Cerebellar infarction after sneezing

INTRODUCTION

Vertebral Artery Dissection (VAD) is a rare condition, with an incidence of 0.97 to 1.5 cases per 100,000 inhabitants per year, affecting mostly young people and adults of both sexes1-3. Dissection generally occurs in the C2 and C3 segments of the vertebral artery and can be caused by a wide neck movement, with dis- tention of the vessel wall2,4. Thus, there is an endo-thelial lesion prone to dissecting blood intrusion into the wall, with probable thrombus formation, stenosis, or aneurysm. The association between these factors is capable of causing ischemia in the cerebellum, an organ irrigated by the vertebral artery, generating symptoms such as nystagmus, occipital headache, cervical pain, and vertigo5. Next, a case of VAD will be presented after a sneezing crisis, which caused cerebellar infarction.

SUMMARY

Vertebral Artery Dissection (VAD) is a rare condition that can be caused by a wide amplitude of neck movement, which injures the vessel wall and can cause ischemia in the cerebellum. We present a 37-year-old man with herniated lumbar disc and allergic rhinosinusitis, which caused sneezing spells. After one of these bouts with a ricochet of the head, he presented C3 misalignment with local pain. Twenty-one days later, affected by a new crisis, he presented left temporal headache, nystagmus, and vertigo. After 3 days, Magnetic Resonance Imaging (MRI) identified 2 regions of cerebellar ischemia and filling failure of the right vertebral artery. After 2 days, Computed Angiotomography (CT Angiography) was performed and showed right VAD with a local thrombus, without aneurysmal signs. Transcranial Doppler did not indicate an increase in blood flow from this artery. The suggested treatment involved administration of anticoagulant Apixaban 5mg, 12/12h, for 3 months, until the condition was reevaluated with new Angio CT and MRI. It was recommended that the patient was released from work for 1 month and forbidden from doing intense physical exercises for 3 months; however, due to setbacks, these deadlines were extended until a new appointment, 4 months after the first visit. The new tests showed no changes, indicating that the condition was stable. This case aims to indicate the possible investigations of the diagnosis and therapeutic options of the rare association between VAD with cerebellar infarction in a well-documented case.

KEYWORDS: Vertebral artery dissection. Cerebral Infarction. Stroke.
Case report

This case was duly submitted to and approved by the Research Ethics Committee, and the patient’s consent was obtained.

A 37-year-old male physician sought a neurological appointment due to complaints of mild left temporal headache and some episodes of vertigo with 1 week of evolution. He had a history of a herniated disc in the lumbar region and allergic rhinosinusitis. He denied smoking and alcohol use, as well as daily medication use. He has a family history of vascular abnormalities: the mother had a Cavernous Sinus aneurysm and the father had an atherosclerotic infarction (not due to vascular fragility).

The patient had frequent sneezing spells in salvo motivated by allergic rhinosinusitis, which were responsible for the bouncing movement of the head. About one month before the appointment, during one of these crises, the patient had a misalignment of the C3 vertebrae that caused local pain, which was minimized by the administration of muscle relaxant and anti-inflammatory medication. The condition was detected 2 weeks later, and there was complete recovery after physiotherapy. Three days before the neurologic appointment, the patient was again affected by sneezing associated with severe headache restricted to the left temporal frontal region. The patient reported having held back some of the sneezing, which indicates that the ricochet was intensified, acting as the cause of the distension of the vessel wall and, because he is a medical professional, there was a well-defined temporality and causal factor. To control the crisis, he used analgesics (Paracetamol) and antihistamines (Dexchlorpheniramine maleate). Right after one of these crises, nystagmus was observed without compromising horizontal balance, for 40 minutes, until it was resolved spontaneously. However, the patient remained with mild left temporal headache and limited episodes of vertigo that motivated the search for a professional three days later. During the neurologic appointment, the physical examination was completely normal, with no vestibular or oculomotor changes, absence of movement, and strength deficits. There were also no relevant changes from other devices.

Investigation

The patient underwent Magnetic Resonance Imaging (MRI) (Fig. 2) on the day of the appointment, which showed two regions of ischemia in the posterior region of the right cerebellar hemisphere and filling failure of the affected right Vertebral Artery. This same region, on a Computed Tomography (CT) (Fig. 1), was hypodense, which is consistent with cytotoxic edema in acute ischemias. Then, two days later, a Computed Angiotomography (AngioTC) (Fig. 1) was performed, which found a right VAD with a local thrombus, without signs of aneurysm. A Transcranial Doppler was also performed, showing no signs of increased blood flow from the same artery.

**FIGURE 1. CT AND ANGIOCT ACUTE VASCULAR INSULT AND RIGHT VERTEBRAL MURAL THROMBUS**

A. Axial angioCT: the right vertebral artery (yellow arrow) shows a mural thrombus restricted to the upper cervical segment with segmental stenosis below 40% (ECST); B: Axial CT (red arrow): hypodense area in the right cerebellar hemisphere, compatible with acute/subacute ischemic vascular insult.

**FIGURE 2. MRI DEMONSTRATING ACUTE VASCULAR INSULT.**

MRI in DWI axial sequence showing a hypersignal area (yellow arrow) with corresponding low signal on the ADC map (red arrow), in figure B. Together with the clinical data, an area with diffusion restriction due to acute/subacute ischemic vascular insult.

**Treatment**

In the same day of the appointment, a therapy plan was created involving anticoagulant Apixabana 5mg, 12/12h, for 3 months, until the condition was reevaluated with a new Angio CT and MRI. Furthermore, he should be removed from work for a month and was forbidden from practicing intense physical exercises for 3 months.
Results and follow-up

Due to delays in the results of the AngioCT and in scheduling a new medical appointment, the patient was advised to stay on the medication until the new appointment with the doctor, which happened 4 months later. On this occasion, after evaluating the new AngioCT and MR, which showed no changes, and given the stability of the case, medication was suspended.

