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Research Article

Hearing Assessment in Patients with Allergic Rhinitis: A Hospital-Based Study of Auditory Dysfunction and Its Reversibility after Eight Weeks of Standard Pharmacotherapy

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ABSTRACT
Background: Allergic rhinitis (AR) is an IgE-mediated inflammatory disorder of the nasal mucosa. The anatomical and functional continuity between the nasal cavity, nasopharynx, Eustachian tube and middle ear provides a plausible route by which nasal inflammation may compromise auditory function, yet hearing is seldom assessed during routine management of AR.
Objectives: To characterise auditory function in adults with AR, to examine whether nasal symptom severity and disease duration relate to audiological abnormality, and to determine whether audiological measures change after eight weeks of standard pharmacotherapy.
Methods: A hospital-based, prospective, single-arm observational study enrolled 150 adults aged 18–65 years with AR diagnosed according to ARIA criteria. Baseline assessment comprised history, otorhinolaryngological examination, otoscopy, tuning fork tests, pure tone audiometry (PTA) at 0.5–8 kHz, 226 Hz tympanometry and distortion product otoacoustic emissions (DPOAE). Symptom burden was quantified with the Total Nasal Symptom Score (TNSS) and disease control with the Rhinitis Control Assessment Test (RCAT). All participants received an intranasal corticosteroid, an oral antihistamine and a leukotriene receptor antagonist for eight weeks, after which every assessment was repeated. Paired t-tests, McNemar and McNemar–Bowker tests, the chi-square test and Pearson correlation were applied, with $p < 0.05$ taken as significant.
Results: Mean age was 31.8 ± 9.3 years, 56.7% were male, 64.0% had persistent AR and mean TNSS was 8.7 ± 1.9. At baseline, some degree of hearing loss was present in 58/150 (38.7%) right and 62/150 (41.3%) left ears; conductive loss predominated (22.7% and 25.3%), with sensorineural loss in 12.0% and 13.3%. Type C tympanograms were recorded in 23.3% of ears in each side and type B in 13.3% and 14.7%. After treatment, the proportion of ears with normal thresholds rose from 61.3% to 80.7% on the right and from 58.7% to 78.0% on the left ($p = 0.01$). Threshold gains were largest at 500 Hz (6.1 dB right, 6.2 dB left; $p = 0.01$) and absent at 8 kHz (1.1 dB; $p = 0.17$ and 0.19). Type A tympanograms increased to 75.3% and 72.7%, and DPOAE pass rates rose from 67.3% to 80.7% ($p = 0.02$) and from 64.7% to 78.7% ($p = 0.03$). The reduction in TNSS correlated modestly with PTA improvement ($r = +0.39$, $p = 0.02$ right; $r = +0.36$, $p = 0.03$ left). Baseline hearing loss increased with AR duration ($p = 0.03$ right, $p = 0.04$ left).
Conclusion: Approximately two in five ears of adults with AR showed measurable hearing loss, predominantly conductive and frequency-weighted towards the lower frequencies, and largely reversible with eight weeks of anti-inflammatory therapy. A smaller, high-frequency component persisted. Audiological screening is warranted in patients with persistent or long-standing AR, and the absence of a control arm means that the observed improvement should be confirmed in a controlled study.

Keywords: Allergic Rhinitis, Hearing Loss, Eustachian Tube Dysfunction, Pure Tone Audiometry, Tympanometry, Otoacoustic Emissions.

INTRODUCTION

Allergic rhinitis (AR) is among the most common chronic inflammatory disorders of the upper respiratory tract, characterised by nasal obstruction, sneezing, rhinorrhoea and nasal itching, often accompanied by ocular symptoms. It arises from an immunoglobulin E (IgE)-mediated hypersensitivity response to inhaled allergens such as pollen, house dust mite, moulds and animal dander [1]. The condition affects an estimated 10–40% of the population in different settings, imposes substantial costs through impaired school and work productivity, and is a frequent reason for primary care and specialist consultation [2]. Global burden estimates confirm that allergic disorders remain a major and persistent source of morbidity across all age strata [3].

Although AR has traditionally been regarded as a disease confined to the nasal cavity, it is now more usefully understood as a regional, and in some respects systemic, inflammatory condition capable of involving structures that are anatomically and physiologically continuous with the nose. The Eustachian tube connects the middle ear cavity to the nasopharynx, and its functions pressure equalisation across the tympanic membrane, middle ear ventilation and clearance of secretions depend on an unobstructed tubal orifice and a mucosa capable of normal active opening [4]. Mucosal oedema and congestion around the tubal ostium in AR may therefore impair opening during swallowing and yawning, generate negative middle ear pressure, and predispose to effusion and conductive hearing loss. This is not merely a theoretical sequence: controlled intranasal provocation with house dust mite has been shown to produce both nasal obstruction and objectively measured Eustachian tube dysfunction in sensitised adults, establishing a direct causal link between an allergic nasal reaction and tubal compromise [5].

