Case Report: Brain Imaging of Cerebrovascular Disease with I-123 HIPDM

Robert J. English, David Summerville, Joseph F. Polak, and B. Leonard Holman

Harvard Medical School and Brigham and Women's Hospital, Boston, Massachusetts

Cerebrovascular disease is the third most common cause of death in the United States, after heart disease and cancer (*1*). Until recently, nuclear medicine imaging of the brain in the evaluation of cerebrovascular disorders has been limited to compounds that only enter brain matter when there is a disruption of the normal blood-brain barrier. With the advent of x-ray transmission computed tomography, the use of traditional brain scintigraphy for the diagnosis of cerebrovascular disease has undergone a profound decline. Recently a family of I-123-labeled amines has been introduced that readily cross an intact blood-brain barrier and are retained by the cortex (*2,3*).

The amine compounds that perform this function are iodinated analogs of naturally occurring compounds, which act as chemical mediators of brain function. Two agents currently undergoing intensive investigation are N,N,N¹-trimethyl-N¹-[2-hydroxyl-3-methyl-5-iodobenzyl]-1,3-propane diamine (HIPDM) and N-isopropyl-*p*-iodoamphetamine (IMP) (Fig. 1). The kinetics of regional distribution is similar in both compounds, as is uptake, which is observed in the lungs and liver as well as the brain.

The distribution of these compounds in the brain reflects local cerebral blood flow. Regions of decreased or absent blood flow, secondary to cerebrovascular disease, appear as "cold" areas on the scintigraphic image. Changes in the pattern of the distribution of these iodinated amines occur early in the course of a cerebral infarction, well before a defect becomes apparent on the x-ray CT study (*4*).

Case Report:

A 64-year-old woman with no prior history of hypertension or neurologic or vascular disease presented with sudden onset of right-sided hemiparesis, aphasia, and restlessness. Physical examination was consistent with a left-sided cerebral infarction. A CT examination of the head on the day of admission was normal (Fig. 2). The patient was immediately referred to the nuclear medicine division for planar and single photon emission tomography using a rotating scintillation camera (400T/STAR System, General Electric Co., Milwaukee, WI). Twenty minutes after the intravenous injection of 5 mCi of I-123 HIPDM, four standard planar images were collected for 300 K counts each, using a 20% window centered on the 159-keV photopeak of the I-123, and collimated with a low-energy general purpose collimator (Fig. 3). A 360° rotation was performed with 64 steps (40 sec/step) collecting 1000 counts per sec.

Reconstruction of transaxial slices was obtained by a standard filtered back projection technique, using a Ramp-Hanning filter with a 0.5 cycle-per-pixel cut-off frequency. Sagittal and coronal slices were obtained from reconstructed transaxial data

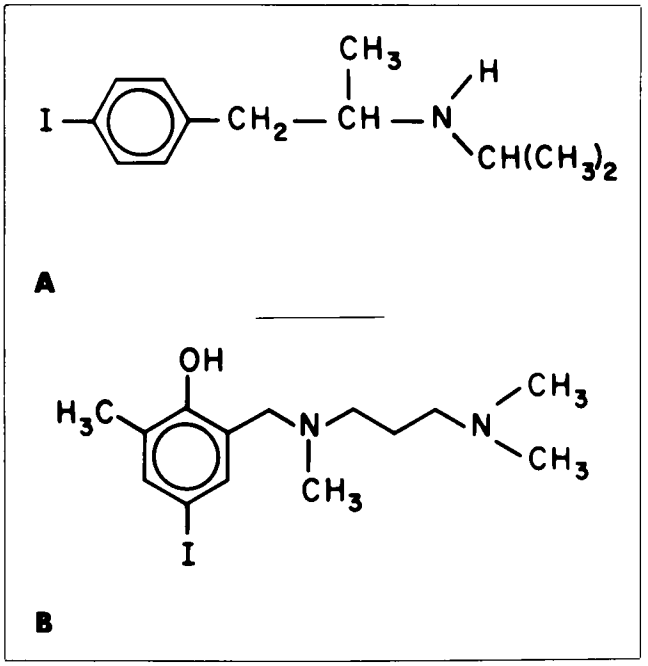

FIG. 1. Chemical structures of (A) HIPDM and (B) IMP.

