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Cerebral Developmental Venous Anomalies Associated with Head and Neck Venous Malformations

Monique Boukobza, Odile Enjolras, Jean-Pierre Guichard, Francoise Gelbert, Denis Herbreteau, Daniel Reizine, and Jean-Jacques Merland

PURPOSE: To study cerebral developmental venous anomalies in patients with extensive venous malformations of the head and neck. **METHODS:** All patients had undergone carotid angiography 10 to 15 years previously. Four-vessel cerebral angiography was carried out in 40 patients with venous malformations. All patients had a physical examination, 16 had CT, and 22 were examined with MR imaging. One patient had MR angiography. **RESULTS:** Eighteen developmental venous anomalies were noted in 8 (20%) of 40 patients. Four patients had multiple anomalies, and these were bilateral in 1 patient. Developmental venous anomalies seen in association with cervicofacial, cutaneous, and mucosal venous malformations were remarkable in their absence of neurologic events and associated cavernoma; significance of ectatic venous convergence, extension, and preponderance of deep drainage routes; and frequency with which they multiple in occurrence. **CONCLUSION:** Developmental venous anomalies have a remarkable prevalence of 20% in patients with extensive superficial venous malformations. Therefore, it is important to search for a cerebral developmental venous anomaly when confronted with a cervicofacial venous malformation.

Index terms: Angioma; Brain, abnormalities and anomalies; Veins, abnormalities and anomalies

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Developmental venous anomalies (DVAs) in cerebral circulation are documented infrequently. These anomalies have long been incorrectly called *cerebral venous angiomas* (1, 2). In fact, according to Lasjaunias et al, "It is possible to demonstrate the 'normality' of these so-called 'venous angiomas' " (3). It is important to differentiate a DVA from a cavernoma. Some investigators consider the cavernoma to be the true venous malformation (4, 5). DVAs consist of dilated intramedullary veins converging into a large draining vein that reaches either the superficial or the deep system in an area in which there is an absence of normal draining veins (6). DVAs are known to be associated with other developmental vascular anomalies, especially cavernomas in the brain (7). The latter are composed of large sinusoidal vascular spaces with no intervening normal brain parenchyma (8).

We found specific patterns of DVAs in patients with extensive venous malformations of the head and neck. Superficial venous malformations of the head and neck are slow-flow vascular malformations (9) that manifest as soft-tissue swellings under normal or bluish skin that are enhanced in the face-down position or during the Valsalva maneuver. The blue cutaneous stain is characteristic and is caused by ectatic venous channels within the dermis. The overlying skin temperature is normal. There is no bruit or thrill.

Ten to 15 years earlier, the patients in our study underwent routine angiographic evaluation of the external carotid artery and cerebral vessels. The aim of this retrospective investigation was to describe the radiologic findings in these patients.

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From the Department of Neuroradiology and Therapeutic Angiography, Hôpital Lariboisière (M.B., O.E., J-P, G., F.G., D.H., D.R., J-J.M.), and the Department of Dermatology, Hôpital Cochin (O.E.), Paris, France.

Address reprint requests to Monique Boukobza, Department of Neuroradiology, Hôpital Lariboisière, 75475 Paris, Cedex 10, France.

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TABLE 1: Demographic and imaging findings in 40 patients with cervicofacial venous malformations

Case*	Age, y	Sex	Location of Venous Malformation	Angiographic Findings	MR Findings	CT Findings
1	16	F	J	N	N	N
2	28	M	OF	N	N	. . .
3	14	F	H	N	. . .	. . .
4	10	M	Bi	DVA	DVA	DVA
5	20	F	H	N	. . .	. . .
6	19	F	J	N	. . .	. . .
7	10	F	H	DVA	DVA	DVA
8	40	F	J+C	DVA	DVA	DVA
9	14	F	H	N	N	. . .
10	19	M	H	DVA	DVA	DVA
11	33	F	Bi	N	N	N
12	20	M	H	N	N	N
13	10	M	H	N	. . .	N
14	26	F	J	N	N	. . .
15	10	F	J	N	N	N
16	7	F	H	N	. . .	. . .
17	21	F	J	N	N	. . .
18	30	M	J	N	. . .	. . .
19	6	F	OF	N	N	N
20	18	F	H	N	N	. . .
21	50	M	H	N	. . .	. . .
22	41	F	H	DVA	. . .	. . .
23	19	M	Bi	N	N	N
24	40	F	J	N	N	. . .
25	14	F	J	N	. . .	. . .
26	33	M	H	N	. . .	. . .
27	20	F	J+C	DVA	DVA	. . .
28	20	F	J+C	N	. . .	. . .
29	5	F	OF	N	N	N
30	11	F	J	N	. . .	. . .
31	16	F	J+C	SS	. . .	SS
32	2	M	H	N	. . .	. . .
33	18	F	H	DVA	. . .	. . .
34	24	F	J	N	N	N
35	9	M	Bi	DVA	. . .	DVA
36	50	M	H	N	N	. . .
37	22	F	H	N	. . .	. . .
38	14	F	OF	N	. . .	. . .
39	15	F	J	N	N	N
40	10	M	H	N	. . .	. . .

