Neuroimaging Highlight

Editors: William Hu, Mark Hudon

Syrinx and Tuberculoma Formation in Tuberculous Arachnoiditis

A.C.F. Hui, Y.L. Chan, R. Kay

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A 21-year-old immunocompetent woman presented with fever and headache. Cerebrospinal fluid revealed a raised protein level of 2.19 g/dL, reduced glucose of 0.9 mmol/L and raised leucocyte count (80% lymphocytes). She was started on anti-TB therapy for presumed tuberculous meningitis: subsequent CSF culture confirmed the diagnosis with the isolation of *Mycobacterium tuberculosis*. This was complicated by hydrocephalus, for which a ventriculo-peritoneal shunt was inserted; postoperative course was uneventful. Six months later she developed paraesthesia of both legs followed by a flaccid paraparesis. MRI of the spinal cord demonstrated arachnoiditis, showing diffuse meningeal enhancement of the pia and arachnoid with extramedullary granuloma formation. Dexamethasone was prescribed, with a good response and was maintained for six weeks. One year later she developed another episode of arachnoiditis. Repeat axial nonenhanced T-1 weighted

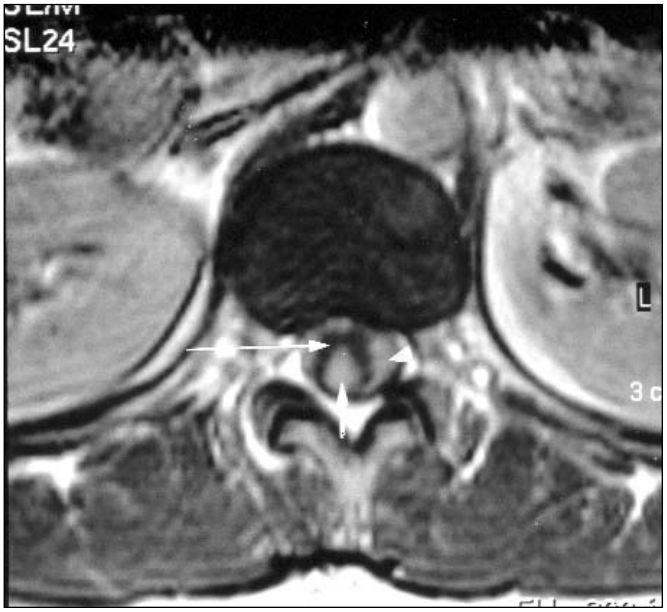

Figure 1: *Axial T1-weighted noncontrast image at the level of the upper end plate of T12 vertebral body. The isointense intramedullary tuberculoma (arrow) is located within the hypointense syrinx (long arrow). The thin rim of cord tissue merges with and cannot be separated from the thick isointense extramedullary arachnoiditis (arrow head) which completely obliterates the thecal sac.*

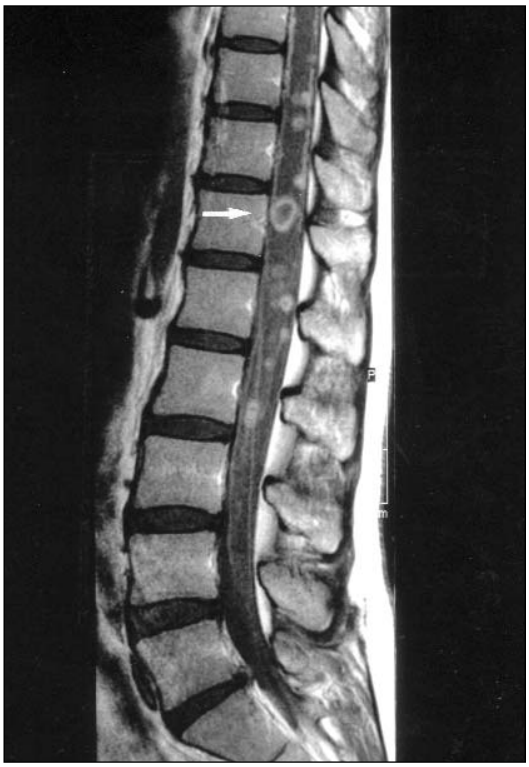

Figure 2: *Sagittal TI-weighted gadolinium enhanced image demonstrates the extensive syringomyelia down to the conus medullaris level. The intramedullary tuberculoma at upper T12 level shows rim enhancement (arrow). Smaller nodular and rim-enhanced intradural extramedullary granulomas are also shown.*

From the Department of Medicine & Therapeutics (ACFH, RK) and the Department of Diagnostic Radiology and Organ Imaging (YLC), Prince of Wales Hospital, Chinese University of Hong Kong, Hong Kong, China

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Reprint requests to: Andrew Che Fai Hui, Department of Medicine & Therapeutics, Prince of Wales Hospital, Chinese University of Hong Kong, Shatin, NT, Hong Kong, China

MR of the spine revealed the formation of a syrinx with a large solid intramedullary tuberculoma at the level of T12 (Figure 1). Post contrast sagittal T-1 weighted images demonstrated smaller ring-enhanced granulomas and widespread enhancement of the wall of the syrinx (Figure 2). Without contrast, the active rim and the caseous material were isointense. She was given a two month course of dexamethasone and transferred to a rehabilitation center. This time the response was less pronounced and the patient eventually required a walking frame.

Myelopathy developing after TB infection may be due to granulomatous arachnoiditis, syringomyelia or cord compression as a result of vertebral osteomyelitis or a solitary tuberculoma.[1-3] Syringomyelia and arachnoiditis following TB meningitis is rare and typically develops in three stages. First there is the acute meningitis followed by a symptom-free interval, which may be years; finally a rapid and steady paraparesis.[4] Treatment with steroids and laminectomy have been reported but a review of 13 cases in the English literature reveals that 11 patients had residual weakness, one died and another was lost to follow-up.[1,3-6] Syrinx formation is thought to be secondary to either an obliterative endarteritis causing ischemic injury and softening or post-inflammatory scarring which leads to spinal block.[7] In this case the presence of a large granuloma may have contributed to the development of a syrinx due to disruption of CSF flow dynamics.

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