

## Lung Cavitory Lesion

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A 35 year-old Caucasian woman with history of Systemic Lupus Erythematosus (SLE) complicated by Lupus glomerulonephritis presented to the Emergency Department complaining of worsening bilateral lower extremity rash and leg pain. The rash had been present for over a month and a recent biopsy of the lesion revealed leukocytoclastic vasculitis. Her usual dose of prednisone 120mg every other day had been increased to 60mg daily over the past few weeks with the worsening rash. In addition to the rash and leg pain, she also reported some right-sided pleuritic chest pain. Otherwise, the patient noted no shortness of breath, cough, fevers, or chills. Of note, she was recently treated for both pneumonia and a pulmonary embolism at an outside hospital.

Past medical history for this patient was significant for the diagnosis of SLE made six months prior along with diffuse proliferative glomerulonephritis for which she had undergone therapy with Cytoxan twice and Cellcept. She was unable to tolerate either of these treatments and was switched to oral prednisone. Two months ago, she was started on intermittent dialysis via a permacath that had been placed.

Physical exam on admission revealed an afebrile young woman with stable vital signs who was visibly uncomfortable due to the pains in her legs. She had normal heart sounds with no murmurs or rubs. Her lung exam was significant for decreased breath sounds over the right lung base, but otherwise her lungs were clear to

auscultation. Abdominal exam was unremarkable. Examination of her lower extremities revealed macular patches of an erythematous rash with scattered patches of palpable purpura that were all tender to touch. Her blood work was remarkable for a white blood cell count of 28,000 with 97% neutrophils, platelets of 73,000, hemoglobin of 10.2g/L, hematocrit of 34%, creatinine of 2.2 mg/dL and a C-reactive protein of 4.8mg/dL.

The patient was admitted to the hospital and treated with pain medications and high dose intravenous steroids for her progressive vasculitis. The leg pain and rash showed signs of improvement over the course of several days, however she continued to complain of worsening pleuritic chest pain. A chest X-ray (Figure 1) revealed a right lower lobe opacity while a ventilation/perfusion scan showed a low probability for pulmonary embolism. CT scan of the chest without contrast (due to her impaired renal function) was performed on day 6 of hospitalization (Figure 2).

On day 7 of hospitalization, the patient developed respiratory distress requiring emergent intubation. Bronchoscopy was performed revealing copious amounts of purulent secretions in the right main-stem bronchus along with its associated branches. An ultrasound-guided thoracentesis of the right side was performed and the fluid was sent for analysis. Figures 3 and 4 show two stains that were obtained from pleural fluid specimen. Please also see inside front cover for another pleural fluid specimen.



Figure 1 – Chest X-ray – Right lobe opacity



Figure 2 – Non-contrast CT of the chest – multiple nodular air space consolidations in the right middle and right lower lobe with cavitation at the right base.

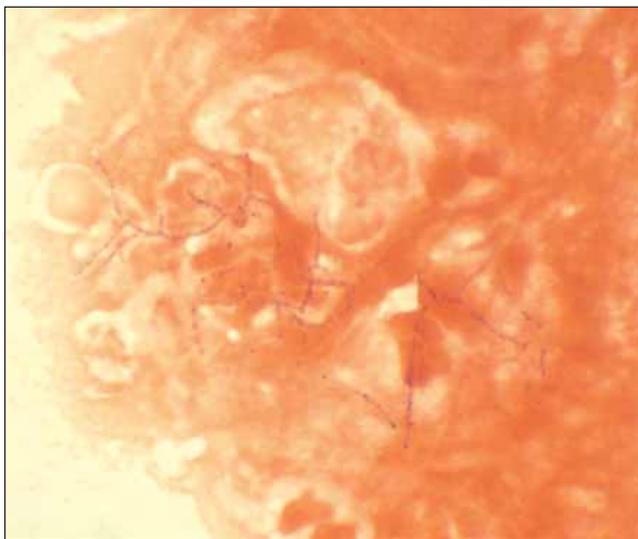


Figure 3 – Gram stain from pleural fluid specimen. Delicate filamentous gram positive rods are seen.

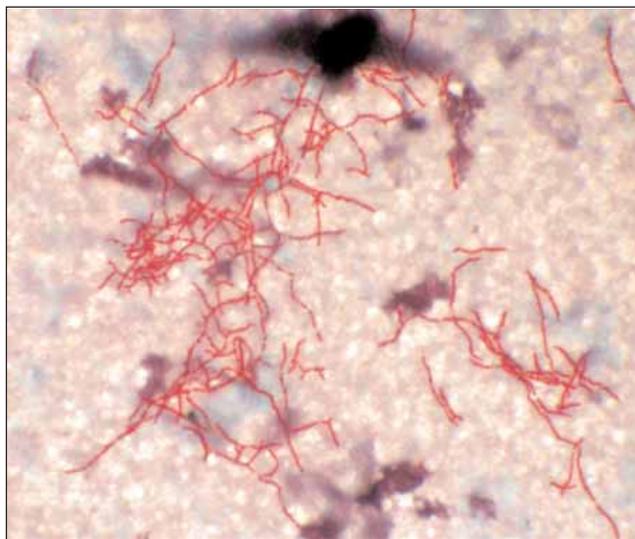


Figure 4 – Modified Acid-Fast Stain (Kinyoun) from pleural fluid specimen. Gram positive branching filamentous rods that are partially acid fast.