In addition, the patient tried to follow the recommendation to resume the practice of physical activities 3 months after the first consultation, but when he tried to exercise, he had mild neck pain. Thus, he preferred to suspend activities until the return visit, a month later, during which a correlation between a new VAD was discarded, which cleared the patient to practice exercise provided no high impact was involved.

After 4 months of treatment, the patient was clinically free from the condition and was instructed only to maintain weight control. Since then, he reported having two new mild attacks of rhinosinusitis, but these did not cause cerebellar symptoms as before. Thereby, the patient was cured and has been neurologically stable for 9 months of follow-up up until now.

DISCUSSION

The reported case shows a Cerebellar Infarction secondary to VAD. Probably, the thrombus that caused the ischemia was formed after an injury to the intimal layer of the artery, caused by ricocheting of the head during an intense sneezing attack. This theory is supported by evidence that proves that the stretching and exaggerated retraction of the neck may be responsible for the damage of vessels in this region.

A literature review on the topic indicated that some of the most common signs and symptoms in VAD are nystagmus, cerebellar ataxia, vertigo, tinnitus, nausea and vomiting, neck pain, paresis, headache, and, more rarely, dysphagia and dysarthria. In this specific case, the initial condition had nonspecific symptoms such as pain in the neck; however, it evolved with neurological impairment, such as nystagmus, vertigo, and left temporal headache.

The diagnosis of VAD associated with cerebellar infarction requires good anamnesis and follow-up with imaging exams. Although arteriography is considered the “gold standard” for diagnosing VAD, since it is an invasive method, it is generally replaced by MRI and CT, which allow the analysis of the main cervical and intracranial arteries.

In the case reported, although the physical examination was normal, due to the report of nystagmus, headache, and vertigo, the diagnostic hypothesis of a tumor was raised and an MRI was requested. The exam revealed a region compatible with ischemic vascular insult, so AngioCT and Transcranial Doppler were indicated for better evaluation. Such tests identified thrombus and alteration in the flow of the vertebral artery, confirming that it was a case of stroke due to VAD, and not a neoplasm as previously thought.

Therapeutic intervention in cases of VAD consists mainly of administrating anticoagulants and antiplatelet agents and should be started as quickly as possible. Evidence on the topic suggests that there is no significant difference between the results of using these two drugs, but many prefer to use antiplatelet drugs because they have a lower risk of bleeding than anticoagulants. When these drugs are not effective, alternative interventions such as reperfusion, intervention in embolism, or surgery can be performed.

For our patient, specifically, anticoagulants were chosen because it is a more conservative approach. The patient reacted well to the adopted therapy and had no complications, so there was no need to try another approach. It is worth mentioning that there was no need for surgery, considering that the area of the vertebral artery that underwent dissection was extracranial, and had no tendency to increase.

The control exams, AngioCT and MRI, 4 months after the event, showed that the penumbra area in the cerebellum was well supplied by the collateral branches, and there was complete resolution of the previous condition (Fig. 3).

FIGURE 3. MRI AND CT CONTROL IMAGES

A: MRI in DWI axial sequence showing a low signal area (yellow arrow); there is no diffusion restriction in this image;
B: CT showing hypodense area with a density similar to CSF (red arrow), indicating previous vascular insult. Orange arrow indicating deviated septum, a potential cause of chronic rhinosinusitis.
CONCLUSION

This report presented a rare and well-documented association between sneezing and cerebellar infarction caused by VAD, highlighting its characteristic manifestations and the measures to be taken in order to assist doctors in quickly identifying and treating this unusual condition, avoiding possible complications and irreversible aftereffects.

Author’s Contribution

GBC, MAR e CACSJ: Participated in the study conceptualization, report organization, literature review, and final revision.

ALMN, ACQ, ALSM e DGPLR: Transcribed the report, organized the text, participated in the literature review, and text drafting.

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RESUMO

A Dissecção da Artéria Vertebral (DAV) é quadro raro que pode ser causado por movimentação de grande amplitude do pescoço, que lesiona a parede desse vaso, podendo provocar isquemia no cerebelo. Apresentamos um homem de 37 anos, com hérnia de disco e rinossinusite alérgica que lhe causava crises de espirros em salva (CE). Após uma dessas crises com ricocheteamento da cabeça, apresentou desalinhamento de C3 com dor local. Vinte e um dias depois, acometido por nova crise, apresentou cefaleia temporal esquerda, nistagmo e vertigem. Decorridos 3 dias, o paciente foi submetido a Ressonância Magnética (RM), que identificou 2 regiões de isquemia cerebelar e enchimento comprometido da artéria vertebral direita. Após 2 dias, foram feitos Angiotomografia Computadorizada (Angio TC), que constatou DAV direita com trombo local, sem sinais aneurismáticos, e Doppler Transcraniano, que não indicou aumento do fluxo sanguíneo dessa artéria. O tratamento sugerido envolvia administração de anticoagulante Apixabana 5mg, 12/12h, por 3 meses, até que o quadro fosse reavaliado com novas Angio TC e RM. Foi recomendado que o paciente ficasse afastado do trabalho por 1 mês e de exercícios físicos intensos por 3 meses, porém devido a contratempos, esses prazos foram prorrogados até nova consulta, 4 meses após a primeira. Os novos exames não apresentaram alterações, indicando que o quadro estava estável. Esse caso tem como objetivo indicar as possíveis investigações do diagnóstico e opções terapêuticas da rara associação entre DAV com infarto cerebelar em caso bem documentado.

PALAVRAS-CHAVE: Dissecção da artéria vertebral. Infarto Cerebral. Acidente vascular cerebral.