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Developmental venous anomaly in the newborn brain

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Abstract
Introduction Cerebral developmental venous anomaly (DVA) is considered a benign anatomical variant of parenchymal venous drainage; it is the most common vascular malformation seen in the adult brain. Despite its assumed congenital origin, little is known about DVA in the neonatal brain. We report here the first cohort study of 14 neonates with DVA.
Methods Fourteen infants (seven preterm) with DVA diagnosed neonatally using cranial ultrasound (cUS) and magnetic resonance imaging (MRI) from three tertiary neonatal units over 14 years are reviewed.
Results DVA was first detected on cUS in 6 and on MRI in 8 of the 14 infants. The cUS appearances of DVA showed a focal fairly uniform area of increased echogenicity, often (86 %) adjacent to the lateral ventricle and located in the frontal lobe (58 %). Blood flow in the dilated collector vein detected by Doppler ultrasound (US) varied between cases (venous flow pattern in ten and arterialized in four). The appearance on conventional MRI was similar to findings in adults. Serial imaging showed a fairly constant appearance to the DVAs in some cases while others varied considerably regarding anatomical extent and flow velocity.
Conclusions This case series underlines that a neonatal diagnosis of DVA is possible with carefully performed cUS and MRI and that DVA tends to be an incidental finding with a diverse spectrum of imaging appearances. Serial imaging suggests that some DVAs undergo dynamic changes during the neonatal period and early infancy; this may contribute to why diagnosis is rare at this age.

Keywords Venous angioma · Vascular malformation · MRI · Ultrasound · Infant

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Introduction

Cerebral developmental venous anomaly (DVA) is considered a benign anatomical variant of parenchymal venous drainage. It is the most common cerebral vascular malformation found in the adult brain with an incidence at autopsy of 2.6 % and in imaging studies of 0.5 to 0.7 % [1–5]. DVA is characterized by an umbrella-like convergence of multiple venules that merge into a dilated collector vein, which drains either superficially into a cortical vein or sinus or into the subependymal deep venous system, or in 10 % of cases, into both [1]. The pathogenesis of DVA is not fully understood, but it is generally thought to be of embryonic origin. A primary dysplasia of capillaries and small transcerebral veins or a compensatory mechanism caused by an intrauterine vascular occlusion resulting in thrombosis of normal venous pathways is discussed [6–8]. Despite its embryonic origin, only four cases

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(one isolated asymptomatic DVA and three associated with other congenital brain abnormalities or complicated by haemorrhage) are reported in the paediatric literature [9–12]. This may be due to the fact that DVA is considered a normal variant and therefore may go unreported at least as an abnormal finding. However, the difference between the few neonatal reports and the large number of publications about DVA in adults is striking. Little is published about the appearances of neonatal cerebral DVA on magnetic resonance imaging (MRI) and even less is known about the findings on cranial ultrasound (cUS)/Doppler ultrasound (US) despite the fact that cUS and increasingly MRI is performed as a screening tool in preterm and term infants undergoing neonatal intensive care and is almost always used as a diagnostic tool in neurologically symptomatic newborns.

This is in agreement with our own observation that DVA is a rare diagnosis in the neonatal period although all our three institutions have a strong focus on neonatal neuro-imaging and the observation of a DVA on either cUS or MRI would always be reported.

The aim of this study was to review 14 neonatal cases of DVA, describe the cUS/ Doppler US and MRI findings, and discuss possible reasons why DVA is a rare diagnosis in the neonatal period.

Materials and methods

We reviewed 14 neonatal cases of DVA diagnosed using cUS and MRI in three tertiary neonatal intensive care units between January 1998 and December 2012. Considering the retrospective design of our study, we are unable to be exact about the total number of infants screened with cUS and MRI during the study period. However, the three institutions have together approximately 1,900 NICU admissions/year. All three have a longstanding tradition in neonatal neuroimaging and in nearly all infants admitted to the NICU, at least one cUS is performed. In recent years, approximately 850 neonatal MRIs/year have been performed for various indications. All neonatal MRIs are reviewed by a paediatric radiologist and a neonatologist experienced in neuroimaging and a DVA would always be reported on whether detected on cUS or MRI in all three institutions.

All DVAs reported here were detected incidentally on imaging performed because of prematurity, growth restriction or neurological problems. Patient characteristics are given in Table 1.

Results

Presentation of DVA and clinical follow-up

For details, see Table 2.

