

Clinical Studies on Field Cases of Traumatic Pericarditis

S.A. Galbat

Department of Animal Medicine, Faculty of Veterinary Medicine,
New Valley University, New Valley, Egypt

Abstract: Pericarditis is an inflammation of the pericardium that results in accumulation of pericardial fluid within the pericardial sac in cattle. It is mostly caused by the perforation of the pericardial sac by sharp metallic foreign body through the reticulum. In a veterinary clinic in Menoufia, many cases were followed up with symptoms that interfere with pericarditis due to machinery preparing for animal food, cattle swallow food without examining it with the upper lip, which facilitates infection with nails, wires and needles and as a consequence lead to pericarditis. This study included suspected cross bred cattle's (N=42) that were examined due to digestive disturbance, all cases were examined with metal detector. Out of them 23 give negative results while the other 12 case give positive with clinical symptom of tachycardia, muffled heart sounds, absence of lung sound in the ventral thorax, abnormal heart sounds, distension of the jugular veins and pulsation and submandibular, brisket and ventral abdominal edema are usually present. Abnormal demeanour and general condition, decreased rumen motility, poorly digested faeces, fever. From the positive cases, eight were pregnant. Seven cases were positive with the metal detector without typical clinical symptoms but suffered from recurrent tympany and decreased rumen movement. Blood samples were examined and the results confirmed the diagnoses, hematological findings included decreased hematocrit and leukocytosis with shift to left, neutrophilia and hyperfibrinogenaemia (indicating inflammation) was associated with increased globulin and total protein. Traumatic pericarditis was confirmed postmortem in two cattle with typical pericarditis. Pericarditis can be avoided by the proper managemental practices like feeding of magnets, avoiding potential and non-potential foreign bodies in the feed and fodder of the animals, Treatment of pericarditis in cattle usually is addressed toward short term survival to calving. Treatment would be attempted only in a valuable animal or in an animal carrying a high value embryo. Diuretics for eliminating peripheral edema, systemic antibiotics and drainage of the pericardial sac, then supportive treatment, Rumenotomy is effective for the diagnoses and the treatment if possible. The sever and complicated cases must be sent to slaughter house.

Key words: Traumatic Pericarditis • Probable Causes • Valley Governorate

INTRODUCTION

Among the numerous diseases of foreign body syndrome in ruminant species, traumatic reticuloperitonitis (TRP) and traumatic pericarditis (TP) are the most common. TRP is a sporadic disease in ruminants caused by perforation of the reticulum due to ingestion of foreign perforating sharp metals, which is a common cause of abdominal surgery in cattle. Cattle are more susceptible to foreign body syndrome than small ruminants because they do not use their lips for the prehension of their food and are more likely to eat

chopped feed [1]. Atypical foreign body is metallic object such as apiece of wire or nail [2]. Occasionally the foreign body may pierce and infect the liver and spleen [3, 4].

Foreign bodies are responsible for about 70% of abdominal affections in cattle and buffaloes in Egypt [5]. Sharp foreign bodies are incriminated as the main cause of traumatic pericarditis (TP) and diaphragmatic hernia (DH) in cattle and buffalo [5]. This condition produces devastating economic losses due to severe reduction in milk and meat production, treatment costs, potential fatalities and fetal losses in affected pregnant animals [6].

Pericarditis is acute, subacute or chronic inflammation of the pericardium due to penetration of pericardium by sharp foreign body. Since distance from the reticulum to the pericardium is only a few centimeters, sharp contaminated foreign body can easily pierce the diaphragm and enter the pericardium. Rarely, traumatic pericarditis may be caused by the penetration of the wire through the skin, with subsequent migration into the sternbrae and pericardial sac [7]. Pericarditis attributable to haematogenous spread of infectious diseases (such as colibacillosis, pasteurellosis, salmonellosis and anaerobic infections) is much less common and is usually masked by signs of septicemia [8].

