

## Case Report

# Atypical Presentation of Constrictive Pericarditis in a Holstein Heifer

**Mohamed M. Elhanafy and Dennis D. French**

*Department of Veterinary Clinical Medicine, Rural Animal Health Management, 1008 W. Hazelwood Drive, 223 Large Animal Clinic, Urbana, IL 61802, USA*

Correspondence should be addressed to Mohamed M. Elhanafy, elhanafy@illinois.edu

Received 4 January 2012; Accepted 19 February 2012

Academic Editors: L. Arroyo and M. Bugno-Poniewierska

Copyright © 2012 M. M. Elhanafy and D. D. French. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

The field diagnosis of constrictive pericardial effusion is often established on the pertinent pathognomonic physical examination findings, but the condition cannot be ruled out based on absence of these cardinal signs. Constrictive pericardial effusion is not always manifested by bilateral jugular venous distention and pulsation, brisket edema, and muffled heart sounds, all of which are considered the key points in the field diagnosis of pericardial effusion and hardware disease. This case will also document that the outcomes of hematology, serum biochemistry panels, and blood gas analysis can be totally inconsistent with passive venous congestion and constrictive pericardial effusion in cattle. Chest radiographic findings revealed radio dense, wire-like objects; the findings were suggestive but not conclusive for pericardial or pleural effusions, due to indistinguishable diaphragmatic outline and cardiopulmonary silhouette. Cardiac ultrasonography was found to be an excellent paraclinical diagnostic procedure for cases that potentially have traumatic pericarditis and constrictive pericardial effusion. Ultrasound-guided pericardiocentesis was also a valuable diagnostic aid in establishing a definitive diagnosis.

## 1. Introduction

The fundamental task of a veterinarian is making a diagnosis and planning the most effective therapeutic approach. Veterinarians face three challenges on a daily basis: establishing a correct diagnosis, selecting appropriate clinical management, and keeping up to date with useful technological advances [1]. In some instances, routine physical examination fails to detect the clinically significant abnormalities of the body system(s) involved even when conducted by highly skilled practitioners. Additionally, hematology and serum biochemistry are not always effective in determining the disease process, and this lack of specificity renders the diagnosis tentative rather than conclusive.

Diagnostic imaging procedures such as thoracic radiographs or cardiac ultrasonography are not frequently applied in large animal practice due to lack of equipment, time, or economic constraints. These factors limit diagnostic capabilities and may lead to incorrect diagnosis, unnecessary treatments, and the potential for a negative impact on the

veterinary-client-patient relationship and unneeded animal suffering.

In large animal patients, this scenario could easily happen with diseases such as constrictive pericarditis or cardiac tamponade. Pericarditis is the most commonly encountered consequence of traumatic reticuloperitonitis in cattle [2] and obtaining a definitive diagnosis can be challenging in certain instances.

It may be assumed that the diagnosis of such cases is often straightforward based on the pertinent cardinal signs and the pathognomonic physical examination findings described in text books such as bilateral jugular venous distention/pulsation, brisket edema, pulmonary edema, and muffled heart sounds [2]. However, variability in history, presenting complaints, physical examination findings, and clinical pathology data often makes the diagnosis somewhat elusive.

This paper describes a challenging, uncommon presentation of constrictive pericarditis in a 9-month-old Holstein heifer and highlights the limitations of routine physical

exam, hematology, and serum chemistry in the diagnosis of the disease.

## 2. Case Presentation

A 9-month-old Holstein heifer was presented to the Veterinary Teaching Hospital, University of Illinois with a three-week history of decreased appetite, decreased fecal output, and intermittent fever. The heifer had been evaluated previously and treated for enzootic pneumonia with oxytetracycline followed by ceftiofur and finally enrofloxacin.

