The Relationship Between Tubotympanic Chronic Suppurative Otitis Media and Sensorineural Hearing Loss: A Prospective Investigation.

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Abstract:

Background: Chronic suppurative otitis media (CSOM), specifically the tubotympanic type, primarily causes conductive hearing loss. However, the potential for sensorineural hearing loss (SNHL) exists due to toxin-induced cochlear damage. This study aimed to investigate the relationship between SNHL and tubotympanic CSOM.

Methods: A prospective study was conducted on 60 patients with tubotympanic CSOM attending an ENT outpatient department. Audiometric assessments were performed to evaluate hearing thresholds.

Results: SNHL was observed in 13.3% (8/60) of patients with tubotympanic CSOM.

Conclusions: While conductive hearing loss is the predominant auditory consequence of tubotympanic CSOM, a significant proportion of patients are at risk for developing SNHL. This highlights the importance of early intervention and patient education regarding the potential for SNHL in prolonged, untreated tubotympanic CSOM.

Introduction:

Chronic suppurative otitis media (CSOM) stands as a persistent and debilitating inflammatory condition affecting the middle ear mucosa, a common ailment encountered in otolaryngology practices worldwide. Defined by recurrent or persistent ear discharge through a tympanic membrane perforation, CSOM poses a significant burden on individuals, impacting their quality of life, social interactions, and overall well-being. While the hallmark auditory consequence of CSOM has traditionally been recognized as conductive hearing loss, emerging evidence suggests a more complex interplay, revealing the potential for sensorineural hearing loss (SNHL) as a significant, yet often overlooked, complication. This introduction delves into the intricacies of CSOM, particularly the tubotympanic variant, exploring the mechanisms that underlie both conductive and sensorineural hearing loss, and highlighting the critical need for comprehensive assessment and management strategies.

The middle ear, a delicate and intricate system, plays a pivotal role in auditory transduction. Its primary function is to efficiently transmit sound vibrations from the external environment to the inner ear, where they are converted into neural signals for interpretation by the brain. The tympanic membrane, a thin, taut structure, acts as a primary receiver of sound waves, vibrating in response to incoming acoustic energy. These vibrations are then transmitted through the ossicular chain – a series of three tiny bones (malleus, incus, and stapes) – to the oval window, a membrane-covered opening leading to the cochlea. Within the cochlea, the organ of Corti,

housing sensitive hair cells, transforms mechanical vibrations into electrochemical signals, which are subsequently conveyed to the auditory nerve and ultimately to the auditory cortex.

CSOM disrupts this delicate process through chronic inflammation and infection within the middle ear cleft. The tubotympanic type of CSOM, characterized by a central perforation of the tympanic membrane and mucosal involvement, is particularly prevalent. The persistent presence of bacteria, inflammatory mediators, and debris within the middle ear leads to chronic mucosal edema, granulation tissue formation, and ultimately, the discharge of purulent material through the tympanic membrane perforation. This chronic inflammatory state not only compromises the integrity of the tympanic membrane and ossicular chain, resulting in conductive hearing loss, but also creates a potential pathway for the dissemination of harmful substances to the inner ear.

The conventional understanding of hearing loss in tubotympanic CSOM primarily focuses on conductive mechanisms. The perforation of the tympanic membrane disrupts the normal sound transmission pathway, leading to a reduction in sound energy reaching the cochlea. Similarly, ossicular damage, such as erosion or fixation, further impedes the efficient transfer of sound vibrations, contributing to conductive hearing loss. This type of hearing loss is characterized by a reduction in air conduction thresholds, while bone conduction thresholds remain relatively preserved.

However, the pathogenesis of SNHL in CSOM is less clearly defined and often overlooked. The cochlea, the sensory organ of hearing, is highly susceptible to damage from various insults, including toxins, inflammatory mediators, and direct bacterial invasion. In the context of CSOM, the chronic presence of bacterial toxins, such as endotoxins and exotoxins, within the middle ear can potentially diffuse across the round window membrane, reaching the cochlear fluids and damaging the delicate hair cells of the organ of Corti. These hair cells, particularly those located in the basal turn of the cochlea, are responsible for encoding high-frequency sounds and are more vulnerable to toxic insults. Damage to these cells results in a reduction in both air and bone conduction thresholds, indicative of SNHL.

