Retinal Artery Embolism: A Complication of Carotid Endarterectomy*

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Since Eastcott et al.,5 in 1954, first successfully relieved a segmental obstruction of the carotid artery, many reports have appeared documenting the beneficial effects of surgery on extracranial occlusive disease of the carotid and vertebral arteries.1,2,4,6,14,18 Complications also should be documented. This case report describes a patient in whom monocural blindness developed following carotid endarterectomy.

Case Report
PBBH 9H167. G.R., a 59-year-old, white, right-handed baker, was admitted Jan. 26, 1961 because of confusion and right-sided weakness.

Eight days prior to admission, while at work, the patient had the sudden onset of confusion. He became unsteady on his feet, began to drop objects, and was noted to be disoriented. He was sent home from work because he was having trouble expressing himself. He had no headache, vertigo or syncope. His condition did not improve and his family brought him to the hospital.

Past history revealed that an unsuccessful operation was performed in 1917 to correct a bilateral exotropia. His only other hospitalization was in 1937 for superficial thrombophlebitis. Since 1953, the patient has had psoriasis.

Examination revealed an afebrile, moderately obese, pellorhagic white male. Pertinent physical findings were: blood pressure, 140/80 in both arms; diffuse psoriasis; no audible bruits over the peripheral vessels; pulses present bilaterally at the subclavian, carotid, superficial temporal, brachial, femoral, dorsalis pedis and posterior tibial arteries.

The patient was disoriented to time and person. He was unable to perform serial sevens or any simpler arithmetic. He had apraxia, right-left and finger agnosia, and perseveration. He had an incongruous right homonymous hemianopia. The fundi showed normal vessels and sharply outlined optic discs bilaterally. The pupils were round, regular and equal and reacted directly and consensually to light and in accommodation. The right corneal reflex was diminished. There was right-sided hypalgia over the face. The masseter muscles were of normal tone and strength. The remainder of the cranial nerves were unaffected. There was weakness of the right hand. There were no signs of cerebellar dysfunction.

The hematocrit was 52 per cent, hemoglobin 16.7 gm. per cent, red blood cells 5,400,000 per c. mm., and platelets 325,000 per c. mm. Prothrombin time was 76 per cent and 85 per cent of normal on two occasions. The count of his white blood cells was 6,900 with a normal differential. Fasting blood sugar on admission was 122 mg. per cent; subsequent fasting blood sugar and 2-hour postprandial blood sugars were each 106 mg. per cent. The cholesterol, uric acid and blood urea nitrogen were normal. Arterial oxygen saturation was 88.88 per cent.

An electroencephalogram showed focal slowing in the left temporal area. Overventilation, photostimulation and carotid compression produced no change in the record. Ophthalmodynamometry was reported as normal in both eyes.

On Feb. 2, 1961, left carotid angiography was performed. The needle was inserted at the carotid bifurcation. Subsequent films suggested stenosis of the left common and internal carotid arteries at the bifurcation but, because of the placement of the needle, were not considered diagnostic. The patient experienced no new symptoms and no exacerbation of old symptoms following angiography. On Feb. 7, 1961, bilateral common carotid angiograms were performed, confirming the diagnosis of stenosis of the left carotid artery at the bifurcation. Again, the patient’s condition was unchanged by the procedure.

Operation. On Feb. 9, 1961, left carotid endarterectomy was performed under local anesthesia. No shunts were used and the patient experienced no symptoms during the procedure. When the carotid sinus was blocked with procaine, the patient’s blood pressure rose from 140/80 to 180/80 and remained at that level throughout the procedure. Prior to the placement of the bulldog clamps, the patient received 75 mg. heparin intravenously. After occluding the common carotid artery, heparinized saline was injected into the artery proximal to the clamp. After the internal and external arteries were occluded, heparinized saline was injected distal to the clamp. A large plaque was removed from the common, external and internal carotid arteries through a longitudinal arteriotomy. The artery was irrigated with heparinized saline. The internal carotid artery was undamaged several times during the procedure to flush out any atheromatous material. Prior to closing the arteriotomy, the lumen was filled with heparinized saline solution. The clamps were removed from the external carotid, common carotid and finally the internal carotid artery in that order. The internal carotid artery had been occluded for a total of 45 minutes.

Course. At the conclusion of the procedure a rapid neurological examination showed no increase in weakness or difficulty in speech. He was able to count fingers with either eye.

Two hours later, the patient complained of difficulty in seeing with his left eye. It was noted that the vessels in the left fundus were much smaller than in the right fundus. He was able to count fingers with either eye at that time. Shortly thereafter ophthalmodynamometry was attempted; the arterioles in the left eye were narrow.

Received for publication January 18, 1963.

* This work was supported in part by USPHS Grant No. HP-4766, and in part by National Heart Institute Grant HE-04766 (Surg.).
and pale; no pulsation or change in caliber could be effected by increasing pressure from the tonometer. The venules showed "box-carrying," i.e., clumping together of the red blood cells with clear areas separating the clumps. Also noted at this time was a highly refractile orange plaque in the inferior temporal branch of the retinal artery. In view of these findings it was concluded that the central retinal artery was occluded.

