# Lung Abscess Due to *Streptococcus mitis*: Case Report and Review

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***Streptococcus mitis*** **is a bacterium traditionally regarded as a normal commensal of the oropharynx, skin, and intestinal and genital tracts. To our knowledge, we describe the first case of bilateral lung abscesses caused by *S. mitis* in an immunocompetent host. The abscesses were successfully treated with clindamycin and gentamicin. Our case illustrates that *S. mitis* should be considered a cause of pulmonary abscesses.**

*Streptococcus mitis* [1] is a species of oral streptococci that has neuraminidase activity [2], does not form acid in the presence of mannitol, inulin, or raffinose broth, and produces extracellular dextran [3–5]. *S. mitis* is part of the viridans group streptococci [3, 4]; it is considered either as a separate strain [3] or (in combination with *Streptococcus sanguis* II) as *Streptococcus mitior* [2]. As other investigators have emphasized, *S. mitis* can be an important pathogen in children [6] and adults [5], even though it is more commonly a contaminant of culture media [5, 7, 8]. Although viridans streptococci have been associated with different pulmonary infections (e.g., pneumonia, empyema, and lung abscess [mainly due to *Streptococcus milleri*]), to our knowledge, cases of lung abscess caused by *S. mitis* have not been reported. We report herein the case of a patient with bilateral lung abscesses due to *S. mitis* and review the world literature. We searched the literature from 1966 to 1993 with MEDLINE by cross-referencing the terms *lung abscess*, *S. mitis*, *S. mitior*, and viridans *Streptococcus*.

## Case Report

A 48-year-old man was admitted to the hospital with a 15-day history of malaise, cough, scanty blood-streaked sputum, and left pleuritic pain. He was a smoker and had chronic bronchitis. He did not receive any treatment. He did not have a history of alcohol consumption, loss of consciousness, neoplastic diseases, or surgical or invasive procedures, and he did not have risk factors for human immunodeficiency virus (HIV) infection. His temperature was 38.8°C, and his blood pressure was 160/95 mm Hg. Oral examination disclosed multiple dental caries and signs of periodontal disease. Physical examination was unremarkable; no cardiac murmurs or peripheral stigmata of infective endocarditis were noted.

The white blood cell count was $20.7 \times 10^9$/L (82% neutrophils, 16% lymphocytes, 1.5% monocytes, and 0.5% eosinophils), the hemoglobin level was 10.7 g/dL, and the erythrocyte sedimentation rate was 80 mm/h. The $PaO_2$ when the patient was breathing room air was 66.5 mm Hg. The patient's tuberculin test was negative, he did not have antibodies to HIV, and his serum immunoglobulin levels and $CD^4$ T lymphocyte counts were normal. Three separate sets of blood cultures were negative. A gram stain of the sputum sample was not done. Culture of the sputum sample on aerobic medium yielded only colonies of gram-positive cocci, which were later characterized as viridans streptococci.

Findings on a chest roentgenogram (figure 1) revealed two cavitary lesions and air-fluid levels, one in the right upper lobe and another in the left lower lobe. In addition, a parenchymal infiltrate in the right middle lobe later became cavitated.

Examination of purulent non-foul-smelling secretions obtained by percutaneous lung aspiration of the left lower lobe abscess showed scanty polymorphonuclear leukocytes and chains of gram-positive cocci; pure growth of these cocci was later noted on both aerobic and anaerobic culture media. These colonies were subsequently characterized as viridans streptococci. The viridans streptococci isolated from the sputum and the secretions were biochemically identified as *S. mitis* by exclusion after the appropriate tests were performed (these $\alpha$-hemolytic strains form acid in the presence of lactose but not in the presence of mannitol; did not produce sorbitol, acetylmethylcarbinol, arginine, and urease; and did not hydrolyze esculin). No mycobacteria or fungi were isolated from the samples, and protozoa were not seen. Malignant cells were not noted. Therapy with gentamicin (120 mg iv twice a day for the first 2 weeks) and clindamycin (600 mg iv four times a day for the first 2 weeks and then 300 mg po four times a day for the next 6 weeks) was given (such antimicrobial therapy was empirically started after percutaneous lung aspiration was done and sputum for culture was obtained but before the results were received). The MICs of

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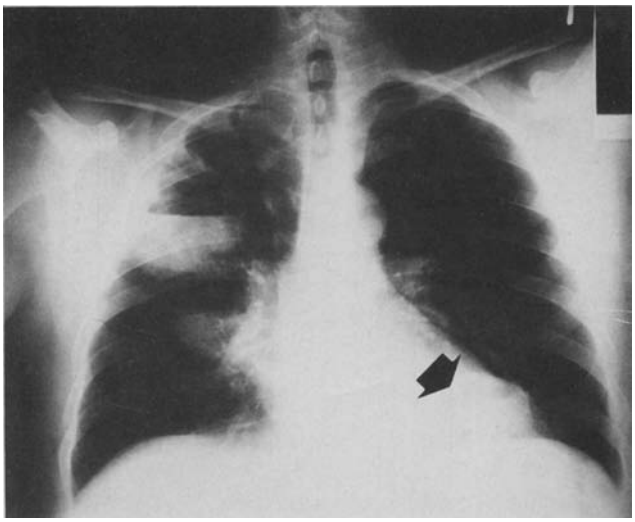

**Figure 1.** Chest roentgenogram demonstrating two cavitary lesions and air-fluid levels, one in the right upper lobe and another in the left lower lobe (*black arrow*). Note the parenchymal infiltrate in the right middle lobe.

gentamicin, clindamycin, and penicillin were ≤1 μg/mL, 2 μg/mL, and 2 μg/mL, respectively. At a follow-up visit 3 months after therapy was started, the patient was asymptomatic, a chest roentgenogram showed only residual interstitial changes, and the erythrocyte sedimentation rate was 8 mm/h.

