CASE REPORT

Perforated Duodenal Ulcer in a Cow

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Summary

A case report of perforated duodenal ulcer in a ten year old Holstein cow is presented. On three occasions, sudden anorexia and rapidly progressing abdominal fluid distension were associated with metabolic alkalosis, hypochloremia and hypokalemia. Rumen fluid at the time of the second episode was acidic and contained an excessive amount of chloride ion. An abdominal mass dorsal to the abomasum involving the pylorus and several loops of small bowel was identified but not corrected at surgery. Necropsy confirmed a 1.5 cm diameter duodenal ulcer 6 cm distal to the pylorus.

Résumé

Perforation d'un ulcère duodénal, chez une vache

Cet article rapporte la perforation d'un ulcère duodénal, chez une vache âgée de dix ans. À trois reprises, elle manifesta une anorexie soudaine et une distension rapide de l'abdomen par du liquide, lesquelles s'accompagnaient d'alcalose métabolique, d'hypochlorémie et d'hypokaliémie. À la deuxième reprise, le liquide du rumen s'avéra acide et il contenait un excès de Cl. La chirurgie révéla la présence d'une masse abdominale, juste au dessus de la caillette; cette masse intéressait le pylore et plusieurs anses de l'intestin grêle. La nécropsie de la vache permit de constater qu'il s'agissait d'un ulcère duodénal de 1,5 cm de diamètre, situé à 6 cm du pylore.

Introduction

Gastric ulceration is common in the bovine (1,7,8,13) whereas duodenal ulceration is rare (11,13). Perforation associated with either problem could cause acute or chronic local peritonitis or acute diffuse peritonitis (1,7,8,11, 13) or fatal hemorrhage (1,7), with or without abomasal reflux and concomitant hypochloremic alkalosis (2,6). This report describes a mature cow which showed recurrent anorexia, abdominal distension, dehydration, alkalosis and hypochloremia attributed to a perforated duodenal ulcer.

History

A ten year old 1600 lb (727 kg) Holstein cow was referred to the Ontario Veterinary College (OVC) with a history of suddenly going off feed three days prior to admission. She had been reared by the owner and although only bred three months previously, was nearly dried off. The cow was at pasture through the day and fed grain in the milking parlor. As rumen activity was reduced she was then treated orally with 225 g of a rumen stimulant and alkalizing preparation.[1] Subsequently that afternoon and the following morning a dose of 112 g had been administered. Little improvement occurred and within 24 hours a profuse diarrhea ensued.

Clinical Findings

When examined at OVC three days after onset of clinical signs (day 1), the cow was alert and the abdomen was moderately distended. The feces were dark and watery, but negative when tested[2] for occult blood.

Fluid sounds and a fluid wave detectable on both sides of the abdomen, were more pronounced on the right side. Auscultation of the left flank revealed two complete contractions/minute. During the examination the cow ate hay and drank. Rectal temperature was 38.9°C, heart rate 60/minute and respiratory rate 24/minute. Based on skin elasticity and prominence of the eyes, the cow was assessed as slightly dehydrated. At this time, a blood sample for blood gas analysis and the determination of plasma electrolytes and aliquots of urine and feces were collected for urinalysis and culture respectively. Rumen pH[3] was evaluated on fluid aspirated *via* stomach tube.

Laboratory Results

The cow was in a state of uncompensated metabolic alkalosis as venous pH was 7.43 (normal 7.32 to 7.42), HCO_3^- was 43 mEq/L (normal 22 to 28) and the base excess was 15 mEq/L (normal ± 3). Plasma K+ at 2.6 mEq/L (normal 4.1 to 5.1) and Cl^- at 84 mEq/L (normal 85 to 105) were also abnormal. The urine did not contain glucose, ketones or protein and had a Ph of 6. Nothing of pathological significance was cultured from the feces.

Diagnosis

Based on these laboratory findings hypokalemic metabolic alkalosis with paradoxical aciduria was suspected. Since the cow showed no evidence of muscle weakness, was eating and had been previously treated orally with 450 g of an alkalinizing agent, no treatment was undertaken and the cow was kept for further observation.

On day 2, the blood pH was identi-

[1]Oxaplus Powder, magnesium hydroxide, sodium phosphate, sodium thiosulfate, antimony potassium tartrite, strychnine (from nux vomica), Rogar/STB, London, Ontario.

