



An 82-Year-Old Woman With Progressive Dyspnea and Bilateral Infiltrates

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An 82-year-old woman was admitted to the hospital with progressive dyspnea and hypoxemia. She had a history of mild asthma and had never been hospitalized. One month prior to admission, a new primary care physician recommended that she try montelukast. Two weeks later, she developed a dry nocturnal cough that progressed to dyspnea at rest and orthopnea. She denied fever or chills. There were no other recent changes in her medications.

Physical Examination Findings

The patient's vital signs were as follows: afebrile; pulse, 94 beats/min; BP, 105/59 mm Hg; respiratory rate, 24 breaths/min; and oxygen saturation, 78% on room air and 88% on 3 L/min oxygen by nasal cannula. Her cardiac examination findings were as follows: jugular venous distension estimated at 10 cm and normal heart sounds with no audible murmur or extra sound. Her thorax had diffusely decreased breath sounds. The patient's extremities showed trace pedal edema bilaterally. No rash or clubbing was observed.

Diagnostic Studies

The patient's WBC count was 11,200/ μ L with normal differential (1% eosinophilia); hemoglobin level was 9.7 g/dL and platelet count, 233,000/ μ L. Electrolyte levels and renal and liver function test results were normal. Serology for antinuclear antibodies and antineutrophil cytoplasmic antibodies was normal. Urinalysis was bland. C-reactive protein levels were 168 mg/L on admission (normal, 0–5 mg/L), 70.9 mg/L on day 3, and 23 mg/L on day 8.

The patient was treated with high-dose amoxicillin clavulanate 1 g po tid on days 1 to 7 and prednisone 40 mg/d on days 3 to 7. Her condition did not improve. Transbronchial lung biopsy for histology was requested by the treating medical team. Chest radiographs and CT scan are shown in Figure 1.

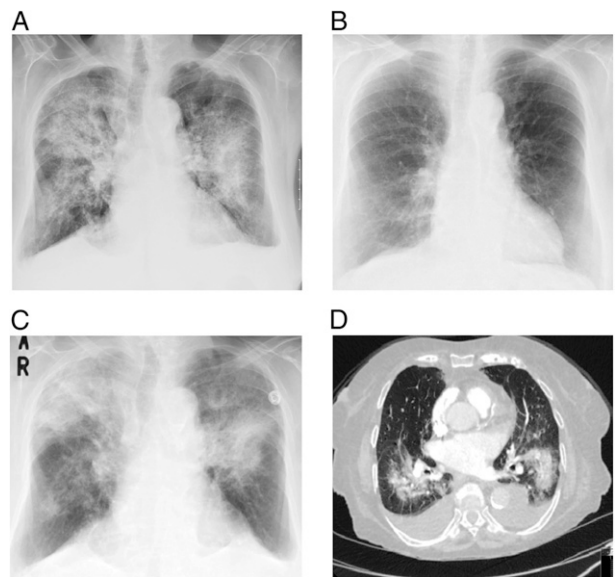


FIGURE 1. A, Posteroanterior (PA) chest radiograph on admission. B, Prior PA chest radiograph (4 years previously). C, PA chest radiograph on hospital day 7. D, Chest CT scan on hospital day 7 with a single-axial section showing bilateral central pulmonary consolidations and bilateral pleural effusions.

What is the diagnosis?

DISCUSSION

In an asthmatic patient with diffuse, nonsegmental areas of consolidation without predilection for any lung zone, the main diagnostic considerations include organizing pneumonia, eosinophilic pneumonia, and Churg-Strauss syndrome (CSS). This radiographic picture, known as bat wing edema, may also represent diffuse alveolar hemorrhage and rapidly developing cardiac failure.

In this case, montelukast therapy raised further concern for CSS, although a causal association is unlikely. The prognosis for untreated CSS or diffuse alveolar hemorrhage associated with autoimmune illness is poor, and immunosuppressive therapy often is initiated without a definitive diagnosis. Furthermore, established diagnostic criteria for CSS often require tissue biopsy, which may cause harm.

