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Case Report

An unusual case of cavitary lung lesion in catheter related blood stream infection

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ABSTRACT

Cavitary lung lesions are commonly caused by *Staphylococcus aureus*, *Klebsiella pneumoniae*. *Pseudomonas* rarely causes such lesion. Here we report a case of cavitary pneumonia due to *pseudomonas* aeruginosa in the setting of catheter related blood stream infection in a 25-year-old male with crescentic IgA nephropathy who was on immunosuppressive medication.

Keywords: *Pseudomonas*, Cavitary lesion, CRBSI

INTRODUCTION

Necrotizing pneumonia refers to consolidation with necrosis resulting in the formation of multiple cavities. Common bacteriological causes of cavitary pneumonia include *Staphylococcus aureus* and *Klebsiella pneumoniae*.[1-3] *Pseudomonas aeruginosa* is a rare cause of necrotizing pneumonia resulting in cavity formation. It has been described in immunocompromised hosts (HIV, immunosuppressive therapy). Here we are reporting a rare case of cavitary pneumonia caused by *Pseudomonas aeruginosa* in the setting of catheter-related bloodstream infection.

CASE REPORT

Our patient is a 25-year-old male, with a case of crescentic IgA nephropathy who was on cyclophosphamide and Prednisolone for the last 2 months. He was hemodialysis (HD) dependent for the last 3 months with vascular access being a chronic tunneled hemodialysis catheter placed in the right internal jugular vein. He received an injection of cyclophosphamide one month prior to the current presentation. A subsequent dose was withheld because of persistent thrombocytopenia. He was taking oral prednisolone for the last 1 month. He presented to us with a history of fever for 4 days (during hemodialysis) and a dry cough for three days. There was associated bilateral chest pain on the day of admission which increased with inspiration and cough. Laboratory investigations revealed neutrophilic leukocytosis (TLC- 14,470/µL; neutrophil-89%), thrombocytopenia (platelet-95,000/µL), procalcitonin of 12.7 ng/ml (Ref: 0-0.5 ng/ml).

Considering the possibility of catheter-related bloodstream infection (CRBSI), piperacillin-tazobactam and vancomycin were started empirically in renal modified dose. Blood culture revealed the presence of *Pseudomonas aeruginosa* sensitive to Levofloxacin, Ciprofloxacin, Piperacillin-Tazobactam and Meropenem. Antibiotics were modified according to the drug sensitivity report (Piperacillin-Tazobactam was continued, Ciprofloxacin was added and Vancomycin was stopped).

Chest radiograph revealed bilateral cavitating lung lesions (Figure 1) which later showed partial resolution after 2 weeks of antibiotic therapy and removal of tunneled HD catheter (Figure 2). HRCT of the chest

revealed consolidation with surrounding ground-glass attenuation and cavity formation in the apical segment of the left lower lobe, a lateral segment of the right middle lobe and a postero-basal segment of the left lower lobe with air-fluid level within. Because of the failure of sputum induction, bronchoalveolar lavage (BAL) washings were collected. CBNAAT and fungal culture sensitivity were negative in BAL fluid. Aerobic culture from BAL fluid showed growth of *Pseudomonas aeruginosa* with a similar drug sensitivity pattern.

Chronic tunneled hemodialysis catheter was removed in view of CRBSI due to Pseudomonas. For the continuation of intermittent HD support, uncuffed femoral HD catheter was inserted. To rule out other possible sites of septic embolization, fundoscopy and echocardiography were done and the results were negative. With dual anti-pseudomonal antibiotic therapy, the patient improved clinically with the normalization of total leukocyte count and declining trend of serum procalcitonin. A repeat chest radiograph two weeks after antibiotic therapy showed partial resolution of the cavitary lesion. The patient was discharged after the placement of uncuffed hemodialysis catheter in the right internal jugular vein after 3-weeks of antibiotic therapy.

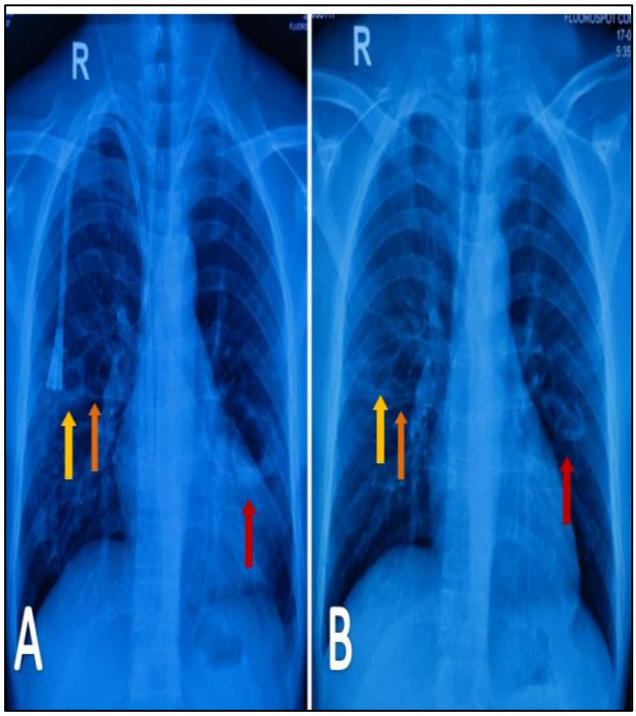

Figure 1 (A and B): Chest radiograph at the time admission showing presence of a thick-walled cavity on the left side and two cavities on the right side (arrow) (Note the presence of tunneled HD catheter on the right side), chest radiograph 2-weeks after antibiotic therapy showing partial resolution of bilateral lung cavities (arrow) (tunneled HD catheter has been removed).

