Cerebral Developmental Venous Anomalies: Current Concepts

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Cerebral developmental venous anomalies are the most frequently encountered cerebral vascular malformation, and as such, are frequently reported as fortuitous findings in computed tomography (CT) and magnetic resonance imaging (MRI) studies. Developmental venous anomalies (DVAs) are generally considered extreme anatomical variations of the cerebral vasculature, and follow a benign clinical course in the vast majority of cases. Here we review current concepts on DVAs with the aim of helping clinicians understand this complex entity. Morphological characteristics that are necessary to conceptualize DVAs are discussed in depth. Images modalities used in diagnosing DVAs are reviewed, including new MRI or CT techniques. Clinical presentation, association with other vascular malformations and cerebral parenchymal abnormalities, and possible physiopathological processes leading to associated imaging or clinical findings are discussed. Atypical forms of DVAs are also reviewed and their clinical significance discussed. Finally, recommendations as to how to manage asymptomatic or symptomatic patients with a DVA are advanced.

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The term cerebral "developmental venous anomaly" (DVA), coined by Lasjaunias et al.,[1] is now widely used as a synonym for venous angioma, cerebral venous malformation, or cerebral venous medullary malformation. DVAs are classified as a type of cerebral vascular malformation, along with capillary telangiectasias, cavernous malformations (CMs), and arteriovenous malformations (AVMs).[2] DVAs are the most frequently encountered cerebral vascular malformations, with an incidence of up to 2.6% in a series of 4,069 brain autopsies.[3] They are often observed fortuitously during routine brain imaging using computed tomography (CT) and magnetic resonance imaging (MRI). DVAs are encountered both in the pediatric and adult populations, with a slight predominance in males.[4,5]

Normal Cerebral Angioarchitecture

A basic knowledge of the normal venous architecture of the brain helps understanding the morphology and the mode of formation of DVAs. Schematically, the veins of the cerebral hemisphere can be divided in two systems (Fig 1)[6–8]:

1. A superficial system draining the cerebral cortex and the subcortical white matter (ie, the outer centimeter of superficial white matter). The superficial system collects into the pial veins that are visible on the surface of the brain;
2. A deep system[8] constituted of the deep medullary veins draining the deep white matter and the striate body. These veins are tributaries of the internal cerebral vein and basal vein of Rosenthal, mainly through the subependymal veins located along the external and superior aspect of the lateral ventricles. The subependymal veins collect into relatively constant trunks distributed along the lateral ventricle and forming triangular venous convergence points corresponding to specific cerebral regions.[9]

Connections between the cortical and deep venous systems through veins that cross the entire thickness of the brain parenchyma have been described as "intracerebral anastomotic veins" or "transcerebral veins."[6,10,11]

The cerebellar venous architecture has been the subject of fewer studies. The principle of superficial and deep venous systems is, however, also applicable to the cerebellum. The superficial venous system of the various surfaces of the cerebellum and vermis collects into the vein of Galen, into the superior petrosal vein and sinus, and into the straight sinus, the transverse sinus, and the torcular herophili.[12] The veins of the cerebellar

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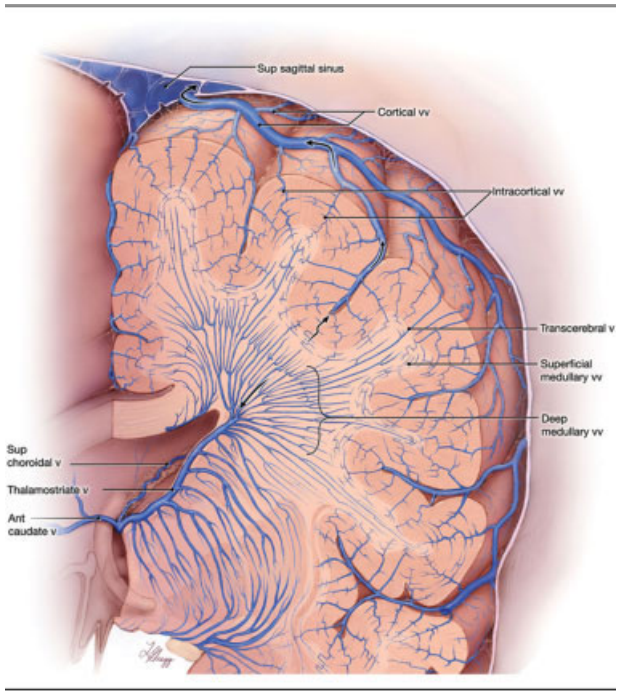

Fig 1. Schematic representation of the venous angioarchitecture, coronal section through the region of the foramen of Monro, anteroposterior view of the left cerebral hemisphere. The superficial venous system draining the cortical and superficial white matter, and the deep venous system draining the deep white matter are depicted. The superficial venous system drains toward the dural venous sinuses by way of the cortical veins, while the deep venous system is collected by the network of subependymal veins (eg, the thalamostriate and anterior caudate veins depicted here) and drains toward the internal cerebral veins, basal vein of Rosenthal, and great vein of Galen. Connections between the two systems may occur through transcerebral medullary veins. The black arrows show the direction of flow of venous blood. Sup sagittal sinus = superior sagittal sinus; sup choroidal v = superior choroidal vein; ant caudate v = anterior caudate vein; v = vein; vv = veins.

white matter, the subependymal region of the fourth ventricle, and the cerebellar nuclei typically converge onto the vein of the lateral recess of the fourth ventricle, one of the tributaries of the superior petrosal vein.[13]

Developmental Considerations

DVAs represent a purely venous entity, an anomalous venous disposition due to the absence of normal pial or subependymal veins. They may be understood as a variation of the venous angioarchitecture described above, where a deep venous territory drains centrifugally toward either the pial veins of the cerebral surface or directly into a dural venous sinus, or, alternatively, where a cortical and subcortical venous territory drains centripetally toward the network of deep subependymal veins. There is still controversy as to how DVAs develop, though it is generally accepted that they form during intrauterine life. Lasjaunias et al.[1] have proposed that DVAs result from an "hemodynamic need," leading to the recruitment of "transhemispheric anastomotic pathways," and must therefore be viewed as anatomical variations. Other authors consider that occlusion, likely by thrombosis, of normal parenchymal veins lead to the formation of DVAs, suggesting a malformative nature of these lesions that should be more correctly designated as medullary venous malformations.[14] Finally, it has also been suspected that DVAs represent the expression of disturbed fetal angiogenesis and regression.[15] Notwithstanding their mode of formation, DVAs generally provide sufficient collateral outflow to the brain parenchyma, as in the vast majority of cases they are not associated with congenital brain parenchymal abnormalities. Rare association between anomalous parenchymal veins, possibly assimilated to DVAs, and migrational brain abnormalities have been established, though in this case the venous anomaly is thought to be a manifestation of the underlying abnormal neuronal migration and not its cause.[16–18]

MORPHOLOGICAL CHARACTERISTICS

DVAs are characterized by a cluster of venous radicles that converge into a collecting vein, resulting in the typical caput medusae appearance of the DVA. The collecting vein crosses a variable length of brain parenchyma to join either the superficial or deep venous system (Fig 2). Occasionally, DVAs may drain into both the superficial and deep venous systems, one of these drainage pathways being predominant. The essential role played by DVAs in the normal cerebral venous drainage is illustrated by cases of catastrophic venous ischemic and hemorrhagic complications resulting from the surgical removal of a DVA.[19,20] In order to prevent such complications, care must be taken to preserve the collecting vein of a DVA during the surgical evacuation of a cerebral hematoma or the resection of a CM located in its vicinity.

