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Frequency and characteristics associated with inherited thrombophilia in patients with intracranial dural arteriovenous fistula

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Abstract

Background—The pathogenesis of dural arteriovenous fistulas (DAVF) remains poorly defined. Prior studies on thrombophilia as a risk factor for DAVF development are limited by small sample sizes and poor generalizability.

Methods—In this prospective cohort study, all patients with intracranial DAVFs evaluated at the University of California, San Francisco from December 1994 through April 2014 were identified. Three thrombophilic mutations: factor V Leiden (rs6025), MTHFR (rs1801133) and prothrombin (G20210A) were genotyped in patients who consented for genetic studies. We evaluated the association of thrombophilia status (presence of any thrombophilic mutation) and clinical and angiographic characteristics using either a two-sample t-test or Fisher's exact test.

Results—We enrolled 116 patients with diagnosed intracranial DAVF. Twenty-five (22%) patients met criteria for thrombophilia. Focal neurological deficits tended to occur more frequently in the thrombophilia group (78% vs. 57%, p=0.09). DAVF angiographic characteristics, including high-risk venous flow pattern, multiplicity of DAVF, and the presence of venous sinus thrombosis, did not differ significantly between the two groups but tended to be more common in the thrombophilic compared to the non-thrombophilic group.

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Conclusions—This is one of the largest studies of thrombophilia and DAVF to date. The frequency of mutations associated with thrombophilia in this study was higher than that of the general population.

Keywords

Dural Arteriovenous Fistula; Thrombophilia; Factor V Leiden; Prothrombin; MTHFR

INTRODUCTION

Dural arteriovenous fistulas (DAVF) account for 10–15% of intracranial arteriovenous malformations.[7] DAVFs can present with a variety of symptoms, including headache, pulsatile tinnitus[31], cranial nerve palsies, ischemic deficits or life threatening intracranial hemorrhage.[22] While the disease course for some DAVFs may be benign or non-progressive, depending on patterns of venous drainage, location of the fistula, and amount of shunting, other DAVFs may become aggressive, resulting in morbidity or even mortality.[5,11,21,29]

Whether DAVFs are congenital or acquired remains controversial, however, the preponderance of evidence points toward DAVFs as likely acquired malformations[1,2,18,32]. Adult-type DAVFs typically present following a venous or venous sinus thrombosis, while congenital DAVFs are often found in childhood.[10] The etiology of DAVF development is likely multifactorial. The most common risk factors for DAVF formation include head trauma, dural venous sinus thrombosis, craniotomy, and intracranial meningioma.[1,3,13,33] Inherited thrombophilic factors may also play an important role in the pathogenesis of DAVFs.

There are several mutations that are considered thrombophilic and can lead to the development of venous clots, including venous sinus thrombosis.[23] These include factor V Leiden, prothrombin G20210A, MTHFR C677T, protein C and S deficiency and antithrombin deficiency, among others.[14] The contribution of prothrombotic states in DAVF development is limited to case reports and small studies, where the clinical significance is not always clear. The goals of this study were to investigate the association between inherited thrombophilia and intracranial DAVF, specifically as it relates to the factor V Leiden, prothrombin and MTHFR mutations. We also compared the clinical and angiographic characteristics of DAVFs in participants with and without thrombophilia.

MATERIALS AND METHODS

Participants

We identified all patients with DAVFs referred for diagnosis or treatment to the University of California, San Francisco (UCSF) from December 1994 through April 2014. Diagnosis of DAVF was made by angiography. All patients with DAVF treated by Neurointerventional Radiology (NIR) or Neurosurgery before December 1999 were entered into a database after chart review (V.S., S.H.). From January 2000 onwards, those presenting to NIR, Neurosurgery or Neurovascular services were enrolled prospectively. Only patients who consented for blood draw for thrombophilia gene mutation testing were included in this

study. Patients were considered to have thrombophilia if they met any of the following criteria: (1) two copies of the MTHFR rs1801133 minor allele, (2) the Prothrombin G20210A mutation, or (3) the Factor V Leiden rs6025 mutation. Patients with only one copy of the MTHFR minor allele were not classified with thrombophilia due to the unclear clinical significance of a heterozygous genotype.[35] Patient characteristics, clinical symptoms, and DAVF features were collected from medical records. Modified Rankin Scores (mRS) were also calculated.

