occurrence of hemorrhage. In our patient, no previous low cranial nerve palsies could be traced.

ECs usually originate from the short segment of the wall of the neural tube from which the tela choroidea develops.[1] Therefore, these cysts may comprise various tissues, such as cilia, choroid plexus, and glial tissue. Some mechanisms by which fluid can accumulate within ependymal cysts include transcellular transport, active secretion, and passive transport.[4] The cyst might enlarge gradually due to breakdown of the ependymal lining and accumulation of secretory material.[12]

The natural history of these ependymal cysts is unknown. We speculate that their asymptomatic nature is responsible for their rarity. Ho et al.[5] reported an asymptomatic patient who was diagnosed at autopsy. Our patient presented only after hemorrhage. Thus, there may be patients who remain asymptomatic. Accordingly, follow-up at regular intervals with clinical examination or imaging studies is a reasonable course of action, unless it causes clinical symptoms or hemorrhagic complications. Several surgical methods for treatment have been described, including open craniotomy with total excision, shunting, fenestration of the cyst into the subarachnoid space, and stereotactic aspiration.[11] However, for hemorrhages and cysts with mass effect, surgical resection might be the most effective strategy.

CPA EC is a rare entity. It is usually observed in young females, and it influences the seventh, eighth, or ninth cranial nerves. Hemorrhagic complications can occur in the elderly, as in our patient. CPA EC can be managed by surgical resection via a retrosigmoid approach. The clinical course and outcome are usually benign and favorable, respectively.

doi:10.1016/j.jocn.2008.02.019

References

1. Friede RL, Yasargil MG. Supratentorial intracerebral epithelial (ependymal) cysts: review, case reports, and fine structure. *J Neurol Neurosurg Psychiatry* 1977;**40**:127–37.
2. Boockvar JA, Shafa R, Forman MS, et al. Symptomatic lateral ventricular ependymal cysts: criteria for distinguishing these rare cysts from other symptomatic cysts of the ventricles: case report. *Neurosurg* 2000;**46**:1229–32, discussion 32–3.
3. Czervionke LF, Daniels DL, Meyer GA, et al. Neuroepithelial cysts of the lateral ventricles: MR appearance. *AJNR Am J Neuroradiol* 1987;**8**:609–13.
4. Pant B, Uozumi T, Hirohata T, et al. Endoscopic resection of intraventricular ependymal cyst presenting with psychosis. *Surg Neurol* 1996;**46**:573–6, discussion 6–8.
5. Ho KL, Chason JL. A glioependymal cyst of the cerebellopontine angle. Immunohistochemical and ultrastructural studies. *Acta Neuropathol (Berl)* 1987;**74**:382–8.
6. Saito K, Morita A, Shibahara J, et al. Spinal intramedullary ependymal cyst: a case report and review of the literature. *Acta Neurochir (Wien)* 2005;**147**:443–6, discussion 6.
7. Nakase H, Ohnishi H, Touho H, et al. Large ependymal cyst of the cerebello-pontine angle in a child. *Brain Dev* 1994;**16**:260–3.
8. Harada A, Takeuchi S, Inenaga C, et al. Hemifacial spasm associated with an ependymal cyst in the cerebellopontine angle. Case report. *J Neurosurg* 2002;**97**:482–5.
9. Sharma RR, Pawar SJ, Kharangate PP, et al. Symptomatic ependymal cysts of the perimesencephalic and cerebello-pontine angle cisterns. *J Clin Neurosci* 2000;**7**:552–4.
10. Monaco P, Filippi S, Tognetti F, et al. Glioependymal cyst of the cerebellopontine angle. *J Neurol Neurosurg Psychiatry* 1995;**58**:109–10.
11. Colnat-Coulbois S, Marchal JC. Thalamic ependymal cyst presenting with tremor. *Childs Nerv Syst* 2005;**21**:933–5.
12. Ciricillo SF, Davis RL, Wilson CB. Neuroepithelial cysts of the posterior fossa. Case report. *J Neurosurg* 1990;**72**:302–5.

Minimal surgery for a cerebellopontine angle lipoma

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Received 2 September 2007; accepted 8 January 2008

Abstract

A 46-year-old man presented with a left cerebellopontine angle lipoma of unusual size. The patient also complained of hearing loss and left trigeminal neuralgia, which were triggered on the same side as the lesion in the resting posture. Surgical treatment with simple debridement of the arachnoid membranes to reduce internal tension in the tumour resulted in stable pain remission and hypoacusia without additional deficit.