Histopathological descriptions of DVAs are scarce and often confusing due to the nonstandardized terminology employed to characterize cerebral vascular malformations, particularly in older publications. Our observations of hyalinized collecting veins with parietal fibrous thickening, absence of elastic lamina, and loosely arranged smooth muscle layers are consistent with the few ultrastructural studies currently available in the literature (Fig 3).[21,22] The clinical significance of these findings is discussed below.

A stenosis is commonly observed on the collecting vein of DVAs.[4,18,23] It is usually located at the point of penetration of the collecting vein into the draining dural venous sinus (see online supplemental material Fig 1). Stenoses, therefore, occur more frequently in superficially draining DVAs. The role of a collecting vein

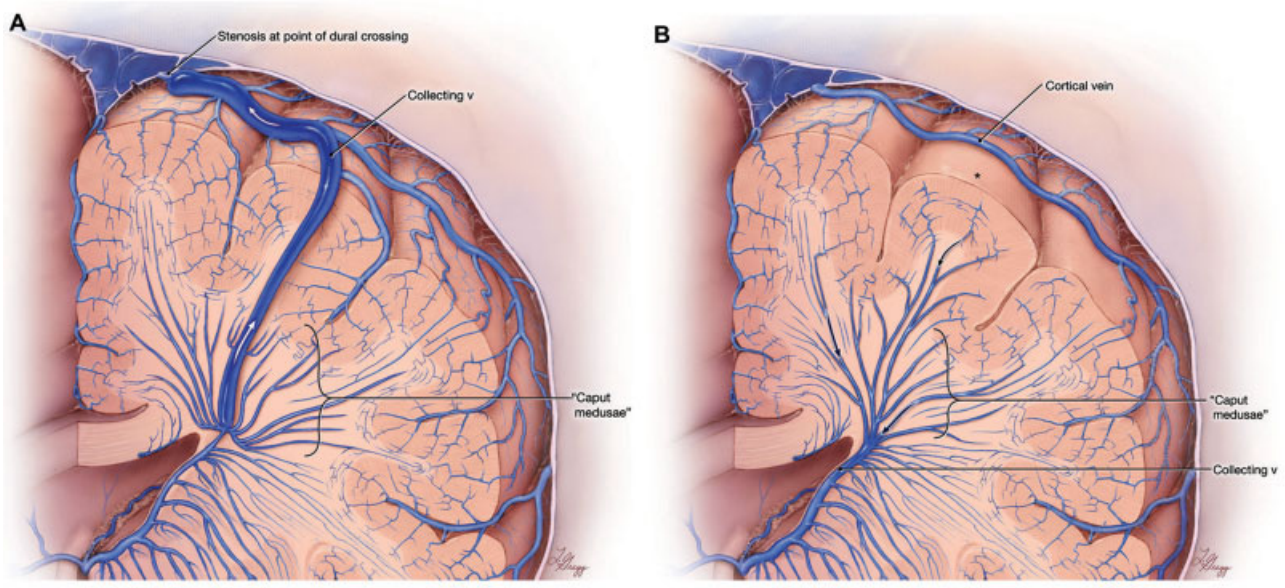

*Fig 2. (A) Schematic representation of the venous angioarchitecture of a superficially draining DVA, same view as Fig 1. In this case, the deep white matter principally drains in an anomalous centrifugal fashion toward the superficial cerebral venous system. These veins converge into a single vein called the collecting vein of the DVA, and form the so-called caput medusae. The collecting vein serves as the major venous outflow of the DVA and continues extraaxially as a dilated cortical vein that joins the superior sagittal sinus. A stenosis on the distal portion of the collecting vein of the DVA as it crosses the dura mater to enter the superior sagittal sinus is depicted. This stenosis may generate an outflow obstruction. A connection between the DVA and the subependymal veins has been depicted, though this is not the rule. (B) Schematic representation of the venous angioarchitecture of a deep draining DVA, same view as in Fig 1 and in (A). In this case, there is absence of the cortical veins (*asterisk*) in a circumscribed region of the brain (*asterisk*) and the intracortical and superficial medullary veins now drain centripetally toward the deep venous system. In this case, a dilated thalamostriate vein is the collecting vein of the DVA.*

stenosis in generating venous hypertension is discussed below. Another frequently encountered abnormality is an ampullary dilatation of the proximal segment of the collecting vein, which can be seen in up to in 27.3% of cases.[4] The cause of this dilatation is not clear in the absence of histological correlation, but the proximal widening of the collecting vein suggests venous stasis and outflow obstruction perhaps caused by focal thickening of the collecting vein wall.

Although the brain tissue drained by a DVA is usually considered to be normal, histological and recent radiological reports have demonstrated that this is far from being the rule (see Associated Cerebral Regional Abnormalities section below).[4,22,24]

DVAs can affect a variable volume of brain parenchyma, ranging from a few sulci or a circumscribed wedge of periventricular white matter to an entire cerebral lobe, or even a whole hemisphere (Fig 4). They are more frequently located at the supratentorial level, with a clear frontal predominance.[4,9] Though the collecting vein is most often unique, several collectors may be observed in about 6% of DVAs (Fig 4).[4] Multiple collectors are more frequently encountered with posterior fossa and large DVAs. In addition, two or more DVAs coexisting in separate regions of the brain have been observed in 1.2% to 16% of cases.[4,9,25]

Imaging of DVAs

The radiological appearance of DVAs is well established.[18,26,27] CT and MRI now permit diagnosing DVAs with confidence in the absence of digital subtraction angiography (DSA). The latter should be reserved for cases presenting with ischemic or hemorrhagic infarction, or whenever an associated vascular malformation is suspected on CT or MRI.

