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Recurrent Spontaneous Pneumothorax in a Maltese Dog with Endogenous Lipoid Pneumonia

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ABSTRACT

Background: Pneumothorax is a clinical condition which can cause respiratory distress. It can have as its origin traumatic causes or even classified as spontaneous, mainly related to diseases of the lung parenchyma. Lipoid pneumonia is rarely described in dogs, and it is characterized by globules of lipid in the alveolar spaces. Endogenous lipoid pneumonia (EnLP) occurs when lesions on pulmonary cells release cholesterol and other lipids in the alveoli. There is no clinical approach established for EnLP in veterinary patients. The aim of this report is to describe a case of a young Maltese dog, with recurrent spontaneous pneumothorax in which EnLP was diagnosed *post mortem*.

Case: A 2-year-old sexually intact male Maltese dog was evaluated for restrictive dyspnea. Clinicopathologic findings included cyanotic, muffled chest auscultation with hypersonic thoracic percussion. Chest x-ray demonstrated an increase in pleuropulmonary radio transparency and a floating-looking heart, indicating pneumothorax. Complete blood counts and biochemical panel results were normal. *Dirofilaria immitis* antigen test results were negative. Computed tomography demonstrated slightly hyper-expanded pulmonary fields, with slightly enlarged reticular marking with areas of mild multicentric panlobular emphysema and a fracture on the sixth left rib. The treatment was focused on improving the breathing pattern through sedation, supplementation with oxygen, and thoracentesis. Owing to the reserved prognosis of the case, the unknown etiology of the recurrent pneumothorax, and the clinical worsening of the patient, the owner opted for euthanasia. Necropsy displayed multiple, circular whitish areas in the lungs, distributed over the surface of all lobes. Histopathological examination revealed pulmonary tissue with the subpleural micronodular foci, multifocal to coalescent, with a moderate accumulation of foamy intra-alveolar macrophages, occasionally multinucleate, associated with cholesterol crystals compatible with endogenous lipid pneumonia.

Discussion: The patient presented with clinical signs and physical examination characteristics of pneumothorax at the first visit. After the pneumothorax diagnosis, and clinical stabilization of the patient. No predisposing factor for the formation of the pneumothorax was identified as the radiography revealed only bronchitis and blood tests were normal, the patient was thus discharged after 24 h, with the recommendations for observing the breathing pattern. Initially, spontaneous pneumothorax was suspected. The antibiotics were administered since bacterial pneumonia, although not confirmed on chest x-ray, is the main cause of pneumothorax in dogs is lung parenchyma disease. With the worsening of the clinical condition of the patient, CT was performed and did not demonstrate any findings that would justify the presence of pneumothorax. Despite the placement of the chest tube for facilitating the management of thoracentesis, there was no stabilization of the condition, enhancing the frequency of centesis procedures, which led to the decision to euthanize. The microscopic examination of the pulmonary alterations was decisive for the diagnostic conclusion. The visualization of the accumulation of foamy intra-alveolar macrophages, occasionally multinucleate, associated with cholesterol crystals, was responsible for the diagnosis of EnLP. This condition is rarely described in dogs and as in the present report, it is a noninfectious inflammatory condition, characterized by intra- or extracellular globules of lipid in the alveolar spaces. In the present report, although it was not possible to determine the etiology of EnLP, we can conclude that although rare, it can affect dogs and can generate severe clinical repercussions.

Keywords: necropsy, lungs, dyspnea, cholesterol crystals.

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INTRODUCTION

Pneumothorax, is a clinical condition which can cause dyspnea, characterized by the presence of air in the pleural space [7,11]. It can have as its origin traumatic or spontaneously [7]. Parenchymal lung diseases are the main causes of spontaneous pneumothorax in dogs [7,11].

Lipoid pneumonia is rarely described in dogs [3,9], and it is a noninfectious inflammatory parenchymal lung condition, characterized by globules of lipid in the alveolar spaces [6]. Endogenous lipoid pneumonia (EnLP) occurs when lesions on pulmonary cells release cholesterol and other lipids in the alveoli, and its pathophysiology is poorly understood [6]. It is proposed that retained epithelial secretions, prolonged hypoxia and chronic respiratory disease may be related [3,4,13]. *Dirofilaria immitis* infection was also reported associated with EnLP in a dog [9] since heartworm disease leads to pulmonary cell injury [2,12]. Other studies suggest that lung neoplasm is involved [13] and it was first described recently in dogs [8].

