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Review

Radiosurgery for dural arterio-venous fistulas: A review

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ABSTRACT

Dural arteriovenous fistulas (dAVFs) are vascular lesions involving direct connections between intracranial dural arteries and venous sinuses. The goal of treatment of these vascular lesions is to alleviate symptoms and prevent future hemorrhage. While endovascular embolization remains the primary method of treatment and obliteration of dAVF recently, stereotactic radiosurgery (SRS) has been used as a treatment modality in select dAVF either alone or in conjunction with endovascular embolization. Considering recent studies examining dAVFs natural history and possible therapeutic interventions, the authors provide a concise review of the literature and discuss the indications, efficacy, and safety of SRS in the management of dAVFs.

Contents

1. Introduction

Stereotactic radiosurgery (SRS) is increasingly being studied and used in the treatment of intracranial vascular anomalies. It was first used for arteriovenous malformations (AVMs) in the 1970s to provide specific radiobiological effects for a defined volume of abnormal vessels. Since then, SRS use has expanded to a range of indications including AVMs and even cavernomas [33]. SRS induces histo-pathologic changes such as perivascular sub-endothelial edema, fissuring of vessel walls, spot hemorrhages, formation of thrombi, and proliferation of smooth muscle [21].

Dural arteriovenous fistulas (dAVFs) are vascular lesions that are commonly thought to be acquired and are distinct from AVMs [33]. They consist of direct connections between intracranial dural arteries and the cerebral venous sinuses, lacking a nidus on angiography, and comprise 10–15% of all brain vascular malformations [16]. In descending order of incidence, they are found to involve the transverse-sigmoid junction, cavernous sinus, tentorial/torcula, and at the cerebral convexities draining in the sagittal sinus.

Similar to AVMs, dAVFs can cause symptoms secondary to hemorrhage as well as seizures and progressive neurologic deficits [12]. Their symptomatology may also depend on their location, ranging from pulsatile tinnitus associated with transverse-sigmoid junction dAVFs, to chemosis, proptosis, and ophthalmoplegia caused by cavernous sinus dAVFs.

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As previously mentioned, unlike other cerebral vascular malformations, dAVFs are more often thought to be acquired lesions from events such as trauma, sinus thrombosis, infection or surgery [8]. Houser et al. [16] illustrated the pathogenesis of this disease entity by showing a correlation between the development of venous sinus thrombosis and the formation of a fistulous connection. DAVFs are commonly thought to occur after venous thrombosis or occlusion leading to venous hypertension. Normal arteriovenous shunts can then enlarge secondarily to this venous hypertension. Moreover, increased blood flow often results in further venous hypertension, forming a positive feedback loop with an increasing volume of the arteriovenous shunting and lesion [33]. Recent studies, regarding blood hemodynamics at the site of a venous thrombosis, have shown that this alteration can lead to abnormal arteriovenous shunting [12]. Other research has gone into identifying individuals with a genetic predisposition to dural AVFs from elevated levels of vascular endothelial growth factors (VEGF) [1,14,31]. However, a definitive and consistent link remains elusive.

2. Treatment indications and natural history

Treatment is necessary for dAVFs when these lesions are symptomatic or at a high risk of hemorrhage. Prevention of hemorrhage or development of neurologic sequelae such as opthalmoplegia, visual loss, or seizures is a strong indication. Treatment may also obviate or relieve symptoms such as tinnitus, headache, or bruit. Digital subtraction angiography is the gold standard imaging modality to assess dAVF while specifically determining the presence of hemorrhagic risk factors.

Studies have reported risk factors such as location, venous drainage, and local flow dynamics. The history of a prior hemorrhage related to the dAVF is potentially the strongest indication for treatment with reports of up to 43% rebleeding within the first few days, with other reports suggesting 35% within two weeks [3,10]. Singh et al. [27] found and discussed five significant hemorrhagic risk factors including cortical venous drainage, focal neurologic deficits, posterior fossa location, male sex, and greater age in their review of 402 patients with dAVFs. Malik et al. [23] reported that dAVFs remotely located from a major sinus have a 7 fold greater risk of hemorrhage than those fistulas located near the sinuses. Awad et al. [1] found a predilection of tentorial dAVFs to present symptomatically with hemorrhage. Eighty percent of tentorial dAVFs presented to their center with intracranial hemorrhage, as opposed to 10–20% of transverse/sigmoid and cavernous sinus dAVFs.

