterility due to congenital and acquired lesions and recommendations for culling
- Application of oestrus induction protocols in the treatment of infertility cases in bovines

PART I

WEEK 1: REPEAT BREEDING SYNDROME IN COWS

1 Introduction, Synonyms, Definition, Prevalence, Economic Importance, Etiology and Classification

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Transcript

Dear and respected veterinarians.

We are going to see about the Repeat Breeding syndrome in cows. It is an important form of infertility, leading to heavy economic loss to the Farmers. How it causes economic loss? Because the number of inseminations will be more, the calving to conception interval will be more, so animals will be repeatedly coming to heat and it is not conceiving, so that may lead to heavy economic loss to the farming community. So, if you would know about these, Repeat Breeding syndrome very well, you can treat the case very easily in the field condition so that farmers' economy can be improved. With that idea, we are going to see about a repeat breeding syndrome in cows.

The Synonyms of the repeat breeding cows are Cyclical non-breeders, Repeaters, and Repeat breeders.

There is a classical Definition for repeat breeding syndrome. Repeat breeder is the one that has been bred two or more times with fertile semen but failed to conceive, which they earned it as

normal or nearly normal estrous cycles and estrous periods along with the following Characteristics:

It should have a normal estrous cycle length of 21 days.

It should be free from palpable abnormalities of the genital tract. The genital tract should be free from any palpable abnormalities.

It should have normal vaginal discharge during the estrous period.

The animal must be in positive energy balance. It should be at least calved once, and

It should be less than 10 years old

One important point to be noted here is, it should be at least calved once. Here we are including heifer also nowadays, but as per the classical definition, the repeat breeder must be at least once calved.

When we talk about the Prevalence of the conditions in cows and buffaloes, crossbred animals have affected the percentage of (17.57%), Buffaloes have the problem of 12.74%, Indigenous cows are less commonly affected, it is 8.64%, 30.4% of the cattle and buffaloes were culled for mainly infertility problem, among the various infertility problem, repeat breeder alone costs about 18 to 20% of the infertility problem. That is why this condition has more importance and relevance to be discussed here.

The Importance in Economy, repeat breeder causes very heavy economic loss as I told you earlier, the cost involved is a Breeding cost, Treatment cost, Feeding cost, and Labor cost. Suppose if an animal is not conceiving a single day, the maintenance cost is about to ₹200, that is the loss is ₹200 per day, nowadays. If one cycle is lost, 21* 200, so ₹4200 is a loss to the farmer. Repeat better even after two or three cycles, it is not getting conceived, that is why the basic economic losses are caused by this delay in conception due to repeat breeding syndrome. Now we are switching over to the Etiology of Repeat breeders:

There are two types of classifications, one is by Casida and co-workers and another one is by Roberts and Zemjanis. So first the Casida co-workers classified the repeat breeding syndrome as

fertilization failure and the early embryonic death. So based on this classification only we are going to discuss Etiology in the future.

Another classification is the Zemjanis and Roberts classification, which includes there are five types of etiological factors, one is Congenital or genetic anatomical defects of the reproductive tract in cows. The second is Congenital or genetic acquired defects of sperm, ovum, and zygote. Third is Infectious or traumatic inflammatory causes, fourth is Endocrine dysfunction, fifth is Managemental and nutritional deficiencies causing repeat breeding syndrome. So, these are the etiological factors classifications.

Now we will go one by one, Fertilization failure. Actually, Fertilization failure constitutes 15% of reproductive wastage.

Ovulatory defects: Ovulation occurs 12 to 14 hours after the end of estrum in cattle. This is important for the ovum to get fertilized with the presence of spermatozoa already deposited in the genital tract, you all know well. Suppose any delay in ovulation, you've already deposited the semen, so all the spermatozoa will die by the time ovum is coming. So delayed ovulation is one of the reasons for fertilization failure. Another reason is Anovulation, sometimes animals may come to heat or estrous and the follicular development will be there, animals will come to heat, but there may not be Ovulation. So, all the spermatozoa which are waiting in the fallopian tube may not involve in fertilization, so that may end up in fertilization failure. Another reason for the ovulatory defect is cystic ovarian degeneration, follicles may develop, they will persist in the ovary, ovulation may not occur, which may lead to ovulatory defect and fertilization failure. So look at this (showing picture), this is the fully matured follicle, it should rupture and produce ovum if any delay or failure to rupture or retention of the follicle in the form of a cystic ovarian degeneration leads to repeat breeding syndrome.

Second is the Ageing of the Ovum; aged ovum: Suppose ovulation takes place, you are inseminating too late when we will inseminate 12 hours after the onset of estrum, we are going to inseminate the animal, that is the classical point,12 hours after the onset of estrous, i.e, from mid estrum to late estrum we have to inseminate the

cow. Suppose you are inseminating after the end of the estrum but after the end of the estrum the sperm has to undergo capacitation reactions, it is not getting ready for fertilization so due to this phenomenon, Ovum is aged. so there may not be any fertilization occurring in the aged ovum.

Defective Ovum: sometimes what will happen is there will be a micro-ovum, macro-ovum, or failure of extrusion of second polar body, so all these causes defect in the Ovum, that also leads to fertilization failure.

Next to failure of male and female gamete union: that may end up in fertilization failure. Also, sometimes unilateral hydrosalpinx, bilateral hydrosalpinx, severe endometritis, all these things will cause the failure of union of male and female gametes.

I told already the Ovaro-bursal adhesions and Tubal obstructions are the major problems causing the fertilization failure.

Stress: stress also, animals are exposed to various types of stress, For example, excessive heat, excessive cold, fear, shyness, all these causes stress, threatening by the owner, everything will cause stress and may lead to improper fertilization or improper movements of gametes in the genital tract and they end up in fertilization failure.

Next, sometimes Antisperm, antibodies are formed in the female genital tract. Because you know very well that, sperm is an antigen, so to this antigen, the antibody will be produced in the genital tract, especially in the cervix. But in the natural cervix, the semen is deposited in the vagina, when the sperm reaches from the vagina to the cervix, these Antisperm antibodies present in the cervix or cervical mucus, will mask the moment of the protozoa, that will end up in fertilization failure. So Antisperm antibody production is there. To bypass this in artificial insemination, we are depositing the semen in the body of the uterus, you know. So, in one way we are passing this barrier of cervical mucus and depositing the semen.

We are continuing with Fertilization failure. As I already told Segmental aplasia.

Segmental aplasia: look at this picture, this is the uterus unicorns, one side of the uterus is not developing,

Hydrosalpinx: this is hydrosalpinx, you look at this fallopian tube is filled with fluid, and

Ovaro-bursal adhesions: here Ovaro-bursal adhesions, you cannot separate the ovary from the rest of the bursal. So, all these leads to fertilization failure because they prevent the union of male and female gametes,

Anti-sperm antibodies: here also you look at this anti-sperm antibody production in the cervical mucus that will prevent the movement of the spermatozoa, masks the moment of the spermatozoa, it will cause the head-to-head attachment or tail to tail attachment of the spermatozoa, so that will end up in fertilization failure.

Dear Vets, we have seen so far various Etiological factors involved in causing Fertilization failure that leads to Repeat Breeding syndrome in cows and buffaloes. So, we will stop here. We will continue with the early embryonic mortality in the next class.

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2 Early Embryonic Mortality

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Transcript

Dear Veterinarians, so far, we have seen various factors involved in causing Fertilization failure in cows and buffaloes, that lead to Repeat breeding syndrome. We have seen in yesterday's class all these factors. In today's class, we are continuing with the Early Embryonic Death in cows and buffaloes, it is the greatest problem causing repeat breeding syndrome in cattle. Fertilization is not the matter; the Embryonic development is the matter for their successful fertility that we will continue today. So, it also constitutes about 25% of the reproductive wastage, I told Fertilizing failure constitutes about 15% but now the recent literature if you see the fertilization is not the matter, almost in 95% of the animals' fertilization takes place, further development of the embryo is the crucial factor in determining the fertility of the cows and buffaloes. So, in our infertility problems, dealing with Early Embryonic Death is more important than Fertilization failure. It constitutes about 25% of reproductive wastage.

What are all the factors causing Early Embryonic Death:

One is Chromosomal Aberrations, this Chromosomal aberration is seemed to be very common in Guernsey breeds, any chromosomal defect in the Ovum or in the Sperm that may cause the death of

the embryo subsequent to the fertilization. In the case of cows 1/29 Robertsonian translocations cause Early Embryonic Death, likewise the sperm defect also, that is any defect in the chromosomes of sperm also causes embryonic mortality. Ovum also causes embryonic mortality.

Aged Ovum: What will happen? Ovum will be released, delayed insemination, after the release of Ovum, may cause penetration of the ovum by more number of spermatozoa, when the ovum becomes aged, the membrane integrity of the zona pellucida is lost, so the membrane integrity will be lost, it will allow more number of sperm to enter into the ovum, therefore Early Embryonic Death occurs in the fertilized Ovum due to more number of chromosomes. Actually, half of the chromosomes are from the male gamete and half of the chromosome from the female gamete forms the Zygote, suppose more sperms enter into the ovum that may lead to the Early Embryonic Death.

Polygyny: During the Fertilization process, Ovum matures followed by Sperm matures then ovum and sperm encounter, these are the steps in the fertilization. But ovum maturation is completed at the time after the time of sperm entry only, ovum released as secondary oocyte i.e., the ovum is released at the time of ovulation as secondary oocyte in cows and buffaloes. Then it continues to mature, when the sperm penetrates, after the penetration second polar body will be extruded, which completes the ovum maturation. But when the second polar body is not extruded at the time of fertilization, then that may end up in a Polygyny. Polygyny means failure of extrusion of second polar body. So, the second polar body will be extruded in a normal course of fertilization, if it is not extruded, that may end up in Polygyny, which in turn will lead to Early Embryonic Death. Why I am telling you about all these etiological factors? If you know this etiological factor only as a field veterinarian, quite interestingly you can treat the animals by knowing the cause, by diagnosing the cause of the repeat breeder.

The next important cause is Specific infections: But if you take Specific infections after the introduction of Artificial Insemination,

these etiological factors have minor significance because the bull is screened for Trichomoniasis, Vibriosis, Brucellosis, IBR, IPV, Tuberculosis, and Johne's disease. So, the semen is prepared or semen is obtained from the genetically proven and bull which is free from infection. So always it is better to use Artificial Insemination to avoid Specific Infections. What are the specific infections in the cattle: It includes Trichomoniasis, Vibriosis, Brucellosis, Tuberculosis, Johne's disease, all these are Specific infections. These Trichomoniasis, Vibriosis, Campylobacterosis, or Brucellosis may cause early Embryonic death. Many of us think these are the agents responsible for abortion, they are not only responsible for abortion but also responsible for embryonic death.

Next is Non-specific Uterine infections: if you take any animal, the vagina will have all our types of organisms' Saprophytic organisms, including Streptococcus, Staphylococcus, E-Coli, all these infections will be there in the vagina. When the animals' resistance goes down, these organisms take the upper hand, and it causes genital infections. Clearly note down this, because always the female genital system, especially the vagina, is contaminated because it has a connection with the environment, and because of these organisms present in the vagina, they may flare up our hit multiplied when the animals' resistance goes down, so this nonspecific uterine infection also many times causes Early Embryonic Death.

There are 7 important micro-minerals that are necessary for successful reproduction, including Selenium, Copper, Zinc, Iron, Cobalt, Magnesium, Manganese is the seven minerals that are necessary for successful reproduction, and they act as a coenzyme for the production of hormones. That is why very, very important mineral mixer supplementation for infertile animals. Next is Environmental factors, so Environment also plays a vital role in causing the repeat breeding syndrome, high temperature, high humidity, all these environmental factors may cause heavy rain, then flood, natural calamities, all these may cause an impact on the fertility of the animal.

The next important thing, which can be avoided by any

Veterinarian, Semen contaminated with infectious agents, we should get semen from approved semen stations or government organizations. So, the Semen, you should ensure it is free from any infectious agents, otherwise, the spread of infections will be more.

Then at the time of AI, we should concentrate on clear hygienic AI, otherwise, any contamination during AI may lead to repeat breeding syndrome. Not only that, it will further aggravate the condition by causing uterine infection.

Next is Oxidative stress. Due to the metabolic activity, there will be free oxygen radicals circulating in the blood. These free oxygen radicals are harmful to the embryo, so that may cause Early Embryonic Death.

Hot sunlight: If the animal, after the insemination, is exposed to heavy or high environmental temperature, under the hot sun, that may cause embryonic mortality. That is why you used to advise after one or two days of insemination, animals must be kept in a shed, so that the environmental temperature may not increase the body temperature. If the body temperature of the animal is increased due to the environmental temperature, it may cause embryonic death. Next is Endocrine Dysfunction: Endocrine dysfunction, any hormonal deficiency, especially progesterone deficiency or LH deficiency, any form of deficiency of hormones may cause a repeat breeding syndrome.

Then another very important is Sub-clinical endometritis: This animal will have clear discharge, but it doesn't mean the uterus is free from infection, many books say that uterine infection without any clear evidence may cause prevention of implantation, that is why it is leading to Embryonic mortality, and this is a thing you have to know Sub-clinical endometritis is another major important cause for repeat breeding syndrome under field condition.

Dear field veterinarians. So far, we have discussed Fertilization Failure and Early Embryonic Death in cows and buffaloes, which lead to a Repeat breeding syndrome in these animals. We will stop here and will continue with the diagnosis of repeat breeding syndrome in the subsequent class.

Thank you!

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3 Diagnosis

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Transcript

Dear Veterinarians, now we are going to start with the diagnosis of repeat breeding syndrome in cows.

Until yesterday's class, we have seen various Definitions and the various Etiological factors causing repeat breeding syndrome, we will continue with that class. Now we are going to see the diagnosis of repeat breeding syndrome.

Diagnosis of repeat breeding syndrome based on the History, actually the owner will be bringing the animal to the fertility ward or gynecology ward for two reasons, one is my animal calved, it does not come to heat so far, till date it did not express any estrous signs, this is one history, the second history is my animal is repeatedly coming to heat, but not yet conceived, so our condition is the second point, i.e, my animal is repeatedly coming to heat, but not yet conceived, this is the clear History from the owner and owner also will tell from the history, it is coming to heat every 18 to 21 days and every time inseminated by the veterinarian and you have to ask the owner how long the animal is in estrous if it is prolonged estrous it indicates delayed ovulation. Prolonged estrous for 3-4 days, it may be anovulation, so we will discuss later how to Diagnose.

The second point is Clinical signs: Clinical signs, I told whenever

you are examining the animal, it will have clear discharge and the animal is free from palpable abnormalities of the genital tract and everything will be normal while doing the rectal examination, but the animal failed to conceive.

The third is I told very important parameter is Endometritis, it is detected by very simple test-by White Side test. How to perform White Side tests? Very easy, you should have potassium hydroxide or sodium hydroxide, you take this cervical mucus. How to take cervical mucus? In AI gun, how the sheath introduced the AI sheath, then introduced into the cervix, suck the cervical mucus or little forward you move suck the uterine fluid, take out the sheath, then collect the fluid in the test tube. An equal volume of 5% sodium hydroxide or potassium hydroxide will be added. and you heat it when the color changes into mild yellow there is mild endometritis. If the fluid is turbid, there is no endometritis. If the color changes to yellow like this (picture shown), it indicates sub-clinical endometritis.

Next is I told you know the fallopian tube obstruction or Ovaro-bursal adhesions, hydrosalpinx. So, such types of conditions may cause repeat breeding syndrome. To Diagnose we have to perform the PSP dye test. PSP dye test phenolsulfonphthalein dye is used, 0.1% solution is used, or Phenol red which is an indicator used in all chemistry labs of (+2) students. So, you can get the Phenol red very easily, then prepare a 0.1% solution. This test is to be conducted only at the time of diestrum. when it should be? Suppose today animal is coming after 7 or 8 days, ask the owner to bring the animal, prepare the phenolsulfonphthalein dye or Phenol red, 0.1%, filtrate then use the Foley catheter, keep it in the one side of the for one side of the uterine horn, inflate the balloon, then inject the dye. The dye will be passing from the uterine horn to the fallopian tube then it will be reaching the peritoneum, then it will be excreted through the urine. Before conducting the test, collect the urine, keep it as a control, then after half an hour of dyeing infusion, again collect the urine by using a urinary catheter, then you see the color change, add any alkali with this example Trisodium orthophosphate, you can

add it, the color of the urine will be changing to a pink color or purple color, so the fallopian tube does not have any obstruction. If there is no change in the color, the color of the urine after the dye infusion is similar to that of the control, it indicates that the side of the fallopian tube is obstructed. The next side of the fallopian tube is to be tested in the next diestrum only, not in the next day. So how long the dye takes to travel through the fallopian tube indicates, the fallopian tube obstruction or patency, this is called the PSP dye test.

Next Endometrial cytology: This is another important thing, don't fear about all these things, very, very easy. See, you can infuse saline into the uterus, then collect the saline after massaging the uterus, centrifuge the collected fluid and take the sediment and smear it on the glass slide and stain it with Hematoxylin-eosin, Giemsa stain, or Leishman stain. Then see for cells, if there are more number of neutrophils, it indicates leukocytic infiltration and endometritis very easily can be diagnosed.

Cervical mucus test: if you take that, you know as a veterinarian, it is the fern pattern, Typical fern pattern will be formed at the time of estrous, you collect the cervical mucus, smear it over the slide, and dry it under the sunlight then you see the fern pattern. The Atypical fern pattern will be seen in repeat breeder, so this is a classical thing, the fern pattern will be Atypical.

Next is a cervical mucus penetration test: Take the cervical mucus of repeat breeder animals, keep the quality semen, put the coverslip. If you examine under the microscope after a few minutes, 5 minutes, 10 minutes, and 15 minutes intervals, there will be more number of sperms that will be penetrating through the cervical mucus, which indicates the cervical mucus is normal, so the repeat breeder cow is not having any infection.

Suppose you can diagnose this sperm viability also if the cervical mucus is from a fertile cow, but here we are testing the female animal, so the female animal that too far repeat breeding. cervical mucus from the repeat breeding animal will not allow more sperms to penetrate, this is how you can diagnose.

Next, Ultrasonography: If you take the ultrasonography,

ultrasound is useful to detect thickening of the endometrial layers, thickening of the uterus, then the tubal obstructions, hydrosalpinx, mucosal sphinx, all these things can be detected by ultrasonography. So, ultrasonography not only detects defective things, sometimes early pregnancy, an early embryo will be there, many veterans failed to diagnose, and they will inseminate that may cause embryonic mortality. So always it is better to use ultrasonography, recent equipment that can be used.

Next is an Endometrial Biopsy: Suppose if you want to confirm the presence of the inflammation in the uterus. All Percutaneous biopsy catheter is there, you could take the endometrial tissue then there only perimetrium gland fibrosis will be there, leukocytic infiltration can be diagnosed. This is the confirmative test to diagnose sub-clinical or clinical endometritis, which in turn causes the repeat breeding syndrome.

Dear Vets! we have seen so far, all the information about the diagnosis of repeat breeding syndrome in cows. We will stop here and will continue with the Treatment aspects of repeat breeding syndrome tomorrow.

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4 Treatment of Fertilization Failure

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Transcript

Dear veterinarians,

As I told you already repeat breeding is a disorder, causes heavy economic loss to the farming community. The important thing expected from veterinarians is the proper treatment.

So in today's class, we will start with the treatment of repeat breeding syndrome. We will continue with the sexual rest. The first and foremost thing is, to suppose, I told about the endometriosis, subclinical endometriosis, if you give one or two cycles, you need not go on breeding the animal continuously. Today it has come to Estrous, you are inseminating after 21 days, again after 21 days, don't do that. Suppose an animal is repeatedly coming to Estrous, give one or two cycles sexual rest, so what for we are giving a sexual rest? The sexual rest will give will cause leukocytic infiltration, it will promote the Uterine Defense Mechanism. Uterine Defense Mechanism UDM, so that infections will be eliminated from the Uterus. In this way, you are allowing nature to cure the condition. So, always say, whenever you are treating for endometriosis, whenever you are treating for repeat breeding syndrome, always give sexual rest one or two cycles. It may not affect the economy or anything, but it is an important thing to be followed in infertility

problems. So sexual rest will promote the Uterine Defense Mechanism and Uterine healthiness and healing of the endometrial layer. So, all these things will be achieved with sexual rest, give sexual rest for one or two cycles.

Next, we are starting, Double AI:

How to do Double AI? I told that there, delayed ovulation may cause repeat breeding syndrome if the ovum is released very late, by that time sperms will die.

So, enough number of spermatozoa will not be available in the isthmus, so you have to make the sperm available when the ovum comes to the fallopian tube. Where the fertilization takes place? It takes place in the ampullar-isthmic junction of the fallopian tube. so, the isthmus should have enough number of spermatozoa when ovulation occurs. When ovulation occurs, at least thousands of sperm should be there in the isthmus. When the sperm moves through the fallopian tube and reaches the site of fertilization, at least hundreds of spermatozoa should be there in the isthmus to fertilize. When the sperm started penetrating, at least 10 sperms must be there. This is how fertilization and embryonic development occur. So, always you should ensure that the availability of the spermatozoa in the genital tract, that is why you are doing today insemination and next day insemination, it is called a Double AI. Double AI means at a single time not doing two straws, instead, it is at an interval of 24 hours, we are doing AI to tackle delayed ovulation. Next, another thing is, to further ensure the ovulation you are doing double AI at an interval of 24 hours but remember you should start doing AI at the onset of estrum. Estrous duration is 18 to 24 hours in cows, the onset of estrum one AI and 24 hours later one AI you do. Second, at the time of AI, you can give hCG, Human Chorionic Gonadotrophin, 1500 IU Intramuscularly available in the name of Chorulon, or you give GnRH, it is available in the name of Gynarich, Ovulanta, all these preparations are available.

So, 2.5 to 5 ml GnRH, that is 10 to 20 microgram GnRH can be given Intramuscularly at the time of first AI, next day you repeat the AI because the GnRH will act it will produce CL surge, it will cause

ovulation. So, at the time of first AI, during double insemination you should give hCG, GnRH intramuscularly. Many reports say and our experience also says at the time of AI, you can give GnRH or hCG. When you are attending at the farm level, this is a fertility drug, Gynarich is a fertility drug. You can inseminate and give one shot of 2.5 ml of GnRH that is 10 micrograms intramuscularly. My advice is you avoid Intra-uterine antibiotic therapy, kindly give antibiotics through the parental route. Parental means intramuscularly you give, what all the antibodies, other than sulfonamides, you can use any antibiotics for example very important is the Streptopenicillin (streptomycin + penicillin), Ampicillin, Amoxicillin, Ceftriaxone, Ceftiofur, Cefuroxime + Sulbactam, so all these antibiotics. But don't use Sulfonamides. Other antibiotics can be given intramuscularly as a post insemination antibiotic therapy. Nowadays we are avoiding Intra-uterine antibiotic therapy because that will affect the Uterine Defense Mechanism. That is why we are going for parental antibiotic therapy, it is preferred, and you need not wait for six to eight hours, after insemination you can give parentally, you can send the animal to the owner's house and the animal may not face any problems, only thing is the proper dose should be given intramuscularly.

So next is a Progesterone, the development of the embryo is crucial, as per the recent report of Arthur is concerned, it is crucial between 6 days to 7 days and 16 to 18 days. The crucial period of embryonic development is 6 to 8 days and also from 6 to 18 days. 6 to 7 or 8 days, why I am telling you is because the hatching of a zona takes place. From the zona pellucida, the embryo has to come out in the uterus, so it has to develop. So, if the hatching fails or improper hatching, any defect due to progesterone deficiency, the embryo may not develop. If the signal is not there from the embryo for 16 to 18 days, then embryonic mortality will occur, that is why to have proper zona hatching in the uterus, we are administering 500 milligrams of progesterone on the 4th day, preferably on the 5th day after AI. So that 6th day or 7th day or 8th day, when the zona hatching takes place, it will be properly taking place.

Then next type of treatment, there are multi, various treatments

available for you to follow in the field. 6th day or 7th day after AI, you can give hCG or GnRH. If you give GnRH, because you know follicular development is a continuous process, 6th day there will be a presence of a dominant follicle. Already there will be a corpus luteum, you are giving GnRH on the 6th day or 7th day, so the follicle presents in the ovary due to the first follicular wave will be Luteinized or causing ovulation, so it will secrete or produce an accessory corpus luteum and more number of progesterone will be secreted. So, on the 6th day or 7th day, you can give one shot of hCG or GnRH. Likewise on the 14th day also you can give hCG or GnRH to Luteinize the third wave follicle. So, these are the recent advancement in the treatment, that is post-AI GnRH, hCG therapy on day 7 or day 6 or day 14. This will cause the formation of accessory corpus luteum and also it will strengthen the already formed corpus luteum. So, this is very very important to be followed, you can follow.

Then I told- No Oxidative stress. Oxidative stress is an important factor for embryonic mortality, so to avoid embryonic mortality due to oxidative stress, you can use vitamin AD3E injection on the 7th day and 14th day after AI, so this will help to avoid oxidative stress. Next is Injection "E-Care Se", another form of antioxidant, this injection E-Care Se also can be used to avoid oxidative stress. So, this also can be given day 7th or 14th after AI. Next is one of the important things is Flunixine meglumine, this is because on day 14th or 15th if the embryonic signal is not there, there will be a prostaglandin release from the uterine endometrium to cause a rise of the CL. To avoid the rise of the CL, if you give Flunixine meglumine, the corpus luteum can be maintained, so that embryo can develop further. So, this is a very very important recent therapy tackling early embryonic mortality in repeat breeding cows. Then during after AI, directly you can insert progesterone into the vagina and they can be kept for 11 to 13 days in the vagina, it will supplement this CIDR or TRIU-B will supplement progesterone, which will favor the growth of embryonic embryo, which is in the uterus, so it will avoid early embryonic death. So, post CIDR therapy

it will be kept in the vagina for 11 to 13 days, after 11 to 13 days if you remove, if the animal is not coming to heat it indicates embryo is there. If it comes to heat, then the animal has not conceived, that advantage is also there for this CIDR therapy.

Next is in case of endometritis, suppose you are diagnosing with white side test I already explained, so you can treat the case with Lugol's Iodine or Povidone Iodine. Again many of the veterinarians are using Povidone-Iodine in the field but it should not be diluted, keep in mind that it should not be diluted. Povidone Iodine is available iodine is 0.5 percent only, so Povidone Iodine should be used as such. But when you are using Lugol's Iodine, how to prepare Lugol's Iodine? You should have the stock solution, you should remember 2-3-4, 2 grams Iodine, 3 grams potassium added 40 ml distilled water two to two two three four this formula 2 grams iodine, 3 grams potassium added and 40 ml distilled water, this makes the stock solution. From the stock solution you can prepare1% to 2 % solution and it can be given through intrauterine route about 30 to 40 ml or a maximum of 50 ml you can give depending upon the size of the uterus and the parity of the animal. So, Lugol's Iodine in 3 days you can administer to treat endometritis. In case of supposing, parental endometriosis is there or uterine infection is confirmed, you can combine with Intra-uterine therapy with the uterine antiseptics along with the parental antibiotics, hence uterine antiseptics Lugol's Iodine or Povidone-Iodine can be used. When you use antibiotics, you can use parental antibiotics along with Intra-uterine therapy with uterine antiseptics.

Dear vets and practitioners of the veterinary profession, we have seen various treatments so far up to the uterine infection-causing repeat breeding syndrome in cows.

