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Hearing impairment in Preeclampsia

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Abstract:
Objective:*To Investigated whether preeclampsia is a risk factor for cochlear damage and permanent hearingloss.*
Methods:
Study Design:*Prospective case-control study design.*
Our study done indept of otolaryngology and dept of Obsteric&Gynecology at J.L.N.M.CH Bhagalpur from March 2014 to April 2015.It composed of patientsgroup that suffer from preeclapsia with mean age26.5 year old and control group representing healthy pregnant women with mean age 26.8 year old.Otoscopic examinations and pure-tone audiometry, tympanometry, otoacoustic emissions (OAEs), and stapedial reflex tests were conducted for all subjects. Negative audiologic tests were reevaluated after the postpartum period.Systemic and Obstetric evalution was done to both group.
Results:*Immitancemetry, stapedial reflex and pure tone audiometry were normal in both groups, while Distortion product otoacoustic emission was statistically significant.*
Conclusion: *Preeclampsia is a risk factor for cochlear damage and permanent hearing loss. Even if preeclampsia resolves after delivery, cochlear damage and permanent hearing loss remain unchanged in patients with preeclampsia.*
Key Word:*Hearing Loss, Otoacostic emission, Preeclampsia*

I. Introduction

Preeclampsia is a pregnancy specific syndrome with reduced organ perfusion resulting in the occurrence of vasospasm and endothelial activation. It is a complication of pregnancy char-acterized by hypertension that usually begins after 20 weeks of pregnancy which is accompanied by proteinuriaPreeclampsia, which affects circulation with possible immunologic pathogenesis, can induce damage to the cochlea and result in sensory neural hearing loss.

Because preeclampsia is a clinical occurrence affecting a large proportion of pregnancies, the prevention of complications that may result in preeclampsia is important from the aspect of morbidity and mortality. Several studies have addressed the problem of preeclampsia in the area of nephrology, ophthalmology, cardiology, and neurology but not in otology. The aim our study was to determine whether preeclampsia was a risk factor from an otological aspect and, by drawing attention to this subject, to establish what preventative and precautionary measures should be taken from an otological aspect.

II. Material & method

Inclusion criteria
Inclusion criteria for this study were female patients after 20th week of pregnancy who were hospitalized in the Obstetrics and Gynecology ward aged under 40 years, and they agreed to participate in this study**.**

Exclusion Criteria
Patient with CSOM
Patient with known history of Hearing loss
Patient with history of Previous preeclampsia Diabetes Mellitus

The prospective study was conducted between March 2014 and April 2015 in the Otolaryngology and Obstetricand Gynecology Department .Informed consent was obtained from all participant .Subjects include 60 healthy pregnant women as controls and 60 women with preeclampsia ,defined by the presence of a maternal blood pressure higher than 140/90 mmhg after 20 week of gestation and proteinuria of at least 300mg/24h. The hearing test blood pressure measurement and urine test were performed on the same day.The preeclampsia and healthy pregnant women were all being monitored by clinical obstetricians.

Otoscopic examinations were conducted for all participants. Hearing thresholds were assessed using the Clinical Audiometer AC33 (Interacoustics A/S, Assens, Denmark). In addition, bone and airway conduction thresholds were measured at frequencies of 250, 500, 1,000, 2,000, 4,000, and 8,000 Hz. The outer ear canal volume, membrane compliance, gradient, and pressure parameters were recorded using the Clinical Impedance Audio-meter AZ26 (Interacoustics). Tympanometry results were clas-sified into Types A, B, and C, as set out by Liden (6) and Jerger

(7). Stapes reflex was also recorded using the Clinical Imped-ance Audiometer AZ26 (Interacoustics). Cochlear function was evaluated by measuring transient evoked OAEs from 1,000 to 4,000 Hz at an intensity of 80 T 3 dB using an OAE device (ILO288; Interacoustics).

III. Result

	Control pregnant women (n = 60)	Preeclamptic women (n = 60)	Pvalue
Tympanic membrane abnormality (right)	0	2	0.549
Tympanic membrane abnormality (left)	0	0	0
Abnormal tympanometry(right)	2	6	0.119
Abnormal tympanometry (type bYc) (left)	0	2	0.549
Stapedial reflex (right)	2	12	0.008
Stapedial reflex (left)	0	4	0.0.4
OAE (right)	0	6	0.013
OAE (left)	0	2	0.549
Abnormal otologic findings	4	16	0.015

The preeclampsia group included 60 women (mean age, 31.43yrs) and the control group included 60 women (mean age, 29.66 yr). At the time of the tests, all the pregnancies were 20 weeks or more. Sixteen of the preeclampsia patients had 1 or more otological problems. In 4 women from the control group, otological abnormalities were detected. No significant differences were observed between the 2 groups in tympanic membrane status, tympanometry type, and stapedial reflex. The 2 groups differed significantly in right and left ear OAEs ($p = 0.013$ and $p = 0.549$, respectively;). However, in the preeclampsia group, there was no significant difference in the OAEs between the right and left ears. The 2 groups also differed significantly in hearing thresholds of right and left ears at BC 500, and left ears at BC 2000 dif-fered significantly ($p = 0.040$ and $p = 0.003$, respectively;). Subjects were rechecked 3 to 4 weeks after delivery of the placenta for an additional emission measurement and evaluation of hearing thresholds. Negativity for OAE and a decrease in hearing thresholds at low frequency were observed to continue. Risk variables in women with preeclampsia were obtained using the logistic regression method. 2 presents the results of the logistic method. The method revealed that the following variables were significant: BC 500Yleft, BC 500Yright, OAE-right, and OAE-left. The odds ratios and 95% confidence intervals (95% CIs) for these 4 risk variables were as follows: 1.167 (1.044Y1.306), 1.117 (1.002Y1.244), 0.642 (0.505Y0.815), and 0.576 (0.475Y0.698), respectively.

