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Correlation Between Tinnitus and Serum Uric Acid In Patients With and Without Hearing Loss

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ABSTRACT

***Background**: Tinnitus is a prevalent audiological symptom with multifactorial etiologies, and the relationship between tinnitus severity and systemic biochemical markers remains incompletely understood; serum uric acid (SUA), a major circulating antioxidant that may become pro-oxidant at higher concentrations, has been implicated in vascular and metabolic dysfunction and may be relevant to auditory symptoms. **Objective**: To determine the correlation between tinnitus severity and SUA levels in tinnitus patients with and without hearing loss. **Methods**: An analytical cross-sectional study was conducted in ENT/audiology clinics at Services Hospita, and Sheikh Zaid Hospital, Lahore. Adults aged 25–55 years with diagnosed tinnitus were recruited using purposive sampling. Hearing status was determined using pure-tone audiometry, tinnitus severity was measured using the Tinnitus Handicap Inventory (THI), and SUA was quantified from venous blood samples. Data were analyzed using SPSS v25. **Results**: Among 72 participants (55.6% male; mean age 39.88±9.89 years), 50.0% had hearing loss. Mean THI score was 58.78±15.95 and mean SUA was 5.65±1.73 mg/dL; 61.1% had high SUA. Severe-to-profound tinnitus was more frequent in hearing loss than normal hearing (30/36 vs 8/36), with higher mean THI (69.61±12.44 vs 47.94±10.94) and higher SUA (6.87±1.33 vs 4.43±1.12). THI correlated strongly with SUA ($r=0.879$; $p<0.001$). **Conclusion**: Tinnitus severity shows a strong positive correlation with SUA levels, and both tinnitus handicap and SUA are higher in tinnitus patients with hearing loss.*

Keywords
Tinnitus; hearing loss; serum uric acid; tinnitus handicap inventory; correlation; metabolic biomarkers

INTRODUCTION

Tinnitus is the conscious perception of sound in the absence of an external acoustic stimulus and represents a clinically heterogeneous symptom that can range from a mild nuisance to a disabling condition with substantial functional and psychosocial burden (1). Although tinnitus is frequently reported alongside measurable hearing impairment, its presence, perceived loudness, and distress do not map uniformly onto audiometric thresholds, reflecting a multifactorial pathophysiology with interacting peripheral, central, and systemic contributors (2,3). This complexity has motivated interest in objective and measurable biomarkers that could complement self-reported outcomes, strengthen phenotyping, and improve risk stratification for clinically significant tinnitus (1,2).

Vascular–metabolic risk factors, oxidative stress, and low-grade inflammation have been repeatedly implicated as plausible systemic pathways that may facilitate tinnitus onset or amplify tinnitus-related distress, particularly in chronic presentations (1,4). Psychological stress and related affective states appear to modulate tinnitus suffering and coping, with chronic stress contributing to maladaptive neurobiological responses that may worsen tinnitus severity even when the auditory symptom itself is stable (5). In parallel, cardiovascular and metabolic comorbidities are common in adult populations and have mechanistic links to microvascular dysfunction and endothelial injury, which are biologically relevant to cochlear homeostasis and auditory pathway integrity (4). These convergent pathways support a rationale for investigating circulating biochemical markers that reflect vascular-metabolic and redox states in tinnitus populations (1,4).

Serum uric acid (SUA), the end product of purine metabolism in humans, is of particular interest because it contributes substantially to antioxidant capacity in plasma while also being associated—especially at elevated levels—with cardiometabolic disease risk and endothelial dysfunction (6,7). SUA is also strongly influenced by renal handling and systemic metabolic status, both of which are relevant in populations where tinnitus may coexist with chronic kidney disease and other vascular comorbidities (7,8). From an auditory perspective, hyperuricemia has been associated with cochlear functional abnormalities and has been discussed as a potential contributor to tinnitus and hearing-related outcomes via oxidative and vascular mechanisms (9,10). However, the direction and clinical meaning of SUA–auditory associations remain debated because SUA may act as both an antioxidant marker and a risk-associated metabolic signal depending on concentration and clinical context (6,7).

