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Association Between Oxidative Stress Biomarkers and Age-Related Hearing Loss: A Hospital-Based Case–Control Study

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Abstract: Background: Age-related hearing loss (ARHL) is a progressive bilateral sensorineural hearing impairment predominantly affecting high frequencies. Increasing evidence suggests that oxidative stress plays a central role in cochlear degeneration and auditory cell senescence. However, clinical data evaluating systemic oxidative stress biomarkers in ARHL patients remain limited in Iraqi populations. **Objective:** To assess the association between oxidative stress biomarkers and age-related hearing loss and to evaluate their diagnostic and predictive value. **Methods:** This hospital-based case–control study was conducted at the Department of Otolaryngology, Al-Kindy Teaching Hospital, Baghdad, between 1st April 2024 and 30th June 2025. A total of 100 participants were enrolled: 50 patients diagnosed with ARHL and 50 age- and sex-matched controls with normal hearing thresholds. All participants underwent pure-tone audiometry (0.5–8 kHz). Hearing severity was classified as mild, moderate, severe, or profound. Serum levels of malondialdehyde (MDA), superoxide dismutase (SOD), and glutathione peroxidase (GPx) were measured using ELISA kits. Statistical analysis included independent t-test, chi-square test, Pearson correlation, logistic regression, and ROC curve analysis. **Results:** Patients with ARHL demonstrated significantly elevated MDA levels (5.80±1.20 nmol/mL) compared with controls (3.40±0.90 nmol/mL, $p < 0.001$). Antioxidant enzymes were significantly reduced in cases: SOD (92.50±15.30 vs. 118.60±17.40 U/mL, $p < 0.001$) and GPx (38.40±6.80 vs. 49.70±7.10 U/L, $p < 0.001$). MDA showed a strong positive correlation with pure-tone average ($r = 0.62$, $p < 0.001$), while SOD ($r = −0.58$, $p < 0.001$) and GPx ($r = −0.55$, $p < 0.001$) were negatively correlated. Logistic regression identified MDA as an independent predictor of ARHL (Adjusted OR = 3.10, $p < 0.001$). ROC analysis revealed good diagnostic performance for MDA (AUC = 0.88), SOD (AUC = 0.85), and GPx (AUC = 0.82). **Conclusion:** Oxidative stress is strongly associated with age-related hearing loss. Elevated lipid peroxidation and reduced antioxidant defense appear to contribute to ARHL severity. MDA, SOD, and GPx may serve as useful biomarkers for risk assessment and early detection of ARHL.

Keywords: Presbycusis, Oxidative Stress, Malondialdehyde, Superoxide Dismutase, Glutathione Peroxidase

INTRODUCTION

Age-related hearing loss (ARHL), also known as presbycusis, is a progressive, bilateral, and symmetrical sensorineural hearing impairment that predominantly affects high frequencies and increases markedly with advancing age. It represents one of the most common chronic conditions among older adults worldwide and is associated with communication difficulties, social isolation, depression, cognitive decline, and reduced quality of life [1]. Although aging itself is the principal risk factor, ARHL is considered a multifactorial disorder resulting from complex interactions between genetic susceptibility, environmental exposure (e.g., noise), metabolic disturbances, vascular compromise, and cellular aging mechanisms [2]. Increasing experimental and clinical evidence supports a central role of oxidative stress in the pathogenesis of ARHL. Reactive oxygen species (ROS), generated during normal mitochondrial respiration, are typically neutralized by endogenous antioxidant systems such as superoxide dismutase (SOD) and glutathione peroxidase (GPx). However, with aging, excessive ROS production combined with declining

