Evaluation of heart rate variability and night-time blood pressure measurements in patients with idiopathic sudden sensory neural hearing loss

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Abstract. – OBJECTIVE: The aim of this study is to investigate the role of the autonomic nervous system in the etiology of idiopathic sudden sensorineural hearing loss (ISSHL) by measuring heart rate variability (HRV) and night-time blood pressure levels.

PATIENTS AND METHODS: A total of 58 patients, 31 ISSHL patients (group 1) and 27 healthy volunteers (control group; group 2), were included in this study. Clinical and ambulatory blood pressure measurements and Holter electrocardiography were performed in both groups. After these evaluations, HRV parameters and night-time blood pressure values were determined.

RESULTS: Mean systolic blood pressure (SBP) and mean diastolic blood pressure (DBP) measured at night-time were higher in group 1 compared to group 2 (p < 0.05). Heart rate variability parameters were lower in group 1 than in group 2.

CONCLUSIONS: In patients with ISSHL, elevated blood pressure at night-time and reduced heart rate variability suggest that autonomic nervous system dysfunction might play a role in the etiopathogenesis of the disease. The measurements of ambulatory blood pressure and heart rate variability can reveal more enlightening data in the determination of the etiology of ISSHL and guiding the treatment.

Key Words: Sensorineural hearing loss, Night-time blood pressure, Heart rate variability, Autonomic nervous system.

Introduction

Idiopathic sudden sensorineural hearing loss (ISSHL) was first described in 1944 as a sudden onset, generally unilateral hearing loss of at least 30dB in at least three consecutive frequencies that develops within 72 hours. The diagnosis is made by exclusion of any other pathology causing sudden sensorineural hearing loss. In 28-57% of patients, vestibular symptoms, fullness in the ear, tinnitus, variable degrees of vertigo and balance disorder can be seen. Although various causes of ISSHL have been suggested such as viral infections, vascular occlusion, autoimmunity, cochlear membrane damage, toxic and vascular reasons, viral infections and vascular pathologies stand in the forefront among these causes. One of the factors that can contribute to the vascular pathology is impairment in the autonomic nervous system. Impairment in the autonomic regulatory mechanisms can result in changes in blood flow to the cranial structures indirectly by causing some dysfunction in the cardiovascular system, which may result in impaired nutrient supply and oxygenation in the inner ear and other structures. Two of the methods that are used to evaluate the effects of autonomic irregularities on the cardiovascular system are 24 hours Holter electrocardiography and ambulatory blood pressure measurement. These two techniques allow us to effectively evaluate the heart rate variability and blood pressure fluctuations. Especially the arterial blood pressure measurements at night-time can provide valuable data in terms of autonomic dysfunction in ISSHL etiology, because the night-time measurements are not affected by the factors such as exercise, smoking, caffeine consumption and stress which can have some influence on autonomic regulation. Additionally, heart rate variability (HRV) is another parameter...
which can show improper autonomic regulation. It has been demonstrated that HRV decreases in diseases such as diabetes mellitus and myocardial infarction\(^4,5\). Especially in patients with ISSHL, determination of autonomic dysfunction can aid in elicitation of etiopathogenesis. The autonomic nervous system works to keep our body in a state of equilibrium against the environmental, psychological and physical stress conditions. The cranial nuclei and secretory glands (both are components of autonomic nervous system) in humans, which are responsible for maintaining our circadian rhythm, are strongly affected by some factors such as chronic sleep deprivation and exposure to light at nights in today’s civilized life\(^6\). Inadequate or excessive regulatory responses in our autonomic nervous system can trigger different pathological processes in many organ systems. ISSHL can as well be such a process. Therefore, in this study we have evaluated heart rate variability and blood pressure changes in patients with ISSHL as determinants of autonomic dysfunction.

