Management of Metabolic and Production Disorders in Cattle

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Contents

Course Description

This course will enhance the knowledge and skill of the veterinarians with recent updates through continuing education enabling them to implement appropriate treatment protocols and control of production disorders for the cattle at the field level thereby enhancing the livelihood and increasing the economy of farmers. Metabolic disorder or production disorder is most common disease entities in lactating dairy animals which leads to severe economic losses in terms of reduction in milk yield and impaired reproductive performance. Dairy production is challenged by the fact that 30 – 50 per cent of dairy cows are affected by one or more forms of metabolic or infectious disease at the time of calving. In cattle, metabolic diseases include Ketosis, Milk fever, Downer cow syndrome, Hypomagnesaemia, Post-parturient haemoglobinuria and Mastitis. These metabolic disease conditions are multifactorial and commonly occur due to high physiological stress or demand for nutrients with late pregnancy and early lactation being key period. Milk fever has been associated with threefold increase in risk of dystocia, uterine prolapse, retained fetal membranes, metritis, abomasal displacement and a nearly ninefold increase in clinical ketosis and Mastitis.

This course will benefit veterinarians to enrich knowledge and skill on sub-clinical and clinical form of metabolic disorders and measures for early diagnosis and management in cattle and small ruminants. This in turn will help to increase the economy of the farmers by saving the life of the animals, preventing the death of cattle from the diseases and by sustaining animal production / productivity.

Course Content

- Hypocalcemia (Milk fever) in cattle
- Downer cow syndrome
- Ketosis in ruminants

- Update in bovine Mastitis
- Post-parturient hemoglobinuria in cattle
- Hypomagnesemic tetany in cattle

Course Audience

- Practicing Veterinarians
- Pre-final, final year and internee of veterinary science
- Veterinarians working in State Animal Husbandry Departments, NGOs
- Veterinarians working in State Veterinary Universities / Veterinary Colleges

Outcomes of this Course

- Understand the diagnosis and staging of milk fever in cattle.
- Know the pathogenesis and differential diagnosis of recumbent cow.
- Diagnose ketosis and management of its complications in ruminants.
- Gain and update knowledge on treatment and alternatives to antibiotics in bovine Mastitis.
- Learn differential diagnosis approach to post-parturient haemoglobinuria.
- Learn the managemental practices and therapeutic approach to hypomagnesemic tetany.

PART I

WEEK 1: HYPOCALCEMIA (MILK FEVER) IN CATTLE

1 Introduction to Milk Fever – Parturient Paresis

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Transcript

Hello! greetings to all,

I welcome all the vets to the agMOOCs program on 'Metabolic Disorders in Cattle', jointly organized by commonwealth learning Canada and the director of extension TANVAS. Milk production is the main source for rural farmers and dairy cattle nowadays, they are going for high yielders, and crossbreeding of a non-descript breed with Holstein Friesian and Jersey cattle breed predisposes them to go for various disorders. So Metabolic disorders or production diseases are ones that mainly occur due to high yielders, deficiency of trace elements, major/ measure minerals, and energy imbalance. and this occurs mainly during periparturient cows during a transition period.

And the occurrence of subclinical metabolic disorders causes it's a silent killer, we can say it causes severe economic loss to the dairy industry. So, this program is designed in such a way to cover all the important metabolic disorders under a Program.

My topic is on a Milk fever or Parturient Paresis in dairy cattle

and this program is designed into different lessons that are a compressing of five lessons starting from 1,2,3,4, and 5.

In this Lesson-1, first I will be covering the Importance of Parturient Paresis or Milk fever in dairy cattle. How does it occur? and the Predisposing factors, risk factors involved, and the Economic importance of Milk fever in cattle. and

Lesson-2; mainly includes how it occurs, that is the effect of hypocalcemia on various systems and the Clinical signs manifested in stage-1, stage-2, stage-3, and the outcome of the disease.

Lesson-3; it's mainly involved in the Complications and the Outcome of Hypocalcemia, and the concurrent ailment and it also covers the how-to diagnose the disease, Differential Diagnosis, and all.

Lesson-4; is an important part of the Program, it is how to Manage Hypocalcemia either with a Calcium treatment like Intravenous therapy or the subcutaneous or oral preparation and then how the animals respond to the treatment for both the unfavorable response and the favorable response the treatment will be covered in lesson-4. And,

Finally, the Lesson-5 is comprised of how to prevent the Hypocalcemia because causes it's subclinical is a major part of a Metabolic disorder, so how to prevent the disease during the dry calf period as well as a post-calving. This is about the full module on Hypocalcemia in Cattle.

Hello! Welcome to Lesson-1 on Introduction of Parturient Paresis in Cattle.

So, in this lesson, we are having a different components like

- Introduction,
- How the Hypercalcemia occurs and
- what are all the risk factors involved and
- the Etiology for Hypocalcemia and
- the economic importance.

Commonly called a Parturient Paresis or Milk fever Hypocalcemia all are synonyms.

- This is an important Metabolic disorder; it constitutes about 10% of a dairy cattle recently calved animals or prone to Hypocalcemia
- In sub-clinical Hypocalcemia that is silent there, it will not be any overt clinical signs, only a reduction in milk yield. so, this clinical hypercalcemia constitutes about 50% of multiparous periparturient dairy cattle, so that is why it forms a greater impact on the economy of the dairy industry, and
- Among the dairy cattle so due to the cross-breeding of our indigenous cattle with Jersey and Holstein Friesian to increase the milk yield, actually, these Jersey breeds are highly prone to Hypocalcemia, they are genetically predisposed, and the intestinal absorption of calcium is less when compared to other breeds of cattle, so that is why it is highly prone to Hypocalcemia. When does it occur? Usually, Hypocalcemia occurs in a transition period, which is 3 weeks prior to calving and 3 weeks after calving is the transition period. During that period, the majority of cases occur immediately after calving, that is within 24 hours of calving there is a high influx of calcium into the milk and rest of the cases occur 24 to 48 hours and in some cases even occur before calving also, and

How does it occur?

In high yielders because it is nothing but there is an imbalance between the input of dietary calcium and the output of calcium in the form of milk. so that is why it occurs immediately after calving and also there is increased loss of calcium in the milk, and

What is the main reason for Hypercalcemia; is there is a reduction in ionized calcium in the blood, so usually the blood calcium is serum calcium is total calcium in that protein-bound form and free forms are there, so ionized calcium is the part of total calcium that exerts its physiological function.

How the level of calcium is maintained in the bloodstream:
Actually, there are two hormones are involved:

- one is a Parathamron and
- another is a 1-5 dihydroxycholicals for all vitamin d3

And there are 3 systems, involved in the maintenance of Calcium in cattle that is ;

1. Kidney and
2. Intestine and
3. Bone

So normally the calcium is that is ingested through the diet, and it is it's called calcium homeostasis. So normal level is maintained by the influence of Parathamron and vitamin D3 through this organ, so normally it is absorbed through the intestine and excess calcium is excreted through kidneys and in the production animal, the calcium is in addition to the physiological function, the additional calcium is excreted in milk also, that is why there is a high demand for calcium in a post parturient animal.

What is ***Calcium Homeostasis***?

That means, there is a feedback mechanism, that is physiologically the animal is going for excess calcium or low calcium in the blood, then there is a hormonal influence, that is a Parathamron hormone, so once the blood level of calcium decreases, then it stimulates the parathyroid gland to secrete a Parathamron. We know the calcium level decreases, it stimulates the parathyroid gland, so to secrete the Parathamron this Parathamron has an effect on the kidney, and it catalyzed that is a 25 hydroxycholef calciferol into 125 hydroxycholesterols, that is colic calciferol, that is a vitamin D3 and it directly increases the reabsorption of calcium from the renal tubules and this vitamin D3 it exerts its function on bone and intestine. So in the bone, it stimulates the osteoclastic activity, thereby there is a mobilization of calcium from the one to the

bloodstream in addition in the intestine it stimulates the absorption of calcium from the intestine so the excess calcium from the bone and bone reception intestinal absorption and the kidney reabsorption is considered by both Parathamron and vitamin D3. Ultimately the calcium level in the blood increases. This adaptive mechanism is a fails in a recently covered animal due to increased demand for calcium more than the input, so this adaptive mechanism is insufficient to meet the demand for calcium in the form of milk or cholesterol leading to Hypocalcemia.

So, what are the **Risk Factors** Involved in Hypocalcemia, how the animals are predisposed to go for a Parturient Paresis?

So mainly it's

- an Animal factor as well as:
- a Dietary and environmental risk factors:

What is the Animal factor; excessive loss of calcium in the colostrum, so the animal usually in the first calving, second calving as the number of calving increases in multiparous animal third lactation or fourth lactation the yield increases, so during the transition period from that is a dry period to post-calving there is a sudden surge of calcium into the colostrum. So, this dramatic increase in the loss of calcium occurs at a faster rate so the influx of calcium to the bloodstream rate is unable to meet the loss in the colostrum and the second thing even though the Parathamron hormone and vitamin D3 functions, due to some reason like there is the occurrence of diarrhea, so the intestinal absorption decreases, there also the calcium level decreases. and then the marked reduction in the mobilization of calcium, for example as the animal age advances the number of receptors that are an osteoclastic activity decreases. So, the response, the rate of mobilization is less in the multiparous animals when compared to a primiparous animal. So, these are all the major animal component that predisposes the animal to go for Hypocalcemia. and another important factor that plays a major role in causing Hypocalcemia is: The *Dietary and*

Environmental factors like the amount of Calcium, Phosphorus, and Potassium and the Dietary Cat-ion, An-ion balance, play a major role.

How the Dietary and Environmental factors causing Hypocalcemia, for example, if the animal during the dry period, the owner may be supplementing high Calcium in the diet with the aim of to prevent Hypocalcemia before calving but it is not that way it causes the less stimulation for Parathamron hormone secretion, so excess calcium decreases the Parathamron need, thereby it is causing Hypocalcemia, and less green pressure, usually potassium-rich fodder that is fed during the transition period or a recently calved animal, this excess potassium causes reduced calcium absorption in the intestine due to an increased level of cat-ion in the feed, and Dietary *Magnesium*; actually, most of the time Hypocalcemia occurs concurrently with the hyper magnesium, what happens is, that magnesium is an important factor for the secretion of Parathamron so whenever there is decreased magnesium secretion, the parathyroid secretion also decreases that's thus predisposing to Hypocalcemia.

Then a dietary excess *Phosphorus* supplementation during a dry calf period; so, for example the animals are fed with excess wheat bran or rice bran, which is rich in Phosphorus and this may cause a reduced calcium level in the blood through Parathamron activity.

Okay, all these factors how affect the Calcium Homeostasis is the excess loss of calcium in the colostrum, that is there is a demand for calcium but the mobilization of calcium from the storage skeleton may not be sufficient to meet the demand for excess calcium in the milk and dietary cat[1]ion and an-ion difference, so this plays a major role. The increase in the cat-ion salts like Sodium and Potassium in the diet may decrease the that is a calcium availability to the animal and apart from that, there is an impairment of absorption of calcium from the intestine as I said earlier due to some compromise in the Intestine like diarrhea and then increase Dietary Phosphorus and the Calcium during the dry period all those factors that it causes reduced Parathamron secretion and

absorption of calcium from the gut and predispose them to go for Hypercalcemia. It is so important because our Indian economy is mainly based on rural industry, especially the dairy industry and this occurrence of Hypocalcemia cannot be prevented by vaccination or any other medication, so it is a metabolic disorder due to there is an impairment of input and output of calcium in the diet in the system.

So, this Milk fever in some cases in multiparous dairy cattle, high yielders the incident occurrence of Milk fever will be again and again it occurs within for example after calving within 10 days, it occurs 3 times or 4 times, it's called as Calcium cycles or Relapse. So relapse of this disease causes severe economic loss and the untreated cases or delays in the management of Hypercalcemia especially if the cow goes for sternal recumbency then it becomes downer cow and the success of management of downer cow is very very limited, even in western countries and this Hypocalcemia may produce for the animal to go for Dystocia and all other reproductive disorders like Retained placenta, Metritis and subsequently it decreases the general systemic state and there will be reduced intake and followed with reduced milk yield and apart from that most of the time it decreases the immune system also and most of the time recumbent cows they are highly predisposed for Mastitis. So once Mastitis is there occurs then also there is a severe loss of milk due to Mastitis and apart from that there is in high illness in will maintained form, usually the Hypocalcemia may predispose them to go for Abomasum displacement and concurrently there will be energy in a negative energy balance it will have Ketosis and finally, all these disorders may force the farmer to Cull the animal. So, it is an economically important disease.

So, in this Lesson-1, we have seen about

- What is Hypocalcemia and which breeds it is so common and usually, it occurs in a multiparous dairy cattle and
- How the Calcium Homeostasis is maintained, mainly the Parathamron and vitamin D3 role and then
- The 3 systems involved Kidney, Intestine, Bone and

- What all are the Risk Factors like the Reduced mobilization of Calcium from bone or reduced intestinal absorption or else the Environmental factor like increased dietary Calcium, Phosphorus and Dietary Cat-ion and An-ion difference, that is increased Cat-ion in the diet and
- How it causes severe economic loss to the farmer and

With this, we are completing the class and we will see in the next class.

Thank you!

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PDF: Introduction to Milk Fever – Parturient Paresis

2 Pathogenesis and Clinical Findings

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Transcript

Hello vets,

Now we are on a second lesson, so in the previous class we saw about the Introduction of Hypercalcemia and How does it occur and the Various Risk factors involved, and the Economic Importance of Hypoglycemia.

Today in this class we are going to see about the Pathogenesis and Clinical signs of Hypercalcemia.

In the Clinical signs, we are going to see about different stages of Hypercalcemia; stage-1, stage-2, and stage-3.

Actually, Calcium is required mainly for membrane stability, that is the nerve cell membrane stability and contractility of the skeletal and smooth muscle and the release of acetylcholine at a neuromuscular junction, thus it aids in the contractility of all muscles.

So what happens if there is Hypocalcemia that is <8mg/per deciliter and this nerve cell membrane becomes stabilities get altered and there will be severe irritability, so always if you used to see excitatory signs due to loss of dysfunction, so the animal becomes there is an increased twitching of a group of muscles called Fasciculation on the shoulder and the head muscles, and

there will be mild titanium, that is the animal will have a disinclination to move, and there is the twitching of muscle and there there is bruxism.

So all these signs are mainly due to decreased membrane stability, and this Hypocalcemia causes loss of muscular contractility in both the skeletal and smooth muscle, then what happens to contractility muscle that is flaccid paresis that is why the animal becomes recumbent. and Smooth muscle loss of smooth muscle contractility, so all the smooth muscles get involved in cardiac muscle, so there is a loss of cardiac contractility, so cardiac output decreases.

And vascular smooth muscle loss of tonicity or contractility leads to vasodilation, so ultimately there is hypotension, which is the major pathognomic sign in Hypocalcemia.

Apart from that, there is reduced rumen motility and intestinal contraction, that's why you are getting scanty feces and the bloat.

And there is a decreased release of acetylcholine, so there is a reduced neurotransmitter secretion at various levels, mainly you can see pupillary dilatation as a cause of reduced acetylcholine release.

There is a certain correlation between serum calcium level and the occurrence of clinical signs, So normal animals you can say it will be 8 to 10.5 milligram/deciliter and in mild Hypocalcemia, sub-clinical Hypocalcemia is very common and there will be a mild reduction in a calcium level like 7.5 milligram/deciliter and there will be reduced mild reduction in milk yield and rumen activity and reduced appetite. Only certain changes are absorbed.

And in Clinical Hypocalcemia to occur the value will be <6 or as low as 2 milligrams also can be recorded. So accordingly the animals may you may get a stage-1, stage-2, and stage-3 from standing to the sternum to lateral recumbency.

But strictly speaking, there is no exact positive correlation between the level of Calcium and Clinical sign occurrence, but however, the ionized calcium level is playing a major role in may influence the occurrence of Clinical signs.

Hypercalcemia is classified as stage-1, stage-2, and stage-3.

Stage-1; is a standing posture, so the animal still is able to stand but it is unable to walk mind the titanium signs, so the calcium values are it can range from 4.9 to 7.5 and

Stage-2; it's a severe form, there is severe muscle flaccidity, now the animal becomes recumbent, so it is sternal recumbency and still severe form is

Stage-3; it is complete there is flaccidity of all muscles, animals become lateral recumbency, and there is a reduced cardiac output, and the animal is mostly comatose

So this is the classical form of Hypercalcemia of Stage-1, it's a Stage of Excitement as I already explained due to that is a nerve cell membrane instability. So, in this stage, if the animals are treated at Stage-1, you can avoid there is they may not go for sternal or lateral recumbency.

So the classical signs of **Stage-1(Stage of Excitement)** are :

- still the animal standing,
- but it has mild titanium signs,
- so it is a disinclination to move,
- disinflation to food, and there will be otherwise the temperature pulse and other normal or
- due to this muscle's increased contractility, there will be that is a stiffening of muscle, an increase in temperature, and
- there will be grinding of teeth and
- protrusion of tongue
- reduced rumen activity
- the volume of dung voided also less and this is the classical form of stage-1 milk fever.
- another important sign at this stage is Fasciculation, which is the twitching of a group of muscles, especially on the shoulder and neck muscles and the head.

Stage-2(Sternal Recumbency): is a severe form of Hypocalcemia the animals are on Sternal Recumbency. In these animals there will be;

- pupillary dilatation,
- reduced rumen activity, and
- reaction reflux, so it will have bloat
- scanty feces
- the temperature may be normal as abnormal temperature due to reduced cardiac output
- peripheral circulatory failure pupillary dilatation and
- here the heart rate is more than is tachycardia but even then you are unable to auscultate that is a weak cardiac contraction, so it's very difficult to hear the cardiac sounds
- likewise, the pulse is also rapid, but it is very weak
- another important sign at a field level you can see there it's very difficult to raise the jugular vein, which is the classical sign due to loss of vascular tonicity.

In this the class we are seeing about :

- the Pathogenesis of Hypercalcemia is how the nerve cell membrane gets affected and
- the loss of acetylcholine release, and
- the reduced contractility of musculature both skeletal and smooth muscle and
- the various stages of milk fever-like –

stage-1 standing posture and
stage 2 sternal recumbency.
So, see you in the next class.
Thank you!

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PDF: Pathogenesis and Clinical findings

3 Third stage Milk Fever

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Transcript

Welcome to lesson-3, before that just recall what we have dealt with in the previous class that is Pathogenesis and the Clinical signs of Hypocalcemia. Mainly how the reduced level of calcium has influenced the skeletal and smooth muscle contractility, causing reduced cardiac function, as well as nerve cell membrane instability, and we have seen the stage-1 Hypocalcemia characterized by a stage of excitement and stage-2 that's the main stronghold recumbency, flaccidity of all the skeletal muscle, and there is loss of muscular tone an animal becomes sternal recumbency and there will be lateral twitching of head and neck and in addition there will be bloat and reduced defecation and there is tachycardia, but the poor intensity of cardiac sound and weak pulse are all the signs we have seen in stage-2 Milk fever.

And today we have that is a third-stage milk fever, and we are going to see about the complications of Milk fever and How to diagnose Hypocalcemia at the field level as well as Differential diagnosis.

Stage-3 really is a challenging one for the veterinarian because most of the animal dies at this stage. See what happens there is a severe reduction in the Calcium level and the animal is in the sternal

recumbency if it is not properly treated it will be going for lateral recumbency and almost you can say it is a comatose stage and the animal there will be lots of complete loss of skeletal muscle and it is unable to raise its head and neck.

So in the sternal recumbency, you could see you can appreciate the lateral kicking of the head and neck. Now in the lateral recumbency, it is unable to raise the head and neck, it is unable to maintain the sitting posture, so now it is on lateral recumbency and there is a severe tachycardia, it is very very difficult to raise the jugular vein and there is abnormal temperature and the animal is comatose and there is reduced defecation, urination and there is bloat.

So if the animal is left in that stage, it will die within 12 to 24 hours of lateral recumbency. So most of the time the hypercalcemic animal will have a concurrent ailment.

Sometimes you may give specific treatment to Hypercalcemia but it will fail because there will be concurrent hypomagnesemia and then hypophosphatemia and in Hypocalcemia, there is reduced rumen activity and intake also reduces.

So, there will be a negative energy balance, and the animal is predisposed to Ketosis. And recumbent animal with additional reduced immune activity, it is highly predisposed to Mastitis, especially Peracute Toxic Mastitis.

And then finally the animal's Sternal recumbency, if there are more than four hours the animal is on recumbent without any treatment it highly predisposes the animal to go for downer cow syndrome and it's a main/major complication of Hypocalcemia.

How to **Diagnose** Hypocalcemia in Cattle:

Usually the classical, clinical signs of different stages, apart from that to confirm the disease we need to go for Blood analysis, that is a Serum Calcium level, either total calcium or Ionized Calcium.

Total calcium is easy to estimate in any laboratory and will give the value of calcium if the sample is given. There is Ionized calcium, it will be estimated in a referral center, that is based on either

the blood gas analyzer or electrolyte analyzer, high-end laboratory facilities are needed to estimate the Ionized calcium level.

Apart from Calcium concurrently you need to measure the Magnesium level as well as a Phosphorous level. There will be less Magnesium and Serum Phosphorus also, so in the clinical[1]pathological analysis, apart from the estimation of calcium in the blood, other components to be estimated are Inorganic Phosphorus, there will be Hypophosphatemia.

Apart from that most animals will have a negative energy balance, so we need to estimate the blood glucose level, if it is less than 50 milligram/deciliter, then we need to give a dextrose treatment also.

Apart from this recumbent animal, there will be the release of muscle enzyme-like Creatine kinase(CK) and Aspartate aminotransferase.

And in blood hematological alterations there will be Neutrophilia, Lymphopenia, and Eosinopenia. These are all but they are not specific for Hypocalcemia but there are changes you can observe.

So apart from clinical signs due to Hypocalcemia Calcium exerts its major effect on skeletal and cardiac muscle contractility. So there will be in ECG you can appreciate prolongation of the acute interval, which is a major change in cardiac activity.

Another important field test to diagnose the Hypercalcemia is Sulkowitch test. In the homeostasis I have clearly mentioned that in a normal animal there is excess calcium is excreted in the urine, so by adding urine with Sulkowitch reagent, a normal animal you will get a white color discoloration or precipitation.

Whereas in Hypocalcemic animals due to the action of Parathamron hormone and vitamin D3, there will be complete resorption of calcium from the urine, so there will no or less Hypocalcemia in the urine analysis. So the clear color there is no white precipitation indicates there is hypocalcemia in animals. So always we need to differentiate the similar disease condition that occurs during the periparturient period, that is a downer cow syndrome following a Milk fever and usually, the animal is recumbent, so immediately after calving common disease or acute

carbohydrate engorgement and hip dysplasia, then that is degenerative myopathy and Obturator nerve paralysis, fat cow syndrome and even severe toxemia like your peritonitis due to foreign body or else coliform mastitis or materials causing the animal to become recumbent. So the common differential diagnosis as we discussed earlier mainly downer cow syndrome, then a hip dysplasia, then a pelvic fracture, then Obturator nerve paralysis, the common fatty cow syndrome, and toxic menstrual mastitis, these are all the common differential diagnosis.

