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SPONTANEOUS DISSECTION OF THE BASILAR ARTERY: CASE REPORT

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Abstract: INTRODUCTION: The main cause of ischemic stroke in young people is arterial dissection due to trauma. Dissections that occur without obvious trauma, structural connective tissue or arterial disease are termed spontaneous. There is a correlation between the presence of a previous history of migraine in patients with spontaneous arterial dissection when compared to patients with stroke due to another etiology. CASE REPORT: Adolescent, female, 16 years old, history of migraine, with no other comorbidities. He presented intense pain in the cephalic region, evolving immediately with loss of consciousness, decerebration, without previous traumatic brain injury. Contrast-enhanced cranial computed tomography performed in the acute phase showed hyperdensity in the basilar artery with filling failure after contrast, suggesting a thrombus in its interior. complete provided. Left triceps, biceps, styloradial, patellar and Achilles hyperreflexia. And the right patellar hyperreflexia. Finger flexor reflex present bilaterally. Tromner and Hoffman positive. Absence of clonus. Plantar cutaneous reflex extended to the left and indifferent to the right. Pseudobulbar affect, left gaze palsy, absent gag reflex. She underwent brain magnetic resonance angiography 25 days after the event, showing a caliber reduction of approximately 50% in the middle third of the basilar artery. During hospitalization, a negative investigation for vasculitis and thrombophilia was performed. FINAL CONSIDERATIONS: In the case report, a cerebrovascular accident was evidenced after spontaneous dissection of the basilar artery in a young patient with previous migraine generating the Basilar Artery Syndrome characterized by motor and oculomotor signs and symptoms and alteration in the level of consciousness.
Keywords: Stroke. Migraine. Basilar artery. Basilar Artery Syndrome.

INTRODUCTION

Arterial dissection consists of separating the layers of the arterial wall. A false lumen appears in the space where the blood penetrates the vessel wall. Subintimal dissections develop an intramural hematoma and cause luminal stenosis or occlusion. This can result in cerebral ischemia due to thromboembolism, hypoperfusion, or a combination of both. Thromboembolism is considered the main cause of ischemic symptoms (PANONE, ET AL 1998).

A common cause of ischemic stroke in young people is arterial dissection from head and neck trauma. Most cases may be due to a transient vasculopathy after minor trauma or infection (NORRIS JW, ET AL, 2000). Dissections that occur without overt trauma are often termed "spontaneous".

The proportion of patients with cranial and cervical artery dissection who are affected by a known connective tissue disorder or vascular disorder is low (DEBETTE, ET AL 2009). However, several connective tissue and vascular disorders have been associated with dissection, including the following: Fibromuscular dysplasia, Ehlers-Danlos syndrome type IV, Marfan syndrome, osteogenesis imperfecta, reticular fiber deficiency, homocystinuria, alpha-1 antitrypsin deficiency, brain aneurysms. There are no established genetic markers for cervicocephalic dissection (GIOSSI A, ET AL 2014).

There is a correlation between the presence of a previous history of migraine in patients with spontaneous arterial dissection when compared to patients with stroke due to another etiology (TZOURIO C., ET AL, 2000).

