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Correlation between Prenatal, Perinatal, and Postnatal Factors with Congenital Hearing Loss

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Abstract: ***Congenital hearing loss can be caused by prenatal, perinatal, and postnatal factors and is difficult to detect. All infants should be screened for hearing loss and intervention should be given as soon as possible to prevent speech and behavior disorders. The study was aimed to examine the correlation between prenatal, perinatal, and postnatal factors with congenital hearing loss in infants. This was an observational analytic research using secondary data from the medical record of infants aged 0-12 months who were screened with otoacoustic emission (OAE) and automated auditory brainstem response (AABR) in Audiology Clinic of Dr. Soetomo General Hospital in 2014-2018. Total of 439 infants met the inclusion criteria. Most of them were ≤ 1 months old (46.2%) and male (53.3%). The largest proportion of prenatal, perinatal, and postnatal factors in this study respectively were Toxoplasma, other agents, Rubella, Cytomegalovirus, Herpes simplex (TORCH) infection (23.2%), low birth weight (67.7%), and febrile convulsion (5.5%). Congenital hearing loss was found in 151 of 439 patients (34.4%). Chi-square test showed a difference between the results of OAE and AABR (p = 0.000). Coefficient contingency test showed a correlation between asphyxia and congenital hearing loss (p = 0.002). Asphyxia as perinatal factor was correlated with congenital hearing loss, whereas prenatal and postnatal factors were not correlated. Using OAE complemented with AABR is recommended in screening for congenital hearing loss.***

Keywords: congenital hearing loss, prenatal factors, perinatal factors, postnatal factors, otoacoustic emission

1. INTRODUCTION

Hearing loss is one of congenital abnormalities which can be caused by prenatal, perinatal, and postnatal factors. According to the Joint Committee on Infant Hearing (JCIH), the risk factors which influence the incidence of congenital hearing loss are mother-to-fetal infection,

family history of hearing loss, gestational diabetes, low birth weight, preterm birth, hyperbilirubinemia, birth asphyxia, mechanical ventilation for 5 days or more, and so on [1].

The initial symptoms of congenital hearing loss are difficult to detect. JCIH recommends all infants should be screened for hearing loss before 1 month of age, repeat tests can be done before 3 months of age, and interventions can be given before 6 months of age so that speech, motor, cognitive, and social interaction disorders can be minimized. The gold standard for hearing loss screening are otoacoustic emission (OAE) and automated auditory brainstem response (AABR), and if necessary, diagnostic tests can be done with brainstem evoked response audiometry (BERA) [1]. OAE aims to determine the function of outer hair cells in the cochlea while AABR is a modification of BERA that used to assess the function of auditory nerve at a frequency of more than 1,000 Hz and stimulation to each ear with a maximum intensity of 40 dB (corresponding to the infant's hearing threshold) [2].

World Health Organization (WHO) states that in Southeast Asia there are 38,000 children born with hearing loss each year. In Indonesia, the prevalence of hearing loss is 4.2% and there is 0.1% of congenital deafness for each live birth rate. Research in Surabaya found 68% of infants and young children were detected with sensorineural hearing loss but the risk factors were mostly unknown (82.23%) [3]. This study was aimed to examine the correlation between prenatal, perinatal, and postnatal factors with congenital hearing loss in infants in Audiology Clinic of Dr. Soetomo General Hospital in 2014-2018.

2. MATERIAL AND METHODS

This was an observational analytic research using secondary data from the medical record in Audiology Clinic of Dr. Soetomo General Hospital Surabaya, East Java, Indonesia. This study was approved by the Hospital Ethics Committee (No.1347/KEPK/VII/2019). Samples were taken by total population sampling. The inclusion criteria were patients aged 0-12 months who were screened with OAE and AABR on January 2014 until December 2018. The exclusion criteria were patients aged > 12 months and incomplete medical record data.

