Short report

Vertebro-basilar ischaemia with thrombosis of the vertebral artery: report of two cases with embolism

B GEORGE,* C LAURIAN†

From the department of Neurosurgery, Hopital Lariboisiere,* and the department of Vascular Surgery, Hopital Saint-Joseph,† Paris, France

SUMMARY Two cases with vertebro-basilar infarcts associated with severe stenosis of the vertebral artery in its cervical part are reported. Complete thrombosis of the vertebral artery was observed after a short delay in both cases. As the carotid arteries were normal and the controlateral vertebral artery was dominant, an embolic mechanism was suspected. No new event occurred in the following one and three years respectively after the stroke. These two cases demonstrate the possibility of embolism from severe stenosis of the cervical vertebral artery. This mechanism could explain some of the infarcts related to vertebral artery occlusion.

The mechanisms causing vertebro-basilar ischaemia with thrombosis of the vertebral artery are not well understood. Sometimes the ischaemia appears likely to be related to low flow in the vertebro-basilar system; in other cases, an embolic mechanism is suggested, but proof is rarely afforded. We report two cases in which we believe that embolism from a severe stenosis occurred, and in which the stenosis subsequently evolved to complete thrombosis after a short delay.

Case reports

Mr P, aged 55 years, was referred having suffered from sudden onset of asthenia and hyperomnia and, 8 hours later, dysarthria, diplopia, and facial palsy. On admission hyperomnia was still present, associated with a Parinaud's syndrome, palsy of the left III, IV, and VII cranial nerves and rotatory nystagmus. CT scan showed a small lucent zone on the left side of the pontopeduncular junction. Bilateral brachial arteriography showed a normal right vertebral artery and a severe stenosis on the left vertebral artery between C1 and C2; the right was the dominant vertebral artery. The patient left the service for one month; on return, most of the symptoms had regressed; only the IV cranial nerve palsy and the rotatory nystagmus remained. On arteriography, the left carotid artery was normal and the left vertebral artery was completely thrombosed without any filling of the vessel (fig 1a). One year later, no new event had occurred and the patient was free of all symptoms apart from the rotatory nystagmus.

Mr D, aged 45 years, was referred for a sudden attack of headache, vomiting, and gait ataxia. The day before, he experienced a short episode of bilateral loss of vision and 6 months before, had had two episodes of vertigo. Neurological examination showed gait ataxia, left dysmetria and hypermetria, and right ptosis. Lumbar puncture was normal; isotopic scanning was normal; bilateral brachial arteriography showed a small ostial stenosis on the right vertebral artery and a severe ostial stenosis on the left vertebral artery (fig 1b). The right vertebral artery was the dominant vessel. Both carotid arteries were normal. Five months later, the cerebellar syndrome had decreased but not disappeared. A second arteriography demonstrated a complete thrombosis of the left vertebral artery. Three years later, the patient has improved but still has a gait ataxia and mild left hypermetria.

These two cases are taken from a series of 66 patients with vertebro-basilar ischaemia seen in a 3 year period. Thirty-two cases were with infarcts of the posterior inferior cerebellar arterial territory (cerebellar or lateral medullary infarcts) (PICA I) and 34 cases with involvement of the basilar trunk territory (brain stem or occipital infarcts). (BT I). Complete arteriographic investigations were performed in 44 cases. Significant lesions of the cervical vertebral artery (stenosis of more than 50%) or occlusion were found in 65% of BT I and in 80% of PICA I (15 significant stenosis and 17 occlusion). Topography of the lesions was mainly ostial for occlusion and ostial and in the third portion for stenosis. Bilateral lesions
were observed in 16 cases (36%). In eight cases arteriography showed insignificant lesions or was normal.

**Discussion**

Many reports deal with vertebro-basilar strokes, most of which are related to occlusion of the vertebral artery and especially of its intracranial portion.\(^1\)\(^-\)\(^11\) Fischer,\(^12\) from anatomical studies, found 14% of stenosis of the vertebral artery in 162 cases, but none was symptomatic. He concluded that lesions of the vertebral artery in its cervical portion, rarely led to stroke.\(^13\) Indications for surgery for cervical stenosis are mainly based on vertebro-basilar insufficiency with transient ischaemic manifestations.\(^14\) The two cases reported here, concern stroke with permanent signs associated with severe stenosis of the cervical vertebral artery.

