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MR Recognition of Internal Carotid Artery Occlusion

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Imaging of vascular structures and blood flow with magnetic resonance (MR) has added a new dimension in evaluating patients with suspected intracranial circulatory disturbances [1]. By using appropriate MR pulse sequences, the capability now exists of diagnosing intracranial circulatory abnormalities without the injection of radiographic contrast agents. We report a case of internal carotid artery occlusion initially diagnosed by MR.

Case Report

A 41-year-old man underwent neurologic evaluation after 3 years of severe, intermittent, bifrontal headaches that at times were associated with paresthesias of the right side of his body. About 2 months before admission, the patient experienced an episode of sudden pain that radiated down the left side of his mandible and was accompanied by severe headache, nausea, vomiting, and scotomata. The patient's condition improved over a period of time except for persistence of his headaches. Eventually, the patient began experiencing difficulty in swallowing and increasing hoarseness. Neurologic evaluation revealed paralysis of the left vocal cord and left palate. Steroids were administered, alleviating his hoarseness and swallowing difficulties; however, his headaches persisted without improvement.

Radiological Studies

An initial MR study elsewhere was submitted for our evaluation. Spin-echo (SE) images in transverse and coronal planes demonstrated increased signal intensity in the cavernous portion of the left internal carotid artery in contrast to absence of signal from the cavernous portion of the right internal carotid artery.

MR was performed at our institution with a 0.6 T superconducting unit (Technicare, Solon, OH) using a 128 × 256 matrix and 1 cm slice thickness. SE sequences with repetition times (TRs) and echo times (TEs) of 500/30 msec and 2120/60, 120 msec in three orthogonal planes demonstrated a discrepancy in signal intensity in the cavernous portions of the internal carotid arteries (figs. 1A–1E). The left intracavernous internal carotid artery demonstrated very intense signal, while the right intracavernous internal carotid artery registered no discernible signal. We concluded that the patient had significant obstruction or complete occlusion of the left internal carotid artery. An angiogram was obtained for definitive diagnosis (fig. 1F).

The left common carotid arteriogram demonstrated total occlusion of the left internal carotid artery about 2 cm from its origin. Intraluminal filling defects were present in the proximal aspect of the left internal carotid artery and in the proximal external carotid artery, suggesting thrombus formation. A right common carotid arteriogram revealed a normal common carotid bifurcation and a normal right internal carotid artery. Cross filling of the left A1 segment and left middle cerebral artery group via the anterior communicating artery was demonstrated on the frontal view of the right common carotid injection.

Discussion

Blood flow imaging using MR has been reviewed in recent studies delineating the basic principles of flow phenomena and their effect on the observable MR image [1–4].

By using SE imaging techniques, variation in signal intensity has been demonstrated for stationary and flowing blood. Mills et al. [1] have shown that a spectrum exists in which signal intensity within vascular structures is related to the velocity of blood flow. Rapidly flowing blood has been shown to emit no detectable signal with MR imaging. This is due to the presence of rapidly moving hydrogen nuclei that escape the imaged volume during the interval between the administered radiofrequency (RF) pulse and the emitted RF signal. As a result, no signal is emitted from within the normal vessel. Slow-flowing blood exhibits what has been termed *paradoxical enhancement*, which can be due to unsaturated protons entering a section or to even-echo rephasing, whereby signal intensity increases with the second echo as a result of the rephasing phenomenon that occurs after each 360° rotation following the initial 90° pulse for all even echoes in a given echo train [5]. This results in a signal intensity greater than that observed with high-velocity blood flow or stationary blood. Stationary blood, on the other hand, gives rise to a signal intensity greater than rapidly flowing blood, but less than slow-flowing blood [1].

Even-echo rephasing and diastolic pseudogating do not play a role in our reported case. If these phenomena were involved, one would expect them to affect both carotid arteries in a similar fashion.

As a result of the signal intensity variability of different blood flow states, one has the potential to predict relative patency of intracranial vascular structures [4]. MR can increase our

