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Oral & Intra Tympanic Corticosteroid effect on Sudden Hearing Loss & Suppressing Tinnitus: A Pilot Study

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Abstract

Systemic glucocorticosteroids ("steroids") are widely used worldwide as a standard of care for primary therapy of idiopathic sudden sensorineural hearing loss (ISSHL). The German ISSHL guideline recommends high-dose steroids without evidence from randomized controlled trials (RCTs) and refers solely to retrospective cohort studies. This RCT aims to assess the efficacy (improvement in hearing) and safety (especially systemic side effects) of high-dose steroids versus standard of care (standard dose systemic steroids) for the treatment of unilateral ISSHL, when given as a primary therapy. There is an unmet medical need for an effective medical therapy of ISSHL. Although sensorineural hearing impairment can be partially compensated by hearing aids or cochlear implants (CI), generic hearing is better than using hearing aids or CIs. Since adverse effects of a short course of high-dose systemic corticosteroids have not been documented with good evidence, the trial will improve knowledge on possible side effects in the different treatment arms with a focus on hyperglycemia and hypertension.

Introduction:

Sudden sensorineural hearing loss (SNHL) is a confusing and debatable issue in otological practice. Sudden sensorineural hearing loss (SNHL) is a common otological emergency. SSHL commonly known as sudden deafness occurs as an unexplained, idiopathic, rapid loss of hearing usually in one ear either at once or over several days. SSNHL is an emergency of otolaryngology characterized by rapid onset of hearing loss or a progressive loss over 24 hours, with an average hearing loss of more than 30 dB on at least three contiguous frequencies within 72 hours. Approximately, 50% patients complain of associated dizziness, tinnitus. Sudden hearing loss is a typical phenomenon that bring about an urgent or emergent visit to ENT [1]. Approx, 4000 cases of sudden hearing loss (SHL) occur annually in the United States, and 15,000 annually worldwide, accounting for approximately 1% of all cases of Sudden HL [2]. A wide spectrum of etiology including local, systemic, retrocochlear diseases are associated with sudden SNHL, like viral diseases, temporary breaks of the inner ear membranes, and immune-mediated reactions. Vascular sclerosis of the microcirculation in the inner ear but still an "idiopathic unilateral" feature of the disease is predominant [3]. Gupta et al stated that out of 37 individuals 35.14% patients had complete recovery while 40.54% and 24.32% patients had partial and no recovery respectively [4]. Idiopathic sudden sensorineural hearing loss remains one of the major unsolved otologic emergencies. The method of clinical staging presented here is based on four elements represented by the acronym HEAR. The individual elements of the staging are hearing threshold (H), elapsed time from onset (E), audiogram shape

(A), and related vestibular symptoms[5]. Some studied reported that the incidence of SSNHL is 5 to 20 cases per 100,000 people per year.1 However, a German study published in 2009 has estimated that there are 160 cases of SSNHL per 100,000 people annually[6]. Epidemiological surveys also show that the incidence of sudden deafness is increasing [7, 8]. The most commonly suspected etiologies of SSNHL include viral infection [9], vascular occlusion[10], abnormal cellular stress responses within the cochlea[11], and immune-mediated mechanisms[12]. Additionally, other studies have shown that antiviral therapy is effective in sudden deafness [13,14], however,the effectiveness of antiviral therapy remains controversial[15-17]. According to a recent epidemiological study, the incidence of sudden sensorineural hearing loss (SSNHL) is increasing yearly [18]. There were no significant differences in degree of improvement between oral and simultaneous oral + intratympanic [19]. Tinnitus is presently viewed as an abnormal, conscious, auditory percept reflecting multiple levels of neuronal dysfunction/dyssynchrony involving either or both the peripheral and central nervous system [20]. The Neurophysiological model of Jasterboff describes distressing tinnitus as reflecting four stage mechanism: generation of peripheral neuronal activity, detection, and perception in the subcortical and cortical auditory areas respectively, and a sustained activation of the auditory related limbic and autonomic nervous system [21]. Shulman proposed an algorithm–based final common pathway model of tinnitus involving the neuroanatomical substrates of sensory, affect and psychomotor components of an aberrant auditory stimulus [22]. It postulates the involvement of, and a complex interaction between, the brainstem, cochlear nucleus, olivocochlear bundle to the inferior colliculus, medial geniculate body, intralaminar-thalamic nuclei, parabrachial nucleus and also the primary ascending reticular activating formation of the lemniscal system to the thalamus (Figure 1).

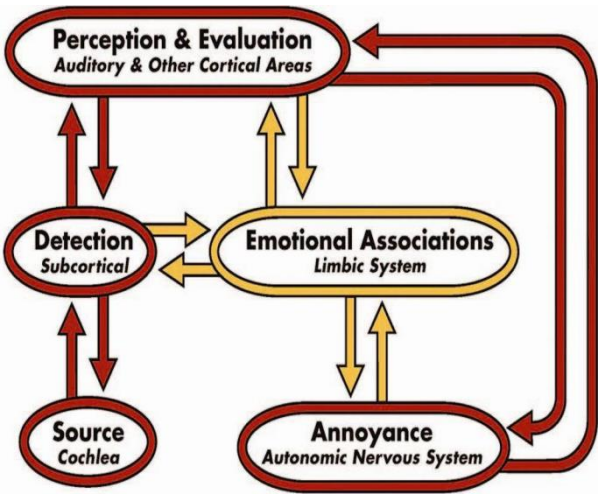

Figure 1: Neurophysiological model of tinnitus (Jastreboff 1990; Jastreboff 2004)

Hyper/depolarization of gamma-aminobutyric acid (GABA) influenced thalamic activity results in thalamocortical oscillations in a synchronous signal at the brain cortex. Reciprocal innervation from the thalamus to the medial temporal lobe system including the amygdala, and hippocampus comprise an endogenous system which is hypothesized to result in the establishment of a “paradoxical memory” for the aberrant auditory sensation (tinnitus) with a reciprocal interaction with the thalamus. These models also highlight the reduction in auditory masking and univocally reflect the importance of the auditory

thalamo-cortical tract and its connections with the limbic and autonomic nervous system, in tinnitus percept.

