

ORIGINAL ARTICLE

Diagnostic Performance of the Eustachian Tube Dysfunction Questionnaire-7 (ETDQ-7) in Predicting Objective Eustachian Tube Patency Among Patients with Chronic Rhinosinusitis: A Cross-Sectional Analytical Study at a Tertiary Referral Hospital in Bali, Indonesia

I Made Wiranadha¹, I Gede Wahyu Adi Raditya^{2*}, Made Prani Windasari²

¹Department of Otorhinolaryngology-Head and Neck Surgery, Faculty of Medicine, Universitas Udayana / Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

²Department of Otorhinolaryngology-Head and Neck Surgery, Faculty of Medicine, Universitas Udayana / Udayana University Hospital, Badung, Indonesia

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*Corresponding author

I Gede Wahyu Adi Raditya

E-mail: dr.gedewahyu@unud.ac.id

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ABSTRACT

Background: Chronic rhinosinusitis (CRS) can extend to the tubal orifice and cause Eustachian tube dysfunction. Concordance between ETDQ-7 symptoms and objective tubal patency in CRS remains uncertain.

Objective: To evaluate the diagnostic performance of ETDQ-7 for objective tubal patency impairment in adults with CRS in Bali, Indonesia.

Methods: This analytical cross-sectional study enrolled 42 consecutive adults with CRS at a tertiary hospital from January to March 2026. ETDQ-7 scores ≥ 14.5 were considered abnormal. Tubal patency was assessed by acoustic impedance tympanometry. Chi-square testing, Spearman correlation, and multivariable logistic regression were performed.

Result: Mean age was 38.9 ± 11.6 years; 57.1% were female. Abnormal ETDQ-7 scores occurred in 24 patients (57.1%), and objective impairment occurred in 26 (61.9%). Impairment was more frequent with abnormal than normal ETDQ-7 scores (83.3% vs 33.3%, $p = 0.002$). Scores correlated with impaired patency ($r = 0.58$, $p < 0.001$). Abnormal ETDQ-7 independently predicted impairment (adjusted OR 7.84, 95% CI 1.74–35.29; $p = 0.007$).

Conclusion: ETDQ-7 is a useful outpatient triage tool for identifying patients with CRS who need objective audiological evaluation; it should not replace confirmatory testing.

1. Introduction

Chronic rhinosinusitis (CRS) represents a widespread, heterogeneously debilitating inflammatory disorder of the nasal cavity and paranasal sinuses that exerts a substantial global public health and socioeconomic burden.¹ As codified by the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS 2020) and international consensus guidelines, CRS is clinically diagnosed by the persistence of two or more cardinal sinonasal symptoms, predominantly nasal obstruction, anterior or posterior mucopurulent rhinorrhea, facial pain or pressure, and olfactory reduction or anosmia, persisting continuously for at least 12 consecutive weeks, corroborated by direct endoscopic visualization of mucosal purulence, polypoid, or edema, or mucosal opacification on computed tomography.¹ Epidemiological surveys across diverse geographical regions estimate that CRS afflicts between 5% and 12% of the global adult population, leading to substantial decrements in health-related quality of life, impaired occupational productivity, and staggering direct and indirect healthcare expenditures that frequently rival those associated with chronic obstructive pulmonary disease and diabetes mellitus.^{2,3} In the Asia-Pacific region, including the Indonesian archipelago, the disease burden of CRS is

particularly aggravated by tropical humidity, high environmental particulate exposure, biomass smoke inhalation, and delayed presentation to specialized tertiary rhinology centers.^{4,5}

Although classic clinical descriptions of chronic rhinosinusitis predominantly emphasize anterior airway impairment and sinus outflow tract blockage, contemporary rhinologic paradigms increasingly recognize that the pathophysiological ramifications of persistent sinonasal inflammation extend far beyond the anatomical boundaries of the osteomeatal complex.² The nasal cavity, the nasopharynx, and the middle ear cleft represent an anatomically continuous, functionally integrated mucosal continuum lined by pseudostratified ciliated columnar respiratory epithelium.^{6,7} Under physiological conditions, the Eustachian tube serves as the critical dynamic osteocartilaginous conduit connecting the lateral wall of the nasopharynx to the anterior wall of the tympanic cavity.^{8,9} Through its specialized physiological opening and closing cycles, the Eustachian tube executes three indispensable homeostatic functions: middle ear ventilation and ambient atmospheric pressure equilibration, continuous mucociliary clearance of middle ear secretions into the pharynx, and acoustic and biological protection against retrograde nasopharyngeal pressure

surges and pathogen reflux.^{8,9} When sinonasal inflammation becomes chronic, contiguous inflammatory spread and microvascular engorgement frequently engulf the pharyngeal orifice of the Eustachian tube, compromising its ventilatory and clearance mechanics.^{2,3}

Eustachian tube dysfunction (ETD) is increasingly acknowledged as one of the most prevalent and bothersome otologic comorbidities among patients suffering from chronic rhinosinusitis.^{3,10} Clinical registries indicate that up to 50% of adult patients presenting for the management of CRS experience prominent concurrent middle ear symptoms, most notably aural fullness, persistent ear pressure, otalgia during atmospheric pressure transitions, muffled hearing, autophony, and recurrent cracking or popping sensations during swallowing.^{10,11} In many instances, these otologic manifestations are so pronounced that they overshadow the patient's primary nasal complaints, severely impairing psychological well-being, disrupting sleep architecture, and degrading overall functional status.¹² Unfortunately, in standard outpatient rhinology practice, secondary Eustachian tube dysfunction is frequently under-recognized or dismissed as nonspecific ear discomfort, leading to fragmented clinical care, unnecessary antibiotic or topical ear drop prescriptions, and missed opportunities for timely targeted intervention.^{3,13}

From a pathophysiological and biomolecular perspective, the mechanistic nexus linking chronic rhinosinusitis to Eustachian tube failure is multidimensional, involving both extrinsic mechanical compression and intrinsic mucosal degradation.^{2,6} In patients with CRS, persistent antigenic stimulation triggers an intense cascade of pro-inflammatory cytokines, including interleukin-4 (IL-4), interleukin-5 (IL-5), interleukin-13 (IL-13), tumor necrosis factor- α (TNF- α), and interleukin-8 (IL-8), that promotes dense eosinophilic and neutrophilic infiltration throughout the respiratory mucosa.^{6,14} These inflammatory mediators induce marked subepithelial edema, microvascular hyperpermeability, goblet cell hyperplasia, and excessive secretion of viscoelastic mucin glycoproteins such as MUC5AC within the mucous membrane surrounding the torus tubarius and the pharyngeal ostium.^{6,15} Furthermore, chronic inflammatory exposure impairs ciliary beat frequency and disorganizes the mucociliary apparatus of the tubal lumen, leading to secretion stasis, persistent mucosal thickening, and colonization by pathogenic bacterial biofilms.^{7,15}

Beyond intrinsic mucosal pathology, inflammatory infiltration of the adjacent parapharyngeal and soft palate musculature directly disrupts the dynamic active opening mechanism of the Eustachian tube.^{9,16} The cartilaginous portion of the tube remains passively collapsed at rest to protect the middle ear from reflux, relying on intermittent active dilation during swallowing, yawning, or mastication.^{16,17} This active opening is executed primarily by the coordinated contraction of the tensor veli palatini (TVP) muscle, innervated by the mandibular division of the trigeminal nerve (cranial nerve V3), with accessory support from the levator veli palatini (LVP) muscle, innervated by the pharyngeal plexus of the vagus nerve (cranial nerve X).^{16,17} In CRS, chronic peritubal edema increases extrinsic tissue pressure and mechanical compliance resistance along the cartilaginous lamina, while submucosal inflammatory dissection into the TVP and LVP myofibers induces local muscular fatigue, functional decoupling, and contractile inefficiency.¹⁷ Consequently, the muscular traction required to rotate the lateral cartilaginous plate and unseal the tubal lumen is compromised, locking the Eustachian tube in a chronically collapsed or hypoventilated state.^{9,17}

Persistent tubal closure initiates a destructive physical sequence governed by the van Liew-Hills mucosal gas absorption model.¹⁸ Once the physiological conduit for atmospheric air replenishment is abolished, the trapped gas volume within the middle ear cleft, primarily nitrogen, oxygen, and carbon dioxide, is continuously absorbed into the dense capillary network of the tympanic mucosa along a positive partial pressure gradient.¹⁸ Because the rigid osseous walls of the temporal bone prevent structural inward collapse, progressive gas absorption generates a severe, uncompensated sub-atmospheric negative middle ear pressure, typically exceeding -100 daPa.^{18,19} This chronic negative pressure pulls the tympanic membrane inward, inducing acoustic impedance mismatch, dampening ossicular chain mobility, and triggering hydrostatic transudation of sterile serous fluid from mucosal microvessels into the tympanic cavity, ultimately precipitating otitis media with effusion (OME) and conductive hearing loss.^{2,8}

Despite the substantial clinical and biological importance of Eustachian tube dysfunction in CRS, the objective evaluation of tubal function has historically been hampered by the deep, inaccessible anatomical location of the tube within the skull base and the dynamic, intermittent nature of its opening cycles.^{9,20} Historically, clinicians relied almost exclusively on patient-reported history or gross physical maneuvers such as the Valsalva and Toynbee tests, which lack quantitative precision, possess poor test-retest reliability, and fail to correlate reliably with true physiological middle ear ventilation.^{13,21} While advanced objective diagnostic modalities, such as acoustic impedance tympanometry, dynamic slow-motion video-endoscopy, tubomanometry (TMM), and sonotubometry, provide quantitative metrics of middle ear pressure, acoustic admittance, and tubal opening pressure, their availability in routine outpatient rhinology clinics in developing healthcare systems is severely constrained by high capital equipment costs, specialized operational training requirements, and lack of universal insurance reimbursement.^{21,22}

In response to the urgent clinical need for a rapid, standardized, and patient-centered assessment instrument, McCoul and colleagues in 2012 developed and validated the 7-item Eustachian Tube Dysfunction Questionnaire (ETDQ-7).²³ The ETDQ-7 was engineered as a disease-specific patient-reported outcome measure comprising seven discrete questions that evaluate the severity of cardinal otologic symptoms over the preceding one-month period on a 7-point Likert scale ranging from 1 (no problem) to 7 (severe problem).²³ A total composite score of 14.5 or greater (or a mean item score ≥ 2.1) was empirically established as the discriminatory threshold defining clinically significant Eustachian tube dysfunction, demonstrating an notable initial sensitivity of 100% and specificity of 100% in distinguishing adult patients with obstructive ETD from healthy control subjects.²³ The questionnaire has since gained widespread international adoption, has been translated and psychometrically validated across multiple languages, including a rigorous Indonesian linguistic adaptation and validation by Restuti et al.²⁴, and is frequently utilized as a primary endpoint in clinical trials assessing medical therapies, endoscopic sinus surgery, and balloon Eustachian tuboplasty.²⁵⁻²⁷

