Heterogenous Uptake on Brain SPECT

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71-YEAR-OLD MAN with coronary artery ${f A}$ disease and hypertension was admitted with a three-day history of fever, productive cough, and mental status changes. He had a past history of steroid-dependent chronic obstructive pulmonary disease (COPD) and a positive PPD with antituberculosis therapy. He had smoked three packs of cigarettes daily for 30 years and was a retired roofer who had been exposed to asbestos. Physical exam on admission revealed a lethargic individual with diffuse wheezing and an enlarged prostate. His blood pressure was 193/66 mm Hg. The chest radiograph on the day of admission showed a left-lower-lobe infiltrate with effusion. PPD was positive on the 4th hospital day. Laboratory findings included WBC 15.2 with 8 bands, Hct 43.7, sodium 130, potassium 3.4. Arterial blood gas data included pH 7.52, PCO₂ 40, PO₂, 56, and oxygen saturation of 92%. The patient was started on Claforan and INH without alleviation of fever and confusion. A second chest radiograph, on the 7th hospital day, showed new right-and left-lower-lobe infiltrates. On the 10th hospital day, the patient developed fever, confusion, headache, nausea, and back pain. On the 12th hospital day a lumbar puncture revealed 40 WBC with 38% neutrophils, 62% monocytes, and no bacteria. For suspected bacterial meningitis the patient was treated with ampicillin and Claforan without improvement. Repeated lumbar puncture on the 16th hospital day showed 7 WBC and 128 RBC, positive for mycoplasma titers and cold agglutinins. Rheumatoid factor, ANA, lyme, legionella, RPR, and Mono TE tests, were all negative. CT studies performed on the day of admission and the 12th hospital day were also negative. Tc-99m HMPAO brain SPECT performed on the 21st hospital day showed irregular uptake throughout both cerebral hemispheres (Fig 1). Surface three-dimensional (3D) displays demon strated scattered multiple areas of hypoperfusion in both cerebral hemispheres (Fig 2). An EEG showed a generalized slowing of background activity and the presence of intermittent delta and theta activity, consistent with encephalopathy. Tc-99m DTPA brain images performed on the 24th day were negative. In addition to fever and deterioration of

mental status, the patient developed abdominal pain. Amylase was 216, and GGT was 276; 2 days later lipase was 305 and amylase decreased to 134. Pancreatitis was suspected and cholescintigraphy suggested chronic cholecystitis. Because the patient became progressively delirious and developed meningeal symptoms, infectious encephalitis versus a toxic metabolic process was considered in the differential diagnosis. The patient died on the 35th hospital day. At autopsy, acute massive intracerebral hemorrhage and a 200-gm of blood clot in the left parieto-occipital lobe was found and considered to be the immediate cause of death. Microscopic exam confirmed cerebral necrotizing angiitis and cerebral amyloid angiopathy. Sporadic medium-to-small meningeal and parenchymal vessels exhibited fibrinoid necrosis of varied severity. Inflammatory cells, mainly lymphocytes, histiocyte, and a few multinucleate giant cells were also present. Occasional, affected vessels contained fibrin thrombi. No features of vasculitis in major basal arteries suggested generalized meningitis or encephalitis. Many of the small and medium-tosmall vessels in the leptomeninges and parenchyma displayed mildly intramural deposition of amyloid, confirmed by Congo-red strains and electron microscopic exam. Other final anatomic diagnoses included chronic microinfarctions of the caudate nucleus and left parieto-occipital cortex, left ventricular hypertrophy, nephrosclerosis, pulmonary edema, hepatomegaly, moderate centrilobular emphysema, bronchopneumonia, and prostate adenocarcinoma.

Cerebral amyloid angiopathy is characterized by the presence of amyloid in the walls of small- and medium-sized arteries and, less often, in the veins of brain parenchyma and adjacent leptomeninges.¹

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Fig 1. Tc-99m HMPAO brain SPECTs ([A] transaxial, [B] coronal, and [C] sagittal sections) show heterogenous pattern of uptake, multiple foci of decreased perfusion, throughout the cerebral cortex.

Cerebral granulomatous angiitis is a rare form of vasculitis. Coexistence of cerebral granulomatous angiitis and cerebral amyloid angiopathy is very rare, only eight cases having been previously reported.¹⁻⁵ Our patient was one of the rare cases of coexistence of granulomatous angiitis and amyloid angiopathy whose brain SPECTs showed cerebral heterogenous uptake throughout. We hypothesize

that narrowing and/or occlusion of small- and medium-sized cerebral arteries, with segmental and focal involvement in granulomatous angiitis changes, resulted in multiple foci of decreased perfusion throughout cerebral cortex on his shown on Tc-99m HMPAO brain SPECT images. Heterogenous pattern or uptake on brain SPECTs perfusion abnormality: multiple foci of decreased perfusion



Fig 1. (cont'd).



Fig 2. Surface 3D displays show irregular, multifocal defects in both cerebral hemispheres worse on the left side. LL, left lateral view; VX, vertex view; RL, right lateral view.

throughout the cerebral cortex may occur in the following disease or status:

Common

- 1. Cocaine abuse⁶
- 2. AIDS dementia complex^{6,7}
- 3. Multiinfarct dementia⁸

Uncommon

- 1. Chronic fatigue syndrome^{7,8}
- 2. Systemic lupus erythromatous⁹
- 3. Lyme encephalopathy¹⁰
- 4. Cerebral angiitis coexisting with amyloid angiopathy*
- *Our case reported here.

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