What Is Your Diagnosis?

In collaboration with the American College of Veterinary Radiology

Figure 1—Left (A) and right (B) lateral and dorsoventral (C) radiographic views of the thorax of a 13-year-old 3.3-kg spayed female domestic shorthair cat with acute respiratory distress and prior history of coughing, weight loss, and hyporexia.

History

A 13-year-old 3.3-kg spayed female domestic shorthair cat was presented laterally recumbent in acute respiratory distress following 48 hours of anorexia and hiding. The cat was indoor only, part of a multi-cat household, and had a 1-month history of cough, progressive dyspnea, decreased appetite, and weight loss (1.4 kg) over the preceding 3 months. Prior treatment with 1 week of an antimicrobial and 2 weeks of steroids by the referring veterinarian yielded minimal improvement. Specific drugs, dosages, and routes were unspecified. The cat had received a dose of antimicrobials on the morning of admission followed shortly thereafter by acute respiratory distress.

Rachel F. Durrwachter, VMD, and Jennifer A. Reetz, DVM*

Department of Clinical Sciences and Advanced Medicine, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA

*Corresponding author: Dr. Reetz (jreetz@vet.upenn.edu)

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A complete physical examination was not possible due to the cat's respiratory distress. Severely increased inspiratory and expiratory effort with expiratory wheezes and soft crackles in the right dorsal lung field and bradycardia were noted. The specific heart rate was not recorded in the medical record. The patient was placed in an oxygen cage. Terbutaline (0.01 mg/kg, IM), dilute 1:10,000 epinephrine (0.01 mg/kg, SC), and sedation (midazolam [0.4 mg/kg] and methadone [0.2 mg/kg], route not specified) were administered. Findings on oral examination with the cat sedated were clinically normal, and the cat was intubated.

A CBC revealed leukocytosis (21.9×10^3 WBCs/ μ L; reference range, 4.04×10^3 to 18.70×10^3 WBCs/ μ L), with a neutrophilia (19.05×10^3 neutrophils/ μ L; reference range, 2.3×10^3 to 14.0×10^3 neutrophils/ μ L) and monocytosis (1.53×10^3 monocytes/ μ L; reference range, 0.0 to 0.7×10^3 monocytes/ μ L). Serum biochemical analyses revealed hyperglycemia (240 mg/dL; reference range, 67.0 to 168.0 mg/dL), mild hypernatremia (158 mmol/L; reference range, 146 to 157 mmol/L), mild hypochloremia (114 mmol/L; reference range, 116 to 126 mmol/L), and high calculated osmolality (312 mOsm/kg; reference range, 267 to 307 mOsm/kg). A coagulation profile was within reference limits, with a prothrombin time of 13 seconds (reference range, 9.6 to 13.2 seconds) and partial thromboplastin time of 14.9 seconds (reference range, 12.6 to 15.7 seconds), and platelets were estimated as adequate by a board-certified clinical pathologist. The cat tested negative for FIV and FeLV. Thoracic radiography with right and left lateral and dorsoventral views was performed (**Figure 1**).

Formulate differential diagnoses, then continue reading.

Radiographic Findings and Interpretation

There was diffuse soft tissue opacification of the right hemithorax seen on the dorsoventral (DV) view (**Figure 2**). On the lateral views, summation of the ill-defined soft tissue opaque region with the contralateral aerated left lung parenchyma and its well-defined pulmonary vasculature created the mixed soft tissue and air opacity region (approx 8.9 X 10 cm) in the caudal thorax. A component of the soft tissue opacification was due to a severe right cranial, middle, caudal, and accessory lung lobe alveolar pulmonary pattern that effaced the lobar bronchi, pulmonary vessels, right diaphragmatic crus, caudal vena cava, and right lateral margin of the cardiac silhouette. The right lung lobes were collapsed, and there was a severe rightward mediastinal shift and cranial displacement of the right diaphragmatic crus, gastric axis, and gastric antrum (DV view). Compensatory hyperinflation of the left lung lobes with extension of these lobes into the right hemithorax was noted.

