few. One such complication was described in a 29-year-old woman who developed signs of meningeal inflammation and an intracranial mass related to the use of gelatin sponge.[4] She was treated with intravenous dexamethasone with complete resolution of symptoms. The radiological findings required 5 months to resolve.

Three cases were reported in which large granulomas developed 13 to 21 months after removal of intracranial meningiomas (two cases) and after operation for an anterior communicating aneurysm.[5] In each of these cases, oxidised cellulose had been left in place for haemostasis. All of the granulomas acted as space-occupying lesions that were removed operatively with good result. Histological study revealed the presence of mononuclear phagocytes and multinuclear giant cells.

Other synthetic materials that may be left in place during intracranial procedures include silicone-coated sheets used as dura mater substitutes, chemotherapy wafers, and agents used for embolisation of highly vascular tumours such as polyvinyl alcohol particles and tris-acryl gelatin microspheres used for embolisation.[1]

There are few reports in the literature that discuss the CT appearance of textiloma. Gondo et al. describe the CT findings for three patients who underwent craniotomies for tumours and in whom was fibrillated collagen applied for haemostasis.[6] CT scans carried out 3 months postoperatively showed contrast enhancement suggestive of brain abscess or residual and recurrent tumour. On repeat craniotomies, however, histological examination showed residual fibrillated collagen with necrotic tissue and granulation.

A search through the literature revealed no other case reports of an intracranial foreign body reaction secondary to embolisation of an AVM using *N*-BCA. The use of *N*-BCA for embolisation of cerebral AVMs is fairly common, and its safety and efficacy has been documented in the literature.[7] In another study looking at tissue reactions induced by different embolising agents in cerebral AVMs, it was shown that irrespective of the agent used, the histological progression consisted of acute inflammation with mural angionecrosis that was soon replaced by prominent chronic granulomatous vasculitis, which remained stable for up to 6 years.[8]

In our case it would have been impossible to make the diagnosis without surgical intervention. This case illustrates the importance of considering foreign body reactions in the differential diagnosis for patients who present with a space-occupying lesion if they have undergone a prior neurosurgical procedure.

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Idiopathic hypertrophic cranial pachymeningitis

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Abstract

Idiopathic hypertrophic cranial pachymeningitis is a rare chronic inflammatory process of unknown origin that can cause neurological deficits owing to thickening of the dura. Patients with this condition commonly present with cranial neuropathy accompanied by localized headache. The clinical features, neuroimaging findings, histopathological features and treatment outcomes for three patients with this condition are reported here. The first patient presented with subacute dull headache in the left temporal area followed by left abducens nerve palsy. The second patient suffered from a cranial nerve IX–XII lesion accompanied by an occipital headache and the third patient presented with left optic neuropathy and mild headache in the frontal area. In all patients, MRI of the brain revealed prominent dural thickening, and histopathological study of the dura revealed chronic inflammatory cell infiltration. Combined therapy with corticosteroid and immunosuppressive drugs was effective, resulting in almost complete resolution of the symptoms and signs, except for visual impairment in one patient.

Keywords: Hypertrophic pachymeningitis; Inflammatory process; Cranial neuropathy; Headache

1. Introduction

Idiopathic hypertrophic cranial pachymeningitis is a rare chronic inflammatory process of unknown origin that can cause neurological deficits owing to thickening of the dura. Three patients with this condition are reported here.

2. Case report

2.1. Case 1

A 60-year-old woman presented in October 2002 with a history of dull headache over the left temple for 8 weeks and double vision for 1 week. A physical examination revealed left cranial nerve (CN) VI palsy. MRI of the brain revealed an enhancing mass along the left dural line of the pre-pontine cistern, encasing the left CN VI, together with thick dural enhancement along the left cavernous sinus and superior orbital fissure (Fig. 1). The erythrocyte sedimentation rate (ESR) was elevated, at 56 mm/h. The results of cerebrospinal fluid (CSF) study were normal. Biopsy of the dura revealed dense fibrous tissue infiltrated by mononuclear inflammatory cells. Stains for bacteria, fungi and acid-fast bacilli yielded negative results. The patient was treated with 1 g of methylprednisolone intravenously for 5 days, followed by prednisolone and azathioprine treat-

