RUMENITIS IN CATTLE*

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Introduction

Rumenitis has been referred to by several clinical terms, including acute indigestion, rumen overload and grain overload. The conditions not included for discussion are bloat, impaction of the rumen, or disorders of the abomasum. The clinical signs in rumen overload result from fermentation of abnormal amounts of carbohydrate in the rumen, leading to profound acidosis within the rumen, chemical rumenitis, dehydration, diarrhea and death. Rumen overload or rumenitis are the terms preferred for the disease and will be used in this discussion.

Rumenitis occurs most commonly among feeder cattle soon after they are placed on high grain rations. The second most common occurrence is when cattle of any type gain access to large quantities of grain by accident, usually by breaking into a granary or feed bin or from being overfed by a well-meaning neighbor who is doing chores for a day or two. If eaten in excess, apples, grapes, bread, sugar beets, mangels and corn will also produce rumen overload. The total amount of grain is not as important as is the amount in excess of what the animals are presently accustomed to being fed.

The history of accidental exposure to, and consumption of, large quantities of grain by dairy cattle, or of increasing the grain to feeder cattle is a very important part of establishing a diagnosis of rumen overload. Usually, the accidental cases have an obvious history. Losses from rumen overload in feedlots will generally be directly proportional to the management. If cattle are placed on grain too quickly or switched from hand feeding to self feeders, trouble may, and probably will, ensue. The minimum time to bring cattle to full feed will vary with management but in general three weeks is necessary. It might appear that taking cattle from full feed by hand, to full feed by self feeders is a harmless step, but this is not true. Large outbreaks of rumenitis often occur when this step is taken. For some reason the cattle will engorge themselves on self feeders. Rumenitis is a constant enemy of the manager who allows newly purchased cattle to mix with those on full feed without a period of relative isolation and adjustment for the newcomers.

Pathogenesis

The ingestion of excessive carbohydrates, usually in the form of milled grain, allows for rapid fermentation within the rumen. The main product of the fermentation is lactic acid and it is this substance in excess which is responsible for the clinical signs and lesions. The excess acidity lowers the pH of the rumen content from a normal value of about 7.0 to levels of 3.5 to 5. In doing so the osmotic pressure within the rumen is increased greatly.

The results are: (1) chemical rumenitis due to the acidity; (2) a massive flow of fluids into the rumen from the body circulation caused by the increased osmotic pressure; and (3) destruction of the protozoan and bacterial flora of the rumen, with the exception of streptococci and lactobaccilli which are the organisms carrying out the fermentation of the grain.

Thus the clinical signs are accounted for by the acidosis and dehydration, and the lesions of rumenitis by the acidity and high osmotic pressure. The animal cannot recover until the pH of the rumen content is lowered and the rumen flora is re-established.

Clinical Signs

The clinical signs are proportional to the excess of carbohydrate eaten and begin with complete anorexia, followed by generalized depression and complete rumenal atony. These signs are evident

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12 to 18 hours after the intake of the excessive carbohydrate; at about 18 hours diarrhea begins. The feces are foul smelling, yellowish in color and pasty to watery in consistency. A serous nasal discharge is very often evident. Affected animals move reluctantly and soon appear obviously dehydrated, with sunken eyes and slight abdominal distension. The temperature may be 103 to 104° F. in the early stages, but is usually subnormal with the onset of dehydration. The rumen is soft on palpation and ballottement indicates a doughy or watery content. Some affected animals are lame, apparently from laminitis. The heart rate is raised to 120 per minute and the respiration rate increases with the degree of acidosis. Severely affected animals stagger and appear to be blind. The affected animal soon becomes recumbent, appears very dull, but still has a profuse diarrhea and a static rumen. A sample of rumen content taken with a stomach tube or with a 6 inch, 18 gauge needle reveals a pH of 3.5 to 5. If examined as a wet mount under the microscope, protozoal activity is absent in the rumen content. Anuria is usually present also. Serum inorganic phosphorus levels tend to be very high, probably because of the large amount of grain in the digestive tract. Hematocrit values may reach 50% from a normal of about 30%.

Pathology

The autopsy lesions are thick, dark blood indicative of dehydration, and a distended rumen having a fluid content which contains excessive grain. The rumenitis is usually confined to the ventral sacs of the rumen. The lesion may not be visible from the mucosal surface and it may be necessary to scrape the lining epithelium of the rumen with a knife. The lamina propria of the rumen is bright red in color and the epithelium detaches with difficulty. Normally the ruminal epithelium can be detached easily two or three hours after death.

