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Vascular

INTRACAVERNOUS CAROTID ARTERY ANEURYSMS:

THE POSSIBLE IMPORTANCE OF ANGIOGRAPHIC DURAL WAISTING. A CASE REPORT

Michael Horowitz, M.D.,*,** Frank Fichtel, M.D.,* Duke Samson, M.D.,* and Phillip Purdy, M.D.*,**,***

Department of Neurological Surgery, Division of Neuroradiology, Department of Radiology,** Department of Neurology,*** UT Southwestern Medical Center at Dallas, Dallas, Texas*

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BACKGROUND
Cavernous carotid aneurysms are generally benign entities. Certain indications exist for their treatment, however, including transient ischemic events, subarachnoid hemorrhage or risk of subarachnoid hemorrhage, epistaxis or its risk, ophthalmoplegia, pain, and progressive visual loss. We feel certain angiographic features may indicate a greater likelihood that cavernous carotid aneurysms extend into the subarachnoid space, thus making their rupture a life-threatening event.

METHODS
A case report of an intracavernous carotid aneurysm, which at surgery extended into the subarachnoid space, is described.

RESULTS
In this particular case, deformation of the aneurysm (waisting) as seen at angiography was in retrospect an indication that the cavernous carotid aneurysm extended into the subarachnoid space, either through the dural ring or through the eroded dural roof of the cavernous sinus. This finding was verified at surgery when the lesion was explored and trapped.

CONCLUSION
Angiographic waisting of a cavernous carotid aneurysm may indicate that the aneurysm extends into the subarachnoid space. Such extension means that rupture would be a life-threatening event. While deformation of the aneurysm may be secondary to compression against the optic nerve or anterior clinoid process with an intact layer of dura overlying the aneurysm, the neurosurgeon confronted with such findings should analyze such lesions carefully and consider surgical exploration.

KEY WORDS
Aneurysm, cavernous carotid, angiography.

Intracavernous carotid artery aneurysms (ICCAA) make up 5% of intracranial aneurysms. Extension of such lesions into the subarachnoid space is a rare but important finding because it influences the surgeon to proceed with prophylactic obliteration to avoid the complications attendant on aneurysm rupture. Despite the advent of new imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI), delineation of the dural margin and its relationship to the aneurysm wall can be difficult. The angiographic appearance of a dural impression on the aneurysm dome can help distinguish between entirely intracavernous and combined intracavernous and extracavernous lesions.

CASE REPORT

A 67-year-old right-handed woman was seen 4 weeks prior to admission to our institution with headache, nausea, vomiting, and diarrhea. Over the next 4 weeks her symptoms resolved, except for the headache, which she described as right retro-orbital and frontal. Worsening cephalalgia prompted a noncontrast cerebral MRI evaluation, which revealed a 3-cm diameter right cavernous carotid aneurysm (Figure 1 **A**, **B**). No evidence of subarachnoid hemorrhage was seen. A cerebral angiogram confirmed the MRI diagnosis, and the patient was transferred to our facility for definitive treatment (Figure 1 **C**, **D**).