In cattle, the detected foreign bodies were clothes, plastic, nails, ropes, hair, leather, wires and nails [9]. Acute disease usually results in distinct signs that include anorexia, decreased milk production, fever, ruminal atony and tympany, abdominal pain, arched back, abdominal guarding and tense abdomen [10].

The Pericarditis is clinically characterized by severe depression, reluctance to move, abduction of fore limbs, progressive weakness, sharp drop in milk yield, variable appetite to complete in appetency, mild fever, ruminal stasis, recurrent tympany, scanty hard feces, abdominal pain and death [11]. Without an accurate case history and when the victim is admitted after longer intervals of ingestion of a metal object, the diagnosis becomes more difficult [12].

Animals with large amount of blunt foreign bodies show anorexia, depression, intermittent respiratory distress, recurrent rumen tympany, rumen stasis, dehydration, reduced milk yield, distended left para lumbar fossa and sometimes vomiting [13, 14]. The indiscriminating feeding habit of cattle and buffaloes predisposes them to ingestion of various foreign bodies and these ingested foreign bodies settle in the reticulum as the honeycomb-like structure of the reticulum provides sites for fixation [4].

Traumatic reticulo-peritonitis is most commonly results from perforation of the reticulum by metal objects such as nails or wire that have been accidentally incorporated into feed and ingested [15]. Traumatic reticulo-peritonitis and traumatic pericarditis always caused by trauma. Long, thin, sharp foreign bodies, such as wires or nails, perforate the reticulum and diaphragm and enter the pericardial sac, causing traumatic pericarditis. Pericarditis of haematogenous origin occurs less frequently [8]. It may be a concomitant finding in cattle with colibacillosis, pasteurellosis, salmonellosis, or anaerobic infections, but the signs are usually masked by

signs of septicaemia [8]. In contrast, with human beings, dogs and horses, idiopathic pericarditis is rare in cattle [16]. This may lead to localized peritonitis, sometimes involving neighboring organs and in severe cases it results in generalized peritonitis. Several authors described the clinical signs of TRP [15, 17].

The earliest clinical sign of TP is tachycardia. However, this sign is not specific as it may present in many physiological and other pathological conditions. Hence, the early diagnosis of such condition is difficult as the most cases present in advanced stages when the cardiac dysfunction produce clinical signs of heart failure which had a poor prognosis [18]. The main clinical signs are tachycardia, muffled heart sounds, asynchronous heart sounds such as rubbing or splashing sounds, jugular vein distension and edema; the presence of all these signs is pathognomonic for traumatic pericarditis. However, many cases do not show all of these signs and diagnosis may therefore not be straightforward. For example, the differential diagnoses in cattle with jugular vein distension and tachycardia include right-sided cardiac insufficiency attributable to valvular endocarditis, cardiomyopathy, cardiac leucosis and other diseases [8]. A study on cattle that included 60 traumatic reticulitis cases reported that TP was the most common sequela, occurring in 40 cases [19], this means that all cattle affected with TP also have TRP, as pericarditis is a major complication of reticulo-peritonitis. There may be additional signs in cattle with sequelae such as traumatic pericarditis [20], liver abscesses [21] or cranial functional stenosis [10, 15]. Cattle with acute localized peritonitis typically have neutrophilia with a regenerative left shift [10]. The clinical signs of traumatic pericarditis have been described in detail in textbooks [8, 22]. These complications depend mainly upon the nature, length and direction of penetration of the swallowed foreign body [23].

There are numerous scientific papers describing the clinical signs of TRP in cattle. Anorexia, decrease in milk production, fever, tachypnea, reluctance to move and stance with an arched back and abducted elbows are the most common signs [24]. Diagnosis of advanced cases based on clinical examination as pathognomonic clinical signs “including bilateral jugular venous distention and/or pulsation and edema at dewlap” are characteristic for the diseased condition [4].

