ORIGINAL ARTICLE

Cranial dural arteriovenous shunts. Part 2. The shunts of the bridging veins and leptomeningeal venous drainage

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Abstract Leptomeningeal venous drainage of cranial dural arteriovenous fistulae is the most important determinant of adverse clinical course. Factors that predispose to its occurrence have not been adequately addressed in the literature. In the present study, we investigated the relation of shunt location to the development of leptomeningeal venous drainage, with regard to the bridging veins. Angiographic data of 211 consecutive patients with cranial dural arteriovenous fistulae treated over 19 years were analyzed. Dural shunts with leptomeningeal venous drainage were found in 107 patients; of these, 71 patients had pure leptomeningeal venous drainage (Borden type 3). The angioarchitecture of the shunt, including pattern of arterial feeders, relation with the bridging veins, primary venous drainage, and venous outflow restrictions were recorded. After analysis of the 71 Borden type 3 shunts with exclusive leptomeningeal venous drainage, three patterns emerged. The commonest was the fistula engaging a bridging vein that had lost its connection to the parent sinus into which it previously drained; it was characterized by an arterial network of feeders converging onto the wall of a bridging vein, with leptomeningeal venous reflux. The other patterns were those of "isolated" sinus segment characterized by arterial feeders converging on to the wall of the dural sinus with leptomeningeal venous reflux following the opacification of the sinus and fistulae in the vicinity of the cribriform plate with two subtypes. The main angioarchitectural features of the 36 Borden type 2 shunts with mixed sinusal-cortical venous drainage were the presence of a diffuse arterial network

of vessels converging onto a site in the wall of the dural sinus, with leptomeningeal venous reflux following the opacification of the sinus. In this group, four exceptions were noticed with arterial feeders converging onto a bridging vein and having a mixed venous drainage to the cortical venous system and the sinuses. We concluded that the exact location of the shunt with regard to the bridging veins is a key factor in the development of leptomeningeal venous drainage. Cranial dural arteriovenous fistulae (CDAVFs) of either Borden type 2 or 3 do not constitute a homogeneous group. The great majority of these shunts present thrombotic phenomena.

Keywords Dural arteriovenous shunt \cdot Dural arteriovenous fistula \cdot Bridging vein \cdot Leptomeningeal drainage \cdot Cortical venous reflux

Introduction

Cranial dural arteriovenous fistulae (CDAVFs) are lesions located within the dura matter of the skull commonly near the dural sinuses. Among the described varieties, fistulae with leptomeningeal venous drainage (LVD) carry the highest risk of aggressive clinical presentation [1, 16, 18, 5, 10, 21]. These lesions are currently described as type 2 and 3 by Borden et al.[4] and type IIb, IIa+b, III, IV, and V by Cognard et al.[8]. Although they share the common feature of LVD, they may drain either into both a sinus and a leptomeningeal vein or only into the leptomeningeal venous system.

However, the question of what determines the venous drainage of a CDAVF has not been sufficiently addressed. The development of LVD has often been associated with venous outflow restrictions (VOR) due to

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partial or complete thrombosis or stenosis of the involved venous sinus, high flow shunt, and the location of the lesion in relation to the cranial epidural spaces. According to the consideration of the cranial epidural spaces, lesions of the lateral epidural space had LVD in 100 % of the studied cases, whereas lesions of the ventral and dorsal epidural spaces developed LVD in the presence of associated VOR, usually thrombosis [15, 7, 14]. However, cases of shunts of the dorsal or ventral epidural space with CVD but without associated outflow restriction have also been described [3, 15].

This study was designed to further investigate the influence of exact shunt location on the development of LVD. In particular, we differentiated CDAVF primarily located in the wall of a sinus from those located primarily in the wall of a bridging vein (BV), which normally drains into the sinus [13].

Materials and methods

From a series of 211 consecutive patients with CDAVF treated in the Department of Neuroradiology, University Hospital Zurich, during the last 19 years, 107 patients for whom complete angiograms were available and had lesions with LVD [2] were analyzed. Mean age was 57 years and male/female ratio 71:36. Among them, 71 patients had dural arteriovenous shunts with only LVD (Borden type 3 and Cognard type III, IV, and V), whereas the remaining 36 patients had lesions with venous drainage to both a sinus and LVD (Borden type 2 and Cognard type IIb and IIab). Cases of pediatric and/or congenital CDAVFs were excluded.