CASE REPORT

Adolescent, Brazilian, female, 16 years old, history of migraine without aura, continuous use of anti-inflammatory drugs for pain without use of prophylactic continuous medication, no other comorbidities, non-smoker, non-drinker, no previous head trauma, performed regular physical activity, weight training. She presented intense pain in the cephalic region, immediately evolving with loss of consciousness and decerebration. She was therefore admitted to the intensive care unit. Computed tomography of the skull with contrast performed in the acute phase showed hyperdensity in the basilar artery with filling failure after contrast, suggesting thrombus inside, without hemorrhagic transformation. The patient was admitted to neurology 14 days after the ictus presenting with tetrassegmental hypotrophy, amyotrophy in the hands, reduced tone and proportionate complete tetraplegia. Left tricipital, bicipital, stylorradial, patellar and achilles hyperreflexia. And on the right patellar hyperreflexia. Finger flexor reflex was present bilaterally. Tromner and Hoffman were positive. Absence of clonus. Plantar cutaneous reflex in extension on the left and indifferent on the right. Absence of meningeal signs. Isophotorreagent pupils. Slowed ocular motricity with conjugate gaze palsy to the left, nausea reflex absent, pseudobulbar affect, with unmotivated laughter and crying. On admission 25 days after the acute event, she underwent skull MRI angiography, which showed heterogeneous contrast impregnation in the pons, with posterior extension to the floor of the IV ventricle to the right of the midline, without diffusion restriction, with a small regional volume increase compatible with subacute ischemic insult and a reduction in caliber of about 50% in the middle third of the basilar artery. During hospitalization, a negative investigation for vasculitis and thrombophilias was performed, including anticardiolipin, antiphospholipid, anti beta 2 glycoprotein, factor V Leiden, lupus anticoagulant, FAN, rheumatoid

factor unreacted, detection of the G20210A mutation in the prothrombin-factor II gene negative anti thrombin III 135%, functional protein C, homocysteine and free protein S within normal limits, serologies for HIV, HTLV, syphilis, hepatitis, herpes negative. Serology for Chagas' disease negative. Liquor without specific alterations. A transthoracic echocardiogram was also performed which showed left atrium 26 mm, ejection fraction (teicholz) 63%, cardiac chambers with normal diameter, interatrial septal aneurysm with mild left-right shunt suggestive of patent foramen ovale.

DISCUSSION

Extracranial dissection is more frequent than intracranial dissection in reports from North America and Europe. However, evidence from several case series suggests that intracranial dissection is more common in Asian populations and in children (DEBETTE, ET AL 2015).

The patient in question is a young Brazilian woman who was diagnosed with intracranial dissection in the middle portion of the basilar artery affecting the pontine paramedian region of probable spontaneous etiology. Because no triggering event such as traumatic brain injury or an underlying predisposition such as connective tissue disorder or vascular disorder was found.

Dissection usually results in ischemic stroke or transient ischemic attack, often associated with local symptoms such as neck pain or headache that may mimic migraine, cluster headache, or have a sudden, severe onset with a thunderclap character. The headache was followed, in most cases, by a motor deficit (MITSIAS P, ET AL 1992; INTERNATIONAL CLASSIFICATION OF HEADACHES, 2018.)

Basilar artery occlusive disease most often presents as pons ischemia. The paramedian pontine base contains the long descending motor tract and the cerebellar fibers that cross it, and the paramedian tegmentum contains mainly oculomotor fibers (FERBERT A, ET AL, 1990). Which justifies the clinical picture presented by the patient.

Most patients with symptomatic basilar artery occlusive disease and pontine ischemia have some transient or persistent degree of paresis and corticospinal tract abnormalities. Initial motor weakness is often lateralized and has been referred to as the "heraldic hemiparesis" of basilar artery occlusion. Hemiparetic patients with basilar artery occlusion may have some motor or reflex abnormalities on the nonparetic side. It is common to have bilaterality of pyramidal symptoms, but with a certain degree of asymmetry. (VOETSCH B, ET AL, 2004; FERBERT A, ET AL, 1990).

Although the possibility that migraine could be a risk factor for intra- and extracranial arterial dissections was described 34 years ago by d'Anglejean-Chatillon et all. This fact has not yet been proven (D'ANGLEJAN-CHATILLON, ET AL 1989).

In a study carried out with 66 patients attended at two tertiary teaching hospitals in the metropolitan region of the city of São Paulo, Brazil, who had a diagnosis of carotid or vertebral artery dissection with no defined cause, more than half of them had a history of previous migraine (PIERI, ET AL 2017).

