GSC Advanced Research and Reviews

eISSN: 2582-4597 CODEN (USA): GARRC2

Cross Ref DOI: 10.30574/gscarr

Journal homepage: https://gsconlinepress.com/journals/gscarr/

(REVIEW ARTICLE)

Check for updates

Diagnosis and management of hearing loss in congenital Rubella Syndrome: A literature review

Ni Putu Oktaviani Rinika Pranitasari *, I Gusti Ayu Mahaprani Danastri and Elviana Lui Tey Seran

Department of Otorhinolaryngology Head and Neck Surgery, Faculty of Medicine, Udayana University, Denpasar, Bali, Indonesia.

GSC Advanced Research and Reviews, 2025, 23(03), 060-066

Publication history: Received on 29 April 2025; revised on 05 June 2025; accepted on 07 June 2025

Article DOI: https://doi.org/10.30574/gscarr.2025.23.3.0148

Abstract

Congenital Rubella Syndrome (CRS) is a condition that results from maternal rubella infection during pregnancy, particularly in the first trimester. This infection can lead to a variety of clinical manifestations in the infant. The virus can cross the placenta and affect fetal development, leading to a range of birth defects. Because of this risk, rubella infection during pregnancy is a major public health concern.

Globally, around 236,000 cases of Congenital Rubella Syndrome (CRS) occur each year in developing countries, with Indonesia reporting a rise in incidence from 3.2 in 2015 to 5.6 per 100,000 in 2017. The risk of CRS is 85% when infection occurs within 12 weeks of gestation and may cause severe congenital defects, including congenital heart defects, cataracts, intrauterine growth retardation with sensorineural hearing loss (SNHL) being the most prevalent. It may be unilateral or bilateral and is often not detected until delays in language development become apparent. SNHL in children impairs speech and language development if untreated, necessitating early intervention. Therefore, newborn hearing screening is essential in infants with suspected or confirmed CRS.

Rubella-induced damage to the cochlea and auditory nerve underlies SNHL, diagnosed through audiological assessments like otoacoustic emissions (OAE), brainstem evoked response audiometry (BERA), and auditory steady-state response (ASSR). Management involves hearing aids, cochlear implants, and speech therapy, with early intervention by 6 months critical to optimize outcomes.

Prevention relies on MMR vaccination and universal newborn hearing screening, particularly in resource-limited settings like Indonesia, where enhanced immunization and screening access are essential to reduce CRS burden.

Keywords: Congenital Rubella Syndrome; Sensorineural hearing loss; Neonatal hearing screening; Rubella vaccination; Hearing diagnostic

1. Introduction

Congenital Rubella Syndrome (CRS) arises from transplacental transmission of the rubella virus during pregnancy, primarily in the first trimester, resulting in congenital anomalies such as sensorineural hearing loss (SNHL), cataracts, congenital heart disease, and mental retardation.[1] SNHL is the most common clinical manifestation, leading to disturbance in speech and language development if not properly managed. [2] However, most parents realize the disturbance only when the kids are 2-5 years old, as their speech and language development are easily observed.[3]

* Corresponding author: Ni Putu Oktaviani Rinika Pranitasari.

The risk of CRS is as high as 85% when maternal infection occurs within the first 12 weeks of gestation, decreasing to 10-20% by weeks 13-16%(4). In Indonesia, the incidence of rubella increased from 3.2 per 100,000 births in 2015 to 5.6 in 2017, though CRS cases at Prof Ngoerah Hospital, Bali, declined from 6.73 in 2021 to 2.85 in 2023. Therefore, more than 100,000 kids have CRS each year.[4]

This review aims to consolidate evidence on the epidemiology, pathogenesis, management, and prevention of SNHL in CRS, emphasizing early detection and intervention strategies applicable to resource-limited settings like Indonesia.

2. Embryology, Anatomy, and Physiology of the Ear

2.1. Embryology of the Ear

The ear's development, structure, and function are critical to understanding the mechanisms of SNHL in CRS, as the rubella virus targets the auditory structure during fetal development. Ear development begins in the third week post-fertilization, driven by the differentiation of the pharyngeal apparatus, which forms brachial arches, pouches, and clefts.[5] These arches, composed of mesoderm, give rise to muscles, blood vessels, and neural crest cells that form bone and nerve tissues. [6,7] Disruptions in this process, such as those caused by rubella infection, can lead to congenital anomalies, including hearing loss.[7]

