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INFECTIOUS DISEASES – PART 2: THE RELATIONSHIP BETWEEN CMV AND HEARING LOSS: FROM INJURY MECHANISM TO CLINICAL FOLLOW-UP

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This bulletin continues the “Infectious Diseases” series, exploring the impact of cytomegalovirus (CMV) on the auditory system. Prior reading of Part 1 (Vol. 40, July 2026) is recommended to broadly understand viral biology and transmission routes. Here, the focus is more clinical and audiological: how the virus damages the cochlea, what audiological patterns are expected, how to diagnose early, and how to conduct longitudinal monitoring and rehabilitation.



REMEMBERING THE INVISIBLE ENEMY

Congenital CMV infection (cCMV) is the most frequent intrauterine infection in humans, with an estimated global prevalence of 0.64% of live births, and approximately 17–20% of those infected develop long-term sequelae (Leruez-Ville et al., 2024). It is the leading non-genetic cause of sensorineural hearing loss (SNHL) in childhood and accounts for approximately 25% of SNHL cases at 4 years of age (Schleiss, 2024).

Three points synthesize Part 1 and anchor this second part:

(I) vertical transmission is more likely and more severe in primary maternal infections, especially in the first trimester;

(II) approximately 10–15% of infected newborns are symptomatic at birth, but the majority of those infected (approximately 85–90%) are asymptomatic—and still at risk for late sequelae;

(III) hearing loss is the most frequent sequela, which may be present at birth or manifest months or years later.



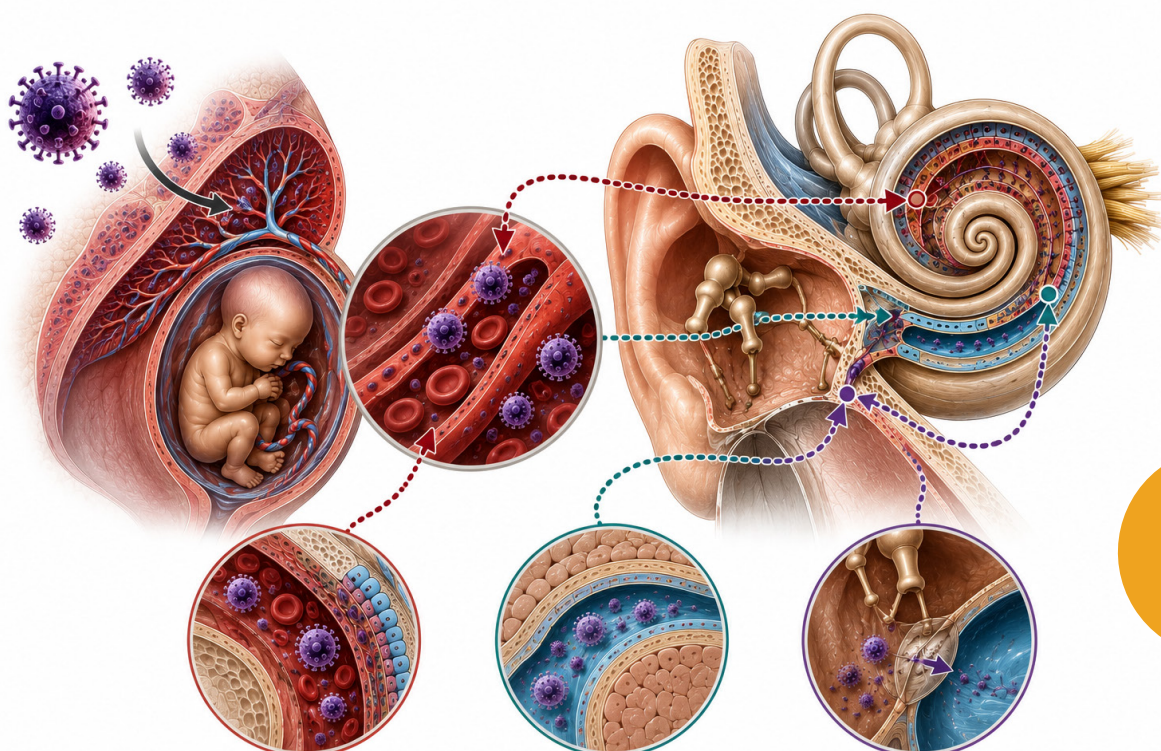
HOW CMV AFFECTS THE AUDITORY SYSTEM (PATHOPHYSIOLOGY)

The exact mechanism of SNHL associated with congenital CMV infection is not yet fully understood, but evidence from mouse models, human stem cell-derived cochlear organoids, and histopathological studies in infected fetuses converge on a multifactorial model (Gabrielli et al., 2013; Carraro et al., 2017; Teissier et al., 2020; Sung et al., 2024).