So in this lesson-3 of a module, we had seen about

- How to diagnose the hypocalcemia mainly based on clinical signs and
- then laboratory technique and
- then complications of Hypoglycemia and
- Differential diagnosis and Diagnosis.

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PDF: Third Stage Milk Fever

4 Treatment

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Transcript

Welcome to lesson-4 so before that, in the previous class we saw about

- What is a stage-3 milk fever,
- What are the classical clinical signs,
- the complication of Milk fever,
- How to diagnose the Milk fever based on laboratory techniques, as well as a field test, and
- What is the Differential diagnosis for Milk fever in cattle?

Now we are going to see about the lesson-4 which mainly comprises Treatment which is an important part of this section and this includes:

- How to manage Hypocalcemia by Intravenous therapy as well as subcutaneous and oral therapy
- What are all the favorable responses immediately after injection of IV calcium, as well as unfavorable responses or poor responses to Calcium supplementation?

So the main goal of Hypercalcemia Treatment is Intravenous administration of rapid administration of Calcium solution. So the IV route is the most preferable route and followed by the subcutaneous route.

Actually, commercially available solutions yield about 8 to10 grams of calcium per bottle. The general thumb rule for calculating the dose of calcium in Hypercalcemia animals is 1 gram per 45 kg body weight. So roughly 450 kg body weight cattle meal you can give 10-gram maximum, that is the main thumb rule, if the animal is 300 kg accordingly we need to reduce the calcium dose.

And sometimes there are 23% calcium is also available but most of the time it uses about 10 grams of calcium in this bottle. IV route is the preferred route for administering calcium to get a rapid response in a Hypocalcemia animal and a calculated dose can be given rapidly between 10 to 15 minutes you can give an IV injection.

And so the important part is we need to monitor the cardiac activity because you are giving a rapid rate of calcium it directly has an effect on the heart rate, so whenever there is severe bradycardia or tachycardia, then we need to manage the flow rate of calcium accordingly. Suppose a case there is a recurrent Hypocalcemia in a dairy cattle it's called Calcium cyclers, in that case, we need to give the calculated dose, half the dose through the subcutaneous route to get a favorable response.

The challenge in the treatment of Hypercalcemia is you may get both relapses of Hypercalcemia after a Treatment or the animal diet during Treatment. So underdosing and overdosing is avoided, they may occur due to various reason.

After the Intravenous administration of calcium, the animal is in sternal recumbency, so the favorable response to the calcium therapy you can get immediately, within a few minutes. So the animal the all the flaccid muscles, they receive the neuro neuronal impulses, and it promotes skeletal and smooth muscle contraction. So there will immediately stimulate the adaptation reflex, the relief of bloat, and the animal cardiac activity increases. So heart rate increases, the intensity of cardiac sound improves, pulse quality

improves, and the urination activity gets stimulated. So the animal will urinate immediately and profuse urination and then defecation the animal will stand and become recumbent to be converted to standing posture and there will be muscular fasciculation, the contractility improves or regained especially from the hindquarter towards the forelimb and later on the animals become normal and it will work normally.

Sometimes the challenge in the Treatment of Hypercalcemia is a poor response or the animal may die while giving calcium Treatment or immediately after treatment it may fall and die. So the reasons are many ;

- Suppose the calcium administered may overdose, so the animal may be thin and the calculated dose exceeds the normal causing severe bradycardia and animal death. and
- Another thing the animal suppose in a high, there is heat stress or it is the animal is recumbency near open space and the high temperature also may cause increase the sensitivity of calcium and the animal may die.
- Another thing most of the time at the field level what will happen in Hypercalcemia, the owner themselves or farmers will give multiple dosages of subcutaneous injection, so after the arrival of a veterinarian if you start giving IV or calcium at a calculated day immediately the peripheral circulation improves and whatever the dose given by the farmer by the subcutaneous route, that also adds on to your calculated dose. So immediately there is increased calcium overdose or toxicity and it compromises the cardia, causing the severe cardiac arrhythmia.
- Apart from that, there are toxic conditions like severe toxemia materials Mastitis, and Peritonitis and these conditions may increase the sensitivity of calcium. So thereby causes even a calculated dose may become toxic to animals.
- Apart from Hypokalemia, and in Hyperkalemia also the sensitivity of calcium to the cardiac muscle also increases.

- So sudden sometimes during treatment, the animal gets frightened or excited because of sudden adrenal release that also causes a sudden cardiac arrest. So always whenever you are going to treat a Hypercalcemia animal you should have an antidote with you that is the atropine sulphate, immediately you should give it intravenously to contract the cardiac arrhythmias.
- Whenever there is an unusual response, that is a relapse occurs in Hypercalcemia after IV administration. In those cases, the subcutaneous route is the best choice, so calculate a dose of 50% you can administer through a sufficient route. At your site usually, 50 to 75 ml you can give is especially on shoulder muscles and the subcutaneous route is suggested mainly for calcium cyclers.
- Apart from parental administration and supplementation, oral supplementation is also suggested. so there are various commercial preparations nowadays available in the form of gel or powder or liquid and these contain mainly calcium chloride or calcium propionate, so according to the manufacturer's prescription, we need to administer carefully you need to drench the cattle for a one or two days in addition to the IV calcium.

In **General management** apart from drug therapy, since the animal is on a recumbent for a few hours then we need to avoid that is you should probe the animal from lateral to sternal recumbency, we should roll the animal from one side to another side, and then we should clean the udder, we should avoid mastitis, so regular milking is also important, always the required contact for feed and water should be adjacent to the animal, and we know the animal tries to get up then we should give assisted lifting to the animal.

Normal the Failure to Hypercalcemia is mainly due to the repeated occurrence of a Hypercalcemia in a multiparous dairy cattle, that is a calcium cycler and the other thing once the animal becomes recumbent it is predisposed to go for downer cow

syndrome, so about 30- 40% of Hypercalcemia untreated cases, they will go for downer cow syndrome.

We have seen the :

- How to Treat Hypercalcemia by various routes, the most preferred route is Intravenous followed by subcutaneous injection,
- the favorable and unfavorable response to Hypercalcemia, and
- How to manage a recumbent cow also

Thank you!

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PDF: Treatment

5 Control

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Transcript

Welcome! to lesson-5 before that the last class that is lesson-4 we have seen about the

How to administer IV calcium, subcutaneous calcium, and oral supplementation of calcium in Hypocalcemia animals, and

How the animal immediately responds to your IV calcium like there is a relief of bloat, immediate urination, defecation, and the animal will walk and

Apart from that Failure to respond to Treatment and there is sudden death during Calcium administration due to cardiac arrest how do prevent and

General Management of recumbent cow we have seen in the last class. and In this class we are going to see about:

How to prevent the occurrence of Hypocalcemia because this is the major part, we need to educate the farmers.

So, it is only a dietary error, Hypocalcemia occurs mainly due to dietary error only. So, the farmers may be giving a calcium excess calcium during a dry cow and after that, it may lead to Hypocalcemia.

So mainly dietary management plays a major role in the prevention and control of Hypocalcemia. So usually, the farmers

give excess calcium during dry cow, so optimum Calcium should be supplemented in the dry cow ration as well as optimum Phosphorus that should not be more phosphorus in the diet, as well as dietary Potassium level, should be reduced because excess potassium may predispose an animal to go for Hypocalcemia and then adequate the Magnesium should be supplemented in the ration. So apart from that the addition of a zeolite that is aluminum chloride and sodium along with so it will increase the an-ion content of the diet and improve the calcium absorption. So, the DCAD plays a major role in the control of Hypocalcemia, so farmers should be educated and the importance of the addition of elements and the avoidance of a certain element. so, the dietary an-ions like the addition of chloride and the sulfur-containing elements in the diet will prevent the occurrence of Hypocalcemia in a parturient cow.

Apart from the administration of vitamin D3 which is 10 million units per cow, approximately one week prior to calving a single intramuscular injection may prevent the occurrence of Hypocalcemia in a parturient cow.

Apart from the oral supplementation of vitamin D3 is also suggested prior to calving and Supplementation of calcium chloride now all the calcium supplementations should be started at the time of calving or after calving. So daily 50 grams of calcium supplementation in the form of a mineral mixture prevents the occurrence of calcium.

To avoid the excess influx of calcium in the colostrum so partial milking is suggested in recently cowed/calved animals in addition to other insufflation during the first day of lactation will reduce the need for calcium in the form of milk. and

The most important part of control is the addition of a balanced mineral mixture during the different stages of lactating and gestation period of cattle.

So, the TANVAS mineral mixture is formulated in such a way to meet the demand for calcium during the dry cow period, at the time of calving, and post-calving, During the lactation period,

liberal supplementation of this mineral mixture will prevent the occurrence of Hypocalcemia in dairy cattle.

As a whole in summary the important points to be remembered in Hypocalcemia or Milk fever:

- The parturient paresis mainly occurs in multiparous dairy cattle and
- subclinical Hypocalcemia is common in 50%of dairy cattle causing severe economic loss to the dairy industry.
- It mainly occurs in Jersey breeds of cattle because of the genetic predisposition and
- this Hypocalcemia occurs due to altered calcium homeostasis,
- the hormones involved in the homeostasis are Parathamron hormone and vitamin D3 and
- the organs involved in the homeostasis Kidney, Intestine, Bone and
- Why there is Hypocalcemia in recently called animals is mainly due to there is an imbalance between dietary input and output of Calcium in the form of milk, so mainly it occurs due to reduced intestinal absorption, reduced reception of bone, and then the genetic predisposition of the cattle.
- so the calcium is mainly required for skeletal and smooth muscle contractility, so reduced calcium causes a flaccid paralysis and there is reduced cardiac activity, reduced cardiac output, and a peripheral circulatory failure,
- so according to the level of calcium and the clinical signs are classified as stage-1 characterized by stage of excitement and standing posture,
- stage-2 it's a become sternal recurrency with the lateral thinking of a head and neck there'll be bloat and it's difficult to raise the jugular vein, scanty feces, pupillary dilatation and
- finally the stage-3 characterized by lateral recumbency, the animals are comatose and the animals are if untreated it becomes it will die within 12 hours of time. and
- How to Diagnose Hypocalcemia, mainly by estimation of Serum

Calcium. if possible Ionized Calcium that is less than the 8-milligram percentage of calcium. It's an indication of a Hypocalcemia and then the

- Management mainly the supplementation of calcium borrow gluconate and the thumb rule for the dose is a one gram per 45 kg body weight and there will be a rapid intravenous administration of calcium. Always a cardiac assault disease must to monitor the cardiac arrhythmia and there will be an immediate response after IV calcium the animal will have improved muscular activity, there will be immediate urination, and there will be muscle fasciculation on the hindquarter, and there will be belching recitation, reflex gets stimulated, relief of bloat, and cardiac intensity increases, and the animal will work. and
- There will be a poor response, or the animal may die during calcium administration due to increased sensitivity of cardiac muscles to calcium due to various reasons.
- Sometimes recurrent Hypocalcemia is common in dairy cattle it's called calcium cyclers and they should be managed by IV calcium followed by subcutaneous calcium also. and
- then at last how to Control the occurrence of Hypocalcemia in dairy cattle are we need to educate the farmers and they should give a balanced mineral mixture during a dry cow as well as after calving. They should reduce the excess calcium feeding and excess phosphorous feeding before calving and after calving sufficient calcium should be supplemented in the form of a mineral mixture.

So with this module on Hypocalcemia, I conclude.

Thank you one and all!

Download

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PART II

WEEK 2: DOWNER COW SYNDROME

1 Introduction to Downer Cow Syndrome

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Transcript

Dear veterinarians! greetings to everyone! I Welcome you all to this session on donor cow syndrome. So, in this session we will be seeing on five classes and regarding the first class we will be seeing about the introduction and etiological factors of downer cow syndrome and in the second class we will be seeing about the clinical examination of the downer cow Syndrome. In the third session we'll be seeing about the metabolic downer cause like hypocalcaemia, hypokalaemia and hypophosphatemia in the fourth session we'll be seeing about the downer cows due to other causes and in the fifth session we'll be seeing about musculoskeletal disorders of downer cows and their management so let us see about in detail about the sessions in the first session we'll be seeing about the introduction and of the downer cow syndrome. So, as we all know treating a downer cow is a herculean task in the field conditions and unless otherwise you identify the etiological factors of the downer cow it will be very much difficult for identifying the downer cow etiology and the treatment aspect. Hence, we should know about the comprehensive knowledge about the downer cow syndrome and what are all the etiological factors involved in that so

that you can be able to easily diagnose the cases and treat it in the field conditions very easily.

So, I hope this session will be giving you an opportunity to learn more things about the downer cow syndrome and it will be helpful for your diagnosis and the treatment aspect in the field conditions. So, downer cow syndrome is otherwise also called as a recumbent cow. They used to call us in two ways they were either downer cow or recumbent cow. The cattle which are cattle or a dairy cow or a cow which is recommend for more than hours are unable to rise for more than hours after initial recumbency is called downer cow syndrome. So, usually the downer cows when they are going for recumbency they go for the secondary recumbency due to the pressure induced damage to the muscles and the nervous tissues. So, this is the etiological factor of the downer cow syndrome. Here let us see about the broad classification of downer cows the first classification we classify downer cows regarding the metabolic disorders and number two abdominal dysfunctions metabolic disorders means that includes hypocalcemia, hypokalemia and hypophosphatemia and abdominal dysfunction that includes traumatic reticuloperetonitis, traumatic Pericarditis, intestinal abstraction, Ileas, in such a conditions all this comes under the abdominal dysfunction, and infectious causes are there and that causes anemia, blood protozoan disease like anaplasma, tailarea, babysis. They cause anemia and the animal becomes downer cow, and also be some cases that will also causes downer cow. So, we have to identify according to the Etiology and intoxication cases like botulism organophosphorus spicing that also leads to recumbent cows and the other category is Musculoskeletal and nervous disorders when the animal is Recumbent. The animal may go for the secondary Recumbency, and you have to identify which nerve is affected and what is the musculoskeletal involvement involved so that our treatment schedule can be planned easily. So, the major elements involved in the recommend cows are the downer cows are the calcium, Phosphorus, Magnesium, Potassium, glucose, vitamin B, niacin and Cobalt. These are the major elements which are

involved in the downer cow syndrome or recumbent cows and in such a cases you have to identify which are all the components which are having a low level and we have to treat it accordingly. So, when you come when you see about the occurrence of the downer cows mostly, mostly the postpartum that undercover occurs mostly during the postpartum period and immediately after calving or during the time of parturition it occurs and the complication mostly the downer cows are the complications of hypercalcemia. We used to come across many conditions like complications of hypocalcaemia will be treating for hypercalcemia but unsuccessful treatment I mean after treating of hypocalcaemia or milk fever the animal cannot able to raise or getup that will lead to secondary Recumbency. So, these are the complications of hypocalcaemia and some of the downer cows during the pregnancy will come across during the pregnancy for example during the last trimester of pregnancy the animal may go for recumbency because of the lack of energy or negative energy balance or malnutrition. So, these are all occurs during the pregnancy period, and during the lactation time also due to the negative energy balance that during the lactation period also many downer cows usual occurrence is there. During the lactation period we have to whenever you come across any downer cow syndrome you have to think about the other causes of any abdominal dysfunction or infectious origin and intoxication that you have to rollouts, some other cows they suddenly they will fell down and they will get injured themselves and they will go for musculoskeletal Disorders. So sudden fell down and the prolonged recumbency that will lead to secondary recumbency and ischemic necrosis to the muscles in hind quarters. I mean when the animal is in recumbent so it will be there for a prolonged Recumbency. The animal will go for secondary recumbency because of this Ischemic necrosis of the muscles under peripheral nervous injury so the animal cannot be able to stand or get up. The pressure induced pressure injury to the superficial nerves for example radial nerve paralysis, peroneal paralysis because the animal is recumbent the pressure-induced nervous injury may lead to the recumbency of

the downer cows. And the pressure induced in the wall injury are vulnerable to obturator and sciatic nerve also so that you will be seeing in coming subsequent sessions in detail. It's a recumbent cows in pregnancy so you have to rule out the pregnancy status whenever you are getting a downer cow syndrome during the pregnancy period you have to verify the pregnancy status – whether the animal is full-time pregnant, or what is the age, determinate age of the pregnancy, whether there is any Hydroallantois is there or not. All these things you have to rule out and if the animal is frequently straining during pregnancy you have to rule out any torsion is present as there or not and you have to rule out any mummified fetus or macerated fetus. All these things you have to keep it in mind and go for the diagnostic evaluations, and sometimes in field conditions you will be coming across unusual downers they are heifers due to anemia, due to anemia the heifers are a calf they will be recumbent for more than weeks or days and you have to rule out the anemia and non-dewormed cases leads to anemia and recumbency in heifers. And sometimes Roman Impacts also leads to recumbent downer cows because of severe dehydration and all and over feeding orgrazing or carbohydrate encouragement like acidosis may also leads to downer cow syndrome and in ephemeral fever also you will be coming across this type of Recumbency. So, in the classification of the downer cows we used to classify them broadly as an alert and non-alert downer. Alert downers and non-alert downers. In alert downers all the feeding habits everything will be normal, the vital signs will be normal, temperature, everything will be in the normal range, heart rate, respiration rate, everything all the vitals will be in the normal Range. The animal will be passing the dung normally, urine will be nodded normally, but the animal cannot able to stand that is the alert downers. So, when you come to the animal may be having Rumination. Rumination may be there drinking water, voiding dung, voiding Urine, everything will be normal this comes under alerted downers. So, in alert downers will be the classification comes like metabolic disorders like calcium, Phosphorus, magnesium, potassium is involved I mean

hypocalcemia, hypokalemia, hypophosphatemia and hypermagnesemia. All this comes under alert downers and musculoskeletal involvement like a pelvic fracture, operator nerve paralysis, gastrocnemius muscle rupture, peroneal nerve paralysis, dislocation of the limbs, and fracture. In coming subsequent lessons, I will tell you in detail how to identify all these musculoskeletal Disorders, and all these nervous injuries. We can easily identify by seeing the posture of the animal. Innon-alert downer cows the animal will be in lateral recumbency, heart rate will be Elevated, respiratory distress may be there, and the dung will be voided appellate in nature or absence of dung will be there, complete absence of the dung or dung will be voided pellety in nature and the animal will be completely anorectic and the respiratory distress will be there. Animal may sometimes be unable to lift the head and the bloat or tympany will be there. All these things are the signs of non-alert downers. So you have to differentiate whether it is alert downer or non-alert downer. So that our treatment I mean the diagnostic aspect can be made very easy. So now I will show the video you can able to see the video of a non-alert downer. So, in this video you can able to see the non-alert downer which is dull and depressed in nature and the extended neck in neck is there. So, the animal is not voiding dung, respiratory disease will be there. The animal is dull and depressed and you can able to differentiate this is a non[1]alert downer. So in this video you can able to see the animal the head is turned towards the flank region and the heart rate will be elevated, respiration distress will be there, dung will be wider, little in nature and animal is completely an aerotic. The animal is placing the chin on the ground. This is all the indicative of non-alert Downers. So, in this first session we have seen about the introduction of the downer cow and also in detail about the etiological factors of the downer cow and also the various classification. How you can classify the downer cow Syndrome, and how you have classified the alert downers and non-alert downers. These are the things we have seen in the first session. In the second

lesson so we'll be seeing in detail about the clinical examination of the downer cows.

Thank you

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PDF: Downer Cow Syndrome

2 Clinical Examination of Downers

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Transcript

Dear veterinarians, I welcome you to the second lesson.

In the first lesson, the previous lesson we have seen about the introduction of the downer cows, and the various etiological factors of the downer cows, and also the classification of how you have to classify the alert and non-alert downers in the first lesson.

In this lesson we will be going in detail about the we are going to see about the clinical examination of the downer cows. So, for example when your downer cow is brought to you or when you are going to examine your downer cow, how to examine a clown downer. In detail about the clinical examination we are going to see in this lesson.

So, the clinical examination of the downers is an important part in the clinically I mean the examination of the downer cows. So first you have to see the temperature of the downer cow or whether the temperature is elevated or not whether it is a normal temperature or subnormal temperature you have to rule out. If it is having subnormal temperature you see whether the animal is having anaemia is there or not or septicaemia is developed or not that you have to Indicate. If the animal is having temperature then you have to think about any infectious origin or septicaemia. Then you have

to evaluate the heart rate of the downer cows, hear that heart rate will be elevated whether you have to see whether it is the normal range or it in the elevated range. If it is in the normal range it is ok if it is in the elevated range i mean tragic idea is that you have to think about any foreign body Syndrome, or any other septicaemia conditions you have to think about. So heart rate evaluation is very very mportant for example in case of acidosis when the animal is recumbent when you are seeing a heart rate if the heart rate is above 100 then you have to evaluate the prognosis. Accordingly, the heart rate will be very much useful in the diagnostic aspect, and you have to see the mucous membrane color whether it is normal, pale, pink in nature or whether it is anaemic in nature or whether it is a trich in nature, or whether it is congested in nature, or not. If it is trich in nature you think about blood protozoan diseases, if it is congested then you have to think about septicaemia. Then you have to see the mental status of the animal whether the animal is in the normal I mean whether any expression is abnormal expression is there or not, or the animal is dull and depressed or not, whether it is active and alert or not that you have to assess the mental status of the animal.

Suppose some animals may be recumbent, but it may show the frenzy behavior so in rabies or hyper magnesium tetany the animal may be excited so you have to see the mental status and the clinical examination and you have to evaluate whether the animal is taking feed or not, whether the animal is ruminating or not, if it is having rumination and taking feed and all it comes under the classification of alert Downer. And you have to evaluate whether the animal is passing dung or not if it is passing little quantity of the dung or nothing no dunk is voided then you have to think about the peritonitis or intestinal obstruction. If the animal is surpassing scanty faeces then you have to think about the foreign body syndrome, peritonitis or for stomach disorders like that you have to classify accordingly, and whether the animal is widening urine or not that you have to assess and the reflexes to the external stimuli for example when you are doing the pinprick stimuli then whether

the animal is responding to your pinprick stimuli is there or not that at all we have to assess.

And regarding the rumen consistency you have to see whether the rumen is hard in nature or if the rumen is having fluid splashing sound if there is fluid flashing sound and diarrhoea it indicates the acidosis, ruminal acidosis, and you have to see whether the animal is trying to get up or not. So in the some conditions when you are going and examining when you are giving some external stimuli the animal may be trying to get up and trying to get up this all the indicators of some science so that you can assess the musculoskeletal disorders whichever it is affected. And you have to see the reflex whether there is any menace reflex is there or not. Menace reflex palpable reflex all these things you have to evaluate during the clinical examination of the animal.

