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Review Article

Update of embolization of intracranial dural arteriovenous fistula

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Abstract

Intracranial dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous communications within the dura locating near a major venous sinus and are supplied by pachymeningeal arteries. DAVFs represent 10–15% of all intracranial arteriovenous malformations. The natural history and clinical manifestations are determined by location of the DAVFs and their angioarchitecture. Aggressive DAVF is usually associated with leptomeningeal venous drains or reflux. It may present with hemorrhagic or nonhemorrhagic stroke. The goal of embolization of DAVFs is total fistula occlusion without interfering with the normal dura–venous drains. Embolization can be performed by transarterial and/or transvenous routes or direct puncture of affected dural sinus. Selection of embolic materials depends on access route and angioarchitecture of the fistula. With the involution of endovascular devices, embolic materials, and high-quality angiography, endovascular embolization of DAVFs has been proved a safe and effective method of treating these complex cerebrovascular lesions.

Keywords: arteriovenous fistula; dura; embolization

1. Introduction

Dural arteriovenous fistulas (DAVFs) can occur in any intracranial dural regions, most commonly in the cavernous sinus or transverse-sigmoid sinus.[1–3] DAVFs are usually fed by dural branches of the external carotid artery. The etiology of DAVFs is not fully understood; some are congenital and others are acquired, but most are thought to be acquired. Different etiologies have been implicated including thrombosis, trauma, or surgery or in the *postpartum* period.[4–6] Patients may be asymptomatic or may experience symptoms ranging from mild symptoms to fatal hemorrhage.[7–11] DAVFs have been classified to benign type with dural sinus drain or aggressive type with leptomeningeal venous drain and/or reflux which may associate with hemorrhage or nonhemorrhagic neurologic deficits.[12–14] Management of DAVFs includes open surgery, radiosurgery, embolization, or combined treatment. In this article, we will discuss the method of embolization, with emphasis on the access routes, embolic materials, and individual DAVF embolization.

2. Symptoms, natural history, and classification of intracranial DAVFs

The clinical presentations of DAVF are variable, depending on the location of the shunt and angioarchitecture of the shunting flow. These symptoms include exophthalmos, bruit, cranial nerve deficits, tinnitus, hemorrhagic or nonhemorrhagic neurological deficits, increased intracranial pressure, papilledema, and cerebrospinal fluid reabsorption abnormalities resulting in ventricular enlargement or congestive heart failure. Several classification systems have been developed to grade the risks and natural course of DAVFs.

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Those devised by Cognard et al[15] and Borden et al[16] are the most widely used. All these classifications emphasize the fistula venous drains. DAVFs with antegrade dural sinus drains and only association with sinus reflux usually present with benign symptoms. By contrast, DAVFs with retrograde leptomeningeal cortical venous drainage channel show a significantly high rate of aggressive symptoms such as hemorrhagic and nonhemorrhagic neurologic clinical manifestation. Although DAVF location is not directly correlated with aggressive behavior, the propensity for dangerous drainage patterns found at initial diagnosis does vary with location.

3. Goal of embolization of DAVFs

The treatment strategy of DAVFs should be tailored individually to patients considering the angioarchitecture, natural history of the DAVFs, and the severity of symptoms. The definitive treatment of DAVFs requires the occlusion of the arteriovenous shunt without interfering with the normal venous drains. Treatment can be performed by conventional open neurosurgery, radiosurgery, or endovascular embolization, or a combination of these modalities. The first reported series of embolization cases was by Halbach et al in the 1980s.[17] With the development of newer techniques and embolic materials in the field of interventional neuroradiology, endovascular therapy has now been developing as the primary treatment strategy to manage DAVFs, particularly in high-risk DAVFs, reserving surgery only if endovascular techniques fail or are unfeasible. The goal of embolization is to achieve an angiographic cure by obliteration of all feeders and proximal draining veins with preservation of the patency of the affected sinus. In those difficult and complex DAVFs with a difficulty of achieving total occlusion of the fistula, the goal of embolization is to achieve partial treatment with reversal of the aggressive type of DAVF to benign type, to facilitate subsequent radiosurgery or neurosurgery.[18–20] Embolization should usually fulfill the following requirements. If a sinus is occluded or severely stenosed, treatment should be directed to reestablish sinus patency[21,22]; if that fails, sinus occlusion with angiographic cure of DAVF is considered. If there is a functional venous pathway that was not previously occluded, it should not be sacrificed. Treatment should not induce redirection of flow to other cortical veins.