2.1. ROUTES OF COCHLEAR INVASION

After primary maternal infection or reinfection/reactivation, CMV crosses the placenta, reaches the fetal circulation, and spreads hematogenously. Three main routes allow the virus to reach the inner ear (Bradford et al., 2015; Almishaal et al., 2022).

- **Direct hematogenous route** — the virus reaches the stria vascularis through cochlear capillaries; it is considered the main route.
- **Cerebrospinal fluid route** — through the cochlear aqueduct, from the infected subarachnoid space.
- **Tympanic route** — through the round window membrane, originating from the middle ear.



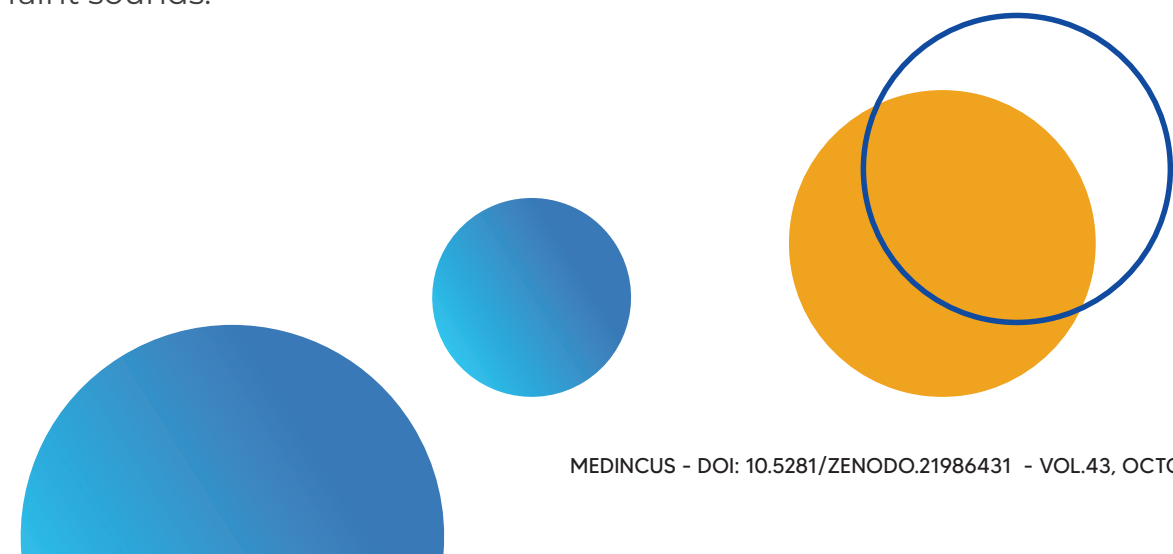
2.2. CELLULAR TARGETS AND INJURY MECHANISMS

Histopathological studies in infected human fetuses show that marginal cells of the stria vascularis are most frequently CMV-positive (Gabrielli et al., 2013). Cochlear injury occurs through two complementary mechanisms, now well documented:

- **Direct cytopathic effect** — viral replication in undifferentiated and mature cells (stria vascularis, spiral ganglion, supporting cells), leading to apoptosis of spiral ganglion neurons and loss of outer hair cells (Schachtele et al., 2011; Carraro et al., 2017).
- **Local immune/inflammatory response** — mononuclear infiltrate (CD3+) and microglial activation, increased reactive oxygen species (ROS), NLRP3 inflammasome activation and release of IL-1 β and IL-18, with disruption of the blood-labyrinth barrier (Zhuang et al., 2018; Sung et al., 2024).

A more understandable explanation of the two aspects mentioned above is that the infection can cause a defensive reaction in the inner ear, with entry of immune system cells and activation of the tissue's own protective cells (microglia). This increases the production of inflammatory substances and molecules that can damage cells (reactive oxygen species). As a consequence, inflammatory proteins (IL-1 α and IL-18) are released and the barrier that protects the inner ear is compromised, further facilitating local damage.

Furthermore, the virus can act directly on ear cells. It multiplies in both young and mature cells, such as those in the stria vascularis, spiral ganglion, and supporting cells. This process can lead to the death of neurons important for hearing and the loss of outer hair cells, which are essential for detecting faint sounds.