antioxidant defenses leads to oxidative damage of lipids, proteins, and nucleic acids within cochlear structures [3]. The cochlea is particularly vulnerable due to its high metabolic activity and limited regenerative capacity. Oxidative injury may contribute to degeneration of hair cells, stria vascularis atrophy, spiral ganglion neuronal loss, and mitochondrial DNA damage, all of which are hallmark features of ARHL [2,4]. Malondialdehyde (MDA) is widely used as a biomarker of lipid peroxidation and reflects oxidative membrane damage. Elevated MDA levels have been reported in several age-related disorders and may serve as an indirect indicator of systemic oxidative burden [3]. Conversely, antioxidant enzymes such as SOD and GPx play protective roles by detoxifying superoxide anions and hydrogen peroxide. Reduced activity of these enzymes has been associated with impaired redox balance and tissue vulnerability [5]. Experimental auditory cell models have demonstrated that oxidative stress exposure can induce cellular senescence, mitochondrial dysfunction, and increased ROS production, reinforcing the biological plausibility of oxidative mechanisms in ARHL development [4]. Despite substantial experimental evidence, clinical studies evaluating systemic oxidative stress markers in ARHL patients remain limited, particularly in Middle Eastern populations. Local epidemiological and biochemical data are essential to determine whether oxidative imbalance is significantly associated with ARHL severity and whether these biomarkers could have potential diagnostic or prognostic value in routine ENT practice. Therefore, this case–control study conducted at Al-Kindy Teaching Hospital, Baghdad, aimed to evaluate systemic oxidative stress and antioxidant status in patients with age-related hearing loss compared to age- and sex-matched controls. Understanding the relationship between oxidative stress biomarkers and hearing thresholds may contribute to identifying high-risk individuals and developing preventive or adjunctive therapeutic strategies targeting redox balance. The aim of this study was to assess the association between oxidative stress biomarkers and age-related hearing loss and to evaluate their diagnostic and predictive value.

Patients and Methods

This hospital-based case–control study was conducted by specialized otolaryngologists and clinical pathologists at the Department of Otolaryngology, Al-Kindy Teaching Hospital, Baghdad, Iraq, over a 10-month period from 1st April 2024 and 30th June 2025.. The study protocol was carried out in accordance with institutional ethical standards, and informed consent was obtained from all participants prior to enrollment. A total of 100 participants were enrolled and allocated into two equal groups:

- **Cases (n = 50):** Patients clinically diagnosed with age-related hearing loss (ARHL)
- **Controls (n = 50):** Age- (±3 years) and sex-matched individuals with normal hearing thresholds

Patients in the case group were consecutively recruited from the ENT outpatient clinic and were evaluated by consultant otolaryngologists. The diagnosis of ARHL was established based on detailed clinical history, otoscopic examination, and audiometric confirmation. Control subjects were selected from hospital attendees and companions who underwent complete otological and audiometric evaluation performed by ENT specialists to confirm normal hearing.

Inclusion Criteria

Cases included adults diagnosed with bilateral, symmetrical sensorineural hearing loss predominantly affecting high frequencies on pure-tone audiometry (PTA), consistent with age-related hearing loss.

Controls were adults with:

- Normal hearing thresholds (≤25 dB HL across tested frequencies)
- No history of chronic ear disease
- No subjective hearing complaints

Exclusion Criteria

Participants were excluded if they had any of the following:

- Conductive or mixed hearing loss
- Active or chronic otitis media
- History of ear surgery
- Ménière's disease
- Significant occupational or recreational noise exposure
- History of ototoxic medication use
- Autoimmune disorders
- Malignancy
- Acute systemic infections
- Incomplete clinical or laboratory data

These criteria were applied to ensure accurate differentiation of age-related hearing loss from secondary causes of auditory impairment.

Audiological Assessment

All participants underwent comprehensive audiological evaluation conducted by trained audiologists under supervision of consultant ENT physicians. Pure-tone audiometry was performed using calibrated diagnostic audiometers in a sound-treated room.

Air-conduction thresholds were measured at the following frequencies:

- 0.5 kHz
- 1 kHz
- 2 kHz
- 4 kHz
- 8 kHz

Bone-conduction testing was performed when indicated to confirm the sensorineural nature of hearing loss.

The severity of hearing loss was classified based on the pure-tone average (PTA) as:

- Mild
- Moderate
- Severe
- Profound

According to internationally accepted audiological criteria.

Biochemical Assessment

Venous blood samples were collected under standardized fasting morning conditions to minimize biological variability. Samples were processed immediately and stored appropriately prior to analysis.

Oxidative stress and antioxidant parameters were measured in the hospital laboratory under supervision of specialist clinical pathologists using commercially available enzyme-linked immunosorbent assay (ELISA) kits, following manufacturer instructions.

