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External Carotid Steal and Lateral Medullary Infarction – is there a Pathogenetic Connection?

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Summary. The case of a patient with occlusion of the external carotid artery is reported; he suffered from vertebrobasilar insufficiency with Wallenberg's syndrome. Arteriography showed stealing of intracranial blood from the vertebral artery via the occipital artery, and thromboendarterectomy was successfully performed. Since no localised processes could be demonstrated could the lateral medullary infarction be due to the haemodynamic changes? In cases of external carotid steal thromboendarterectomy might be the appropriate treatment.

Key words: External carotid steal – Occipital artery – Wallenberg's syndrome – Thromboendarterectomy

Zusammenfassung. Es wird der Fallbericht eines Patienten mitgeteilt, der bei einem Verschluß der A. carotis externa das Bild einer vertebrobasilären Insuffizienz mit Wallenberg-Syndrom bot. Die Arteriographie zeigte ein Steal-Syndrom aus der A. vertebralis über die Occipitalarterie. Eine erfolgreich durchgeführte Thrombendarteriektomie brachte Beschwerdefreiheit. Bei fehlenden lokalisierten Gefäßveränderungen muß die Möglichkeit der hämodynamischen Verursachung des lateralen Oblongatasyndromes diskutiert werden. In solchen Fällen könnte die Thrombendarteriektomie eine kausale Behandlungsmethode darstellen.

Schlüsselwörter: Wallenberg-Syndrom – Intrakranielles Steal-Syndrom – Occipitalarterie – A. carotis externa – Thrombendarteriektomie

Introduction

Since the description of the subclavian steal syndrome [3] haemodynamic factors for the development of ischaemic brain lesions became of greater interest [11]. In

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this condition the reversed blood flow in the vertebral artery causes recurrent brain stem ischaemia–transitory symptoms are more common than brain infarctions [7]. Similar extracranial steal syndromes have been reported in occlusions of the brachiocervical trunk and the common carotid artery.

Fazio [5] demonstrated intracranial steal phenomena in patients with variations of the circle of Willis. Brain infarctions on the opposite side of a vascular occlusion seem to be due to the diversion of blood flow.

Vertebrobasilar symptoms caused by haemodynamic factors in occlusions of the external carotid artery have been described by Barnett et al. [1]; they observed such symptoms as vertigo, drop attacks and visual loss. Most of their cases had atherosclerotic lesions beside the external carotid occlusion. While the development of brain infarction in subclavian steal syndrome is easily explained to be due to the haemodynamic changes it is rather difficult to prove the theory of external carotid steal.

We would like to report the case of a patient with an occlusion of the external carotid artery accompanied by Wallenberg's syndrome in view of the possible connection between the two findings.

Case Report

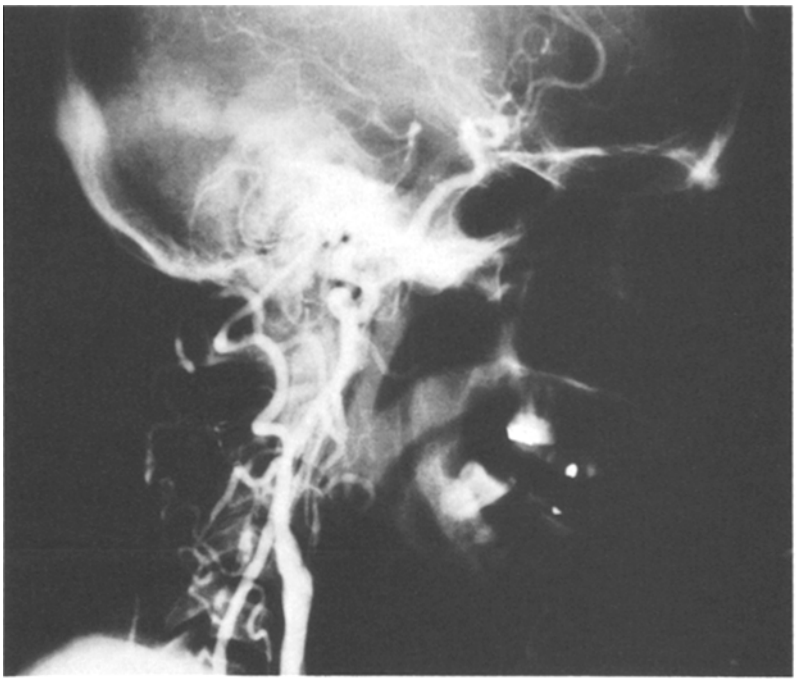
A 52-year-old bricklayer was admitted to our clinic in October 1979. On September 26th whilst walking outside he experienced sudden vertigo, followed by a sensory disturbance of the left side and dysarthria. Because of increasing unsteadiness in walking and repeated vertigo attacks the patient was transferred to us from his home town hospital.

