Hot Tub Lung: Case Report and Review of the Literature

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ABSTRACT

Introduction: Nontuberculous mycobacteria-related hypersensitivity pneumonitits (NTM-HP), otherwise known as hot tub lung, is an uncommon disease produced by exposure to aerosolized hot tub water containing nontuberculous mycobacteria. Patients usually present with nonspecific, prolonged respiratory symptoms and require a thorough respiratory workup, including radiography and even pulmonary biopsies.

Case Presentation: We present the case of a 58-year-old patient with chronic respiratory symptoms and history of exposure to a hot tub.

Discussion: There is little data on why certain patients develop NTM-HP; however, it seems to be an immunologic response to the nontuberculous mycobacteria, not a primary infection. The treatment, as in this case, is typically just hot tub avoidance.

Conclusion: To our knowledge, this is the first case of NTM-HP reported from Wisconsin. NTM-HP can mimic nontuberculous mycobacterial disease and should be on the differential diagnosis for patients with unclear chronic respiratory problems.

INTRODUCTION

Nontuberculous mycobacteria-related hypersensitivity pneumonitits (NTM-HP), otherwise known as hot tub lung, is an uncommon disease produced by exposure to aerosolized hot tub water containing nontuberculous mycobacteria. Patients usually present with nonspecific, prolonged respiratory symptoms and require a thorough respiratory workup, including radiography and even pulmonary biopsies.

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CASE PRESENTATION

A 58-year-old man with chronic obstructive pulmonary disease, obesity, essential hypertension, and obstructive sleep apnea was evaluated in fall 2019 for progressive dyspnea, fatigue, and occasional nonproductive cough for the past 4 years. His dyspnea was advancing, and he was unable to walk on a level surface without resting. He used continuous positive airway pressure (CPAP) at night for his sleep apnea and had a diagnosis of chronic obstructive pulmonary disease (COPD) and was on home oxygen, although he used it sparingly. He denied edema, orthopnea, fever, and weight loss.

He was a lifelong nonsmoker and worked in medical cyclotron with intermittent exposure to radiation at 800 mr (0.008 Gy) per quarter in the last 16 years; however, cumulative dosing was not felt to be above level of safety (< 20 Gy). He used a hot tub at home twice daily for the past 5 years.

The patient was afebrile at presentation, body mass index was 31.19 kg/m^2, blood pressure 144/110, tachycardia with a heart rate of 105, respiratory rate 18 per minute, and oxygen saturation at 94% with 1L of intranasal oxygen. His respiratory system examination showed bilateral inspiratory crackles with no wheezing noted. The remaining clinical examination was unremarkable.

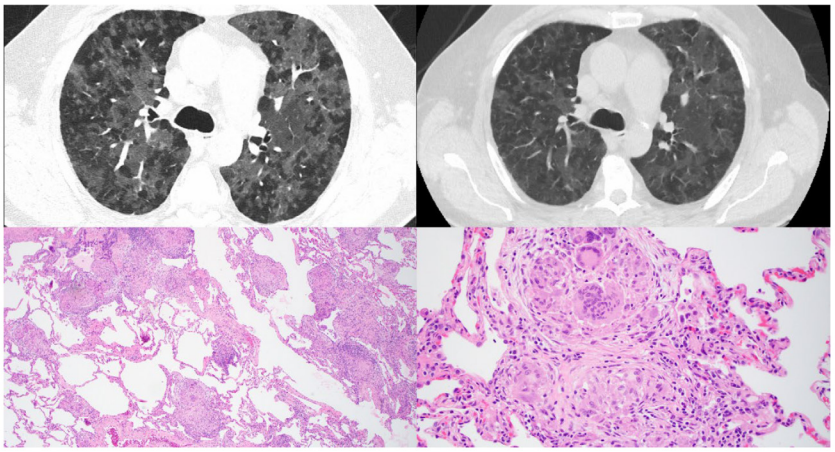
Laboratory examination of the peripheral blood showed the following findings: white blood cells (WBC) 8,200 cel/uL, absolute eosinophils 200/uL, creatinine 0.85 mg/dL, calcium 9.9 mg/dl. Chest computed tomography (CT) showed diffuse bilateral ground glass opacities with mosaic attenuation and no evidence of emphysema; sedimentation rate was 10 mm/h,

