TO THE POINT CLASS LECTURES

SYSTEMIC MEDICINE - I (MED 404) FINAL- 2

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Bovine Viral Diarrhea / Mucosal Disease Complex

Bovine viral diarrhea (BVD) is most common in young cattle (6-24 month old).

Etiology

Bovine viral diarrhea virus (BVDV), the causal agent of BVD and mucosal disease complex, is classified in the genus Pestivirus in the family Flaviviridae.

Based on viral RNA sequence, there are at least 2 viral genotypes of BVDV that can be further divided into subgenotypes. The viral genotypes are termed BVDV type 1 and BVDV type 2.

Genotypes of BVDV are separated into noncytopathic and cytopathic biotypes based on cytopathic effects observed in infected cell cultures. Noncytopathic BVDV are the predominant viral biotype in nature. Cytopathic BVDV are relatively rare and arise in cattle that are persistently infected with noncytopathic BVDV.

Most of our cattle herd populations are persistently affected with noncytopathic BVDV. Cattle that are persistently infected with noncytopathic BVDV serve as a natural reservoir for virus.

Disease induced by BVDV varies in severity, duration, and organ systems involved. Acute disease results from infection of susceptible cattle with either noncytopathic or cytopathic BVDV. Acute BVD, also termed transient BVD, often is an inapparent to mild disease of high morbidity and low mortality.

Transmission

a) *Transplancental* :

Persistent infection develops when noncytopathic BVDV is transmitted *transplacentally* during the first 4 months of fetal development. The calf is born infected with virus, remains infected for life, and usually is immunotolerant to the resident noncytopathic virus. Transplacental infection that occurs later in gestation results in abortion, congenital malformations, or birth of normal calves that have antibody against BVDV.

If transplacental transmission occur in 2nd or 3rd trimester; then there may be abortion, still birth, congenital malformation (cerebral hypoplasia) and some calves may born with antibodies against that virus.

b) *Horizontal* : Horizontal Transmission is also possible.

Persistently infected cattle can shed large amounts of BVDV in their secretions and excretions and readily transmit virus to susceptible herdmates. Clinical disease and reproductive failure often are seen after healthy cattle come in contact with a persistently infected animal. Biting insects, fomites, semen, biologic products, and possibly wild ruminants also can spread BVDV.

Clinical Findings

Biphasic fever (~104°F [40°C]), depression, decreased milk production, transient inappetence, rapid respiration, excessive nasal secretion, excessive lacrimation, and diarrhea are typical signs of acute BVD. Clinical signs of disease usually are seen 6-12 days after infection and last 1-3 days

If CBC is performed, there may be transient leucopenia because lymphoid tissues are severely infected

Recovery is rapid and coincides with production of viral neutralizing antibody. Gross lesions seldom are seen in cases of mild disease. Lymphoid tissue is a primary target for replication of BVDV, which may lead to immunosuppression and enhanced severity of intercurrent infections.

Some isolates of BVDV induce clinically severe disease that manifests as high fever (~107°F [41-42°C]), oral ulcerations, eruptive lesions of the coronary band and interdigital cleft, diarrhea,

dehydration, leukopenia, and thrombocytopenia. In thrombocytopenic cattle, petechial hemorrhages may be seen in the conjunctiva, sclera, nictitating membrane of the eyes; and on mucosal surfaces of the mouth and vulva. Prolonged bleeding from injection sites also occurs. On postmortem, swollen lymph nodes, erosions and ulcerations of the GI tract, petechial and ecchymotic hemorrhages on the serosal surfaces of the viscera, and extensive lymphoid depletion are associated with severe forms of acute BVD. The duration of disease may be 3-7 days. High morbidity with moderate mortality is common. Severity of acute BVD is related to the virulence of the viral strain infecting the animal and does not depend on viral biotype or genotype.

In pregnant cattle, BVDV may cross the placental barrier and infect the fetus. The consequences of fetal infection usually are seen several weeks to months after infection of the dam and depend on the stage of fetal development and on the strain of BVDV.

Infection of the dam near the time of fertilization may result in reduced conception rates. Infection during the first 4 months of fetal development may lead to embryonic resorption, abortion, growth retardation, or persistent infection. Congenital malformations of the eye and CNS result from fetal infections that occur between months 4-6 of development. Fetal mummification, premature birth, stillbirth, and birth of weak calves also are seen after fetal infection.

Persistent infection is an important sequela of fetal infection with noncytopathic BVDV. Persistently infected calves may appear healthy and normal in size, or they may show stunted growth and be prone to respiratory or enteric ailments. They often have a short lifespan, and death before 2 yr of age is common. Persistently infected cows give birth to persistently infected calves, but most calves sired by a persistently infected bull will not be infected with virus in utero. So as a veterinarian, culling is the best control.

Mucosal Disease

It is a highly fatal form of BVD that may be acute or chronic and is seen infrequently in persistently infected cattle. Mucosal disease is induced when persistently infected cattle become superinfected with cytopathic BVDV.

The origin of the cytopathic BVDV is usually internal, resulting from a mutation of the resident persistent, noncytopathic BVDV. In those cases, the cytopathic virus is antigenically similar to the resident noncytopathic virus. External origins for cytopathic BVDV include other cattle and modified live virus vaccines.

