Lead encephalopathy simulating a cerebral neoplasm in an adult

Case report

JAMES M. POWERS, M.D., STEPHEN E. RAWE, M.D., AND GARNETT R. EARLYWINE, M.D.

Departments of Pathology and Neurosurgery, Medical University of South Carolina, and Veterans Administration Hospital, Charleston, South Carolina

The clinical and pathological findings in an adult with lead encephalopathy due to moonshine consumption are presented. The remarkable focality of the edema led both the clinicians and radiologists to consider this a cerebral glioma.

KEY WORDS • pseudotumor • lead • lead encephalopathy • neoplasm

LEAD encephalopathy, previously believed to be rare in adults, is now being recognized with increasing frequency in this age group. It is usually due to the consumption of illicit moonshine whiskey and presents as diffuse cerebral edema with increased intracranial pressure. Lateralizing neurological signs have also been observed in 35% of adult patients with this disease. Despite these lateralizing signs, however, focal lesions have not been identified with angiography, brain scan, echoencephalogram, electroencephalogram, or postmortem examination. We are reporting the clinical profile and demonstrating focal pathological lesions in an adult with lead encephalopathy which presented as a cerebral neoplasm.

Case Report

This 47-year-old right-handed, non-hypertensive black man had a 24-year history of excessive alcohol intake, most of which was moonshine whiskey. For the past 3 years, he increased his consumption of moonshine whiskey to approximately 1 quart per day. During that time he experienced an occasional generalized seizure that was controlled by intermittent Dilantin (sodium phenytoin) medication. Two weeks before admission he had another seizure, this time with a mild residual right hemiparesis. This hemiparesis increased until the day of admission. He was admitted when a right-sided focal seizure became generalized.

Examination. In the emergency room his blood pressure was 160/100 mm Hg, pulse rate was 100, and temperature was 97.2°F. His neck was supple, and there were no signs of head trauma. No bruits of the extracranial neck vessels were heard. The remainder of the general physical examination was normal.

On neurological evaluation the patient was unresponsive, in status epilepticus, with a mild left anisocoria, no evidence of papilledema, and conjugate deviation of the eyes to the right. Seizures were controlled with Valium (diazepam) and Dilantin. Routine
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skull, cervical spine, and chest x-rays were normal. Arterial blood gases, blood glucose, and serum studies of electrolytes, calcium, and toxicology screen (heavy metals not included) were normal. The patient had a hypochromic, microcytic anemia with a hemoglobin of 9.7 gm% and hematocrit of 26%. Basophilic stippling was observed.

After the cessation of seizures, the patient never regained consciousness and continued to display a mild left anisocoria. In addition to this, a right central facial palsy, right hemiparesis with a Babinski response, and semipurposeful left-sided movements to painful stimulation were observed. An emergency computerized axial tomography scan was carried out with enhancement to rule out a left-sided supratentorial mass lesion. The scan demonstrated a left-to-right ventricular shift with an increased density in the gyral patterns of the temporal and parietal lobes on the left. Subsequent left carotid angiography also showed evidence of a mass lesion primarily in the left temporoparietal area with extension into the posterior frontal lobe. This was interpreted as an extensive glioma.

Operation. The patient's neurological condition deteriorated further and an emergency craniotomy was performed. At surgery the left temporal, parietal, and posterior frontal lobes were found to be full, but there were no obvious cortical or gyral discolorations or distortions. No mass could be palpated. A ventricular needle was inserted into the temporal, parietal, and frontal lobes in various directions without encountering any cystic lesion or change in consistency of the white matter. A left anterior temporal lobectomy was performed for decompressive purposes. Although the white matter appeared to be somewhat necrotic and edematous, no other change in its consistency was observed. Frozen section was nondiagnostic. Postoperatively the patient's condition did not improve, and he died 2 days after surgery.

General Postmortem Findings. Significant gross observations were confined to hepatomegaly (2110 gm) and hepatic congestion, marginal left ventricular hypertrophy, and minimal renal arteriolonephrosclerosis. Microscopic examination of the affected organs revealed chronic passive congestion of liver, mild hemosiderosis of spleen and liver, and mild hyaline arteriolosclerosis. More importantly, the nuclei of hepatocytes and proximal convoluted tubules contained eosinophilic, acid-fast and aldehyde fuchsinophilic intranuclear inclusions typical of lead intoxication.

