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Histologically Classified Venous Angiomas of the Brain: a Controversy

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Abstract

The term ''venous angioma'' (VA) usually refers to a developmental venous anomaly (DVA). However, a group of vascular malformations called VAs shows no venous abnormalities on angiography. The clinical and histological features of histologically classified VAs were studied in eight patients who presented with hemorrhage or seizures to reevaluate these venous anomalies. Angiography showed no venous abnormalities in six patients. Histological study included immunostaining for smooth muscle actin and glial fibrillary acidic protein. Surgical specimens of 10 cases of cavernous angiomas, 10 cases of arteriovenous malformations, and two cases of capillary telangiectasias were studied to compare these types of VAs. Angiographically occult VAs were surgically removed safely, whereas removal of DVAs was complicated by brain swelling and hemorrhagic infarction of the brain. Histological examination found angiographically occult VAs contained malformed and compactly arranged vessels with partly degenerated walls, whereas DVAs had dilated thin-walled vessels that were diffusely distributed in the normal white matter. This study of our cases and a review of the reported cases of VAs suggests that two different clinical and pathological entities are commonly categorized as ''VA,'' angiographically occult VAs and DVAs. These two entities should be carefully distinguished.

Key words: cerebral vascular malformation, cerebral vein, cerebral venous angioma

Introduction

Venous angiomas (VAs) or venous malformations are angiographically demonstrated venous anomalies with a caput medusae-like appearance, and thus can be considered as a developmental venous anomaly (DVA). However, VAs have also been found as angiographically occult vascular malformations (AOVMs).[4,9,13,26,47,55,62] The histological findings of these occult malformations were consistent with those of VAs, that is containing numerous venous channels that were separated by brain parenchyma. However, some authors deny the existence of VAs other than DVAs.[43,56] Whether angiographically occult VAs should be classified as VAs or as atypical cases of some other subtype of vascular malformations remains controversial.

The present study investigated the clinical and histological features of malformations that were previously histologically classified as VAs to reevaluate these vascular lesions as two groups: angiographically occult VAs and DVAs. Histologically, ''angiographically occult VAs'' were found to contain malformed and compactly arranged blood vessels with partly degenerated walls, whereas DVAs possessed dilated thin-walled vessels that were diffusely distributed in the normal white matter.

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Materials and Methods

This study is a retrospective analysis of the records of eight patients treated at six different medical centers: one at Saga Medical School, one at Kyushu Kouseinenkin Hospital (Kitakyushu, Fukuoka), three at Kitasato University School of Medicine, one at Massachusetts General Hospital (Boston, Mass., U.S.A.), one at Nagasaki Rosai Hospital (Sasebo, Nagasaki), and one at Mitsui Memorial Hospital (Tokyo). Six patients had lesions with histological diagnosis of VA, and two patients with angiographical DVAs that were subsequently resected in 1982 and 1986. Six of the eight cases were previously described[38,40,58] or included in the study of AOVMs.[2]

Table 1 Clinical summary of eight cases of histologically classified venous angioma of the brain

Case No.	Age/Sex	Presentation	Location	Size (diameter, cm)	Angiographic findings	CT findings	Operative (autopsy) findings
1	9/M	seizure	parietal lobe	3	avascular mass	not done	dark brownish mass with calcification
2	21/F	seizure	frontal lobe	2	normal	area of high attenuation	partly calcified mass
3	9/F	hemorrhage	cerebellar hemisphere	2	normal	intraparenchymal hemorrhage	conglomeration of abnormal vessels
4	40/M	hemorrhage	pons	1	normal	intraparenchymal hemorrhage	dilated blood vessels and hemorrhage
5	28/F	hemorrhage	frontal lobe	2	faint stain	intracerebral hemorrhage	conglomeration of abnormal vessels
6	42/F	hemorrhage	pons	1.5	normal	intraparenchymal hemorrhage	encapsulated mass containing old hematoma
7	50/F	hemorrhage	frontal lobe	3	caput medusae-like venous anomaly	intracerebral hemorrhage	large vein and vascular brain parenchyma
8	38/F	hemorrhage	cerebellum	2	caput medusae-like venous anomaly	intraparenchymal hemorrhage	large vein and vascular brain parenchyma

CT: computed tomography.

Clinical features and outcomes were summarized from the records and diagnostic imaging studies were reviewed.