The same examiner who had performed the preoperative ophthalmodynamometry repeated this test. A systolic pressure of 120 was found in the right eye, zero in the left. During the performance of this test "about a dozen" refractile bodies were noted in various branches of the retinal arteries. The patient had no useful vision in the left eye; he claimed he could see bright light.

The patient was without any other neurological change. Since he had shown no symptoms or signs during operation, their absence could not be relied on as ruling out thrombosis of the internal carotid artery. With ophthalmodynamometry lost as a diagnostic tool, it was felt imperative to determine the patency of the internal carotid artery. The patient was therefore returned to the operating room.

2nd Operation. The incision was reopened and the bifurcation was exposed. The common carotid artery and both branches were found to be pulsating well. The wound was closed in layers.

Postoperatively the patient was started on 5 per cent CO₂-95 per cent O₂ inhalation by nasal catheter for 10 minutes every 90 minutes. On the 7th day the sutures were removed, and the patient was discharged on Feb. 17, 1961. He had no improvement in vision during his hospital stay.

Follow-up visits have shown some return of vision in the superior nasal quadrant of the left visual field. The fundus has remained pale, and the arteries have remained extremely narrow.

Discussion

In several large series dealing with the treatment of carotid occlusive disease by endarterectomy, the authors reported no cases of blindness following operation.1,2,4,9,18 It should be pointed out that these authors were not discussing complications of surgery, but rather were evaluating endarterectomy as the treatment of choice in occlusive disease of the extracranial portion of the carotid arteries.

Tarlov and his associates,16 in 1952, reported a case of occlusion of the retinal artery which occurred in the ipsilateral eye following open carotid arteriography.

Pereze et al.13 described a case of temporary blindness caused by embolism of the retinal artery. They also documented the lesion and its resolution with photographs of the retina taken at various dates after the incident. This episode was reported as a complication of carotid arteriography.

In 546 angiograms, one episode of transient blindness was reported by Coddon and Krieger.8 This episode was ascribed to spasms of the retinal artery.

Falls et al.6 reported 2 cases of temporary blindness which lasted from 10 to 30 minutes, also following arteriography.

Lindner and his colleagues12 reported 1 case of embolism of the retinal artery occurring in their series of 951 patients on whom angiography was performed.

In our clinic, 1 patient had occlusion of the central retinal artery in the left eye immediately following bilateral arteriography. His vision did not return in spite of stellate ganglion blocks, amyl nitrate inhalation and intermittent 5 per cent CO₂-95 per cent O₂ inhalations. Craniotomy was performed. No abnormality of the meninges or chiasm was found. The vessels were noted to be extremely tortuous and diffusely atherosclerotic at the time of operation.

Walsh and Smith17 reported 2 cases of visual disturbances. In 1 patient they found edema of the macula with no change in the caliber of the arterioles. In their other patient bilateral homonymous hemianopsia developed. They also described in detail the blood supply to the visual apparatus and illustrated how occlusions of various arteries affect visual acuity.

The association of transient episodes of unilateral blindness and contralateral hemiplegia with occlusive disease of the internal carotid artery was emphasized by Fisher in 1932.1 He also pointed out the tendency for the amaurosis fugax to cease when the internal carotid artery became completely occluded. At the time of his report and exhaustive review of the literature, he attributed the attacks to vasospasm. He made a strong point of the fact that this assumption was not based on observation of the fundus during one of these attacks. When the occasion presented itself, he made detailed observations of the fundus in a patient who was experiencing an attack of blindness.8 This patient was proved to have stenosis of the internal carotid artery by angiography. The cause of temporary blindness in this patient was established firmly as an embolus to the central retinal artery. The progression of the embolus through the superior temporal arteriole of the retina was described beautifully.

Hollenhorst10,11 described the appearance of bright plaques in the retinal arterioles of 27 patients of a group of 235 with occlusive disease of the carotid arterial system. He also noted these plaques in 4 of a group of 98 patients with occlusive disease of vertebral system. In his series, 35 patients underwent endarterectomy; in 5 of these patients retinal plaques developed during or immediately following the operation.

The appearance of the plaque in the patient described in this report corresponds to the description of Hollenhorst. The clumping together of red cells in the venules of the affected eye ("box-carrying") was also described by Fisher as occur-
ring in his patient with an embolus to the retina. This phenomenon ceased spontaneously as the occluding embolus moved distally.

Two possibilities exist to explain how the embolization occurred. The first is that a thrombus formed at the operative site, was dislodged and produced the embolus to the retinal artery. It would seem that this is unlikely in a patient who received adequate anticoagulation.

The second possibility is that the atheromatous material was flushed into the external carotid circulation and reached the eye through orbital anastomoses.

Dr. William H. Sweet has suggested that instead of completely releasing the clamp on the external carotid artery, it be replaced above the superior thyroid and/or lingual arteries. When the clamp of the common carotid artery is released any atheromatous material would be flushed into a less critical portion of the external carotid circulation.

Summary

In spite of the usual precautions taken to prevent dislodging of emboli from the site of endarterectomy to the intracranial vessels, a patient suffered an embolus to the retinal artery which corresponded to the descriptions of other authors of atheromatous emboli to retinal vessels.

References

15. Sweet, W. H. Personal communication.