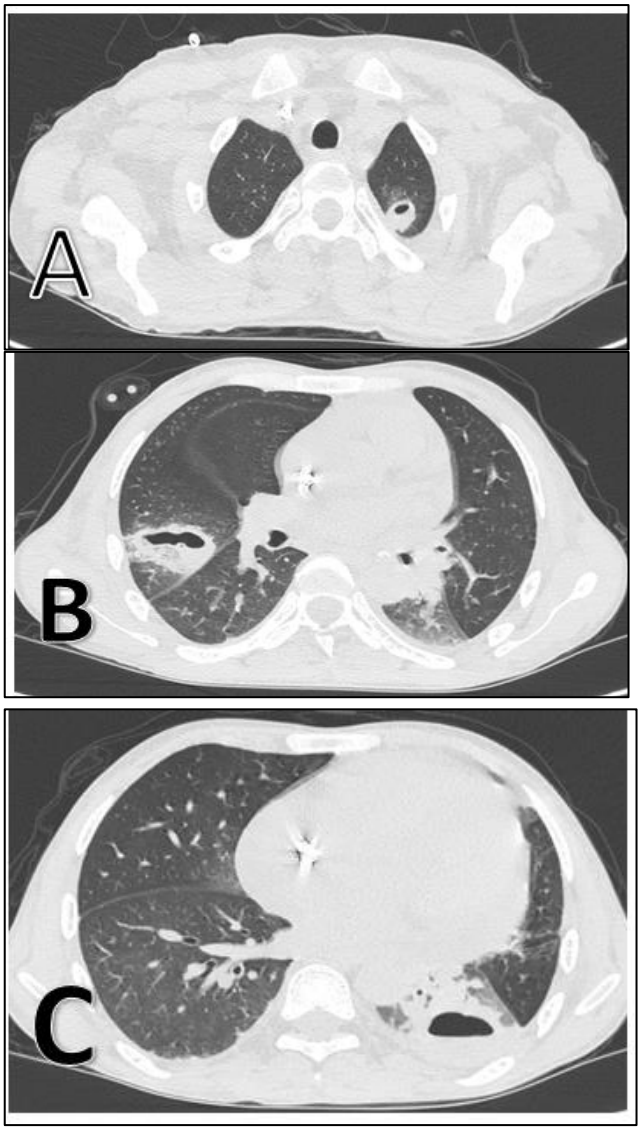

Figure 2 (A-C): HRCT of chest (CT (axial view) showing cavitary lesion in apical segment of left lower lobe, lateral segment of right middle lobe and posterobasal segment of left lower lobe.

DISCUSSION

Cavities develop in a wide variety of pathological processes involving the lung. Cavitary lung lesions[1] are caused by a bacterial infection (*Staphylococcus aureus*, *Klebsiella pneumoniae*, *Nocardia* species), *Mycobacterium tuberculosis* infection, bronchogenic carcinoma, autoimmune disease (rheumatoid nodule, granulomatosis with polyangiitis). Pseudomonas aeruginosa rarely causes cavitary lung lesions and is mostly described among immunocompromised patients secondary to human immunodeficiency virus (HIV).[2,3]

A cavity is the result of a number of pathological processes including suppurative necrosis (e.g., pyogenic lung abscess), caseous necrosis (e.g., tuberculosis), ischemic necrosis (e.g., pulmonary infarction), cystic

dilatation of lung structures (e.g., ball valve obstruction and *Pneumocystis* pneumonia), or displacement of lung tissue by cystic structures (e.g., *Echinococcus*).[6] The likelihood that a given process will cavitate depends upon both host factors and the nature of the underlying pathogenic process. The predilection to form necrotic cavities may be due to the priming of the inflammatory response by the concurrent aspiration of stomach acid or factors specific to the organism, such as endotoxin Unfortunately, there is no single common factor that differentiates organisms that are frequently associated with pulmonary cavitation from organisms that are rarely associated with pulmonary cavitation.[7,8]

Salciccioli et al reported a case of cavitating lung lesion due to *Pseudomonas aeruginosa* in a 31-year-old female patient of systemic lupus erythematosus (SLE) who was taking immunosuppressive therapy with Prednisolone and Azathioprine.[9] Kawakami et al presented a case of multiple cavitary lung lesions with an air-fluid level in a 59-year-old male who received chemotherapy about a month ago for adenocarcinoma of the lung. [10]

This case presented to us with a history of fever for 4 days (during hemodialysis) and dry cough for 3 days. Chest radiograph showed bilateral cavitating lung lesions that possibly resulted as part of septic embolization from catheter-related bloodstream infection (CRBSI) due to *Pseudomonas aeruginosa* in a patient of crescentic IgA nephropathy who was taking immunosuppressive therapy for last one month.

CONCLUSION

Pseudomonas rarely causes cavitary pneumonia. It has been reported in immunosuppressed patients (HIV, immunosuppressive medication). A 25-year-old male with crescentic IgA nephropathy who was on immunosuppressive therapy with steroid and cyclophosphamide presented with *Pseudomonas* related catheter related blood stream infection. He developed bilateral cavitary pneumonia due to *Pseudomonas* caused by septic embolization. The pneumonia resolved after appropriate antibiotic therapy and removal of tunneled hemodialysis catheter. This case highlights the importance of keeping a high index of suspicion of *Pseudomonas* as etiology of cavitary pneumonia in the context of *Pseudomonas* related CRBSI.

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