Neuropathological Findings. The brain weighed 1110 gm. The cerebral hemispheres...
were diffusely swollen with the left posterior frontal, parietal and, temporal lobes demonstrating a conspicuously greater degree of gyral flattening and sulcal narrowing. The latter area was fluctuant, but not discolored nor distorted. Inspection of the inferior surface of the left cerebral hemisphere revealed loss of the anterior tip of the temporal lobe with hemorrhagic necrosis of the adjacent anterior temporal lobe. The left uncus was herniated toward the midline and was notched. Horizontal sections of the brain disclosed a soft and wet enlargement of the left centrum semiovale, particularly in the posterior frontal and parietal lobes (Fig. 1). There was blurring of the gray-white junction. The subcortical white matter also demonstrated petechial hemorrhages and greenish discoloration. The left lateral ventricle was partially compressed; the basal ganglia were unremarkable. A single secondary hemorrhage (Duret) was also noted in the upper midbrain at the level of the red nucleus. The microscopic features of the edematous left cortical mantle consisted of perivascular, inspissated protein droplets, a few microglial cells, many reactive astrocytes, petechial hemorrhages, acute neuronal necrosis, severe neuronal loss, perivascular and perineuronal clear spaces, and endothelial swelling found particularly in the deeper layers. The white matter exhibited similar glial and vascular changes with a severe edematous alteration. This consisted of separation of myelinated fibers by clear spaces, often containing extravascular protein droplets (Fig. 2) and reactive astrocytes. Myelin stains of the white matter confirmed the preservation, but distortion, of individual myelin sheaths with a marked loss of overall staining. The Bodian axonal stain demonstrated frequent, periodic constrictions in axons. A small amount of myelin debris and oligodendrocytic pyknosis was also present. Corresponding sections from the contralateral hemisphere revealed the same perivascular protein droplets, but a much milder reactive astrogliosis (mostly subpial), microgliosis, neuronal loss, and edematous change. The white matter was minimally involved. The remaining portions of the neuraxis contained scattered microglial-astrogliotic clusters (glial shrubs), often associated with protein droplets in the cerebellum and brain stem. Perivascular lymphocytes were infrequently observed, usually in the brain stem. Although the cerebellum did exhibit a substantial reduction in Purkinje cells with a mild Bergmann's gliosis, no other significant alteration in the cerebellum was observed. Endothelial swelling was present, predominately in the edematous left cerebrum, but true endothelial proliferation was not identified. Sections of cauda equina demonstrated focal areas of demyelination with axonal preservation (segmental demyelination).

Other Studies. After the microscopic sections of brain were examined, formalin-fixed pieces of brain from the severely edematous left cerebrum and corresponding sections from the right cerebrum were analyzed by atomic absorption spectrophotometry for lead content. Results in μg/100 gm wet and dry weight were: right cerebrum 322 (wet), 1709 (dry); left cerebrum 181 (wet), 904 (dry); normal brain <80 (wet), 780 (dry). Formalin lead was only 3 μg/dl.

Discussion

Both the clinical profile and the neuroradiological studies pointed to a mass lesion in this patient's left cerebral hemisphere. The history of moonshine consumption was not obtained until after his death. Even if lead intoxication had been entertained during this patient's course, it is unlikely that the diagnosis would have been seriously considered. Lateralizing signs are not rare in adult lead encephalopathy, but a clinical presentation such as this is most unusual. Moreover, focal abnormalities in the neuroradiological studies mitigate strongly against the diagnosis of lead encephalopathy.

The diagnosis was suspected only after the brain microscopic sections were examined and was confirmed by the demonstration of elevated brain lead. The levels of "normal" brain lead vary somewhat, but the values obtained in this patient's brain tissue were clearly elevated, even after 10 days fixation in formalin. The hematological finding of a hypochromic microcytic anemia and basophilic stippling, as well as the intranuclear lead inclusions in liver and kidney are significant pieces of supporting evidence.

Pathological verification of focal abnormalities in lead encephalopathy is extremely rare. Most large series consistently
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emphasize the generalized nature of the brain swelling, if there is any at all. The cause for the focality of the edema in our patient remains unclear, but it correlates well with the clinical profile, and provides a possible pathological substrate for the lateralizing clinical signs seen in a substantial number of adults with lead encephalopathy. The course of the present case also emphasizes the fact that chemical agents, such as lead, can induce toxic lesions in the brain which may simulate a cerebral neoplasm.

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References


Address reprint requests to: James M. Powers, M.D., Department of Pathology, Medical University of South Carolina, 80 Barre Street, Charleston, South Carolina 29401.