Chapter

Evaluation of Current and Future Diagnostic and Prognostic Techniques for Traumatic Pericarditis in Cattle

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Abstract

Pericarditis in cattle can be classified as traumatic pericarditis (TP), idiopathic haemorrhagic, secondary to neoplasia or septic pericarditis due to haematogenous spread of organisms, such as *Colibacilli* or *Pasturella*. In cattle, pericardial disease most commonly develops from traumatic reticuloperitonitis (TRP). Bovine cardiac disease often has a poor prognosis, and this is worsened when clinical manifestations of heart failure are present. Euthanasia is the treatment of choice in many cases, but for pregnant or high value animals, treatment of disease is often the preferred option and an early diagnosis can provide a better prognosis in these cases. This chapter looks at the anatomy, physiology and presentation of TP. In addition, a more in depth look at cardiac troponin is presented alongside present and future diagnostic and prognostic methods, in addition to treatment options surrounding this clinically important problem.

Keywords: biochemistry, cardiac abnormality, cardiac troponin, cattle, haematology

1. Introduction

Pericarditis is defined as inflammation of the pericardium resulting in accumulation of fluid or exudate between visceral and parietal pericardium and can be classified as traumatic pericarditis (TP), idiopathic haemorrhagic [1], neoplastic or septic [2, 3]. Haematogenous spread of organisms, such as *Colibacilli* or *Pasturella* species which commonly originate from pleuropneumonia, are most commonly involved. In cattle, [4] the most common classification of pericarditis is TP, resulting from traumatic reticuloperitonitis (TRP), the ingestion of sharp objects which migrate through the anterior wall of the reticulum, across the diaphragm and penetrate the pericardium, in some cases extending into the myocardium [3, 4]. This can lead to pericarditis; which is defined as inflammation of the pericardium that results in accumulation of fluid or exudate between the visceral and parietal pericardium [2].

TP is one of the most common heart diseases reported in cattle [3]. From 1989 to 1995, TP was one of the top 10 findings following necropsy examination of 321 cattle at the University of Glasgow [5]. Clinical signs of the cardiovascular condition