In contrast, congestive heart failure (CHF) is a more common diagnosis. In this case, CHF is suggested by the radiophysiologic signs of increased circulating volume (discussed next) and the concomitant presence of bat wing edema. Serial chest radiographs after IV diuresis provided further support for this diagnosis and helped to preclude the need for invasive procedures.

Bat Wing Edema

Bat wing edema describes the central, nongravitational distribution of alveolar opacities with sparing of the peripheral lung. It is seen in < 10% of cases of pulmonary edema and usually is attributed to rapidly developing cardiac or renal failure. Although cardiogenic pulmonary edema is typically symmetric and bilateral, it may be unilateral or predominate in non-dependent lung zones. The most common cause is morphologic changes in the lung parenchyma. Emphysema or fibrosis will direct pulmonary edema to regions less affected by these disease processes. In this case, COPD is suggested by the long history of asthma and hyperinflation seen on the chest radiograph.

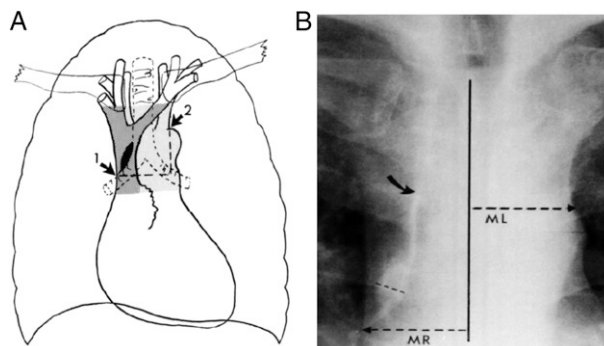


FIGURE 2. A, Measurement of the vascular pedicle. The venous portion is entirely to the right of midline and is shown with darker shading. Measuring points for the vascular pedicle width includes the point at which the superior vena cava crosses the right main bronchus (1) and the point of takeoff of the subclavian artery from the aorta (2). Vascular pedicle width is measured (mm) from point 1 to a perpendicular dropped from point 2. B, Alternative measurement of the vascular pedicle. The right (MR) and left (ML) components of the vascular pedicle are measured (mm) from a midline vertical reference line. The curved arrow points to the paratracheal stripe, which appears to expand inferiorly and enclose the azygos vein. The azygos vein is identified and measured at its greatest short axis (mm). ML = left of midline; MR = right of midline. (Reprinted with permission from Figs 3 and 6 in Milne et al [1984].)

The Vascular Pedicle of the Heart and the Azygos Vein

Dilatation of the neck veins logically accompanies dilatation of the intrathoracic brachiocephalic vessels with which they are immediately continuous. These great vessels are readily accessible for measurement from a standard upright posteroanterior (PA) chest radiograph. Milne and coworkers called this structure the vascular pedicle and proposed two methods of measuring its width (Fig 2). The azygos vein may be seen en face as it arcs over the right-side main bronchus to enter the back of the superior vena cava (Fig 2B).

In the patient presented here, the initial PA chest radiograph demonstrates a significant widening of the vascular pedicle and an increase in the size of the azygos vein width in the presence of bat wing edema (Figs 3, 4, Table 1). The findings are particularly

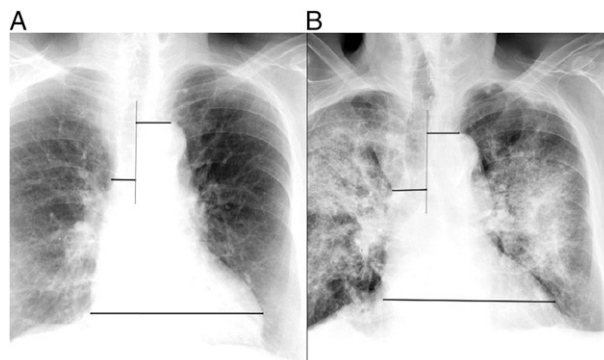


FIGURE 3. A, Milne measurements for the vascular pedicle width (VPW) on prior chest film (4 years previously) was 46 mm. B, Milne measurements for VPW while symptomatic on admission was 52 mm (Table 1).

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