VALUE OF CEREBRAL ISOTOPE FLOW STUDIES IN TIMING OF SURGERY FOR RUPTURED ANEURYSMS WHEN THERE IS VASOSPASM AND NEUROLOGIC DEFICIT

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There are a variety of guidelines concerning the timing of surgery for intracranial aneurysms when there has been aneurysmal rupture with subarachnoid hemorrhage. However, there are almost no guidelines regarding the timing of surgery when there is angiographic vasospasm. We have observed in the particular clinical situation when there is angiographic vasospasm and a focal neurologic deficit that a cerebral isotope flow study will reveal abnormalities in both the "arterial" and "capillary" phases. Furthermore, serial flow studies can be used to determine the resumption of a normal flow pattern and thus a safer time for intracranial surgery. Since a neurologic deficit may remain fixed because of brain infarction, clinical testing may be an inadequate method of determining focal cerebral blood flow. We recommend a combination of bedside exams, serial isotope flow studies, and interval angiograms as a good method for assessing cerebral blood flow after there has been subarachnoid hemorrhage, vasospasm, and a focal neurologic deficit. Two case reports are indicative of our recent experience concerning eight patients.

The timing of surgery for intracranial aneurysms following a spontaneous subarachnoid hemorrhage involves balancing the unknown daily risk of a catastrophic rebleed against the favorable effect of delaying surgery until the patient has returned to an optimal condition (*1*). The optimal condition is often defined in terms of mental status, overall neurologic function, CSF pressure, CSF color, and systemic arterial blood pressure (*2–5*). In addition, attention is often directed to whether or not there is angiographic vasospasm because patients with vasospasm fare less well than similar patients without vasospasm, regardless of the mode of therapy (*6–12*). There are no clear guidelines regarding the timing of surgery when there is angiographic vasospasm.

Our comments pertain to the particular clinical situation when there is angiographic vasospasm and a focal neurologic deficit following a subarachnoid hemorrhage. The persistence of the neurologic deficit could be due either to ischemia or to the evolution of an ischemic infarct. Clinical testing cannot distinguish between ischemia or infarct in this situation, yet surgery might be delayed with the former but not with the latter condition. We have found that such patients could be followed after the initial radiographic angiogram with serial cerebral isotope flow* and brain scan studies, that ischemia could be distinguished from infarct, and that such distinctions help in selecting the time for surgery.

CASE REPORTS

Case 1. A 32-year-old man was admitted to Harbor General Hospital after 6 days of severe, intermittent, bitemporal headache followed by a syncopal episode on the day of admission. The patient was lethargic. His blood pressure was 120/60, and his pulse was 55. The patient had a stiff neck, a left homonymous hemianopsia, and a left hemiparesis

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* This is also referred to as "intravenous radionuclide angiogram," "nuclear angiogram," or "serial dynamic study" (in contrast to "static" delayed brain scan).

mildly involving the leg and profoundly involving arm. A lumbar puncture was done and clear fluid at an opening pressure of 230 mm H_2O was obtained. A ^{99m}Tc-pertechnetate brain scan* was done and no abnormality was detected; a cerebral isotope flow study* showed decreased perfusion in the distribution of the right middle cerebral artery (Fig. 1, Nov. 10, 1972). The patient gradually regained a normal, alert state and the neck stiffness resolved over 6–10 days; the hemiparesis remained unchanged. One week after admission an angiogram was obtained revealing a right internal carotid artery aneurysm, prominent spasm in the horizontal portion of the right middle cerebral artery, and right temporal lobe swelling (Fig. 1, Nov. 16, 1972). Neurosurgery consultation was obtained: epsilon–aminocaproic acid (24 gm/day) and dexamethasone (4 mg q6h) were begun. Followup cerebral isotope flow studies (Fig. 1, Nov. 20, 1972 and Nov. 29, 1972) showed a considerably reduced perfusion abnormality 20 days after admission and brain scan showed a pattern of increased isotope uptake consistent with an infarct pattern in the distribution of the right middle cerebral artery. At this time the patient had regained more leg strength but remained almost monoplegic in the left upper extremity. A second angiogram, 21 days after admission, was consistent with the recently obtained isotope flow study: the vasospasm was greatly reduced and temporal lobe swelling was gone (Fig. 1, cf. Nov. 29, 1972 flow study with Nov. 30, 1972 angiogram).

