Fluid Therapy and Management of Clinical Syndrome in Cattle and Small Ruminants

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Contents

Part VI. Week 6: Intestinal Stasis in Cattle

Course Description

Livestock especially ruminants play a significant role in global food security, rural livelihoods and economies of developing nations. They are providers of income and employment for farmers besides providing diverse range of products such as milk, meat, hides, wool, heat and energy. The milk and meat from ruminants are also an important source of calories, high quality proteins and micro-nutrients. Under-consumption of animal proteins is linked to malnutrition and stunting with serious health outcomes globally. 17% of calories and 33% proteins consumed worldwide comes from animal sources. This necessitates to maintain good health of ruminants by adopting the best health management practices. Fluid therapy is an important component of management for many diseases that affect cattle and small ruminants. Dehydration and hypovolemia can occur due to decreased fluid intake or excessive fluid loss due to diarrhoea, haemorrhage, excessive salivation etc.

This agMOOCs course will enhance the knowledge and skill of the veterinarians across Asia and Africa to save animals and sustain productivity thereby enabling them to institute appropriate treatment protocol for the sick ruminants at field level thereby making the animal productive and enhancing the livelihood and increasing the economy of farmers.

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Course Content

- Fluid therapy in ruminants

- Anaemia and blood transfusion in ruminants
- Rumen and Omasal disorders
- Abomasal disorders
- Traumatic reticuloperitonitis and pericarditis in ruminants
- Intestinal stasis

Course Audience

- Practicing veterinarians
- Pre-final, final and internee veterinary students
- Veterinarians working in State Animal Husbandry Departments, NGOs, development departments and livestock farms
- Veterinarians working in State Veterinary Universities / State Agricultural Universities / Veterinary Colleges

Outcomes of this Course

- Gain knowledge and skill on clinical aspects of fluid therapy.
- Identify clinical features of anemia and blood transfusion protocol.
- Diagnose and treat of important clinical syndrome in cattle and small ruminants.
- Identify the etiology, pathogenesis, clinical signs and management of ruminal and abomasal disorders.
- Identify the etio-pathogensis, clinical signs, differential diagnosis of traumatic reticulo-peritonitis and pericarditis.
- Diagnose and manage ileus in cattle.

PART I

WEEK 1: FLUID THERAPY IN CATTLE

1 Fluid Distribution in Body and Assessment of Dehydration

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Transcript

Welcome to all! Now we are going to deal with fluid therapy in cattle.

This is Dr.Vijaykumar, Professor of Veterinary Clinical Medicine and Therapeutics, working in Tamilnadu Veterinary Animal Sciences University.

So we will split this course into 5 lessons and we will see what we are going to see in this class step-wise.

Now, first, we should know, why this Fluid Therapy has been taken?

To know this, we should know, what are the functions of Water?

Basically, Water is involved in

- the transport of nutrients and hormones,
- it helps in maintaining the body temperature,
- it acts as a source for eliminating the waste from the body,
- it provides input for the production of milk,
- saliva to the cattle, and
- moreover, it is important in maintaining the osmotic pressure

So what are the economic importance for this topic :-

Basically, any disease involves a reduction in feed intake and water and the animal becomes of it and there is dehydration. So because of this the animal also goes down in milk production and ultimately the farmer suffers a loss of production. In certain cases, the animal dies.

So farmer meets out with the reduction in milk production as well as mortality. This ultimately affects it's per capita daily, so that is why this fluid therapy is taken.

Adding on to this there is heat stress in the case of cattle as well as in the case of buffaloes when there is heat stress:

- there is a 0.23 kg per day loss in the feed intake,
- 1 kg of milk loss is there for every degree centigrade rise in the environmental temperature,
- a record says there is a 2% loss in milk production in India because of the heat stress,
- during humid and warm climate there is 50% reduction in the milk production, and
- certain animals like buffalo go for silent heat and we are not able to detect the estrum and conception becomes low.

We are taking Dairy cattle requires approximately 15 to 30 gallons of water. This shows how much water should be taken by cattle to maintain its maintenance and production. So, NRC cattle 2011, has given an indication of water requirement by a means of a formula which also includes the milk yield, sodium intake, and average temperature in the environment. In this slide, we are going to discuss, what are the lessons we are going to take up in this course? These are split up into 5,

1. In the first lesson, we are going to deal with Fluid distribution in the animal and how to diagnose dehydration.
2. Lesson number two, we are going to deal with the calculation of fluid deficit and what is the choice of fluid for us.

3. Chapter number three, we are going to discuss Acid-base imbalances (part I) and
4. continuation of that, we are going to deal in chapter number four, because that is a very big fairly big topic (part II), and
5. chapter number five, we are going to deal about Heat stress fluid therapy in cattle now we are going to discuss in the first lesson.

Fluid therapy in cattle:

Now we are going to discuss in the first lesson:

Fluid therapy in cattle namely the fluid distribution in animals and how to assess dehydration. Of the total body weight, we already understand 60% is composed of water and 40% is others. The 60% is composed of 40% intracellular fluid and 20% in extracellular fluid. We are more bothered about extracellular fluid because this constitutes 5% plasma, less than 1% transcellular fluid (<1%), and 50% interstitial fluid.

Why we are bothered about plasma, because we cannot enter into the animal without plasma, so transcellular fluid is less than 1% (<1%), what is that these are the meningeal fluid, cerebral sternal fluid, and the fluid in between the joints.

The take-home point is of the 60% water – 20% ECF which is 1/3rd and 1/4th of ECF is plasma, so to put it in nutshell, the total body water 1/12th is the plasma. why I am restressing is we need this for the calculation of fluid deficit in the animal.

Total deficit = TD

ECF = TD * 1/3

PLASMA = ECF * 1/4

OR = (TD * 1/3) *1/4

= TD * 1/12 (Showing the Slide)

So we are explaining the Sunken Eyeballs in the cattle, by means of a cattle, these two beautiful pictures explain how the eyeball has gone deep inside the orbit which is called as enophthalmos, which is important clinical sign in dehydration.

The next slide shows the Skin Tent test: how to do this Skin Tent

test, pull the skin on the neck, and then assess the time for the pulled skin to regain the original status this is called a Skin Tent test, one of the important parameters we use for assessment of dehydration, and

Next slide we are going for how to calculate Capillary Refill Time: this beautiful video shows, how to calculate the Capillary Refill Time, so we have to press the gum and allow the gum to regain its original color. so we are pressing the gum to make it blanched and assess the time for the blanched mucous membrane to become the normal color, so you calculate the time as

- 1000-1
- 1000-2
- 1000-3
- 1000-4
- ……

At what point exactly the blanched mucous membrane regains the original color, is taken as the second so don't look for a sports watch or electronic watch or android watch to calculate it because you will as you move like this you get spondylitis, so best is calculate 1000-1,1000- 2,1000-3,1000-4,…… and this is a universal way to calculate the Capillary Refill Time in case of animals. T

his is a video that shows CRT in a calf, as a beautiful video, we will reassess it, because this is more important for assessing the dehydration case of the calf. So, this the person clinician is blanching the mucous membrane and counting it to assess the CRT in case of the calf.

(Showing the Slide)This shows assessment of dehydration on the left side we have got % of dehydration and on the right side we have got the clinical signs:

- Less than 5%(<5%), the clinical signs is not detectable,
- 5-6%, there is a subtle loss in skin elasticity, which means the increase in the Skin Tent time,

- 6-8%, there is a marked loss in skin elasticity, that is a Skin Tent test, which is more than 2-4 seconds and Capillary Refill time is 2-3 seconds, the mucous membrane becomes pink and hardy and sticky,
- 8-10% , please note this point is more important, 8-10% the Skin Tent is going more than 5 second , Capillary Refill time is 4-5 second and there is a marked Sunken Eyeballs what do you call as enophthalmos and the mucous membrane becomes dry,
- The dehydration is 10-12%, there is a skin elasticity increased to 6-10 seconds, capillary refill time goes for 6-8 seconds, there is a severe enophthalmos and mucous membrane becomes dry,
- the last one which is 12-15% dehydration skin elasticity becomes 20 seconds, marked and capillary refill time is more than 8 second(> 8 second), enophthalmos, the animals Recumbent
- shows early signs of shock, you have got fixed eyeballs, moribund, and death is imminent.

This is more important please note, down these clinical signs because it is needed for pronouncing the prognosis to the farmer and intensive critical care has to be taken if at all if you want to save the life of the animal.

(Showing the Slide)So this table shows the dehydration in the calves, more or less similar to that what we have seen in the case of cattle.

Here we have got the 4 columns: first is the percentage % dehydration, next is Sunken Eyeballs Skin Tent test and Mucous membrane.

Please note down, the last two rows which are important for us, because 9-10%, where we need to give your Fluid therapy by intravenous roots, may be less than that we can face the fluid therapy by oral root, wherein 9-10% we get the Sunken Eyeball and the % mm of the Sunken Eyeballs going away from the orbit is more than 5 mm(> 5mm), in case 11-12 % the gap is 5-10 mm gap.

Dehydration in Calves:

% Dehydration	Eyeball Status	Skin Tent (in seconds)	Mucus membranes
0	None	<1	Moist
1-5	None to Slight	1-4	Moist
6-8	Slight Separation	5-10	Tacky
9-10	<5 mm gap	11-15	Tacky to Dry
11-12	5-10 mm gap	16-45	Dry

please note down this is dehydration in calves, so which is a thumb rule, the Dehydration is equal to the degree of eye recession into the orbit in mm multiplied by 1.6. I.e.,

Dehydration = (degree of eye recession into the orbit in mm) X (1.6)

This will give a rough idea of how much fluid has to be lost. please again I repeat dehydration is equal to the degree of eye recession in millimeter multiplied to 1.6.

This gives a laboratory analysis based upon Packed Cell Volume (PCV), Total Solid (TS), and pH.

So we have given 3 rows, in this first row is a Normal and next one in which animal needs a Fluid Therapy (FT) and the last row which shows the Unfavorable, namely the PCV of more than 60% (> 60%), Total Solid more than 12% (>12%), pH of more than 7 or 7.1(>7.00-7.10), is a critical thing where the animal is having an unfavorable prognosis.

Laboratory Analysis

	PCV	TS	pH
Normal	30-40	6-7.5	7.35-7.45
Need FT	50-60	8-10	7.20-7.30
Unfavourable	>60	>2	7.00-7.10

(Showing the Slide) This slide shows the cattle which is in the standing posture becomes Recumbent and that shows how much severe is the dehydration and the next stage becomes lateral recumbency, the animal goes for death.

Coming on to summary, in this lesson 1, we have seen:

- What are the importance of Fluid Therapy?
- How it is being distributed in the body and
- We have taken the points in the calculation of the Dehydration in cattle and calves.

In the next lesson we are going to see:

- How to calculate the Fluid Deficit, and
- What are the choices we have for us in the field to meet out the Dehydration?

Thank you!

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PDF: Fluid distribution in body and Diagnosing dehydration

2 Calculation of Fluid Deficit and Choice of Fluids

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Transcript

Welcome back to Fluid therapy in the cattle.

In lesson 1, we have seen the distribution of fluid in the body and how to assess dehydration in the case of cattle and calves.

Now we are going to see lesson 2 in which we are going to see how to calculate the Fluid deficit and what are the choices of fluid we have to meet this dehydration.

Now the calculation of fluid requirement in the animal, there is a formula.

The fluid deficit in liters = the percentage of dehydration multiplied with bodyweight

= % dehydration * body weight

Now in this, we have got an example. Always when you solve with an example, it is best remembered in our mind. So we have solved 1 question practically. Please follow us. You can also solve it.

To calculate the Fluid deficit for a cow weighing 360 kg body weight and 10% dehydrated. In this problem we have 2 things in our hands already, what is called bodyweight 360 and there is a formula

fluid in deficit required is the percentage of dehydration multiplied with body weight, so substituting the available things in the formula, namely the 10% and this 360 KG, so if we multiply, we get 36 liters, so 36 liters is the dehydration requirement in case of this cattle.

Body weight = 360 kgs

Fluid deficit in litres = % dehydration * body weight

= 10% * 360 = 10/100 * 360 = 36 litres

Substituting this in the water distribution, as we have seen in the last class, the total deficit is 36 liters, ECF is the total deficit multiplied by one-third. So substituting 36 multiplied with 1/3 is 12 liters. So ECF deficit is 12 letters.

Total deficit = 36 litres ECF = TD * 1/3 = 36 * 1/3 = 12 litres

Now please substitute this into the Plasma, what is Plasma?

Plasma is 1/4 of ECF, so 12 liters multiplied by one-fourth, we get 3 liters.

Plasma = ECF * 1/4 = 12 * 1/4 = 3 litres OR

Plasma = TD * 1/12 = 36 * 1/12 = 3 litres

We can also do the plasma estimation by 1/12th of the total deficit. So what is called 36 / 12 we get 3 liters.

Why we have estimated this because Plasma requirement has to be infused into the animal because that is the one which is reflecting the severe dehydration. So this is already lost. It needs immediate pumping into the animal. How fast you can do it to the animals, there is a chance of restoring the circulating volume. Now the plasma requirement that you have calculated is 3 liters. Please give this requirement immediately within a matter of half an hour to one hour. So with 50%, you have to give it in the first 6 hours, namely, the 9 liters and the remaining 24 hours kindly give in the remaining 12 to 24 hours, so that is a thumb rule. If it is an acute case, give it more rapidly. If it is a chronic case, give it slowly. Now here we are going to see how to calculate the bodyweight because the bodyweight forms an important thing in the percentage of dehydration calculation.

So we have got 4 methods;

1. one is by so formula, 2. another is by Android application, 3. another is a weight measuring tape, and 4. 4th is the weighing machine.

Let us see about the formula, which is the commonly used Shaffer's formula. W = LG2 / 300 , where L is the length of the animal from point of shoulder to point of buttocks in inches, and G is the chest girth of the animal in inches.

(showing image in the slide) There is a beautiful photo in that which explains how to measure L and how to measure G. So substitute that in the formula. We will be able to get the animal's body weight. So, there are different applications available in the Google Play Store. You can download any one of them which suits you in this. (showing image in the slide) We have shown the TANUVAS FEED CALCULATOR, where there is an app, which you can see which gives length and girth. So you measure using a tape and substitute that in this app. What you have to do is, you have to move that dot towards the measurement which you have taken in the L as well as in G. The bodyweight is immediately given by the app, with that you can substitute in the formula, percentage dehydration multiplied with bodyweight, that is how it is made very simple.

(showing image in the slide) This shows how to measure the body weight using tape, this tape is a beautiful application where if you are going to look into the tape it gives the bodyweight immediately and there are measurements in centimeters as well as inches. What you need to do is, take the tape and measure the chest girth, as soon as you measure, it gives in centimeters and inches, immediately by the side of it, there is a bodyweight. So, this is very much applicable in the case of the field where it gives spontaneous body weight, but you need to know this tape is specific for different breeds, maybe for a jersey, it has got the different tape and HF different tape, and indigenous cattle we have got a different tape.

Calculation of Total Fluid requirement: this fluid requirement we have got 3 components;

1. one is the Volume of Replacement, which you have calculated based upon your formula, 2. number 2 is the Volume required for the Maintenance of the animal, and 3. the 3 is the Volume of the Production requirement.

So you add up all three, which gives the total fluid requirement of the animal.

Now, this gives take-home points; these are all the thumb rules, please make a note of it.

- Each litter of milk produced requires one liter of water
- for every rise in environment temperature, body temperature 1 degree, we require about 4.5-5 liters of water on a per-day basis, the normal body temperature is in the range of 101.5 – 102.5 F, so if there is an increase in one-degree rise in the body temperature, you have to supplement a minimum of 4.25 liters, this is the thumb rule.

Now let us work out with an example; where we substitute the Fluid requirement in the case of a cattle with a temperature of 103.5 degrees Fahrenheit and milk yield of 5 liters. Now, as you substitute for the elevated temperature of 1 degree Fahrenheit, we have to give 5 liters of water, which is the requirement is 5 liters for milk production for every litter of milk we need one liter of water. So substituting in that, we get 5 liters, 5 liters= five-liter for the temperature. we get a total of 10 liters. This is over and above the maintenance and dehydration requirement. Now we are going to see the importance of fluid therapy in adults and how we are going to face it. Most likely it is dehydration and does not happen to be a shock unless it is a severe case of sepsis or a very severely decompromised disease. It is usually negative base, excess and mature cattle are always likely to be alkalotic except in certain diseases like acidosis or case of diarrhea. And many of the conditions diseased condition or disorder is accompanied by hypochloremia, hypokalemia, and please consider hypocalcemia,

which is to substitute calcium. We are going to see the Routes of fluid administration; we've got 2 routes,

1. one is Parenteral Route and another is
2. the Enteral Oral Route.

When you consider Parenteral Route, IV is best and the replacement is immediate and more so in the case of neonates. In terms of the Enteral Oral Route, when the animal is not severely dehydrated, you can go for Enteral Oral Route, thinking the IV fluids are very costly. So first 4 to 6 hours, the corrective therapy is 100 to 150 ml/kg BW, and if it is for 20 to 24 hours, the maintenance therapy will be 60 to 150 ml/kg BW per 24 hours, you can give intravenous or oral.

Fluid therapy: here we are going to see the quantity, as we have taken from the percentage of dehydration. The rate of fluid therapy, if it is for routine, is 10 to 20 ml if it is a case of Crystalloid and in case of colloid, we are going to give at 5 to10 ml per kg body weight, this is when you are going to give as a routine.

But in case of a shock, the rate is different. For example, in the case of calves, 50-80 ml/kg IV you have to give and see that within 30 to 60 minutes the animal response. But the dose rate is different in the case of cattle which is 30 ml/kg/IV in the case of the first hour, so if you can increase the fluid rate, you can even go up to 50 ml, but the restriction should be in the IV set. In the normal IV set, we may not be able to reach even 30 ml/kg, that's why the rate we have decided to give at 30 ml/kg.

This gives Rapid Intravenous(IV) fluid administration, which has been compromised by restriction of the rate of fluid by the routine Intravenous fluid is being overcome by rapid intravenous fluid administration.

(showing image in the slide)What is beautiful in this, here the diameter of the IV set is more and we are going to choose an intravenous needle or hypodermic needle to the extent of 14 gauge. So there will be a Rapid fluid administration, in which you can

administer 50 ml/kg BW, that facilitates the fluid resuscitation therapy in case of shock.

(showing image in the slide) In this slide, we see a Buffalo receiving fluid therapy through Rapid Intravenous fluid administration, in the right slide you can see we have almost got 30 sachets of fluid, where the fluid goes very rapidly and the 500 ml of fluid takes roughly about 2 minutes 10 seconds. So you can imagine how much amount of time we can conserve.

Here we are going to see the Parenteral therapy, we have got the two fluids Crystalloid and Colloids.

Crystalloid: These are the solution that contains small particles that can easily go into the bloodstream to cells and also into the tissues. So these are used as maintenance.

Colloids: The colloids are the fluids that have got a larger molecular weight, which helps to retain the fluid within the circulating system.

Isotonic solution: These are called so because they have got 280-300 mOsm/liter and they are used for expanding circulating volume and replacing the actual fluid losses. These are the Routine fluids that we are going to use in our day-to-day life, namely the Normal Saline, which has got 0.9% sodium chloride(NaCl), again it is isotonic and provides sodium and chloride. The Ringer's solution is again isotonic, it provides Sodium(Na^+), Chloride(Cl^-), Potassium(K^+), and Calcium(Ca^{2+}). Next is Ringer's lactate solution, which is also isotonic, provides Sodium(Na^+), Chloride(Cl^-), Potassium(K^+), Calcium(Ca^{2+}) chloride, in addition it gives Lactate. Next is favorable which we are routinely using is DNS(Dextrose Normal Saline), which provides 0.9% sodium chloride and 5% dextrose. Again, it is isotonic.

Hypotonic solution: Here we have got 0.45% sodium chloride or have 0.25% sodium chloride. Here we've got 5% dextrose or 2.5% dextrose. These are called hypotonic solutions.

Hypertonic solutions: So these are to reestablish equilibrium in electrolytes and acid-base imbalances. In addition, we have got 10% dextrose, 5% protein hydrolysate, high electrolyte concentrates

like 5 or 7.5% sodium bicarbonate, calcium gluconate, or calcium borogluconate. These are all hypertonic solutions, these are given very slowly and rapid infusion causes catastrophic changes.

Colloids: so as we discussed in the previous slides, Colloids are those solutions, which contain large molecules, help to retain the fluids within the bloodstreams. So these are used for plasma volume expanding and they require less volume and short time and even blood or albumin or hypertonic solutions or what is called colloids.

(showing image in the slide)This slide shows colloid, on the left side we have got Dextran and right side we have got Gelatin. These are commonly used as colloids.

So we have seen the Hydroxyethyl Starch on the left side and right side you got blood both are called colloids which we are using for Fluid therapy. Oral dehydration; Oral restoration of Cattle, so in substitute to parenteral administration, namely IV, we have to go through the oral route. For the oral route, we need to have the types of equipment namely, the infusion pump, the tube that connects the infusion pump, and the Rumen tube, and we need to have a Rumen tube that is firm and strong. After assembling this, then we can pass the Rumen tube into the mouth we take utmost precaution not to damage the esophagus, once it is in the Rumen, we can connect the pre-set infusion pump into the Rumen tube, then you can start pumping the fluids of maybe 20 to 25 liters into the Rumen and it hardly takes 3 to 5 minutes.

(showing image in the slide)Here on the left side, we have an infusion pump, which we have been using in larger, maybe the horse that can also be used in the case of cattle, the only thing it has to be suitably adapted and attached to the Rumen pump, then it becomes very easy so that you can use the same pump for cattle as well as horses.

Now have known how to give the oral solution, pumping into the Rumen, we need to know what is the ORS composition. The ORS for the adult and the Calf are different.

For the adult we use;

- Sodium chloride-160 gram,
- Potassium chloride-20 gram,
- Calcium chloride-10 gram,
- propylene Glycol-300 ml and
- mix it with 20 liters of Water,

and then you can pump through the Rumen pump.

Calf: so it is

- Sodium chloride- 4gram,
- Potassium chloride-1gram, in addition, we have got
- Sodium acetate-4gram,
- Dextrose-10gram,
- mix it in 8 liters of water.

This can be pumped into the case of cattle and One more ORS is available for cattle for routine maintenance, which is called standard. We have got 7 grams of Sodium chloride, 1.25 grams of Potassium chloride, 5 gram of Calcium chloride, please mix it with 1litre water and this is going to give you 120mmol of Sodium chloride, 16.8mmol of Potassium chloride, and 4.5mmol of calcium chloride making Osmolarity of 287 mOsm per liter. You may wonder how we can pump in 20-25 liters into the Rumen. please note that Rumen volume is 13%-17% of the body weight, so it can easily accommodate 50-65 liters in an Indian cattle, which weighs about 350kg to 400kg bodyweight.

(showing image in the slide) So this slide gives a beautiful ORS Composition for Rumen impaction. What is that? Magnesium oxide 500 gram, mix it with 5 liters of water, make it to the homogeneous solution and pump it into the animal. So in addition to maintaining or compensating the dehydration by IV Fluid, you will see that the animal passes on dung taking precaution that there is no mechanical obstruction. Here we are taking an example of a

For Bullock, which has not been passed dung for 6 days and an Anorectic rectal examination reveals Mucus and the absence of

dung. So we have used this ORS impaction protocol, that is 500 grams Magnesia oxide with 5 liters of water and the animal passed dung.

(showing image in the slide)This slide shows left side is before treatment and the right side is after treatment for a Bullock with Rumen impaction.

For Calf, with less than 8% dehydration, you can go with ORS. With more than 8% dehydration it is a must, that you go fluid resuscitation with intravenous route. So less than 8%, we have got over his composition of 2.5-grams Sodium chloride, 1.5-gram Potassium chloride, 5-gram Sodium acetate, and 28-gram Dextrose mix it in 1 liter of water and give it 2 liters at a time and repeat it 3-4 times a day. So remember if it is less than 8% dehydration, go by oral route. More than 8% of dehydration go by intravenous route.

This ORS is exclusively for Recumbent Cows, where we have got Sodium chloride-44 gram Potassium chloride-180 gram, mix it with 24 liters of water and then you pump into the Rumen and it is a beautiful oral solution for the Recumbent cow because potassium happens to be one of the contributing factors for continued recumbents, so give potassium the animal is facilitated for ambulation.

In this lesson, we have seen how to calculate body dehydration and what are the choices of the fluids and what are the routes. The Intravenous route, the Parenteral route and effectively we have dealt with an example.

In the next lesson, we are going to see the fluid basic deficit, acid-base imbalance, and how to manage the dehydration or acid-base imbalance in the animals.

Thank you!

Download

PDF: Calculation of Fluid Requirement

3 Acid-base Imbalances and Rehydration Management Part:1

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Transcript

Welcome! back to the Fluid Therapy lesson.

In the last class we have seen, what are the choices of the Fluid, how to calculate this Fluid deficit, and what are the roots of administration- namely the intravenous root and the parental therapy.

In this class, we are going to see about

- Acid-Base Imbalances and
- how to do Rehydration Management in various disorders.

Now we have got a thumb rule:

for maintenance requirement, we have got 50ml/kg/day

Maintenance requirement = 50ml/kg/day and

the Fluid requirement in liters, there is a formula, Fluid requirement in liters is equal to surface area multiplied 2L/square meter

Fluid requirements in Litres = surface area * 2L/sq.m

So far, we have been calculating the Fluid deficit using

bodyweight, now we will have a comparison with the surface area of the body.

Now let us solve this, by taking an example:

Calculate the Fluid requirement in a 450 kg body weight?

Let us proceed, with how to calculate based upon the body weight and surface area.

Kg	SA(Sq.m)	Total Fluid requirements
1	0.10	0.20
5	0.29	0.58
10	0.46	0.91
20	0.72	1.44
50	1.32	2.64
100	2.10	4.20
250	3.83	7.65
450	5.64	11.28
500	6.04	12.10
800	8.24	16.50
1000	9.55	10.10

This chart gives the conversion factor from kg to surface area and the fluid requirement.Again this last column is based upon the formula surface area multiplied by 2 liters.

so substituting this namely the 450 kg body weight, the surface area is 5.64 and the requirement is 11.28 liters, so fluid requirement based upon the surface area is 5.64 multiplied by 2, it is approximately 11.28 liters but if you take the same thing based upon the body weight, so bodyweight multiplied by 50 ml per kg, it will be best equal to 22.5 liters. so consider surface area given 11.28 liters, whereas body weight given 22.5 liters, consider there is a hundredfold increase from 10 kg to 1000 kg but there is only 20 fold increase if you are taking to the surface area.

