INTRACEREBRAL HEMORRHAGE DEMONSTRATED BY NUCLEAR CEREBRAL ANGIOGRAM: CASE REPORT

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Two intracerebral hemorrhages were detected because of vascular displacement on the radionuclide cerebral angiogram. The static brain images in both patients were normal.

Only rarely will an intracranial space-occupying mass be detected by the dynamic phase of the brain alone (*1,2*). We would like to report two cases of intracranial hemorrhage that were detected by the nuclear cerebral angiogram. The conventional radionuclide image of the brain, dependent on blood-brain barrier alteration, was normal.

CASE REPORTS

Case 1. A 62-year-old man entered the hospital because of a sudden onset of headache, confusion, disorientation, and aphasia. For several years the patient had been treated for hypertension which had been controlled by oral medication.

On admission the blood pressure was 220/120 and neurologic examination was normal. A lumbar puncture was done. The opening pressure was normal but the cerebrospinal fluid was xanthochromic and the protein elevated (104 mg%). An echoencephalogram revealed a borderline left-to-right shift of the midline. An electroencephalogram was interpreted as showing a recent type of encephalopathy involving the deep structures in the left hemisphere, most likely due to a cerebrovascular insult.

The following day the patient became incontinent and began to exhibit a peculiar affect. A nuclear cerebral angiogram and brain scan were done the same day on a scintillation camera. The scintiphotos were obtained after intravenous injection of 15 mCi of ^{99m}Tc-pertechnetate preceded by an oral choroid plexus blocking dose of 200 mg of potassium perchlorate.

The cerebral perfusion study (Fig. 1) was performed with the patient in the anterior position. Serial images were obtained every 1–2 sec, synchronizing hand-pulled Polaroid films with the timer on the camera. The nuclear cerebral angiogram showed a left-to-right shift of the anterior cerebral arteries. Throughout the dynamic study there was diminished activity deep in the left hemisphere, compatible with a large mass effect due to vascular dis-

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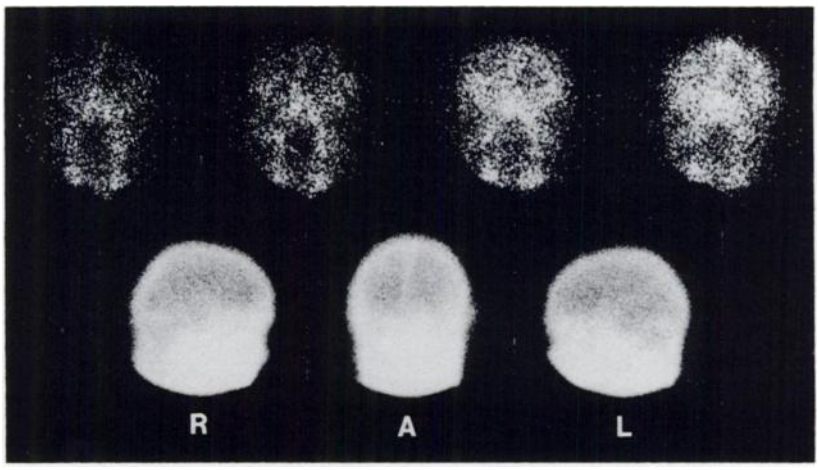

FIG. 1. Case 1. Nuclear cerebral angiograms and brain scans demonstrating left hemisphere vascular displacement on perfusion study. Static scintiphotographs are negative. R, right; A, anterior; and L, left.

FIG. 2. Case 2. Left common carotid angiogram. Anteroposterior view. Anterior cerebral arteries are displaced to right and lenticulostriate arteries are displaced laterally due to left centrosylvian mass.

placement. Ninety-minute postinjection brain scans were normal. The radionuclide studies were compatible with a mass in the left frontal region, most likely due to an intracerebral hemorrhage, a low-grade glioma, or a metastatic neoplasm.

The following day percutaneous left carotid angiography (Fig. 2) revealed a large left frontal pole mass, probably due to an intracerebral hemorrhage or hypovascular neoplasm.

The patient improved slightly but then plateaued. Fourteen days after the onset a craniotomy was performed with evacuation of a large frontal hematoma. The patient steadily improved thereafter.

Case 2. A 70-year-old hypertensive man was admitted to the hospital with a 24-hr history of progressive loss of consciousness. He had been maintained on oral sodium warfarin for approximately 10 months following multiple pulmonary emboli. Two years previously the patient had a right hemisphere cerebrovascular accident.

The blood pressure was 300/140. On neurologic examination a right central nerve deficit, a right hemiparesis, and a right Babinski sign were present. A lumbar puncture was carried out. The opening pressure was 30 cm of cerebrospinal fluid. The fluid itself was grossly bloody and its supernate was xanthochromic.

