Imaging diagnosis

Computed tomography of traction bronchiectasis secondary to pulmonary fibrosis in a Patterdale terrier

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Abstract

An 8-year-old, Patterdale terrier was referred for evaluation of tachypnoea, exercise intolerance, and weight loss. Computed tomographic images showed pneumomediastinum, diffuse ground glass opacity, and marked dilatation of peripheral bronchi, but no thickened bronchial walls. The histopathologic diagnosis was diffuse pulmonary interstitial fibrosis, type II pneumocyte hyperplasia, and bronchiectasis. The lack of evidence of primary bronchitis supports a diagnosis of traction bronchiectasis. Traction bronchiectasis can occur as a sequela to pulmonary fibrosis in dogs.

Key words: computed tomography, dog, pneumomediastinum, pulmonary fibrosis, traction bronchiectasis

Running head: Traction bronchiectasis in a Patterdale terrier

Signalment, history and clinical findings

An 8-year-old female neutered Patterdale terrier had worsening tachypnoea, exercise intolerance and weight loss over a period of 4 months. A short course of broad-spectrum antibiotics and anti-inflammatory drugs had no apparent effect on clinical signs. At referral, there was marked tachypnea (120 breaths per minute) and inspiratory dyspnea. No abnormal breath sounds were identified on auscultation. Mucous membranes were slightly tacky with a normal capillary refill time. Cardiovascular evaluation was unremarkable and body temperature was normal (38°C). Body condition score was 3/9. Haematological and biochemical evaluation revealed slightly increased urea, creatinine, creatine kinase and total protein. Echocardiography revealed no abnormalities. Blood antigen test for canine lungworm (*Angiostrongylus vasorum*) was negative. Shortly after arrival, the patient's respiratory rate and effort increased. The dog became hypoxic and was placed in an oxygen chamber.

Imaging diagnosis and outcome

Thoracic radiography at the primary care practice 3 months prior to referral found a pneumomediastinum and diffuse, unstructured interstitial pattern affecting all lung lobes. To further characterize the pulmonary changes computed tomography (CT) of the thorax was performed using a 16-slice scanner (Mx8000 IDT, Philips, Best, The Netherlands) with the dog in ventral recumbency under general anesthesia. The CT settings were 120 kVp, 150 mA, 16 x 1.5 mm collimation, pitch 1, tube rotation time 0.5s, and 3mm reconstruction slice thickness. Images were reconstructed using medium and high frequency algorithms. Images were acquired pre- and post-

intravenous bolus injection of contrast medium at 600mgI/kg body weight (Omnipaque, iohexol, 300 mg I/mL, GE Healthcare AS, Nycoveie 1–2, NO-0401 Oslo, Norway). There was a large volume pneumomediastinum and diffuse, uniform pulmonary ground-glass opacity (700HU), and a lack of normal tapering and dilatation of the peripheral bronchi, but no apparent thickening of the bronchial walls (Fig. 1).

Dilatation of the peripheral bronchi was considered to be the major finding, indicative of bronchiectasis, whereas pneumomediastinum and pulmonary ground-glass attenuation were non-specific findings. Differential diagnoses for bronchiectasis include primary bronchial disease (i.e. chronic bronchitis) or traction bronchiectasis secondary to pulmonary fibrosis. The history, clinical signs, and lack of bronchial wall thickening supported the latter diagnosis.

A post-CT bronchoalveolar lavage revealed a mild neutrophilic inflammation. No bacterial growth was noted after four days of incubation and further culture for *Mycoplasma* spp. was negative. A short trial of systemic steroids and inhaled bronchodilator (Salbutamol) was initiated, and the dog continued to receive supplemental oxygen; however, signs continued to worsen with increasing respiratory rate and effort. The dog was euthanized at the owners' request.

At necropsy, there was pneumomediastinum and the lungs were firm and diffusely pale. Multiple small (<2mm diameter) raised foci were noted on the visceral pleural surface, which was thickened and had a wrinkled contour. Histologic examination of the lung found marked thickening of the alveolar septa and subpleural space with fibrous connective tissue. Multiple alveoli were lined by plump cuboidal cells consistent with type II pneumocyte hyperplasia. In addition, the alveolar spaces contained proteinaecous material, foamy macrophages and multinucleate

hemosiderin-containing cells (hemosiderophages). Markedly dilated terminal bronchioles were identified adjacent to the pleural surface (Fig. 2). The histologic diagnosis was marked, diffuse, chronic interstitial fibrosis and chronic-active alveolitis with secondary traction bronchiectasis.

