Original Article

The Silent Sound of Hair Loss: Heat Shock Protein 70 (HSP-70), Hearing Sensitivity, and Alopecia Areata

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Abstract

Aim: This cross-sectional investigation aimed to delineate the role of circulating heat shock protein 70 (HSP-70) in alopecia areata (AA) pathogenesis, to assess its correlation with disease severity, and to evaluate its potential association with auditory function.

Materials and Methods: A cohort comprising 30 AA patients and 30 matched, healthy, AA-free subjects underwent comprehensive assessment. Circulating HSP-70 levels were quantified using enzyme-linked immunosorbent assay, and the Severity of Alopecia Tool (SALT) score was used to objectively assess AA severity. Audiological integrity was evaluated through pure-tone audiometry across standard frequencies. The predictors of AA were identified using multivariate logistic regression.

Results: Revealed a statistically significant elevation of serum HSP-70 in AA patients compared to controls (2.5 ± 0.8 ng/mL vs. 1.3 ± 0.6 ng/mL; $P < 0.001$). Furthermore, AA patients exhibited significantly higher mean auditory thresholds across various frequencies; however, the clinical significance of these differences requires further investigation. Receiver operating characteristic analysis demonstrated robust diagnostic utility for HSP-70, with an area under the curve of 0.847 ($P < 0.001$) and an optimal diagnostic threshold exceeding 1.4 ng/mL. Multivariate analysis indicated that each unit increase in HSP-70 was associated with ninefold higher odds of AA (odds ratio = 9.213, $P < 0.001$). A positive correlation was observed between HSP-70 levels and disease duration (r = 0.504, $P = 0.004$), although no significant associations were found between HSP-70 levels and SALT score or auditory thresholds.

Conclusion: Elevated HSP-70 levels show promise as a biomarker for AA and correlate with disease chronicity. Further research is imperative to elucidate HSP-70's mechanistic contributions to AA pathogenesis and its nuanced relationship with auditory function.

Keywords: Alopecia areata, biomarker, hearing sensitivity, heat shock protein 70, pure-tone audiometry

Introduction

Alopecia areata (AA) is a prevalent immune-triggered disorder with well-defined non-scarring areas of hair loss on different body areas-preferentially scalp and beard- as typical presentations.[1] Nail abnormalities, including pitting and trachyonychia, may also be present.[2]

AA has been associated with various etiological factors, including genetic predisposition, immunological reactions, and oxidative damage. Among these, disturbance of the hair follicular immune privilege is considered a significant pathogenic mechanism, with melanocyte-associated autoantigens serving as the main immunological targets.[3]

Melanocytes are vital for maintaining endocochlear potential, controlling inner hair cell function, and protecting against free radical damage. Similar to hearing loss observed in vitiligo, which may arise from melanocyte depletion, the immune-

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triggered, melanocyte-related pathogenic mechanisms in AA may be associated with sensorineural hearing loss (SNHL). Scant literature comments on a link between AA and hearing loss.[4,5]

Heat shock proteins (HSPs) are a class of proteins that are produced in response to a variety of stimuli, such as heat, toxins, heavy metals, or infections. They play an important role in protecting cells from stress by assisting in the proper packaging of other proteins and are therefore commonly known as molecular chaperones. HSPs accumulate in numerous diseases and malignancies.[6]

Among the various HSP families, heat shock protein 70 (HSP-70) has received particular attention due to its dual role in maintaining cellular homeostasis and modulating immune responses. Intracellularly, depending on their location, HSP-70 fights stress-induced damage and promotes protein production and transfer and inhibits protein folding errors and instability. Extracellularly, or when anchored to the cell surface, HSP-70 plays an important role in activating immunological responses.[6]

This study aims to investigate circulating levels of HSP-70 in patients with AA, to explore its relationship with disease severity, to assess its potential association with audiological abnormalities in these patients, and to generate hypotheses for future research.

