A 14-YEAR-OLD girl had severe, sharp midback pain for 3 weeks and nontender, gradually enlarging left chest wall swelling for 2 weeks. It was unrelieved by change of position. One month before symptoms started, she had fallen, striking her left chest. No bruise remained and the tenderness subsided; however, 2 weeks later, swelling began in the area of injury. She had lost 10 pounds over the last 2 months while dieting. She had no fever, cough, chills, sweats, joint pain, or rash. Last year, she lost a filling from a right upper molar, but otherwise, her last dental care was 6 years ago. She was receiving 325 mg of ferrous sulfate per day for iron deficiency anemia diagnosed 1 month earlier. Findings from physical examination revealed an obese female (height, 139.7 cm; weight, 83.3 kg). Her temperature was 38.5°C; pulse, 120 beats per minute; respirations, 20 breaths per minute; and blood pressure, 122/88 mm Hg. Spinal examination showed scoliosis but no vertebral or costal tenderness. A 4-cm mass was palpated over the left rib cage at the anterior axillary line in the 10th interspace. This mass was ovoid, nontender, soft, and cool to the touch without erythema. A large cavity was present in the upper left molar.

Laboratory results included hemoglobin, 5.9 g/dL; hematocrit, 21.2%; mean corpuscular volume, 67.4 fl; red cell distribution width, 18.6%; reticulocyte count, 2.6%; prothrombin time, 16 seconds; and partial thromboplastin time, 33 seconds. Chest radiograph showed scoliosis of the lower thoracic and upper lumbar spine, cardiomegaly, a retrocardiac consolidation, and left pleural effusion. Fine-needle aspiration of the chest wall mass yielded 20 mL of purulent material; the chest wall abscess was incised and drained. Serum iron was 10 µg/dL, total iron binding capacity, 159 µg/dL; and ferritin, 397.2 ng/mL. Erythrocytes were hypochromic and microcytic, and the neutrophils had toxic granules. Erythrocyte sedimentation rate was greater than 140 mm/h. The rheumatoid factor was positive, and IgG and IgM were increased to 2234 mg/dL and 627 mg/dL, respectively.

Treatment was started with antimicrobials, analgesics for back pain, and vitamin K injections for prolonged prothrombin time. The prothrombin time remained prolonged despite vitamin K administration and infusion of fresh frozen plasma. Anticardiolipin antibody was positive. Routine cultures from the abscess grew no organisms. The tuberculin skin test result was negative, and an anergy panel was positive. Computed tomography (CT) of the chest and abdomen (Figure 1), lateral radiography (Figure 2), and then magnetic resonance imaging scan of the spine were performed. A CT-guided biopsy of the paraspinal mass was performed. Pathologic findings are shown in Figure 3 and Figure 4.
Denouement and Discussion

**Thoracic Actinomyces**

Figure 1. Midabdominal computed tomographic scan demonstrates destruction of the T12 vertebra with adjacent left paravertebral soft tissue mass causing lateral displacement of the left kidney. At the same level, note the large left chest wall mass with an intra-abdominal component.

Figure 2. Lateral spine radiograph shows T12 vertebral body collapse with adjacent irregularities of vertebra T11 and L1.

Figure 3. Medium-power hematoxylin-eosin stain demonstrates fibrous connective tissue with extensive inflammatory reaction.

Figure 4. Gomori methenamine silver stain of actinomyces granule.

Actinomyces are branching, filamentous, gram-positive, microaerophilic anaerobes that are normal commensals of the mouth and tonsillar crypts. Sulfur granules commonly associated with actinomyces are suggestive of the organism. Microscopically, the sulfur granule is a colony of organisms. Staphylococcus aureus, Nocardia species, Actinomadura madurae, and some Streptomyces species may produce colonies resembling sulfur granules (Figure 3 and Figure 4). Diagnosis is made by identifying branching filament colonies. Special staining and anaerobic culture techniques help confirm diagnosis since cultures are positive in only 24% of cases; diagnosis is often based on histopathologic findings.

Actinomyces produce focal granulomatous disease with abscess formation. Actinomyces israelii is the most common pathogen, though other species (Actinomyces meyeri) can cause disease. It advances by contiguous spread and disregards tissue planes. The 3 major forms are cervicofacial (65%), thoracic (15%), and abdominal (20%). Cervicofacial disease is from infection of the teeth and gingiva; abdominal disease may be from perforation of the appendix or cecum. Pulmonary actinomycosis occurs from aspiration of infected material from the oral cavity or carriage into the lung by a foreign body, hematogenous seeding, or spread of cervicofacial or abdominal infection through tissue planes. Thoracic actinomycosis often involves pulmonary, pleural, and chest wall tissue disease. Pericardial involvement occurs in few cases (<2%). Pericarditis with pleural effusion is the most common manifestation.

Possible aspiration of oral secretions may have been the cause of lung infection in our patient, spreading to the pericardium, pleura, paraspinal region, and the T12 vertebra. The paraspinal infection extended to the epidural space through intervertebral foramina. Lateral spread along a track at the costal margin resulted in the subcutaneous abscess. Posttraumatic actinomycosis has been described, but trauma is usually coincidental. Vertebral actinomycosis is a chronic febrile illness, with back pain, weight loss, malaise, and draining sinuses or subcutaneous abscesses. Paravertebral abscesses are adjacent to the involved vertebrae and cause spread to bone. The bacterium invades the transverse processes, body, pedicles, laminae, and spinous processes, yet usually spares the intervertebral disks, distinguishing itself from tuberculosis of the spine. Anemia and thrombocytosis are caused by iron deficiency and chronic inflammation. Antibiotic treatment includes penicillin or clindamycin and tetracycline in case of penicillin allergy. Prompt surgical debridement is necessary in the presence of an abscess. With proper therapy there is a 90% cure rate. Untreated, the mortality rate is 75% to 100%.

Accepted for publication July 12, 2000.

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