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Inferior Turbinate Hypertrophy and SFAR-Defined Allergic Rhinitis in Chronic Suppurative Otitis Media: A Cross-Sectional Study at Ngoerah Hospital, Bali

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ABSTRACT

Background: Allergic rhinitis (AR) is an IgE-mediated inflammatory disease of the nasal mucosa and a recognised risk factor for chronic suppurative otitis media (CSOM) through Eustachian tube dysfunction, yet Indonesian data on this comorbidity remain scarce.

Objective: To characterise Score for Allergic Rhinitis (SFAR)-defined AR and its association with inferior turbinate hypertrophy among CSOM patients at a tertiary ENT referral centre in Bali.

Methods: A descriptive cross-sectional study, reported in accordance with the STROBE statement, consecutively enrolled 26 CSOM patients attending the ENT outpatient clinic of Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, from November to December 2025. AR was screened using the validated SFAR questionnaire (cut-off ≥7). Data were analysed with Wilson 95% confidence intervals (CI), bivariate odds ratios (OR), multivariable logistic regression, and receiver-operating-characteristic (ROC) analysis.

Results: SFAR-defined AR was identified in 17 patients (65.4%; 95% CI 46.2–80.6). Most were female (65.4%) and aged 30–39 years (26.9%). Predominant symptoms were sneezing (42.3%), rhinorrhoea (34.6%) and nasal obstruction (23.1%); house dust was the leading trigger (94.1%). Inferior turbinate hypertrophy (80.8%) was strongly associated with AR (OR 12.8; 95% CI 1.15–142.6; p=0.034), and the SFAR score discriminated turbinate hypertrophy with an area under the curve of 0.776 (95% CI 0.527–1.00).

Conclusion: Comorbid AR is highly prevalent among CSOM patients. Routine SFAR screening, particularly when inferior turbinate hypertrophy is present, may enable earlier AR detection and more comprehensive middle-ear management in Indonesian ENT practice.

All authors have reviewed and approved the final version of the manuscript.

1. Introduction

Allergic rhinitis (AR) is an immunoglobulin E (IgE)-mediated inflammatory disease of the nasal mucosa and one of the most prevalent chronic respiratory conditions globally, affecting an estimated 10-30% of adults and up to 40% of children, with a rising trend across Asia driven by urbanisation and environmental exposure.[1-2] In tropical Southeast Asia, perennial sensitisation to house-dust mite predominates, sustaining a high year-round symptom burden.[3-4] Chronic suppurative otitis media (CSOM), defined by persistent middle-ear inflammation with a non-intact tympanic membrane and recurrent otorrhoea, remains a leading cause of preventable hearing loss; a recent systematic review estimated a global prevalence of 3.8%, affecting approximately 297 million people, disproportionately in low- and middle-income countries including Indonesia.[5-6]

The pathophysiological link between the upper airway and the middle ear is well established within the unified-airway paradigm. Chronic inflammation of the nasal and nasopharyngeal mucosa in AR promotes mucosal oedema and inferior turbinate hypertrophy, which impair the ventilatory and clearance functions of the Eustachian tube.[7-8] The resulting persistent middle-ear under-ventilation predisposes to effusion and, over time, to suppurative and structural middle-ear disease; a meta-analysis of paediatric risk factors identified atopy as a significant contributor to CSOM development.[9-10] Turbinate-reduction studies have demonstrated measurable improvements in Eustachian tube function after relief of nasal

obstruction, reinforcing the mechanistic relevance of rhinologic inflammation to otologic outcomes.[7-8]

International frameworks, including the International Consensus Statement on Allergy and Rhinology: Allergic Rhinitis (ICAR-AR) 2023, standardise AR classification and endorse structured case-finding among comorbid populations.[11] The Score for Allergic Rhinitis (SFAR) is an eight-item, internationally validated questionnaire (score range 0-16; cut-off ≥7) that identifies AR in epidemiological and clinical settings without requiring allergy testing, making it well suited to resource-conscious ENT clinics.[12-13] A recent Mendelian-randomisation study provided genetic evidence of a causal effect of AR on otitis media, and systematic reviews confirm a consistent association between AR and middle-ear disease.[9,14]

In Indonesia, ENT services are delivered through centres affiliated with the Indonesian Society of Otorhinolaryngology-Head and Neck Surgery (PERHATI-KL), with tertiary referral hospitals such as Prof. Dr. I.G.N.G. Ngoerah General Hospital in Denpasar managing complex otologic disease for Bali and the surrounding Nusa Tenggara region. National studies confirm that house-dust-mite sensitisation and rhinitis are common among Indonesians, yet systematic local data quantifying comorbid AR and evaluating the SFAR questionnaire in CSOM populations remain limited.[15-16] The tropical climate favouring perennial house-dust-mite sensitisation further shapes the local epidemiology of these patients.[3,17]

