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Abducens nerve palsy as the sole presenting symptom of petrous apicitis

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Abstract
Petrous apicitis as a potentially fatal complication of suppurative otitis media presents in a variety of forms. Gradenigo's triad of abducens paralysis, deep facial pain due to trigeminal involvement and acute suppurative otitis media rarely occurs. The conflicting symptoms reported in the literature usually result in the delayed recognition of the condition with potentially disastrous consequences. The VIth nerve palsy is considered to be the least reliable sign as it is least often present. We present a case in which it was the sole presenting symptom.

Key words: Petrous Bone; Otitis Media, Suppurative; Abducens Nerve Injury

Introduction

The incidence of petrous apicitis and the intracranial complications of meningitis, extra dural and intracranial abscess, lateral sinus thrombosis and death have greatly decreased since the 1940s with the widespread use of antibiotics for the reatment of acute otitis media.[1–3]

However, petrous apicitis continues to occur and is often diagnosed late due to the variable presentation of the disease. The most common clinical findings are purulent otorrhoea, retrobulbar and/or facial pain, VIth and VIIth cranial nerve palsies and vertigo. Chole and Donald[4] in their historical review illustrated how authors have continued to debate the most useful symptoms in diagnosis of the disease since Gradinego first decribed the classical findings in 1904. These included abducens paralysis, deep facial pain due to trigeminal involvement and acute suppurative otitis media. In his original description of the disease, only 42 per cent of the cases he described actually had the classical symptom triad.[5]

The triad is thought to come about through the direct extension of infection from the petrous apex to involve the gasserian ganglion of the trigeminal nerve and the abducent nerve as it courses through Dorello's canal under the petroclinoid ligament.[3,4]

The relatively few case reports published have illustrated the variable presentation of the disease. In a significant number of cases, the diagnosis was only made at postmortem.[4] The authors have tried to determine the most common symptoms, the majority claiming deep facial pain (retrobulbar) and purulent otorrhoea to be the most useful in diagnosis.[5] Abducens palsy appears to be the least common sign and is often absent.[4,5] Chole and Donald conclude that this is the least realiable sign to use for making the diagnosis of acute petrous apicitis.

Case report

A seven-year-old girl was admitted under the Paediatric Department's care with a two-week history of horizontal diplopia. She had seen an Optometrist with sudden onset of blurred vision and had been referred to the Ophthalmologist with a note that the 'left eye didn't turn to the left'.

The Ophthalmologists noted a left lateral rectus palsy in an otherwise well child and ordered a magnetic resonance image (MRI) of her head. This was performed five days later in an unsupervised session so that the patient went home before the films were reported. The scans showed a mass lesion in the left petrous apex and evidence of inflammatory disease of the middle ear cavity. The diagnosis of petrous apicitis was suspected and the child was brought back to the department for contrast enhanced scans, under the care of the paediatricians.

The ENT department was requested to perform a tympanotomy for culture purposes. We encountered a very well child who was apyrexial with a normal pulse rate. She had not been on any antibiotics and had no otalgia, otorrhoea or decrease in her hearing. There was no deep face pain or headache and no signs of meningism. The right tympanic membrane was normal and the left was dull and bulging with no signs of an acute infection. She had a left abducens nerve palsy (Figure 1) and a small haemangioma on the left lower eyelid, that had first been noted at two months of age.

In the light of the clinical picture, the diagnosis of petrous apicitis was questioned and the contrast MRI reviewed with the Senior Head and Neck Radiology consultant. The new scans (Figure 2) revealed a ring-enhancing mass lesion in the left petrous apex, extending anteriorly to involve the posterior aspect of Meckle's cave. There was displacement of the adjacent carotid artery and enhancement of the adjacent dura, particularly in the posterior fossa extending into the left internal auditory meatus. The diagnosis of petrous apicitis was therefore confirmed. Of concern was that the inflammatory changes were immediately adjacent to the left tranverse sinus with the risk of venous sinus thrombosis. The patient was, therefore, booked for an emergency mastoid exploration and lumbar puncture under general anaesthetic.

