Focally Increased Activity on Scinticisternography: Report of Two Cases

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Two cases where focally increased activity was noted on scinticisternography are reported. One case involves a documented arteriovenous malformation, and the other, documented embolic vascular disease. After a review of the pertinent literature, various other pathologic entities associated with similar scan findings are described. Possible causative mechanisms are discussed.

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Scinticisternography is an established procedure for the determination of cerebrospinal-fluid flow patterns when shunting procedures are being considered. Less well known, however, are the focal scinticisternographic abnormalities found in various pathologic conditions. We report two cases in which focally increased activity was associated with arteriovenous malformation and embolic cerebral vascular disease, respectively.

MATERIALS AND METHODS

Scinticisternography was performed with ^{99m}Tc-human serum albumin (^{99m}Tc-HSA). Three millicuries of ^{99m}Tc-HSA, diluted to 3 ml with 10% dextrose solution, was injected into the lumbar subarachnoid space. (The dextrose was added to make the solution hyperbaric and to enhance movement of ^{99m}Tc-HSA to the head.) Scintigrams of the head were taken at 1, 2, 4, and 24 hr, and a scintigram of the injection site was taken 1 hr after injection to confirm the subarachnoid location of the radiopharmaceutical.

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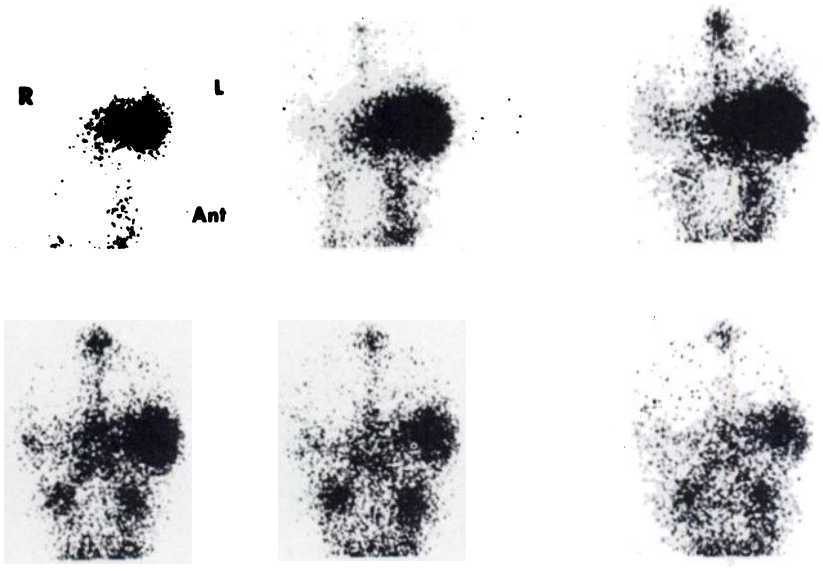

FIG. 1. Case 1. Cerebral bloodflow study shows marked early arterial activity in left temporal region and relatively diminished activity during late venous phase. Findings are typical of large left temporal arteriovenous malformation.

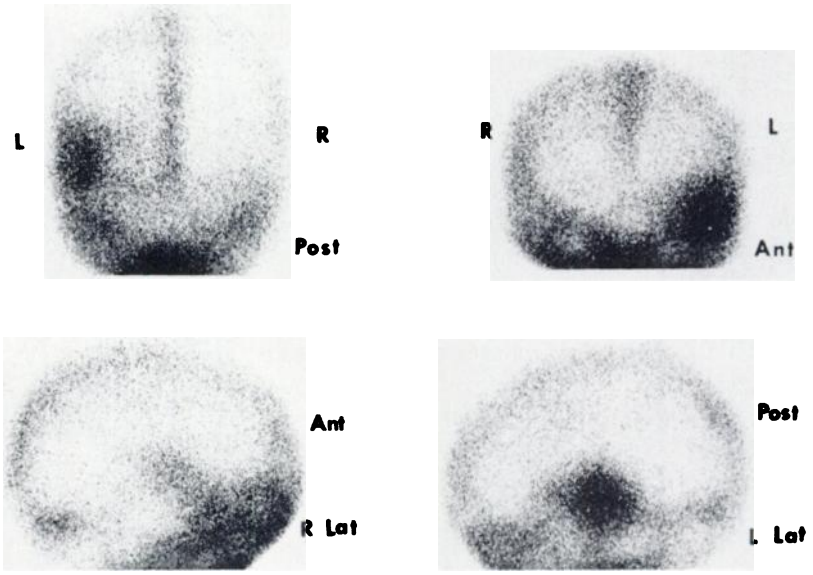

FIG. 2. Case 1. Static brain scan shows focally increased activity in left temporal region corresponding to increased activity noted during bloodflow study

