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Investigation of the Effect of Allicin on Noise-Induced Hearing Loss in Rats

Yakup Şen[1], Canberk Kertmen[2], Serpil Mungan Durankaya[3], Osman Yılmaz[4], Günay Kırkım[3], Safiye Aktaş[5], Yüksel Olgun[6], and Abdullah Dalğıç[1,2]

[1]Department of Otorhinolaryngology/Head and Neck Surgery, İzmir Bozyaka Education and Research Hospital, University of Health Sciences Turkey, İzmir, Türkiye
[2]Department of Otorhinolaryngology, Head and Neck Surgery, University of Health Sciences Türkiye, İzmir City Hospital, İzmir, Türkiye
[3]Department of Otorhinolaryngology, Unit of Hearing Speech and Balance, Dokuz Eylül University Faculty of Medicine, İzmir, Türkiye
[4]Department of Laboratory Animals, Dokuz Eylül University Institute of Health Sciences, İzmir, Türkiye
[5]Department of Basic Oncology, Dokuz Eylul University Institute of Oncology, İzmir, Türkiye
[6]Department of Otorhinolaryngology, Dokuz Eylül University School of Medicine, İzmir, Türkiye

Background and Objectives: Noise-induced hearing loss (NIHL) is a major cause of sensorineural hearing loss. However, effective pharmacological treatments remain limited. The aim of this study was to investigate the therapeutic effects of allicin on NIHL in a rat model. **Materials and Methods**: Forty-two female Wistar albino rats were randomly divided into six groups (n=7 per group): saline (SF), allicin, dimethyl sulfoxide (DMSO), noise+SF, noise+allicin+DMSO, and noise+DMSO. Noise exposure was performed using 4 kHz octave-band noise at a sound pressure level (SPL) of 120 dB for 4 hours. Allicin (20 mg/kg/day, intraperitoneally) was administered for 7 days following noise exposure. Auditory brainstem response (ABR) thresholds (dB SPL) were measured at 8, 12, 16, 20, and 32 kHz at baseline, 24 hours, and day 7. Histopathological evaluation of the cochlea was performed after euthanasia. **Results**: Noise exposure significantly increased ABR thresholds in all noise-exposed groups. On day 7, the noise+allicin+DMSO group demonstrated significantly lower ABR thresholds at 16, 20, and 32 kHz compared with the noise+SF group and at 20 and 32 kHz compared with the noise+DMSO group ($p<0.05$). No significant differences were observed at lower frequencies. Histopathological analysis showed a trend toward reduced cochlear damage in the allicin-treated group; however, the differences were not statistically significant. **Conclusions**: Allicin treatment improved high-frequency hearing thresholds in rats with NIHL. However, the absence of significant histopathological and mechanistic findings indicates that further molecular and region-specific studies are required.

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Address for correspondence
Canberk Kertmen, MD
Department of Otorhinolaryngology, Head and Neck Surgery, University of Health Sciences Türkiye, İzmir City Hospital, İzmir 35000, Türkiye
Tel +90-5315041325
E-mail kertmencanberkk@hotmail.com

Introduction

Noise-induced hearing loss (NIHL) is one of the most common causes of acquired sensorineural hearing loss worldwide and remains a major public health problem, particularly in industrialized societies [1-4]. NIHL may occur acutely after a single exposure to intense noise or gradually as a result of chronic exposure, leading to permanent auditory threshold shifts, especially at high frequencies [3-6].

Experimental and clinical studies have demonstrated that exposure to noise levels exceeding 115 dB SPL can result in irreversible cochlear damage, particularly within the 3–6 kHz frequency range [3-6]. Auditory brainstem response (ABR) testing is a widely used, objective method for assessing auditory pathway integrity in both clinical and experimental settings [7].

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The pathophysiology of NIHL involves complex mechanisms, including mechanical damage to cochlear structures, vascular compromise, and metabolic disturbances. Intense noise exposure leads to excessive displacement of cochlear fluids, disruption of the basilar membrane, edema of the stria vascularis, impaired cochlear blood flow, and alterations in endocochlear potential [8]. One of the most critical mechanisms implicated in NIHL is the excessive production of reactive oxygen species (ROS), which induces oxidative stress, mitochondrial dysfunction, and subsequent apoptosis or necrosis of cochlear hair cells and spiral ganglion neurons [9-12].

