PEER REVIEWED

Generalized Peritonitis in Cattle: 31 Cases (1993-1997)

Mohammed Ebeid, *BVSc, MVSc, Dr.med.vet.*
D. Michael Rings, *DVM, MS, Dipl. ACVIM*
Department of Veterinary Clinical Sciences
Ohio State University
Columbus, OH 43210

Abstract

In a retrospective study, 31 cases of generalized, severe peritonitis in cattle were reviewed for the period between 1993-1997. Historical, clinical, laboratory and necropsy findings were analyzed. Physical examination findings were non-specific. The more consistent findings were depression, anorexia, decreased fecal output, and varying degrees of dehydration. Most cases of generalized peritonitis in cattle occurred peripartum. Clinical examination alone was not always adequate to establish the diagnosis. Hematologic findings were non-specific, with both neutrophilia and neutropenia found in affected cattle. The clinicopathologic findings did not correlate significantly with the duration of the condition. Serum chemistry profiles revealed hyponatremia, hypochloremia, and hypokalemia. Twenty three cases showed hypocalcemia and hyperphosphatemia was noticed in 15 cases. Serum urea nitrogen and creatinine were increased in 11 cases. Increased plasma fibrinogen was recorded in four of the five cases in which it was estimated. Abdominocentesis, performed in 11 animals, was helpful in reaching a clinical diagnosis. Perforating abomasal ulcers (type V) caused peritonitis in 7 of the 20 cases where a cause of peritonitis could be determined.

Introduction

Peritonitis is an inflammation of the mesothelial membrane that lines the peritoneal cavity and covers the intra-abdominal viscera.[14] Several causes of peritonitis have been reported in cattle: traumatic reticulitis, perforating abomasal ulcer, inflammation and/or penetration of the urogenital tract, penetrating trauma of the abdominal wall and intra-abdominal surgical contamination.[1,3] Generalized peritonitis has also been described in three cows following flushing of the uterus in the immediate postpartum period.[11]

Despite the importance of generalized peritonitis, there is a paucity of information in the literature. Therefore, the objective of this study was to review the causes, clinical findings, clinicopathologic changes and postmortem findings in 31 cases of generalized peritonitis presented to the Department of Veterinary Clinical Sciences, The Ohio State University, between January 1993 and December 1997.

Materials and Methods

The medical records of 62 cattle with a diagnosis of peritonitis were reviewed. Thirty-one cases of generalized peritonitis with complete medical records were selected. Data retrieved included the history, physical findings upon examination and clinicopathologic results (complete hemogram and serum chemistry profiles). Abdominocentesis was performed in 11 cases and the peritoneal fluid was examined for physical characteristics, specific gravity, total protein concentration and cell content. Necropsy records of all 31 cases were reviewed to determine the possible etiology of the disease.

Statistics

All data were not normally distributed, therefore they are presented as median and range. Data were analyzed using nonparametric techniques, i.e. Wilcoxon Rank test for paired data and the Mann-Whitney test for non-paired data.

Results

Breeds of the affected cattle were Holstein-Friesian (22) 70.9%, Brown Swiss (5) 16.3%, Guernsey (1) 3.2%, Jersey (1) 3.2%, Hereford (1) 3.2% and Shorthorn (1) 3.2%. The age ranged from 1.9-12 years with a median of 4 years. The majority were females (29; 93.5%),

with two males representing only 6.5%. Nineteen cows (61.5 %) were in the immediate postpartum period (within 8 weeks), three (9.6%) were lactating, non-pregnant cows, and seven cows (22.5%) were late gestation dry cows. The duration of clinical illness (based on owner history) ranged from 1 to 90 days with a median of 4 days. In 19 animals, the duration of clinical disease was less than one week and in 12 cases the duration of disease was more than one week.

Clinical Findings

Depression, anorexia and varying degrees of dehydration were consistently found in all animals presented. In three cows (9.6%), diarrhea was a prominent clinical sign; one was hemorrhagic. In three additional animals (9.6%) bloating was the main presenting clinical sign.

Heart rates ranged from 42-152 beats per min. with a median of 100 beats per min.; respiratory rate varied from 16-96 per minute with a median of 40 per minute. Respiration was typically shallow and labored. Rectal temperature ranged from 98.8-105.1°F (37.1-40.6°C) with a median of 100.9°F (38.2°C). Ruminal stasis was recorded in all cases. Rectal examination was non-revealing and nonspecific. Fecal material was not present in the colorectal lumina of 27 (87%) animals. Pain, characterized by grunt during palpation, was recorded in three animals (9.6%).