However, substantial diagnostic controversies and methodological dilemmas have emerged regarding the real-world performance and clinical fidelity of the ETDQ-7 in complex upper airway populations.^{9,28} Recent critical appraisals in unselected audiology cohorts and tertiary ENT

clinics have raised serious concerns regarding the potential for symptom overlap and false-positive diagnostic classification.²⁸ Because several ETDQ-7 items, such as aural fullness, head pressure, and muffled hearing, can be provoked by sinonasal mucosal congestion, allergic rhinitis, temporomandibular joint dysfunction, or superior semicircular canal dehiscence without actual middle ear pressure abnormalities, relying on the ETDQ-7 as a stand-alone diagnostic gold standard risks substantial diagnostic misclassification.^{13,29} Several contemporary investigations have observed notable discordance between subjective questionnaire scores and objective physiological parameters derived from tympanometry or tubomanometry, emphasizing that subjective perception of ear fullness does not invariably mirror mechanical tubal patency failure.^{28,29}

This diagnostic dilemma is especially acute in developing nations such as Indonesia, where tertiary referral academic medical centers, such as Prof. Dr. I.G.N.G. Ngoerah General Hospital in Bali, serve as apex clinical hubs managing highly complex, medically refractory CRS populations across vast archipelagic catchment areas.^{5,30} In resource-constrained public healthcare settings, establishing the precise predictive accuracy, sensitivity, and multivariable strength of the ETDQ-7 against objective diagnostic standards is essential.³⁰ If a strong, independent statistical relationship exists between abnormal ETDQ-7 scores and objectively verified tubal impairment, the questionnaire can be deployed with high confidence as a cost-effective, zero-equipment triage instrument to prioritize high-risk CRS patients for detailed audiological and endoscopic evaluation.^{23,24} Conversely, delineating the extent of subjective-objective divergence will safeguard clinicians against over-diagnosis and premature invasive interventions.^{9,28}

The core scientific novelty of the present investigation lies in establishing the first rigorous diagnostic accuracy profile, continuous non-parametric correlation, and multivariable logistic regression model evaluating the ETDQ-7 score against objective acoustic impedance tympanometric patency specifically within an Indonesian adult chronic rhinosinusitis cohort managed at a top-tier tertiary referral center in Denpasar, Bali. To resolve existing clinical controversies, the primary aim of this study was to determine the precise association between ETDQ-7 symptom scores and objective Eustachian tube patency status, to evaluate whether symptom severity independently predicts objective tubal impairment after rigorous statistical adjustment for confounding demographic factors (age and sex) and clinical CRS phenotypic subtypes (CRSwNP vs CRSsNP), and to delineate actionable clinical triage algorithms for secondary and tertiary rhinology practice in the Asia-Pacific region.

2. Methods

2.1. Study Design, Clinical Setting, and Ethical Governance

This study was conceptualized, designed, and executed as an observational, analytical cross-sectional diagnostic accuracy investigation in strict compliance with the Standards for Reporting of Diagnostic Accuracy Studies (STARD 2015) guidelines.³¹ The investigation was conducted at the Department of Otorhinolaryngology-Head and Neck Surgery, Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia, over a consecutive three-month enrollment period extending from January 2026 through March 2026. Prof. Dr. I.G.N.G. Ngoerah General Hospital serves as the primary top-tier national tertiary academic medical center, regional oncology hub, and apex referral institution for the province of Bali and the adjacent Lesser Sunda Islands (West Nusa

Tenggara and East Nusa Tenggara). The study protocol strictly adhered to the ethical principles codified in the Declaration of Helsinki (1964 and its subsequent amendments) and was formally reviewed and granted institutional ethical clearance by the Research Ethics Committee of the Faculty of Medicine, Udayana University under formal ethical approval number 3021/UN14.2.2.VII.14/LT/2026. Written informed consent was obtained from all eligible participants prior to enrollment following detailed counseling regarding the study objectives, diagnostic procedures, and voluntary participation rights.

2.2. Target Population and Consecutive Recruitment Protocol

The target population encompassed all adult patients presenting to the Otorhinolaryngology outpatient rhinology clinic who met the standardized diagnostic criteria for chronic rhinosinusitis as codified by the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS 2020) and Indonesian national clinical guidelines.^{1,5} Eligible patients were consecutively recruited using a total consecutive sampling strategy to minimize selection bias. Inclusion criteria were: (1) chronological age of 18 years or older at the time of examination; (2) definitive diagnosis of chronic rhinosinusitis established by a board-certified otorhinolaryngologist based on persistent characteristic symptoms (nasal obstruction, anterior/posterior rhinorrhea, facial pain/pressure, or hyposmia) lasting ≥ 12 weeks with objective endoscopic confirmation of sinonasal mucosal disease; (3) structurally intact, non-perforated bilateral tympanic membranes verified on high-magnification pneumatic otomicroscopy; and (4) cognitive capacity and willingness to complete the diagnostic questionnaire and audiological testing protocols.

2.3. Strict Eligibility and Exclusion Safeguards

To eliminate confounding clinical conditions that could independently alter middle ear pressure, acoustic admittance, or tubal biomechanics, strict exclusion criteria were enforced. Patients were excluded if they presented with: (1) acute upper respiratory tract viral or bacterial infections within the preceding two weeks; (2) history of previous otologic surgical interventions, including myringotomy, tympanostomy tube insertion, tympanoplasty, or mastoidectomy; (3) pre-existing chronic suppurative otitis media, cholesteatoma, otosclerosis, or congenital craniofacial malformations (such as cleft palate or Down syndrome); (4) history of head and neck radiotherapy or skull base surgical resections; (5) benign or malignant neoplasms of the nasal cavity, paranasal sinuses, or nasopharynx (e.g., nasopharyngeal carcinoma or inverted papilloma); (6) active systemic granulomatous disorders (such as granulomatosis with polyangiitis or sarcoidosis); or (7) incomplete questionnaire responses or uninterpretable audiological tracing data.

2.4. Sample Size Estimation and Statistical Power

A priori sample size calculation was performed using the standard single-proportion formula for diagnostic observational investigations: $n = [Z^2 * P * (1 - P)] / d^2$, where Z represents the standard normal deviate corresponding to a two-sided 95% confidence level ($Z = 1.96$), P represents the expected prevalence of Eustachian tube dysfunction among adult patients with chronic rhinosinusitis derived from pooled meta-analytic estimates by Chen et al.³ ($P = 0.472$ or 47.2%), and d represents the acceptable margin of absolute error set at 0.15 (15%). Inserting these empirical parameters into the formulation yielded a baseline sample size requirement of 42 subjects. Because recruitment was maintained consecutively throughout the designated three-

month enrollment window, exactly 42 fully evaluable adult CRS patients meeting all rigorous eligibility criteria were accrued and completed the full diagnostic evaluation protocol.

2.5. Standardized Rhinologic Examination and Phenotypic Subtyping

All enrolled participants underwent detailed clinical and rhinologic assessment performed by two independent senior otorhinolaryngologists. Rigid diagnostic nasal endoscopy (0-degree and 30-degree 4.0 mm endoscopes; Karl Storz Endoskope, Tuttlingen, Germany) was executed following topical mucosal decongestion and local anesthesia using 0.5% oxymetazoline hydrochloride and 2% lidocaine spray. Endoscopic findings were systematically mapped to assess mucosal edema, presence and character of purulent middle meatal drainage, and the presence, morphology, and extent of sinonasal polyposis. In accordance with EPOS 2020 diagnostic guidelines, participants were formally stratified into two primary clinical phenotypic subtypes: chronic rhinosinusitis without nasal polyps (CRSsNP) and chronic rhinosinusitis with nasal polyps (CRSwNP).¹ Furthermore, direct endoscopic inspection of the nasopharyngeal vault, torus tubarius, fossa of Rosenmüller, and the pharyngeal ostium of the Eustachian tube was performed to rule out mechanical neoplastic obstruction or adenoidal hypertrophy.

2.6. Symptom Burden Assessment via the Bahasa Indonesia ETDQ-7

Subjective severity of Eustachian tube dysfunction symptoms was evaluated using the 7-item Eustachian Tube Dysfunction Questionnaire (ETDQ-7).²³ The ETDQ-7 evaluates seven discrete symptom domains experienced over the preceding one-month period: (1) pressure or fullness in the ears; (2) pain or aching in the ears; (3) feeling that the ears are clogged or plugged; (4) ear symptoms when you have a cold or sinus problem; (5) crackling or popping sounds in the ears; (6) ringing in the ears; and (7) feeling that your hearing is muffled.²³ Each item is scored on a 7-point Likert scale ranging from 1 ('no problem at all') to 7 ('severe problem'), yielding a total composite score ranging from 7 to 49 points (or an item mean ranging from 1.0 to 7.0).²³ The questionnaire was administered using the psychometrically validated Bahasa Indonesia version established by Restuti et al.,²⁴ which demonstrated outstanding internal consistency (Cronbach alpha = 0.91) and reliable test-retest reliability. In accordance with established international diagnostic standards, a composite score of ≥ 14.5 (corresponding to a mean score ≥ 2.1) was defined as an abnormal ETDQ-7 score indicative of clinically significant Eustachian tube dysfunction, whereas a composite score < 14.5 was classified as normal.^{23,24}

2.7. Objective Acoustic Impedance Tympanometric Patency Evaluation

Objective assessment of Eustachian tube patency was executed within 60 minutes of questionnaire completion using acoustic impedance tympanometry (Madsen Zodiac 901 Clinical Impedance Audiometer; Otometrics, Taastrup, Denmark) in a calibrated sound-attenuated audiological chamber complying with ANSI S3.1 standards. Equipment calibration was performed daily using a 2.0-cc artificial test cavity. Following verification of an unobstructed external auditory canal and an intact tympanic membrane via pneumatic otoscopy, an appropriately sized hermetic silicone probe was seated within the canal. Using a standard 226-Hz probe tone presented at 85 dB Sound Pressure Level (SPL), pneumatic pressure was swept continuously from +200 daPa to -400 daPa at an automated sweep rate of 50 daPa/sec.