He had a history of vision disorder with a convergent strabismus and nystagmus since birth, and had had a stroke in 1966 with a left hemiparesis, which improved within a few weeks.

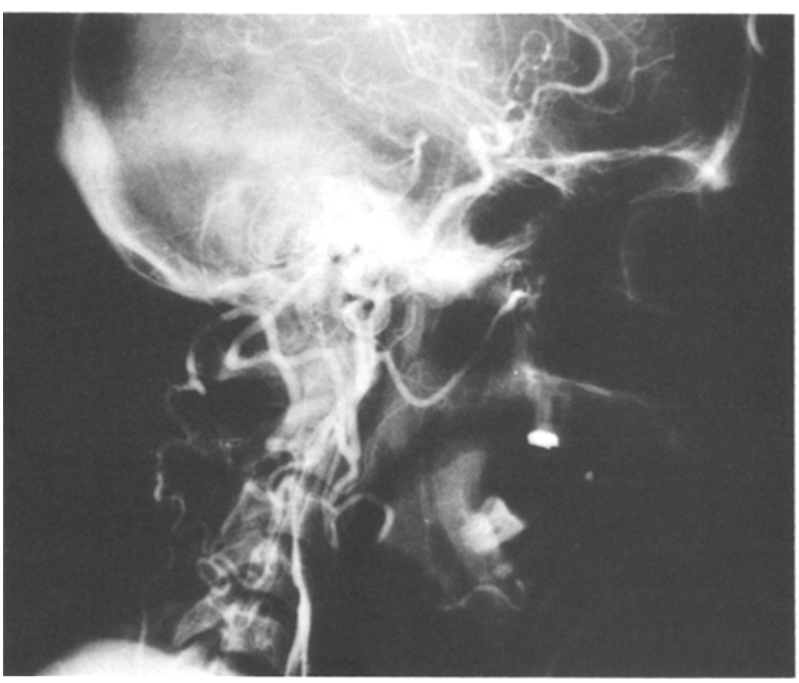
On neurological examination he had in addition to his ophthalmological symptoms a sensory loss in the region of the trigeminal nerve with a diminished corneal reflex and a palate paresis on the right side. There was ataxia of the right arm and leg in the finger-to-nose and heel-to-skin tests combined with disturbed alternating movements. On the left side there was a hypalgesia and diminished temperature perception. The deep tendon reflexes were brisker on the left with a positive sign of Babinski (residuals of the old supratentorial infarction). Apart from a minimal stenosis of the right vertebral artery at its origin arteriography showed an occlusion of the right external carotid artery, which was filled retrograde from connections between the occipital artery and muscular branches of the vertebral artery (Fig. 1a and b). Beside this anastomosis there were collaterals from the ascending and deep cervical arteries. Left retrograde brachial injection showed adequate filling of the vertebral artery, and there were normal findings on cranial computed tomography.

During his hospitalization the patient suffered from repeated vertigo attacks, even during treatment with acetylsalicylic acid and dextran infusions. At the end of October 1979 the patient underwent surgical treatment: thromboendarterectomy of the carotid bifurcation and plastic reconstruction with a vein patch from the leg.

Though there was a complete occlusion of the external carotid artery at its origin the blood pressure measurement during operation was 90 mm Hg in the artery indicating the sufficiency of the collateral supply. There was no hypertrophy of the thyroid artery, the lingual or the facial artery; and it seemed unlikely that the blood came via these branches. The operation was successfully carried out, and at follow-up the patient remained symptom free,–in particular he had had no more vertigo attacks. Pathological examination of the vessel revealed an atherosclerotic lesion. On re-examination in February 1981 there was no palate paresis, the disturbance of pain and temperature perception had diminished and the hemiataxia had completely disappeared.



a



b

Fig. 1a, b. Right retrograde brachial injection (preoperative) showing occlusion of the external carotid artery. Retrograde filling of the occipital artery (**a**) via muscular branches of the vertebral artery (**b**)