So this is to stress there is a non-linear relationship between body weight and maintenance Fluid requirement. why this is being emphasized, as the bodyweight keeps on increasing maybe 900 or 1000 as it goes the more important is you have to concentrate on the Surface area.

Now we will see step-wise, how to deal with the Acid-base Imbalances in various disorders.

- The first ones are D-lactic acidosis, urinary tract diseases, SI(small intestines) triangulation, obstruction, and choking. so, the disorder is mainly the metabolic acidosis low bicarbonate (HCO3)and there is severe dehydration.
- So the treatment or the management is you have to give sodium bicarbonate initially followed by isotonic fluids or electrolytes.
- Here we are going to see what is the changes we encounter in Neonatal calf diarrhea: the changes are metabolic Acidosis, low sodium bicarbonate, there is severe dehydration, loss of sodium Na, and that is hyperkalemia.
- what is the treatment? The treatment is a mixture of isotonic saline, isotonic bicarbonate HCO3 and please add on 5% dextrose because there will be hypoglycemia in the calf. If you want to save the calf, please add on 10 grams of glucose per liter of water.

We have been discussing Isotonic sodium bicarbonate, how to get that, so use 5and ½ of ampoules of 10 ml each, so it makes about 155 ml, mix it in 845 ml of water, so this will become Isotonic sodium bicarbonate. This can be used wherever there is an Isotonic sodium bicarbonate requirement, you can use it intravenously.

A base deficit in calf, there is a thumb rule, if it is less than 1 week(<1 week), it is 10-15 milliequivalent per liter(10-15 mEq/l), if it is more than 1 week(>1 week), the Base deficit is 15 to 20 milliequivalent per liter(15-20 mEq/l), kindly substitute this in the requirement of acid[1]base balance and you will get the result.

What is the acid-base balance or changes you encounter in RDA(right side displacement of amazon); impaction, torsion, vagal indigestion, Caecal dilatation, or Caecal torsion, the predominant change is metabolic Alkalosis, which is reduced chloride, and severe dehydration? And the Treatment: use of balanced electrolytes and high potassium with chloride and add on acidifying solution.

Let us see what is the acid-base changes in Intestinal obstruction; the predominant change is metabolic Alkalosis, which is reduced to chlorine and reduced potassium.

The Treatment: use balanced electrolyte, high potassium, and chloride with an acidifying solution. Acute diffuse peritonitis, the changes; Dehydration and slight metabolic Alkalosis,

Treatment: please use balanced electrolytes in heavy quantity.

Per acute mastitis; here we encounter severe dehydration, Mild electrolyte defect, hypercalcemia and if there is diarrhea you also encounter acidosis.

So the treatment will be balanced electrolytes for 24 to 48 hours.

Ruminal Alkalosis/Putrefaction; here you use oral acetic acid, please use a stomach tube or rumen tube, to pass on this acetic acid into the rumen.

So the percentage is 2.5%, use 1-2 liters orally for 2-3 days. In addition to that, you need to use sodium chlorate, a dextrose normal saline intravenous. For the Putrefaction, you use an oral antibiotic namely tetracycline at 20 milligrams per kg body weight for 2-3 days followed by cut transplantation. If it is very severe go for Rumenotomy.

We have been discussing the Hypertonic Saline solution. So let us consider how to prepare Hypertonic saline solution. So this is 3-7.5% sodium chloride(3-7.5% NaCl), prepare this percentage is, highly useful for hemorrhagic, septic, and endotoxic shock. Now, what is the rate? The rate is 3-5 ml per kg(3- 5ml/kg) intravenous, it produces a very good effect, but don't forget to use Isotonic solutions following Hypertonic saline.

In the case of Ketosis, we are using dextrose, hypotonic dextrose 20/50%, in addition, we may be using Isoflupredone, what happens

is, this Isoflupredone causes excretion of potassium and there is hypokalemia.

(showing the slide) In the next slide, now we can see the twist, the neck twist in the case of cattle which indicates hyperkalemia. So don't forget if you are using Isoflupredone continuously so there will be hypokalemia and substitute this potassium so that the animal becomes normal. Hypokalemia, in such cases you need to supplement potassium, please administer potassium very slowly even slower than the calcium administration, the rate should be very slow.

The availability is 10 ml ampoules and this hypokalemia occurs, even during treatment for rumen lactacidosis, and

(showing the slide) This next slide also beautifully shows, how the neck is twisted that is very muscle becomes very weak animal becomes Recumbent.

The rate is 0.5 milliequivalents per kg per hour (0.5 mEq/Kg/hr), never increasing beyond this rate. So 1.15 percent potassium chloride(1.15% KCl) gives about 3.2 ml per kg per hour (3.2 ml/Kg/hr). So there is a Scott thumb rule, which is indicated in the table, where the first column is the Serum concentration of potassium, the second is how much milliequivalent of potassium you are going to dissolve in 500 ml of saline, and the last one is the rate ml/per/kg/hr.

- 1.15%KCl = 3.2ml/kg/hr
- = 0.5 mEq/kg/hr Serum concentration mEq/500ml ml/kg/hr

Screen concentration	mEq/500 ml	ml/kg/hr
<2	40	6
2.1-2.5	30	8
2.6-3.0	20	12
3.1-3.5	14	18
3.6-5.0	10	25

Kindly refer this Scott thumb rule and then you can determine the milligram or milliequivalent of potassium, to be dissolved in one sachet and you can control the speed using the last column. If you are going to increase the speed then there will be the death of the animal. In many cases we use dextrose, rapid infusion of dextrose causes hypophosphatemia so whether in your case of ketosis you may be using this, or as a nutrient supplement to get effective oxaloacetate you may be using dextrose.

In such cases so see that hyper positivity does not occur if it occurs, you have to supplement phosphorus. In this lesson, we have detailedly seen The acid-base imbalances and the electrolyte requirement in various disorders of ruminant. The important disease for disorder we have seen in this class. In the next class, we are going to see how to calculate the ml of bicarbonate required based upon the formula and the millimole of bicarbonate equal to 0.3 multiplied by body weight multiplied by base deficit, because that calculation is more important for field conditions.

Thank you!

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PDF: Acid-base Imbalances and Rehydration Management

4 Acid-base Imbalances and Rehydration Management Part: 2

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Transcript

Welcome! back to Fluid therapy in cattle.

In the last class, we have seen about the various electrolyte acid-base imbalances and how to tackle that. In this class, we are going to see how to calculate the bicarbonate requirement in specific conditions.

(showing the slide)So, in this slide, we are seeing the Rumen Lactacidosis, which is mainly because of rapid ingestion of fermentable carbohydrates in animals, where we get roman lactic acidosis.

How to calculate the bicarbonate requirement? It is based upon the formula : millimole of bicarbonate required is equal to 0.3 multiplied with bodyweight multiplied with the base deficit.

mmol of HCO3 required= 0.3 * bodyweight * base deficit

This formula shows how to calculate the base deficit, but in field conditions, it is very difficult to calculate the base deficit, as the base deficit can be assessed by a blood gas analyzer, which is not available in the field. If it is available to fine, if it is not available, we can use the thumb rule.

We can classify this type of acidosis into Mild, Moderate, and Severe.

If it is a Mild acidosis, you can use the best deficit as(-5), if it is Moderate use the base deficit as (-10), if it is Severe use the base deficit as(-15). If you substitute this in the base deficit formula: 0.3 multiplied with bodyweight multiplied with the base deficit, we will get the millimole of bicarbonate requirement. Now we will always understand when we are going to deal or substitute in a case of an example.

Now let us take an example:

Calculate the bicarbonate required for 100 kg cattle.

So we know the formula, the availability of the formula is, bicarbonate requirement is equal to body weight multiplied with 0.3 multiplied with base deficit

HCO3 requirement = bodyweight * 0.3 *base deficit

considering this cattle as a Moderate acidotic or Moderate Rumen Lactacidosis, we substitute 100 kg multiplied with 0.3 multiplied with (-10), which will give you (-300) millimole of bicarbonate,

= bodyweight * 0.3 *base deficit

= 100 kg * 0.3 * (-10 mmol) = -300 mmol

This is the required bicarbonate that has to be given to this animal.

On the contrary to the calculated millimole, the availability is only in ml. What is that? We have got 5% ampoule as a -10 ml ampoule or 7.5% 10 ml ampoule or 25 ml ampoule. So the calculated is millimole or bicarbonate, but it is available as a percentage. Infield condition, how to calculate millimole from the percentage solution? There is a thumb rule:

Each ml of 7.5% = 0.89 mmol of bicarbonate

So for 0.89 mmol, we need 1 ml of 7.5% solution

For 300 mmol we need = (300 * 1)/0.89 = 337 ml

Thus 337 ml of 7.5% NaHCO3 will provide 300 mmol of bicarbonate

Each ml of 7.5% will give you 0.89 millimole of bicarbonate, so substituting this in the example which we are dealing with, for 0.89

millimoles, we need 1 ml of 7.5% solution, to rewrite this so 300 millimoles we need 337 millimoles or ml of sodium bicarbonate. Thus in a correct 337 ml of 7.5% sodium bicarbonate, it will require 337 ml re-emphasize this thus 337 ml of 7.5% sodium bicarbonate will provide 300 millimoles of bicarbonate. Here we are going to see an example with a small ruminant namely the goat weighing 10 kg, having a Moderate acidosis:

So the formula we already know, bicarbonate requirement is equal to body weight multiplied with 0.3 multiplied with the base deficit.

HCO3 requirement = body wt * 0.6 * base deficit

= 10Kg * 0.6 * (-10 mmol)

= -60 mmol

Kindly note in the case of a small ruminant, you have to substitute 0.6, so the bicarbonate requirement is equal to body weight multiplied with 0.6 multiplied with the base deficit. That is the only difference, instead of 0.3 you are going to put as 0.6. So, substitute that 10 kg multiplied with 0.6 multiplied with (-10), we will get (-60) millimole because it is a deficit, it is in the negative sign. So 60 millimole is required for this 10 kg goat. The difficulty faced in the cattle is also known in the case of goats, so substituting bicarbonate requirement is equal to (-60) millimole. So for 0.89 millimoles we require 1 ml of 7.5% solution, for 60 millimoles will require 67.4 ml, this is based upon your 7.5% solution. Suppose, if your own availability is a 5% solution, what you will do? the 5% will supply 0.6 millimoles of bicarbonate. so substituting that 0.6 millimoles, we require 1ml of 5% solution, for 60 millimoles we require 100 ml of 5% sodium bicarbonate. Bicarbonate requirement is (- 60) millimole, if you are using 7.5% sodium bicarbonate we need 67.4 ml, if you are using a 5% solution we require 100 ml of 5% solution. But some people substitute this acidosis or compensate this acidosis with Lactated Ringer's Solution(LRS), if it is to be given by Lactated Ringer's Solution(LRS), kindly note there is a thumb rule 500 ml will supply only 25 millimoles of bicarbonate. To rewrite 1 ml will supply 0.05 millimole of bicarbonate, so we need 1.2 liters of Lactated Ringer's Solution to compensate for this 60 millimole. So for 10

kg goat, we need 1.2 liters. You can understand our Indian cattle weighs about 350 to 450 kg bodyweight, you can calculate how many sachets of 500 ml of Ringer's Lactate is needed to effectively treat this condition. So, if at all if it is the acidosis and if you want to compensate or neutralize that, kindly give sodium bicarbonate at the rate of body weight multiplied with 0.3 multiplied with the base deficit, if it is a cattle but if it is a goat kindly use 0.6 instead of 0.3.

Now we have seen 2 examples, wherein each

Case A is 100 kg cattle which required 300 millimoles so we gave 337 ml of 7.5% solution.

Case B: we had 10 kg of goat, we required 60 millimoles and we were given 67.4 ml of 7.5% solution.

Both required treatment with sodium bicarbonate. Case A did not die, but Case B died, as soon as we gave IV bolus of sodium bicarbonate. Why? The answer to this puzzle of the question is take-home points: never exceed 2 millimoles per kg per minute(2 mmol/kg/min).

If you are administering sodium bicarbonate never exceed 2 millimoles per kg per minute. So if you are substituting with Case A and Case B:

Case A is 100 kg, 2 millimoles are 200 millimoles per minute(200/minute).

Case B, it is 2 millimole multiplied with 20 kg, it is 40 mmol, approximately 40 ml.

So this 40 ml, we are usually taking in a syringe and then doing is an IV bolus, which exceeded the 2 millimoles per kg per minute, so it has caused the death. So take-home lesson is never to administer sodium bicarbonate IV bolus if the body weight is less than 50 kg. So what to do? if it is 50 kg and less mix the sodium bicarbonate in saline or dextrose normal saline and then give IV so that the rate of administration of sodium bicarbonate will not exceed 2 millimoles per kg per minute. so thereby saving the animal. Now we also have 8.4% of sodium bicarbonate in the field, in such cases never administering faster than 1 ml per kg body weight per minute, so the

same like 7.5%, so your caution should be exercised it should not be more than one number per kg per minute.

Now in this lesson, we have seen about How to calculate the bicarbonate requirement in the case of cattle and calf.

Remember for cattle we have used 0.3 as the standard factor, for small ruminants you use 0.6. Another important thumb rule is never to administer more than 2 millimoles per kg per minute as far as sodium bicarbonate is concerned, which is very crucial to save a life.

In the next lesson, we are going to see about the Importance of Heat stress and how to overcome it.

Thank you!

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PDF: Acid-base Imbalances and Rehydration Management-Part II

5 Heat Stress

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://opentextbooks.colvee.org/fluidtherapycattlesmallruminants/?p=54#oembed-1

Transcript

Welcome! back for the Fluid therapy in cattle.

In the last class, we have seen about the calculation of the bicarbonate requirement in cattle as well as in calves and how careful we have to administer the sodium bicarbonate to the animals otherwise it will go for mortality in the case of animals.

In this class, we are going to see about Heat stress, which is an important thing because it reduces the milk yield and sometimes the death of the animal thereby reducing the economy of the farmer.

Now let us see what is Heat stress: Heat stress is a point where the animal is not able to dissipate the heat and maintain the body temperature. So what are the risk factors for Heat stress:

- one is an abnormal or sudden increase in the environmental temperature,
- the continued high temperature on a particular day,
- high temperature combined with high humidity on a particular day with low wind speed, and
- the wide difference between the day temperature as well as night temperature.

These all are important risk factors for the animal, ultimately leading to Heat stress. The situation like air temperature of 940 F, Humidity of 90%, and animal not provided with effective shade, so what happened the radiation from the sun and there is reduced conduction and convention from the animal, because of the increased environmental temperature the animal is not able to dissipate enough heat, there is reduced evaporation from the animal, all these add to the heat production, which exceeds the net heat loss from the animal, ultimately ending in Heat stress of the animal.

Now, this shows the Temperature Humidity Index(THI);

Temperature Humidity Index(THI) THI = 0.72(Cdb + Cwb) + 40.6

Where Cdb = dry bulb temperature (OC)

Cwb = wet bulb temperature (OC)

There is a formula that gives us THI is equal to 0.72 dry bulb thermometer and wet bulb thermometer. So here the thumb rule is when the THI exceeds 72, the animal is under stress. (showing the chart in the slide)That is beautifully explained by this slide, which shows the chart between the Temperature as well as the Relative Humidity and what is all there in the yellow, which shows the animal is in stress. what is in red color, that shows the animal is already in a heavily stressed condition and going to die.

Now we will see what are the direct effect of Heat stress: The direct effect is:

1. The feeds intake is reduced,
2. there are changes in the behavior and it will become restless, lies down,
3. then there is a metabolic change production loss, and
4. the immunity is going to get reduced and succumb to infection.

Buffalos are more sensitive to Heat stress and what happens is milk yield, growth, fertility, and conception are all going to be reduced because of heat stress, moreover, buffalo is in silent heat, and

because of the stress by heat, the silent heat will not be exhibited there will be a prolongation of the inter estral period and that goes for the affection of the economy. This shows the indirect effects of the heat stress on the animal:

- increases severity and the distribution of livestock diseases and parasites,
- there is a spread of disease and the parasites into the new regions, and
- there is an increase in the incidence of the diseases

these are the indirect effect.

(showing pictures in the slide) Now, these photos and this slide indicates the various clinical signs noticed in the heat stress namely :

- the reduced feed intake,
- heavy salivation,
- reduced milk yield,
- open mouth breathing, and
- sometimes goes for recumbency.

you can see the animal with

1. protruded tongue,
2. rapid shallow respiration
3. Depression
4. sometimes goes for comatose and
5. imminent death

Heat stress increases

- the metabolic state
- thereby increasing the oxygen demand
- there is hypoxemia and muscular weakness in addition because of lactic accumulation and the animal becomes weak goes for

recumbency
- there is a deterioration of cellular integrity
- going for Disseminated Intravascular Coagulation(DIC)
- GI integrity is lost so endotoxin is absorbed and sometimes there will be bleeding and
- cerebral and cardiac dysfunctions are noticed

Now we will see what are the strategies we can adopt for managing heat stress: very commonly we use:

1. **NSAID**– the non-steroidal anti-inflammatory drug we commonly use for reducing this hypothermia but none of the NSAID works effectively for the management of heat stress.
2. So what is effective? effective is **Fluid therapy**; as we discussed in the previous lessons calculate the fluid therapy requirement and then pump IV, thereby we can reduce the temperatures.
3. what else we can do? we can do **Anema**, do cool water enema two or three times, till the temperature comes down.
4. you can also go for gastric **Lavage**, again it is a cool gastric lavage, put into the room and lavage room and you do lavage repeatedly you do three or four times.
5. Then provide adequate **drinking water** to the cattle that also reduces.

But the best method is an effective physical method of cooling; by evaporation from the wet skin. (showing video in the slide) this shows how the pouring of water or rinsing the animal with water effectively cools down the temperature.

We can also do environmental modifications to reduce the heat stress, what are those :

1. we can provide **shades**, shade can be provided by means of providing trees. one such tree is in a Neem tree which provides adequate shade,
2. you can also increase the **ventilation**, how to increase the

ventilation; you can provide a fan, you can give sprinklers, you can give mist, and you can spray water onto the animal.

3. it is necessary to increase the evaporative cooling by **wetting** the skin, so you can pour the water onto the animal
4. you can increase misting by spraying the mist onto the animal and also to the environment.
5. kindly increase the **wind speed** and more than 5 km/hr,
6. provide a fan or
7. you can use a sprinklers

So these are the strategies by which you can environmentally modify the heat stress. So here we are able to see a modification in the protein, fiber, supplementation of potassium, supplementation of sodium, and you can also give antioxidants; like vitamin E and Selenium to the diet. You can give adequate salts, yeasts to the animal and these various strategies effectively control the heat stress.

In this lesson we have seen Heat stress, we have seen what are the various factors that contributed to heat stress, and we have followed the strategies and methods we can control heat stress.

The take-home lesson never uses the non-steroidal anti-inflammatory drug for the management of hypothermia.

Please understand physical methods of cooling are the best method, please use cool water anema and gastric lavage.

Thank you! very much

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PDF: Heat Stress

PART II

WEEK 2: ANEMIA AND BLOOD TRANSFUSION IN RUMINANTS

1 Anemia in Ruminants

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Transcript

Introduction

This lesson is about anemia and blood transfusion for ruminant practice. We have about five modules one on anemia evaluating the anemia in case of ruminants, then there is another module about basics of transfusion practice, then there is one module on bovine blood collection and, the other module on how to do transducer for bovines, the last module will be about small ruminant blood transition. In each module there are some of the subdivisions like how to evaluate for anemia, what sort of clinical signs you can see it in anemia, and what laboratory tests you need to do for, how to evaluate an anemic patient.

The second one about the transfusion basics – you need to know what is the importance of PCV for in the donation blood collection practices as well as transition practices, and how to select a good cow for donation and which are the need equal for blood transfusion and about the blood groups which we need to know both for bovines and small ruminants, and the use of anticoagulants, which is the good anticoagulant which we need to know use. And how much we need to use and, next we see about how much blood can be collected from a donor and how much can be transfused and the

rate of administration of the blood for the of both cattle and small ruminants and how to monitor for transfusion reactions and what sort of transfusion reactions commonly occurs. And if there is a transfusion reaction occurs what drugs we need to use to manage transfusion reactions. And at the end we will also be seeing about the small ruminants practice and the recent developments in the field of veterinary transfusion medicine

Thank You

Greetings vets

This class will be about Anemia in Ruminants. But, we know that animea is one of the very commonly occurring diseases in livestock practice either bovine or small ruminant that is the most important thing. Now what is basically anemia? Anemia is the reduction in the blood oxygen levels how we discussed, there is a reduction in the haemoglobin level or RBC count and both of which reduces the PCV. So, the total oxygen carrying capacity gets reduced. So that is how anemia ensues. Now what are the mechanism by which anemia comes? It could be either hemorrage, or due to hemolysis, or even due to the decreased production of the RBC itself. For example, in bullets some of the adult bullets, senior bullets, there may be possibility for some nasal tumors usually the ethmoid tumors that will be profusely bleeding so, that causes hemorrage in cattle cows which undergo prolapse or dystopia there will be bleeding at that time so that causes haemorrhage. So, what happens there is a presence of hypoxemia in the renal erythropoietin producing cells basically in the kidney.

Now this erythropoietin induces proliferation of the differentiation of erythriod that brings in red blood cells and normally takes care of the blood products. Now this is how they have to regenerate whenever there is acute blood loss. Now when there is anemia or bone marrow diseases this may not happen always. Now there are many many causes for anemia. The causes in ruminants are almost like same across cattle, sheep and goat many of the causes.

Now ectoparasites are the most important one like linognathus

similarly, gi parasites. Gi parasites usually the Haemonchus, then Fasciola, Paramphistomum these are the commonest ones. Then there is Abomasal ulceration, whenever there is a bleeding ulcers that also causes anemia. Hemorrhagic bowel syndrome there is one of the commonest diseases in case of crossbred cattle caused by clostridium pathogen species and that produces hemorrhage in the bowel and that's why it's called as hemorrhagic bowel syndrome. Similarly, bleeding in other sides or other systems like respiratory system or Gi tract, then presence of some of the injuries or tumors, then presence of hemostatic disorders similarly some poisonings like carbon tetracyclide, tetrachloride all causes anemia.

Now coming to the infectious agent many times blood parasites like Babesia, Theileria and Anaplasma, Trypanosoma and sometimes some of the Mycoplasma infections and Stephanofilarial or yellow lamb diseases, Bacillary hemoglobinuria, these are the commonest one. Most importantly in buoyant practice especially those in urban areas Leptospira is also a commonest cause. So under the toxic many many plants species like Allium or Brassica sometimes even long-acting tetracyclines can also cause some sort of impact on the blood production then trace minerals like copper, zinc arsenic these are also the important culprits for the causes of the anemia.

Deficiency mainly copper, iron, selenium, phosphorous all these things are a possible causes for anemia. Most importantly immune mediated some of the times immune mediated diseases are also causing the issues. Many times anemia can be caused by decreased production of the RBCs. One of the major causes nutrient deficiency for example micro minerals, rice minerals, cobalt and iron. Then there is presence of chronic diseases like lumpy skin disease, Mycoplasma paratuberculosis, paratuberculosis, chronic toxicities and Blue tongue. All these things are causing the decreased production of rbcs due to many factors. Sometimes bone marrow dysfunction and deficiencies that also causes the anemia.

Now what are the Basic Clinical manifestations – All mucous membrane then there is exercise intolerance, weakness, Tachypnea, Dyspnea. Now when you auscultate the heart there is increase in

the heart sound, then increase in the quality of the sounds, what sometimes they call it as anemic thrills. The severity of clinical signs are related to the amount of blood loss. How do you diagnose? Always diagnosis for chronic diseases is clinical science, laboratory examination, sometimes even bone marrow assessment. In some of the cases physical examination helps, but all these cases laboratory examination especially simply at least haemoglobin, PCB or BCR are essential. And Hemolysis occurs and that produces Jaundice, Splenomegaly, these are the easily palpated I mean palpated or using the physical exam tools you can find out.

Now one of the important thing is using some of the diagnostic aids like FAMACHA chart. This FAMACHA chart uses predetermined colour code that corresponds with the level of the haemoglobin used throughout the world and using the different colours the mucous membrane colour is correlated and the level of anemia is determined. Then the laboratory evaluation like your regular blood smear study, then the complete blood count, agglutination testing, these are the essential ones to diagnose anemia. Then most essentially the Fecal examination.

Then Urinalysis the commercially available urine strips like this that will help us to diagnose hypoproteinemia whenever there is hypoproteinemia anemia will be there. Similarly, when there is Hemolysis there is a difference in the pigmentation that can be identified.

Most importantly whenever there is a death of the animal when you cut open the bone marrow like this now this is the healthy bone marrow. Now the one with yellow gelatinous material like this this indicates the animal underwent a chronic disease and the bone marrow has been totally replaced by some sort of gelatinous fatty substance and there is nothing called marrow there to produce blood cells.

Now sometimes we may require additional Serological testing, molecular testing for diagnosing for either infectious diseases or some sort of antigen screening or antibody screening. Whenever you suspect poisoning, you need to identify with the chemical

assessment. So, with this the evaluation of anime is over. You have additional reading materials for the evaluation of anemia and you can go through these materials and whenever there is any doubt you can feel free to contact us.

Thank You

Download

PDF: Anemia in Ruminants

2 The Practice of Transfusion Medicine in Bovines

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Transcript

Greetings

My dear veterinarians, in the last class we saw about how to evaluate Anemia. Now once anemia is confirmed then our the next point is we need to go for a treatment options in the therapeutic drugs. Sometimes if it is a mild anemia your drug therapy will help. Now if it is a severe one you need to go for Transfusion.

How to do transfusion in the large animal practice? So that is what this class will be describing about. Now basically, why we need transfusions? Whenever there is acute disease acute hemolysis, acute hemorrhage there will be volumes of volumes of blood that will be lost. So, in such cases we need to have transfusion and whenever there is a chronic anemia then there is a best way is to go for a blood transfusion. Animals with hemostatic disorders which may not often get responded to regular drug therapy so, they require blood transfusion and whenever there is a critical care acute care is needed many of the patients will be requiring blood transfusion.

