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Nutritional disorder in ruminant animals

1. **Yusuf Ahmed Hassen**

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Abstract

In this review we focused on nutritional disorders in ruminant can result poor animal health, lowered production, and animal loss. Mineral imbalances, sudden shifts from high roughage to high concentrate diets, and consumption of foreign objects are some of the factors associated with nutritional disorders in beef cattle. The common nutritional disorders in this review are bloat, acidosis, hardware and liver abscess.

Key words: nutrition, disorder, bloat, acidosis and liver abscess

Introduction

Livestock play vital roles for human being(Zereu, 2016), they utilize and convert grains and roughages feeds in to human utilizable forms. Nutritional disorders in ruminant can result in poor animal health, lowered production, and animal loss. Mineral imbalances, sudden shifts from high roughageto high concentrate diets, and consumption of foreign objects are some of the factors associated with nutritional disorders in beef cattle(States, 2005). Conditions associated with mineral imbalances include grass tetany, water belly, polioen cephalomalacia, white muscle disease, and milk fever. Nutritional disorders outlined in this review include bloat, acidosis, liver abscess and hardware disease. Bloat is a common digestive disorder in beef cattle. It occurs most often in feedlot cattle but affects cattle in all production phases. It results when cattle cannot belch (eructate) or release gases produced from microbialfermentation in the rumen. The animal may produce more gas than it can eliminate. Rumen expansion from gases puts pressure on the diaphragm and lungs. This compression reduces or cuts off the animal's oxygen supply and can eventually suffocate cattle. Frothy bloat (feedlot bloat) is the most common type ofbloat. It happens when foam in the rumen stops the animalfrom expelling rumen gases. Acidosis occurs as a consequence of the extensive use of highly processed grain and low levels of roughage in beef cattle finishing diets. In the survey of nutritional and management practices in US feedlots, 61.3% of feedlots with a capacity of greater than 8000 animals used steam-flaked grain in finishing diets. Summaries of in vivo data have documented that ruminal, intestinal, and total digestibility of starch is greater with steam-flaked corn and sorghum than with their dry-rolled counterparts.

Processing cereal grains by heat and moisture methods like steam flaking gelatinizes starch, increasing both the rate and extent of starch

fermentation in the rumen, with associated acid production and increased risk for acidosis. (Galyean & Rivera, 2003)Described both acute and sub-acute acidosis as conditions that can occur in cattle fed high-concentrate diets, acute acidosis is an overt illness that occurs in conjunction with consumption of an excessive quantity of readily fermented carbohydrate, which increases the total acid supply from the rumen. Lactic acid, normally found at very low concentrations in the rumen, can increase substantially, along with increased concentrations of volatile fatty acids (VFA). As a result of the increasing acid load, cattle with acute acidosis experience a drastic decrease in ruminal pH, with a pH of 5.2 considered as a benchmark. Absorption of acids into thebloodstream can lead to systemic acidosis, stiffness in the legs, and laminitis or founder, severe ruminal lesions, and sometimes death. In contrast to acute acidosis, cattle with sub-acute acidosis do not typically show outward signs of illness, but often exhibit decreased feed intake and lower performance. Liver abscesses are a common disorder in feedlot cattle inthe United States, with occurrence rates ranging from 0 tonearly 100%. Liverabscesses accounted for 46% of liver condemnations in the United States, which was more than double the rate for liverflukes (22%), the second highest in canadian 14% were approved for use in pet food, and 72% were approved for human consumption. Liver abscesses were responsible for the majority. The occurrence of liver abscesses is generally considered to be closely related to acidosis because it is thought that a continual highacid load in the rumen can lead to parakeratosis or similarinsults to the ruminal wall, leading, in turn, to clumping andnecrosis of ruminal papillae. These ulcerative lesions, along with hairs or other foreign objects that become bedded in the ruminal epithelium provide routesof entry into the portal system for microbes that cause liverabscesses; however, the exact mechanism of pathogenesis isunknown. Therefore bloat, acidosis, liver abscess and hardware are mutual nutrition disorder of ruminant anima(States, 2005)l.

Objective

To briefly review nutritional disorder in ruminant animals

Nutritional disorders in ruminant animals

Nutritional disorders in beef cattle can result in poor animal health, lowered production, and animal loss. Losses caused by digestive problems such as bloat and acidosis. Cattle death from digestive problems increased as herd size decreased. Mineral imbalances, sudden shifts from high roughageto high concentrate diets, and consumption of foreign objects are some of the factors associated with nutritional disorders in beef cattle. Conditions associated with mineral imbalances include grass tetany, water belly, polioen cephalomalacia, white muscle disease, and milk fever(States, 2005).. There are also several nutritional, metabolic, and digestive motility disorders that may disrupt nutrient assimilation and cause loss of condition(Asín et al., 2021).

