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REVIEW ARTICLE

PERICARDIAL EFFUSION IN DOGS: DIAGNOSTIC FEATURES OF RADIOGRAPHY, ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY

***[1]Mudasir Bashir Gugjoo, [1]Mozammel Hoque, [1]Abhishek Chander Saxena, [1]Malik Mohammed Sham Suz Zama and [2]Mohd Iqbal Yatoo**

[1]Division of Surgery, Indian Veterinary Research Institute-Izatnagar, Bareilly, UP. India- 243122
[2]Division of Medicine, Indian Veterinary Research Institute-Izatnagar, Bareilly, UP. India- 243122

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ABSTRACT

Pericardial effusion (PE) is an abnormal accumulation of fluid between epicardium and pericardium and may occur due to tumour masses, infectious agents or can be idiopathic in nature. It is more prevalent in large male dog breeds with higher predisposition of Golden retrievers. There are number of modalities that can be useful in diagnosing the pericardial effusion, however, each in one way or other the lack potential for confirmatory diagnosis and thus multiple diagnostic modalities are employed. Radiographic features that are evident in PE include the increase in cardiac silhouette and a Globoid/basket ball appearance of heart. On electrocardiography, decrease in QRS complex voltage and presence of electrical alternans are the usual findings observed. Echocardiography aids in the detection, localization and quantification of pericardial effusion in dogs and is the most sensitive method available for detection of pericardial effusion in dogs. Although, these techniques enable us to diagnose the condition, however, They may not be sufficient enough to arrive at the aetiology of PE. The present review covers the role of radiography, electrocardiography and echocardiography in the diagnosis of pericardial effusion.

INTRODUCTION

Pericardial disorders are emerging as an increasingly common and important in small animal practice and have been found to comprise approximately 8% of the total canine cardiac disorder case load at referral institutions (Tobias, 2010; Smith and Rush, 2000). Pericardial diseases are manifested in a number of ways ranging from trivial pericardial effusion to cardiac tamponade (Pepi and Muratori, 2006) from thickened to the constricted pericardium, with pericardial effusion (PE) being the most common type of abnormality in dogs (Buchanan, 1992). Pericardial effusion is more prevalent in large breeds with predominance in males and middle aged dogs (Berg and Wingfield, 1984; Gibbs *et al*., 1982). However, there are also reports which show no effect of gender on the occurrence of PE (Stepien *et al*., 2000). With regard to breed predisposition, Golden retriever is most commonly reported. The other dog breeds with higher incidence include Labrador, German shepherd, St Bernard, Newfoundland and Great Dane (Johnson *et al*., 2004; Aronsohn and Carpenter, 1999; Dunning *et al*., 1998; Cobb and Brownlie 1992; Gibbs *et al*., 1982; Berg *et al*., 1984). Majority of the cases of pericardial effusion occur without any obvious underlying disease (idiopathic pericardial effusions), and tend to affect middle-aged to older large breed dogs. Tumours are also common causes of pericardial in dogs (Campbell, 2006). Other causes are chest trauma, myocardial rupture and pericarditis, though not as common. Aetiology of PE in dogs cannot be detected solely on the basis of physical examination (Miller and Sisson, 1995; Stepien *et al*., 2000), but may be identified based on the results of different diagnostic modalities *viz*., radiography (Thomas and Reed, 1986; Tobias and McNiel, 2008), electrocardiography, haematological and pericardial fluid examination (Sisson and Thomas, 1999; Tobias and McNiel, 2008), and echocardiography (Berg and Wingfield, 1984; Cobb and Brownlie, 1992; Tobias and McNiel, 2008). The present review covers the role of radiography, electrocardiography and echocardiography in the diagnosis of pericardial effusion and/or cardiac tamponade in dogs.

Radiography

Thoracic radiography is often the first diagnostic imaging technique used for cardiac diseases, although, stressful for dogs with pericardial effusion. Ventrodorsal views of the thorax are preferable to dorsoventral or lateral views as ventrodorsal views lessen the patient's stress (Smith and Rush, 1999). Forcing a patient into a position in an attempt to obtain a perfect radiograph may cause the patient to decompensate and die and, therefore, should be done cautiously. Radiography is generally considered an insensitive indicator in deducting the cause of effusion although it might detect the pulmonary masses which represent the primary cardiac tumors (Johnson

Corresponding author: Mudasir Bashir Gugjoo,
Division of Surgery, Indian Veterinary Research Institute-Izatnagar, Bareilly, UP. India- 243122.

