European Review for Medical and Pharmacological Sciences 2023; 27: 5153-5158

Is atrial fibrillation a risk factor for hearing loss?

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Abstract. – OBJECTIVE: In the present study, we sought to evaluate the results of hearing loss in AF patients.

PATIENTS AND METHODS: This study involved 50 patients with AF, as determined by means of electrocardiogram, and 50 patients without AF. The pure-tone audiometry (PTA) threshold values were measured at low, medium and high frequencies for both ears. The signal-to-noise ratio (SNR) DPOAEs and TEOAEs were also analyzed for both ears separately.

RESULTS: Both the airway and bone conduction PTA thresholds at 3, 4 and 6 kHz (kilohertz) were significantly lower in the AF group than in the control group ($p<0.05$). The AF patients exhibited worse hearing and worse TEOAE results at 1, 2, 3 and 4 kHz. In fact, the TEOAE amplitudes of the AF group were significantly lower in both the right and left ears at 2, 3 and 4 kHz when compared with the control group ($p<0.05$). Moreover, the DPOAE amplitudes in the AF group were statistically significantly lower at 3.4 kHz in both ears when compared with the control group ($p<0.05$).

CONCLUSIONS: In light of these findings, we believe that AF is a risk factor for hearing.

Key Words:
Atrial fibrillation, Hearing loss, Cochlea, Cardiac output.

Introduction

The most common cardiac arrhythmia is atrial fibrillation (AF). It significantly increases both morbidity and mortality, in addition to leading to a significant decrease in quality of life[1]. AF is associated with a high risk of complications such as thromboembolic events, coronary events and heart failure[2]. Moreover, atrioventricular synchrony disorder can develop in cases of AF, leading to a decrease in cardiac output and the deterioration of the left ventricular filling. As a result, cerebral hypoperfusion can develop[3].

Hearing loss occurs when one or more of the pathways that provide hearing are impaired. One of these ways, the cochlea, carries out sound transmission in the inner ear[4]. Cochlear blood supply plays an important role in hearing, and cochlear blood vessels are responsible for maintaining the blood-labyrinth barrier, transporting systemic hormones for ion homeostasis, and providing nutrients for metabolic functions[5,6]. As a consequence of hypoperfusion or hypoxia that develops in the cochlea, inflammatory factors such as nitric oxide are released. Nitric oxide causes cochlear dysfunction through damaging the cochlear hair cells[7]. Increasing severity of hypoxia affects first the inner hair cells and then the outer hair cells, causing increasing damage from the cochlear apex to the cochlear base[8]. While high-frequency sound waves vibrate near the cochlear base, low-frequency sound waves vibrate near the cochlear apex[9].

There are some studies[10-12] reporting that cochlear functions are adversely affected by decreased cochlear blood flow as a result of cardiovascular diseases. We think that it is possible that embolic infarctions developed as a result of AF and the resulting decrease in cardiac output may cause hearing loss by affecting cochlear blood flow. As far as we know, there is no study investigating the presence of hearing loss in AF patients using audiological examinations. Therefore, in this study, we aimed to determine whether hearing loss occurs in AF patients by using audiological examinations.

Patients and Methods

Study Population

This study involved 50 patients (27 men and 23 women) with AF, as determined by means of electrocardiogram, and 50 patients (28 men and 22 women) without AF who were aged between

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18 and 60 years and who applied to the Cardiology Clinic of Diyarbakır Gazi Yaşargil Training and Research Hospital. All patients signed an informed consent form before starting the study. Any patients who had undergone ear surgery, had certain hearing impairments (e.g., rubella infection, head trauma), smoked, had any metabolic disorder (e.g., diabetes, chronic kidney disease, hyperthyroidism) or vascular disorder (e.g., stroke), had noise-induced hearing loss, worked in noisy environments, had previously taken ototoxic drugs and/or had systolic left ventricular dysfunction were excluded from the study. However, AF patients with hypertension and/or heart valve disease were included in this study.

Data Collection

All patients (i.e., both with and without AF according to their electrocardiogram) were also evaluated by means of echocardiography (ECO). Blood pressure measurements were taken. Moreover, the patients' plasma glucose, urea, creatinine, thyroid hormones, total cholesterol, triglyceride, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) levels were measured. After the patients' otoscopic examinations were performed in the ear, nose and throat clinic, pure-tone audiometry and otoacoustic emission tests were performed in both ears to evaluate their hearing.

