The Journal of Laryngology & Otology (2007), ***121***, *897–901*.

doi:10.1017/S0022215107006032
Printed in the United Kingdom
First published online 26 February 2007

Clinical Record

Role of surgery in the management of otogenic meningitis

Y Slovik, M Kraus, A Leiberman, D M Kaplan

Abstract

Meningitis is a life-threatening complication of otitis media. The appropriate management and the role of surgical intervention are still controversial, and there are no evidence-based guidelines in this regard.

We report three cases of otogenic meningitis, initially treated with parenteral antibiotics and myringotomy, followed by surgery. Two patients had an emergency mastoidectomy and one patient underwent surgery one month post-recovery due to the suspicion of bone erosion on a computed tomography scan. In two cases, a canal wall up procedure was performed, and one patient underwent revision of a radical mastoidectomy. In all cases, no pus or granulations were seen in the mastoid. Two patients fully recovered and one patient died.

We review the literature and critically discuss the role, timing and preferred type of surgery for otogenic meningitis.

Key words: Meningitis; Otitis Media; Antibacterial Agents; Tomography, Computed Xray

Introduction

Acute bacterial meningitis remains a serious disease with high morbidity and mortality rates.[1] Otitis media is a common predisposing factor for acute bacterial meningitis, being responsible for up to 25 per cent of cases.[2] Acute otitis media and chronic otitis media are found with equal frequency among cases of otogenic meningitis,[3,4] and early diagnosis and treatment of the otologic infection is a key determinant of prognosis.[1] The accepted treatment of otogenic meningitis comprises parenteral antibiotics and surgical drainage.[3–9] However, the timing and the type of surgery are controversial.[3–10] Some authors advocate urgent mastoidectomy, while others postulate that myringotomy is the only surgical procedure required.[10]

We present three cases of otogenic meningitis which were surgically treated in our department between January 2003 and February 2004. While all patients underwent a mastoidectomy, the rationale and timing differed for each. The role, timing and extent of surgery are discussed.

Case reports

Case one

A 52-year-old man was admitted to our hospital in an agitated state with severe headache, vomiting and left otalgia. Relatives reported that the patient had complained of otalgia without fever for the previous 24 hours. Twelve hours prior to his presentation, the patient had been diagnosed with acute otitis media by his general practitioner; oral antibiotic treatment (amoxicillin 1.5 g/24 hours, divided into three doses) was initiated but the patient took only one dose. There was no prior history of ear disease and the patient was otherwise healthy.

On arrival, the patient's body temperature was 38.2°C, heart rate was 104 bpm and blood pressure was 105/60 mmHg. The initial physical examination showed neck stiffness, and otoscopy revealed an opaque, bulging left tympanic membrane. The patient's white blood cell count (WBC) was 15.7 10^3 cells/mm^3 (88 per cent neutrophils, 8 per cent lymphocytes). Due to the patient's agitated state, he was intubated.

Cranial and temporal bone computed tomography (CT) were performed, demonstrating sclerotic changes and fullness of the left mastoid air cells. Air bubbles were seen near the left petrous bone at the suprasellar cisterna and along the tentorium (Figure 1). Effacement of temporoparietal sulci was seen, with no evidence of intracranial abscess.

Cerebrospinal fluid (CSF) analysis showed a protein level of 861 mg/dl, a glucose concentration of 47 mg/dl and a WBC of 5860 cells/mm^3 (90 per cent granulocytes). Gram staining showed multiple leukocytes, with no evidence of bacteria.

Tympanocentesis was performed, with drainage of pus, and the culture was positive for β-lactamic sensitive *S. pneumoniae*.

Treatment was initiated with intravenous dexamethasone 1 mg/kg and vancomycin 1 g, thrice daily. Cerebrospinal fluid cultures were positive for β-lactamic sensitive *S. pneumoniae*, and vancomycin was replaced with ceftriaxone 2 g, twice daily. Local treatment to the left ear included repeated microsuction and application of 2 per cent boric acid drops.

Twenty-four hours after commencing treatment, the patient's condition improved, with temperature and WBC count returning to normal values, and he was extubated after three days. On the fifth day, the cranial and temporal bone CT scans were repeated, demonstrating normal brain tissue, opacity of the left mastoid air cells and suspected bone destruction of the posterior plate of the temporal bone (Figure 2).

