

History of Premature Birth and Low Birth Weight as Risk Factors for Hearing Impairment in Children

Dina Alia¹, Nuzul Afrizal²

¹ Department of Otorhinolaryngology, Head and Neck Surgery, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Aceh, Indonesia

² Residency Program in Department of Otorhinolaryngology, Head and Neck Surgery, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Aceh, Indonesia

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Abstract

Background: Infants with a history of low birth weight (LBW) and prematurity are at increased risk of developing hearing loss, along with other health complications. Since normal hearing is closely linked to speech and language development, untreated hearing loss in these infants can significantly impair communication skills. Therefore, early hearing screening is essential.

Objective: The aim of the study was to understand the significance of LBW and prematurity as risk factors for hearing loss in infants.

Case Report: We presented a case of a 3-year-old boy with bilateral sensorineural hearing loss. The patient had multiple risk factors, including prematurity, LBW, congenital heart disease, asphyxia, and hyperbilirubinemia. Hearing evaluations using otoacoustic emissions (OAE), brainstem evoked response audiometry (BERA), and auditory steady-state response (ASSR) confirmed the diagnosis. The child was subsequently fitted with hearing aids and underwent behavioral and speech therapy.

Method: Literature studies were conducted manually to deepen understanding of the relationship between prematurity, LBW, and hearing loss.

Conclusion: Infants with LBW and prematurity are at increased risk of hearing loss. Early detection and timely intervention with hearing aids, combined with rehabilitation therapy, play a critical role in optimizing language and communication development.

Corresponding Author: dinaalia@usk.ac.id (Dina Alia)

INTRODUCTION

Hearing loss arises when the transmission of sound from the external ear to the brain is impaired. Such impairment may occur at different stages of auditory processing, either before or after the cochlea, and is typically classified as conductive, sensorineural, or mixed. In infants, the risk factors for hearing loss are generally categorized into three groups: prenatal, perinatal, and postnatal.¹ Prenatal risk factors include intrauterine infections, craniofacial anomalies, genetic syndromes associated with congenital deafness, and a family history of hereditary hearing loss. Perinatal risk factors are those present at birth, such as preterm delivery, very low birth weight (<1,500 g), hyperbilirubinemia, and low Apgar scores. Postnatal risk factors include prolonged administration of ototoxic medications (≥ 5 days), concurrent use of loop diuretics, bacterial meningitis, and admission to the neonatal intensive care unit (NICU) for five days or longer, particularly when mechanical ventilation is required.² Prematurity and low birth weight (LBW) are strongly associated with hearing impairment, as these conditions may contribute to delayed growth, immature organ

development, and compromised neurological function.³

According to the World Health Organization (WHO), approximately 0.5 to 5 per 1,000 newborns present with moderate to severe sensorineural hearing loss. Genetic factors account for up to 50% of these cases, even in the absence of other identifiable risk factors. Infants admitted to the neonatal intensive care unit (NICU) have a disproportionately higher incidence of risk factors associated with hearing impairment.¹⁴ More than 80% of pediatric hearing loss is attributable to congenital causes, either present at birth or manifesting shortly thereafter. The prevalence of hearing loss among high-risk infants is estimated to be 10 to 20 times greater than in infants without risk factors and, in some cases, may be as high as 10 to 50 times greater.⁵

Data from the Otorhinolaryngology (ENT) Community Division at Dr. Zainoel Abidin General Hospital indicate a substantial burden of pediatric hearing loss. Between 2018 and 2021, 203 cases were documented. In 2022, 78 children were newly diagnosed with hearing loss. In 2024, among 117 patients assessed, 28 were premature infants with low birth weight (LBW); of these, 17 experienced