Regardless of the imaging modality employed, the diagnosis of a DVA relies on demonstrating a typical caput medusae draining into a collecting vein. Because of its higher temporal resolution, DSA remains the best imaging modality to study the hemodynamic behavior of DVAs. Typically, DVAs are opacified during the venous phase of the angiogram, concomitantly to the normal cerebral veins. Delayed outflow of a DVA suggesting drainage impairment may sometimes be observed, even in the absence of a demonstrable collecting vein stenosis.

On noncontrast CT, the collecting vein of the DVA may appear to be isodense or slightly hyperdense to the cortex if patent, or markedly hyperdense if acutely thrombosed. In addition, noncontrast CT may disclose an associated hemorrhage, parenchymal calcifications, and atrophy or white matter lesions in the drainage territory of the DVA.[4] Both the collecting vein and the

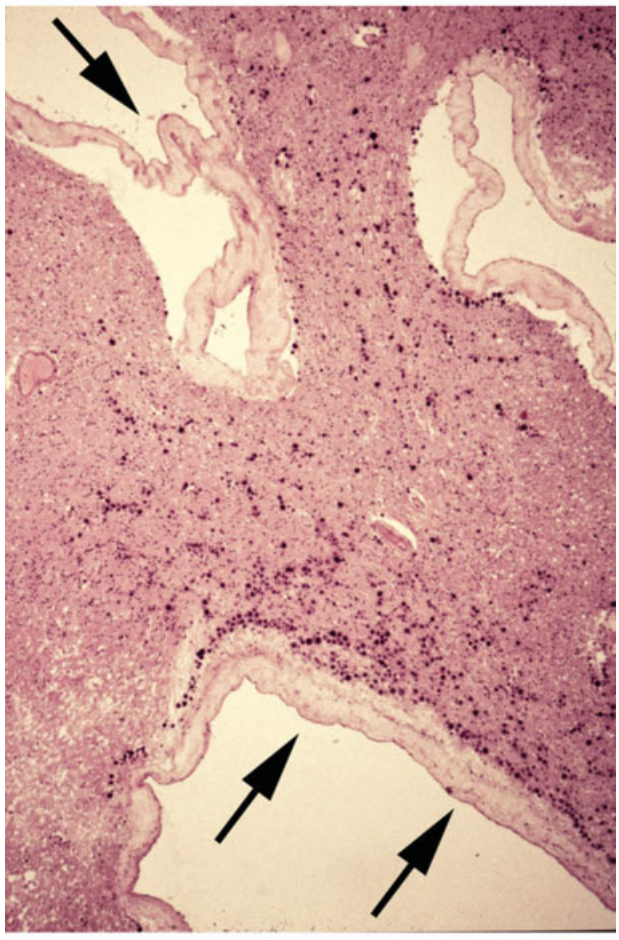

*Fig 3. Hematoxylin-eosin stain, histological demonstration of a parietal DVA (magnification 40×). The venous lakes correspond to dilated veins forming the DVA. The walls are markedly thickened by hyaline material (*black arrows*). Courtesy of Dr Gian Paolo Pizzolato, Department of Clinical Pathology, Geneva University Hospital, Switzerland. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]*

caput medusae enhance after administration of contrast material and are best demonstrated by thin-section CT venography. Unprecedented morphological and hemodynamic analysis of DVAs is now possible with newly developed CT technologies, such as 320-multidetector row CT (320-MDCT), that produce dynamic subtracted CT angiography and perfusion of the whole head (Fig 4C, D).[28]

Noncontrast T2-weighted and T1-weighted MRI may demonstrate flow voids and phase-shift artifact produced by the collecting vein of a DVA and by the larger venous radicles of the caput medusae.[9] As with CT, the caput medusae and the collecting vein readily enhance after gadolinium administration on T1-weighted sequences (Fig 5A, B). Three-dimensional contrast-enhanced gradient-echo T1-weighted imaging allowing for multiplanar reconstructions in the axial, sagittal, or coronal planes, provides excellent morphological assessment of the DVA, including the depiction of a potential stenosis of the collecting vein (see online supplemental material Fig 1). MRI is superior to CT in demonstrating associated parenchymal abnormalities such as white matter lesions, locoregional cerebral atrophy, and CMs.[4] Because CMs are frequently associated with DVAs, they should be specifically sought for using hemosiderin or deoxyhemoglobin-sensitive sequences such as gradient-echo T2-weighted images or susceptibility-weighted images (SWI).[29] CMs are low-pressure and slow-flow vascular entities associated with intralesional hemorrhages at various stages of evolution. On MRI, a typical CM appears as a well-circumscribed lesion of mixed signal intensity on T1-weighted and T2-weighted sequences, with variable enhancement after contrast administration, a hypointense rim corresponding to hemosiderin deposition, and characteristic "blooming effect" resulting from the susceptibility dephasing effect caused by hemosiderin. CMs may be difficult to identify in the setting of a DVA presenting with acute hemorrhage and may only become apparent after the hemorrhage is fully reabsorbed, typically 3 to 6 months after the initial event.

Clinical Presentation

Before the advent of CT and MRI, DVAs were considered rare entities often causing cerebral hemorrhages and seizures. Nowadays, DVAs are frequently encountered on routine diagnostic imaging, in particular with MRI. DVAs are associated with one or more regional CMs in 13% to 40% of cases.[4,30] These CMs are now thought to be responsible for the vast majority of symptomatic cases previously attributed to DVAs themselves (see online supplemental material Fig 2).[31,32] Garner et al.[33] retrospectively evaluated the risk of hemorrhage truly associated with DVAs to be 0.22% per year, while McLaughlin et al.[31] prospectively found that risk to be 0.68% per year, though only one-half of their patients were symptomatic. It was initially proposed that DVAs located in the posterior cranial fossa had a higher propensity to bleed than supratentorial DVAs,[34] but recent evidence suggests that this is not the case,[31] though hemorrhages in the posterior fossa carry a worse prognosis than at the supratentorial level.