The data compiled included the name, age, gender, date of birth, prenatal factors, perinatal factors, postnatal factors, OAE and AABR examination results and interpretation. The operational definition of congenital hearing loss is a decrease in hearing acuity in one or both ears showed by the OAE result confirmed by AABR result is refer. The data was analysed using SPSS Statistics for Windows version 22.0 (Armonk, NY: IBM Corp). The Chi-square test was used to compare the results of OAE and AABR examination and the coefficient contingency test was conducted to correlate prenatal, perinatal, and postnatal factors with congenital hearing loss. $P < 0.05$ was considered as statistically significant.

3. RESULTS

Table 1. Distribution of age

Age (month)	**Total (%)**
≤ 1	203 (46.2)
> 1-3	126 (28.7)

> 3-6	69 (15.7)
> 6	41 (9.3)
Total	439 (100)

There were 439 patients who met the inclusion criteria. Most patients were in the age group of ≤ 1 month with 203 patients (46.2%) where the mean age was 2.46±2.36 months (Table 1). The patients consisted of 234 males (53.3%) and 205 females (46.7%) in a ratio of 1.14:1.

Table 2. Distribution of risk factors

Risk factors	**Total (%)**
Prenatal factors	
Family history of hearing loss	8 (1.8)
TORCH infection	102 (23.2)
Preeclampsia	67 (15.3)
Gestational diabetes	17 (3.9)
Perinatal factors	
Prematurity	279 (63.6)
Low birth weight	297 (67.7)
Asphyxia	82 (18.7)
Hyperbilirubinemia	162 (36.9)
Postnatal factors	
Mechanical ventilation	8 (1.8)
Febrile convulsion	24 (5.5)

TORCH: *Toxoplasma*, Other agents, *Rubella*, *Cytomegalovirus*, *Herpes simplex*

The most common prenatal, perinatal, and postnatal factors in this study respectively were TORCH infection (23.2%), low birth weight (67.7%), and febrile convulsion (5.5%). Details of the results are as described in Table 2.

Table 3. Comparison between the results of OAE and AABR examination

OAE	**AABR**		**Total**	**p-value**
	Pass	**Refer**		
Pass	220	44	264	0.000
Refer	68	107	175	
Total	288	151	439	

OAE: otoacoustic emission. AABR: automated audiometry brainstem response

Screening with OAE obtained 264 patients (60.1%) with pass results while with AABR obtained 288 patients (65.6%) with pass results. The Chi-square test showed there was a significant difference between the screening results of OAE and AABR (p = 0.000) (Table 3). Among 439 patients, congenital hearing loss was found in 151 patients (34.4%), where 85 patients (19%) were bilateral, 66 patients (15%) were unilateral, and 63 patients (14%) had auditory neuropathy.

Table 4. Correlation between prenatal factors and congenital hearing loss

Prenatal factors	**Congenital hearing loss**		**p-value**
	Yes (%)	**No (%)**	
Family history of hearing loss			
Yes (%)	2 (1.3)	6 (2.1)	0.720
No (%)	149 (98.7)	282 (97.9)	
TORCH infection			
Yes (%)	38 (25.2)	64 (22.2)	0.565
No (%)	133 (74.8)	224 (77.8)	
Preeclampsia			
Yes (%)	21 (13.9)	46 (16.0)	0.666
No (%)	130 (86.1)	242 (84.0)	
Gestational diabetes			
Yes (%)	6 (4.0)	11 (3.8)	1.000
No (%)	145 (96.0)	277 (96.2)	

Out of 151 patients with congenital hearing loss, 2 patients (1.3%) had family history of hearing loss, 38 patients (25.2%) had experienced of TORCH infection, 21 patients (13.9%) had history of preeclamptic mothers, and 6 patients (4.0%) had history of gestational diabetic mothers. The coefficient contingency test showed there was no statistically significant correlation between each prenatal factors and congenital hearing loss (p > 0.05) (Table 4).

