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Abstract

Hyperacusis is increased hearing sensitivity to the ordinary environmental sounds. Tinnitus and hyperacusis co-occur in a wide range of patients including those having neural, sensory or psychological pathologies, head injury, temporomandibular junction disorders, ototoxicity, etc. Sudden loud noise exposure is also a major cause of hyperacusis and tinnitus. The present case report highlights the occurrence of hyperacusis and tinnitus with normal hearing thresholds after exposure to loud music for two hours in a rock music concert. The functioning of middle ear, cochlea, vestibular system, auditory nerve and stapedius muscle was examined and found to be normal. Johnson's hyperacusis quotient revealed mild hyperacusis in both ears and tinnitus handicap inventory showed bilateral moderate tinnitus. Medical treatment and tinnitus desensitization therapy was given to the client elsewhere, but no significant improvement in hyperacusis and/or tinnitus perception was reported by the client. Thus, the possible central nervous system involvement in hyperacusis and tinnitus may be considered and hence, tinnitus retraining therapy was suggested to the client.

Keywords: Tinnitus retraining therapy, sound injury, rock music, loud sound.

Background

Hyperacusis is defined as reduced tolerance to ordinary environmental sounds (Vernon, 1987). It is characterized by increased sensitivity to certain frequency and intensity of sound. It accounts for approximately 5-15% of the hearing impaired population seeking medical help (Andersson, Lindvall, Hursti, & Carlbring, 2002; Fabijanska, Rogowski, Bartnik, & Skarzynski, 1999). The prevalence of hyperacusis along with tinnitus is widely noted. Among the patients

with primary complaint of tinnitus, the prevalence of hyperacusis range from about 40% (Jastreboff & Jastreboff, 2000) to as high as 86% (Anari, Axelsson, Eliasson, & Magnusson, 1999).

A number of clinical conditions have considered to be leading to hyperacusis. These include ototoxicity, neural pathology, sensory disorders, psychological problems, head injury, temporo-mandibular joint disorders and others (Andersson et al., 2005; Baguley, 2003; Batuecas-Caletrio, del Pino-Montes, Cordero-Civantos, Calle-Cabanillas, & Lopez-Escamez, 2013; Zarchi, Attias, & Gothelf, 2010). The most common cause of hyperacusis is overexposure to very high intensity sound (Størmer & Stenklev, 2007). Continuous exposure to loud sound (usually above 110 dB SPL) for long duration or sudden exposure to sound above 140-150 dB SPL are reported to be commonly associated with hyperacusis, tinnitus and hearing loss. Noise exposure damages the delicate structures of the ear, resulting in hearing loss, tinnitus, diplacusis, distortion and hyperacusis (Lie et al., 2016).

Various researchers have attempted to explain the cause of hyperacusis as secondary to noise exposure. According to Robels and Ruggero (2001) noise damages the hair cells in the inner ear resulting in disturbing the non-linearity of cochlea. This causes loss of compression of the amplitude of the basilar membrane vibration. The loudness growth in the auditory nerve activity with sound levels has also been proposed as a possible mechanism behind abnormal perception of loudness (Heinz & Young, 2004). Perhaps, the most common cause of hyperacusis following noise exposure is damage to the stapedius muscle of the middle ear. The stapedius muscle contract in response to the loud sound exposure, and hence protect the cochlear structures from damage due to noise. However, excessively loud noise (~120 dB and above) damages the stapedial tendon and hence fails to protect the cochlea. This results in acoustic trauma leading to

sensori-neural hearing loss. Most of the reviewed cases of hyperacusis, secondary to noise exposure, also reported sensori-neural hearing loss (Salloum, Yurosko, Santiago, Sandridge, & Kaltenbach, 2014; Sun, Deng, Jayaram, & Gibson, 2012; Turner & Larsen, 2016). The present case report describes a case who developed hyperacusis and tinnitus following noise exposure, but had normal hearing sensitivity.

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Case Report

A 37 year old male patient visited department of Audiology with the complaint of irritability to sound and continuous ringing in the ear. Detailed case history revealed that the client's problem started immediately after attending a rock music concert for two hours at USA. The client was sitting immediately below the loudspeakers. The music was not very loud (as reported), but the client felt buzzing sensation in both ears. Next morning, he had slight pain in the ears with buzzing sound, and later the client noticed oversensitivity to even soft sounds in the close vicinity.

The medical and audiological evaluation were carried out in a hospital at California, and the available reports revealed normal hearing sensitivity with reduced loudness discomfort level. The client was diagnosed as having hyperacusis and tinnitus and was suggested to follow the medical line of treatment. One month course of certain analgesics and antibiotics, showed no significant improvement in tinnitus and hyperacusis perception. However, the ear pain reduced significantly. On follow up visit, sound desensitization therapy was suggested to the client, by using ear level noise generators, and the therapy lasted for 3 months. No marked relief from tinnitus and hyperacusis was reported, even after this therapy.