Now what are the indications for the acute hemorrhage because

acute hemorrhage is the one wherein, we need to go for a blood transfusion. Do acute hemorrhage could be caused by the rupture of the vessels during caesarian section, whenever there is any complications or umbilical muscle damage in case of calfs, some sort of trauma, or accident and then some surgical procedures where unexpectedly some bleeding happens or the vessel get cut we are unable to control. So, these are the ones that causes acute hemorrhages. When the ulcer in the abomination is severe enough that may also cause acute hemorrhages. Similarly, Tick born protozoa diseases. Sometimes massive anemia can come even with the Tick born diseases. Then whatever pathologies that causes acute destruction of the erythrocytes all of these are the causes for the acute hemorrhage.

Similarly, chronic hemorrhage basically Parasites are the one that causes the chronic hemorrhage the best example is the Haemonchus then Strongyles species that causes a typical white mucous membrane, there is not even a small sign of pink. Similarly, hemolysis this is caused by blood parasites – Theileria, plasma everything. The other important thing especially in case of buffalos Post-Parturian Hemoglobin- uria. This also produces the anemia. Then Bacillary Hemoglobinuria.

Now all these cases in bovine practice the most commonest thing is whole blood transition. In small level practice we go with blood component therapy like a separate RBC, separate platelet, separate plasma this can be used. For largen your practice because high volume of blood is needed many of the time it is the whole blood transfusion that is happening. So, this is the blood bags available for large animals. In some of the countries larger blood bags are also marketed in India too we have large animal thing, okay. Now the advantage is that it gives some amount of relief and oxygen carrying capacity supports the oxygen carrying capacity. Sometimes, if we need only RBC we can do that. So, when to use RBC transfusion? The decision to transfuse RBCs is always determined by the clinical science and not by any pre[1]selected or predetermined PCV levels. The acute anemia cases will show weakness, tachycardia, tachypnea

even at a higher PCV than animals with the chronic anemia. So, the amount of RBCs is required to alleviate clinical sign will generally increase the PCV above 20 percent.

Sometimes we can even go with the platelet and plasma transfusion. Platelet numbers will rise rapidly after hemorrage so many of the times platelet may not totally be required because they somehow manage and increase their count. Plasma that helps to equilibrate the interstitial space unless there is a massive hemorrhage plasma is not generally required and where the plasma is most useful those with coagulation disorders that will be benefited by your plasma therapy especially those with von Willebrand factor deficiency whereas the plasma therapy will be most useful. The other component therapies use of platelets or platelet-rich plasma. In case of thrombocytopenia this will be of use, in case if it is immune mediated you cannot expect much relief from that.

Now, what is the basic Blood Volume? Domestic animals will have about seven to nine percent of their body weight i.e., the Blood Volume. So by determining the recipient's Blood Volume and knowing the animals PCV the requested replacement of RBC can be calculated and always never collect more than 20 percent of a donor's blood so, that is the most important thing. Whenever you are collecting never collect more than 20 percent of the donors Blood Volume. Some of the facts which you need to keep in mind for your Clinical practice is the normal blood volume is eight percent of the body weight and, the adult bovine PCV is like 24 to 43 percent and Haemorrhagic shock may happen when there is 30 to 40 percent of the total blood volume. Blood Volume is lost many times a cow with a PCV of 10 percent would have lost almost two thirds of the circulating red cells. still the animal may be standing. So, these are sometimes misguiding us. Now if we are using one liter of blood this may raise PCV sometimes by 75 percent, it depends on the case varies from case to case. The clinical difference between a cow with a PCV of 8 percent and another cow with 14 percent is always highly remarkable and the red cell turnover happens probably once in two

to three days. But what is the most important thing is whenever you are using a repeated transfusion, second transfusion that may end up in the transfusion reactions. And the most important thing is red cell survival happens only for few hours so, within which it has to improve.

So, with this in this presentation we saw about basics of the Blood Transfusion for a large annual practice. So, what are the basic Clinical Facts you need to know before moving into the actual transfusion practice and there are reading materials available you can refer them if you have any query kindly contact us.

Thank You

Download

PDF: The Practice of Transfusion Medicine in Bovines

3 Bovine Blood Groups

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Transcript

Greetings dear veterinarians

In the last presentation we saw the basics of large animal transfusion with those clinical facts now we move into the next stage where we need to know about the bovine blood groups and how to collect blood from the donor cause.

Now blood types are basically representing certain specific antigens. These are present on the surface of the erythrocytes. In human we have the ABO system but the same is not used in animals, in bovines we have a different system a species specific antigen presentation is there. The number of blood groups in cattle are bovines it varies between species many times in the bovines or cattle it exceeds 80 whereas in case of Camelidae it's only one in the new world Camelidae and then there are certain kind of antibodies to blood group antigens we need to know about. So there could be a naturally occurring antibody or the antibodies that may get acquired after an exposure to the particular blood antigen. Now what happens the naturally occurring antibodies occur in the most species and they vary in their pathological significance, some even may not even produce a transfusion reaction.

The acquired antibodies what happens they get developed or

acquired after exposure to an incompatible blood group or any blood product that contains a particular antigen. Now when such antigen exposure is happen these acquired antibodies are produced and this causes some sort of reactions sometimes it may even induce a hemolytic reaction or agglutination or hemolysis of the cells.

So to avoid these sort of transfusion reactions we need to go for the blood typing. Bovine blood typing is not available in common conditions or in the field scenario which is available only in the referral centers, IR institutions and not much companies are available who manufacture the kits only very few diagnostic labs are available. Now compatibility testing, right so we use cross matching to test the compatibility generally we send the sample to the laboratory and we can do that or if you have a point of care lab at your own practice we can do that. Many times the administration of typed negative blood will not prevent a transfusion reaction so don't expect that even after typing the reaction may not happen it can happen, varied antigens are there that may cause such a consequence.

Now from the clinician's standpoint these antigens are sometimes the practices should be able to familiarize with that. But due to the lack of commercially available kits practices are finding it difficult to know the antigen systems in the in their respective practice area. Now many times these antigens gets coupled with platelets leukocytes and plasma proteins and induce immune mediated reactions. So we need to be careful whenever incompatible blood is used and erythrocyte antigens themselves can produce antibodies in the recipient. So many many disadvantages occurs whenever there is a incompatible antigen usage.

Now these are the common blood groups in the cattle so they are named with different alphabets like A, B, C then F, J, L then there is M, O, R, S T and Z these are the ones until now has been identified and in some of the referral labs the kits are available for typing these 11 groups. Now out of these 11 groups, group B and J are the most important one and clinically relevant. But what happens is even in

within the B itself there are more than 60 antigens. So, we can't perfectly do and identify a blood that is very safe for the recipient because 60 antigens can be varying. We can't basically do any very close matching for such cases.

And J antigen now this is a lipid found in the body fluids and gets absorbed into the erythrocytes. So basically it is not a true antigen but still it can produce some interferences. Newborn calves may not have this antigen but it gets acquired in the first six months of life. Even sometimes the J negative animals can develop antibodies. so, we need to be very very careful when we are using uh such cases.

And in case of younger animals Neonatal Isoerythrolysis (NI) is a possibility. It's not a naturally occurring phenomena but Bouts of neonatal isolation can happen after a blood dried vaccine is being used for example some of the vaccines they use for anaplasmosis, babesiosis. These can bring in Isoerythrolysis and most common antigens that cattle get sensitized is the A and F systems.

Now in the next part we will see how to do donor blood collection from the bovines. For small volume blood collection we can use donors at the hospital setup. In some of the countries where the big slaughter houses are there people go and collect blood from the slaughter houses also. But in our small practices we use donor cows, a healthy donor cow about four and five year old and possibly it should be a dry animal or at least one month after calving, these are the preferred animal. A larger sized cows with a body condition score of 5 that will be good enough and most importantly we need to contain their excitement we provide allowing care for them so that it cooperates for your collection. Basically, we don't need to have any specific treatment for the blood donors. After donation you can just send them home and always calculate the PCV for both donor and recipient. For donors sometimes PCV may go less than 15 but animal may look like not even a sick, so you need to take into account.

Now we look at the use of Anticoagulants for the Bovine blood collection. For bovine donations either you can use a sodium citrate normally 12 grams of sodium citrate dissolved in 300 ml of saline

will be enough to collect 5 liters of blood, and basically about 9 liters of blood will be more than sufficient to keep a cow alive and sometimes even as low as 3 liters can be enough but you need to aim for a collection of 5 liters so that this will improve the PCVand the relieve the Anemia significantly. You can even store the blood and you can use it but whenever you are using with stored blood preferably do it within the 24 hours, and for this reason we always collect fresh blood whenever there is a immediate requirement, we don't collect and store them. A cow with PCV of 8 may even stand and walk around, so we think it is a good but suddenly it may deteriorate. So pcv alone will not be good guidance you need to decide based on each case.

Now collecting at the clinical setup now how to collect for example if it is in your farm you can use the human blood bag like this, and put it put the you can either use a jugular catheter, or the collection set available with the standard blood bags, put the needle into the jugular vein and start collecting and the bag has to be keep agitated, if it is in the hospital you can do it with a blood collection monitor that keep on agitating the blood bag. The most important thing is maintain the donor in a quite calm situation if required you can even use mild sedation and do a very perfect aseptic preparation of the jugular vein and then put the needle into the jugular vein and then collect. Probably it will take less than few minutes for getting the end tablet collected, and what is the needle size or catheter size 14 gauge will be useful even with the 16 gauge needle you can do that and normally 400 ml of 3.8 percent sodium citrate can be used, add it to a four litre collection bag if it is a bigger volume collection a four litre collection bag or even a bucket where you can add this uh three point eight percent sodium citrate and you can collect the blood and start transfusing to the max and use that. The other formula is to use about 38 gram of sodium state into one litre of Hartmanns solution and then that will support for two cows.

Now what are the basic limitations is that the limitation here is no more than 20 % of the blood should be collected from the donor

so that is the most important point you need to keep in mind never collect more than twenty percent of the bonus blood volume. And all the collection storage as well as transfusion should be done in a very very aseptic manner because you are going and dealing with the jugular vein, it's a central vein we can't take risk even a small change can bring in infection or massive bleedings. So, we need to be very cautious in that, so now we need to know how to collect from the donors and there are additional reading materials available about the care of the donor cows and what are the things to be done for a donor, if you have any doubt kindly let us know will be there to clarify.

Thank you

Download

PDF: Bovine Blood Groups and Blood Collection from Donors

4 Case selection for Blood Transfusion in Cattle

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Transcript

Greetings veteririans,

In the last presentation we saw about how to collect the donor blood from the donor cows. Now in this we will go and see how to use the collected blood, and how to transfuse them, and out what are the cases, how to be select such cases, right.

The most important thing is the practitioner should assess the essentiality for the transfusions, okay. Since, transfusion is always a little expensive offer, a time consuming one, lot of stressful thing to the animals, so we need to be very very cautious and to decide that. Acute loss of 20 to 25 percent of the blood volume will result in market clinical science like anemia and then maniacal behavior sometimes tachycardia, so identify for such science and decide these things. Then along with your clinical guidance use the PCV as the guidance and acutely if the animal develops a pc of 15 person or less then that is the case you need to go for the transfusion and some of the cases with the chronic PCV or very low PCV with 7 to 12 percent they may even tolerate that low levels without transfusion, but if it gets reduced probably we need to go for a transfusion so

PCV has to be taken into account but don't decide purely based on the PCV because PCV sometimes that's what I say even with the seven percent PCV the animal may not exhibit such alarming signs so always take into clinical presentation plus PCV for transfusion.

Now we need to know certain basics about the transfusion practices. 75% of the transfused RBCs will get destroyed within 48 hours of the transfusion, so that is the most important thing we need to keep in mind. Whatever we do this RBC will get destroyed within 48 hours almost 70 percent gets destroyed in this point of time. And, what is the transfusion rate we use to administer the blood? We need to use a rate of 10 to 20 ml per kg that will appreciably increase the PCV levels so 10 to 20 ml per kg is the rate with which we need to use. Basically, a non[1]pregnant donors can contribute into 10 to 15 liters of body weight, okay and you can even collect it at two to four weekly intervals, so that is the benefit for the case. Now this is a field collection where just using restraining the animal and with a few people and start using the human blood bag and collect and somebody has to agitate the bag so that it mix up easily. Now once collected you can take them to the laboratory and check at the use the blood weighing monitoring monitor weigh the blood and you can calculate the amount of blood collected and then you can use the transfusion set.

This is the most important thing you can't use your regular intravenous fluid therapy lines these transfusion sets are special filters, so these transfusions sets should be purchased and used, not the regular IV sets, that is the most important thing and again always ensure which sort of anticoagulant is present in the particular bag. Sodium citrate that is very effective but the other one is ACD- acid citrate dextrose. When we use acid citrate dextrose commonly when you prefer to store the blood we use the ACD if you are going for a direct transfusion you can use the sodium citrate, and once you are deciding for the storage don't store for more than two weeks, okay, try to use them within that. And use of Heparin not normally a good one. So heparin is basically an unsuitable anticoagulant.

Now the most important point is doing a cross matching you need to do major versus minor cross matching, Okay. In the field conditions it is always easy how we do? Cross matching is just inject 200 ml of the donor blood into the adult, then wait for about 10 minutes, just infuse 200 ml a small volume, wait for 10 minutes, if there is no transfusion reaction occurs then slowly start transfusing the remaining blood, if there is any transfusion reaction occur within this 10 minutes or the first 15 minutes then don't use that blood. So adverse reactions normally, they are seen in the very young animal, and the pregnant animal, and the adult you may not able to see, and don't massively transfuse all the volume in a rapid rate, always maintain the rate very very slow.

Which are the needy cases where you need to do? Mainly the Tick borne (TBD) diseases especially a condition called taba, theileria associated bovine anemia where, almost invariably the drug therapy alone is not sufficient, we need to keep transfusing lot of blood for making the animal to survive. Those cases which we do not do blood transfusion many of them succumbs so tawba or theileria associated bone anemia is a typical case where blood blood is blood has to be collected and transfuse. Blood transfusion helps is the clients are very happy when we are making some effort and we do blood transfusion, because they realize the importance how much the doctor takes to save these cattle. So, it is a kind of Public Relationship Building for the veterinary practices and the farmer feels how much effort he and his team has put in. So always make all the best efforts to do transfusions of the field level and the Recipient Cows.

Now which start of the cows we need to select? Any cow which is less than 10 percent PCV. So, these are the ones they require transfusion and those cows between 10 and 15 PCV they also get benefited. So, they will also get benefited from the transfusion. And again please keep it in mind the cows don't actually look sick until the PCV falls below 12 percent so PCV may get misguide us. So always ensure clinical science plus PCV to do the transfusion decision. Always prepare to watch for transfusion reactions in any

case transfusion reaction can happen, if you are doing the transfusion, always keep ready some of the Antihistamine. then adrenaline at least five to 10 ml of adrenaline keep it ready so that if any reaction comes you can use.

Now the veterinarians need to know basically the transfusion reactions in the bovines. It is not a similar one like what you see in the human medicine or small animal practice. Many times you may not even realize these things the reason is we are almost 11 to 13 blood groups, and Bovine red cells do not agglutinate that much easily, and we need to keep in mind the isohaemolysins. Sometimes preformed isohaemolysins may present in a very low low quantity so in such cases repeated transfusions within seven days they carry the risk for transfusion reactions. So, though it is not a very common one transfusion reactions can occur especially if we are going for the repeated transfusions.

Now what sort of transfusion reactions you can see whenever there is things but that's what I said, usually the reactions are rare and very mild. The commonly observed transfusion reactions are like hiccup, the tachycardia, Tachypnea, sweating. Sometimes tremors, tremors is the most easiest one we can identify, saliva and cough all of a sudden there will be increase in salivation. Lacrimation mostly if it is a incompatible blood lacrimation will happen. Then after transfusion there is every possibility that hematuria and hemoglobinuria can occur. If it is very severe the animal may even collapse. So these are the transfusion reactions you need to watch for in the cattle.

The rate with which you need to administer is 10 ml/ kg/hour. So, it's a very slow transfusion 10 ml per kg per hour is the transfusion faster rates. Sometimes you can use whenever there is a per acute hemorrhage. You want to rapidly replace blood to prevent collapse and plasma this can be used when there is failure of absorption of the antibodies, especially with the protein losing entropy or protein losing nephropathy you can consider use of plasma. And whenever the protein levels are getting reduced less than three grams and if

the admin goes less than 1.5 gram these are the cases where you can use plasma.

Now when you encounter any transfusion reaction how would you manage such transfusion reaction. So usually epinephrine is the one we use, intravenously epinephrine, one in dilution, one in thousand dilution for example about point two to point five ml intravenous are at the rate of four to five ml intramuscularly which you can use if there is a transducer reactions. Either, you can go with low dose iv or about 5ml intramuscularly. Some of the field practice people start using antipyretics even before transfusion is being done, but that is a double edged weapon we may not even able to identify if there is a temperature rise after transfusion. So, it will be better if you go with after the transfusion and to as a time there is a reactions, okay, and blood the other easiest thing is blood or plasma if you give it rapidly it may end up in cardiovascular overload. So, a rapid transfusion result in overload and sometimes acute collapse can also happen. Acute heart failure or hypertension collapse will be happened. And the other drug is Furosemide, why we are using furosemide is when you are giving rapidly there is buildup of overload, to decrease this overload you can use furosemide.

And for transfusion of plasma you can store it using your household refrigerator or even the freezer. The freezer is the best one because at the household we can't store for longer years, but if you have a professional grade freezer you can store plasma at minus 15 to 20 for almost like a year, because plasma is rich in coagulation factors. If you start using early fresh it will be good, but after two to four months the coagulation factors will start coming down as they are getting destroyed. If you want to really maintain the viability of these coagulation factor then the best alternative is to use minus 80 degree freezes to store your buoyant plasma, almost up to an year or less than 12 months we can use it.

The Administering Plasma okay, again the same kind of special built-in filter should be used at the transfusion set not a regular iv set. And whenever you thawed the most important thing all the

frozen plasma has to be thawed before use. Sometimes there could be precipitations and these microaggregates may result in a fatal reaction. So when you thaw the plasma always check it, it is free from the microaggregates. If you just take it in a, and shake it you can easily visualize the microaggregates.

So, in this presentation we saw how to transfuse the collected donor blood as well as the stored blood products like plasma to the cattle. We have a reference material and reading materials given for your further reading. You can go through them if you have any queries please let us know we will be clarifying that.

Thank you

Download

PDF: Case selection for Blood Transfusion in Cattle

5 Blood Transfusion in Small Ruminants

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Transcript

Dear vets

In the previous presentations we discussed and dealt about the blood transfusion practices in bovines. Another important ruminant species is the small ruminants and anemia is again a common disease in the small Romanians. So, whether in the field level we can do blood transfusion for the small ruminants. Currently, not many places they do but there are field practices doing small ruminant blood transfusions. So, in this presentation we see about how to go with the blood transfusion in the small ruminants.

When we talk about the small Ruminants the history itself is very important. The first transfusion happened with the lamb to the human. So, this is how first transfusion happened somewhere in the year 16th century about 1692.

Now what is the basic reason to go with transfusion in the small ruminants. The internal parasites are the most important reason for example Hemonchus is the most important one for which blood transfusion is being done. And any external parasites uh besides internal parasites can cause severe anemia that requires blood transfusion. Similar to bovines there are certain blood groups in the small ruminants. So about seven blood group systems are there

like A,B,C,D, M, R and X. Similar to cattle, the B system is highly polymorphic in the small Ruminants also. The R system is similar to theJ system in the cattle because it is soluble. Now one of the uniqueness about the M and L system is they are involved in the active red cell potassium transport and any polymorphism happening in the ML that produces sheep with varying erythrocyte potassium levels.

The other important thing we need to know about is the Neonatal isoerycthrolysis similar to bovines. It can happen in the lambs when you feed the lamb with bovine colostrum. In many practices, in many areas people used to feed bovine colostrum to small kid's lambs. So they developed a neonatal isoerycthrolysis because there is a presence of antibodies to sheep isoerycthrolysis which is present in the bovine colostrum and this causes the isoerythrolysis. So we need to be very careful with that.

Now we move on to the next one Blood Groups in case of Goats. These are almost like similar to sheep and there are special laboratories available which types and helps to form us to identify the blood groups but not commonly available. There are five major systems of blood groups as far as goats are concerned so A, B, C the M and J. So here also in goats J is a soluble antigen as in case of cattle.

This is how a field blood collection. You can use the human blood bags what you get it in the commercial blood bags. You can use your human blood bag put the jugular vein prepare the jugular vein aesthetically and similar to any small practice put the needle into the jugular vein carefully and start the blood flow started collecting via into the blood bag and again the bag has to be keep on tilted so that uniform mixing up of the anticoagulants happens.

Now what are the basic parameters where you need to keep in mind. 10 to 20 ml per kg you can collect from a healthy donor. So you can safely start with 10 ml but if you need a higher blood you can go with the 20 ml and the collection at the rate of 10 to 15 ml equates to roughly about 500 to 700 ml for a goat weighing 50 kg. Many of the times it is comparatively lesser we don't find 50 kg goat

we'll be finding less than 20 kg or 30 kg only and, all the human blood bags what you can use are for the human medicine practice they can be very well serving the purpose for our goat. There are some field practice they can collect the blood from the sterile IV bags which are added with the sodium citrate. For example 100 ml of four percent sodium citrate this can be added to a one liter bag. You yourself can make a bag, add about 100 ml of four percent sodium citrate to one liter bag and use this bag for collecting the blood. Now the most important point always is agitate I mean keep on turning the bag to prevent the clotting that happens inside.

Now once you collect it you can transfuse similar to your IV injection giving a jugular injection you just put a jugular catheter or the needle and start transducing the uh collected blood into that.

Now what are the basic transfusion tips you need to keep in mind when you are dealing with the small ruminants. Always go with this slow drip infusion right very very slow drip for the first 15 Minutes, because we need to know watch for transfusion reactions, if there is no transfusion reactions happening then we can go with the regular rate. Normally, reactions are rare in the small ruminants. We don't see much often especially with the first transfusion the reactions are comparatively less, but the reactions will become very frequent if we are going for a subsequent transfusion or repeated transfusions. Similar to bovina practice you always keep it in mind Epinephrine. So Epinephrine will help to manage transfusion reaction. If there is any severe transfusion reaction occur you need to stop the blood that blood cannot be used possibly, you need to collect it from a different donor and again test it and use it. Recently there are papers emerging where some trial studies where they used bovine whole blood for Goat Anemia. So, these are all research studies probably in the future we may able to get the field level implications for such cross pieces transfusions. It may take another few years to get such results available for the field. And always keep it in mind transfusions are not total solutions it is only a temporary measure. You need to find out the cause for the anemia and treat that anemia and whenever you transfuse the entire

Donor erythrocytes get cleared within the eight days. And similar to bovine practice always can go for the cross matching and find out if it is unfit you don't use that and, monitor most importantly the heart rate, respiratory rate and the rectal temperature you need to monitor. Any changes in them then that may indicate the possibility for transfusing. So we need to monitor them every 30 minutes that will be good then hourly monitoring and even you can monitor up to 16 days after transfusion. Right. Sometimes, reactions can happen as a delayed reactions.

Now the commonest reaction here is tachycardia, tachypnea tremors fever and pruritus. So these are the commonly observed one sometimes that can be hematurian and hemoglobinuria. But almost 50 percent of the cases there will be hyperthermia. So that's why we advise you to frequently monitor the rectal temperature whenever you do a transfusion reaction. The other important thing whenever you are using a stored blood there are every chances it even can also cause reactions. Stored Blood for example you are collecting blood you can use the CPDA -1 solutions and store the blood for 35 days right, but sometimes stored blood may have some sort of metabolic changes within that the using the stored blood may also produce some reactions we need to be very careful with that.

So in this presentation we saw about how to do small ruminants blood transfusion. We have additional reading material given to you can refer very well and many of the large animal practice journals are also having materials around boving blood transfusions and small remaining transfusions you can very well learn from them if you have any queries kindly let us know

Thank you

Download

PDF: Blood Transfusion in Small Ruminants

PART III

WEEK 3: ABOMASAL ULCERS AND DISPLACEMENT

1 Introduction to Abomasal Ulcers and Displacement

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Transcript

Dear Vets,

Greetings to all!!

In this module, I am going to discuss Abomasal ulcers and displacement in cattle. The module has five lessons:

- In lesson one, we are going to discuss Abomasal ulcers up to Etiopathogenesis
- Lesson two includes clinical signs, diagnosis, and treatment of Abomasal ulcers
- lesson three covers left displacement Abomasum
- lesson four covers right displacement of Abomasum with valveless
- lesson five is going to cover Abomasal bloat and Abomasal impaction.

Dear vets, greetings to all.

Lesson 1: we are going to discuss the Abomasal ulcers;

Introduction

As we know the Abomasum is a true stomach of the ruminants,

so all the digestion process is going to happen and any dysfunction of the Abomasum is leading to metabolic alkalosis, hypochloremia, hypokalemia. Here the glandular stomach of the ruminants is more vulnerable to any stress, so this stress is a major factor for the development of the Abomasal ulcers, apart from stress, so many other causes also attribute to this.

So first we will see the **Anatomy** of the Abomasum. The abomasum is lying in the cranial right quadrant. In the non-pregnant animal, it is positioned below the rumen, almost ventral to the abdomen, predominantly on the right side. So, in the case of pregnant animals, it will be pushed a little bit forward, so the pregnancy is going to influence any displacement, so that is a measure because it is a movable organ it is going to have a displacement very easily.