We will continue with the estrous induction program and other treatment aspects of repeating syndrome in the next class.

Thank you.

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PDF: Treatment of Fertilization Failure

5 Treatment for Early Embryonic Mortality

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Transcript

Dear veterinarians,

We have started the Treatment aspects of repeat breeding syndrome in cows and buffaloes, today we will continue with yesterday's class.

We are starting with the Improper estrous detection and Untimely insemination: It is a major factor causing repeat breeding syndrome, we will see how to tackle it. Embryonic mortality is a natural way of eliminating unwanted genotypes at a low biological cause. So sometimes we need not favour embryonic development also in some cases, because the congenital or heredity anomaly may develop. The important thing in the field as far as repeat breeding is concerned is the improper estrous detection and untimely insemination is the major cause of repeat breeding syndrome, it comes under managemental cause, so improper estrous detection and untimely insemination is the major cause, so this cause can be avoided by using the estrous induction and synchronization program.

Two principles are there: one is Shortening the luteal phase another one is extending the luteal phase. Shortening the luteal phase with PGF2 Alpha, Extending the luteal phase with

Progesterone treatment. On the 10th day, how to induce in, suppose one or two animals are brought, so 10th day you ask the owner to bring the animal, 10th day means 10 days after the estrous. Palpate the corpus luteum in the ovary, then give PGF2 Alpha intramuscularly, then after 72 hours, you do AI at 96 hours. At the time of doing AI, at 72 hours, you can give GnRH 2.5 ml or corion or hCG 1500IU intramuscularly.

The very important thing I told already, the importance of minerals in the etiology, there are important 7 minerals: Copper, Zinc, Iron, Cobalt, Selenium, Manganese, Magnesium, all these minerals all in bolus, or in the form of powder, so that can be supplemented when you start the therapy. Before starting any therapy for repeat breeders, you give mineral mixture for at least 10 to 15 days, but continuously you have to supplement minerals orally so that your treatment response will be improved. Already I told you, they act as coenzymes for the production of hormones and if it is a heifer showing repeatedly repeat breeding syndrome problem, you have to test the dung material for the presence of parasitic ova and if there is a parasitic ova presence, you can do deworming according to the type of egg found in the dung. Then after doing AI irrespective of whether it is a normal animal or repeat breeding animal, do Clitoral massage for 4-5 seconds, Clitoral massage can be done for 4-5 seconds after AI, which will improve the conception rate because it is set to produce the effect through the advancement of LH surge by 4 hours. What is LH surge? The LH surge is important for the final maturation of the follicle in ovulation, I told you ovulation defects cause embryonic failure that is fertilization failure, so the clitoral massage, simple clitoral massage will improve the conception rate in repeat breeding cows.

Now we are moving to how to prevent repeat breeding syndrome in cattle. Ensure the proper timing of AI, I told you the best time of breeding is from mid to late estrum or 12 hours after the onset of estrum. So any early insemination or very delayed insemination may cause repeat breeding syndrome. Ensure that the insemination techniques are proper, that is you should ensure the quality of the

semen, proper deposition of the semen in the proper site i.e, the body of the uterus. You should not deposit the semen in the horn with the enthusiasm that we will advance the sperm migration, it is not correct. Suppose ovulation takes place in the right ovary if you are inseminating in the left horn, then there will be a fertilization failure, so always inseminate the cow within the body of the uterus, and you should not damage the endometrium at the time of AI, that may cause endometritis and the production of prostaglandin PGF2 Alpha, then the conception failure.

Then animals should not be inseminated, when it is early pregnant, many many of the veterinarians are the other fellows, neglecting the early pregnancy and doing AI because early pregnant uterus will develop tonicity that will confuse the estrous. So early pregnancy should be detected properly to avoid repeat breeding syndrome. Identify and treat the cow with abnormal discharge before doing artificial insemination and all uterine infections should be settled, then only we should do AI.

Don't start breeding too soon after calving, some animals may come to heat within 25 days, 30 days give one cycle rest, plan for next cycle, that is you should give a gap of 45 to 60 days after the calving in many many crossbred animals. So that is also very important for proper involution of the uterus.

Next, minimize the stress at AI. In our experience, we found doorstep AI has increased conception rate, then when the animals are brought to the veterinary hospitals. So don't make the animal walk for a long distance and do AI instead, you can do doorstep AI to improve the conception rate.

Dear friends, in the series of lectures we have discussed the Definition, Etiological factors, various Diagnostic procedures, Clinical signs, and Managemental practices to be followed for treating or alleviating repeat breeding syndrome in cows and buffaloes. I think all the classes will be useful to you and if you have any doubts in the future, don't hesitate to contact me at this address and you can contact me through email or mobile.

Thank you very much. I Thank one and all.

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PART II

WEEK 2: ANESTRUM

1 Introduction, Definition, Prevalence, Economic Importance and Classification

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Transcript

Hi! Friends,

I am glad to introduce myself as Dr. S.Raja, Assistant Professor in the Department of Veterinary gynecology and obstetrics. Veterinary College and Research Institute, Namakkal, which is a constituent college under Tamil Nadu Veterinary and Animal Sciences University, Tamil Nadu, India.

I am sure you all are enjoying the online courses from MOOCs in collaboration with IIT Kanpur and Commonwealth of learning Canada. I am happy to say, I am the content creator for the course "Anestrum" with a special reference to Anestrum in dairy animals. Let's move on to the Objectives and this Anestrum course has 5 series of lectures and the course outline which includes the 1st lecture, which is on an Introduction to Anestrum. For any condition or any disease condition, understanding the introduction is very important followed by prevalence. Understanding the prevalence gives an idea about the pattern of the reproductive disorder, prevalence not only in the State among the entire Nation and

followed by economic importance. So why do we need to study Anestrum and what way it is contributing to the economy and what way farmers are getting affected or benefited? So, we will be discussing the economic importance followed by etiology and classification concludes lecture 1. so, lecture 2 will be focusing on the main classification, we will be discussing False Anestrum. So, what is meant by False Anestrum followed by the types? What are all the subtypes under False Anestrum and its etiology? What are all the etiological factors which are being contributed under False Anestrum? Followed by in lecture 3, we will be continuing the false Anestrum but the focus is more on the Diagnostic methods, what are all the Diagnostic methods under the False Anestrum can be identified because it needs to be differentiated from the true Anestrum, so diagnosis plays an important role in approaching therapeutic interventions for false Anestrum, followed by the therapeutic methods or any intervention protocols that concludes lecture 3. Followed by in lecture 4, under the main classification will be discussing on true Anestrum, so what is mean by true Anestrum, what are all the subtypes and what are all the etiological factors which are being contributed under true Anestrum and in the final lecture, we will be continuing the true Anestrum with focusing on various diagnostic methods as well as a various therapeutic intervention and finally, we will be concluding with the summary of entire Anestrum. So, with this objective's background, we will move on to the Introduction.

So, in general, from in farmer's point of view Infertility is being expressed in two ways, one[1]whether the animal is not showing any signs of estrum, so that is Anestrum which we will be discussing in the session in a very detailed way, that is one. And the second problem is the animal is cyclic but not getting conceived, so which means that the animal is repeatedly cyclical inactivity, repeatedly approaching for the breeding but even then, the animal is not getting conceived. So that is a topic under repeat breeding, which will be covered separately.

So, we will be discussing Anestrum, so Anestrus is characterized

by failure of estrus, failure in the sense, absence of estrus. So, the term Anestrus meaning without cyclicity. So means the word 'An' means absence. so, the absence of estrus that is- Anestrus. So, the absence of estrus is Anestrum, which means the absence of cyclicity, literally the word means. So, Anestrus is observed more commonly either in postpartum condition, so that is after parturition or sometimes in a pre-service condition also, because to understand this Anestrum can happen in normal physiological conditions also like before puberty or during pregnancy, but these conditions are often physiological in nature, which means that we need not worry about it. So only thing is we should know when the absence of cyclicity that is there has to be considered under Anestrum. So, this means that after puberty the animal has to show cyclical signs at the period of the interval of 18 to 21 days, if that is getting delayed then probably, we can put it under Anestrum. Similarly, after parturition, we need to wait for the period of around 2 months to 3 months, the average period is 60 days, if it is going beyond 90 days even though the animal is not showing any signs of estrum, then probably we can be put under the category of Anestrum. So, with this introduction, we will move on to what is Anestrus? what Anestrus is being defined? So, Anestrus is a functional disorder of the reproductive cycle which is characterized by either the absence of overt signs of estrum manifested either due to lack of expression of estrum or failure of its detection, so what it means here 3 points I would like to insist one is the absence of overt signs, overt signs mean it is not being exhibited, the other one is lack of expression of estrum or failure of detection. Here are the 3 points which include 1) is the animal itself not showing any signs of estrum, which means that ovaries are also not functional, functional in the sense, in terms of cyclicity, the ovary is not functional, the animal is not showing any signs of estrum. 2)The second thing is the animal is showing signs of estrum, but we are a failure in detecting, for example in large farm conditions, labors have been not properly educated and they may be skipping the estrus signs, that is an absolutely managemental error which is also coming under

Anestrum. 3)The other condition is, the ovary is functional, but the animal is not showing signs of estrum, so which means that physiologically the ovary is developing a follicle, the follicle is getting ovulated and there is a formation of corpus luteum, but the only thing is there are no behavioral signs of estrum, so which is also coming under Anestrum. So Anestrum means the absence of signs of estrum, which means that exhibition is not there, either it can be due to lack of expression or failure in detection, so this is what Anestrum means.

Next is Prevalence- for any reproductive disorder or any disease condition we should understand the prevalence, by understanding the prevalence, we could able to get an idea of how effectively or how far this disease is causing a loss or how effectively the population is being affected, which means the prevalence of Anestrum, for example in Tamil Nādu is around 16.6% in case of cattle almost throughout the entire Nation the percentage is more than 15%, so you could able to predict around 15% of the cattle population is being affected with Anestrum so rough estimate. So next thing is by understanding the prevalence we will be able to get an idea about the economic importance of why Anestrum is linked with economic importance or what way it is being linked. So, Anestrus has a great economic impact. so, what kind of an impact? For example, if the animal is having an Anestrum, which leads to economic losses through increased inter-calving interval, so which means that the time period or the gap between the two successive calving's is getting delayed. The optimal period is a calf a year, which means that within a period of 12 months or a maximum of 15 minutes I can say the animal has to go for the next subsequent calving if suppose if the animal is entering into the period of Anestrum, this inter-calving period is getting extended. So, what happens overall is affecting the annual calf production of the cow, for example, in a lifetime of the cow the overall calf production is getting reduced, this in turn affecting the production loss, How? Often the farmers are thinking, the increased or greater lactation length is a permanent profit, but strictly speaking, the greater or the longer

lactation period is only a temporary profit. so, what comes the permanent profit, so in a shorter duration of time the maximum number of pregnancies or maximum calving has to happen, then only it is a permanent profitable way. So increased or greater lactation length even though the animal is having an Anestrum, it is only in temporary profit, so the farmer should understand within a short period of time the maximum pregnancies and maximum calving has to happen which in turn increases the production. If suppose it doesn't happen, the calving inter[1]calving period gets increased, overall, it is affecting the production so that the farmer is forced to involve in treatment expenses. In turn the cost of replacing mature animals with the first calving heifer. Overall, the farmer is sacrificing some portion of his profit for the treatment. The other one is an estimated loss roughly from Anestrus is around rupees 200 per day in case of purebred and rupees 250 in case of the crossbred, so you can able to imagine if the animal or if the expression of heat signs is getting delayed by one day it is almost giving a loss of rupees 200. A per animal. rupees loss of 200 is affecting the farmer, the farmers are directly contributing to the state economy, the state economy is directly proportionate to the Nation's economy. So, each and every cow, for example, I can say each and every day if Anestrum is happening our estrus signs is getting delayed it is directly affecting the nature's economy. So overall I can say each and every day, if it is due to Anestrum it is affecting the GDP of our country. So, each and every cow is being contributed.

So, with this importance of the economy, we will move on to the Classification, In general, in the clinical aspect, the Anestrum is categorized into two types: one is Class I Anestrum and the other one is Class II Anestrum. So, Class I Anestrum is also referred to as False Anestrum so Class II is also referred to as True Anestrum.

What is the major difference between a Class I Anestrum and Class II Anestrum or between False and True Anestrum? In the case of False Anestrum, the animal is having a functional CL, in the case of True Anestrum, the animal is having No functional CL, so

what it means for example if False Anestrum, the ovary is having a corpus luteum, this secreting progesterone and because of that, the animal is not showing any signs of estrus but in the case of the True Anestrum the ovary is smooth, smooth doesn't mean that no structures, it can have varying degrees of structure which include multiple small follicles or sometimes you can find medium[1]sized follicles or sometimes you could able to find a dominant follicle also but there will not be any CL. The only thing is the ovary will be having a variety or kind of sizes of the follicle but there will not be any estrus signs. Often, we will be under the impression, in the case of true Anestrum there will not be any development of follicles, but strictly speaking, when you go perform ultrasound examination regularly, you could able to find in case of True Anestrum also you will be finding a different size of the follicles even more than 8 to 10 mm in diameter. Even though the animal will not show any clinical signs of estrus, so that is True Anestrum. In general, False Anestrum means with CL, True Anestrum means No functional CL. So, with this understanding of Classification, we will try to conclude today's Session. So we had seen the introduction about Anestrum and its importance followed by the Prevalence in Tamil Nadu as well as in overall India's nation and the importance in terms of economy and what way it is affecting the economy followed by a broad classification of Anestrum which includes True Anestrum and False Anestrum, with this, we will conclude today's session and we will continue in the next session.

Thank you!

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PDF: Introduction, Definition, Prevalence, Economic Importance, and Classification

2 False Anestrum and its Types

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Transcript

Hi! Friend's greetings,

In the last session, we were discussing on introduction to Anestrum, Prevalence followed by Economic importance and Classification of Anestrum. In session 2, we'll be continuing with the classification of False Anestrum, as we all know we have discussed in the last session also, the False Anestrum means there will be the presence of corpus luteum, the animal will not show any clinical signs of estrous. So what are the factors or what are the etiological factors which are being contributed to False Anestrum? The first one is Anestrus due to pregnancy. During pregnancy what happens, this ovary has a corpus luteum and the uterus is being occupied by the embryo or fetus, so what happened, this corpus luteum is kept on secreting progesterone, and progesterone is having negative feedback on the hypothalamus and suppress GnRH. So whenever the corpus luteum is there, it secretes progesterone, progesterone blocks, or suppresses the GnRH release from the hypothalamus by a negative feedback mechanism. So, during pregnancy, there will not be any clinical signs of estrum which is considered to be a normal physiological phenomenon. so, we need not worry about it. The second is Anestrum associated with corpus luteum of pregnancy,

which has been terminated early and not recognized, so what it means, the animal would have been conceived, you could be able to find this is a slaughterhouse specimen of early pregnancy, you would able to find there will not be any major clinical changes or topographical changes in the uterine structures. The animal would have been pregnant but because of some reasons embryonic mortality would have happened and which results in the expulsion of the embryo which may be noticed, or which may be unnoticed also, but it has been not detected by the corpus luteum which is present in the Ovary. So, what happens, this corpus luteum will be kept on persisting in the ovary and it secreting progesterone. Even though the animal has lost its pregnancy during early stages, this corpus luteum is secreting key progesterone, as I said earlier, this progesterone is having negative feedback at the hypothalamus to suppress the GnRH, so till progesterone is there, GnRH will not be secreted and because of that there will not be any clinical signs of estrum, so this is not physiological phenomena, which are in pathological which we need to be intervened.

The next thing is under False Anestrum, which is due to Anestrum due to persistent Corpus Luteum, here the term 'persistent' is somewhat new. So, when this persistent corpus luteum will be there? persistence means it keeps on persisting, apart from the normal duration. The conditions such as associated with uterine pathology, so this persistent CL is almost associated with some uterine pathological conditions. For example, Pyometra means pus in the uterus. The second one is a Mummified Fetus, so what happened the fetus would have been dead for some reason, and it has been keeping on preserved in the uterus. So, you could able to find this in a mummified fetus that has been evacuated out, but this mummified fetus will keep on persisting in the uterus. Another condition is Fetal Maceration because, for some reason, the fetus would have been undergone death followed by autolysis by bacterial contamination, followed by cervical closure and only the fetal remanence will be keep retained in the uterus, which is also a pathological condition. And other disease conditions such

as Mucometra, presence of Mucometra mucus or any kind of a liquid I can say mucometra or hydrometra, based on the change in viscosity, based on the uterine accumulation, prevents the release of prostaglandin and other condition is Pyometra, so I would like to insist that in all condition the uterus is being occupied with something either the fetus or dead fetus or fetal remanence or abnormal fluid in terms of the difference in viscosity or in terms of pus also. So uterus is occupied by something, which prevents the release of prostaglandin, so what happened unless the Prostaglandin F2 Alpha is being secreted, there will be no lysis of the corpus luteum, so what I would like to say is that in all conditions, the corpus luteum is persisting, so what happened this corpus luteum is kept on secreting progesterone and the animal is not showing any signs of estrum, these are all the different conditions where you can expect Anestrum because of persistent corpus luteum. So other than Anestrum due to persistent corpus luteum under the different mechanism. Usually what happens, in the ovary, the follicle keeps develops and this follicle secretes estrogen and estrous behavior will happen followed by ovulation and which results in the formation of the corpus luteum. This is a normal mechanism follicle growth proceeds undergo ovulation and there is the formation of corpus luteum, so after that in a normal mechanism what happens, this corpus luteum will undergo lysis and the animal will enter into the next period of Anestrum but in this condition after CL formation, the animal will enter into the period of Anestrum, due to failure of luteal regression. So what could be the cause for it, so usually what happened during the time of luteal regression, there will be one dominant follicle either it may be in the same ovary or the other ovary and this follicle secretes a little bit of estrogen and it potentiates the lysis mechanism but in this condition, the absence of estrogenic dominant follicle at the time of luteal regression favors the period of Anestrum. So till luteal formation, it is normal but luteal regression is not happening and because of that, the animal is entering into the period of Anestrum. Here I would like to insist that, persistent CL does not occur in the

normal cyclical animal often many Vets are getting confused with the cyclical CL with the persistent CL, so here I would like to make it very clear how to differentiate persistent CL and normal CL. For example, if you want to declare the animal is having a persistent CL, you are requested to perform a rectal examination at least two times with intervals of a minimum of 10 to 11 days so when you perform two times rectal examination and you are finding same ovaries having a corpus luteum, then probably you can declare the animal is having a persistent corpus luteum. If suppose during your second examination you are not finding the same ovary that has this corpus luteum, then you should not declare that as in persistent CL, so that is a cyclical CL. So easy way to differentiate a persistent and cyclical CL, is you need to go for two repeated examinations at the interval of 10 to 11 days. So False Anestrumand.

The next etiology is, next type is, Subestrous or Silent Estrous or Quite Ovulation. so what it means, Sub, Silent, and Quiet? The term Sub means a little less, little less than the normal so what it means so usually the estrous signs will be exhibited but, in this condition, estrous signs are not getting exhibited properly, another one is Silent as the name indicates silently ovulation happens but there are no behavioral signs of estrum, that is Silent ovulation. Another one is Quite ovulation, quite ovulation is also almost similar meaning. So, the synonyms are Sub estrum, Silent estrum, or Quite Ovulation. So, what happens in this condition, is being characterized by the failure of overt symptoms of estrum, It means that there are no clinical signs of estrum, usually what happens, in this case, is the follicle development and ovulation occur without manifestation of overt signs of estrum. It is a beautiful condition physiologically ovulation takes place, follicular development takes place, lysis happens again next follicular development ovulation happens, but there are no behavioral signs of estrum under this condition. So, it is common during the post-pubertal period in heifers and early in high-yielding dairy cows, it happens. The reason could be the progesterone, which has been secreted from the regressing field of the previous cycle, potentiates the action of estrogen and seems to favors the

manifestation of the estrous cycle in the next one. So what happens, the progesterone which is being secreted from the regressing CL of the previous cycle, potentiates the action of estrogen and probably I can say it is dominating and the animal is not showing any signs of estrum. so these all coming under silent. So one beautiful thing is after parturition, usually, the animal will show estrous signs, so whatever the estrous signs, which we are observing after parturition is not the first sign of estrous, it is usually the second sign. The reason behind this is, usually, the first estrum after parturition is a silent one, the reason behind this is the hypothalamus needs to get primed by the progesterone. Usually, what happens, the best example I can say is that during estrum little quantity of estrum is being secreted which results in the expression of estrum, but during calving, larger quantities of estrogen are being secreted for the relaxation of sacrociatic ligament, vulva, etc. Followed parturition during follicular development and ovulation, only a little quantity of estrogen is being secreted. So the animal could not able to differentiate between the larger quantity and the smaller quantity, so for which to differentiate that priming is required. So after parturition, the first ovulation happens, results in the formation of corpus luteum, and this corpus luteum secretes progesterone, and this progesterone primes the hypothalamus so that in the next follicular development, a follicle secretes estrogen, it could able to sensitize the hypothalamus properly and the expression of estrous seems to be in a better way. So Subestrum etiology, as I said the physiological basis is not exactly known, but it may be due to lack of estrogen and a potentiating action of Progesterone. So unobserved estrum may be due to managemental deficiencies in a short period of use. So other one is a managemental error, as the term indicates, the farmer/ the herdman, which has been involved in the farm is unnoticing the estrous signs, which are being exhibited by the animal. It is absolutely a man-made error, even though from the farm animal side everything is normal, even though the animal is showing the signs regularly or exhibiting the signs properly the farmer or the person who is involved in the farm

activity is not able to observe these signs properly, it may be due to lack of awareness on estrous signs. For example, in the case of developed countries, there is only a short number of large farms are there but in the case of a developing country like India, we could able to find there are a large number of small farmers out there because of animal husbandry activity is part of our livelihood. So, each and every small family in the village is being involved in animal husbandry activity, especially a dairy activity and it is being a part of the livelihood. It is not that subsequent generation will be having awareness on the estrous signs and it is our duty to educate them in a better way to understand or brief about the clinical science of anestrum so that they could be able to understand and observe these signs properly and they could able to avoid this False Anestrum. We have come to the end of today's session, so we have seen what is False Anestrum, so which means that the animal or the ovary will be having in CL, and because of that CL, the animal is not exhibiting signs of estrous and we have seen the etiological factors such as a normal physiological condition such as pregnancy and pathological conditions also. We have seen examples such as Mummified maceration, Pyometra, Mucometra, etc., and in addition to that we also had seen Sub estrum, Silent estrum also and these are all the factors which are being contributed for False Anestrum.

So, with this, we will conclude today's session and we will continue the diagnostic and treatment protocols in the next session.

Thank you.

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PDF: False Anestrum and its Types

3 False Anestrum Diagnosis and Treatment

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Transcript

Hi! Friends greetings,

In the last session, we were discussing False Anestrum and the etiological factors such as pregnancy, which seems to be a physiological factor for False Anestrum. The pathological factors such as Persistent corpus luteum, sub-estrum, etc., we have discussed. In continuation that we will be focusing today on the Diagnosis of False Anestrum, for any condition or any disease condition, Diagnosis plays a very important role, the reason behind this, unless we diagnose the condition correctly, our clinical intervention or therapeutic intervention may not be successful, hence we need to give more importance in diagnosis and for diagnosing the False Anestrum the diagnostic methods which include a basic History briefing by the farmer above the animal, the other one is clinical signs. Of course, for this condition, there will not be any clinical signs, that is the clinical signs which need to be understood by the farmer and it needs to be briefed. The other one is the Rectal examination, and the other one is Ultrasonography. There are only four methods, however endocrinology or assessing the hormones if it is required, then we may proceed or with simple

rectal examination and ultrasound examination itself, we would be able to diagnose this condition in a very better way.

History: why is history important? Because Vets are not the person who is always present for the animal, a farmer is the only person who is available at the animal's nearby station or nearby position to observe the clinical signs, so whatever the clinical signs are being exhibited it can be easily observed by the farmer and it can be briefed to the veterinarian which helps well in diagnosing what the condition could be. So, in history, based on the information such as failure of displaying of estrum or overt signs of estrum by the animal such as after attaining puberty or after postpartum two to three minutes. The farmer also should have basic ideas. For example, after puberty, the farmer is expecting the heifer should be showing clinical signs with a period of 18 to 21 Days or after the first cycle, if the animal is not showing any signs of estrum, that has to be observed by the farmer. Similarly, in the case of postpartum animals, the farmer has to at least observe the clinical signs after a waiting period of around 60 to 90 days, even after 90 days if the animal is not showing any clinical signs of estrum, then probably that has to be noticed by the farmer and it has to be acted accordingly. Often we can see the farmers waiting for the period of six months, eight months, nine months because their objective is only focusing on the lactation and they may be forgetting about the clinical signs. So at no more than 90 days, if the animal is not showing signs of estrum, it needs to be brought to the knowledge of the veterinarian.

The second one is Clinical signs, as we said earlier the definition itself says, there is the absence of overt signs of estrum, symptoms of estrus shown with cyclicity which subsequently ceased and revert into the period of estrus, the animal may show signs of estrum, after the period of more than 21 days or more than one month, if the animal is not showing signs of estrum, then that has to be noticed.

The next one is the Rectal Examination so when I say about the rectal examination, it is a basic or fundamental procedure for any veterinarian and moreover, it is a very simple technique to perform

a gynecological examination, so rectal examination plays a vital role in the gynecological examination. So, in the rectal examination, you could able to find a mature CL in one of the ovaries and a flaccid uterus, which means that the animal is in the period of diestrum, so when you palpate the ovary, you could able to find a corpus luteum, then probably you could able to feel the crown of the corpus luteum, then probably it is in the period of diestrum, which is indicating the animal is cyclic. The problem is either it is not showing signs of estrum, or it is being missed or the second possibility is the uterus will be having mild tonicity. either there will be regressing CL with follicle or a developing CL, which is indicating the animal is in pro estrum or metestrus or sometimes estrum also. At all stages you would be able to find, either a prominent corpus luteum or a regressing or developing corpus luteum, which is indicating the ovaries functional, the problem is either there are no clinical signs or if it is showing clinical signs, it is being missed out. So, from the rectal examination, you would be able to easily find whether the ovary is functional or not.

The next thing is the examination of Postpartum anestrus cows at days 60 to 90 gives an idea about whether the animal is entered into the period of cyclicity or not. The permissible period is 60 to 90 days, after that, if there are no signs, perform a simple rectal examination and you will be able to find it. So, you would be able to find a prominent corpus luteum in the ovary, indicating the animal is cyclical.