Given the central role of oxidative stress and apoptosis in NIHL, numerous antioxidant and antiapoptotic agents have been investigated; however, no pharmacological treatment other than corticosteroids has demonstrated consistent efficacy in humans. Allicin, a sulfur-containing compound derived from garlic (Allium sativum), exhibits potent antioxidant, anti-inflammatory, and antiapoptotic properties [13-17]. Previous studies have shown that allicin can reduce ROS formation, inhibit caspase activation, and protect cells from oxidative stress–induced apoptosis in various experimental models [16,17].

Despite these promising biological effects, the role of allicin in NIHL has not been previously investigated. Therefore, the present study aimed to evaluate the potential protective and therapeutic effects of allicin in an experimental rat model of NIHL.

Material and Methods

This study was approved by Dokuz Eylul University Animal Experiments Local Ethics Committee (approval number: 05/2022, date: 05/03/2022).

Forty-two Wistar outbreeding animals, each weighing 150–200 g (young–adult) were used in the study. Forty-two female Wistar albino rats were obtained from the Experimental Animal Research Laboratory of Dokuz Eylül University Faculty of Medicine. During the study, they were kept at room temperature (20±2°C) under a 12-hour light/dark cycle, fed standard pellet rat food, and given free access to water. Before the study, rats were monitored for one week and their adaptation to the environment provided. All rats underwent otoscopic examination. Any debris or plugs in the external ear canal were removed, and the tympanic membrane was confirmed to have a normal appearance.

Rats were randomly divided into 6 groups.

Group 1: Control group, group given only saline (SF) (20 mL/kg/day) (n=7).

Group 2: Allicin (20 mg/kg/day ip) only group (n=7).

Group 3: Group given only dimethyl sulfoxide (DMSO) (20 mg/kg/day ip) (n=7).

Group 4: Noise+SF (20 mL/kg/day ip) given group (n=7).

Group 5: Noise+allicin (20 mg/kg/day ip)+DMSO given group (n=7).

Group 6: Noise+DMSO (20 mg/kg/day ip) group (n=7).

Experimental timeline

- Day -7 to 0: Acclimatization.
- Day 0: Baseline ABR measurement.
- Day 0: Noise exposure (Groups 4–6).
- Day 0–7: Allicin/DMSO/SF administration.
- Day 1: Post-noise ABR measurement.
- Day 7: Final ABR measurement.
- Day 7: Sacrifice and tissue collection.

Allicin was dissolved in DMSO and administered intraperitoneally at a dose of 20 mg/kg/day. Control groups received equivalent volumes of SF or DMSO to ensure consistency. The selected dose of allicin was based on previous experimental studies demonstrating antioxidant and antiapoptotic effects.

Noise exposure was performed in a sound-attenuated chamber. Animals were placed in individual cages and exposed simultaneously to a speaker positioned 2 cm above the cages. The noise stimulus (4-kHz octave band) was calibrated using a sound level meter before each experiment. All animals were awake during exposure (Fig. 1).

ABR recording

ABR measurements were performed under anesthesia using ketamine (50 mg/kg) and xylazine (10 mg/kg). Subdermal needle electrodes were placed at the vertex (active), mastoid (reference), and back (ground) (Fig. 2). Tone-burst stimuli at



Fig. 1. Rats were exposed to noise in a noise chamber in an attempt to induce noise-induced hearing loss.

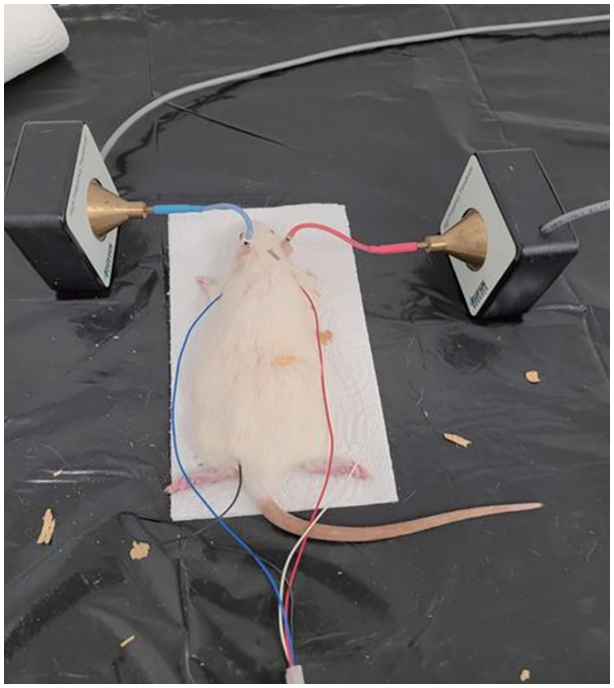


Fig. 2. Electrodes were attached to measure auditory evoked brainstem potentials in rats experiencing hearing loss.