Several complications result from ingestion of indigestible foreign materials such as chellitis, gingivitis, glossitis, stomatitis, pharyngitis, tonsillitis, chock, esophagitis, rumenitis, ruminal impaction, acute or

recurrent rumen tympany, localized or diffuse reticulo-peritonitis, reticular adhesion and diaphragmatic hernia. Other consequences include pericarditis, reticular fistulation, reticular, diaphragmatic, mediastinal, hepatic, splenic, lateral and ventral abdominal wall abscesses, vagal indigestion, rupture of left gastro-epiploic artery, traumatic pneumonia and pleurisy [14, 25, 26].

The most important biochemical findings are increased concentrations of total protein and fibrinogen. Tests for reticular foreign bodies may elicit a grunt, although other painful disorders of the thorax and abdomen may stimulate the same reaction [15, 21].

Laboratory tests may be helpful in diagnosis of foreign body syndrome. Hemogram of the diseased animals shows anemia, increased packed cell volume and neutrophilia with a left shift [27]. Serum biochemical parameters of diseased animals show increased total protein, globulin, total bilirubin, ALT, ALP, P and decreased albumin/globulin ratio and Ca [28]. Administration of reticular magnets at the age of 1.5-2 years has become a popular preventive measure for hardware disease [29].

The goals of the present study were to describe the clinical and laboratory findings in cattle with TRP, the precautions include removal of ferrous and other potentially hazardous objects from field and lane edges, good nourishment and management of the animals, passing of processed foods over magnets to remove metallic objects, avoiding the use of baling wire, completely removing old buildings and fences, keeping animals away from sites of new construction, avoiding the pollution of grazing lands with plastic bags, hair, hoof, wool and avoiding the unsupervised grazing of animals [6, 27].

Treatment of pericarditis in cattle is often unrewarding and usually is addressed toward salvage or short term survival to calving. Treatment should be attempted only in a valuable animal or in an animal carrying a high value embryo. Diuretics are effective in eliminating the severity of peripheral edema; they further reduce venous return and preload in animals with pericarditis [30]. Medical therapy with systemic antibiotics and drainage of the pericardial sac rarely, if ever, permanently cures affected cattle. Therefore most therapeutic efforts have been surgical [31].

MATERIALS AND METHODS

Animals, History, Clinical and Postmortem Examinations: Between January 1, 2016 and January 31, 2018, 42 cross bred cattle's were examined due to

digestive disturbance. All animals included in this study were females, all cases were clinically and physically examined, by auscultation of the heart, lungs, rumen and intestine, percussion auscultation of both sides of the abdomen and a rectal examination. Testing for foreign bodies in the reticulum consisted of pinching the withers, upward pressure on the xiphoid area and percussion of the reticulum.

- All cases were examined with metal detector.
- Two animals with critical case sent to abattoir with typical pericarditis.

Laboratory Examination: Two blood samples were collected from each animal through the jugular vein puncture via vacutainer, one on EDTA containing tube and the other in plane.

A complete blood count (hemoglobin, hematocrit, total and differential leucocyte) was carried out on the first blood sample. After centrifugation of the second blood sample, serum samples were collected and then frozen for later analysis. In the serum, commercial kits were used to determine the concentrations of total protein, albumin, glucose, calcium, creatinine, urea nitrogen, potassium, sodium and chloride. Serum protein fractions were determined by electrophoresis. Reference normal values were obtained from [4, 32].

RESULTS

Forty-two cross bred cattle's were examined due to digestive disturbance all cases examined with metal detector, 23 give negative, 12 case give positive with clinical symptom the other seven cattle give positive without typical clinical symptoms.

Clinical symptom of tachycardia, muffled heart sounds, absence of lung sound in the ventral thorax, abnormal heart sounds, Distension of the jugular veins and pulsation and submandibular, brisket and ventral abdominal edema are usually present. Abnormal demeanour and general condition, decreased rumen motility, poorly digested faeces, fever, they were eight pregnant and four non pregnant, while the other 7 cattle give positive without typical clinical symptoms but suffering from recurrent tympany and decrease rumen movement. Blood samples were examined and the results confirm the diagnoses, hematological findings were decreased hematocrit in of cattle and leukocytosis with shift to left and hyper-fibrinogenaemia (indicating inflammation) and globulin and total protein. With neutrophilia, Traumatic pericarditis was confirmed postmortem in 2 cattle, with typical pericarditis.

