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Ischemic Stroke Secondary to Dissection of the Internal Carotid Artery: A Case Report and a Brief Review

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Abstract

Background: Internal carotid artery (ICA) dissections generally occur when a tear occurs in the intima-media layer, either of traumatic or spontaneous origin. In this scenario, multifocal cerebral thrombosis may be a clear consequence of untreated ICA dissection. The incidence of internal carotid artery dissection in the US is estimated to be 1.72 per 100,000 population per year. Objective: To make a brief review of the literature and to explain, through a clinical case, the scenario of multiple ischemic cerebrovascular events is associated with the dissection of the internal carotid artery, in a young patient with no relevant history. Presentation of the Case: A 46-year-old female patient, with no significant history, who came to the emergency service presenting loss of strength in the right pelvic limb, disordered language, and difficulty walking for 3 hours, in the neurological examination evidence of decreased tendon reflexes and loss of strength of the right side of the body. Imaging studies show compromise of the M2 and M3 segments of the left middle cerebral artery, as well as multiple areas of associated ischemia in this hemisphere. Additional studies identify concomitant left ICA dissection. Based on the findings, it was decided to perform a thrombectomy of the affected area, as well as subsequent placement of a stent in the area of dissection. In the control angioTC study, revascularization of the M2 and M3 segments was observed, and in the follow-up of the patient, complete recovery of the symptoms was observed, with muscle strength, tendon reflexes, and preserved language functions. Conclusions: The ischemic cerebrovascular event is a

frequent pathological entity in the emergency service; in the scenario of a young patient as mentioned above, it is important to take into consideration the different causes of this pathology. We are obliged to familiarize ourselves with the different imaging modalities for an adequate diagnostic approach, as well as the treatment methods according to the time of evolution.

Keywords

Ischemic Stroke, Dissection, Internal Carotid Artery

1. Introduction

A tear occurs in the intimal layer of the carotid artery secondary to trauma or of spontaneous origin. The tear causes a continuous flow towards the false lumen, generating an increase in the thickness of the wall and subsequent stenosis [1]. Neurologic abnormalities, a history of recent trauma, and head, neck, or face pain, are the initial clues for diagnostic suspicion [2]. The incidence of internal carotid artery dissection in the USA is estimated to be 1.72 per 100,000 population per year [3].

Magnetic resonance imaging can help determine the timing of an ischemic cerebrovascular event (CVE). Signal intensity on FLAIR images varies after stroke. However, the FLAIR imaging findings are positive 6 - 12 hours after the onset of symptoms, showing arterial hyperintensity at the beginning of the stroke, within 0 - 2 hours after the onset of symptoms [4]. ADC maps can represent hypointense foci within minutes of stroke onset and are more sensitive than diffusion-weighted sequences, which demonstrate hyperintensity. Darkening on ADC maps distinguishes the trace from the "T2 glow", a later finding that occurs after infarction and appears bright on both diffusion-weighted images and ADC maps [3]. The duplex examination may show an absent flow signal in the internal carotid artery, bulbous biphasic flow, high-resistance flow pattern from the ipsilateral common carotid artery, signs of collateral flow through the circle of Willis, and low flow in the cerebral artery average in transcranial insonation [5].

The ultimate goals of carotid dissection treatment include stabilizing the dissection, improving complications, and healing the false lumen [6]. Endovascular treatment consists of the placement of the endoprosthesis in the ICA beginning with balloon angioplasty of the dissected segment, which allows the lesion to be recognized, especially at the point of entry to the dissection. Next, the stent is released from distal to proximal until the entire length of the dissection is covered and, more importantly, the entry point. With this technique, successful dilation of the ICA of up to 83.3% has been achieved [7].

2. Objective

Presentation of the clinical case and a brief review of the literature, in the setting

of an ischemic cerebrovascular event of multiple foci, conditioned by the dissection of the internal carotid artery of spontaneous origin.