Table 1 Patient characteristics

No.	Gestational age (weeks+days)	Birth weight (g)	IUGR	Sex
1	28+1	870	Yes	Female
2	29+4	1,430	No	Male
3	31+1	1,600	No	Female
4	32+4	1,650	No	Male
5	33	2,130	No	Female
6	35+2	2,970	No	Female
7	36+4	1,790	Yes	Female
8	38+2	2,015	Yes	Male
9	39+4	2,650	Yes	Female
10	40	2,680	Yes	Male
11	40+5	3,280	No	Male
12	41	3,210	No	Female
13	41	3,345	No	Female
14	42+1	2,700	Yes	Male

Presentation on cUS and Doppler US findings

The cUS appearances of DVA were of a focal fairly uniform area of increased echogenicity, adjacent to the lateral ventricle in 86 % of the cases. Representative images are presented in Fig. 1. The intensity of the hyperechogenicity varied between infants and also intra-individually over time in some infants (see below). Doppler US visualized blood flow in the collector vein, typically localized in the centre of the hyperechogenicity. Blood flow velocity in the dilated collector vein showed a very slow venous flow pattern in ten and an arterialized flow pattern in four cases. In one case, Doppler US failed to visualize flow on day 1 although the DVA was visible on the 2D real-time image (a video is provided as a Supplementary material). When this infant's cUS was repeated at 1 month of age, the venous flow in the collector vein appeared to have increased and was easy to detect with Doppler US. At 4 months of age, the flow was detectable but was decreased and the hyperechogenicity was barely visible (Fig. 2).

Presentation on conventional MRI, diffusion-weighted imaging, susceptibility-weighted imaging, and angiography

All infants underwent conventional MRI and 13/14 also had diffusion-weighted imaging (DWI). Conventional angiography was only performed in one infant (case 10, Figs. 3 and 4). As in adults, DVAs were predominantly located in the frontal lobe (58 %). Interestingly, in this neonatal cohort, DVAs were exclusively found supratentorially and all but one in the cerebral hemispheres. Figure 5 shows the uncommon localization of a DVA in the thalamus (case 7).

The appearance on conventional MRI was comparable to findings in adults. Signal abnormalities, generally of long T1 and T2, in the surrounding parenchyma on conventional MRI

Table 2 Presentation of DVA and clinical follow-up

Case	Reason for imaging	Location of DVA	Flow pattern on Doppler sonography	DVA first detected on	Signal intensity abnormalities in surrounding parenchyma	Other cerebral abnormalities	DWI abnormal	Serial cUS	Serial Doppler	Serial MRI	Age at last follow-up	Neurodevelopmental status at follow-up
1	Prematurity	Left frontal	Venous	MRI	cUS yes/MRI no	No	No	Yes	No	No	24 months	Normal
2	Prematurity	Left frontal	Venous	MRI	cUS yes/MRI no	Small haemorrhage adjacent to DVA	No	Yes	No	Yes	3 years	Normal
3	Prematurity	Right parietal	Venous	cUS	cUS yes/MRI no	Punctate lesions in periventricular white matter	No	Yes	Yes	No	5 months	Normal
4	Prematurity	Right parietal	Arterialized	cUS	CUS yes/MRI no	No	No	Yes	Yes	No	14 months	Normal
5	Prematurity	Right frontal	Arterialized	MRI	cUS yes/MRI yes	No	No[b]	Yes	Only 1	No	11 years	Normal
6	Group B streptococcal meningitis	Right parietal	Venous	cUS	cUS yes/MRI no	Meningitis, left MCA infarction, and extensive DWI abnormalities in the BGT	Yes[a]	Yes	No	No	3 weeks	Died
7	Seizures	Left frontal	Venous	MRI	cUS yes/MRI yes	No	No	Yes	No	Yes	19 months	Normal
8	IUGR	Left frontal	Venous	cUS	cUS yes/MRI no	No	No	Yes	Yes	No	2 years	Normal
9	Seizures	Right temporal	Venous	MRI	cUS yes/MRI no	Small haemorrhage adjacent to DVA	No	Yes	Yes	Yes	30 months	Normal
10	IUGR	Left frontal	Arterialized	cUS	cUS yes/MRI no	Cavernous malformation and haemorrhage	Yes	Yes	Yes	Yes	26 months	Normal
11	Seizures	Right parietal	Arterialized	MRI	cUS no/MRI no	No	No	No	No	Yes	2 years	Normal
12	Seizures	Left thalamus	Venous	MRI	cUS no/MRI no	No	No	Yes	No	No	19 months	Normal
13	Asphyxia	Right frontal	Venous	cUS	cUS yes/MRI no	No	No	Yes	No	Yes	5 years	Normal
14	Asphyxia	Left frontal	Venous	MRI	cUS no/MRI yes	BGT lesions associated with HIE	No	Yes	No	Yes	3 years	Dyskinetic cerebral palsy DQ 83

BGT basal ganglia and thalami, *DWI* diffusion-weighted imaging, *HIE* hypoxic–ischaemic encephalopathy, *IUGR* intrauterine growth restriction, *MCA* middle cerebral artery, *NA* not applicable

