Spontaneous pneumothorax secondary to granulomatous pneumonia caused by Angiostrongylus vasorum in a dog in Denmark

Spodsberg, Eva-Maria Hohneck; Miles, James Edward; McEvoy, Fintan; Willesen, Jakob

Published in:
Journal of Small Animal Practice

DOI:
10.1111/jsap.12023

Publication date:
2013

Document Version
Early version, also known as pre-print

Citation for published version (APA):
Spontaneous pneumothorax secondary to granulomatous pneumonia caused by Angiostrongylus vasorum in a dog in Denmark

- A case report

EVA-MARIA HOHNECK-SPODSBERG, JAMES EDWARD MILES, FINTAN MCEVOY OG JAKOB WILLESEN
UNIVERSITY HOSPITAL FOR COMPANION ANIMALS, DEPARTMENT OF VETERINARY CLINICAL AND ANIMAL SCIENCES, FACULTY OF HEALTH AND MEDICAL SCIENCES, UNIVERSITY OF COPENHAGEN, DENMARK

Introduction
Canine pulmonary angiostrongylosis is caused by the metastrongyloid nematode Angiostrongylus (A.) vasorum (1).
Angiostrongylosis occurs worldwide with endemic foci in Europe in the UK, Ireland, Denmark and France (2,3,4,5,6). A. vasorum has an indirect life cycle with a definitive host in dogs and related canids. Adult worms reside in the pulmonary arteries within the definitive host and, after hatching of eggs shed by the female worms, the first-stage larvae (L1-larvae) migrate from the capillaries through the pulmonary parenchyma and into the smaller airways. Hence, the most commonly reported clinical signs are related to the respiratory tract and include coughing, exercise intolerance and dyspnoea, although many other clinical signs associated with A. vasorum infections have been reported (4,6). These include haemorrhagic diathesis of the lungs, brain, eyes, skin or abdomen (4,7,8,9) and haemothorax and haemoabdomen (10,8).
Pneumothorax has been mentioned in association with angiostrongylosis (11), but has not been described in detail so far.
Pneumothorax can be seen secondary to trauma or spontaneously with emphysematous bullae, neoplasia, lung lobe necrosis and parasitic infections such as Filaroides osleri (12) or Dirofilaria immitis.
(13). Recently, pulmonary thromboembolism was identified as the cause of spontaneous pneumothorax in a dog with pituitary-dependent hyperadrenocorticism (14). Identification of the underlying cause is important for the prognosis and treatment of pneumothorax.

Case History
A 2-year-old, female, intact Whippet presented to the University Hospital for Companion Animals at the University of Copenhagen with a 2-days history of rapidly progressing dyspnoea, exercise intolerance and anorexia. The dog was current on vaccinations, had no travel history outside Denmark and had not been wormed for the last 12 months. It lived in a multi-dog household in a rural environment and none of the other dogs (all Whippets) were reported to show signs of illness.

Physical examination confirmed tachypnoea (respiratory rate 80 breaths/minute), a severely increased, mainly expiratory, respiratory effort, bilaterally muffled dorsal lung sounds and increased ventral lung sounds.

On thoracocentesis, 760 ml air was evacuated from the left hemithorax while 7200 ml air was emptied from the right hemithorax without achieving a vacuum on this side. Nonetheless the dog’s condition improved and the respiratory rate decreased to 40 breaths/minute. Thoracic radiography and ultrasound, a complete blood count, serum biochemistry, urinalysis and a faecal analysis (collected rectally for a direct faecal examination) were performed.

On thoracic radiographs, a gas-filled bulla in the right caudal lung lobe and marked bilateral lobar collapse was seen in addition to a bilateral pneumothorax (Figure 1). With no history of trauma or oesophageal foreign body, spontaneous pneumothorax with ongoing leakage of air into the right pleural space was suspected. Echocardiography revealed no signs of primary cardiac involvement. No larvae were found on the direct faecal smear (15). Mature neutrophilia (15·37×10⁹/l, reference limits 3·2 to 12·1×10⁹/l) and mild hyperfibrinogenaemia (4·61 g/L, reference limits 1 to 4 g/L) were the only other abnormalities.

Under general anaesthesia, a 14-gauge small-bore wire-guided chest drain (MILA Chest Drain; MILA International Inc.) was placed in the right hemithorax using a modified Seldinger technique and continuous drainage instituted. Subsequent CT examination of the thorax confirmed pulmonary consolidation, collapse and a bulla (3 cm in diameter) in the caudal part of the right caudal lung lobe (Figure 2).

On right intercostal thoracotomy, continuous air leakage was observed from the area of the bulla. The region comprised consolidated, granuloma-like lung tissue which was removed by lobectomy.
using a stapling device (Proximate Linear Stapler TX60B, Ethicon) (Figure 3).

