Hypomagnesemic tetanies

Tetany associated with depression of serum magnesium levels is a common occurrence in ruminants

The syndrome associated with hypomagnesemia is relatively constant , irrespective of the causes , but the group of diseases in which it occurs has been divided into hypomagnesemic tetany of calves , which appears to be due specifically to a deficiency of magnesium in the diet , and group of hypomagnesemias in ruminants characterized by lactation tetany , in which there may be a partial dietary deficiency of magnesium but in which nutritional or metabolic factors reduce these availability , or increase the body loss of the element so that serum magnesium levels fall bellow a critical point .

In general, the occurrence of hypomagnesemic tetany is related to three sets of circumstances:

Most common is the occurrence in lactating cows turned out on to lush, grass- dominant pasture in the spring often wintering in closed housing.

Wheat pasture poisoning may occur when any types of cattle or sheep is grazed on young, green cereal crops.

The third occurrence is in beef or dry dairy cattle running at pasture in the winter, usually when nutrition is inadequate and where no shelter is provided in changeable weather rather than in severe, prolonged cold.

Less common forms occur in housed animas on poor feed.

Hypomagnesemia of sheep, although it is less common, occurs in the same general groups of circumstances as the disease in cattle.

Hypomagnesemic tetany; lactation tetany; grass tetany;

Grass staggers; wheat pasture poisoning

Etiology:

Magnesium is the major intracellular divalent cat ion, and is an essential element in a large number of enzymatic activities in the body .however, because of the peculiarities of absorption 0of magnesium in the ruminant fore stomachs, and the use of animal and pasture management systems that can lead to marginal magnesium uptake, ruminants are at risk of hypomagnesemia.

Magnesium homeostasis:

There is no feedback regulatory mechanism to control concentrations of magnesium in the body of ruminants.

As a consequence, magnesium concentrations in blood and extracellular fluid are essentially determined by the balance between dietary intake of magnesium, loss in feces and milk, and the modulating effect of magnesium homeostasis by the kidney.

Dietary intake:

In normal circumstances, magnesium absorbed from the diet is sufficient to meet the requirements of the body and excess amounts are excreted in the urine.

Renal excretion:

The kidney is the major organ of homeostasis and can act to conserve magnesium. Magnesium is freely filtered across the renal glomerulus and is reabsorbed within the renal tubules, the degree of reabsorption acting in homeostasis.

When the dietary intake of magnesium is decreased, blood and interstitial fluid magnesium concentrations fall, excretion of magnesium in the urine will cease when serum concentrations fall below 1.8 mg / dl.

The renal threshold for magnesium excretion is partially under the control of parathyroid hormone and increased levels of parathyroid hormone will act to conserve magnesium.

Magnesium reserves

There are large stores of magnesium in the body , especially in bone .these are available to the young calf but mobilization decreases with age , and in the adult ruminant there is little mobilization in response to short –term deficits of magnesium .

In ruminants, this control mechanism for magnesium can maintain adequate concentrations of magnesium in bodily fluid in most production circumstances but it can fail where there is a high requirement for magnesium coupled with a decreased intake.

This combination leads to hypomagnesemia and hypomagnesemic tetany is a possible outcome.

Lactation

Increased requirement for magnesium is almost always associated with the loss of magnesium in the milk during lactation. Whereas the amount of magnesium in milk is not high (12 mg /kg) the loss of magnesium to milk represents a significant proportion of the dietary intake of magnesium.

As a consequence of this drain , most instances of hypomagnesemia occur in lactating animals around the period of peak milk production , although in some circumstances the demands of late pregnancy are the cause of the increased requirement .

The decreased intake of magnesium can result from an absolute deficiency of magnesium in the diet or because the availability or absorption of magnesium from the diet is impaired.

Factors influencing absorption of magnesium:

In the adult ruminants, magnesium absorption occurs in the fore stomach with little absorption in the abomasums and small intestine. Some absorption occurs in large intestine, particularly in sheep.

Na: K ratio in rumen:

Magnesium ids transported across the epithelium of the fore stomach by active sodium – linked ATPase – dependent transport system.

Absorption, and the serum magnesium concentration, is influenced by the Na: K ratio in the rumen, which is determined by the dietary and salivary concentrations of sodium and potassium.

Absorption of magnesium increases with an increasing Na: K ratio to plateau at a ratio of 5: 1.

Absorption is significantly impaired if the Na: K ratio is less than 3:1.

