

In utero magnetic resonance imaging for diagnosis of dural venous sinus ectasia with thrombosis in the fetus

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Abstract

Background Dural venous sinus ectasia with thrombosis (DVSET) in the fetus is a rare condition that can be diagnosed prenatally with the use of fetal MR imaging, yet with limited indication of long-term clinical significance.

Objective To describe and evaluate the diagnostic value of fetal MR imaging in the prenatal diagnosis of dural venous sinus ectasia with thrombosis and its clinical significance.

Materials and methods We report a series of nine fetuses with dural venous sinus ectasia with thrombosis. The mothers, located in four fetomaternal centres, were referred for fetal MR imaging due to space occupying lesions identified on second-trimester antenatal ultrasound.

Results In all but one case the dural venous sinus ectasia with thrombosis was in the vicinity of the venous confluence (VC) with various extension in the posterior dural

sinuses. Antenatal follow-up imaging was performed in seven cases and showed progression in one, stable appearances in one and regression in five cases. Three pregnancies were terminated. In the remaining six cases there was no reported neurological deficit at up to 44 months of clinical follow-up.

Conclusion This is among the largest series of postnatal clinical follow-up in cases of prenatal diagnosis of dural venous sinus ectasia with thrombosis in the literature. Clinical follow-up suggests a good prognosis when antenatal follow-up shows partial or complete thrombus resolution.

Keywords MRI · Fetus · In utero · Dural venous sinus thrombosis

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Introduction

Dural venous sinus ectasia with thrombosis is a potentially life-threatening condition because of the high risk of venous ischaemia and haemorrhage. It can occur in the fetus and can be detected by MRI, although only approximately 50 cases had been described in the world literature at the time of this report [1–18].

Knowledge about the cause and long-term clinical significance of dural venous sinus ectasia with thrombosis in the fetus is limited. Most cases diagnosed on prenatal imaging involved the venous confluence (also known as the torcular Herophili) with variable extension into the adjacent dural sinuses. Because most venous drainage of the brain normally passes through that structure, fetal dural venous sinus ectasia with thrombosis has been associated with a poor outcome and this has been reported in some cases [2, 5, 9, 13]. There are, however, several reports of spontaneous resolution and normal clinical outcomes [1, 3, 7, 8, 10, 11, 14–16, 18].

We report nine cases of fetal dural venous sinus ectasia with thrombosis and describe the fetal MR features and outcomes of the pregnancies.

Materials and methods

The fetuses in the study were located from the records of four tertiary feto-maternal units in the UK and fetal MR imaging was performed at three of those centres. At two sites (Southampton and Cardiff) all of the cases were performed as clinical studies and written consent to be included in this report was obtained retrospectively. The other fetal MR examinations were performed at Sheffield either as part of an on-going research study or as a clinical study. Full written consent was obtained prospectively in the research cases under the guidance of the South Sheffield Research Ethics Committee. The clinical cases at Sheffield reported here were included after relevant review and approval was obtained from the Institutional Clinical Effectiveness Unit and Research Department of the institution. Two cases were excluded on the basis of their autopsy results and absence of comments on the space-occupying cystic lesion. Initial US examinations were performed in the tertiary referral centre as part of routine second-trimester screening. In all cases the reporting radiologist was aware of the US report or previous fetal MR imaging.

In all cases fetal MR was performed at 1.5-T MR magnets using different protocols across the three scanning centres but these always included T2-W single-shot fast spin-echo (SSFSE) sequences and some form of ultrafast T1-W sequence. An echoplanar imaging diffusion-weighted sequence was not routinely performed.

Results

Table 1 summarizes the clinical and MR imaging characteristics of our cases.

The evolution of the thrombus was variable. In four cases ($n=4$, 44.4%) there was complete prenatal resolution as follows:

Case 1. Dural venous sinus ectasia with thrombosis was identified first on US at 21 weeks' gestational age. The thrombus measured 1.5 cm in maximum linear diameter on fetal MR at 22 weeks' gestational age and had completely resolved by 30 weeks.

Case 2. Dural venous sinus ectasia with thrombosis was identified first on US at 21 weeks' gestational age when the thrombus measured 2.9 cm in maximum linear diameter. Fetal MR at 22 and 32 weeks showed an incremental decrease in size, with complete resolution on US by 34 weeks' gestational age.

