Int.J.Curr.Microbiol.App.Sci (2021) *10*(03): 447-459

International Journal of Current Microbiology and Applied Sciences
***ISSN: 2319-7706* Volume 10 Number 03 (2021)**
Journal homepage: http://www.ijcmas.com

Review Article https://doi.org/10.20546/ijcmas.2021.1003.060

Disease Developed in Ruminant Animals due to Faulty Feeding

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Keywords

Ruminant animals, Faulty feeding

Article Info

Accepted:
07 February 2021
Available Online:
10 March 2021

A B S T R A C T

Ruminants are herbivorous, forgot fermentors. They are able to acquire nutrients from plant based foods by fermenting it in a specialized stomach having four-compartment stomach. Any sudden change in feeding pattern or an improper feeding regimen is bound to disturb the balance of the various types of microbes, leading to an over population of certain unfavorable microbes and changes in rumen environment that may cause severe problem in animals.

Introduction

Ruminants are herbivorous mammals that are able to acquire nutrients from plant-based food by fermenting it in a specialized stomach having four-compartment stomach, which includes, the rumen (paunch), the reticulum ("honeycomb"), the omasum ("manyplies") and the abomasum ("true stomach"). The digestive process is based on the action of various types of microbes which break down the feed and provide nutrients to the animal (Hungate, 1966). The process, which takes place in the front part of the digestive system and therefore is called foregut fermentation typically requires the fermented ingesta (known as cud) to be regurgitated and chewed again. The process of rechewing the cud to further break down plant matter and stimulate digestion is called rumination.

Any sudden change in feeding pattern or an improper feeding regimen is bound to disturb the balance of the various types of microbes, leading to an over population of certain unfavorable microbes and changes in rumen environment that may cause various problems like laminitis or even death in severe cases (acute acidosis). Such conditions/disease may also arise from interference in the process of natural gas expulsion produced as a by-product of microbial digestion. It is important to understand the common conditions/disease so that timely advice or treatment can be sought from a veterinarian. The following diseases are described under this section:

Bloat
Rumen acidosis
Laminitis
Pica
Hardware Disease
Acute mycotoxicosis
Nitrate Poisoning
Prussic Acid (Hydrocyanic Acid) Poisoning
Acute Bracken Poisoning

Bloat

Bloat is over distension of the rumen-reticulum with gases of fermentation (Cole and Boda, 1960; Davis *et al.,* 1965; Rick, 2010; Bowen, 1996; Majak, 2003). Bloat is a form of indigestion marked by excessive accumulation of gas in the rumen. Bloat can occur when the animal grazes on lush young pasture, particularly if the pasture is wet. Some plants, e.g. clover, Lucerne and alfalfa are especially dangerous in causing bloat but any fast growing plants can cause it. When the animal grazes on lush young pasture, particularly if the pasture is wet. Some plants, e.g. clover, lucerne and alfalfa are especially dangerous in causing bloat. Choking due to foreign objects (esophageal obstruction) will also cause bloat by preventing gas release and causing accumulation of gas in the rumen.

Types (on basis of forms of gas)

Primary or frothy bloat/nutritional.
Secondary or free-gas bloat

Plants factors: Forage species and their potential for causing bloat in cattle

High Potential	Low Potential	Considered Safe
Alfalfa	Arrow leaf clover	Birds foot trefoil
Sweet clover	Spring wheat	Cicermilkvetch
Red clover	Oats	Crownvetch
White clover	Rape	Lespedeza
Alsike clover	Perennial ryegrass	Fall rye
Winter wheat	Berseem clover	Most grasses

(Aiello, S.E., and Moses, M.A. 2016)