Young rapidly growing grass is low in sodium and high in potassium and can significantly depress the Na: K ratio in the rumen fluid, causing impairment of magnesium absorption.

Depression is observed at dietary potassium concentrations of greater than 22 g / kg dry matter.

Saliva

Normally has a high Na: K ratio but where there is a deficit of sodium in the diet , absorption of sodium in saliva may be replaced with potassium under the influence of aldosterone , which further negatively influences the uptake of magnesium

Approximately 40%of the total magnesium available in extracellular fluid is secreted daily in saliva and 20% of this is reabsorbed in the fore stomach.

When animals are on tetany – prone grass, this absorption is impaired, which accounts for the susceptibility of ruminants to hypomagnesemia compared to monogastric animals.

Others factors influencing absorption:

Young grass fertilized with nitrogenous fertilizers has an increased crude protein which is readily fermentable and leads to increased ammonia concentrations.

A sudden rise in ruminal concentrations of ammonia impairs magnesium absorption in the rumen.

The uptake of magnesium is also influenced by the carbohydrate content of the diet; magnesium absorption is improved with increasing amounts of readily degradable carbohydrates.

Volatile fatty acids provide the energy for the active transport of magnesium across the rumen wall and increase magnesium absorption.

Other dietary substances have been proposed to influence the absorption of magnesium including calcium and phosphorus, organic acids such as citric acid, fatty acids and aluminum, but the significance of their role is controversial.

Magnesium in pastures and tetany hazard:

The dietary intake of magnesium in grazing animals is directly related to the magnesium concentration in pasture but other elements in pastures also influence magnesium absorption by the ruminant.

Required magnesium concentrations

Hypomagnesemia can result from the ingestion of pastures that have insufficient magnesium to meet dietary requirements.

The estimated magnesium concentration in pasture required to meet the dietary requirement for pregnant or lactating cattle varies from 1.0- 1.3 g / kg dry matter for pregnant cattle, depending upon the stage of pregnancy, and 1.8- 2.2 g /kg dry matter for lactating cattle with both estimates assuming minimal interference of absorption by other elements in the pasture.

The recommended minimal safe concentration of magnesium in pastures is 2 g /kg dry matter for lactating and pregnant cattle with a preference for a concentration of 2.5 g /kg dry matter.

Magnesium availability in pastures and hazard:

Hypomagnesemia can also occur in animals grazing pastures with adequate concentrations of magnesium but that contain high conc4ntrations of potassium and nitrogen impaired absorption of magnesium in the rumen.

Pastures with concentrations of potassium of greater than 30 g K / kg dry matter and nitrogen greater than 40 g N / kg dry matter are considered hazardous.

Winter hypomagnesemia:

Hypomagnesemia also occurs in the winter periods .In housed lactating dairy cattle being fed conserved feeds.

It also occurs in cattle out wintered on poor quality feed.

Hypomagnesemia and hypocalcemia:

In some outbreaks of hypomagnesemic tetany ., there is also hypocalcemia , there is increasing evidence that the actual onset of clinical tetany may be associated with a rapid fall in serum calcium levels superimposed on pre – existing hypomagnesemia .

This is particularly true for the wheat pasture poisoning but can also apply to outbreaks with different predisposing factors.

Chronic hypomagnesemia can have a profound effect on calcium homeostasis .hypomagnesemia reduces the production and secretion of parathyroid hormones reduced hydroxylation of vitamin D in the liver , and also causes target organ insensitivity to the physiological effects of parathyroid hormone and 1,25 dihydroxy vitamin D3.

Chronic subclinical hypomagnesemia can increase susceptibility to milk fever and can predispose to episodes of milk fever and downer cows in lactating dairy cows during the period of peak lactation.

Summary of etiology:

A number of factors are capable of causing hypomagnesemia in ruminants out that under particular circumstances one or other of them may be of major importance.

In lactation tetany of cows and ewes, turned into lush pasture in the spring, a primary dietary deficiency of magnesium or the presence of high relative concentration of potassium and nitrogen in the diet reduces the absorption of magnesium and possibly calcium.

In wheat (cereal) pasture poisoning the ingestion of abnormally large amounts of potassium and low levels of calcium in the diet leads to hypomagnesemia and also hypocalcemia.

Hypomagnesemic tetany in cattle wintered at pasture, and exposed to inclement weather is associated with low magnesium intake and in adequate caloric intake, and possibly to the resultant hyperactivity of the thyroid gland.

