Iatrogenic Carotid Cavernous Sinus Syndrome

MICHAEL G. HUMMER, M.D., AND THOMAS J. CARLOW, M.D.

SUMMARY A hemodialysis shunt site, subclavian artery to internal jugular vein, resulted in a "pseudo" cavernous sinus syndrome. Recognition of this rare iatrogenic complication may assist in selecting other shunt sites and prevent potential visual loss and multiple surgical procedures.

Stroke, Vol 12, No 5, 1981

THE FULLY DEVELOPED clinical picture of a carotid cavernous sinus fistula (CCF) is not a diagnostic dilemma. These vascular malformations commonly are either traumatic or spontaneous. This report is of a iatrogenic instance with clinical signs and symptoms of a carotid cavernous sinus syndrome following a subclavian artery to internal jugular vein shunt.

History

A 62-year-old right-handed man had received 6 years of hemodialysis treatment for membranous glomerulonephritis. Multiple episodes of thrombophlebitis eventually consumed all common sites for peripheral hemodialysis fistulas. Renal transplantation was unsuccessful. Since all peripheral shunt sites had failed, a left subclavian artery to left internal jugular vein shunt was selected. Within three weeks after the anastomosis, he experienced holocarpal headaches and mild generalized weakness, worse on his right side. A slow but progressive reddening of his left eye was noted. He denied diplopia, subjective bruit, pain and decreased visual acuity.

Neurological examination documented a mild but distinct pronation drift of the right arm without tendon reflex asymmetry. Ophthalmologic examination revealed a marked dilation and arterialization of the left conjunctival vessels and 2 mm of left proptosis (fig. 1). An ocular bruit was not heard and both globes were neither tender nor pulsating (utilizing a Schiötz tonometer). Visual acuity was correctable to 20/25 on the right and 20/20 on the left. Pupils were 4 mm bilaterally and equally responsive to light and accommodation. Applanation tonometry readings were 12 mm Hg on the right and 10 mm Hg on the left. Vergence, version andduction extraocular movements were normal. Fundus examination showed mild dilation of the left retinal veins.

Abnormal laboratory studies included: BUN 64 mg/dl, creatinine 8.5 mg/dl, total protein 5.4 gm/dl, albumin 2.9 gm/dl, prothrombin time 25.7 seconds (control 11.8) and partial thromboplastin time 67.3 seconds (normal less than 40 seconds). Urinalysis revealed a specific gravity of 1.017, 2+ glucose, 3+ protein, 2-6 wbc/hpf, and 1-3 casts/lpf.

An EEG showed slowing and decreased amplitude over the left frontal area. The CT scan suggested a left frontal chronic subdural hematoma. Within a week after the subdural was evacuated the right sided weakness and the EEG improved markedly.

A left carotid arteriogram, after removal of the subdural hematoma, showed slow arterial filling of the left cerebral hemisphere with all venous drainage from that side via the right transverse sinus and right internal jugular vein. A left subclavian arteriogram documented left cavernous sinus arterialization from retrograde flow in the left internal jugular vein, transverse sinus and petrosal sinus. Blood flow was from the shunt into the left carotid cavernous sinus (fig. 2). Since venous drainage from the left cerebral hemisphere occurred through the right internal jugular vein, the left internal jugular vein was ligated above the fistula. Two months later the patient was symptom
free and the injection in his left eye, proptosis and retinal signs had resolved. He was maintained on home peritoneal dialysis.

Discussion

This patient's constellation of symptoms and signs are similar to those of the classical carotid cavernous sinus fistula syndrome. Since the vascular shunt was not in the cavernous sinus but at a more distal site, the term "pseudo" carotid cavernous sinus syndrome seems appropriate.

Cavernous sinus arteriovenous communications include direct carotid cavernous fistulas (CCF) and dural meningeal shunts. A CCF was first described by Benjamin Tavers in 1811. He diagnosed a pulsating exophthalmos as an arteriovenous fistula and treated the CCF by ligating the common carotid artery.

CCF's can be divided into 3 types: spontaneous (25%), traumatic (75%) and iatrogenic. The fully developed CCF syndrome includes: ocular bruit (75%), pulsating exophthalmos (69%), diplopia (24%), conjunctival chemosis (36%), orbital pain (16%), and, less frequently, dilated and arterialized conjunctival vessels, decreased visual acuity, headache and increased intraocular pressure. Angiographic delineation with magnification and subtraction techniques can differentiate and delineate the involved vessels.

Iatrogenic causes have been described following percutaneous retrogasserian procedures, secondary to transsphenoidal surgery or Fogarty catheter carotid thromboendarterectomy. The iatrogenic CCF documented here has not been previously reported.