Fig. 3. The rats were sacrificed before their temporal bones were resected.

8, 12, 16, 20, and 32 kHz were delivered. Responses were recorded using a band-pass filter (100–3,000 Hz), with a repetition rate of 21.1/s and 1,024 sweeps averaged per stimulus level. ABR thresholds were defined as the lowest stimulus intensity (dB SPL) at which a reproducible wave III response was

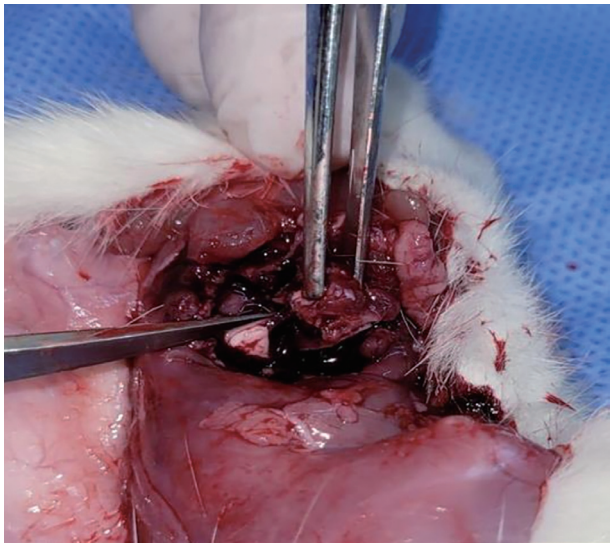


Fig. 4. After rats were sacrificed, their temporal bones were resected to examine their cochleas.

visually identified. Stimuli were presented in 5 dB steps.

Audiological examination

Intraperitoneal injection of 10 mg/kg xylazine and 50 mg/kg ketamine to all groups under basal hearing values in hand to be done for auditory evoked brainstem potential (AEBP) test was performed. Groups 4, 5, and 6 were exposed to 4 kHz octave band noise at 120 dB SPL for 4 hours. The test was repeated for all groups 1 day and 7 days after the noise. With the test, 8, 12, 16, 20, and 32 kHz were measured in each measurement. In frequencies, hearing thresholds were recorded.

Histological examination

All groups were sacrificed after the treatment was completed. The right and left temporal bones of all rats were sacrificed from the process immediately later dissected by being was removed (Figs. 3 and 4). The volume of 10 thick amount of 10% formalin solution. Fixed at room temperature for 48 hours. Tissue examples, 5 day 5% glacial acetic acid in solution decalcification was released.

The decalcification process, a section was taken and checked and placed in tap water for 1 hour, washed and cassetted and taken for routine paraffin tissue monitoring. Five of the prepared paraffin blocks micron sections by taking Hematoxylin - Eosin painting with histopathologically Striae were evaluated as histopathological evaluation criteria for ototoxicity. Vascularis, organ of corti, and spiral ganglion were determined. Striae were used as histopathological evaluation criteria for ototoxicity. Vascularis, organ of Corti and spiral ganglion were determined. The 4-point ototoxicity scoring system described by de Freitas, et al. [18] was used.

Statistical analysis

The AEBP results are presented as mean±standard deviation using descriptive statistics. For the analysis of differences between measurable data Kruskal–Wallis variance analysis was used to determine which group caused the difference. The Mann–Whitney U test was used for between-group comparisons. For within-group comparisons, the Wilcoxon signed-rank test with Bonferroni correction was used. Statistical analyses of histological data were performed using SPSS 22.0 program (IBM Corp.). Continuous variables were normally distributed. Kolmogorov–Smirnov and Shapiro–Wilk tests were used to determine whether continuous variables were not normally distributed between groups. Mann–Whitney U test (group number 2) was used for comparison and the results were presented as median (min-max) values. Categorical variables between groups in comparison chi-square and Fisher's exact test was used. Results were presented as numbers (percentages). In all analyses, the significance level was determined as 0.05. The primary endpoint was defined as the change in ABR threshold (dB SPL) at 20 kHz on day 7. Secondary endpoints included thresholds at other frequencies. A mixed-effects model was used to analyze repeated measurements over time and across frequencies. Post hoc comparisons were performed with Bonferroni correction. A p-value <0.05 was considered statistically significant.