2.2. Anatomy of the Ear

The inner ear, or labyrinth, comprises a bony labyrinth (semicircular canals, vestibule, cochlea) and a membranous labyrinth (semicircular canals, utricle, saccule, cochlea).[8] The cochlea, a 35-mm bony canal coiling 2½ turns around the modiolus, is central to auditory function. The basilar and vestibular (Reissner's) membrane divides the cochlea into three compartments: scala vestibuli, scala media (cochlear duct), and scala tympani. The scala vestibuli and scala tympani, containing perilymph (high Na^+, low K^+, similar to cerebrospinal fluid), connect via the helicotrema at the cochlear apex, with the scala vestibuli linked to the oval window and the scala tympani to the round window.[9]

On the other hand, the scala media contains endolymph (high K^+, low Na^+) produced by the stria vascularis that maintains an 80 mV endocochlear potential critical for auditory signaling.[5,6] The organ of Corti, located on the basilar membrane, houses sensory hair cells (3,500 inner, 12,000 outer) that convert mechanical vibrations into electrical signals.[6] Inner hair cells, bottle-shaped, act as primary mechanoreceptors, while outer hair cells, cylindrical, enhance frequency selectivity via the cochlear amplifier.[1] The basilar membrane's elasticity varies—rigid and narrow at the base (high-frequency sensitive) and flexible and wider at the apex (low-frequency sensitive).[8]

2.3. Hearing Physiology

Sound vibrations captured by the auricle travel through the ear canal, vibrating the tympanic membrane. The vibration is then relayed via the ossicles to the cochlear oval window, moving perilymph in the scala vestibuli. The pressure transfers through the helicotrema to the scala tympani, displacing the round window, and propagates via Reissner's membrane to the basilar membrane, with high frequencies peaking at the rigid base and low frequencies at the flexible apex.

The basilar membrane movement bends hair cell stereocilia, which open the mechanotransduction channels to allow K^+ influx, driven by the +80 mV endocochlear potential, triggering auditory nerve signals during depolarization. [6,8] The propagated signals travel through the cochlear nuclei, the inferior colliculus, the medial geniculate body, and finally, the auditory cortex.[6]

3. Epidemiology

CRS remains a significant public health challenge, particularly in developing countries, where WHO estimates approximately 236,000 cases occur annually, increasing tenfold during epidemics.[10] In Southeast Asia, rubella cases declined from 10,361 in 2016 to 4,386 in 2017, but CRS cases rose from 319 to 754 in the same period.[11] In contrast, effective vaccination program have reduced CRS incidence in developed regions, with rates during 2005–2015 dropping to <2 per 100,000 live births in the United States, 1.7 in Israel, 1.5 in Singapore, and 0.5 in Malaysia.[10] Limited studies in Indonesia suggest there is still a high rubella incidence, with Riskesdas 2011 reporting approximately 400 CRS cases, and WHO estimating 5,000–10,000 infants born with deafness annually in 2012.[10] Moreover, according to the Indonesian Ministry of Health, immunization coverage declined in 2015 compared to previous years, emphasizing a significant public health issue requiring effective prevention efforts.[12]

4. Etiopathogenesis

The rubella virus, a single-stranded RNA virus of the Togaviridae family, is transmitted via respiratory droplets during coughing or sneezing, replicating in the nasopharynx and regional lymph nodes before spreading systemically via viremia 5–7 days post-exposure. The virus is highly contagious, particularly one week before and four days after rash onset, with an incubation period of 14–21 days.[1] Transplacental infection during maternal viremia, typically 5–7 days post-inoculation, targets the developing fetus, with congenital infection rates exceeding 80% in the first 12 weeks (43% in month 1, 51% in first trimester), 54% at 13–14 weeks, and 25% by the end of the second trimester, with no CRS risk after 16 weeks. [13,14]

Placental infection causes focal necrosis in chorionic villi epithelium and capillary endothelial cells, desquamating into the fetal circulation as infected emboli, leading to organ infection and damage. The virus inhibits cell division, induces apoptosis, and causes cellular necrosis without inflammation in early pregnancy due to the fetus's immature immune system.[15] In CRS, rubella-induced apoptosis and vascular endothelial damage in the cochlea, organ of Corti, and stria vascularis disrupt auditory development, leading to SNHL, the most common defect.[4]

Infants with CRS excrete the virus in nasopharyngeal secretions (84% in month 1, declining to 3% by year 2) and urine, remaining infectious for up to 27 months, typically less than one-year[25].[16]