Etiology: Let us discuss Abomasal ulcers; the Abomasal ulcers are nothing but like a monogastric animal, there is gastritis, here above an Abomasitis is going to have. So, what is the reason behind the Abomasal ulcers are primary causes. primary etiology or primary ulceration is caused by Abomasal hyperacidity. So, stagnation of the hyperacidity will lead to damage. Then mechanical abrasions due to any straw which is escaping through the reticulo omasal orifice. Bacterial infections like Clostridium perfringes type A and fungus-like Aspergillus fumigatus and some of the Mucor spp species also be involved with the Abomasal ulcers. Concurrent deficiency of any minerals like copper deficiency, transport stress, calving, lactation stress altogether is making up Abomasal ulcers. These are the primary causes. Secondary ulcers are mainly because of the some of the diseases, like bovine viral diarrhea(BVD), Rinderpest, bovine malignant catarrha (BMC), and some of the cases, there may be a chance of getting reflux of bile into the abomasum, that will also damage the abomasal mucosa and Lymphosarcoma and in case of Theileriosis, it's especially in case of blood protein disease, Theileriosis it is going to cause the punctuated ulcers in the abomasum, that is having a heavy bleeding animal is going to develop anemia.

Another more thing is non-steroidal anti-inflammatory drugs (NSAIDS) indiscriminate use of the non-steroidal anti-inflammatory drugs will lead to Abomasal ulcers.

Pathogenesis: The injury to the gastric mucosa will lead to diffusion of the hydrogen ion into the wall of the Abomasum, it is permitting the other pepsin to enter into the wall of the abomasum, which is causing the damage. So, ultimately there will be ulcers due to the hydrochloric acid. Now let us come to the Classification:

Classification of Abomasal ulcers; according to the severity we are going to classify type 1 to type 4.

- Type 1 it is non-perforating ulcers only the internal,
- Type 2 ulcers with severe blood loss,
- Type 3 perforating ulcers with local peritonitis,
- Type 4 perforating ulcer with diffuse peritonitis.

So let us discuss it one by one now.

Type 1 is a non-perforating ulcer, it is having an incomplete penetration, there is no involvement of the layer beyond the muscular layer, so there won't be any bleeding.

Type 2 is ulcers with severe blood loss because the penetration of the blood vessels in the Abomasal wall also are involved and there are intra-luminal hemorrhages, there is a lot of accumulation of fluids in the Abomasum, it is leading to Metabolic alkalosis, hypochloremia, hyperkalemia.

Type 3 is a perforating ulcer with localized peritonitis, the entire wall is having an opening and the Abomasal content is discharged into the peritoneal cavities leading to peritonitis. It is confined only to a particular area that is called localized peritonitis.

Type 4 diffuse peritonitis, perforating ulcer in the abomasum, discharging the content of the peritoneal cavity, leading to diffuse peritonitis. It is more severe, and it is leading to the death of the animal.

In this lesson 1, we have discussed

- the Introduction of the Abomasum
- the location of the Abomasum- Anatomy
- Abomasal ulcers regarding etiopathogenesis
- types of ulcers

That is lesson 2, we are going to discuss

- the clinical signs,
- diagnosis, and
- the treatment of the Abomasal ulcers.

Thank You!!!

Download

PDF: Introduction to Abomasal ulcers and displacement

2 Abomasal Ulcers – Clinical Signs, Diagnosis & Treatment

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Transcript

Dear vets,

Greetings to all!

Last class lesson, we have discussed Abomasal ulcers, Etiopathogenesis, and Types of ulcers. In this lesson 2, we are going to discuss the Clinical signs, Diagnosis, and Treatment of Abomasal ulcers.

The Clinical signs: mainly the Abomasal ulcers will have a Melena, a tarry colored feces. so automatically leading to anemia. The animal will have anemia means, automatically having a Tachycardia, Tachypnoea. The distinction of right lower corner of the abdomen also be there and abdominal pain because of ulcer abdominal pain will be there. The animal is going to develop Bruxism, which is grinding of teeth because of continuous irritation in the Abomasum, animal sips water, continuously sipping the water and it will be staying in the area of water turf and continuously sipping. It will have severe dehydration, metabolic alkalosis, hypochloremia, hypokalemia is going to happen in case of the Abomasal disorders, and in severe cases, there will be cold extremities and the

perforating ulcers in animals will lead to acute diffuse or acute localized peritonitis in animals.

(showing the chart in the slide) In this picture there are tarry-colored feces, it is indicated that Abomasal ulcer.

Abomasal ulcers Diagnosis: so you have to diagnose based on the history of any Melena or Bruxism. Clinical signs; also have to take into account. Clinical signs of sipping the water, grinding of teeth, pain in the abdomen, Ultrasound scan: so you can also scan the ultrasound of the abomasum by the ultrasound scanner. Sometimes if there is an Abomasitis or Abomasal ulcers, there may be thickening of the wall, which you cannot identify the light lower quadrant. Abdominocentesis; suppose type 4 or type 3 Abomasal ulcers will lead to either diffuse or localized peritonitis, so that can be identified by Abdominocentesis, if there is a copious fluid discharge in the Abdominocentesis, we can claim that it is a case of petronetics due to Abomasal. Clinical pathology: so, you have to assess the hematological values, there will be metabolic alkalosis, serum biochemistry, and hematological values. There will be metabolic alkalosis hypochloremia, hypokalemia, potassium will be low. and the Occult blood test is one of the important tests, you can identify the Abomasal ulcers. Suppose by the naked eye, we are not able to see the blood present in the dung, so it will be having some hidden factors like. So, if you are having traces of blood in the dung, you have to subject the dung to an Occult blood test. So Occult blood test is an ortho-tolidine tablet test (Hematest) or Guaiac paper test. This test will develop into a blue color, it will confirm that traces of blood in the dung and then those animals are affected with the Abomasal ulcer having Leukocytosis, neutrophilia, and elevated fibrinogen level. In the case of an Abomasal ulcer fibrinogen level will be elevated, and another more important recent test is Plasma Gastrin level will be elevated.

Treatment :

- Blood transfusion: In case of severe anemia, Pale mucous membrane, Tachycardia, Tachypnoea, if it is below 15% of PCV

or 5 mg per liter of hemoglobin, we need to go for blood transfusion as per the standard operating procedure from a healthy animal to the donor you have to give.

- Fluid therapy: massive fluid therapy, when you are having a fluid requirement you have to assess the anemic status of the animal. If anemia is there you have to judicially use the fluids, you have to select colloids in case of anemia. Hetastarch, Pentastarch, or Haemacce, are the colloids 5 to 6 ml per kg bodyweight you can give followed by crystalloids like regular selected DNS you can give.
- Coagulants; to arrest the bleeding you can also advise coagulants but it is having doubtful value. common coagulants used are Tranexamic acid and angiogram semicarbazone.
- The mixture of the Kaolin pectin; In The field condition, you can also use the Mixture of the Kaolin pectin mixtures, that 2-3 liters if you are administering, that will coat the ulcers and further damage will be prevented.
- Antibiotics: in case of perforating ulcers, antibiotics that are broad-spectrum antibiotics for 5-7 days you have to give.
- Antacids: definitely will be useful for us to treat the case perfectly, it includes. H2 blockers- Cimetidine, Ranitidine.
- The proton pump inhibitors like Pantoprazole, Omeprazole.
- Neutralizing agents- neutralizing secreted acids like Aluminum hydroxide, Magnesium hydroxide, and Magnesium oxide at 500 grams per animal, if you are giving for 2-3 days it will be helpful for coating over the ulcers. Further secretion of the acid will not damage the wall of the Abomasal.
- In the case of Theileriosis, especially theileriosis directly involved with the Abomasal ulcer, the animals should be treated with buparvaquone and oxytetracycline (OTC) and
- In any Tick infestation, that will be covered with Iverment.

In this class 2, we have discussed the Clinical signs, Diagnosis, and Treatment of Abomasal ulcers in cattle. Lesson 3 is going to cover the Left displacement of Abomasum in cattle.

Thank you!

Download

PDF: Abomasal Ulcers – Clinical Signs, Diagnosis & Treatment

3 Left Displacement of Abomasum

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Transcript

Dear vets,

Greetings to all!

Last class, we have discussed the Abomasal ulcers in cattle that too Clinical signs, Diagnosis, and Treatment.

In this class, we are going to discuss the Left displacement of Abomasum in cattle. So, usually, the Abomasum is a movable organ, it's associated with pregnancy and rumen fullness. whenever the rumen is full. it will be a little bit pushed forward and the gradual push is there that will push the Abomasum forward. So during the course of any pregnancy or rumen dysfunction, the Abomasum is displaced, so if it is displaced on the left side, it is a common one, when is associated with the calving, just a few weeks of calving, it is more commonly seen. So why it is happening? Because of gaseous distension and hypomotility, so when the concentrated feeding is more during the prepotent period that will make the Abomasum to have a gaseous distinction because of movement of more volatile flaccid acid there will be a mildly acidic, that will also lead to hypomotility.

There will be a chance of reduced actually forage feeding, which is also another more attributing factor for the Abomasal displacement.

Pathogenesis; the advanced stage of pregnancy, the abomasum is a little bit pushed forward, the rumen is lifted, and after calving the rumen is coming to the original position that time the The abomasum is having hypomotility and gaseous distension, the illest condition of the abomasum, the pyloric part is slowly going to the trapped underneath the rumen and sidewall of the left side, so that is leading to Left side displacement of Abomasum.

What are the **Clinical Signs**?

- The inappetence to anorexia,
- drop in milk yield,
- bruxism,
- the signs of Ketosis, the animal because of Abomasum is in what signs of ketosis will be there, so cow side test will be helpful for us to identify the ketosis also, that is a secondary one,
- slap sided left side abdomen,
- decreased ruminal movements, when you are doing the combined percussion and auscultation, the 8th to 12th intercostal space or 9th to 12th intercostal space on the left side there will be a ping sound, the oval area, you can see the oval area (showing picture in the slide) that will have a ping sound.

(showing picture in the slide)So you have to do the combined percussion, in this picture I am doing the combined percussion of the, you have to first demarcate the area of ping sound, that is a ping sound is nothing but dropping a coin into a metallic vessel or ringing sound, that the ping sound how it is happening it occurs in a hollow organ, it is filled with fluid and some air is also there under the pressure. The fluid medium and air medium is going to contact in the hollow organ, when you are percussing on the top the air movement will be vibrating and hitting on the fluid and returning back as an echo, which is leading to a ping sound.

So here the abomasum is trapped in between the rumen and

the left abdominal wall, that is why it is causing the pressure in the air present in the abomasum leading to a ping sound. (showing video in the slide) This video is showing the combined percussion, auscultation of the left displacement abomasum. So, you have to confirm whether the distended area and the ping sound area are due to our abomasum displacement or due to some other reason, sometimes it may be done due to rumen collapse or auscultation condition also.

So we have to take the needle, spinal needle, puncture the area where you are getting, this video telling that the abomasal ping sound area, we are injecting the needle and collecting the grayish fluid (water fluid), from the abomasum. It is suspected of the abomasum.

In the ping area, you have to aspirate, so you have to see the pH and protozoa. The normal pH of the abomasum is 2-4, but there won't be any protozoa. So, in this picture, you are able to see the pH of 2 and there won't be any protrusive mobility, so the technique is called as Liptak test. The Liptak test is confirming the displacement of the abomasum.

Ultrasonography examination also reveals the left side displacement of the abomasum, you can visualize the hyperechoic wall with the hyperechoic content of the abomasum on the left side. Then other diagnostic techniques are:

- if you are doing a Rectal examination, you can palpate the abomasum on the left side,
- clinical pathological conditions like Metabolic alkalosis, hypochloremia, and hypokalemia,
- sometimes it may also have an elevated beta hydroxy butyric acid and AST level as aspartate aminotransferase, then
- mild hypocalcemia also is there due to abomasum displacement.

Differential diagnosis; the abomasum displacement can be differentially diagnosed with the

- Simple indigestion,
- Astomia
- Primary Ketosis
- Traumatic reticulopetonitis,
- Vagus indigestion,
- Fat cow syndrome

Treatment:

- The abomasum displacement on the left side should be surgically corrected, first, it should be surgically corrected, or else
- you can go for roll and toggle pin method, suturing method keeping the animal on the dorsal recumbency, you can roll this side and that side, then positioning the abomasum in the right paramedian side, then you have to go for a pin, suturing the areas.
- Then lavish fluid therapy to correct the electrolyte abnormalities and dehydration,
- the transformation of the rumen gut, that is we have to collect from the slaughterhouse give 2-3 days, two to three liters.
- Treatment of the primary ketosis is to be addressed here.

In this class, we have discussed the left displacement of the abomasum. In the next class, we are going to discuss the right displacement of the abomasum.

Thank you!

Download

PDF: Left Displacement of Abomasum

4 Right Displacement of Abomasum

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Transcript

Dear vets,

Greetings to all!

In the last class, we have discussed the Left displacement of Abomasum Etiopathogenesis, Clinical signs, Treatment, and Differential diagnosis.

In this class, we are going to discuss, the Right displacement of Abomasum, when compared to the Left displacement, Right displacement is very very rare. The left displacement of Abomasum is most commonly associated with the calving, that too 3-6 weeks after calving is going to develop, but in the case of Right displacement, it is also be associated with the calving and it is also unassociated with the calving. Right displacement of Abomasum always is combined with the volvulus, so if on the right-side ping sound is there, we have to suspect for the right displacement of Abomasum. It is mainly due to the Atony of the Abomasum.

Pathogenesis: it is mainly the Atony of the Abomasum leads to accumulation of the fluid and gas and it will be slowly distending and it will be displaced caudal to the normal position and there is a lot of secretion of HCl hydrochloric acid, Sodium chloride, and the electrolytes other Potassium into the Abomasum. It is leading to

muscular weakness because of metabolic alkalosis, hypochloremia, hypokalemia. Any Abomasum involvement, there will be metabolic alkalosis, hypochloremia, hypokalemia, that is electrolyte and acid-base imbalances. There may be severe dehydration, this should be noticed in the RDA.

The Clinical signs are:

- Anorexia to inappetence,
- depression
- dehydration: the animal is going to develop dehydration
- the right side paralumbar fossa is distended
- you have to go for percussion and auscultation of the right side distended area usually the right displacement of Abomasum is wider in the area when compared to the other areas of ping sound,
- there is a decreased quantity of dung voiding,
- a drop in milk production both LDA and RDA Abomasals there will be a drop in milk yield
- The rumen will be static and doughy in nature
- Rectal examination: you can feel the blind end of the Abomasum, you can palpitate.

Diagnosis is mainly based on the combined percussion and auscultation of the right paralumbar fossa.

(showing picture in the slide) In this picture you are seeing the Right side ping area, here you are seeing that the wider cranial to caudal up to the middle area middle third of the right paralumbar fossa wider area is in involved means that is due to right displacement of Abomasum. More caudal and more upper quadrant means, there may be a chance of Caecal Volvulus and Spiral colon.

Ultrasonography examination of the Abomasum on the 9th to 12th intercostal pair in the midline of the right paralumbar fossa will reveal a hyperechoic wall with hypocrite content. So that area you have to locate, we need to go for the Liptak test. Liptak test is confirming both LDA and RDA, wherever you are getting a ping

sound on combined percussion and auscultation, we need to rule out whether it is the involvement of the Abomasum by doing the Liptak test.

So here also you are puncturing the area of ping and collecting the liquid from the Abomasum. The pH and protozoa motility will be assessed the pH will be 2-4, the protozoa motility will be nil, so that is confirming the RDA. Suppose in case of Caecal dilatation and Spiral colon involvement that won't be there, the Liptak test is negative.

Differential diagnosis: it should be differentially diagnosed with the right-side ping areas like

- Caecal dilatation,
- Ascending colon,
- Pneumoperitoneum,
- Impaction of the Abomasum,
- Abomasum ulcers, and
- Fetal dropsy conditions.

Suppose right side distention is there you can think about the dropsical condition of the fetus also either Hydramnios or Hydrallantois.

Treatment:

- 500 ml of Calcium borogluconate because here the hypomotility of the Abomasum is one of the reasons for the displacement so if you are improving the motility or that smooth muscle contraction by giving Calcium borogluconate, it will be altered or it will go to the original position.
- Then you have to provide adequate good quality hay because the hay more of salivation, it will buffer and it reduces the acidity,
- grain feedings to be discontinued for a while,
- fluid therapy either intravenously or oral is to be given for 2-3 days,

- Mineral oils, so you can also evacuate the content by giving mineral oils 5-10 liters per day you can give either liquid paraffin or vegetable oils also can give,
- Antacids: Magnesium hydroxide, you can give 500 grams per hourly, which will help for neutralizing the more acidity, and
- Hyocine-n-butyl bromide; it's a pain killer that can also be administered to animals that are affected with the right displacement of Abomasum.

So far, we have discussed the Right displacement of Abomasum in this class, in this lesson. and the next class we are going to discuss the Abomasal Atony and Abomasal tympany or Abomasal Impaction.

Thank you!

Download

PDF: Right Displacement of Abomasum

5 Abomasal Bloat and Impaction

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Transcript

Dear vets,

Greetings to all!

Last class, we have discussed the Right displacement of the Abomasum, in that Etiopathogenesis, Clinical signs, Treatment.

In this class we are going to discuss two diseases; Abomasal Bloat and Abomasal Impaction

Let us see **Abomasal Bloat** first: the Abomasal Bloat is mainly seen in the case of calves and lambs;

- due to Clostridium perfringens Type A infection, Sarcina species, Salmonella species Lactobacillus species. So, these are the bacteria, that will have a multiplication, will lead to Abomasal Bloat
- mainly the exact etiology is not yet been identified even then these all the other etiological agents postulated,
- another thing is the calves, lambs fed with milk replacers,
- either in the form of too chill and too warm that will have putrefaction in the stomach, so the abomasum is going to be bloated and
- sometimes high glucose concentration administration for any

kid or lambs, calves not taking adequate milk forcibly if you are giving that will also increase the fermentation leading to of Abomasal bloat.

The **Pathogenesis** just distended abdomen due to gas and it will be compressing the thoracic and abdominal viscera and blood vessels that will occlude the abdominal vessels. So, automatically lead to Asphyxia by compressing the diaphragm, the breathing difficulties will come and it will also affect the circulation. It is leading to acute circulatory failure and loss of venous return to the heart, it is going to be happening and leading to shock. The animal will be going to develop shock.

The **Clinical signs**: major clinical signs are:

- Acute bloat suddenly will develop good bloat
- the anorexia not taking milk and it is restless, it's not willing to sleep, not listening to any stimuli
- it will be dull and depressing
- diarrhea will be developing because of the bacterial overload in the abomasum leading to indigestion which leads to diarrhea
- some of the causes may show Colic science
- when you are doing the percussion auscultation of the abdomen either both sides, will have a ping sound as I already told you it is because the liquid medium is there in the hollow organ and the air is under the pressure, top of the liquid medium air is under the pressure, it is leading to ping sound and
- the cold extremities due to shock
- Tachycardia, Tachypnoea will be there.

Treatment:

- you have to have a lavage, passage of stomach tube maximum possible you evacuate the content and remove the air also, so for further fermentations to be avoided by passage stomach

tube you have to remove as much as possible that fetid fluid from the abomasum and

- some of the cases not responding properly not yielding for the gastric levels, you go for a Laparotomy and you can evacuate the abomasum
- you need to advise the owner to have proper milk replacers, it should be in adequate heat and it should not be kept for a prolonged time, so that will be putrefying then it will lead to problem. so adequate management of milk replacers supplementation to be there.

The next disease is **Abomasal Impaction**: it is commonly seen in the case of large ruminants like cattle, buffalos;

Primary causes

- it is mainly because of some fibrous feed intake, suppose the more fibrous, indigestible fibrous material is taken that is escaping through the reticulo-omasal orifice entering the abomasum, will have an Impaction
- sometimes lack of water intake, the animal which is there in the winter is not taking adequate water, which will also lead to the development of the Abomasal Impaction.

Secondary causes

- Pyloric outflow obstruction, any tumor condition, or any inflammatory condition of the pylorus will lead to Pyloric obstruction. Stagnant of the fluid, only a stagnation of the feed material in the abomasum, only the fluid portion will be drained to the intestines.
- then adhesions, sometimes any displacement, adhesions, actually adhesions due to abomasal ulcers may also influence the Abomasal Impaction and
- Lymphosarcoma and
- Conditions like Peritonitis, TRP there is a fluid accumulation in

the peritoneal cavity will alter the motility of the abomasum, it is going to develop Impaction and finally

- the vagal nerve, which is also supplying, I think posterior functionalities already we discussed, isn't it, in the vagal digestion that is having some role Vagal nerve injury will also influence over the Abomasal Impaction.

What are the **Clinical signs** we can expect in Abomasal Impaction; there are no specific clinical signs, all are like Abomasal Involvement:

- Progressive abdominal distension on the right lower quadrant
- Intermittent appetite, the animal will not take proper feeding
- the dung material will be loose and it will be somewhat watery in nature and reduced quantity
- the animal is going to develop weight loss, chronic Abomasal Impaction is going to develop weight loss,
- rectal examination, if you are doing there is a dorsal and ventral sac of the rumen is distended, because the abomasal distention is there, automatically the content will be staying in the forestomach,
- the animal feels pain and is exhibiting a Bruxism
- you can do the palpation on the right lower quadrant in the abdomen, you will get a deep pain, elicitation can have, and
- all the vital signs like temperature, heart rate, pulse rate, the respiratory rate will be normal
- then Bradycardia- secondary to the vagal nerve in irritations and
- Metabolic alkalosis as I told it is having Metabolic alkalosis, hypochloremia, hypokalemia.

Diagnosis: only thing is you have to go for right side Laparotomy and open the area or else you can go for Rumenatomy and you can through the ruminative wound you can also examine the abomasum, whether it is impacted or distended.

Treatment:

- during the Laparotomy or in the Rumenatomy, you have to pass the tube into the abomasum, if you are doing Rumenatomy, you pass the stomach tube or some tubes through the reticulo omasal orifice to enter into the abomasum, you have to infuse some mineral oil and some other materials like dioctyl sodium succinate.
- you have to give adequate laxatives to propel the content from the abomasum
- you have to increase the motility by giving Ruminotorix
- and the recent treatment, researchers have given is, Coffee-one pound is given to the rumen, it is also one of the important materials which help to alleviate or rectify the Abomasal Impaction.

In this module, we have seen:

- Abomasal Ulcers,
- Left displacement Abomasum,
- Right displacement Abomasum,
- Abomasal Bloat in lambs and calves, and
- Abomasal Impaction

I hope that these modules would have helped you to identify the field problems and are able to treat them successfully in the field condition.

Thank you!

Download

PDF: Abomasal Bloat and Impaction

PART IV

WEEK 4: RUMEN AND OMASAL IMPACTION AND DYSFUNCTION IN CATTLE

1 Introduction to Rumen and Omasal Impaction and Dysfunction in Cattle

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Transcript

Dear vets,

Greetings to all!

In this module, I am going to discuss Rumen and omasal impaction and dysfunction in cattle. As we know, the Rumen reticulum Abomasum comes under the category of four stomachs. Any four stomach dysfunction is directly affecting the production, so in this module, we are going to learn about; Etiopathogenesis, Clinical findings, Diagnosis, and Treatment of the four stomach disorders. So that will help us to treat the cases effectively in the field condition and by the way, it is improving the economic status of the farmers.

In this module I am going to discuss five lessons;

- Lesson one will cover the Introduction and a list of Rumen omasal Impaction and disorder dysfunctions in cattle and Simple indigestion in ruminal impaction,
- Lesson two covers Ruminal lactic acidosis,
- Lesson three; is going to cover three diseases subacute ruminal acidosis (SARA), Ruminal drinkers, Ruminal alkalosis,

- Lesson four is going to cover Ruminal tympany, and
- Lesson five includes Vagal indigestion and omasal Impaction.

Let us enter into Lesson one:

The **Introduction**

You know that Rumen is a hollow organ, which is responsible for the microbial digestion and production of volatile fatty acids (VFA) like acetic acid, propionic acid, and butyric acid so these volatile fatty acids are mainly responsible for milk production. So, any dysfunction of the rumen reticulum omasum will directly affect the production of the volatile fatty acids, in turn, it affects the reflection on the production.

In this module I am going to cover the following diseases:

- Simple indigestion
- Rumen impaction
- Acute carbohydrate engorgement
- Subacute ruminal acidosis
- Ruminal drinkers
- Ruminal alkalosis
- Ruminal tympany
- Vagus indigestion
- Omasum Impaction

Simple indigestion:

What do you mean by Simple indigestion; any change in the feeding regimen automatically affects the digestibility of the ruminants, so ruminants are very keenly responsible for the change in the feed, so whenever you are changing automatically it will affect the digestibility of the animal.

Let us see **Etiopathogenesis**: so, any derangement in the feeding in the ruminants automatically will affect the rumen function, so the main outcome is Ruminal atony, what are the reasons for which it is going to occur: one is excessive carbohydrate engorgement, Putrefaction of protein, moldy feeds of protein sources if it is not

good quality and it is due to limited water access and it is due to sometimes Anaphylactic or allergic reactions because of high Histamine production will affect rumen and motility. So ultimately the animal will have Rumen atony, so Rumen atony will reflect on the digestibility of the animal, so automatically it will reflect on the production.

What are the **Clinical signs**: it is associated with simple indigestion,

- sudden change in the feed
- there is a dull and depressed animal
- suspended rumination
- The rumen will be a little bit distended and
- palpation will be a doughy rumen and
- there will be reduced rumen motility
- the temperature, pulse rate, heart rate, will be normal and
- milk production will be reduced

So, in simple indigestion, if you are seeing the dung initially will be (in the picture you have seen) that dry pellet dung, drier than the normal level, and in case of 24 to 48 hours after simple indigestion, there may be profuse diarrhea, maybe a voluminous and malodorous, it is called as Dietary diarrhea. How to confirm the disease, you need to take Rumen liquor, you have to collect the Rumen liquor bypassing the stomach tube and with the help of the suction apparatus, we have to collect the Rumen liquor, immediately you have to transform into the closed containers, that should be subjected to detailed investigation like protozoa motility and pH and MBRT Cellulitic Cellulose digestion test, etc.

In the picture, we are seeing that Altered Rumen pH, normal Rumen pH is 6.2 to 7.2, if it is below that it is called Acidosis, and if it is above that it is called Alkalosis. You need to go for an assessment of the Rumen liquor in another way, by assessing the protozoal count and protozoan motility.

In this video, we are able to see some small protozoa moving

in the low-power microscope. so that indicates that if the Rumen microflora is altered now only the regeneration is going to start. In the next slide, next video, we could able to see the medium size, small size, large size protozoans, they are humpy numbers, more than 30 plus the per low power microscopic slide, it is indicating that three-plus. so that could be a good protozoan that is the normal protozoa.