Tinnitus frequently follow the symptoms of idiopathic sudden hearing loss (ISHL) (70% of the cases) and diziness is sometimes present (up to 40% of the cases), thus completing the triad of Ménière. About 10% of dizziness cases may be incapacitating and associated to nausea and vomit [23-25]. Studies stated that there was no increases in dizziness or tinnitus lasting longer than 24 hours were observed after injections.There was statistically significant improvement in 4-frequency pure-tone average and speech discrimination score at 1 month after treatment [26]. The incidences of viral conversion and sudden hearing loss track one another closely, suggesting that viral infection is a major cause of ISHL [27].

Aim:

To identify and define the disease at earliest and to study the efficacy of multidrug high dose steroid treatment & also suppression of Tinnitus after intratympanic doses.

Materials and Methods:

The research proposal was approved by the research approval and ethical committees of the respective affiliated institutions of the authors and the approvals are available on request. Signed consents procured from all participants. Patients attending the Department of ENT at CK Birla Hospital(CMRI), Kolkata, India & few private clinic at BURDWAN, Kolkata, India. During the period of Jan 2021 to July 2024 with complaints of sudden SNHL were included in the study.

- **Instrumentation:**

Tinnitus handicap inventory (THI); pure tone audiometer: Madsen Itera II diagnostics, immittance audiometer: Inventis Flute; Auditory evoked potential instrument: VIVOSONIC pro auditory evoked potentials systems

Subjects:

An experimental group was constructed with 75 patients of age-range from 20 to 50 years with mean age = 29.175 years (51 male and 24 female) with complaint of sudden SNHL with Moderate to Severe degree of tinnitus & blockage sensation in the poorer ear.

Those with age >20years and with mixed and conductive hearing loss (HL) were excluded.

- **Evaluation:**

A complete Protocol Based and ENT examination was done audiometric evaluation included pure tone audiogram, impedance audiometry and if required imaging. Pure tone audiometry, Tympanometry & THI were repeated on day 4, 3 weeks and 6 weeks after the onset of HL.

Table 1: Age wise demographic data of individuals

Gender	Mean(Year)	SD
Male	26.825	7.91
Female	32.015	7.21
Both	29.175	7.56

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The identification of pathologies affecting the middle ear or the auditory meatus (including ceruminal plugs) is performed by ear microscopy and tuning fork tests. The current version of the German S1 guideline on ISSHL recommends the following elements as necessary diagnostics

- Intensive general and specific history taking
- ENT-specific physical examination

Blood pressure measurement

- Ear microscopy
- Hearing tests (tuning fork, pure tone audiogram)
- Tympanometry
- preliminary vestibular testing (ENG/VNG, VEMP, ECoCHG)

RESULT AND DISCUSSION:

Regarding the design of the studies, 63.3% were prospective, 30% and 6.7% retrospective prospective and retrospective. Only three studies (10%) were controlled and randomized. Three other studies (10%) were randomized and only two (5%) were controlled.

As to the objectives of the studies analyzed, 76.7% sought to evaluate the use of intratympanic therapy salvage after failure of conventional treatment. Treatment therapy with systemic steroids was considered as conventional therapy; 52.2% of the studies used oral administration and 47.8%, intravenous administration. Intratympanic therapy was used as the primary treatment 23.3% of the studies.

It is noteworthy that 100% of the studies used auditory evaluation by pure tone audiometry as a method of checking the effectiveness of the therapy chosen, mainly using as improvement criteria 20 dB increase in the mean frequencies of 0.5, 1, 2, and 4 kHz. Of these, some studies included evaluation through speech audiometry and impedance.

Analyzing the articles published on sudden hearing loss and use of intratympanic corticosteroid therapy, the data showed that this is a promising treatment modality, especially for cases where there was a failure in the conventional treatment, as well as in those where systemic corticosteroid therapy is contraindicated. It can be seen that in most of the analyzed studies, the authors made three intratympanic corticosteroid applications, usually every other day. However, dosages were different, ranging from one to five applications.

Pure tone audiometry was the audiological test used in all the studies analyzed. However, because of the possibility of sudden deafness is multifactorial and affects the inner ear and/or auditory pathways, the authors suggest testing electrophysiological objectives accompanying a patient.

CONCLUSION:

The study by Rauch et al. in 2011 compared the non-inferiority in the two therapeutic approaches for sudden deafness (intratympanic and oral), and 250 subjects were followed for a period of six months. They found that non-inferiority was defined as a difference of less than 10 dB in hearing improvement between treatments. In the group treated with oral prednisolone (n = 121), the pure tone average (PTA) improved 30.7 dB compared to the group receiving intratympanic corticosteroid therapy (n = 129), who presented improvement of 28.7 dB.4 Similarly to these authors, Dallan et al., in Italy, also followed patients for six months. However, their study examined the efficacy of intratympanic corticosteroid therapy after failure of oral therapy.

Other researchers followed-up for up to three months, and most of the time, during only one month after starting treatment with intratympanic corticosteroid.
The following studies are presented according to the type of therapy used.

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