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Article

Impact of Cytomegalovirus and Toxoplasma Infections on Hearing in Children in Early Childhood and Preschool Age: Clinical and Audiological Study

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Abstract: Objective: The present investigation was designed to characterize in detail the clinical manifestations and audiological profile of sensorineural hearing loss (SNHL) in pediatric patients with concurrent Toxoplasma gondii and cytomegalovirus (CMV) infections.

Materials and Methods: A total of 115 children aged between 1 and 7 years diagnosed with SNHL in association with toxoplasmosis and CMV infection were enrolled as the main study cohort. The control group consisted of 30 children with SNHL in whom no evidence of toxoplasma or CMV infection was detected. Patients with hereditary forms of hearing loss, central nervous system pathology, congenital anomalies of the auditory system, acute or chronic purulent otitis media, prior exposure to ototoxic agents, or a history of ear trauma were excluded from the analysis. Auditory function was assessed using short-latency auditory evoked potentials (SLAEP).

Results: Analysis of the primary cohort demonstrated that mild (grade I) SNHL was present in 14.8% of cases, moderate (grade II) in 30.4%, severe (grade III) in 46.1%, and profound (grade IV) in 8.7% of patients. In the comparison group, grade I SNHL was identified in 13.3% of children, grade II in 33.3%, while severe and profound impairment (grades III–IV) were observed in 26.7% of cases with equal distribution. Bilateral involvement was documented in all examined patients, with no cases of unilateral hearing loss detected. Importantly, a predominance of severe and profound bilateral SNHL (grades III–IV) was noted in both cohorts, particularly in children with infectious comorbidity.

Conclusions: The obtained data indicate a tendency toward more pronounced bilateral sensorineural hearing impairment in children with combined toxoplasma and CMV infection, with nearly half of the patients exhibiting severe degrees of hearing loss. These findings emphasize the clinical importance of своевременной audiological screening and structured follow-up in this high-risk pediatric population.

Keywords: cytomegalovirus infection, toxoplasmosis, sensorineural hearing loss, pediatric audiology, auditory evoked potentials

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1. Introduction

Despite substantial advancements in otorhinolaryngology, sensorineural hearing loss (SNHL) remains a prominent contributor to pediatric morbidity. According to World Health Organization data, the prevalence of hearing impairment in children ranges from 1% to 2%, with SNHL accounting for approximately 0.02% of these cases [1],[2],[3],[4]. Timely identification of SNHL associated with toxoplasmosis and cytomegalovirus infection (CMVI) in early and preschool children remains a significant clinical challenge . Determination of etiological factors, comprehensive assessment of obstetric and perinatal history, and initiation of etiopathogenetic interventions, combined with early rehabilitation strategies, constitute essential components of pediatric otorhinolaryngologic care.

Study Aim. To delineate the clinical and audiological profile of SNHL in children with concurrent toxoplasmosis and CMV infection, facilitating early diagnosis and optimizing management strategies.

2. **Materials and Methods**

The present study encompassed 115 children aged 1–7 years presenting with sensorineural hearing loss (SNHL) concomitant with toxoplasmosis and cytomegalovirus infection, constituting the primary study cohort. The control cohort comprised 30 children diagnosed with SNHL in the absence of toxoplasmosis or CMV infection. Exclusion criteria encompassed offspring of consanguineous unions, individuals with a hereditary predisposition to hearing impairment, central nervous system pathologies, congenital malformations of the auditory apparatus, acute or chronic suppurative otologic conditions, prior exposure to ototoxic pharmacological agents, or a history of cranial or otologic trauma.

Endoscopic evaluation was performed utilizing a Suntem ENT workstation (China) in conjunction with rigid endoscopes of 4.0 mm and 2.7 mm diameters with a 0° viewing angle (Delon, Germany), enabling detailed visualization of the tympanic membrane under both physiological and pathological conditions. Functional assessment of membrane mobility was concurrently performed. The endoscopic system projected a magnified real-time image of the tympanic membrane onto a monitor, while an LED 300 computer system (Delon, Germany) facilitated high-resolution archiving of photographic and video documentation.

Auditory Brainstem Response (ABR) recordings were obtained using surface electrodes positioned according to the standardized montage: A1 and A2 on the mastoid processes of both temporal bones, Fz (active electrode) at the midline of the hairline, and Fpz (ground electrode) at the midline of the forehead, inferior to the active electrode. Inter-electrode impedance was maintained below 5 kΩ. Acoustic stimuli were delivered via EARtone 3A air-conduction headphones and a B 70A bone vibrator. Dual-channel recording was employed to minimize potential artifact contamination. Detailed stimulation and recording parameters are summarized in Table 1.

Table 1. Parameters of registration of short-latency auditory evoked potentials.

Parameter	Clicks, CHIRP, Tone Bursts (2 kHz, 4 kHz)	Tone Bursts (0.5 kHz, 1 kHz)
Electrode Placement	**Non-inverting (+):** High forehead (avoiding fontanelle), back of the neck. **Inverting (-):** Ipsilateral mastoid, earlobe. **Ground:** Contralateral mastoid, forehead (closer to the bridge of the nose).	
Stimulus Polarity	Alternating; condensation/rarefaction for CM (Cochlear Microphonic)	
Stimulus Duration	100 µs; 2-1-2 cycles; Blackman window; 7.75 ms CHIRP	
Stimulus Rate	47 Hz–57 Hz; 21–29 Hz (for Wave I and Bone Conduction)	37–39 Hz
Rejection Level	3–10 µV	
Frequency Filters	30–1500 Hz (Children); 100–2000 Hz (Adults)	
Analysis Window (Epoch)	15–20 ms	20–25 ms
Number of Averages	Standard: 2000 (up to 5000). 1000–1500 possible for CHIRP.	
Display Parameters	X-axis (Time): 1–2 ms/div Y-axis (Amplitude): 0.1–0.2 µV/div	

The subsequent phase of the study aimed at corroborating the preliminary findings involved recording standard Auditory Brainstem Responses (ABR) across a spectrum of stimulus intensities ranging from 10 to 70 dB. Evaluation criteria encompassed: (1) latencies of ABR wave peaks (I, III, V); (2) interpeak intervals (IPI) between waves I–III, III–V, and I–V; (3) the intensity–latency function during ABR acquisition; and (4) interaural discrepancies in the aforementioned parameters [5],[6],[7].