Hemorrhagic or ischemic infarction around a DVA may result from acute thrombosis of the collecting vein[35-49] (Fig 5), although it may also be observed in rare instances in the presence of a patent venous collector.[50,51] A review of the literature revealed 19 documented cases of symptomatic thrombosed DVAs (Table) presenting with venous ischemic infarction (53%), parenchymal hemorrhage (37%), subarachnoid and intraventricular hemorrhage (5%), and no intraaxial or

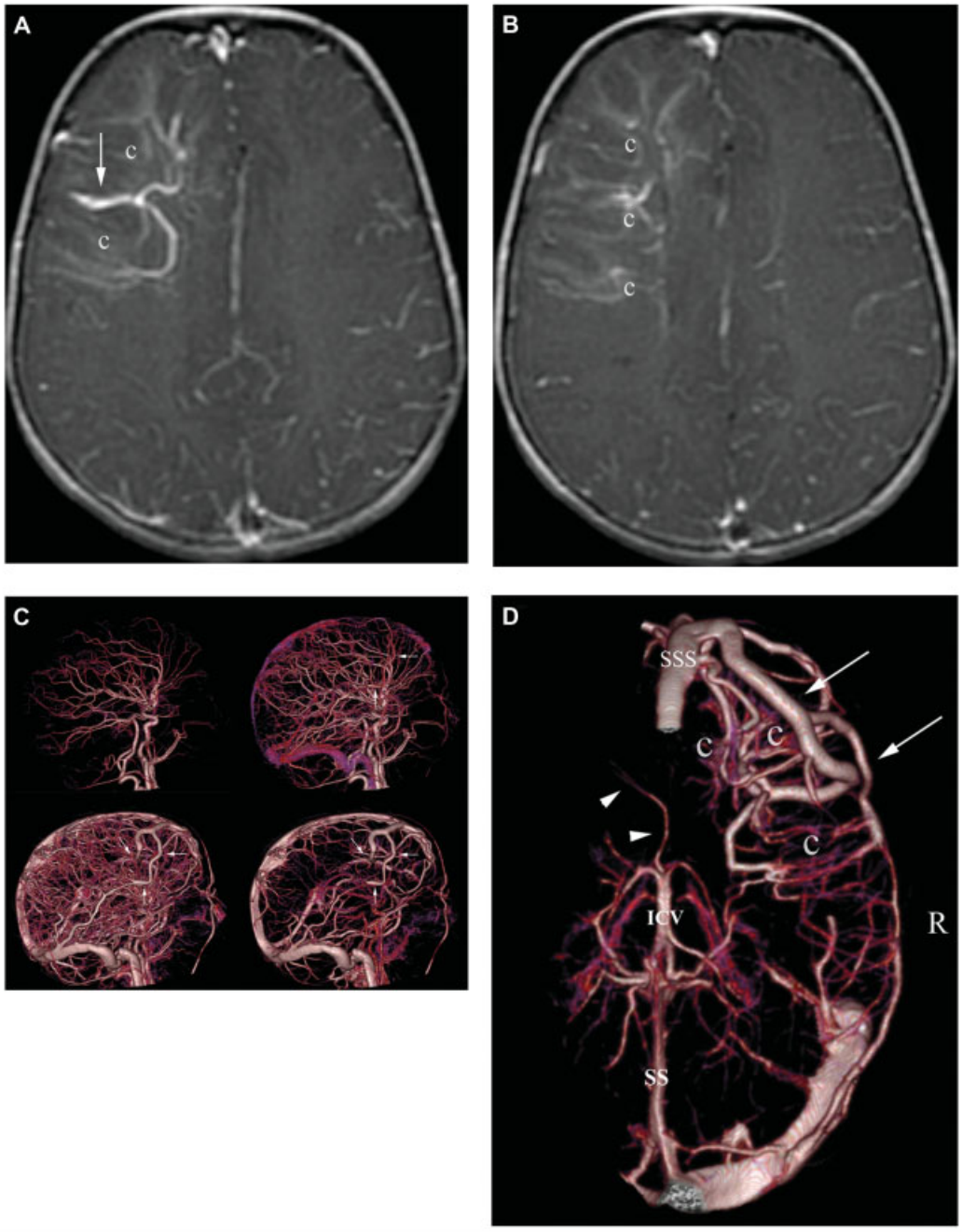

*Fig 4. A 4-year-old boy investigated for seizures. (A, B) Gadolinium-enhanced gradient-echo (GE) T1-weighted MRI showing a complex lobar frontal DVA draining superficially into the superior sagittal sinus through several collecting veins (*arrows*). The caput medusae is well depicted (*c*); 3D-volume-rendered reconstruction from dynamic subtracted 320-multidetector row CT angiography. (C) Right lateral projection, sequential filling of the intracranial circulation (only four out of the 14 whole-head volumes obtained during dynamic acquisition are shown): arterial phase (top left), beginning of venous filling (top right), intermediary phase with arteries and veins (bottom left), and venous phase (top bottom). The high temporal resolution of the this dynamic subtracted sequence suggests normal filling of the DVA during the venous phase (collecting veins,* arrows*). Absence of arterialization of this DVA was confirmed through DSA; (D) superior view, the DVA, the deep venous system, and the anterior third of the superior sagittal sinus (SSS) have been segmented. The caput medusae of the DVA (*c*) are demonstrated along with the various collecting cortical veins that drain into the anterior third of the superior sagittal sinus. The anterior septal vein (*arrowheads*), tributary of the internal cerebral vein (ICV), is visible on the left side, and is developmentally absent on the right. The DVA drains the territory of the latter. SS = straight sinus; R = right side.*