The aneurysm of the internal carotid artery was directly clipped on the 22nd hospital day. A small zone of brain infarction above and below the Sylvian fissure was seen. A final angiogram on the tenth postoperative day showed the aneurysm obliterated, the clip in good position, and further improvement in arterial diameter (Fig. 1, Dec. 11, 1972). The patient has regained independent walking ability but still has a left upper extremity monoplegia. Mental function is normal.

Case 2. A 27-year-old woman suddenly lost consciousness at home and struck her head as she fell. She remained lethargic and was taken by her husband to a physician who diagnosed a concussion, and the patient was returned home. She was brought to Harbor General Hospital 4 days later because of continued lethargy, and was admitted on the neurosurgery service. The patient was lethargic but easily arousable and oriented to person, time, and place. The blood pressure was 130/90 and the pulse 64. There was a board-like stiff neck and a mild left

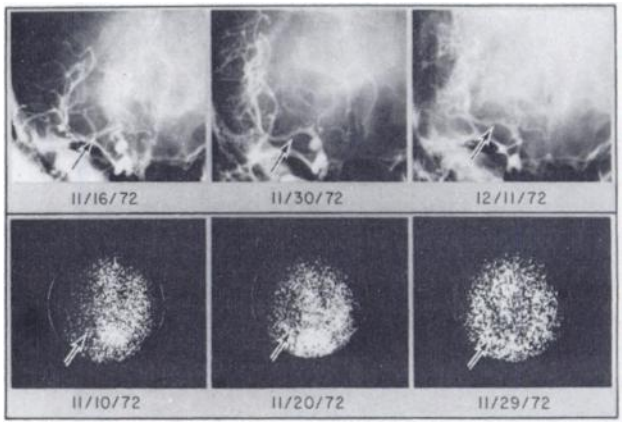

FIG. 1. Representative views from three angiograms and three cerebral isotope flow studies done on patient reported in Case 1. (Angiographic and flow studies were done only on dates indicated in illustration. Each isotope study shown was obtained from 3-sec integration at comparable late arterial phase). Note that cerebral isotope flow study was done first (Nov. 10, 1972), and angiogram was done 6 days later. Internal carotid artery aneurysm is seen on Nov. 16, 1972 and Nov. 30, 1972 and clip obliteration of aneurysm is seen on Dec. 11, 1972. Arrow indicates that portion of middle cerebral artery showing most prominent angiographic and isotope flow abnormalities initially. Only metallic aneurysm clip can be seen in vicinity of aneurysm in Dec. 11, 1972 study.

hemiparesis. A lumbar puncture revealed bloody, xanthochromic fluid with an opening pressure of 350 mm H_2O. Within hours of admission the patient's condition worsened—she became more lethargic and the left hemiparesis worsened with all muscle groups retaining antigravity strength (3/5)* except in the hand where all function was reduced to a flicker movement (1/5). An angiogram done on the day of admission revealed a large aneurysm at the genu of the right middle cerebral artery, temporal lobe swelling, and profound vasospasm in the supraclinoid carotid artery, the horizontal portion of the middle cerebral artery, and the first portion of the anterior cerebral artery (Fig. 2, Jan. 13, 1973). A cerebral isotope flow study (Fig. 2, Jan. 16, 1973) showed greatly decreased perfusion in the distribution of the right middle cerebral artery. The patient was treated with dexamethasone (4 mg q6h), epsilon–aminocaproic acid (24 gm/day), methyl dopa, and frequent lumbar punctures. Over a 10-day period the spinal fluid cleared, mental status returned to normal, and the left hemiparesis virtually resolved except for sustained profound left hand weakness (2/5). Followup cerebral isotope flow studies revealed that the perfusion abnormality was greatly improved on the 13th hospital day (Fig. 2, cf. Jan. 19, 1973 with Jan. 26, 1973). A repeat angiogram on the 16th hospital day showed considerable im-

* All cerebral isotope flow and brain scan studies referred to in this report were done with ^{99m}Tc-pertechnetate.

* Using the standard British Medical Research Council muscle strength scale of 0/5 (no contraction) to 5/5 (normal power).

provement in the vasospasm with only focal lumen narrowing in the supraclinoid carotid artery (Fig. 2, Jan. 29, 1973).

On the 17th hospital day the large aneurysm was dissected free from the middle cerebral artery trifurcation and was clipped. A small zone of infarction above the Sylvian fissure was noted prior to the clipping. The postoperative course was uncomplicated and the patient went home 2 weeks after surgery. She returned 1 month after surgery at which time an angiogram was done: the size and contour of the major intracranial vessels was normal, the aneurysm was obliterated, and the clip was in good position (Fig. 2, March 1, 1973). Improvement in left hand finger function was definite within the first week following surgery and some grip ability had returned by the time the patient went home. Six months following surgery there was no detectable left-sided abnormality except some clumsiness in the left hand during rapid movements.

