Stroke from bilateral carotid dissection after dental procedure: case report

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ABSTRACT

Introduction: Carotid artery dissection (CAD) has the ischemic stroke as one of the main repercussions in previously healthy young individuals. It is common that in traumatic CAD, as in automobile accidents, the patient is subjected to a more complex investigation with imaging exams. These, allow an early diagnosis and, therefore, a treatment, reducing the chances of sequelae. However, non-traumatic causes with CAD outcomes have already been reported, such as dental causes. **Objective:** To contribute to the diagnostic valuation of non-traumatic CAD and subsequent stroke prophylaxis. **Methods:** We report a case of non-aneurysmatic dissection of the internal carotid arteries (ICA) after a dental procedure with evolution to bilateral stroke outcome in a 52-year-old patient. **Results:** Due to the strong association of CAD with traumatic factors, in a first contact with the patient there was no suspicion of CAD or stroke (absence of focal deficit). However, late, ICA occlusion on the left and stenosis on the right ICA were identified, resulting in two strokes and severe functional disability at discharge. **Conclusion:** The diagnosis of CAD is a challenge, especially since it is a multifactorial lesion. However, its early recognition will directly affect the patient's outcome. Therefore, in the presence of any warning symptoms, meticulous questioning about the individual's latest activities is necessary. This study alerts to the adoption of prophylactic conducts in the occurrence of causal factors such as prolonged cervical hyperextension or sudden movements of the cervical.

Keywords: Carotid artery dissection internal, Stroke, Dental implantation.

INTRODUCTION

One of the main repercussions of carotid artery dissection (CAD) is ischemic stroke[1]. With an incidence of around 2% of strokes in the general population, this percentage increases to 10 to 20% when considering the age group below 50 years[2]000, but reliable epidemiologic data for vertebral artery dissection (VAD. The pathophysiology of ischemic mechanisms secondary to CAD is related to luminal stenosis with distal ischemia due to brain hypoperfusion, being aneurysmal or embolic mechanism[3]. However, studies related to potential risk factors, the presentation of symptoms and CAD treatment are dependent on the type of dissection, with little consensus among them[4,5].

Spontaneous onset CADs are not associated with severe trauma (eg, automobile), but with structural changes in the artery (hereditary disorders of the wall and connective tissue)[5] or minor cervical trauma[4], such as hyperextension of the neck[6], cervical manipulations[7], or even, they are related to infectious diseases[8]. There are controversies regarding vascular risk factors such as hypertension and hypercholesterolemia in this type of occurrence[9].

Dental procedures can be associated with causal factors of CAD by simply maintaining posture in prolonged cervical hyperextension with

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anesthetized patients during the dental procedure[10]. To our knowledge, to date, only four studies have reported the occurrence of CAD after a dental procedure, with bilateral[10–12] and unilateral occurrence resulting in stroke[8].

With so many causal factors often not considered and an insidious symptomatic picture, as the carotid lesion may present different clinical signs at the time of dissection of the cervical arteries[9], the result may be a late diagnosis reflecting on the prognosis. The repercussion of CAD in ischemic stroke can only be confirmed by direct visualization of the brain injury, narrowing or occlusion of the cervical artery lumen through imaging exams. Angiography shows the arterial lumen and shows irregularities in the wall[13]. Although this is the gold standard modality to the CAD diagnosis, magnetic angiography (angioMR) and/or computed angiotomography (angioTC) are non-invasive alternatives and are currently preferred neuroimaging methods[13].

Considering this information, and aiming to contribute to the enhancement of the diagnosis, we describe a case of non-aneurysmatic dissection of the internal carotids on the left and right, resulting in bilateral stroke and severe functional disability at discharge. The etiopathogenic mechanisms of this unique case of CAD are widely discussed and compared with the main records in the literature.

CASE REPORT

A 52-year-old female patient, hypertensive and a smoker, sought the emergency service with discomfort in the left cervical region and difficulty raising her left arm. The symptoms started after a dental extraction procedure and a dental implant lasting hours. At the initial examination, she was conscious, oriented, walking normally, blood pressure: 140/80 mmHg, afebrile, suprapapular pain. Motor strength grade 3 for abduction of the arm and flexion of the left shoulder, grade 5 in the other muscle groups and limbs. Initially seen as musculoskeletal pain secondary to the dental procedure (extraction of four teeth and implant), which was initially treated with common analgesics and discharged from the emergency room.