Upon presentation, the heifer was bright, alert, and responsive and had normal ambulation. Rectal temperature was 40.3°C [104.5°F] (reference range 37.8–39.2°C; 100–102.5°F). The respiratory rate was 36 breaths/minute (reference range 24–36 breaths/minute) with an abdominal respiratory pattern. Chest auscultation revealed audible moist rales over the middle third of lung fields bilaterally and high-pitched, hissing rhonchi dorsally. It was noted that there was a slight abduction of both elbows. The heart rate was 72 beats/minute (reference range 55–80 beats/minute) with normal rhythm. Cardiac sounds were barely audible and were overwhelmed by the breathing sounds. The withers pinch test appeared to be normal and no jugular distention or pulsation was detected. Furthermore, the brisket was of normal size and consistency. She appeared slightly dehydrated based on her eye recession. The rumen was completely atonic, the intestines were hypomotile, and the rectum was full of firm dry feces upon rectal exam.

Complete blood count revealed leukocytosis ( $15.3 \times 10^3/\mu\text{L}$ ; reference range  $4.0\text{--}12.0 \times 10^3/\mu\text{L}$ ) with neutrophilia ( $12.3 \times 10^3/\mu\text{L}$ ; reference range  $0.6\text{--}4.0 \times 10^3/\mu\text{L}$ ) and monocytosis (963/ $\mu\text{L}$ ; reference range 25–800/ $\mu\text{L}$ ).

Serum chemistry profile showed decreased albumin (1.8 gm/dL; reference range 2.1–3.6 gm/dL) and increased globulin (5.0 gm/dL; reference range 3.6–4.5 gm/dL). Hepatic enzymes and renal values were within normal limits. There was moderate hypocalcemia (7.5 mg/dL; reference range 9.7–12.4 mg/dL), hypokalemia (3.6 mmol/L; reference range 3.9–5.8 mmol/L), hypochloremia (91 mmol/L; reference range 95–100 mmol/L), and hyponatremia (130 mmol/L; reference range 132–152 mmol/L).

Based on the arterial blood gas analysis the heifer was neither hypoxic nor hypercapnic, with moderate alkalosis (pH 7.54; reference range 7.35–7.50). The  $P_{\text{a}}\text{O}_2$  (103.0 mm Hg; reference range 85–105 mm Hg),  $P_{\text{a}}\text{CO}_2$  (29.9 mm Hg; reference range 34–45 mm Hg), oxygen saturation (98.0%; reference range 97–99% at sea level), and the blood *l*-lactate (0.9 mmol/L; reference range up to 2.9 mmol/L) were within normal limits.

To rule out pulmonary disease, thoracic radiographs were taken. Images revealed a round circumscribed mass measuring approximately  $2.4 \times 1.8$  cm with a fluid line and a large area of gas dorsally. This mass was superimposed over the cardiac region. Multiple small, round to irregular, gas opacities could be seen throughout the ventral aspect of the thorax. A diffuse interstitial to alveolar pattern was observed on the caudal lung lobes as well as one thin metallic

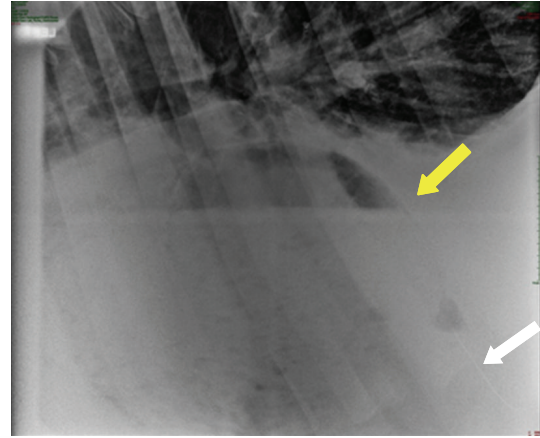


FIGURE 1: Laterolateral radiograph showing circumscribed mass with a fluid line and an area of gas shadows dorsally superimposing in the cardiac region (yellow arrow), thin opaque foreign material in the region of the caudal thorax (white arrow).

