Case Report

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Tuberculous Meningitis complicated with thoracic cord myelitis; a diagnostic conundrum.

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Abstract

Tuberculosis is known as the great masquerader for its ability to present atypically. We report a case of a young man presented with progressive tuberculous meningitis and subsequently developed paraplegia due to thoracic cord myelitis, where diagnosis was confirmed bacteriologically. We highlight the presence of acute gastric dilatation as a warning sign of thoracic cord involvement and the place for CSF examination and spinal imaging in arriving at a clinical diagnosis, as treatment needs to start without delay to avoid permanent sequelae. Patient showed a good response to a combined approach of anti-tuberculosis treatment, systemic steroids and intravenous immunoglobulin.

Keywords: *Tuberculous meningitis, Tuberculous myelitis, Intravenous immunoglobulin*

BACKGROUND

Tuberculosis (TB) is an airborne infection caused by *Mycobacterium tuberculosis* with a history of more than four thousand years. In Sri Lanka, the prevalence rate is 103 per 100,000 populations which show that it is one of the low TB prevalence countries in the region [1]. Central nervous system TB is common in TB prevailing developing

countries. Within the wide spectrum of presentations of Central nervous system TB, paraplegia due to thoracic cord myelitis is an uncommon presentation which leads to diagnostic dilemma. Delay in initiating treatment causes poor outcome with permanent neurological sequelae.

CASE REPORT

We report a case of a 31-year-old male, presented to Teaching Hospital Peradeniya with headache, fever and vomiting for 3 days. He had mild occipital headache associated with photophobia which has been worsening over one week. He had loss of appetite and weight for two months, but no cough, abdominal pain, altered urinary or bowel habits. He was a teetotaler and non-smoker and had no past history or contact history of tuberculosis. He had taken treatments from general practitioner which partly subsided his symptoms without improving his general ill-health.

On examination he was thin-built, ill looking, mildly dehydrated and febrile. There were no rash or enlarged lymph nodes. His cardiovascular and

respiratory system examination were unremarkable except for mild tachycardia (104/min). He had soft abdomen without organomegaly or renal angle tenderness. He was conscious, cranial nerves and bilateral upper and lower limb examinations were normal and had good bladder and bowel continence. He had mild neck stiffness, but Kernig's and Brudzinski's signs were negative.

His serology was normal (Table 1) and cerebrospinal fluid (CSF) analysis (Table 2) showed turbid appearance with lymphocytosis, high proteins and low sugars. CSF pyogenic culture revealed no growth along with negative blood and urine cultures. Patient was treated with intravenous Ceftriaxone 2g twice daily and intravenous Acyclovir 500 mg twice daily for possible partially treated bacterial meningitis.

Table 1: Serological Investigations

Test	Value
Leukocyte count	9.42 x 10 ⁹ /l
Neutrophils	8.11 x 10 ⁹ /l
Platelet count	340 x 10 ⁹ /l
Haemoglobin	11.9 g/dL
C-reactive protein (CRP)	8.8mg/dl
Aspartate aminotransferase (AST)	40.7 U/l
Alanine aminotransferase (ALT)	38.5 U/l
Serum creatinine	50.8 micromoles/L
Potassium	3.5 mmol/L
Sodium	137 mmol/L
Calcium	2.15 mmol/
Magnesium	0.89 mmol/L
Retroviral antibodies	Negative
Erythrocyte sedimentation rate in 1 hour	105

Table 2: Cerebrospinal Fluid (CSF) analysis

Test	Value
Appearance	Turbid
Red blood cells (RBC)	4 Cells/mm ³
White blood cells (WBC)	54 cells/mm ³
Differential counts	Lymphocytes 97%, Polymorphs 03%
Protein	113 mg/L (<45)
Glucose	1.6 mmol/L (serum glucose - 6.4 mmol/L)
Adenosine deaminase	21U/L (TB meningitis cut-off 12.23+/- 4.92)
TB PCR	Not detected
Cryptococci	Negative
AFB stain	Negative
Pyogenic culture	No growth
Fungal culture	No growth
TB culture	positive for <i>Mycobacterium tuberculosis</i>

On second day of admission patient developed gradual onset bilateral lower limb weakness which progressed in to paraplegia with power of zero, mild hypotonia, absent reflexes and equivocal planter response within two-days duration. This was subsequently associated with drowsiness, difficulty in breathing, slurring of speech without involvement of lower cranial nerves, abdominal distension and urinary retention. Upper limb examination was normal.

Contrast enhanced computed tomography (CECT) of brain and cerebral venogram showed no features suggestive of venous sinus thrombosis, infarctions or haemorrhage and Electroencephalogram (EEG) revealed generalized slowing with intermittent seizures suggestive of encephalitis. Nerve conductions on bilateral common peroneal and posterior tibial nerve were normal, but delayed F waves and conduction blocks suggestive of severe demyelinating polyneuropathy were noted. Chest radiograph was poorly inflated and abdominal X ray showed marked gastric dilatation. His erythrocyte sedimentation rate in first hour was 105mm, CSF adenosine deaminase became positive, though CSF PCR was negative for TB. His retroviral screen was negative.