Histological specimens were stained with hematoxylin and eosin, as well as with elastic van Gieson. Multiple sequential sections were also cut and prepared for immunostaining of smooth muscle actin (SMA) and glial fibrillary acidic protein (GFAP) to detect the presence of smooth muscle and gliotic brain tissue, respectively. Mouse monoclonal anti-alpha SMA antibody (concentration 1/50; DAKO, Glostrup, Denmark) and mouse monoclonal anti-GFAP antibody (concentration 1/100; Novocastra Laboratories, Newcastle, U.K.) were used for immunostaining. Surgical specimens of well-defined vascular malformations (10 cases of cavernous angiomas, 10 cases of arteriovenous malformations [AVMs], and 2 cases of capillary telangiectasias) were prepared and stained before being compared to the VAs.

Results

I. Clinical findings

The six patients with VAs (Cases 1–6, Table 1) were four females and two males aged 9 to 42 years (mean age 25 years). Four patients presented with hemorrhage and two patients presented with seizures. Two lesions were located in the frontal lobes, two in the pons, one in the parietal lobe, and one in the cerebellum.

The two patients with DVAs (Cases 7 and 8) were both females aged 50 and 38 years. Both presented with hemorrhage. The lesions were located in the frontal lobe and in the cerebellum.

II. Imaging findings

Angiography demonstrated no abnormalities either in the arterial phase or in the venous phase in four patients with VAs. However, angiography demonstrated an avascular mass lesion in Case 1, and a faint opacity related to the VA from the late arterial phase to the venous phase in Case 5. Computed tomography (CT) and magnetic resonance (MR) imaging demonstrated intraparenchymal hemorrhage in Cases 3–6. CT with contrast medium demonstrated either no (Cases 3, 5, and 6) or slight (Case 4) enhancement of the lesions (Fig. 1). Radiography or CT demonstrated calcified lesions in the two patients who presented with seizures (Cases 1 and 2).

CT showed hemorrhage in the two patients with DVAs. CT with contrast medium showed a linear enhancing lesion near the hematoma in Case 7. Angiography demonstrated a caput medusae-like venous structure in the venous phase in both cases.

III. Surgical findings and outcome

All patients with VAs except Case 4 underwent direct surgery. The preoperative diagnosis was either AOVMs (Cases 3 and 6) or AVM (Case 5) in the patients who presented with hemorrhage. Hemato-

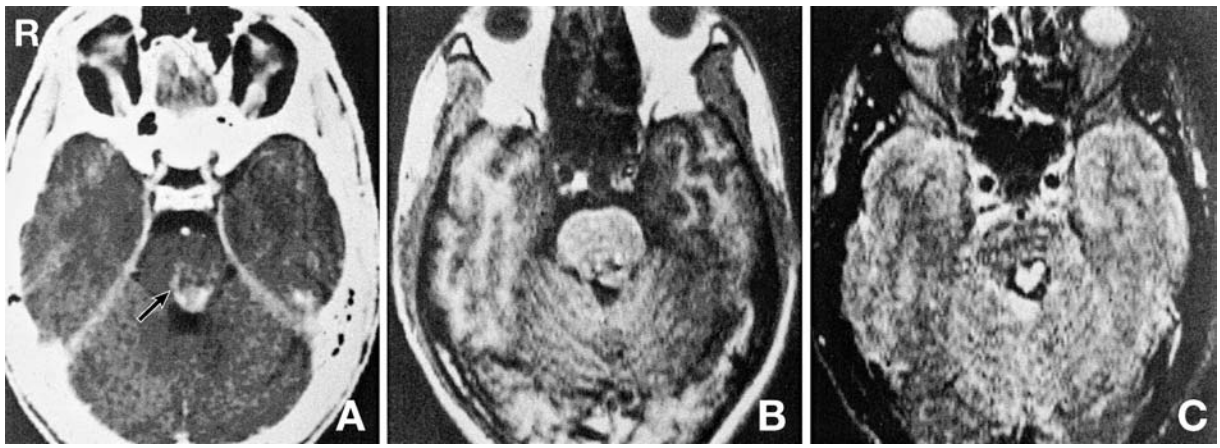

Fig. 1 Case 4. A: Computed tomography scan with contrast medium showing an area of high attenuation in the pontine tegmentum surrounded by slight enhancement suggestive of well-vascularized structures (*arrow*). B, C: Magnetic resonance images showing a lesion with signal characteristics of an old blood clot.