Addressing this gap, the present study aimed to describe the characteristics of SFAR-defined allergic rhinitis among patients with CSOM, to identify its predominant symptoms and allergen triggers, and to quantify its association with inferior turbinate hypertrophy at the ENT outpatient clinic of Prof. Dr. I.G.N.G. Ngoerah General Hospital, thereby providing novel Indonesian evidence to inform routine AR screening in CSOM.[18-19]

2. Methods

Study design

This was a descriptive, single-centre, cross-sectional study reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies, following COPE and ICMJE standards for the responsible conduct and reporting of research.

Setting and participants

The study was conducted at the ENT Outpatient Clinic of Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia, a tertiary ENT referral centre, over the period November to December 2025. Eligible participants were patients with a clinical diagnosis of CSOM established by history and otoscopic examination (persistent or recurrent otorrhoea through a non-intact tympanic membrane for at least two weeks), consistent with contemporary CSOM criteria.[5-6] Inclusion criteria were all CSOM patients recorded in the medical records who attended the clinic during the study period and were willing to complete the SFAR questionnaire. Exclusion criteria were patients who declined to complete the questionnaire or had incomplete records.

Sample size

The sample size was estimated using the single-population-proportion formula:

$n = Z^2_{\alpha/2} \times P(1 - P) / d^2$

where $Z\alpha/2 = 1.96$ for a 95% confidence level, $P = 0.65$ (expected AR prevalence among CSOM patients from prior comparable studies), and $d = 0.20$ (absolute precision). This yielded a minimum of 22 patients; allowing for approximately 15% non-response, the target was 26 patients. The 26 patients enrolled achieved a two-sided 95% confidence-interval half-width of approximately 18% for the primary prevalence estimate and greater than 80% power to detect a large association (odds ratio of approximately 5) at $\alpha = 0.05$.[9,14]

Sampling

Consecutive sampling was applied: every eligible CSOM patient attending the clinic during the study period who met the inclusion criteria was invited to participate until the accessible population was exhausted. Consecutive sampling was selected to minimise selection bias within a fixed recruitment window in a single tertiary centre.

ORL assessment and instruments

Allergic rhinitis was screened using the SFAR, an eight-item validated questionnaire assessing nasal symptoms, perceived allergic cause, triggers, seasonality, personal and family history of atopy, and prior allergy testing; the total score ranges from 0 to 16, and a score of ≥7 was classified as indicative of AR.[12-13] Predominant nasal symptoms (sneezing, rhinorrhoea, nasal obstruction) and self-reported allergen triggers were recorded from the questionnaire. Inferior turbinate hypertrophy was documented by anterior rhinoscopy and, where indicated, nasal endoscopy performed by the attending otolaryngologist. CSOM was confirmed by otoscopic examination. Diagnostic definitions for AR were aligned with the ICAR-AR framework.[11,19] Because assessment of turbinate hypertrophy is examiner-dependent, examinations were standardised using a common proforma; formal inter-observer reliability (Cohen's kappa) was not computed given the single-centre design and is acknowledged as a limitation.

Confounders

Potential confounders of the AR-CSOM relationship, including age, sex, and inferior turbinate hypertrophy, were identified a priori and controlled through multivariable logistic regression. Occupation was

examined descriptively as a proxy for allergen-exposure environment.

Statistical analysis

Data were analysed using a reproducible numerical pipeline (Python 3.10; NumPy; fixed random seed). Categorical variables were summarised as frequencies and percentages; the SFAR score was summarised as mean ± standard deviation (SD) and median with interquartile range (IQR). Prevalence estimates were reported with 95% Wilson confidence intervals. Bivariate associations between AR status and candidate predictors were assessed using odds ratios (OR) with 95% CI, Fisher exact tests (with the Haldane-Anscombe correction for zero cells), and the Cramer's V effect size. A multivariable logistic-regression model (ridge-stabilised maximum likelihood) estimated adjusted odds ratios, with performance summarised by the Nagelkerke R^2 and the Hosmer-Lemeshow goodness-of-fit test. The discriminatory performance of the SFAR score for inferior turbinate hypertrophy was evaluated by ROC analysis, reporting the area under the curve (AUC) with DeLong 95% CI, the Youden-optimal cut-off, sensitivity, specificity, predictive values, and likelihood ratios. The number needed to screen was derived from the absolute risk difference. All tests were two-sided with $\alpha = 0.05$.