Clinical observation supports the same association. Allergic rhinitis is highly prevalent among children with persistent otitis media with effusion, in whom hearing thresholds are significantly poorer than in non-allergic controls and improve appreciably once allergy-directed therapy is started [6]. Similar associations have been reported between AR and otitis media with effusion in case-control settings [7], and AR has been identified as an independent risk factor for chronic suppurative otitis media in adults [8].

Interest has more recently extended beyond the middle ear. The cochlea was long regarded as immunologically privileged, but it is now recognised to contain a resident macrophage population and to recruit circulating monocytes under stress, organising inflammatory responses that can produce collateral injury to cochlear cells [9]. Macrophage activation and associated cytokine release in the inner ear are increasingly implicated in the pathogenesis of non-infective cochlear injury [10]. Against this background, several audiological studies in AR have described high-frequency sensorineural threshold shifts, abnormal otoacoustic emissions and altered auditory brainstem responses, suggesting that the seat of damage may in part be cochlear rather than purely conductive [11–13]. Sub-clinical, or "hidden", auditory dysfunction has been demonstrated in adolescents with AR using extended high-frequency audiometry, distortion product otoacoustic emissions (DPOAE), electrocochleography and speech-in-noise testing, in the absence of any subjective hearing complaint [14]. Isolated reports of sensorineural loss recovering after anti-allergic therapy raise the further possibility that at least part of this dysfunction is reversible [15].

Despite this accumulating evidence, audiological evaluation is not part of routine AR care. Patients frequently attribute mild fluctuating hearing change or aural fullness to transient congestion and do not report it, while clinicians understandably prioritise nasal symptoms. The consequence is a gap between what is measurable and what is measured. Few studies have combined a standardised nasal severity instrument with a complete audiological battery and then repeated the entire battery after a defined course of therapy, which would allow the reversibility of any deficit to be assessed directly.

The present study was therefore designed to determine the prevalence and pattern of auditory dysfunction in adults with AR, to examine its relationship with nasal symptom severity and disease duration, and to assess whether middle ear and cochlear measures change after eight weeks of guideline-concordant pharmacotherapy.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based, prospective, single-arm observational study with paired pre- and post-treatment assessment, conducted in the Department of Otorhinolaryngology of a tertiary care teaching hospital. Baseline data were collected at the time of enrolment and all

assessments were repeated after eight weeks of therapy. The study was approved by the Institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant before enrolment.

Participants

One hundred and fifty consecutive adults aged 18–65 years with a clinical diagnosis of AR, classified according to the Allergic Rhinitis and its Impact on Asthma (ARIA) criteria as intermittent or persistent and as mild or moderate–severe [1,2], were enrolled. Patients were excluded if they had a perforated tympanic membrane, previous otological surgery, chronic suppurative otitis media, a history of ototoxic drug exposure, occupational or recreational noise exposure, head injury, known sensorineural hearing loss of identified aetiology, nasal polyposis, gross septal deviation requiring surgery, acute upper respiratory infection within four weeks, or pregnancy. Patients on systemic corticosteroids or already receiving an intranasal corticosteroid were also excluded.

Clinical and Symptom Assessment

All participants underwent history taking with particular attention to duration of AR, aural symptoms (fullness, tinnitus, fluctuating hearing) and atopic comorbidity, followed by a complete otorhinolaryngological examination including anterior rhinoscopy and otoscopy. Tuning fork tests (Rinne with a 512 Hz fork, and Weber) were performed as a bedside screen.

Symptom severity was quantified using the Total Nasal Symptom Score (TNSS), in which nasal congestion, rhinorrhoea, sneezing and nasal itching are each rated from 0 (absent) to 3 (severe), giving a total of 0–12; the minimal clinically important difference for this instrument has been estimated at approximately 0.55 units [16]. Disease control was recorded using the Rhinitis Control Assessment Test (RCAT), a validated six-item patient-reported instrument scored from 6 to 30, with higher scores indicating better control [17]. TNSS was pre-specified as the severity variable for correlation analysis; RCAT was collected to describe control status.