Hearing Assessment

Trained professionals performed the patients' audiometric tests in a soundproofed room using the OTOsuite program of an Otometrics MADSEN Astera audiometer (United Kingdom). Air conduction hearing thresholds were measured using pure tones at six frequencies (0.5, 1, 2, 3, 4 and 6 kHz for each ear separately). The pure tone frequencies were then measured at 0.5, 2, 3, 4 and 6 kHz. The pure-tone audiometry (PTA) threshold values were measured at low, medium and high frequencies for both ears. PTA thresholds of low, medium, and high frequency were calculated using the mean of the PTA at 0.5 and 1 kHz, the mean of the PTA at 1 and 2 kHz, and the mean of the PTA at 3,4 and 6 kHz, respectively. We defined the hearing levels of the subjects included in the study as normal hearing (PTA threshold $\leq$ 25 dB) and hearing loss (PTA threshold > 25 dB).

In addition, TEOAEs and DPOAEs, which are the basic tests used to reveal cochlear function by evaluating the integrity of the outer hair cells of the inner ear, are audiological evaluation methods that can be used. The TEOAE and DPOAE amplitudes were measured using the OTOsuite program of an Otometrics MADSEN Capella (United Kingdom) device with the aid of a microphone in a probe that was placed in the external auditory canal. The acoustic stimuli obtained with an 80 dB peak SPL (peak-equivalent sound pressure level) were recorded for the evaluation of the TEOAEs. DPOAEs were evaluated using acoustic stimuli obtained using two pure tones of different frequencies and intensities (65 dB peak SPL and 55 dB peak SPL). Both TEOAEs and DPOAEs were analyzed in the frequency bands of 1, 2, 3 and 4 kHz. The signal-to-noise ratio (SNR) DPOAEs and TEOAEs were also analyzed for both ears separately. A record was made when the SNR value was $\geq$6. The SNR amplitudes in each frequency band were taken into account during both tests.

Statistical Analysis

All the gathered data were analyzed using IBM's Statistical Package for the Social Sciences (SPSS) Statistics 20 software (IBM Corp., Armonk, NY, USA). The Chi-square test was used to compare the categorical variables between the groups, while the independent samples *t*-test was used to compare the numerical variables. Statistical significance was determined to be $p<0.05$.

Results

Both the AF group (34 men and 16 women) and the control group (31 men and 19 women) consisted of 50 patients. The patients' ages ranged from 26 to 58 years (mean: 46.9±6.81 years). The groups included in this study were similar in terms of age or gender ($p>0.05$). The AF patients' systolic and diastolic blood pressures were found to be significantly higher than those of the control patients ($p<0.05$) (Table I).

With regard to the pure-tone audiometry, there was no statistically significant difference observed between the two groups in terms of either the airway or bone conduction at PTA thresholds between 0.5 and 2 kHz ($p>0.05$). The obtained PTA values were within normal limits for the patients in both groups. However, both the airway and bone conduction PTA thresholds at 3, 4 and 6 kHz were significantly lower in the AF group than in the control group ($p<0.05$) (Table II).

Table I. Clinical characteristics of AF and control groups.

	AF group	Control group	*p*-value
Age	46.6 ± 6.96	47.3 ± 6.71	$p > 0.05$
Systolic Blood Pressure	130.8 ± 11.5	103.9 ± 10.7	$\mathbf{p < 0.05}$*
Diastolic Blood Pressure	83.8 ± 9.3	64.1 ± 7.8	$\mathbf{p < 0.05}$*

Data are expressed as mean ± SD. AF: Atrial Fibrillation. *p statistically significant.

Table II. Comparison of PTA between AF group and control group.

	AF group	Control group	*p*-value
Right ear			
Low Frequency PTA	14.10 ± 3.59	14.20 ± 3.55	$p > 0.05$
Medium Frequency PTA	25.44 ± 7.65	22.10 ± 7.69	$p > 0.05$
High Frequency PTA	49.7 ± 9.64	30.6 ± 8.72	$\mathbf{p < 0.05}$*
Left ear			
Low Frequency PTA	14.20 ± 3.69	14.30 ± 3.78	$p > 0.05$
Medium Frequency PTA	18.8 ± 5.58	18.10 ± 5.88	$p > 0.05$
High Frequency PTA	46.10 ± 8.88	28.40 ± 7.72	$\mathbf{p < 0.05}$*

Data are expressed as mean ± SD. AF: Atrial Fibrillation, PTA: Pure-tone audiometry. *p statistically significant.

