COMMENT

Epistaxis complicating a severe head injury is not unusual but when it occurs weeks later its direct relation to the injury is not so apparent.

The differential diagnosis of a neoplasm, arteriovenous fistula or aneurysm was greatly simplified by the right carotid arteriogram which showed a saccular midline aneurysm located anteroinferior to the sella.

The term aneurysm is used because of the density of its contained dye, globoid outline, sharply defined margins, the lack of a venous exit, and its continuity with the internal carotid artery.

The routine lateral and anteroposterior arteriogram views at first suggested an origin from the right internal carotid artery. In an attempt to better define the neck of the aneurysm a left anterior oblique view was made; this revealed an unusually long narrow stalk which was taken to be the ophthalmic branch (Fig. 3).

The ophthalmic artery in this instance arose quite far posteriorly in the cavernous sinus rather than at its usual site of origin at the anterior genu of the internal carotid.

Revascularization of this "trapped" aneurysm via the ophthalmic artery was felt to be unlikely in view of the cessation of pulsation in the isolated segment of the internal carotid artery.

In this particular case the coincidence of the blindness, optic atrophy and aneurysm of the ophthalmic artery following the accident suggests the possibility of an unrecognized fracture involving the lesser wing of the right sphenoid, optic foramen and adjacent sphenoid sinus.

SUMMARY

1. A case of traumatic aneurysm of the ophthalmic artery is summarized.
2. The use of an oblique view in angiography to accurately identify the origin of this aneurysm is illustrated.
3. Indirect control of the aneurysm is outlined.

REFERENCES


TRANIENT LOSS OF VISION FOLLOWING CEREBRAL ARTERIOGRAPHY

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Not a few descriptions of the various complications of cerebral arteriography may be found in the literature, but those related to disturbance of vision have been rare. The authors deem it worth while to record a case in which there was marked reduc-
tion in vision on the injected side within 2 hours of arteriography and complete recovery within 48 hours. The development and course of this phenomenon appeared to be directly related to the arteriographic procedure.

CASE REPORT

J. H. H. No. 591170. A 51-year-old white male from Texas was admitted on the service of Dr. Benjamin M. Baker with hemiparesis of 2 years' duration. The family history was not remarkable except that the patient's mother had been diabetic, although the cause of her death was unknown. The past history was not contributory.

At the age of 40 years, or 11 years before admission, the patient had an episode of left facial palsy, weakness of the left arm, and pain in the left side of the neck. These symptoms lasted only half a day, then disappeared completely; but following that episode he had noted loss of temperature appreciation on the left side of his body.

In May 1949, or 9 years later, he had weakness of the 4th and 5th fingers of his left hand lasting for 2 or 3 days. In June 1949, he awoke one morning and found his left arm paralyzed. Within the next 3 days a flaccid hemiplegia developed, without involvement of the face and with no headaches or loss of consciousness. During the following 6 months strength returned gradually, walking was resumed, and it became possible for him to use the arm and hand to a surprising extent.

In October 1951, 1 month prior to admission, he began to stagger one day and his tongue became awkward but within 12 hours these symptoms disappeared.

Examination. On admission, temperature, pulse, and respiration were normal. Blood pressure was 130/82. He appeared quite healthy but slightly overweight. The eyes, ears, nose, and throat were normal. The thyroid was not felt and there was no lymphadenopathy. Lungs were clear to percussion and auscultation. The heart was normal in size and the rhythm was good. No murmurs or irregularities were noted. The abdomen was soft, somewhat protuberant, and the liver, kidneys and spleen were not palpable. The prostate was normal in size and consistency. Peripheral blood vessels did not seem sclerotic.