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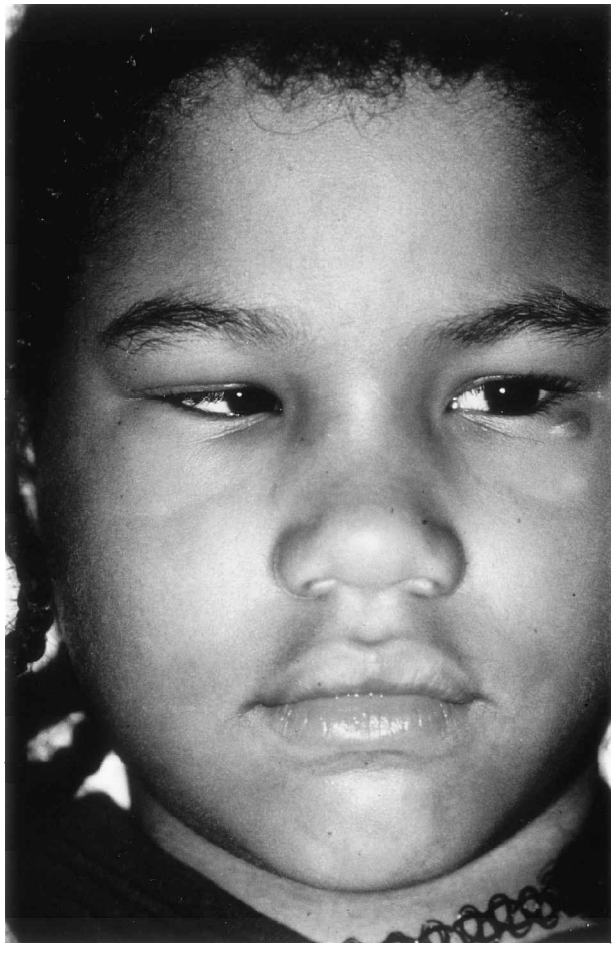

FIG. 1

Photograph demonstrating the left lateral rectus palsy and the haemangioma on the left lower lid.

Her full blood count revealed a normal white cell count (6.4) and a mildly elevated ESR (45) but a normal CRP (<4). Her CSF pressure was elevated (32 cm CSF) but clear with no organisms seen and a white cell count of 26/μl. Intraoperative findings revealed mucopus in the middle-ear cavity after performing a myringotomy. A pus swab was taken down the external auditory meatus and a Shah grommet inserted. An attico-antrostomy revealed almost all air cells to be filled with granulomatous tissue and some pus. Numerous specimens were sent for microbiological culture and for histology. The child was commenced on high dose intravenous Augmentin® and vancomycin.

The neurosurgeons were consulted with respect to draining the intracranial abcess. They requested a computed tomography (CT) scan of the temporal bones (Figure 3) and on this evidence and her clinical condition it was decided not to attempt any further surgery unless her condition deteriorated significantly.

She remained apyrexial throughout her stay in hospital and the ESR returned to normal after six days of antibiotics. Her blood cultures, CSF and tissue cultures were negative after prolonged incubation. The pus swab grew normal skin flora. The mantoux was negative as was staphylococcal serology and serum ACE and ANCA. The histology report showed chronic inflammation. Her PTA revealed a high frequency sensorineural deafness (Figure 4). Unfortunately there were no pre-operative PTAs for

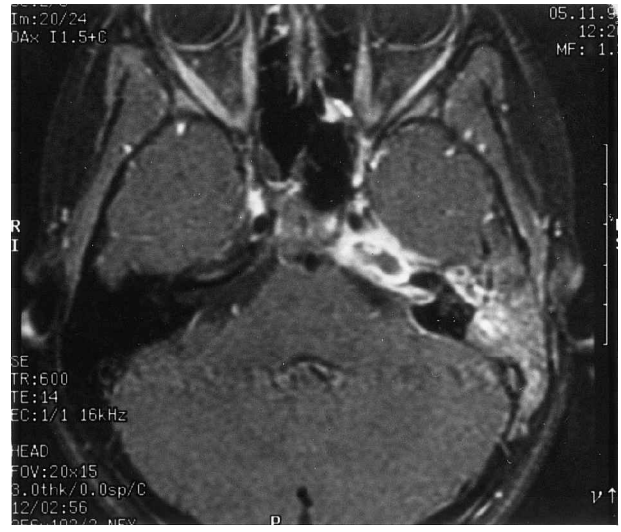

FIG. 2

T1 fat-suppressed MRI scan with contrast revealing a ring-enhanced mass lesion in the left petrous apex extending into the internal auditory meatus.

comparison. After nine days of antibiotics her lateral rectus palsy began to improve and the patient was discharged on a six-week course of oral clindamycin and amoxycillin.