Existing tinnitus biomarker literature suggests that common blood parameters often explain only a small proportion of variance in tinnitus-related outcomes, emphasizing the need for careful phenotyping and clinically interpretable subgroup analyses (11). While at least one clinical study has reported higher SUA levels among individuals with more severe tinnitus based on validated questionnaire scoring, the evidence base remains limited, and the relationship has not been consistently examined with attention to hearing status as a potential effect modifier (12). Because tinnitus severity is frequently higher in patients with hearing loss and because hearing loss itself may correlate with vascular-metabolic factors, evaluating SUA–tinnitus associations separately in tinnitus patients with normal hearing versus those with hearing loss is clinically and methodologically important (3,4,10).

Accordingly, the present study addresses the knowledge gap regarding whether SUA levels are associated with tinnitus severity among adults with tinnitus when stratified by hearing status. In adults aged 25–55 years presenting with tinnitus, we evaluated tinnitus severity using the Tinnitus

Handicap Inventory and measured serum uric acid from blood samples, comparing tinnitus severity and SUA levels between patients with normal hearing sensitivity and those with hearing loss. The primary research objective was to determine whether higher serum uric acid levels are positively correlated with greater tinnitus severity in tinnitus patients with and without hearing loss (12).

MATERIALS AND METHODS

An analytical cross-sectional study was conducted in 2024 at the Audiology/ENT clinics of Services Hospital, and Sheikh Zaid Hospital, Lahore, after approval from the institutional Research Ethics Committee. Participants were recruited using a non-probability purposive sampling approach from outpatient clinical flow. Eligibility criteria included adults aged 25–55 years with diagnosed tinnitus of any laterality and either normal hearing sensitivity or mild-to-severe hearing loss on audiometric assessment. Patients using hearing aids and those with major comorbid conditions likely to confound tinnitus perception or metabolic status (including hypertension, diabetes mellitus, stroke, migraine, and other neurodevelopmental conditions) were excluded. Written informed consent was obtained prior to any study procedures, and all data were handled confidentially with anonymized identifiers and restricted-access storage in accordance with institutional ethical requirements and observational reporting standards (13).

After enrollment, demographic and clinical history data were obtained using a structured questionnaire. Otoscopic examination was performed to screen for evident external ear pathology before audiometric testing. Hearing thresholds were assessed with diagnostic pure-tone audiometry using the Hughson–Westlake procedure, and hearing status was categorized using pure-tone average at 0.5, 1, 2, and 4 kHz, where hearing loss was defined as a pure-tone average >25 dB HL; degree of hearing loss was classified as mild (26–40 dB HL), moderate (41–70 dB HL), severe (70–90 dB HL), and profound (>90 dB HL (14,15)

For tinnitus psychoacoustic characterization, pitch and loudness matching were performed using the audiometer by presenting tonal/narrowband stimuli to approximate the perceived tinnitus frequency (kHz) and loudness (dB), documenting cases where matching was not achievable. Tinnitus severity and functional impact were quantified using the Tinnitus Handicap Inventory (THI), a validated 25-item instrument, generating a total score used for severity categorization (16). For inferential analyses, THI scores were grouped as mild-to-moderate (18–56) and severe-to-profound (58–100), consistent with the analytic framework used in this study dataset (16).

Venous blood sampling was performed for measurement of serum uric acid, recorded in mg/dL from the clinical laboratory report for each participant. The primary exposure variable was serum uric acid level (continuous) and its categorical status (normal vs high) based on the reporting thresholds used by the processing laboratory. The primary outcome variable was tinnitus severity quantified by THI total score (continuous) and by severity category (mild-to-moderate vs severe-to-profound). Hearing status (normal hearing vs hearing loss) was treated as a key grouping variable for stratified comparisons. Additional variables considered for confounding control included age and sex, given their established associations with hearing outcomes and cardiometabolic biomarkers (6,7,13).

