The Journal of Laryngology & Otology (2006), ***120***, *875–878.*

doi:10.1017/S0022215106000843
Printed in the United Kingdom
First published online 14 August 2006

Clinical Record

Coexistent acute pyogenic and tubercular petrous apicitis: a diagnostic dilemma

A Sethi, MS, D Sethi, MBBS, S Mrig, MBBS, J C Passey, MS, N Srivastav, MD*

Abstract

We report the case of a 16-year-old boy who presented to us with acute otitis media, facial weakness and retro-orbital pain. Computed tomography and magnetic resonance imaging (MRI) scans of the head and temporal bone revealed otitis media with petrous apicitis. The patient responded to broad-spectrum, parenteral antibiotics, with disappearance of facial weakness and reduction in pain. One month following the completion of treatment, the patient continued to have dull retro-orbital pain and developed ear discharge. A repeat MRI of the temporal bone revealed a persistent inflammatory lesion in the petrous apex, with a nodular, ring-enhancing lesion in the cerebellum, strongly suggestive of tuberculosis. The ear discharge stained positive for acid-fast bacilli and the patient's serum enzyme-linked immunosorbent assay for tuberculosis was reactive. The patient responded well to anti-tubercular treatment and was disease free eight months following the completion of treatment.

Key words: Petrous Bone; Otitis Media; Mastoiditis; Tuberculosis

Introduction

Petrous apicitis is characterized by the infection of the air cells or the marrow spaces of the petrous portion of the temporal bone.[1] It may result from an acute or chronic inflammatory process of the temporal bone.[2,3] Such a chronic inflammatory process usually involves the petrous apex as a result of bone destruction and direct extension of the disease, whereas thrombophlebitis is usually the cause in an acute inflammatory process.[4] The majority of cases result from pyogenic organisms.[5] Tuberculosis is an extremely rare cause of petrous apicitis.[3] Tuberculous involvement of the petrous bone is usually secondary to pulmonary tuberculosis and is rarely primary (i.e. the temporal bone as the primary focus of infection).[3,6]

Although the diagnosis of petrous apicitis can be made on the basis of computed tomography (CT) and magnetic resonance imaging (MRI) scans of the temporal bone,[7] these cannot differentiate between tubercular and pyogenic pathology. Thus, a coexistent tubercular and pyogenic petrositis may pose a diagnostic problem, delaying initiation of definitive management for the patient. We describe one such rare case of coexistent acute and tubercular petrositis.

Case report

A 16-year-old boy presented with complaints of fever and left earache of three days' duration and retro-orbital pain, headache and ear discharge of one day's duration. There was no history of any neurological weakness, visual disturbances, nausea or vomiting. However, the patient gave a history of five to six episodes of scant, clear, non-foul-smelling discharge from the left ear over the past two months. There was no history of past tuberculosis.

Examination of the left ear revealed a non-foul-smelling, mucopurulent discharge in the left external auditory canal and a congested tympanic membrane with a small anteroinferior perforation. The patient was found to have a mild conductive hearing loss. There was no spontaneous nystagmus. A lower motor neuron-type left facial nerve paresis was present. There was no neck stiffness or any other neurological abnormality. The right ear and the rest of the otolaryngological examination was normal.

A diagnosis of left acute suppurative otitis media with facial paresis and a possible intracranial complication was made, and parenteral antibiotics, in the form of crystalline penicillin, gentamicin and metronidazole, were started. Steroids were included in the treatment in view of the facial nerve involvement. Meanwhile, a CT scan followed by an MRI scan of the head and temporal bone were performed, which revealed an inflammatory lesion in the left petrous apex suggestive of petrous apicitis, without any evidence of an intracranial complication (Figures 1 and 2). Routine blood and urine investigations and chest X-ray were normal. An enzyme-linked immunosorbent assay (ELISA) for human immunodeficiency virus was non-reactive.