Detailed imaging of the individual venous anatomy for cortical venous drainage (CVD) is necessary due to its traditional correlation with hemorrhage. van Dijk et al. [32] reported a 15% annual risk of hemorrhage or progressive neurologic deficits with the persistence of cortical venous reflux. In these lesions they also reported a 10% annual mortality rate. Cognard et al. [8] noted hemorrhage in 40% of dAVFs with cortical venous drainage and an even higher rate of 65% of those with CVD and venous ectasia. Traditionally, dAVFs with retrograde drainage or CVD have been more aggressively treated, as opposed to asymptomatic dAVFs without CVD where a case for close observation can be made. However, some studies have differed with regards to the actual risk associated with CVD in patients without a previous hemorrhage. Soderman et al. [28] reported a 1.5% annual risk of hemorrhage in 53 patients with unruptured dAVF with CVD. Similarly, Strom et al. [29] reported on 17 patients with CVD without a prior history of hemorrhage and found an annual hemorrhage rate of 1.4%. These studies highlight the need for treatment options with low side effect profiles for dAVF with CVD and without a history of hemorrhage if treatment is going to be undertaken.

Other documented high-risk features are also observed occasionally. Some examples include drainage primarily to the vein of Galen, a large venous varix seen on angiogram, as well as a thrombosis of venous outflow pathways distant from the fistula [1,15,17]. Pseudoaneurysms also may appear along with dAVF to increase the risk of ischemic stroke or hemorrhage. Some studies suggest that the morbidity and mortality of hemorrhage from dAVF may rival that of aneurysmal SAH with a mortality rate between 20% and 30% [4].

3. Traditional treatment perspectives

The decision to treat dAVF involves understanding the risk/benefit analysis involved with each treatment strategy compared to the natural history of the lesion. Manual compression of afferent carotid and jugular vessels was discussed by Halbach et al. [15] as a method to promote cavernous sinus thrombosis and sigmoid fistulas. Traditionally, open surgical strategies have been reported as a means of direct fistula excision, or if not feasible, obliteration via feeding vessel ligation or venous sinus packing. When able, the goal of surgery has evolved to the disconnection of the veins bridging the dAVFs and brain parenchyma [33]. Recently, dAVFs are more commonly treated by embolization either transarterially or transvenously. With advances in endovascular techniques and catheter technologies, its role has expanded over the past twenty years. For all modalities the goal remains fistula obliteration to remove the risk of hemorrhage and if present cure symptoms. The rationale for the prioritization of surgical excision or endovascular embolization remains the ability to provide immediate improvement or complete resolution, as well as the elimination of cortical venous hypertension [33].

4. Stereotactic radiosurgery

The goal of SRS for any intracranial lesion, including dAVF, is to selectively provide radiation to a precise, abnormal volume, sparing normal surrounding structures from any damage. Stereotactic radiosurgery has been utilized in the treatment of dAVFs after its demonstration of utility in other vascular malformations. SRS is reported mainly for those dAVFs with a high treatment risk involved in surgical excision or embolization; as well as for lesions that fail those more traditional modalities or require further adjunctive therapy [21]. In some instances, a complex arterial supply or vascular anatomy with treacherous intracranial anastomosis creates lesions that are unable to achieve endovascular obliteration with an appropriate low risk. Koebbe et al. [17] also reported the use of SRS in a patient with renal failure as a medical contraindication to endovascular embolization. SRS is generally reserved for those patients failing the more traditional treatment strategies or for those with a small nidus and benign course. A major disadvantage is the necessary latency period until obliteration could be achieved with radiosurgery. Conversely, the treatment latency of radiosurgery could be an advantage in preventing the risk of venous hypertension and infarction involved with sudden complete obliteration by endovascular methods. Nevertheless, some authors advocate SRS use in those dAVFs with a low chance of spontaneous resolution and presenting with disabling deficits.

SRS targets the arterial feeders of the dAVF nidus to induce diminishing shunting and eventually obliteration. It is not entirely clear how SRS exactly achieves obliterations; however, one theory points to radiation-induced small muscle expansion, adventitial fibrosis, and an intimal response leading to small vessel occlusion [21]. Another potential theory is that an inflammatory process in the adjacent dura causes external pressure on small arteries leading to reduced blood flow and thrombosis. DAVF may reside in