The patient returned the following day with worsening of the condition, presenting disproportionate left hemiparesis with a predominance of proximal facio-brachial, blood pressure: 160/80 mmHg, alert, Glasgow Coma Scale: 15, without deviation from the conjugated look, score 5 in the National Institute of Health Stroke Scale (NIHSS). Emergency computed tomography (CT) of the skull showed cortical hypodensity in the parieto--occipital region of the right hemisphere suggestive of acute ischemic injury. At the time, thrombolysis with Tissue Plasminogen Activator (tPA) or mechanical thrombectomy was not performed, as it was already outside the therapeutic window. Support measures for stroke were promptly initiated and tests for etiological investigation were requested.

While waiting for the complementary exams, the patient evolved with reentrant seizures (state of convulsive illness) refractory to the initial measures, requiring orotracheal intubation and ventilatory support. The comparative evaluation with a second cranial CT results showed new hypodensities, affecting the entire territory irrigated by the left middle cerebral artery (MCA). (Figure 1).

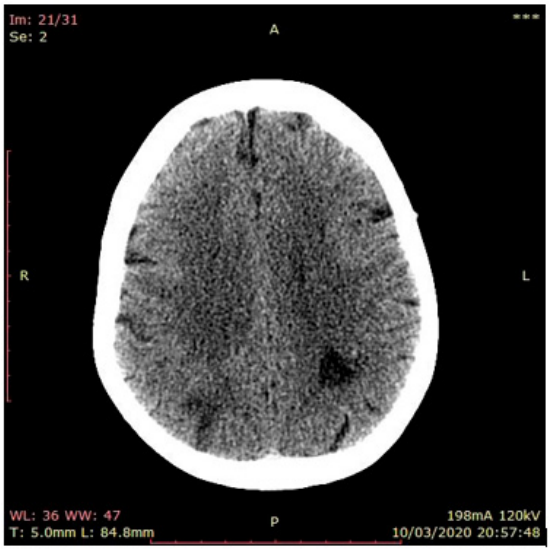

Figure 1: Axial CT scan of the skull (axial plan) showing hypodensities in the parieto-occipital region of the right hemisphere (ischemic insult type watershed) and the territory of the left Middle Cerebral Artery (MCA).

Due to the condition, a transthoracic echocardiogram was performed at the bedside, which

did not show embolic sources. Doppler of the carotid and vertebral arteries showed occlusion of the left common carotid artery without changes in the right carotid circulation and in the vertebral arteries. The cranial and cervical angioTC showed occlusion of the internal carotid artery (ICA) on the left and stenosis on the right ICA, both with characteristics compatible with CAD (Figure 2) and a marked reduction in vascularization in the left hemisphere, these being supplied by anastomoses (Circle of Willis) and collaterals (Figure 3).

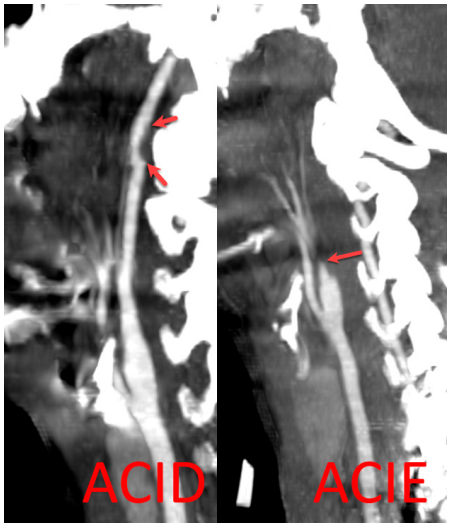

Figure 2: Sagittal section of Cervical Angiotomography showing stenosis and occlusion of the Internal Carotid Arteries in its extracranial segment (ACID: Right Internal Carotid Artery; ACIE: Left Internal Carotid Artery).

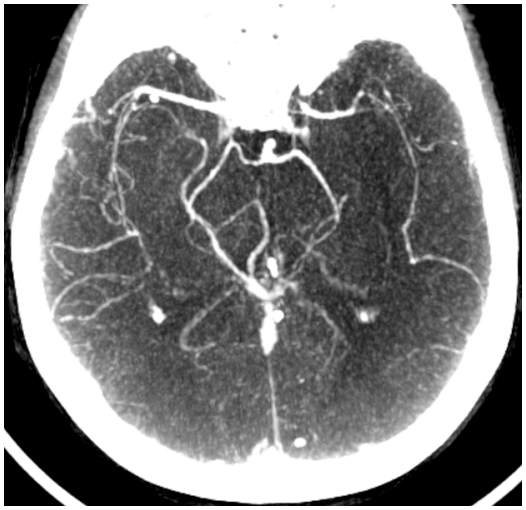

Figure 3: Axial section angiotomography of the skull showing a marked reduction in vascularization in the left hemisphere and filling of the vessels by contrast through anastomoses and collaterals.