et al., 2004). A large globoid cardiac silhouette (Basket ball/Football heart) which may or may not be merged with pericardium is supportive of pericardial effusion (Tobias, 2010; Davidson *et al*., 2008; Stepien *et al*., 2000; Bouvy and Bjorling, 1991). In acute cases, however, such a finding may be absent as there is lack of time for the pericardial sac to stretch (Adeyanju *et al*., 2012; Tobias, 2010). Pleural effusion may also be noted on radiographs affected with PE (Smith and Rush, 1999; Berg and Wingfield, 1984; Gompf, 1980). Caudal vena cava distention, hepatomegaly, ascites, and pleural effusion may be present if the patient has developed congestive heart failure (Miller and Sisson, 2000). Sometimes, an elevated trachea may be observed in lateral radiographs due to the overall increase in the size of heart (Adeyanju *et al*., 2012). There are some measurement techniques *viz*., vertebral heart score (VHS), intercostal space method and cardiothoracic ratio wherein the relative size of the heart is calculated. VHS (Fig.1) is the most accurate radiographic index for identifying pericardial effusion (PE) and differentiating it from other cardiac diseases in dogs (Gugjoo *et al*., 2013a; Gugjoo *et al*., 2013b; Guglielmini *et al*., 2012). The mean VHS for dogs is 9.7±0.5 and 10.2±0.83 vertebrae in lateral and ventrodorsal radiographs, respectively (Buchanan and Bucheler, 1995). However, there is considerable breed variation with regards to normal heart size and shape. So, it is important to consider the specific breed value whenever the heart is being evaluated (Gugjoo *et al*., 2013a). Also, cardiac silhouette in some cases of PE is lost and it may be difficult to select the proper reference points for calculation of VHS (Adeyanju *et al*., 2012).

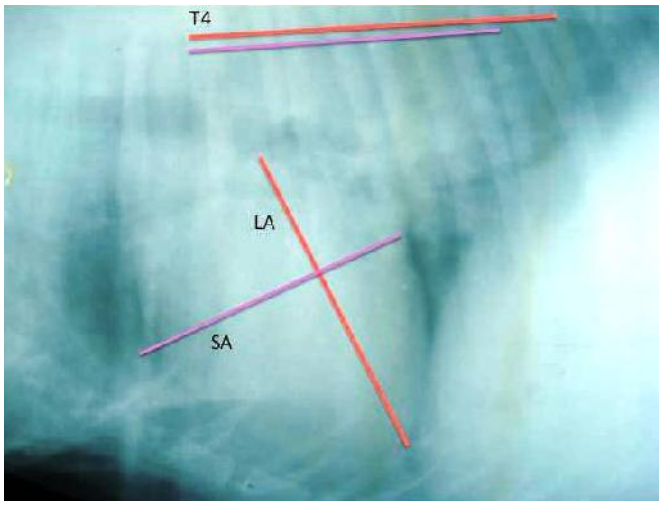

Fig.1. VHS calculation from radiograph taken in lateral recumbency. LA: Long axis; SA: Short axis; T4: 4th Thoracic vertebrae

Other cardiac measurement techniques that can be useful are comparison of cardiac silhouette with the intercostal spaces (intercostals space method) and thoracic cavity size (cardiothoracic ratio), on thoracic radiographs. A general guideline of 2.5 to 3.5 intercostal spaces for dogs with a deep and wide thorax, respectively, has been used as an indicator of normal heart size in lateral radiographic views (Owens, 1985; Kealy, 1987). Any increase in such values can be suspected for pericardial effusion. Cardiothoracic ratios, however, are not generally useful in detecting the pericardial effusion as there is consequent increase in thoracic size with the increase in cardiac silhouette. Both the above mentioned techniques can detect the increase in cardiac silhouette but cannot confirm reason behind such increase. Increase in cardiac silhouette can occur due to number of causes like dilatation cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM), pericardial effusion (PE) (Gugjoo *et al*., 2013a).

Electrocardiography

Electrocardiography is generally considered to have a low sensitivity in the diagnosis of pericardial effusion. Low voltage QRS complex (Fig. 2) is often a valuable sign for diagnosis of pericardial effusion, however, not observed in all cases of pericardial effusion. There are different reports on the incidence of low voltage QRS complexes in PE (Johnson *et al*., 2004; Stepien *et al*., 2000; Bonagura 1981). Low voltage is said to be present when QRS amplitude is less than 1.0 mV in Lead II and 0.5 mV in other leads (Kumar *et al*., 2011; Jinks, 2001; Stepien *et al*., 2000; Berg, 1984; Bonagura, 1981).