Quantitative parameters recorded comprised external ear canal volume (V_{ea} , cc), static acoustic admittance / peak compliance (Y_{peak} , mmho or cc), tympanometric peak pressure (TPP, daPa), and tympanometric width/gradient (TW, daPa).¹⁹ Tympanometric waveforms were classified in accordance with the standardized Jerger classification system:¹⁹ (1) Type A (normal curve: peak compliance 0.3–1.6 cc, TPP between -100 and +100 daPa), reflecting normal middle ear aeration and physiological tubal patency; (2) Type B (flat curve: negligible peak compliance < 0.2 cc across the sweep), diagnostic of middle ear fluid accumulation, severe mucosal immobilization, or otitis media with effusion; and (3) Type C (retracted curve: distinct peak compliance shifted to severe negative pressure < -100 daPa), diagnostic of significant Eustachian tube hypoventilation and sustained negative intratympanic pressure.^{9,19} For primary dichotomous classification, Eustachian tube patency was categorized as 'Patent' (Type A tympanogram) or 'Impaired' (pathological Type B or Type C tympanogram).⁹ In patients with bilateral CRS, the ear demonstrating the more severe functional pathology was designated as the index ear for statistical correlation analysis.

2.8. Comprehensive Biostatistical Analysis Plan

All clinical, endoscopic, questionnaire, and audiological variables were entered into a standardized electronic research database and analyzed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA) alongside MedCalc Diagnostic Calculator Version 20.0 (MedCalc Software, Ostend, Belgium). Continuous demographic and clinical variables were evaluated for distributional normality using the Shapiro-Wilk test; normally distributed continuous data are reported as mean $\pm$ standard deviation (SD) and range, whereas non-normally distributed parameters are expressed as median and interquartile range (IQR). Categorical parameters are summarized as absolute frequencies and percentages [n (%)]. Univariable descriptive analysis was utilized to characterize the baseline profile of the study cohort. Bivariable comparisons of categorical variables (ETDQ-7 category, sex, CRS phenotype vs. patency status) were evaluated using the Pearson Chi-Square test with Yates continuity correction where appropriate. The monotonic relationship between continuous ETDQ-7 scores and objective patency was assessed using the Spearman rank-order correlation coefficient (r) with 95% confidence intervals (CI). Receiver operating characteristic (ROC) curve analysis was generated to quantify diagnostic discriminative performance via the Area Under the Curve (AUC), and 2x2 contingency matrices were constructed to calculate sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (LR+), negative likelihood ratio (LR-), diagnostic odds ratio (DOR), and overall accuracy.³¹ Multivariable binary logistic regression analysis (Enter method) was constructed to identify independent predictors of impaired Eustachian tube patency, incorporating variables with bivariable screening $p < 0.25$ alongside clinically essential biological covariates (age, sex, CRS phenotype, and ETDQ-7 category). Model calibration and goodness-of-fit were appraised using the Omnibus Test of Model Coefficients, the Nagelkerke pseudo- R^2 statistic, and the Hosmer-Lemeshow goodness-of-fit test. Two-sided p -values < 0.05 were established as the threshold for statistical significance across all inferential tests.

3. Results

3.1. Baseline Sociodemographic Profile and Clinical Presentation

A total of 42 consecutive adult patients with confirmed chronic rhinosinusitis completed the detailed clinical, endoscopic, symptom-based, and audiological evaluation protocol. As comprehensively detailed in Table 1 and illustrated across the five panels in Figure 1, the baseline sociodemographic profile and clinical presentation established a representative adult cohort. The chronological age of the participants ranged from 18.0 to 68.0 years, with an overall mean age of 38.9 ± 11.6 years and a median of 39.0 years, demonstrating that the sample was composed predominantly of working-age adults in their fourth and fifth decades of life, as depicted in the age histogram in Figure 1A. Regarding biological sex distribution, as detailed in Table 1 and displayed in Figure 1B, female participants demonstrated

a slight preponderance, comprising 57.1% ($n = 24$) of the cohort compared to 42.9% ($n = 18$) male participants, yielding a female-to-male sex ratio of 1.33:1. Diagnostic nasal endoscopy classified 59.5% ($n = 25$) of the cohort as chronic rhinosinusitis without nasal polyps (CRSsNP), whereas 40.5% ($n = 17$) presented with chronic rhinosinusitis with nasal polyps (CRSwNP), as detailed in Table 1 and shown in Figure 1C. Across the entire cohort, the total composite ETDQ-7 score exhibited an overall mean of 18.6 ± 6.1 (median: 17.5, range: 7.0–34.0 points). Utilizing the established international diagnostic threshold of 14.5, abnormal ETDQ-7 scores were identified in 57.1% ($n = 24$) of patients, whereas 42.9% ($n = 18$) had normal scores, as documented in Table 1 and visualized in the stacked histogram in Figure 1D. Objective acoustic impedance tympanometry revealed that impaired Eustachian tube patency was documented in 61.9% ($n = 26$) of subjects, while 38.1% ($n = 16$) maintained normal tubal patency, as detailed in Table 1 and illustrated in Figure 1E.

Table 1. Baseline Sociodemographic and Clinical Characteristics of the Adult Chronic Rhinosinusitis Cohort (N = 42).

Variable	Category / Metric	Frequency (n)	Percentage (%) / Mean $\pm$ SD
Age (years)	Mean $\pm$ SD	—	38.9 ± 11.6
	Median (Range)	—	39.0 (18.0–68.0)
Biological Sex	Male	18	42.9%
	Female	24	57.1%
CRS Phenotypic Subtype	CRS without nasal polyps (CRSsNP)	25	59.5%
	CRS with nasal polyps (CRSwNP)	17	40.5%
ETDQ-7 Total Score	Mean $\pm$ SD	—	18.6 ± 6.1
	Normal (< 14.5)	18	42.9%
	Abnormal (≥ 14.5)	24	57.1%
Eustachian Tube Patency	Patent (Normal Ventilation)	16	38.1%
	Impaired (Pathological Patency)	26	61.9%

Values are presented as absolute frequencies with percentages n (%) for categorical variables or mean $\pm$ standard deviation (SD) and median (range) for continuous variables. CRS: chronic rhinosinusitis; CRSsNP: chronic rhinosinusitis without nasal polyps; CRSwNP: chronic rhinosinusitis with nasal polyps; ETDQ-7: Eustachian Tube Dysfunction Questionnaire-7.

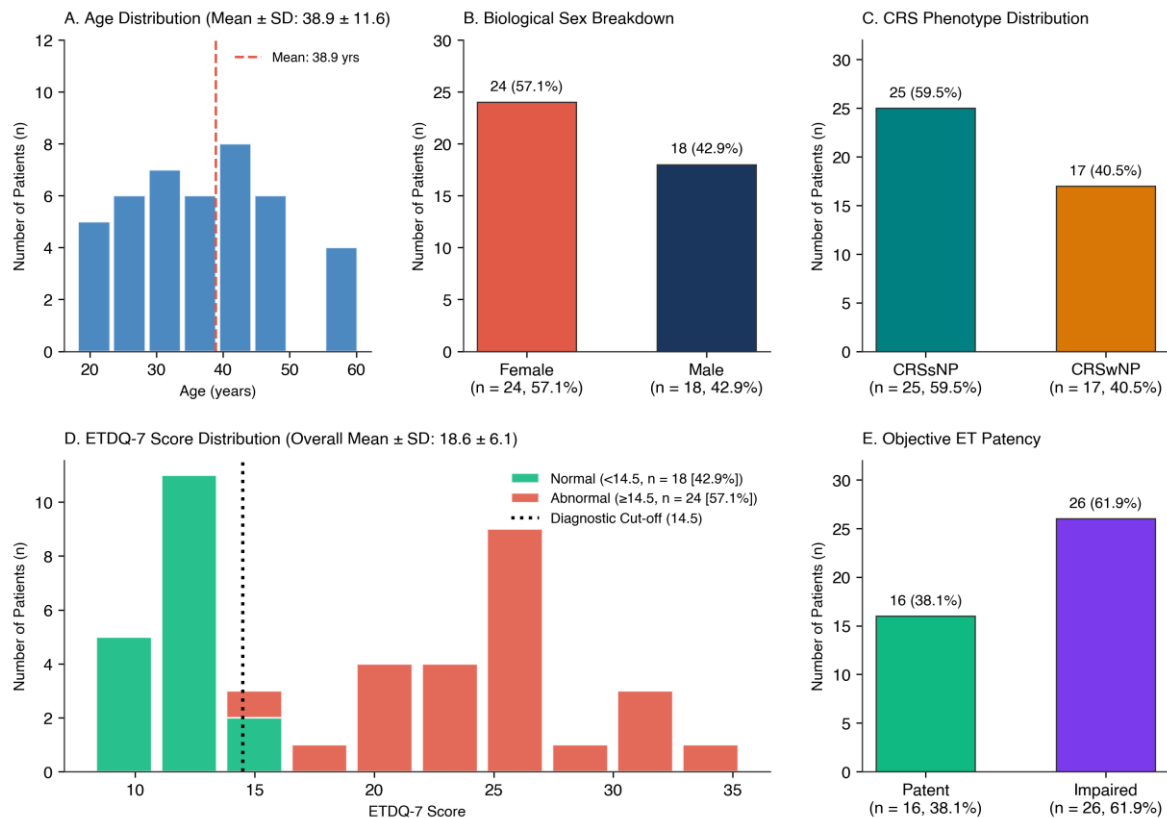


Figure 1. Baseline sociodemographic distributions and clinical disease profiles of the adult chronic rhinosinusitis cohort (N = 42). (A) Histogram and normal distribution curve of patient age (mean ± SD: 38.9 ± 11.6 years, range: 18–68 years). (B) Biological sex breakdown illustrating a slight female preponderance (57.1% vs 42.9%). (C) Distribution of chronic rhinosinusitis phenotypic subtypes: CRSSNP (59.5%, n = 25) vs CRSwNP (40.5%, n = 17). (D) Stacked histogram of total ETDQ-7 scores across the cohort (mean ± SD: 18.6 ± 6.1), categorized by the diagnostic cut-off of 14.5 into normal (42.9%, n = 18) and abnormal (57.1%, n = 24). (E) Prevalence of objective Eustachian tube patency status determined by acoustic impedance tympanometry: patent (38.1%, n = 16) vs impaired (61.9%, n = 26).