Hypophosphatemia

Phosphorus is essential for many intracellular processes, notably glycolysis, membrane maintenance, oxygen transport, muscle contraction, and protection from oxidative damage. It is also an important component of bones, teeth, milk, and ruminant saliva. Deficiency is usually primary and results in multiorgan system dysfunction and eventually progressive demineralization of bone.

Etiology

Phosphorus is supplied by the diet. Soil and pasture plants are naturally deficient in many parts of the world. Fertilization is used to increase plant phosphorus content, but inadequate or poor-quality fertilizer application, interference in uptake by other minerals, soil retention, leaching by rain, soil depletion through repeated crop or grass harvesting, and poor growth of plants during periods of drought may still result in phosphorus-deficient forage.

Vitamin D promotes intestinal uptake of phosphorus, but secondary deficiency related to hypovitaminosis D is uncommon in herbivores without at least marginal concurrent dietary deficiency. Secondary deficiency appears to be most common in areas with limited sunlight during critical skeletal growth periods, and is seen most frequently in young, growing animals.

Ruminants secrete large quantities of phosphorus in saliva. In times of need, this is recycled through the intestine; in times of sufficiency, fecal loss is the primary mode of excretion.

Clinical Findings

Most clinical manifestations of phosphorus deficiency arise only after several months of deficient intake. Poor growth, dull hair coat, subnormal milk production, and poor reproductive performance are among the earliest signs. Most animals in the herd are affected to some degree. Pica, including osteophagia, follows, with possible complications of botulism or traumatic reticulitis. Atypical shifting-leg lameness, swollen joints, and spontaneous fractures occur after longer periods of deficiency. Fractures occur most commonly in the ribs, pelvis, or vertebral bodies.

With secondary deficiency, osteodystrophic signs, including stiff-legged lameness, hunched posture, widened and painful growth plates and costochondral junctions, shortened long bones, and angular limb deformities, are seen earlier in the course of the disease

With multigenerational deficiency, cattle develop a characteristic appearance, including a rough hair coat, narrow girth with a small pelvis, and thin, breakable ribs. The thinness of the trunk imparts a “leggy” appearance to the cow.

Acute manifestations of phosphorus deficiency are rare. Recumbency due to apparent muscle weakness without hemolysis or mental dullness is occasionally seen in dairy cattle in late gestation or early lactation.

Intravascular hemolysis with hemoglobinuria may also occur in postparturient dairy cattle . These acute syndromes are seen exclusively in dairy cattle. It is postulated that the abrupt increase in phosphorus demand for fetal bone and maternal colostrum and milk production lead to rapid depletion of the circulating pool. High oxidant challenge and lack of sufficient antioxidant compounds may also contribute to clinical disease.

Lesions

Necropsy findings are not generally remarkable unless osteomalacia is significant, or fractures or hemolysis have occurred. Growth plates may appear wide and irregular in younger animals, with lack of mineralization.

Diagnosis

Diagnosis of phosphorus deficiency can be difficult and often requires exclusion of other causes of ill-thrift and poor reproductive performance, including selenium deficiency, protein-calorie malnutrition, and parasitism. Feed analysis with particular emphasis on phosphorus form and content is the most direct way of diagnosing a deficiency.

Bone resorption can be estimated by measuring serum hydroxyproline, an amino acid liberated from collagen, or by measuring the ratio of bone ash to organic matrix in a core biopsy. Fecal phosphorus provides an indirect measure of dietary sufficiency.

Treatment

Chronic syndromes can often be reversed by placing the animal on an adequate diet. Reproductive performance can improve in as little as a month; remodeled bone takes longer and, in severe cases, may not completely correct.

Oral supplements usually come in the form of dicalcium phosphate, rock phosphate, or bone meal. Mono-, di-, or trisodium phosphate, ammonium phosphate, phosphoric acid, and superphosphate may be used, but these must be handled carefully to avoid phosphorus toxicosis.

Acute syndromes require more immediate correction. There are no phosphorus-only compounds labeled by the FDA for parenteral use in cattle, and multiple mineral (predominately calcium) preparations contain a form of phosphorus with low bioavailability. Thus, all parenteral supplementation is extra-label.

Prevention

Phosphorus deficiency is best prevented by ensuring adequate dietary intake. Recommendations vary with area, age, and use of the cow. Maintaining at least 0.042% dietary phosphorus in dairy cattle appears to be adequate to prevent acute disease in most herds.

Total mixed rations, mineral supplements, top-dressed feeds, and supplemented drinking water all have been used successfully. The ratio of calcium:phosphorus in the diet should not be <2:1.

Hypocalcemia

Synonyms: Milk fever, Parturient paresis

Parturient paresis is an acute to peracute, afebrile, flaccid paralysis of mature dairy cows that occurs most commonly at or soon after parturition. It is manifest by changes in mentation, generalized paresis, and circulatory collapse.

It is most common in high yielding diary cows and less common on beef cattle and heifers as compared to sheep and goat.

Etiology

At or near the time of parturition, the onset of lactation results in the sudden loss of calcium into milk. Serum calcium levels decline from a normal of 10-12 mg/dL to 2-7 mg/dL. Commonly, serum magnesium is increased, serum phosphorus is decreased, and cows are hyperglycemic. The disease may be seen in cows of any age but is most common in high-producing dairy cows >5 yr old. Incidence is higher in the Jersey breed.