The angiographic images of all cases were analyzed, focusing on the exact location of the shunt and its relation to the bridging or emissary veins (EV). For this purpose, morphologic aspects of the shunt's angioarchitecture, including its pattern of network of arterial feeders, the relation of this network and shunt site with the BVs, and the sequence of contrasting vascular structures during selective and superselective injections were recorded. The structure which opacified first and with the highest contrast density was defined as the primary drainage. Associated VOR as complete or partial sinus and/or BV thrombosis or stenotic appearance were also recorded.

Results

The common features of the group of 36 patients with Borden type 2 lesions were the diffuse pattern of the feeding arterial network and its convergence to a certain segment of the sinus wall (sinus-based shunt). As a rule, the draining BVs varied in distance from the shunt site, and the sinus was opacified prior to the BVs (Fig. 1).



Fig. 1 External carotid injection opacifying a sinus-based dural shunt of the distal sigmoid sinus, with venous drainage to the ipsilateral jugular bulb, the posterior condular vein and retrograde to the proximal sigmoid and distal transverse sinus, and through a temporal BV to the cortical venous system. The shunt site is clearly distinct from the site of leptomeningeal venous reflux

There were four exceptions. The first was a shunt next to the superior sagittal sinus (SSS) with the sinus faintly opacified and almost simultaneously with a parietal BV. The feeders were converging rather to the parietal BV than to the wall of the sinus. The second and third exceptions were shunts at the ostium of a temporal BV next to the transverse sinus in which faint opacification of the sinus after the BV was clearly evident after selective (in one shunt) or superselective (in the other) injection of the external carotid and the distal middle meningeal feeder, respectively (Fig. 2). The fourth exception was a shunt with feeders converging to the superior petrosal vein, with primary drainage to the BV and secondary drainage into the superior petrosal sinus and through the cavernous sinus to the inferior petrosal sinus.

Complete sinus thrombosis combined with stenothrombotic appearance of the patent segment of the sinuses was found in 15 (41.5 %) patients, whereas a stenothrombotic appearance without occlusion was recorded in another 15 patients (41.5 %). In total, 83 % of lesions of this group presented thrombotic phenomena detectable in angiography. Of the remaining six cases, four showed evidence of an extensive and high flow shunt. In one case, an anatomic, non-thrombotic disconnection of the transverse sinus from the torcular was noted.

The 71 cases with exclusive LVD fell into three groups of lesions: One group consisted of lesions with sinus-based shunts, a second group consisted of BV-based shunts, and



Fig. 2 a Selective external carotid injection of a temporal bridging veinbased dural shunt. The middle meningeal feeders converge to the BV and the shunt drains primarily into the BV. **b** In the superselective injection, a "leak" to the parent sinus demonstrated by the faint and slightly later opacification of the distal transverse sinus is visible, presumably showing that the BV exit to the sinus is not completely occluded

the last group was the so-called shunts of the anterior cranial fossa.

The first group included 18 patients with a shunt located in the sinus wall, secondarily presenting an LVD due to the complete disconnection of that segment of the sinus from the rest of the sinuses. We called the lesions of this group "isolated sinus shunts" (ISS). Two types of disconnection were found; the first type was associated with occlusion of the sinus proximal and distal to the ostium of the BV, most likely due to thrombosis (Fig. 3). The second type was due to an occlusion of the distal segment of the sinus associated with a preexisting proximal anatomical isolation of the sinus, remote from the site of the lesion (Fig. 4). The networks of feeders were converging on the sinus wall, the sinus was opacified first, and the BV(s) was/were located at a varying distance from the shunt site. The preferred sites of these shunts were the transverse and sigmoid sinuses.