Discussion

Traction bronchiectasis is an irreversible dilation of the bronchioles that occurs secondary to pulmonary fibrosis.[1] In humans, it is often associated with end-stage lung disease but has also been identified with other chronic lung diseases.[2] Three mechanisms of bronchial dilation have been described: damage to the bronchial wall, obstruction of the lumen, and traction from surrounding fibrotic tissue.[2] Damage to the bronchial wall is usually secondary to infection and the associated inflammatory response with release of inflammatory mediators including neutrophil elastases, which degrade mural connective tissue. In chronic bronchitis, thickening of bronchial walls, dilatation of bronchi, and mucus plugging the bronchial lumen may be observed. In traction bronchiectasis, tension arising from contraction of surrounding fibrous tissue dilates the bronchial lumen without other signs of bronchial disease. In each form of bronchiectasis, there is chronic irreversible damage to the supportive connective tissue within the bronchial and bronchiolar wall.[2]

The term “honeycombing” has been associated end-stage interstitial lung fibrosis and describes clusters of subpleural cystic airspaces. Histologically, there is complete loss of acinar architexture.[3] In the present case, there was intervening lung

between dilated airspaces, hence it would be incorrect to use the term "honeycombing".[4]

Traction bronchiectasis has been reported as a sequela to pulmonary fibrosis in dogs[5], but not illustrated. *Ante mortem* diagnosis of pulmonary fibrosis is difficult and relies on exclusion of other types of infiltrative disease such as interstitial pneumonia, neoplasia, and non-cardiogenic edema. Definitive diagnosis depends on lung biopsy, which is an invasive procedure with significant morbidity.[6] In humans, CT findings alone can now be used to diagnose idiopathic pulmonary fibrosis without the need for tissue confirmation.[7] Traction bronchiectasis is a key criterion for CT diagnosis of pulmonary fibrosis in humans.[7] Increasing severity of traction bronchiectasis correlates with a poorer prognosis in humans.[8] However such a correlation has not been made in veterinary medicine. In the present case, the CT finding of peripheral bronchiectasis was an important sign of pulmonary fibrosis.

Pneumomediastinum (and pneumothorax) have been observed in dogs with bronchiectasis.[9] In these cases, air leaks through sites of alveolar rupture, tracks along the bronchovascular interstitium and accumulates within the mediastinum. This is the Macklin effect.[10] The specific site of air leak causing pneumomediastinum was not identified in the present case. It is possible that a sudden increase in the volume of mediastinal air in this patient may account for the acute respiratory decompensation following admission.

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Legends

Figure 1. Transverse (A) and oblique (B) CT images of the thorax showing pneumomediastinum (*), diffuse ground glass pulmonary opacity, and uneven dilation of the peripheral bronchi in left caudal lobe (arrowheads) and accessory lobe (arrow) compatible with bronchiectasis.

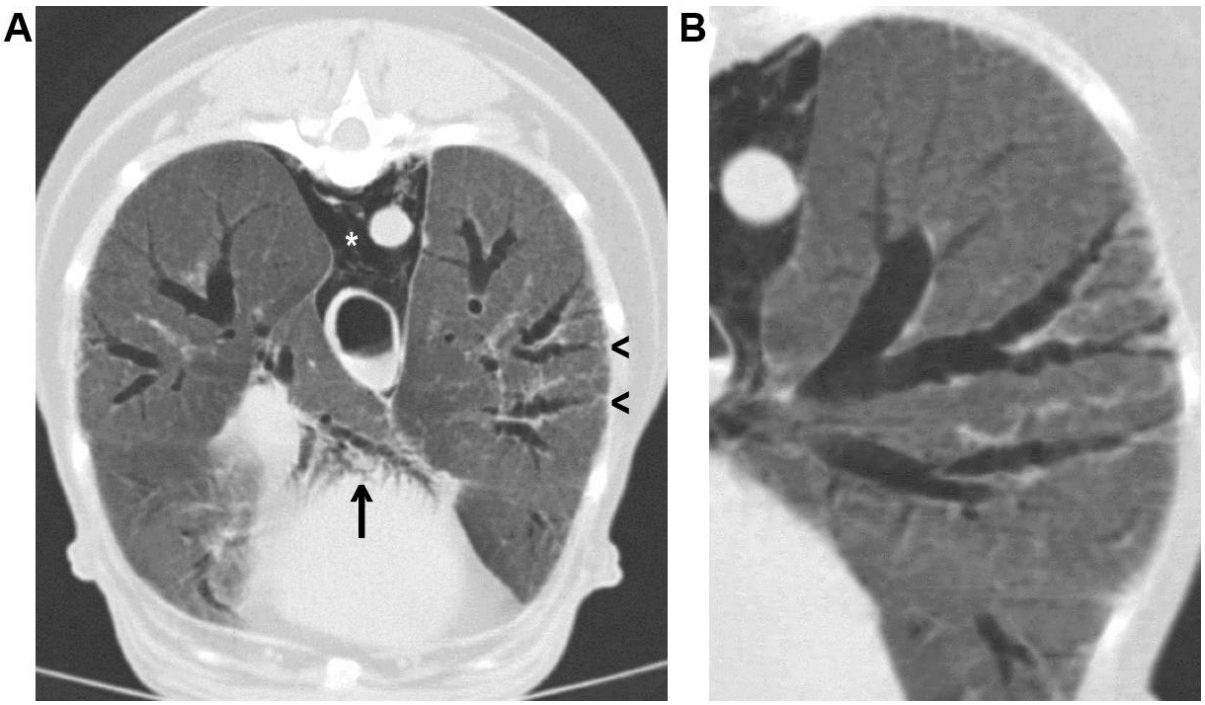

Figure 2. Hematoxylin and eosin-stained histologic sections of lung (A, x20 magnification) and (B, x20 magnification) showing a dilated bronchioles (B). The visceral pleural surface of the lung (arrowheads) appears normal, but there is marked fibrosis of the subpleural parenchyma (F). There is a lack of abnormalities affecting the respiratory epithelium of the small bronchiole (arrow). Bar = 1.5 mm