The Natural History of Dural Arteriovenous Shunts: The Toronto Experience

To the Editor:

With interest we have read the article “Natural history of dural arteriovenous shunts” by Söderman et al1 in Stroke. We agree that the dural arteriovenous shunt still is a puzzling neurovascular entity that poses a challenge in knowing the natural disease course. Therefore, every attempt to analyze large series and particularly the events during follow-up should be eluded to. However, things should also be seen in perspective and so, to avoid erroneous conclusions by the reader, we want to make some critical comments.

First of all, the authors differentiate 3 separate periods of time at risk: the first period from the debut of symptoms to the verification of the diagnosis by digital subtraction angiography (DSA; before diagnosis); the second period from the diagnostic DSA to the time of embolization or radiosurgery (radiological follow-up); and the third period from the last DSA showing the DAVS to the death of the patient (clinical follow-up). This differentiation is confusing because it implies that the risk of a DAVS is dependent on whether the diagnosis is angiographically verified or not. It is improbable that the risk ‘before diagnosis’ is different from the risk during follow-up. Furthermore, it is difficult—if possible at all—to determine the exact time at risk ‘before diagnosis,’ because a benign DAVS without cortical venous reflux produces signs and symptoms too, and conversion from a benign to an aggressive DAVS (with cortical venous reflux) is a possibility. In this point of view, the only ‘before diagnosis’ cohort worth studying are the patients presenting with hemorrhage, of which remarkably in the short follow-up from hemorrhage to DSA (mean 10 weeks), already 2 rebleeds occurred. The ‘radiological follow-up period’ corresponds with the period of follow-up used in other studies, but again this follow-up period is very short (mean 0.6 years). Still, the annual incidence of an intracranial hemorrhage is calculated 6.1%. The ‘clinical follow-up period’ is the most obscure of the 3 periods. The authors attempt to make a risk calculation based on 5 patients that not only refused a treatment, but also a repeated DSA. It is, therefore, not clear whether a spontaneous cure occurred. Nevertheless, all 5 patients died after mean 11.7 years, of which 4 patients died of supposed non-DAVS–related causes.

Beside these confusing timeframes and the remarkable exclusion of 14 patients (only because they came from another institute,) another flaw of the Söderman study is the short mean time of patient observation. They collected a large group of patients that apparently was awaiting treatment and added up the small individual bits of time to patient-years. In comparison, in our study on the natural history of DAVS with persistent cortical venous reflux the average follow-up per patient was 4.3 years, almost 7 times as long.2 Taken the fact that hemorrhages in our series occurred at a median of 2.3 years, the reader should realize that the conclusions by the group of Söderman are to be questioned. In our experience the DAVS still is a hazardous vascular entity that mandates prompt diagnosis and treatment.

Disclosures

None.

J. Marc C. van Dijk, MD, PhD
Department of Neurosurgery
University Medical Center Groningen
Groningen, the Netherlands

Karel G. TerBrugge, MD
Robert A. Willinsky, MD
Department of Medical Imaging
Toronto Western Hospital
Ontario, Canada

M. Christopher Wallace, MD, MSc
Department of Neurosurgery
Toronto Western Hospital
Ontario, Canada

