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SPECT Imaging in Cerebral Vasospasm Following Subarachnoid Hemorrhage

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Cerebral vasospasm is a frequent complication after subarachnoid hemorrhage and contributes to overall morbidity and mortality. Arteriography is the standard test for determining the presence of vasospasm. A retrospective review of 16 patients with cerebral aneurysm was undertaken to assess the sensitivity and specificity of SPECT for diagnosis of vasospasm. Fourteen patients were hospitalized after subarachnoid hemorrhage and 2 patients were hospitalized for elective aneurysmal clipping. The patients' condition on discharge was correlated to clinical and SPECT evidence of vasospasm. **Methods:** Vasospasm was defined as the new onset of neurological signs and symptoms not explained by rebleed or hydrocephalus. A total of 20 SPECT studies were performed for 16 patients during their admission and 14 of 16 patients had a single angiographic study. **Results:** Thirteen of 16 patients had 14 episodes of clinical evidence of vasospasm and 14 SPECT studies were performed in these 13 patients. The sensitivity and specificity of SPECT in this retrospective study were 89% (8/9) and 71% (5/7), respectively. Our small sample of arteriograms yielded in comparison a sensitivity of 67% (2/3) and specificity of 100% (9/9). The one false-negative SPECT study occurred in conjunction with the one false-negative arteriogram in the presence of clinical findings consistent with vasospasm. Three false-positive SPECT studies occurred in 2 patients who had perfusion abnormalities in areas of normal CT findings without clinical or arteriographic evidence of vasospasm. Five of 5 patients who died became unresponsive as a result of clinically presumed vasospasm and 4 of 5 of these patients had diffuse or hemispheric SPECT perfusion defects. Of the 11 patients who survived, none became unresponsive; 1 of 11 had positive diffuse or hemispheric perfusion defects. **Conclusion:** SPECT is a sensitive and fairly specific test for corroboration of clinical findings of vasospasm. A negative SPECT study may obviate the need for arteriography. Unresponsiveness is the best predictor of poor outcome; however, hemispheric SPECT perfusion deficits are also associated with poor outcome.

Key Words: cerebral vasospasm; arteriography; subarachnoid hemorrhage

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Cerebral vasospasm after subarachnoid hemorrhage is clinically defined as a worsening of neurologic status that is not explained by hydrocephalus or rebleed. In subarachnoid hemorrhage due to ruptured aneurysm, the most significant cause of disability and death is cerebral vasospasm, which accounts for 33.5% of the combined mortality and morbidity. Among patients with vasospasm, about half will be disabled, the other half will die. It is estimated that 28,000 people in North America will sustain rupture of an intracranial aneurysm each year; by extrapolation from a cooperative study (*1*), this indicates that 4000 people per year will die or become disabled because of vasospasm.

The arterial spasm is presumed to be secondary to oxyhemoglobin released from blood clots adjacent to the adventitia. Vasospasm associated with subarachnoid hemorrhage generally occurs 4–14 days postbleed. Vasospasm before this time may be secondary to an earlier small (sentinel) bleed (*2*).

Treatment of vasospasm to prevent cerebral infarction involves volume expansion and the administration of hypertensive agents (*3*). Aneurysmal clipping is the treatment of choice for subarachnoid hemorrhage; however, this procedure may be contraindicated in the presence of vasospasm. Doppler ultrasound, although very effective in larger vessel spasm, is less successful in detecting spasm of smaller vessels (*4*). Cerebral arteriography has been considered the technique of choice, but it has the potential of triggering an ischemic event in the presence of vasospasm. Cerebral SPECT, using perfusion agents, is a promising noninvasive procedure for the evaluation of vasospasm. In this article, we present a retrospective comparison of clinical assessment, cerebral arteriography and SPECT in the evaluation of the course and outcome of cerebral vasospasm in subarachnoid hemorrhage.

MATERIALS AND METHODS

Patient Population

Sixteen patients (12 women, 4 men; age range 31–73 yr; mean age 50 yr) who were admitted to the neurosurgical service after subarachnoid hemorrhage between January 1994 and January 1996 were included in this study. Thirteen patients were admitted within 24 hr of onset of bleeding and the remaining 3 patients were transferred to the Boston Medical Center at 4, 5 and 16 days after bleed. All patients presented with symptoms of subarachnoid hemorrhage, including drowsiness, headache, meningismus, confusion and poor responsiveness. Three of the patients also presented with unilateral hemiplegia or hemiparesis. Aneurysm clipping was performed in 11 of 16 patients; 7 of 11 were performed within 5 days of bleed and 4 of 11 were performed between 10 and 17 days after bleed. CT studies were performed during the patients' hospitalization.

