TRAUMATIC ARTERIOVENOUS FISTULA BETWEEN THE MIDDLE MENINGEAL ARTERY AND THE SPHENOPARIETAL SINUS: A CASE REPORT AND REVIEW OF THE WORLD LITERATURE

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A fistula between a lacerated middle meningeal artery and the sphenoparietal sinus was discovered in a patient who had increased intracranial pressure following trauma.

Only 23 cases of angiographic demonstration of fistulae involving the middle meningeal vessels have been reported in the world literature.[1-12] These fistulae involved the following venous structures: greater petrosal sinus (four cases), middle meningeal veins (12 cases), diploic veins (three cases), sphenoparietal sinus (two cases), and "pterygoid" sinus (one case). Eighty-seven percent of these fistulae were associated with a demonstrable skull fracture. One case was believed to be spontaneous; sixty-seven percent were associated with concomitant hematoma.

CASE REPORT

The patient, a 37-year-old male, was brought to the hospital by ambulance. He was confused and combative. He had a seizure in the treatment area whereupon he gradually became comatose and areflexic. No other history was obtainable. Physical examination revealed a moderately developed, somewhat undernourished male in an unconscious state. His blood pressure was 150/90 mmHg, pulse 116/minute, and his heart was normal. Ecchymosis and swelling were found over both occipitotemporal areas. Neurological examination revealed dilated pupils, bilateral papilledema, blood in both auditory canals, and blood pigment stains behind both ears (positive Battle signs). The patient was unresponsive to deep pain stimuli. Further investigation revealed a 31.3 percent hematocrit, 14,700 white cell count, normal urine, 21 mm/100 ml blood urea nitrogen (BUN), normal blood glucose, and noncontributory electrolyte values. A lumbar puncture was not attempted because of increased intracranial pressure.

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RADIOGRAPHIC FINDINGS AND CLINICAL COURSE

Plain film examination of the skull revealed a linear fracture through the right occipitotemporal area. A right frontal sinus osteoma was also noted. Left common carotid angiography was performed via direct needle puncture, and rapid sequence filming was carried out. The left internal carotid artery (Figure 1) did not fill above the level of the first cervical vertebral body. No evidence of collateral filling of any of the cerebral branches of the internal carotid artery was seen, despite adequate filling of the external carotid circulation. Extravasation of contrast from the left middle meningeal artery into the sphenoparietal sinus is seen in Figure 1. Figure 2 is a close-up showing the middle meningeal artery, the sphenoparietal sinus, and early drainage into the pterygoid plexus (white ar-

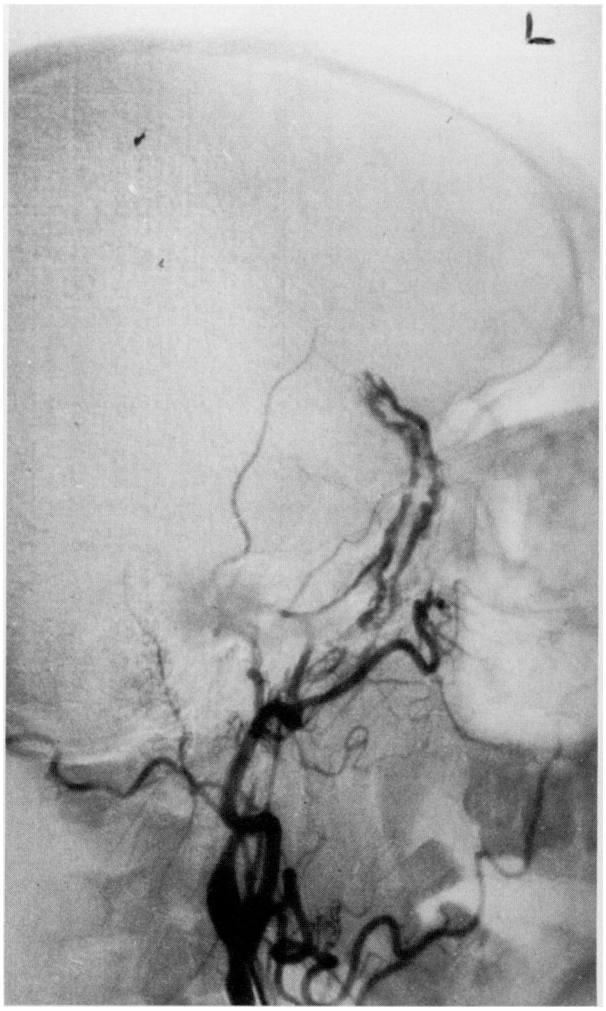

Figure 1. Left common carotid angiogram, lateral view, demonstrating nonfilling of the internal carotid artery and filling of the fistula at 1½ seconds

row). Displacement of the sphenoparietal dural sinus (arrows) away from the inner table of the calvarium is seen on the corresponding anteroposterior (AP) view (Figure 3).