Note.—H indicates hemifacial; Bi, bilateral; J, jugal; C, cervical, OF, orbitofrontal; N, normal; DVA, developmental venous anomaly; and SS, surgical sequelae.

* Underlined numbers indicate patients in whom angiography showed a DVA.

Materials and Methods

Between 1978 and 1985, 40 consecutive patients with cervicocephalic congenital venous malformations underwent internal and external carotid and vertebral arteriography. The group consisted of 13 male and 27 female subjects who were 2 to 50 years old (mean age, 20 years) at the time of the angiographic procedure (Table 1). Twenty-four patients were younger than 20 years old at the time of radiologic study. None of the 40 patients were examined because of neurologic symptoms; rather, cerebral angiography was part of a systematic investigation of the brain done in parallel with a study of their facial venous malformations. Intraarterial cerebral arteriography was performed with the use of film-screen or digital-subtraction techniques. In addition, all patients had a physical examination performed by an interdisciplinary team of physicians specializing in vascular anomalies.

Sixteen of the 40 patients also had computed tomography (CT) of the face and brain, in which both precontrast and postcontrast studies were obtained. Sections were 5 mm thick below the tentorium and 10 mm thick above the tentorium.

In 22 patients, magnetic resonance (MR) imaging was performed on a 0.5-T magnet using routine T1-weighted (400/30 [repetition time/echo time]) and T2-weighted (2300/25,100) spin-echo imaging. MR angiograms were