The second group included 44 cases with involvement of a BV and exclusive drainage into the leptomeningeal venous system. We called the lesions of this group "bridging vein shunts" (BVS). The detailed analysis of the selective and superselective angiographic images showed that the feeders were converging to the BV and the adjacent sinus or plexus was not primarily opacified (Fig. 5). The preferred site in this group was the tentorium (28 lesions or 63.5 %), followed by the parietal parasagittal BV shunts and the medullary BV shunts. Among the tentorial lesions, 15 were petrosal BV shunts.

Nine patients had a shunt at the anterior cranial fossa. Two different subtypes were distinguished: The first subtype is characterized by the convergence of the ethmoidal feeders to a BV (normally emptying into the SSS) located above and anteriorly to the level of the lamina cribriformis (Fig. 6). Three shunts of our series belonged to this subtype, which for descriptive reasons we called "ethmoidal BV shunts" but otherwise are identical to the rest of BV shunts of the second group. The second subtype is characterized by the convergence of the ethmoidal feeders to a vein located at the level of



Fig. 3 An example of the isolated sinus shunt (ISS) variety of the sinusbased shunts group, due to occlusion of the distal transverse (in this case) sinus on either side of the arterialized segment



Fig. 4 An example of the isolated sinus shunt (ISS) variety of the sinusbased shunts group, due to occlusion of the distal sigmoid sinus at the shunt site and anatomic disconnection of the ipsilateral transverse sinus from the contralateral transverse and superior sagittal sinus at the level of the torcular. The retrograde flow is conducted to the ipsilateral transverse sinus and straight sinus as well as to the superficial cortical veins, which drain secondarily to the SSS

the lamina cribriformis (Fig. 7). Four shunts of our series belonged to this subtype, which we called "cribrosal dural shunts" (CDS). In two cases, the quality of angiographic



Fig. 6 An example of an ethmoidal bridging vein-based dural shunt, supplied by the anterior ethmoidal artery. The superselective injection shows the shunt site and the cortical draining vein, which are located anteriorly and superiorly to the level of the lamina cribrosa. This vein corresponds to a BV that normally would drain to the lowermost end of the SSS

images did not permit a precise evaluation. All lesions emptied secondarily either to the SSS and/or through an olfactory vein to the basal vein.



Fig. 5 An example of a bridging vein-based dural shunt, supplied by the occipital and middle meningeal artery and located in a posterior parietal BV, which through superficial venous anastomoses, empties secondarily to the straight sinus



Fig. 7 An example of an ethmoidal cribrosal dural shunt, supplied by the anterior ethmoidal artery. The superselective injection shows the shunt site, which is located at the level of the lamina cribrosa. The draining vein, through a venous tangle that most likely lies around the olfactory bulb, empties to the olfactory (cortical) vein

Discussion

The analysis of all our cases with LVD, based on angioarchitectural characteristics, helped in distinguishing features, beyond those described in the known classification systems, which may explain the appearance of LVD. It also defined groups of lesions such as BV shunts, so far not clearly distinct. A BV shunt is defined as a shunt involving the dural segment of the BV at the BV-sinus junction. Such a shunt will have always a direct LVD, which by definition is a (thus defined) primary retrograde LVD. How much long or short the retrograde-flow course is depends entirely on the individual local venous anastomotic background. If near the involved BV, an efficient collateral venous connection channels the retrograde flow into a recipient of another BV, then the flow will drain secondarily, in an anterograde fashion into a nearby sinus. If no efficient collateralization is anatomically available, then the retrograde-flow course to a favorable sinus-exit will be longer. The pathomechanism of this shunt type is now known. We do not know if in all cases the original lesion was only located in the BV, and not in the sinus wall. Theoretically, some BV shunts could be the result of partial thrombosis of a previously more extensive sinus-based shunt. Partial thrombosis of the sinus lumen in a sinus-based lesion can be a self-treating process since the venous component of these lesions is the key for their treatment. Such a thrombosis would leave behind a more focal lesion with its own draining exit (i.e., the BV), which keeps the shunt active. However, the fact that among our cases, no shunt with mixed BV and sinus wall location was found speaks against such a scenario.

