Sudden Hearing Loss After Dental Treatment

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A 66-year-old man presented with impaired balance, tinnitus, sensation of blockage, and hearing loss in his left ear, which developed after dental treatment for dental pain 4 days previously. Treatment of the carious left upper second molar tooth had included pulp extirpation, canal expansion, and tooth filling under local anesthesia with articaine and epinephrine. Impaired balance decreased spontaneously within 3 days of dental treatment, but tinnitus and hearing loss persisted. Pure tone audiogram showed profound sensorineural hearing loss in the left ear, with a downslope from 40 to 100 dB, and an abnormal speech discrimination score (50%). Treatment included intravenous prednisolone, intratympanic dexamethasone, and oral betahistine and trimetazidine. The patient had improved hearing and resolution of tinnitus. Sudden hearing loss is rare after dental treatment, and awareness of this complication may prompt early referral for treatment and may improve recovery and prognosis.

The United States National Institute on Deafness and Other Communication Disorders defines sudden hearing loss as a decrease in hearing of at least 30 dB that occurs within 3 days and that affects at least 3 consecutive frequencies of hearing impairment in either ear or both ears.1,3 Sudden hearing loss is frequently associated with tinnitus (52% to 70% of patients), dizziness (≤40% of patients), vertigo (26% of patients), fullness in the ear, and headache.1,4,5 In 10% of patients who have dizziness, the dizziness may be incapacitating and associated with nausea and vomiting. Sudden sensorineural hearing loss affects 5 to 20 of 100,000 people per year in the United States.5,6

The most common causes of sudden hearing loss include infectious, vascular, metabolic, traumatic, neoplastic, postoperative, and miscellaneous causes. Idiopathic sudden hearing loss may occur when no cause is identified.4,5,7 Sudden hearing loss may occur after ear surgery, but is rare after dental treatment.6 Treatment of sudden hearing loss may include systemic and topical steroids, antiviral agents, rheologic agents, diuretics, minerals, vitamins, herbal preparations, hyperbaric oxygen, pharmacologic drugs, middle ear surgery for fistula repair, and observation.1,8

This report describes the case of a man who had sudden hearing loss in the left ear after endodontic treatment. Early evaluation and treatment resulted in improved hearing.

Report of Case

A 66-year-old man on holiday from Norway presented to the otolaryngology department because of impaired balance, tinnitus, sensation of blockage, and hearing loss in his left ear. These symptoms developed after dental treatment 4 days previously for dental pain. Impaired balance decreased spontaneously within 3 days of dental treatment, but tinnitus and hearing loss persisted.

Dental evaluation 4 days previously had shown caries in the left upper second molar tooth. Local anesthesia included articaine with epinephrine, and treatment of the root canal included pulp extirpation, canal expansion, and tooth filling. Drilling was performed with a low-powered surgical drill, and treatment was completed within 30 minutes.

The patient’s medical history was notable for hypertension associated with anger. There was no history of hearing loss or related illness, arterial or venous thrombotic disease, chronic disease, or systemic disorder. There was no family history of hearing loss or related illness.

Physical examination of the head, neck, and ears, including otoscopy, was normal. Blood pressure was
normal. Pure tone audiogram showed profound sensorineural hearing loss in the left ear, with a downslope from 40 to 100 dB, and an abnormal speech discrimination score (50%); the right ear had presbycusis (Fig 1). Blood tests were normal, including complete blood cell count with differential, electrolytes, coagulation profile, liver function tests, erythrocyte sedimentation rate, lipid profile, thyroid function tests, and glucose level. Magnetic resonance imaging of the internal acoustic canal showed no acoustic neuroma or other abnormality.

The patient was admitted to the hospital and immediately given prednisolone (150 mg, intravenous bolus). On the second hospital day, he was given another dose of prednisolone (1 mg/kg, intravenous bolus) and started on betahistine (24 mg orally, 3 times daily) and trimetazidine (20 mg orally, 3 times daily). Blood pressure remained normal on steroid therapy.