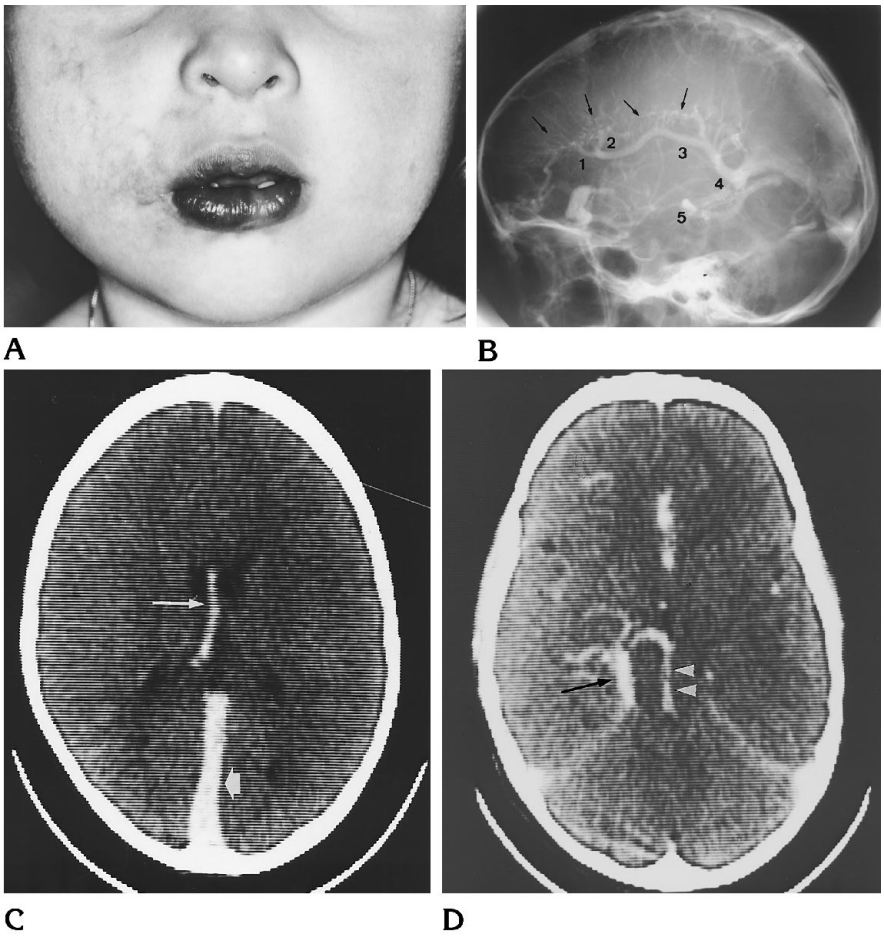

Fig 1. *A*, Patient 7: 10-year-old girl with hemifacial venous malformation. There is blue discoloration of the skin with slight distortion of the facial structures when standing upright.

B, Lateral view of right carotid angiography. The early venous phase shows a lack of cortical veins in the frontal lobe. Numerous dilated deep medullary veins in the periventricular area (*arrows*) drain into large veins situated along the wall of the lateral ventricle (subependymal veins), which drain at the junction of the internal cerebral vein and the great vein of Galen. The septal vein (*1*), the thalamostriate vein (*2*), the internal cerebral vein (*3*), and the vein of Galen (*4*) are dilated and tortuous. The dilated deep medullary veins have a fanlike shape. The basal vein is also dilated (*5*). The pattern of venous drainage in the frontal area is similar to that of case 27 (Fig 3).

C and *D*, Postcontrast CT scans show a markedly dilated internal cerebral vein (*long arrow* in *C*) and straight sinus (*short arrow* in *C*); the ectatic right basal vein (*arrowheads* in *D*) and an abnormal draining vein crossing the brain stem (*arrow* in *D*) are also seen.

obtained in one patient with the use of a two-dimensional phase-contrast technique. Acquisition parameters of the gradient-echo sequences were 60/26 with a 25° flip angle. The two-dimensional phase-contrast technique was preferred to the time-of-flight technique for detecting slow-flow vessels that comprise DVAs. MR imaging of the superficial venous malformations was done on the same magnet in all 22 patients with the same spin-echo T1-weighted and T2-weighted sequences in the axial, sagittal, and coronal planes.

DVA patterns were classified first as juxtacortical/subcortical or paraventricular, depending on the location of the dilated medullary veins, and second as deep or superficial, depending on their type of drainage (10).

Results

The superficial venous malformations were facial and bilateral in 4 patients, hemifacial in 17, jugal and cervical in 15, and orbital and frontal in 4. Except for the 4 patients who had frontal and orbital malformations, all patients also had intraoral mucosal malformations. One woman (case 31) had a frontal cerebral cavernoma treated surgically following cerebromeningeal hemorrhage and hemiplegia. She was neurologically normal at the time of her examination in our unit for extensive jugal and cervical superficial venous malformations; DVAs were not seen on the angiographic study we performed.

A diagnosis of DVA was made in 8 of the 40 patients (cases 4, 7, 8, 10, 22, 27, 33, and 35). Of these patients, 3 were male and 5 were female. All 8 had extensive cutaneous venous malformations: hemifacial in 5 (Fig 1), facially bilateral in 2 (Fig 2), and located in the cheek and neck in 1. All patients had oral venous malformations (Fig 3). None of the 4 patients with orbitofrontal malformations had a DVA.

DVA was, in every case, an incidental finding. None of these patients had had neurologic deficits, although they occasionally complained of headaches. None were shown by CT or MR imaging to have hemorrhaged from their DVAs. The arterial and capillary angiographic phases

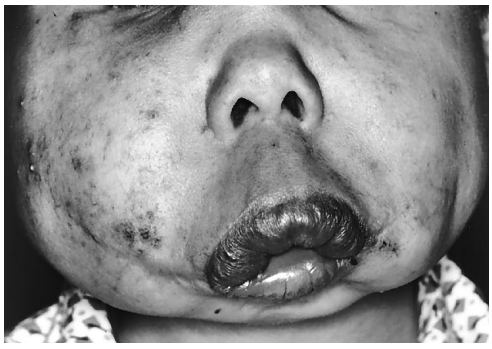

Fig 2. Patient 35: 9-year-old boy with bilateral facial venous malformation. The skin is blue, the features distorted, and cosmetic and functional consequences are severe.

were normal. The dysmorphic venous drainage was observed in the venous phase. In most cases there was poor opacification of the cortical veins and superior sagittal sinus.

