Tympanic Membrane Perforation: An Unusual Complication of Varicella-Zoster Virus Infection

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Otological complications associated with varicella-zoster virus infection are common; however, tympanic membrane involvement is rarely reported. We describe a patient with herpes zoster in whom hemorrhagic otitis media with tympanic membrane perforation developed. To our knowledge, this is the first report of an HIV-infected patient with this unusual presentation. [Infect Med. 2008;25:561-562]

Primary infection with varicella-zoster virus (VZV) causes chickenpox, whereas reactivation of latent VZV causes herpes zoster (also known as shingles), typically a localized infection confined to one dermatome or adjacent dermatomes. Both types of infection are known to have a vast array of presentations and complications among HIV-infected patients.

Case report
A 38-year-old man with stage C3 HIV infection and hepatitis C virus (HCV) coinfection and obstructive sleep apnea presented with a painful rash on the left cheek that had a distribution consistent with the third branch of the left trigeminal nerve. The patient was given famciclovir and gabapentin for management of presumed zoster, which was confirmed by a direct fluorescent antibody test.

Two days later, the man returned to the clinic with left ear pressure, pain, and diminished hearing. Physical examination revealed a temperature of 37°C (98.6°F), a blood pressure of 128/68 mm Hg, and a heart rate of 83 beats per minute. The vesicular lesions that were noted on the left side of the face 2 days earlier were still present and several new lesions had developed on the left portion of his scalp. The patient did not experience visual symptoms and no lesions developed on the tip of the nose. No facial asymmetry or weakness was present. The left tympanic membrane was bulging, and a hemorrhagic middle ear effusion was discovered (Figure).

Findings from an examination of the right ear were unremarkable. An otolaryngologist visualized a normal external auditory canal and a bulging, erythematous tympanic membrane with an effusion that did not appear to be purulent. An audiogram revealed mild conductive hearing loss in the affected ear and normal hearing in the other ear. A tympanogram was not obtained because of the patient's ear pain. Amoxicillin/clavulanic acid was prescribed to prevent secondary infections. The otolaryngologists...
chose noninvasive management, but they planned to reconsider tympanocentesis if symptoms did not improve.

The following day, the patient returned to the clinic complaining that blood had drained from his ear during the night. On examination, a small slit perforation, with scant amounts of drainage, was found in the tympanic membrane. Attempts were made to culture the drainage but insufficient fluid was obtained. No vesicular lesions were noted within the canal.

When the patient returned for follow-up a week later, dramatic improvement was noted, with complete resolution of his ear pain and recovery of his hearing. The physical examination revealed only crusted, healing skin lesions; a normal auditory canal; and a mobile left tympanic membrane with a small slit-like opening. His Weber test result was midline, and his bilateral Rinne test results were positive (normal).

The patient had begun highly active antiretroviral therapy (lamivudine/zidovudine and atazanavir) 5 months earlier, continued it for 2 months, and then interrupted therapy for a month because of elevated transaminase levels, presumably caused by immune reconstitution and HCV infection. He then restarted antiretroviral therapy 8 weeks before zoster and ear symptoms developed. Laboratory tests at the time that antiretroviral therapy was initiated revealed a CD4+ cell count of 29/?L and a plasma HIV-1 RNA level of 549,000 copies/mL. When the patient presented with ear symptoms, his CD4+ cell count was 100/?L and his plasma HIV-1 RNA level was 302 copies/mL.

**Discussion**

This case is notable for several reasons. First, although otological complications associated with varicellazoster virus infections are common, they typically involve the external auditory canal; tympanic membrane involvement is unusual. Second, otic zoster (also known as Ramsay Hunt syndrome, or herpes zoster oticus) typically presents as a polycranial neuritis manifesting as a vesicular facial rash, severe otalgia, inflammation of the pinna, and occasionally unilateral sudden facial paralysis. The rash is often present on the external ear and the anterior two-thirds of the tongue. Other symptoms can include vertigo, tinnitus, hearing loss, loss of taste, decreased salivation or lacrimation, regional lymphadenopathy, and hyperacusis.1,2

When tympanic membrane involvement has been reported, symptoms included dizziness, vertigo, severe sensorineural hearing loss, facial palsy, dysphagia, impaired gag reflex, and vocal cord paralysis.3–5 Our patient had none of these findings, perhaps because he began therapy soon after the development of symptoms consistent with zoster. A review of the clinical presentations of zoster in HIV-infected patients has revealed no cases of tympanic membrane involvement.6,7

It is possible that our patient's otitis media and tympanic membrane perforation were unrelated to the zoster; however, this is unlikely considering the timing, the findings of zoster lesions on the same side of the face and scalp as the unilateral otitis, and the absence of any upper respiratory tract symptoms or other likely cause of the otitis.

This case serves as a reminder that unusual presentations of common infections can occur in HIV-infected patients, especially in the setting of immune restoration.

**References:**


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