Arterial Hypertension Is Associated with Symptomatic Spinal Dural Arteriovenous Fistulas

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■ OBJECTIVE: **To determine possible systemic factors that may induce or be associated with the pathogenesis and pathologic course of spinal dural arteriovenous fistulas (SDAVFs), the most common type of arteriovenous disorder of the spinal cord and its meninges.**

■ METHODS: **We assessed the role of possible systemic (vascular) risk factors (arterial hypertension, diabetes mellitus, fat metabolism disorders, and nicotine dependence) by comparing the prevalence of these risk factors in an SDAVF cohort of 59 patients with the prevalence in the general population.**

■ RESULTS: **Age-corrected prevalence of arterial hypertension in the SDAVF cohort was significantly higher than in the general population ($P < 0.001$). Prevalence of diabetes mellitus ($P = 0.150$.), nicotine dependence ($P = 0.561$), adiposity ($P = 0.217$), and fat metabolism disorders ($P = 0.125$) did not differ from prevalence of comparable cohorts in the general population.**

■ CONCLUSIONS: **Our results and data from the literature suggest that arterial hypertension may play an important role in the development of SDAVF-related symptoms or the development of SDAVFs in the presence of other predisposing factors.**

INTRODUCTION

Spinal dural arteriovenous fistulas (SDAVFs) are the most common type of arteriovenous (AV) disorder involving the spinal cord and its meninges and account for 70% of all spinal AV shunts.[1,2] Clinical and radiologic characteristics of SDAVFs and treatment strategies have been the subject of various studies in the last 2 decades.[1,3] Nonetheless, the pathophysiology, in particular, the etiology, of SDAVFs is not yet fully understood.[4-10] It is currently assumed that SDAVFs are acquired lesions.[3] However, clinical factors that may induce or be associated with SDAVFs are currently unknown. The purpose of our study is to determine possible systemic factors that may induce or be associated with the pathogenesis and the pathologic course of SDAVFs.

MATERIALS AND METHODS

After obtaining permission from the local ethics board, we retrospectively searched our medical database for all patients who underwent spinal digital subtraction angiography in our institution between 2006 and 2016 and identified 59 consecutive patients with angiographically verified SDAVFs. The initial diagnosis of SDAVF was based on clinical and radiologic criteria that comprised 1) clinical sequelae of myelopathy resulting in motor or sensory disturbances with or without vegetative bladder/bowel dysfunctions and 2) magnetic resonance imaging findings of congestive myelopathy of the spinal cord and/or visibly engorged perimedullary veins. All suspected cases of SDAVF were verified by digital subtraction angiography. All clinical data, including demographics, clinical and neurologic presentation, comorbidities, and premedication, and radiologic data were assessed by the primary treating physicians and reevaluated for this study. To assess the role of possible systemic (vascular) risk factors, we compared the prevalence of cardiovascular diseases, including arterial hypertension, diabetes mellitus, fat metabolism disorders, and nicotine dependence, in our SDAVF cohort with the comparable prevalence in the general population in Germany. The investigated risk factors were defined according to respective societies.[11,12]

Key words
- Arterial hypertension
- Congestive myelopathy
- Risk factors
- Spinal arteriovenous malformation

Abbreviations and Acronyms
AV: Arteriovenous
SDAVF: Spinal dural arteriovenous fistula

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Citation: World Neurosurg. (2017) 103:360-363.
http://dx.doi.org/10.1016/j.wneu.2017.04.050

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

Table 1. Vascular Risk Factors of Patients with Spinal Dural Arteriovenous Fistula

