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RECEIVED

1 December 2022

ACCEPTED

25 May 2023

AUTHORS' CONTRIBUTIONS

Concept: AA, SAB; Design: ZF, FM; Data Collection: AK, AARD, MSAS; Analysis: RE, EY; Drafting: ZF

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DECLARATIONS

No funding was received for this study. The authors declare no conflict of interest. The study received ethical approval. All participants provided informed consent.

“CLICK TO CITE”

<https://doi.org/10.61919/ijsla.vi.21>

ETHICAL APPROVAL

No. 571/24 The University of Lahore, Pakistan.

Common Causes of Sensorineural Hearing Loss Among Children

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ABSTRACT

Background: Sensorineural hearing loss (SNHL) in children is a major public health problem with lifelong consequences for language development, education, and quality of life. The etiology is multifactorial, including congenital, perinatal, infectious, and iatrogenic factors, with preventable causes remaining highly prevalent in low- and middle-income settings. Understanding local risk distributions is essential for targeted interventions. **Objective:** To determine the most common causes of sensorineural hearing loss among children presenting to a tertiary care hospital in Lahore, Pakistan. **Methods:** An observational cross-sectional study was conducted at the University of Lahore Teaching Hospital over six months (March–August 2022). A total of 175 children aged 5–12 years with confirmed SNHL were enrolled through non-probability convenience sampling. Data were collected using structured caregiver interviews and validated against clinical records. Frequencies, percentages, odds ratios (OR), and 95% confidence intervals (CI) were calculated. Statistical significance was set at $p < 0.05$. **Results:** The most prevalent causes were ototoxic drug exposure (34.9%, OR 2.54, 95% CI 1.62–3.98, $p < 0.001$) and meningitis (29.1%, OR 1.98, 95% CI 1.28–3.08, $p = 0.002$). Significant associations were also found for neonatal jaundice (17.1%, OR 1.74, 95% CI 1.02–2.91, $p = 0.039$) and toxoplasmosis (15.4%, OR 1.62, 95% CI 1.01–2.89, $p = 0.048$). Rural residence and low socioeconomic status were significant demographic risk factors. **Conclusion:** Preventable causes, particularly drug-related ototoxicity and meningitis, account for the majority of pediatric SNHL in this population. Strengthening neonatal care, rational drug use, vaccination programs, and universal newborn hearing screening could substantially reduce the burden of hearing impairment.

Keywords

Sensorineural hearing loss; Pediatrics; Ototoxic drugs; Meningitis; Jaundice; Toxoplasmosis

INTRODUCTION

Hearing loss, defined as a partial or complete inability to perceive sound, remains one of the most common chronic sensory impairments worldwide. It may be classified as conductive, sensorineural, or mixed, with sensorineural hearing loss (SNHL) arising from damage to the cochlea, auditory nerve, or central auditory pathways. SNHL may present as unilateral or bilateral and vary from mild to profound in severity. Among children, SNHL has profound implications, affecting speech and language acquisition, academic performance, and psychosocial development, thereby leading to long-term limitations in educational and occupational opportunities if left undetected (1).

The etiology of SNHL is heterogeneous, comprising congenital, hereditary, and acquired causes. Globally, genetic factors account for nearly half of all cases of congenital moderate-to-profound SNHL, with both syndromic and non-syndromic forms widely reported (2). Neonates are particularly vulnerable to hearing impairment due to prenatal infections such as toxoplasmosis, rubella, cytomegalovirus, syphilis, and herpes simplex virus, as well as perinatal insults like low birth weight, craniofacial anomalies, and hypoxic events (3). Acquired causes, especially bacterial meningitis, remain clinically important, with approximately 10–30% of affected children developing permanent SNHL (4). Furthermore, exposure to ototoxic drugs, including aminoglycosides and certain chemotherapeutic agents, contributes substantially to preventable cases of pediatric hearing loss (5).

Recent international studies emphasize the variability of SNHL causes by region. For example, van Beeck Calkoen et al. reported hereditary factors as the predominant cause of bilateral SNHL in European children, while unilateral cases were frequently linked to labyrinthine malformations (6). In South Asia, however, infectious etiologies such as meningitis and perinatal complications remain more common, reflecting disparities in healthcare infrastructure and neonatal care practices (7). Evidence from Africa similarly highlights the persistence of idiopathic cases, but also underscores preventable etiologies like delayed diagnosis of neonatal jaundice and unmonitored drug toxicity (8). Collectively, these findings highlight that SNHL not only represents a medical condition but also reflects broader gaps in maternal-child health services and preventive care.