[a] Not associated with DVA

[b] DWI not performed

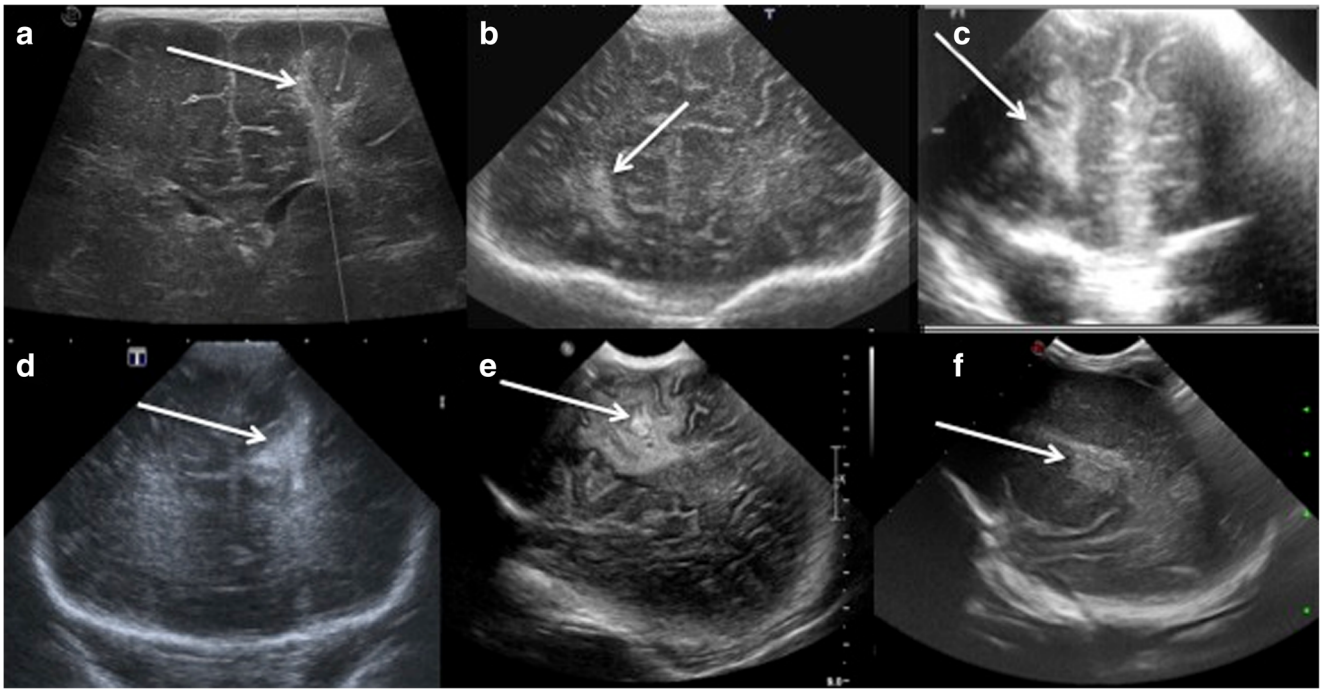

Fig. 1 Representative cUS images: **a** cases 8, **b** 13, **c** 5, **d** 7, **e** 10, and **f** 13. DVA (*arrow*) presents as an area of increased echogenicity initially difficult to distinguish from arterial or venous infarction

were found in 3/14 cases (Fig. 6). DWI was performed in 13/14 infants and was normal in 12. One infant had persistent extensive diffusion changes in the surrounding parenchyma at the age of 3 months. Figures 3 and 4 show the findings in this infant who had an arterialized flow pattern in the DVA and also a cavernous malformation (CM) and haemorrhage (case 10). Angiography in this infant showed a capillary stain, but no arterial feeders could be detected.