Table 2 Clinical outcome and histological findings of eight cases of histologically classified venous angioma

Case No.	Postoperative course	HE staining	Elastic van Gieson staining	SMA staining	GFAP staining
1	uneventful	dense fibrous vessel wall	no elastic laminae	no smooth muscle layer	intervening gliotic tissue
2	uneventful	dense fibrous vessel wall	no elastic laminae	no smooth muscle layer	intervening gliotic tissue
3	uneventful	dense fibrous vessel wall	no elastic laminae	no smooth muscle layer	intervening gliotic tissue
4	died at 8 mos	dense fibrous vessel wall	no elastic laminae	no smooth muscle layer	intervening gliotic tissue
5	uneventful	fibrous vessel wall	no elastic laminae	compact smooth muscle layer	intervening gliotic tissue
6	uneventful	thin vessel wall	no elastic laminae	no smooth muscle layer	intervening gliotic tissue
7	acute brain swelling	fibrous vessel wall	no elastic laminae	loose smooth muscle layer	normal brain tissue
8	acute brain swelling	fibrous vessel wall	no elastic laminae	loose smooth muscle layer	normal brain tissue

GFAP: glial fibrillary acidic protein, HE: hematoxylin and eosin, SMA: smooth muscle actin.

mas were evacuated and the abnormal vascular structures in the walls of the hematoma cavity were removed. Macroscopically, the lesions were a conglomeration of abnormal vessels and an encapsulated mass containing old hematoma. The calcified lesions were removed in the patients who presented with seizures (Cases 1 and 2). The lesions were dark brownish masses, with partial calcification. The postoperative courses were uneventful (Table 2). One patient (Case 4) died 8 months after radiosurgery. Postmortem examination revealed hemorrhage and an area of necrosis in the pontine tegmentum.

The two patients with DVAs underwent surgery. A dilated vein was seen running toward the superior sagittal sinus in Case 7, which was resected along with removal of the hematoma. Acute cerebral swelling immediately followed this procedure. The left frontal lobe was partially removed to ameliorate the edema. The right hemiparesis and dysphasia worsened postoperatively. Postoperative CT showed cerebral edema with intracerebral hematoma. The patient underwent reoperation that same evening for external decompression of the brain and evacuation of the hematoma. After rehabilitation, she was discharged in an ambulatory condition with mild disability. A large vein and vascular brain parenchy-

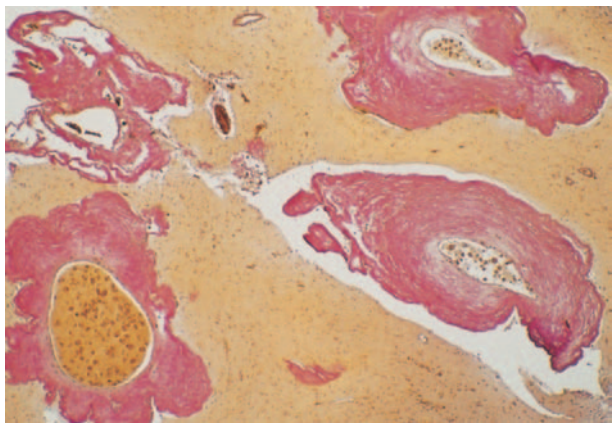

Fig. 2 Photomicrograph showing a cavernous angioma containing abnormal blood vessels with irregularly thick dense fibrous walls surrounded by gliotic brain tissue at the periphery of the lesion. Elastic van Gieson stain, original magnification ×40.

ma adjacent to the hematoma were resected in Case 8. Acute cerebellar swelling immediately followed this procedure. One third of the left cerebellum was removed to ameliorate the swelling. CT performed the following day demonstrated edema of the left cerebellum with hemorrhage. The patient underwent reoperation for cerebellar decompression. The patient recovered and was discharged with mild incoordination of the left limbs. Postoperative CT findings were interpreted as hemorrhagic infarction in both patients.

IV. Histological findings

Histological examination of 10 cases of cavernous angiomas showed clusters of sinusoidal vessels with dense fibrous vessel walls and without intervening brain tissue. The walls of the blood vessels were irregularly thickened, with such degenerative changes as hyalinization and calcification, and without elastic tissue as determined by elastic van Gieson staining. SMA staining showed positive cells only in the subendothelial layer and within pericytes, and no smooth muscle layer. GFAP staining showed a small amount of gliotic tissue among some of the vessels in three cases. Some abnormal vessels with markedly thickened walls were surrounded by the gliotic brain at the periphery of the lesion in two cases (Fig. 2). Ectatic vessels often contained thrombus. Histological examination of 10 cases of AVMs showed clusters of abnormal arteries and veins with fibrous walls, and a small amount of intervening gliotic brain tissue. A compactly arranged smooth muscle