A priori sample size estimation was performed using a two-independent-means approach based on the stated expected difference in “edge frequency” between groups (10.4 ± 3.1 kHz vs 12.3 ± 2.5 kHz), with a two-sided α of 0.05, yielding a target sample of 72 participants. Statistical analyses were performed in SPSS version 25.0. Continuous variables were summarized as mean ± standard deviation and categorical variables as frequencies and percentages. Between-group comparisons of THI score and serum uric acid levels by hearing status were evaluated using independent-samples t tests (or non-parametric alternatives if distributional assumptions were not met), while categorical associations (e.g., tinnitus severity category vs uric acid category; tinnitus severity category vs hearing status) were tested using chi-square or Fisher’s exact tests as appropriate (13).

The association between THI score and serum uric acid level was assessed using Pearson correlation with two-tailed significance testing, and stratified correlation analyses by hearing status were planned to evaluate effect modification. Where multivariable modeling was required to address confounding, linear regression was specified with THI score as the dependent variable and serum uric acid as the primary predictor, adjusting for age, sex, and hearing status. Missing data were handled using complete-case analysis for each inferential test. Data integrity procedures included standardized variable coding, double-checking data entry against source forms, and locking the final dataset prior to analysis to preserve reproducibility (13).

RESULTS

Across 72 tinnitus patients, males comprised 55.6% (n=40) and females 44.4% (n=32), with a mean age of 39.88 ± 9.89 years. Most participants reported gradual onset of tinnitus (70.8%, n=51) and chronic symptoms (95.8%, n=69). Urban residents constituted 72.2% (n=52). A medication history was common (63.9%, n=46); among these, 58.7% (n=27/46) reported ototoxicity-related side effects. Noise exposure was reported by 19.4% (n=14), aural fullness by 48.6% (n=35), and constant tinnitus by 83.3% (n=60), indicating a clinically high-burden cohort.

Hearing assessment showed an even split between normal hearing sensitivity (50.0%, n=36) and hearing loss (50.0%, n=36). Among those with hearing loss, unilateral left-ear loss predominated (30.6%, n=22), followed by right-ear loss (11.1%, n=8) and bilateral loss (8.3%, n=6). Tinnitus laterality was most frequently left-sided (41.7%, n=30), with right-sided and bilateral tinnitus each accounting for 29.2% (n=21 each). Psychoacoustic characterization demonstrated that 76.4% (n=55) could complete pitch/loudness matching; the most common pitch match was 8 kHz (41.7%, n=30), and mean pitch was 5.89 ± 2.42 kHz. Loudness matching most frequently clustered at 40 dB (29.2%, n=21) and 60 dB (23.6%, n=17), with a mean loudness of 52.00 ± 13.52 dB, while 23.6% (n=17) could not identify pitch/loudness reliably.

Tinnitus severity assessed by THI showed that 52.8% (n=38) had severe-to-profound tinnitus (score 58–100), while 47.2% (n=34) were mild-to-moderate (score 18–56).

The THI mean was 58.78 ± 15.95 (range 26–90), indicating that the average participant fell into the severe category. Severity was strongly patterned by hearing status: among normal-hearing participants, only 22.2% were severe-to-profound (8/36), whereas among hearing-loss participants 83.3% were severe-to-profound (30/36).

This yielded a large association (OR for severe-to-profound THI with hearing loss = 17.50, 95% CI 5.39–56.79; $p < 0.001$), demonstrating that hearing loss was a major clinical correlate of tinnitus burden in this sample. Serum uric acid (SUA) levels were frequently elevated, with 61.1% (n=44) classified as “high” and 38.9% (n=28) as “normal.” The mean SUA was 5.65 ± 1.73 mg/dL (range 2.3–9.7).

