Clinico-Biochemical Parameters, Treatment, and Prognostic Indicators of Peritonitis in Buffaloes

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Abstract: *Background*: The present study aimed to describe clinical findings, hemato-biochemical changes, response to medical treatment, and outcomes for buffaloes suffering from peritonitis. Another objective of this study was to determine the prognosis of the disease.

Methods: The study was conducted on 25 buffaloes suffering from peritonitis and presented to the Large Animal Hospital of Guru Angad Dev Veterinary and Animal Science University, Ludhiana, Punjab, India. The buffaloes were subjected to detailed clinical examination. Hematological, biochemical, and peritoneal fluid analysis was carried out along with treatment.

Results: Peritonitis was more prevalent in pregnant buffaloes, being septic peritonitis in 60% of cases. The important clinical findings were depression, anorexia, reduced water intake, scanty feces, dehydration, and absence of rumen motility. The hematological findings were hemoconcentration, left shift, and toxic changes in neutrophils. Biochemical analysis revealed increased total bilirubin, AST, ALP, GGT, glucose, triglycerides, BUN, creatinine, lactate, fibrinogen, and rumen chloride, whereas albumin, fibrinogen ratio, potassium, chloride, calcium, and phosphorus were decreased. Abnormal peritoneal fluid changes were altered physical parameters and the presence of degenerated neutrophils, bacteria, and gut contents. Nucleated cell count and total peritoneal fluid protein were not reliable indicators of peritonitis. The absence of rumen motility, marked left shift, toxic changes in neutrophils, higher BUN, lower potassium (≤3.6 mmol/L), and unfavorable peritoneal fluid changes were the negative prognostic signs. Treatment with broad-spectrum antibiotics and supportive therapy led to a recovery in about 50 percent of cases, with diffuse peritonitis cases being unresponsive. Long time survival rate was good, and there was no recurrence.

Conclusion: The prognosis of peritonitis in buffaloes has to be precisely assessed on the basis of clinical, hemato-biochemical, and peritoneal fluid alterations. Standard classification of transudate and exudate did not apply in the majority of buffaloes with peritonitis.

Keywords: Buffalo, biochemistry, peritonitis, peritoneal fluid, prognosis, treatment.

INTRODUCTION

Peritonitis means inflammation of the peritoneal cavity and is one of the main causes of animal deaths. Peritonitis may be localized or generalized and may occur as a primary or secondary part of a specific disease. As a primary disease, it commonly results from injury of the serosal surface of the alimentary tract [1]. Less commonly, there is perforation of the abdominal wall from the exterior by penetrating foreign bodies or intra-peritoneal injection of non-sterile solutions/irritating substances, or exploratory laparotomy [1,2]. The perforated abomasal ulcers may also result in localized or diffuse peritonitis [3-5]. The other causes include abomasal volvulus or/ and rupture, necrosis of the intestine after intestinal obstruction [1,6,7], as well as rectal and jejunal tears [8]. The clinical signs resulting from toxemia and gastrointestinal paresis are non-specific. The signs include abdominal rigidity, abdominal distension, sclera hyperemia, fever, anorexia, decrease in milk production, ruminal atony, scanty feces, abdominal pain, and there may be diarrhea in chronic cases [1,9,10].

The available literature about hemato-biochemical parameters of peritonitis in buffaloes is scarce and involves only a few parameters [5]. The data on the treatment of peritonitis is also scarce. The production performance of buffaloes in relation to the long-term

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outcome of this disease has not been evaluated previously, and the data about prognostic indicators are lacking. Thus, the present study described signalment, clinical findings, hemato-biochemical changes, response to medical treatment, and outcome in buffalo cases suffering from peritonitis. Another objective of this study was to determine prognostic factors associated with the outcome of the disease, fetal survival, and milk production in the current and subsequent lactation.

MATERIALS AND METHODS

Selection of Animals and Criteria for Diagnosis of Peritonitis

The study was conducted on 25 buffalo cases suffering from peritonitis. The study was performed in compliance with the institutional ethical guidelines. All owners gave their consent for the animals to be included in the study and undergo the testing procedures. The diagnosis of peritonitis was made based on clinical examination and peritoneal fluid examination [5,11]. Before predicting the diagnosis, traumatic reticuloperitonitis and perforated abomasal ulcer were ruled out by physical examination and other ancillary tests like radiography and ultrasonography.

Signalment, Anamnesis, and Physical Examination

Data related to age, sex, parity, calving, pregnancy status, and duration of illness were recorded in all the buffaloes, along with the detailed history of the disease. Each animal was subjected to a general physical examination and a special examination of the gastrointestinal system [12,13]. As previously described by us, each animal was subjected to rectal examination [14]. The rectal examination was performed for rectal mucosa, the quantity of feces in the rectum, consistency of feces, fecal color, rumen size and fill, movement of the hand, intestines, and other abnormal findings. Fecal samples were examined for fecal occult blood by commercially available HEMOSPOT test cards (Crest Biosystems, A Division of Coral Clinical Systems, Goa, India) based on the standard Guaiac method. The fecal samples negative for occult blood were re-examined after 24 and 48 hours [15].

Laboratory Analyses and Diagnostic Procedures

The blood samples (2ml) collected in EDTA coated vials (Accuvete-PLUS, Quantum Biologicals Pvt. Ltd.) were used for the determination of hematological parameters by automatic hematology analyzer (ADVIA ® 2120 Hematology system, Siemens Healthcare Diagnostics Inc., USA). The differential leukocytic count was performed manually in a blood smear stained by Leishman stain [16]. A thorough examination of stained blood smear was also done to determine left shift and toxic changes in neutrophils if any. Serum was used to estimate biochemical parameters except for glucose and fibrinogen. For glucose and fibrinogen estimation, blood samples were collected in sodium fluoride and sodium citrate coated vials (Accuvote Disposables), respectively. The concentration of total globulin was calculated by subtracting the albumin concentration from the total protein concentration. Fibrinogen was estimated with the help of a handheld refractometer [16]. The fibrinogen ratio was calculated by subtracting the fibrinogen value from the total protein concentration and then dividing that value by fibrinogen concentration. VITROS DT-II Chemistry system (Ortho-Clinical Diagnostics, Johnson and Johnson Company) was used to estimate other biochemical parameters.

Rumen fluid samples were collected as described by Hussain *et al.* [13]. Rumen fluid samples were evaluated for pH and chloride concentration. Rumen chloride was estimated after filtering the rumen fluid through a double layer muslin cloth and then centrifuging. Abdominocentesis was done in all the buffaloes [11]. Representative peritoneal fluid samples were collected in EDTA coated vials by free hand centesis using a 16-18 gauge, 1.5-inch-long hypodermic needle. The peritoneal fluid was evaluated for volume, color, consistency/turbidity, and specific gravity. Specific gravity was measured with a handheld refractometer. Peritoneal fluid samples were subjected to cytological examination [11]. White blood cell count (WBC), neutrophil count, and total protein were estimated by the same methods as used for blood.

Reticular radiographs were taken in right lateral recumbency as described [17]. Ultrasonography of the abdomen was performed with Wipro GE Logiq III Expert/ Aloka 500V in real time B mode and B+M mode with a 3.5/5.0 MHz convex transducer by standard procedure [18].

Treatment and Follow-Up

Out of 25 buffaloes, one died on the day of presentation, while the owner of another buffalo was not willing to treatment due to a poor prognosis. The rest 23 buffaloes were treated with intravenous fluids, antibiotics, and other supportive treatments. Depending

upon the severity of hemato-biochemical and peritoneal fluid alterations, the antibiotics were administered as per our clinic's protocol [3,19,20]. Treatment included intravenous administration of 5-10 liters of normal saline and 5 liters of dextrose normal saline once daily for 3-5 days, one dose of intravenous calcium therapy (450ml of MIFEX, Novartis India Limited, India), 200mL of liquid POTKLOR (containing 20 g of potassium chloride for 3 days) orally, 8-10 ml intramuscular injection of belamyl (B complex liver extract with vitamin B12 injection, Zydus AH, India) for 3-8 days and 100 g charcoal (as anti-bloat agent) once daily till resolution of tympany. Meloxicam (0.5mg/kg, injection Melonex, Intas Pharmaceuticals Ltd, India), administered once daily for three days. Ceftiofur (2.2mg/kg once daily, injection Xnel, Zoetis India Ltd), ampicillin (20mg/kg twice daily, injection Roscillin, Ranbaxy Laboratories Limited), gentamicin (2.2mg/kg once daily, injection E-Gmicin Vet, Facmed Pharmaceuticals Private Ltd, India) and enrofloxacin (5mg/kg once daily, Injection Fortivir, Virbac Animal Health, India) were administered intramuscularly while metronidazole (20mg/kg twice daily, injection Flagyl, Hindustan Agencies, India) was administered intravenously. Follow-up information was obtained from the owners by telephonic conversation. The owners were contacted at 3 days intervals for 15 days, weekly till 2 months, then at regular intervals of three months for a period of 24 months. The owners were questioned about the general health status of the buffaloes and the born calves (for pregnant buffaloes), any effect on milk yield in current and subsequent lactation, and any recurrence of disease in the next pregnancy.

