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Carotid Endarterectomy in a Patient with Persistent Proatlantal ArteryJulia Schoof^a, Martin Skalej^c, Zuhir Halloul^b,
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On admission, a 37-year-old man presented with an acute right-sided sensorimotor hemiparesis and nonfluent aphasia (NIH Stroke Scale score 11) persisting for more than 12 h. As cerebrovascular risk factors, the patient exhibited untreated arterial hypertension and chronic nicotine abuse (42 pack-years). Cranial computer tomography revealed a left-sided posterior border zone infarction. An electrocardiogram on admission, subsequent ECG monitoring and transthoracic echocardiography indicated no significant pathological findings. Extracranial color-coded duplex sonography revealed an occlusion of the left internal carotid artery (ICA) directly after the bifurcation. About 1.5 cm more distally, a high-frequency flow signal in projection on the lumen of the left ICA (fig. 1) was detected. Transcranial duplex sonography showed a diminished blood flow velocity of the left middle and anterior cerebral arteries. The left vertebral artery was hypoplastic.

Digital subtraction angiography confirmed the extracranial occlusion of the left ICA. Furthermore, it revealed a persistent proatlantal intersegmental artery originating from the V₃ segment of the vertebral artery and discharging into the ICA distally from the carotid occlusion (fig. 2).

In addition, the patient presented a fetal blood supply type (with hypoplastic P₁ segments), i.e. the posterior arteries were supplied by the intracranial distal internal artery via the posterior communicating arteries. An additional hypoplasia of the right A₁ segment made an anterior cross-flow impossible and led to the critical situation that the entire left hemisphere was supplied only by the persistent intersegmental proatlantal artery.

Fluctuation and slight deterioration of the patient's neurological symptoms despite hypertensive systolic blood pressure values between 160 and 200 mm Hg and additional small-sized infarcts of terminal vessels demarcated in a follow-up cranial computer tomography emphasized the critical hemodynamic situation. A revascularization procedure was seen as indicated. The patient underwent thrombendarterectomy (TEA) of the occluded left ICA 6 days after the onset of stroke symptoms. Afterwards, the neurological deficits did not improve in general but fluctuations were no longer observed and the patient could be transferred to a

Fig. 1. Color-coded duplex sonography showing occlusion of the extracranial left ICA and about 1.5 cm distally an intraluminal high-frequency flow signal.

Fig. 2. Digital subtraction angiography revealing a persistent proatlantal intersegmental artery type I, originating out of the V₃ segment of the vertebral artery discharging into the ICA more distally from the carotid occlusion.

rehabilitation center 7 days after TEA (NIH Stroke Scale score 11, modified Rankin Scale 4). Three months later, a mild right-sided hemiparesis was the only clinical residuum (NIH Stroke Scale score 1, modified Rankin Scale 2).

During embryonic development, 5 segmental arteries (A. trigeminalis, A. otica, A. hypoglossica, A. intersegmentalis proatlantis and A. intersegmentalis cervicalis) interconnect the anterior (carotid) and posterior (basilar) circulation systems on the brainstem and medullospinal level. These segmental arteries are named after their accompanying cranial nerves and usually involute with the development of the posterior communicating arteries (between days 30 and 40 of fetal life). A persistent intersegmental proatlantal artery is extremely rare, and only a handful of cases are reported in the literature [1, 2]. With regard to their origin, the proatlantal arteries can be classified into type I and type II [3]. Type I originates from the ICA close to the bifurcation, usually at the level C₂–C₃ [4]. Suboccipitally, it takes a horizontal course parallel to that of the vertebral artery with the noteworthy difference that it does not pass through any foramen transversarium of the vertebral bodies. Type II arises from the external carotid artery. Both type I and type II enter the skull through the foramen magnum. The persistence of embryonic carotid-basilar anastomoses leads to an altered dynamic blood flow at their origin which favors the formation of atherosclerotic plaque [4]. This may explain the occlusion of the left ICA in our patient, whereas the other cerebrovasculature showed no or only very mild atherosclerotic changes.

The persistence of embryonic arteries is not only associated with an altered blood flow between the anterior and posterior cranial circulation systems, but also leads to a suppression of the normal development of the cerebrovasculature. This explains that an ipsilateral or bilateral hypoplasia of the vertebral artery is often observed in patients with a persistent proatlantal artery [4, 5].

In extracranial ICA occlusion, the intracranial collateral blood supply depends on the integrity of the circle of Willis. In our case, a hypoplastic A₁ segment of the anterior cerebral artery prevented an anterior cross-flow from the right to the left hemisphere, and the additional hypoplasia of both P₁ segments of the posterior cerebral arteries furthermore hindered a posterior cross-flow to the anterior circulation. This insufficient expression of the circle of Willis together with the persistence of the proatlantal artery reflect the altered embryonic vasculogenesis in our patient. Usually, however, the posterior communicating arteries and not the P₁ segments are hypoplastic, and a persistent intersegmental proatlantal artery normally serves as an anastomosis from the anterior to the posterior circulation system. In our case however, the occlusion of the left ICA led to a change of the vascular pressure conditions resulting in a modification of blood flow direction: now the proatlantal artery transferred blood from the posterior to the anterior circulation system. As a result, the blood supply of the left hemisphere entirely depended on the persistent intersegmental proatlantal artery, which resulted in a critical hemodynamic situation and led to the decision to perform a TEA of an occluded ICA.

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Clinicoradiological Characteristics of Cerebral Air Embolism

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Since the first description of a patient with cerebral air embolism (CAE), seen on brain computed tomography (CT), due to an esophagoatrial fistula [1], dozens of similar patients have been described [2–7]. However, the clinical and radiological characteristics of CAE, including infarct patterns on diffusion-weighted image (DWI) [4, 5], have rarely been reported. Here, we describe the clinical and radiological features of 4 patients with CAE.

Subjects

Between August 2002 and December 2005, 4 patients developed CAE at the Asan Medical Center. Initial neurological evaluations were performed on the first day of symptom onset in all 4 patients. Magnetic resonance imaging (MRI) evaluation was performed using a 1.5-tesla MRI unit.

Case Summaries

Patient 1. A 54-year-old woman with a history of pulmonary tuberculosis developed sudden serious respiratory difficulty following recurrent massive hemoptysis. Percutaneous transcatheter embolization of the right pulmonary artery was immediately performed to control massive bleeding. During subsequent artificial ventilation, quadriplegia was noticed by the duty physician, and she was promptly referred to the neurological department. She was alert and cooperative but quadriplegic on initial neurological examination. There was no limitation or nystagmus in eye movements. Facial expression was preserved, but detailed cranial nerve evaluation and sensory evaluation were not possible due to endotracheal intubation. Brain CT was performed 38 h and DWI 46 h after embolization (fig. 1A). After comprehensive rehabilitation treatment for 3 months, she remained