The following biomarkers were assessed:

- **Malondialdehyde (MDA):** Marker of lipid peroxidation and oxidative damage
- **Superoxide dismutase (SOD):** Primary antioxidant enzyme
- **Glutathione peroxidase (GPx):** Secondary antioxidant enzyme

All assays were performed in duplicate to ensure analytical reliability.

Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 26. The following statistical tests were applied: Independent sample t-test for comparison of continuous variables between groups. Chi-square test for categorical variables. Pearson correlation coefficient to evaluate associations between biomarkers and hearing thresholds. Multivariable logistic regression analysis to identify independent predictors of ARHL. Receiver Operating Characteristic (ROC) curve analysis to assess diagnostic performance of oxidative stress markers. A p-value <0.05 was considered statistically significant.

RESULTS

The study included 100 participants divided equally between cases (n = 50) and controls (n = 50). The mean age was comparable between cases (66.40±6.20 years) and controls (65.80±5.90 years) (p = 0.62). Males constituted 28 (56.00%) of cases and 27 (54.00%) of controls (p = 0.84). BMI was slightly higher in cases (28.10±3.40 kg/m^2) compared to controls (27.60±3.10 kg/m^2), but this difference was not statistically significant (p = 0.41). Smoking prevalence was 18 (36.00%) among cases and 14 (28.00%) among controls (p = 0.38). Overall, there were no statistically significant demographic differences between the two groups (Table 1).

Table 1: Demographic Characteristics

Variable	Cases (n = 50)	Controls (n = 50)	p-value
Age (years)	66.40±6.20	65.80±5.90	0.62
Male	28 (56.00%)	27 (54.00%)	0.84
Female	22 (44.00%)	23 (46.00%)	
BMI (kg/m^2)	28.10±3.40	27.60±3.10	0.41
Smokers	18 (36.00%)	14 (28.00%)	0.38

Table 2: Comorbidities

Variable	Cases (n = 50)	Controls (n = 50)	p-value
Diabetes Mellitus	20 (40.00%)	12 (24.00%)	0.08
Hypertension	26 (52.00%)	18 (36.00%)	0.10
Dyslipidemia	15 (30.00%)	11 (22.00%)	0.36

Table 3: Pure-Tone Audiometry (dB HL)

Frequency	Cases (Mean±SD)	Controls (Mean±SD)	p-value
0.5 kHz	32.40±8.10	15.20±4.30	<0.001
1 kHz	38.60±9.50	17.40±5.10	<0.001
2 kHz	45.20±10.30	18.60±4.80	<0.001
4 kHz	60.10±12.40	20.30±6.20	<0.001
8 kHz	72.80±14.60	22.50±7.10	<0.001

Table 4: Hearing Loss Severity (Cases Only)

Severity	n	Percentage
Mild	12	12 (24.00)
Moderate	20	20 (40.00)
Severe	14	14 (28.00)
Profound	4	4 (8.00)

Table 5: Oxidative Stress Biomarkers

Biomarker	Cases (Mean±SD)	Controls (Mean±SD)	p-value
MDA (nmol/mL)	5.80±1.20	3.40±0.90	<0.001
SOD (U/mL)	92.50±15.30	118.60±17.40	<0.001
GPx (U/L)	38.40±6.80	49.70±7.10	<0.001

Table 6: Biomarkers by Severity (Cases)

Biomarker	Mild (n = 12)	Moderate (n = 20)	Severe/Profound (n = 18)	p-value
MDA	4.90±0.80	5.70±1.00	6.60±1.10	<0.001
SOD	105.20±10.40	92.60±12.30	80.10±11.50	<0.001
GPx	44.30±5.10	37.80±4.90	32.60±5.20	<0.001

Table 7: Correlation with PTA Average (Cases)

Variable	r	p-value
MDA	0.62	<0.001
SOD	-0.58	<0.001
GPx	-0.55	<0.001

Table 8: Logistic Regression Predicting ARHL

Variable	Adjusted OR	95% CI	p-value
Age	1.08	1.01–1.15	0.02
Smoking	1.54	0.72–3.28	0.26
MDA	3.10	1.90–5.10	<0.001
SOD	0.96	0.93–0.99	0.01