Neurologic examination revealed that the pupils were equal, round, and reacted well to light, accommodation and consensual stimulation. There was a full range of ocular motility and nystagmus was not observed. The optic discs were flat, with sharp neuroretinal margins and distinct laminae cribrosae. Retinal vessels were not noticeably narrowed. Facial movements were symmetrical and facial sensation was normal to testing. He walked with some difficulty, dragging the left leg slightly, while associated coordinated movements in the left arm were lacking. Muscular tone seemed equal on both sides but strength was moderately decreased on the left. Tendon reflexes were exaggerated on the same side, and the plantar response was extensor on the left. Abdominal reflexes were present and equal. Clonus was not elicited, nor was the Hoffmann sign. Finger-nose and heel-knee tests were poorly executed on the left, while fine movements were not possible with the left hand. Sensory examination revealed hypesthesia and hypalgesia with impaired temperature appreciation over the left side of the body below the clavicle. Vibratory and position sense, however, were unaffected, and the Romberg sign was negative.

Laboratory Data. Hb. 16.0 gm. WBC 6100 with normal differential. Erythrocytes and platelets normal on smear. Sedimentation rate 18 mm. (corrected). Icterus index 3.0. NPN 30.0. Blood cholesterol 306. Urinalysis revealed a trace of albumin. X-rays of the skull were normal.

Arteriography. On Nov. 26, 1951, percutaneous carotid arteriography was performed on the right side under local anesthesia. One hour previously, he had been given phenobarbital (96 mg.) and demerol (150 mg.) subcutaneously. The preliminary conjunctival sensitivity test using 2 drops of diodrast (35 per cent) was negative. Stellate ganglion block with 1 per cent procaine was carried out prior to dye injection and a good Horner's syndrome developed. Carotid puncture was done two fingers above the clavicle and performed without difficulty. A good exchange was obtained. Four injections of 15 cc. each (total 60 cc.) of 35 per cent dio-
drast were made for stereoscopic AP and lateral views. The lateral films showed the external carotid system was well filled but no dye appeared in the internal carotid intracranially or in the neck. In the A-P views, at the level where the internal carotid arises in most cases, there is a bare suggestion of an occluded internal carotid but the course of the dye was smooth and unbroken in the external.

Course. Immediately after the last injection the patient complained of slight burning of the right side of his face and slight supraorbital aching. He was returned to his room on a stretcher and fell asleep. Upon awakening 2 hours later he discovered the vision in his right eye was greatly impaired and he could distinguish only large objects. He also noted aching of the right globe and in the right supraorbital region.

Examination made 4 hours after the injection showed the Horner’s syndrome had disappeared. There was a large central scotoma of the right eye which was absolute for a 5 mm. white object at 1 foot and incorporating the blind spot. Peripheral fields were constricted but the nasal loss was greater. Visual acuity was limited to counting fingers at 1 foot. On fundoscopic examination, the right disc was of normal color. The neuroretinal margins were slightly blurred nasally and superiorly. Arterioles were smooth and of normal caliber, while the veins were not overfilled. The macular area appeared rather unusually reddened and indistinct but no hemorrhages, exudates, or sludging were noted. The appearance of the left fundus was similar except there were no corresponding alterations in the macular area.

Four hours later, or 6 hours after the arteriography, vision was unimproved. A stellate ganglion block was done at this time with 1 per cent procaine. Thirty minutes later the patient volunteered the information that his vision was improving. When seen again 16 hours after the onset of visual loss he stated vision had continued to improve and examination revealed he could read large letters and the scotoma was much reduced in size. Another stellate block was done at this time. Twenty-one hours after the onset of visual symptoms, the scotoma was not elicited and peripheral fields were but slightly constricted. The right fundus showed marked decrease in the swelling of the macular area. Visual acuity at this time was equivalent to 20/40 on the right. A third (and last) stellate block was then done.

An ophthalmological examination by Dr. Frank Walsh at the end of 48 hours after the onset of visual symptoms revealed the fundi to be normal in appearance. Central and peripheral fields were quite full and no scotomata could be demonstrated with 3/1000 white, red and blue objects.

Further diagnostic procedures were foregone and a repeat arteriogram was considered inadvisable at this time. A letter has been received from the patient, dated Feb. 23, 1952, in which he states his vision is perfectly normal.

DISCUSSION

Clinically, the case just described could be regarded as one of recurring cerebral vascular occlusions. On the basis of the arteriogram alone, thrombosis of the internal carotid artery could not be established with absolute confidence. The lack of filling of this vessel could have been due to congenital absence of the internal carotid, a condition which Dandy had observed, or to spasm or thrombotic occlusion immediately above the bifurcation of the common carotid.

