Received: 17 December 2023 | Accepted: 20 January 2024

DOI: 10.1002/rcr2.1293

CASE REPORT

Respirology Case Reports OPEN ACCESS APSR WILEY

A case of hot tub lung secondary to mycobacterium avium complex in Victoria, Australia

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Associate Editor: Arata Azuma

Abstract

Hot tub lung (HTL) is a type of Hypersensitivity Pneumonitis (HP) caused by inhalation of aerosols containing non-tuberculous mycobacteria (NTM). We report the first case of hot tub lung in Victoria, Australia. A 31-year-old female presented with 3 weeks of dyspnoea, fevers and malaise, despite a course of antibiotics. She had used an indoor spa frequently for the past several months. She was hypoxic saturating 86% on room air. Examination revealed fine bilateral basal crepitations without wheeze. Inflammatory markers were elevated. A high resolution CT (HRCT) chest demonstrated extensive bilateral ground glass changes and centrilobular nodular opacities involving upper and lower zones. Bronchial washings were obtained and isolated Mycobacterium Avium Complex. Clinically, the patient had excellent response to empirical corticosteroids and avoidance of hot tub. This case highlights the importance of clinical history taking and suspicion of Hot Tub Lung as a differential diagnosis in a patient with a compatible clinical history, radiology and microbiology and exposure to a hot tub.

KEYWORDS

hot tub lung, hypersensitivity pneumonitis, interstitial lung disease, mycobacterium avium complex

INTRODUCTION

Hypersensitivity pneumonitis (HP) is a type of interstitial lung disease that occurs as an abnormal immunologic response to an antigen in a genetically susceptible individual. Hot tub lung (HTL) is a type of HP caused by the inhalation of aerosols containing non-tuberculous mycobacteria (NTM). Presenting symptoms include cough, shortness of breath and fever. Imaging findings on high resolution computer tomography (HRCT) chest include diffuse centrilobular nodules and ground glass opacities.[1] Clinical suspicion of HTL is important as the treatment differs from NTM pulmonary infections. The first case report was published in 1997[2] and to our knowledge, there have been only a few described case reports in Australia.[3,4] We report the first case of hot tub lung in Victoria, Australia (Figures 1 and 2).

CASE REPORT

A 31-year-old female presented to our hospital with 3 weeks of progressive shortness of breath, intermittent fevers, and malaise. There had been no improvement with oral antibiotics in the community. She had no significant past medical history and had been a lifelong non-smoker. She worked as a professional horse jockey but had minimal exposure to hay and other animals. Further questioning revealed that she used indoor spa regularly in the past year.

On presentation, oxygen saturations were 98% with 4 L/minute oxygen supplementation and her blood pressure and heart rate were stable. Pulmonary findings on physical examination demonstrated fine bi-basal crepitations without wheeze. There was no finger clubbing or cervical lymphadenopathy and no signs of decompensated heart failure.