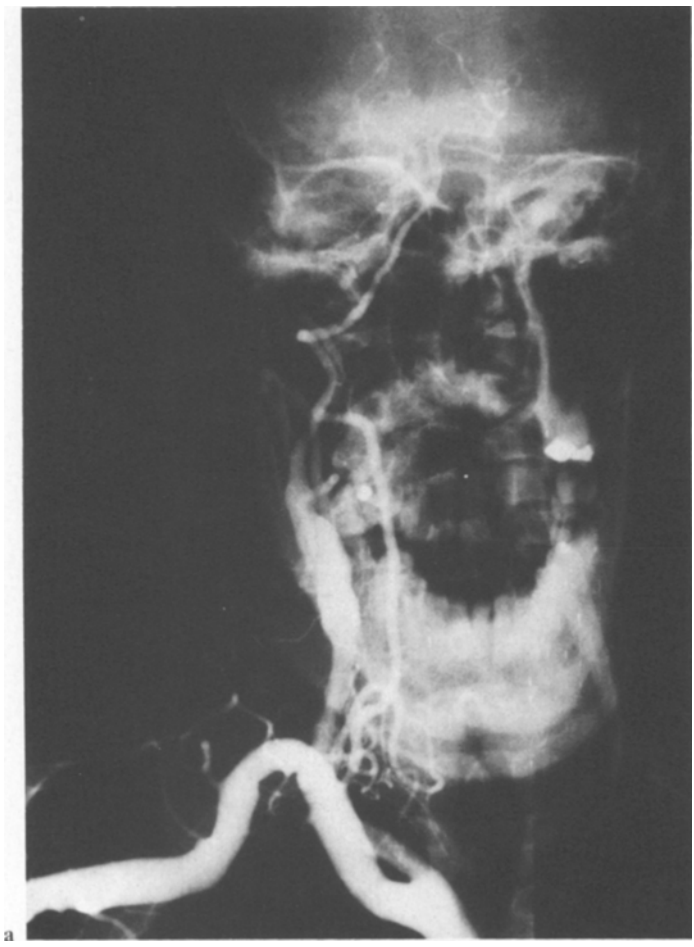


Fig. 2a, b. Right retrograde brachial injection (postoperative) anteroposterior (**a**) and lateral projections (**b**). Carotid and vertebral vessels are well visualized. Circulation in the region of operation is restored. No retrograde filling of the external carotid artery via collaterals

An angiographic study was performed using a retrograde right brachial injection, and this time the carotid arteries were normal with a normal lumen. There was no longer any retrograde filling of the external carotid artery from the vertebral artery, and there was no change in the slight stenosis at the origin of the vertebral artery (Fig. 2a and b).

On re-examination on October 22nd 1982 there was still a discrete disturbance of pain and temperature, though the patient had been symptom free. Angiographic study again showed normal findings at the site of the operation, but this time there was a stenosis of the left internal carotid artery at its origin. A thromboendarterectomy of this asymptomatic stenosis was carried out successfully and pathological examination again showed an atherosclerosis of the vessel.

Discussion

Since Wallenberg reported in 1895 [15, 16] a case of lateral medullary infarction caused by an embolic occlusion of the posterior inferior cerebellar artery the

