Reversible Increased Technetium-99m-HMPAO Cerebral Cortical Activity: A Scintigraphic Reflection of Luxuriant Hyperperfusion

Arnold Strashun, Eddy K. Dunn, Salil S. Sarkar, William Abel, Gwendolyn Hotson, and Salvatore Sclafani

Department of Radiology, Division of Nuclear Medicine, Division of Interventional Radiology and Division of Neuroradiology, State University of New York, Health Science Center at Brooklyn, New York

A hemiparetic and aphasic patient, 3 days after acute traumatic transection of the left internal carotid artery requiring life-saving total embolic occlusion, revealed ipsilateral increased peripheral hemispheric ^{99m}Tc-HMPAO activity. Ten days postocclusion, HMPAO peripheral cortical flow normalized as hemiparesis and aphasia significantly cleared. The initial lateralized HMPAO hyperactivity pattern may reflect reactive hyperemia, a sign previously identified by contrast angiography and often associated with a better prognosis in evolving CVA. Evanescent peripheral cerebral hyperemia may represent beneficial cortical collateralization of the periinfarct area of a deeper lacunar (white matter) CVA.

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Inoperable basal carotid aneurysms have been treated with intracarotid arterial occlusion. Temporary balloon occlusion has been monitored preoperatively by EEG, clinical neurologic testing, and conventional angiography to predict adequate collateral circulation post-carotid occlusion. This procedure is known as the Balloon Test Occlusion (BTO). Preliminary testing in small patient trials suggests that preservation of distal cerebral perfusion monitored by stable xenon CT quantitation of uptake post-BTO could better predict a successful surgical outcome than EEG or classical neurological signs (*1*).

We would like to describe sequential neurospect ^{99m}Tc-HMPAO cerebral perfusion patterns which parallelled the clinical course of recovery in a patient with acute traumatic left internal carotid artery laceration necessitating emergency total occlusion via arterial embolization. This clinical setting is analogous to BTO.

MATERIALS AND METHODS

Computed tomograms (CT) were obtained on a General Electric 9800 detector with generation of contiguous axial 8.00 mm slice reconstructions (Fig. 1). Tc-HMPAO SPECT scintigraphy was obtained 20 min after intravenous injection of 20 mCi Tc-HMPAO on a triple-headed dedicated neuro-SPECT detector (Triad®) utilizing GAP collimators. Forty point-and-shoot 10-sec planar acquisitions produced raw SPECT data. Chang attenuation correction and count adaptive ramp filters were applied to SPECT raw data to generate orthogonal plane transaxial, coronal, and sagittal 6 mm slice SPECT reconstructions of ~50,000 counts each. Equivalent positioned transaxial slices at the level of the basal ganglia are depicted from Day 3 (Fig. 2) and Day 10 (Fig. 3).

CASE REPORT

A 49-yr-old policeman suffered a gunshot wound which transected his left internal carotid at the siphon, requiring coil embolization within minutes to prevent exsanguination. He was initially comatose. On Day 2, the patient remained comatose. Computed tomography revealed slight edematous swelling of the left cerebral hemisphere (Fig. 1). On Day 3, he was arousable, aphasic, right hemiparetic. HMPAO neuro-SPECT showed hyperperfusion (? luxuriant) global left cerebral cortex (closed arrows) and absent left thalamus and left basal ganglia activity (Fig. 2, open arrows). By Day 10, the patient was oriented to time,

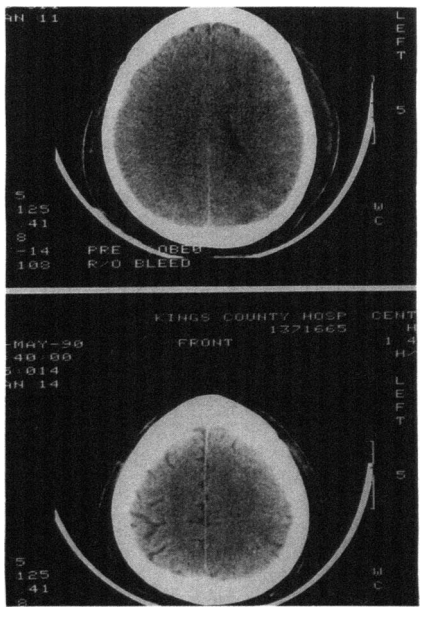

FIGURE 1. Transaxial computed tomography obtained acutely (Day 2) reveals slight edema of left cerebral cortex.

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For reprints contact: Arnold M. Strashun, MD, Division of Nuclear Medicine, Box 1210, SUNY Health Science Center at Brooklyn, 450 Clarkson Ave., Brooklyn, NY 11203.

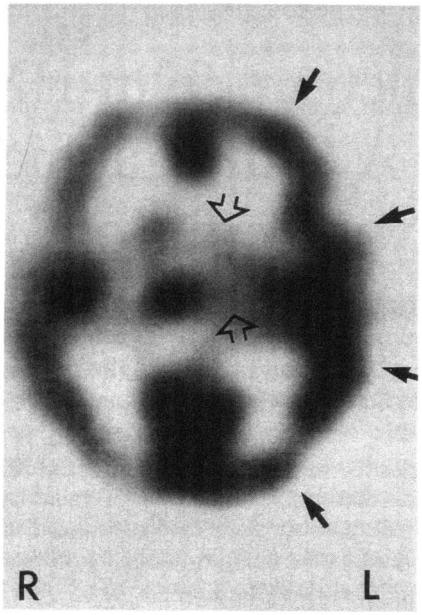

FIGURE 2. Transaxial reconstruction view of Tc-HMPAO neuro spect (Day 3) reveals asymmetric increase in left cerebral cortical flow (closed arrows) as well as absent left thalamus and left basal ganglia perfusion (open arrows) at time of clinical right hemiparesis and aphasia.

space, person and fully responsive. On that day, he then demonstrated mild right hemiparesis and mild expressive aphasia. Repeat HMPAO neuro-SPECT (Fig. 3) showed balanced cerebral cortical flow (reversal of increased left hemispheric activity, partially recovered activity of the left thalamus, and normalized left basal ganglia uptake. Progressive clinical improvement allowed the patient to be discharged 1 mo later. One year later, he had persistent mild expressive aphasia but was otherwise neurologically recovered.

