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Rhodococcal lung abscess in a renal transplant recipient

Koh-Wei Wong, Bharathan Thevarajah

Nephrology Unit, Department of Medicine, Queen Elizabeth Hospital, Kota Kinabalu, Sabah, Malaysia

Summary

Background: Rhodococcus species are relatively rare human pathogens, but are being increasingly recognized as causes of infection especially in immunosuppressed patients.

Case Report: We present a case of Rhodococcus lung abscess in a patient 10 months post-cadaveric renal transplant, successfully treated with a combination of antibiotics. She required a prolonged course of oral antibiotics for 6 months. She did not require surgical intervention. Chest X-rays and CT thorax showed complete resolution of the initial lesion. We also review the medical literature related to Rhodococcus infection in patients with renal transplantation. Rhodococcus infection should be considered as in the differential diagnosis of immunosuppressed patients who present with lung abscess/mass.

Conclusions: A literature review indicates this is a potentially fatal condition with disseminated sepsis/abscesses.

key words: ***Rhodococcus*** • **lung abscess** • **renal transplantation**

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Author's address: Koh-Wei Wong, Nephrology Unit, Department of Medicine, Queen Elizabeth Hospital, Kota Kinabalu, Sabah, Malaysia, e-mail: drwongkohwei@gmail.com

Background

Rhodococcus species (*R. equi* especially) have for the past 3 decades been recognized as causes of pulmonary infection, especially among immunosuppressed patients. They are aerobic, intracellular, gram-positive coccobacilli that cause granulomatous inflammation [1–4].

We present a case of lung abscess due to *Rhodococcus* species in a patient who had a renal transplant less than a year before.

Case Report

A 34-year-old woman with a cadaveric renal transplant was admitted with a history of fever for 2 days and productive cough.

She had undergone a cadaveric renal transplantation in July 2010, 10 months before this hospitalization. The immunosuppression for her renal transplant consisted of IV methylprednisolone and IV thymoglobulin, followed by oral tacrolimus, mycophenolate acid (Myfortic) and prednisolone. She had delayed graft function, requiring 3 sessions of hemodialysis immediately post-transplantation. The case was further complicated by an episode of acute cellular rejection (Banff 4b, type 1A), which responded to IV methylprednisolone for 3 days. Subsequent graft biopsy showed severe hypertensive changes, with only mild tubulitis. Prior to the transplantation she was on hemodialysis for 14 years (since 1996, primary etiology was unknown). She was a nonsmoker, and a housewife.

On admission, she was noted to have high-grade fever and mild tachypnea. She was otherwise hemodynamically stable. The result of lung examination was essentially normal. An initial chest X-ray revealed a mass in the right upper zone, which developed into a cavity with an air-fluid level (chest X-ray 2 days post-admission) (Figures 1, 2). A CT scan of the thorax showed a cavity in the right lung, which could represent an infective lesion with endobronchial spread. Her renal function deteriorated acutely, and serum creatinine rose from her baseline of 110 umol/L to more than 200 umol/L. Her CRP was raised, at 11.4 mg/dL (normal <0.30 mg/dL), and ESR was 43 mm/hour. Her white blood cell count was not raised, at 6.5×10^3/uL. She was initially treated with IV ceftriaxone 2 g daily and oral azithromycin 500 mg daily.

On her 4th day of admission, her blood culture grew *Rhodococcus* species, sensitive to cefuroxime (parenteral), linezolid, rifampicin, vancomycin and meropenem, and resistant to chloramphenicol and tetracycline. In view of the culture findings and her persistent spiking fever, the antibiotics were switched to IV meropenem and metronidazole, her dose of mycophenolic acid (Myfortic) was halved, and her tacrolimus dosage was also reduced.

On her 10th day of admission, she was still having persistent spiking fever; IV fluconazole was added to cover the possibility of fungal infection, and her mycophenolic acid was stopped. Bronchoscopy was done, and the findings showed pus secretions from the lateral segment of the right upper lobe, and due to bleeding, transbronchial lung biopsy was not performed.