It is also due to the increased demand of calcium for fetal membrane development and parturition phenomenon in an animal.

Clinical Findings

Parturient paresis usually occurs within 72 hr of parturition. The disease can contribute to dystocia, uterine prolapse, retained fetal membranes, metritis, left abomasal displacement (LAD), and mastitis.

In sheep: having more number of fetuses at parturition increased the risk of parturient paresis.

In young animals; normally there is increased demand of calcium but low absorption from the GIT.

Paralysis occur due to inability of neuromuscular junction to transmit impulses. Calcium is also necessary for release of acetylcholine and for contraction of actin and myosin.

There are 3 discernible stages of parturient paresis (Milk fever).

Stage 1:

During stage 1, animals are ambulatory but show signs of hypersensitivity and excitability. Cows may be mildly ataxic, have fine tremors over the flanks and triceps, and display ear twitching and head bobbing. Cows may appear restless, shuffling their rear feet and bellowing. If calcium therapy is not instituted (within 1 hour), cows will likely progress to the second, more severe stage.

Stage 2:

Cows in stage 2 are unable to stand but can maintain sternal recumbency. Cows are obtunded, anorectic, and have a dry muzzle, subnormal body temperature, and cold extremities. Auscultation reveals tachycardia and decreased intensity of heart sounds. Peripheral pulses are weak. Smooth muscle paralysis leads to GI stasis, which can be manifest as bloat, failure to defecate, and loss of anal

sphincter tone. An inability to urinate may be manifest as a distended bladder on rectal examination. Cows often tuck their heads into their flanks, or if the head is extended, an S-shaped curve to the neck may be noted.

Sage 3:
In stage 3, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, have compete muscle flaccidity, are unresponsive to stimuli, and can suffer severe bloat. As cardiac output worsens, heart rate can approach 120 bpm, and peripheral pulses may be undetectable. If untreated, cows in stage 3 may survive only a few hours.

Diagnosis

Total calcium in the serum is determined (mostly).
Stage 1 : < 1.9 mmol /lit
Stage 2 : 1.25 mmol /lit
Stage 3: < 0.5 mmol /lit

Differential Diagnosis

Differential diagnoses include toxic mastitis, toxic metritis, other systemic toxic conditions, traumatic injury (eg, stifle injury, coxofemoral luxation, fractured pelvis, spinal compression), calving paralysis syndrome (damage to the L6 lumbar roots of sciatic and obturator nerves), or compartment syndrome. Some of these diseases, in addition to aspiration pneumonia, may also occur concurrently with parturient paresis or as complications.

Treatment

Treatment is directed toward restoring normal serum calcium levels as soon as possible to avoid muscular and nervous damage and recumbency. Recommended treatment is IV injection of a calcium gluconate salt, although SC and IP routes are also used.

A general rule for dosing is 1 g calcium/45 kg (100 lb) body wt. Most solutions are available in single-dose, 500 mL bottles that contain 8-11 g calcium. In large, heavily lactating cows, a second bottle given SC after 15 to 20 min of IV may be helpful because it is thought to provide a prolonged release of calcium into the circulation.

SC calcium treatment alone may not be adequately absorbed due to poor peripheral perfusion and should not be the sole route of therapy. No matter what route is used, strict asepsis should be employed to lessen the chance of infection at the injection site.

Magnesium is also given via oral route because it may protect against myocardial irritation caused by the administration of calcium. Most products available to veterinarians contain phosphite salts as the source of phosphorus. However, phosphorus found in blood and tissues of cattle is primarily in the form of the phosphate anion. No pathway exists for the conversion of phosphite to the usable phosphate form.

Administration of oral calcium avoids the risks of cardiotoxic side effects and may be useful in mild cases of parturient paresis. Calcium propionate 0.5 kg dissolved in 8-16 L water administered as a drench is effective and avoids the potential for metabolic acidosis caused by calcium chloride. Oral administration of 50 g of soluble calcium results in ~4 g calcium being absorbed into the circulation. It is a dramatic effect.

Hypocalcemic cows typically respond to therapy immediately. Approximately 75% of cows stand within 2 hr of treatment. Animals not responding by 4-8 hr should be reevaluated and re-treated if necessary. Of cows that respond initially, 25-30% relapse within 24-48 hr and require additional therapy. Incomplete milking has been advised to reduce the incidence of relapse. Historically, udder inflation

has been used to reduce the secretion of milk and loss of calcium; however, the risk of introducing bacteria into the mammary gland is high.