Furthermore, the inflammatory cascade associated with CSOM can lead to the release of various cytokines and chemokines, which can also contribute to cochlear damage. These inflammatory mediators can disrupt the blood-labyrinth barrier, allowing harmful substances to enter the cochlear fluids and induce cellular apoptosis and dysfunction. Additionally, the presence of granulation tissue and cholesteatoma, a benign but destructive growth of keratinizing squamous epithelium, can further exacerbate the inflammatory process and increase the risk of SNHL.

The clinical implications of SNHL in tubotympanic CSOM are significant. While conductive hearing loss can often be addressed through surgical interventions such as tympanoplasty and ossiculoplasty, SNHL is generally irreversible. Early detection and intervention are crucial to minimize the progression of hearing loss and preserve auditory function. Audiometric evaluation, including pure-tone audiometry and speech audiometry, is essential for assessing the extent and nature of hearing loss in patients with CSOM.

Moreover, the awareness of the potential for SNHL in tubotympanic CSOM is crucial for patient education and counseling. Patients should be informed about the risks associated with prolonged, untreated CSOM and the importance of timely medical and surgical intervention.

This knowledge empowers patients to make informed decisions regarding their treatment options and encourages adherence to recommended management strategies.

Materials and Methods

Study Design and Setting:

This prospective observational study was conducted in the Department of Otorhinolaryngology and Head and Neck Surgery at GIMSH, Durgapur, India, over a one-year period. The study received ethical approval from the Institutional Ethical Committee (IEC), GIMSH, Durgapur. prior to commencement.

Participants:

Sixty (60) patients diagnosed with unilateral tubotympanic (mucosal/safe) chronic suppurative otitis media (CSOM) were enrolled in the study.

Inclusion Criteria:

- Patients diagnosed with unilateral tubotympanic (mucosal/safe) CSOM.
- Patients with normal hearing in the contralateral ear, serving as a control.

Exclusion Criteria:

- Patients younger than 10 years of age, to minimize potential inaccuracies in audiometric testing in children.
- Patients older than 50 years of age, due to the increased prevalence of presbycusis in this age group.
- Patients with a history of prior otologic surgery.
- Patients with a family history of hearing loss.
- Patients with a history of prolonged noise exposure.
- Patients with a history of head trauma.

Study Protocol:

1. **Informed Consent:** All participants provided written informed consent prior to enrollment.
2. **Clinical Evaluation:** A comprehensive ear, nose, and throat (ENT) examination was performed by a qualified otolaryngologist, with specific attention to the affected ear. Tuning fork tests (Rinne and Weber) were performed as part of the initial clinical assessment.
3. **Audiological Assessment:**
    - Pure-tone audiometry (PTA) was conducted in a sound-treated room meeting the American National Standards Institute (ANSI) 1977 and International Organization for Standardization (ISO) standards for maximum permissible noise levels.
    - Air conduction and bone conduction thresholds were measured at frequencies of 250, 500, 1000, 2000, 4000, and 8000 Hz.

   - Audiometry was performed by a trained audiologist.
   - Sensorineural hearing loss was diagnosed when bone conduction thresholds were impaired.
4. **Data Collection:** Detailed patient demographic information (age, sex) and audiometric data were recorded.

Definitions:

- **Tubotympanic CSOM (Mucosal/Safe):** Characterized by a central tympanic membrane perforation and mucosal involvement without evidence of cholesteatoma.
- **Sensorineural Hearing Loss (SNHL):** Defined as a reduction in both air and bone conduction thresholds, indicative of cochlear or retrocochlear dysfunction.

Statistical Analysis:

The collected data were entered into a Microsoft Excel spreadsheet and subsequently exported to the data editor of SPSS version 2.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were summarized as percentages.