3.2. Bivariate Association Between ETDQ-7 Categories and Tubal Patency

The central primary empirical finding of this investigation, the direct cross-tabulation and bivariate association between ETDQ-7 symptom category and objective Eustachian tube patency, is comprehensively detailed in Table 2 and graphically demonstrated in Figure 2. Among patients presenting with a normal ETDQ-7 score (< 14.5, n = 18), the majority demonstrated preserved, normal Eustachian tube patency, accounting for exactly 66.7% (n = 12), whereas only 33.3% (n = 6) exhibited objective patency impairment. In sharp contrast, among patients classified with an abnormal ETDQ-7 score (≥ 14.5, n = 24), only 16.7% (n = 4) retained patent Eustachian tubes, whereas an overwhelming 83.3% (n = 20) demonstrated objective impairment of tubal patency. As shown in the bivariate cross-tabulation in Table 2, this marked divergence in objective patency across symptom categories achieved high statistical significance upon Pearson

Chi-Square testing with continuity correction ($\chi^2 = 9.87$, degrees of freedom $df = 1$, $p = 0.002^*$). The crude odds of presenting with objective tubal patency impairment among patients with abnormal ETDQ-7 scores were exactly 10.00 times higher (95% CI: 2.23–44.75) compared to patients reporting normal questionnaire scores. Furthermore, as demonstrated in the box-and-whisker and jittered distribution in Figure 2, patients with objective tubal patency impairment exhibited a markedly higher mean ETDQ-7 score of 22.3 ± 5.6 points (range: 12.0–34.0 points) compared to those with preserved patent tubes, who exhibited a mean score of only 12.6 ± 2.9 points (range: 7.0–19.0 points), representing an absolute mean score difference of +9.7 points. Non-parametric Spearman rank-order correlation analysis confirmed a highly significant, moderate-to-strong positive monotonic correlation between continuous ETDQ-7 symptom scores and worsening Eustachian tube patency status (Spearman $r = 0.58$, 95% CI: 0.33–0.75, $p < 0.001^{***}$).

Table 2. Cross-Tabulation and Bivariate Association Between ETDQ-7 Symptom Category and Objective Eustachian Tube Patency (N = 42).

ETDQ-7 Symptom Category	Patent ET, n (%)	Impaired ET, n (%)	Total Cohort (N)	Chi-Square (χ^2)	p-value
Normal ETDQ-7 (< 14.5)	12 (66.7%)	6 (33.3%)	18 (100.0%)	9.87	0.002*
Abnormal ETDQ-7 (≥ 14.5)	4 (16.7%)	20 (83.3%)	24 (100.0%)	—	—
Total Cohort	16 (38.1%)	26 (61.9%)	42 (100.0%)	—	—

Data presented as absolute frequencies with row percentages n (%). Statistical testing performed via Pearson Chi-Square test with continuity correction ($\chi^2 = 9.87$, $df = 1$). *Statistically significant at $\alpha = 0.05$. ETDQ-7: Eustachian Tube Dysfunction Questionnaire-7; ET: Eustachian tube.

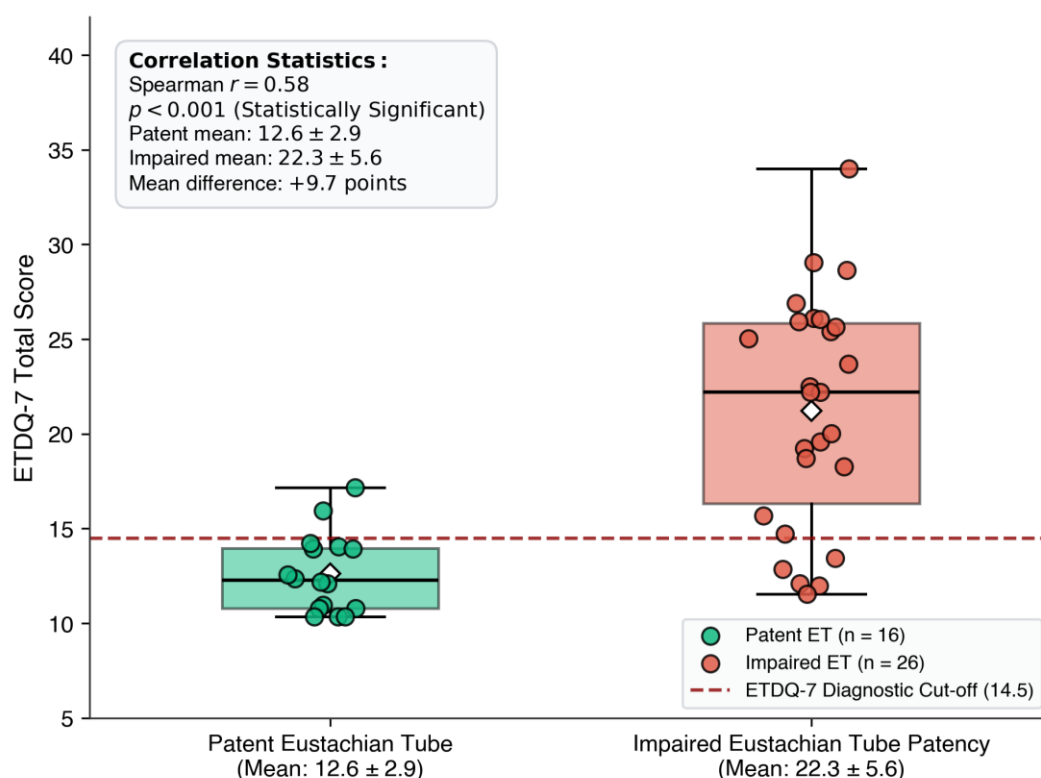


Figure 2. Correlation between continuous ETDQ-7 symptom scores and objective Eustachian tube patency status. Box-and-whisker plots with overlaid jittered scatter points illustrate that patients with impaired tubal patency exhibited a markedly higher mean ETDQ-7 score (22.3 ± 5.6 points, $n = 26$) compared to patients with preserved tubal patency (12.6 ± 2.9 points, $n = 16$; mean score difference: +9.7 points). Spearman rank-order correlation demonstrated a highly significant, moderate-to-strong positive monotonic association (Spearman $r = 0.58$, 95% CI: 0.33–0.75, $p < 0.001^{***}$). Horizontal dashed line indicates the validated clinical diagnostic cut-off of 14.5.

3.3. Bivariable Screening of Demographic and Clinical Risk Factors

Bivariable screening analysis of sociodemographic and clinical factors associated with impaired Eustachian tube patency is outlined in Table 3 and depicted in the stratified clustered bar charts in Figure 3. Chronological age evaluated as a continuous predictor was slightly higher among patients with impaired patency compared to those with patent tubes, but this trend did not achieve statistical significance in univariable logistic screening (crude OR = 1.04 per year increase, 95% CI: 0.98–1.10, $t = 1.35$, $p = 0.184$). Regarding biological sex, impaired patency was documented in 66.7% ($n = 16$ of 24) of female patients compared to 55.6% ($n = 10$ of 18) of male patients, representing a modest, non-significant

difference (crude OR = 1.60, 95% CI: 0.45–5.67, $\chi^2 = 0.54$, $p = 0.421$), as outlined in Table 3. In terms of chronic rhinosinusitis phenotypic subclassification, patients diagnosed with CRSwNP exhibited a substantially higher rate of objective tubal impairment (76.5%, $n = 13$ of 17) compared to those diagnosed with CRSsNP (52.0%, $n = 13$ of 25; crude OR = 3.00, 95% CI: 0.76–11.83, $\chi^2 = 2.58$, $p = 0.108$), as outlined in Table 3 and illustrated in Figure 3B. In comparison, the stratification by ETDQ-7 symptom category, as depicted in Figure 3A and outlined in Table 3, demonstrated a massive and highly significant disparity (83.3% vs 33.3%, $p = 0.002^*$), confirming that symptom burden captured by the questionnaire was far more predictive of objective patency than gross phenotypic polyposis or demographic covariates.

Table 3. Bivariable Screening Analysis of Sociodemographic and Clinical Predictors Associated with Impaired Eustachian Tube Patency (N = 42).

Potential Predictor Variable	Impaired Patency, n / N (%)	Crude Odds Ratio (95% CI)	Test Statistic	p-value
Age (continuous, per year increase)	—	1.04 (0.98–1.10)	$t = 1.35$	0.184
Biological Sex				
• Male (reference)	10 / 18 (55.6%)	1.00 (Reference)	—	—
• Female	16 / 24 (66.7%)	1.60 (0.45–5.67)	$\chi^2 = 0.54$	0.421
CRS Phenotype				
• CRSsNP (without polyps, reference)	13 / 25 (52.0%)	1.00 (Reference)	—	—
• CRSwNP (with polyps)	13 / 17 (76.5%)	3.00 (0.76–11.83)	$\chi^2 = 2.58$	0.108
ETDQ-7 Symptom Score Category				

Potential Predictor Variable	Impaired Patency, n / N (%)	Crude Odds Ratio (95% CI)	Test Statistic	p-value
• Normal ETDQ-7 (< 14.5, reference)	6 / 18 (33.3%)	1.00 (Reference)	—	—
• Abnormal ETDQ-7 (≥ 14.5)	20 / 24 (83.3%)	10.00 (2.23–44.75)	$\chi^2 = 9.87$	0.002*

Continuous age screened via univariable logistic regression. Categorical variables evaluated via Pearson Chi-Square test. *Statistically significant at alpha = 0.05. CI: confidence interval; CRSsNP: chronic rhinosinusitis without nasal polyps; CRSwNP: chronic rhinosinusitis with nasal polyps; ETDQ-7: Eustachian Tube Dysfunction Questionnaire-7.