Combinations of the above factors and other factors have etiological significance in individual outbreaks of the disease

The worst combination of the causative factors and the most common circumstances, in which the disease occurs, is inadequate energy intake with a low dietary content of magnesium (grass pasture) in recently calves' cows during as Pell of cold, wet and especially windy weather.

Epidemiology:

Occurrence and risk factors for lactation tetany.

Lactation tetany in dairy and beef cattle turned out to graze on lush, grass- dominant pasture after winter- housing is common in northern Europe.

With housed cattle, or cattle fed conserved feed during the winter, most cases occur during the first 2 weeks after the cattle are turned out to spring pasture.

Pasture which has been heavily top dressed with fertilizers rich in nitrogen and potash is potentially the most dangerous.

Cattle in the first 2 months of lactation and 4-7 years of age are most susceptible, which probably reflects an increased risk due to a higher loss of magnesium in milk.

Occurrence and risk factors for wheat (cereal) pasture poisoning

Wheat pasture poisoning is a misnomer as it can occur with grazing of any small grain cereal pasture.

It is most prevalent where young cereal crops are utilized for winter grazing.

The risk is with young rapidly growing pasture, either in the spring or in the autumn and winter with pastures planted in late summer.

Morbidity and mortality

In all of these forms of the disease, the morbidity rate is highly variable reaching as high as 12% in individual herds, and up to 2% in particular areas.

The incidence varies from year to year depending largely on climatic conditions, and management practices, and the disease is often limited in its occurrence to particular farms and even to individual fields.

Although an effective treatment is available, the case – fatality rate is high because of the short course, since animals die before they are observed to be ill.

Hypomagnesemia in sheep

It is more common in ewes bred for milk and lamb production.

In outbreaks, ewes with twins are more liable to develop clinical disease than those with single and the main occurrence is in ewes 1-4 weeks after lambing with cases up to 8 weeks after lambing.

Disease is often precipitated by management procedure involving movement and temporary food deprivation and cases will occur within the first 24h following this and for a few days after wards.

As in cattle, disease occurs when ewes are placed on lush grass pastures, but it is especially common where ewe in early lactation is placed on young cereal pastures.

Cases also occur in sheep which are exposed to inclement weather when on a low nutritive intake.

Pathogenesis

Most evidence points to hypomagnesemia as the cause of the titanic signs observed but the concurrent hypocalcemia may have a contributory effect and in many instances may even be the dominant factor.

Most clinical cases of the disease have serum magnesium levels below 1 mg / dl. Compared with the normal kevels in cattle of 1.7- 3 mg /dl, and there is a striking relationship between the incidence of the clinical disease and the occurrence of a seasonal hypomagnesemia.

The reduction in serum levels of magnesium in concurrent with a marked fall in the excretion of magnesium in the urine.

In affected herds and flocks, many clinically normal cows and sheep have low serum magnesium levels.

In some of these circumstances a concurrent hypocalcemia may be the precipitating cause.

Magnesium has many influences on impulse transmission at the neuromuscular system, including effects on the release of acetylcholine, on the sensitivity of the motor end plate, on his threshold of the muscle membrane and on activation of the cholinesterase system.

Magnesium concentrations in the cerebrospinal fluid are more predictive of clinical disease than those in serum, which would indicate that alterations in CNS function are more important than alterations in peripheral nerve function.

It is also evident that CSF levels of magnesium in hypomagnesemic animals rise significantly after treatment with a magnesium salt.

Clinical findings:

Lactation tetany is described in acute, sub acute and chronic forms.

Acute lactation tetany:

The animal my be grazing at the time and suddenly cease to graze, adopt a posture of unusual alertness and appear uncomfortable, twitching of the muscles and ear is also evident.

There is severe hyperesthesia and slight disturbances precipitate attacks of continues bellowing and frenzied galloping,

The gait becomes staggering and the animal falls with obvious tetany of the limbs, which is rapidly followed by clonic convulsions lasting for about a minute.

During the convulsive episodes there is:

Opisthotonos

Nystagmus

Champing of the jaws

Frothing at the mouth

Picking of the ears

Retraction of the eyelids

Between episodes, the animal lies quietly but a sudden noise or touch may precipitate another attack.

The temperature rises to 40- 40.5 oc after severe muscle extension

The pulse and respiratory rates are also high.

The absolute intensity of the heart sounds is increased so that they can be heard some distance away from the cow.

Death usually occurs within 1 h and the mortality rate is high because many die before treatment van is provided.

The response to treatment is generally good if the animal is treated early.