SPECT

The patients' heads were gently immobilized and tomographic imaging was performed 1 hr after the intravenous injection of 740–1110 MBq ^{99m}Tc-HMPAO. Sixty-four 40-sec images were obtained over a 360° arc using a single-headed GE 400AC/T camera (Milwaukee, WI) with a high-resolution collimator (FWHM at 10 cm = 0.8 cm). The tomographic data were prefiltered with a Butterworth filter (0.55 cutoff, power of 10) and backprojected with a ramp filter. Sagittal slices were reoriented parallel to the interhemispheric line, and transaxial views were reoriented parallel to the orbitomeatal line. Images were viewed in one-pixel slices (approximately 0.5 cm/pixel). Using a color scale, the cerebellum was assigned a value of 100% perfusion. Cerebral defects were present if perfusion was < 60% of the perfusion of the deep ipsilateral cerebellum. Defects were registered only if they were transcortical, seen in three projections and in at least two slices per projection (*5*). SPECT studies were interpreted by two independent observers. Defects on SPECT studies due to nonischemic abnormalities detected on CT (such as parenchymal bleed and postoperative edema) were excluded from the analysis. However, SPECT defects that were much larger than nonischemic defects were included as suspicious for concomitant ischemia.

CT

All CT scans were performed on a Picker PQ 5000 (Picker International, Highland Heights, OH). Four-millimeter-thick contiguous slices were obtained from the base of the skull to the level of the tentorium followed by 8-mm contiguous slices to the vertex. The images were filmed at standard brain window settings (level = 40, window = 80). The images were evaluated for the presence of parenchymal abnormalities. All CT scans were read by two neuroradiologists.

Arteriography

Cut film and digital subtraction arteriography were performed using selective catheterization of bilateral internal carotid and vertebral arteries. Images were evaluated for the presence of vasospasm in the intradural portions of the internal carotid arteries, basilar artery and the anterior, middle and posterior cerebral arteries. Vasospasm was graded using a four-point scale (none; mild = < 25% narrowing of the native vessel diameter; moderate = 25%–75%; marked = > 75%). All studies were read independently by two neuroradiologists.

Analysis and Statistics

For each patient, the intervals between imaging studies and onset of neurological signs and symptoms of vasospasm were calculated. SPECT results were compared to neurological status at the time of the SPECT study to determine sensitivity and specificity for cerebral vasospasm. Results of SPECT and arteriography in a subset of patients who had these studies performed within 3 days of each other were compared. Patients were divided into subsets based on discharge clinical status (Group 1 = normal neurological status; Group 2 = mild sequelae such as cranial nerve palsy, unsteadiness and mild memory deficit; Group 3 = more serious sequelae such as hemiparesis or hemiplegia; and Group 4 = death). SPECT findings in these clinical groups were compared.

RESULTS

Clinical Course

There were a total of 14 events consistent with clinical signs of symptoms of vasospasm in 13 of 16 patients during the course of their admission; 6 of 14 episodes occurred in patients who developed vasospasm 0–10 days posthemorrhage and before surgery (if performed). Eight of 14 patients had vasospasm 0–9 days postoperatively. Of the total group of 16 patients, 2 patients were discharged without sequelae, 5 patients with mild sequelae, 4 patients with more serious sequelae and 5 patients died. Results are summarized in Table 1.

SPECT Studies

In the 13 patients with clinical evidence of vasospasm, 14 SPECT studies were performed between 11 days before and 20 days after onset of symptoms (median = 3 days; mean = 4 days between SPECT and onset of new symptoms). In the 2 patients who did not develop evidence of vasospasm, SPECT studies were performed within 2 days of subarachnoid hemorrhage. SPECT abnormalities in the location of nonischemic CT findings were discounted. The resulting defects were compared with the presence or absence of clinical evidence of vasospasm at the