Mainly a Diagnosis of simple indigestion is done by:

- History of any change of feeding, sudden change of feeding, or giving any new feed
- Clinical signs of doughy Rumen suspended rumination and decreased Rumen motility and
- Based on the Laboratory tests like Rumen fluid examination, we are able to diagnose the cause as simple indigestion. The simple indigestion is going to occur because of a reduction in the feed intake, that's an ultimate clinical sign we are going to observe.

So, this is going to happen in all other diseases also, that should be differentially diagnosed. In this context one is Acetonemia- that is the ketosis and acute carbohydrate engorgement, Ruminal alkalosis, left displacement Abomasum, right displacement Abomasum, Traumatic reticuloperitonitis, and other conditions like deficiencies say hypocalcemia all are included in this differential diagnosis.

We have to go with the differential diagnosis and treatment for simple indigestion.

The **Treatment** of simple indigestion is very simple, you have to go with the normal Stomachic, stomachic means- it is having bitters, it will increase the salivation by the way it improves the appetite.

Next is Probiotics; if there is any protozoan motility is low and protozoa are very less in number, so we need to go for probiotics to improve the microbial status of the Rumen liquor, but that will increase the digestibility.

Then another one more thing is if the Rumen atony is there we need to go for Ruminatorics; that is tartar emetic, ginger- sometimes ginger is also another Ruminatorics, and you can also go for some preparation available in the market like Rumentas it is having an Antimony Potassium Tartrate, Cobalt Chloride, and a Ferrous Sulfate, so the Antimony Potassium Tartrate-it is irritating the rumen by the way it is improving the motility of the rumen. okay

Parasympathomimetics– you can also give Carbamylcholine, Physostigmine, Neostigmine, this will increase the motility of the rumen and you can also give some Metoclopramide at the dose rate of 0.3 milligrams per kg body weight if you are giving that will be acting as a progenetic, it increases the rumen motility, it will be useful for in case of vagal nerve damage also.

Laxatives– you have to give adequate Laxatives to improve the rumen output, then lavish fluid therapy- you have to give a fluid therapy. Then if this is Acidosis you have to go for alkalizing agents and if it is Alkalosis you have to go for acidifying agents. another more important thing is in case of simple indigestion, the flora and fauna are affected, we need to go for Cud Transfaunation- cud Transfaunation we can have it from healthy animal/healthy cattle, we can collect it and you can drench, 2-3 liters per day for consecutive three days, if it is not available you can go for slaughterhouse collection of the cud Transfaunation, you can collect from the sheep or goat or cattle, you can take the ruminal content, only the watery portions to be drenched without aspiration ammonia for 3 days 2 liters per day. The other management aspects regarding simple indigestion are:

- provision of the palatable hay, it will improve the resistibility by the way of increasing the saliva and
- if there is atony of the rumen you have to go for Calcium borogluconate, In the ruminants always whenever you are going for managemental advice, we have to tell the owner that gradual inclusion of a new feeding diet 7 to 14 days interval you have to take to introduce a new feeding.

Let us discuss **Ruminal Impaction**:

Etiopathogenesis: It is almost like simple indigestion, it is due to indigestible feed material or any foreign bodies. The metallic foreign bodies are directly causing some damage to the wall and leading to Traumatic reticuloperitonitis and non-metallic foreign bodies like plastic bags wire, rope, cloths, leather, will be stay in the reticulum omasal orifice and it will be having a significant alteration in the rumen dysfunction.

The **Clinical signs** are:

- the distended left side paralumbar fossa,
- when you are palpating the rumen it will be doughy or sometimes it may be harder in nature, firm in nature
- the animal is going to lose its weight, over a while it is going to lose weight,
- lack of feces in the rectum, reduced quantity of feces dung will be there in the rectum, ● abdomen distension and lack of symmetry
- abdominal will be dispensed on the left side
- Foamy salivations
- Recumbency and then the advanced stage of any Traumatic reticuloperitonitis is going to recumbent.

Diagnosis:

- based on the indigestible feed material or metallic foreign body injection
- another thing is based on the clinical findings like emaciation, rumen distension, reduced or dung voiding, and
- You can also do the ultrasound examination on the left side of the abdomen at the level of the 11th or 12th intercostal space using the 3.5-megahertz ultrasound probe. You have to infuse 1.5 to 2 liters of water before going for an ultrasound examination, then only you will be able to properly visualize any foreign bodies which are present or any alteration in the

rumen wall also you can assess.

- The next thing is Oesophageal and ruminal endoscopy- you can also use endoscopy to identify the foreign body lodging in the choke in the Oesophagus or any foreign bird which is there in the reticulum or rumen.

Treatment of the rumen impaction;

- If it is due to the indigestible feed material, we need to go for oral infusion therapy along with the laxatives.
- Laxatives you can use magsulf and
- along with the water you have to trench into the rumen by using a rumen infusion pump, so that will increases the consistency of the rumen content is to be dissolved and it will be moving towards the hindgut
- if it is due to any metallic or non-metallic foreign bodies, it will be lodged in the rumen, so the only treatment is you have to go for Rumenotomy. so, with that, we can save the animal.

Dear vets in this first lesson, we have discussed the

- Introduction ● list of rumen omasal impaction and dysfunction in cattle
- Simple indigestion
- Ruminal impaction
- Etiopathogenesis
- Clinical signs diagnosis and
- Treatment

In the second lesson, we are going to discuss Ruminal Lactic Acidosis which is otherwise called Carbohydrate engorgement.

Thank you!

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Introduction Rumen and Omasal Impaction and Dysfunction in Cattle

2 Ruminal Lactic Acidosis

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Transcript

Dear vets,

Greetings to all!

Last lesson- lesson one we have discussed the Introduction of Rumen omasal impaction and dysfunction in cattle, a List of disorders related to the Rumen omasum in cattle, and Simple indigestion and Ruminal impaction, in which we have discussed Etiopathogenesis, Clinical findings, Diagnosis, and Treatment of Simple indigestion and Ruminal impaction. In this second lesson, we are going to discuss a very important disease in the field condition, it is having a lot of influence and diagnosis skills at the field level. So, it is mainly because of the man-made problem because of the management aspect, excessive feeding that is called acute carbohydrate engorgement or Rumen lactic acidosis. This is a main problem in the field conditions, if you have diagnosed the condition, it is very easy to treat. For that, we need to learn something regarding Etiopathogenesis, Clinical signs, Diagnosis skills, and Treatment.

Let us enter into the topic of proper **Etiopathogenesis**; excessive carbohydrate ingestion will lead to the production of lactic acid, the lactic acid is responsible for so many pathophysiological changes

in the rumen. So let us take one by one it is also increasing the carbon dioxide production in the cellular respiration, by this way it is leading to Kussmaul's respiration that is called commentary hyperventilation, so increased elimination of carbon dioxide, that's the first animal is brought with the symptoms of some respiratory distress, in case of lactic acidosis and another thing is the Gram-positive bacteria will be dominating like a Gram-negative bacteria is decreasing, Gram-positive bacteria are dominating. So, it leads to the lactic acid's corrosive nature. Sometimes it is going to leads to some damage in the ventral half of the rumen and there is a chance of getting perforation, which is leading to acute peritonitis and it will also lead to toxemia.

The next thing is that lactic acid is going to cause Chemical rumenitis and Mycotic rumenitis, there is a chance of three to four days after the lactic acid accumulation in the rumen it will favor the multiplication of absidia, mucor, rhizopus this fungus will be more predominant. So that is leading to Fungal ruminant. Sometimes the lactic acid is absorbed in the system and it will go for systemic academia low pH and lactic acidemia are there in the system circulation, which is leading to further anaerobic metabolism, the production of further lactic acid in the system. That is again reducing the blood pressure, which is leading to hypotension. The ruminal vessels will be damaged through the rumen vessels, the fusobacterium necrophorum will enter and go to the liver, and there it is causing liver abscess, small nodular liver abscess, that is called sawdust liver.

Then histamine is released because of the acute carbohydrate enrichment in the rumen and that is leading to laminitis, it is going to accumulate in the current region and animals will be finding difficulty in walking. Then the growth of the rumen papilla, will be more and it will be altered so that will be leading to ruminal parakeratosis, thus all the clinical-pathological changes are going to occur because of the lactic acid in the rumen.

Let us discuss Clinical signs: In mild cases suppose ingestion of carbohydrates source in the morning, rice, or whatever memorize

crude oil in the morning. So, the animal will be showing the symptoms of anorexia, dullness, depression and sometimes it is a mild dose of carbohydrate engorgement, the animal may recover spontaneously within 3-4 days, so the initial period of indigestion only is going to happen.

Next severe cases- suppose if it is a severe case, the animal may go for recumbents also sometimes, in acute cases there may be a severe production of or heavy production of lactic acid is going to happen. Here the lactic acid is hygroscopic in nature, so that's why drawing the water from the systemic circulation leads to severe dehydration and if you are seeing the left paralumbar fossa, it will have a fluid splashing sound and rumen motility will be reduced, suspended rumination is there and the animal will be going to develop a drunken gait, so incoordination, ataxia will be there due to this dehydration and lactic acidemia and the animal is having diarrhea, it is initially it will be somewhat semi-solid nature and it is voluminous it is small waters due to that, the dietary diarrhea is prognostic in the phosphate prognostic indicator it should be there, so the purified materials should come out, then only the animal is going to recover. So diarrhea animals may recover, the animal which does not have diarrhea in acidosis is going to suffer, in severe cases, and the animal is going to have laminitis. In the picture you could able to see the sunken eyeball, you have to see that there is a gap between the eyeball under the eyelids, and another test is to be there to assess the dehydration by skin tenting test, you have to pinch the grass the neck skin and leave it for a second it is 1-2 second within that you have to it should have normalcy. If it is persisting as a tent that is indicating dehydration more than 8% or 10% like that. You have to do the succussion, so to identify whether the animal is having any fluid splashing sound or not you have to do the succussion on the left paralumbar fossa, which will have a fluid splashing sound over the left paralumbar fossa. You can also occult it or you can also hear the normal with the distance also. You have to collect the rumen liquor and you have to go for a physical examination. The color of the rumen liquor in case of acidosis is

milky green and it is sour-smelling and watery in nature. Then if you are seeing the pH and protozoa, protozoan won't be available. The protozoan is nil almost all protozoans would have been dead and the low pH is not conducive for the survival of the protozoan then pH will be below the normal range of 6.2 -7.2 is a normal range it is below that 5-6 is the normal range. you can see the rumen fluid with a pH of around 5-6.

In the picture, the **Diagnosis** of rumen lactic acidosis or carbohydrate engorgement is mainly of history. History of excessive intake of carbohydrates. Sometimes the normally, routinely is having a lot of carbohydrates but a little bit more excess will also lead to carbohydrate engorgement.

Then **Clinical signs**: Sunken eyeball, fluid splashing sound, abdominal distension, laminitis all together is going to give some idea. Then rumen fluid examination is going to confirm the ruminal lactic acidosis disease. This disease is going to be differentially diagnosed with simple indigestion, Toxemia- some animals going for recumbency. In case of severe acidosis also, animals are going to be recumbents. So, you have to go to examine the udder and any peritonitis is there, you have to go for peritonitis synthesis. So, the peritonitis or the coliform mastitis will lead to toxemia and subsequent recumbency in animals will also simulate acute carbohydrate engorgement and you have to differentiate diagnosis with the subacute rumen acidosis, here subacute rumen acidosis – the animal is fed with a continuous carbohydrate source, the animal would have been adopted for that but even then the animal is having diarrhea and suddenly sometimes we may have indigestion problems. So, we have to look for subacute rumen acidosis by history, it is a herd problem.

Parturient paresis- that means milk fever, in case of milk fever the animal will be going for recumbency, the lateral kink of the neck will be there. So, to differentiate milk fever from acute carbohydrate engorgement, we need to take the rumen liquor and you have to examine and you have to do the succussion on the left paralumbar fossa, if the fluid splashing sound is there that is a major clinical sign

related to the acute carbohydrate engorgement or ruminal lactic acidosis.

Treatment in Parturient paresis: emergency treatments to be there. The first thing is we need to lavage the rumen as much as possible to remove the lactic acid present which is there in the rumen to be evacuated as much as possible, bypassing the stomach tube and sodium bicarbonate administration. Sodium bicarbonate administration, you can do it with the millie equivalent of sodium bicarbonate requirement is equal to 0.3 multiplied with a base deficit that will be measured based on the clinical signs and laboratory examination, and body weight. so that will give a milliequivalent of sodium bicarbonates to be administered to the animal.

mEq of $NaHCO_3$ = 0.3 * base deficit * BWT(kg)

So, if it is not done we can go for 5% – five liters of sodium bicarbonate intravenously over 30 minutes and 1.3% of this isotonic sodium bicarbonate solution that can be administered 6-12 hours like a 150 ml per kg body weight. But at the field level, you will calculate the sodium bicarbonate requirement and you can administer it, while giving the sodium bicarbonate it is very simple you are having a 7.5%, 8.4% sodium bicarbonate solution available in the market. We are going to dilute with the distilled water to administer the sodium bicarbonate at the rate of 5% only we are going to administer to the animals. That too you have to mix it with the normal saline, so in mild cases, Ringer's lactate can also be given, when the circulation is proper. The lactate is going to be liver and it will be converted into bicarbonate that will take care of the rumen acidosis, but in case of severe dehydration you have to try to avoid Ringer's lactate, so because the circulation is Ringer's lactate, we are giving the periphery it is not going to the liver and it is not going to convert. Again, the lactate is going to deteriorate the condition. Here two things are there: L lactic acid, D lactic acid. The D lactic acid is actually persisting, lactic acid is metabolized.

And another treatment is Oral: so, our aim is first to evacuate the content and give bicarbonate to systemic acidosis and we are

going to give some oral antacids like a Magnesium hydroxide or Magnesium oxide at the dose rate of 500 grams per animal, you have to mix it with the 10 liters of water and so that will help neutralize the pH in the rumen. other Ancillary treatments like Antihistamine because of histamine release there will be laminitis, you have to give Antihistamines, Chlorpheniramine we can give and because of inflammatory changes is going to happen in the rumen ventral wall, we need to give Non-steroidal anti-inflammatory drugs (NSAIDs), but that would also be judicially used and Thiamine, the rumen is only in ruminants the rumen is responsible for the synthesis of B complex, vitamins we need to submit the B complex, vitamins especially Thiamine, which is responsible for the so many biochemical reactions in the body and you can also move that content by giving Laxatives[1]Parasympathomimetics and Calcium borogluconate. Calcium will also be low in some cases of Lactic acidosis, we need to supplement calcium borogluconate and Oral antibiotics as we know that there is a Gram-positive bacteria domination usually there should be Gram-negative bacteria. So, we need to just kill the Gram-positive bacteria by giving oral antibiotics, especially Sulfonamides and oxidative cycling. Then one of the important things the next day, first today you have to treat this, then probiotics you want to give probiotics that will help for the regeneration of the microflora and it will also help for the metabolism of the lactic acid. The Cut transplantation; as we discussed in this simple indigestion we need to collect from the healthy animal or from the slaughterhouse, which should be administered 2-3 liters within half an hour to one hour of collection. Should not be stored for a long time. During the transit, it should be closely, tightly capped, and should not be having exposure to the environment. The other important management advice to the farmer is to restrict the water intake, in case of ruminant lactic acidosis the animal is willing to take more water it is willing to take but it is going to engorge, and it will develop some aspects here it will engorge the rumen and it will compress the diaphragm, it is leading to some motility in animals. So, you have to give water

but in a restricted manner. So, half bucket, two hours after that you are given another bucket. Another thing is we have to go for giving the palatable hay, so that will help rumen motility by irritating the rumen mucosa, so the animal regains its normal appetite.

In lesson two we have discussed Etiopathogenesis, Clinical signs and Diagnostic Aspects, and Treatment of ruminal lactic acidosis in cattle.

In lesson three we are going to discuss three topics: Subacute ruminal acidosis, Ruminal drinkers, Ruminal alkalosis.

Thank you!

Download

PDF: Rumen Lactic Acidosis

3 SARA, Ruminal Drinkers and Ruminal Alkalosis

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Transcript

Greetings to all,

In lesson two we have discussed rumen lactic acidosis. In that Etiopathogenesis, Clinical signs, Diagnosis, and Treatment and control you have discussed. I hope that would have been very useful to you. In this lesson, that is lesson three we are going to discuss three topics one is Subacute ruminal acidosis, Ruminal drinkers, and Ruminal alkalosis.

Let us first discuss the,

Subacute ruminal acidosis: is a management problem so here the acidosis is going to be there in a continuous way in the herd. So the animals which are continuously taking excessive carbohydrate source and low number/less amount of fiber diet, it is going to happen in the herd problem, it is going to be a herd problem, so what will happen in this situation, excessive carbohydrate is taken animal will adopt for that carbohydrate source and it will maintain the pH from 5-6, but it is not going to happen as alike clinical sign like that of acute carbohydrate engorgement, the animal will have signs like diarrhea, laminitis, and it is having some reduction in the

milk yield, low milk fat like that. So here is what will happen, that will lead to cellulose digestion bacteria/fiber digestion bacteria will be low, other cellulose bacteria will be low and the sugar starch digestion bacteria will be dominating and there will be lactate replacing that is producing bacterias also more.

So let us discuss Pathogenesis; so continuous ingestion of the carbohydrate source, the less amount of roughages intake, will lead to the subacute ruminal acidosis by reducing the rumen pH, by the way of producing multiple fatty acids and also lactate content, so it will affect the lactate and the high level of volatile fatty acid leading to low pH, that will affect the cellulolytic bacterial activity and fiber digestion is impacted. So that will lead to production loss in the case of cattle.

Clinical signs like diarrhea intermittently semi-solid nature, mold waterous in nature and some of the animals may have a distended abdomen, loss of bodily conditions and some animals going to show some laminitis, hoof problems. So, the animal had some laminitis problems in the herd. So collectively we have to examine the herd, we should inquire about the history, if excessive carbureted sources are given continuously, that should be addressed.

In some cases, it may also lead to Haemoptysis and epistaxis associated with venal caval thrombosis and pulmonary hemorrhage. In this case, there is fiber is to be there to have more production of the acetic acid, if the acetic acid production is less in case of carbohydrate intake, that will lead to milk fat depression that's one of the important things, so the owner may report that milk fat depression is there in a herd and the diarrhea is there and the animal is having laminitis, you have to think about subacute ruminal acidosis.

The Clinical signs: you can see the picture, you will be able to see there some other hoof abnormalities, so it may be a

- Ridges development in the hoof or overgrown hoof,
- Sole ulcerations,
- Sole hemorrhages,

- The white line in the hooves and
- there will be misshapen hooves.

So these are the things that are there in the herd, we need to think about some common problems, it's maybe because of excessive carbide administration or it may be due to some mineral deficiencies.

The **Treatment**; we have to go for

- Antacid treatment, so Magnesium hydroxide 500 gram for 450 kg body weight, you can give orally,
- the buffering diet: diet should be modified in a way it should have a buffering,
- so, the animal should be provided adequate palatable hay, by the way, it is improving the mastication, so more amount of saliva is secreted, so that will go for a buffering in the rumen,
- you have to fit the feeding of concentrate in different quantities at different times morning and evening how to split and give, by this way you can reduce a load of volatile fatty acid and lactate levels in the rumen, and
- Another thing is feeding the roughages first, then you have to go for a concentrate that will help the animal to elevate from this condition.

Next, let us discuss the **Ruminal drinkers**: it is seen mostly in the case of calves. calves that are weaned on the first day itself from the form. From the mother, the form condition, so that will lead to this type of condition when it is administered the milk, is administered in the form of bale feeding, that is drinking the milk as such, not suckling the milk, so what will happen drinking the milk will not induces the suckling reflex, so the suckling reflex is very much useful or needed for the closer of the esophageal groove. As we know that young calves may be having more dominant abomasum and an ill-developed rumen reticulum omasum, so in the third, to fourth month only the rumen reticulum abomasum is going to

be a normal structure, as in the case of adult animals. So in this case what will happen because of the ill-developed rumen, is the rumen reticular esophageal groove is not properly closed because of suckling reflex is not there, so the milk is directly entering the rumen and there will be putrefaction, the putrefaction will lead to casein clots and graze rumen liquor and animal having abdominal distension and depression, dehydration, it is going to be recumbent and it will also have a clay-like feces and when you are just doing the succussion on the left paralumbar fossa, it will have a fluid splashing sound. So when you are going for any detailed investigation of any dead calves, there will be villainous atrophy, and marker ruminal parakeratosis will be there because it is favoring the growth of a dark color rumen epithelium.

Diagnosis is based on the history of bale feeding and clinical signs, which we have discussed, and another one more test you can adopt in the field condition is the Acetaminophen absorption test which is to be done in a field condition.

- The paracetamol at the dose rate of 20/25 to 30 milligram per kg body weight mixed with the 2 liters of milk, that can be administered to the calves, then you have to wait for some time, you have to take the sequential blood level and you have to estimate the paracetamol level.
- The calf with the ruminal drinkers with the flat Acetaminophen absorption curve will be there so that is indicating that the animal is affected by the rubric drinkers.

The management you have to provide the nipple feeding of milk, in the nipple feeding the nipple pore will be very small. The calf should have more force to suck then only the esophageal groove will be closing, which will make the milk directly enter into the abomasum. The next one is you have to give a milkman's hand or fingers to suck, so bale feedings to be avoided.

The **etiological** factor for **Ruminal alkalosis** is very simple, it is sometimes maybe because of protein putrefaction or NPN

(NonProtein Nitrogen) sources excessive administration of urea toxicity. In the urea toxicity, sudden death will be there with the bloating so when you are going for an autopsy you will be able to find out that ammonia generation is more in the groove. But when compared to the other ruminal disorders, ruminal alkalosis is very rare, it is not going to happen in the field condition but confirmation is done only with the rumen fluid collection and examination.

The Clinical signs are there will be a

- Ruminal hypomotility,
- Tympany,
- Vomiting, some animals may have a regurgitation,
- Severe abdominal pain,
- Muscle tremors, the animal will have trembling muscle tremors,
- Muscular weakness,
- Incoordination ataxia,
- Tachypnea, and
- CNS excitation, the animal will develop CNS excitation and die.

In case of urea toxicity, suddenly after injection of the heavy amount of urea, it will be developing hyperammonemia and ammonia is more in the rumen and it will be circulated in the blood and there will be a CNS toxic and sudden death will be there.

Diagnosis:

- We have to collect the rumen liquor and you have to examine the rumen liquor, the pH is more than 7.5, that is indicating that there is a Rumen alkalosis, the reason may be either it may be a protein denaturation or it may be due to urea or NPN (NonProtein Nitrogen) sources excessive injection.
- So one more urea injection is through the urea-treated straws, if it is excessively fed sometimes the urea is more concentrated in the straws which will also affect the condition, rumen dysfunction will lead to alkalosis.

- The strong ammonia odor of the rumen content will be there, so in this picture, we will be able to see the alkaline pH of 9, which we can see in the ruminal alkalosis.

Treatment:

- We have to give Acetic acid or vinegar, so the alkalinization agent is to be utilized as an acidic one. Give acetic acid around the 2-6 liter into the intra-ruminally you have to give bypassing the rumen infusion pump. So, if you want you can also dilute with the water and you can administer.
- Then intravenously normal saline should be administered and other B-complex, Vitamins Supplements to be given.

In this lesson 3, we have discussed the

Subacute Rumen Acidosis, Ruminal drinkers, Ruminal alkalosis in detail.

So, in the next lesson, that is lesson 4, we are going to discuss the Ruminal Tympany.

Thank you!

Download

PDF: SARA, Ruminal Drinkers and Ruminal Alkalosis

4 Ruminal Tympany

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Transcript

Dear vets,

Greetings!

In lesson three, we have discussed Subacute Ruminal Acidosis, Ruminal drinkers, Ruminal alkalosis. In this lesson four, we are going to discuss the Ruminal tympany.

Ruminal tympany: it is nothing but an accumulation of air in the rumen, it is either in the form of a persistent foam or a free gas in the rumen. So, the persistent foam is because of either leguminous fodder administration, or it may be because of feedlot bloat. The gas bubbles in the persistent foam, it does not coalesce together to form a bigger gas bubble to eruct it. Once the cardia is touched by the bigger gas cap, then only they will have eructation. Small gas bubbles are trapped, it is not going to eructate, it is making the bulk in the rumen fluid, is leading to Frothy bloat.

The types of bloat, as we already discussed: it is primary ruminal tympany or secondary ruminal tympany. Secondary ruminal tympany is mainly because by some other conditions like infectious conditions, choking, or any other disease conditions affecting the esophageal lumen and it is leading to eructation failure. Primary ruminal tympany mainly occurs in two ways one is by the ruminant's

ingestion of leguminous fodder, it is more gas-producing nature, it is leading to pasture bloat and another one more thing is the carbohydrate source to the rumens is finely ground and given that will lead to feedlot bloat, so the primary ruminal tympany is the dietary origin, we need to treat accordingly.

What are the other conditions responsible for the ruminal tympany in cattle? one is *Vagal indigestion*: in vagal indigestion initially, there will be a hypermotility of the rumen. So here the eructation failure is there, the frothiness of the rumen liquor is there, so there won't be any proper eructation, and bulkiness of the rumen is going to occur. so Vagal indigestion is going to happen in two ways that are; Anterior functional stenosis and Posterior functional stenosis. Both will lead to the accumulation of content in the room, and it is leading to bloat.

In the case of *Diaphragmatic hernia* DH: the diaphragm is having some wind the reticulum is herniated into the thoracic cavity, which is trapped, so normal motility of the reticulum is needed for the eructation mechanism, so the primary or secondary ruminal cycles will be eructed. so, the eructation mechanisms won't be proper, and another thing is during the diaphragmatic hernia, hypermotility of the rumen is there, again it will lead to free gas bloat is turned into frothy bloat. Then Unusual posture: when the animal is in recumbency, either the Lateral recumbency or Sternal recumbency, the content in the ventral abdomen is just compressed up, so that is the fluid medium is touching the cardia, the cardia is not opening when the fluid is touched. so the fermentation is keep on going, it is leading to again bloat. so, the Sternal recumbent animal Lateral recumbent animal, bloat is because of this condition. Then in the case of calves, as we already discussed in ruminant drinkers the abomasal bloat, maybe having distension abdomen, that is having a tympanic resonance.