C reactive protein was 7.0 mg/dL, antinuclear antibody (ANA) was negative, cytoplasmic neutrophil antibody was negative, rheumatoid factor was negative. An allergen panel for detecting IgE antibodies to some hypersensitivity pneumonitis etiologic agents like *Aspergillus fumigatus, Alternaria alternate* and others allergens that are common in Michigan, Minnesota, and Wisconsin was negative. Serum IgE was within normal range, and the patient's antibody test was negative for *Aspergillus fumigatus, Thermoactinomyces vulgaris, Thermoactinomyces sacchariis, Aurobasidium pullulans, Micropoly faeni*, and pigeon serum.

Echocardiogram revealed normal left ventricle size, with normal left ventricular ejection fraction; moderate pulmonary hypertension was present, and there was no pericardial effusion. Spirometry and flow volume loop revealed moderate obstruction without significant change following albuterol (FEV1 53%). Diffusion capacity was normal at 84% of predicted capacity, and when corrected for alveolar volume, it was elevated at 134%. This, in combination with the CT scan, argued against his previous COPD diagnosis. He was trialed on fluticasone furoate, umeclidinium, and vilanterol inhalation powder. However, in January 2020, he had not improved and a lung biopsy was performed to solidify a diagnosis. The biopsy showed numerous nonnecrotizing, mostly well-formed granulomas in bronchocentric distribution. These granulomas were composed of multinucleated cells, epithelioid histiocytes, and small lymphocytes. There also were some poorly formed granulomas with histiocytes, lymphocytes, and occasional multinucleated cells. Histochemical stains for acid-fast bacilli (AFB) and for fungal organisms were negative (Figure). AFB culture grew *Mycobacterium avium*.

The patient was advised to avoid using his hot tub in late January and was treated with daily prednisone (20 mg). He was not given antimycobacterial medications. His repeat CT scan 2 months later (see Figure) showed improvement in his ground glass opacities, and he was able to come off of home oxygen and had marked clinical improvement.

Figure. Patient's Computed Tomography (CT) Scans and Biopsy Images



Initial CT scan (upper left) and 2 months after hot tub avoidance (upper right). Biopsy showing numerous nonnecrotizing, mostly well-formed granulomas in bronchocentric distribution (lower left), poorly formed granulomas with histiocytes, lymphocytes, and occasional multinucleated cells (lower right).

Table. States With Reported Cases of Hot Tub Lung (N=42)

State	N (%)
Minnesota	21 (50)
Florida	10 (24)
Illinois	4 (10)
Massachusetts	2 (5)
Pennsylvania	2 (5)
Vermont	1 (2)
Georgia	1 (2)
California	1 (2)

DISCUSSION

Nontuberculous mycobacteria (NTM) are ubiquitous environmental organisms found mostly in soil and water and cause lung, sinus, lymph node, joint, central nervous system, catheter-related, and disseminated infections in susceptible individuals.[1] Recently, an NTM pulmonary disease syndrome with a presentation similar to hypersensitivity lung disease has been recognized and called "hot tub lung." The first case of hot tub lung was reported in 1997,[2] with other reports made in North America, Europe, Japan, and New Zealand. See Table.

Hot tub lung is described like a hypersensitivity pneumonitis (HP) in patients who are exposed to water aerosols of hot tubs contaminated with *Mycobacterium avium* complex (MAC). The association of NTM-HP to exposure to *Mycobacterium fortuitum*[3] and *Mycobacterium phocaicum*.[4] had been described, but is uncommon.