Case 3. Dural venous sinus ectasia with thrombosis was identified first on US at 21 weeks' gestational age. The thrombus measured 1.7 cm in maximum linear diameter and had completely resolved on imaging by 28 weeks.

Case 4. Dural venous sinus ectasia with thrombosis was identified first on US at 18 weeks' gestational age. US at 24 weeks showed a reduction in size and low velocity flow on colour Doppler (Fig. 1). Fetal MRI performed at 24 weeks showed a decrease in size, with complete resolution on US at 32 weeks' gestational age (Fig. 1).

In one case ($n=1$, 11.1%) there was partial antenatal resolution with subsequent postnatal incomplete resolution:

Case 5. Dural venous sinus ectasia with thrombosis was identified first on US at 21 weeks' gestational age and on fetal MR at 23 weeks (Fig. 2). The thrombus was increasing in size and at 30 weeks measured 4.6 cm in maximum linear diameter. At 34 weeks the lesion had decreased in size and at 35 weeks' gestational age measured 2.8 cm in maximum linear diameter. Postnatal MR imaging on day 2 after birth showed further reduction in size, with near-complete resolution at 3 months (Fig. 2).

In one case ($n=1$, 11.1%) the volume of the thrombus remained stable antenatally with subsequent postnatal partial resolution:

Case 6. Dural venous sinus ectasia with thrombosis was identified first on US at 20 weeks' gestational age when the thrombus measured 3.5 cm in maximum linear diameter. Fetal MR imaging at 32 weeks showed stable appearances of the lesion in terms of size. MR imaging at 6 months of age showed a partially thrombosed dural sinus fistula and normal brain.

In three cases ($n=3$, 33.3%) the dural venous sinus ectasia with thrombosis was complicated with significant mass effect also commonly associated with obstructive hydrocephalus, and the pregnancies were terminated:

Case 7. Dural venous sinus ectasia with thrombosis was identified first on US at 19 weeks' gestational age. The thrombus measured 4.8 cm in maximum linear diameter on fetal MRI at 20 weeks (Fig. 3). All features were in status quo at 21 weeks' gestational age; however at 22-weeks the thrombus had increased in size (Fig. 3). Autopsy showed a blood-filled cystic space representing the posterior wall of the superior sagittal sinus. This was in continuity with an expanded and distended transverse sinus. Within the cyst were a blood-coloured and a white nubbins of tissue. The dark red thrombotic nodular tissue was confirmed as fresh thrombus with some fibrin strands.

Table 1 Characteristics, imaging findings and outcomes of each case

Case	Maternal history	Location of thrombosis	Extension	Signal intensity (SI) (SSFSE T2-W and T1-W)	Brain appearance	Outcome of pregnancy	Postnatal imaging or fetopathological correlation	Postnatal follow-up
1	25-year-old Gravida 1 para 0 G1 P0 Coagulation/ thrombophilia and TORCH: Negative or normal	Vicinity of the straight sinus	Venous confluence	High on T1-W in the centre Heterogeneous SI and susceptibility artefact in the periphery on T2-W	Normal brain Mild mass effect but no structural abnormality No evidence of dural sinus fistula	Delivery at 39 weeks* GA Male	MR at 3 months: normal	Normal development at 3 months
2	27-year-old G1 P0 Thrombophilia, Neonatal alloimmune thrombocytopenia-negative (NAIT) and TORCH: normal or negative	Venous confluence	Straight, transverse and sagittal sinuses	Intermediate on T2-W	Mild mass effect with displacement of the posterior cerebral hemispheres No structural abnormality	Delivery at 39 weeks* GA Male	MR at 26 months: normal	Normal development at 26 months
3	38-year-old G4 P3	Venous confluence	Right transverse sinus	High on T1-W and intermediate to low on T2-W	No structural abnormality	Delivery at 41 weeks* GA Male	Cranial US at 1 month: normal	Normal neonatal period
4	40-year-old G2 P0+1	Venous confluence	Superior sagittal sinus	High on T1-W and low on T2-W	No structural abnormality	Delivery at 39 weeks* GA Female	N/A	Normal development at 36 months
5	23-year-old G2 P1	Venous confluence	Superior sagittal sinus	High on T1-W and low on T2-W with a focus of very low SI	Displacement of the posterior cerebral hemispheres but no structural abnormality	Delivery at 37 weeks* GA Female	MR at 3 months: Near-complete resolution	Normal development at 44 months
6	32-year-old G2 P1	Venous confluence	N/A	Intermediate on T2-W with a focus of high on T1-W and very low on T2-W	Displacement of the posterior cerebral hemispheres but no structural abnormality	Delivery at 38 weeks* GA Female	MR at 6 months: partly thrombosed dural sinus fistula	Normal neonatal period
7	26-year-old G2 P1 TORCH: Immunity to parvo virus and rubella. Negative for cytomegalovirus and toxoplasma	Venous confluence	Superior and inferior sagittal sinuses	Intermediate on T2-W with a focus of high on T1-W and very low on T2-W SI	Mass effect on brainstem No structural abnormality	TOP at 23 weeks* GA Male	DVSET on autopsy	N/A