Molecular confirmation of toxoplasmosis and cytomegalovirus (CMV) infections in biological specimens was performed through detection of pathogen-specific DNA. Qualitative and quantitative assessment of toxoplasma and CMV DNA in plasma

samples was achieved using real-time polymerase chain reaction (qPCR) with hybridization-fluorescence detection on a qTOWER analyzer (Germany), employing the commercial reagent kits *AmpliSens® Toxoplasma-screen/monitor-FL* and *AmpliSens® CMV-screen/monitor-FL*. The analytical characteristics of the assay included a sensitivity threshold of 400 copies/ml and a linear quantification range of 400–10,000,000 copies/ml. Values exceeding 10,000,000 copies/ml were reported as >10,000,000 copies/ml, whereas values below 400 copies/ml were reported as <400 copies/ml. Validation of assay specificity was performed using the AD169 CMV reference strain.

Patient monitoring was conducted both in the inpatient pediatric neurology unit and on an outpatient basis within the consultative polyclinic department of the multidisciplinary clinic at SamSMU. The diagnosis of bilateral sensorineural hearing loss (H90.3, ICD-10) adhered to WHO criteria and was substantiated by comprehensive anamnesis, clinical examination, laboratory investigations, and instrumental diagnostics. All subjects underwent general clinical, immunological, and virological assessments. Confirmation of toxoplasmosis and CMV infection relied upon enzyme-linked immunosorbent assay (ELISA) and PCR. Additionally, all participants underwent endoscopic otoscopy, tympanometry, otoacoustic emissions (OAE), and ABR testing.

The study cohort included children aged 1–7 years. Following contemporary age classifications [8],[9], participants were stratified into two subgroups:

- Subgroup 1 (1–3 years): 65 children (56.5%) in the primary cohort exhibited PCR-confirmed concomitant toxoplasmosis and CMV infection; the control cohort included 14 children (46.7%).
- Subgroup 2 (3–7 years): 50 children (43.5%) in the primary cohort and 16 children (53.3%) in the control cohort demonstrated PCR positivity for both infections.

3. Results and discussion

Virological assessment through continuous-sampling PCR revealed that, among 355 children evaluated, 115 (32.4%) with chronic SNHL exhibited concomitant toxoplasmosis and CMV infection.

Analysis of the clinical manifestations of SNHL associated with the combined infections indicated specific indirect indicators. In the primary cohort aged 1–3 years with first-degree hearing loss, these manifestations were absent. However, in children with second-degree hearing loss, 22 (33.8%) exhibited limited vocabulary, 7 (10.7%) produced simple words with multiple phonetic errors, and 13 (20.0%) demonstrated articulatory inaccuracies (Fig. 1).

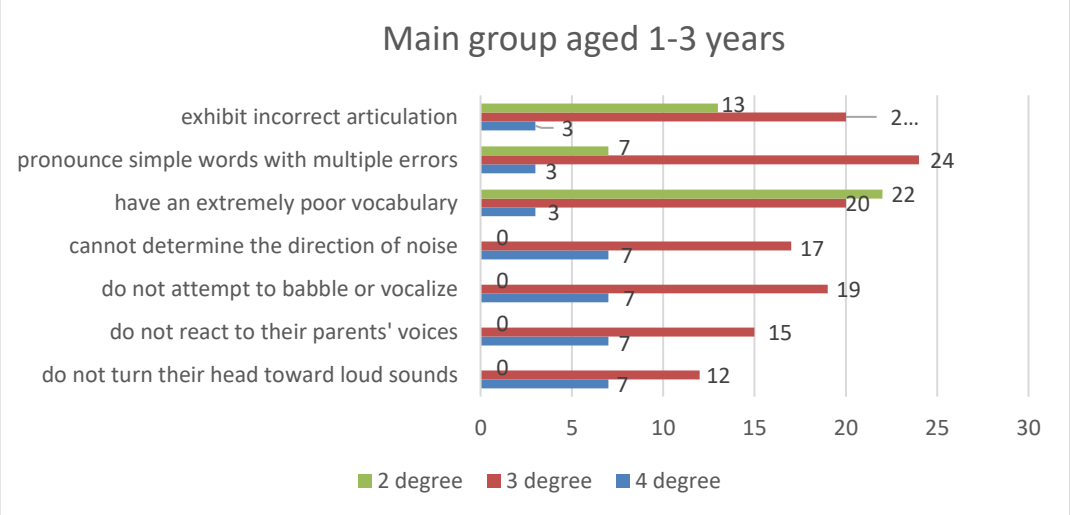


Figure 1. Clinical criteria of SHL in children aged 1-3 years in the main group

In the control group, children aged 1–3 years with degree I hearing loss did not exhibit any indirect signs. Among children in the control group with degree II hearing loss, 2 (14.3%) had a limited vocabulary, 2 (14.3%) pronounced simple words with multiple errors, and 1 (7.1%) patient exhibited incorrect articulation (Fig. 2).

