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DELAYED APPEARANCE OF ANTERIOR CEREBRAL ARTERIES ON ISOTOPIC CEREBRAL FLOW STUDY: A SIGN OF BLEEDING ANTERIOR COMMUNICATING ARTERY ANEURYSM

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Delayed appearance of the anterior cerebral arteries on an isotopic cerebral flow study in association with a right frontal area of decreased activity was seen in a patient with a hemorrhage from a ruptured anterior communicating artery aneurysm. It is suggested that closer scrutiny of the major vessels, in addition to the usual grosser hemispheric observations, would prove this finding to be more frequent. The importance of making this observation of delayed appearance of a major vessel, particularly in conjunction with a hemispheric abnormality, is that it raises the suspicion of an aneurysm as the cause of the cerebral vascular accident. This suspicion alters the further evaluation and care of the typical stroke patient because a lesion potentially correctable by surgery may otherwise be overlooked.

The isotopic cerebral flow study is currently regarded as the most sensitive noninvasive procedure for diagnosing cerebral vascular accident (CVA). In arteriographically proven acute or subacute CVA, the isotopic flow study has been shown not only to identify the abnormality in all patients with positive brain scans but also to diagnose correctly an equal number of patients whose scans are negative. The overall sensitivity of the flow study alone in acute or subacute CVA is 0.83 compared with 0.43 for the brain scan alone (*1*).

The criterion for diagnosing an abnormality based on the flow study is unilateral hemispheric decrease in tracer activity contralateral to the affected side of the body on at least a successive pair of scintiphotos with or without a later phase reversal in asymmetry (*2*). The normal flow pattern is symmetric with the appearance of the circle of Willis at 10.5–12.0 sec, both middle and anterior cerebral arteries (the "trident") at 12.0–13.5 sec, and the venous phase at 13.5–15.0 sec (*1*). Resolution is generally considered too poor to interpret abnormalities in the major vessels unless they are totally absent.

A recent case suggests that diagnostic information is being overlooked by not paying close attention to the trident.

CASE REPORT

A 42-year-old white man was admitted following a postcoital generalized clonic seizure. There was no prior history of seizure, head trauma, or serious illness. On physical examination he was confused and disoriented with a fluctuating level of consciousness. Initially, he had no focal signs but rapidly evolved a left positive Babinski and then bilateral positive Babinskis. His vital signs, fundi, and the remainder of his neurologic examination were normal.

An EEG showed normal background with marked 2–4 Hz right focal slowing. A skull series showed a calcified pineal midline in position. His CBC, electrolytes, and creatinine were normal.

The patient then had a major motor seizure with subsequent bilateral pyramidal tract signs, left greater than right, and then developed papilledema and marked rigidity.

An emergency four-vessel cerebral arteriogram performed several hours after his initial seizure re-

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vealed a 1-cm anterior communicating artery aneurysm with a right frontal avascular mass causing a localized anterior displacement to the left of the anterior cerebral arteries, both of which are supplied by the right internal carotid artery (Fig. 1). Mild hydrocephalus was also noted.

A lumbar puncture immediately after angiography revealed an opening pressure of 190 mm, 40,000 RBC/mm^3, and elevated proteins.

The patient remained clinically stable. An isotopic cerebral flow study (Fig. 2) performed 12 days following admission revealed an area of decreased uptake in the right frontal region corresponding to the mass lesion seen on angiography. Delayed appearance of the anterior cerebral artery group was also noted.

A subsequent flow study and brain scan 22 days after admission were normal and a repeat cerebral arteriogram 4 days later showed diminution in the size of the right frontal hematoma, slight further ventricular enlargement, and no arterial spasm. The aneurysm was operatively coated successfully 32 days after admission and the patient's course has been one of steady recovery.

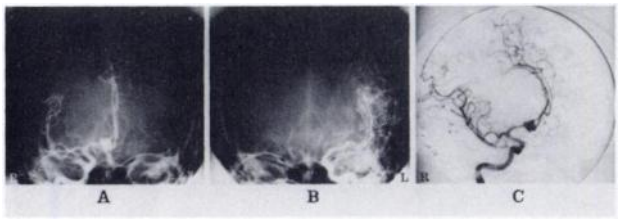

FIG. 1. (A) Anterior projection of right internal carotid artery (ICA) injection showing 1-cm anterior communicating artery aneurysm and left anterior displacement of anterior cerebral arteries both of which are supplied by right ICA. (B) Normal left ICA injection with lack of opacification of anterior cerebral arteries. (C) Second-order subtraction LPO right ICA injection demonstrating aneurysm to better advantage. No arterial spasm is evident on this examination.