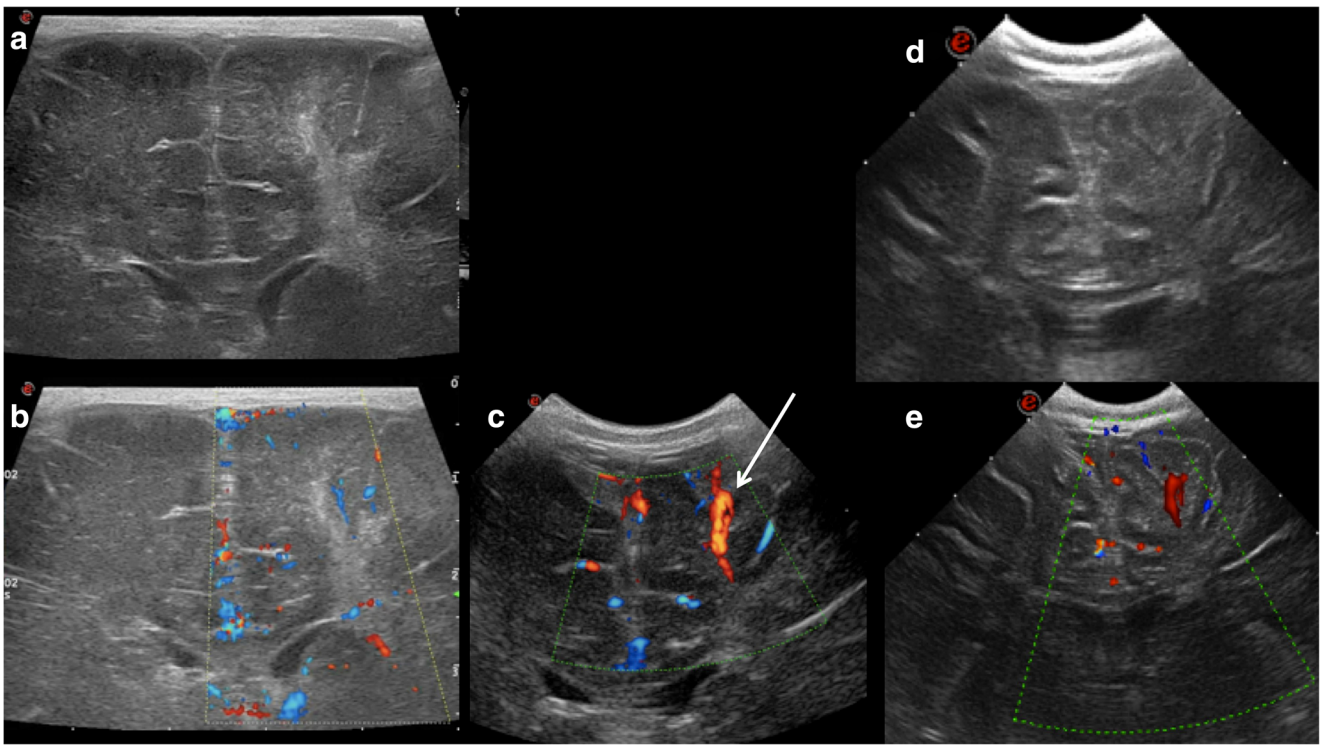

Fig. 2 cUS and Doppler US findings in case 8 at the age of 1 day (**a**, **b**), at the age 1 (**c**) and 4 months (**d**, **e**). Note the change in the hyperechogenicity over time (**a**, **d**). While Doppler US failed to visualize flow in the collector vein on day 1 (**b**), flow velocity increased and was easily detectable at 1 month of age (**c**, *arrow*). At 4 months, the dilated vein is still visible, but flow velocity decreased (**e**)

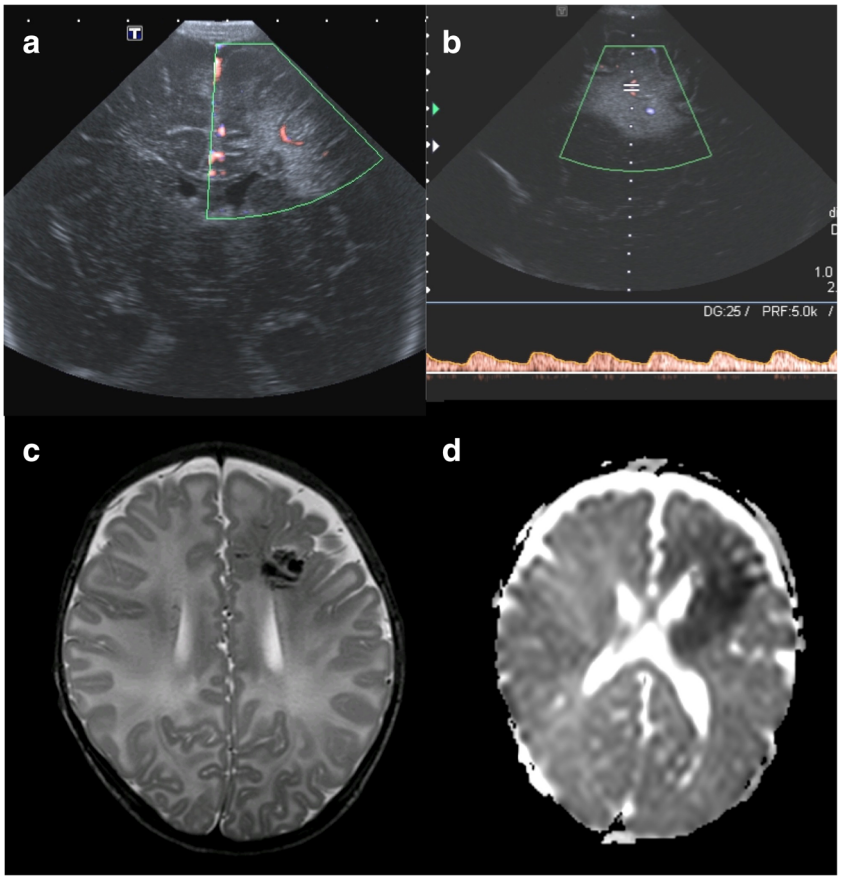

Fig. 3 Case 10 with an arterialized DVA, associated cavernous malformation and haemorrhage: on coronal cUS/Doppler US images at the age of 30 days (**a**). The arterialized flow pattern in the collector vein could be detected with Doppler US (**b**). While axial T2-weighted images showed a stable appearance at 30 days (**c**) and 3 months, DWI at 3 months (**d**) revealed extensive diffusion changes in the surrounding parenchyma

