# Hearing Impairment in Pre-eclampsia 

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#### Abstract

Introduction: Pre-eclampsia is marked by increase in blood pressure and a raised level of protein in the urine and women will often also have swelling in the feet, legs, and hands. This condition usually appears during the second half of pregnancy; the pathological changes that happen during pre-eclampsia will be consequence of vasoconstriction of blood vessels, endothelial malfunction and ischemia. Hypertension and hemo-vascular disorder that accompany the disease can influence any organ including inner ear. The aim of the present study is to study the effect of pre-eclampsia on the inner ear by using Distortion product otoacoustic emission (DPOAEs).

Patients and Methods: Our study is a prospective one that done at Otorhinolaryngology, Obstetric and Audiology clinics at Minia University Hospital, from March 2011 to December 2012. It composed of patient's group that suffer from pre-eclampsia with mean age 25.3 years old and control group representing healthy pregnant women with mean age 25.4 years old. The entire study sample was subjected to audiological evaluation (Distortion product otoacoustic emission, Immitancemetry, Stapedial reflex and pure tone audiometry). Systemic and Obstetric evaluation was done to both groups.


Results: Immitancemetry, stapedial reflex and
pure tone audiometry were normal in both groups, while Distortion product otoacoustic emission was statistically significant lower amplitudes in nearly one third of the patient's groups at all F2 frequencies except at 6250 Hz , while none of the ears of the control group had abnormal DPOAEs.

Conclusion: Pre-eclampsia can influence cochlear function and be a risk factor for sensory neural hearing loss in affected women.

Key words: Pre-eclampsia, Hearing, Cochlea

## Introduction:

Preeclampsia is defined as increase in blood pressure and proteinuria after 20 weeks of pregnancy in a woman who not suffer previously from hypertension, if left untreated; preeclampsia can lead to serious and fatal complications for both the mother and her baby ${ }^{1}$. Preeclampsia is a serious condition that puts pregnant women at risk for failure of multiple organs including inner ear, due to vascular events and systemic toxemia ${ }^{2}$.

Preeclampsia is a common disorder that is a consequence of vasospasm, endothelial dysfunction and ischaemia ${ }^{3}$, Preeclampsia, which affects circulation with possible immunologic pathogenesis, can induce damage to the cochlea and result in sensory neural hearing loss ${ }^{4}$. The aim of our study is
to evaluate cochlear changes by using Otoacoustic emission in pre-eclamptic women.
Subjects and Methods:
The study was approved by The Research Ethics Committee of the faculty of Medicine, Minia University. A written consent was signed by all women that enrolled in the study. The current study is a prospective study that done on 16 female patients, that suffer from pre-eclampsia (the study group); and 18 healthy pregnant mothers represent the control group. The mean age of study group was 25.3 years and that of control group was 25.4 years old.

The study was done at Otorhinolaryngology, Obstetric and Audiology clinics at Minia University Hospital, from March 2011 to December 2013.

All patients and control groups subject to:

1. Detailed medical history
2. Obstetric examination
3. Laboratory investigation in the form of (complete blood count, blood glucose, renal function tests, liver function tests and urine analysis)
4. Full Otorhinolaryngological examination
5. Medical examination including measurement of blood pressure.
The patient is diagnosed as having pre-eclampsia if she passed 20 weeks gestation; blood pressure is $140 / 90 \mathrm{mmHg}$ or higher-documented on two measures, at least six hours apart but no more than seven days apart and there is protein in urine after urine analysis.
Exclusion criteria:
$\alpha$. Patients with chronic suppurative otitis media (unilateral or bilateral)
$\beta$. Patients with previous ear surgery
$\chi$. Patients with known history of hearing loss
$\delta$. Patients with otosclerosis
$\varepsilon$. Patients with previous medical diseases like Diabetes, Hypertension, autoimmune diseases.
Audiological evaluation:
6. Immitancemetry using Zodiac 401 middel ear analyser to measure middle ear preassure and stapedial muscle reflex at frequencies 500, 1000,2000 , and 4000 Hz .
7. Pure tone audiometry using Amplaid_309 audiometer for assessment of hearing sensitivity. Air conduction (AC) threshold was obtained for the frequency range 250-8000 Hz at single octave intervals through using TDH 49 ear phone, while bone conduction (BC) threshold was obtained for the frequency range $500-4000 \mathrm{~Hz}$ at single octave intervals using B71 bone vibrator.
8. Distortion product otoacoustic emission (DPOAE) to test the cochlear function. DPOAE was recorded using the Intelligent Hearing ${ }^{\circledR}$ system with Smart OAE 4.5 software. Two tones were used; $\mathrm{L} 1=65 \mathrm{~dB}$ SPL and $\mathrm{L} 2=55 \mathrm{~dB}$ SPL, while f2/f1 was 1.22. Both the amplitude of response of the distortion product (DP) at 2 f1-f2 and background noise ( Ns ) were obtained at 9 points corresponding to $f 2$ frequencies of 553, 783, 1105, 1560, 2211, 3125, 4416, 6250 and 8837 Hz . These measurements were used to build DP-gram by displaying the DP against the F2 frequency. The SNR was measured (SNR=DP-Ns) at each of these 9 points. DPOAE was considered normal and thereby reflecting normal cochlear function if the SNR was equal to or greater than 3 dB SPL on at least 70\% of the tested frequencies.