Bloat

Bloat is a common digestive disorder in beef cattle. It occurs most often in feedlot cattle but affects cattle in all production phases. It results when cattle cannot belch (eructate) or release gases produced from microbialfermentation in the rumen. The animal may produce more gas than it can eliminate. Rumen expansion from gases puts pressure on the diaphragm and lungs. This compression reduces or cuts off the animal's oxygen supply and can eventually suffocate cattle. Frothy bloat (feedlot bloat) is the most common type ofbloat. It happens when foam in the rumen stops the animalfrom expelling rumen gases. The foam can cover the cardia(esophageal entrance from the reticulorumen) and prevent the animal from belching. Frothy bloat occurs in cattle fed high-grain diets but is not a major concern for many Mississippi cattle producers. "Feedlot" bloat is a concern,though, with cattle on high-grain diets, such as bulls on feed-based bull development programs. Consuming forages with high levels of soluble protein (such as alfalfa, winter wheat, and white clover) contributes to stable form production. This is called frothy pasture bloat or legume bloat. Legumes that contain leaf tannins help break up the foam in the rumen and are rarelyassociated with bloat. These tannin-containing legumes include arrow leaf clover, berseem clover; birds foot trefoil, sericea lespedeza, annual lespedeza, and crown vetch. Tropical legumes such as kudzu, cowpea, perennial peanut, and alyceclover rarely cause bloat. Bloat can also occur on lush annually grass or small grain pastures, particularly in spring(Galyean & Rivera, 2003).

Factors Affecting Feedlot Bloat

An overview of factors affecting feedlot bloat adapted. As with acidosis and liver abscesses, feedlot bloat is largely a disease that results from the feeding of high-grain diets. Thus, factors associated with the feeding of grains will affect the incidence and severity of bloat. Because cereal grains differ in the rate and extent of fermentation of starch, differences exist in the rate of acid and gas production among grains. Among cereal grains, the ranking of ruminal and total tract starch digestionis oats = wheat > barley > corn > sorghum. Differences in the nature of endosperm protein also exist among grains, which are associated with differences in ruminal starch digestion. indicated that the total tract protein digestibility of corn, sorghum, and barley(Galyean & Rivera, 2003)

Bloat Signs

Cattle suffering from bloat swell rapidly on the left side and may die within an hour. Sudden death from bloatis frequently cited in feedlots as a cause of cattle losses. Cattle may show signs of discomfort by kicking at their sides or stomping their feet. Susceptibility to bloat varies with individual animals. Some animals tend to bloat whenothers do not(States, 2005).

Management of bloat

Do not turn shrunk or hungry cattle out onto lush legume or small grain pastures without first filling them up on hay. Bloat is still possible on these forages, even after a frost. Bloat risk is lower later in plant growth when legumes begin to flower. Use of forages containing condensed tannins can help prevent bloat. Slowly adapt cattle from forage-based diets to grain-based diets over a period of at least three weeks. Manage the nutritionalprograms of chronic bloaters carefully. Provide poloxalene in a salt-molasses block or as a topdressing according to label recommendations. If providing poloxalene blocks, make sure cattle consume the blocks at least three days before placing the cattle on a pasture with a significant bloat risk. Remove other sources of salt,and place poloxalene blocks (30 pounds per four to five animals) where they will be easily accessible to the cattle.Feeding monensin can reduce the risk of both feedlot and pasture bloat. Monensin is reported to be more effective than lasalocid in controlling bloat, while poloxalene is more effective than monensin for bloat prevention(Mcguffey et al., 2001). A veterinarian or experienced person can administer poloxalene through a stomach tube to help break up the stable foam and let the animal belch. Do not drench a bloated animal because of the danger of inhalation and theresulting possibility of

pneumonia or death. Feed coarsely chopped roughage as 10 to 15 percent of the ration in a finishing diet. A bloat needle (6 to 7 inches long) or a trocarcan be used in extreme cases to puncture the rumen wall on the left side of the animal to relieve pressure inside the rumen. Consider this as a last resort, because severe infections may result. Although there is no label claim, research indicates that monensin reduces the incidence and severity of frothy bloat(Mcguffey et al., 2001).

Acidosis

Acidosis occurs as a consequence of the extensive use of highly processed grain and low levels of roughage in beef cattle finishing diets. In survey of nutritional and management practices in US feedlots, 61.3% of feedlots with a capacity of greater than 8000 (States, 2005)animals used steam-flaked grain in finishing diets. Summaries of in vivo data have documented that ruminal, intestinal, and total tract digestibility of starch is greater with steam-flaked corn and sorghum than with their dry-rolled counterparts. Processing cereal grains by heat and moisture methods like steam flaking gelatinizes starch, increasing both the rate and extent of starch fermentation in the rumen, with associated acid production and increased risk for acidosis. (Gadberry & Powell, n.d.)described both acute and sub-acute acidosis as conditions that can occur in cattle fed high-concentrate diets. Acute acidosis is an overt illness that occurs in conjunction with consumption of an excessive quantity of readily fermented carbohydrate, which increases the total acid supply from the rumen. Lactic acid, normally found at very low concentrations in the rumen, can increase substantially, along with increased concentrations of volatile fatty acids (VFA). As a result of the increasing acid load, cattle with acute acidosis experience a drastic decrease in ruminal pH, with a pH of 5.2 considered as a benchmark. Absorption of acids into the bloodstream can lead to systemic acidosis, stiffness in the legs, and laminitis or founder(Asín et al., 2021) , severe ruminal lesions, and sometimes death. In contrast to acute acidosis, cattle with sub-acute acidosis do not typically show outward signs of illness, but often exhibit decreased feed intake and lower performance. In sub-acute acidosis, ruminal pH drops substantially, and decreased feed intake follows the severity of the acid load. A ruminal pH of 5.6 has been considered as a benchmark of sub-

acute acidosis. In their excellent review article, described the sequence of events leading to acidosis and discussed potential points of control. The disease process begins when the animal consumes a large quantity of starch or other rapidly fermented carbohydrate. Glucose, normally found at very low concentrations in the rumen.