The patient recovered fully and was discharged from hospital after seven days. At one month follow up, his physical examination and audiometry results were completely normal. The patient underwent elective canal wall up

From the Department of Otolaryngology – Head and Neck Surgery, Soroka University Medical Center, Beer Sheva, Israel.
Presented at the Annual Meeting of the Israeli Otolaryngology – Head and Neck Surgery Society, 17 March 2005, Eilat, Israel.
Accepted for publication: 30 October 2006.

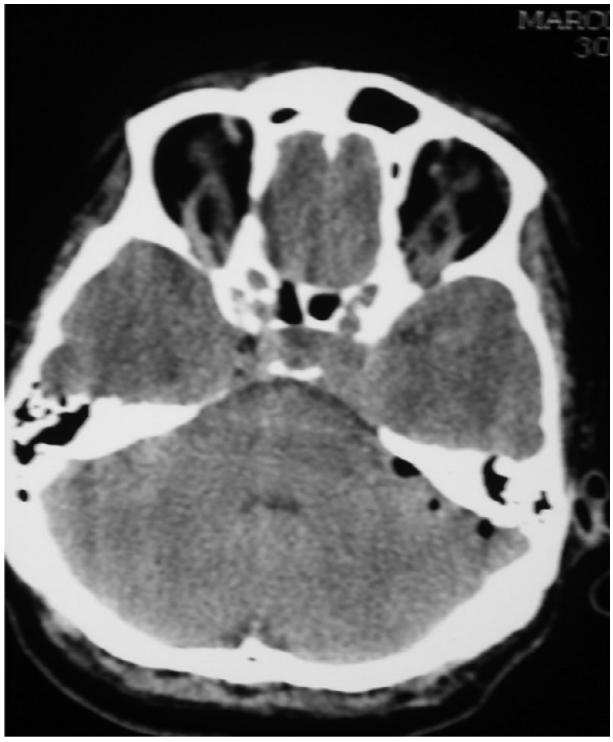

FIG. 1

Case one. Cranial computed tomography scan performed on admission, demonstrating effacement of temporoparietal sulci, with air bubbles near the left petrous bone, near the suprasellar cisterna and along the tentorium.

mastoidectomy. The mastoid cavity was sclerotic, and only minimal granulation tissue was found. The bony plate of the posterior fossa was intact, with no bone destruction. At 18 month follow up, the patient was well.

Case two

A 79-year-old man was admitted to our hospital with confusion, fever and vomiting. Two days previously, he had been seen by his general practitioner due to an exacerbation of his chronic obstructive lung disease and left-sided otalgia. He had been treated with oral roxithromycin 150 mg, twice a day, as well as systemic and local analgesia. The patient's previous medical history was significant for diabetes mellitus, chronic obstructive lung disease and arterial hypertension.

On admission, the patient was confused, with a body temperature of 38.8°C, pulse rate of 76 bpm and blood pressure of 146/69 mmHg. The initial physical examination showed positive meningeal signs, and the left tympanic membrane was hyperaemic on otoscopy.

A bedside tympanocentesis was performed, with drainage of mucoid fluid. The cultures were positive for *Moraxella catharalis* and β-lactamic sensitive *S. pneumoniae*.

A cranial and temporal bone CT revealed fullness of the left mastoid air cells, with air bubbles near the middle cranial fossa (Figure 3).

On lumbar puncture, the CSF was yellowish and cloudy, and analysis showed a protein level of 441 mg/dl, a glucose concentration of 55 mg/dl and three cells/mm^3. Gram staining showed multiple leukocytes, with no evidence of bacteria.

The patient was admitted and treated with intravenous ceftriaxone 4 g/day. Twenty hours later, his neurological status deteriorated and he became comatose. The patient was intubated and transferred to the intensive care unit.

Due to the patient's deterioration, surgery was delayed until approximately 40 hours after presentation. A canal wall up mastoidectomy was performed and a ventilation tube was inserted into the tympanic membrane. At surgery, minimal serous discharge was found, with no evidence of cholesteatoma, granulations or bone erosion.

One day post-surgery, the patient was successfully extubated and made a dramatic recovery. Blood culture was positive for *S. pneumoniae* and enterococcus. The CSF culture was positive for *S. pneumoniae.* The patient was discharged from hospital after 10 days. At one month follow up, the ear and mastoid cells were well aerated and the ventilation tube was in place. There was no neurologic deficit. At 20 months follow up, the patient was well.