TABLE 1
Clinical and Imaging Data for Patients with Cerebral Vasospasm

Patient no.	Aneurysm clipped (day)	Symptoms admission (day)	New deficit (day detected)	SPECT defects (day)	Arteriographic vasospasm (day)	Condition discharge (day)
1	None	Right, VI palsy, slurred speech, meningismus (0)	None	Normal	None	Right VI palsy
2	Left MCA, left Pcom, Acom (3)	Drowsiness, right foot weak, meningismus (0)	None	Normal (2)	None (1)	Mild memory
3	Left Pcom (1)	Lethargy (0)	Lethargy, right hemiparesis (2)	Left basal ganglia, left inferofrontal (5)	None (1)	Dysphagia (28)
4	None	Headache, meningismus (0)	Poor responsiveness (2)	Diffuse reduction (3)	None (1)	Death
5	Right Pcom (19)	Headache, dizziness (0)	Left hemiparesis, dysarthria (8)	Right temporal, right parietal (16)	None (16)	Mild left hemiparesis (25)
6	Acom (17)	Drowsiness, headache, meningismus (16)	Left VI palsy, right arm weak (18)	Left hemisphere (18)	—	Left VI palsy (28)
7	Bilateral callosomarginal (28)	Mutism, paraplegia (4)	None	Bilat frontal* (18)	—	Flat affect, unsteadiness (42)
8	Basilar tip (11)	Drowsiness (1)	III palsy lethargy (11) Right hemiparesis (20)	Normal (16)	None	III palsy, right hemiparesis (36)
9	Right Pcom (11)	Lethargy, leg weakness (0)	Unresponsive (12)	Bilateral post-temporal (2)	None (2)	Death (13)
10	Acom (5)	Meningismus, mild confusion (0)	Fluctuating disorient, mild dysphasia (7)	Bilat frontal, left post-temporal* (10)	—	No deficit (21)
11	None	Meningismus left, III seizure (0)	Confusion, right arm weak (5)	Normal (2)	—	Left III palsy (9)
12	Acom (13)	Meningismus (1)	Lethargy (19)	Left frontal and temporal (11, 2)	None (—, 2)	Mild confusion (36)
13	Acom (2)	Confusion, left hemiparesis (0)	Confusion, left hemiparesis (0)	Normal (1)	None (1)	No deficit (12)
14	None	Unresponsive, right hemiplegia (0)	Poor brainstem function (5)	Left hemispheric* (1)	Moderate right A1 (1)	Death (16)
15	Right Pcom (5)	Drowsy, VI palsy seizure (0)	Unresponsive (5)	Normal (3)	Mild terminal basilar (1)	Death (21)
16	None	Poorly responsive, right hemiparesis (0)	Unresponsive (10)	Left hemisphere (15)	Distal internal carotid, left M1 (7, 17)	Death (25)

*SPECT defects larger than CT abnormalities.
All days are number of days following subarachnoid hemorrhage.
MCA = middle cerebral artery; Pcom = posterior communicating; Acom = anterior communicating; V1 palsy = sixth nerve palsy; M1 = segment of the MCA.