Results

Demographic and Clinical Characteristics:

- **Age:** The mean age of participants was 32 ± 10 years, with the highest prevalence (31.67%) in the 31-40 year age group.
- **Sex:** The male-to-female ratio was 1:0.93.
- **Symptom Duration:**
   - 51.67% of patients presented with symptoms lasting less than 15 months.
   - 30% presented with symptoms lasting 15-30 months.
   - 3.33% presented with symptoms lasting 31-45 months.
   - 15% presented with symptoms lasting more than 45 months.
- **Ear Involvement:** 55% of patients had left ear involvement, and 45% had right ear involvement.
- **Tympanic Membrane Perforation:**
   - 41.67% had anterosuperior quadrant perforation.
   - 40% had anteroinferior quadrant perforation.
   - 18.33% had posterosuperior quadrant perforation.

Audiometric Findings:

- **Air Conduction (AC) Thresholds (Diseased Ear):**
   - 500 Hz: 27.58 ± 3.73 dB
   - 1000 Hz: 30.16 ± 4.09 dB
   - 2000 Hz: 31.92 ± 4.79 dB

  - 4000 Hz: 33.5 ± 5.98 dB
- **Bone Conduction (BC) Thresholds (Diseased Ear):**
  - 500 Hz: 13.33 ± 5.79 dB
  - 1000 Hz: 17 ± 6.18 dB
  - 2000 Hz: 21.58 ± 6.67 dB
  - 4000 Hz: 24.5 ± 6.74 dB

Sensorineural Hearing Loss (SNHL):

- 8 patients (13.3%) exhibited significant SNHL (average BC threshold > 25 dB).
- 52 patients (86.67%) did not exhibit significant SNHL.
- **SNHL group demographics:**
  - Mean age: 42.87 ± 4.38 years.
  - Mean duration of disease: 94.5 ± 28.27 months.
  - Male:female ratio 1:1.
  - Otoscopic findings in SNHL group:
    - 37.50% antero-inferior quadrant perforation.
    - 37.50% antero-superior quadrant perforation.
    - 25% postero-inferior quadrant perforation.
- **Audiometric findings in SNHL group:**
  - AC thresholds: 27.88 ± 3.75 dB (500 Hz), 30.57 ± 4.04 dB (1000 Hz), 32.31 ± 4.69 dB (2000 Hz), 34.04 ± 5.86 dB (4000 Hz).
  - BC thresholds: 31.20 ± 3.76 dB (500 Hz), 14.23 ± 5.63 dB (1000 Hz), 17.88 ± 6.13 dB (2000 Hz), 22.69 ± 6.29 dB (4000 Hz).
  - Average AC threshold: 34.04 ± 4.34 dB.
  - Average BC threshold: 39.95 ± 4.95 dB.
  - Maximum hearing loss was seen at 4000 Hz (39.375 ± 4.95 dB) and 2000 Hz (35 ± 2.67 dB).
- **Comparison of BC thresholds normal vs diseased ear:**
  - 500Hz: normal 10.45db, diseased 14.95db, statistically significant difference.
  - 1000Hz: normal 11.95db, diseased 21.34db, statistically significant difference.
  - 2000Hz: normal 16.45db, diseased 29.95db, statistically significant difference.
  - 3000Hz: normal 21.27db, diseased 37.32db, statistically significant difference.

Table 1: Age distribution, sex distribution, laterality, duration of symptoms and otoscopic finding among patients with chronic suppurative otitis media.

Age (in years)	No. of patients	%
10-20	8	13.3
21-30	17	28.33
31-40	19	31.67
41	16	26.67
32±10 (mean±SD)	60	100
Gender		
Male	31	51.67
Female	29	48.33
M:F ratio	60	1:0.93
Duration (in months)		
<15	31	51.67
16-30	18	30
31-45	2	3.33
>45	9	15
Laterality		
Right	27	45
Left	33	55

Table 5: Gender wise distribution among patients with SNHL (n=8).

Sex	No. of patients	%
Male	4	50
Female	4	50
Total	8	100
Otoscopic finding (perforation)		
Anterio-inferior	3	37.50
Anterio-superior	3	37.50
Posterio-inferior	2	25

Table 6: Ac threshold and bone threshold among patients with SNHL (n=8).