**What's Your Diagnosis?**  
Turn the page for the diagnosis and discussion.

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### Diagnosis: *Nocardia*

*Nocardia* is part of the actinomyces species which includes *Mycobacterium* and *Actinomyces*. They are considered aerobic bacteria which appear as filamentous, branching gram-positive rods on gram stain. In contrast to *Actinomyces*, *Nocardia* species are positive by modified acid-fast stains (Kinyoun stains) secondary to the mycolic acid content in their cell walls.

*Nocardia* is found worldwide in the soil, decaying vegetable matter, and aquatic environments. It can become airborne and ultimately inhaled or directly inoculated as a result of trauma, surgery, catheters, or animal bites and scratches. Initially, the host response involves neutrophils and macrophages engulfing the bacteria. Then a T cell-mediated response occurs which enhances this phagocytic process and helps eliminate the bacteria. Since cell-mediated immunity is so important, *Nocardia* causes disease mainly in immunocompromised individuals such as those with HIV/AIDS, prolonged steroid use, hematological malignancies, transplant recipients, and even diabetics and alcoholics (~ 60% of cases). However, nocardiosis can also occur in those with intact immune systems (~ 40% of cases).

*Nocardia* is known for its ability to affect any organ system. The most common organ involved is the lung, followed by skin and then central nervous system (CNS). Lung involvement can be acute, sub-acute, or chronic and manifested by a variety of symptoms such as cough, SOB, fevers/chills, night sweats, hemoptysis, or pleuritic chest pain. Imaging studies may reveal lobar consolidation, interstitial infiltrates, single or multiple nodules, lung masses (with or without cavitations), or pleural effusions. Skin involvement is also diverse and can be manifested by cellulitis, ulcerations, pyoderma, or subcutaneous abscesses. CNS involvement occurs as a result of special affinity of *Nocardia* to neuronal cells and results in brain abscesses with signs and symptoms such as headaches, fevers and chills, meningismus, seizures, and focal neurological deficits.

There have been no prospective randomized trials ever performed to determine what the best treatment options are for nocardial infections. Most of the data in the literature stem from experienced clinicians, microbiology/antibiotic studies, and animal models. The antibiotic of choice is trimethoprim-sulfamethoxazole (TMP-SMX) which has good tissue penetration

including the CNS. Other agents include amikacin, imipenem, 3rd generation cephalosporins, and minocycline. In addition to antibiotics, surgical intervention is necessary for any abscesses.

Former cases of nocardiosis suggest it has a tendency to relapse, but those treated for a prolonged period of time can achieve complete remission. It is recommended that immunocompetent patients with pulmonary or non-pulmonary involvement, other than CNS, be treated for 6 months. Patients who are immunocompromised or have CNS involvement should be treated for 12 months. Cure rates depend on the virulence of the *Nocardia* species and the host immune response, but in general: skin ~100%, pulm ~90%, non-pulm/non-CNS ~60%, and CNS ~50%.

### Discussion

Our young patient fits the profile of those who become susceptible to disseminated nocardiosis. She was indeed immunocompromised not only from her chronic high dose steroid use but also from her past exposure to Cytoxan and Cellcept therapies. The fact that the organisms were so readily visualized by gram stain from pleural fluid samples emphasizes the large organismal burden as well as its dissemination. Upon modified acid-fast stain confirmation, the patient was started on high dose TMP-SMX. Since speciation and sensitivities were still pending at that time, she was also started on combination therapy with ceftriaxone and linezolid. The organism was eventually confirmed as *Nocardia asteroides* sensitive to TMP-SMX. Ceftriaxone was stopped but linezolid was continued for possible synergy in combination therapy as had been recently reported. A chest tube was placed on the right lung for drainage of the purulent effusion. Three days after being intubated, the patient was extubated without complications. A head CT showed no signs of any intracranial cavitary lesions. Her steroid regimen was quickly tapered over the next two weeks and all plans for restarting Cytoxan treatment were put on hold. On day 24 of hospitalization, the patient was discharged home on oral prednisone 5mg twice a day, oral linezolid 600mg twice a day, and TMP-SMX DS two tablets twice a day.

### Reference

1. Chapman S. Diagnosis and treatment of nocardiosis. UpToDate version 12.2.2004. Available at [www.uptodate.com](http://www.uptodate.com). Accessed October 2004.
2. Moylett et al. Clinical Infectious Diseases. 36(3):313-8, 2003.