The patient responded promptly to treatment, with complete recovery of facial functions within 10 days. Steroids were tapered over the next 10 days and antibiotics were continued for six weeks, following which the patient was discharged. On discharge, the patient had a dry left ear with a small anteroinferior perforation.

During follow up, the patient continued to have dull retro-orbital pain and, one month following discharge, he presented to us again with ear discharge and retro-orbital pain of increased severity.

From the Departments of ENT & Head and Neck Surgery, and *Radiology, Maulana Azad Medical College and associated L N Hospital, New Delhi, India.
Accepted for publication: 18 November 2005.

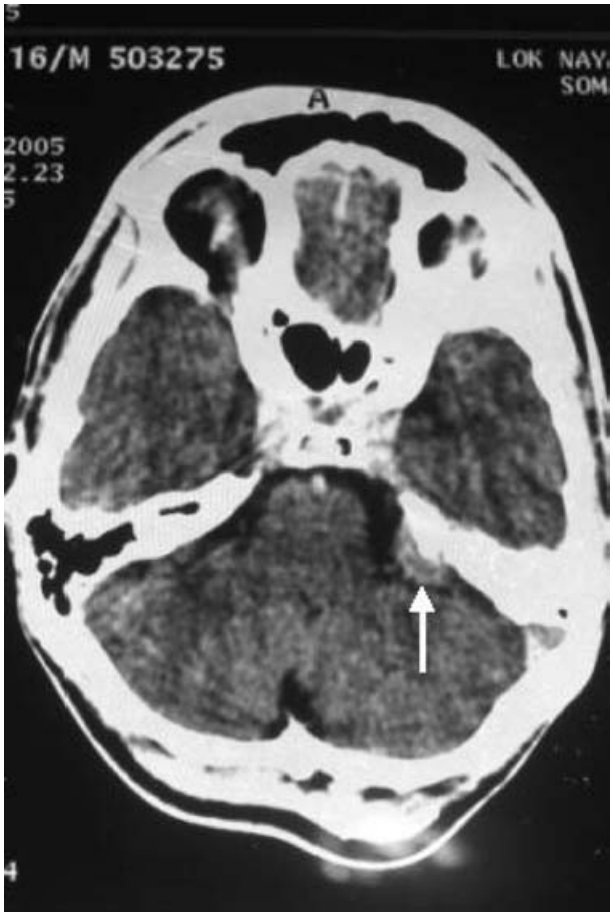

FIG. 1
Computed tomography scan (axial cut) showing an inflammatory lesion in the left petrous apex (white arrow).

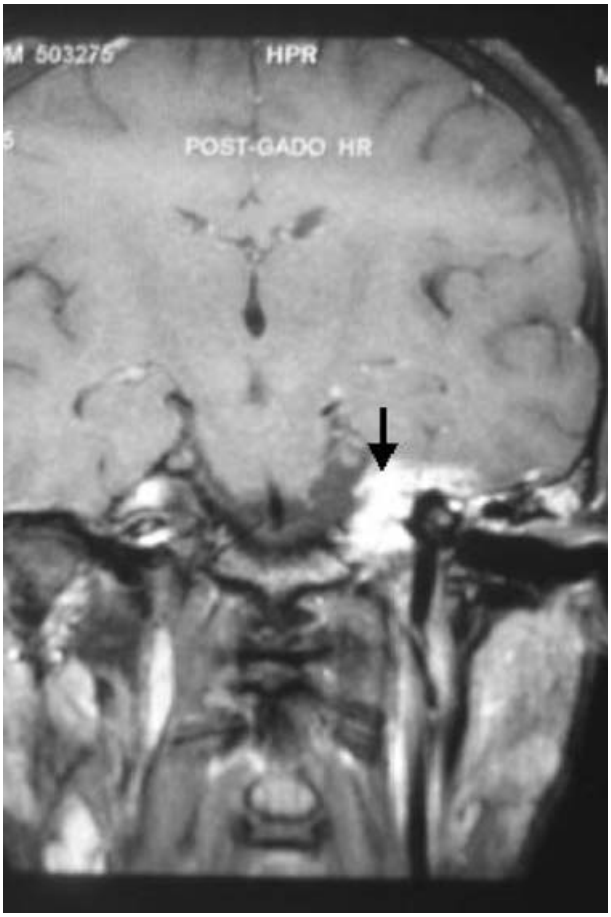

FIG. 2
Magnetic resonance imaging (coronal cut) showing inflammation of the left petrous apex (black arrow).