So in regarding the reflexes you can see the pinprick reflex, and menace reflex. Menace reflex means just you can have you take your fingers and just open it near the eyes so that whether the animal is blinking is there or not that you can see very well and the reflexes are the palpebral reflexes present or not or corneal reflexes present or not all these things you can Evaluate. And you have to evaluate the other examination is important sometimes in downer cows the animal may be having in mass studies. so, it may be because of mass status it could have developed a septicaemia it may be recumbent. So, you have to examine the whether the milk is normal in nature or watery in nature and you have to assess the color of the milk and the consistency of the milk. And you have to palpate the both the hind limbs and you have to see whether both the hind limbs are symmetrical in nature so whether it is having symmetrical nature or any other one limb is extended or not that you have to evaluate so that you can identify the dislocated limb very easily. And you have to evaluate the Achilles tendon I will show the pictures and do the rectal Examination. Rectal examination is very very important only then the clinical examination will be completed and see whether that is a dung material is present or not or complete absence of the dung so that you can able to assess your diagnosis. If there

is complete absence of the dung it indicates intestinal abstraction or iliac. If it is a scanty faeces it indicates for stomach Disorders and see whether the animal is in the Posture, posture of the animal whether the animal is in the sternal recumbency or whether the animal is the lateral Recumbency. so you have to assess the things and knuckling of the fetlock is will be that sometimes when you are able to lift the animal the animal may be may not able to bear the weight so knuckling of the fetlock will be there. That will be seeing in detail in the coming lesson the musculoskeletal disorder in detail. So here in this picture you can able to see the actually stand on so, you have to see whether the actually standard is very intact or very loose in nature. So you can palpate the actual standard very easily and you can assess whether it is intact in nature or flexible in nature. So in this picture you can able to see the hind limb. I am palpating the hind limb so just to see how i am palpating the hind limbs in one hand by one hand you keep your hand on the hip joint in another thing you keep your other hand on this stifle joint and see any crepitation is there or not this is one of the important clinical signs in the examination of the downer cow. See in this video you can able to see I'm examining the hind limb so I'm examining the hind limb and able to flex the hind limb and whether you are seeing any crepitation in the stifle is there or not and also the stifle the hind limb the stifle joint should not go for the 90 degree flux 90 degree angle deviation. So, in this case the 90 degree angle deviation is there so that indicates the stifle dislocation. So, this is one of the important clinical sign you have to do in all the cases whether there is any repetition in the stifle joint is there or not and the 90-degree angle deviation of the stifle joint is there or not so that you can assess. If the animal is a normal animal it won't come behind the 40-degree angle. So, if it is coming above 90 degrees then you have to think about stifle dislocation. So, these are all the important clinical examination that you have to keep it in mind during the examination of the downer cow and regarding the serum biochemical profile for examining the downer cow, you can collect

the serum and you can go for the evaluation of calcium, phosphorus, Potassium, magnesium.

All these things are very important in assessing the etiological factors of the downer cow and also, you can able to see the creatinine level creatine kinase not creatine it's a creatine kinase creatinine kinase can be given, and also AST is the important the prognostic indicator in case of downer Cow.

So, in this lesson we have seen about in detail about the clinical examination of the downer cow and in detail about the clinical examination of the stifle joint and hip joint in detail and regarding the biochemical parameters what are the biochemical parameters you have to collect in case of downer cow syndrome so in the next lesson third lesson we will be seeing about the downer cows due to metabolic disorders in detail

Thank you

Download

PDF: Clinical Examination of Downers

3 Downers due to Metabolic Syndrome

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Transcript

So dear veterans I welcome you all for this third lesson greetings to everyone. So, in the last class we have seen in detail about the clinical examination of the downer cow and also the biochemical, what are the biochemical parameters you have to Examine, I mean go you go for a review in case of downer cows.

In this we will be seeing about the downer cows due to metabolic disorders. So Downer cow due to metabolic disorders you can come across hypocalcemia, Hypophosphatemia, and hypokalemia, and hypomagnesemia. These are the four conditions will be coming across in the field conditions. For example the recently in milk fever in hypocalcemia it occurs within 72 hours of the calving. So, most of the time it occurs mostly around the 72 hours within calving period and the animal may be recumbent and the history will be very much obvious after calving animal will go for recumbency. In the early stage there will be shifting of the both the high limbs, the animal may be in restlessness and protrusion of the tongue will be there, then animal will be showing the teeth and the next the animal will be going for recumbency. So here and after recumbency the animal if it is not treated, if it is not attended, the animal may go for sternal recumbency and the head is turned towards the flank region and

the animal will be going for the third stage of milk fever. In the third stage of milk fever the animal will be in the lateral recumbency and in case of milk fever hypercalcemia the anal sprinters are very much relaxed and the corneal reflux is completely absent and the pupil will be dilated. So, these are all the symptoms we will be assessing in detail I mean very easy to diagnose the milk fever in field conditions and you will be treating the cases with Calcium Borogluconate. When you diagnose the case as a downer cow I mean the downer cow due to hypocalcemia you are giving Calcium Borogluconate half dose IV and half dose subcutaneous and immediately the animal responds well to the hypocalcemia, and the animal becomes normal.

And here we will see some of the science which are response to the Calcium. When you are giving calcium to the animal some response should be there and that is the animal belching. Belching will be there and the muscle tremors will be there and muscle tremors will be there the animal will void urine, the animal will void dung will be there and Slightly, there will be development, I mean increase in the amplitude of the, pulse will be there and increasing the intensity of the heart sounds will be there, and the sweating of the muscle will be there, and defecation will be there, the animal will void dung. These are all the positive signs of calcium. Even when you are giving calcium if you are not able to go for any response to the calcium the animal is not voiding Urine, the animal is not voiding dung, then you have to re-evaluate your diagnosis, then that is the case, not a case of Hypocalcemia. So, whenever you are administering calcium you have to keep it in mind some response to these were for calcium treatment the animal should void urine and they pass the dung. If it is not passing the dung or urine you have to re-evaluate your diagnosis and, if it is not responding to the calcium treatment it could be a case of Toxemia, it could be a case of Septicemia, it could be a case of Peritonitis, Metritis and Rumen Acidosis. All these things you have to evaluate by doing the clinical examination. For example, in acidosis you will be having fluid splashing sound in the rumen and the animal will be passing semi-solid dung so that you can easily identify the Rumen Acidosis.

And Relapses, in some cases the animal may get up again it will lie down and if you are giving calcium the animal may get up again it will lie down, or sometimes it may not get up so animal will be completely recumbent. Even after giving calcium that will be Recumbency means in the sternal Recumbent, always in the sternal recumbency. The animal may be taking feed and everything Normal, but they're not able to stand so you have to think about the two things one thing Hypophosphatemia and another thing is Sternal I mean Musculoskeletal disorders. So, you can apply you can in the relapses cases you can ask the owners to have a partial milking don't go for the complete milking and oral administration of the calcium can be given for the follow-up cases and Oral and subcutaneous administration of the calcium can be given for lapsing cases of hypocalcemia and re-evaluate the cases in some aged factor age factor also involved in the relapses of the downer cow due to Hypocalcemia.

In hypophosphatemia the animal will be everything will be normal it may respond to the calcium treatment one shot ways again it may go for downer I mean the recumbency it cannot be able to get up, all other signs will be normal, vitals will be normal, so in such a cases you have to go for supplementation of Phosphorus, oral phosphorus. So, we have the best thing is the supplementation of the oral phosphorus which is inorganic in nature. So you can supplement inorganic phosphorus so that it is more efficacy in case of ruminants that is sodium dihydrogen Orthophosphate. So, you can give sodium dihydrogen orthophosphate 100 to 150 gram orally so that will be very much effective for correcting the Hypophosphatemia. After supplementing sodium dihydrogen orthophosphate the phosphorous level will be elevated in the blood so that the animal become normal and just if you can manage the animal to just lift the animal and keep the animal in the sling or just if you are supporting the animal to get up it will get up from the it will recover well from hypophosphatemia.

And, here in this picture you can able to see the animal is in the Lateral Recumbency. Unsuccessful treatment you have treated

with all the calcium, phosphorus everything but the animal is not responding to your treatment. You think about hypokalemia. Hypokalemia means low potassium level in the blood. So here the animal is the lateral recumbency. When you are lifting the neck the neck muscle weakness will be there. The animal cannot able to lift the neck so, the animal may be in the lateral recumbency. See this is a picture of hypokalemia due to the lateral recumbency and here the animal is in the head is turned towards the flank. Even after treating with the calcium, phosphorus Everything, the animal is not responding to the treatment. Here the animal head is turned towards the flank region because of low level of potassium in the blood serum, that is hypokalemia. So in hypokalemia muscle weakness will be there, neck muscle weakness will be there. So that is a clear indication of hypokalemia. So here also you can able to see the lateral kink of the neck because of the muscular Weakness, the muscle weakness, the animal cannot be able to maintain the posture of the neck. So here you can supplement potassium chlorate syrup, for example in hypokalemia cases you can supplement potassium chloride powders. I mean potassium chloride is there you can supplement 100 gram orally in drinking water or you can supplement potassium chlorate syrup are available in the market that potassium chloride syrup you can give orally so that the animal will get will recover very eventfully. So, for example potassium chlorate are available for IV preparations also, ampules are Available. So, you can give this IV preparation 0.5 million percent body weight but the disadvantage in this iv preparation is that you have to take care very, you have to give it very very very slowly if you are giving very fast it will lead to arrhythmia the animal will collapse immediately. So, you have to take precautions during administration of potassium chloride in iv preparation. Normally we used to give IV preparation in the normal saline. So 0.5 milli equivalent per kg body weight you have to calculate and and it should be mixed with the normal saline not more than 20 to 30 ml in a normal saline sachet so that you can avoid untoward reactions. If you are not comfortable or not confident in the administration of potassiu m

chlorate IV better, you can go for oral preparation of potassium chloride powders or potassium chloride syrup that is the best thing and avoid IV preparations because it will lead to cardiac arrhythmia. So, you have to take precautions in this one. So here in this picture you can able to see the Oral rehydration therapy we are giving the electrolytes for the recumbent cow and the animal will become recover very eventfully.

So, in this lesson we have seen about the downer cows due to hypocalcemia and hypophosphatemia and hypokalemia in detail under the treatment of these Metabolic disorders. So, in the fourth lesson we will be seeing about the downer cows due to other etiological cases not we have seen about the metabolic in the fourth lesson we will be seeing the downer cows due to other etiological factors in detail.

Thank you!

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PDF: Downers Due to Metabolic Syndrome

4 Other Downers

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Transcript

Greetings to everyone so in the last lesson we have seen in detail about the metabolic disorders like hypocalcemia Hypophosphatemia, hypokalemia and how to identify and how to manage this type of Cases. in this lesson we will be seeing about the recumbent cows due to other etiological causes I mean downer due to other etiological causes. So, in this picture you can able to see the animal is recumbent and the animal is having you can able to easily appreciate the bilateral abdominal distension so the abdomen is bilateral they distension bilaterally and you have to think about whether the animal is pregnant or non-pregnant. So, if it is pregnant you have to think about Hydroallantois. So, you do the rectal examination you cannot able to palpate the fetus so it is a clear case of Hydroallantois so, you manage Accordingly. So, if it is a non-pregnant animal and if it is having bilateral abdominal distension then you think about retinitis so in this picture you can able to see the animal is having bilateral abdominal distension with the lateral kink of the neck, and lateral deviation of the head. Neck is there and it's a non[1]pregnant animal the dung is not voider so it is a clear case of peritonitis. So, I will show you in the coming lesson how to do the Abdominocentesis, identify the peritonitis. So, in this

picture you can able to see the bilateral abdominal distension and it's also a case of peritonitis, recumbent cow due to peritonitis.

Here you can able to see the picture of Abdominocentesis. It's a very important clinical examination the field condition to rule out peritonitis in cases of downer cows or whatever the standing animal or recumbent animal abdominocentesis is an important technique. Here you can simply see the video see this is the peritoneal fluid which is being collected from the abdominocentesis. See here the animal is the lateral recumbency from the xiphoid you go for the 5 centimeters 10 centimeters caudal 5 to 10 centimeters caudal and in the lower portion just apply the povidone iodine and insert the 18 gauge or 16 gauge needle so that you can able to clearly identify the Peritonitis. So, you can able to identify the free flow of peritoneal fluid from the abdomen so it is a clear case of Peritonitis. So, this cow is recumbent or downer due to Peritonitis. So, in such a cases the prognosis will be unfavorable but the diagnosis will be very easy for evaluating these cases.

So, in this picture you can able to see the bilateral abdominal distension is there on the rectal examination the animal is not the rectal examination there is no dung material in the rectum so it indicates that it's a case of Ileus or intestinal abstraction. These are all the recumbent cows due to ileus or international abstraction and is acute abdomen this is also having bilateral abdominal distension and you are doing the rectal examination and evaluating whether the dung is present or not if there is no dung or a complete absence of dung it is a case of intestinal abstraction and in this case also you do the abdominocentesis and rule out peritonitis.

So, in this picture you can able to see the prolapse of the rectum and this is a clear case of ileus or intestinal abstraction. So, the recumbent the animal is recumbent due to intestinal Abstraction, so you have to manage accordingly. Here you have to go for fluid therapy and you have to maintain the case and you treat it accordingly. So you have to go for intestinal abstraction means so you have to go for surgical interventions. Here also this picture shows the bilateral abdominal distension and the complete absence

of the dung and the rectal examination no dung material is present. So here also you can able to see the prolapse of the rectum in the case of ileus cases. These are all the downer cow due to ileus so, in the Rectal examination you can able to see the dung material whether it is scanty in nature or complete absence in nature. Scanty in nature indicates for stomach disorder, pellet dung indicates peritonitis and if it is a complete absence of the dung it is indicative of intestinal abstraction. So here you can able to appreciate in this picture the dark tarry colored dung is voided and it is indicative of Abomasal Ulcers This is a case of downer cow due to above Abomasal Ulcers. So you have to treat it accordingly for our Abomasal Ulcers. You have to go for pantoprazole iv preparations. Pantoprazole giving iv and fluid therapy and orally you can give magnesium oxide or magnesium carbonate 0.5 to 1 gram per kg body weight so that you can manage this type of cases, and this picture the animal is in the lateral recumbency and you can able to see the tail is away from the body and the animal is having a frequent pedalling will be there, frequent pedalling will be there here in this picture the tail is away from the body it is due to Botulism. Clostridium botulinum mostly the animals which are reared around the poultry farm area poultry farm premises most of the cases we use to get botulinum from the poultry farm area so because of the Clostridium botulinum because of the toxin produced, the preformed toxin is ingested and it causes placid paralysis. See here in this picture you can able to see because of the frequent pedalling you can able to see the semicircular drawing mark in the ground third is the classical sign of botulism. So, during the lateral recumbency the animal be having frequently having a pedalling of the limbs will be there and the semicircular drawing mark you can able to appreciate in case of botulism and also continuous salvation will be there, and the important thing is the abdominal respiration abdominal respiration is predominant.

So here you can able to appreciate the salivation in this picture because of botulism and the abdominal respiration is a predominant clinical sign for identifying the botulism cases, and some of the

downer cows are due to Mastitis. Gangrenous mastitis are equally form Mastitis. the animal may go for Septicaemia the animal may go for downer so you have to manage the dower cow because of Mastitis with the fluid therapy accordingly so that you can manage these type of cases.

So, in this lesson we have seen about the factors various conditions of a downer cow due to other etiological factors like Peritonitis, traumatic reticular Peritonealises, ileus, intestinal abstraction and botulism. so, in the next class we will be seeing about the downer cows because of due to the musculoskeletal involvement and the management of the downer cow syndrome.

Thank you

Download

PDF: Other Downer

5 Downers due to Musculo-skeletal and Nervous Disorders

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Transcript

So, greetings to every veterinarians. I welcome you all to this fifth lesson of downer cow due to musculoskeletal and nervous disorders. The previous lesson we have seen about the various etiological factors of I mean the downer cow due to other etiological factors like peritonitis and botulism, septicemia due to Mastitis and all.

In this lesson we will be seeing exclusively regarding the musculoskeletal nervous disorders. See in this picture so already we have seen the previous lessons like how to treat hypocalcemia, hypophosphatemia hypokalemia and all the other conditions. Already you have given calcium, Phosphorus, potassium, everything and all the animal recovered everything, but the animal is still in recumbency you cannot able to make the animal stand. So here what you have to do you have to make the animal lift the animal and stand make it stand for one time and see the posture so that you can easily identify the which nervous system, which nervous musculoskeletal disorder is there very easily you can identify. For example in this case in this picture you can see this animal the

animal is the left hand limb is abducted. The outward extension of the left hind limb is noticed. So you can easily identify this is a case of clear case of hip dislocation. By seeing this posture you can easily identify this is a case of hip dislocation. In the previous classes I have already told how to identify the stifle dislocation, if dislocation is by clinical examination you do the clinical examination and correlate and you can easily diagnose it. And here this picture you can able to see the outward extension of the hind limb is there so the right hand limb is here in this picture the right hand limb is affected and the abduction of the right hand limb by seeing this we can easily identify this is the stifle dislocation of the right hind limb. And here the stifle joint injury because of the stifle joint injury the animal is the lateral deviation of the left left hind limb and the abduction of the left hand limb is there so you do the clinical examination keep your hand on the stifle joint and flux it and see any crepitation is there or not and identify the stifle injury.

Here also the animal is having the abduction of the towards the right side so this is also a case of stifle injury. And this video you can able to see the stifle joint injury the animal is having the lateral deviation of the right hand limb and it's a clear indication so it's not a case of hypocalcemia or hypokalemia and All, it's a purely a case of stifle injury due to musculoskeletal disorder, and again I am showing this video in this lesson you can able to flux the hind limb you can able to flux a hind limb, I mean the left hand limb is affected and when you lift to the hind limb it should not go for 90 degree angle deviation. If it is going for 90 degree angle deviation it is a clear case of stifle dislocation. So very easily you can diagnose and you can say the prognosis for these type of Cases. This picture you can able to see the both the hind limbs are extended towards the elbow, so in this animal both the hind limbs are extended towards the elbow it is a clear case of Obturator Nerve Paralysis. Obturator Nerve Paralysis usually occurs during the time of Calving. prolonged calving period or a very big fetus may injure the Obturator Nerve and it will lead to this type of Posture. Animal won't get up irrespective of all the treatment the animal won't respond to any type of treatment. Both

the hind limbs will be extended towards the elbow region, it's a clear case of obturator Nerve Paralysis. See in this picture also you can able to identify the both the hand limbs extended towards the elbow region.

And this picture you can able to see both the hand limbs are extended outward and unable to abduct so, it's a clear case of obturator nerve paralysis. In abductor muscle paralysis also this type of clinical science will be noticed. And when you are lifting the animal and keeping the animal in the sling both the abduction of the both the hand limbs will be noticed in case of obturator Nerve Paralysis the both the high limbs normally both hind limbs should be very it should be within a small gap should be there. If there is abduction of both the hind limbs that indicates after obturator Nerve Paralysis. so it will take a long time you have to keep the animal in the sling and go for the physiotherapy slowly the animal will recover in such a cases. Here in this case you can able to see there is a huge swelling in the thigh region and when you make the animal lift the animal cannot be able to bear the weight on the fetlock or the foot the animal is bearing the weight on the hog joint the hog giant resting on the ground is a clear indication of Gastrocnemius Muscle Rapture. So, you can tell the prognosis when you lift the animal the animal will bear the weight on the hog joint, the hog joint touching the ground or hog joint resting on the ground is a clear case of gastrocnemius muscle rupture.

And this picture also can you can able to see the animal in the standing posture, but the hog joint is resting on the ground so it is a clear case of gastrocnemius muscle rupture. So here in this picture you can able to see the hog joint is dropped, Dropped Hock with the Partial Knuckling of the Fetlock. So here the Dropped Hock is that I mean the hog joint is dropped downward and the knuckling of the fetlock is there that indicates Sciatic Nerve Paralysis indicates of Sciatic nerve paralysis and in this picture the animal is put on the sling and the animal is put on the sling and the hock joint is dropped downwards so it is indication of Sciatic nerve injury and you can able to see the knuckling of the fetlock is there that's a clear

indication of Peroneal Nerve Paralysis. So, in this case it is affecting it is having Sciatic nerve injury with the peroneal nerve paralysis. So, you can manage accordingly you have to keep the animal the sling and go for physiotheropical treatment. And here in the animal is put in the sling and you can able to see the dropped elbow I mean the droopiness of the droopiness of the four limb is there, dragging of the forelimb is there, the elbow is dropped, that is a clear indication of Radial Paralysis, it's a class of radial paralysis. See when the animal is recumbent the forelimb is extended forward so that indicates, this posture indicates of Radial nerve paralysis.

See in this picture also the extended forelimb in this Sternal Recumbency indicative of radial nerve paralysis. All these things are muscular skeletal Disorder. You have to keep the animal in the sling and you have to manage in the Sling, sling management is very very important. See in this picture there is a knuckling of the partial knuckling of the fetlock complete knuckling of the fetlock indicator of peroneal nerve paralysis. Partial knuckling of the fetlock indicator of tibial nerve injury.

See in this picture you can able to see the partial knuckling of the fetlock indicator of tibial nerve injury. Here complete knuckling of the fetlock is noticed indicative of peroneal nerve Paralysis. So these are all the musculoskeletal disorders are nervous involvement of all these downer cows so once you lift the animal in the sling and you can easily identify the posture you can manage accordingly very easily instead of going for poly pharmacy.

So here management regarding the management of downer cow you have to keep the animal in the Sandpit or floor or bedding for the Treatment, don't keep the downer cow in the concrete floor and treat it it will be a failure one. So keep the animal in the sand pit or the bedding should be provided and treat the animal accordingly, and assisted lifting should be there assisted lifting should be there with the slings and the turn the animal side by side so every once or two hours three hours you have to turn this animal this side and that side so that the muscular ischemia, muscle ischemia and ischemic necrosis can be prevented, and the wound if there is any

wound wound care management should be there and monitor the order of the downer cow daily whether it has developed Mastitis is there or not, and the formentations can be given hot formentation can be given for the affected limb, and treatment regarding the treatment we have discussed in detail in the previous lessons for example hypocalcemia you are giving calcium, for hypokalemia you are giving potassium, for hypophosphatemia you are giving Potassium, I mean hypophosphatemia you are giving phosphorus, oral supplementations you are giving and electrolyte supplementations, fluid Therapy. All these things are manageable for the downer cow syndromes and you can also go for vitamin b complex supplementations for the management of downer cow syndrome. So here you have to make the animal Lifting, lifting is very important after the treatment the animal is active and alert taking feed everything normal so you have to keep the animal you have to make the animal lift by manually and if possible you can keep the animal the sling you can able to keep the animal the sling and you go for the physiotherapy in the management of the downer cow after lifting. So, this is the movable sling so you can make the animal stand in the sling and you can see the identify the posture and manage it accordingly. We have seen in detail about the musculoskeletal and nervous disorders of various affections of downer cows in detail, and the management of the downer cows. So greetings to all the veterinarians so in this lesson of downer cow syndrome you have seen comprehensive knowledge regarding the various etiological factors. So, in the lesson previous lessons we have seen about the various classification of downer cows' course like metabolic disorder, abdominal Dysfunction, infectious causes, Intoxication, musculoskeletal disorders, and all these things we have seen in detail. We are also seen in detail about the alert and non[1]alert downers. How we have to identify alert and non-alert downers and also later we have seen about the clinical examination of the downer cow. When the animal is brought to you how we have seen how to examine it clinically in detail we have seen and, also we have seen about the metabolic disorders of downer cows

like Hypocalcemia, hypophosphatemia, hypokalemia in detail under their Management, and also we have seen the downer cow due to abdominal dysfunction like peritonitis, intestinal Abstraction, ileus and hydraulics all these conditions we have seen. And also we have seen about the botulism in infectious causes we have seen about the botulism and regarding the other etiological causes of musculoskeletal disorders we have seen about the peroneal nerve paralysis, shear technology paralysis tibial Nerve paralysis and stifle dislocation and stifle injury in detail we have exam.

