Clinical Neurology and Neurosurgery 115 (2013) 241–251

Contents lists available at SciVerse ScienceDirect

Clinical Neurology and Neurosurgery

journal homepage: www.elsevier.com/locate/clineuro

Review

Endovascular management of intracranial dural arteriovenous fistulas: A review

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ARTICLE INFO

Article history:
Received 7 April 2012
Received in revised form
11 November 2012
Accepted 24 November 2012
Available online 31 December 2012

Keywords:
Dural arteriovenous fistula
Endovascular
Embolization
Transarterial
Transvenous
Onyx

ABSTRACT

Dural arteriovenous fistulas (DAVFs) are rare pathological entities presenting with a diverse clinical course, ranging from benign to life-threatening. Digital subtraction angiography remains the gold standard in the diagnosis of clinically suspected DAVFs. This article reviews the ethiopathogenesis, natural history, classification systems, clinical and angiographic features, and the current treatment strategies for these complex lesions. The management of DAVFs may include conservative treatment, endovascular intervention, microsurgery, and stereotactic radiosurgery. A multidisciplinary approach involving a neurosurgeon, interventional neuroradiologist, and neurologist is required before considering any type of treatment modality. The indication for the best therapeutic alternative must be individualized for each patient.

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Abbreviations: AVF, arteriovenous fistula; AVM, arteriovenous malformation; BFGF, basic fibroblast growth factor; CS, cavernous sinus; CT, computed tomography; CTA, computed tomography angiography; DAVF, dural arteriovenous fistula; ECA, external carotid artery; ICA, internal carotid artery; ICH, intracranial hemorrhage; ICP, intracranial pressure; IJV, internal jugular vein; ILT, inferolateral trunk; IMA, internal maxillary artery; IOP, intraocular pressure; IOV, inferior ophthalmic vein; IPS, inferior petrosal sinus; MHT, meningohypophyseal trunk; MMA, middle meningeal artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NBCA, *N*-butyl cyanoacrylate; OA, occipital artery; OCP, oral contraceptive pills; OphA, ophthalmic artery; PMA, posterior meningeal artery; PVA, polyvinyl alcohol; SOV, superior ophthalmic vein; SPS, superior petrosal sinus; SPhS, sphenoparietal sinus; SSS, superior sagittal sinus; STA, superior temporal artery; TSS, transverse/sigmoid sinus; TDAVF, tentorial dural arteriovenous fistula; VA, vertebral artery; VEGF, vascular endothelial growth factor.

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0303-8467/$ – see front matter. Published by Elsevier B.V.
http://dx.doi.org/10.1016/j.clineuro.2012.11.021

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1. Introduction

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal shunts between dural arteries and a dural venous sinus or cortical (leptomeningeal) vein. They account for 10–15% of all intracranial vascular malformations [1,2], comprising roughly 6% of supratentorial and 35% of infratentorial AVMs [2]. Generally, the arterial supply to DAVFs is by meningeal arteries, although "parasitization" of pial vessels may occur as the DAVF increases in size, causing a subsequently greater degree of shunting [3]. The fistulous connection is typically contained in the leaflets of the dura mater, within a venous sinus or plexus, but there is possible transdural, retrograde flow into cortical and/or leptomeningeal veins [4]. In a small proportion of cases, a parallel channel, probably within the dural wall of the sinus, is the primary recipient of drainage from the fistula, with secondary drainage into the primary sinus and/or cortical veins [5,6]. DAVFs associated with leptomeningeal venous drainage or a venous varix are considered high risk with a poor natural history [7–10]. The overall hemorrhage risk from a DAVF is approximately 1.5% per year [11]. Given the anatomic complexity and variability of DAVFs, the decision to treat should be based on the venous drainage pattern, the natural history of the lesion, the severity of presenting symptoms including previous hemorrhage, the patient's age and general condition, angiographic features, the location of the DAVF, and the morbidity and mortality rates of the procedure under consideration. Although there are several classifications systems for intracranial DAVFs, Cognard and Borden's is most widely used and accepted (see Table 1). Cognard's classification is based on Djindjian and Merland classified DAVFs into four types based on the venous drainage pattern. Type I shows an immediate drainage into a dural sinus or meningeal vein, type II presents with initial drainage into the sinus and retrograde flow into other sinuses or cortical veins. Type III shows an initial drainage into a cortical vein and type IV an initial drainage into a cortical vein with venous ectasia.

