Case Report

Fulminant holocord intramedullary tubercular abscess with enigmatic presentation

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Abstract

Background: Intramedullary and subarachnoidal tubercular abscesses are rare forms of spinal tuberculosis as compared with extradural collections secondary to vertebral tuberculosis.

Case Description: We herein present a 33-year-old, apparently healthy male patient who presented clinically as transverse myelitis, with a lesion at detected at conus cauda, developing fulminant holocord intramedullary tubercular abscess, treated with surgical evacuation and much later with anti-tubercular drugs. Atypical clinical, serological, imaging findings in addition to lack of knowledge of occurrence of fulminant intramedullary tuberculosis led to the delay in starting anti-tubercular treatment.

Conclusion: Early diagnosis requires a high index of suspicion, search for a primary focus of tubercular infection, investigation with magnetic resonance imaging (MRI) of spinal cord, biopsy, and confirmation with microscopy and culture, even in immunocompetent individuals. Early diagnosis, prompt treatment with surgical evacuation of abscess, and anti-tubercular drugs can lead to a good neurological recovery.

Key Words: Filum terminale, intramedullary, spinal tuberculosis, subarachnoidal, tubercular abscess

INTRODUCTION

Tuberculosis is a chronic bacterial disease caused by Mycobacterium tuberculosis, characterized by formation of granulomas and rarely by abscesses in immunocompetent individuals. Spinal involvement usually involves the vertebral bodies and secondarily compression of the thecal sac. Spinal intradural tuberculosis is very rare. A few cases of intramedullary and a few cases of subarachnoidal tuberculomas have been reported in the literature.[2,7,10,11,13,14,16,18,19] Less than 10 cases of intradural tubercular abscesses have been reported.[1,4,8,12] To the best of our knowledge, this is the second case report of a holocord intramedullary tubercular abscess, along with subarachnoidal locus of tubercular abscesses.

CASE REPORT

A 33-year-old male, apparently healthy manual laborer, presented with history of intermittent fever associated
with chills and rigors for 6 days and sudden onset of weakness in both the lower limbs, complete loss of sensations below the level of nipples and urinary retention for 5 days, prior to presentation to our hospital. There was no history of weight loss, loss of appetite, or chronic cough. He was febrile with a temperature of 100°F. The lower limbs were hypotonic with a power of 0/5, and absent deep tendon, superficial reflexes. Bilateral plantars were unelicitable. There was bilateral and complete loss of sensations to all modalities (touch, pin prick, temperature, and vibration) below D4 level. Perianal sensations were absent and anal sphincter was lax. Neck rigidity and Kernig’s sign were negative. There was no spinal tenderness or lymphadenopathy. On the basis of sudden onset of flaccid weakness following a brief period of febrile illness, a working diagnosis of acute transverse myelitis (probably viral) was considered and was investigated further.

Complete hemogram, random and fasting blood sugars, total leukocyte count (9700/mm³), erythrocyte sedimentation rate (ESR 4 mm in 1st hour), and X-ray chest were normal. Enzyme-linked immunosorbent assay (ELISA) for human immunodeficiency virus (HIV) was negative. Blood culture and urine cultures were negative. Cerebrospinal fluid (CSF) was positive for antiherpes simplex type I immunoglobulin G (IgG) antibodies, varicella zoster IgG antibodies, antidengue virus IgG and IgM antibodies, which was suggestive of para-infectious acute transverse myelitis. However, CSF microscopic examination showed plenty of polymorphs, reduced sugars, and elevated proteins, which were unusual for viral etiology. No organism was grown on CSF culture. Plain and gadolinium contrast enhanced magnetic resonance imaging (MRI) scans of cervical spine; dorsal spine and screening of lumbar spine was performed. The MRI images showed mild thickening of the spinal cord from C4 to D2 level with hyperintensities in the T2W images, with no enhancement after gadolinium injection [Figure 1a and b]. This radiological picture of central T2 hyperintensity of spinal cord spanning greater than three segments along with the clinical presentation of sudden onset paraplegia with a sensory level and early bladder involvement simulated acute transverse myelitis. But a closer look at the screening MRI of lumbar spine (sagittal sections) revealed a small mass isointense to cord on both T1W and T2W images with no enhancement on contrast attached to conus and filum terminale. Plain and contrast enhanced MRI scans of lumbar spine were performed showing the lesion surrounded by cord matter [Figure 2]. Neutrophil preponderance in CSF and abnormal mass lesion in lumbar region prompted us for surgical exploration with a revised diagnosis of superadded bacterial infection (abscess) in an acute viral transverse myelitis.