5. Clinical manifestations

Rubella infection is asymptomatic in 20–50% of cases, but symptomatic cases resemble other rash-associated diseases, presenting acutely with maculopapular rash, fever (>37.2°C), arthralgia/arthritis, lymphadenopathy, and conjunctivitis in adults or pregnant women[26]. Maternal rubella infection in the first trimester, particularly before 8 weeks, poses a 50–80% risk of fetal infection, with congenital defect risk approaching 90%, decreasing to 10–20% by week 16, and becoming rare after 20 weeks.[13,15] Affected organs include the eyes, ears, heart, and central nervous system, characterized by four major defects[1]

5.1. Sensorineural hearing loss (SNHL)

The most common defect, often the sole manifestation, occurring if infection precedes 8 weeks of gestation, defined by WHO as permanent hearing loss ≥26 dB. [10]

5.2. Cardiac defects

Including PDA, VSD, and pulmonary stenosis.

5.3. Ocular defects

Cataracts and glaucoma, rarely occur in isolation.

5.4. Mental retardation

Affecting neurological development.

SNHL in CRS infants commonly manifests as speech delay, necessitating evaluation if developmental milestones are missed: no babbling or sound imitation by 12 months, no single meaningful words by 18 months, fewer than 10 words by 24 months, or no two-word phrases by 30 months.[17] Auditory developmental milestones should also guide SNHL suspicion

- 0–4 months: Reflexive responses, such as startling to loud noises or waking from sleep (e.g., auropalpebral or Moro reflexes).
- 4–7 months: Inconsistent head turns toward horizontal sound sources, becoming more consistent by 7 months, with stronger neck muscles.
- 7–9 months: Precise localization of sound sources with rapid, firm head turns.
- 9–13 months: Active seeking of sounds, including from above, with rapid multi-directional localization by 13 months.
- 2 years: Reduced responses to repeated stimuli due to anticipatory prediction of sound sources, requiring careful clinical observation.

6. Diagnostic Tests

Laboratory tests support the diagnosis of rubella virus infection and assess immunological status using specimens such as throat swabs, blood, urine, cerebrospinal fluid, amniotic fluid, or saliva.[1] Due to the complexity and cost of virus isolation, serological and RNA-based tests are preferred for routine diagnosis. These tests are categorized into three approaches:

6.1. Virus Isolation

Rubella virus can be isolated from nasal secretions, blood, throat swabs, urine, or cerebrospinal fluid, with pharyngeal isolation possible from one week before to two weeks after rash onset. Cell cultures (e.g., Vero, African green monkey kidney [AGMK], RK-13) detect cytopathic effects (CPE), providing a definitive diagnosis[2]. However, this method is rarely used due to its complex, time-consuming procedures, making it unsuitable for routine diagnostics.

6.2. Serological Testing

Serological tests, primarily enzyme-linked immunosorbent assay (ELISA), diagnose congenital and postnatal rubella infections and evaluate immunity status, particularly in pregnant women exposed to rubella. The serum is tested for rubella-specific IgM to confirm acute or congenital infection, with repeated testing after 5 days for worsening symptoms. IgG levels (≥15 IU/mL in early pregnancy) indicate protective immunity.

6.3. RNA Testing

Molecular methods detect rubella virus RNA, offering high sensitivity.

- Reverse Transcription Polymerase Chain Reaction (RT-PCR): Routinely used in the UK, RT-PCR detects RNA in amniotic fluid (87–100% sensitivity) or saliva (for surveillance). Amniocentesis is optimal at <8 weeks post-infection and >15 weeks of gestation.[1]
- Reverse Transcription-Loop-Mediated Isothermal Amplification (RT-LAMP): An alternative to RT-PCR, with 77.8% sensitivity (vs. 66.7% for RT-PCR, 33.3% for isolation).[1] Results are assessed via turbidity after turbidimeter incubation

7. SNHL in CRS

Typically presenting within the first 6–12 months of life, SNHL in CRS may also be evident at birth, significantly impacting speech and language development. It can manifest unilaterally or bilaterally with varying severity. The mechanisms underlying rubella-induced SNHL are not fully elucidated but involve direct viral damage to auditory structures.[18] The rubella virus enters the inner ear via the stria vascularis blood supply, causing cell death in the organ of Corti, stria vascularis, and cochlear duct, altering endolymph composition critical for auditory signaling.[18] Additionally, vasculitis triggered by the virus directly damages cochlear cells and impairs myelination of the auditory nerve, further exacerbating hearing loss. Vascular endothelial damage may also cause hypoxia, with emboli leading to thrombosis in small blood vessels, compounding cochlear injury.[19]

