Neurological Deficits Following Therapeutic Collapse of Intracavernous Carotid Aneurysms

Report of Two Cases

LAWRENCE STRANGER, M.D.
St. Vincent’s Hospital, and Medical Center, New York, New York

These two cases demonstrate the efficacy of common carotid ligation in dealing with an intracavernous aneurysm, but also point out the possibility of undesirable sequellae associated with the treatment.

Case Reports

Case 1. (J.Q.) A 54-year-old Spanish-speaking housewife from Puerto Rico was admitted on January 15, 1962, complaining of double vision. Her diplopia was progressive and of 6 months duration.

History. She had been hospitalized in Puerto Rico in 1952 for sudden loss of consciousness. At that time, she was diagnosed as being hypertensive and having an abnormality of the cerebral arteries (not described). She recalled no special examinations being performed. The patient had been suffering from generalized headaches which had been occurring more frequently during the past year.

Examination. On admission the blood pressure was 170/110 mm. Hg. There was a left lateral rectus paresis, ptosis of the left lid, and anisocoria, with a larger left pupil. Both pupils reacted to light, fundoscopy revealed moderate arteriosclerotic hypertensive changes. The rest of the examination was normal.

The electroencephalogram was normal. Roentgenograms of the skull showed a radiolucency in the left occiput that suggested a malignant neoplasm. An air encephalogram and examination of the cerebrospinal fluid performed on January 24 were normal. On January 29, cerebral angiography revealed a large saccular aneurysm of the intracavernous portion of the left internal carotid artery (Fig. 1). Cross-compression studies indicated adequate cross filling.

Operation. Manual compression of the left carotid (Matas) confirmed the angiographic impression of collateral adequacy, and on February 2, the left common carotid artery was ligated in continuity.

Postoperative Course. The next morning a total left ophthalmoplegia was noted, including a fixed dilated pupil and ptosed lid. No paresis of the face or limbs was evident. Her blood pressure was erratic.

The second postoperative day she complained of severe left-sided head pain. This persisted and on February 5, a lumbar puncture was performed and clear cerebrospinal fluid was found under 400 mm. H2O pressure. The protein content was 70 mg.%. The patient was placed on reserpine, and her blood pressure stabilized between 140-120/100-70 mm. Hg. Her headache persisted as did the ophthalmoplegia. No change in visual acuity was noted.

Beginning February 10, the patient was gradually elevated from her heretofore flat position, and was allowed out of bed on February 14. The pain tended to localize about the left eye. The corneal reflex remained intact.

Postoperative left carotid angiography performed on February 15 (Fig. 2) demonstrated minimal filling at the site of the sac with the patient either prone or supine.

Because of the severity of the pain about the eye the patient remained hospitalized. On March 1, Dilantin was instituted and narcotics stopped. Her discomfort ameliorated in a few days requiring only salicylates for control. When she was discharged on March 10, 1962, there was still a residual left ophthalmoplegia.

Case 2. (E.M.) A 55-year-old West Indian colored woman was referred to St. Vincent’s Hospital from another institution on February 12, 1962. She had complained of a throbbing pain behind her right eye for 2 weeks. It had begun suddenly and became progressively more severe.

History. Three years before admission, her right eye had begun to “turn in.” There had also been loss of some visual acuity so that she was not troubled by diplopia. During the same interval she had “blackouts” on two occasions resulting in hospitalization at another institution. Lumbar punctures and electroencephalography reportedly revealed nothing abnormal. For the past 2½ years she had suffered from frequent paroxysmal right frontal headaches which had responded to Anacin.

Examination. She was an obese, excitable female with a blood pressure of 210/120 mm. Hg. She had a right lateral rectus palsy and a depressed right corneal reflex. The visual acuity was O.D. 20/100, O.S. 20/40 corrected; the right optic disc was pale. Visual fields (with eccentric fixation) revealed a superior nasal defect on the right. The rest of the examination was normal.

An electroencephalogram indicated a mild to moderate dysfunction of the right cerebral hemisphere, slightly more prominent in the right posterior frontal and parietal regions than elsewhere. Roentgenograms showed enlargement of the right superior orbital fissure and destruction of the adjacent wall of the optic foramen. The sella, posterior clinoids and floor of the right middle fossa showed demineralization. These findings suggested an infra-clinoid aneurysm. This diagnosis was confirmed by angiography on February 16 (Fig. 3). Cross-compression studies indicated adequate cross circulation. This impression was supported by carotid compression (Matas).