Materials and Methods

Design and Population

This cross-sectional study included 60 participants: 30 patients with clinically diagnosed and dermoscopically confirmed AA and 30 matched AA-free controls. The study was conducted over six months, from October 2023 to March 2024. The Benha University Research Ethics Committee approved the protocol (approval number: Ms 17-10-2023, approved on October 17, 2023; final document issued: 29.12.2024), and all subjects provided signed informed consent prior to participation.

Eligibility Criteria

Patients of both genders aged 18 years or older who were clinically diagnosed with AA and had the diagnosis confirmed through dermoscopic examination were included.

Excluded participants were those who had any condition associated with hearing loss from known causes, including acoustic neuroma, central lesions, Meniere's disease, multiple sclerosis, drug or noise exposure, or prior ear surgery. Additional exclusion criteria included the presence of dermatological conditions other than AA; systemic or autoimmune disorders; cardiovascular or cerebral ischemic heart disease; recent use (within the past month) of antibiotics, hormonal medications, compounds containing nitric oxide, or heavy metals; and non-epithelial cancers, such as ovarian, breast, lung, or pancreatic cancer.

Participants were stratified into two equal groups: a patient group comprising individuals diagnosed with AA, and a control group of AA-free volunteers matched for age (P = 0.161) and gender (P = 0.165).

All patients in this study will undergo the following:

History and Clinical Examination

All patients underwent detailed history-taking, including personal information, disease characteristics, and family history of AA; this was followed by a comprehensive clinical examination to exclude other dermatological conditions and to assess the site, number, and size of alopecia patches.

Dermoscopic evaluation of scalp patches was performed using a DermLite® 4 dermoscope (California, USA), and images were documented. AA severity was determined using the Severity of Alopecia Tool (SALT) score,[7] which quantifies hair loss in four scalp regions, with scores measured visually and corroborated by photographic analysis.

Hearing Assessment

Pure-tone audiometry (PTA) was used to assess hearing thresholds in patients and controls. To minimize bias, audiologists were blinded to the group status (AA patient vs. control) during the assessments. This was conducted in a soundproof cabin using a Resonance R37A audiometer (Italy) with frequencies ranging from 250–8000 Hz. Air-conduction thresholds were measured in both ears. Bone conduction audiometry was not performed because the primary aim was to assess overall hearing sensitivity in relation to AA; air conduction thresholds are sufficient for this initial screening. Pure-tone averages were calculated using thresholds at 500, 1000, and 2000 Hz. Hearing loss was defined as a threshold of ≥ 26 dB, categorized as hypoacusis.

Serum HSP-70 Analysis

Venous blood samples were collected from all participants under aseptic conditions and allowed to clot at room temperature for 15–20 minutes. Samples were then centrifuged at 3000 rpm for 20 minutes to separate serum, which was aliquoted into sterile Eppendorf tubes and stored at –70 °C until analysis. Hemolyzed samples were excluded. Serum levels of total HSP-70 were measured using a human HSP-70 enzyme-linked immunosorbent assay kit (Catalogue

No. 201-12-1814; SunRed Biological Technology Co., Ltd., Shanghai, China) according to the manufacturer's instructions in the Clinical and Chemical Pathology Department. This kit measures total HSP-70 (both constitutive and inducible forms).

Sample Size Calculation

Sample size was calculated using G*Power software version 3.1.9.2, based on a previous study by Ghaderi,[8] which investigated serum HSP-70 levels in patients with AA. The study reported a large effect size (d) for differences in HSP-70 between patients with AA and controls. The effect size from Ghaderi[8] was chosen due to its direct relevance to HSP-70 levels in AA patients, providing a comparable basis for our power calculation. The alpha level and statistical power were set at 0.05 and 0.8, respectively.

Statistical Analysis

SPSS version 28 (IBM, Armonk, NY, USA) was employed for statistical analysis. Parametric data were expressed as mean ± standard deviation (SD), non-parametric data as median and interquartile range, and categorical data as frequencies and percentages. HSP-70's diagnostic performance was evaluated using receiver operating characteristic (ROC) analysis, which yielded area under the curve (AUC) values, cut-off points, and diagnostic indices. Multivariate logistic regression analysis was used to identify determinants of alopecia and to estimate odds ratios (ORs) with 95% confidence intervals (CIs). A P-value of < 0.05 was used to define statistical significance.