Over the next 12 hr the patient's condition progressively deteriorated, and he became comatose without response to pain.

A nuclear cerebral angiogram and brain scan were performed with the same technique described previously except that potassium perchlorate administration was omitted. The dynamic study (Fig. 3) showed decreased perfusion of the right cerebral hemisphere in the distribution of the right middle cerebral artery. This was attributed to the patient's previous cerebrovascular accident. Elevation and medial displacement of the left middle cerebral complex were also demonstrated by the nuclear cerebral angiogram. The brain scan begun 1 hr after injection was interpreted as normal. The displaced middle cerebral vasculature and negative static scintiphotographs suggested a hypovascular left temporal mass such as an intracerebral hematoma or perhaps left middle cerebral compression by an acute subtemporal subdural hematoma.

Because of the patient's worsening condition and the findings on the radionuclide brain studies, a left percutaneous carotid angiogram was performed immediately. The frontal projection (Fig. 4) showed a square shift of both anterior cerebral arteries to the right; the lenticulostriate and left middle cerebral arteries were displaced considerably toward the midline. The ascending portion of the middle cerebral axis was stretched and also displaced medially. This study indicated the presence of a large hypovascular left temporal lobe mass.

The patient was then taken to surgery where a left temporal osteoplastic craniotomy was carried out. A very large intracortical hematoma was found extending from the temporal into the frontal lobe. The hemorrhage, which had extended into the temporal

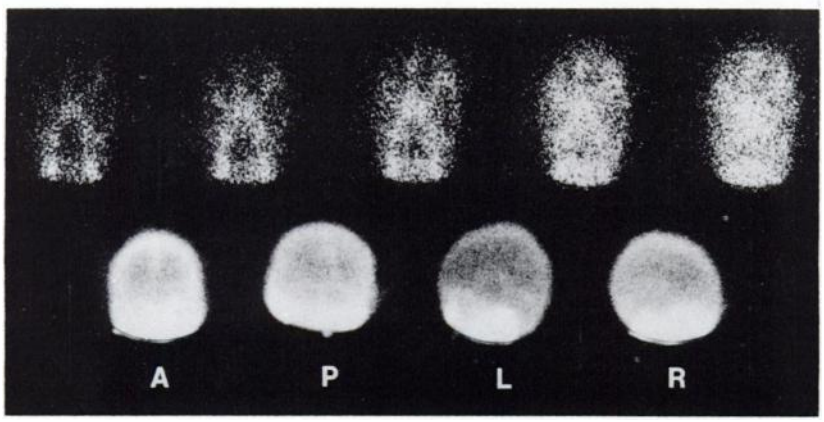

FIG. 3. Case 2. Serial sequential scintiphotos portraying reduction of radioactive transit through right hemisphere. More significant is elevation and medial displacement of left middle cerebral vessels. Static scintiphotos at 1 hr were negative. A, anterior; P, posterior; L, left; and R, right.

FIG. 4. Case 2. Left common carotid angiogram. Anteroposterior view showing square shift of both anterior cerebral arteries to right. Lenticulostriate arteries are displaced medially. Middle cerebral vessels are shifted to left, stretched, and elevated.

ventricular system, was evacuated and a subtemporal lobectomy performed. The patient died approximately 18 hr after surgery.

DISCUSSION

Recent studies have indicated that ^{99m}Tc static brain scans were more often normal than abnormal in patients with intracerebral hemorrhage. In two recent series, Shivers, et al (*3*) and Sharma and Quinn (*4*) reported positive results in four of nine brain images and one of nine patients, respectively.

This is in striking contrast to earlier reports in which the brain scans were performed with ^{197}Hg-chlormerodrin or ^{203}Hg-chlormerodrin. Overton, et al (*5*) and Glasgow, et al (*6*) found an almost 100% incidence of brain scan abnormalities in patients with intracerebral hemorrhage. Ciric, et al. (*7*) found either equivocal or definitely abnormal brain scans in 8 of 13 individuals with intracerebral hematomas.

One of the reasons for discrepancies in the results may be due to the difference in the biologic behavior of the tracers, ^{99m}Tc and radiochlormerodrin (*4*). Thus, the use of ^{99m}Tc may be a disadvantage when attempting to detect intracerebral hemorrhages by static brain scanning. An advantage of high flux, short-lived ^{99m}Tc is that the cranial vasculature can be imaged following an intravenous bolus injection. Use of rapid sequential cranial scintiphotography was responsible for detection of the intracerebral hemorrhages in these two patients. Perhaps this dynamic technique will be of value in detecting other intracerebral masses that displace vasculature but do not produce a focal abnormality on the static brain image.

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