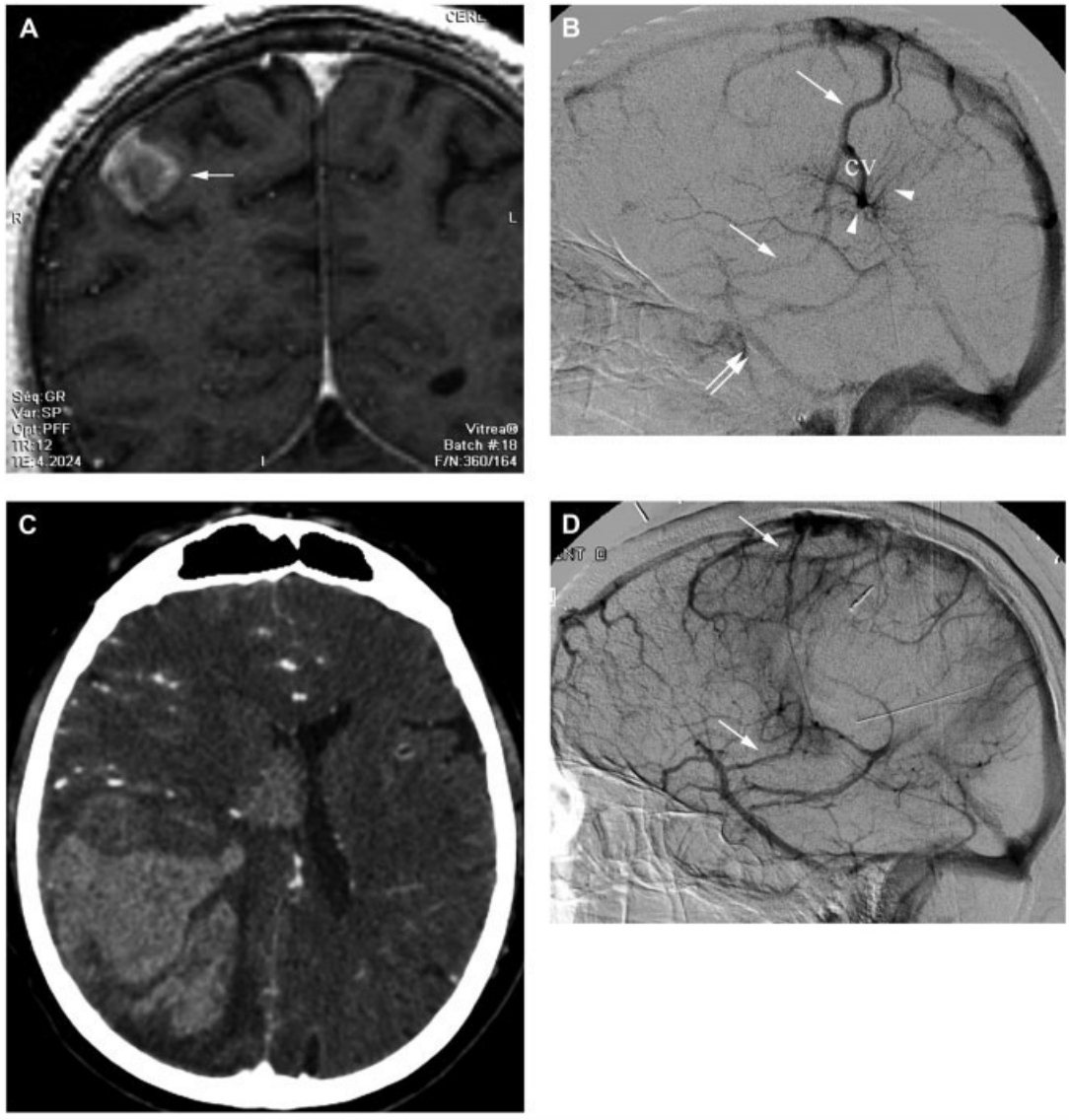

*Fig 5. A 50-year-old man who presented with a small post-Rolandic hemorrhage. (A) Coronal, Gd-3D T1-weighted image showing the hemorrhagic lesion (*arrow*). (B) Right carotid artery injection, lateral view of the venous phase showing the typical caput medusae of the DVA (*arrowheads*) and the collecting vein (*cv*) draining into the greater anastomotic vein of Trolard (*arrows*). The latter was connected to a lesser anastomotic vein of Labbé (*double arrow*). No thrombosis of the DVA was discernable at that time. (C) Four years later, the patient presented with a lobar fronto-parieto-temporal and intraventricular hemorrhage, seen here on this axial CT angiography (CTA) source image. (D) Right carotid artery injection, lateral view of the venous phase; the DVA and the middle segment of the greater anastomotic vein of Trolard are no longer visible due to acute thrombosis. The proximal and distal portions of the greater anastomotic vein of Trolard are still patent (*arrow*).*

extraaxial lesions (5%).[35–49] Management was conservative in eight cases (42%), and involved anticoagulation in seven (37%) and decompression surgery for evacuation of a compressive hematoma or mass effect secondary to venous infarction in four (2%). Clinical outcome was favorable in 16 out of 19 patients (84%) with complete recovery or persistence of mild neurological symptoms, though modified Rankin scores were not made available. Although no large series or controlled studies exist to support systemic anticoagulation over conservative management in cases of thrombosed DVAs, experience gained from treatment of cortical or dural venous sinus thrombosis suggests that systemic and prolonged anticoagulation should also be of benefit in preventing clot propagation, in favoring recanalization, and in promoting reversibility of the symptoms in such cases.[52,53] Finally, screening for prothrombotic conditions should be carried out as with cortical and dural venous sinus thrombosis. Symptomatic hematomas and cerebral edema warrant decom-

Table. **Documented Cases of Symptomatic Thrombosed DVAs**

Case	Reference (Publication Year)	DVA Location	Presentation	Risk Factors	Management	Outcome
1	Walsh et al.[43] (2008)	Right parietal	Initially ataxia, nausea, and vomiting, then unresponsive from a right parietal hemorrhage	None reported	Craniectomy and hematoma removal	No response to verbal command at time of discharge
2	Walsh et al.[43] (2008)	Left frontal	Unresponsive then right facial droop and dysarthria; no parenchymal abnormalities	None reported	Systemic anticoagulation (type not specified)	Complete recovery
3	Gama et al.[44] (2008)	Right frontal	Left sided numbness and weakness and seizures from right frontal edema and small hemorrhage	None reported	Low-molecular-weight heparin	Good recovery
4	Seki and Sahara[45] (2007)	Left parietal	Headache, decreased conscious level and right hemiplegia from left temporoparietal hematoma with mass effect	Negative prothrombotic screening	Craniectomy and hematoma removal	Improvement, persistent moderate aphasia, mild right hemiparesis, and homonymous hemianopsia at 1 month
5	Vieira Santos and Saraiva[46] (2006)	Left frontoparietal	Right hemiparesis from frontoparietal hemorrhage	Homozygous for 4G allele of PAI-1	Low-molecular-weight heparin	Improvement of right hemiparesis
6	Lovrencic-Huzjan et al.[47] (2004)	Inferior vermian	Headache and stiff neck from subarachnoid and IVth ventricle hemorrhage	None reported	Conservative	No neurological sequelae, no recurrence of symptoms
7	Peltier et al.[48] (2004)	Left cerebellar	Headache, vomiting, and coma from venous ischemic infarction of the posterior fossa	Negative prothrombotic screening	Conservative/ intraventricular drainage for hydrocephalus	Recovery with mild residual symptoms
8	Hammoud et al.[41] (2002)	Left frontoparietal	Right sided numbness and weakness from left frontoparietal venous ischemic infarction	40 days after delivery, oral birth control, and smoking, negative prothrombotic screening	Conservative	Recovery with residual minimal sensory deficit and gait disturbance
9	Masson et al.[86] (2000)	Left parietal	Seizure and right motor and sensory hemisyndrome from left frontoparietal venous ischemic infarction	Negative prothrombotic screening	Anticoagulation (3 months)	Complete recovery at 3 months
10	Masson et al.[86] (2000)	Left frontal	Seizures, headache, right hemiplegia, and aphasia from left frontal venous ischemic infarct	Negative prothrombotic screening	Anticoagulation	Slight motor deficit of the right hand
11	Lai et al.[38] (1999)	Right parietal	Left hemiparesis and seizures from right parietal venous infarction	None reported	Conservative	Good recovery
12	Konan et al.[40] (1999)	Bilateral cerebellar	Vomiting, ataxia, and right facial palsy, followed by coma from right cerebellar venous ischemic infarction	None reported	Conservative	Independent, with persistent right facial palsy and cerebellar ataxia
13	Thobois et al.[42] (1999)	Right parietooccipital	Headache, seizures, and left homonymous hemianopsia from right parietooccipital hemorrhage	Oral contraceptives	Anticoagulation	Good recovery, residual hemianopsia
14	Guerrero et al.[49] (1998)	Right cerebellar	Headache, diplopia, ataxia, and vertigo from right cerebellar venous ischemic infarction	None reported	Conservative	Mild recovery
15	Merten et al.[35] (1998)	Left basal ganglia	Right-sided hypoesthesia and motor aphasia from left basal ganglia hemorrhage	Negative prothrombotic screening	Anticoagulation (phenprocoumon for 2 years)	Complete recovery
16	Kim et al.[39] (1996)	Right temporoparietal	Vomiting, ataxia, and left hemiplegia, followed by lethargy from right temporoparietal and basal ganglia venous ischemic infarction	None reported	Decompression craniectomy	Death
17	Field et al.[87] (1995)	Right temporal	Headache, left homonymous hemianopsia and papilledema from right temporooccipital hemorrhage	None reported	Conservative	Not specified, patient discharged after 9 days
18	Yamamoto et al.[37] (1989)	Right parietal	Headache and left-sided weakness, followed by coma from parietal hemorrhage	Postpartum	Decompression craniectomy	Good recovery, slight apraxia, and agnosia of the left hand
19	Bouchacourt et al.[36] (1986)	Left frontal	Headache, seizures, and right hemiparesis from left frontal venous ischemic infarction	None reported	Conservative	Good outcome