Animal factor

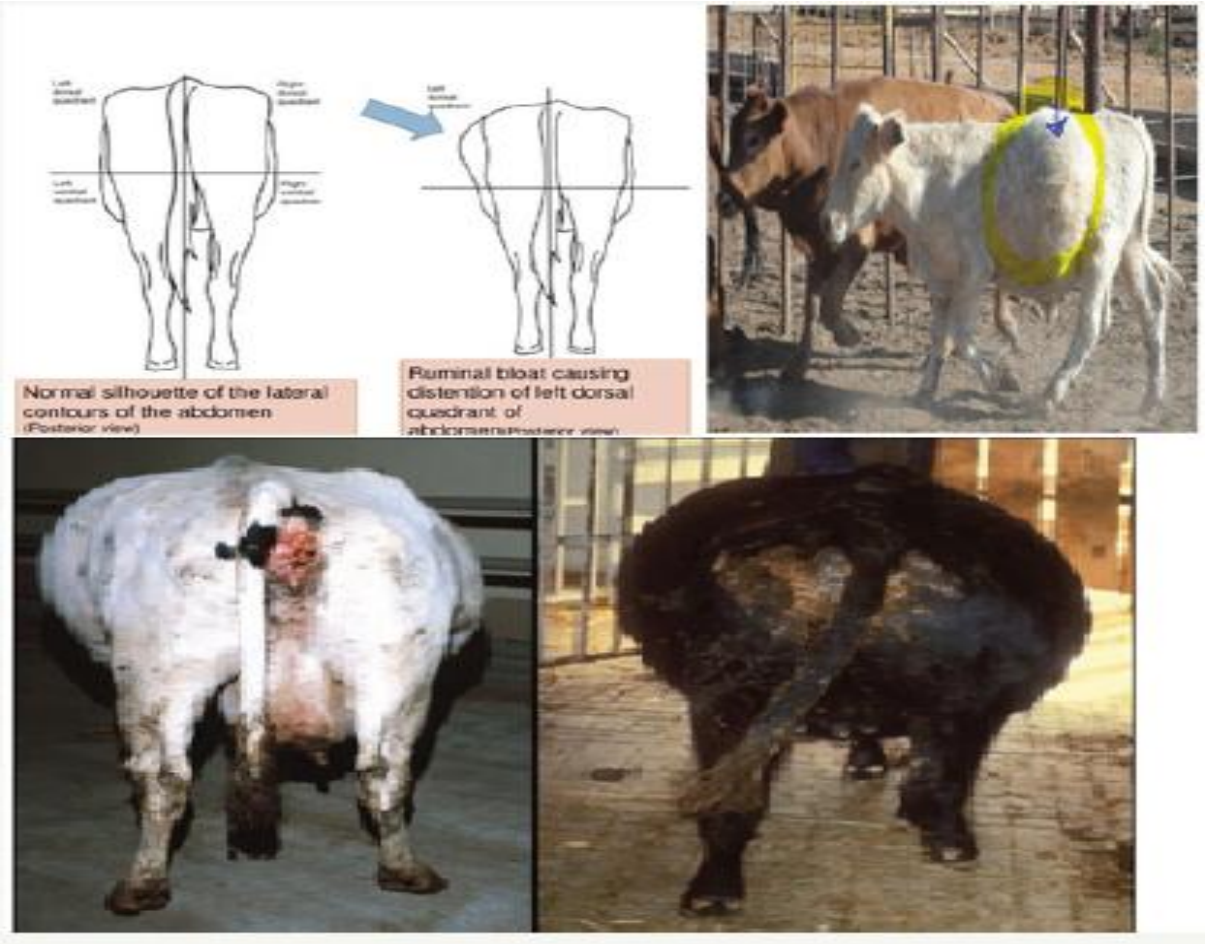
It is fairly well established that young animals are more susceptible to acute and severe bloat than older animals, and it is suspected that animals get used to eating bloating pastures and are less susceptible after exposure. Variation in incidence and severity of bloat between animals grazing the same pasture may be partially related to animal factors such as differences in diet selection, forage intake and saliva production (Rutter *et al.,* 2004).

Symptoms of bloat

Clinical sign, which observed in pasture (or frothy) bloat, is the distention of abdomen. Distension is usually more obvious in the upper left flank, although the whole of the rumen can be enlarged. Some clinical sign is

observed like getting up and lying down frequently, defecate frequently, kick at the abdomen and rolling over in attempting to relieve the discomfort.