Address reprint requests to: Michael Horowitz, M.D., Division of Neuroradiology, Department of Radiology, UT Southwestern Medical School, 5323 Harry Hines Blvd., Dallas, TX 75235-8896.
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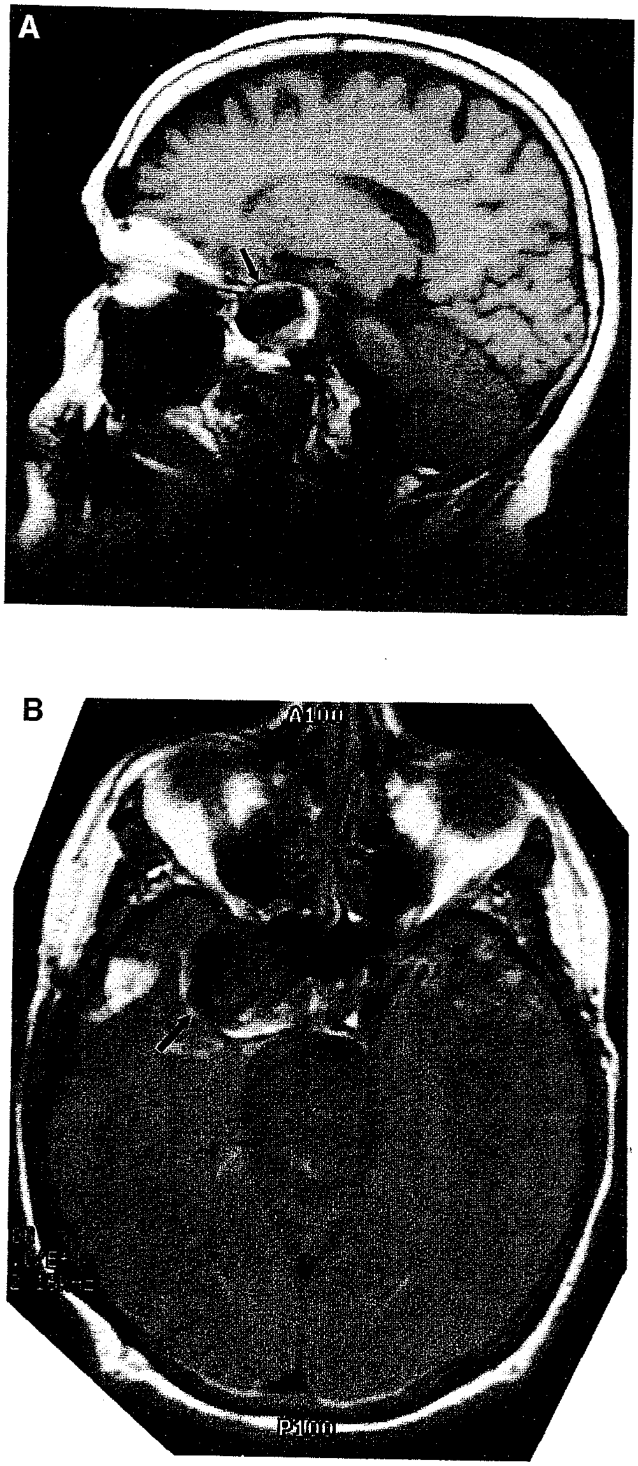