[2]Hematest Tablets, Ames Co.: Division, Miles Lab. Ltd., Rexdale, Ontario.

[3]pH indicator stick, Em Lab. Inc., 500 Executive Building, Elmsford, New York 10523.

cal (7.43) but HCO_3^- concentration had dropped to 27 mEq/L and urine pH had increased to 7.0. Heart rate varied throughout the day between 48 and 60 beats/minute. Over the next three days the cow, although untreated, continued to eat well. The abdominal distension gradually decreased and the feces became soft in consistency. An atropine test (4) conducted at that time was considered positive as the heart rate increased by 25% within 15 minutes after injection of 30 mg atropine sulphate subcutaneously.

The cow appeared unchanged clinically on the morning of the sixth day but her condition deteriorated that afternoon. Feed intake was reduced but abdominal distension was obvious on the left flank and fluid sounds and a fluid wave were detectable over both sides of the abdomen. Fecal consistency was normal. Venous blood pH and HCO_3^- values were 7.55 and 50 mEq/L respectively, indicating a severe uncompensated metabolic alkalosis. Hypokalemia (2.3 mEq/L) and hypochloremia (74 mEq/L) were also revealed. Urine had become alkaline (pH 8.5). Rumen fluid contained 68 mEq/L of chloride ion (normal range 16 to 32) and was slightly acid (pH 6.5).

These clinical and laboratory findings were consistent with an abomasal or pyloric problem with sequestration and reflux of abomasal secretions into the rumen. Treatment consisted of 90 litres of an acidifying solution composed of two parts isotonic sodium chloride (0.85%), one part isotonic potassium chloride (1.1%) and one part isotonic dextrose (5%). The fluid mixture was administered by continuous intravenous drip over a 48 hour period to cope with the dehydration, alkalosis and electrolyte imbalances.

On the morning of day 8, while the cow was still on intravenous fluids, a left flank exploratory laparotomy was performed. Old adhesions were detected on the anterior surface of the reticulum and diaphragm. Also, a large mass, 20 cm in diameter, was palpated dorsal to the abomasum. Since it was impossible to identify this mass, the left flank incision was closed and the cow continued to receive supportive therapy. During days 8 and 9 the cow appeared clinically normal.

On day 10 the cow again showed a sudden reduction in feed intake and progresive abdominal distension and concomitant dehydration, metabolic alkalosis, hypochloremia and hypokalemia. The cow was anesthetized and a right paramedian laparotomy was performed on day 11. The mass was exteriorized with difficulty. It involved the abomasum, the pylorus and sevèral loops of small intestine. Because the abomasum appeared impacted, an abomasotomy was performed to remove the dry, fibrous roughage. During this surgery the animal died suddenly.

Necropsy Findings

Pertinent findings at necropsy included fatty necrosis of the mesentery associated with the abdominal mass at the junction of the abomasum and the duodenum. Dissection of this mass revealed a perforated duodenal ulcer, 1.5 cm in diameter, 6 cm distal to the pylorus. Although several loops of small intestine were incarcerated in the mass, there was no evidence of physical obstruction of the small intestine. three linear ulcers 0.5 by 2 cm were present on the mucosal folds of the abomasum.

The histological changes due to inflammation and to necrosis of the fatty tissues in the mass were considered to be three to four weeks old. Sections from the ulcerated abomasum revealed necrosis, hemorrhage and large numbers of neutrophils which in places extended to the submucosa.

Discussion

The decision to leave the cow untreated in spite of the diarrhea, alkalosis, hypochloremia, hypokalcemia and aciduria was based on the following reasons. Therapy prior to admission could have initiated the alkalosis and diarrhea (9,12) which could explain the hypochloremia and hypokalemia (9,10,14). The cow exhibited no evidence of generalized muscular weakness and had a normal appetite, clinical features not consistent with previously reported cases of metabolic alkalosis and paradoxical aciduria in the bovine (6,10). Decreased plasma HCO_3^- concentration and increased urine pH on day 2 and continued normal appetite until the afternoon of day 6 support this decision.