Audiological Assessment

Pure tone audiometry (PTA) was performed in a sound-treated room using a calibrated clinical audiometer. Air conduction thresholds were obtained at 500 Hz, 1, 2, 4 and 8 kHz and bone conduction thresholds at 500 Hz to 4 kHz. Hearing was classified as normal (< 25 dB HL), mild (25–40 dB HL) or moderate (> 40 dB HL) on the four-frequency average, and as conductive, sensorineural or mixed on the basis of the air–bone gap.

Tympanometry was performed with a 226 Hz probe tone over a pressure sweep of +200 to −300 daPa and curves were classified by the Jerger convention as type A, As, Ad, B or C, a type C curve being defined by a peak pressure below −100 daPa [18]. Distortion product otoacoustic emissions were recorded and reported as pass or refer according to standard signal-to-noise criteria. Ears were analysed separately throughout, giving 150 right and 150 left ears.

Intervention and Follow-Up

After baseline assessment, all participants received standard pharmacotherapy for AR in line with ARIA recommendations [2]: an intranasal corticosteroid, an oral second-generation antihistamine and a leukotriene receptor antagonist, for eight weeks. No placebo or comparator arm was used. Adherence was reinforced at a mid-point telephone contact. At the end of eight weeks, otoscopy, tuning fork tests, PTA, tympanometry, DPOAE, TNSS and RCAT were repeated by the same assessors using the same equipment.

Statistical Analysis

Data were analysed using SPSS. Continuous variables are presented as mean ± standard deviation and categorical variables as frequency and percentage. Paired pre- and post-treatment threshold comparisons were made with the paired t-test. Dichotomous paired categorical outcomes (Rinne result, DPOAE pass/refer) were compared with the McNemar test and polytomous paired categorical outcomes (otoscopic findings, hearing severity, hearing loss type, tympanogram type) with the McNemar–Bowker test. The association between duration of AR and baseline hearing loss was tested with the chi-square test. The relationship between the change in TNSS and the change in mean PTA threshold was examined using the Pearson correlation coefficient. A two-sided p-value below 0.05 was considered statistically significant.

RESULTS

Socio-Demographic and Clinical Profile

The mean age of the 150 participants was 31.8 ± 9.3 years, with a clear predominance of younger adults: 41.3% were aged 18–30 years and 38.7% were aged 31–45 years, while only 20.0% were older than 45 years. Males constituted 56.7% of the cohort. Persistent AR accounted for 64.0% of cases, nearly twice the proportion with intermittent disease. Mean duration of AR was 2.4 ± 1.1 years and mean baseline TNSS was 8.7 ± 1.9, reflecting a moderate to severe symptom burden (Table 1).

Table 1. Socio-Demographic and Clinical Characteristics of Patients with Allergic Rhinitis (N = 150)

Variable	Category	n (%) or Mean ± SD
Mean age (years)	—	31.8 ± 9.3
Age group (years)	18–30	62 (41.3)
	31–45	58 (38.7)
	> 45	30 (20.0)
Gender	Male	85 (56.7)
	Female	65 (43.3)
Mean duration of AR (years)	—	2.4 ± 1.1
ARIA classification	Intermittent	54 (36.0)
	Persistent	96 (64.0)
Mean TNSS	—	8.7 ± 1.9

AR, allergic rhinitis; ARIA, Allergic Rhinitis and its Impact on Asthma; SD, standard deviation; TNSS, Total Nasal Symptom Score.

Baseline Hearing Status

At baseline, normal hearing was present in 92/150 (61.3%) right and 88/150 (58.7%) left ears. Conductive hearing loss was the commonest abnormality, affecting 34 (22.7%) right and 38 (25.3%) left ears. Sensorineural loss was identified in 18 (12.0%) right and 20 (13.3%) left ears, and mixed loss was least frequent, in 6 (4.0%) and 4 (2.7%) ears respectively. Overall, 58/150 (38.7%) right and 62/150 (41.3%) left ears had some degree of hearing loss (Table 2).

