A Possible Case of Spinal Tuberculosis in a HIV-Positive Male

The Editor

Sir,

A 37-year old male who was known to be HIV-positive for two years, presented with a three and a half month history of lower back pain that increased in intensity over the prior three weeks. He described the pain as severe, aggravated by movement and radiating around the waist. There were no known factors that could relieve the pain, he had constitutional symptoms: fever and a 30-pound weight loss over the prior three months. He had no recent contact with anyone known to have tuberculosis and he had no history of intravenous drug use or recent incarceration. He denied having any bladder or bowel dysfunction and there were no pulmonary lower extremity motor or sensory symptoms. He has been on antiretroviral therapy (ARV) for the past two years but has been non-compliant. His ARV regime consisted of efavirenz and truvada. His physical examination revealed pallor, mild cachexia without generalised lymphadenopathy or oral candidiasis. He had normal chest findings. There were no focal deficits involving his motor and sensory systems. His haemoglobin level was 5.6 g/dL and his white blood cell count 4.3 x 10^9 /L. His ESR was 130 mm/hr. His CD4 cell count was 17 cells/ml, but no viral load assessment was attained. His chest radiograph was normal and one out of three early morning sputum samples was positive for atypical acid fast bacilli. Tuberculin skin testing was done and showed no reaction. His MRI (Fig 1–4) revealed collapse of the L2/L3 disc space with sclerotic adjacent bone edges. A small epidural collection was seen spanning L2 to L3 which did not appear to be compressing any surrounding structures. A small anterior collection at the same level was also visualized and there was no significant canal compromise noted. An open bone biopsy was performed to confirm the diagnosis and to isolate the organism for drug susceptibility testing. Unfortunately, the sample was deemed inadequate for histopathologic evaluation and no pathogens were isolated.

On the basis of his clinical presentation and MRI, a diagnosis of spinal tuberculosis was made. He was started on a four drug anti-tuberculosis regime (rifampin, isoniazid, ethambutol and pyrazinamide) for two months with the plan to subsequently continue rifampin and isoniazid for ten to twelve months. He was continued on his first line regime of truvada and efavirenz with the goal of eventually switching to a protease inhibitor based regime.

DISCUSSION

Extra-pulmonary tuberculosis has a higher incidence in HIV-positive patients (56.5%) than in HIV-negative patients [35.7%] (1). Skeletal tuberculosis accounts for approximately 35% of cases of extra-pulmonary tuberculosis (2).

Microbiologic and histological confirmation via a biopsy or aspirate should be done. This will confirm the diagnosis as well as provide information on drug resistant strains. This is done usually via CT guided biopsy or percutaneous needle aspiration/biopsy. In this index case, an open
bone biopsy was chosen as the method of sampling. Unfortunately, a sufficient sample was not obtained for evaluation. Magnetic Resonance Imaging (MRI) is currently the best diagnostic tool for spinal TB (3–6). It is more sensitive than plain radiographs and more specific than CT in the diagnosis of spinal TB. The soft tissue and disc involvement yields greater specificity with the MRI. Magnetic resonance imaging can also provide the diagnosis of TB of the spine four to six months earlier than conventional methods, offering the benefits of earlier detection and treatment (6).

The World Health Organization (WHO) recommends, for first line treatment of tuberculosis in HIV co-infected patients, a quadruple regime including rifampicin. Rifampicin containing regimes have been associated with a decreased risk of treatment failure and relapse (7, 8).

In contrast to the HIV-negative population, HIV co-infected patients should receive their anti-TB medication on a daily basis and not on reduced frequency regimes such as once or twice weekly dosing. These reduced frequency regimes have been associated with an increased risk of rifampicin associated resistance (9, 10). The length of time for which treatment should be continued has not been clearly proven and hence there remains some ambiguity on the issue (11, 12). Short-course anti-tuberculous therapy can be an appropriate option in select patients who are HIV co-infected, however, longer periods of therapy have been recommended for patients with spinal and extra-pulmonary disease, extending up to 12 months (12, 13).

The co-administration of ARVs and anti-TB therapy in these patients also presents some challenges. The timing of commencement of ARV therapy in TB co-infected patients remains a somewhat controversial issue, although a couple of recent studies have shown improved outcomes in patients who commence antiretrovirals within the initial two months of anti-TB therapy (24). The options of ARVs to be included in this regime are significantly affected by the presence of rifampicin. Rifampicin induces the activity of the cytochrome P450 system, particularly the CYP3A4 isoenzyme. This is the common pathway for the metabolism of many ARVs and hence great limitation is placed on the available options. Recommended ARVs include an efavirenz-based regime or a double boosted protease inhibitor regime. Triple Nucleoside Reverse transcriptase regime is reserved for those patients in whom efavirenz is contraindicated (15).

**Key words:** Antiretrovirals, HIV, tuberculosis.

**REFERENCES**