CASE REPORT

Torsion of the Abomasum in a One Month Old Calf

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Summary

The clinical and clinicopathological findings of a one month old Holstein heifer calf presented with severe abdominal distention are reported. Preoperative evaluation and therapy were initiated and followed by an exploratory laparotomy (paracostal right flank) which revealed a 360° counterclockwise torsion (viewed from the rear) of the abomasum. Severe vascular compromise of the abomasum precluded salvage of the animal. Following euthanasia and postmortem evaluation, the calf was found to have irreversible changes within the abomasum.

Key words: Abomasal surgery, abomasal torsion, blood gas analysis, calf, metabolic changes.

Résumé

Torsion de la caillette, chez un veau âgé d'un mois

Ce rapport commente les observations cliniques et pathologiques relatives à une génisse Holstein, âgée d'un mois, qui présentait un ballonnement marqué. L'auteur procéda à la thérapie et à la préparation préopératoires conventionnelles; elle effectua ensuite une laparatomie exploratrice, par une approche paracostale, dans le flanc droit. Cette intervention révéla la présence d'une torsion de la caillette de 360°, en sens inverse des aiguilles d'une montre, quand on la regardait d'en arrière. Les constatations significatives, lors de la chirurgie, incluaient une grave atteinte des vaisseaux de la caillette, incompatible avec la survie de la génisse. Après l'euthanasie et la nécropsie, on confirma la présence de lésions irréversibles de la caillette.

Mots clés: chirurgie de la caillette, torsion de la caillette, analyse des gaz sanguins, veau, changements métaboliques.

Introduction

Abomasal torsion is recognized as being a common cause of severe abdominal distention in young calves (1). The condition is seen across all areas of intensive dairy and beef practice and is usually associated with a poor prognosis. Despite its recognized status, there is little information available describing both the condition and the clinicopathological findings of this disease. There are even fewer descriptions of acid-base and electrolyte findings in such affected animals, the tendency being to equate abomasal torsion in calves with abomasal problems seen in adult animals. The purpose of this paper is to describe a calf with abomasal torsion and its coincident clinical, acid-base, electrolyte and pathological findings. Research of the literature revealed only one instance of induced abomasal torsion in "calves" which did not coincide with the results found in this particular calf (2).

History and Clinical Findings

A one month old Holstein heifer calf was presented to the Western College of Veterinary Medicine Large Animal Clinic with severe bilateral abdominal distention. Twelve hours earlier the calf was normal in all respects and had a healthy appetite. High quality, freshly cut alfalfa hay was added to the diet that day in addition to the whole milk and oat chop the calf normally received. Over the next few hours the calf developed a very distended abdomen resulting in severe distress. An attempt was made by the owners to relieve the abdominal pressure by opening the rumen with a pocket knife. However, the distention on the right side prevailed. Following the owner's treatment with one-third cup of turpentine and two cups of milk per os, the calf was referred to the clinic for evaluation. Physical examination revealed a subnormal temperature of 37.5°C, a heart rate of 80 beats per minute, and a respiratory rate which was shallow and elevated. The calf was moderately dehydrated with cool extremities, pale mucous membranes and a slow capillary refill time. The abdomen of the calf was distended bilaterally. The stab wound to the left flank was present and allowed gas and fluid content to escape to the outside. A taut distended mass, extending over a large area on the right side of the calf, elicited a metallic ping upon percussion and auscultation. The rectum contained blood and mucous with a minimal amount of fecal material present.

Abdominocentesis produced a blood-tinged, nonturbid fluid. The red and white blood cell counts of the fluid were found to be within normal limits.

As seen in Table I the laboratory test revealed a mixed acidosis without abnormal electrolyte concentrations. On the basis of clinical and laboratory findings a tentative diagnosis of

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abomasal torsion was made and surgery was performed.