The right internal carotid artery did not fill above the level of the second cervical vertebral body. Again, no filling of any of the cerebral branches of the right internal carotid artery was seen, despite adequate filling of the external carotid circulation.

A craniotomy was not attempted because it was felt that irreversible brain damage had already occurred secondary to increased intracranial pressure. The patient was given intravenous hypertonic invert sugar (mannitol) and corticosteroids for cerebral edema. He did not regain a higher level of consciousness and finally succumbed on the sixth hospital day.

In addition to the radiographically demonstrable epidural hematoma and fistula, the autopsy disclosed bilateral subdural hematomas and intracerebral hemorrhage.

DISCUSSION

Angiographic findings in epidural bleeding were first described by Lohr in 1936.[13] The classical angiographic signs of epidural bleeding are: (1) extravasation of contrast from the middle meningeal artery[13,14]; (2) displacement of the dural sinuses

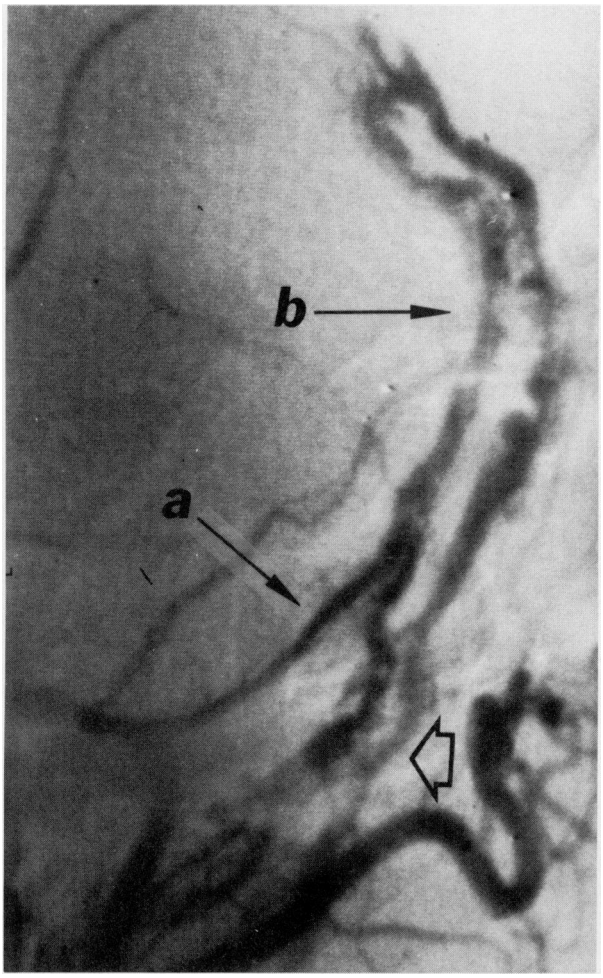

Figure 2. Lateral close-up of the fistula shows the middle meningeal artery (a) filling the sphenoparietal sinus (b), and drainage into the pterygoid plexus (white arrow) at 1 1/2 seconds

from the inner table[13,14]; (3) displacement of the middle meningeal arteries from the inner table; (4) biconvex (lentiform) extravascular space in cranial vault[13,14]; and (5) localized stretching and attenuation of underlying arteries ("compression cone" effect).[14]

Extravasation of contrast from the middle meningeal artery may either be in the form of an amorphous collection of contrast[15] or in a fistula[13,14] with the middle meningeal veins ("tram-trak" sign),[13,15] the sphenoparietal sinus, the greater petrosal sinus,[5] or the diploic veins in the calvarium if the artery is traveling in a bony tunnel at the site of rupture.[13,15] Fistula formation was the first described by Leslie in 1962.[13]

Displacement of meningeal arteries from the calvarium probably occurs with all epidural hematomas but is rarely seen because meningeal arteries are so small and there are usually many superimposed external and internal carotid branch vessels. However, when seen, this sign is pathognomonic for epidural bleeding. Some of the vascular twigs seen above the sphenoparietal sinus (Figure 3) may represent middle meningeal branches displaced away from the calvarium; however, superimposed occipital branches preclude defini-