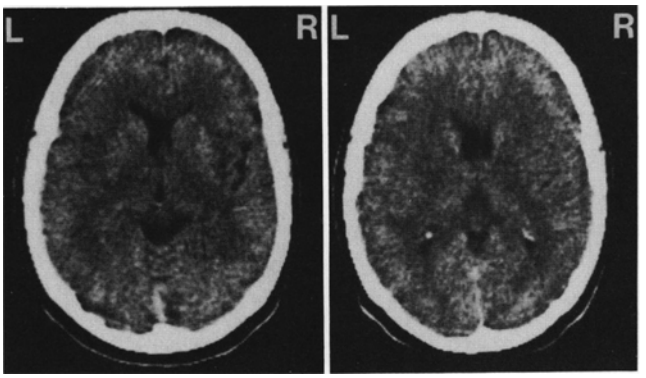

FIG. 2. Midbrain transmission CT images on day of admission.

For reprints contact: Robert J. English, Division of Nuclear Medicine, Brigham and Women's Hospital, 75 Francis St., Boston, MA 02115.

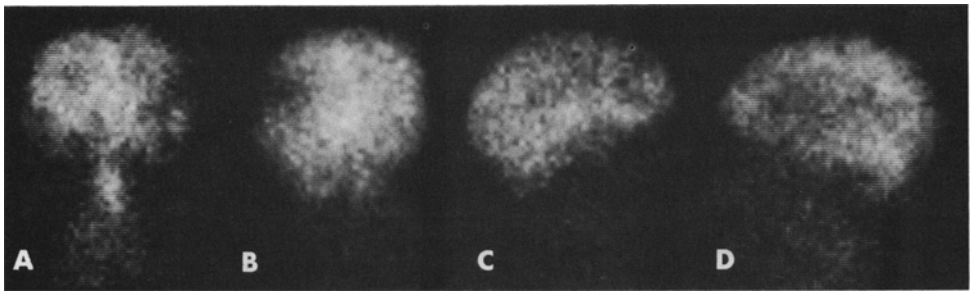

FIG. 3. Planar images of I-123 HIPDM: (A) anterior, (B) posterior, (C) right lateral, and (D) left lateral.

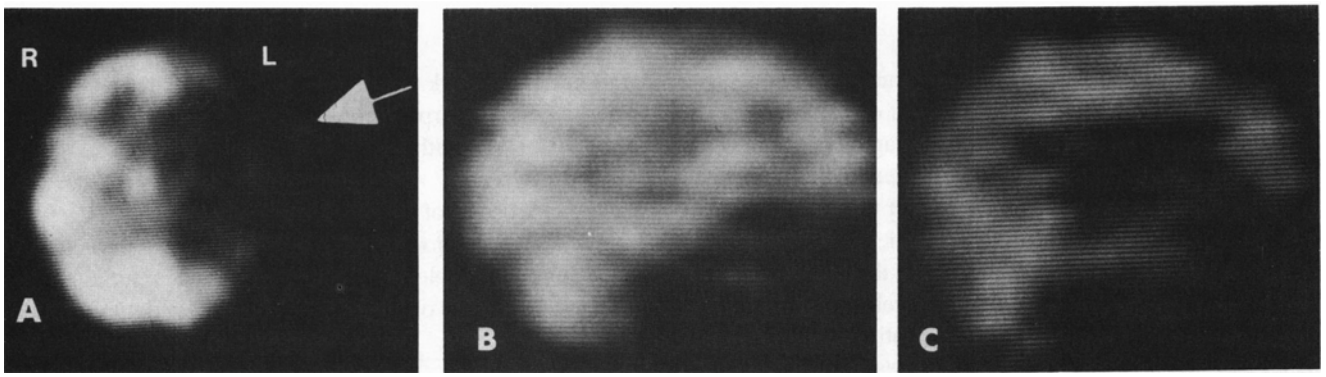

FIG. 4. (A) Midbrain transaxial slice by SPECT imaging obtained at onset of symptoms. *Note:* R and L differ for transaxial SPECT and transmission CT images. (B) right hemisphere sagittal slice, (C) left hemisphere sagittal slice.

