This case, only the second of bilateral facial palsy in the literature, underscores the tragedy of a fragmented social infrastructure and health care system where multiple factors conspire to ensure an increasing prevalence of tuberculosis (TB).

The patient was first seen at Red Cross Children’s Hospital in September 2001, aged 3 years and 2 months. He was living with his unemployed single-parent mother in an informal settlement. He had bilateral suppurative submandibular lymphadenitis, requiring incision and drainage. Two weeks after discharge, pus swabs revealed a positive culture for Mycobacterium tuberculosis. The local TB clinic was notified, but his mother could not be contacted, having relocated to central Cape Town where she had obtained temporary employment. In March 2002 he was seen at Somerset Hospital, Cape Town, with a suppurative discharge from both ears. Pus swabs again cultured M. tuberculosis. Attempts at contact via the local TB clinic faced the same problems as previously and the follow-up outpatient appointment was not kept. The third contact with the health care system was in May 2002, when his aunt, who had taken over his care, brought him to Red Cross Hospital. He had bilateral profuse suppurative discharges from his ears, discharging neck sinuses, facial palsies, profound conduction deafness, microcytic anaemia and kwashiorkor. There was no evidence of exposure to HIV. Pus swabs from the ears and neck yielded mixed bacterial and M. tuberculosis culture. Computed tomography (CT) of the temporal bones demonstrated extensive destruction of the mastoid bone and ossicular chain bilaterally, consistent with tuberculous mastoiditis (Fig. 1).

Tuberculous involvement of the temporal bone was described early in the 18th century, more than a century before Koch’s isolation of the tubercle bacillus in 1882. Early diagnosis of tuberculous otitis media is dependent on a high index of suspicion, especially in areas of high tuberculosis prevalence. It classically presents with an intractable, antibiotic-resistant, profuse, painless discharge from the ear, which may become muco-purulent as secondary infection supervenes. A large central perforation of the tympanic membrane is usual, but a total perforation or multiple perforations may also be present. The handle of the malleus is often denuded, the middle ear mucoa pale and the middle ear cavity packed with pale, soft granulation tissue, which may protrude through the tympanic membrane perforation. Hearing loss can be profound. Pre-auricular lymphadenopathy is now less common than in the early descriptions of the condition.

Extension to mastoiditis may occur, manifesting as a post-auricular abscess. Facial nerve palsy is a common complication in children, tending to be unilateral, even in bilateral disease, with the incidence inversely related to age. Two otitic aphorisms are: ‘A child with a facial nerve palsy and a discharging ear has tuberculous otitis until disproved’ and ‘A patient with a discharging ear and known tuberculosis at another anatomical site has tuberculous otitis until disproved’.

Bacteriology is unreliable since pus swab microscopy and culture are positive in less than one-fifth of cases, hence the importance of histological diagnosis and repeated biopsies if results are inconclusive.

Antituberculous chemotherapy is the treatment of choice. The role of surgery is limited to biopsy for diagnostic purposes, incision and drainage of a post-auricular abscess, the management of intracranial complications and for removal of bony sequestra.

High-resolution CT is the imaging modality of choice, providing better bony detail than magnetic resonance imaging (MRI). CT allows evaluation of the extent of mastoid air cell and cortical destruction, the presence of extradural extension, integrity of the ossicular chain, facial nerve canal and bony labyrinth.

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