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Normalization of Brain Tissue Lactate after Hyperbaric Oxygen Therapy in a Progressive Stroke Patient

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Introduction

In contrast to experimental results [1, 2], clinical trials with hyperbaric oxygen therapy (HBOT) have failed to show benefit in acute ischemic stroke [3] and its application has been discussed controversially [4, 5]. We used proton magnetic resonance spectroscopic imaging (^{1}H-MRS) to detect the effect of HBOT in a progressive ischemic stroke patient.

Case Report

A 56-year-old right-handed woman was admitted because of worsening of a right-sided hemiparesis and dysphasia for 20 days. Vascular risk factors were smoking (40 pack-years) and a family history of cerebrovascular diseases. Neurological examination showed decreased spontaneous movement, expressive dysphasia, a right-sided hemiparesis and a left-sided arm paresis (MRC scale: 3+/5). Duplex sonography (Aplio XV; Toshiba), magnetic resonance angiography (fig. 1a; 3 Tesla MRT; Trio; Siemens) and cere-

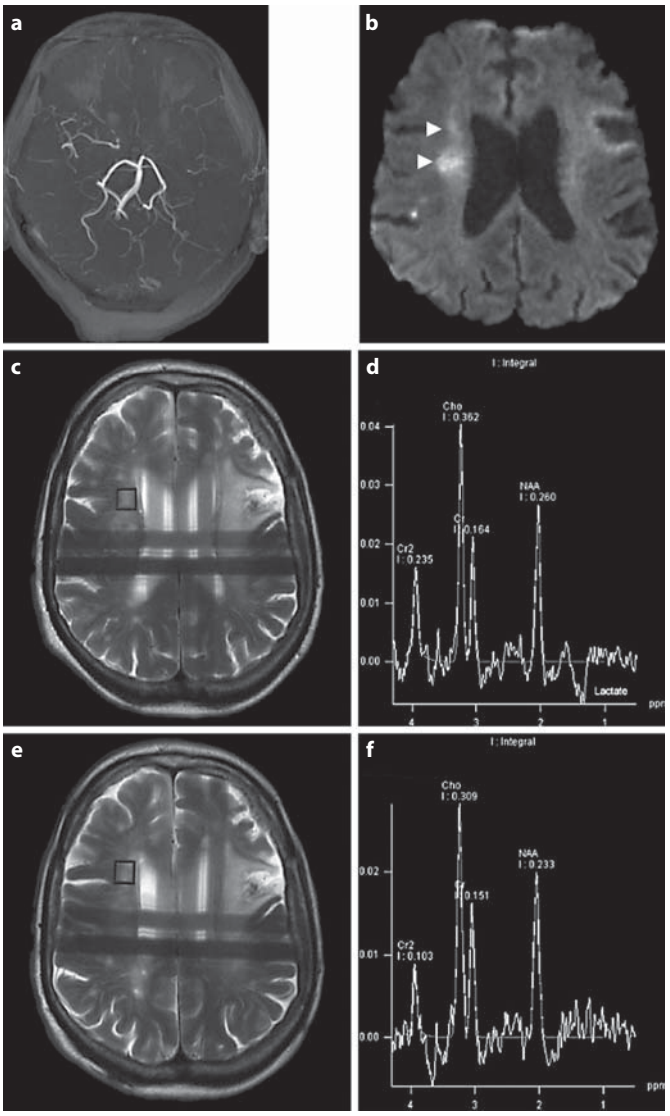

Fig. 1. a Axial MR time-of-flight angiography indicates bilateral occlusion of the internal carotid arteries (3D gradient echo sequence, TR = 22 ms, TE = 3.7 ms, FOV = 105 × 200 mm, image matrix = 202 × 384, 3 slabs, 40 image sections, section thickness 0.65 mm). **b** Axial diffusion weighted-image brain MRI shows new ischemia in the centrum semiovale of the right hemisphere (arrowheads) pointing to an acute hemodynamic watershed infarct (Echo planar imaging sequence, TR = 3,200 ms, TE = 92 ms, image matrix = 192 × 192, FOV = 230 mm, slice thickness = 5 mm, gap = 1.5 mm, 20 slices, b-value = 0; 500; 1,000 s/mm^2 in 3 orthogonal directions combined to a diffusion-weighted imaging trace image). **c** Axial T_2-weighted sequence shows voxel for ^{1}H-MRS planning prior to HBOT (Turbo spin echo sequence, TR = 5,000 ms, TE = 100 ms, section thickness = 5 mm, gap 1.5 mm, FOV = 240 mm, image matrix = 384 × 512, 2 averages, images acquired in 3 orthogonal directions). **d** Corresponding ^{1}H-MRS reveals a negative double peak at 1.3 parts per million (ppm) attributable to lactate in the new ischemic area prior to HBOT. Other peaks represent N-acetyl-aspartate (NAA), choline (Cho) and creatine (Cr, Cr2) (Spectroscopic imaging sequence, TR = 1,700 ms, TE = 135 ms, FOV = 160 mm, image matrix = 16 × 16, section thickness = 15 mm, voxel size = 10 × 10 × 15 mm, 3 averages). **e** Axial T_2-weighted sequence shows voxel for ^{1}H-MRS planning after 8 HBOT units. **f** After 8 HBOT units corresponding ^{1}H-MRS detects no pathologic lactate peak in the same area.

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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). ^{1}H-MRS revealed a lactate peak in these ischemic areas (fig. 1c, d). A 550 MBq ^{99m}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 ^{1}H-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, ^{1}H-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 ^{1}H-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.

^{1}H-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 infrapapilar choroidal coloboma, have been reported in a small number of Moyamoya patients [3, 4].