CASE REPORTS

CAROTID Cavernous Fistula with Signs on Contralateral Side

CASE REPORT

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(Received for publication July 21, 1952)

The fact that an arteriovenous fistula between the internal carotid artery and the
cavernous sinus, because of anatomical variations, is apt to present clinical findings
on the side opposite to that of the pathological lesion, has been previously reported
by Doctors Dandy and Walsh. However, their cases were not verified by arteriogra-
phy and, to the best of our knowledge, there is only one other case (personal com-
munication of Sir Geoffrey Jefferson from England) in which careful and well done
arteriographic studies demonstrated evidence of an internal carotid-cavernous sinus
fistula on the side opposite to that on which the clinical signs were present.

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No64436. S.E., a 54-year-old right-handed white woman, was admitted to the Neuro-
surgical Service of the Neurological Institute on Oct. 21, 1951. Her chief complaint was a
pounding noise inside her head, related mostly to the left ear, since May 31, 1950.

On May 30, 1950, approximately at midnight, and for an unknown reason, she had
fallen while going up the front stairs of her house. She was unconscious for 2–5 minutes
and there was no apparent injury resulting from the fall. The patient went to sleep and
the next day as usual she was at work. During the whole day she complained of headache and
pain over her eyes, and the left eye began to feel “swollen.” She returned home and then
about 10:00 p.m. on May 31, 1950 she noticed the sudden onset of a pounding noise “like
hammering” inside her head, mostly localized to her left ear. This was a continuous noise,
with exacerbations synchronous with her pulse. Immediately afterward her left eye became
“harder” and completely closed, remaining that way for about 5 days. At the same time there
was marked ecchymosis around the left eye, which progressively subsided in a few days. The
left-sided ptosis gradually improved during the next 4 months. When her left eye began to
open she noticed “double vision.” This disappeared within a period of 2 months. Since May
31, 1950 her left eye had appeared to be “blood shot,” mostly in the outer portion of the eye-
ball. There had been progressive protrusion of the left eye since the onset of her illness.

Examination. The left upper eyelid was full with congested veins. The conjunctiva showed
generalized chemosis, greater in the left lower quadrant. There was a left, nonpulsating exoph-
thalmos of 2 mm. Auscultation over the closed left eye revealed a loud bruit synchronous
with her pulse. The bruit was not audible over the right eye. The bruit was not eliminated
by left, right or bilateral carotid compression. Visual acuity: O.D. 20/20; O.S. 20/70 (not
corrected). Visual fields were normal. Ophthalmoscopic examination showed engorgement of
the veins of the left fundus. There was no papilledema. The III, IV and V cranial nerves
were normal. There was no diplopia, but there was weakness of the left external rectus muscle.
The rest of the cranial nerves were normal.

Laboratory Studies. Complete blood count, urinalysis, sedimentation rate, and Mazzini

* Presented at the meeting of the New York Neurosurgical Society, New York, November 20, 1951.
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test were negative. The CSF pressure, cytology and chemistry were normal. Calories and audiometrics were essentially normal. Skull x-rays, including special projections, were reported as negative.

**Impression.** Considering the history and physical findings, the diagnosis of arteriovenous fistula of the left internal carotid artery and the left cavernous sinus was made by all observers.

**Arteriography.** On Oct. 23, 1951, under local anesthesia, left common carotid percutaneous arteriography was performed. This failed to show any abnormality. The right common carotid artery was then entered and films revealed a large arteriovenous fistula in the right cavernous sinus. This drained through the anterior portion of the circular sinus into the opposite cavernous sinus and left superior ophthalmic vein (Figs. 1 and 2). Since the bruit was not obliterated by bilateral common carotid compression, percutaneous right vertebral arteriography was done. This showed a tremendous right posterior communicating artery supplying part of the carotid circulation, and explained why the bruit could not be eliminated by bilateral carotid compression (Fig. 3).