Response to Letter by van Dijk et al

Response:

We thank Drs van Dijk et al for their comments on our recent article about the natural history of dural arteriovenous shunts.1 We agree that these lesions are enigmatic and that data should be interpreted with caution, in particular because all studies on the subject, including ours, are small and surely biased. Van Dijk et al has raised several questions and we will try to answer them in order.

Firstly, why did we differentiate time at risk for hemorrhage from a DAVS into three time periods?

We quote directly from our paper: “The time at risk comprised three different periods. The first period was from the first indication of a DAVS until diagnosis by angiography. Any number of patients may have dropped out prior to diagnosis and this data has limited value. The period from diagnosis until censoring was split into the radiological follow-up period and the clinical follow-up period. The rationale is that DAVSs may close spontaneously.2–3 Therefore the time at risk is certain only if there is a neuroradiological examination at the end of the period, proving the DAVS to be still present. In the patients who died from other causes or refused further radiological follow up, we do not know whether the DAVS was present or not until censoring. Therefore the two periods have different weight as evidence.”

Van Dijk et al propose that “this differentiation is confusing since it implies that the risk of a DAVS is dependent on whether the diagnosis is angiographically verified or not.” We do not understand this point by van Dijk et al considering the cited text above.

Secondly, van Dijk et al propose that “it is difficult—if possible at all—to determine the time at risk ‘before diagnosis,’ since a benign DAVS without cortical venous reflux (CVR) causes signs and symptoms too, and conversion from a benign to an aggressive DAVS (with CVR) is a possibility.” A DAVS may of course change its type of drainage over time. However, van Dijk et al have previously shown that it is very unusual for a DAVS, that initially does not have CVR, to change its type of drainage to develop CVR.4 We cite from van Dijk et al: “our goal was to make it absolutely clear that 98% of DAVFs without CVD (CVD=CVR) follow a benign disease course and do not demonstrate a conversion into the more aggressive type of lesion associated with CVD.” Has van Dijk et al suddenly arrived at a conclusion contrary to what they have published previously? Anyway, as stated above, the time at risk before diagnosis has limited value in a risk calculation, because patients may die or drop out for a number of reasons and never come to diagnosis. For the same reason we find it surprising that van Dijk et al propose that the reported 3.3% annual incidence of rebleeding prior to diagnosis for DAVSs with CVR presenting with hemorrhage, should imply a high risk for hemorrhage in general for patients with DAVSs and CVR.

The next point van Dijk et al have made is that our material partially treated patients (5). However, it is not explained in their letter why this should be a flaw. Some authors suggest a high risk for very early rehemorrhage4 and if this is correct a short mean follow-up time may cause an overestimation of the annual risk for hemorrhage. If on the contrary the risk for hemorrhage increases over time this could hypothetically cause an underestimation of the annual risk for hemorrhage, when based on our material. However, there is to our knowledge no data suggesting such a natural course for patients with DAVSs.

A fourth question is why we excluded the 14 patients coming from other hospitals and only included patients primarily investigated at our hospital? This is basic epidemiological practice. Any number of patients from other hospitals could have died or for other reasons never been examined at our center. Thus there would probably be a selection toward patients at low risk for hemorrhage. Patients with the first angiography performed elsewhere were excluded with the aim to avoid inclusion bias. In this context it should be noted that DAVSs not presenting with hemorrhage probably carry a low bleeding risk (about 1.5% annual incidence in our material) whereas those that present with hemorrhage seem to be more prone to bleed (about 7.4% annual incidence in our material).

Both the present study and the previous study by van Dijk et al have obvious limitations and data from the studies are partly contradictory. Additional analysis of the natural history of this disease is probably required to achieve best patient care. We believe that the present study adds important new information that should be taken into consideration when designing treatment protocols.

Disclosures

None.

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