Clinicopathologic Findings

The results of hemograms and serum chemistry profiles are presented in Table 1. There were no significant differences in the clinicopathologic findings between animals with short duration of illness (less than one week) and those with a longer period of illness (more than one week).

Leukocytosis (total white cell counts > 14.0 x 10^9/litre) with neutrophilia (polymorphonuclear leukocyte count > 7.0 x 10^9/litre) was present in six animals. Neutrophilia with a left shift but with a total leukocyte count in the normal range was present in seven animals. Leukopenia (WBC count < 4.5 x 10^9/litre) and neutropenia (PMN count < 2.0 x 10^9/litre) were present in eleven animals.

Serum chemistry profiles were hyponatremic, hypochloremic, and hypokalemic. Hypocalcemia (< 8.7 mg/dl) was found in 23 cases. Hypophosphatemia (< 4.5 mg/dl) was noticed in four animals and 15 cases had hyperphosphatemia (> 7.2 mg/dl). Serum urea nitrogen (> 30 mg/dl) and creatinine (> 2.0 mg/dl) were elevated in eleven animals. Creatine kinase (CK) was increased (> 280 IU/L) in fifteen cases. The total serum protein varied. Eleven cases had panhypoproteinemia (< 6.5 g/dl) and in two cases panhyperproteinemia was found (> 8.9 g/dl). Normal plasma protein was found in 18 cases. Plasma fibrinogen (> 500 mg/dl) was elevated in four out of the five animals measured.

Table 1. Hemogram and serum chemistry profile findings in 31 cases of peritonitis

Parameter	Range	Median	Reference range
PCV	21-54	34.5	26-44
Total leukocytic count x 10^9/L	1.4-25.1	9.05	4.5-14
Band neutrophils x 10^9/L	0.05-10.07	0.55	0-0.1
Segment neutrophils x 10^9/L	0.4-20.8	3.6	0.6-4
Lymphocytes x 10^9/L	0.9-12.4	3	2.5-7.5
Calcium mg/dl	5.4-11.3	7.9	8.7-11.4
Phosphorus mg/dl	3.4-14.6	7.8	4.5-7.2
Creatinine mg/dl	0.8-6.7	1.8	0.7-1.5
BUN mg/dl	3-104	36	6-32
Na mEq/L	123-143	137	139-149
K mEq/L	2.1-5.2	3.7	3.5-5.1
Cl mEq/L	62-112	88	84-102
Total protein g/dl	4.4-9.4	6.8	6.5-8.9
CK IU/L	45-58761	1236	35-280
GGT IU/L	16-467	37	5-28
Fibrinogen mg/dl	269-985	713	200-500

Abdominocentesis

Peritoneal fluid analysis was performed in 11 cases. The color of the collected sample was normal in 3 cases with the remaining 8 samples having changes in color ranging from orange to brown. Turbidity of the peritoneal fluid was apparent grossly in all cases. The specific gravity ranged from 1.016-1.035 with a median of 1.026. The total protein concentration varied from 2.5-6.5 gm/dl with a median of 4.2 gm/dl. Total leukocyte counts were between 700-74600/µl with a median of 3250/µl. Neutrophils comprised 29-93 % of the total white blood cell count with a median of 77%. Degenerative or toxic neutrophils were not detected in any case. Bacteria were detected in two peritoneal fluid smears that were examined.

Postmortem Findings

None of the cases survived. They died (20 cases) or were euthanized (11 cases) due to economic reasons. The most common cause of generalized peritonitis in this retrospective study was rupture of a viscus (9), with seven of nine due to perforation of abomasal ulcers. Postoperative surgical complications (8), traumatic reticulitis (1), fat necrosis (1) and trocarization of the rumen (1) were also identified as a primary cause. In 11 cases, the etiology of the peritonitis could not be estab-

lished due to the wide spread nature of the problem. No evidence of gut perforation or history of previous surgery was reported in these 11 animals (Table 2).

Table 2. Causes of generalized peritonitis in 31 cases

Cause	Number of cases	%
Fat necrosis	**1**	**3.2**
Visceral perforation	**9**	**29.2**
Abomasum	7	
Omasum	1	
Uterus	1	
Postoperative complication	**8**	**25.8**
Ventral abomasopexy	1	
Omentopexy	4	
Intestinal anastomosis	2	
Cesarean section	1	
Traumatic reticuloperitonitis with liver abscess	**1**	**3.2**
Secondary to ruminal trocarization	**1**	**3.2**
Undetermined	**11**	**35.4**

Discussion

Cattle have been considered more resistant to generalized peritonitis than other species. This has been largely attributed to the greater omentum, which serves as a physical barrier between the peritoneum and the intestines. In addition, cattle can rapidly deposit fibrin and seal areas of infection.[13] Whether peritonitis becomes generalized, or is contained locally, is largely determined by whether the inflammation can be confined to the area outside the omental sling. Peritonitis secondary to traumatic reticulitis or perforated abomasal ulcers is usually partitioned away from the intestines by the greater omentum. On occasion evidence of prior peritonitis is found during routine surgery, but without a history suggesting peritonitis and without clinical signs in the patient. Bacterial contamination inside the omental sling is much more difficult to control and extensive adhesions often form between bowel loops. This can adversely affect intestinal motility and may create strictures or occlusions of the bowel.