Table 2. Baseline Hearing Status on Pure Tone Audiometry (Ear-Wise)

Hearing status	Right ear, n (%)	Left ear, n (%)
Normal hearing	92 (61.3)	88 (58.7)
Conductive hearing loss	34 (22.7)	38 (25.3)
Sensorineural hearing loss	18 (12.0)	20 (13.3)
Mixed hearing loss	6 (4.0)	4 (2.7)
Any hearing loss	58 (38.7)	62 (41.3)

Otoscopic Findings Before and After Treatment

Otoscopic appearances improved markedly over the treatment period. In the right ear, the proportion with a normal tympanic membrane rose from 62/150 (41.3%) to 120/150 (80.0%) (p = 0.01), while dullness fell from 50 (33.3%) to 16 (10.7%) (p = 0.01) and retraction from 30 (20.0%) to 10 (6.7%) (p = 0.02). The left ear followed the same pattern, with normal appearances rising from 58 (38.7%) to 116 (77.3%) (p = 0.01). Visible fluid or air bubbles were uncommon at baseline and their reduction did not reach statistical significance in either ear (Table 3).

Table 3. Otoscopic Findings Before and After Treatment (Ear-Wise, N = 150 Ears per Side)

Otoscopic finding	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Normal tympanic membrane	62 (41.3)	120 (80.0)	0.01
Dull tympanic membrane	50 (33.3)	16 (10.7)	0.01
Retracted tympanic membrane	30 (20.0)	10 (6.7)	0.02
Fluid / air bubbles	8 (5.4)	4 (2.7)	0.29
Left ear			
Normal tympanic membrane	58 (38.7)	116 (77.3)	0.01

Dull tympanic membrane	54 (36.0)	18 (12.0)	0.01
Retracted tympanic membrane	30 (20.0)	12 (8.0)	0.02
Fluid / air bubbles	8 (5.3)	4 (2.7)	0.31

McNemar–Bowker test. $p < 0.05$ considered statistically significant.

Tuning Fork Findings

Bedside tuning fork testing showed a consistent shift towards normality. In the right ear, a positive Rinne was recorded in 106/150 (70.7%) before and 133/150 (88.7%) after treatment ($p = 0.01$), with negative results falling from 36 (24.0%) to 12 (8.0%) ($p = 0.01$). In the left ear, positive Rinne rose from 102 (68.0%) to 130 (86.7%) and negative Rinne fell from 40 (26.7%) to 14 (9.3%) (both $p = 0.01$). Equivocal results were few and changed little (Table 4).

Table 4. Rinne Test Before and After Treatment (Ear-Wise, N = 150 Ears per Side)

Rinne result	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Positive	106 (70.7)	133 (88.7)	0.01
Negative	36 (24.0)	12 (8.0)	0.01
Equivocal	8 (5.3)	5 (3.3)	0.45
Left ear			
Positive	102 (68.0)	130 (86.7)	0.01
Negative	40 (26.7)	14 (9.3)	0.01
Equivocal	8 (5.3)	6 (4.0)	0.61

McNemar test. $p < 0.05$ considered statistically significant.

Pure Tone Thresholds

Mean air conduction thresholds improved significantly at the lower and mid frequencies. In the right ear, thresholds fell from 28.4 ± 7.6 to 22.3 ± 6.8 dB HL at 500 Hz (gain 6.1 dB, $p = 0.01$) and from 27.1 ± 7.2 to 21.9 ± 6.5 dB HL at 1 kHz (gain 5.2 dB, $p = 0.01$), with smaller but significant gains at 2 kHz (3.9 dB, $p = 0.02$) and 4 kHz (2.8 dB, $p = 0.03$). The change at 8 kHz was 1.1 dB and did not reach significance ($p = 0.17$). The left ear mirrored this gradient, with a 6.2 dB gain at 500 Hz ($p = 0.01$) and no significant change at 8 kHz (1.1 dB, $p = 0.19$). The magnitude of improvement therefore declined monotonically with increasing frequency (Table 5, Figure 1).

Table 5. Mean Pure Tone Audiometry Thresholds Before and After Treatment, In Db HL (Ear-Wise, N = 150 Ears per Side)

Frequency	Pre-treatment, Mean ± SD	Post-treatment, Mean ± SD	Mean gain (dB)	p-value
Right ear				
500 Hz	28.4 ± 7.6	22.3 ± 6.8	6.1	0.01
1 kHz	27.1 ± 7.2	21.9 ± 6.5	5.2	0.01
2 kHz	25.3 ± 6.9	21.4 ± 6.3	3.9	0.02
4 kHz	24.6 ± 6.7	21.8 ± 6.4	2.8	0.03
8 kHz	23.8 ± 6.5	22.7 ± 6.3	1.1	0.17
Left ear				
500 Hz	29.2 ± 7.9	23.0 ± 7.0	6.2	0.01
1 kHz	27.8 ± 7.4	22.2 ± 6.6	5.6	0.01
2 kHz	26.0 ± 7.1	21.7 ± 6.4	4.3	0.01
4 kHz	25.2 ± 6.8	22.1 ± 6.5	3.1	0.03
8 kHz	24.1 ± 6.6	23.0 ± 6.4	1.1	0.19

Paired t-test. SD, standard deviation. $p < 0.05$ considered statistically significant.