Ethics

This study received ethical approval from the CMHC Ethics Committee, Indonesia (Approval No. CMHC/EC/2025/114). Written informed consent was obtained from all participants prior to completion of the SFAR questionnaire, and the study was conducted in accordance with the Declaration of Helsinki and COPE and ICMJE standards.

3. Results

A total of 26 patients with CSOM were consecutively enrolled between November and December 2025. The cohort comprised 17 women (65.4%) and 9 men (34.6%). The most frequent age group was 30-39 years (7 patients, 26.9%), followed by the 20-29 and 50-59 year groups (6 patients each, 23.1%), 10-19 years (4 patients, 15.4%), 40-49 years (2 patients, 7.7%), and 60-69 years (1 patient, 3.8%). The mean SFAR score was 8.12 ± 3.50 (median 8.5; IQR 5.2-11.0). The full demographic and ORL clinical profile of the cohort is detailed in Table 1.

Table 1. Patient baseline and ORL clinical characteristics (N = 26), ENT Outpatient Clinic, Prof. Dr. I.G.N.G. Ngoerah General Hospital.

Characteristic	Category	n	%
Sex	Female	17	65.4
	Male	9	34.6
Age group (years)	10-19	4	15.4
	20-29	6	23.1
	30-39	7	26.9
	40-49	2	7.7
	50-59	6	23.1
	60-69	1	3.8
Occupation	Private employee	10	38.5
	Student	8	30.8
	Housewife	6	23.1
	Sanitarian	1	3.8
	Unemployed	1	3.8
SFAR-defined AR	Present (SFAR ≥ 7)	17	65.4
	Absent (SFAR < 7)	9	34.6
Predominant symptom	Sneezing	11	42.3
	Rhinorrhoea	9	34.6
	Nasal obstruction	6	23.1
Inferior turbinate hypertrophy	Present	21	80.8
	Absent	5	19.2
Leading trigger (AR, n = 17)	House dust	16	94.1
	House-dust mite	1	5.9

As shown in Table 1, SFAR-defined AR was identified in 17 of 26 patients, a prevalence of 65.4% (95% CI 46.2-80.6). The mean SFAR score was markedly higher among patients classified as having AR than among those without (10.24 ± 2.02 versus 4.11 ± 1.62; Mann-Whitney p<0.001). The predominant nasal symptom was paroxysmal sneezing (11 patients, 42.3%), followed by rhinorrhoea (9 patients, 34.6%) and nasal obstruction (6 patients, 23.1%). Inferior turbinate hypertrophy was observed in 21 patients (80.8%; 95% CI 62.1-91.5). Among the 17 patients with AR, house dust was the dominant self-reported trigger (16 patients, 94.1%). The AR prevalence with its 95% confidence interval and the distribution of predominant symptoms are illustrated in Figure 1.

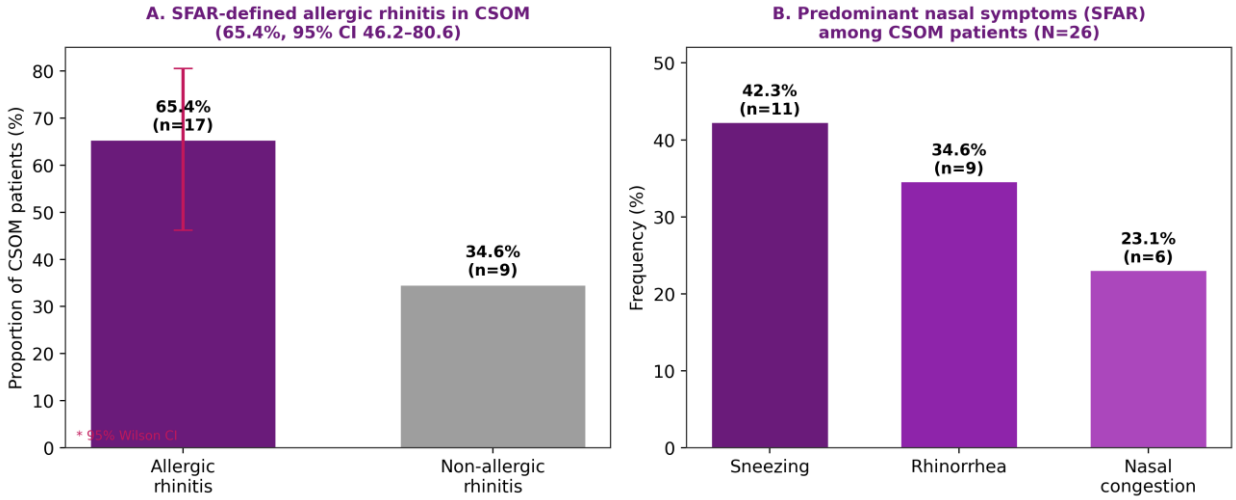

Figure 1. SFAR-defined allergic rhinitis prevalence with 95% Wilson confidence interval (panel A) and predominant nasal symptoms (panel B) among CSOM patients (N = 26).

