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Internal Carotid Artery Dissection after Tonsillectomy in an Adult Woman

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Introduction

Cervical artery dissections (CAD) are a frequent cause of strokes in young people, which may be due to major traumas of the cervical arteries but also occur spontaneously or after minor traumas [1]. We report the first case of an internal carotid artery (ICA) dissection occurring 24 h after a tonsillectomy performed in an adult woman.

Case Report

A 50-year-old woman with a medical background of obesity (BMI = 33) and moderate untreated dyslipidemia had recurrent episodes of tonsillitis for several months. Three months after the last episode, a bilateral tonsillectomy was planned and performed under general anesthesia, without any complications. Twenty-four hours after waking up, the patient abruptly presented with right hemiparesis, aphasia and impairment of consciousness. The

Glasgow Coma Scale score was E3 V1 M6, the blood pressure 130/70 mm Hg, the pulse 80/min, and she was afebrile. The NIH Stroke Scale score was 25. Throat examination revealed a mild inflammation with swelling of the peritonsillar area. Blood examinations were normal. Brain MRI with diffusion-weighted images demonstrated a large cerebral infarction in the left middle cerebral artery territory, MR angiography showed an irregular stenosis of the left ICA extending from the bulb to the base of the skull. The MR axial scans looking for mural hematoma were non-interpretable due to the clouding of consciousness and agitation, but CT angiography showed widening of the external diameter of the ICA with a crescentic hypodensity around the enhanced narrowed artery (fig. 1) consistent with a mural hematoma. Ultrastructural examination of the skin did not show abnormalities of the elastic tissue, and Ehlers-Danlos syndrome, Marfan syndrome, polycystic renal syndrome, α 1-antitrypsin deficiency and fibromuscular dysplasia were excluded. The MR angiography performed 3 months after the onset of symptoms showed recanalization of the left ICA with a slight persistent narrowing of the artery (fig. 2).

Discussion

Although incompletely known, the main risk factors usually admitted for CAD are: genetic or inborn factors, migraine, trivial trauma (especially manipulative therapy of the neck), hyperhomocystinemia and recent infections [2]. They usually result from the association of a mechanical factor with an underlying disorder of the vessel wall. Minor traumas can cause arterial wall disruption by hyperextension and rotation of

Fig. 1. Findings in a 50-year-old woman with a left ICA dissection, 24 h after an adenotonsillectomy. CT angiography: widening of the external diameter of the ICA with a crescentic hypodensity around the enhanced narrowed artery (arrow), consistent with a mural hematoma.

Fig. 2. Cervical angio-MRI at 3 months. Recanalization of the left ICA with a slight persistent narrowing of the ICA.

the neck or abrupt full flexion. The ICA may be compressed between the angle of the mandible and upper cervical vertebrae [3].

In our patient, there was no obvious causal factor for CAD, since common vascular risk factors have never been demonstrated to be associated with arterial dissections and heritable connective tissue disorders have been ruled out [1, 2]. The chronic tonsillitis may have contributed to vessel weakening of the upper respiratory tract, due to infections, by secretion of proinflammatory cytokines, free radicals or proteases, which increase the susceptibility of the arterial wall [2, 4]. The precipitating causal factor in our case seems to be the surgical procedure. A case of ICA dissection 24 h after adenotonsillectomy has already been reported in a 7-year-old child, but until 12 years of age, there is a close proximity between the ICA and the tonsillar bed, so a blunt or penetrating trauma may occur during the procedure [5]. In adults, due to the cervical anatomy, a direct trauma occurring during the procedure seems very unlikely and would have been obvious before the end of the surgery. The forced opening of the mouth with a cervical hyperextension, associated with the administration of the anesthesia by a mask, may have constituted the traumatic factor in our patient, where the obesity was a technical difficulty.

Interestingly, 2 cases of CAD have been reported occurring a few hours after surgical procedures performed under general anesthesia with tracheal intubations. The causal role of the mask anesthesia administration, with a compressive effect of the fingers holding the anesthesia mask and anchoring it by extending over the mandible and encroaching on the carotid bifurcation, has been suggested [6, 7].