Table 1 (continued)

Case	Maternal history	Location of thrombosis	Extension	Signal intensity (SI) (SSFSE T2-W and T1-W)	Brain appearance	Outcome of pregnancy	Postnatal imaging or fetopathological correlation	Postnatal follow-up
8	25-year-old Mother was Rh- and NAIT-negative	Venous confluence	Superior sagittal, transverse and sigmoid sinuses	Intermediate on T2-W with a central focus of high SI on T1-W and very low on T2-W	Mild ventriculomegaly, brainstem kinking and volume loss of posterior cerebrum	TOP at 21 weeks, GA Male	Autopsy not performed	N/A
9	20-year-old G3 P0+2 Normal thrombophilia	Venous confluence	Right transverse and sigmoid sinuses	Iso-intense to grey matter	Significant mass effect and hydrocephalus	TOP at 22 weeks, GA	Autopsy not performed	N/A

GA gestational age, NAIT neonatal alloimmune thrombocytopenia, TOP termination of pregnancy, TORCH toxoplasmosis, other infections, rubella, cytomegalovirus infection and herpes simplex, DVSET dural venous sinus ectasia with thrombosis, Gx Py gravida para

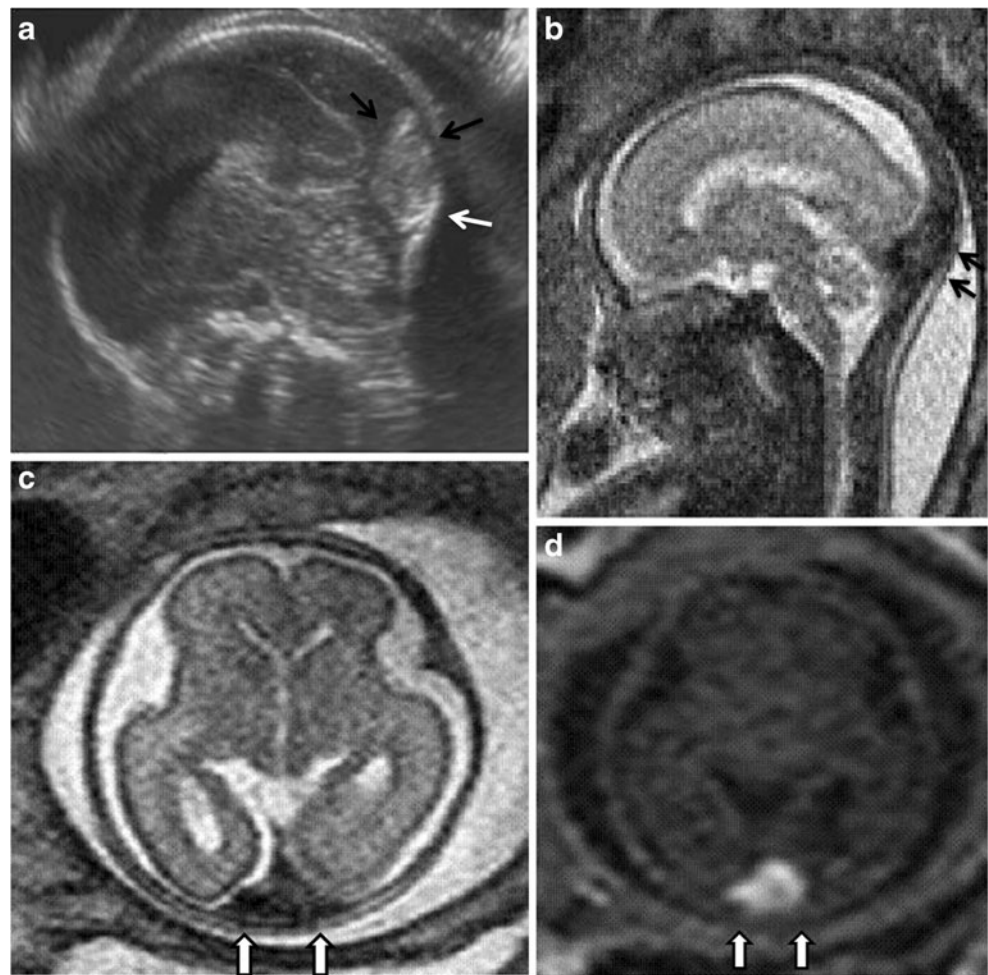
The white nubbin of tissue showed amorphous pale degenerate tissue, predominantly fibrin consistent with old thrombus. Focally calcification was seen, consistent with old thrombus. The appearances of the cyst wall was compatible with the macroscopic findings of cystic degeneration of the wall of the dural venous sinus with thrombosis. Sectioning of the brain showed no gross focal abnormality. Case 8. Dural venous sinus ectasia with thrombosis was identified first on US at 21 weeks' gestational age. The size of the thrombus remained stable between the initial US and fetal MR and was associated with significant mass effect. There was no other antenatal follow-up imaging. No autopsy was performed.

