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Case Report

The dural vascular plexus in subdural hematoma: Illustration through a case of dural arteriovenous fistula

Julie Lebeau[1], Martin Moïse[2], Pierre Bonnet[3], Didier Herman Martin[1], Bernard Otto[2], Felix Scholtes[1,4]

Departments of [1]Neurosurgery, [2]Medical Imaging, [3]Human Systematic Anatomy and [4]Neuroanatomy, University of Liège, Liège, Belgium.

E-mail: Julie Lebeau - jlebeau@chuliege.be; Martin Moïse - martin.moise@chuliege.be; Pierre Bonnet - p.bonnet@uliege.be; Didier Herman Martin - didier.martin@chuliege.be; Bernard Otto - bernard.otto@chuliege.be; *Felix Scholtes - felix.scholtes@uliege.be

***Corresponding author:**
Felix Scholtes,
Departments of Neurosurgery and Neuroanatomy, University of Liège, Liège, Belgium.

felix.scholtes@uliege.be

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ABSTRACT

Background: The initiation of chronic subdural hematoma (cSDH) is traditionally explained by rupture of bridging veins. Recent descriptions of the embryology and anatomy of the meninges and their vascularization, however, point to the dural vascular plexus (DVP) as a plausible origin of cSDH. This dural plexus is supplied by meningeal arteries. Their endovascular occlusion is efficient in cSDH treatment. Dural arteriovenous fistulae (dAVF) may also present with subdural hematoma.

Case Description: A 65-year-old female patient presented with parietal parasagittal dAVF and bilateral cSDH requiring surgical disconnection followed by complete clinical and imaging resolution of dAVF and cSDH.

Conclusion: In common cSDH, pressure in the DVP may be normal and subdural bleeding may occur due to mechanical traction on the DVP. In the setting of dAVF, it may be the increase in pressure due to the fistula, within the DVP, that causes subdural hematoma. The DVP, supplied by meningeal arteries, thus not only allows for convergent pathophysiological explanation of subdural bleeding in both cSDH and dAVF but may also be the actual target of the emergent endovascular treatment of cSDH trough meningeal artery embolization.

Keywords: Bridging veins, Dural arteriovenous fistula, Dural vascular plexus, Meningeal artery embolization, Subdural hematoma

INTRODUCTION

Intracranial dural arteriovenous fistulae (dAVF) are shunts between meningeal arteries and cerebral veins which result in cerebral venous hypertension. Most become symptomatic through parenchymal hemorrhage or nonhemorrhagic neurological deficits associated with edema or ischemia.[8,15] A minority of cases presents with subdural hematoma.[6,20] Subdural bleeding, in the setting of dAVF or not, has traditionally been attributed to bridging vein rupture. This concept has been challenged by recent advances in the understanding of meningeal microanatomy, as well as the efficacy of meningeal arterial embolization in the treatment of chronic subdural hematoma (cSDH). We describe a case of chronic SDH due to dAVF, integrating these recent concepts.

CASE PRESENTATION

A 65-year-old woman, without known head trauma, craniotomy, or dural sinus thrombosis, presented with a 2-month history of headache and neck pain which had progressively worsened

and were associated with nausea over the 5 preceding days. There was no neurological deficit at examination. Cranial computed tomography (CT) and magnetic resonance imaging showed bilateral cSDH, as well as abnormal left parasagittal vascular structures. Digital subtraction angiography confirmed these to be a dAVF, fed bilaterally by parieto-occipital branches of the middle meningeal artery (MMA) draining directly into a unique dilated left parietal cortical vein without venous ectasia and secondary drainage within the superior longitudinal sinus (Cognard Grade 3) [Figure 1].

An endovascular attempt to disconnect the fistula failed because of difficulties navigating within the draining vein and failure to reach the shunt point with injected liquid embolization agent from the arterial side. Postembolization CT showed recent hemorrhage within the left subdural collection in the absence of clinical deterioration. The fistula was approached through a left parietal craniotomy and located within a group of parasagittal calcifications. After temporary clipping, demonstrating dearterialization of the draining vein, the fistula was definitively clipped and sectioned. The deflated draining vein's color changed, as expected, from bright red to dark purple [Figure 2] and intraoperative Doppler confirmed the disappearance of arterial-type flow within those vessels. Postoperative neurological status was unchanged. Follow-up angiography showed no persisting shunt and follow-up CT the regression of the subdural hematoma over the following months [Figure 2].