Case three

A 43-year-old Bedouin Arab woman was admitted to our hospital due to severe vertigo, left tinnitus, nausea and

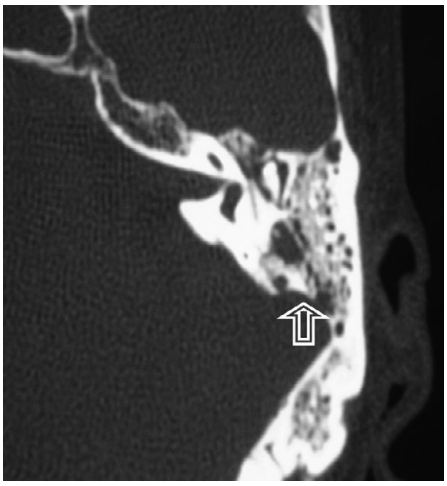

FIG. 2

Case one. Delayed high resolution temporal bone computed tomography, demonstrating left mastoid fullness with suspected bone destruction at the posterior plate of pyramid near the rostrum (arrow).

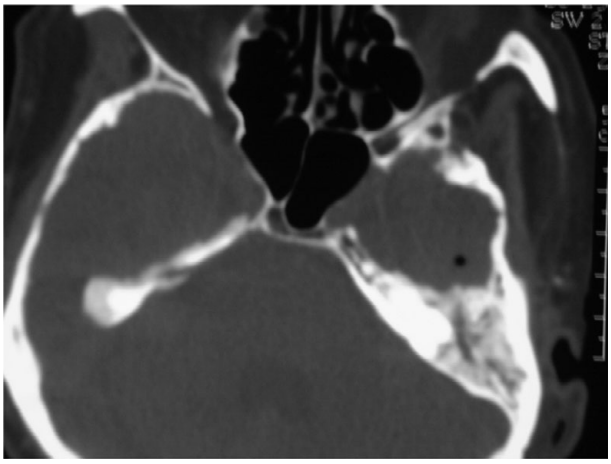

FIG. 3

Case two. Temporal bone computed tomography performed on admission, demonstrating fullness of left mastoid cells, with petrous bone destruction.

https://doi.org/10.1017/S0022215107006032 Published online by Cambridge University Press

vomiting, which had begun several hours prior to admission. Twenty-five years previously, she had undergone bilateral mastoidectomy due to chronic suppurative otitis media without cholesteatoma. She had not been followed up in recent years.

On admission, the patient's body temperature was 37.1°C, her pulse rate was 98 bpm and her blood pressure was 123/75 mmHg. The physical examination did not reveal meningeal signs. On otoscopy, an obliterated left external canal was seen, with discharge from a retroauricular fistula. The patient had spontaneous, right-beating horizontal nystagmus with a slight rotatory component, and her gait was wide. The WBC was 13.1×10^3 cells/mm^3.

The following night, the patient developed a fever of 39.1°C, with no other changes in physical examination findings. Parenteral antibiotic treatment with cefuroxime 750 mg, thrice daily, was commenced. The next morning, the patient became confused and displayed a degree of neck stiffness.

On the basis of clinical symptoms, a cerebral and temporal bone CT was performed, revealing fullness of the left mastoid cavity, with no intracranial abnormality. During the CT scan, the neurologic status of the patient deteriorated and she became comatose.

The patient was intubated and transferred to the intensive care unit. On lumbar puncture, cloudy fluid with elevated pressure was obtained. The CSF contained 5120 cells/mm^3, with a glucose concentration of 61 mg/dl (plasma glucose was 82 mg/dl) and a protein concentration of 351 mg/dl. The patient was taken to the operating theatre one hour later, and revision radical mastoidectomy was performed on the left side. During surgery, limited granulations were found in the mastoid cavity, with no evidence of an intralabyrinthine fistula. Cultures of CSF and blood and from the mastoid cavity were all positive for β-lactamic sensitive *S. pneumoniae*.

Following surgery, there was no change in the patient's comatose state. Her pupils became dilated and she developed syndrome of inappropriate anti diuretic hormone secretion, and she was diagnosed as brain dead. The patient died three days later.

Discussion

Incidence of otogenic meningitis

While the overall incidence of central nervous system complications of infectious ear disease has dramatically declined during the antibiotic era in terms of absolute numbers, the relative distribution of these complications remains similar to that in the pre-antibiotic era.[10] The commonest intracranial complication of otitis media is acute bacterial meningitis, followed by lateral sinus thrombosis.

