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Sudden sensorineural hearing loss (SSHL) following a local anesthetic dental procedure

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ABSTRACT

Acute sensorineural hearing loss is an uncommon phenomenon in dentistry. We describe the case of a 79-year-old male who presented with acute sensorineural hearing loss occurring 2 days after a tooth extraction procedure under local anesthesia. Possible mechanisms are discussed. He was treated with vasodilators (Ginaton and Alprostadil Injection) and Mecobalamin injection with benefit. High dose oral steroids (1 mg/kg) and low molecular weight dextran were used.

1. Introduction

Idiopathic sudden onset sensorineural hearing loss (SSHL) is a recognized phenomenon and its reported annual incidence is5–20 individuals per year per 100,000 (Stachler et al., 2012; Mattucci et al., 1982; Farrell et al., 1991). In the United States, SSHL is commonly regarded as sudden deafness (SD) by the National Institute on Deafness and Other Communication Disorders (NIDCD), which is defined as unexplained, rapid loss of hearing by at least 30 dB, within three days and influencing at least three consecutive frequencies in both ears or one ear (Stachler et al., 2012). Patients with SSHL may have concomitant symptoms including tinnitus, dizziness, vertigo, fullness in the ear and headaches (Stachler et al., 2012; Mattucci et al., 1982; Lazarini et al., 2006). The 2015 Chinese guidelines on sudden deafness treatment and diagnosis define SSHL as unexplained sensorineural hearing loss of 20 dB or more affecting at least 2 consecutive frequencies which occurs within 72 h (three days) (Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery.2015).

A number of etiologies have been postulated for SSHL, but in about 90% of these patients the exact etiology cannot be found (Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery. 2015). One of the possible but uncommon causes of SSHL is medical procedures. There have been SSHL cases reported after cardiopulmonary bypass, with an incidence of 1 in 1000. This is characterized by micro-emboli from the cardiopulmonary tree (Plasse et al., 1981). Cases resulting from cervical spine manipulation and vertebral artery injury have also been reported (Brownson et al., 1986). On the other hand, SSHL is unusual after general anesthesia (GA) for non-cardiac and non-otologic procedures (Evan et al., 1997). There were merely 44 cases reported in the literature to date, mostly after gynecologic, abdominal, spinal and ophthalmic procedures (Goodhill et al., 1976). SSHL following dental procedures are rarely documented. This paper presents a case of SSHL occurring after a dental extraction procedure under local anesthesia. Evaluation and treatment results were reported, and relevant literature were reviewed.

2. Case report

2.1. The patient and medical history

A 79-year-old man complained of an acute hearing loss in left ear occurring after a tooth extraction under local anesthesia. The patient had myringitis mycotica and otitis externa since 2010 with

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no other ENT diseases or trauma/barotrauma history. The patient reported some baseline hearing loss (symmetrical) in both ears, which were not documented on audiogram.

2.2. Onset of hearing loss

The patient received a dental extraction procedure under local anesthesia (4 ml of 4% articaine hydrochloride with adrenaline-sat1:1000). He experienced acute vertigo and hearing loss in left side immediately after the procedure. Vertigo resolved spontaneously the next day but the hearing loss remained.

2.3. Tests

Pure tone audiometry indicated worse mostly sensorineural hearing loss in left ear, especially involving low frequencies (Fig. 1-a). The 4FA (four-frequency average of 0.25, 0.5, 1 and 2 kHz) threshold was 38.75 dB HL in right ear and 48.75 dB HL in left ear. Air-bone gap (ABG) was also seen in left ear at 250, 500 and 1000 Hz. Rinne test was positive (air conduction > bone conduction) on left side, while Weber test was at the middle. Tympanogram was type A in both ears. No nystagmus was found in vestibular examination.

Common protocols of Magnetic Resonance Imaging (MRI) of the brain and internal acoustic meatus excluded the possibility of posterior circulation ischemia causing SSHL and showed no evidence of a neurological disorder. Hematological and biochemical tests were unremarkable.

2.4. Treatment

The subject was given vasodilators (Ginaton and alprostadil) and mecobalamin injection. Oral steroids (prednisone 1 mg/kg/d) and low molecular weight dextran were also used. Hearing started to improve 3 days after treatment was started. After one week of treatment, 4FA remained at 38.75 dB HL in the right ear and 47.75 dB HL in left ear, with no change in ABG (Fig. 1-b). Ginaton, alprostadil and mecobalamin were continued for one more week, when 4FA was 36.25 dB HL in the right ear and 45 dB HL in left ear, with ABG slightly decreased (Fig. 1-c).