The otoscopic examination showed normal external auditory canal and tympanic membrane. Tympanometric testing showed bilateral 'A' type tympanogram indicating normal middle ear function. The acoustic reflexes were present in both the ears, focusing on normal stapedius muscle function (Table 1). Pure tone audiometry for conventional and extended high frequencies revealed 'bilateral normal hearing sensitivity' (Figure 1) and speech audiometry results were in agreement with pure tone average thresholds (SRT±10 dB of PTA; SIS≥85%). Distortion product and transient evoked oto-acoustic emissions were also present with good signal to noise ratio (Table 1). Tone decay test was done to assess the auditory nerve involvement causing tinnitus and hyperacusis. Negative tone decay at octave frequencies from 500 to 4000 Hz ruled out auditory nerve pathology (Table 1).

Insert table 1 here

Insert Figure 1 here

The severity of hyperacusis was measured using Johnson's hyperacusis quotient (JHQ) (Hawkins, 1980). The loudness discomfort level (LDL) was measured at the octave frequencies from 250 Hz to 8000 Hz (Figure 1), and the mean LDL value was rated according to JHQ severity rating. The results revealed mild hyperacusis in both ears. The tinnitus severity was measured using tinnitus handicap inventory (THI) (Newman, Jacobson, & Spitzer, 1996). The THI scores were '40' indicating moderate tinnitus. The pitch and the loudness of tinnitus was also matched by adapting the procedure suggested by Mitchell, Vernon and Creedon (1993). In the right ear tinnitus pitch was matched at 125 Hz with the perceived loudness of 24 dB HL and in left ear, the pitch was matched at 125 Hz with loudness of 21 dB HL. Residual inhibition was present in both the ears at 5 dB SL.

Thus, the patient was diagnosed as a case of "Hyperacusis and Tinnitus secondary to noise exposure". Tinnitus retraining therapy (TRT) was suggested to the client which involved counseling and sound therapy. TRT has been a successful method for treatment of hyperacusis and tinnitus as suggested by Jastreboff and Jastreboff (2000), and thus, the same was advised to this patient also. A significant reduction in the hyperacusis and tinnitus perception was reported by the client after 3 months of TRT.

Discussion

The case report highlights the presence of hyperacusis and tinnitus in a 37 year old male after exposure of moderately loud music. The client had no stapedius muscle injury, peripheral hearing loss, auditory nerve pathology as well as no symptoms of vestibular system involvement. The client's audiological features were unique as available literature indicates the presence of either sensori-neural hearing loss (Salloum et al., 2014; Sun et al., 2012; Turner & Larsen, 2016) or stapedial muscle damage (Asha'ari, Mat Zain, & Razali, 2010; Liriano, Magalhães, Barros, Testa, & Fukuda, 2004).

The audiological findings of the client indicates probable central nervous system involvement in the origin of hyperacusis and tinnitus, i.e., increased sensitivity to spinal neurons as proposed by Woolf (2004), as one mechanism indicating central involvement in individuals with hyperacusis. Other mechanisms involved in tinnitus and hyperacusis include reduced central inhibition (Schwenkreis et al., 2003), disturbed inhibitory-excitatory balance associated with multiple biological changes in the nervous system (Woolf & Mannion, 1999) and disturbed sensori-motor processing (de Klaver et al., 2007). The type 1 auditory nerve fibers alter the excitation of the glutamate which result in inducing hyperacusis, and contribute to the induction,

maintenance and exacerbation of tinnitus (Sahley, Nodar, & Musiek, 1999). Thus, any of these plausible explanations can be applied to the present case in explaining the symptoms shown by him.

Conclusion

The present case study highlights the occurrence of hyperacusis and tinnitus with normal hearing thresholds in a patient after exposure to loud music. The functioning of the peripheral auditory system including middle ear, cochlea, vestibular system and auditory nerve were normal in the client. Thus, the possible central nervous system involvement in hyperacusis and tinnitus may be considered.

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Figure Legend

Figure 1: Pure tone audiometry results measuring hearing thresholds from 250 Hz to 14 KHz and loudness discomfort level (LDL) from 250 Hz to 8 KHz.

Table 1: The hearing thresholds for various tests measured across different frequencies.

Tests	Right Ear				Left Ear			
	500 Hz	**1 KHz**	**2 KHz**	**4 KHz**	**500 Hz**	**1 KHz**	**2 KHz**	**4 KHz**
ART (Ipsi)	80 dB	85 dB	90 dB	NR	85 dB	85 dB	90 dB	95 dB
ART (Contra)	85 dB	90 dB	90 dB	95 dB	80 dB	90 dB	95 dB	NR
DPOAE's (SNR)	23	18	21	16	14	16	16	15

TEOAE's (SNR)	20	22	17	12	9	11	13	12
TDT*	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative

*Negative TDT indicate that the client was able to hear the tone at the specific frequency for one minute.

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