Table 5. Correlation between perinatal factors and congenital hearing loss

Perinatal factors	**Congenital hearing loss**		**p-value**
	Yes (%)	**No (%)**	
Prematurity			
Yes (%)	97 (64.2)	182 (63.2)	0.911
No (%)	54 (35.8)	106 (36.8)	
Low birth weight			

Yes (%)	102 (67.5)	195 (67.7)	1.000
No (%)	49 (32.5)	93 (32.3)	
Asphyxia			
Yes (%)	41 (14.2)	41 (14.2)	0.002
No (%)	110 (72.8)	247 (85.8)	
Hyperbilirubinemia			
Yes (%)	53 (35.1)	109 (37.8)	0.644
No (%)	98 (94.9)	179 (62.2)	

Among 151 patients with congenital hearing loss, prematurity was found in 97 patients (64.2%), low birth weight was obtained in 102 patients (67.5%), asphyxia was found in 41 patients (14.2%), and hyperbilirubinemia was obtained in 53 patients (35.1%). The coefficient contingency test showed there was a significant correlation between asphyxia and congenital hearing loss (p = 0.002), while other perinatal factors were not correlated ($p > 0.05$) (Table 5).

Table 6. Correlation between postnatal factors and congenital hearing loss

Postnatal factors	**Congenital hearing loss**		**p-value**
	Yes (%)	**No (%)**	
Mechanical ventilation			
Yes (%)	2 (1.3)	6 (2.1)	0.720
No (%)	149 (98.7)	282 (97.9)	
Febrile convulsion			
Yes (%)	11 (7.3)	13 (4.5)	0.321
No (%)	140 (92.7)	275 (95.5)	

Out of 151 patients with congenital hearing loss, 2 patients (1.3%) had history of mechanical ventilation and 11 patients (7.3%) had experienced of febrile convulsion. The coefficient contingency test showed there was no statistically significant correlation between each postnatal factors and congenital hearing loss ($p > 0.05$) (Table 6).

4. DISCUSSION

The largest proportion of age group in this study was ≤ 1 month of age (46.2%). This was in accordance to JCIH recommendation for hearing loss screening before 1 month of age [1]. Auditory development functionally begins at 25 weeks gestation. In the first 6 months of life is a critical period of sensory and neural development of the hearing system so it needs to be stimulated, such as talking to the babies, hearing music or sound from the environment so that babies can develop their speech and learning perceptions [4]. Seeing congenital hearing

loss is quite difficult to detect, early detection and intervention of hearing impairment should be done to all infants so that speech delay can be prevented.

The gender distribution in this study showed there were more male (53.5%) than female (46.7%) with a ratio of 1.14:1. Another study in Istanbul also showed similar results and it was stated there was no correlation between gender and congenital hearing loss [5].

Several studies have shown variations in the distribution of risk factors. The largest proportion of prenatal, perinatal, and postnatal factors in this study respectively were TORCH infection, low birth weight, and febrile convulsion. A study in India showed the most common prenatal factors were gestational diabetes and preeclampsia [6]. Another study in Yogyakarta showed the most common perinatal factor was hyperbilirubinemia, while the largest proportion of postnatal factor was mechanical ventilation [7]. The different proportion of risk factors were according to distribution of characteristics in each study site.

In this study, the refer results in OAE were higher than AABR. However, not all patients with refer OAE results had congenital hearing loss because some patients passed the AABR examination. A study in India stated that OAE is a simple and fast examination tool, but it has poor sensitivity and specificity when used independently. AABR is considered better than OAE because it has a lower failure rate, but it is more time-consuming. Using both OAE and AABR is effective for early screening of all high-risk infants [8]. A study in developing countries showed the difference between OAE and AABR results depended on the time of the screening done. In infants aged ≤ 48 hours, the refer rate of AABR (16.7%) was lower than OAE (37.9%). In infants aged > 48 hours, the failure rate for OAE decreased to 26%, but in AABR there was almost no failure (3%). It showed for infants who would be sent home early should be screened using AABR, while those who would be sent home later could be screened by OAE [9]. Another study in China showed in high-risk neonates, examination by OAE alone had limited diagnostic value, so it was advisable to check with AABR first then followed by OAE [10]. This may explain the difference between hearing tests with OAE and AABR ($p = 0.000$) in this study.

