CHEST™

Streptococcus anginosus Infections
Crossing Tissue Planes

Bernie Y. Sunwoo, MBBS; and Wallace T. Miller Jr, MD

Streptococcus anginosus has long been recognized to cause invasive pyogenic infections. This holds true for thoracic infections where *S anginosus* has a propensity for abscess and empyema formation. Early diagnosis is important given the significant morbidity and mortality associated with thoracic *S anginosus* infections. Yet, distinguishing thoracic *S anginosus* clinically is difficult. We present three cases of thoracic *S anginosus* that demonstrated radiographic extension across tissue planes, including the interlobar fissure, diaphragm, and chest wall. Few infectious etiologies are known to cross tissue planes. Accordingly, we propose *S anginosus* be considered among the differential diagnosis of potential infectious etiologies causing radiographic extension across tissue planes. CHEST 2014; 146(4):e121–e125

ABBREVIATIONS: CXR = chest radiograph; MIC = minimum inhibitory concentration

The *Streptococcus anginosus* group, consisting of three distinct species, *Streptococcus intermedius*, *Streptococcus constellatus*, and *Streptococcus anginosus*, has long been recognized to cause invasive pyogenic infections. This holds true for thoracic infections where thoracic *S anginosus* has a propensity for abscess and empyema formation. Cases of mediastinitis have been reported.[1-3] Early diagnosis and a high clinical suspicion are essential given the significant morbidity and mortality associated with thoracic *S anginosus* infections.[4]

Few pulmonary infections are known to cross pleural surfaces, leading to chest wall, mediastinal, or abdominal invasion. Infections reported to cause these problems include actinomycosis, nocardiosis, blastomycosis, TB, and mucormycosis.[5-11] As such, an imaging demonstration of extension of infection across pleural boundaries into the adjacent tissues is of use in suspecting these unusual infections as a cause of the patient's clinical illness.

We present three cases of thoracic *S anginosus* that demonstrated radiographic extension across tissue planes. We propose *S anginosus* be considered among the differential diagnosis of potential infectious etiologies causing extension across tissue planes.

Case Reports

The study was approved by the center's institutional review board (IRB#7,

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AFFILIATIONS: From the Department of Medicine, Division of Pulmonary, Allergy, Critical Care Medicine, Division of Sleep Medicine (Dr Sunwoo) and Department of Radiology (Dr Miller), Perelman School of Medicine, The University of Pennsylvania, Philadelphia, PA.

CORRESPONDENCE TO: Bernie Y. Sunwoo, MBBS, Department of Medicine, Division of Pulmonary, Allergy, Critical Care Medicine, Division of Sleep Medicine, Perelman School of Medicine, Perelman Center for Advanced Medicine, University of Pennsylvania, 1st Floor, W Pavilion, 3400 Civic Center Blvd, Philadelphia, PA 19104; e-mail: bernie.sunwoo@uphs.upenn.edu

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Protocol 813882), and informed consent was waived due to the retrospective nature of the project.

Case 1

A 62-year-old white man with a history of bipolar disorder was hospitalized to the psychiatric unit for mania. A chest radiograph (CXR) was performed on admission for cough and right-sided chest pain that revealed right middle lobe opacity. He was treated with levofloxacin and clindamycin for 1 week but had continued chest pain with progressive productive cough, low-grade fevers, and weight loss. He was smoking one pack of cigarettes daily but denied other illicit drug or alcohol use. He had no pets and denied recent travel or occupational exposures. On physical examination, he was afebrile, had poor oral dentition with no cervical lymphadenopathy, globally reduced breath sounds, and crackles in the right lower zone. A chest CT scan was performed for continued chest pain following a second course of levofloxacin; it showed mass-like consolidation in the peripheral right middle lobe extending across the oblique fissure into the adjacent anterior right lower lobe (Fig 1). Sputum cultures showed moderate mouth flora. Bronchoscopy was performed, revealing copious purulent secretions emanating from the right middle lobe with no endobronchial lesions seen. BAL and transbronchial biopsies showed many polymorphonuclear cells and *S anginosus* susceptible to penicillin (minimum inhibitory concentration [MIC], 0.094). Cultures were negative for anaerobes, mycobacteria, and fungi, and no evidence of malignancy was seen. Blood cultures remained negative. HIV testing was negative. His course was complicated by empyema requiring multiple chest tubes. He was treated with IV ampicillin/sulbactam for 2 weeks followed by oral amoxicillin/clavulanate for an additional 4 weeks with clinical and radiographic improvement.

