Brainstem encephalitis with serological evidence of St Louis encephalitis

SIR,—St Louis encephalitis is caused by an arbovirus that is transmitted to man by the mosquito.¹ This form of encephalitis usually presents in endemic areas in the summer and early autumn months with typical clinical manifestations of fever, headache, change in sensorium and tremor.² We report here a patient with serological evidence of St Louis encephalitis infection who presented with brainstem signs and symptomatology as the major manifestations of her illness.

A 49-year-old black female school janitor from southern Louisiana had the onset of fever, low back pain and myalgia in September, 1976. She was evaluated by a local physician and treated with ampicillin with a presumptive diagnosis of a urinary tract infection. Two days later she was admitted to a local hospital because of headache, fever, vertigo and unsteadiness of gait. Physical examination was said to be normal except for fever (38°). Skull and chest X-rays, routine electrolytes and CBC were normal. The spinal fluid contained 20 polymorphonuclear leukocytes/mm³, protein of 0.37 g/l and a glucose of 2.94 mmol/l. Spinal fluid culture was negative. The patient was treated with acetylsalicylic acid and bed rest but had progressive difficulty with walking and coordination, profuse nausea and vomiting. She was transferred to Parkland Memorial Hospital in Dallas, Texas three weeks after the onset of her illness. She was afebrile, alert with a normal mental status. General physical examination was unremarkable. The neurological examination showed a scanning dysarthria without dysphagia or palatal weakness; the gag reflexes were intact. There was nystagmus in all directions of gaze without gaze palsy or scotoma. The pupils were equal and reactive to light and the fundi were normal. Facial sensation and

movements were intact. The auditory acuity was normal and the patient denied tinnitus. There was gross dysmetria of the arms and legs bilaterally and marked truncal ataxia. Occasional head titubation was observed. Bilateral pyramidal tract weakness was present with hyperactive reflexes throughout and sustained clonus at the knees. The plantar responses were flexor. No sensory abnormalities were found.

Normal or negative laboratory results included complete blood count, VDRL, routine urinalysis, latex fixation, anti-nuclear antibodies, routine electrolytes and serum enzymes and coagulation factors. The erythrocyte sedimentation rate was 38 mm in the first hour. PPD was negative. Spinal fluid contained 14 lymphocytes/mm³ and 3 neutrophils/mm³, glucose was 74 mg%, protein was 68 mg%, and the IgG was 12.7 mg%. Cultures for bacteria including *M tuberculosis* and fungi were all negative. Computed tomography showed two areas of decreased attenuation in the brainstem. The area on the left measured about 1.5 cm in diameter and was located lateral to the upper portion of the fourth ventricle. The second zone of diminished density was smaller and located on the right side immediately lateral to the fourth ventricle. There were no areas of either abnormal, diminished or increased density in the cerebral hemispheres. The ventricular system, bony structures and cisterns all appeared to be normal. Following the intravenous administration of renografin there was no enhancement in the areas of diminished density noted in the unenhanced scan. Acute and convalescent serum samples were obtained for viral titres. The haemagglutination inhibition titre for St Louis encephalitis virus three weeks after onset of illness had a titre of 1:160; 10 weeks after the onset titre was equal to or greater than 1:320. The complement fixation titre on both occasions was 1:16. The

patient gradually recovered and two months after the onset of her illness had no neurological abnormalities and had remained well for three years.

The important feature of this case is that on clinical grounds it would not have been recognised as an encephalitis by the criteria used in previous investigations of St Louis encephalitis. Although the major clinical manifestations of St Louis encephalitis are headache, fever, nuchal rigidity, confusion and tremor, involvement of the brainstem is also apparent in about half the patients, with cranial nerve palsies, dysdiadochokinesia, and nystagmus.² In this patient, signs and symptoms of brainstem involvement were the predominant clinical manifestations. Obviously, in an area in which St Louis encephalitis is endemic, it is important to consider the possibility of this disease during the summer and early autumn months in patients who present with restricted disease of the nervous system, especially with brainstem involvement. In the absence of tissue examination, we cannot determine if this case represents a primary encephalitis or a infectious encephalitis.

References

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