The dominant functional consequence is a reduction of endocochlear potential (EP) due to stria vascularis damage, which has been proven in mouse models (Almishaal et al., 2022). This explains three classic audiological findings of CMV:

- **(I) fluctuating thresholds:** because the EP can oscillate along with inflammatory activity;
- **(II) late progression:** because the loss of spiral ganglion neurons is cumulative;
- **(III) relative preservation of the auditory nerve,** which explains the good results of cochlear implants in most cases (Aldè et al., 2023).

2.3. CENTRAL AUDITORY PATHWAY INVOLVEMENT

CMV injury is not restricted to the cochlea. Recent studies show viral tropism for the temporal lobe and auditory cortex, with microglial activation proportional to tissue viral load (Carraro et al., 2024).

This finding has direct clinical implications: part of the auditory/cognitive difficulties observed in children with cCMV, even with thresholds close to normal, may reflect retrocochlear and cortical involvement, not just peripheral involvement. Therefore, the inclusion of electrophysiological assessments (ABR, P300, MMN) and auditory processing in the follow-up protocol is justified.



3. TABLES

Table 1: Summary of Target Structures, Predominant Mechanisms, and Audiological Consequences (Ferreira et al., 2026)

Target Structure	Predominant Mechanism	Audiological Consequence
Stria vascularis	Marginal cell injury; Blood-labyrinth barrier disruption; Endocochlear potential drop.	Fluctuating sensorineural loss; Progressive worsening.
Spiral ganglion	Neuronal apoptosis by direct cytopathic effect and chronic inflammation.	Progressive loss; Possible neural dyssynchrony (auditory neuropathy spectrum. disorder).
Organ of Corti	Outer hair cell dysfunction (murine models).	OAE alterations before threshold elevation.
Cortex and central pathway	Temporal lobe tropism; microglial activation.	Processing difficulties even with preserved thresholds.



Table 2: Comparison of Symptomatic versus Asymptomatic Patients at Birth

Characteristic	Symptomatic	Asymptomatic
SNHL Frequency	30–65% (up to 74% in follow-up to 18 years).	7–15% (up to 20% in longitudinal follow-up).
Age of Onset	Often at birth.	Predominantly late onset.
Predominant Degree	Moderate to profound.	Mild to moderate, with profound cases.
Follow-up Implications	Intensive evaluation; antivirals indicated.	Normal NHS does not exclude risk; maintain surveillance.

Critical Point for Practice

The absence of abnormalities in newborn hearing screening (NHS) should not authorize audiological discharge in children with confirmed cCMV. Approximately half of CMV-associated losses are of late or progressive onset. The asymptomatic group at birth requires the same monitoring rigor as the symptomatic group, although with a schedule adaptable to the risk profile (trimester of maternal infection, viral load, antiviral use).



4. THE CRUCIAL ROLE OF SCREENING AND EARLY DIAGNOSIS

4.1. THE 21-DAY DIAGNOSTIC WINDOW

The diagnosis of congenital CMV infection requires detection of the virus in saliva, urine, or blood (viral DNA by PCR) in the first 3 weeks of life. After this period, a positive sample does not allow one to distinguish congenital infection from postnatal infection (via breast milk or contact), a finding which completely changes the prognosis and management (Leruez-Ville et al., 2024; Schleiss, 2024).

Among available materials, saliva has high sensitivity and ease of collection, being the most used sample in screening programs; however, urine is considered the gold standard for confirmation. Dried blood spot (DBS), although attractive because it is already collected in newborn screening, has lower sensitivity (around 30–70% depending on the method).

Dried Blood Spot (DBS)

- A blood collection method in which a few drops of blood (usually from a newborn's heel prick or finger) are dropped onto a specific filter paper card.
- This blood is allowed to dry on the paper, forming small dried spots that are later cut out and analyzed in the laboratory.
- The great advantage is that very little blood is needed, collection is quick and minimally invasive, and the dried material is stable and easy to store and send to a laboratory (which is why it is widely used in newborn screening).

4.2. TARGETED VS. UNIVERSAL SCREENING

There are two main cCMV screening models:

- **Targeted screening (“hearing-targeted”):** for investigating CMV in newborns who have failed NHS. It is a low-cost strategy, but fails to identify the majority of cases (some 85–90% of children with cCMV pass NHS at birth). This is the main strategy today where there are structured programs.
- **Universal screening** — saliva PCR (with urine confirmation) in all newborns. In an analysis of 96,785 newborns in the USA, universal screening was found to be more cost-effective and prevented more cases of profound loss than targeted screening, even in low-prevalence regions (Gantt et al., 2024)..

