Case Report
A Rare Case of Syphilitic Mastoiditis Concomitant with Neurosyphilis
Zhigang Zhao, MD1, Qian Gao, MD1, and Penglong Song, MD2

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Neurosyphilis is an infection of the brain and spinal cord caused by dissemination of Treponema pallidum to the cerebrospinal fluid (CSF) and meninges.1 The infection can occur at any stage of the disease process, and one-third of patients in the tertiary stage develop neurosyphilis. Treponema pallidum causes syphilitic otitis media and mastoiditis usually characterized by severe sensorineural hearing loss and vertigo.2 There are few literature reports on syphilitic otitis media and mastoiditis. The patient in this study had syphilitic mastoiditis concomitant with neurosyphilis, which is the only case reported so far.

Case Report
A previously healthy 34-year-old woman presented with a 3-month history of distending headache. The patient had no history of hearing loss, vertigo, nausea, vomiting, or language disorders. The ear, nose, and throat examination, including nasopharyngeal endoscopy, was normal except for bilateral tympanic membrane retraction. The audiologic evaluation revealed bilateral mild conductive hearing loss and bilateral type C tympanogram (see Supplemental Figure S1 at www.otojournal.org). Computed tomography (CT) revealed clouding of mastoid air cells by soft tissue shadow with a bone defect of mastoid (Figure 1A). Mastoid magnetic resonance imaging (MRI) revealed an isointense lesion (Figure 1B,D). A brain MRI revealed large areas of long T1 and T2 signals in the right temporal lobes, which showed high signal in fluid attenuation inversion recovery (FLAIR) sequences (Figure 2A-C). The thickening of the right temporal cerebral dura, especially that close to the mastoid, was clearly enhanced after injection of gadolinium-diethylenetriamine pentaacetic acid (Figure 1C and Figure 2D). The brain MRI displayed abnormal strengthened signals in both the right temporal cerebral dura and mastoid, as well as the erosive lesion of mastoid extending into the middle cranial fossa; this was due to a bone defect of the mastoid (Figure 1C). A biopsy of the mastoid implied syphilis infection. Both T pallidum hemagglutination assay and rapid plasma reagin tests of serum and CSF were positive (see Supplemental Figure S2 at www.otojournal.org). The patient was finally diagnosed with syphilitic mastoiditis concomitant with neurosyphilis and treated with standard penicillin therapy for 14 days. Thereafter, the patient’s headache was significantly relieved, and the patient is now under follow-up. This

1Department of Otorhinolaryngology/Head and Neck Surgery, The First Affiliated Hospital, Harbin Medical University, Harbin, People’s Republic of China
2Hearing Center of the Otolaryngology Head and Neck Surgery Department, The First Affiliated Hospital, Harbin Medical University, Harbin, People’s Republic of China

Corresponding Author:
Penglong Song, MD, Hearing Center of the Otolaryngology Head and Neck Surgery Department, The First Affiliated Hospital, Harbin Medical University, Harbin 150010, Heilongjiang Province, People’s Republic of China.
Email: s_pl2008@hotmail.com

Figure 1. (A) The large black arrow indicates the bone defect, and the small black arrow indicates Körner’s septum. (B, D) Axial T1 and T2-weighted magnetic resonance imaging (MRI) revealed an isointense shadow in the right mastoid. (C) The coronal contrast-enhanced MRI image. The white arrow indicates line strengthening and thickening dura. The black arrow indicates lesions in the mastoid.
study received approval from the ethical committee of Harbin Medical University.

**Discussion**

Meningeal neurosyphilis usually manifests with the clinical features of acute meningitis, including hydrocephalus, cranial neuropathies, and the formation of leptomeningeal granulomas, called gummas. Our patient’s brain MRI scan revealed abnormally strong signals in both the right temporal cerebral dura mater and mastoid. It also showed an erosive lesion of the right mastoid extending into the temporal cerebral dura mater by a bone defect of hanging wall of mastoid, which was probably attributed to the gummas. This speculation was further confirmed by pathology. For patients with syphilitic meningitis, the cortex is often involved secondary to invasion and direct extension by gummas. The patient had cortical atrophy in the right temporal lobe and long T1, long T2, and high signals on the FLAIR sequence in the subcortical regions. This imaging is consistent with imaging characteristics described in other studies. Generally, a high T2 signal in the temporal lobe represents edema and glial proliferation. In this study, the high signal on the FLAIR sequence excluded edematous temporal lobes; therefore, glial proliferation was a strong consideration. Glial proliferation in subcortical regions is usually secondary to microvascular ischemic changes induced by infection.

Syphilitic otitis media is usually due to a retrograde infection of the nasopharyngeal gumma or ulceration in the tertiary stage. The imaging results indicated that due to blockage of Körner’s septum, the right mastoid had fused without involvement of the tympanic cavity and antrum, which is hardly explained by a retrograde infection. We speculated that the syphilitic mastoiditis was likely to be from a hematogenous infection, and then mastoid gumma led to destruction of the tegmen mastoideum and direct intracranial extension.

The tympanic membrane retraction correlating to a type C tympanogram may cause mild conductive hearing loss by restricting sound-induced vibrations of the eardrum. According to normal results of CT and MRI imaging of the tympanic cavity and nasopharyngeal endoscopy, the bilateral tympanic membrane retraction was more likely produced by eustachian tube dysfunction. The patient did not present with signs and symptoms suggesting the diagnosis of syphilitic otitis media or mastoiditis, such as foul discharge, destruction of the eardrum, and fluctuating hearing loss, which can easily lead to misdiagnosis or missed diagnosis. Although syphilitic mastoiditis concomitant with neurosyphilis is very rare, it is important for otolaryngologists to be aware of this disease process.

**Author Contributions**

Zhigang Zhao, contribution to design, revising, final approval, accountability for all aspects of the work; Qian Gao, contribution to design, revising, final approval, accountability for all aspects of the work; Penglong Song, contribution to design, drafting, final approval, accountability for all aspects of the work.

**Disclosures**

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**Supplemental Material**

Additional supporting information may be found at www.otojournal.org/supplemental.

**References**