Statistical Analysis

The categorical/qualitative data were presented as frequency. The quantitative data were expressed as mean ± standard deviation. The student's t-test was used to analyze the significance of the difference between survivors and non-survivors. A Chi-square test was used to compare binary variables between survivors and non-survivors. The significance level was kept at $P<0.05$ for all statistical procedures.

RESULTS

History and Clinical and Diagnostic Imaging Findings

The buffaloes suffering from peritonitis were 2–10-year-old females (mean=6.16±1.88). Sixteen buffaloes (64 %) were in 2^{nd}-4^{th} lactation, two (8%) were heifers, four (16%) were in the third lactation, and three (12%) were in 5^{th}-7^{th} lactation. The majority of buffaloes (60%) were in different stages of pregnancy (12 were > 6month pregnant), and 40% were non-pregnant (six had calved <3 months back, three had calved 3.5-10 months back, and one was heifer). The duration of the illness ranged from 2 to 30 days (mean 11.92±8.1, median=10 days), being less than a week in 32%, 1-2 weeks in 32%, >2-3 weeks in 28%, and 30 days in 8% buffaloes. The history and clinical examination findings are presented in Tables **1-2**. Sixty percent of buffaloes had complete anorexia, and others had chronic inappetence. The majority of buffaloes had a history of fever followed by reduced or scanty defecation. The fall in milk yield was sudden in 86% of lactating buffaloes, and 40% of buffaloes had at least one episode of tympany. Colic was observed in six (24%) buffaloes. Before referral to our clinic, all except one buffalo had been treated symptomatically.

Table 1: Presentation and Clinical Findings of 25 Buffaloes with Peritonitis

Characteristic	Findings	Number of Animals (%)
Feed intake	Reduced	10 (40)
	Absent	15 (60)
Water intake	Normal	08 (32)
	Reduced	17 (68)
History of fever	Present	18 (72)
	Absent	07 (28)
History of abdominal pain	Present	06 (24)
	Absent	19 (76)
History of tympany	Recurrent	02 (08)
	Persistent	05 (20)
	Absent	15 (60)
	Once	03 (12)
Reduction in milk yield[b]	Sudden	02 (14)
	Gradual	12 (86)
Defecation	Reduced	08 (32)
	Scanty	09 (36)
	Absent	07 (28)
	Normal	01 (04)

[b]Only 14 animals were lactating.

Clinically, the majority of buffaloes were depressed, dehydrated, and had congested mucous membranes, tachypnea, and atonic or hypomotile rumen with variable rumen consistency (Table **2**). Pneumoperitoneum was observed in three buffaloes,

Table 2: Clinical Examination Results of 25 Buffaloes with Peritonitis

Characteristic	Finding	Number of Animals (%)
General condition	Alert	11 (44)
	Disturbed	14 (56)
Visual examination	No abnormality	14 (56)
	Mild distension on left side	04 (16)
	Moderate distension on left side	05 (20)
	Severe distension on left side	01 (04)
	Bilateral distension	01 (04)
Mucous membrane	Normal	08 (32)
	Congested	15 (60)
	Anemic	02 (08)
Dehydration	Not appreciable	03 (12)
	Mild	09 (32)
	Moderate	12 (48)
	Severe	01 (04)
Temperature	Low (<37.2^{0}C)	01 (04)
	Normal (37.2-38.9^{0}C)	13 (52)
	Increased (>38.9^{0}C)	11 (44)
Heart rate/minute	Normal (60-80)	19 (76)
	Low (<60)	03 (12)
	Slightly increased (81-90)	03 (12)
Respiration rate/minute	Normal (15-25)	11 (44)
	Slightly increased (26-35)	10 (40)
	Moderately increased (36-45)	03 (12)
	Severely increased (>45)	01 (04)
Rumen motility/2 minutes	Normal (3)	03 (12)
	Reduced (1)	04 (16)
	Reduced (2)	04 (16)
	Absent	13 (52)
	Increased (>3)	01 (04)
Rumen consistency	Doughy	11 (44)
	Mushy	11 (44)
	Hard	03 (12)

and fluid splash on swinging palpation of the abdomen was recorded in three buffaloes. Rectal examination revealed reduced or scanty pasty consistency feces, sticky rectal mucosa, and normal intestines in the majority of buffaloes (Table **3**). On rectal examination, peritoneal fluid was appreciable in two buffaloes, and rough peritoneum was palpable in one buffalo. Vomiting was observed in two buffaloes (8%), and a fecal occult blood test (FOBT) was positive in ten (40%) buffaloes.

All animals were negative for foreign body syndrome based on the radiography findings, although non-potential foreign bodies were observed in five buffaloes (20%). Ultrasonography (n=20) revealed absence of reticular motility (n=12, 60%), increase in peritoneal fluid (n=11, 55%) and diffuse peritonitis (n=4, 20%) and fibrinous peritonitis (n=4, 20%). The mean rumen pH was 6.83±0.5 (median= 7), and the rumen chloride was increased in all the buffaloes.

Table 3: Rectal Exploration Findings in 25 Buffaloes with Peritonitis

Characteristic	Finding	Number of Animals (%)
Rumen size	Normal	13 (52)
	Moderately distended	07 (28)
	Severely distended	05 (20)
Intestines	Normal	18 (72)
	Distended	07 (28)
Rectal mucosa	Normal	09 (36)
	Sticky	14 (56)
	Bleeds easily	02 (08)
Hindered Hand movements	No	17 (68)
	Yes	08 (32)
Fecal quantity in rectum	Normal	04 (16)
	Reduced	02 (08)
	Scanty	15 (60)
	Absent	06 (24)
Fecal color*	Normal	12 (48)
	Black	07 (28)
Fecal consistency*	Normal	07 (28)
	Pelleted	01 (04)
	Hard	04 (16)
	Loose	02 (08)
	Pasty	05 (20)

*Feces were absent in six buffaloes.

Hematobiochemical Findings

The hematological and biochemical parameters are presented in Table **4**. The mean hemoglobin and lymphocyte count were within the normal reference range, while hematocrit, WBC, and neutrophil count were higher than the reference values. The WBC was 7400-10000 in six (24%), 10001-12000 /µL in three (12%), 12001-16000/µL in ten (40%), 16001-20000 in four (16%) and >20000/µL in two (8%) buffaloes. The toxic changes in neutrophils were observed in 76% of cases (mild in eight, moderate in eight, and severe in three), and left shift was observed in 20 cases (80%), being mild in six (24%), moderate in three (12%) and marked in eleven (44%). The mean levels of cholesterol, triglycerides, glucose, total bilirubin, aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), fibrinogen, blood urea nitrogen (BUN), creatinine, lactate, and rumen chloride were higher than the reference values, while albumin, fibrinogen ratio, potassium, chloride, calcium and phosphorus were lower than the reference values. The other biochemical parameters were within the normal reference range values.

