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RESEARCH

Hearing screening test in neonates born to COVID-19–positive mothers

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Abstract

SARS-CoV-2, the responsible virus for the COVID-19 pandemic, has demonstrated neurotropic properties indicated by cases presenting with auditory and vestibular system insults. The expression of ACE-2 receptors in the placenta and the detection of IgM antibodies against the virus in the fetuses of pregnant women suffering from COVID-19 render vertical transmission of the infection to the fetus possible. Thus, our study aims to examine whether, similar to other viruses like CMV, SARS-CoV-2 is responsible for congenital hearing loss. This is a retrospective study in a regional pediatric hospital. The medical records of newborns ($n = 111$) born by mothers positive for COVID-19 during pregnancy who underwent screening hearing tests with Transient Evoked Otoacoustic Emissions (TEOAE) and Automatic Auditory Brainstem Response (AABR) from February 2020 to June 2022 were reviewed. Neonates with additional aggravating factors for congenital hearing loss were excluded from the study. For the study period, nine mothers were found positive during the first trimester, twenty mothers in the second trimester, and eighty-three mothers in the third trimester. TEOAEs test and AABR test scored PASS bilaterally in all neonates tested.

Conclusion: Infection with COVID-19 during pregnancy was not a risk factor for hearing loss, similar to other studies.

What is known:

- *The pathogenetic mechanism of the viral-induced impairment of the organ of Corti includes direct damage to the hair cells and indirect damage due to the induction of the innate inflammatory response.*
- *Early data suggested that the SARS-CoV-2 virus also has neurotropic properties with manifestations from the sensory epithelia.*

What is new:

- *Although the intrauterine infection remains controversial, the expression of the ACE-2 receptor on the placenta and the detection of IgM antibodies, as well as the covid-19 genome in fetuses, make the vertical transmission tenable.*
- *In our study, the newborn hearing screening results indicate that COVID-19 infection during pregnancy is not a risk factor for hearing loss.*

Keywords COVID-19 · Congenital hearing loss · Screening audiology tests · TEOAEs

Abbreviations

AABR	Automatic auditory brainstem response
ACE-2	Angiotensin-converting enzyme 2
CMV	Cytomegalovirus
HIV	Human immunodeficiency virus
IgM	Immunoglobulin M
IL-6	Interleukin 6
RT-PCR	Real–Time polymerase chain reaction
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
SSNHL	Sudden sensorineural hearing loss
TEOAE	Transient evoked otoacoustic emissions
Th-17	T-helper 17
TNF-α	Tumor necrosis factor-α
TORCH	Toxoplasma, rubella, cytomegalovirus, herpes
Treg	T regulatory lymphocyte

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Introduction

Congenital hearing loss is one of the most prevalent chronic pathologies that affect 1.33/1000 newborns in developed countries [1]. As it constitutes a highly burdening condition for individual quality of life and medical social services [1], elucidation of the pathogenetic factors and early detection with neonatal hearing screening protocols are essential.

Congenital hearing loss may be attributed to genetic and perinatal environmental factors [1]. Intrauterine infection by specific pathogens like the TORCH (toxoplasma, rubella, cytomegalovirus, herpes, HIV, treponema pallidum, and other agents) is a typical etiologic condition that exerts a detrimental impact on the developing fetus leading, among others, to sensorineural hearing loss [1]. The pathogenetic mechanism of the viral-induced impairment of the organ of Corti includes direct damage to the hair cells and indirect damage to the organ of Corti due to the induction of the innate inflammatory response [2, 3].

Infection by the coronavirus SARS-CoV-2 appeared as a pandemic in 2020, primarily with lower airway and lung symptoms [4]. Early data suggested that the new virus also has neurotropic properties with manifestations from the sensory epithelia, like the olfactory epithelium in 33–68% of the cases [5]. The labyrinth is another sensory organ that can be affected by neurotropic viruses. In line with the neurotropic properties of SARS-CoV-2, there are reports of auditory [6–8] and vestibular pathology [9–11] in adults and children suffering from the COVID-19 infection.

Although the intrauterine infection remains controversial [12], the expression of the ACE-2 receptor on the placenta [13] and the detection of IgM antibodies [13], as well as the COVID-19 genome [13] in fetuses, make the vertical transmission tenable.

In the current retrospective study, based on the database of the audiology department of our tertiary pediatric hospital, we examine the possible impact on neonatal hearing screening tests by maternal infection by SARS-CoV-2 virus during pregnancy, along with a review of the literature.

Materials and methods

This retrospective study was carried out in a regional tertiary pediatric hospital after the approval of the Institutional Review Board (IRB) of Karamandanio Pediatric Hospital, Patras, Greece.

