Letter to the Editor

To the Editor

Intramedullary Spinal Tuberculoma and Syringomyelia

I read with interest the paper by Hui et al[1] 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]

José Berciano
Santander, Spain

1. Hui ACF, Chan YL, Kay R. Syrinx and tuberculoma formation in tuberculous arachnoiditis. Can J Neurol Sci 2001;28:148-149.
2. Sanchez-Pernaute R, Berciano J, Rebollo M, Pascual J. Intramedullary tuberculoma of the spinal cord with syringomyelia. Neuroradiology 1996;38:S105-S106.

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.

Andrew CF Hui
New Territories, Hong Kong

1. Thwaites G, Chau TTH, Mai NTH, et al. Tuberculous meningitis. J Neurol Neurosurg Psychiatry 2000;68:289-299.
2. Donald PR, Schoeman JF, Beyers N. Concentrations of interferon γ tumour necrosis factor α, and interleukin-1 β in the cerebrospinal fluid of children treated for tuberculous meningitis. Clin Infect Dis 1995;21:924-929.
3. Girgis NI, Farid Z, Kilpatrick ME, et al. Dexamethasone adjunctive treatment for tuberculous meningitis. Pediatr Infect Dis J 1991; 10:179-183.
4. Teoh R, Humphries MJ, O'Mahony G. Symptomatic intracranial tuberculoma developing during treatment of tuberculosis: a report of 10 patients and review of the literature. Q J Med 1987;63(241):449-460.

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To the Editor

Dr. Alastair Buchan's eloquent editorial on our review of education in the clinical neurosciences (Can J Neurol Sci 2001; 28:281-282) missed the point. Resident training program committees are not bureaucratic regulatory bodies, and do not set call-schedule, morning-after or any other type of mandatory rules. They are committees to oversee and administrate training programs, and among other things ensure their adherence to contracts and guidelines set by governments and the Royal College. If they don't do a reasonable job of that, their programs will not be accredited and will ultimately be suspended. Our review pointed out that it has become impossible for some, and perhaps many, programs to meet these requirements due to diminishing resident numbers and increasingly demanding clinical teaching units – Dr. Buchan himself refers to modern stroke care as "extreme neurology"! One solution that Dr. Buchan took exception to is "hospitalists" to complement the resident staff. Since that job includes night time in-hospital call, it has been difficult to attract Canadian-trained doctors to such positions. With special approval from our provincial College we, in Edmonton, have been able to acquire the services of foreign medical graduates, otherwise unqualified to practice private medicine in Canada, following a period of supervised training. These doctors are hired (by the hospital) precisely to "keep Canadian residents in their beds" as Dr. Buchan worries. While they are fine doctors, they are not, as Dr. Buchan mistakes, foreign neurologists, neurosurgeons or neuroscientists seeking to "light their torches at the fire of the Ancients". There are, in fact, no work regulations regarding international fellows on our services, and thank goodness for that, since all of them are on the stroke team!

We are sorry that Dr. Buchan, and perhaps others, found our review "depressing reading". The constant lament that "things are not what they used to be" may, in the case of medical specialty training today, actually be true for a change. Recognizing this, and dealing with it proactively and effectively, is the progressive approach, not going "back to the future", as Dr. Buchan would suggest.

J. Max Findlay
Edmonton, Alberta

Notes and Announcements

Neuroscience Conference in Banff

The Department of Clinical Neurosciences, University of Calgary, will hold its 9th Annual Banff Conference at the Canmore Radisson Hotel and Conference Centre, near Banff. The Conference will run from Friday, March 15, 2002 to Sunday, March 17 2002. There will be three invited guest speakers from Canada, the United States and the United Kingdom. Topics include spinal cord repair, MRI and multiple sclerosis, and hydrocephalus. There will be case presentations and research presentations from the Department of Clinical Neurosciences, University of Calgary. All neurologists, neurosurgeons, physiatrists, and other interested physicians and researchers are invited.

For more information please contact Ms Danielle Sikander at 403-670-1260 or e-mail at sikander@ucalgary.ca.

Erratum

Methodology for the Canadian Activase for Stroke Effectiveness Study (CASES). Can. J. Neurol. Sci. 2001; 28: 232-238.

Two members of the CASES Investigators were mistakenly omitted from the CASES Investigators list. They are: Vance Makin (Lions Gate Hospital, North Vancouver), and JH Warwick Pexman (Foothills Hospital, Calgary).

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