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Middle ear infection and sixth nerve palsy

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Abstract
Three cases of patients with acute middle ear infections complicated by a sixth nerve palsy are described. The possible causes of sixth nerve palsy in such cases are discussed. We propose that where there is a sixth nerve palsy without petrositis and without raised intracranial pressure, it can arise as a result of spreading phlebitis along the inferior petrosal sinus from the lateral sinus.

Key words: Abducens nerve; Mastoiditis; Otitis Media, Gradenigo syndrome

Introduction

The incidence of acute mastoiditis and the intracranial complications of middle ear cleft infection has greatly diminished since the introduction of antibiotics (Ludman, 1988). The incidence of acute mastoiditis in the Wessex Region was estimated at 3.3/million/year in 1992 (Pearson *et al.*, 1994).

Complications arising from acute mastoiditis occur most commonly in children and adolescents (Singh, 1993). In children, 10–15 per cent of cases are associated with intracranial complications (Ogle and Lauer, 1986). The commonest of these complications are meningitis, extradural abscess and cerebral abscess. Although the incidence of mastoiditis and intracranial complications has declined, the mortality of the latter has not and remains around 20 per cent (Ludman, 1988).

In a patient presenting with a sixth nerve palsy and a middle ear infection, the complications which are recognized as a cause are otitic hydrocephalus, Gradenigo's syndrome and cavernous sinus thrombosis. All of these complications are rare.

Case reports

Case 1

A 15-year-old girl presented with a week's history of right otorrhoea, followed by headache, vomiting and diplopia. On examination she was found to have a pyrexia, a left (contralateral) sixth nerve palsy, neck stiffness and bilateral papilloedema. She had a right attic defect and was found to have tenderness and erythema over the right mastoid area. She was commenced on intravenous antibiotics.

A CT scan showed an opacified and poorly developed mastoid system on the right side with erosion of the malleus and incus, and a loculus of gas in the sinodural angle. The left side was normal.

At surgery, the findings were mastoiditis, with a small sclerotic mastoid system containing cholesteatoma. The head of malleus and body of incus were eroded but the tegmen tympani/antri and sinus plate were not. There was an extradural parasinus abscess and lateral sinus thrombosis from the mastoid tip to the horizontal sinus (which was discernible on subsequent magnetic resonance imaging (MRI). A modified radical mastoidectomy was carried out, and an extradural drain left in situ. The patient made a full neurological recovery three days post-operatively.

Case 2

A six-year-old boy presented with a four-day history of left otalgia followed by drowsiness and diplopia. On examination he was found to have a pyrexia, a red left tympanic membrane, erythema and tenderness over the left McEwan's triangle and a left sixth nerve palsy. There was no papilloedema or any other neurological signs. After being commenced on intravenous antibiotics, a computed tomography (CT) scan was carried out and showed opacification of the left mastoid antrum but no involvement of the petrous apex or surrounding area. There were no other abnormalities.

A cortical mastoidectomy was carried out and a 6 ml extradural parasinus collection of pus was drained. This collection extended from the sinodural angle towards the jugular bulb. The lateral sinus itself was firm and coated with perisinus fibrin deposition but it was patent, with a free flow of blood on aspiration. He made a full recovery post-operatively with the sixth nerve palsy resolving after two weeks.

Case 3

A 10-year-old girl presented with a week's history of left otalgia and two days of diplopia. On examination she was febrile and had a red left tympanic membrane with tenderness over McEwan's triangle. She had a left sixth nerve palsy, with no other neurological signs including papilloedema. She was commenced on intravenous antibiotics. A CT scan showed clouding of the left mastoid antrum but there were no other abnormalities, in particular the region of the petrous apex was normal (Figure 1).

She underwent a cortical mastoidectomy which showed mucosal oedema of the antrum with no cholesteatoma, pus

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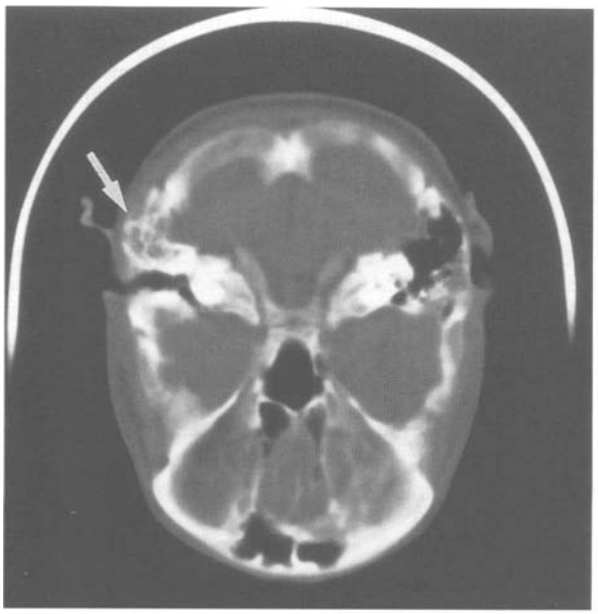

FIG. 1

Case 3. Transverse CT scan showing opacification of left mastoid system as the only abnormal finding.

or signs of petrous apex involvement. The lateral sinus was firm, being covered with fibrin but it was patent and a free flow of blood was demonstrated on aspiration.