We reviewed the results of the hearing screening test of 111 neonates born by mothers positive for COVID-19 during pregnancy for the period February 2020 to June 2022. The age of our subjects ranged from 0.5 to 5.5 months, with a median age of 2 months. Neonates were grouped according to the gestation trimester when their mothers were diagnosed with the COVID-19 infection. For the study period, nine mothers were found positive during the first trimester, twenty mothers in the second trimester, and eighty-three mothers in the third trimester. Thus, for the study period in our center, 75.7% were cases of COVID-19 infection in the third trimester of pregnancy and only 7.2% in the first trimester, similar to other studies referring to COVID-19 infection [14–16], but also similar to data referring to TORCH infections [17].

Neonates with additional aggravating perinatal factors, like mothers positive for TORCH infection during pregnancy, NICU admission for more than 5 days, intravascular ototoxic antibiotics, lower birth weight than 1500 g, and prematurity of fewer than 33 weeks gestational age were excluded from the study. Additionally, none of our subjects were born to mothers who had taken antiviral agents due to the severity of manifestations of their COVID-19 infection.

The audiological evaluation was performed with a combined transient evoked otoacoustic emission (TEOAEs) and automated auditory brainstem response (AABR) test. We preferred to use AABR rather than solely TEOEAs to avoid misdiagnosing auditory neuropathy as one of the neuropathic effects of the virus. In case of a *Refer* result in one test, we repeated only the failed test within a month.

Results

In our sample, we only detected one case (0.01%) with failed screening hearing tests, probably due to technical reasons like concurrent otitis media with effusion. The tests were repeated 1 month later, concluding with a *Pass* result.

Based on the literature reports and the absence of other aggravating factors for auditory impairment, we did not schedule any follow-up, except for the cases where parents express any concerns for their child's hearing, depending on the acquisition of auditory and language developmental milestones.

Discussion

Intrauterine viral infections are established etiologic agents for congenital sensorineural hearing loss, with CMV infection being the most prevalent case in western countries [18]. SARS-CoV-2, the etiologic virus for the COVID-19 disease that outbroke as an epidemic in 2020, appears with characteristic neurotropic properties making

the study of its association with the organ of Corti insult warranted. Even if it remains controversial, accumulating literature attributes sudden Sensorineural hearing Loss (SSNHL) cases in adults to SARS-CoV-2 [6–8]. In line with that, a possible intrauterine infection with SARS-CoV-2 could also account for congenital hearing loss.

An initial concern is whether the SARS-CoV-2 viremia of an infected pregnant woman can reach the fetus via the placenta. SARS-CoV-2 infects human cells through the angiotensin-converting enzyme 2 (ACE2), activating transmembrane serine protease 2 (TMPRSS2), hence enhancing viral uptake [19]. Since the placenta expresses the ACE-2 receptor, it seems possible that covid-19 can be transmitted to the fetus [20]. Moreover, the SARS-CoV-2 virus has been detected with the reverse transcriptase–polymerase chain reaction (RT-PCR) technique in the placenta, amnion, and blood of newborns born to mothers infected with the SARS-CoV-2 virus [21]. This vertical transmission was also confirmed with newborns positive for IgM antibodies against the SARS-CoV-2 virus, which, unlike IgG, cannot cross the placenta and transmit passively from maternal blood to the fetus [13]. However, despite the possible intrauterine transmission from a pathophysiological standpoint, actual in utero infections appear to be rare, estimated at 2% [22], rendering the vast vertical virus transmission still controversial in the literature.

Data deriving from pathophysiology studies on adult cases of SSHL in the context of COVID-19 infection indicates that although neurologic impairment can be the consequence of hypoxia, hypotension, and liver and renal insufficiency that is characteristic of the most severe cases of COVID-19 infection, several specific mechanisms are considered the leading causes of central and peripheral nervous system damage [23]. It has been supposed that SARS-CoV-2 may infect olfactory neurons and subsequently invade the brainstem, basal ganglia, and cortex [24]. Viruses can also damage the organ of Corti directly as the ACE-2 receptor is expressed in the stria vascularis and spiral ganglion in mice [25] or through the inflammatory response of the host immune system [26]. Host response to viral infection induces the production of proinflammatory cytokines, like IL-1β, IL-6, and TNFα [27]. This cytokine storm can subsequently activate the complement subunits C3a and C5a resulting in decreased differentiation of T regulatory cells (Treg) and increased differentiation of T helper 17 (Th17) that ultimately conclude in an uncontrolled inflammatory response that can have a detrimental impact on central and peripheral neural tissues and sensory organs [28].