Surgical and Pathological Findings

The preparation for surgery consisted of intravenous fluid therapy with Ringer's solution, aseptic preparation of the right flank as well as a line block using 1% xylocaine with epinephrine. A laparotomy incision was made extending from the xiphoid in a straight line directly towards the wing of the ileum for approximately 30 cm. Exploration of the abdominal cavity revealed a grossly dilated abomasum discolored to a grey-black color on its serosal surface. A 360° counterclockwise torsion of the abomasum about its long axis as viewed from the rear was evident. Other findings included flaccid loops of normal-colored small intestine and fibrin tags throughout the abdominal cavity. The coloring of the intestine was within normal limits. The severe discoloration of the abomasum indicated irreversible changes to the whole structure and based on these findings, euthanasia was performed. Just prior to euthanasia, a section of discolored abomasum was biopsied, placed in formalin and sent for histopathological examination.

Postmortem examination revealed a dilated, discolored abomasum filled with dark brown fluid with very few changes apart from the fibrin tags in the abdominal cavity. The rumen contained a large amount of the newly introduced alfalfa hay. Histopathological findings of the abomasal wall biopsy revealed marked congestion of the lamina propria and fibrin thrombi within the venules. The epithelial layer was completely denuded exposing the basement membrane of the abomasal mucosa. Changes present in the rumen included hydropic degeneration and vacuolization of the stratum corneum and focal areas of ulcerative rumenitis.

Discussion

The cause of abomasal torsions in young calves is not clear. Histories seldom reveal any previous illness, suggesting accidental causes (1). It may be of significance to note the diet change that occurred the day this calf became ill. High quality alfalfa hay was added to the diet and as indicated by the full rumen and rumenitis at necropsy, the calf had consumed large quantities of the hay. It is possible the rumenitis and digestive upset contributed to acute physical and chemical changes in the abomasum leading to dilatation and torsion.

TABLE I
RESULTS OF LABORATORY EVALUATION PERFORMED BEFORE SURGICAL INTERVENTION

	Calf[a]	Normal Range[b]
pH	7.11	7.30 — 7.35
pCO_2(mmHg)	64.7	40 — 50
pO_2(mmHg)	37.9	40
HCO_3(mmol/L)	20.4	18 — 24
BE	-10.4	(-)2.0 — (+)2.0
Na(mmol/L)	135.2	132 — 152
K(mmol/L)	5.5	3.9 — 5.8
Cl(mmol/L)	102.2	95 — 110

[a]Venous sample.
[b]Normal values at Western College of Veterinary Medicine Clinical Pathology Laboratory.

From Table I it is interesting to note that a mixed acidosis was present, even though serum electrolytes (Na^+, K^+, Cl^-) were within normal limits. Sequestration of large quantities of gastric secretions in the abomasum produces a state of metabolic alkalosis, hypokalemia and hypochloremia in cows with abomasal torsions (3,4,5, 6). Along with these characteristic metabolic changes, a paradoxic aciduria also develops related to the low serum potassium levels (5,6). The metabolic state of cows with abomasal torsion sharply contrasts the acidosis and normal electrolyte findings of this young calf. Unfortunately a urine sample was not obtained from the calf, so the pH of the urine cannot be compared with the paradoxic aciduria condition. The respiratory component (increased pCO_2) of the mixed acidosis was probably due to respiratory embarrassment caused by the abdominal distention and pressure on the thorax while the metabolic component may have occurred secondary to visceral pooling of blood and the resulting state of shock. With the acute course of this disease it is possible electrolyte shifts could not, or did not occur in that short time period or that the abomasum of the young calf does not have the ability to secrete and sequester fluids as in the adult abomasum.

Related conditions of gastric dilatation and torsion in dogs and high intestinal obstructions in horses often cause a metabolic acidosis. As in cows, horses may develop a metabolic alkalosis if large amounts of fluid are sequestered in the stomach however, acute high intestinal obstructions result in metabolic acidosis (1,8). Dogs can develop metabolic acidosis or alkalosis subsequent to a gastric dilatation and torsion. Although each case must be evaluated, metabolic acidosis and hypokalemia are commonly present in acutely affected dogs (9,10,11). While adult ruminants are consistently alkalotic with decreased serum concentrations of Cl^- and K^+ when the abomasum is vascularly compromised, calves may not follow this pattern. The metabolic state in this calf with abomasal torsion would appear to more closely resemble that of monogastrics with similar gastric abnormalities.

