A 17-year-old white male was admitted to the hospital with a six-day history of sore throat, three-day history of right neck pain, and temperature of 102°F (38.8°C). Three weeks prior to admission he had had his teeth cleaned and several caries filled. He denied rigors, dyspnea, cough, sputum production or hemoptysis. There was no history of intravenous drug abuse.

The patient was acutely ill. His blood pressure was 100/60 mm Hg; pulse rate, 110 beats per minute; and oral temperature, 102°F (38.8°C). Examination of his head and neck showed mild right tonsillar hypertrophy without exudate, a firm right submandibular lymph node, and mild diffuse tenderness along the right sternocleidomastoid muscle. The lungs were clear to auscultation. A grade 1/6 systolic murmur was heard at the cardiac apex.

Laboratory findings were: hematocrit, 41.6 percent; white blood cell count, 21,700/cu mm with 43 percent neutrophils, 32 percent bands, 15 percent lymphocytes, and 10 percent monocytes. Chest roentgenogram showed bilateral scattered ill-defined densities less than 3 cm in diameter. PA tomograms demonstrated noncavitated nodular densities (Fig 1). Results of soft tissue roentgenograms and an ultrasound examination of the neck were unremarkable. Intravenous nafcillin sodium at 1 g every four hours was administered. Chest roentgenogram obtained four days later showed cavitation of the previously seen nodules with no air-fluid levels (Fig 2).
Diagnosis: Septic pulmonary emboli secondary to parapharyngeal abscess (postanginal sepsis)

The right neck became edematous and fluctuant. On the fifth hospital day an abscess in the parapharyngeal space was surgically drained under general anesthesia. Cultures grew Bacteroides melanogenicus and Peptostreptococcus. Blood cultures also grew Bacteroides melanogenicus. The nafcillin sodium was discontinued and intravenous clindamycin at 600 mg every eight hours was started. The patient had an uneventful and complete recovery.

Septic pulmonary emboli may originate from infected thrombi anywhere in the venous system or cardiac chambers, or may be related to other systemic infections. Common sites from which septic embolization can occur are: the arm and leg veins (particularly in patients who have a history of intravenous drug abuse, or have long-term indwelling peripheral or central venous catheters), the skin and subcutaneous tissues, the pelvis and abdomen (particularly in the postoperative period), and the right-sided cardiac valves. Less common sites from which septic embolization can occur are: the middle ear, brain, liver, kidney, bone, arteriovenous fistulas (used for hemodialysis), left-sided cardiac valves (through left-to-right cardiac shunts), and the internal jugular vein and its tributaries. Septic emboli to the lung may also be seen in systemic illnesses such as: rickettsial diseases, plague, and certain fungal and parasitic infections. Jaffe and Koschmann reviewed the roentgenographic manifestations of septic pulmonary emboli. The lesions usually appear as small ill-defined, round or wedge-shaped densities in the peripheral lung fields. Septic pulmonary emboli are almost always multiple and may be the same size or vary in size. They may appear to migrate. Cavitation frequently occurs and may be rapid. The resulting cavities are characteristically thin-walled without clearly evident air-fluid levels. Large lung abscesses may be seen from the coalescence of several smaller abscesses or infarcts. Pleural effusions (empyema) or pneumothorax (bronchopleural fistula) may also be seen. With appropriate therapy, the lesions may completely resolve or leave areas of pleural scarring or pulmonary fibrosis.

Postanginal sepsis is the name given to sepsis originating in the internal jugular venous system usually from a parapharyngeal abscess following an oropharyngeal infection. This condition was well described by Abt in 1932, Hall in 1939, and Boharas in 1943. The source of this condition is pharyngitis or tonsillitis, but it may occur following odontogenic infections, or infections of the nose, sinuses, ear, jaw or cervical vertebrae. Usually, the infection extends from the throat into the retro- or parapharyngeal space through venous or lymphatic drainage, or through the pharyngeal muscular wall. Septic thrombophlebitis of the internal jugular venous system ensues from which septic emboli originate. Organisms most commonly isolated from oropharyngeal infections include aerobes and anaerobes: Streptococcus, Peptostreptococcus and Bacteroides. The onset of the illness is insidious. Often the sore throat is mild and easily overlooked, and the neck examination initially may be unimpressive. Symptoms of fever and rigors, heralding the onset of sepsis, may occur shortly after the primary infection, but may be delayed for as long as four weeks. The most common and serious complications are pulmonary abscess formation and pulmonary infarction. Other complications include: mediastinitis due to extension of infection along fascial planes into the mediastinum, hemorrhage from erosion into the internal or external carotid arteries, paralysis of the hypoglossal or recurrent laryngeal nerves from abscess formation in the parapharyngeal space, and metastatic abscesses of the brain, liver, kidneys, bones, joints and muscles. Venography before therapy can confirm thrombophlebitis in the internal jugular venous system, but has at least the theoretic danger of vessel perforation by the contrast-instilling catheter. Treatment of postanginal sepsis involves surgical drainage of the parapharyngeal infection and appropriate antibiotic therapy such as penicillin, clindamycin or metronidazole.

The modern antibiotic era has not completely invalidated Hall's warning in 1939:

Be not deceived by a comparatively innocent appearing pharynx, as the veins of this pharynx may be carrying the death sentence for your patient.

References

7. Mitre RJ, Rotheram EB. Anaerobic septicemia from thrombophlebitis of the internal jugular vein, successful treatment with metronidazole. JAMA 1974; 230:1188-9