Case 9. Dural venous sinus ectasia with thrombosis was identified first on US at 20 weeks' gestational age. The size of the thrombus on fetal MR at 21 weeks measured 4.6 cm in maximum linear diameter. There was associated significant mass effect and compression upon the adjacent brain parenchyma and brainstem and obstructive hydrocephalus. There was no other antenatal follow-up imaging. No autopsy was performed.

Discussion

The nomenclature of the pathology described in this case series is not straightforward. Authors describing the same disease process have used a range of terms including dural sinus malformation (with thrombosis), dural venous sinus thrombosis and dural venous sinus ectasia (with thrombosis). Dural sinus malformations can be divided into midline with giant pouches, often associated with arteriovenous shunting within the wall of malformed sinus, and lateral involving the jugular bulb [12, 18]. Proponents of “dural sinus malformation” usually indicate the presence of dural fistulae (direct communications between arteries and a dural venous sinus) as the cause of the enlargement of the vessel and secondarily causing thrombosis. We were not able to show any in utero evidence of fistula formation in our cases of enlarged thrombosed structures in the vicinity of the venous confluence. Fistula might be difficult or impossible to confirm or refute on fetal imaging at present, hence we elected not to use the term “dural sinus malformation”. In contrast, “dural venous sinus thrombosis” does not emphasize the marked enlargement of the vessel that characterises our cases. It is known that a sign of sinus thrombosis postnatally is enlargement of the vessel by clot expansion but this is never to the degree seen in the fetuses reported here and in the literature, perhaps reflecting the different pathophysiological mechanism. The situation is complicated further by the fact that fistulae can arise as a result of sinus thrombosis and vice versa. We chose to use the term “dural venous sinus ectasia with thrombosis” because it is the best descriptor of the pathology in the absence of known predisposing factors or

Fig. 1 Case 4: Dural venous sinus ectasia with thrombosis with complete prenatal resolution. **a** US at 24 weeks' gestational age. Sagittal image shows a well-defined echogenic lesion above the cerebellum in the vicinity of the venous confluence (*arrow*) in continuation with the superior sagittal sinus (*arrowheads*). **b** Fetal MR imaging at 24 weeks' gestational age. Sagittal T2-W image shows an abnormal triangular structure in the midline in the vicinity of the venous confluence, continuous with the superior sagittal sinus, which returns low signal intensity (*black arrows*). **c** Corresponding axial T2-W MR image at 24 weeks shows the low signal intensity lesion in the vicinity of the venous confluence (*white arrows*). **d** Axial T1-W MR image at 24 weeks shows the lesion with high signal intensity (*white arrows*)



causes in our cases. Possible mechanisms have been proposed [1, 4, 5, 18], but in most cases the aetiology is unknown and this is true for all of our cases.