We have seen all these conditions and also the management of downer cows and how to put the animal in the sling management. So I hope this all this lesson would have given you the comprehensive knowledge about identifying the etiological factors of downer cow syndrome

Thank you

PART III

WEEK 3: KETOSIS IN RUMINANTS

1 Definition, Importance of Ketosis, Etiology of Ketosis

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://opentextbooks.colvee.org/metabolicandproductiondisorderscattle/?p=82#oembed-1

Transcript

I welcome all the practicing Vets for this class on Ketosis.

As you all know, Ketosis is an important production disease, which more commonly occurs in the immediate postpartum period. It is a recent trend in selecting the dairy cows genetically for higher milk yield results in Ketosis.

So, Ketosis has adverse effects or it can have a direct and indirect effect on the economy of the animal. Directly it affects the milk yield and also the production of the animal and in turn, it also affects the health of the animal. So, the cattle are exposed to or are predisposed to the other metabolic disorders in the periparturient period and indirectly or the latent effect of this Ketosis especially the sub-clinical Ketosis is, that it will make the animal succumb to cystic ovaries, there will be an increase in the calving to the first service, increase in the interval between calving to the first service and increase in the interval between calving to the last service. And all the other reproductive failures will occur because of subclinical ketosis.

Now we are going to see about Ketosis in five classes.

Lesson-1; will deal with the Definition and the Importance of Ketosis and its Etiology,

Lesson-2; the second class is about the Etiology and also the Epidemiology of both the Ovine and Bovine Ketosis,

Lesson-3; third class is about the Pathophysiology of Bovine or Ovine Ketosis, and

Lesson-4; fourth class is regarding the Clinical Signs, Clinical Pathology, the Diagnosis and Differential Diagnosis of both Bovine and Ovine Ketosis and

Lesson-5; fifth class we are going to deal with the Treatment Management and Control of the Bovine and Ovine Ketosis.

Let's start with the topic. So, in first class, we will see about:

The **Definition of Ketosis**: Ketosis is defined as a multifactorial disorder of energy balance, it is a basic metabolic disturbance resulting from negative energy balance during early lactation reduction in blood glucose and also the liver glucose level, and an increased fat mobilization that results in elevated Ketone body concentration. It is characterized by abnormally elevated concentrations of Ketone bodies and Hypoglycemia. So, Ketone bodies include acetoacetic acid, beta-hydroxybutyrate, and acetone.

Next, we'll see something about the **Importance of Ketosis**: why ketosis is so important as a production disease. See nowadays in modern agriculture practice animals are selected for the increase in milk yield. Genetically animals are selected for increased milk yield, so this increase in milk yield exceeds the capacity of the animal to ingest sufficient feed to meet the requirements for energy, especially during lactation. So, the net result is the negative energy balance, this results in the negative to offset the negative energy balance, mobilization of the fat and protein occurs, this results to the that is, it is transported as triglyceride and amino acids to the liver, where it undergoes the gluconeogenesis and ketogenesis for the production of energy. Always in the lactating animal, there will be a certain degree of Ketosis. Cows' pregnancy undergoes a partition of nutrients during pregnancy and lactation and is always

in a lipolytic stage in early lactation and or at a higher risk for Ketosis during this early lactation period, that is immediately after postpartum.

When does ketosis become a disease condition? It is when the absorption and the production of Ketone bodies exceed their use by the ruminants as an energy source, that results in an elevated blood Ketone, which is free or non-esterified fatty acids.

So, coming to Etiology, that is **Etiology of Bovine Ketosis**, before going deep into the Etiology of Bovine Ketosis, understanding why the Ketone bodies are formed and how the Ketone bodies are formed are essential. This understanding of the glucose metabolism, energy metabolism in the case of animals, the role of insulin and glucagon, ketone body formation, and how this ketone body formation is going to affect the Hepatic insufficiency or it leading to the Hepatic insufficiency in cattle is more important.

So how glucose metabolism is more important in the case of cattle. So, maintenance of adequate concentration of glucose in the plasma is always essential for the regulation of the energy metabolism. In the case of ruminants, they will absorb very little hexose sugar as carbohydrates, the remaining carbohydrates are fermented in the rumen into short-chain fatty volatile fatty acids like acetate, propionate, and butyrate. In this acetate and butyrate are ketogenic and the propionate is glycogenic.

Propionate and amino acids are the important precursors for gluconeogenesis, whereas the glycerol and lactate are less important for gluconeogenesis. So, the propionate that is formed is its most important glucose precursor and it is produced in the rumen from starch fiber and proteins. So, this enters the portal circulation and reaches the liver and it is efficiently removed by the liver. And the production of the propionate is increased, or it is favored by the inclusion of the starch in the diet.

The amino acids: coming to amino acids the importance of amino acids is it is gluconeogenic or it's a glucogenic factor and it is an important precursor for gluconeogenesis you all know dietary protein is an important source or it is the most important

quantitative source for the amino acids and the second source is the Labile pool of body protein, that is particularly of the skeletal muscles, is also an important source. This contributes to energy synthesis, milk protein synthesis, and milk lactone synthesis.

So, coming to energy metabolism; see all high producing dairy cows will always be in a negative energy balance, especially during the first few weeks of lactation. This is because the high dry matter intake does not occur until 8 -10 weeks after calving, but the animal will go for peak lactation within 4-6 weeks. Hence the energy intake does not keep up with the production, so the net result is the animal will go to a negative energy balance and there will be low serum glucose and low serum insulin will be there. So, this will lead to the or will make the cow mobilize the adipose tissues in the form of, that is present the form of triglycerides, with a subsequent increase in the non-esterified fatty acids. So, this subsequent increase in the serum concentration of leads to the mobilization of the fat as free fatty acids and non-estrogen fatty acids to the liver will increase the serum concentration of the ketone bodies like beta-hydroxybutyrate, acetoacetate, and acetone. So the Hepatic mitochondrial metabolism of fatty acids promotes both glyconeogenesis and also ketogenesis.

How Ketone bodies are formed?

So there are two main sources for the Ketone bodies production in the ruminants.

1. One is Butyrate in the Rumen: this is mainly produced by the rumen fermentation of the diet, where it is converted into beta-hydroxybutyrate in the ruminal epithelium and is absorbed as such.
2. whereas the second one is the Mobilization of the fat: where mobilized fat will be transported to the liver and will be oxidized to acetyl CoA and nicotinamide adenosine dinucleotide plus hydrogen ion (NADH). so, what happens to the acetyl-CoA that is formed it has to be oxidized via the TCA cycle, so for this oxidation auxiliary state is required supply of

oxaloacetate is required for which the precursor propionate is required as I told the carbohydrates are converted into short-chain fatty acids like acetate, butyrate, and propionate. The propionate plays a major role, or the importance of the propionate lies over here, where it is important for the supply of oxaloacetate. Once this oxaloacetate is propionate is decreased, in turn, the auxiliary state is going to be decreased, so this will affect the TCA cycle, that is acetyl-CoA oxidation in the TCA cycle. So, the next is what happens is this acetyl CoA will be metabolized to acetyl-CoA where again it will be converted into acetoacetate and beta-hydroxybutyrate thereby leading to Ketone body formation. And in the case of cattle any animal that is in recent calving or the immediate postpartum period, some degree of hepatic insufficiency or some degree of fatty liver is more common. So, uptake of fatty acids by the liver leads to the fatty liver, which will lead to hepatic insufficiency. See as I told you any animal that is in the immediate postpartum period will have a certain degree of negative energy balance, okay so the degree of the negative energy balance usually depends upon the level of milk yield high production or it is a medium producing animal. So, this depends upon the production level or how much milk yield is there in each animal. So, what happens is there will be an uptake of fatty acid by the liver which leads to the formation of fatty liver. There will be a certain degree of hepatic insufficiency, so this hepatic insufficiency occurs in cows that are pre-disposed to ketosis, by over-feeding, especially during the dry period.

So based on the hepatic insufficiency presence of hepatic insufficiency ketosis can be divided into:

Type 1

Type 2 and

Type 3.

So in the case of Type 1 ketosis what they say is it is spontaneous

ketosis. Here what happens is gluconeogenesis pathways are maximally stimulated, so what happens is whenever there is a negative energy balance or this negative energy balance is offset by the mobilization of the fat, ketosis occurs when the demand for the glucose increases or outstrips the capacity of the liver to go for gluconeogenesis due to insufficient glucose precursor. So as I told you there will be a decrease or there will be a deficiency of propionate leading to the deficiency of the oxaloacetate, so the acetyl-CoA oxidation via the TCA cycle will be impacted, this will lead to the mobilization so there will be a decrease in the energy level, there will be a rapid entry of NEFA into the hepatic mitochondria resulting in a higher rate of the ketogenesis. So there will be high plasma or serum ketone concentration will be there, see whatever the NEFA that is remaining that is a free fatty acid so the non-esterified fatty acids will be again converted into triglyceride and it will be stored in the liver or it will be converted as fatty liver.

So, in Type 1 ketosis, there will be a little conversion of the NEFAs to triglycerides hence little fat accumulation in the liver.

Whereas in the case of Type 2 Ketosis: it is also called a fatty liver, the gluconeogenic pathway. You have to understand the difference between Type 1 and Type 2:

In the case of Type 1, gluconeogenic pathways are maximally stimulated, so what happens is whatever the NEFA and the free fatty acids, that are mobilized to the liver will be resynthesized as triglycerides and it will be used for the ketone body production, whatever the remaining NEFA and this thing will be stored as the fat in the liver crossing the fatty liver, but the degree of fat deposition differs in Type 1 and Type 2 whereas the Type 2 is truly called as a fatty liver.

In this **Type 2 Ketosis**, gluconeogenic pathways are not maximally stimulated, so the NEFA obtained by the cytosol is not active, NEFA becomes esterified in the cytosol forming triglyceride. So, the capacity of the cattle to transport triglyceride from the liver is low, this will result in the accumulation of the triglyceride in the liver causing fatty liver. So, the animal will go for fatty liver, this fatty liver

will further depress the gluconeogenesis and excess butyrate the already present glucose decrease in the gluconeogenesis.

Type 3 Ketosis: here this is because the animals are due to feeding the animal with a high butyrate-producing diet, especially maize.

Coming to the **Role of insulin and glucose:** see insulin and glucagon, these two hormones are more important to regulate the energy metabolism in ruminants. Both the hormone has got a counteracting effect. The counteracting effects play a major role in maintaining the homeostatic control of glucose metabolism. Low insulin glucagon ratio will stimulate it will be there in the immediate postpartum period or lactating animals this will stimulate the lipolysis from the adipose tissues and result in the ketogenesis in the liver.

Cows in early lactation will have low insulin and low glucagon ratio.

There will be an elevated ketone body or the presence of the elevated ketone body will stimulate insulin production and it will have a negative feedback effect, and regulation is also indirectly governed by the hormone Somatotrophin. Somatotrophin is again lipolytic and it is also an important determinant of milk yield in ruminants.

So the Importance of Ketosis is mainly due to the presence of the subclinical ketosis it not only causes affects the production of the animal and also the reproduction, but it will not allow the animal to respond to the underlying disease or respond to the treatment given to the primary diseases, thereby decreasing the production of the animal.

So **Subclinical Ketosis**: In this subclinical ketosis, there will be an elevated concentration of blood ketones without clinical disease and this is more common than the clinical ketosis, it causes a significant economic loss to the farmer and it is common in high producing dairy cows, especially during 2-7 weeks postpartum period and small, see what happens is the animal will be in subclinical ketosis, so any minor nutritional insult diffusion

deficiency or any metabolic insult is going to be there this will lead to the development of the **Clinical Ketosis**.

We have seen the Definition of Ketosis, and also the Importance of Ketosis in ruminants and followed by the Etiology, and in that Etiology how the ketone bodies are produced and why the ketone bodies are produced, and how it is going to affect the health of the animal by going for the Sub-clinical ketosis and Clinical ketosis, and

Next class, I will talk about the remaining Etiology and Pathophysiology.

Thank you!

Download

PDF: Defenition, Importance of Ketosis, Etiology of Ketosis

2 Types of Ketosis, Etiology of Bovine and Ovine Ketosis, Epidemiology of Bovine and Ovine Ketosis

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Transcript

In the first class on Ketosis, we have seen the Definition of Ketosis, the Importance of Ketosis in dairy farming, and also the Etiology- How the ketone bodies are formed, and Why the Ketone bodies are formed. How subclinical ketosis is present? How does it become a Clinical Ketosis? Now in the second class, we will see about the Etiology, Epidemiology, and Different types of Ketosis of Bovine and Ovine Ketosis right. So here we have got five different types of Ketosis which are primary Ketosis, also called the production ketosis. This is the ketosis of most herds and it is also called the estate acetonemia.

Primary ketosis occurs in cows that are in very good condition or a very good body condition scoring and it has a high lactation potential so a greater proportion occurs, as a case of seeing in the case of primary ketosis, mostly there will be subclinical ketosis there is an increased concentration of circulating ketone bodies, but the animal will not show it clinically, so affected cattle will recover with the corrected feeding and ancillary treatment.

In the case of **Secondary ketosis**: secondary ketosis is mainly due to the presence of the other diseases, metabolic disorders, or any diseases that are present in the immediate postpartum period, which will affect the feeding of the animal, which is causing anorexia, in such cases, the secondary ketosis will develop. This may be due to Traumatic reticulitis, Herd with a higher incidence of fluorosis, and other diseases in the postpartum period like Mastitis, Metritis, and Abomasal Displacements. Other than this once the animal is going to go for calving in the immediate postpartum period, there will be a decrease in the immunity of the animal, which is making the animal be prone to other disorders or other diseases, that will cause an increase in the Secondary ketosis.

Alimentary Ketosis: This is mainly due to as I told you in the case of Type 3 ketosis, feeding the high butyrate silage. It is commonly subclinical in nature and it predisposes the development of the production of primary ketosis.

Starvation Ketosis: see ketosis will not only occur in animals that are well fed or over-conditioned body, it will also be formed in the animals that are starved or undernourished. okay, So cattle with poor body conditions and also that are fed with poor quality diet or poor quality feedstuffs and the animals that are deficient in propionate and protein from the diet. So if the starch, as I told you propionate is formed from the fermentation of the starch in the rumen, if the starch quality is not good or if it is below the quality, so what happens is the propionate formation will be affected, which in turn is going to cause the ketosis.

And limited capacity of the gluconeogenesis from body reserves, see whenever the animal is not having sufficient energy or if the animal is in negative energy balance, what happens is the body reserves will be mobilized or whatever the body reserves are there that fat will be mobilized but in case of starved animal or if the body condition of the animal is thin or it is undernourished what happens is there will not be sufficient body fat to mobilize for ketogenesis so in such animals the Starvation ketosis is more common and the affected cattle will recover with corrected feeding. okay and

Another type of ketosis here is ketosis resulting from the Deficiency of specific nutrients, as I told you, see in the case of the TCA cycle- Cobalt and Vitamin B12, play a major role in the form of coenzyme for oxidation of the acetyl-CoA in the TCA cycle. Suppose if there is any deficiency of this vitamin B12 or Cobalt, this will end up in Ketosis, so reduction in the intake of total digestible nutrients and if there is any Phosphorus deficiency is there, any Cobalt deficiency is there or the failure of the animal to metabolize propionic acid so as I told so if there is any Cobalt deficiency of Vitamin B12 deficiency, there will be a failure of the metabolism of propionic acid in the TCA cycle, this will result in Ketosis.

So coming to in nutshell, what is the **Etiology of Bovine Ketosis,** we will see about the in nutshell about the etiology of Bovine ketosis and Ovine Ketosis.

Bovine ketosis: it is common in the case of heavily producing cows and ruminants are prone to this because very few carbohydrates are observed as such and it requires a direct supply of glucose is essential for tissue metabolism, particularly for the formation of lactose. So whatever the glucose is absorbed is very little, so the animal has to utilize the volatile fatty acids for energy production in the liver and it is also dependent upon the available glucose.

So ketosis is more common in the immediate postpartum period, so the period between calving and peak lactation has a demand for glucose and there will be an increased demand for glucose. Low blood glucose levels will lead to low blood insulin and so the long-chain fatty acids will be released from the body stores under the influence of low insulin, this insulin glucose ratio and also because of the presence of high somatotrophin, there will be ketogenesis. Etiology of Ovine ketosis so coming to:

Ovine Ketosis: see Bovine ketosis and Ovine ketosis is not similar. okay, I'll tell you about the biochemical differences between Ovine Ketosis and Bovine Ketosis in the subsequent classes. Here in the case of Etiology of Ovine ketosis includes, if there is a decline in the plane of nutrition, especially during the last two weeks of pregnancy particularly when the animals are carrying twins or triplets and it

will also be seen in the case of animals that are well fed in the early or in the mid[1]pregnancy. Biochemical differences and elevation of the plasma cortisol level and a significant hepatic dysfunction will be there.

Here we can divide it into :

- Primary pregnancy toxemia and
- Secondary pregnancy toxemia

Primary pregnancy toxemia is due to the fall in the plane of the nutrition, especially during the latter half of pregnancy and there will see if the animal is in a shorter period of fasting especially when the animal is subjected to the managemental procedures like shearing and drenching and other management of procedures, this will aggravate the deficiency leading to Ketosis in Ovine and another one is that is going to cause the Ovine Ketosis is an accustomed diet or cold inclement weather. In the case of Ovines, fat ewe pregnancy is mainly due to the overfat condition in late pregnancy, this is mainly because there will be a reduction in the rumen size due to the increase in the pressure from the fetus and the abdomen fat and

the second one is Starvation pregnancy ketosis, this is more common in the case of Ovines that are reared in an extensive grazing system. See in such a condition what happens whenever there is going to be a prolonged drought condition is there and if there is no alternate feed supplement or feed supply available then it will lead to Starvation Ovine Ketosis and secondary pregnancy toxemia, this is mainly because of the presence of other intercurrent diseases like foot rot, worm infestation, and other sporadic diseases.

So coming to **Epidemiology**: How Epidemiology will give you at what stage, at what postpartum period, the Clinical ketosis is more common, and at what parity the Clinical ketosis is more common than all these details will be given by Epidemiology. So Clinical ketosis will increase with parity, especially peaking at the fifth to sixth lactation and it is also important ketosis is more common in

overfeeding, especially during the late lactation period. So definitely it will predispose the animal to ketosis in the next lactation. And the ketosis is more common during the first month of the lactation and less common in the second month and also it is common in diets that are less than 8% protein before calving or the diets that are having more protein that is more than 20% dry matter after calving and 30-40% of the cases is usually complicated by the concurrent diseases such as Metritis, TRP, Abomasal Displacement. As I told you, this will lead to Secondary Ketosis because the animal will not sufficiently take food because of the primary disease. So this will lead to anorexia and the patient will go for Secondary Ketosis another one is in the case I told the animals with Subclinical Ketosis will have a reproductive failure that is mainly because the animal will not show any Clinical signs, Subclinical Ketosis will not show any clinical signs, in such animals cystic ovarian condition increased, calving to first service period or increased calving to last service period, is more commonly same.

In the case of **Pregnancy Toxemia**: it is primarily a disease of the intensive farming system it occurs only in cases of easts that are in the last six weeks of pregnancy and it is usually during the last month of pregnancy is carrying twins or triplets are more prone for this disease and also the presence of other intercurrent diseases in late pregnancy will cause the Ovine Ketosis.

So in summary in the second class, we have seen something about :

- the remaining Etiology and
- also the Epidemiology.
- How and When the ketosis will occur both in the case of Ovine and also the Bovine Ketosis. And the
- remaining Pathophysiology
- Clinical findings and
- Treatment

we will see in the next subsequent classes.

Download

PDF: Types of Ketosis, Etiology of Bovine and Ovine Ketosis, Epidemiology of Bovine and Ovine Ketosis

3 Pathogenesis of Bovine and Ovine Ketosis

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Transcript

So, in the second class, we have seen about the Etiology and also the Epidemiology. How and When Clinical Ketosis and Subclinical Ketosis will occur in the case of Bovine Ketosis and in the case of ovine Ketosis.

Now we will see something regarding the Pathophysiology. How Physiology becomes Pathology due to the presence of Ketosis. Whenever there is a demand for glucose there will be fat mobilization, this will lead to the Clinical ketosis in ruminants, especially during the early post partisan period.

In the case of ruminants, the energy is, or the energy requirement is met by the production of volatile fatty acids in the rumen. So, in the case of ruminants, very few carbohydrates are absorbed as hexose sugars and the remaining carbohydrates will be converted into or it will be fermented into short-chain volatile fatty acids like Acetate, Butyrate, and Propionate. In this Acetate, 70% of the acetate will be produced and this is mainly used for fat synthesis, and it is ketogenic whereas Butyrate again 20% it is produced it is again ketogenic this will be condensed into an acetyl-CoA, this will be either oxidized to ketone bodies or it will be transformed into acetyl- CoA and this acetyl-CoA will enter the TCA cycle for

oxidation and the remaining Propionate that is only 10% is produced that is glycogenic, here it enters the TCA cycle at the level of the succinyl CoA. So, what happens is whatever the acetyl-CoA, I told in the previous slide whatever the acetyl- CoA that is formed it has to get oxidized via the TCA cycle, and this oxidation depends upon the adequate supply of oxaloacetate for which propionate is the main precursor for the production of the oxaloacetate. Once propionate is deficient, oxaloacetate will be insufficient this will make which will affect the oxidation of the TCA that is acetyl-CoA via the TCA cycle. So the acetyl[1]CoA will be diverted to the formation of ketone bodies, so to increase the gluconeogenesis and offset so this will result in, see this acetyl-CoA which is diverted to the formation of ketone bodies, so there will be insufficient glucose so the animal will enter into the state of negative energy balance and to offset this negative energy balance, more triglycerides will be mobilized from the adipose tissues as NEFA and free fatty acids to the liver. Once it enters the liver this will be converted again into triglycerides or triglycerides will be resynthesized from these free fatty acids and NEFA and the ketone bodies will be produced and in the liver triglycerides are resynthesized from the free fatty acids and will be stored by the liver or it will be exported as very-low-density lipoproteins.

But whereas in the case of Ruminants, Ruminants have inherently low capacity to export this VLDL, so what happens is excess of the free fatty acids or the NEFA that is that attempt to enter the TCA cycle through the acetyl-CoA in the absence of sufficient oxaloacetate, is partially oxidized to acetoacetyl-CoA which allows the formation of Ketone bodies and so the negative energy balance that occurs in the postpartum period, further reduces the available carbohydrates and this accelerates the further mobilization of the body fat and further production of the ketone body.

So all these together will cause Ketonemia, Ketonuria, Ketolactia, Hypoglycemia, and lower levels of the Hepatic glycogen.

In case how/ what is the path of Physiology in case of Ovine Ketosis? So there is an inadequate energy intake in the late

pregnancy, which will lead to ineffective gluconeogenesis, so there will be hypoglycemia and lipid mobilization this will lead to the accumulation of ketone bodies and there will be an increase in the Cortisol level.