3. Case Report

A 46-year-old female patient, who came to the emergency department reporting loss of strength in the lower extremities, predominantly in the right pelvic limb, a language disorder, and difficulty walking for 3 hours of evolution. On neurological examination, the patient was alert, with conduction aphasia, reduced tendon reflexes, and muscle strength. A magnetic resonance imaging of the brain was performed in Flair sequence, without apparent alterations (**Figure 1**). The DWI and ADC sequences (**Figure 2**) show at least 4 lesions located in the cortico-subcortical, insular, operculoparietal, periatrial, and ipsilateral corona radiata, findings consistent with areas of ischemia-infarction in the acute phase.

Cerebral magnetic resonance angiography, where decreased vascularization is visualized in the branches of the left middle cerebral artery (MCA), segments M2 and M3 (**Figure 3**).

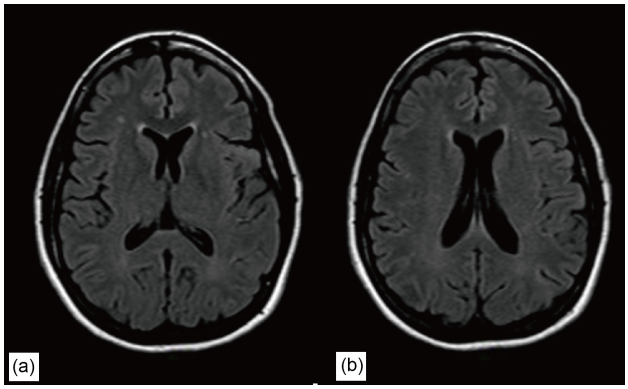

Figure 1. Axial Brain MRI in FLAIR sequence. Panels (a) and (b) show no significant findings on the FLAIR sequence.

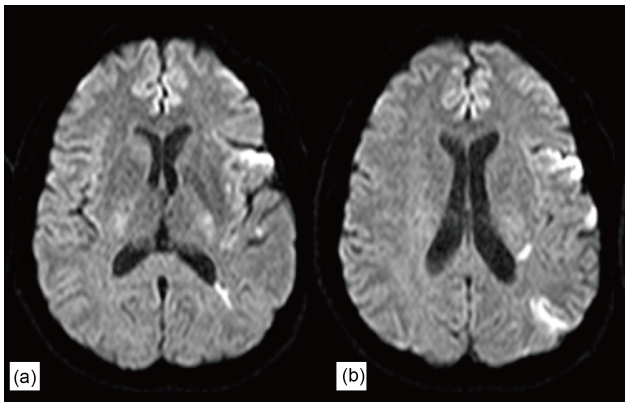

Figure 2. Axial Brain MRI in DWI sequence. Panels (a) and (b) show diffusion restriction at the cortico-subcortical, insular, operculoparietal, periatrial, and ipsilateral corona radiata levels, findings compatible with areas of ischemia/infarction in the acute phase.

Doppler of the carotid arteries was requested, where an image of heterogeneous morphology, ovoid and anterior intramural location, with extension from the bulb to the proximal third of the internal carotid artery (ICA) is visualized, conditions a reduction in the lumen of the 70%. In power Doppler, there is saturation in this region, and in spectral Doppler, there are no data of hemodynamic repercussion, findings compatible with left ICA dissection and intramural thrombus/hematoma (Figure 4).

Angiography is requested and the diagnosis will be corroborated, an absence of opacification is identified at the level of the M2 and M3 segment, as well as an intimal flap at the level of the left ICA. Navigation was performed with a hydrophilic microguide to segment M2 and M3 of the left middle cerebral artery and thrombectomy was performed with the Solumbra technique, which involves the use of a retractable stent and concomitant aspiration. Subsequently, balloon plasty was performed at the level of the ICA dissection, with the placement of a Zilver self-expanding stent (Figure 5).

Control angiotomography is requested, showing satisfactory opacification of the M2 and M3 segments of the left middle cerebral artery (Figure 6).

The patient was followed up, observing satisfactory recovery of muscle strength, tendon reflexes and language functions. The prognosis of the patient was good, and periodic neurological evaluation was indicated, as well as controls with Doppler ultrasound of the carotid arteries.