So the next beautiful method of diagnosis is, by means of using Ultrasonography, so ultrasonography is considered to be the third eye for the gynecological person, by means of performing ultrasonography you would be able to exactly find the ovarian status of the animal. So what way is ultrasonography an advantage over the rectal examination? When you find that sometimes in the case of buffalos or sometimes in the case of cattle also, you were able to find ovaries with corpus luteum in a prominent way that means by rectal examination you were able to feel the crown but not in all the cases. Often in buffaloes, this CL will be embedded, which means

that it will be buried inside or sometimes it is encapsulated, in such conditions your thumb finger may not be able to differentiate the crown of the corpus luteum, and often we will be diagnosing this as muthuri, in such condition ultrasonography plays a very important role in diagnosing accurately the condition of presence of corpus luteum. In this case, you could able to find the same animal the left ovary which is having multiple small follicles, the same animal, the right ovary is having, a prominent corpus luteum, with the help of the doppler mode we could able to find the boundary also very clearly if the size is more than 15mm in diameter then probably it is a prominent CL. Prominent CL can be noticed during the period of diestrus. I'll show the video and this video clearly explains the presence of multiple small follicles in the ovary, you could able to find the left ovary is having a small follicle which is anechoic, fluid is anechoic, you could able to find only the presence of small follicles which is indicating the ovary smooth. Similarly, from the same animal, if you see the right ovary, you could able to find a hypoechoic boundary defined structure that is a corpus luteum and it measures more than 15 mm diameter and in this case, it is around 23.5 mm in diameter which is absolutely indicating the animal lesion period of diestrum because of its prominent corpus luteum. So, the other diagnostics with the help of an Ultrasound, if any uterine pathology is it you would be able to identify in a very easy way and probably here you would be able to find a condition of a mucometra, where the uterus is completely occupied by the fluid. Usually, in the case of non[1]pregnant animals, there should not be any uterine involvement but here you could able to find that the uterus is completely occupied by the very clear fluid, so which means that the uterus is occupied by the fluid either the ovary may be having a follicle with CL or without CL also. So uterine pathology, which is also a contributing factor for False Anestrum, can be diagnosed easily with the help of an ultrasound.

Next, let's move on to the Treatment: so, understanding the basic principle diagnostic methods helpful in approaching the case with proper therapeutic intervention, the treatment protocols the first

simple thing is educating the farmer are herdsmen. As I said earlier the farming community, there are subsequent changes in generations and more number of peoples have been engaged commercially, they may not be having a clear-cut idea on basic signs or basic estrous signs of an animal, hence the basic signs of the estrum animal has to be brief to the farmer such as estrous signs, which includes there may be a bellowing or the animal will be bit excited and drop in milk production, frequent urination in terms of buffaloes, the vulva may be bit swollen and a clear vaginal discharge, these are the simple signs can be briefed to the farmer for which they could able to identify the estrous signs in a better way. The second one is unobserved estrous, in unobserved estrous, mainly due to poor farm conditions, if the person is not involved properly then he may be skipping the estrous signs, they may be focusing usually in case of commercial farms, the laborers who are mainly involved in milking cleaning and all, so observation skills will be lacking. In such conditions we need to educate them to improve the management practices, a poor management condition also being a contributing factor in suppressing the expression of estrous signs, hence the farm conditions also have to be looked out by the veterinarian and accordingly suggestions have to be given. For example, with proper ventilation, proper feeding, etc. So increased regular observation thrice a day for estrous, for which we need to maintain the records, if suppose an animal is calved this month and accordingly a period after two to three minutes we need to give more importance or in case of the cyclical animal, if one cycle is being exhibited today after the period of 18 to 20- 21 days, we need to give more time in observing the animal whether the animal is exhibiting estrous signs or not. Another one is the provision of adequate lighting to improve the estrous detection and in case of the farm conditions, you can even try estrous detection. In other advanced conditions, probably you can go for teaser bulls in identifying the estrum in a larger group of animals or you can do a careful and frequent examination of the cows for predicting and confirming the estrum inbreeding, by means of maintaining

records. A specific treatment using a prostaglandin or progesterone therapy and fixed-time artificial insemination is highly effective in approaching a case of False Anestrum and this hormonal or controlled breeding is separately discussed in another topic as Estrous Synchronization, so in that topic, you could able to understand what are all the different protocols which have been implemented in synchronizing the estrum as well as ovulation. But being a False Anestrum, a simple drug, if you're diagnosing the ovaries having a functional or persistent corpus luteum, then the best therapeutic approach is by means of using prostaglandin-based treatment. Prostaglandin is a treatment of choice for persistent corpus luteum and inducing estrum. Natural or Synthetic analog of prostaglandin F2 Alpha (PGF2 Alpha) as a single dose has been used with a reasonable degree of success for the management of silent estrous cattle and buffaloes. You will be able to find the synthetic prostaglandin which contains Cloprostenol sodium, the total dose for synthetic one is 500 micrograms. Similar to other preparations of Natural prostaglandin, which contains Dinoprost tromethamine, the total dose is 25 milligrams. Both synthetic and natural prostaglandin can be given in intramuscular root, the other route is also there Intravulvar submucosal route, you can reduce the half dose. The intramuscular dose is easy to administer, and you would be able to find better success by using the prostaglandin[1]based approach.

So, coming to the end of the session and this session we have discussed what is False Anestrum and the diagnosis by different methods which includes History, Clinical science the most important thing is Rectal examination and Ultrasonography and followed by the treatment protocols according to the type of an Anestrum. For example, if it is in managemental error, accordingly we need to be addressed. There are two different methods, one is the Management method and the other one is the Hormonal method. In the case of hormonal methods, the best drug of choice is prostaglandin and accordingly synchronization protocols can be

adapted to successfully approach this condition and we will continue in the next session with True Anestrum.

Thank you.

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4 True Anestrum, Types, Etiology and Pathogenesis

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Transcript

Hi! Friends greetings,

In the last session, we discussed False Anestrum, Diagnostic methods, and Therapeutic interventions. In continuing the lecture on Anestrum, we will be focusing today on True Anestrum, as we discussed in lecture 1, the True Anestrum means, the ovary is smooth, there will not be any corpus luteum on the ovary, so it means in True Anestrum you could able to find small inactive ovaries, often it will be spindle-shaped also, without a functional corpus luteum. It may be due to insufficient release of gonadotropins or the failure of ovaries to respond, often you could be able to find the ovaries there will not be any corpus luteum rather you could be able to find small multiple follicles. In all structures of the True Anestrum, you will be finding, the ovary will be smooth either with the presence of a small follicle medium or large molecule also but there will not be any functional corpus luteum. So, what are all the factors which are being contributed for True Anestrum:

Etiological factors-

The first one is a low plane of nutrition if the animal is not in

a proper nutritive status, which is the main primary problem for True Anestrum, so it is the most common cause of the lack of energy and protein deficiency of minerals especially phosphorus(P), cobalt(Co), iron(Fe), copper(Cu), iodine(I), and manganese(Mn) along with vitamin A. If the mineral deficiency is there, definitely the animal will enter into the period of True Anestrum, there will not be sufficient quantities of GnRH release, in turn, estrous signs will not be exhibited, so nutrition plays a very important role in regulating the reproductive cyclicity. In addition to that, the second most important factor is heavy lactation, so negative energy balance. So often we are encountering cases like the transition, during the transition period, before calving, during calving, and after calving. So during the last few weeks of parturition, most of the farmers are not interested in giving enough for surplus feeding to the animal, the reason behind this is that if they give more feed, they are having an impression that the fetus will be growing larger which will end up in Dystocia, but that was a condition that has been noticed many decades back when we are using an imported summons, but right now all the bulls which have been reared under local conditions and accordingly it has been inseminated, so that concept is absolutely wrong. So, during the transition period, the farmer has to give additional importance to maintaining the regulation of the dam as well as the fetus, so during the transition period if the nutritive status is not good then definitely during lactation or even during the calving itself the animal will go for downer, and it will enter into the period of negative energy balance.

The other one is Chronic debilitating diseases like joining disease, tuberculosis, etc. If the animal is very poor, then probably animal will not show clinical signs of estrum, that is why I prefer to say that the reproduction of breeding is a sophisticated one, which means that if normal physiological functions of the animal are quite okay, then only the animal will enter into the period of breeding. So other causes which include seasonal and environmental influences, huge variation in season also can affect the release of the gonadotropins, for example, extreme summer affect the release of the

gonadotropins, which is commonly noticed in the case of buffaloes. The other one is closely confined dark stables, lack of exercise combined with nutritive factors, if the farm is poorly managed then that is also one of the factors for contributing to the True Anestrum.

So Suckling-During suckling, the prolactin reduces the ovarian sensitivity, so unless suckling is where the animal will be in the period of Anestrum, that is why here weaning plays an important role in entering the dam into the next cyclicity.

So Pathogenesis: in what way is the pathogenic mechanism being involved in regulating the estrum in Anestrum, so usually the concentration of gonadotropin is almost negligible in late gestation, so high progesterone, which is being secreted by the corpus luteum during pregnancy or end of gestation along with the placenta, also secret progesterone and estrogen from the placenta during the last trimester of pregnancy and put together these hormones causing negative feedback, which results in inhibition of hypothalamic pituitary-axis and because of that sufficient quantities of GnRH are not being released in addition to that FSH and LH is also being affected, in turn, the animal is entering into the period of True Anestrum with reduced ovarian activity.

So now Types of True Anestrum:

In the type, one is Physiological, another one is Pathological: – In physiological Anestrum: you would be able to find Pre-pubertal Anestrum, which means that before animals attaining puberty there will be Anestrum, which you will be finding, small follicles or medium size follicles which are quite a normal one. Another one is Gestational Anestrum, so which means that the animal will be pregnant, the ovary will be having a corpus luteum, this secretes progesterone and this suppresses the GnRH and because of that animal is entering into the period of Anestrum and this is also very common in pregnancy.

The next one is Postpartum Anestrum, all the animals after parturition, will undergo a period of Anestrum, which is quite normal. In the case of cattle, it is comparatively shorter than the buffaloes during this period. This period is referred to as

Puerperium, this is a period from the period of calving till the resumption of the next ovarian cyclicity and during this period all the uterine involution and uterus will come back to their original position, and after that, the ovarian rebound will happen and subsequently, the follicular development activity will be initiated. Lactational Anestrum, during lactation Anestrum, as we said earlier prolactin is a dominant hormone which is suppressing the GnRH activity, so during lactation, the animal will be in the period of an Anestrum, that is why weaning plays a very important role in the resumption of next ovarian cyclicity. Almost all Prepubertal, Gestational, Postpartum, and Lactational are Physiological.

If this Postpartum Anestrum is going beyond two to three months, then it is considered to be Pathological.

The next etiological cause is the Congenital and Hereditary cause of True Anestrum. These three conditions are Ovarian agenesis or dysgenesis, Ovarian hypoplasia, and Freemartin.

Ovarian agenesis means the complete absence of an ovary, this condition is very very rare, it is due to autosomal dominant genes and in this condition, the animal will not be expressing any signs of estrum.

The other one is Ovarian Hypoplasia, this is due to the simple autosomal recessive gene with incomplete penetration and in this condition probably the ovary is not developed properly, it may be unilateral or even bilateral. In this condition also the animal will not show any signs of estrum.

Another one is Freemartin, this is a sterile female born co-twin with the male, because of the placental anastomosis and hormonal exchange, there may not be proper development of the ovary which results in absence of expression of heat signs. So, another type of True Anestrum is Pathological True Anestrum: Here I have categorized it into one, two, and three. Type one is Emergence, type two is Deviation, and type three is Growth. So, this chart will be able to explain in a better way about the Pathological Anestrum based on the recent concepts, based on ovarian dynamics. From the Follicular

Pool, there are four types of follicle growths are happening, For example: In the case of type 1, there will be a small follicle, which is being recruited from the follicular pool and after that, there is no progress, so no deviation is happening. So, in this condition, you could be able to correlate in diagnosis you will be able to find only small follicles in the ovary. In type 2, there will be a recruitment of the follicle, the follicular will be developing into the next stage, that is Growth is happening followed by Atresia. Here you could find the size of the follicle is comparatively larger, but it is ending up with Atresia. Another one is type 3, in this, you could be able to find, the follicle will be recruited from the wave pool, and after that development is happening and after that, it is attaining the dominant size also but there is no ovulation is happening. So, either the large follicle will persist, and it gets regressed or sometimes the presence of the follicle persistent results in follicle assist or sometimes after follicle assist, partial luteinization happens and it becomes a luteal cyst, all possibilities are there.

The other type is, there will be a development of the follicle, growth happens to attain dominance after that ovulation happens but after that, there will not be any regression of corpus luteum. So it is being acted as prolonged corpus luteum, in this, we will exclude type 4 because CL is there, we will take only till type 3, so in this condition what is happening often we could able to find, one follicle is getting developed in a smaller size that is Emergence so Emergence of the follicle is happening after that no activity, so this is Deviation, so after Emergence, the follicle is developing further but after that, it is getting deviated and the 3rd one is, it is attaining to the dominant stage, it has attained the growth but ovulation is not happening. So at any True Anestrum, you could able to find all these three types of varieties of follicles based on the size and this condition is mainly due to low pulse of LH release and increased sensitivity to negative feedback of estrogen on gonadotropin and some of the growth factors like insulin growth factor, epidermal growth factors, also contributing factor for this. Often, we are examining the animals of True Anestrum with the follicle size of

more than 6mm diameter, so this is the recent concept in Anestrum classification based on the variant dynamics. So, the summary of today's lecture, we have seen what is mean by True Anestrum and what are all the causative factors which are being contributed for True Anestrum, in addition to that we have seen the Types of True Anestrum in which Physiological Anestrum, Congenital Anestrum, and Pathological Anestrum and our intervention will be mainly focusing on how to approach a case of Pathological Anestrum.

With this, we will conclude today's session and we will continue the Diagnostic and Therapeutic portion in the next session.

Thank you.

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5 True Anestrum Diagnosis and Treatment-prevention Summary

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Transcript

Hi! Friends greetings,

In the last session, we have been discussing what is True Anestrum, what are all the contributing factors for True Anestrum, and what are all the types of True Anestrum. In which we have seen Physiological, Congenital and Pathological. In continuing, we will be discussing today on the Diagnosis of True Anestrum, like in the case of False Anestrum, the same measures only, one is History, Clinical signs, Rectal examination, and Ultrasonography examination.

History: So, based on the information provided by the farmer, the failure of displaying the overt signs of estrum, will be briefed by the farmer to the veterinarian, which will be the first thing in Diagnosing or suspecting the animal could be in Anestrum. The second thing is Rectal examination, as I said earlier Rectal examination is the primary and fundamental procedure, for all the veterinarians should know about it for the gynecological examination. By means of performing Rectal examination, you could able to find either small and smooth ovaries in buffaloes, which is almost spindle-shaped and it should be confirmed by repeated

examination at 10 days interval, the reason is, when you are examining the animal by Rectal examination, you could be able to find an ovary, it may be in any stage. For example, there may be a regressing corpus luteum or developing corpus luteum also, the animal would have been expressing in heat signs, it may be unnoticed also in such conditions when you perform rectal examination if you are finding both ovaries are smooth, then probably you can suspect it could be of True Anestrum, anyway, it is better to re-examine the animals after 10 days interval to re-confirm whether the ovary is smooth if suppose if it is not a True Anestrum, then after 10 days interval, you will be finding a prominent corpus luteum. If, suppose no prominent corpus luteum is there, then you can diagnose the condition as True Anestrum. Another method is Ultrasonography, as I said earlier, Ultrasonography is an accurate method in diagnosing the ovarian status of the animal. Small to developing follicles often you will be finding in True Anestrum, without corpus luteum.

These are all different animal images: (showing images) In this condition (1st image), you would be able to find multiple small follicles. Here (2nd image) you could able to find a medium-sized follicle is there, and Here (3rd image) this image, with all the different animals, you would be able to find almost a dominant follicle.

So we had seen in the classification of Pathological Anestrus, type 1(1st image), type 2(2nd image), and type 3(3rd image), according to the size of Emergence, Deviation, and Growth. This is a classical example, so in 1st image, you could able to find only mall follicles are there that is Emergence is happening, in the 2nd image you could able to find a medium-sized follicle it is growing but deviated so it is Deviation, the 3rd image you could be able to find a dominant follicle. Often the dominant follicle indicates the animal is in the estrum, but it is not. If CL is there, then only we can declare the animal is cyclic and in Anestrum, the follicle can even up to the size of 8 to 10 mm in diameter, which is normal in the case of Anestrum but there will not be any clinical signs of Anestrum. During estrum 8

mm follicles also can be able to secrete estrogen and better clinical signs of estrum can be noticed. So, this is small to developing follicles.

Another one is progesterone estimation. So, progesterone estimation the presence of basal level, which is 0.5 to 1 nanogram (0.5-1 ng/ml) of progesterone in the blood sample at an interval of 8 to 10 days up to examination, with confirmation diagnosed the animal has not entered into the period of the luteal phase, which is indicating the absence of estrum. If progesterone is being noticed in the blood circulation, then probably we can declare the animal is cyclic.

On two subsequent examinations of hormonal progesterone estimation at 8 to 10 days interval, it is showing less than one nanogram (< 1ng) is clear cut indicative of True Anestrum. The other one is Treatment, so when we recollect the etiological factors, the prime factor for causing True Anestrum is nutrition. so, the first thing is to improve the nutrition and supplement minerals.

So, improving nutrition in the sense, balanced nutrition, balanced nutrition is a rationalized one along with green fodder as well as dry fodder, so ad libitum green fodder, dry fodder is there it is preferable along with the rationalized balanced nutrition according to the status of the animal. In addition to that you need to supply a mineral supplementation, in addition, that you need to go for mineral supplementation. a patent preparation according to area-specific mineral mixture availability is there, so accordingly you can prefer that, and you can go for mineral supplementation. In addition to that, you need to go for managemental care under small farm conditions. So other management condition which includes, summer management, especially in case of buffalos, a summer Anestrum is very common, so the reason could be, during summer it causes suppression of GnRH released from the hypothalamus, heat stress cause suppression of GnRH released from the hypothalamus, in turn, the gonadotropins are also getting reduced and because of that the follicle is not developing properly hence estrous signs are being not properly exhibited, in summer condition how this

can be clinically or very simple approach can be undertaken by means of going for water sprinkling and this video is showing a small farm condition which is having a water sprinkling for the period of around 15 to 20 minutes for around 3 to 4 times a day will definitely help in approaching summer infertility in case of buffalos.

So, the other one is treatment, again treatment includes Hormonal as well as Non-hormonal approaches. The first simple approach is a simple Utero-Ovarian massage. For example, in farm conditions, a simple Utero-Ovarian massage induces 40 percent estrum in Anestrum buffaloes, by means of palpating or massaging you are increasing the circulation to the gonads which results in expression of signs, development of the follicles, etc. So simple Utero-Ovarian massage results in the induction of estrum.

Another one is Lugol's Iodine, intrauterine administration of Lugol's Iodine, acts as an antiseptic, and also iodine supplementation provided by the Lugol's Iodine results in good managemental practices to improve Infertility in Anestrous cows. Hormonal approaches: Hormonal approaches place very important and successful results, a simple approach is GnRH, Gonadotropins releasing hormone at the rate of 0.5 milligrams, can be repeated even after 10 days for the induction of estrum in Anestrum animals and GnRH analogs, like Buserelin, can be administered at the rate of 0.02 milligram as a total dose that can be administered intramuscularly, which results in induction of follicular growth and expression of heat signs.

Another one is Pregnant Mass Serum Gonadotropin (PMSG) or FSH was advised earlier but it is not recommended now because it can even cause superovulation or sometimes it can result in follicular cysts, because of its moral of life, etc. and because of that PMSG or FSH is not recommended recently. Another one is Short-term progestogens, either in the form of Controlled Internal Drug Release (CIDR) or Progesterone Releasing Intravaginal Devices (PRID) or along with ear implants that induce heat even in an anestrum animal. So CIDR, Progesterone Impregnated Intravaginal

Device (PRID) in the form of a CIDR or vaginal sponge is highly effective in the case of True Anestrum animals.

So, another hormonal protocol which includes progesterone injection followed by hCG or a combination of Progesterone + PMSG + Estrogen is also recommended. Another one is Clomiphene Citrate: Clomiphene citrate at the dose rate of 300 milligrams, daily for five days drenched as a suspension, after drenching of copper sulphate (CuSO4) solution. So, when Clomiphene citrate can be used or then Progesterone therapy can be used? a simple approach, when you are diagnosing a True Anestrum with only multiple small follicles, then Progesterone therapy is more effective and when you are diagnosing the True Anestrum with medium to a dominant follicle then Clomiphene citrate seems to be very effective. So, we could be able to find more than 70% of the cases by means of Clomiphene citrate therapy resulting in induction of estrum, not only estrum, ovulation also it has been helped. Another one is Insulin Based therapy, which is the recent one, use of insulin for induction of estrum in animals either alone or in combination is a fairly recent development and results in encouragement. The recommended dose is 0.25 international units per kg (0.25 IU/KG) in a subcutaneous route. It helps in follicular development, follicular maturation which results in better expression of the estrum.

Another one is the Non-hormonal approach but a plant-based one, so recently ethnoveterinary practices have become very advanced or are currently being implemented in veterinary science also.

In that, the plants synthesize varieties of phytochemicals such as alkaloids, glycosides, terpenes, and tannins, as a part of their normal metabolic activity and many of these have therapeutic actions when consumed by the animals. For example, Moringa leaves, daily administration of a handful of Moringa lives for the period of 10 days, it supplies, it increases the mineral contribution, so it induces the follicle growth.

Another one is Kalonji leaves, kalonji seeds also can be administered. Another one is Ashoka leaves

Another one is in Bambusa arusa leaves. All these administration by means of oral, which has been proved in terms of follicular growth development.

So other commercial plant-based heat inducers which include Prajana (HS), Janova, and Himfertin, also have been shown to have successful results in the case of True Anestrum. The dose rate is 3 capsules per day for 2 days, then wait for 10 days, if heat signs are not noticed then you can probably repeat for the same dose.

So, coming to the Prevention protocol:

The First thing is you can maximize the appetite and after calving. Usually, after calving, the animal enters into the period of Anestrum and it is getting extended, when you are managing the nutritive status, then probably you could be able to prevent the Anestrum. Second thing is to provide a palatable well-balanced and highly digestible diet to allow cows to meet their nutritive balances and you improve or maintain the immune status of the animal which helps in preventing the disease. After parturition or during the transition, there is the possibility of increased nepha which in turn results in increased heat in the body's production, in turn, it affects on entering into the period of Anestrum, if more ketone bodies are there, so you prevent that. Another one is to maintain minerals such as calcium and magnesium at and after calving. Animals must be maintained in a less stressful environment around parturition, so reduce the Weaning period, so here I would like to conclude one in Nutrition, another one is Management and the other one is Weaning. This management surrounding the periparturient, surrounding the transition period, surrounding the calving period, plays a very important role in inducing Anestrum during the postpartum period. So during calving or at the end of the gestation, we need to give extra cautious of the animal. So, what I want to conclude in Anestrum, two major things have to be approached: one is Anovulatory ovaries, the other one is an Ovulatory response. When we address Anovulatory ovaries and Ovulatory response, we can achieve success. These two contribute to Anestrum.

Anovulatory ovaries: Anovulatory ovaries can be addressed by

means of going progesterone therapy in the form of CIDR, Vaginal sponge, etc., By means of going for a Progesterone therapy you can be able to expect better estrum in case of Anestrum animals then, By means of using LH or any GnRH you can be able to address the ovulatory response, there is no point in inducing estrum followed by without ovulation, so inducing estrum is equally important along with ovulatory responses. By means of using LH or GnRH, you are not only ensuring ovulation, but you are also ensuring the quality of corpus luteum because sometimes due to luteal insufficiency the animal may be repeating or implantation or subsequent pregnancy may not happen, that also can be overruled by means of using this LH, hence by means of using Progesterone therapy and LH you could be able to expect a better conception.

Hi friends! With this I am concluding this session on Anestrum, I guess this lecture would have been useful to you for application in your field practice. In case of any clarification, you can contact me on either mobile or with the email id mentioned here. So, thank you for the opportunity given.

Thank you all!

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PART III

WEEK 3: ENDOMETRITIS

1 Introduction, Types, Incidence, Fertility and Normal Postpartum Events

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Transcript

Greetings!

Happy to reach you all in this virtual media. I am Dr.S.Sathesh Kumar, professor in the subject of animal reproduction. Currently heading the Department of Veterinary Gynecology and Obstetrics at Veterinary College and Research Institute, Orathanadu, a constituent unit of prestigious Tamil Nadu Veterinary and Animal Sciences University, situated at Tamil Nadu. In this series of lessons entitled management of infertility in cattle, I am going to take classes on Endometritis and in this session, we are going to deal with the basic Introduction about uterine infections as well as the Types and incidences of various uterine infections. The fertility of dairy cattle is a gateway of production and the backbone of the dairy industry and rural economy. Our country stands first in milk production globally, but the bovine population behind this production scenario is also huge, ever-rising infertility problems seem to be the major hurdle in the production status of the animals, if these obstacles could be resolved, we can attain a mammoth structure of milk production, a real white revolution. One of the

main factors that impact fertility, is the occurrence of uterine infections. The disturbed uterine environment, disrupts the Utero-ovarian function, leading to infertility. Hence, it is important to understand all the details about Endometritis condition in dairy cattle, right from its incidence, causes, diagnosis, treatment, and prevention in order to improve fertility. A parturition is an event of new life coming into being. At the same time, it's also a period of high risk for the mother and the offspring as well, and cattle is no exception, we can expect all sorts of problems at the time of parturition also.

After parturition, there is often an upsurge of microbial infection in the cow that ultimately has a greater impact on the health and reproductivity of the animal. Most microbial contaminants are eliminated from the uterus during the first three weeks of pregnancy by uterine involution and activation of innate immune defenses. However, failure in this comeback mechanism leads to various degrees of uterine infections. Puerperal Metritis, is one of the serious conditions among uterine infections. It's the inflammation of the inner lining and all the deeper layers of the uterus. It occurs within 21 days after parturition and it is also characterized by foetid reddish-brown uterine discharge and it's also associated with signs of systemic illness like fever, decreased milk yield, dullness, and signs of toxaemia, in that way Puerperal Metritis is one of the serious conditions after the parturition.

Then comes a lesser degree of uterine infection, that is Endometritis, this is the Inflammation of the endometrial layer of the uterus, the superficial layer of the uterus. It is characterized by the presence of purulent or muco-purulent uterine discharge. About 15% of dairy cows have clinical signs of uterine disease, that persist beyond 3 weeks postpartum. Next is Sub-Clinical Endometritis, this is also the inflammation of the uterine endometrium but where lies the difference? Here, there will not be any overt signs because this infection is characterized by a clear uterine discharge and interestingly it affects 30-35% of dairy cows between 4 to 9 weeks postpartum. Since there are no signs to be

found because of this Sub-Clinical Endometritis, this is considered to be the 'Silent Killer' of Fertility.

The major events that must be completed after parturition before a cow is likely to conceive again are:

Uterine involution,

Regeneration of the endometrium,

Elimination of bacterial contamination, and

Return of ovarian cyclical activity

The pre-requisite for all these normal postpartum events are the normal expulsion of the foetus, foetal membranes, and associated fluids at calving. Uterine Involution: Involution of uterus involves—

- physical shrinkage,
- necrosis and sloughing of caruncles and
- regeneration of endometrium.

Uterine caruncles are usually sloughed by 12 days after parturition. The sloughed caruncles along with the remains of the fetal fluids and blood from the ruptured umbilicus will be discharged as the lochial. This is a normal discharge in the postpartum period that is called lochial.

Epithelial Regeneration: Epithelial regeneration is completed about 25 days after parturition. Deeper layers of tissues require 6 to 8 weeks for a complete restoration, after parturition. It is followed by the Evacuation of Microbial Load: The postpartum environment of uterine lumen supports the growth of a variety of aerobic and anaerobic bacterias. Normally they are removed from the uterine environment by the defense mechanisms.