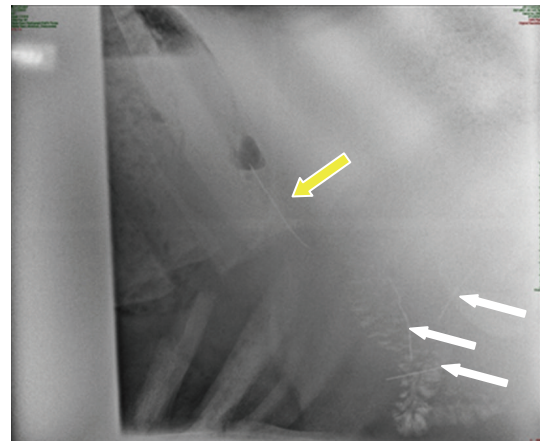


FIGURE 2: Laterolateral radiograph taken at the level of the reticulum and diaphragm. The intrathoracic foreign body and gas shadows could be seen cranial to the reticulum (yellow arrow). The lung and diaphragm could not be differentiated because of the increased soft tissue opacity. Three thin metallic opaque foreign bodies could be seen in the cranial epigastric abdomen in the region of the reticulum (white arrows).

opaque foreign object in the region of the caudal thorax (Figure 1). Three thin metallic opaque foreign bodies were also noted in the cranial abdomen in the region of the reticulum (Figure 2).

Ultrasound was used to better characterize the thoracic mass identified on radiographs. The ultrasonographic evaluation of the left hemithorax revealed moderate-to-large amounts of hypoechoic fluid within the ventral pleural space. Multiple hyperechoic, linear, undulating projections were imaged throughout the pleural cavity indicating chronic fibrinous pleurisy (Figure 3). The surface of pericardium was rugged, with multiple hyperechoic linear pericardial projections imaged throughout, indicative of chronic inflammatory changes with fibrin strand deposition (Figure 4). A large

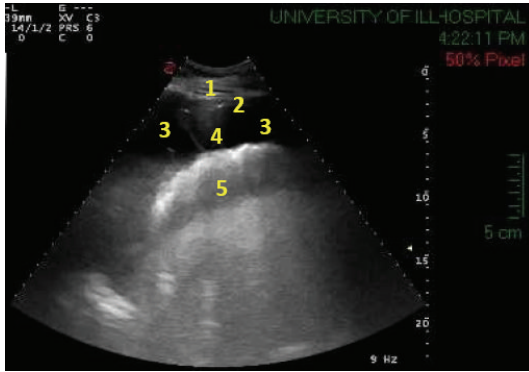


FIGURE 3: Ultrasonogram of the left thorax at the seventh intercostal space using a 3.5 MHz convex transducer showing pleural effusion. 1: thoracic wall; 2: costal pleura; 3: pleural effusion; 4: fibrin strands; 5: lung.

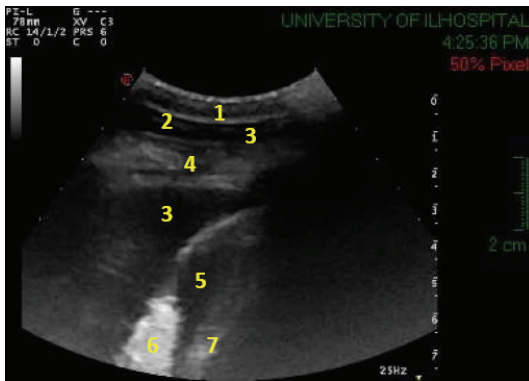


FIGURE 4: Ultrasonogram of pericardial effusion with strands of fibrin on the epicardium obtained by 3.5 MHz convex transducer placed in the fifth intercostal space on the left thorax. 1: thoracic wall; 2: parietal pleura; 3: pleural effusion; 4: lung; 5: hypoechoic pericardial effusion; 6: thickened pericardium with fibrin strands; 7: atrial wall.

hypoechoic area with irregular margins was also observed in the cranioventral thorax. This was thought to be a massive pericardial effusion that made the evaluation of the cardiac silhouette difficult (Figure 5). Free echogenic fibrin strands were occasionally detected floating within the epicardium.

Ultrasound-guided pericardiocentesis was aseptically performed using a 3-inch 18-gauge needle inserted into the lower third of the 4th intercostal space. It yielded a turbid, gray-colored fluid that contained 4.5 gm/dL (reference range < 2.5 gm/dL) total protein.