Liberated from starch, resulting in ruminal glucose concentrations that can exceed those in the blood, these high glucose concentrations can cause several negative consequences, including: growth of organisms like Streptococcus bovis and other microbes that produce lactic acid; growth of coliforms and amino-acid decarboxylating microbes that produce endotoxins and amines like histamine that can subsequently contribute to the development of laminitis; and increased ruminal osmolality, which exacerbates ruminal acidity by inhibiting VFA absorption from the rumen. For reasons unknown at present, ruminal glucose concentrations remain high for an extended period during acute acidosis. Glucose is subsequently fermented via glycolysis through pyruvate to lactate or VFA. Ruminal microbes that ferment glucose to lactate tend to be fairly insensitive to low pH, whereas most lactate-utilizing microbes in the rumen are sensitive to low pH, thereby favoring production and accumulation of lactate. Lactate is produced as both the D and L isomers, with concentrations of greater than 40 mm considered reflective of severe acute acidosis. Total VFA concentrations increase along with lactate, and ruminal pH decreases substantially because increased ruminal osmolality decreases absorption of acids. As pH continues to decrease and acid concentrations increase, absorption proceeds by passive diffusion(Galyean & Rivera, 2003). The capacity of the ruminal wall and liver to metabolize acids can be overwhelmed, which leads to high blood concentrations of acids that can potentially overpower the bicarbonate buffering system. In addition, blood osmolality increases because increased ruminal osmolality pulls fluid from the blood into the rumen and because acids that have accumulated in the blood have a direct effect on osmolality. If the animal is unable to restore homeostasis, death can result; however, even when animals recover, damage to the ruminal epithelium, laminitis, and subsequently lowered or erratic feed intake can have significant long-term effects on

productivity. Rumen acidosis (ruminal acidosis, rumen lactic acidosis, grain overload, rumenitis) is a metabolic disorder that occurs after the ingestion of readily fermentable carbohydrates and their subsequent rapid ruminal fermentation. The disease occurs frequently in cattle and small ruminants, and may be acute, sub-acute, or chronic. Ruminal acidosis associated with ingestion of excess carbohydrate in sheep is usually associated with intensive breeding systems. While acute acidosis usually causes death, sub-acute and chronic acidosis are responsible for loss of production. Sub-acute and chronic acidosis causes rumenitis, which is also important as a port of entry for *Fuso*- bacterium necrophorum and fungi, thus leading to secondary ruminal and hepatic infections. Primary tympany (frothy bloat) is another complication of rumenitis. The pathogenesis of acute ruminal acidosis is usually the consequence of sudden in gestion of excess carbohydrate in animals not accustomed to it. These carbohydrates include, amongst others, grain, bread, waste baked goods, brewers' waste, root crops, and apples. The amount of carbohydrate necessary to produce acidosis is highly variable and the individual tolerance increases when those carbohydrates are introduced progressively. Hence, the sudden introduction or increase of carbohydrates is more critical than the actual amount(Asín et al., 2021).

Factors Affecting Acidosis

In commercial feedlots, cattle are adapted gradually to a final high-grain diet(Galyean & Rivera, 2003). This adaptation process is designed to minimize the risk of acidosis. It is presumed that careful bunk management designed to provide cattle with relatively constant day-to-day feed intake during the adaptation period is important in preventing acute and sub-acute acidosis(Galyean & Rivera, 2003). However, because pens of cattle are fed in commercial feed- lots, and wide swings in intake by individuals within the pen are possible, this transition period represents the time during which acidosis is most likely to occur. Roughages typically have lower fermentable carbohydrate content than grains do, so increasing roughage decreases the chance for an acute ruminal acid load. The ability of roughage to stimulate chewing and saliva flow also should aid in moderating ruminal pH.

Inherent buffering capacity of roughage could be a factor, but suggested that buffering by feeds would most likely occur at a pH less than 5 and that the potential for direct buffering by dietary ingredients was a small fraction of buffering by saliva. Sources of fiber other than those provided by traditional roughages might have beneficial effects on acidosis. For example, based on results with individually fed cattle used to measure variation in feed intake and on ruminally cannulated cattle exposed to an acidosis challenge model, that a digestible fiber source like wet corn gluten feed did not eliminate acidosis but decreased the time that cattle were exposed to an acid insult, animal-to-animal variation in the ability to handle an acid load seems fairly substantial, even in model systems where attempts have been made to apply a relatively constant acid load. For example, used a sub- acute acidosis model that involved adapting ruminally cannulated steers (five per treatment) to a 50% concentrate diet at a restricted intake [1.7% of body weight (BW)], with-holding feed the day before the challenge, and then dosing a challenge diet (all-concentrate, wheat corn mixture) into the rumen at a rate of 1.5% of BW. Similarly, the acute acidosis model used by involved adapting five ruminally cannulated steers to grass hay, withholding feed the day before the challenge, and then dosing steam- flaked corn (3% of BW) into the rumen over a 4-h period. After analyses of the ruminal and blood samples taken following the challenges, concluded that, on the basis of ruminal pH, ruminal and plasma lactate concentrations, and dry matter (DM) intake, two of the five acute-challenge steers did not experience acute acidosis, and one of these two did not experience sub- acute acidosis. Likewise, only one of the five sub-acute- challenge steers experienced sub-acute acidosis. These results suggest that beef cattle have a fairly remarkable ability to adapt to abrupt changes in acid load. Moreover, given the degree of variation among animals noted by(Galyean & Rivera, 2003), the relatively low rates of occurrence of and mortality from digestive disorders in feedlots noted previously are not surprising.