There was no decrease of symptoms by the third hospital day. Therefore, the patient was given a left ear intratympanic injection of dexamethasone (8 mg/mL; dose, 0.5 mL) with a 1-mL syringe and 22-gauge needle through the postero-inferior quadrant of the tympanic membrane. The procedure was performed with the patient lying supine, no anesthetic, and visualization with an operating microscope. The treated ear was positioned upward, with the patient lying still, for 45 minutes after injection.

On the fourth hospital day, the patient noted marked improvement in hearing and tinnitus. Repeat pure tone audiogram showed improved hearing to 40 dB at 250 to 2,000 Hz and a downslope from 45 to 100 dB at 4,000 to 8,000 Hz (Fig 2). The speech discrimination score was improved (68%). The daily intravenous prednisolone was tapered by 20 mg every 2 days.

On the fifth hospital day, the patient was discharged from the hospital on oral prednisolone, betahistine, and trimetazidine, and intravenous prednisolone was tapered by the 10th day of treatment. He returned for a daily outpatient intratympanic injection of dexamethasone (total, 7 intratympanic injections). On the 10th day of treatment, the pure tone audiogram and speech discrimination score were unchanged from the fourth hospital day (Fig 3), and tinnitus was completely resolved. The patient returned to his country after treatment and no additional follow-up was available.

**Discussion**

The patient had idiopathic sudden hearing loss after dental surgery, which recovered after intravenous prednisolone and a daily intratympanic injection of dexamethasone.

The pathophysiology of sudden hearing loss may include a decreased inner ear blood supply or viral inflammation of the cochlea or inner ear.\(^8,9\) Ischemia may occur from vascular spasm, thrombosis, hemorrhage, or hyperviscosity and sludged blood, and cochlear tolerance to ischemia is very limited.\(^5,10\) Histopathologic studies have shown that hair cells,
ganglionic cells, and spiral ligament are affected within 30 minutes of total circulation blockage. Spasm of the internal auditory artery may occur from autonomic dysfunction and cause anoxia, sludged blood, release of toxic substances, increased capillary permeability, localized disturbance in fluid and electrolyte balance, and hydrops.

Structural lesions found in the temporal bones of patients with sudden hearing loss may be similar to those in temporal bones affected by viral infection, and this suggests an association between viral infection and sudden hearing loss. Viral particles may affect capillary blood flow in the inner ear because of increased erythrocyte agglutination, capillary wall edema, or a hypercoagulable state. The incidence of dyslipidemia is increased in patients with sudden hearing loss.

Sudden hearing loss may occur after dental surgery because of noise exposure from drilling and the transmission of noise from the teeth to the ear. The noise intensity and duration of drilling may be risk factors for sudden hearing loss. Medical and dental histories may be important, and the present patient had dental treatment of an upper tooth ipsilateral to the affected ear.

Noise from tooth drilling is 120 dB, which is 64-fold (36 dB) greater than noise from the same source held 32 cm from the ear. The equivalent continuous noise level for dentists may range from 60 to 84 dB. Noise exposure of 120 dB may be above the pain threshold, and exposure for 28 seconds at 120 dB is equivalent to exposure for 8 hours at 90 dB. However, these estimates are based on measurements of air conduction experienced by a dentist, and bone conduction experienced by the patient may be greater than air conduction.

Communication between the teeth and the ear may result from: nociceptive afferent signals along the trigeminal nerve that cause a reflex on the inner ear; an autonomic reflex along antidromic fibers that may duplicate in the branches of the internal auditory artery; release of microemboli into the circulation, including potentially pathogenic bacteria; or venous channels that deliver ototoxic substances in high concentrations to the ear. Furthermore, local anesthesia with a vasoconstrictor such as epinephrine, used in the present patient, may cause localized vasospasm of the cochlear division of the internal auditory artery.