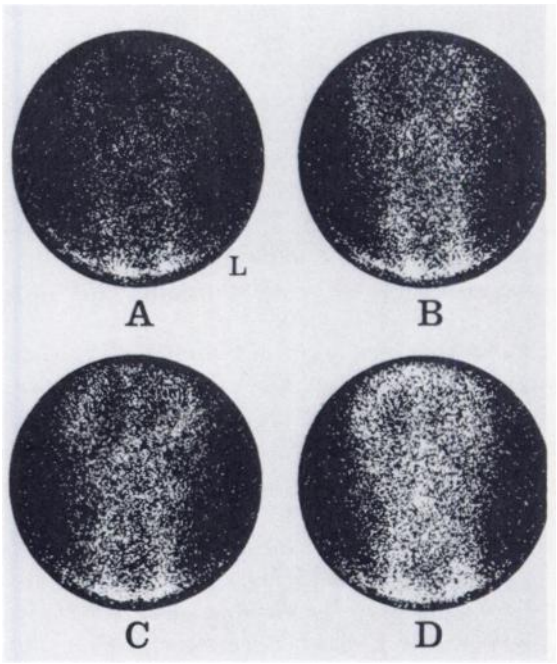

FIG. 2. Hand-pulled Polaroid prints were taken in anterior position every 3 sec for total of 24 sec on Searle Radiographics Pho/Gamma HP after rapid 2-cc bolus injection of 20 mCi of ^{99m}Tc-pertechnetate into antecubital vein. Scintiphotos, 7–9 sec (A) and 10–12 sec (B), show arterial phase. There is decreased tracer in anterior cerebral artery group. In 13–15 sec (C) scintiphoto, anterior cerebral artery group appears late, as early hemispheric asymmetry is evident. Venous phase at 19–21 sec (D) shows persistently decreased tracer in right frontal area.

DISCUSSION

No cases of delayed appearance of the anterior cerebral arteries (ACA) on isotopic cerebral flow studies have been found in reviewing the literature. Because most vascular accidents affecting the anterior cerebral group are unilateral and the resolution limits of the camera do not permit resolving the two ACAs, this "middle prong" of the trident will often appear normal.

On the other hand, 50–80% of nontraumatic intracranial hemorrhages are due to aneurysms (*3*) and the anterior communicating artery (ACoA) is the most common site of intracranial aneurysm (*4*). In view of these observations, this sign probably occurs more often than is recognized.

Delayed appearance is probably the result of arterial spasm. Spasm is maximal (85%) 1–2 weeks after aneurysmal rupture (*5*). This may explain why it is seen on the flow study 12 days after the hemorrhage and not on the arteriogram performed the same day as the hemorrhage or on the arteriogram performed 26 days later.

Delayed appearance of a major arterial group on isotopic cerebral flow study suggests the presence of arterial spasm. This is generally the result of hemorrhage with ruptured aneurysm being the most likely nontraumatic cause. Therefore, in a patient with the clinical diagnosis of CVA, delayed appearance of a major arterial group on isotope cerebral flow study raises the suspicion of the presence of an aneurysm. Unlike a simple CVA, aneurysm often requires operative intervention. Since most simple CVA patients do not undergo cerebral arteriography, we should consider using the isotopic cerebral flow study to screen for aneurysm. The sensitivity of the flow study may not, in fact, be low if it is performed 1–2 weeks after the acute episode and if delayed appearance of major arterial branches is sought.

An unanswered question remains regarding the generalization from this case, i.e., what role, if any, does the fact that both ACAs are supplied from a single internal carotid artery play in producing this sign? Unfortunately, we cannot be certain with-

out greater experience. A ruptured ACoA aneurysm, however, may well induce spasm in both ACAs because of its unique position regardless of the internal carotid artery supply to the ACAs.

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