Reminder of important clinical lesson

A 14-year-old girl with tuberculous otitis media and brain abscess

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Summary

Tuberculosis is a rare cause of chronic suppurative otitis media and mastoiditis. Intracranial complications are rare. The authors report a case of a 14-year-old girl with chronic otitis media of her right ear for 2 years. Her presentation was as follows: 10 days before admission she had a high fever, headache, alteration of consciousness and a generalised seizure. CT scanning of her brain showed right otomastoiditis with early brain abscess of the right cerebellum. She underwent right radical mastoidectomy and aspiration of the cerebellar abscess. The pus from the mastoid and cerebellar abscess was stained positive for acid-fast bacilli (AFB). The pus culture grew *Mycobacterium tuberculosis* (MTB). Her discharge diagnosis was tuberculous mastoiditis (TM) with cerebellar abscess. Long-term treatment plans included antituberculous drugs for 1 year.

BACKGROUND

Mycobacterium tuberculosis (MTB) was a common cause of mastoiditis in the first half of the twentieth century, manifesting itself as chronic ear drainage. Recently, this organism has been recovered only rarely from children or adults with long-standing otitis media.¹ In some communities MTB accounts for up to 4% of all cases of otitis media. In such cases the middle ear can become infected through direct transmission from the lungs, larynx, pharynx and nose via the Eustachian tube, or through haematogenous spread from other primary sites. Tuberculous mastoiditis (TM) is usually a complication of unrecognised, and therefore untreated, tuberculous otitis media. Complications associated with infection of the middle ear and mastoiditis caused by MTB include hearing loss, facial palsy, subperiosteal abscess formation, postaural fistula formation and labyrinthitis. Intracranial complications such as meningitis, sigmoid sinus thrombosis, tuberculoma and abscess formation are rare.² There are uncommon case reports of tuberculous otitis media and mastoiditis.^{3–9} This is the first case reported in Thailand and is a reminder of a historically significant infection with the potential to resurface more frequently.

CASE PRESENTATION

A 14-year-old girl was referred to our tertiary medical centre from a provincial hospital with a history of high fever for the last 10 days as well as progressively worsening headaches, altered mental status and generalised seizure activity. She reported having had chronic otitis media of the right ear for the previous 2 years. During the time period prior to hospitalisation she was treated with multiple courses of oral antibiotics for chronic otitis media without resolution of symptoms.

Apart from the chronic ear symptoms detailed, she had no significant medical history. There was no history of

tuberculous contact. Her growth and development were normal for her age and gender. Her immunisation history was unknown.

On physical examination, she was febrile to 38°C with otherwise normal vital signs. Her neurological examination revealed several abnormalities: her Glasgow Coma Score was 11/15 (E3V2M6), her pupils were 3 mm in diameter and reactive to light bilaterally, her left eye deviated medially at rest, fundoscopic examination revealed bilateral papilledema, and there were signs of cerebellar dysfunction, including ataxic swaying during the finger-to-nose test. Additionally, she had yellow discharge from her right ear canal. Lymph nodes of her head and neck were not palpable. Other aspects of physical examination were within normal limits.

INVESTIGATIONS

Initial laboratory studies showed a white blood cell count of 11.1×10^9 cells/l (92.1% neutrophils, 5.4% lymphocytes, 2.3% monocytes, 0% eosinophil and 0.2% basophil), a haemoglobin level of 10.3 g/dl, and a platelet count of 573×10^9 /l. Electrolyte and liver function panels were within normal limits for her age as were a chest radiograph and a tuberculin skin test.

A CT scan of her brain (figures 1 and 2A) showed a hypodense lesion in the right cerebellar hemisphere, measuring about $3.1 \times 3.4 \times 2.3$ cm. Neurosurgical intervention led to mastoid and brain abscess drainage. Initial staining of the purulent drainage was positive for acid-fast bacilli (AFB) 3+ and grew MTB in culture.

DIFFERENTIAL DIAGNOSIS

TM with right cerebellar abscess.

TREATMENT

Therapy consisted of a right radical mastoidectomy with aspiration of the sigmoid venous sinus and brain abscess.

Figure 1 Hypodense lesion at right cerebellar hemisphere and hyperdensity of air-fluid level in the right mastoid air cell.

She was treated first with a combination of rifampicin, isoniazid, pyrazinamide and ethambutol for 2 months, and then with a combination of rifampicin and isoniazid for 10 months. Additional follow-up included regular clinical reassessment and serial CT scans.

OUTCOME AND FOLLOW-UP

Nine months after the onset of treatment a repeat CT of the patient's brain (figure 2B) showed a complete resolution of the abscess in the right cerebellar hemisphere. Clinically, the neurological deficits noted on her initial physical exam had resolved entirely. She noted no ongoing drainage from her ears and denied any other neurological

impairment. Clinically and by report she appeared to be back to her baseline.

DISCUSSION

The prevalence of MTB is increasing due in part to the rising incidence of immunocompromised hosts (especially those with HIV infections). Although tuberculous otitis media has been shown to be more common in HIV-infected than in HIV-uninfected children, the incidence of it in immunocompetent patients may be increasing. The clinical features of tuberculous ear disease are similar to those of chronic otitis media caused by other microorganisms: long-term discomfort and drainage unresponsive to antibiotic treatment. Clinical features that increase the suspicion for tuberculous otitis media and mastoiditis include facial paralysis associated with chronic suppurative otitis media, a history of pulmonary tuberculosis, suppurative otitis media affecting another anatomical site, pale granulation tissue in the middle ear or in a previously created mastoid cavity and a postauricular abscess.² Diagnosis is made by isolation of AFB either by microscopy or culture from middle ear contents.

More severe complications can include erosion of the roof of the antrum leading to temporal lobe abscess and extension into the lateral sinuses (which can cause septic thromboses).¹⁰ Further complications associated with middle ear and mastoid tuberculosis include hearing loss, facial palsy (especially in children) and intracranial lesions. Early therapy is paramount to avoid complications and is very often associated with a rapid resolution of the infections.²

Treatment consists of systemic antitubercular therapy. Surgical involvement includes biopsy for diagnostic purposes, incision and drainage of abscesses and further management of intracranial complications. With early diagnosis and treatment, outcomes are generally good.^{2 5 11 12} In the case of our patient, a long-term antimicrobial regimen (as outlined above) along with appropriate surgical intervention led to a return to baseline.

Figure 2 (A) Seven days after right mastoidectomy with aspiration the sigmoid venous sinus and brain abscess showed an irregular shaped rim-enhancing hypodense lesion in the right cerebellar hemisphere (3.0×2.8×2.3 cm) with surrounding brain oedema. (B) Complete resolution of the abscess in the right cerebellar hemisphere.

Learning points

- ▶ Tuberculous mastoiditis (TM) and otitis media were a common finding in the first half of the twentieth century, but providers are much less likely to see them today.
- ▶ While TM and otitis media are unusual manifestations of tuberculosis it is important that they be considered in patients with chronic otitis in endemic areas.
- ▶ Early diagnosis and treatment of TM and otitis media result in better outcomes.

Competing interests None.

Patient consent Obtained.

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