Intermediate strategies, such as saliva pooling screening (Yinon et al., 2024) and expansion to high-risk groups (premature, low birth weight, NICU admissions) have been implemented internationally as a transition towards universalization.



4.3. CRITICAL ANALYSIS FOR THE BRAZILIAN SCENARIO

In Brazil, newborn hearing screening (NHS) is one of the main warning signs for possible hearing loss in babies, and congenital CMV is recognized as part of the STORCH+Z group (Joint Committee on Infant Hearing, 2019; Ministry of Health, 2025). However, systematic collection of CMV PCR in neonates who fail NHS, or in risk groups, has not yet been uniformly incorporated into the Unified Health System. Some identified bottlenecks are:

- Heterogeneous laboratory availability: not all maternity hospitals have saliva or urine PCR within 21 days.
- Low return adherence: non-attendance rates at the diagnostic stage (>20% in some Brazilian series) compromise the critical collection time.
- Absence of a single national protocol for management after confirmed cCMV (antiviral use, monitoring frequency, referral).
- Lack of systematic prenatal education on behavioral prevention: despite evidence of reduction in maternal seroconversion with hygiene measures.

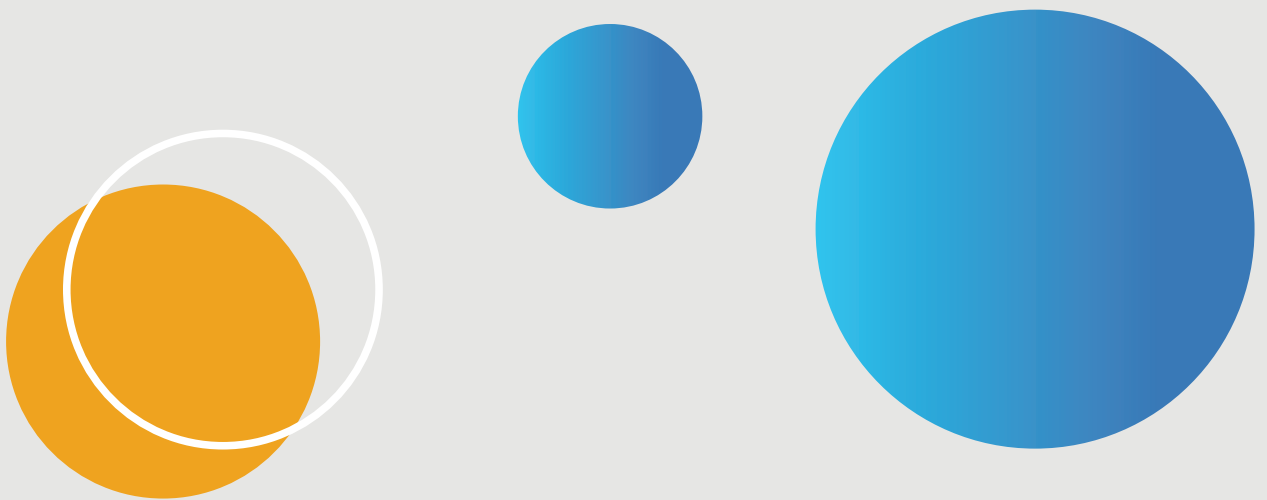
Applied Research Opportunity

There is the potential for Brazilian multicenter studies evaluating:

- (1) the cost-effectiveness of universal saliva screening compared to the current targeted strategy;
- (2) the feasibility of saliva pooling in public maternity hospitals;
- (3) the impact of early valganciclovir initiation in symptomatic Brazilian newborns on audiological outcomes at 24 months;
- (4) o desempenho de estratégias híbridas combinando DBS + saliva.

CHECKLIST – EARLY DIAGNOSIS OF cCMV

- Collect saliva or urine for PCR before the 21st day of life; preferably before the 1st month.
- Confirm positive saliva sample with urine (which rules out breast milk contamination).
- For collection done after 21 days with a positive PCR, one needs to evaluate retroactive DBS to differentiate congenital from postnatal infection.
- Complete clinical evaluation in newborns with cCMV: weight, head circumference, neurological examination, complete blood count, transaminases, bilirubin, fundoscopy, among others.
- Request neuroimaging (transfontanellar ultrasound or brain MRI) for symptomatic/asymptomatic classification.
- Refer to specialized audiology service before 3 months of age, even with normal NHS.





5. AUDIOLOGICAL MONITORING: A CONTINUOUS JOURNEY

There is not yet an internationally standardized protocol for auditory follow-up of children with cCMV, but three high-level references converge: the American Academy of Audiology (AAA), the Joint Committee on Infant Hearing (JCIH, 2019), and the European ECCI consensus (Leruez-Ville et al., 2024). The synthesis recommended by Aldè et al. (2024) is the most used in tertiary centers.