2.5. Hearing following up

The patient was followed for 3 years after treatment was completed with repeated audiometric evaluations.4FA thresholds gradually increasedto55 dB HL in left ear, but remained relatively stable in right ear (with some fluctuations), with minimal change in ABG at 250 and 500 Hzandreport of tinnitus and transient dizziness (Fig.1 d to i). As a result, for the period of follow up, interaural 4FA differences gradually increased (Fig. 2-a), mainly from changes at 250 Hz (Fig. 2-b) and 500 Hz (Fig. 2-c) but not so much at 1000 Hz or 2000 Hz (Fig. 2-d and 2-e).

3. Discussion

3.1. Medical cause of SSHL

SSHL should be considered as a medical emergency. The exact cause of SSHL is unknown in 85%–90% of SSHL patients, even with extensive testing. Only in 10%–15% of the patients who are diagnosed with SSHL, a definite cause can be identified (Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery. 2015).

SSHL may frequently be caused by multiple factors. Some suspected etiologies include viral orbacterial infections of the cochlea or inner ear (Wilson et al., 1983), trauma (mechanical, iatrogenic, noise-induced and barotraumas), micro-emboli, autoimmune diseases, acoustic neuroma, ototoxic drugs, neurotoxicity, anesthesia, circulation disorders, metabolic disorders, vascular diseases, inner ear disorders (e.g. Ménière's disease) and neurologic diseases (such as multiple sclerosis) (Goodhill et al., 1976).

While often idiopathic, SSHL can be secondary to infections (Stachler et al., 2012). Virus particles may impact capillary flow in the inner ear as a consequence of increased erythrocyte agglutination, hypercoagulable stateorcapillary wall edema (Mattucci et al., 1982). Immunological disorders including autoimmune conditions (e.g. Cogan's syndrome) may be another possible cause (Bachor et al., 2005). Certain kinds of tumors, such as vestibular schwannoma (Sauvaget et al., 2005) oracoustic neuroma, cangive rise to SSHL. Perilymph fistula may also cause SSHL (TS Tan et al., 2007), although relatively rare. Drug overdose, from accidental intravascular injection or direct injection into the nerve may result in neurotoxicity in local anesthesia. Nitrous oxide (NO) may be related to hearing loss in general anesthesia (Evan et al., 1997). Other possible contributing factors in general anesthesia (GA) include Valsalva maneuvers, perilymphatic fistula, micro-emboli, sneezing and coughing (Goodhill V et al., 1976). Blood flow change in the inner ear is considered one of the pathophysiological mechanisms in SSHL. Correlations to blood viscosity (Grgic et al., 2005) and arterial flow (Mierzwa et al., 2004), as a result of circulatory system disorders, have been reported as major etiologies of SSHL.

The cochlea is sensitive to ischemia, which can result from blood sludging, hyper-viscosity, vascular spasm, thrombosis, or hemorrhage (Mattucci et al., 1982; Marcucci et al., 2005). His topathologic studies have demonstrated that 30 min of block age of blood supply to the cochlea causes changes in the spiral ligament, hair cells and ganglionic cells (Lazarini et al., 2006; Merchant et al., 2005). Spasm of the internal auditory artery can lead to ischemia and release of otoxic substances in the inner ear, as well as increased capillary permeability, electrolyte balance disturbances and hydrops (Mattucci et al., 1982; Lazarini et al., 2006), which have direct impact on hair cells.

3.2. SSHL and dental procedure

The present patient had ipsilateral SSHL after a dental procedure, potentially related with the following factors.

(1) Vasoconstrictor: The recorded 4 ml of 4% articaine hydrochloride indicates no drug overdose in this case, although unintentional direct intravascular injection cannot be completely ruled out. However, vasoconstrictor (epinephrine) was used, which potentially could cause spasm of the internal auditory artery and ischemia of the cochlea. Four cases of SSHL after dental procedures were reported by Farrel et al., of which three involved local anesthesia (LA) and one involved general anesthesia (GA) (Farrell et al., 1991). The extensive vascular communications in the head and neck region may take a drenaline used in local anesthetic to the internal auditory artery, leading to local vascular spasm and subsequently hearing loss.
(2) Micro-emboli: Micro-emboli can be released into blood during dental extraction, causing ischemia and infarction of the cochlea and subsequent hearing loss when they end up in inner ear circulation, similar to what can potentially occur following a cardiopulmonary bypass procedure (Farrell et al., 1991).
(3) Hypo-perfusion: During tooth extraction, the necessary movement of cervical spine and wide jaw opening and