Genotyping

Genomic DNA was extracted from blood or saliva using standard procedures. We genotyped three thrombophilia gene mutations: Factor V Leiden (rs6025), MTHFR C677T (rs1801133) and Prothrombin G20210A (rs1799963). Genotyping was performed in two different laboratories over the course of the study: primary UCSF Lab Medicine, Laboratory of Neurogenetics, and at the UCSF Center for Cerebrovascular Research (CCR). Genotyping in the CCR laboratory was performed using the template-directed dye-incorporation assay with fluorescence polarization detection and Taqman assays (Applied Biosystems, Foster City, CA) according to manufacturers' instructions.

Statistical Methods

We calculated the association between each variable and thrombophilia status using either a two-sample t-test or Fisher's exact test. We calculated 95% exact binomial confidence intervals for the proportion with thrombophilia and the proportions of each of the three genetic mutations. Data analysis was performed with Stata 13.1 (StataCorp. 2013. *Stata Statistical Software: Release 13*. College Station, TX: StataCorp LP).

RESULTS

We enrolled 116 patients with diagnosed DAVF by angiography during the study period. There were no differences in patient demographics between the thrombophilic and non-thrombophilic groups (Table 1). Similarly, clinical symptoms were not significantly different between the thrombophilic and non-thrombophilic groups. However, focal neurological deficits tended to occur more frequently in the thrombophilia group but did not reach statistical significance (78% vs. 57%, p=0.09). This difference became statistically significant if MTHFR heterozygotes were included in the thrombophilic group (72% vs. 49%, p=0.02).

In this cohort, 25 patients (22%; 95% CI 14%–30%) met criteria for thrombophilia (Table 2). Of these patients, 17 patients (15%; 95% CI 9%–22%) carried two copies of the MTHFR minor allele, 7 patients (6%; 95% CI 2%–12%) carried the Prothrombin G20210A mutation, and 3 patients (3%, 95% CI 1%–7%) carried the factor V Leiden mutation; 2 patients (2%) fulfilled both the MTHFR and Prothrombin criteria.

When stratifying our analysis by presence of venous sinus thrombosis, we found that 83 of the 116 patients (72%) had venous sinus thrombosis. The patients with venous sinus thrombosis included the majority of patients with thrombophilia mutations. Of the 83

patients with venous sinus thrombosis, 21 patients had at least one thrombophilia mutation (25.3%, 95% CI: 16.3–36.0%).

DAVF angiographic characteristics (Table 3), including venous flow pattern, location, multiplicity of DAVF, and presence of venous sinus thrombosis did not differ significantly between the two groups, but tended to be more common in the thrombophilic compared to the non-thrombophilic group.

We also compared modified Rankin Scores (mRS) across patients with thrombophilia and patients without. Here, mRS represents treatment outcome determined by the technical difficulty of DAVF treatment, not disability due to the DAVF directly. While there was no statistically significant difference between groups ($p = 0.56$), more patients without thrombophilia (29% versus 17%) received a mRS of 0, referring to absence of disability.

