

CASE 1

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The patient, a 27-year old male, was in his usual state of health until he developed severe frontal headache a few days after he swam in a fresh water lake. During the next two days he developed persistent nausea and vomiting, and was treated with oral ampicillin. On the day before admission he complained of pain on moving his eyes, was irritable and restless and occasionally asked inappropriate questions. On the morning of admission he was stuporous and poorly responsive, and had a generalized seizure.

Physical examination revealed a combative, post-ictal patient with blood pressure 140/75, pulse 120, respirations 24 and temperature 105<sup>o</sup>F. The neck was stiff and Kernig's sign was present. There was mild papilledema. The rest of the physical examination was not remarkable.

Laboratory studies revealed WBC 24,000 with 93% neutrophils and 7% lymphocytes. STS negative. Hematocrit 45%. Lumbar puncture revealed thick, purulent cerebrospinal fluid which would not rise in the monometer: WBC 9,000 (93% neutrophils), RBC 24,000. Examination of the wet unstained cerebrospinal fluid revealed many large cells which showed ameboid movement. Gram stain and cultures for bacteria and fungi were negative.

Intravenous ampicillin was continued and sulfadiazine was added to the treatment regimen. The patient became totally unresponsive. Although movement in both upper extremities, withdrawal to pain and corneal reflexes returned after treatment with intravenous mannitol, the patient died the day after admission, or four days after the onset of frontal headache.

Except for acute bronchopneumonia, pertinent pathology was limited to the central nervous system where an acute, hemorrhagic meningoencephalitis involved frontal, parietal and cerebellar cortical gray matter as well as the leptomeninges covering the base of the brain. In many perivascular spaces, amebae with central large nuclear karyosomes surrounded by clear halos were identified.

The stain is Hematoxylin and Eosin. Cellular detail was more distinct with trichrome stain. Points for consideration are: the route of infection in amebic meningoencephalitis and the pathogenicity of various species of free-living amebae.