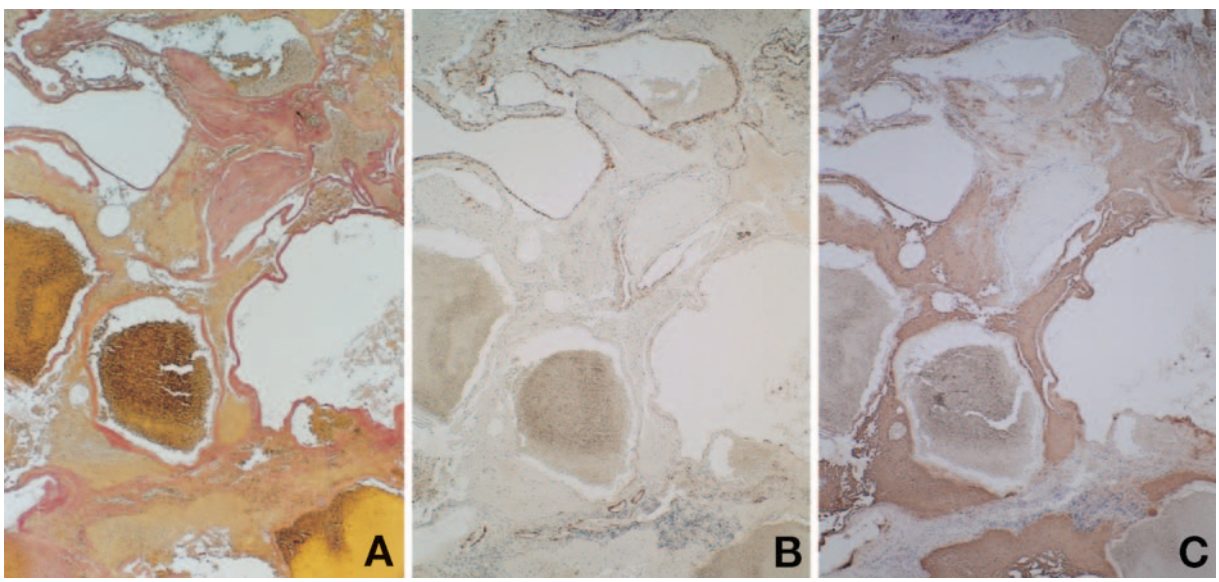

Fig. 3 Case 2. A: Photomicrograph showing abnormal blood vessels and intervening brain tissue, areas of thinning as well as fibrous thickening of vessel walls and no elastic laminae in the walls of the blood vessels. Elastic van Gieson stain, original magnification ×40. B: Photomicrograph showing positive smooth muscle actin staining of cells only in the subendothelial layer of some vessel walls, and no smooth muscle layer. Original magnification ×40. C: Photomicrograph showing gliosis in the brain parenchyma interspersed between the venous channels. Glial fibrillary acidic protein stain, original magnification ×40.

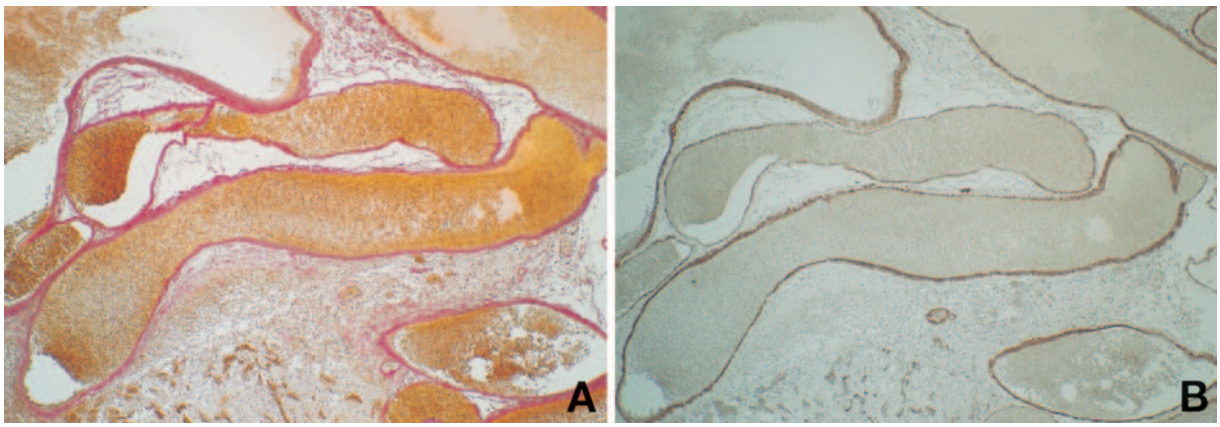

Fig. 4 **Case 5. A: Photomicrograph showing clusters of large blood vessels, with small amounts of intervening gliotic tissue, and no elastic laminae in the abnormal blood vessels. Elastic van Gieson stain, original magnification ×40. B: Photomicrograph showing a compactly arranged smooth muscle layer in the vessel walls. Smooth muscle actin stain, original magnification ×40.**