Embryologically the development of the dural venous sinuses is closely related to the development of the dura mater, a detailed description of which is beyond the scope of this paper but the interested reader is directed to [19–21]. In short the galenic and dural venous systems start to develop during the third gestational month. The internal cerebral vein, posterior thalamic veins and basal veins unite to form the vein of Galen, which in turn drains into the straight sinus. The superior sagittal sinus is formed from the fusion of the bilateral marginal sinuses, although they initially remain unfused posteriorly and so drain into the transverse sinuses on each side separately. During the sixth fetal month the two limbs unite to form the venous confluence. There are many anatomical variations in the structure of the venous confluence and most can be explained by the presence of interconnecting vascular channels between the two limbs of the posterior part of the superior sagittal sinus in the fetus. At roughly the same time and until the sixth fetal month, the transverse sinuses enlarge from a lateral to medial direction followed by a subsequent decrease in size. In a similar

fashion the occipital sinuses, originating from the primitive torcular plexus, enlarge rapidly between 4 months' and 5 months' gestational age before reducing in size again [21].

Despite knowledge of the development of the dural sinuses, the natural history, triggering event and pathophysiology of the dural venous sinus ectasia with thrombosis is unclear. One hypothesis suggests that the persistence of the developmental ballooning of the dural sinuses at 4–6 months' gestational age persists and causes venous hypertension and secondary dural fistulae formation [1, 2, 4, 7, 18], which is likely to contribute to the dural venous malformation. Another proposed mechanism is related to excessive and disorganized dural sinus development (without fistulae) during the transition period when the dilated sinuses should decrease in size [1]. Local hypothetical mechanisms that could lead to thrombosis include immaturity of the sinus, altered flow and modification of the endothelial wall [1], the last being consistent with the findings of the autopsy in our seventh case. The dural venous sinus ectasia with or without identifiable thrombus can regress spontaneously. The wide variation in the anatomical arrangement around the venous confluence [22] could explain the variable outcome in cases of thrombosis by providing potential anastomotic channels to redirect the venous drainage of the prosencephalic

Fig. 2 Case 5: Dural venous sinus ectasia with thrombosis with partial antenatal resolution and incomplete postnatal resolution. **a** Fetal MR imaging at 23 weeks' gestational age. Sagittal T2-W image shows a well-defined lesion with low signal intensity. The lesion is in the vicinity of the venous confluence, continuous with the superior sagittal sinus (grey arrows), with a focus of very low signal intensity (black arrows) at its inferior aspect likely representing blood products of a different age. **b** Postnatal MR imaging on day 2 after birth. Sagittal T1-W image shows reduction in the size of the thrombus, which returns high signal intensity (white arrows). **c** Corresponding axial T2-W MR image on day 2 shows reduction in the size of the thrombus, which returns high signal intensity (white arrows) on both T2-W and T1-W sequences. **d** Postnatal MR imaging at 3 months. Axial T2-W image shows a small residual thrombus in the posterior inferior part of the superior sagittal sinus (white arrows)