The remaining diagnostic research with an electrocardiogram showed sinus rhythm and no signs of ischemia. Laboratory tests without noticeable changes. The patient was referred to the Intensive Care Unit (ICU) where she stayed for 35 days, persisting with a state of refractory disease.

She was discharged from the ICU in a minimally conscious and totally dependent state, with a score 5 on the Rankin Scale (mRS). After five months of discharge, in the outpatient follow-up, the patient did not present new strokes. However, she is still severely disabled (mRS 5 on August 3 of the current year). This study was approved by the Research Ethics Committee of the proposing institution (nº 3.609.943) and the legal guardian signed the Informed Consent Form authorizing the case report.

DISCUSSION

Our study presented a case report of a patient with bilateral CAD episode after a dental procedure, with initial presentation of neck pain and shoulder motor deficit, both on the left. Later, there was a severe neurological evolution due to 'two strokes' with consequent hemiparesis and severe functional disability at discharge. Occlusion was identified in the left IC with brain injury in the region between MCA and the right posterior artery. It is known that when there is the involvement of the innmRSost layers of the vessels (intimate and middle), luminal stenosis occurs, promoting distal ischemia by hypoperfusion[9]. The patient also presented stenosis in the right IC artery with ischemia in the left MCA territory compatible with embolism.

On the first patient's visit to the emergency department, the only signs were neck pain and a deficit in shoulder elevation. Thus, the absence of other signs suggestive of CAD led to an initial diagnosis of musculoskeletal pain due to the intraoperative positioning itself. According to the literature, other suggestive signs are those secondary to ischemic events, including the association of loss of consciousness, hemiparesis, aphasia and Horner syndrome, with a previous history most often of cervical trauma[9,14]. However, studies show that the association of these signs is

present in only one third of CAD cases[5,9], and may occur in isolation, or even the dissection may be asymptomatic[3,5,9]. It is common that in traumatic CAD, for instance automobile accidents, the patient is subjected to a more complex investigation with imaging tests that end up diagnosing CADs early, which are treated before the neurological deficit is installed1. However, the same does not occur in spontaneous CAD or minor trauma, as in the patient in question, predisposing the initial diagnostic imprecision. Because, in addition to the causal factor not raising the diagnostic hypothesis, the patient at the first moment did not have any focal deficit. In contrast to the retrospective study by Campos *et al*[15], in which, all patients had focal deficit.

However, even with associated clinical signs suggestive of CAD, the diagnosis is only proven by direct visualization of the intramural hematoma and narrowing or arterial occlusion of the lumen[13]. The most frequent question concerns the performance of invasive exams in the face of suspected CAD, such as angiography for surgical indication or whether non-invasive methods such as ultrasound (US) mode B, would be sufficient[13]. With AngioMR and AngioTC, the best imaging test to diagnose CAD has become even more complex. Most tertiary centers start investigating with AngioMR and/or AngioTC, reserving angiography (Gold Standard) for inconclusive cases.

The problem with performing imaging methods is the high cost, which restricts the performance of the Unified Health System (SUS). So, if there are no onset factors to motivate an investigation in this area, they end up not being carried out, a fact that directly affects the investigation and, consequently, the diagnosis confirmation. However, it is known that the cost of a post-stroke patient to SUS is R$ 6 thousand reals on average, with a cost of R$ 640 per day in cases of mild sequelae to R$ 32 thousand for severe sequelae patient and one month hospitalization periods. The early diagnosis of CAD can minimize neurological events and even prevent stroke, also reducing the economic burden on the country's health service[16].

The patient had pronounced hemiparesis twenty-four hours later, which was verified on return to the emergency service. According to the literature, the intervals between CAD and the appearance of signs and symptoms suggestive of the injury can be variable[3,15], as the dissection may be ongoing. Therefore, the evolution of severity in the clinical picture was probably due to an increase in the flap with a progressive decrease in the arterial lumen[9]. The flow was reduced causing a progressive cerebral infarction starting at the periphery ("watershed" infarction). So, due to the lack of clinical signs for the establishment of an early diagnosis, the "therapeutic window" was lost, which probably caused an increase in the central ischemic zone with the recruitment of penumbra zone cell [9].