Table 4: Haematological and Biochemical Analytes of Blood in 25 Buffaloes with Peritonitis (Mean ± S.D)

Measurement	Reference range	Findings
Hemoglobin (g/dl)	8.5-12.2	10.57±2.05
Haematocrit (%)	22-33	35.2±7.7
WBC (/µl)	4900-12000	13605±4596
Neutrophil (/µl)	1800-6300	9791±3938
Lymphocyte (/µL)	1600-5600	3733±1963
Cholesterol(mg/dL)	65-220	61.8±30.3
Triglyceride (mg/dL)	0-14	34.0±21.11
Glucose (mg/dL)	45-75	102.2±46.8
Total bilirubin (mg/dL)	0.01-0.5	2.19±2.8
AST (U/L)	78-132	384.2±263
ALP (U/L)	27-107	108±88.2
GGT (U/L)	6.1-17.4	63.7±90
Total protein (g/dL)	5.7-8.1	7.19±0.94
Albumin (g/dL)	3.03-3.55	2.92±0.43
Globulin (g/dL)	2.9-4.9	4.27±0.74
Fibrinogen (mg/dL)	0.2-0.7	0.8±0.45
Fibrinogen ratio	>15	10.87±7.71
BUN (mg/dL)	6-27	36.96±20.8
Creatinine(mg/dL)	1-2	2.44±1.08
Lactate (mmol/L)	0.6-2.2	7.57±3.64
Sodium (mmol/L)	132-152	136.8±8.23
Potassium (mmol/L)	3.9-5.8	3.55±0.74
Chloride (mmol/L)	95-110	88.24±0.74
Calcium (mg/dL)	9.7-12.4	8.04±1.23
Phosphorus (mg/dL)	5.6-6.5	5.3±2.54
Magnesium (mg/dL)	1.7-3.0	3.12±1.05
Rumen chloride	10-25	47.4±14.3

Peritoneal Fluid Examination Findings

The collected peritoneal fluid ranged from 4 ml to >2 liters. More than one liter of turbid fluid was drained from the post xiphoid site in two buffaloes. In another buffalo, foul smelling >2litre pus, like fluid was obtained from every site tapped in the abdomen. In one more buffalo that became recumbent soon after presentation, there was massive peritoneal fluid, and free-flowing

peritoneal fluid could be collected on paracentesis of even lateral abdomen. The peritoneal fluid was crystal clear in 44% and yellow in 32% cases (Table **5**). The consistency was watery in 40% and turbid in 60% of cases. Peritoneal fluid was off-smelling in three (12%) buffaloes. The specific gravity was increased in 80% of cases, while WBC was <5000 in 60% of cases. The total cell count of peritoneal fluid ranged from 350 to >100000/µL. The majority (80%) of buffaloes had a neutrophil count of >50%, but the total protein was > 3g/dL in 60% of cases only.

Table 5: Peritoneal Fluid Analysis Results of 25 Buffaloes with Peritonitis

Characteristic	Finding	Number of Animals (%)
Colour	Crystal clear	11 (44)
	Yellow	08 (32)
	Reddish	04 (16)
	Pus-like	02 (8)
Consistency	Watery	10 (40)
	Slightly turbid	07 (28)
	Turbid	08 (32)
Volume	Up to 6 ml	08 (32)
	>10 ml	17 (68)
Specific gravity	1.005-1.015	05 (20)
	1.016-1.025	05 (20)
	1.026-1.040	15 (60)
WBC (/µL)	<3000	10 (40)
	3001-5000	07 (28)
	6000-7000	05 (20)
	>12000	03 (12)
Neutrophils (/µL)	<1500	07 (28)
	1501-3000	07 (28)
	4101-6000	05 (20)
	>6000	06 (24)
Neutrophils (%)	<50	02 (08)
	50-80	11 (44)
	81-90	08 (32)
	>90	04 (16)
Total protein (g/dL)	0.9-3.0	10 (40)
	3.1-4.5	08 (32)
	4.6-6.0 5	07 (28)
Bacteria	Absent	10 (40)
	Present	15 (60)

The abnormal findings on cytological examination of peritoneal fluid included neutrophilia, variable numbers of degenerated neutrophils, the aggregate of macrophages, bacteria, and gut contents (Figures **1-3**). In some cases, fibroblasts and mesothelial cells were also observed. Microscopic examination of peritoneal fluid smear revealed bacteria in 60% of cases. Bacteria were cocci in six (24%) samples, bacilli in three (12%), both cocci and bacilli in six (24%), and additionally other shapes in two samples. The bacteria were seen as free or phagocytosed by neutrophils (Figures **4-5**). Furthermore, bacteria were observed in isolated fields,

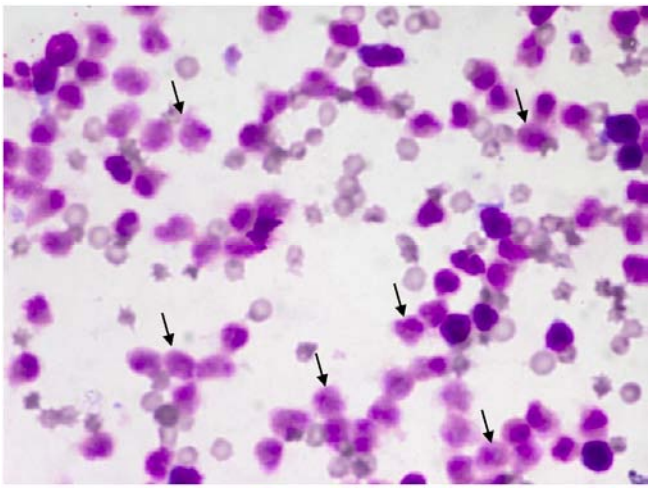

Figure 1: Stained peritoneal fluid (Leishman stain) showing a large number of degenerated neutrophils (arrows). The buffalo was 8 months pregnant, having inappetence and scanty defecation from 20 days. The vital parameters were within the normal reference range except rectal temperature (40^0C). WBC was 13750/µL with 84% neutrophils. Left shift and toxic changes in neutrophils were mild. The buffalo recovered after 8 days of treatment.

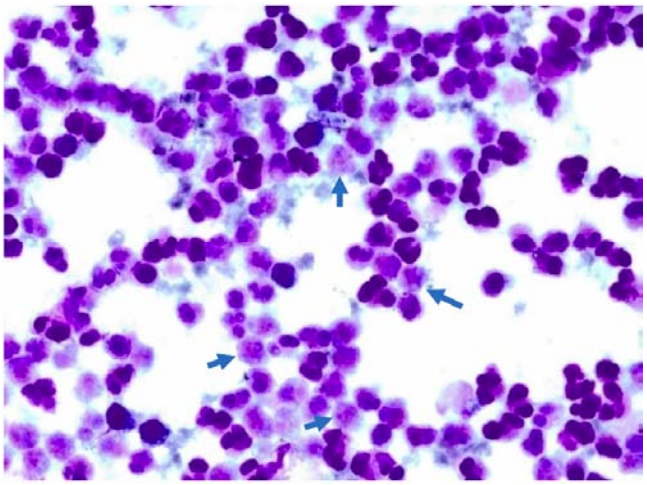

Figure 2: Stained peritoneal fluid (Leishman stain) showing massive neutrophilia along with degenerated neutrophils (arrows). The buffalo was ill for 10 days having anorexia, gradual fall in milk yield, fever, scanty defecation, bradycardia, and neutrophilia without any toxic changes in neutrophils or left shift. The buffalo recovered after 11 days of treatment.

as chains or as aggregates. Severe sepsis (n=4) was characterized by massive neutrophilia and markedly degenerated neutrophils with engulfed bacteria along with a high count of activated macrophages and mesothelial cells (Figure **6**). The presence of gut contents in the peritoneal fluid was observed in three cases. The gut contents included plant fiber and actinomycete-like organisms (Figures **7-8**). Adhesive peritonitis was diagnosed in four (Figure **9**) and overwhelming peritonitis in two cases (Figure **10**).

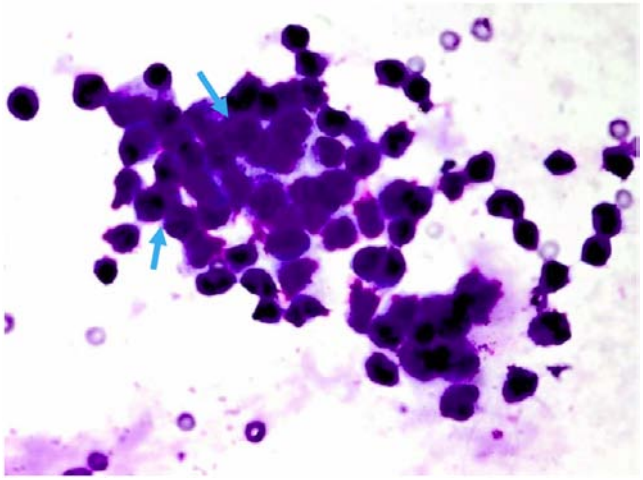

Figure 3: Stained peritoneal fluid (Leishman stain) showing the aggregate of neutrophils, macrophages, and mesothelial cells (arrows). The buffalo (9.5month pregnant) had inappetence, mild ruminal tympany, and melena from 3 days. The vital parameters and blood picture were normal, and the buffalo recovered uneventfully after 6 days of treatment.

