Case 2

Submitted by: Uwamie Tomiyasu, M.D. and K. A. Ranganath, M.D.
Veterans Administration, Wadsworth Hospital Center
Los Angeles, California 90073

This 47-year-old man had a history of transient seizures at age 12, usually on an empty stomach. The terminal illness probably began 8 years prior to death, when he, for the first time, began to have headaches around the right eye, difficulty in speaking, dizziness, numbness of the right leg and transient loss of consciousness. Work-up at that time included a spinal tap, skull X-rays, EEG, bilateral carotid angiograms and pneumo-encephalogram, all of which were negative. The following year, a CSF protein was found to be elevated to 78 mg.%. There were 7 WBC's, 5 RBC's and a glucose of 76 mg.%. He was maintained on Dilantin 100 mg. t.i.d. He continued to have occasional seizures the following 6 years. Two years prior to death, he developed some features of Parkinson's disease with reduced facial expression and rigidity. Twenty-one months prior to his death, he was hospitalized for seizures, transient right hemiparesis and aphasia. The EEG showed left fronto-temporal slowing, but a brain scan was negative. A left carotid angiogram was interpreted as showing ventricular dilatation. Pneumoencephalogram was interpreted as showing paraventricular atrophy and bilateral cortical atrophy with a delayed 24 hour air study showing a large left parietal parasagittal collection of air. He recovered from this episode with clearing of the hemiparesis and aphasia and was maintained on Dilantin. Five months prior to his death, he was admitted with status epilepticus. At this time, he responded only to pain. EEG showed a slow focus over the left fronto-temporal area. Now the brain scan revealed a large concentration of isotope uptake in the left temporal region. Later, he again developed a right persistent and progressive hemiparesis, global aphasia and bilateral horizontal nystagmus. There was cog-wheel rigidity in both wrists and symmetrical deep tendon reflexes. No abnormal reflexes were present. CSF at this time showed an opening pressure of 190 mm. H_2O, was slightly cloudy, xanthochromic, with protein of 440 mg.% and sugar of 116 mg.%. He developed fever and died in coma.

Necropsy - showed the main abnormality to be in the central nervous system. There were massive random, recent intracerebral hemorrhages with secondary compression and hemorrhages in the brain-stem.

Gross Kodachrome photographs will be shown.

The slides distributed are stained with Hematoxylin and Eosin.

Point for Discussion:

1. What is the nature of pathologic process and why?

2. Possible etiology?

3. Why is the CNS the predominantly involved organ in this case?