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Deficiency of zinc and cobalt in living organisms

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Summary:- Dietary deficiency of zinc are resulting parakeratosis. A zinc deficiency in young, growing pigs can cause parakeratosis, but it is not due to a simple zinc deficiency. The availability of zinc in the diet is adversely affected by the presence of phytic acid, a constituent of plant protein sources such as soybean meal. The disease occurs most commonly during the period of rapid growth and from 20 to 80% of pigs in affected herds may have lesions, and the main economic loss is due to a decrease in growth rate. The sign of zinc include Poor growth, stiff gait, swelling of the coronets, hocks, and knees, alopecia, wrinkling of the skin of the legs, hemorrhages around the teeth and ulcers on the dental pad. Skin biopsy will confirm the diagnosis of parakeratosis. Cobalt deficiency is a disease of ruminants ingesting a diet deficient in cobalt, which is required for the synthesis of vitamin B12. The disease is characterized clinically by in appetence and loss of body weight. Cobalt is unique as an essential trace element in ruminant nutrition, because it is stored in the body in limited amounts only and not in all tissues. No specific signs are characteristic of cobalt deficiency. A gradual decrease in appetite is the only obvious clinical sign. The treatment of deficiency of both cobalt and zinc is simply adding cobalt and zinc in to diet.
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1. Introduction

Malnutrition causes inappropriate growth and low body weight. Zinc and cobalt is very important source of elements for appropriate growth and to manage malnutrition problems of living organisms.

1.1. Zinc deficiency (parakeratosis)

1.1.1. Definition

Dietary deficiency of zinc and factors which interfere with zinc utilization are resulting parakeratosis.

1.1.2. Etiology

A zinc deficiency in young, growing pigs can cause parakeratosis, but it is not due to a simple zinc deficiency. The availability of zinc in the diet is adversely affected by the presence of phytic acid, a constituent of plant protein sources such as soybean meal. Much of the zinc in plant protein is in the bound form and unavailable to the monogastric animal such as the pig. Another unique feature of the etiology of parakeratosis in swine is that an excess of dietary calcium (0.5-1.5%) can favor the development of the disease, and the addition of zinc to such diets at levels much higher (0.02% zinc carbonate or 1001: mg/kg zinc) than those normally required by growing swine prevents the occurrence of the disease.

1.1.3. Epidemiology

Parakeratosis in pigs was first recorded in North America in rapidly growing pigs, particularly those fed on diets containing growth promoters. The disease occurs most commonly during the period of rapid growth, after weaning and between 7 and 10 weeks of age. From 20 to 80% of pigs in affected herds may have lesions, and the main economic loss is due to a decrease in growth rate. There are naturally occurring cases in cattle, sheep, and goats. The disease is well-recognized in Europe, especially in calves. It is common in some families of cattle and an inherited increased dietary requirement for zinc is suspected. The inherited disease occurs in Friesian and Black Pied cattle and is known as lethal trait A46.5 Signs of deficiency appear at 4-8 weeks of age. The main defect is an almost complete inability to absorb zinc from the intestine; zinc administration is curative. Outbreaks of the disease have occurred in Sudanese Desert ewes and their lambs fed on a zinc-deficient diet of Rhodes grass containing less than 10 mg/kg of zinc.

1.1.4. Pathogenesis

The pathogenesis of zinc deficiency is not well understood. Zinc is a component of the enzyme carbonic an hydrase, which is located in the red blood cells and parietal cells of the stomach, And it is related to the transport of respiratory carbon dioxide and the secretion of hydrochloric acid by the gastric mucosa. Zinc is also associated with RNA function and related to insulin, glucagon, and other hormones. It also has a role in keratinization, calcification, and wound healing, and somatic and sexual development. Because it has a critical role in nucleic acid and protein metabolism a deficiency may adversely affect the cell mediated immune system.

1.1.5. Clinical findings

Papules 3-5 mm in diameters, which are soon covered with scales followed by thick crusts. Crusts are most visible in areas about the limb joints, ears and tail and are distributed symmetrically in all cases. Crusts develop fissures and cracks, become quite thick (5-7 mm) and eaSily detached from the skin and diarrhea of moderate degree is common.