Operation. Common carotid ligation in continuity was performed under general anesthesia on February 19.

Postoperative Course. The immediate postoperative course was uneventful other than for the prompt cessation of the retro-orbital pain. The other neurological
Fig. 1. Case J.Q. Preoperative arteriogram.

Fig. 2. Case J.Q. Postoperative arteriogram.
deficits persisted. She refused postoperative angiography, and after progressive mobilization, was discharged on March 1, to be followed as an outpatient.

Second Admission. She returned to the hospital on March 14 because of severe right retro-orbital pain. The right eye was amblyopic and a superior and inferior rectus palsy was noted in addition to the pre-existing lateral rectus paralysis. Carotid angiography revealed non-filling of the aneurysmal sac (Fig. 4). In spite of therapy with analgesics and tranquilizers the intense pain about the eye persisted along with a dull right frontal headache which seemed to radiate downward and posteriorly into the neck. She was discharged on April 7.

Third Admission. The discomfort persisted and the patient was readmitted on May 22 for consideration of trigeminal rhizotomy. The right optic disc appeared totally atrophic. No other significant objective changes were noted. During the period of observation she developed a left axillary hydradenitis with abscess formation requiring drainage. In the convalescent stage from this procedure, the pain subsided considerably. She was discharged on June 12, 1962.

Course. At clinic visits she reports occasional discomfort about the right eye relieved by aspirin. She has remarried, keeps house and is working. There has been no change in the neurological deficits. She is blind in the right eye and the right pupil reacts only consensually to light. The corneal reflex is diminished, but present. The extra-ocular palsies are unchanged.

Discussion

The terms infracarotid (or subclinoid) and intracavernous are both used to describe these aneurysms. The larger series2,9,10 show that these lesions occur most frequently in middle-aged women, often associated with hypertension. Not all infracarotid aneurysms, however, are purely intracavernous.

Fig. 3. Case E. M. Preoperative arteriogram.

Fig. 4. Case E. M. Postoperative arteriogram.

The infracarotid group has 3 general forms: fusiform, serpentine, and saccular. The fusiform and serpentine lesions tend to rise out of the sinus, and cause changes in the overlying visual apparatus. The serpentine lesions may also affect the innervation of the ocular musculature.5-14 Saccular aneurysms generally remain within the sinus affecting the structures in the lateral wall, but upward extension will affect the visual apparatus.4,5 Ipsilateral facial and auditory nerve function and smell have been impaired by large saccular lesions.1,11

Evidently, the various collected series of infraclinoid aneurysms represent a heterogeneous group. Close scrutiny reveals the clinical, radiological and pathological behavior of these lesions to be diverse, as is their response to therapy.

In our 2 cases, common carotid ligations were followed by obliteration of angiographic evidence of an aneurysmal sac. The patients, however, experienced serious new or increased deficits.

Dandy4 and Hamby9 thought that the development of orbital pain suggested enlargement of intracavernous aneurysms. In our cases, collapse or thrombosis of the sac resulted in similar complaints, and in addition, aggravated the ophthalmoplegia and even led to progressive loss of vision (E. M.). Levitt7 reported a similar situation in his case where the lesion was identified at
craniotomy and treated by carotid ligation. An analogous syndrome was found in Fisher and Goran’s case. 11
The mechanism producing these signs is obscure. The collapsed sac may cause traction upon adjacent structures including the dura of the lateral wall of the cavernous sinus. The latter is innervated by the nervus meningeus medius. This nerve has been described 12,13 as having fascicles arising from all 3 divisions of the trigeminal and from the sympathetic plexus of the internal carotid artery. The implications of referred pain are obvious.
Postoperative nonvisualization of the lesion at angiography may not mean that it has really collapsed. Interference with the small vessels arising from the intracavernous carotid artery 12 such as the artery to Meckel’s cave may have caused local ischemia. This seems unlikely considering the available anastomoses and the patency of the parent and adjacent vessels.
Although the pathophysiology is admittedly enigmatic, the mechanical concept of traction by collapse of the thrombosed sac seems most tenable.

Summary
We have reported 2 cases of intracavernous carotid aneurysm which showed (angiographically) collapse of the sac following common carotid ligation, but developed severe additional postoperative neurological deficits. We have postulated a mechanism for the production of these untoward effects.

References
2. DANDY, W. E. Intracranial arterial aneurysms.