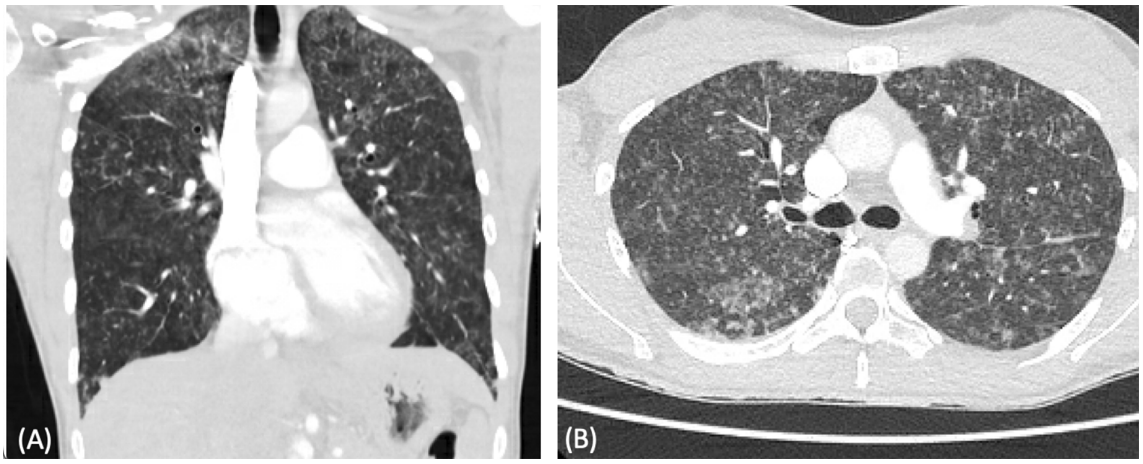

FIGURE 1 High resolution computer tomography chest on presentation with coronal (A) and axial (B) views demonstrating bilateral ground glass opacities and centrilobular nodules in a bilateral distribution.

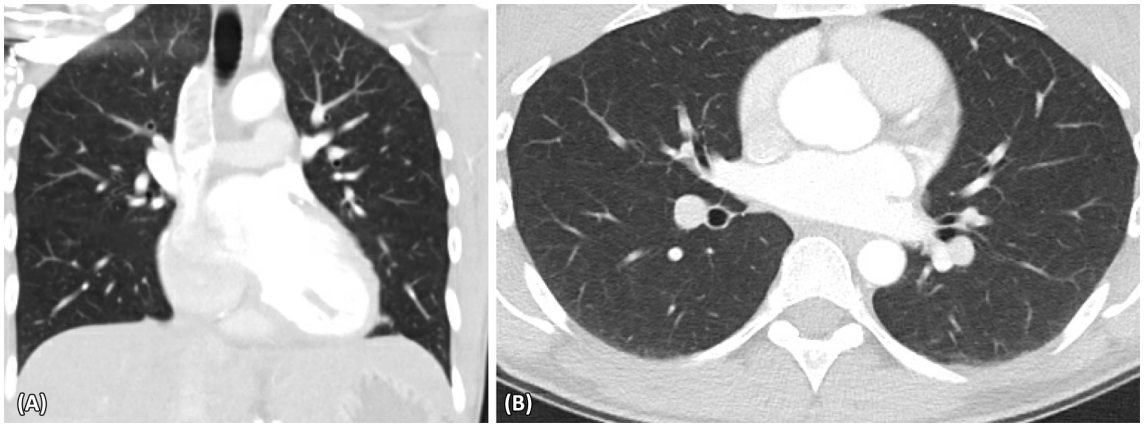

FIGURE 2 High resolution computer tomography chest after corticosteroid treatment demonstrating interval resolution on coronal (A) and axial (B) views.

Full blood examination showed a mildly elevated white cell count of 12.8 $\times$ 10^{-9}/L. Eosinophil count was not elevated. Her electrolytes, renal function and liver function tests were unremarkable. Inflammatory markers were raised with an erythrocyte sedimentation rate of 31 mm/h and C-reactive protein of 38.1 mg/L. Autoimmune serology including anti-nuclear antibodies, anti-neutrophilic cytoplasmic antibody and extractable nuclear antigens were negative. Aspergillus precipitins were negative. Blood cultures were negative and sputum cultures were unable to be obtained in the absence of a productive cough.

Chest x-ray demonstrated widespread bilateral infiltrates. HRCT of the chest demonstrated extensive bilateral ground glass changes and centrilobular nodular opacities involving upper and lower zones, with changes more prominent in the upper and middle lobes (Figure 1). There was no intrathoracic lymphadenopathy. Spirometry was within normal limits and the diffusing capacity of carbon monoxide was normal.

A bronchoscopy was performed, with bronchoalveolar lavage (BAL) and washings taken from the right upper lobe. Unfortunately, cell count differential was not performed but microscopic evaluation revealed frequent alveolar macrophage cells, lymphocytes, and neutrophils. There was no evidence of malignant cells or fungal elements. Staining for acid-fast bacilli (AFB) and fungal organisms were negative. Culture for AFB was performed and subsequently grew Mycobacterium Avium Complex (MAC). Specific culture for fungal organisms and Aspergillus polymerase chain reaction (PCR) tests were also performed on BAL and washings and were negative.