5.1. Proposed Schedule for Auditory Follow-up of Children with cCMV

Age Range	Evaluation Frequency	Recommended Battery
0–3 months	Complete diagnostic evaluation.	ABR-click and tone burst, OAE, tympanometry.
3–12 months	Every 3–6 months.	VRA (visual reinforcement audiometry), OAE, tympanometry; ABR if necessary.
1–3 years	Every 6 months.	Conditioned audiometry, OAE, tympanometry, language milestones.
3–6 years	Annual.	Pure tone and speech audiometry; processing assessment if school complaints.
>6 years	Annual or biennial, individualized.	Audiometry, central auditory processing, electrophysiology if indicated.

5.2. Vestibular Assessment is Often Forgotten but Let us Remind You

Vestibular dysfunction is highly prevalent in children with cCMV (estimates range from 30–70%) and impacts motor milestones, balance, and school performance (Aldè et al., 2024).

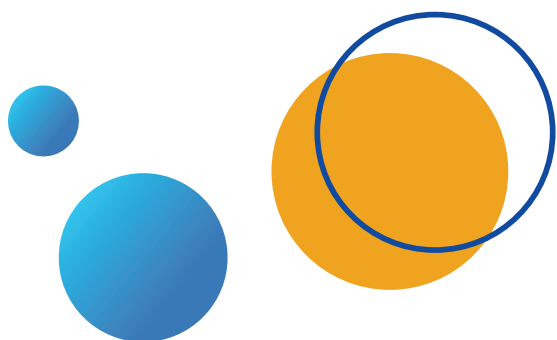
It is recommended to include, whenever available, vHIT (video Head Impulse Test) and cVEMP (cervical vestibular evoked myogenic potentials) in the initial evaluation and periodic reviews, especially in symptomatic children or those who will receive cochlear implants.

5.3. Risk-Based Personalization

Aldè et al. (2024) propose stratification of follow-up by risk factors:

- trimester of maternal infection;
- viral load in amniotic fluid;
- valganciclovir use;
- neuroimaging findings;
- presence of neurodevelopmental comorbidities.

This stratification optimizes resources and reduces the risk of missing the intervention window, a topic little explored in Brazilian studies and which is a promising research agenda.



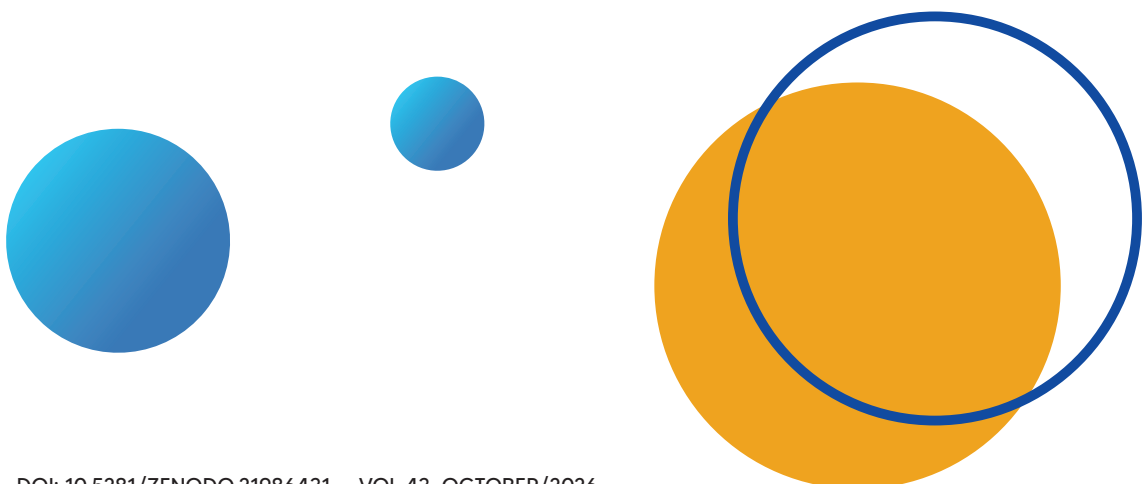
6. INTERVENTION AND AUDITORY REHABILITATION

6.1. ANTIVIRAL TREATMENT: WHAT CURRENT EVIDENCE SHOWS

Oral valganciclovir for 6 months is the treatment of choice for symptomatic newborns with cCMV, ideally started before 30 days of life (Kimberlin et al., 2015; Leruez-Ville et al., 2024), with monitoring of neutropenia and liver function being important.

