SPONTANEOUS CAROTID DISSECTION WITH HYPOGLOSSAL NERVE PALSY AS RESIDUAL DEFICIT

The importance of magnetic resonance evaluation

Marcio Luiz Escorio Bezerra, José Luiz Pedroso, Alexandre Pieri

Spontaneous dissection of the carotid artery was once considered uncommon, but nowadays neuroimaging progress has lead to an increased recognition of the pathology. It is an important cause of stroke in young patients, representing 10 to 25% of the cases\(^1,2\). Most patients affected are in the fifth decade of life\(^2,3\). It is not definitely established whether the cardiovascular risk factors are important in spontaneous carotid dissection; however, tobacco use, migraine and respiratory tract infections have been shown to be relevant in different studies\(^1-4\). In spite of the term spontaneous dissection, minor precipitating events, such as rotation or hyperextension of the neck, are frequently found\(^5\). The most frequent initial symptom is headache, which is reported in up to 90% of the cases, usually preceding the neurological deficit. Few patients present the classical triad of headache, ischemic stroke and Horner syndrome. Cranial nerve palsy is found in about 12% of the cases, but unusually it is the sole manifestation\(^1\). We report a case of carotid artery dissection presenting as lower cranial nerve palsy and headache. The diagnosis was made through magnetic resonance imaging (MRI), using fat suppression technique, highlighting its importance in such cases.

CASE

A 45 year-old right handed man with previous history of systemic hypertension experienced severe right cervical pain followed by pain in the ipsilateral ear. He reported no traumatic or strenuous event in the previous days. The pain remitted with regular analgesics, but two days later it returned with the same qualities, what made the patient seek medical counseling. The systemic blood pressure was high, and analgesics and antihypertensive...
medications were administered. On this same day, he felt that his tongue was numb on the right posterior area and he noticed difficulty in manipulating food in the mouth. The next morning he woke up with tongue paresthesia, dysphagia and dysarthria. He was then taken to the hospital. Upon admission, general examination was unremarkable. Neurological examination disclosed right hypoglossal palsy, in addition to ipsilateral palsy of the glossopharyngeal and vagus nerves. Cranial CT showed no abnormalities and brain MRI was highly suggestive of right carotid artery dissection (Fig 1A). Carotid and vertebral Doppler ultrasonography was also performed, but showed no abnormalities. Antiplatelet therapy was started. Four months later a second MRI and MR angiography showed no signs of dissection (Fig 1B). Follow-up clinical examination detected a mild hypoglossal palsy (Fig 2), but there was no longer dysarthria or dysphagia.

DISCUSSION

Lower cranial palsy has a large range of differential diagnoses, such as malignant tumors in the base of the skull, neurinomas, leptomeningeal carcinomatosis, infectious diseases or may even be idiopathic, but carotid or vertebral dissection should always be considered. The absence of ischemic stroke and the fact that clinically it presented as lower nerve palsy, may have delayed the diagnosis in this case.

Carotid dissection usually arises from an intimal tear of the artery. The intramural hematoma may grow towards the intima, which cause stenosis of the arterial lumen or towards the adventicia, resulting in aneurismal dilatation. Considering that there were only local symptoms (cranial nerve palsies) and that brain MRI showed only intramural hematoma and no vascular lumen abnormalities, it was probably an adventicia dissection. In contrast, with the intimal tear, the vascular lumen is affected and tends to cause distal ischemia due to embolization.

The gold standard diagnostic method for carotid dissection is angiography, which is limited by its invasiveness. This case highlights the importance of MRI with MR angiography as a diagnostic method. Its resolution approaches the conventional angiography and is able to show intramural hematoma, especially when fat suppression techniques are used. The complementary exam also showed that the dissection extended to the petrous portion of the carotid artery, which is an uncommon event because usually the temporal bone represents a barrier to the pathologic process. Also, it became evident that even thought ultrasonographic techniques are useful, as the dissection is a dynamic process, the abnormalities may appear only in an initial moment.

The follow-up MRI with no abnormalities emphasizes the dissection as a transitory process with a good prognosis. Treatment involves antithrombotic or antiplatelet therapy, as was the case in our patient. Systemic anticoagulation may bring some benefits by reducing the risk of embolization and aiding intimal healing, but there is no conclusive evidence in current literature for one choice over the other.

REFERENCES