



Case Report

A Rare Association of Brain Arteriovenous Malformation and no Decussating Corticospinal Tracts Presenting as Ipsilateral Hemiparesis - A Case Report

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Citation: Campos CMS, Teixeira RRC, Marussi VHR, Yonekura BS, Neto MR, et al. (2022) A Rare Association of Brain Arteriovenous Malformation and no Decussating Corticospinal Tracts Presenting as Ipsilateral Hemiparesis - A Case Report. J Surg 7: 1643. DOI: 10.29011/2575-9760.001643

Received Date: 22 November, 2022; **Accepted Date:** 25 November, 2022; **Published Date:** 28 November, 2022

Abstract

Background: Brain Arteriovenous Malformation (AVMs) are rare disorders and an important cause of morbidity and mortality. The treatment of brain AVMs is complex, most of the times consisting of a multidisciplinary approach. The topography of the lesion, as well as the involvement of motor tracts are crucial to the treatment planning and follow up. The lack of corticospinal tract decussation has few descriptions on the medical literature, and the association with concomitant brain AVM has not previously been described on the medical literature until the date, and the recognition of this disorder is important for better management of these complex conditions.

Case Presentation: We present a case of a 53 years old woman complaining of headache and visual symptoms due to a right parietal brain arteriovenous malformation. The large nidus size associated with high-flow arteriovenous fistula, creating a complex angioarchitecture, as well as eloquent localization, were key points to apply a multidisciplinary approach to the treatment of this patient with a combination of endovascular plus surgery. She was first submitted to three sessions of embolization, however in the immediate post-embolization after the third session she presented ipsilateral hemiparesis (right side). A magnetic resonance imaging done a couple of hours after the clinical manifestation onset revealed cytotoxic edema, with areas of restricted diffusion surrounding the nidus malformation, suggestive of acute ischemic event affecting the right motor tract. The tractography study performed at the same time revealed a lack of corticospinal tract decussation.

Conclusion: An extremely rare association in clinical practice of brain arteriovenous malformations with concomitant lack of corticospinal tract decussation, highlights the possibility of occurrence, allowing the understanding the pathophysiology of these phenomena.

Keywords: Brain arteriovenous malformation; Case report; Corticospinal tracts; Interventional neuroradiology

Introduction

Brain Arteriovenous Malformations (AVM) are vascular lesions consisting of a direct arterial to venous communication in the brain circulation, associated with a vascular nidus in between, as a classic angioarchitecture feature. They are rare and usually single. The incidence of brain AVMs is rising, possibly due to the development of new radiology techniques that help identify these lesions [1]. The clinical presentation of these vascular malformations is heterogeneous, some of them are asymptomatic, but they may be associated with seizures, hemorrhage, focal neurologic deficits or headache [2]. Most of the brain AVMs are considered congenital, but there are some reports of acquired lesions [3]. The management usually involves neurosurgery, embolization, radiotherapy and some of the patients may have benefits in a conservatory treatment, mainly those with low risk of rupture or topography, for example specific brain stem AVM topography[2]. Abnormalities of the pyramidal tract decussation are rare, and for our knowledge, no case reports in the literature have been published so far, with the association of lack of corticospinal tract decussation and brain arteriovenous malformations.

Case Report

A previously healthy 53 years old woman presented to neurological evaluation due to a new onset of holocranial headache and visual disturbance. In the complementary investigation, she was diagnosed with a parieto occipital arteriovenous malformation (Figure 1). She had no familial history of neurological disorders.

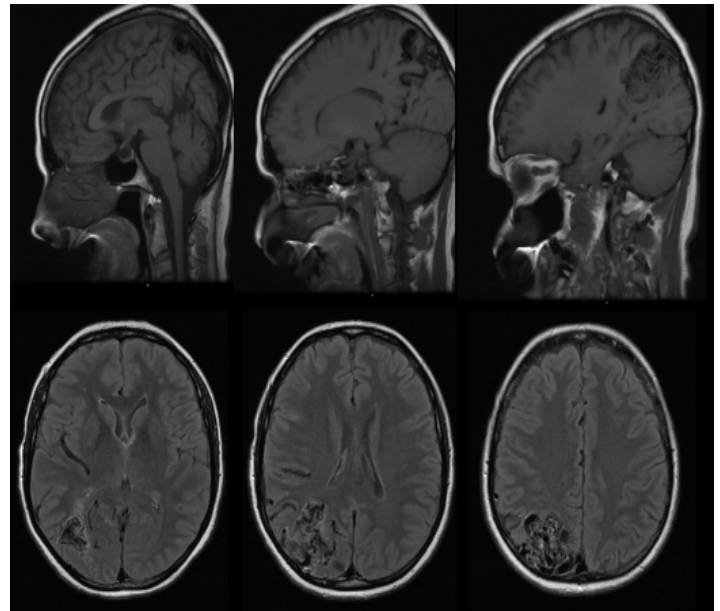


Figure 1: Brain magnetic resonance imaging showing an arteriovenous malformation in the right parietal lobe, with the nidus occupying the intraparietal sulcus and the adjacent white matter. It is associated with hypertrophy of middle and posterior cerebral arteries branches ipsilateral to the lesion. The main venous drainage of the malformation is carried out to the superficial venous system.