Unilateral DVAs were discovered in 7 patients (cases 4, 7, 8, 10, 27, 33, and 35); in one patient (case 22), a woman with severe hemifacial venous malformations, the DVAs were multiple and bilateral. A total of 18 DVAs were found in the 8 patients; they were multiple in 5 cases and solitary in 3.

Deep drainage was documented in 14 of these DVAs, and superficial drainage was seen in 4. Drainage was superficial in the 3 patients with a solitary DVA (cases 8 and 10 in the cerebellar hemisphere; case 33 in the frontal lobe). In one patient (case 35), drainage was both superficial and deep.

The majority (n = 15) of the DVAs were supratentorial. In three patients (cases 7, 8, and 10). DVAs were in the posterior cranial fossa. In two patients (cases 8 and 10), the DVA was in the cerebellar hemisphere. Another patient (case 7) had a draining vein passing through the brain stem at the pontomesencephalic junction, easily depicted on enhanced CT scans (see Fig 1); this patient also had a supratentorial DVA. In three patients the entire corpus callosum was involved (cases 7, 27, and 35), and in one patient (case 22) the genu and splenium of the corpus callosum had anomalous venous drainage.

In five of seven patients with supratentorial DVAs, several uncommon pathways of cerebral venous drainage were found through the white matter. In these patients (cases 4, 7, 22, 27, and 35) the draining veins ended variously, fanning out toward an enlarged basal vein, an internal cerebral vein, the septal vein, or the vein of Galen confluence. All drainage pathways and routes of drainage, for the eight DVAs are listed in Table 2.

Enhanced CT scans and/or MR images were obtained in six of the eight patients in whom a DVA was found at angiography (Table 1). In all, five CT and five MR studies were available from which we identified the deep dilated veins that were seen at angiography. CT scans showed deep dilated veins in the torcular region or in the region of the vein of Galen confluence, as well as in the midline (Figure 1B and C). In one patient (case 27), T1-weighted MR images and the much better T2-weighted images showed stripes of flow void in the midline, corresponding to the dilated internal cerebral vein and vein of Galen confluence (Fig 3A–C). Dilated medullary veins were not seen. In the same patient, the dilated collectors in the midline were clearly documented by MR angiography (Fig 3D). Additionally, in one patient (case 8) CT and MR imaging revealed a sphenoidal meningocele.

Discussion

In most patients, opacification of a DVA occurs within the usual time sequence in the angiographic venous phase, just as for normal veins. The arterial and parenchymal phases are usually normal. In some cases, a capillary blush is seen in the territory drained by the DVA. Unenhanced MR images show DVAs as tubular or small round structures with low signal intensity caused by flow void, or with high signal intensity caused by slow flow rephasing (11), which enhances after intravenous administration of contrast material (12). Contrast-enhanced MR imaging and MR angiography have proved to be superior in the visualization of DVAs (12, 13).

DVAs of the brain are variations of the normal cerebral venous drainage pathways. In most people, DVAs are an incidental finding and are only rarely symptomatic. They are venous deviations in which brain parenchyma is normal between the dilated veins. They are well tolerated (3), and most authors consider them to be uncommon and to be clinically silent venous patterns (14–18). As such, DVAs cannot be termed abnormal. It is more accurate to say that DVAs are uncommon trajectories of venous drainage within areas of normal white matter. Nonetheless, DVAs are less flexible than the normal venous pathways (3). Some authors be-