## Discussion

*S. mitis* is a normal inhabitant of the human mouth, pharynx, bowel, vagina, and skin [5, 9, 10]. Since the original description of *S. mitis* in the early 1900s [9, 11], the spectrum of clinical manifestations associated with this organism has considerably enlarged. However, the true scope of the pathogenic importance of *S. mitis* is not yet well defined because many reports on viridans streptococci do not contain enough information for identifying these microorganisms to the species level.

*S. mitis* has been associated with bacteremia [12], infections of the digestive [5, 13] and cardiovascular systems [5, 9, 14], meningitis [14, 15], periodontal disease [16], and other infections [5, 10, 12, 17–20] (table 1). Moreover, pleural empyema, tracheoesophageal fistula with mediastinitis, and pneumonia [5, 21, 22] caused by *S. mitis* have also been reported. Although several investigators have reported cases of lung abscess due to *Streptococcus* species, including viridans group streptococci [23–30], in our review of the literature we did not find any cases of lung abscess due to *S. mitis*. There are several explanations for the fact that respiratory infections caused by *S. mitis* and other oral streptococci are not recognized more frequently. First, the same antimicrobial agents that have activity against bacteria causing other pulmonary infections have activity against these streptococcal strains. Second, there is nothing distinctive about the clinical presentations of infections due to these organisms that suggests the etiologic pathogen [23]. Third, the significance of these organisms may easily be undervalued in sputum since they are usually part of the normal flora of the mouth. Finally, the taxonomy and nomenclature of streptococci are not reliable and consistent.

Bacteremia (the mouth being the main portal of entry [14, 21]) and aspiration of oral secretions [5] or esophageal rupture [5] may be risk factors for respiratory infections secondary to *S. mitis* infection. In our case periodontal disease was believed to be the predisposing factor for the lung abscesses.

Although the incidence and severity of *S. mitis* infections may be increased among neonates [31], children with cancer [6], intravenous drug users [14], and adults with neoplastic diseases [12, 23], the case reported herein underscores the fact that serious pulmonary infection can also develop in healthy hosts. On the other hand, production of dextran, which is detected in 13%–25% of *S. mitis* isolates, has been recognized as a virulence factor of special significance [5, 10, 32]. This characteristic could aid in elucidating the reasons for severity of some clinical infections. We assume that this microbiological feature played a role in the present case since our patient had three lung abscesses in the absence of other underlying factors. Unfortunately, it was not determined

**Table 1.** Summary of data on *S. mitis* infections reported in the world literature since 1966.

| Type of infection(s) | [Reference(s)] |
|---|---|
| Respiratory and mediastinal infections | |
| Pneumonia | [21, 22] |
| Pleural empyema | [5] |
| Mediastinitis | [5] |
| ARDS-like syndrome* | [12] |
| Bacteremia and cardiovascular system infection | |
| Bacteremia (with or without sepsis) | [5, 12, 14] |
| Endocarditis | [9, 14] |
| Pericarditis | [5] |
| Septic thrombophlebitis | [5] |
| Miscellaneous infection | |
| Eye infection (keratitis, conjunctivitis) | [17, 18] |
| Otitis media | [20] |
| Periodontal infection | [16] |
| Meningitis | [14, 15] |
| Genital tract infection | [10] |
| Peritonitis | [5, 13] |
| Osteomyelitis and bone prosthesis infection | [5] |
| Soft-tissue infection (CHIF, FP†) | [5, 19] |

NOTE. ARDS = adult respiratory distress syndrome; CHIF = close to hip infected fracture; FP = forearm phlegmon.

* With eventual development of encephalopathy and palmar desquamation.

† *S. mitis* was isolated in association with other microorganisms.

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whether the *S. mitis* isolate from the patient described herein produced dextran.

Antimicrobial agents are considered the primary mode of therapy for lung abscess, and surgical resection or drainage is rarely required [26, 30]; therefore, most strains of *S. mitis* remain sensitive to penicillin [4, 5, 14]. Other antibiotics with less activity against *S. mitis* include erythromycin, clindamycin, cephalosporins, and vancomycin [4]. However, reports have been published that have demonstrated resistance of *S. mitis* to penicillin [12]; these reports have also showed that higher concentrations of antibiotics were needed for inhibiting and killing *S. mitis* than for inhibiting and killing other viridans streptococci [33].

In summary, clinicians should be aware that *S. mitis*, although often a contaminant, can be associated with severe disease in both immunocompromised and immunocompetent patients. Our case of bilateral lung abscesses caused by *S. mitis* serves to illustrate that the spectrum of infections due to this microorganism is growing.

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