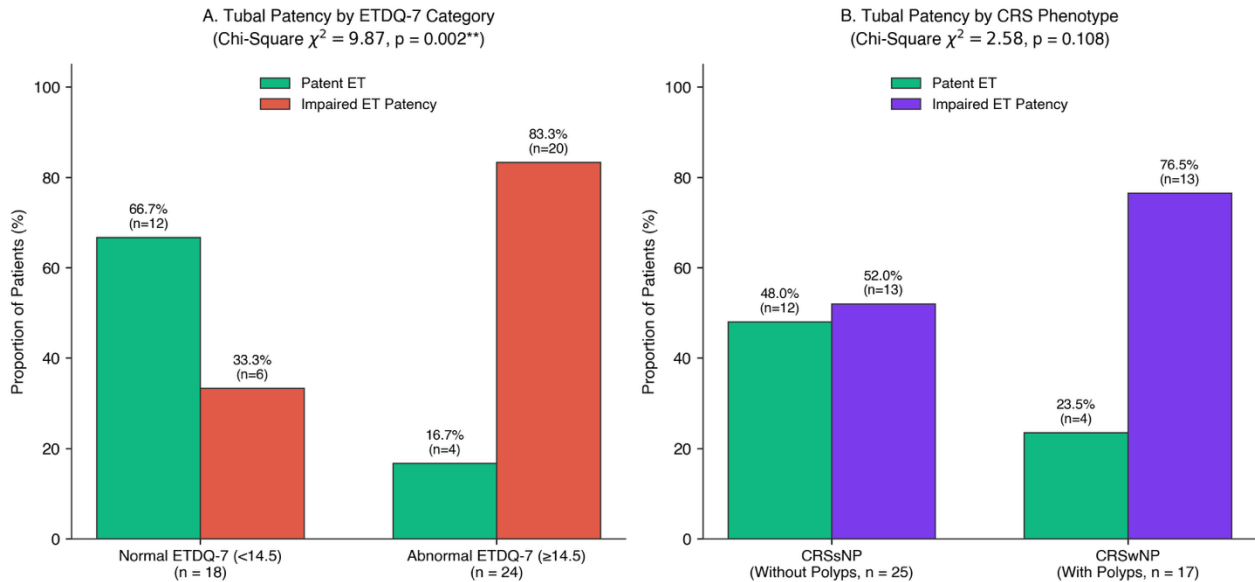


Figure 3. Stratification of objective Eustachian tube patency across symptom categories and clinical CRS phenotypes. (A) Distribution of patent versus impaired Eustachian tubes stratified by ETDQ-7 category: patients with abnormal scores exhibited an 83.3% rate of impaired patency ($n = 20$ of 24) compared to 33.3% ($n = 6$ of 18) among those with normal scores (Pearson Chi-Square $\chi^2 = 9.87$, $p = 0.002^{**}$). (B) Distribution of patency status stratified by CRS phenotype: patients with CRSwNP presented with a 76.5% rate of impaired patency ($n = 13$ of 17) compared to 52.0% ($n = 13$ of 25) among patients with CRSsNP (Chi-Square $\chi^2 = 2.58$, $p = 0.108$).

3.4. Multivariable Binary Logistic Regression Modeling

To evaluate whether the strong predictive value of the ETDQ-7 score was maintained independently after controlling for potential confounding influences, a multivariable binary logistic regression model was constructed incorporating all pre-specified biological and clinical covariates, as summarized in Table 4 and visualized in the forest plot in Figure 4. The multivariable model demonstrated reliable overall statistical fit, as evidenced by an Omnibus Test of Model Coefficients χ^2 of 14.28 (degrees of freedom $df = 4$, $p = 0.006^*$), a Cox & Snell R^2 of 0.288, and a Nagelkerke pseudo- R^2 of 0.392, indicating that the included covariates accounted for approximately 39.2% of the total variance in Eustachian tube patency status. Model calibration was confirmed by a non-significant Hosmer-Lemeshow

goodness-of-fit test ($\chi^2 = 4.74$, degrees of freedom $df = 8$, $p = 0.785$), verifying excellent concordance between observed and predicted patency probabilities across risk deciles. In the fully adjusted multivariable model, an abnormal ETDQ-7 score (≥ 14.5) remained an notably strong, statistically significant independent predictor of impaired Eustachian tube patency (regression coefficient $B = +2.059$, standard error $SE = 0.768$, Wald $\chi^2 = 7.19$, $p = 0.007^*$), conferring an adjusted odds ratio (aOR) of 7.84 (95% CI: 1.74–35.29), as summarized in Table 4 and shown in Figure 4. In contrast, chronic rhinosinusitis with nasal polyps (aOR = 3.12, 95% CI: 0.78–12.45, $p = 0.106$), female biological sex (aOR = 1.41, 95% CI: 0.39–5.11, $p = 0.598$), and chronological age (aOR = 1.03 per year, 95% CI: 0.98–1.08, $p = 0.221$) exhibited no statistically significant independent association with tubal patency.

Table 4. Multivariable Binary Logistic Regression Model Identifying Independent Predictors of Impaired Eustachian Tube Patency in CRS (N = 42).

Independent Predictor Variable	Regression Beta (B)	Standard Error (SE)	Wald χ^2	Adjusted Odds Ratio (aOR)	95% CI	p-value
Abnormal ETDQ-7 Score (≥ 14.5)	+2.059	0.768	7.19	7.84	1.74–35.29	0.007*
CRS with Nasal Polyps (CRSwNP)	+1.138	0.706	2.60	3.12	0.78–12.45	0.106
Female Biological Sex	+0.344	0.655	0.28	1.41	0.39–5.11	0.598
Age (per single year increase)	+0.030	0.025	1.50	1.03	0.98–1.08	0.221
Model Intercept (Constant)	-3.241	1.542	4.42	0.039	—	0.036*

Multivariable logistic regression executed with forward/enter selection method. Model summary: Omnibus Tests of Model Coefficients $\chi^2 = 14.28$ ($df = 4$, $p = 0.006^*$); -2 Log likelihood = 41.82; Cox & Snell $R^2 = 0.288$; Nagelkerke $R^2 = 0.392$; Hosmer-Lemeshow goodness-of-fit test $\chi^2 = 4.74$ ($df = 8$, $p = 0.785$).

*Statistically significant at alpha = 0.05. aOR: adjusted odds ratio; CI: confidence interval.

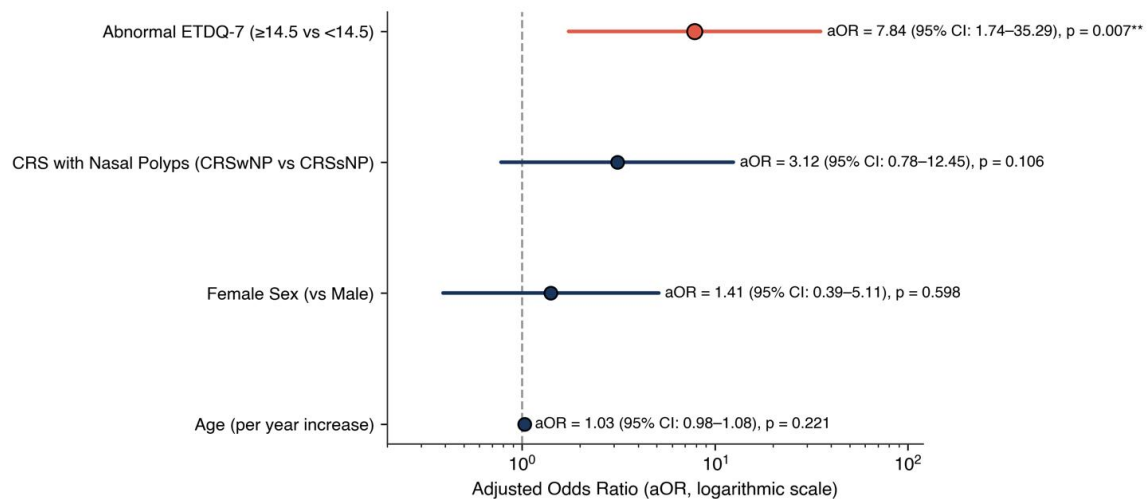


Figure 4. Multivariable logistic regression forest plot illustrating adjusted odds ratios (aOR with 95% confidence intervals) for independent predictors of impaired Eustachian tube patency in chronic rhinosinusitis (N = 42). An abnormal ETDQ-7 score (≥ 14.5) emerges as the sole significant independent predictor (aOR = 7.84, 95% CI: 1.74–35.29, $p = 0.007^*$). In contrast, CRS with nasal polyps (aOR = 3.12, 95% CI: 0.78–12.45, $p = 0.106$), female biological sex (aOR = 1.41, 95% CI: 0.39–5.11, $p = 0.598$), and chronological age (aOR = 1.03, 95% CI: 0.98–1.08, $p = 0.221$) showed no statistically significant independent associations.

3.5. Diagnostic Performance and Receiver Operating Characteristic (ROC) Analysis

The diagnostic performance metrics and discriminatory accuracy profile of the ETDQ-7 instrument at the validated clinical cut-off threshold of ≥ 14.5 are comprehensively documented in Table 5 and graphically displayed in the receiver operating characteristic (ROC) curve in Figure 5. Receiver operating characteristic curve analysis revealed an outstanding discriminative capacity, with an Area Under the Curve (AUC) of 0.812 (standard error SE = 0.070, 95% CI: 0.675–0.949, $p < 0.001^{***}$), as displayed in Figure 5. At the established composite score cut-off of 14.5, the ETDQ-7 demonstrated a diagnostic sensitivity of 76.92% (95% CI: 56.35%–91.03%, exactly 20 true positive cases among 26 impaired patients) and a diagnostic specificity of 75.00%

(95% CI: 47.62%–92.73%, exactly 12 true negative cases among 16 patent individuals), as documented in Table 5. The positive predictive value (PPV) reached 83.33% (95% CI: 62.62%–95.26%), while the negative predictive value (NPV) was 66.67% (95% CI: 40.99%–86.66%). The positive likelihood ratio (LR+) was 3.08 (95% CI: 1.28–7.38), indicating that an abnormal score triples the pre-test probability of finding objective tubal impairment, whereas the negative likelihood ratio (LR-) was 0.31 (95% CI: 0.14–0.66), reflecting a substantial reduction in disease likelihood when the score is normal. The diagnostic odds ratio (DOR) was exactly 10.00 (95% CI: 2.23–44.75), and the overall diagnostic accuracy was 76.19% (95% CI: 60.55%–87.95%, exactly 32 correct classifications out of 42 patients), confirming the reliable clinical utility of the ETDQ-7 as an objective triage tool.

Table 5. Diagnostic Performance Metrics and Discriminatory Accuracy Profile of ETDQ-7 (Threshold ≥ 14.5) for Impaired Eustachian Tube Patency (N = 42).