7.1. Diagnosis

Early diagnosis of SNHL in CRS is critical to enable timely intervention and mitigate developmental delays, particularly in speech and language. While CRS diagnosis relies on clinical criteria and laboratory confirmation, audiological assessments are essential for detecting SNHL, the most common CRS manifestation. Newborns exhibit early auditory development, responding to sounds with reflexive behaviors such as the auropalpebral reflex (blinking), increased heart rate, widened eye, cessation of suckling, or facial grimacing, which can indicate intact auditory function[32]. Failure to demonstrate these responses or delays in auditory milestones warrants detailed audiological evaluation.[19] Gold-standard techniques, including otoacoustic emissions (OAE), brainstem evoked response audiometry (BERA), and auditory steady-state response (ASSR), offer rapid, non-invasive, and highly sensitive (nearly 100%) methods for SNHL detection.[5] These methods, described below, are particularly valuable for newborns and uncooperative patients, ensuring early identification of hearing loss in CRS infants.

7.1.1. Otoacoustic Emission

Otoacoustic emissions are low-intensity sounds produced by the outer hair cells of the cochlea and recorded in the external auditory meatus, either without acoustic stimulation (spontaneous emissions) or in response to acoustic stimulation (acoustically evoked emissions) or electrical stimulation (electrically evoked emissions). The sounds

captured by the cochlea are very faint, around 30 dB, but potentially audible. Otoacoustic emissions occur spontaneously due to continuous sound circulation within the cochlea but typically require prior stimulation. Otoacoustic emissions are generated only when the organ of Corti is near normal and the middle ear functions well.[5]

7.1.2. Brainstem Evoked Response Audiometry (BERA)

BERA is a technique for measuring nerve activity or responses to sound stimuli.[5] Conditions recommended for BERA testing include newborns to prevent speech/language development delays. If a child experiences speech delays or impairments, one potential cause may be an inability to receive sound stimuli due to ear dysfunction.[20] BERA can also be used to determine the source of hearing loss (cochlear or retrocochlear), evaluate brainstem function, and distinguish between psychological and physical causes of hearing loss. The procedure is relatively safe, painless, and without side effects, making it suitable for medical check-up screening.[20] Although BERA provides information on hearing function and sensitivity, it is not a substitute for formal hearing evaluation, and results should be correlated with standard audiometry if available.[20,21]

7.1.3. Auditory Steady State Response (ASSR)

ASSR is an objective test used to measure hearing ability in children who cannot yet undergo subjective tests such as play audiometry or pure-tone audiometry. ASSR is also an objective test employed to evaluate hearing ability in children unfit for traditional audiometric testing. [20,21] Like Auditory Brainstem Response (ABR), ASSR can estimate hearing thresholds for individuals unable to participate in conventional testing methods. Therefore, the primary benefits of ASSR include diagnostic follow-up assessments in infants, infants in neonatal intensive care units (NICUs), unresponsive or comatose patients, and others.[20]

7.2. Treatment

SNHL in CRS has no specific treatment and is managed supportively. The Joint Committee on Infant Hearing (JCIH) 2000 recommends that hearing loss in children should be detected by 3 months of age, with appropriate interventions initiated by 6 months. These interventions include amplification through hearing aids, cochlear implantation, and special education (speech therapy, schools for the deaf) as rehabilitation efforts, aiming to optimize the child's speech and language abilities.[22]

7.2.1. Hearing Aid

Hearing aids are designed to amplify stimuli for the damaged sensory cells in the inner ear, enhancing responses to external sounds and noises. They include a microphone that converts sound waves into electrical energy, which is then amplified by an amplifier to increase sound volume and transmitted to a speaker in the ear.[23] The most commonly chosen hearing aid for children is the behind-the-ear model. The American Academy of Audiology recommends this type for children due to its lower risk of being swallowed than in-the-ear hearing aids. During the first two years of hearing aid use, the Paediatric Working Group recommends audiological consultations every 3 months to monitor audiological status and ensure the hearing aid's appropriateness, given the rapid growth of the ear canal during this period.

