"Granulomatous Tenosynovitis of the Hand Due to Mycobacterium kansasii in an Immune-Competent Host"

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Introduction

M. kansasii was first described as causing human disease in 1953 [1]. M. kansasii is a nontuberculous mycobacterium found in many places such as soil and various water sources; however, it is almost exclusively recovered from municipal sources of water as opposed to environmental sources [2]. M. kansasii, though relatively rarely seen, is the second most common cause of nontuberculous mycobacterial (NTM) disease in the United States [3]. To culture, M. kansasii an acid fast culture is used with a Lowenstein-Jensen medium which limits growth to Mycobacterium species [4]. Regarding its classification, it is considered a "photochromogen" (Runyon I), meaning it is non-pigmented when grown in the dark but produces a yellow-orange pigment when exposed to bright light. Colonies that do grow will appear rough upon culturing. Beyond culturing, another way to positively identify M. kansasii is via Gen Probe which has a 100% specificity and 85-100% sensitivity via detection of the 16s RNA of the mycobacterium [2].

Regarding M. kansasii, infection in humans typically presents as pulmonary disease in immunocompromised patients [1]. The most commonly affected demographic is middle-aged white men especially in the presence of other risks factors including chronic lung disease, i.e., pneumoconiosis, COPD, and previous mycobacterial pulmonary disease; malignancy; alcoholism; and immunodeficiency [2]. When M. kansasii causes disease, it presents in a way nearly identical to tuberculosis [5]. As such, symptoms often include fever, weight loss, night sweats, hemoptysis, etc. [2]. Furthermore imaging often shows cavitary infiltrates with upper lobe predilection; nodular/bronchiectatic lung disease, mostly in the middle and lower lobes; and pleural effusions [2].

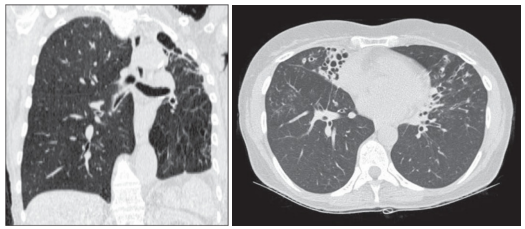

Figure 1 (left): Depicted is a CT scan of an M. kansasii pulmonary infection showing reticular opacities and volume loss [2].
Figure 2 (right): Depicted is a CT scan of an M. kansasii pulmonary infection showing bronchiectasis, atelectasis, and nodules [2].

Diagnosis of an M. kansasii infection is made based on symptoms, radiographic findings (i.e., pulmonary disease) or pathologic findings, and microbiologic findings [2]. There are numerous manifestations of M. kansasii on pathology. These include abscesses; granulomas without giant cells but with large areas of central, eosinophilic necrosis with numerous neutrophils and nuclear debris; well-organized granulomas without giant cells or necrosis but with a mononuclear cell infiltrate; areas of eosinophilic, granular necrosis with scattered clusters of epithelioid histiocytes; and spindle-cell proliferations with scattered clusters of neutrophils [3]. Musculoskeletal manifestations of M. kansasii are incredibly rare and can present in many forms. Granulomatous synovitis due to M. kansasii is rarely described, mentioned in only a few case reports in the literature, and is typically described in chronically immunosuppressed patients (e.g., those diagnosed with rheumatoid arthritis or having received a transplant) [1]. Granulomatous synovitis due to M. kansasii is challenging to both diagnose and manage, as it often requires a prolonged course of combination antimicrobial therapy with multiple surgical interventions/reconstruction [2]. A case and systematic review of 26 cases of granulomatous synovitis showed that the best results were treatment via a combined surgical and chemotherapeutic approach with the duration of antibiotics varying greatly (3-18 months) [6].

Patient History

The patient for this case study was a 50-year-old man who first went to an orthopedic clinic in 2019 with a small bump on the dorsal side of his left wrist, and he recalled no initiating injury to his left wrist. Regarding social history, the patient worked in construction and landscaping since his high school years. He worked at various types of construction sites including post-Hurricane Katrina sites dedicated to rebuilding damaged homes. The patient's main method of transportation was via bike, and though he had occasionally fallen he recalled no significant injuries. Upon review of medical history, he indicated a surgical history of an uneventful right knee arthroscopy in 2007. He also had a history of chronic Hepatitis C which was treated with Epclusa a year prior in 2018. An ultrasound completed in 2017 was concerning for cirrhosis; however a follow-up scan indicated an elastography fibrosis score of F0-1 indicating little-to-no fibrosis. In addition to hepatitis C, the patient also had a history of bipolar II disorder and depression. Though, the patient was not currently under or seeking psychiatric treatment because the patient attributed much of his past psychiatric symptoms to be due to past drug use. The patient had a history of IV drug use and alcohol abuse which ceased two years prior in 2017. He reported previous use of heroin, cocaine, and methamphetamines. He also reported often using dirty or shared needles. For this, he was being treated with suboxone 8 mg/2 mg three times a day at the time of his presentation. The patient also reported a 60 pack-year smoking history which ceased in 2020, about a year after his initial presentation. For this, he was taking bupropion 300 mg daily. Regarding family history, the patient's father had been diagnosed with gout.