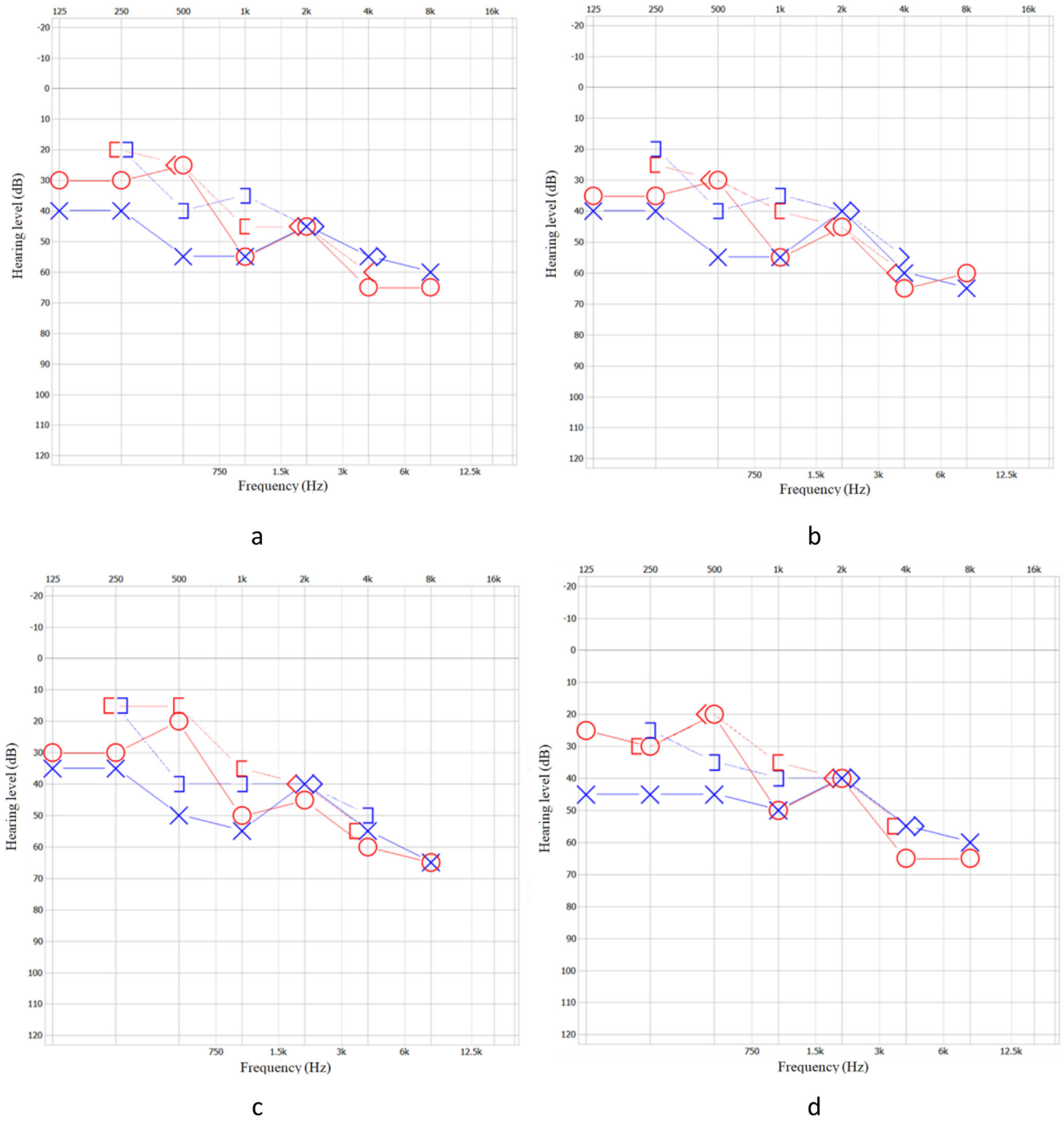
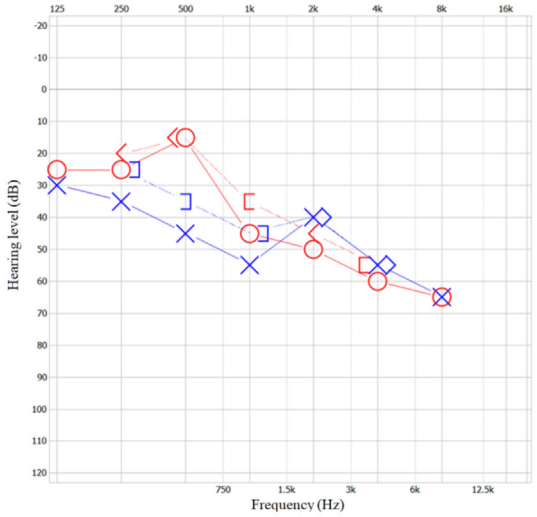