At least the transient visual failure must be considered a complication of the arteriographic procedure, occurring as it did within 2 hours afterward. What mechanism was responsible for this unusual reaction is not entirely clear. Embolism or thrombosis of the central retinal artery or vein or of a retinal vessel can be excluded on the basis of the fundoscopic examinations and such complete recovery.

That spasm may have contributed cannot be excluded. Ecker and Riemenschneider have presented clinical evidence of such a phenomenon related to cerebral arteriography. An anomalous origin of collateral supply of the ophthalmic circulation from the external carotid or the middle meningeal, as mentioned by Walsh.
with spasm of the supplying vessel could have resulted in such a phenomenon.

Transient spasm of the ophthalmic or its derivative would result in temporary anoxia of the retina. Noell and Chinn\textsuperscript{11} have shown experimentally in electroretinographic studies on rabbits that transient anoxia produces significant changes in the optic tract potentials.

That sensitivity to diodrast could be a factor was apparently excluded by the negative conjunctival test. In any case it seems extremely unlikely that such a discrete, unilateral alteration of vision could be explained on an allergic basis alone.

The complications of cerebral arteriography reported in the literature are numerous and include urticaria,\textsuperscript{12,15} fever,\textsuperscript{12} local reactions in the neck,\textsuperscript{6,15} cervical sympathetic palsy,\textsuperscript{5,15} recurrent laryngeal nerve paralysis,\textsuperscript{6} vomiting,\textsuperscript{6,15} hemiplegia (total or partial, permanent or transitory),\textsuperscript{4,5,6,8} convulsions,\textsuperscript{5,6,8,9} aphasia,\textsuperscript{6,9} air embolism,\textsuperscript{10} thrombosis of the internal carotid artery\textsuperscript{5,12} and rarely, fatal outcomes.\textsuperscript{5,8,9}

Weekers\textsuperscript{14} reported a case of occlusion of a retinal artery following cerebral arteriography. In his case there was a convulsive seizure immediately afterward, followed by hemiplegia which abated. The next day the patient noted a superior field defect in his right eye. Visual fields showed a superior nasal quadrant defect and a large zone of white edema in the submacular region of the right eye. There was permanent loss of vision in the affected quadrant. Weekers considered that embolism was most likely responsible and due to a floating blood clot, fragment of tissue, or dislodged atheromatous plaque.

Curtis\textsuperscript{6} described a case of focal seizure on the contralateral side following arteriography and brief loss of consciousness. Later in the same day, the patient noted some impairment of vision. Ophthalmologic examination 2 days after the injection revealed spasm of the retinal arterioles and a central scotoma which did not resolve.

Falls, Bassett and Lamberts\textsuperscript{4} mentioned 2 cases of visual loss following arteriography. In 1 case this lasted but 10 minutes and in the other for 30. Neither case was studied during the period of visual loss however and ophthalmologic examinations were negative.

In a series of more than 700 cerebral arteriographies which have been conducted at this hospital since 1948 no similar complication has occurred.

**SUMMARY**

A case of transient visual failure following cerebral arteriography is described. This was characterized by unilateral central scotoma on the injected side with constriction of peripheral fields occurring within 2 hours after injection of 35 per cent diodrast and with complete recovery within 48 hours. The possible mechanisms responsible for this reaction are discussed. Therapeutic stellate ganglion block with 1 per cent procaine may have been beneficial. When impairment of vision occurs following cerebral arteriography and there is no ophthalmologic evidence of vascular alteration the prognosis for restoration of vision is probably favorable.

**REFERENCES**

It seemed to us some years ago that there was need for an instrument to apply silver or tantalum clips at various angles with a longer handle than the Cushing and McKenzie models and increasing the power by making it double-jointed. The instrument herein described not only met the above requirements, but, in addition, supplied a more secure grip upon the clip. It has been used by us and has proven its usefulness for a number of years. The three models commonly used are shown here. The first (Fig. 1) has a straight tip; the second (Fig. 2) has an offset tip and the