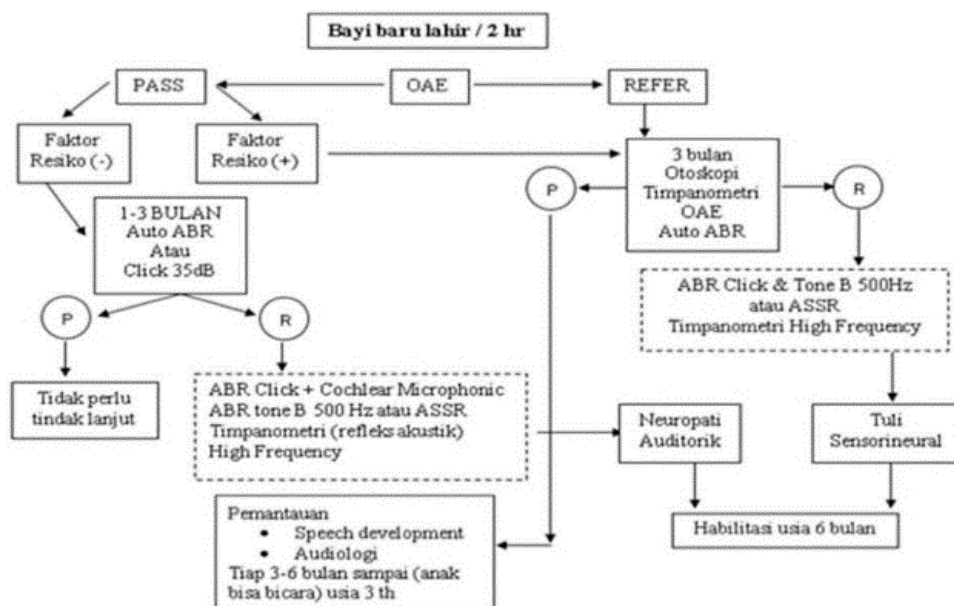


Figure 1. Newborn Screening Algorithm

hearing loss. Research conducted in 2023 further demonstrated that prenatal factors were the most frequent contributors (77.5%), followed by postnatal factors (44.9%) and perinatal factors (36.7%).²

If undetected and untreated, hearing loss can significantly impair language development and may lead to a spectrum of secondary complications, including behavioral problems, cognitive delays, motor deficits, intellectual disabilities, and other developmental disorders.² Early detection is particularly challenging in infants, who are unable to report symptoms, thereby underscoring the importance of routine hearing screening.⁶ The Joint Committee on Infant Hearing (JCIH) recommends universal newborn hearing screening within the first 48 hours of life, confirmation of diagnosis by 3 months of age, and initiation of appropriate intervention by 6 months of age (Figure 1).⁴

Hearing screening for newborns can be conducted through hospital-based newborn hearing screening, which is divided into targeted newborn hearing screening, which involves screening all babies at risk of hearing impairment (high-risk babies), babies admitted to the NICU (Neonatal Intensive Care Unit) or PICU (Perinatal Intensive Care Unit), and Universal

newborn hearing screening, which is performed on all live-born babies with or without risk factors for hearing loss.

Hearing function testing in children is divided into two types: subjective tests based on observing the child's behavior or response to sound stimuli, such as Behavioral Observation Audiometry (BOA) and Visual Reinforcement Audiometry (VRA), and non-behavioral or objective tests using electrophysiological instruments, such as Auditory Brainstem Response (ABR/BERA), Auditory Steady State Response (ASSR), and Otoacoustic Emission (OAE).⁶

CASE DESCRIPTION

A 3-year-old male patient was brought to the clinic with concerns of delayed speech and absence of verbal communication. The child was unable to produce meaningful words and did not respond to auditory stimuli from any direction. Currently, he vocalizes only nonspecific sounds, such as "baa..baa.." and "mamm..," without forming intelligible speech. Additionally, there was no eye contact when spoken to. According to the parents, these concerns were first observed when the child was approximately 18