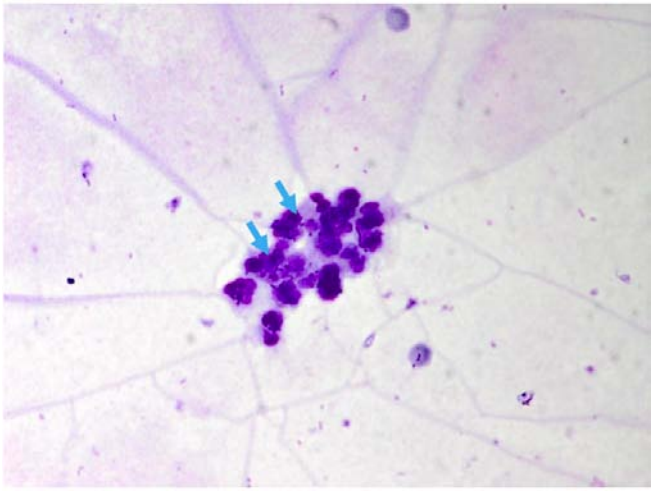

Figure 4: Stained peritoneal fluid (Leishman stain) showing a bunch of neutrophils with engulfed bacteria (arrows). The buffalo had anorexia, fever, and a sudden reduction in milk yield from 2 days. There was neutrophilic leukocytosis, hypoproteinemia, and hyperlactatemia. There was a slight response to treatment, but the buffalo died after 32 days of treatment.

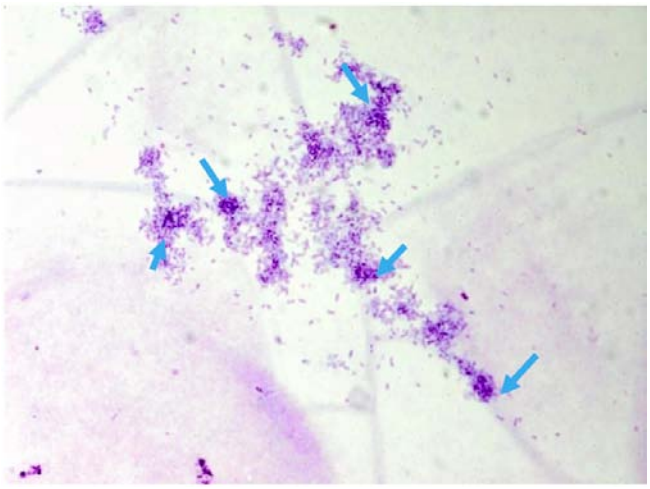

Figure 5: Stained peritoneal fluid (Leishman stain) showing free bacterial colonies (arrows). The buffalo (6month pregnant) had been ill for 20 days and had a history of inappetence, colic, fever, melena, and scanty defecation. There was tachycardia, atonic rumen, and neutrophilic leukocytosis (WBC=14600), marked left shift and toxic changes in neutrophils, and elevated lactate levels (8.6mmol/L). The buffalo died after 3 days of treatment.

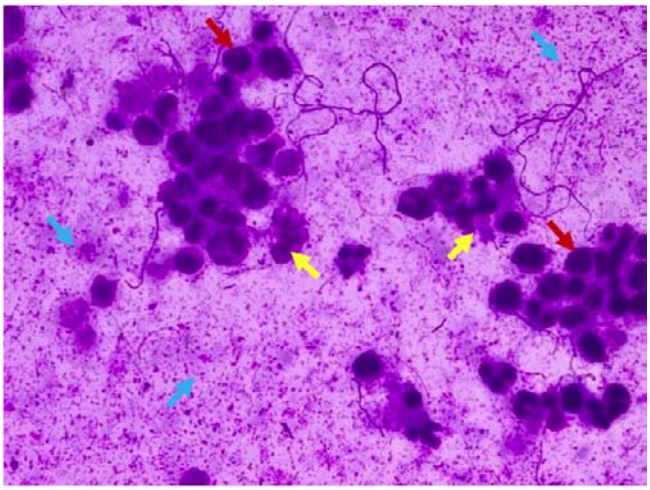

Figure 6: Stained peritoneal fluid(Leishman stain) suggestive of a severe sepsis-large number of bacteria (blue arrows), intact (red arrows), and degenerated neutrophils(Yellow arrows). The buffalo had anorexia, fever, and colic from 12days, atonic rumen, heart rate of 71bpm, WBC of 13260/µL, a moderate degree of toxic changes in neutrophils and marked left shift, low albumin (2.9g/dL), azotemia (BUN=68 mg/dl, creatinine=2.6mg/dL), hypokalemia (2.4mmol/L) and severe hyperlactatemia (9.4mmol/L). The buffalo died after 5 days of treatment.

Treatment and Follow Up

Medical treatment was initiated in 23 buffaloes, which included the administration of intravenous fluids, antibiotics, and other supportive therapy. Four (17%) buffaloes died within three days of the treatment, six (26%) after 4-7 days, and one after 32 days of treatment. The buffalo, whose owner was not willing to

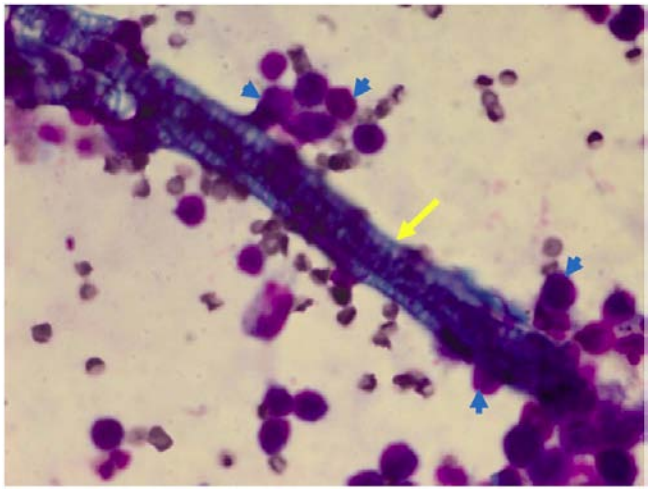

Figure 7: Stained peritoneal fluid (Leishman stain) showing plant fiber (Yellow arrow) surrounded by neutrophils (Blue arrows). This 6-month pregnant heifer had anorexia from 4 days, scanty feces, atonic rumen, normal WBC (7700/µL) and neutrophil count, marked left shift, low albumin, and severe hyperlactatemia (9.1 mmol/L). The buffalo died after 4 days of treatment.

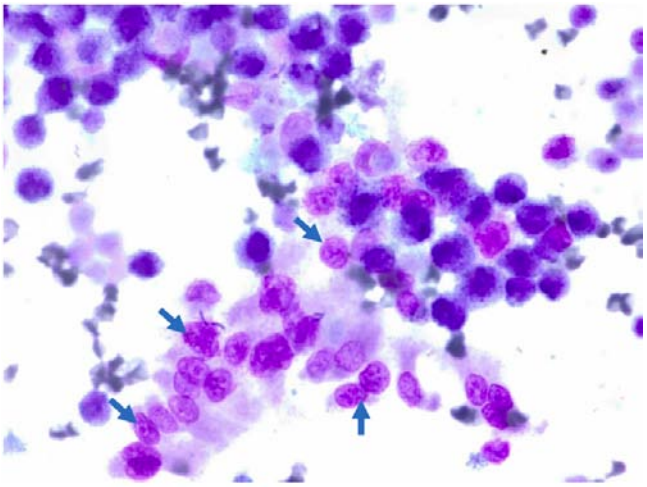

Figure 9: Stained peritoneal fluid (Leishman stain) showing the high count of fibroblasts (Arrows) suggestive of adhesive peritonitis. The buffalo had been ill for 7 days with inappetence, scanty defecation, gradual fall in milk yield, rumen was motile with normal vitals. WBC was 14812/µL, with mild left shift but no toxic changes in neutrophils. The buffalo recovered after 14 days of treatment.