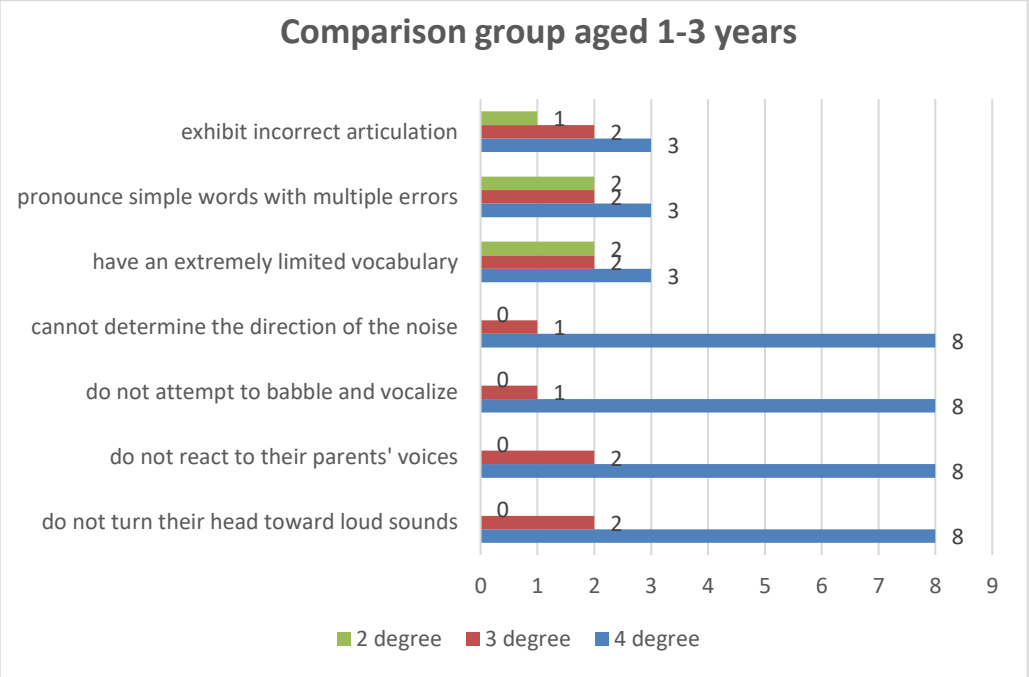


Figure 2. Clinical criteria of SHL in children aged 1-3 years in the comparison group

Within the primary cohort, among children exhibiting third-degree (III) hearing loss in this age category, 12 (18.4%) failed to orient their head toward a salient auditory stimulus, 15 (23.0%) demonstrated no response to parental vocalizations, 19 (29.2%) exhibited absent vocal babbling or spontaneous vocalizations, 17 (26.1%) were unable to localize the direction of ambient noise, and 20 (30.7%) manifested a restricted lexicon accompanied by articulatory inaccuracies. Additionally, 24 (36.9%) of these children produced words with multiple phonetic errors (Fig. 1). In the control cohort with comparable III-degree hearing loss, 2 (14.3%) children did not orient to salient auditory stimuli or parental voices, presented with extremely limited vocabulary, produced simple words with multiple errors, and exhibited defective articulation; only 1 (7.1%) patient failed to initiate babbling or vocalization and was unable to localize noise sources (Fig. 2).

Among children with fourth-degree (IV) hearing loss in the primary cohort, 7 (10.7%) did not respond to loud auditory stimuli or parental vocalizations, failed to produce vocalizations, and were unable to discern the direction of sound. Notably, 3 (4.6%) children with IV-degree hearing loss had been fitted with hearing aids; despite amplification, these children displayed extremely limited vocabulary, articulatory errors, and produced simple words with multiple phonetic inaccuracies (Fig. 1). In the control cohort, 8 (57.1%) children with IV-degree hearing loss demonstrated absent orienting to loud sounds and parental voices, lack of babbling or vocalization, and inability to localize auditory stimuli; 3 (21.4%) exhibited severely restricted vocabulary, articulatory distortions, and produced simple words with multiple errors. Hearing aid fitting had been performed in this group prior to evaluation (Fig. 2).

Figures 3 and 4 illustrate the clinical assessments of children aged 3–7 years with SNHL in both primary and control cohorts. In the primary group with first-degree (I) hearing loss, 1 (2%) patient exhibited markedly reduced speech intelligibility, syllabic distortions, erroneous phoneme usage, and lexical errors affecting phrase and sentence construction (Fig. 3).

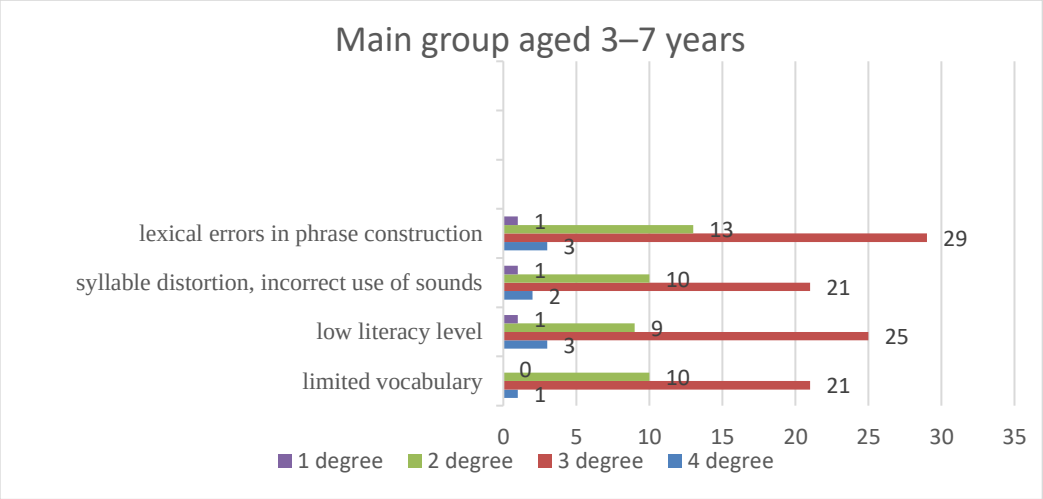


Figure 3. Clinical criteria of SHL in children aged 3-7 years old in the main group