Intake and decreased BW gain are often associated with sub-acute acidosis, but results of production-level research to verify these effects are limited. Feed bunk management is an important aspect of nutritional consulting; however, the extent

to which feeding management systems affect the incidence of metabolic disorders like acidosis is largely unknown. Research is needed to identify the relationships between feeding management practices (e.g., restricted feeding, programmed feeding, and clean bunk management), feeding patterns, and the incidence of acidosis. Variation in feed intake has been suggested to increase the incidence of metabolic disorders like acidosis, and found that varying intake decreased performance by feedlot cattle. Nonetheless, reported no effects, and in some cases slight positive effects, of variation in intake on feedlot cattle performance. And suggested that given the complexity of metabolic disorders like, acidosis a comprehensive evaluation of the relationship of feed intake variation to performance and the incidence of acidosis and other metabolic disorders is needed. Feeding behaviour might affect acidosis, but the extent of its impact is largely a matter of speculation because research in this area is limited. Because feedlot cattle are typically fed in large groups, social behavior patterns (e.g., dominance structure in a pen of cattle, role models, previous experience, and so on) might affect eating patterns and the incidence of metabolic disorders like acidosis. Research is needed to establish baseline data on the feeding and social behaviors of beef cattle fed high-concentrate diets. Studies conducted under commercial feedlot conditions in large pens would likely be the most useful. The effects of previous nutritional history on feeding behaviors and potential interactions of feeding behavior with nutrition and management need to be evaluated. If feeding and social behavior patterns affect the incidence of metabolic disorders, quantifying their effects should allow development of more effective model systems that would allow the use of individual animals to mimic metabolic disorders in feedlot cattle(Galyean & Rivera, 2003).

Feed additives can be an important tool for managing acidosis. Monensin has been shown to decrease day-to- day variation in feed intake, which, coupled with a potentially greater frequency of smaller meals, a lower total feed intake, and direct effects on inhibi tion of lactate-producing bacteria, might decrease the odds of high acid production in the rumen. Ionosphores like lasalocid and laidlomycin propionate do not seem to have major effects on feed intake, and their effect on acidosis and other

metabolic disorders is not as well documented as with monensin. The role that dietary buffers play in feedlot acidosis is not well defined, nor is the role of dietary cation-anion difference. Changes in dietary cation-anion difference might affect both ruminal acid production and the ability of the animal to compensate for a metabolic acid load. Further research with buffers and effects of dietary cation-anion difference is needed(Galyean & Rivera, 2003).

Acidosis Signs

Effects of acidosis on cattle may include slowing or stopping of gut movement (rumen stasis), diarrhea,and dehydration. Cattle appear weak, anorexic, and uncoordinated. Manure is often soft, gray, and foamy. Nutrient absorption may be impeded after animals recoverfrom a bout of acidosis. Cattle with subacute acidosis may exhibit reduced but variable feed intake and decreased performance. Susceptibility to subacute acidosis may vary among animals. Acute acidosis can result in heart and lung failure and death. Both subacute and acute acidosis can lead to rumenitis (infection of the rumen wall). The low pH from acidosis creates lesions in the rumen wall. Damage to the rumen wall from sharp objects (such as wire or nails) predisposes the animal to abscess formation. When rumenitis develops, liver abscesses often follow. Bacteria(*F. necrophorum, Actinomyces pyogenes, Bacteriodes spp*.) from the rumen that cause liver abscesses enter the blood supply through ulcerative lesions, hairs, or foreign objectsembedded in the rumen wall. These bacteria then travel via blood to the liver(States, 2005). While small ruminant signs of acute ruminal acidosis may be observed within 12 h after ingestion of concentrate, and they include lethargy, anorexia, bruxism, nasal discharge, head press-ing, ataxia, hyperpnea, recumbency, dehydration, scleral injection, muscle twitching, tachycardia, fluid abdominal sounds, reduced or absent motility of the rumen, mild colic,diarrhea and pH reduction to 5 or 6. In severe cases, shock and coma leading to death may also occur. In subacute and chronic ruminal acidosis, clinical signs include reduced or cyclic feedintake, decreased milk production, and

reduced fat, poor body condition and diarrhea. Additionally, unusually high rates of culling or unexplained deaths may be noted in the flock.Diarrhea is inconsistently seen. Laminitis is a traditionally suggested sequel of all forms of rumen acidosis, however, the pathogenesis for acidosis and laminitis hasbeen recently disputed in cattle, since there seems to be lack of clear evidence to support the classic laminitis hypothesis. Further research into the mechanistic basis of the association between ruminal acidosis and laminitis in sheep is necessary. The gross findings of acute ruminal acidosis are not specific and are characterized bysigns of dehydration and hypoxia, including sunken eyes, dense and dark blood, and general venous congestion. Soon after the ingestion of large amounts of carbohydrates, the rumen content has a porridge-like appearance with a distinct fermentative odor. There may or may not be a large amount of grain or other sources of starch, but care should be taken not to overlook finely ground concentrate. In sub-acute or chronic cases of acidosis, the ruminal content may appear more or less normal, but the intestinal contents remain watery. There may be a poorly defined slight blue discoloration in the ventral sac of the rumen and reticulum and in the omasum, visible through the serosa. When the epithelium is detached, the lamina propria is patchy hyperemic. In some cases, the epithelium appears to have undergone fixation due to low pH, and is difficult to peel. Patches of flat, white ruminal mucosa devoid of papillae, and multiple abscesses or foci of necrosis may be evident in the liver.

