

Comorbidity Of Spinal Cord Neurocysticercosis And Tuberculosis In A HIV-Positive Patient

H Foyaca-Sibat, L Ibanez-Valdes

Citation

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Abstract

We report a HIV-positive patient on HAART who presented a spinal cord syndrome due to TB spine and an associated intramedullary neurocysticercosis confirmed by T-2 MRI and ELISA test on CSF. Patient improved remarkably with medical treatment and surgical procedure was done.

INTRODUCTION

As far as medical history is known, it is likely that arachnoiditis (ARC) was present in the spines of some Egyptian mummies (estimated to have been buried over 5,000 years ago) in whom typical lesions of spinal tuberculosis were found. This dreadful disease is characterized by longstanding inflammation of the two innermost layers of the sac surrounding the spinal cord (SC) which contains the cerebrospinal fluid (CSF), and not uncommonly expands into the nerve roots, and the cauda equina, occasionally deforming the dural sac by scarring. TB is the commonest infection associated with HIV/AIDS, but TB spine is not the common presentation. Despite the availability of effective preventive measures and chemotherapy, the prevalence of tuberculosis (TB) is increasing in the developing world and in much of the industrialized world as well. In developing countries the annual risk of tuberculosis infection in children is 2- 5 per cent. Nearly 8-20 per cent of the deaths caused by tuberculosis occur in children. Early diagnosis plays a vital role in control of TB. Although acid fast bacilli (AFB) microscopy, and conventional Lowenstein Jensen (L-J) culture remain the cornerstone for the diagnosis of TB, these traditional methods are either slow or their sensitivity is quite low.¹ Worldwide cestode infection "neurocysticercosis" is the commonest parasitic disease of the central nervous system; it may involve the brain parenchyma, the meninges or ventricles and, infrequently it involves the spinal cord. Involvement of spinal cord has variably been reported to be 1-5%² Other report its ranging from 0.7%-5.8%³ Between 1978 and 1989 only forty-five cases of intramedullary

cysticercosis have been reported in the literature, presenting as quadriplegia or paraplegia. Up to 1998 no more than 49 confirmed cases have been reported in the literature,⁶ but none have been reported in the conus medullaris region until 2003,⁷ Therefore the spinal forms of neurocysticercosis are rather rare. The more common presentation is the leptomeningea form. The current theory of downward migration of the parasites from the cerebral to the spinal subarachnoid space cannot explain primary spinal forms, and it is suggested that retrograde flow through the epidural vertebral veins provides an alternative route.⁸

Spinal cysticercosis can be either leptomeningeal (which responds like subarachnoid disease) or intramedullary. A review of 95 published cases of medullary cysticercosis since 1856 shows the incidence of this condition.⁹ As far we know this is the first report about comorbidity of TB spine and NCC spine in HIV-positive patient.

CASE REPORT

A 27-years-old female presented with burning pain on both lower limbs and gradual onset weakness for 6 months leading to inability to walk. She was diagnosed as HIV-positive since 2 years back and she was on HAART (Stavudine 30 mg. lamivudine 150 mg. efavirenz 600 mg.) for the past seven months. After five months of symptoms on lower limbs, she developed acute onset retention of urine with overflow incontinence along with constipation. By this time, she also complained of pins and needle sensation on both legs and the diagnosis of peripheral neuropathy HIV's related was made and then treated empirically with amitriptyline (25 mg po BD) and lamotrigine (200mg por

TDS) without improvement. In the next two weeks, she gradually became bedridden. she did not give a history of root pain, low backache, trauma to back, prolonged fever or claudicating pain, weight loss, anorexia, diabetes, tuberculosis or any opportunistic infection.

