Vertebral artery dissection as a rare cause of vertigo: case report

Dissezione dell'arteria vertebrale come causa rara di vertigine: presentazione di un caso clinico

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Key words

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Parole chiave

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Summary

Vertebral artery dissection is one of the more frequent cerebral-vascular disorders in the young adult. The initial symptoms rarely consist of vertigo with clinical characteristics of Selective Monolateral Acute Vestibular Deficit Syndrome. The case is described of a patient, who arrived with intense rotatory vertigo associated with neurovegetative symptoms and spontaneous nystagmus, which we initially diagnosed as right neuronitis. About 48 hours later, the symptoms of vertigo disappeared spontaneously, and prevalently nuchal cephalea appeared. Since the symptoms were atypical and the otoneurologic study revealed normal canalar and otholithic function, a cerebral nuclear magnetic resonance, with contrast, was carried out which showed the presence of multiple areas of cerebellar ischaemia, prevalently on the left, and at the level of the right occipital lobe. Study of the patient was completed with an intracranial angio-nuclear magnetic resonance of the neck arteries and cerebral angiography the findings of which were compatible with left vertebral artery dissection. It is important to emphasize, as reported in the literature, that in cases in which atypical evolution of the pathology appears, or instrumental data do not confirm initial suspicions, a more scrupulous study is always necessary in order to find a possible central cause. Among the central causes, it should not be forgotten that multiple small cerebellar strokes (more frequent in elderly patients) and even more rarely also vertebral artery dissection (which is typical of younger patients) may become evident in a clinical picture that is almost identical to that seen in selective monolateral acute vestibular deficit syndrome.

Riassunto

La dissezione dell'arteria vertebrale (VAD) è una delle cause più frequenti di patologia cerebrovascolare nel giovane adulto. Raramente la sintomatologia di esordio è quella di una sindrome vertiginosa con caratteristiche cliniche di sindrome da deficit vestibolare acuto selettivo monolaterale (SDVASM). Descriviamo il caso di un paziente, giunto con un'intensa vertigine rotatoria associata a sintomatologia neurovegetativa e nistagmo spontaneo, in cui inizialmente ponemmo il sospetto diagnostico di neuronite destra. Dopo circa 48 ore la sintomatologia vertiginosa si risolse spontaneamente e comparse una cefalea prevalentemente nucale. Poiché la sintomatologia era atipica e lo studio otoneurologico rivelò che la funzionalità canalare e otolotica risultavano nella norma, si eseguì una RMN cerebrale con contrasto che rivelò la presenza di aree ischemiche multiple a livello cerebellare, prevalentemente a sinistra e a livello del lobo occipitale di destra. Lo studio venne completato con angioRMN intracranica, angioRMN dei vasi del collo e angiografia cerebrale che evidenziarono dei reperti compatibili con dissezione dell'arteria vertebrale sinistra. Ci è sembrato importante sottolineare, in accordo con la letteratura, che nei casi in cui compaiono delle atipie nell'evoluzione della patologia o i dati strumentali non confermano i sospetti iniziali è sempre necessario approfondire le indagini in ordine ad una possibile causa centrale; tra le cause centrali bisognerebbe ricordare che piccoli infarti cerebellari multipli, più frequenti nell'anziano, e più raramente anche la VAD, tipica del giovane, possono presentarsi con un quadro clinico sovrapponibile a quello di una SDVASM.

Introduction

Vertebral artery dissection (VAD) is one of the more frequent causes of cerebral or cerebellar ischaemia in young adults. Together with the dissection of the internal carotid artery, it represents 20% of all cerebrovascular disorders [1]. The average age for its development is about 40 years old. The cause is unknown and in the majority of cases is spontaneous [2]. Onset has been described, following cervical trauma or after a rapid neck-movement [1 3], chiropractic adjustment [4], or swimming. It can arise from acute subarachnoid haemorrhage, as well as cerebral, or more frequently cerebellar [1-6], ischaemia, or in spinal manifestations [7]. The symptoms can also develop more gradually, in most cases with cephalea, prevalently with migraines or intense nuchal pain, neurological focal deficits, vertigo and oscillopsy [8]. Angiography, and, more recently, angio-magnetic resonance (MR), are essential examinations if a diagnosis of VAD is

suspected. The pathognomic signs can be irregular restrictions of the lumen with aneurysmatic dilatation; the appearance of a double lumen is typical but not usual [9]. The treatment of choice is anticoagulant therapy with a 70% chance of healing having been reported in the literature [3].
In a few rare cases, described in the medical literature, vertigo, with the clinical characteristics of Selective Monolateral Acute Vestibular Deficit Syndrome (SDVASM), presents as the only initial sign of VAD [3 9].

