A NEW ASPECT OF COLLATERAL CIRCULATION IN OCCLUSION OF INTERNAL CAROTID ARTERY

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Since Egas Moniz (1937) first demonstrated a case of occlusion of the internal carotid artery by angiographic means, the appearance of this condition has become familiar to all workers in this field, and many cases have been published and analyzed.

Spontaneous occlusion of the internal carotid artery is nearly always caused by thrombosis, secondary to atherosclerosis, and most commonly occurs in the sinus region, i.e., just distal to its origin from the bifurcation of the common carotid artery. Next in order of frequency is the cavernous region followed by the cerebral and, more rarely, the petrous portions. The cervical region is the area least likely to be affected.

Samuel, in a careful study of 82 random autopsy cases, supported this incidence by detection of the degree of atherosclerosis present in the internal carotid arteries examined microscopically. Of these cases, 74 showed atherosclerosis of varying degree; 15 showed gross narrowing of at least one internal carotid artery amounting to near, or almost complete, obstruction; 4 cases showed complete obstruction of one internal carotid artery, and in 1 further case the common carotid artery itself was obstructed.

This evidence strongly supports the conjecture that in occlusion of the internal carotid artery it is far more common for an adequate collateral circulation to be formed than not, as judged by the absence of neurological defect in most of these cases. It is very possible that in these cases there was a gradual onset of the occlusive process, whereas a sudden occlusion is much more likely to produce neurological changes. Development of surgical techniques for the gradual production of carotid occlusion has been necessitated by the need for collateral circulation to become effective.

Several workers, among these Torkildsen and Koppang, Taveras et al., Thiébaut et al., and Finkemeyer, from their angiographic studies, described various anastomotic pathways that are available to such patients and that may limit the initial disability following such a vascular accident.

The vascular routes described are as follows:

I. Through the circle of Willis.
   A. Via the anterior communicating artery. Filling of the cerebral circulation of the affected side is obtained from the opposite internal carotid artery via the anterior communicating artery.
   B. Via the posterior communicating artery. Although the authors have not seen such a case demonstrated by vertebral angiography, a study by Hutchinson and Yates leaves no doubt that the contribution from the vertebral arteries may be of great importance.

II. Via the external carotid artery of the same side.
   A. Between the lateral nasal and angular branches of the external carotid and the ophthalmic artery.
   B. Between the anterior branches of the superficial temporal artery and the ophthalmic artery.
   C. Between orbital branches of the middle meningeal artery and the ophthalmic artery.
   D. Between infraorbital branches of the internal maxillary artery and the ophthalmic artery.
   E. Between the middle meningeal artery and a meningeal branch of the internal carotid artery.
   F. Between inconstant branches perforating the dura mater to anastomose with arteries on the surface of the brain.

III. Via anastomoses among end-arteries of the anterior, middle and posterior cerebral arteries, probably their meningeal branches.

We have seen several cases in which collateral circulation through the ophthalmic artery is well demonstrated (Case 1).

In a recent case (Case 2), however, the unusual order of filling of the internal carotid artery distal to a demonstrated occlusion of this vessel suggested strongly the presence of a further collateral circulation which feeds blood into the internal carotid artery in its petrous portion, as well as by the usual ophthalmic route.

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FIG. 1. Case 1. Showing anastomotic filling of the carotid siphon and its branches via a distended ophthalmic artery (arrow).

CASE REPORTS

Case 1. D.S., a woman of 47, was seen in hospital on Nov. 19, 1957. At the age of 37, while in good health and with a normal blood pressure, sudden left hemiparesis developed, more severe in the arm. There was moderate headache for 4 days. Good recovery from the hemiparesis followed.

Since that time there had been persistent deep-seated pain in the left side of the body with pronounced hyperesthesia. It was for this that the patient was admitted to hospital.

Neurological examination revealed a blood pressure of 130/80. A minimal left hemiparesis, complete homonymous hemianopsia sparing the macula, and pronounced left-sided cortical sensory changes, including astereognosis and the extinction phenomenon, were noted. Hyperesthesia on the left side of the body was conspicuous.