Experimentally produced cases exhibit the following signs include the poor growth, a stiff gait, swelling of the coronets, hocks, and knees, soft swelling containing fluid on the anterior aspect of the hind fetlocks, alopecia, wrinkling of the skin of the legs, scrotum and on the neck and head, especially around the nostrils, hemorrhages around the teeth and ulcers on the dental pad.

1.1.6. Clinical pathology

1.1.6.1. Skin scraping

Laboratory examination of skin scrapings yields negative results, But skin biopsy will confirm the diagnosis of parakeratosis.

1.1.6.2. Zinc in serum and hair

Serum zinc levels may have good diagnostic value, normal levels are 80-120 llg/dL (12.2-18.2 Ilmol/L) in sheep and cattle, levels of zinc in the blood are very labile and simple estimations of it alone are likely to be misleading, For example, other intercurrent diseases commonly depress serum calcium and copper levels and In addition, zinc levels in plasma fall precipitately at parturition in cows; they are also depressed by hyper thermal stress.

1.1.6.3. Necropsy findings

Necropsy examinations are not usually performed, but histological examination of skin biopsy sections reveals a marked increase in thickness of all the elements of the epidermis. Tissue levels of zinc differ between deficient and normal animals but the differences are statistical rather than diagnostic.

1.1.7. Differential diagnosis

1.1.7.1. Sarcoptic mange

May resemble parakeratosis, but is accompanied by much itching and rubbing. The parasites may be found in skin scrapings. Treatment with appropriate parasiticides relieves the condition.

1.1.7.2. Exudative epidermitis

Is quite similar in appearance, but occurs chiefly in un weaned pigs. The lesions have a greasy character that is quite different from the dry, crumbly lesions of parakeratosis.The mortality rate is higher.

1.1.7.3. Diagnosis

Response to therapy is the best diagnosis. Serum and hair levels for zinc can be low or normal; thus, these tests are not diagnostic. A skin biopsy shows evidence of parakeratotic hyperkeratosis.

1.1.8. Treatment

In outbreaks of parakeratosis in swine, zinc should be added to diet immediately at the rate of 50 mg/kg DM (200 mg of zinc sulfate or carbonate per kg of feed). The injection of zinc at a rate o f 2--4 mg/kg BW daily for 10 days is also effective. Zinc oxide suspended in olive oil and given 1M at a dose of 200 mg of zinc for adult sheep and 50 mg of zinc for lambs will result in a clinical cure within 2 months. The oral administration of zinc at the rate of 250 mg zinc sulfate daily for 4 weeks resulted in a clinical cure of zinc deficiency, in goats in 12-14 weeks.

1.1.9. Prevention and control

The calcium content of diets for growing pigs should be restricted to 0.5-0.6%. Weight gains in affected groups are appreciably increased by the addition of zinc to the diet The addition of oils containing unsaturated fatty acids is also an effective preventive For cattle, the feeding of zinc sulfate (2-4 g daily) is recommended as an emergency measure followed by the application of a zinc- containing fertilizer. The creation of subcutaneous depots of zinc by the injection of zinc oxide or zinc metal dust has been demonstrated. A soluble glass bolus containing zinc, cobalt, and selenium was able to correct experimentally induced zinc deficiency in sheep. Zinc-methionine, an organic zinc supplement for dairy goats improved udder health and enhanced the absorption of nitrogen and increased nitrogen retention.

2. Cobalt deficiency

2.1. Definition

Cobalt deficiency is a disease of ruminants ingesting a diet deficient in cobalt, which is required for the synthesis of vitamin B12. The disease is characterized clinically by in appetence and loss of body weight.

2.2. Epidemiology

Occurred primary in cattle and sheep unsupplemented with cobalt worldwide where soils are deficient in cobalt. Associated with ovine white liver diseases. Cobalt deficiency occurs in Australia, New Zealand, the UK, North America, the Netherlands/ and probably occurs in many other parts of the world. Where the deficiency is extreme, large tracts of land are unsuitable for the raising of ruminants, and in certain areas suboptimal growth and production may be limiting factors in in the husbandry of sheep and cattle. Australian investigations in the 1930s of two naturally occurring diseases, 'coast disease' of sheep, and 'wasting disease' or enzootic disease, of cattle.