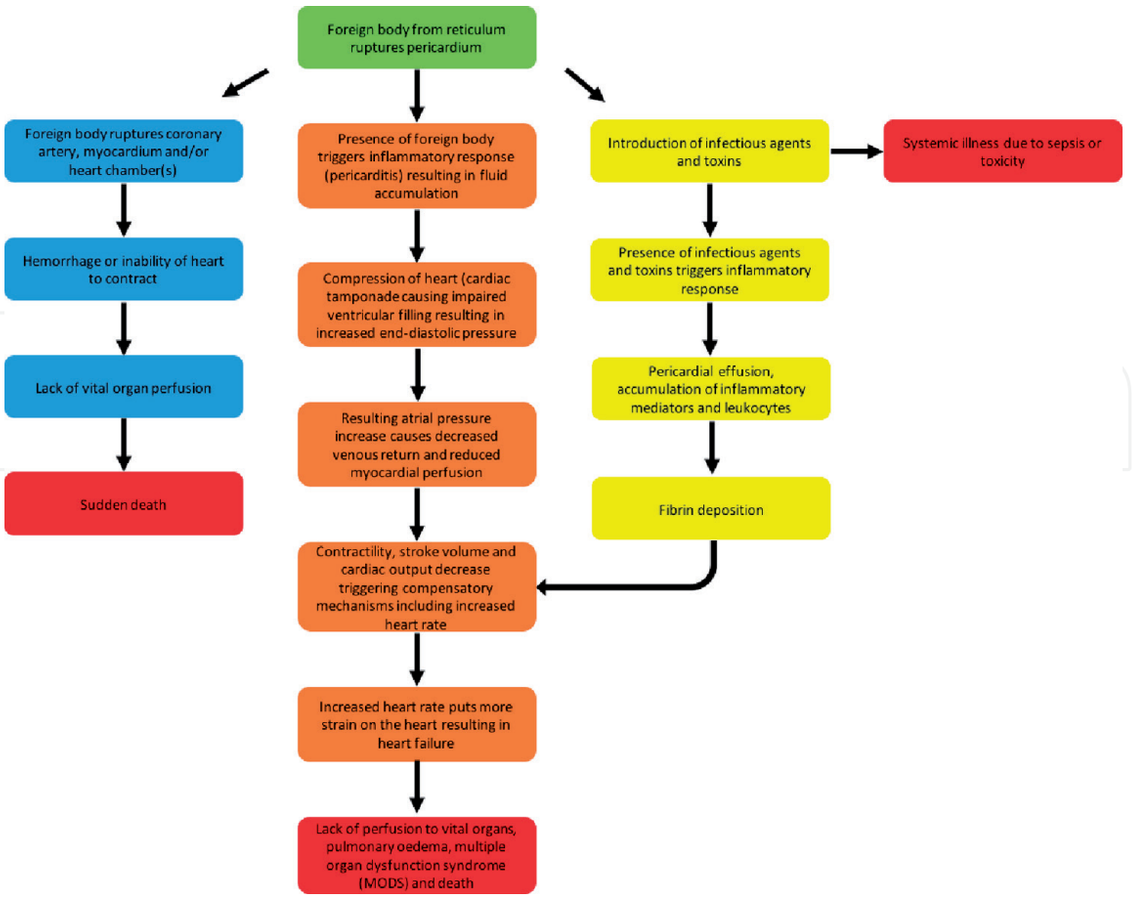

Figure 1.
Flowchart explanation of three potential outcomes of TP, with clarification of how HF can occur as a result of TP. Adapted from [10].

can vary greatly as they depend on the extent of disease progression, location of the foreign object, route of migration [6, 7], and potential for other diseases to present with similar signs [8]. Cases of TP do not commonly present until the later stages of the disease process when subsequent heart failure (HF) is evident [9] (see **Figure 1**). Due to varying clinical signs and late presentation of those signs, diagnosis can be difficult and often requires more than a clinical examination. Commonly used methods include echocardiogram, pericardiocentesis and blood analysis which help to determine the presence and extent of disease [11]. Unfortunately, these diagnostic techniques are frequently not economically viable for clients in farm practice where expenses are usually focussed on herd health as opposed to the health of individual stock.

Bovine cardiac disease often has a poor prognosis, which is worsened when clinical manifestations of HF are present. Euthanasia is the treatment of choice in many cases [11]. However, for pregnant or high value animals, treatment of disease is often the preferred option and an early diagnosis can provide a better prognosis in these cases [11]. With regards to TRP, recognition of pericardial involvement (TP) needs to be recognised as early as possible in the disease process to prevent further suffering of the animal and economic losses for the farmer. Ideally, a cheap and quick method of diagnosing pericarditis in cases of cattle with TRP should be established. In this review, we assess the recent research focusing on concentrations of cardiac biomarkers (specifically cardiac troponin 1 (cTnI)) in the serum of TP cattle and evaluate both the current and future capabilities of such tests. The advantages and limitations of other diagnostic techniques are also explored, with reference to survival rates of TP affected cattle.

2. Clinical examination

The main presenting complaints in cases of TP are often vague and non-specific; relating to milk drop, anorexia, lethargy, and weight loss [4, 10]. A review study demonstrated that in 60 animals diagnosed with fatal traumatic reticulitis (15 of which developed TP), the most common presenting sign was a sudden reduction in milk yield, which was observed in 68% of the cases.

Early signs of pericarditis, which can be detected through a clinical exam, include muffled heart sounds with associated splashing sounds, pericardial friction rubs, tachycardia, and pyrexia [10, 12]. Fluctuations in parameters such as rumen turnover, faecal output, heart rhythm, lung sounds and demeanour may also be found [12].

Pain is also an important component of TP which may present as bruxism, grunting, unwillingness to move, abducted elbows or a positive response to the cranial bar and withers test [10, 12]. Not all signs are present in every case, and they are also not exclusive to TP [12], other pathologies and physiological changes can be associated with such signs [11].

The signs mentioned above are also commonly seen in TRP cases irrespective of known TP development [7]. Clinical examinations alone have been shown to miss instances of TP. In a group of 28 animals which had TP, confirmed by necropsy, only 15 were diagnosed with TP using clinical examinations alone [13]. The lack of consistency in clinical signs between TP cattle is attributed to individuals presenting at different stages of disease with some showing evidence of HF. In addition, differences are observed as a result of the location of the foreign object, which specific anatomical structures the object has penetrated and the volume of fluid present in the pericardium [10]. Pregnancy and parturition have been also highlighted as possible contributing factors, due to increased pressure on the reticulum [13]. The variable degree of clinical signs and contributing factors associated with TP emphasises that only a tentative diagnosis can be made on clinical examination alone [4, 12, 14].