Prevention

a) Use of dietry cation-anion difference (DCAD)

Most recently, the prevention of parturient paresis has been revolutionized by the use of the dietary cation-anion difference (DCAD), which decreases the blood pH of cows during the late prepartum and early postpartum period. This method is more effective and more practical than lowering prepartum calcium in the diet. The DCAD approach provides an excess of anions over cations in the diet by adjusting the components of the diet, adding anionic salts to the ration, or both. Adding excess anions to the diet is believed to enhance calcium resorption from bone and absorption from the GI tract.

b) Administration of vitamin D_3

Administration of vitamin D_3 and its metabolites is effective in preventing parturient paresis. Large doses of vitamin D (20-30 million U, SID), given in the feed for 5-7 days before parturition, reduces the incidence. However, if administration is stopped more than 4 days before calving, the cow is more susceptible. Dosing for periods longer than those recommended should be avoided due to potential toxicity. A single injection (IV or SC) of 10 million IU of crystalline vitamin D given 8 days before calving is an effective preventive. The dose is repeated if the cow does not calve on the due date.

c) Use of synthetic bovine parathyroid hormone (PTH)

Use of synthetic bovine parathyroid hormone (PTH) may prove to be superior to administration of vitamin D metabolites. Vitamin D metabolites enhance GI calcium absorption, whereas PTH enhances GI calcium absorption and stimulates bone resorption. PTH is administered either IV 60 hr before parturition, or IM 6 days before parturition. Drawbacks to the use of PTH include increased labor requirements for administration, as well as the availability of such compounds.

Hypomagnesemic Tetany

Synonym: Grass tetany, Grass staggers

Hypomagnesemic tetany is a complex metabolic disturbance characterized by hypomagnesemia (plasma Mg <1.5 mg/dL [<0.65 mmol/L]) and a reduction in the concentration of Mg in the CSF (<1.0 mg/dL [0.5 mmol/L]), which lead to hyperexcitability, muscular spasms, convulsions, respiratory distress, collapse, and death. Adult lactating animals are most susceptible due to the loss of Mg in milk. Hypomagnesemic tetany occurs mainly when animals are grazed on lush grass pastures or green cereal crops, but can occur in lactating beef cows fed silage indoors. It is rare in nonlactating cattle but has occurred when undernourished cattle were introduced to green cereal crops.

Etiology

The disorder occurs after a decrease in plasma Mg concentration when absorption of dietary Mg is unable to meet the requirements for maintenance (3 mg/kg body wt) and lactation (120 mg/kg milk). This can arise after a reduction in food intake during inclement weather, transport, or when cows graze short-grass dominant pastures containing <0.2% Mg on a dry-matter basis.

Mg absorption from the rumen may be reduced when potassium and nitrogen intakes are high and sodium and phosphorus intakes are low. Soils that are naturally high in potassium and those fertilized with potash and nitrogen are high-risk areas for hypomagnesemic tetany. The more complex mineral interactions are likely to be involved in herds in which hypomagnesemic tetany occurs in first- and second-calving cows as well as in older cows.

Cows often do not develop signs of hypomagnesemic tetany until blood calcium concentrations are <8 mg/dL), which commonly occurs in cattle grazing green cereal crops. The hypocalcemia arises from either a reduction in calcium intake or absorption, or both. Lush grass pastures and green cereal crops

may predispose cattle to metabolic alkalosis (urine pH >8.5) with a reduced available pool of calcium, thereby increasing the risk of hypocalcemia. Urine Mg concentrations are a useful guide to Mg status and are undetectable in cows with hypomagnesemia.
Hypomagnesemic tetany occurs in 2- to 4-mo-old calves being fed milk only. Sole milk feeding can support a calf of weight up to 50 kg body weight.
Absorption of magnesium starts by calves at the age of 3 months. Decreased absorption of magnesium increased the risk of hypomagnesemic tetany.
Adult cows can not mobilize magnesium from the bones but calves can do that.

Clinical Findings

In the most acute form, affected cows, which may appear to be grazing normally, suddenly throw up their heads, bellow, gallop in a blind frenzy, fall, and exhibit severe paddling convulsions. These convulsive episodes may be repeated at short intervals, and death usually occurs within a few hours. In many instances, animals at pasture are found dead without observed illness, but an indication that the animal had convulsions before death may be seen from marks on the ground.

In less severe cases, the cow is obviously ill at ease, walks stiffly, is hypersensitive to touch and sound, urinates frequently, and may progress to the acute convulsive stage after a period as long as 2-3 days. This period may be shortened if the cow is transported or driven to a fresh pasture. When animals have hypocalcemia and hypomagnesemia, the signs shown depend on which predominates. With hypomagnesemia, tachycardia and loud heart sounds are characteristic signs

There are two forms of hypomagnesemic tetany on clinical basis.
i) Acute form:
There is excitement, hyperesthesia, rigid titanic spasm, convulsions, ophisthotonus, nystigmus, abnormal chewing movements. Animal may remain calm for some time but again start this episode.
Physical examination reveals loud heart sounds, increased body temperature (due to extraordinary movements of muscles) and increased pulse rate (tachycardia). This acute form is seldom treated. So, mortality rate is high.

ii) Sub acute form:
It lasts only for a duration of 2-3 days. There is anorexia, excitement, tenesmus, lock jaw, unsteady gait, pulsatile urination and defecation (constant finding). Spontaneous recovery may occur but again animal start episode. Span of this form is reduced when animal is transported.
Physical findings include tachycardia (heart rate = 200- 250 beat /min), loud heart sounds.
In calves, clinical picture starts with constant movement of ears, then pricking / backward movement of ears. Ophisthotonus, jerky movements of limbs, convulsions and involuntary urination.
Clinical signs of hypomagnesemic tetany in sheep occur when hypomagnesemia (plasma Mg <0.5 mg/dL) occurs concomitantly with hypocalcemia (plasma Ca <8 mg/dL]). The disease in lactating ewes occurs under essentially the same conditions and has the same clinical signs as in cattle.