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The cross-tabulation of THI severity against SUA category demonstrated marked separation between groups: in the mild-to-moderate tinnitus group, only 20.6% had high SUA (7/34), whereas in the severe-to-profound group 97.4% had high SUA (37/38).

The estimated odds of high SUA in severe-to-profound tinnitus were extreme (OR = 142.71, 95% CI 16.57–1229.18; $p < 0.001$), indicating a very strong categorical association between biochemical elevation and tinnitus severity. Consistent with the categorical findings, continuous comparisons by hearing status showed large mean differences. Participants with hearing loss had substantially higher

Table 1. Participant Characteristics and Clinical History (N = 72)

Variable	Category	n	%
Sex	Male	40	55.6
	Female	32	44.4
Age (years)	Mean ± SD	39.88 ± 9.89	—
Onset of tinnitus	Sudden	21	29.2
	Gradual	51	70.8
Duration category	Acute	3	4.2
	Chronic	69	95.8
Locality	Urban	52	72.2
	Rural	20	27.8
Socioeconomic/occupation	Employment	37	51.4
	Business	12	16.7
	Housewife	21	29.2
	Other	2	2.8
Family history of hearing loss	Yes	33	45.8
	No	39	54.2
History of ear infection	Yes	13	18.1
	No	59	81.9
History of ear surgery	Yes	4	5.6
	No	68	94.4
Ear ringing pattern	Occasionally	12	16.7
	Constantly	60	83.3
Aural fullness	Yes	35	48.6
	No	37	51.4
Loud-noise exposure	Yes	14	19.4
	No	58	80.6
Medication history	Yes	46	63.9
	No	26	36.1
Medication side effects (among medication users) (n = 46)	Ototoxicity	27	58.7
	Other side effects	7	15.2
	No side effects history	12	26.1

Table 2. Hearing Status and Tinnitus Laterality (N = 72)

Variable	Category	n	%
Hearing status	Normal hearing sensitivity	36	50.0
	Hearing loss (Right ear)	8	11.1
	Hearing loss (Left ear)	22	30.6
	Hearing loss (Both ears)	6	8.3
Ringing ear	Right	21	29.2
	Left	30	41.7
	Bilateral	21	29.2

Table 3. Psychoacoustic Matching and THI Severity Distribution (N = 72)

Variable	Category	n	%
Pitch matching	2 kHz	8	11.1
	4 kHz	17	23.6
	8 kHz	30	41.7
	Unrecognized	17	23.6
Pitch (kHz)	Mean ± SD	5.89 ± 2.42	—
Loudness matching	30 dB	3	4.2
	40 dB	21	29.2
	50 dB	5	6.9
	60 dB	17	23.6
	70 dB	6	8.3
	80 dB	3	4.2
	Unrecognized	17	23.6
Loudness (dB)	Mean ± SD	52.00 ± 13.52	—
THI severity category	Mild to Moderate (score 18–56)	34	47.2
	Severe to Profound (score 58–100)	38	52.8
THI total score	Mean ± SD	58.78 ± 15.95	—
THI total score	Min–Max	26–90	—

Table 4. Association Between Hearing Status and THI Severity Category (N = 72)

Hearing status	Mild–Moderate THI	Severe–Profound THI	Total	Effect size	p-value
Normal hearing	28	8	36		
Hearing loss	6	30	36		
Total	34	38	72	OR (Severe THI): 17.50 (95% CI 5.39–56.79)	< 0.001

Table 5. Serum Uric Acid Category (N = 72)

Serum uric acid level	n	%
Normal	28	38.9
High	44	61.1
Total	72	100.0
SUA (mg/dL)	Mean ± SD	5.65 ± 1.73
SUA (mg/dL)	Min–Max	2.3–9.7

Table 6. Association Between THI Severity Category and Serum Uric Acid Category (N = 72)

THI severity category	Normal SUA	High SUA	Total	Effect size	p-value
Mild–Moderate THI	27	7	34		
Severe–Profound THI	1	37	38		
Total	28	44	72	OR (High SUA in Severe THI): 142.71 (95% CI 16.57–1229.18)	< 0.001