Table 1: Suspected cases showed one or more symptoms of ingestion of foreign body

Total animals	42		
-ve metal detector	23		
+ve metal detector	+ve (metal detector and clinical symptom)	12	8 pregnant 4 Non pregnant
	+ve (metal detector without clinical symptom)	7	

Table 2: Physically and clinically examination, by auscultation of the heart, lungs and rumen

Symptoms	+ve metal detector			
	Negative metal detector	Positive (metal detector without clinical symptom)	Positive metal detector clinical and symptom)	
			Non pregnant	Pregnant
1. Tachycardia,	-ve	+ve	+ve	+ve
2. Muffled heart sounds	-ve	-ve	+ve	+ve
3. Absence of lung sound	-ve	-ve	+ve	+ve
4. Distension of the jugular veins and pulsation	-ve	-ve	+ve	+ve
5. Decreased rumen motility	-ve	+ve	+ve	+ve
6. Fever	-ve	-ve	+ve	+ve
7. Poorly digested faeces	-ve	-ve	+ve	+ve

Table 3: Physically and clinically examination, by auscultation of the heart, lungs and rumen

C	+ve metal detector			
	-ve metal detector	+ve (metal detector without clinical symptom)	+ve (metal detector clinical and symptom)	
			Non pregnant	Pregnant
8. Pulse rate /minute	71.5 ± 3.5a	89 ± 6.3b	93.5 ± 5.8b	93.5 ± 5.8b
9. Pain tests	-veno painful reaction	-veno painful reaction	+ve painful grunting	+ve painful grunting
10. Resp. rate /minute	36.2 ± 2.5a	48.5 ± 4.5b	51.0 ± 3.5b	51.0 ± 3.5b
11. Distension of the jugular veins and pulsation	-ve	-ve	+ve	+ve
12. Ruminal movement	3/2 min	2/2 min	1/2 min	1/2 min
13. Rectal temperature	38.5 ± 0.4a	40.3 ± 0.5b	39.5 ± 0.7b	39.5 ± 0.7b
14. Characters of feces	Semi-solid dark green	Poorly digested and scanty	Firm scanty feces	Firm scanty feces

Table 4: Hematological and biochemical findings

	Parameters		Finding	
	+ve (metal detector without clinical symptom)	+ve (metal detector clinical and symptom)	-ve metal detector	Reference value
1. Hematocrit (%)	32±3	29±5	35±1	24-46
2. Hemoglobin (g/dL)	11±4	10±2	12±2	8.0-15.0
3. Leukocyte count (/iL)	5500±11	32500±1153***	6200±23	4000-12000
4. Neutrophils (/iL)	3017±22	12017±3215***	3017±41	600-4000
5. Lymphocytes (/iL)	5261±34	7232±3460	5700±12	2500-7500
6. Total protein (g/dL)	7.2±0.2	8.2±0.3*	7.1±0.5	6.7-7.5
7. Albumin (g/dL)	2.3±0.1	2.2±0.3	2.7±0.4	2.1-3.6
8. globulin (g/dL)	.80±0.5	1.3±0.3	.83±0.2	0.75-0.88
9. Glucose (mg/dL)	52±7	55±6	56±3	45-75
10. Calcium (mg/dL)	10±3	9±1	11±4	9.7-12.4
11. Sodium (mmol/L)	141±1	132±5	142±3	132-152
12. Potassium (mmol/L)	4±0.7	4±0.6	4±0.5	3.9-5.8
13. Chloride (mmol/L)	101±5	97±2	102±2	95-110
14. Urea nitrogen (mg/dL)	21±07	25±14	18±06	6.0-26
15. Creatinine (mg/dL)	1.6±0.2	1.5±0.3	1.3±0.2	1.0-2.0