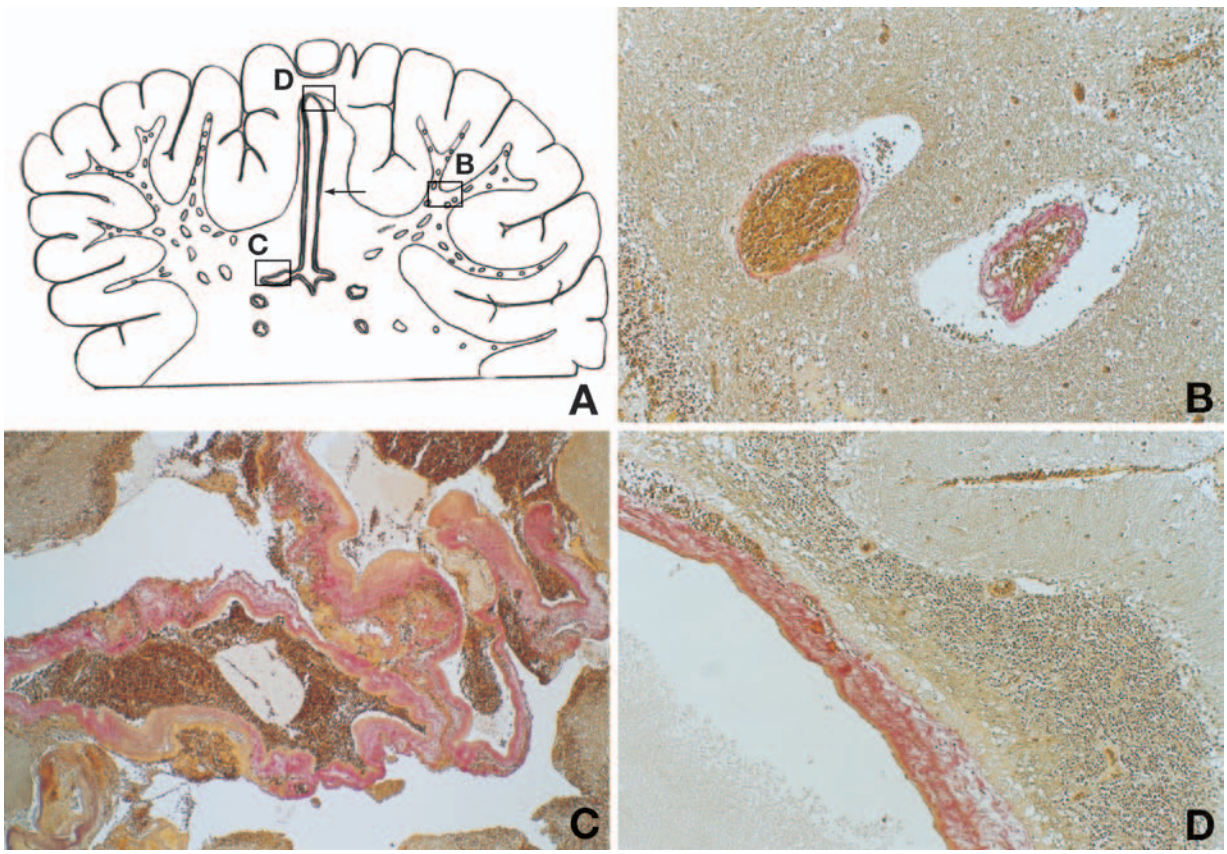

Fig. 5 **Case 8. A: Drawing of the resected specimen of the cerebellum illustrating how multiple dilated veins were distributed diffusely in the white matter and drained the normal brain tissue. *Squares with letters* indicate the areas of the photomicrographs. Original magnification ×3. B–D: Photomicrographs showing multiple dilated thin-wall blood vessels within the normal white matter (B), which converged into larger vessels with thick walls (C), and a large draining vein (*arrow* in A) traversing the cerebellum had a thick, collagen wall (D). Elastic van Gieson stain, original magnification ×100 (B), ×40 (C), ×100 (D).**

layer was observed in most of the vessel walls with SMA staining. Histological examination of two cases of capillary telangiectasias showed collections of dilated capillaries with thin vessel walls and intervening brain tissue.