Breathing is difficult or labored (a condition known as dyspnea) and occurs through the mouth. The animal protrudes the tongue, salivates, and extends the head. Its respiratory rate increases to up to 60 inhalation-exhalation cycles per minute.



Ruminal movements are usually much increased in the early stages and may be almost continuous, but the sounds are less audible because of the frothy nature of the ingesta. Later, when the distension is extreme, the movements are decreased and may be completely absent. The tympanic note or drum sound produced by percussion (tapping on the distended rumen) is characteristic. Before severe bloat (known as clinical tympany) occurs, a temporary increase in eructation and rumination can be noted, but both disappear with severe bloat.

Death may occur quickly, but usually does not take place until 2 to 4 hours after the onset of bloat. When the bloat becomes severe enough, the animal collapses and dies quickly, almost without a struggle. Death is likely caused by suffocation, when the distended rumen pushes against the diaphragm and prevents inhalation (Radostits *et al.*, 2010).

Diagnosis

On the clinical symptom

History of access to lush pasture
Passing a stomach tube will distinguish between gassy and frothy bloat. If it's gassy bloat a stomach tube passed into the rumen will allow the gas build-up to escape through the tube. No such gas is seen in frothy bloat.

The best method in diagnosis of bloat animals is using general clinical examination like auscultation and palpation (Abdisa, 2017).

Prevention and treatment

Avoid moving animals to wet pasture in the morning (Majak *et al.,* 2003; Rick *et al.,* 2010; Radostits *et al.,* 2010).

Do not allow very hungry animals to graze a pasture.
Offer dry, cut grass first before turning out to graze.

Home remedies may be used in mild cases.

In severe cases, puncturing the left flank with a sharp knife to release the gas is necessary, it will be necessary for you to act quickly as any hesitation could lead to the death of the animal.

A variety of antifoaming agents are effective, including vegetable oils (e.g, peanut, corn, soybean) and mineral oils (paraffins), at doses of 250-500mL (Radostits *et al.,* 2010).

Feeding the ionophores monensin or lasalocid to the animal (Radostits *et al.,* 2010)

Poloxalene (25-50g, PO) is effective in treating legume bloat but not feedlot bloat (Radostits *et al.,* 2010).

Placement of a rumen fistula provides short-term relief for cases of free-gas bloat associated with external obstruction of the esophagus (Aiello and Moses, 2016; Radostits *et al.,* 2010).

Home remedies for adult animals

Drench coconut / vegetable / peanut oil: 300-500ml once a day for 2-3 days till recovery. Or; Above plus 30-40 ml turpentine oil.

Drench 1 tablespoon of detergent in half liter of water once. Or;

Feed 4-6 banana leaves (mild bloat)

Rumen acidosis

Acidosis may be clinical or sub- clinical.

Feeding large quantities of rapidly digestible carbohydrate (concentrate) in a short period of time and amount of forage or roughage in the diet decreases causes clinical acidosis and takes a huge toll of profit from dairy enterprise often unnoticed by farmer. It costs double loss to farmer as productivity and health of dairy animal is compromised along with feed loss as dairy farmer is feeding costly grain ration of which considerable amount passes undigested in feces (Kleen *et al.,* 2003).

Ruminal acidosis when occurs in chronic form is called as Sub-acute ruminal acidosis (SARA).

Sub-acute or sub-clinical ruminal acidosis (SARA) is considered to be one of the major threats to the welfare of lactating dairy cows and may affect up to 20% of cattle in early to mid lactation. (Rushen *et al.,* 2008)

Subclinical rumen acidosis is defined as a condition where rumen fluid pH is below 6 while acute rumen acidosis is when rumen pH is below 5.5 associated with rumen motility that is weak or ceased (Plaizier *et al.,* 2009).

Low pH supports growth of lactic acid-producing bacteria. Lactic acid is very strong and reduces rumen pH even more.