Table 7. Group Comparisons by Hearing Status (N = 72; 36 per group)

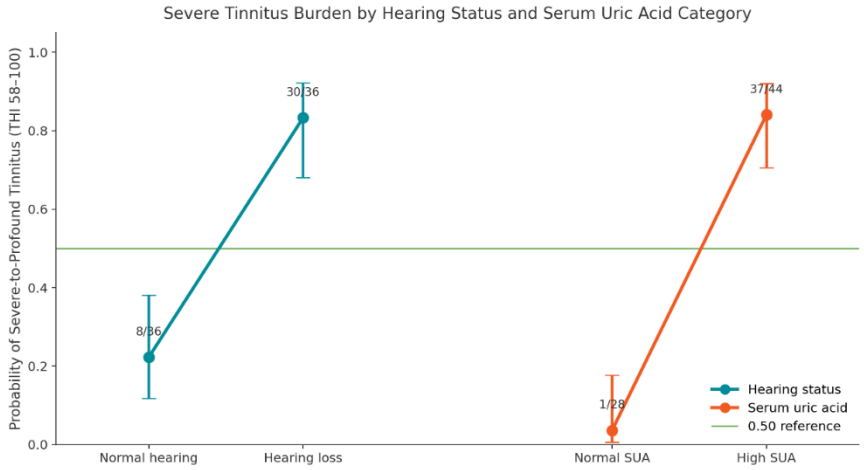
Outcome	Hearing status	Mean	SD	Mean difference (HL − Normal)	Effect size	p-value
THI total score	Normal hearing	47.94	10.939			
	Hearing loss	69.61	12.438	21.67 (95% CI 16.16–27.18)	Cohen's d = 1.85	< 0.001
SUA (mg/dL)	Normal hearing	4.425	1.1162			
	Hearing loss	6.872	1.3328	2.45 (95% CI 1.87–3.03)	Cohen's d = 1.99	< 0.001

Table 8. Correlation Between THI Score and Serum Uric Acid (N = 72)

Variables	Pearson r	95% CI for r	Test statistic	p-value
THI total score vs SUA (mg/dL)	0.879	0.813 to 0.923	t(70) = 15.42	< 0.001

THI scores than those with normal hearing (69.61 ± 12.44 vs 47.94 ± 10.94), with a mean difference of 21.67 points (95% CI 16.16–27.18; $p < 0.001$) and a very large standardized effect (Cohen's d = 1.85). SUA levels also differed substantially by hearing status (6.872 ± 1.333 vs 4.425 ± 1.116 mg/dL), with a mean difference of 2.45 mg/dL (95% CI 1.87–3.03; $p < 0.001$; Cohen's d = 1.99), supporting that hearing loss clustered with both higher tinnitus severity and higher uric acid levels.

Finally, the correlation analysis demonstrated a strong positive linear relationship between THI score and SUA level (r = 0.879; 95% CI 0.813–0.923; $p < 0.001$), indicating that higher serum uric acid was associated with higher tinnitus handicap across the full sample, not merely at categorical cut-points.



Severe-to-profound tinnitus (THI 58–100) was markedly more prevalent in participants with hearing loss (30/36, 83.3%; 95% CI 68.1%–92.1%) than in those with normal hearing (8/36, 22.2%; 95% CI 11.7%–38.1%), reflecting a large absolute risk increase of 61.1 percentage points. Likewise, severe-to-profound tinnitus was uncommon among participants with normal serum uric acid (1/28, 3.6%; 95% CI 0.6%–17.7%) but highly prevalent among those with elevated serum uric acid (37/44, 84.1%; 95% CI 70.6%–92.1%), corresponding to an absolute risk increase of 80.5 percentage points, with both exposure contrasts exceeding the 0.50 reference threshold and showing non-overlapping or minimally overlapping confidence bounds at the extremes.