Susceptibility-weighted imaging (SWI) was performed in 9/14 cases. In one infant, the SWI was of poor quality due to movement artefacts. In the other eight cases, SWI was found to be helpful as the DVA could be delineated more clearly than

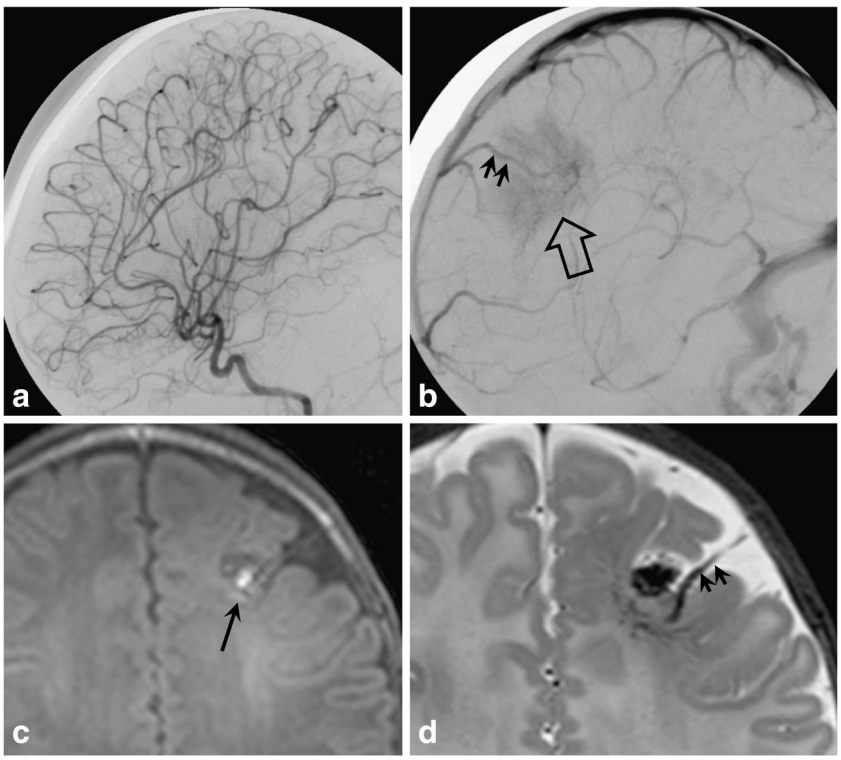

Fig. 4 Angio-DSA (**a**, **b**) and axial T1 (**c**) and T2SE (**d**) MR sequences of case 10. No arteriovenous shunt is seen on the arterial phase on DSA (**a**). However, a DVA with a deep capillary stain (**b**, *empty arrow*) that is draining through a cortical vein (**b**, *double arrows*) is clearly identified on the venous phase. The cavernoma was diagnosed on a follow up MRI (**c**, **d**) that showed the classical findings of a type 2 cavernoma. The lesion is partially hyperintense on T1-weighted images and next to the DVA (**d**, the *double arrows* indicate the draining vein)

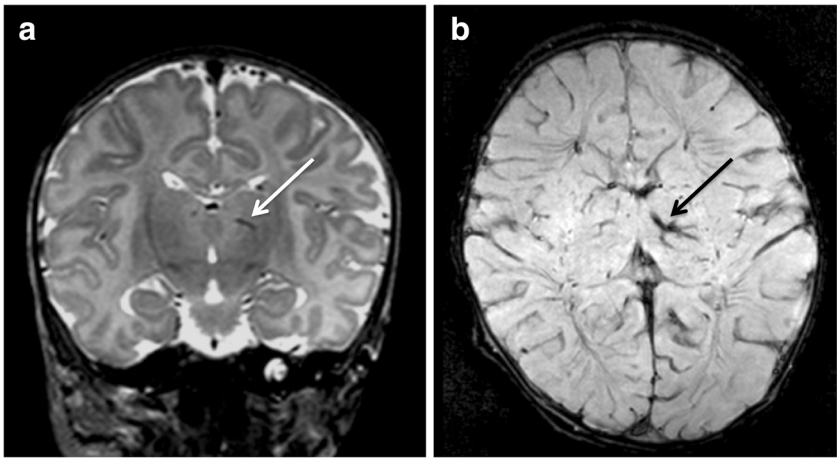

Fig. 5 **a** Coronal T2-weighted and **b** axial susceptibility-weighted image of case 12 showing the relatively uncommon localization of a DVA (*arrow*) in the left thalamus

on conventional MR imaging. However, in none of the infants did the SWI add findings not seen on conventional MR imaging.

