What Is Your Diagnosis?

In collaboration with the American College of Veterinary Radiology

Figure 1—Left lateral (A), right lateral (B), and dorsoventral (C) radiographic images of a 17-year-old 10.9-kg spayed female spaniel mixed-breed dog evaluated because of a 2-day history of progressive coughing and respiratory distress.

History

A 17-year-old 10.9-kg spayed female spaniel mixed-breed dog was presented for evaluation because of a 2-day history of progressive coughing and respiratory distress. Earlier that morning, the owner noticed

that the dog had markedly increased respiratory rate and effort. Medical history included prior immune-mediated thrombocytopenia and chronic cough of undetermined etiology. These were being effectively managed with prednisone (1.25 mg, PO, q 24 h) and fluticasone inhaler (q 12 h).

On initial examination, the patient was tachypneic (66 breaths/min; reference range, 18 to 24 breaths/min) with markedly increased respiratory effort. Thoracic auscultation revealed diffuse crackles in all lung fields. No stridor, stertor, or wheezes were noted. A grade 4/6 left apical systolic heart murmur with a regular rhythm, and rate of 120 beats/min was auscultated. Femoral pulses were fair, symmetric, and synchronous, and the extremities were mildly cold.

Abnormalities on the CBC analyses included mild normocytic, normochromic regenerative anemia

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(Hct, 34.3%; reference range, 40% to 55%) (68,900 reticulocytes/ μ L; reference range, 7,000 to 65,000 reticulocytes/ μ L), mild leukocytosis (15,630 WBCs/ μ L; reference range, 6,000 to 13,000 WBCs/ μ L), mild neutrophilia with a regenerative left shift and slight toxicity (13,129 mature and 1,407 bands; reference range, 3,000 to 10,500 mature and rare band neutrophils/ μ L), and moderate lymphopenia (313 lymphocytes/ μ L; reference range, 1,000 to 4,000 lymphocytes/ μ L). Thoracic radiography was performed (**Figure 1**).

Formulate differential diagnoses, then continue reading.

Diagnostic Imaging

Findings and Interpretation

Thoracic radiography revealed a marked complex pulmonary pattern. There were diffusely increased and thickened bronchial markings as well as obscuration of the pulmonary vascular markings connoting an interstitial-to-coalescing alveolar pulmonary pattern, affecting all lung lobes. The most severe changes were present in the ventral aspects of the

right middle and caudal subsegment of the left cranial lung lobes, where large air bronchograms could be seen (**Figure 2**). The pulmonary vasculature, though somewhat obscured by this pulmonary pattern, was deemed within normal limits. There was minimal bulging of the margin of the left atrium on the lateral projections; however, it was not enlarged on the dorsoventral view, with no double cardiac silhouette margin nor any splaying of the mainstem bronchi. Incidental findings included mild hepatomegaly, smooth focal expansion of the eighth right rib (likely a healed fracture), and a small pyloric mineral foreign body.

Given the long-term history of cough, the primary differential was chronic inflammatory lower airway disease with concurrent infectious bronchopneumonia. Another consideration was pulmonary edema associated with pulmonary hypertension secondary to chronic inflammatory lower airway disease. Other noncardiogenic causes for pulmonary edema (eg, postobstructive), immune-mediated inflammatory airway disease (eg, interstitial pneumonitis), and hemorrhage (eg, coagulopathy) were also considered. Given the lack of definitive left atrial or

Figure 2—Same radiographic images as in Figure 1. There is a marked complex pulmonary pattern, with diffuse bronchointerstitial and peribronchial pulmonary pattern. In the ventral aspect of the right middle and caudal subsegment of the left cranial lung, there is a heavy alveolar pulmonary pattern (white arrows). Minimal bulging of the caudal margin of the left atrium is seen on the lateral projections (white arrowhead); no left atrial enlargement is appreciated on the dorsoventral projection. The pulmonary veins are radiographically normal where visualized. There is incidentally a small smoothly margined expansion of the right eighth rib (white circle). There is also a small mineral foreign body (asterisk) in the pylorus, deemed incidental given the lack of relevant history.

pulmonary venous enlargement, left-sided congestive heart failure was considered unlikely.

To interrogate for pulmonary hypertension and to characterize the ausculted heart murmur, an echocardiogram was performed. This revealed a mildly dilated left atrium (left atrial-to-aortic root ratio in right parasternal short-axis basilar view of 1.76; reference limit, < 1.68), mildly increased left ventricular cavity size with relative systolic dysfunction, moderately thickened mitral valve leaflets, and severe mitral valve regurgitation. A flail posterior leaflet pointing toward the left atrium during systole was identified, consistent with ruptured chordae tendineae. The pulmonary veins entering the left atrium were subjectively enlarged, and there was mildly elevated right ventricular systolic pressure (peak tricuspid regurgitant gradient of 37 mm Hg; reference range, 15 to 30 mm Hg), most likely representing pulmonary venous hypertension secondary to elevated left atrial pressures. Acute left-sided congestive heart failure (L-CHF) secondary to chordae tendineae rupture associated with myxomatous mitral valve disease (MMVD) was the working diagnosis.