In the control cohort, the aforementioned clinical manifestations of hearing loss were not observed (Fig. 4). Within the primary cohort, among 10 children (20%) presenting with second-degree (II) hearing loss, restricted vocabulary, syllabic distortions, and improper phoneme usage were documented. Additionally, 9 children (18%) exhibited markedly reduced literacy skills, and 13 children (26%) demonstrated lexical errors affecting phrase and sentence construction (Fig. 3). In the comparison cohort of children aged 3–7 years with II-degree hearing loss, 3 children (18.7%) presented with limited vocabulary and lexical inaccuracies in phrase formation. Only 1 child (6.2%) exhibited low literacy, whereas 5 children (31.2%) displayed syllabic distortions and improper phoneme articulation (Fig. 4).

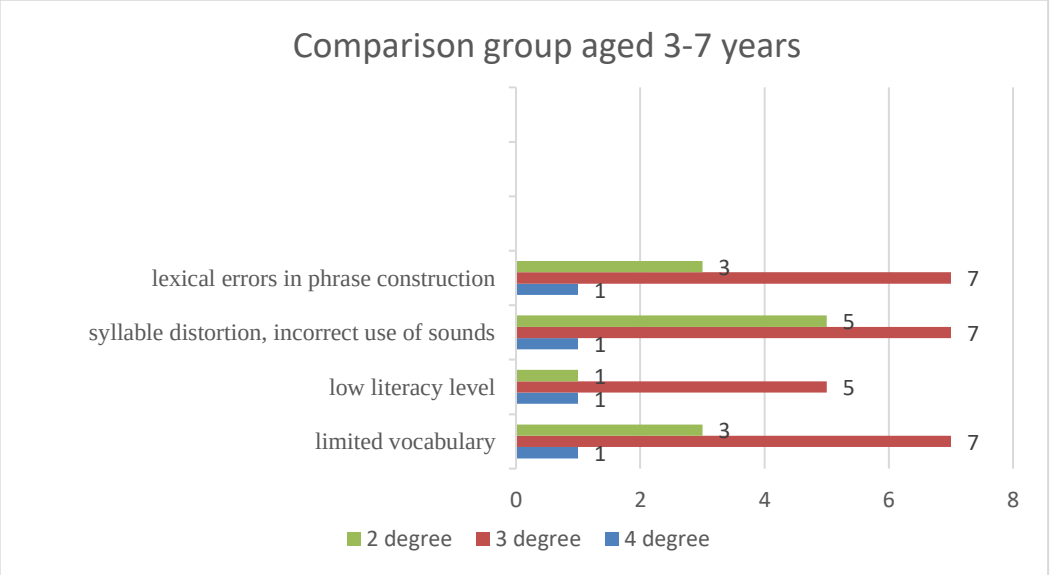


Figure 4. Clinical criteria of SHL in children aged 1-3 years in the comparison group

Among children aged 3–7 years in the primary cohort with third-degree (III) hearing loss, 21 (42%) demonstrated restricted vocabulary, syllabic distortions, and incorrect phoneme usage, 25 (50%) exhibited markedly reduced literacy skills, and 29 (58%) manifested lexical errors in sentence construction (Fig. 3). In the corresponding comparison cohort, 7 children (43.7%) exhibited limited vocabulary, syllable distortion with incorrect phoneme articulation, and lexical errors, while 5 children (31.2%) presented with low literacy levels (Fig. 4).

Within the 3–7-year age category of the primary cohort with fourth-degree (IV) hearing loss, 1 child (2%) exhibited restricted vocabulary, 2 children (4%) displayed syllabic distortions with incorrect phoneme usage, and 3 children (6%) demonstrated both low literacy and lexical errors in sentence construction (Fig. 3). In the comparison cohort, a

single patient (6.2%) with IV-degree hearing loss exhibited all indirect clinical indicators of hearing impairment (Fig. 4).

Audiological evaluation was performed to determine the severity of hearing loss in the study population. Interpretation of findings was conducted in accordance with WHO guidelines, and the diagnosis of sensorineural hearing loss was classified according to the internationally recognized degrees of hearing impairment endorsed by the WHO (Table 2).

WHO International Classification of Hearing Loss . Table 2

Degree	Decibel
I-degree	26- 40 dB
II-degree	41-55 dB
III-degree	56-70 dB
IV-degree	71-90 dB
Deafness	over 91 dB

Based on the classification, the degree of hearing loss in the speech frequencies of the better-hearing ear was evaluated as follows: 26–40 dB was assessed as degree I; degree II was 41–55 dB; degree III was 56–70 dB; degree IV was 71–90 dB; and deafness was more than 91 dB.

The results of the conducted study regarding the degree of hearing loss are reflected in the following diagram (Fig. 5).