Fig. 1. Pure tone audiometry results. a: at initial visit; b: after one week of treatment; c: after two weeks of treatment; d: at one week follow up; e: at two weeks follow up; f: at one month follow up; g: at 9 months follow up; h: at two years follow up; i: at three years follow up. Red circle: right ear air conduction; blue cross: left ear air conduction; red [: right ear bone conduction; blue]: left ear bone conduction.

associated crushing and stretching of the vertebral artery may lead to cochlea hypo-perfusion, causing the SSHL in the present case.

(4) Noise: Noise exposure is another possible cause of SSHL, especially in dental procedures, generally from drilling noise transmitted from the teeth to the ear mainly by bone conduction. The duration of drilling and noise intensity play an important role. Measurement at the position of the dentist shows constant air conduction noise levels from 60 to 84 dB SPL. For the patient who receives the energy by bone conduction, the noise level may potentially be even greater (Farrell et al., 1991). High level exposure over long period of time can potentially impair hair cells in the cochlea.



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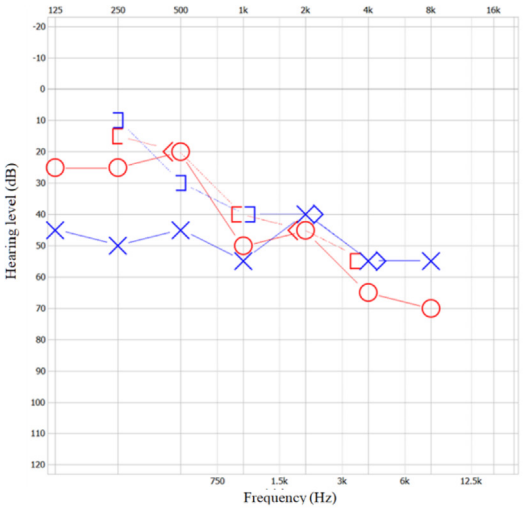

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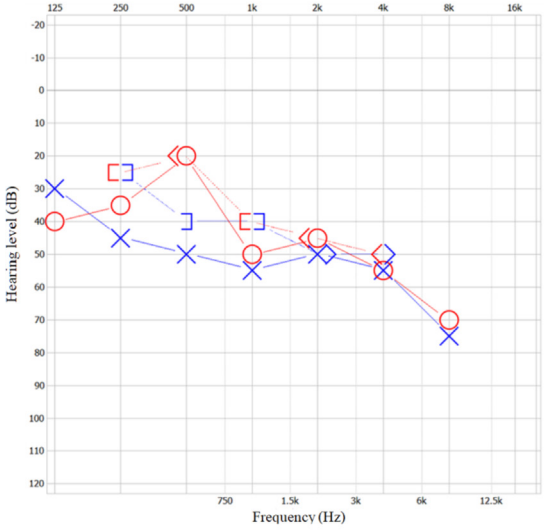

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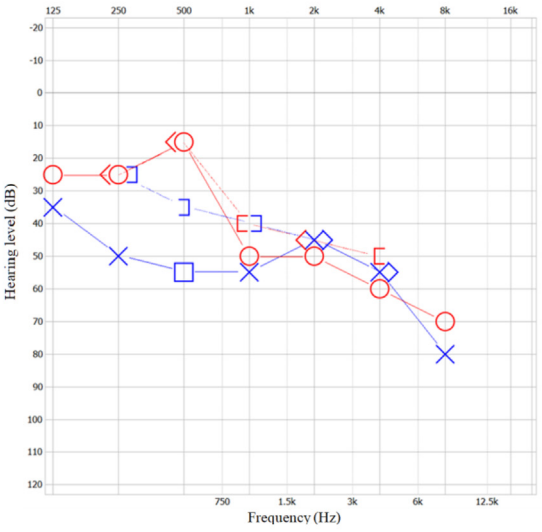