CASE REPORTS

Case 1. A 58-year-old white woman had had grand mal seizures since the age of 16. At age 43 she had a left carotid angiogram, which revealed a large left temporal arteriovenous malformation fed primarily from the left middle cerebral artery. A left temporal craniotomy was performed, but the malformation was judged to be unresectable. Since that time the patient has had no documented episodes of bleeding from the arteriovenous malformation, but she is an unreliable historian. The patient has had numerous psychiatric problems, including progressive dementia. She was admitted to the hospital for the evaluation of her dementia. A cerebral bloodflow study (Fig. 1) revealed marked early arterial activity in the left temporal region, which rapidly diminished during the late venous phase. A brain scan (Fig. 2) revealed focally increased activity in the left temporal region. These findings correlated well with the arteriovenous malformation documented 15 years previously. Scinticisternograms (Fig. 3) all showed focal activity in the left temporal region, best seen on the 4-hr scintigrams. Again, the increased activity corresponded well with the arteriovenous malformation documented by the bloodflow study and the static scan. A ventricular shunting procedure for decompression was not recommended since there was no lateral ventricular activity on the 24-hr scintigrams.

Case 2. A 44-year-old woman with mitral rheumatic heart disease had had four documented episodes of cerebral emboli involving both cerebral hemispheres over a 14-year period. The last episode, which occurred 1 year before the present admission, left the patient with some residual left hemiparesis and hyperreflexia. A radiotracer cerebral bloodflow study at that time showed decreased perfusion in the territory of the right middle cerebral artery. Serial brain scans were normal. Cerebral angiography showed moderate bilateral small-vessel disease due to recurrent emboli, with some leptomeningeal collateralization to the right side from the right posterior cerebral artery.

The present admission was for nausea, vomiting, and dizziness. Neurologic examination confirmed the residual left hemiparesis and hyperreflexia but gave no focal findings. A radiotracer cerebral bloodflow

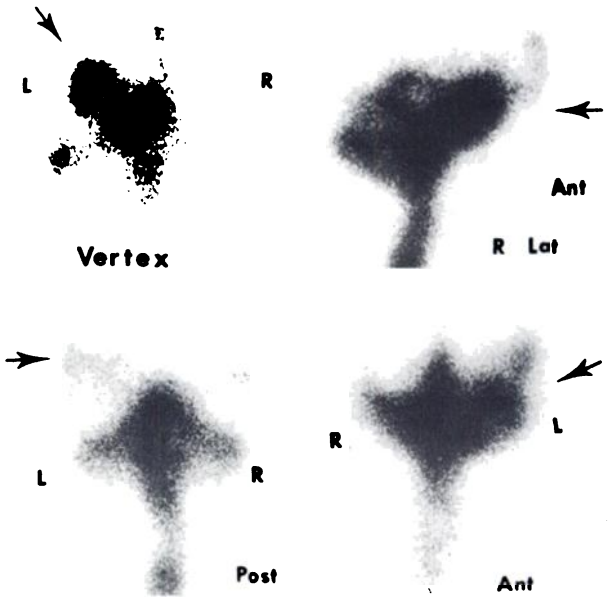

FIG. 3. Case 1. Scintigrams at 4 hr show discrete subarachnoid activity in left temporal region corresponding to findings in bloodflow study and static brain scan.

study again revealed decreased perfusion in the territory of the right middle cerebral artery, and the brain scan remained normal. Computerized axial tomography was performed without contrast enhancement, and a 13-mm-thick slice, centered about 33 mm above the base of the skull (Fig. 4A), revealed a right frontal area with low density (low absorption coefficients) consistent with postinfarction changes of encephalomalacia, edema, or porencephaly of indeterminate age. Scinticisternography (Fig. 4B) revealed a discrete peripheral area of increased activity, noted on the 24-hr scintigrams only, corresponding to the low-density area in the tomographic scan. A ventricular shunting procedure for decompression was not recommended since there was no lateral ventricular activity on the 24-hr scintigrams.

