INTERESTING IMAGES

Vein of Galen aneurysmal formation

Case report

A three-year-old child presented with delayed developmental milestones and seizures. On examination the head circumference was below the 10th percentile. There was no cardiac failure or bruit.

Discussion

This patient has cerebral atrophy with evidence of chronic cerebral ischaemia resulting from the vascular steal and cerebral venous hypertension. This is the chronic phase of the disorder where endovascular occlusion of the fistula is unlikely to improve symptoms.

In VGAM, the large draining vein, the promesencephalic vein, is the embryonic precursor to the Vein of Galen. The feeding arteries are the choroidal vessels and subependymal vessels from the posterior Circle of Willis. This disorder must be differentiated from the Vein of Galen malformation, in which the Vein of Galen is well-formed.

Figure 1a and b: CT scan pre- and post-contrast injections, demonstrated global cerebral atrophy with focal parenchymal calcification and a large central draining vein with serpiginous vessels suggesting a Vein of Galen aneurysmal malformation (VGAM).

Figure 2a and b: T2 weighted MR and time of flight MR confirms a central promesencephalic draining vein with a fistula arising from the anterior and posterior choroidal arteries.
Vein of Galen aneurysmal formation

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Figure 3: Carotid angiogram confirms the fistula and draining vein of a VGAM

The natural history of VGAM is variable. In the infant, presentation with macrocrania and hydrocephalus are more common. These are due to venous congestion and / or thrombosis. In the child delayed developmental milestones and convulsions are common, arising from the subependymal atrophy and chronic venous ischaemia.

Primary hydatidosis of the thyroid gland: a case report

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centre the incidence of carcinoma in patients with solitary nodules is 70%, compared to 17% found by McCall et al. We feel that the reason for this discrepancy is that at our centre we analyze the scan during both dynamic and static phases. The lesion is considered malignant if a photopenic area seen on static images shows perfusion during the dynamic phase of the study. The lesion is considered benign if a photopenic area on static image shows no vascularity during the perfusion phase of the investigation. Our case displayed benign features on scintigraphy as shown on Figure 1. Evidence of diminished function in a Tc-99m scan is an indication for FNA of a nodule. Aspiration cytology showed multiple calcified bodies and very few cells, with groups of scolices resembling Echinococcus granulosus. Should ultrasound have been performed, the potential problem of spillage could have been avoided. Ultrasound may confirm the diagnosis of hydatid disease by demonstration of a ‘cyst within a cyst’ or other features.

After the left thyroid lobectomy, histological examination revealed a parasite membrane with a degenerated germinal layer. Many degenerated scolexes and hooklets were seen with extensive dystrophic calcification. The surrounding thyroid tissue was severely inflamed with many foreign body giant cells being seen, thus allowing a diagnosis to be made.

In conclusion although our results were satisfactory, we hope that this rare case of primary hydatidosis of the thyroid will prove useful to justify routine use of ultrasound in the evaluation of thyroid nodules, especially where a cystic lesion is suspected. With surgery still the treatment of choice, we believe that scintigraphy combined with ultrasound prior to FNA will make the surgeon aware of this possibility so as to follow the special therapeutic policy necessary to avoid dangerous spillage of the parasite.

References