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Research Article

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Elevated Oxidative Stress and Impaired Antioxidant Defense in Idiopathic Sudden Sensorineural Hearing Loss: A Case–Control Study

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Abstract

Objective: Sudden sensorineural hearing loss (SSNHL) is an acute otologic emergency that can profoundly affect quality of life. Although various mechanisms, including viral, vascular, and immune-mediated pathways, have been proposed, most cases remain idiopathic. Oxidative stress has been suggested as a potential contributor by disrupting the balance between reactive oxygen species (ROS) and antioxidant defences within the cochlea. This study aimed to investigate serum oxidative stress markers and antioxidant enzyme activities in patients with idiopathic SSNHL compared with healthy controls.

Materials and methods: This case–control study included 30 patients diagnosed with idiopathic SSNHL and 30 age- and sex-matched healthy volunteers. SSNHL was defined as a ≥30 dB loss over at least three contiguous frequencies within 72 hours, with secondary causes excluded. Blood samples were collected at presentation before treatment initiation. Serum malondialdehyde (MDA) levels and antioxidant parameters, superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH), were measured using commercial spectrophotometric assay kits. Between-group comparisons were performed using appropriate statistical tests.

Results: Patients with SSNHL exhibited significantly elevated oxidative stress. Mean serum MDA levels were 1.66±0.29 µmol/L in the SSNHL group versus 0.69±0.09 µmol/L in controls ($p<0.001$). Antioxidant enzyme activities were markedly reduced in patients with SSNHL: SOD (1.63±0.73 vs. 6.85±0.85 U/mL), Gpx (0.06±0.04 vs. 0.21±0.006 U/mL), CAT (0.09±0.13 vs. 0.29±0.09 U/L), and GSH ($2\times10^{-5}\pm1\times10^{-5}$ vs. $1\times10^{-4}\pm5\times10^{-6}$ U/L) were all significantly lower ($p<0.001$ for all comparisons).

Conclusion: Idiopathic SSNHL is associated with heightened oxidative stress and a significant reduction in key antioxidant defences. These findings support the role of oxidative injury in SSNHL pathogenesis and suggest that therapies targeting redox imbalance, such as antioxidant adjunctive treatments, may offer potential benefits alongside standard management.

Keywords Sudden sensorineural hearing loss · Oxidative stress · Antioxidant enzymes · Malondialdehyde · Superoxide dismutase · Hearing loss

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INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency characterised by a rapid onset of hearing impairment, typically defined as a loss of at least 30 dB in three consecutive frequencies occurring within 72 hours (1). It affects approximately 5–20 per 100,000 persons annually (2). SSNHL can lead to significant disability, impacting social interactions and quality of life (3). Despite its clinical importance, the cause of SSNHL is identified in only 10%–15% of cases; the vast majority are termed *idiopathic* because no definite aetiology is confirmed (4).

Multiple hypotheses have been proposed for idiopathic SSNHL. Viral infections, autoimmune inner ear disease, and especially vascular ischaemia of the cochlea are major contributors (5). For instance, a viral insult or reactivation (such as herpes virus) could damage the inner ear, or a sudden compromise of the blood supply (e.g. labyrinthine artery spasm or thrombosis) could induce cochlear injury (6). Immune-mediated mechanisms and other factors (trauma and metabolic disturbances) have also been implicated, but collectively these factors do not fully explain all cases (7). These multifactorial hypotheses highlight the complexity of SSNHL and the need for further research into its underlying mechanisms.

One mechanism of interest in SSNHL is oxidative stress, which refers to an imbalance between the production of reactive oxygen species (ROS) and the capacity of antioxidant systems to neutralise them; when occurring in the cochlea, this can injure hair cells, neurons, and microvasculature (8). Under normal conditions, the inner ear, as in other tissues, maintains redox homeostasis through endogenous and exogenous antioxidants that continuously scavenge ROS produced by cellular metabolism (9). Experimental evidence demonstrates that ROS can directly alter the morphology and viability of cochlear hair cells (10). Moreover, an imbalance favouring oxidation can disrupt the vascular endothelium, which in the inner ear could worsen cochlear ischaemia or permeability, compounding hearing injury (11).

Oxidative stress has been implicated in many forms of hearing loss. For example, noise-induced and age-related hearing loss, as well as other otologic disorders such as noise trauma and ototoxic drug injury, have all been linked to ROS-mediated cochlear damage (12). These findings suggest that different cochlear insults, including noise, toxins, and ageing, may converge on ROS overproduction and subsequent hair cell death.