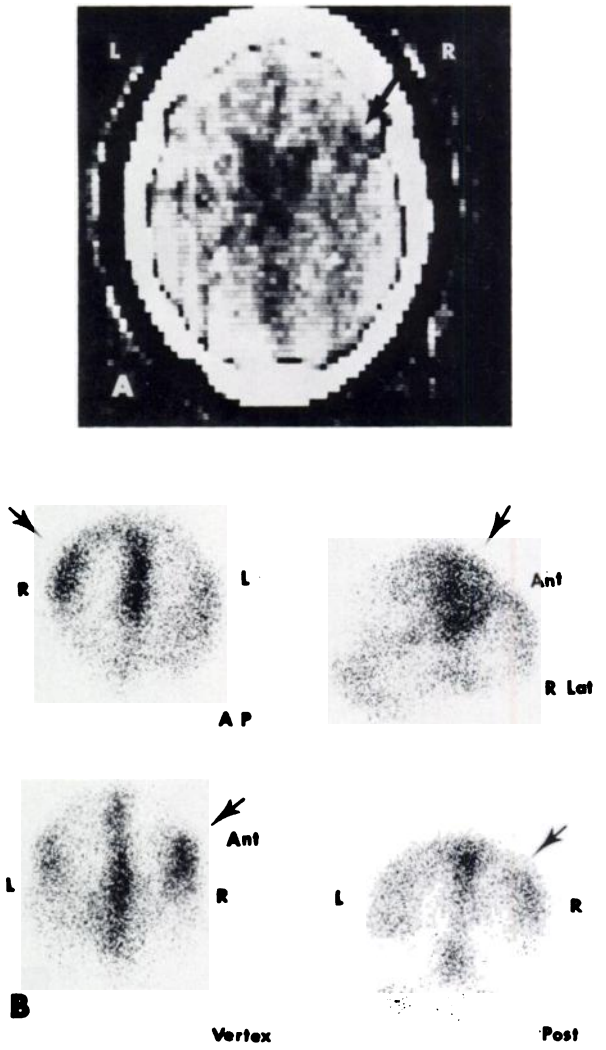

FIG. 4. Case 2. (A) Computerized axial tomographic cut, taken parallel to base of skull and centered about 33 mm above it, shows low-density area in right frontal region consistent with postinfarction changes of encephalomalacia, edema, or porencephaly of indeterminate age. (B) Scintigrams at 24 hr show focally increased activity in right posterofrontal region corresponding to low-density area seen in CAT scan. Note discrete wedge-shaped configuration in right lateral scintigram.

DISCUSSION

To the author's knowledge the literature has reported only one case of focal scinticisternographic activity associated with arteriovenous malformation and only one such case associated with occlusive vascular disease (*1*). Many other focal scinticisternographic abnormalities have been reported: e.g., in subdural hematomas (*2,3*); various types of acquired or congenital cysts (*1,4,5*); meningoceles and myelomeningoceles (*4*); focal atrophy secondary to surgery or other trauma (*1,4*); and degenerative and developmental conditions (*1,4*). In the posterior fossa focal areas of increased activity have been found associated with acquired or congenital cysts, Paget's disease of the skull, achondroplasia, and subdural injection of radiopharmaceutical (*4*).

Two possible mechanisms are postulated for the scinticisternographic findings in Cases 1 and 2:

1. Pathologic alterations in the subarachnoid membranes or tissues causing increased affinity for the radioactive material.
2. Secondary focal atrophy allowing pooling or puddling of the tracer in localized enlargements of the subarachnoid space.

Arteriovenous malformations cause alterations in adjacent tissues by recurrent bleeding, ischemia secondary to rapid arteriovenous shunting, and pulsatile mass effects. Some of these alterations would appear as increased uptake on the static brain scan. Conceivably, in Case 1 the tissues and membranes adjacent to the subarachnoid space were altered, causing changes in the cerebrospinal fluid–brain barrier or increased affinity for tracer (Fig. 3). Such mechanisms have been postulated for the occasional subdural hematoma associated with focal scintigraphic darkening (*2,3*). Perhaps extravasated blood, common to both subdural hematomas and arteriovenous malformations, is the agent responsible for these tissue alterations. The discrete well-defined dark areas seen in Case 1 support the concept of altered tissue affinity for the radiopharmaceutical, since atrophy should be more irregular and poorly defined.

Arteriovenous malformations can cause atrophy of adjacent tissues due to ischemia or pulsatile mass effects. Patient 1 also had a craniotomy adjacent to the malformation about 15 years previously, and some postsurgical atrophy is possible. Pooling or puddling of the tracer adjacent to such regions of focal atrophy has been suggested as a common cause of focal scinticisternographic activity (*1,4,5*). Atrophy, therefore, may have been responsible for the findings in Case 1.

In Case 2 either atrophy or focal tissue alterations could have caused the focal increased activity. The patient had proven embolic cerebral vascular disease, which is more often hemorrhagic than the nonembolic type. Again, blood may have caused the alterations, increasing the affinity of the tissue or membrane for the subarachnoid radiopharmaceutical. Conceivably, ischemic changes could also be implicated. The discrete wedge-shaped picture in Case 2 would favor membrane or tissue changes rather than focal atrophy.

Focal atrophy could, nevertheless, be responsible for the picture in Case 2 since the occlusive right frontal episode occurred about 1 year before the present admission. Old occlusive vascular disease is a known cause of focal cerebral atrophy (*1*). Figure 4A does not show dilatation of the subarachnoid space adjacent to the area of encephalomalacia, but due to instrumental limitations, one cannot exclude such focal dilatation.

Lastly, it is not known whether the different radiopharmaceuticals used for scinticisternography all respond similarly to the pathologic entities discussed above. James et al (*4*) state that "in general the informational content of the studies is not significantly altered by the radiopharmaceutical or the instrumentation. . . ." They found that ^{169}Yb-DTPA, ^{99m}Tc-HSA, and ^{131}I-HSA all showed similar behavior in the cerebrospinal fluid spaces. Cases 1 and 2 of this report confirm that ^{99m}Tc-HSA can show focally increased activity, at least when associated with arteriovenous malformation and occlusive vascular disease.

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