The likelihood of generalized peritonitis in cows increases in the peripartum period due to a greater prevalence of abomasal ulcers, uterine infections, and conditions necessitating abdominal surgery (abomasal displacements, cesarean sections).

In our retrospective study, and in one other study,[10] perforating ulcers (type V) caused about one-third of the cases of generalized peritonitis in which an etiology could be determined. Stressors such as feed changes, concurrent infections (metritis, mastitis), parturition (hormonal changes), and increasing milk production all predispose dairy cows to abomasal ulcers during this period.[3]

Metritis can occur in the immediate postpartum period. If the inflammation extends through the uterine wall, the resulting perimetritis is characterized by weeping of inflammatory proteins through the uterine serosa and into the abdomen. Such infections can result in adhesions of the uterine horns to abdominal structures, i.e. body wall, rumen, omentum, or may transfer bacteria inside the omental sling. Additionally, the oviducts are open immediately postpartum and uterine lavage or large volume uterine flushes may force bacteria through the ducts and into the abdomen.[11]

Surgical contamination is an important cause of peritonitis. External contamination of the abdomen is not the only reason for generalized peritonitis associated with surgery. Abdominal exploration in cattle with existing fibrinous peritonitis can lead to extension of the infection if the adhesions are disturbed. Adhesions wall off infections. When adhesions are broken down, the infection is no longer contained and the surgeon can become contaminated as well. Further exploration of the abdomen can carry the infection into new body sites. Adhesions, especially those outside the omentum, should not be disturbed.

The manifestations of generalized peritonitis may be subtle, and intuition based on history, clinical experience and the rule out of other conditions may be the primary basis for a diagnosis of peritonitis. Tachycardia, labored respiration, depression, reduced fecal output, and lack of rumen contractions were the most consistently observed signs in this retrospective study. Another report[1] suggested that a diagnosis of peritonitis should be considered when a cow or a bull is febrile and there are no signs specific to other diseases. However, in our study most of the animals with generalized peritonitis had normal or subnormal temperatures. Dehydration was present in all animals. This is usually caused by the massive outpouring of fluids and plasma proteins into the abdomen,[14] but can also be caused by gastrointestinal stasis which can sequester water in the forestomachs. If cattle with generalized peritonitis are treated, the dehydration should be corrected aggressively. Colic signs (kicking at the abdomen, rolling) were not observed in any of the 31 animals presented. Decreased fecal output was found in 27 of 31 cattle. This is most likely caused by ileus, secondary to intestinal adhesions.

Examination of peritoneal fluid was a valuable aid in the antemortem diagnosis of peritonitis. The technique of abdominocentesis in cattle and interpretation

of the results have been described.[6-9,15] Cytologic examination of peritoneal fluid supported the clinical diagnosis in our cases (700-74600 cells/μl). In two cases bacteria were detected in the fluid. While we recommend that abdominocentesis be performed in suspected cases of peritonitis, failure to recover fluid does not discount the possibility of peritonitis. Cattle can wall off infection, therefore the likelihood of recovering fluid increases if the abdominocentesis is done close to the site of suspected inflammation.

The hematologic findings from 31 cattle did little to establish the diagnosis of peritonitis or even to establish a trend. Total white counts ranged from leukopenia to leukocytosis, with or without left shifts. The duration of the condition (based on owner history) had no significant influence on the hematologic data, which was unexpected. We expected that cattle with chronic conditions would have elevated, mature white counts and that acute (and overwhelming) infections would result in leukopenia and degenerative left shifts, but this was not the case. This is difficult to explain but might be due to the small number of the cattle in this study.