7.2.2. Cochlear Implant

Cochlear implants (CI) directly stimulate spiral ganglion cells, the primary sensory neurons in the auditory pathway. Though severe complications such as facial nerve trauma (0.39%), cerebrospinal fluid or perilymphatic fistula (0.25%), and meningitis (0.11%) can occur, CI is still considered to have low complication rates. Cochlear implants have been clinically proven effective.(24) Colletti et al. reported improved receptive language development (20–100%) after 9 years of cochlear implant use. [24] Several studies also indicate that the age at implantation affects hearing and language outcomes, with many children implanted before age two achieving speech and language abilities comparable to their peers without hearing impairment.[25]

7.3. Prevention and Screening

The primary strategy to control the incidence of CRS is achieved through immunization programs. The Centers for Disease Control (CDC) recommends the first dose of the Measles, Mumps, and Rubella (MMR) vaccine at 12–15 months of age and the second dose at 4–6 years.[26] The first dose reduces CRS complications, while the second prevents rubella virus transmission. Adults who have never been vaccinated can receive a single MMR dose. Women of childbearing age may be vaccinated one month before pregnancy.

Prevention also involves testing for rubella-specific IgM and IgG antibodies using ELISA in all women planning pregnancy and pregnant women with rubella infection symptoms. An IgG level of ≥15 IU/mL is generally considered

protective against rubella for the fetus. The Advisory Committee on Immunization Practices (ACIP) recommends avoiding pregnancy for 28 days after each dose of a live vaccine.[27] Passive immunization does not prevent fetal infection if the mother is infected.

As for the SNHL manifestation, hearing loss should be diagnosed and addressed before 6 months of age. Early detection in all newborns should occur by 1 month, diagnosis by 3 months, and intervention by 6 months. Otoacoustic emissions (OAE) and Auditory Brainstem Response (ABR) are commonly used techniques for hearing loss screening.

8. Conclusion

Congenital Rubella Syndrome (CRS) remains a significant cause of sensorineural hearing loss (SNHL) in developing countries. SNHL impairs speech and language development, necessitating early diagnosis through audiological assessments like OAE, BERA, and ASSR, ideally by 3 months, with interventions such as hearing aids, cochlear implants, and speech therapy by 6 months to optimize outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Ezike E, Steele R. Pediatric Rubella [Internet]. 2025 [cited 2025 May 12]. Available from: https://emedicine.medscape.com/article/968523-overview

[2] Hendryastuti S. Sindrom Rubela Kongenital [Internet]. 2016 [cited 2025 May 12]. Available from: https://www.idai.or.id/artikel/klinik/keluhan-anak/sindrom-rubela-kongenital

[3] Smith RJH, Bale JF, White KR. Sensorineural hearing loss in children. Lancet Lond Engl. 2005 Mar 5;365(9462):879–90.

[4] Karimi-Boroujeni M, Zahedi-Amiri A, Coombs KM. Embryonic Origins of Virus-Induced Hearing Loss: Overview of Molecular Etiology. Viruses. 2021 Jan;13(1):71.

[5] Rosen, Clark A. J Jonas T. Bailey's Head and Neck Surgery: Otolaryngology. 6th ed. Philadelphia: Lippincott Williams & Wilkins (LWW); 2022.

[6] P. Ashley W, James B. S. Ballenger's Otorhinolaryngology 18 Head and Neck Surgery. Vol. 1: Otology and Neurotology. P. Ashley Wackym, ed. Shelton, Connecticut, USA: People's Medical Publishing House. [Internet]. 18th ed. Vol. 1. Connecticut, USA: People's Medical Publishing House; 2016 [cited 2025 May 12]. Available from: https://journals.lww.com/otology-neurotology/citation/2018/06000/ballenger_s_otorhinolaryngology_18_head_and_neck.2.aspx

[7] Clark A. R. Development of the ear. In: Bailey's Head & Neck Surgery Otolaryngology [Internet]. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2022 [cited 2025 May 12]. Available from: https://www.wolterskluwer.com/en/solutions/ovid/baileys-head--neck-surgery-otolaryngology-12625

[8] Dhingra S, Dhingra PL. Diseases of Ear, Nose and Throat & Head and Neck Surgery. 2020.

[9] Organ of Corti. In: Wikipedia [Internet]. 2025 [cited 2025 May 12]. Available from: https://en.wikipedia.org/w/index.php?title=Organ_of_Corti&oldid=1268690484

[10] Kadek K, Darmadi S. GEJALA RUBELA BAWAAN (KONGENITAL) BERDASARKAN PEMERIKSAAN SEROLOGIS DAN RNA VIRUS. Indones J Clin Pathol Med Lab. 2007;13(2):63–71.