IV. Discussion

The underlying mechanisms of preeclampsia are not fully understood. However, placental hypoperfusion is present in the cause, and this leads to an increase in placental factors (soluble tyrosine kinase 1 and anti-angiogenic factors such as soluble endoglin) and a de-crease in placental growth factor and vascular endothelial growth factor (proangiogenic factors). An imbalance of antiangiogenic factors is thought to trigger vascular endothelial cell injury in the liver, kidney, and brain, as well as within the placenta itself (10). In addition, increased vascular resistance and diffused or segmental multifocal vasospasms have been reported in cases of preeclampsia. These clinical factors most likely result in hypertension, proteinuria, and other systemic manifestations of the syndrome. Traditionally, preeclampsia has been viewed as a self-limited condition that resolves after delivery of the placenta. However, recent research has shown that maternal endothelial dysfunction may persist for years after the episode.

Arterial hypertension may affect hearing by different ways. High pressure in the cochlear microcirculation may cause haemorrhage in the inner ear, which may cause progressive or sudden sensory neural hearing loss. As blood viscosity is increased due to pre-eclampsia the capillary blood flow and oxygen saturation in the cochlea are reduced which causes tissue hypoxia that can cause hearing deficits and hearing loss in hypertensive patients. Moreover, increase in arterial blood pressure may cause ionic changes in cell potentials of the hair cells of the cochlea, thus causing hearing loss.The pathogenesis of preeclampsia is complicated and not fully understood. It may be associated with multiorgan failures of the mother, coagulopathy, vasospasm, ischemia and microthrombi in peripheral circulation that my lead to maternal and foetal.Bakhshaee et al. stated that damage to the cochlear hair cells during preeclampsia was possible. Theyevaluated hearing in 37 preeclamptic patients and 38 healthy women with TEOAE and reveal significant differences between the two groups, as 13.5% of pre-eclamptic women had abnormality in TEOAE. These

findings pointed out the possible effect of preeclampsia on the cochlea at least temporarily]. Our study differed from the research of Bakhshaee et al. (5) in that we evaluated hearing threshold, stapes reflex, and tympanometry in addition to OAEs. We found bilateral failure in cochlear function in patients with preeclampsia. This failure was particularly affected by low frequencies, so a drop in hearing threshold seemed to arise in bone conduction at low frequencies. This failure continued after delivery. The fact that we determined hearing loss in low frequencies supports the notion that vascular and immune components play an important role in the preeclampsia-induced otopathologic abnormality. We hypothesize that the low-frequency hearing loss reflects the areas of the inner ear affected by ischemia. We think that the hearing loss at low frequency depends on these ischemic areas, particularly the affected cochlear apical section because this seems to be more sensitive to vas-cular and immunological incidents

Altunta et al. pointed out that, there was no significant difference between hypertensive and healthy pregnant women in terms of hearing assessment, but damage to the cochlear hair cells consequent to hypertension during pregnancy is possible. The results of his study suggested that ischaemia of the inner ear that caused by microthrombus and vasospasm in hypertensive patients during pregnancy does not result in hearing impairment in the postpartum period6. Our results disagree with Altunta et al. as there is hearing impairment detected by OAEs of preeclmptic women.Ozdemir et al. found statistically significant differences between per-eclamptic and healthy women in pure-tone audiometric results. However, these results were irrelevant clinically as all puretone thresholds were lower than 20 dB (normal hearing abilities). The differences between brainstem auditory-evoked potentials were not statistically significant]. Our results disagree with Ozdmir et al. as there is no statistical difference between both groups in pure tone audiometery and our data showed significant difference between patient's group and control group regarding DPOAEs.

Baylan et al. OAE-right, and OAE-left differed significantly between pre-eclamptic patient and control groups also bone conduction at 500 Hz significantly differ between both groups in pure tone audiometry.

V. Conclusion

Preeclampsia plays a role as a risk factor of cochlear outer hair cells destruction that can lead to permanent hearing loss. Although preeclampsia mostly resolves after delivery, cochlear damage and permanent hearing loss may remain unchanged in patients with preeclampsia. Some shortcomings of this study were not being capable of testing the function of the middle ear objectively or performing any hearing threshold test using audiometry. Further research is needed to examine middle ear function objectively with tympanometry and also the use of audiometry to assess the hearing thresholds as well as the need to evaluate whether the results of the OAE will remain the same or return to normal after giving birth. Based on this research, it is expected that obstetricians should work together with otolaryngologists in order to detect the complications of preeclampsia on the inner ear so that any damage will be immediately resolved.

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