Critical points based on recent evidence:

- **Symptomatic with onset <30 days:** the Collaborative Antiviral Study Group trial showed audiological and neurodevelopmental benefit of 6-month treatment compared to 6 weeks (Kimberlin et al., 2015). This is the scenario with the most robust evidence.
- **Onset after 30 days:** the placebo-controlled randomized trial by Kimberlin et al. (2024) showed that starting valganciclovir after the first month of life did not improve audiological outcomes, despite reducing viral load in saliva and urine. This finding restricts the indication for antiviral outside the neonatal window.
- **Asymptomatic with isolated hearing loss:** the ValEAR trial (Park et al., ongoing) seeks to answer whether there is benefit from antivirals in this group – currently without clear evidence and routine treatment is not recommended.
- **Asymptomatic without hearing loss:** there is no indication for antivirals; maintain monitoring.



6.2. HEARING AIDS

For mild to severe sensorineural hearing loss, hearing aids remain the first line. Certain features of cCMV call for caution:

- **Fluctuating thresholds** may require more frequent re-evaluations and adjustments.
- **Progressive losses** require periodic reprogramming and anticipation of criteria for cochlear implantation.
- **For unilateral losses**, CROS/BiCROS technologies and FM/RM systems are particularly useful in school environments.

6.3. COCHLEAR IMPLANT (CI): CURRENT OUTCOMES

For bilateral severe to profound losses, cochlear implantation is now the standard of care/rehabilitation. The Italian multicenter study by Forli et al. (2025), with 53 children with cCMV implanted at 5 tertiary centers, offers recent and clinically relevant data:

- 47% of children achieved CAP level ≥ 6 (understanding speech in everyday conversation).
- 23% achieved level 6 on the Bates scale (advanced expressive language).
- Symptomatic status at birth correlated weakly, but significantly, with worse auditory and language scores.
- Neurodevelopmental comorbidities (present in 36% of the sample) were the main negative predictor of outcome.
- Brain MRI findings classified by the Alarcón scale showed that moderate to severe impairments were associated with comorbidities, but not in isolation, with worse post-CI results.

This analysis reinforces that a CI offers substantial benefits even in children with moderate comorbidities, but family counseling should be realistic: the outcome is a function of CMV plus the associated neurodevelopmental profile, and not just the device.

6.4. SPEECH THERAPY AND LANGUAGE STIMULATION

Speech-language therapy intervention is a key factor and should begin before the device is defined, along with family guidance, parental training, and early auditory stimulation.

Recent evidence (Donaldson et al., 2026) showed that children with cCMV have a higher rate of speech dysfunction than previously reported, including in children without diagnosed hearing loss, which expands the indication for early speech-language evaluation for all confirmed cases, regardless of initial auditory status.

The principles of intervention, according to the JCIH early identification and intervention approach (1-3-6) are:

- Hearing screening in the 1st month.
- Definitive audiological diagnosis by the 3rd month.
- Intervention initiation (hearing aids, speech therapy, family program) by the 6th. month.

Children identified and beginning intervention before 6 months show significant gains in receptive and expressive language compared to those with later intervention (Yoshinaga-Itano et al., 1998 and subsequent replications).