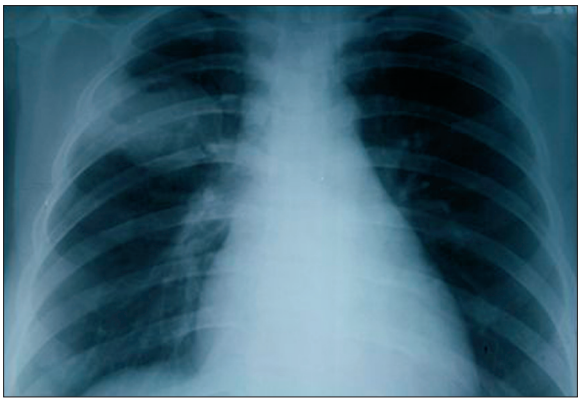

Figure 1. Day 1 of admission.

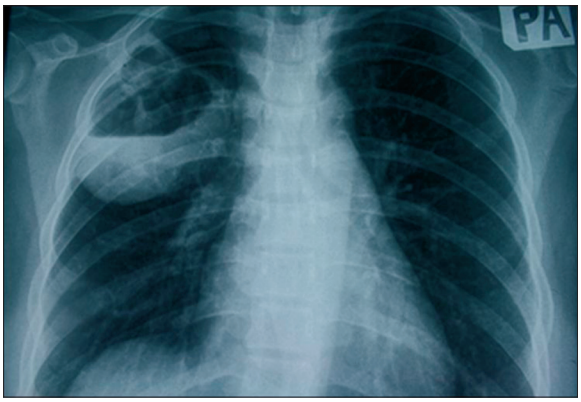

Figure 2. Day 3 of admission.

Her fever eventually subsided on day 12 of admission, with improvement in her cough. The bronchial washing results showed a negative culture, with no evidence of acid-fast bacilli or malignant cells. Endobronchial biopsy showed no evidence of granuloma or malignancy. Her serum creatinine slowly improved to her baseline of 110 umol/L.

IV metronidazole and fluconazole were stopped after 2 weeks, and she was continued on IV meropenem 1 g bd, and oral azithromycin 250 mg od was added. She remained afebrile, and her renal function was stable on tacrolimus and prednisolone. Her CRP and ESR were coming down to normal level as well. She was given IV meropenem for a total duration of 4 weeks, and was then put on a prolonged course (we initially planned for at least 2 months) of oral azithromycin and ciprofloxacin. Mycophenolic acid was restarted at a low dose (180 mg bd). She was discharged after a month of hospitalization.

One month after her discharge she developed fever and cough again, with greenish/chocolate coloured sputum. The repeated chest X-ray showed the same lesion (Figure 3), with no evidence of reduction in size. Repeated blood and sputum cultures, and tuberculosis work-up had all been negative. She was continued on oral azithromycin and ciprofloxacin, while IV meropenam was restarted and her mycophenolic acid was stopped again. A repeat CT scan of the thorax showed reduction of the cavity (from 7.3×9.3 cm to 5.6×5.7 cm). She was treated with meropenem for another week, and was discharged after 2 weeks of hospitalization. Again, mycophenolic acid was started at a low dose (180 mg bd).

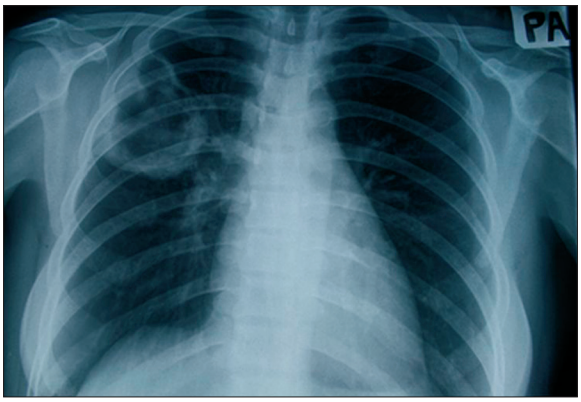

Figure 3. One month after first presentation.

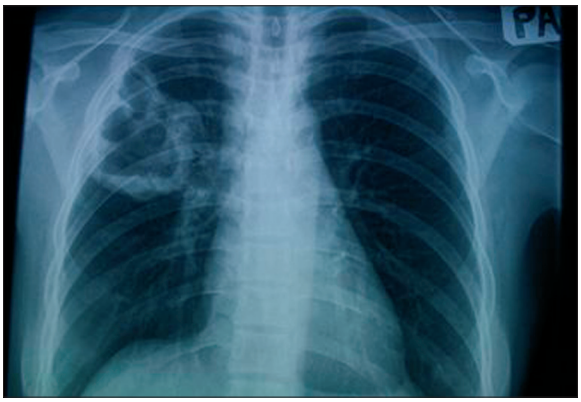

Figure 4. Two month after first presentation.