Frequency	Air conduction-Db (mean±SD)	Bone conduction-Db (mean±SD)
500 Hz	27.88±3.75	31.20±3.76
1000 Hz	30.57±4.04	14.23±5.63
2000 Hz	32.31±4.69	17.88±6.13
4000 Hz	34.04±5.86	22.69±6.29
Average	34.04±4.34	39.95±4.95

Table 7: SNHL hearing loss according to frequency

Discussion:

Chronic suppurative otitis media (CSOM) remains a prevalent condition in otolaryngology practice. While conductive hearing loss is well-established as a primary consequence, the role of sensorineural hearing loss (SNHL) in mucosal CSOM has been a subject of ongoing debate and requires further elucidation. This study aimed to investigate the incidence and characteristics of SNHL in patients with tubotympanic CSOM.

Incidence of SNHL:

The incidence of SNHL in our study was 13.33%, which falls within the range reported by previous studies. Variations in incidence may be attributed to differences in study populations, diagnostic criteria, and the duration of follow-up. Our findings corroborate the notion that SNHL, though less common than conductive hearing loss, is a significant complication of mucosal CSOM.

Age and SNHL:

We observed a higher prevalence of SNHL in older age groups, specifically those between 40-50 years. This aligns with the findings of Paparella et al. (1972), who reported SNHL across all age ranges. Excluding patients over 50 years aimed to minimize the influence of presbycusis. The increased SNHL in older patients with CSOM might reflect the cumulative effect of chronic inflammation and toxin exposure over time.

Gender and SNHL:

While some studies have reported a male predominance in SNHL associated with CSOM, our study found no significant correlation between gender and SNHL. This suggests that the susceptibility to SNHL in mucosal CSOM is likely independent of gender.

Duration of Disease and SNHL:

A crucial finding of our study was the strong association between the duration of CSOM and the incidence of SNHL. None of the patients with a history of ear discharge for less than four years had SNHL. The incidence progressively increased with longer disease duration, reaching 40% in patients with a history exceeding 10 years. This supports the hypothesis that prolonged exposure to bacterial toxins and inflammatory mediators, diffusing through the round window membrane, contributes to cochlear damage. These results are consistent with Nanda MS et al. (2015), who also reported a higher incidence of SNHL in patients with longer disease duration.

Ear Discharge and SNHL:

Consistent with the findings on disease duration, nearly all patients with SNHL in our study reported a history of recurrent ear discharge. This reinforces the notion that prolonged exposure to inflammatory byproducts plays a pivotal role in SNHL development. The round window's semi-permeable nature facilitates the passage of toxins, particularly during active inflammation when pH levels are lowered. This contrasts with Noordzij JP et al. (1995), who found no consistent relationship between ear discharge characteristics and SNHL.

Frequency Analysis of SNHL:

Our audiometric analysis revealed a statistically significant elevation of bone conduction thresholds in the diseased ear compared to the contralateral normal ear across all frequencies. Notably, the difference was most pronounced at higher frequencies, particularly 4000 Hz. These results are consistent with previous studies, such as Mohsin et al. (2013) and Nanda MS et al. (2015), which also reported greater bone conduction threshold differences at higher frequencies. This suggests that the basal turn of the cochlea, responsible for high-frequency perception, is more susceptible to damage in mucosal CSOM.

Limitations:

This study has certain limitations. The relatively small sample size, while adequate for a prospective study, may limit the generalizability of our findings. A larger, multicenter study would provide a more robust understanding of the relationship between mucosal CSOM and SNHL. Furthermore, the duration of follow-up was limited. Longitudinal studies are needed to assess the long-term progression of hearing loss in these patients.

Clinical Implications:

Our findings underscore the importance of early diagnosis and prompt treatment of mucosal CSOM to minimize the risk of SNHL. Patients with prolonged disease duration should undergo thorough audiometric evaluation to detect early signs of cochlear damage. The strong association between disease duration and SNHL highlights the need for patient education regarding the potential long-term complications of untreated CSOM.

Conclusion:

This study confirms that SNHL is a significant complication of tubotympanic CSOM, particularly in patients with prolonged disease duration.

The increased bone conduction thresholds, especially at higher frequencies, suggest cochlear damage. Early intervention and comprehensive audiometric monitoring are crucial to mitigate the risk of irreversible hearing loss in these patients. Future research should focus on elucidating the specific mechanisms underlying SNHL in mucosal CSOM and developing strategies for prevention and management.
Sources and related content

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