Fig. 2. Lead II of bipolar limb lead system showing low voltage QRS complexes (arrows)

This decrease in QRS amplitude may result from, electrical current short circuiting by the accumulated fluid within the pericardial space causing change in the position of the heart (Toney and Kolmen, 1966; Badiger *et al*., 2012), increasing distance from the current generator to the recording electrodes (Gonzalez and Basnight, 1991; Rokey *et al*., 1991), decrease in cardiac chamber size and volume and changes in the generation and propagation of electrical currents in the myocardium (Karatey *et al*., 1993), which may or may not exist together in decreasing the QRS voltage. Sometimes insulating effect of fibrin also lowers the amplitude and removal of fluid in such cases does not give positive response (Jinks, 2001; Johnson *et al*., 2004). P wave amplitude however, remains normal due to absence of effusion over the posterior surface of atria, which is a part devoid of pericardial duplication (Jung *et al*., 2010). Electrical alternans, characterised by beat-to-beat alterations in the QRS complex and arising from same focus, is characteristic of PE, reflecting the swinging of the heart in the pericardial fluid (Johnson *et al*., 2004; Tilley, 1985). It is relatively specific but not very sensitive for tamponade; rarely, this phenomenon is seen with very large pericardial effusions alone (Badiger *et al*., 2012; Kumar *et al*., 2011; Cobb and Brownlie, 1992). In some cases of PE beat to beat alterations may not be observed (Gugjoo *et al*., 2013c; Jinks, 2001). In man, the presence of QRS alteration during sustained narrow QRS supraventricular tachycardia is highly indicative of a retrograde accessory AV pathway in the tachycardia circuit (Green *et al*., 1975). Other less commonly documented electrocardiographic features of PE in dogs are atrial fibrillation, ventricular premature beats, supraventricular premature complexes (Johnson *et al*., 2004). All these above mentioned signs though can lead to the diagnosis of PE, but are not sufficient to confirm the cause of the PE.

Echocardiography

Cardiac ultrasound, echocardiography, aids in the detection, localization and quantification of pericardial effusion in dogs (Pepi and Muratori, 2006). It is the most sensitive method available for detection of pericardial effusion in dogs (Gugjoo *et al*., 2013c; Christensen and Bonte, 1968) and the sensitivity in detecting pericardial fluid is as high as 20 ml of fluid inside the pericardium can be visualized (Kumar *et al*., 2011; Tobias, 2010; MacDonald *et al*., 2009). Normal pericardium appears as a bright, dense layer of echoes inseparable from the epicardial echo in M-mode or two-dimensional echocardiography while echocardiogram of pericardial effusion shows an echo-free space between epicardium and pericardium (Adeyanju *et al*., 2012; Tobias, 2010; Pepi and Muratori, 2006; Rush *et al*., 1990; Freestone *et al*., 1987; Pipers and Hamlin, 1980). This echo free space, however, is not visible behind the left and right atrium due to the more adherence of pericardium to the epicardium at the base of the heart and is helpful in differentiation of pleural effusion from pericardial effusions (Boon, 1998; Bonagura and Pipers, 1981). Pericardial examination should be done in as many views as possible, including off-axis views. In fact, gain settings also have a significant effect on accuracy of diagnosing the pericardial diseases. By setting the gain low, pericardial effusion may be more precisely defined while the fibrin strands, masses and clots within the pericardial sac may be missed. Therefore, it is must to optimise the ultrasound gain and gray scale throughout the examination to get the correct information (Bonagura and Pipers, 1981). Generally, it is not possible to characterize the fluid, as serous effusion, hemopericardium and chylopericardium appear as equally clear spaces. However, hematomas and neoplastic diseases may be suspected in the presence of solid masses of echoes inside the pericardium; fibrin strands are frequently seen in chronic serous or serous–hematic effusions. Echogenic masses attached to the visceral pericardium may suggest the presence of metastases (Pepi and Moratori, 2006). There are however, some conditions which may mimic the signs of pericardial fluid. Among the most common are epicardial fat and pleural effusions. Left pleural effusions may be differentiated from pericardial effusion by utilizing all echo-windows including the posterior approach. The latter facilitates the recognition of pleural space, pericardial layers and posterior pericardial effusion. As a rule in the different views pericardial fluid reflects at the posterior atrioventricular groove, whereas pleural effusion continues under the left atrium, posterior to the descending aorta (Pepi and Moratori, 2006; Bonagura and Pipers, 1981). Other diagnostic pitfalls are: cysts, and left ventricular pseudoaneurysm (Pepi and Moratori, 2006).