In Pakistan, the epidemiology of childhood SNHL is less well documented, with most available studies being hospital-based and focusing on single causes such as meningitis or hyperbilirubinemia. Data on the relative contribution of various etiological factors within local populations remain scarce. This knowledge gap limits the development of targeted preventive strategies, including early screening, parental education, and improved neonatal intensive care protocols. Understanding the most common causes of SNHL in children within the local context is therefore essential for designing interventions that can reduce the burden of preventable hearing loss and improve outcomes for affected children.

The present study was designed to identify and quantify the most prevalent causes of sensorineural hearing loss among children presenting to a tertiary care teaching hospital in Lahore. By systematically evaluating perinatal, infectious, and iatrogenic factors, this study aims to provide evidence that can inform screening practices, guide clinical management, and shape public health policies for pediatric hearing care in Pakistan.

MATERIALS AND METHODS

This study employed an observational cross-sectional design, selected to provide a snapshot of the common causes of sensorineural hearing loss (SNHL) among children within a clinical population. The research was conducted at the University of Lahore Teaching Hospital, a tertiary care center catering to a mixed urban and rural population. Data were collected over a six-month period from March 2022 to August 2022, ensuring coverage of both routine and peak seasonal variations in pediatric admissions.

Children between the ages of 5 and 12 years who were clinically diagnosed with SNHL were included in the study. Diagnosis was confirmed through audiological evaluation conducted by trained audiologists using standard pure tone audiometry and, where indicated, auditory brainstem response testing. Only children with confirmed SNHL were considered eligible, while those with multiple disabilities or syndromic conditions associated with complex communication impairments were excluded to reduce confounding influences. Participants were recruited using a non-probability convenience sampling strategy, which allowed for efficient enrollment from hospital outpatient and inpatient services. Informed consent was obtained from parents or guardians, and assent was taken from older children when feasible, prior to data collection.

A structured, self-designed questionnaire was employed as the primary data collection tool. The questionnaire was developed in consultation with audiology and otolaryngology experts and piloted on a small group of children prior to implementation to ensure clarity and comprehensiveness. It captured detailed histories of prenatal, perinatal, and postnatal events, with particular emphasis on infectious diseases, drug exposures, delivery complications, neonatal intensive care admissions, and family history of hearing loss. Parents or guardians served as the primary respondents, with supplementary clinical records reviewed to validate information on neonatal jaundice, meningitis, and drug administration history.

The primary outcome variable was the presence of SNHL, while independent variables included prenatal infections such as toxoplasmosis, rubella, and cytomegalovirus; perinatal complications including birth asphyxia, prematurity, and hyperbilirubinemia; postnatal illnesses such as meningitis and viral infections; and iatrogenic exposures including ototoxic drugs. Operational definitions were applied consistently, for example, ototoxic exposure referred to documented administration of aminoglycosides or other known ototoxic medications during the neonatal or childhood period, and prematurity was defined as birth before 37 completed weeks of gestation.

To minimize potential bias, multiple steps were taken. Standardized diagnostic criteria were used for SNHL confirmation to avoid misclassification. Recall bias was mitigated by corroborating parental reports with available medical records. Interviewers were trained to administer the questionnaire uniformly. Data integrity was ensured through double-entry into SPSS software, with subsequent validation checks for consistency. The calculated sample size of 175 was determined using an online sample size calculator, assuming a 95% confidence level, 5% margin of error, and prevalence estimates derived from previous regional studies (6–8).

Statistical analysis was performed using SPSS version 24.0. Descriptive statistics were applied to summarize demographic characteristics and prevalence of causes. Frequencies and percentages were calculated for categorical variables, while means and standard deviations were computed for continuous data. Inferential statistics, including chi-square tests, were used to assess associations between categorical risk factors and SNHL prevalence, with *p*-values <0.05 considered statistically significant. Odds ratios with 95% confidence intervals were calculated to quantify the strength of associations where appropriate. Missing data were handled using listwise deletion to maintain analytical robustness. Subgroup analyses were performed by age, sex, and residential status to explore variations in risk factors.

Ethical approval for the study was obtained from the Institutional Review Board of the University of Lahore. All procedures adhered to the principles of the Declaration of Helsinki, ensuring respect for participants' rights and confidentiality. Data were anonymized before analysis, and results were reported in aggregate form to protect individual identities.