Based upon the protracted history of the case, radiographic and ultrasound images and the character of the fluid obtained via pericardiocentesis, a grave prognosis for use and life was made. The owner elected for humane euthanasia and the heifer was submitted for necropsy.

The gross pathology revealed approximately 8 liters of clear yellow fluid in the pleural cavity mixed with yellow gelatinous material (Figure 6). Portions of the ventral aspects of the left and right lungs were adhered to the diaphragm, pericardium, and ventral aspect of the thorax. The intercostal,

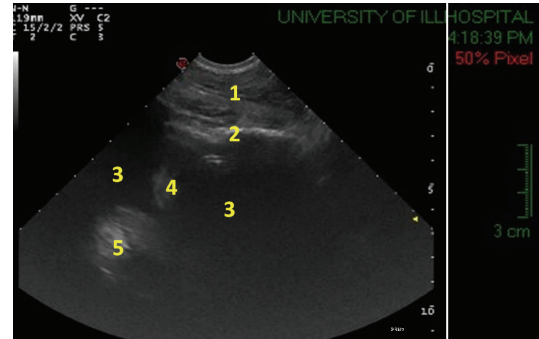


FIGURE 5: Ultrasonogram of pericardial effusion obtained by 3.5 MHz convex transducer placed in the distal fifth intercostal space on the left thorax. 1: thoracic wall; 2: thickened pericardium; 3: massive hypoechoic pericardial effusion; 4: fibrin strands; 5: ventricular wall.

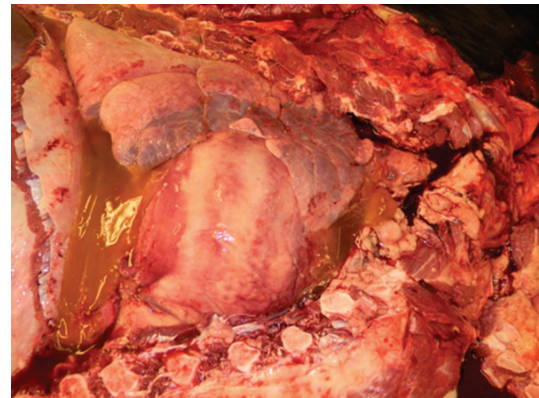


FIGURE 6: Postmortem image of the thoracic cavity showing severely distended pericardium displacing the lung dorsally and yellow gelatinous material within the pleural cavity.

sternal, cranial and caudal mediastinal, and tracheobronchial lymph nodes were diffusely enlarged. The pericardium was severely distended, displacing the lung dorsally (Figure 6). Several portions of the pericardium were tightly adhered to the adjacent diaphragm and ventral aspect of the thorax. The pericardial cavity contained approximately 7 liters of clear yellow malodorous fluid admixed with moderate amounts of tan to white friable material. The parietal pericardium was severely thickened measuring approximately 2.5 cm. The pericardial sac and was totally lined by a large mat of thick yellow fibrinous material (Figure 7). Several small to moderately sized abscesses were scattered throughout the abdominal wall, thoracic wall, liver, and reticular serosa. One of the abscesses located on the diaphragmatic surface of the left lobe of the liver contained a thin black 6 cm wire (Figure 8). A similar wire was found free in the reticulum.

### 3. Discussion

Constrictive pericarditis or cardiac tamponade is the abnormal accumulation of exudate in the pericardial sac. It is the most common sequelae of hardware disease in cattle caused



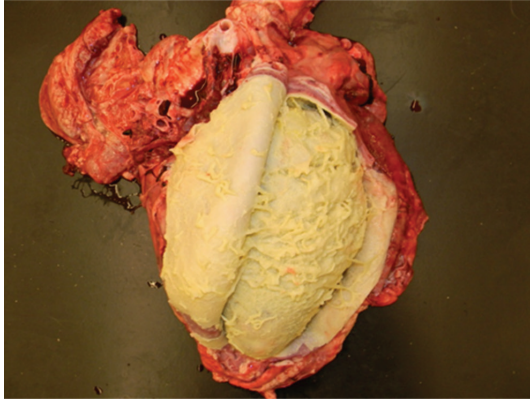


FIGURE 7: Postmortem image of the heart and pericardial sac, which has been opened. Note that the heart is covered with yellow fibrin.

