CARDIAC TAMPONADE SECONDARY TO INTRAPERICARDIAL TUMOR IN A DOG. CASE REPORT

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Abstract

Presence of an intrapericardial tumor can produce effusion accumulation, sometimes in a great amount, creating pressure over the myocardium and reducing the cardiac diastole. This process is called cardiac tamponade and brings together a series of changes, both in cardiac activity and the entire cardiovascular system. The pericardial effusion associated with the presence of a cardiac tumor is uncommon in dogs, with a frequency of 7%, while the cardiac tumors without pericardial effusion amount to 3% of the total cardiac diseases studied. A 12 years old male Mioritic shepherd dog was referred to the Clinics of Veterinary Faculty of Iași, showing signs of apathy, anorexia, severe dyspnoea, that have been lasting for four days prior to examination. After the clinical examination, other special exams were recommended. Radiological examination, cardiac ultrasonography, electrocardiogram, pericardial centhesis and examination of the pericardial effusion have been performed. Clinical examination revealed severe dyspnoea and abdominal respiratory efforts, fatigue and mucosal cyanosis. In auscultation, cardiac sounds were dimmed. Femoral pulse palpation revealed cardiac asynchrony. X-ray showed cardiac enlargement and pulmonary oedema. Electrocardiography indicated high cardiac frequency, ventricular contraction reduction, electrical alternance of the R wave, signifying the presence of the pericardial effusion. Ultrasonography showed high amounts of pericardial effusion and a hyperechoic structure attached to the right cardiac free wall. Two hundred milliliters of sanguinous liquid were extracted during pericardiocentesis. The clinical examination is the one that helps suspect the presence of the pericardial effusion, but the certain diagnosis can only be established through special exams. The etiology can only be established through cytological or histological examination, but the presence of the pericardial effusion and the tumor can be confirmed through ultrasonography, radiography and the mechanical alterations of the heart can be highlighted through electrocardiography.

Key words: cardiac tumor, dog, pericardial effusion.

INTRODUCTION

The presence of cardiac tumor can produce the accumulation of pericardial fluid, sometimes in large amount, thereby influencing the activity of the myocardium, through the fluid’s pressure on the heart. This phenomenon is called cardiac tamponade and sums up a series of changes not only in the heart but also of the entire vascular system and whole organism.

The pericardial effusion associated to cardiac tumor is rare in dogs, being encountered in 7% of cardiac diseases, and neoplasms without effusion in 3% of the total cardiac diseases (Fox et al., 1999). The excessive accretion of pericardial fluid appears due to various factors such as neoplasms (57,1%), hemangiosarcoma (33,3%), chemodectoma (11,9%), other tumors like mesothelioma, lymphosarcoma, or metastasis (11,9%), cardiac primary causes (11,9%), traumatic (4,8%), infectious (2,4%), atrial...
rupture (2.4%), or metabolic diseases (2.4%) (Chetboul, 2005). In humans, among the cardiac tumors which produce pericardial effusion and cardiac tamponade, the most common is the angiosarcoma, usually located in the right atrium (Masauzi et al., 1992). The pericardial effusion accumulation process induces certain restrictions over the heart mechanism known as cardiac tamponade, which manifests through the rapid increase of pericardial pressure, exceeding at some point the right ventricular pressure, then the left one, compromising the ventricular diastolic function and inducing the specific systemic effects. The goal is to describe and explain the course of examination and the differential diagnosis in cardiac tumors with generation of pericardial fluid and the occurrence of cardiac tamponade, presented on a practical case.

MATERIALS AND METHODS

A 12 years old male Mioritic shepherd dog with torpidity, anorexia and severe respiratory restrain lasting for four days was referred to the medical clinic from the Faculty of Veterinary Medicine from Iași. The medical history and the clinical examination has been performed and further investigations have been recommended in order to establish a diagnostic of certitude. Cardio-thoracic radiography, cardiac ultrasonography, electrocardiography, and pericardiocentesis have been performed for the examination of the pericardial fluid. Within the clinics of the Faculty of Veterinary Medicine in Iași, the radiologic exam has been performed in the Roentgen-diagnostic service, using the Intermedical basic 4006 mobile x-ray machine, in the two classical incidences (right lateral and dorsal-ventral), the cardiac ultrasonography has been performed with the Esaote Aquila Pro Vet ultrasound machine, with convex transducer, on a 5 MHz frequency, with the patient in a standing position, using the right parasternal ultrasonographic window, in B-mode transverse and longitudinal view of the heart and M-mode of the transversal mid-ventricular view (Chetboul, 2005; Kealy, 2011; Pennick, 2008). The electrocardiography has been performed with the Poly-Spectrum 8E/8V device, on the vigil animal, in sternal-abdominal recumbency, before and all during the pericardial puncture process (Bexfield, 2010). The pericardial centesis was carried out on the fully awake animal, in a sternal-abdominal position, in the 5th intercostal space, parasternal, under ultrasonography guidance. The extracted fluid was examined cytological on a MGG smear.