Treatment and Outcome

Trial treatment for L-CHF was initiated, consisting of furosemide (2 mg/kg IV q 6 h), pimobendan (5 mg PO q 12 h), and supplemental oxygen as needed. Given the atypical radiographic findings, the patient was also treated with unasyn (30 mg/kg IV q 8 h) to cover for a possible infectious pulmonary process.

Within 2 hours following initiation of furosemide, the patient began showing marked respiratory improvement. Overnight the patient's respiratory rate dropped to 20 to 28 breaths/min, and by the following day was comfortably breathing room air and had improvement in pulmonary crackles. A second set of radiographs were obtained (**Figure 3**). A bronchial

Figure 3—A right lateral projection of the same patient 1 day after starting a furosemide treatment trial. The marked interstitial-to-coalescing alveolar pulmonary pattern is now resolved; however, a diffuse bronchial pulmonary pattern remains.

pulmonary pattern remained, whereas the diffuse interstitial and ventral alveolar pattern had resolved. L-CHF secondary to MMVD and subsequent chordae tendineae rupture was thus confirmed. The antimicrobials were subsequently discontinued, and the patient was discharged to the owner with appropriate medications. At 1 month of follow-up, the patient remained well with no further respiratory distress.

Comments

Radiographs are the diagnostic modality of choice to diagnose L-CHF with the typical roentgen signs including an enlarged cardiac silhouette (specifically, definitive left atrial enlargement with regards to MMVD), enlarged pulmonary veins, and an interstitial-to-alveolar pulmonary pattern most predominantly affecting the perihilar and caudodorsal lung fields (indicative of cardiogenic pulmonary edema).¹⁻³ However, radiography can have reduced sensitivity and specificity with concomitant chronic lung disease and acute heart failure secondary to chordal rupture, both of which likely occurred in this patient.^{2,4}

The radiographic diagnosis of acute L-CHF secondary to MMVD and chordae tendineae rupture is described in both veterinary and human medicine literature.^{1,5} In a retrospective study of 114 dogs with MMVD, 7% of the dogs had chordal rupture and marked mitral valve regurgitation showed no evidence of left atrial enlargement.⁵ Similarly humans with this condition usually display minimal to no atrial enlargement.⁶ Left atrial size and duration of valvular insufficiency positively correlate.^{1,6} Typically, with MMVD, clinical signs of L-CHF take multiple years before incipience; the left atrium can expand and become more compliant to accommodate slowly progressive mitral regurgitation. Instead, with chordal rupture in early MMVD, the left atrium has not undergone substantial remodeling and is not able to suddenly adapt to the large and sudden increases in mitral regurgitation. This can lead to fulminant L-CHF without substantial echocardiographic or radiographic evidence of left atrial enlargement.

The lack of enlarged pulmonary veins may be attributed to the presence of pulmonary edema and the subsequent inability visualizing pulmonary vasculature.² However, even when visible, not all cases of congestive heart failure present with enlarged pulmonary veins. This is seen frequently in settings where patients have already received furosemide prior to the radiographic study.

The diffuse, predominantly ventral peribronchial and alveolar pulmonary pattern is atypical of congestive heart failure; normally, a caudodorsal interstitial-to-alveolar pattern is the dominant feature.^{2,4} This is likely multifactorial. First, the patient had a chronic cough of open etiology, and the bronchial pulmonary pattern remained despite furosemide treatment. Chronic inflammatory lower airway disease

is a common disease of middle-aged to older dogs. When present, this disease causes bronchial thickening creating the classic “bronchial” pattern.² Second, a “peribronchial” pattern can occasionally be seen as a manifestation of acute pulmonary edema secondary to acute L-CHF.³ Anatomically, the pulmonary veins follow the bronchi intimately; as such, when edematous fluid first begins to exit the vessel, it first gathers around the bronchi in the loose connective tissues, creating a “peribronchial” pulmonary pattern.³

Radiographs are the gold standard modality for diagnosis of congestive heart failure and are widely available.² Echocardiography is not readily available in most clinical situations, though it is necessary for definitive characterization of cardiac disease.⁴ This case highlights an uncommon variation of the radiographic presentation L-CHF, to which clinicians may have a lower sensitivity. It is important to recognize that chordal rupture specifically may result in acute L-CHF that may not display typical radiographic features.² In these cases, when high-grade, new, or progressive murmurs are heard, echocardiography is a valid diagnostic modality and will help direct

therapeutics and further diagnostics. Echocardiography is not always available. If L-CHF remains a differential, in addition to basic stabilization and oxygen support, a furosemide trial followed by radiographic monitoring may be considered a useful therapeutic and diagnostic plan.

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