Case report

In November of 2001, a 32-year-old male patient came to our attention. with symptoms of rotatory vertigo and intense neurovegetative symptoms, all of which appeared within 24 hours of a flu episode.
The patient was hospitalised in our unit with a suspected diagnosis of right neuritis due to the presence of a harmonic vestibular syndrome with spontaneous nystagmus type II towards the left and asymmetric deviation towards the right. The neurological examination resulted negative. The remote pathologic case history was negative. The patient was a truck driver and often loaded heavy weights which he carried on his left shoulder.
He underwent the standard tests used for acute vertigo syndromes. The audiometric tonal examination showed a mild perceptive hypoacusis limited to 6 KHz. Timpanometry and auditory brainstem response were within normal limits. The Vestibular Evoked Myogenic Potentials (VEMPS) were present on both sides. Electronystagmography (ENG) revealed a bilateral normoreflectivity to bithermic caloric stimulation (test). Stabilometry was within normal limits.
Approximately 48 hours after hospitalization, the symptoms of vertigo spontaneously disappeared, as well as the signs of vestibular decompensation, and a nuchal headache appeared. Since the symptoms were atypical and the canalar and otolithic functions were normal, a cerebral nuclear magnetic resonance (NMR) with contrast was performed. This revealed the presence of multiple areas of cerebellar ischaemia, prevalently on the left side, as well as at the level of the right occipital lobe. The patient was then transferred to the Neurology Department, where an intra-cranial angio-NMR was performed, which revealed a left vertebral artery slightly inferior in calibre, especially in relation to the subarachnoid distal portion. The right vertebral artery, the basilar artery, and the posterior cerebral arteries were all regular. An angio-NMR of the neck arteries showed that the left vertebral artery presented a homogeneous 15 mm reduction in calibre; partial parietal thickening also seemed to be present. Cerebral angiography showed marked changes in calibre of the left vertebral artery

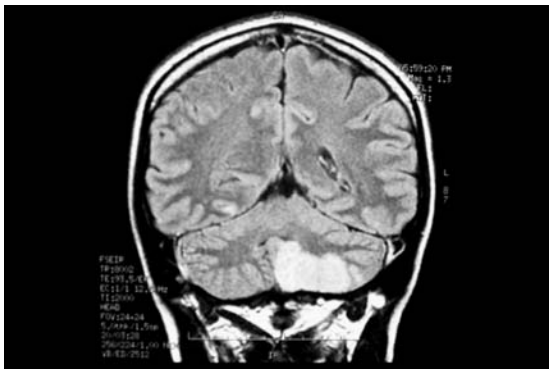

Fig. 1. Cerebral NMR: ischaemic areas at cerebellar level (coronal projection).

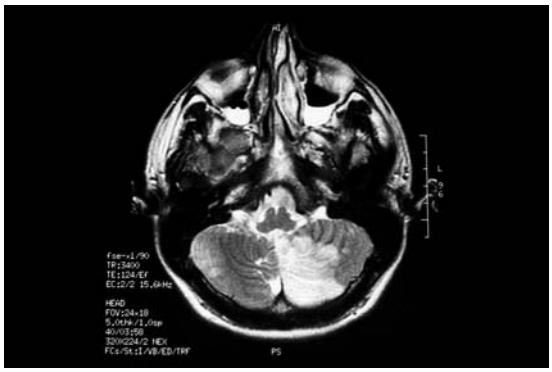

Fig. 2. Cerebral NMR: ischaemic areas at cerebellar level (axial projection).

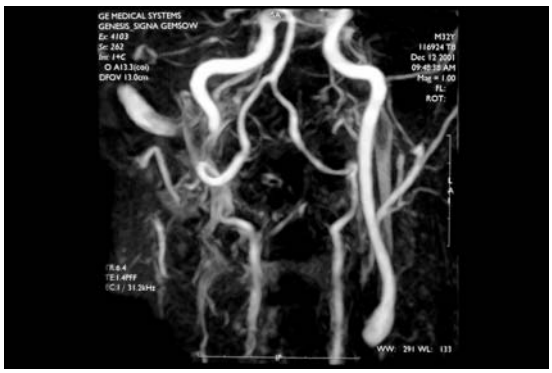

Fig. 3. Cerebral angio-NMR: left vertebral artery, reduced caliber.