This distinct entity of CDAVFs was previously described as "extrasinusal" shunt [23]; later, focusing on the medullary and petrosal shunts, Mitsuhashi et al.[22] used the term "medulla bridging vein-draining fistula" for the former, they noticed the exact shunt location on the petrosal vein for the latter and highlighted their similarities with spinal dural shunts. Nevertheless, the literature so far is not clear that the BV shunts constitute a distinct group of shunts, located on the BV itself with the characteristics described above and being the most important group among those with LVD.

The analysis of the cases classified as Borden type 2 lesions disclosed that almost all these shunts are located in the wall of the sinus and not in the wall of a BV. The four cases bearing characteristics of a BV shunt with drainage through both the leptomeningeal venous system and the sinuses [22] show that BV shunts only rarely present a patent connection of the BV with the corresponding sinus when they become symptomatic. The analysis of the Borden type 3 lesions and the identification of almost all BV shunt cases as part of the group with exclusive LVD essentially confirm the above observation. In other words, in most of BV shunts, the exit of the BV to the sinus was occluded at the time of diagnosis. This may imply that most BV shunts become symptomatic after the connection of the BV to the sinus is occluded. The fact that the first case of the four cases was symptomatic at the time of diagnosis does not necessarily contradict this conclusion since the peculiar angiographic features of the shunt can explain the symptoms. It is known that some cerebral areas are devoid of notable venous anastomoses [11]. In fact, as the angiographic investigation of the first case showed, the tributaries of the involved BV did not display any anastomoses with neighboring superficial veins, and therefore the shunt drained retrograde and exclusively into the gyral medullary venous system. This may explain both the mode of presentation (epilepsy) and why the lesion became symptomatic before the occlusion of the BV exit to the sinus. The second of the four cases with faint opacification of the sinus in the superselective injection most likely corresponds to a lesion with a nearly completely occluded BV exit to the sinus. Interestingly and in accordance with the above concept, this shunt was still asymptomatic and incidentally discovered. The third and fourth cases presented with benign symptoms (tinnitus and headache). The LVD observed in the Borden type 2 lesions was accompanied by or related to a stenosis or partial thrombosis or complete thrombosis of the sinus in 83 % of the cases, showing that thrombosis is a key element for the development of LVD in these shunts.

Cognard et al. [8] defined the type 3 CDAVFs as those draining directly into a leptomeningeal vein; in the definition of the Borden [4] type 3 lesions, on the other hand, the type of the isolated sinus due to thrombosis on both sides of the arterialized site was also included. The three different groups that we described, all classified as lesions type 3, show that lesions of this type are inhomogeneous. The LVD observed in the group of ISS was clearly related to the thrombosisisolation of the sinus and the redirection of the flow into the leptomeningeal veins, an entity already well known [4]. The LVD observed in the group of BV shunts was primarily related to the fact that the shunt was located in the wall of the BV at the BV-sinus junction and secondarily to the disconnection of the BV from the sinus due to thrombosis of the BV exit, an almost universal characteristic of BV shunts at the time of diagnosis. The location of a shunt in the BV, as primary factor for LVD development, has not been clearly stated in the literature. The LVD observed in the cribrosal subtype of ethmoidal shunts was apparently related to the location of the shunt on the wall of a vein, which represents a direct durocortical venous connection, similar to a bridgingemissary vein configuration, seen in the spinal cord (and intracranially seen only in this area). Thrombosis-occlusion of this connection is almost always observed at the time of diagnosis [12]. From the above, it becomes clear that all type 3 lesions are accompanied by thrombotic phenomena. Therefore, the vast majority—94.3 % in our series—of CDAVFs with LVD (Borden types 2 and 3) are associated with thrombosis (complete or partial), in contradiction with the oftencited statement that the majority of CDAVFs do not have evidence of preexisting or coexisting thrombosis [20] and in accordance with previous reports advocating consistent accompanying thrombotic phenomena [26]. This raises the old question of a potential cause-effect relation between thrombosis and CDAVFs [17, 24, 6], which is beyond the scope of this report.

The pattern of the network of feeding vessels, the sequence of enhancing structures, the shunt site, and its relation with the BV, in ISS lesions, were clearly different in comparison with the BV shunts and this justified their distinction from the angioarchitectural point of view [13].