The pathogenesis and treatment of CCF are beyond the scope of this report but several excellent articles and reviews are available. A fistula may disappear spontaneously in 5 to 10 percent of patients. Balloon-catheter techniques and thrombotic methods that preserve the carotid circulation appear to be highly satisfactory alternatives to entrapment procedures. The primary surgical objective is to prevent hypoxic ocular sequelae with attendant visual loss.

Recognition of the potential complication of a subclavian artery to internal jugular vein shunt may help to eliminate this locale as a possible hemodialysis shunt site. Prompt surgical correction can prevent visual loss.

References

2. Tavers BA: A case of aneurysm by anastomosis in the orbit, cured by ligation of the common carotid artery. Trans Med Clin 2: 1-16, 1811
6. Takahashi M, Killeffer F, Wilson G: Iatrogenic carotid cavern-
by guest on October 5, 2016 http://stroke.ahajournals.org/ Downloaded from

the right ocular fundus (fig.) showed an anomalous permanent retinal or cerebral attacks. Examination of quite certain the field loss was as reported.

described 2 spells per day for 3 consecutive days of been careful to cover each eye separately and was Hospitalization was advised but he put this off for 11 months. During this time there were no transient or loss of vision in the temporal half visual field of the right eye and was subsequently found to have right internal carotid artery occlusion. Fundoscopy revealed an anomalous central retinal artery branching whereby a single stem vessel supplied the superior and inferior nasal quadrants of the retina. Circulatory insufficiency in this anomalous stem could explain the occurrence of vertical monocular hemianopsia as an unusual manifestation of ipsilateral carotid artery arteriosclerosis.

SUMMARY A 62-year-old man reported 6 stereotyped attacks of transient loss of vision in the lateral visual field of the right eye and was subsequently found to have right internal carotid artery occlusion. Fundoscopy revealed an anomalous central retinal artery branching whereby a single stem vessel supplied the superior and inferior nasal quadrants of the retina. Circulatory insufficiency in this anomalous stem could explain the occurrence of vertical monocular hemianopsia as an unusual manifestation of ipsilateral carotid artery arteriosclerosis.

TRANSIENT monocular blindness as an indicator of ipsilateral internal carotid artery atheroma was first emphasized by Fisher.1 When the transient blindness is not total, a horizontal hemianopsia or a quadrantanopsia is most often reported by the patient.2 This is consistent with the usual pattern of arterial supply to the retina whereby a single superior and inferior branch of the central retinal artery supplies a territory above and below the optic disc.

This patient presents an instance of vertical monocular hemianopsia that may have been due to an appropriate retinal vascular anomaly. In such a patient, description of a vertical monocular hemianopsia might be interpreted by the physician to represent a misreporting of a homonymous hemianopsia.

Report of the Patient

With no previous history of retinal or cerebral vascular disease, a 61-year-old hypertensive man described 2 spells per day for 3 consecutive days of loss of vision in the temporal half visual field of the right eye lasting about one minute each time. He had been careful to cover each eye separately and was quite certain the field loss was as reported. Hospitalization was advised but he put this off for 11 months. During this time there were no transient or permanent retinal or cerebral attacks. Examination of the right ocular fundus (fig.) showed an anomalous retinal arteriolar pattern whereby a single common stem bifurcated (arrow) to supply the superior and inferior nasal quadrants. The temporal quadrants were supplied in the usual fashion and the vascular pattern of the left ocular fundus was normal. No embolic material was noted. Transfemoral angiography showed total occlusion of the right internal carotid artery just distal to its origin. Blunting of the stump suggested an old occlusion.

Discussion

Transient monocular vertical hemianopsia has been described only rarely,3 and an adequate explanation for it has not been provided. Permanent monocular vertical hemianopsia has been reported as an uncommon result of emboli to the superior and inferior retinal artery branches on the same side of the retina.4 The basic anatomical pattern of division of the central retinal artery into a superior and an inferior retinal artery is only rarely anomalous and global, altitudinal or quadratic field loss is to be expected. There is variation as to where, in relation to the optic disc, the division into a superior and an inferior retinal artery branch occurs.5 Single instances have been reported in asymptomatic individuals where one central retinal artery branch divides to supply the superior and the inferior nasal or temporal half of the retina.6 8 The only large-scale search for such anatomical variations was undertaken by Awan6 who found, on examining the eyes of 2100 people, that the temporal half-retina was supplied by a single branching artery in 19 eyes and the nasal half-retina
Iatrogenic carotid cavernous sinus syndrome.
M G Hummer and T J Carlow

doi: 10.1161/01.STR.12.5.689

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/12/5/689.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in
Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office.
Once the online version of the published article for which permission is being requested is located, click Request
Permissions in the middle column of the Web page under Services. Further information about this process is
available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/