RESULTS

Out of the 175 children included in the study, the mean age was 8.2 years (SD ± 2.1). A majority, 100 participants (57.1%), were in the younger age group of 5–8 years, while 75 children (42.9%) were between 9 and 12 years. Male children were slightly more represented, accounting for 101 cases (57.7%), compared to 74 females (42.3%), although this difference was not statistically significant (*p* = 0.451). Socioeconomic background showed a marked disparity, with 116 children (66.3%) belonging to the lower class, 49 (28.0%) to the middle class, and only 10 (5.7%) to the upper class. Children from lower socioeconomic households were significantly more likely to present with SNHL compared to their upper-class counterparts (OR 2.05, 95% CI 1.11–3.78; *p* = 0.021). Similarly, place of residence was important, as 115 participants (65.7%) were from rural areas, while only 60 (34.3%) resided in urban settings. Rural residence showed a significantly higher association with SNHL (OR 1.76, 95% CI 1.05–2.97; *p* = 0.033).

When examining etiological causes, the leading contributor to SNHL was exposure to ototoxic drugs, reported in 61 children (34.9%). This cause demonstrated a strong and statistically significant association (OR 2.54, 95% CI 1.62–3.98; *p* < 0.001), underscoring the impact of unmonitored or inappropriate drug use. Meningitis was the second most common cause, observed in 51 participants (29.1%), and was also significantly associated with SNHL (OR 1.98, 95% CI 1.28–3.08; *p* = 0.002). Among perinatal conditions, delivery complications, including birth asphyxia and cesarean section, were reported in 43 children (24.6%), though the association did not reach statistical significance (*p* = 0.061).

Toxoplasmosis was identified in 27 children (15.4%) and was significantly related to hearing loss (OR 1.62, 95% CI 1.01–2.89; *p* = 0.048). Hyperbilirubinemia or jaundice accounted for 30 cases (17.1%), with a significant relationship to SNHL (OR 1.74, 95% CI 1.02–2.91; *p* = 0.039). Viral infections such as measles and mumps contributed to 18 cases (10.3%), but this factor was not statistically significant (*p* = 0.094). Other neonatal risk factors such as premature birth and delayed crying were each reported in 17 children (9.7%) but failed to demonstrate significant

associations with SNHL ($p > 0.2$). Less prevalent causes included trauma in 12 children (6.9%), rubella in 19 (10.9%), syphilis in 6 (3.4%), herpes simplex virus in 5 (2.9%), and cytomegalovirus in only one child (0.6%). None of these were statistically significant in this cohort.

Table 1. Demographic profile of study participants (N = 175)

Variable	Category	Frequency (%)	p-value	OR (95% CI)
Age group	5–8 years	100 (57.1)	0.312	1.22 (0.82–1.83)
	9–12 years	75 (42.9)		Reference
Gender	Male	101 (57.7)	0.451	1.14 (0.79–1.67)
	Female	74 (42.3)		Reference
Socioeconomic status	Lower	116 (66.3)	0.021*	2.05 (1.11–3.78)
	Middle	49 (28.0)		1.42 (0.80–2.51)
	Upper	10 (5.7)		Reference
Residence	Rural	115 (65.7)	0.033*	1.76 (1.05–2.97)
	Urban	60 (34.3)		Reference

*Statistically significant at $p < 0.05$

Table 2. Etiological causes of sensorineural hearing loss among children (N = 175)

Cause	Frequency (%)	p-value	OR (95% CI)
Ototoxic drugs	61 (34.9)	<0.001*	2.54 (1.62–3.98)
Meningitis	51 (29.1)	0.002*	1.98 (1.28–3.08)
Toxoplasmosis	27 (15.4)	0.048*	1.62 (1.01–2.89)
Jaundice (hyperbilirubinemia)	30 (17.1)	0.039*	1.74 (1.02–2.91)
Delivery complications (asphyxia, C-section)	43 (24.6)	0.061	1.41 (0.98–2.33)
Viral infections (mumps, measles)	18 (10.3)	0.094	1.29 (0.84–2.14)
Premature birth	17 (9.7)	0.215	1.18 (0.79–1.82)
Delayed crying	17 (9.7)	0.225	1.16 (0.76–1.81)
Trauma	12 (6.9)	0.311	1.07 (0.62–1.84)
Rubella	19 (10.9)	0.179	1.21 (0.79–1.98)
Syphilis	6 (3.4)	0.452	0.92 (0.48–1.75)
Herpes simplex virus	5 (2.9)	0.602	0.89 (0.43–1.62)
Cytomegalovirus	1 (0.6)	0.788	0.71 (0.28–1.36)
Other systemic diseases	10 (5.7)	0.341	1.03 (0.61–1.74)

*Statistically significant at $p < 0.05$

The findings indicate that the most significant contributors to pediatric SNHL in this population were ototoxic drug exposure, meningitis, toxoplasmosis, and neonatal jaundice, with rural residence and lower socioeconomic status acting as key sociodemographic risk factors. Other causes such as prematurity, viral infections, and congenital conditions were present but did not show statistically significant associations in this sample.