Another one more condition is an enlargement of the thymus in animals.

Pathogenesis of the Frothy bloat: it is occurring in two ways.

one is pasture- so the animal taking leguminous fodder, it is

having chlorophyll, the chloroplast, that is negatively charged, it is going to be combined with the sodium, potassium, and calcium in the rumen, so any cat-ions are there, that ionic substance of chloroplast will be combined, it is favoring some of the micro-organisms, colonize together to form an entrapped gas bubbles. These gas bubbles have more surface tension, it is not collaged together to form a small gas cap, so all small gas bubbles are persistent as such that is making the bulk, that is leading to frothy bloat.

The next one is Feedlot: when the concentrate is highly powdered like a less than 388 nano micrometer in diameter that is favoring some bacteria like Streptococcus Bovis, it is coming to act on that particular area that is producing slime, some thick foamy like thing is there that will coat over the gas bubbles, it is not making the gas bubbles to collage together to form a bigger gas bubble. So what will happen this will lead to the bulkiness of the rumen liquor it is called feedlot bloat.

What are the causes associated with the Secondary Ruminal tympany – physical obstruction of the oesophagus and some infectious like Tetanus, Listeriosis, Botulism, and Granulomatous lesions in the either intraluminally or extra luminally in the obstructing the oesophagus or Amphistomiasis which is clogging the cardia and some of the other conditions like Anaphylaxis Hypercalcemia and Atony of rumen due to acidosis ruminitis, whether the primary or secondary ultimately there will be a persistent distention of the abdomen it is leading to Hypoxia that is decreasing the lung capacity by compressing the diaphragm and leading to hypoxia and death.

Clinical findings are:

- in case of bloat, sudden death will be there, the animal is grazing in a land which is having leguminous fodder, coming to the shed, night hours it is going to develop bloat and unnoticed it is going to develop a sudden death. Sudden death is another one more clinical entity of the frothy bloat.

- Then the animal will have a distended abdomen,
- projectile vomiting, regurgitation may be there, the animal will be restless, and soft frequent dung elimination,
- Hypermotility of the rumen in the initial stage will also lead to hypermotility in another stage,
- then Systolic murmur, in some cases may be absorbed.

In the picture you can able to observe the full distension, the left paralumbar fossa is fully distended, how to confirm the frothy bloat or free gas load, you can percuss the left paralumbar fossa, if it is tympanic resonance it is free gas bloat, it is frothy bloat there will be a dull sound. Another one more way you can pass this stomach tube is if free gas bloat is there it will be relieved as such quickly and in frothy bloat you will get, you can see the picture there is a frothiness, this is making the bulk so when compared to the free gas bloat, frothy bloat is to be addressed first.

Differential diagnosis; what are all the conditions associated with this boat, already we have discussed :

- Vagal indigestion,
- Infectious causes,
- Diaphragmatic hernia,
- The sudden death of any bloating cases may be different diagnosis black Hodder, lightening stroke, Anthrax, snake bites these are the causes we can very well attribute for sudden death, but bloat is also having sudden death. and
- Postmodern findings of any Actinobacillosis or pillometa and Carcinomas are there in the rumen or reticulomasal orifice
- another more important condition is Swellers, it's a moderately bloating animal when it is making when you are allowing the animal to walk after giving some surface tension reducing agent, it will resolve its bloat automatically, that is called as Swellers.

Treatment: treatment in emergency cases,

- you have to go for Rumenotomy or
- else you can pass the stomach tube to relieve the gas,
- in the emergency situation, you can also go for Trocharization but Trocharization is also having some disadvantage of developing peritonitis,
- we can tie a Bit like structure over the mouth and you can make the animal just have more salivation to be secreted by just licking the Bit and that will be a buffering agent for the foams to collage together.
- then you have to drench the sodium bicarbonate (NaHCO3) with 150 to 200 grams of sodium bicarbonate mixed in the water you can give and mineral oils or vegetable oils you can give for cattle and sheep and
- emulsified detergents like dioctyl sodium sulfosuccinate
- synthetic surfactants
- another more important point that the material that is available in the market is Silicone dimethicone as a trading name of Bloatosil or Bloatonil and so many other trade names are there that is reducing the surface tension, by the way, the small gas bubbles collage together to form this bigger gas bubbles that will touch the cardia, the eructation will be started, the animal will be recovering and
- in case of any choke, you have to remove if the cervical oesophagus is having any material, in this picture you are seeing the coconut kernel, which is to be taken through the oral cavity by applying the mouth gag manually. one person to push the content towards the fairings and another one for the person to enter through the oral cavity to get it out so by the way you can reduce the chance of a free gas blow choke can be relieved, and
- you can also diagnose based on the Endoscopy, the endoscopy will identify and another thing is the choke where it is before retrieving you just pass the stomach tube and just locate the area if it is in the cervical oesophagus, you can take it by oral. if it is entered into the thoracic oesophagus, you have to go for

Rumenotomy and you have to take it out.

The **Control**:

Pasture bloats, what you have to do is you have to spill the or spray the oils on the pastures and you can also go for some sustained-release monensin, administration to the animals. then Feedlot bloat, we have to give crushed or whole grain to the animals rather than going for the powdering the concentrate. Generally, the strip gazing is to be advised, to avoid the frothy bloat and leguminous fodder always to be combined with the other fodders to avoid frothy bloat development.

In this class four, we have discussed Etiopathogenesis, Clinical signs, Diagnosis, Differential diagnosis, and Treatment of the pasture bloat, feedlot bloat, and secondary Ruminal Tympany.

In the next class lesson five, we are going to discuss the Vagus indigestion and Omasal impaction.

Thank you!

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PDF: Ruminal Tympany

5 Vagus Indigestion and Omasal Impaction

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Transcript

Dear vets,

Greetings to all!

In lesson four, we have discussed the Ruminal tympany, in that we have discussed Etiopathogenesis, Clinical signs, Diagnosis, Differential diagnosis, Treatment, and Management of the frothy and free gas bloat. In lesson five, we are going to discuss two diseases one is Vagus indigestion and Omasal impaction.

Let us first discuss **Vagus indigestion**; what do you mean by Vagus indigestion, I think you know that the vagal nerve is the 10th cranial nerve, supplying the stomach and all other internal organs in the abdomen. Suppose if it is affected due to some other reason, some of the conditions, automatically it will lead to some vagal nerve problem associated with the digestive disturbance. So, it is otherwise called Hoflund syndrome, the person who has identified the syndrome in his name is called Hoflund syndrome. Here are two areas to be covered:

1. Proximal functional stenosis and
2. Distal functional stenosis

Or

1. Anterior functional stenosis and
2. Posterior functional stenosis

Etiology: mainly the Dorsal vagal nerve or Ventral vagal nerve damage, that is due to some inflammation or scar. so what will happen Dorsal nerve damage, there is an achalasia reticulo omasal orifice, that is achalasia means, there is an opening but the content is not moving. In the case of Ventral vagal nerve affection, there is stenosis or achalasia of the pyloric part of the abomasum it is going to be there. Apart from that, there will be reticular adhesions, that will compress the vagal nerve or any other conditions associated with the nerve root or nerve track, compression-like Actinobacillosis, in sheep-Cysticercus tenuicollis, Fibropapilloma and Abomasal impaction, Omasal impaction, then Indigestion in the late pregnancy. Suppose the rumen reticulum omasum is fully distended during the late pregnancy and the pregnancy also has a gravid uterus, that is compressing the vagal nerve will lead to this type of Vagal indigestion.

Pathogenesis:

here Anterior functional stenosis- there is an achalasia reticulo omasal orifice, that will lead to failure of Omasal transport because the omasum is not allowing the content to move to the abomasum. So here the Paralysis of the forestomach and abomasal walls will be there and the accumulation of content in the rumen is making the left side fully distended, so when you are doing the rectal examination you feel the L-shaped rumen because of the Dorsal sac and the Ventral sac both are distended with the content, and it will lead to hyper-motility of the rumen. Here another one more thing is decreased reticular motility because of the distended rumen and vagal nerve damage, there is a decreased reticular motility. The decreased reticular motility is having significance in the development of Anterior functional stenosis. Here what will happen Achalasia and Atony of the rumen and here the Particulate matter,

will be the fibrous material and the liquid portion of the rumen liquor, separation is very difficult he is going to be impaired, so that will be affecting the outflow of the content to the Omasum. Sometimes there may be a coarser fiber present in the feces like 0.5 centimeters and more length diameter is going to be present in the dung material, which is an indication of the reticular adhesions. And the animal may develop vagal tone mediated Bradycardia and in the case of Anterior functional stenosis, there won't be any metabolic alkalosis and dehydration.

In the case of Posterior functional stenosis- there is a Pyloric obstruction; the abomasal pyloric end is having Achalasia, so the failure of the outflow of the content to the pylorus region. Here the abomasal fluid keeps on accumulating and sometimes it may be regurgitated which is called internal vomiting, regurgitated into the rumen, so the rumen chloride content will be elevated because available hydrochloric acid that will be regurgitated into the rumen, is going to be elevated. so that is one of the significant findings when you are going for a rumen fluid examination. So, in the case of posterior functional stenosis due to pyloric obstruction or pyloric stenosis, it is leading to severe dehydration, metabolic alkalosis, hypochloremia, hypokalemia. So, the animal may develop a flask Paralysis.

What are all the Clinical signs you can expect in vagal indigestion,

- the animal may develop a papple shaped abdomen-papple shaped abdomen means apple on the left side, pear on the right side,
- it will have inappetence to anorexia, the vagal indigestion animal will be having slow progression, it will have development slowly, over a period of one week it will be developing so it may be due to either it is initially the indigestion problem or bloat or it will look like a foreign body syndrome but slowly it's not going to respond to your treatment,
- it will be progressing to the adverse level, so it is going to pass

the scanty pasty feces and the animal will be emaciated and that abdominal distension will be more progressive,

- and initially, there will be a hypermotility followed by hypomotility of the rumen, so the rumen distention with hypermotility is going to lead to L-shaped rumen and Bradycardia and the rumen contents will be macerated and frothy in nature,
- then rumen discharge with atony, the content will be very soft porridge-like, atony of the rumen, persistent foams will be there, scanty feces is also there, and
- Pyloric obstruction is seen in late pregnancy.

Diagnosis: mainly based on the History, Clinical signs, and Exploratory, Laparotomy, and Rumenotomy. Another one more test is very useful in the field condition Atropine test, so to identify whether the Bradycardia is going to be modified or not, so you have to administer the Atropine 40 mg at 1% level subcutaneously to the animal, we have to examine the heart rate for every 5 minutes once the 15 minutes after the administration of the atropine sulfate the minimum of 15.8% increase in the heart rate is indicating that positive for vagal indigestion.

Differential diagnosis: it should be

- Traumatic reticular paternities-TRP
- Abomasal impaction
- Phytobezoar
- Foreign body syndrome
- Abomasal ulcerations
- Omasal impaction and
- the distension of the abdomen due to late pregnancy also be addressed.

Treatment :

- The prognosis of this vagal indigestion case is unfavorable, so

slaughter for salvage is one of the ultimate aims. But some of the cases can be tried with Rumenotomy, Rumen lavage you can do,

- Fluid and electrolyte therapy is to be given for 3-5 days,
- Mineral oils– we can have 5-10 liters of mineral oils can administer every day for 3 days,
- then if the late pregnant animals, you have to go for induction or parturition that will help to elevate the problem.

The next disease is **Impaction of Omasum** :

- It is very rare in cattle and most of the time, it is not diagnosed that is undiagnosed because the omasum is not accessed by your palpation, percussion, auscultation.
- It will be there in the right midline in the central third of the abdomen,
- It is spherical in shape, and it is having a leaf-like structure inside that, it is filtering the content which is coming from the rumen,
- It is also having so many other functions like volatile fatty acid (VFA) absorption, electrolyte absorption, water absorption so that is the main function,
- then is Impaction of the omasum is secondarily involved with other diseases like any other advanced stage of pregnancy or any obstruction in the pyloric.

The **Clinical signs** are:

Because it is very difficult to palpate percussion auscultate, that's why based on the clinical signs we can conclude that it may be due to because of Omasal impaction, only an autopsy will confirm the disease. The clinical signs are:

- Anorexia,
- Decreased ruminal movements,
- Cessation with defecation

- Empty rectum- the animal will be having empty rectum and not void dung
- Subacute abdominal pain will be there,
- The sudden drop in milk yield and
- Pain can elicit on the 7th – 9th intercostal space on the right side right thorax.

Diagnosis: it is only done at Autopsy, the Omasal impaction is difficult to diagnose on the anti[1]bottom clinical examination. It will be having an enlarged, excessively hard, dry powdery appearance in the postmark so which indicates Omasal impaction.

Treatment: there is no specific treatment you have to go for

- Rumenotomy- through the Rumenotomy that event, you can introduce a tube you can infuse some oils, mineral oils to the omasum and you can also knead just massage the omasum through the medial wall of the rumen and we'll able to just to make the omasal impaction to be resolved.

So far we have discussed this in five lessons. It has covered

1. Rumen dysfunction and Omasal impaction in cattle, Simple Indigestion, Ruminal impaction,
2. Ruminal lactic acidosis,
3. Subacute Ruminal acidosis, Ruminal drinkers, and Ruminal alkalosis
4. Ruminal tympany and
5. Vagus Indigestion and Omasal Impaction.

I hope that these 5 lessons would have created some diagnostic capabilities and treatment modalities in your liberal practice.

Thank you!

Download

PDF: Vagus Indigestion and Omasal Impaction

PART V

WEEK 5: TRAUMATIC RETICULO - PERITONITIS AND PERICARDITIS

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Transcript

Hi! Greetings everyone,

This is Dr. Ranjit Kumar, so I am going to present the Traumatic Reticulo-peritonitis and Pericarditis in five modules.

In module 1 we are going to see.

- How the animal develops Traumatic Reticulo-peritonitis,
- What are all the predisposing factors,
- What is the epidemiology,
- What are all the materials which are penetrating animals?

In module 2 we are going to discuss;

- Various Pathologies are involved in Traumatic Reticulo-peritonitis and Pericarditis.

In module 3 we are going to discuss;

- Different clinical pictures, of what the animal is exhibiting at the field level, so this is what we are going to discuss.

In module 4 we are going to discuss the

- What are all the different ways to diagnose this disease, and

In module 5

- How to approach these cases,
- How to approach medical management followed by surgical corrections,

This is what we are going to discuss in this presentation. So, let's begin with a presentation, so we will go through directly module 1, Thank you.

In this module we are going to discuss;

- What is Traumatic Reticulo-peritonitis,
- Why the cattle are getting more Traumatic Reticulo-peritonitis and Pericarditis,
- What are all the reasons,
- Where the foreign material is penetrating,
- What is the Epidemiology, and
- What are all the materials which are penetrating animals?

This is what we are going to present in this module, so here if you see this Traumatic Reticulo[1]peritonitis is because by the penetrating metallic foreign bodies either by the Peritoneum or by the Pericardium, so generally this is more common in cattle and buffaloes when compared to other ruminants, particularly the small ruminants like ship and goat. Why cattle and buffalo are getting more Traumatic Reticulo-peritonitis and Pericarditis, so there are four reasons:

1. Number one is so generally the cattle and buffaloes, they never use their lips to catch the materials and to differentiate the materials which are animate and inanimate, so this is number one,
2. Number two they use their tongue by out-swept curling method, so generally, they use for out-swept curling method

this is to grasp the grasses and swallow them, so this is number two

3. Number three there is limited mastication in cattle and buffaloes, so generally, cattle and buffaloes used to prefer remastication, particularly during the rest period, when in the grazing time so there is limited mastication is there that is why they are unable to differentiate the metallic foreign bodies which are present in the ingesta,
4. Number four is the Honeycomb anatomical structure of the reticulum, so once the foreign material is entered into the reticulum, they just logged in the honeycomb structure of the reticulum so this is the four reasons why cattle and buffalo are getting Traumatic Reticulo-peritonitis and Pericarditis.

(Showing video in the slide)So this is the grazing pattern of the cattle if you see this video you can very easily see that the animal uses its tongue to out-swept curling method, so you can very easily see this topographical anatomy of the cattle if once the ingesta is entering to the cattle and directly enter into the reticulum, so this is the honeycomb structure of the reticulum anatomy of the reticulum.

So, as I told the metals they generally gravitate, directly enter into the reticulum which is from the oral cavity, they directly enter into the reticulum and hold by the reticular folds, particularly the honeycomb structure of the reticulum.

The other reason, why the cattle are predisposed is because of the pregnancy, generally, the pregnancy pushes, because of the enlarging fetus so they generally push the rumino reticulum into the cranial which is another reason why the cattle and buffalo are prone to Traumatic Reticulo-peritonitis and Pericarditis.

The other reason is Pica, particularly this is due to the deficiency of various minerals, proteins, and all those various reasons. So again, Pica is one of the predisposing factors for the animals to get Traumatic Reticulo-peritonitis and Pericarditis. Where the foreign

material is penetrating there are three places the foreign material is penetrating generally so:

1. Anterior-ventral region/wall,
2. 2. The ventral region, and
3. 3. Anterior region

So, these are the three places where the foreign material penetrates most commonly in animals. So once the foreign material enters the reticulum, so what will happen is generally if the foreign material lies quietly it won't disturb at all and for over some time particularly six weeks to one year period, they generally corroded, and it used to get away, so this is when the foreign material when it lies freely. If the foreign material is attached to the wall of the reticulum, it generally produces inflammatory reasons and necrosis develops within 72 hours, so this creates inflammation and other consequences. Immediately there will be a development of acute peritonitis and other consequences so on.

And if the penetration is not there or if the penetration is coming back, then again, the animal will get recovered from that and the retrieved foreign body they generally corroded, and it easily gets away.

Then **Epidemiology** of this disease; see generally this disease is more common in developing countries, so where the industrialization is more, where this area is more vulnerable to Traumatic Reticulo-peritonitis and Pericarditis. So, 90% of cases, they generally occur in the dry season, and in most cases, they generally occur in the female animals for one year of age. And the majority of the cases if you see these cases last term of pregnancy or within one month after the delivery or calving. So generally, it is estimated in India the prevalence rate is 14% in forestomach disorders, so this is the only study available in India how what is the incidence of the Traumatic Reticulo-peritonitis, so there is 14% in forestomach disorders, not overall cases, the incidence is recorded. And the complication is a reticular abscess, the reticular

abscess is more common in buffalos rather than in cattle, so where the foreign material pair travels, where it penetrates, and what are all the organs affected, I have depicted here in this slide. But if you see this generally the foreign body is used to enter, it used to move cranially and it used to penetrate the Pericardium and once it penetrates the Pericardium, it creates Pericarditis. If it travels Lateral and Ventral, it penetrates the Peritoneum and is used to produce Peritonitis. If it travels Medial and Dorsal, then the liver is located it is used to produce a liver abscess. If it travels Laterally and Dorsally, their Spleen is located and here splenic abscess it used to produce. Sometimes the foreign body is used to move in an aberrant way and if it enters the lungs, lung parenchyma, is used to produce Pleuropneumonia or else sometimes it used to be very rarely it used to rupture the major arteries and veins. For example, if you take it sometimes it may rupture the Coronary artery or it may rupture the Reticular vein. So, in both the conditions, it used to produce acute death in animals.

(Showing pictures in the slide) So if you see this picture, this is the liver abscess and this is a splenic abscess produced by the reticular foreign bodies. This animal is affected by Traumatic Reticulo-peritonitis with that the animal also has severe Pleuropneumonia. So, there is severe Peritonitis along with the Pleuropneumonia, so that is why there is open mouth breathing and other respiratory distress in this animal.

So, what are all the materials used to penetrate the animal, so generally it starts from the needle, hairpin, any sharp material which lies in the ground, or which lies which is grasped by the animal, that directly enters into the reticulum and which is used to penetrate the animal and it produces Traumatic Reticular-peritonitis and Pericarditis. These materials generally penetrate the animal's reticulum and they produce Traumatic Reticulo-peritonitis.

(Showing pictures in the slide) So these are the materials recovered from Rumenotomy and these materials, generally penetrate the reticular wall you see these materials which are recovered from Rumenotomy in an animal.

So, in this class, we have seen about

What is the reason for Traumatic Reticulo-peritonitis in cattle and buffalo,

What is the Epidemiology, and

What are all the materials used to penetrate the animal?

Thank you!

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PDF: Introduction to Traumatic Reticulo Peritonitis and Pericarditis

2 What Happens When Foreign Body Penetrates

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Transcript

Hi! Welcome everyone,

So, the last module we have seen about

- What is Traumatic Reticulo-peritonitis,
- Why there is the incidence of Traumatic Reticulo-peritonitis more in cattle and buffalo,
- What is the Epidemiology,
- What are all the materials which are penetrating the cattle and buffalo?

so, what is the Pathophysiology of Traumatic Reticulo-peritonitis and Pericarditis? So, we will see in this module, here once the foreign material, I told generally it used to travel Anterior ventral part and Anterior or Ventral part. So, this is the most common place where the foreign material is used to travel, so if it is traveled in an aberrant manner it is used to produce various types of diseases, particularly starting from a splenic abscess to a liver abscess to Pleuropneumonia and even it may cause a sudden death also.

So, if you leave all those things, what is happening, if it travels

into the Anterior-ventral region particularly when it penetrates the peritoneum and pericardium what happens in these animals, this is what we are going to see in this module. See once the foreign material is used to penetrate so here immediately produces an inflammatory reaction, this inflammatory reaction that it happens within 24 hours. So immediately there will be an establishment of Foci be created. so, this inflammatory Foci are used to reduce the Rumino[1]reticular motility and if the Rumino-reticular motility is gets reduced naturally, it is used to produce bloat and all other sequences.

So, before that, if the foreign material gets lodged in the Oesophagus, so there will be vomit. So, there is the development of acute peritonitis, particularly in acute cases where the penetration occurs just immediately, so this acute peritonitis occurs within 24 hours, particularly if the penetration stands a long time or if the material is so strong or if it is lengthier if it is if the material is like having large diameter this used to produce chronic peritonitis.

Then because of the penetration, there are **Four Outcomes** in animals:

1. Acute peritonitis,
2. Chronic local peritonitis,
3. Diffuse peritonitis, and
4. Sudden death.

So, we will see them one by one:

(Showing pictures in the slide) If you see this picture; when there is the penetration of the foreign material, see generally the foreign material lies in the reticulum, I told the foreign material is generally used to travel either anteriorly or in the Ventral regions, so if it travels in the Ventral region or the Anterior region, then there is the development of inflammatory foci. So, this inflammatory foci, develop here and create Acute local peritonitis. And if the inflammatory foci are so large inflammatory focus, gets disturbed sometimes or if the infection seepage is there everywhere,

particularly in the peritoneum, then it creates Diffuse peritonitis. So, the entire peritoneum gets inflamed here, if you see this picture in this module there will be a development of entire Diffuse peritonitis.

And in Acute local peritonitis, so generally this occurs within 24 hours and there is the development of ruminal atony and abdominal pain will be there and if you see here there will be a development of adhesions, generally, it used to produce adhesions immediately whatever the material penetrates inflammatory foci develop and this inflammatory foci it used to create adhesions so once adhesions are developed, sometimes the foreign material is used to move back or the once the adhesions develop so this inflammation is segregated there itself and sometimes or slowly it will be corroded away. so, if it knots sometimes if the foreign material comes back so the animal generally recovers. So here once the recovery is there or the foreign material comes back, the motility gets restored, adjacent they generally disappear within six months of the period. So sometimes there is the development of reticular abscess here, so that is the complication in Acute peritonitis.

Then Chronic local peritonitis: as I told when the foreign material is if it penetrates or persists over a period then there is the development of Chronic local peritonitis. So chronic local peritonitis generally there will be an adhesion formation, this adhesion formation gets broken when the animal moves or when the animal plays, or when the animal rubs, jump when these adhesions break and there is reduced Reticulo-rumen motility. So, because of the adhesion formation the intestinal motility and there will be a reduction in the Reticulo-rumen motility. See I told you there is the adhesion formation, and this persists over some time from this, or the bacterial colonies are entering the systemic circulation and they dispersed the entire body. So, when they dispersed it to the entire body used to reach the different localities of the heart is used to produce Endocarditis, when it reaches joints, it is used to produce arthritis, and when it reaches the kidney it is

used to produce nephritis, and sometimes it used to produce lung abscess also.

And Diffuse peritonitis; this Diffuse peritonitis is generally because by the accumulation of fibrous material or purulent material because of the long-standing inflammation or breakage of the fibrinous adhesions and there is severe mental depression occurs in this diffuse peritonitis. See here there is the development of three things;

1. Toxemia,
2. Alimentary tract stasis,
3. Severe dehydration and Shock

And sudden death; so, if you see here what all the reasons for Sudden death are: So

- Perforation of the coronary artery, so when there is perforation of the coronary artery occurs there is the development of Cardiac tamponade and
- Cardiac tamponade is used to produce compliance failure in the heart and because of the compliance failure the animal dies immediately this is number one,
- There is a rupture of the left gastroepiploic artery or else reticular vein, so when these two both reticular veins are left gastroepiploic artery gets ruptured, there is profuse bleeding because of the profuse bleeding the animal dies.

Cardiac System:

There is simple penetration doesn't mean that the animal will get Pericarditis it never happens, so generally when the foreign body travels it used to take some of the bacterial colonies also or bacterial foci also. These foci enter into the Pericardium and then are used to produce Pericarditis, majority of the time there will be a development of Toxemia and there is an accumulation of fluid.

So, there are three types of Pericarditis develops in animals:

1. Fibrinous pericarditis
2. Effusive pericarditis
3. Constrictive pericarditis

So, because of the development of fibrinous pericarditis, there is constriction used to happen in and around the heart. So, this is Constrictive pericarditis.

What happens in Effusive pericarditis: In Effusive pericarditis, there is a large accumulation of fluid particularly protein-rich fluid is getting accumulated in the pericardium, so Effusive pericarditis produces compliance failure in animals.

Development of congestive heart failure is more common, so this is number one, Number two when the animal develops fibrinous pericarditis, the fibrinous material gets accumulated in the opposite that is the posterior part where the foreign body penetrates in the posterior part the fibrinous material gets accumulated in the pericardium and there is a development of fibrinous adhesions with the myocardium and pericardium. So, this inhibits the Myocardial contractions this is number two.