Diagnosis is reached basically by cytological examination based on bronchoalveolar lavage or histopathological exam [5,6,10]. Chest x-ray and computed tomography may be normal or altered by secondary complications such as bacterial infections and pneumothorax [6].

There is no clinical approach established for EnLP. Secondary infections must be treated when presented and supportive care must be provided as necessary [6].

The aim of this report is to describe a case of a 2-year-old Maltese dog, with recurrent pneumothorax, in which EnLP was diagnosed *post mortem*, with no lung tumors or infections related.

CASE

A 2-year-old male Maltese dog presented with a restrictive pattern of dyspnea, with a history that he was never very active like other dogs and that always were easily tired. On physical examination, he presented with cyanotic, muffled chest auscultation with hypersonic thoracic percussion. The patient was initially placed on oxygen therapy and sedated with de butorfanol¹ [Torbugesic - 0.3 mg/kg, IV]. After dyspnea stabilization, thoracic radiograph was recorded, which demonstrated an increase in pleuropulmonary radio transparency and a floating-looking heart, indicating the presence of pneumothorax.

After the radiographic examination, thoracentesis was performed, with the improvement of dyspnea, and the patient was admitted to the hospital for observation, remaining stable for the next 24 h. Thoracic radiograph was recorded as a follow-up examination and demonstrated mild remaining pneumothorax associated with mild bronchitis. Blood samples were collected for complete blood cell count (CBC), serum biochemistry, and *Dirofilaria immitis* antigen test² (SNAP 4Dx Plus). CBC and biochemistry examinations were in normal ranges and the *D. immitis* antigen test was negative. After being clinically stable, the patient was released with a prescription of amoxicillin with potassium clavulanate [20 mg/kg/PO/BID/15 days].

Three days later, the dog was admitted presenting with similar symptoms. The protocol of thoracentesis, sedation, and oxygen was repeated, and it was kept under observation for 48 h and then discharged. After 14 days, the episode recurred. At that moment, after clinical stabilization by thoracentesis and oxygen therapy, a computed tomography (CT) scan of the thorax was requested, occurring without complications.

The CT scan demonstrated slightly hyperexpanded pulmonary fields, with slightly enlarged reticular markings with areas of mild multicentric panlobular emphysema, predominantly in the middle and caudal lobes, without images of edema, masses, or bubbles. Loss of continuity was noted in the middle third of the sixth left rib, accompanied by a slight increase in peripheral soft tissue volume.

After remaining stable for 15 days, the patient presented with a new episode of respiratory distress and was brought to emergency care. Sedation, oxygen supplementation, and thoracentesis were performed. However, the formation of pneumothorax was not detected for a maximum period of 6 h. Based on the rapid decompensation, a chest tube was placed. Owing to the reserved prognosis of the case, the unknown etiology of the pneumothorax, and the clinical worsening of the patient, making the stabilization challenging, the owner opted for euthanasia.

After authorization from the dog owner, necropsy was performed. Multiple, circular whitish areas were observed in the lungs, with a diameter ranging from 0.1 to 0.3 cm, distributed over the surface of all lobes (Figure 1). On dissection, the whitish areas did not enter the parenchyma.

On the microscopic examination, histological preparation of heart muscle and valves did not demonstrate any microscopic changes. In the pulmonary tissue, the subpleural micronodular foci, multifocal to coalescent, with a moderate accumulation of foamy intra-alveolar macrophages, occasionally multinucleate and associated with cholesterol crystals (Figure 2) were observed. These pulmonary morphological findings were compatible with those of endogenous lipid pneumonia.

DISCUSSION

As described in the literature, the patient presented with clinical signs and physical examination characteristics of pneumothorax at the first visit, such as expiratory dyspnea, hypersonic sound to chest percussion, muffled lung, and cardiac sounds on auscultation and cyanosis [1,7,11]. As there was respiratory pattern stabilization after emergency care, chest x-ray was recorded before thoracentesis. No predisposing factor for the formation of the pneumothorax was identified as the radiography revealed only bronchitis and blood tests were normal. The patient was thus discharged after 24 h, with the recommendations for observing the breathing pattern. Initially, spontaneous pneumothorax was suspected.

Although bacterial pneumonia was not detected on chest radiography, antibiotics were administered since pneumonia is a common cause of lung parenchyma related to pneumothorax in dogs is lung parenchyma disease [7,11]. With the worsening of the clinical condition of the patient, CT was performed to investigate possible lesions that had not been detected on chest x-ray [1]. Despite the CT did not demonstrate any findings that would justify the presence of pneumothorax, with the loss of continuity in the sixth rib that was not present at the time of recording the first radiograph, it is possible to infer that it was probably resulting from the patient's chronic respiratory effort [11].