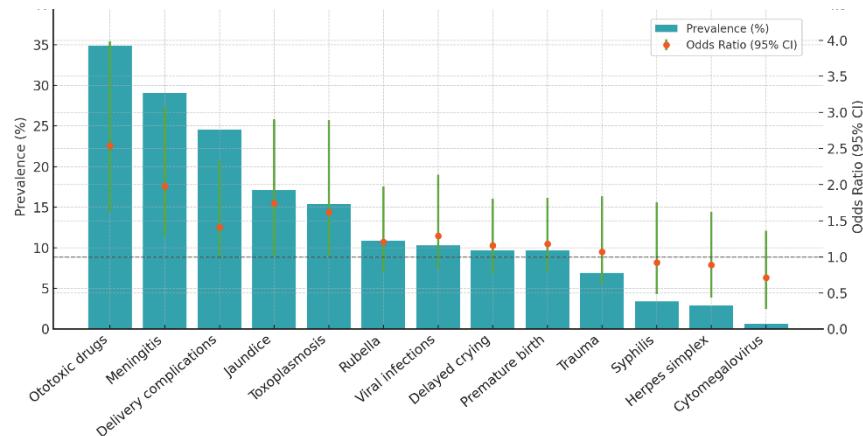


Figure 1 Prevalence and Risk Strength of Causes of Pediatric Sensorineural Hearing Loss

The figure provides a dual-axis visualization of the causes of pediatric sensorineural hearing loss, integrating both prevalence rates and relative risk strength. Ototoxic drug exposure emerged as the most dominant factor, affecting 34.9% of the children, with a markedly elevated odds ratio of 2.54 (95% CI 1.62–3.98), underscoring its role as the strongest predictor in this cohort. Meningitis followed closely, with a prevalence of 29.1% and an odds ratio of 1.98 (95% CI 1.28–3.08), confirming its significance as a leading acquired cause. Delivery complications, including perinatal asphyxia and surgical deliveries, were reported in 24.6% of cases, though their risk estimate of 1.41 (95% CI 0.98–2.33) approached but did not reach statistical significance. Among congenital and perinatal factors, jaundice (17.1%, OR 1.74, 95% CI 1.02–2.91) and toxoplasmosis (15.4%, OR 1.62, 95% CI 1.01–2.89) were both notable contributors with significant associations. Viral infections such as measles and mumps accounted for 10.3% of cases with a modest odds ratio of 1.29 (95% CI 0.84–2.14), while rubella was implicated in 10.9% (OR 1.21, 95% CI 0.79–1.98). Other perinatal factors including premature birth and delayed crying each accounted for 9.7% of cases but showed weaker associations, with odds

ratios close to 1.1. Less common but clinically relevant causes included trauma (6.9%), syphilis (3.4%), herpes simplex virus (2.9%), and cytomegalovirus (0.6%), all of which demonstrated non-significant risk estimates with confidence intervals crossing unity. Collectively, the visualization highlights that while multiple etiologies contribute to pediatric SNHL, preventable exposures particularly ototoxic medications and meningitis carry the highest burden and strongest associations in this population.

DISCUSSION

The present study identified ototoxic drug exposure and meningitis as the most significant causes of sensorineural hearing loss (SNHL) among children, with prevalence rates of 34.9% and 29.1%, respectively. These findings are consistent with previous literature that highlights the disproportionate role of preventable and acquired factors in low- and middle-income countries. For instance, Safdar *et al.* reported that nearly one-third of children with bacterial meningitis developed permanent hearing loss, emphasizing the long-term neurological sequelae of inadequately treated infections (9). Similarly, Jiang and colleagues in China estimated that nearly 30% of pediatric SNHL cases could have been prevented through early vaccination, prompt infection management, and cautious use of ototoxic medications (10). The current results align with these findings, underscoring the dual challenge of infection control and rational pharmacological practices in preventing pediatric deafness.