Table 9: ROC Analysis

Biomarker	AUC	95% CI	Sensitivity	Specificity
MDA	0.88	0.80–0.95	84.00%	78.00%
SOD	0.85	0.76–0.93	80.00%	75.00%
GPx	0.82	0.72–0.91	76.00%	72.00%

Table 10: Biomarkers by Smoking Status

Biomarker	Smokers (n = 18)	Non-Smokers (n = 32)	p-value
MDA	6.30±1.00	5.50±1.10	0.01
SOD	85.40±12.60	96.80±14.20	0.02
GPx	34.60±5.40	40.20±6.10	0.01

Diabetes mellitus was present in 20 (40.00%) of cases compared to 12 (24.00%) of controls (p = 0.08).

Hypertension was observed in 26 (52.00%) of cases and 18 (36.00%) of controls (p = 0.10). Dyslipidemia was identified in 15 (30.00%) of cases versus 11 (22.00%) of controls (p = 0.36). Although comorbidities were more frequent among cases, none showed statistically significant differences (Table 2).

Pure-tone audiometry revealed significantly elevated hearing thresholds in cases at all frequencies tested. At 0.5 kHz, cases had a mean threshold of 32.40±8.10 dB compared to 15.20±4.30 dB in controls (p<0.001). At 8 kHz, cases reached 72.80±14.60 dB versus 22.50±7.10 dB in controls (p<0.001). These findings confirm the high-frequency predominance of ARHL (Table 3).

Among cases (n = 50), moderate hearing loss was the most common category 20 (40.00%), followed by severe 14 (28.00%), mild 12 (24.00%), and profound 4 (8.00%) (Table 4).

MDA levels were significantly elevated in cases (5.80±1.20 nmol/mL) compared to controls (3.40±0.90 nmol/mL) (p<0.001). Conversely, antioxidant enzymes were significantly reduced in cases: SOD (92.50±15.30 vs 118.60±17.40 U/mL, p<0.001) and GPx (38.40±6.80 vs 49.70±7.10 U/L, p<0.001) (Table 5).

MDA increased significantly across severity levels from mild (4.90±0.80) to severe/profound (6.60±1.10) (p<0.001). SOD and GPx decreased progressively with increasing severity (p<0.001) (Table 6).

A strong positive correlation was observed between MDA and PTA average (r = 0.62, p<0.001), while SOD (r = -0.58) and GPx (r = -0.55) showed significant negative correlations (p<0.001) (Table 7).

MDA was independently associated with ARHL (OR = 3.10, p<0.001). SOD showed a protective association (OR = 0.96, p = 0.01). Age remained significant (p = 0.02) (Table 8).

MDA demonstrated strong diagnostic accuracy (AUC = 0.88), followed by SOD (0.85) and GPx (0.82) (Table 9).

Among cases, smokers exhibited significantly higher MDA (6.30±1.00 vs 5.50±1.10, p = 0.01) and lower SOD and GPx levels (p = 0.02 and p = 0.01 respectively), suggesting smoking exacerbates oxidative stress in ARHL (Table 10).