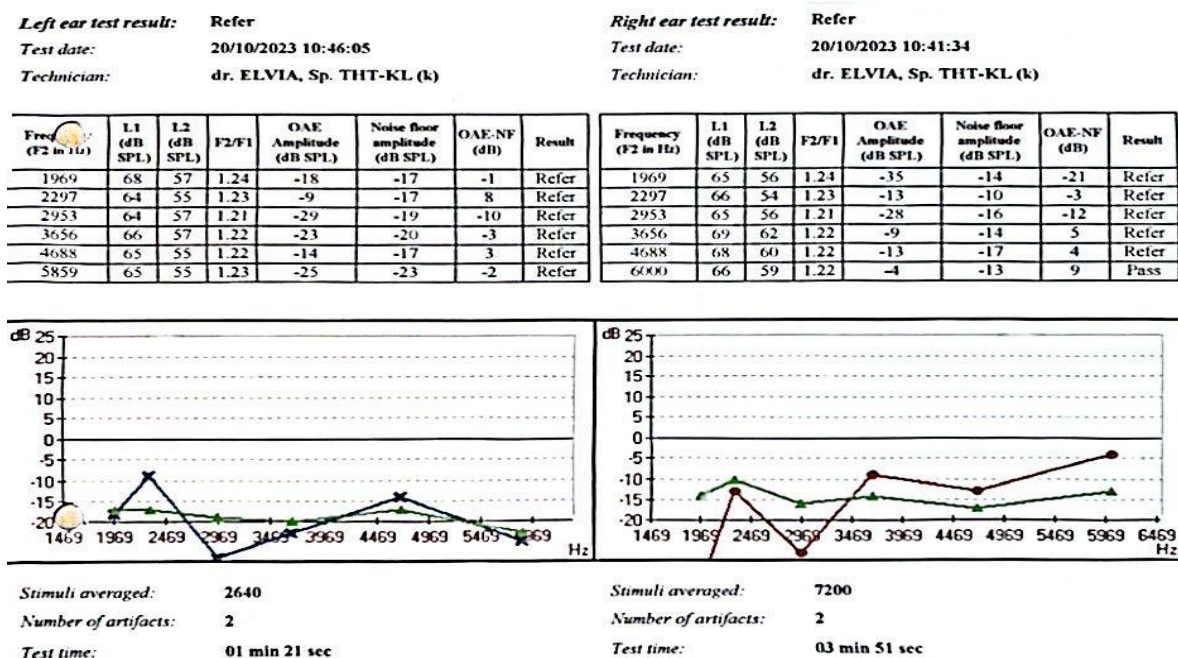


Figure 2. OAE Examination

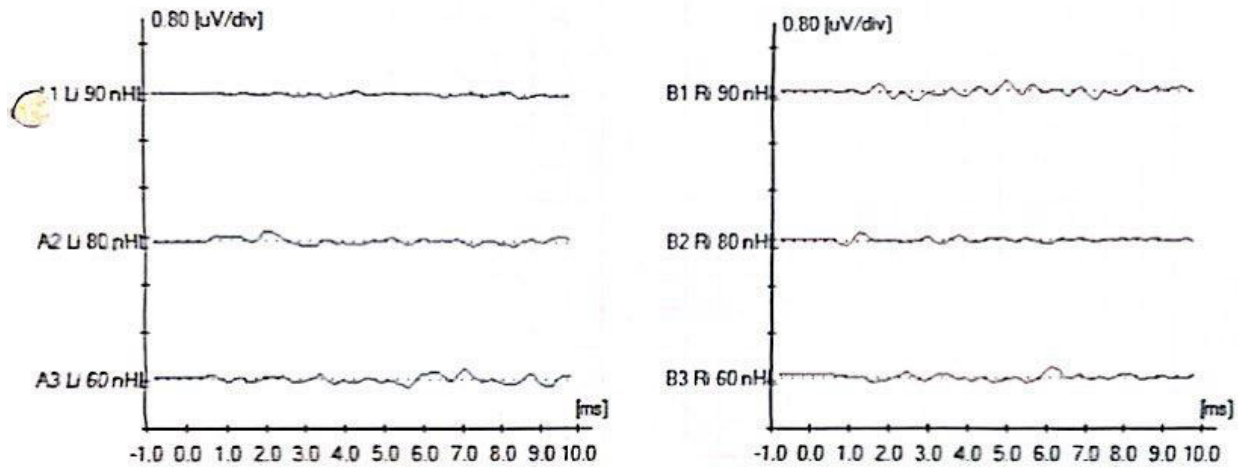


Figure 3. BERA Examination

months old. Despite these deficits, his physical growth and motor development were appropriate for age. Immunization history was incomplete.

The mother reported no history of maternal infections during pregnancy and denied the use of medications or herbal remedies. The child was delivered via cesarean section at 26–27 weeks of gestation, with a birth weight of 1,900 g. At birth, the neonate did not cry immediately and required admission to the neonatal intensive care unit (NICU)

for 14 days. The NICU admission was necessitated by prematurity, low birth weight, perinatal asphyxia, congenital heart disease, and hyperbilirubinemia.

