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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.

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

- A case report

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Summary
Granulomatous pneumonia secondary to *Angiostrongylus vasorum* was diagnosed in a dog presenting with bilateral pneumothorax. A 2-year-old, female, intact Whippet presented to the University Hospital for Companion Animals at the University of Copenhagen with an acute history of progressing dyspnoea, exercise intolerance and anorexia. 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. Thoracic radiographs revealed a gas-filled bulla in the right caudal lung lobe with marked bilateral lobar collapse in addition to a bilateral pneumothorax. Thoracic CT examination confirmed pulmonary consolidation, collapse and a bulla in the caudal part of the right caudal lung lobe. On lobectomy of the right caudal lung lobe, consolidated, granuloma-like lung tissue was identified. Histological examination revealed numerous, often coalescing, granulomata centred on parasitic *A. vasorum* larvae and eggs in the collapsed and fibrotic lung parenchyma. Faecal samples were positive for *A. vasorum* L1-larvae, and the dog was treated with fenbendazole for 20 days. The dog made a full recovery.

Sammendrag

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.
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 hyperfibrinogenemia (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). No other potential causes of pneumothorax were identified.

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.
This dog presented with a spontaneous weight. The dog made a full recovery.

Bayer) for all dogs according to their body prid 10 % spot-on solution (Advocate, laxis with moxidectin 2.5 % / imidaclo -
to provide monthly

been initiated. The owner was instructed
and treatment with fenbendazole had
infection on Baermann analysis

been subsequently diagnosed with A.
vasorum infection (Figure 4).

On day 3 the dog was bright and alert without respiratory signs and was dis-
charged with instructions to complete fenbendazole treatment and to keep the
dog at rest. A follow-up examination was
performed on day 21. Clinical examina-
tion, haematology, biochemistry and tho-
deracic radiographs did not reveal any
abnormalities. No larvae were found on
Baermann analysis. The owner reported
that all other dogs in the household had

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, hae-
mothorax and haemoabdomen have been
described frequently in association with
canine angiostrongylosis (8,10,21). Inter-
estingly, 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 thick-
ening or any signs of disseminated granulo-
matous pulmonary changes which could
have indicated canine angiostrongylosis.
Radiographic changes in dogs experiment-
tally or naturally infected with A. vasorum
commonly show a bronchial, alveolar
and/or interstitial pattern and bronchial
thickening mainly affecting the periph-
eral and dorso-caudal parts of the lung
(22,23). The duration of infection with A.

Discussion
This dog presented with a spontaneous
eventide pneumothorax which had developed se-
condary to a single ruptured granuloma-
tous lung lesion, ultimately caused by A.
vasorum, indicating that canine angio-
strongylosis 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 indi-
cate 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 pre-
cent bilaterally with a unilateral lesion as
the mediastinum of most dogs is fene-
strated, 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 ultim-
ately 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, hae-
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(22,23). The duration of infection with A.

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 (Duro-
gesic 50 mcg/h, Janssen-Cilag), and sup-
pplemented with a lidocaine infusion
(Xylocain 10 mg/ml, AstraZeneca) for the
first 24 hours. Faecal samples for Baer-
mann analysis collected during postoper-
ative 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 myco-
logical culture of lung tissue was sterile.
Histological examination of the necrotic
lesion revealed numerous, often coales-
cing, granulomata centred on parasitic A.
vasorum larvae and eggs in the collapsed
and fibrotic lung parenchyma and irregu-
lar muscular hypertrophy of the larger
pulmonary vessels giving a diagnosis of
chronic granulomatosus pneumonia due to
A. vasorum (Figure 4).

Figure 4. Lung histology, Hematoxy-
lin and Eosin (HE) stain x400. The coale-
sing granulomas centred on the larvae
and embryos are illustrated including
some heterokaryons. The lung paren-
chyma is collapsed, fibrotic and contains
innumerable, often coalescing granuloma-
tata containing macrophages, eosin-
ophils and multinucleate giant cells cen-
tred on parasitic larvae and eggs. No
adult forms were identified. There is
moderate, irregular muscular hypert-
rophy of the larger pulmonary vessels,
and scattered haemosiderotic macroph-
hages in the periarteriolar stroma in pla-
ces. This is a chronic granulomatosum
pneumonia associated with immature
stages of a lungworm. The concommit-
tant vascular changes suggest that Angi-
strongylus vasorum is the most likely
cause.
Pulmonary infiltrates may be masked during the partial lobar collapse of pneumonia, they would have been expected in the post inflation, delayed studies. 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.

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