Case History
Aneurysmal malformation of the vein of Galen was suspected on the basis of the MRI findings, and the patient was referred for further evaluation. Postnatal DSA corroborated the findings. Glue embolisation was subsequently done, with successful obliteration of the malformation. However, the child died at 29 hours of age from intractable congestive heart failure.

Discussion
Vein of Galen aneurysmal malformations (VGAM) are rare congenital vascular malformations characterised by shunting of the arterial flow into an enlarged cerebral vein dorsal to the tectum. Most of these malformations present in early childhood, often causing congestive heart failure in the neonate.1

VGAM is defined as an aneurysmal dilatation of the vein of Galen, with arterial input from one or more major intracranial
arteries, either directly or via an interposed angiomatous malformation. Arteriovenous malformations arise when fistulas develop in positions where primitive vessels cross in the embryo, which is most prominent near the choroid plexus. Fistula formation in the deep midline region, therefore, results in a malformation consisting predominantly of the choroidal vessels drained by the deep venous system, of which the vein of Galen is the main channel.

Although VGAMs constitute only 1% of all cerebral vascular malformations, they comprise up to 30% of all paediatric vascular malformations. VGAM develops between the 6th and the 11th week of gestation, after the development of the circle of Willis. Other venous anomalies such as anomalous dural sinuses and sinus stenoses are commonly present in association with VGAM. A fully developed vein of Galen malformation may vary considerably in complexity from an aneurysmal dilatation fed by the branches of posterior choroidal or posterior cerebral vessels, to an extremely complex malformation fed by all major intracranial vessels. Antenatal MRI can show the malformation in three dimensions and depict the exact anatomy of the dilated channels and thrombosis if any. Magnetic resonance angiography (MRA) with 2D-TOF may be a useful additional technique for evaluating foetal VGAM. Embolisation of the feeding arteries is the preferred therapeutic modality for a patient with severe cardiac failure. MRI is mandatory before endoarterial treatment, to assess the brain parenchyma. If there is severe parenchymal damage, endovascular treatment cannot compensate for the irreversible melting-brain process. MRI has a prognostic value, allowing the decision for therapeutic approach. Angiography is mandatory only at the time of endovascular treatment, while MRA and MRI have a role in follow-up.

Antenatal USG and Colour Doppler can make the diagnosis of VGAM; however, MRI can not only make the diagnosis but also evaluate any associated parenchymal damage and thrombosis to a better extent, because of excellent soft-tissue contrast and lack of interference by bony structures. In conclusion, MR imaging and MR angiography can indicate the major vessels of supply, tortuosity of accessible arteries, venous anatomy, and parenchymal / ventricular status even antenatally and aid in the management and follow-up of such lesions.

References