Interpretation of the post-mortem rumen pH may be challenging. Some authors consider that because ruminal fermentation continues after death, the pH of rumen contents declines postmortem(Asín et al., 2021). Microscopic changes are usually absent in the rumen of animals dying of acute rumen acidosis, unless pre-existing subacute lesions were present before the onset of acute disease. Microscopic examination of the rumen in cases of sub-acute or chronic acidosis reveals lesions consistent with chemical rumenitis, including enlarged ruminal papillae, cytoplasmic vacuolation of the epithelial cells often

leading to vesiculation, and a mild for the diagnosis of acidosis in the live animal, measurement of the rumen fluid pHis helpful. Collection of rumen fluid may be performed by rumenocentesis or by aspiration through a tube. A pH of 5.5 or less is strongly suggestive of rumen acidosis. However, animals accustomed to a high grain ration may have rumen pH close to 5. On microscopic examination of rumen fluid, the numbers of protozoa are significantly reduced or absent, and Gram-positive bacteria predominate.

Management of acidosis

When cattle are exposed to a high-concentrate diet too quickly(States, 2005), acidosis may result. Fluctuations in eating behavior are often observed. To reduce the incidence of acidosis, use a warm-up feeding period, introducing high-concentrate feeds gradually over three to four weeks. Keep at least 10 percent roughage in the final diet to helpmoderate rumen pH. Forages and cottonseed hulls are both good roughage sources high in effective fiber. While acidosis most often occurs during adaptationto concentrate-rich diets, chronic acidosis may continue into the feeding period. Feeding a combination of grains orfeeding a dry grain with a high-moisture grain can reduce the risk of acidosis. Potential for acidosis is higher when feeding wheat than corn. Processing grains less thoroughly and limiting the quantity of feed offered should reduce acidosis incidencebut may also lower animal performance. Good bunk management where all feed is consumed before the next feeding may lessen daily fluctuations in feed intake and reduce acidosis risk. Feeding ionophores (monensin or lasalocid) can help to reduce the incidence of acidosis. Ionophores may reduce intake and help moderate concentrate intake when calvesbegan on higher concentrate diets. Adding bicarbonate, fat(up to 8 percent of the diet), probiotics, virginiamycin, or thiamin to the diet or increasing protein in the diet may decrease acidosis risk(Mcguffey et al., 2001).

liver abscesses

Liver abscesses are a common disorder in feedlot cattle in the United States, with occurrence rates ranging from 0 to nearly 100%. Liver abscesses accounted for 46%

of liver condemnations in the United States, which was more than double the rate for liver flukes (22%), the second highest cause of condemnation. (States, 2005) indicated that 14% of livers were condemned in Canadian processing plants, 14% were approved for use in pet food, and 72% were approved for human consumption. Liver abscesses were responsible for the majority. The occurrence of liver abscesses is generally considered to be closely related to acidosis because it is thought that a continual high acid load in the rumen can lead to parakeratosis or similar insults to the ruminal wall, leading, in turn, to clumping and necrosis of ruminal papillae. These ulcerative lesions, along with hairs or other foreign objects that become embedded in the ruminal epithelium provide routes of entry into the portal system for microbes that cause liver abscesses; however, the exact mechanism of pathogenesis is unknown(Galyean & Rivera, 2003). The incidence of liver abscesses can be high in certain geographical regions of the United States, especially the Midwest feeding belt and northern Great Plains region. Affected livers are condemned at slaughter, which represents a loss of carcass weight of approximately 2%. Sheep with severely abscessed livers (one or more large, active abscesses) have a lower BW gain and are less efficient than cattle with no abscesses, but performance by cattle with less severely abscessed livers (but nonetheless condemned at slaughter) is not affected greatly(Asín et al., 2021).

Controlling Liver Abscesses

Liver abscesses are visible at slaughter, and as noted previously, only severe abscesses affect animal performance; hence, pre-slaughter detection of feedlot cattle with abscesses is virtually impossible. Ultrasonography has proven useful for monitoring experimentally induced abscesses, but its value in practice has been deemed negligible. Because detection of liver abscesses in the live animal is impractical, prevention is the only feasible approach(Galyean & Rivera, 2003).

Hardware Disease

Hardware disease is the common name for traumatic gastritis and traumatic reticulitis. It may occur when cattle consume sharp, heavy objects, such as nails or wire. These objects fall to the rumen floor and are swept into the reticulum by muscle

contractions. Cattle may ingest these objects and never have hardware disease, muscle contractions may cause these sharp objects to puncture the reticulum wall, diaphragm, and heart sac. Forceful abdominal movement during calving may force a sharp object through the reticulum wall. This leads to severe damage to and infection of the abdominal cavity, heart sac, or lungs(Gadberry & Powell, n.d.).

Signs of hardware disease

Signs of hardware disease vary, depending on where the puncture occurs. Loss of appetite, depression,reluctance to move, arched back, and indications of painare common signs. The animal may grunt when forced to walk. Recurring bloat may be noticed on the upper left side, with fluid accumulation on the lower right. If the heart sac has been punctured, fluid may accumulatearound the heart and in the brisket. Fatal infection can occur if the object penetrates close to the heart(States, 2005).