Bovine Ketosis: the principal metabolic disturbance will be Hypoglycemia and Ketonemia and the nervous signs that occur in the case of Bovine ketosis are mainly due to the production of Isopropyl alcohol which is a breakdown product of acetoacetic acid in the rumen and secondly because of the presence of Hypoglycemia, which is more important for the nervous function. And Ovine Ketosis: In the case of Ovine Ketosis Encephalopathy is mainly due to Hypoglycemia in the early stage of the disease. Once the Encephalopathy comes the disease is not reversible unless it is treated in the early stage. A high level of Cortisol and adrenal steroid diabetes contributes to the pathogenesis so adrenal cortical diabetes plays a major role by increasing the cortisol and it causes the remaining clinical signs or it is the cause of the clinical signs like Encephalopathy and other signs in Ovine ketosis.

To summarize this class, we have seen the Pathophysiology, how normal Physiology has become a Pathology because of the Ketosis, and How and why do those individual signs develop in Ketosis, especially in the case of Ovine Ketosis and in the case of Bovine Ketosis.

So, the factors that are causing the clinical signs in the case of Bovine and Ovine Ketosis have been seen. We have seen the cause for those things, and

Next class, we will see about the Clinical Findings and Treatment.

Thank you!

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PDF: Pathogenesis of Bovine and Ovine Ketosis

4 Clinical Findings and Clinical Pathology of Bovine and Ovine Ketosis

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Transcript

Last class we have seen about the Pathogenesis of the Ketosis and How each Clinical Sign develops or Why Clinical signs like the Nervous form of Ketosis and other Encephalopathy develop in the case of Ovine Ketosis, everything has been seen.

In this class, we will see about the Clinical findings, Clinical pathology, Diagnosis, and Differential Diagnosis of Bovine and Ovine Ketosis.

So, coming to Clinical findings, we have got two forms of Bovine Ketosis:

- one is the Wasting form
- another one is the Nervous form

In the case of Wasting form: this is the most common form of ketosis in bovines:

- there will be a decrease in the appetite
- there will also be a decrease in the milk yield over 2-3 days

- cows will refuse to eat grain but continue to eat hay, that is why the owners will tell its roughage is taken normally whereas the animal is not eating grains
- This is the commonest complaint we get from the owners.
- So, and there will be a rapid loss of body weight, which is why it is called a Wasting form
- because of the loss of the subcutaneous fat, the animal will go for a woody appearance
- there will be a disappearance of the subcutaneous fat and there will be firm and dry feces
- The animal will be depressed
- it will have a disinclination to eat and move
- there will be a fruity odor from the breath, that is the sweet odor in-breath and

Nervous form of the ketosis: Nervous form of ketosis:

- the animal will be in a stage of delirium rather than frenzy
- the animal will be walking in circles crossing of legs which will be the first sign seen in case of the Nervous form of ketosis, once the animal starts developing Hypoglycemia, the owner will complain that the crossing of limbs is seen like that
- there will be head pushing
- apparent blindness is also more common
- aimless wandering and movement of the animal are seen
- vigorous licking of the skin is one of the most important Clinical signs in case of Nervous form of ketosis
- Chewing movement and salivation
- Hyperesthesia will be there
- Bellowing
- Tremor and Tetany: tremor or tetany will be there most often these nervous form of ketosis resembles that of the Rabies in the case of bovines.

Pregnancy Toxemia: in ovine says:

- separation from the group
- failure to come up with feeding
- there will be an apparent blindness
- constipation, feces will be dry and scanty
- grinding of teeth is also seen
- Nervous signs there will be tremors of the muscles of the head, twitching of the lips, and also the champion of jaws will be seen
- salivation will be there
- there will be tonic and clonic convulsions
- Hyperaesthesia will be there
- cogwheel type clonic contractions of the cervical muscle system
- there will be dorsiflexion and lateral deviation of the head as seen
- stargazing posture will be seen
- so circling tonic-clonic convulsions
- abnormal postures as I told stargazing posture
- in coordination falling and
- recumbency will be occurring in three to four days
- there will be a profound depression and
- there will be difficulty in lambing and the animal will recover after lambing

Now I will show the slides showing:

the Clinical signs the animal is recumbent

there is a stargazing poster

the third animal is recumbent with the stargazing posture

so, this is again a stargazing poster in pregnancy toxemia, In the same animal, we were able to see a Hypoglycemia the glucose level is only 31 milligram per deciliter and ketonemia so the ketometer values showed more than 3.1, which indicates that the animal is going for there is an increase in the ketone body production.

Clinical Pathology; how are we going to diagnose the presence of ketosis is by Diagnosis of the Hypoglycemia Ketonemia and

Ketonuria. so, we can see the presence of ketone bodies in the serum and also in the urine.

So, *Glucose level*

The normal range is between 50 to 65 milligram per deciliter

whereas in the case of Ketosis the range can be between 20 to 40 milligram per deciliter and in case of secondary Ketosis, there may be a Hyperglycemia

Ketone bodies:

beta hydroxy butyrate (BHB)

Normal will be less than 1.0 millimole per liter

whereas in the case of clinical ketosis it will be more than 2.5 millimoles per liter

milk and urine ketone bodies can be diagnosed or tested for the diagnosis of ketone bodies; this can be deducted by the reaction of the acetoacetate with sodium nitroprusside.

sodium nitroprusside reaction is more sensitive with acetoacetate than acetone acetone is only deductible when the concentration is greater than 600 millimole per liter

Sodium Nitroprusside Test

so, this is the test showing the sodium nitroprusside test that is called a Rothera's Test in the urine and we have got commercially available strips for diagnosis of the urine ketone bodies.

It can be diagnosed by saying

Hypoglycemia

Ketonemia

Ketonuria

Serum beta hydroxy butyrate (BHB) level will be more than 3.0 millimole per liter

Metabolic acidosis, one of the most important signs in the case of the ovines is metabolic acidosis will be there

Terminal uremia is more common and

Abnormal liver function test because as I told fatty liver is one of the important signs in case of ovine ketosis and

The elevated plasma cortisol level will be there more than 10 nanograms per ml.

Milk fat to Milk protein Ratio:

There will be a difference in milk fat to milk for protein ratio this can also be used for the diagnosis of ketosis.

Fat to Protein ratio should be greater than 1.5, if it is less than that then definitely there is Ketosis, that is why the owner used to complain that the milk is watery, when we are stripping the milk there is no frothiness or the froth is breaking frequently, and the milk is watery that will be the primary complaint from the farmers.

In *serum biochemistry*, there will be elevated non-estrified fatty acids will be as elevated total bilirubin because the liver function will be affected and there will be a decreased plasma cholesterol level because of the liver dysfunction and low plasma cortisol concentration.

Diagnosis: is mainly based on the

History of the animal

Clinical signs, in the case of bovine ketosis diagnosis, are based on the history, clinical signs,

Biochemical examination,

Ketonemia and

Ketonuria

these are necessary to establish the diagnosis.

Differential Diagnosis: Wasting form and Nervous form both are to be differentiated with the different diseases.

The wasting form will be there because it is occurring immediately after the postpartisan period, we need to differentiate it between the disorders or diseases that are occurring in the immediate postpartisan period so including the abomasal displacement, Traumatic reticulitis, Traumatic peritonitis, and Primary indigestion because that is occurring in immediately after the postpartisan, cystitis and pyelonephritis, diabetes mellitus.

Nervous form as we all know in Rabies also, we will see similar signs like that of the Nervous form of the ketosis, so one of the important differential diagnoses is Rabies.

Hypomagnesemia, again hypomagnesium tetrad also is more

common so we need to differentiate it between hypomagnesemia also. and bovine spongiform and cephalopathy.

In the case of Sheep Diagnosis can be made with the clinical signs, history, history of pregnancy, and it should be differentiated from Listeriosis Cerebral abscess and Rabies.

So, in nutshell in this class, we have seen about the

- Clinical findings or Clinical signs that are seen in case of the Bovine ketosis and Ovine ketosis,
- different forms that as the Wasting form, Nervous form, and how it will be seen in case of the ovine ketosis and
- how to Diagnose this with the blood and also the urine and
- we have seen the diseases that are to be differentially or that are to be differentiated from the ketosis in both Ovine and the Bovine

In the next class or lesson number 5, we will see the Treatment and Management and Control of the Ketosis.

Thank you!

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PDF: Clinical Findings and Clinical Pathology of Bovine and Ovine Ketosis

5 Treatment of Bovine and Ovine Ketosis

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Transcript

In lesson 4 we have seen the Clinical signs of Bovine and Ovine Ketosis, Clinical Pathology, How to Diagnose it in the serum and the urine, and the Differential Diagnosis of the Bovine Ketosis in both the Wasting and the Nervous form and also of the Ovine Ketosis.

In this class, we will see about the Treatment, Management, and Control of the Ketosis of both Ovine and Bovine Ketosis.

Treatment

Treatment of the Bovine Ketosis: so actually the animal should be treated in the early stage itself, in order to prevent the occurrence of the Nervous form the or the animal from becoming worse in condition, so it should be treated earlier.

It starts with the Replacement therapy so Replacement therapy can be done with 50% dextrose at the rate of 500 ml Intravenously.

Intraperitoneal injection can also be given at the rate of 20% dextrose can be given. Still, there is a chance of infection, so it is of lesser importance, this type of treatment is very rarely used. For Primary ketosis, an Intravenous injection of 100-500 ml of 50% of the glucose is given and it gives a marked clinical improvement, and the Hyperglycemic state will remain for 4-6 hours. so, this shows

that there are subsequent treatments with this type of solution are essential.

Ketosis Secondary to the butyrate content of the silage can be corrected by modifying the diet or by eliminating or diluting the silage that is causing the increase in the butyrate level.

So, the second line of Treatment is by giving, the Oral **Glucose Precursors** so as such as : Propylene glycol and glycerin: so, propylene glycol can be given as a drench at the rate of 225 grams twice daily for two days and followed by 110 grams twice daily for two days and for Glycerol can also be given at the rate of 500 grams twice daily for 10 days.

But again so Other **Glucose Precursors** that can be used are: Sodium propionate 110-225 grams daily, ammonium lactate 200 grams for five days, and sodium lactate one gram per kg initially followed by 0.5 gram per kg for 7 days PO twice daily.

It can be used as a feed additive and an alternative glucose source. So, all these Glucose Precursors can be added to the feed as a feed additive. In the case of sheep; early treatment is essential because it's important, it will result in the response will be good if the animals are treated at an early stage, as we all know the changes that are seen in the case of Ovine ketosis are not reversible, as in case of Bovine ketosis. So Treatment should be started at an early stage for better protection of the animal.

So 5-7 grams of Glucose Intravenously every 6-8 times, that is 6-8 times a day with 20 to 40 units of Zinc, Protamine insulin Intramuscular, every other day for 3 days can be given and Propylene glycol can be given at the rate of 110 gram per OS. Oral therapy includes 160 ml of a solution containing 45 grams of glucose every 4-8 hours along with 8.5 grams of sodium chloride and 6- 17 grams of glycine and electrolytes.

So this can be given orally in order to treat the Ovine ketosis and the best line of treatment if it is in the later stages of the gestation and if the animal is developing signs of Pregnancy Toxemia, the best way to treat is the removal of the fetus by cesarean section or

bringing the termination of pregnancy in order to save the life of the dam.

Hormonal Therapy: this includes Glucocorticoid therapy, so 10 milligrams of Dexamethasone 21 Isonicotinate produces the Hyperglycemia state for 4-6 days, and Dexamethasone sodium phosphate can be given at the dose rate of 40 milligrams and Flumethasone can be given at the rate of 5 milligrams per kg body weight. These are short-acting glucocorticoids, this can be given in order to see to treat the ketosis, and Isoflupredone acetate can be given at the rate of 10 to 20 milligrams, one of the main disadvantages of giving this Isoflupredone is it will cause Hypokalemia in the treated animal. Both glucocorticoid and mineralocorticoid action will be seen because of the use the Isoflupredone. and

We can also use **Anabolic steroids** to Treat the Bovine ketosis, that is 60 milligrams and 120 milligrams of trenbolone acetate, Repeated treatment will cause profound Hypokalemia, the use of this glucocorticoid is to prolong the Hyperglycemic effect; one is by increasing the tissue uptake of glucose, and another one is it reduces the milk production for the next 2-3 days so thereby decreasing or it brings down the ketotic signs.

And another one is the use of **Insulin**: Insulin, lower doses of long-acting insulin that is 200 International units of proton zinc can be given once every 48 hours.

Ruminants are insulin resistant during early lactation which is why the patient goes for the lipolysis,

So Pancreatic secretion of insulin is also reduced so, insulin will assist in giving insulin. It will assist in suppressing the fatty acid mobilization and improve the tissue uptake of glucose while stimulating the hepatic glycolysis.

We can also go for Lipotrophic Agents: such as Choline can be given at the dose rate of 25-50 grams per day orally daily PO. Choline is a precursor of phosphatidylcholine which is required for phospholipid synthesis.

L-Methionine can also be given, it is also a precursor for

phospholipid in the synthesis of apolipoprotein because the presence of apolipoprotein is more essential in order to export the very low-density lipoproteins(VLDL), so the triglycerides will be converted into very-low-density lipoproteins and this will be exported. But the ruminants have a very low capacity, inherently low capacity to export the very low-density lipoprotein, for this apolipoprotein is most essential. so L-Methionine can be given in order to export the very-low-density lipoproteins.

Miscellaneous Treatment: includes Cysteamine which is given at the rate of 750 milligrams Intravenous every 2-3 days. Amino acid lysine can also be given which also plays a major role in the apolipoprotein synthesis, as I told which will improve the VLDL secretion. and

Vitamin B12 as I told is an important cofactor for the coenzyme taking part in the oxidation of the acetyl-CoA via the TCA cycle, so Vitamin B12 can also be given, it can be given, it is useful in the metabolism of propionate as it enters the TCA cycle.

Nicotinic acid or nicotinamide can be given at the dose rate of 6 grams per OS daily for 10 weeks, it can be started 10 weeks after calving up to 10 weeks after calving.

Nicotinamide enzymes were reduced in mammary glands in ketotic animals.

Niacin can be given, it decreases the blood ketone levels and increases the blood glucose. Niacin is an anti-lipolytic agent, it can be supplemented two weeks prior to 12 weeks after parturition.

Chloral hydrate: Chloral hydrate will increase the breakdown of starch in the rumen, why we need to improve the breakdown of starch in the rumen is, by breaking down the starch propionate level can be increased which will improve the oxidation of the acetyl-CoA via the TCA cycle.

Chloral hydrate will improve the breakdown of starch in the rumen and influence the production of the propionate. The initial dose of 30 grams followed by 7 grams twice daily for several days can be given, one of the added advantages of giving this chloral

hydrate is, that it has a sedative effect in the treatment of the Nervous form of ketosis. And,

Monensin: nowadays I mean most of the daily farm Monensin is used as a feed additive. so Monensin reduces the clinical and subclinical ketosis, the main thing is it decreases the acetate to propionate ratio in the rumen. As we all know the energy synthesize in the case of ruminants, ruminants will absorb the carbohydrates as hexose sugar for a very little quantity only, the remaining carbohydrates will be fermented in the rumen as acetate butyrate and propionate. By giving Monensin, we can decrease the acetate to propionate ratio in the rumen because of its effect on the rumen fermentation.

Increases the availability of the propionate as a glucose precursor, so that it helps to suppress the fat mobilization and ketone in turn ketone body formation can be decreased.

Supportive therapy: It is also a supportive therapy for reducing ketosis and the remaining supportive therapy includes rumen transfaunation, provision of a variety of palatable feed, and also exercise.

So, these are the line of treatment, that is used for treating the Bovine and Ovine Ketosis.

Control: How it can be controlled or it's before or preventing the occurrence of Ketosis. How it can be controlled.

Preparation of next lactation; it should start or it should begin about 4 weeks prior to calving itself, the preparation for next lactation should start.

So silage, hay, or pasture should be given as a maintenance ration and

it should be supplemented with 1 kg per day concentrate and gradually it should be increased to 5 kg per day as the animal is approaching the calving time.

And the concentrate should be increased gradually as the production increases at the rate, so hay should be given at the rate of 3 kg per 100 kg body weight for maintenance and 1 kg of grain should be given for every 3 kg of milk production, so by this, we

can prevent the formation of ketosis in the immediate postpartum period.

Another one is carbohydrates whatever we are supplying should be readily digestible, it can be in the form of like what they say is oats or maize should be crushed, which will make the carbohydrates more easily digestible,

and adequate amounts of cobalt, phosphorus, and iodine should also be given in order to prevent the nutritional deficiency thereby leading to secondary ketosis.

Prophylactic feeding of sodium propionate at the rate of 110 grams per day for six weeks from calving will also prevent the occurrence of ketosis.

Propylene glycol can be given at the rate of 356 ml per day for 10 days after calving, that is 6%, it should be 6% of the concentration for 8 weeks can be given in order to prevent the formation of ketosis in the immediate postpartum period.

Monensin, which is given as a growth stimulant at the rate of 25 milligrams per day in a grain feed mix will also prevent ketosis.

In the case of the Sheep, to prevent the Ovine ketosis, body condition scoring should be maintained at 2.5 to 3 at 90 days of gestation, whereas the last two months are important where we have to go for 70% of the lamb's birth weight will be achieved in the last two months of the pregnancy, especially during the last 6 weeks of pregnancy. So maintaining the animal during the last six weeks of pregnancy will prevent Ovine ketosis.

Concentrate should be given at the rate of 0.25 gram, that is kg per day increasing to 1 kg per day in the last 2 weeks period of gestation and avoid so one of the most important points in avoiding the Ovine ketosis is there should not be any sudden change in the feed, so sudden change in the feed the animal will not take the feed which will lead to any short period of inhabitants or if the animal is not taking feed, immediately the animal will go for Pregnancy Toxemia. To avoid sudden changes in the feed. and

Another most important point is providing shelter, which will protect the animal from the cold and inclement weather.

So, in lesson number five, we have seen the

- Treatment, what are all the lines of treatment for Bovine Ketosis and Ovine Ketosis and
- How can we go for the Management and Control in order to prevent Ketosis in the immediate postpartum period?

Thank you! for all of your kind attention and for your patience in listening to the class.

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PDF: Treatment of Bovine and Ovine Ketosis

PART IV

WEEK 4: UPDATE IN BOVINE MASTITIS

1 Epidemiology of Bovine Mastitis

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Transcript

Hello friends!

Welcome to the agMOOCs session on Bovine Mastitis. So, this session is divided into 5 lessons for ease.

In lesson one, we will deal with the Epidemiology of Bovine Mastitis and

In lesson two, we will deal with the Immune system and Pathogenesis of Mastitis

In lesson three, we will deal with the Clinical signs and Diagnosis of Mastitis

In lesson four, we will discuss the Treatment of Mastitis

In lesson five, we will deal with the Alternate Therapies, Prevention and Control of Mastitis

Now we will move on to lesson-1, which is Epidemiology of Mastitis

So in about Epidemiology of Mastitis, we will be seeing a brief;

1. Introduction to the Mastitis
2. Its social and economic impact on the disease,
3. Prevalence and Incidence Mastitis and also
4. the Risk factors and the Pathogens involved in the occurrence

of Mastitis.

To have a brief **Introduction** the word Mastitis is derived from the Greek word that is 'Mastose' meaning breast, 'it is' is inflammation as it is, so there comes the word Mastitis. We started domesticating the cow 8300 years back for milk purposes and Mastitis has been recorded since then, so it is an older disease that is recorded ever.

Social and Economic Importance: yes, we are the highest producer of milk worldwide we have the largest population of buffalo and we have the second largest population of white cattle in the world. And even our babies are dependent on cow's milk for their nourishment. So in that way Mastitis is very important as far as India is concerned and also that is the occurrence of repeat breeders, that is the reduction in conception is mainly due to the animals which are affected with Mastitis rather than any other disease, the antimicrobial residues which are in the treated milk and in the meat, so they can be consumed by the human being posing antimicrobial resistance in the human being or other drug-related problems in human being also.

Coming to the *economic importance*, it has not spared any of the countries the even the US is facing a loss of 2 billion dollars because of this Mastitis, whereas the UK is facing 4 billion dollars because of this Mastitis and as far as India is concerned according to a study in 2019, there was a loss of 8333 crores just because of this Mastitis and the world wide loss because of this Mastitis it was 35 billion dollars.

Coming to the Prevalence, it has not spared any country and as far as India is concerned, there is more than 18% of clinical Mastitis Prevalence and 43% of subclinical Mastitis Prevalence in India, West Bengal. Actually, in West Bengal there is more Prevalence, that is with 75% of subclinical Mastitis and the Andaman has the least that is 12% of subclinical Mastitis.

Moving to **Risk Factors for Mastitis**: Mastitis risk factors can be broadly classified due to **Pathogen factors, Environmental factors, and Host factors.**

So among these, the Host factor is the majority because the risk of Mastitis increases with the age and increases with the calving,

there is a particular period that is between 21 to 90 days of lactation,

the incidence or the risk of Mastitis is going to be high.

the Milking Interval: longer the milking interval, there is high the risk for Mastitis

the Milk Yield: normally we go for a selection of high yielders, with high milk flow but both these characters pose them for Mastitis

the dry period is the riskiest period for the development of Mastitis, during which the new intra memory infections could occur

then the other thing is *Udder Confirmation*, even before purchasing the animal we have to check for the Udder confirmation because the distance between the ground and the teat tip plays a major role in the development of Mastitis

Also, they should be placed uniformly you would have seen the ladder-type of the udder and if you see the shapes of the teat, there are many like cylindrical, pencil-shaped, conical shaped, they have their own merits and demerits. For example, a cone-shaped teat will be highly prone to Mastitis development if it is having a machine type of milking.

The other factors are previous inflammation of the teat, so any teat like this can lead to the occurrence of Mastitis and even the carryover of the previous infection to the next lactation can also pose a risk of Mastitis.

Even the presence of other diseases can also cause immune deficiency leading to the risk of Mastitis in those animals.

Coming to **Pathogen Factors**: it depends upon what type of pathogen is affecting, its virulence, its load, its properties like the ability for addition, its ability for biofilm formation, and even blind treatment without assessing or identifying the causative pathogen can also lead to antimicrobial resistance which also leads to the risk of Mastitis in those animals.

Coming to **Environmental Factors**: it starts with the dietary habit, what we are giving, deficiency of vitamin A and Selenium can also

force the animals to the risk of Mastitis and the bedding type, what we use whether it is either wooden shreds or dried straws, they also can harbor organism and lead to the development of Mastitis.

You know that there are four types of milking: Stripping, Knuckling, Whole Hand Milking, and Whole Milking with stripping.

So among these Whole Hand Milking is suggested as the better one, which will cause the least damage to the teat. Normally small farmers do milk with their hands and on large farms, they use milking machines. Any faulty attachment of these machine knobs can lead to the occurrence of Mastitis and even if it is not cleaned well, it can transmit the disease from one animal to the other. Commonly the Bacterial causes are more and even among that worldwide. staphylococcus, streptococcus, E-coli, and mycoplasma Mastitis are the common ones.

Though they are common pathogens more than 250 strains have been identified or isolated in Mastitis milk.

Coming the **Virus causes**, virus either directly or indirectly they cause Mastitis. The causatives can be Herpes viral mammalitis or in cases of FMD or in cases of LSD.

The picture you are seeing is the Ulcerated wound, which is caused due to LSD. So apart from that Enzootic bovine leukosis and Bovine mammalitis immunodeficiency viruses can also cause Mastitis.

