An internal carotid artery dissection presenting with facial peripheral palsy treated with endovascular therapy.

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INTRODUCTION

We present the case of a left peripheral facial palsy as symptom of a left internal carotid artery (ICA) pseudoaneurysm which required endovascular treatment as second choice after medical therapy failure. A 39 years old men suffering from ICA dissection and pseudoaneurysm worsened despite medical therapy. The review of literature showed that peripheral facial nerve palsy can be a very rare presentation of ICA dissection and its pathophysiology remains uncertain. To our knowledge this is the sixth case described in literature [1, 2, 3, 4, 5]. Furthermore we report our single case experience of the good outcome of endovascular treatment as a safe and effective alternative for ICA dissection in case of medical therapy failure.

CASE REPORT

A 39 years old male suffering from hemicrania came to our emergency department because of the onset of left eye ptosis and anysocoria after physical effort. The day before he had suffered from cephalalgia and left sided neck pain. The neurological examination confirmed a left incomplete Horner syndrome. No other focal signs were present. Brain CT and AngioCT showed left ICA ectasia about 7 cm after the bifurcation, followed by narrowed and absent contrast signal until the end of the petrous tract. The patient was admitted to our stroke unit and a cerebral DSA showed a 7x5 mm pseudoaneurysm of the last portion of extracranial left ICA. Patient was discharged with dual antiplatelet therapy.

17 days after the first symptoms onset the patient came back to our hospital suffering from left sided upper and lower facial palsy. Brain AngioCT and MRI showed increased size of the left ICA pseudoaneurysm and left hemisphere mean transit time delay in PWI sequences. The following day cerebral DSA was performed which confirmed the pseudoaneurysm enlargement and showed further narrowing of the petrous tract of left ICA.

Because of the worsening of the clinical and radiological picture despite medical therapy, together with the neurointerventionalists, we decided to try endovascular treatment. An Enterprise (4,5 x 14mm) Stent was deployed in the petrous tract of left ICA just downstream the pseudoaneurysm in order to diverge the blood flow from the pseudoaneurysm and maintain the intracranial ICA patent. Twelve days after, the patient was discharged with dual antiplatelet therapy, neurological examination was negative and left ICA stent was patent at a final control AngioCT.

DISCUSSION

Carotid and vertebral artery dissection represent 1–2% of all ischaemic strokes, but considering just the under 50 years old population they account for 10–25% of strokes [6, 7]. However, several cases remain undiagnosed because of the poor clinical signs making patients overlooking the pathology. The average annual incidence rate for ICA dissection is estimated to be 1.72 per 100,000 population [8]. Main risk factors from ICA dissection are age, trauma, infections, genetic or inborn predisposition/disorders such as connective tissue disease and vessel abnormalities, atherosclerosis and other vascular risk factors [9].

