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Cerebral Hemodynamic Failure Presenting as Limb-Shaking Transient Ischemic Attacks

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Key Words
Limb-shaking transient ischemic attacks · Ultrasound examination · Hemodynamic failure

Abstract
Limb-shaking transient ischemic attacks (TIA) may occur in patients with insufficient brain perfusion due to an underlying occlusive disease. We present the case of a 64-year-old patient who suffered from repetitive TIA presenting with shaking movements of the right-sided extremities and accompanying speech arrest. Symptoms are documented in the online supplementary video (www.karger.com/doi/10.1159/000327683). These episodes were frequently triggered in orthostatic situations. The diagnosis of limb-shaking TIA was established. The diagnostic workup revealed pseudo-occlusion of the left internal carotid artery, a poor intracranial collateral status and, as a consequence, an exhausted vasomotor reserve capacity. At ultrasound examination, symptoms were provoked by a change of the patient's position from supine to sitting. During evolvement of symptoms, a dramatic decrease of flow velocities in the left middle cerebral artery was observed. This case thus documents the magnitude and dynamics of perfusion failure in a rare manifestation of cerebral ischemic disease.

Introduction

Hemodynamic failure is a relatively rare cause of transient ischemic attacks (TIA). In these cases, TIA may be triggered by a change from the supine into the upright position in patients with compromised hemispheric perfusion and exhausted cerebral vasoreactivity following severe obstruction of the internal carotid artery (ICA) [1]. Typically, TIA may present with repetitive shaking movements of the effected limbs, which is referred to as limb-shaking TIA.

In this report, we describe the case of a patient with highly frequent TIA as a consequence of severely compromised left hemispheric perfusion.

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Case Report

A 64-year-old female patient was referred to our hospital because of TIA. Three years before presentation, a routine assessment of the brain-supplying arteries had led to the diagnosis of left ICA occlusion. On admission, the patient reported transient episodes occurring up to 10 times per day. The episodes affected the right arm and leg and mainly consisted of involuntary non-rhythmic shaking movements, limb weakness and speech problems that she referred to as a 'mental block'. Sometimes the mental block was the leading symptom. The patient was responsive but appeared to be markedly slowed in her reactions. After 1–2 min, her clinical status normalized again. She reported that the episodes mainly occurred after standing up from a supine position or when walking. We observed these episodes on several occasions after the patient had been lying and was then asked to sit or stand up (online suppl. video; www.karger.com/doi/10.1159/000327683). An ultrasound examination revealed subtotal stenosis of the left extracranial ICA with extensive lumen narrowing in the whole extracranial segment. The B-mode finding was suggestive of a past arterial dissection. Doppler analysis showed pendular flow, indicating vessel patency with absent hemodynamic function. Transtemporal insonation demonstrated postocclusive flow in the left middle cerebral artery (MCA). Additionally, hyperventilation apnea testing indicated an exhausted cerebrovascular reserve (**fig. 1**). Moving the patient from a supine to a sitting position further resulted in a marked decline of MCA flow velocities. At the same time, the patient experienced typical limb-shaking symptoms, which resolved after 2 min. Flow velocities also returned to pretest values within 2 min (**fig. 2**). The A1 segment of the left anterior cerebral artery (ACA) was not detectable despite good insonation conditions, indicating poor collateral quality. High velocities were found in the right A1 and the left P2 segments, demonstrating collateral flow via leptomeningeal anastomoses. The results of the native and perfusion CT are shown in **figure 3**.

Repeated EEG tracing during the course of the patient's hospital stay revealed discrete focal theta slowing over the left hemisphere without epileptiform phenomena. An antiepileptic treatment attempt with valproic acid was stopped after she showed no improvement of her symptoms.

The diagnosis of limb-shaking TIA due to compromised left hemispheric perfusion was established. Since the patient had experienced the symptoms for several years on a much less frequent basis, acute heart failure was thought to be the cause of the cerebral hemodynamic deterioration. Best medical treatment of her chronic heart disease resulted in an only moderate improvement of her left ventricular ejection fraction from 23 to 35% (measured by echocardiography), which, however, did not result in a lasting neurological improvement. As a consequence, the patient was confined to bed and revascularization of the left ICA territory was discussed. Due to the small caliber of the peripheral arterial branches, extra-intracranial bypass surgery was thought to result in a primarily insufficient bypass and was therefore discarded. Finally, angioplasty and stenting of the proximal ICA were performed as a treatment attempt. The stent could be placed without complications and angioplasty was performed within the whole cervical segment of the ICA. However, although the stent was sufficiently expandable, there was only a moderate improvement of the distal lumen narrowing (**fig. 4**). Furthermore, it was now clear that the distal ICA was nearly occluded and, therefore, no possibility for a further recanalization could be found. Ultrasound controls up to 3 weeks later showed unchanged hemodynamics with persisting pendular flow patterns within the ICA.