Results

All AA patients (n = 30) exhibited a sudden onset and a gradual course, with a median disease duration of 18 months, ranging from 5 to 48 months. No patients had nail involvement, a history of medication use, a positive family history, or any associated diseases. Regarding the SALT score, S2 exhibited the highest percentage, 36.7% (n = 11), followed by S1 and S3, each 26.7% (n = 8). S4 had the lowest representation, accounting for only 10.0% (n = 3). Dermoscopic findings revealed that all participants in the study (100%) exhibited yellow dots, black dots, and exclamation mark hairs. The majority (73.3%) showed vellus hair, while 90% displayed broken hair. This high prevalence of characteristic dermoscopic signs likely reflects our stringent inclusion criteria, which focused on patients with active AA and may have introduced selection bias. We acknowledge that this might not fully represent the spectrum of dermoscopic findings across all AA patients.

In the right ear, patients showed significantly higher mean auditory thresholds at 250 Hz (30 ± 6 vs. 22 ± 3, $P < 0.001$), 500 Hz (26 ± 8 vs. 21 ± 4, $P = 0.003$), 1000 Hz (22 ± 3 vs. 20 ± 4, $P = 0.047$), 4000 Hz (25 ± 12 vs. 17 ± 6, $P = 0.002$), and 8000 Hz (25 ± 9 vs. 17 ± 5, $P = 0.002$). In the left ear, significant differences were noted at 250 Hz (27 ± 6 vs. 22 ± 2, $P < 0.001$), 500 Hz (27 ± 7 vs. 20 ± 4, $P < 0.001$), 1000 Hz (22 ± 4 vs. 20 ± 4, $P = 0.029$), 2000 Hz (23 ± 8 vs. 16 ± 5, $P < 0.001$), 4000 Hz (22 ± 7 vs. 17 ± 7, $P = 0.005$), and 8000 Hz (21 ± 7 vs. 18 ± 3, $P = 0.042$). A total of 15 AA patients exhibited auditory thresholds exceeding 26 dB, indicating hypoacusis (Table 1, Figure 1A and B).

Table 1. Auditory thresholds in the studied groups

		Patients (n = 30)	Controls (n = 30)	*P*-value
Right ear				
250 Hz	Mean ± SD	30 ± 6	22 ± 3	**< 0.001***
500 Hz	Mean ± SD	26 ± 8	21 ± 4	**0.003***
1000 Hz	Mean ± SD	22 ± 3	20 ± 4	**0.047***
2000 Hz	Mean ± SD	21 ± 6	19 ± 3	0.106
4000 Hz	Mean ± SD	25 ± 12	17 ± 6	**0.002***
8000 Hz	Mean ± SD	25 ± 9	17 ± 5	**0.002***
Left ear				
250 Hz	Mean ± SD	27 ± 6	22 ± 2	**< 0.001***
500 Hz	Mean ± SD	27 ± 7	20 ± 4	**< 0.001***
1000 Hz	Mean ± SD	22 ± 4	20 ± 4	**0.029***
2000 Hz	Mean ± SD	23 ± 8	16 ± 5	**< 0.001***
4000 Hz	Mean ± SD	22 ± 7	17 ± 7	**0.005***
8000 Hz	Mean ± SD	21 ± 7	18 ± 3	**0.042***

n: Number, SD: Standard deviation, Hz: Hertz, * = Significant P-value at $P < 0.05$

The serum level of HSP-70 was significantly higher in patients (mean ± SD: 2.5 ± 0.8) compared to controls (mean ± SD: 1.3 ± 0.6), with a *P*-value of < 0.001 (Figure 2).

The ROC analysis was performed to evaluate the ability of HSP-70 to predict alopecia. It revealed an AUC of 0.847 (95% CI, 0.739–0.955), indicating good diagnostic performance. The best cut-off point was > 1.4, at which sensitivity, specificity, positive predictive value, and negative predictive value were 83.3%, 90%, 89.3%, and 84.4%, respectively (*P*-value < 0.001) (Figure 3).