**Patients and Methods**

**Patient Population**

Patients admitting to our Ear, Nose and Throat Outpatient Clinics with the complaint of sudden hearing loss between 2014-2015 were included in this study. ISSHL was defined as the sudden onset hearing loss of at least 30dB in at least three consecutive frequencies in audiometry test that developed progressively within 72 hours and presented in one or both ears in the absence of any other etiological factors as determined by patient history, physical examination, laboratory evaluation and imaging. A total of 79 patients with the diagnosis of ISSHL were evaluated, and those with hypertension, cardiac, vascular, metabolic, neurologic, otologic, and psychologic disease, and those receiving any medical treatment were excluded. A total of 58 patients, 31 with ISSHL and 27 healthy volunteers, were included in the study. An ethical approval was obtained from Erzincan University local Ethics Committee and the study was conducted according to the Declaration of Helsinki. All patients were informed before the study and their consents were obtained.

**Study Design**

In this study patient group and control group was designated as group 1 and group 2, respectively. In order to investigate the secondary causes of hearing loss patients were assessed by Ear-Nose-Throat, Neurology, Internal Medicine and Psychiatry Outpatient Clinics. Those patients with a diagnosis of ISSHL were also evaluated by cardiology clinic. Conventional echocardiographic evaluation was performed to exclude cardiac pathologies. A 24 hours blood pressure monitoring was obtained by ambulatory blood pressure measurement and 24 hour electrocardiographic Holter was used to determine the HRV. All of the evaluations were performed by two cardiologists.

**Measurement of Clinical Blood Pressure**

Blood pressure measurements were performed according to the standard blood pressure measurement rules, manually using a sphygmomanometer after a rest of 5 minutes in sitting position. Those with a mean value of ≤ 140/90 mm Hg of three consecutive measurements were included in the study.

**Evaluation of Ambulatory Blood Pressure**

24 hours ambulatory systolic (SBP) and diastolic blood pressure (DBP) measurements were taken automatically by oscillometric portable monitor (SpaceLabs, Medical Inc, Model: 92512, Redmond, WA, USA) from the non-dominant arm. The measurements were taken once in every 20 min between 07:00-22:00 (day-time) and once in every 30 min between 22:00-07:00 (night-time). The cuff sizes were chosen in accordance with the arm circumference of the individuals. All patients and healthy controls were advised to carry on their routine daily activities and to avoid excessive exercise, smoking, alcohol and caffeine containing drinks. Ambulatory blood pressure data was recorded digitally. The blood pressure values during sleep and awake state were recorded.

**Determination of Time Related Variability in Heart Rate**

A 24 hours Holter electrocardiography monitoring was undertaken. The ‘Holter WIN-PV plus’ software was used during monitorization. All the Holter recordings were evaluated manually to avoid interference of any artefacts and then ‘time domain’ variability in heart rate parameters were calculated automatically. The following parameters were chosen for evaluation: 24 hours SDNN (the standard deviation of all normal R-R intervals), SDNN-i (the mean of standard deviation...
tions of all R-R intervals within the 5 min of the recordings), SDANN (the standard deviation of the means of all R-R intervals within the 5 min of the recordings), pNN_50 (the ratio of adjacent R-R intervals that demonstrate a difference of more than 50 milliseconds), RMSSD (the square root of the difference between the consecutive R-R intervals), NN (the distance between two QRS segments). Patients were instructed not to take any medications and caffeinated drinks up to 48 hours before the application of Holter electrocardiography.

**Conventional Echocardiographic Evaluation**

The transthoracic echocardiographic evaluations were performed by a Vivid 7 Dimension® (GE Vingmed Ultrasound AS N-3190 Horten, Norway) echocardiography device with the use of a 2.5 MHz transducer. Patients were evaluated in left lateral decubitus position after a 5 min rest. The pericardium, heart valve morphology and wall motion were evaluated by M mode and 2 dimensional echocardiography.