On Nov. 21, 1957 right carotid angiography was performed under local anesthesia (Fig. 1). There is obstruction of the internal carotid artery as it is not seen filled except distal to the origin of the ophthalmic artery throughout the series. The ophthalmic artery is markedly dilated and anastomotic vessels are seen well filled around the orbital region. There is good filling of the middle cerebral artery and its branches. There is poor filling of the anterior cerebral group.

It is noted that there is no retrograde flow of contrast medium down the carotid siphon, nor is there filling of the carotid artery at the site of entry into the carotid canal.

Subsequently, the patient was discharged under medical treatment.

Case 2. J.H., a man of 62 years, was first seen on Jan. 23, 1958. He had been known to have moderate hypertension for a number of years. In 1955 an attack of weakness and numbness of the left arm occurred, slowly subsiding in 2 months. There was no headache. Late in 1956 attacks of numbness without weakness ensued in the left face and arm. These continued intermittently until seen in 1958. Their duration was approximately 30 minutes and they were sometimes accompanied by throbbing in the right side of the head. In the period since the onset of these attacks the patient had become aware of slowly progressing clumsiness in the left arm.

Neurological examination revealed mild weakness and clumsiness of the left arm. The hand could still be used for fastening buttons. Slight weakness of the right side of the face was also apparent. The visual fields were full. There were no sensory changes, and no convincing asymmetry of the reflexes could be demonstrated. The remainder of the neurological findings were normal.

On Feb. 16, 1958, the patient was seen again on admission to hospital. A striking change had taken place. Marked bilateral weakness and clumsiness were apparent. Large buttons on the pajama jacket could not be fastened or unfastened using both hands. The patient was very confused. He walked with difficulty using a shuffling gait. There was no defect of vision or speech.

Roentgenograms of the skull were normal and lumbar puncture yielded normal cerebrospinal fluid. On Feb. 19, 1958 a very pronounced right hemiparesis was apparent. Speech and visual fields remained apparently unaffected. The patient was bedfast. Right carotid angiography was performed under local anesthesia. The first phase (Fig. 2) shows complete occlusion of the internal carotid artery about 1 cm. distal to its origin. The internal carotid is seen filled more distally at two separate sites, the higher of the two being in the carotid siphon and the lower of the two being in the carotid canal. The filling of the siphon is derived from the oph-
Fig. 2. Case 2. Anteroposterior and lateral films taken immediately after injection of contrast material show filling of the internal carotid artery in the cavernous and petrous parts. The upper two arrows show the separation of the two areas of contrast medium. The lowest arrow in the anteroposterior view (left) shows the lowest limit of filling of internal carotid artery and is separate from the prominently filled internal maxillary artery. The lowest arrow on the lateral view (right) shows the occluded internal carotid artery.

[In the original films, the internal carotid artery in the carotid canal is clearly visible. For purposes of adequate reproduction, the petrous portion of the temporal bone has been darkened slightly on the photographs to provide greater contrast.]

The course was rapidly downhill. Within 24 hours the patient was in coma, responding only to deep pain. Supportive treatment was given. Death occurred on Mar. 12, 1959. Autopsy was refused.

DISCUSSION

Case 1 shows the common ophthalmic anastomotic pathway expected in these cases. In our opinion, the films of Case 2 appear to indicate two separate anastomotic pathways supplying the internal carotid artery. One of these is the well known channel derived from distal branches of the internal maxillary artery feeding the ophthalmic artery. The other is a communication which must exist between the external carotid system and the petrous portion of the internal carotid artery. Such an anastomosis is described anatomically in Gray's Anatomy. Two branches of the internal carotid artery arise in its petrous portion, the caroticotympanic artery and the artery of the pterygoid canal. The caroticotympanic artery anastomoses with the anterior tympanic branch of the internal maxillary artery and with the stylomastoid artery; the artery of the pterygoid canal is a small inconstant branch and anastomoses with a branch of the internal maxillary artery.

Although demonstration of these minute vessels has not been achieved in these films, such a communication appears to exist in this patient.
SUMMARY

A case of occlusion of the internal carotid artery is discussed. The carotid arteriogram demonstrates two separate anastomotic channels. The one, via the usually described route of the ophthalmic artery, produces filling of the cavernous portion of the internal carotid artery. The other produces filling of the petrous portion of the internal carotid artery and a suggested pathway is described.

A second case of occlusion of the internal carotid artery, showing the common type of anastomosis, is presented for comparison.

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REFERENCES