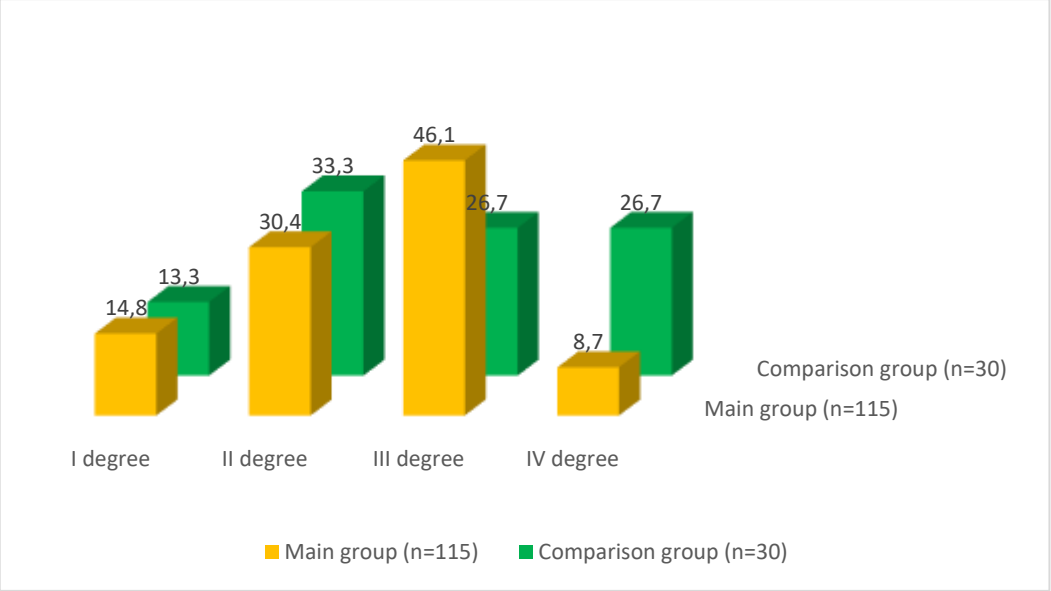


Fig.5. Degrees of SHL according to the results of SHLAEP in the examined groups.

The obtained results based on ABR showed that in the main group, degree I SNHL was recorded in 17 (14.8%) patients, while in the control group, it was found in 4 (13.3%) children. Degree II SNHL was recorded in 35 (30.4%) children in the main group and 10 (33.3%) in the control group. Degree III SNHL was recorded in 53 (46.1%) children of the main group and 8 (26.7%) children of the control group. Degree IV SNHL was recorded in 10 (8.7%) children of the main group and 8 (26.7%) children of the control group.

Using this classification, the reaction to whispered and conversational speech was additionally determined in our examined children to evaluate the degree of hearing loss. Table 3 presents the data on the degree of hearing loss based on the ratio of whispered and conversational speech in the study groups.

Comparative results of the degree of SHL in children in both groups in relation to whispered and spoken speech

Table 3

Degree of hearing loss	Parameters	Whispered speech			
		Main group		Comparison group	
		Amount (115)	%	Amount (30)	%
I degree (26-40 dB)	Up to 3 meters	8	6,9	2	6,6
II degree (41-55 dB)	Up to 0.5 meters	14	12,2	5	16,7
III degree (56-70 dB)	At the ear	12	10,4	3	10,0
IV degree (71-90 dB)	Not audible	7	6,1	5	16,7
		Conversational speech			
I degree (26-40 dB)	Up to 6 meters	9	7,8	2	6,6
II degree (41-55 dB)	Up to 3 meters	21	18,3	5	16,7
III degree (56-70 dB)	Up to 0.5 meters	41	35,7	5	16,7
IV degree (71-90 dB)	Loud speech at the ear	3	2,6	3	10,0

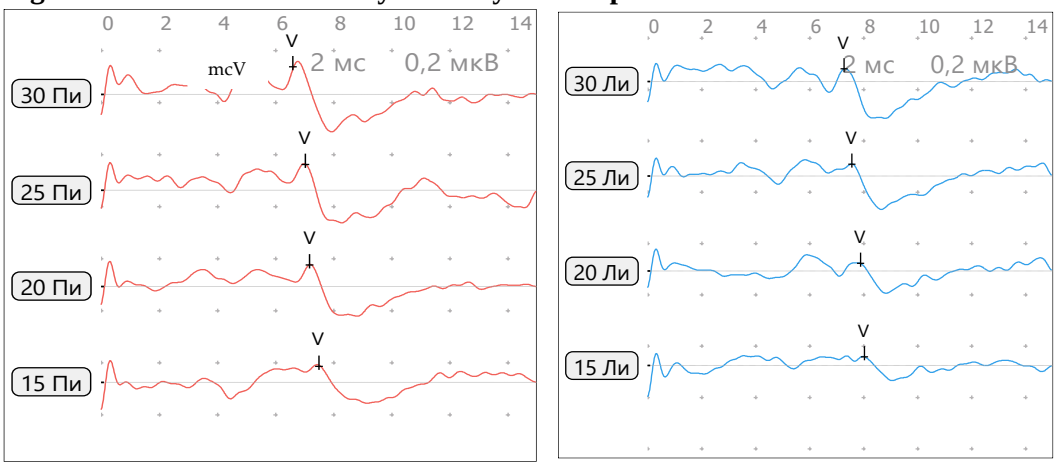
As presented in Table 3, within the primary cohort, first-degree (I) hearing loss was detected via whispered speech assessment at a distance of up to 3 meters in 8 children (6.9%), whereas in the comparison cohort, this parameter was observed in 2 children (6.6%). Whispered speech perception at a proximity of 0.5 meters was identified in 14 children (12.2%) with second-degree (II) hearing loss in the primary group and in 5 children (16.7%) in the control group. Among children with third-degree (III) hearing loss, 12 (10.4%) in the primary cohort demonstrated auditory responses to whispered speech presented at the concha (near the ear), compared to 3 children (10.0%) in the comparison group. In cases of fourth-degree (IV) hearing loss, 7 children (6.1%) from the primary cohort and 5 children (16.7%) from the comparison cohort exhibited no response to whispered speech stimuli.