At that point, the patient rejected further invasive treatment and was therefore discharged to rehabilitation in an only slightly improved clinical status.

Discussion

We report a patient with severe left cerebral hemodynamic insufficiency as a consequence of subtotal ICA stenosis and poor collateral function. Pendular flow within the ICA indicated a 'functional closure' of the vessel. Intracranial Doppler analysis demonstrated insufficiency of the main collateral pathways, a finding that has previously been associated with a state of maximal hemodynamic compromise [2]. The severity of our findings is further underlined by the exhausted vasomotor reactivity and by the documentation of a severely pathological reduction of MCA flow velocities in orthostatic situations (fig. 2). As a consequence, limb-shaking TIA was repeatedly triggered by a

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change of position from supine to sitting or standing. According to this observation, limb-shaking TIA may be considered as a vascular paroxysmal dyskinesia [3], triggered by an orthostatically induced decrease of cerebral blood flow in already critically hypoperfused arterial territories. Bogousslavsky and Regli [4] found focal limb-shaking symptoms in 12% of 51 patients with infarcts in watershed cerebral territories.

Treatment of low-flow TIA focuses on measures to improve cerebral blood flow. In our patient, severe cardiac insufficiency may have contributed to the exacerbation of TIA frequency, because maximally dilated arteries are unable to increase their capacity in stress situations such as decreased cardiac output. However, medical treatment only moderately improved cardiac output and did not result in a neurological improvement. Extra-intracranial bypass surgery as a treatment option in this high-risk patient group has been shown to improve symptoms in a series of 5 patients who underwent the procedure [5]. Nevertheless, absence of satisfactory arterial branches made us refrain from this approach. Unfortunately, carotid stenting did not result in significant cerebral reperfusion due to the irreversible narrowing of the distal ICA in the 3-week follow-up period.

Epilepsy as a potential differential diagnosis was ruled out due to the presenting symptoms and the circumstances of their manifestation, the complementary imaging and EEG findings, and the missing response to antiepileptic treatment. In a previous report of 4 limb-shaking TIA cases [6], focal delta slowing in EEG, without noteworthy structural lesions on imaging, was found. The authors discuss a structural/EEG mismatch attributable to cerebral hypoperfusion. In our case, left hemispheric theta slowing may have been a consequence of the documented white matter lesions. Retrospectively, in this context, it would have been an interesting task to compare the hemodynamic changes in the MCA with EEG abnormalities during change of position from supine to sitting. Future investigations on this disease should take this aspect into consideration.

Conclusion

Limb-shaking TIA is a rare but typical manifestation of cerebral vascular disease. It may occur in patients with occlusive disease of major brain-supplying arteries and poor collateral conditions. Transcranial Doppler is well suited to document hemodynamic instability from exhausted vasomotor reactivity and orthostatic decrease of MCA blood supply.

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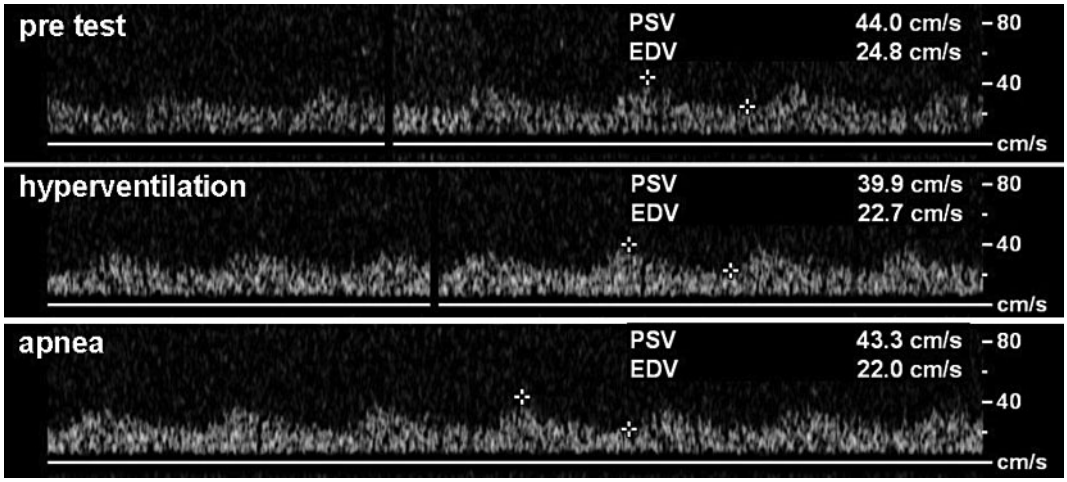

Fig. 1. Hyperventilation for 30 s followed by apnea for 30 s showed no relevant flow velocity changes in the M1 segment, reflecting exhausted reserve capacity.