6.5. SUMMARIZED DECISION FLOW

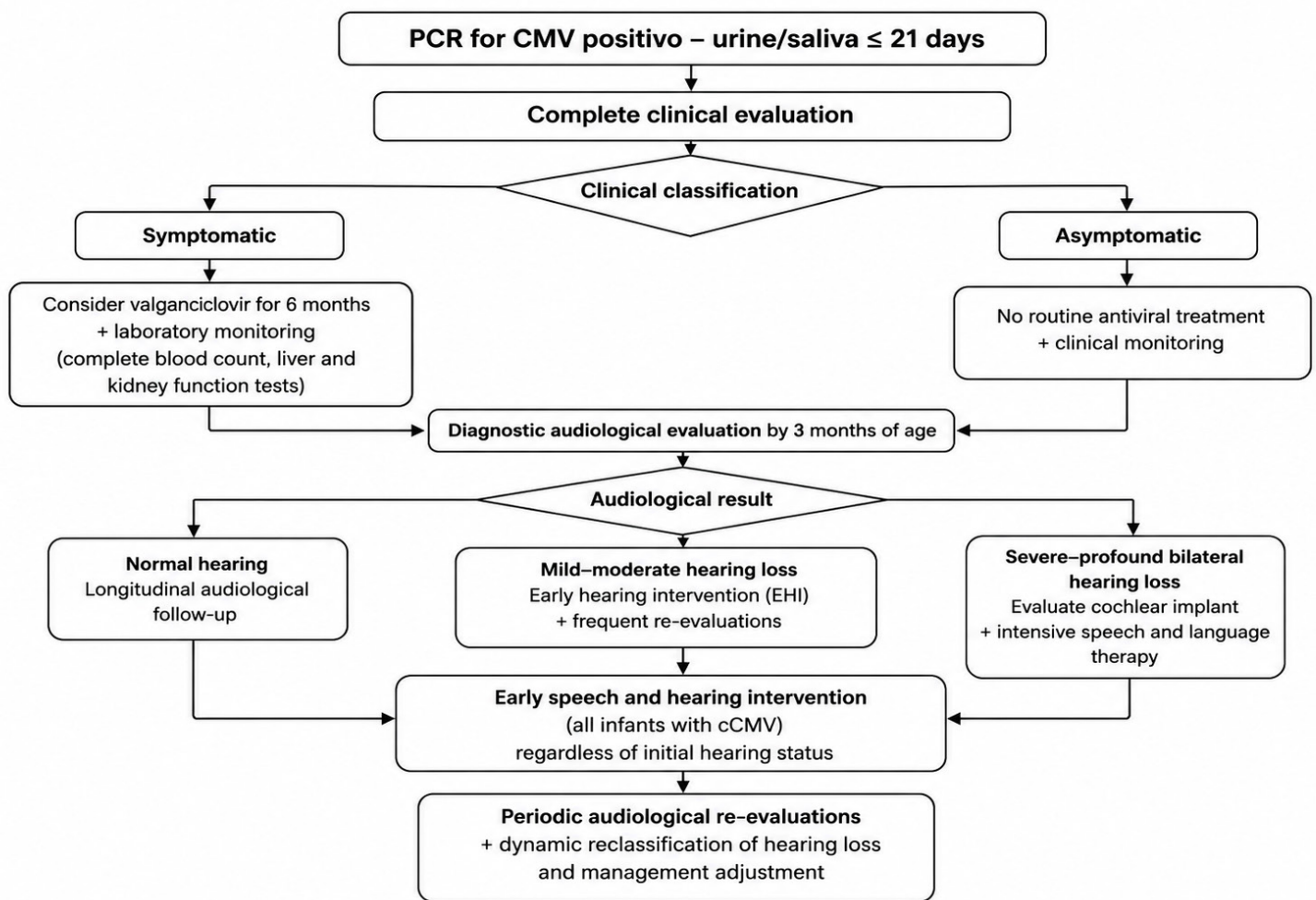


Figure 1: Management Flowchart for Newborns with cCMV.

7. CRITICAL ANALYSIS

7.1. STRENGTHS OF CURRENT KNOWLEDGE

- Increasingly well-characterized pathophysiological mechanisms (stria vascularis, spiral ganglion, inflammation).
- Robust evidence for 6-month valganciclovir in symptomatic newborns started <30 days.
- Consensual monitoring schedules (AAA, JCIH, ECCI 2024).
- Good success rates with a CI when there is multidisciplinary support and absence of severe comorbidities.

7.2. GAPS AND BOTTLENECKS

- Absence of available effective maternal vaccine (despite candidates in advanced phases).
- Absence of conclusive evidence for treatment of asymptomatic cases with isolated loss (awaiting ValEAR).
- Lack of standardized vestibular protocols, despite high prevalence of dysfunction.
- Underdiagnosis of central auditory involvement; tools such as P300 and auditory processing are poorly applied in this group.
- In Brazil: heterogeneity of access to CMV PCR within 21 days; absence of unified national protocol.

8. CLOSING REMARKS

Hearing loss associated with congenital CMV is heterogeneous, often late, and partially modifiable when there is timely diagnosis, multidisciplinary intervention, and longitudinal follow-up.

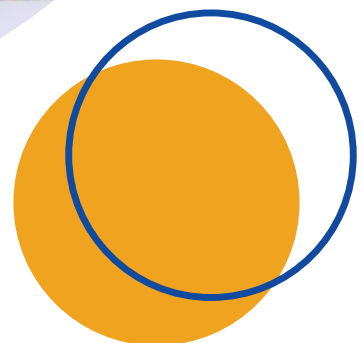
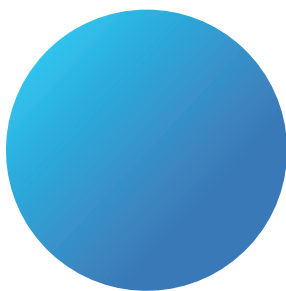
The JCIH 1-2-3 model remains valid as a clinical goal, but needs to be adapted to the reality of cCMV, since NHS evaluation alone is not enough and monitoring extends to school age.