Ear examination revealed a small anteroinferior perforation with mucoid discharge. The discharge was collected on a sterile swab and sent for acid-fast bacilli (AFB) staining and culture. A multiplanar MRI scan was taken, which revealed an inflammatory lesion of the left petrous apex, with a nodular lesion showing thick peripheral enhancement in the left cerebellar hemisphere (Figures 3 and 4). The possibility of tuberculosis was suggested by the radiologists. Another chest X-ray was taken, which was normal. A Mantoux test gave a 10 mm result and the erythrocyte sedimentation rate was 24 mm in the first hour. The ear discharge stained positive for AFB. A serum ELISA for anti-mycobacterial immunoglobulin M was performed and was found to be reactive at 1:200 dilutions.

The patient was commenced on four-drug antitubercular chemotherapy in the form of isoniazid, rifampicin, ethambutol and pyrazinamide. The patient responded well to treatment and was disease free (radiologically and symptomatically) eight months following treatment.

Discussion

Like the majority of infections, the incidence of acute and chronic petrous apicitis decreased markedly in the post-antibiotic era.[8] As a result, the experience of current otologists with this condition is relatively limited. Most of the cases of petrous apicitis are as a result of pyogenic infections, with a negligible incidence of tubercular petrous apicitis.[3,5,6] It is for this reason that tuberculosis is not considered routinely in the differential diagnosis of petrous apicitis and is liable to be misdiagnosed.

Tubercular petrous apicitis is usually secondary to pulmonary involvement.[3] However, our patient seems to have developed primary tubercular petrous apicitis, as no other focus of tuberculosis was identified. Tubercular otitis usually presents with scant, painless otorrhoea and

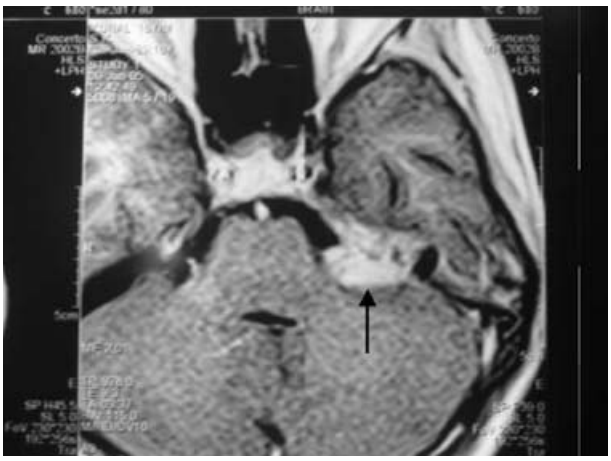

FIG. 3
Magnetic resonance imaging (axial cut, fluid attenuated inversion recovery image) showing an irregular, enhancing lesion in the left petrous apex.

https://doi.org/10.1017/S0022215106000843 Published online by Cambridge University Press