Our data was collected and analyzed statistically by using student t-tests
Results:
Our results revealed statistically significant lower DPOAE amplitudes in the study groups at all F2 frequencies except at 6250 Hz , while none of the ears of the control groups had abnormal DPOAEs (Tables 1 and 2). Show Mean and SD of DPOAE amplitudes in dB SPL of the right and left ear-consequently for both study and control groups; in addition to $t$ and $p$ values of the student $t$ test.

The results of the present study revealed that $36.4 \%$ of patients of the study group had abnormal DPOAEs (i.e., the DPOAEs was greater than 3 dB SPL in less than $70 \%$ of the tested frequencies)

Our results pointed out that there is no statistical difference between patient and control groups in Immitancemetry and stapedial muscle reflex, as they were normal in both groups. Our results showed also, that pure tone audiometry at examined frequencies were normal in both groups

## Discussion:

Otoacoustic emissions especially the distortion

| F2 Frequency (kHz) | Group | Mean | SD | T value | $P$ value |
| :---: | :---: | :---: | :---: | :---: | :---: |
| 1105 | Study Group Control Group | $\begin{aligned} & 7.89 \\ & 14.2 \end{aligned}$ | $\begin{aligned} & 8.51 \\ & 6.87 \end{aligned}$ | 3.6 | 0.001 |
| 1560 | Study Group Control Group | $\begin{gathered} 10.00 \\ 23.4 \end{gathered}$ | $\begin{gathered} 11.66 \\ 4.88 \end{gathered}$ | 5.6 | 0.000 |
| 2211 | Study Group Control Group | $\begin{gathered} 17.14 \\ 28.0 \end{gathered}$ | $\begin{aligned} & 5.94 \\ & 5.02 \end{aligned}$ | 6.3 | 0.000 |
| 3125 | Study Group Control Group | $\begin{gathered} 16.29 \\ 25.9 \end{gathered}$ | $\begin{gathered} 7.78 \\ 8.0 \\ \hline \end{gathered}$ | 3.6 | 0.001 |
| 4416 | Study Group Control Group | $\begin{gathered} 10.00 \\ 17.8 \end{gathered}$ | $\begin{gathered} 5.10 \\ 9.0 \end{gathered}$ | 4.3 | 0.000 |
| 6250 | Study Group Control Group | $\begin{gathered} 9.27 \\ 9.5 \end{gathered}$ | $\begin{aligned} & 7.01 \\ & 3.93 \end{aligned}$ | 0.19 | 0.84 |

Table 1: Mean and SD of DPOAE amplitudes in dB SPL of the right ear for both study and control groups; in addition to the $t$ and $p$ values of the student $t$ test.

| F2 Frequency (kHz) | Group | Mean | SD | T value | P value |
| :---: | :---: | :---: | :---: | :---: | :---: |
| 1105 | Study Group Control Group | $\begin{gathered} 7.75 \\ 15.75 \end{gathered}$ | $\begin{aligned} & 8.55 \\ & 4.15 \end{aligned}$ | 3.56 | 0.001 |
| 1560 | Study Group Control Group | $\begin{aligned} & 13.25 \\ & 20.44 \end{aligned}$ | $\begin{gathered} 11.38 \\ 5.36 \end{gathered}$ | 5.62 | 0.000 |
| 2211 | Study Group Control Group | $\begin{gathered} 15.13 \\ 24.9 \end{gathered}$ | $\begin{gathered} 9.2 \\ 3.81 \end{gathered}$ | 6.42 | 0.000 |
| 3125 | Study Group Control Group | $\begin{gathered} 15.63 \\ 22.9 \end{gathered}$ | $\begin{aligned} & 7.66 \\ & 6.98 \end{aligned}$ | 3.5 | 0.001 |
| 4416 | Study Group Control Group | $\begin{aligned} & 7.65 \\ & 19.7 \end{aligned}$ | $\begin{aligned} & 6.78 \\ & 7.45 \end{aligned}$ | 4.33 | 0.000 |
| 6250 | Study Group Control Group | $\begin{aligned} & 9.5 \\ & 8.3 \end{aligned}$ | $\begin{gathered} 5.7 \\ 4.56 \end{gathered}$ | . 18 | 0.81 |

