Introduction

Cervicocranial arterial dissection is the cause of 10–20 % of ischaemic strokes in young adults [1]. The pathogenesis of dissections is not clear in most cases, although extrinsic factors (e.g. trauma and hypertension) and intrinsic factors, such as primary disease of the arterial wall, have been suggested [2–4].

Case report

A 39-year-old previously healthy man was referred with suspected left internal carotid artery (ICA) occlusion. He reported two episodes of right periorbital tingling, numbness and paraesthesia in the right arm together with a feeling of weakness and difficulties in finding words most probably due to transient aphasia. During the first episode he also had left-sided headache, most marked in the parieto-occipital region. Each episode had lasted several minutes. There was no history of trauma and/or neck pain before or during these events, or of cardiovascular disease, previous stroke or other vascular risk factors. There were no other medical or lifestyle risk factors. Examination was normal. All 15 measurements recorded on eight consecutive days showed a blood pressure \( K_{130} \text{ mmHg} \) systolic and \( K_{80} \text{ mmHg} \) diastolic.

Colour-coded duplex sonography suggested a pseudo-occlusion of the left ICA, and cerebral angiography demonstrated dissection of the left ICA and both vertebral arteries. Angiography 6 months later was completely normal. This underlines the importance of four vessel angiography in young patients with dissections of cervical arteries.

Abstract

A 39-year-old healthy man had several transient ischaemic attacks suggesting left internal carotid artery (ICA) occlusion. There were no vascular risk factors and no preceding trauma. Colour-coded duplex sonography suggested a pseudo-occlusion of the left ICA, and cerebral angiography demonstrated dissection of the left ICA and both vertebral arteries. Angiography 6 months later was completely normal. This underlines the importance of four vessel angiography in young patients with dissections of cervical arteries.

Key words Dissection, internal carotid artery, vertebral artery
small string at the skull base (Fig. 1 A). Dissection of both vertebral arteries and a small pseudoaneurysm of the left were also detected (Fig. 1 B, C). There was no angiographic sign of fibromuscular dysplasia, and no abnormality of the intracranial arteries.

The patient was given dicumarol (international normalised ratio 2–3.5) and discharged without further ischaemic symptoms. At follow-up 3 months later, no further ischaemic events had occurred. The flow in the left ICA had markedly improved on sonography showing only a slightly abnormal profile. Six months after the first episode the patient underwent further intra-arterial pan-angiography which demonstrated all previously dissected vessel to be normal; the pseudoaneurysm of the left vertebral artery was no longer visible (Fig. 1 D–F).

Treatment was stopped, and the patient was scheduled for further follow-up sonography.

Discussion

We report a patient with multiple cervical artery dissections without a cardiovascular risk profile or a history of other possible precipitating factors. Thus, the dissections should be designated spontaneous. We can assume that the dissections occurred simultaneously. Multiple spontaneous dissections have been described in 28–66 % of reported cases [5–7] but, interestingly, although vascular risk factors are said to be present, a common cause of spontaneous multiple-vessel dissection has not usually be discussed.

It appears questionable whether simultaneous dissection of three cervical vessels should be designated “spontaneous”. There may have been an underlying