With the high clinical suspicion on tuberculous meningitis, patient was started on standard anti TB medication and Intravenous dexamethasone, followed by intravenous immunoglobulin (IVIG)

0.4g/kg daily due to progressive neuropathy. Gastric decompression achieved with nasogastric tube. Meanwhile patient developed poor respiratory effort which warranted intubation and mechanical ventilation for five days.

Patient underwent magnetic resonance imaging (MRI), which showed normal MRI brain (figure-1) with a segment of dilated thoracic central canal/syrinx (T8-T9) with thoracic cord atrophy at T6, T7, representing chronic changes of TB myelitis (Figure-2). There were no abnormalities seen in vertebral bodies, intervertebral disks or para-spinal soft tissues. CSF culture was positive for *Mycobacterium tuberculosis* after 10 weeks of incubation which bacteriologically confirmed the diagnosis.

Anti TB treatment is planned to continue for 12 months, with 4-6 weeks of steroids. Limb physiotherapy and ambulatory rehabilitation was continued. Patient was discharged after six weeks of inward care, where he was able to walk with support on discharge.

Figure 1: Normal MRI brain (Sagittal section -T1 weighted)

Figure 2: T2 weighted sagittal and axial sections of spine

A segment of dilated thoracic central canal/syrinx (T8-T9) with thoracic cord atrophy at T6, T7, representing chronic changes of TB myelitis

DISCUSSION

Central nervous system TB commonly presents as meningitis or tuberculoma, though it has wide spectrum of uncommon manifestations including radiculomyelitis (Arachnoiditis), myelitis, TB abscess and syringomyelia [2,3]. Paraplegia may be common in tuberculous spondylitis with compressive myelopathy (Pott's disease), however, spinal tuberculomas and radiculomyelitis remain rare causes [2,4].

Myelitis is an acute inflammatory disorder of the spinal cord. It leads to spinal cord swelling, ischemia and necrosis leading to loss of function. Most commonly affected area is the cervico-thoracic spine (80%) [5]. Vasculopathy, immunological mediation and direct invasion by bacilli are the postulated reasons for TB myelitis [5,6]. Patients with myelitis present with features of non-compressive myelopathy including paresthesia, paraparesis/ tetraparesis, sensory impairment, bladder involvement (Urinary incontinence), bowel involvement and sexual dysfunction. Symptoms may evolve within hours to few weeks. Fever, headache, blurring of vision, recurrent vomiting, loss of appetite and loss of weight are common with CNS involvement [2,5,6]. Acute gastric dilatation is one of the important features which warns that there will be thoracic spinal involvement in appropriate clinical context [7].

CSF full report will indicate lymphocytosis, high CSF protein and low CSF glucose. CSF ADA is not used for routine diagnosis as it lacks specificity and the level can be raised in several other conditions [5,8]. On MRI, these lesions are hyper intense on T2-weighted images without contrast enhancement. Radiological findings associated with poor outcome include syrinx formation and cord atrophy or cavitation [5]. In our patient there was no significant intramedullary signal intensity abnormality seen while having mild syrinx formation at T8, T9 and mild cord atrophy noted at T6, T7 due to chronic changes. Bacteriological or molecular evidence is needed for confirmation of tuberculous myelitis.

However, there is lack of consensus on management of tuberculous myelitis. Since the postulated pathogeneses for TB myelitis include immune-mediated inflammation and direct bacillary invasion, high dose intravenous steroids along with standard anti-TB therapy including Isoniazid, Rifampin, Pyrazinamide and Ethambutol have a beneficial role [2,5,6,8]. Use of IVIG along with anti TB therapy and steroids to treat resistant cases of tuberculous myelitis and syrinx formation in some studies have been shown the clinical improvement with favorable outcome and recognized benefits were there for the patients with acute deterioration as well [9,10]. Limb physiotherapy and ambulatory rehabilitation is equally important for a better clinical outcome in a patient with significant disability.

Mixed pattern of outcome can be observed in the available literature where complete or near complete resolution [5], partial resolution [2,10] and no change in neurological status [11] may occur. The time taken for recovery is usually few months but it can be vary from few days to months [5,6] where our patient was able to engage in activities of daily living independently at the end of six months follow up.

CONCLUSION

In conclusion, tuberculous myelitis is an uncommon complication of TB meningitis which needs to be suspected in a meningitis patient with features of myelopathy. Along with anti-TB medications and steroids, IVIG can be considered as a treatment option in a patient with progressive symptoms.

Author declaration

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Author Contributions

All authors examined, assessed and involved in the management of the patient. All authors collected and analyzed data. All authors read and approved the final manuscript.

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Ethics approval and consent to participate

Written informed consent was obtained from the patient.

Competing interests

The authors declare that they have no competing interests.

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