Adolescent With Fever, Hypotension, and Respiratory Distress

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A previously healthy 15-year-old White girl presented to the pediatric clinic with chest pain and difficulty breathing. During the preceding week she had experienced a fever, sore throat, nonproductive cough, significant fatigue, and multiple episodes of nonbloody, nonbilious vomiting. She reported markedly reduced oral intake and decreasing urine output. She appeared fatigued and in severe respiratory distress.

Her medical history was negative for smoking, drug use, or recent invasive dental procedures or orofacial illnesses. Her immunizations were up to date. She had no known drug allergies. A review of systems was negative for headache, diarrhea, rash, or joint pain. She lived with her mother and stepfather and had no known sick contacts.

In the clinic she received a nebulized albuterol treatment, with no improvement in her respiratory status. She was transferred to the pediatric emergency department, where she received a normal saline solution fluid bolus and was treated with intramuscular ceftriaxone. She was then admitted to the pediatric intensive care unit (PICU) because of continued hypotension, respiratory distress, and suspected septic shock.

On arrival in the PICU, she was in moderate respiratory distress. Her vital signs were as follows: temperature, 38.3°C; respiratory rate, 50 breaths per minute; heart rate, 124 beats per minute; and blood pressure, 78/38 mmHg. She had a mean arterial pressure of 63 mmHg and oxygen saturation of 92% on 2 L per minute of oxygen administered by nasal cannula. She appeared jaundiced and had significant edema of the neck, abdomen, and lower extremities. Her head,

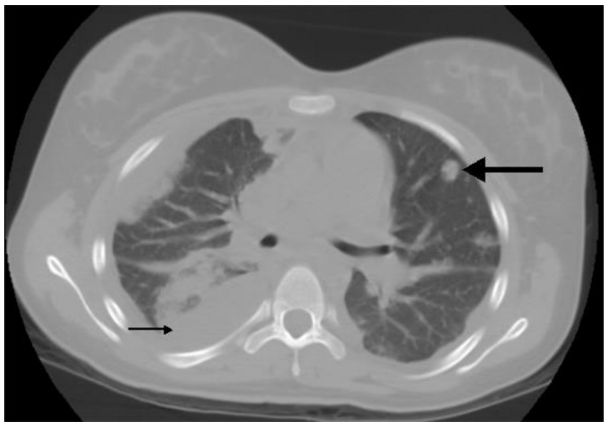
eye, ear, nose, and throat examination revealed a 1-cm, palpable, nontender right anterior cervical lymph node. Chest auscultation revealed crackles in all fields with decreased breath sounds at the bases. Her cardiac rhythm was regular with a mildly hyperdynamic preccordium. Her pulses were weak, with cool extremities and delayed capillary refill. Abdominal examination was significant for a liver edge palpable 3 cm below the right costal margin and a spleen tip palpable 2 cm below the left costal margin. She was electively intubated as a result of her respiratory distress. An infusion of norepinephrine was initiated for inotropic support. Broad-spectrum antibiotics also were initiated to treat the presumed septic state.

Initial laboratory studies revealed a white blood cell count of 12,500/mm^3 with 65% neutrophils, 28% bands, 5% monocytes, and 2% lymphocytes; hemoglobin, 12.1 gm/dL; hematocrit, 34%; and platelet count, 30,000/mm^3. A peripheral blood smear showed few poikilocytes, burr cells, ovalocytes, and anisocytes. The C-reactive protein and erythrocyte sedimentation rate were elevated at 201 mg/dL (normal, 0 to 0.5 mg/dL) and 37 mm/hour (normal, 4 to 20 mm/hour), respectively. Her basic metabolic profile was normal except for slightly decreased serum sodium (130 mEq/L; normal, 133 to 146 mEq/L) and bicarbonate (11 mmol/L; normal, 22 to 26 mmol/L) and elevated creatinine (2.2 mg/dL; normal, 0.5 to 1 mg/dL). Her coagulation profile, fibrinogen, and D-dimer were normal. A hepatic function panel demonstrated low total protein (5.6 g/dL; normal, 6 to 9 g/dL) and albumin (2.3 g/dL; normal, 3.2 to 5 g/dL) levels but elevated conjugated bilirubin (3.1 mg/dL; normal, 0.3 to 1.2 mg/dL), unconjugated bilirubin (0.9 mg/dL; normal, <0.2 mg/dL), and lactate dehydrogenase (529 U/L; normal, 100-190 U/L) levels. Urinalysis was normal except for 1 + protein and 2 + bilirubin. A rapid test for group A streptococcus via a throat swab specimen was negative.

An abdominal ultrasound demonstrated hepatosplenomegaly. A chest radiograph showed bilateral pleural effusions and a 4-cm round density in the left lung close to the mediastinum. A chest computed tomography (CT) scan was obtained to investigate the lung density. The CT scan revealed multiple bilateral noncalcified pulmonary nodules of varying sizes, as well as atelectasis of the lung bases (Figure 1). On the third day of hospitalization, the patient underwent a thoroscopy with a pulmonary nodule biopsy, which demonstrated loculated pleural effusions containing serosanguineous fluid and fibrinous debris. Cultures of the patient's blood, pleural fluid, and lung tissue all demonstrated the presence of *Fusobacterium necrophorum*.

The patient's presumptive diagnosis was Lemierre syndrome, which is also called necrobacillosis or postanginal sepsis. A neck CT scan (Figure 2) revealed partial thrombosis involving the anterior walls of both internal jugular veins (IJVs), confirming the diagnosis

FIGURE 1. Multiple bilateral noncalcified pulmonary nodules (arrows) of varying sizes and atelectasis of the lung bases.



of Lemierre syndrome. The patient's coagulation laboratory panel remained normal. Because the etiology of the thrombosis was thought to be infectious, anticoagulant medications were not administered, as no evidence-based protocols support anticoagulant use in persons with Lemierre syndrome. The antibiotic therapy was changed to intravenous clindamycin and metronidazole pending susceptibility results as per standard recommendations (Kristensen & Prag, 2000; Ramirez et al., 2003; Riordan & Wilson, 2004).

After 1 week in the PICU, the patient became afebrile and was weaned off inotropic medication and the ventilator. Her hepatic enzymes and renal functions normalized, and repeat blood cultures were negative. Based on susceptibility results, the clindamycin was discontinued. A follow-up neck CT obtained 10 days after

FIGURE 2. A neck computed tomography scan shows partial thrombosis involving the anterior walls of both internal jugular veins (arrow), confirming the diagnosis of Lemierre syndrome.