The bivariate analysis of predictors of SFAR-defined AR is presented in Table 2. Inferior turbinate hypertrophy was strongly and significantly associated with AR (OR 12.80; 95% CI 1.15-142.58; p=0.034; Cramer's V = 0.47). Female sex (OR 4.06; 95% CI 0.72-22.86; p=0.194) and younger age (<40 years; OR 4.06; 95% CI 0.72-22.86; p=0.194) showed positive but non-significant associations. As further detailed in Table 2, the absolute risk of AR was 76.2% among patients with turbinate hypertrophy versus 20.0% among those without, an absolute risk difference of 56.2% (number needed to screen = 1.8).

Table 2. Bivariate analysis of predictors of SFAR-defined allergic rhinitis in CSOM patients (Fisher exact test).

Predictor	OR (95% CI)	p-value	Cramer's V
Inferior turbinate hypertrophy	12.80 (1.15-142.58)	0.034	0.47
Female sex	4.06 (0.72-22.86)	0.194	0.32
Age <40 years	4.06 (0.72-22.86)	0.194	0.32

Note: Female sex and age <40 years share an identical 2×2 cross-classification in this sample, hence identical odds ratios; this is a coincidence of the small sample, not a transcription error.

In the multivariable logistic-regression model, summarised together with the diagnostic-performance metrics in Table 3, inferior turbinate hypertrophy retained the largest adjusted effect (adjusted OR 2.47; 95% CI 0.60-10.15; p=0.209), although the association did not reach statistical significance after adjustment, consistent with the limited sample. The model explained a moderate proportion of variance (Nagelkerke R^2 = 0.272) and showed adequate calibration (Hosmer-Lemeshow chi-square = 0.335, p=0.563). As detailed in Table 3, the SFAR score discriminated inferior turbinate hypertrophy with an AUC of 0.776 (95% CI 0.527-1.000); at the Youden-optimal cut-off of SFAR ≥ 7, sensitivity was 76.2%, specificity 80.0%, positive predictive value 94.1%, negative predictive value 44.4%, positive likelihood ratio 3.81, and negative likelihood ratio 0.30. The corresponding ROC curve is shown in Figure 2, and the adjusted odds ratios are displayed as a forest plot in Figure 3.

Table 3. Multivariable logistic regression (upper rows) and diagnostic performance of the SFAR score (lower rows).

Parameter	Estimate (95% CI)	p-value
Female sex (adjusted)	1.68 (0.43-6.59)	0.455
Age <40 years (adjusted)	1.68 (0.43-6.59)	0.455
Inferior turbinate hypertrophy (adjusted)	2.47 (0.60-10.15)	0.209
Nagelkerke R^2	0.272	—
Hosmer-Lemeshow p	0.563	—
SFAR AUC for turbinate hypertrophy	0.776 (0.527-1.000)	—
Youden cut-off SFAR ≥ 7: Sn / Sp	76.2% / 80.0%	—
PPV / NPV	94.1% / 44.4%	—
LR+ / LR−	3.81 / 0.30	—

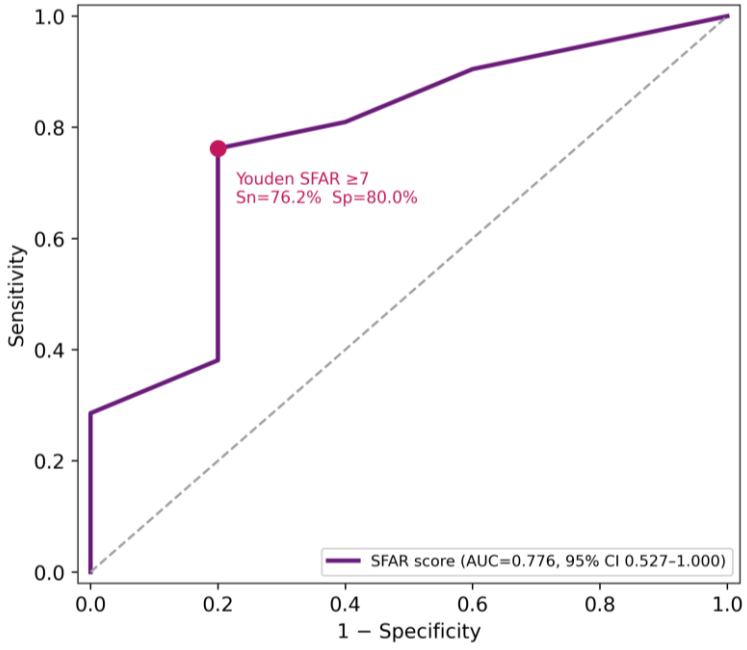

Figure 2. Receiver-operating-characteristic curve of the SFAR score for detecting inferior turbinate hypertrophy (AUC 0.776; DeLong 95% CI 0.527-1.00).