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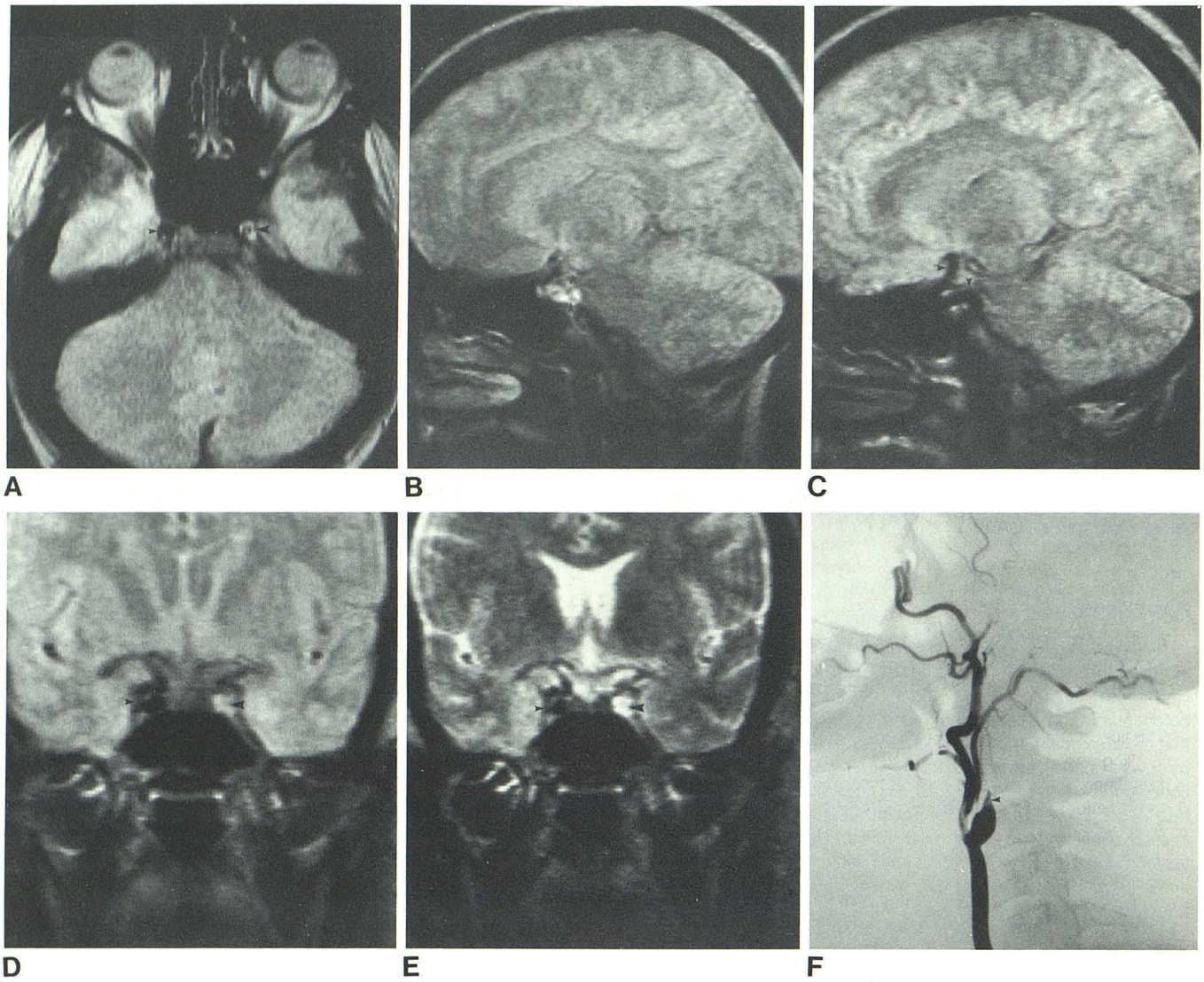

Fig. 1.—**A**, Transaxial SE image, 2120 msec TR, 60 msec TE. Thrombus formation in left intracavernous carotid artery is represented by area of increased signal (*large arrowhead*) in contrast to low signal in patent right intracavernous carotid artery (*small arrowhead*). **B**, Sagittal SE image, 2120 msec TR, 60 msec TE. Area of increased signal (*arrowheads*) in region of left cavernous sinus represents thrombus formation in intracavernous left internal carotid artery. Small low-signal areas in high-signal region within cavernous sinus may represent areas of recanalization within thrombus (cf. **A**). C, Sagittal SE image, 2120 msec TR, 60 msec TE. In contrast, intracavernous and supraclinoid portions of right internal carotid artery (*arrowheads*) are well demonstrated with low signal in this patent vessel due to flow-void phenomenon. **D**, Coronal SE image, 2120 msec TR, 60 msec TE. Thrombus formation in intracavernous left internal carotid artery is again represented by area of increased signal (*large arrowhead*) in contrast to low signal in patent right intracavernous carotid (*small arrowhead*). Good visualization of supraclinoid portions of internal carotid arteries bilaterally due to cross filling via anterior communicating artery. **E**, Coronal SE image, 2120 msec TR, 120 msec TE. Signal intensity from thrombus in intracavernous left internal carotid artery (*large arrowhead*) has increased due to its long T2 characteristics. Again note patency of right intracavernous carotid artery in comparison (*small arrowhead*). **F**, Lateral view of left common carotid angiogram. Complete occlusion in proximal left internal carotid artery (*arrowhead*) at level of C2–C3 about 2 cm distal to its origin. Intraluminal thrombus in origin of external carotid artery.

ability to diagnose patients with significant vascular disease that may not be diagnosed with CT. This case of internal carotid artery occlusion diagnosed by MR demonstrates the value of MR in the evaluation of intracranial abnormalities that may have a vascular etiology.

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