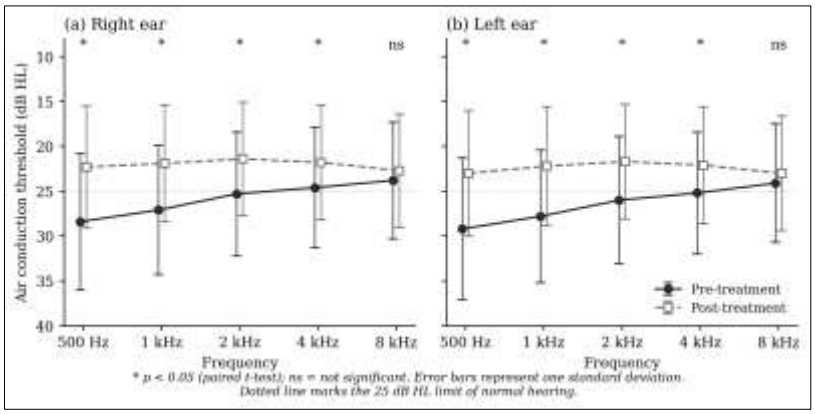

Figure 1. Mean Pure Tone Air Conduction Thresholds From 500 Hz To 8 Khz Before And After Eight Weeks Of Treatment, Shown Separately For (A) The Right Ear and (B) The Left Ear (N = 150 Ears Per Side). Error Bars Denote One Standard Deviation And The Dotted Line Marks The 25 Db HL Limit Of Normal Hearing. Thresholds Improved Significantly At 500 Hz To 4 Khz In Both Ears, With The Magnitude Of Improvement Declining Monotonically As Frequency Increased; The Change At 8 Khz Was Not Significant (Right Ear P = 0.17, Left Ear P = 0.19). * P < 0.05, Paired T-Test; Ns, Not Significant.

Severity and Type of Hearing Loss

The distribution of hearing loss severity shifted significantly towards normal. In the right ear, normal hearing increased from 92/150 (61.3%) to 121/150 (80.7%) (p = 0.01), mild loss fell from 50 (33.3%) to 26 (17.3%) and moderate loss from 8 (5.4%) to 3 (2.0%). In the left ear, normal hearing increased from 88 (58.7%) to 117 (78.0%) (p = 0.01) and mild loss fell from 55 (36.7%) to 30 (20.0%) (Table 6). Analysed by type, the conductive component accounted for most of the gain. Right-sided conductive loss fell from 34 (22.7%) to 16 (10.7%) and left-sided from 38 (25.3%) to 19 (12.7%), whereas sensorineural loss showed a smaller reduction, from 18 (12.0%) to 11 (7.3%) on the right and from 20 (13.3%) to 12 (8.0%) on the left. Mixed loss was infrequent throughout (Table 7).

Table 6. Hearing Loss Severity Before and After Treatment (Ear-Wise, N = 150 Ears per Side)

Severity	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Normal (< 25 dB)	92 (61.3)	121 (80.7)	0.01
Mild (25–40 dB)	50 (33.3)	26 (17.3)	
Moderate (> 40 dB)	8 (5.4)	3 (2.0)	
Left ear			
Normal (< 25 dB)	88 (58.7)	117 (78.0)	0.01
Mild (25–40 dB)	55 (36.7)	30 (20.0)	
Moderate (> 40 dB)	7 (4.6)	3 (2.0)	

McNemar–Bowker test applied to the overall distribution within each ear. p < 0.05 considered statistically significant.

Table 7. Type of Hearing Loss before and After Treatment (Ear-Wise, N = 150 Ears per Side)

Type	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Normal hearing	92 (61.3)	121 (80.7)	0.01
Conductive hearing loss	34 (22.7)	16 (10.7)	
Sensorineural hearing loss	18 (12.0)	11 (7.3)	
Mixed hearing loss	6 (4.0)	2 (1.3)	
Left ear			
Normal hearing	88 (58.7)	117 (78.0)	0.01
Conductive hearing loss	38 (25.3)	19 (12.7)	
Sensorineural hearing loss	20 (13.3)	12 (8.0)	
Mixed hearing loss	4 (2.7)	2 (1.3)	

McNemar–Bowker test applied to the overall distribution within each ear. $p < 0.05$ considered statistically significant.