Low rumen pH can cause rumenitis, metabolic acidosis, lameness, hepatic abscesses formation, pneumonia, and even death. (Bramley *et al.*, 2008).

Aetiology

Accidental ingestion of excessive carbohydrate containing foods (rice, wheat, oats, barley).
Feeding of decomposed silage, Overnight kept boiled rice
Excessive feeding of left over kitchen garbage.
Sudden change in quality and quantity of concentrate feeds.

Clinical signs

Effects of acidosis on cattle may include slowing or stopping of gut movement (rumen stasis), diarrhea, and dehydration.

Animals may be recumbent within 24-48 hours, which may resemble cases of milk fever. One of the way for field diagnosis of SARA is to see the hair around the skin of rectum i.e. perianal region, hairless shiny skin will indicate SARA due to chronic acid damage to skin. (Bramley E. *et al.*, 2008).
Cattle appear weak, anorexia within 8-12 hr of feeding. Manure is often soft, gray, and foamy containing gas bubbles and appearance of undigested grain, mild diarrhoea, reduced rumination (cud-chewing). (Owens *et al.*, 1996).

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 objects embedded in the rumen wall. These bacteria then travel via blood to the liver. Liver abscesses are most often seen in feedlot cattle. Severe liver abscesses may reduce feed intake, weight gain, feed efficiency, and carcass yield.

Stages of Acidosis, Rumenitis, and Liver Abscess Development

rapidly fermentable grain fed to cattle
↓
acidosis (low rumen pH)
↓
gut lesions
↓
Fusobacterium necrophorum bacteria populations increase
↓
rumen wall abscesses, inflammation, and tissue death
↓
bacteria travel to liver via blood
↓
liver abscesses

Treatment

Selection of treatment for acidosis depends on the severity of the clinical symptoms.
Mild signs: diarrhoea, they should be fed a diet with smaller proportion of grain or increased amount of roughage.
Cows with more severe signs such as severe diarrhea, off feed, and in a depressed state should be removed from the grain diet and fed roughage only.
Cows should be given orally sodium bicarbonate and feeding of ionophores (monensin or lasalocid) can help reduce the incidence of acidosis. (Stone W.C. 2004; Goff J.P., 2006).
Administration of Anti histamines and cortical steroids I/M for each of several days to help prevent intoxication and laminitis.

Prevention

Never provide the animal with large quantities of rapidly digestible carbohydrates. (Owens, 1996; Stone, 2004; Goff, 2006).
Animal should be provided a roughage diet before feeding concentrates or immediately after feeding grain/molasses so that an adequate quantity of saliva is produced. (Stone, 2004; Goff, 2006).
Adding probiotics to the diet may decrease acidosis risk in animals.

Laminitis

Commonly seen in mature dairy cows during peak lactation, often a herd issue.
Higher concentrate/protein, low forage, mastitis, metritis, acidosis etc predispose. (Cook *et al.*, 2004).
Laminitis is a pathophysiologic disturbance of the microstructure of the dermis or corium of the claw. Occurs in sub-clinical, acute and chronic form.
Damage of the ruminal epithelium induced by acidosis allows absorption of histamine and endotoxins into the blood which comes from the lysis of gram negative bacteria in rumen due to low pH. Histamine and endotoxins cause vasoconstriction and inflammation of lamella of hoof causing laminitis (Stone 2004).
Cows fed the higher level of crude protein may have increased incidence and duration of lameness (Baird *et al.*, 2009).
It is considered that products of degradation of protein excess in the rumen may be the causative agents for lameness. It has also been shown that nutritional supplements such as biotin and zinc can help reduce lameness through improving claw horn quality (Baird *et al.*, 2009).

Prevention

Prevented by feeding for stable rumen conditions i.e. addition of buffers, feeding of good quality roughages, etc. (Stone, 2004).
Foot baths containing 2–5% copper sulphate particularly in wet conditions is beneficial for lame animals. (Stone, 2004)
Nutritional supplements like biotin and zinc (Zn) can help reduce lameness through improving claw horn quality. Other vitamins like vitamin A and vitamin E also may play important roles in maintaining claw integrity (Cook, 2004; Baird *et al.*, 2009).