About one third (34.4%) of the total study sample had congenital hearing loss where most of them were bilateral (19%). There were 63 patients (14%) had auditory neuropathy, in which these patients experienced hearing nerve dysfunction so that they only passed the OAE examination. A previous study in Surabaya obtained different results where 68.3% of infants and children were detected congenital hearing loss in 2011-2013 [3]. However, seeing the high percentage showed congenital hearing loss is quite common in infants. This must be a concern because hearing loss has an impact on social relations and education in the future.

In line with previous study in Istanbul [5], there was no association between family history of deafness from birth and congenital hearing loss ($p = 0.720$). Family history of congenital hearing loss was one of the risk factors, but the evidence of its relevance was still low, which was only 1.43% [11]. A study in South Africa found that 19.6% of children with congenital deafness had a family history of hearing loss since birth. Based on WHO data, the prevalence of congenital hearing loss in South Africa is quite high, 19 per 1,000 births [12]. This showed the tendency for countries with high prevalence of hearing loss to have a high family history

of hearing loss so that this risk factor should not be ignored [11]. Research in Australia suggested that children who had family history of hearing loss and passed the hearing screening at birth needed to be supervised throughout childhood because it might be delayed onset [13].

In this study, there was no correlation between TORCH infection and congenital hearing loss ($p = 0.565$). This was different from a research in Yogyakarta which showed an association between TORCH infection and congenital hearing loss detected by OAE and BERA ($p = 0.002$) where children with TORCH infection had 15.63 times risk of having cochlear and auditory nerve damage [14]. TORCH infection in pregnancy can be transmitted to the fetus and interfere organogenesis. This infection can cause central nervous system disorders that have an impact on neurological, hearing, and vision disorders [15]. Another study stated that hearing loss due to TORCH infection is commonly moderate to severe degrees. The manifestation sometimes appeared several months after birth, for example hearing loss in Cytomegalovirus infection can appear at the age of 27-33 months [16]. Therefore, hearing screening should be done periodically so that congenital hearing loss can be detected immediately.

This study showed there was no correlation between preeclampsia and congenital hearing loss ($p = 0.666$). Preeclampsia is a disorder in pregnant women associated with vasospasm, endothelial dysfunction, and ischemia. Preeclampsia can affect the microcirculation in the cochlea associated with local oxygenation disorders which is potentially causing inner ear damage and resulting in sensorineural hearing loss [17]. A study in Iran showed there was a significant correlation between preeclampsia and the results of the first OAE examination ($p = 0.001$), but not significant with the results of the second OAE ($p = 0.646$) and BERA ($p = 0.573$) so it was suspected that preeclampsia might have a temporary effect on congenital hearing loss [18].

In this study, there was no association between gestational diabetes and congenital hearing loss ($p = 1,000$), similar with a study in Brazil that it was not correlated in infants of diabetic gestational mothers without complications [19]. Different result showed in an American research that there was a correlation between gestational diabetes and congenital hearing loss, especially sensorineural and bilateral type [20]. The inner ear has complex metabolic activities which make it sensitive to changes in body homeostasis. Changes in blood sugar and insulin levels in the blood can cause hearing loss and vestibular disorders [21]. Gestational diabetes can cause complications for infants, such as hyperbilirubinemia, low birth weight, and prematurity, where these complications are also risk factors for congenital hearing loss [22].

Contrary to the findings in a Romanian research [23], there was no correlation between prematurity and congenital hearing loss ($p = 0.911$). Premature infants, especially < 33 weeks gestational age, are more at risk of experiencing congenital hearing loss because their auditory nerve development is still incomplete [24]. Cochlea which is immature both anatomically and physiologically will interfere the development of outer hair cells [7]. A study in Poland stated that infants who only had prematurity might not have a severe impact

on hearing because they are often linked to other risk factors, including mechanical ventilation, very low birth weight, and craniofacial anomalies [25].

