Pericardial effusion and cardiac neoplasia in dogs

The presence of a pericardial effusion most often indicates one of two things: idiopathic, self-limiting inflammation, or a neoplastic process in a vital organ. As such, approaching dogs with a diagnosis of pericardial effusion can present challenges in decision making and client communication. Here, we review how to perform pericardiocentesis as well as various considerations and treatment options for dogs diagnosed with tumours of the heart. Novel approaches and methods of palliating incurable neoplasia are available which can help to improve quality of life and prolong survival.

Key words: Neoplasia; heart; dog; haemangiosarcoma; tamponade

Introduction

Tumours of the heart and recurrent, unexplained pericardial effusions seem like an insurmountable obstacle for most clinicians – sometimes, they prove to be. However, recently through the use of more advanced diagnostic, interventional and surgical techniques, we may be able to help treat these most challenging of cases. This article aims to review the causes of pericardial effusion in dogs and discuss a practical approach to pursuing a diagnosis. We will focus on the three most common types of cardiac neoplasia and discuss possible treatment options, case selection, and expected outcomes.

The pericardium: structure and function

The pericardium is a sac which surrounds the heart and serves to anchor the heart in place within the thorax, provide a degree of protection to the heart from outside structures, and act as a barrier against the spread of infection. It is effectively a reflection of the pleura, and comprises two layers; the fibrous (outer) and serous (inner) pericardium. The pericardium covers not only the heart but the proximal parts of the great vessels and venous inflow to the atria. The serous pericardium is known as the epicardium where it is in contact with myocardial tissue. In the normal animal, a small volume of fluid is present in the potential space formed by the layers. This is thought to help lubrication of structures during cardiac motion. The phrenic nerve passes over the pericardium, approximately one quarter to one third of the distance from heart base to apex.

Pericardial effusion

The pericardial sac has a blood supply and lymphatics, like most other bodily structures, so can be subject to inflammation, fluid transudation/exudation and haemorrhage. This most often manifests as clinically significant fluid accumulation in the potential space between the fibrous and serous pericardial layers.

The pericardium is a relatively non-distensible structure. If fluid accumulates within the pericardial space, the pressure within the pericardium (i.e. around the heart) will rise. If the pressure reaches a level that exceeds the pressure within the right atrium – the lowest pressure cardiac chamber – then systemic venous inflow to the heart is impeded. This is manifest on echocardiography as right atrial collapse, which changes in a dynamic manner dependent on the phase of the cardiac cycle and respiration (Figure 1). Owing to reduced stroke volume, clinical signs of low cardiac output (“forward failure”) and, later, fluid accumulation (“congestive heart failure”) are seen. The syndrome of right sided heart failure secondary to a pericardial effusion is called “cardiac tamponade”.

In patients with gradual accumulation of pericardial fluid, the pericardium may stretch over time to accommodate the fluid. Eventually, clinical signs will occur, but this phenomenon can lead to relatively large volumes of fluid being present. In our clinic, the current record volume is from a German Shepherd Dog, where we obtained approximately 2.5 litres of effusion at pericardiocentesis. The opposite situation – where fluid accumulates rapidly within the pericardium – can cause cardiac tamponade with a relatively low volume of fluid, because the pericardium does
not have time to stretch and therefore the pressure rises more rapidly to significant levels. It is inferred by many clinicians that cardiac tamponade associated with smaller volume pericardial effusion is more likely to represent true haemorrhage, rather than a haemorrhagic effusion (where the PCV is usually in the range 3-15%), and therefore have a neoplastic cause.

Identification of a pericardial effusion

**History:** May be vague, with lethargy, depression and reduced appetite common but non-specific findings. Exercise intolerance is almost always present. A surprising number of dogs present with gastrointestinal signs (vomiting or diarrhoea), presumably because of altered perfusion within the abdomen. Many owners report weight gain or abdominal distension, reflecting the presence of ascites. Some dogs present with syncope, and rarely a cough may be reported (possibly because of cough receptors in the pericardium itself). Exposure to toxins should be interrogated, in case of exposure to warfarin-type substances, and a foreign travel history may raise suspicion of fungal disease (area dependent).

**Physical examination:** Ascites is common and, in combination with jugular vein distension and variable filling (timed with respiration), is a typical finding of pericardial effusion. Clipping of the haircoat over a jugular vein will significantly aid identification of these cases in practice. Heart sounds may be quieter than expected, or a previously reported heart murmur may be absent due to muffling of sound as it travels through pericardial fluid. Tachycardia and a regular rhythm (loss of sinus arrhythmia) are typical findings. Pulse quality may be weak, or may even vary with respiration — so called pulsus paradoxus. This latter finding is pathognomonic for pericardial disease in dogs (Box 1).

**Radiographic findings:** The cardiac silhouette appears globoid and very clear, because the fluid distended sac is not moving as rapidly or frequently as the heart during contraction, so borders are more clearly identified radiographically. The lungs may appear hypovascular because of poor right heart output, and the caudal vena cava is often distended and sagging due to increased systemic venous hydrostatic pressure. Hepatomegaly and a loss of abdominal definition (ascites) may be present, caused by cardiac tamponade. For cardiac neoplasia to be visible, it must be a large mass lesion (Figure 2).

**Echocardiographic findings:** A “black” appearing, anechoic space around the heart indicates pericardial fluid. This method of imaging is far more specific than radiography, and may identify a mass lesion on the heart, or large thrombus within the pericardium — both of which should influence clinical decision making and owner counselling. Pleural effusion may be mistaken for pericardial fluid in some cases, but this can be differentiated by identification of lung lobes surrounded by fluid.

**Laboratory findings:** A pre-renal azotemia may be present in the face of reduced cardiac output, and hepatic congestion can

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**Box 1:** What is pulsus paradoxus?

In the normal dog, palpable pulse quality changes marginally — often imperceptibly — with respiration. On inspiration, negative intrathoracic pressure returns more blood to the right heart. This increases cardiac output (according to the Frank-Starling Principle) from the right ventricle, through the pulmonary vessels, back to the left heart, and then out to systemic arteries. In other words, increased right heart filling leads to increased left heart output and arterial pulse pressure on inspiration. On expiration, the reverse occurs: reduced right heart filling is associated with reduced left heart output.

In a dog with pericardial effusion causing cardiac tamponade, the ability of both chambers to expand simultaneously is lost because of the increased pressure surrounding the heart. Now, inspiration fills the right heart but this causes compression of the left heart within the pericardium, and an inability of the left heart to fill. As a result, left heart output is reduced on inspiration and increases upon expiration. This paradoxical flow in time with respiration is pathognomonic for cardiac tamponade.

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