Histological examination of four cases of VAs (Cases 1–4, Table 2) showed similar features of numerous venous channels separated by brain parenchyma. The walls of blood vessels were irregularly thickened without elastic tissue as determined by elastic van Gieson staining (Fig. 3A). SMA staining showed positive cells only in the subendothelial layer of some vessel walls, and no smooth muscle layer (Fig. 3B). The brain parenchyma that was interspersed between the venous channels showed gliosis in all cases by GFAP staining (Fig. 3C) and calcification and liquefaction in two cases (Cases 1 and 2). Thrombi were seen inside ectatic vessels. There was fresh hemorrhage and a layer of hemosiderin-laden macrophages around the blood vessels (Cases 3 and 4). The characteristics of the vessel walls in these four cases of VAs (Cases 1–4) resembled those of cavernous angiomas.

Histological examination of Case 5 of a VA showed clusters of large blood vessels with an area of dilated capillaries. There was a small amount of intervening gliotic tissue between these abnormal blood vessels. No elastic laminae were seen in the walls of large blood vessels by elastic van Gieson staining (Fig. 4A). SMA staining showed a compactly arranged smooth muscle layer in most of the vessel walls (Fig. 4B). The characteristics of the vessel walls in this case resembled those of the abnormal venous walls in AVMs.

Histological examination of Case 6 of a VA showed clusters of dilated blood vessels with thin walls, and intervening gliotic tissue between these blood vessels. These abnormal blood vessels had no elastic laminae. SMA staining showed positive cells in the subendothelial layer of the vessel walls, but no smooth muscle layer. The characteristics of the vessel walls in this case resembled those of capillary telangiectasia.

Histological examination of Cases 7 and 8 revealed large numbers of dilated thin-walled vessels, and each patient had a large caliber vein traversing the brain (Fig. 5A). These thin-walled vessels were diffusely distributed within the white matter, which otherwise looked normal (Fig. 5B). These thin-walled vessels drained into the large caliber vein (Fig. 5C). The large caliber vein was composed of thick fibrous, collagen walls with no elastic laminae (Fig. 5D) and loosely arranged smooth muscle layers by SMA staining. The histological and angiographic information both suggested the diagnosis of DVA.

The histological findings of DVAs (Cases 7 and 8) mimicked those of angiographically occult VAs (Cases 1–6), but lacked certain features that are commonly seen in vascular malformations, such as malformed blood vessels, partly degenerated vascular walls, organizing intravascular thrombi, hemosiderin-laden macrophages, and gliosis of the interspersed brain parenchyma.

Discussion

I. VAs in the classification of vascular malformations

Virchow[63] classified these angiomatous anomalies as follows: Cavernous angiomas; and vascular changes in which glia or nerve tissue is present between the individual blood vessels as telangiectasias and racemose angiomas. Cushing and Bailey[11] classified only the second group as a malformation, and regarded cavernous angiomas as true neoplasms. Racemose angiomas in the second group were later subdivided into AVMs and VAs. The current terminology used for vascular malformations[21,31,50] thus includes a group called ''VAs,'' which refers to racemose angiomas that consist only of vessels resembling veins. This histological classification allows arteries and veins, and even capillaries to be present in the AVMs, whereas only capillaries or veins are allowed in either capillary telangiectasias or VAs.[32,33] However, the absence of any types of arteries in the resected specimens was difficult to confirm. Old reports of VAs have been questioned and the described lesions are often reinterpreted as AVMs rather than pure venous malformations.[6] Simple histological classification without angiographic study could erroneously interpret secondary parenchymal venous engorgements from dural arteriovenous fistulae as VAs. So-called spinal VAs, which had long been considered as a common type of spinal angiomas,[50] actually represent secondary intrathecal venous engorgements from dural arteriovenous fistulae.[17,48]

A series of regular and linear vascular channels that represent dilated medullary veins has been designated as VAs or medullary venous malformations.[18] Although described as malformations, these lesions would be more accurately categorized as DVAs, given that they provide the functional venous drainage for the involved tissue.[24,25]

II. VAs of the brain other than DVAs

Radiologically, vascular malformations can be divided into two groups: angiographically visible lesions and AOVMs.[2] Cavernous angioma is a frequent histological subtype among resected AOVMs,

but AVMs, VAs, and capillary telangiectasias are also common.[4,9,13,26,41,47,55,57,62,64] We carefully reviewed well-documented cases of VAs other than DVAs.[4,9,13,55] Thirteen cases of VAs were found excluding our cases. Histological study showed numerous venous channels that were separated by gliotic brain parenchyma in all cases. The walls of these blood vessels were irregularly thickened, with degenerative changes such as hyalinization and calcification, but without elastic tissue. The histological diagnosis was VA.

