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Lung abscess caused by *Streptococcus intermedius* in an immunocompetent patient

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Abstract
Lung abscesses are most commonly polymicrobial, being caused by both anaerobic and aerobic bacteria, usually from the oral flora. A particular pathogen present in the oral flora, *Streptococcus intermedius*, has been known to cause aggressive pyogenic infections such as abscesses, most often on the soft tissues, liver and brain. Though less common, these infections can also occur in the lungs of immunocompetent individuals without preceding risk factors. In such cases, a presentation with productive cough and fever can be misdiagnosed as tracheobronchitis or pneumonia. We present the case of an immunocompetent patient without significant underlying risk factors, who was initially misdiagnosed as recurrent sinusitis, that was found to have a lung abscess due to *S. intermedius* infection.

Keywords: Lung abscess, *Streptococcus intermedius*, immunocompetent, pulmonary infection

Introduction
Lung abscesses are microbial infections of the lung parenchyma characterized by a localized area of liquefactive necrosis and formation of cavities measuring more than 2cm in diameter which can contain purulent fluid and necrotic debris [1, 2]. Despite being commonly encountered in clinical practice they are usually diagnosed late in the disease course given their indolent presentation [3, 4]. Patients typically present with features that mimic pneumonia or upper airway infections, these may include fatigue, cough, shortness of breath, fever, chills, among others. Such nonspecific presentation often leads to misdiagnosis, especially when advanced imaging studies are delayed [5]. In regards to their etiology, lung abscesses are often a result of polymicrobial infection, with anaerobes being among the most common causative organisms. These infections are usually a consequence of aspiration particularly in patients with predisposing factors such as seizure disorder, alcohol use, and poor dentition. *Streptococcus intermedius* is a highly pyogenic organism found within the oral flora, and it can be involved in abscesses on different organs such as liver and brain [6, 7, 8]. However, lung infections with *S. intermedius* typically are not common in immunocompetent patients, with only a few cases reported to date [9, 10]. We describe a patient who had initially presented to our emergency department with complaints of recurrent upper airway disease. It was later discovered that this patient was harboring a large lung abscess as a result of *S. intermedius*, which required percutaneous drainage and prolonged antibiotic therapy for resolution.

Case Report
A 48-year-old male presented to our hospital with a chief complaint of cough with productive yellow sputum, intermittent hemoptysis, postnasal drip, decreased appetite, and a 20-pound weight loss occurring over the past 3 months. One week prior to admission, he visited an urgent care center and was prescribed a regimen of oral doxycycline for sinusitis, however despite treatment he had no significant improvement. Given the persistence of his symptoms he returned to the urgent care center. This time a chest X-Ray was done (Figures 1A and 1B) and revealed a very large opacity posterior to the heart. He was referred to our hospital's emergency room for further evaluation. His physical exam findings were benign with the exception of poor oral dentition and an absence of breath sounds from the left lung base up to the level of the scapular spine on auscultation; however, no lymphadenopathy, testicular mass or other abnormal breath sounds were noted. Vital signs revealed blood