She returned to clinic six weeks later and her palsy had completely resolved. Prolonged tuberculosis cultures were negative. She had had no further trouble with her ear and the left drum looked healthy. The grommet was *in situ* and patent. An MRI scan had been performed approximately a week before her visit. This scan revealed that the enhancement of the petrous apex had decreased in size and the central area of relative non-enhancement had disappeared. The enhancement of the middle-ear cavity and mastoid air cells had largely resolved.

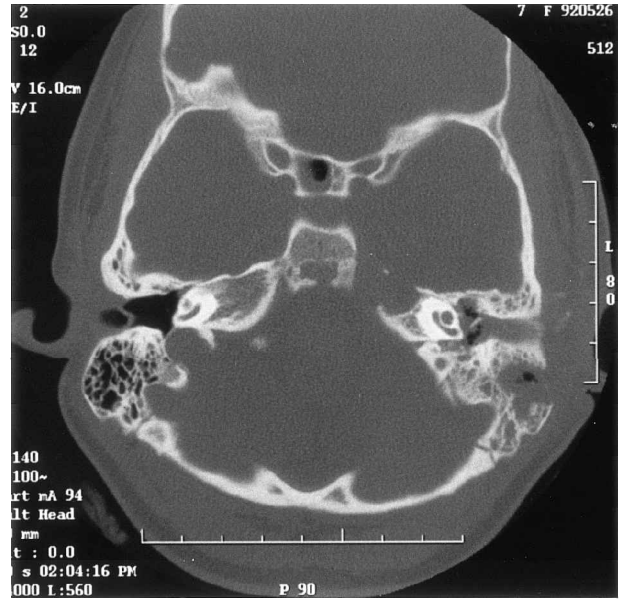

FIG. 3

CT-scan of the temporal bones showing destruction of the left petrous apex.

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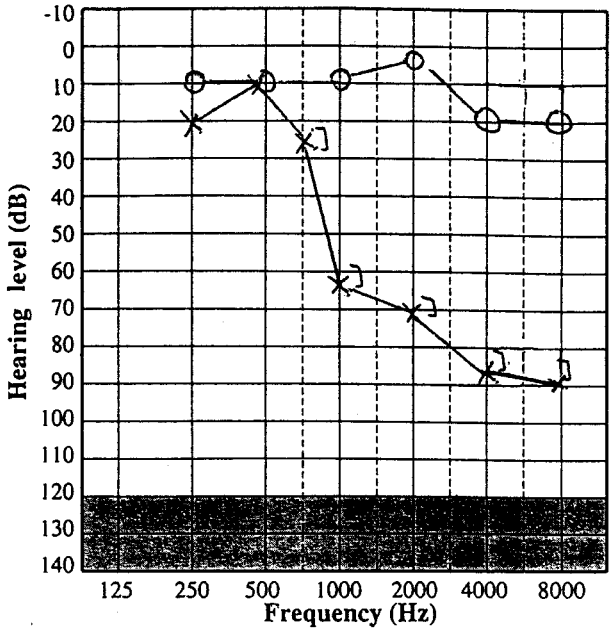

FIG. 4
An audiogram showing left-sided sensorineural deafness.

A follow-up MRI scan at 16 months has shown complete resolution of the enhancement seen in the previous scans and the petro-mastoid air cells are, once more, air containing. The grommet remains *in situ* and apart from the sensorineural hearing loss, which remains unchanged, she is symptom free.

Discussion

The young lady in question is unique in the literature in that she had no clinical sign of infection and the only sign suggestive of petrous apicitis was the isolated VIth nerve palsy. This is not considered to be a reliable sign and all the previous cases reported in the literature had some otological symptoms and signs that were absent here. This illustrates once again the variable presentation of the condition.