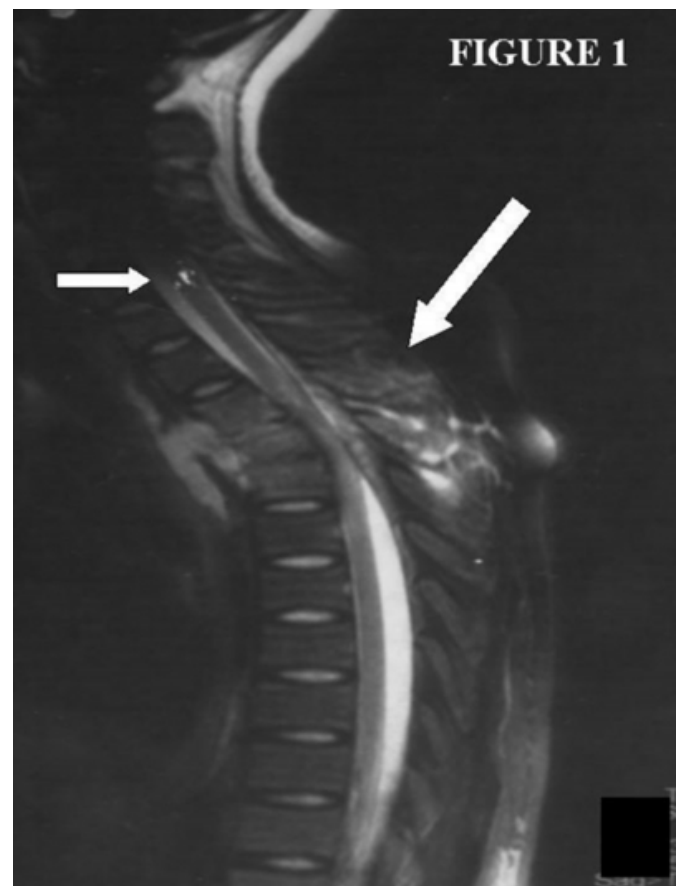
On clinical examination, she had a symmetric flaccid paraplegia with sensory bladder, bowel involvement, sensory level for light touch, pin-prick and signs of spinal shock. Spinal tenderness and mild kyphotic deformity were observed at the upper back. Generalized lymphadenopathy and oral candidiasis were confirmed. The rest of the general and systemic examinations were non-contributory.

She had a normal FBC, liver function test, urea and electrolytes, coagulation screen, and erythrocyte sedimentation rate, CD4 count level increased from 75 to $297 \times 10^6/L$ and viral load was undetectable. (Immune Reconstitution Syndrome?)

CT Scan of the head was normal. X-rays of the thoracic spine bone showed decrease intervertebral space with collapse of the vertebral bodies between T3/4, lumbosacral spine and chest were normal. Contrast magnetic resonance imaging from thoracic spine revealed a small cigar-shape semi-calcified lesion at C-7 surrounded by perilesional edema. Incomplete collapse of T3 vertebral body is noted with pathological signal at T2, T3, and T4 level. There is an anterior and paraspinal soft tissue component noted in association with elevation of the anterior longitudinal ligament in with sub ligament spread of infection. Disruption of the anterior longitudinal ligament with retro pharyngeal space infective collection is noted. Elevation of the posterior longitudinal ligament noted with impingement on the thecal sac and epidural fat. Pathological high signal is noted within the cord at this level in keeping with compressive myelopathy. A gentle kyphotic deformity is noted. Pathological signal in the posterior elements and para spinal muscle may be in keeping with infective change. A posterior epidural collection is noted to efface the posterior epidural fat and push the cord anteriorly in keeping with posterior epidural infection. The remaining vertebral bodies demonstrate normal signal intensity. A quantitative ELISA for immunodiagnosis of neurocysticercosis using CSF and purified fraction from *Taenia solium* cysticerci (PCF-ELISA) showed a strongly positive result. CSF routine and microscopy revealed normal findings with normal lactate dehydrogenase (LDH) and β micro globulin. CSF-ADA level was >15 units.