The analysis revealed a significant positive correlation between serum levels of HSP-70 and disease duration (r = 0.504, *P* = 0.004). No significant correlations were observed between serum HSP-70 levels and age (*P* = 0.711), SALT score (*P* = 0.618), or right ear frequencies at 250 Hz (*P* = 0.188), 500 Hz (*P* = 0.655), 1000 Hz (*P* = 0.492), 2000 Hz (*P* = 0.456), 4000 Hz (*P* = 0.295), and 8000 Hz (*P* = 0.534). Similarly, no significant associations were found with the left ear frequencies at 250 Hz (*P* = 0.691), 500 Hz (*P* = 0.845), 1000 Hz (*P* = 0.952), 2000 Hz (*P* = 0.130), 4000 Hz (*P* = 0.259), and 8000 Hz (*P* = 0.249) (Table 2, Figure 4).

The SALT score did not show a statistically significant correlation with hearing thresholds at any frequency in either the right or the left ear. For the right ear, the *P*-values were as follows: 250 Hz (*P* = 0.46), 500 Hz (*P* = 0.99), 1000 Hz (*P* = 0.59), 2000 Hz (*P* = 0.231), 4000 Hz (*P* = 0.495), and 8000 Hz (*P* = 0.848). For the left ear, the *P*-values were: 250 Hz (*P* = 0.171), 500 Hz (*P* = 0.409), 1000 Hz (*P* = 0.491), 2000 Hz (*P* = 0.975), 4000 Hz (*P* = 0.981), and 8000 Hz (*P* = 0.196).

A multivariate logistic regression analysis was performed to predict alopecia. It revealed that a one-unit increase in serum HSP-70 was associated with a ninefold increase in the odds of alopecia (OR = 9.213, 95% CI = 3.357–25.282, *P* < 0.001), controlling for age and gender. However, this OR should be interpreted cautiously, as the small sample size may lead to an overestimation of the true effect size. (Table 3).[3]

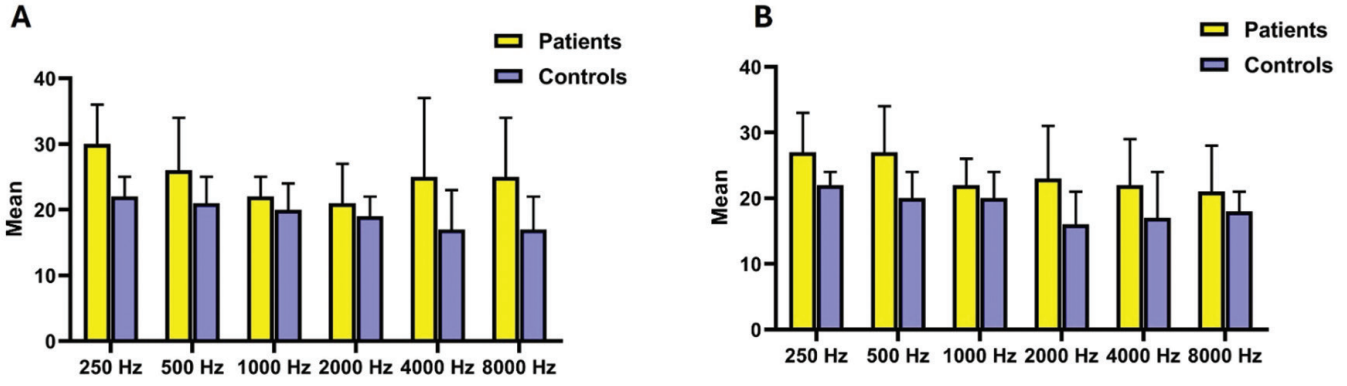

Figure 1. (A) Right and (B) left ear auditory thresholds in the studied groups
Hz: Hertz