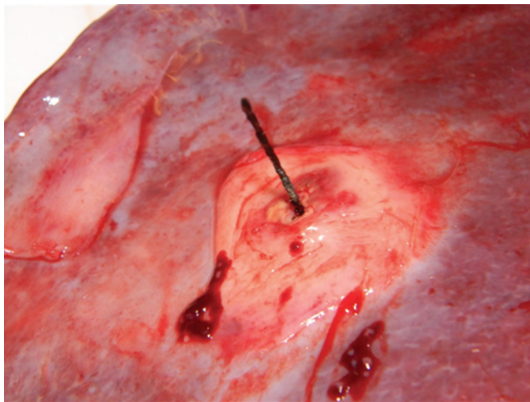


FIGURE 8: Postmortem image revealing a thin black 6 cm wire penetrating the diaphragmatic surface of the left lobe of the liver.

by traumatic perforation of the wall of the reticulum by a sharp foreign body [2]. The classic presentation of constrictive pericarditis has been described in standard text books [2] and some recent case reports [3–5] as muffled heart sounds, bilateral jugular venous distention/pulsation, along with brisket, and pulmonary edema. Brisket edema has been reported to be the most common symptom associated with pericarditis. In 27 out of 40 clinical cases of traumatic pericarditis in water buffaloes, edema of the brisket was recorded as a clinical sign and 37 of the 40 animals had constrictive pericardial effusion on postmortem examination [6]. However, none of these signs were observed in this case despite the presence of voluminous constrictive pericardial effusion recorded by ultrasonography and observed on postmortem examination. The absence of the clinical symptoms considered typical for the disease syndrome made arriving at the final diagnosis challenging.

Constrictive pericardial effusion is expected to lead to the development of generalized venous congestion and edema, as it impairs venous return to the heart. Auricular compression is usually inevitable in the course of the events [2, 4, 7]. Accordingly, some authors [4] related the degree of jugular congestion and brisket edema to the degree of pericardial

effusion. This case presentation illustrates that cardiac tamponade may not always be manifested by venous congestion and brisket edema: the heifer had neither of these signs as part of her physical examination findings. The lack of these signs may have been associated with compensation by cardiac reserve. These compensatory responses comprise the redistribution of the blood flow and increased heart rate [2]. An elevation of heart rate alone is a significant factor in increasing cardiac output and maintaining circulatory equilibrium. It has been documented that cattle can increase their heart rate to four times their resting values [2]. For the case presented here, the heart rate was still normal at presentation suggesting the heart was able to compensate for the pericardial restriction through mechanisms such as increased contractility. Given the severity of the pericarditis, it was surprising that the heart rate was normal. Accumulation of 7 liters of exudate in the pericardial sac would be expected to create sufficient intrapericardial pressure to cause collapse of auricles. However, the fibrinous adhesions between epicardium and the parietal pericardium at the level of the atria may have added extra support to the auricular wall, apparently preventing the collapse.

Muffled heart sounds usually indicate pericardial effusion [8] while heart sound audibility does not rule out pericardial effusion. It has been previously reported that 21 of 28 clinical cases of pericarditis in cows had audible heart sounds on both sides of the chest even though they had pericardial effusion [9]. Fifteen out of 40 reported clinical cases of traumatic pericarditis in water buffaloes had audible heart sounds [6].

In most literature,  $\gamma$ -glutamyltransferase ( $\gamma$ -GT), aspartate aminotransferase (AST) activities, and serum bilirubin concentration were found to be increased with hepatic congestion and right-sided heart failure [10]. Furthermore a strong correlation has been established between liver enzymes activities, especially  $\gamma$ -GT, in cattle and right-sided cardiac insufficiency which indicated that the increased  $\gamma$ -GT is usually a sign of liver congestion and not one of primary liver disease. It has also been reported that some cattle with right-sided cardiac insufficiency were often misdiagnosed with liver disease because of elevated liver enzyme activities, even though they may have had jugular vein distension on clinical examination [10].

