to have episodes of cervical pain, but an MRI and CT and bone scans did not show any active infection or signs of osteomyelitis.

The primary etiology of the deep-neck infection in this case remains unknown, which is not unusual [1]. The patient did not have any known predisposing factors for CEA, such as diabetes, iv drug use, recent spinal surgery, trauma, or documented metastatic spread of infection [2]. S. aureus is the most common causative organism in cases of CEA [3].

It has been postulated that osteomyelitis is a necessary prerequisite for CEA, but Lasker and Harter [3] also maintain that infection may spread directly into the veins of the cervical epidural space, thus creating an epidural abscess. Direct extension through the deep fascia and the foramen of the cervical column was the most probable path of the infection in our case, given that the abscess was located near the vertebral column, the epidural abscess developed fairly rapidly, and osteomyelitis was not detected on the preoperative CT scan.

Symptoms of CEA include neck stiffness and cramping, as well as radicular pain involving both the arms and the lower extremities. Weakness or paralysis of the extremities occurs 2 days to several weeks after the onset of the neck pain. Occasionally, bladder dysfunction occurs. Radicular pain and bladder dysfunction were present in our case, but motor weakness did not develop. The case we describe suggests that CEA can be managed conservatively if there is no motor weakness, a view also supported by Leys et al. [4].

**Petri Koivunen, Heikki Löppönen, and Hannu Syrjälä**

Departments of Otolaryngology and Infection Control, Oulu University Hospital, Oulu, Finland

**Retrosternal Abscess: A Prominent Manifestation of Infection Due to Staphylococcus aureus**

*Staphylococcus aureus* is a frequent cause of bacteremia with resulting metastatic infections at sites distant from the primary site of infection; this primary source of infection may be minor or subclinical. Isolation of *S. aureus* from blood cultures should prompt a thorough evaluation to rule out concurrent endocarditis, osteomyelitis, or other infections. We describe a patient with a retrosternal abscess due to *S. aureus* infection.

A 43-year-old man was admitted to the hospital for evaluation of fever, chills, and retrosternal pleuritic chest pain that was relieved when he leaned forward. He reported a history of hypertension.

Physical examination revealed a temperature of 39.5°C, blood pressure of 160/100 mm Hg, pulse of 140/min, and respirations of 24/min. Auscultation disclosed a precordial friction rub, and the patient was admitted to the cardiology department. Laboratory studies revealed the following values: hematocrit, 38%; WBC count, 16 × 109/L (80% neutrophils); and platelet count, 155 × 109/L. Biochemical indices and the results of a urinalysis were normal. An electrocardiogram showed sinoatrial tachycardia. Findings on a chest radiograph were within normal limits, and transthoracic and transesophageal ultrasonography of the heart revealed no abnormalities. The next day the patient complained of vague abdominal pain; he was febrile (temperature, 40°C) and was perspiring profusely. He had mild diffuse abdominal tenderness, but neither rebound tenderness nor guarding was present. Abdominal ultrasonography was negative.

The patient was referred to the internal medicine department. His temperature was 39.8°C; blood pressure, 110/60 mm Hg; pulse, 115/min; and respirations, 20/min. A meticulous physical examination disclosed a small, well-demarcated, healing erysipelas-like lesion on the lower half of the shin, mildly tender to palpation. Cultures of a swab of the lesion were negative. The patient provided a history of a minor trauma that had occurred while gardening 1 week before admission. Leukocytosis persisted. The C-reactive protein level was 252 mg/dL, and serology for rheumatoid factor was negative. Blood cultures (seven samples) were positive for methicillin-susceptible *S. aureus*, and the patient started receiving iv vancomycin, 500 mg q.i.d., and rifampin, 300 mg po b.i.d., because the MIC for semisynthetic anti-staphylococcal penicillins was relatively high. However, the patient’s subclinical discomfort continued. A thoracic CT scan revealed a retrosternal mass lesion, extending from the level of the aortic arch to the level of the ascending aorta, with no bony involvement (figure 1A). CT-guided drainage was unsuccessful; the patient refused surgery. The fever abated after 7 days, and he became afebrile on the 12th day of treatment. A repeated thoracic CT scan showed significant reduction in the size of the mediastinal mass lesion (figure 1B). Rifampin therapy was continued for 15 days overall.

On the 20th day of therapy, the patient was febrile (temperature, 38°C). Laboratory studies revealed the following values: hematocrit, 31%; WBCs, 2.6 × 109/L; and platelets, 394 × 109/L. Evaluation of a peripheral blood smear revealed 1% neutrophils (0.26 × 109/L), 45% lymphocytes, 45% monocytes, 1% basophils, and 2% metamyelocytes. Evaluation of a bone-marrow aspirate revealed a hypercellular marrow and the myeloid-to-erythroid-line ratio was 1.2:1. Blood cultures were negative. Treatment with vancomycin was discontinued, and that with iv dicloxacillin, 2 g q.i.d., was instituted. Three days later the patient was afebrile, and his WBC count was 10.5 × 109/L (78% neutrophils). The anti-staphylococcal therapy was continued for 6 weeks. Seven weeks after dis-
charge, the patient’s hematologic and biochemical indices returned to normal. A repeated thoracic CT scan showed no abnormalities (figure 1C).