Serum biochemical findings were dehydration, loss of electrolytes (sodium, potassium and chloride) and hypoproteinemia (11 cases). This is attributed to the effusion of fluid, electrolytes and plasma protein into the abdominal cavity. In addition, the fluid accumulation or sequestration within the intestines due to ileus may account for fluid and electrolyte losses.[5] Cattle in the early stage of disease may show hyperproteinemia. Animals with normal plasma protein values might have both dehydration and protein loss at the same time.[12] Hypocalcemia (< 8.7 mg/dl), found in 23 cases, may be due to anorexia secondary to the toxic state of peritonitis.[2] Both hyperphosphatemia (> 7.2 mg/dl; 15 animals) and hypophosphatemia (< 4.5 mg/dl; four animals) were found. Elevations of serum phosphorus in cattle are most often associated with impaired renal excretion[5] but may occur in severe toxic states through the release of intracelluar phosphorus associated with cell death or with death of ruminal flora. Hypophosphatemia might be explained by increased losses through either the urine or the saliva or decreased intake. Elevations of serum creatinine and urea nitrogen were found in 11 animals. This is the renal response to the dehydration caused by the massive exudative loss of fluid into the peritoneal cavity with the resultant hypovolemia and decreased renal filtration.[4]

Fibrinogen, an inflammatory protein, was determined in five animals. Four of the five cows had blood fibrinogen concentrations above 500 mg/dl. Although fibrinogen production is a non-specific reaction to inflammatory insults in cattle,[13] elevated fibrinogen levels, combined with other diagnostic parameters and physical findings, can help the veterinarian reach a diagnosis of peritonitis.

The important causes of peritonitis in this study were the perforation of abdominal organs, mainly the abomasum, or as a complication of intra-abdominal surgery. The cause of generalized peritonitis might be difficult to determine even after postmortem examination. Organized fibrinous adhesions inside the omental sling and between the loops of the bowel and mesentery may easily conceal intestinal perforation. This makes determination of the exact inciting cause impossible. Peritonitis outside the omental sling can usually be narrowed down to traumatic reticuloperitonitis, perforating abomasal ulcer or ruptured liver abscess. In the 11 cases in which the cause could not be determined, the lesions were too overwhelming to allow for identification of the source.

Conclusions

Diagnosis of peritonitis is a challenge for the practicing veterinarian. The clinical signs are similar to several other diseases. Clinicopathologic findings are not specific but may direct the clinician toward the diagnosis. Abdominocentesis is probably the best diagnostic test but suffers from low specificity due to the cow's ability to wall off the infection sites. The causes of advanced stages of peritonitis might be difficult to diagnose, even at postmortem examination. Controlled studies are needed to establish diagnostic and prognostic criteria for peritonitis in cattle.

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CVM UPDATE

FDA, Center for Veterinary Medicine

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February 5, 1999

Update on Human Food Safety of BST

FDA's Center for Veterinary Medicine (CVM) has reexamined the human food safety of recombinant bovine somatotropin (rbST) in response to recent inquiries about the safety of this product. FDA's CVM approved Monsanto Company's rbST product, Posilac® in November 1993 after a comprehensive review of the product's safety and efficacy, including human food safety. CVM has issued a detailed report based on a careful audit of the human food safety sections of this approval. CVM's finding upholds the Agency's original conclusion that milk from cows treated with rbST is safe for human consumption.

The new concerns about the safety of Posilac®, currently the only rbST product approved for increasing milk production in dairy cattle in the U.S., were stimulated by the product's review for approval in Canada. In April 1998, while the review process was underway, the Health Protection Branch (HPB) of Health Canada prepared an internal memorandum, entitled "rBST (Nutrilac) 'GAPS Analysis' Report," which was critical of the review method used by the HPB, and identified areas of human food safety concern.

In particular, the Canadian report claimed that a 90-day oral toxicity study in rats had been "misreported" by FDA, and cited allegations of significant absorption of oral rbST based on serum antibody levels in the rats, and toxicity to the rats. Both the memorandum and the circumstances under which it was made public became highly controversial in Canada.

Following the publication of the Canadian document several groups and individuals in the United States raised questions about the safety of milk from rbST-treated cows. In response to these concerns, CVM prepared a "Report on the Food and Drug Administration's Review of the Safety of Recombinant Bovine Somatotropin." The Report affirmed the original review of the 90-day rat oral toxicity study, which concluded that there were no biologically significant observed effects in either the thyroid or the prostate.

In addition, CVM conducted a review of the report cited by Health Canada of the antibody response to oral rbST. While CVM concurred that oral exposure to high doses of rbST results in antibody production, there is no evidence for biologically significant absorption of intact rbST from the gastrointestinal tract.

The "Report on the Food and Drug Administration's Review of the Safety of Recombinant Bovine Somatotropin" is available on the CVM's Internet Home Page, which is located at http://www.fda.gov/cvm.

Issued by: *FDA, Center for Veterinary Medicine, Office of Management and Communications, HFV-12 7500 Standish Place, Rockville, MD 20855*
Telephone: (301) 594-1755
FAX: (301) 594-1831
Internet Web Site: http://www.fda.gov/cvm