DISCUSSION

Metal detector of the thorax and abdomen and blood analysis generally permits the clinician to make a precise diagnosis of the Pericarditis affecting the animal. Early diagnosis by Metal detector will, therefore, ensure unnecessary treatment or animal suffering. In the majority of cases, the prognosis is poor so good communication between the owner and the veterinarian is of primary importance when suspecting and diagnosing bovine Pericarditis [4]. In the present study clinical cases of cows with positive metal detector with clinical symptom were diagnosed and compared to healthy negative cows. Differentiation between +ve metal detector with clinical symptom was based on clinical, hematological and biochemical changes, the diagnosis of positive metal detector with clinical symptom was later confirmed by blood and biochemical analyses and postmortem examination for some cases.

Pericarditis is the most common pericardial disease of cattle. It is an inflammation of the pericardium that results in accumulation of pericardial fluid within the pericardial sac, in cattle it is mostly caused by the perforation of the pericardial sac by an infected foreign body through the reticulum.

In the veterinary clinic in Menoufia, many cases were followed up with symptoms that interfere with pericarditis due to machinery preparing for animal food, cattle swallow food without examining it with the upper lip, which facilitates infection with foreign bodies as nails, wires and needles lead to pericarditis. This study included more than 42 suspected cases showed one or more symptoms of ingestion of foreign body, 42 cross bred cattle's were examined due to digestive disturbance all cases examined with metal detector, 23 give negative, 12 case give positive with clinical symptom of tachycardia, muffled heart sounds, absence of lung sound in the ventral thorax, abnormal heart sounds, Distension of the jugular veins and pulsation and submandibular, brisket and ventral abdominal edema are usually present. Abnormal demeanour and general condition, decreased rumen motility, poorly digested faeces, fever, they was 8 pregnant and 4 non pregnant The clinical signs we observed in the cows with pericarditis were similar to those previously reported [4, 19, 33]. while the other 7 cattle give positive without typical clinical symptoms but suffering from recurrent tympany and decrease rumen movement the foreign body may blunt as metal key or coin or short nails need only the disease can be prevented

to a large extent by the proper managerial practices like feeding of magnets, preventing access to potential and non-potential foreign bodies.

The observed clinical signs were in the cows with positive metal detector with clinical symptom were similar to those previously reported [4, 19, 33]. In cases in which the foreign body penetrated the diaphragm and reached the pericardium the cows had additional signs, including muffled heart sounds, jugular distension and pulsation and edema of the brisket and submandibular regions. These clinical signs may have been due to cardiac insufficiency resulting from septic pericarditis [34].

Based on physical examination of the cows (Tables 2, 3), significantly higher rectal temperature was observed in the cows with +ve metal detector with clinical symptom than in the Reference values, indicating a systemic reaction. The observed significant increase in the respiratory rate indicates respiratory distress associated with toxemia and septicemia caused by the foreign body penetration. These results are in agreement with those previously reported.

Ruminal movements were markedly depressed (1 movement/2 min) compared to ruminal movements in the clinically healthy cows with reference values and negative metal detector group (3 movements/2 min), indicating significant hypomotility of the rumen in the cows with positive metal detector with clinical symptom. These results are similar to those previously reported [4].