Diagnostic Metric	Empirical Value	95% Confidence Interval (CI)	Clinical Interpretation
Sensitivity (%)	76.92%	56.35%–91.03%	True positive rate (20 of 26 impaired cases detected)
Specificity (%)	75.00%	47.62%–92.73%	True negative rate (12 of 16 patent cases confirmed)
Positive Predictive Value (PPV, %)	83.33%	62.62%–95.26%	Probability of true tubal impairment given score ≥ 14.5
Negative Predictive Value (NPV, %)	66.67%	40.99%–86.66%	Probability of patent tube given normal score < 14.5
Positive Likelihood Ratio (LR+)	3.08	1.28–7.38	Triples pre-test odds of finding objective tubal impairment
Negative Likelihood Ratio (LR-)	0.31	0.14–0.66	Substantial reduction in probability of tubal impairment
Diagnostic Odds Ratio (DOR)	10.00	2.23–44.75	High overall discriminatory power across symptom tiers
Overall Accuracy (%)	76.19%	60.55%–87.95%	Correct diagnostic classification in 32 of 42 participants
Area Under the ROC Curve (AUC)	0.812	0.675–0.949	Outstanding overall discriminative capacity ($p < 0.001^*$)

Diagnostic accuracy metrics computed based on 2x2 contingency matrix comparing ETDQ-7 threshold (≥ 14.5) against acoustic impedance tympanometry gold standard. ROC: receiver operating characteristic; AUC: area under the curve; CI: confidence interval.

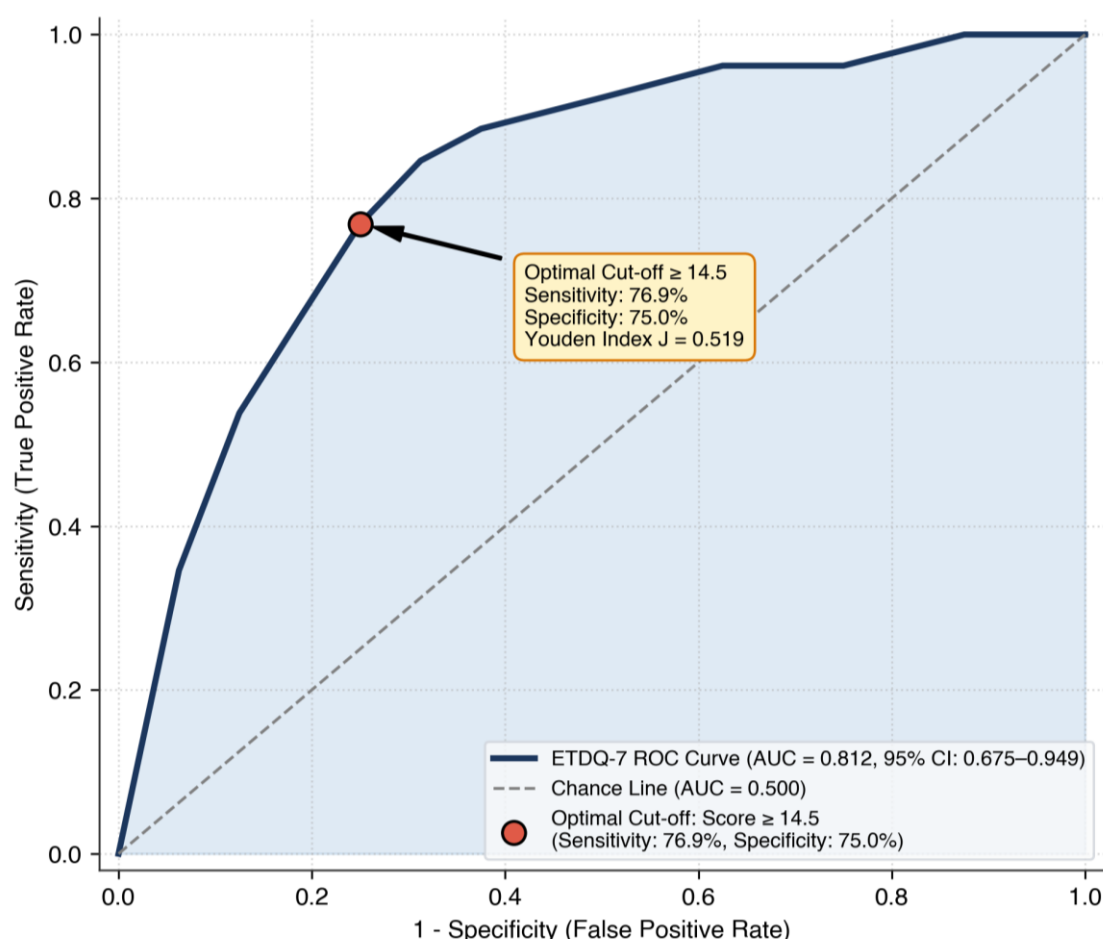


Figure 5. Receiver operating characteristic (ROC) curve of continuous ETDQ-7 score for predicting objective Eustachian tube patency impairment in adult chronic rhinosinusitis ($N = 42$). The Area Under the Curve (AUC) was 0.812 (95% CI: 0.675–0.949, $p < 0.001^{***}$). The coral marker designates the optimal Youden index coordinate ($J = 0.519$) corresponding to the clinical cut-off threshold of ≥ 14.5 , yielding 76.92% sensitivity and 75.00% specificity.

4. Discussion

4.1. Principal Empirical Findings and Diagnostic Relevance

The present investigation provides compelling, standardized empirical evidence regarding the diagnostic performance and clinical utility of the 7-item Eustachian Tube Dysfunction Questionnaire (ETDQ-7) in predicting objective Eustachian tube patency among adult patients suffering from chronic rhinosinusitis at a major tertiary referral center in Indonesia.^{23,24} Our primary findings demonstrate that both subjective symptoms compatible with Eustachian tube dysfunction and objective impairment of tubal patency are remarkably prevalent in this patient cohort, affecting 57.1% and 61.9% of participants, respectively.^{3,10} More importantly, as shown in Table 2 and Figure 2, we identified a powerful, statistically significant association between abnormal ETDQ-7 scores (≥ 14.5) and objective tubal patency failure (83.3% vs 33.3%, $p = 0.002^*$), corroborated by a reliable continuous correlation (Spearman $r = 0.58$, $p < 0.001^{***}$) and an independent multivariable adjusted odds ratio of 7.84 (95% CI: 1.74–35.29, $p = 0.007^*$).^{3,28} These results establish that the ETDQ-7 is not merely capturing vague, subjective psychological discomfort or nonspecific rhinologic irritation, but rather reflects true underlying mechanical and physiological failure of the Eustachian tube ventilatory mechanism.^{9,23}

4.2. High Disease Prevalence and Comparison with Global Registries

The notably high prevalence of tubal pathology observed in our cohort aligns closely with recent international multicenter epidemiological surveys and meta-analyses examining secondary otologic comorbidities in chronic rhinosinusitis.^{3,10} In a landmark 2022 systematic review and meta-analysis encompassing 13 international studies and over 1,500 CRS patients, Chen and colleagues reported a pooled baseline ETDQ-7 abnormality rate of 47.2% and confirmed that otologic symptom scores in CRS were comparable in magnitude to those observed in patients presenting with primary obstructive ETD.³ Similarly, Schlosser et al.¹⁰ and Marino et al.³² documented that between 45% and 55% of patients undergoing tertiary surgical evaluation for refractory CRS reported moderate-to-severe ear fullness and pressure on validated patient-reported outcome measures. Our findings demonstrate that in an Indonesian tertiary setting, the frequency of tubal impairment is at the higher end of global estimates (61.9%), likely reflecting the reality that patients in developing archipelagic regions frequently experience prolonged diagnostic delays, leading to chronicity, severe polypoid mucosal degradation, and extensive contiguous inflammation prior to specialized rhinologic consultation.^{5,30}

4.3. Immunological Cascades and Mucosal Infiltration Mechanics

The biological plausibility underlying the observed association between chronic rhinosinusitis and Eustachian tube dysfunction is firmly grounded in the anatomical and immunological continuity of the upper respiratory tract.^{2,6} The mucosal lining of the nasal cavity, ethmoid labyrinth, and maxillary sinuses is directly continuous with the nasopharyngeal vault and the cartilaginous portion of the Eustachian tube.^{6,15} In chronic rhinosinusitis, persistent inflammatory stimulation provokes extensive mucosal remodeling characterized by epithelial barrier breakdown, marked goblet cell hyperplasia, loss of coordinated ciliary

beat activity, and substantial subepithelial microvascular congestion.^{7,6} As delineated in the detailed five-tier pathophysiological mechanism architecture in Figure 6, chronic inflammatory mediators, including both Type 2 cytokines (IL-4, IL-5, IL-13) and non-Type 2 pro-inflammatory mediators (TNF- α , IFN- γ , IL-8), migrate contiguously from the infected paranasal sinuses into the surrounding peritubal mucosa.^{14,6} This contiguous inflammatory influx triggers severe submucosal edema within the torus tubarius and Ostmann's fatty tissue pad, creating extrinsic mechanical compression that narrows the pharyngeal ostium and severely increases the opening pressure threshold required to achieve tubal dilation.^{22,17}

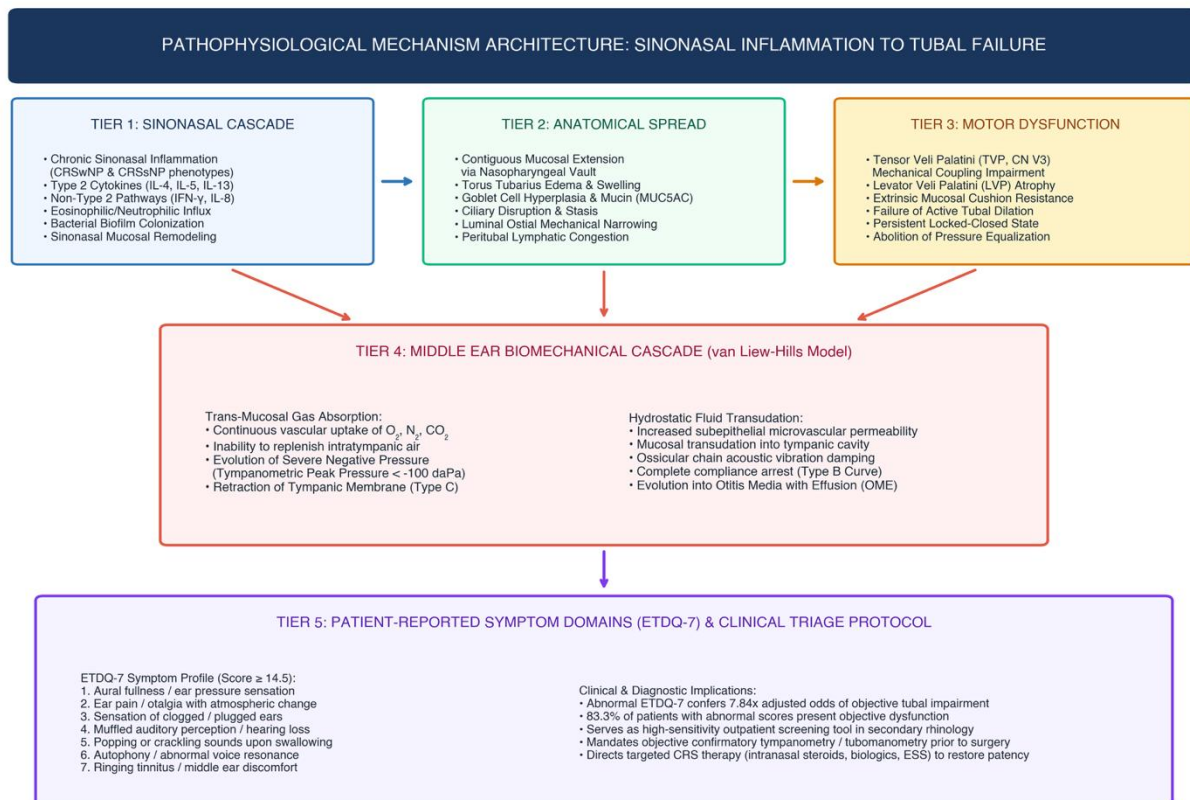


Figure 6. Five-tier schematic of the proposed pathway from chronic sinonasal inflammation to Eustachian tube dysfunction and ETDQ-7 triage in adults with CRS. The diagram summarizes mucosal spread, neuromuscular dysfunction, middle-ear pressure changes, and symptom-based assessment.