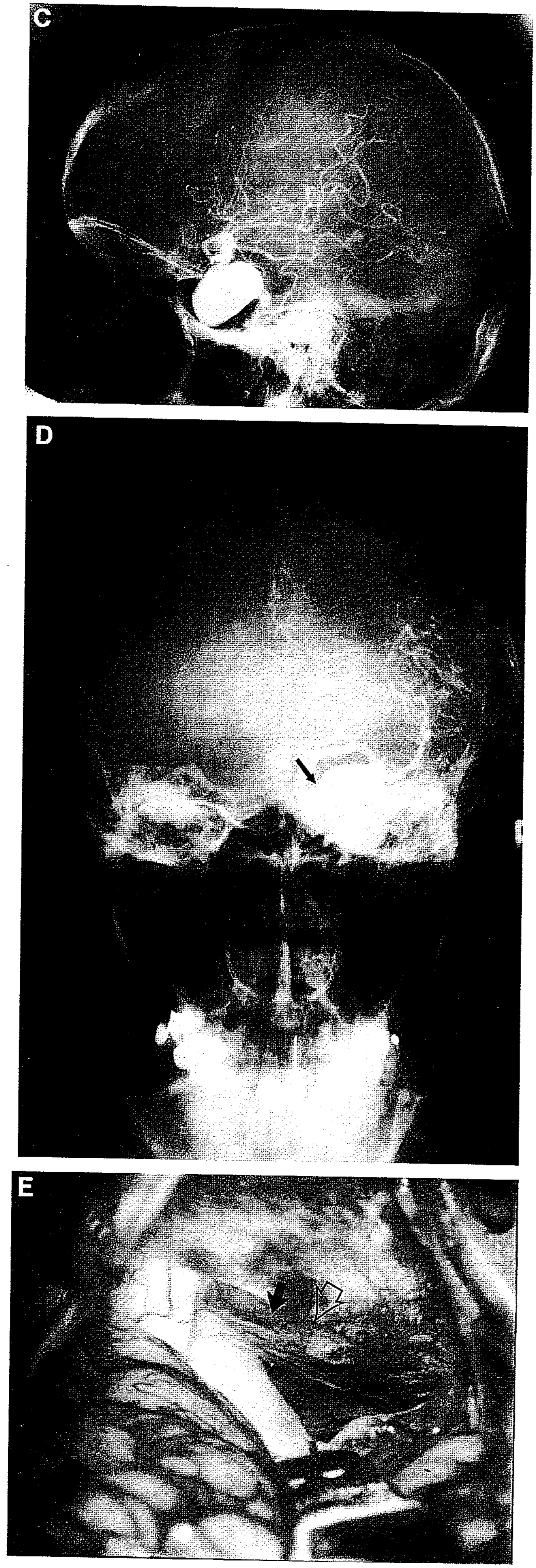

1 (**A, B**) Sagittal (**A**) and axial (**B**) T_1-weighted MRIs demonstrating the internal carotid artery aneurysm located within the cavernous sinus (*arrow*). (**C**) Lateral internal carotid artery cerebral arteriogram demonstrating the cavernous carotid artery aneurysm. (**D**) AP internal carotid artery cerebral arteriogram demonstrating the cavernous carotid artery aneurysm. Note the dural waist along the medial aspect of the aneurysm dome (*arrow*). (**E**) Intraoperative photograph demonstrating the interface (*open arrow*) between the cavernous carotid dura and the superior dome of the aneurysm (*solid black arrow*). The interface corresponds to the waist seen on the AP arteriogram. The optic nerve can be seen to the left of the retractor between the frontal lobe and the carotid artery.

Upon admission, the patient's neurologic examination was normal, except for a mild right sixth nerve paresis. Her history revealed intermittent diplopia over the previous weeks. A selective right internal carotid artery arteriogram confirmed the presence of a giant right ICCAA. The dorsal anteromedial aspect of the lesion was slightly indented with what appeared to be a waist along the aneurysm dome. A Medetech (Boston Scientific Corp., Watertown, MA) 8-mm balloon with a 0.026-inch lumen was then placed over a 0.025-mm Medetech Glide exchange wire into the internal carotid artery (ICA). The balloon was inflated for 30 minutes, and the ICA was occluded approximately 4 cm distal to the left common carotid artery bifurcation. Serial neurologic examinations during this time period remained normal. The balloon was deflated and removed at the end of the trial.

Having passed a trial of ICA occlusion, the patient was brought directly to the operating room where she underwent a right cervical carotid exposure and pterional craniotomy. To eliminate the risk of retrograde aneurysm filling and antegrade embolization from the partially thrombosed aneurysm, the plan was to trap the aneurysm with proximal ICA ligation and distal clip ligation superior to the aneurysm and proximal to the ophthalmic artery. After dissecting the supraclinoid carotid artery and drilling the roof of the optic canal, the proximal supracavernous carotid artery was exposed. At this point, it was noted that vascular tissue seemed to extend superior and medial to the cavernous sinus roof and through the dura. With further dissection, it became clear that this tissue represented the cavernous carotid aneurysm that had extended out of the cavernous sinus and into the subarachnoid space (Figure 1 **E**). The internal carotid artery was ligated in the neck and clipped proximal to the ophthalmic artery, thus completing the trapping procedure.

The patient's postoperative course was complicated by transient loss of light perception in her right eye. Vision returned to normal in less than 18 hours. The etiology of her deficit was never elucidated, although compression from postthrombotic expansion of the aneurysm or transient ischemia are two considerations. A CT performed during the period of visual loss revealed increased density within the cavernous sinus, which was consistent with thrombus within the aneurysm sac.