On physical examination, the ears, nose, and throat were within normal limits. However, further evaluation with OAE examination demonstrated bilateral refer/refer results, consistent with outer hair cell dysfunction of the cochlea in both ears (Figure 2).

BERA examination revealed that electrophysiological response thresholds for waves

Physiologic Audiogram

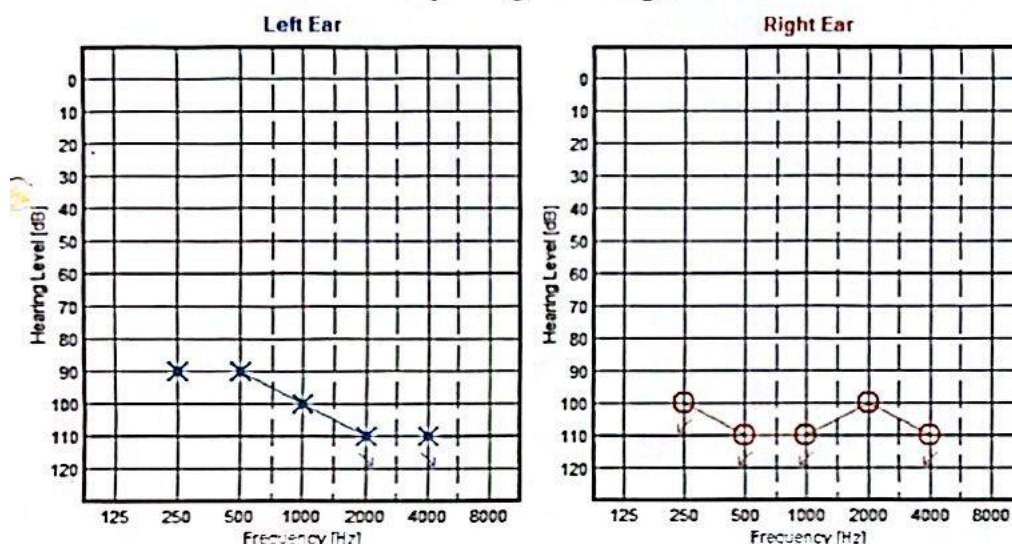


Figure 4. ASSR Examination

I, III, and V were absent up to 90 dB in both ears (Figure 3)

ASSR examination (Figure 4) demonstrated peripheral hearing thresholds of 102.5 dB in the right ear and 107.5 dB in the left ear, consistent with bilateral profound sensorineural hearing loss.

The patient was diagnosed with severe bilateral sensorineural hearing loss. ABD fitting was recommended, followed by behavioral therapy and speech therapy. Cochlear implants may be the next intervention option.

METHOD

Literature search through PubMed, Science Direct, Springer Link, Dental Sciences, and relevant journals using the search facility with keywords “risk factors for hearing loss in children,” “hearing loss in premature and low birth weight infants,” and “early detection of hearing loss in children.”

DISCUSSION

In this case, the parents recognized that their child was not developing communication skills appropriate for his age. However, due to both economic constraints and parental inexperience, particularly as the child was their firstborn, they delayed seeking medical evaluation until the child was 3 years old. At presentation, the primary complaints included lack of response to auditory stimuli, absence of head-turning when called, and failure to produce meaningful speech.

According to established developmental milestones for speech and expressive language, by the age of 3 years, a child should be able to answer simple questions, describe the actions of others, talk about objects or people not present, produce intelligible speech in approximately 75% of utterances, and articulate consonant sounds such as /k/, /g/, /f/, and /y/ with clarity. Failure to meet these milestones is a clinical indicator of possible hearing impairment. One of the recognized red flags in expressive language development at this age is a vocabulary of fewer than 200 words, which was

evident in the present case.⁷

The patient was male, which is notable given that several studies have reported a higher prevalence of hearing impairment among boys. One study documented that 237 children (50.9%) of the total subjects with hearing loss were male. Similarly, a retrospective study conducted at Dr. Soetomo General Hospital between 2011 and 2013, involving 556 infants and children, found that 199 subjects (52.79%) were male.^{8,9} To date, however, no definitive explanation has been established regarding this apparent gender-related trend, and it remains uncertain whether the predominance of cases among males is attributable to anatomical or genetic factors or whether it is incidental.