The case report cannot bring specific direct correlations. But other published studies suggest that migraine and arterial dissection may share common pathogenic mechanisms and underlying susceptibility factors. Considering that current theories accept the concept that migraine is a neurovascular disorder.

It is considered that vascular disorders can act as triggers of neurological manifestations. There is a hypothesis that serum levels of alpha1-antitrypsin, a proteinase that

inhibits the proteolytic effect of elastase and collagenase, are reduced in patients with migraine, which leads to an increase in elastase, a metallopeptidase that degrades elastin, increasing the risk of degradation of elastin. extracellular matrix (TZOURIO C., ET AL, 2000).

It is possible that molecular mechanisms that affect vascular function may be involved in migraine susceptibility. And the extracellular matrix defects represent a predisposing condition for spontaneous arterial dissection and for the patent foramen ovale (BRANDT T, ET AL, 2001).

FINAL CONSIDERATIONS

The literature on the relationship between migraine and specific etiological subtypes of ischemic stroke is scarce, but some studies demonstrate the clinical correlation between migraine and spontaneous arterial dissection. In the case report, an ischemic cerebrovascular accident was evidenced after dissection of the basilar artery in a patient with migraine without previous aura, compromising the brainstem and generating Basilar Artery Syndrome characterized by motor and oculomotor signs and symptoms and alteration in the level of consciousness.

IMAGES

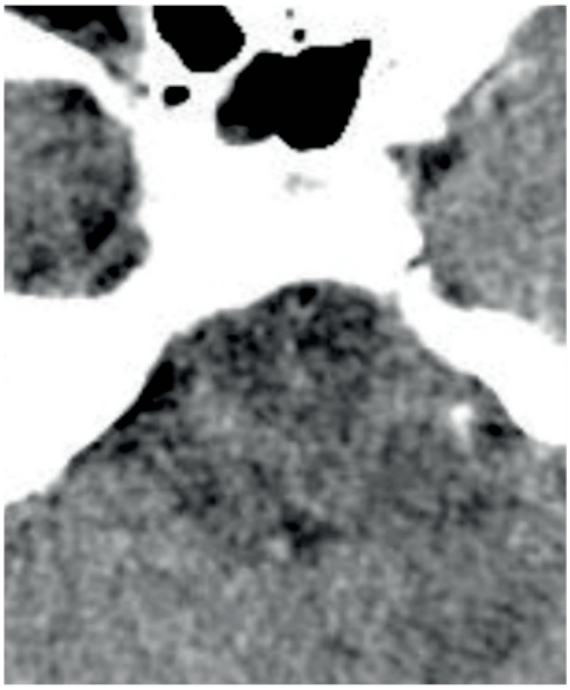

Figure 1:CT Skull with contrast. Posterior peripheral flow.

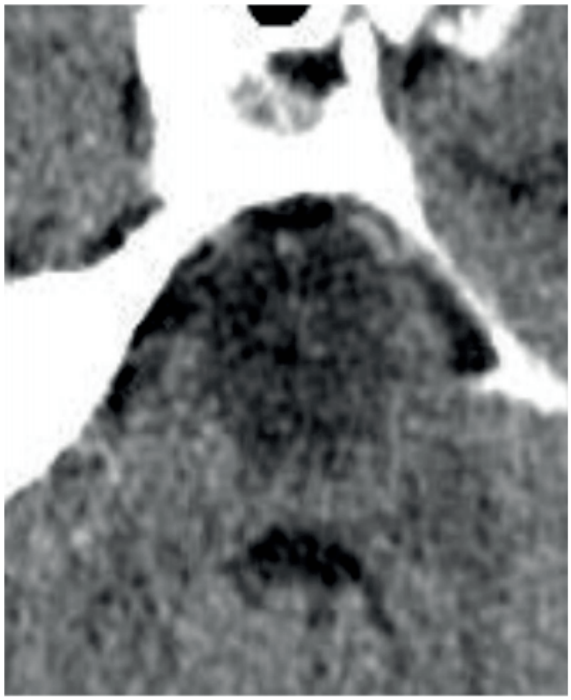

Figure 2:CT Skull with contrast. Anterior peripheral flow.

