Supplementary Figure 1.

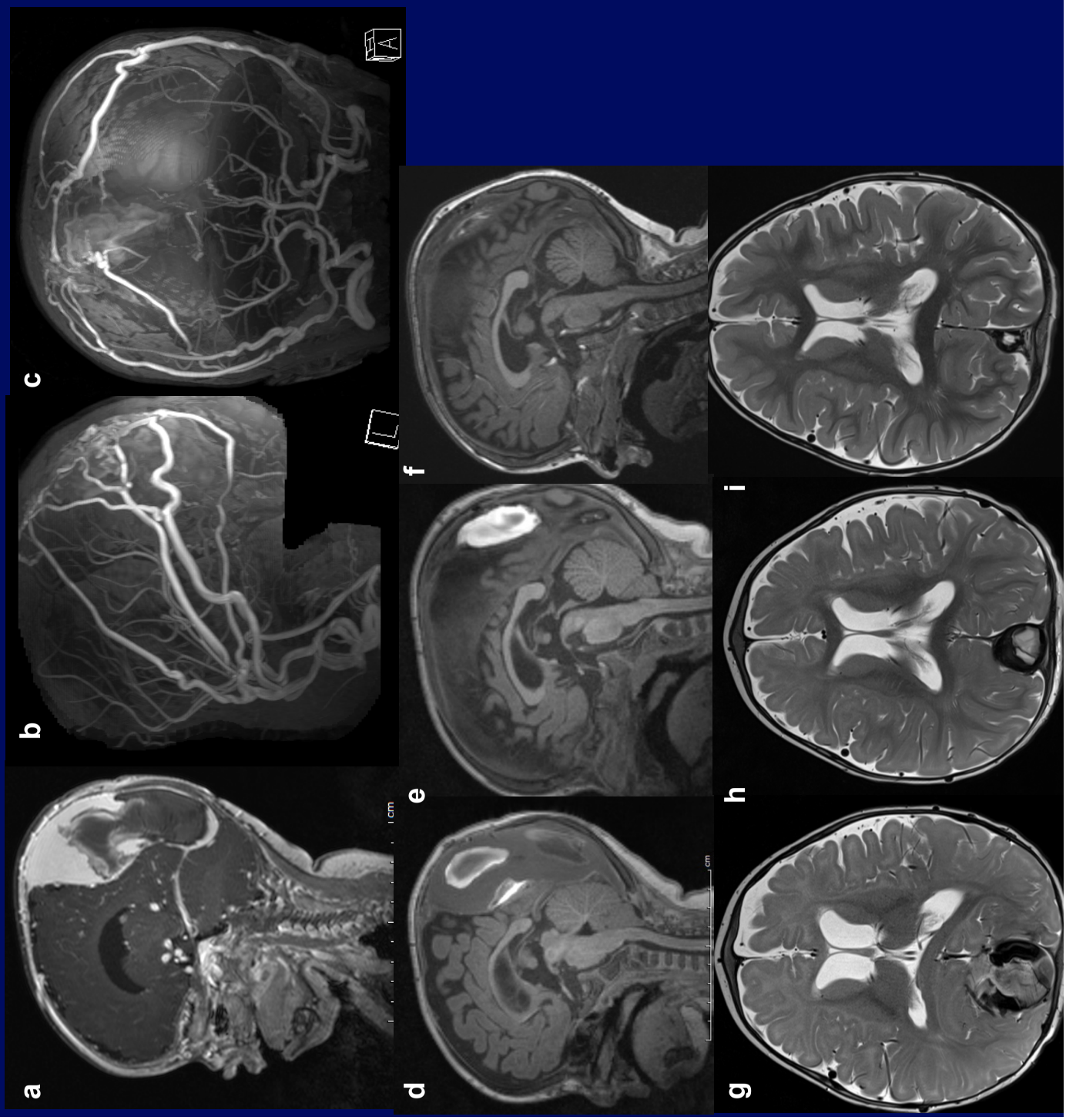
Torcular DSM with underlying arteriovenous fistula detected *in utero* (case 1). Imaging obtained at 33 weeks (a-e), 37 weeks (f), first day of life (g-k), 3 months of age (l-p), and eight months (q-r). Transverse gray scale (a) and color/M-mode Doppler (b, c) at 33 weeks demonstrates florid arterialization of the torcular an internal area of echogenicity consistent with non-occlusive thrombus. Fetal MRI at 33 weeks (d, e) demonstrates a moderate tDSM on axial HASTE imaging (d) and enlargement of the middle meningeal arteries terminating at the tDSM on VIBE T1 weighted imaging (shown as a MIP, e). The tDSM slowly enlarged without loss of arterialization or worsening thrombosis through 37 weeks gestation as shown in a representative axial HASTE image from 37 weeks (f). Axial T2 (g, h, l, m), SWI (i, n), MRA transverse MIP (j, i), and sagittal MRV MIP (k, p) from day of life 1 (g-k) and two month follow-up (l-p) MRI/MRA/MRV. Both studies demonstrate presence of enlarged MMA (green arrows) as well as occipital artery (red arrows) feeders to the tDSM on MRA. Although there was evidence of some spontaneous regression of the tDSM between day of life 1 and 2-month follow-up MRI and MRA, as well as some mineralized mural material suggestive of scar (yellow arrows, i, n), the onset of increased ventricular and subarachnoid space prominence at two months (l, m) prompted embolization of the middle meningeal artery feeders. An eight month follow-up (q-r) demonstrated regression of the tDSM and ventriculomegaly with normal brain myelination (q) though some arterial feeders persisted (r). NOTE: Supplementary Figures 1d, 1e, and 1j correspond to Figures 1a, 1b, and 1c.

