**Audiological Profile of Vitiligo Patients**

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**Abstract:** Vitiligo Is A Systemic Disorder Influencing The Whole Pigmentary System Including Melanocytes In The Inner Ear. The Loss Or Reduction Of Melanocytes In The Inner Ear May Have A Critical Effect On Hearing; Most Vitiligo Patients Are Asymptomatic For Audiological Abnormalities. The Aim Of The Present Study Was To Evaluate The Auditory Function In Vitiligo Patients. The Study Group Included 15 Cases Of Various Types Of Vitiligo. Fifteen Age- And Sex-Matched Non Vitiligo Cases Were Included As Controls In The Study. A Thorough Audiological Examination Including Pure Tone Audiometry, Transient Evoked Otoacoustic Emission And Auditory Brainstem Response Were Carried Out In All Patients And Controls. The Result Shows Decreased Hearing At High Frequencies Which May Indicate More Serious Damage At The Base Of The Cochlea. Transient Oto-Acoustic Emission Result Shows A Significant Decrease In Reproducibility Percent Of Emission In The Vitiligo Group. Auditory Brainstem Response Result Shows A Statistically Significant Decrease Of The Peak I Latency And Increase Of The I-III Interpeak Latency In The Patients As Compared To The Controls.

**Key Words:** Vitiligo, Transient Evoked Otoacoustic Emission, Auditory Brainstem Response.

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**I. Introduction:**

Vitiligo is an acquired hypomelanotic disorder characterized by circumscribed depigmented macules or patches resulting from the loss of functional melanocytes and of melanin from the epidermis [4]. Vitiligo is not transmitted by simple mendelian mechanism and its inheritance pattern is more consistent with that of a polygenic trait [5]. Many possible causes of vitiligo have been proposed, including stress, infections, mutations, neural factors, melatonin receptor dysfunction, and impaired melanocyte migration and/or proliferation. In addition, the accumulation of toxic intermediate products of melanin synthesis [6], the breakdown of free radical defence [7] and the build up of excessive quantities of hydrogen peroxide [8] have all been suggested to result in the self-destruction of pigment cells. Although loss of melanocytes from the skin is almost always the primary and initial symptom in vitiligo, other pigment cells in the body can be affected.

Melanocytes are located in the inner ear [3] and vitiligo-associated auditory problems have been reported in some patients [10,11]. Damage can also occur to melanocytes within the eye. The affection of extracutaneous melanocytes in some vitiligo patients suggests that systemic immunological reactions directed at pigment cells might play a role in the development of the disease [4].

**Need For The Study:**

Although the loss or reduction of melanocytes in the inner ear may have a critical effect on hearing, most vitiligo patients are asymptomatic for audiological abnormalities. Melanocyte distributions and melanocyte-associated diseases differ, however. In various racial groups. To date, there have been very few studies of hearing loss in Asian patients with vitiligo, and no analysis of any relationship between hearing loss and severity of vitiligo. We therefore assessed differences in hearing parameters between Indian vitiligo patients and normal Indians.

**Objective Of The Study:**

The aim of the present study was to evaluate the auditory function in vitiligo patients to detect possible subclinical abnormalities of the auditory system in comparison to healthy persons.

**Subjects And Methods**

This study was conducted in the department of otorhinolaryngology, Guwahati Medical College & Hospital, Guwahati between April 2016 to June 2017. All subjects in this study were submitted to a full history taking, general medical and skin examination, otological history and otological examination.

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Patients Suffering From Metabolic, Vascular Or Autoimmune Diseases Or Any Systemic Disease Like Tuberculosis Were Excluding. Hearing Assessment Was Conducted In The Audiology Unit Of The Hospital. Conventional Pure-Tone And High-Frequency Audiometric Tests Were Obtained, And Pure-Tone Average Hearing Thresholds Were Calculated For 30 Consecutive Cases Of 10 To 20 Years (Mean Age Of 15.6 Years) With Vitiligo And 30 Sex- And Age-Matched Controls.

Transient Evoked Otoacoustic Emission (TEOAE) Using Condensation Click Stimulus At Intensity Equal To 70 Dbspl. The Software Determined The Amplitude Of TEOAE In Three Frequency Bands (1, 2, 3 KHz). The Analysis Was Also Performed For The Amplitude And Reproducibility Percent. Auditory Brain Stem Evoked Response (ABR) Was Performed Also With The Following Parameters:

- Stimulus: Rarefaction Click With A Duration Of 100 Msec.
- Rate: Regular Repetition Rate At 27.7pulses. Absolute Latencies Of Wave I, III And V And Interpeak Latencies I-III, III-V And I-V At Regular Repetition Rate. Also, Absolute Latency Of Wave V At High Repetition Rate Was Recorded.

**Statistical Analysis:**

Pure-Tone Audiometry, Auditory Brainstem Responses, And TEOAE Results Were Compared Between The Vitiligo And Control Groups And Using Student’s T-Test And Pearson’s Correlation Coefficient. All Statistical Analyses Were Performed Using SPSS Software (Version 12.0; SPSS Inc.; Chicago, IL). A P Value < 0.05 Was The Threshold Of Significance.

**II. Results:**

Using Pure Tone Audiometry, We Found That Hearing In The Right Ears Of The Vitiligo Group Was Significantly Lower Than In The Control Group At 125 Hz, 1,000 Hz, 4,000 Hz, 6,000 Hz, And 8,000 Hz (P < 0.05), And That Hearing In The Left Ears Of The Vitiligo Groups Was Significantly Lower Than In The Control Group At 1,000 Hz, 2,000 Hz, 4,000 Hz, 6,000 Hz, And 8,000 Hz (P < 0.05).