Pica

In cows at unusual behaviors, including eating and licking stones, soil, plastic objects and cow track surfaces. This unusual behavior can be defined as ‘pica’.
Three most common causes of pica include; phosphorous and sodium deficiency or a shortage of fiber in the diet.
Symptoms: Stiffness in gait and fractures also common.
Treatment: Mineral mixture in diet and including wheat bran & bone meal in the ration that are rich in phosphorous.

Hardware disease

Common name: traumatic gastritis and traumatic reticulitis.
Common in cattle fed on prepared feeds, especially those fed inside for part of the year. It is almost un known in cattle fed entirely on pasture. Much more common in winter months in the northern hemisphare.

Fencing wire which has passed through a chaff cutter, feed chopper or forage harvester is one of the common causes


Cattle may ingest these objects and never have hardware disease, or muscle contractions may cause these sharp objects to puncture the reticulum wall, diaphragm, and heart sac. This leads to severe damage to and infection of the abdominal cavity, heart sac, or lungs.

Clinical signs

Signs of hardware disease vary, depending on where the puncture occurs. Loss of appetite, depression, reluctance to move, arched back, and indications of pain are 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 accumulate around the heart and in the brisket. Fatal infection can occur if the object penetrates close to the heart.

Management guidelines

Restrict 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.” only surgical removal of the object works.
Controlling infection is important after the object is removed for successful recovery.

Acute mycotoxicosis

Ingestion of mycotoxins by cattle from a variety of feedstuffs such as maize infected with fungal /moulds.

Under certain conditions some fungi will produce potentially harmful toxins.
The condition usually affects adult cattle.
These include neurological conditions, hemorrhagic syndromes and fever with dermatitis.

Aflatoxins

The most widespread and the most studied group of all the mycotoxins,.
Aflatoxins are primarily produced by fungi of

the genus *Aspergillus* (*Aspergillus flavus, Aspergillus parasiticus, and Aspergillus nomius*), and are found in dairy feeds and human food products.

Major forms of Aflatoxins found in feeds include Aflatoxins B1, B2, G1 and G2; with Aflatoxin B1 being the most common and toxic.

Aflatoxin M1 is found in milk and milk products. (NDDB, 2019)

Deoxynivalenol (DON, Vomitoxin)

Deoxynivalenol is produced by species of mould primarily *Fusarium graminearum* and may co-occur with other mycotoxins in contaminated commodities.

DON in cattle and buffaloes has been associated with reduced feed intake and lower milk production when fed with feed containing more than 5 parts per million (ppm). (NDDB, 2019)

Zearalenone

Zearalenone is produced primarily by *Fusarium graminearum* and *Fusarium roseum*.

Responsible for reproductive disorders because of its estrogenic effect.

In dairy animals, the clinical manifestations such as udder enlargement, decreased milk yield, vaginal discharge, continuous estrus, infertility and abortions are observed when the level of zearalenone in the feed is more than 0.5 ppm. (NDDB, 2019)

Limits for Aflatoxin B1 in dairy feed: (NDDB, 2019)

COUNTRY	Aflatoxin B1 (µg/kg or ppb)
United States (FDA)	20
European Union (for milking animals)	5
European Union (for calves)	10
India (BIS)	**20**

- **Limits for other mycotoxins in dairy feed: (NDDB, 2019)**

Mycotoxins (EU Guidelines)	Limits (mg/kg or ppm)
Deoxynivalenol: Adult animals Calves	**5 2**
Zearalenone: Adults and calves	**0.5**
Fumonisins B1 and B2: Adult animals: Calves	**50 20**

Clinical sigs

Milk producers may not be able to see visual symptoms of aflatoxicosis in the animals at low level Aflatoxin in the feed. However, high concentrations of Aflatoxins and/ or prolonged duration may cause visual symptoms in dairy animals.