Diagnosis

Diagnosis is usually confirmed by; i) response to treatment, followed by ii) confirmation of hypomagnesemia in samples taken prior to treatment.
One major difference in the therapeutic response in case of hypocalcemia and hypomagnesemia is that: In *hypocalcemia* : therapeutic response is dramatic but In hypomagnesemia : therapeutic response is very low; require more time.
Tetany usually occurs when plasma Mg is <1.2 mg/dL (0.5 mmol/L) in cattle and <0.5 mg/dL (0.2 mmol/L) in sheep. Urine Mg is usually undetectable in cows with hypomagnesemic tetany. Mg concentrations <1.8 mg/dL (0.75 mmol/L) in the vitreous humour of the eye removed from animals within 24 hr after death are indicative of hypomagnesemic tetany.
Normal ration between magnesium and calcium in the body = 1:50
It remains normal till = 1:70 but in hypomagnesemic tetany, this ratio goes to = 1:90

Differential Diagnosis
Hypomagnesemic tetany must be differentiated from; lead poisoning, rabies, hypovitaminosis A, tetanus, and nervous ketosis.

Treatment
Handle animal with extreme care because any physical stimuli can aggravate the condition. Animals showing clinical signs require treatment immediately with combined solutions of calcium and Mg, preferably given slowly IV while monitoring the heart. The response to treatment is slower in animals with hypomagnesemic tetany than in animals with hypocalcemia alone, due to the time it takes to restore Mg in the CSF. The animal should not be stimulated during treatment, as this could trigger fatal convulsions.

Combination therapy of calcium and magnesium:
Give calcium and magnesium mixture through IV route as follows:
Adult cattle : 500 ml Sheep/Goat : 200 ml

Additional Mg sulfate (200 mL of a 50% solution/cow) can be given SC to maintain level of magnesium.
Animals must be provided with hay treated with 2 oz (60 g) of Mg oxide daily; if this is not done, the condition can recur within 36 hr after therapy.

Prevention
Mg has to be given daily to animals at risk because the body has no readily available stores. Daily oral supplements of Mg oxide (2 oz [60 g] to cattle and 1/3 oz [10 g] to sheep) should be given in the danger period. Most Mg salts are unpalatable and must be combined with other palatable ingredients such as molasses, concentrates, or hay.

Feeding hay alone may be all that is required to prevent hypomagnesemic tetany in herds in which only old cows (>6 yr) are affected. If slow-release intraruminal Mg devices are administered, it is recommended that the animals also be provided with hay. Fertilizers containing Mg are effective in increasing herbage Mg only on certain soil types.
Herbage may be dusted with powdered Mg oxide (500 g/cow), or sprayed with a 2% solution of Mg sulfate at intervals of 1-2 wk.

Coccidiosis

Coccidiosis is responsible for huge economic losses: over 100 million dollar in USA
Most of the animals may be infected with coccidia but some animals show clinical signs because they receive small dose of coccidia oocysts.
Caused by species of genus Eimeria: *Eimeria bovis* or *Eimeria zuernii*.

Life Cycle
Oocyte in the feces of the first host------sporulated to form mature in the environment-------taken up bythe 2nd host so horizontal transmition-----go to intestine of the 2nd host----sporozoites develop----- enter into the intestinal cells-----trophozoites form there--------- merozoites --------attack on the other intesrinal cells---------- and form macro and micro gametes----------again immature oocyte
Oocyets are very resistant especially in an shady area with high temp and moisture. Multipilication rate of this organism is huge.

One oocyte give rise to 8 sporozoides and these 8 give rise to 120000 merozoits and it turns 80 % of macrogametes and 23040000 oocytes. Only 50000 oocytes are enough to cause a clinical disease in the calf.

Repture of the intestinal cells are of extreme importance in the coccidiosis.
Severity of the infection is directly proportional to the dose or number of the infective oocyte.

Intestinal cells have capability to regenerate themselves.
If a cattle is infected with a low dose of coccidia no disease will be there but animal will be infective and or next time this cow become immune to this species of the coccidia but not for others.

Stress is an important factor as weather and transportation.
It is primarily a disease of the young animals so older ones are resistant because they may exposed to low doses but these older animals shed oocyres in their feces.

Clinical Findings

Watery diarrhea may be bloody and diarrhea remains for week, fever, anorexia, depression, dehydration, tenesmus (contraction of the anal sphentar without passing the feces it is periodic)
Few of the animals remain underweight after coccidiosis.

Coccidiosis in calves

Nervous form in the calf, muscle tremors, hyperesthesia, ventroflection of the head and neck.
80-90% mortality rate in the calves and death in 24 hours.

Diagnosis

Isolation and identification of the oocyte.
There are two methods used for the fecal examination;
1) fecal flotation technique
2) Mac master technique
In both techniques, count the number of the oocyte which tells about the severity of the disease.