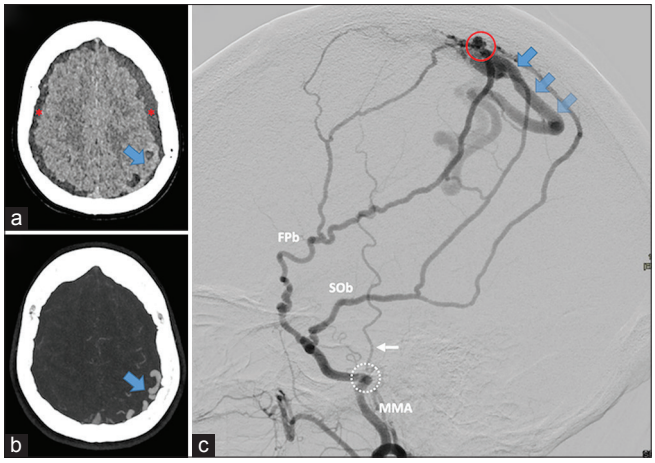

Figure 1: Preoperative imaging. (a and b) Head computed tomography scan. A (noncontrast acquisition): bilateral cSDH (red asterixis) and left extra-axial structures suspected to be abnormal vessels (blue arrow). (b) (Maximum Intensity Projection [MIP] reconstruction from CT angiography): the structures are identified as dilated cortical veins (blue arrow). C (left middle meningeal artery [MMA] digital subtracted angiography [DSA], lateral view): dAVF depending on frontoparietal and squamo-occipital MMA branches (respectively, FPb and SOb). The shunt (red circle) is directly on a tortuous left parietal cortical vein (blue arrows) with secondary drainage within the superior longitudinal sinus (Cognard Type III dAVF). The foramen spinosum (white dotted circle) and the temporal artery (white arrow) are depicted as anatomical landmarks.

DISCUSSION

Subdural hematoma in dAVF has traditionally been attributed to ruptured arterialized bridging veins.[1,3,19,20] However, first, this does not explain subdural hemorrhage in Cognard Type 1 fistula with a direct connection between the meningeal artery and the sinus without cortical venous reflux.[4,10,20] Second, although the bridging vein rupture hypothesis prevailed for a long time in common cSDH, the

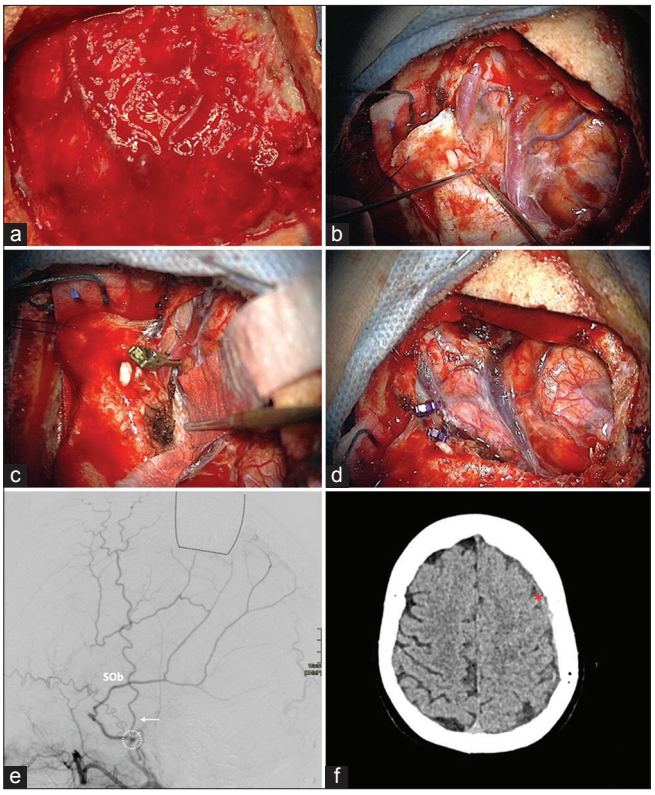

Figure 2: Intraoperative images of different stages of the operation (A-D) and postoperative imaging. (a) Turgescent dural vessels. (b) Intradural view of turgescent veins located within a group of parasagittal calcifications. (c) Transient clipping of the posterior vein: deflation and color change indicating the dearterialization of the draining veins. (d) Clipping and division of the coagulated vein with macroscopic normalization of the venous system. (e) (Lateral view of postoperative MMA DSA, 18 days): no residual early venous opacification. Now that the shunt has been disconnected, the occipital component of the SOb is more clearly observed. The proximal part of the FPb had been occluded during the embolization attempt. The localization of the craniotomy flap (back dotted lines), the foramen spinosum (white dotted circle), and the temporal artery (white arrow) are depicted as anatomical landmarks. (f) (Noncontrast CT, 34 days): regression of the left SDH (red asterisk).

latter appears to actually originate from the dural vascular plexus (DVP). This is a rich capillary network located in the dural border cell layer, and particularly near the midline, fed by branches of the MMA [Figure 3].[2,13,16,17,18]

Common cSDH mainly occurs in elderly patients with relatively atrophic brains. Bleeding may be initiated by traction on the inner dural border cell plexus in these aging meninges [Figure 3].[17] The subdural accumulation of blood can lead to an inflammatory response, resulting in the formation of neomembranes surrounding the hematoma.[5,17] Angiogenesis in these neomembranes with leaky capillaries sustains cSDH through repeated microbleeds.[5,17,18] Their neovascularization also appears to depend on meningeal arterial supply.[5,7] This has been used as a rationale for MMA embolization in the treatment of cSDH and to explain its observed clinical efficacy.[7,17] The microanatomy of the meninges illustrated in the present case [Figure 3] indicates a plausible alternative or complementary explanation: the decrease in DVP pressure resulting from endovascular meningeal artery occlusion may decrease recurrent bleeding into the cSDH which might help cSDH regression.