2.3. Pathogenesis

Cobalt is unique as an essential trace element in ruminant nutrition, Because it is stored in the body in limited amounts only and not in all tissues. The effect of cobalt in the rumen is to participate in the production of vitamin B12 (cyanocobalamin), And compared with other species the requirement for vitamin B12 is very much higher in ruminants.The essential defect in cobalt deficiency in ruminants is an inability to metabolize propionic acid. Lack of vitamin B12 results in accumulation of methylmalonic acid which can be measured in the serum. A prolonged moderate cobalt deficiency in beef cattle (83 mg/kg) for 43 weeks results in several changes in lipid metabolism in addition to impaired growth. The efficiency of cobalt in preventing staggers in sheep grazing pasture dominated by (Phalaris tuberosa). And possibly by canary grass (Phalaris minor) or rhompa grass, a hybrid Phalaris spp., is unexplained. The pathogenesis of ovine white liver disease is unclear.

2.4. Clinical findings

No specific signs are characteristic of cobalt deficiency. A gradual decrease in appetite is the only obvious clinical sign. It is accompanied by loss of body weight, emaciation, and weakness. Pica is likely to occur, especially in cattle. There is marked pallor of the mucous membranes and affected animals are easily fatigued. Increased percentage of stillbirths, and increased neonatal mortality.

2.5. Clinical pathology

2.5.1. Biochemical criteria to determine cobalt and vitamin B12 status

Changes in the concurrent serum concentrations of methylmalonic acid and vitamin B12 of ewes and their lambs on cobalt deficient pastures. And their response to cobalt supplementation can be evaluated and monitored. These measurements are common y done along with recording live weight gains, Analysis of pasture for cobalt content at the sampling times for blood MMA and vitamin B12. Growth responses to cobalt or vitamin B12 supplementation is anticipated when cobalt levels in herbage fall below 0.08-0.1 mg/K DM.

2.5.2. Serum and hepatic cobalt and vitamin B12 concentrations Serum cobalt

Cobalt concentrations in the serum of normal sheep are of the order of 1-3 mg/dL (0.17-0.51 mmoI/L), and in deficient animals these are reduced to 0.03-0.41-mol/L. Serum vitamin Bu. Clinical signs of cobalt deficiency in sheep are associated with serum vitamin B12 levels of less than 0.20 mg/mL, and serum vitamin B12 1evels are used as a laboratory test of cobalt status in animals. Levels of 0.2-0.25 iJ-g/L are indicative of cobalt deficiency.

2.6. Necropsy findings

At necropsy, emaciation is extreme. The livers of sheep affected with white liver disease are pale and fatty. In most cases of cobalt deficiency, the spleen is dark due to the accumulation of hemosiderin. The microscopic changes of ovine white liver disease include hepatocellular dissociation and intracytoplasmic accumulations of lipid and ceroid-lipofuscin within hepatocytes. Biochemical assays reveal very high iron levels in the liver and spleen, and low cobalt levels in the liver. In normal sheep, cobalt levels in the liver are usually above 0.20 mg/kg DM, but in affected sheep are typically less than 0.05 mg/kg DM. Liver cobalt levels in cattle fed excessive amounts of cobalt and thought to be affected by cobalt poisoning can be as high as 69 mg/kg DM. Normal levels of vitamin B12 in the liver are of the order of 0.3 mg/kg, falling to 0.1 mg/kg in deficient lambs. In cattle, clinical signs occur with liver vitamin B12 levels of less than 0.10 mg/kg, and levels of more than 0.3 mg/kg of liver are necessary for optimum growth. Normal levels of the vitamin of cattle in New Zealand are 0.70-1.98 mg/kg of liver. Since serum B12 levels reflect cobalt status, it is often useful to submit sera from surviving in herd mates when attempting to confirm the diagnosis.

2.7. Differential diagnosis

Cobalt deficiency must be differentiated from other causes of 'ill-thrift' or 'enzootic Marasmus.