On the DV view, to the right of T11, there were several slightly irregular, stippled mineral opaque

foci that could have been within the right caudal lung lobe or could have been part of the chondral portion of the ninth rib. Several irregularly margined 0.7- to 1-cm-diameter gas foci were noted ventral to T10–T11, to the right of midline. The presence of a thin pleural fissure line (lateral views) suggested a small volume of pleural fluid, which likely contributed to the soft tissue opacification of the right hemithorax. On both lateral views, a 1.3-cm soft tissue opaque structure with a cranially convex margin occupied the caudal tracheal lumen with distention of the intrathoracic trachea cranial to the carina (diameter approx 1.3 cm; reference range, 0.5 to 0.7 cm).¹ A radiographically normal air-filled carina was not seen. The left caudal lobar pulmonary artery was equivocally enlarged, and both the left cranial and caudal lobar arteries were larger than the corresponding veins; the pulmonary veins were subjectively small. The cardiac silhouette was radiographically normal in size. In the included abdomen, there was moderate gas distention of the visible intestinal tract, attributed to dyspnea. Finally, scalloping of the visible dorsal paralumbar soft tissues demonstrated the patient's lean body condition with loss of muscle mass.

Figure 2—Same images as in Figure 1. There is diffuse soft tissue opacification of the right hemithorax with complete collapse of the right cranial, middle, caudal, and accessory lung lobes and compensatory hyperinflation of the left lung, with a severe rightward mediastinal shift and effacement of the right lateral aspect of the cardiac silhouette (C). Summation of this ill-defined soft tissue opaque region (general area delineated by short black arrows in A and B) with the aerated left lung creates a mixed soft tissue and air opacity region in the caudal thorax on the lateral views. There are faint pleural fissure lines (short white arrow in A and B), consistent with a scant volume of pleural fluid. Irregular gas foci in the right caudal lung lobe, suggestive of cavitation (white arrowheads in A through C), and possible dystrophic soft tissue mineralization associated with malignancy (long black arrow in C) support the presence of a lung mass. Notice the severely enlarged tracheal diameter (solid white line in A and B) with complete effacement of the carina by an approximately 1.3-cm, cranially convex soft tissue opaque mass occupying the caudal tracheal lumen (black asterisk in A and B). Left lobar arteries (white asterisk in A through C) are larger than corresponding veins. Combined findings were consistent with surgically confirmed right caudal lung lobe mass with extension into the other right lung lobes, lobar bronchi, and caudal trachea.

Based on patient signalment, clinical signs, and the radiographic features of a caudal tracheal luminal or mural soft tissue mass with complete collapse of the right lung lobes, we suspected a cavitated right caudal pulmonary lesion (versus a small volume of pneumothorax), possible mineralization in the right caudal lung lobe, and small volume of pleural fluid. Our primary differential diagnosis was an infiltrative broncho-obstructive cavitary right caudal lung lobe neoplasm, such as primary pulmonary carcinoma or less likely lymphoma, with infiltration of the tracheal lumen, wall, or both. The collapse of the right cranial, middle, and accessory lung lobes was likely secondary to bronchial obstruction with or without multilobar neoplasia. The right caudal lung lobe cavitation was suggestive of necrosis versus less likely, gas-producing bacterial infection or small volume loculated pneumothorax. The small volume of pleural fluid could have been from neoplastic effusion (eg, carcinomatosis), inflammatory effusion, or a small volume of hemorrhage. The possible mineral foci in the right caudal lung lobe could have been dystrophic mineralization, such as associated with malignancy or incidental coalescing bronchial mineralization. Given the patient's geriatric age, clinical presentation, and constellation of radiographic findings, nonneoplastic processes (eg, infectious granulomatous disease, pulmonary abscess, or bronchial obstruction due to inhaled foreign body lodged at the carina) were deemed much less likely but are presented here to demonstrate other processes that might mimic this radiographic appearance, especially in younger patients. Although the effacement of the right diaphragmatic crus by the right hemithoracic soft tissue, along with the cranial displacement of the right diaphragmatic crus and gastric axis, could have suggested chronic diaphragmatic hernia as a differential diagnosis, the severe ipsilateral mediastinal shift concurrent with the other radiographic features (eg, suspected cavitation and tracheal mass) was not fitting with this diagnosis.

The equivocal left pulmonary artery enlargement may have represented shunting of blood to the aerated left lung field, whereas the subjectively small pulmonary veins were likely due to hypovolemic shock.

Treatment and Outcome

After the thoracic radiography, an abbreviated abdominal and thoracic ultrasonographic examination was performed (complete ultrasonographic examination was not performed due to the patient's unstable respiratory status). No clinically important findings were noted on abdominal ultrasonography. Thoracic ultrasonography, focused on the caudal aspect of the right hemithorax, showed scant pleural effusion and right caudal lung lobe consolidation with a clustered region of air-reverberation foci,

which possibly represented the cavitated region seen on radiography.