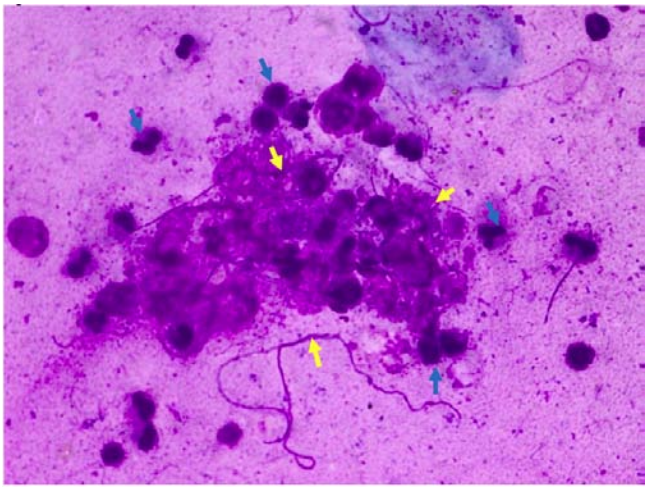

Figure 8: Stained peritoneal fluid (Leishman stain) showing high protein background, actinomycete-like organisms (Yellow arrows), and neutrophils (Blue arrows). The buffalo had anorexia, loss of defecation, fever, heart rate of 75bpm, WBC of 14270/µL with a mild degree of toxic changes in neutrophils and mild left shift, low albumin (2.5g/dL), and hyperlactatemia (7.1mmol/L). The buffalo died after 14 days of treatment.

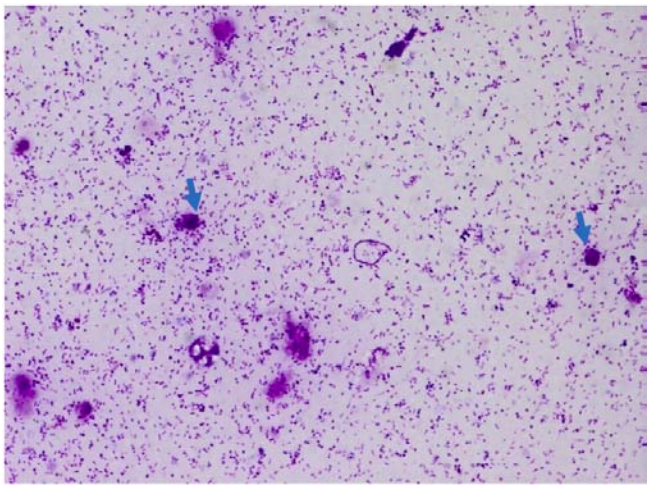

Figure 10: Stained peritoneal fluid (Leishman stain) showing overwhelming bacteria and few severely degenerated neutrophils (arrows). The buffalo had anorexia, fever, and melena for 10days. There was neutrophilic leukocytosis with a moderate degree of toxic changes in neutrophils and marked left shift, azotemia, and severe hyperlactatemia (12.8mmol/L). The buffalo died after 2 days of treatment.

treatment, died after a period of 14 days. Six (26%) buffaloes recovered after 6-9 days, five (22%) after 11-15 days, and one buffalo recovered after 23 days (Table **6**). All the survivors were healthy at the time of the last feedback. The pregnant buffaloes had calved normally, and some non-pregnant buffaloes had become pregnant and then calved normally during follow-up. After recovery, there was no significant effect on the milk yield in the current or subsequent lactation.

Survival Analysis and Prognostic Indicators

The age, duration of illness, clinical signs, and clinical examination parameters did not differ significantly ($p<0.05$) between survivors and non-survivors. None of the buffaloes with pneumo-peritoneum survived. Hence peritoneal tympany could be regarded as a poor prognostic sign. Rumen motility was absent in 13 buffaloes, and only two of them recovered after a period of 12 and 23 days,

Table 6: Antibiotic Combinations used for the Treatment of Peritonitis in 23 Buffaloes and their Outcome

Case no.	Duration of illness (Days)	Septic peritonitis	Antibiotics	Outcomes	Outcome Period
1	10	No	CAM	Recovered	12
2	5	No	CAM	Died	1
3	20	Yes	CAM	Died	4
4	5	Yes	CAM	Recovered	15
5	20	No	CAM	Recovered	8
6	30	No	CAM	Died	3
7	4	Yes	AGM	Died	4
8	7	Yes	AGM	Died	6
9	12	Yes	AGM	Died	5
10	3	No	AG	Recovered	6
11	7	No	AG	Recovered	14
12	8	Yes	AEM	Recovered	6
13	15	Yes	AEM	Died	6
14	10	Yes	AEM	Died	2
15	15	Yes	AEM	Recovered	7
16	20	Yes	AEM	Died	3
17	4	No	AEM	Recovered	15
18	20	Yes	AE	Recovered	8
19	2	Yes	AE	Died	32
20	20	Yes	AE	Recovered	23
21	6	No	AE	Died	7
22	10	No	AE	Recovered	11
23	3	No	AE	Recovered	9

*In column 3, C=Ceftiofur, A=Ampicillin, M=Metronidazole, G=Gentamicin, E=Enrofloxacin.

respectively. A significantly ($p<0.05$) higher percentage of non-survivors had an absence of rumen motility than the survivors. Marked left shift, and toxic changes in neutrophils were observed significantly ($p<0.05$) in higher percentage of non-survivors than the survivors. The non-survivors had significantly ($p<0.05$) higher BUN (46±20.8 vs. 27.3±16.4) than the survivors. Although, potassium did not differ ($P=0.09$) between survivors and non-survivors (3.8±0.75 vs. 3.3± 0.66), significantly ($P<0.05$), a higher percentage of non-survivors had potassium concentration ≤ 3.6mmol/L than the survivors.

The physical characteristics and quantitative parameters of peritoneal fluid did not differ significantly ($P<0.05$) between survivors and non-survivors. Out of 15 buffaloes with septic peritonitis, only five survived. The non-survivors had a greater degree of degenerative changes in peritoneal fluid neutrophils than the survivors. None of the buffaloes with diffuse peritonitis, severe sepsis, overwhelming peritonitis, and the presence of gut contents in peritoneal fluid survived.

DISCUSSION

Peritonitis was diagnosed based on peritoneal fluid examination supported by clinical examination [11,21]. Due to melena, loss of defection, and colic, the important abdominal disorders to be ruled out were perforated abomasal ulcer, abomasal volvulus, intussusception, and hemorrhagic bowel syndrome [1,22]. Hemorrhagic bowel syndrome and abomasal volvulus have not been reported in the area of this study, but intussusception and abomasal ulceration are prevalent [4,6,23]. These disorders were ruled out by clinical evaluation and other ancillary tests like ultrasonography. However, type 1 abomasal ulceration could not be ruled out with certainty, and it was assumed to be secondary ulceration. Type 1 ulcers are reported to cause no or mild clinical signs [15,24] and hence were less likely to result in the clinical signs observed in the present study. Similar to an earlier study [15], retesting of fecal samples after storage did not increase their positivity for occult blood. Like the present study, other abdominal disorders have been reported to result in secondary melena like traumatic

reticulo-peritonitis [20], traumatic pericarditis [25], and reticular diaphragmatic hernia [26]. These disorders were also ruled out in the present study. Type 3 abomasal ulcer could not be ruled out as we did not examine the abomasum directly.

The majority of buffaloes were pregnant (60%), and 16% had calved <2 months back. So, pregnancy seemed to be a greater risk factor than recent calving in the present study. The clinical findings were non-specific and resembled other abdominal disorders like traumatic reticuloperitonitis, abomasal ulceration, and reticular diaphragmatic hernia [4,26,27], but were similar to published findings in cattle and buffaloes [5,11,28]. It is important to mention that the signs of complete anorexia, colic, tympany, loss of defecation, depression, dehydration, tachycardia, and tachypnoea were less pronounced than reported for peritonitis in cattle [11]. Further, in contrast to peritonitis in cattle [11] higher percentage of buffaloes in this study had normal rumen consistency and normal abdominal contour. The main complaint by the owners in the majority of cases was fever and scanty defecation. History of fever, a common sign in the initial stages of peritonitis, corroborates with observations of Hosgood and Salisbury [29]. The fecal output can be normal or reduced and stale in peritonitis because of prolonged transit [10,11]. Before referral to our clinic, the majority of animals had been treated with antibiotics along with rumen tonics, liver tonics, anti-bloat compounds, calcium infusion, and/or purgatives. It is also worthwhile to mention that in some cases, previous treatment had resulted in mild improvement in appetite and fecal output, but clinical recovery was not complete. Signs of abdominal pain were observed less frequently than other abdominal disorders in bovines like traumatic reticuloperitonitis, intestinal obstruction, caecal dilatation, omasal impaction, abomasal ulcers, and abomasal volvulus [4,6,10,13,22,27,30]. Pneumo-peritoneum was characterized by the presence of gas on tapping of the paralumbar fossa without puncturing the rumen. Pneumoperitoneum not associated with surgery has been reported to be indicative of bacterial peritonitis and the presence of gas-producing bacteria [10]. Like peritonitis, vomiting has been reported to be an infrequent sign in other abdominal disorders like intestinal obstruction [6] and reticular diaphragmatic hernia [26].