Conversational speech evaluation also demonstrated high clinical significance in this population. In the primary cohort, 9 children (7.8%) with I-degree hearing loss responded appropriately to questions delivered at distances of up to 6 meters; in the comparison group, 2 children (6.6%) exhibited similar responses. Among children with II-degree SNHL, 21 (18.3%) in the primary cohort responded to conversational stimuli from a distance of up to 3 meters, whereas 5 children (16.7%) in the control cohort demonstrated comparable responses. Responses to questions delivered at a distance of 0.5 meters were observed in 41 children (35.7%) with III-degree SNHL in the primary group and in 5 children (16.7%) in the comparison group. Notably, an equivalent number of patients with IV-degree SNHL—3 children (2.6%) in the primary cohort and 3 children (10.0%) in the comparison cohort—exhibited responses to conversational stimuli.

Early identification of hearing impairment is a pivotal component in implementing effective auditory-verbal rehabilitation strategies for children with SNHL. Conventional audiometric assessment using pure-tone air-conduction audiometry during the first years of life provides limited insight into the functional status of the auditory system, thereby constraining the optimization of hearing aid fitting and auditory-verbal rehabilitation planning.

In contemporary clinical practice, short-latency auditory brainstem response (ABR) recording has emerged as the method of choice for objective evaluation of auditory function in young children.

Figure 6. Normal short-latency auditory evoked potentials



Typically, ABR results are interpreted through the analysis of wave peak registration (Fig. 6). The methodology is based on the recording of electrophysiological potentials elicited in response to a series of acoustic stimuli presented as a rapid packet of six consecutive signals. The intensity of each successive stimulus is incremented by 10 dB. This technique is primarily designed to isolate Wave V of the ABR, and its diagnostic significance is attributed exclusively to the latency of this wave across varying stimulus intensities, thereby enabling the determination of the intensity–latency function.

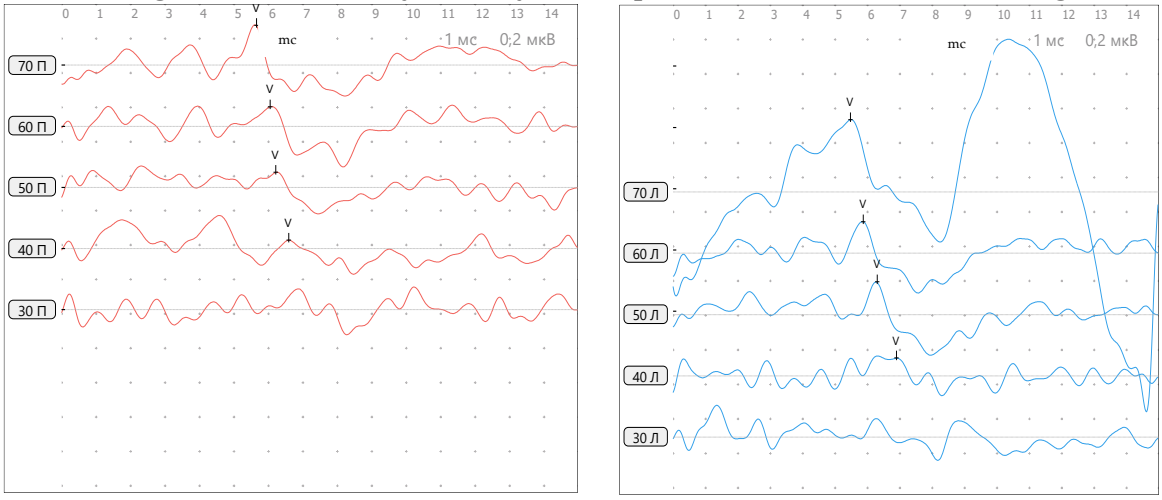
Assessment of auditory function via this approach constitutes a critical step in pediatric evaluation, as it permits individualized identification of the minimal stimulus intensity necessary to evoke Wave V in each child. Subsequent ABR recordings are then performed at intensities no lower than this threshold, optimizing test efficiency while minimizing patient fatigue and distress [10],[11].

To validate these findings, the study proceeded with standard ABR recordings across a stimulus intensity range of 10–70 dB. Data analysis was conducted according to the following parameters:

1. Latencies of ABR wave peaks (I, III, V);
2. Interpeak intervals (IPI) between waves I–III, III–V, and I–V;
3. The intensity–latency function derived from ABR recordings;
4. Interaural differences of the aforementioned measures.

In cases of pathology, the recording of ABR Wave V is noted starting from 30 dB and higher (Fig. 7).

Figure 7. Short-latency auditory evoked potentials in sensorineural hearing loss



ABR amplitude is inherently low even under normative conditions, and in our study cohort, it was not feasible to analyze amplitude measures due to incomplete

myelination in the pediatric participants. To elucidate the relationship between age at diagnosis of chronic SNHL and the severity of bilateral hearing impairment, a detailed analysis was performed across both cohorts.

Analysis of ABR data revealed that, within the primary cohort, first-degree (I) hearing loss on the right ear differed significantly from the comparison group, with mean latencies of 30.67 ± 1.32 ms versus 23.00 ± 2.83 ms, respectively ($p < 0.05$), and on the left ear, 37.22 ± 1.56 ms versus 28.50 ± 3.54 ms ($p < 0.05$). For combined I–II degree hearing loss on the right, mean latencies were 40.00 ± 2.02 ms in the primary cohort versus 39.00 ± 2.50 ms in controls ($p < 0.1$), and on the left, 45.88 ± 1.77 ms versus 39.00 ± 2.50 ms ($p < 0.05$).