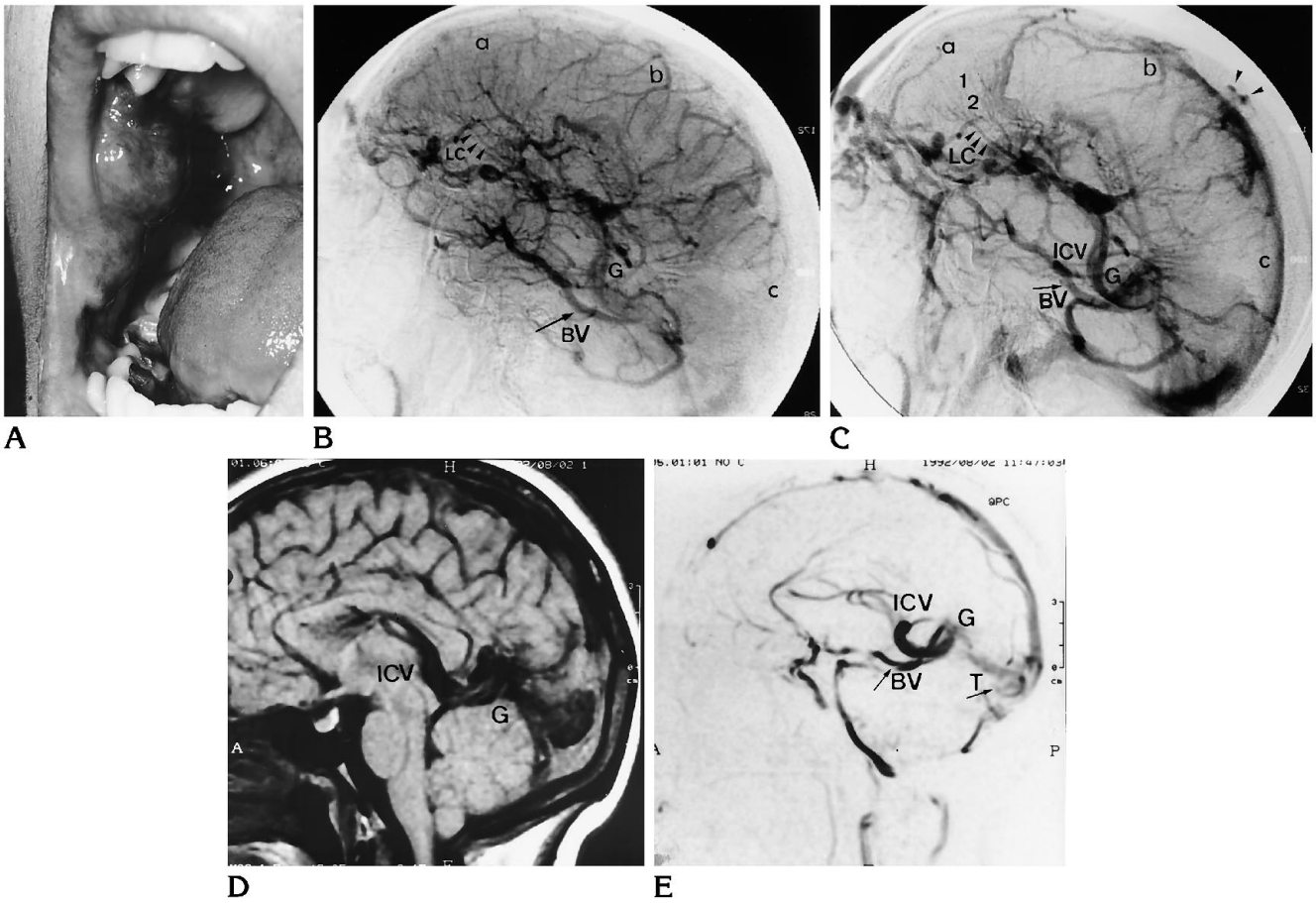

Fig 3. Patient 27: 20-year-old woman with intraoral venous malformation causing blue venous pouches in the jugal mucous membrane (*A*).

Lateral view of right carotid angiography in early (*B*) and middle (*C*) venous phase. There is a striking lack of cortical veins in the frontal and occipital regions, and the anterior part of the superior sagittal sinus is not seen. Numerous dilated fine deep medullary veins (*1*), clearly visible in the right frontal (*a*) and occipital (*c*) areas of the subcortical regions, drain into marked dilated deep medullary veins (*2*), then into subependymal veins, then into an ectatic septal vein (*arrow*), and finally join the internal cerebral vein. There is an enlargement and a tortuous aspect of the internal cerebral vein (*ICV*), the basal vein (*BV*), and the vein of Galen (*G*). In the periventricular area (*b*), numerous dilated deep veins drain into the longitudinal caudate vein (*LC*, *arrowheads*). Finally, typical radiating medullary veins are seen coming from cortical areas, connecting to large draining veins, and draining into the internal cerebral vein. As in Figure 1, the dilated deep medullary veins are fanlike in shape. Diploic veins (*arrowheads*) are filled, probably by the superior sagittal sinus.