Middle-ear infection has been reported as the source of acute bacterial meningitis in 20–25 per cent of adult cases.[1,2] This high prevalence justifies investigation for this potential source in every case of acute bacterial meningitis. In the pre-antibiotic era, the mortality rate from intracranial complications of ear disease was in the order of 80 per cent. Accordingly, the standard recommended treatment included performing a radical mastoidectomy within the first 24 hours.[8] With the advent of antibiotics, the recovery rate from otogenic meningitis improved dramatically. Nowadays, the mortality rate for otogenic meningitis is 5–10 per cent.[5,7,10] Thus, the role of surgical drainage and the timing of the procedure have now became controversial.

We present three cases of otogenic meningitis in adults, in which surgical intervention had a major role in management. Below, we review several important aspects of management of such patients, and we attempt to summarise a rational approach to this condition.

Immune status and otogenic meningitis

Barry *et al.*[3] found that six out of 79 adults with otogenic meningitis had diabetes mellitus, with a slight predominance among patients with acute otitis media. Felisati *et al.*[4] presented three patients with otogenic meningitis, of whom two were immunocompromised – one due to hepatitis C infection with liver cirrhosis and the other due to pregnancy. Both these patients made a full recovery.

In our report, two of the three patients were immunocompetent, while one (case three) had diabetes mellitus type two, with no target organ failure. Although one cannot be certain, it is quite possible that the underlying diabetes mellitus type two in this patient contributed to the disease pathogenesis, the delayed diagnosis and the tragic outcome.

Pathogenesis of otogenic meningitis

A suppurative process in the ear may access the central nervous system by either direct or indirect pathways. These infection pathways may be seen in both acute otitis media and chronic otitis media. Direct spread occurs via normal anatomical pathways (e.g. the round and oval windows) or via areas of bone erosion. The latter is most commonly caused by chronic infection, such as granulations and cholesteatoma. Acute infection may also spread directly or haematogenously, due to systemic bacteraemia, or by thrombosis of the emissary veins of the dura.[11]

Gower and McGuirt[10] reviewed 76 patients with otogenic meningitis, and found that all paediatric cases were secondary to acute otitis media, while in the adult group acute otitis media was slightly more common than chronic otitis media. Barry *et al.*[3] reviewed 79 adults with otogenic meningitis. Among the patients with chronic otitis media, there were similar numbers of patients with and without cholesteatoma. Similar results were obtained by Rupa and Raman.[12] In contrast, Kangsanarak *et al.*[7] reviewed a total of 43 patients with otogenic meningitis; 42 had chronic otitis media and almost all had cholesteatoma. In the present study, we report two patients with acute bacterial meningitis following acute otitis media and one patient with a history of chronic otitis media and an occluded ear canal. In none of these patients was there a clear path of local spread. In case three, we assume that, in the absence of an intralabyrinthine fistula, the round or oval window may have been involved, accounting for the patient's vestibular symptoms.

Otogenic meningitis with a normal tympanic membrane

Felisati *et al.*[4] reported three cases of otogenic meningitis with a normal tympanic membrane. At surgery, the mastoid was found to be filled with pus in all cases, but the middle ear appeared normal. The authors postulated that the antrum had become obstructed by mucosal hypertrophy and the middle ear remained free of disease. The implication of otogenic meningitis with an apparently normal tympanic membrane is that local drainage by myringotomy may have a limited effect, and that delivering antibiotics and performing a mastoidectomy may be more important. In our two patients with acute otitis media, the tympanic membrane was intact but with signs of middle-ear suppuration, so initial myringotomy may have played an important factor in recovery.

https://doi.org/10.1017/S0022215107006032 Published online by Cambridge University Press

Bacteriology

Beta-lactamic sensitive *S. pneumoniae* has previously been reported as the leading causative pathogen in acute bacterial meningitis in adults, accounting for approximately half of isolates cultured from the CSF.[1,2] Although *Haemophilus influenzae* type B was previously the most common pathogen,[10] it has now been mostly eliminated in the western world since the introduction of vaccination.[1] *Streptococcus pneumoniae* was also found to be the leading pathogen in patients with otogenic meningitis caused by acute otitis media. *Proteus mirabilis* and klebsiella are the leading pathogens in cases of otogenic meningitis caused by chronic otitis media.[3,7]

In the present report, β-lactamic sensitive *S. pneumoniae* was cultured in all three cases. In two cases, the same pathogen was detected in the CSF and ear cultures. In one case, the CSF culture was sterile. Interestingly, according to Barry and colleagues' review of previous studies,[3] the same pathogen was recovered from both the ear and the CSF in only 17 per cent of otogenic meningitis cases.