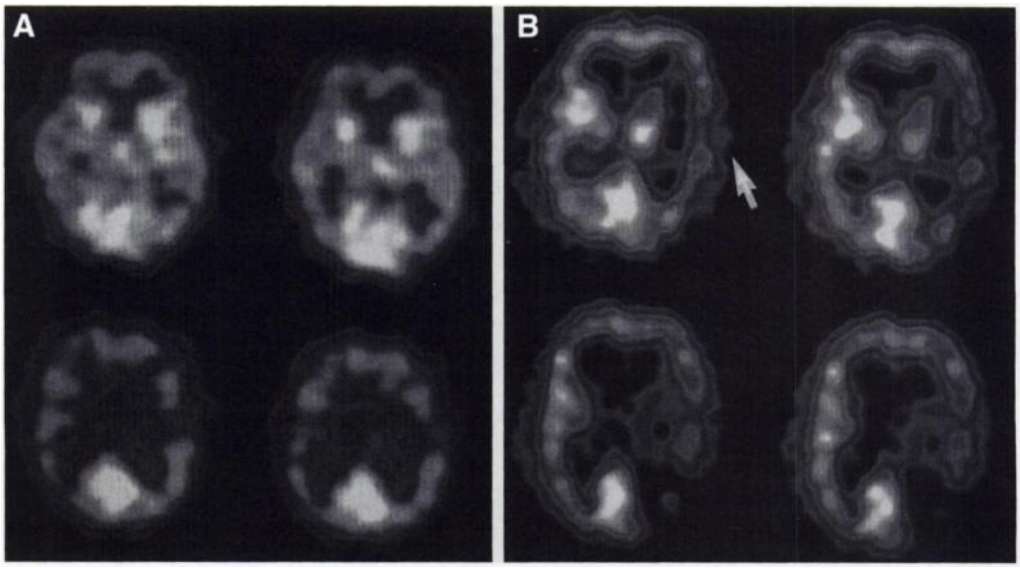

FIGURE 1. (A) A representative transaxial slice from Patient 1. The patient had no clinical evidence of vasospasm. This study was normal. (B) A transaxial slice from Patient 16. This study demonstrates reduced perfusion in the left hemisphere (arrow) consistent with clinical evidence of vasospasm. The patient died 10 days after this study.

time of each study and yielded a 89% (8/9) sensitivity and 71% (5/7) specificity for the detection of vasospasm. Two representative images are shown in Figure 1.

Arteriography Studies

Ten of the 13 patients with clinical evidence of vasospasm received a total of 11 angiographic studies. Arteriography was performed between 10 days before onset of symptoms and 9 days after onset (median = 7 days; mean = 6 days). The 2 patients without evidence of vasospasm received arteriography 2 days after subarachnoid hemorrhage. The majority of arteriograms were performed after subarachnoid hemorrhage, but before clinical evidence of vasospasm. One of the three studies performed after vasospasm was false-negative, yielding a sensitivity of 67%. The overall specificity was calculated at 100%.

Combined SPECT and Arteriography Results

There were a total of 10 paired SPECT and angiographic studies (interval of 3 days or less between studies, pairs occurring before or after clinical symptoms of SPECT). Seven of 10 cases had concordant readings for the presence or absence of vasospasm. The results of the three discordant pairs were compared to clinical findings. One of these three studies had a positive SPECT study and a negative arteriogram that were performed 8 days after the onset of clinical evidence of vasospasm. Two pairs had positive SPECT studies and negative arteriography in the time period before clinical evidence of vasospasm.

SPECT Results Compared to Clinical Outcome

The normal, mild and moderate sequelae groups showed no apparent differences with respect to SPECT perfusion abnormalities. However, when the test results in the group of patients who died were compared with those who survived it was noted that 88% (n = 5) of those patients who died had diffuse or hemispheric SPECT perfusion defects as opposed to 9% (n = 11) of those who survived. All 5 patients who died became unresponsive before their demise (clinically assessed as secondary to vasospasm). None of 11 of the patients who survived became unresponsive during their hospitalization.

DISCUSSION

Detection of Vasospasm

Prompt medical treatment of ischemia secondary to vasospasm can prevent permanent ischemia (*6*). Early detection of cerebral ischemia due to vasospasm after subarachnoid hemorrhage is therefore important.

Angiography has been the gold standard for imaging vasospasm, but it has associated morbidity and mortality. Transcranial Doppler is very promising for the detection of vasospasm in the larger proximal arteries but is less effective in smaller arteries. SPECT is promising for the detection of vasospasm in smaller vessels.

SPECT perfusion studies have been investigated for their use for identifying patients with subarachnoid hemorrhage who are at risk for vasospasm or permanent ischemia. Naderi et al. (*7*) found SPECT to be highly sensitive but with reduced specificity for the detection of borderline areas of ischemia at risk for vasospasm: 22 of 25 (88%) patients had SPECT abnormalities and 68% had angio-positive vasospasm. In our retrospective review of 16 patients, we found good sensitivity (89%) and acceptable specificity (75%) for the detection of vasospasm using HMPAO SPECT studies. Rigorous comparison of SPECT and CT findings was essential to avoid misinterpretation of SPECT abnormalities due to other pathology (such as atrophy, mass lesions, etc.) from those due to reduction in perfusion. Rosen et al. (*8*) emphasized the importance of correlation with anatomical imaging in postoperative studies; 17 of 20 studies in 9 patients in his study demonstrated perfusion abnormalities that could be explained by postoperative changes. The false-negative SPECT scan in our study was corroborated by a false-negative arteriogram for vasospasm. It is conceivable that the clinical vasospasm was very transient and therefore was not detected within 24 hr of the symptoms. The false-positive SPECT scans from our study may represent subclinical vasospasm; however, further research is necessary before this conclusion can be justified.