Management of hardware disease

Manage cattle so they cannot ingest heavy, sharp objects. Keep pastures, paddocks, and feed bunks free of wire, nails, fencing staples, and other sharp objects (even heavy plastic items) that could be swallowed. Debris from structures and equipment may appear in areas where cattle graze after high winds(States, 2005). Deteriorating steel-belted tires also pose a risk. Place magnets on feeding equipment to catch some ofthe metal objects in feed. An intraruminal magnet can be inserted into the rumen to trap metal fragments. Ingestedmetal is drawn to the magnet instead of working its way through the stomach wall. The magnet eventually “fills. up” if enough metal is ingested. Administer a second magnet if signs of hardware disease persist. Magnets are relatively inexpensive, particularly when compared to thecost of surgery. It is often difficult to diagnose hardware disease. Seek veterinary advice for suspect cases. Administer an intraruminal magnet when hardware disease cannot be ruled out. Confining cattle and limiting feed intake may allow puncture sites to heal in less serious cases. If infectionis suspected, administer a broad-spectrum antibiotic. The severity of infection and how long the condition has been present affect the animal’s treatment outlook. Cattle with extensive infection in the heart or abdomen havea very poor prognosis. Even with attempts to encourage

feed consumption, these animals often die of starvation. Sometimes only surgical removal of the object works. Controlling infection is important after the object is removedfor successful recovery. Surgery is often not a cost-effectiveoption unless the cattle affected are very valuable(States, 2005).

Mineral deficiency

This section enquires into different mineral/trace element deficiencies that may contribute to states of ill-thrift and disorder. The most common symptoms of mineral deficiency are stunted growth and discolored skin. The symptoms of a mineral deficiency depend partly on the function of that nutrient in the feed, For example, poor production performance, mastitis, susceptible to disease, loss of appetite, low feed intake and loss body weight, Irritability, tetany-increased excitability and reduced reproductive efficiency. The relationship between a mineral deficiency and its symptoms is less distinct. Indices to evaluate the risk of deficiency disorder have been reviewed for some of these mineral, although these values may vary depending on the geographical location, diet, method of measurement(Asín et al., 2021)etc.

Copper deficiency

Copper (Cu) is an essential component of many enzymes, and it is vital for a proper development and maintenance of several organ systems, such nervous, integumentary, skeletal, hematopoietic, immune, and digestive systems. Cu deficiency in sheep may be primary, due to decreased intake, or secondary, due to altered absorption, reduced tissue availability, or enhanced excretion. In rumi- nants, Cu has a complex array of interactions with dietary molybdenum (Mb) and sulfur that, when present in excess, will lead to production of molybdates and thiomolybdates(Asín et al., 2021), which bind Cu and decrease its absorption and utilization. Other elements, such as zinc (Zn), iron, cadmium, and lead may act as Cu-antagonists and interfere with its bioavailability. Gastrointestinal parasites are another cause of conditioned hypocuprosis. The pathogenesis of Cu deficiency is related to decreased activity of many Cu-de- pendent enzymes in several tissues. Cu is a component of several enzyme systems that are essential for neural function and, therefore, lesions

are characterized by pri- mary neuroaxonal degeneration with secondary myelin loss. Impaired function of mitochondrial cytochrome C oxidase, superoxide dismutase (SOD), and ceruloplasmin have been proposed as the most important molecular bases of the disease, since there is suppression of mitochondrial respiration and phospholipid synthesis, with possible neu- ronal damage by superoxide radicals. Loss of embryonic cells during brain devel-opment has also been involved in the pathogenesis of congenital cerebral cavitation. In addition to nervous tissue, many other systems may be affected by Cu deficiency.

Poor wool quality and hypopigmentation are caused by imperfect oxidation of sulfhydryl groups in prekeratin, and by decreased melanogenesis due to depressed tyrosinase activity, respectively. Impaired production of ceruloplasmin and hephaestin reduce gas- trointestinal iron absorption, which leads to decreased hemoglobin synthesis, altered mat- uration of erythroid precursors, and eventually anemia after prolonged periods. Moreover, increased oxidative stress due to Cu depletion in erythrocytes and muscle fi- bers can lead to hemolysis secondary to Heinz body formation, as well as to muscle necrosis. Decreased functions of Cu-dependent enzymes on intestinal mucosa interfere with digestion, motility, and inflammatory responses to parasites, which might account for growth retardation and increased susceptibility to gastrointestinal parasites

Cobalt

A regular dietary cobalt (Co) supply is essential for survival, health, and efficient production. Sheep are particularly susceptible to Co deficiency due to their high S-aminoacid requirements for wool growth(Spears, 2018). Co deficiency is of economic importance in weaning lambs, as it causes an ill-thrift syndrome, clinically indistinguishable from that caused by starvation, occasionally associated with hepatic disease, and known as ovine white liver disease. The major cause of Co deficiency in sheep is inadequate dietary supply. Clinico-pathologic consequences derive from insufficient vitamin B12 synthesis by ruminal microorganisms, leading to decreased tissue availability of its coenzymes, methylcobalamin and deoxyadenosylcobalamin, which assist methionine synthase and methylmalonyl coenzyme Amutase, respectively. Methionine synthase is a methyl

transferase involved in the synthesis of methioninefrom homocysteine, an essential amino acid with important roles in DNA and protein synthesis. Reduced methionine availability causes ineffective production of cells, particularly those with rapid turnover and, consequently, impaired normal growth and development. Moreover, homocysteine accumulation increases lipid peroxidation in the liver, which is considered an important mechanism involved in the pathogenesis. Methylmalonyl coenzyme A mutase performs a key role in the intermediary energy metabolism of ruminants, facilitating the metabolism of propionate via succinate, which serves for gluconeogenesis. Metabolic abnormalities resulting from alterations of this enzymatic pathway and accumulation of intermediary metabolites in plasma, such as methylmalonic acid and propionate, are important in the pathogenesis and other alterations related to Co deficiency. Methylmalonic acid becomes incor-porated into branched-chain fatty acids, which may alter lipid composition in the liver, and also inhibits the oxidation of fatty acids mobilized to supply the energy deficit causedby anorexia in affected animals(Smart & Christensen, 1982). Poor reproductive performance in ewes has been attributed to impaired gluconeogenesis in addition to generalized poor body condition. Increased propionate levels in the portal blood flow induce powerful satiety sig- nals in sheep and has been suggested as a cause of anorexia in Co-depleted lambs,but this hypothesis in considered unlikely by others.

