CASE REPORT

Pseudomonas aeruginosa as an unusual cause of cavitating lung lesionJustin D Saliccioli,¹ Hannah Woodcock,² Mathina Darmalingam²

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SUMMARY

A 31-year-old woman with systemic lupus erythematosus presented to the emergency department with cough for 1 week. Chest radiograph demonstrated cavitating lesion in the right upper zone with surrounding ground-glass change. Blood culture results from the day of hospital presentation grew *Pseudomonas aeruginosa*. Sputum sample and pleural fluid grew *P. aeruginosa* and were negative for acid-fast bacilli. *P. aeruginosa* is a rare cause of cavitating lung lesion and has been associated with immunocompromised hosts. Most reports of cavitating *P. aeruginosa* lesions have been identified in patients who are immunocompromised secondary to HIV. The current case highlights the potential for infection in patients who are immunosuppressed therapeutically and appropriate investigations are necessary to rule out common causes of cavitating lung lesions.

BACKGROUND

This case highlights the fact that therapeutic immunosuppression carries additional clinical risks for the patient including less common causes of lung pathology. The case also examines relevant investigations to rule out common causes of cavitating lung lesions.

CASE PRESENTATION

A 31-year-old woman with a 12-year history of systemic lupus erythematosus (SLE) presented to the emergency department with cough for 1 week. The cough was associated with fever and right-sided chest pain, worsening overnight on the day of presentation. There were no other constitutional symptoms. Following initial diagnosis of SLE, the patient had been treated for 6 years with immunosuppression and this was stopped due to leucopenia. Immunosuppression therapy was restarted 2 months prior to the current presentation and medications on admission included hydroxychloroquine 200 mg twice daily, prednisolone 5 mg once a day, azathioprine 50 mg once a day and omeprazole 20 mg once a day. There were no known drug allergies. Clinical examination revealed raised temperature and a single episode of coughing frank blood. Laboratory tests were remarkable for raised inflammatory markers with leucocyte count $11.5 \times 10^9/L$ (neutrophil 10.2; lymphocyte 1.1; monocyte 0.2; eosinophil 0.0; basophil 0.0) and C-reactive protein of 18 mg/L. D-dimer was raised at 1.14 ug Fibrinogen Equivalent Units/mL (Ref.: 0.08–0.18). She was admitted for investigation of possible pulmonary embolism (PE).

INVESTIGATIONS

Chest radiograph demonstrated cavitating lesion in the right upper zone with surrounding ground-glass change and without other obvious concerning features (figure 1). CT pulmonary angiogram (CT-PA) was performed to rule out PE. CT-PA demonstrated an unexpected large right-sided pneumothorax with mediastinal shift as well as consolidation of the posterior segments of the right upper lobe with a 15 mm cavity (figure 2). There was no evidence of PE. A right-sided intercostal chest drain was inserted for treatment of pneumothorax and pleural fluid analysed for microbiology, culture and sensitivity as well as acid-fast bacilli (AFB) for *Mycobacterium* spp. Sputum sample and bronchoalveolar lavage washings were collected for microbiology and AFB.

Blood culture results from the day of hospital presentation grew *Pseudomonas aeruginosa*. Sputum sample and pleural fluid grew *P. aeruginosa* and were negative for AFBs. Bronchial washings showed scanty Gram-negative rods with cellular smears containing alveolar macrophages, neutrophil polymorphs and lymphocytes. There were no

Figure 1 Chest radiograph showing a well circumscribed cavitating lesion in the right upper zone.

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Figure 2 Transverse section of CT pulmonary angiogram reveals a large right-sided pneumothorax with mediastinal shift and a 15 mm cavity in the right upper lobe.

fungal elements or features of *Pneumocystis pneumonia* and no granulomatous formations.

TREATMENT

The patient received a 2-week course of Tazocin (4.5 g three times a day, per hospital protocol) for treatment of *P. aeruginosa* bacteraemia of chest origin and improved systemically with therapy. The pneumothorax improved with intercostal chest drain and this was removed on hospital day 9 without complication.

OUTCOME AND FOLLOW-UP

The patient remained otherwise clinically stable during the hospital course and she was discharged to resume regular follow-up with the rheumatology service for management of SLE.

DISCUSSION

Pseudomonas aeruginosa is a rare cause of cavitating lung lesion and has been associated with immunocompromised hosts.¹ Most reports of cavitating *P. aeruginosa* lesions have been identified in patients who are immunocompromised secondary to HIV and the current case highlights the potential for infection in patients who are immunosuppressed therapeutically.^{2 3} Cavitating lung lesions may be caused by neoplasm (eg, squamous cell carcinoma), inflammatory disorders (eg, rheumatoid nodules or granulomatosis with polyangiitis) or infectious aetiologies (eg, *Mycobacterium tuberculosis*, *Nocardia* spp, *Coccidioidomycosis immitis*) and appropriate investigations are necessary to rule out common causes of cavitating lung lesions.

Learning points

- ▶ Pulmonary cavitory lesions may be caused by neoplastic, inflammatory or infectious disorders.
- ▶ Appropriate investigation is necessary to exclude common infectious causes of cavitating lesions.
- ▶ *Pseudomonas aeruginosa* may cause cavitating lung lesions and should also be considered in immunocompromised hosts.

Contributors JDS and HW were responsible for management of the subject and MD was the respiratory consultant of record during the hospital admission. JDS and HW drafted the report, obtained patient consent and performed relevant literature search for the case. MD was responsible for critical review and editing of the manuscript.

Competing interests None declared.

Patient consent Obtained.

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