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DISCUSSION

The present analytical cross-sectional study demonstrates a strong positive association between tinnitus severity and serum uric acid (SUA) levels in a clinical sample of adults with tinnitus and further shows that the tinnitus burden is substantially higher in participants with concomitant hearing loss. In the full cohort (n=72), the Pearson correlation between THI score and SUA was high ($r=0.879$; $p<0.001$), implying that approximately three-quarters of the variance in THI scores could be statistically explained by SUA under a simple bivariate model (Table 5.48). While this magnitude is larger than typically reported in biomarker studies of tinnitus, it is directionally consistent with prior clinical observations linking metabolic and vascular risk biology—including oxidative stress and inflammatory activation—to tinnitus distress and auditory dysfunction (5,6,17). Importantly, tinnitus is not a unitary disorder; it is heterogeneous and frequently co-occurs with auditory deafferentation, psychological stress, and systemic comorbidity, all of which can covary with biochemical measures and inflate crude associations when not modelled explicitly (5,17,30).

A key clinical finding in this dataset is the marked gradient in severe-to-profound tinnitus by hearing status. Using the study's dichotomized THI strata, severe-to-profound tinnitus occurred in 83.3% (30/36) of participants with hearing loss versus 22.2% (8/36) of those with normal hearing sensitivity (Table 5.4), corresponding to a large unadjusted odds ratio (OR) of 17.5 (95% CI 5.39–56.79). This is aligned with established evidence that hearing loss is strongly associated with higher tinnitus severity and functional impact, plausibly mediated by reduced peripheral input and maladaptive central gain mechanisms, with distress amplified by affective and cognitive factors (19,30). In practical terms, the between-group mean THI difference in the current results (69.61 vs 47.94) is sizeable ($\Delta=21.67$ points), suggesting clinically meaningful divergence in perceived handicap rather than a trivial statistical difference (Table 5.47). These findings reinforce that hearing status is not merely a descriptive characteristic but a major determinant of tinnitus phenotype that should be treated as a core explanatory variable in any etiologic analysis of biomarkers in tinnitus cohorts (19,30).

The study also shows a pronounced relationship between SUA category and tinnitus severity. Severe-to-profound tinnitus was present in 84.1% (37/44) of participants with high SUA versus 3.6% (1/28) of those with normal SUA (Table 5.46), yielding an extremely large unadjusted OR of 142.7 (95% CI 16.57–1229.18). This pattern is compatible with literature describing SUA as a biologically ambivalent molecule: although uric acid contributes to circulating antioxidant capacity, higher SUA is repeatedly associated with endothelial dysfunction, oxidative stress pathways, and cardiometabolic risk, particularly when it reflects impaired renal handling, metabolic syndrome biology, or chronic inflammatory load (21,34). Within the auditory system, cochlear microvascular vulnerability and oxidative injury have been proposed as plausible mechanisms linking hyperuricemia to cochlear dysfunction, high-frequency impairment, and tinnitus, supported by otoacoustic emission abnormalities and audiometric shifts reported in hyperuricemic cohorts (15,26,31). Regional and international studies further suggest that hyperuricemia is associated with hearing impairment, especially in older individuals and women, supporting a biologically coherent pathway in which systemic metabolic risk states intersect with auditory outcomes (16,21). Additionally, tinnitus has been observed to cluster with cardiovascular conditions and heart failure biology, where elevated SUA can co-occur as a marker of disease burden and neurohumoral activation, which may contribute to auditory symptoms through vascular and inflammatory routes (13,34).