Otoacoustic Emissions

DPOAE outcomes improved in both ears. On the right, pass results rose from 101/150 (67.3%) to 121/150 (80.7%) and refer results fell from 49 (32.7%) to 29 (19.3%) ($p = 0.02$). On the left, pass results rose from 97 (64.7%) to 118 (78.7%), with refer results falling from 53 (35.3%) to 32 (21.3%) ($p = 0.03$). The magnitude of change was comparable between sides (Table 8).

Table 8. Distortion Product Otoacoustic Emission Findings Before and After Treatment (Ear-Wise, N = 150 Ears per Side)

DPOAE result	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Pass	101 (67.3)	121 (80.7)	0.02
Refer	49 (32.7)	29 (19.3)	
Left ear			
Pass	97 (64.7)	118 (78.7)	0.03
Refer	53 (35.3)	32 (21.3)	

McNemar test. DPOAE, distortion product otoacoustic emissions. $p < 0.05$ considered statistically significant.

Tympanometry

Tympanometric findings showed the clearest treatment effect. In the right ear, type A curves increased from 78/150 (52.0%) to 113/150 (75.3%) ($p = 0.01$), type B fell from 20 (13.3%) to 6 (4.0%) ($p = 0.04$) and type C from 35 (23.3%) to 10 (6.7%) ($p = 0.01$). The left ear showed the same pattern, with type A rising from 74 (49.3%) to 109 (72.7%) ($p = 0.01$), type B falling from 22 (14.7%) to 7 (4.7%) ($p = 0.03$) and type C from 35 (23.3%) to 12 (8.0%) ($p = 0.01$). Type As and Ad curves were uncommon and essentially unchanged (Table 9).

Table 9. Tympanometry Findings Before and After Treatment (Ear-Wise, N = 150 Ears per Side)

Tympanogram type	Pre-treatment, n (%)	Post-treatment, n (%)	p-value
Right ear			
Type A	78 (52.0)	113 (75.3)	0.01
Type As	12 (8.0)	15 (10.0)	
Type Ad	5 (3.3)	6 (4.0)	
Type B	20 (13.3)	6 (4.0)	0.04
Type C	35 (23.3)	10 (6.7)	0.01
Left ear			
Type A	74 (49.3)	109 (72.7)	0.01
Type As	14 (9.3)	16 (10.7)	
Type Ad	5 (3.3)	6 (4.0)	
Type B	22 (14.7)	7 (4.7)	0.03
Type C	35 (23.3)	12 (8.0)	0.01

McNemar–Bowker test. $p < 0.05$ considered statistically significant.

Correlation between Symptom Change and Threshold Change

The reduction in TNSS correlated positively and significantly with the improvement in mean PTA threshold in both ears: $r = +0.39$ ($p = 0.02$) on the right and $r = +0.36$ ($p = 0.03$) on the left. Patients with the greatest fall in nasal symptom burden therefore tended to show the greatest threshold gain, although the correlation was moderate and accounted for only about 13–15% of the variance in hearing improvement (Table 10, Figure 2).

Table 10. Correlation between Reduction in TNSS and Improvement in Pure Tone Audiometry Thresholds (Ear-Wise)

Ear	Correlation	r-value	p-value
Right ear	ΔTNSS vs ΔPTA improvement	+0.39	0.02

Left ear	ΔTNSS vs ΔPTA improvement	+0.36	0.03

Pearson correlation. Δ denotes change from baseline to eight weeks. PTA, pure tone audiometry; TNSS, Total Nasal Symptom Score. $p < 0.05$ considered statistically significant.

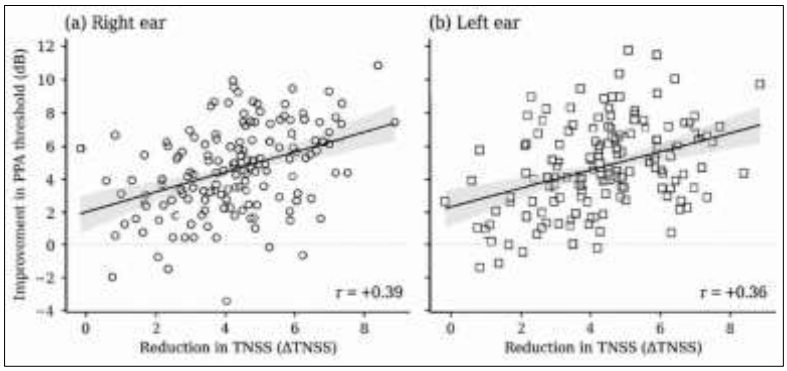

Figure 2. Scatter Plot of the Reduction in Total Nasal Symptom Score (ΔTNSS) Against the Improvement in Mean Pure Tone Audiometry Threshold (ΔPTA, Db) For the Right and Left Ears, With Fitted Regression Lines (Right Ear R = +0.39, P = 0.02; Left Ear R = +0.36, P = 0.03)