Ovarian Rebound: This is one of the important features for the animal to come into the next cycle. After parturition, the concentrations of the steroid hormones will decrease, the concentration of the follicle-stimulating hormone will increase, and it is very important in the stimulation of the first follicular wave emergence after parturition. The first dominant follicle that is selected from this follicular wave, will be around 10 to 12 days after

calving. The fate of this dominant follicle decides the cyclicity or the onset of cyclicity of the animal, after the parturition.

The Clinical signs of Endometritis: The nature of vaginal discharge is a mirror of uterine health status, it's always known, we can see that based on the nature of discharge, we can actually classify the conditions into Clinical Endometritis or Sub-Clinical Endometritis. First, we are going to see about Clinical Endometritis: here in the case of clinical endometritis the discharge will be purulent or muco-purulent, it will be readily detected from the vaginal discharge. The physical character and odour of vaginal mucus can be scored to assess the degree of infection. This is called the Clinical Endometritis Score. This score indicates the prognosis of the condition and the type of treatment we have to undergo. Basically, it has been characterized and scored as 0, 1, 2, and 3. If it is score 0, the discharge seems to be clear and translucent mucus. If the discharge is going to have some flecks of white pus, that can be graded as score 1, If the discharge is contained less than 50% white or yellowish pus, then it can be graded as score 2, If this discharge is going to have more than 50% of white, yellow or reddish pus, then it can be scored as 3. So, this will be given the various degrees of Clinical Endometritis condition.

Then we come to the Sub-Clinical Endometritis, as we have told in the last class itself, here the vaginal discharge is often clear, and the animal is not showing any signs of infection Typically copious clear discharge with thin consistency is a characteristic feature of subclinical uterine infection. The reproductive performance of these cows affected by subclinical endometritis is usually diminished, which will be reflected as repeat breeding syndrome. So, in this class, we have seen the various clinical signs and the various degrees of infection in the case of Clinical Endometritis as well as Sub-Clinical Endometritis. In the next session, we can see about the Etiology and Pathogenesis of the infected conditions. See you all.

Thank you!

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PDF: Endometritis

2 Etiology and Pathogenesis

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Transcript

A warm welcome to you all,

In the last sessions, we have seen the types and incidence of uterine infections as well as, the clinical signs and various degrees of effection of these uterine infections. In this session, I am going to deal with the Etiology and Pathogenesis of Endometritis. This is one of the important features, we have to know before attempting any treatment or prevention. In that way, this Aetio[1]pathogenesis is very important in the Endometritis lesson. If you see the causative factors, Endometriosis is a multifactorial disease, so only it is having importance. Determining its causative risk factors has great potential as a source of information, that must be considered for the treatment and prevention of endometritis. The major causative risk factors for the incidence of uterine infections are Extrinsic factors as well as Intrinsic factors.

The Extrinsic factors include the calving season as well as the environment and nutrition, as far as the season is concerned, we all know that the rainy season is more vulnerable for most of the infections, the general health status of the animal will be decreasing during the rainy season, a lot of endemic diseases are coming into being during this rainy season. And the environment, if it is having

endemic infectious agents then it is also a problem for this utility infections.

Nutrition's very very very very important:

The Negative energy balance usually happens during the postpartum period,

The Protein deficiency or excess,

Vitamin deficiency especially A, B, C, and E,

Mineral deficiency especially Selenium.

Then comes the Intrinsic factors:

Dystocia and Retention of the placenta (ROP) is a very important thing. How do you handle the animal during the parturient period, is very important and is one of the Intrinsic factors: Inappropriate and indiscriminate handling of the uterine environment during Dystocia and manual removal of retained placenta, is one of the predisposing factors for uterine infections. The Incidence of twinning, how twinning is going to affect or cause the endometritis, because of Dystocia and Retention of the placenta, there are more incidences of chances for dystopia during twinning in case of cattle.

Metabolic disorders: this is again going to be linked with nutrition, Hypocalcemia, Ketosis, all these conditions will pave the way for the entry of uterine pathogens.

After parturition, the anatomical barriers of the vulva, vagina, and cervix are breached, introducing bacteria into the uterus, including pathogens, along with the bacteria that is normally found in the uterine environment and this is one of the reasons, the breaching of the genital tract for the incidence or occurrence of endometritis.

What are the important pathogens that are going to affect or cause endometritis? The uterine disease is commonly caused by Escherichia coli (E-coli), Arcanobacterium pyogenes, Fusobacterium necrophorum, and Prevotella species.

The most prevalent pathogens are E-coli and Arcanobacterium pyogenes.

E-coli infections appear to precede and pave the way for other infections, in that way E-coli is a very very important pathogen in the cause of endometritis.

Arcanobacterium pyogenes, Fusobacterium necrophorum, and Prevotella species act synergistically and increase the risk of clinical endometritis and its severity. The uterine immune response to the microbes leads to the influx of neutrophils from the peripheral circulation into the endometrium and uterine lumen.

The presence of pathogenic bacteria in the uterine lumen and the associated inflammation of the endometrium, preclude the successful development and implantation of the embryo. so these animals cannot conceive while they are affected.

Normal implantation process: we should know about the normal implantation process, what is happening to the normal implantation, before going into the pathology.

Normally a blastocyst will elongate, and it will slowly get a position with the uterine endometrium, and it gets added so that it will end up in the implantation, it's a normal process blastocyst elongation> opposition> as well as addition.

In the case of endometriosis, especially sub-clinical endometritis, there is a secretion of Mucin 1(Muc 1), the expression of Mucin 1(Muc1), increases in the bovine endometrium in response to the subclinical infection.

It is an epithelial cell glycosylated transmembrane protein, that has a role in microbial defense, it is appearing as a role of defense mechanism.

But this Muc 1, is an anti-adhesion factor that prevents the conceptus from attachment and implantation. So, this is how it is going to affect.

In the normal implantation, we can see the blastocyst coming and attaching to the receptors in the uterine endometrium and it is going for the normal implantation.

In the case of Sub-clinical uterine infection, what happens? A layer of Muc 1, is going to appear between the receptors as well as the blastocyst, this will prevent the implantation of the blastocyst and blastocyst will be lost. There will be early embryonic mortality and conception will not occur and impaired implantation will lead to conception failure.

The next important factor is the Endotoxin:

As a part of the pathogenesis of uterine inflammation, the bacteria or the pathogens will be having bacterial endotoxins that are called the Lipopolysaccharides (LPS). These are the components of the outer cell wall of the gram-negative and gram-positive bacteria that are highly immunostimulatory.

The peripheral plasma of animals with E-coli infection of the endometrium have higher concentrations of LPS.

What happens? It is going to affect the Hypothalamus, the Ovary, the Follicle, as well as the Corpus Luteum. Here the LPS from the bacteria can attack the Hypothalamus and it can interfere with the pre-ovulatory sequence of endocrine events via suppression of GnRH and LH pulsatility and thus the ovulation is going to be affected.

The LPS from the bacteria can act at the follicular level, it will reduce follicular growth and it can also inhibit or suppress the protection of our synthesis of Oestradiol from the granulosa cell. Again, the LPS can attack the corpus luteum level also, the luteal phase of the animal will be prolonged, in the case of animals with endometritis or sub-clinical endometriosis. why? It can suppress the PGF2 Alpha and accumulate PGE, in that way the luteal phase/ luteolysis is affected and most of the time it will be having a prolonged luteal phase, and these are the problems how the LPS will affect the endocrine system.

And again, the Cytokines, the inflammatory products: These are the immune or inflammatory challenges that can affect reproduction at the level of the hypothalamus, pituitary gland, and gonads. How these Cytokines affect the theca and granulosa cells and production of androstenedione and oestradiol, respectively. In that way, the entire inflammation can affect the endocrine system.

In the case of Chronic Inflammation, the consequences are a little bit more actually. The chronic inflammation of endometrium may lead to conception failure due to-

- chronic scarring of the endometrium.

- obstruction of the uterine fallopian tube and over bursal addition will lead to complete sterility of the animal also.

In this session, we have seen in detail about the Etiology and Pathogenesis of Endometritis as well as clinical endometritis.

See you all in the next session

Thank you!

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PDF: Aetio-pathogenesis of Endometritis

3 Diagnosis of Endometritis

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Transcript

Happy to invite you all to this session. In the previous sessions, we have seen various aspects from Types, Incidents, Clinical signs, as well as the Etiology and Pathogenesis of Endometritis and Sub-clinical Endometritis. In this session we are going to see about the Diagnosis of Endometriosis, this is one of the important application parts in the case of Endometritis. Clinical endometritis, as we have already dealt with, there are very clear signs in the case of clinical endometritis. We can see the observation of vaginal discharge with pus, it's a very clear indication, this is one of the first methods to Diagnose. Observation of the vaginal discharge, if it is going to have a purulent discharge, pus, then we can say that animal is having a uterine infection.

The next thing we have seen about the observation when the animal is going to be in natural condition, then in case of Clinical Examination, the contents of the vagina can be examined by passing a gloved hand, clean sterile gloves into the vagina, then you can get the secretions of the or the mucus from the vaginal passage. It's very clear if there is any pass, the animal is going to have an infection. We have seen about the manual vaginal examination, and this is here another method with the manual technique using a

vaginal speculum, the Vaginoscopy. Using this Vaginoscopy, vaginal speculum, we can inspect the mucus flowing out of the cervical loss, bypassing the vaginal speculum into the vaginal canal, you can just see the cervical loss and if there is any discharge coming out of that cervix, we can notice that one. And this is one more instrument or equipment Metricheck, this is going to be a stainless-steel rod. it has a hemisphere made of rubber in the tip of the stainless-steel rod, this can be passed into the vagina, and you can go and back rake it, while taking back it will be coming along with the discharge and based on the nature of discharge you can grade it as we already discussed we can grade it.

(showing score on pictures 1,2,3, and 4) This is very clear that is score 0, this is with some sort of flex of pus that is going to be score 1, this is with less than 50% of pus(<50%) it is going to be can be scored as 2 and more than 50% pus(>50%) it is going to be scored as 3 and this is one of the important latest equipment that is being used for identifying the degree of infection. Sub-Clinical Endometriosis: all the time we have seen about clinical endometritis, but it's very easy because it's all known the discharge is there and you can see the nature of discharge, you can also get that pus material and everything, you can very easily say that animal is having an endometritis condition.

But Sub-clinical endometritis, as we already told there are no clinical signs at all, the discharge is very much clear, so the problem here we have to Diagnose, and it also leads to the repeat breeding conditions. so, it is very important to Diagnose the Subclinical endometritis first, especially in case of repeat breeders. There is a massive infiltration of the endometrium we have already seen that and uterine lumen with the neutrophils/the Polymorphonuclear leucocytes (PMN) cells.

The detection of neutrophils/the PMN cells, is one of the important things, in the case of diagnosis of Sub-clinical endometritis. The first thing the White side test: it is going to be a very simple, field-level, rapid 'Cow-side' test. It can be performed very easily, you have to collect the cervical mucus aseptically, mix

1ml of cervical mucus with 1ml of 5-10% sodium hydroxide solution, mix it well and allow it to boil in a spirit lamp or any lamp. once it gets boiled, cool it under running tap water, that is a very important thing, there will be some color changes. If it is going to be clear, without any color change, then it is normal, no infection indicates that there is no infection at all. But the degree of color will change from yellow color, light yellow to dark yellow, light yellow color indicates mild infection and yellow color indicates moderate infection, the dark yellow color has a severe infection. The positive reaction for the White side test is due to the presence of neutrophils/leukocytes. These neutrophils or leukocytes will react with the sodium hydroxide and the color change occurs. In the case of normal cervical mucus, there will be very few numbers of neutrophils so the color change is not evident, but in the case of sub-clinical endometritis, the cervical mucus contains a higher number of leukocytes or neutrophils, that will evince or change the color of the reaction.

And the next method is Endometrial cytology: this is one of the reliable methods for diagnosing sub-clinical endometritis. Endometrial cell samples can be subjected for cytological examination, the methods used for collecting the endometrial samples are –the Uterine lavage technique as well as the Cytobrush technique.

First, we will see about the Uterine lavage technique– a small volume of 10-20 ml of 0.9% sodium chloride solution, that is normal saline, is infused into the uterine body with a syringe attached to a sterile AI sheath. The uterus was massaged. The fluid was recovered by negative pressure aspiration, and it is being transferred to the collection tube and can be centrifuged. The sedimented cells can be taken and they can be streaked onto a sterile slide and can be dried and stained with a normal Giemsa stain.

Next is the Cytobrush technique: It's one of the recent techniques, adapted from human technologies. The Cytobrush will be having a sanitary plastic sleeve, it will be having a brush-like appearance in the tip, and it can be covered with a sanitary plastic sleeve. It can be

passed into the uterus transposing the cervix and it can be rotated along the uterine wall. The endometrial cytology or the cell samples can be taken from this brush by rolling over the slide, The slide can be dried, and it can be stained as described previously.

How to Interpret? here is a slide after staining, you can see the different types of cells– the endometrial cells, the pathogen, and the inflammatory cells. So, by counting 100 to 200 cells in each field, you can now know the percentage of polymorphonuclear (PMN) cells. Different thresholds of PMN cells during di. rent periods of postpartum will indicate the degree of sub-clinical endometritis. During the postpartum 20 to 30 days postpartum period, when you are going to collect the endometrial samples, if the PMN cells are going to be more than 18% (>18%)then it is considered as sub-clinical endometritis, if it is 30 to 40 days postpartum more than 8% (>8%)PMN cells will indicate sub-clinical endometritis, and if it is 40 to 60 days postpartum more than 5%(>5%) PMN cells itself will indicate the animal is having sub-clinical endometritis and thus this is one of the reliable methods to diagnose the sub-clinical endometritis.

This is another method the Ultrasonography: it has been used as a method to diagnose sub[1]clinically based on the presence of intrauterine fluid and the evaluation of uterine wall thickness. Usually, you'll be having, you could see this is a normal one (showing 3 different ultrasonography pictures), where there is no fluid and here the uterine lumen with a little bit of fluid and here the uterine lumen with more fluid and this indicates the degree of infection. What's the biggest thing here is even in estrous, you will be having some sort of nutrient fluid accumulation. So, some of the reports are saying that ultrasonography is not the most reliable method for diagnosing sub-clinical endometritis, but anyway, the cytological study is one of the important ones for diagnosing sub-clinical endometritis.

To put it in nutshell, as well as a different Diagnosis of endometritis, as well as the sub-clinical endometritis, Clinical endometritis can be based on clinical signs, that will be very good

we could see the purulent or mucopurulent discharge and vaginal contents should be inspected for the presence of pus, then we can diagnose it as Clinical endometritis, but in case of sub-clinical endometritis, there are no clinical signs, the clear discharge is one of the biggest problems, some animals will show a copious discharge that is one of the indications thin consistency, so in such animals, we can suspect for subclinical endometritis and usually the animals with subclinical endometriosis will be having a repeat breeding condition and the most reliable method for diagnosis of clinical endometriosis is the White side Test as well as the Cytology Test, it can be a field test or a laboratory test. So, this is one way of diagnosing sub-clinical endometritis.

So, we have seen in this session the various methods of diagnosing endometritis as well as sub-clinical endometritis. This is one of the important steps before deciding on the treatment, so we have to see about the clinical status, the nature of vaginal discharge, as well as based on the laboratory test you diagnose the condition, then we can go for the treatment. So, the Treatment we are going to see in the next session.

See you all.

Thank you!

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PDF: Diagnosis of Endometritis

4 Therapeutic Approach of Endometritis

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Transcript

Happy to see you all in this session.

So far, we have seen the basics of the Types, Incidence, the Causative factors, and Pathogenesis of Endometriosis conditions. In the last session we have seen about the practical applicability, that is the Diagnosing the endometriosis condition and especially the sub-clinical endometritis which needs elaborate study and it's very important also. Once we have Diagnosed the condition, we need a remedy, for that only we are going to see about the Therapeutic approaches for the endometritis conditions. Basically, we should know that there is a difference between the influences of Progesterone as well as the Oestradiol in the uterine environment. A high progesterone environment usually suppresses the uterine gland and cervical mucus secretions, myometrial contractility, the phagocytic activity of the uterine neutrophils but a high Oestrodiol content will increase the vascular porosity of the uterine lumen, promotes the migration of leukocytes and eosinophils, increase concentration of immunoglobulins, favors the physical clearance of inflammatory fluid. So basically, the progesterone environment will be a dry one and is high oestradiol will be a little bit more secretey in nature.

Progesterone influence thus is permissive to uterine infection on the contrary Oestrodiol environment facilitates the removal of microorganisms, this is a basic one for the Therapeutic approach.

We all know that Prostaglandin is one wonder drug, that can cut off the luteal phase by inducing luteolysis.

The benefit of PGF2 Alpha administration arises from the induction of oestrous in cows having a responsive corpus luteum, PGF2 Alpha will be very effective in inducing the estrous. The PG-induced oestrous leads to the physical expulsion of bacterial contaminants as well as inflammatory products.

PGF2 Alpha is not only a luteolytic activity but is also pro-inflammatory.

In that way, the PGF2 Alpha is one of the important agents in cleansing the uterine environment.

Uterine lavage is another technique, as we have already seen in the case of Diagnosing the sub-clinical Endometrium for the Cytological study, here we are going to see the same technique used as a Treatment, this can be again done the uterine lavage with a sterile normal saline solution, it's a common treatment for the endometrial inflammation. Uterine lavage should be conducted for three consecutive days from the day of oestrus, in terms of cleansing the uterine environment. Uterine lavage can be done by infusing a sterile normal saline solution into the uterine body, using an intrauterine stainless-steel catheter, just by massaging the uterine lumen you can collect the fluid back, by back racking or by aspiration, so that the uterine environment is totally cleansed.

PGF2 Alpha treatment and Uterine Lavage favours the elimination of inflammatory products and induce uterine contractions that facilitate the evacuation of contaminants. Next comes the Antibiotic Therapy: this is also one of the important areas as well as a crucial area in the treatment of endometritis. There is a lot of chances for the development of antimicrobial resistance if you are going to use indiscriminate antibiotics, without any normal study. The Intrauterine Antibiotic Therapy is not indicated in clinical endometritis, why? the efficacy of these antibiotics in the local

environment is diminished by the presence of pus and organic debris in the uterine fluid, as well as the oxygen-deficient atmosphere, which will also prevent the action of antibiotics. Again, most of the antibiotics are irritant to the uterine endometrium.

The systemic administration gives a better distribution in the tubular genital tract and eliminates the risk of damage to the endometrium. Broad-spectrum antibiotics such as oxytetracycline (in the dose rate of 22 milligram/kg body weight) will provide an effective minimal inhibitory concentration in the lumen and uterine tissues.

Systemic Streptopenicillin: is one of the simple basic antibiotics, which is having covered a wide range of infections. A Systemic Streptopenicilline (2.5 to 5 gram) total dose results in the genital tract tissue and lumen concentration, similar to that of the blood plasma concentrations.

Other anti-microbials such as metronidazole, ciprofloxacin, and cephalosporin are also administered effectively in the treatment of endometritis. Sub-clinical endometritis is again a special one, here intrauterine treatment of antibiotics can be followed because there is no pus or debris to affect the action of antibiotics and usually intrauterine fluoroquinolones just like Levofloxacin or Ofloxacin or Ciprofloxacin are found to be effective in the treatment of subclinical endometritis.

A combination of Levofloxacin or Ofloxacin + Ornidazole + alpha-tocopherol provides better recovery of uterine infections.

Levofloxacin or Ofloxacin is having a bactericidal action,

Ornidazole is having action against the anerobic bacteria,

Alpha-tocopherol is a powerful antioxidant, we have to see that there is a lot of oxidative stress at the time of endometritis and this alpha-tocopherol will take care of it again by an antioxidant action.

The next one is the Intrauterine Antiseptic Therapy and the most preferred one is a povidone[1]iodine that is the polyvinylpyrolidone-iodine is a broad-spectrum microbicide with the potency to inactivate bacteria, fungi, as well as protozoa, it's a wide range of activity. A 2% povidone-iodine infusion into the uterus for three

consecutive days from the estrous, will improve the fertility of the animal. What can be a successful approach? after seeing all these things, we have seen about the Antibiotics, Antiseptics and PGF2 Alpha, after seeing all these therapies, what is a successful approach? A Combo therapy, a combination of systemic antibiotics, we are not going for intrauterine antibiotics.

A combination of systemic antibiotic + intrauterine antiseptic + Hormone therapy that is PGF2 Alpha and sexual rest in the induced oestrous helps in successful conception in case of endometriosis condition, especially align of treatment can be suggested: Injection of streptopencillin 5 grams intramuscularly (5 gms; IM) along with a diluted Povidone-Iodine for three consecutive days from the day of estrous. Followed by Prostaglandin (PGF2 Alpha- Inj. Cloprostenol 500 micrograms/ Inj.Dinoprost tromethamine 25mg; IM) administration during the mid-luteal phase and an induced estrous will be there, you have to leave that estrous, give a sexual rest and then you can go for the breeding from the next cycle. This is one of the successful approaches for treating endometritis conditions in the case of cattle.

One more recent Treatment is being reported and still, it has not come into the field, but we have to know this because in the future Platelet Concentrate therapy (PC) will be enforced in many conditions, here the Platelet Concentrate enriches the uterine environment with factors necessary for embryo development and counteracts eventual sub-clinical endometritis by its anti-inflammatory properties. It has been reported that administration of Platelet Concentrate (PC) 10ml into the uterus of the animals at 48 hours after insemination, here we are going to breed the animal and then we are going to infuse the platelet by 48 hours after insemination, it produces encouraging results in case of repeat breeding animals. Yet a lot has to be studied using Platelet Concentrate therapy (PC) and it can be applied in the field condition in the near future.

One more alternative therapy we are going to have for the endometrial condition, any Uterine infections:

We are talking a lot about antimicrobial resistance nowadays, so indiscriminate usage of antibiotics leads to resistance conditions. We have a lot of literature that has been documented, ancient literature documenting with a lot of herbal therapy. So here we are going to have two important components of natural origin, that can be effective in treating the endometritis condition: – One is Radish (Raphanus sativus), it is going to have a very good anti-inflammatory activity because of its 'raphanin' content, that is having antibacterial activity, antifungal property and it's also strongly active against the E-Coli. So Raphanus, that is radish is one of the important components in treating.

The next Cumin (Cuminum cyminum), this is having the volatile oil of this Cuminum cyminum /cumins is active against E-Coli Staphylococcus and other bacterial agents. We have already seen that E-Coli is one of the important uterine pathogens that is going to cause uterine infections, both these radish as well as the cumins are having an effect against the E-Coli and they are having a multifactorial effect on the antifungal and the antibacterial also.

How to apply:

A 10 gram of wet grounded Cumins can be smeared on the tongue of the animal, One piece of Raphanus sativus i.e., is Radish (150-200 g/day), it can be given, Both of them can be given orally for five days from the onset of estrous, and you can inseminate the animal or breed the animal in the next cycle and there are reports that there is about 85 percent recovery by giving this treatment. It can be tried in repeat breeding conditions where we can avoid the indiscriminate usage of antibiotics.

So, we have seen in this session, the various, Therapeutic approaches ranging from the PGF2 Alpha treatment, Uterine Lavage, Antibiotic treatment as well as the alternative treatment with the natural product of plant origin.

We have to decide as a veterinarian, as a clinician, we have to decide first the Degree of infection and based on the diagnosis you just decide, what is the line of treatment you want. As far as the antibiotics are concerned, it's again one more concern is, you

have to select an antibiotic based on the sensitivity in your area, in our experience Streptopenicillin is going to work very nicely, in most of the conditions. In the recent reports, we are seeing a lot of reports about the subtuophore but still, it can be decided by the veterinarians in that region by going for a sensitivity test in the lab. I hope you are now clear about the Therapeutic approaches for the endometritis condition in cattle.

See you all.

Thank you!

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PDF: Therapeutic Approaches for Endometritis

5 Prevention of Endometritis and Summary

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Transcript

Last session in this series of lessons about Endometritis. All along in this series, we have seen the basics of the Types and Incidences, the Causative factors, the Etiology and Pathogenesis, the Diagnosis of the clinical conditions, clinical signs and we have also seen the Therapeutic approach to treat the sub-clinical and endometritis. In this session, we can see a little bit about the preventive measures. We can take to prevent the incidence or occurrence of uterine infections. Cows with clinical endometritis were 1.7 times more likely to be culled for reproductive failure, than cows without endometriosis. This shows the seriousness of the condition, even though we see that it is only infection but the incidence of culling because of this condition is very wide. Good management practices in the transition period i.e., the prepartum and postpartum period can minimize or even avoid cow uterine infections and prevent the prevalence of endometritis disease. So transition co-management is one of the important factors in preventing uterine infections.

So Major risk factors that are going to affect this transition period that is the pre-parturition and the postpartum period.

Periparturian period:

First is Hygiene, it may be animal hygiene or environmental hygiene,

The second one is, the Immune status of the animal in general, and

The third one is, the Nutrition and Metabolic status,

sometimes all these three factors are intermingled but anyway we are going to see one by one. The Hygiene, it's very clear or well known that the very hygiene environment will be free of pathogens or reduced pathogens so that the animals are not being affected. So, paying attention to the hygiene of the cattle accommodation and calving facilities will pay dividends in terms of cleaner surfaces and less contamination for animals. So, this is very important the housing and management of the drainage everything will take care of this hygiene of the environment as well as the animal.

Immuno modulation is one of the important terms and has a very big role in preventing or controlling uterine infections. Understanding of uterine microbiome has created opportunities for the development of preventive measures to improve the management of uterine diseases. Avoidance of endometriosis depends on how effective the integrated process of adaptive events like well-regulated immune response is at limiting the burden of effects of bacterial pathogens.

So in that way animals defend themselves against pathogens using two important mechanisms is Tolerance and another one is Resistance.

What is Tolerance?

The Tolerance of the animal is the ability to limit the disease severity induced by a given pathogen burden and Resistance is the ability of the animal to limit the pathogen burden through the development of immunity. So, this Tolerance and Resistance are important factors in the immune status of the animal. Metabolic stress associated with the transition period and lactation compromise both Tolerance and Resistance. So only there is a problem in the Incidence of uterine infections. When we say about the Metabolic status: Negative energy balance favors the

development of metabolic disorder this is a very basic one. What is the negative energy balance? How it can be affecting the Metabolic disorder? What are the major Metabolic disorders?

Hypocalcemia

Ketosis

High non-esterified fatty acids and

Triacylglycerol

These are all important metabolic disorders in the postpartum period, during the transition period, which can increase the risk factors for Endometritis by 10 folds. So, it is having very big impact on the uterine defense mechanisms, as well as the incidence of Endometritis.

We have to concentrate on the Nutrition of the transition cow is very important because poor nutritional management of dairy cows during this transition period leads to metabolic disorders. The deficiency of micronutrients such as Selenium and Vitamin E can suppress immunity. So, the formulation of the appropriate diet during the transition and lactation period is important for reducing the incidence of metabolic disorders nutrition has to be concentrated during the pre-parturient period, parturient period as well as post parturient period because there will be a lot of metabolic changes in the animal, based on the parturient status of the animal.