The sudden deterioration in the clinical state of the cow on day 6, was unexpected. The alkalosis and acid pH and high chloride ion concentration in the rumen fluid were considered to be supportive evidence for an abnormality of either the abomasum or pylorus with sequestration of HCl in the forestomachs (2,15). The bradycardia (as low as 48/minute) and the positive atropine test (day 2) were consistent with increased vagal tone which could be associated with either the adhesions around the reticulum and the pyloric or abomasal ulceration (4). Although histologically severe, the abomasal ulcers, small and without evidence of recent homorrhage, were not considered clinically significant. The duodenal ulcer and adhesions resulting from perforation were considered of prime clinical importance. Critical retrospective review of this case would lead one to conclude that the episodes of anorexia and abdominal distension, and the sudden final episode on day 10 were related to the duodenal ulcer.

Duodenal ulcers in man have been associated with recurrent pylorospasm (3,5). That could explain the sequestration of abomasal fluid in the abomasum and rumen of this cow. In spite of the pathologist's impression of the age of the lesion (approximately three to four weeks) the authors are suspicious that perforation of the ulcer occurred at the time of the initial episode of abdominal distension two weeks prior to death. It would seem unlikely that perforation prior to this time would have gone unnoticed as the cow was fed and observed and milked twice daily.

Duodenal ulceration should be considered along with abomasal and pyloric problems as a cause for reflux of abomasal secretions into the rumen.

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BOOK REVIEW

Comparative Diagnosis of Viral Diseases, Volumes III, IV. Edited by E. Kurstak and C. Kurstak. Published by Academic Press, Inc., Publishers, New York. 1981. Volume III, 429 pages. Price $54.00. Volume IV, 694 pages. Price $79.00.

These two volumes are the first that we have seen of this series. Volume III is dedicated to the study of desoxyribose nucleic acid viruses while volume IV deals with ribonucleic acid viruses. They offer a comparative study of the physicochemical, molecular, structural, genetic properties of the various diseases implicated in disease.

Immunological characteristics are well covered, and give a good understanding of the complexity of the detection and identification of the disease causing virus. The sheer number of viruses studied exemplifies well the problems faced by the virologist.

The symptoms of the disease are well described in the various host species. Their evolution is followed and outcome of the disease is explained when required. Modern methodology makes rapid and specific diagnosis possible and it is explained in enough details. Presently accepted means of control and prevention are also given.

Whenever possible, the classification recommended by the International Committee of Taxonomy of Viruses has been followed.

Individual chapters dealing with a given viral family have been written by a recognized authority in this given area. This indicates the present research directions in the comparative diagnosis and in the prevention of viral diseases. It serves also to highlight the relevant current findings.

These two volumes from a beautiful unit, and will undoubtedly be appreciated by virologists, researchers and many others. In spite of the obvious complexity of the subject, the authors have succeeded to make this treatise a most interesting and useful tool.

C. Gardell

Veterinary Virology. S.B. Mohanty and S.K. Dutta. Published by Lea & Febiger, Philadelphia. 1981. 372 pages. Price $28.25.

This book is the result of a graduate course in veterinary virology taught by the authors at the University of Maryland.

Two sections make up the framework of the book. The first deals with general animal virology, and covers classification, cultivation of viruses, their effects on the host cells, the pathogenesis of viral infection, resistance to viral infections and immunity, the epizootiology of viral diseases. A chapter deals with vaccines and anti viral drugs. The last chapter studies the laboratory diagnosis of viral diseases.

The second section covers specific viruses. These are grouped according to their host species (equine, canine etc. . .) (one chapter is devoted to the laboratory, wild and zoo animals, another to the slow viruses (scrapie, maedi/visna, etc. . .). One chapter deals with oncogenic viruses and the last one with viral diseases of animals which are transmissible to man.

This book is concise, and at times, somewhat too concise. This is specially true where the zoonoses are concerned. This chapter is an enlarged list of viruses and could or should supply more information.

The chapter dealing with laboratory diagnosis of viral diseases is specially interesting and well written. It will prove of advantage to many, as it explains the techniques used, while, usually only the results of the tests are seen.

The section covering the specific viruses is indeed very well written and supplies a lot of valuable information.

This book is a more basis text book than Andrewes, Pereira and Wildy's Viruses of Vertebrates, and as such more accessible to unspecialized students and professionals. *C. Gardell.*