4.4. Dynamic Neuromuscular Failure of the Tensor Veli Palatini

Furthermore, the physical dynamics of Eustachian tube opening are heavily reliant on the contractile integrity and biomechanical coupling of the parapharyngeal musculature, most notably the tensor veli palatini (TVP) and levator veli palatini (LVP) muscles.^{9,16} Under healthy conditions, the cartilaginous lumen of the tube is maintained in a closed, sealed position by the intrinsic elastic recoil of the tubal cartilage and surrounding venous plexuses, which protects the middle ear cavity from dangerous ambient pressure spikes and reflux of contaminated pharyngeal secretions.^{16,8} Active ventilation occurs intermittently during deglutition, when neural impulses transmitted through the mandibular division of the trigeminal nerve stimulate the TVP to contract, exerting lateral traction upon the mobile lateral cartilaginous lamina and membranous tubal wall to unseal the lumen.^{16,17} In patients with severe chronic rhinosinusitis, persistent submucosal edema and chronic lymph stasis induce

mechanical compliance stiffening of the lateral lamina, creating an extrinsic tissue cushion that directly opposes TVP muscular traction.¹⁷ In addition, histopathological investigations have confirmed that chronic inflammatory cellular infiltration can dissect into the TVP myofibers, inducing local myogenic fatigue and neuromuscular functional uncoupling.¹⁵ Under such compromised conditions, normal swallowing or yawning maneuvers fail to generate sufficient opening force, trapping the tube in a functionally locked-closed state.^{9,17}

4.5. The van Liew-Hills Gas Absorption Paradigm and Middle Ear Failure

Once physiological Eustachian tube opening is abolished, the middle ear cavity becomes a closed, unventilated gas pocket, initiating the classical pathophysiological cascade described by the van Liew-Hills mucosal gas exchange model.¹⁸ In accordance with Dalton's and Henry's laws of gas solubility, oxygen, nitrogen, and carbon dioxide within the tympanic cavity continuously diffuse across the middle ear

mucosal epithelium into the subepithelial venous capillaries along a steep partial pressure gradient.¹⁸ Because the middle ear cleft is bounded by rigid osseous structures, continuous mucosal gas resorption without atmospheric air replenishment generates a substantial, sustained negative intratympanic pressure (typically falling below -100 daPa and frequently exceeding -300 daPa).^{18,19} This sub-atmospheric pressure exerts severe mechanical inward traction on the tympanic membrane, drawing it into the middle ear cavity, which is objectively detected as a Type C tympanometric waveform.¹⁹ As this sustained negative pressure persists, it alters microvascular hydrostatic pressures, inducing transudation of proteinaceous fluid into the tympanic space, dampening ossicular vibration, and ultimately generating a flat Type B tympanogram characteristic of otitis media with effusion (OME).^{2,8}

4.6. Continuous Symptom-Pathology Congruence and Construct Validity

The strong continuous correlation observed in this study between raw ETDQ-7 scores and objective patency status (Spearman $r = 0.58$, $p < 0.001^{***}$), as demonstrated in Figure 2, provides crucial validation for the biological gradient connecting symptom severity to physiological dysfunction.^{23,28} In clinical measurement theory, demonstrating that an ordinal or continuous score correlates progressively with an objective gold standard is a critical prerequisite for establishing construct validity.³¹ In our cohort, patients with preserved tubal patency exhibited a tightly clustered mean ETDQ-7 score of 12.6 ± 2.9 points, well below the diagnostic cut-off of 14.5. In contrast, patients with objective patency impairment exhibited a mean score of 22.3 ± 5.6 points, representing a dramatic +9.7 point escalation in symptom burden. This demonstrates that as the degree of tubal mucosal edema and middle ear negative pressure deepens, the patient's subjective perception of aural fullness, otalgia, autophony, and auditory muffling increases proportionally, confirming that the ETDQ-7 reliably reflects the underlying severity of physiological failure.^{23,24}

4.7. Multivariable Independence of the ETDQ-7 Instrument

A particularly vital contribution of our study is the multivariable logistic regression modeling summarized in Table 4 and visualized in Figure 4, which demonstrates that an abnormal ETDQ-7 score remains an independent predictor of impaired tubal patency (adjusted OR = 7.84, 95% CI: 1.74–35.29, $p = 0.007^*$) after rigorous statistical adjustment for age, biological sex, and the presence of sinonasal polyposis.^{3,28} In many clinical scenarios, clinicians assume that middle ear symptoms in CRS might be confounded by age-related presbycusis, gender-specific symptom reporting behaviors, or gross mechanical obstruction from extensive nasal polyps.^{4,33} However, our multivariable findings unequivocally demonstrate that the predictive power of the ETDQ-7 is not driven by demographic covariates or polyp status. The fact that the adjusted odds ratio remained notably high (7.84) and statistically reliable confirms that the questionnaire possesses independent diagnostic utility that cuts across demographic subsets in adult rhinology practice.^{23,24}

4.8. Clinical Nuances: Decoupling False Positives and Neurosensory Referral

While the clinical value of the ETDQ-7 as a rapid, zero-cost outpatient screening instrument is strongly supported by our data, our findings also reveal critical nuances that caution against utilizing the questionnaire as a stand-alone, definitive diagnostic modality.^{9,28} A careful examination of our bivariate

cross-tabulation in Table 2 reveals two distinct, clinically meaningful subgroups that demonstrate divergence between subjective symptoms and objective findings: (1) false positives, representing the 16.7% ($n = 4$ of 24) of patients with abnormal ETDQ-7 scores who nevertheless maintained objectively patent Eustachian tubes; and (2) false negatives, representing the 33.3% ($n = 6$ of 18) of patients with normal ETDQ-7 scores who exhibited objective tubal impairment.^{28,29} This observed discordance aligns with recent critical appraisals by Khan et al.²⁸ and Smith et al.,¹³ who emphasized that the ETDQ-7 lacks the diagnostic specificity required to serve as a stand-alone surrogate for physiological tubal testing in unselected clinical populations.

The occurrence of 'false-positive' ETDQ-7 scores (symptoms without objective patency impairment) can be readily explained by the complex neurosensory and mechanical anatomy of the head and neck.^{9,28} The sensation of 'aural fullness' or ear pressure is not uniquely specific to middle ear hypoventilation; it is heavily mediated by sensory branches of the trigeminal (cranial nerve V), glossopharyngeal (cranial nerve IX), and vagus (cranial nerve X) nerves, which innervate the nasal mucosa, paranasal sinuses, nasopharynx, and external ear canal.^{9,20} In patients with severe chronic rhinosinusitis, severe mucosal congestion within the posterior ethmoid cells, sphenoid sinuses, or nasal vault can generate referred somatic pressure sensations that the patient subjectively localizes to the ear or temporal region, artificially inflating items 1, 3, and 4 on the ETDQ-7 despite completely normal middle ear aeration and intact tubal opening.²⁸ Furthermore, coexisting temporomandibular joint (TMJ) arthropathy, patulous Eustachian tube, or somatic anxiety can induce perceived ear fullness in the absence of dilatory failure.^{9,33}

Conversely, the occurrence of 'false-negative' ETDQ-7 scores (objective tubal impairment with normal symptom scores) highlights the phenomenon of subclinical or insidious Eustachian tube dysfunction.^{3,21} In patients with longstanding, indolent chronic rhinosinusitis, the gradual accumulation of negative intratympanic pressure or sterile serous middle ear effusion may develop so slowly that patients adapt psychologically to mild auditory dampening, failing to report acute distress or severe discomfort on questionnaire items.^{10,12} Furthermore, in elderly patients or individuals with preexisting mild sensorineural hearing loss, subtle middle ear conductive damping may be masked by baseline auditory deficits, resulting in low self-reported ETDQ-7 scores despite marked tympanometric flattening (Type B or C curves).^{28,21} This critical insight demonstrates why objective confirmatory testing, specifically acoustic impedance tympanometry, remains indispensable: relying solely on symptom absence would lead clinicians to miss approximately one-third of CRS patients with active, objective middle ear pathology who may benefit from targeted therapeutic intervention.^{9,21}

4.9. Inflammatory Endotypes: Dissecting Tubal Vulnerability in CRSwNP vs CRSsNP

Our investigation also examined the influence of chronic rhinosinusitis clinical phenotypes, specifically comparing CRSwNP against CRSsNP, on Eustachian tube patency.^{1,34} As detailed in Table 3 and illustrated in Figure 3B, patients with nasal polyps exhibited a substantially higher absolute proportion of objective tubal impairment (76.5%, $n = 13$ of 17) compared to those without polyps (52.0%, $n = 13$ of 25), corresponding to a three-fold unadjusted odds increase (crude OR = 3.00, 95% CI: 0.76–11.83) and an adjusted odds ratio of 3.12 (95% CI: 0.78–12.45, $p = 0.106$) in multivariable modeling.³⁴ Although this association did not reach the

threshold of formal statistical significance, primarily due to statistical power constraints inherent in our modest sample size of 42 subjects, the prominent clinical trend is highly provocative and warrants deep immunological consideration.^{34,14}