Supplementary Figure 1

Supplementary Figure 2.

Postnatal presentation of thrombosed tDSM with associated arteriovenous fistula (case 10). After presentation with vomiting/lethargy at 5 months, postcontrast sagittal T1 MPRAGE (a) imaging demonstrated subtotal thrombosis of the superior sagittal sinus and torcular, and sagittal/coronal MIPs of MRA data (b, c) demonstrated enlarged middle meningeal arteries which emptied directly into the tDSM in keeping with a dAVF. The patient was taken for embolization and started on an anticoagulation regimen. There was no angiographic residual at 3 month follow-up catheter angiogram. Sagittal T1 (d, e, f) and axial T2 (g, h, i) images from the presentation at age 5 months (d, g), at 9 months of age (e, h), and at 21 months of age (f, i) demonstrate progressive involution of the tDSM and clot as well as decreased ventricular/subarachnoid prominence. Note that the clot appears sequestered from the scarred vessel lumen as the tDSM clot contracts (g-i). Although the superior sagittal sinus has remained occluded following treatment, there has been development of drainage through right cavernous capture (not shown).

Supplementary Figure 2



Supplementary Figure 3.

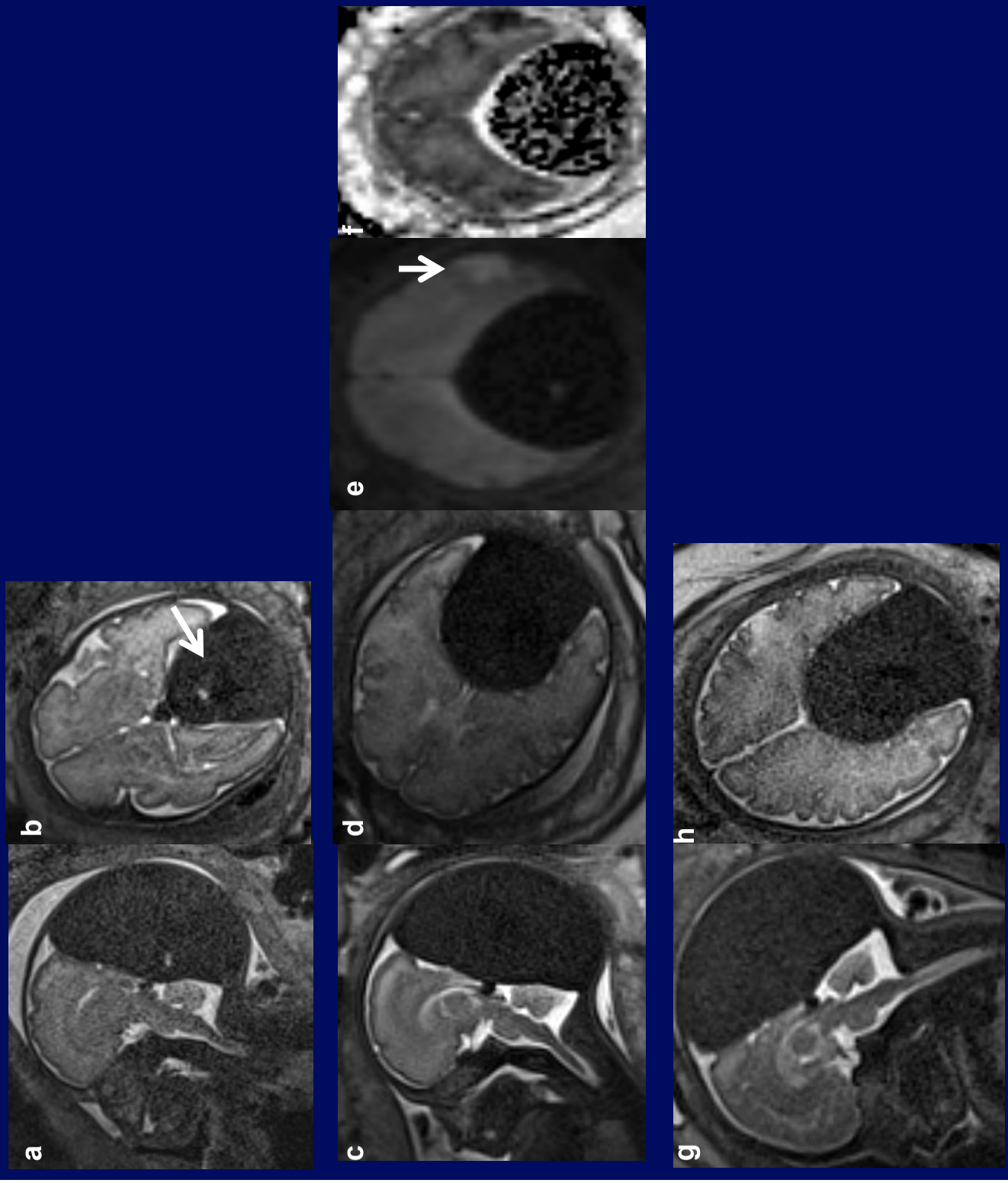
Prenatally diagnosed tDSM with spontaneous involution of thrombosis (case 4). Fetal color Doppler ultrasound (a) and sagittal/axial HASTE (b, c) at 28 weeks gestational age demonstrates an enlarged torcular complex consistent with a tDSM. Note the two-tone thrombus within the torcular, having echogenic/non-echogenic as well as T2 hyperintense/hypointense characteristics. Follow-up fetal imaging at 31 weeks GA (d-f) and 35 week GA (g-i) demonstrated a decrease in overall size of the tDSM; sagittal HASTE (d, g), sagittal T1 (e, h), and axial HASTE (f, i). However, the size of the clot and the percentage of the tDSM filled by clot increased. Postnatal sagittal T1 (j, m), axial T1 (k, n), and sagittal MIP MRV data (l, o) at 3 weeks (j, k, l) and 8 months (m, n, o) of age demonstrated progressive incorporation of the clot into the wall of the dural sinus, with T1 shortening suggestive of mineralization (n), and dural sinus recanalization by MRV. NOTE: Supplementary Figures 3b, 3h, 3m, and 3n correspond to Figures 2a, 2b, 2c, and 2d.