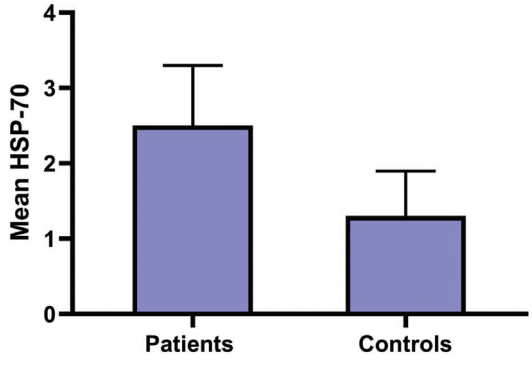

Figure 2. HSP-70 in the studied groups
HSP-70: Heat shock protein 70

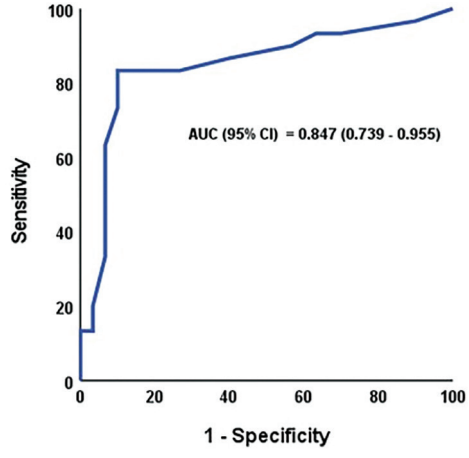

Figure 3. Receiver operating characteristic analysis of heat shock protein 70 to predict alopecia
AUC: Area under the curve, CI: Confidence interval

Table 2. Correlation between serum level of HSP-70 and other parameters

	Serum level of HSP-70	
	r	*P*
Age (years)	-0.071	0.711
Disease duration	0.504	**0.004***
SALT score	-0.095	0.618
Rt 250	-0.247	0.188
Rt 500	0.085	0.655
Rt 1000	0.13	0.492
Rt 2000	-0.141	0.456
Rt 4000	-0.198	0.295
Rt 8000	-0.118	0.534
Lt 250	0.076	0.691
Lt 500	0.037	0.845
Lt 1000	-0.012	0.952
Lt 2000	-0.283	0.13
Lt 4000	-0.213	0.259
Lt 8000	-0.217	0.249

*Significant *P*-value at $P < 0.05$; r: Correlation coefficient, *P*: Probability value, Rt: Right, Lt: Left, HSP-70: Heat shock protein 70, SALT: Severity of Alopecia Tool

Table 3. Multivariate logistic regression analysis to predict alopecia

	OR (95% CI)	*P*-value
Age (years)	0.946 (0.873–1.026)	0.179
Gender	1.949 (0.388–9.801)	0.418
Serum level of HSP-70	9.213 (3.357–25.282)	**< 0.001***

*Significant *P*-value at $P < 0.05$, OR: Odds ratio, CI: Confidence interval, HSP-70: Heat shock protein 70, *P*-value: Probability value

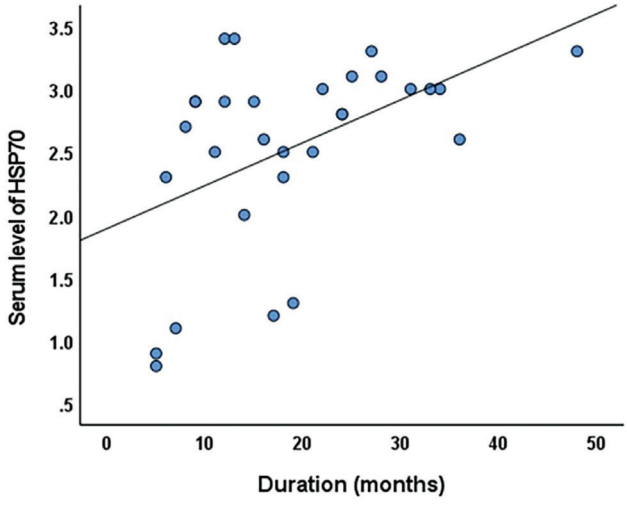

Figure 4. Correlation between HSP-70 and duration
HSP-70: Heat shock protein 70

Discussion

The current study explored the role of circulating HSP-70 in the pathogenesis of AA, its correlation with disease severity, and its potential association with auditory function. Our findings reveal a statistically significant elevation of serum HSP-70 in AA patients compared with healthy controls and significantly higher mean auditory thresholds across various frequencies in these patients. Furthermore, HSP-70 demonstrated robust diagnostic utility for AA, correlating positively with disease duration. These results contribute to the growing body of evidence suggesting a complex interplay among immune responses, stress proteins, and systemic manifestations in autoimmune conditions such as AA. However, given the cross-sectional design, causality cannot be inferred; elevated HSP-70 may simply reflect systemic inflammation, and our findings should be interpreted as hypothesis-generating.

