Spinal Epidural Abscess Due to *Mycobacterium tuberculosis* in a Patient with AIDS: Case Report and Review of the Literature

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Spinal epidural abscess (SEA) is a rare infectious disorder that often has delayed diagnosis and is associated with significant morbidity and mortality rates. We present a case of an AIDS patient with a SEA due to *Mycobacterium tuberculosis*. This type of SEA in AIDS patients is characterized by localized spinal pain and prolonged fever. Magnetic resonance imaging is the method of choice in the diagnostic process. Early diagnosis, followed by specific therapy (surgical decompression combined with antituberculous drugs), is necessary to improve the prognosis of these kinds of patients. Key Words: Spinal epidural abscess, *Mycobacterium tuberculosis*, AIDS.

The AIDS epidemic in Latin America has resulted in a parallel increase of all forms of tuberculosis (TB), including spinal TB. Spinal epidural abscess (SEA) is a rare infectious disorder, often with a delayed diagnosis and associated with significant morbidity and mortality rates. SEA was first described in 1761. It is a severe pyogenic infection of the epidural space, which frequently requires neurosurgical intervention for etiological diagnosis in order to avoid permanent neurological sequelae [1].

We report a case of an AIDS patient who developed a SEA due to *Mycobacterium tuberculosis*.

Case Report

A 35 year-old homosexual man, infected with the human immunodeficiency virus (HIV), with persistent fever and thoracic back pain that lasted for three months was admitted to our AIDS division. He had developed lower extremity weakness and anal and bladder sphincter dysfunction for 15 days. On admission he was alert and oriented; neurological examination revealed signs of spinal cord compression, with spastic paraparesis and sensitivity at the thoracic level at D7-D8. Relevant laboratory findings were: hemoglobin 14 g/dL, hematocrit 40%, erythrosedimentation rate 76 mm/1st h, white blood cells 8.6 x 10³/μL, platelets 329,000 /mm³, alkaline phosphatase 474 U/L. A lumbar puncture was performed; cerebrospinal fluid (CSF) revealed albumino/cytological dissociation. CSF smears were negative for pyogenic bacteria, fungi and parasites, Gram, Giemsa and Ziehl-Neelsen stains, VDRL and polymerase chain reaction (PCR) for Herpesvirus, as were the serological tests for toxoplasmosis, Chagas' disease and HTLV I-II infections. Sputum smear cultures were negative for microbial pathogens.

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Chest radiography was normal. The CD₄⁺T cell count was 156 cell/µL (10%), and the plasma viral load was 55,090 copies/mL (log₁₀ 4.8). Magnetic resonance imaging (MRI) showed contiguous multilevel involvement at D6-D8, with decreased signal intensity of vertebral bone marrow on T-1weighted images, intervertebral disk space compromise, multiple vertebral deformities, plus homogeneous tissue with paravertebral and prevertebral irregular contrast enhancement and epidural extension of the infectious process (Figures 1 and 2).

The patient underwent surgical decompression and debridement. Surgical findings were pus and granulation tissue in the epidural space, with spondylitis of the D7 and D8 vertebrae. The neurosurgical outcome was good.

Histopathological examination of the smears obtained during surgery revealed a granulomatous inflammation, with necrosis and acid-fast bacilli. Culture of the biopsy smears yielded *Mycobacterium tuberculosis* (definitive diagnosis). The patient went through two months of standard therapy for TB, based on isoniazid, rifampin, pyrazinamide and ethambutol, with a good clinical and neurological response. After one month of antituberculous therapy, highly active antiretroviral therapy (HAART), was initiated. Four months later, the CD₄T cell count was 358 cell/µL (14%) and the plasma RNA-HIV viral load was undetectable (< 50 copies/µL). A two drug (isoniazid plus rifampin) antituberculous treatment consolidation phase was maintained for seven months.

Discussion

TB osteitis of the spine (Pott's disease) accounts for 1% of all TB infections [2]. *Mycobacterium tuberculosis* infection generally spreads to the spine by the hematogenous route, or by paraspinal extension. Any level of the spine can be affected; but lesions are more commonly found in the lower thoracic region, as in our patient. The lumbar and cervical spine areas are less frequently affected. Two or three adjacent vertebral bodies can be involved, as in our patient, but noncontiguous sites of infection can also be found [3]. TB spondylitis is generally indolent, with gradual symptoms over months, as in

Figure 1. Coronal T1 weighted magnetic resonance imaging (MRI) imaging demonstrating involvement of T7-T8 vertebral bodies.