The AF patients exhibited worse hearing and worse TEOAE results at 1, 2, 3 and 4 kHz. In fact, the TEOAE amplitudes of the AF group were significantly lower in both the right and left ears at 2, 3 and 4 kHz when compared with the control group ($p<0.05$) (Table III). Moreover, the DPOAE amplitudes in the AF group were statistically significantly lower at 3.4 kHz in both ears when compared with the control group ($p<0.05$) (Table IV).

Discussion

It is believed that embolic infarctions and low cardiac output in patients with AF cause both cerebral and cochlear hypoperfusion. In addition, blood pressure was found to be high in patients with AF in our study. Therefore, the coexistence of AF and hypertension is thought to reduce cochlear and cerebral hypoperfusion. It has been suggested that such cognitive impairment may be associated with hearing loss. In the present study, we detected high-frequency sensorineural hearing loss in the AF patients when compared with the control patients based on hearing tests, such as PTA, TEOAE and DPOAE. We consider that, as a result of cochlear hypoperfusion, the AF patients may have experienced damage to their inner and outer hair cells, which will have gradually increased from the cochlear apex, where low-frequency sound waves vibrate, to

Table III. Comparison of TEOAE measurements between AF group and Control group.

	AF group	Control group	*p*-value
Right ear			
1 kHz	6.10 ± 3.92	6.60 ± 3.63	$p > 0.05$
2 kHz	4.97 ± 2.60	8.03 ± 0.96	$\mathbf{p < 0.05}$*
3 kHz	3.60 ± 1.36	8.05 ± 1.23	$\mathbf{p < 0.05}$*
4 kHz	1.92 ± 1.29	8.57 ± 1.25	$\mathbf{p < 0.05}$*
Left ear			
1 kHz	4.53 ± 2.24	4.85 ± 2.50	$p > 0.05$
2 kHz	3.59 ± 2.55	8.10 ± 0.93	$\mathbf{p < 0.05}$*
3 kHz	2.36 ± 1.25	8.27 ± 1.24	$\mathbf{p < 0.05}$*
4 kHz	2.45 ± 2.30	8.57 ± 1.30	$\mathbf{p < 0.05}$*

Data are expressed as mean ± SD. AF: Atrial Fibrillation, kHz: kilohertz. *p statistically significant.

Table IV. Comparison of DPOAE measurements between AF group and control group.

	AF group	Control group	*p*-value
Right ear			
1 kHz	8.04 ± 2.04	8.3 ± 1.19	$p > 0.05$
2 kHz	8.94 ± 3.53	7.4 ± 1.76	$p > 0.05$
3 kHz	3.08 ± 1.19	8.1 ± 1.6	$\mathbf{p < 0.05}$*
4 kHz	2.08 ± 0.96	8.16 ± 1.25	$\mathbf{p < 0.05}$*
Left ear			
1 kHz	6.34 ± 2.78	6.94 ± 2.77	$p > 0.05$
2 kHz	7.8 ± 3.44	7.46 ± 1.72	$p > 0.05$
3 kHz	2.38 ± 1.25	8.06 ± 1.59	$\mathbf{p < 0.05}$*
4 kHz	6.12 ± 3.14	8.08 ± 1.15	$\mathbf{p < 0.05}$*

Data are expressed as mean ± SD. AF: Atrial Fibrillation. **p* statistically significant.

the cochlear base, where high-frequency sound waves vibrate, thereby causing hearing loss. In other words, we think that, in addition to hearing loss due to cognitive impairment in AF patients, hearing loss may also occur due to cochlear hypoperfusion.

Conen et al[13] demonstrated the presence of both cortical and non-cortical infarctions in clinically asymptomatic AF patients using magnetic resonance imaging (MRI). Moreover, they reported that the cognitive functions were worse in these patients. Similarly, Malavasi et al[14] found AF to be associated with cognitive impairment and dementia. Shamloo et al[15] reported that cerebral hypoperfusion, changes in the cerebral blood flow, cerebral microhemorrhages, microembolism, vascular inflammation, cerebral small vessel diseases and brain atrophy are all among the underlying mechanisms associated with cognitive impairment in AF patients. We believe that similar pathologies may occur in the cochlear and cerebral vessels of AF patients, thereby altering the cochlear blood flow and potentially leading to the development of hearing loss.