The rumen-reticular motility depends upon the excitatory and inhibitory inputs to the gastric center [1,10]. The suspended or reduced rumen motility may be attributed to toxemia, peritoneal adhesions, or generalized gastrointestinal tract atony. Reduced ruminal motility may also be attributed to the observed hypocalcemia. Hypomotility corroborates the earlier findings [11]. In contrast to the present study, Kumar *et al.* [31] reported normal rumen motility in bovines with diffuse peritonitis. The mushy rumen consistency may be attributed to prolonged ingesta retention in the rumen due to ineffective or absent rumen contractions. Rectal examination is a valuable diagnostic procedure in bovine gastroenterology and helps to diagnose many gastrointestinal disorders in bovines like rumen impaction intestinal obstruction, and caecal dilatation [6,14,30], and could also help in the subjective assessment of omasal impaction [13]. In this study, the rectal findings were not diagnostic except in three buffaloes.

The main hematological findings were increased hematocrit and neutrophilia or neutrophilic leucocytosis with left shift and the presence of toxic neutrophils and were similar to earlier reported findings in peritonitis [1,11,32]. The changes in hemogram may be attributed to an immunological response to peritonitis [33]. The mean WBC count was higher than reported for other bovine gastrointestinal disorders like rumen impaction, reticular diaphragmatic hernia, intestinal obstruction, caecal dilatation, and vagal indigestion [6,14,26,30,34] but comparable to that reported for omasal impaction, peritonitis, and traumatic pericarditis [11,13,25].

The increased total bilirubin and other liver enzymes indicated impaired hepatic function and/or hepato-biliary circulation. The possible cause of higher liver enzymes could be degenerative lesions in the peritoneum and liver. The probable explanation for increased glucose is that all these animals were transported to our clinic, which could have triggered the release of stress-related hormones such as cortisol and adrenalin, increasing blood concentrations of glucose [35]. In addition, the stress of the disease could have also resulted in hyperglycemia. The lower cholesterol level may be attributed to inflammatory response and hepatic malfunction impairing the lipid transport [36-38].

Change in serum proteins following tissue destruction and inflammation is expected [39]. Leukogram and peritoneal fluid alterations indicated an inflammatory response in our study. So the expected change in total protein would have been an increase in its concentration. But on the contrary, the total protein concentration was within the normal reference range (5.7-8.1 g/dL). Albumin level was towards the lower side of the reference range and resembled earlier findings in diffuse peritonitis of buffaloes [5]. There

could be two possible reasons for lower total protein and albumin. The First could be third space loss, and another cause could be reduced synthesis of albumin (negative acute phase protein) by the liver. The noted hemoconcentration, yet a tendency for normal total protein, suggested significant third space losses [6]. It has been shown that during inflammation, the synthesis of positive acute phase proteins in the liver increases while the synthesis of negative acute phase proteins such as albumin decreases, and hence albumin concentration may fall in inflammatory and infectious diseases [39,40].

The fibrinogen was elevated due to peritoneal inflammation. The fibrinogen ratio in bovines gives a more realistic picture of increased fibrinogen in inflammatory conditions and rules out any alterations due to dehydration. The lower fibrinogen ratio indicated a marked increase in fibrinogen [16]. In some acute diseases like intestinal obstruction, the fibrinogen and fibrinogen ratio may be misleading and should be interpreted with caution [6]. However, we believe that the fibrinogen and fibrinogen ratio reflected true changes in the disease in the present study.

The main causes for the increase in blood lactate are increased anaerobic metabolic activity and a decrease in hepatic utilization. Under clinical situations, the most common cause of increased lactate is dehydration [6,13,30]. Furthermore, not only hypovolemia or oxygen debt but also metabolic conditions such as hepatic malfunction with decreased lactate uptake may induce hyperlactatemia [41]. In the present study, most cases had multiple organ dysfunctions, as indicated by biochemical analysis. So, the increased lactate had multi-factorial etiology in the present study. The important probable causes could be hypovolemia, reduced hepatic utilization, and decreased renal clearance due to azotemia. Abomasal reflux (indicated by increased rumen chloride concentration) could be the cause of dehydration, hypochloremia and azotemia [1,26,34]. As forage is the main source of potassium, anorexia could be the cause of hypokalemia [42]. Although lower albumin may have caused alterations in the ratios of chloride and potassium, it seems more probable that in peritonitis, abomasal reflux precedes the third space loss. Third space losses may have resulted in low sodium levels, but the compensating renal responses causing water retention may have resulted in about normal sodium concentration [10]. The lower calcium concentration was ascribed to decreased absorption and anorexia [1,10].

Peritoneal fluid analysis has been considered a supplementary aid to the hematological and clinical examination in the diagnosis of bovine abdominal disorders [1,17]. Assessing the volume, cellularity, and protein concentration of peritoneal fluid can give an indication of inflammatory changes in the peritoneal cavity [43,44]. However, there are no definite ranges for WBC and total protein in peritoneal fluid for classifying samples as inflammatory or non-inflammatory, and the values may overlap between normal and severely infected samples [1]. An increased number of peritoneal fluid leukocytes is commonly associated with inflammation [45], but the sensitivity and specificity of this parameter are low due to numerous false positive and false negative results [46]. Peritoneal fluid nucleated cell count of greater than 6000/μLhas been reported to be consistent with a diagnosis of peritonitis in cattle [47-50]. The nucleated cell count criterion did not hold well in the present study. Rather differential cell count of peritoneal fluid was more useful in diagnosing peritonitis as WBC was ≤5000 in 68%cases. The present study's consistent findings on peritoneal fluid examination were increased specific gravity and >50% neutrophils. A total protein concentration of>3g/dL in the peritoneal fluid has normally been used as the cut-off value for exudates established in the classical transudate-exudate system [45]. The reference range for total protein has been redefined to 0.56-4.18 g/dL in peritoneal fluid of healthy cows [51]. However, higher cut-off values might increase the risk of false negatives and therefore lower the specificity of total protein in peritoneal fluid as a measure of peritoneal inflammation, as demonstrated in the present study.

The common cytological abnormalities of peritoneal fluid were an increased number of neutrophils with degenerative or toxic changes and resembled earlier findings of Ziemer [52]. The samples with the presence of bacteria were categorized under septic peritonitis as per Hirsch and Townsend [48]. A probable cause for the presence of gut contents in the peritoneal fluid could be trocarisation of rumen done at the farm level. Overwhelming peritonitis was characterized by the presence of a massive number of bacteria, greater than the cells in peritoneal fluid with markedly degenerated neutrophils. Overwhelming peritonitis, along with the presence of gut contents in the peritoneal fluid, was indicative of a poor prognosis.

In the present study, the treatment of peritonitis was a conservative medical approach with systemic antibiotics, fluid therapy, and supportive care. The

individual buffaloes were treated with different antibiotic combinations based on inflammatory changes in leukogram and peritoneal fluid. All the antibiotics were administered intramuscularly at the standard dose rates. Depending upon the severity of dehydration, all these animals received intravenous fluids. The replacement of fluids is necessary so that adequate tissue perfusion could begin again, kidneys can adequately excrete the metabolic by-products again, and anaerobic metabolic processes can be curtailed. The controlled trials for determining different drug concentrations in peritonitis are not available, and peritoneal lavage is not of much use [1]. Also, the intra-peritoneal administration of antimicrobials has been discouraged as it had no advantage over the use of parental antibiotics. Moreover, it is associated with the risk of local adhesion formation [1].

Smith *et al.* [53] have advocated stabilization of animals with supportive therapy combined with the use of antimicrobials in generalized peritonitis. The specific treatment directed to contain the infection is the use of antimicrobials, and third generation cephalosporin or synthetic penicillin have been reported to be good choices in peritonitis [32]. So, both were used in the present study and were administered parentally for a period of ≤12 days in the majority of animals. From Table **6**, it can be inferred that antibiotic combination did not seem to affect the final outcome, e.g., an equal number of animals treated with ceftiofur, ampicillin, and metronidazole recovered and died. The electrolyte and mineral abnormalities should be identified and corrected. Meloxicam was administered to prevent the synthesis of more inflam-matory mediators and help pain management [32].