D, T1-weighted (600/25) sagittal spin-echo MR image reveals enlarged deep veins with typical signal voids corresponding to a dilated ICV and vein of Galen (*G*).

E, Two-dimensional phase-contrast MR angiogram (60/26, 25° flip angle) in the sagittal plane reveals an enlargement of the ICV, vein of Galen (*G*), torcular Herophili (*T*), and basal vein (*BV*).

lieve that DVAs can cause intracranial hemorrhage (7, 19–21), epilepsy (20, 22, 23), focal neurologic deficit (23), and headaches. However, in some of those symptomatic cases diagnosed before the advent of MR imaging, there might have been a cerebral cavernoma, which would have gone undetected on angiographic studies and CT scans.

Some of our patients with cervicofacial superficial venous malformations had cerebral DVAs, which appeared to be well tolerated. They consisted of anatomic variants of the cerebral venous system, with channels that drained the normal brain and normal arterial territory. The atypical venous drainage functioned normally and the DVAs were discovered by chance. All patients had unobstructed veins, which might account for the universal absence of neurologic symptoms.

Some investigators have reported the occurrence of neurologic symptoms related to intracerebral hemorrhage among patients who had DVAs in association with cavernoma. Symptoms were attributable to the area of the brain in

TABLE 2: Classification of developmental venous anomalies by location of dilated medullary veins (subcortical or paraventricular) and by drainage route (deep or superficial)

Case	Drainage Pathway	Site of Developmental Venous Anomaly	Drainage Route
4	Septal vein to internal cerebral vein	Paraventricular	Deep
	Subependymal vein to internal cerebral vein	Subcortical	Deep
	Basal vein	Paraventricular	Deep
7	Septal vein to internal cerebral vein	Paraventricular	Deep
	Subependymal vein of lateral ventricle to internal cerebral vein	Subcortical	Deep
	Subependymal vein of lateral ventricle to thalamostriate vein	Subcortical	Deep
	Transpontine vein to anterior pontomedullary vein	Subcortical	Deep
8	Inferior cerebellar vein	Subcortical	Superficial
10	Inferior cerebellar vein	Subcortical	Superficial
22	Subependymal vein of lateral ventricle to internal cerebral vein	Subcortical	Deep
	Septal vein to internal cerebral vein (bilateral)	Paraventricular	Deep
27	Septal vein to internal cerebral vein	Paraventricular	Deep
	Subependymal vein of lateral ventricle to internal cerebral vein	Subcortical	Deep
	Subependymal vein of lateral ventricle to thalamostriate vein	Subcortical	Deep
33	Superficial frontal cerebral vein	Paraventricular	Superficial
35	Septal vein to internal cerebral vein	Paraventricular	Deep
	Subependymal vein of lateral ventricle to internal cerebral vein	Subcortical	Deep
	Superficial frontal cerebral vein to sagittal superior sinus	Paraventricular	Superficial

which both the DVA and the cavernoma were located (16, 17, 24, 25). The reported frequency of a cavernoma occurring in association with a cerebral DVA ranges from 8% to 33% (26). Such an association was not found in our series. Only one patient (case 31) of the 40 studied had a frontal cavernoma that had been treated previously, and this young woman did not have a DVA.

Our patients with combined venous malformation and DVA had atypical transparenchymatous cerebral venous drainage. Multiple draining vessels in the white matter converged, in a fanlike pattern, toward enlarged venous channels. Anomalies included an enlargement and a tortuosity of the internal cerebral vein, the basal vein and its tributaries, and the vein of Galen confluence. In case 7, a draining vein passing through the brain stem was observed. A similar pattern was described in 1 case reported by Ostertun and Solymosi (26) and in 4 of 11 cases reported by Damiano et al (27). In our series, the venous drainage anomalies were most commonly unilateral (7 of 8 cases). They were multiple in 5 cases. In the literature, multiple DVAs have been reported to occur in about 25% of the cases (16, 17). In 1 of our patients, a woman with unilateral hemifacial venous malformations (case 22), the DVAs were bilateral.