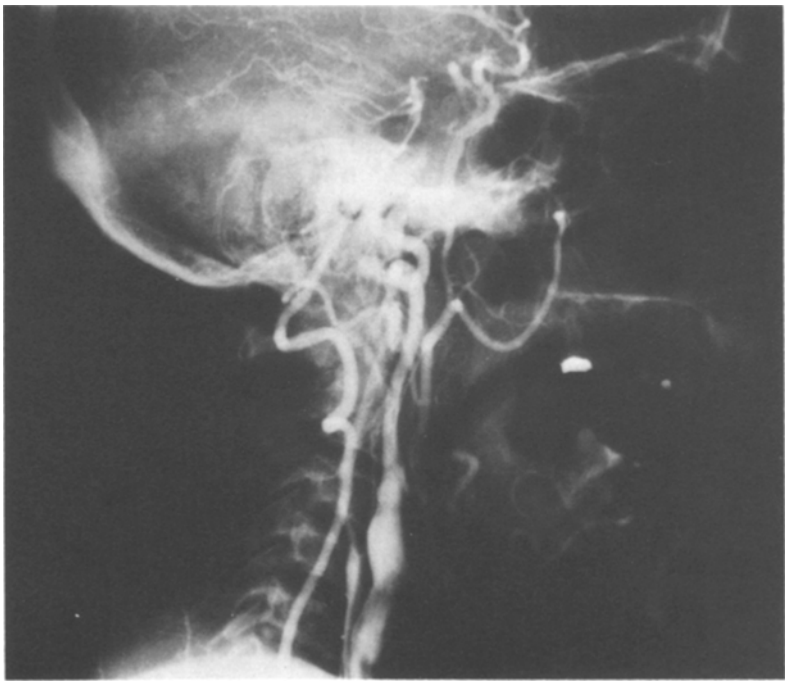


Fig. 2b

combination of cranial nerve V, IX, X-paresis, Horner's syndrome, ataxia ipsilateral and loss of pain and temperature sensation on the contralateral side is well known as Wallenberg's syndrome. The most common causes are occlusions of the A. cerebellaris inferior posterior, the vertebral artery [4, 6, 7], or lesions of the A. cerebellaris inferior anterior [17]. Our patient suffered from the typical symptom-combination of Wallenberg's syndrome. Since no occlusion of the inferior cerebellar arteries or the vertebral artery was demonstrable one has to suppose the lateral medullary infarction to be related to a circulatory abnormality.

Beside the external carotid obliteration our patient had a slight stenosis at the origin of the right vertebral artery while the remaining vertebral-basilar vessels had no atheromatous changes. Angiographic examination revealed a diversion of vertebral flow into the occipital artery. This vessel is the most important connection between the vertebral and the external carotid territory [2, 7, 10, 12–14]. From the radiological point of view there is no doubt, that the external carotid artery in this case was "stealing" intracranial blood via the vertebral artery. But could this finding possibly be related to the oblongata syndrome?

The collateral circulation in external carotid occlusion may take place in four different arterial anastomoses:

1. from the external carotid artery of the opposite side;
2. from the internal carotid artery via the ophthalmic artery;
3. from the ascendent and the deep cervical artery, and
4. from the vertebral artery of the same side via the occipital artery via muscular branches [1–2, 7–10, 12–14].

Of these four connections the second and the fourth are of special clinical interest, because they are extra-intracranial anastomoses, which may cause neurological symptoms in external carotid occlusion. Clinical deficits related to the internal carotid territory may be caused by external carotid obliteration because of interruption of important collaterals in internal carotid occlusive disease [7]. On the other side "steal" of blood from the vertebral artery can produce clinical signs directly. Barnett et al. [1] reported five patients with vertebrobasilar symptoms in external carotid occlusion; they described vertigo, drop attacks and transient bilateral visual loss. Three of the patients had stenoses of the subclavian, innominate or vertebral artery in addition to external carotid occlusion. Multiple vascular lesions are probably an important factor in this steal syndrome.

It is most likely that both the slight vertebral artery stenosis at its origin and the external carotid steal in our case caused the neurological deficits diminishing the blood flow to the dependent region. This supposition is supported by the high blood pressure in the external carotid artery measured during the operation and the reversal of blood flow in arteriography. Since the remaining branches of the external carotid artery showed no hypertrophy the occipital artery has to be responsible for the blood supply of the occluded vessel. There still remains the possibility of an embolic occlusion followed by fragmentation of the embolus with a negative result in the arteriography. But in vascular medullary syndromes an embolic aetiology is rather seldom [17], and our patient suffered from a multilocular atherosclerosis.

The appropriate treatment of extracranial arterial occlusive disease is—depending on the clinical findings and the condition of the other brain supporting vessels—the thromboendarterectomy [1, 4, 7]. Barnett et al. [1] found improvement of cerebral circulation in three of five surgically treated patients with freedom from symptoms. Our patient remained completely free of vertebrobasilar complaints such as vertigo attacks, and the deficits related to his lateral medullary infarction clearly diminished. The improvement of cerebral circulation was further confirmed by angiography. These findings support the theory of a haemodynamic aetiology in our patient.

This case may illustrate the significance of a complete angiographic study of all brain supporting vessels in cerebral infarction. An occlusion of the external carotid artery may cause vertebrobasilar symptoms depending on haemodynamic changes—one has to consider the possibility of external carotid blood steal at the expense of the brain. In these cases surgical treatment by thromboendarterectomy should be considered.

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