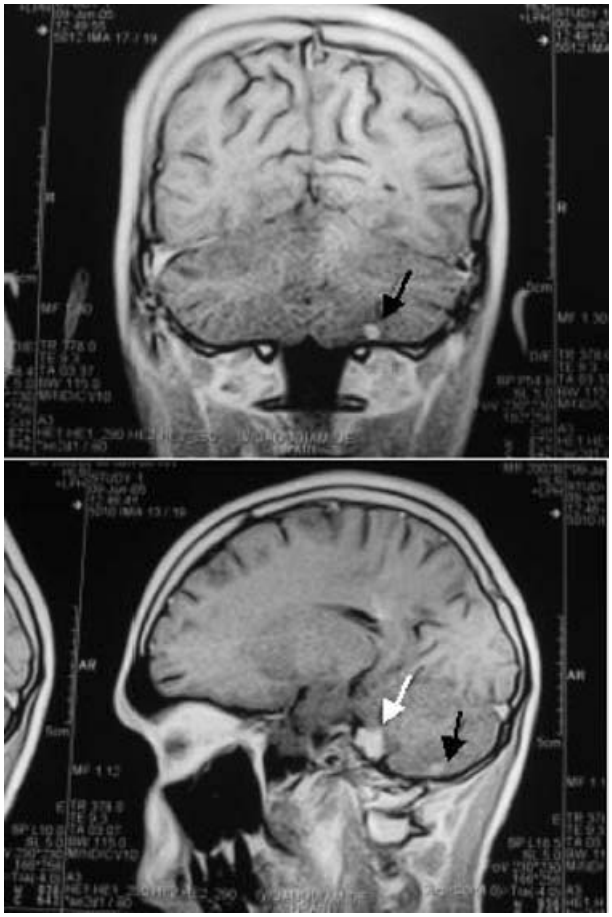

FIG. 4

Magnetic resonance imaging (coronal and sagittal cuts) showing the petrous apex inflammatory lesion (white arrow) and a ring-enhancing nodular lesion in the ipsilateral cerebellar hemisphere (black arrow).

a mild to moderate conductive hearing loss.[3,9] Our patient gave a history of episodes of recurrent otorrhoea and was found to have a mild conductive hearing loss. However, the patient did not present to us with otorrhoea; rather, he presented with acute otitis media and facial paresis. Facial paralysis may arise as a result of acute as well as tubercular otitis media.[10,11] In our case, the facial palsy was attributable to acute otitis media as it responded promptly to broad-spectrum antibiotics and steroids. Our patient also presented with retro-orbital pain, a feature common to both acute and chronic petrous apicitis.[8,12] In our patient, the retro-orbital pain was initially considered to be as a result of acute petrous apicitis due to the presence of coexistent signs and symptoms suggestive of this condition. However, the persistence of the pain in spite of resolution of the acute otitis media suggested the presence of a chronic, non-pyogenic inflammation of the petrous apex that was initially masked by an acute suppurative process.

The diagnosis of petrous apicitis was confirmed on the basis of CT and MRI of the temporal bone in our patient. An MRI scan was done in our case as it is considered to be a better imaging modality for the petrous apex. However, radiological imaging is not useful in differentiating tuberculous from acute pyogenic petrous apicitis.[7] The most reliable means of diagnosis is histopathological evaluation of the tissue from the infected ear.[13] In our patient, no tissue was obtained from the affected ear as the patient presented to us with acute otitis media, and, as a result, the possibility of tuberculosis was not considered. Moreover, physical examination revealed no pathological tissue (i.e. granulations or polyps) that could be sent for histological analysis.

- **Tubercular petrous apicitis is an extremely uncommon entity that may pose a significant diagnostic challenge, requiring a high index of clinical suspicion**
- **An acute pyogenic petrous apicitis may coexist with a tubercular petrositis, thus lowering the index of clinical suspicion and making the diagnosis of the latter more difficult**
- **However, an acute pyogenic process of the petrous apex not responding completely to conventional broad-spectrum antibiotics must alert the clinician to the possibility of a coexistent, underlying tuberculous infection**

The possibility of diagnosing AFB in the ear discharge is less than 20 per cent in ears with tubercular otitis media.[14] In our patient, the ear discharge showed positive staining for AFB. However, the culture for mycobacterium showed no growth. The newer tests available for diagnosis include serum ELISA and polymerase chain reaction (PCR) of the ear discharge.[15,16] Serum ELISA for *Mycobacterium tuberculosis* was reactive in our patient. Due to the unavailability of PCR as a routine diagnostic test in our institute, it could not be performed. Thus, the diagnosis in our case was established on the basis of detection of AFB in the ear discharge and a reactive serum ELISA. It was confirmed by a complete response to anti-tubercular chemotherapy.