Some publications have emphasised that auscultation of the heart is the most significant aspect of a clinical exam in identifying pericarditis [3, 9]. Evidence of muffled heart sounds or other abnormal sounds such as splashing, tinkling, and rubbing are also commonly associated with pericarditis [11]. In a previous study, it was shown that muffled heart sounds had a sensitivity of 92% and specificity of 93% to pericarditis disease in 39 confirmed cases [6]. It should be noted that the presence of muffled heart sounds does not specify the type of pericarditis and the absence of such sounds does not necessarily rule out pericarditis [12]. Muffled heart sounds have also been demonstrated in other, non-cardiac diseases. One study observed muffled heart sounds in all 7 cases of pleurisy and all 5 cases of mediastinal abscesses, compared to only 39 of 55 TP cases [15]. This study also found that splashing and tinkling heart sounds were only found in the TP cattle, and not those with pleurisy or mediastinal abscesses. The presence or absence of tinkling sounds might help to rule in but not rule out TP.

Abnormal heart sounds do not always remain constant within TP cases, their presentation can change daily and the severity and time frame of disease progression of each animal should also be taken into account [16]. This highlights the need to reassess clinical signs in dubious cases. As well as auscultation, a full cardiac clinical exam should include assessment of mucous membrane colour, capillary refill time, a description of heart sound audibility and intensity with appreciation for cardiac rhythm and rate. In addition, pulse strength, rate, quality and the rhythm of both jugular and mammary veins, auscultation of lung fields and percussion of

the cardiac region to assess level of cardiac dullness should be conducted [12, 17]. This comprehensive assessment allows for clarification of the clinical status of each cow and indicates whether there is potential disruption of the cardiac system. This can help indicate the most likely disease responsible, however, further diagnostic approaches are needed to fully determine the underlying aetiology and type of pericarditis, if present.

HF is the penultimate stage of all heart disease due to failure of the compensatory mechanisms [18, 19] (see **Figure 1**). Any heart disease seen in cattle can present with or without clinical signs of HF, as demonstrated in previous cases of pericarditis [19, 20]. Most clinical signs associated with chronic late stage TP relate to congestive right sided HF which can present with signs such as jugular distension, submandibular and brisket oedema [4].

It should be noted that a lack of any of the aforementioned signs of TP found on clinical examination does not mean that TP is not present. A study using clinical examination alone identified that 2 of 28 cattle under investigation had TRP with no signs of TP. However, on post mortem examination it was discovered that all 28 cases had profound signs of TP. This demonstrated that the development of TP in cases of TRP cannot be excluded via clinical assessment alone.

TP has been reported as the most common cause of pericardial disease and congestive heart failure (CHF) in cattle [15]. Despite this, other diseases should be considered when signs of congestive HF are apparent. Differentials include other causes of pericarditis, bacterial endocarditis, primary dilated cardiomyopathy, congenital heart disease, cardiac lymphoma, mediastinal abscess and non-cardiac exudative pleurisy with much research evident on these other possible diseases too [10].

In conclusion, all differential diagnoses should be taken into careful consideration by the veterinarian when considering the presenting signs in suspected cases of TP. This is especially important in cases where presenting signs are vague or inconsistent. TP cannot always be ruled out by clinical examination alone and TP should always be considered when cattle present wit TRP. A full, cardiac clinical exam (**Table 1**) is paramount when reviewing the possibility of cardiac disease, to accurately provide diagnostic and prognostic information to the client.

3. Haematology and biochemistry

The most frequent reported haematological finding with TP is reduced clotting time indicated by the glutaraldehyde test in 93% of 28 TP cases which had presented with a heart rate of more than 100 bpm, distended jugular veins and abnormal heart sounds [12]. Other recorded signs include hyperfibrinogenaemia and leucocytosis with neutrophilia and lymphocytopenia [12]. Hyperproteinaemia, hypoalbuminaemia and hyperglobinaemia have also been consistently found in buffalo TP cases [21].

Haematology and biochemistry results only support findings indicated by clinical examination and provide limited additional information on underlying disease processes. It has been previously stated that biochemistry and haematology results alone were not enough to differentiate between endocarditis, pericarditis and congenital heart defects in cattle and it could be argued that farmers' money would be better spent on the use of other diagnostic techniques [6]. However, it could also be said that haematology and biochemistry results are necessary in order to rule out disease secondary to HF such as liver congestion which is commonly found secondary to HF. Haematology and biochemistry also gives an indication to the severity of the HF and therefore can be used to determine an accurate prognosis.