In the setting of dAVF, on the other hand, subdural bleeding is attributed to venous hypertension resulting from the arteriovenous shunt. Since the DVP drains directly into the cerebral sinus, separately from the sites of entry of the bridging veins,[9,18] localized sinus hypertension could increase DVP pressure [Figure 3] and, thus, result in subdural bleeding [Figure 3d]. A local pressure increase in the sinus has indeed been demonstrated in dAVF,[12] and global sinus hyperpressure, resulting in clinical intracranial hypertension, can result from dAVF, including Type 1.[4,11]

Therefore, increased pressure within the DVP provides a plausible explanation for the occurrence of subdural hematoma in the setting of dAVF, including the case we describe, which is only the second case of bilateral SDH associated with dAVF reported in the literature to the best of our knowledge.[14] In cases of dAVF responsible for subdural hematoma, the disconnection of the shunt is expected to lead to normalization of pressure within the cortical vein, thus the sinus, and therefore DVP, eliminating the driving factor for bleeding. In the present case, we did, indeed, observe definitive regression of subdural hematoma after surgical dAVF disconnection.

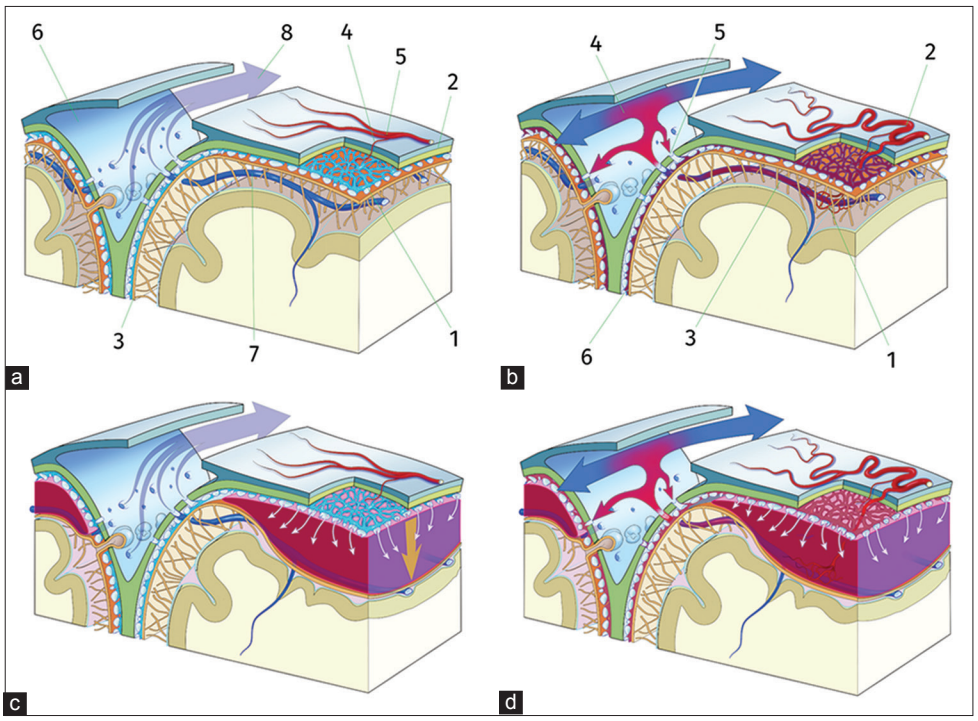

Figure 3: (a) The DVP (1) is located in the dural border cells layer between the dura mater (2) and the arachnoid membrane (3). It is fed by branches (4) of the meningeal artery (5) and it drains directly into the superior sagittal sinus (6) and its lateral pouches, independently from cortical bridging veins (7), with unidirectional flow (8). (b) Common cCSH: traction (arrow) on the dural border cells layer, for example, with minor head trauma and subsequent blood accumulation, leading to hemorrhage from the inner dural plexus (white arrows) creating cSDH by disruption of the border cell layer joining arachnoid membrane and dura mater. (c) Cognard Type III dAVF (1) between the tortuous meningeal artery (2) and a cortical vein. Blood flow is arterialized (3), leading to local pressure increase in the SSS (4) and creating an high pressure reflux (5) into the DVP (6) and abnormal blood flow in the SSS. (d) Increase in DVP pressure leads to rupture and, thus, subdural bleeding.

CONCLUSION

The DVP is a plausible origin of subdural hematoma. The present case of bilateral SDH due to dAVF can satisfactorily be explained by sinus hypertension that increased pressure in the DVP. Changes in the pressure regime of the DVP may also play a role in the efficacy of the endovascular meningeal arterial devascularization in common cSDH.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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