The Fungal causes commonly the Candida species, Aspergillosis, Trichophotons, and Penicillium can also cause Mastitis. The problem with the Fungal Mastitis is that if we are not diagnosing it properly and if you are straight away going for antibiotics, these antibiotics oxytetracycline and penicillin will be utilized for the nourishment which contains nitrogen.

So with this, we are concluding lesson-1,

so in lesson-1

we have seen a brief Introduction to Mastitis

we have seen the Prevalence and Incidence of Mastitis

we have seen the Risk factors for Mastitis

we have seen the Common Pathogens which cause Mastitis.

So in lesson-2, we will continue with the Immune System of Udder and Pathogenesis of Mastitis.

Thank you

Download

PDF: Epidemiology of Bovine Mastitis

2 Immune System of Udder and Pathogenesis of Mastitis

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Transcript

Hi Vets,

In the last class, we have seen about the Epidemiology of Mastitis.

In this lesson, that is in lesson-2, we will see about the Immune system of Udder and the Pathogenesis of Mastitis.

Historically the milk from the cow's udder was thought to be sterile, but the evolution of the concept of Udder Microbiota has completely changed the thought, that the milk from the udder was sterile. because even in the normal milk, which doesn't have any disease we could be able to isolate more than 250 strains of bacteria.

And among these Microbiota some are pathogenic, which may be harmful to the udder and others are actually beneficial, which may prevent the occurrence of Mastitis.

The Udder Microbiota can also be classified based on the niches, that is udder microbiota of the teat affects the udder microbiota of the teat canal and the milk itself.

So among the microbiota of the teat apex, teat canal, and the milk, the number of or the diversity of the microbiota is more in the teat

canal, that is more than 165 strains have been isolated from the teat canal.

Among these, Lactobacilli and the Lactococcy, have the property of aggregation and they have hydrophobic property and colonizing properties. So with this, they could compete with the pathogens which try to colonize and infect the udder.

Coming to **Mammary Gland Defense Mechanism**; that can again be divided into the first line of defense mechanism, that is the Anatomical barrier, the second one is the Innate immune system of the udder, and the third one is the Recruitment of Neutrophils.

So the Anatomical defense barriers are actually considered as the first line of defense mechanism, which includes the Teat skin, Teat system, and Teat canal.

Actually, in Teat skin, the stratified squamous epithelium is covered with a layer of keratin, which prevents the colonization of bacteria.

The Teat canal is also lined by Keratin, and a thin layer of lipid forms the physical plug and prevents the entry of pathogens into the udder.

Any microorganism which tries to enter the Teat canal in between the milking, will be trapped by the keratin layer and they will be flushed out along with some layer of keratin, during the next milking and this is called as keratin flush.

Even if they escape this and ascend, they have to face the challenge of the rosette of sternberg which is a ring of neutrophils that will destroy this bacteria.

Coming to the **Innate immune response**; the macrophages located in the alveoli, sensitize the bacteria which are entering into the udder and they engulf the bacteria by a process called Phagocytosis and they shred them into pieces and release these pieces, which will be again sensitized by the neutrophils and the neutrophils will be recruited to that place which again destroys these pathogens.

Apart from this, udder has many antimicrobial peptides and glycoproteins, one among them is Lactoferrin whose iron-binding

capacity is 300 times more than the transferring. Actually, it is having a bactericidal effect on Staphylococcus aureus and a bacteriostatic effect on E-coli. So having all these immune systems how a pathogen causes Mastitis, prepare loss of keratin layer and post milking dilatation of the teat canal, this facilitates the entry of microorganism into the udder.

So once the organisms enter into the udder, they are being facilitated by the udder environment. Even they get horizontal signaling from the udder microbiota which is already residing in the udder.

Next, some of the bacteria like staphylococcus aureus and even E-coli go for Biofilm formation and Internalization.

So during this process even if the macrophages engulf, they go for encapsulation and remain dormant and live in the macrophages and also release proteases and toxins like alpha and beta, which on one side destroy the antibodies and on the other side they damage the epithelial cells and get internalized into the epithelium, where they reside for a long time with low activity and they again cause chronic Mastitis in due course of time.

On the other side, the presence of pathogens is sensitized by the neutrophils and they enter into alveoli, while entering into the alveoli, they displace the secretory cells leading to increased somatic cell count in the milk.

Aggregation of these leukocytes and blood clotting factors forms clots, which abstract the flow of milk out of the udder and they also cause scar formation and proliferation of connective tissue. And in the first lesson we have also seen, that is the main reason for reduced conception rates in cattle, so that is due to the mechanism that bacterial invasion releases LPS that is lipopolysaccharides, which increases the level of cytokines, PGF2 alpha, and increased body temperature, which in turn reduces the level of GnRH, LH, estradiol, and progesterone, these changes are harmful to oozing maturation, ovulation, embryo implantation, and early embryonic development, this results in the decrease of reproductive efficiency of dairy cows.

So far we have seen about

- the Microbiota of the udder,
- the Defense mechanism of the udder, and
- Pathogenesis

In the next class, we will see about the Clinical signs and Diagnosis of Mastitis.

Thank you!

Download

PDF: Immune System of Udder and Pathogenesis of Mastitis

3 Clinical Signs and Diagnosis of Mastitis

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Transcript

Hi Vets,

In the last class, we have seen about the Microbiota of the udder, the Defense Mechanism of the udder, and Pathogenesis.

So in this lesson, that is lesson-3, we will see about the Clinical signs of Mastitis and the Diagnosis of Mastitis.

So Mastitis can be classified

Based on Epidemiology as Contagious and Environmental.

Based on Inflammation as Clinical and Sub-clinical and

Again on Severity as Acute, Chronic, and Peracute.

Contagious Mastitis is one, which spreads from the disease to a quarter to a healthy quarter or even from the diseased animal to the healthy animal, mostly during milking. Examples of this Contagious Mastitis causing organism are:

Staphylococcus and Corynebacterium.

Environmental Mastitis commonly the Pathogen will be in the cow's environment and may be in the soil, bedding, or water. So the common pathogens are Streptococcus and Escherichia Coli. So second definition is based on Clinical signs; we have seen it has Clinical and Sub-clinical Mastitis.

Sub-clinical Mastitis can be defined as inflammation of the udder

in the absence of any physical changes in the milk and also in the udder. It will not be usually visualized by the farmer. In some cases, he may report only the reduction in milk yield.

whereas Clinical Mastitis is farmer identified disease, he can very well identify changes in the milk or in the udder even in the animal.

So, the Clinical Mastitis can be further divided into 3 based;

Abnormal Secretion

Abnormal Quarter and

Abnormal Cow.

In Abnormal secretion, we may find flakes or watery milk or even blood tinge in the milk but there won't be any change in the udder or in the animal.

In Abnormal udder;

There may be Inflammation of the udder, that is swollen or rigorous.

There may be pain on palpation and

There may be local or diffused fibrosis and

In an abnormal cow, the animal will show signs of toxemia, there may be pyrexia, there may be inheritance and even there may be recumbency.

And in ChronicMastitis, the udder may be regressed, feeling empty, and there may be scanty secretion or even no secretion and on careful palpation, you will be able to feel the fibrosis.

In *clinical examination*; we have to examine all the quarters and the teat for similarity or any inflammation or fibrosis and we have to check the secretion for any changes and we have to check for the confirmation of the teat, whether are there any deviation or convergence are to be considered.

Field tests include pH, Strip cup test, White site test, California Mastitis test, and others. So normally the pH of milk should be within the range of 6.3 to 6.7, so if the pH is more than 6.7 or less than 6, it can be considered Mastitis even without any changes in the milk.

And CMT is considered the cheapest and best reliable field site test, wherein the principle is that the CMT reagent will lie in the cell

and interact with the DNA which coagulates to form gel formation. So here we have to take 5 ml of milk with an equal quantity of the reagent and we have to stir it with this toothpick and the result should be read within 20 to 30 seconds.

And for the *Strip cup* test, we have to use a black cup which will show the presence of any clots or any other change in the milk well.

And the other test is *Electrical conductivity* which is based on the increased level of Sodium and Chloride in the milk even in the subclinical level of damage to the udder, in such conditions the electrical conductivity will be increased.

Somatic cell count is another field site test that can be done for individual animals or even for bulk tank milk, so

- if the reading is less than 1 lakh it is taken as normal
- if the reading is more than 2 lakhs it is considered the presence of Mastitis and
- if the reading is more than 3 lakhs it requires examination of that individual animal

The other test is the *White side test*, wherein we have to take a few drops of milk on a glass slide and we have to add the same quantity that is two or three drops of 4% percent Sodium Hydroxide and stir well, it will lead to the formation of clots or clumps or aggregation, that indicates the presence of Mastitis.

The other test is the *Rapid Catalyst* test, though it is an old method of testing, that is also giving reliable results. So in that again we have to take two or three drops of milk on a glass slide and add an equal quantity of 9% Hydrogen Peroxide and within one minute, you can see the bubble formation which indicates a positive case.

The next is *Bacterial Isolation and Identification*, wherein we have to go for culturing a specific medium that can be used for diagnosing or isolating specific bacteria and it has limitations like all the bacteria cannot be identified because low growing bacteria and some bacteria cannot be cultivated cannot be diagnosed with this method.

If we collect from a milking machine, there is another test called the String test, wherein we collect the milk from the positive pressure side of the machine, which indicates the presence of Mastitis at a herd level.

And coming to *Culture Interpretation*, if the culture is positive in the first week- that is on a day 0 and if it is negative on 7, 21, and 28, it is taken as negative, and if it is negative for the first test and if it is positive for the subsequent test that indicates new Intra memory infection. And the presence of 3 or 4 sites of bacteria is considered Contamination.

Diagnosis:

And the other advanced techniques include PCR and many variants of PCR and among these 16s rRNA sequencing and whole metagenome sequencing are considered authentic or reliable methods of Diagnosis.

Apart from that, we have Eliza, Biomarkers, Biosensors, and Protein-based Diagnosis. So, coming to another useful animal site test Ultrasonography, helps a lot. So, by doing Ultrasonography will be able to assess, even there is any damage to the udder or if there is any fibrosis to the udder, or if there is any obstruction in the teat kennel, so these things can be identified by ultrasonography.

These are some of the videos and pictures, which show the clinical condition and normal ones. Again, with ultrasound, we can assess whether that particular quarter is functional or not. so, for that, we have to administer 10 to 20 international units of Oxytocin and we can examine the functioning of the teat.

So, if you focus on the Lactiferous ducts on the collection tubules, you can find the accumulation of milk. So, in this case you can see the collection of milk, so this is functional. It won't be like this in cases of the diseased or completely fibrous quarter, where there is no functioning. So, in this lesson, we have seen the Clinical signs, Classification, and Diagnosis of Mastitis.

And in the next class, we will see about the Treatment of Mastitis.

Thank you!

Download

PDF: Clinical Signs and Diagnosis of Mastitis

4 Treatment of Mastitis

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Transcript

Hi Vets,

In the last lesson, we have seen about the Clinical signs of Mastitis and the Diagnosis of Mastitis. In this lesson, we will see about the Treatment Strategy, Antibiotic therapy, Antimicrobial resistance, and Non-antibiotic therapies.

The first one is *Treatment Strategy*; a veterinary needs to assess whether Antimicrobial therapy is needed for that particular case or that can be managed with supplements alone, considering the antimicrobial resistance and antimicrobial residues in the milk.

So normally there are recommendations that based on the clinical signs a vet can wait or can directly go for treatment. For example, if it is a change only with the milk and if the udder is normal, and if the cow is normal, it is recommended that a vet can wait for two days and by that time he can go for and if the rapid culture is negative, it can be left without any treatment which can result on its own and if there is any growth we have to go for particular treatment against that organism.

This holds good even with if there is any change in the milk and the udder, but if there is an abnormal cow we need not wait for

any test results, we have to directly go for the administration of antibiotics and the supportive therapies.

Then after that which route is needed for administration, for example, if at least one quarter is affected the administration of the parent drug is not warranted, just administration of intra memory infusion will do. But if it is an abnormal cow we have to go for parental administration.

And the *third strategy* is the selection of antibiotics.

Actually, there are like two approaches-

one is based on the previous experience and the records that we have and

the other is going for the cultural result and the treatment according to that.

Actually, we cannot wait for two days in all the cases, so we need to go with our previous experience and the response to therapy may also differ from animal to animal and even the efficacy of Intra memory drug that we infuse into the udder may differ because of the presence of microabscesses or the presence of inflammation, the tissue protein binding, and the difference in the pH.

And we all know that the antibiotic sensitive may differ from place to place and season to season, so it is a big question whether we can wait for the result so it is better to go for treatment with our previous records of response and our previous results of the culture.

Then comes the *fourth strategy*; for how long, so it is normally recommended that we have to go for extended and aggressive therapy for seven to eight days, instead of the recommended two to three days. and the dose can also be a little bit higher than the recommended dose rate, but I doubt this may go for early onset of antimicrobial resistance, here I am presenting a table that indicates the pathogens and the relevant possible clinical signs and the relatively effective treatment.

So this is for your reference you can go through this table for a better understanding of the Clinical signs, related Pathogens, and the Treatment.

So far we have seen the Treatment of Lactating cows and the next one is the Dry cow therapy, which is very important in eliminating the existing Intra memory infections and preventing the occurrence of new Intra memory infections because I have been told that is a period when the occurrence of new Intra memory infections are common but the efficacy is going to be good if we treat the dry cows that is the dry cow therapy because there won't be much of secretion during this period which may neutralize the efficacy of antibiotics.

So here again there are two types of treatments- one is Selective therapy and the other is Blanket therapy.

In Blanket therapy- we will be treating all the quarters or all the animals in the herd, whereas in Selective therapy- will be treating the affected teat or the affected animal. So normally we will be infused with long-acting intra memory antibiotic preparation preferably Cephalosporins but along with this if some internal heat sealant is utilized, the efficacy is going to be better the examples are Bismuth subnitrate and Chlorhexidine. So apart from this using a product from isella Siberia, which is a lactic acid bacterium useful as an internal teat sealant.

So apart from the usage of NSAIDs, supportive like intravenous fluids as in cases of Parachute and Acute cases, we have to infuse plenty of intravenous fluid even four to five liters of hypertonic saline can be given and immediate access to the drinking water should be given in such cases or isotonic fluids at the rate of 0.5 liters per minute can be administered with the help of Mcdougall needle, which helps in flushing out of the toxins and better dehydration.

Again there is a study that the usage of Oxytocin, at the dose of 10 to 20 international units, can help in flushing out the pathogens and also it helps in the migration of the neutrophils.

Even with this failure to treatment can happen, because of the presence of microabscesses especially increased cases of chronic Staphylococcus Mastitis and inefficient killing of the bacteria and

internalization of the bacteria, which is not available for the drug to act, and due to increased antimicrobial resistance.

The last-mentioned antimicrobial resistance has gained its importance because most pathogens have gained multiple drug resistance, this can be due to unethical over usage of antimicrobials, extensive application of toxic chemicals, and the gut microbiome.

Actually, the concept of gut microbiota is contradictory in ruminants, but it is well established in the human beings that the microbiota of the gut, gets transferred to the mammary gland without any damage, which can lead to horizontal transmission of signaling even to the pathogens which will lead to the development of antimicrobial resistance to the pathogens and there is every possibility of mutation with every organism and again there is a risk of transfer of antimicrobial resistance from the human being to the animal strains.

One of the examples of this is Methicillin-resistant, Staphylococcus aureus, and the other minor reason is that there was a great gap in the invention of antimicrobials from 1970 to 2000. The treatment of Mastitis was started with the Sulfonamides in the 1930s and Pencilline in the 1940s and with other drugs and there was a gap of more than 30 years from 1970, where Quinolones were introduced to 2000.

Talking about the severity of antimicrobial resistance who has also released a list of critically resistant drugs against which new antimicrobials are to be developed, so that high priority is given to enterobacteria which include E-coli and Klebsiella, on the second high-risk bacteria are Staphylococcus aureus. So it is mentioned one thing that they are involved in Mastitis development in cattle.

So apart from Antibiotic therapy, Non-antibiotic therapy includes immunotherapy, nanoparticles, natural cytokines, herbal extracts, probiotics, laser radiation, lysozymes, propolis, and others. Even intra memory infusion of lactoferrin or ozone in addition to traditional medicine to manage antimicrobial resistance is successfully documented in 2019 by Malinowski.

So Immunotherapy can be done by the administration of

microbeads, which contain specific antibodies against E-coli, Streptococcus, or Staphylococcus. And a single injection of interleukin 2 also provides resistance against most of the pathogens which causes Mastitis and infusion of extract of saccharomyces also increase the activity of the immune cells and again specific IgY from the egg yolk, is gives protection against E-coli and Staphylococcus aureus and it also increases the phagocytic activity.

And nowadays nanoparticles have gained importance in all the fields of science so here gold, and silver, nanoparticles are used in the treatment of Mastitis, their combination with antibiotics act as a synergistic and they increase the spectrum and the efficacy of the antibiotic which is administered.

The application of gold nanoparticles with honey is known to be highly antimicrobial against most pathogens, which cause Mastitis in bovines.

A combination of safety offers plus silver nanoparticles as Intra memory infusion has 99% efficacy over most of the pathogens which are causing Mastitis in bovines.

Nano Soya bean oil has also recorded its efficacy against some of the pathogens which cause Mastitis.

The next one is Photodynamic therapy; the principle is by the release of nascent oxygen and hydroxide, through sensitization of a nanoparticle such as safranine-O, here there is a selective killing and there is no development of antimicrobial resistance, which is an advantage of photodynamic therapy.

The next one is Acoustic pulse therapy; where the mechanical stimulus is converted into biochemical changes, which enhances the healing, and promotes the recovery even in affected animals.

The next one is *Radiation therapy*; here we use a low-intensity laser this enhances the regenerative capacity reduces pain and inflammation and also enhances the phagocytic activity.

The next is *Stem cell therapy*; here we use stromal stem cells of adipose tissue, they have antimicrobial properties, they have good regenerative properties, and they have angiogenesis, these are required for earlier recovery from the Mastitis.

The next one is the *usage of Probiotics*; Probiotics are nowadays commonly used in human Mastitis, they have a better effect than the usage of antibiotics, which are administered parentally or orally. But in ruminants, the study is only in the primary stage and better documentation or researchers are needed in this area.

The next one is *Antimicrobial Peptides*; actually, the antimicrobial peptides of a unicellular organism are like back resins whereas the antimicrobial peptides of multicellular organisms are defensins and cathelicidins, they also have good antimicrobial properties which will help in fighting against the Mastitis causing pathogens, but the limitations are they have a short half-life and the production cost is high and its efficacy is studied only in vitro and on the other hand they have a more cytotoxic effect.

The next one is *Bacteriophage*; they are very selective against the specific pathogens, injection of phage license, instead of this bacteriophages have a better effect and nowadays combination or cocktail of bacteriophages are being used instead of using single bacteriophage. The limitation is quicker development of antimicrobial resistance, snake venom of Bothrops are used, which is proved to disrupt the biofilm formed by Staphylococcus aureus.

If no treatment is effective, what next to do we use to dry that particular quarter? So for that 30- 60 ml of 3% silver nitrate can be infused or else 20 ml of 5% copper sulfate or 100-300 ml of 5% povidone-iodine 3 days continuous infusion of 60 ml of chlorhexidine can also be tried.

So this concludes lesson-4, so here we have seen ;

- The various Strategies of Treatment,
- Antimicrobial choice,
- The duration and the length of Treatment required,
- Usage of NSAIDs,
- Usage of Supportive therapy,
- Usage of Oxytocin and Non-antibiotic therapy

So in the next class, we will see about Alternative Therapy, Prevention, and Control of Mastitis.

Thank you!

Download

PDF: Treatment of Mastitis

5 Alternate Therapy, Prevention and Control of Mastitis

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Transcript

Dear friends,

In the last lesson, we have seen about the Treatment of Mastitis and

In this present lesson, we will see about the Alternate Therapies available for treating Mastitis. the Prevention and Control of Mastitis.

Nowadays the increased demand for organic products, let it be meat or milk and any vegetables, and the concern the antibiotic residues and the antibiotic resistance, have attracted even the modern farmers towards the usage of a natural way of Treatment.

So in this lesson, we will see about the available Ayurvedic and Herbal medicines, which are used commonly for the Treatment of Mastitis. And Homeopathy drugs, are commonly used in the treatment of Mastitis, the prevention, and the control of Mastitis. Though N number of Herbal medicines and Ayurvedic preparations are available throughout the world, as far as India is concerned, we are using the combination of Aloe vera, Lime, and Turmeric

powder, which is applied over the udder, which very much reduces the inflammation even in clinical Mastitis.

This video shows how the preparation is made and how it is to be applied.

Other herbs used are Coconut, Babassu, and Palm oils, which are known to control bacterial contamination by minimizing the bacterial colonization of water.

Also, garlic and butter and black cumin, and wheat flour can be administered orally against E-coli and Staphylococcus aureus Mastitis.

It is also confirmed that Honey inhibits and eliminates the biofilm produced by Pseudomonas, Streptococcus, and E-coli.

Apart from this Goat weed, Jam fruit, Turmeric, Pudina, Tulasi, and Kadukkai were also very much helpful in Treating Mastitis.

Coming to Homeopathy Therapy, many drugs are commonly used in the treatment of Mastitis, but there are combinations like Belladonna, Bryonia, and Urticaria which are used in all kinds of Mastitis.

And for Intra memory usage, the combination of Belladonna, Calendula, Dulcamara, and Echinacea are used.

And for Internal usage combination of Phytolocca, Silicea, Belladonna, Calc. Fluor, Arnica, and Conium were used and a combination of Phytolacca, Calendula, Apis, and Belladonna can be used as external use to apply over the udder, which can be used in cases of fissures, wounds, ulcers, congestions, hematomas, and to reduce inflammations.

Prevention is mainly by Managemental practices, Maintaining good nutritional status, Genetical selection, and Vaccinations. Good udder health management includes Washing the udder and keeping the udder clean without any dirt, keeping good bedding material, and swiping or dipping the teats before and after milking.

So out of this dipping Iodine based tip in 10% glycerin is considered the golden standard for teat dip.

Coming to *Vaccines*: though vaccines are available against Mastitis, their efficacy is questionable. And normally the vaccine

is focused on E-coli Mastitis and Staphylococcus Mastitis and the reduced efficacy can be due to other factors like Host factors, Environmental factors,s or the Pathogen factor.

And recently Intranasal vaccines have also been introduced against Staphylococcus aureus, but it's only a pilot study, and its clinical efficacy needs to be assessed.

These are all some of the examples of vaccines, here it is against Coliform Mastitis, normally it does not prevent new Intra memory infections but it will significantly reduce the clinical severity of the infection. The dose rate is 5ml subcutaneously to be administered 3 times. and

This table indicates various trials which are run for this E-coli vaccine and this is an example of the vaccine against Staphylococcus aureus, here five different phases of Staphylococcus aureus are used and dosage is again 5ml but intramuscular to be repeated in 14 days and the first dose is at 6 months but the effect is not as good and these are all the variable field trials which are done with the vaccine.

The next one is Genetic selection, so genetic selection is mainly based on the sire index so that is by selecting the proper sire. If milk production, fat level, protein level, fore-udder attachment udder depth, and udder support are included in the selection index, the udder health can be improved by genetic selection.