Evolution and regression on serial imaging

Serial cUS scans were performed in all neonates and serial MRI scans in 7/14 cases (Table 2). These revealed that cUS, Doppler US, and MRI appearance of the DVAs were stable in some, but varied considerably in 10/14 cases (Figs. 2 and 6; Table 3); marked changes to the extent and intensity of the hyperechogenicity of the DVA and its surrounding parenchyma on cUS and tissue signal intensity changes on MRI, as well as changes in the blood flow velocity in the collector vein measured by Doppler US were found. The changes varied considerably between subjects. While hyperechogenicity and blood flow velocity increased in some, it decreased in others. No characteristic pattern of changes over time could be found. This information is detailed in Table 3.

Associated cerebral abnormalities and neonatal complications

One infant had a CM in addition to the DVA (case 10, Figs. 3 and 4); no congenital cerebral abnormalities other than the DVA were detected in the other infants. Punctate lesions in the unmyelinated periventricular white matter (high signal intensity on T1-weighted and hypointensity on T2-weighted images) were found in one preterm infant. These kind of white matter punctate lesions are a common finding in preterm infants [13–15] and are unlikely to be related to the DVA. Cerebral haemorrhage complicated the clinical course of three infants, including the one with a CM. Seizures were observed in four infants and were attributed to perinatal asphyxia in two and cerebral haemorrhage in one. In one neonate, the cause of the seizures remained unclear but was most likely unrelated to the DVA. Case 6 suffered from severe group B streptococcal meningitis and developed an extensive left-sided middle cerebral artery infarct, which appeared to be unrelated to the DVA. This infant died.

Atrophy around the DVA was observed in two cases (cases 9 and 10) and was likely related to the associated haemorrhage in that area rather than the DVA per se.

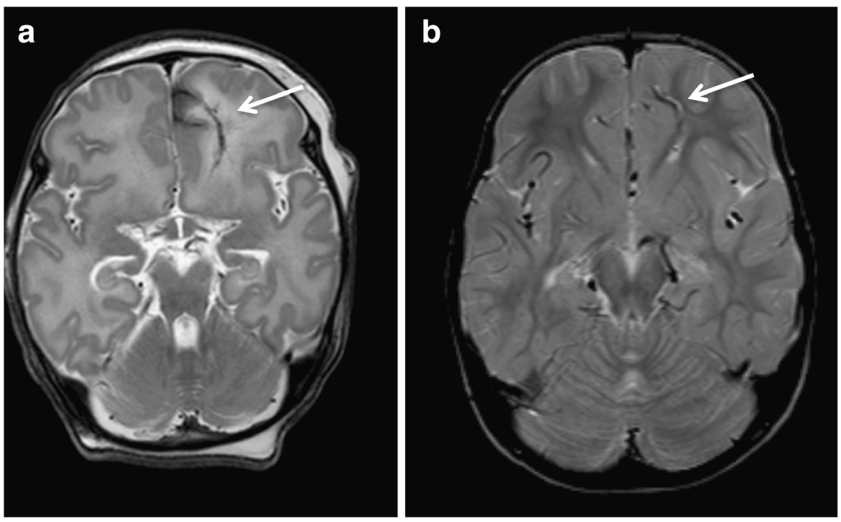

Fig. 6 Axial T2-weighted images of case 14 at the age of 4 days (**a**) and 24 months (**b**): while at the age of 4 days (**a**), the DVA presents with a prominent collector vein and high signal intensity in the surrounding parenchyma (*arrow*), the findings at the age of 24 months (**b**) are subtler

Table 3 Summary of changes over time observed on serial imaging

cUS	Appearance of the DVA/collector vein	Unchanged	Less clear/no longer detectable	Clearer/easier to detect	
		9/14	4/14	1/14	
	Echogenicity in the surrounding parenchyma	Unchanged echogenicity	Hyperechogenicity increased/developed	Hyperechogenicity decreased/no longer detectable	Cystic evolution in the adjacent haemorrhage
		6/14 (3 hyperechoic, 3 normal)	2/14	3/14	3/14
	Doppler findings in the collector vein	Unchanged	Increased	Decreased	First increased and later decreased
		2/5	2/5	0/5	1/5
MRI	Appearance of the DVA/collector vein	Unchanged	Less clear/no longer detectable	Clearer/easier to detect	
		6/7	0/7	1/7	
	Signal intensity (SI) in the surrounding parenchyma	Unchanged and normal	High SI increased/developed	High SI decreased/no longer seen	Cystic evolution in the adjacent haemorrhage
		2/7	1/7	1/7	3/7
	Diffusion-weighted imaging (DWI)	Normal on all MRIs	DWI abnormalities detected on follow up imaging		
		5/6	1/6		