(Figs. 4–6). Reconstruction of data in each of the three planes was performed with a thickness of one slice, where one pixel measures 0.63 cm.

A large perfusion defect was present on both planar and tomographic images, involving the left temporal and posterior frontal lobes (middle cerebral artery distribution). Tomography was superior to planar imaging for determining the extent, severity, and location of the perfusion defect. The most intense reduction in flow was in the temporal lobe with blood flow reduced, but preserved on the borders of the infarct.

Subsequent Doppler examination with oculoplethysmography sonography showed probable occlusion of the left internal carotid artery, which was confirmed with carotid arteriography. A repeat CT exam of the head performed approximately two weeks later demonstrated evidence of cerebral infarction in the region of distribution of the left middle cerebral artery (Fig. 6). The patient had only slight resolution of her neurologic dysfunction after several weeks, and was discharged for further rehabilitation.

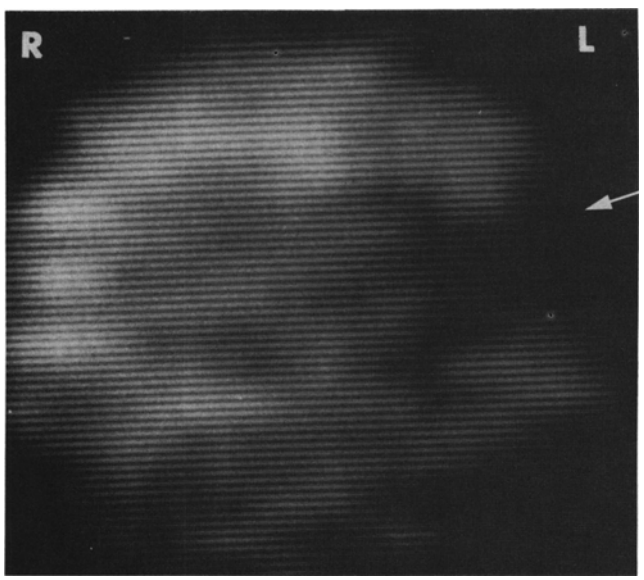

FIG. 5. Midbrain coronal slice (arrow indicates perfusion defect).

Discussion

This case demonstrates the utility of I-123-labeled amine imaging in the early diagnosis of acute cerebral infarction, particularly when the CT findings may be normal for several days after the onset of symptoms. Satisfactory images were obtained by both planar imaging and tomographic reconstructions, however, the lesion appeared better delineated on the tomographic images. Both planar and tomographic images of diagnostic quality can be obtained with I-123 HIPDM, and a commercially available single-head rotating Anger camera system (5).

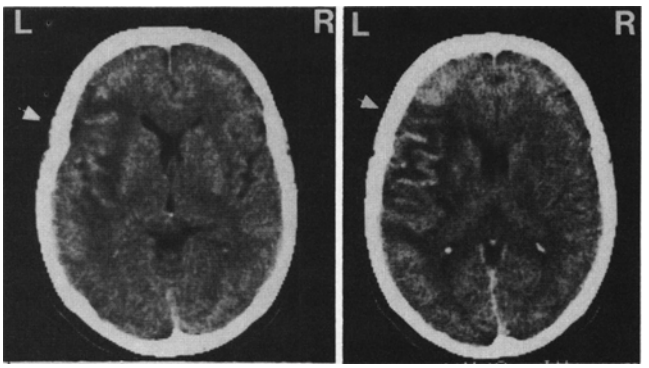

FIG. 6. Transmission CT slice performed two weeks after symptom onset, demonstrating cerebral infarct (arrow).

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