She made a full recovery post-operatively. The sixth nerve palsy fully resolved after three weeks.

Discussion

Guiseppe Gradenigo first described a syndrome of paralysis of the sixth cranial nerve, retro-orbital pain and middle ear infection in 1904 (Gradenigo, 1904). This triad is classically attributed to petrositis, the trigeminal ganglion and sixth cranial nerve being adjacent to the petrous apex. The abducent nerve pierces the dura mater lateral to the dorsum sellae after ascending the clivus and bends acutely over the superior edge of the petrous bone, entering a fibro-osseous canal (Dorello's canal) formed by the apex of the petrous bone and the petrosphenoidal ligament (Williams *et al.*, 1989).

The actual pathological mechanism is local pachymeningitis secondary to the petrositis with resultant inflammation of the adjacent abducent nerve (de Graaf *et al.*, 1988).

Gradenigo's syndrome can also be caused as a result of an extradural abscess or an area of pachymeningitis overlying the petrous apex without there necessarily being petrositis per se (Ransome, 1987).

In the absence of petrositis, the possible pathways of infection or inflammation to spread from the middle ear cleft to the dura over the petrous apex include: the dura itself as pachymeningitis, an extension of an extradural abscess, or via phlebitis of the lateral sinus extending along the inferior petrosal sinus.

The inferior petrosal sinus runs postero-inferiorly from the cavernous sinus in a groove between the petrous and basilar occipital bone. Shortly after leaving the cavernous sinus, it is closely related to the sixth nerve as the nerve pierces dura (Williams *et al.*, 1989).

The superior petrosal sinus is not closely related to the sixth nerve, running on top of the trigeminal nerve immediately after leaving the cavernous sinus.

In otitic hydrocephalus, the cause of the sixth nerve palsy is raised intracranial pressure with resultant stretching of the nerve. The palsy can be either side and is hence a 'false localizing sign'. The raised intracranial pressure itself is probably due to a combination of lateral sinus thrombosis impeding cranial venous outflow and spread to the superior saggital sinus impeding CSF absorption by pacchionian bodies (Pfaltz and Griesemer, 1984).

Interestingly, it has been noted that the sixth nerve palsy in cases of otitic hydrocephalus is most often ipsilateral (O'Connor and Moffat, 1978). There is no reason for this if it is assumed that the cause of the palsy is raised intracranial pressure. Contralateral and ipsilateral palsies should occur in approximately equal numbers. An explanation of this observation is that in some cases, there is spread of infection from the lateral sinus which is nearly always thrombosed (Ludman, 1988) to the petrous apex dura via the inferior petrosal sinus.

In *Case 1*, there were signs and symptoms suggesting raised intracranial pressure (headache, vomiting, papilloedema); the sixth nerve palsy was contralateral; and there was a lateral sinus thrombosis. There is consequently little doubt about the cause of the palsy as being due to otitic hydrocephalus.

In the second and third cases, the sixth nerve palsy was ipsilateral to the site of mastoiditis and acute otitis media respectively. In each case there was no evidence of petrositis, nor of raised intracranial pressure. However, there was phlebitis of the lateral sinus in both cases, with there being a parasinus abscess in *Case 2* and phlebitis alone in *Case 3*. In these cases, the most plausable cause of the sixth nerve palsy is pachymeningitis around the petrous apex secondary to the spread of phlebitis from the lateral sinus via the inferior petrosal sinus. This hypothesis is further reinforced by the absence of trigeminal nerve involvement in the symptoms of the patients in these two cases. If the sixth nerve palsy arose from pachymeningitis of the petrous apex in the area where the inferior petrosal sinus is in close proximity to the nerve, one would not expect trigeminal nerve involvement. This is in contrast to classical Gradenigo's syndrome where the pachymeningitis/extradural abscess of the petrous apex involves the superior aspect in close proximity to both the fifth and sixth cranal nerves.

In conclusion, not all cases of sixth nerve palsy complicating a middle ear cleft infection are Gradenigo's syndrome due to petrositis or due to otitic hydrocephalus. Nevertheless, it is always a serious sign that requires: (a) high dose blood brain barrier-penetrating antibiotics, (b) high quality imaging, (c) attention to other possible sequelae of middle ear infection and (d) operative intervention.

Acknowledgement

Case 1 has previously been reported with a discussion of the imaging of lateral sinus thrombosis (Irving *et al.*, 1991).

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