Supplementary Figure 3

Supplementary Figure 4.

Prenatally diagnosed tDSM complicated by (venous) infarction (case 2). Fetal MRI from 30 weeks (a, b), 32 weeks (c-f), and 33 weeks (g, h) demonstrate a large tDSM with nonocclusive thrombus. While large, initial sagittal and axial HASTE images at 30 weeks (a, b) demonstrate no signs of hydrocephalus or parenchymal injury. However, the 32 week study demonstrated enlargement in the size of the tDSM and cytotoxic edema in the left parietal lobe on diffusion trace (e) and ADC (f) images. Follow-up imaging at 33 weeks demonstrated evolution to encephalomalacia at the site of injury detected at 32 weeks on axial HASTE (h).

Supplementary Figure 4



Supplementary Figure 5.

Favorable evolution of tDSM. Case 5, as imaged with axial HASTE at 21 weeks (a), axial T1 VIBE at 25 weeks (b), axial T2 at 5 months (c), and axial CT at 7 months (d). With progressive decrease in size of the thrombus and the tDSM, there is residual peripheral calcification along the margins of the tDSM. Case 7, as imaged by axial HASTE at 24 weeks (e), axial T2 (f) and MRV coronal MIP at 13 days of life (g), and axial CT at 4 months of age (h). Note that with dramatic reduction of the thrombus by 13 days of life, there was patency of the dural sinuses (g). At the last available follow-up, there was faint peripheral calcification at the torcular which appeared extradural on careful review (h). In case 8, a large torcular thrombus was visible by sagittal (i) and axial (j) HASTE imaging at 26 weeks. By 33 weeks gestation, the thrombus had contracted and developed peripheral echogenicity with posterior acoustic shadowing (arrow, k) suggestive of calcification; peripheral areas of pulsatile flow previously seen on Doppler ultrasound had resolved. At term delivery, there was residual clot which appeared centered within the torcular leaflets apart from an apparent dural rent on the sagittal T1 images (arrow, l) suggesting decompression from the occipital sinus into the intradural space. An axial T1 postcontrast MPRAGE image obtained during this perinatal scan demonstrated smooth borders of this clot and preserved opacification of the dural sinuses (m), also consistent with intradural location. By the 3 month follow-up, this intradural clot had contracted and the area of dural sinus rent had resolved on postcontrast MPRAGE imaging (n).

Supplementary Figure 5

Supplementary Figure 6

Case 12 imaged at 2 months of age (a-c), 7 months of age (d-f), and 19 months of age (g-j). After presenting with prominent scalp veins at 2 months, sagittal T1 (a) and axial T2 (b) images demonstrate torcular thrombus. While not having convex outward borders, the torcular still demonstrates remodeling with abnormal dilation of the torcular characteristic of a tDSM (i.e. greater than the brainstem in AP dimension). A coronal oblique (LAO projection) MIP of a time of flight MRV demonstrates lack of flow related enhancement through the torcular. No abnormal arteriovenous communication was demonstrated by MRA at 2 months (not shown), and a catheter angiogram at 7 months also demonstrates no abnormal arteriovenous communication though there is evidence of redirected venous drainage into the superficial system (d-f): A right external carotid artery injection lateral projection (d) and right vertebral artery injection anterior projection (f) are shown in the arterial phase with a lateral projection right internal carotid injection in the venous phase (e). Most recent follow-up at 19 months demonstrates contraction of clot and tDSM size on sagittal T1 SPGR postcontrast (g) with axial reformat (h) as well as on the axial T2 sequence (i). The residual clot appears sequestered inside the torcula with smooth margins and torcular contrast enhancement along the periphery. A coronal oblique MIP (LAO projection) of a time of flight MRV demonstrates peripheral recanalization around the sequestered residual clot.

Supplementary Figure 6