pression craniectomy taking particular care to preserve the drainage of the DVA during clot aspiration. Anecdotal publications have reported the association of DVAs with choreoballismus,[54] obstructive hydrocephalus through compression of the cerebral aqueduct by the DVA,[55] ophthalmoplegic migraine,[56] or nerve root compression.[57,58] In contrast to initial beliefs, there seems to be no demonstrable relation between uncomplicated DVAs and epilepsy or headaches.[59–61]

Association With Other Vascular Malformations

DVAs are associated with sporadic CMs in 13% to 40% of cases.[4,30] The CM is typically located in the region of the caput medusae. This close topographic relation between CMs and DVAs, as well as reports describing the de novo formation of CMs or CM-like lesions in the proximity of a DVA.[23,62–64] suggest a causative link between CMs and DVAs (see online supplemental material Fig 3). Evidence of subclinical microhemorrhages may be found in the parenchyma surrounding a DVA (see online supplemental material Figs 4,5), possibly resulting from blood diapedesis through the walls of the venous radicles of the caput medusae, or rupture of the latter.[15,23] Repeated microhemorrhages around DVAs are thought to induce the formation of CM-like lesions by activating angiogenic growth factors such as the vascular endothelial growth factor (VEGF), and lead to reactive angiogenesis with vessel formation and coalescence, a process that has been referred to as hemorrhagic angiogenic proliferation.[65,66] The hemorrhagic risk of CMs associated with DVAs may be higher[67] than the 2.6% and 3.1% per patient-year reported for isolated CMs.[68,69] DVAs may occasionally drain completely or partially into a sinus pericranii. Sinus pericranii consists of anomalous extracranial drainage of the intracranial circulation occurring through diploic emissary veins into an enlarged varicoid venous pouch connected to the subgaleal and scalp venous systems. In a recently published series, a DVA was found in eight out of 15 patients with a sinus pericranii.[70] This finding emphasizes the need for precise analysis of the cerebral venous anatomy when planning the treatment of a sinus pericranii. When surgical or endovascular closure of a sinus pericranii that drains a DVA is contemplated, particular care must be taken in demonstrating an alternative drainage pathway of the DVA (see online supplemental material Fig 6). If no such pathway exists, closure of the sinus pericranii is precluded by the risk of cerebral venous infarction secondary to interrupting the outflow of the DVA.

The association between DVAs and head and neck superficial venous malformations is well established. DVAs may be encountered in up to 20% of patients with a large superficial venous malformation, a much higher incidence than in the general population (see online supplemental material Fig 7).[71] DVAs are also associated with lymphatic or lymphaticovenous malformations of the orbit or periorbital regions, with an incidence as high as 60.6% in a recent series of 33 patients.[72] These patients presented with other concomitant intracranial vascular abnormalities, such as CM (6.1%), dural arteriovenous fistulas (12.1%), pial arteriovenous malformations (3%), and sinus pericranii (3%). Finally, DVAs may be part of the central nervous system manifestations of neurocutaneous disorders, such as the blue-rubber-bleb syndrome characterized by the occurrence of multiple bluish hemangiomas of the skin and gastrointestinal tract.[73]

Atypical Forms of DVAs: Arterialized DVAs

Early angiographic opacification of a DVA during the mid or late arterial phases may exceptionally be observed, typically in large supratentorial DVAs. There is no accepted term designating this subtype of DVAs; they are sometimes called "arterialized DVAs" (the term used herein), mixed-type vascular malformations, arteriovenous malformations with venous predominance, or intracerebral venous angiomas with arterial blood supply.[14,23,65,74–79] DSA remains necessary to adequately characterize this particular form of DVA. Based on the available literature and our personal experience, three types of arterialized DVAs may be distinguished angiographically:

1. Typical DVAs preceded by a caput medusae blush during the mid or late arterial phases, with no demonstrable arterial feeders or AVM nidus. We have never observed a hemorrhagic presentation with this type of DVAs (see online supplemental material Fig 8). The early caput medusae blush may represent increased transit time through the enlarged venous collectors. On the other hand, Dillon[23] has postulated that venous hypertension within the DVA promotes arteriovenous shunting at the precapillary level, leading to the early appearance of the DVA;
2. Arterialized DVAs with enlarged arterial feeders to the caput medusae, without an angiographically demonstrable AVM nidus (Fig 6). In a recent report of 15 cases of this type of DVA, eight patients presented with a hemorrhage and one with seizures.[75] Im et al.[75] concluded that these lesions were in fact AVMs, for which they coined the term of "venous-predominant parenchymal AVM." They based their conclusions on the histology findings in three of their cases, in which arterialized dilated veins with hyalinized walls containing elastic laminae interspaced with normal brain tissue were observed. Similar findings have been described previously.[77]
3. DVAs draining an angiographically demonstrable AVM.[76,78–81]