Treatment initially included intravenous antibiotic therapy with Piperacillin/Tazobactam and azithromycin. Upon discussion and review of the clinical case in a multidisciplinary meeting, clinical suspicion of HTL was raised and consensus was reached to commence prednisolone. Prednisolone was commenced at a dose of 50 mg daily for 1 week, followed by 25 mg daily for 1 week, 15 mg daily

for 1 week, 10 mg daily for 1 week and 5 mg daily for 1 week. Pneumocystis Jiroveci prophylaxis was also commenced. Her symptoms improved significantly, her oxygen requirements resolved within 2 days and she was subsequently discharged. We also advised her to cease using her indoor spa. Unfortunately by this time, the patient had her indoor spa cleaned and we were unable to test the spa water for micro-organisms.

She was reviewed in subsequent outpatient respiratory clinic visits in the following months. She had removed her indoor spa. Her symptoms had completely resolved and repeat imaging showed complete resolution of the previous infiltrates, without evidence of fibrotic change (Figure 2). She had also returned to her premorbid physical activity levels. Given her improvement with antigen avoidance, corticosteroid therapy and isolation of MAC on BAL samples, without culturing of other potential culprit pathogens such as fungi, there was clinical consensus that our patient's diagnosis was Hot Tub Lung secondary to MAC.

DISCUSSION

HP can be a challenging diagnosis as there are numerous inciting culprit antigens ranging from bird to mould to farming exposures.[5] Identifying the correct culprit antigen in HP is important as it guides subsequent management. Moreover, untreated HP can lead to fibrosis and a poor prognosis.[6] NTM are atypical mycobacteria found ubiquitously in the environment,[7] often in soil and water distribution systems, including showers, swimming pools and spas. NTM such as MAC are resistant to chlorine[8] and thus, poor or infrequent maintenance of hot tub or other water systems can predispose to the development of hot tub lung via inhalation of soil or water aerosols containing NTM. Though it is important to note that exposure to MAC does not always lead to HTL, suggesting that individuals affected have a genetic predisposition. Furthermore, fungi such as Trichosporon species can be found in water systems, which can lead to fungi-induced HP[9] and thus, should also be considered in the diagnostic work-up.

Diagnosis of HTL requires a suggestive clinical history, exposure to hot tub, compatible radiology and microbiology. Culture of NTM (most commonly MAC) is often obtained from respiratory tract (e.g., sputum, bronchial washings or lavage) and whilst not obtained in our case, collection of culprit water samples can be subsequently cultured for mycobacteria.[10,11] Broncho-alveolar lavage fluid is predominantly lymphocytic. Radiological findings of HP include abnormalities such as ground glass opacities, centrilobular nodules and gas trapping, commonly in a diffuse distribution. A high index of clinical suspicion with the appropriate radiological and microbiological findings can obviate the need for more invasive investigations such as lung biopsies, though histopathological examination can demonstrate non-necrotising granulomas and a mild chronic interstitial pneumonia.[1] Other histopathological patterns including organizing pneumonia and necrotising granulomas have been reported.[11]

Treatment involves hot tub avoidance and corticosteroids in the majority of cases. Anti-tuberculous therapy may be considered in patients who do not improve, but the utility and efficacy is unclear.[11] In our case, although, the water of the spa could not be tested and thus the origin of the exposure is limited to circumstantial evidence, the patient improved dramatically with hot tub avoidance and a weaning course of prednisolone.

HP can be a challenging diagnosis due to the numerous potential causes. Furthermore, HTL is rare and is limited to a few case reports in the Australian context,[3,4] and is often not considered in the differential diagnoses. Our case highlights the importance of a thorough clinical and exposure history and clinical suspicion of HTL in managing patients with undifferentiated interstitial pneumonitis.

AUTHOR CONTRIBUTIONS

All authors were involved in the preparation of the manuscript.

ACKNOWLEDGMENTS

This case was presented as an abstract at the APSR congress, Singapore, 2023.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analysed in this study.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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How to cite this article: Chhor L, Tumali RS, Östberg C, Sachdev A. A case of hot tub lung secondary to mycobacterium avium complex in Victoria, Australia. Respirology Case Reports. 2024; 12(2):e01293. https://doi.org/10.1002/rcr2.1293