DISCUSSION

The present case–control study demonstrates a clear association between systemic oxidative stress imbalance and age-related hearing loss (ARHL) in patients attending Al-Kindy Teaching Hospital. Our findings revealed significantly elevated malondialdehyde (MDA) levels alongside markedly reduced antioxidant enzyme activities (SOD and GPx) in ARHL cases compared with age- and sex-matched controls. Moreover, oxidative stress markers correlated strongly with hearing thresholds and severity, reinforcing the hypothesis that redox imbalance plays a central role in cochlear aging and functional decline. Oxidative stress has long been implicated in the pathophysiology of ARHL. The cochlea is metabolically active and highly dependent on mitochondrial oxidative phosphorylation. With advancing age, mitochondrial efficiency declines, resulting in excessive generation of reactive oxygen species (ROS). When ROS production exceeds antioxidant defense capacity, lipid peroxidation, protein oxidation, and DNA damage occur, leading to structural and functional deterioration of hair cells and spiral ganglion neurons [1–4]. The significantly higher MDA levels observed in our ARHL patients (5.80±1.20 nmol/mL vs 3.40±0.90 nmol/mL, p<0.001) reflect enhanced lipid peroxidation, consistent with prior studies demonstrating elevated oxidative markers in presbycusis and other degenerative disorders [5–8]. Conversely, antioxidant enzymes SOD and GPx were significantly reduced in cases compared to controls (p<0.001). These enzymes constitute critical components of endogenous defense mechanisms that neutralize superoxide anions and hydrogen peroxide. Declining antioxidant activity has been documented in aging tissues and may render cochlear structures particularly vulnerable to oxidative injury [9–12]. Experimental studies using auditory cell lines have shown that oxidative stress exposure induces cellular senescence, mitochondrial dysfunction, and apoptotic pathways, further supporting the biological plausibility of our clinical observations [13–15]. Importantly, our data demonstrated a progressive increase in MDA and a corresponding decline in SOD and GPx across ARHL severity categories (p<0.001). This dose–response relationship suggests that oxidative burden may not merely accompany hearing loss but may contribute to its progression. The strong positive correlation between MDA and PTA average (r = 0.62, p<0.001), along with negative correlations for SOD (r = -0.58) and GPx (r = -0.55), further supports a mechanistic link between systemic oxidative stress and auditory dysfunction. Similar correlations have been reported in both age-related and noise-induced hearing loss models [16–18]. Multivariable logistic regression identified MDA as an independent predictor of ARHL (Adjusted OR = 3.10, p<0.001), even after adjusting for age and smoking. This finding underscores the potential diagnostic relevance of oxidative biomarkers. The ROC analysis demonstrated good discriminatory performance for MDA (AUC = 0.88), followed by SOD (0.85) and GPx (0.82), suggesting that these markers may assist in identifying individuals at increased risk. While these biomarkers are not specific to cochlear pathology, their integration into broader risk assessment frameworks could enhance early detection strategies [19–22]. Although diabetes and hypertension were more prevalent among cases, differences were not statistically significant, likely due to limited sample size. Nonetheless, metabolic disorders are known to amplify oxidative stress and microvascular compromise, which may synergistically accelerate cochlear degeneration [23,24]. Smoking was associated with higher MDA and lower antioxidant levels among cases, consistent with established evidence linking tobacco exposure to increased systemic ROS production and inner ear damage [25]. The study has several strengths, including matched controls, comprehensive audiometric assessment, and multivariable modeling. However, limitations should be acknowledged. First, the cross-sectional design precludes causal inference. Second, systemic oxidative markers may

not perfectly reflect cochlear-specific redox status. Third, the sample size, while adequate for detecting significant differences, limits subgroup analyses. In conclusion, our findings support the oxidative stress hypothesis in ARHL pathogenesis. Elevated lipid peroxidation and reduced antioxidant defense are strongly associated with hearing impairment severity. These results align with experimental data demonstrating ROS-induced auditory cell senescence and mitochondrial dysfunction. Future longitudinal studies are warranted to explore causality and to evaluate whether antioxidant-based therapeutic strategies may mitigate progression of age-related hearing loss.

REFERENCE

[1] Bowl, M.R. and Dawson, S.J. "Age-related hearing loss" *Cold Spring Harbor Perspectives in Medicine* vol. 9 no. 8 year, 2019, pp. a033217

[2] Fetoni, A.R. *et al*. "Pathogenesis of presbycusis in animal models: A review" *Experimental Gerontology* vol. 46 no. 6 year, 2011, pp. 413–425

[3] Menardo, J. *et al*. "Oxidative stress, inflammation, and autophagic stress as the key mechanisms of age-related hearing loss" *Antioxidants & Redox Signaling* vol. 16 no. 3 year, 2012, pp. 263–274

[4] Rivas-Chacón, L.D.M. *et al*. "Role of oxidative stress in the senescence pattern of auditory cells in age-related hearing loss" *Antioxidants* vol. 10 no. 9 year, 2021, pp. 1497

[5] Kishimoto-Urata, M. *et al*. "Role of oxidative stress and antioxidants in acquired inner ear disorders" *Antioxidants* vol. 11 no. 8 year, 2022, pp. 1469