For this case, the serum biochemistry liver enzymes were within normal limits. This indicates the animal had not progressed to the point of having significant hepatic venous congestion. The animal's cardiovascular system was still able to compensate for the significant pericardial effusion.

Arterial blood gas analysis was of limited diagnostic value in this case. It revealed the  $P_aO_2$ ,  $P_aCO_2$ , oxygen saturation and the blood *l*-lactate concentrations to be within normal values. Arterial blood gas is not useful for definitely diagnosing pericarditis but it has some value in being able to rule out a significant loss of functional lung capacity as might occur with diffuse bronchopneumonia, severe pleural effusion, or extensive pulmonary edema.

The anatomical and physiological features of the respiratory system of cattle predispose them to the development of hypoxia faster and more profoundly than other farm animal

species [2]. The bovine lung is relatively smaller than other farm animals in relation to the animal size. The ratio of tidal volume to lung volume is much lower in cattle when compared to other large animals such as horses. Cattle also have a small physiological gaseous exchange capacity [11].

Therefore, the 8 liters of pleural fluid effusion in addition to the dorsal displacement of the lung by pericardial distention with 7 liters of fluid would have been expected to build up serious compression atelectasis, ventilation failure, and anoxic anoxia. The respiratory rate on this heifer was assessed to be in the high normal range, but it was difficult to appreciate significant lung disease on physical examination and her  $P_{aO_2}$  levels were found to be within the normal range.

Radiographs are a helpful diagnostic tool for traumatic reticuloperitonitis with some limitations. The size of the animal makes it challenging to obtain a conclusive radiograph with clear thoracic details. The lack of available equipment in field situations and even in teaching hospitals precludes routine use. In addition, transportation of most bovine patients to a suitable radiology unit may require animal sedation which may further compromise the animal. Furthermore, positioning the animal for imaging requires a team effort and increases risks for handling personnel and potential for excessive radiation exposure.

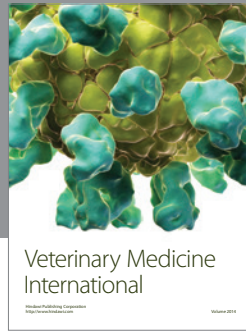
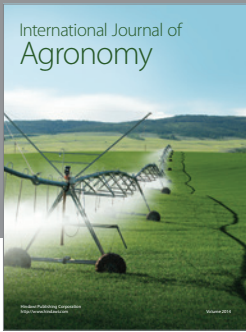
Definitive diagnosis of this condition by radiography can only be achieved when a foreign body is found perforating the cranial reticular wall and diaphragm (Figure 1), or is located close to the region of the reticulum (Figure 2). A published report [12] suggested that observation of traumatic perforation of the pericardium with radiographs is definitive in only one out of five clinical cases. Chest radiographic findings were suggestive, but not conclusive for pericardial or pleural effusions, due to indistinguishable diaphragmatic outline and cardiopulmonary silhouette.

Cardiac ultrasonography has been suggested as the method of choice for imaging and evaluating the severity of constrictive pericardial effusion [13–15]. Early diagnosis by ultrasonography will prevent unnecessary treatment or animal suffering. In most cases, a large amount of hypoechogenic fluid is seen in the thorax, sometimes containing strands or free clots of fibrin (Figures 4 and 5). In the case presented here, a poor prognosis was concluded based upon the severe disease identified via ultrasonography. The presence of large quantities of fibrin with massive pericardial effusion has been suggested as an important prognostic parameter in cases of pericarditis in human beings [16] and also in cattle [2]. Ultrasound-guided pericardiocentesis was an additional valuable tool that lead to a conclusive diagnosis.