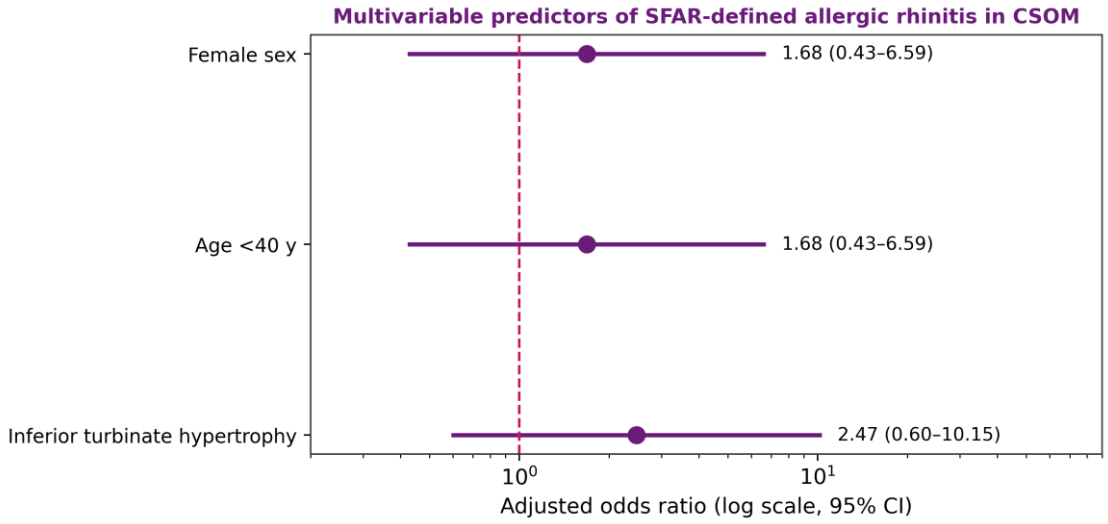

Figure 3. Forest plot of adjusted odds ratios from the multivariable logistic-regression model for SFAR-defined allergic rhinitis.

4. Discussion

In this cross-sectional study of 26 CSOM patients at a tertiary ENT referral centre in Bali, SFAR-defined allergic rhinitis was highly prevalent (65.4%; 95% CI 46.2-80.6), and inferior turbinate hypertrophy emerged as the strongest correlate of AR (bivariate OR 12.8; p=0.034; Table 2). These findings quantify, in an Indonesian setting, the clinically suspected but locally under-documented convergence of upper-airway allergy and chronic middle-ear disease.[14]

The observed AR prevalence is consistent with, and toward the upper range of, prior reports linking allergic airway disease to chronic otitis media. Systematic-review and Mendelian-randomisation evidence confirms a significant association between AR and otitis media, and a meta-analysis of paediatric risk factors identified atopy as a contributor to CSOM.[9-10,14] The comparatively high prevalence in our cohort (Table 1) likely reflects the tertiary-referral case-mix, the perennial house-dust-mite-rich tropical environment, and the sensitivity of the SFAR instrument for symptomatic AR.[3,18]

The predominance of female patients (65.4%; Table 1) aligns with international observations of higher atopic-disease prevalence among adult women after puberty, attributed to the immunomodulatory effects of oestrogen, which enhance humoral responses and mast-cell reactivity.[20] The clustering of cases in the 20-40 year productive age range echoes Sandbhor et al., who identified 16-40 years as the peak age band for AR, reflecting greater environmental and occupational allergen exposure; the lower frequency beyond 60 years is consistent with immunosenescence and declining allergen-specific IgE.[2,18]

Symptom distribution in our cohort, dominated by paroxysmal sneezing (42.3%) and rhinorrhoea (34.6%) as shown in Figure 1, mirrors the early-phase, histamine-mediated allergic response, whereas nasal obstruction (23.1%) reflects the late-phase inflammatory component with mucosal oedema driven by type-2 cytokines (IL-4, IL-5, IL-13).[1,21] This symptom pattern is mechanistically important in CSOM: persistent rhinorrhoea and mucosal swelling obstruct the nasopharyngeal orifice of the Eustachian tube, impairing middle-ear ventilation and drainage.[19,22]

