

What Is Your Diagnosis?

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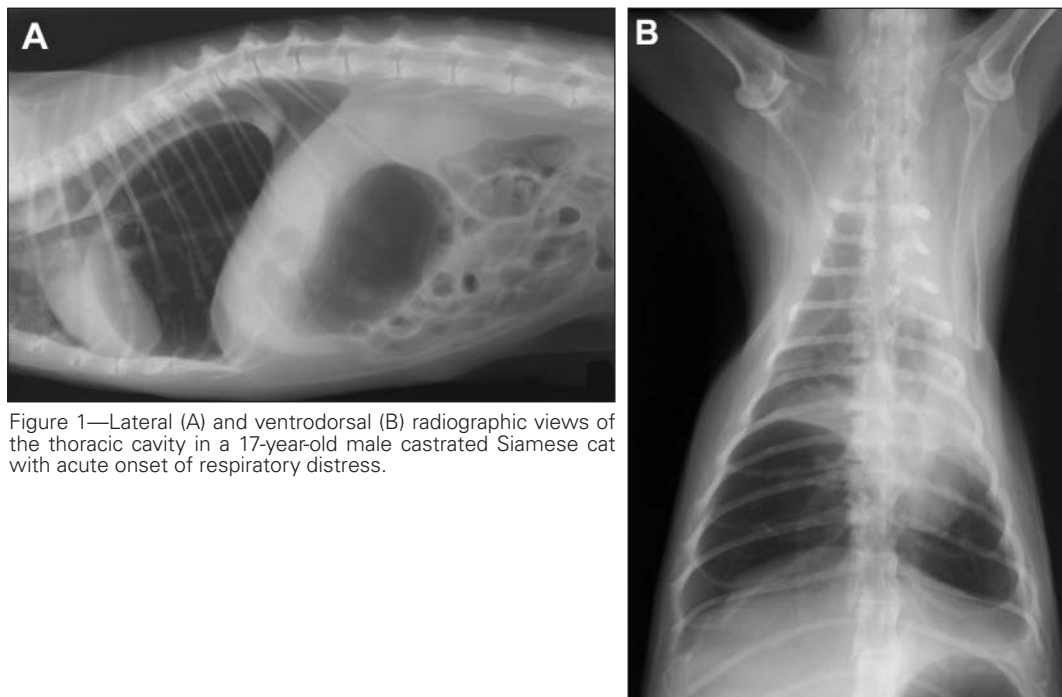


Figure 1—Lateral (A) and ventrodorsal (B) radiographic views of the thoracic cavity in a 17-year-old male castrated Siamese cat with acute onset of respiratory distress.

History

A 17-year-old castrated male Siamese cat was examined because of open-mouth breathing and cyanosis that had developed acutely within the preceding 30 minutes. The cat did not have a history of dyspnea or pulmonary disease. No known trauma was associated with acute onset of clinical signs. The cat had hyperthyroidism and was receiving methimazole. The only abnormality detected on serum biochemical analysis was mildly high serum thyroxine concentration. At the time of admission, the cat was not tested for heartworm disease or gastrointestinal parasites. The cat was severely dyspneic, and decreased airway sounds were detected in the dorsal aspect of the thorax during auscultation. Thoracic radiography was performed (Figure 1).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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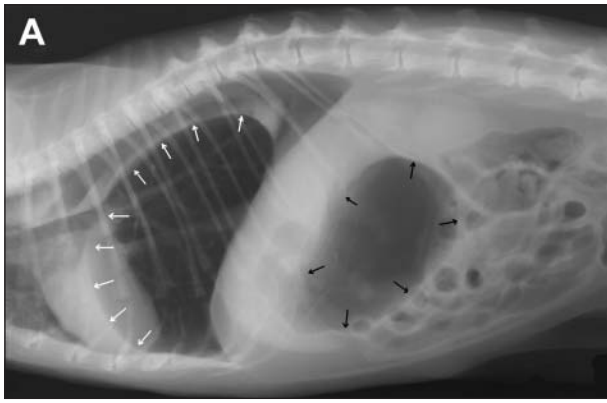


Figure 2—Same radiographic views as in Figure 1. On the lateral view, notice the hyperlucent caudal lung lobe (white arrows) and aerophagia (black arrows). On the ventrodorsal view, both the left and right caudal lung lobes are hyperlucent (white arrows). The caudal portion of the left cranial lung lobe is collapsed, and the right middle lung lobe is consolidated (black arrows). A left mediastinal shift is present.