Table 2: Shows mean and SD of DPOAE amplitudes in dB SPL of the left ear for both study and control groups; in addition to the $t$ and $p$ values of the student $t$ test.
product type (Distortion product otoacoustic emissions; DPOAEs) is a sensitive test for cochlear function. It has excellent test retest reliability and it can detect sub-clinical cochlear abnormality before these abnormalities manifest clinically in the conventional audiogram. Therefore it has been used to monitor cochlear function in patients taking ototoxic medications as gentamycin and subject under noise exposure. Compared to the transient otoacoustic emissions (TOAEs), DPOAEs is more frequency specific and test wider frequency range than TEOAEs ${ }^{5}$.

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 ${ }^{6-9}$.

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 ${ }^{4,10}$.

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 ${ }^{4}$. Our results showed that $36.4 \%$ of the ears of pre-eclamptic women had abnormal DPOAEs
which agree with Bakhshaee et al. and as we use of DPOAEs which is more sensitive than TEOAE, explain the higher results that we have.

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 period ${ }^{6}$. Our results disagree with Altunta et al. as there is hearing impairment detected by DPOAEs in $36.4 \%$ 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 ${ }^{11}$. 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 ${ }^{12}$. Our results agree with Baylan in OAE results but differ in pure tone audiometery as our results pointed out that no statistical difference between both groups regarding pure tone audiometery.

## Conclusion:

Our results pointed out that pre-eclampsia can influence the cochlea and in turn it may be a risk factor for sensory neural hearing loss in pregnant women, this may be temporarily. We recommend doing this study on wide scale of patients and following them for a while in postpartum.

## References

1. Myo clinic (2010) Pre-eclampsia
2. Bakhshaee M, Boskabadi H, Hassanzadeh M (2008) Hearing impairment in the neonate of preeclamptic women. Otolaryngol Head Neck Surg 139: 846-849.
3. Roberts JM, Pearson G, Cutler J (2003) Summary of the NHLBI working group on research on hypertension during pregnancy. Hypertension 41: 437-445.
4. Bakhshaee $M$, Hassanzadeh $M$, Nourizadeh N, Karimi E, Moghiman T, et al. (2008) Hearing impairment in pregnancy toxemia. Otolaryngol Head Neck Surg 139: 298-300.
5. Vinck BM, Van Cauwenberge PB, Leroy L, Corthals (1999) Sensitivity of transient evoked and disortion product otoacoustic emissions to direct effects of noise on the human cochlear. Audiol 38: 44-52
6. Altuntaş EE, Yenicesu AGI, Mutlu AE, Muderris S, Çetin M, et al. (2012) An evaluation of the effects of hypertension during pregnancy on postpartum hearing as measured by transient-evoked otoacoustic emissions. Acta Otorhinolaryngol Ital 32: 31-36.
7. Moraes L, Almeida E, Matsuo T (2006) Hypertension as a factor associated with hearing loss. Braz J Otorhinolaryngol 72: 533-540.
8. Nagahara K, Fisch U, Yagi N (1983) Perilymph oxygenation in sudden and progressive sensorineural hearing loss. Acta Otolaryngol 96: 57-68.
9. Maddox PT, Saunders J, Chandrasekhar SS (2009) sudden hearing loss from PDE-5 inhibitors: a possible cellular stress etiology. Laryngoscope 119: 1586-1589.
10. Yinon Y, Kingdom JC, Odutayo A (2010) vascular dysfunction in women with a history of preeclampsia and intrauterine growth restriction: insights into future vascular risk. Circulation 122: 1846-1853.
11. Ozdemir I, Yilmaz S, Albayrak M, Yıldızbas S, Guven DG, et al. (2011) Hearing evaluation in preeclamptic patients.Gynecol Obstet Invest 72: 1-4.
12. Baylan MY, Kuyumcuoglu U, Kale A, Celik Y, Topcu I (2010) Is preeclampsia a new risk factor for cochlear damage and hearing loss? Otol Neurotol 31: 1180-1183.
