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ORIGINAL ARTICLE

Spinal arteriovenous fistulas in adults: management of a series of patients treated at a Neurology department☆

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KEYWORDS
Spinal arteriovenous fistula;
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Spinal vascular disorder;
Myelopathy;
Spinal angiography

Abstract
Objective: Spinal arteriovenous fistulas (SAVFs), a rare type of vascular malformation, account for 3% of all spinal cord lesions. Without early treatment, the associated morbidity is high; furthermore, SAVFs pose a major diagnostic challenge. Our purpose was to evaluate the clinical characteristics of SAVFs and review their progress after treatment to determine whether they may be too late for treatment in some cases.
Methods: We present a retrospective series of 10 patients diagnosed with SAVFs and treated at a tertiary hospital during a 3-year period.
Results: In our sample, SAVFs were found to be significantly more frequent in men (80%). Mean age in our sample was 65.4 years. The most common initial symptom was intermittent claudication/paraparesis (70%). In most patients, symptoms appeared slowly and progressively. At the time of diagnosis, the most common symptoms were motor, sensory, and sphincter disorders. Mean time from symptom onset to diagnosis was 24.3 months. Initial diagnosis was erroneous in 60% of the patients. Spinal magnetic resonance imaging was diagnostic in 90% of these cases and arteriography in 100%. The most common location of the fistula was the lower thoracic region and the most frequent type was dural (seven cases). All patients were treated with embolisation, surgery, or both, and 70% improved after fistula closure regardless of progression time.

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Conclusions: Diagnosis of SAVFs is difficult and often delayed, which leads to poorer patient prognosis. We should have a high level of suspicion for SAVFs in patients with intermittent claudication or paraparesis exacerbated by exercise. Early treatment should be started in these patients. Treatment should always aim to improve the quality of life or stabilise symptoms, regardless of progression time.
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PALABRAS CLAVE
Fístula arteriovenosa espinal;
Fístula arteriovenosa dural espinal;
Malformación vascular espinal;
Enfermedad vascular espinal;
Mielopatía;
Arteriografía espinal

Fístulas arteriovenosas espinales del adulto. Manejo de una serie de casos desde una planta de Neurología

Resumen
Objetivo: Las fístulas arteriovenosas espinales (FAVE) son excepcionales y representan el 3% de las lesiones espinales. Asocian gran morbilidad sin tratamiento precoz, pero el diagnóstico constituye un reto. Nuestro objetivo es evaluar sus características clínicas y revisar la evolución tras el tratamiento. ¿Puede ser tarde para tratar?
Métodos: Presentamos una serie retrospectiva de 10 casos diagnosticados y tratados en 3 años en un hospital terciario.
Resultados: Se observó un predominio masculino (80%). La edad media fue de 65,4 años. El síntoma inicial predominante fue la claudicación de la marcha/paraparesia (70%). En la mayoría de los pacientes la clínica fue lentamente progresiva. Al diagnóstico, lo habitual fue la combinación de síntomas motores, sensitivos y esfinterianos. El tiempo medio desde el inicio de los síntomas hasta el diagnóstico fue de 24,3 meses. El 60% tenía un diagnóstico inicial erróneo. La RM espinal fue diagnóstica en el 90% de los casos; la arteriografía, en el 100%. La localización más frecuente fue dorsal baja y el tipo anatómico predominante fue FAVE dural (7 pacientes). Todas fueron tratadas con embolización, cirugía o con ambas y el 70% mejoró tras su cierre, independientemente del tiempo de evolución.
Conclusiones: El diagnóstico de las FAVE es difícil y generalmente tardío, lo que empeora el pronóstico de los pacientes. Se debe tener un alto nivel de sospecha ante síntomas de mielopatía o claudicación de la marcha exacerbadas con el ejercicio e intentar tratamiento precoz. Consideramos que el tratamiento siempre está indicado, independientemente del tiempo de evolución, al mejorar la calidad de vida o conseguir la estabilización.
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Introduction

Spinal arteriovenous fistulas (AVFs) are extremely rare. Despite accounting for 70% of all vascular malformations of the spinal cord, they represent only 3% of all spinal cord lesions.[1,2] Spinal AVFs are thought to be acquired lesions and rarely present in individuals younger than 50 years, although the exact aetiology is yet to be determined. From a pathophysiological viewpoint, an AVF is an abnormal, low-flow connection, or shunt, between an artery and a vein, and is associated with potentially severe complications. Spinal dural AVFs (also known as thoracic intradural AVFs) constitute the most frequent subtype; in these, the shunt is located in the trajectory of the corresponding nerve root, within the dura mater, and is fed by radiculomeningeal arteries draining centripetally via a radicular vein into perimedullary veins. The other two subtypes, spinal pial AVFs (ventral intradural or perimedullary) and spinal epidural AVFs (extradural), are less frequent; these are supplied by a radiculomedullary or a segmental artery, respectively.[3,4]

Spinal dural and pial AVFs drain into perimedullary veins, leading to venous hypertension and dysregulation of spinal blood vessel flow, which results in blood—brain barrier disruption and progressive spinal cord oedema.[5] Over time, the arteriovenous pressure gradient decreases, leading to spinal cord ischaemia and consequently to progressive myelopathy; the reversibility of the latter depends on progression time. The pathogenic mechanism of these shunts is yet to be understood. It has been hypothesised that progressive fibrosis or thrombosis of radicular veins (due to advanced age and accelerated by the presence of a shunt), acting until then as alternative draining routes from the arterialised perimedullary veins, may play a relevant role in the pathogenesis of hypertension as a result of the decreased outflow.[6,7] Given that the thoracic and lumbar regions of the spinal cord have fewer venous drainage channels than other spinal regions, venous congestion is transmitted caudocranially along the spinal cord, which may explain why the first symptoms usually reflect dysfunction of the conus medullaris.[8] These include slowly progressive non-specific

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