The next very important one you have to deal with is the Handling of Parturient Disorders: It is always known that the uterus is bestowed with powerful systems of natural repair and recovery, provided if it is undisturbed. In the case of normal parturition, without handling the incidence of uterine infections, is very much reduced, so that shows that if there is any indiscriminate handling then that paves the way for the infection of the uterus, especially during dystopia we have seen it in the training also the normal Dystocia (we have seen it in the twinning also, the normal Dystocia). If it is a prolonged case, we have to handle more, in such conditions, there are a lot of chances of breaching the uterine environment and allowing the entry for the invasion of the pathogen that will lead to

endometriosis. One more reason is with handling the retention of the placenta, so nowadays we are telling that there is no need to handle match or manipulate intrauterine for removing the placenta. It is one of the reasons to prevent the unwanted invasion of uterine pathogens. so, you could see in field conditions sometimes they are trying a lot of weights some stones or blocks or whatever the thing to remove. These are all very indiscriminate practices that will lead to further consequences and sometimes for infertility or sterility, so to be very careful unnecessary manipulation either per vaginum or per rectum and indiscriminate dosing of drugs should be discouraged to facilitate the uterus to experience the natural process of repair and recovery leading to better postpartum reproductive functions. It is being proved actually. So, the best current advice for the prevention of uterine infection is to provide a better environment to optimize animal nutrition and management and to handle parturient disorders judiciously in order to increase the tolerance of the parturient and postpartum animals so that they are better able to limit the impact of uterine pathogens without developing the uterine disease.

We all know that there's a lot of multifactorial facts, a lot of factors, right from the LPS Lipopolysaccharides or the ROS, negative energy balance, everything put together makes the animal into an infertile condition in the postpartum period. So, the best preventive measures during the parturient period will prevent the occurrence of uterine infections definitely.

In a conclusion, we can say that the proper understanding of endometritis like the various Degrees of affection, the Causative factors for the infection, the Effect on fertility, Selection of appropriate therapy, and adopting preventive measures will reduce the incidence of endometritis and it will ensure the improvement in Fertility among the dairy cattle. So we have seen Endometritis in detail. About the types of Incidences, the Causative factors the diagnosis Therapeutic approach as well as the Preventive measures for controlling the Endometriosis condition.

I hope the lessons will be useful for you and you can apply them in the field conditions to contact us for any queries in the future.

Thank you all, see you again in some other sessions.

Thank you!

Download

PDF: Prevention of Endometritis

PART IV

WEEK 4: CYSTIC OVARIAN DEGENERATION

1 Introduction, Hormonal Regulation of Ovarian Activity, Predisposing Factors

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Transcript

Dear friends

Greetings to all!

First of all, I would like to introduce myself Dr. K Krishna Kumar Professor and Head, Department of Veterinary Gynaecology and Obstetrics, Madras Veterinary College, Chennai, Tamil Nadu, Veterinary and Animal Sciences University, Tamil Nadu, India. We are going to discuss about Cystic Ovarian Degeneration in cattle. The first lesson comprises of Introduction, Hormonal regulation of Ovarian activity, Predisposing factors, Signs and Symptoms of Cystic Ovarian Degeneration.

Introduction:

Cystic Ovarian Degeneration is a common, clinically recognized cause of infertility in dairy cattle. The incidence has been 6-19%. The huge incidence of Cystic Ovarian Degeneration is recognized as a serious cause of reproductive failure. Cystic Ovarian Degeneration is characterized by one or more large anovulatory follicles greater than 2.5 centimeters in diameter, present in one or both ovaries that persist for at least 10 days in the absence of the corpus luteum with

abnormal estrous behavior. Here the diameter of 2.5 centimeters, is meant for large size breeds like HF or Jersey like that, in our Indian breeds or in small breeds like Vechure, that may have 100KG body weight or in case of other indigenous breeds may have 250KG body weight, this 2.5-centimeter diameter is not applicable, hence another definition is required, that is one or more large follicles greater than 17 millimeters, that is 1.7 centimeter in diameter, failed to ovulate and subsequently do not regress and persist for at least 10 days, but maintain growth and steroidogenesis.

What is a Hormonal Regulation of Ovarian Activity? First, we have to know the basics of follicular development, Ovulation, and Endocrinological events that occur in a normal Physiology. First, the GnRH is secreted from the hypothalamus, which will act on the anterior pituitary that in turn release FSH and LH from the anterior pituitary. This FSH is highly essential for the growth of the follicle. This FSH will act on the follicle, so the follicle can get maturity. So, the matured and developing follicle can secrete a sufficient amount of estrogen, that estrogen is having a positive feedback effect on hypothalamus and pituitary, that in turn release of LH, that LH is highly essential for ovulation. This LH will act on the ovarian follicle. Matured follicle ovulation occurs. After ovulation, the corpus luteum formation occurs, this corpus luteum is going to secrete progesterone, this progesterone is highly essential for the negative feedback effect on FSH, then only further development of follicle may not occur, but this negative feedback is not applicable for LH because this LH is highly essential for the corpus luteum maintenance. This is the normal physiological Hormonal Regulation of Ovarian activity in cattle.

What are the Predisposing Factors for Cystic Ovarian Degeneration?

Most prevalent in high-producing dairy cows, especially which occurs 30 to 60 days after calving, mostly up to the first ovulation, 60% of the animals may have cystic ovarian degeneration condition, among this 40% of the animals may get recovery spontaneously, another 20% only may persist. The second one is it is common in

closely confined and stabled animals, normally the moment of the animal and exercise is highly essential for the normal reproductive management and maintenance of the animal. Here it is closed confinement and the stabled animals may not have sufficient exercise, that is lack of exercise is a common cause for the Endocrinological disturbance.

The next one is it occurs during winter than summer and fall months. It is clearly indicated that during the winter months, it is closely confined. In the summer months, it is freely movable. Intake of estrogen-containing forages is another cause of Cystic Ovarian Degeneration, because these forages may have a high quantity of estrogen, for example, Alfalfa, Zearalenone, Red Clover like plants.

Then Increased Stress at calving; that is also having negative energy balance.

Metabolic diseases, like calcium deficiency, Ketoses like diseases are predisposing this Cystic Ovarian Degeneration.

Excess negative energy balance.

Hereditary- especially Twinning is highly correlated with the Hereditary.

High protein diet intake.

Uterine infection any postpartum complications like dystocia, RFM, metritis, etc are acting as a predisposing factor for Cystic Ovarian Degeneration.

This picture (showing picture) demonstrates that how heat, stress, and any stress may act as predisposing factors. Here, heat stress, not only heat stress, any stress condition, especially in postpartum condition, animals feed intake will be reduced, and the respiratory rate will be increased, that in turn, negative energy balance occurs. This negative energy balance causes a reduction in GnRH and LH and FSH secretions and increases in CRH and ACTH secretions. This CRH and ACTH secretion may involve in an increase of Glucocorticoid secretion, Catecholamine, and cortisol secretion, which in turn reduction in estradiol secretions, so it will lead to a reduction in follicular dominance, reduction in estrous expression, reduction in Oocyte quality, and finally fertility will be affected.

Another cause is the Subnormal Luteal level of Progesterone which is observed in high-producing dairy cows, especially energy-compromised animals. So, any subnormal luteal levels lead to inadequate formation of LH receptors, in the presence of inadequate formation of LH receptors leads to decreased sensitivity to LH of the growing follicles, so that in turn creation of a persistent dominant follicle and cyst formation occurs. So once the creation of persistent follicle and cyst formation occurs, leads to arrest of the next to follicular waves before dominance and ovulation. Finally, end up in anovulation and anestrous.

So far, we discussed about Introduction, Hormonal Regulation of Ovarian Activity, predisposing factors, Signs and Symptoms, so the remaining parts will be discussed in the next class.

Thank you!

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PDF: Cystic Ovarian Degeneration

2 Ultrasound Image of Normal Ovary, Classification of COD, Pathogenesis

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Transcript

Greetings! to all.

Today we are in Lesson 2, the last class, we discussed the Introduction, Hormonal regulation of Ovarian activity, Predisposing factors, Signs, and Symptoms of cystic ovarian degeneration. In this class, the objective is a discussion of Ultrasonographic images of normal Ovarian Structures, Classification of cystic ovaries, Mechanism of cyst formation, Signs and symptoms of Cystic Ovarian Degeneration. This is the picture having Ultrasonographic images of normal ovarian structures, that is normal ovary during the time of estrous, it is always having dominant follicle even though the presence of regressing CL with numerous follicles, one follicle may be dominated during estrous that may be observed during the estrous, second one is the coherence of follicles, during any stage of the estrous cycle, multiple follicles, small follicles, may be observed in proestrus, it may be developing follicle with regressing CL and during the time of estrous, that is developed follicle or matured follicle with regressed corpus luteum and late time of estrous that

is corpus haemorrhagecam may be observed during this estrous cycle and after the formation of corpus luteum, that corpus luteum may be cavity corpus luteum or Non-cavity corpus luteum may be observed in either one of the ovaries.

Cavity corpus luteum sometimes contains fluids also, the central cavity comprises or there is an accumulation of fluids and the Non-cavity corpus luteum may or may not have any fluids. So the next one is Classification of cystic ovaries: How to classify? that is based on the number of cysts, the texture of the cyst, and then fluid accumulation present in the cyst. how long the cyst persists in the ovary, based on that, the cyst is classified into the Follicular cyst, Luteal cyst, and Cystic Corpora Lutea.

What is meant by a Follicular cyst? The follicle cyst is defined as one or more than one thin[1]walled anovulatory follicles and greater than 2.5 centimeters in diameter with a fluid-filled cavity, Persists for 10 or more than 10 days in the absence of a functional CL, and is accompanied by either nymphomania or frequent estrous.

In the case of Luteal cyst: it is defined as thick-walled, partially luteinized anovulatory follicles, more than 2.5. Centimeter in diameter, which persists for a prolonged period characterized by anoestrous.

In the case of Cystic Corpora Lutea: it is defined as non-pathological CL containing greater than 7-millimeter fluid filled the central cavity with a distinct ovulation papilla, which produces a sufficient concentration of progesterone, that is about 7 to 8 nanograms per ml (7-8 ng/ml) which is sufficient for maintaining pregnancy, Most of the researchers stated that 100 micrograms of progesterone P4, in the circulation, is sufficient to support the pregnancy. It is often slightly fluctuating inconsistency also, So based on that Causes and Persistence and Conception, it is Classified as the Follicular cyst, and Luteal cyst and are always considered Pathological and Cystic Carpora Lutia is considered as a Physiological cyst.

Suppose the animal is not able to conceive, what will happen in the Cystic Corpora Lutea, the regression of corpus luteum occurs

within a certain period of time and then cyclicity will be initiated, that's why it is called as Non-pathological cyst or Physiological cyst. Mechanism of cyst formation: In spite of much researchers demonstrated and done research in so many aspects, the exact mechanism of cystic ovarian degeneration is still unclear. However, the generally accepted mechanism is disruption of the Hypothalamo-Pituitary Gonadal axis, which comprises Intrinsic factors and Extrinsic factors. Mechanism of cyst formation- The first one is the Intrinsic factors; here the Hypothalamus secreting GnRH will act on the anterior pituitary and release FSH and LH, that FSH will act on the follicle, it is involved in the development of follicle, which in turn secretion of estrogen, that estrogen positive feedback is highly essential for the LH surge and then LH will act on the ovary and ovarian follicle and ovulation occur. This is a normal mechanism. But here fails to elicit or miss-timed or delayed GnRH or LH surge at the Hypothalamus pituitary level will cause the failure of ovulation by the dominant follicle. So what will happen after that one? The follicle can grow continuously, however, aberrant follicular growth also maybe there, so it will lead to alterations in receptor expression, and steroidogenesis activity will be affected, finally, it becomes a cyst. That is called Intrinsic factors involved in cystic formation.

The next one is Extrinsic factors: extrinsic factor, the similar way the Hypothalamus, GnRH, Anterior pituitary, FSH and LH, estrogen and everything will occur in normal animals, however low insulin level or insulin-like IGF1 growth factor one, concentration is very low means low proliferative follicular cells occurs. Follicle cell proliferation will be affected, which leads to low estradiol production. In the presence of low estradiol production, means that may not able to induce LH surge, so LH surge is also low, however, reduction of Gonadotropin-releasing hormone will be there, which will end up in retarded dominant follicle growth and pattern with estradiol production that will disrupt the Hypothalamo pituitary-adrenal axis, finally development of cystic follicles occurs.

The next one is the Signs and Symptoms of Cystic Ovarian

Degeneration. The first symptom observed in follicle or cyst is concerned Nymphomania, what is Nymphomaniac? It is a frequent, irregular, prolonged, or continuous sign of estrous absorbed in the affected animals, those animals are affected, means those animals are having often nervous, restlessness and bellow frequently, and then it is frequently attempting to ride other cows, but refused to stand to be riden and by other cows. Affected animals always have aggravated homosexual characteristics that are called Bullers. In the Bullers and in Nymphomania cows, the uterus and cervix are always larger in size edematous, and flaccid. Uterus and Cervix are concerned, at the time of examination, you can reveal that the presence of a large edematous and flaccid uterus, the cervical canal is always dilated and relaxed, permitting one finger you can insert or a pencil may be inserted inside the cervix. The endometrium is smooth, moist, semi-transparent, and edematous. Vagina Clitoris Vulva is always swollen in condition. That is called Nymphomania.

The next one is Sterility hump: affected animals that are chronically affected animals are having excess relaxation of the pelvic ligament lead to the tipping of pelvis and elevation of the tail head, In long-standing cases of Nymphomania tipping of the pelvis is very commonly observed, that is(showing picture) this is tipping of the pelvis area. This is the tipping of the pelvis area that is sacroiliac ligaments and all the ligaments are excessively relaxed and the tail head is elevated in nature, The ligaments, even after recovery, failed to regain their tone, and even after conception animal may be maintained with that excess relaxation of the pelvic ligament and sterility hump situation itself.

The tipping of the pelvis may result in an unsteady gait and predispose to injuries. More common these injuries are absorbed in aged animals and later stage of the pregnancy in more number of calved animals. The next one is Adrenal Virilism: commonly observed in chronic follicular cyst cases, those affected animals exhibit a muscular behavior and appearance. That is, those animals always have increased levels of 17 beta keto steroids in urine from the adrenal gland. And then the appearance of the animal is

masculinization of head and neck is also noticed that is called Steer like appearance. This is the region, neck region, and head region is having a masculine appearance. The animal looks like bull-like. It just looks like a Steer like appearance, that is bull[1]like appearance.

The next one is Mucometra or Hydrometra: What is meant by Micrometra? It means accumulation of mucus.

Hydrometra means an accumulation of watery-like contents, that is chronically affected cases often noticed with Mucometra, and Hydrometra. In these cases Hyperplasia of the mucosa is always noticed, Cystic dilatation of the endometrial glands is also noticed. Marked cystic dilatation develops a typical Swiss cheese appearance. This is the Swiss cheese appearance of the endometrium(showing picture).

Always in the affected cases, the uterus accumulated with 100-1000 CC of watery mucus. Affected animals may have affected the single horn or portion of the horn, if infection occurs may lead to Byometra also, that is an accumulation of pus inside the uterus.

So far we discussed Ultrasonographic images of normal ovarian structures, Classification of cystic ovaries, Mechanism of cyst formation that is Extrinsic factors and Intrinsic factors, Signs, and Symptoms of the cystic ovary, that is, Nymphomania, Sterility hump, Adrenal Virilism, Mucometra and Hydrometra, the remaining portion will be discussed in the next class.

Thank you!

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PDF: Cystic Ovarian Degeneration – Objectives

3 Luteal Cyst Signs

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Transcript

Greetings to all!

We are going to discuss in the next lesson 3 on Signs and symptoms of the luteal cyst, Diagnosis. Before that, in the last class, we dealt with Ultrasonographic images of normal ovarian structures, Classification of cystic ovaries, Mechanism of cyst formation due to the Extrinsic factors and Intrinsic factors, signs, and symptoms, Nymphomania, Sterility hump, Adrenal Virilism, Mucometra, and Hydrometra even formation of Pyometra. In this class, we are going to discuss the Signs and Symptoms of the luteal cyst and the Diagnosis of Cystic Ovarian degeneration. Signs and Symptoms of Luteal Cyst: that is affected animals are always having Prolonged anestrous periods and also Erratic changes in milk production. Surprisingly those animals may have higher milk production on that particular day, within a few days it may be reduced few liters also, so the fluctuation of milk production may be absorbed in those cases. Nervous tension is always absorbed. Disturbed with feeding and rumination, Progressive emaciation is noticed in those animals particularly in farm conditions. Either a Follicular cyst or Luteal cyst, one cow may be affected, all the cows may have a reduction in body weight due to the affected cow being

involved in that. That is always agitating all the animals, so the remaining animals may not be able to graze normally.

Diagnosis:

The diagnosis based on History and Clinical signs: The first one is Follicular cyst- those cases already we discussed Relaxation of vulva and perineum is absorbed, Nymphomania is commonly observed in the follicular cyst, The irregular estrous cycle that may be a short cycle or frequent estrous or long cycle may be absorbed in the follicular cyst, Sterility hump is noticed in follicular cysts, and also at the time of examination we can absorb Tougher, more tenacious, and opaque vaginal mucus with a mucopurulent appearance discharge, and these animals always have High milk yield, and Adrenal virilism is also observed in those cases. But in the case of Luteal cyst- always having Prolonged anestrous opposite to Follicular cyst, Follicular cyst that may have an irregular estrous cycle, here it may have prolonged anestrous period, and Erratic changes in milk production, Rough dry hair coat, Nervous tension, Disturbed feeding and rumination, and Progressive emaciation are the common Clinical Signs observed in the luteal system.

The diagnosis based on the Rectal Examination: First is Follicular cyst- External os of the cervix is usually larger in size and highly relaxed, The uterus is Turgid and doughy in consistency, Single or multiple follicles may be absorbed, the multiple follicles are more commonly observed than a single cyst, and Enlarged, thin-walled fluid filled follicular structures are observed, and Voluminous cervical discharge may be observed especially during examination it is confirmed, Chronic cases accumulation of mucus with the debris is also, this debris may be accumulated in the part of the cervix. Then in the case of Luteal cyst – rectal examination revealed It is a single cyst mostly most of the time it is a presence of a single cyst, The enlarged ovary is observed more than lemon size, Thick-walled structure, Flaccid uterine horns, and A closed cervix is always observed.

The diagnosis based on the Ultrasonographic Examination: In Follicular cyst The fluid-filled cavity that is the Anechoic area is

observed by ultrasonographic examination, The Diameter of the follicle is about >2.5 cm, The Follicular wall thickness is, this is (showing picture) the follicular wall thickness, which is about less than <3mmin diameter, and also Swiss cheese appearance is noticed in chronic follicular cyst cases. In the case of Luteal cystIt is always single in nature, and Enlarged ovaries observed, the size of the ovary is enlarged but at the time of examination the part of the cyst is palpable because the remaining cyst may be embedded in nature, and then A thick-walled structure is observed, here the thick-walled means that the follicular wall layer is always >3 mm in diameter, Always the uterine, uterus, and uterine horns are Flaccid in nature, The Cervix is always closed.

The diagnosis based on the Progesterone Concentration: In the Follicular cyst – The Serum Progesterone Concentration is less than one nanogram per ml (Serum P4< 1ng/ml) and Serum Estrogen concentration is approximate that range is about 10 to 13.3 picogram per ml (Serum E2:10-13.3 pg/ml) In the case of Luteal cyst[1]Serum Progesterone Concentration is more than one nanogram per ml (Serum P4 >1ng/ml) and Serum Estrogen Concentration always less than 10 picograms per ml (Serum E2< 10 pm/ml) is observed. Based on the Rectal examination, based on Ultrasonographic examination, based on Progesterone Concentration examination, we can find out whether it is a Follicular cyst or Luteal cyst.

The next one is Prognosis: As far as this is concerned, in most cases, recovery is not assured, until the cows are pregnant. More number of Treatments are required in some cases; Suppose the earlier diagnosis is made better Prognosis is there, and Minimum number of cysts are there in either one of the ovaries or both the ovaries better Prognosis, Single ovarian follicular cyst always better Prognosis, Hereditary causes better Prognosis may be there but some of the cases spontaneous recovery, If the presence of daughters may have this same condition it may be culled the Prognosis is very poor, the incidence of a daughter with cystic ovaries are highly poor, Severe cystic degeneration of endometrium and atrophy of the uterus in hydrometra and mucometra, the

Prognosis is always poor, Rare cases of much-cervix are also there these types of cases the pregnancy may not be able to maintain because the cervix is the gateway of the uterus, it may not be able to prevent the infection, so the Prognosis is poor.

So far we discussed Signs and Symptoms of Luteal cyst, Diagnosis comprises of History Clinical signs, Rectal examination, Ultrasound examination, Serum progesterone concentration, Prognosis. So remaining parts will be discussed in the next class.

Thank you!

Download

PDF: Signs and Symptoms of Luteal Cyst_Diagnosis

4 Diagnosis and Treatment

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Transcript

The next one is lesson 4.

In the 3rd lesson already, we discussed the Signs and Symptoms of the luteal cyst, Diagnosis, History and Clinical signs, Rectal examination, Ultrasound examination, Serum progesterone concentration, and Prognosis of the Cystic Ovarian Degeneration.

Here we are going to discuss Treatment and Diagnosis.

So as far as Treatment is concerned, in the olden days onwards, the manual rupture was followed. It is the earliest Treatment, just introduce your hand and place your fingers on the cyst and just pinch of the cyst and the rupture of the cyst we have to do, in that case, the single cyst is there it is possible to do, but repeated manual rupture is required for most of the times. Again, suppose the remnants of cystic cells are there, tissues are there, the accumulation of fluid again is possible, so repeated manual rupture is highly essential between 6-10 days interval. Most of the time the thick-walled follicles are there, we may not be able to do manual rupture, it is not possible also.

What are the Disadvantages? This means most of the time haemorrhage occurs, due to that haemorrhage, Ovaria-bursal adhesions are also possible, so it may have sterility also, it may

lead to sterility. So, it is not popularised, this treatment was not popularised.

Specific Treatment for Luteal Cyst: That is, the first one is PGF2 Alpha.

Two types of PGF2 Alpha are available in the market: One is Synthetic, another one is Natural. Natural: Dinoprost Tromethamine, for example, Lutalyse, 25 milligrams, intramuscularly we have to administer, that is the total dose not based on per KG body weight.

Synthetic: Cloprostenol Sodium, for example, Pragma is available in the market, the dose is about 500-750 micrograms intramuscularly.

Sometimes the cyst is not responsive for PGF2 Alpha, in those cases, you have to go for GnRH+PGF2 Alpha Treatment. On day 0, GnRH administration will be done, or LH administration may be adopted, GnRH receptor that is the example it comprises of Busarelin acetate 20 mcg is administered, LH means Chorulon that comprises of HCG (Human chorionic Gonadotropin, 3000IU (international units) may be administered.

After that, on Day 7 or 10, again Dinoprost Tromethamine may be administered, or Cloprostenol Sodium may be administered. If you go for GnRH, once the GnRH will act on the anterior pituitary, and then LH release will be there, complete luteinization of the follicle is possible or sometimes ovulation may also be possible. In the case of LH, it will act directly on the ovaries and directly act on the persistent follicle or cystic follicle and luteinization of the follicle occurs or ovulation occurs.

Nowadays, researchers are telling that GnRH is more effective than LH. And after Day 10, just go for insemination also, at 72 hours after PGF2 Alpha, that is Dinoprost Tromethamine, the second insemination will be adopted 96 hours post-administration of PGF2 Alpha.

The next one is the Specific Treatment for Follicular Cyst: In the case of the Follicular cyst, if you go for Dinoprost Tromethamine directly no effect will be there, hence you have to go for GnRH or

HCG on Day 0 and then on Days 7 and 10, Dinoprost Tromethamine is administered, then after administration of prostaglandin 72 hours and 96 hours of insemination may be carried out, that is called Fixed Timed Insemination, which will be carried out.

The next one is most of the bedding veterinarians, even experienced veterinarians were not able to differentiate that follicle or cyst and luteal cyst. So, in those cases, just go for this type of therapy, combined therapy, the general treatment for follicular cyst and luteal cyst. You have to go for GnRH and its analogues with PGF2 Alpha and its analogues. If you go for GnRH, after administration of GnRH, that will cause an immediate release of LH that LH will act on that follicle, that is a cyst, complete luteinization of cyst occurs or even sometimes ovulation of anovulatory follicles also occurs.

So finally, the cyst that is luteinized follicle or ovulated that is the formed corpus luteum is highly responsible for PGF2 Alpha. The progesterone secretion level, secretary ability are increased and it is highly responsive to PGF2 Alpha. The responsiveness of the Hypothalamus to the positive feedback effect of estrogen is also possible, so it leads to LH surge and normal ovulation may take place, animal return to normal ovarian cyclicity. So based on this, this treatment protocol is adopted Day 0, Busarelin acetate 20 mcg or HCG 3000 IU (International units) administered intramuscularly and Day 7 Cloprostenol Sodium 500 mcg is administered, at the time of administration, you can visualize the presence of follicle, that follicle after administration of Busarelin acetate, that follicle may get luteinization that is called completely luteinized follicle, in this(showing picture) follicle is not an ovulated one and then Day 9 again, Busarelin acetate is injected with Fixed Time Artificial insemination after 72 hours of Cloprostenol injection, Here lysis of CL or luteinized follicle occurs, new emergence of the follicle is observed here and then at the time of Day 9 injection of Busarelin acetate cause hastening of ovulation also that is the synchronization of ovulation with artificial insemination and then on Day 10, second

Fixed Time Artificial Insemination is done. On day 45 pregnancy Diagnosis is made with Ultrasound.

So far, we have discussed various Treatments like:

- Manual rupture like cystic follicles,
- Specific treatment for luteal cyst, follicular cyst, and
- GnRH and its analogues with PGF 2A and its analogues for the treatment of either Follicular cyst or Luteal cyst.

Next class, we will discuss the remaining Treatment portion and Prevention and Control.

Thank you!

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PDF: Treatment and Diagnosis

5 Treatment, Prevention and Summary

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Transcript

Dear friends

Greetings to all!

Today we are going to discuss lesson 5, before that up to the last class we have dealt with various Treatments like Manual rupture, Specific treatment for Luteal cyst, Follicular cyst, and general treatment for GnRH and its analogues with PGF2 Alpha and its analogues.

Today we are going to discuss the remaining Treatment available for Luteal cysts and Follicular cysts, Prevention and Control of Luteal cysts and Follicular cysts, and the Summary also.

In the case of Follicular cysts and Luteal cysts, these may be treated with Progesterone based equipment and Prostaglandin based protocols: On Day1, CIDR/PRID/P4, Progesterone sponges may be inserted per vaginally and this CIDR, PRID, and Progesterone contains Synthetic Progesterone, so after insertion, this Progesterone may be observed by the system, which causes suppression of Gonadotropin release; On Day8, administration of PGF2 Alpha should be done, which helps to Lysis of CL or complete formation of luteinized cysts;

On Day9, the sudden withdrawal of these Progesterone P4 devices

is to be done, which in turn releases a surge of Gonadotropin release, which helps in the emergence of new follicular waves. Progesterone based treatment protocols Day1 that is Day0, insertion of CIDR + Buserelin Acetate 20 microgram; On Day8 Cloprostenol 500 microgram; On Day9 withdrawal of CIDR to be done; On Day11 Fixed time Artificial Insemination along with Buserelin Acetate 10 microgram, at the time of initiation of treatment you have to give 20 micrograms on Day11 at the time of AI 10 microgram of Buserelin Acetate is sufficient; Day12 again Fixed time Artificial Insemination you can go for pregnancy diagnosis on Day45.

