Paradoxical progression of conus tuberculoma during chemotherapy of tuberculous meningitis

Sir,

Simultaneous presentation of both spinal arachnoiditis and intramedullary tuberculoma, which deteriorate
during antituberculous chemotherapy, is too rare. We report a rare case and study the clinico-pathological manifestations of spinal arachnoiditis and intramedullary tuberculoma.

A 21-year-old girl was admitted to our hospital due to fever and increased headache since one-and-a-half month before admission. The patient appeared ill and had neck stiffness. Chest X-ray was normal. A plain brain CT scan revealed dilated ventricles with periventricular edema. Cerebrospinal fluid analysis yielded 2600 leukocytes/µl of which 70% were lymphocytes; total protein level was 240mg/dL, glucose level 40mg/dL. Simultaneous blood glucose was 110mg/dL. No organisms were seen on gram stain or in AFB (acid-fast bacilli) smear of CSF and gastric lavage fluid. Polymerase chain reaction (PCR) test specific for M. tuberculosis was reported positive. HIV antibody was negative. The patient was started on four-drug antituberculous chemotherapy and steroid. Neurological consultation revealed progressive loss of consciousness, symmetric movements of the limbs, increased deep tendon reflexes, neck stiffness, Babinski sign and blurred optic discs. A medium-pressure ventriculo-peritoneal shunt was inserted immediately. Over four weeks postoperatively she got better but she couldn’t move her feet perfectly and got blind. Left abductor palsy was noted. Motor function was graded 2/5+ proximally and 1/5 distally in both lower limbs with bilateral Babinski sign. Deep tendon reflexes were absent. Another CSF analysis yielded the same result. Electromyographic study revealed secondary axonal injury of the tibialis and common peroneal nerves due to multiple lumbosacral radiculopathies. On myelography a huge filling defect was present at the level of L1-L2 vertebrae, candle guttering appearance at the level of T11-12 and root adhesion of the cauda equina region [Figure 1]. The surgical procedure included laminectomies at T11-L3, dural incision and midline posterior myelotomy at the level L1-2. There was white to yellow cheesy soft suctionable tissue including small calcified particles. Pathologic tissue extended from the conus medullaris to cauda equina. Histopathologic examination revealed granulomatous lesion that contained caseating necrosis, Langhans’ giant cells and perivascular lymphocytic infiltration in the specimens.

The first documented case of paradoxical reaction during treatment of tuberculosis was reported by Citow et al.\cite{1} and similar cases have been reported occasionally.\cite{2,3} These phenomena generally occurred within three months after treatment initiation. Decreased penetration of antituberculous drugs into the brain or spinal cord and restoration of blood-brain barrier might lead to reactivation of latent foci. It does not usually represent treatment failure but the most likely explanation for these phenomena is an interaction between the host’s immune response and the direct effects of mycobacterial products. This suggest that despite an adequate response to treatment, an ongoing inflammatory process takes place in the arachnoid and spinal cord tissue.\cite{4} Campbell proposed that rapid killing of bacilli by effective treatment can cause the release of large amount of tuberculoprotein and other cell-wall products.\cite{5} It is logical to assume that the overall inflammatory response to M. tuberculosis reflects both the number and function of the appropriate immune cells and the amount of the antigen that they encounter.\cite{2}

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References