

Early Syringomyelia in Tubercular Meningitis

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Abstract

Tubercular meningitis (TBM) can have various complications. Sometimes syringomyelia can also occur as a late complication of tubercular meningitis. Although syrinx formation in early stage of TBM is very rare. There are only four published case reports of syringomyelia in acute stage of TBM. Here we report a patient with tubercular meningitis who developed syringomyelia in early course of illness.

Introduction

The most frequent manifestation of central nervous system tuberculosis is tuberculous meningitis. TBM can have varied early and delayed complications including hydrocephalus, cranial nerve palsies, vasculitic infarction, arachnoiditis and tuberculoma involving brain or spinal cord. Although syringomyelia can occur sometimes as a late complication of tubercular meningitis,¹ its occurrence in early stages of TBM is very rare. There are only four published case reports of syringomyelia in acute stage of TBM.²⁻⁴ We are reporting a patient with tubercular meningitis who developed syringomyelia in early disease course and pertinent review of literature.

Case Report

A 20-years-old male presented with history of fever, headache and vomiting for last two months. Clinical examination revealed bilateral papilledema and signs of meningeal irritation. Patient was drowsy. Neurological examination including cranial nerves and motor system examination was normal. His blood investigations were normal. Plain and Contrast Enhanced Computed Tomography (CECT) Scan of head was suggestive of communicating hydrocephalus and brain oedema (Figure 1 A, B). Cerebrospinal fluid (CSF) analysis revealed leukocyte count 150 cells/cubic mm with lymphocyte count 90%, glucose 30 mg% (Corresponding blood sugar 90 mg %; ratio 1/3) and protein level of 150 mg%. CSF acid fast Bacilli (AFB) staining was negative. He was started on first line antitubercular drugs, steroids and antiedema drugs and urgent ventriculoperitoneal shunt

surgery was done. Intraoperatively the CSF pressure was markedly raised and CSF was grossly clear. Patient became conscious and oriented on next day of surgery. After 7 days he developed weakness of all four limbs and inability to control micturition. Motor system examination revealed hypotonia of all four limbs. Motor weakness was 1/5 in upper limbs and 2/5 in lower limbs. Deep tendon reflexes were absent in upper limbs and brisk in lower limbs. Planter reflex were extensor bilaterally. Magnetic Resonance Imaging (MRI) spine showed long segment syrinx extending from Cervico-medullary junction to T10 level (Figure 2 A, B, C, D). He was continued on antitubercular drugs and steroids for 2 weeks with no

improvement in neurological status. Foramen magnum decompression was planned but patient left the hospital against advice.

Discussion

Syringomyelia is an uncommon and usually late complication of TBM. The time interval between TBM and syrinx formation varies from 7 - 28 years.³ In present case syringomyelia developed in early stages of TBM during the treatment, which is very rare. Daif et al first time described two cases of syringomyelia in early course of tubercular meningitis (after 11 days and 6 weeks).² Pandey et al similarly reported a case of syringomyelia in early course of TBM.⁴

Exact pathogenesis of syrinx formation in TBM is not known. Several mechanisms have been proposed in the syrinx formation after tubercular meningitis. Syrinx formation may be secondary to either an obliterative endarteritis causing ischemic injury or softening of spinal cord or may be because of post inflammatory scarring.^{5,6} Tubercular endarteritis

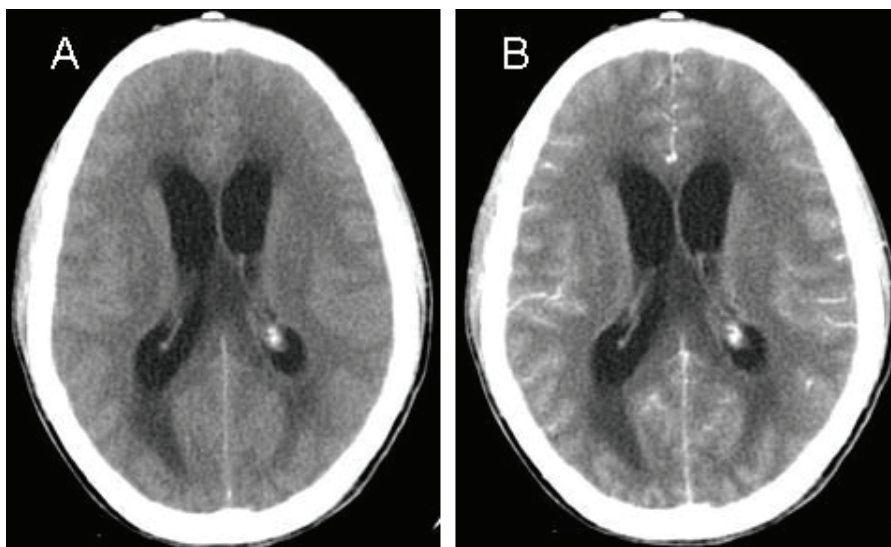


Fig. 1 A, B: Plain and contrast enhanced CT scan of head suggestive of gross communicating hydrocephalus.