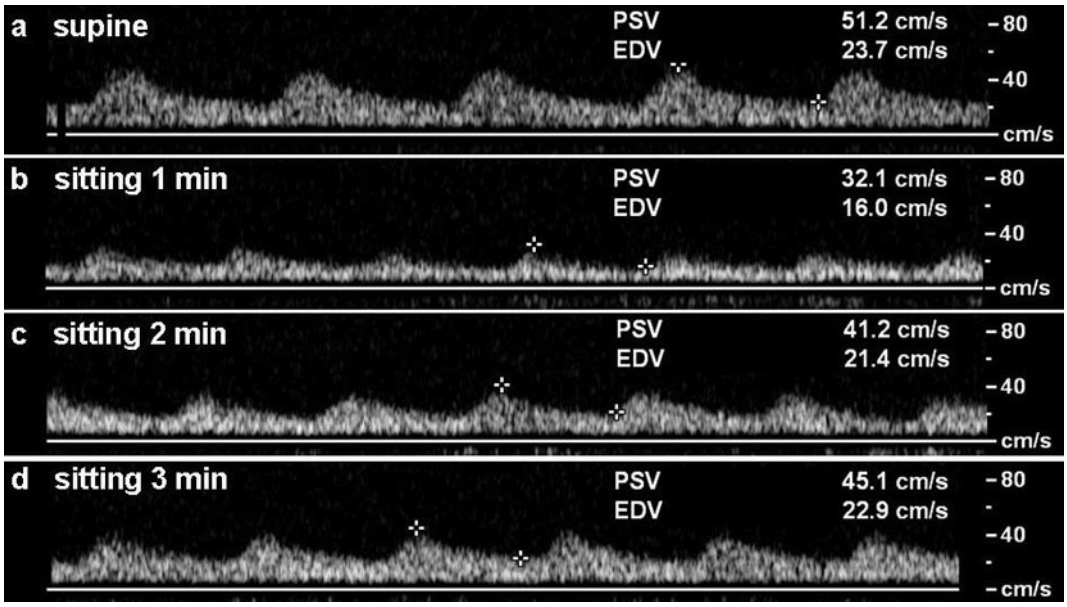

Fig. 2. Doppler examination of the left MCA in a supine (**a**) and a sitting (**b–d**) position. Within 30 s after sitting up, the patient developed symptoms similar to those seen in the supplementary video lasting 1 min. Simultaneous Doppler showed deceleration of systolic and diastolic velocities (**b**), which slowly returned to pretest values within 2 min (**c**, **d**).

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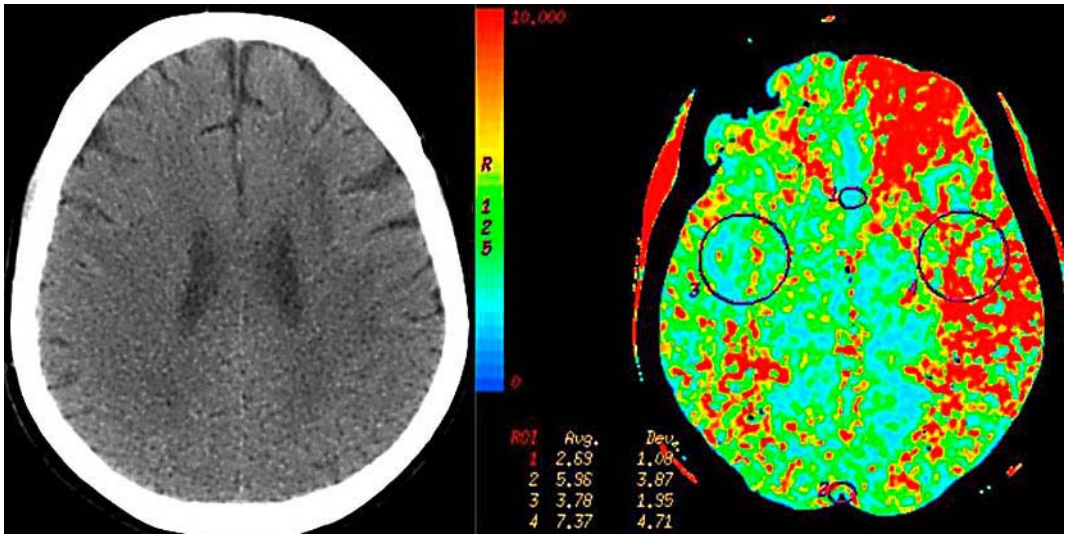

Fig. 3. Cranial CT: left-sided white matter disease. Perfusion CT: mean transit time was markedly prolonged within the left MCA and ACA territories (7.4 vs. 3.8 s on the right territories).