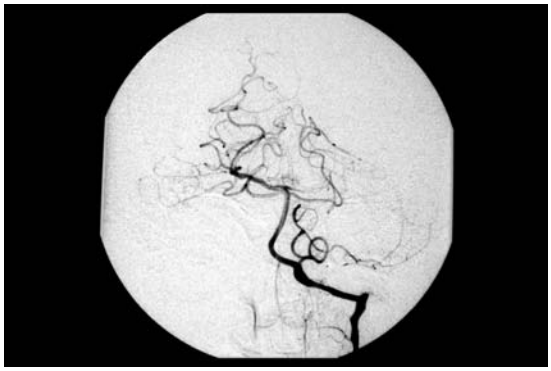

Fig. 4. Cerebral angiography: marked alterations of calibre of left vertebral artery in extra-cranial tract before PICA artery origin that appears scarcely represented in distal branches.

in the extracranial and the distal intracranial tract, before the origin of the postero-inferior cerebellar artery (PICA) artery that appeared poorly represented in the distal branches; these findings were compatible with left VAD. We proposed treatment with anticoagulants which, however, the patient refused due to the fact that he had no symptoms.

Discussion

A problem often facing the ORL specialist, in the case of vertigo, is that of distinguishing a central pathogenesis from a peripheral disorder. In the case described here, the patient came to our attention with initial problems that led us towards a diagnosis of possible vestibular neuronitis. The patient had had a recent episode of flu, an intense rotary vertigo and typical nystagmus that we had interpreted as a hypofunction of the right labyrinth. The following evolution of the symptoms and otoneurological studies showed later atypical results suggesting a possible central cause of the condition. The atypical results consisted of the rapid resolution of the symptoms, with the disappearance of nystagmus, the development of cephalea, the absence of canalar function deficit to caloric and otolithic stimulation (VEMPS within the normal range) and the normal stabilometry.

According to the literature [10], in those cases presenting an atypical evolution of the condition or when instrumental data do not confirm our suspicions, it is necessary to seek a possible central cause.

Vicini et al. [11] have recently published a revision of the literature on "so-called vestibular neuritis" in which they outline the diagnostic criteria for SD-VSAM, pointing out that the presence of a significant deficit in unilateral canalar function, transient or persistent, is essential for this diagnosis.

Among the central causes to be taken into consideration in the differential diagnosis, spontaneous vertebral artery dissection, albeit rare, is a pathological condition which is typically found in the age range of our patient (30-50 years). In fact, 68% of post-traumatic VADs, and 80% of those of spontaneous appearance are found among patients within this age group [3]. The sudden appearance of headache and occipital pain often represent the initial symptoms. In this case, the more likely aetiology is post-traumatic, since the day before the development of symptoms the patient had repeatedly loaded a heavy weight on his neck and left shoulder.

There are few cases described in the medical literature in which VAD had a similar onset. Braverman et al. [9], in 1999, described a case in which vertigo was the only symptom and they described it as a singular case. Strome et al. [3], in 1997, report that the initial symptom of VAD was cocleo-vestibulopathy, and, also in this case, cervical pain was present as well as the recollection of a recent trauma (head injury while playing soccer). In the cases more frequently found in the medical literature, small cerebellar strokes are often described in the areas of the PICA and the antero-inferior cerebellar artery (AICA), generally with a benign evolution, which revealed clinical signs that are very similar to those of monolateral vestibular peripheral deficit [10]. Braverman et al. [9] hypothesized that the development of symptoms of vertigo could be connected to ischaemic damage at the level of the vestibular nuclei due to the fact that, since the cochlea is more sensitive than the posterior vestibule to ischaemic damage, if there were selective labyrinthis damage, one would also expect cochlear damage. According to Strome et al. cochlear-vestibular symptoms are a consequence of AICA occlusion; the lack of other neurological alterations should show the presence of collateral flow through the PICA and the branches of the superior cerebellar artery.

In conclusion, in our opinion, it is important to emphasize how, in the clinical setting compatible with syndromes of monolateral periphery deficit, central motivations must also be taken into consideration in the differential diagnosis. In-depth studies, with neuroradiological research, should be carried out when the clinical trend of signs and symptoms shows any atypical aspects, i.e., different from what might have been expected. An analogous approach should be adopted when instrumental vestibular studies do not provide data confirming the diagnosis initially suspected. The onset of nuchal cephalea and related disorders together with eventual cervical damage, should suggest a vertebral VAD.

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