Response to Letter Regarding Article, “Intracranial Dural Arteriovenous Fistulae: Clinical Presentation and Management Strategies”

We thank Gross and Du for their comments on our recent review of dural arteriovenous fistula (DAVF). We disagree with their suggestion that meningeal vessels exclusively supply DAVFs. Although pial supply is rare, we have observed it on several occasions, and it has been described in the literature.

We recognize the contributions made by Houser et al and Djindjian in the identification of cortical venous drainage (CVD) as a risk factor for hemorrhage associated with DAVF. However, the aim of our article was not to provide an historical overview of the development of our modern understanding of these lesions. We highlighted the report by Malik et al because they had also performed a fairly extensive meta-analysis of 27 previously published cases of DAVF associated with hemorrhage (out of a total of 213 lesions), putting into perspective the risk posed by CVD in a larger patient population.

As we emphasized in our article, reported annualized rates of hemorrhage, nonhemorrhagic deficits, and mortality from DAVF with CVD have varied considerably in the literature. We agree with Gross and Du that this variability probably reflects heterogeneity in the population of DAVF’s included in individual reports. Consequently, we felt that reporting single, averaged, annualized rates for these sequelae may be misleading, and instead opted to describe the findings of 3 representative studies. We would like to point out that the 6% annual hemorrhage rate of DAVF with CVD cited by Gross and Du is well within the range that we present in our review (4%–19%).

We agree with Gross and Du that aggressive clinical presentation and venous ectasia are risk factors for DAVF-associated hemorrhage. In our article, we emphasized that lesions with CVD often have more aggressive clinical presentations, and we provided Cognard classification system of DAVF, which highlights venous ectasia. However, the key factor determining management of DAVF remains the presence or the absence of CVD. On the basis of our review of the literature and current understanding of the natural history of aggressive DAVF, patients with fistula demonstrating CVD, even if they are asymptomatic, or do not have associated venous ectasia, should be offered treatment.

In our review, we clearly state the limitations of the article by Narvid et al, which evaluated the ability of computed tomography angiography to detect arterial feeders of DAVF. It is important to note that cross-sectional imaging techniques have made tremendous strides over the last several years, and they can be a reasonable screening study in low-risk patients (eg, those presenting with pulsatile tinnitus). However, catheter angiography maintains its place as the imaging gold standard and should be performed if the clinical suspicion for DAVF is high.

We stand by the statement that a majority of intracranial DAVF may be cured by modern endovascular methods, with few lesions lacking a safe route of transarterial or transvenous access. To the best of our knowledge, multidisciplinary groups often defer to endovascular therapy because of its minimally invasive nature and high cure rate. Although surgery remains an excellent treatment option for DAVF, it is increasingly being reserved for lesions that either cannot be approached endovascularly or which fail endovascular therapy. In cases where endovascular therapy fails, it typically will not interfere with a subsequent open surgical approach to the DAVF. However, the reverse is not always true, supporting the strategy of reserving surgery for more challenging cases that are not cured by endovascular means.

It was not our intention to equate gamma knife surgery with stereotactic radiosurgery. The authors point is well taken that other types of stereotactic radiosurgery are applicable to DAVF.

Disclosures

None.

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