RESULTS AND DISCUSSIONS

The clinical examination has revealed severe respiratory restrain with abdominal breathing, effort intolerance, the cyanosis of the mucous membranes. On the auscultation the cardiac sounds were dimmed. On the palpation, the femoral artery revealed pulse asynchrony. The cardiothoracic x-ray, on lateral recumbency, revealed a mixed pulmonary alveolar, interstitial and bronchial pattern. Alveolar filling with aerated bronchia in the right cardiac lobe was observed. The primary bronchi walls appear radio opaque and intense perihilar radiopacity with diffuse aspect – compatible with the cardiogenic pulmonary edema. Presence of small amount of pleural fluid that separates the dorsal border of the lungs from the thoracic wall has been noticed. The heart line was covered by the radio opaque pulmonary lobes and the pleural effusion (Figure 1). The cardiac silhouette was hardly detectable, the heart extends in a cranial-caudal direction from the 4th to the 9th rib, the heart height was 76% of the thorax height measured at the same point, the vertebral heart score (VHS) was 12, the caudal vena cava was not visible; the dorsal incidence indicated alveolar and interstitial filling in the perihilar space, flattened right cardiac lobe and compact pulmonary area on the right thorax between the 3rd and 4th intercostal spaces, with aerated bronchogram, pleural effusion. Global cardiomegaly with round cardiac silhouette was visible, specific for pericardial effusions. The heart extended cranial-caudally from the 4th rib to the 10th, and the width is 84% of the total thorax width measured at the same point.
The electrocardiography indicated a heart rate of 190 bpm, sinus rhythm, the P wave with an amplitude of 0.12 mV and a length of 0.06 s, the R wave amplitude of 0.90 mV and a length of 0.06 s, the amplitude of wave T of 0.36 mV, the PQ interval of 0.09 s, and the QT interval of 0.18 s, medium R-R of 0.31 s. The increased heart rate indicated the diminished degree of diastolic filling and the organ necessity to increase the overload.

Figure 1. Cardiothoracic x-ray. A – right lateral incidence shows the presence of pulmonary infiltrations in the diaphragmatic lobes, high radio opacity on the heart projection area and modified cardiac silhouette; B – dorsal incidence, highlights the round cardiac line that occupies almost all the thorax, radio opacity of the left hemi-thorax pulmonary area and the flattening of the right cardiac lobe;

The low voltage of the R wave (under 1 mV) which is explained by a decrease of the ventricular contraction and the electric alternation defined by the voltage variation of the R wave from a complex to another are specific effects of the accumulation of pericardial fluid (Jinks, 2001; Michael 1999).

Figure 2. Electrocardiogram. A – the specific complexes for each derivation I-III, aVL, aVR, aVF; B –
Electrocardiogram in the 2nd derivation.

Ultrasonography sections have been performed through the right parasternal window, through the long and short axis of the heart, in the two-dimensional mode. It has been observed the presence of an anechoic strip between the myocardial wall and the pericardium, with a width of 3.57 cm, measured at the base of the heart, from the free wall of the right atrium towards the pericardium which corresponds to an elevated quantity of pericardial fluid, the presence of an intra-pleural anechoic area which signifies the presence of pleural effusion. The right atria and ventricular wall appear thinned, the ventricular filling movements appears modified by an abnormal kinetics of the right ventricular free wall and the presence of diastolic collapse (Sisson et al., 1999). On the right atria free wall it has been noticed a hyperechoic, circular area, measuring over 2 cm, without intracardiac proliferation. The same hyperechoic and circular area has been observed in the right parasternal trans-aortic view through the base of the heart, hence the origin of the formation could not be specified (Figure 3 a-f).
The cytological diagnosis was modified transudate, intracavitary hemorrhage, tumoral activity with suspicion of epithelioid hemangiosarcoma or epithelioid mesothelioma. The ultrasonographic guided pericardiocentesis has been performed for diagnosis and treatment objectives. Approximately 200 ml of serosanguinous fluid have been extracted, with expressed deposit, from which the cytological exam has been performed.

