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ORIGINAL ARTICLE

Sudden Sensorineural Hearing Loss: An Otologic Emergency

H. Vijayendra · Greeshma Buggaveeti · Bhavin Parikh · R. Sangitha

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Abstract The study purpose was to determine the efficacy of steroids, volume expanders and antivirals in the management of idiopathic sudden sensory neural hearing loss and to establish importance of early medical intervention. In this prospective study, thirty-four patients presenting with idiopathic sudden hearing loss of 30 db or more were enrolled in study group between 2005 and 2009. Patient variables as they related to recovery were studied and include patient age, time to onset of therapy, status of contralateral ear, presence of diabetes, severity of hearing loss, pattern of hearing loss in audiogram and presence of associated symptoms, (tinnitus, vertigo). Treatment protocol with intravenous hydrocortisone, intravenous dextran and oral anti-viral agent was followed. Pre-treatment and post-treatment pure tone average was analyzed. With combination therapy the overall improvement in pure tone threshold was seen in 27 patients (79.4%). A statistically significant association was found between the time at which medical intervention was started and hearing improvement. Early intervention in patients presenting before 3 days has given 77.8% complete type 1 recovery Idiopathic sudden sensory neural hearing loss is a medical emergency. It should not be misdiagnosed. Early detection and management with volume expanders, steroids and antivirals will improve the chances of complete recovery.

Keywords Sudden sensorineural hearing loss · Idiopathic hearing loss

H. Vijayendra · G. Buggaveeti · B. Parikh (✉) · R. Sangitha
Vijaya ENT Care Centre, Malleswaram, Bangalore, India
e-mail: drbhavinparikh@hotmail.com

Introduction

Idiopathic sudden sensory neural hearing loss (SSNHL) has been defined as 30 dB or more sensorineural hearing loss over at least three contiguous audiometric frequencies occurring within three days or less. 15,000 new cases are reported annually world wide accounting for approximately 1% of all cases of sensorineural hearing loss [1]. Hearing loss is usually unilateral and frequently is accompanied by tinnitus (70%), vertigo or milder sensation of spatial disorientation is present in 50% of cases. Hearing loss may be mild in a restricted frequency range or it may be total [2].

The etiology of SSNHL is idiopathic but viral infection, vascular compromise, disruption of cochlear membrane; immunological diseases and otological tumors are discussed [2]. Upto one-third of patients have upper respiratory prodrome preceding SSNHL suggesting the role of viruses in the etiology of this disease [3]. The alterations in microcirculation of cochlea have long been suggested to be a cause of SSNHL [3]. The blood supply to cochlea is maintained by labyrinthine artery, which has no collateral vasculature. Vascular disorders may cause SSNHL because of cochlear injury and dysfunction secondary to anoxia or hypoxia [4].

Treatment of SSNHL should be started as soon as possible, should be aggressive and should cover those disorders that are most likely to benefit [1]. Treatment is not helpful after 30 days have passed because active disease may have resolved and damage may be permanent. High dosage systemic steroid therapy is currently the mainstay of the treatment for SSNHL as a result of its high anti-inflammatory effect [5]. They also reduce cytotoxic immune response, increase the micro vascular blood flow in the cochlea and decrease the onset of endolymphatic

hydrops. Low molecular weight dextrans cause expansion of plasma volume and increase in cardiac output, increase in vascular perfusion and microcirculation, reduction in blood viscosity; platelet adhesiveness and rouleau formation [2]. Antiviral agents like acyclovir or valacyclovir should be prescribed for SSNHL considering the viral infection being the probable etiology. In the present study we reviewed our experience with combination regime of low molecular weight dextrans, steroid therapy, vasodilators and antiviral.

Materials and Methods

The present prospective study was carried out in 34 patients presenting with idiopathic SSNHL between 2005 and 2009. Evaluation and management of SSNHL is considered as a medical emergency. A careful history regarding trauma, intense noise exposure is noted. Most of the patients complain of sudden loss of hearing on drinking cold fridge water, on exposure to cool breeze, on taking cold water head bath early in the morning, traveling in air conditioned bus or train. More than 50% complain of blocked sensation in the ear or paraesthesias in front of ear. Patients with Meniere's disease, fluctuating HL were excluded. General physical examination is done. Otoscopy, fistula testing, neurotological examination including cranial nerves, cerebellar function is performed [1]. Tuning fork tests with 256, 516 and 1024 Hz is done in all patients. Hearing loss was measured with pure tone audiogram (PTA). Extended Fletcher Index (EFI) was used to calculate hearing loss. EFI is mean hearing loss for 500, 1000, 2000 and 3000 Hz [6].

For all 34 patients standard treatment protocol was followed.

1. Intravenous dextrans in 5% dextrose 500 ml was infused slowly over 4–6 h. This is followed 2 h later by another 500 ml if the patient had subjective improvement. Most of the patients have remarkable improvement of tinnitus following first dose of dextrans. Patient is kept under continuous supervision while giving dextrans as they may cause cardiac overload. Audiometry is repeated on the second day. If the patient has improvement another two units of 500 ml of 5% dextrose with dextrans are infused over 6–8 h. From third day onwards patient is put on oral xanthinol nicotinate 150 mg twice daily for 1 month.
2. Intravenous hydrocortisone 100 mg was given twice daily for 2 days. This is followed by oral steroids in full therapeutic dose (either prednisolone 1 mg/kg or methylprednisolone 0.8 mg/kg) for 5 days. Dose is tapered over the next 10 days.
3. Oral Valacyclovir 500 mcg three times a day was given for 10 days to those patients who presented within 10 days.
4. Neurovitamins were given.
5. Aspirin 75 mg twice daily is given to prevent thrombus formation.

In diabetics dextrans were infused along with normal saline instead of 5% dextrose and oral steroids were avoided.