Patient was immediately taken up for surgery with a plan to debride the area and obtain tissue for histopathology, culture and sensitivity. D12 to L3 laminectomy was performed and dura was opened. Arachnoid was thin, clear, and transparent. Yellowish, purulent material was seen loosely attached to conus medullaris and filum terminale through the intact arachnoid [Figure 3]. Arachnoid was opened. The CSF was clear. Yellowish, thick, slimy material was seen loose in the subarachnoid plane and on further evacuation was found attached to the conus but was easily separable with a jet of saline. The material was sent for histopathological examination, fungal, tubercular, and bacterial smears and cultures. Histopathological examination was suggestive of nonspecific inflammation with abundant neutrophils and few lymphocytes. KOH mount, gram stain, and smear for acid fast bacilli (AFB)
and immunoflorescence staining for tubercular bacilli were negative. Bacterial (aerobic, anaerobic), and fungal cultures were negative. As the CSF was showing plenty of neutrophils and histopathological examination showed abundance of neutrophils, he was started on empirical IV antibiotics (Cefoperazone 2 g TID, amikacin 750 mg OD, and metronidazole 500 mg TID) with a provisional diagnosis of pyogenic abscess. After a week, patient complained of paraesthesias involving both the upper limbs. Repeat MRI scan of spinal cord showed evidence of increase in size of cervico-dorsal lesion, which by now had involved the cord from C4 to D12 (holocord). Patchy enhancement of meninges of dorsal spine (D2-D4) was seen [Figure 4a and b]. Cervico-dorsal laminectomy (C4-D4) was performed and cord was explored with multiple midline myelotomies at C5, C7, and D2 regions. There was yellowish-white cheesy material in the depth of white matter with no clear plane from surrounding neural tissue. Multiple biopsies were taken and were sent for culture and histopathological examination. Grams stain, fungal stains, and AFB smears were again negative. Cultures (as per protocol for aerobic, anaerobic, fungal, and tubercular) failed to grow any organism. Histopathological examination showed features of nonspecific inflammation with neutrophils. No granulomas or signs of demyelination were noted. He was continued on empirical antibiotics, supportive care for bowel, bladder, and back, low molecular heparin, and regular physiotherapy. At 6 weeks, tubercular culture in 12 B medium of BACTEC, 460 TB system showed growth of colonies, having sensitivity to all the first line drugs. Spinal tuberculosis was diagnosed and he was started on anti-tubercular drugs. He was further worked up for his immune status with total proteins, serum albumin, and CD4 counts were within normal limits. His Mantoux was 24 mm (strongly reactive). The patient is under constant follow up, but even at 8 months after starting anti-tubercular drugs, he failed to show neurological improvement.

**DISCUSSION**

Tuberculosis is endemic in developing countries like India. Central nervous system is affected in about 10% of patients with tuberculosis. Pachymeningitis, leptomeningitis, tuberculomas, and abscesses are the various pathological presentations of spinal cord tuberculosis with intradural and subarachnoidal forms being quite rare. Intramedullary and intradural extramedullary tuberculomas are rare and have been reported in the literature. Intradural tubercular abscesses are much rarer. Less than 10 cases of intradural tubercular abscesses have been reported. Approximately seven cases of holocord intramedullary abscesses have been reported and only in one case the abscess was of tubercular etiology. Only one case of intradural-extramedullary tubercular abscess has been reported. To the best of our knowledge, this is the second case report of a holocord intramedullary tubercular abscess, along with subarachnoidal lobe of tubercular abscesses.

Spinal cord tuberculosis is usually secondary to tuberculosis elsewhere in the body. Tubercular abscesses are more common in immunocompromised individuals as compared with tuberculomas seen in immunocompetent individuals. But our patient was immunocompetent and had no evidence of tuberculosis elsewhere. Clinical presentation of spinal cord abscesses is highly variable, ranging from progressive neurologic deficit to acute transverse myelitis like presentation. The possible explanation to holocord involvement has been ascribed to the tracking of the purulent material along the path of least resistance—along white matter tracts of the spinal cord. Occurrence in the subarachnoidal space and rapid spread prompts us to hypothesize that the pus might have tracked along the central canal and

Figure 3: Intraoperative picture. After D12 to L3 laminectomy was done and dura was opened. The arachnoid was thin and transparent. Through the arachnoid yellowish, thick pus attached to conus and filum terminale is seen.

Figure 4: Magnetic resonance images of dorsal spine after first surgery, (a) T2 weighted sagittal image of dorsal spine showing hyperintensity of cord extending from cervical cord down to D12 level, (b) Post gadolinium T1 weighted sagittal image of cervico-dorsal spine showing enhancement of meninges at D2, D3, and D4 levels along with patchy enhancement of adjacent cord.
then exteriorized at the conus. Constitutional symptoms like fever, weight loss may not be conspicuous in CNS tuberculosis[20] as are serological markers like ESR, C-reactive protein.[8] Tubercle bacilli were also not seen in microscopic examination of the lesion, which is unusual for tubercular abscesses, which are classically described to be steaming with AFB. Histopathological examination showed only features of nonspecific inflammation with no evidence of granulomas or tubercle bacilli. Diagnosis was made only after the tubercular colonies were grown on culture media. This had caused a considerable delay in initiating antitubercular chemotherapy.

MRI is the best and most sensitive investigation to detect mass lesions, cord changes and defining its extent. Tubercular abscesses are iso to hypointense in T1 weighted images, iso to hyperintense in T2 weighted images and so show ring enhancement after contrast administration.[8,12] There can be enhancement of the overlying meninges. However, these imaging features are shared by bacterial abscesses, demyelination, and intramedullary tumors. Biopsy along with microscopic examination and culture are essential to confirm the diagnosis. In our patient, both intramedullary and extramedullary lesions were non-enhancing. The central T2 hyperintesity in the cord, extending more than three vertebral levels and the acute presentation were suggestive of acute transverse myelitis. This atypical radiological picture has delayed our diagnosis.

As the number of tubercular abscesses reported in the literature is limited, definitive guidelines cannot be laid down for their management. Surgery is aimed for biopsy and prompt management may offer a favorable prognosis, even in cases having severe neurological deficits.[9]

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CONCLUSION

Intramedullary and subarachnoidal tubercular abscesses are rare forms of spinal tuberculosis, presenting with acute or subacute, progressive motor and sensory deficits. MRI features are nonspecific and a definitive diagnosis is often difficult on the basis of radiological features alone. In countries where tuberculosis is endemic, a high index of suspicion is necessary for early diagnosis and treatment with anti-tubercular drugs. With early diagnosis and treatment, good neurological recovery can be expected.