The management of tubercular infection of the temporal bone is primarily medical, in the form of anti-tubercular chemotherapy, with surgery reserved for the management of subperiosteal abscess and the removal of sequestrum.[13] Our patient was managed medically.

References

1 Martin JP. Apical petrositis. Report of a case. *N Z Med J* 1967;**66**:10–13
2 Kangsanarak J, Fooanant S, Ruckphaopunt K, Navacharoen N, Teotrakul S. Extracranial and intracranial complications of suppurative otitis media. Report of 102 cases. *J Laryngol Otol* 1993;**107**:999–1004
3 Kearns DB, Coker NJ, Pitcock JK, Jenkins HA. Tuberculous petrous apicitis. *Arch Otolaryngol* 1985;**111**:406–8
4 Ludman H. Complications of suppurative otitis media. In: Kerr AG, Grones J, eds. *Scott-Brown's Otolaryngology*, 5th edn. London: Butterworth, 1987;264–91
5 Al-Ammar AY. Recurrent temporal petrositis. *J Laryngol Otol* 2001;**115**:316–18
6 Saunders NC, Albert DM. Tuberculous mastoiditis: when is surgery indicated? *Int J Pediatr Otolaryngol* 2002;**65**:59–63
7 Palacios E, Rojas R, Ramirez MG. Apical petrositis. *Ear Nose Throat J* 2004;**83**(6):386
8 Chole RA, Donald PJ. Petrous apicitis. Clinical considerations. *Ann Otol Rhinol Laryngol* 1983;**92**:544–51
9 Lucente F, Tobias G, Parisier SC, Som PM. Tuberculous otitis media. *Laryngoscope* 1978;**88**:1107–16
10 Turner AL, Fraser JS. Report for the year 1914 from the Ear, Nose and Throat Department of the Royal Infirmatory, Edinburgh: III. Tuberculosis of the middle ear cleft in children: a clinical and pathological study. *J Laryngol Rhinol Otol* 1915;**30**:209–47

https://doi.org/10.1017/S0022215106000843 Published online by Cambridge University Press

11 Popovtzer A, Raveh E, Bahar G, Oestreicher-Kedem Y, Feinmesser R, Nageris BI. Facial palsy associated with acute otitis media. *Otolaryngol Head Neck Surg* 2005; **132**:327–9
12 Minotti AM, Kountakis SE. Management of abducens palsy in patients with petrositis. *Ann Otol Rhinol Laryngol* 1999;**108**:897–902
13 Singh B. Role of surgery in tuberculous mastoiditis. *J Laryngol Otol* 1991;**105**:907–15
14 Yaniv E. Tuberculous otitis media: a clinical record. *Laryngoscope* 1987;**97**:1303–6
15 Mathai E, Rajkumari R, Kuruvilla PJ, Kirubakaran H, Brahmadithan KN, Imbamalar U. Evaluation of serological tests for the diagnosis of tuberculosis. *Indian J Pathol Microbiol* 2002;**45**:303–5
16 Inoue T, Ikeda N, Kurusawa T, Sato A, Nakatani K, Ikeda T *et al*. A case of middle ear tuberculosis: PCR of the otorrhoea was useful for the diagnosis. *Kekkaku* 1999;**74**:453–6

Address for correspondence:
Dr Ashwani Sethi,
E-80, Naraina Vihar,
New Delhi 110028, India.

E-mail: dr_sethi@rediffmail.com

Dr A Sethi takes responsibility for the integrity of the content of the paper.
Competing interests: None declared