Duration of Allergic Rhinitis and Baseline Hearing Loss

Baseline hearing loss, defined as a PTA threshold of 25 dB or more, increased progressively with the duration of AR. In the right ear, it was present in 13/38 (34.2%) ears of patients with disease of less than one year, 34/72 (47.2%) of those with one to three years, and 23/40 (57.5%) of those with more than three years ($p = 0.03$). The left ear showed the same gradient, from 15/38 (39.5%) to 36/72 (50.0%) to 24/40 (60.0%) ($p = 0.04$) (Table 11).

Table 11. Association between Duration of Allergic Rhinitis and Baseline Hearing Loss (Ear-Wise)

Duration of AR	Total ears	Hearing loss, n (%)	p-value
Right ear			
< 1 year	38	13 (34.2)	0.03
1–3 years	72	34 (47.2)	
> 3 years	40	23 (57.5)	
Left ear			
< 1 year	38	15 (39.5)	0.04
1–3 years	72	36 (50.0)	
> 3 years	40	24 (60.0)	

Chi-square test. Hearing loss defined as a pure tone average of ≥ 25 dB HL. AR, allergic rhinitis. $p < 0.05$ considered statistically significant.

DISCUSSION

This study found measurable hearing loss in approximately two in five ears of adults with AR, a conductive pattern in the majority, and substantial reversal of both middle ear and audiometric abnormalities after eight weeks of anti-inflammatory therapy. Three features of the data deserve emphasis: the predominance of the conductive component, the frequency gradient of recovery, and the persistence of a smaller high-frequency deficit.

The Middle Ear Component

The tympanometric profile provides the most coherent account of the findings. Nearly a quarter of ears showed a type C curve at baseline, indicating negative middle ear pressure, and a further 13–15% showed a type B curve consistent with effusion. Both categories fell steeply after treatment, while type A curves rose by more than twenty percentage points in each ear. This is precisely the sequence predicted by the Eustachian tube hypothesis: peritubal mucosal oedema impairs active tubal opening, negative pressure develops, effusion follows in a proportion of ears, and the process reverses when the mucosal inflammation is suppressed. Experimental support for the first step of this chain is direct — intranasal challenge with house dust mite in sensitised adults produced

objectively measured tubal obstruction in more than half of ears with previously normal function [5] — and the clinical corollary has been observed in children with persistent otitis media with effusion, in whom hearing thresholds improved significantly after allergy-directed treatment [6].

The frequency gradient of threshold recovery is consistent with this mechanism. Gains were largest at 500 Hz and declined steadily through 1, 2 and 4 kHz, becoming negligible at 8 kHz. A negative-pressure or effusion-related conductive loss characteristically affects the lower frequencies most, and its resolution should therefore produce exactly this pattern. Our findings agree with a hospital-based series in which mild conductive loss was the commonest audiological abnormality in AR and in which resolution of secretory otitis media was accompanied by return of normal hearing [11].

The Sensorineural Component

Sensorineural loss was present in 12.0–13.3% of ears at baseline and fell only modestly after treatment, and the 8 kHz threshold did not change significantly in either ear. This residual, high-frequency-weighted deficit is consistent with a body of work suggesting genuine cochlear involvement in AR. In a study of 200 subjects, high-frequency sensorineural loss and abnormal otoacoustic emissions were more prevalent in AR patients than controls, the authors concluding that the likely seat of damage was the inner ear [12]. Comparable abnormalities of transient evoked and distortion product emissions, together with altered auditory brainstem response latencies, have been reported in other case-control series [13,19]. More recently, adolescents with AR were shown to have poorer extended high-frequency thresholds, reduced DPOAE signal-to-noise ratios at 6–10 kHz, raised summating-to-action potential ratios and worse speech-in-noise performance than matched controls, a constellation described as hidden hearing loss [14].