Hemorrhage was the presenting symptom in five patients, and seizures in eight patients. Twelve lesions were located supratentorially and one in the cerebellum. The diameter of the lesions was greater than or equal to 2 cm, indicating that they were not angiographically occult because of their small size. Angiographic studies showed normal results or findings consistent with an avascular mass in 11 cases, faint staining in one, and early venous filling in one. CT showed demarcated areas of high attenuation, suggesting lesions with calcification, thrombosis, or hemorrhage. CT with contrast medium showed either no enhancement or only slight enhancement. MR imaging often demonstrated signal characteristics indicative of nonacute intraparenchymal hemorrhage. The macroscopic descriptions of the lesions included the following: partially calcified mass, encapsulated dark brownish mass, and conglomeration of abnormal vessels, resembling cavernous angiomas and AVMs.

VAs account for 7% to 10% of a large series of AOVMs.[26,64] There is no discernible location preference for VAs, whereas capillary telangiectasias occur most commonly in the pons.[15,33,59] None of the clinical features or findings of diagnostic imaging are typical of VAs or exclusive of other histological subtypes. The natural history of VAs is not known because the diagnosis is usually only established after surgery, and thus cannot be followed up. Surgical resection should be recommended for patients who have suffered massive or recurrent hemorrhage. Retrospective analysis revealed that the histological subtype of VAs did not influence surgical mortality and morbidity.[26]

Vascular malformations containing numerous venous channels separated by brain parenchyma can be histologically classified as VAs. However, the histological distinction of VAs from cavernous angiomas is not always clear. Intervening brain tissue can be present at the periphery of cavernous angiomas. The absence of any kinds of arteries is difficult to confirm and the distinction between dilated capillaries and veins with thin walls seems quite arbitrary.[50] Therefore, reported cases of VAs could be variants of cavernous angiomas, AVMs, or capillary telangiectasias.

III. DVAs

DVAs are relatively common incidental findings at autopsy,[52] as well as frequent incidental discoveries during radiologic studies, with a prevalence as high as 0.6% (50 of 8200 craniospinal MR images).[16] Neurological symptoms or complications in patients with DVAs include hemorrhage, seizures, focal neurological deficits, and headaches. Reports of the AOVMs associated with DVAs are increasing.[1,3,8,10,12,19,30,37,45,46,53,60,61,66] Neurological symptoms or complications have been attributed to AOVMs rather than DVAs. The exact nature of the association is unknown, but certain DVAs are assumed to cause regional venous hypertension, which may facilitate formation of AOVMs.[8,10,27] Other possible causes of the symptoms may be longstanding venous hypertension and venous infarction,[23,29,35] but in many cases, as well as our two cases of DVAs, the exact causes of the symptoms have not been clarified.

The neuroradiologic diagnostic features are a caput medusae-like appearance on angiography, linear or curvilinear enhancement on CT, and a tubular signal void on MR imaging.[16,22,45,51] DVAs can be divided into two angiographical types, with and without early-appearing vessels (capillary blush in the arterial phase and early venous filling).[39] The nature of the early-appearing vessels has not yet been clarified. CT often shows a nodular or globular area of high attenuation with faint enhancement after contrast administration.[42,49] This finding is assumed to be the associated AOVM.[3,45]

Histological studies of resected DVAs[6,44,65] as well as autopsy studies[12,17,46,52,54,67] showed characteristics similar to those in Fig. 5, dilated thin-walled vessels within the normal white matter and a large caliber vein with a thicker wall. These histological findings of DVAs were interpreted as either VAs or telangiectasias.[7,12,46] We would rather emphasize that secondary changes such as degenerating vessel walls, organizing intravascular thrombi, hemosiderin-laden macrophages, gliosis, and liquefaction of interspersing brain parenchyma, which are all essentially common to any subtype of vascular malformations, have rarely been demonstrated in DVAs.

