CLINICAL NOTE

Internal Carotid Artery Dissection Following Rigid Esophagoscopy

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A case of internal carotid artery dissection that developed after rigid esophagoscopy is described. The diagnosis was suggested by the clinical presentation and confirmed by the findings of radiological examinations. Internal carotid artery dissection is a rare condition of controversial etiology. Most frequently, the cause is unknown and the condition is termed *idiopathic*. A few cases have occurred after forceful cervical extensions and manipulations. The pathogenesis in our case is uncertain: while the rigid esophagoscopy is the most probable cause, the intubation and spontaneous carotid artery dissection cannot be ruled out.

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Serious complications of esophagoscopy include anesthesia-related cardiorespiratory complications, dental injuries, bleeding, aspiration, which can result in pulmonary infections of various seriousness, and perforation of the esophageal wall.[1] The exact incidence of complications associated with rigid esophagoscopy is still unclear, the majority of publications dealing with endoscopies performed for foreign body removal and reporting mainly serious complications, such as esophageal perforations and deaths. Giordano et al,[2] in reviewing reports of esophageal foreign body extractions prior to 1980, found a perforation incidence rate of 0.34% and a mortality rate of 0.05% in approximately 7000 cases. We describe a case of internal carotid artery (ICA) dissection that developed after an apparently uneventful esophagoscopy.

REPORT OF A CASE

A 39-year-old man underwent a rigid esophagoscopy for a suspicion of an esophageal foreign body (chicken bone). Tracheal intubation was uneventful. No foreign body was found, but a small superficial erosion, located 3 cm below the upper esophageal sphincter, was visualized. The patient awoke without complaints, was able to resume oral intake, and was discharged within 12 hours.

One month later, he complained of a pulsatile right retromandibular pain, irradiating toward the eye and the frontoparietal area, with blurred vision in the right eye. The findings of the head and neck examination were unremarkable. An ophthalmologic examination showed right pseudoenophthalmia, miosis, and anisocoria, which increased in darkness. A right Horner syndrome was confirmed by a cocaine test (5% cocaine solution administered as eyedrops).[3]

Sinus films and a neck-facial-cerebral computed tomographic scan showed no lesions or evidence of infection. A pulse-wave Doppler scan demonstrated a severely reduced blood flow, with a high resistance and a reduction of diameter in the right ICA. A dilatation of the right external carotid artery, with lower vascular resistance, was found. The pulse-wave Doppler scan results corresponded to a right ICA dissection. A magnetic resonance image showed a double-lumen artery with an intimal flap and an intramural hematoma (**Figure**).

The patient was told to take aspirin and given recommendations to avoid sudden neck movements and traumatic activities. Two months later, the headaches and blurred vision were improved. A second pulse-wave Doppler scan showed nor-

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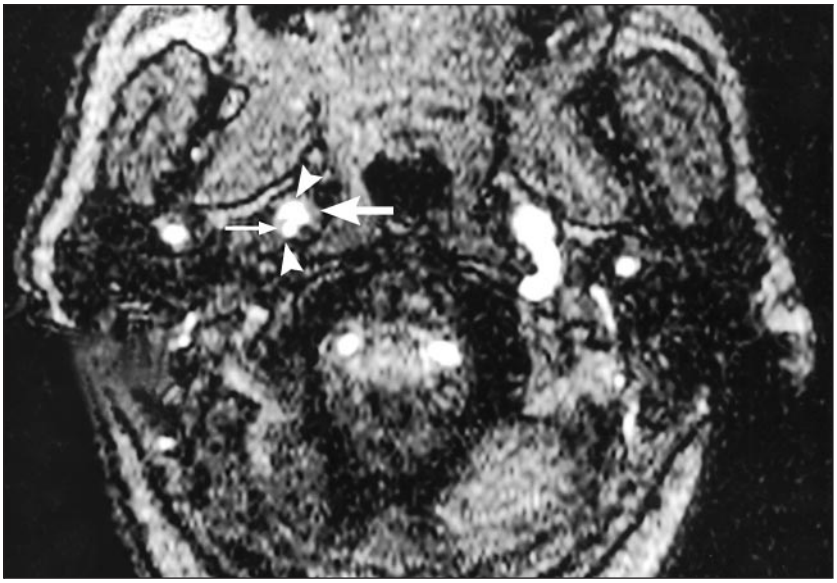

Source image from the 3-dimensional time-of-flight magnetic resonance angiogram, obtained 1 month after rigid esophagoscopy, showing the characteristic aspect of an internal carotid artery dissection. The true and false lumina (arrowheads) are seen as areas of increased-signal intensity, while the intimal flap separating these lumina is seen as a thin hypointense band (thin arrow). Note a semilunar hypointense area surrounding the right internal carotid artery corresponding to a persistent intramural hematoma (thick arrow).

mal resistance and flow in the right ICA, despite a persistent diameter reduction. Eighteen months after the esophagoscopy, the patient was asymptomatic, but the Horner syndrome was still present.

COMMENT

Dissection of the ICA is a rare condition, usually involving the distal cervical segment of the artery, contrary to atherosclerosis, which is located at the level of the carotid bulb and ICA takeoff. The petrosal portion of the ICA is spared, probably owing to protection by the surrounding bone.