Sub acute lactation tetany:

In this form of the disease, the onset is more gradual. Over a period of 3-4 d., there is slight in appetence, wildness of the facial expression and exaggerated limb movement.

The cow often resists being driven and throws her head about as though expecting a blow.

Spasmodic urination and frequent defecation are characteristic.

The appetite and milk yield are diminished and ruminal movements decreases.

Muscle tremor and mild tetany of the hind legs and tail with an unsteady, straddling gait may be accompanied by retraction of the head and trismus.

Sudden movement, noise, the application of restraint or insertion of a needle may precipitate a violent convulsion.

Animals with this form of the disease may recover spontaneously within a few days or progress to a stage of recumbency with a similar but rather milder syndrome than in the acute form.

Treatment is usually affective but there is a marked tendency to relapse.

Chronic hypomagnesemia:

Many animals in affected herd have low serum magnesium levels but do not show clinical signs.

There may be sudden death.

A few animals do evidence a rather vague syndrome including dullness, unthriftiness and indifferent appetite and may subsequently develop one of the more obvious syndromes.

In lactating cows, this may be the development of paresis and a milk fever – like syndrome that is poorly responsive to calcium treatment.

Depressed milk production has also been attributed to chronic hypomagnesemia.

The chronic type may also occur in animals which recover from the sub acute form of the disease.

Parturient paresis with hypomagnesemia:

This syndrome consists of paresis and circulatory collapse in an adult cow which has calved within the preceding 48 h but in which dullness and flaccidity are replaced by hyperesthesia and tetany.

Clinical pathology:

Serum or urinary magnesium concentrations can be used for clinical cases. Where an animal is dead and hypomagnesemia is suspect, presumptive diagnosis can be made from samples taken from other at risk animals in the group, or from the vitreous humor of the dead animal.

Serum magnesium concentrations

Normal serum magnesium concentrations are 1.7-3 mg /dl.

These levels in cattle are often reduced in seasonal subclinical hypomgnesemia to between 1 and 2 mg /dl. But risk for tetany is not present until the levels fall to below 1.2 mg /dl.

The average level at which signs occur is about 0.5 mg /dl and in sheep it is suggested that clinical tetany does not occur until the serum magnesium level is below 0.5 mg /dl.

Serum magnesium in some animals may fall to as low as 0.4 mg /dl without clinical illness.

This is due to individual animal variation in the degree of ionization of the serum magnesium and the difference between serum and CSF concentrations.

Total serum calcium levels are often reduced to 5-8 mg /dl and this may have an important bearing on the development of clinical signs.

Serum inorganic phosphate levels may or may not be low.

In wheat pasture poisoning of cattle there is hypocalcemia, hypomagnesmia, and hyperkalemia.

In acute tetany, serum potassium levels are usually dangerously high and may contribute to the high death rate.

Urine magnesium concentrations

The occurrence of low urine magnesium levels is good presumptive evidence of hypomagnesemia.

Differential diagnosis

Cattle

Acute lead poisoning

Rabies

Nervous ketosis

Bovine spongiform encephalopathy

Treatment

IV administration of preparation contain magnesium or magnesium and calcium are used,

Response rates and recovery rates are much higher in cases treated early in the clinical course.

IV chloral hydrate may be administered to reduce the severity of convulsions during treatment with magnesium.

Case fatality, even with therapy, can be high, especially in advanced cases.

The safest general recommendation is to use a combined calcium magnesium preparation (e. g 500 ml of a solution containing 25% calcium borogluconate and %5 magnesium hypophosphite for cattle, 50 ml for sheep

IV followed by a SC injection of a concentrated solution of magnesium sat.

A combination of 12 % magnesium adipate and5% calcium gluconate at a dose rate of 500 ml is also used.

Hypomagnesemic tetany of calves

Etiology

The disease results when the dietary intake of magnesium is inadequate for the requirement of the calf.

Affected animals may have concurrent hypocalcemia.

Magnesium homeostasis in the calf

Milk has low concentrations of magnesium. a milk diet provides adequate magnesium for the requirements of a growing calf up to a body weight of approximately 50 kg , but if milk is the sole diet , the intake of magnesium will be inadequate for requirements once his body weight is reached ,.,.

In the young calf, magnesium is absorbed in the intestine

Te efficiency of magnesium absorption decreases markedly up to about 3 months of age, when maximum susceptibility to the disease occurs.

The efficiency of absorption is decreased by a reduction in transit time in the intestine caused by diarrhea.