Clinical signs of Co deficiency are most commonly observed in weaned lambs at pasture during late summer/autumn. Affected animals show lethargy, reduced appetite, growth retardation, poor wool quality, small size, and poor body condition despite adequate nutrition. Lachrymation and wool alterations might be the earliest visible abnormalities. In chronic stages, animals develop anemia with pallor of the mucous membranes, and progressive anorexia with marked weight loss, emaciation, muscular wasting, pica and decreased wool growth; without intervention, death ensues. Additionally, clinical signs related to hepatic dysfunction such as icterus, photosensitization, or neurological signs secondary to hepatic encephalopathy might be present in animals affected. An increased susceptibilityto gastrointestinal parasitic

infections has been also reported in Co deficient lambs. In adult ewes, Co deficiency during pregnancy may cause infertility, poor mothering, and a reduction of the viability of the offspring(Asín et al., 2021)

Selenium

Selenium (Se) is an essential element that interferes through selenoproteins (SePs) in many physiological processes of livestock(Spears, 2018). In many instances, Se deficiency must be addressed with vitamin E deficiency. Se and/or vitamin E deficiency in sheep are well- known causes of nutritional myopathy or white muscle disease, but subclinical forms with ill-thrift and reproductive issues may be the most important manifestations of Se deficiency in economic terms. The major cause of Se and/or vitamin E deficiency is inadequate dietary supply. Se deficiency occurs in many regions, particularly in those with soils of granitic or volcanic origin, and in areas of heavy rainfall. It can also be associated with intensive farming practices. Vitamin E deficiency is independent of soil type. Green pasturesand fresh legumes are good sources; therefore, vitamin E deficiency is widespread in weaner sheep flocks over long, dry summer and autumn, when animals are fed on dry feed, hay, and grain with little or no access to green pastures for long periods. Prolonged storage of feedstuffs also causes important loss of vitamin E content. Pathophysiological consequences of Se deficiency derive from dysfunction of many SePs with important roles in antioxidant defense, cell signaling and transcription, metab-olism of thyroid hormones, and in immune and reproductive systems. Se and vitamin E play complementary but independent functions as cellular antioxidants, which protect cells from injury by reactive oxygen species generated during normal oxidative cellular metabolism. Se is a component of glutathione peroxidase, a family of selenoen- zymes that inactivates peroxides thus preventing membrane damage; vitamin E acts as free radical scavenger, providing protection against peroxidation of polyunsaturated fatty acids(Spears, 2018). Failure of antioxidant mechanisms caused by Se/vitamin E deficiency leads to intracellular accumulation of free radicals with subsequent membrane damage and intracellular influx of calcium (Ca^{2+}); this is first moved from the cytosol to the mitochondria, causing damage in the latter, and ATP depletion. Eventually, raised

Ca^{2+} cytosolic levels result in myofibril hypercontraction and cellular necrosis with secondary dys-trophic mineralization. Cells or tissues that undergo rapid increases in oxidativemetabolism, such as striated muscle and red blood cells, are particularly susceptible to theoxidative injury that underlies NM, Heinz-body anemia, and reproductive failure relatedto Se/vitamin E deficiency in sheep. In addition to oxidative damage, dysfunc- tion of a series of deiodinases involved in the synthesis of thyroid hormones are also im- portant contributors to growth retardation and infertility problems in Se-depleted lambs and sheep.

The most classical clinical manifestation of Se/vitamin E deficiency in sheep is NM, although many cases are related to Se deficiency, only. The incidence may vary from 1 to 30% depending on the geographical region. It primarily affects young lambs andpresents as two major clinical forms, which can develop in- or ex-utero. The congenital form is rare, but may be associated with high mortality; stillbirths or extremely weak neonates occur, and the latter usually die within a few days, often from acute cardiac arrest. Delayed or acquired forms are usually encountered in 2–6-week-old lambs that have recently turned out onto the first green pasture, but they can also occur in younger or older animals. Mortality rates in outbreaks among lambs from one day to two months may be low or reach 50%. A second peak of incidence, frequently with lower mortality but moderately higher incidence of subclinical or minimal forms occurs at 4–8 months of age, when weaned lambs are put onto lush pastures or feedlots(Asín et al., 2021).