Radiographic Findings and Interpretation

Both caudal lung lobes are hyperlucent, indicating overinflation, particularly on the right side (Figure 2). There is lung volume loss in the left hemithorax, causing a mediastinal shift to the left. The right middle lung lobe is consolidated. A large amount of air in the stomach and small intestines is indicative of aerophagia.

Comments

The cat initially responded to treatment with oxygen and antimicrobials but later developed a pneumothorax that was nonresponsive to repeat thoracocentesis. A thoracic tube was placed, but the cat continued to accumulate air within the thoracic cavity at an alarming rate and was taken to surgery. A right lateral thoracotomy revealed the right caudal lung lobe was severely hyperinflated and actively leaking in 2 places. The remaining right lobes and accessory lobe were atelectic. The left lung lobes were collapsed and not inflating sufficiently with manual ventilation. Hyperinflation was not seen on the left side as it was radiographically; however, surgery was performed hours after the radiographs were taken. A right caudal lung lobectomy was performed. Upon removal, a large mucus plug oozed from the cut surface of the bronchus. The right lung lobe and the mucus plug were submitted for histologic evaluation and microbial culture, respectively.

Radiographic findings indicative of pulmonary hyperinflation are caused by air trapping, a condition where air becomes trapped in the lung tissue during expiration. In normal respiration, the diameter of the airway lumen is smaller during expiration than during inspiration. This narrowing can lead to complete airway obstruction in a patient with an existing obstructive lesion (ie, mucus accumulation, bronchoconstriction, or foreign body). Thus, the inspired air is allowed in but cannot escape upon expiration and accumulates in the affected lung. The lung tissue expands and will appear hyperlucent on radiographic images. Other radiographic findings may include increased thoracic cavity size, flattening of the diaphragm, and aerophagia. Air trapping is clinically important because it

alone can cause dyspnea as the result of decreased air flow throughout the affected lung.

Histologic evaluation of the right caudal lung lobe revealed emphysema, ruptured bulla, and multifocal atelectasis. No evidence of neoplasia or pneumonia was found. Results of microbial culture of the mucus plug were negative (cytologic examination was not performed). Although these findings are not

specific, they do suggest a chronic nature to the lung disease. A diagnosis of chronic allergic bronchial disease was considered most likely on the basis of the overall findings. Heartworm disease was also a possibility but considered less likely because cats are not the normal host of *Dirofilaria immitis*. Findings in this cat resembled chronic bronchitis with emphysema, a category of feline asthma.¹ This disease is common in old Siamese cats and is characterized by chronic air trapping, emphysema, and permanent hyperinflation of the lungs. It is believed the cat of this report had end-stage disease, which progressed to emphysema and, eventually, ruptured bullae. Cough may be the only clinical sign observed. Prognosis for full recovery is generally poor, but clinical signs may be relieved by aggressive treatment for asthma (ie, corticosteroids and bronchodilators). If hyperinflation is present, bronchoscopy may be indicated to remove mucus plugs. Early detection is beneficial in treating the disease but can be difficult in cats with mild or no clinical signs. Routine workup may provide hints that underlying disease is present. Affected cats may have an eosinophilia or hyperproteinemia on CBC and serum biochemical analyses, respectively.¹ Examination of a fecal sample and heartworm testing can be performed to rule out parasitic disease and to determine whether further testing is warranted.

The cat in this report did poorly after surgery. Despite aggressive supportive care and ventilation, the cat underwent respiratory arrest within hours after surgery and died. The owner elected not to perform a necropsy examination; thus, the cause of respiratory arrest is speculative. Because of the presence of atelectasis in the remaining lung lobes at surgery, multifocal bronchial obstructions, poor reexpansion of the remaining lobes, or conversely, reexpansion pulmonary edema were considered the most likely contributors to the poor outcome in this cat.

1. Dye JA, Moise NS. Feline bronchial disease. In: Kirk RW, Bonagura JD, eds. *Kirk's current veterinary therapy XI*. Philadelphia: WB Saunders Co, 1992.