Radiology Quiz

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Clinical History

A 38-year-old Bangladeshi male presented to the Emergency Room with acute confusional state for 3 days, constitutional symptoms for 3 months and testicular mass for 2 months. Clinical examination revealed that he was febrile, confused, agitated with normal cranial nerves, normal motor system with bilateral down going planters, and there were no cerebeller sign. Systemic examination showed no lymphodenopathy or abnormal breath sound, there was tender liver with firm immobile epididymal swelling. The patient was started on ceftriaxone, and computed tomography of brain was carried out with normal findings. Cerebrospinal fluid was pale yellow, 31 WBCs differential: PMN 95%, mononuclear cells 5%, protein 2.1 gm, glucose 0.8 mmol. Erythrocyte sedimentation rate 8, CBCs normal. With this clinical and CSF picture, the patient was started empirically on anti-TB medication. Repeated CSF revealed 160 WBCs, and 8 gm protein (traumatic sample). The patient showed marked improvement after 10 days of starting anti-TB drugs. Liver biopsy confirmed presence of granuloma. Epyndidymal biopsy was normal. The patient was discharged from the hospital after 20 days in stable condition.



Figure 1 - T2 image showing tiny ring like lesions on the left hemisphere.



Figure 3 - Coronal flair image showing the same lesion.



Figure 2 - T1 with contrast showing multiple tiny rounded ring like lesion scattered within the cerebellum, mesencephalon posteriorly and posterior pons.



Figure 4 - T1 with contrast showing multiple tiny rounded ring like lesion scattered within the cerebellum, mesencephalon posteriorly and posterior pons.

Questions

- 1. What are the radiographic abnormalities?
 - 2. What is the diagnosis?

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Figure 1 - T2 image showing tiny ring like lesions on the left hemisphere.



Figure 3 - Coronal flair image showing the same lesion.



Figure 2 - T1 with contrast showing multiple tiny rounded ring like lesion scattered within the cerebellum, mesencephalon posteriorly and posterior pons.



Figure 4 - T1 with contrast showing multiple tiny rounded ring like lesion scattered within the cerebellum, mesencephalon posteriorly and posterior pons.

Findings and Discussion

Tuberculous meningitis and tuberculoma

Tuberculosis is the world's leading cause of death from a single infectious agent; neurological complications of tuberculosis are not uncommon. Tuberculous meningitis may occur as an isolated event, due to rupture of an asymptomatic cerebral tuberculoma into the subarachnoid or ventricular space, or it may be part of the picture of miliary (disseminated) tuberculosis. This latter situation prevails in approximately 75% of patients and evidence of tuberculosis outside the nervous system, most frequently in the lung, can usually be found. When meningitis results from rupture of a solitary subependymal tubercle the diagnosis depends upon findings in the CSF. The illness may begin acutely but more often, the onset is insidious, with gradual development of headache, low-grade fever and signs of meningitis. The chronic meningitis is most marked at the base of the brain, and the thick gelatinous exudate often involves cranial nerves with production of cranial nerve palsies. As the disease progresses the level of consciousness may be diminished and focal neurological deficits may appear. Cerebrospinal fluid examination typically reveals pleocytosis with 100-500 cells/mm3, with predominance of lymphocytes, elevated protein (100-500mg/ml), and glucose concentration which may be normal or diminished. Acid-fast bacilli are seen in stained smears of centrifuged CSF in less than one third of patients; the yield may be increased by repeated examinations and by staining the pellicle which forms in the CSF on standing in a test tube. Computed tomographic scanning may reveal the chronic basilar meningitis and tuberculomata within the brain. Tuberculous meningitis may be treated with drug regimens effective in the treatment of pulmonary tuberculosis, such as isoniazid plus rifampin for 6 months, with pyrazinamide given during the first 2 months. If the level of consciousness is diminished or if focal

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neurological deficits are present, addition of corticosteroids for the first few weeks of treatment may be beneficial. Since culture of the organism from CSF may require several weeks, it may be necessary to begin treatment empirically; clinical improvement after a week or two of therapy provides some support that the meningitis is due to mycobacterium tuberculosis. In the absence of meningitis, tuberculomata usually presents as space-occupying lesions, often with onset of seizures. Computed tomography scanning usually reveals multiple avascular mass lesions surrounded by edema. Magnetic resonance imaging is the investigation of choice in detecting the signs of TB meningitis and tuberculoma, as in this case it was helpful in detecting small tuberculoma in the early stage of the disease. Isotope scanning may reveal one or more areas of increased uptake. When the diagnosis is known, therapy with antituberculous drugs should be initiated and surgery avoided if possible, as fewer neurological sequelae result from medical therapy. Concomitant corticosteroid therapy may reduce cerebral edema and result in improvement of symptoms. If the diagnosis is made by biopsy, no further excision should be performed and antituberculous therapy should be instituted.

References

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