[11] World Health Organization. Global and Regional Immunization Profile [Internet]. 2020. Available from: https://cdn.who.int/media/docs/default-source/immunization/data_statistics/gs_gloprofile.pdf?sfvrsn=e932592c_6

[12] Kartasasmita C, Purbadi IMY, Tandy G, Sundoro J. Petunjuk Teknis Kampanye Imunisasi Measles Rubella (MR) [Internet]. Direktorat Jenderal Pencegahan dan Pengendalian Penyakit Kementrian Kesehatan RI; 2017.

Available from: https://cdn.who.int/media/docs/default-source/searo/indonesia/non-who-publications/2017-mr-guidance-immunization-campaign-moh-bahasa.pdf?sfvrsn=4c49454a_2

[13] CDC. Manual for the Surveillance of Vaccine-Preventable Diseases. 2025 [cited 2025 May 12]. Chapter 15: Congenital Rubella Syndrome. Available from: https://www.cdc.gov/surv-manual/php/table-of-contents/chapter-15-congenital-rubella-syndrome.html

[14] Martínez-Quintana E, Castillo-Solórzano C, Torner N, Rodríguez-González F. Congenital rubella syndrome: a matter of concern. Rev Panam Salud Publica. 2015;

[15] Nguyen TV, Pham VH, Abe K. Pathogenesis of Congenital Rubella Virus Infection in Human Fetuses: Viral Infection in the Ciliary Body Could Play an Important Role in Cataractogenesis. EBioMedicine. 2015 Jan;2(1):59–63.

[16] Giambi C, Filia A, Rota MC, Del Manso M, Declich S, Nacca G, et al. Congenital rubella still a public health problem in Italy: analysis of national surveillance data from 2005 to 2013. Euro Surveill Bull Eur Sur Mal Transm Eur Commun Dis Bull. 2015 Apr 23;20(16):21103.

[17] Soetjipto D. Tuli Kongenital [Internet]. 2015 [cited 2025 May 12]. Available from: https://id.scribd.com/presentation/440202557/TULI-KONGENITAL

[18] Lee JY, Bowden DS. Rubella Virus Replication and Links to Teratogenicity. Clin Microbiol Rev. 2000 Oct;13(4):571–87.

[19] Aminullah A, Sugiarno I, Rohsiswanto R, Batubara JRL, Dewi R. Buku Panduan Bayi Baru Lahir di Rumah Sakit [Internet]. 2010 [cited 2025 May 12]. Available from: https://www.scribd.com/doc/296543270/Buku-Panduan-Tatalaksana-Bayi-Baru-Lahir-di-RS-pdf

[20] Johnson KC. Audiologic assessment of children with suspected hearing loss. Otolaryngol Clin North Am. 2002 Aug;35(4):711–32.

[21] Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-step Learning Guide. Thieme; 2005. 442 p.

[22] Dewi YA, Agustian RA. Karakteristik Gangguan Dengar Sensorineural Kongenital pada Anak yang Dideteksi dengan Brainstem Evoked Response Audiometry. Maj Kedokt Bdg. 2011 Jun 17;43(2):77–82.

[23] Gürtler N. Hereditary Hearing Impairment. In: Lalwani AK, editor. Current Diagnosis & Treatment Otolaryngology—Head and Neck Surgery [Internet]. 4th ed. New York, NY: McGraw-Hill Education; 2020 [cited 2025 May 12]. Available from: accessmedicine.mhmedical.com/content.aspx?aid=1169079982

[24] Colletti L, Mandalà M, Zoccante L, Shannon RV, Colletti V. Infants versus older children fitted with cochlear implants: performance over 10 years. Int J Pediatr Otorhinolaryngol. 2011 Apr;75(4):504–9.

[25] Arief W, Zizlavsky S, Priyono H, Wahyuni LK, Medise BE, Prihartono J. Gambaran persepsi auditori CAP-II pada anak tuli prelingual bilateral 6–12 bulan pasca implantasi koklea. Oto Rhino Laryngol Indones. 2018 Jun 28;48(1):1–10.

[26] CDC. Measles (Rubeola). 2025 [cited 2025 May 12]. Measles Vaccination. Available from: https://www.cdc.gov/measles/vaccines/index.html

[27] CDC. Pregnancy and Vaccination. 2025 [cited 2025 May 12]. Guidelines for Vaccinating Pregnant Women. Available from: https://www.cdc.gov/vaccines-pregnancy/hcp/vaccination-guidelines/index.html