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Gout with hearing loss

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Dear Editor,

Gout is regarded as an autoinflammatory disease induced by the deposition of monosodium urate crystals in the synovial fluid and periarticular tissues. It is characterized by joint symptoms and uric acid nephropathy, as well as other organs involved due to inflammation. Here, we presented a rare case of gout with hearing loss.

A 26-year-old Chinese man presented to the Otolaryngology Department with a 1-year history of bilateral ears hearing loss. He had no history of ear injury, fever, rash, headache, nasitis, red eyes, hypertension, and diabetes, and denied exposure to loud noise or ototoxic drugs. Otoscopic examination showed that eardrums were normal. Enhanced magnetic resonance imaging (MRI) scanning detected no abnormalities of inner acoustic meatus or intracranial space-occupying lesion. Auditory brainstem responses (ABR) indicated that the right threshold was increased to 50, and the latency of left V wave and left I–V waves interval were slightly prolonged. Pure tone audiometry (PTA) demonstrated elevated thresholds of 35 dB at 8 kHz in the left ear, while 75 dB and 55 dB at 4 kHz and 8 kHz in the right ear, respectively. Testing for distortion product otoacoustic emissions (DPOAE) found abnormal otoacoustic emissions in the 6–8 kHz frequency of the left ear, and in the 4 kHz and 6–8 kHz frequency of the right ear. He was diagnosed with nerve deafness by the otolaryngologist. Meanwhile, we learned that he had a 3-year history of gout. The right first metatarsophalangeal joint was hurt once; the symptom alleviated voluntarily. Annual follow up showed that serum uric acid increased but without any treatment. Physical examination showed no joint swelling and tenderness, and no ectopic tophus. Laboratory findings showed an increased serum uric acid of 730 μmol/L. Complete blood count, liver and kidney function, C-reactive protein, and erythrocyte sedimentation rate were normal, while anti-nuclear antibodies and anti-neutrophil cytoplasmic antibodies were negative, and renal ultrasound showed no abnormality. Based on the clinical manifestations and laboratory results, he was diagnosed as gout complicated with nerve deafness. After 3 months of allopurinol monotherapy (100 mg, thrice a day), he had no arthritis and the serum uric acid level was decreased to 362 μmol/L. Intriguingly, his symptom of hearing loss was obviously relieved. Meanwhile, the results of ABR follow-up was improved with normalization of waves, but PTA and DPOAE results showed no remarkable changes.

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Hearing loss caused by gout is comparatively rare and the pathogenesis is not completely clear. A recent research had pointed out that gout was associated with a higher risk of hearing loss in the adults aged ≥65 years,[1] which suggested that gout had a close relationship with dysaudia to some degree. Nevertheless, the limitation of the aforementioned study was that the conclusion was only generalizable to the elderly, yet could hardly reveal the characteristics in different age stages.

As we know, joints are the most common deposition sites of gouty tophi. However, there have also been increasing reports of unusual localizations of tophi, such as the middle ear. Mutlu *et al*.[2] reported a gout patient who complained of hearing loss. Otoscopic examination found the perforation of tympanic membrane perforation and white middle ear plaques, which were monosodium urate crystals confirmed by polarized light. A similar case was also reported by Gargula *et al*. [3] These findings indicated that the middle ear was a predilection site of unusual localizations of tophi. It might be one of the pathogenesis factors of hearing loss induced by gout. But unfortunately, this kind of organic lesion was not found in our patient.

Interestingly, pathogenesis of hearing loss in gout has been enriched by current researches that hyperuricemia is closely related to inflammation[4] and oxidative stress.[5] Prasad *et al*.[6] demonstrated that oxidative stress and pro-inflammatory cytokines could influence the expression of microRNAs via mediating their stability and transcription. Since microRNAs are abundantly expressed in the normal inner ear, accordingly, the function of the hair cells would be affected, which ultimately lead to the hearing disorders.[7] Besides, Sung *et al*.[8] proved that spiral ganglion neurons (SGN), synapses, and neurites connecting the cochlear hair cells and SGN were significantly affected under inflammatory conditions. These data together support the view that inflammation and oxidative stress may lead to the inner ear and auditory nerve damage in gout patients. As DPOAE and ABR examination could reflect the function of hair cells and auditory nerve, respectively, the abnormal results in our gout patient suggested that his hearing loss might due to hair cells and auditory nerve damage.

Although the patient's hearing loss improved after treatment for reducing uric acid, and the follow-up ABR results indicated that his auditory nerve injury were recovered with the decrease of uric acid level, we should also point out that there was no significant improvement of PTA and DPOAE. It might be due to the long-term hyperuricemia with no treatment, which resulted in irreversibly damage of the hair cells, or we need to prolong the follow-up further. This thought-provoking case suggested that rare organ damage caused by inflammation should be recognized in gout, the common autoinflammatory disease. Early and appropriate treatment is essential to avoid irreversible complications of gout.

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Patient Informed Consent Declaration

The authors certify that they have obtained all appropriate patient consent documents. In the documents, the patient has given his consent for his images and other clinical information to be published in the journal.

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Conflict of Interest

The authors declare that no conflicts of interest exist.

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References

[1] Singh JA, Cleveland JD. Gout and Hearing Impairment in the Elderly: A Retrospective Cohort Study Using the US Medicare Claims Data. BMJ Open. 2018;8:e022854.

[2] Mutlu A, Dündar E, İşeri M, *et al*. An Unusual Presentation of Gout: Tophi in the Middle Ear. J Int Adv Otol. 2016;12:216–218.

[3] Gargula S, Poillon G, Daval M, *et al*. Tophaceous Gout of the Middle Ear. J Otol. 2019;14:155–157.

[4] Haryono A, Nugrahaningsih DAA, Sari DCR, *et al*. Reduction of Serum Uric Acid Associated with Attenuation of Renal Injury, Inflammation and Macrophages M1/M2 Ratio in Hyperuricemic Mice Model. Kobe J Med Sci. 2018;64:E107–E114.

[5] Glantzounis GK, Tsimoyiannis EC, Kappas AM, *et al*. Uric Acid and Oxidative Stress. Curr Pharm Des. 2005;11:4145–4151.

[6] Prasad KN. Oxidative Stress and Pro-Inflammatory Cytokines May Act as One of the Signals for Regulating microRNAs Expression in Alzheimer's Disease. Mech Ageing Dev. 2017;162:63–71.

[7] Prasad KN, Bondy SC. MicroRNAs in Hearing Disorders: Their Regulation by Oxidative Stress, Inflammation and Antioxidants. Front Cell Neurosci. 2017;11:276.

[8] Sung CYW, Seleme MC, Payne S, *et al*. Virus-induced Cochlear Inflammation in Newborn Mice Alters Auditory Function. JCI Insight. 2019;4:e128878.