We demonstrated a significant elevation of serum HSP-70 levels in patients with AA compared to healthy controls (2.5 ± 0.8 ng/mL vs. 1.3 ± 0.6 ng/mL; $P < 0.001$). This finding aligns with previous research suggesting the involvement of HSP-70 in autoimmune diseases, including AA.[5,8,9] HSPs are known as molecular chaperones, crucial for maintaining cellular homeostasis and protecting cells from various stressors. However, their role in autoimmune conditions is complex and can be dual-edged; while intracellular HSP-70 protects cells, extracellular or cell surface-anchored HSP-70 can activate immunological responses.[6]

The observed increase in HSP-70 in AA patients could reflect an ongoing cellular stress response within the hair follicles or a systemic immune activation. Given that AA is an immune-mediated disorder characterized by the loss of hair follicle immunological privilege, the elevated HSP-70 might be a compensatory mechanism to mitigate cellular damage or, conversely, contribute to the autoimmune cascade by acting as an autoantigen or an immune stimulator.[3,6] While our study demonstrates a significant association between elevated HSP-70 and AA, further research is needed to determine whether this increase is specific to AA pathogenesis or represents a broader response to systemic stress. Future studies could investigate the correlation between HSP-70 and other specific markers of AA activity or of other autoimmune conditions to better delineate its specificity.

The positive correlation between HSP-70 levels and disease duration ($r = 0.504$, $P = 0.004$) further supports its involvement in the chronicity of AA, suggesting that sustained cellular stress or immune dysregulation might be features of long-standing disease.

These results are consistent with Ghaderi[8] and Elgendy et al.[9], who also reported increased HSP-70 levels in AA

patients. These studies, along with ours, highlight HSP-70 as a potential adjunct biomarker for AA. The robust diagnostic utility demonstrated by ROC analysis (AUC = 0.847, $P < 0.001$) with an optimal diagnostic threshold exceeding 1.4 ng/mL indicates that serum HSP-70 could serve as a valuable tool to aid in the diagnosis of AA, particularly in differentiating it from other forms of hair loss or monitoring disease activity. However, external validation in larger, independent cohorts is mandatory before HSP-70 can be considered for clinical diagnostic use.

Another notable finding of our study is that AA patients exhibited statistically significantly higher mean auditory thresholds across various frequencies than controls. While this suggests a potential link between AA and auditory dysfunction, specifically SNHL, these statistically significant differences do not necessarily indicate clinically significant SNHL for all individuals; mild dB differences may not be clinically meaningful. This association is biologically plausible given the shared embryological origin and immunological characteristics of melanocytes in hair follicles and the inner ear.[4] Melanocytes play a crucial role in maintaining endocochlear potential and protecting against free radical damage in the inner ear.[4,10]

The immune-mediated destruction of melanocytes, a hallmark of AA pathogenesis, could extend to cochlear melanocytes, leading to SNHL. This hypothesis is supported by observations in other autoimmune conditions like vitiligo, where melanocyte depletion is linked to hearing loss.[4] Previous systematic reviews and meta-analyses have also explored the association between AA and SNHL, with some studies indicating a higher prevalence of hearing impairment in AA patients.[4,10,11] Our findings, showing elevated auditory thresholds in both right and left ears across multiple frequencies, further strengthen this proposed connection.