Evaluation of the pericardial sac in cardiac tamponade should be performed carefully through all the echocardiographic windows in order to quantify pericardial fluid and to differentiate diffuse circumferential effusions from loculated regional ones. Excessive cardiac motion up to the so-called 'swinging heart' is frequently seen in severe pericardial effusion with chronically accumulated effusion and a minimum of adhesions (Tobias, 2010). This movement can be observed in malignancies, chronic tuberculous pericarditis and also benign viral pericarditis. An exaggerated inspiratory expansion of the right ventricle (RV) and simultaneous compression of the left ventricle is a nonspecific sign of increased direct interdependence and has therefore a low specificity (Leimgruber *et al*., 1983). M-mode and two-dimensional echocardiographic evaluation of diastolic collapse of the right atrium (RA) and RV free walls are accepted signs of cardiac tamponade (Little and Freeman, 2006; Maisch *et al*., 2004; Battle and LeWinter, 1990; Berry *et al*., 1988; Singh *et al*., 1984). Transient invagination of the RV free wall which occurs in early diastole and a transient invagination of RA wall, which occurs in late diastole and early systole (Fig. 3) is diagnostic of cardiac tamponade. The timings of these two collapses are related to the lowest intracavitary pressures occurring in the two chambers in early (RV) or late (RA) diastole, respectively (Little and Freeman, 2006; Maisch *et al*., 2004; Kochar *et al*., 1990). At the earliest appearance of RV diastolic collapse, there is the 21% reduction in the cardiac output (Leimgruber *et al*., 1983). These two signs of tamponade may be too sensitive on the one hand and lack specificity on the other (Reydel and Spodick, 1990; Armstrong *et al*., 1982). Duration of the collapse should therefore be taken into account which in fact is directly related to severity of tamponade (Reydel and Spodick, 1990). As the severity increases, RA collapse tends to begin earlier and RV collapse to extend later in diastole. Even though these two signs are too sensitive, the presence of both RA and RV collapses always indicates that the effusion is hemodynamically significant. The sequence and characteristics of RA collapse long before hemodynamic alterations of fully established cardiac tamponade has been reported in an open chest dog model by Lopez-Sendon *et al*. (1988). RA compression becomes apparent as a quick inward motion of a small portion of the posterior RA wall, and as the intrapericardial pressure increases, a wider portion of the RA wall presents an abnormal motion. With a further increase in intrapericardial pressure, RA inversion throughout the entire cardiac cycle becomes progressively apparent, and finally the complete distortion of the RA shape and dimensions indicates an extreme situation of cardiac tamponade. These experimental studies depicts the concept that echocardiography may be helpful in detecting early tamponade, and that we may always consider tamponade as a continuum of events: in the early phase of cardiac compression even minor elevations of intrapericardial pressure produce some effects on ventricular filling and echocardiographic signs could be present even in the absence of overt clinical tamponade (Fowler, 1993; Eisenberg *et al*., 1991).

There are few exceptions to the use of right-sided collapse for diagnosing tamponade: right ventricular hypertrophy and high RV intracavitary pressures may prevent the occurrence of these collapses (Leimgruber *et al*., 1983). Left atrial and left ventricular collapse is rarely seen in patients with cardiac tamponade. This is mainly due to local factors; first, the left ventricle is much thicker and stiffer than the other chambers and for that reason it resists collapse. The left atrium is posteriorly positioned and it is tightly clasped by the pericardium; rarely, in cases with very large effusions, fluid does get behind the left atrium causing wall collapse. Facilitating factors of left atrial and ventricular wall collapse are therefore, posteriorly loculated effusions and conditions in which pressures in these chambers are relatively low. Normal heart Doppler studies show change in flow velocities across different valves during inspiration. Slight decrease in flow velocity across mitral and aortic valves occurs along with

increase in flow velocity across tricuspid and pulmonic valves (Tobias, 2010). These respiratory changes of flow velocity in humans become markedly accentuated with pericardial effusion, even in absence of clinical signs of cardiac tamponade. Such Doppler indicators have been observed in dogs with experimentally induced tamponade. However, the clinical utility of Doppler studies in the evaluation of pericardial effusion in small animals is yet to be evaluated (Tobias, 2010).

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