In addition to the 20% rate of occurrence of cerebral DVAs that were associated with extensive superficial venous malformations, our cases are remarkable in several aspects: absence of neurologic complications, absence of cerebral cavernoma in association with DVA, significance of ectatic venous convergence, frequency of multiple DVAs in the same patient, and a preponderance of deep drainage patterns. In our group of patients with cutaneous and mucosal venous malformations of the head and neck, the DVAs usually consisted of hypoplasia of the superficial venous system draining to deep enlarged venous collectors through the white matter.

Some of the enlarged deep veins we saw in our patients could represent venous channels that were normally wider during the embryonic period, such as the veins of the choroid plexus.

This conjecture was proved in studies of some patients with Sturge-Weber syndrome (28, 29).

Saito and Kobayashi (14) have suggested that DVAs are the result of an error in vascular embryogenesis. They hypothesized that occlusion and maldevelopment of the normal venous drainage pathway in part of the brain results in dilated medullary veins converging toward an enlarged transcerebral vein. This theory is supported by the finding of a complete absence of normal draining veins in the region involved. It is possible that this particular anatomic variant of the cerebral venous drainage pattern mimics an early embryonic stage of venous system development. Nonetheless, at the present time, the embryogenesis of DVA is unclear, as is the embryogenesis of cervicofacial venous malformations.

Conclusion

DVAs occur in about 0.05% to 0.25% of the general population (19, 26, 30). In contrast, the frequency of DVAs in patients with extensive cervicofacial venous malformations was found to be 20% in our series of 40 patients. We do not know the true prevalence of DVAs in patients with venous malformations, as only patients with large cervicofacial superficial anomalies underwent arteriographic examination. The frequency of DVAs in patients with a spectrum of minor to major superficial cervicofacial venous malformations is under evaluation in a prospective study using MR imaging and MR angiography. We no longer use arteriography to examine patients with superficial venous malformations, because it is an inefficient technique for studying this type of anomaly. MR imaging is the most reliable noninvasive technique for documenting superficial cutaneous or mucosal vascular malformations of the head and neck. MR imaging and MR angiography are also the favored imaging methods for detecting DVAs in the brain and for making the differential diagnosis between DVAs and cavernomas (12, 13). We think MR imaging is important in cases of vascular malformations because of their high rate of association with DVAs. MR angiography combined with MR imaging offers a noninvasive diagnostic strategy adequate for evaluating DVAs.