Dissections of the ICA can occur spontaneously or after a known head and neck injury. Spontaneous ICA dissection was described in 1959,[4] and its yearly incidence has been estimated to be 2.6 cases per 100 000 persons.[5] The mean age at occurrence is the early 40s, with a slight predominance in females.[6,7] Spontaneous dissections of the ICA are responsible for 2.5% of stroke cases in patients younger than 50 years.[8]

In spontaneous dissections, the role of hypertension and trivial trauma, such as coughing,[9] straining,[9] and delivery,[10] has been emphasized.[6] Other diseases, such as fibromuscular dysplasia, Marfan syndrome, Ehler-Danlos disease, and α_1-antitrypsin deficit, are predisposing factors for arterial dissections and are found in 15% to 20% of cases.[6-8,11] Exceptionally abrupt or exaggerated neck movements have been the supposed cause. The presumed pathophysiological mechanism involves hyperextension and lateral flexion of the neck, stretching the ICA over the transverse process of the upper cervical vertebrae.[12] In the literature, 3 cases have been reported after chiropractic cervical manipulation,[13,14] 1 case after prolonged mask ventilation,[15] and 1 case after intubation.[15] In all these cases, the symptoms occurred within 1 to 5 days.

Our patient presented with retromandibular and homolateral headaches associated with blurred vision. Unilateral headaches are the most common clinical symptom of ICA dissections, usually followed, after a delay of a few minutes to a few weeks,[6,16] by focal cerebral ischemic symptoms or by ipsilateral incomplete Horner syndrome.[7,8,16] Headaches are unilateral, in frontoorbital or periauricular regions.[7] Pulsatile tinnitus is sometimes present.[7] Besides Horner syndrome, ocular symptoms can include blurred vision,[16] as in our patient. More rarely, spontaneous ICA dissections present with lower cranial nerve palsies,[16] the most frequently affected nerve being the hypoglossal.[6]

Occasionally, spontaneous ICA dissections are asymptomatic and are detected incidentally. The overall frequency of ischemic events, ranging from transient ischemic attacks to complete strokes, varies from 50% to 95%.[6,8] However, the majority are transitory events, followed by a complete neurological recovery.[5]

The pathogenesis of ICA dissection is still unclear. The dissection may be caused by tearing of the intima, followed by penetration of circulating blood into the vessel wall, or by a primary intramural hemorrhage from the vasa vasorum that secondarily ruptures into the true lumen.[6] This hematoma may extend along the vessel wall, more often distally than proximally, and may be a source of thrombosis and emboli. If it ruptures back into the lumen, it will create a double-lumen artery.[17] Sometimes, the hematoma expands toward the adventitia and causes an aneurysmal dilatation, also called a *dissecting aneurysm*.[7,17]

The symptoms and signs associated with ICA dissection result from decreased blood flow, an embolization from the aneurysm, or an intramural thrombus. The Horner syndrome is caused by the compression or stretching of sympathetic fibers as a result of the enlargement of the artery. This syndrome is incomplete, because the sympathetic fibers responsible for facial sweating follow the external carotid artery and its branches.[7] The irritation of the wall of the carotid artery could explain the unilateral facial pain and headache.[9]

Ultrasonography (with duplex sonography, Doppler colorflow imaging and transcranial Doppler examination) is a very sensitive tool in the early diagnosis and follow-up of ICA dissection.[8] Doppler color imaging permits the determination of the degree of obstruction and the extension of the hematoma, which is often hypoechogenic. Ultrasonograms may be important during follow-up evaluation, showing lumen recanalization and normalization of the blood flow stream.[8] Angiography was consid-

ered the "gold standard" for the diagnosis of arterial dissections but detects only a decrease in the vessel diameter and lacks sensitivity.[6,17] Computed tomography can be used to demonstrate an intramural hematoma, but it, too, is not considered a reliable and sensitive screening technique.[18] Magnetic resonance imaging is the only noninvasive technique that can demonstrate an intramural hematoma, show its longitudinal extension, evaluate the degree of wall expansion, and determine the relationship of the involved artery with the surrounding tissues.[18] The role of magnetic resonance angiography is currently under evaluation.

The management of the ICA dissection is directed at the prevention and propagation of the thrombus and distal embolization. Since spontaneous ICA dissection may be complicated by a stroke up to 1 month after the onset of symptoms, a preventive treatment, initiated as soon as possible, is suggested. The best treatment remains unclear: heparin is most widely used, but aspirin is sometimes favored, particularly in the absence of ischemic signs.[7]

The evolution is usually favorable, with improvement or complete recovery in at least 80% of patients.[6,7,9] In case of persistence of severe stenosis, floating thrombus, or residual aneurysm after 3 months of medical treatment, surgical treatment is proposed.[19]

Our patient presented with an ICA dissection after undergoing an uneventful rigid esophagoscopy under general anesthesia. His symptoms, other than the Horner syndrome, resolved. The respective roles of endoscopy and tracheal intubation are speculative. Nevertheless, usual oral intubation requires much less neck extension than esophagoscopy, so we tend to attribute the ICA dissection to the endoscopic procedure. Also, the distinction between true spontaneous ICA dissection and a dissection that develops after minimal trauma, such as neck extension during esophagoscopy, is somewhat arbitrary.

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