Transverse–Sigmoid Sinus Dural Arteriovenous Fistulae

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Transverse–sigmoid sinus dural arteriovenous fistulae are abnormal arteriovenous communications within the dural wall of the transverse–sigmoid sinuses. They present with a variety of clinical features, ranging from benign bruits to intracranial hemorrhage and neurologic deficits. The presentation and natural history of these fistulae are largely determined by the pattern of venous drainage. Knowledge of natural history and careful study of the angioarchitecture by angiography is therefore mandatory for correct management of these lesions. In this review, anatomy and pathology, principles of management, and the various factors that influence treatment decisions are discussed, with a focus on endovascular therapy. Indications for endovascular treatment, therapeutic goals, approaches, and techniques are reviewed. The role of surgical treatment is also briefly discussed.

INTRODUCTION

Dural arteriovenous fistulae (DAVFs) consist of arteriovenous fistulae of blood confined within dural leaflets (51). Studies have shown the crude risk of hemorrhage for DAVF to be 2% per year (7). The most common locations of dural arteriovenous malformations (DAVMs) are the transverse and sigmoid venous sinuses (56). The endovascular cure rate of DAVF involving other locations (e.g., tentorium or cavernous sinus) has been dramatically improved up to 80%–100% in recent years (23, 36, 39). However, in the case of transverse–sigmoid sinus dural arteriovenous fistulae (TSDAVFs), the rate was lower than 40% because of its complex anatomic factors (35).

Key words
- Arteriovenous fistula
- Transverse–sigmoid sinus

Abbreviations and Acronyms
AV: Arteriovenous
DAVF: Dural arteriovenous fistula
DAVM: Dural arteriovenous malformations
NBCA: N-butyl cyanoacrylate
TSDAVF: Transverse–sigmoid sinus dural arteriovenous fistula
TSS: Transverse–sigmoid sinus

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CLINICAL PRESENTATION OF DAVFs

Patients with TSDAVFs may present with subjective pulse-synchronous tinnitus, bruit, insomnia, headache, visual decline, seizure, and/or altered mental status, including dementia (3, 41). The most common symptoms of TSDAVFs were pulsatile tinnitus (81% of cases), headaches (15%), and intracranial hemorrhage (10%) (21, 25). Even trigeminal neuralgia has been reported (11). However, a history of pulsatile tinnitus was reported in 50% of the case reports of trigeminal neuralgia caused by a DAVF (11). Dural AVFs can also result in life-threatening brain edema, hemorrhage, and venous infarction (24, 37, 57). Those with a retrograde leptomeningeal venous drainage are regarded as high-risk, having the potential to hemorrhage and result in serious neurologic complications requiring urgent treatment (27). Neurologic dysfunction is thought to be due to venous hypertension, with resulting venous ischemia or hemorrhage (43, 58). Drainage of the fistula into cerebral surface veins, as opposed to dural sinuses, probably predisposes to an aggressive course and merits surgical or endovascular therapy. Pulsatile tinnitus, heard by the patient or by auscultation over the mastoid, may be an indication for magnetic resonance imaging or angiography (3, 59, 60). Conventional magnetic resonance imaging is often positive in DAVF with retrograde cortical venous drainage or retrograde flow in the venous sinus with venous congestion. DAVFs with retrograde cortical venous drainage or retrograde flow in the venous sinus with venous congestion often has prominent flow voids on the surface of the brain. High-intensity lesions in the deep white matter on T2-weighted images are secondary to the venous hypertension and venous congestion (**Figure 1**). The combination of prominent flow voids on the surface of the brain and high-intensity lesions in the deep white matter on T2-weighted images is highly suggestive of a DAVF (48).

ANATOMY

The transverse sinus extends from the confluence of sinuses to the opening of the superior petrosal sinus, beyond which it continues as the sigmoid sinus and then the jugular bulb. The transverse sinus always has venous cranial afferents, whereas the sigmoid sinus never receives cerebral veins. The potential for retrograde leptomeningeal venous drainage evolving in these locations is therefore not the same. Septations may occur within the dural sinuses and result in separate venous channels, which may run parallel to each other (5). On occasion, one of these channels may be draining the brain while the other is exclusively used for drainage of the DAVFs, providing a specific target for treatment (56).

TSDAVFs have two types of arterial suppliers (6, 30). The first type is transosseous arterial supply, which is mainly tortuous and multiple and comes from a single arterial trunk, normally the transosseous branches of the occipital artery and/or superficial temporal artery. In addition to representing the main path for most of the blood flow for the DAVF, it is also difficult to perform superselective catheterization of