Perinatal complications emerged as another important contributor, with 24.6% of cases associated with delivery-related factors such as birth asphyxia and operative deliveries. Although not statistically significant in the current analysis, this trend is consistent with reports by van Beeck Calkoen *et al.*, who found perinatal hypoxic events to be a recurrent risk factor for congenital and early-onset hearing loss in European cohorts (6). The association between jaundice and SNHL, observed in 17.1% of the study population, also mirrors the findings of Boskabadi *et al.*, where severe hyperbilirubinemia was identified as a significant independent predictor of auditory neuropathy spectrum disorder (11). These findings suggest that inadequate perinatal monitoring, especially in rural and resource-constrained settings, continues to expose neonates to preventable risks of hearing impairment.

Congenital infections contributed substantially, with toxoplasmosis accounting for 15.4% and rubella for 10.9% of cases. The significant association with toxoplasmosis reinforces previous observations by Dimopoulos *et al.*, who demonstrated the relationship between low birth weight, congenital toxoplasmosis, and subsequent auditory impairment (12). The contribution of rubella is also noteworthy, particularly in the context of limited vaccination coverage in certain regions of South Asia. Caroça *et al.* reported that nearly 18% of pregnant women in Sub-Saharan Africa were at risk of rubella infection, placing neonates at considerable risk for congenital SNHL (13). The persistence of congenital infections as causes of hearing impairment in this study reflects gaps in prenatal screening, maternal immunization, and early neonatal care that demand urgent policy-level interventions. Although less common, other viral etiologies such as measles, mumps, cytomegalovirus, and herpes simplex virus were also observed, albeit with lower prevalence. International studies, particularly those conducted in high-income countries, have consistently shown cytomegalovirus to be the leading congenital infection associated with SNHL, with prevalence rates up to 20% in certain cohorts (14). The very low prevalence (0.6%) observed in this study may reflect underdiagnosis due to limited access to molecular testing rather than true rarity, suggesting that diagnostic gaps remain an important limitation in assessing the burden of congenital viral infections in Pakistan. Sociodemographic analysis revealed that rural residence and lower socioeconomic status were significantly associated with SNHL. These findings parallel the work of Chakrabarti and Ghosh, who reported disproportionately higher rates of severe and profound SNHL among children in socioeconomically disadvantaged regions of India (15). Rural households often have limited access to neonatal intensive care, audiological services, and vaccination programs, which exacerbates the risk of preventable hearing loss. In this study, 65.7% of children were from rural backgrounds, reflecting how geographic disparities intersect with medical risk factors to worsen outcomes.

The strengths of this study include the use of audiological confirmation of SNHL, systematic exploration of prenatal, perinatal, and postnatal risk factors, and analysis of sociodemographic determinants. However, several limitations must be acknowledged. The cross-sectional design precludes causal inference, and the reliance on convenience sampling limits generalizability. Recall bias from parental reporting, especially for early neonatal exposures, may have influenced accuracy despite cross-checking with clinical records. Additionally, the absence of genetic screening and advanced virological testing restricted the study's ability to identify hereditary and subclinical infectious contributors, which have been increasingly reported in global literature. Despite these limitations, the study highlights actionable implications for both clinical practice and health policy. The strong associations observed for ototoxic drug use and meningitis point to the need for antibiotic stewardship programs, parental counseling on drug safety, and improved management protocols for pediatric infections. The associations with perinatal factors and jaundice underscore the necessity of strengthening neonatal intensive care facilities and ensuring routine bilirubin monitoring in at-risk newborns. Expanding maternal vaccination programs, particularly against rubella, and implementing universal newborn hearing screening could substantially reduce the burden of undiagnosed and untreated SNHL.

CONCLUSION

The findings of this study demonstrate that sensorineural hearing loss in children is predominantly associated with preventable causes, most notably exposure to ototoxic drugs and post-meningitic complications, followed by perinatal conditions such as jaundice and delivery-related factors, and congenital infections including toxoplasmosis and rubella. Less frequent contributors such as trauma, syphilis, herpes simplex, and cytomegalovirus were identified but carried limited statistical significance. The observed associations with rural residence and lower socioeconomic status highlight the role of social determinants in exacerbating disease burden. These results emphasize the urgent need for comprehensive preventive measures, including strict regulation and monitoring of ototoxic drug use, improved neonatal care and bilirubin screening, early management of infectious diseases, and expansion of maternal immunization programs. Establishing universal newborn hearing screening and awareness campaigns in underserved communities could further aid in early detection and timely intervention. Collectively, these strategies can significantly reduce the burden of pediatric hearing loss, improve developmental outcomes, and enhance the quality of life for affected children.

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