CONGENITAL PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA (PPDH) IN A MIXED BREED DOG

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Abstract

Congenital pericardial diseases are rare in dogs and cats and most part of them were reported as incidental findings on radiography or necropsy examinations. In this case report, a definitive diagnosis of congenital peritoneo-pericardial diaphragmatic hernia (PPDH) in a three year old male mixed breed dog (Canis lupus familiaris) is described. The dog was referred in relatively shock status with a history of anorexia, dyspnea, cough, abdominal distension, progressive weight loss, but died after conservative treatment was applied. At necropsy examination, a cranial displacement of abdominal viscera (portion of right hepatic lobe) into the pericardial sac was observed, while the pleural space was intact. Supplementary, another dog from the same nest/littermates (3-year-old female), which died in identical conditions was submitted for postmortem investigations and showed no congenital abnormalities.

Keywords: PPDH, congenital diaphragmatic hernia, pericardial disease

INTRODUCTION

Peritoneopericardial diaphragmatic hernia is a common congenital abnormality which involve the pericardium of dogs and cats. These congenital abnormalities were reported as incidental radiographic findings while evaluating other problems or at necropsy examination (Keirandish et al. 2014, Statz et al. 2007). PPDHs occurs as a result of an embryonic development defect of the dorsolateral septum transversum in the socalled sternocostal triangle structure. Persistent communication between the peritoneal and pericardial cavities allows the abdominal organs to herniate into the pericardial sac without involving the pleural space (Berry et al. 1990, Evans et al. 1980, Keirandish et al. 2014, Nelson et al. 2014). Other congenital anomalies such as umbilical hernias (most frequent). hydrocephalus, sternal defects, cranial midline abdominal hernias, abnormal swirling of hair on the ventral abdomen, intracardiac defects can be associated with this anomaly (Bellah et. al. 1989, Evans et al.

1980, Neiger 1996, Hunt et al. 2003, Statz et al. 2007, Wright et al. 1987). The initial onset of clinical signs associated with PPDH can occur at any age (ages between 4 weeks and 15 years have been reported). The majority of cases are diagnosed during the first 4 years of life. In some animals, clinical signs never develop. Males appears to be more affected than females. Literature mentions Weimaraners among the dog breeds that show a predilection for this disorder, as PPDH with a percentage of 0.5% of their congenital cardiac diseases (Evans et al. 1980, Nelson et al. 2014). A similar percentage of feline peritoneopericardial diaphragmatic hernia is frequently identified in Persian cats, whose pattern of inheritance is consistent with an autosomal recessive trait (Dêbiak et. al. 2009, Neiger 1996).

MATERIAL AND METHODS

A mixed-breed dog (3-years-old male of *Canis lupus familiaris*) weighting around 12 kg, body lenght of 45 cm was submitted for postmortem

investigations: necropsy and histopatological examinations.

RESULTS AND DISCUSSIONS

Postmortem changes prove a period of approximately 24±8 hours after the installation of death. To sustain this observation specific postmortem changes appear. as corneal dehvdration and hemoglobin imbibition. General appearance attests а good maintenance.

The pericardial sac was severely deformed with a marked increased volume and presenting a breach in the intimate contact with the diaphragm in the form of hernial ring about 2 cm width, through which a portion of the diaphragm conjunctly with about 35% of the right medial lobe of liver (Fig. 1).



Fig. 1 – Abnormal topography of liver, mixed breed dog, 3 years



Fig. 2 – Section in the pericardial sac with heart and portion of liver

The diaphragmatic membrane hernias's portion was intact, engaging with it a liver lobe which is strangled by the hernial ring described above. The part of liver spotted in pericardial cavity with the diaphragm shows a whitish pink color, suggesting an increased fibrosis consistency and presenting a visible stricture that demarcate the affected portion of adjacent liver tissue (Fig. 3).



Fig. 3. - Part of right medial lobe liver spotted in pericardial cavity

The hepatic fragment mentioned and the diaphragm hernias portion showed no adherence to adjacent tissues (Fig. 4).



Fig. 4. Increased hepatic fibrosis and ring of strangulation (black arrow)

The right atrium presented an increased volume (partial atrial dilatation),the left ventricle being reduced. Endocardium presented thickening of right atrioventricular valve with corresponding features for vegetative endocarditis and left ventricular hypertrophy.

The lesion from the lungs was represented by incipient pulmonary oedema (frothy exsudate was present in the trachea and bronchi but not in the nasal cavity). The spleen presented lack of normal tissue continuity (fragmented) with marked areas of fibrosis, suggesting an previous injury/trauma. The urinary bladder showed a moderate amount of urine (aprox. 100 ml) and insignificant quantity of yellow-orange dry, putty-like material. The stomach presented scant red fluid and no distension. Postmortem discoloration occurred in intestine. The kidneys were normally situated and the capsules striped easily revealed a smooth surface. The corticomedullary demarcation was obscured by congestion.

Histopathology examination:

Lungs presented diffuse distension of alveolar and interstitial capillaries. The alveolar space was occupied by protein-like material mixed with red blood cells (hemorrhages and oedema). The interstitial space was increased by moderate mononuclear infiltrate, also discreet in some areas. The bronchioloalveolar space was frequently blocked by mucus, desquamated cells and inflammatory cells (neutrophils and macrophages).

Liver: The hepatic parenchyma entrapped in the pericardial sac presented abundant connective tissue especially in the porto-biliary space. Additionally, fibrosis area were associated with marked bile duct hyperplasia. The spleen keeps histological architecture with a clear demarcation between the red and the white pulp.

The most severe lesions identified in gross and histological examination were identified in respiratory system expressed as interstitial pneumonia and oedema, the other injuries being discrete changes, secondary to respiratory causes and agonic state. The congenital anomaly, incidentally detected, caused functional disorders of cardiac activity without major pathological implications. The lesions described in lung may be the result of infectious disease (Distemper/ Kennel cough). The other dog from the same nest which was examinated had the same macroscopical lesions excepting the peritoneopericardial diaphragmatic hernia. As with other diaphragmatic herniations, clinical signs of affected animals may vary from none to severe respiratory impairment, depending on herniated organs and degree of organ damage.

The liver and gall bladder are herniated most frequently, followed by small intestine, spleen and stomach. (Ettinger et. al.2017). Peritoneopericardial diaphragmatic hernias are rare but should be included as a differential diagnosis in dogs with enlarged cardiac silhouette. The main tool for diagnosis of congenital diaphragmatic hernia is radiography. In this case the injuries provoked by congenital abnormality didn't cause the death of the studied animal. The case reports available in the literature indicate that the intensity of clinical symptoms vary according to size of diaphragm defect, as well as the kind and volume of the herniated organs (Dêbiak et al. 2009).

CONCLUSIONS

Peritoneopericardial diaphragmatic hernia in dogs can be an incidental finding during necropsy examination.

Depending on the volume of the herniated organ can be appreciated if it is a cause of death. The fragment of liver surprised in the pericardial sac presented ischemia with degeneration and secondary fibrosis. The cause of death in the examinated dog was respiratory insufficiency.

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