Rumen overload has been produced experimentally via rumen fistula in cattle (1). The opportunity arose to examine rumen epithelium during the early phases of the disease. Fourteen liters of lactic acid at a pH of 4.0 were placed in the washed out rumen of the experimental animals and rumen villi were removed at varying periods thereafter. It was necessary to add more lactic acid to keep the pH at about 4. The experimental animals could bring the pH up to 7 in 6 hours mainly by the action of saliva.

The lesion began as a hydropic change in the epithelial cells in and below the stratum lucidum, followed by rupture of these cells to form microvesicles involving most of the prickle cell layer of the epithelium. Intense accumulation of neutrophils occurred in the microvesicles, followed by sloughing of parts of the vesiculated epithelium. The capillaries in the submucosa were dilated and filled with blood. The hydropic change in the epithelium was apparent at 6–10 hours, with the most advanced stage at 24 hours. In areas of thick epithelium on large villi, only part of the epithelium sloughed so that ulceration did not occur. In areas of thin epithelium on small villi, however, ulceration occurred. These microscopic lesions have been observed in natural cases as well as experimental, and serve to explain the pathogenesis of the gross lesions.

In natural cases, ulceration of the lining epithelium will lead to the next sequence of events which may be regarded as a sequel to a mild expression of the disease. The rumenitis-liver abscess syndrome in feedlot cattle is very likely a direct result of a degree of rumen overload and rumenitis. Bacteria, mainly Spherophorus necrophorus, infect the ulcers produced by the chemical rumenitis and produce foci of infection. These foci appear as more or less circular areas on the mucosa, with a raised, necrotic surface. The lesions do not extend to the serosa. Healed lesions appear as puckered scars devoid of villi. Bacterial emboli from these foci are carried to the liver and result in focal necrosis and abscessation. If liver abscesses are found in feeder cattle at slaughter or at necropsy, a lesion of a previous rumenitis, if looked for, will almost certainly be found. Liver abscesses cause great economic loss to the meat industry each year.

In severely affected animals that live a few days the sequel to the chemical rumenitis is mycotic rumenitis. Fungi, normally present but perhaps selectively aided by the high carbohydrate media, invade the ulcerated areas and penetrate to the submucosa and serosa. The fungal

infection has much more serious consequences than bacterial infection. The fungal hyphae invade blood vessels and produce extensive thrombosis of the vessels of the rumen wall. Necrosis extends from the mucosa to the serosa. The lesions appear grossly as circular or irregular-shaped areas, thickened when compared to the normal areas of the rumen wall, and dark red to blue to black in color. Lesions caused by bacteria alone are not usually visible from the serosal surface but can be felt from the serosal surface. On the mucosal surface the mycotic lesions are circular or irregular in shape, raised, and have a reddish granular or dark blue color. In areas with short villi, the early lesion has a pattern similar to ringworm on the skin of cattle. The fungi may spread to the liver as emboli and into the abdominal cavity by direct invasion, resulting in fibrinohemorrhagic peritonitis and focal hepatitis. In severe or prolonged cases mycotic lesions extend into the lower digestive tract. Parts of the leaves of the omasum are necrotic, black in color and clearly demarkated from the normal mucosa. A hemorrhagic to diphtheritic inflammation is present over variable portions of the abomasum and in patches along the small and large intestinal tracts. The thickened black patches on the serosal surface of the rumen are characteristic of mycotic rumenitis and in advanced cases may become confluent in the ventral sacs of the rumen. Acute cases of rumen overload often appear to respond to treatment for a few days but then relapse and die. Such animals will very likely have mycotic rumenitis.

Therapy

The principles of treatment of the acute disease follow from an understanding of the pathogenesis. Once the mycotic stage is reached treatment is likely to be in vain. Large quantities of fluid intravenously, alkalizing agents (probably magnesium hydroxide) orally, antihistamine and antibiotics parenterally are necessary in treatment of the disease. The fluid therapy is particularly vital. Antibiotics may be infused directly into the rumen to reduce the lactic acid fermenting bacteria. Rumenotomy is necessary in severe cases and is advised in valuable animals. In large outbreaks emergency slaughter may be the only solution. A transplant of rumen flora from a normal animal is usually necessary for a prompt recovery and is one of the best forms of therapy. It is possible to overtreat with oral alkali and cause alkalosis rather than return the animal to normal.

