

# Case Reports

## Influenza B Encephalitis

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SEROLOGICALLY CONFIRMED influenza B encephalitis has not been reported in adults. This disorder has been described for many years, with an association being inferred from a temporal relation between neurologic disorders and influenza epidemics.<sup>1,2</sup> Horner has presented serologic evidence of encephalitis caused by influenza A.<sup>3</sup> A teenaged girl has been reported to have an acute encephalitis associated with a nasopharyngeal culture positive for influenza B, but serologic testing was not reported.<sup>4</sup> During an influenza B outbreak in Chicago in 1971, 16 grade school students with encephalopathy (of which half had Reye's syndrome) showed serologic evidence of influenza B infection.<sup>5</sup> In the following report, encephalitis developed in an adult patient with a serologically documented influenza B infection.

### Report of a Case

The patient, a 41-year-old man, was admitted to hospital because of confusion of 18 hours' duration. One week before admission the patient had a febrile illness with cough and rhinitis. This resolved spontaneously within three days and he returned to work for two days. On the morning of admission the patient complained of headache and was noted by his wife to be confused. This confusion progressed, prompting transport to the emergency room.

There was no previous history of neurologic problems, and the patient was not known to smoke, drink alcohol or take medication. No recent head trauma or loss of consciousness was reported.

The temperature was 38°C (100.4°F), the pulse was 100 per minute, the respirations 34 and the blood pressure was 184/90 mm of mercury. On examination the patient was disoriented and lying still. He was normocephalic and resisted neck flexion. Pupils were equal, 5 mm and briskly reactive to light, the fundi appeared normal and the eyes moved in a wandering, conjugate fashion. The tympanic membranes were gray with normal landmarks. Cardiorespiratory and abdominal examinations showed no abnormalities. The patient did not respond to verbal requests, but on tactile stimulation would withdraw appropriately and say "don't." The face was symmetric with normal corneal reflexes bilaterally. He had a normal gag reflex and moved all four extremities strongly. Deep tendon reflexes were normoactive and symmetric. Babinski's sign was absent.

A urine specimen on analysis was normal. The hematocrit was 40.9% and a leukocyte count was 9,300 per  $\mu$ l, with 92% neutrophils, 6% lymphocytes and 1% basophils. A serum sodium level was 137 mEq per liter, potassium 4.0 mEq per liter, urea nitrogen 15 mg per dl, glucose 235 mg per dl (after administration of a solution of 50% dextrose in water), serum

aspartate aminotransferase (glutamic-oxaloacetic transaminase) 15 units per liter and blood ammonia 24 mmol per liter (normal 11 to 35). A cerebrospinal fluid (CSF) specimen was clear, with 6 leukocytes (80% lymphocytes and 20% monocytes) and 0 erythrocytes per  $\mu$ l. The CSF glucose level was 81 mg per dl and the protein 54 mg per dl (normal 15 to 45). A Gram's stain and india ink preparations were negative for organisms. Computed tomography revealed no abnormalities of the brain.

On the first day of admission the patient had progressive deterioration of his sensorium, a temperature elevation to 39°C (102.2°F) and a generalized tonic-clonic seizure. A regimen of phenytoin was started. By 36 hours after admission he was unresponsive to deep pain. Magnetic resonance imaging of the brain was normal. A repeat lumbar puncture showed the CSF to be clear with 21 leukocytes (77% lymphocytes, 20% monocytes and 3% neutrophils) and 1 erythrocyte per  $\mu$ l. A glucose level was 79 mg per dl and protein 65 mg per dl. The patient became responsive to pain on the fourth hospital day and was conversant by the eighth hospital day. He remained febrile until the fifth hospital day. He returned to work within a month of his hospital admission and has remained without neurologic sequelae.

Bacterial culture of blood, urine and CSF specimens was negative. Viral screening cultures of CSF, nasopharynx and rectal specimens were negative. Viral antibody titers (complement fixation method) were evaluated from serum specimens drawn on the day of and 18 days after admission. The titer for influenza B showed a fourfold rise, from 1:16 to 1:64. No change in titer was noted for Rous sarcoma virus, herpes simplex, mumps or influenza A.

### Discussion

Many neurologic complications are linked with influenza B infection. These include Reye's syndrome, transverse myelitis, the Guillain-Barré syndrome and encephalitis.<sup>1,2,4-6</sup> Both acute and postinfectious influenza B encephalitides have been described, with the former being confirmed by viral culture.<sup>4</sup> This case represents the first serologic confirmation of influenza B encephalitis in an adult. The time course of the illness suggests a postinfectious encephalitis, which would account for the negative culture results.<sup>7</sup>

Influenza B is generally considered to be a mild disease in otherwise healthy adults.<sup>8</sup> This report documents a serious neurologic complication of influenza B infection and reemphasizes the need to monitor influenza type B antibodies in patients with wintertime encephalopathic illnesses.<sup>5</sup>

### REFERENCES

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