Case Report

Cutaneous *Mycobacterium kansasii* infection in a patient with AIDS post initiation of antiretroviral therapy

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Abstract

The HIV pandemic has resulted in unique clinical presentations in patients, and their diagnosis and management pose challenges to physicians in the developing world. Due to limited resources and difficulties in laboratory diagnosis, most physicians treat according to the most likely etiological agent that might be causing the disease. In South Africa, when acid-fast bacilli are detected, anti-tuberculous treatment is commenced. However, it must be realized that not all acid-fast bacilli are *Mycobacterium tuberculosis*, and that there are nontuberculous mycobacteria that can cause infections. Clinicians should work closely with the medical microbiologist when unique cases arise to ensure optimal microbial detection, identification, and patient management. This paper describes a very rare case of self-resolving cutaneous *Mycobacterium kansasii* infection following the initiation of antiretroviral therapy and potentially associated with immune reconstitution inflammatory syndrome.

Key words: HIV; AIDS; IRIS; *Mycobacterium kansasii*

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Introduction

In South Africa, the presence of acid-fast bacilli (AFB) in any tissue specimen implies *Mycobacterium tuberculosis* (MTB) and usually mandates the initiation of anti-tuberculosis (TB) therapy. However, not all AFBs are MTB as nontuberculous mycobacteria (NTM) also stain AFB-positive. The high incidence of human immunodeficiency virus (HIV) infection has led to a concomitant rise in NTM infection [1]. We present a rare case of cutaneous NTM infection in an HIV-positive patient after the commencement of highly active antiretroviral therapy (HAART - which is a combination of three or more antiretroviral medications), and document its spontaneous resolution.

Case report

A 33-year-old HIV infected female with a CD4 count of 147 cells/µl and a background history of pulmonary cryptococcosis on treatment, as well as squamous cell carcinoma of the right eye, presented with a small pustule on the right upper hip posteriorly, with a few smaller non pustular papules extending to the posterior aspect of the knee. The patient had not received any injections nor had there been any trauma at the site of the lesions. The patient did not have respiratory complaints and the rest of the general and systemic examination was normal. Chest radiograph did not reveal any new infective changes since the diagnosis of pulmonary cryptococcosis.

The patient received HAART approximately four weeks prior to the eruption of the skin lesions. The lesions were treated conservatively with povidone-iodine paste and the patient was reviewed one month later. The pustule was now draining pus and a swab of the exudate was taken. At follow-up a further one month later, the lesions had ulcerated and were healing. The satellite lesions had also disappeared. The direct microscopy for acid-fast bacilli was negative and the routine culture of the pus swab was negative. However, the TB culture using the BACTEC MGIT 960 (BD, Franklin Lakes, USA) detected acid-fast bacilli after 19 days incubation. Due to the spontaneous resolution of the lesions, anti-TB therapy was deferred pending the final identification of the microorganism cultured.

The patient had made a dramatic improvement over a three-month period with a small healing ulcer where the initial pustule was situated. The final culture results were obtained 25 days after the initial positive culture result. The microorganism was identified as *Mycobacterium kansasii* with the Genotype Mycobacterium CM (Hain Lifescience, Nehren, Denmark). The patient had been treated with only fluconazole prophylaxis and HAART.

Due to the unique clinical presentation and the organism cultured, a punch biopsy of the lesion was obtained, even though it was healing. The histology demonstrated fibrosis, scarring, and non specific inflammation. There was more intense inflammation at the deep margin with associated multinucleated giant cells. No organisms were detected on Periodic acid-Schiff and Ziehl-Nielsen stains. TB culture of the biopsy, with the BACTEC MGIT 960 was negative after 43 days of incubation.

The skin lesion had completely healed approximately five months after the initial presentation. There was a documented improvement of the CD4 count, which increased from 147 cells/μl to 321 cells/μl. Due to limited resources and budget constraints, the CD4 count was repeated after six months on ARV's.

Discussion

M. kansasii is a slow growing, photochromogenic nontuberculous *Mycobacterium* which when exposed to light produces a yellow pigment [2]. The microorganism has five subtypes. It is an environmental pathogen most commonly located in freshwater sources [2,3]. *M. kansasii* is not transmitted from person to person and infection is always from the environment [2,3,4].

M. kansasii is the most virulent NTM and is both microbiologically and clinically similar to MTB and its presentation and features are similar to tuberculosis [4]. The disease manifestations are primarily bronchopulmonary, which occurs in patients with pre-existing lung disease [1]. *M. kansasii* can also cause meningitis, arthritis, tenosynovitis, osteomyelitis, lymphadenitis as well as disseminated disease, primarily in immunosuppressed individuals [1,5]. Skin disease is an uncommon manifestation and tends to occur in disseminated disease in immunosuppressed patients; isolated skin disease due to *M. kansasii* is less frequently observed [2,4,6,7]. The clinical presentation in the HIV infected population is variable with atypical clinical and histological features [3,4]. Clinically, such patients may present with sporotrichoid nodules, erythematous plaques, papules, pustules, verrucous plaques or abscesses [2,3,4,6]. Histological evaluation does not always reveal granulomas in immunosuppressed patients and specimens may not stain positive for AFB [3].

The detection of *M. kansasii* is rarely a contaminant and its detection generally indicates infection, as evident in our patient [1,4,6]. The presentation and the resolution of infection in this case are unusual. We postulate that the cause and subsequent resolution of the lesions is due to an immune reconstitution inflammatory syndrome (IRIS) phenomenon. The skin eruption occurred approximately one month after commencing HAART and resolved after three months of treatment. It is likely that the infection had resolved spontaneously with improvement in immune function. There have been documented cases of IRIS caused by *M. kansasii* in the skin and the lungs [8,9].

The treatment of cutaneous *M. kansasii* infection is the same as that for pulmonary disease and multisystemic disease. The treatment regimen includes rifampin 600 mg daily, isoniazid 300 mg daily and ethambutol 15 mg/kilogram daily but may need to be adjusted in patients on HAART [10]. Untreated lesions tend to have a slowly progressive course [3,4]; however, it has been documented that skin lesions can resolve without any specific treatment [6].

This case highlights important aspects in the management of HIV positive patients with rare dermatological manifestations. Clinicians in developing countries must be cognizant of the fact that although AFB positive specimens may suggest MTB in an endemic setting with a high HIV prevalence, NTM should also be considered. Anti-tuberculous therapy should not be instituted routinely in HIV positive patents on HAART as this increases their pill burden and may cause unnecessary adverse effects.

Close clinical follow-up and the identification of the mycobacterial species are beneficial in this subset of patients. A multi-disciplinary team, which includes a medical microbiologist, is necessary to optimize diagnosis and treatment. This case also highlights that the unusual presentations of IRIS can include infections with NTM as well as other pathogens. These infections might heal spontaneously with an improvement in the host's immune function.

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