Radiographs on day 2 showed almost completely inflated lungs, and the chest tube was removed as no air could be evacuated. Analgesia was provided with methadone (Metadon 10 mg/ml, Nycomed Danmark) as needed, combined with a transdermal fentanyl patch (Durogesic 50 mcg/h, Janssen-Cilag), and supplemented with a lidocaine infusion (Xylocain 10 mg/ml, AstraZeneca) for the first 24 hours. Faecal samples for Baermann analysis collected during postoperative hospitalisation were positive for A. vasorum L1-larvae, and treatment with the antiparasitic agent fenbendazole in a dosage of 25mg/kg SID orally for 20 days (Panacur vet. 500 mg, Intervet) was started (16). Bacteriological and mycological culture of lung tissue was sterile. Histological examination of the necrotic lesion revealed numerous, often coalescing, granulomata centred on parasitic A. vasorum larvae and eggs in the collapsed and fibrotic lung parenchyma and irregular muscular hypertrophy of the larger pulmonary vessels giving a diagnosis of chronic granulomatous pneumonia due to A. vasorum infection (Figure 4).

On day 3 the dog was bright and alert without respiratory signs and was discharged with instructions to complete fenbendazole treatment and to keep the dog at rest. A follow-up examination was performed on day 21. Clinical examination, haematology, biochemistry and thoracic radiographs did not reveal any abnormalities. No larvae were found on Baermann analysis. The owner reported that all other dogs in the household had been subsequently diagnosed with A. vasorum infection on Baermann analysis and treatment with fenbendazole had been initiated. The owner was instructed to provide monthly A. vasorum prophylaxis with moxidectin 2.5% / imidacloprid 10% spot-on solution (Advocate, Bayer) for all dogs according to their body weight. The dog made a full recovery.

Discussion

This dog presented with a spontaneous pneumothorax which had developed secondary to a single ruptured granulomatous lung lesion, ultimately caused by A. vasorum, indicating that canine angiostrongylosis should be considered as a possible cause of this clinical condition. There were no other signs of illness prior to the acute onset of dyspnoea to indicate A. vasorum as the cause in this dog.

Pneumothorax as a main presenting sign of angiostrongylosis has not been described previously, although it has been mentioned in one case series with the suggestion that the pneumothorax may have been secondary to the dog’s dyspnoea (11). Pneumothorax can present bilaterally with a unilateral lesion as the mediastinum of most dogs is fenestrated, allowing free communication between the two pleural sacs (17). The bilateral presentation of pneumothorax in this case is therefore not surprising despite the unilateral nature of the lesion. Pulmonary bullae can develop from destruction, dilatation, and confluence of adjacent alveoli secondary to pulmonary pathology (18). Rupture results ultimately in pneumothorax as seen in our case.

Granulomatous pneumonia has been a consistent finding in dogs infected with A. vasorum (19,20). Rupture of vessels due to tissue and vascular damage induced by A. vasorum resulting in severe blood loss, haemopneumothorax, haemothorax and haemoperitoneum have been described frequently in association with canine angiostrongylosis (8,10,21). Interestingly, bleeding to the thoracic cavity was completely absent in this case.

Postoperative and 3-weeks-follow-up thoracic radiographs did not reveal an abnormal lung pattern, bronchial thickening or any signs of disseminated granulomatous pulmonary changes which could have indicated canine angiostrongylosis. Radiographic changes in dogs experimentally or naturally infected with A. vasorum commonly show a bronchial, alveolar and/or interstitial pattern and bronchial thickening mainly affecting the peripheral and dorso-caudal parts of the lung (22,23). The duration of infection with A.

Figure 4. Lung histology, Hematoxylin and Eosin (HE) stain x400. The coalescing granulomas centred on the larvae and embryos are illustrated including some heterokaryons. The lung parenchyma is collapsed, fibrotic and contains innumerable, often coalescing granulomata containing macrophages, eosinophils and multinucleate giant cells centred on parasitic larvae and eggs. No adult forms were identified. There is moderate, irregular muscular hypertrophy of the larger pulmonary vessels, and scattered haemosiderotic macrophages in the periarteriolar stroma in places. This is a chronic granulomatous pneumonia associated with immature stages of a lungworm. The concommitant vascular changes suggest that Angiostrongyulus vasorum is the most likely cause.
The clinical pathological findings registered in this case report were unspecific which is often reported in dogs naturally infected with A. vasorum (24). Larvae were not identified on a direct faecal smear while subsequent Baermann analysis made a diagnosis of A. vasorum possible. This report emphasises that it is important to consider A. vasorum as a cause of spontaneous pneumothorax secondary to underlying pulmonary pathology in dogs especially in areas endemic for this infection even in the absence of radiological changes supportive of an A. vasorum infection.

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

Reference list

17. AGUT, A. Radiology of the Mediastinum, Proceedings WSAVA 2002