DISCUSSION

This is one of the largest studies of thrombophilia and DAVF to date. Of 116 patients diagnosed with DAVF, 25 (22%) patients met criteria for thrombophilia. The frequency of some mutations associated with thrombophilia in this study was higher than estimated in the general population, though not as high as rates seen in cerebral venous sinus thrombosis, a known risk factor for DAVF development.[23] The prevalence of the prothrombin G20210A gene mutation ranges from 0.7 to 4.0 percent, with a higher preponderance among Caucasian populations.[14] The prevalence of prothrombin G20210A was higher in our study (6%; 95% CI 2%–12%). One population study in Wisconsin calculated a prevalence of the MTHFR homozygous mutation to be approximately 8%,[25] though it may be present in as low as 1.4% of the population.[14] The frequency of MTHFR minor allele homozygotes was much higher than expected in our DAVF cohort (15%; 95% CI 9%–22%). The prevalence of factor V Leiden heterozygosity varies widely among populations and ranges from 1 to 8.5 percent,[14], and is greater among Caucasians than minority populations within the United States.[26] The prevalence of factor V Leiden heterozygosity was not elevated in our DAVF cohort (3%, 95% CI 1%–7%).

The contribution of prothrombotic states in DAVF development is limited to case reports and small studies. One of the initial investigations was led by Kraus and colleagues, who illustrated that the genetic procoagulant risk factors for venous thrombosis were also germane to DAVF.[15–17] In a 44-person case-control study, they found a significantly higher frequency of a mutation in the factor V Leiden gene, leading to activated protein C resistance, in patients with DAVF compared with those without.[17] Here, approximately 22% of patients with DAVF featured the factor V Leiden mutation, which was much larger than in our population. Similarly, aggregating 121 cases of DAVF and 178 control subjects, van Dijk et al. found significant associations between presence of factor V Leiden and prothrombin G20210A allele and the development of DAVF (odds ratios 4.69 and 10.87, respectfully).[34] Kraus and colleagues later expanded to additional thrombophilic mutations including prothrombin G20210A, MTHFR C677T, and others, but did not find a significantly increased prevalence of these risk factors in patients with DAVF.[16] Gerlach and colleagues studied 25 patients with intracranial DAVF and 18 patients with spinal DAVF to determine if

there were differences in thrombophilic risk factors for these two DAVF types.[8] Both groups featured a greater prevalence of the factor V Leiden G20210A mutation than the general population, and more patients with intracranial DAVF than with spinal DAVF carried this mutation.[8]

While thrombophilic genetic mutations may be associated with an increased risk of developing a DAVF, they are likely not sufficient for DAVF formation. The studies discussed here demonstrate that only a minority of patients with DAVF has thrombophilia. Most patients with hypercoagulable states will never be diagnosed with a DAVF. There are likely additional unknown risk factors that predispose people with thrombophilia to develop DAVF. Similar to the development of venous sinus thrombosis, these factors may include dehydration, concomitant illness or inflammatory states, or additional causes of prothrombotic states such as pregnancy, malignancy, or use of certain medications such as hormones.[6,27,28]

Testing for thrombophilic mutations remains controversial.[12,24,30] Common criticisms surrounding thrombophilia testing include the high cost of testing, low prevalence of procoagulant disorders, difficulty with test interpretation, and the uncertain clinical significance of positive results.[12,24] The detection of inherited coagulation disorders is clinically relevant for several reasons. The diagnosis of a thrombophilic mutation may provide insight into subsequent risk for recurrent events and choice of prevention strategies.[30] The patient can be educated on addressing predisposing factors for thromboembolism, such as hormonal medications, pregnancy, surgery, and extended travel.[20] Detection of these mutations may result in a more in depth assessment of concomitant traditional stroke factors, such as hypertension, diabetes, and hyperlipidemia, which can then be modified aggressively. Patients with prothrombin, factor V Leiden, or protein S and C mutations may be started on anticoagulation. Lastly, family members can be screened for inherited disorders, which may be of special importance to women of childbearing age.[4]

This study features several advantages. Our data are generalizable as our study population is ethnically diverse and includes participants from multiple regions within the United States due to our large referral catchment area. This also resulted in a much larger sample size than in many prior studies. We investigated three of the more common mutations associated with thrombophilia in order to maximize clinical saliency for this patient population. These mutations are also more encountered in patients with cerebral venous sinus thrombosis.