Serial cUS was available in 14/14, Doppler examinations in 5/14, conventional MRI in 7/14, and DWI in 6/14 cases

Neurodevelopmental outcome

All 13 surviving infants took part in the standardized clinical follow-up program of the local university hospital. They were examined by paediatricians experienced in neurodevelopmental assessment. The age at follow up ranged between 5 months to 11 years (see Table 2). Depending on the age and the institution, follow-up protocols included the Bayley Scales for Infant Development II or III or the Griffiths Mental Developmental Scales; all infants had a formal neurological examination. Neurodevelopmental outcome was judged normal in 12 of the 13 children. One term infant who suffered from perinatal asphyxia and associated abnormalities in the basal ganglia developed dyskinetic cerebral palsy and had a developmental quotient of more than 2 standard deviations below the mean on the Griffiths Mental Development Scales (case 14). The neurodevelopmental impairment was unlikely to be related to the DVA.

Discussion

This case series underlines the fact that that the neonatal diagnosis of DVA is possible with carefully performed cUS and MRI. The paucity of neonatal reports in the literature and of the diagnosis in our own centres is striking and might suggest that a significant proportion of DVAs are missed on neonatal imaging. However, serial imaging in our cohort suggests that some DVAs undergo dynamic changes during the neonatal period and early infancy; this may contribute to why the diagnosis is rare at this age.

In 6 of our 14 cases, the DVA was diagnosed using cUS. However, while the cUS appearance of a dilated collector vein surrounded by increased echogenicity was striking in some cases (those who were diagnosed primarily on cUS), it was subtle in others and only detected on cUS retrospectively, once the diagnosis was made with MRI. In some instances, a DVA could be mistaken for a periventricular haemorrhagic infarction because it presented as a focal lesion of increased triangular shaped echogenicity adjacent to the lateral ventricle. However, its appearance and evolution were different, without atrophy, cystic breakdown or the formation of a porencephalic cyst and often with persistent rather uniform echogenicity. Some DVAs may initially resemble focal ischaemic infarction related to distal perforator arteries perfusing the periventricular white matter.

In this neonatal cohort, all DVAs were located supratentorially, as opposed to 17–29 % of DVAs being found infratentorially in adults [2, 16, 17]. This maybe due to a selection bias, as almost half of our cases were first detected during a routine cUS scan done for prematurity or growth restriction. While the supratentorial area is easily accessible for cUS, a detailed visualization of the posterior fossa can be difficult, especially in term infants and without routine use of the mastoid window for scanning. Therefore, infratentorial DVAs are more likely to be missed on cUS.

SWI has been shown to be a valuable tool for identifying cerebrovascular lesions which may not be seen using conventional MR sequences [18]. In our cohort SWI was available for analysis in eight cases. In all eight, it was found to be helpful and aided in the differential diagnosis. However, in none of the cases did the SWI add new findings that were not visible on conventional MR imaging.

Doppler US may distinguish DVA from other entities by visualizing blood flow in the collector vein, typically localized in the centre of the hyperechogenicity. However on the first cUS exam in one of our 14 infants colour Doppler US failed to detect the very slow flow in the collector vein although it was

visible on the 2D real-time image. When this infant's cUS was repeated at 1 month of age the venous flow in the collector vein had increased and was easily detected with Doppler US, but at 4 months the hyperechogenicity surrounding the DVA was barely visible and the flow in the collector vein had decreased again (Fig. 2).

While in the majority of cases infants had a venous flow profile with low velocity, four infants had arterialized flow in the collector vein. Arterialized DVAs, also referred to as atypical DVAs, mixed-type vascular malformations, arteriovenous malformations with venous predominance, or intracerebral venous angiomas with arterial blood supply [19–24], have been recently reviewed and classified into three subtypes according to the angiography findings by San Millan Ruiz [7, 25]. In our neonatal cohort, only one of the three infants with arterialized flow underwent conventional angiography. It showed a capillary stain pattern, but no arterial feeders could be detected. These findings correspond to type 1 arterialized DVA according to the classification system of San Millan Ruiz [7, 25]. Type 1 has been thought to be generally benign, while types 2 and 3 follow a more aggressive clinical course with a risk of haemorrhage similar to AVMs. Recently, Roccatagliata et al. published a case series of seven adult patients with DVAs with capillary stain on angiography, who became symptomatic (progressive neurological deficits, seizures, or cerebral haemorrhage) [26]. The neonatal case reported here (case 10) had an associated CM and haemorrhage on initial imaging, but was neurologically asymptomatic at least up to two years. Whether the CM and the haemorrhage were a consequence of the arterialized DVA remains speculative.