Precise description of a shunt location, based on the abovementioned features, makes sense because it signifies its angioarchitecture, which seems to be inherent to each particular location. Typically, in the literature of the dural arteriovenous lesions, the characterization of a shunt with exclusive LVD is either connected to a sinus as for instance "transverse sinus CDAVF type 3" or to a dural structure as "tentorial CDAVF type 3" or to a specific anatomic location as "ethmoidal CDAVF" or "jugular foramen CDAVF," etc. The former designation has theoretically the potential to express precisely the location of a shunt. However, the same term may indicate either a shunt of an isolated sinus with indirect LVD or a shunt primarily located on a BV, which normally would drain into the transverse sinus, but its exit is now thrombosed. Functionally, both types present exclusive LVD but in terms of exact location and relation to the BV and VOR are different. In the first case, the lesion is located in the wall of the sinus, and the sinus is the primary site of the disease and its lumen is thrombosed on both sides of the arterialized segment, whereas in the second case, the shunt affects the wall of the BV in which is located and the exit of the BV to the sinus is (usually) occluded and the sinus itself remains patent or only partially thrombosed. In the first case, the shunt is in fact a sinusal lesion and should be called in our example "isolated transverse sinus CDAVF" for the sake of clarity and preciseness, whereas in the second case, it is a disease of the BV and it should be called accordingly, addressing the corresponding BV.

Regarding the characterization based on a dural structure as for instance "tentorial shunt," normally, it should be straightforward (yet vague), with this term to describe only shunts of the various BVs, which drain into the tentorial sinuses. But due to its current usage, the difficulty is similar because under this term, a shunt of a superolateral cerebellar hemispheric BV is included with a shunt of a persisting tentorial sinus or with a tentorial shunt of an inferior temporal BV or even with a shunt of a peduncular BV at the tentorial incisura. Additionally, the petrosal shunts are often included in this group, and they can be either superior petrosal BV shunts (often) or superior petrosal sinus shunts (rarely). When the shunt involves directly a Labbé vein, which may drain to a tentorial sinus before it exits a short distance into the transverse sinus, the designations as "transverse sinus CDAVF" or "tentorial CDAVF" can be deemed equally "correct" and therefore nonspecific and imprecise. Thus, discrepancies among studies in the incidence of described locations and in correlations between location and clinical presentation as well as confusion due to incongruity of characterization should be suspected even if not clearly highlighted so far. It is important and very useful to notice that all the above-described types of tentorial lesions, except the one of the superior petrosal sinus, which should describe a sinus-based lesion, are BV shunts. Therefore, it would be both convenient and precise if the designation of "tentorial CDAVF" was reserved only for BV shunts of the tentorium, which in fact constitute the real tentorial dural lesions.

A similar concept applies for the shunts in the area of the anterior cranial fossa since these lesions present a peculiar venous anatomy. The presence of two different types of veins and the absence of a strictly defined emissary vein, in the floor of anterior cranial fossa, make the anatomy of the so-called ethmoidal CDAVFs peculiar and the understanding of their nature seemingly problematic. A shunt of an orbitofrontal BV is located almost at the level of the floor of the anterior fossa but anteriorly and slightly superiorly to the lamina cribriformis. A shunt of the dural ethmoidal veins which are connected directly with the leptomeningeal veins of the olfactory bulbs [25, 19, 27] is located at the level of the lamina cribriformis. Although both present a similar architecture of the arterial feeding network and venous drainage, their exact location and the nature of the underlying veins are different. In either case, their characterization as ethmoidal or anterior cranial fossa CDAVFs does not describe precisely the nature of the lesions. Whether this distinction corresponds to a distinct clinical behavior remains to be clarified. We suggest the more precise term ethmoidal BV shunts for lesions involving a BV to the lowermost SSS, in order to distinguish them from shunts at the level of the lamina cribriformis, termed "cribrosal shunts."