Emerging clinical evidence suggests that oxidative stress may contribute to idiopathic SSNHL (13). Recent studies have demonstrated impaired oxidant and antioxidant balance in affected patients, including alterations in thiol disulphide homeostasis and increased overall oxidative status compared with healthy controls (14, 15). Collectively, these findings support the hypothesis that oxidative injury may be involved in SSNHL pathogenesis (13-16).

Given this background, we hypothesised that idiopathic SSNHL is associated with heightened oxidative stress and reduced antioxidant defence. To test this, we designed a study to measure serum markers of oxidative stress in patients with SSNHL at presentation. Malondialdehyde (MDA) was chosen as a key indicator of oxidative stress, as it is a reactive by-product of lipid peroxidation when ROS attack polyunsaturated fatty acids in cell membranes. Elevated MDA reflects increased membrane oxidative damage (17, 18). Concurrently, we assessed the major components of the antioxidant defence system: superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and reduced glutathione (GSH). These represent enzymatic and non-enzymatic antioxidants that normally protect cochlear cells from ROS injury. SOD catalyses the dismutation of superoxide radicals into hydrogen peroxide, CAT converts hydrogen peroxide into water and oxygen, GPx reduces peroxides (using GSH as a cofactor), and GSH is a ubiquitous ROS scavenger (9, 12, 19, 20).

By comparing these markers in patients with SSNHL versus healthy controls, we aimed to clarify whether an oxidative stress imbalance is present in idiopathic SSNHL and to what extent. Ultimately, demonstrating such an imbalance would support the concept that oxidative stress plays a role in SSNHL and could open avenues for antioxidant-based adjunct therapies.

MATERIALS AND METHODS

This study was approved by the Clinical Research Ethics Committee of Yüzüncü Yıl University Faculty of Medicine (Decision No. 07, Date: 27.10.2015). All procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki, and written informed consent was obtained from all participants.

Study design and participants

This was a prospective case–control study. We enrolled 30 patients diagnosed with idiopathic SSNHL who presented to the Ear-Nose-Throat (ENT) department of a tertiary medical centre. All patients met the clinical definition of SSNHL (rapid-onset sensorineural hearing loss ≥30 dB over at least three contiguous frequencies within 72 hours). All patients underwent a standard etiologic evaluation before being classified as idiopathic, including detailed medical history, physical examination, assessment for recent trauma and ototoxic drug exposure, magnetic resonance imaging to exclude retrocochlear pathology, and targeted laboratory testing for infectious or autoimmune aetiologies when clinically indicated. Bilateral SSNHL was defined as simultaneous sensorineural hearing loss involving both ears within the same 72-hour episode. Because bilateral SSNHL has a broader differential diagnosis than unilateral disease, these cases were reviewed with particular scrutiny for secondary causes within the same diagnostic framework. Only patients in whom no identifiable cause was found after this evaluation were classified as having idiopathic SSNHL. Patients were excluded if they had an

established cause of hearing loss or significant comorbid conditions that could independently influence oxidative stress parameters. Specifically, individuals with uncontrolled hypertension, diabetes mellitus, thyroid disorders, active infections, chronic inflammatory diseases, or malignancy were excluded. Patients who had received antioxidant supplements or corticosteroid treatment before blood sampling were also excluded to avoid confounding effects.

A control group of 30 healthy volunteers was recruited, frequency-matched to the patient group by age and sex. Controls had no history of hearing loss or ear disease and no chronic systemic illnesses (such as diabetes, cardiovascular disease, or chronic inflammatory conditions). They were confirmed to have normal hearing on audiometric screening. The control subjects were selected to be as similar as possible in baseline characteristics (other than hearing loss) to the patient group. The mean age of participants in both groups was in the mid-40s (range 18–80 years in the SSNHL group, with a similar range in controls), and the patient group was 63% male (19/30), which was matched in the control group.