The strong association between inferior turbinate hypertrophy and AR (80.8% prevalence; adjusted OR 2.47; Tables 2 and 3) is the central clinical finding. Turbinate hypertrophy is a morphological marker of chronic nasal mucosal inflammation; posterior-end enlargement exerts mechanical effects on nasopharyngeal airflow and the tubal orifice, and radiofrequency turbinate reduction has been shown to improve Eustachian tube function, particularly in atopic patients.[7-8] The discriminatory performance of the SFAR score for turbinate hypertrophy (AUC 0.776; Figure 2) and the low number needed to screen (1.8) support combining a validated symptom questionnaire with simple rhinoscopic assessment to flag patients warranting integrated upper-airway and otologic management.

The occupational profile, dominated by private-sector employees (38.5%), students (30.8%), and housewives (23.1%) as tabulated in Table 1, provides context on allergen-exposure environments. House dust as the dominant trigger (94.1%) is congruent with Indonesia's tropical climate, which favours proliferation of *Dermatophagoides pteronyssinus*, *D. farinae*, and *Blomia tropicalis*, the principal mite allergens in tropical house dust, and is consistent with national Indonesian data documenting high house-dust-mite sensitisation.[15,17] These findings also match Sandbhor et al., who identified mites and dust as the leading allergens.[3,18]

For otolaryngologists practising at Prof. Dr. I.G.N.G. Ngoerah General Hospital and comparable tertiary ENT centres in Indonesia, these findings support incorporating routine SFAR screening into the CSOM care pathway, with a low threshold for nasal examination for turbinate hypertrophy.[11] Identifying and treating comorbid AR, through allergen avoidance, intranasal corticosteroids, and antihistamines, may reduce Eustachian tube dysfunction and complement established CSOM management, for which contemporary reviews emphasise topical therapy, aural toilet, and, in refractory cases, surgery.[19,23]

Within the Indonesian ENT context, PERHATI-KL-aligned care and national health-insurance (BPJS) coverage of ENT consultation make a low-cost questionnaire-based screening approach both feasible and scalable across secondary and tertiary centres, particularly in tropical regions with heavy perennial mite exposure.[15-16] Systematic screening could support earlier, more equitable identification of a modifiable comorbidity.

A closer comparison with the literature situates our estimates within a coherent evidence base. Cross-sectional series across South and Southeast Asia have reported AR prevalences of 40-70% among patients with chronic middle-ear disease, and Mendelian-randomisation analysis supports a causal contribution of AR to otitis media.[9,14,18] Our prevalence of 65.4% (Figure 1) sits at the upper end of this range, best explained by the tertiary-referral case-mix, the house-dust-mite-saturated tropical environment of Bali, and the symptom-and-history basis of the SFAR instrument.[1,3]

Mechanistically, the association is biologically plausible and reinforced by the immunology of type-2 airway inflammation. Allergen capture drives naive CD4+ T-cell polarisation toward a Th2 phenotype, with IL-4 and IL-13 promoting IgE class-switching and IL-5 mobilising eosinophils; the resulting mediator release produces the glandular hypersecretion underlying rhinorrhoea and the tissue remodelling underlying inferior turbinate hypertrophy.[2,21] Because the nasopharyngeal mucosa is continuous with the

Eustachian tube lining, this inflammatory milieu degrades tubal function and establishes the effusion-prone middle-ear environment that predisposes to suppurative chronicity, as reflected in the strong turbinate-hypertrophy signal in Table 2.[7-8]

From an implementation standpoint, the findings translate into a pragmatic, low-cost screening pathway: every CSOM patient completes the SFAR at registration, anterior rhinoscopy documents inferior turbinate status, and patients meeting the SFAR threshold or exhibiting turbinate hypertrophy are referred for structured AR management.[11,19] With a number needed to screen below two (Table 2) and negligible marginal cost within BPJS-funded consultations, such a pathway is realistic for secondary and tertiary Indonesian centres.[16]

It is important to interpret the multivariable results with appropriate statistical humility. The attenuation of the turbinate-hypertrophy association after adjustment, from a bivariate odds ratio of 12.8 (Table 2) to an adjusted odds ratio of 2.47 (Table 3), is expected when a small sample is partitioned across several correlated predictors and a ridge penalty is applied; it reflects reduced precision and conservative shrinkage rather than absence of effect, as evidenced by the retained direction of association and the moderate Nagelkerke R^2 of 0.272. The adequately fitting Hosmer-Lemeshow statistic (p=0.563) indicates that the model is not miscalibrated.[9-10]

The audiological dimension also deserves emphasis. Allergic rhinitis has been associated with measurable conductive and subclinical auditory changes, and in CSOM, superimposed allergic Eustachian tube dysfunction may compound conductive hearing loss and complicate the timing of tympanoplasty.[23-24] Integrating SFAR screening with baseline audiometry would allow clinicians to characterise the allergic contribution to the hearing deficit.