Stenosis of the vertebral artery, as in the carotid artery, may cause symptoms by two mechanisms. The most frequent is a reduction of flow in the vertebro-basilar system. This mechanism is suspected in bilateral lesions or when associated with carotid and vertebral lesions. In our series, 20 occlusions were discovered after a stroke in 17 patients.\(^11\) In 16 cases bilateral lesions were found presumably causing a fall of blood flow. But in 16 other cases, the lesion was unilateral, and the contralateral vertebral artery was of good size, as in the two cases reported here. In these cases, we suspect an embolic mechanism, particularly in a case with cerebellar or occipital infarct with a normal posterior-inferior cerebellar artery and posterior cerebral artery.

Embolism is frequently advanced to explain infarcts in the carotid artery territory, but usually it may only be presumed in the vertebro-basilar system.\(^4\) Nevertheless, embolism in the posterior circulation has been documented on post-mortem examination.\(^1\)\(^-\)\(^7\) Meyer\(^15\) and Sundt\(^16\) also have
emphasised the rôle of embolism from proximal vertebro-basilar occlusion. Strokes from non-atheromatous lesions also have been reported.\textsuperscript{15–18} Fischer\textsuperscript{18} states that only 4% of vertebral atheromatous stenosis show ulceration. Severe stenosis, at least, seems to produce a high risk of embolism such as on the carotid artery. As occlusions of the vertebral artery are often diagnosed late after the onset of ischaemia (average 1 month in our cases), ischaemia could sometimes be related to embolism from steno-
sis changing to thrombosis when arteriography is done. These points should be taken into account when new techniques of revascularisation (occipital artery to posterior-inferior cerebellar artery)\textsuperscript{9–16} and carotid artery to vertebral artery\textsuperscript{19–20} are discussed. The best indications should be bilateral lesions and unilateral symptomatic stenosis.

References


\textsuperscript{2} Sypert GW, Alvord EC. Cerebellar infarction. A clinicopathological study. \textit{Arch Neurol} 1975;32:357-63.

\textsuperscript{3} Peiffer J, Haas H, Boelldart J. Basilaris-vertebralis-
aneurysmen als ursache schneiaber halswirbelsaulen-
syndrome. Klinische und morphologische untersun-

\textsuperscript{4} Caplan LR. Occlusion of the vertebral or basilar artery. Follow-up analysis of some patients with benign outcome. \textit{Stroke} 1979;10 3:177-82.

\textsuperscript{5} Yates PO, Hutchinson EC. Carotido-vertebral stenosis. \textit{Lancet} 1957;1:2-8.

\textsuperscript{6} Yates PO, Hutchinson EC. Cerebral infarction. The role of stenosis of the extracranial cerebral arteries. \textit{Spec Rep Sec Med} 1961;300:1-95.


\textsuperscript{9} Khodadad G, Singh RS, Olinger CP. Possible prevention of brain stem stroke by microvascular anastomosis in the vertebral system. \textit{Stroke} 1977;8:316-21.


\textsuperscript{12} Fischer CM, Gore I, Okabe N, White PD. Atherosclero-
sis of the carotid and vertebral arteries. Extra-

\textsuperscript{13} Fischer CM. Occlusion of the vertebral arteries causing transient basal symptoms. \textit{Arch Neurol} 1970;22:12-19.

\textsuperscript{14} Cormier JM, Laurian C. Surgical management of vertebo-basilar insufficiency. \textit{J Cardiovas Surg} 1976;17:205-23.


\textsuperscript{17} Mauruierre F, Bouillat J, Ferry AM, Goutelle A, Garde A. Syndrome de Walleberg dû à un anévrysmse fusiforme de l’artère vertébrale droite extracra-
nienne d’origine traumatique probable. \textit{Rev Oto-

\textsuperscript{18} George B, Laurian C, Derome P, Guilmet D. Pontage sous-clavier vertebral en C1-C2 pour dysplasie anévrismale. Intérêt et possibilité de l’abord chir-

\textsuperscript{19} Corkill G, French BN, Michas C, Cobb CA, Mims TJ. External carotid vertebral anastomosis for vertebo-

\textsuperscript{20} George B, Laurian C. Surgical approach to the whole length of the vertebral artery with special reference to the third portion. \textit{Acta Neurochirurgica} 1980;51:259-72.