Duration of illness may be another factor that differs between adult and young ruminants. This calf progressed from a normal state to lateral recumbency in less than 12 hours. Severe irreversible changes were present in the abomasum at the time of surgery leaving little hope of survival even with correction of the torsion. Cows may develop a right abomasal dilatation and torsion acutely or quite commonly, the course of illness extends over a period of days (1, 12). Surgical correction of these torsions is often successful even after several days of illness, with postsurgical outcome correlated to serum Cl^- concentrations and pulse rates preoperatively (7).

The abomasal torsion in this one month old calf with resulting acidosis and normal serum electrolytes suggests an acute disease, resembling acute gastrointestinal problems in monogastrics. This particular case may have been caused by the digestive upset and rumenitis from the excessive alfalfa hay consumed. It is important to note the contrasts presented here to abomasal torsion in adult ruminants. Each acutely ill animal should be evaluated individually, without speculation, so that proper effective treatment can be given accordingly.

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BOOK REVIEW

Prolactin. Volume 8. D.F. Horrobin. Published by Eden Press, Westmount, Quebec. 1981. 152 pages. Price $37.50.

The book entitled *Prolactin* is the eighth in a series of reviews by Dr. Horrobin which contain recent information about prolactin and its functions. This is the final edition which was published in 1981 and contains research information through 1979 on a variety of topics including the structure of prolactin, its secretion, metabolism, clearance and receptors. Also discussed are the cellular and metabolic effects of prolactin as well as its role in regulation of such diverse phenomena as water and electrolyte balance and endocrine, especially reproductive, events. The discussion emphasizes the human clinical aspects of prolactin activity and includes a treatment of the role of prolactin in carcinogenesis and mental illness. The book reviews information from more than 300 references and its comprehensive approach in itself is valuable to anyone interested in this hormone.

A particularly interesting portion of the book deals with the mechanism of action of prolactin on its diverse population of target cells. Prolactin receptors are found on hepatic cells, kidney tubules, adrenal cells, adipose tissue, and the function of the hormone at these sites is unknown. The greatest concentrations of receptors are found on mammary tissue and in the testis and ovary, where specific stimulatory and inhibitory effects of prolactin have been described. Horrobin's section on receptors, in which he postulates action through a second messenger, presages the later discovery of a membrane associated factor which stimulates specific mRNA synthesis in isolated mammary nuclei in response to the addition of prolactin (Teyssot *et al.* PNAS 1981; 78: 6729.

A lengthy and interesting review of the relationship between the hepatic prolactin receptor and circulating sex steroids describes an apparent stimulatory effect of estrogens on receptor populations and an inhibition by testosterone. The text is replete with obscure findings such as prolactin potentiates the norepinephrine induced contractions of the vas deferens in the rat. The author alludes to the now familiar story of prolactin as the principal mechanism of control of gill mineral and electrolyte transfer in fish.

The review is often speculative, bringing together information from diverse and unrelated experiments in hopes of rationalizing the wide range of different physiological activities of prolactin. Clinical experiments receive critical review, and in one case, papers on the treatment of prolactin-secreting tumors are scathingly described as "me-too papers describing only a small number of patients and not illustrating or demonstrating any new points" (p. 104).

This book is of considerable interest to the scientist working in the area of metabolic or reproductive endocrinology, because, despite the fact that it is a few years out of date, it provides a comprehensive overview of a hormone which is highly conserved from an evolutionary point of view. The target organ adaptation, that is the capacity of animals to utilize this hormone to whatever end it suits is continually amazing. Dr. Horrobin's review provides us with an appreciation for that diversity. It is regrettable that this series has not continued to include the fast moving research which has characterized the study of prolactin in recent years. *B.D. Murphy.*