De novo dural arteriovenous fistula

The development of de novo dural arteriovenous fistula(s) (dAVF) following endovascular embolization of a prior high-flow pial arteriovenous fistula (PAVF) has not previously been reported and the natural history is unknown.

Among 16 cases of PAVFs treated by endovascular embolization, four developed de novo dural arteriovenous fistulas during treatment or on follow-up that were not present before treatment. Information was collected from the clinical case records, imaging by MRI on presentation and during follow-up, all angiographic images and records during each of the procedures and during follow-up.

The time interval between the last embolization and identification of a dural fistula ranged from 3 to 14 months. Ten fistulas were identified in four patients, seven of which were embolized, four with glue, two with Onyx18 and one with absolute alcohol. None recanalized, while one patient developed fistula in an adjacent location that was subsequently treated with radiosurgery. Not all fistulas need treatment; small fistulas with a minimal flow can safely be observed.

De novo dural fistulas following endovascular embolization of high-flow PAVFs is not an uncommon development. They are mostly asymptomatic and develop anywhere along the drainage of the fistula, maturing over time and diagnosed during follow-up studies, emphasizing the need for follow-up angiography. They can be effectively treated by endovascular embolization. Localized refractory dural fistulas can be dealt with by radiosurgery [1].

Etiology

A possible role of local tissue hypoxia as the initial step causing neoangiogenesis and a low degree of endothelial proliferation in DAVFs. Such hypoxia might be caused by venous hypertension or venous thrombosis [2].

Remote dAVFs can develop after transarterial embolization and may shed light on the etiology of this occurrence.

Although the etiology remains unclear, it is important to recognize that such lesions can develop so that patients can be managed and followed appropriately following endovascular treatment [3].

Several theories have been proposed. One theory is that manipulation of the venous anatomy with microcatheters may injure the veins, leading to sinus thrombosis, venous obstruction, and subsequent development of a dAVF

A second possibility is that the architecture of the dAVF was present before treatment but was not visible at the time of angiography [4].

A third possibility is that the initial embolization resulted in venous turbulence through the sinus, thereby initiating thrombosis that lead to the development of an AVF as the thrombus was recanalized [5].

1)
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