Role of imaging

While a cranial CT is commonly performed in the immediate investigation of meningitis, prior to a lumbar puncture, this imaging modality is not useful in delineating middle-ear disease. High resolution temporal bone CT and a post-contract scan of venous blood flow can demonstrate middle-ear disease, as well as rule out thrombosis of the sigmoid sinus.[11] However, in the emergency scenario, high resolution temporal bone CT may not always be practical. Therefore, the imaging is usually not a major determinant in deciding whether, when and how to operate on these patients. High resolution temporal bone CT is indicated in both acute otitis media and chronic otitis media at a later stage, to determine whether reconstructive ear surgery is needed in order to prevent future acute bacterial meningitis development via eroded bone.

Role and timing of surgical treatment

The role of surgical treatment in otogenic meningitis has dramatically changed with the advent of modern antimicrobial agents. House[13] demonstrated that the recovery rate for meningitis improved from 10.6 to 85.7 per cent after the introduction of antibiotics. Based on this data, Singh and Maharaj[8] attempted a more conservative surgical approach. They reserved radical mastoidectomy for cases of cholesteatomatous chronic otitis media; all other cases underwent cortical mastoidectomy. These authors found similar death rates for patients in these two groups. Canal wall up mastoidectomy was found to be a sufficient strategy for controlling otogenic meningitis, according to Felisati *et al.*,[4] Samuel *et al.*,[5] and Samuel and Fernandes.[9] Based on these data, it seems reasonable to perform urgent canal wall up mastoidectomy in any case of non-cholesteatomatous ear disease, and to reserve radical mastoidectomy for patients with cholesteatoma.

The rationale for urgent mastoidectomy, as explained by Samuel *et al.*[5] and Samuel and Fernandes,[9] is that the mastoid cavity is the source of the organisms invading the meninges, and that eradicating it thus prevents further seeding of the CSF and promotes rapid recovery. In contrast, Gower and McGuirt[10] initially treated their 76 otogenic meningitis patients with parenteral antibiotics alone. Only four of these patients failed treatment and required surgical drainage. For two patients, the only surgical procedure performed was myringotomy. Barry *et al.*[3] recommended initial treatment with antibiotics and myringotomy alone. In their view, urgent mastoidectomy should be reserved for cases of neurological deterioration or lack of improvement after 48 hours of drainage and antimicrobial treatment. However, in this series, recurrent meningitis developed in four of 13 patients undergoing canal wall up mastoidectomy, suggesting that a non-aggressive initial approach may not be entirely riskfree.

Two of our patients were initially treated with parenteral broad spectrum antibiotics and myringotomy. One patient (case one) responded to antibiotics and underwent electively surgery one month later due to suspected bone erosion of the mastoid. Another patient (case two) continued to deteriorate under similar treatment, and therefore underwent surgery within the first 48 hours. During surgery for these two cases, the minimal tympanic cavity and mastoid changes seen led us to perform a canal wall up mastoidectomy. The third patient (case three) also underwent emergency mastoidectomy, including exploration of her obliterated radical cavity; again, no pus or recent bone erosion was seen. This patient suffered a fulminant course and probably died because of the delay in diagnosis and in antimicrobial treatment, rather than because of a delay in, or failure of, surgery. The rationale for surgery, in this patient's case, was the dramatic deterioration in her condition; the additional risk of the procedure was thought to be comparatively minimal, given her condition.

Case one underwent elective surgery one month after his emergency admission, but he probably received little benefit from the procedure, as no evidence of bone erosion was seen during the procedure. This over-diagnosis of bone dehiscence on CT scan has been previously reported,[14] and this possibility should always be considered by the surgeon. Case two showed a dramatic improvement and full recovery following surgery. However, as the middle-ear and mastoid findings were so subtle, it is quite possible that recovery would have occurred regardless of this intervention.