pressure, 140/76 mmHg, pulse rate, 71 bpm, respiratory rate, 18 breaths per minute, body temperature, 98.8 Fahrenheit, body mass index of 30.1 m^2/kg, pulse oximetry, 99% on room air. Laboratory testing revealed the following abnormalities: leukocytosis of 20,700 per µl with 74% of white blood cells represented by neutrophils, hemoglobin of 9.4 g/dL with an MCV of 85 fL, platelet count of 527,000 per µl, lactic acid of 0.9 mmol/L, albumin 3.0 g/dL with a normal albumin/globulin ratio of 0.7 and a procalcitonin level of 0.27 ng/mL. A CT scan of the chest with IV contrast obtained after hospitalization (Figures 2A, 2B and 2C) revealed an oval and very well circumscribed cystic lesion measuring 14.1 x 11.5 x 13.2 cm within the left lung parenchyma with minimal pleural effusion present at the base, differential diagnosis for this new found lesion at the time included a round pneumonia with central necrosis, a substantially large left lung abscess, or malignancy with a central area of necrosis. Of note, this was in general a healthy gentleman - his past medical history was significant only for right sided testicular cancer with metastasis to the lumbar spine which was successfully treated 19 years prior to admission and had since remained in remission; he was otherwise a healthy, nonsmoker, middle aged male with no previous episodes of pneumonia or significant alcohol consumption. Given his clinical presentation and CT findings he was subsequently hospitalized. Respiratory sputum samples were appropriately obtained for Gram stain and culture, and an antibiotic regimen of IV Ceftriaxone and IV Metronidazole was initiated. Although he denied fevers prior to presentation, he did experience febrile episodes which occurred only during the first 3 days of his hospitalization with maximum temperatures peaking at 101.6 Fahrenheit. Pulmonary service was consulted early during his hospital course and decided to obtain a CT-guided biopsy along with needle aspiration of the cystic mass on day 2 of his hospitalization; however, after an initial 40 mL of free-flowing fluid revealed a frankly purulent appearance without putrid odor, a percutaneous pigtail catheter connected to a drainage system was placed and appropriate samples were obtained for microbiologic and cytologic analysis. Chest tube drainage equaled a total of 815 mL of purulent fluid over the next 3 days, however drainage on the 4th and 5th days post chest tube placement was consistently less than 50 mL and appeared to be light yellow to clear resembling pleural fluid, at which time the decision to extract the chest tube was made and was successfully completed 5 days after placement. Daily chest X-Rays and a chest CT with IV contrast obtained one day prior to chest tube removal were done for close monitoring; X-Rays (Figures 3A and 3B) revealed progressive resolution of the cystic lesion with repeat chest CT (Figures 4A, 4B and 4C) revealing a 5.7 x 5.3 x 0.6 cm cavity mostly filled with air along with a persistent but small pleural effusion on the same side and a newly revealed airspace opacity that involved the majority of the left lower lobe. Culture of the aspirated purulent fluid revealed *Streptococcus intermedius* which was resistant to clindamycin and erythromycin. Initial respiratory sputum cultures were also positive for Methicillin-sensitive Staphylococcus aureus, however given the patient's presentation this was considered most likely a contaminant. He agreed to be tested for the presence of HIV, with negative results. During hospitalization he received IV Ceftriaxone and IV Metronidazole for a total of 6 days and was subsequently discharged on an antibiotic regimen consisting of Amoxicillin with Clavulanic Acid. Prior to discharge, his symptoms had significantly improved and he had been afebrile since the third day of hospitalization. He was instructed to follow up with a pulmonologist within 1 week of hospital discharge and was scheduled to undergo a repeat CT scan of the chest 3 weeks after discharge for further assessment of the underlying pneumonia and residual left lower lobe cavity.

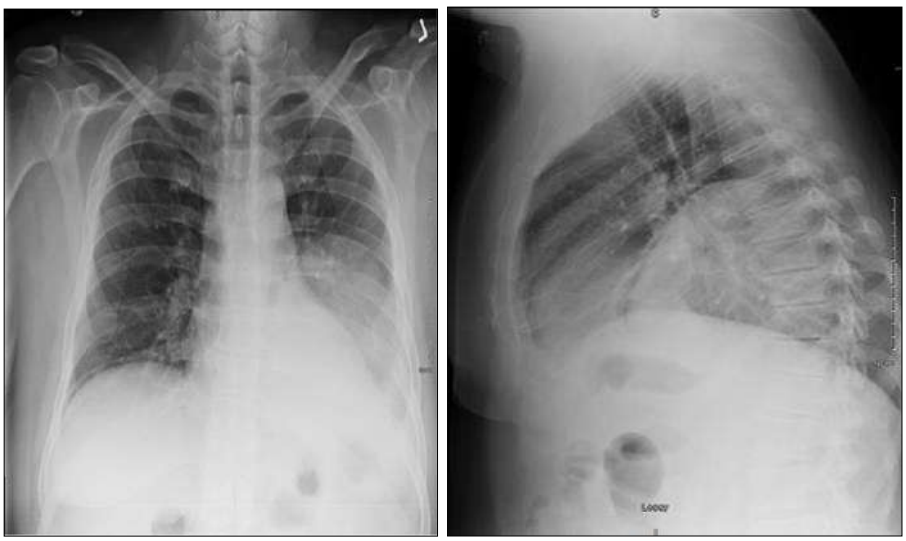

Fig 1: A and 1B: Admission chest X ray (PA and lateral views) - Opacity is seen posterior to the heart occupying the entire lower half of the left chest.