Clinical data collection

For each SSNHL patient, baseline demographic and clinical data were recorded, including age, sex, affected ear(s), and severity of hearing loss. Hearing loss severity was categorised by pure-tone audiometry at diagnosis: thresholds were classified as mild (20–39 dB HL), moderate (40–69 dB), severe (70–89 dB), or profound/total (≥90 dB) based on the average hearing level across four frequencies. Among the 30 patients, hearing loss ranged from mild to profound, with approximately one-third presenting with profound losses. The presence of accompanying symptoms, such as tinnitus or vertigo, was also noted. These clinical features were documented to allow correlation analyses with biochemical markers. All patients received oral prednisolone as first-line treatment at a dose of 1 mg/kg/day with gradual tapering over 14 days. Treatment was initiated immediately after diagnosis and blood sampling. In addition, 26 of the 30 patients underwent adjunctive hyperbaric oxygen therapy (HBOT) and completed at least 10 sessions, whereas 4 patients could not receive HBOT because of systemic contraindications. Hearing outcomes were assessed 1 month after completion of treatment using Siegel's criteria.

Biochemical measurements

Fasting blood samples were collected from each subject (patients and controls) in the morning to minimise diurnal variation. Importantly, patient samples were obtained before starting any treatment (e.g., before steroid or hyperbaric oxygen therapy) to reflect the acute untreated state of SSNHL. Venous blood was drawn into plain tubes, allowed to clot, and then centrifuged to isolate the serum. The serum samples were immediately frozen at −80°C until analysis.

Serum oxidative stress and antioxidant parameters were measured using commercial spectrophotometric assay kits according to the manufacturers' instructions. Serum MDA levels were measured using the Lipid Peroxidation MDA Assay Kit (Elabscience, Houston, TX, USA; Cat. No. E-BC-K025-M) at 532 nm. SOD activity was determined using the SOD Assay Kit (Cayman Chemical, Ann Arbour, MI, USA; Cat. No. 706002) at 450 nm. CAT activity was measured using the CAT Assay Kit (Cayman Chemical, Ann Arbour, MI, USA; Cat. No. 707002) at 540 nm. GPx activity was assessed using the GPx Assay Kit (Cayman Chemical, Ann Arbour, MI, USA; Cat. No. 703102) at 340 nm. GSH levels were measured using the GSH Assay Kit (Cayman Chemical, Ann Arbour, MI, USA; Cat. No. 703002) at 405 nm. Absorbance measurements were obtained using a BioTek Epoch 2 microplate spectrophotometer (BioTek Instruments, Winooski, VT, USA). All assays were performed in duplicate by laboratory personnel blinded to group allocation, and the mean of the 2 measurements was used for statistical analysis. Quality control procedures were conducted in accordance with the manufacturers' recommendations, including calibration with assay standards and internal controls. Samples showing an approximate difference greater than 10% between duplicate measurements were reanalysed according to the laboratory's standard operating procedure.

Routine laboratory parameters, including complete blood count and fasting blood glucose, were also evaluated to exclude overt infection or metabolic abnormalities that could confound oxidative stress measurements.

Statistical analysis

Data analysis was conducted using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD) or median (with range) as appropriate. We first tested all quantitative data for normality using the Shapiro–Wilk test. For comparisons between the SSNHL and control groups, an independent samples *t*-test was used for normally distributed variables, whereas the Mann–Whitney *U* test was used for non-normally distributed variables. Categorical data (e.g., sex distribution and symptom presence) were compared by chi-square test. A *p*-value <0.05 was considered statistically significant for all analyses. We also explored correlations between oxidative stress markers and clinical features (such as initial hearing loss severity and recovery outcome) using Spearman or Pearson correlation coefficients as appropriate. However, given the sample size, these secondary analyses were exploratory. As a sensitivity analysis, the primary between-group comparisons were repeated after exclusion of patients with bilateral SSNHL. The results are presented with their corresponding *p*-values and 95% confidence intervals where relevant.

RESULTS

Participant characteristics

A total of 30 patients with idiopathic SSNHL and 30 healthy controls were included. The groups were comparable in age and sex distribution (both p>0.05), and baseline clinical and laboratory parameters did not differ significantly. Details of the affected ear, accompanying symptoms, and initial hearing loss severity are presented in Table 1.