Considering the causative factor of CAD, several anamnesis details of hyperextension or sudden neck rotation often precede the development of CAD. Sudden movements of the cervical can mechanically injure the arterial wall[17], the prolonged inclination of the head[18], sports activities[19], cervical manipulations[7], medical and surgical procedures such as anesthesia[20]. In the case of the patient in question, the dental procedure can be considered etiologically[10,11] due to the prolonged neck slope and the sudden movements of the neck through tooth extraction. The opening of the mouth reduces the distance between the angle of the mandible and the upper cervical vertebrae, favoring the compression of the cervical carotid arteries against the transverse processes of the upper cervical vertebrae or other bony regions[17]. The mechanical stretching of the soft tissues of the neck can also cause the rupture of the intima and media tunic with preservation of the more elastic adventitia[21]. Thus, these are hypotheses of CAD mechanisms to be considered.

Similar cases after a dental procedure have been previously reported[8,10–12]. Two of them showed an association of periodontal infection with CAD, however, in our study, given the results of laboratory tests, the possibility of the involvement of infectious processes can be ruled out. In the case portrayed by Cerrato *et al*. (2004), the patient presented sensorimotor impairment in the upper limb (UL) and initial hemianopsia due to unilateral CAD of the left IC and a single stroke in the parieto-occipital region[8]. Aghaebrahim *et*

al. (2013)[10] also report that the patient in their study presented sensorimotor impairment in UL due to bilateral CAD and a single stroke in the parietal region10. Both patients in the aforementioned studies were similar in age to the patient in this study, as well as, with similar sensorimotor symptoms. Unlike previous studies, the patient in this study had a stroke in the two hemispheres resulting from bilateral CAD, a fact that strongly compromised her clinical status and prognosis. Having this in mind, these cases highlight the importance of applying a protocol to investigate the occurrence of CAD due to minor traumatic events, such as after a dental procedure, enabling early diagnosis.

Regarding drug treatment, the prescription of anticoagulants and antiplatelet agents is recommended for CAD suspected, without evidence of superiority of one therapy over the other[22]. However, Santamarta-Fariña *et al*. (2004) report that despite the use of anticoagulants, the patient suffered a Transient Ischemic Accident in the hemisphere on the same side as the previous injury[23]. The same happened in the Aghaebrahim *et al*. (2004) study in which new areas of infarction were identified in imaging exams even after management with anticoagulation therapy in bilateral CAD after a dental procedure[10]. In this same study, the authors questioned the effectiveness of this therapy within the patient's clinical condition. Also, in this context, Daou *et al*. (2017) showed that, of the cases analyzed, there is a percentage with new or recurring events, despite the administration of antiplatelet therapy and anticoagulation in the CAD treatment[24]. However, research about ischemic stroke in CAD through maps of vascular territories and etiologies obtained by imaging studies (angiography, CT or magnetic resonance) suggest that most infarctions are probably embolic and not hemodynamic origin[25]. According to this mechanism, anticoagulant and antiplatelet therapies appear to be a logical and appropriate treatment in the initial stage of CAD. However, it is known that in some cases with persistent ischemic symptoms, the aforementioned therapies may not prevent new strokes, and the surgical approach would be the most recommended[3].

The literature reports a 5% mortality rate associated with cervical arterial dissections, the prognosis being directly dependent on the severity of the ischemic event[3]. This percentage may increase when considering carotid dissections, and even more so when bilateral. Campos *et al*. (2004), in a retrospective study of 48 patients with carotid and vertebral arterial dissection, found the occurrence of bilateral CAD in three patients (10.3%), two of them died[15]. Thus, considering the case presented in this study, with the evolution of CAD in two strokes and severe disability at discharge, it was possible to assume that the patient could remain with some level of functional disability. In outpatient follow-up, after five months of discharge, the patient was severely disabled (mRS 5) and has been assisted by the rehabilitation team.

CONCLUSION

In general, the prognosis after CAD shows good functional recovery and a low rate of recurrent dissection, ischemic and hemorrhagic complications. However, it was not found in the reported case, possibly due to a late diagnosis due to the low suspected, resulting from the sum of the atypical causal factor and the few initial findings. A cervical dissection can go unnoticed if it is not associated with relevant pain or neurological deficits that motivate the search for medical services, which makes it difficult to assess the exact incidence of this disease in the population. Thus, this study highlights that factors such as minor trauma may go unnoticed or be underestimated. Therefore, in the presence of any warning symptoms, a meticulous questioning about the individual’s latest activities is necessary, and the establishment of diagnostic criteria with the request for exams that may corroborate the clinical suspicion. Prophylaxis can also be adopted in the occurrence of causal factors such as prolonged cervical hyperextension or sudden movements of the cervical. Finally, the diagnosis of CAD is a challenge, especially since it is a multifactorial lesion. However, its early recognition will directly affect patient outcomes.

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