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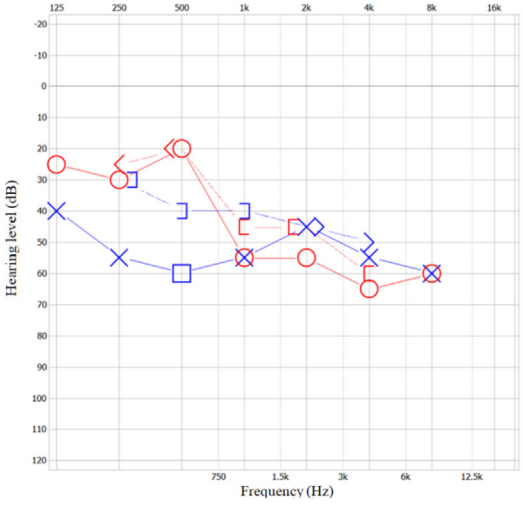

i

Fig. 1. (*continued*).

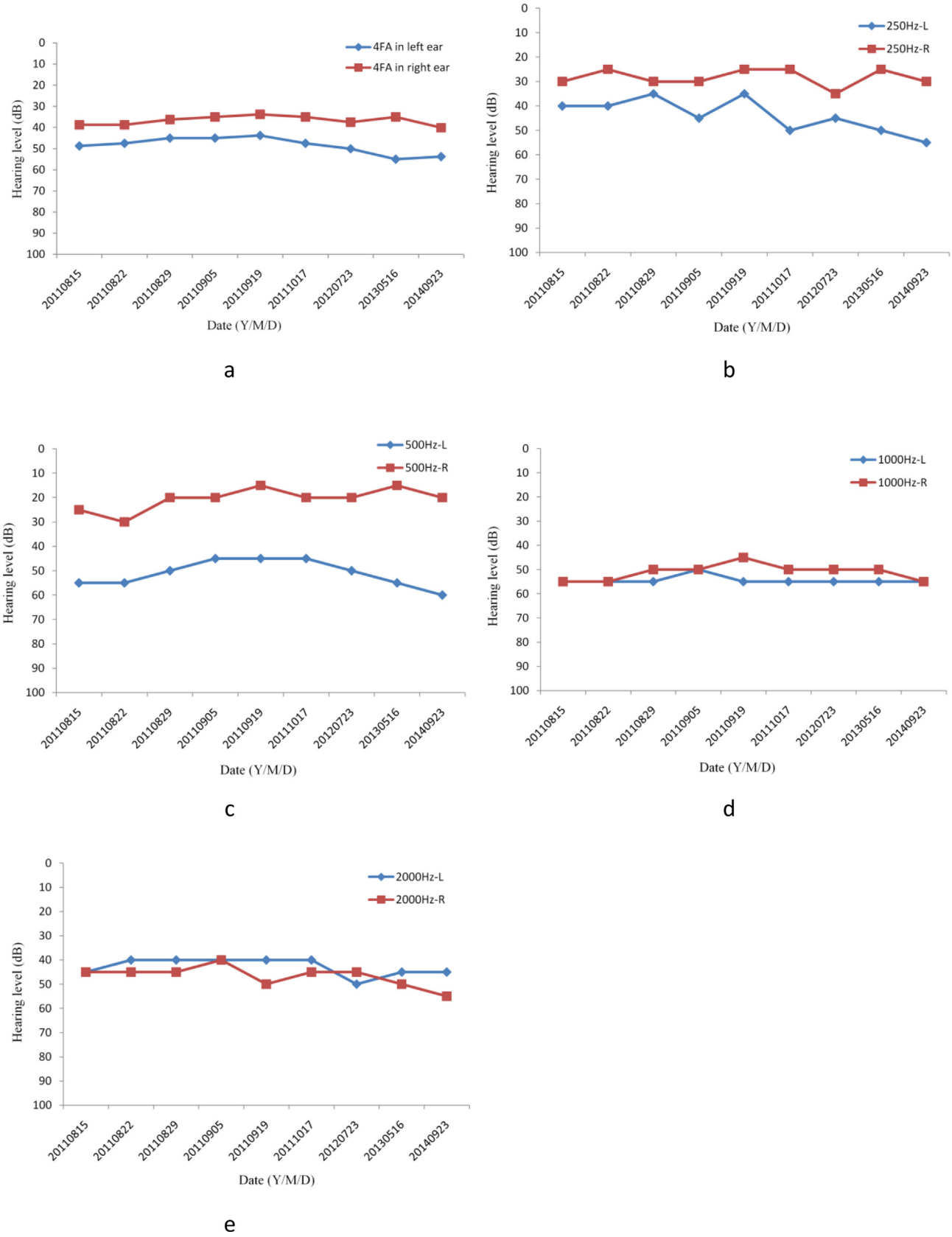

Fig. 2. Threshold changes at different frequencies over time. a:four frequency (250, 500, 1000 and 2000 Hz) average (4FA); b: at 250 Hz; c: at 500 Hz; d: at 1000 Hz; e: at 2000 Hz.