Table 1. Baseline demographic and clinical characteristics of SSNHL patients and controls

Variable	SSNHL patients (n=30)	Control group (n=30)
Age, years, mean±SD	45.3±14.2	44.1±13.7
Sex, n (%)	Male 19 (63.3) Female 11 (36.7)	Male 18 (60.0) Female 12 (40.0)
Affected ear, n (%)	Right 12 (40.0) Left 11 (36.7) Bilateral 7 (23.3)	Not applicable
Accompanying symptoms, n (%)	None 11 (36.6) Tinnitus only 16 (40.2) Tinnitus + vertigo 3 (10.0) Vertigo only 0	Not applicable
Baseline hearing loss severity	Mild: 8 (26.7) Moderate: 6 (20.0) Severe: 5 (16.7) Profound: 11 (36.7)	Not applicable

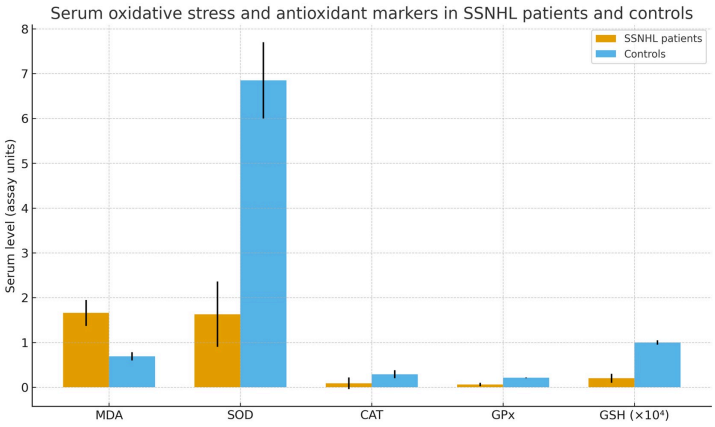
Oxidative stress and antioxidant markers

Patients with SSNHL had significantly higher serum MDA levels than controls (1.66±0.29 vs 0.69±0.09 µmol/L, p<0.001) (Figure 1).In contrast, antioxidant markers were significantly lower in the SSNHL group: SOD, 1.63±0.73 vs 6.85±0.85 U/mL; CAT, 0.09±0.13 vs 0.29±0.09 U/L; GPx, 0.06±0.04 vs 0.21±0.006 U/mL; and GSH, $2\times10^{-5}\pm1\times10^{-5}$ vs $1\times10^{-4}\pm5\times10^{-6}$ U/L (all p<0.001). These between-group differences remained significant after adjustment for age and sex. Sensitivity analyses excluding the 7 patients with bilateral SSNHL showed the same overall pattern, with materially unchanged between-group differences. Biochemical results are summarised in Table 2.

Table 2. Serum oxidative stress and antioxidant markers in SSNHL patients and controls

Marker (unit)	SSNHL patients (n=30) mean±SD (min–max)	Control group (n=30) mean±SD (min–max)	p-value
MDA (µmol/L)	1.66±0.29 (1.12–2.21)	0.69±0.09 (0.52–0.85)	<0.001
SOD (U/mL)	1.63±0.73 (0.54–3.08)	6.85±0.85 (5.42–8.21)	<0.001
CAT (U/L)	0.09±0.13 (0.01–0.42)	0.29±0.09 (0.14–0.46)	<0.001
GPx (U/mL)	0.06±0.04 (0.01–0.15)	0.21±0.006 (0.20–0.22)	<0.001
GSH (U/L)	0.00002±0.00001 (0.00001–0.00004)	0.00010±0.000005 (0.00009–0.00011)	<0.001

SSNHL: Sudden sensorineural hearing loss, MDA: Malondialdehyde, SOD: Superoxide dismutase, CAT: Catalase, GPx: Glutathione peroxidase, GSH: Reduced glutathione, SD: Standard deviation, min: minimum, max: maximum.



SSNHL: Sudden sensorineural hearing loss, MDA: Malondialdehyde, SOD: Superoxide dismutase, CAT: Catalase, GPx: Glutathione peroxidase, GSH: Reduced glutathione.

Figure 1. Serum oxidative stress and antioxidant enzyme levels in SSNHL patients and healthy controls.

Exploratory correlations with clinical features

At presentation, correlations between baseline pure-tone average and oxidative markers were weak and not statistically significant, including MDA (Spearman r=0.20, p=0.29) and SOD (r=−0.18, p=0.34); the other markers showed similarly non-significant correlations (all p>0.10). Among the 28 patients with available 1-month follow-up data, recovery according to Siegel's criteria was complete in 9 (32.1%), partial in 5 (17.9%), and absent in 14 (50.0% patients). Baseline oxidative markers did not differ significantly across outcome categories on omnibus testing, although median values tended to reflect higher MDA and lower antioxidant levels in the no-improvement group.

DISCUSSION

In this study, idiopathic SSNHL was associated with marked redox imbalance. Compared with matched healthy controls, patients with SSNHL had significantly higher serum MDA levels and significantly lower SOD, CAT, GPx, and GSH levels. To our knowledge, this is among the first case-control studies to assess both lipid peroxidation and multiple antioxidant defence markers in idiopathic SSNHL. These findings support the role of oxidative stress in SSNHL pathophysiology.