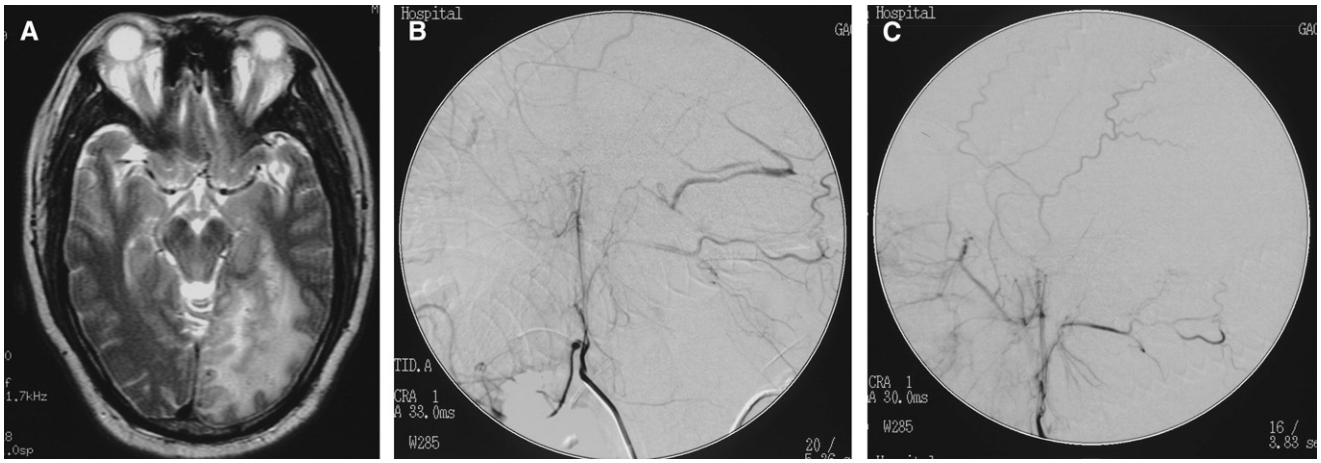

Figure 1. A, T2-weighted image. Diffuse white matter hyperintensity is seen in occipital lobe on the left side. **B,** Left external carotid angiogram shows the dural arteriovenous fistula (DAVF) fed by posterior auricular artery with drainage into the left isolated sigmoid sinus. Note reflux into the cortical veins, occlusion of the left sigmoid sinus and severe stenosis of the left transverse sinus. **C,** Left external carotid angiogram obtained after transarterial embolization shows that the DAVF was completely obliterated and there is no reflux into the cortical veins.

this transosseous arterial supply. The second type of vascularization for TSDAVFs is meningeal, the petrosquamous branch of the middle meningeal artery is frequently recruited in TSDAVFs of the transverse–sigmoid sinus. Although the meningeal arterial system may appear to be a supplementary afference network for DAVFs (**Figure 2**), it provides a natural access to the venous collector system of these lesions (30). Contrary to the transosseous arterial supply, the meningeal arterial system penetrates the cranium through the base, thus making the anatomy more navigable and, consequently, more favorable to microcatheterization (**Figure 3**).

PATHOGENESIS

TSDAVFs may be of either congenital or acquired origin. Congenital lesions are much rarer and thought to develop in the first trimester as a result of persistent communication between future arterial and venous segments of the primitive vascular plexus as the intervening capillary network fails to develop (22). Sinus thrombosis and increased venous pressure are recognized contributors to the development of acquired TSDAVFs (20). Nishijima et al. (46) reported a 72% rate of sinus thrombosis concomitant with TSDAVFs. In experimental models, increased venous pressure associated with sinus thrombosis and venous outflow obstruction resulted in the opening of preexisting arteriovenous (AV) fistulae in the normal dura mater (22). Ishikawa et al. found that normal AV fistulae exist within the dura and DAVFs may subsequently evolve from these normally occurring fistulae if the conditions are favorable (22). Congenital factors, namely the persistence and enlargement of primitive dural arteriovenous communications that normally involute during development, are thought by some authors to be causal. The occurrence of DAVFs during childhood, however, is rare. When they do occur in children, these lesions tend to be complex and bilateral, occur more often in male patients, and are associated with cardiac failure and a high mortality rate (38%) (2). Arnautovic et al. (2), in their comprehensive meta-analysis, outlined three possible stages in the natural history of DAVMs: 1) sinus thrombosis with engorged dural venous collaterals and the opening of embryonic arteriovenous communications; 2) arteriovenous shunting, which favors the recruitment of arterial feeders into the nidus with secondary venous hypertension; and 3) leptomeningeal retrograde venous drainage, with possible subsequent varicose and aneurysmal dilation. Hamada et al. (16) hypothesized that the venous hypertension in patients with DAVMs is based on two factors: 1) the increased blood flow through the draining vein caused by a direct shunt into this vein and 2) the restricted venous outflow, which arises distal to the DAVMs because of the increased blood flow, elevated pressure, and turbulence in the draining vein. According to Hamada et al. (16), these stresses combine to restrict the venous outflow and, in turn, decrease cerebral compliance, elevate intracranial pressure, and even cause hydrocephalus in some patients (2). If blood flow through existing dural AV fistulae increases because of sinus thrombosis, trauma, or sinus or venous hypertension, AV fistulae may develop into DAVFs. Thus, the involvement of AV fistulae in the normal dura mater in the etiology of DAVFs cannot be ruled out (22). It might be postulated that sinus hypertension, caused by stenoocclusive disease of the venous sinuses (at least in some cases), and arterial hypertension force open abnormal connections between arteries and veins in the dura mater, which may result in increasingly dilated venules and the formation of DAVFs (22). After such an abnormal arteriovenous fistula has been formed in the wall of the venous sinus, thickening and stenoocclusive sinus lesions ensure that the fistula also recruits arterial blood from numerous dural and, later, pial arteries. Increased sinus pressure promotes the progression of the disease process, and thus a vicious cycle might be created (19).

PATHOPHYSIOLOGY

Histopathologically, DAVFs consist of multiple abnormal direct communications (so