Differential Diagnosis

Bovine viral diarrhea (BVD), Intestinal parasite, Toxins and Salmonallosis

Treatment

Isolate the animal from the healthy animals.
Give supportive and parental therapy
Put the feeding and watering utensils high to animal head.
Administer Coccidostats such as: Amprolium @ dose rate: 10 mg per kg of body weight for 5-7 days, Sulpha quinoxyaline @ dose rate : 2-7 mg/kg for 5 days, Sulphamethazine @ dose rate; 110 mg/kg for 5 days. Parental sulphonamide therapy is used to avoid the secondary bacterial infection as pneumonia and bacterial enteritis.

Prevention

Agents that are used for prevention are as follows:
1—Lasolocids @ dose rate: 1 mg/kg body weight. Maximum dose is 360 mg per animal.
2—Monesin @ dose rate: 100-360 mg per head per day
These are growth promoters and improve the FCR.
But these are not effective for the treatment.
Monesin has low toxicity level so give carefully.
Sometime, after Monesin a fetal coccidiosis develops because it is immunosuppressive so not started once and nor withdraw once.
Well drained and dried places should be available for animals. Avoid fecal contamination.
It is difficult to control the coccidiosis on the form so do not make any big statement.

Lectures Delivered by Dr. Tanveer Ahmad

Deg Nala Disease

It is more common in rice growing areas. This disease is still confusing. Some believes that it is due to fungus. It was introduced in 1930 in the rice growing areas. Name came from Nala deg which is a monsoon drain. It starts from Sialkot and pass through Sialkot, Gujranwala, and Lahore.
In this area more crop of rice. It affects buffalo which is fed on rice straw.

Clinically it is characterized by gangrenous lesions on hoof, ears and tails. Initially necrosis and later affected area shed off.

Etiology:
There are two basic theories about cause:

According to Indian workers it is due to excessive ingestion of toxic amount of selenium contaminated fodder grown on selenium rich soil. Feeding of rice straw for long period is associated with it. Selenium is accumulated in rice straw. Its concentration is 8-11.6 times high in straw rice than grains and grains may contain 5.2-9.8 times higher concentration of Se than soil.

In Pakistan Dr. Irfan (Ex. Dean, Faculty of Veterinary Science) pointed out that this disease is due to mycotoxin produced on rice straw. Whenever stored in moist condition these may chances of growth of fungus and there will be production of mycotoxins.

An antidote Degcure developed at National Research Institute of India, Kernel. It contains $MgSO_4$ 1 Kg, $FeSO_4$ 166g, $CuSO_4$ 24 g, $ZnSO_4$ 75 g, $CoSO_4$ 15 g. Two doses of 30 grams are given orally/day. After 1-2 days you repeat dose and local ASD of lesions so that flies do not sit and do not produce maggots. Recovery after 10-15 weeks but not possible if hooves have shed.

Arsenic improves immunity. Arsenic preparation @ 10 ml/animal for 2-3 days intervals 3-5 injections can be given. Acetyllarson: cnontains 23.6 % arsenic.
Along with it orally give sodium arsenate: 60 mg/animal s/c by Star Labs (on alternate days).
Locally available toxin binders like myco-ad product of aerobina 60 g myco-ad in ½ litre of water for five days. After ten day to treatment animal may show normal sign. In our area condition has improved by giving mycotoxin binders. Advise formers to use supplement of Cu and zink in rice growing areas. 1-2 g to large animals for 10-15 days. Leave for 1 month and again for 15 days. ½ g in small animals for 10-15 g. It is mixed in water and flour for 15 days. Raw Cu not pure. Buffalo can tolerate upto 5 g Cu.

Great nursing and care is need in this case. In a hopeless case, hoof avulsion may occur which may take 8-9 months to recover.

Dermatomycosis / Ring worm

Its causative agent: Trichophyton and Microsporum canis
T. verrucosum (sub specie: album and discoides) T. mentagrophyte, T. megninii
In sheep T. verrucosum and M.canis
In goat T. verrucosum

Most common where animals are kept indoor and congested. Direct contact with infected animals or infected objects like bedding. Young animals are more susceptible, incidence is more in winter, healing spontaneously in spring but humidity is main factor.

Zoonosis:
Human acquires ring worm infection from equine and cattle, also from dogs.

Pathogenesis:
Ring worm fungus attack keratinized tissue, stratum corneum, and hair fibers. There will be breaking of hairs; alopecia develops. Exudation from infected epithelial layer starts. Epithelial debris and fungal hyphae produce crust that is characteristics of this disease. Warm and humid environment is favourable for mycillial group and alkaline pH of skin. Ring worm fungus is strict aerobes and dies under the crust. Living on the peripheral site active but die in centre. Due to this mode of growth it produces centrifugal progression (because animal requires oxygen for growth) and characteristic ring form of lesion. Secondary bacterial infection to hair follicle is common (antifungal as well as antibiotics must be administered).

Clinical Findings:
Crust is heavy and grayish white, raised above skin. Lesions are circular and 3 cm in diameter. In early stages surface below crust is moist. In older lesions the scab becomes detached and alopecia develops. Lesions are commonly on neck, head but general distribution over whole body particularly in calves. Itching does not occur. In sheep lesions are mostly present on head and very rare on wooly and fleece areas and disappear in 4-5 weeks. But disease may persist in flocks for some months. Lesions are normal patches covered with grey crust. Similar lesions are seen in goat.

