Extracranial Carotid Artery Occlusion by an Anomalous Digastric Muscle

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On infrequent occasions, lesions other than arteriosclerosis have been encountered as a cause of extracranial carotid artery occlusion with resultant cerebrovascular insufficiency. By and large such lesions are perivascular, producing their effect by mechanical compression, kinking or displacement of the vessel. Neoplasms, trauma to the head or neck, post-traumatic bands and adhesions, as well as bony abnormalities, have been identified as the basic cause. The present report concerns a hitherto unrecognized possible cause, namely an anomaly of the digastric muscle in the neck.

Case Report

R. L., a 47-year-old, self-employed salesman, was admitted to the Neurological Institute of New York on August 14, 1964. He had been in excellent health until 8 days before admission when, at 3 a.m., he went to the bathroom, suddenly lost complete use of the left arm and leg and dropped to the floor. There was no loss of consciousness or evidence of convulsive activity. He was helped to bed and then admitted to a local hospital where the physician noted the presence of a complete left hemiplegia.

For approximately 1 week prior to the present illness the patient had had an upper respiratory infection with nasal stuffiness and sore throat. He had consulted a physician on the day before the vascular accident because of the persistent "sore throat" which was more evident on the right side of the neck. The past history revealed no evidence of a familial disease, hypertension, diabetes, or collagen disease. He had never experienced transient ischemic attacks or blindness.

Examination. He was alert and cooperative, though depressed. Blood pressure was 140/90 in both arms; and the pulse rate 84 and regular. Examination of the neck revealed equal and normal carotid pulsations with no bruits. The carotid compression test was not done.

Neurological findings included a complete flaccid left hemiplegia with a left central facial paresis, left-sided hyperreflexia with a left Babinski sign, and marked cortical sensory loss in the left arm and leg. The fundi appeared normal and the visual fields were full to gross confrontation. The other cranial nerves were normal.

Laboratory examination showed a normal complete blood count and urinalysis. The sedimentation rate was elevated on 2 occasions, with values of 65 mm. and 63 mm. Fasting blood sugar was 100 mg. per cent and the BUN 31 mg. per cent. A complete battery of liver tests was within normal limits. Three lupus erythematosis preparations were negative. The cerebrospinal fluid The initial clinical impression was occlusion of either the middle cerebral or intra- or extracranial internal carotid artery. On August 18, 1964, a right brachial arteriogram was performed and complete occlusion of the right internal carotid artery, approximately 1 cm. distal to its origin in the neck, was demonstrated (Fig. 1). The right vertebral and external carotid arteries were normal. A left common carotid arteriogram showed a normal appearance of the extracranial portion of this vessel. Both anterior cerebral arteries filled from the left. A left brachial arteriogram performed on August 24, 1964, showed a normal left vertebral artery.

Operation. On August 25, 1964, under nitrous oxide and oxygen anesthesia, the right extracranial carotid circulation was explored. Dissection was begun at the common carotid artery. As this was carried cephalad the most proximal part of the carotid sinus was noted. At this point there was a large muscle, approximately 3 cm. in width, stretched across the origin of the internal and external carotid arteries (Fig. 2). The mass of muscle appeared to be half tendinous at this point. The constriction of the arterial origins was so marked that it was difficult to insert scissors between the muscle and the internal carotid artery. Numerous large lymph nodes were noted; some were found under the muscle and surrounded the arteries. Dissection was carried upward and the posterior belly of the digastric muscle was definitely identified. The first muscle encountered appeared to take identically the same course as the posterior belly of the digastric, except at a lower level. The sternohyoid muscle and hypoglossal nerve were in normal position.

As the lower muscle was transected, the visible internal carotid artery appeared to be "released." Numerous surrounding matted lymph nodes were removed to facilitate dissection up the vessel. The internal carotid artery was opened and a firm organized thrombus, about 3 cm. long, was removed. The wall of the vessel appeared smooth and glistening with no evidence of atheromata. Attempts at total removal of the thrombus and restoration of flow were unsuccessful. The wound was closed in routine fashion.

Postoperative Course. Pathological examination of the lymph nodes demonstrated chronic lymphadenitis. The patient made an uneventful recovery. With intensive physical therapy, he regained function in the left leg and arm rapidly so that 3 weeks postoperatively he was walking with a brace. Very minimal function in the hand has returned $2\frac{1}{2}$ months postoperatively.

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showed an initial pressure of 200 mm. with crystal clear fluid. There were 5 white blood cells, a sugar of 72 mg. per cent and a protein of 73 mg. per cent. The spinal fluid Kolmer test was negative. An electroencephalogram showed poor organization of the alpha activity on the right side and increased right-sided slowing, maximal in the right temporal region.



FIG. 1. Right brachial arteriogram showing patent vertebral and external carotid arteries and complete occlusion of internal carotid artery 1 cm. distal to origin.

Discussion

With the present increasing interest in extracranial occlusive vascular lesions as one of the causes of cerebrovascular accidents, it seems desirable to report unusual instances of mechanical compression of major arterial channels.

Perhaps the most frequently observed mechanical precipitating factor has been that of kinking of the carotid vessel.^{1-4,7,9,10,11} Head injuries, penetrating injuries of the palate and pharynx, wounds and contusions of the neck, cervical rib and neoplasm have also been reported in a smaller number of observations.^{6,8}

In this particular case, an anomalous duplicated posterior belly of the digastric muscle was present. This was stretched diagonally across the origin of the internal and external carotid artery markedly compressing these structures. The posterior belly of the digastric muscle was present above this in its usual position. Obviously this anomalous condition had been present for some time and one must speculate that the precipitating cause of the occlusion may well have occurred as a result of the associated marked lymphadenopathy surrounding these structures. Apparently the latter was secondary to a recent upper respiratory infection. Certainly arteriosclerosis does not appear to have been a factor since thorough examination of the lumen of the vessel revealed no evidence of atherosclerotic plaque formation. A review of anatomical texts shows that anomalies of the digastric muscle may be present in multiple forms. Gray's⁵ anatomical text notes that the anterior belly may be double and the posterior belly may be connected by a slip to the middle or inferior constrictor muscle.



FIG. 2. Occlusion of internal carotid artery by anomalous digastric muscle. (Redrawn from photograph of operative field.)

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This condition may be bilateral and a similar anomaly on the opposite side may well exist in this patient.

Summary

An unusual occurrence of compression of the extracranial portion of the carotid artery by an anomalous digastric muscle is presented. The anatomical relationship of this anomaly to the carotid vessels in the neck is discussed.

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Addendum

The patient was readmitted to the Neurological Institute on April 15, 1965. Two weeks prior to this he had what appeared to be a grand mal seizure, while asleep. Two days later he had an aching pain over the left sternocleidomastoid area which subsided in 2 days. At this time there developed intermittent tingling of the fingers of the right hand and episodic numbness of the right foot. No headaches, visual changes, motor weakness or speech problems were observed.

Examination at the time of readmission showed persistence of the old left hemiparesis. The patient could walk without assistance; however, he had almost no function of the left hand and arm. There was no bruit or tenderness in the neck. The symptoms could not be reproduced by movements of the head or neck. The patient had not recently had an upper respiratory infection.

Because of these unilateral symptoms referable to the opposite cerebral hemisphere and the abnormal muscle previously demonstrated, it was felt that surgical investigation of the left carotid region was indicated. At operation on April 19, 1965, no anomalous constricting structures were found. However, the left internal jugular vein overlay the common carotid vessel and was lateral to it. No lymphadenopathy was seen and the vessels themselves appeared normal.

Since operation, the patient had noted none of the original symptoms. Their exact cause remains unexplained.