Furthermore, the venous drainage of a lesion type 3 is usually described as "drainage through a leptomeningeal or subarachnoid or cortical vein." Such a vein is described as if it was a no-name vein, whereas in reality, these veins are often high-profile vessels, with a specific draining territory, specific functional significance according to the location and the particular anatomic configuration and variation and perhaps with specific veno-dural interface [9] depending on the location, the size, or the connection to a sinus or to a plexus. In our previous example, a characterization as "CDAVF of the Labbé vein" is precise in terms of anatomic location, equally precise as the term "galenic CDAVF," and signifies by definition the existence of direct and (usually) exclusive LVD.

Conclusion

In addition to the previously described factors, the exact location of the shunt in relation to the BVs is a major determining factor for the development of LVD in CDAVFs. BV shunts present LVD by definition and the vast majority have their exit to the corresponding sinus thrombosed. CDAVFs type 3 is not a homogeneous group. They may be BV-based lesions with LVD without involvement of the sinus; they can be lesions of an isolated sinus due to thrombosis or combined with an anatomic variation, with only venous drainage through the BVs, or they may be lesions at the area of the lamina cribriformis. These subgroups of lesions share the common feature of exclusive LVD, but they have different angioarchitectures. Their different anatomic backgrounds may portend a different clinical expression and natural course, a subject, which needs further investigation. As reported previously, dural shunts with LVD are frequently associated with sinus thrombosis and/or stenosis and, in our series, were seen in almost all cases.

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Conflict of interest We declare that we have no conflict of interest.

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References

- Awad IA, Little JR, Akarawi WP, Ahl J (1990) Intracranial dural arteriovenous malformations: factors predisposing to an aggressive neurological course. J Neurosurg 72(6):839–850
- Baltsavias G, Valavanis A (2014) Endovascular treatment of 170 consecutive cranial dural arteriovenous fistulae: results and complications. Neurosurg Rev 37(1):63–71
- Barnwell SL, Halbach VV, Dowd CF, Higashida RT, Hieshima GB, Wilson CB (1991) A variant of arteriovenous fistulas within the wall of dural sinuses. Results of combined surgical and endovascular therapy. J Neurosurg 74(2):199–204
- Borden JA, Wu JK, Shucart WA (1995) A proposed classification for spinal and cranial dural arteriovenous fistulous malformations and implications for treatment. J Neurosurg 82(2):166–179
- Castaigne P, Bories J, Brunet P, Merland JJ, Meininger V (1976) Meningeal arterio-venous fistulas with cortical venous drainage. Rev Neurol 132(3):169–181
- Chaudhary MY, Sachdev VP, Cho SH, Weitzner I Jr, Puljic S, Huang YP (1982) Dural arteriovenous malformation of the major venous sinuses: an acquired lesion. AJNR Am J Neuroradiol 3(1):13–19