It is striking that we found an arterialized pattern in 29 % of our neonatal cases, while arterialized DVA is an exceedingly rare finding in adults [19–22]. One might speculate whether the typical imaging features of DVAs evolve earlier and are more pronounced in the arterialized type and are therefore more likely to be depicted during the neonatal period. Alternatively, an "arterialized phase" could be common step in the development of all DVAs, with subsequent loss of the arteriolization in the majority once they have reached a hemodynamic equilibrium. Unfortunately none of our cases underwent serial DSA imaging to either prove or disapprove this hypothesis.

Serial imaging during the neonatal period and early infancy revealed that DVA cUS and MRI findings may vary over time (in 10/14 cases, see Table 3). The extent and intensity of the hyperechogenicity/hyperintensity as well as the flow velocity measured by Doppler US in the collector vein changed remarkably during the first months after birth in some infants. The changes showed large inter-individual differences and no clear pattern could be found. These findings are intriguing as classical venous DVAs are usually considered a stable normal variation of the cerebral venous angioarchitecture of embryonic origin [1–7]. However, the potential for postnatal evolution of DVAs has already been suggested by Okudera et al. [27, 28] and is supported by the report of Leach and colleagues who were able to observe the postnatal evolution/maturation of a DVA in a young infant between 5 and 25 months of age [29].

The essential role of DVAs in venous drainage has been demonstrated by severe complications such as venous infarction and haemorrhage that have occurred when DVAs were occluded. We speculate that the observed tissue changes in the neonatal period and early infancy are related to changes in cerebral blood flow and tissue perfusion. The development of in- and outflow imbalance within the DVA and its surrounding parenchyma might also play a role.

Consistent with the changes we found in some neonates it has recently been reported in adults that DVAs may undergo dynamic changes. San Millan Ruiz et al. observed parenchymal abnormalities associated with DVAs on MR imaging [30]. They postulate that these abnormalities occur secondarily, likely during postnatal life, as a result of chronic venous hypertension. Outflow obstruction, progressive thickening of the walls of the DVA, and their morphological organization into a venous convergence zone are thought to contribute to the development of venous hypertension in the DVA [7, 30]. The possibility of postnatal changes in and around a DVA is also underlined by reports from Cakirer and Nussbaum et al. on patients with DVA, in whom a de novo formation of an adjacent CM [31] and several AVMs close to and draining into a large DVA [32] were observed. Subclinical microhaemorrhages in the form of haemosiderin-laden macrophages surrounding a DVA have been reported by San Millan Ruiz et al. [7] supporting the hypothesis of blood diapedesis through the walls of the venous radicles or rupture of one of the radicles may occur and play a role in the evolution of CMs associated with DVAs. Haemosiderin and microhaemorrhages might contribute to the echogenicity seen on cUS in infants with DVA, but we did not find supporting evidence for this on MRI in any of the infants. An imbalance between the in- and the outflow to and from the DVA was found to be the leading pathogenic mechanism in rare cases where DVAs become symptomatic [12]. It is interesting that the neonate with an associated CM, capillary stain on angiography, and haemorrhage showed abnormal MR diffusivity in a large area surrounding the DVA on follow-up imaging at 3 months. Despite these striking diffusion abnormalities, the infant did not have any clinical symptoms at the time of scan or on clinical follow-up to 2 years of age. To the best of our knowledge, persisting MR diffusion abnormalities surrounding DVAs have not been reported so far.

One might hypothesize that the above mentioned evolving pathogenic mechanisms contribute to DVA being rarely diagnosed neonatally or in early infancy despite the fact that many neonates have detailed and repeated cUS and also MR brain

imaging. However, DVA might be structurally present at birth but it may not be easily detectable because the characteristic imaging findings are yet to evolve.

Intrauterine growth restriction (IUGR) was present in 6 infants in our cohort. In the pathogenesis of both IUGR and the development of DVA, prothrombotic factors, intrauterine vascular occlusion and disturbed angiogenesis are currently discussed [33–36]. However, we can only speculate whether these two entities are linked or whether IUGR itself being an indication for neonatal imaging may have biased this finding.

In conclusion, this report is the first case series describing cUS imaging, Doppler US, and MRI findings of DVA in neonates. It illustrates a diverse spectrum of cUS and MRI findings of DVA in the newborn brain. Serial imaging suggests that some DVAs may undergo dynamic changes, evolution, and regression in the neonatal period and early infancy. Now that large cohorts of preterm infants are scanned routinely with MRI at term equivalent age and some again at school age, we should be able to learn more about the neonatal incidence and evolution of this embryonic vascular variant.

Ethical standards and patient consent The authors declare that all human and animal studies have been approved by the local ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. The authors declare that all patients gave informed consent prior to inclusion in this study.

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

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