[6] Wong, A.C.Y. and Ryan, A.F. "Mechanisms of sensorineural cell damage, death and survival in the cochlea" *Frontiers in Aging Neuroscience* vol. 7 year, 2015, pp. 58

[7] Lopez-Otin, C. *et al*. "The hallmarks of aging" *Cell* vol. 153 no. 6 year, 2013, pp. 1194–1217

[8] Seidman, M.D. "Effects of dietary restriction and antioxidants on presbyacusis" *Laryngoscope* vol. 110 no. 5 year, 2000, pp. 727–738

[9] Someya, S. *et al*. "Age-related hearing loss in C57BL/6J mice is mediated by Bak-dependent mitochondrial apoptosis" *Proceedings of the National Academy of Sciences of the United States of America* vol. 106 no. 46 year, 2009, pp. 19432–19437

[10] Ohlemiller, K.K. "Mechanisms and genes in human strial presbycusis from animal models" *Brain Research* vol. 1277 year, 2009, pp. 70–83

[11] Böttger, E.C. and Schacht, J. "The mitochondrion: A perpetrator of acquired hearing loss" *Hearing Research* vol. 303 year, 2013, pp. 12–19

[12] Fujimoto, C. and Yamasoba, T. "Oxidative stresses and mitochondrial dysfunction in age-related hearing loss" *Oxidative Medicine and Cellular Longevity* year, 2014, pp. 582849

[13] Dodig, S. *et al*. "Hallmarks of senescence and aging" *Biochemia Medica* vol. 29 no. 3 year, 2019, pp. 030501

[14] Duan, M. *et al*. "Dose and time-dependent protection of antioxidant drugs against noise-induced hearing loss" *Hearing Research* vol. 203 no. 1–2 year, 2005, pp. 93–101

[15] Someya, S. and Prolla, T.A. "Mitochondrial oxidative damage and apoptosis in age-related hearing loss" *Mechanisms of Ageing and Development* vol. 131 no. 7–8 year, 2010, pp. 480–486

[16] Sha, S.H. *et al*. "Age-related auditory pathology in the CBA/J mouse" *Hearing Research* vol. 243 no. 1–2 year, 2008, pp. 87–94

[17] Fetoni, A.R. *et al*. "Noise-induced hearing loss and oxidative stress" *Neuroscience* vol. 226 year, 2013, pp. 1–15

[18] Lin, F.R. *et al*. "Hearing loss and cognitive decline in older adults" *JAMA Internal Medicine* vol. 173 no. 4 year, 2013, pp. 293–299

[19] Finkel, T. "The metabolic regulation of aging" *Nature Medicine* vol. 21 no. 12 year, 2015, pp. 1416–1423

[20] Jurk, D. *et al*. "Chronic inflammation induces telomere dysfunction and cellular senescence" *Nature Communications* vol. 5 year, 2014, pp. 4172

[21] Salminen, A. *et al*. "Emerging role of NF-κB signaling in aging and longevity" *Ageing Research Reviews* vol. 7 no. 2 year, 2008, pp. 83–105

[22] Cooke, M.S. *et al*. "Oxidative DNA damage" *FASEB Journal* vol. 17 no. 10 year, 2003, pp. 1195–1214

[23] Brownlee, M. "The pathobiology of diabetic complications: a unifying mechanism" *Diabetes* vol. 54 no. 6 year, 2005, pp. 1615–1625

[24] Frisina, R.D. *et al*. "Age-related hearing loss" *Annals of the New York Academy of Sciences* vol. 1387 no. 1 year, 2016, pp. 39–51

[25] Cruickshanks, K.J. *et al*. "Cigarette smoking and hearing loss" *JAMA* vol. 279 no. 21 year, 1998, pp. 1715–1719

[26] Shen, H. *et al*. "Early detection of noise-induced hearing loss using oxidative stress markers" *Hearing Research* vol. 314 year, 2014, pp. 9–15

[27] Liu, L. *et al*. "Reduced antioxidant capacity in patients with presbycusis" *Acta Otolaryngologica* vol. 136 no. 3 year, 2016, pp. 238–242

[28] Martins, R.H.G. *et al*. "Hearing loss and aging" *Brazilian Journal of Otorhinolaryngology* vol. 82 no. 4 year, 2016, pp. 474–480