Open Journal of
Clinical & Medical
Case Reports

Volume 5 (2019)
Issue 17

ISSN: 2379-1039

A rare case of mycobacterial kansasii infection with atypical presentation in a newly diagnosed HIV infection

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Abstract

Mycobacterial kansasii together with Mycobacterium avium complex are the most popular non tuberculous mycobacterial infection in the US. Most clinical cases occurs in immunocompromized patients. It was more common in HIV infection prior to the effective Highly Active Antiretroviral Therapy (HAART) regimen. Since starting the HAART regimen for HIV patients, M. kansasii becomes rarer. We report M. Kansassi infection in a newly diagnosed HIV patient presenting with atypical symptoms.

Keywords

Mycobacterial kansasii; HIV; AFB

Case Presentation

A 48-year-old man with unknown PMH brought to the Emergency Department (ED) for an episode of seizure. Physical examination was irrelevant. Laboratory workup was only significant for leucocytosis. Chest X ray showed left lower lobe consolidation. EKG demonstrated sinus tachycardia. Urinalysis was without evidence of urinary tract infection (UTI). CT head demonstrated findings suspicious for old infarcts within the anterior and posterior right parietal and left temporal lobe. EEG was normal and required no medical management. Cerebrospinal fluid (CSF) analysis didn't show any signs of infection.

Because of the low O_2 saturation in the low 90s, CT angiography was done and showed multiple consolidation in the superior segments of the left lower lobe. With suspected cavitation.

Urine histoplasma, blastomyces, pneumococcal, and legionella Antigens were negative. Patient started treatment for community acquired pneumonia (CAP), however his clinical course worsened. Procalcitonin was low on two separate occasions, and atypical pulmonary process was suspected, so the antibiotics were stopped.

Bronchoscopy was done and sputum AFB was positive. The patient was started on Rifampicin, Isoniazid, Pyrizanimide and Ethambutol (RIPE regimen). Later, Quinteferon was negative but the patient was positive for HIV type 1, CD4 count was < 20.

Per micro lab, respiratory AFB grew after one day in M.Bactec bottle, the microscopic appearance was not suggestive of M. Tubeculosis as it has elongated structure. After 1 month, the AFB isolate was determinate to be M. Kansassi (Figure 1), and the patient responded to HAART regimen, rifampicin, Isoniazide, and ethambutol for 18 months.

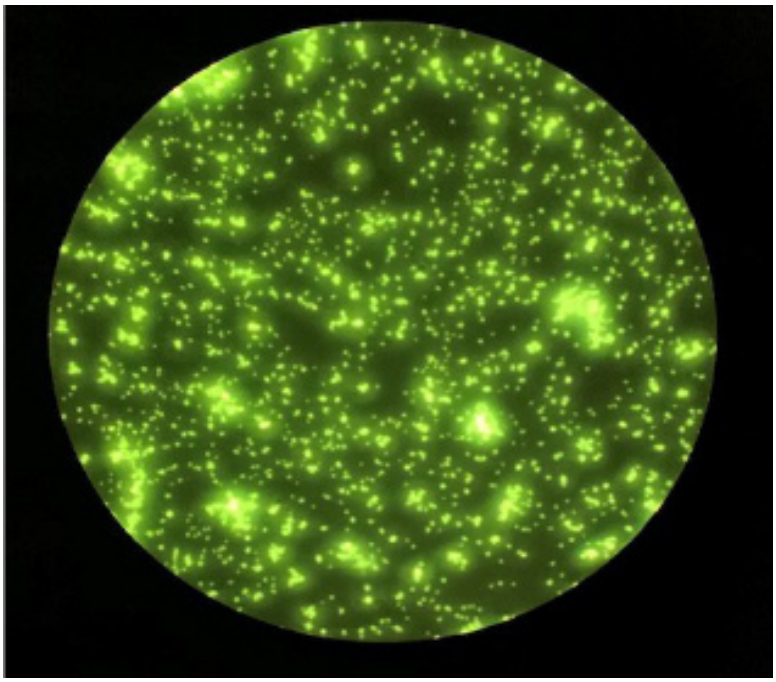

Figure 1: Acid fast isolate showing Mycobateria Kansasii

Discussion

The regional domain of Mycobacterium kansasii is not well known. However, it shows to be active around the central prairie states such as Illinois and northern Texas and along the southeastern and southern coastal region states such as Louisiana and Florida [1,7].

Unlike the Non Tuberculous Mycobacteriae, M. kansasii has never been found in soil or in natural water supplies. It is isolated from tap water in cities and concentrated in urban areas [11].

A microbiology lab survey performed between 1992 and 1996 in northern california identified that M. kansasii infection incidence was found to be 2.4 cases per 100,000 adults annually [2]. There were a total number of 270 cases, of which 187, roughly 69% were HIV positive, 33 HIV negative and 50 with unknown HIV status. The individuals from which M. kansasii respiratory specimens were isolated had a symptomatic illness. These individuals were also associated with homelessness and lower socioeconomic status. With relation to CD4 count, a case-control study conducted in Spain showed that while the clinical symptoms and dissemination rates were about the same, the patients with M. kansasii infection had lower CD4 cell counts than M. tuberculosis [8].

There are several risk factors associated with M. kansasii infection. Some of them are associated with structural pre-existing lung disease like COPD, and others are associated with immunocompromise of the host including alcohol abuse, malignancy, immunosuppressive drugs, and HIV infection [3-6]. Some occupations also are at an increased risk as painters, welders, miners, and sandblasters [3].

Our patient has underlying lung disease as CT chest was suggestive, giving the long history of smoking and alcohol drinking that we knew later, and it is likely having advanced HIV infection (AIDS), all of these risk factors made our patient at higher risk for the rare infection of M. Kansasii.

The standard therapy for pulmonary M. Kansassi infection is Isoniazid, Rifampin, and Ethambutol. Unlike MTB, Pyrizinamide is not effective against M. Kansasii. The treatment is carried out for 1 year after negative sputum culture is obtained [9]. Newer Regimen by replacing INH with macrolides, quinolones, or trimethoprim-sulfamethoxazole have shown efficacy in the management of M. Kansassi infection as well [10].

Our patient was on the previous regimen for a year, was following up in the HIV clinic and infectious disease specialist was following till completing the full course of antibiotics.

Conclusion

M. kansasii is a considerably less virulent organism than M. tuberculosis, but clinical symptoms and radiographs are quite similar. However, M. kansasii has shown to have a higher incidence of disseminated infection, and extrapulmonary involvement as pericarditis, bacteremia, and osteomyelitis as patients usually are immunocompromised.

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Manuscript Information: Received: July 07, 2019; Accepted: November 04, 2019; Published: November 29, 2019

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Citation: Shaker M, Bose A, Shehata M, Vogel N. A rare case of mycobacterial kansasii infection with atypical presentation in a newly diagnosed HIV infection. Open J Clin Med Case Rep. 2019; 1600.

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