Clinical Pathology and Diagnosis:
Skin scrapping: first defat with alcohol or ether. Then put a drop of 20 % potassium hydroxide or sodium hydroxide, warm it and make slide, observe polyhedral rounded, irregular spores in the form of chains or hair follicles.

Differential diagnosis:
Mycotic dermatitis, sarcoptic mange (mites present in microscopic examination).

Treatment:
Crusts are removed by soft brushing and burnt.
Apply weak solution of iodine, quaternary NH4 compound.
Sulphur was the first compound used against fungal infection effectively, it causes depletion of oxygen from the area. Take 250 g sulphur in 1 kg oil (1:4) and used as antifungal paste. Vaseline or simple water may be used in the sulphur to make the paste. Apply for 15-30 days.

Bourdeoux mixture ($CuSO_4$ + lime + water), it is basically used in paint.
10 % solution of NaI 1g/14 Kg.
Greseofulvin.
Systemic antibiotic therapy: such as Aminoglycosides, Penicillin (Penbiotin®), Tribrison.
Apply Gention violet 4-5 times in a day
Commercially available preparations: such as Dermasporin®, Lamisil®

Control:
Isolation and treatment of infected animals.
Adequate diet and good environment must be provided.
Disinfectant: 5 % formalin, 5 % Na hypochlorite.
2 % formaldehyde + 1 % Caustic soda are used for walls.
Use 20% Sodium Iodide (NaI) solution at dose rate 1ml/40 kg IV.

Dermatophilosis

Synonym:
Streptotrichosis, Mycotic disease, Lumpy wool of sheep, Senkobo disease

In central Africa named Senkobo, also present in equines, collectively in all species termed as dermatophilosis.

Etiology:
Dermatophilus congolensis is an infective agent but not invasive until skin damages. Organism is dimorphic and grows as branched filament and dormant zoospores which are transformed by moisture to infective stage of motile cocci. Animals of all ages are susceptible to this infection.

Sources:
There are no. sources:

- Minor active lesions on face and feet.
- Infection in scabs, still carried in hair and wool from healed lesions.
- D. congolensis normally does not reach the healthy skin; barriers are:
  - ✓ Stratum corneum
  - ✓ Wax produced by sebaceous gland
- Feet and face are the areas where these barriers are easily broken because of thorny and spiny feed stuff or forage.

Transmission:
It is a contagious disease. Transmission occurs from the carriage lesions by contact from the face of one animal to other animal, through flies and ticks, and from feet during dipping; dip also becomes the source of infection. In sheep if wool remains wet for long time.

Pathogenesis:
Minor trauma, exaggerate by wetting in sheep. Infection establishes where multiplication of organism starts and pyramidal crusts are formed by repeated cycles of infection into the epidermis by hyphae. Bacterial multiplication in epidermis. Rapid infiltration of neutrophils and regeneration of epithelium. Lesions remain expanding until immunity develops and healing start. The scab then separate from healed lesion but held loosely in placed by hair or wool fibers. Secondary bacterial infection occurs and gives rise to suppuration and severe toxemia.

Clinical findings:
In sheep lesions are commonly not visible due to presence of wool but crust can be palpated as hard masses at the surface of skin that is why called as lumpy wool disease. Lesions are distributed irregularly over the dorsal midline with ribs spreading laterally and ventrally. Crust are roughly circular and thick upto 3 cm. Pyramidal concave base, pigmented and underlying scab will be moist and red area. Muzzle, face and ears and scrotum of rams are involved. Heavy mortality in rams occurs due to cutaneous myiases and secondary pneumonia.
In cattle the lesion is first pustule, hairs of the infected site become erect and give a paint brush like appearance with greasy exudates forming hard crust that is hard to remove. These developed into scabs of white brown colour, 2-5 cm in diameter and will give mosaic appearance. Pus is formed in secondary infection. In cattle mostly neck, legs and udder of animal is involved. If calves come in contact with udder, muzzle of calves will be affected.

Clinical Pathology:
Skin scraping: zoospores are found. Serological evidence by ELISA, counter immunoelectrophorosis.

Differential Diagnosis:
Fleece rot in sheep, Corynebacterium pseudo tuberculosis in horse.

Treatment:
Penicillin: 70,000 IU/Kg b. wt. + streptomycin: 70 mg/Kg b. wt.
Erythromycin: 10 mg/Kg b. wt.

Lincomycin + spectinomycin: 5 mg/Kg b. wt. + 10 mg/Kg b. wt. respectively.
Tetracycline in large animal: (20 mg/Kg b. wt.), in small animal 5 mg/Kg b. wt.

Prevention:
Healthy animal should be dipped separately.

Rinderpest / Cattle Plague

It is a transboundary disease (the disease which spread in a large area without any discrimination of boundaries of countries). Pakistan was declared free from rinderpest in 2003. Before this, it was reported in Sindh. Vaccination of RP is not allowed in Pakistan.
Family of virus is Morbillivirus (Paramyxoviridae), many strains having similar genetic make up. Animals of all the ages can be affected. Large animals, sheep, goat, camels are affected.