The complex arteriovenous malformation demanded a multidisciplinary strategy, with the initial planning of interventional neuroradiology embolizations. She was submitted to two sessions of embolization, but immediately after the third embolization the patient complained of loss of motor strength in the right hemicorpus, ipsilateral to the lesion. Normally, the deficit would be contralateral to the lesion due to the corticospinal tract decussation. To help understand this clinical presentation, she was submitted to an urgent magnetic resonance imaging with tractography (Figures 2,3).

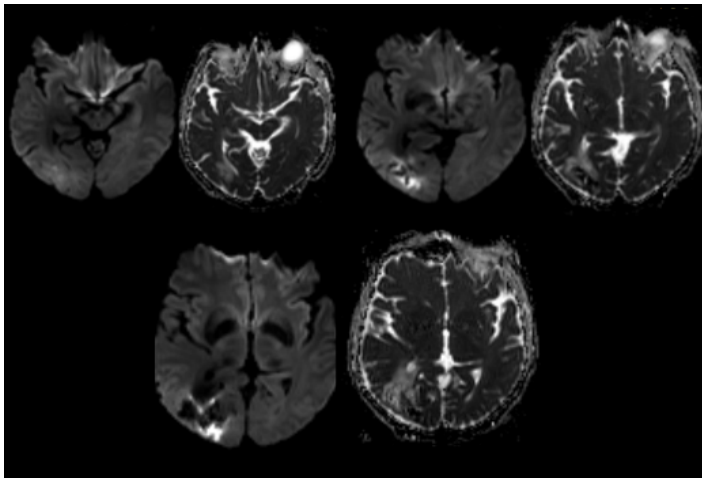


Figure 2: Magnetic resonance imaging showing restricted diffusion in the right occipital and parietal lobe, compatible with recent ischemia.

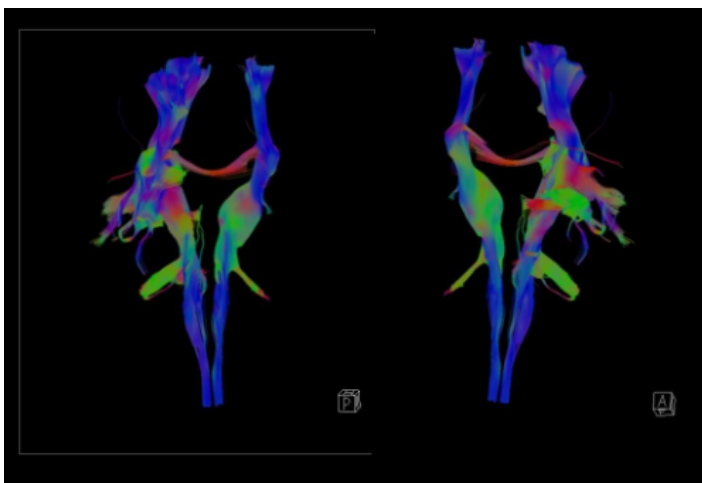


Figure 3: Magnetic resonance tractography showing lack of corticospinal tract decussation

She was submitted to a supportive therapy with neurointensive care and blood pressure management, with improvement of the motor deficits. She remained with no new clinical deficits until the date, and the brain AVM is stable.

Discussion

The pathophysiology of brain AVMs is complex, and many of them are congenital in origin. Some of them have complex genetic features associated, like in Osler-Weber-Rendu, Wyburn Mason [2], and Cowden syndrome [4]. The majority of the brain AVMs cases, however, are not associated with these syndromes and are usually single, like in our case. The choice of the intervention involves many variables, many of them based on the Spetzler-

Martin grading score, that accounts for the AVM size, pattern of venous drainage and eloquence, described initially in 1986 [5] and further improved. The eloquence, so, in our case, was influenced by the involvement of the right corticospinal tract due to the lack of corticospinal decussation. The medical literature show correlations of corticospinal decussation absence in medical disorders like mirror movements, Dandy Walker syndrome and Joubert syndrome [6-8], for example, but literature correlating abnormalities of corticospinal tract and cerebrovascular malformation is lacking. The pattern of motor connections involved by the vascular abnormality is important in treatment planning, because it may influence the eloquence. The genetic basis of the AVMs and corticospinal tract abnormalities are yet in study, and for now we found no information in medical literature showing a genetic basis that connects both disorders.

Conclusion

We present a rare association of brain arteriovenous malformation and lack of corticospinal tract decussation that may contribute to the genetic basis of both diseases and for further identification of new correlations between the two disorders, which may provide a better treatment decision and follow up of these complex patients.

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