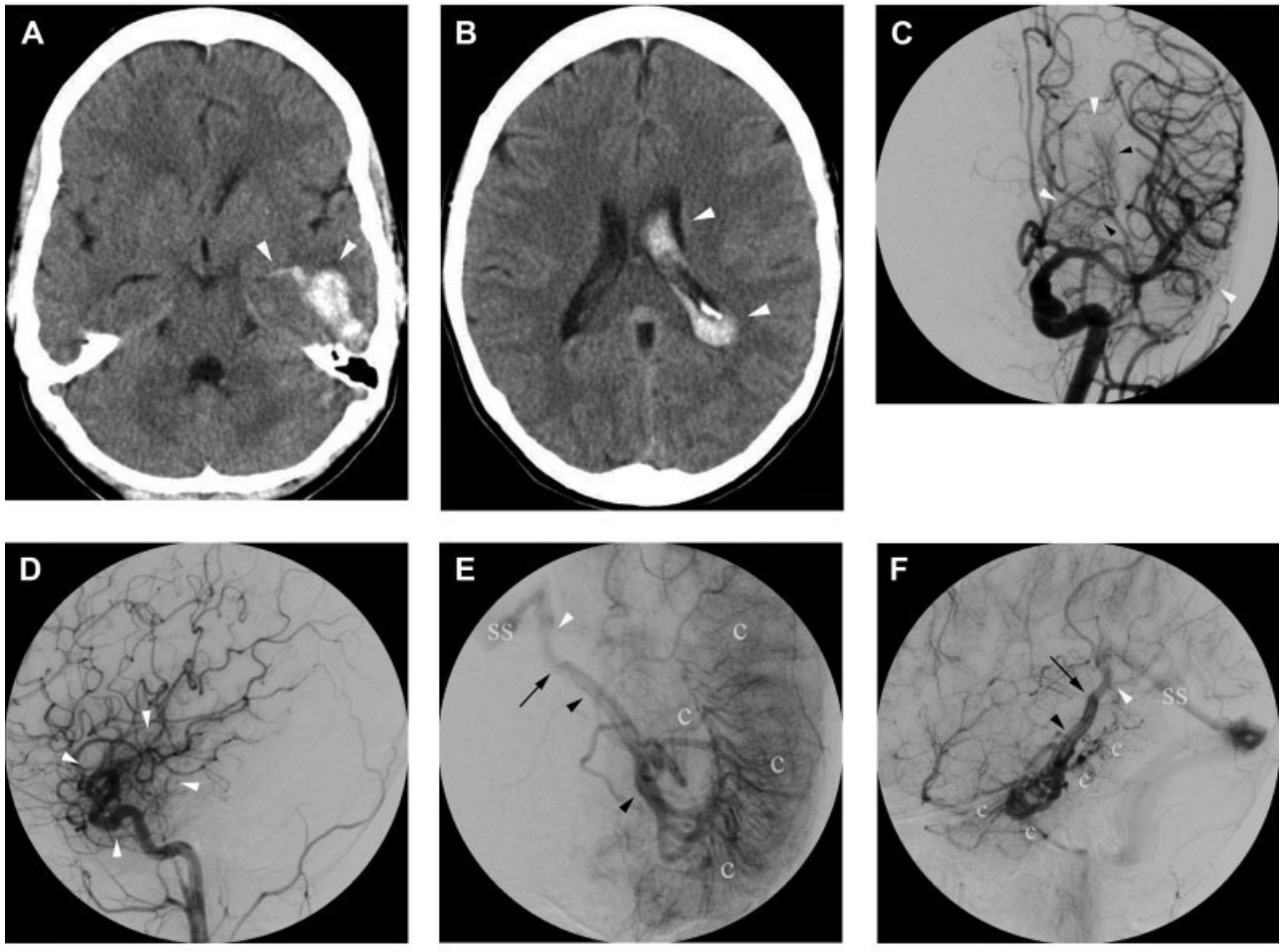

*Fig 6. A 32-year-old woman presenting with left temporoparietal headaches and word-finding difficulties. Noncontrast CT disclosed a left temporal lobe and left lateral ventricle hemorrhage. MRI demonstrated a patent complex left temporal lobar DVA draining into the basal vein of Rosenthal. Because of the hemorrhagic presentation, DSA was performed, revealing a Type 2 arterialized DVA. (A) Noncontrast CT showing an intraparenchymal hemorrhage of the left temporal lobe (*arrowheads*) extending into the temporal horn of the lateral ventricle; (B) hemorrhage in the left lateral ventricle; (C–F) DSA, left internal carotid artery injection, anterior-posterior (AP) (C) and lateral projections (D) of the mid-arterial phase showing slightly enlarged lateral lenticulostriate arteries (*black arrowheads*) and a vascular blush (*white arrowheads*). There is no evidence of an AVM nidus, but the enlarged lenticulostriate arteries are consistent with a Type 2 arterialized DVA; (E, F) same injection and projection as (C, D), venous phase. The large complex DVA of the left temporal lobe is now clearly visible. Note the slight stenosis (*black arrow*) at the junction of the collecting vein (*black arrowhead*), in this case the basal vein of Rosenthal, and the great vein of Galen (*white arrowhead*). c = caput medusae; SS = straight sinus.*

Management of arterialized DVAs must be tailored to each individual presentation. Treatment strategies will be defined by the presence of compressive parenchymal hemorrhage and morphological characteristics such as the neurological eloquence of the lesion site and the presence of an identifiable arteriovenous shunt with feeding arteries or an AVM nidus. In our opinion, incidentally discovered Type 1 arterialized DVAs with no demonstrable AVM nidus or enlarged arterial feeders do not warrant treatment, as they do not seem to be associated with a higher risk of hemorrhage. The natural history of Type 2 and 3 arterialized DVAs has not been studied longitudinally, but their clinical behavior, particularly in their propensity to hemorrhage, make them appear closer to AVMs than DVAs.[75] In cases of parenchymal hemorrhage needing surgical evacuation of the hematoma or resection of an AVM nidus, care must again be taken to preserve the DVA collecting vein in order to prevent ischemic or hemorrhagic complications. Indeed, in the Im et al.[75] series, both patients undergoing partial surgical resection of the DVA developed significant venous infarction, one of which was fatal.[75] If surgical resection of the DVA itself is contemplated, radical "en-bloc" resection of the DVA and the surrounding brain parenchyma seems warranted, an option that is

only available at distance from eloquent brain parenchyma.