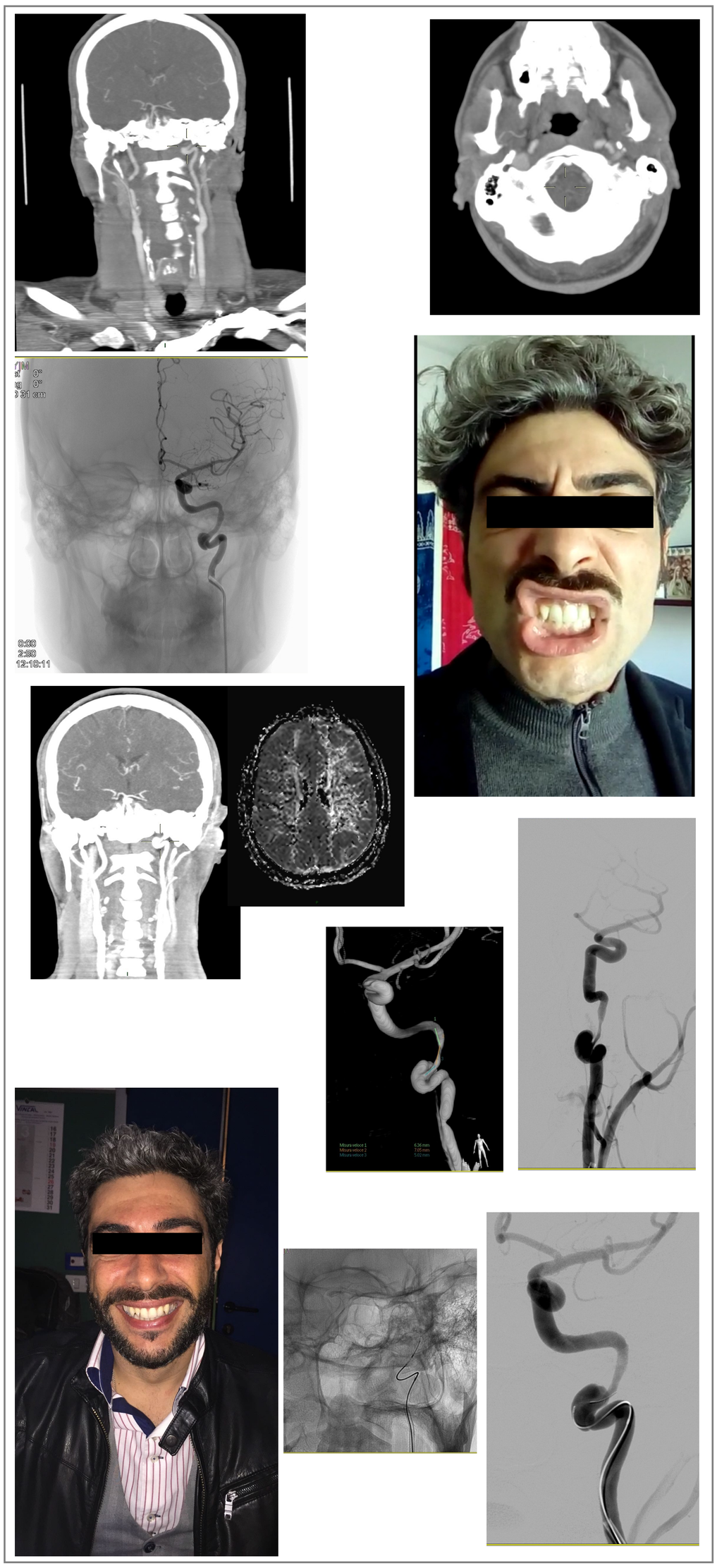
The most frequent clinical manifestations included head or neck pain, cerebral ischemic symptoms (TIAs or stroke), Horner syndrome, or ocularsympathetic paresis (OSP). To our Knowledge there are just 5 cases in literature describing peripheral facial palsy as a manifestation of ICA dissection, all of them reported a Cavernous tract pseudoaneurysm. Conversely our patient suffered from petrous ICA dissection but pseudoaneurysm was located in the final tract of cervical ICA [1, 2, 3, 4, 5].

As an option peripheral facial palsy can be caused by compression of geniculate ganglion, but bone erosion would be necessary and in our case there was not any sign of it. A pathophysiologic explanation includes feeding artery of nerve to be involved. Usually the VII cranial nerve receives feeding support by branches of the external carotid artery via meningeal medial system, but different collaterals and anatomic abnormalities are described, including feeding artery of facial nerve originating from ICA [10]. These vessels lie under the resolution power of DSA. We believe that the dissection of petrous ICA diagnosed in our patient may have compromised left facial nerve blood supply by haemodinamic effect more than by embolic source. This hypothesis is supported by the palsy regression after endovascular treatment.

According to Italian and European stroke guidelines [11, 12], medical therapy is the best first approach for ICA dissection, both anticoagulant and antiplatelet agents are safe and effective in order to prevent embolization, anyway the choice of the last ones can be preferred in case of large infarcts or high risk of subarachnoid hemorrhage. Endovascular treatment can be considered as second line in case of worsening of the clinical and radiological picture despite medical therapy, this approach includes either artery occlusion, when tolerated, or supporting patency of the artery and excluding the pseudoaneurysm by stent deploying. As ICA dissection is a quite uncommon pathology still there are not clinical trials with sufficient patients testing endovascular treatment's efficacy and safety. However several retrospective studies and clinical series support its use. Moreover the rapid development of new generation devices in neurointerventional field might support the spreading of this technique [13, 14, 15]. We decided to consider the endovascular treatment for our patient because of the new clinical sign and the data coming from neuroimaging suggesting progression of the pathology.

CONCLUSIONS

ICA dissection represent an important cause of juvenile stroke, for this reason it is important to recognize it promptly in order to carry out therapeutic intervention and prevent new episodes. Most of times clinical presentation of the disease allows a rapid diagnosis but in some cases its symptoms can be misleading and cause mistakes and delays in treatment. Peripheral facial paralysis is a rare manifestation of ICA dissection and a careful medical history and examination can reveal its origin and guide the diagnostic path. Cerebral DSA remains the gold standard for the detection of the pathology and can give important information about the cerebral hemodynamic. Moreover endovascular techniques must be considered as a possible and useful alternative for ICA dissection treatment when medical therapy is not effective.



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