Number three because of the fibrinous development, there is constriction used to develop this. There is constriction over the Myocardium and is used the fibrinous material used to contract the Myocardium this is used to produce constrictive failure, constrictive pericarditis, and finally, there is a development of cardiac tamponade and death.

So, this is the Differential diagnosis for Traumatic Reticulo-peritonitis and Pericarditis

- Abomasal impaction and Volvulus, there is impacted Abomasal material, and you can very easily diagnose it by using the Ultrasound or some other method or by using your hand by Rectal examination, Impacted abomasum can be palpated, and it can be diagnosed
- Perforated Abomasal ulcers, these are grade four Abomasal ulcers, so if you see there will be severe abdominal pain.

number one so here because of the perforation there is the development of Diffuse peritonitis. So here you need to differentiate this Diffuse peritonitis is due to an abomasal ulcer or because of Traumatic Reticulo-peritonitis. so, this mimics Traumatic Reticulo-peritonitis many times, so because this is a grade four Abomasal ulcer, generally it used to produce severe Abomasum, severe abdominal pain and tucked up abdomen all those things and there will be black terry feces which is more common in abomasal ulcers and

- Acute pleuritis, so you know that this is generally because of the penetration of the foreign body in the lung parenchyma. so if you leave these things primary pleuritis they mimic has a Traumatic Reticulo-peritonitis and
- Lymphosarcoma: Lymphosarcoma again there is the development of lymphadenopathy in lymphosarcomas, but generally that won't happen in Traumatic Reticulo-peritonitis.

In this module we have seen there are four things that happen when there is a foreign body penetrates the peritoneum, so there is a development of Acute peritonitis, Chronic local peritonitis, and Diffuse peritonitis, Sudden death. so, this is what happens when the foreign body penetrates in and around the peritoneum.

When there is development when the foreign body penetrates the pericardium, so there is a development of three types of pericarditis:

- Effusive pericarditis
- Fibrinous pericarditis
- Constitutive pericarditis

This is what happens in the Pericardium when the foreign body penetrates the pericardium.

Thank you!

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PDF: What happens when Foreign Body Penetrates

3 What are the Signs?

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Transcript

Hi everyone!

Welcome back to module 3. So, in module 2 last module, we have seen about the different types of Peritonitis and different types of Pericarditis.

There are four types of Peritonitis and three types of Pericarditis. So that what is happen when the foreign body penetrates the peritoneum and pericardium, as we have seen in the last module.

So, this module we will see about the Clinical picture; so what are all the Clinical pictures depicted by the animals.

So generally, if you see whenever the foreign material penetrates, I told in the last module that there is a development of Acute peritonitis within 24 hours and that generally persists for about 72 hours. So that I told in the last module.

When there is the development of Acute local peritonitis, what are all the Clinical pictures, the animal exhibits we will see in this module. So generally, if you speak Traumatic Reticulo[1]peritonitis, the Clinical picture is vague in nature. It is a non-specific, generally non-specific Clinical picture produced by the animals and it depends upon the size shape of where the foreign material travels and how long it is there, so all these things determine the Clinical

picture. It is not only by the Peritoneum inflammatory reaction, it is also decided by the what organ is penetrated, how long it is penetrated, all these things produce the severity of the Clinic picture.

Then, when you give attention to an animal, so when you suspect an animal is having a Traumatic Reticulo-petronitis and Pericarditis.

Two things:

1. When the animal is having grunting,
2. When there is bruxism, the animal may have Traumatic Reticulo-peritonitis and Pericarditis. This you have to think of immediately.

So the majority of the time, there will be cranial abdominal pain, when the animal exhibits cranial abdominal pain then you have to think it of Traumatic Reticulo-peritonitis and Pericarditis.

(Showing video in the slide) I hope you have listened to the grunting sound from the animal each and every expiration this animal produces, there is a grunting sound. During expiration, there is a sudden opening of the epiglottis and generally is used to produce the grunting sound. So again, if you see this video, this animal is having Traumatic Reticulo-peritonitis and brisket edema and it used to produce a grunting sound.

So now we will see when the animal is having Acute peritonitis what is the Clinical picture as I told the Acute peritonitis, develops within 24 hours. So generally, there will be a sharp decline in the milk yield. particularly the milk yield goes below 50%, this is number one.

Number two, there will be the development of Pyrexia and there is the development of abdominal pain, so because of the Subacute abdominal pain, the animal there will be a 'tucked[1]up' abdomen, so the animal is used to seeing there is if the animal having pain, it pulls the abdomen back so there will be a 'tucked-up' abdomen will be there.

Then there will be a reduced or absent rumination is one of the indications of Acute peritonitis.

(Showing pictures in the slide) If you see this picture there is arching of the back and tucked-up abdomen.

Then there will be firm and pelleted dung in when the animal develops Acute peritonitis, so if you see this material, see here if you see there are pellet feces. These pellet feces indicates that there is some ruminal stasis and there is some abdominal pain. These pellet feces indicate one thing dehydration and the second thing because of the long stasis in the GI tract. (Showing pictures in the slide) And if you see this animal is kicking the abdomen because of the cranial abdominal pain, not only abdominal pain, when the animal is having Acute peritonitis generally these animals, they will have ruminal tympany, mild ruminal tympany will be there, so as I told before there will be in grunting and reluctant to move and so these animals, they remain standing for a long period. So once the foreign material moves back or the Acute peritonitis subsides these animals recover within 3-5 days.

The next one is **Chronic local peritonitis**: In Chronic local peritonitis, there is a loss of body condition and when the animal moves, there will be a slow and careful gait. So, this is again suggestive of Chronic local peritonitis and there will be an arched back, so when the animal is having pain in the abdomen, naturally the animal exhibits the arched back. There will be a tense abdomen so when I told you naturally the animal has pain, an arched back, tensed abdomen.

See another things are **Chronic moderate bloat**; so moderate bloat will be there and here one more important criterion is there will be an increased undigested material in the feces, so when there is increased undigested material always you have to think of the animal is having Chronic local peritonitis and of course, the animal will have grunting and naturally there will be a reticular abscess. If the reticular abscess is there, the animal there will be in poor body condition, the entire lifespan will be poor, or else we can say in the other words the quality of the life always goes down.

And if you see the feces will be firm and it will be coated with mucus, so this is another typical example so one thing undigested material increase in the undigested material and the second one is mucus coated firm mucous coated dung. So, this is again indicative of Chronic local peritonitis.

(Showing video in the slide) So this is one of the videos that indicate that the animal is having Reticular abscess.

Diffuse peritonitis: In Diffuse peritonitis in spite of Anorexia, there will be an increase in the abdomen. So that is the typical indication that the animal is having Diffuse peritonitis. As I told there will be an absent or limited rumen movement, so because of the limited rumen movement and one thing the second one is there is Diffusion in the peritoneal fluid is accumulated with a lot of fluids because of that there is severe dehydration and this severe dehydration that leads to Scanty feces. Once scanty feces are there then you can suspect the animal is having Diffuse peritonitis this is one thing.

Another one is because of these Scanty feces and dehydration, and it is prone to Hypovolemia. Once Hypovolemia is there then the heart rate gets increased, generally the heart rate will be more than 120 beats per minute.

If you do a rectal examination, there will be pain in the visceral organs, so severe pain will be avenged by the animal. and

As I told in the last module itself, there will be a marked mental depression because of the fluid secretion in the peritoneum severe Dehydration, Hypovolemia, there is the development of Toxemia and finally, there is Shock, and the animal goes for recumbency and Death in these animals.

(Showing pictures in the slide) If you see this animal, they see the rectal examination there is scanty feces and these scanty feces indicates black tarry scanty feces. So, this always indicates that the animal is having Toxemia because of Diffuse peritonitis.

(Showing pictures in the slide) If you see this picture, when you do a rectal examination, there is finely powdered material the physical

material which is strictly adhered to the gloves, so it is very difficult to remove this fecal material, so thick sludge-like, tenacious dung.

(Showing pictures in the slide) It is which is difficult to remove from the gloves, so this is again indicative of Diffuse peritonitis adhered to the gloves because of the long stasis it is adhered to the gloves, again it is very difficult to remove. so again, this indicates Paralytic ileus because of the Diffuse peritonitis.

So that is what happens when the animal is having Acute peritonitis, Chronic local peritonitis, and Diffuse peritonitis.

When the animal develops Pericarditis what will happen, when the animal develops Pericarditis there is an increase in the heart rate, so generally the increase in the heart rate will be more than 130 beats per minute. So definitely the animal will have more than 100 and it may reach 130 and above also. So, this is one of the indications that an increase in the heart rate that is Tachycardia, particularly above 100 indicates that the animal is having Pericarditis this is number one. Number two, there is the development of Congestive signs, where the Congestive signs develop in the loose space, where the loose space is there? in the intermandibular space, in the brisket region, and in the ventral region.

So, this is where the Oedema develops because of the congestive heart failure.

Number three is there is the development of Positive venous pulse, and there is bilateral engorgement of the jugular veins, so this is number three.

Number four will be, there is mild diarrhea or scanty feces will be there, so this mild diarrhea or scanty faces indicates that there is congestion in the intestinal tract because of the congestion, the nutrients won't be absorbed properly this is number one and number two there will be a mild effusion in the intestinal ingester because of that there is the development of diarrhea and scanty faces. Then number four the Cardiac sign is because of Congestive heart failure, there will be the development of Oedema, particularly in the Conjunctival mucous membrane that looks like grapes, and

that much Oedema is used to develop in the Conjunctival mucous membrane.

(Showing pictures in the slide) If you see this picture, you just see this there is engorgement of Jugular vein and there is the development of brisket Oedema here and you see this brisket Oedema and abducted limbs. So, this is again indicative of Pericarditis. (Showing pictures in the slide) If you see this picture just see this there is a huge development of Oedema in the brisket region, inter mandibular region, and the ventral region. So don't think that the animal is having udder edema, so here the udder is absolutely normal. So, the edema in the ventral region, brisket region, and in the inter mandibular region, all these things because of the congestive signs because of the Pericarditis.

In this module, we have seen what all the Clinical pictures are.

Number one and the majority of the time you will get the vague clinical picture, so when there is grunting, and bruxism, definitely you have to suspect Traumatic Reticulo-peritonitis and Pericarditis. When the foreign body penetrates the peritoneum so different types of peritonitis develop. What are all the Clinical pictures in Acute peritonitis, Chronic local peritonitis, and Diffuse peritonitis we have seen in this module?

And what are all the Clinical pictures that when the foreign body penetrates the Pericardium and there is the development of Congestive failure, congestive heart signs?

So that we have seen in this module.

Thank you!

Download

PDF: What are the signs

4 How to Diagnose it?

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Transcript

In the last module, we have seen what all the Clinical pictures of different types of Peritonitis are and what are all the different types of Pericarditis. What is all the clinical pictures animal develops, all those things we have seen in the last module.

In this module How to Diagnose Traumatic Reticulo-peritonitis and Pericarditis, what are all the multimodal approaches various ways we have to diagnose, these cases we will see in this module.

So, as I told in the last module itself, whenever the animal is having Bruxism or Grunting so that animal is suspected of Traumatic Reticulo-peritonitis and Pericarditis, so that is the foremost Clinical sign when any clinician has to suspect the animal is having Traumatic Reticulo-peritonitis and Pericarditis. So apart from that, there are pen site tests that mean which can be done in the field itself. So, the Slope test, Wither pinch test, Pole test, and so on. We will see one by one, what are all those tests:

(Showing video in the slide) This is the **Slope test**: In the slope, test an animal is made to walk on the slope in an up and down direction. When the animal walks in an up and down direction there will be a grunting sound, see when the animal goes down if the intra-abdominal organs push the thorax the presence of foreign

material creates intra-thoracic pain and grunting sound in animals. so generally, in the Slope test when the animal walks downstairs or in the upstairs, there will be a grunting sound. so, this indicates that there is Traumatic Reticulo-peritonitis.

(Showing video in the slide) and if you see this so this animal is examined with a **Pole test**: so, with the wooden pole, we have to push the ventral thorax at the level of the point of the elbow, then we have to push it upwards when there are intra-abdominal foreign bodies the animal reveals the pain.

(Showing video in the slide) and if you see this is **Wither pinch test**: if there is any foreign material, the animal exhibits pain so that is Wither pinch test.

So apart from the Pole test, Slope test, Wither pinch test, there are some more Ancillary tests that can be Diagnosed through nearby laboratories or in the laboratory which is available in your locality.

See here if you see any Traumatic Reticulo-peritonitis and Pericarditis, definitely, there will be an increase in the Leukocyte count, and most probably these animals all majority of the times they have Neutrophilia this is number one.

number two this increase in the Leukocyte count depends upon whether the animal is having Acute local peritonitis or Diffuse local diffuse peritonitis. If the animal is having Acute local peritonitis, there will be a Regenerative left shift.

If the animal is having Diffuse peritonitis, there will be a Degenerative left shift will be there. and in Chronic cases majority of the time, there will be a persistent leukocytosis with neutrophilia is the indication that the animal is having Chronic peritonitis.

Apart from the leukogram, there are some other indications, particularly there will be an increase in the fibrinogen level in the blood, which again indicates that the animal is having Traumatic Reticulo-peritonitis, so if you see the level of the acute-phase proteins, particularly serum amyloid-A (SAA), Haptoglobin (Hp) and all other serum acute-phase proteins levels will be elevated. There are some more cardiac biomarkers, particularly Cardiac troponin-

I and Cardiac troponin-T both elevated in Traumatic Reticulo-peritonitis and Pericarditis cases. These are all the commercial kits available in the market for the estimation of Fibrinogen and Cardiac troponin.

The next one is **Abdominocentesis**: generally, Abdominocentesis is carried out in four places; so, four places so abdominal synthesis once you collect the fluid so it indicates that there will be an increase in the protein levels, particularly the protein in the abdomen peritoneal fluid will be more than 3 gram per deciliter, this is one of the indications that the animal is having Peritonitis.

Then there will be an increase in the nucleated cells will be more than 6000 cells per microliter, if the level is so high that indicates again that the animal is having Peritonitis. so this clearly indicates that there is some ongoing pathology is there in the Peritoneum because of the Peritonitis.

Then sometimes rarely, we may not get adequate fluid in Abdominocentesis, but that doesn't mean that the animal is not having peritonitis. so even failure to get peritoneal fluid also, it won't exclude that the animal is not having peritonitis.

(Showing video in the slide) For the collection of peritonitis, you see this video the peritoneal fluid collection and we can very easily diagnose the peritonitis through the collection of peritoneal fluid. Once you collect the peritoneal fluid if the fibrinogen level is so high and the material is so purulent, then we can be very easily diagnosed by seeing the appearance one thing and another thing you can smell it sometimes there will be foul-smelling.

(Showing pictures in the slide) And once you collect the fluid, it depends upon the quantum of the fibrinogen level, if the fibrinogen level is so high there will be cake formation immediately so so you see this picture, there will be a fibro purulent material, so there will be in cake formation immediately and if you smell it will be foul-smelling. so that means some purulent material is accumulated in the peritoneum.

Then **Pericardiocentesis**; at the level of the third and fourth intercostal space that prior to that you have to auscultate and

identify the maximum intensity of the heart. Once you identify the maximal intensity, then you can locate it and most probably you can identify the fourth and fifth intercostal space. If particularly on the left side, so left fourth and fifth intercostal space is the right place to do the Pericardiocentesis, so say if you insert the needle and if you touch the myocardium if you attach it with ECG, sometimes there will be an Arrhythmia, if you touch the myocardial tissue then there will be an Arrhythmia.

So, if you are not touching the myocardial tissue, then there won't be any Arrhythmia and very easily if there is an Effusive Pericarditis or Fibronous Pericarditis, both the things you can get fluid.

(Showing video in the slide) So you see this video just pull the limbs, forelimbs, cranially as much as possible then locate the maximal intensity of the heart and directly you can identify the place, right place, and you can insert the needle.

Once you insert the needle there will be a pericardial fluid will come out and you can collect it and send it to the laboratory.

And the next one is **Metal detection**: so Metal detectors very easily, we can identify the foreign materials it is a non-invasive technique and very cheap also and the only lacuna with metal detectors are, so we cannot identify, we cannot differentiate the whether the foreign material is penetrating one or nonpenetrating one.

We are using Ferroscopy and this is the ferroscopy most commonly used in animals to identify the foreign materials, particularly for Traumatic Reticulo-pertinotis so and this is the screening of the ferroscopy over the reticular area. Whatever the foreign material is there particularly penetrating foreign bodies, that can be easily identified by the Ferroscopy.

And the next technique is **Radiography**, it is done in the cranial abdomen to identify the penetrating foreign bodies, metallic foreign bodies. And here the metallic foreign bodies which are more than one centimeter in length and that lie around 30 degrees that lie over the floor that is 30 degrees the floor where it can be diagnosed easily by the Radiography. By radiography, we cannot differentiate

the peri-reticular and the hepatic abscesses, that is the only difficulty is the only lack of Radiography.

(Showing pictures in the slide) This is the position of the cassette, where we have to keep the cassettes while taking radiography and this is the metallic foreign body see you can see this arrow-headed point, this point of the arrowhead can be easily revealed in the Radiography. So, the next technique is Ultrasound: by using Ultrasound we can easily diagnose the anechoic areas particularly, the fluid accumulations over the Pericardium, Peritoneum, and Pleural fluids. So, all these things wherever the fluid is there, we can easily be diagnosed by the anechoic fluid accumulation.

Then not only the fluid accumulation so we can identify the gap between the reticulum and the abdominal wall. The other things are so we can easily identify the Perireticular abscess Perireticular adhesions, particularly the reticulo-rumen motility, why the reticulo-rumen motility is decreased, there are effusions particularly because of the Pericardial thoracic or Pleural effusions are there. This can be very easily diagnosed by using Ultrasound.

(Showing video in the slide) If you see this video there is an intestine and outside the intestine, fibrinous material is floating, if you see this video there is fibrinous material along with the pericardial effusion.

And next one is the **Postmortem examination**: In the postmaster examination, we can easily identify the extensive adhesions number one.

Number two multiple abscesses, and see if there is severe Diffuse peritonitis, there will be a foul-smelling material, particularly there will be a huge quantum of Peritoneal fluid in the Peritoneum and this can be easily diagnosed in the Postmortem Diagnosis and sometimes you may get puss between the layers of the pericardial tissues, so that can also be diagnosed by using postmortem.

(Showing pictures in the slide) So this is the picture you can see the Fibro purulent material, which is a deposit, this is because of Traumatic Reticulo-pericarditis.

So, in this module we have seen different modes of how to

Diagnose the Traumatic Reticulo[1]peritonitis and Pericarditis, particularly we have seen the pen site tests and then we have Ancillary tests and then Pericardiocentesis and Abdominocentesis, Radiography and Ultrasonography, and finally the Postmortem examination.

Thank you!

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PDF: How to Diagnose it

5 How to treat these cases?

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Transcript

Hi! Everyone,

In the last module, we have seen the various modes of Diagnosing Traumatic Reticulo[1]peritonitis and Pericarditis.

In this module, we will see How to Treat these cases, that is what we will see in. Before going to this presentation, the first and foremost thing is:

1. Where the Foreign material is located and
2. What are all the facilities, we have
3. Again, the foremost thing is whether the economy is suitable for the therapy or not.

So, these are the three things you have to decide, what are all the facilities, whether the economy is suitable, and where the foreign material is located. So, these are the three things you have to decide and go through it before going through the therapy.

See before initiating any therapeutic approach, we have to go for medical management and before approaching Surgical management. Generally, Medical management is initiated if the medical management is failed, then we can go for Surgical

corrections. Generally, it needs a long-time approach, and particularly long-time antibiotic is required. So, the goals of the therapy are antimicrobial therapy, Anti-inflammatory therapy, Management of fluid and electrolytes, and Pain management, these are the four things we need to keep in our mind. These are the goals of any animal which is suffering from Traumatic Reticulo-peritonitis and Pericarditis.

Generally, Traumatic Reticulo-peritonitis and Pericarditis animals have adhesions, as I told in the previous modules, when these adhesions are there, they generally inhibit the gut motility once the gut motility is inhibited there will be a development of ileus, so this alias needs to be kept in our mind always before treating the animal and this ileus always needs to be treated with prokinetic drugs if warrant in these animals. Because of the toxin's signs, once the adhesion is developing, it is always better to maintain the animal, or it is always better to restrict the movement of the animal to promote the adhesions. see if there is breakage of the adhesions there will be extensive diffuse peritonitis used to develop, so we don't want that because of that we always promote the adhesion formation. Once the adhesion formation is there, particularly in acute conditions, I told in the previous module's adhesions formed within 72 hours, once these additions are formed and the inflammation is isolated, these animals generally recover within 3-5 days. And for these things two things are required:

1. Restricting the movement of the animal, then
2. Elevation of the four-quarter, see always the four-quarter should be elevated like this, that is the four-quarter and hindquarter should be 25 centimeters at least a difference. see the four quarters should be elevated and hindquarters should be below that, so we need to restrict the further movement of the foreign bodies.

Then some of the **Pharmacological options** are:

Generally, Tetracyclines and Beta-lactam antibiotics are used, and

most commonly the steroid drugs and or NSAIDs non-steroidal anti-inflammatory drugs are warranted here. Again, if required you can go for anti-inflammatory drugs, as you can administer these drugs over a period, particularly by using these drugs in CRI infusion, and generally these drugs and anti-inflammatory drugs along with antimicrobial drugs. The minimum is at least required for about a period of five days.

Then **Administration of Magnets**, so magnets need to be administered on day one, when you diagnose the case as having Traumatic Reticulo-peritonitis, immediately you administer the Magnet. So, keep in mind, this magnet length should be more than >3 inches, and if the magnet is situated in an upright position, so it is very ideal for capturing the penetrating foreign bodies... So generally, if the magnet does not retrieve the penetrating foreign bodies, at least it should not allow the penetrating foreign bodies to progress further so that is how it will help. If not retrieved back at least it will prevent the progression of the foreign material penetrating foreign material further.

(Showing pictures in the slide) These are all the magnets that are administered to the reticulum of an animal and if you see this picture, this is after post-mortem they identified these magnets which are having penetrating foreign bodies around them and

The next one is **Fluid therapy**: Generally, I told diffuse peritonitis always has a Hypovolemic shock, so once Hypovolemic shocks are there, these patients require a large quantum of fluids, generally, these patients are vulnerable to Hypochloremia and Hypokalemia, so metabolic alkalosis is suspected in these patients. So, Hypochloremic, Hypokalemic, and metabolic alkalosis are suspected in these patients so that is why the best fluid is Isotonic saline's. Along with that, generally we used to think about whether we can go for oral fluids or not. So here when you suspect an animal is having Traumatic Reticulo-peritonitis or Pericarditis, the oral fluid efficacy is not much, so that is why oral fluid therapy is not indicated particularly when the animal is in diffuse peritonitis, it is not indicated. Along with the normal Isotonic saline, if we want, we

can add Potassium and Magnesium to that fluids and we can go for fluid therapy which is containing Potassium and Magnesium-rich fluids.

Then **Rumenotomy**: so, Rumenotomy, you know it Rumenotomy is generally done at the left flank.

And the Indications for Rumenotomy are Perotonitis, Peri-reticular/rumen adhesions, Peri[1]reticular abscess, and retrieving the foreign materials in the intraluminal space.

And suppose if a peri-reticular abscess is there so we want to lance it again we can go for Rumenotomy.

(Showing pictures in the slide) So this is the Rumenotomy, you know it very well, and see once the material is removed and we have to lance it properly, lavage it, then again, we have to close this surgical site in a proper way. This is the material recovered from Rumenotomy.

Then **Thoracotomy**: two things are done in Thoracotomy

1. Pericardiectomy
2. Pericardiotomy

So, both things can be done in Traumatic Reticulo-peritonitis and Pericarditis cases. So, the main indication is Septic pleuritis, Pyothorax, and Pericarditis, so these three are the main indications where Thoracotomy is indicated.

Generally, the fifth and sixth rib section is indicated for Thoracotomy and once Thoracotomy is done the outcome, particularly for unilateral Pyothoraxand and unilateral Septic pleuritis cases, the outcome will be good when compared to the other cases.

(Showing video in the slide) And if you see this tube attachment in the Thoracocentesis cases and there will be draining of the septic material from the Pericardium.

(Showing video in the slide) So this is the Ultrasonographic examination, after drip, you see this there is the quantum of fluid accumulated in the pericardial space, this is after therapy after

seven days, particularly with the tube Thoracocentesis and generally, when you do the Thoracocentesis it should be done in a slow manner, you should not drain as quickly as possible, that is not indicated and generally the second-intention healing is always advisable, majority of the cases the second-intention healing will take a bit long time.

In a summary, in nutshell, so the penetration of the foreign body occurs mostly in the cranio[1]ventral regions which is one of the main places where the foreign material penetrates. So, the incidence of Traumatic Reticulo-peritonitis and Pericarditis is 14% particularly, in the forestomach disorders in India.

And there are 4 types of Pathogenesis: Acute peritonitis, Chronic local peritonitis, Diffuse peritonitis, and Sudden death, particularly in Traumatic Reticulo-peritonitis cases.

And there are 3 different types of Pericarditis: Effusive pericarditis, Fibrinous pericarditis, Constitutive pericarditis.

And the suspected Clinical signs are Grunting and Bruxism, whenever an animal is having bruxism and grunting you have to suspect the animal may have Traumatic Reticulo-peritonitis and Pericarditis.

So, the Multimodal Diagnostic approach needs to be carried out, it depends upon the availability of the facilities in your locality. Generally, the Prognosis is unrewarding in Traumatic Reticulo-peritonitis and Pericarditis Cases.

Thank you!

Download

PDF: How to treat these cases

PART VI

WEEK 6: INTESTINAL STASIS IN CATTLE

1 Types of Ileus- Anamnesis and Clinical Signs of Ileus

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Transcript

A warm Greetings to all,

This class is on Intestinal Ileus in cattle.

Why Intestinal Ileus has been taken as a separate topic, is to know that it is one of the most challenging cases in bovine medicine and most of the owners become frustrated and become nervous because they want to know whether this case will become all right or they are going to face a severe economic loss because of the death of the animal. In that context, these Intestinal Stasis has been taken as a separate topic and we are going to see why there is economic importance.