For speech-language pathologists, otolaryngologists, pediatricians, and screening teams, three operational premises summarize what the recent literature supports:

- **Diagnosis before 21 days:** the laboratory window is narrow and directly impacts antiviral indication.
- **Structured longitudinal monitoring:** half of losses only appear after birth; audiological discharge of children with cCMV should not be given without clear criteria.
- **Multidisciplinary intervention before 6 months:** audiology, speech-language pathology, pediatric infectious diseases, neuropsychiatry, otolaryngology, working with shared objectives and defined timelines.

We thank you for following us in reading this bulletin and expanding your knowledge about cCMV and its auditory aspects. We hope that the analyses, decision flows, and synthesised evidence contribute to the care of babies and children in your practice.

We invite you to follow us in upcoming editions, where we will address other critical topics about infectious diseases related to audiology.



Quiz

1. An asymptomatic pregnant woman presents serology compatible with primary cytomegalovirus (CMV) infection in the first trimester. Regarding the risk of sensorineural hearing loss (SNHL), which statement is most appropriate?

- a) cCMV is a rare cause of SNHL in childhood, accounting for less than 5% of cases.
- b) The majority of newborns with cCMV are symptomatic at birth, with a high rate of major malformations.
- c) cCMV is the leading non-genetic cause of SNHL in childhood and is responsible for a relevant proportion of cases at 4 years of age.
- d) Hearing loss related to cCMV is always present at birth and does not have a progressive or fluctuating character.

2. Regarding the routes of inner ear invasion by CMV, which alternative describes ONE of the proposed routes and its mechanism?

- a) Exclusively tympanic route, through the eustachian tube, without participation of other structures.
- b) Direct hematogenous route, with the virus reaching the stria vascularis through cochlear capillaries.
- c) Direct bone route, with erosion of the otic capsule and virus entry into the cochlea.
- d) Exclusively cerebrospinal fluid route, starting from the spinal cord toward the auditory nerve.

3. According to the text, which combination represents a typical finding of cochlear pathophysiology in cCMV and its functional consequence?

- a) Increased endocochlear potential due to stria vascularis hyperactivity, resulting in auditory hypersensitivity.
- b) Complete preservation of outer hair cells, with exclusive auditory nerve impairment.
- c) Reduction of endocochlear potential due to stria vascularis injury, with fluctuating and progressive auditory thresholds.
- d) Isolated auditory cortex injury, without alteration of cochlear peripheral structures.



4. Regarding the diagnostic window and laboratory methods for confirmation of congenital CMV infection, select the CORRECT alternative:

- a) Detection of viral DNA by PCR at any age defines congenital CMV infection.
- b) Saliva is considered the gold standard for confirmation, while urine is only for screening.
- c) Confirmation of cCMV requires virus detection in the first 3 weeks of life, with urine being the gold standard.
- d) Dried blood spot (DBS) has sensitivity close to 100% and completely replaces saliva and urine.

5. According to the material, which alternative best describes the indication for prolonged audiological monitoring in children with cCMV?

- a) Hearing losses related to cCMV are invariably stable from birth, dispensing with follow-up after the first year.
- b) Approximately half of hearing losses associated with cCMV are of late or progressive onset, requiring a structured schedule until school age.
- c) The risk of hearing loss is limited to the symptomatic group at birth, with no need for follow-up of the asymptomatic group.
- d) A normal newborn hearing screening (NHS) test is sufficient for definitive discharge in all cases of confirmed cCMV.

6. Regarding the use of oral valganciclovir in cCMV management, select the alternative that best reflects the evidence presented:

- a) Treatment with valganciclovir for 6 months presents robust audiological benefit even when started after the first month of life.
- b) In symptomatic newborns, 6-month treatment started by 30 days of life showed better audiological and neurodevelopmental outcomes than shorter regimens.
- c) In asymptomatic children without hearing loss, there is routine indication of valganciclovir to prevent late sequelae.
- d) Reduction of viral load in saliva and urine guarantees, by itself, improvement in audiological outcomes at any treatment start age.

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Correct answers:

1. Correct answer: C
2. Correct answer: B
3. Correct answer: C
4. Correct answer: C
5. Correct answer: B
6. Correct answer: B

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