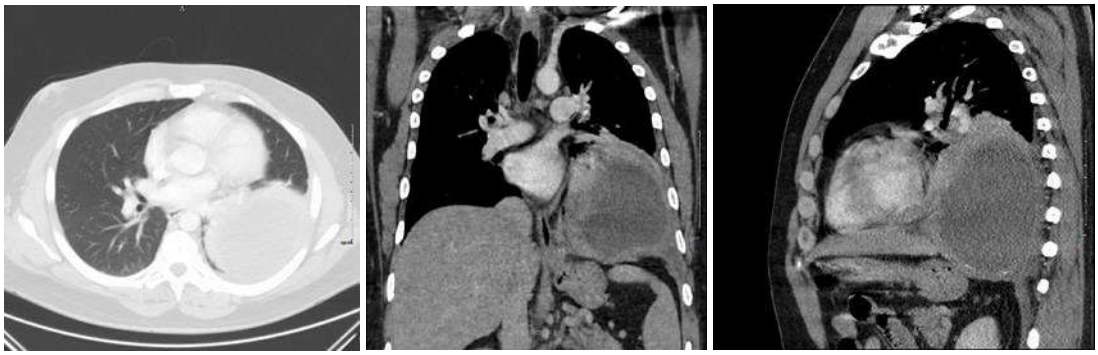

Fig 2: A 2B and 2C: Admission chest CT transverse, coronal and sagittal views – Note the oval and very well circumscribed cystic lesion measuring 14.1 x 11.5 x 13.2 cm within the left lung parenchyma with minimal pleural effusion present at the base.

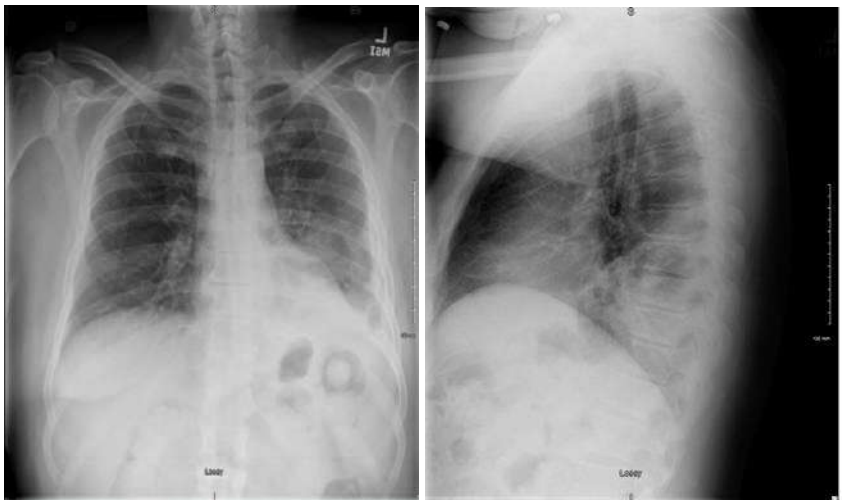

Fig 3A and 3B: Chest X ray (PA and lateral views) - Minimal residual left lower lung pneumonitis.