A biological substrate for such involvement is now plausible. The cochlea maintains a resident macrophage population and recruits circulating monocytes under stress, and the resulting inflammatory response, although protective in intent, can produce off-target injury to cochlear cells [9]. Macrophage activation and cytokine release in the inner ear have been implicated in several forms of non-infective cochlear damage [10]. Isolated reports of sensorineural loss recovering substantially and durably after intranasal corticosteroid, systemic corticosteroid and antihistamine therapy suggest that at least part of this cochlear dysfunction may be inflammatory and reversible rather than structural [15].

One caveat must be stated plainly. Otoacoustic emissions require an intact forward and reverse transmission path through the middle ear, so an ear with negative middle ear pressure or effusion may yield a refer result despite entirely normal outer hair cell function. In this cohort, the ears whose tympanograms normalised and the ears whose DPOAE results converted from refer to pass are likely to overlap considerably. The improvement in DPOAE pass rate should therefore not be read as direct evidence of cochlear recovery; a proportion of it almost certainly reflects restored middle ear transmission. Disentangling the two would require analysis restricted to ears with type A tympanograms at both time points, which was not pre-specified here.

Symptom Severity, Disease Duration and Clinical Implications

The correlation between symptom improvement and threshold improvement was statistically significant but moderate ($r = +0.36$ to $+0.39$), explaining only a small share of the variance. This is informative in itself: nasal symptom burden is an imperfect proxy for auditory involvement, and a patient whose TNSS improves little may still gain hearing, while a patient with a large symptomatic response may not. It follows that clinicians cannot reliably infer auditory status from nasal symptoms alone.

The association between longer disease duration and baseline hearing loss, rising from roughly a third of ears in patients with under a year of AR to nearly 60% in those with more than three years, points in the same direction and echoes a previous report of a relationship between hearing loss severity and duration of AR [11]. Although cross-sectional and therefore incapable of establishing causation, it is compatible with cumulative injury from repeated or sustained tubal dysfunction and argues for earlier rather than later audiological assessment.

Taken together, these findings support incorporating a brief audiological screen — otoscopy, tympanometry and pure tone audiometry — into the assessment of patients with persistent, moderate-to-severe or long-standing AR, particularly when aural fullness, tinnitus or fluctuating hearing are reported.

Given that type C tympanograms also become more frequent with age and are independently associated with poorer thresholds [18], the young mean age of this cohort makes age an unlikely explanation for the abnormalities observed here.

Limitations

Several limitations temper these conclusions. The most important is the absence of a control or comparator arm. In an uncontrolled, unblinded, single-arm design, regression to the mean, natural seasonal fluctuation in allergen exposure, spontaneous resolution and learning effects on repeated audiometry cannot be separated from a genuine treatment effect, and the magnitude of improvement reported here is therefore likely to be an upper bound. Second, atopy was defined clinically according to ARIA criteria without skin prick testing or serum specific IgE, so a proportion of participants may have had non-allergic rhinitis. Third, the analysis treated right and left ears of the same patient as independent observations; this ignores within-patient clustering and will tend to understate standard errors, and a mixed-effects or generalised estimating equation approach would be more appropriate. Fourth, the audiological battery did not include extended high-frequency audiometry, auditory brainstem response or speech-in-noise testing, which are the measures most likely to detect the sub-clinical cochlear involvement suggested by recent work [14]. Fifth, follow-up was limited to eight weeks, so neither the durability of the gains nor the long-term trajectory of the residual high-frequency deficit could be assessed. Finally, although RCAT was recorded, correlation analyses were pre-specified around TNSS, and control status was not analysed as an independent predictor.

CONCLUSION

Measurable hearing loss was present in approximately 40% of ears in adults with allergic rhinitis, predominantly conductive in type, weighted towards the lower frequencies, and accompanied by a high prevalence of type B and type C tympanograms indicating Eustachian tube dysfunction. Eight weeks of intranasal corticosteroid, oral antihistamine and leukotriene receptor antagonist therapy was associated with normalisation of otoscopic and tympanometric findings and with significant threshold gains at 500 Hz to 4 kHz, while the 8 kHz threshold and a minority of ears with sensorineural loss did not improve. Longer disease duration was associated with a higher baseline prevalence of hearing loss. These observations support routine audiological evaluation in patients with persistent, severe or long-standing allergic rhinitis, and indicate that much of the associated auditory deficit is inflammatory and potentially reversible. Because the design was uncontrolled, the extent of that reversibility should be confirmed in a randomised or at least controlled study incorporating extended high-frequency audiometry and longer follow-up.

Declarations

Ethics Approval and Consent to Participate: The study was approved by the Institutional Ethics Committee of and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for Publication: Not applicable.

Availability of Data and Materials: The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests: The authors declare that they have no competing interests.

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