Because arterialized DVAs rarely have enlarged arterial feeders and/or a demonstrable AVM nidus or arteriovenous shunt, they are generally not amenable to endovascular embolization. Embolization plays a role in Type 2 or 3 arterialized DVAs, where an arterial feeder or an AVM nidus can be documented by angiography. In such instances, it seems possible to close the distal segments of the arterial feeders, or the AVM nidus itself if present, with the same recommendations as for "standard" AVM treatment. Reports in the literature regarding endovascular treatment of arterialized DVAs are rare. Nussbaum et al.[79] described a case of an AVM draining into a cerebellar DVA, which was embolized with a good clinical outcome. Oran et al.[76] recently described a case of DVA draining an AVM, which presented with a frontal hemorrhage. The lesion was managed with combined embolization and surgical resection of the AVM and the DVA, resulting in improvement of the clinical condition with persistent mild hemiparesis at the 6-month follow-up.[76] Finally, Pereira et al.[51] have reported five cases of arterialized DVAs presenting with hemorrhage, one with an associated AVM and four with microshunts whose morphological characteristics were not specified. These patients were successfully managed with endovascular embolization, without additional neurological deficit.[51]

There is no evidence yet as to the effectiveness of gamma-knife surgery (GKS) for the treatment of arterialized DVAs. GKS has mostly been reserved for patients presenting with hemorrhage, with or without a demonstrable AVM nidus, though the limited numbers and short-term follow-ups do not allow for solid conclusions to be drawn.[75,76,78,80,81] Some authors have reported complete obliteration of the AVM nidus and arteriovenous shunt in arterialized DVAs with an AVM nidus.[80,81] Radiation should be directed at the AVM nidus or the component containing a demonstrable arteriovenous shunt, thus sparing the DVA itself. However, this strategy is not always possible, particularly when addressing large and diffuse arterialized DVAs or DVAs without an identifiable AVM nidus.[81]

Associated Cerebral Regional Abnormalities

In a recent series of 84 consecutive DVAs explored by MRI and CT, San Millán Ruíz et al.[4] have found parenchymal abnormalities (other than CMs) within the drainage territory of the DVA in 65% of the cases. Though several reports in the literature mention abnormalities of the brain around DVAs,[14,18,23–25,30,82] their incidence has been largely underestimated. Locoregional cerebral atrophy was the most frequent abnormality, being present in close to a third of the cases (29.7%), followed by white matter lesions (28.3% on MRI, 19.3% on CT), and dystrophic calcification (9.6% of CT investigations). Parenchymal abnormalities were equally observed in small-, medium-, and large-sized DVAs, suggesting that size is not a determining parameter for their development.

Locoregional atrophy around a DVA may be cortical, subcortical, or corticosubcortical, and may be present both at the supratentorial and infratentorial level. White matter lesions are better evaluated with MRI, and involve both the superficial and deep supratentorial white matter. These lesions characteristically respect the boundaries of the drainage territory of the DVA, they are stable over time, they show no contrast enhancement, diffusion restriction, or mass effect, and behave similar to leukoaraiosis both on CT and MRI. In our experience, these lesions are asymptomatic, though Dillon[23] has reported one patient presenting with slowly progressive left-sided hemiparesis from a right-sided centrum semiovale lesion with partial calcification around a relatively large DVA. Augustyn et al.[82] have found white matter abnormalities adjacent to DVAs in four of their seven cases.

The CT and MRI characteristics of the white matter abnormalities surrounding DVAs are reminiscent of the white matter lesions observed in cases of leukoaraiosis, which have been shown to consist of a spectrum ranging from edema on one end, to demyelination, loss of oligodendrocytes, and gliosis on the other.[83] Similar histological findings were reported by Noran,[24] who observed zones of "demyelination, degenerative alterations of nerve cells, gliosis and leukomalacia . . ." around DVAs. It is likely that the similarities observed at the histological level between the lesions of leukoaraiosis and the white matter abnormalities linked to DVAs translate into a similar appearance by CT and MRI, although confirmation by direct anatomic–radiologic correlation is still lacking.

Calcifications around DVAs typically occur in the supratentorial or cerebellar white matter, the basal ganglia, and the caudate nucleus. They may result from old hemorrhages or from longstanding cerebral ischemia or venous hypertension. Calcifications deposit in the surrounding brain parenchyma or the walls of the DVA.[21]

Physiopathology Considerations

There is a substantial body of evidence from the literature to support the idea that venous hypertension (VHT) is the underlying mechanism leading to the spectrum of brain lesions associated with DVAs. Impaired brain perfusion attributed to venous congestion was documented by several authors in areas drained by either small or large DVAs.[84,85] Intraoperative evidence of increased venous pressure within a DVA was produced by Dillon.[23] Venous outflow obstruction due to a stenosis of the collecting vein may account for VHT in a substantial number of cases. Parietal thickening of

the veins forming the DVA may also contribute to the development of VHT, even in the absence of demonstrable stenosis, by reducing the size and compliance of the vessel lumen, increasing the resistance to flow, and diminishing the vessel's capacity to adapt to pressure modifications.[4] In addition, DVAs represent a point of venous confluence, at which a single collecting vessel drains an abnormally large venous territory, resulting in a relative volume overload that may also contribute to the development of VHT.[4]

Conclusions

Cerebral DVAs are frequently encountered on routine imaging studies of the brain and are currently considered extreme variations of the cerebral venous angioarchitecture that puts them at risk of developing venous hypertension. In the vast majority of cases, however, DVAs follow a benign clinical course and do not require follow-up imaging studies or specific medical management. However, their association with other vascular malformations, in particular with CMs, is frequent, and usually accounts for cases presenting with cerebral hemorrhage or seizure activity. Associated vascular malformations must therefore be sought for with specific MRI protocols employing susceptibility-sensitive sequences wherever a DVA is encountered on CT, MRI, or DSA. On rare occasions, a DVA itself may be responsible for neurological complications secondary to thrombosis of the collecting system of the DVA, or to mass effect exerted by the dilated collecting vein. Thrombosed DVAs should be managed as cortical or dural venous sinus thrombosis. In cases where a compressive brain hematoma or edema requires surgical management, particular care must be taken in preserving the DVA collecting vein to avoid catastrophic venous infarction. There is an increasing awareness to rare and atypical forms of arterialized DVAs that generally follow an aggressive clinical course and likely carry a hemorrhagic risk similar to AVMs. Three types of arterialized DVAs may be defined based on the angiographic demonstration of abnormal arterial supply to the DVA or an associated AVM that drains into the DVA system. DSA is required to establish this diagnosis and should be proposed for all cases of DVAs presenting with a cerebral hemorrhage where CT or MRI fail to demonstrate a collecting vein thrombosis or a CM. The management of this subtype of DVAs is not yet clearly established in the literature due to the small number of reported cases, though it may include surgery, endovascular embolization, or GKS depending on individual case presentation. Clinicians should be aware that, though generally benign, DVAs represent a complex entity with potential for clinical complication requiring additional imaging investigations and specific medical management in some cases.

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