- Cognard C, Casasco A, Toevi M, Houdart E, Chiras J, Merland JJ (1998) Dural arteriovenous fistulas as a cause of intracranial hypertension due to impairment of cranial venous outflow. J Neurol Neurosurg Psychiatry 65(3):308–316
- Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, Chiras J, Merland JJ (1995) Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. Radiology 194(3):671–680
- Dagain A (2008) Junction between the great cerebral vein and the straight sinus: an anatomical, immunohistochemical and ultrastructural study on 25 human brain cadaveric dissections. Clin Anat 21: 389–397
- Djindjian R, Merland JJ, Theron J (1978) Superselective arteriography of the external carotid artery. Springer, Berlin, Heidelberg, New York
- Duvernoy HM, Delon S, Vannson JL (1981) Cortical blood vessels of the human brain. Brain Res Bull 7(5):519–579
- Fang YB, Li Q, Yang PF, Huang QH, Zhao WY, Xu Y, Hong B, Liu JM (2013) Treatment of blood blister-like aneurysms of the internal carotid artery with stent-assisted coil embolization. Clin Neurol Neurosurg 115(7):920–925
- Gaston A, Chiras J, Bourbotte G, Leger JM, Guibert-Tranier F, Merland JJ (1984) Meningeal arteriovenous fistulae draining into cortical veins. 31 cases. J Neuroradiol 11(3):161–177
- Geibprasert S, Krings T, Pereira V, Pongpech S, Piske R, Lasjaunias P (2009) Clinical characteristics of dural arteriovenous shunts in 446 patients of three different ethnicities. Interv Neuroradiol 15(4):395– 400
- Geibprasert S, Pereira V, Krings T, Jiarakongmun P, Toulgoat F, Pongpech S, Lasjaunias P (2008) Dural arteriovenous shunts: a new classification of craniospinal epidural venous anatomical bases and clinical correlations. Stroke 39(10):2783–2794
- Houser OW, Baker HL Jr, Rhoton AL Jr, Okazaki H (1972) Intracranial dural arteriovenous malformations. Radiology 105(1): 55–64
- Houser OW, Campbell JK, Campbell RJ, Sundt TM Jr (1979) Arteriovenous malformation affecting the transverse dural venous sinus-an acquired lesion. Mayo Clin Proc 54(10):651–661
- Kosnik EJ, Hunt WE, Miller CA (1974) Dural arteriovenous malformations. J Neurosurg 40(3):322–329
- Lang J (1981) Klinische Anatomie des Kopfes. Neurocranium, Orbita, kraniocervikaler Übergang. Springer, Berlin-Heidelberg
- 20. Lasjaunias P, Berenstein, A., ter Brugge, K. (2001) Surgical neuroangiography: dural arteriovenous shunts, vol 2.2. Springer
- Lasjaunias P, Chiu M, ter Brugge K, Tolia A, Hurth M, Bernstein M (1986) Neurological manifestations of intracranial dural arteriovenous malformations. J Neurosurg 64(5):724–730
- 22. Mitsuhashi Y, Aurboonyawat T, Pereira VM, Geibprasert S, Toulgoat F, Ozanne A, Lasjaunias P (2009) Dural arteriovenous fistulas draining into the petrosal vein or bridging vein of the medulla: possible homologs of spinal dural arteriovenous fistulas. Clinical article. J Neurosurg 111(5):889–899
- Piske RL, Lasjaunias P (1988) Extrasinusal dural arteriovenous malformations. Report of three cases. Neuroradiology 30(5):426– 432
- Piton J, Guilleux MH, Guibert-Tranier F, Caille JM (1984) Fistulae of the lateral sinus. J Neuroradiol 11(3):143–159
- 25. San Millán Ruíz DGP, Rüfenacht DA, Yilmaz H, Fasel JH (2006) Anomalous intracranial drainage of the nasal mucosa: a vein of the foramen caecum? AJNR Am J Neuroradiol 27(1):129–131
- Uranishi R, Nakase H, Sakaki T (1999) Expression of angiogenic growth factors in dural arteriovenous fistula. J Neurosurg 91(5):781– 786
- 27. Whitnall E (1921) The anatomy of the human orbit and accessory organs of vision. R.R. Clark, Edinburgh

Comments

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The authors analyzed the regional venous architecture of CDAVFs from their large case series and clearly demonstrated the bridging veins functioning as drainage system of CDAVFs with high-quality angiographic images. The exact location of the shunt in relation to the BVs is a major determining factor for the development of LVD in CDAVFs. This conclusion well clarifies the diversity of angioarchitecture of this pathology.

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Dr. Baltsavias and colleagues highlight in this manuscript the importance of precise localization of the DAVS with respect to the bridging vein (BV) or the Dural Sinus (DS). The expected drainage pattern associated with each location can be modified by local or regional availability of the draining veins distal to the BV or DS mostly determined by coexisting thrombosis. This frequent association with thrombosis is implied because of the absence of the expected drainage in the region. The authors suggest that an AVS at the level of the BV is more likely to become symptomatic when the to-be expected connection with the adjacent DS is interfered with, likely because of focal thrombosis. Correct analysis of the exact shunting point will enable clinicians to make a more accurate interpretation of the risks involved related to the to-be expected versus the observed drainage pattern of the DAVS.

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The current study investigated the venous drainage of cranial dural arteriovenous fistulas and sheds light on the influence of exact shunt location on the development of leptomeningeal venous drainage, with regard to the bridging veins. Based on a considerable large number of cases, the authors provided excellent new data that may further refine the current classification systems. Furthermore, their findings may provide important clues to better define the natural course of these lesions.