There are several causes of VIth nerve palsy not associated with middle-ear pathology. They include:

(1) raised intracranial pressure resulting in the false localizing VIth nerve palsy;
(2) nerve trunk infarction by diabetes, arteritis and atherosclerosis;
(3) inflammatory disease of the cavernous sinus and aneurismal dilatation of the carotid siphon within the cavernous sinus are likely to involve the nerve as it passes through the sinus;
(4) acute and chronic meningitic processes may involve the nerve in its long intracranial course;
(5) carcinoma infiltrating the orbit through the inferior orbital fissure from the nasopharynx may also cause a palsy;
(6) direct involvement of the nerve by acoustic neuroma is very rare.[6]

The causes of a VIth nerve palsy in association with a middle-ear infection are otitic hydrocephalus, Gradenigo's syndrome and cavernous sinus thrombosis. However, Gradenigo's syndrome can also be caused by an extradural abscess or an area of meningitis overlying the petrous apex without there being petrous apicitis.[7]

In this case, the diagnosis could only be made with certainty, radiologically. In recent years, increasing numbers of petrous apex lesions have been encountered as a consequence of improved imaging techniques. CT scan, by virtue of its sensitivity and low false-positive rate, is the screening examination of choice in a patient suspected of having a destructive petrous apex lesion.[8] It is particularly good at evaluating bony erosion and will detect most intracranial complications.[3] The post-operative CT performed three days after surgery clearly illustrates the destruction of the left petrous apex (Figure 3).

Once a lesion is identified, it is often necessary to perform an MRI as it provides information about the composition of the lesion. Enhancement with gadolinium DTPA is a valuable additional aid for recognizing changes in the dura and arachnoid in meningitis.[3] Figure 2 illustrates this very well. It is usually possible to differentiate between petrous apicitis, cholesterol granuloma, cholesteatoma and neoplasms such as schwannoma, meningioma, chondroma and chordoma[8] on MRI imaging.

No causative organism was isolated from the numerous specimens sent for culture. The organisms commonly involved in acute otitis media and mastoiditis include *Streptococcus pneumoniae* and beta haemolytic streptococci, *Haemophilus influenzae*, *Moraxella* (*Branhamella*) *catarrhalis*, *Staphylococcus aureus* and coagulase-negative staphylococci (e.g. *Staph epidermidis*, *Pseudomonas aeruginomas* and a variety of anaerobes.[9]

Anaerobes are most frequently found in ears with extensive cholesteatoma or granulation tissue formation.[10] The types of anaerobes implicated are *Peptococcus* sp, *Actinomyces* sp, *Clostridium* sp, *Bacteroides* sp and *Fusobacterium* sp.[11] In the case presented, there was extensive granulation tissue so the organisms involved may have been anaerobic. The negative culture result may have been due to the susceptibility of the organisms to exposure to oxygen and the need for special culture transport media, devoid of oxygen.[11] *Mycobacteria*, both *M tuberculosis* and *M avium*, should always be considered and the prolonged culture for these organisms was negative.

Her high frequency sensorineural hearing loss in the affected ear may have been due to several factors. There may have been some pre-existing hearing loss but without previous audiograms to compare with, it is not possible to say whether this was the case. The ototoxic side-effects of the antibiotics (vancomycin) was also considered but the loss is usually bilateral and symmetrical and therefore it is doubtful whether the antibiotics were causative. It is possible that there was some sensorineural damage caused by the surgery but again there are no pre-existing audiograms. It was felt that the most likely cause of her hearing loss was a direct result of the infection involving the internal auditory meatus and cochlear nerve as seen on the MRI scans.

In conclusion, any patient presenting with any of the symptoms associated with petrous apicitis even in the absence of any otological signs requires urgent admission for CT and MRI imaging as the mortality for intracranial complications of mastoiditis remains at 20 per cent.[2] Once the diagnosis is made, treatment with both high dose intravenous antibiotics and surgical decompression of the mastoid cavity is the management of choice.[5,7,12]

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