Spinal dural arteriovenous fistulas (SDAVF): Follow-up

Jellema et al. followed patients treated for SDAVF for 0.7 to 15 years (median 5.7 years). After treatment, 70% of patients rated their activities of daily life to be better than before treatment. In most patients gait disturbances and muscle strength had improved after treatment, with reduced disability.

Surgery vs endovascular treatment of spinal dural AVF

Commentary by Elad I. Levy, MD, and L. Nelson Hopkins, MD

Not much is known concerning the long-term outcome of endovascular treatment of spinal dural AVF. Although some have reported favorable long-term outcomes following embolization with liquid embolic agents such as n-butyl 2-cyanoacrylate (NBCA), the treatment of AVF remains controversial. Most clinicians agree that treatment of dural AVF is indicated because of the progression of spinal deficits once the lesion becomes symptomatic. Spinal dural AVF may induce a subacute necrotizing myelopathy that is seen as a hyperintense signal on T2-weighted MRI. The suggested mechanism for this myelopathy is venous medullary hypertension.

In the Jellema et al. report on the long-term follow-up of endovascular treatment of spinal dural AVFs in 44 patients, complications included transient leg weakness in 1 patient, persistent lower extremity paresthesias or pain in 9 patients, and postoperative pain attributed to muscle ischemia in 23 patients. Importantly, 31 (70%) patients described functional improvement at a mean follow-up period of 5.7 years. These findings are similar to those of others.

Several groups report excellent results following surgical interruption of the pathway between the arterial feeder(s) and drainage pathways for AVF with low morbidity rates. Nonetheless, some lesions are not easily amenable to surgical ligation, and often patients are not suitable surgical candidates. Further discussion of the anatomical and clinical characteristics of patients in this article indicates which patients are likely to benefit from surgical vs endovascular therapy. For example, intradural dorsal AVF with single feeding pedicles (Type A) may be amenable to surgery or endovascular treatment, whereas ventral AVF (Type IV-b and c) may be more suited to endovascular therapy alone or in combination with surgery owing to the complexity of the pathology and a ventral location relative to the spinal cord. Additionally, AVF with easily accessible pedicles that allow microcatheter delivery of NBCA into the venous draining vessel allow for an endovascular cure and preclude the need for open surgery.

In addition to anatomic considerations in determining optimal surgical or endovascular management for spinal cord arteriovenous shunts, it is useful to understand the pathophysiology and etiology contributing to these symptoms. Surgical approaches may be preferable if the symptoms arise from spinal cord compression as a result of venous engorgement, whereas endovascular approaches may be indicated for poor surgical candidates with ischemic symptoms. Endovascular techniques can address steal-induced hypoperfusion but may be less successful at ameliorating long-term mass effect resulting from a dilated venous complex.

References


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