Clinical sign	% affected (*N*)	Reference
Tachycardia	85.7 (28)	[13]
Decreased rumen turnover	82.1 (28)	[13]
Milk drop	78.8 (85)	[4, 13]
Weight loss	63.1 (57)	[4]
Jugular vein distension	62.4 (85)	[4, 13]
Tachypnoea	60.7 (28)	[13]
Muffled heart sounds	57.6 (85)	[4, 13]
Oedema	51.8 (85)	[4, 13]
Anorexia	48.2 (85)	[4, 13]
Pain (bruxism, muscle fasciculations or Grunting)	42.9 (28)	[13]
Splashing heart sounds	25.0 (28)	[13]
Pericardial friction rubs	7.14 (28)	[13]
Tachyarrhythmia	3.57 (28)	[13]

Table 1.
Commonly observed clinical signs associated with TP in order of their frequency in scientific studies.

4. Radiographic imaging

Radiographic imaging is considered a useful method to identify metallic objects within the reticulum and adjacent structures, as well as highlighting findings such as cardiomegaly and abnormal cardiac shape [21]. One caveat of using radiography in late-stage pericarditis is that often, fibrinous deposits, adhesions, pleural effusion and pneumopericardium can be so severe that there is a profound loss in thoracic detail making it difficult to identify offending object(s) [6, 10, 22]. A lack of a foreign object seen on thoracic radiographs does not rule out TP, and as a result, the sensitivity of radiography as a diagnostic technique is limited [11, 22]. Additionally, in cases of extensive fluid accumulation or concurrent pleural effusion with TP, radiographic changes will be indistinguishable from other cardiac pathologies such as pleuritis [10].

5. Echocardiography

Ultrasonography is currently recognized as the preferred diagnostic method for investigating cardiac abnormalities, including pericardial effusion in cattle [11]. Echocardiography is non-invasive and can be performed on the standing and compliant animal on site [5]. This does not include evaluation via Doppler due to higher expense and problems with accessibility in farm practice. When carrying out an echocardiographic assessment it has been recommended that a 3–3.5 MHz transducer is used observing caudal long, caudal short and cranial long axis views on the right side and caudal long and cranial long axis views on the left side [23, 24].

Common signs noted within the literature relating to thoracic ultrasonography and echocardiography in TP cases include displacement of the heart away from the thoracic wall, increased echogenicity of the heart, generalized thickening of the pericardium and the presence of a hypoechoic fluid exudate between the parietal and visceral pericardium, which often contains echogenic fibrin deposits [6, 25]. As a result of fluid accumulation in the pericardium, causing a subsequent cardiac

tamponade, compression of ventricles and reduced ventricular motility can also be observed [11, 26–28]. The ultrasonographic findings described provide strong evidence for the diagnosis of pericarditis.

Factors to consider when using echocardiogram include the training experience of the clinician, the views obtained and the restraint and position of the cow. To ensure a full and complete assessment, ultrasound images should also be obtained from the lung fields, mediastinum pleural space and abdominal region, extending the scan from the 3rd intercostal space to the 12th [15]. It has been suggested that a productive and conclusive echocardiogram would take 20 minutes to perform in a farm setting [29]. Studies have also highlighted the importance of assessing the reticulum in retrospect to the diaphragm in order to interpret signs of abscess formation, adhesions and peritonitis, which may occur alongside TP [21]. This subsequently adds additional time to the scanning session but is performed at the farmer's request. In a study of 51 healthy cattle, it was shown that differences in cattle temperament, body condition score and rib width can result in a lack of consistency between thoracic ultrasonography assessments [26]. This further emphasises the importance of clinician experience when producing echocardiograms in cattle, alongside good anatomical comprehension to prevent over interpretation.

6. Ultrasound-guided pericardiocentesis

Ultrasonography alone is unable to explicitly determine the type of pericarditis which may be causing fluid accumulation in the pericardial sac. Pericardiocentesis is needed to characterise the fluid present and is often performed at the left fifth intercostal space, 2.5–10 cm dorsal to the olecranon [10]. Samples obtained from cases of TP are often malodourous, purulent, have elevated protein content (>3.5 g/dl) and on cytology show elevated white blood cells (>2500/μl) (mainly neutrophils) and the presence of mostly commensal bacteria [10]. In comparison to TP, idiopathic pericarditis is rare and in these cases pericardiocentesis is often haemorrhagic. Clinical signs may also improve with the pericardiocentesis, unlike with TP [1, 28].