Prediction of Clinical Outcome

A few studies have looked at the correlation between SPECT readings after subarachnoid hemorrhage and/or surgical correction and clinical outcome. Tranquart et al. (*9*) performed SPECT scans on 26 patients at 3, 8 and 15 days after surgical correction of cerebral arterial aneurysm and found that the studies at 8 days allowed prediction of clinical outcome. Kimura et al. (*10*) identified a subset of patients on Day 8 after subarachnoid hemorrhage with more extensive reduced perfusion reserve after a Diamox challenge in which 52% had a subsequent infarct. Lewis et al. (*2*) studied 10 patients before and after cerebral arterial angioplasty and noted that improve-

ment in SPECT perfusion scores corresponded to improvement in clinical signs and symptoms. In our study, we found an association between the finding of severe perfusion defects and the outcome of death, although clinical findings of unresponsiveness secondary to vasospasm remained the best predictor of death as an outcome. There did not appear to be a correlation between SPECT findings and less severe outcomes; this may, however, be due to our small sample size.

CONCLUSION

The results of this investigation would indicate that SPECT should be considered as the first test for the detection of clinically suspected vasospasm and may obviate the need for invasive studies before the onset of treatment. In addition, SPECT may aid in the identification of those patients with a poor prognosis. In this preliminary study, we found that SPECT brain perfusion studies substantially contributed to the diagnosis of vasospasm complicating subarachnoid hemorrhage and allowed early initiation of specific therapy to treat this serious illness.

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HMPAO Brain SPECT in Acute Carbon Monoxide Poisoning

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Technetium-99m-hexamethylpropylene amine oxime (HMPAO) brain images with fanbeam SPECT, in combination with surface three-dimensional display, were used to detect basal ganglion and cerebral cortex anomalies in the acute phase of carbon monoxide (CO) poisoning. **Methods:** Ten patients, aged 16–29 yr, with acute CO poisoning and no past history of neurologic disorders were enrolled in this study. After oxygen treatment, all 10 patients were investigated using ^{99m}Tc-HMPAO brain images with fanbeam SPECT and surface three-dimensional display. Meanwhile, 6 of 10 patients also received a brain CT scan. **Results:** CT scan findings were negative in all 6 patients. Fanbeam SPECT demonstrated unilateral or bilateral hypoactivity of basal ganglia in 6 patients. Local hypoactivity anomalies were found in the brain cortex of 7 patients, using surface three-dimensional display of the brain. Only 2 of 10 patients had normal ^{99m}Tc-HMPAO brain images. **Conclusion:** This study suggests that, in comparison with traditional brain imaging techniques, ^{99m}Tc-HMPAO brain imaging with fanbeam SPECT in combination with surface three-dimensional display is a better tool for early detection of regional cerebral anomalies in acute CO poisoning.

Key Words: technetium-99m-hexamethylpropylene amine oxime; fanbeam collimator; SPECT; surface three-dimensional display; carbon monoxide poisoning

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Acute carbon monoxide (CO) poisoning is a frequent and often fatal event. Of those who survive, 10%–40% suffer permanent neuropsychiatric complications (*1–3*), the nature of which cannot be predicted in the acute phase by clinical, EEG or brain CT scan findings (*1,3,4*). Brain imaging with ^{99m}Tc-hexamethylpropylene amine oxime (HMPAO) has been used for the assessment of regional cerebral blood flow (rCBF) and has proven accurate for detecting various neurological and psychiatric diseases (*5,6*).

SPECT is essential for depicting brain abnormalities, because it improves image contrast by separating overlapping structures (*7,8*). Particularly when a fanbeam collimator is used to replace the conventional parallel-hole collimator, both system resolution and sensitivity improve by approximately 20% (*9,10*). In addition, if the fanbeam collimator has an FWHM close to 6.5 mm (*11*), deeper lesions within the brain, such as lesions of the basal ganglia, can be clearly demonstrated. However, when brain lesions are evaluated by SPECT, the interpreter must bring together every slice of the transaxial, coronal and sagittal sections to make a whole for accurate localization of lesions. To avoid this, surface three-dimensional images of the brain can be used. Surface three-dimensional images can enhance continuity of structures and improve understanding of spatial relationships (*12–14*). Although a standard surface three-dimensional display cannot depict lesions within the brain, such as those of the basal ganglia (*14,15*), this technique has been clinically applied to the evaluation of rCBF in patients who have suffered stroke (*14–16*), seizure (*16*), depression (*17*) or slow progressive apraxia (*7*).