GLOSSOPHARYNGEAL NEURALGIA
CAUSED BY COMPRESSION OF THE NERVE BY AN Atherosomatous Vertebral Artery

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Apart from reports relating to intra- and extracranial tumours, few observations have been published on glossopharyngeal neuralgia in which a precise cause of this disorder was discovered. Pope reported a case of neuralgia caused by pressure on the glossopharyngeal nerve by a thrombosed vertebral artery, and Lilie and Craig published another in which the cause was an anomalous artery in the cerebellopontile angle. Ferey et al. and Deparis described cases of glossopharyngeal neuralgia caused by arachnoiditis and perineural fibrositis.

In the case presented here, partial degeneration of the glossopharyngeal nerve resulted from compression by an atherosomatous vertebral artery.

CLINICAL NOTES

Van A. J., a woman of 77, had had recurrent attacks of angina pectoris since the age of 65. She was first admitted to hospital in February, 1953, suffering from a blood dyscrasia caused by nutritional deficiency. She was readmitted on May 24, 1953, complaining of a neuralgic syndrome which had commenced suddenly 5 days previously.

She had paroxysms of excruciating stabbing pain of short duration brought on by swallowing and movements of the tongue. The pain radiated from the left tonsillar fossa to the left ear and prevented her from eating and swallowing. Short intervals of relief occurred between the attacks of pain. Cocainization of the tonsillar region produced rapid though transient relief.

Examination. The patient showed a generalized severe degree of atheroma. The blood pressure was 160/90 mm. Hg. Otolaryngological and neurological findings were negative. Wassermann’s reaction was negative. The blood urea was 26 mg. per cent. The blood platelets were decreased in number: 129,000 per c.mm. on the first occasion, 48,000 per c.mm. on the second occasion. The bleeding and coagulation times were normal. The prothrombin time was 65 per cent of normal. The cerebrospinal fluid contained 3 cells per c.mm.; total protein was 22 mg. per cent. The patient died suddenly on June 9, 1954.

Postmortem examination disclosed myocardial infarction, purulent bronchiolitis, bilateral hydronephrosis and a dissecting aneurysm of the abdominal aorta. A small focus of softening was present in the left putamen. No other macroscopic lesion was found in the brain.

The cerebral arteries showed a severe degree of atheroma and even the smallest parenchymatous vessels were affected. The left vertebral artery ran on the lateral surfaces of the cervical spinal cord and medulla oblongata, then turned towards the basilar sulcus of the pons and alone formed the basilar artery. Below this transverse groove the artery gave rise to the left posterior inferior cerebellar artery. The right vertebral artery, after running its normal course, curved backwards and outwards at the level of the transverse groove separating medulla oblongata and pons and terminated in the right posterior inferior cerebellar artery (Figs. 1 and 2). The origin of the vertebral arteries in the neck was not investigated. At the level of the lower part of the medulla oblongata, the diameter of the right vertebral

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artery was 2 mm. and the diameter of the left vertebral artery was 6 mm. The circle of Willis showed inequality in the calibre of the two posterior communicating arteries.

At the level of the transverse groove, the enlarged, atheromatous, left vertebral artery made a depression on the lateral surfaces of the medulla oblongata and pons and on the undersurface of the cerebellum (Fig. 3). In the bottom of this depression, the roots of the nerves IX, X, and XI and the posterior inferior cerebellar artery were compressed. The trigeminal nerves were normal.

The roots of the right and left cranial nerves IX, X, XI, and XII were embedded in paraffin after mordanting in fluorochrome. The cranial nerves IX and X were embedded together. The nerves were cut transversely and stained by the Weigert-Pal method: some sections were counterstained by van Gieson's method.

Fig. 1. Inferior aspect of the brain. The vertebral arteries are not united: the basilar artery is formed by the left vertebral artery only.

Fig. 2. The dissected circle of Willis demonstrates nonunion of the vertebral arteries.
Fig. 8. The left vertebral artery is retracted to demonstrate the depression on the left of the transverse groove. It gives off the left posterior inferior cerebellar artery which runs downwards behind the roots of cranial nerve XII. Cranial nerve IX cannot be clearly distinguished from X.

The right cranial nerves IX, X, XI and XII and the left cranial nerves XI and XII were unaltered but demyelination was seen in several bundles of radicular fibres of the left cranial nerves IX and X (Fig. 4). One of these bundles was almost completely demyelinated; the others were partly demyelinated. Portions of the roots of the left and right cranial nerves IX and X were teased and stained by Sudan: neutral fat was not observed in them.

Fig. 4. (Above) Radicular fibres of the right cranial nerves IX and X. Their myelin sheaths are intact. (Below) Radicular fibres of the left cranial nerves IX and X. Some myelin sheaths are partly and others severely degenerated (Weigert-Pal method).
DISCUSSION

The nonunion of vertebral arteries was regarded as an extremely rare anomaly. McMinn⁷ published 1 case, discovered accidentally at dissection, and quoted only 2 other similar observations, one of Berry and Anderson⁸ and the other of Batueff.¹ Frequent use of vertebral angiography has shown, however, that this absence of union of the vertebral arteries is not a rare anomaly. Among 221 patients subjected to vertebral angiography, Radner⁹ saw 7 cases—that is to say, nonunion of the vertebral arteries in 3.1 per cent.

Our observation is interesting chiefly because of the etiology of the neuralgia which was related to the pressure of the atheromatous elongated left vertebral artery on the brain stem rather than because of the anatomical anomaly. The paroxysms of pain had the clinical character of “essential” neuralgia in spite of their organic origin. The objective sensory changes in the glossopharyngeal area were not investigated in the present case. If the frequency of the neuralgia among old people is taken into account (30 per cent beyond the age of 60 years according to Schachter¹⁰) one can imagine that such a compression of radicular fibres of the nerve by a lengthened atheromatous artery is not exceptional. The clinical problem of the dolicho-arteries put forward by Leriche¹¹ should be considered at the cephalic level. It remains to be explained, however, why the pain occurred so recently, while the malposition of the artery was apparently present for a long time.

SUMMARY

A report on a patient aged 77 years with advanced atheroma who suffered from “essential” glossopharyngeal neuralgia is presented. Postmortem examination disclosed compression of the radicular fibres of the nerve by an elongated, atheromatous vertebral artery. The compressed nerve roots showed partial demyelination. This observation suggests that, in some cases, essential glossopharyngeal neuralgia may be caused by compression of the nerve by an atheromatous artery.

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REFERENCES