Discussion

The prognosis is proportional to the time and adequacy of treatment. In severe cases treatment must indeed be heroic. Very often affected animals manage to survive on minimal or moderate treatment but do not improve and die in a few days. In this disease there is a great difference between an animal being alive in three or four days time and an animal making a complete recovery.

The conditions to be considered under differential diagnosis, at least in the early phases of the disease, are so-called simple indigestion, traumatic reticulitis and subacute lead poisoning. Of these, simple indigestion is likely to be the most confusing when making a diagnosis.

Simple indigestion usually has the following features: (1) rumen usually full of feed; unable to ballotte a watery content; (2) normal feces; (3) bright attitude; (4) improvement in 12–24 hours; and (5) rumen pH normal, or up to 7.5 or 8.

Simple indigestion is usually caused by too much good hay, too much silage, or too much high protein concentrate. If caused by damaged hay or frozen food, there is usually a mild bloat along with the simple indigestion. When cattle eat an excess of protein or urea, a rumenitis may also develop. In these cases the rumen pH is 8–10. Indigestion in a recently calved or post parturient cow may be due to eating the placenta. This can cause severe illness with rumen stasis, diarrhea, rumen pH of 10, and destruction of protozoa from the rumen flora.

In a group of animals, the main points for establishing a diagnosis are history, rumen pH and clinical findings. The most important point is the pH of the rumen content. Very often, ordinary pH paper will suffice to demonstrate the rumen acidity; however, this method may not be accurate enough in some cases and laboratory assistance may be necessary. Examination of rumen fluid under a coverslip on a slide is very useful in demonstrating the

lack of motility of the protozoal flora and is an indication of the acidity.

In an individual animal, without a history of taking in excessive grain, it may be very difficult to arrive at a clinical diagnosis. The pH of the rumen content, examination for protozoan activity and serum phosphorus may give the only positive indications in an otherwise confusing clinical picture. The evaluation of the rumen pH and flora in dead animals is a problem because normals are not established. It is not known if, or how quickly, the pH of the rumen content changes or how quickly the protozoa die following death in a normal animal. An estimate of the time after death during which pH and protozoal changes in the rumen should reflect ante mortem conditions would be six hours.

This entire story could be repeated for the disease in sheep, up to the point of mycotic rumenitis. It appears that sheep are more likely to die in the acute phase of the disease than are cattle, and seldom live long enough to develop the mycotic infection. In sheep, anorexia and profuse diarrhea are the main clinical signs.

Summary

Rumen overload is likely to be accidental, except in feedlots where it is a management problem. The pathogenesis of the condition results from fermentation of excessive carbohydrate in the rumen, leading to increased osmotic pressure within the rumen, a drop in pH to 3.5 to 5, dehydration, diarrhea and death. A sequel to the acute disease may be mycotic rumenitis. Milder cases of rumenitis or cases that have responded to treatment may subsequently develop liver abscesses. The most important factors in establishing a diagnosis are the history, and an evaluation of the pH of the rumen content.

Résumé

L'impaction du rumen est ordinairement accidentelle, sauf dans les parcs d'alimentation où l'hygiène alimentaire est déficiente. On explique la pathogénie de la maladie par une fermentation excessive des hydrates de carbone à l'intérieur de l'organe, qui suscite une augmentation de la pression osmotique, une élévation du pH de 3.5 à 5, de la déshydration, de la diarrhée et finalement la mort. La forme aigüe de l'affection provoque éventuellement une ruminite mycotique. Des abcès hépatiques peuvent se développer comme séquelles de ruminites bénignes ou de ruminites qui ont été traitées avec succès.

L'étude de l'anamnèse et du pH du contenu stomacal constituent les deux facteurs les plus importants à l'élaboration d'un bon diagnostic.

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Reference

1. Ahrens, F. M.Sc. thesis. Cornell University, Ithaca, New York. 1965.

ABSTRACT

Störiko, K. R. (1966). (Live foot and mouth disease vaccine for pigs.)—Bull. Off. int. Epizoot. 65, 359–360 (F.) (Bundesforschungsanstalt, Waldhäuser Höhe, Tübingen).

Strains of Types O, A and C were modified by passage on calf kidney cells, until loss of virulence (and immunogenicity) for pigs. The time required for loss of virulence was related to the temp. (shorter at 28° or 22° than at 37°C). Completely avirulent strains gave good immunity at high doses only. Results indicate that these modified strains are not to be used for the preparation of live vaccines.

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