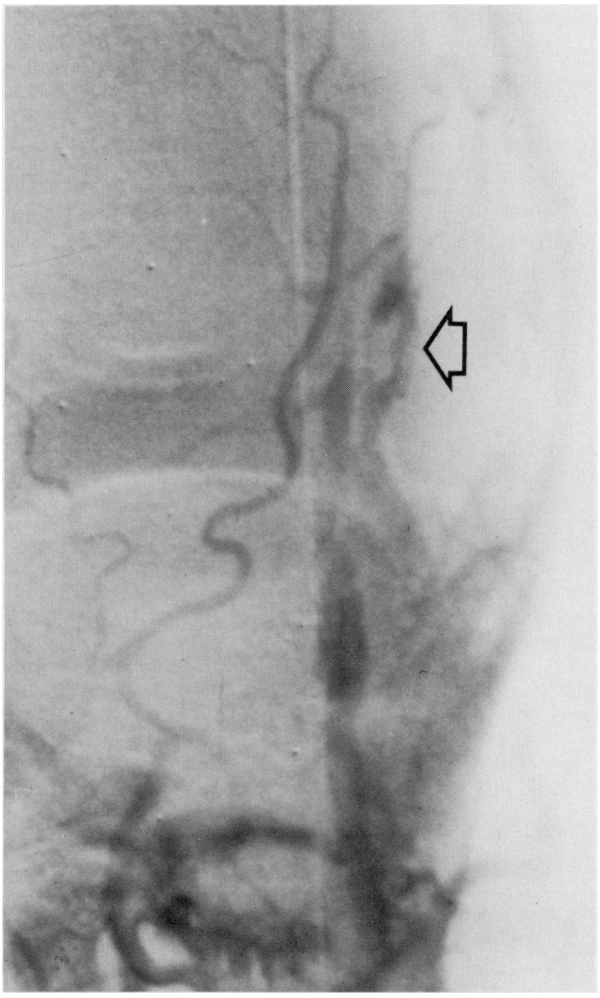

Figure 3. Anteroposterior view showing displacement of the sinus away from the calvarium

tive identification of such vessels in this case.

Lack of internal carotid filling in this patient enables us to see displacement of the dural sinuses away from the calvarium clearly on the AP projection (Figure 3).

A biconvex extravascular space may be seen in patients in whom the cerebral branches of the internal carotid arteries are filled. Localized stretching and attenuation of underlying internal carotid branch arteries also may be seen when these vessels fill.

Lack of filling of the cerebral portions of the internal carotid arteries has been attributed to reduced cerebral blood flow secondary to suddenly increased intracranial pressure. Compression of cerebral veins and capillaries beyond a critical point, causing an acute increase in cerebrovascular resistance, appears to be the mechanism for this phenomenon.[16] No evidence of intraventricular hemorrhage was found at autopsy, contrary to the theory proposed by some authors that the acute increase in intracranial pressure must be accompanied by intraventricular hemorrhage before nonfilling of the internal carotid circulation occurs.[17]

Extravasation from torn meningeal arteries is usually seen in proximity to a fracture.[15] This patient's fracture probably extended around the skull

posteriorly to involve the left as well as the right side of the calvarium; the portion of the fracture actually crossing the torn vessel is probably radiographically invisible.[13]

Although computerized tomography has made the diagnosis of intracranial epidural hematoma an easy one in most cases, when a fistula is encountered the vascular anatomy can only be precisely defined with cerebral angiography.

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ACUTE LOW-DOSE HYDRALAZINE INDUCED LUPUS SYNDROME (HILS)

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A case of acute hydralazine induced lupus syndrome is described. No case has previously been described in which a small daily dose and a small accumulation dose have evoked this syndrome in such a short period of time. A pertinent and selective review of this syndrome is presented. The need for awareness of this syndrome manifesting in a patient exposed to small doses of hydralazine for short periods of time is emphasized.

It has long been recognized that hydralazine can cause an illness resembling systemic lupus erythematosus (SLE).[1-5] In general, it has been accepted that the hydralazine induced lupus syndrome (HILS) occurs in 6-10 percent of patients taking hydralazine, 400 mg or more daily, for more than six months.[6] To date no known case has been reported to occur after 50 mg/day of hydralazine or less than 1 gm total dose of hydralazine.[7] The case reported herein is unique in these respects.

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