		Age Group (Years)							
Risk Factor	**Cohort**	**50–59**		**60–69**		**70–79**		**>80**	
Hypertension	General population	20%	$P < 0.001$*	25%	$P < 0.001$*	31%	$P < 0.001$*	32%	$P = 0.003$*
	SDAVF	64%		80%		76%		86%	
Diabetes mellitus	General population	7%	$P = 0.96$	14%	$P = 0.627$	19%		21%	$P = 0.173$
	SDAVF	7%		10%		6%		0%	
Adiposity	General population	18%	$P = 0.325$	21%	$P = 0.114$	20%	$P = 0.797$	16%	$P = 0.869$
	SDAVF	27%		35%		22%		14%	
Nicotine dependence	General population	24%	$P = 0.672$	14%	$P = 0.481$	7%	$P = 0.416$	3%	$P = 0.621$
	SDAVF	27%		20%		11%		0%	
Fat metabolism disorders	General population	35%	$P = 0.291$	48%	$P = 0.038$*	53%	$P = 0.141$	38%	$P = 0.012$*
	SDAVF	27%		25%		39%		86%	

Age-corrected prevalence of clinical (vascular) risk factors (50–59 years, $n = 14$; 60–69 years, $n = 20$; 70–79 years, $n = 17$; >80 years, $n = 7$). One 36-year-old patient with adiposity was excluded from this analysis to ensure comparability with the literature. *P* values were calculated with the original data provided by the Robert Koch Institute Department of Epidemiology and Health Monitoring.[11,12]

SDAVF, spinal dural arteriovenous fistula.

*Significant *P* value.

Statistical Analysis

We compared the prevalence of possible risk factors in our cohort and the general population using the Pearson χ^2 and Fisher exact tests depending on data distribution. *P* values ≤ 0.05 were defined as significant. All statistical analyses were performed with IBM SPSS Statistics for Windows Version 23 (IBM Corp., Armonk, New York, USA).

RESULTS

Mean age of all patients (N = 59) was 66.86 years ± 10.38 (median, 68 years; range, 36–85 years). There were 45 male patients (77%). All patients presented with various combinations of progressive neurologic deficits. There were 43 (69%) patients with paraparesis, 28 (47%) patients with ataxia, 26 (44%) patients with sensory disturbance, and 12 (20%) patients with bladder/bowel dysfunction. The average symptom duration before diagnosis was 20 months ± 23 (median, 12 months; range, 1–120 months). Fistula locations ranged from Co to S3. Most SDAVFs were located in the thoracolumbar region (n = 31).

Vascular risk factors in our cohort and the general population are compared in **Table 1**. The age-corrected prevalence of arterial hypertension in our cohort was consistently significantly higher than in the general population ($P < 0.001$).[12] The prevalence of diabetes mellitus ($P \geq 0.173$), nicotine dependence ($P \geq 0.416$), and adiposity ($P > 0.114$) did not differ from the prevalence of comparable cohorts in the general population, whereas the prevalence of fat metabolism disorders correlated inconsistently with prevalence in the general population ($P = 0.012–0.291$).[11,12]

DISCUSSION

The first studies dealing with SDAVFs were published by Kendall and Logue in 1977 and Merland et al. in 1980.[13] Although SDAVFs are the most common type of AV disorders of the spinal cord and its meninges, little is known about their etiology.[1,13] In contrast to SDAVFs, various theories about the natural history of intracranial dural AV fistulas have been established.[14-16] The presence of microscopic dural communications between arteries and veins at the proximity of cranial venous sinuses has been previously described in the literature.[17,18] It was hypothesized that venous sinus thrombosis with associated intravenous hypertension and venous outflow obstruction might initiate the opening of these channels and form AV shunts into the venous sinuses.[14,19] An additional angiogenesis, as a part of the inflammatory process that organizes and recanalizes the thrombosed sinuses, was considered to influence the progression of these fistulas.[19] Concerning SDAVFs, Merland et al.[20] assumed that an insufficient venous outlet of the spinal cord induced the development of these fistulas and their clinical symptoms.

It has been hypothesized that once a fistula is present, the progressive fibrosis or thrombosis of radicular venous outlets could reinforce the medullar venous hypertension due to a decreased venous drainage.[20,21] However, the relationship between an AV fistula of the spinal dura and meningeal venous thrombosis has never been proven.[13,14] In the past, the pathophysiologic focus in most publications dealing with SDAVFs has been on local changes.[5,6,22] It is generally assumed that anatomic predispositions and local hemodynamic changes favor the development of SDAVFs.[8] Accordingly, we hypothesized that systemic diseases that affect the vascular system may be associated with