

## Letters to the Editor

Dear Editor,

## CEREBRAL PROLIFERATIVE ANGIOPATHY: A RARE FORM OF VASCULAR MALFORMATION

Cerebral proliferative angiopathy (CPA) is a relatively recently described entity and differs from other arterio-venous malformations (AVMs) in its angio-morphology, histology, epidemiology, natural history and clinical presentation. In a case series,<sup>1</sup> CPA was diagnosed in 3.4% of brain AVMs and was more common in young females. The natural history of CPA differs from other AVMs in that haemorrhagic events are less common. On histology, CPA is characterised by the presence of normal-appearing neural tissue intermingled between the vascular channels. Ischaemia is the major cause of symptoms and is multifactorial in origin, possibly secondary to incompetent angiogenesis, 'steal' phenomena, arterial stenosis and capillary wall involvement.<sup>1</sup>

We describe a 16-year-old, right-handed, white girl who was referred with progressively worsening episodic headaches associated with right-sided motor weakness, ongoing since the age of 8 years. They would be followed by intense, bi-frontal, pulsatile headaches that would last for anything from 30 minutes to a few hours. General physical and neurological examination was normal.

Magnetic resonance imaging (MRI) showed a diffuse network of densely enhancing vascular spaces over the left parietal region with intermingled normal brain parenchyma (Fig. 1). Cerebral angiography demonstrated a widespread angiopathy supplied by numerous arterial branches and a diffuse network of vascular spaces with relatively slow shunting, suggestive of CPA (Fig. 1).

The episodes of transient weakness in our patient were likely to be secondary to ischaemia, and the risk of stroke due to haemorrhage was low. Surgical intervention was not felt to

be appropriate because of relatively low risk of haemorrhage in the long term without intervention and high risk of significant morbidity due to interspersed normal neural tissue with intervention.

Clinically, patients with CPA are more likely to present with headaches, seizures and progressive neurological deficits rather than acute haemorrhage, when compared with other AVMs. Primary indications for invasive intervention in CPA include haemorrhage, identifiable fragile angio-architecture and persistent disabling symptoms. Various treatment options include partially targeted embolisation in non-eloquent areas or calvarial burr-holes in cases where the spontaneous transdural supply is poor. Burr-holes increase the cortical blood supply by recruiting additional blood vessels as employed in other similar conditions such as moyamoya disease.<sup>2</sup>

Evidence of ongoing angiogenesis with elevated levels of vascular endothelial growth factor (VEGF) has been described in patients with CPA; however, it is currently not clear if treatment with monoclonal antibody against VEGF could be of benefit.<sup>3</sup>

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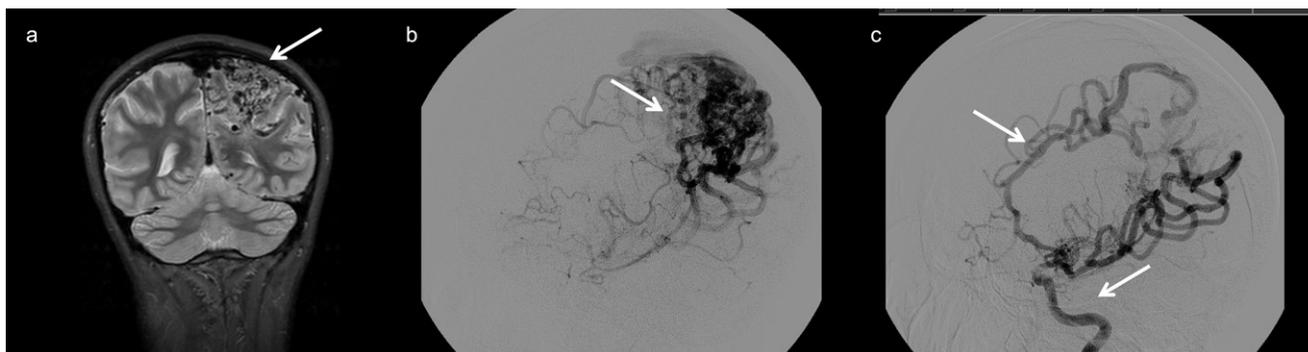
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**Fig. 1** (a) T2-weighted coronal image from MRI brain showing large left parietal vascular malformation with multiple flow voids, (b) diffuse circa 5 cm fronto-parietal nidus on the left side and (c) the nidus being fed by equally dilated multiple branches of the anterior and middle cerebral arteries and lenticulostriate arteries (transmantle).

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Conflict of interest: None declared.

## References

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Dear Editor,

### MULTIPLE MAGNET INGESTION IN CHILDREN: NEAR-FATAL ATTRACTIONS

We report two recent cases of multiple magnet ingestion in children. The children, aged 18 months and 20 months, presented with vomiting, reduced oral intake and irritability and were initially thought to have viral gastroenteritis. On presentation, the children's physical examinations were only remarkable for signs of dehydration. Neither child had abdominal radiographs at admission due to a lack of clinical signs. With persistent vomiting on day 2 of admission, now bile-stained, and abdominal distension, abdominal radiographs were performed. Plain abdominal X-rays revealed the presence of multiple radiodense objects intra-abdominally with associated small bowel obstruction (see Fig. 1).

Each of the children underwent emergency laparotomy and required resection of significant portions of small bowel due to the presence of multiple perforations. The individual magnets (five in one patient, seven in the other), in separate parts of the small intestine, had clumped together, thereby trapping intestinal wall between them. The trapped bowel wall between the magnets had become necrotic and had perforated (Fig. 2). Neither child had peritonitis as the perforations were localised by the magnets. Both children had unremarkable postoperative recoveries. On subsequent questioning, both sets of parents recalled recent events where magnet ingestion may have occurred.

The accidental ingestion of magnets by children is becoming increasingly common due to the rise in popularity of toys and novelty products containing small magnets.<sup>1</sup> Unfortunately, the symptoms of magnet ingestion are vague,<sup>2,3</sup> and obtaining a history can be difficult, as many caregivers may be unaware that the ingestion has occurred. Most solitary magnets will pass through the gastrointestinal tract without complication<sup>4</sup>; however, the ingestion of multiple magnets is dangerous, as magnets in adjacent bowel loops attract to one another, potentially leading to bowel perforation, obstruction, volvulus or fistula formation.<sup>2</sup> Interestingly, the localisation of the bowel trauma between the magnets can prevent overt peritonitis, thereby delaying investigations, diagnosis and surgery.

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**Fig. 1** A plain abdominal X-ray showing multiple intra-abdominal opacities (individual magnets clumped together) with associated bowel obstruction.



**Fig. 2** An intra-operative picture showing magnets extruding through perforations in the small intestine.