Resection of VAs can be performed without major morbidity,[28] but it is now widely accepted that resection or occlusion of a DVA causes acute brain swelling and venous infarction.[5,54] Some previous VA cases showed the clinical and histological features of DVAs and others showed those of

angiographically occult VAs.[28] Use of the term ''VA'' that may include these two entities can cause confusion. These two entities should be carefully distinguished and discussed separately. Management of DVA patients should be conservative.[14,34] Vascular malformations associated with DVAs can be managed by surgical resection, but DVAs should be carefully protected from inadvertent injury during manipulation of the vascular malformations.[20,36,37,45,53]

IV. Conclusion

The term ''VA'' apparently includes two different clinical and pathological entities, angiographically occult VAs and DVAs. Use of the term ''VA'' that may include these two entities can cause confusion which has implications for treatment. These two entities should be carefully distinguished and discussed separately.

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Commentary on this paper appears on the next page.

Commentary

As some cerebral venous anomalies do not appear on angiography, the authors attempted to distinguish angiographically demonstrable developmental venous anomalies (DVAs) from angiographically occult venous anomalies (AOVAs) by histological examination. They found that AOVAs contained malformed and compactly arranged vessels, whereas DVAs had dilated thin-walled vessels, which were diffusely distributed in the white matter. They suggest that a DVA should be distinguished from an AOVA, as the surgical removal of a DVA can result in venous infarction. CT and MRI can detect both AOVAs and DVAs. Many hemorrhagic AOVAs have been shown to be cavernous angiomas, or cryptic vascular malformations. If a patient has both an AOVA, with a nearby DVA, and develops an intracerebral hemorrhage, the source of hemorrhage might be an AOVA located near the DVA, not a DVA itself. Therefore, the surgical removal should be restricted to the AOVA for the prevention of venous infarction.

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There has been considerable confusion in the classification and terminology of vascular malformations involving the CNS parenchyma. This confusion seems to derive from the presence of mixed forms or transitional forms that show the features of more than one of the histologically discrete categories of vascular malformations. Inappropriate use of the terminology of ''angioma,'' still fairly often used to indicate nonneoplastic malformative vascular lesions, is also aggravating this issue. The present study aimed at analyzing the clinicopathologic diversities of venous malformative lesions of the brain. The authors reported 8 cases of so-called ''venous angioma'' and concluded that these lesions should be separated into two distinct categories, angiographically occult venous malformations and developmental venous anomalies showing the ''Medusa's head'' characteristic on angiography. The authors' argument is mostly justified by their clinical, radiographical and histological observations except for incorporating these two entities into one ambiguous heading, ''venous angioma'' ab initio. This ambiguity of terminology, however, is not fully the fault of the authors, because a recent review article (ref. 7 of this article) and even Greenfield's Textbook of Neuropathology use the term venous malformation and venous angioma synonymously. In Russell & Rubinstein's textbook, vascular hamartomas are classified into 4 entities: capillary telangiectases, cavernous angiomas, arteriovenous malformations and venous malformations. Venous malformation is defined as an abnormality composed only of a single enlarged and tortuous vein, or of a group of such veins. ''Venous angioma'' is carefully eliminated in the text and the Medusa's head type lesions are not included in the vascular hamartomas of the CNS. In this context, it is suggested that venous malformations and developmental venous anomalies should not be included in a single category, and ''venous angioma'' should be carefully used as a synonym of developmental venous anomaly, if this terminology is unavoidable.

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*The authors present the surgical experience and pathological findings of eight patients with venous angioma. In six patients, there were no venous anomalies on angiography (angiographically occult venous malformation), and all patients except one underwent surgical resection without complications. However, two patients with developmental venous anomalies, appearing as dilated vein on angiography, underwent surgical resection which was followed by acute brain swelling and neurological deficits. As the authors described, the term ''venous angioma'' usually refers to developmental venous anomalies showing the well-known ''caput medusae'' feature on angiography. In some textbooks of neuropathology (*Greenfield Neuropathology, Ellison's Neuropathology *etc.), the term ''venous angioma'' is used just to describe developmental venous anomalies. However, some (arterio-)venous malformations like ''angiographically occult venous malformations'' are called ''venous angioma'' but this is terminological misinterpretation. Due to this terminological confusion, the authors have suggested that the ''venous angioma'' should be distinguished from angiographically occult venous malformations, because ''venous angioma'' cannot be surgically resected without complications of brain swelling and hemorrhagic infarction. From the surgical point of view, we should use the term ''venous angioma'' carefully only for developmental venous anomalies.*

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