This patient was delivered by cesarean section at less than 37 weeks of gestation (premature) with a birth weight below 2,500 g, thus fulfilling the criteria for prematurity and low birth weight (LBW).³ Both prematurity and LBW are well-established perinatal risk factors for hearing impairment, as they are associated with a spectrum of health complications ranging from impaired growth and incomplete organ maturation to neurological dysfunction.³ In the present case, prematurity and LBW likely contributed to the patient's hearing impairment.

Several studies have demonstrated a significant association between these factors and hearing outcomes. One study reported a strong correlation between combined prematurity and LBW with the degree of hearing impairment in children.⁹ Bambang et al. identified the most frequent risk factors for deafness as hyperbilirubinemia (53.0%), prematurity (36.1%), mechanical ventilation (32.5%), and LBW (19.3%). A chi-square analysis revealed LBW as a statistically significant risk factor for hearing impairment ($p = 0.001$).¹⁰ Moreover, neonates born before 37 weeks of gestation are particularly vulnerable to disturbances in neurogenesis, as the cochlear organ may not yet be structurally and functionally mature. This immaturity often results in impaired function of the outer hair cells within the cochlea.^{9,11}

Risk factors such as LBW rarely act in isolation

and are frequently interrelated with other perinatal conditions. Evidence indicates that prematurity with LBW is significantly associated with the severity of hearing impairment ($r=5.533$; 95% CI=0.728–42.051; $p=0.046$).^{12,13} The frequent co-occurrence of prematurity and LBW in children with congenital deafness underscores the multifactorial nature of risk and highlights the need for targeted screening in this population.

Children with a history of admission to the neonatal intensive care unit (NICU), as in this case for 14 days, are at increased risk of developing hearing loss, particularly when compounded by factors such as hyperbilirubinemia or exposure to aminoglycoside antibiotics.¹⁴ Prolonged use of mechanical ventilation, especially for more than five days, has also been identified as a risk factor for neonatal hearing impairment. The application of positive inspiratory pressure during mechanical ventilation elevates intrathoracic pressure, which in turn increases pulmonary vascular resistance. This hemodynamic alteration disrupts blood flow from the heart to the lungs and back, ultimately reducing cardiac output.¹⁵

In the present case, the patient had a history of perinatal asphyxia, evidenced by failure to cry immediately after birth and the subsequent need for NICU care. Perinatal asphyxia is a recognized cause of long-term complications, including sensorineural hearing loss, due to injury to the cochlear nerve structures. In the pathophysiological mechanism, asphyxia induces hyperpolarization of cochlear hair cells, resulting in diminished neurotransmitter release and reduced auditory nerve activity. Furthermore, hypoxia associated with asphyxia can lead to ischemia, reducing oxygen and nutrient delivery to neural tissues and causing secondary neuronal injury. This hypoxic-ischemic state is also characterized by excessive glutamate release into the synaptic cleft, which disrupts synaptic transmission and induces excitotoxicity, ultimately leading to permanent neuronal damage.¹⁶

A history of hyperbilirubinemia, as observed in this patient, can result in selective damage to the auditory nuclei of the brainstem, as well as injury to

the auditory nerves and spiral ganglion cells. This pathology is associated with disturbances in neuronal intracellular calcium homeostasis. Interestingly, the organ of Corti and the thalamocortical auditory pathway are typically spared from bilirubin-induced damage. De Vries et al. reported that among premature infants with elevated bilirubin levels (≥ 14 mg/dL), those with birth weights $\leq 1,500$ g demonstrated a higher risk of developing deafness compared with infants whose birth weights exceeded 1,500 g.¹⁷

In addition, the patient had a history of congenital heart disease (CHD), a condition recognized to increase the risk of sensorineural hearing loss (SNHL) and neurodevelopmental disorders. SNHL in children with CHD frequently manifests as delayed speech and impaired language acquisition. While the overall prevalence of SNHL in the general pediatric population is estimated to be approximately 0.2% at birth and increases to 0.35% during adolescence, the risk is substantially higher in children with CHD. A study by Gopinetti et al. found that among 172 children with CHD, 20 (11.6%) were diagnosed with hearing loss.¹⁸ The mechanisms underlying this increased risk are multifactorial and may include physiological alterations, medication-related ototoxicity, and complications directly attributable to the underlying cardiac condition.