We report this interesting case for two reasons. First, this patient developed a retrosternal abscess as the predominant manifestation of S. aureus bacteremia, without any significant findings either at the primary site of infection (i.e., the skin) or involving other organs that are frequent sites of infection in cases of bacteremia due to S. aureus [1–3]. An exclusive, localized staphylococcal infection at this site is very rare. To our knowledge, there is only one report of staphylococcal bacteremia presenting as a retrosternal abscess and endocarditis; this case was in a drug-addict [2]. Second, our patient developed agranulocytosis after the administration of vancomycin, and the condition reversed rapidly after the agent was discontinued.

Among nonhospitalized patients, staphylococcal bacteremia results from blood-stream invasion via carrier sites or minor superficial skin lacerations. In these cases of primary bacteremia, the most common sites of metastatic infection are the lungs, heart, CNS, bones, joints, and muscles [2, 3]. In contrast, our patient developed a retrosternal abscess in the context of staphylococcal bacteremia without any obvious endocardial, thoracic, or mediastinal-structure involvement.

In addition, although the long-term administration of vancomycin has been implicated in cases of leukopenia [4–7], severe granulocytosis due to vancomycin has been reported rarely. Adrouny et al. [8] described a case of severe vancomycin-induced agranulocytosis, postulating a drug-related toxic effect on the bone marrow. Nonetheless, in our case, there were no clinical or laboratory indications of a hypersensitivity reaction (rash or eosinophilia), as in reported cases of mild vancomycin-induced leukopenia [4–7]. Therefore, periodic leukocyte counts should be mandatory during long-term treatment with vancomycin.

Haralampus Milionis, Christos Tatsis, and Moses Elisaf
Department of Internal Medicine, Medical School, University of Ioannina, Ioannina, Greece

References
Recent reports [1–3] focus on the classification of different forms of tracheobronchial aspergillosis (TA); however, the paucity of symptoms associated with this disease and early systematic use of diagnostic procedures such as CT scanning [4] and bronchoscopy should be emphasized in order to avoid the usual fatal outcome. We describe the simultaneous occurrence of two forms of tracheobronchitis in a patient with acute lymphocytic leukemia (ALL).

A 64-year-old diabetic woman was admitted to our facility in Alicante, Spain, for evaluation of sudden worsening of a mild dyspneic condition of 4 months’ duration. Two years before, a diagnosis of ALL had been made, and chemotherapy was prescribed and was followed by complete remission. Two months before admission ALL recurred, again remitting after chemotherapy. The day of admission the patient experienced a sudden worsening of dyspnea while swallowing a pill, and bronchial aspiration was suspected.

Physical examination revealed a patient in severe respiratory distress. Her blood pressure was 100/60 mm Hg, and her temperature was 36.5°C. Auscultation of the chest revealed scattered rhonchi and diminished breath sounds over the left hemithorax. Laboratory studies revealed the following values: WBC count, other, PNBA. Although attempts at classifying TA are clearly of diagnostic capacity than chest radiographs, such as CT scanning [4] and bronchoscopy [5], a prompt clinical suspicion of this unusual disease is unlikely. Pseudomembranous necrotizing bronchial aspergillosis (PNBA) was first described [5] in a patient with AIDS and hemophilia. The most striking feature in this variant of TA was the presence of a well-defined pseudomembrane covering almost the entire bronchial tree. Clarke et al. [1] characterized two morphological variants of fungal tracheobronchitis. The first is the most likely form of serious fungal infection of the airways to be missed clinically and consists of diffuse intraluminal growth with only superficial mucosal invasion, whereas the second variant consists of localized plaques that can penetrate beyond the bronchial wall. Kramer et al. [2] proposed a classification system for invasive TA consisting of three entities, each with distinct clinical and/or pathological features: aspergillosis bronchitis, ulcerative aspergillus bronchitis, and PNBA. Recently, Denning [3] added a fourth entity to this classification system, consisting of obstructing thick mucous bronchial plugs full of Aspergillus hyphae, with little or no evidence of inflammation and without invasion of the bronchial wall.

The patient we describe had bronchoscopic evidence of two of these forms of TA, one with obstructing mucous plugs, and the other, PNBA. Although attempts at classifying TA are clearly of pathogenic concern, we believe that for granulocytopenic patients, an effort should be made to focus, even in the absence of clinical symptoms, on the early systematic use of procedures of higher diagnostic capacity than chest radiographs, such as CT scanning.