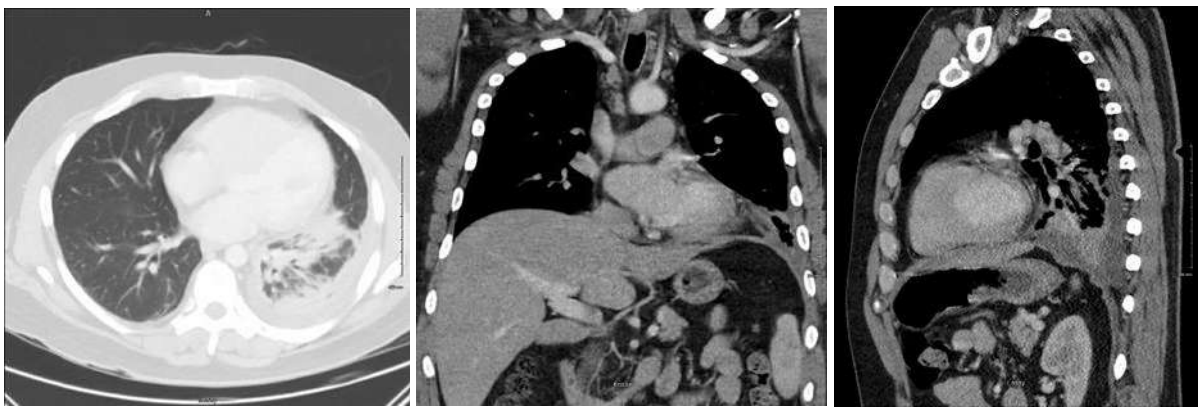

Fig 4A, 4B and 4C: Chest CT done after drainage - transverse, coronal and sagittal views - Interval near complete drainage of the cavitary lesion in the left lung base. Small residual thin curvilinear cavity remains. Airspace opacity involving the majority of the left lower lobe.

Discussion

The incidence and mortality from lung abscesses have reduced significantly over the past 50 years. The advent of antibiotics and advanced drainage techniques has greatly impacted the management, complications, and prognosis of lung abscesses. Despite this, the overall mortality rate remains variable between 2% to 38% [11]. Lung abscesses can be classified by time duration into acute (< 6 weeks) or chronic (>6 weeks), and the vast majority are caused by polymicrobial bacterial infections, encompassing both anaerobic and aerobic bacteria. This includes, *Bacteroides fragilis, Fusobacterium capsulatum, Streptococcus* Spp, *Klebsiella pneumonia, Pseudomonas aeruginosa, Haemophilus influenza* (type B), *Acinetobacter* spp, *Escherichia coli, and Legionela* [12, 13]. In patients with persistent abscesses or worsening clinical condition despite empiric antibiotic therapy, suspicion for other organisms such as *Mycobacterium* Spp, mycotic organisms, and parasitic infections should be considered [1, 14]. Neoplasms and autoimmune illnesses can also be a part of the differential diagnosis, given the fact that they may be associated with cavitary lesions. Aspiration of mixed oral and respiratory flora is still the most common mechanism that leads to infection and formation of a lung abscess, and one of the most common predisposing factors for aspiration has been alcohol abuse [1]. Symptoms of lung abscesses can often be confused with pneumonia or upper respiratory infections. Those include fever, dry cough or cough with

sputum production, chest pain, shortness of breath, and weight loss. Hemoptysis frequently occurs as a result of the involvement of surrounding minor vascular structures. History and physical examination are crucial, but they should be associated with a comprehensive work-up to increase the diagnostic yield. Diagnostic modalities include imaging studies (chest X-ray, CT scan, thoracic ultrasound) and microbiological studies (sputum smears and culture) should be used [5]. Invasive techniques such as bronchoscopy with bronchioalveolar lavage, needle aspiration, or video assisted thoracoscopic surgery (VATS) are also used to aid in the diagnosis and identification of causative organisms, and may also work as a treatment intervention since they can provide drainage of the purulent material. Empiric broad spectrum antibiotic therapies should be used and they must cover the most common organisms, particularly anaerobes and other mixed oropharyngeal bacterial flora. Since there has been increasing resistance of anaerobic organisms to penicillin and similar beta-lactam medications due to production of beta-lactamase penicillins should be combined with a beta-lactamase inhibitor or metronidazole for extended polymicrobial and anaerobic coverage. Intravenous clindamycin may also be used [15, 16]. Other antimicrobial agent classes such as carbapenems, cephalosporins, and newer generation fluoroquinolones can also be considered [17]. If initial antibiotic therapy is unsuccessful in clearing the infection, or if source control is required, invasive drainage techniques including percutaneous and endoscopic drainage are performed [18, 19]. Drainage is particularly essential in cases of ruptured lung abscesses. Open surgical resection, on the other hand, is seldomly done, and carries a high mortality rate [1, 20].