Feed refusal are reduced growth rate the predominant signs of chronic Aflatoxin poisoning.

In addition, listlessness, weight loss, rough hair coat and mild diarrhea may occur. The disease may also impair reproductive efficiency, including abnormal estrous cycles (too short and too long) and abortions.

Other symptoms include impaired immune system response, and increased susceptibility to diseases. (NDDB, 2019)

Diagnosis

The demonstration of mycotoxins in feedstuffs is useful in diagnosis when suspicious clinical signs are seen. (NDDB, 2019). However, often the 'toxic* feed has been consumed before sampling occurs, and the failure to demonstrate mycotoxins does not rule out the diagnosis.

Treatment: Symptomatic treatment, nutritional modifications.

Nutritional modifications

Nutritional routes for protection against mycotoxins include higher levels of methionine, selenium and vitamin supplementation of affected diets. Some plant and herbal compounds including chlorophyll derivatives are also used for protection against mycotoxins. This method is partially beneficial but not cost effective. (NDDB, 2019)

Nitrate toxicity

Nitrates are relatively nontoxic, as ruminants convert nitrate of the plants to nitrite in rumen an then to ammonia by the action of ruminal and intestinal microbes. Ammonia produced as a source of nitrogen by rumen microflora for protein synthesis. (Leng, 2008)

Animal feed, especially forage, typically contains nitrate as one form of nitrogen. In normal conditions, nitrate in feed is generally negligible.(Leng, 2008)

Factors affecting nitrate concentration in forage include forage maturity, soil conditions (e.g., moisture content), and application of fertilizers to soil. (Leng, 2008)

Depending on the reduction rate of nitrate by rumen microbes, nitrate and nitrite can accumulate in the rumen. (Jones, 1972)

Nitrate absorbed into blood through the rumen wall. Nitrate that appears in blood is not toxic, but nitrite is toxic.

A portion of nitrate in blood will be recycled back to the gut, excreted in urine, or might be incorporated in nitric oxide metabolism. (Lewicki *et al.*, 1998; Gilchrist *et al.*, 2010).

But nitrite in blood binds to red blood cells and changes the ferrous (Fe^{2+}) form of hemoglobin to the ferric (Fe^{3+}) form (methemoglobin). (Lundberg *et al.*, 2008; Bruning-Fann and Kaneene 1993; Sinderal and Milkowski 2012) while the nitrite itself is oxidized to nitrate. (Lewicki *et al.*, 1994, 1998)

Acute cases

Gasping, Rapid respiration, Salivation, vomition, diarrhoea, colorless urine, Accelerate heart rate, anoxic convulsions, death in 2-24 hrs.

Bluish is colouration of mucus membrane an chocolate brown colour blood - characteristic of nitrite poisoning (Bruning-Fann and Kaneene, 1993; Sinderal and Milkowski, 2012)

Feed intake is one of the most important factors affecting animal productivity (i.e., milk yield and weight gain), and there are many factors altering feed intake of ruminants. (NRC 1996, 2001)

Lower feed intake is one of the symptoms of mild nitrate-poisoning, which subsequently can affect productivity (Hegarty *et al.*, 2013).

Diagnosis

History

Clinical signs

Chocolate brown colour of blood and tissues

Analysis of stomach/ ruminal/ intestinal contents

Detection of nitrite in urine

Response to methylene blue treatment.

Serum nitrite levels and ocular fluid levels.
Sampling of feed or water

Treatment

Remove the source of poison.
Objective of treatment in nitrite poisoning is to reduce met-Hb to Hb by using a suitable Methylene blue (1%w/v) solution in isotonic solution is the specific antidote used and administered by slow IV injection @ 8.8mg/kg body wt. in cattle.

Prevention

Stop access of the animals to offending fields, or fodders.
Supplement the diet with chlortetracycline (30 mg /kg of feed) for two weeks to suppress gastro intestinal flora to reduce conversion of nitrate to nitrite in the rumen.