The studies we reviewed were all retrospective and thus could not be used to determine whether performing a mastoidectomy was beneficial to the outcome of otogenic meningitis. Furthermore, these studies do not assist in defining clear indications for surgical intervention or the type of surgery warranted.

- **Antimicrobial therapy has drastically changed the prognosis of otitic meningitis. As a result, the role of surgery in the management of otitic meningitis may have become limited**
- **Surgery is indicated only in patients who fail to respond adequately to antimicrobial therapy in the first 48 hours**
- **High resolution temporal bone computed tomography is recommended in cases of conservatively managed patients with clinical suspicion of eroded bone, to determine whether reconstructive ear surgery is required**

Conclusion

Antimicrobial therapy has been the main factor in the improved survival of patients with acute bacterial meningitis. Early evaluation by an otolaryngologist is essential in every case of meningitis in order to rule out an otologic as well as a paranasal sinus source. When ear disease is suspected, myringotomy with culture should be performed. Surgery is indicated only in the limited number of patients

https://doi.org/10.1017/S0022215107006032 Published online by Cambridge University Press

who fail to respond adequately to the initial treatment with myringotomy and antimicrobial therapy within the first 48 hours. All cases of Omen, whether managed conservatively or surgically, require adequate follow up. A delayed high resolution temporal bone CT scan should be performed in cases in which there is suspicion of defects that may predispose to recurrent infection. In such cases, definitive treatment, including mastoidectomy with reconstruction of the defect, should be undertaken as soon as possible.

References

1 Van de beek D, de Gans J, Spanjaard L, Weisfelt M, Reistma JB, Vermeulen M. Clinical features and prognostic factors in adults with bacterial meningitis. *N Engl J Med* 2004;**351**:1849–59
2 Ayaz C, Mehmet FG, Hosoglu S, Mustafa KC, Akalin S, Omer FK. Characteristics of acute bacterial meningitis in southeast Turkey. *Indian J Med Sci* 2004;**58**:327–33
3 Barry B, Delattre J, Vie F, Bedos JP, Ge'hanno P. Otogenic intracranial infections in adults. *Laryngoscope* 1999;**109**:483–7
4 Felisati G, Di Berardino F, Maccari A, Sambataro G. Rapid evolution of acute mastoiditis: three case reports of otogenic meningitis in adults. *Am J Otolaryngol* 2004; **25**:442–6
5 Samuel J, Fernandes CMC, Steinberg JL. Intracranial otogenic complications: a persisting problem. *Laryngoscope* 1986;**96**:272–8
6 Tarantino V, D'agostino R, Taborelli G, Melagrana A, Porcu A, Stura M. Acute mastoiditis: a 10 year retrospective study. *Int J Pediatr Otorhinolaryngol* 2002;**66**:143–8
7 Kangsanarak J, Navacharoen N, Fooanant S, Ruckphaopunt K. Intracranial complications of suppurative otitis media: a 13 years' experience. *Am J Otol* 1995;**16**:104–9
8 Singh B, Maharaj TJM. Radical mastoidectomy: its place in otitic intracranial complications. *J Laryngol Otol* 1993;**107**: 1113–18
9 Samuel J, Fernandes CMC. Otogenic complications with an intact tympanic membrane. *Laryngoscope* 1985;**95**: 1387–90
10 Gower D, McGuirt WF. Intracranial complications of acute and chronic infectious ear disease: a problem still with us. *Laryngoscope* 1983;**93**:1028–33
11 Dew LA, Shelton C. Complications of temporal bone infections. In: Cummings CW, Harker C, eds. *Otolaryngology Head Neck Surgery*. Missouri, St. Louis: Mosby-Year Book, 1998;**4**:3047–73
12 Rupa V, Raman R. Chronic suppurative otitis media: complicated versus uncomplicated disease. *Acta Otolaryngol* 1991;**111**:530–5
13 House HP. Otitis media comparative study of results obtained in therapy before and after introduction of sulfonamide compounds. *Arch Otolaryngol* 1946;**43**:371–8
14 Migirov L. Computed tomographic versus surgical findings in complicated acute otomastoiditis. *Ann Otol Rhinol Laryngol* 2003;**112**:675–7

Address for correspondence:
Dr Slovik Youval,
Sansana Mobile post Negev 3,
Israel 85334.

Fax: 972 8 6403037
E-mail: youvals@bgumail.bgu.ac.il

Dr S Youval takes responsibility for the integrity of the content of the paper.
Competing interests: None declared