Progesterone alone may be given however administration of Buserelin Acetate along with the insertion of CIDR is having advantages one this Buserelin Acetate involved in the complete luteinization of follicle or ovulation, so it makes such the formation of corpus luteum and that corpus luteum is highly responsive to Prostaglandin injection, so most of the time all the animals coming to estrous, you can achieve more than 60% of conception rate also. Irresponsive Follicular cyst cases: mostly 60% of the cases can respond to the treatment of Hormonal therapy, remaining 20% – 30% that is delayed cases or chronic cases or not having response with the earlier treatment, those cases are treated with ultrasound-guided transvaginal follicular cyst aspiration technique, this is the follicular transvaginal probe(showing picture), that is an ultrasound-guided transvaginal probe with the needle, this should be inserted per vaginally with the help of parental examination, the follicles or the ovaries are guided with your fingers and the needle is inserted and the fluid is aspirated, this is the aspirated fluid(showing picture) it looks like yellow or amber-colored if bleeding occurs it looks pink in color.

This is a technique (showing pictures), it is an intact cystic follicle, so after the insertion of the needle, you can see/visualize the needle present here after respiration is a collapsed follicle is absorbed and then after collapsed follicle within 10 days, 9 to 12 days interval, whatever maybe you can observe the presence of any estrous signs, during that signs you can absorb or visualize the emergence of

new follicles present on the ovary, so during this time you can go for Artificial Insemination with Receptal injection 10 micrograms of Receptal is injected and then after 24 hours again repetition of AI should be done and then pregnancy diagnosis made at 45th-day.

Treatment response: here the incidents are concerned, authors have recorded various incidences, so an overall incidence of Follicular cyst and Luteal cyst is about 5-45%, always the Follicular cyst formation is higher than that of Luteal cyst formation and then here 14% of all cows develop a cyst in life, especially during the postpartum condition. In postpartum condition, 20% of the animals after cyst formation can recover spontaneously remaining 40% of the animal go for cyst and delayed ovulation or anovulation or cystic formation. Then postpartum calving to conception interval may be increased, that is recovery changes without treatment is possible in 30-70% of the animals which were recorded by so many researchers.

Treatment response with the Hormonal therapy is possible 60-70% in Follicular cysts, 70-80% in Luteal cysts, and treatment response with Follicular fluid aspiration technique this is only for non-responsive cases that are about 30% in Follicular cyst. So, Prevention and Control: the first one is the culling and selective breeding; this culling and selective breeding are adapted to farm conditions, every year 10% of the culling is highly essential, mostly aged animals and infertile animals, these types of chronically affected cystic ovarian conditions animals, maybe culled, then only the form will be viable. Rational use of Hormones; the hormones should be used rationally because it is like a swad, suppose if you use either a high dose of hormone or a lower dose of hormone, that will affect the reproductive cyclicity of the animal.

Implementation of synchronization technique during the postpartum period may help to improve the conception rate, not only improve the conception rate but also helps to prevent and control cystic ovarian degeneration.

Reducing the length of the postpartum negative energy balance period is highly essential because, in farm conditions, balanced

feeding is highly essential. Usually, most of the farms' scientific feeding schedule is followed, that is up to 5 liter of milk yield they used to give 2 kg or 2.5 kg of concentrate and in addition each 1 liter of milk you have to give half kg of concentrate. Suppose a 10 kg milk production animal is there means, you have to go for 4.5 kg of concentrate then only the negative energy balance will be prevented. and green fodder administration is highly essential, especially during summer 25 kg of Green fodder feeding is highly essential.

Maximizing dry matter intake and reducing protein content in diet especially during postpartum is highly essential to prevent and control the cystic ovarian degeneration condition. Minimizing the occurrence of metabolic diseases is also highly essential.

So far, we have discussed in 5 classes that are: Introduction, Hormonal regulation of ovarian activity, Predisposing factors, Signs and Symptoms, Ultrasonographic images of normal ovarian structures, Classification of cystic ovaries, Mechanism of cyst formation, Signs and Symptoms of cystic ovarian degeneration, Diagnosis and Prognosis, Treatment and Prevention.

Thank you!

Download

PDF: Treatment Continue – Prevention – Summary

PART V

WEEK 5: CONGENITAL AND ACQUIRED DEFECTS OF REPRODUCTIVE TRACT

1 Introduction, Congenital and Acquired Defects of Ovary and Oviduct

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://opentextbooks.colvee.org/managementinfertilitycattle/?p=151#oembed-1

Transcript

Dear Veterinarian and my Dear students.

My topic is Congenital and acquired defects of the reproductive tract causing infertility in bovines. So before going to the subject just, I would like to introduce myself, I am doctor T. Sarath and working as an Assistant Professor in the Department of Clinics, Madras Veterinary College, Chennai, and TANUVAS.

As you all know that the Infertility is a major problem in the field and every veterinarian is facing Infertility problems in cattle. So, this is one of the major causes which limits the profit of farmers in the field. So as far as causes are concerned, there are so many causes for infertility problems in bovines like Repeat breeding syndrome and Nutritional deficiencies and Anoestrous, so on. So that the Congenital and acquired defects of the reproductive tract causing infertility is a major one.

Introduction:

Congenital anomalies are mostly due to:

Hereditary defects, which may be due to single gene effects,

Certain genes adversely affect both cows and bulls whereas others are sex-limited in their effect.

Some of the Congenital anomalies include Ovarian agenesis,

Ovarian hypoplasia,

White heifer diseases,

Freemartin

Congenital lack of endometrial glands,

Uterus Didelphis,

Uterus unicornis,

Double cervix,

Persistence of Gartner's ducts

So, these all are Congenital anomalies, and

As far as Acquired lesions of reproductive tracts are concerned, these are all some of the acquired lesions, especially Ovaro Bursal adhesions, Ovarian tumour, Endometritis, Metritis, and Pyometra, Cervicitis, Tumour of the vagina, Cystic Ovarian Degeneration (COD) Perineal laceration, and Rectal fistula.

So, in a nutshell, these are all the Congenital and acquired lesions that will cause infertility in bovines. So, in the coming classes, we will discuss one by one. As far as this topic is concerned, I have divided the lessons into 5.

So, in the 1st Lesson: We are going to discuss

Congenital and acquired defects of the Ovary and Oviduct: In that, the Ovarian agenesis first one, so the condition of complete failure of development of gonads or lack of one or both gonads are otherwise called as Ovarian agenesis,

Ovarian agenesis means the ovaries, not at all present, probably it may be absent, so if at all there, it may be a functionless, very very small, or pea size ovary that cannot be able to produce ovum as well as the hormones.

This is probably an inherited autosomal dominant gene and these gonadless heifers or the animals appeared normal until breeding age. So, until breeding age, it is normal, it's not showing any symptoms. But if you see the clinically there won't be any presence of estrum behaviour, as well as the udder also not showing any

proper development and The genital tract of these heifers is also looking like juvenile and undeveloped. So, this is about Agenesis. and

The next one is Ovarian Hypoplasia:

Ovarian hypoplasia is a little more common than Ovarian agenesis, In these Ovaries, the Oocytes and follicles are virtually absent. It is also due to a single recessive autosomal gene with incomplete penetration. So, the affected ovaries may be partially or totally hypoplastic, which means it is not fully grown at all. Depending upon the severity of hypoplasia and whether the condition is unilateral or bilateral accordingly, infertility or sterility will result. In bilateral cases, the affected heifer is anestrous, so in bilateral hypoplasia, because the ovary is almost hypoplasia, it is not able to produce a sufficient amount of estradiol and further no other ovarian steroids, so that the animal is not able to show the estrous behavior. So, in cows, left-sided ovaries are most commonly affected with hypoplastic ovaries, so the incident is more on the left side, the reason we still learn is we don't know, and Diagnosis and only repeated rectal examination are necessary.

And in this image, you can see the right-side ovary is completely developed, I mean oval and round in shape, and if we see the left side, there is a hypoplastic ovary and it looks like a band of tissue and it's like an elongated in nature. So, in this image, you can see this area, so this is an elongated ovary, and it is a streak of tissue, so it is otherwise called a Streak Gonad. So definitely this ovary is a functional one and probably it is not going to produce any amount of hormones, so probably the animal may be an infertile one.

Ovarian Hypoplasia: So, the hypoplastic ovary undergoes incomplete development and part of the whole ovary lacks a normal number or complement of primordial follicles. If you see normal animals, 50,700 primordial follicles will be that it ranges from 6,800 to 1,00,000 in both the ovaries. But in affected heifers, one ovary was totally or partially hypoplastic and the primordial follicles are very few, especially it ranges from 19,000 to 23,000. So partially affected ovaries with bilateral hypoplasia, the primordial follicle number is very very less, so it is lesser than the 500. So, in the case

of totally hypoplastic ovaries with bilateral hypoplasia, no follicle is at all present. So definitely it will be a sterile animal.

So, in Unilateral hypoplasia, the tubular portion of the genital tracks developed normal, so in unilateral, one ovary is maybe normal, another one is hypoplasia ovary, so in that case, the tubular tract, especially the uterus, cervix, and vagina may be developed normally. However, in the case of bilateral total hypoplasia, the genital tracts remain infantile, very smaller, or underdeveloped.

These gonadless heifers appeared normal until breeding age, but no estrum and normal udder development also lagging. So, the genital tract of these heifers is almost juvenile and undeveloped, so estrum does not occur because both ovaries are probably hypoplastic, so it's not able to secrete a sufficient quantity of estradiol so, the animal remains as an anestrous animal there will be, so the estrous behavior will be absent. So, in bilateral total hypoplasia, the heifer is like a steer with long legs, a narrow pelvis, and a poorly developed udder with small teats and a firm uterus. So, these kinds of symptoms indicate probably the animal is in sterile in nature.

The next one is Oophoritis, so this is an acquired lesion, and it is a somewhat rare lesion of the ovary, Usually seen as the adventitious findings at postmortem examination. So, in tuberculous oophoritis, brucella-induced oophoritis, and ovarian abscessation in animals were reported that have had generalized pyaemia, so the pyaemia is the most common finding, so, the causes may be many, the Enucleation of the corpus luteum for ovarian abscesses is one of the reasons for the oophoritis.

So next condition is Neoplasia:

Neoplasia is also a rare lesion in the bovine ovary. The most commonly encountered tumours are Granulosa cell tumours and Fibromas. Most Granulosa cell tumours are reported from non-pregnant animals and in pregnant animals also. So, these tumours may produce ovarian steroids, the tumour that secret estrogen will result in persistent estrous behaviour. So, in longstanding cases, virilism may occur and the Progesterone or Androgen-secreting

tumours resulted in anoestrous, the animal will remain as an anestrous animal. The unaffected ovary-typically regressive and inactive because the tumour will produce a large quantity of ovarian steroids, so which may suppress the unaffected ovary, so it may be inactive and rigorous in nature.

The GCTs, especially the Granulosa Cell Tumours, are generally regarded as a brain tumour, and sometimes metastasis is also not common, so it is an uncommon one. Other occasional tumours are carcinomas, fibromas, thecomas, and sarcomas, and these are generally benign and often massive in size, very larger in size.

In this image, you can see Granulosa cell tumours, while rectal palpation, so it may look like a smooth surface and firm in rectal palpation. So, the presence of the corpus luteum on the contralateral ovary suggested that the tumour is endocrinology active, so the Granulosa cell tumours will produce mostly estradiol, and Another type is Large Granulosa cell tumours, so these types of tumours may have some lobulation in the affected area, bull-like behaviour or aggressive behaviors. The next one is a Parovarian Cyst: this is due to remnants of mesonephric ducts and that is sometimes present in the mesosalpinx of the cows. A tiny para ovarian cyst of a few millimeters in size and is common incidental findings at the time of slaughter, so it is not having any serious complications in the reproductive performance of the animal.

The next acquired lesions are Ovarian bursal disease: so, the ovarian bursal disease is one of the common problems which will end up with sterility or infertility, so what are all the things which cause ovarian bustle adhesions? In older days many veterinarians used to treat cystic ovarian degeneration by rectal palpation or manual enucleation. For example, if the animal is having a follicular cyst, so sometimes this manual rupture or the manual enucleation of the corpus luteum may result in bleeding in the ovarian region, so that will cause further inflammation and it will end up with the ovarian bursal adhesion or ovarian bursal disease. So other causes like injection or infusion of the irritant solution, for example, some of the reports say that the infusion of Lugol's Iodine solution is also

one of the causes for ovarian bursal diseases or *** manipulation or *** manipulation of the rectal examination. So, this is about Ovarian Bursal disease.

In the fallopian tube, there will be common findings of Hydrosalpinx and Pyosalpinx. So, the hydrosalpinx is Cyrus-like fluids that will be accumulated in the fallopian tubes, so this is called hydrosalpinx. Suppose if the puss is accumulated in the fallopian tubes, it means it is otherwise called pyosalpinx. So, in this image, you can see the enlarged fallopian tube same as the fluid in the fallopian tubes, so this will affect or occlude the lumen of the fallopian tube, so which will definitely affect the gamete transport.

So, the summary of the day one lectures we have discussed:

Congenital defects like Ovarian agenesis,

Ovarian hypoplasia,

Oophoritis

Ovarian Neoplasia,

Acquired lesions like Ovarian bursal disease, Hydro, and Pyosalpinx.

So, with these, acquired lesions we conclude the day 1 lecture.

Thank you very much!

Download

PDF: Congenital and Acquired Defects of Reproductive Tract Causing Infertility in Bovines

2 Congenital and Acquired Defects of Uterus

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Transcript

Dear Veterinarian,

Good day and in the last class we have discussed the sum of congenital anomalies. Today we are going to discuss Congenital and acquired lesions of the uterus.

Uterus Unicornis: What do you mean by Uterus unicornis? Segmental aplasia involving the uterine horns is not uncommon and if only one horn is involved, the condition is called a uterus unicornis. (showing picture) The left horn is fully developed and the right horn is not developed, so congenital absence of right horn and if you see the ovaries almost normal and both ovaries are present and functional. However, when ovulation occurs in the ovary, unaccompanied by the horn until coming to the estrum. So because the ovaries are normal, the ovaries will produce a sufficient amount of estradiol as well as progesterone, so because of the estradiol, the animal exhibit the estrous behaviour, and subsequently the animal remain as an anestrous animal, because the corpus luteum will be formed up, so it may not be luteinized, or it may not be regressed away, because the uterus is involved in

the synthesis of luteinizing, so which is the hormone responsible for the lysis of the corpus luteum. But in this case absence of one uterine are the reason for the poor secretion or poor synthesis of luteinizing, which is essential for the lysis of the corpus luteum. So, the animal that remains here is an anestrous animal. So even after a manual rupture or manual inoculation of corpus luteum resulted in the animal will come into the estrum and subsequently, the animal remains is an anestrous animal, due to the persistent presence of corpus luteum.

So, in this image also you can see the left one is absent, the left uterine horn is absent and the right one is fully developed, both the ovaries are normal and functional, so this is about Uterine unicornis. And the next one Uterus Didelphys: This is double Uterus, so the complete failure of fusion which resulted in the double cervix and each uterine horn connecting with the vagina by a separate cervical canal. There is often also a division of the cranial portion of the vagina, as this is also derived from the paramesonephric ducts. So, in this image, you can see a double cervix, so both the external horns are leading to a separate cervical canal which enters directly into the uterine horn. So, this is a typical case of uterus didelphys. So, these animals may conceive and if at all provided with insemination in the horn, which is ipsilateral to the ovulation, and it will carry the calves to term and it also gives the birth normally. Suppose if you do the artificial insemination in the contra uterine horn that means the ovulation is another side and you are doing insemination on another side, so resulted in the conception failure because the ovulation resulted in the transport of the gametes in other horn, but someone deposited in another horn. So definitely the animal will not get conceived.

Another anomaly is Congenital lack of endometrial glands: So congenital lack of endometrial gland in the bovine uterus has been absorbed in few heifers. These heifers exhibited a failure of the estrum and a retained or persistent corpus luteum apparently due to failure of the endometrium to produce the luteolytic factor or prostaglandin necessary for the involution of corpus luteum. So,

you all know that the endometrial glands are very important for the synthesis of luteinizing especially PGf2 Alpha, which is responsible for the lysis of the corpus luteum.

Manual removal of the corpus luteum also will result in the lysis of the corpus luteum. So, the animal will return into the estrum within 3-4 days of manual inoculation, however, in the next cycle, again corpus luteum will persist. So definitely the animal will be either infertile one or sterile one, this is due to genetic or congenital is still unknown. So, this is about a congenital lack of endometrial glands.

So next Inflammation of Uterus: Endometritis, Metritis, Perimetritis, Parametritis. Sclerotic metritis, and Chronic endometritis, there are some of the Inflammation of the uterus and you know what does mean by inflammation of endometritis? this is basically the lumen, layers, inner layer of the uterus is endometrium. Inflammation of the endometrial layer of the uterus is called Endometritis, Inflammation of the myometrium of the uterus that is the middle layer is called Metritis, Inflammation of serosal layer, the especially outer layer of the uterus is called perimetritis, Inflammation of adjacent structures to the uterus is called parametritis, Inflammation of all three layers of uterus and fibrosis is called sclerotic metritis, and chronic inflammation of endometrium is called chronic endometritis. So, these all are some of the Inflammation of the uterus.

So in the next slides, you can see some of the images; endometritis,perimetritis, and parametritis, you always will get confused about how would you diagnose clinically, so you have to do the rectal examination obviously and you have to palpate the uterus. All of you know how you can suspect endometritis, for example, if you do the rectal palpation, you can be able to see the thickening of the endometrium, especially in the bifurcation region. So along with the clinical signs, you can easily diagnose the endometritis because affected cows will show purulent discharge, so easily you can diagnose the endometritis. As far as perimetritis is concerned, it is somewhat difficult, but the entire uterus is inflamed and a little bit you can able to see the tonicity, adhesion may be absorbed in the

adjoining region also, and the clinical signs, the animal may not be conceived, so it may be repeated one and infertile one.

Parametritis: so definitely this is chronic inflammation, so adhesion may happen so the other adjoining structure, like ovarian bursal and peritoneum and other structures, will get inflamed, and probably adhesion may happen. So, this is about inflammation of uterine layers. So as far as Chronic endometritis is concerned and so you can see this image, because of chronic inflammation of the endometrium, there is an accumulation of secretions, so that will be continuously accumulated and that will be inspissated and it looks like hard mosses and sometimes very thick moss. So definitely such kinds of cases will not get conceived because the entire layer is get affected and probably the uterine function is getting lost due to the chronic inflammation and sometimes the chronic inflammation leads to the fibrosis of the entire uterus, so once the fibrosis happened and then definitely the animal will get not get conceived, so probably it may be sterile ones only treatment is slaughter, so you cannot save the animal.

So, the next condition is bilateral cystic endometrial hypoplasia: So, some animals may continuously be exposed to estrogen, especially pyro estrogen or estrogen implants, or some estrogen-like substances. So, you know the estrogen is normally a secreting natural hormone, so it continuously stimulates the endometrium so that the endometrium will become secrete in nature, sometimes the endometrial glands will get enlarged, and there will be some cystic[1]like structure formed. So, in this image, you can easily identify the presence of cystic endometrial hypoplasia, so definitely these kinds of animals also will not get conceived probably sterile in nature, so there is no treatment for such kind of cases.

The next thing is Hydrometra, Mucometra, and Pyometra.

So, in Hydrometra, you know the water-like fluid is accumulated in the uterus and it will look like a pregnant uterine horn, so how will you diagnose or how will you differentiate between the pregnancy and the hydrometra, mucometra, and pyometra. So, you know Hydrometra means accumulation of water-like fluid inside the

uterus, Mucometra means accumulation of mucus inside the uterus, and Pyometra means the accumulation of pus inside the uterus. Suppose if you cannot be able to identify these positive signs of pregnancy and you may assume that these may be the reasons and sometimes, we clinically also these conditions are evident. For example, In the case of Hydrometra, continuous water-like discharge maybe happen so this is one, In Mucometra continuous mucus-like discharge may be present, and In Pyometra also continues pus-like discharge may be there. So based on the clinical conditions you can correlate, and repeated examination also is necessary and re-examine the animal in 10 days intervals or 15 days intervals and you can identify these conditions. And if the animal is pregnant, definitely the foetus may enlarge, may grow, and further enlargement is that we will visualize and easily identify. So based on the ultrasonography findings, you can easily differentiate. So, this is about Hydrometra, Mucometra, and Pyometra.

The next condition is Uterine Tumours, so the sum of tumours is rare, but Leiomyomas, Fibromyomas, Fibromas, Lymphosarcomas, and Adenocarcinoma of the uterus, these tumours accounted for almost 77% of the total tumours. These are often the incidental findings at the time of slaughter, definitely sometimes you cannot be able to confirm through the rectal examination unless you don't have an ultrasound machine. suppose if you have ultrasound easily you can identify but in olden days it was very difficult to identify, so only at the time of slaughter you can encounter these kinds of tumours. So often large and they can potentially be mistaken for the presence of mummified foetus on palpation per rectum, so most of the veterinarians mistakenly think that it may be a mummified foetus, because the tumour looks like a hard and firm mass, the mummified foetus also looks like a hard doughy mass, because all the fluid gets reserves inside the uterus, so leaving only a hard mass or doughy mass. So occasionally these tumours are massive in size. Most of the time it is smaller in size, only occasionally it may be a massive one. The Adenocarcinoma present as a moderately enlarged, firm and constricted lesion of the uterine wall and have

a high rate of metastases, so only adenocarcinoma metastases one, Affected animals often present clinically as having a chronic wasting disease, so the animal may be wasting in nature, probably sterile, very weak in nature right. So, you can easily diagnose and quickly you can dispose of the animal because of wasting natural unproductive animals. This is about the uterine tumour, and

Uterine Adhesions: so, this is a common sequel of some of the obstacle's procedure as well this is the instruction. Uterine adhesions are the common sequel of perimetritis also the adhesion may be present as fibrinous state tags over the surface of the uterus, adhesion that involve the ovarian bursa, or adhesion to other pelvic/abdominal viscera. A similar lesion may follow uterine rupture or retention of the foetus after dystocia also. So, as I told you that after Caesarean most of the time, some cases will develop uterine adhesion also because of leaking of any discharge or uterine discharge may enter into the peritoneal cavity, so that will cause inflammation and subsequently it will end up the adhesion with adjacent structures like peritoneum and intestines and abdominal wall and other ovarian bursal structures. So, such lesions definitely are more frequently associated with sterility because adhesion will affect entire reproductive performance like the gamete transport, ovulation and the secretion of hormones and all, so it completely blocks the normal reproductive functions, definitely, the animal may remain as a sterile animal, so the treatment you know already.

So, the summary of the second-day lecture we have discussed, Uterus Unicornis, Uterus, Didelphys, Congenital lack of endometrial glands, Endometritis, Metritis, Perimetritis, Parametritis, and Sclerotic metritis, Chronic endometritis and Endometritis Hyperplasia, Hydrometra, Mucometra, and Pyometra, Uterine Tumours and some Uterine adhesions.

So, thank you!

PDF: Congenital and Acquired Anomalies of Uterus

3 Segmental Aplasia of Mullerian Duct

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Transcript

Dear Veterinarians,

Good day! and today we are going to discuss segmental aplasia of the Mullerian duct or paramesonephric duct. So how these failures will affect the entire reproductive system? We will discuss. In the last class, we have discussed some of the anomalies in the uterus, so in this class, we are going to discuss again the failure of fusion of the Mullerian duct which resulted in the segmental aplasia of the reproductive tract.

So, in that first one is Segmental Aplasia or Hypoplasia of the Uterus: So, these defects are mostly hereditary in origin, and some may be congenital. So how does it happen? Because the Mullerian Duct has to be fused at the time of embryonic development so that the entire tubular structure will be formed accordingly.If the tube or Mullerian duct or paramesonephric duct failed to fuse means, either one of the tubular tract or the part of the uterus or part of the vagina or part of the cervix may be absent. Segmental aplasia of the uterine tube is very rare. The most severely affected cattle with Segmental aplasia of the paramesonephric duct are sterile due to the bilateral nature of the defects. These may be characterized by hymenal constriction; absence of either the cranial part of the

vagina or the cervix or the uterine body, including the part of the horns, and cystic dilation of the uterine horns due to a narrow band of aplasia or a defect nearby the uterine body. More commonly just the apices of the uterine horns containing yellow, tan to dark reddish-brown mucus are present because these secretions may be accumulated inside and the secretion will be changed like these in due course of time. This cystic apical dilation may vary from the size of the horn to the approximate size of a four-month pregnant uterus containing a few ml of or gallon or even more thick gummy or inspissated mucus will be present in the uterus. so, you can very well see this image, the congenital absence of one uterine on resulting in the dilation of the cyst or the endometrial glands inside the developed uterine horn and finally end up with the accumulation of the mucus or inspissated mucus. so, this is about the Segmental aplasia of the uterus or hypoplasia of the uterus.

So next is Segmental aplasia of the Cervix with secondary Hydrometra: So, this is also a genetic or congenital anomaly of the cervix of the cattle not uncommon. Segmental aplasia may rarely occur, resulting in mucometra or a cystic enlargement of the cervix. A high incidence of developmental defects of the paramesonephric duct system is associated with infertility and sterility especially in cattle like Jersey and Shorthorn heifers. Besides segmental uterine aplasia, infertile heifers had sacculations that range from 1-4 centimeters in diameter and diverticulums ranging from 1-2 centimeters in deep, and dilation of the cervix due to the defects at the 3rd or 4th cervical rings. These defects are invariably filled with thick mucus and most affected heifers will result in a conception failure. These may often be diagnosed by rectal palpation of the cervix aided by a small metal probe such as a uterine catheter.

The next one is Segmental aplasia or Hypoplasia of the Vagina: The vagina is usually short and narrow in such condition or may have an enlarged or dilated caudal portion containing mucus or occasionally pus and submucous vaginal channels may be present. These cordlike structures are considered to be primitive vestiges of arrested development of the Mullerian ducts. They might be

vestiges of the primitive Wolffian ducts also. In rare instances, there is a marked arrest in development, aplasia, or hypoplasia of the vagina and Mullerian duct system characterized by a lack of normal vagina, cervix, or uterus.

White Heifer disease with imperforate hymen: So, this is a classical form of failure of Mullerian duct or paramesonephric duct. So segmental aplasia of the Mullerian or paramesonephric ducts and especially an imperforate hymen have been called "White heifer disease", so why was the name White heifer diseases coined? Because the defect is more correlated with white color, especially this condition is more commonly observed in Shorthorn cattle, the coat color also is white, so the name is White Heifer disease. So especially the Mullerian ducts fuse and develop in the bovine embryo at the time of embryo when the embryo is 5-15 centimeters in length or around day 35-120 days of age. So, during this embryonic stage, the Mullerian duct will fuse so that it will follow the regular development of the tubular structure, especially the entire reproductive tract. So White heifer disease was so named because it occurs most commonly in White heifers of the Shorthorn breed. As I told you, this condition is considered to be caused by a single, recessive, sex-limited gene with linkage to the gene for white color, so the condition called arrested development of the Mullerian ducts occurred in the Holstein herd also it's mainly due to the inbreeding of a valuable sire upon his own daughters. so, because of inbreeding the white heifer's disease also occurred in the Holstein herd.

So, you can see these images, the abnormality of the tubular structure because of cystic dilatation, the secretion will be getting accumulated, and it will stay, and it cannot be evacuated because of the hymenal constriction.