2. Etiology and pathophysiology

Given the variability in location and the inherent complexity of dural AVFs, there are multiple etiologies including trauma, iatrogenic (i.e. post-surgical), secondary to aneurysmal rupture, spontaneous, congenital, and idiopathic [13]. Although originally believed to be congenital in nature, there is mounting evidence over the last 30 years supporting that they may be acquired lesions resulting from impairment of venous outflow [12]. Thus, the majority of DAVFs encountered are believed to be the result of venous thrombosis or venous stenosis. Hormonal factors have also been postulated as causative [4]. The etiology of sinus thrombosis may be traumatic, infectious, or related to a thrombophilic condition (Factor V Leiden, protein C or S deficiency, pregnancy, OCPs). Initial studies proposed sinus thrombosis-induced inflammation resulting in initiation of angiogenic activity, with recanalization of the sinus with formation of a connection between the dural arterial supply and venous sinus. A contribution by angiogenic factors to the formation of DAVFs is supported by the finding that basic fibroblast growth factor (BFGF) and vascular endothelial growth factor (VEGF) are expressed in this type of lesions [14]. Although the formation of DAVF following venous sinus thrombosis is well described, in the majority of cases there is no demonstrable preceding venous sinus thrombosis, indicating that there may also be other factors promoting angiogenesis. Venous hypertension following thrombosis has been postulated to cause DAVFs formation in sites remote from the location of thrombosis [15], and this association has been demonstrated in a rodent model [3,16]. Venous hypertension may induce the opening of potential arteriovenous connections by means of the vasa vasorum to the dural sinus, or the opening of embryonic remnants of the dural venous plexus [5,17].

3. Dural AVFs of aggressive course

The clinical presentation of DAVFs primarily depends on location and pattern of venous drainage. Dural AVFs with an aggressive course have characteristic angiographic features such as cortical/leptomeningeal venous drainage and/or an associated varix. The source of hemorrhage from a DAVF is usually not within the nidus of the fistula but rather the distended leptomeningeal venous varices [18]. In a meta-analysis, Awad et al. reported hemorrhage in 88% and severe neurological deficit in 12% of 100 aggressive cases amongst 377 patients with DAVFs; where leptomeningeal venous drainage, variceal or aneurysmal (venous) dilations, and galenic drainage correlated significantly with an aggressive neurological presentation [7]. In 102 intracranial dAVFs, Davies et al. reported bleeding rates of 13% for Cognard type IIb to 75% for Cognard type V DAVFs [19]. Dural AVFs with retrograde venous drainage have a 35% risk of rebleeding within 2 weeks after the initial hemorrhagic presentation [20]. Neurological complications can also be caused by parenchymal venous congestion, neurovascular compression and local mass effect by dilated varices [21–25]. Due to the aggressive clinical behavior and poor clinical outcome associated with these types of lesions, they should be treated promptly. Indications for early treatment include the presence of leptomeningeal drainage, ICH, increased ICP, increased IOP causing visual loss, and/or neurovascular compression producing neurological deficits [18]. Hurst et al. reported venous hypertensive encephalopathy with dementia in 12.5% of 40 patients with DAVFs [26].

Based on the pattern of venous drainage, Collice et al. [21] identified two types of high-grade lesions, those that drain into a large dural sinus with retrograde filling of leptomeningeal veins and those that drain directly into leptomeningeal veins without involvement of a large dural sinus. For the first type, the aim of treatment is to occlude only the arterialized fistulous sinus and the most proximal portion of the refluxed leptomeningeal vein, thus preserving the uninvolved segments of the same sinus and distal veins that join the arterialized leptomeningeal veins functioning in normal parenchymal venous drainage. Occlusion of these functional veins may result in venous hypertension and venous infarction [18]. In this type of fistula, surgery is more difficult and carries a higher risk of complications, therefore endovascular embolization is the