The following characteristics of hot tubs promote growth of MAC:

- NTM can growth in a wide range of temperatures; the optimal temperature for most nontuberculous mycobacteria cultures is 28 °C to 37 °C.[1] However, the growth of MAC is not inhibited by temperature as high as 42 °C, which is above most hot tub temperatures.[5]
- MAC is approximately 1000 times more resistant to chlorine than *Escherichia coli*.[6] The combination of poor hot tub hygiene and maintenance can presumably lead to the development of NTM-HP.[7]

A diagnosis is difficult to achieve based on 1 clinical parameter. Correlation of clinical history, images, and pathologic and microbiologic findings are necessary to establish a diagnosis of hot tub

lung.[8] Diagnostic criteria of hot tub lung include (1) persistent respiratory symptoms; (2) diffuse lung infiltrates on chest radiography or CT; (3) exposure to hot tub before onset of illness; (4) MAC isolated from respiratory secretion, hot tub water sample, or lung tissue biopsy; and (5) no other identifiable cause for the illness.[9]

In contrast, patients with NTM pulmonary disease have respiratory symptoms, such as productive cough and shortness of breath, and/or systemic symptoms, such as fatigue, night sweats, and weight loss. NTM typically occur in patients with baseline pulmonary disease such as COPD or bronchiectasis. Imaging findings are nodular or cavitary in nature on high-resolution CT. Microbiologic diagnosis is needed for NTM and includes positive cultures from 2 separate sputum samples, 1 positive bronchial alveolar lavage or transbronchial or other lung biopsy with both histologic and microbiologic findings suggestive of NTM. Patients with NTM are typically treated with a 3-drug regimen consisting of a macrolide, rifamycin, and ethambutol for 12 to 18 months.[10]

The most common symptom in NTM-HP is dyspnea, followed by cough and fever. Unintentional weight loss, chest tightness, and other nonspecific symptoms are less common.[11] In the largest clinical case series of 21 NTM-HP patients in Minnesota, the duration between hot tub exposure and symptom onset was highly variable, from 2 months to 17 years (median, 26 months). Furthermore, the median duration of symptoms prior to diagnosis was 5 months (range 1-54 months). In addition to the clinical findings, this study reported variable results in the pulmonary tests: 10% had normal pulmonary function results, an obstructive pattern was seen in 29%, restrictive pattern in 19%, isolated reduction in diffusing capacity for carbon monoxide was found in 19%, and no specific pattern was found in 24%.[9]

There is little information on why patients develop NTM-HP. One study demonstrated that MAC isolated from a patient with hot tub lung disease brought about HP-like inflammatory responses in exposed mice. This suggests that there may be an immunostimulatory aspect of inhaled mycobacterial particles that are important pathogenic factors of HP.[12] The susceptibility of the host and duration and intensity of the exposure are other potential etiologies of NTM-HP, but studies in the areas where the NTM-HP had been reported are necessary to clarify the factors associated with it.

Our patient showed diffuse bilateral ground glass opacities. This image and nodular opacities with diffuse centrilobular distribution are the most frequently reported in patients with NTM-HP. Chest CT showed opacities in 100% of the patients with NTM-HP, which is more sensitive than chest x ray, which showed abnormalities in 80% of the cases.[13]

The most prominent histopathology finding in NTM-HP is the presence of multiple, non-necrotizing well-formed granulomas in bronchocentric distribution.[9] The main diffrential diagnosis includes sarcoidosis and hypersensitivity pneumonitis. Hypersensitivity pneumonitis typically has mostly poorly formed granulomas. The recovery of MAI and absence of mediastinal adenopathy make a diagnosis of sarcoidosis less likely.

In the majority of cases, the clinical and radiographic findings improve with avoidance of hot tubs and the use of systemic corticosteroid.[9] This suggests that NTM-HP acts more like hypersensitivity pneumonitis than an infectious disease. Furthermore, antituberculous therapy has not shown clear benefit and might expose individuals to unnecessary adverse drugs events and is, therefore, not recommended. Our patient was treated without antituberculous therapy and showed clinical and radiographic improvement after 6 months of hot tub abstinence.

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