Prussic acid (Hydrocyanic Acid) poisoning

It occurs mainly in the form of cyanogenic glycoside.
In plants the Glucoside is non-toxic in the intact tissues.
These glycosides can be hydrolyzed to prussic acid or hydrocyanic acid (HCN) by the enzyme usually present in the same plant or as they are being digested by animals.(Clarke ML *et al.,* 1981)
Cyanide is taken to the body, it's rapidly absorbed and circulated, then it's merged with methemoglobin and forms cyanomethemoglobin.
The circulating cyanide inactivates cytochrome oxidase enzyme by binding ferric (Fe+++) iron which is within this enzyme the enzyme-cyanide complex prevents this task from being performed.
Because of that the enzyme can not combine with oxygen and electron transportation become inhibited.& cellular respiration stops immediately.As a result of this process, death occurs due to histotoxic anoxia. (Akgül *et al.,* 2013; Gurnsey *et al.,* 1977)

There are three distinct glycosides

Amygdalin: Almonds
Dhurrin: Jowar and other immature grasses
Linamarin: Pulses, Linseed, cassava.
The plants are considered as toxic if they contain over 200 ppm of these glycosides. (Gracia and Shepherd, 2004)
The lethal dose of HCN for cattle and sheep is about 2mg/ kg of body weight. (Hydrogen Cyanide, 1970; Gurnsey *et al.,* 1977; Clarke *et al.,* 1981; Vetter, 2000).

Treatment

Sodium nitrate and sodium thiosulfate should be rapidly administered intravenously to affected cattles. (Clarke, 1981; Vetter, 2000; Arnold and Gaskill, 2014).

Prevention

Sorghum and sudan grasses should not be grazed when they are in an immature state.
They must be allowed these forages to attain a height of 15 to 18 inches before grazing. (Nobrega JR *et al.,* (2006)
New varieties of sudan grass and sorghum with lower prussic acid content should be considered when selecting seed.
Plants must not be grazed during drought periods when growth is severely reduced or the plant is wilted or twisted.
Animals must have been provided sufficient feed, like hay, so they will not be hungry when they enter fresh pastures (Clarke, 1981; Nobrega *et al.,* 2006)

Acute bracken poisoning

Acute bracken poisoning is caused by ingestion of large quantities of bracken fern (*Pteridium aquilinum*).

Some places where bracken is cut and used as bedding material. (Sanderson, 2002)

Peak incidence is in young cattle aged between 1 and 3 years.
It is the only known higher plant that is carcinogenic in animals when ingested (Norton, 2008). Bracken fern rhizomes were used as food in times of draught. (Smith and Seawright, 1995; Alonso-Amelot and Avendaño, 2002).

Clinical signs

Haemorrhage, dullness and fever. intermittent hematuria and anemia (Anjos *et al.*, 2008)

Enzootic hematuria, the most common form of bracken fern poisoning, primarily affects cattle and less frequently affects sheep. (Shahin *et al.*, 1998; Perez-Alenza *et al.*, 2006) .

Diagnosis: based on the clinical signs and history of grazing bracken infested pasture, PM findings. (Bischoff and Smith, 2011)

Treatment

Affected animals should be given high doses of antibiotics (penicillin, streptomycin) over a prolonged period.

Prevention

Avoid use of bracken infested pastures at time of greatest risk, i.e. when grazing is in short supply.

Reduce the level of bracken contamination by cutting or burning.
Avoid using bracken as bedding material.

In conclusion, ruminant's animals, feedstuffs should be given in proper ways and amount (roughages and concentrate ratio) to avoid such type of health problems.
Feeding of undesirable things should be restricted in ruminants to prevent such type of health issues.
Balanced feeding and nutritional supplement are important ways to enhance health and prevent disease in ruminant animals.

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How to cite this article:

Manju Lata and Mondal, B. C. 2021. Disease Developed in Ruminant Animals due to Faulty Feeding. *Int.J.Curr.Microbiol.App.Sci.* 10(03): 447-459.
doi: https://doi.org/10.20546/ijcmas.2021.1003.060