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References

1. Valavanis A, Schefer S, Wichmann W. Cavernomas and venous angiomas of the brain. *Riv Neuroradiol* 1990;3(suppl 2):89–93
2. Augustyn GT, Scott JA, Olson E, Gilmor RL, Edwards MK. Cerebral venous angiomas: MR imaging. *Radiology* 1985;156:391–395
3. Lasjaunias P, Burrows P, Planet C. Developmental venous anomalies (DVA): the so-called venous angioma. *Neurosurg Rev* 1986; 9:233–244
4. Gomori JM, Grossman RI, Goldberg HI, Hackney DB, Zimmerman RA, Bilaniuk LT. Occult cerebral vascular malformations: high-field MR imaging. *Radiology* 1986;158:707–713
5. Rapacki TFX, Brantley MJ, Furlow TW, Geyer CA, Toro VE, George ED. Heterogeneity of cerebral cavernous hemangiomas diagnosed by MR imaging. *J Comput Assist Tomogr* 1990;14: 18–25
6. Huang YP, Patel SC, Robbins A, Chandhary M. Cerebral venous malformations and a new classification of cerebral vascular malformations. In: Kapp JP, Schmidek HH, eds. *The Cerebral Venous System and Its Disorders*. New York, NY: Grune & Stratton, 1984: 373–474
7. Thron A, Petersen D, Voigt K. Neuroradiologie, Klinik und Pathologie der zerabralen venösen angiome. *Radiologe* 1982;22:389–399
8. Simard JM, Garci-Bengocheat F, Ballinger WE, Mickle JM, Quisling RC. Cavernous angioma: a review of 126 collected and 12 new clinical cases. *Neurosurgery* 1986;18:162–172
9. Enjolras O, Mulliken JB. The current management of vascular birthmarks. *Pediatr Dermatol* 1993;10:311–333
10. Valavanis A, Wellauer J, Yasargil MG. The radiological diagnosis of cerebral venous angioma: cerebral angiography and CT. *Neuroradiology* 1983;24:193–199
11. Uchino A, Imada H, Ohno M. Magnetic resonance imaging of intracranial venous angiomas. *Clin Imaging* 1990;14:309–314
12. Wilms G, Demaerel P, Marchal G, Baert AL, Plets C. Gadolinium MR imaging of cerebral venous angiomas with emphasis on their drainage. *J Comput Assist Tomogr* 1991;15:199–206
13. Marchal G, Bosmans H, Van Frayenhoven L, et al. Intracranial vascular lesions: optimization and clinical evaluation of three-dimensional time-of-flight MR angiography. *Radiology* 1990;175: 443–448
14. Saito Y, Kobayashi N. Cerebral venous angiomas: clinical evaluation and possible etiology. *Radiology* 1981;139:87–94
15. Martin NA, Wilson CB, Stein BM. Venous and cavernous malformations. In: Wilson CB, Stein BM, eds. *Intracranial Arteriovenous Malformations*. Baltimore, Md: Williams & Wilkins, 1984:234–245
16. Rigamonti D, Drayer BP, Johnson PC, Hadley MN, Zabramski J, Spetzler RF. The MRI appearance of cavernous malformations (angiomas). *J Neurosurg* 1987;67:518–524
17. Rigamonti D, Spetzler RF, Drayer BP, et al. Appearance of venous malformations on magnetic resonance imaging. *J Neurosurg* 1988;69:535–539
18. Abe M, Asfora WT, DeSalles AA, Kjellberg RN. Cerebellar venous angioma associated with angiographically occult brain stem vascular malformation: report of two cases. *Surg Neurol* 1990;33: 400–403

19. McCormick WF, Hardman JM, Boulter TR. Vascular malformations (angiomas) of the brain with special reference to their occurring in the posterior fossa. *J Neurosurg* 1968;28:241–251
20. Maehara T, Tasaka A. Cerebral venous angioma: computerized tomography and angiographic diagnosis. *Neuroradiology* 1978; 16:296–298
21. Malik GM, Morgan JK, Boulos RS, Ausman JI. Venous angiomas: an underestimated cause of intracranial hemorrhage. *Surg Neurol* 1981;30:350–358
22. Tomaccini D, Venturi C, Scarfo GB. Cerebral venous angioma, cutaneous angioma, facial asymmetry and recurrent stroke: a case report. *Eur Neurol* 1981;20:29–32
23. Garner TB, Del Curling O, Kelly DL, Laster DW. The natural history of intracranial venous angiomas. *J Neurosurg* 1991;75: 715–722
24. Goulao A, Alvarez H, Garcia Monaco R, Pruvost P, Lasjaunias P. Venous anomalies and abnormalities of the posterior fossa. *Neuroradiology* 1990;31:476–482
25. Wilms G, Bleus E, Demaerel P, et al. Simultaneous occurrence of developmental venous anomalies and cavernous angiomas. *AJNR Am J Neuroradiol* 1994;15:1247–1254
26. Ostertun B, Solymosi L. Magnetic resonance angiography of cerebral developmental venous anomalies: its role in differential diagnosis. *Neuroradiology* 1993;35:97–104
27. Damiano TR, Truwit CL, Dowd CF, Symonds DL. Posterior fossa venous angiomas with drainage through the brain stem. *AJNR Am J Neuroradiol* 1994;15:643–652
28. Bentson JR, Wilson GH, Newton TH. Cerebral venous drainage pattern of the Sturge-Weber syndrome. *Radiology* 1971;101:111–118
29. Vogl TJ, Stemmler J, Bergman C, Pfluger TH, Eggr E, Lissner J. MR and MR angiography of Sturge-Weber syndrome. *AJNR Am J Neuroradiol* 1993;14:417–425
30. Sarwar M, McCormick WF. Intracerebral venous angioma: case report and review. *Arch Neurol* 1978;35:323–325