Discussion

Three to 5% of intracranial aneurysms arise from the intracavernous carotid artery, and 14% of internal carotid artery aneurysms are located within the cavernous sinus [4–6,8,11]. ICCAAs have been described to arise from four segments of the cavernous carotid artery named in caudal to rostral orientation the posterior genu, horizontal segment, anterior genu, and ophthalmic segment [6]. The majority of such aneurysms remain asymptomatic. Enlargement, however, may lead to bony erosion and compressive cranial nerve neuropathy. Rupture of an ICCAA most commonly presents with the development of a carotid cavernous fistula and its attendant signs and symptoms, including epistaxis, retro-orbital pain, proptosis, chemosis, cranial nerve neuropathy, cephalic bruit, and pulsatile exophthalmos. ICCAA presenting with subarachnoid hemorrhage (SAH) has been reported in as many as 9% of patients in some series [1,10]. SAH is most likely to occur when an ICCAA either arises at the anterior genu and extends through the cavernous sinus dura, or when the ICCAA erodes into the pituitary sella [6].

Indications for the treatment of ICCAAs include the following: transient ischemic events secondary to embolization of sac thrombus, SAH or risk of SAH, epistaxis or its risk, severe ipsilateral facial or orbital pain, progressive ophthalmoplegia, and progressive visual loss [7]. Many of these indications are easily elucidated by history and physical examination. Evaluation of the risk for SAH can often be difficult if the aneurysm abuts the dorsal cavernous sinus dura, as it can be hard to discern on MRI studies and angiographic images whether the lesion extends above the dural margin and into the subarachnoid space or simply bows the dura in a dorsal direction. Lombardi's radiographic series of 24 cases mentioned the finding of a fusiform dilation when ICCAA involved both the infraclinoid and supraclinoid carotid artery [9]. The case described here points to the importance of the presence of an indentation or waist along the aneurysm dome on angiographic images as evidence that the lesion may protrude through the dura and into the subarachnoid space. The waist itself is created by dural indentation of the aneurysm fundus as it protrudes into the subarachnoid space. When faced with this type of angiographic image, the neurosurgeon should consider prophylactic obliteration of the lesion, whether by an open surgical or endovascular route, because such an image may indicate a partially subarachnoid location.

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COMMENTARY

The authors have herein described a case of a 67-year-old woman with an intracavernous aneurysm that was found at surgery to have extended into the subarachnoid space. Retrospectively, the arteriogram showed a slight indentation or "waisting" of the aneurysm fundus along its anteromedial border at the point where it had eroded through the cavernous sinus dura into the subarachnoid space.

The figures are of high quality and show excellent correlation between angiographic and operative findings. Recognition of the indentation of an aneurysm dome can be an important clue to proximity of the aneurysm to surrounding structures (i.e., an ophthalmic segment aneurysm against the optic nerve). In this case, the indentation correctly predicted extension of the aneurysm into the subarachnoid space. While SAH from intracavernous aneurysm is rare, it carries the same dire consequences as hemorrhage of purely subarachnoid aneurysms, and this factor should be carefully considered in treatment planning.

Arthur L. Day, M.D.
Joel D. MacDonald, M.D.
Department of Neurological Surgery
University of Florida
Gainesville, Florida

This is an interesting documentation of a giant intracavernous ICA aneurysm extending into the subarachnoid space, with probable correspondence to the angiographic finding of deformation. We had a case of a giant intracavernous ICA aneurysm, which presented with subdural hematoma. At surgery, the giant aneurysm was found to be exposed without dural covering in the anterior temporal fossa. Although the aneurysm was somewhat irregular in shape on the angiogram, we did not correlate the deformity with the operative findings, as in the present case.

The reason for the dural waist formation in the present case would be that the aneurysm was denuded anteriorly near the site where the carotid artery penetrated the dura. In this region, the dura is not of even thickness, having such structures as fibrous (distal) dural ring and falciform process (dural fold over the optic nerve); these anatomic structures, together with nearby bone structures such as the anterior clinoid process, optic canal, and optic strut eroded variously by the giant aneurysm, may have caused the angiographic deformity. Intraoperative angiography would have proved the correspondence between the waist on the angiogram and the site of dural erosion.

Shigeaki Kobayashi, M.D.
Department of Neurosurgery
Shinshu University School of Medicine
Matsumoto, Japan