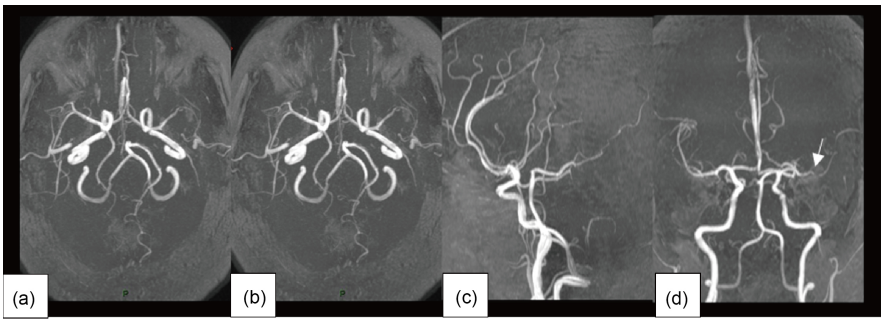

Figure 3. Brain MRI Angiography. Panels (a), (b), (c), and (d) show decreased vascularization in the branches of the left MCA, segments M2 and M3 (white arrow).

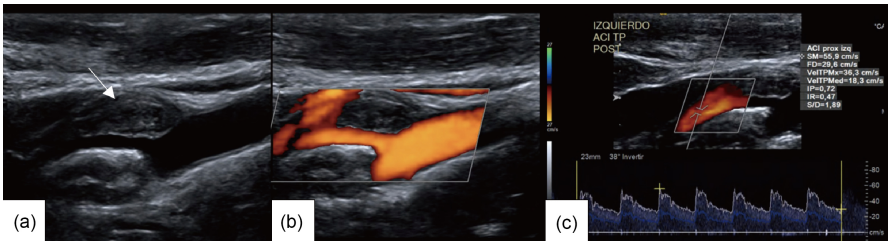

Figure 4. Doppler of the left carotid arteries. Panel (a) shows in (b) mode, an image of heterogeneous morphology, ovoid, and anterior intramural location (white arrow), with extension from the bulb to the proximal third of the ICA, conditions a 70% reduction in light. Panel (b) shows at power mode, saturation in this region. Panel (c) shows the spectral Doppler, absence of hemodynamic repercussion. Findings were consistent with left ICA dissection and intramural thrombus/hematoma.

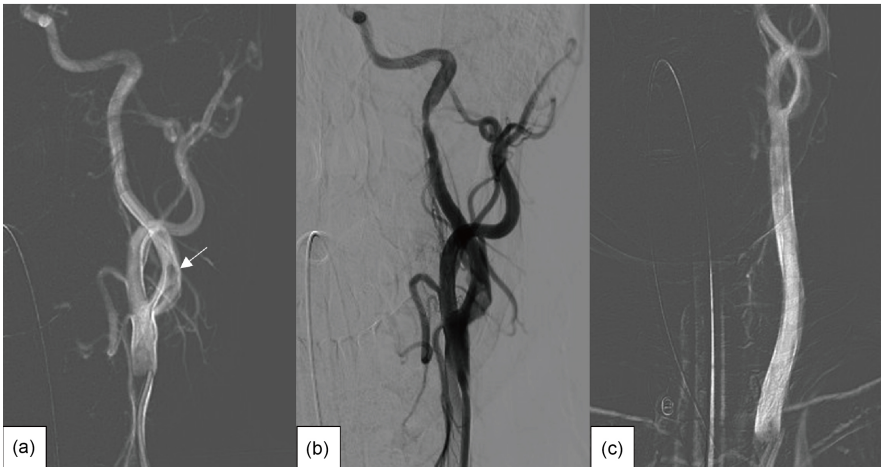

Figure 5. Digital subtraction angiography. Panels (a), (b), and (c) show the intimal flap at the level of the left ICA (white arrow). After identifying the affected site and its extension, thrombectomy, angioplasty, and stent placement are performed.

Figure 6. Brain angiotomography. Hyperdense vascular pathways of the anterior and posterior circulation are observed, as well as the permeability of M2 and M3 segments of the left MCA (white arrow).