This study showed there was no correlation between low birth weight babies and congenital hearing loss (p = 1.000). A research in Yogyakarta showed a different result that there was an association between low birth weight and impaired cochlear outer hair cell function [7]. Infants with low birth weight have inadequate intrauterine fetal development. It impact on poor development of the auditory nerve so that it is potentially causing hearing loss. Another study showed the prevalence of hearing screening failure was higher in neonates with low birth weight due to fluid accumulation in the middle ear. This was temporary so that a few weeks after discharge from the hospital will improve. Infants with low birth often had other risk factors that trigger congenital hearing loss, both early and delayed onset [26].

In line with previous study in Netherland [27], there was a correlation between history of asphyxia and congenital hearing loss (p = 0.002). Hearing loss is a secondary impact of hypoxia in the dorsal cochlear nucleus of the brainstem and cochlea [28]. Cochlea can function properly if the oxygenation and perfusion of these organs are adequate. Severe hypoxia can cause irreversible degeneration of outer hair cells and stria vascularis in the cochlea [25]. This statement is supported by a research in Central Java showed infants with history of asphyxia are 21 times more likely to have decreased function of cochlear outer hair cells [29]. Until now, the limit level of hypoxia that causes congenital hearing loss remains unknown [26].

In this study, there was no correlation between hyperbilirubinemia and congenital hearing loss (p = 0.644). A similar result was reached by a study in Yogyakarta that hyperbilirubinemia was not associated with disruption of cochlear outer hair cells [7]. Hyperbilirubinemia causes selective damage to the cochlea and vestibular nucleus in the brainstem, auditory nerve, and spiral ganglion cells, while the organ of Corti and thalamocortical auditory pathway are not affected. The common form of hearing loss caused by hyperbilirubinemia is auditory neuropathy which can be detected by AABR or BERA. This type of hearing loss sometimes resolves spontaneously depending on the duration of hyperbilirubinemia [26]. Another study stated that the association between hyperbilirubinemia and congenital hearing loss was influenced by other risk factors, such as prematurity and low birth weight babies [25].

In line with previous study in Yogyakarta [7], there was no correlation between mechanical ventilation and congenital hearing loss (p = 0.720). Different results showed in a study in Netherland that there was a significant correlation between mechanical ventilation for $\geq$ 5 days and congenital hearing loss [27]. Mechanical ventilators will pump air into the lungs during inspiration and provide positive pressure. The intrathoracic pressure will rise followed by increasing pulmonary blood pressure which in long-term use can cause complications, such as bronchopulmonary dysplasia and neonatal respiratory distress. In addition, these infants will support the loss of oxygen levels which are getting higher and higher so that it can damage the sensory cells of the inner ear [30]. Various aspects have been associated with a greater incidence of hearing loss in the use of mechanical ventilators, including noise level of the equipment, duration of mechanical ventilation, and lung abnormalities involved [31].

This study showed there was no correlation between febrile convulsion and congenital hearing loss (p = 0.321), different from a research in India showed different results that febrile seizures had a significant association with hearing loss in infants admitted to NICU [32]. One of the causes of febrile seizures is viral or bacterial infections in the postnatal period, such as meningitis, meningoencephalitis, and sepsis. Prolonged febrile seizures can cause epilepsy and a risk of nerve damage, especially in the mesial temporal lobe area [33].

However, further studies are needed due to delayed onset of congenital hearing loss which still can be detected in children up to 18 years of age and, if possible, adding the results of the BERA examination as a diagnostic test to determine the degree of hearing loss.

5. CONCLUSION

From this study, it is concluded that asphyxia as perinatal factor was correlated with congenital hearing loss, whereas prenatal and postnatal factors were not correlated. Using OAE complemented with AABR is recommended in screening for congenital hearing loss. Enhancing awareness of the risk factors and easy access to primary healthcare can help to detect congenital hearing loss earlier and reduce the adverse effect.

6. CONFLICT OF INTEREST

There is no conflict of interest in this study.

7. FUNDING

This study is free of supporting charge.

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