3.3. Treatment and prognosis

For SSHL, treatments are often initiated without knowing the exact etiology. Despite the various possible causes, treatment for SSHL is often similar (Stachler et al., 2012; Mattucci et al., 1982), In 32%–65% of patients with SSHL, hearing can recover spontaneously within two weeks of onset (Stachler et al., 2012). Total recovery is possible in one fourth of these cases. In25% of patients, SSHL does not recover spontaneously (Lazarini et al., 2006). Due to variable etiologies in SSHL, several treatment strategies have been tried, including systemic and topical steroids to reduce the inflammatory process, carbogen to increase blood supply in the inner ear, and dextran 70 to improve circulation. Pharmacologic interventions (antibiotics, anti-inflammatories, antivirus, mineral supplements, vitamins, rheologic agents, diuretics, vasodilators, antihistamines, osmotic agents, plasma expanders, anticoagulants and herbal preparations), hyperbaric oxygen, carbon dioxide and observation have also been used in treating SSHL (Goodhill et al., 1976). Surgical exploration is sometimes indicated for perilymph fistulae or acoustic neuroma, especially in the presence of vertigo (Stachler et al., 2012; Arslan et al., 2011).

Treatment of SSHL varies from one medical center to another, but systemic corticosteroid therapy is widely used, especially in cases without known causes (Arslan et al., 2011). There is so far no consensus on the use of steroid regarding the type, dosage and route of delivery. Ten to fourteen days of prednisone (maximum dose at 1 mg/kg/d for 4 days, followed by a taper by 10 mg every 2 days) is one of the recommended regimens. Side effects by systemic steroids have been reported, including cardiovascular, musculoskeletal, gastrointestinal and dermatologic reactions. Direct topical use of therapeutic agents can potentially reduce side-effects (Seggas et al., 2011). Intra-or trans-tympanic delivery may provide higher steroid concentrations in the inner ear than systemic administrations (Stachler et al., 2012). The eardrum-delivered steroid may reach the inner ear via the pathway of the middle ear, round window, annular ligament of the oval window, blood vessels and lymphatic channels (Arslan et al., 2011). The combination of intra-tympanic and systemic steroids may enhance treatment effects.

The definition of effective hearing improvement varies between reports. According to the 2015Chineseguidelines on sudden deafness, improvement by 30 dB at affected frequencies on pure tone audiogram is considered as excellent, and improvement by no less than 15 dB is probably still clinically meaningful (Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery. 2015). By this criterion, unfortunately, the present case did not obtain a clinically meaningful result.

Significant prognostic indicators in SSHL include the severity of initial hearing loss, frequencies affected, accompanying symptoms, treatment timing, patient age et al. (Stachler et al., 2012; Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery. 2015). It has been reported that outcomes may be poor in patients older than sixty years or younger than fifteen years. Patients with more severe hearing loss or vertigo at onset, and those receiving delayed diagnosis and treatment, may obtain less favorable results (Stachler et al., 2012; Editorial Board, Chinese Journal of Otorhinolaryngology Head and Neck Surgery. 2015).

Early diagnosis and treatment are crucial in SSHL. Hearing improvement is seen in 49%–79% of patients treated within one week of hearing loss onset, while treatment delay by six weeks decreases the chance of hearing improvement to 43%. Only one fourth of patients may notice hearing improvement if treatment is delayed by more than six weeks (Mattucci et al., 1982).

4. Summary

Although the exact cause of SSHL in this patient remains unclear, the sudden onset of hearing loss following a dental treatment appears to be more than coincidental. The rarity of reports on such cases may be attributed to poor recognition of the correlation between SSHL and dental procedures. Most possible causes mentioned in previous reports can be entailed in this case. Earlier diagnosis and earlier treatment may lead to a better result. It is also prudent and extremely important for dentists to realize possible complications in oral procedures, including hearing loss, and timely referral to the otolaryngologist for comprehensive evaluation and treatment is mandatory and may improve prognosis and outcomes.

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