The dural vascular plexus in subdural hematomas: a pivotal neuro-anatomical structure in their physiopathology and the indirect target of their management?

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Purpose Subdural hematomas (SDH) have classically been attributed to bridging vein rupture. Recent advances in the understanding of meningeal microanatomy and the efficacy of meningeal arterial embolization in the management of chronic SDH (cSDH) could underline the pivotal role of the dural vascular plexus (DVP) in both development and treatment of SDH.

Materials and Methods First, we report the case of a 65-year-old woman presenting with a 2-month history of worsening headache. CT showed bilateral cSDH as well as dilated left parietal cortical veins. Digital substracted angiography (DSA) confirmed the diagnosis of dAVF. The shunt was directly located onto a tortuous left parietal cortical vein (Cognard type 3) with secondary drainage within the superior longitudinal sinus and was fed by bilateral middle meningeal arteries (MMA). After an unsuccessful transarterial embolization, surgical clipping and section of the fistula allowed for dearterialization of the draining vein as proven by visual examination and peroperative Doppler. Follow-up DSA and CT confirmed the exclusion of the shunt and the disappearance of the SDHs over the following months. Second, we illustrate the efficacy of MMA embolization for cSDH through the case of a 58-year-old man who developed a 2 cm thick left SDH under anticoagulant regimen for deep venous thrombosis. After its termination, imaging follow-up showed recurrent bleeding and development of membranes under single antiplatelet therapy, in keeping with chronification. Left MMA embolization was performed by selective microparticles injections and follow-up CT showed significant reduction of the subdural collection and its hyperdense compound.

Discussion The DVP is a rich capillary network located in the dural border cell layer preferentially found near the midline. It is fed by branches of the middle meningeal artery and drains into dural sinuses separately from the connecting end of bridging veins. It could represent a central hub in subdural collections development and management. In dAVF, sinus hypertension might increase DVP hydrostatic pressure and thus result in subdural collection and bleeding. Disconnection of the shunt leads to pressure normalization within the cortical vein and therefore the sinus, indirectly eliminating the driving factor of DVP hyperpressure. In the setting of common cSDH, bleeding might be initiated by traction on the inner dural bordel cell layer. Inflammatory response secondary to bleeding results in neovascularized membranes responsible of further leakage and microbleeds. MMA embolization might decrease DVP pressure (in addition to membrane devascularization?) and favour cSDH resorption.

Conclusion We hypothesize the crucial role of the DVP in both development and management of subdural collections as depicted by cases of dAVF-related SDHs and common cSDH treated by MMA embolization. We further provide original anatomical drawings illustrating the underlying convergent mechanisms.