

Letter to the Editor

TO THE EDITOR

INTRAMEDULLARY SPINAL TUBERCULOMA AND SYRINGOMYELIA

I read with interest the paper by Hui et al¹ describing the case of a woman presenting with tuberculous meningitis treated with anti-TB therapy. Six months later she developed spastic paraparesis related to spinal arachnoiditis illustrated by MRI study. Good response occurred with dexamethasone given for six weeks. One year later she developed another episode of arachnoiditis. MRI study now showed spinal arachnoiditis, a large solid intramedullary tuberculoma at T12, and syringomyelia down to the conus medullary level. She was given a two-month course of dexamethasone and transferred to a rehabilitation center, but response was less pronounced.

With colleagues, I reported a 37-year-old man with a presumed intramedullary tuberculoma presenting with a three-week history of paresthesiae in the left chest wall and numbness in the right leg and abdomen.² Examination revealed right hypoesthesia to touch and pain with a T6 level and sacral sparing. MRI showed an expanding and ring-enhancing annular lesion at T5 and a syringomyelic cavity extending 70 mm below the presumed granulomatous lesion. There was evidence of active tuberculous prostatitis. Cerebrospinal fluid examination revealed no abnormalities. He was treated only with anti-TB therapy, a few weeks later becoming asymptomatic. After 10 months of treatment there was reduction of the space occupying lesion and collapse of the syrinx.

As stated by Hui et al,¹ spinal arachnoiditis following TB meningitis is the usual cause of syringomyelia complicating neurotuberculosis. Steroids and eventually laminectomy are the treatment of choice. Exceptionally however, syringomyelia, as in the reference cases, is associated with an intramedullary tuberculoma. Treatment now consists of initiation or continuation of anti-TB therapy; steroids should be restricted to cases with associated arachnoiditis. MRI proves invaluable in confirming the resolution of the structural lesion as the symptoms disappear.²

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REPLY

I appreciate the observations from Berciano and agree that chemotherapy is the mainstay of treatment; this should be continued for one to two years.¹ The maximal benefit of steroids is seen in cases with tuberculous meningitis of intermediate severity.² It is often prescribed for patients who develop arachnoiditis but the mechanism of action is unclear as steroids do not seem to reduce CSF proinflammatory cytokines.³

Tuberculomas that are not causing significant compression and are not located in a strategic location do not require resection and may resolve with anti-TB treatment. Paradoxical expansion of tuberculomas after appropriate drug treatment has been reported and this should be considered in patients who deteriorate despite treatment with anti-TB drugs.⁴

Tuberculous meningitis complicated by both syringomyelia and intramedullary tuberculoma is uncommon and there is insufficient experience to dictate management of patients. The patient's response to conservative treatment and the relationship between the tuberculoma and the syrinx are important considerations. For example, if hydrocephalus is present and MRI shows that there is a patent central canal communicating with the fourth ventricle, shunting would be indicated in this situation. On the other hand, surgery would not be useful if the cavity is caused by obliteration of the spinal vasculature. Serial MRI and CSF flow studies to monitor progress is recommended. Further long-term studies are needed to evaluate the role of surgery as the etiology and the natural history of the syrinx is unclear.

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