The strengths of this study include its systematic use of a validated screening instrument, consecutive sampling within a defined window, STROBE-concordant reporting, and an analytical approach providing effect sizes, confidence intervals, and diagnostic-performance metrics rather than descriptive frequencies alone. To our knowledge, it is among the first Indonesian studies to quantify SFAR-defined AR and its association with turbinate hypertrophy specifically within a CSOM population. Several limitations nonetheless warrant caution. The single-centre design and modest sample (N=26) limit statistical power and precision, widening confidence intervals and attenuating associations after multivariable adjustment (Table 3); the study is therefore hypothesis-generating rather than confirmatory. The cross-sectional design precludes causal inference regarding the temporal AR-CSOM relationship. SFAR identifies probable AR by symptoms and history rather than by allergen-specific IgE or skin-prick testing, and the tertiary-referral setting may limit generalisability. Formal inter-observer reliability for turbinate assessment was not quantified. Larger, multicentre, prospective studies incorporating objective allergy testing, audiometric outcomes, and longitudinal Eustachian-tube-function assessment are needed to confirm these associations and to establish whether treating AR modifies middle-ear outcomes.[5,7,10,24]

5. Conclusion

Comorbid allergic rhinitis is highly prevalent among patients with chronic suppurative otitis media at this tertiary ENT referral centre, affecting 65.4% (95% CI 46.2-80.6) of the cohort, and is strongly associated with inferior turbinate hypertrophy (OR 12.8; 95% CI 1.15-142.6), with the SFAR score discriminating turbinate hypertrophy at an AUC of 0.776. Routine SFAR screening, combined with simple rhinoscopic assessment, offers a low-cost, scalable strategy for the early detection of allergic rhinitis in CSOM patients in Indonesian ENT practice, and may support more comprehensive management aimed at reducing Eustachian-tube-mediated middle-ear complications. Larger multicentre and prospective studies with objective allergy testing are warranted to confirm these findings.

Declarations

Ethics approval and consent to participate

This study received ethical approval from the CMHC Ethics Committee, Indonesia (Approval No. CMHC/EC/2025/114). Written informed consent was obtained from all participants prior to completion of the SFAR questionnaire, and the study was conducted in accordance with the Declaration of Helsinki and COPE and ICMJE standards.

Consent for publication

Not applicable; the manuscript contains no individually identifiable participant data or images.

Availability of data and materials

The datasets generated and analysed during the present 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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Authors' contributions

N.L.S.S. and M.P.W. conceived and designed the study; N.L.S.S. collected the data; M.P.W. and N.M.P.S. performed the statistical analysis and interpretation; N.L.S.S. drafted the manuscript; M.P.W. and N.M.P.S. critically revised it for important intellectual content. All authors have reviewed and approved the final version of the manuscript.