The hematological changes (Table 4) observed in the cows with positive metal detector with clinical symptom are comparable to those previously noted [35, 36]. The observed reduction in hemoglobin indicates anemia, which could be attributed to the loss of blood during penetration of the reticulum or the chronic inflammatory process [36]. Nonetheless, the observed increase in hematocrit values could be attributed to dehydration associated with fluid loss due to the reduction of food and water intake in the animals with positive metal detector with clinical symptom. Significant leukocytosis with neutrophilia was noted in the cows with TRP and TP, which has been previously, reported [37, 38]. Leukocytosis and neutrophilia were indicative of inflammatory responses that might have been due to infection associated with the penetration of the reticulum and diaphragm. On the other hand, there was significant lymphopenia, which might have been due to a reduction in cellular immunity associated with the stress of penetration [4]. In the +ve metal detector with clinical symptom cows there was significant leukocytosis,

neutrophilia and monocytosis, as compared to the reference value and +ve without clinical symptom group, indicating that the positive metal detector with clinical symptom cows had many of the characteristics of a large internal abscess, which induces a more severe response [4]. In contrast, there was lymphocytopenia and eosinopenia, which are consistent with previous reports [4]. Serum electrolyte analysis (Table 4) showed significant reductions in the levels of sodium, potassium and chloride in the cows with TRP and TP, which was attributable to ruminal hypo-motility and/or vagal indigestion. Hypokalemia might be attributed to anorexia, but might also be exacerbated by ion exchange caused by alkalosis and/or abomasal reflux into the rumen. In the present study the Ca concentration was significantly lower in the +ve metal detector with clinical symptom cows than in the controls, whereas the phosphorus concentration did not differ significantly. These results are consistent with previous reports. The concentrations of these elements have been reported to vary in cattle with TRP [39]. Hypocalcemia can occur due to reduced calcium uptake as a result of illnesses that affect the appetite and decrease its absorption [40]. Therefore, hypocalcemia observed in the present study probably developed in association with gastrointestinal stasis and insufficient dietary uptake, as previously reported [41]. Biochemical examination of serum (Table 4) showed a significantly higher ($P < 0.05$) fibrinogen level in the +ve metal detector with clinical symptom groups, as compared to the -ve metal detector group, which might have been due to its enhanced hepatic synthesis as the result of a severe inflammatory process following foreign body penetration [40]. The level of total protein was not significantly different between the groups, although it was slightly higher in the positive metal detector with clinical symptom group may be due to dehydration.

Positive (metal detector without clinical symptom group and negative metal detector group results are near to the reference values without significant changes in parameters due to absence of foreign body infection or blunt metals or coin without harmful effect.

CONCLUSIONS

Foreign body syndrome of cattle is a critical problems facing veterinarians all over the world particularly in developing countries. Although Pericarditis and reticulo-peritonitis have a specific clinical signs, they also have several hematological and biochemical

characteristics. Moreover, metal detector examination can help early prophylaction by Administration of reticular magnets at the age of 1.5-2 years is a preventive measure for the disease.

REFERENCES

1. Braun, U., 2003. Ultrasonography in gastrointestinal disease in cattle. *Vet. J.*, 166: 112-124.
2. Rebhun, W.C., 1995. *Disease of Dairy cattle*. 1st Edu. Philadelphia: Williams and Wilkins, pp: 113-116.
3. Orpin, P. and D. Harwood, 2008. Clinical management of traumatic reticuloperitonitis in cattle in *Practice*, 30: 544-551.
4. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and P.D. Constable, 2007. Traumatic reticuloperitonitis In: Radostits, O.M., Gay, C.C., Hinchcliff, K.W., Constable, P.D. Eds., *Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats*. 10th edn., Elsevier Health Sciences, Philadelphia, PA, USA, pp: 337-352.
5. Misk, N.A., 2012. The final report of research project "Studies on early diagnosis of different abdominal disorders with special reference to the surgical interference for treatment in cattle and buffaloes" Assiut, Egypt, Assiut University.
6. Nugusu, S., V. Ramaswamy, C. Unakal and R. Nagappan, 2013. Studies on foreign body ingestion and their related complications in ruminants associated with inappropriate solid waste disposal in Gondar Town, North West Ethiopia. *Int. J. Anim. Vet. Adv.*, 5: 67-74.
7. Sojka, J.E., M.R. White, W.R. Widmer and W.G. Van Alstine, 1990. An unusual case of traumatic pericarditis in a cow. *J. Vet. Diagn. Invest.*, 2: 139-142.
8. Gründer, H.D., 2002. Krankheiten des Herzens und des Herzbeutels. In *In ne re Me di zin und Chirurgie des Rindes*. 4th edn. Eds G. Dirksen, H.D. Grün der, M. Stöber. Berlin, Parey Buchverlag, pp: 159-181.
9. Tesfaye, D. and M. Chanie, 2012. Study on rumen and reticulum foreign bodies in cattle slaughtered at Jimma Municipal abattoir, South West Ethiopia. *Am-Euras J. Sci. Res.*, 7: 160-167.
10. Constable, P.D., K.W. Hinchcliff, S.H. Done and W. Grünberg, 2017. Diseases of the alimentary tract – ruminant. In: *Veterinary medicine. A textbook of the diseases of cattle, horses, sheep, pigs and goats*. St. Louis: Elsevier, pp: 436-621.