In contrast to adult cattle, young calves can mobilize body stores of magnesium, which are principally located in the skeleton.

Epidemiology

Occurrence:

The disease is not common

Cases may occur sporadically or a number of deaths may occur on the same farm within a short period of time.

Risk factors:

The disease can occur under a number of different circumstances

Most commonly ; hypomagnesemic tetany occurs in calves 2-4 months of age or older which are fed solely on a diet of whole milk , and calves receiving the greatest quantity of milk and growing most rapidly are more likely to be affected because of their greatest need for magnesium for incorporation into developing soft tissues .,

It is most likely to occur in calves being fattened for veal .those cases which occur on milk replacer appear to be related to chronic scours and low magnesium content of the replacer.

A significant loss of magnesium in the feces also occurs in calves allowed to chew fibrous materials such as bedding; the chewing stimulates profuse salivation and creates greater loss of endogenous magnesium. Peat and wood shaving are bedding materials known to have this effect.

Cases have also been reported in calves fed milk – replacer diets or milk, concentrates and hay, and in calves running at pasture with their dams.

Deaths due to hypomgnesemic tetany have also occurred in 3-4 months old calves whose hay and silage rations were low in magnesium content.

Hypomagnesemia also occurs in young cattle, about 6 months of age, which are being fattened intensively in doors for the baby beef market. The phosphorus content of their diet is high and a lack of vitamin D is possible.

The hypomagnesemia is accompanied by a hypocalcemia.

Pathogenesis:

On affected farms, calves are born with normal serum magnesium levels of 2- 2.5 mg / dl, but the levels fall gradually in the succeeding 2-3 months, often to below 0.8 mg / dl.

Tetany does not occur until the serum magnesium galls below this concentration and is most severe at concentration below 0.6 mg /dl, although some calves in a group may have concentrations even lower than this and show few clinical signs.

Magnesium deficiency inhibits the release and action of parathyroid hormone and this is believed to be the genesis of the concurrent hypocalcemia.

It is probable that depression of the serum calcium level precipitates tetany in animals rendered tetany prone by low serum magnesium levels.

Tetanic convulsions can occur in hypocalcemic calves in the absence of hypomagnesemia.

Clinical findings:

Experimentally

Constant movement of the ears .the temperature is normal and the pulse rate accelerated.

Hyperesthesia to touch. And grossly exaggerated tendon reflexes with clonus, are present.

Shaking of the, opisthotonus, ataxia without circling, and a droopy, backward carriage of the ears is constant.

There is difficulty in drinking due to the animal's inability to get to the bucket.

Initially, the calves are apprehensive, show agitation and retraction of the eyelids when approached, and are hypersensitive to all external stimuli but show no tetany.

Later, fine muscle tremors appear, followed by kick in at the belly, frothing at the mouth and spasticity of the limbs.

Convulsions follow, beginning with stamping of the feet, head retraction, champing of the jaws and falling

During the convulsions the following signs are present jaws are clenched

Respiratory movements cease

There are tonic and clonic movements of the limbs

There is involuntary passage of urine and feces.

There are cycles of protrusion and retraction of the eye balls.

The pulse rate rises to 200- 250 \ min and the convulsions disappear terminally

The pulse becomes impalpable and cyanosis appears before death.

Older calves usually die within 20- 30 min of the onset of convulsions but young calves may recover temporarily only to succumb to subsequent attacks.

Cases which occur in young calves with scours , usually at about 2-4 weeks of age, show ataxia , hyperesthesia , opisthotonus and convulsions as the presenting signs .

The convulsion is usually continued and the calves die within 1 h.

Clinical pathology

Serum magnesium levels below 0.8 mg / dl indicate severe hypomagnesemia and clinical signs occur with levels of 0.3 – 0.7 mg / dl. Normal values are 2.2 – 2.7 mg / dl

Serum calcium levels tend to fall when serum magnesium levels becomes very low and are below normal in most clinical cases

Differential diagnosis

Acute lead poisoning

Enterotoxemia cause by clostridium perfrenges type D

Polioencehalomalacia

Tetanus

Vitamin A deficiency

Meningitis

Treatment:

Response to magnesium injections (100 ml of a 10% solution of magnesium sulfate) is only transitory because of the severe depletion of bone reserves of magnesium. Followed – up supplementation of the diet with magnesium oxide or carbonate is advisable

Chloral narcosis or tranquilization with an ataractic drug may be essential to avoid death due to respiratory paralysis during convulsions.