An exploratory right lateral thoracotomy was performed. A firm 4 X 5-cm mass was identified in the right caudal lung lobe, but extension into the right cranial and middle lobes and associated bronchi was observed (comments regarding the accessory lung lobe were not present in the medical record); subsequently, a right-sided hemipneumonectomy was performed. The right lung lobes en bloc were submitted for histopathologic evaluation, and the diagnosis was pulmonary adenocarcinoma. After worsening clinical progression under mechanical ventilation following thoracotomy, euthanasia was elected the following day. Diagnostic necropsy revealed a firm, tan irregular mass occupying the lumen of and protruding from the trachea and right principal bronchus; grossly and histopathologically, the lesion was consistent with pulmonary carcinoma with severe multifocal necrosis and invasion into the hilar bronchi and tracheal wall and lumen. Moderate chronic multifocal interstitial pneumonia with mild multifocal histiocytosis and multifocal type II pneumocyte proliferation was also noted throughout the lungs, likely due to a chronic systemic inflammatory response associated with neoplasia.

Comments

In cats, primary pulmonary neoplasia (PPN) is uncommon. Respiratory signs are typically not present until late in the disease course, and cats may also have nonspecific signs such as weight loss and hyporexia,² as did the cat of the present report. Common signs in one study² were weight loss (46.5%), lethargy (39.5%), dyspnea (39.5%), anorexia (30.2%), and tachypnea (30.2%). The most common histologic diagnosis of pulmonary neoplasia in cats is pulmonary adenocarcinoma,³ as was the case for the cat of the present report.

Typical radiographic features for cats with PPN include discrete solitary or multiple soft tissue to cavitary pulmonary masses, consolidation of entire lung lobes, tracheobronchial lymph node enlargement, pneumothorax, and pleural effusion.^{2,4,5} Diffuse lung changes, including interstitial, peribronchial, or patchy alveolar infiltrates, are less common.⁶ In this case, morphological intraluminal and mural tracheobronchial invasion and obstruction by primary pulmonary carcinoma were noted. The tracheal invasion was characterized by abrupt termination of the trachea, with the luminal tracheal air abutting a soft tissue opaque structure at the carina. Although bronchial invasion is not unexpected for PPN, tracheal invasion is atypical.^{1,6-8} Right lung lobe airway obstruction was complete and without collateral ventilation (ie, small alternate connections to alveoli that bypass usual the airway conduits were not present),⁶ as illustrated by the presence of complete atelectasis of the right lung lobes and severe ipsilateral

mediastinal shift. Complete right hemithoracic airway obstruction resulted in a lack of visible principal or lobar bronchi (air bronchograms) radiographically. This type of atelectasis is classified as “resorption” atelectasis, in which residual air from the collapsed lobe is resorbed after bronchial obstruction and air can no longer enter the affected lobe.⁶ It should be noted that pulmonary neoplasia can be associated with lobar bronchial obstruction (such as due to luminal invasion) and resorption atelectasis, yet the luminal soft tissue mass component may not be radiographically visible.⁶ The compensatory hyperinflation of the left lung lobes in this case was presumably in response to the right-sided atelectasis; however, tracheal obstruction with air trapping and intact collateral ventilation is also possible.

Differential diagnoses for radiographic findings of discrete or multiple pulmonary masses generally include PPN, metastatic disease, abscess, or granuloma (such as with fungal disease).⁶ Though brief thoracic ultrasonography did not yield a definitive diagnosis, it supported the index of suspicion for a cavitated pulmonary mass, given that the gas noted was intraparenchymal, and confirmed the presence of scant pleural effusion. Although not utilized in this case, CT would have provided an ideal evaluation of combined lesions. In cats with PPN, common findings include visceral pleural contact, bronchial compression, gas cavitation, mineralization, intrapulmonary metastases, and pleural effusion, with less common findings of bronchial invasion and pulmonary vascular thrombosis.^{1,7} Definitive diagnosis in patients with a pulmonary mass may be achieved through an airway wash (eg, bronchoalveolar lavage), evaluation of ultrasonographic or fluoroscopic guided fine-needle aspirate samples, surgical biopsy via lung lobectomy, or necropsy.⁴

Treatment in cats largely consists of surgical resection of affected lung lobes.⁵ Postoperative median survival time for surgically resected PPN is 11 days when there are poor prognostic indicators, including the presence of clinical signs or pleural effusion at time of presentation; lymph node, pleural,

or distant metastasis; and histologically poorly differentiated tumors.⁵

In conclusion, the patient described herein was presented with severe acute clinical signs due to atypical multilobar neoplastic right lung involvement with resorption atelectasis and tracheal invasion. The clinical and radiographic features were negative prognostic indicators associated with a survival time of only 1 day postoperatively. In patients with the aforementioned clinical features, pulmonary neoplasia with regionally invasive behavior should be a prioritized differential diagnosis.

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