The findings with respect to the use of antimicrobials in buffalo peritonitis can be further confirmed by a clinical study with the randomized allocation of antibiotics. Whether supportive therapy had or had not had any effect on the improvement of the animals could not be concluded. The recovery period was 1-2 weeks in the majority of buffaloes. The treatment success rate in the present study was low, but it reflects the clinical reality. However, it is worth mentioning that the treatment success rate was far higher than reported for peritonitis in cattle [11]. Maybe a greater number of animals could have recovered after treatment if they had been diagnosed and treated at an early stage of the disease.

The duration of illness and other clinical parameters did not seem to play a role in prognosis. Rather, the absence of rumen motility was the important negative prognostic sign. Barely detecting rumen motility was associated with a poor prognosis. Marked left shift and toxic changes in neutrophils, higher BUN, and ≤3.6 mmol/L potassium were indicative of poor prognosis. The abnormalities of peritoneal fluid of non-survivors were diffuse and septic peritonitis, overwhelming bacterial infection, massive neutrophilia along with degenerated neutrophils, and the presence of gut contents. Five survivors had bacteria in peritoneal fluid (septic peritonitis), but degenerative changes in neutrophils of peritoneal fluid were mild or absent. In buffaloes with mild inflammatory leukogram and less severe peritoneal fluid changes, the severity of infection could have increased with the passage of time, resulting in death. It is always advisable to take multiple samples at different time intervals or stages of the disease to predict the exact prognosis [26]. Prognosis, as predicted on a single sample basis, holds true for that animal at that point in time and may change subsequently with improvement or worsening of condition and constitution of therapeutic interventions or response to therapy. Therefore, a good prognosis may worsen with time, and a guarded prognosis may improve [26]. No recurrence was recorded during the follow-up period, and there was no significant effect on the milk yield in the current or subsequent lactation.

CONCLUSION

Peritonitis in buffaloes usually occurs during pregnancy but can also occur at any stage of lactation. The main clinical features of peritonitis were fever, anorexia, dehydration and scanty defecation. The hematological analysis revealed hemoconcentration and neutrophilia. Biochemical alterations revealed hepatic and renal malfunction, hypocalcemia, hypokalemia, hypochloremia, hyperlactatemia, and increased rumen chloride concentration. In treatment, the deficient electrolytes and minerals should be corrected in addition to the use of antibiotics and liver tonics. The response to medical treatment was fair. However, for substantiation of the use of antibiotics, studies on a large number of clinical cases with proper design about randomization should be conducted. Long time survival rate was good, and there was no recurrence. The absence of rumen motility marked left shift, toxic changes in neutrophils, higher BUN, lower potassium (≤3.6 mmol/L), and unfavorable peritoneal fluid changes were the negative prognostic signs.

ETHICS APPROVAL

All procedures have been conducted as per the guidelines by Guru Angad Dev Veterinary and Animal

Science University Institutional Animal Ethics Committee (IAEC), constituted as per article number 13 of the CPCSEA rules laid down by the Government of India. The procedures being performed were routine clinical procedures, and all owners gave consent for such procedures. Ethical approval was waived by the IAEC as handling and measurements of the animals were done by qualified veterinarians, and the study was on clinical cases.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest in publishing this manuscript.

DATA AVAILABILITY

All the data are present in the manuscript. However, the rough data may be provided on request.

REFERENCES

[1] Constable PD, Hinchcliff KW, Done SH, Grunberg W. Veterinary Medicine. A textbook on the diseases of cattle, horses, sheep, pigs, and goats. 11th ed. Elsevier, St. Louis, Missouri. 2017.

[2] Cramers T, Mikkelsen KB, Anderson P, Enevoldsen C, Jensen HE. New types of foreign bodies and the effect of magnets in traumatic reticulitis in cows. Vet Rec 2005; 157: 287-289. https://doi.org/10.1136/vr.157.10.287

[3] Hussain SA, Uppal SK, Randhawa CS, Sood NK. Frank exudative peritonitis due to perforated abomasal ulceration in a crossbred cow. Int J Agro Vet Med Sci 2011; 5(5): 447-51. https://doi.org/10.5455/ijavms.20111019032526

[4] Hussain SA, Shafi TA, Nanda A, Sood NK. Meloxicam induced abomasal ulceration in a buffalo. Int J Liv Res 2017; 7(3): 175-180. https://doi.org/10.5455/ijlr.20170124052017

[5] Elgioushy M, Abdelaal A, Gouda S, Noseer E, Hashish EA, Elgami SA. Modulation of biochemical parameters in Egyptian buffaloes affected by diffuse peritonitis. Ital J Anim Sci 2019; 18(1): 963-970. https://doi.org/10.1080/1828051X.2019.1608135

[6] Hussain SA, Uppal SK, Randhawa CS, Sood NK. Bovine intestinal obstruction: Blood gas analysis, serum C reactive protein, and clinical, hematological, and biochemical alterations. J Appl Anim Res 2015; 43: 224-230. https://doi.org/10.1080/09712119.2014.963093

[7] Bhutia CN, Uppal SK, Hussain SA and Sood NK. Bovine intestinal obstruction: Changes in cytological and biochemical parameters of blood and peritoneal fluid. Exp Anim Med Res 2019; 19 (1): 37-41. https://www.animalmedicalresearch.org/Vol.9_Issue-1_June_2019/BOVINE%20%20INTESTINAL%20%20OBSTRUCTION.pdf

[8] Tyler JW, Shore MD, Weaver DM, Croucher S, Shephard L. Surgical management of rectal and jejunal tears associated with dystocia in cow. Vet Rec 1998; 143: 280-281. https://doi.org/10.1136/vr.143.10.280

[9] Fubini S, Divers TJ. Non-infectious diseases of the gastrointestinal tract. In: Rebhun's Diseases of Dairy Cattle [Divers T, Peek S. (eds.)] 2nd ed. Saunders Elsevier, St Louis. 2007.

[10] Smith BP. Diseases of the alimentary tract. Large Animal Internal Medicine. 3rd Edn. Mosby St. Louis Missouri USA 2002.

[11] Hussain SA, Uppal SK. Haemato-biochemical changes and peritoneal fluid cytology in clinical cases of bovine peritonitis. Indian J Anim Res 2014; 48(2): 188-193. https://doi.org/10.5958/j.0976-0555.48.2.039

[12] Rosenberger G. Clinical examination of cattle. Paul Parey, Berlin, Hamburg, Germany. 1990.

[13] Hussain SA, Uppal SK, Randhawa CS, Sood NK and Mahajan SK. Clinical characteristics, haematology, and biochemical analyses of primary omasal impaction in bovines. Turk J Vet Anim Sci 2013; 37: 329-336. https://doi.org/10.3906/vet-1205-31

[14] Hussain SA, Uppal SK. Rumen impaction in buffaloes: A haemato-biochemical study. Indian J Anim Sci 2012; 82: 369-373. https://doi.org/10.5958/0973-970X.2015.00057.7

[15] Hussain SA, Uppal SK, Sood NK. Clinicopathological diagnosis of Type-I abomasal ulceration in cattle and buffaloes. Indian J Vet Pathol 2015; 39: 239-242. https://www.indianjournals.com/ijor.aspx?target=ijor:ijvp&volume=39&issue=3&article=009

[16] Feldman B F, Zinkl JG, Jain NC. Schalm's Veterinary Haematology. 5th ed, Lee and Febiger, Philadelphia 2000.

[17] Hussain SA, Uppal SK, Sood NK and Bhat RR. Peritoneal Fluid Examination: A Potential Tool for the Diagnosis of Bovine Diseases. In: Sreedhar S and Bindumadhuri S. Current Research in Animal Husbandry and Veterinary Sciences (Volume - 3). Integrated Publications, Delhi, India 2021; pp 1-17. https://www.researchgate.net/profile/Papita-Gourkhede/publication/356536286_Complete_Book-min/links/619f94cd43dedf033a7d3eb6/Complete-Book-min.pdf#page=5

[18] Braun U. Ultrasonography of the gastrointestinal tract in cattle. Vet Clin North Am Food Anim Pract 2009; 25: 567-590. https://doi.org/10.1016/j.cvfa.2009.07.004

[19] Kumar A. Diagnostic and therapeutic approach to functional forestomach disorders in cattle buffaloes. Ph.D. Thesis, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, India 2009.