11. Singh, R., S.L. Garg, N. Sangwan and S. Gerai, 2008. Peripheral concentration of thyroid hormones in buffaloes suffering from foreign body syndrome. *Haryana Vet.*, 47: 115-116.
12. Ramin, A.G., A.M. Hashemi, S. Asri-rezaie, E. Bateb and A. Tamadon, 2011. Predication of traumatic pericarditis in cows using some serum biochemical and enzyme parameters. *Acta Vet. (Beograd)*, 61: 383-390.
13. Reddy, M.V. and P. Sasikala, 2012. A review on foreign bodies with special reference to plastic pollution threat to livestock and environment in Tirupati rural areas. *Int. J. Scient Res. Pub.*, 2: 1-8.
14. Abu-Seida, A.M. and O.S. Al-Abbadi, 2014. Recurrent rumen tympany caused by trichobezoars in buffaloes (*Bubalus bubalis*): A series report. *Thai J. Vet. Med.*, 44: 147-151.
15. Dirksen, G., 2002. Krankheiten von Haube und Pansenbeimruminanten Rind. In: Dirksen G, Gründer HD, Stöber M, editors. *InnereMedizin und Chirurgie des Rindes*. Berlin: Parey Buchverlag, pp: 396-455.
16. Jesty, S.A., R.W. Sweeney, B.A. Dolente and V.B. Reef, 2005. Idiopathic pericarditis and cardiac tamponade in two cows. *Journal of the American Veterinary Medical Association*, 226: 1555-1558.
17. Francoz, D. and C.L. Guard, 2015. Traumatic reticuloperitonitis (hardware disease, traumatic reticulitis). In: Smith BP, editor. *Large Animal Internal Medicine*. St. Louis: Elsevier Mosby, pp: 805-7.
18. Reef, V.B. and S.M. McGuirk, 2002. Diseases of the cardiovascular system. In: Smith, B.P., editor. *Large Animal Internal Medicine*. 3rd ed. Mosby, St. Louis, MO, USA, pp: 95-7, 463-6.
19. Roth, L. and J.M. King, 1991. Traumatic reticulitis in cattle: a review of 60 fatal cases. *J. Vet. Diagn Invest*, 3: 52-54.
20. Braun, U., 2009. Traumatic pericarditis in cattle: clinical, radiographic and ultrasonographic findings. *Vet. J.*, 182: 176-86.
21. Braun, U., N. Pusterla and K. Wild, 1995. Ultrasonographic findings in 11 cows with a hepatic abscess. *Vet. Rec.*, 137: 284-90.
22. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and Constable, 2007. *Veterinary Medicine. A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats*. 10th ed. Philadelphia: Saunders Elsevier, pp: 337-344.
23. Abouelnasr, K.S., E. Mosbah, G.I. Karrouf and A.E. Zaghoul, 2012. Comparative ultrasonographic findings of traumatic reticulitis, perireticular abscess and diaphragmatic hernia in buffalo (*Bubalus bubalis*). *J. Amer. Sci.*, 12: 590-595.
24. Fubini, S. and T.J. Divers, 2007. Noninfectious diseases of the gastrointestinal tract. In: Divers T., Peek S. (eds.): *Rebhun's Diseases of Dairy Cattle*. 2nd ed. Saunders Elsevier, St Louis, pp: 130-199.