Risks with pericardiocentesis may be reduced when it is performed with ultrasound guidance rather than blind. However, these risks are still plausible and can include pneumothorax, cardiac puncture, pericardial fluid leakage and formation of arrhythmias [13]. Pericardiocentesis has been described as a successful treatment option alongside pericardial lavage, pericardiostomy and rib resection in some cases of TP but evidence still remains variable [19]. Studies have shown that performing pericardiocentesis can prolong life when an animal has TP and is pregnant, so that it may deliver [30].

7. Necropsy

A definitive diagnosis of TP can be provided via post-mortem examination and this is carried out in most academic studies to allow correlation to other clinical findings [6, 11]. On necropsy, a malodourous, fibrinous to purulent pericarditis with extensive adhesions is found in TP cases. Other changes are also frequently found within the thorax and abdomen; these commonly include a congested liver, hepatomegaly, liver abscessation and peritoneal effusion [6, 11]. Sometimes the foreign object is located penetrating through the reticulum, diaphragm and pericardium, but not always [4, 11].

8. Serum cardiac troponin

Studies across many species have demonstrated the specificity of cardiac troponin to the heart muscle, and elevated levels of cardiac troponin subunits, especially cTnI, which shows a positive correlation with acute myocardial damage [31, 32]. Cardiac biomarkers have therefore been studied in farm practice following the need for an accessible and cheap method of detecting cardiac damage. Analysis of cardiac biomarkers in serum samples of TRP affected cattle could provide information on the prognosis and has the potential to surpass other diagnostic techniques especially in reference to TP [33–35].

There is a significant difference in cTnI levels between healthy cattle and those with cardiac disease. A study found that serum cTnI was higher in 4 out of 5 confirmed pericarditis cases, compared to 34 healthy control animals [36]. In a different study which used a cut off value of 0.08 ng/ml, elevated levels of cTnI was observed in 14/18 pericarditis cases, 12/15 endocarditis cases, 5/10 congenital heart defect cases, 3/7 mediastinal abscesses, 3/5 caudal vena cava thrombosis cases and in 4/13 cattle with chronic suppurative pneumonia [33]. All of these conditions were confirmed at necropsy. By investigating the concentrations of cTnI in 40 perceptively healthy cattle another study concluded that the normal range of cTnI in normal, healthy, lactating (Holstein) dairy cattle was 0.00–0.05 ng/ml (using iSTAT- immunoassay). Although, serum cTnI concentrations are usually increased with cardiac disease in cattle, this parameter cannot be used to differentiate pericarditis from other heart diseases (**Figure 2**), or to differentiate primary cardiac disease from other non-cardiac, intrathoracic diseases [33, 36, 37]. Despite the research conducted to date, further studies are also necessary in order to confirm the normal range of cTnI in healthy cattle which can then be used to determine specific cut off values for use in disease assessment. However, standardization of cTnI assays is difficult due to the use of different antibodies in differing assays [38]. Additionally, the assays used in cTnI investigations were designed for use in human medicine, cattle specific assays may be more appropriate.

Whilst comparison of serum cTnI levels between cardiac diseases has not been fully established, an additional complicating factor is non-cardiac disease. Elevated cTnI levels have been demonstrated in non-cardiac intrathoracic and non-intrathoracic diseases in cattle. The concentrations of serum cTnI in cattle with metritis, mastitis, left displaced abomasum, downer cow syndrome and other calving and post-calving complications determined that in 43 of the 53 diseased cattle cTnI

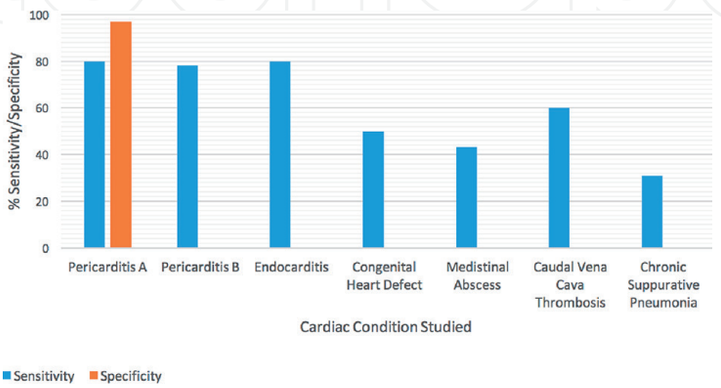

Figure 2.
Sensitivity and specificity of cTnI as a test for various cardiovascular diseases including two studies on pericarditis. Information adapted from Pericarditis A [37], and Pericarditis B, endocarditis, congenital heart defect, mediastinal abscess, caudal vena cava thrombosis, chronic suppurative pneumonia [34].

