Commentary on “Surgical Treatment with or without Embolotherapy for Arteriovenous Malformations”

Byung-Boong Lee, MD, PhD, FACS

Arteriovenous malformations (AVM) is relatively infrequent congenital vascular malformations (CVMs). But its clinical risk/significance is far exceeding to other CVMs like venous malformation (VM) or lymphatic malformation (LM). Because, AVMs are the outcome of birth defect involving the vessels of both arterial and venous origins. It maintains direct communications between the different size vessels or a meshwork of primitive reticular networks of dysplastic minute vessels. Such unique condition results in shunting of high velocity, low resistance flow from the arterial vasculature into the venous system so that its hemodynamic consequences is quite serious and often progresses to potentially life or limb threatening condition.

Besides, almost all the AVMs are ‘extratruncular’ lesions originated from the early stage of embryogenesis (cf. truncular lesion from the later stage) so that they possess unique mesenchymal cell (angioblast) characteristics to grow when stimulated by various conditions (e.g. female hormone, menarche, pregnancy, trauma, surgery). Ill planned treatment strategy with incomplete excision, for example, often stimulates the residual lesion to grow back.

To make the condition more difficult to handle, all these ‘extratruncular’ AVM lesions are diffusely infiltrating into the surrounding tissue by its nature so that the complete excision of such lesion is extremely difficult if not impossible unless risking severe morbidity and complication (e.g. massive bleeding).

Therefore, surgically ‘inaccessible’, if not difficult, infiltrating lesions should be considered for the endovascular therapy as the first choice, and only for surgically ‘accessible’ lesions (e.g. localized limited lesion), surgical resection may be the treatment of choice with a chance of optimal control. But the preoperative sclerotherapy or embolization should be considered whenever feasible to supplement to subsequent surgical excision by reducing the morbidity (e.g. operative bleeding) and defining the lesion borders. Such a combined approach may provide an excellent potential for a curative result as the authors claim.

Nevertheless, an aggressive control of the nidus of the AVM lesion itself is essential to prevent recurrence and/or fast deterioration of the AVM lesion from much complicated hemodynamic consequences. And previous approach to the AVM only with the strategy to shut off the feeding artery (e.g. ligation, embolization), leaving the nidus of the lesion intact, would make the condition worse, only provoking a more aggressive neovascular recruitment by this primitive lesion without exception.

Due to such critical nature of the AVM lesions, all the AVM lesions should be considered for the treatment whenever feasible in earliest possible time. However, right treatment strategy warrants accurate diagnosis of AVM as well as accurate assessment of its extent, severity, and progress, which is critical to avoid/minimize the complication and morbidity by currently available therapies.

And the ‘controlled’ aggressiveness has to be exercised for the management of AVM in general within the accepted boundary of the “palliative” concept only when the benefit should exceed the morbidity following the treatment. The temptation to intervene AVM aggressively therefore, must be tempered with realistic assessment of the long-term prospect of the treatment outcome.

Critical role of careful assessment of the treatment strategy based on benefit versus risk of morbidity before the commitment cannot be overemphasized, and the ultimate goal of the treatment has to be clearly defined with realistic expectation.
References