Otoacoustic emission (OAE) testing was performed to evaluate cochlear function, specifically the activity of outer hair cells (OHCs). OAE represents an acoustic response to auditory stimuli generated by normally functioning OHCs. The cochlea itself functions to differentiate sound frequencies and transmit them through the auditory nerve pathways and brainstem to the brain, where they are perceived as sound. Damage to OHCs may result from infection, exposure to ototoxic agents, or impaired cochlear blood flow. Prematurity is also a recognized risk factor, as it may cause structural and functional immaturity of the cochlea, including the OHCs. Javiolo (2013) reported that neonates with a gestational age of <32 weeks had an 8.69-fold higher risk of OHC dysfunction compared to term infants. In the present case, OAE

results revealed refer/refer findings in both ears, indicating bilateral OHC dysfunction consistent with the patient's history of prematurity.¹⁵

To further assess auditory pathway integrity, brainstem evoked response audiometry (BERA) was conducted. BERA evaluates the electrical activity generated by neural responses to auditory stimuli, thereby assessing conduction along the auditory nerve pathway from the cochlea to the brainstem. In this patient, the BERA results demonstrated absence of wave V across all intensities in both ears, suggesting SNHL with involvement of the auditory nerve system, thereby corroborating the OAE findings.¹⁹

Following confirmation of bilateral severe SNHL, ASSR test was performed to determine precise hearing thresholds and guide appropriate hearing aid fitting. In this case, ASSR results demonstrated thresholds of 102.5 dB in the right ear and 107.5 dB in the left ear, consistent with bilateral profound hearing loss. ASSR testing provided frequency-specific threshold information, which is essential for accurate HA programming and optimization tailored to the patient's auditory needs.

SNHL, if left untreated, can lead to profound language development disorders, as demonstrated in this case and corroborated by parental reports. Habilitation strategies for such patients include the use of auditory brain devices (ABD) or cochlear implants. Selection of an appropriate hearing device requires careful consideration of multiple factors, including amplification needs and output capacity, frequency range (bandwidth), ear canal size and morphology, anticipated growth-related changes in conchal cartilage and ear canal dimensions, skin sensitivity, and the necessity for additional features such as directional microphones, telecoils, direct auditory input, or integrated FM receivers. Comfort, the potential for occlusion effects, and cosmetic acceptability are also important determinants in device selection.¹⁴

Use of ABDs should be complemented by structured behavioral and speech therapy, typically under the supervision of a specialist in physical medicine and rehabilitation (Sp.KFR). The effectiveness of speech development is strongly influenced by

parental adherence to therapy programs, including the consistent implementation of prescribed home exercises. Such compliance is essential, as children may initially resist the ABD, perceiving it as a foreign object, or may experience difficulty adapting to newly perceived sounds. In addition, individual factors such as cognitive capacity and the presence of other congenital conditions significantly affect speech outcomes in children using ABDs.

Optimal management of pediatric SNHL requires a multidisciplinary approach. Collaboration among pediatricians (Sp.A), otorhinolaryngologists (Sp.T.H.T.B.K.L.), and rehabilitation specialists (Sp.KFR) ensures timely detection, appropriate intervention, and comprehensive habilitation. This interprofessional synergy provides a holistic, sustained, and effective strategy to maximize the child's communication abilities, quality of life, and long-term developmental potential.

CONCLUSION

Infants with a history of prematurity and low birth weight are at significantly increased risk of developing hearing impairment, which can adversely affect language acquisition and communication skills. Delayed detection, as demonstrated in this case, may result in the loss of the critical period for auditory and language-related brain development, thereby impeding both linguistic and cognitive outcomes. Consequently, newborn hearing screening—both universal screening for all live births and targeted screening for high-risk populations—must be regarded as an essential and non-negotiable public health priority. Timely diagnosis by 3 months of age, followed by intervention no later than 6 months, represents a pivotal strategy for safeguarding a child's potential for optimal growth, learning, and communication. Ultimately, early detection and intervention in hearing impairment should be recognized as an investment in human capital and the nation's future.

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