Cerebrovasc Dis 2006;21:208–210
DOI: 10.1159/000090793

Dissection of the Common and External Carotid Artery

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Case Report

A 37-year-old man was admitted to the Department of Neurology with left-sided neck pain. The pain had started 6 days before admission after a foot kick to the left side of the patient's neck by his son while playing. He went to his general practitioner who performed an ultrasound examination of the brain-supplying arteries. Ultrasound examination was suspicious of a mural hematoma in the common carotid artery (CCA) and the patient was sent to our department. We performed a neurological examination, a CT angiography, a 3-tesla MRI scan of the neck arteries and an ultrasound of the brain-supplying arteries.

The neurological examination in our department was completely normal apart from a mild miosis on the left side. We immediately performed a cranial CT scan including CT angiography which demonstrated a small mural swelling of the carotid bulb continuing to the external carotid artery (ECA) (fig. 1). The mural swelling of the ECA led to a mild stenosis of the vessel. We diagnosed a cervical artery dissection and the patient was put on intravenous heparin. The ultrasound examination 3 days later revealed an echolucent structure at the same site of the vessel without any visible intimal lesion (fig. 2). The reduction of the vessel lumen of the ECA was less than 50%. A 3-tesla MRI scan of the neck was performed. The MRI scan demonstrated an intramural hematoma of the left carotid bulb and of the ECA (fig. 3, 4). The internal carotid artery was not affected and no intimal lesion could be found. During the hospitalization, the left-sided miosis disappeared completely. The neck pain was treated symptomatically. Since no intimal lesion could be found, the patient was put on aspirin 100 mg for antiplatelet aggregation.

Discussion

In the literature, there is a large amount of knowledge about dissections of brain-supplying extracranial cervical arteries, but nearly nothing is known about dissections of the CCA and/or ECA.

To our knowledge, only few studies on traumatic lesions of the ECA have been published. One report [1] describes a spontaneous fistula between the ECA and the jugular vein after repetitive hyper-

Fig. 1. Axial picture of the CT angiography of the neck arteries. The arrow marks the narrowing of the ECA lumen due to a vessel hematoma.

extension of the neck. Another report [2] describes 54 patients with blunt and sharp carotid artery injuries but without mentioning dissections. In 20% of the patients, the ECA was affected, but the trauma included stab wounds and gunshot wounds. Campbell et al. [3] describe a case of ECA pseudoaneurysm from hyoid bone fracture. In 1994, Minion et al. [4] report also a case of a pseudoaneurysm of the ECA and performed a review of the literature. They found only 6 other cases of ECA pseudoaneurysms. In a case report of Chen et al. [5], a spontaneous dissection of the CCA is described and a review of the literature from 1960 to 2003 revealed a further 8 patients with CCA dissection; no patient with a dissection of the ECA was found. Up to now, there has been no systematic investigation about the frequency and incidence of dissections of the CCA and ECA. In a comprehensive review by Brandt [6], dissections of the CCA and/or ECA are not mentioned, due to their rareness. Out of a series of 126 cervical artery dissections (78 patients with dissection of the internal carotid artery, 46 patients with dissection of the vertebral artery and 2 patients with a combined dissection) registered in our Department from 1992 to 2001 [7], there was no case

Fig. 2. Axial picture of a duplex ultrasound investigation of the left ECA (left-sided) and internal carotid artery (right-sided). The echolucent structure marked by an arrow shows the vessel hematoma in the ECA.

Fig. 3. Axial picture of a 3-tesla MRI of the neck arteries (T₂-weighted). The arrow marks the vessel hematoma in the carotid bulb on the left side.

Fig. 4. Axial picture of a 3-tesla MRI of the neck arteries (T₂-weighted). Mural hematoma in the left-sided ECA is marked by an arrow.

of CCA and/or ECA dissection. Thus, we estimate the prevalence of this type of dissection to be less than 1% of all cervical artery dissections. The reasons for the low frequency might be a different ultrastructure of the vessel wall in the CCA and ECA, but underdiagnosis due to a lack of attention regarding dissection of the ECA is also possible because dissection occurs without severe consequences/sequelae for the patients.

Conclusion

Dissections of the CCA and/or ECA are very rare. We assume that a systematic review of neuroimaging investigations would reveal a higher incidence of dissections at these sites.

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visual changes, confusion, and seizures. Magnetic resonance imaging (MRI) characteristically shows an increased T₂-weighted signal in the white matter of the parietal and occipital lobes, usually without restricted diffusion on diffusion-weighted imaging (DWI). The hallmark of the disease is clinical and MRI reversibility with aggressive treatment and/or withdrawal of the inciting agent. MRI brainstem abnormalities have also been described, but signs and symptoms specific to this location are more rare. We present a case of PRES with prominent bulbar manifestations that completely resolved with treatment.

Case Report

A 77-year-old man with a past medical history of hypertension, a greater than 100 pack-year history of tobacco smoking, and right internal carotid artery 90% stenosis was admitted to our institution after new onset dysarthria and inappropriate behavior. His neurological examination showed left hemineglect, confusion, and a blood pressure of 250/150 mm Hg. In addition, there was prominent dysarthria and dysphagia, with characteristic bulbar features of hypophonia and oropharyngeal weakness. There was no seizure activity, headache, or visual changes. Laboratory studies were unremarkable (sodium 139 mEq/l) except for a creatinine of 2.5 mg/dl (baseline). He underwent MRI (fig. 1c, d) which showed T₂-weighted hyperintensities in bilateral posterior cerebral white matter as well as in the pontine and ventral medullary white matter, which had not been observed on prior neuroimaging (fig. 1a, b). DWI showed no evidence of cytotoxic edema (data not shown). The patient was transferred to the cardiac care unit, where with aggressive blood pressure control, his confusion, hemiparesis and neglect promptly improved over days. The dysarthria and dysphagia also improved over the ensuing 2 weeks. Serial MRIs were performed that showed interval resolution of prior white matter abnormalities (fig. 1e, f); these findings correlated with his clinical improvement. On discharge, he no longer required a nasogastric tube for feeding, his speech was no longer dysarthric, and he had returned to his functional baseline.

Discussion

Hypertensive encephalopathy is a disease entity that has recently been well characterized in the literature. A broader category for this neurological syndrome has been coined: reversible posterior leukoencephalopathy or PRES [1]. Besides acute severe hypertension, several other conditions have been associated with PRES, including renal disease, immunosuppressive and cytotoxic drugs, collagen vascular disorders such as systemic lupus erythematosus, eclampsia, and hematological disorders, including thrombotic thrombocytopenic purpura or hemolytic uremic syndrome [2]. On MRI, the most commonly observed characteristics include hyperintensities on the T₂-weighted sequences in the parietal and occipital lobes, and isointense or hyperintense signals on apparent diffusion coefficient maps, suggesting a vasogenic edema pattern [3].

While the exact underlying mechanism of PRES is unknown, there are two prevailing theories regarding its pathogenesis and predilection for areas of the brain supplied by the posterior circulation. The most widely accepted theory proposes that the myogenic component of autoregulation in the posterior circulation, with its sparse sympathetic innervation [4], becomes overwhelmed by either elevated blood pressure or endothelial toxins, leading to a capillary leak phenomenon and vasodilatation, resulting in vasogenic

Cerebrovasc Dis 2006;21:210–212
DOI: 10.1159/000090794

Hypertensive Encephalopathy with Prominent Bulbar Presentation

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a well-described clinical entity, typically presenting with features of parieto-occipital cortical dysfunction and associated with vasogenic edema on neuroimaging. Patients often present with headaches,