Results

AEBP findings

According to the Kolmogorov-Smirnov normality test, the data was not normally distributed (p<0.05); therefore, non-parametric tests were preferred. In the Kruskal–Wallis variance analysis test, no significant difference was found in the baseline measurements between the groups. The baseline measurements of the 6 groups were similar to each other. No statistically significant difference was observed (p>0.05).

After noise was applied to the groups 4, 5, and 6, according to the 1st day results of all groups, there was a statistically significant difference between the groups in the Kruskal–Wallis test in the 1st day measurements (p<0.001).

According to these data, noise+SF, noise+allicin, it was statistically demonstrated that we created NIHL in the noise+DMSO groups the Kruskal–Wallis test, a statistically significant difference was found between the groups in terms of measurements on day 7. No statistically significant difference was observed between the hearing thresholds taken on day 7 and the thresholds taken at baseline and day 1 measurements of the groups that were given only SF, allicin, DMSO and did not receive noise (groups 1, 2, 3) (p>0.05).

Mann–Whitney U test with Bonferroni correction was performed to determine which group caused this difference. According to this test, in terms of 7th day measurements, no statistically significant difference was observed between the 7th day responses of the 5th (noise+allicin) and 6th (noise+DMSO) groups at 8, 12, 16 kHz after noise (p>0.05) (Table 1).

No statistically significant difference was observed between the responses of the 4th (noise+SF) and 5th (noise+allicin) groups at 8 and 12 kHz on the 7th day after noise (p>0.05). However, significant differences were obtained between the two groups at 16, 20, and 32 kHz. The responses of the 5th group were significantly better at 20, and 32 kHz (p<0.05) (Table 2). Seven days after noise ip, a statistically significant difference was obtained in the group receiving allicin treatment compared to the group receiving SF. These results proved that allicin treatment after noise has significant effects on hearing in rats, especially at frequencies of 20 kHz and above.

While the AEBP responses of the 1st, 2nd, and 3rd groups (SF, allicin, DMSO) were at similar levels to the basal and day 1 measurements, the threshold values of the 4th, 5th, and 6th groups (noise+SF, noise+allicin, noise+DMSO) increased compared to the basal measurements. The AEBP responses in the 5th group, the noise+allicin group, were significantly improved, especially at 20 kHz and 32 kHz, compared to the control groups (groups 4 and 5).

No statistically significant difference was observed between the 7th day responses of the 4th and 6th groups after noise (p>0.05). According to these results, no significant difference was observed in the group given DMSO after noise compared to the SF group. It was shown that DMSO treatment had no therapeutic effect on hearing loss.

Histological findings

The median damage scores for the organ of Corti, spiral gan-

Table 1. Auditory evoked brainstem potential values (dB SPL) obtained on the first day after noise in the 5th and 6th groups, their standard deviations and p-values

	Group 5	Group 6	p-value
8 kHz	83.93±11.47 (55–90)	78.21±10.67 (60–95)	0.114
12 kHz	79.29±13.28 (55–95)	72.50±11.73 (50–95)	0.137
16 kHz	76.79±13.24 (55–95)	69.29±11.74 (50–95)	0.077
20 kHz	76.07±12.12 (45–90)	69.29±13.99 (50–95)	0.137
32 kHz	82.50±11.73 (65–95)	72.14±13.69 (55–100)	0.056

Values are presented as mean±standard deviation (range).