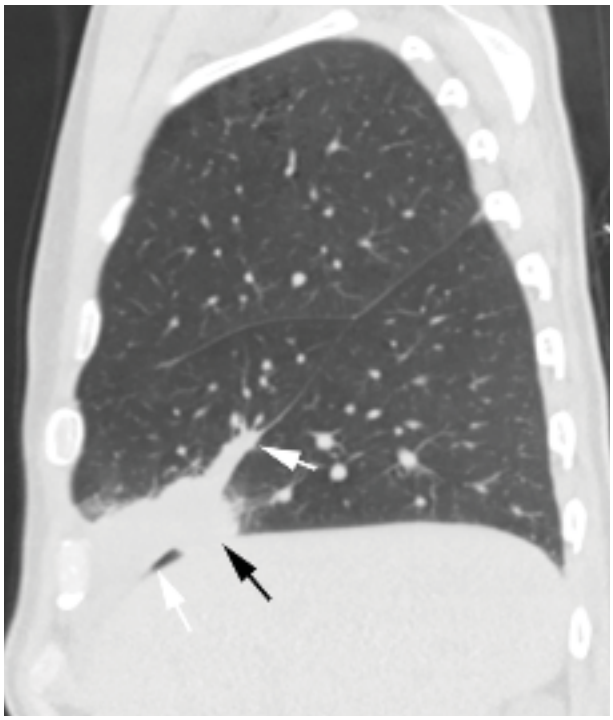

Figure 1 – *Invasion across fissures. Sagittal reconstruction from an unenhanced CT scan demonstrates a focal area of consolidation (black arrow) in the right middle lobe that has invaded across the major fissure (white arrows) into the right lower lobe.*

Case 2

A patient in his 20s with Crohn's disease (status: postileostomy) on no medications presented with 3 weeks of left-sided pleuritic chest pain and dyspnea. He was smoking one pack of cigarettes per week but denied illicit drug or alcohol use. He had no pets and denied recent travel or occupational exposures. On physical examination, he was afebrile but diaphoretic, tachycardic, tachypneic, with BP 113/58 mm Hg without pulsus paradoxus. The jugular venous pressure was elevated with distant heart sounds. The chest was clear. He had diffuse abdominal tenderness maximal over the right upper quadrant with no peritonism, and his ileostomy site was intact. ECG showed diffuse ST elevation and PR depression. Laboratory data were significant for the following: WBC count 31.7 with 19% bands, hemoglobin 8.2 g/dL, platelets 734, acute renal failure, negative cardiac enzymes, total bilirubin 4.1 mg/dL, alkaline phosphastase 229 units/L, aspartate aminotransferase 59 units/L, alanine aminotransferase 84 units/L, and lactic acidosis 3.3 mg/dL. CXR revealed massive cardiomegaly but clear lung fields. A transthoracic echocardiogram was performed showing a moderate to large pericardial effusion with evidence of increased intrapericardial pressure that did not meet criteria for tamponade, ejection fraction of 50%, and no vegetations. Pericardiocentesis was attempted but was unsuccessful with an apparent loculated effusion. Right-sided heart catheterization revealed no tamponade physiology. An abdominal CT scan was obtained, showing new hypodense areas occupying the entire left lobe and portions of the right lobe of the liver and thickening of the gallbladder wall with extension of the abscess through the diaphragm into the adjacent pericardial space (Fig 2). The patient was initiated on broad-spectrum antibiotics and taken to the operating room for a pericardial window and drainage of his liver. Foul-smelling,