If the Intestinal Ileus is not properly approached, the animal can die. There will be a reduction in the production, namely the milk yield. Sometimes the whole animal may die, We are going to see this Intestinal Ileus in 5 chapters:

1. In the first chapter, we are going to see an anamnesis and its clinical sign,
2. In the second lesson, we are going to see the Clinical investigations that are going to perform on the animal,

3. In the third lesson, we are going to see Ultrasonography involving the thoracic diseases,
4. The fourth lesson involves Ultrasonography of abdominal diseases, and
5. In the fifth lesson, we are going to see the Management of Ileus and special emphasis on the pregnancy and pregnancy-associated Ileus.

Now we will go on to lesson one, where we are going to see the importance of Ileus and anamnesis. The Ileus is broadly classified into:

- Mechanical Ileus and
- Functional Ileus.

Mechanical Ileus: it has got a wide variety of causes, it can be luminous or extraluminal causes. Mechanical alias there are many important there are many causes, but the important diseases or syndrome that we need to focus on:

- Traumatic reticulitis or Traumatic Reticulo-peritonitis
- Right-sided displacement (RDA) of abomasum or volvulus
- Diaphragmatic hernia or Vagal indigestion
- Intestinal obstruction
- Mesenteric volvulus
- Caecal dilatation,

these are all on the Intestinal part, there are two more important things as far as the obstacle involvement is concerned;

- Uterine torsion
- Dystocia caused by the fetus

So, you have to rule out all these things and take appropriate measures to identify the Functional Ileus because by exclusion we have to rule out each Etiology then only we can approach the

Functional cause. If you are not ruling out the Mechanical cause and going for treatment of the Functional cause, then there will be a metallic, that is why a Mechanical Ileus is being dealt with when you are going to deal with the functional Ileus.

Of the **Functional Ileus**; there is no exact Etiology so far it has been proved, but it is only hypothesized that these may cause a Functional Ileus. In Functional Ileus, there are no specific clinical signs, except for the reduction in the quantum of the dung produced.

The etiology can be:

- Dietary factors, for example, the owner would have changed the type of fodder or suddenly he would have gone for a change in the concentrate which would have caused Impaction and other things.
- It can be a parasitic infection or
- maybe because of peritonitis or
- it can be pregnancy-associated areas where the pregnant fetus presses onto the intestinal segments and causes Ileus or
- The mere electrolyte abnormality, namely hypercalcemia, a reduction in potassium and phosphorus can also be attributed to the functional Ileus.

Now we are going to see how we are approaching this Intestinal Ileus called the Intestinal Stasis in cattle. Firstly we will be taking up an Anamnesis or how far we are going to stress on the history, then we will go on to Clinical signs, then Clinical investigation, Laboratory analysis, Radiography, and Ultrasonography- how it is going to involve in the thoracic and abdominal cavity.

Anamnesis: In this stress has to be given whether the owner has changed the diet, because the proportion of the fodder and the concentrate plays an important role or whether he has given the excess fodder or irrational fodder, this can go on for the ruminal impaction, then, later on, develop into Intestinal Ileus or whether the importance has to be given to note on the dung, what are the

parameters to be noticed on dung consistency whether it is hard or it is loose, whether it is accompanied by any other things may be mucus or blood or foreign material.

Please note the pregnancy status also, as in the advanced stage this can go for Functional Ileus due to pregnancy.

Then previous medication, whether it has been treated with an antibiotic or any anti[1]inflammatory or has been treated with any other diseases. so because these factors may go from hypermotility or atony of the rumen.

Clinical examination of the animal: invariably in all these cases the change in the dung is most conspicuous, what is that? There is a reduction in the quantity of the dung, maybe atony and the complete absence of the dung or change in the consistency of the dung may be hard, a little pasty like that.

The marked things he noticed here are *Dehydration*, *Rumen atony*, or *hypomotility*. In some cases like volvulus or mesentery, we may get *Colic* signs.

Traumatic reticulitis or Traumatic reticulo-peritonitis: the animal may have Fever, Tachycardia, Arched back when you go for a Slope test, what is a Slope test- make the animal walk on an inclined plane, the animal will be a little bit comfortable when it walks up the hill but when it comes down the hill it has difficulty either it grunts or going for a mild groaning is there.

The next test is Pain Percussion, so if you go for deep pain percussion, the animal elicits pain or chronic. There will be Rumen atony or hypomotility, Rectal examination- inevitably there is scanty dung or absent dung.

Diaphragmatic hernia; here there is recurrent bloat and as a clinician, they are not able to pass the stomach tube, sometimes if they pass the stomach tube, the content is usually frothy and again the bloat occurs.

Next is *Vagal indigestion:* vagal indigestion as you see (Showing pictures in the slide) the abdomen from the end we get a papple-shaped abdomen. what is that left side is papple shape and right

side of the abdomen is pear shape which is a significant as far as the vagal indigestion is concerned.

Displacement of abomasum: it can be Right or Left, usually Right causes a significant change as far as the Intestinal Ileus is concerned. In the Left side displacement of the abomasum, we get a ping sound, either on the left paralumbar fossa or a penultimate intercostal space. What we need to do is start the auscultation and combine the percussion from the left elbow to the paralumbar fossa. So we will be able to get a ping sound, so where we get the ping sound we are supposed to get the Liptack test. Introduce the needle, collect the fluid, and assess the pH, so that is going to confirm the left side displacement of the abomasum.

As far as the Clinical sign again Right side displacement of the abomasum, we are going to get a ping sound on the right side, what is the area, from the elbow to the paralumbar fossa? Do a combined auscultation percussion, we will get a ping sound. During a rectal examination we may get the caudal part as close as your pelvic inlet, the round viscous is palpable on the cranial right side of the abdomen.

Mesenteric Volvulus: here there is a rapid debilitation and progression to a moribund state and the Colic is often severe in this type of mesenteric volvulus. Rectal examination reveals multiple loops of the distended small and large intestine, the normal spatial arrangement of the viscera is usually not present.

Cecal dilatation: here it is on the right side there is abdominal distension and tympanic resonance is noticed on right paralumbar fossa to 1-4 rib spaces cranially. In the rectal examination, we find the dilated round apex of the cecum protruding into the pelvic inlet, and the small intestine of various segments are palpable. Uterine torsion and Dystocia can be easily assessed by vaginal examination and rectal examination and for those clinical signs, we have to see P1 P2 P3.

In **Functional Ileus** no specific clinical signs, except for a reduction in the dung quantity.

(Showing pictures in the slide1) On the left side, we can see the

Rectal examination and the dung that is smeared on the hand which is in the case of a normal animal, and right side we can see the animal where the fodder is of a brown type of hay is being given. The left side is of the green folder, the right side is of the hay or other things.

(Showing pictures in slide2) here we can see on rectal examination, a reduction in the quantity and a slight change in the consistency.

(Showing pictures in the slide3) Rectal examination reveals dung which is having a near absence of dung, what you call an empty rectum, on the right side we are seeing the dung of dark color. We can say this is called a *raspberry jam* appearance.

(Showing pictures in the slide4) The appearance of the dung on the left side is a strawberry jam appearance, on the right side is a *Raspberry jam* appearance.

(Showing pictures in the slide5)This gives an important take-home lesson namely, jam-like appearance, what is it? The left side has a *Strawberry jam-like* appearance. We get frank bleeding as in the case of polyps or tumors which are from the rectum or the colon.

On the right side, we get a raspberry jam appearance which we notice in case of intussusception, so if we get a raspberry jam appearance or dark red colored dung, then we should immediately think of intussusception.

(Showing pictures in the slide6) There is a deviation from the previous slide, now we are going to see a blackberry jam appearance. *Blackberry jam* appearance is nothing but a dark-colored/ black color dung which we encountered in abomasal ulcer as appreciated from the right side slide.

The important Clinical tips to re-emphasize :

- if you get Strawberry jam appearance- it is a frank bleeding
- if you get Raspberry jam appearance it is indicative of intussusception
- if you get a Blackberry jam appearance it is an abomasal ulcer.

In this class, we have seen the various Etiologies that are responsible for Intestinal Ileus or Intestinal Stasis in cattle and we have elaborately seen Anamnesis and Rectal examinations. In the next class, we are going to see the Clinical Investigation we are going to undertake on Intestinal Ileus in cattle.

Thank you!

Download

PDF: Intestinal Stasis in Cattle

2 Clinical Investigation

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Transcript

Welcome back to the Intestinal Ileus in cattle.

In the last lesson, we have seen about Anamnesis and the rectal examination of the Intestinal Ileus in cattle. In this lesson, we are going to see the various **Clinical investigation** that we are focused on on Intestinal Ileus.

Restraining the cattle for doing Thoracocentesis is more important; put the animal in the Travis, keep the particular side, left side, or right side forelimb in a forward stance, how to do that?- tie the forelimb in a forward stance on the Travis, so that the area just began the elbow is free for your investigations. Aesthetically prepare the area by shaving the particular area, and applying surgical spirit.

Thoracocentesis or Pleurocentesis: the site which we are going to use is the 6th or 7th intercostal space and then prepare the area as we have been doing it for surgical conditions and what is the material needed? the material needle is a sixteenth gauge needle or Thoracocentesis needle and a syringe.

(Showing pictures in the slide) this shows the Thoracocentesis being done with the help of a catheter and the fluid is watery. On the

right side, we get zero sanguineous fluid. So this shows the different stages of the disease in these two cattle.

(Showing video in the slide) Thoracocentesis that is being done with the catheter, here we can see the free flow of the fluid, normally we do not get more than 1 or 2 ml at the most. so here you get a free flow of fluid and watery and crystal clear. The most important is the sediment of this fluid and the subject to cytology.

(Showing pictures in the slide) This is another case of Thoracocentesis, which is being done in a buffalo, where we can collect more than 5 liters of fluid, so there is already one can with the 5 liters the another can we are collecting the fluid.

Thoracocentesis or Pleurocentesis following collection of the fluid, we are subject to analysis of the fluid, normal volume is very little but in case of an effusion, we get a lot of fluids. We need to analyze this fluid bacteriologically as well as cytologically, the important point is these fluids do not clot and the nucleated cells are less than 10*109 /liter mostly Neutrophils, mononuclear cells, mesothelial type of cells.

Thoracocentesis do we have any complications; rarely will get complications sometimes there is a collapse of the animal, Pneumothorax or Puncture of Heart/lung may be encountered, but if the proper site is being selected, properly restrain is being followed, no on toward incidence has been encountered.

The procedure is **Pericardiocentesis**; the site has 4th or 5th intercostal space, again the restrain is the same as we have discussed for Thoracocentesis.

(Showing pictures in the slide) describes the Clinical signs of animals with pericardial involvement where there is jowl edema, brisket edema, and positive stasis test. Pericardiocentesis was done with a chest drainage tube, we can get the serous angular fluid, copious amount, and in large volume.

(Showing video in the slide) This is a Pericardiocentesis in other cattle where you get purulent fluid effusion.

(Showing video in the slide) here we have fixed the chest drainage

tube and we have been doing lavage with warm saline and then subsequently irrigation with metronidazole.

Abdominocentesis: Abdominocentesis is otherwise called paracentesis abdominis, this we are doing it to rule out Ascites, Peritonitis, Uroabdomen, and Neoplasia. So let us see what are the site? the site is 8-10 centimeters caudle to the xiphoid and 8-10 centimeters lateral to the midline.

This site aseptically prepares the area following shaving the area and then application of surgical spirit. Introduce the needle very carefully till you get a blup sound and a free flow of fluid is obtained. Normally 0.5 to 1ml is obtained, in the case of peritonitis, it is a free flow of fluid is obtained. So this site which you have described is called a cranial site.

Paracentesis abdominis; this is a caudal site, where we are going to introduce the needle on the right side lower flank in front of the udder. So have you are going to get fluid on the caudal side of the cranial side, it is localized peritonitis. If we get fluid on both sides then it is generally spectronitis.

Paracentesis abdominis; In this as we have seen in Thoracocentesis, subject the fluid for analysis both physically as well as microbiologically and the sediment needs to be analyzed. It is amber-colored and is normal. Cloudy indicates the increased concentration of the protein, Serosanguineous indicates Ischemic necrosis and if it is Turbid fluid then devitalization of the intestine.

Thoraco-reticulocentesis: this is a special procedure we are adopting to diagnose diaphragmatically. What is the material needed? It is a 16 gauge needle, the site is the identification of the peristaltic sound by auscultation in the thoracic area. Once that area is identified and then shaving the area, the surgical spirit is applied, and introduce the needle very slowly and collects the fluid as we do it for the Liptak test. The collected fluid is tested for its pH as well as microbial assessment.

(Showing pictures in the slide) This slide describes the site for Thoraco-reticulocentesis, we are introducing the needle fixed with the 5ml syringe, introduce the needle very slowly and keep

aspirating in the area which has been predetermined by the area of auscultation, where you get peristaltic sound.

Liptak test: the collected fluid is checked for pH, where the pH is alkaline. The alkaline indicates the reticulum as herniate into the thorax and a drop of this fluid is kept under a microscope and examined. You will be able to see the Rumen protozoa again which indicates the reticular rumen is herniated into the thorax.

Clinical Pathology/ Laboratory analysis: usually complete blood count indicates inflammatory changes and hyperglycemia, hypochloremia, and hyperkalemia are noticed in the blood changes.

The summary, now in this lesson, we have seen the Clinical exploration or interpretation in the case of Thoracocentesis, Abdominocentesis, and Thoraco-reticulocentesis we have seen elaborately following the surgical precautions.

In the next class, we are going to see the other investigation namely the Radiography and Ultrasonography.

Thank you!

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PDF: Clinical Investigation

3 Radiography and Ultrasonography

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Transcript

Welcome! back to the lesson on Intestinal Ileus in cattle.

In the previous class, we have seen about Anamnesis, Clinical investigation, and Clinical interpretation on how to proceed with the approach of Intestinal Ileus in cattle. In this class, we are going to see about Radiography and Ultrasonography examination of animals with Intestinal Ileus.

(Showing pictures in the slide1) In the radiograph of a normal healthy animal, where D indicates the Diaphragm, H indicates the Heart, and Re indicates Reticulum. so where we can clearly see the Diaphragmatic border, Heart, and Reticulum.

(Showing pictures in the slide2) This is a radiograph of an animal using Contrast, here also we can see a clear Diaphragmatic border, H indicates Heart, and Re indicates Reticulum, there is a clear distinguish between the thorax as well abdomen.

(Showing pictures in the slide3) This is a radiograph of an animal where we get a *Foreign body*, the arrow indicates the foreign body and it is very clear in the case of a radiograph.

(Showing pictures in the slide4) This is a radiograph of an animal with the *Diaphragmatic Hernia* you can see the Reticulorumen is herniated into the thorax and it is pointed out by a pointer.

Ultrasonography: Now let us see about Ultrasonographic investigation in these animals.

(Showing pictures in the slide1) the first is Pericarditis, so echocardiography the site is 3rd – 6th intercostal spaces, and what you have to do is the cranial limb on the side left side or right side should be proximally placed, apply a liberal quantity of gel or spirit, and place the probe of ultrasound to investigate into the heart.

Pericarditis: the ultrasonic features of pericarditis include pericardial effusion, which can be hypoechogenic or echogenic, and sometimes we will be able to see fibrin sheds within the anechoic fluid. The note points or take-home lesson is there is a thick echogenic membrane surrounding the heart.

(Showing video in the slide) This gives an ultrasonic picture of a heart where LV indicates left ventricle, RV indicates right ventricle, this is ultrasonography in the case of pericarditis where there is an accumulation of echogenic fluid material around the heart.

(Showing video in the slide) this slide shows both the normal animal, which is on the left side, and on the right side we have got an animal with Pericarditis. In a normal animal, you can appreciate the heart without any surroundings. On the right side, as facilitated through the arrow, I have put so echogenic fluid surrounding the heart.

(Showing video in the slide) this slide shows the echocardiography of an animal with Pericarditis. The pericardial diffusion can be anechoic or hyperechoic or there can be extensive Fibrin sheds.

(Showing video in the slide) This slide shows anechoic fluid.

(Showing pictures in the slide) This echocardiographic image shows echogenic fluid.

(Showing pictures in the slide) This slide shows a mild accumulation of Fibrin, while the next slide shows the extensive accumulation of fibrin with its network.

(Showing pictures in the slide) this is a postmortem collected sample of an animal, which has got an ion movement within the heart. This was scanned before and the ultrasonographic pictures show the needle.

(Showing pictures in the slide) The slide of echocardiography shows the line which is echogenic and there is a casting of shadow. We can appreciate the echogenic dot which is caused by this iron foreign body and there is an acoustic shadow- the acoustic shadow is by black color as indicated by a pointer.

(Showing pictures in the slide) **Pleural effusion**: an animal with pleural effusion is scanned and we can get anechoic fluid surrounding the lungs and you can see the movement of the fibrin segments which is hyperechoic.

(Showing pictures in the slide) In this slide, we can see the pericardial fluid as well as the pleural effusion. So pericardial fluid you can see indicated by an arrow and there is a pleural effusion also.

In this lesson, we have appreciated Ultrasonographic investigation in Pericarditis as well as Pleural effusions, where there was echogenic or anechoic, or mixed fluid in this. In the next class, we will see about Ultrasonographic findings on the abdomen.

Thank you!

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4 Ultrasonography of Abdomen Disorder

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Transcript

Warm greetings! to all. In the last lesson, we have seen the Ultrasonic features of the thoracic cavity in those animals affected by Intestinal Ileus.

In this lesson, we will see the Ultrasonography features of the abdominal cavity.

Reticulum: so reticulum is usually on the ventral aspect of the thorax and the left side or right side of the sternum. so follow the area and the left or right side lateral thorax up to the elbow. A normal reticulum is half-moon-shaped, this slide shows the ultrasonic features of the reticulum in healthy cattle. we can see R as the reticulum as viewed by a half-moon shape, and M is the Musculophrenic vein.

(Showing pictures in the slide) This is an echocardiographic picture of an animal with Traumatic reticuloperitoneitis. R is Reticulum, Ru is Rumen, and H is Hyperechoic Adhesions. The features are deposits of fibrinous tissues with fluid packets, and Reticular wall we can see the serosal surface.

Reticular abscess: so Reticular abscess, the echogenic features or echogenic capsule, homogeneous, hypoechoic to moderately echogenic center.

(Showing pictures in the slide) The picture shows A is an Anechoic abscess with the echogenic content and R is Reticulum.

(Showing pictures in the slide) so this shows a case of Reticular abscess in another cattle and R is the Reticulum and outside A is an abscess. Echocardiography features reticular adhesions, where we can see the reticulum with the extensive fibrous adhesion which is echogenic.

Peritoneal effusion: here the efficient is usually black colored or what you called anechoic. There are extensive fibrin sheds that forms a carb egg like appearance.

(Showing pictures in the slide) These are ultrasonic features of the **Uroabdomen**: so bladder which has been ruptured and the fluid collected is anechoic seen as a black color.

(Showing pictures in the slide) This is an ultrasonic feature of a **Diaphragmatic Hernia**, where H is the Heart and R is the Reticulum. Now we need to appreciate the close association of the reticulum, which moves in association with the heart, as the heart moves the reticulum also moves, because of the absence of the diaphragm.

(Showing pictures in the slide) These ultrasonographic pictures on the left side show pointing at the reticulum and on the left show the heart. Please follow this to understand the next slide.

(Showing video in the slide) This video shows the movement of the heart, as well as the movement of the reticulum, which is in unison with the heart indicating a Diaphragmatic hernia.

(Showing video in the slide) This is another video in the case of a cattle with a Diaphragmatic hernia, we can appreciate the movement of the heart and the reticulum in association with the heart.

(Showing pictures in the slide) **Displacement of abomasum**: this is a cattle on the left side as well as right side these are affected with left side displacement of the abomasum.

(Showing pictures in the slide) This picture shows where we need to image and place the probe for abomasal displacement. Initially the abomasal is seen from the xiphoid caudal up to the pelvic area and then since there is a displacement so this area towards the right

side on the flank region, as well as on the left side from the elbow towards the left paralumbar fossa has to be extensively investigated for the abomasum. Or the area where there is a ping sound, that area can be visualized using a probe.

(Showing pictures in the slide) **Left side displacement of abomasum**: the left side photo indicates an animal with a left side displacement abomasum. There is an area demarcated with chalk, that indicates the area with a ping sound.

The right-side top photo indicates the Liptak test where we introduce the needle and collect the fluid. The right bottom slide shows the Liptack test, in this the pH is acidic. The acidic indicates it is the abomasum. Usually, the area where we have introduced the needle should be alkaline, indicating the reticular rumen. So the Liptak test in this left side displacement abomasum indicates acid confirming the displacement of the abomasum.

(Showing video in the slide) These are ultrasonic features of the left side displacement abomasum, where the abomasum is filled with the content, which is fluid, that is anechoic and content is echogenic. As it starts moving you can see the abomasal folds which are echogenic.

(Showing pictures in the slide)These are other ultrasonographic pictures, depicting the movement of the abomasum along with abomasum volvulus and anechoic and echogenic content.

(Showing pictures in the slide) This is a case of right side displacement abomasum, we have got two cattle and the site for investigation is from the elbow towards the paralumbar fossa. Apply liberal quantities of gel or spread and place the probe.

The left side indicates the ultrasonic features of right side displacement of the abomasum, where the abomasum is distended with the fluid anechoic and echogenic content. The right side picture shows the leaf-like structures, these are abomasal folds that are echogenic.

In this lesson, we have seen various ultrasonographic features in the abomasum as well as the reticulum and other features of

the abdominal cavity. In the next lesson, we are going to see the Management of Elias in the case of cattle.

Thank you!

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5 Management of Ileus

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Transcript

A warm Greetings! to all,

We have been seeing about Intestinal Ileus in cattle and in the previous lesson we have seen about Ultrasonography features of the thorax as well as the abdominal cavity.

In this lesson we are going to see the Ultrasonic features of Pregnancy and more important is the ultrasonic features of the Intestine and the Management of the Ileus.

Ultrasonography of pregnancy: here we need to appreciate the fetal heart movement and movements of the fetus and we have to recognition of the gestation sac, fetal parts, and heartbeat.

(Showing pictures in the slide) this ultrasonic feature depicts the chest cage of the fetus, where we can see the central part, and the heartbeat is also noticed.

Pregnancy:(Showing pictures in the slide) On the left side we can see the live fetus, where the heartbeat is also seen, and the fetal also actively moves as indicated by the arrow. On the right side, there is another fetus with the movement of the fetus.

Pregnancy ultrasonographic examination: here we can see the fetal head and the active movement of the fetal head, this is a frozen image that is used for assessment of the fetal age. Ultrasonic feature

of an early fetus: So, where you can see the entire thoracic cavity and the movement of the heart.

(Showing pictures in the slide) this slide shows where we are going to place the probe for ultrasonic investigation of the intestine, here we are going to investigate from tuber coxae to the 8th intercostal space, transverse process of the vertebrae to the linear album on the right side, so the usual diameter is usually 2-4 centimeter.

Normal small intestine: now we can see the cross-sections of a healthy small intestine, where you can see sacculations with echogenic content. So, the loops are in cross-section mostly in cross-section, occasionally we see it in longitudinal sections.

(Showing pictures in the slide) These are ultrasonic features of the small intestine where there is a beautiful cross-sectional image of the Intestine with echogenic content.

Dilated Caecum: it is usually with gas and the wall of the large intestine- the outer part is visualized as a thick echogenic line. Sometimes as echogenic semicircular lines, the internal content is usually not visualized.

This is the **Ileus**: where the concentration has to be done on Diameter, Motility, and Evidence of peritonitis. Here the diameter is usually increased by more than 4 centimeters and there will be hypermotility and usually, there will be an accumulation of fluid or peritoneal diffusion in case of delayed cases.

Ultrasonic examination of the Intestine: In this case, we can appreciate dilated intestine and the diameter is more than 4 centimeters. Here ultrasonic features of the intestine in the case of Ileus in addition to the increase in the diameter we can see anechoic fluid in between the Intestine.

Intussusception: Intussusception in the future has to be identified by the target lesion, as we see as round concentric rings of anechoic and hyperechoic lines.

(Showing video in the slide) the left side shows a rectal examination of an intussusception, there is an echogenic lumen, that is intestine and in the caudal or ventral part, we can see

concentric anechoic and hyperechoic lines indicative of intussusception.

(Showing pictures in the slide) This case is shown by anechoic intestinal loops and echogenic intestinal loops on the ventral part. So anechoic intestinal loops are with fluid, and echogenic is with the intestinal content. so, this indicates Intestinal obstruction.

Ileus: after having identified functional Ileus, by excluding mechanical Ileus and elimination by individual disease or disorder, a functional Ileus diagnosis is made. Once the diagnosis is made the important part is fluid therapy, the fluid has to be calculated based upon the dehydration as well as the maintenance requirement and it has to be given by rapid intravenous fluid administration set, to achieve the effective circulating volume. Usually, this will be supplemented with Calcium Potassium, and Phosphorus, the various dose are Calcium at 1gram/45 kg BW Potassium at 0.5 milliequivalent/kg BW, Phosphorus usually 4 (Dimethylamino)-2(Methylphenyl phosphonic acid) is administered at the rate of 10 milligram/kg BW IV, all these have to be diluted in a normal Saline.

Management of Ileus can be done with the 3 drugs namely:

- the Neostigmine
- Metoclopramide and
- Erythromycin Neostigmine- the dosage is 0.02 gram/kg BW and it has to be diluted in normal saline and can be given.

Another drug for the management of a functional Ileus is Metoclopramide at the dosage of 0.1 milligrams/kg BW essentially needs to be diluted in normal saline and administered. The effective drug when compared to the Metoclopramide and Neostigmine is Erythromycin, so at 1.1 milligrams to 8.8 milligrams per kg IV is very effective in producing the normal motility from 20 hours to 24 hours.

We have been discussing the various Etiology and how to Differentiate the Mechanical and Functional Ileus, the

Ultrasonography features of various disease conditions, and how to Manage the Intestinal Ileus.

This has to be followed to avoid the mortality in the animal because the loss of animals is a reduction in the economy of the farmer.

Thank you very much.

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PDF: Management of Ileus