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Spontaneous Thrombosis of a Direct Carotid Cavernous Sinus Fistula: Confirmation by Gd-DTPA-Enhanced MR

Case Report

A 38-year-old man was admitted for possible embolization of a left carotid cavernous sinus fistula. Two months previously, the patient had been admitted to the hospital after a motorcycle accident. Twenty-four hours later, he was noted to have left proptosis, chemosis, and a third-nerve palsy. An angiogram was performed and showed a carotid cavernous sinus fistula (Fig. 1A). The patient was referred to our institution for embolization. Physical examination revealed marked left proptosis and chemosis, but no orbital bruit was present. A repeat angiogram failed to show the fistula (Fig. 1B). Thrombosis of the left cavernous sinus was suspected, and two sets of MR images were obtained, one set before and one set after gadolinium-DTPA injection (Figs. 1C and Fig. D).

Discussion

Spontaneous thrombosis of a carotid cavernous sinus fistula (CCF) is a rare event mainly seen in the indirect (dural) type of fistula [1, 2]. Seeger et al. [1] reported nine cases of CCF that spontaneously thrombosed. Only two of these fistulae were the direct (high-flow) type. In one of these two patients, the CCF did not thrombose completely and surgical treatment was necessary. In their second patient with a direct CCF, an abnormal venous blood-flow pattern was noted during angiography and was presumed to represent cavernous sinus thrombosis. However, this patient refused angiographic follow-up, and it remains unknown whether the findings actually were related to cavernous sinus thrombosis.

Mechanisms thought to be responsible for the spontaneous thrombosis of CCF include slow flow leading to venostasis and damage to the vascular lining of the cavernous sinus caused by venous hypertension [3, 4]. Another possible explanation is that the injection of iodinated contrast media exaggerates leukocytic accumulation, promotes RBC aggregation, and has a direct effect on the vascular endothelium, all of which can lead to thrombosis [5-7].

Our patient underwent angiographic evaluations 2 months apart; between the two angiographic studies, complete thrombosis of a direct CCF had occurred, a segment of narrowing had developed, and two pseudoaneurysms of the left internal carotid artery had formed (Figs. 1A and 1B). Although we cannot definitely explain why the CCF thrombosed, we hypothesize that the blood flow through the carotid artery became slow because of the presence of the previously described narrowed segment (which was most likely due to an undetected posttraumatic dissection) and the development of the two pseudoaneurysms, and that this decreased blood flow caused thrombosis of the fistula. Since the most superior pseudoaneurysm occurred at the exact fistulous tract, it may have compressed the fistula mechanically, leading to its thrombosis. Another plausible explanation could be related to the accentuation of pre-existing partial thrombosis caused by the injection of contrast material during the initial angiogram.

In order to confirm the diagnosis of a thrombosed cavernous sinus, a gadolinium-enhanced MR study was obtained. In a previously reported case [8] of cavernous sinus thrombosis, T1-weighted MR images showed increased signal intensity within the involved sinus. In our patient, the T1-weighted images obtained before gadolinium injection showed questionable enlargement of the left cavernous sinus and a small area of increased signal intensity within it (Fig. 1C). Because we were uncertain whether this small abnormal area could account for the complete obliteration of the CCF in our patient, a gadolinium-enhanced MR was obtained, and the T1-weighted images obtained immediately after gadolinium injection showed marked increased signal intensity in both cavernous sinuses. Enlargement of the left cavernous sinus was definitely present, and an area of low signal intensity was identified within it and thought to represent thrombus (Fig. 1D).

We recommend MR as the noninvasive method of choice for the evaluation of clinically suspected cavernous sinus thrombosis regardless of whether it is associated with CCF. However, as in our patient, if the thrombosis is relatively acute and sufficient methemoglobin is

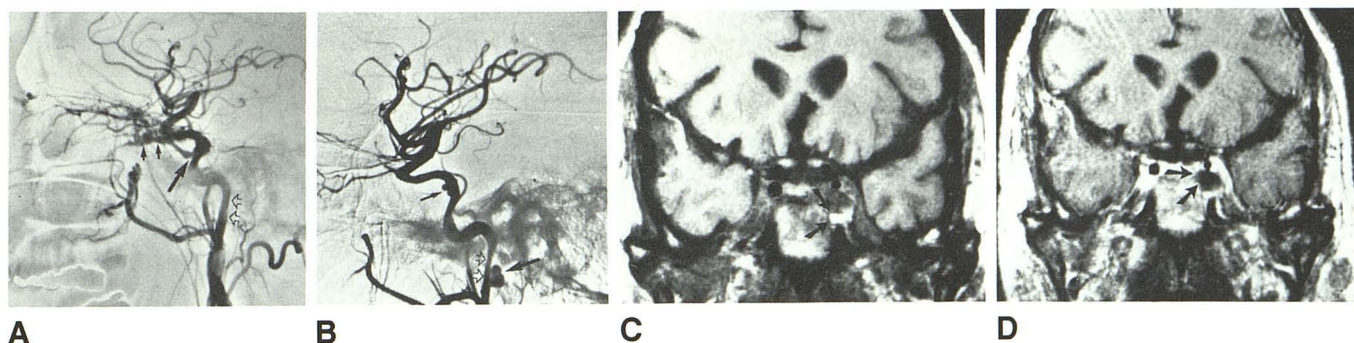


Fig. 1.—A, Initial angiogram, lateral view. Internal carotid artery communicates with cavernous sinus (large arrow). Small black arrows indicate left cavernous sinus. There is questionable narrowing of internal carotid at base of skull (open arrows).

B, 2 months after A, previously documented carotid cavernous fistula is not seen. At base of skull, there is narrowing of common carotid (open arrows) and a bilobed pseudoaneurysm (large arrow). A smaller bilobed pseudoaneurysm (small black arrow) is present at site of previous carotid cavernous sinus fistula.

C, MR image (600/20) through region of interest shows questionable enlargement of left cavernous sinus with a small abnormal area (arrows) of high signal intensity within it. This could represent methemoglobin caused by thrombosis.

D, MR image (600/20) immediately after gadolinium administration through same level as C. Left cavernous sinus is enlarged and contains an area of low signal intensity (arrows) thought to represent clot without methemoglobin. Notice that the previously seen area of high signal intensity is now indistinguishable from surrounding enhanced cavernous sinus. Compare with right cavernous sinus.

not present, the clot may not be visible and enhancement with gadolinium is helpful.

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