Thrift:-

In young animals, in which this situation is most often encountered, nutritional deficiencies of copper, selenium, and vitamin D are possible causes of ill-thrift. Lack of total digestible nutrients is the most common cause of thin animals. But owners are usually aware of the shortage and do not present their animals for diagnosis. However, it does happen, especially with urban people who become farmers and are unaware of the actual needs of animals. So it is best to check the feed supply and also to check whether or not the animals have any teeth.

2.8. Internal parasitism

Careful necropsy or fecal examination will determine the degree of helminthes infestation, But cobalt-deficient animals are more susceptible to parasitism and the presence of a heavy parasite load should not rule out the diagnosis of primary cobalt deficiency. Common for parasitic disease and cobalt deficiency to occur together in the one animal.

2.9. Diagnosis

2.9.1. Samples for confirmation of diagnosis

Toxicology - 50 g liver (ASSAY (Co)), 2 mL serum (ASSAY (B12)), Histology - formalin-fixed liver (LM).

2.10. **Treatment**

2.10.1. Cobalt and vitamin B12

Affected animals respond satisfactorily to oral dosing with cobalt or the IM injection of vitamin B12. Oral dosing with vitamin Bl2 is effective, but much larger doses are required. Oral dosing with cobalt sulfate is usually at the rate of about 1 mg cobalt/d in sheep and can be given in accumulated doses at the end of each week. On the other hand, the monthly dosing of lambs with oral doses of 300 mg cobalt is sufficient greatly to reduce deaths and permit some growth at suboptimal levels. The response to dosing is very quick, significant elevation of serum vitamin Bl2 levels being evident within 24 hr. Vitamin Bl2 should be given in 100-300 mg doses for lambs and sheep at weekly intervals. One injection of 1 mg provides protection to lambs for 14 weeks, and for weaners, protection for up to 40 weeks. Treatment of lambs with ovine white liver disease with hydroxocobalamin results in an immediate beneficial response and treatment is repeated 10 days later.

2.10.2. Cobalt toxicity

Overdosing with cobalt compounds is unlikely, But toxic signs of loss of weight, rough hair coat, listlessness, anorexia, and muscular in coordination appear in calves at dose rates of about 40-45 mg of elemental cobalt per 50 kg BW/day. Pigs have tolerated up to 200 mg cobalt/kg of diet. At initials of 400 and 600 mg/kg there is growth depression, anorexia, stiff legs, in coordination and muscle tremors. Supplementation of the diet with methionine, or with additional iron, manganese and zinc alleviates the toxic effects.

2.11. Prevetion and control

2.11.1. Supplement diet with cobalt

The recommended level of cobalt in the diet for sheep and cattle has for many years been about 100 mg/kg DM. Supplementation of the diet with cobalt is necessary. Calves reared on cobalt deficient pastures require cobalt or vitamin Bl2 supplementation prior to weaning.

2.11.2. Top dressing of pastures with cobalt

Cobalt deficiency in grazing animals can be prevented most easily by the topdressing of affected pasture with cobalt salts. The amount of top-dressing required will vary with the degree of deficiency. Recommendations include 400-600 g/ha cobalt sulfate annually or 1.2-1 .5 kg/ha every 3-4 years. The response to pasture treatment is slow, requiring some weeks to complete.

Supplementation of the diet with 0.1 mg cobalt/d for sheep and 0.3-1.0 mg/d for cattle are required, and can be accomplished by inclusion of the cobalt in salt or a mineral mixture. Cobalt can also be supplied to cattle in their drinking water supply.

2.11.3. Cobalt-heavy pellet

The use of 'heavy pellets' containing 90% cobalt oxide is an alternative means of overcoming the difficulty of maintaining an adequate cobalt intake in a deficient area.

The pellet is in the form of a bolus (5 g for sheep, 20 g for cattle) which, when given by mouth, lodges in the reticulum and gives off cobalt continuously in very small but adequate amounts. Administration of the pellets to lambs and calves less than 2 months old is likely to be ineffective because of failure to retain them in the undeveloped reticulum.

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