A Unique Case of Hot Tub Lung Worsening during the Winter

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Abstract

Summer-type hypersensitivity pneumonitis includes a spectrum of granulomatous lung diseases that result from the inhalation of Trichosporon species in the summer. Hot tub lung is a granulomatous lung disease caused by the inhalation of water aerosols containing non-tuberculous mycobacteria. We herein describe a case of hot tub lung that deteriorated during the winter season. Every winter, the patient’s symptoms, laboratory findings and chest images worsened. Genetically identical Mycobacterium avium strains were detected in his sputum and bathtub. The circulation of bathtub water during the winter months only exacerbated his symptoms in the winter.

Key words: hot tub lung, winter season, Mycobacterium avium


Introduction

Summer-type hypersensitivity pneumonitis (HP) is caused by the inhalation of Trichosporon asahii or mucoides during the hot and humid summer season, while hot tub lung is a diffuse granulomatous lung disease that results from the inhalation of water aerosols containing non-tuberculous mycobacteria (NTM). We herein describe a case of hot tub lung that deteriorated in the winter with genetically identical Mycobacterium avium (M. avium) isolates detected in samples obtained from the patient’s sputum and bathtub circulation adapter.

Case Report

A 59-year-old man with a cough and dyspnea presented to a local doctor around December 2010. Chest computed tomography (CT) showed diffuse ground glass opacity (GGO) in the bilateral lung fields. After several months, the patient’s symptoms and chest CT findings improved, whereas the serum level of Krebs von den lungen-6 (KL-6) remained high; therefore, he was referred to the Kinki-Chuo Chest Medical Center in March 2011. The patient’s medical history included diabetes mellitus starting at 53 years of age and surgery for colon cancer at 57 years of age. He was an ex-smoker with no pets, humidifiers or exposure to dust, and used a down-filled quilt. He lived in a 17-year-old wooden house and worked in air condition management in an office building; his family had no specific past medical history. The results of a physical examination performed on admission were as follows: height =168 cm, body weight = 73 kg, no palpable surface lymph nodes, no fine crackles on chest auscultation, and no clubbed fingers or skin rashes. Meanwhile laboratory examinations of the peripheral blood conducted on admission showed the following findings: white blood cells (WBCs) =6,500/μL, lactate dehydrogenase (LDH) =201 IU/L, C-reactive protein (CRP) =0.08 mg/dL, KL-6 =2,485 U/mL, pulmonary surfactant protein-D (SP-D) =308 ng/mL, pulmonary surfactant protein-A (SP-A) =48.9 ng/mL, angiotensin-converting enzyme (ACE) =14.9 U/L, brain natriuretic peptide (BNP) =2.3 pg/mL and anti-tuberculous glycolipid (TBGL) antibodies =9.7 U/mL. Tuberculin reactions were negative; however, chest radiography...
revealed bilateral GGO and a high-resolution CT (HRCT) of the chest showed GGO in a centrilobular pattern in all lung fields (Fig. 1). The results of a pulmonary function tests and an arterial blood gas analysis were within the normal ranges. Bronchoalveolar lavage (BAL) was performed in the right B4a; the rate of recovery of bronchoalveolar lavage fluid (BALF) was 78%, and the total cell count in the fluid was 3.95×10⁷/mL, with 24.5% macrophages, 75.0% lymphocytes, and 0.2% neutrophils. The CD4/CD8 ratio of lymphocytes was 7.1. No microorganisms were detected in the BALF. Transbronchial lung biopsies (TBLBs) of the right B4b and B3a showed epithelioid granulomas and mural alveolitis with the infiltration of lymphoid cells (Fig. 2). However, no microorganisms were identified in the lung biopsy specimens with using Ziehl-Neelsen and Grocott’s staining. After recovering in the hospital, the patient returned to his house for two days. Consequently, the PaO₂ decreased from 92.2 mmHg to 83.6 mmHg on room air, the

Figure 1. Chest radiography revealed bilateral ground glass opacity (GGO). A high resolution CT (HRCT) scan of the chest showed GGO in a centrilobular pattern in all lung fields.

Figure 2. The transbronchial lung biopsy (TBLB) specimens obtained from the right B4b and B3a showed epithelioid granulomas and mural alveolitis with the infiltration of lymphoid cells.
CRP level increased from 0.13 mg/dL to 0.53 mg/dL and the GGO on chest HRCT increased slightly. Based on these findings, he was suspected of having HP, although the antigen was not clarified. The patient was subsequently advised to avoid feather products and have others clean up any environmental mold. After discharge, his condition progressed well for a few months; however, the GGO on chest HRCT deteriorated and the KL-6 level increased in December 2011. He was therefore hospitalized again, and his laboratory data improved over a few days without medication. At that time, Mycobacterium avium was cultured from his sputum. While following the patient in the outpatient setting, his condition subsequently worsened and he was again hospitalized in January 2013, at which time Mycobacterium avium was cultured from...
Discussion

We herein presented a case of hot tub lung that deteriorated in the winter season. The combination of inadequate hot tub maintenance and poor personal hygiene promoted the growth of *Mycobacterium avium* complex (MAC) organisms, and jet aerosolization with the subsequent inhalation of large amounts of MAC presumably led to the development of the disease (1).

In the present case, *M. avium* was detected on the bathtub faucet, drainage components and the filter of the bathtub circulation adapter. The warm water in the bathtub was not aerosolized, but rather circulated automatically throughout the day in winter in order to prevent freezing of the pipes. Therefore, we speculate that *M. avium* proliferated under the conditions of warm circulating water, especially in the winter. The patient’s exposure to large amounts of *M. avium* in the winter compared to that observed in other seasons may explain his seasonal deterioration. Indeed, it has been reported that the inhalation of high doses of MAC within a short period is required to develop hot tub lung (2).

The clinical course of the present patient was seasonal, similar to that of summer-type HP, the most prevalent form of HP in Japan. Summer-type HP is caused by *Trichosporon asahii* or *mucoides* in which the symptoms are worse during the humid summer season, and among residents of old, wooden houses (3, 4). In the current case, the patient’s symptoms worsened only in the cold winter. There is the potential for HP associated with the use of feather products or humidifiers in the winter. However, this possibility was excluded in this case because the patient and his family avoided feather products and did not use humidifier.

According to the American Thoracic Society (5), a diagnosis of hot tub lung may be made in patients with the subacute onset of respiratory symptoms associated with hot tub exposure, positive mycobacterial cultures from respiratory samples and water and characteristic radiographic findings. These guidelines do not require the genetic identification of NTM strains from respiratory samples and water, although determining the genetic type of NTM is important specifying the causal relationship, as NTM is distributed ubiquitously in the environment (6). There are few cases of hot tub lung in which the genetic type of NTM in specimens obtained from both the patient and the environment has been confirmed (7-9), with an especially limited number of such cases in Japan (10). Therefore, the present case is valuable, as hot tub lung was confirmed based on the genetic identity of the bacteria. Further more, avoiding the inhalation of NTM is essential for treating hot tub lung (5). In the current case, after an adequate periods of antimycobacterial therapy, the patient’s laboratory parameters, including the KL-6 and SP-D levels, increased slightly, the patient exhibited no symptoms and his chest CT imaging findings improved.

The authors state that they have no Conflict of Interest (COI).

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References


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http://www.naika.or.jp/imonline/index.html