Data Analysis

Severity of SSNHL [7] is classified as Mild: 41–55 dB, Moderate: 56–70 dB, Severe: 71–90 dB and Profound $>$ 90 dB HL.

Definition of Recovery

The specific definition of what constitutes "improvement" or "recovery" after an SSNHL is not uniform among studies and reports [8]. Since most patients did not have audiograms before the SSNHL episode, the hearing in unaffected normal ear as measured at the time of diagnosis was used as standard [9]. We adopted following definition for recovery considering normal hearing contralateral ear as indicator of status of affected ear prior to hearing loss.

1. Complete (Type 1) recovery: The recovery of hearing to within 10 dB of contralateral ear hearing pure tone average.
2. Partial (Type 2) recovery: The recovery of hearing to within 50% or more of contralateral ear hearing loss pure tone average.
3. No (Type 3) recovery: Less than 50% recovery of hearing.

Results

Thirty-four patients with unilateral sudden onset sensory neural deafness were included in the study. The mean age was 43.2 years (range 24–62). There were 24 women and 10 men. The right and left years were affected in 26 and 8, respectively. Most of the patients complained of concomitant onset of tinnitus with hearing loss. Twenty-four patients complained of vertigo (70%). According to the level of initial hearing loss, patients were divided into four classes of hearing loss. Moderate hearing loss was detected in 3 (8.8%), moderate-severe hearing loss in 11 (32.3%), severe hearing loss in 18 (52.9%) and profound hearing loss in 2 (5.8%) patients.

Types of hearing loss pattern in pure tone audiogram were observed as ascending in 7 (20.5%), descending in 4

Table 1 Comparison of extent of recovery with the severity of hearing loss at the time of presentation

Type of recovery	Moderate	Moderate severe	Severe	Profound	Total
Complete (type 1)	3	9	6	0	18
Partial (type 2)	–	2	6	1	9
No recovery (type 3)	–	–	6	1	7
Total	3	11	18	2	34

Table 2 Comparison of extent of improvement and the time of presentation after sudden sensorineural hearing loss

Time of presentation at hospital	Improved	Not improved	Total
<72 h (3 days)	17	1	18
>72 h	10	6	16
Total	27	7	34

Fishers exact test: Two tailed P value = 0.0348 (statistically significant)

(11.8%), flat in 20 (58.8%) and total in 3 (8.8%). According to classification for extent of recovery, complete recovery (Type 1) was seen in 18 (52.9%), partial recovery (Type 2) was seen in 9 (26.4%) and no recovery (Type 3) was seen in 7 (20.8%) (Table 1). For the purpose of statistical analysis, complete recovery and partial groups are combined in recovery group. 79.4% of the treated patients got improvement. Subjective improvement of tinnitus was perceived in all the patients with type 1 and type 2 recoveries after first dose of intravenous dextrans. Of the 16 patients with vertigo, 13 got relief (81.23%). Two out of seven patients with type 3 recovery had diabetes. They presented with severe to profound deafness and they did not improve with intravenous dextrans and antivirals. Steroids were avoided in these patients.

Patients have been divided into two groups depending on the time gap between onset of sudden hearing loss and their presentation at the tertiary care centre (Table 2). Fourteen patients (77.8%) presenting before 3 days had type 1 recovery whereas only four patients (25%) presenting after 3 days had type 1 recovery. With combination therapy the overall improvement in pure tone threshold was seen in 27 patients (79.4%) which were more than spontaneous recovery. A statistically significant association was found between the time of presentation and hearing improvement.

Discussion

Evaluation and management of sudden sensory neural hearing loss should be considered as a medical urgency, if not an emergency [1]. A surprising number of patients report that the hearing loss was noticed immediately on awakening suggesting that hearing loss occurred during sleep [8]. Some patients say that they heard a pop sound in the ear following which they had become deaf. Delicacy of the structures involved makes elucidation of and interference with the pathophysiology difficult [6]. The possible mechanism for vascular cause of SSNHL includes hemorrhage, thrombosis, embolism, vasospasm and hypercoagulability [4].

In our series, history suggested that most of the patients developed hearing loss following exposure to cool breeze, traveling in air conditioned bus. Senior author suspects vasospasm as the most common etiology in these cases. History, simple tuning fork tests and pure tone audiogram help us to arrive at the correct diagnosis. Often, SSNHL is mistaken for Meniere's disease due to vestibular symptoms.

The duration of time from the onset of symptoms until presentation at the otolaryngologists office may be one of the most important factors in determining a patients prognosis. Most patients do not seek advice immediately at the onset of symptoms and the typical presentation is generally delayed 48 to 96 h [8]. In our series out of the 18 patients who presented within 3 days, 15 (77.8%) had type 1 recovery, 2 (11.1%) had type 2 recovery and 1 (5.5%) had type 3 recovery. 4 (25%) of the patients presenting after 3 days had type 1 recovery, 6 (37.5%) had type 2 recovery and 6 (37.5%) had type 3 recovery.

Medical treatment should be started immediately after the diagnosis without wasting valuable time. Recently a retrospective study conducted by Zadeh and Storper et al. [10] demonstrated that antiviral therapy and increased length of steroid treatment has given 73% recovery. Combination therapy of vasodilators, steroids and antivirals has given improvement in 79.4% in our case series. Mattox et al. [11] found that shape of the initial audiogram was related to recovery and patients with low frequency or mid frequency (u-shaped) audiogram contour showed a better recovery. The results of present study showed that more severe hearing loss, delayed presentation and associated diabetes were poor prognostic factors.

Conclusion

SSNHL is a medical emergency. It should not be misdiagnosed. Early detection and management with vasodilators, steroids and antivirals will improve the chances of complete recovery.

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