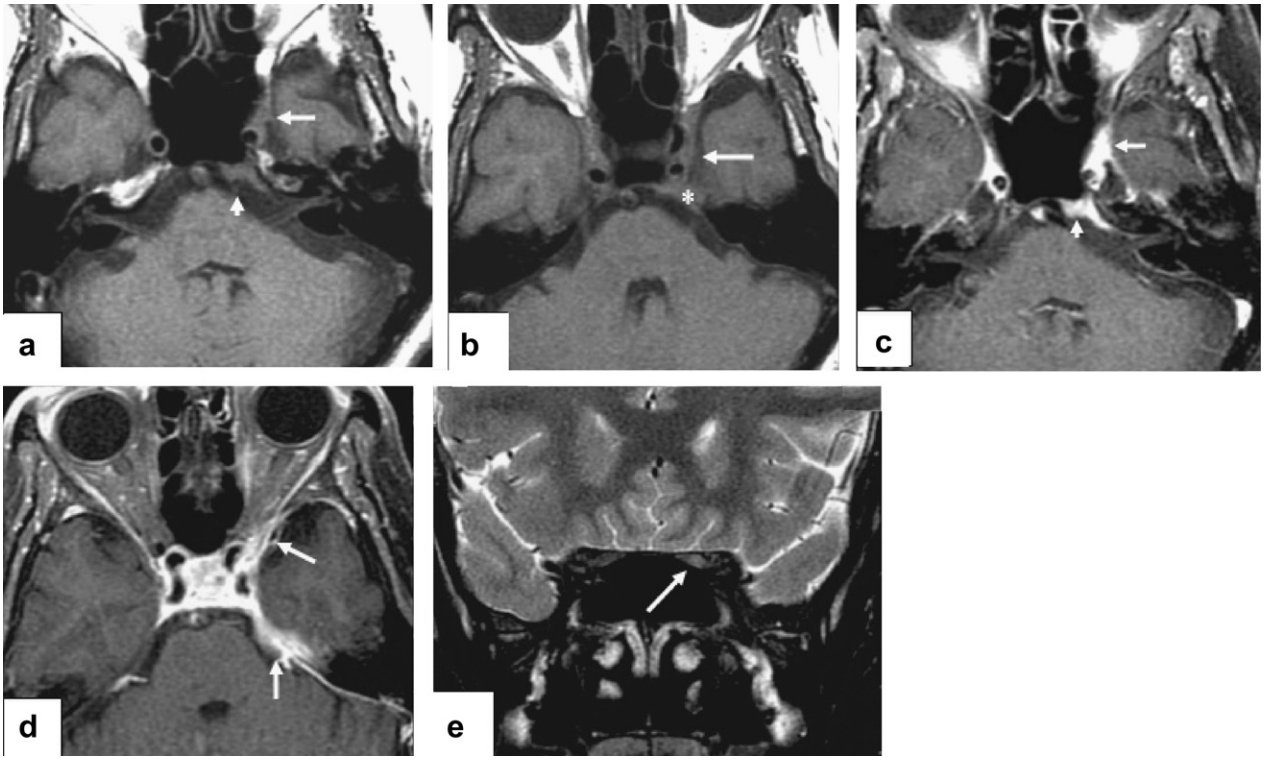

Fig. 1. Case 1. Axial T_1-weighted MR images with fat suppression before (a,b) and after (c,d) gadolinium administration reveal an enhancing isosignal T_1 lesion along the left dorsal aspect of the clivus at the Dorello foramen (arrows in a and c) and along the left cerebellopontine angle cistern as well as the left tentorial sleeve and lateral cavernous sinus wall to the superior orbital fissure (arrows in b and d). The lesion also occupies the left Meckel's cave (white asterisk in b). A coronal fast spin-echo T_2-weighted fat suppression MR image (e) shows an abnormal hypersignal T_2 change of the left optic nerve (arrow in e), probably secondary to compressive ischaemic insult.