So Imperforate Hymen: the hymenal constriction also is common to these animals may be fertile or infertile, usually the latter, with prolonged intervals between estrus periods and repeated services per conception because conception cannot occur if ovulation takes place on the side of the abnormal horn and the normal involution

of the Corpus luteum CL may not occur due to prostaglandin deficiency caused by the missing uterine horn or arrested development of uterine horn.

The heredity nature of these defects of the tubular portion of the bovine genital tract and the danger of their spread by artificial insemination (AI) bulls are also noted. So, you can see this image, imperforate hymen, secretion will be accumulated in the uterus, and it results in the Mucometra or Mucocervix or Mucovagina also.

So, Summary of Day 3 lectures,

Segmental aplasia or Hypoplasia of Uterus,

Segmental aplasia of Cervix with secondary hydrometra,

Segmental aplasia or Hypoplasia of the Vagina, and

White heifer disease with an imperforate hymen.

So, with this third-day lecture, I conclude, and

Thank you very much.

Download

PDF: Segmental Aplasia of the Mullerian Duct or Paramesonephric Duct

4 Miscellaneous Hereditary Forms of Infertility

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Transcript

Dear Veterinarian,

Good day! and today we are going to discuss about the Miscellaneous Hereditary forms of Infertility in bovines, so in that, we are going to discuss about Freemartinism, this is one of the common congenital anomalies which is encountered in the majority of bovine breeds. So Freemartinism is a distinct form of intersexuality due to vascular anastomosis of the adjacent chorioallantoic sacs of heterozygous fetuses in twin pregnancies. The majority of female fetuses in male/female twin pregnancies are affected. So vascular anastomosis occurs as early as 30 days of gestation. So, occurs when the blastodermic vesicles from each developing zygote meet and fuse in the uterus about day 18-20 of pregnancy, so it is possible for single born freemartins to occur if there is a death of the male twin of a heterozygous pair after the time of vascular fusion with the another being carried to term.

This has been demonstrated as a cause of infertility in heifers with apparently normal external genitalia but with sex chromosome

chimerism that is a mixture of both female and male cells. The external genitalia freemartins may appear relatively normal. and

The clitoris is characteristically enlarged, and prominent and coarse hairs are present at the ventral commissure of the valva.

The internal genitalia is grossly abnormal.

The gonads are typically vestigial, although they undergo masculinization in mild cases.

So the structure is derived from the paramesonephric ducts are almost entirely absent or grossly hypoplastic.

In animals, with a significant degree of masculinization, the gonads resembled testes to the extent that their parenchyma contains recognizable tubules and interstitial tissue. The development of mesonephric (Wolffian)ducts is related to the degree of masculinization of the gonad. So, in extreme cases, well-developed epididymides, vas differentia, and Vesicular glands are also observed. More typically, the vestigial gonads of freemartins are devoid of oocytes and follicles and have parenchyma that consists largely of degenerating sex cords. You can absorb prominent clitoris and tuft of hair at the ventral commissure of the vulva, but these signs are not always reliable. and Freemartins can be identified on the basis of the length of the vagina and the absence of the cervix also So in the adult, the vagina is normally 30 centimeters in length compared with the 8-10 centimeter length of the vagina in Freemartin females. The rectal palpation will fail to identify the cervix because the cervix is absent in promoting freemartin females. So, in calves of the 1-4 weeks of age. the vagina is normally 13-15 centimeters in length as compared with 5-6 centimeters in a freemartin female calf. Diagnosis at this age can be made using Fincher's test, so this is one of the tests which is very useful under field conditions, so you can use a blunt probe or blunt rod, which should be inserted initially at an angle of 45 degrees below the horizontal for 5 centimeters and then angled downwards to avoid impinging on the hymen.

We have encountered so many twin births in our surgical ward and most of the time we used to test then and there, so especially

in twin foetus, in twin calf, one is male and another one is female means we usually observe the clinical signs like a tuft of the vulva, a tuft of vulval hair as soon as the prominent clitoris and simultaneously we use to do the Fincher's test and accordingly we used to advise the farmer also for these conditions so that we can prevent as well as we can avoid such kind of sterile animal in the field conditions. See very well in these pictures, especially a tuft of vulval hair in the freemartin heifer and the prominent clitoris, especially in bovine the clitoris is buried in nature, so it is not well developed as compared to other animals.

For example, if you take Mare, the clitoris is very well developed, but in the case of cattle and buffalo, the clitoris is poorly developed and buried in nature in the ventral commissure of the vulva, but in the case of freemartin heifer the clitoris is very well developed and very prominent and in the ventral commissure of the vulva, there are so much of hairs that is a tuft of vulval hair characteristic symptoms in freemartin female heifer.

So, in the dissociation section so you can see the vestiges of the female reproductive tract also and being very very small pea size to ovaries in the reproductive tract and so it is underdeveloped one and only a band of tissue is available instead of fully developed cervix and the uterus and subsequently the uterine horn.

So, Diagnosis: most accurate method of diagnosis is a demonstration of sex chromosome chimerism in cultured lymphocytes, you have to collect the blood cells and you have to culture the lymphocytes and you have to identify the sex chromosomes. So, there will be a mixture of both male and female cells so that itself indicates the sex chromosomes chimerism, so these indicate, or this confirm the freemartin cases.

So unfortunately, the distribution of male cell percentage in freemartin appears to be random also sometimes. Thus, animals with low male percentages in the blood are as common as those with high male percentages. Diagnosis by a polymerase chain reaction (PCR) for the presence of the Y chromosome in the blood cells is a confirmative diagnosis. Confirmation of freemartin also

and you can go for the culture of lymphocytes by identifying these X chromosomes and further you can use the PCR test for the confirmation of the freemartin also.

So, these are some of the diagnostic methods for the identification of the freemartin heifers. The next one is Intersexes or Hermaphrodites: So, Intersexes or Hermaphrodites occur most commonly in goats and pigs and less commonly in horses and dogs. It is occasionally present in sheep and cattle and is rare in the cat.

So, Intersexes are individuals in which the diagnosis of sex is confused because of congenital anatomical variations and abnormalities of the genital organs. Conditions in Intersexes include hermaphroditism, abnormalities of the accessory genital organs, gonadal dysgenesis, and freemartinism. Other forms of intersexuality are pseudohermaphroditism have been also reported, in rare cases, the XY sex reversal and true hermaphroditism were also reported. Intersexes or Hermaphrodites are commonly observed in naturally polled goats also. These intersexes are genetic females and although the polled condition is dominant the Hermaphroditic defect is recessive, sex-limited, and incompletely penetrant. Most of the Hermaphrodites are phenotypic females most with their ovotestes and may exhibit a projecting vulva and enlarged clitoris. Both ovary cells and as well as the testes the male cells, both cells are present, so it typically exhibits intersexuality.

The more the affected animal source masculine phenotypes in parents and also penile-like clitoris, hypospadias, and hypoplastic testes often in the inguinal region. Probably it may be retained, and it may not be a descendant one. Variable development or lack of development of the mesonephric and paramesonephric ducts are seen at the time of autopsy. The vagina in these Hermaphrodites is very hypoplastic and a small glass rod will only penetrate about an inch past the vulva.

So, the summary of these day 4 lectures, we have discussed:

Freemartinism, Theories of freemartins, Clinical Signs and how it will be Diagnosed and confirmed, and Intersexes or Hermaphrodites.

So, thank you very much!

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PDF: Miscellaneous Hereditary Forms of Infertility

5 Defects of Cervix, Vagina and External Genitalia

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Transcript

Dear Veterinarians, Good day!

At the last class we have discussed Freemartinism and intersexes and in this class today we are going to discuss about Defects of the Cervix, Vagina, and External Genitalia.

The first one is the Double External OS of Cervix: So, in this condition of uterus didelphys, failure of fusion is complete, so there is a double cervix, and each uterine horn connects with the vagina by a separate cervical canal already we have discussed.

Such animals may conceive, provided that the insemination takes place into the horn ipsilateral to the ovulation.

A more common lesion is partial duplication of the cervical canal, often with a single internal ostium and double external Ostia.

Affected animals conceive normally, but may show dystocia due to fetal limbs entering into both cervical canals; the expulsion is very difficult, so the dystocia maybe a sister one, then only we can able to relieve the foetus. Similar complications may arise in the cattle showing dorsoventral post cervical band also.

So, you can see this image and you can appreciate the presence of

a double external OS. The cervical canal assumes the configuration of the Y shape. So, this image (2nd image), you can see the true double cervix, so there is two separate cervical OS, and it is connected with the two separate uterine horn, so this is a true double cervix.

Double Pseudocervix and lack of cervical rings:

So other anomalies of the bovine cervix may include.

- a short cervix with one or more transverse rings missing,
- a lack of development of the cervical ring also

So, 1-3% of heifers may have a tortuous, sharply curved cervix or a hypertrophied transverse cervical ring so that artificial insemination. In collegial language, we usually called tilt cervix, so the cervix may be bent, in such cases we usually advise for natural cervix, so that the animal will get conceived and is followed by the normal parturition may also be taken place. In heifers with a short or underdeveloped cervix, infertility is often due to endometritis, because whatever secretion is there, it may be accumulated and because of the short cervix it may not be closed completely so the even microorganism from the vagina may travel through the cervix and that way the animal may develop endometritis.

So, the bent, tortuous, or stenotic cervix often is filled with thick mucus, and infertility may be associated with the inability of large numbers of spermatozoa to traverse the cervical canal, See the image of double psuedocervix So in that, you can absorb the presence of two external OS and connect with a normal external OS which will enter in the common tube to the uterine body, so during embryonic development, the anterior portion of the paramesonephric duct fails to fuse creating two pseudo externals cervical opening which connected by an antechamber with a normal external OS of the cervix and in this image(2nd image) you can see the absence of the transverse fold or cervical rings, so you know the cervical rings play a vital role for the close of this cervix, if it is getting absent, the particular affected case may be very well prone

for the development of endometritis. So next one is Cervicitis and Fibroma of Cervix: Cervicitis and Fibroma of Cervix, so these are some of the acquired lessons of the cervix. So, cervicitis is almost invariably accompanied by puerperal metritis and is common in cases of delayed involution of the uterus or power retention of the placenta or after birth.

Tumors of the cervix occur very occasionally, especially Leiomyomas and fibromas are the most common encountered tumors, from these pictures, so you can realize the presence of a tumor. And cervicitis, so you can see the enlarged cervix is a very common sequel after postpartum complications.

The next one is the Developmental defect of the Vagina: so, the Developmental defect of the Vagina is largely limited to the partially persistent or imperforate hymen. Obliteration of the caudal portion of the vagina. the persistence of the median wall of the paramesonephric duct in the cranial portion of the vagina.

Most commonly seen as a fleshy band, just caudal to the external os of the cervix or as a median septum extending 3 to 6 inches caudally resulting in a septate vagina associated with a double cervix. These defects are also probably hereditary in nature, although a few may be congenital.

The Developmental Defects-Infantile and hypoplastic vulva:

Rectovaginal constriction (RCV), Anovestibular Stenosis or an infantile, hypoplastic, and small vulva, all are some developmental defects of the vagina. The vulva was so small that during parturition, the animal will end up with dystocia, so in such condition, the episiotomy operation is necessary to relieve the dystocia, so the episiotomy involves the dissection of the vulva lips to expand the birth passage so that the delivery is easy, so in such cases, if we failed to do the episiotomy, we cannot able to do it or the episiotomy is failed means or we can go for Caesarion section, so it is necessary for such condition.

Copulation and defecation can occur, but rectal examinations in both bulls and cows and the parturition in affected cows cannot occur and they may end up with dystocia. A strong band of

connective tissue causes stenosis or constriction in the vulva, which encircles both the anus and vestibule.

So, another one is Abnormal Wolffian or Gartner's duct:

So Abnormalities of Gartner ducts, which are vestiges of the primitive Wolffian or mesonephric ducts in the embryo are rather common in the cow. It is not known whether these defects are hereditary. In hyperkeratosis due to chlorinated naphthalene poisoning- the Gartner's ducts become greatly enlarged due to the metaplastic changes in the epithelial lining of the ducts. Normally, these ducts are located beneath the mucosa of the floor of the vagina. They are two in number and are usually difficult to detect. The duct may develop multiple cysts along its course towards the cervix or form a long, sometimes rather coiled or cordlike range from 0.5-1.5 centimeter in diameter, which is distended with fluid.

In these images, you can very well recognize accumulated fluid and the duct is accumulated so you can very well recognize the Cystic Gartner's duct in the floor of the vagina.

Abnormal Wolffian or Gartner's ducts:

In rare instances, infection of the duct may occur with small abscesses forming along its course. The ducts may be incised freely, if necessary, to correct these abnormalities, but in most cases, they are ignored as they do not affect fertility. There may be present in rare cases a cul-de-sac extending forward 5-7 centimeters on either side of the urethral opening. This may be small and admit one finger or less, or it may be large admitting 3-4 fingers. This cul-de-sac represents an abnormally large terminus of Gartner's duct and may interfere with coitus.

So next one is Atresia of Vulva:

An abnormally small vulva has been described as a cause of dystocia in some breeds like Friesian and Jersey heifers. In such cases, episiotomy or Caesarean operation may be required to allow delivery. The defects have been seen to affect many of the progeny of particular Jersey bull indicating that it is likely to be hereditary in origin. It is occasionally seen in newborn calves in conjunction with atresia ani also.

Next is Urovagina:

Vaginal urine pooling or Eurovagina is the condition in which the urine accumulates in the anterior part of the vagina, so it leads to inflammation of both the cervix and vagina. It may extend into the uterus, causing endometritis also. Stretching of the suspensory apparatus of the genital tract as a result of several pregnancies may be a factor for causing urovagina. It is diagnosed at the time of post-calving examination.

Tumors of the Vagina and Vulva: Some of the tumors like Fibropapilloma in the vulva are common in cattle, Lymphosarcoma in the vagina are found occasionally, meta in or carbon and lymphosarcoma, occasionally found in the vagina. A squamous cell carcinoma of the vulva occurs in unpigmented areas of the skin of the cattle that are exposed to high levels of solar radiation. Warts and melanomas are occasionally seen on the perineum. So, you can see Fibropapilloma in the vulva and Lymphosarcoma in the vaginal area also.

So Acquired Lesion of the Vagina, Vestibule, and Vulva:

These acquired lesions during obstetrical operations especially laceration of the perineum, sometimes it may be impaired fertility especially in affected cows and Second-degree perineal lacerations sometimes lead to pneumovagina if the confirmation of the vulva is compromised. The surgical correction of such malconformation of the vulva is possible by performing Caslick's operation, in that you can correct the pneumovagina also. Dystocia and severe calving trauma- a third-degree perineal rupture at calving. So third-degree perennial rupture may be a more complicated one. So probably the animal will end up with the infertile one, so this is about acquired lesions of the vagina, vestibule, and vulva.

Summary of Day five lectures. We have discussed:

Double external os of the cervix, Double pseudo cervix and lack of cervical rings, Cervicitis and Fibroma of cervix, Developmental defects of the vagina, Developmental defects like infantile, hypoplastic vulva, Abnormal Wolffian or Gartner's ducts, Atresia of

the vulva, Urovagina, Tumours of the vagina and vulva, and Acquired lesions of the vagina, Vestibule, and Vulva.

So, in a nutshell we have discussed some of the congenital lesions of the ovary like ovarian hyperplasia, ovarian agenesis, and ovarobursal adhesions, oophoritis. So, this is about the overall view of the congenital or acquired lesions of the entire reproductive tract, which causes infertility in bovines.

So, thank you very much everyone and my dear veterinarian, and if you have any questions or queries you, please put a message on my number or to my mail ID.

Thank you once again, thank you very much!

PART VI

WEEK 6: ESTROUS SYNCHRONISATION

1 Introduction, Estrous Cycle, Endocrine Regulation and Hormonal Concentration

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Transcript

Greetings friends!

Today we are going to see about Estrous Synchronisation in bovines. It is one of the Important topics to tackle the various Infertility problems in the field condition also. You know Animal husbandry is the backbone of the rural economy, many farmers of rural India are depending on the animals' productivity for their daily income. Their daily bread and need are satisfied by the income of the Animal husbandry activity. Although we are the highest milk producers in the World, the milk yield of the cow in India is very less, so the number of cattle herds available in India is very high. And our Desi cows with because of the low genetic makeup for milk production, are low producers. But if you take Exotic and Crossbred cows, although they have early maturity and high milk yield, there is less disease resistance and more Infertility problems.

After the introduction of the Cross Breeding program in India, the Infertility problem has become very high. So, to tackle the Infertility problem many recent reproductive techniques are available, with the reproductive Biologist. So, one such technique is the Estrous

Synchronisation program. In this Topic, we are going to discuss various Principles, Advantages, Disadvantages, and the methods of the Estrous Synchronisation program in cows and buffaloes. If you adopt these techniques in the field condition, you can treat Infertility problems, at the same time even without Infertility problems in your Herd or at the Farm level, you can synchronize many animals at a time to bring into Estrous.

Now, we are going to see -What is the fixed expectation from an ideal cow? Ideal dairy cows should have aged at puberty -18 months, age at first calving about 30 months, conception date which would have 80%, AI per consumption is 1.3 to 1.7. The calving interval must be 12 to 18 months, it should not exceed beyond this level. Calving to conception must be 60 to 90 days. So, Ideal dairy cows should have all these parameters so that the farmer can get benefit out of rearing the cow.

The Delay in consumption rate, after 90 days of parturition, led to the loss of milk production up to 32% loss. Then medicine costs 13%. Calf loss 13%. The added Breeding cost comes around 11%. Then dairy animal maintenance comes to 31%. So, this is how the reproductive parameter, that is infertility causes, heavy economic laws to the farming community. To avoid this, there are very many techniques available in our hands, to manipulate the reproductive system to help the farmer to get more benefit from Animal husbandry.

Before entering into the Estrous Synchronisation program, as per this topic, we must know about the Bovine Estrous Cycle. It is the basic thing, then only you will understand what the Estrous Synchronisation program is. As you all know, what is the Estrous cycle? The estrous cycle is a Physiological functional rhythm of the reproductive system. It comprises Proestrous, Estrous, Metestrus, and Diestrus. That is, Proestrus and the Estrous together is called the Follicular phase. Metestrus and Diestrus are together called the Luteal phase. The cows and buffaloes are throughout the year cyclical, which is why they are called PolyEstrous.So, we can manipulate the Estrous cycle at any point in time in the year, that

is why we call this a Polyestrous Animal and the total length of the Estrous cycle varies from 18 to 21 days. We all know how the expressive stage of the Estrous Cycle is only Estrous. So, always Estrous is 18 to 24 hours duration in cows and buffaloes. Of course, in buffaloes, it is a little bit high. Estrus Duration is 8 hours high; it may go up to 30 hours. Although it is a silent Estrous animal, the duration of Estrous is a little bit higher in buffaloes than in cows. So, Estrous, if you take 18 to 24 hours i.e., one day, the manifestation of Estrous can be seen from outside. So, when we calculate the Estrous cycle we will take it as a day 0, but the duration of Estrous is one day. Then following Estrous is Metestrus, it is 3 days. So up to day 1, day 2, day 3, it will be in Metestrus and then Diestrous comes, it is about 12 to 14 days. Then Proestrus comes, it is 3 days. If you want to remember the duration of the Estrous stages (3-1-3-13, you can remember in this way, 3 days Proestrous – 1-day Estrous – 3 days Metestrus – 13 days Diestrous).

What are we studying these things for? There are two structures present in the Ovary, one is Follicle, and another is Corpus Luteum. When we are doing an Estrous Synchronisation program or Synchronisation of Ovulation program, we cannot manipulate the Follicle, but we can manipulate the Corpus Luteum, so what we are doing is, we are implementing the program during Diestrus. So, the best period we have is Diestrus that too I told you to start this is on day 0, Metestrus on day 3, then again in 3 days, you take in Diestrus. So, after six days you will have matured Corpus Luteum in the Ovary, either one of the Ovary will have matured Corpus Luteum. So, from day 6 to day 16, another 10 days are there. If you deduct 3 days from this,9 to 10 days are available for us, so from day 6 to day 16, there will be definitely the presence of matured Corpus Luteum in the Ovary. This Corpus Luteum you can manipulate.

You can cause regression of this Corpus Luteum. Once at the end of the Diestrous, what happens in normal animals, if there is no conception, no embryo in the uterus then the uterus will secrete PGF2 Alpha, the PGF2 Alpha will cause a rise up the Corpus Luteum during the Proestrus period, during this period Corpus Luteum

regresses. So complete regression takes place during these 3 days, then when it comes to Estrous, no Corpus Luteum and Progesterone level will be very less. Very less means, less than 0.5 nanograms per ml (< 0.5 ng/ml) in the Serum. So, at the end of Diestrus, if there is no conception in the uterus, the Corpus Luteum is regressed by the secretion of PGF2 Alpha from the uterus. So, the animal again comes to heat, this is what happens during the cyclicity. So you remember, the ovulation takes place in the case of cows and buffaloes 12 to 14 hours after the end of Estrum,i.e., during the Metestrus period it takes place. So, during Proestrous there is a Follicle development and during Estrous the Follicle reaches the maximum diameter and the animal shows Estrous signs, Ovulation takes place during a Metestrus and Corpus Luteum development occurs. The corpus luteum fully matures in the Diestrous. At the end of the Diestrous, if there is no pregnancy, the animal enters into the Proestrum. During Proestrum, Corpus Luteum regression takes place. This is the whole cyclicity, if you know this one, it will be very easy to understand the Synchronisation program.

So, this is a Chart form–Estrous Cycle=>Proestrous–>Estrous–>Metestrus–>Diestrous I already told these two i.e (Proestrous–>Estrous) is called a Follicular phase or Estrogenic phase, because as the follicle grows, it secretes Estrogen at its prime central nervous system and it primes the genitalia. So, the animal comes to Estrous.

Once Metestrum comes, Corpus Luteum is formed, Progesterone comes and these two i.e (Metestrus–>Diestrous) is completely dominated by Progesterone. That is why it is called a Progestational phase or Luteal phase. So, I told you Ovulation takes place during Metestrus. The cervix relaxes only during Estrum and Estrous is about 12 to 24 hours in the case of cows and buffaloes.

This is a Hormonal background, just for information. And before going to the Estrous Synchronisation program, this is very important for any field to know this. So, this is the Hypothalamus, Hypothalamus Secretes GnRH, it goes to the Anterior pituitary and the Anterior pituitary secretes FSH, this happens during Proestrus,

from FSH reaches the Ovary, all this hormone will be secreted in one place, and it will be acting on the target organ. so pituitary secrets, but the target organ is Ovary, within the ovary the target structure is the follicle. So, under the influence of FSH, the follicle starts growing. When the follicle matures, it produces Estrogen. Estrogen acts on the central nervous system and also it improves the blood supply to the genitalia. It causes increased secrete activity in the genitalia. When the Estrogen reaches the peak, it stimulates the Hypothalamus to increase LH secretion. Estrogen after reaching the peak level suppresses the FSH. But it favors the release of LH and also Inhibit secreted from the follicle, suppresses the release of FSH but favors the release of LH.So the Luteinizing hormone is secreted, this Luteinizing hormone again comes and acts on the follicle. The final maturation of the follicle is brought up by LH, LH not only causes Ovulation, but the final maturation of the follicle is also caused by LH. So, after the final maturation Ovulation takes place. Up to the final maturation of the follicle, it is in the Estrous stage. Once it enters into the Metestrus phase, Ovulation takes place. After the Ovulation again, LH is necessary for the maintenance of the Corpus Luteum, then in Luteinization, full Corpus Luteum is formed. I told you from Metestrum it enters into the Diestroum, fully. matured Corpus Luteum is formed, then the end of the Estrous cycle day 16 or 17, if there is no conceptus in the Uterus, PGF2 Alpha will be secreted from the uterus Luteinization occurs again, when the Corpus Luteum secretes progesterone, it will suppress the release of FSH and LH, so during the Diestrous stage animal should not come to heat except only a few animals that may come to heat during the mid-cycle also, that we will discuss later.

But strictly speaking, the progesterone suppresses the release of FSH and LH, so animals will not ovulate, and the animal will not come to heat during Diestrus. So, at the end of Diestrous, if there is no conception, Corpus Luteum gets sliced because of thePGF2 Alpha. Again, animals enter into the Proestrus. So, what I wanted to stress is, the Corpus luteum can be manipulated from Day 6 to 16 of the Estrous cycle. This is what I wanted to stress.

Going to see the Graphical Representation of the Hormonal secretion during the Estrous Cycle. As I told you, when there is no pregnancy, the progesterone level goes down because of the secretion of PGF2 Alpha from the Uterus. When the progesterone level goes down during the Proestrus, the progesterone block on the GnRH release is removed, so the FSH starts to secrete. When FSH starts to secrete, it acts on the follicle, so Estrogen starts to come to play a vital role. Estrogen acts on the central nervous system and the genital tract. Once Estrogen reaches the peak, it selectively suppresses the release of FSH but stimulates the release of LH from the anterior pituitary. When the LH reaches the peak during the Estrous Period, it causes the final maturation of the follicle. Again, it prefers the follicle for Ovulation. Ovulation takes place during the Metestrus period. Once Corpus Luteum is formed, again Progesterone level increases.

Dear friends!

So far, we have seen the Bovine Estrous cycle and the Endocrinology of Bovine Estrous Cycle and Hormone secretion during the Estrous Cycle that too particularly with Follicular Phase. We will stop here today. We will continue with the Basic principles and Approaches of Estrous Synchronisation in the next class.

Thank you.

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PDF: Introduction, Estrous Cycle, Endocrine Regulation and Hormonal Concentration

2 Principle, Benefit, Criteria for Controlled Breeding

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Transcript

Greetings to all!

Principle:

Today in this class we are going to see the basic principle and approaches of Estrous Synchronization in cows and buffalos. What is Estrous Synchronization- Bringing a group of animals into Estrous at a predetermined time by using an exogenous hormone. The very important thing is "predetermined time", suppose if you have a hundred non-pruned animals, all the hundred non-pruned animals can be brought into estrous at a particular time, this is called Estrous Synchronization. So, the very important thing is, the improper Estrous Reduction and Untimely Insemination is the major cause of infertility in the field. That is why if you know this – Estrous Synchronization, it is very easy to tackle the infertility problem. The basic principle is the regulation of the lifespan of the Corpus Luteum (CL). There are two approaches available for Estrous Synchronization-one is shortening the luteal phase by using PGF2 Alpha and another one is extending the luteal phase by using Progesterone. So, these are the two fundamental approaches

available for the Estrous Synchronization Program -one is shortening the luteal phase by using PGF2 Alpha or Prostaglandin F2 alpha another one is extending the luteal phase by using Progestational preparations. Then if you take the Estrous Synchronization protocols 1) Prostaglandin based protocol 2) Progesterone based protocol and 3) GnRH based protocol. So, by using these three protocols we can manipulate the Estrous cycle of cows and buffalos to achieve a high consumption rate.

Benefit:

What are the benefits of Estrous Synchronization?