Initial Presentation

The mass appeared spontaneously on the dorsal surface of the patient's left wrist. The patient reported that that mass felt like pressure in the area of his left wrist. The mass was typically not painful unless the patient performed repetitive hand movements which caused some soreness. Furthermore the mass was not erythematous or associated with draining. An MRI (figure 3) was ordered which confirmed the diagnosis of tenosynovitis. A tenosynovectomy was scheduled, but the mass spontaneously resolved causing the patient to not have the surgery.

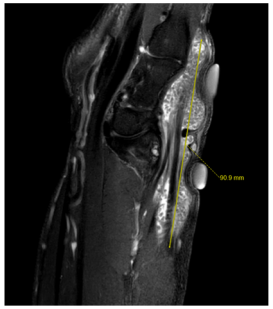

Figure 3: A T2 MRI, sagittal view of the left wrist shows as reported "Findings consistent with tenosynovitis of the extensor compartment with rice bodies. Rice bodies are multiple, small loose bodies that macroscopically resemble polished grains of rice. Seen in synovial fluid or bursae. Unclear pathogenesis. Possibly due to shedding of infarcted synovium, or may develop independently as a part of the inflammatory process and is subsequently encased by fibrin. Differential includes any chronic synovitis such as rheumatoid, tuberculous, etc. Dorsum is to the right, edema is of the extensor tendons of the fourth and fifth digits."

Second Presentation

In March 2020, the mass on the dorsal surface of the patient's left wrist reappeared. This time it was larger, and a second mass developed directly adjacent to the first. Again it was not tender, erythematous, or productive of drainage. The pressure and soreness began to interfere with his ability to work which prompted him to seek care from orthopedics. The patient denied fever, chills, weight loss, or night sweats. The patient had no rashes or other joint pain. He did have a chronic, non-productive cough, which he attributed to past tobacco use. The patient was right-handed and had injected IV drugs into his left hand numerous times; however, he did not remember a specific trauma or infection in that area. A chest X-ray was ordered and showed no acute intrathoracic processes or acute osseous abnormalities making the x-ray overall unremarkable.

Treatment

Upon reappearance and growth of the mass, a tenosynovectomy was scheduled and performed. Surgical pathology reports indicated, "Fragments of benign tenosynovium with multifocal fibrin deposits along partially denuded synovial lining, subepithelial edema, subepithelial chronic inflammation with infiltrate of small lymphocytes and plasma cells, and focal loose aggregate of histiocytes suspicious for non-caseating granulomatous inflammation." Various cultures were performed on the surgical findings. Aerobic and anaerobic cultures appeared negative, as did fungal cultures. However, acid fast bacterial (AFB) smear on Lowenstein-Jensen medium was positive. A Gen Probe was used for final identification of Mycobacterium kansasii based on the release of target 16S rRNA from the organism [2].

Currently, there are no recommendations from the American Thoracic Society (ATS) for skeletal M. kansasii infection. There are, however, Infectious Disease Society of America (IDSA) recommendations for M. kansasii pulmonary disease. According to IDSA, "In patients with rifampicin-susceptible M. kansasii pulmonary disease, we suggest a regimen of rifampicin, ethambutol, and either isoniazid or macrolide (conditional recommendation, very low certainty in estimates of effect)" [7]. As well, there are ATS recommendations for a localized, skeletal mycobacterium avium complex (MAC) infection: "a combination of excisional surgery (or surgical debridement) and chemotherapy," along with the same drug regimen recommended for MAC pulmonary disease though optimal treatment duration and regimen is unknown [2].

Conclusions

For our patient's treatment plan, we adapted the ATS recommendations for skeletal MAC infection, as there were no recommendations for skeletal M. kansasii infection. As with many patients, choice of antibiotic therapy is complicated by concerns for resistance, drug-drug interactions, and side effect profiles. Susceptibilities have been sent and are still pending; ATS recommends testing only for rifampin resistance. Rifampin will likely interact with this patient's suboxone causing decreased efficacy. Due to the patient's history of liver disease, we opted for macrolide as opposed to isoniazid. Repeated HIV testing has been and will be performed, with negative results thus far, and AFB blood cultures have been and will be ordered to check for disseminated disease. Empiric rifampicin, azithromycin, and ethambutol have been prescribed, pending susceptibilities. We will also trend his ESR, CRP, and CMP monthly to monitor liver enzymes (given his history of hepatitis C and possible cirrhosis).

In summary, this patient presented with a rare, solely musculoskeletal manifestation of an M. kansasii infection. This was diagnosed via both appropriate AFB culturing and Gen probe. Likely due to the rarity of this course of presentation, no suggested treatment plan was available, so the skeletal MAC infection recommendations were adapted to treat the patient.

Sources

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