Despite the placement of the chest tube for facilitating the management of thoracentesis [7,11], there was no stabilization of the condition, enhancing the frequency of centesis procedures, which led to the decision to euthanize.

The microscopic examination of the pulmonary alterations was decisive for the diagnostic conclusion [6]. The diagnosis could have been made *in vivo* through thoracotomy and lung biopsy, although there is no effective treatment for EnLP.

The visualization of the accumulation of foamy intra-alveolar macrophages, occasionally multinucleate, associated with cholesterol crystals, was responsible for the diagnosis of endogenous lipid pneumonia [6,10]. This condition is rarely described in dogs [3,9], and as in the present report, it is a noninfectious inflammatory condition, characterized by intra- or extracellular globules of lipid in the alveolar spaces [6].

In the present report, although it was not possible to determine the etiology of EnLP, we can conclude that although rare, it can affect dogs and can generate severe clinical repercussions. Therefore, more studies should be conducted for a better understanding of the disease as well as possible diagnostic and therapeutic approaches.



Figure 1. *Post mortem* examination of the dog demonstrating multiple, circular whitish areas (arrows) in the lungs, distributed over the surface of all lobes.



Figure 2. Lung photomicrograph. Multinucleate macrophages are associated with cholesterol crystals (arrow) and accumulation of foamy intraalveolar macrophages (arrowhead) [H&E; obj.40x].

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REFERENCES

- 1 Au J.J., Weisman D.L., Stefanacci J.D. & Palmisano M.P. 2006. Use of computed tomography for evaluation of lung lesions associated with spontaneous pneumothorax in dogs: 12 cases (1999-2002). *Journal of American Veterinary Medical Association*. 228(5): 733-737.
- 2 Castleman W.L. & Wong M.M. 1982. Light and electron microscopic pulmonary lesions associated with retained microfilariae in canine occult dirofilariasis. *Veterinary Pathology*. 19(4): 355-364.
- 3 Corcoran B.M., Martin M., Darke P.G.G., Anderson A., Head K.W., Clutton R.E., Else R.W. & Fuentes V.L. 1992. Lipoid pneumonia in a rough collie dog. *Journal of Small Animal Practice*. 33(11): 544-548.
- **4 Hadda V. & Khilnani G.C. 2010.** Lipoid pneumonia: an overview. *Expert Review of Respiratory Medicine*. 4(6): 799-807.
- 5 Lauque D., Dongay G., Levade T., Caratero C. & Carles P. 1990. Bronchoalveolar lavage in liquid paraffin pneumonitis. *Chest.* 98(5): 1149-1155.
- 6 Norris C.R. 2004. Lipid Pneumonia. In: King L.G. (Ed). *Textbook of Respiratory Disease in Dogs and Cats*. St. Louis: Elsevier, pp.456-460.
- 7 Pawloski D.R. & Broaddus K.D. 2010. Pneumothorax: A review. *Journal of the American Animal Hospital Association*. 46(6): 385-397.
- 8 Pérez-Accino J., Liuti T., Pecceu E. & Cazzini P. 2020. Endogenous lipoid pneumonia associated with pulmonary neoplasia in three dogs. *Journal of Small Animal Practice*. 62(3): 223-228.
- 9 Raya A.I., Fernández-de Marco M., Núñez A., Afonso J.C., Cortade L.E. & Carrasco L. 2006. Endogenous lipid pneumonia in a dog. *Journal of Comparative Pathology*. 135(2-3): 153-155.
- 10 Silverman J.F., Turner R.C., West R.L. & Dillard T.A. 1989. Bronchoalveolar lavage in the diagnosis of lipoid pneumonia. *Diagnostic Cytopathology*. 5(1): 3-8.
- 11 Sumner C. & Rozanski E. 2013. Management of respiratory emergencies in small animals. *The Veterinary Clinics* of North America Small Animal Practice. 43(4): 799-815.
- 12 Sutton R.H. & Atwell R.B. 1985. Lesions of pulmonary pleura associated with canine heartworm disease. *Veterinary Pathology*. 22(6): 637-639.
- 13 Tamura A., Hebisawa A., Fukushima K., Yotsumoto H. & Mori M. 1998. Lipoid pneumonia in lung cancer: radiographic and pathological features. *Japanese Journal of Clinical Oncology*. 28(8): 492-496.