Keywords: Trigeminal neuralgia; Cerebellopontine angle lipoma; Lipoma

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1. Introduction

Intracranial lipomas are very rare lesions, accounting for 0.08% of all brain tumours.[1] Owing to their particular location, lipomas in the cerebellopontine angle (CPA) may cause symptoms mimicking trigeminal neuralgia in about 10% of patients.[1,2] It is still unclear if these lesions are true tumours and if their growth is due to an increased cellular population or to cellular volume. Lipomas cause an enlargement of the basal cisterns and grow in the subarachnoid space, thus keeping close contact with the vessels and nerves. Surgical removal of lipomas, where indicated, requires great caution.[1–3]

2. Case report

A 46-year-old man was admitted to our neurosurgery department in 2001 with a 12-year history of left-side hearing loss followed by mild ipsilateral trigeminal neuralgia, which had been sustained without therapy. An MRI performed 10 years previously had revealed the presence of a left cerebellopontine angle lipoma measuring $35 \times 28 \times 20$ mm (Fig. 1). In March 2000 the trigeminal neuralgia became severe and frequent within a short time. Therapy with carbamazepine was scheduled, with a dosage progressively increasing to 1200 mg/day, but its effects were only mild. On admission in 2001, the severity of the neuralgia was incompatible with good quality of life. The trigeminal neuralgia was peculiar in that it depended on the patient's posture. Pain was triggered or worsened when he lay on his left side and disappeared or was alleviated when he lay on his right side. Therefore, the patient could only sleep on the right side. A general examination revealed moderate obesity (body weight 80 kg and height 168 cm), which had developed during the previous 10 years. Total serum cholesterol values were 288 mg/dL, triglycerides 147 mg/dL and blood sugar 190 mg/dL. A neurological examination showed severe left hypoacusia, right horizontal nystagmus, second trigeminal branch hypoesthesia, and left masseter hypotrophy. Pain was easily triggered in the malar region. Despite the severity of the symptoms, a new MRI (March 2001) failed to demonstrate any increase

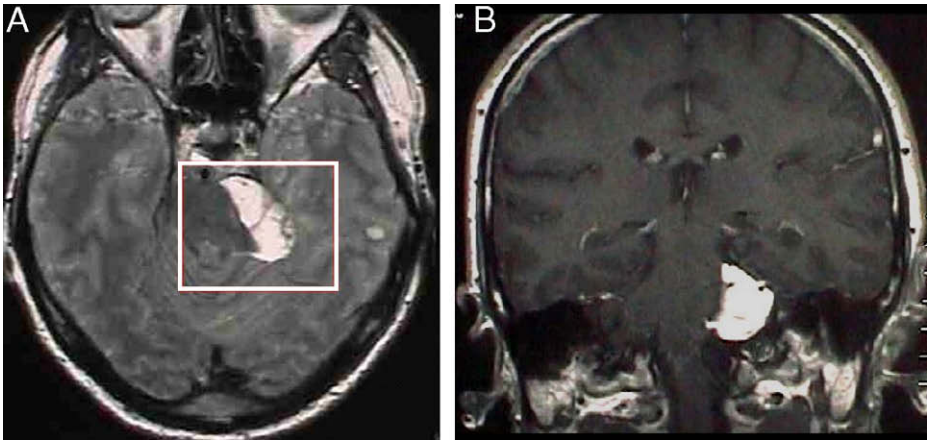

Fig. 1. (A) Pre-operative axial and (B) coronal T1-weighted MRIs showing (in the white box) a cerebellopontine angle lipoma compressing the brainstem at the level of the incisura, with the trigeminal root pushed upward and the peculiar vascular supply of the brainstem with small arteries trespassing the lipoma from the posterior cerebral artery P1 tract to the brainstem.

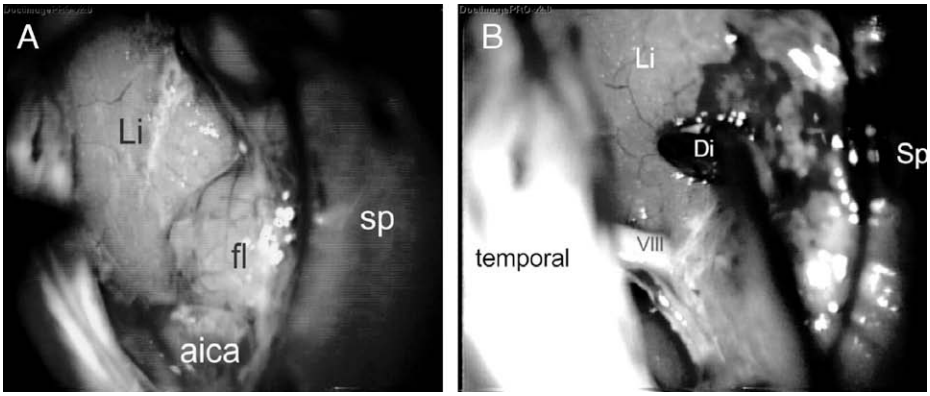

Fig. 2. Intra-operative images. (A) The surgical field at the opening showing the lipoma enveloped by arachnoid. (B) The caudal part of the lipoma after the flocculus had been dislocated. In the inferior part the acoustic nerve is visible before entering the meatus separately to the facial nerve. Di, dissector; fl, flocculus; Li, lipoma; sp, Sp spatula; VIII, acoustic nerve; aica, anterior inferior cerebellar artery.