Zink Deficiency

Zn is an essential nutrient and plays an important role in many metalloenzymes thatregulate metabolic processes, tissue growth, maturation, and repair(Smart & Christensen, 1982). Zn also modulates many aspects of the immune and inflammatory responses because of its structuralrole in the vast majority of transcription factors involved in immune system development. Features of Zn deficiency include loss of appetite and growth retardation, reproduc-tive disorders, depression of the immune response, hematologic abnormalities, impair- ment of central nervous system development, decreased wound healing, alopecia and keratinization defects in

epidermis, hair, wool, and horny appendages. Loss of appetite is an early sign of Zn deprivation and there are changes in the pat- terns of food intake from ‘meal-eating’ to ‘nibbling’ in ruminants. Zn deprivation in-creases the expression of the gene that encodes for the appetite-regulating hormone cholecystokinin, which induces satiety, there is also increased expression of leptin, a cytokine hormone whose secretion from adipocytes also promotes a satiety signal. This reduction in appetite is selective, with carbohydrates being avoided and proteins and fatspreferred. Zn deprivation in pregnant ewes does not cause congenital malformations, although the number of born lambs and their weights may be reduced due to restricted fetal growth. Diets very low in Zn during pregnancy reduce survival of the newborn lamb, and pregnancy toxemia may occur as a secondary consequence of anorexia in the ewe. Testicular hypoplasia occurs experimentally in Zn-deprived male lambs. In that ex- periment, spermatogenesis almost entirely ceased within 20 weeks on a diet containing very low amounts of Zn, but recovered completely during a repletion period. Zn has many roles in immunity and disease resistance as well. In a study, there was a reduction in the percentage of lymphocytes in the peripheral blood of lambs fed with a Zn-deficient diet. Animals with hereditary Zn deficiency have a hypoplastic thymus and, consequently, secondary infections are common because of associated immune sys- tem dysfunction involving both humoral and cell-mediated immunity. Thickening, hardening, and fissuring of the skin due to hyperkeratosis is a late sign of Zn deprivation in all species. In lambs, frequently affected sites include periocular areas, nose, coronary band, scrotum, and pressure points. Lesions can also affect strat-ified epithelia lining the tongue and esophagus, and are similar to those induced by vita-min A deficiency. Hyperkeratosis also affects the forestomachs. The rate of heal-ing of skin wounds is retarded in Zn-deprived animals, and wounds caused by ectopara-sites or other skin infections will probably exacerbate the effects of hyperkeratosis. In horned lambs, the normal ring structure disappears from new horn growth, and the horns are ultimately shed, leaving soft spongy outgrowths that bleed continuously. Ab- normal hoof growth may lead to soreness and the adoption of a kyphotic stance. Sheep also have thin, easily epilated, redbrown colored wool and

loss of fleece. Wool eat- ing and drooling are prominent clinical signs. The characteristic histologic lesion is severe and diffuse hyperkeratosis. In sheep with naturally occurring Zn deficiency, the hyperkeratosis is predominantly of the orthokeratotic rather than the parakeratotic type. In addition, skin from Zn-deficient sheep contains apoptotic bodies in follicle bulbs and the wool fibers are improperly keratinized as indicated by retained cell nuclei and fiber distortion in the distal parts of some follicles.

Iodine

Iodine (I) has only one known, though vital, function since it is an essential constituent of thyroid hormones. Thyroid hormones have a thermoregulatory role, increasing cellular respiration and energy production, as well as widespread effects on intermediary metabolism, growth, muscle function, immune defense, and circulation(Asín et al., 2021). These are particularly important in facilitating the change from fetal to a free-living stage. Seasonality of reproduction in ewes is related to changes in thyroid activity. Iodine deficiency disorder (IDD) may arise from simple dietary lack or due to low content in the soil. IDD is also induced by exposure to goitrogens contained in brassicas, white clover, or legume crops. These act by disrupting I metabolism, either by impairing its uptake by the thyroid gland, or by promoting iodination of tyrosine residues. However, widespread addition of iodized salt to animal diets prevents most of the outbreaks nowadays, and fewer animals tend to be affected if they occur. Goiter (enlarged thyroid glands) is a clinical manifestation of IDD(Asín et al., 2021). Goiter is manifested predominantly in the newborn animal, which is usually delivered by a clinically normal dam. In sheep, insufficient I intake by pregnant ewes and subsequent fetal hypothyroidism results in late term abortions, increased perinatal mortality, and birth of weaker lambs with visibly enlarged thyroids and delayed skin and wool follicle development . Delayed bone growth and maturation may also occur if the ovinefetus is exposed to I deficiency up to the last trimester of gestation. Gestation is oftenprolonged, and most severe cases of thyroid enlargement may cause dystocia, with a tendency to retain the fetal placenta. The degree of thyroid enlargement increases with the level and duration of I deprivation, Size tends to be indicative of compensatory at- tempts for insufficient thyroid hormone production.

Asphyxiation may also result from pressure by the enlarged gland on the trachea and adjacent structures. RetardedCNS development has been observed in severely depleted fetuses, and behavioral abnor malities may thus ensure after birth(Asín et al., 2021).

Conclusion

Knowledgeable of beef cattle producers can reduce or eliminate risk factors for bloat, acidosis, and hardware disease. Watch cattle for signs of nutritional disorders to facilitate early intervention and treatment. Seek veterinaryassistance when developing and implementing treatment programs for suspect cases of nutritional disorders. Correct nutritional management is a key factor to prevent disorder in ruminant. Feed has to be offered not only in enough amounts, but also with a qualitative composition that ensures an appropriate intake of all the necessary minerals, vitamins, and other micronutrients. In addition to emaciation and loss of performance, some mineral deficiencies produce lesions that may facilitate a tentative diagnosis. Such diagnosis must be confirmed by demonstrating low levels of the suspected imbalanced nutrient in organs or body fluids. Restituting these depleted levels is the therapeuticapproach to take in most cases, either by selecting feeds with optimal amounts of such nutrient/s or by using supplements and mineral correctors.

Recommendation

We would like to suggest that ruminant herd farmers should communicate veterinary technicals when they suspect heavy nutritional disorder in their cattle and sheep.

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