values were above the cut off value of 0.02 ng/ml [39]. Cattle with downer cow syndrome showed the highest levels of serum cTnI out of these diseases, with a maximum value of 27 ng/ml. This illustrates the need to consider secondary myocardial involvement as a result of other disease processes [39] and again, it reflects the importance of a thorough clinical exam. The research also found that non-surviving diseased cows (which died or were euthanized) had significantly higher median cTnI than surviving cows and that the cTnI in healthy cows was in the undetectable range (<0.02 ng/ml). However, it was concluded that further standardization studies are necessary to confirm such claims.

Using cTnI to detect TP and therefore determine the prognosis for cattle with TRP has been investigated [40]. Investigations found that measurement of serum cTnI in cattle with TRP could potentially provide an earlier diagnosis of TP. Making an early diagnosis of TP could save time and money for the vet and farmer and reduce suffering for cattle, since treatment, at present, is generally unrewarding and the prognosis is poor particularly when diagnoses are made further along the disease process [10].

Concentrations of cTnI have been recorded in cattle with confirmed TRP to try and show possible myocardial degeneration [37]. In 55% of confirmed TRP cases, significantly elevated levels (>0.3 ng/ml) of cTnI were observed, with the mean cTnI recorded at 3.26 ng/ml (standard deviation of 2.1 ng/ml). This compares to 0.052 ng/ml mean serum cTnI (standard deviation 0.001 ng/ml) recorded in 10 healthy control animals that were used in the study.

It should be considered that elevated cTnI levels in the serum of TRP affected cattle may not correlate directly with the severity of myocardial damage. No post-mortem examinations, echocardiograms or thorough clinical examinations were described in the report, therefore potential correlations of cTnI concentration and the stage/severity of disease could not be established [37]. A positive correlation between the magnitude of cTnI increase and the severity of myocardial damage on histopathology has been demonstrated in cattle with monesin toxicosis [41] and in calves with Foot and Mouth disease [35]. Another study [36] concluded that increased serum cTnI might be more likely in acute presentations of TP compared to chronic cases, as is seen in human medicine. This emphasizes a need to consider the magnitude of the rise in serum cardiac biomarker levels over a significant timeframe.

A more recent study [42] assessed other cardiac biomarkers including heart-type fatty acid-binding protein (H-FABP), Pentraxin-3 (PTX-3) and thrombomodulin (TM) in cases of TP in cattle. There were significantly elevated H-FABP, TM and PTX-3 levels in the 25 Holstein TP positive cattle compared to 10 healthy control animals. The elevations in these biomarkers positively correlated with elevations in cTnI. However, it was concluded that there is a need to correlate cTnI levels with other cardiac biomarkers not just in TP but also other cardiac diseases. Additionally the need to correlate H-FABP, PTX-3 and TM to necropsy and histological findings in TP has been highlighted. The use of multiple cardiac biomarkers in such cases could help to further confirm thoughts of pericardial involvement when no cardiac signs are observed clinically and this could assist with determining prognosis before the expense of treatment is pursued [42].

9. Conclusion

In this review, we have identified the need for a gold standard cardiac examination to be performed in all ruminant cases where cardiac disease is suspected. We have also re-emphasised the ability for echocardiogram and pericardiocentesis to

provide a diagnosis of TP. These methods are also required to help review and evaluate the use of cTnI as a possible indicator of prognosis in TRP with TP. We highlight the need to correlate serum cTnI levels and other cardiac biomarkers to the severity of myocardial damage present, and the correlation of such values to the stage of TP via findings on echocardiogram, necropsy examination and histopathology. There is also a need to carry out further research into serum cTnI in larger cohorts of cattle over a significant time-frame starting from initial TRP through to severe TP cases, in order to validate its use as a commercially viable and dependable parameter. In order to do this, specific cut off values for disease level and severity, and a normal cattle cTnI reference range still needs to be defined.

However, this review suggests that with further investigation and if proven to be reliable, serum cardiac biomarkers such as cTnI have the potential to revolutionise diagnosis of traumatic pericarditis in cattle.

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Conflict of interest

The authors declare no conflict of interest.

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