Streptococcus intermedius is a microaerophilic bacterium and a member of the *Streptococcus anginosus* group. Infections from *S. intermedius* have been known to be highly pyogenic and cause invasive purulent abscesses in multiple organ systems [21]. Their presence in the normal oropharyngeal flora makes them a potential pathogen that could lead to pulmonary disease, particularly in patients risk factors such as aspiration and poor dentition [22]. Their virulence can be linked to inhibition of neutrophil cytotoxins as well as production of enzymes that aid in tissue destruction and liquefaction [23, 24]. However, despite its virulence and presence in the oropharyngeal flora, only a few cases of pulmonary infection by *S. intermedius* have been described in literature, with the bacteria being more often associated with abscesses of soft tissue, liver, and brain [25, 26, 27, 28]. In such rare patients with lung infections by *S. intermedius*, the duration of symptoms prior to hospitalization is highly variable, ranging from 10 days to 5 months. *S. intermedius* has been shown to be generally susceptible to beta-lactam antibiotics, whereas metronidazole alone may not be effective. Vancomycin has been shown to be effective as an alternative if resistance to beta-lactams is detected. In cases of pleural space collections and empyema, patients are more likely to benefit from invasive drainage procedures [21]. Despite the virulence of the pathogen, the outcome and prognosis when appropriate antibiotic therapy is provided is usually favorable [28, 29].

Conclusion

Although uncommon, infections with *S. intermedius* can lead to abscess formation within the lung parenchyma even in immunocompetent individuals. Given the nonspecific and insidious clinical presentation, the diagnosis can often be delayed since the disease can be confused with other respiratory pathologies such as pneumonia or upper airway infections. Diagnostic tools such as chest radiograph, CT scan, and needle aspiration are usually required for appropriate diagnosis and identification of the infectious agent. Treatment of *S. intermedius* pulmonary abscesses requires antimicrobials, particularly beta-lactams, which should often be associated with beta-lactamase inhibitors given the coexistence of polymicrobial infection including anaerobes. Alternative regimens with different antibiotic classes may be attempted in cases of resistance. Some patients should undergo invasive procedures both for diagnostic and therapeutic reasons. These include percutaneous or imaging guided needle aspiration, catheter drainage, or video-assisted thoracoscopic surgery (VATS). Approaches that combine antibiotic therapy and some form of drainage usually provide good outcomes.

References

1. Kuhajda I, Zarogoulidis K, Tsirgogianni K. Lung abscess-etiology, diagnostic and treatment options. Ann Transl Med, 2015; 3(13):183.
2. Moreira JS, Camargo JJP, Felicetti JC, Goldenfun PR, Moreira ALS. Lung abscess: analysis of 252 consecutive cases diagnosed between 1968 and 2004. J Bras Pneumol. 2006; 32(2):136-43.
3. Hagan JL, Hardy JD. Lung abscess revisited. A survey of 184 cases. Ann Surg. 1983; 197(6):755-62.
4. Ko Y, Tobino K, Yasuda Y, Takuto S, Nishizawa S, Yoshimine K *et al.* A Community-acquired Lung Abscess Attributable to *Streptococcus pneumoniae* which Extended directly into the Chest Wall. Intern Med. 2017; 56(1):109-11.
5. Reddy S, Singh K, Hughes S. Liver Abscesses Caused by *Streptococcus intermedius* in an Immunocompromised Patient. Cureus, 2018; 10(1):2107. Published 2018 Jan 24. doi:10.7759/cureus.2107
6. Khaja M, Adler D, Lominadze G. Expressive aphasia caused by *Streptococcus intermedius* brain abscess in an immunocompetent patient. Int Med Case Rep J, 2017; 10:25-30.
7. Ramhmdani S, Bydon A. *Streptococcus intermedius*: an unusual cause of spinal epidural abscess. J Spine Surg. 2017; 3(2):243-249.
8. Mohapatra MM, Rajaram M, Mallick A. Clinical, Radiological and Bacteriological Profile of Lung Abscess - An Observational Hospital Based Study. Open Access Maced J Med Sci. 2018; 6(9):1642-1646.
9. Trabue C, Pearman R, Doering T. Pyogenic brain and lung abscesses due to *Streptococcus intermedius*. J Gen Intern Med. 2013; 29(2):407.
10. Khatib R, Ramanathan J, Baran Jr J. *Streptococcus intermedius*: A Cause of Lobar Pneumonia with Meningitis and Brain Abscesses. Clinical Infectious Diseases. 2000; 30(2):396-397.
11. Patradoon-Ho P, Fitzgerald DA. Lung abscess in children. Paediatric Respiratory Reviews, 2007; 8:77-84.
12. Bartlett JG. Anaerobic bacterial infection of the lung. Anaerobe. 2012; 18(2):235-9.
13. Nicolini A, Cilloniz C, Senarega R, Ferraioli G,