Advantages:

If you know the uses, you can use the Technology very well. So fundamentally, it is used for better control of calving intervals. You can reduce the consumption to calving interval, thereby you can produce a calf per year with the use of this important technology. So, you can reduce the calving interval, so the inter calving period can be reduced. Once if you synchronize the Estrous and do on many numbers of AIs similarly, a group of females will calve simultaneously. All the animals' calves in a particular period, so Maintenance of cows and Vaccination program, Management procedures are very easy by using this technology. It reduces the time required for Estrous reduction. When you do an Estrous Synchronization program, it is otherwise called Controlled Breeding or Fixed Time Breeding or Programmed Breeding. So, you can program the Breeding. So thereby you can avoid the labor involved in the Estrous Reduction program. And many times, in villages animals will be in a particular site and the owner will be having his home two kilometers or three kilometers away from the animal shed in the Indian scenario, so they may not detect the Estrous properly. They are not with the animals during the night hours, most of the animals are coming to heat during the cooler part of the day, that is during night hours and early morning. So, since owners are staying away from the animal shed, they cannot detect the Estrous that may lead to infertility problems. So, such types of problems can be avoided by the Estrous Synchronization Program. Then you

can do Estrous detection with Timed Artificial Insemination, which will save the time required for Estrous Reduction. From which you can increase the production. Particularly, suppose in the month of summer, there will be a reduction in the milk yield from many animals. But if you want to have more calving peak milk yield at that time, you can program breeding based on this Estrous Synchronization program. So, since we are reducing the consumption to calving interval and in the calving interval number of cows produced per cow in its lifetime can be increased with this important technology. Then, Infertility- Estrous Synchronization is not only a management tool but also it is helpful to Alleviate or Treat or Manage the infertility problems under field condition. So, these are all the benefits of the Estrous Synchronization program in cows and buffalos.

Criteria:

For Controlled Breeding or Fixed Time Breeding or Programmed Breeding or Estrous Synchronization or Estrous Induction. You must know the difference between Estrous Induction and what is Estrous Synchronization. In India, particularly in Tamil Nadu and other places, owners owning one or two cows are more than the Farming system. The Farming system is not common in the Southern part of India, so owners will be owning one or two animals. You can't do synchronization in one animal or two animals. If you induce Estrous in many animals or hundreds of animals, it is called Estrous Synchronization. If you induce Estrous in a single animal, it is called the Estrous Induction program. So, the Estrous Induction and Synchronization program requires all these requirements.

Animal requirement:

You cannot do this program on a sick, debilitated, and weak animal. Even if you do this program, you cannot achieve the success of fertility, so animals must be disease-free animals. Adequate nutrition of the body condition score must be three and four. It should not be too lean; it should not be too fat. So, animals must be in good body condition with adequate nutrition and the adequate postpartum interval. The animal should have at least passed 45 to

60 days and should not do 20 days of calving. Next to Normal Non-pregnant Reproductive Tract: It should not have Adolescence, it should not have Infections, it should not have hydrosalpinx mucosulphings, etc. So, the normal genital tract must be there, and the animal must be cycling. In case of when you use prostaglandin-based protocol animals must be cycling. So, all these are Important Criteria.

Management requirement:

Proper timing should be maintained. The proper schedule of the Estrous Synchronization program should be correctly followed. Good quality Semen must be (very very important, I am always stressing this one) used. When you do Estrous Synchronization program in hundreds of animals or infertile animals, the Semen must be of a good quality You should do AI (Artificial Insemination) perfectly, good AI techniques must be followed otherwise the program will be a failure. When you use the Prostaglandin-based protocol, it will act only in the functional CL, that is it will act on days 6 to 16 of the cycle only. So, always you should have Matured Corpus Luteum on the Ovary, either the Right Ovary or Left Ovary should have Matured Corpus Luteum. Developing CL or Regressing CL will not respond to PGF2 Alpha. So, this is what we wanted to say. When there is a Developing CL from day zero to six that is three days of Metestrum and three days of Diestrum there is a Developing CL, that CL will not respond to PGF2 Alpha. Even if you give PGF2 alpha CL will not Regress instead it will grow. And what is Regressing CL, at the end of Diestrum, when it enters into the proestrus stage it will be a Regressing CL-that CL also will not respond to. So, during proestrus or Estrous and Metetrous and early Diestrous, you should not use PGF2 Alpha. There is no harm if you use PGF2 Alpha, but it will not produce the proper effect. So, one thing you should remember regressing Cl that is during proestrus or developing CL will not respond to PGF2 Alpha therapy. Only matured CL will respond to PGF2 Alpha therapy. When matured CL will be there from day 6 to 16th day of the Estrous cycle, the

matured corpus luteum will be there, so you can use PGF2 alpha during this time to synchronize or to induce Estrous.

Examples:

These are some of the examples as a Field Veterinarian what are all the Commercial Preparations available, these are all Synthetic Prostaglandins:

Vetmate is available

Clostenolis available

Pragma is available

PGF2 Alpha available

This dose of each ml contains 250 micrograms, so 2ml we will give Intramuscularly, 500 micrograms synthetic prostaglandin will be administered. Whereas natural prostaglandin, 25 milligrams will be administered Intramuscularly. It is only the total dose, not per kg, it is only the total dose. Remember 500 micrograms total doses intramuscularly synthetic prostaglandins like Pragma, Clostenol, or Vetmate or Estrumate. If it is natural, prostaglandin utilizes 25 milligram 5ml Intramuscularly.

Dear friends, so far, we have seen about

The Principles of Estrous Synchronization.

Protocols of Estrous Synchronization.

Benefits, Animal requirement and Management requirement

Following the Prostaglandin method of Estrous Synchronization

To understand these procedures, we will continue the class about the Follicular wave pattern in cattle.

Thank You!

Download

PDF: Principle, Benefit, Criteria for Controlled Breeding

3 Follicular Wave Pattern, Synchronization using Prostaglandin

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Transcript

Dear Field Practitioners!

Actually, I used to enjoy taking a Follicular wave pattern in cattle because this is fundamental to achieve success and manipulating the Estrous cycle in Bovines. We will continue with the Follicular wave pattern today. You know Follicular development is a continuous process. It occurs even during Anestrous that too is true Anestrous even during pregnancy. The only thing is that a Corpus Luteum formation is prevented. So, there are various types of Follicular wave patterns. The animal may have two-wave or three-wave or four-wave Follicular wave patterns. Sometimes four waves also. Depending upon the number of waves the Estrous cycle length also varies. Those animals which are having two-wave will have a lesser Estrous cycle length and those animals which have three-wave Follicular waves will have more Estrous cycle length than the two wave animals.

So, what is a Follicular wave? The follicular wave consists of, Recruitment, Selection, Dominance, and Atresia or Ovulation. These are the words.

What is Recruitment? Suppose! Today the animal is coming to heat, tomorrow it is going to Ovulate. So, at the time of Ovulation, on too many animals, many numbers Follicles start to grow. So, this is called Recruitment.

Among these many numbers of follicles, few follicles will be selected, they will be growing. This process is called a Selection.

Among the selected follicles, one follicle will reach a large diameter, which is called Dominance. This follicle will dominate, and it will suppress the growth of subordinate follicles. So, in Recruitment more follicles will be recruited, and the few follicles will be selected in Selection, among the few follicles, one follicle will be reaching the highest diameter. It is called the Dominant follicle. This Dominant follicle will suppress the growth of subordinate follicles. Then it will be growing and reaching the maximum size. But since Progesterone is secreted, no LH surge occurs. So, this will not Ovulate, and it will undergo Atresia. If it is a second wave, the progesterone level goes down GnRH will be released, then. FSH, LH will come, then LH will cause Ovulation, ovulating follicle. So, what I want to stress is Dominant follicle of the last wave is the ovulating follicle. Suppose if two waves are there, the first wave dominant follicle undergoes Atresia, so the Dominant follicle of the second wave undergoes Ovulation. If there are three waves in a cycle, the dominant follicle of the first wave and dominant follicle of the second wave undergoes Atresia. The dominant follicle of the third wave is the Ovulating Follicle. This is how it is happening.

So, for understanding the Estrous Synchronisation program, you must know the Follicular wave pattern also. I already told you this is the two-wave Follicle. You look at the dominant follicle of the first wave undergoes Atresia, the dominant follicle of the second wave is going for Ovulation, because, in a cyclical animal, the progesterone level goes down, then animals start to secrete GnRH and LH that results in Ovulation. This is the second wave. When you do induction of ovulation, if we give PGF2 Alpha on day 7, then this dominant follicle of the first wave will be the ovulating follicle. When you give this on day 6, PGF2 Alpha, the dominant follicle of the first

wave will be the Ovulating follicle. You are not allowing the animal to undergo a second follicular wave pattern. Again, this is the induction of Ovulation with the first wave. So, with the first wave, instead of undergoing Atresia, you can make this follicle Ovulate with the Estrous induction program.

So, now we will go into detail about how with the PGF2 Alpha we are doing the Estrous Synchronisation program. I already told you there are two types of Prostaglandins available. One is Natural Prostaglandin, the other is Synthetic Prostaglandin. Both Synthetic Prostaglandin and Natural prostaglandin can be used for Estrus Synchronization. The Natural Prostaglandin is available as Lutalyse. It is available in 5 ml and 10 ml vials, each ml contains 5 milligrams per ml. Whereas Synthetic Prostaglandin is available in the form of Pragma, Clostenol, Repregna, Pragma, Clostenol, Repregna, Entremets etc. So, these are available as 2 ml vials.

There are 3 programs available with PGF2 Alpha.

Programme A -If there are groups of animals or a single animal, whatever it is in the field condition. You palpate the ovary. If there is a presence of Corpus Luteum in any of the ovaries, inject PGF2 Alpha and do AI at 72 and 96 hours. I told when you are doing this, the animal must have matured CL. The matured CL is very important to respond. This is a program A. (Repetition)Animal is brought to you for examination, you are examining the ovary. Matured corpus luteum is present in the ovary and you are injecting the PGF2 Alpha and do AI without Estrous deduction, you can do AI 72 and 96 hours.

Program B- There is no palpation of the ovary. And inject PGF2 Alpha into all the animals. Suppose 20 animals are there is a camp, inject all the animals PGF2 Alpha. Either Natural prostaglandin or Synthetic prostaglandin, whatever you have, you inject the dose I told synthetic prostaglandin as 500 micrograms Intramuscularly, Natural prostaglandin 25 milligrams Intramuscularly. Then deduct heat and breed, that is 72 and 96 hours you can breed. Animals that have not expressed. Estrous sign, so after 11 days give PGF2 Alpha. Those animals which have come to heat, you breed or give

PGF2 Alpha. Those animals which have not come to heat, give PGF2 Alpha, then breed animals 72 and 96 hours. (Repetition) Program B in which, there is no palpation of the Ovary, you need not palpate the ovary, suppose 20 animals are coming for you in the camp or you identified 20 animals as infertile, then to all the 20 animals give PGF2 Alpha, either Natural prostaglandin or Synthetic prostaglandin. If it is Natural prostaglandin give 25 milligrams, Synthetic prostaglandin 500 micrograms Intramuscularly. Deduct heat, suppose out of 20 animals, 12 animals have come to eat, breed those animals, or do AI at 72 and 96 hours. Those animals which have not come to heat, give injection PGF2 Alpha 11 days, 11 days after the first injection, then breed the animal at 72 or 96 hours.

Programme C- There was no palpation of the ovary. Give PGF2 Alpha to all the animals, repeat PGF2 Alpha after 11days. This is a typical double injection schedule. Then after giving PGF2 Alpha, do AI at 72 or 96 hours. Second PGF2 Alpha – After the first PGF2 Alpha, you need not inseminate, after the entire second PGF2 Alpha you can inseminate all the animals. This is the PGF2 Alpha program.

Dear friends!

So far, we have discussed two-wave follicular wave patterns, three-wave follicular wave patterns in cows and buffaloes, followed by the prostaglandin use in the Estrous Synchronisation program. I think you would have understood the Estrous Synchronisation technique with PGF2 Alpha thoroughly. We will move to the next class, that is with Estrous Synchronisation by using Progestogens.

Thank you!

PDF: Follicular Wave Pattern, Synchronization Using Prostaglandin

4 Synchronization Using Progesterone and Advantages

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Transcript

Greetings friends!

Today we are going to see about the Progestogens in the Estrous Synchronization program. We are going to see about all the types of progesterone used to synchronize Estrous and how the CIDR is used in Estrous Synchronization, followed by how the TRIU-B is used in the Estrous synchronization program. The advantage with this progesterone-based protocol is that, whether the animal is cyclical or non-cyclic the Estrous synchronization can be done. For prostaglandin to be used in the Estrous synchronization program, animals must be cyclical, but when you use progesterone or progesterone, the animal may be in true anestrous, both ovaries are smooth, or animals may be cyclical. So, in these two categories, progesterone can be used to synchronize the estrous, which is the advantage with the progesterone compounds. What progesterone is doing- It is lengthening the Estrous cycle, that is it is lengthening or extending the luteal phase. Normally when the progesterone level goes down at the end of the diestrous, the animal comes to proestrus and it comes to estrous, but instead of going to the

proestrus we are extending the luteal phase with the exogenous progesterone, so once the progesterone is withdrawn, exogenous progesterone is withdrawn suddenly, there is a copious amount of secretion of GnRH from Hypothalamus, which in turn stimulates the anterior pituitary to release the FSH. The FSH will act on the Ovary to cause follicular development and again it will start secreting estrogen, which will stimulate LH surge, which will cause Ovulation. This is what happens with the exogenous progesterone administration in the Estrous synchronization program. I told you to know when the progesterone is withdrawn, there is follicular growth, Estrous and ovulation occur. This is the basic fundamental principle involved in the synchronization of the Estrum with the progesterone compounds. Progesterone is available in the form of Injection, it can be given orally, oral preparations are available. It was available in the form of Implant, now it is not available, and it is available as a pessary, Intravaginal pessaries example CIDR (Control Internal Drug Release Device) or TRIU-B. So, all these are Intravaginal treatments or Intravaginal sponges also available. So various preparations are available for our convenience. When the progesterone is used, how it works in the animal. You look at this is the Estrous cycle, normal Estrous cycle, the animal comes to Estrous, Ovulation occurs, this is the ovulation point. You see this is a follicular wave pattern, first wave follicle, second wave follicle then the third wave follicle, it goes like that. So, if you see the progesterone is released from the corpus luteum and it reaches and it reaches and regresses, you look at this in the normal, even when you are doing a synchronization program. When you insert the progesterone, insert here, progesterone containing product here intravaginally or orally or injectable form. It will extend the diestrous phase. Once it is withdrawn, if you remove this exogenous progesterone source, animals come to heat. So, this is what happens during the Estrous synchronization program with Progesterone.

Advantage of using progesterone: Corpus Luteum need not be identified here, the only thing is you must confirm the animal is non-pregnant. All the animals can be administered at the same time

when you use the progesterone all the animals can be inserted with CIDR, even if the animal is not cycling, I told already true anestrous animals also can be synchronized with progesterone. All other conditions with the prostaglandins and also applicable to progesterone for success. What all the animals" requirements or management requirements, everything is also held good for progestational compounds. So, the greatest advantage of progestational commodities is, even they can be used in non-cyclical animals or true anestrous animals. So, to induce Estrous in a true anestrous animal, this is the drug of choice available in our hand. So, progesterone injection, how to administer progesterone injection, 50-milligram daily injection or 500 milligrams every 10 days interval, 2 injections at an interval of 10 days. In India we don't have aqueous preparation of progesterone, so we cannot use aqueous preparation here, so 500 milligrams inject Intramuscularly, 10 days later you inject another 500 milligrams, so within 5 to 7 days animal will come to heat, then you can breed. It is the cheapest treatment; the cost is very low that precision of the Estrous may not be there with injection form. Some animals will come to heat on the third day of the second injection, some animals will express Estroys signs after the first injection itself after three to four days of the first injection. Few animals will inject will come to heat, five days after the second injection like that the close synchrony will not be there with the injection form of progesterone. But injections are available in the name of P[1]Depot, Duraprogen, and Prolutil. So, all these are available in the market you can use them for the Estrous Synchronization program in cows.

This is about oral progesterone, it is available as a MAP, CAP, and MGA.

MAP means — 6 methyl- 17 acetoxy progesterone, 500 mg daily it is given in the food orally for 21 days.

CAP means — 6 chloro- 6 dihydro-17 acetoxy progesterone, 100 mg/day for about the whole length of the Estrous cycle you have to give.

Then MGA means — Melengesterol Acetate, another oral form of progestogens, it is given in the feed for 9 days.

However, there are two types of therapies with progestogens, if you administer orally, all animals will not take an equal dose and a prolonged administration like this long-term projection therapy will cause a defect in the sperm transport, it will affect the sperm transport, it will affect the cleavage process. So, it will affect the consumption rate. So, to avoid this type of continuous therapy, long-term progesterone therapy, we have to use short-term progesterone therapy, but we are not using oral progesterone therapy nowadays in our country. The advantage of oral progesterone therapy is that it is simple to Implement, Inexpensive and Heifers do not have to be handled. Although these advantages are there, long-term progesterone therapy will affect the sperm transport, cleavage process, and consumption rate. So next very important drug is this, Controlled Internal Drug Releasing Device (CIDR), it contains 1.38 gram of progesterone, natural progesterone, it is inserted in the vagina and I told you when it is alone kept in the vagina it should be kept for a longer period 13 to 15 days, but it is combined with PGF2 Alpha or GnRH, you can reduce the length of treatment in CIDR therapy. I will tell you later. It is kept in the vagina for 9 days and 8th day we have to give PGF2 Alpha and on the 9th day, you remove it, On the 11th day we can go for AI that is 48 hours after the CIDR removal, and 72 hours after the CIDR removal you can do AI. When you use prostaglandin alone, you are doing AI at 72 and 96 hours. When you use a CIDR, we are doing AI at 48 hours to 72 hours after the withdrawal. First, you have to do a Rectal examination, the animal must be non-pregnant, and you can differentiate whether the animal is cyclical or non-cyclical also because the cyclical animal will have more consumption rate than anestrous animals. Next is the CIDR, which contains natural process progesterone 1.38 gram, and this is the applicator provided with the CIDR.

Next is we have to insert like this the T-shaped end should be folded together, it should be inserted inside the applicator, this

tail portion will be hanging then after application of CIDR into the applicator, you sterilize the CIDR with poured on Iodine, it not only sterilizes or cleans the CIDR applicator but also it will prevent the infections. This is the protruding portion of this CIDR, this is the tail portion of the CIDR, this is the applicator with CIDR. Then we have to restrain the animal properly, mostly we will be doing in the rural part, so the strong tree can be used to restrain the animal, wash the external genitalia with water. Then wipe the external genitalia very nicely, then evert the vulvar lips, it is inserted into the vagina like this then one single jerk it should be pushed and suddenly you have to remove the applicator, after removing the applicator we have to cut the tail portion to about to 6 centimeters. So, the whole length need not be there.

Another form of the vaginal insert containing progesterone is a TRIU-B, it has 1,2,3,4 ring, so each green ring has 186-milligram progesterone, and the white color is a dummy. Only these 3 are enough. Additionally, one more pink color ring is there, it is 400 mg Progesterone containing a thing. So, it should be inserted into the vagina, before that we have to load this TRIU-B inside the applicator, insert fully then, after insertion it will be like this. This is the cylinder or applicator, and another plunger is there, after inserting into the vagina, we have to push the TRIU-B inside the vagina, then the tail portion will be hanging. It should be cut only leaving a 6 centimeter from the vulva.

Friends, so far, we have seen how to apply CIDR in animals and how long it should be kept in the vagina followed by how you have to apply TRIU-B for the Estrous Synchronization program. We will stop here; we will continue with the CIDR in combination with other hormones in the next class for Estrous synchronization.

Thank you!

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PDF: Synchronization Using Progesterone and Advantages

5 Progesterone Protocols, Synchronization of Ovulation and Summary

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Transcript

Greetings friends!

As I told you in the last class, today we are continuing with CIDR and PGF2 Alpha and various combinations and it will be interesting to watch.

This slide shows the CIDR/ TRIU-B +PGF2 Alpha combination for the Synchronisation of Estrous. You consider today's (day 0) animal Estrous. Either day 10 means that it is in Diestrus or you can use it on day 6. From day 6 onwards day 10 you are inserting 8 days you keep the CIDR. On the 18th day you give PGF2 Alpha.19th day you remove the CIDR. 21st day and 22nd day you do AI (Artificial Insemination). Once again, I will repeat. Day 0 is Estrous day, day 10 insert CIDR/TRIU-B, day 18 give PGF2 Alpha, day 19 remove the CIDR, day 21 and 22 do AI. This is the best protocol for the Synchronisation of Estrous. Suppose if the animal is anestrous, insert the CIDR on day 0 then 8th day instead PGF2 Alpha, give GnRH 5ml (20 micrograms) Intramuscularly or eCG about 500 IU to 1000 IU you give Intramuscularly. 9th day you remove CIDR and day 11 and 12 do AI. At the time of AI again you can give GnRH, 2.5 ml

Intramuscularly. So, this is for two hornist animals, both the ovaries are Smooth. Previously I told day 10 CIDR, day 18 PGF2 Alpha, day 19 removal of CIDR, day 21 and 22nd doing AI. It is in the cyclical animals. If it is in an anestrous animal, this protocol is found to be the best protocol.So a combination of CIDR plus GnRH or eCG. It is not PGF2 Alpha, it is GnRH /PGF2 Alpha in true anestrous animals.

OK, the next protocol we are using is a Synchronisation of Ovulation. This is called synchronisation of Ovulation. So, whatever we used so far, it is Synchronisation of Estrous.

So the next step in this synchronisation program is the Synchronisation of Ovulation. Precisely it will synchronise the ovulation so that the consumption rate will be increased. The first protocol in the synchronisation of Ovulation is the Ovsynch protocol.

Suppose GnRH is given on day 0 and day 7 you give PGF2 Alpha, then on day 9 after 48 hours give GnRH again. After the 2nd GnRH injection, 16 to 18 hours later go for AI. This is called GPG protocol. Suppose an animal is cyclical, day 0 is Estrous, day 6 or7 you start this program. Suppose on day 6 you are starting the GnRH then 7 days later,i.e., on day 13, you will give PGF2 Alpha. Then two days later, on day 15, you will be going for GnRH injection, then on day 16, you will do AI. So, this protocol is the best protocol for the precise synchronization of Ovulation. This is called GPG protocol. Here the GnRH and the PGF2 Alpha are used. This (PPT) is very well explaining the Ovsynch protocol, that is GnRH, where Receptacle or Fertagyl is used to ovulate the dominant follicle. It may be present on day 6 or day 5, so you can give GnRH day 6 then 7 days later give PGF2 Alpha, then 48 hours later, i.e., after the PGF2 Alpha give GnRH, then 16 to 18 hours later, go for a Fixed Time AI.So this (G) is for synchronization of Estrous, this (P) protocol is for 16 to 24 hours before AI, this (G)we are giving GnRH to synchronize Ovulation also. So this is called a GPG Protocol or Ovsynch Protocol.

The scientists have improved the Ovsynch Protocol. So, if you don't know which day the animal expresses the Estrous, 12 days before the GnRH injection, give PGF2 Alpha to the cyclical animal.

So, animals will come to heat within 3 days, and it will be on day 6 or 7 again start the GnRH Protocol, GPG protocol i.e., GnRH- PGF2 Alpha-GnRH. This is called Presynch- Ovsynch Protocol. Then 16 to 18 hours later go for AI, this is called Presynch-Ovsynch Protocol.

Next is Pre synchronization: Presynch Ovsynch can be done with two PGF2 Alpha. First give PGF2 Alpha then 14 days later you give PGF2 Alpha, again 14 days later start the GPG protocol i.e., (GnRH-PGF2 Alpha-GnRH), then go for AI. So, if you see this protocol, you can follow any time. So this is very important and it is giving more chances for consumption in animals.

Next, we will see a G6G program.

Actually, in the Presynch Ovsynch Protocol, we give PGF2 Alpha then 14 days later we will give PGF2 Alpha again and 14 days or 12 to 14 days later we will start the Synchronisation Ovulation program. But in the G6G program, we will give PGF2 Alpha,2 days later we will give GnRH, 6 days later we will again give GnRH.This is the first GnRH of the Ovsynch program. Then we will continue the Ovsynch protocol. This proved that it is giving more consumption rate than any other protocol.

Next, the Double Ovsynch protocol is also recommended. That is double the time you are doing synchronization of the Ovulation program. Give GnRH, 7 days later give PGF2 Alpha, again 3 days later give 2nd GnRH, then 7 days later you start the 3rd GnRH, and you are continuing the Ovsynch protocol. This is called a Double Ovsynch. So, there are many protocols available, and I have told you, whichever protocol you prefer in the field according to the availability of the drug in your area, you can choose. Many avenues are available in reproduction to improve the fertility rate in infertile animals.

Now, look at this Cosynch Programme. In GnRH, we have to visit the GPG protocol many times. But in the Cosynch Programme, there is a difference. The difference between Ovsynch and Cosynch is that 2 days later, we will give a PGF2 Alpha,48 hours after the PGF2 Alpha injection. But hereafter the PGF2 Alpha injection, at the time of AI we will go for GnRH injection. So, 16 to 18 hours before the AI we

will give a GnRH injection. But here we are not doing that, you can have these at day 10, So on day 10 along with AI, you can go for GnRH injection also. So, one more visit you are avoiding in Cosynch protocol. so, at the time of AI itself, you are administering the GnRH. This is called a Cosynch Programme.

Next is the Heatsink Programme. Suppose if you don't have GnRH. This is Heatsynch Protocol, day 0 you are giving GnRH, day 7 PGF2 Alpha, day 8 giving E2 (Esterol Benzoate) or Caproate. So Esterol Benzoate any Estrogen can be given, then day 10 we are doing Timed Artificial Insemination(TAI). So, this is called Heatsynch Protocol.

The next protocol is the CIDR protocol. So Ovsynch + CIDR you can combine together.GnRH insert CIDR, 7th day give PGF2 Alpha and remove CIDR, 9th day give GnRH and 10th day you can go for Breeding. In Cosynch + CIDR also give GnRH insert CIDR,7th day give PGF2 Alpha, then 10th day or after the 72 hours of PGF2 Alpha injection, give GnRH and Breed the animal. So, this is Cosynch +CIDR.

In Select D Synch + CIDR, give GnRH insert CIDR, remove the CIDR on day 7, breed the animal by deducting the estrous. This is called Select D Synch. So, Select D Synch means to give GnRH, insert CIDR, 7th day give PGF2 Alpha, remove the CIDR, breed the animal by detecting the Estrous. So, there are many protocols available. All these protocols can be used to treat Repeat breeders, Anestrus and Cystic vary. So, in Cystic Ovary also this can be used. So, by way of using the Estrous Synchronisation Programme, many infertility cases can be treated.

I think all of you will utilize this program for improving the fertility of infertile cows and buffaloes under field conditions.

What all are the points to be remembered in the Estrous Synchronisation program:

- Observe the cyclicity pattern of the animal.
- Select the appropriate protocol according to the owner's status, ability, patience, and everything. You select the best

protocol for cyclical and Anestrous animals. Best is Prostaglandin for cyclical animals and Progesterone for cyclical or non-cyclical animals.

- Ensure ovulation by adding GnRH in the selected protocols
- Recommended performing AI at 24 hours intervals in Estrous Synchronisation programs.
- So, you can do double AI in any synchronization program.

These are the points to be remembered.

Dear friends, so in the entire series of classes we have seen the Estrous synchronization technique in cows and buffaloes, which is an important technique to augment fertility both in normal cycling and the infertile animals, so you can use this technique for improving fertility under field condition to increase the economy of the poor farming community of rural India.

And at anytime and anyplace from anywhere, you can contact me at this address.

Thanks to one and all.

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