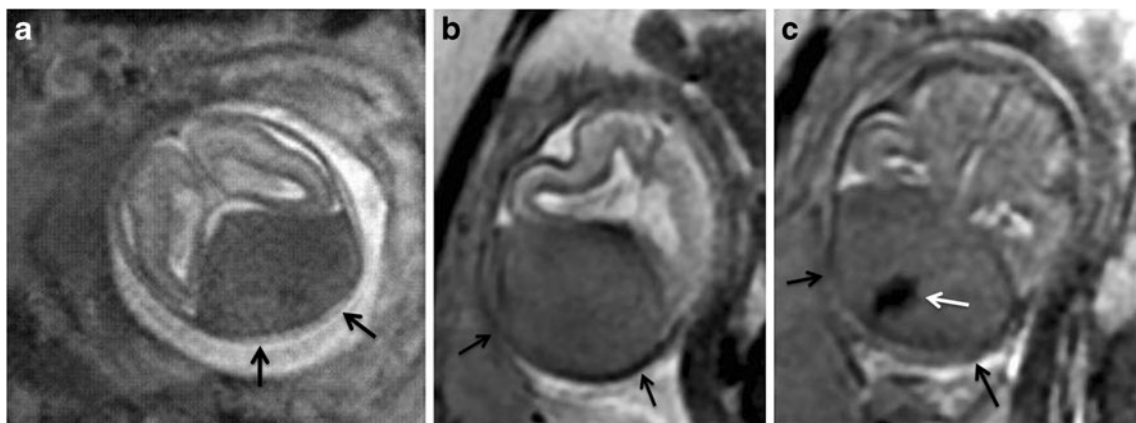
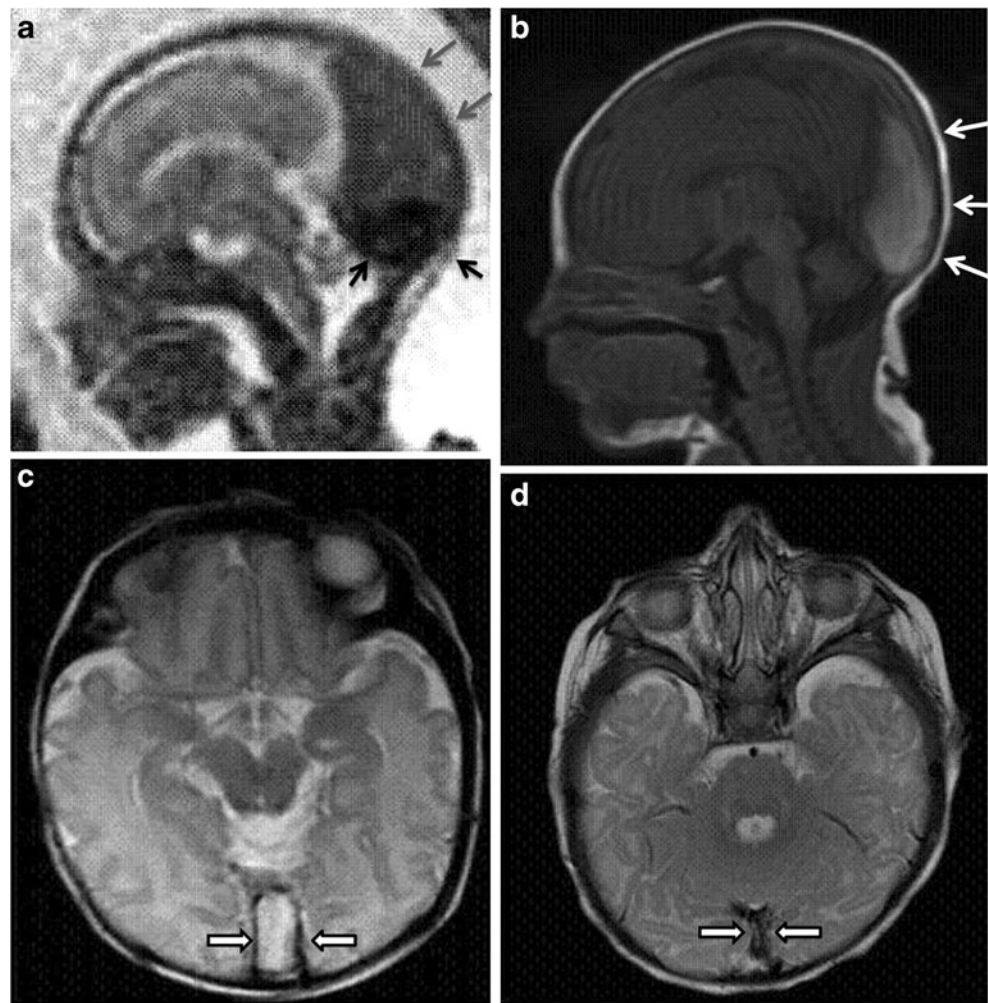


Fig. 3 Case 7: Dural venous sinus ectasia with thrombosis with prenatal increase in the size of the thrombus associated with significant mass effect. **a** Fetal MR at 20 weeks' gestational age. Axial T2-W image shows a well-defined lesion with intermediate signal intensity. The lesion is occupying the posterior half of the cranial cavity (black arrows), with underlying mass effect and displacement of the cerebral

hemispheres. **b** Fetal MR at 22 weeks' gestational age. Axial T2-W image shows increase in the size of the thrombus (black arrows) and more mass effect on the cerebral hemispheres. **c** Corresponding axial T2-W MR image at a lower level (at 22 weeks) shows a focus of very low signal intensity (white arrow) at the posterior aspect of thrombus (black arrows)

structures [16], thereby protecting the underlying brain parenchyma during the regression of the pathology without underlying brain injury and leading to a normal outcome.