Reduction Of Transient Evoked Otoacoustic Emission Amplitude Was Statistically Significant At 4 Khz In Patients With Vitiligo (P = 0.023). There Was No Significant Difference In Reproducibility, Stimulus Intensity, Stability, And Average Transient Evoked Otoacoustic Emission Amplitudes In Patients With Vitiligo.

**Figure 1.** Comparison Of Conventional Pure-Tone And High Frequency Audiometry Results In Patient And Normal Group.

**Figure 2.** Comparison Of Transient Evoked Otoacoustic Emission Signal-To-Noise Ratios In Patient And Control Group.
When We Compared Auditory Brainstem Responses, We Found That, In Right Ears, The Cases Showed Significant Decreases In Wave I And Wave III Latencies And Significant Increases In Third Amplitude And Interpeak Latencies I-III And I-V (P < 0.05). For Left Ears, The Disease Group Showed A Significant Decrease In Wave I Latency And Significant Increases In Interpeak Latencies I-III And I-V (P < 0.05) Compared With The Normal.

### Table 1. Comparison Of ABR Parameters Between Vitiligo Patients And Normal Subjects For Right-Ear Stimulation.

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<tr>
<td>Normal Subjects</td>
<td>1.56 ± 0.17</td>
<td>3.72 ± 0.17</td>
<td>5.60 ± 0.23</td>
<td>3.28 ± 0.12</td>
<td>0.28 ± 0.14</td>
<td>0.26 ± 0.13</td>
<td>2.13 ± 0.15</td>
<td>1.90 ± 0.14</td>
<td>4.04 ± 0.21</td>
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<td>Vitiligo Patients</td>
<td>1.60 ± 0.07</td>
<td>3.84 ± 0.18</td>
<td>5.68 ± 0.22</td>
<td>3.25 ± 0.13</td>
<td>0.24 ± 0.11</td>
<td>0.27 ± 0.12</td>
<td>2.23 ± 0.15</td>
<td>1.83 ± 0.14</td>
<td>4.07 ± 0.20</td>
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### Table 2 Comparison Of ABR Parameters Between Vitiligo Patients And Normal Subjects For Left-Ear Stimulation.

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<tbody>
<tr>
<td>Normal Subjects</td>
<td>1.56 ± 0.17</td>
<td>3.71 ± 0.17</td>
<td>5.62 ± 0.23</td>
<td>3.30 ± 0.12</td>
<td>0.26 ± 0.14</td>
<td>0.29 ± 0.13</td>
<td>2.15 ± 0.11</td>
<td>1.89 ± 0.15</td>
<td>4.05 ± 0.18</td>
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<tr>
<td>Vitiligo Patients</td>
<td>1.57 ± 0.10</td>
<td>3.81 ± 0.19</td>
<td>5.66 ± 0.23</td>
<td>3.28 ± 0.15</td>
<td>0.26 ± 0.16</td>
<td>0.31 ± 0.14</td>
<td>2.13 ± 0.49</td>
<td>1.76 ± 0.40</td>
<td>3.89 ± 0.88</td>
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### III. Discussion:

**Pure Tone Audiometry:** Using Pure Tone Audiometry, We Found That Average Hearing Thresholds In The Control Group Were 7.12 Db In Right Ears And 7.52 Db In Left Ears. In Comparison, Thresholds In The Vitiligo Group Were 9.69 Db In Right Ears And 10.18 Db In Left Ears. Although All Subjects Were Within Normal Hearing Ranges, We Found That Hearing Decreased At High Frequencies In The Vitiligo Group Compared With The Control Group. Decreased Hearing At High Frequencies May Indicate More Serious Damage To The Stria Vascularis, Especially To Intermediate Cells At The Base Of The Cochlea. In Correlations Of Auditory Sensation Areas And Optimal Frequencies Of Basement Membranes, High Frequencies Are Recognized At The Base Of The Cochlea, Whereas Low Frequencies Are Recognized Toward The Apex.

**Transient Oto-Acoustic Emission (TEOAE):** Using Another Audiologic Test For Evaluation Of Cochlear Function (TEOAE), Revealed A Significant Decrease In Reproducibility Percent Of TEOAE In The Vitiligo Group. The Reproducibility Percent Was 33.3 In The Study Group Compared To 98% In The Control Group. Significant Differences In TEOAE At Different Frequencies Were Also Detected. In A Study By [2] The Mean Of The Whole Reproducibility Percentage Was 49% For Vitiligo Group With More Than 10 Years Duration. The Lost Cochlear Emission In Vitiligo Group Was Explained Previously By [13]. They Stated That Hypo Pigmentation Disorders For A Long Duration May Lead To Degeneration Of The Outer Hair Cells Beginning From The Basal Turn Of The Cochlea While Inner Hair Cells Remain Structurally And Functionally Intact.

**Auditory Brainstem Response (ABR):** Patients With Vitiligo Have Found A Statistically Significant (P < 0.01) Decrease Of The Peak I Latency And Increase Of The I-III Interpeak Latency In The Patients As Compared To The Controls. It Is Explained That The Decrease Of First Peak Latency To Be Due To Numerical Decrease Of Active Melanocytes In The Inner Ear Resulting In An Impairment Of The Ion Exchange Between The Endolymph And Perilymph With Disturbance Of The Transduction Of The Auditory Stimulation In The Inner Ear. The Increased I-III Interpeak Latency Is Explained To Be Due To Abnormal Synaptic Activity And Transmission Of The Action Potential From The Auditory Nerve To The Superior Olive. Similarly, A Statistically Significant Decrease Of Wave I Latency Was Found. It May Be Due To Cochlear Lesion Present In All Melanin Deficient Patients.

### IV. Conclusion:


### References:
