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Short Report

A Case of Reversed Robin Hood Syndrome: A Prognostic Indicator for an Urgent Therapy

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ABSTRACT

Introduction: In acute stroke, the diagnosis of reversed Robin Hood syndrome (RRHS) by transcranial Doppler (TCD) helps to identify patients at high risk for neurological deterioration.
Report: A patient with left intracranial internal carotid artery (ICA) dissection and concomitant inadequate collateral circulation suffered from recurrent ipsilateral ischaemic symptoms, not prevented by the best medical treatment. TCD showed an RRHS. Stenting of ICA could restore an adequate flow with disappearance of the RRHS and prevention of further episodes.
Discussion: An invasive emergency treatment should be considered in those stroke patients in which TCD detects an inadequate haemodynamic status.

Introduction

Neurological deterioration or symptom recurrence can occur in 20–40% of patients with acute stroke. One of the possible mechanisms is a haemodynamic impairment that can render penumbral tissue particularly vulnerable to alterations in cerebral perfusion. The insufficiency of cerebral autoregulatory mechanisms resulting from stroke and the presence of an inadequate collateral circulation are the two major causes of altered haemodynamics. Several studies demonstrated the importance of transcranial Doppler (TCD) in detecting in real time the haemodynamic status of acute ischaemic stroke patients.[1] In this field, Alexandrov et al. recently described, as reversed Robin Hood syndrome (RRHS), a condition characterised by a paradoxical decrease in middle cerebral artery (MCA) flow velocities during hypercapnia. This condition is due to an exhausted vasomotor reactivity, distal to a proximal arterial occlusion, associated with a flow steal towards normal vessels with least resistance.[2] TCD monitoring in the acute phase of ischaemic stroke can help to identify patients at high risk for stroke progression.

Report

A 45-year-old man was hospitalised for severe headache and high blood pressure (240/140 mmHg). Physicians diagnosed multiple subacute ischaemic lesions in left hemisphere due to the dissection of the homolateral internal carotid artery (ICA). He was discharged with anticoagulant and anti-hypertensive treatment. After 2 weeks of well-being, he came to our department complaining of left retrobulbar pain and transient paraesthesias in the right arm and referring an episode of transient motor aphasia 2 days before. Neurological examination showed left miosis, restriction of the left eyelid and motor awkwardness in the right arm. Angiographic computerised tomography documented left ICA subocclusion in the intrapetrous segment, confirming an extensive wall dissection; foetal origin of both posterior cerebral arteries (PCAs) and hypoplasia of A1 segment of left anterior cerebral artery (ACA). Brain magnetic resonance imaging (MRI) showed new acute ischaemic lesions in the left hemisphere. Extracranial and intracranial colour-Doppler ultrasound revealed reduced flow velocities in left ICA, MCA, ACA and PCA. TCD monitoring showed during voluntary breath holding transient decrease of the mean flow velocities in the affected side and normal response in the contralateral MCA, as described in RRHS (Fig. 1(B)). During hospitalisation, despite the best medical treatment, he complained, while taking the orthostatic position, of transient episodes of motor aphasia, right brachio-crural hypoesthesia and right emianopia. A second brain MRI documented new acute ischaemic lesions in the previously affected territory. Considering the neurological deterioration and the haemodynamic impairment, we decided on a percutaneous transluminal angioplasty with stenting (PTAS) of the intracranial ICA. One week before the procedure, the patient replaced anticoagulants with double anti-platelet therapy (acetylsalicylic acid

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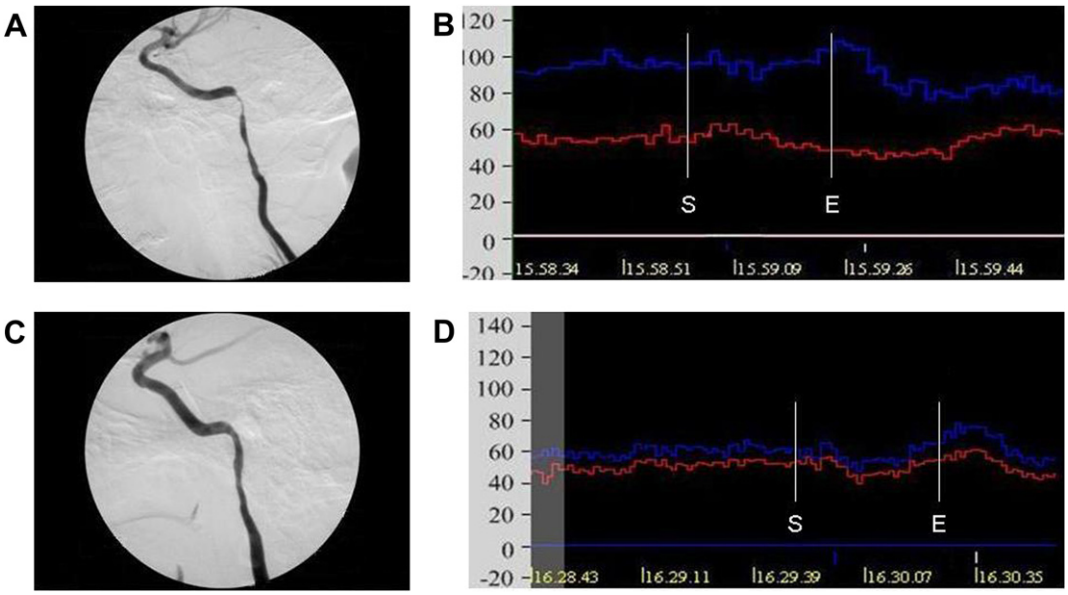

Figure 1. Stenting of ICA determines disappearance of the steal phenomenon with improvement of cerebral haemodynamics. A. Angiogram of left ICA before stenting, showing intracranial dissection. B. TCD monitoring of vascular reactivity to hypercapnia before left ICA stenting, showing asymmetry of MCA flow velocity with normal response in right (blue-coded) MCA and paradoxical response in left (red-coded) MCA as described in RRHS. S = start of breath holding. E = end of breath holding. C. Angiogram of left ICA after stenting, showing restoring of the vessel lumen. D. TCD monitoring after ICA stenting, showing restoration of symmetry of MCAs flow velocity with normal response to hypercapnia in both MCA. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

(ASA) 100 mg plus Clopidogrel 75 mg). A neurodedicated stent (Pharos Vitesse, Micrus 4.5 × 26 mm) was positioned in the prepetrous and intrapetrous segments of the left ICA, restoring an adequate flow in the distal hypoperfused territory (Fig. 1(A) and (C)). TCD after the procedure showed an improvement of cerebral haemodynamics, with disappearance of the steal phenomenon (Fig. 1(D)). Double anti-platelet therapy was then maintained for 3 months. At 24 months, the patient was still free from new neurological symptoms.

Discussion

The outcome of patients with acute ischaemic stroke due to carotid occlusion is linked to the adequacy of collateral flow and cerebral autoregulatory mechanisms.[1] In patients with RRHS, blood vessels distal to the occlusion are not able to further dilate in response to chemical or pressure stimuli, demonstrating an exhausted vasodilatory response. The consequence is a flow steal from ischaemic to non-affected territories (with a normal arteriolar vasodilation) and recurrent ipsilateral strokes of haemodynamic origin.[3] In our patient, RRHS co-existed with the absence of a collateral circulation through ipsilateral PCA and ACA. Therefore, transient reduction of arterial blood pressure (during the orthostatic position) determined the recurrence of hypoperfusion episodes leading to new ischaemic lesions. Even in the presence of adequate medical treatment, we could not prevent symptoms until the endovascular approach was applied, with regression of RRHS.

As regards the positioning of an intracranial stent, despite the recent results of the SAMMPRIS (Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis) trial, which showed the superiority of medical treatment versus PTAS in patients with atherosclerotic intracranial arterial stenosis,[4] there are a few or no studies on endovascular treatment of dissective intracranial stenosis.[5] Our case confirms the efficacy and safety of PTAS in the treatment of carotid intracranial dissection in those patients with haemodynamic impairment where medical treatment (anticoagulation and blood pressure management) does not appear satisfactory.

TCD allows to demonstrate an altered haemodynamics and to select patients with acute ischaemic stroke at high risk for neurological deterioration, for whom an invasive emergency treatment should be considered.

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Conflict of Interest Statement

The authors report no conflict of interest.

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