Figure 1: T-2 MRI shows: Features are in keeping with a Tuberculous involving T2,T3 and T4 vertebral bodies and intervertebral disc with sub ligamentus spread of disease anteriorly to the vertebral bodies in the pre and para vertebral spaces as well as in the retro pharyngeal space a disruption of the anterior longitudinal ligament. Anterior and posterior epidural collections are noted which effaces the cord and the cord demonstrates features of compressive myelopathy.

Figure 1



When treated with anti-TB medication (Rifapourin), prednisolone (1.5 mg/kg) for 3 days and one day treatment with praziquantel (3 tablets of 600mg each, 2 hourly 4 times), she showed a remarkable improvement after two weeks. Pain disappeared first and she started walking unsupported and regained continence of bladder and bowel. At the end of the third month of anti-TB treatment, she became normal with return of bladder/bowel sensations, decreased deep tendon reflexes, hypoalgesia on both lower limbs and 3/5 muscle power. Follow-up MRI studies revealed almost complete resolution of spinal cord lesion. No surgical intervention was done.

DISCUSSION

Ritesh Agrawal, in 2007, concluded that spinal intramedullary cysticercosis represents a diagnostic challenge and surgery is required to decompress the cord, confirm the diagnosis and provide a route for definitive therapy. Patient recovery may be variable. Despite promising reports, the safety and efficacy of medical treatment remains unproved.

Although spinal NCC is relatively rare, it represents a distinct clinical entity that can have devastating consequences for the patient. Because of the limited size of the spinal canal, the mass effect of these lesions is poorly tolerated. Most spinal NCC occurs in the subarachnoid space where mass effect can cause spinal cord compression, although obstruction of cerebrospinal fluid pathways due to scarring of the subarachnoid space can also cause symptoms, therefore most of patients need surgical management for spinal cord decompression (laminectomy) as soon as possible, but in places where surgical facilities are not always available, medical treatment can be another choice

References

1. Vijayasekaran D, Kumar RA, Gowrishankar NC, Nedunchelian K, Sethuraman S. Mantoux and contact positivity in tuberculosis. *Indian J Pediatr* 2006;73:989-993.
2. Garg RK, Nag D. Intramedullary spinal cysticercosis: response to albendazole: case reports and review of literature. *Spinal Cord*. 1998 ;36(1):67-70
3. Agakhani N, Comoy J, Tadie M, Lacro C. Isolated intramedullary cysticercosis case report. *Neurochirurgie* 1998;44:127-31.
4. Firemark HM. Spinal cysticercosis. *Arch Neurol* 1978;35:250-251
5. Venkataraniya NK, Jain VK, Das BS, Rao TV. Intramedullary cysticercosis. *Clin Neurol Neurosurg* 1989;91:337-41.
6. Escobar A, Herrera MP, Escobar W , Vega R Spinal intramedullary cysticercosis: A case report . *Neuropathology* 1998;18 (3):343-346.
7. Singh NN, Verma R, Pankaj BK, Misra S. Cauda-conus syndrome resulting from neurocysticercosis. *Neurol India* 2003;51(1):118-120
8. Bandres JC; White AC; Samo T; Murphy EC; Harris RL. Extraparenchymal neurocysticercosis: report of five cases and review of management. *Clin Infect Dis*;1992;15(5):799-811
9. Agrawal R, Chauhan SPS, Misra V, Singh PA, Gopal NN. Focal Spinal Intramedullary Cysticercosis. *The Internet Journal of Neurosurgery*. 2007. Volume 4 Number 1. Full text is available on the URL : <http://www.ispub.com/ostia/index.php?xmlFilePath=journals/ijns/vol4n1/focal.xml>

Author Information

H. Foyaca-Sibat

Department of Neurology, Nelson Mandela Academic Hospital, Faculty of Health Sciences. Walter Sisulu University

LdeF Ibanez-Valdes

Department of Neurology, Nelson Mandela Academic Hospital, Faculty of Health Sciences. Walter Sisulu University