DISCUSSION

Lipophilic, small (<500 molecular weight) radiopharmaceuticals have been developed which readily cross the blood-brain barrier to be incorporated within the myelinated neurons in direct relationship to perfusion. Technetium-labeled HMPAO is one of this class of so-called neuroSPECT agents that reflect grey matter perfusion and have shown clinical value in the study of CVA, epilepsy, and dementia reflecting the site and degree of increase or decrease in regional cerebral grey matter flow. HMPAO functions much like a tagged microsphere in acutely reflecting regional cerebral blood flow (rCBF) to target grey matter within minutes of injection, i.e., an acute phase reactant or indicator of flow (*2,3*).

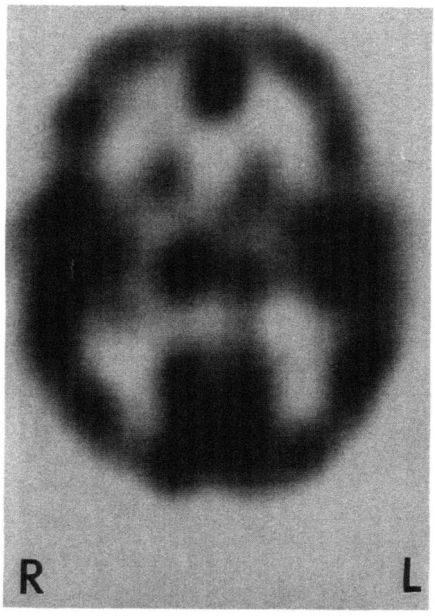

FIGURE 3. Repeat Tc-HMPAO neurospect (Day 10) reveals balanced cerebral cortical flow as well as normalized left basal ganglia activity and partially recovered left thalamic flow. Patient has major improvement in aphasia and right hemiparesis.

We describe a case of increased cerebral flow in an area (the left internal carotid watershed) that clinically was anticipated to demonstrate decreased or absent flow by all clinical indicators and by the body of experience of the study of acute CVA with HMPAO SPECT: classical CVA should produce both white and grey matter infarct with absent flow (HMPAO activity) to the targeted grey matter (*3*). Demonstration of what proved to be an acute and reversible hyperperfusion in the occluded left internal carotid watershed suggested luxuriant or collateral flow via intact large caliber vessels presumably via the circle of Willis from unoccluded posterior vertebral basilar and right internal carotid vessels. The unilateral absence of flow to the thalamus and basal ganglia on the ipsilateral occluded left internal carotid side suggested acute loss of flow to vessels (thalamostriate branches) supplying these arteries.

The clinical course of recovery in this patient paralleled improved uptake of HMPAO by the left thalamus and basal ganglia, implying that the initial clinical deficit was probably flow-related to compromised deeper structures. The presence of an evanescent hyperperfusion surrounding the deep left basal structures at risk was first described in the angiographic literature as luxuriant perfusion and chronologically seen to peak at 2 to 5 days post-acute CVA (*4*). This was consistent with the clinical time course of our scintigraphic findings which could best be explained by classic cerebral ^{133}Xe blood flow studies by Lassen et al. as a vasodilitatory hyperperfusion mechanism felt to be an autoregulation effect of local cerebral hypoxia and especially hypercapnia with secondary intracellular acidosis (*5*). This acidosis in noninfarcted vessels can produce as much as 40% augmentation in rCBF. It might be inferred that a similar mechanism has occurred in uninfarcted vessels surrounding the left basal ganglia defect immediately following our patient's acute traumatic injury.

Increased peripheral neurospect radiopharmaceutical activity peripheral to a deep vascular compromised structure is an unusual sign not generally seen in acute CVA. When seen, it has been described as a generally fixed, late sign of CVA which is neither early nor evanescent (*6,7*). More commonly, a lacunar infarct, especially when localized to white matter as is seen in hypertensive CVA or other small vessel (watershed) strokes, has been reported to be a scintigraphic "false-negative" with the criterion that

all cerebral perfusion defects produce absence of Tc-HMPAO activity or "cold" defects. The autoregulatory vasodilation of adjacent tissue could produce increased rather than decreased activity as was demonstrated in our case if the adjacent rim of cortical grey matter remains viable as manifest by vasoreactivity. Such vasoreactivity could also prognostically imply possible partial or reversible neurologic deficit as evolved in our patient. Finally, our experience in this case suggests that Tc-HMPAO has potential analogous value in predicting clinical outcome of internal carotid balloon occlusion to treat basilar intracerebral aneurysms after BTO. HMPAO injected at the time of temporary balloon occlusion, if taken up by cortical grey matter within the carotid flow distribution, could imply adequate collateralization to tissue at risk, whereas diminished or absent uptake would suggest a poorer prognosis following permanent carotid occlusion. The rapid uptake of Tc-HMPAO would allow accurate test scintigraphy to be performed outside the catheterization lab after cessation of temporary balloon occlusion without complicating the BTO procedure itself.

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