**Statistical Analysis**

All of the data was recorded in the Statistical Package for Social Sciences 20.0 software programme (SPSS Inc., Chicago, IL, USA). Parametric variables were expressed as mean ± standard deviation whereas non parametric data were expressed as percentages. A distribution test was used in comparison of demographic and clinical features. A Chi square test was used to compare ratios of parametric data when the distribution was normal and a *t*-test was used in comparing parametric data with non-parametric data. A Student *t*-test and Mann-Whitney U-test were used to compare numeric variables that are normally distributed and not normally distributed, respectively. A *p* value of < 0.05 was designated as statistically significant in all analyses.

**Results**

The comparison of the groups in terms of demographics, clinical features and blood pressures is shown in Table I. There was no statistically significant difference between the groups in terms of age, gender and heart rate. Also there was not any significant difference between groups in terms of day-time and clinical systolic and diastolic blood pressures. But the night-time systolic blood pressure was significantly higher in Group 1 compared to Group 2 (Group 1: 130.6 ± 5.1; Group 2: 124.1 ± 5.1, respectively, *p* < 0.05) (Figure 1). Also nighttime diastolic blood pressure values was significantly higher in Group 1 compared to Group 2 (respectively Group 1; 83.2±3.8; Group 2; 80.2 ± 4.7, *p* < 0.05) (Figure 2).

The comparison of the groups in terms of heart rate variability in relation to time parameters is shown in Table II. NN was significantly lower in Group 1 compared to Group 2 (*p* < 0.05). SDNN was significantly lower in Group 1 compared to Group 2 (*p* < 0.05). SDANN and SDNN-i was significantly lower in Group 1 compared to Group 2 (*p* < 0.05). RMSSD was significantly lower in Group 1 compared to Group 2 (*p* < 0.05). pNN_50 was significantly lower in Group 1 compared to Group 2 (*p* < 0.05).

**Table I.** Comparison of the groups in terms of demographics, clinical features and blood pressures.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group 1 (n: 31)</th>
<th>Group 2 (n: 27)</th>
<th><em>p</em>-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48 ± 17</td>
<td>49 ± 14</td>
<td>0.985</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>20/11</td>
<td>17/10</td>
<td>0.675</td>
</tr>
<tr>
<td>Heart rate (beat/minute)</td>
<td>81 ± 8.3</td>
<td>78 ± 7.7</td>
<td>0.095</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>121.8 ± 9.6</td>
<td>122.3 ± 5.8</td>
<td>0.881</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>80.1 ± 6.2</td>
<td>78.9 ± 3.9</td>
<td>0.643</td>
</tr>
<tr>
<td>24-hour SBP (mmHg)</td>
<td>123.4 ± 7.1</td>
<td>120.8 ± 6.6</td>
<td>0.123</td>
</tr>
<tr>
<td>24-hour DBP (mmHg)</td>
<td>79.6 ± 7.9</td>
<td>78.9 ± 4.5</td>
<td>0.760</td>
</tr>
<tr>
<td>Day-time SBP (mmHg)</td>
<td>122.6 ± 6.2</td>
<td>122.4 ± 4.8</td>
<td>0.642</td>
</tr>
<tr>
<td>Day-time DBP (mmHg)</td>
<td>79.2 ± 5.8</td>
<td>79.4 ± 3.2</td>
<td>0.867</td>
</tr>
<tr>
<td>Night-time SBP (mmHg)</td>
<td>130.6 ± 5.1</td>
<td>124.1 ± 5.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Night-time DBP (mmHg)</td>
<td>84.2 ± 3.8</td>
<td>80.2 ± 4.7</td>
<td>0.010</td>
</tr>
</tbody>
</table>