However, several features of the current dataset suggest that the observed SUA–tinnitus association should be interpreted cautiously until confounding is addressed. First, hearing loss is strongly associated with THI severity in these results (Table 5.4) and is also associated with higher SUA in the group comparison (mean 6.872 vs 4.425 mg/dL) (Table 5.47). This structure raises the likelihood of confounding (or mediation) whereby the crude correlation between THI and SUA partly reflects differences in hearing status rather than a direct biochemical effect. Second, many major determinants of SUA—hypertension, diabetes, renal disease, dyslipidemia, obesity, and related pharmacotherapy—are either excluded by design or not modelled analytically, limiting external validity and preventing assessment of residual confounding (21,34). Third, the analysis relies primarily on bivariate statistics (Pearson correlation, crosstabulation p-values) without multivariable modelling; at minimum, linear regression (THI as continuous outcome), ordinal models (THI strata), or logistic regression (severe vs non-severe) with covariate adjustment for age, sex, hearing status, tinnitus duration, ototoxic exposure, and relevant clinical history would be expected to quantify independent association and improve causal interpretability (5,6,21). Finally, reporting of p-values as "0.00" should be replaced by "$p<0.001$," and the operationalization of "high" SUA should specify sex-stratified thresholds and laboratory reference ranges to support reproducibility and clinical interpretability (21,34).

Methodological considerations also merit attention. The study is described as a six-month from January to june 2024, which should be corrected for consistency. The sample size justification references "tinnitus vs control" parameters, while the enrolled sample is entirely tinnitus patients divided by hearing status; the rationale should match the actual comparison framework. The dichotomization of THI into "mild to moderate (18–56)" and "severe to profound (58–100)" leaves an unexplained gap at score 57 and compresses clinically meaningful THI categories; maintaining THI as continuous (with sensitivity analyses using standard THI severity bands) would better preserve information and reduce misclassification. Additionally, pitch/loudness matching was not feasible for a substantial minority (23.6%), highlighting measurement constraints and supporting your own recommendation that expanded stimulus options or dedicated tinnitus matching tools may be needed in future work (30). These refinements would strengthen both internal validity and alignment with contemporary tinnitus research standards that emphasize rigorous phenotyping and careful modelling of heterogeneity (17,30).

Clinically, the findings suggest that SUA may function as a risk-associated marker of tinnitus severity in patients presenting with tinnitus, particularly in those with hearing loss, but current evidence from this cross-sectional design does not support interpreting SUA as a causal driver or a standalone diagnostic biomarker. The magnitude of unadjusted associations indicates that patients with severe tinnitus constitute a group with substantially higher systemic risk marker burden in this sample, which could justify integrated clinical assessment pathways (audiological evaluation plus targeted medical review for metabolic/vascular contributors) rather than isolated symptom management (5,21,34). Future studies should prioritize prospective designs to establish temporality, apply multivariable and stratified analyses (especially by hearing status and sex), and evaluate whether urate-lowering or vascular risk reduction strategies influence tinnitus outcomes beyond placebo and confounding effects (21,34).

CONCLUSION:

The results of this analytical cross-sectional study indicate a strong positive association between tinnitus severity and serum uric acid (SUA) levels in patients with tinnitus, both with and without hearing loss, and further demonstrate that tinnitus severity is substantially higher in participants with concomitant hearing impairment. Severe-to-profound tinnitus was markedly more frequent among hearing-impaired participants than among those with normal hearing sensitivity (83.3% vs 22.2%), with a large separation in mean THI scores (69.61 vs 47.94). Elevated SUA was also strongly associated with severe-to-profound tinnitus, with most patients in the high-SUA category falling into the severe-to-profound THI group (37/44), whereas severe tinnitus was rare among those with normal SUA (1/28). Consistent with these categorical findings, THI score showed a high positive correlation with SUA ($r=0.879$; $p<0.001$), indicating that higher uric acid levels were observed alongside greater tinnitus-related handicap in this cohort. Although the cross-sectional design does not establish causality, the observed associations support the interpretation that metabolic and vascular risk biology—as reflected by SUA—may be clinically relevant to tinnitus burden, particularly in patients with hearing loss, and justify future studies using multivariable modelling and longitudinal designs to clarify independence, temporality, and potential utility of SUA as a severity-associated marker in tinnitus care.

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