Second-degree (II) hearing loss was significantly identified on the right in 47.71 ± 0.71 ms versus 41.25 ± 2.02 ms ($p < 0.05$) and on the left in 52.14 ± 0.67 ms versus 50.00 ± 0.26 ms ($p < 0.05$). For II–III degree hearing loss, right-ear latencies were 59.79 ± 2.69 ms versus 52.50 ± 1.87 ms, and left-ear latencies were 62.50 ± 1.19 ms versus 58.83 ± 0.91 ms, both with $p < 0.05$. Third-degree (III) hearing loss was significantly observed on the right at 62.27 ± 0.63 ms versus 58.25 ± 1.44 ms, and on the left at 66.46 ± 0.72 ms versus 62.75 ± 1.44 ms ($p < 0.05$).

For III–IV degree hearing loss, mean right-ear latencies were 66.67 ± 1.34 ms versus 57.50 ± 3.60 ms, while left-ear latencies were 66.67 ± 1.34 ms versus 80.00 ± 2.07 ms ($p < 0.05$). Fourth-degree (IV) hearing loss was significantly identified on the right at 76.70 ± 1.48 ms versus 70.71 ± 2.48 ms and on the left at 95.20 ± 2.22 ms versus 86.43 ± 2.56 ms in the control group ($p < 0.05$) (Table 4).

Correlation of the obtained results of SHLAEP in children of the main and comparison groups

Table 4.

Degree of hearing loss	I degree		I-II degree		II degree		II-III degree		III degree		III-IV degree		IV degree	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
I group Main n= 115	30,67±1,32	37,22±1,56	40,00±2,02	45,88±1,77	47,71±0,71	52,14±0,67	59,79±2,69	62,50±1,19	62,27±0,63	66,46±0,72	66,67±1,34	74,83±0,91	76,70 ±1,48	95,20±2,22
II group Comparison n= 30	23,00**±2,83	28,50**±3,54	39,00*±1,12	39,00**±2,50	41,25**±2,02	50,00**±0,26	52,50**±1,87	58,83**±0,91	58,25**±1,44	62,75**±1,44	57,50**±3,60	80,00**±2,07	70,71**±2,48	86,43**±2,56

Note: P1, P2 – significance of differences between the main and comparison groups *p < 0.1; **p < 0.05.

According to extant literature, all patients diagnosed with sensorineural hearing loss (SNHL) should undergo tympanometric assessment to exclude concomitant middle ear pathology [12],[13]. Tympanometry results are conventionally represented as graphical plots, in which the presence or absence of peaks, their number, and amplitude provide diagnostic information that allows for an accurate characterization of middle ear status. Based on the Jerger classification, six distinct tympanometric types are recognized: A, B, C, D, E, and Ad.

In our study, Type A tympanograms were consistently observed in all children across both the primary and comparison cohorts, indicating normal middle ear function.

Complementary to tympanometry, otoacoustic emission (OAE) testing is recommended in young children to evaluate the functional integrity of outer hair cells [14],[15]. OAE is categorized into spontaneous and evoked responses. Spontaneous otoacoustic emissions (SOAE) can be detected in the external auditory canal without external acoustic stimulation.

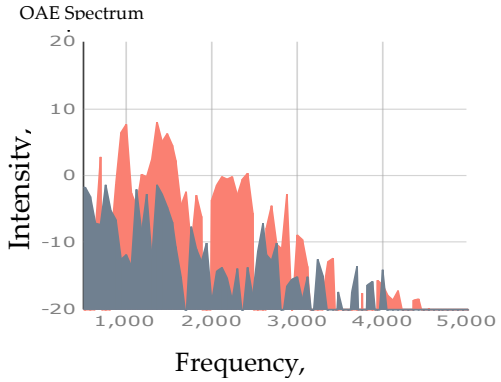
Evoked OAE, in contrast, is elicited in response to controlled sound stimuli and is further subdivided based on the nature of the stimulus:

- Transient-evoked otoacoustic emissions (TEOAE);
- Distortion-product otoacoustic emissions (DPOAE);
- Stimulus-frequency otoacoustic emissions (SFOAE).

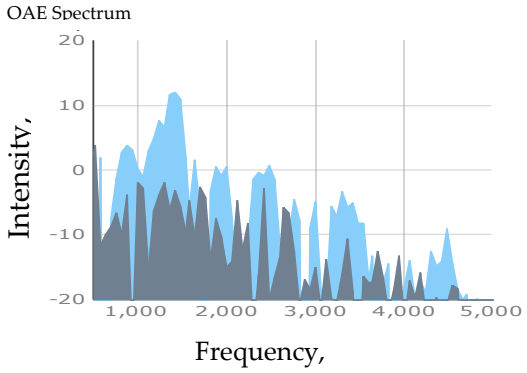
Figures 8 illustrate the results of TEOAE recordings obtained in healthy children, providing reference normative data for comparison with children exhibiting SNHL.