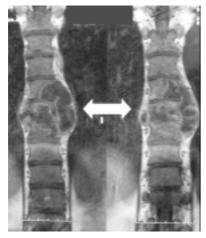


Figure 2. An axial T1 weighted MRI view gadolinium enhancement shows the spinal canal to be severely obstructed by granulomatous tissue and an abscess.

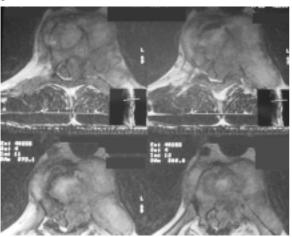
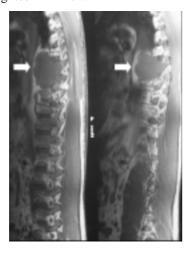


Figure 3. Destruction of the anterior and middle columns of the bodies with a large anterior epidural abscess is seen on a sagital T1 weighted MRI view.



our patient. Reihsaus et al. [1] reviewed 915 cases of SEA published in the medical literature until 2000. Seventy-one percent of the patients had back pain as an initial manifestation, while only 66% had fever. SEA is a rare and a late complication of spinal TB that compresses the spinal cord (Pott's paraplegia). Signs and symptoms are unspecific and include spinal pain, fever and night sweats as the most common clinical features at the time of diagnosis [4]. Only 10% to 20% of patients have the SEA diagnosed before the onset of neurological symptoms. After several days, there is a rapidly progressing paraparesis and paraplegia, associated with sensory loss in the lower extremities, sphincteric paralysis, and urinary and fecal retention, as also found in our patient. Percussion of the spine is associated with pain at the site of infection. Physical examination also reveals signs of a transverse cord lesion at the spinal level, also found in our patient. CSF examination yields a small number of white cells (usually fewer than 100/mm³) and a relatively high concentration of proteins, with normal glucose, as also seen in our patient.

SEA is primarily a bacterial infection, and *Staphylococcus aureus* is the most common etiological agent [5]. Koppel et al. [6] reported 18 cases of SEA with disk space infection, amongst which two were due to *Mycobacterium tuberculosis*. The two most-common pathogens found in 29 patients with a diagnosis of SEA who were analyzed by Lu et al. [4] were *Staphylococcus aureus* and *Mycobacterium tuberculosis* (62%).

A history of prolonged fever, night sweats, elevated peripheral white cell blood counts, and elevated erythrocyte sedimentation rates, is common to both spinal TB and pyogenic infections. The presence of pulmonary infiltrates on the chest radiography, evidence of immunodeficiency and a direct smear or culture sputum positive for acid-fast bacilli, is suggestive of TB. However, chest radiography may not reveal evidence of TB, as in our patient.

MRI is the method of choice for the diagnosis of SEA. Huang et al. [7] evaluated the MRI findings in 24 patients with a diagnosis of infectious spondylitis. Decreased signal intensity of the vertebral marrow at T1 was most frequent in pyogenic infections. Contiguous multilevel involvement was observed only in patients with tuberculous spondylitis, as in our case (Figure 3). SEA was found in 15 patients of Huang's series; MRI showed dense tissue, with homogeneous enhancement after gadolinium injection. In comparison with pyogenic abscesses, spondylitis and SEA due to Mycobacterium tuberculosis are characterized by a predilection for spinal deformity, subligamentous spread and contiguous multilevel involvement, as in our patient. Spinal TB should be distinguished from malignant metastatic lesions [8]. Metastases characteristically spare the disc space, and they may involve multiple noncontiguous vertebra, which does not occur with spinal TB. Paravertebral and epidural abscesses, and subligamentous spread, are more frequent in spinal TB [9,10].

SEA is a severe complication of spinal TB that requires urgent neurosurgical intervention (laminectomy), combined with specific antituberculous therapy. This is the treatment of choice in order to avoid neurological sequelae. Diabetes mellitus, alcoholism, intravenous drug abuse and HIV/AIDS disease are the most common risk factors to develop a SEA following spinal TB. Age, early diagnosis, and the degree of neurological involvement are the most important prognostic factors in patients with SEA. Osteomyelitis of the vertebral bodies, tuberculous abscesses, granulation tissue and pus or caseum were found in most cases, as was seen in our patient.

We conclude that SEA due to *Mycobacterium tuberculosis* affects AIDS patients, provoking localized spinal pain and prolonged fever. The method of choice in the diagnosis process is MRI. Early diagnosis, followed by specific therapy (surgical decompression combined with antituberculous drugs), is necessary to improve the prognosis of this kind of patient.

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