In our cases the typical finding on US, which could be detected as early as 18 weeks' gestational age, was a large predominantly hypoechoic mass between the posterior portions of the cerebral hemispheres and – as with many cases in the reported literature – a range of incorrect diagnoses were suggested, such as tumours, cysts and Dandy-Walker malformation. In 3/9 of our cases US suggested the presence of thrombosis (cases 3–5) and in two others intracranial haemorrhage was described (cases 1 and 8). In the other four, however, a vascular cause was not suspected on US. The fetal MR imaging features in eight of our cases were similar to those described in the literature, namely a well-defined rounded or triangular extra-axial lesion that could be localised to the venous confluence with variable extension into the adjacent dural sinuses in the majority of the cases. In our series one fetus had thrombosis in the straight sinus only (case 1). In all cases there was mass effect and displacement of the adjacent brain but no associated parenchymal complication or any other focal abnormalities. The signal characteristics of the lesions are in keeping with those described in the literature [1–18]. As Byrd et al. [18] described, the lesion commonly returned intermediate signal with central areas of very low signal intensity on T2-W sequences and high on T1-W sequences. Correlating the imaging findings with the autopsy results in case 7, this likely reflects different ages of the thrombus. When diffusion-weighted imaging was performed, it did not demonstrate associated parenchymal ischaemic changes. Contrary to the finding of migrational disorder in one of the cases of Byrd et al. [18] series, in our cases the underlying parenchyma was structurally normal except for the mass effect associated in some cases with ventriculomegaly (cases 8 and 9). Follow-up imaging after the first fetal MR examination was performed in seven cases using US with or without repeat fetal MR.

The relatively small number of cases of dural venous sinus ectasia with thrombosis with clinical follow-up in the literature shows that a good neurological outcome can be expected in up to 70% of cases [1]. Moreover the involvement of the venous confluence does not appear to be related to poor outcome as reported in previous series [2]. Six deaths have been reported involving the venous confluence [1–3, 5, 9], one of which was peri-operative [5]; in two cases there was mild [6] and in one case significant [18] developmental delay and in one case a severe neurological deficit was reported [2].

US imaging is the principal modality for screening for fetal brain abnormalities and is also used as the first line of more detailed assessment by fetomaternal experts if a brain problem is suspected on screening. It appears that fetal MR is clinically useful in the diagnosis of dural venous sinus ectasia with thrombosis primarily because of higher anatomical delineation of the

exact location of the mass and its relationship to the venous dural sinus system and its ability to accurately characterise acute and subacute thrombus. It is possible, however, that awareness of dural venous sinus ectasia with thrombosis is limited among many fetomaternal experts and consideration of this pathology is important when a posterior mass lesion is shown. The use of colour Doppler studies can be of great assistance in assessing the dural system and can show lack of blood flow [5]; however colour Doppler might not demonstrate an underlying dural arteriovenous fistula. The evolving literature suggests that most cases of dural venous sinus ectasia with thrombosis resolve and are not associated with long-term disability, although longer follow-up periods in more children are required. Generally it is difficult to find features on antenatal imaging that correspond to good or poor outcomes. As described above, the potential for brain injury and poor clinical outcome might be related to the degree of venous or dural sinus flow in anastomoses around the thrombus resulting in cerebral ischaemia or haemorrhage or other associated complications. Unfortunately antenatal imaging methods available at present cannot do this reliably. In our cases reduction in the size of the lesion occurred as late as 34 weeks' gestational age (case 5). Hence we suggest serial antenatal imaging follow-up with either US or fetal MR at regular time intervals (e.g., 4 weeks) to monitor evolution of the lesion (changes in the size or signal characteristics), underlying brain parenchyma and ventricular size changes. When fetal MRI is used we suggest single-shot fast spin-echo (SSFSE) T2-weighted sequences in the three orthogonal planes (axial, sagittal and coronal), ultrafast T1-W sequences and echoplanar imaging diffusion-weighted sequences.

Conclusion

Fetal MR imaging appears to be clinically useful in the diagnosis of dural venous sinus ectasia with thrombosis by demonstrating the location of the lesion and signal characteristics of the thrombus. The evolving literature suggests that most cases of dural venous sinus ectasia with thrombosis resolve and are not associated with long-term disability, although longer follow-up periods in more children are required to determine features on antenatal imaging that correspond to either good or poor outcomes.