4. Embolization access routes

There are three embolization routes to manage DAVFs: transarterial embolization (TAE) of arterial feeders (Figs. 1 and 2), transvenous embolization (TVE; Figs. 3 and 4) or direct puncture of the affected sinus. Selective TAE requires

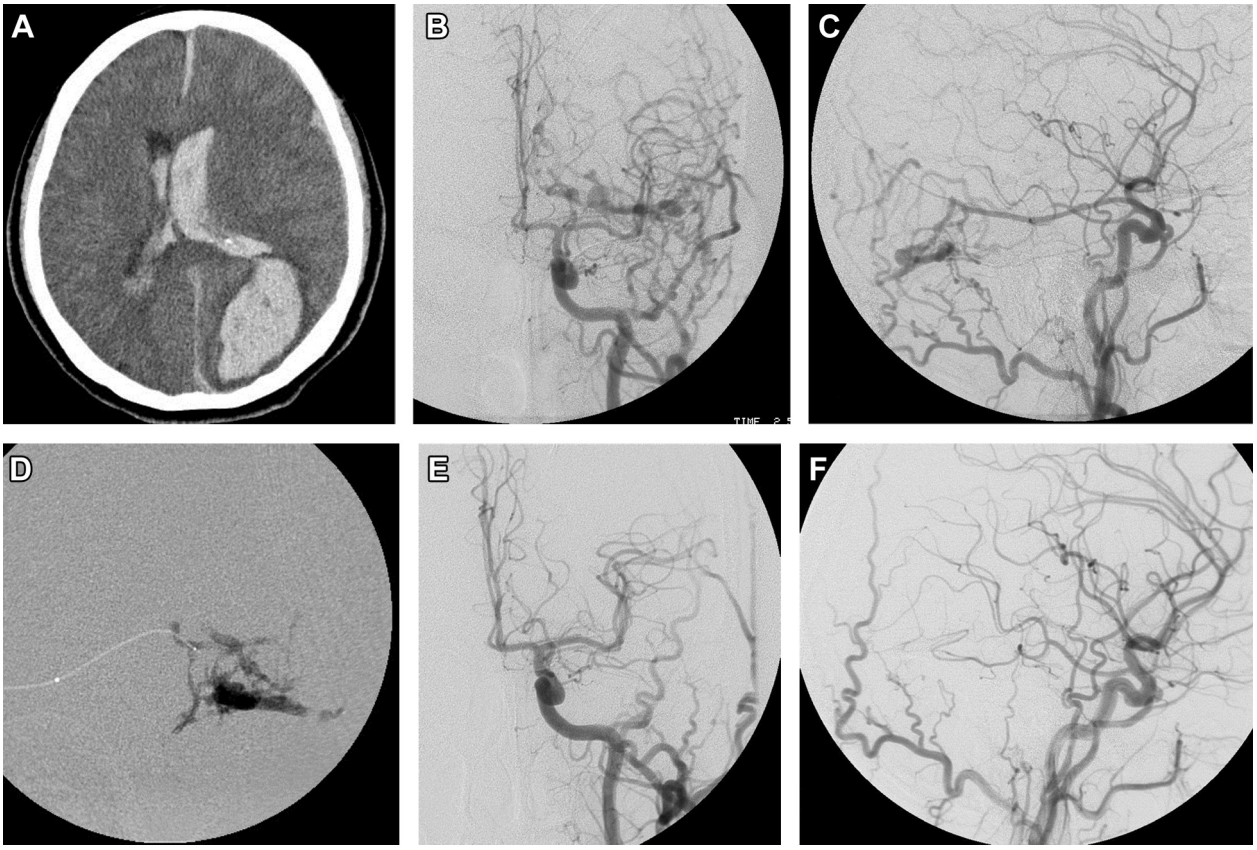

Fig. 1. A 61-year-old woman presented with consciousness change. (A) Brain computed tomography demonstrated intracerebral hemorrhage with extension to ventricular systems; subdural hematoma was found as well; (B, C) left carotid angiograms depicted Cognard type IV dural arteriovenous fistulas at the left posterior tentorium; (D) the patient underwent transarterial (middle meningeal artery) n-butyl-2-cyanoacrylate embolization; and (E) postembolization angiograms demonstrated total fistula occlusion.