Tonsillectomy is a common procedure rarely performed in middle-aged adults, where it is longer and more difficult than in children, especially in obese patients. Therefore, mouth and throat surgeons should carefully monitor young patients after long surgical procedures because stretching the soft neck tissues may cause delayed irreversible infarctions.

References

- 1 Schievink WI: Spontaneous dissection of the carotid and vertebral arteries. *N Engl J Med* 2001;344:898–906.
- 2 Rubinstein S, Peerdesman SM, Van Tulder M, Riphagen I, Haldeman S: A systematic review of risk factors for cervical artery dissection. *Stroke* 2005;36:1575–1580.
- 3 Lee WW: Bilateral internal carotid artery dissection due to trivial trauma. *J Emerg Med* 2000;19:35–41.
- 4 Guillon B, Berthet K, Benslamia L, Bertrand M, Bousser MG, Tzourio C: Infection and the risk of spontaneous cervical artery dissection: a case-control study. *Stroke* 2003;34:79–81.
- 5 Wraige E, Ganesan V, Pohl KRE: Arterial dissection complicating tonsillectomy. *Dev Med Child Neurol* 2003;45:638–639.
- 6 Cerrato P, Giraud M, Bergui M, Baima C, Grasso M, Rizzuto A, Lentini A, Gallo G, Bergamasco B: Internal carotid artery dissection after mandibular third molar extraction. *J Neurol* 2004;251:348–349.
- 7 Gould DB, Cunningham BS: Internal carotid artery dissection after remote surgery: iatrogenic complications of anesthesia. *Stroke* 1994;25:1276–1278.

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Reversible Posterior Leukoencephalopathy Syndrome in Catastrophic Antiphospholipid Syndrome

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A 47-year-old woman with a history of systemic lupus erythematosus (SLE) and secondary antiphospholipid syndrome (APS), treated by aspirin 160 mg q.d. and hydroxychloroquine 200 mg b.i.d., presented with subacute onset of bilateral blindness associated with headache 5 days after cholecystectomy (for acute cholecystitis). Aspirin treatment had been discontinued 1 week prior to cholecystectomy. At the age of 35 years, the diagnosis of APS had been made after recurrent deep venous thrombosis of the leg with high titres of anticardiolipin antibodies and positive lupus anticoagulant test. Coumarin treatment had been started but changed to aspirin 10 years later because of traumatic subdural brain haemorrhage.

On admission, clinical examination showed drowsiness and complete bilateral blindness with preserved pupillary reflexes. The blood pressure was 189/97 mm Hg. Fundus examination was normal. Activated partial thromboplastin time was increased (108 s, for a reference value inferior to 34 s) and slight hypoalbuminaemia was found. Platelet count, renal function, CRP, magnesiumaemia and cholesterolaemia were normal. Brain CT showed white matter hypodensity in the right fronto-parieto-occipital and the left parieto-occipital lobe, together with a right parietal and left parieto-occipital haemorrhage (fig. 1A–C). The day after, left hemiplegia appeared. MRI showed areas of increased white matter signal on T₂, FLAIR and ADC sequences (within the same distribution as white matter abnormalities on CT) and confirmed both brain haemorrhages (fig. 1D–F), in the absence of venous sinus thrombosis.

Intravenous nicardipine was started in order to lower the blood pressure. Three days later, left hemiplegia was almost completely recovered and visual field deficit improved, leaving binocular inferior altitudinal defect. The 24-hour urinary protein content was elevated. Renal involvement was probably not SLE-associated, as no complement breakdown products or circulating immune complexes were found. One week after admission, adrenal insufficiency (revealed by hyponatraemia, hyperkalaemia, hypoglycaemia and hypotension) was diagnosed by low morning plasma cortisol. Abdominal CT showed bilateral adrenal haemorrhage. Hydrocortisone treatment was started.

A diagnosis of probable catastrophic antiphospholipid syndrome (CAPS) was made because of multiple organ involvement (adrenal gland, kidney and brain) and rapid development of manifestations. Intravenous unfractionated heparin treatment was started.

One month after admission, complete recovery of visual field deficit and hemiplegia was seen. MRI showed a marked regression of brain lesions (fig. 1G–I). A diagnosis of reversible posterior leukoencephalopathy syndrome (RPLS) was made.