Accordingly, in the developing auditory neural pathway of the fetus, even in the absence of fetal viral infection or severe maternal symptoms, a placental infection can trigger a fetal inflammatory response, leading to multiorgan system damage and a predisposition for negative developmental consequences [29]. Moreover, the COVID-19 infection can cause placental dysfunction, resulting in intrauterine hypoxia [30]. In a mouse model, endothelial and thrombotic alterations in the placenta were associated with the altered vascular flow to the fetus and subsequent neural inflammation [31]. Perinatal hypoxia is one established cause of newborns' transitory or permanent hearing loss [32].

An additional concern is whether the timing of maternal infection can have a possible role in the auditory system insult by SARS-CoV-2. The inner ear development begins early in embryogenesis. By the end of the eighth gestational week, the labyrinth has formed its characteristic coiled shape. By the end of the seventh month of fetal age, the labyrinth reaches its mature anatomic configuration and its functional ability [33]. As organogenesis occurs in the first trimester, an infectious agent insult during this period can be way more detrimental. For instance, the risk for complications from congenital cytomegalovirus (CMV) is higher in the first trimester [26]. Even fever during the early stages of pregnancy can harm neuroblast development [34]. Thus, the timing of the viral infection is a significant factor as the placental transmission of some infections is more likely in particular trimesters, and the trimester when exposure occurs will alter the risk of developing an anomaly. Nevertheless, our study did not detect any enhanced vulnerability to hearing loss with fetal exposure to COVID-19 during different trimesters of pregnancy.

Following our sample, most relevant studies include only a minority of women infected during the first trimester of pregnancy [14–16]. Since it is not warranted to assume that women showed various levels of precaution for COVID-19 infection during the different trimesters, and since data exist that do not support any enhanced incidence of miscarriages in COVID-19–infected women early in pregnancy [35], the difference in the incidences of symptomatic infection could be possibly attributed to an enhanced vulnerability, given the suppression of the immune system, expressed by the reduced number of T-cells, the decreased cell-mediated cytotoxicity, and the overall diminished lymphocyte proliferative response that occurs as pregnancy progresses [36].

Apart from in utero exposure, a neonatal infection may occur during the labor (intrapartum) or even in the immediate newborn period (postpartum) from the infected mother, through close contact or breastfeeding, and from asymptomatic hospital staff. Nonetheless, even postpartum infection can result in hearing loss, as is evidenced in several studies on adult cases of sudden sensorineural hearing loss due to COVID-19 infection [6–8].

Similar to our findings, most studies published during the COVID-19 pandemic do not support strong evidence of auditory system insult due to intrauterine infection by the SARS-CoV-2 virus mirrored on normal newborn hearing

screening tests [14, 37–41]. However, other studies are showing a possible negative impact on hearing of newborns of mothers infected by COVID-19 during pregnancy [15, 16]. Interestingly, one study shows suppressed amplitudes of high-frequency otoacoustic emissions, suggesting an insufficiency in the medial olivocochlear efferent system in infants exposed to SARS-CoV-2 intrauterine [16].

Screening audiology tests, TEOAEs, and AABR, are electroacoustic and electrophysiologic exams, respectively, that provide indications for the normal function of the organ of Corti and the generation and transmission of a neural sign along the brainstem. Although there is no strong evidence that the organ of Corti can be affected by COVID-19, the neurotropic properties of the new virus reaching CNS hematogenous or via retrograde transmission through the olfactory epithelium [23] and the detection of ACE-2 receptors on glial cells, neurons, and brain vasculature, cannot exclude a long-term sequelae of the virus to the cerebral centers of hearing in the developing fetal temporal lobe that could manifest as central auditory processing disorders.

One limitation of our retrospective study was that we did not group our sample according to the severity of the mother's symptoms. There is evidence that the severity of maternal COVID-19 infection correlates with an infant's possibility of being infected [22]. However, even asymptomatic infection in pregnant women may lead to the virus's intrauterine transmission [42]. An additional limitation is that, in our cases, we did not consider the different variants of the SARS-CoV-2 virus.

Conclusion

In our study, the newborn hearing screening results indicate that COVID-19 infection during pregnancy is not a risk factor for hearing loss. However, large-scale, multicenter studies of pregnant women are needed to support our results and to make a definite judgment regarding neonatal outcomes.

Authors' Contributions Goulioumis Anastasios and Asimakopoulos Athanasios had the idea for the article. Goulioumis Anastasios, Angelopoulou Maria and Tsiakou Magdalini performed the literature search and data analysis. Goulioumis Anastasios and Angelopoulou Maria drafted the work and Goulioumis Anastasios and Kourelis Konstantinos and Mourtzouchos Konstantinos revised the work.

Data Availability The data presented in this study are available on request from the corresponding author.

Declarations

Ethics approval This is an observational study. The Institutional Review Board (IRB) of Karamandanio pediatric hospital, Patras, Greece has confirmed that no ethical approval is required.

Conflict of interest The authors declare no competing interests.

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