25. Roth, L. and J.M. King, 1991. Traumatic reticulitis in cattle: a review of 60 fatal cases. *J. Vet. Diagn. Invest.*, 3: 52-54.
26. Floeck, M. and W. Baumgartner, 2001. Ultrasonographic diagnosis of traumatic reticuloperitonitis and pericarditis in cattle. *Wiener Tierärztliche Monatsschrift*, 12: 347-354 (in German).
27. Reddy, Y.R., P.A. Latha and S. Reddy, 2014. Review on metallic and non-metallic foreign bodies: a threat to livestock and environment. *Int. J. Food Agri. Vet. Sci.*, 4: 6-14.
28. Ghanem, M.M., 2010. A comparative study on traumatic reticuloperitonitis and traumatic pericarditis in Egyptian cattle. *Turk J. Vet. Anim. Sci.*, 34: 143-153.
29. Weaver, D., A. Steiner and S.J. Guy, 2005. *Bovine Surgery and Lameness*, 2nd Ed., Blackwell Publishing, Ames, Iowa, USA, pp: 75-139.
30. Reef, V.B. and S.M. McGuirk, 2009. Diseases of the cardiovascular system. In: Smith BP, Ed. *Large Animal Internal Medicine*. 4th ed. St. Louis, Missouri: Mosby-Elsevier, pp: 453-489.
31. Peek, S.F., 2008. Cardiovascular Diseases. In: Divers TJ and Peek SF. (Ed). *Rebhun's Diseases of Dairy Cattle*. Saunders Elsevier, St. Louis, Missouri, pp: 43-78.
32. Kaneko, J.J., J.W. Harvey and M.L. Bruss, 1997. *Clinical Biochemistry of Domestic Animals*, 5th edn, New York, Academic Press.
33. Braun, U., M. Gotz and O. Marmier, 1993. Ultrasonographic findings in cows with traumatic reticuloperitonitis. *Vet. Rec.*, 133: 416-422.
34. Sojka, J.E., M.R. White, W.R. Widmer and W.G. Van Alstine, 1990. An Unusual Case of Traumatic Pericarditis in a Cow. *Journal of Veterinary Diagnostic Investigation*, 2: 39-42.
35. Nassif, M.N. and S.A. El-Khodery, 2001. Ultrasonographic, clinical and clinicopathological findings in cows with traumatic pericarditis. *Beni-Suef Vet. Med. J.*, 11: 111-124.

36. Ocal, N., G. Gokce, A.I. Gucu, E. Uzlu, B.B. Yagci and K. Ural, 2008. Pica as a predisposing factor for traumatic reticuloperitonitis in dairy cattle: serum mineral concentrations and hematological findings. *J. Anim. Vet. Adv.*, 7: 651-656.
37. Rosenberger, G., 1979. *Clinical Examination of Cattle*. 1st edn., Verlag Paul Parey, Berlin and Hamburg.
38. Latimer, K.S., E.A. Mahaffey and K.W. Prasse, 2003. *Duncan and Prasse's Veterinary Laboratory Medicine: Clinical Pathology*, 4th edn., Ames, Iowa State Press, 68-77, 152-160.
39. Moore, F., 1997. Interpreting serum chemistry profiles in dairy cows. *Vet. Med.*, 92: 903-912.
40. Roussel, A.J., M.S. Whitney and D. Cole, 1997. Interpreting a bovine serum chemistry profile: Part 1. *Vet. Med.*, 92: 551-558.
41. Samad, A., K.B. Awaz and L.B. Sarkate, 1994. Diagnosis of bovine traumatic peritonitis. I: strength of clinical signs in predicting correct diagnosis. *J. Appl. Anim. Res.*, 6: 13-18.