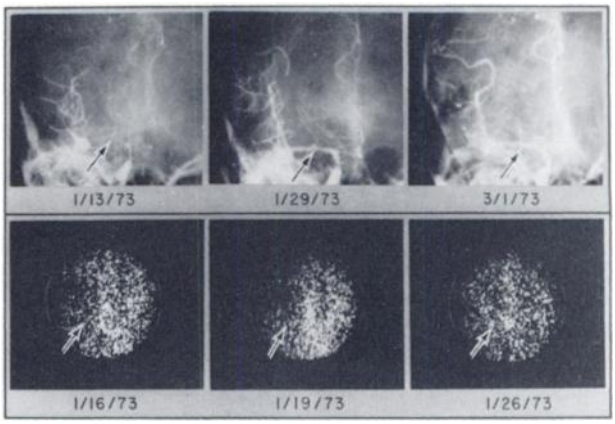

FIG. 2. Representative views from three angiograms and three cerebral isotope flow studies done on patient reported in Case 2. (Angiographic and flow studies were done only on dates indicated in illustration. Each isotope study shown was obtained from 3-sec integration at comparable late arterial phase.) Note that angiographic study was done first (Jan. 13, 1973) and then three cerebral isotope flow studies were done before repeating preoperative angiogram. Middle cerebral artery aneurysm is seen on Jan. 13, 1973 and Jan. 29, 1973, and clip obliteration of aneurysm is seen on Mar. 1, 1973. Arrow indicates that portion of middle cerebral artery showing most prominent angiographic and isotope flow abnormalities initially. Only metallic aneurysm clip can be seen in vicinity of aneurysm in Mar. 1, 1973 study.

COMMENT

Our specific concern is that the combined factors of angiographic vasospasm and a continuing neurologic deficit may be due to brain ischemia. Intracranial surgery during ischemia could be quite hazardous. First, in the early stages of widespread ischemia there is sufficient edema to complicate brain retraction and aneurysm exposure (Fig. 1, Nov. 16, 1972; Fig. 2, Jan. 13, 1973). Second, even if the edema does not handicap brain retraction, there is the risk that the ischemia will be considerably worsened by the techniques used in surgery: retraction, hypotension, vessel manipulation, temporary proximal occlusion, etc. And finally, the bedside examination cannot distinguish between ischemia and infarction. We suggest that the cerebral isotope flow study is an objective assessment of cerebral blood flow that can distinguish whether or not there is continuing ischemia in patients with angiographic vasospasm and a neurologic deficit following rupture of an aneurysm. In both patients presented, the focal neurologic deficit persisted for many days as the cerebral blood flow gradually improved and the day of surgery was eventually selected primarily on the flow data (and the associated repeat angiogram).

Several types of clinically useful information related to cerebral blood flow have been derived from intravenously injected isotopes. Detection of arrival time of the isotope at a cranial counter along with measuring isotope washout has been used to calculate "transit time" (*13*). Further manipulation of such data, i.e., uptake curves and transit time, has been used to calculate actual cerebral blood flow (*14*), and this quantitative approach has been used to assess ischemia of brain and therefore the timing of aneurysm surgery. We have been more interested in the qualitative data derived from gamma camera detection of intravenously injected isotope since this technique permits visualization of an "arterial" and "capillary" phase (*15–17*) and has been used previously to visualize giant aneurysms and arteriovenous malformations (*18*).

Our experience now includes eight consecutive patients in whom we could detect a major abnormality in the cerebral isotope flow study, often in both the "arterial" and "capillary" phases, when there was angiographic vasospasm and a focal neurologic deficit. In patients with recent subarachnoid hemorrhage, angiographic vasospasm, and no neurologic deficit, the isotope flow study is most often normal. Also, we have been unable to detect by a cerebral isotope flow study a perfusion abnormality when a posterior fossa aneurysm rupture has caused vertebrobasilar vasospasm and associated brain stem signs. However, since aneurysms of the anterior portion of the circle of Willis are much more common than those of the posterior fossa, we want to emphasize that cerebral isotope flow studies contribute practical information to the neurosurgeon when such studies are correlated with the clinical status of the patient and with the conventional radiographic angiogram. This information may be helpful in selecting on optimal time for surgery.

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