CASE 9

Submitted by: Ursula T. Slager, M.D., Rancho Los Amigos Hospital, Downey, California.

Ref. No. ER-21-63.

This 33 year old white male developed headaches, low back pain, and muscular aches. When seen by his family physician, he was afebrile, had a WBC of 10,500, 58% lymphocytes, and normal urinalysis. Six days after the onset of the symptoms he was lethargic and staggered slightly, according to his wife. That night he was booked for drunken driving; after twelve hours in jail his state of consciousness had not changed and he was sent to the hospital. Here, he was comatose; there was no papilledema and the only abnormal physical findings were a right Babinski and Hoffmann with hyperactive reflexes on the right. Spinal fluid was clear, with an opening pressure of 380, closing pressure 290, normal chemistries, 68 cells, 79% lymphocytes, and sterile on culture. A right cerebral angiogram was within normal limits as were chest x-rays and EKG;s. Serum titers for mumps, poliomyelitis, Q fever, herpes simplex, psittacosis, St. Louis encephalitis, and western equine encephalitis were less than 1:8 on admission and showed no rise two weeks later. There were no salicylates or heavy metals demonstrable in the urine. He had been given both Salk and Sabin vaccines and "flu" immunization one year ago with a booster two months ago. The patient was treated with urea for cerebral edema. He remained comatose, never responded to voice and had vacillating and equivocal changes in neurologic findings. Ankle clonus and Babinski ~~was~~ were demonstrable and then absent on one side or the other. Eventually there was a flaccid paralysis of the left arm and leg. Repeat lumbar punctures were within normal limits. He died 38 days after admission with pneumonia and lung abscess.

At autopsy the lungs showed bilateral pneumonia with an abscess in the right lower lobe. The rest of the organs, including the salivary glands, were within normal limits.

The brain showed numerous punched out, sharply circumscribed lesions with central softening and yellow-gray to green. They were scattered throughout the cortical white matter, one crus cerebri, pons, and anterior columns at T-8.

Submitted are: 1 Kodachrome of the gross lesions, and 1 slide stained with Luxol Fast Blue-PAS-H & E.

Points for discussion:

1. Diagnosis?
2. Etiology?
3. Does brain edema play a role in the pathogenesis of these lesions, and could they have been prevented had the patient been treated 12 hours earlier to reduce cerebral edema?