Figure 8. Otoacoustic emissions in normal hearing

Test Result (Right Ear): PASS



Test Result (Left Ear): PASS



OAE Analysis - Right Ear

Stimulus, dB	74.8
Stimulus stability, %	99.3
A&B mean, dB	16.3
A-B mean, dB	11.67
Response, dB	15.9
Reproducibility, %	84.0
Noise level, dB SPL	40.0

OAE Analysis - Left Ear

Stimulus, dB	75.4
Stimulus stability, %	99.9
A&B mean, dB	18.8
A-B mean, dB	13.65
Response, dB	18.5
Reproducibility, %	86.1
Noise level, dB SPL	40.0

OAE Frequencies (Right Ear)

Freq., (kHz)	1	2	3	4	5
Reprod., %	84.8	90.2	80.2	49.7	29.2
Signal, dB	13	9.7	2.7	-7.8	-18
Noise, dB	5.6	0.1	-3.3	-7.8	-14
SNR, dB	**7.5**	**9.7**	**6.0**	**0**	**3.9**

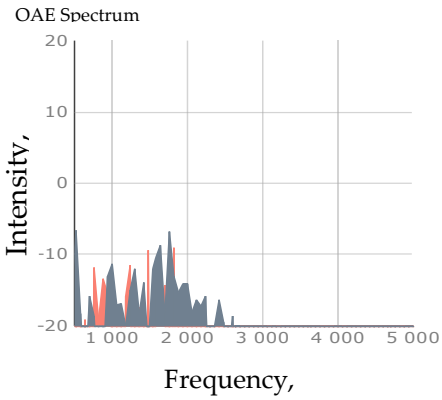
OAE Frequencies (Left Ear)

Freq., (kHz)	1	2	3	4	5
Reprod., %	88.9	86.8	81.6	71.1	68.5
Signal, dB	15	12	4.5	-2.8	-9.5
Noise, dB	6.3	3.4	-1.7	-6.1	-13
SNR, dB	**8.7**	**8.2**	**6.3**	**3.3**	**3.1**

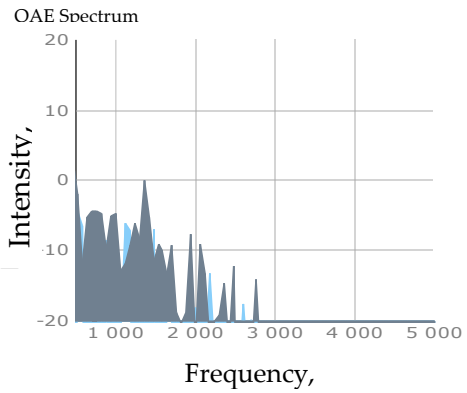
In our study, we used transient evoked otoacoustic emission (TEOAE) to determine the presence of damage to the outer hair cells. The results were as follows: all 115 examined children from the main group and 30 children from the comparison group "failed" the TEOAE test. These data are presented in Figure 9.

Figure 9. Results of the DEOE test

Test Result (Right Ear): FAIL



Test Result (Left Ear): FAIL



OAE Analysis - Right Ear

Stimulus, dB	75,5
Stimulus stability, %	99,2
A&B mean, dB	0,2
A-B mean, dB	4,64
Response, dB	-5,0
Reproducibility, %	17,9
Noise level, dB SPL	40,0

OAE Analysis - Left Ear

Stimulus, dB	75,3
Stimulus stability, %	99,8
A&B mean, dB	5,0
A-B mean, dB	11,91
Response, dB	0,0
Reproducibility, %	-10,5
Noise level, dB SPL	40,0

OAE Frequencies (Right Ear)

Freq., (kHz)	1	2	3	4	5
Reprod., %	31,2	15,2	1,9	-7,4	-0,5
Signal, dB	-7,0	-10	-31	0	0
Noise, dB	-3,5	-2,6	-14	-19	-23
SNR, dB	**-3,5**	**-7,5**	**-17**	**0**	**0**

OAE Frequencies (Left Ear)

Freq., (kHz)	1	2	3	4	5
Reprod., %	-3,7	-7,4	-10,0	7,0	7,3
Signal, dB	0	0	0	-27	-31
Noise, dB	5,4	-0,9	-10	-16	-20
SNR, dB	**0**	**0**	**0**	**-11**	**-11**

4. Conclusion

Test failed on both sides.

The primary criterion for normal otoacoustic emission (OAE) is the signal-to-noise ratio (SNR), which quantifies the difference between the amplitude of the evoked response generated by outer hair cells (signal) and the background cochlear activity (noise). SNR measurement is performed by mathematically decomposing the response into discrete frequency bands; in the case of transient-evoked OAE (TEOAE), five frequency bands are analyzed. In our patient cohort, TEOAE testing yielded negative results, indicating a sensory-level hearing loss exceeding 30 dB HL.

Conclusions:

- In the primary cohort, ABR analysis identified first-degree (I) SNHL in 14.8% of cases and second-degree (II) hearing loss in 30.4%.
- Third-degree (III) SNHL was observed in 46.1% of children, while fourth-degree (IV) SNHL was diagnosed in 8.7% of the primary cohort.
- In the comparison cohort, I-degree SNHL was present in 13.3% of children, II-degree in 33.3%, and III–IV degrees were equally represented in 8 children each

(26.7%).

- Bilateral SNHL of degrees III and IV was significantly more prevalent in both primary and comparison cohorts.
- All patients exhibited bilateral SNHL; no unilateral cases were identified.

Analysis of ABR parameters focused on interpeak intervals (IPI) I–III, III–V, and I–V, peak latencies (LP), and amplitudes of waves I, III, and V. Comparison of ABR outcomes revealed statistically significant differences between children with SNHL associated with Toxoplasma and CMV infections and the control cohort.

The principal ABR alterations observed in the primary cohort involved prolongation of the I–III interpeak interval, while the III–V interval remained within normal limits, suggesting localized slowing of neural conduction between the cochlea and brainstem. Peak I latency remained normative, whereas latencies of peaks III and V were elevated. Overall, the prolonged latency observed at near-threshold stimulus intensities normalized at supra-threshold levels, indicating stimulus-dependent recovery of conduction velocity.

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