This study has several limitations. Our study period spanned multiple decades in order to maximize enrollment. The number of procedures per participant varied due to the differences among DAVF types. Not all clinical characteristics were known or recorded for each participant limited our ability to investigate associations. Despite the relatively large size of the cohort, it is still limited by small patient numbers. However, every patient who was approached for this study enrolled, which minimized sampling bias from the population.

Future directions for this research include increasing the number of thrombophilic mutations that are tested for, as well as expanding this study to a larger DAVF population.

CONCLUSION

Thrombophilic polymorphisms, including factor V Leiden, prothrombin and MTHFR mutations, are not uncommon in patients with DAVF. The frequency of mutations associated with thrombophilia were higher in patients with DAVF than in the general population. Other undiscovered genetic and environmental factors may also play a role in DAVF development and warrant investigation.

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Table 1

Clinical characteristics of patients with DAVF by thrombophilia status.

Clinical Characteristics	Thrombophilic (N = 25)	Not Thrombophilic (N = 91)	P-Value
Mean Age at Angiography in Years	56 ± 16	57 ± 15	0.69
Female	15 (60%)	52 (57%)	0.82
Race			0.18
Asian	2 (8%)	18/89 (20%)	
Black	0 (0%)	4/80 (4%)	
Hispanic	4 (16%)	9/89 (10%)	
Native American	1 (4%)	0/89 (0%)	
Caucasian	18 (72%)	58/89 (65%)	
History of head injury	3/21 (14%)	24/76 (32%)	0.17
History of pregnancy	4/14 (29%)	11/40 (28%)	1.00
Oral contraceptive use	4/4 (100%)	15/17 (88%)	1.00
Hormone replacement use	4/4 (100%)	13/23 (57%)	0.26
Family history of clotting disorder	1/21 (5%)	7/69 (10%)	0.68
Symptoms on Presentation			
[1]Neurological deficit	18/23 (78%)	43/75 (57%)	0.09
Hemorrhage	3 (12%)	13/89 (15%)	1.00
Seizure	4/19 (21%)	6/62 (10%)	0.23
Pulsatile tinnitus	18/23 (78%)	47/78 (60%)	0.14
Headache	16/20 (80%)	57/76 (75%)	0.77
Visual symptoms	6/18 (33%)	22/64 (34%)	1.00

[1]Neurological deficits include presence of hemiparesis, hemisensoryloss, and cranial neuropathies.

Values are mean ± standard deviation, n (%), or n/total (%) if missing data is present.

Table 2

Frequencies of thrombophilia and associated genetic mutations in DAVF cohort.

Mutation	[1]Total (%)	95% CI
Thrombophilia (Total)	25 (22%)	14% – 30%
Factor V Leiden (rs6025)	3 (3%)	1% – 7%
Prothrombin (G20210A)	7 (6%)	2% – 12%
MTHFR (rs1801133) homozygotes	17 (15%)	9% – 22%

[1]Two patients fulfilled both the MTHFR and Prothrombin criteria.

Abbreviations: CI – Confidence Interval.

Table 3

DAVF angiographic characteristics by thrombophilia status.

Angiographic Characteristics	Thrombophilic (N = 25)	Not Thrombophilic (N = 91)	P-Value
High risk venous flow	12 (48%)	29 (32)	0.16
Location of DAVF			0.80
Cavernous	4 (16%)	16 (18%)	
Ethmoidal	1 (4%)	4 (4%)	
Large Sinuses: Transverse, Sigmoid, Sagittal	17(68%)	52 (57%)	
Posterior Fossa: Spheno-palatine, Tentorial, Incisura	3 (12%)	29 (32%)	
Presence of multiple DAVF	7 (28%)	12 (13%)	0.12
Venous sinus thrombosis	21 (84%)	62 (68%)	0.14