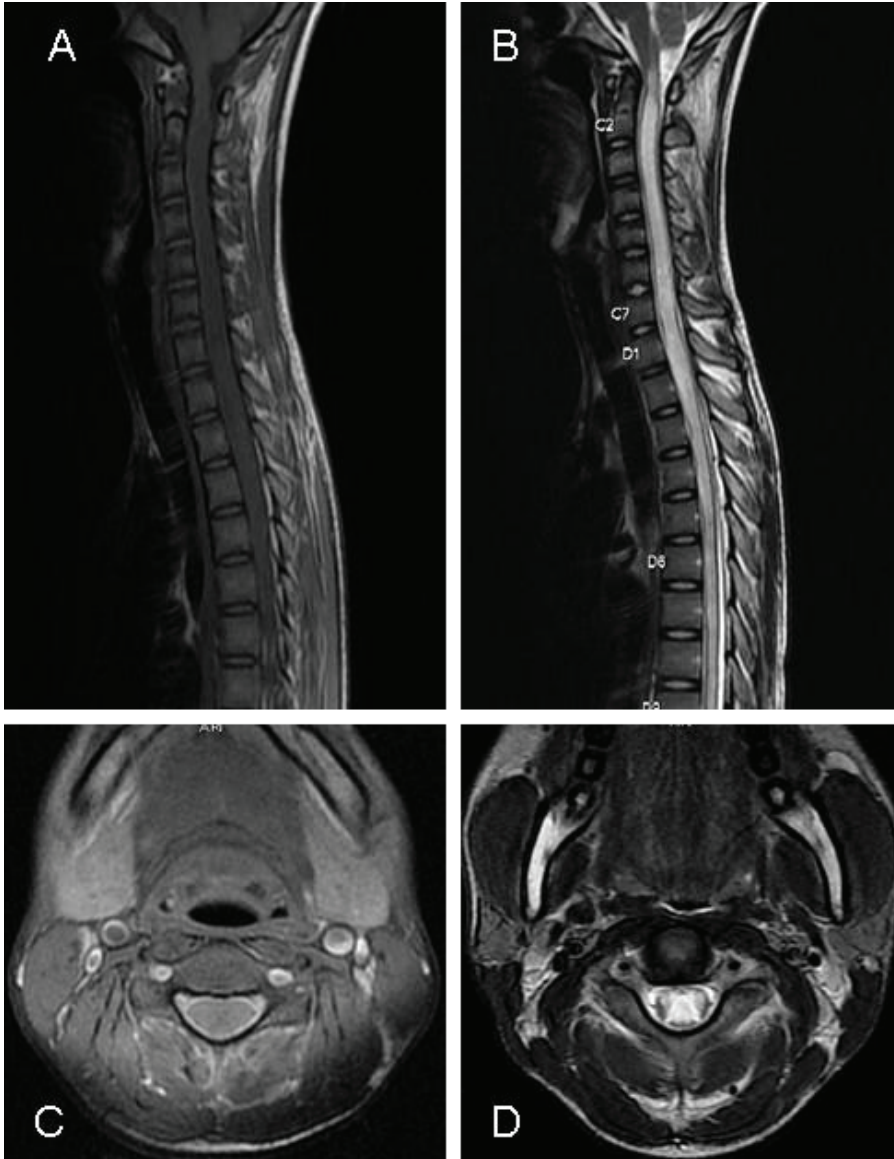


Fig. 2 A, B, C, D: T1 and T2 weighted Sagittal and Axial images of MRI spine showing cervico-dorsal syrinx.

producing softening of the cord, spinal subarachnoid space scarring leading to reduction in the compliance of the

subarachnoid compartment and the patent Virchow-Robin spaces in the spinal cord providing a conduit for the

CSF to enter into the central canal are three basic mechanisms in the formation of syrinx in TBM. Development of communicating hydrocephalus and acute rise in intracranial tension may contribute in pathogenesis of syrinx formation⁴ as occurred in our case.

No medical treatment is known for patients with syringomyelia following TBM. However, a chronic, stable clinical course is common. Surgical treatment most likely will be necessary^{7,8} though ideal treatment of syrinx following TBM is not known.

Conclusion

Acute-onset syringomyelia should be suspected in any patient being treated for tuberculous meningitis who subsequently develops limb weakness and/or sphincteric dysfunction.

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