Our findings align with and extend the growing body of literature linking oxidative mechanisms to inner ear diseases (8, 12, 13). The increased MDA levels observed in patients with SSNHL indicate enhanced lipid peroxidation, a hallmark of oxidative cell injury (17), and are consistent with previous reports showing MDA accumulation in tissues exposed to ischaemic or toxic insults (21). In SSNHL, one plausible explanation is transient cochlear ischaemia followed by reperfusion, leading to excessive ROS generation and oxidative damage to cochlear cell membranes (22). This mechanism, analogous

to ischaemia-reperfusion injury in other organs, has also been proposed in the inner ear as a contributor to sudden hearing loss (13).

Concomitantly, the antioxidant defence systems in patients with SSNHLappeared overwhelmed or depleted. The activities of SOD, CAT, and GPx were all dramatically lower in patients than in controls. Under normal conditions, these enzymes form a first-line defence against oxidative damage: SOD removes superoxide radicals, and CAT and GPx remove peroxides. A significant reduction in their activity suggests either consumption/inactivation of these enzymes due to an excessive ROS burden or downregulation/dysfunction possibly related to the underlying condition of the patients. One possible explanation is that a surge of ROS (as indicated by high MDA) acutely inactivates some enzyme molecules or consumes cofactors (for example, GPx activity depends on the availability of GSH and selenium) (23). Additionally, GSH, which we found to be five-fold lower in patients, is often consumed during periods of intense oxidative stress as it directly quenches free radicals and is utilised by GPx to detoxify peroxides (24). The low GSH levels in patients with SSNHL may reflect an exhaustion of this vital antioxidant reserve despite overwhelming ROS. Alternatively, some patients may have had a chronically weaker antioxidant system even before the SSNHL event, which could predispose them to oxidative damage upon an insult. For instance, nutritional deficiencies or genetic variations (such as polymorphisms in antioxidant enzymes) might result in inherently lower SOD or GPx activity, making the inner ear more vulnerable. Indeed, a recent genetic study found an association between a polymorphism in the SOD2 gene and increased risk of SSNHL, supporting the idea that innate antioxidant capacity can influence susceptibility to sudden hearing loss (25).

Our results are consistent with those of other clinical studies that have examined the oxidative status in SSNHL. Çetin et al. investigated oxidant and antioxidant molecules in SSNHL and found that “the oxidant–antioxidant and thiol–disulphide balances were impaired in the idiopathic SSNHL group” (14). They reported significant differences in total oxidant status and thiol levels between patients and controls, corroborating that an oxidative tilt exists. Similarly, Dinc et al. measured dynamic thiol/disulphide homeostasis as a novel oxidative stress marker in SSNHL and concluded that SSNHL patients experience significant oxidative stress (16). Our study adds direct evidence of increased lipid peroxidation (MDA) and quantifies the deficit in specific antioxidant enzymes, thereby providing a more granular picture of this redox imbalance.

Not all studies have reported reductions in every antioxidant parameter. For example, Gul et al. found higher total oxidant status and oxidative stress index in patients with SSNHL, whereas total antioxidant status and some individual antioxidants, such as paraoxonase and thiols, were unchanged (15). These differences may reflect variations in patient characteristics, timing of sample collection, and assay methods. Because TAS is a global measure, overall antioxidant capacity may appear preserved despite reductions in specific components, such as SOD or GSH (26). In our study, the significantly lower enzymatic antioxidants suggest impairment of these specific defences during the acute phase of SSNHL. Antioxidant levels may also vary over time, and longitudinal studies are needed to clarify these dynamics (27).

The clinical implications of oxidative stress involvement in SSNHL are noteworthy. If ROS-mediated damage contributes to cochlear injury, therapies that either limit ROS formation or strengthen endogenous antioxidant defences may be useful as adjuncts to standard care (12, 13). At present, systemic and/or intratympanic corticosteroids remain the mainstay of idiopathic SSNHL treatment and act primarily through anti-inflammatory and immunomodulatory pathways, although they may also modulate redox defences in some settings (4). Nevertheless, a substantial proportion of patients experience incomplete recovery, underscoring the need for additional strategies. Small exploratory studies have evaluated antioxidant-based adjuncts (such as high-dose vitamins, N-acetylcysteine, melatonin, or hyperbaric oxygen protocols) with mixed but intriguing results, and larger randomised trials will be required to determine whether targeting oxidative stress can meaningfully improve hearing outcomes in SSNHL (8,27-29).