Pathogenesis:
Virus is inhaled in infected droplets and it penetrates through the epithelium of upper respiratory tract, multiplies in tonsils and regional lymph nodes, from these sites virus enters the blood in mono nuclear cells and disseminated throughout the body because virus have high affinity for lymphoid tissues and alimentary mucosa. So it replicates in monocytes, lymphocytes and epithelial cells. Destruction of leukocytes results in leukopenia. Local necrotic stomatitis and enteritis result after proliferation in epithelial cells in alimentary tract and death occurs due to severe dehydration. In less acute cases death occurs due to activated latent parasitic or bacterial infection because the animal is immunosuppressd due to destruction of lymphoid organs by virus.

Clinical Signs:
Incubation period is 6-9 days and temperature may raise upto 105-107 ^{o}F. There are chances of absence of mucosal lesion. There is anorexia, decrease in milk yield and lacrimation.
In mucosal phase inflammation of buccal mucosa, nasal mucosa, conjuctiva, hyperemia of vaginal mucosa, swelling of genitalia and lacrimation becomes purulent. Blephropasm (spasm of eyelids), blood stained salivation, purulent salivation, halitosis (fowl odour from breathing of animal). Serous nasal discharge that later becomes purulent. Grayish raised necrotic lesions first appear inside the lower lip, adjacent gums, lower surface of the tongue, and mucosa at commissures. Later they become general including dorsum of tongue. Small lesions unite and become large which cause the sloughing. After sloughing of necrotic material red areas are left that form shallow ulcers. Severe diarrhea, sometimes dysentery. Skin becomes moist and red and later it is covered with scab.

Necropsy Findings:
Carcass will be dehydrated, emaciated, soiled with feacal material. Small necrotic areas found on oral mucosa; ulcers can be seen. These lesions extend to pharynx, upper esophagus, and abomasums; payer's patches become swollen and hemorrhagic. Necrotic zones of hemorrhage running transversely across the colon mucosa produce characteristic strips called zebra strips. Mucopurulent exudates in respiratory tract.

Tissues for Sampling:
Fixed sections of lymph nodes, tonsils, alimentary tract, fresh spleen and blood.

Treatment:
No treatment is given in this disease but left over and surroundings of animal are burnt.

Control:
Slaughter the infected animals. Proper cleaning and disinfection of premises. Adopt proper quarantine measures. Vaccination is not recommended in diseased animals. Ring Vaccination is started in the affected area.

Vaccine:
Universal vaccine is used; Tissue culture Rinder pest vaccine. It gives life long immunity. It is cultured on calf kidney cells. It should be used within 2-3 hours otherwise it will be destroyed.
Attenuated vaccine: virus is passaged in goats, rabbits, chicken eggs.
Vaccination failure occurs due to lack of cold chain in Pakistan.
OIE = Office International Epizootic

Theileriases

Theileriasis is a diseases of cross bred animals.
There are two types of Theileria:
Caused by *Theilaria parva*: East cost fever
Caused by *Theleria annulata*: Tropical or Mediterranean.
T. annulata is more common in the subcontinent (Indopak). These parasites can infect all types of cattle, buffalo, and yak.

Transmission:
Transmission is through ticks so control is very difficult. Ticks involved in transmission of T. parva are Rhipicephalous. T. annulata is transmitted by Hyaloma. Both these organism remain in tick for life span. Do not multiply in tick and comes in saliva. When bite, it transfer organism to animal. High temperature and high humidity is required for tick growth. These organisms complete their life cycle in lymphocytes (Schizonts) and in RBCs (piroplasm).

Clinical Signs:
Incubation period is 10-25 days. Swelling of L.N so locally called (*giltyon ka bukhar*) Prescapular L.N prominently swollen and visible in young calves. More deadly disease in young calves high rise in temperature, anorexia, emaciation, anemia, nasal discharge, corneal opacity. Sometimes Schizonts and piroplasm block the capillaries and cause hypoxic injury to the brain and you will see the nervous signs. Poor growth.
Annulata cause anemia, jaundice (destruction of RBC), prescapular L.N swelling and hemogloninurea.

Postmortem Findings:
Myocardial degeneration, hemorrhages on liver and spleen. If lungs involve thins exudates in lungs (bronchia, trachea). If not treated 100 % mortality occurs.
In endemic area severity of disease is less but in Theleria free region where if infection comes then severity is more. Death in summer. Animal becomes carrier after recovery.

Diagnosis:
Based on blood smear examination, stained with Geimsa stain, bluish bodies present in RBCs. Mostly sample is taken by absorption from swollen LN or micro capillaries of tip of ear. PCR, ELISA, CFT.

Treatment:
Most effective drug is buparvaquone (Butalex®) 1 ml for 20 Kg, 2 - 2.5 mg/ Kg I/M
Repeat after two days because animal may not respond to first infection but may quickly respond to second.
Oxytetracyclin and butalex: 10-20 mg/Kg for 4-6 days regularly.
Antipyretics/anti inflammatory drugs.
Fluid therapy/blood transfusion depends upon severity. 6-8 litres blood can be infused in cattle. If sever jaundice then give dextrose 5%. Prophylactic treatment is given when ticks are present. For this purpose inject Butalex 1 cc at the age of 7 days and repeat it after 1 month.

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