The labyrinth artery, a branch of the anterior inferior cerebellar artery, gives off many branches and provides blood flow to the inner ear, which consists of the cochlea and vestibule. In addition, the blood feeding the cochlea constitutes 10-7 of the total cardiac output[16]. Anselmino et al[17] explained the mechanism by which the hemodynamic parameters of the distal cerebral circulation at the arteriolar and capillary levels are affected in AF patients. More specifically, they stated that hypoperfusion at the arteriolar level and a hypertensive event at the capillary level during AF may reduce the blood flow. Furthermore, it has been reported that atrioventricular synchronic disorder in those with AF causes a decrease in the cardiac output and the deterioration of the left ventricular filling. As a consequence, cerebral hypoperfusion develops[3]. Wang et al[18] reported that hearing loss is a factor associated with cognitive impairment in patients with AF. They also suggested that AF is a risk factor for both cognitive impairment and dementia. Although the underlying mechanisms are known to be multifactorial, strokes and embolic brain infarctions are thought to contribute to this phenomenon. It has also been reported to be one of the underlying causes of cerebral hypoperfusion[18]. Similar to the findings of this study, we believe that hearing loss may develop in AF patients. However, we emphasize that this hearing loss may develop due to cognitive impairment stemming from cerebral hypoperfusion and/or cochlear hypoperfusion as a result of low cardiac output and embolic infarctions in AF patients.

Fanaei et al[19] evaluated hearing loss due to bilateral carotid occlusion in rats. They reported that, in rats with ischemia, the basal part of the cochlea is more sensitive to damage caused by ischemia and, as a result, hearing loss develops more at high frequencies than at low frequencies[19]. Similarly, we found that, as a result of hypoperfusion and ischemia in the cochlea of AF patients, the basal part of the cochlea is more affected than the apex and also that sensorineural hearing loss develops at high frequencies in these patients. However, it is important to determine whether the cause of AF is ischemia, which suggests that further studies involving larger study populations and longer follow-up periods are required.

Conclusions

This study found that the otoacoustic emission amplitudes were lower in patients with AF and also that hearing loss at high frequencies was more common in these patients when compared with patients without AF. Considering these findings, we believe that AF is a risk factor for hearing loss, which indicates that patients with AF should be evaluated in terms of hearing loss. In this study, we emphasized that while investigating many etiologies in patients with hearing loss, it should not be forgotten that AF is a disease that causes hearing loss.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Authors' Contribution

Conceptualization: Mehmet Zulkif Karahan (MZK), Sermin Can (SC): Data curation: MZK, SC; Formal analysis: MZK, SC; Funding acquisition: MZK, SC; Investigation: MZK, SC; Methodology: MZK, SC; Project administration: MZK; Resources: MZK, SC; Software: MZK, SC; Supervision: MZK, SC; Validation: MZK, SC; Visualization: MZK, SC; Roles/Writing-original draft: MZK, SC; Writing-review & editing: MZK, SC.

Funding

The authors received no financial support for this article's research, authorship and/or publication.

Ethics Approval

The Local Ethics Committee approved the study protocol (Gazi Yaşargil Training and Research Hospital; No. 705, March 5, 2021). The study was conducted following the Declaration of Helsinki's Ethical Guidelines for Human Testing (2013).

Availability of Data and Materials

Data are available upon reasonable request to the corresponding author. De-identified data might be available after the consent of all authors and the privacy policy of Diyarbakır Gazi Yaşargil Training and Research Hospital.

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Informed Consent

All patients signed an informed consent form before starting the study.