SBP: Systolic blood pressure, DBP: Diastolic blood pressure.
In this study we demonstrated an increased night-time systolic and diastolic blood pressures and decreased time related variability in heart rate in patients with ISSHL. Approximately half of the patients with ISSHL have the symptoms that result in an impairment in quality of life such as balance disorder, fullness in the ear, tinnitus and vertigo. It is very important to understand the underlying etiology in order to guide the treatment. Vascular pathologies were accused initially in the etiology of ISSHL. The reason for considering a vascular pathology as an offender is that it occurs suddenly just like myocardial infarction and frequently coexists with a systemic vascular disease. It can be the case that vascular physiopathology can lead some irregularity in the blood flow to the cranial structures which can then contribute to the development of ISSHL. Also autonomic nervous system (ANS), being the main regulator of the cardiovascular functions, can play a key role in the vascular physiopathology of the ISSHL. The regulatory function of the ANS is briefly as follows: the day-night time cycle occurring as a result of sun rise and sunset allows us to maintain our circadian rhythm regularly in normal circumstances. Circadian rhythm activates many regulatory processes through neurohormonal effects such as immune system, musculoskeletal system, cardiovascular system, metabolism and regulation of reparative and reconstructive activities in other organ systems. These processes reveal their effects through the nuclei and endocrine glands within the ANS. It has been shown that the impairment in the circadian rhythm and chronic sleep deprivation result in abnormalities in the autonomic functions which cause different pathologic processes in the development of ISSHL.

### Table II.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group 1 (n: 31)</th>
<th>Group 2 (n: 27)</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NN (ms)</td>
<td>510 ± 39.1</td>
<td>549 ± 74.4</td>
<td>0.030</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>81 ± 5.3</td>
<td>89 ± 8.0</td>
<td>0.036</td>
</tr>
<tr>
<td>SDANN (ms)</td>
<td>71 ± 3.0</td>
<td>83 ± 2.2</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>SDNN-i (ms)</td>
<td>63 ± 3.4</td>
<td>71 ± 2.3</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RMSSD (ms)</td>
<td>60 ± 4.1</td>
<td>66 ± 3.0</td>
<td>0.032</td>
</tr>
<tr>
<td>pNN50 (%)</td>
<td>11.5 ± 1.2</td>
<td>13 ± 0.7</td>
<td>0.012</td>
</tr>
</tbody>
</table>

NN; the distance between two QRS segments, SDNN; the standard deviation of all normal R-R intervals, SDNN-i; the mean of standard deviations of all R-R intervals within the 5 min of the recordings, SDANN; the standard deviation of the means of all R-R intervals within the 5 min of the recordings.
pressure can play a role in the etiology of the ISSHL. Mutoh et al. in their mammalian animal study, demonstrated an increase in the sympathetic activity and a decrease in the vagal autonomic regulation can explain the increase in heart rate variability and the occurrence of ISSHL. Especially in elderly patients heart rate variability is reported to decrease as a result of ANS dysfunction. Also in the elderly ISSHL can be seen. But there was no difference between the patient group and the control group in terms of age in our study.

Electrical signals are carried from the nerve endings located in the inner ear to the brain. A pathology in these nerve endings or in the pathways that carry the signals to the brain can lead to autonomic dysfunction and sensory neural hearing loss. In this situation night-time blood pressure variability can occur too. For this reason the concomitant occurrence of ISSHL and autonomic dysfunction can lead some elevation in night-time measured blood pressures and decrease in heart rate variability. But it is hard to say whether ISSHL is due to autonomic dysfunction or vascular pathology.
Conclusions

Impaired autonomic nervous system functions can play an important role in the etiology of the ISSHL. Determination of blood pressure changes and heart rate variability in cardiovascular system can be an important tool in the clinical practice to evaluate autonomic dysfunction. In clinical practice, instead of a single measurement to predict the existence of autonomic dysfunction, a 24 hours follow up of the blood pressure can provide us more informative data. Chronic irregularities in the autonomic nervous system and their role in the physiopathology of many diseases have been reported in various studies. We think that larger scale clinical studies designed in detail can demonstrate the role of autonomic nervous system dysfunction in the etiology of ISSHL.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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