[20] Shah SAH. Studies on prevalence, clinico-haemato-biochemical alterations and therapy of gastrointestinal impaction in dairy animals. M.V.Sc. Thesis, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, India 2010.

[21] Abdelaal A, Floeck M, El Maghawry S, Baumgartner W. Clinical and ultrasonographic differences between cattle and buffaloes with various sequelae of traumatic reticuloperitonitis. Vet Med 2009; 54(9): 399-406. https://doi.org/10.17221/128/2009-VETMED

[22] Braun U, Gerspach C, Nuss K, Hassing M, Hilbe M, Reif C. Clinical and laboratory findings, treatment and outcome in 145 cows with type-2 abomasal ulcer. Res Vet Sci 2019; 124: 366-374. https://doi.org/10.1016/j.rvsc.2019.04.020

[23] Hussain SA, Uppal SK and Sood NK. The prevalence, frequency and topographic distribution of Type 1 abomasal ulcers in water buffalo (Bubalus bubalis): a case-control study. Vet Arhiv 2019; 89(3): 317-329. http://vetarhiv.vef.unizg.hr/papers/2019-89-3-5.pdf

[24] Braun U, Bretscher R, Gerber D. Bleeding abomasal ulcers in dairy cows. Vet Rec 1991; 129(13): 279-284. https://doi.org/10.1136/vr.129.13.279

[25] Hussain SA,Uppal SK, Sood NK, Randhawa CS, Mahajan SK, Bansal BK. Hemato-biochemical and blood acid-base gas alterations in bovine traumatic pericarditis. SKAUST J

Res 2018; 20(1): 43-48. https://www.indianjournals.com/ijor.aspx?target=ijor:skuastjr&volume=20&issue=1&article=007

[26] Hussain SA, Uppal SK, Mahajan SK and Sood NK. Reticular diaphragmatic hernia in water buffalo: Clinical characteristics, hematology, biochemical analytes and prognostic indicators. Comp Clin Pathol 2021; 30: 605-615. https://doi.org/10.1007/s00580-021-03251-y

[27] Braun U, Nuss K, Warislohner S, Reif C, Oschlies C, Gerpach C. Diagnostic reliability of clinical signs in cows with traumatic reticuloperitonitis and abomasal ulcers. BMC Vet Res 2020; 16: 359. https://doi.org/10.1186/s12917-020-02515-z

[28] Abdelaal A, FloeckM. Clinical and sonographic findings in buffaloes *(Bubalus bubalis*) with traumatic reticuloperitonitis. Vet Arhiv 2015; 85(1): 1-9. http://wwwi.vef.hr/vetarhiv/papers/2015-85-1-1.pdf

[29] Hosgood GL, Salisbury K. Pathophysiology and pathogenesis of generalized peritonitis. Problem in Vet Med 1989; 1: 159-167.

[30] Hussain SA, Uppal SK, Randhawa CS, Bhutia CN, Hassan N, Dar LM. Clinical findings and haemato-biochemical alterations in caecal dilatation of buffaloes (*Bubalus bubalis*). Int J Liv Res 2012; 2(3):127-132. http://ijlr.org/issue/clinical-findings-haemato-biochemical-alterations-caecal-dilatation-buffaloes-bubalus-bubalis/

[31] Kumar M, Mohindroo J, Kumar A, Singh SS. Clinical, haemato-biochemical and ultrasonographic features of diffuse peritonitis in bovines. Indian J Vet Surg 2008; 29(1): 34-36. https://indianjournals.com/ijor.aspx?target=ijor:ijvs&volume=29&issue=1&article=011

[32] Fecteau G. Management of peritonitis in cattle. Vet Clin Food Anim Pract 2005; 21: 155-171. https://doi.org/10.1016/j.cvfa.2004.12.007

[33] Zadnik T. A comparative study of the hemato-biochemical parameters between clinically healthy cows and cows with displacement of the abomasum. Acta Vet Beograd 2003; 53(5-6): 297-309. https://doi.org/10.2298/AVB0306297Z

[34] Hussain SA, Uppal SK, Sood NK and Mahajan SK. Clinico-hematobiochemical findings, clinical management, and production performance of bovines with late pregnancy indigestion (Type IV vagal indigestion). Vet Med Int 2014. https://doi.org/10.1155/2014/525607

[35] Holtenius K, Sternbauer K, Holtenius P. The effect of the plasma glucose level on the abomasal function in dairy cows. J Anim Sci 2000; 78: 1930-1935. https://doi.org/10.2527/2000.7871930x

[36] Abeni F, Calamari L, Stefanini L. Metabolic conditions of lactating Friesian cows during the hot season in the Po valley 1. Blood indicators of heat stress. Int J Biometeorology 2007; 52: 87-96. https://doi.org/10.1007/s00484-007-0098-3

[37] Bobe G, Young JW, Beitz DC. Pathology, etiology, prevention, and treatment of fatty liver in dairy cows. J Dairy Sci 2004; 87: 3105-3124. https://doi.org/10.3168/jds.S0022-0302(04)73446-3

[38] Vogel KD, Claus JR, Grandin T, Oetzel GR, Schaefer DM. Effect of water and feed withdrawal and health status on blood and serum components, body weight loss, and meat and carcass characteristics of Holstein slaughter cows. J Anim Sci 2011; 89: 538-48. https://doi.org/10.2527/jas.2009-2675

[39] Saleh MA, Rateb HZ, Misk NA. Comparison of blood serum proteins in water buffaloes with traumatic reticuloperitonitis and sequelae. Res Vet Sci 2008; 85: 208-213. https://doi.org/10.1016/j.rvsc.2007.11.007

[40] Eckersall PD. Proteins, Proteomics, and the Dysproteinemias. In: Kaneko JJ, Harvey JW, Bruss ML, Eds. Clinical Biochemistry of Domestic Animals. 6th ed. New York: Academic Press Inc. 2008; pp. 117-155. https://doi.org/10.1016/B978-0-12-370491-7.00005-2

[41] Vary TC, Siegel JH, Rivkind A. Clinical and therapeutic significance of metabolic patterns of lactic acidosis. Perspect Crit Care 1988; 1: 85-132.

[42] Sattler N, Fecteau G. Hypokalemia syndrome in cattle. Vet Clin North Am Food Anim Pract 2014; 30: 351-357. https://doi.org/10.1016/j.cvfa.2014.04.004

[43] Oehme FW. Cytological examination of the peritoneal fluid in the diagnosis of cattle disease. J Am Vet Med Assoc 1969; 155: 1923-1927.

[44] Oehme FW, Noordsy JL. Examination of peritoneal fluid in deferential diagnosis of bovine disease. Vet Med 1970; 65: 54-59.

[45] Kopcha M, Schultze AE. Peritoneal fluid. Part I. Pathophysiology and classification of non-neoplastic effusions. Compend Contin Educ Pract Vet 1991; 13: 519-526.

[46] Wittek T, Grosche A, Locher LF, Furll M. Diagnostic accuracy of D-dimer and other peritoneal fluid analysis measurements in dairy cows with peritonitis. J Vet Int Med 2010; 24: 1211-1217. https://doi.org/10.1111/j.1939-1676.2010.0548.x

[47] Ducharme NG. Surgery of the bovine forestomach compartments. Vet Clin North Am Food Anim Pract 1990; 6: 3713-97. https://doi.org/10.1016/S0749-0720(15)30867-7

[48] Hirsch VM, Townsend HGG. Peritoneal fluid analysis in the diagnosis of abdominal disorders in cattle - a retrospective study. Can Vet J 1982; 23: 348-354. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1790266/

[49] Smith DF. Bovine intestinal surgery. Part IV. Modern Vet Pract 1985; 66: 277-81.

[50] Ward JL, Ducharme NG. Traumatic reticuloperitonitis in dairy cows. J Am Vet Med Assoc 1994; 204: 874-877.

[51] Wittek T, Grosche A, Locher LF, Alkaassem A, Fürll M. Biochemical constituents of peritoneal fluid in cows. Vet Rec 2010; 166: 15-19. https://doi.org/10.1136/vr.b5584

[52] Ziemer EL. Cytological analysis of large animal body fluids. Vet Med 1989; 6: 574-83.

[53] Smith DF, Munson L, Erb HN. Abomasal ulcer disease in dairy cattle. Cornell Vet 1983; 73: 213-214.

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