References

1) Andrade J, Khairy P, Dobrev D, Nattel SJCr. The clinical profile and pathophysiology of atrial fibrillation: relationships among clinical features, epidemiology, and mechanisms. Circ Res 2014; 114: 1453-1468.
2) Schnabel RB, Pecen L, Engler D, Lucerna M, Sellal JM, Ojeda FM, De Caterina R, Kirchhof P. Atrial fibrillation patterns are associated with arrhythmia progression and clinical outcomes. Heart 2018; 104: 1608-1614.
3) Hui DS, Morley JE, Mikolajczak PC, Lee RJAhj. Atrial fibrillation: a major risk factor for cognitive decline. Am Heart J 2015; 169: 448-456.
4) Casale J, Kandle PF, Murray I, Murr N. Physiology, Cochlear Function. StatPearls Publishing, Treasure Island; 2022.
5) Shi XJHr. Physiopathology of the cochlear microcirculation. Hear Res 2011; 282: 10-24.
6) Trune DR, Nguyen-Huynh A. Vascular Pathophysiology in Hearing Disorders. Semin Hear 2012; 33: 242-250.
7) Orita Y, Sando I, Miura M, Haginomori S-I, Hirsch BEJO, neurotology. Cochleosaccular pathology after perinatal and postnatal asphyxia: histopathologic findings. Otol Neurotol 2002; 23: 34-38.
8) Olivetto E, Simoni E, Guaran V, Astolfi L, Martini AJHr. Sensorineural hearing loss and ischemic injury: Development of animal models to assess vascular and oxidative effects. Hear Res 2015; 327: 58-68.
9) Kim Y, Kim J-S, Kim G-WJSr. A novel frequency selectivity approach based on travelling wave propagation in mechanoluminescence basilar membrane for artificial cochlea. Sci Rep 2018; 8: 1-8.
10) Torre P 3rd, Cruickshanks KJ, Klein BE, Klein R, Nondahl DM. The association between cardiovascular disease and cochlear function in older adults. J Speech Lang Hear Res 2005; 48: 473-481.
11) Telischi FF, Stagner B, Widick MP, Balkany TJ, Lonsbury-Martin BLJTL. Distortion-Product Otoacoustic Emission Monitoring of Cochlear Blood Flow. Laryngoscope 1998; 108: 837-842.
12) Mom T, Telischi FF, Martin GK, Lonsbury-Martin BLJHr. Measuring the cochlear blood flow and distortion-product otoacoustic emissions during reversible cochlear ischemia: a rabbit model. Hear Res 1999; 133: 40-52.
13) Conen D, Rodondi N, Müller A, Beer JH, Ammann P, Moschovitis G, Auricchio A, Hayoz D,

Kobza R, Shah D, Novak J, Schläpfer J, Di Valentino M, Aeschbacher S, Blum S, Meyre P, Sticherling C, Bonati LH, Ehret G, Moutzouri E, Fischer U, Monsch AU, Stippich C, Wuerfel J, Sinnecker T, Coslovsky M, Schwenkglenks M, Kühne M, Osswald S; Swiss-AF Study Investigators. Relationships of overt and silent brain lesions with cognitive function in patients with atrial fibrillation. J Am Coll Cardiol 2019; 73: 989-999.

14) Malavasi VL, Zoccali C, Brandi MC, Micali G, Vitolo M, Imberti JF, Mussi C, Schnabel RB, Freedman B, Boriani G. Cognitive impairment in patients with atrial fibrillation: Implications for outcome in a cohort study. Int J Cardiol 2021; 323: 83-89.

15) Sepehri Shamloo A, Dagres N, Müssigbrodt A, Stauber A, Kircher S, Richter S, Dinov B, Bertagnolli L, Husser-Bollmann D, Bollmann A, Hindricks G, Arya A. Atrial fibrillation and cognitive impairment: new insights and future directions. Heart Lung Circ 2020; 29: 69-85.

16) Nakashima T, Naganawa S, Sone M, Tominaga M, Hayashi H, Yamamoto H, Liu X, Nuttall AL. Disorders of cochlear blood flow. Brain Res Brain Res Rev 2003; 43: 17-28.

17) Anselmino M, Scarsoglio S, Saglietto A, Gaita F, Ridolfi LJSr. Transient cerebral hypoperfusion and hypertensive events during atrial fibrillation: a plausible mechanism for cognitive impairment. Sci Rep 2016; 6: 1-8.

18) Wang WJ, Lessard D, Abu H, McManus DD, Mailhot T, Gurwitz JH, Goldberg RJ, Saczynski J. Hearing loss and cognitive decline among older adults with atrial fibrillation: the SAGE-AF study. J Geriatr Cardiol 2020; 17: 177.

19) Fanaei H, Pourbakht A, Jafarzadeh SJB, Neuroscience C. Bilateral carotid artery occlusion and cochlear oxidative stress and hearing loss in rats. Basic Clin Neurosci 2020; 11: 821.