Here the list of main genotypes which are linked to Mastitis is listed, and their Gene/locus and properties are listed over here. Considering the nutritional status, trace minerals like Selenium, Copper, Zinc, and Vitamins like Vitamin A/beta-carotene and Vitamin E, are more concerned with the health of the udder. Even the injectables with Zinc, Magnesium, and Copper have very much reduced the incidence of chronic Mastitis on the other hand cattle with negative energy balance are very much prone to Ketosis, and such animals are at double risk of developing Mastitis.

Dietary zeolites with their antibacterial immunostimulating and detoxifying properties can reduce the incidence of Mastitis and even fight against the pathogens.

Altered nutrition or malnutrition or even altered gut microbiota, can detrimentally affect the udder health by gut-brain access signaling.

Control is mainly by eliminating the existing infections and preventing the occurrence of new infections.

Eliminating the existing infection can be done by Dry cow therapy, Lactating cow therapy, and culling the animals which are not responding to treatment.

Preventing new infections can be done again by Dry cow therapy, proper teat dipping, proper milking method, attaching proper milking machine, environmental and nutritional management, and quarantining newly purchased animals, again endpoint Mastitis control program has been recommended by the UK, which is not actually existing in India according to NDDB, that is establishing other health goals, maintaining clean, dry, and comfortable environment, proper milking procedures, proper maintenance, and use of milking equipment, good record keeping, management of clinical Mastitis during lactation, effective dry cow management including blanket dry cow therapy, maintenance of good biosecurity for contagious pathogens, regular monitoring of another health status, and periodic review of the Mastitis control program.

Mastitis is a million-dollar disease, causing economic loss to the farmers, not only by reduced milk yield but also by reduced reproduction. Maintaining the good health of animals can be done by improved nutrition, environmental sanitation, use of teat sealants, and selection of disease-resistant genetic traits.

And apart from this more rapid pathogen detection, sensitivity, usage of proper antibiotics, and sufficient course of treatment would eliminate the infection and also reduce the occurrence of antimicrobial resistance and improve the effectiveness of treatment.

Dear vets hope this lecture was useful to you in understanding the Pathogenesis and the Advancement in the treatments by means of antibiotics, non-antibiotics, and alternate medicines.

Thanks for listening

Thank you!

Download

PDF: Alternate Therapy Prevention and Control of Mastitis

PART V

WEEK 5: POST-PARTURIENT HEMOGLOBINURIA IN CATTLE

1 Definition, Etiology and Epidemiology

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Transcript

Greetings to all!!

Today's topic is about Bovine Postpartum hemoglobinuria. It consists of four lesson. Lesson number one is about definition of the Disease, importance of the disease, and Causes, and second lesson is about the Etiopathogenesis, third lesson is about the clinical findings and clinical Pathology, fourth lesson is about the treatment and management of postparturient hemoglobinuria.

Now coming to lesson number one, first slide we are going to see about what is the definition of the bovine postpartum hemoglobinuria. It is a non-infectious hemolytic syndrome of adult cattle and buffaloes. This is mainly characterized by intra-vascular hemolysis, Hemoglobinuria, severe anemia and death is mainly due to anemic anoxia. So, the phosphorus deficiency that is occurring during the early stages of the pregnancy or sorry early stages of the lactation is widely believed to be associated with this condition.

Exact pathogenesis is not completely understood and it is commonly seen in case of high yielding dairy cause especially during the third to sixth lactation and multiparous animals are affected more commonly, and the disease is found to be, the prevalence is found to be more common during the first month of

lactation or after carving, and the higher incidence, this is the third slide I am going to talk about and it is high most commonly seen in case of high yielding dairy cause during the winter month. And risk factors what are all the risk factors that are associated with or which predisposes the animal for postpartum hemoglobinuria is ingestion of the cruciferous plant such as cabbage, onion, cauliflower and radish, and this cruciferous plants or beet pulp are low in phosphorus. Another one of the most important factor, this is the fourth slide I'm talking about another most important factor that is predisposing the animal to postpartum hemoglobinuria is copper deficiency. So, low copper status which is related to the application of high molybdenum or sulphur will lead to copper deficiency. And another one is, another common important factor is phosphorus deficient soils and drought conditions also predisposes the animal to hypophosphatemia and the last important factor that predisposes the animal to postpartum hemoglobinuria is ingestion of excess of cold water or exposure of the animal to extreme cold weather will precipitate an episode of postpartum hemoglobinuria. So, in this first lesson we have seen about the definition, the factors or the causes that are leading to the Hypophosphatemia. In the coming session, we will see about the etiopathogenesis and clinical findings, clinical pathology, and treatment.

Thank you

Download

PDF: Defenition, Etiology and Epidemiology

2 Etiopathogenesis

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Transcript

Coming on to lesson two here i'm in last class we have dealt about the what is the definition, what is what are all the causes, or the conditions that are predisposing to the post hypophosphatemia and the occurrence of the postparturient haemoglobinuria. Now in this class we will see something about what is the pathogenesis?

How postparturient haemoglobinuria is occurring due to hypophosphatemia or other conditioning factors. Etiopathogenesis, as I told you, too many causes are there for Etiopathogenesis of postparturient hemoglobinuria. In case of mammalian red blood cells or dependent on the glucose pathway as the main source of energy in order to maintain the viable structure and function and So, I will deal about this in the next slide. Now coming to how I have given a flow chart showing how this will lead to the pathogenesis of the condition. So the slide is showing about the oxygens that are present in the feed and so how it is going to cause the postparturient Hemoglobinuria. So, oxidants in the feed it will cause the denaturation of the red blood cell skeletal proteins. This will lead to the denaturation of the Hemoglobin. And, the next important thing is heavy drainage of the phosphorus in lactating animals, and also the low phosphorous diet. Both these will lead to

hypophosphatemia which in turn is going to cause a oxidative stress in erythrocyte cell wall. So, this will lead to the peroxidation of the phospholipid on erythrocyte lipid bilayer. So, all these and again the third causes Hypocuprosis or Molybdenosis excess of molybdenum or lower level of copper.

This will lead to see, copper as you all know copper is essential for the or it is a cofactor, or it is a important component of certain enzymes like ceruloplasmin and superoxide dismutase. These two are important as an antioxidant factor. Okay so, alteration of this or if there is a copper deficiency this will lead to alteration of the activities of the biological antioxidants like superoxide dismutase and ceruloplasmin. This will result in weakening of the so any antioxidant pathway is affected this will result in weakening of the biological antioxidant system of the erythrocytes will be affected. All this will lead to peroxidation of the Phospholipid, phospholipids of erythrocytes lipid bilayer.

So, this is going to cause the decreased deformability of the red blood cell in micro circulation which will lead to the rupture of the erythrocytes, and that is going to be the release of haemoglobin which will result in the postpartum haemoglobinuria. This is one Etiopathogenesis.

We will see the next flow chart that is showing the another Etiopathogenesis. Here, as I told you the phosphorus deficiency it will lead to hypophosphatemia in detail. I will tell in the next slide how it is causing. So, you see the flow chart. So, hypophosphatemia will be there. This will lead to an impairment of the glycolytic pathway of red blood cells. So, there will be depletion of the ATP synthesis. This depletion of ATP or decrease in the ATP is going to cause the haemoglobinuria in two ways. One is it will lower the glutathione enzyme and also nucleotide sorry nicotinamide Dinucleotide, adenosine phosphate or nicotinamide Adenosine, dinucleotide that combine with the hydrogen ion. This will lead to oxidative stress and further this will cause the intravascular hemolysis. The second way how the depletion of the ATP is going to affect the or it is going to cause the postparturient

haemoglobinuria. It is going to make the RBC's prone for hemolysis because ATP synthesis is decreased so, there will be a decrease in the energy source to maintain the normal function and viability of the red blood cells. So, this will lead to the erythrocytes will become more fragile and there will be a rupture of the erythrocytes releasing the hemoglobin. So, erythrocyte is more prone for hemolysis there will be haemoglobinuria and further haemoglobinuria will be there.

This is one of the flowcharts that is showing the how the haemoglobinuria is going to occur due to copper deficiency. So, reduced activity so, once there is going to be a copper deficiency there is going to be a reduced activity of the copper containing enzymes, like superoxide dismutase. This enzymes are most important because they provide protection against the oxidative stress for the erythrocytes. So, once there is going to be a copper deficiency, there is going to be a oxidative injury to RBC. This will lead to the hemolysis and subsequent haemoglobinuria will be there.

And, next slide we will we are going to see the actual pathogenesis. As I told mammalian red blood cells, it all depends on the glucose metabolism in order to maintain the normal or viable function on the structure. So, it is highly vulnerable to factors that causes the or it impasse the glycolytic pathway. So, what happens is whenever there is a hypophosphatemia there is a decrease in the red blood cell glycolysis which will result in the decrease in the ATP synthesis. So, this abnormal concentration of the atp synthesis is going to alter the normal function and structure of the red blood cell, and there will be a loss of normal deformability, and there will be an increase in the fragility of the red blood cells. And so, this will lead to hemolysis so, there is a release of haemoglobin causing the haemoglobinuria.

Another one, is haemoglobinemia and hemoglobinuria will be the subsequent result of this fragility and hemolysis. And, next thing is the changes, the most important point that is to be remembered in this postparturient haemoglobinuria is changes that are occurring

in the red blood cells are irreversible so, and also the lifespan of this red blood cells will be diminished because they are unable to regain its normal or regain its original structure and function. And, the second reason as I told copper and selenium this also plays an important role in the postparturient haemoglobinuria because it provides some protection against the effects of orally ingested hemolytic agents that are present in the cruciferous plants. So, the clinical findings are those of the Hemolytic anemia and death is mainly due to anemic anoxia in postparturient haemoglobinuria. So, in this class we have seen about how individual factors or how individual causes are causing the or leading to the postparturient haemoglobinuria. In the next class we will see something about related to the clinical findings and clinical pathology.

How are we going to diagnose the postparturient haemoglobinuria?

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PDF: Etiopathogenesis

3 Clinical Findings, Diagnosis

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Transcript

In the previous classes we have seen about the Definition, and the Risk factors that are causing the postpartum haemoglobinuria and, in the second class second lesson we are seeing about the Etiopathogenesis and in the third lesson now what we are going to see is about the Clinical findings and the diagnostic methods to diagnose the postpartum haemoglobinuria. So, Clinical findings include as you all know because it is causing haemoglobinuria it is the most important sign so haemoglobinuria will be there the animal will be in appetent or anorectic it will not eat and, weakness will develop the most important sign is the weakness will develop suddenly. There will be a severe depression of the milk yield will be seen, and dehydration will develop quickly because of severe postpartum haemoglobinuria and as you all know haemoglobinuria will cause us severe deficiency of this haemoglobin so the cardiac impulses and the jugular pulses are augmented in postpartum Haemoglobinuria, and there will be a moderate rise in temperature, body temperature and the faces will be because of severe dehydration the faces will be dry and it will be firm. And as you all know again there will be hypoxia so, there will be a Obvious

Dyspnoea will be there and red dark Colored, coffee colored urine and because of hemoglobinuria there will be a pale mucous membrane and once the intravascular hemoysis is going to get increased there will be a the mucous membrane will become icteric, and there will be tachycardia, and shallow breathing is more common, and the patient the animal will be having decreased milk production. PICA, pica is more common during the convulsion period. Course of the disease acute disease it will extend from three to five days, and the cows will become First, it will become weak, then it will become staggery and finally the animal will become recumbent, and because of the loss of the blood supply there will be gangrenous, and sloughing of the tip of the tail or the digits are more common in Hyposthenia, and death will occur within few days. non-fatal cases recovery will take three weeks and PICA can be seen during the recovery stage and This is a picture of showing the haemoglobinuria and this is again a buffalo with post parturient haemoglobinuria and the urine is post parturient haemoglobinuria that is seen this is during the first month of the Lactation.

So how are we going to diagnose ? So the diagnosis is mainly based on the history of the recent calving that is a recent calving or that is first month of lactation and the clinical signs like haemoglobinuria and pale mucous membrane everything will suggest that the patient is going for postpartum haemoglobinuria. And, next one is the urine analysis? What we do is to discriminate between whether it is haemoglobinuria or haematuria make the collect the urine and allow it to stand for some time so that if the erythrocytes settle down then it is due to haematuria, if does not it is due to Haemoglobinuria.

So that will be in a case of urine analysis there will be haemoglobin and Albumin and Higher urine pH will be same and microscopic examination as I told uniformly reddish brown erythrocyte free urine is seen. And hematology as you all know death is due to anemic anoxia and most important sign is hemolytic anemia. So, there is anemia will be most important hematological Diagnosis. Polychromasia will be the Anisocytosis, macrocytosis, basophilic

Striplings, reticulocytosis and increased number of metarubricytes will be seen.

And main important type of anemia is Heinz body anemia and biochemical analysis reveals low phosphorus concentration will be seen, that is less than two milligram per decilitre and there will be a low copper status and high molybdenum status will be Same, and another one factor or some literatures they are dealing about Lecithin cholesterol acyltrasferase (LCAT) level.

So one article has given about the importance of this LCAT especially in case of diagnosis of ketosis and postpartum haemoglobinuria in the recently calved Animal. What is the importance of this? See what they tell us in case of hemolysis there will be there will be a decrease in the haemoglobin level, hypoxia is more common So, this will affect the function of the liver which is essential for the synthesis of LCAT. So, once there is going to be a severe hemolysis, hypoxia will be there, this will lead to the, this will impact the hepatic function in turn LCAT synthesis will be affected so, there will be a decreased level of LCAT in the immediate postpartum period in animals that are suffering from postpartum haemoglobinuria and in ketosis.

Thank you

So in the third lesson we have seen about the normal, what are all the clinical signs the animal will develop in postpartum haemoglobinuria and the diagnostic parameters to diagnose the postpartum haemoglobinuria in subsequent classes we will see the treatment and management of postpartum haemoglobinuria.

Thank you

Download

PDF: Clinical Findings, Diagnosis

4 Treatment

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Transcript

So in previous lessons we have seen about the definition,

Etiopathogenesis, clinical findings, and the diagnostic methods of postpartum Hemoglobinuria.

In lesson number four what we are going to see is the treatment and management. So, first and foremost treatment that is going to be that is most important in case of cause with postpartum hemoglobinuria is blood transfusion. So whole blood transfusion should be done in severe cases. See a delay of 12 hours will result in or will cause an irreversible change. So, the amount of blood required is 5 liter of blood for a 450 kg cow. So additional transfusion will be required if the patient is weak and if the mucous membrane is pale. Supportive therapy is to be given in order to prevent the hemoglobinuric nephrosis so, in order to avoid the danger of the hemoglobinuric Nephrosis. Phosphorus can be administered, or next is the supplementation of phosphorus. Phosphorus can be administered as sodium acid phosphate intravenously at the rate of 60 gram in 300 ml distilled water followed by a subcutaneous injection, and further subcutaneous injections can be given at 12 hours interval on three occasions, and similar doses can also be given orally daily. This is regarding

the phosphorus, and other sources of phosphorus includes Oral dosing with diet bone meal that is 120 gram of bone meal twice daily or dicalcium phosphate or a suitable source of phosphorus and calcium daily for five days can be given. It can be included in the ration. And haematinics because there is the animal becomes more anaemic. Haematinics should be given it can be given in the convalescence period and as I told you Ketosis is also one of the most important disorder along with postpartum hemoglobinuria in the immediate postpartum period. Ketosis is a common complication and hence additional treatment is to be given in order to treat the ketosis in along with the postpartum hemoglobinuria treatment. And next one is the as you all know Ascorbic Acid. It is an important antioxidant along with phosphorus treatment should be given in order to provide an antioxidant property to prevent the rupture of the erythrocytes, and Copper again as I told one of the important causes for postpartum hemoglobinuria is copper so copper deficiency. Copper can be supplemented in the form of glycinate copper at the rate of 1.5 milligram per kg dissolved in 500 ml of normal saline can be given intravenously, or else copper sulphate can be given at the rate of 3.5 gram orally it can be given to supplement copper. Another thing is fibrinolytic agents can be given in order like Epsilon amino caproic acid (EACA) at the rate of 20 gram in 540 ml of normal saline intravenously (NS-IV). Para amino benzoic acid (PAMBA) at the rate of 300 milligram in 540 ml of normal saline can be given intravenously (NS-IV) and because it's having haemoglobinuria we can go for Botropase at the rate of 10 ml in 20 ml of normal saline intravenously (NS-IV) other than this vitamin A, D and E injection can be given at the rate of 5 ml per animal because D will supplement. It will improve the intestinal absorption of the Phosphorus. So, this will help to prevent the animal in getting postpartum haemoglobinuria.

So, I hope you would have understood about the postpartum haemoglobinuria and, the definition about the causes and how the causes leads to the genesis of the postpartum haemoglobinuria, clinical findings and diagnostic methods and also the treatment

and management of postpartum in postpartum haemoglobinuria in immediate postpartum period of dairy cattle.

Hope you all enjoyed the class.

Thank you for your attention.

Download

PDF: Treatment

PART VI

WEEK 6: HYPOMAGNESAEMIC TETANY IN CATTLE

1 Introduction, Etiology, Risk Factors

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Transcript

Greeting to all vets,

I welcome you all to the agMOOCs program on Metabolic disorders in Cattle, jointly organized by the Commonwealth of learning Canada and directed by extended education TANUVAS.

In this program, you could have seen a series of lectures on various Metabolic disorders and this is the one among the common Metabolic syndrome in cattle, is Hypomagnesaemic Tetany in cattle otherwise known as Lactation Tetany, grass staggers, Transit Tetany, and Wheat pasture poisoning.

Normally Hypermagnesaemic Tetany occurs in lactating dairy cows, which is a high yielder, and in an acute form, there will be peracute mortality, so there will be excitatory neurological signs and the animal will die immediately, which is common in mainly inclement weather, especially cattle raised on a lush green pasture or a cereal grass crops.

And now the Hypercalcemia, the deficient dietary deficiency is rare in normal cattle only some metabolic factors influence the absorption of magnesium in lactating cow, and Parakeet form is common and Chronic form is characterized by that is a mostly occur concurrently with Hypercalcemia, so most the time Hypercalcemia

cow they will not respond to routine treatment that is the specific treatment for calcium and their combined treatment of calcium magnesium will give a good result.

And Hypomagnesaemic Tetany in a calf is very common that are mainly raised on a whole milk or milk replacer diet without any roughage feeding those calfs the neurological signs are uncommon and the animal may die.

So, in this class, we are going to see about Hypomagnesaemic Tetany in dairy cattle. We are going to cover 5 lessons.

so lesson-1 is about the occurrence of Hypermagnesiuma and how what are all the risk factors involved in causing the disease and then the Etiology of Hypermagnesiuma and

In the lesson-2, we are going to see about the effect of Hypermagnesemia on various systems and then the various stages of Clinical syndrome manifested in animals and

then in lesson-3, we are going to see about how to Diagnose the Hypermagnesemia by various laboratory methods and then a differential Diagnosis and…

In lesson-4, the main part of the program is how to Manage Hypermagnesemia through different treatment protocols and then the Control of Hypermagnesemia.

finally, in lesson-5, we are going to see about the Occurrence of Hypomagnesaemic Tetany in calves and what are all the Clinical manifestation and how does it occurs, and the Treatment and Management.

Now we are moving on to lesson-1; so in a Hypomagnesaemic Tetany of cattle. In the first lesson, we are going to see is about -What is Hypomagnesaemic, How does it occur, What are all the risk factors involved in causing the Hypomagnesaemic and Magnesium Homeostasis, and the Etiology of Hypomagnesemia.

See Hypomagnesaemic is mainly occurring in lactating dairy cattle and mainly in the normal cow the deficiency will not occur because dietary input is sufficient, but certain factors predispose the cattle to go for a magnesium deficiency and the main magnesium is a major Intra solar divalent element and the excess of

Magnesium is deposited in the bone and then muscles and tissue. So the main role of magnesium is it aids in the various enzymatic activity and mainly it exerts its function on the neuromuscular junction for the release of neurotransmitters, hence the deficiency mainly causes neurological signs in cattle.

So as I said it's an important element for enzymatic activity and muscular function through the release of a neurotransmitter at a motor end plate and it mainly occurs in cattle raised on lush green pasture as well as cereal grass crops.

Now we are going to see how the Magnesium level in the serum is maintained in cattle. Normally unlike a hyper calcium homeostasis it is mainly under the control of a hormone in Hypercalcemia that is a Parathermone and Vitamin D3, but here there is no feedback mechanism to maintain the Magnesium status in cattle and mainly kidney playing a major role in the conservation of a magnesium whenever there is excess demand in cattle so you can see in cow the input of magnesium is through dietary source only then it once reaches the rumen where it's mainly absorbed in the rumen epithelium through the sodium linked ATAPase mechanism, for that the rumen environment should have sufficient sodium potassium ratio of 5:1, that is important for the active transport of magnesium through the rumen epithelium and to certain extent the magnesium is absorbed in the small intestine and as well as abomasum and the excess is excreted in the feces and the absorbed Magnesium it goes to the kidney where the excess level is excreted in the tubules and in lactating dairy cattle apart from that is excretion in the urine phases there is excess loss of magnesium in the milk and normally the magnesium absorption is also deposited on the major bones, that is the reserve for magnesium as well as in the skeletal muscle to give the normal physiological activity.

So input and output whenever there is a dietary deficiency or some factors influence the absorption of magnesium on the rumen, then it predisposes the cattle to go for hypo magnesium so hence there is no feedback mechanism for a Hypomagnesaemic, that is the

maintenance of magnesium in the serum it is only a kidney playing a role for the excretion or reabsorption of magnesium.

Now we are going to see about all the predisposing factors for the causation of Hypomagnesaemic, as the terminology says it's a Wheat pasture poisoning or Grass Tetany, mainly cows that are raised on a lush green pasture, that is in grasses, especially during the winter season in grasses as well as they are fed with some cereal crop grass like wheat or barley or rice. So they are the main source, they contain excess potassium, so once the potassium content is more than sodium, then the ratio will get altered. So normal absorption of sodium is to potassium is 5:1, now the ratio decreases if the rate of potassium increases and sodium decreases is a 3:1. So the active transport of Magnesium through the rumen epithelium gets compromised, so this is the major cause of Hypomagnesaemic in dairy cattle.

Apart from that, there is a severe winter, and there will not be enough fodder to feed the anima, so the dietary deficiency of Magnesium also contributes to Hypomagnesemia. Apart from that, there is acid soil, like leaching of soil may lead to Hypomagnesaemic in a cattle. Fertilization of a pasture may cause that is excess Potassium containing fertilizers causing an altered sodium-potassium ratio in the rumen, so reduce the availability of Magnesium to the animal.

Causes: So as a whole Hypomagnesaemic; mainly it is caused by the factors like even Animal factors that is reduced dry matter intake and normally the concentrate feeds that favor the magnesium absorption the increased volatile fatty acids constituted by the constitute feeding improve the Magnesium absorption, so reduced dry matter is given to the animal, then the reduced volatile fatty acid then we reduce the absorption of magnesium in the rumen, and

Apart from that the period of there being an animal being transported during inclement weather then there also there is chances are there is a deprivation of food and sudden excitement like epinephrine or release, stress, and all these factors may predispose the animal to go for Hypomagnesemia.

The **Etiology** for Hypomagnesaemic is multi-factorial because even though one thing is dietary deficiency but it rarely occurs. Most of the time even though the diet is adequate, the altered sodium-potassium ratio may predispose the animal to go for Hypomagnesemia.