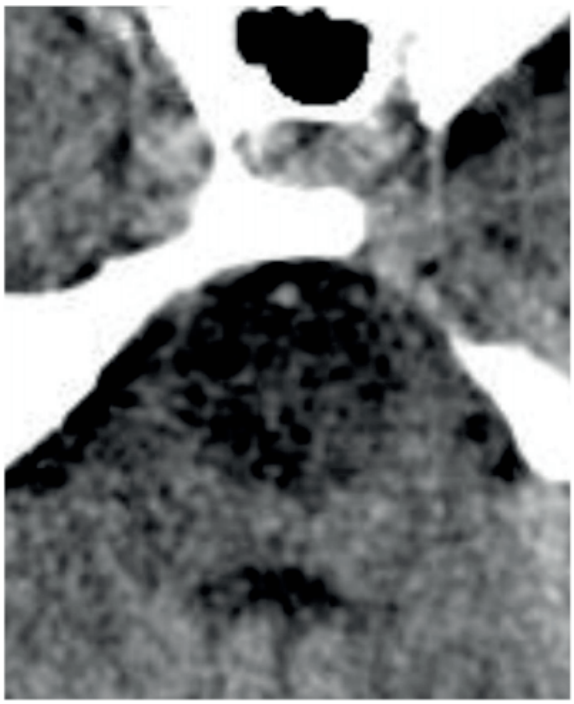

Figure 3: CT Skull without contrast. Spontaneous hyperdensity of a. basic.

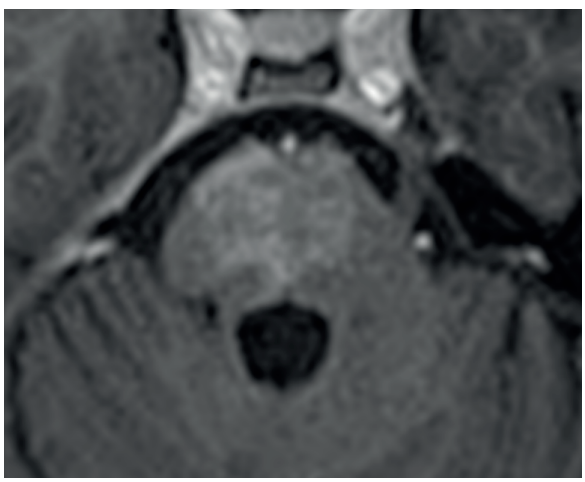

Figure 4: Contrast-enhanced T1 skull MRI. Enhancement at the pontine base denoting subacute infarction.

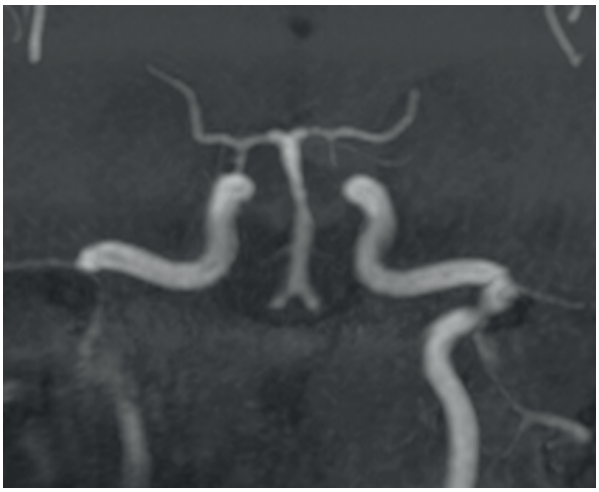

Figure 5:AngioRNM Skull. Tapering of a. basic.

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