However, our study failed to find a significant correlation between serum HSP-70 levels and auditory thresholds or between SALT score and hearing thresholds. This lack of direct correlation might suggest that, while both AA and auditory dysfunction may share underlying immune-mediated mechanisms involving melanocytes, their specific pathways or the timing of manifestation may differ. For instance, while melanocyte destruction in the scalp leading to hair loss might be a more rapid and acute process, the impact on inner ear melanocytes and subsequent hearing loss could be more insidious and develop over a longer period. It is also possible that HSP-70's role in auditory dysfunction is more indirect or involves different isoforms or cellular compartments not captured by circulating serum levels. Further research, perhaps involving more direct assessment of cochlear function or specific immune markers related to inner ear melanocytes, would be beneficial to fully elucidate this complex relationship.

Study Limitations

Despite these significant findings, our study has several limitations that warrant consideration. First, the restricted sample size (30 AA patients and 30 controls) and single-centre recruitment limit the generalizability of the findings. This small sample size also increases the risk of model overfitting in logistic regression and necessitates external validation of the ROC model. Second, the cross-sectional design precludes the establishment of causality. While we observed associations between elevated HSP-70 and AA, and between AA and auditory dysfunction, we cannot definitively conclude that HSP-70 causes AA or that AA directly causes hearing loss. Longitudinal studies are imperative for understanding the temporal relationships and causal pathways. Furthermore, our patient cohort, characterized by the absence of nail involvement, comorbidities, or a family history of AA, may not fully represent the broad spectrum of the AA population. This is a consequence of our stringent exclusion criteria, designed to minimize confounding factors and focus on the primary research questions. Future studies should aim to include a more heterogeneous AA population to enhance the generalizability of the findings.

Furthermore, while serum HSP-70 levels provide valuable insights, they may not fully capture the localized expression or specific cellular roles of HSP-70 within the hair follicles or inner ear. The absence of assessment of other systemic inflammatory markers (e.g., CRP, ESR) is a limitation, as elevated HSP-70 could reflect systemic inflammation or other unidentified stressors. Future research should include a comprehensive panel of inflammatory markers to better contextualize HSP-70 levels in AA pathogenesis. Future research could also explore tissue-specific HSP-70 expression, different HSP-70 isoforms, and their interactions with various immune cells and pathways involved in AA pathogenesis and auditory function. Advanced audiological assessments beyond PTA, such as otoacoustic emissions or auditory brainstem responses, could provide a more detailed understanding of the cochlear and neural auditory integrity in AA patients.

Investigating genetic polymorphisms related to HSP-70 and their association with AA susceptibility and severity, as well as with hearing loss, could also offer valuable insights.[12]

Finally, the precise mechanistic contributions of HSP-70 to AA pathogenesis and its nuanced relationship with auditory function remain to be fully elucidated. Further experimental studies, including *in vitro* and *in vivo* models, are needed to dissect the molecular mechanisms by which HSP-70 influences immune responses, melanocyte function, and hair follicle integrity in the context of AA. Understanding these mechanisms could pave the way for novel therapeutic targets and interventions for AA and its comorbidities.

Conclusion

Our study provides compelling evidence that elevated circulating HSP-70 levels are a promising diagnostic biomarker for AA and correlate with disease chronicity. We also highlight a significant association between AA and elevated auditory thresholds, suggesting a potential link to SNHL. While further research is warranted to establish causality and elucidate the underlying mechanisms, these findings underscore the systemic nature of AA and the potential for HSP-70 to serve as both a diagnostic marker and therapeutic target.

Ethics

Ethics Committee Approval: The Benha University Research Ethics Committee approved the protocol (approval number: Ms 17-10-2023, approved on October 17, 2023; final document issued: 29.12.2024).

Informed Consent: All subjects provided signed informed consent prior to participation.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.G.A., S.E.A., A.O.A.E-G., R.M.A.; Concept: A.M.H., G.M.S.; Design: A.M.H., G.M.S.; Data Collection or Processing: M.G.A-S., S.E.A., G.M.S.; Analysis or Interpretation: A.O.A.E-G., G.M.S.; Literature Search: A.O.A.E-G., R.M.A., G.M.S.; Writing: A.M.H., A.O.A.E-G., R.M.A., G.M.S.

Conflict of Interest: The authors declared that they have no conflict of interest.

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