The smear examination from the deposit (MGG coloration) has revealed rich sanguine cellularity: numerous erythrocytes, rare neutrophils, lymphocytes, monocytes; isolated or 2-3 mesothelial cell groups, macrophages (numerous eritrophagocyte); pleomorphic cellular groups: anaplastic mesenchymal cells, with epithelioid aspect, abundant cytoplasm, basophilic, numerous vacuoles, metachromatic granules, round, oval, kidney-shaped nucleus, with chromatin blocks, 1-3 nucleoli that are poorly viewed. The cellular conglomeration reveals a low intercellular adhesion and moderate malignity characteristics: anisocytosis, anisokaryosis, intranuclear and cytoplasmic vacuolization, but have intense phagocytic activity (Figure 4);

![Echogram](image1)

**Figure 3.** Echogram: view through the right parasternal window A, B, transverse section through the heart base (trans-aortic), underlining an anechoic intra-pericardial strip represented by fluid (PF); C – long axis of the heart with the marking out of the 5 chambers (left ventricle – LV, left atrium – LA, right ventricle – RV, right atrium – RA, aorta – Ao); D,E,F – right parasternal view, trans-aortic section, highlighting a suspect tumoral formation (STF) on the free wall of the right atrium and the presence of pericardial fluid (PF).

![Pericardial fluid](image2)

**Figure 4.** Pericardial fluid obtained through pericardiocentesis. A. Group of neoplastic, hyper-basophilic, epithelioid cells, with anisocytosis, anisokaryosis, cytoplasmic vacuolization and erythrophagocytosis. B. Mesenchymal cells, oval, star-shaped, with a medium basophilic cytoplasm. Two cells highlight erythrophagocytosis. In the upper side there is a group of 4 mesothelial cells. Co MGG, x 1000
The pathological process is a classical one, through the mechanical and biochemical changes that take place, having most of the times a bad prognosis, with a fast evolution towards exitus. The neoplastic formation is the starting point of the changes through the pericardial effusion it produces. The presence of pericardial effusion is normal, in the dog being between 1-15 ml (Jinks, 2001). Forward, the intrapericardial pressure modifies, with effects on the myocardium and especially on its diastolic function. While the fluid accumulates in the intra-pericardial space, the right ventricular pressure equalizes and the right cardiac tamponade appears, then the left one, producing the left cardiac tamponade, therefore the ejection volume drops significantly.

The diagnosis is based on the clinical exam, corroborated with paraclinical exams such as cardio-thoracic x-ray, cardiac ultrasonography, electrocardiography, and the etiological diagnosis based on the cytological examination of the pericardial fluid. (Ikede, 1980). Computed tomography is recommended for localizing the tumoral suspect formations thus replacing the radiological exam (Cote et al., 2013). The ultrasonography is the most important exam for the diagnosis of pericardial effusion (Vogtli et al., 1997), but in order to decide the fluid nature, other special exams are required.

The neoplasia is the most common cause of the pericardial effusion in the dog (Bomassi, 2004).

Among the cardiac neoplasms, the hemangiosarcoma (HAS) is ten times more frequently met in dogs than the other types of cardiac tumors (chemodectoma, mesothelioma, ectopic thyroid carcinoma ) (Jinks, 2001). The epithelioid versions of HAS or mesothelioma are rarely reported in veterinary medicine and could be a diagnosis challenge in the case of cytological examinations of cavitary effusions (Shor et al., 2009).

In a classic way, if the cytological examination of the pericardial effusion reveals angiod structures and hemosiderin (the metachromatic granules revealed in this case could be hemosiderin granules) or erythrocytes in the cytoplasm (obvious in this case), should be suggested the diagnosis of hemangiosarcoma (Funahashi et al., 1995). In many of these cases, due to the difficult differentiation of the cellularity in the mesothelioma from the one seen in the mesothelial reactivity caused by the presence of a large quantity of pericardial effusion, the cytological suspicion of mesothelioma, has proved to be hemangiosarcoma at the histopathological examination (Shor et al., 2009; Ellison, 2010).

In general, in the pericardial effusion associated to a tumor, the prognosis is reserved to bad because of the increased quantity of accumulated pericardial fluid, which compresses the myocardium and depreciates the diastolic function, and the treatment is often ineffective due to the fast rate of the fluid renewal. Diuretics are ineffective, even contraindicated because they increase the risk of creating a fall of the refill pressure, therefore increasing the cardiac tamponade (Bomassi, 2004). Neither is the pericardiectomy indicated. The only recommended treatment is the surgical removal when possible, chimiotherapic and cardiac treatment for the improvement of the cardiac function.

CONCLUSIONS
The clinical exam is the one which orientates the diagnosis towards the presence of pericardial effusion, but the confirmation is made only with paraclinic and laboratory exams. The etiological diagnosis can be established only with cytological or histopathological examinations, but the confirmation of the presence of pericardial fluid and tumoral formations can be decided based on the ultrasonography, while the mechanical changes of the heart are shown with the help of electrocardiography.

In the case of pericardial effusion secondary to cardiac tumors the prognosis is bad due to its fast accumulation. The treatment is of less use and a surgical treatment requires a high-performance equipment.