Despite thorough evaluation, the underlying cause of sudden hearing loss is unknown at presentation in 85% to 90% of patients. Treatment decisions typically are made without knowing the cause. Therefore, different idiopathic cases may be treated similarly, although the causes may vary. Spontaneous recovery may occur within 2 weeks of onset of symptoms in 32% to 65% of patients with sudden hearing loss. Spontaneous recovery may be total in 25% of patients, partial in 50% of patients, and absent in 25% of patients.

The most common treatment for sudden hearing loss includes systemic or intratympanic corticosteroids, used in the present patient. Other treatments may include antiviral agents, antibiotics, diuretics, vasodilators, anti-inflammatory agents, antihistamines, osmotic agents, plasma expanders, anticoagulants, mineral supplements, hyperbaric oxygen, or carbon dioxide. Treatment methods for sudden hearing loss may vary between different centers, but systemic steroid therapy is most commonly used. The efficacy of steroids in treating sudden hearing loss may occur from anti-inflammatory activity and increased cochlear blood flow.

Systemic steroids may have adverse effects, including cardiovascular, musculoskeletal, gastrointestinal, dermatologic, neurologic, and endocrine problems. Direct intracochlear application of therapeutic agents may minimize these adverse effects. Intratympanic or transtympanic delivery may provide higher concentrations of steroid to the inner ear than systemic administration. The applied steroid may enter the inner ear through the round window membrane.

**FIGURE 3.** Pure tone audiogram on the 10th day of treatment showed the same finding as the pure tone audiogram on the fourth day of treatment, namely improved hearing to 40 dB at 250 to 2,000 Hz and a downslope from 45 to 100 dB at 4,000 to 8,000 Hz.

annular ligament of the oval window, blood vessels, and lymphatic channels. In patients with sudden hearing loss, intratympanic steroids are used as initial treatment without systemic steroids, adjunctive treatment concomitant with systemic steroids, or salvage therapy after failure of systemic steroids. The addition of intratympanic dexamethasone to systemic treatment decreased hearing loss and tinnitus after failure of initial systemic prednisolone.

Intratympanic steroid treatment may vary by drug (dexamethasone or methylprednisolone), concentration (dexamethasone range, 10 to 24 mg/mL), and frequency. Adverse effects of intratympanic corticosteroids are infrequent but include pain, transient dizziness, infection, persistent tympanic membrane perforation, and vasovagal reaction or syncope during injection. Other factors that may limit this treatment include cost and multiple office visits.

The definition of successful improvement of hearing after therapy may vary between studies, and there are no definitive criteria that define recovery in patients treated for sudden hearing loss. Criteria for improvement range from any improvement on pure tone audiogram, or in speech discrimination scores to an improvement of 30 dB on pure tone audiogram, or 25% in speech discrimination scores. Improvement by at least 10 dB on pure tone audiogram or at least 15% in speech discrimination scores may be clinically relevant. By these criteria, the present patient had a successful outcome, with improvement on the pure tone audiogram from 100 to 40 dB and in the speech discrimination score from 50% to 68%.

The prognosis for recovery may be worse in patients younger than 15 years or older than 60 years, and in those with presence of vertigo at onset, greater levels of hearing loss, audiometric configuration, impaired hearing level in the opposite ear, and delayed diagnosis and treatment. Tinnitus and elevated erythrocyte sedimentation rate may have little prognostic value. Early recognition and treatment of sudden hearing loss are important because patients treated within the first week of onset have a better prognosis for improvement; patients treated within 7 days may have 50% improvement, but a delay of 6 weeks may decrease improvement to the 43% level of hearing. Only 25% of patients may have any improvement when treatment is delayed beyond 6 weeks. Therefore, it is prudent for dentists to be aware of this possible complication of dental treatment, and early referral to the otolaryngologist for treatment of sudden hearing loss may improve recovery and prognosis.

References