So it is a multifactorial disorder and it mainly occurs when the dietary magnesium concentration decreases from the normal value, that is 1.7 milligram to 3 milligrams, if it is less than 1.7 it is 1 milligram, then it will show neurological signs.

So What is Hypomagnesaemic, How it Occurs, and How the normal Magnesium Homeostasis is maintained in cattle, What are all the various Risk factors involved in that, and the function of a Magnesium.

Download

PDF: Introduction, Etiology, Risk factors

2 Pathogenesis and Clinical Findings

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Transcript

Welcome to lesson-2, so before the previous lesson, you have seen that is How, What is Hypermagnesaemic in dairy cattle, What are the risk factors that predispose the cattle to go for Hypermagnesaemic and the Role of Magnesium, that is a How the Magnesium level is maintained, Magnesium Homeostasis, and the various Risk factor involved with and the Etiology for Hypermagnesaemic.

In today's class, we will see about the Pathogenesis and the Clinical signs of Hypermagnesaemic here that is various stages of Paracute form and a Subacute and Chronic form of Lactation Tetany in cattle. So I have already explained about the Pathogenesis or Homeostasis of Hypermagnesaemic, normally the source of Magnesium is through diet only and it reaches the rumen and the normal sodium-potassium ratio is 5:1, there will be active absorption of the Magnesium through the rumen wall. Even the source of sodium and potassium is through saliva also, so the saliva that reaches the rumen, that maintains the normal sodium-potassium ratio of 5:1 and absorbed magnesium majorly it goes for a skeletal tissue, as well as body tissue and the excess, is excreted in the urine and feces.

Then whenever there are various risk factors as we have seen, the sodium-potassium ratio is get altered is reduced ratio of 3:1, and the absorption of magnesium decreases, so naturally like Hypermagnesaemic here there will not be any mobilization of Magnesium from the bone reserve except in calves, so the only the Homeostasis is maintained by reabsorption of Magnesium or reduced excretion whenever there is demand for magnesium at a serum level. So the reduced magnesium in the serum in spite of the reabsorption of magnesium from the urine causes a severe deficiency and neurological signs.

The major effect of magnesium even though exits an effect on various enzymatic activities, the acute deficiency of magnesium, mainly causes neurological signs. The reason behind this there is acute whenever there are serum magnesium decreases, equally the magnesium level of CSF also decreases, hence there will be the central nervous system signs are more predominant than the peripheral nerve, and as we as I already mentioned Magnesium is mainly required for the release of neurotransmitters that is esterase choline and for the transmission of impulses at the motor end plate. So whenever there is a deficiency it causes muscle irritation and all the neurological signs. The reason for the Clinical signs are: as we already discussed there are various Risk factors that are causing a reduced Magnesium level in the serum, like inclement weather, and then feeding or grazing on lush green pasture, as well as the cereal crop grasses like wheat pasture or barley or a rice cereal crop grasses, and then excess fertilization of the feedstuff, that is a pasture land causes increased potassium content of grasses and predisposed to go for Hypermagnesaemic as well as the excess urea feeding also increases the increased Ammonium content of rumen that impair the Magnesium absorption and finally the reduced dry matter intake, reduces the volatile fatty acid current of rumen decreases the magnesium level. And the stress and other factors that predispose the animal to go for Hypermagnesaemic and finally we are getting Clinical signs. So there are major 3 forms of

Hypermagnesaemic here encountered Acute form, Subacute form, and Chronic form.

Acute Hypermagnesaemic Tetany: So this is a more challenging one to treat and here there is an acute reduction of Magnesium in Lactating dairy cattle that may cause sudden death. So what happens the animal will normally be on grazing land it suddenly stops grazing and the animal will have a disinclination to move and it has a tremor in the body and even slight external stimuli, the animal becomes a hyperaesthetic to external stimuli and there will be a violent behavioral activity, frenzy behavior, you can appreciate. So this is the classical portion of Hypermagnesaemic, so the animals are immediately after showing hyper setting signs and then trimmers of muscles, the twitching of ears, there will be mild tetany, and the animal falls, and it attains there will be an extension of all the limbs and then the deviation of head and neck towards the laterally and it attains opposite onus posture and the animal will have stiffening of limbs and there will be twitching of muscles and then frothing at the mouth and the animal will show tonic-clonic convulsions.

So in this you can see, this is actually a young cattle that is a fed with a green fodder immediately after that within few hours, the animal showed some frenzy behavior and it is a highly hyperaesthetic to external stimuli and there will be sprinkler ears and the animal behaves violently. So here you can see the animal is uncontrollable and it is moving here and there and there are excitatory behavioral signs, we can appreciate in the animal, it is also Hypermagnesaemic here. So other than neurological signs, usually after the animal show tonic-clonic convulsion, the animal will die finally due to respiratory arrest. And sometimes the episodes of signs will occur 3 to 4 times and if the animal is untreated, then it will die.

So apart from neurological signs, the animal will have an increase in temperature and respiration and the heart rate- the intensity of the cardiac sound will be more pronounced, so even with some distance you can hear the loud cardiac sound otherwise known as

thumbs and then usually the death occurs in animal within few hours of manifesting the neurological signs.

Subacute Hypermagnesaemic Tetany: this is the duration of the onset of clinical signs or 3 to 4 days, so slow onset of signs so the animal is hyperaesthetic and there will be tremors of muscles and the ears pricked up, and the animal will have apprehension, and it may have some frenzy behavior and stamping of feet and the appetite may be sluggish and milky less, this adult dairy cow that is a high yielder cow presented with excitatory signs, and it has a frenzy behavior and it's a pricked up ear and it's a responding to the hyperesthesia that is excessive responsibilities to external stimuli. In the Sub-acute form, the major clinical signs are tremors and tetany of limbs, but whenever the animal is exposed to external stimuli or sudden excitement, the animal will go for tonic-clonic convulsion and it may die also.

Chronic Lactation Tetany: this is the common form we can see because it's a hidden cause for most of the downer cows. What happens is the animal will have only unthriftiness and then reduced appetite, and reduced milk yield, and can currently because of reduced hypo magnesium level, magnesium is important to source for a secretion of parathyroid gland hormone that is Parathormone that we have already seen in a Hypocalcemia class, so what happens is the chronic deficiency of magnesium may predispose the animal for hypercalcemia so hyper magnesium and hypercalcemia occurs concurrently and most of the time in making the animal downer cow, so it is an important cause for downer cow along with hypercalcemia, most of the time the animal will not respond to calcium treatment unless you include magnesium there will not be any response. So Tetany signs are common in this stage.

In this lesson, we have seen what is a Hypermagnesaemic is a Pathogenesis of Hypermagnesaemia and various stages of clinical manifestation like Acute form, Subacute form, and Chronic form, mainly Neurological signs and concurrent occurrence of Hypocalcemia in cattle.

Download

PDF: Pathogenesis and Clinical Findings

3 Diagnosis and Differential Diagnosis

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Transcript

So today, we are going to see about lesson-3, before that in the previous lesson, we have seen about the Pathogenesis of Hypermagnesaemia and the various risk factors, How it causes hyper magnesium and the Clinical manifestation that is a major form is Acute Hypermagnesaemic characterized by neurological signs, tonic-clonic convulsion Tetany, and a Subacute form that is a more gradual onset of clinical signs, and the common Chronic form which is only unthriftiness and reduced appetite and milky and this form is always accompanied with hypocalcemia and most of the time it will predispose the cattle to go far downer cow syndrome.

And today in lesson-3, here we are going to see about How to diagnose the Hypermagnesaemic based on the various laboratory techniques at the field level.

The major pathogenic in Hypermagnesaemia is reduced serum magnesium level, so estimation of Magnesium from the blood is an important diagnostic too, normal value is 1.7 to 3 milligram per deciliter. So in Hypermagnesaemic, the levels will be less than 1.7.

To occur Tetany and convulsion the value should be too low, that is less than 1.2 and since most of the time Hypermagnesaemi concurrently there will be hypocalcemia, estimation of Calcium is

also important, always there will be Subclinical hypercalcemia that is less than 7.5-milligram deciliter of calcium will be estimated apart from this there will be release or increased Acute-phase inflammatory response you can see, that is a severe leukocytosis also you can appreciate in a Hypermagnesaemic animal. So in addition to serum analysis of magnesium since the main Homeostasis is the kidney, whenever there is Hypermagnesaemia, you may expect a reduction of magnesium in the urine, so the filter useful is a Xylidel test, so the urine magnesium decreases that indicates the animal is having Hypermagnesaemia.

So, one challenge is that field the animal is given is on grazing land and the animal shows severe neurological signs, you cannot collect even a blood sample, and most of the time the animal may be found dead. So if the animal is found dead, the diagnosis purpose will be to collective various samples and if you have a suspicion of Hypermagnesaemic here we need to collect the CSF fluid and we need to estimate the magnesium level, because CSF is a predictor for a Hypermagnesaemia and we can collect CSF up to 12 hours of death, that is reliable so you can estimate the magnesium till the even after 12 hours of the death of carcass animal and so the collection of magnesium from atlantooccipita region is more reliable than the Lumbar puncture. So the values are less than like it's almost equal to serum magnesium, if the values are too low 0.4 or 0.5 milligrams it could have died of convulsion and other severe neurological signs.

If the animal doesn't have a convulsion, then it is in subacute form, you can estimate the magnesium from even Vitreous humor, so you can use a 14 gauge needle to collect CSF from Vitreous humor, even in a dead animal also Vitreous humor sampling is useful to estimate the magnesium level and to prove Hypermagnesaemia and by adding formaldehyde to the sample we can estimate the still prolong the estimation of magnesium level in the prosumer fluid. Since Hypermagnesaemia occurs mainly it is manifested as a neurological disease as I said earlier mainly Tetany is stiffness of limb and the excitatory signs like prickly ears and that is an

uncontrollable neurological sign like frenzy expression and then very difficult to control so these are exciting neurological signs should be differentiated from similar diseases like mainly the common neurological adult dairy cattle acute Lead poisoning, there also you will get neurological signs and another important thing in a high yielding dairy cattle- nervous signs of like the frequent licking of body, frenzy behavior, and pica and other signs, that is a nervous form of ketosis is also important.

And another important this is to be differentiated that is an important neurological disorder at the field level is Rabies; in Rabies also you will get excitatory signs, like attacking others and the animal is uncontrollable and it's very difficult to restrain frequent getting up and walking. In addition to the neurological sign, each disease will have the Pathogens based on that we need to differentiate and mostly in western countries other important neurological signs to be differentiated are moments of spongy form Encephalopathy.

In this lesson, we have seen how to Diagnose Hypermagnesaemia by various laboratory techniques by estimation of Magnesium from the serum and then the estimation of Magnesium in the urine and then in dead animals especially CSF fluid and Vitreous humor.

How they are useful in the Diagnosis of Hypermagnesaemia as well as What are all the Diseases to be differentiated from Hypermagnesaemia especially Rabies, Lead poisoning, and Ketosis?

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4 Treatment and Control

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Transcript

In the previous class, that is a lesson-3, we had a discussion on Hypermagnesaemic- How to Diagnose based on various laboratory techniques like Serum analysis, Urine analysis, and then in a dead animal usually how to Diagnose based on a series of magnesium status as well as a Vitreous humor level of magnesium.

And today's class which is a lesson-4 is an important part of the lecture because of How to Manage Hypermagnesemia, so here in this class, we are going to see about the various route of Magnesium administration and How to Prevent and Control the Hypermagnesemia.

The important part of Treatment is Intravenous Magnesium administration, that is since most of the time Hypercalcemia and Hyperminosia occur concurrently, it is the best choice of Treatment and commercially available preparation containing Calcium Boro Gluconate 25% and then a Magnesium hypophosphate 5%. So this is very widely available in the market, you can go for this combined form for concurrent Hypermagnesemia as well as Hypercalcemia.

Apart from combined therapy, you need to go for a specific treatment that is a magnesium administration in various forms Magnesium chloride, Magnesium sulfate, and Magnesium oxide,

there are different preparations, but the one thing important in Magnesium administration is we need to prepare the solution that is magnesium especially if you are going to use Magnesium sulfate, crystals are available we need to dilute with distilled water and prepare the IV solution. Now since the Magnesium sulfate causes severe medullary depression, so you can very very carefully we need to administer the Magnesium sulfate as a sole treatment so the recommended dilution is you can use a 20% solution of Magnesium sulfate as an intravenous injection and you need to monitor the respiration of the animal also. So always you should have Calcium as an antidote for Magnesium, whenever the animal is having respiratory depression. Then you can go for even Subcutaneous injection for maintenance of a Magnesium at the blood serum level, so there are various levels like you can go up to 20 ml of 50% solutions are suggested, if you go for a Subcutaneous injection. Apart from that IV and the Subcutaneous injection, even there will be better absorption of Magnesium at the intestine that is rectum, so rectal enema, that you can give about 50% solution of that is Magnesium chloride- 30 gram in the water you can administer as a rectal enema, so it will give an added advantage to the maintain the Magnesium level in the blood. So apart from parental injections, the supplementation by oral route, there are various preparations available, you can go for a Magnesium oxide or Magnesium chloride like that. You need to go for drenching if you go for overdosing, it may cause diarrhea. So you should give careful dosing and drenching in adult cattle.

So in this video you can see how high exited the signs of a cattle with that is a Hypomagnesemic sign, after treatment, the animal had a normal appetite and it is controllable, and the excitatory signs are decreased.

So in this case also, you can see in the excitatory sign before Treatment and after Magnesium and Calcium administration, you can see the favorable response in the animal become normal.

This is the most important part of the Management of Hypermagnesemia because most of the production disorders we

need to *Control* by various factors, which cause Hypermagnesemia. So as you have seen in the earlier classes how the Hypermagnesemia may occur there is a factor, mainly the cereal grass pasture, that is an increase in the Potassium level of lush green pasture as well as the cereal grass, and then the increased urea top dressing and a fertilizer top, these are all the cases.

Another thing India, whenever the cattle are allowed for grazing so the high yielding animal should not be allowed on a lush green pasture, so rotational grazing system should be followed and apart from that you can go for a Magnesium oxide supplementation orally and there should be sufficiently dry matter intake that is concentrated, should be given to the animal to increase the absorption of Magnesium. And to increase the Sodium Potassium ratio, we can supplement Salt blocks that are available, so the Lactating cattle can be supplemented with this, they are allowed to lick on the salt block, which forms the source of Sodium as well as Magnesium thereby improving the absorption in dairy cattle.

Magnesium-containing solutions can be mixed with feed especially grasses.

The main disadvantage of Magnesium supplementation; oral supplementation is its very less palatability, so to improve the palatability along with the magnesium-containing preparation *molasses* should be added to the feed, to increase the feed intake of the cattle.

Apart from that *Pellets* are available, that is Magnesium-rich pellets that can be fed to the cattle. So in western developed countries, a normal dairy cattle practice, what they are doing in to control Hypomagnesemia Magnesium Bullets, Magnesium-rich bullets are placed in their reticulum, so that it will release the magnesium very slowly and avoid the occurrence of Hyper magnesium. Apart from that, you can go for a top dressing of a Magnesium spray on the fodder on pasture land. So, in this lesson-4, you have seen that is How to Treat Hypomagnesemia, mainly you just remember Calcium along with the Magnesium administration will give a favorable response in most the dairy cattle. And apart

from that followed with Magnesium sulfate solution by IV injection, so it should be prepared and a high concentration should be avoided because it may cause severe material depression, and animals will die of respiratory failure.

The Subcutaneous route is also suggested and a rectal enema also improves the Magnesium absorption and ...

In addition to that and the Control practices, there are various Magnesium preparation, and supplementation along with *Molasses* as well as we need to increase the dry matter intake, top dressing of a Magnesium, Magnesium Bullets are suggested in the prevention of Hypermagnesium in cattle.

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PDF: Treatment and Control

5 Hypomagnesaemic Tetany in Calves

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Transcript

Now we are going to see about lesson-5, before that we will see what we have discussed in the previous class, that is- What is Treatment for Hypermagnesaemia and which is mainly Calcium along with the Magnesium containing solutions or a preferred drug of choice.

Apart from that injectable Magnesium Sulfate solution of 10 to 20% by IV injection followed with a high concentrated subcutaneous injection even rectal enema of Magnesium containing solution improve the absorption of Magnesium. And supplementation of Magnesium Oxide and Chloride acid drench but only excess feeding of Magnesium may cause diarrhea, so overdosing should be avoided, and the control measures top dressing of foliage or pasture land with a Magnesium containing solution and then a Magnesium salt lick, and then magnesium bullets and a high concentrate diet and the rotational grazing of animal on the pasture land.

Finally, this is the last session of Hypermagnesaemia, lesson-5, which is a **Hypermagnesaemic Tetany in Calves** and we are going to see about how it occurs in calves and What all the Etio reasons for Hypermagnesaemia and Clinical sign Manifestation and the

Management. That is a Treatment of Hypermagnesaemia here in calves, so as I said earlier Hypermagnesaemia occurs in 2 forms:-

that is in *Lactating dairy cattle*– it mainly occurs due to decreased availability of a Magnesium and it causes Acute to Chronic form.

Whereas in *Calves*– it is mainly a calves age of a 2-4 month it occurs and unlike adult cattle here the absorption occurs at the level of a small intestine and whenever diarrhea and animal are done only on milk it predisposed them to go for hypermagnesemia, always hypermagnesemia here also occur concurrently with hypocalcemia.

Why there is Hypermagnesaemia in the Calves- one interesting thing is cows in preliminary calves, if they are fed only on whole milk, so no other roughage or concentrate is given, even after two months which fed on only milk source, are sometimes they are raised on a milk replacer diet and or else with the poor quality of roughage food, so all these may cause a dietary deficiency of Magnesium because milk is a poor source of Magnesium. So the calves that are fed on only this milk replacer diet, are mainly predisposed to Hypomagnesemia.

Apart from that if the calves are raised on a dry bedding material, that is wooden shavings or anything so the constant chewing causes excess loss of Magnesium in the saliva thereby the availability of magnesium to the animal decreases and more magnesium is excreted in feces and the animal has diarrhea. So in that condition also the absorption of magnesium in the intestine decreases and it predisposes the animal to go for Hypermagnesemia.

So and the absorption of Magnesium from the Intestine, that is immediately after birth, the absorption will be 85% but it slowly decreases within two months period as low as 30% in three months period the absorbing capacity or the level of the intestine decreases, that is why the disease occurs if the animal is only maintained on a milk diet and then without any roughage or concentrate.

In the newly born calf, there is rapid absorption of Magnesium from the small intestine nervous and it slowly decreases as the age advances.

So whenever the animal is maintained only on milk which is a poor source of magnesium, then there will be a composite mechanism by the way of mobilization of magnesium from the bone, but they're also the reserve capacity decreases as the age advances. So the Tetany signs are mainly seen when the serum level of Magnesium decreases by less than 0.6 milligrams per decilitre. So the Clinical findings absorbed in the Hypomagnesium calves are like they are in Cattle only. So there is an anxiety sign, all the neurological signs, initially, the animal will have mild tetany tremors of muscle, shaking of the head, and then a pricked ears, and the animal will have circling and even mild external stimulation will agitate the animal, it will go for ataxia and other signs. Later on, the increased muscle twitching followed with an animal will have severe tetanic and it will fall down, it will have a tonic-clonic convulsion, frothing of mouth, urination defecation, and opacity on exposure, whatever the convulsive series of signs absorbed in your typical conversion, you can see in calves, apart from that if the animal may have diarrhea due to the feeding of a milk replacer diet.

So in this, you can see very well appreciate the tonic-clonic convulsion and then opacity on exposure and all the excitatory signs in a calf affected with Hypermagnesaemia. So in this, you can see a delta calf showing stamping of feet, the excitatory sign in Hypermagnesaemia.

See how to diagnose Hypermagnesaemia because neurological signs we need to confirm there are other various diseases also may commonly occurring neurological disorders in calves are *Polio Encephalomalacia*– that is a thiamine deficiency, hypovitaminosis and then could Lead poisoning and Intro toxemia, Rabies. So to differentiate this we need to go for analysis or to prove the Hypermagnesaemia here we need to collect the serum sample and the levels less than 2 milligrams the animal will have neurological signs and titanic signs very low less than 0.6 milligrams you will get a convulsion. So the most important part of Hypermagnesaemia because one way of diagnosis of Hypermagnesaemia at the field

level suppose calves are presented with diarrhea, the neurological signs, then we can try Magnesium injections.

So IV solution of 10% Magnesium sulfate, may give an immediate response to the treatment followed because it is transient and the response to bone resorption is also very low then we can go for subcutaneous injection as well as a rectal enema of magnesium-containing solution. Apart from the magnesium supplementation in the form of oral drenching is recommended in Hypermagnesaemia calves.

So, in this case, you can see the calf- it's represented with diarrhea and then it had recumbency and a crawling and this goes after Treatment you can see the very good improvement, it can stand and walk normally and it had other clinical signs become normal.

In this series of lessons on Hypermagnesaemia, here I think you could have a better understanding of Hypermagnesaemia. So in a nutshell- what are all the points to be remembered in Hypermagnesaemia are :

The Lactation Tetany- is common in high yielders, mainly those that are fed with cereal grain crops lush green fodder and then the feed that is rich in fertilizers of potassium-rich fertilizer or urea containing fodder and mainly it altered the sodium-potassium ratio so they reduced absorption of magnesium and the common manifestation of Hypermagnesaemia and neurological signs. So Acute Hypermagnesaemia, you will get sudden mortality before an animal dies there will be severe tonic-clonic conversion, you can see and subject you to Chronic forms are common in Sub-acute form the onset of signs is more gradual whereas in Chronic Hypermagnesaemia there is unthriftiness and mostly it is a concurrently occurred with the Hypocalcemia and causing downward cow. So you always say whenever you manage Hypocalcemia, Hypermagnesaemia also should be included.

And the **Diagnosis** of Hypermagnesaemia is mainly by serum level of estimating the magnesium in the serum and a dead animal

especially CSF fluid and vitreous humor or for diagnostic importance.

Then the common neurological disorder to be **differentiated** with Hypermagnesaemia is Acute lead poisoning, Rabies, Nervous form of Ketosis is a metabolic disorder.

The **Management** mainly Hypermagnesaemia usually combine the solution of Calcium and Magnesium preparation should be used followed by a Magnesium Sulphate IV solution whenever you go for IV preparation always you should carefully monitor the respiratory sign, it will cause severe sudden respiratory arrest, so that diluted 10 to 20 magnesium sulfate suggested for IV preparation and high concentration of even 50% suggested for subcutaneous injection even Rectal enema is useful in improving the blood magnesium level.

And in the **Control**, we need to go for supplementation of a salt lick as well as a top dressing of foliage with a Magnesium containing solution and overall magnesium-drenching solution.

And finally, the important part of Hypermagnesaemia is the **Hypermagnesaemic Tetany** in calves that are maintained only on a milk-replace diet or whole milk and are maintained on a rough paddy bedding material, predisposed animal to go for neurological signs and the animal may die after convulsion. So in a pre-rumen calf after the second month of birth, they should be given sufficient concentrates and a roughage diet to avoid the occurrence of Hypermagnesaemia in calves.

Thank you!

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PDF: Hypomagnesemic Tetany of Calves