## References

- [1] O. M. Radostits, I. G. Mayhew, and D. M. Houston, "Making diagnosis," in *Veterinary Clinical Examination and Diagnoses*, pp. 11–52, Elsevier Saunders, Philadelphia, Pa, USA, 1st edition, 2000.
- [2] O. M. Radostits, C. C. Gay, K. W. Hinchcliff, and P. D. Constantine, "Diseases of the pericardium," in *Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats*, pp. 430–432, Elsevier Saunders, Philadelphia, Pa, USA, 10th edition, 2007.
- [3] E. W. Fisher and H. M. Pirie, "Traumatic pericarditis in cattle: a clinical, physiological and pathological study," *British Veterinary Journal*, vol. 121, no. 12, pp. 552–567, 1965.
- [4] S. A. Jesty, R. W. Sweeney, B. A. Dolente, and V. B. Reef, "Idiopathic pericarditis and cardiac tamponade in two cows," *Journal of the American Veterinary Medical Association*, vol. 226, no. 9, pp. 1502–1558, 2005.
- [5] J. Laureyns, S. de Vlieghe, I. Kolkman, L. Vandaele, and A. de Kruijff, "Traumatic pericarditis with "steel band-effect" sounds in a young heifer," *Vlaams Diergeneeskundig Tijdschrift*, vol. 74, no. 2, pp. 146–148, 2005.
- [6] S. Fubini and T. J. Divers, "Noninfectious diseases of the gastrointestinal tract," in *Rebhun's Diseases of Dairy Cattle*, pp. 130–199, Elsevier Saunders, St. Louis, Mo, USA, 2nd edition, 2007.
- [7] T. Mohamed, "Clinicopathological and ultrasonographic findings in 40 water buffaloes (*Bubalus bubalis*) with traumatic pericarditis," *Veterinary Record*, vol. 167, no. 21, pp. 819–824, 2011.
- [8] L. Roth and J. M. King, "Traumatic reticulitis in cattle: a review of 60 fatal cases," *Journal of Veterinary Diagnostic Investigation*, vol. 3, no. 1, pp. 52–54, 1991.
- [9] U. Braun, B. Lejeune, G. Schweizer, M. Puorger, and F. Ehrensperger, "Clinical findings in 28 cattle with traumatic pericarditis," *Veterinary Record*, vol. 161, no. 16, pp. 558–563, 2007.
- [10] U. Braun, "Traumatic pericarditis in cattle: clinical, radiographic and ultrasonographic findings," *Veterinary Journal*, vol. 182, no. 2, pp. 176–186, 2009.
- [11] G. J. Gallivan, W. N. McDonell, and J. B. Forrest, "Comparative pulmonary mechanics in the horse and the cow," *Research in Veterinary Science*, vol. 46, no. 3, pp. 322–330, 1989.
- [12] S. Imran, S. P. Tyagi, A. Kumar, A. Kumar, and S. Sharma, "Ultrasonographic application in the diagnosis and prognosis of pericarditis in cows," *Veterinary Medicine International*, vol. 2011, Article ID 974785, 10 pages, 2011.
- [13] U. Braun, M. Flückiger, and F. Nägeli, "Radiography as an aid in the diagnosis of traumatic reticuloperitonitis in cattle," *Veterinary Record*, vol. 132, no. 5, pp. 103–109, 1993.
- [14] T. Moeller, "Clinical and ultrasonographic findings in a cow with pericarditis and ascites -a case report," *Praktische Tierarzt*, vol. 78, no. 5, pp. 403–405, 1997.
- [15] S. Buczinski, "Cardiovascular ultrasonography in cattle," *Veterinary Clinics of North America*, vol. 25, no. 3, pp. 611–632, 2009.
- [16] M. M. Lewinter, "Pericardial diseases," in *Brunwald's Heart Disease, A Textbook of Cardiovascular Medicine*, P. Libby, R. O. Bonow, D. L. Mann, and D. P. Zipes, Eds., pp. 1829–1854, Saunders, Philadelphia, Pa, USA, 8th edition, 2007.





# Hindawi

Submit your manuscripts at  
<http://www.hindawi.com>