One month after her second discharge, the repeated chest X-ray showed the same lesion (Figure 4), her ESR has come down to 14 mm/hour and CPR was 0.10 mg/dL. Although lobectomy was considered by the cardiothoracic team, the patient was reluctant.

A repeat CT scan of thorax was done 6 months after her first presentation, and it showed resolution of the abscess, and her chest X-ray also showed the lesion was cleared up (Figure 5). Her oral azithromycin and ciprofloxacin were stopped after 6 months. She remained well 7 months after the initial presentation, with her renal function at the post-transplantation baseline level (110 umol/L).

Discussion

Rhodococcus species are still an uncommon cause of infection in clinical practice, though they have been reported with increasing frequency among immunosuppressed patients [1–3].

It was first identified as a pathogen in 1923 from the lungs of foals. The first human infection was reported in 1967 from a young man treated with corticosteroid and 6-mercaptopurine [1,2]. Since then it has been reported sporadically, mainly among immunocompromised patients, such as those with acquired immune deficiency syndrome, hematological malignancies, and solid organ transplantation recipients [1–4].

It is a gram-positive obligate aerobic coccobacillus, which may be confused morphologically as diphtheroids and disregarded as contaminant. We managed to isolate this organism from a blood culture, but the cultures from endobronchial washings were negative.

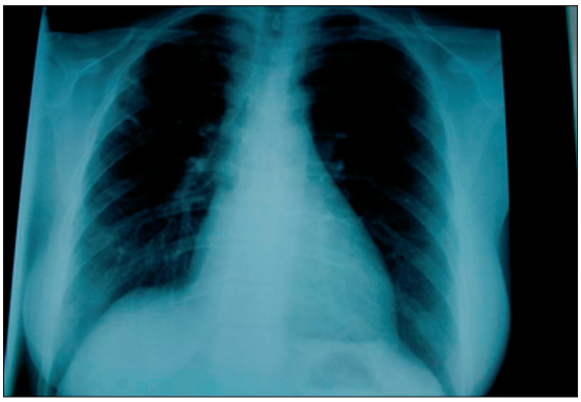

Figure 5. Six months after first presentation.

A literature search on PubMed at the time of writing revealed about 20 case reports on *Rhodococcus* infection in renal transplant patients [5–19]. Combination synergistic antibiotics were used in most cases, and some cases required surgical drainage. Most cases involved the lungs, suggesting this organ as the primary site of involvement. Pneumonia is the most common clinical manifestation. Other extra-pulmonary infected sites reported were disseminated abscesses, brain, pericarditis, lymphadenitis, kidney (allograft), osteomyelitis and skin [5,10,17–19]. Those extra-pulmonary sites were most likely infected via hematogenous spread.

As mentioned above, the commonest presentation is pneumonia, and micronodular necrotizing pneumonia with abscesses with cavities are the prevalent pathology; only a few reports mentioned lung masses, as in our patient. One required surgical resection, and others seemed to resolve without the need of surgical intervention.

The antimicrobial treatment for *Rhodococcus* is varied from the cases reported. Use of synergistic antibiotics with high intracellular penetration is recommended. Carbapenem, vancomycin, ciprofloxacin, clarithromycin and rifampicin have all been used with success [6–8,10,11,13]. After the initial clinical response, most reports advocated 4 to 6 months of antibiotics. In our case, we used IV meropenam and oral azithromycin for 4 weeks, followed by 6 months of oral azithromycin and ciprofloxacin.

As immunosuppression is the major risk factor of Rhodococcal infection, most authors advocated the reduction of immunosuppressive medications [7,10]. We initially reduced the dosage of mycophenolic acid and tacrolimus, and eventually stopped the mycophenolic acid during her hospitalization. We then cautiously reintroduced it later, when the infection appeared to be under control.

Conclusions

In conclusion, Rhodococcal infection should be one of the many differential diagnoses of pulmonary infection or abscess among immunosuppressed patients, along with other common opportunistic infections such as mycobacterial infection.

Conflict of interest

The authors declare that they have no conflicts of interest.

Acknowledgement

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