Beyond potential treatments, our findings may help explain certain clinical observations in SSNHL. For instance, SSNHL has worse prognoses in older patients and those with cardiovascular risk factors (30). Both ageing and cardiovascular diseases are associated with heightened oxidative stress and reduced antioxidant capacity systemically (31). One could speculate that patients with those backgrounds have less “redox reserve” in the cochlea, so an idiopathic insult triggers greater oxidative damage, leading to more severe or irreversible hearing loss. This is in line with the idea of endothelial dysfunction (often driven by oxidative stress) contributing to cochlear vascular problems in SSNHL (32). Indeed, Gul et al. suggested that their oxidative findings point to endothelial dysfunction in SSNHL etiopathogenesis (15). Our demonstration of an oxidative imbalance provides biochemical evidence supporting this theory.

Clinical correlations and prognostic implications

Although our cohort exhibited a consistent high-oxidative/low-antioxidant signature relative to controls, baseline serum redox markers did not correlate with initial hearing thresholds or predict recovery using Siegel’s criteria (33). The lack of association likely reflects both statistical and biological factors: limited power in a modest sample (risk of type II error) and etiologic heterogeneity of idiopathic SSNHL (ischaemic, viral, immune endotypes), in which oxidative injury may be a common downstream pathway but not the sole rate-limiting determinant of threshold shift or steroid

responsiveness. Methodological issues may also contribute. Single time-point sampling at presentation likely captures a transient oxidative burst whose amplitude depends on symptom to blood draw latency rather than on the eventual recovery biology. In addition, serum indices are systemic surrogates and may not mirror perilymphatic redox status; enzymatic activities are sensitive to preanalytical handling and micronutrient cofactors (e.g., selenium for GPx). Clinically, these findings argue against using isolated baseline MDA/SOD/CAT/GPx/GSH as stand-alone prognostic tests. Future work should track longitudinal trajectories (baseline/early follow-up/post-therapy), integrate redox markers with vascular and immune indices in multivariate models, and evaluate composite algorithms with ROC analysis. Etiology enriched stratification (e.g., presence of vertigo, cardiovascular risk, and audiometric configuration) and adjustment for time to treatment may unmask subgroup-specific prognostic value.

Study limitations

Several limitations should be acknowledged. First, the sample size was relatively modest (n = 30 per group); while sufficient to detect the large differences in oxidative markers, it may have limited power to detect more subtle associations with hearing outcomes. Second, we measured serum markers only. Ideally, inner ear fluid or tissue markers would directly reflect cochlear oxidative status, but these are not feasible to obtain in living patients; serum markers are a proxy and may be influenced by systemic conditions. We mitigated this by excluding patients with major systemic inflammatory or metabolic diseases and using well-matched controls, but residual confounding cannot be ruled out. In addition, the relatively high proportion of bilateral cases in our cohort should be interpreted cautiously because bilateral SSNHL has a broader differential diagnosis than unilateral disease. However, all bilateral cases underwent the same comprehensive etiologic evaluation as the rest of the cohort, including detailed history, physical examination, MRI, and targeted laboratory testing when clinically indicated, and no secondary cause was identified. Nevertheless, some residual etiologic heterogeneity cannot be excluded. Third, oxidative stress markers were measured only at a single time point, at presentation. Therefore, we cannot determine causality or distinguish whether oxidative stress was a contributing factor or a secondary consequence of cochlear injury. Experimental data indicate that ROS can induce cochlear injury, but reverse causation remains possible, for example, if primary tissue damage leads to increased ROS production via inflammation. Finally, we did not evaluate longitudinal changes in these markers after treatment. It would be informative to determine whether patients who recover normalise their oxidative marker levels and whether those with poor recovery exhibit persistent oxidative abnormalities, which could strengthen the case for oxidative stress as a therapeutic target.

CONCLUSION

Idiopathic SSNHL was associated with increased oxidative stress and impaired antioxidant defence in this case–control study. Higher serum MDA levels with lower SOD, CAT, GPx, and reduced glutathione levels support a role for redox imbalance in SSNHL pathophysiology. These findings suggest that oxidative stress may represent a relevant therapeutic target and that redox-targeted adjunctive strategies warrant further clinical investigation.

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