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6. References

1. Nur Husna SM, Tan HT, Md Shukri N, et al. Allergic rhinitis: a clinical and pathophysiological overview. Front Med (Lausanne). 2022;9:874114. doi:10.3389/fmed.2022.874114
2. Siti Sarah CO, Mohd Ashari NS. Exploration of allergic rhinitis: epidemiology, predisposing factors, clinical manifestations, laboratory characteristics, and emerging pathogenic mechanisms. Cureus. 2024;16(10):e71409. doi:10.7759/cureus.71409
3. Lee YZ, Kow ASF, Jacquet A, et al. House dust mite allergy in Malaysia: review of research gaps in the current scenario and the way forward. Exp Appl Acarol. 2023;91(4):509-539. doi:10.1007/s10493-023-00857-5
4. Lam K, Au E, Ip WK, et al. Inhalant mediated allergy: immunobiology, clinical manifestations and diagnosis. Clin Rev Allergy Immunol. 2025;68(1):43. doi:10.1007/s12016-025-09053-2
5. Onifade A, Katolo HW, Mookerjee S, et al. Epidemiology of chronic suppurative otitis media: systematic review to estimate global prevalence. J Epidemiol Glob Health. 2025;15(1):55. doi:10.1007/s44197-025-00396-9
6. Khairkar M, Deshmukh P, Maity H, et al. Chronic suppurative otitis media: a comprehensive review of epidemiology, pathogenesis, microbiology, and complications. Cureus. 2023;15(8):e43729. doi:10.7759/cureus.43729
7. Martines F, Dispenza F, Sireci F, et al. Eustachian tube function assessment after radiofrequency turbinate reduction in atopic and non-atopic patients. Int J Environ Res Public Health. 2021;18(3):881. doi:10.3390/ijerph18030881
8. Prakash P, Singh RK, Sinha R. The impact of inferior turbinate reduction on middle ear function in adults with nasal obstruction. Cureus. 2023;15(11):e48535. doi:10.7759/cureus.48535
9. De Corso E, Cantone E, Galli J, et al. Otitis media in children: which phenotypes are most linked to allergy? A systematic review. Pediatr Allergy Immunol. 2021;32(3):524-534. doi:10.1111/pai.13431
10. Heward E, Saeed H, Bate S, et al. Risk factors associated with the development of chronic suppurative otitis media in children: systematic review and meta-analysis. Clin Otolaryngol. 2023;49(1):62-73. doi:10.1111/coa.14102
11. Wise SK, Damask C, Roland LT, et al. International consensus statement on allergy and rhinology: allergic rhinitis - 2023. Int Forum Allergy Rhinol. 2023;13(4):293-859. doi:10.1002/alr.23090
12. Annesi-Maesano I, Didier A, Klossek M, et al. The score for allergic rhinitis (SFAR): a simple and valid assessment method in population studies. Allergy. 2002;57(2):107-114. doi:10.1034/j.1398-9995.2002.1o3170.x
13. Cheah SC, Hamizan AKW, Zahedi FD, et al. The modification, translation, and validation of the Malaysian version of the Score for Allergic Rhinitis (SFAR) questionnaire. Egypt J Otolaryngol. 2023;39(1):52. doi:10.1186/s43163-023-00413-3
14. Wang F, Yu C, Liu R. Causal relationship between allergic rhinitis and otitis media: a Mendelian randomization study. Medicine (Baltimore). 2024;103(39):e39671. doi:10.1097/MD.0000000000039671
15. Norback D, Hashim JH, Hashim Z, et al. Fractional exhaled nitric oxide (FeNO) among school children in Java and Sumatra, Indonesia: associations with respiratory symptoms, house dust mite sensitization and the home environment. J Asthma. 2024;61(12):1772-1780. doi:10.1080/02770903.2024.2383627
16. Pratama YA, Marhaeny HD, Rohmah L, et al. Allergic rhinitis behavioral changes after Indonesian house dust mites allergenic extract administration as immunotherapy. J Public Health Afr. 2023;14(Suppl 1):2510. doi:10.4081/jphia.2023.2510
17. Caraballo L, Lockey R, Puerta L, et al. *Blomia tropicalis*: a 50-year history. J Allergy Clin Immunol Pract. 2024;13(6):1289-1297. doi:10.1016/j.jaip.2024.11.007
18. Sandbhor A, Jain S, Deshmukh P, et al. Pattern and severity of allergic rhinitis correlated with patient characteristics: a rural hospital-based cross-sectional study. Indian J Otolaryngol Head Neck Surg. 2024;76(1):514-522. doi:10.1007/s12070-023-04198-y
19. Siddiqui ZA, Walker A, Pirwani MM, et al. Allergic rhinitis: diagnosis and management. Br J Hosp Med (Lond). 2022;83(2):1-9. doi:10.12968/hmed.2021.0570
20. Gutierrez-Brito JA, Lomeli-Nieto JA, Munoz-Valle JF, et al. Sex hormones and allergies: exploring the gender differences in immune responses. Front Allergy. 2025;5:1483919. doi:10.3389/falgy.2024.1483919
21. Maspero J, Adir Y, Al-Ahmad M, et al. Type 2 inflammation in asthma and other airway diseases. ERJ Open Res. 2022;8(3):00576-2021. doi:10.1183/23120541.00576-2021
22. Sharma K, Akre S, Chakole S, et al. Allergic rhinitis and treatment modalities: a review of literature. Cureus. 2022;14(8):e28501. doi:10.7759/cureus.28501
23. Hura N, Xia A, Santa Maria PL. Management strategies for chronic suppurative otitis media and why they fail. J Assoc Res Otolaryngol. 2025;26(4):389-396. doi:10.1007/s10162-025-00996-z
24. Mahajan A, Manhas M, Kalsotra P, et al. A prospective study of audiological manifestations in patients of allergic rhinitis. Indian J Otolaryngol Head Neck Surg. 2022;74(Suppl 2):1256-1261. doi:10.1007/s12070-020-02343-5