Table 2. Auditory evoked brainstem potential values (dB SPL), standard deviations and *p*-values of the 5th and 6th groups taken on the 7th day after noise

	Group 5	Group 6	*p*-value
8 kHz	68.75±15.24 (40–85)	76.07±9.64 (60–95)	0.403
12 kHz	56.25±21.65 (15–80)	69.29±10.16 (55–95)	0.231
16 kHz	55.42±15.59 (20–70)	64.64±9.99 (55–95)	0.322
20 kHz	53.75±14.79 (25–65)	66.43±10.64 (50–95)	0.046
32 kHz	55.83±15.50 (20–75)	69.64±11.51 (60–100)	0.041

Values are presented as mean±standard deviation (range).

glion, and stria vascularis in Group 4 (noise+SF) and Group 5 (noise+allicin) are presented in Table 3. Morphologically, milder degrees of nuclear degeneration and outer hair cell loss were observed. Although signs of cytoplasmic vacuolization were present, no significant morphological changes were observed in the organ of Corti, spiral ganglion, or stria vascularis. No statistically significant difference was found between the groups in terms of vascular damage degrees (p=0.295, p=0.101, p=0.138, respectively) (Table 3).

The median damage scores of the organ of Corti, spiral ganglion, and stria vascularis for Group 5 (noise+allicin) and Group 6 (noise+DMSO) are presented in Table 4. Morphologically, there was a milder level of nuclear degeneration, outer hair cell loss, cytoplasmic. Although signs of vacuolization were seen, organ of Corti, spiral ganglion and stria, no statistically significant difference was found between the groups in terms of vascularis damage degrees (p=0.295, p=0.234, p=0.295, respectively). No morphological and statistically significant difference was observed between the 5th and 6th groups (p>0.05) (Table 4).

Table 3. Comparison of damage scores of group 5 and group 4

	Group 5	Group 4	*p*-value
Organ of Corti, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.295
Spiral ganglion, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.101
Stria vascularis, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.138

Histopathologically, the cases were categorized into four grades, and all statistical analyses were conducted according to this grading system.

Table 4. Comparison of damage scores of group 5 and group 6

	Group 5	Group 6	*p*-value
Organ of Corti, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.295
Spiral ganglion, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.234
Stria vascularis, median (min–max)	2.0 (2.0–3.0)	3.0 (2.0–3.0)	0.295

Histopathologically, the cases were categorized into four grades, and all statistical analyses were conducted according to this grading system.

Discussion

NIHL remains one of the most prevalent causes of acquired sensorineural hearing loss worldwide, and effective pharmacological treatment options are still limited. In the present study, we investigated the potential therapeutic effect of allicin, a biologically active sulfur-containing compound derived from garlic, in an experimental rat model of NIHL. The main finding of this study is that allicin administration after noise exposure significantly improved ABR thresholds, particularly at high frequencies (16, 20, and 32 kHz), compared with both SF- and DMSO-treated noise-exposed control groups.

The pathophysiology of NIHL is multifactorial and involves mechanical disruption of cochlear structures, vascular compromise, oxidative stress, and activation of apoptotic pathways [18-22]. Among these mechanisms, excessive generation of ROS following acoustic trauma plays a central role in cochlear hair cell and spiral ganglion neuron degeneration [9-12]. Increased ROS production leads to lipid peroxidation, mitochondrial dysfunction, impaired cochlear blood flow, and subsequent apoptosis or necrosis [19-22]. Therefore, antioxidant and antiapoptotic agents have been widely investigated as potential therapeutic strategies for NIHL.

Allicin has been shown in previous experimental studies to exert strong antioxidant and cytoprotective effects by reducing ROS formation, suppressing inflammatory cytokines, and inhibiting caspase-mediated apoptosis [13-17]. Wu, et al. [16] demonstrated that allicin protected cochlear hair cells and spiral ganglion neurons from cisplatin-induced apoptosis by modulating the Bcl-2/Bax pathway and reducing caspase-3 and caspase-9 activation. Similarly, Cai, et al. [17] reported that allicin significantly reduced stria vascularis damage and apoptotic activity in a cisplatin ototoxicity model. Although these studies focused on drug-induced ototoxicity, their findings support the hypothesis that allicin may also be beneficial in NIHL through similar biological mechanisms.

In the present study, allicin-treated rats demonstrated significantly improved ABR thresholds at high frequencies compared with both noise+SF and noise+DMSO groups. High-frequency regions of the cochlea are known to be more vulnerable

to noise-induced damage, particularly in the basal turn. The observed frequency-specific functional improvement is therefore consistent with the known tonotopic susceptibility of the cochlea to acoustic trauma. However, although histopathological evaluation showed a trend toward reduced spiral ganglion degeneration, less stria vascularis damage, and better preservation of the organ of Corti in the allicin-treated group, these differences did not reach statistical significance.