Barlascini C. Lung abscess due to *Streptococcus pneumoniae*: a case series and brief review of the literature. Pneumonol Alergol Pol. 2014; 82(3):276-85.
14. Miki M. Standard and Novel Additional (Optional) Therapy for Lung Abscess by Drainage Using Bronchoscopic Endobronchial Ultrasonography with a Guide Sheath (EBUS-GS). Intern Med. 2018; 58(1):1-2.
15. Bartlett JG. How important are anaerobic bacteria in aspiration pneumonia: when should they be treated and what is optimal therapy. Infect. Dis. Clin. North Am. 2013; 27(1):149-55.
16. Fernández-Sabé N, Carratalà J, Dorca J, Efficacy and safety of sequential amoxicillin-clavulanate in the treatment of anaerobic lung infections. Eur J Clin Microbiol Infect Dis. 2003; 22(3):185-7.
17. Ott SR, Allewelt M, Lorenz J. Moxifloxacin vs ampicillin/sulbactam in aspiration pneumonia and primary lung abscess. Infection. 2008; 36:23-30.
18. Klein JS, Schultz S, Heffner JE. Interventional radiology for the chest: Imaging-guided percutaneous drainage of epidural effusions, lung abscess, and pneumothorax. AJR Am J Roentgenol. 1995; 164(3):581-8.
19. Herth F, Ernst A, Becker HD. Endoscopic drainage of lung abscesses: technique and outcome. Chest 2005; 127(4):1378-81.
20. Singhal S, Lakhkar BN. Ruptured lung abscess: Often a result of delayed diagnosis and treatment. Respiratory Medicine CME. 2009; 2:73-76.
21. Gana TM, Awolaran O, Akhtar S. *Streptococcus milleri* and Recurrent Intra-Abdominal Abscesses: A Case Report and Literature Review. Case Rep Surg., 2016, 6297953. DOI: 10.1155/2016/6297953.
22. Ng KW, Mukhopadhyay A. *Streptococcus constellatus* bacteremia causing septic shock following tooth extraction: a case report. Cases J, 2009; 2:6493. doi: 10.1186/1757-1626-2-6493.
23. Wanahita A, Goldsmith EA, Musher DM, Clarridge JE, Rubio J, Krishnan B. *et al.* Interaction between human polymorphonuclear leukocytes and *Streptococcus milleri* group bacteria. J Infect Dis. 2002; 185(1):85-90.
24. Nagamune H, Whiley RA, Goto T, Distribution of the intermedilysin gene among the anginosus group streptococci and correlation between intermedilysin production and deep-seated infection with *Streptococcus intermedius*. J Clin Microbiol. 2000; 38(1):220-6.
25. Whiley RA, Beighton D, Winstanley TG, Fraser HY, Hardie JM. *Streptococcus intermedius*, *Streptococcus constellatus*, and *Streptococcus anginosus* (the *Streptococcus milleri* group): Association with Different Body Sites and Clinical Infections. Journal of Clinical Microbiology. 1992; 30(1):243-244.
26. Clarridge JE, Attorri S, Musher DM, Herbert J, Dunbar *S. Streptococcus intermedius*, *Streptococcus constellatus*, and *Streptococcus anginosus* ("*Streptococcus milleri* Group") Are of Different Clinical Importance and Are Not Equally Associated with Abscess. Clinical Infectious Diseases. 2001; 32(10):1511-1515.
27. Tran MP, Caldwell-McMillan M, Khalife W, Young VB. *Streptococcus intermedius* causing infective endocarditis and abscesses: A report of three cases and review of the literature. BMC Infect Dis, 2008; 8:154. Published 2008 Nov 10. DOI: 10.1186/1471-2334-8-154.
28. Hannoodi F, Ali I, Sabbagh H, Kumar S. *Streptococcus intermedius* Causing Necrotizing Pneumonia in an Immune Competent Female: A Case Report and Literature Review. Case Rep Pulmonol. 2016; 2016:7452161.
29. Tracy M, Wanahita A, Shuhatovich Y, Goldsmith EA, Clarridge JE, Musher DM. Antibiotic susceptibilities of genetically characterized *Streptococcus milleri* group strains. Antimicrob Agents Chemother. 2001; 45(5):1511-4.