4. Discussion

Studies conducted in the United States have reported an incidence of spontaneous internal carotid artery dissection of 1.72 per 100,000 population per year [3]. 2% of all cases of cerebral ischemic events are attributable to dissection, originating from the internal carotid artery. Generally, the patient presents with headache, neck or face pain, as well as partial Horner's syndrome and cerebral ischemic event. The classic triad is found in less than 30% of patients; If at least two are present, the diagnosis should be suspected [8]. Intimal tearing and continued blood penetration into the artery wall lead to false lumen formation with intramural hematoma, which can lead to stenosis, vascular occlusion, and thrombus formation [9].

The causes of ICA dissection can be: traumatic or spontaneous, the latter being attributed to collagen pathologies. In a publication of 2 case reports by L A

Kalashnikova *et al*., It is proposed that vascular connective tissue damage in patients with Turner syndrome is associated with a deficiency of extracellular matrix protein biglycan, which interacts with collagen and elastin to strengthen the arterial wall. The gene for this protein is linked to the X chromosome that is totally or partially missing in Turner syndrome, resulting in a biglycan deficiency. However, the association between sex hormone deficiency and arterial wall weakness is uncertain and more studies are needed to date [10]. In another case report publication by Hajime Ikenouchi *et al*., dissection of the internal carotid artery associated with multiple ischemic infarcts is mentioned in a 30-year-old patient with a history of Ehlers-Danlos syndrome [11]. In the classic Ehlers-Danlos presentation, the kyphoscoliotic subtype shows molecular defects in the PLOD12 and FKBP1416 genes, which are associated with vascular rupture [12]. This last case presents clinical similarity to the case presented in this article, however, we do not know if there was similar pathogenesis generated by some type of collagen disease.

Ultrasound with the application of color, power, and spectral Doppler is a non-invasive and low-cost method that allows us to characterize: the mural hematoma, the percentage of stenosis, the blood flow towards the false lumen, the changes in the systolic velocity, and the spectral modifications.

Computed tomography has high sensitivity in identifying false lumen and wall hematoma, showing increased wall thickness without changes in vessel diameter. Magnetic resonance imaging offers us the magnetic susceptibility sequence (SWI), which will allow us to characterize the presence of wall hematoma. However, digital subtraction angiography (DSA) remains the study of choice to define this pathology, showing the location and degree of involvement [13].

Currently, angiography is the method of choice to characterize defects in vascular brain permeability and internal carotid artery dissections. The technique used to solve this pathology begins by placing an introducer in the common femoral artery, with subsequent advancement of guides and catheters to the site of interest under fluoroscopy, identifying the sites of obstruction by angiography. After this, a mechanical thrombectomy is performed, which can be performed using a retractable stent, aspiration, or the Solumbra technique [13]. The widening of the wall and the stenosis generated by the thrombosis of the internal carotid artery dissection can be treated with balloon plasty and placement of a self-expanding metallic stent, showing good results [14]. In a scenario of cerebral thromboembolism of the middle cerebral artery and concomitant internal carotid artery dissection, endovascular treatment should be considered as the first therapeutic option [15].

5. Conclusion

The ischemic cerebrovascular event is a frequent pathological entity in the emergency service. In the scenario of a young patient as was previously mentioned, it is important to take into consideration the different causes of this pathology. We

are obliged to familiarize ourselves with the different imaging modalities for an adequate diagnostic approach; magnetic resonance will allow us to define the affected sites as well as the time in which the event occurred, diffusion being one of the main sequences to establish a cerebral hyperacute ischemic event. However, given that in the case presented, the ischemic event is secondary to a dissection of the intima of the internal carotid artery; Doppler ultrasound is a method that, unlike magnetic resonance imaging, will show important findings such as the percentage of stenosis, hemodynamic repercussion data, and location of the affected site. Diagnostic angiography remains the definitive method, showing the exact site of the condition and allowing adequate treatment planning. As we observed in the present case and according to the literature reviewed, thrombectomy with stent placement was the method of choice to release vascular occlusion and stabilize the dissection, thus reducing future complications.

Informed Consent

The authors attest that the patient provided written informed consent for the purpose of this publication.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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