One important limitation of this study, as noted by the reviewers, is that histopathological analysis was not performed in a frequency-specific, tonotopically matched manner. While ABR findings indicated significant improvements at higher frequencies, cochlear sections were evaluated using conventional light microscopy without precise localization to basal, middle, or apical turns. Therefore, direct correlation between frequency-specific functional outcomes and region-matched histological changes could not be established. This limitation has now been explicitly acknowledged, and future studies using segmental cochlear analysis or electron microscopy are warranted to better elucidate region-specific protective effects of allicin.

Another important methodological consideration is the use of DMSO as a solvent for allicin due to its hydrophobic nature. To address this, a noise+DMSO control group was included. Consistent with previous studies demonstrating the otological safety of DMSO, no significant differences were observed between noise+DMSO and noise+SF groups in either ABR thresholds or histopathological findings [23]. These results indicate that the observed therapeutic effects were attributable to allicin itself rather than the solvent.

Despite the absence of statistically significant histopathological differences, the functional improvement observed in ABR measurements suggests that allicin may exert protective effects at the cellular or molecular level that are not fully captured by routine histological scoring. Early biochemical or mitochondrial protection, reduced oxidative stress, or partial preservation of synaptic integrity may account for these findings. Similar discrepancies between functional and morphological outcomes have been reported in previous NIHL studies [24].

To our knowledge, this is the first experimental study to evaluate the effects of allicin in a NIHL model. The findings suggest that allicin may represent a promising adjunctive therapeutic agent for NIHL due to its antioxidant and antiapoptotic properties. However, translation of these findings to clinical practice requires further investigation, including dose–response studies, molecular pathway analysis, and ultimately, well-designed clinical trials.

One important limitation of this study is that the observed functional improvement was limited to high frequencies without statistically significant histopathological confirmation. This may be explained by the greater vulnerability of the basal turn of the cochlea to noise exposure. However, cochlear sampling in this study was not performed in a tonotopically specific manner.

Additionally, no molecular or biochemical analyses were performed to evaluate oxidative stress or apoptotic pathways. Future studies should include assessment of oxidative stress markers (e.g., MDA, SOD), apoptotic markers (e.g., caspase-3), and quantitative hair cell analysis to better elucidate the underlying mechanisms.

Conclusion

Despite many studies, NIHL is a disease that has no successful and definitive treatment in humans. In this study, AEBP measurements demonstrated that allicin exerts a therapeutic effect on NIHL in rats. Our histopathological examinations further revealed that allicin treatment attenuated the histopathological changes induced by noise exposure. It has been shown that cochlea and spiral ganglion damage is less morphologically seen. Our study on allicin in NIHL is important because it is the first to demonstrate its effectiveness. However, the effect of allicin treatment on people with NIHL is investigated. Further studies are needed to elucidate the underlying mechanisms and confirm these findings. Although allicin improved ABR thresholds at high frequencies, the lack of consistent effects across all frequencies and absence of significant histopathological findings indicate that these results should be interpreted with caution.

Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization:Yakup Şen. Data curation: Canberk Kertmen. Formal analysis: Osman Yılmaz. Investigation: Serpil Mungan Durankaya. Methodology: Günay Kırkım. Project administration: Safiye Aktaş. Resources: Yüksel Olgun. Software: Abdullah Dalğıç. Supervision: Yakup Şen. Validation: Yakup Şen. Visualization: Osman Yılmaz. Writinig—original draft: Canberk Kertmen. Writing—review & editing: Canberk Kertmen. Approval of final manuscript: all authors.

ORCID iDs

Yakup Şen	https://orcid.org/0000-0002-4454-6190
Canberk Kertmen	https://orcid.org/0009-0005-5413-0139
Serpil Mungan Durankaya	https://orcid.org/0000-0003-4236-434x
Osman Yılmaz	https://orcid.org/0000-0001-7817-7576
Günay Kırkım	https://orcid.org/0000-0003-4170-5317
Safiye Aktaş	https://orcid.org/0000-0002-7658-5565
Yüksel Olgun	https://orcid.org/0000-0003-1769-4224
Abdullah Dalğıç	https://orcid.org/0000-0002-6958-4169

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