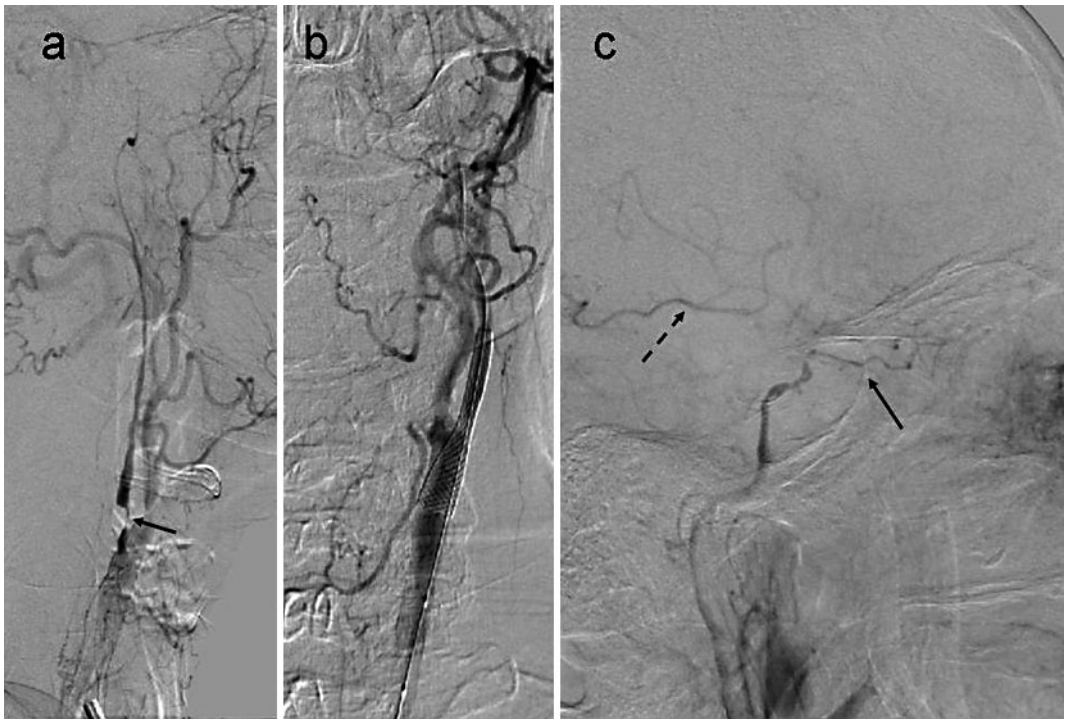

Fig. 4. **a** Angiography showing subtotal stenosis of the left ICA (arrow) and poststenotic collapse of the patent ICA. **b** Stent placement in the proximal ICA and PTA with acceptable results.
c Postinterventionally persisting lumen narrowing with contrast filling of the ophthalmic artery (arrow) and 1 small MCA branch (dotted arrow).

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References

1 Baumgartner RW, Baumgartner I: Vasomotor reactivity is exhausted in transient ischaemic attacks with limb shaking. J Neurol Neurosurg Psychiatry 1998;65:561–564.

2 Smith HA, Thompson-Dobkin J, Yonas H, Flint E: Correlation of xenon-enhanced computed tomography-defined cerebral blood flow reactivity and collateral flow patterns. Stroke 1994;25:1784–1787.

3 Ali S, Khan MA, Khealani B: Limb-shaking transient ischemic attacks: case report and review of literature. BMC Neurol 2006;6:5.

4 Bogousslavsky J, Regli F: Unilateral watershed cerebral infarcts. Neurology 1986;36:373–377.

5 Baquis GD, Pessin MS, Scott RM: Limb shaking – a carotid TIA. Stroke 1985;16:444–448.

6 Bearden S, Uthman B: Cerebral hemodynamic compromise associated with limb shaking TIA and focal EEG slowing. Am J Electroneurodiagnostic Technol 2009;49:225–243.