Conflicts of interest None.

References

1. Merzoug V, Flunker S, Drissi C et al (2008) Dural sinus malformation (DSM) in fetuses. Diagnostic value of prenatal MRI and follow up. *Eur Radiol* 18:692–699

2. Barbosa M, Mahadevan J, Weon YC et al (2003) Dural sinus malformations (DSM) with giant lakes, in neonates and infants. Review of 30 consecutive cases. *Intervent Neuroradiol* 9:407–424
3. Laurichesse Delmas H, Winer N, Gallot D et al (2008) Prenatal diagnosis of thrombosis of the dural sinuses: report of six cases, review of the literature and suggested management. *Ultrasound Obstet Gynecol* 32:188–198
4. McInnes M, Fong K, Grin A et al (2009) Malformations of the fetal dural sinuses. *Can J Neurol Sci* 36:72–77
5. Visentin A, Falco P, Pilu G et al (2001) Prenatal diagnosis of thrombosis of the dural sinuses with real-time color Doppler ultrasound. *Ultrasound Obstet Gynecol* 17:322–325
6. Komiyama M, Ishiguro T, Kitano S et al (2004) Serial antenatal sonographic observation of cerebral dural sinus malformation. *AJNR Am J Neuroradiol* 25:1446–1448
7. Legendre G, Picone O, Levallant JM et al (2009) Prenatal diagnosis of a spontaneous dural sinus thrombosis. *Prenat Diagn* 29:808–813
8. Grigoriadis S, Cohen JE, Gomori JM (2008) Prenatal thrombosis of torcular Herophili with spontaneous resolution and normal outcome. *J Neuroimaging* 18:177–179
9. Schwartz N, Monteagudo A, Bornstein E et al (2008) Thrombosis of an ectatic torcular Herophili: anatomic localization using fetal neurosonography. *J Ultrasound Med* 27:989–992
10. Spampinato MV, Hardin V, Davis M et al (2008) Thrombosed fetal dural sinus malformation diagnosed with magnetic resonance imaging. *Obstet Gynecol* 111:569–572
11. Jung E, Won HS, Kim SK et al (2006) Spontaneous resolution of prenatally diagnosed dural sinus thrombosis: a case report. *Ultrasound Obstet Gynecol* 27:562–565
12. Rossi A, De Biasio P, Scarso E et al (2006) Prenatal MR imaging of dural sinus malformation: a case report. *Prenat Diagn* 26:11–16
13. Breyssem L, Witters I, Spitz B et al (2006) Fetal magnetic resonance imaging of an intracranial venous thrombosis. *Fetal Diagn Ther* 21:13–17
14. Clode N, Cardoso C, Tavares J et al (2004) Prenatal diagnosis of thrombosis of dural sinuses. *Ultrasound Obstet Gynecol* 24:330
15. Emamian SA, Bulas DI, Vezina GL et al (2002) Fetal MRI evaluation of an intracranial mass: in utero evolution of hemorrhage. *Pediatr Radiol* 32:593–597
16. Gicquel JM, Potier A, Sitrik S et al (2000) Normal outcome after prenatal diagnosis of thrombosis of the torcular Herophili. *Prenat Diagn* 20:824–827
17. Zerah B, Zerah M, Swift D et al (2010) Giant dural venous sinus ectasia in neonates. *J Neurosurg Pediatr* 5:523–528
18. Byrd SE, Abramowicz JS, Kent P et al (2012) Fetal MR imaging of posterior intracranial dural sinus thrombosis: a report of three cases with variable outcomes. *Pediatr Radiol* 42:536–543
19. Padget DH (1955) The cranial venous system in man in reference to development, adult configuration and relation to the arteries. *J Neurosurg* 12:307–355
20. Raybaud CA, Strother CM, Hald JK (1989) Aneurysms of the vein of Galen: embryonic considerations and anatomical features relating to the pathogenesis of the malformation. *Neuroradiology* 31:109–128
21. Okudera T, Huang YP, Ohta T et al (1994) Development of posterior fossa dural sinuses, emissary veins, and jugular bulb: morphological and radiologic study. *AJNR Am J Neuroradiol* 15:1871–1883
22. Widjaja E, Griffiths PD (2004) Intracranial MR venography in children: normal anatomy and variations. *AJNR Am J Neuroradiol* 25:1557–1562

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