bral digital subtraction angiography revealed a bilateral occlusion of the internal carotid arteries and the right vertebral artery, with the retrograde right ophthalmic artery as the only vessel supplying the brain. Further MRI sequences showed, besides old left hemispheric infarcts, new ischemic lesions in the right centrum semiovale, pointing to a recent watershed infarct (fig. 1b). ^1H -MRS revealed a lactate peak in these ischemic areas (fig. 1c, d). A 550 MBq $^{99\text{m}}\text{Tc}$ -HMPAO brain perfusion scintigraphy showed hypoperfusion in both middle and anterior cerebral artery territories with severely impaired functional cerebrovascular reserve capacity.

Because of the hemodynamic cause of these infarcts, early treatment consisted of antiplatelet agents and hypotension prevention. We decided to support the oxygen supply of the brain with HBOT in order to bridge the time until neurosurgical treatment with an extracranial to intracranial bypass.

Hyperbaric oxygen treatment units were administered once daily for 8 days. Each treatment unit consisted of 90 min surplus pressure with 100% oxygen at 2.0 bar in a hyperbaric chamber (Sayers-Hebold, Germany). Pressure and duration of each HBOT session were chosen according to human and experimental animal data in cerebral ischemia [1–3, 6].

After 8 hyperbaric oxygen treatment units and 11 days, an MRI and ^1H -MRS detected neither new ischemic lesions nor lactate peaks in the same areas (fig. 1e, f) and the patient showed clinical improvement. The patient was then treated with an extracranial to intracranial bypass, which preserved the spectroscopic and clinical result.

Discussion

There is no consensus on the efficacy of HBOT in ischemic stroke [4, 5]. Here, the first MRI showed new ischemic lesions and, additionally, ^1H -MRS indicated hypoxic tissue with lactate due to severely impaired cerebral perfusion.

The absence of the lactate peak in the new ischemic areas after HBOT might be induced by HBOT rather than by spontaneous decline, since lactate has been reported to remain at constantly high levels for more than 1 month after ischemia [7] and the period between the first and second ^1H -MRS was only 11 days.

Furthermore, lactate has been described as a prognostic marker for a significantly poorer clinical outcome in ischemic stroke [8]. The number of HBOT sessions was set at 8, based on the time until neurosurgical treatment could be performed.

Our results suggest that HBOT increases brain tissue oxygenation in ischemic areas and may serve as a treatment option in severely hemodynamically compromised patients until definitive revascularization.

^1H -MRS may be useful for monitoring HBOT efficacy, although more experimental and clinical data are necessary to confirm our assumption.

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Moyamoya Syndrome Associated with Optic Nerve Coloboma and Mental Retardation

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Moyamoya is an intracranial arteriopathy of undetermined etiology that is characterized by the progressive obliteration of the major arteries of the anterior and, in some cases, the posterior cerebral circulation, and their replacement by a meshwork of small collateral vessels at the base of the brain. Moyamoya encompasses two distinct entities: (1) Moyamoya disease which is mostly seen among children and young adults with no other pathological conditions; (2) Moyamoya syndrome which is mostly seen in western countries, and its arterial abnormalities are considered as epiphenomena in response to another vascular process and have been associated with a series of conditions [1].

Mental retardation has been described in several patients with Down syndrome associated with Moyamoya [2]. In addition, ocular malformations, such as morning glory disk anomaly, optic disc and infrapapillary choroidal coloboma, have been reported in a small number of Moyamoya patients [3, 4].

Fig. 1. T₂-weighted MRI in the axial (a) and sagittal (b) plains, and a turbo-FLAIR-weighted MRI in the axial plain (c) demonstrate a coloboma of the left optic nerve (white arrow) in a patient with typical Moyamoya arteriopathy affecting the anterior and posterior cerebral circulation, as visualized on digital subtraction angiography (d) and magnetic resonance tomography (a, c; black arrows).

We present the case of a now 42-year-old woman with a history of unilateral amblyopia, due to an optic nerve coloboma (fig. 1a–c), and mental retardation since infancy, who developed a severe gait disorder over the last decade due to multiple ischemic cerebral lesions. Diagnostic evaluation established, by means of digital subtraction angiography, a diagnosis of Moyamoya arteriopathy affecting both the anterior and posterior circulation as the cause of relapsing brain ischemia (fig. 1d).

Optic nerve coloboma has been associated with mental retardation in a series of distinct syndromes [5–7]. To our knowledge, this is the first report of congenital optic nerve coloboma associated with mental retardation that is also accompanied by a progressive ischemic encephalopathy due to Moyamoya arteriopathy. It describes one more association of Moyamoya syndrome with other diseases.

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Rapidly Progressive Cerebrovascular Stenosis and Recurrent Strokes Followed by Improvement in HIV Vasculopathy

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Human immunodeficiency virus (HIV) infection is associated with an increased incidence of stroke [1–3]. Infected children and rarely adults have been reported to develop cerebral vasculopathy with aneurysms [4, 5]. We report here an AIDS patient with no other obvious risk factor for strokes, with rapidly progressive vascular stenosis and recurrent cerebral infarcts, who showed vascular and neurological improvement on highly active anti-retroviral therapy (HAART).

Case Report

A 33-year-old man with a HIV+ status for the past 1.5 years, noncompliant with medications, was admitted with confusion for 1 week. He was alert, but did not know the date or time. He understood simple 1-step commands, but had difficulty in word finding and he had a right central facial weakness. A cranial MRI scan revealed a left thalamic infarct (fig. 1a). Antinuclear, antineutrophil cytoplasmic, rheumatoid factor, anti-DNA, anti-Jo, and hepatitis B and C antibodies were absent. The erythrocyte sedimentation rate was normal and C-reactive protein was mild-