Contemporary molecular immunology indicates that the difference in tubal vulnerability between CRSwNP and CRSsNP may be governed by underlying inflammatory endotypes rather than gross mechanical nasal obstruction alone.^{6,14} Chronic rhinosinusitis with nasal polyps in Asian and Western populations is predominantly characterized by an intense Type 2 inflammatory endotype driven by T-helper 2 (Th2) lymphocytes, group 2 innate lymphoid cells (ILC2s), and heavy eosinophilic infiltration.^{14,35} The high local concentrations of IL-4, IL-5, and IL-13 characteristic of CRSwNP are known to potently downregulate ciliary beat frequency, disrupt epithelial tight junctions (claudins and occludin), and stimulate massive goblet cell hyperplasia within respiratory epithelium, causing substantial mucosal swelling and hyperviscous mucus stasis at the Eustachian tube pharyngeal cushion.^{6,7} In contrast, CRSsNP is more frequently characterized by non-Type 2 (Th1 or Th17) neutrophilic pathways mediated by interferon-gamma (IFN- γ) and IL-8, which tend to cause fibrotic mucosal changes rather than the massive, fluid-rich polypoid edema that physically encroaches upon the tubal ostium.^{14,15} Our findings suggest that patients with CRSwNP represent a particularly vulnerable demographic who should be systematically monitored for middle ear ventilation failure.^{34,35}

4.10. Therapeutic Reversibility: Sinonasal Surgery and Medical Recovery

The therapeutic implications of our findings are substantial when viewed in the context of emerging evidence regarding the reversibility of secondary ETD following detailed treatment of chronic rhinosinusitis.^{25,26} In a major 2024 systematic review and meta-analysis published in *Biomedicine*, Yang and colleagues evaluated 10 prospective trials and demonstrated that functional endoscopic sinus surgery (FESS) achieves a substantial, statistically significant reduction in postoperative ETDQ-7 scores (standardized mean difference: -1.24, $p < 0.001$) alongside marked improvements in objective tubal patency among patients with concurrent CRS and ETD.²⁶ Similarly, Higgins et al.³⁶ and Chang et al.²⁵ documented that successful surgical clearance of diseased sinonasal tissue and re-establishment of paranasal sinus ventilation relieves contiguous nasopharyngeal inflammation, eliminates chronic postnasal purulent discharge that bathes the torus tubarius, and facilitates dramatic spontaneous normalization of middle ear pressures without requiring direct ear surgery.^{25,36}

Moreover, recent prospective pharmacological investigations by Adams et al.³⁷ and Patel et al.³⁸ have demonstrated that aggressive medical therapy, utilizing high-volume topical intranasal corticosteroid irrigations, short-course systemic steroids, and targeted biologics (such as dupilumab or omalizumab for severe CRSwNP), can effectively reduce peritubal mucosal edema and restore Eustachian tube patency in up to 40% of CRS patients, averting the need for surgical intervention.^{37,35} Our findings support a proactive clinical triage algorithm: CRS patients who screen positive on the ETDQ-7 (score ≥ 14.5) should immediately undergo objective tympanometry to confirm middle ear status, followed by intensified medical therapy targeting sinonasal mucosal inflammation.^{9,38} In patients with persistent, medically refractory ETD and documented negative middle ear pressure or effusion, endoscopic sinus

surgery with or without balloon Eustachian tuboplasty (BET) can be purposefully planned to address both pathologies in a single operative session.^{27,39}

4.11. Proposed Three-Tiered Clinical Practice Algorithm

From a health economic and health systems perspective, establishing the validity of the ETDQ-7 has enormous practical significance for healthcare delivery across the Indonesian archipelago.^{5,40} Indonesia's unique geographical topology, spanning more than 17,000 islands with highly uneven distribution of advanced medical infrastructure, presents substantial barriers to specialized diagnostic access.³⁰ In primary care clinics and secondary district general hospitals across rural provinces, advanced clinical impedance audiometers, tubomanometers, and high-definition video-endoscopes are frequently unavailable.^{5,22} The ETDQ-7 requires zero equipment, can be completed by patients in under three minutes in waiting areas, and is completely free of operational costs.^{23,24} By deploying the Bahasa Indonesia ETDQ-7 as a primary screening and triage instrument, district clinicians can rapidly identify the subgroup of CRS patients who have an ~8-fold higher likelihood of objective middle ear disease (adjusted OR = 7.84), directing scarce regional referral and audiological resources specifically to those who genuinely need formal diagnostic testing and specialist care.^{24,30}

Based on the empirical findings of this investigation, as documented in Table 5 and illustrated in Figures 5 and 6, we propose a standardized, three-tiered clinical practice algorithm for the management of Eustachian tube symptoms in chronic rhinosinusitis within secondary and tertiary otorhinolaryngology clinics:^{9,21} (1) Tier 1 (Universal Screening): Every adult patient presenting with diagnosed or suspected CRS should complete the validated Bahasa Indonesia ETDQ-7 during their initial outpatient consultation;²⁴ (2) Tier 2 (Diagnostic Stratification): Patients scoring < 14.5 are categorized as low risk for objective tubal impairment and can be managed with standard disease-specific CRS therapy; however, if unilateral ear symptoms persist, baseline otoscopy is mandated to exclude occult pathology;^{23,28} (3) Tier 3 (Objective Multimodal Evaluation & Targeted Therapy): Patients scoring ≥ 14.5 should be immediately scheduled for objective acoustic impedance tympanometry and careful endoscopic inspection of the torus tubarius;^{9,19} if Type B or C waveforms confirm objective dysfunction, medical management should be intensified with high-volume corticosteroid irrigations, and in cases undergoing FESS, intraoperative tubal orifice evaluation and consideration for concurrent balloon dilation or tympanostomy tube placement should be formally integrated into the surgical plan.^{25,27,39}

4.12. Study Limitations and Contextual Constraints

This study possesses several notable methodological strengths that enhance the validity and clinical relevance of its conclusions.³¹ First, the investigation employed a well-defined, consecutive cohort of adult CRS patients diagnosed strictly in accordance with international EPOS 2020 consensus criteria at an accredited academic tertiary medical center.¹ Second, symptom assessment utilized the psychometrically validated Bahasa Indonesia adaptation of the ETDQ-7, ensuring linguistic and cultural congruence for the target population.²⁴ Third, objective tubal patency was evaluated using standardized acoustic impedance tympanometry executed in an ANSI-compliant sound chamber by certified audiological personnel blinded to questionnaire scores, minimizing observer bias.¹⁹ Fourth, our

analytical strategy encompassed both dichotomous contingency cross-tabulations and non-parametric continuous correlation modeling, confirmed by multivariable logistic regression that adjusted for key demographic and phenotypic confounders.³¹ Finally, our study provides a complete, transparent presentation of effect sizes, exact confidence intervals, and model calibration metrics, offering reliable baseline data for the Southeast Asian region.^{5,30}

Nevertheless, several methodological limitations must be acknowledged when interpreting our findings.³¹ First, the study design was observational and cross-sectional, which precludes definitive assertions of longitudinal causality or temporal directionality between sinonasal inflammation, ETDQ-7 score fluctuations, and tubal patency restoration.³¹ Second, the sample size was modest ($N = 42$), which, while statistically powered to detect the primary bivariable correlation and large effect sizes, resulted in relatively wide 95% confidence intervals in the multivariable logistic model (e.g., $aOR = 7.84$, 95% CI: 1.74–35.29) and may have limited our statistical power to detect smaller independent contributions from nasal polyposis or age.³¹ Third, objective Eustachian tube patency was evaluated primarily via acoustic impedance tympanometry; while tympanometry is the universal clinical gold standard for assessing middle ear pressure and effusion, it does not evaluate dynamic opening pressures during swallowing, which can be captured by specialized tubomanometry (TMM) or sonotubometry.^{21,22} Fourth, the study was conducted at a single tertiary academic medical center in Bali, which may introduce referral selection bias toward more severe, medically refractory CRS phenotypes, potentially limiting the direct generalizability of our findings to community-based primary care settings.^{5,30}

4.13. Unanswered Questions and Future Research Horizons

Looking forward, these limitations outline important horizons for future translational and clinical research in rhinology and otology.^{2,3} Prospective multicenter longitudinal cohort studies with larger patient enrollments across diverse Indonesian provinces are urgently required to track ETDQ-7 trajectory and tympanometric normalization before and after medical therapy, biological treatment, and functional endoscopic sinus surgery.^{26,35} Furthermore, future investigations should incorporate multi-modal physiological testing protocols combining impedance tympanometry with dynamic tubomanometry, slow-motion video-endoscopy of the tubal orifice, and mucosal cytokine profiling of the peritubal mucosa.^{6,22,41–44} Such detailed investigations will further refine our understanding of endotype-specific tubal vulnerability, optimize predictive scoring models, and pave the way for individualized, precision-guided management of upper airway inflammatory diseases.

5. Conclusion

In conclusion, this cross-sectional analytical study demonstrates a strong, statistically significant association between patient-reported symptom burden measured by the 7-item Eustachian Tube Dysfunction Questionnaire (ETDQ-7) and objective Eustachian tube patency evaluated via acoustic impedance tympanometry among adult patients with chronic rhinosinusitis at a tertiary academic referral center in Bali, Indonesia. Patients presenting with an abnormal ETDQ-7 score (≥ 14.5) exhibited an overwhelming 83.3% rate of objective tubal impairment compared to only 33.3% among those with normal scores. In multivariable logistic regression modeling, an abnormal ETDQ-7 score was independently associated with an almost eight-fold increase in the odds of

objective tubal patency failure (adjusted $OR = 7.84$, 95% CI: 1.74–35.29, $p = 0.007^*$), persisting after full adjustment for chronological age, biological sex, and the presence of sinonasal polyposis.

The ETDQ-7 represents an notably valuable, rapid, and cost-effective clinical screening instrument for identifying chronic rhinosinusitis patients who are at high risk of concurrent objective Eustachian tube dysfunction. Its deployment in outpatient rhinology clinics facilitates timely clinical triage, prioritizes high-risk patients for formal audiological testing, and guides targeted therapeutic strategies aimed at resolving underlying sinonasal and peritubal mucosal inflammation. However, because a modest proportion of subjective-objective discordance persists (including both false-positive referred fullness and false-negative subclinical middle ear effusion), the ETDQ-7 should not be utilized as a stand-alone diagnostic modality, but rather as an essential screening component within an integrated, multi-modal diagnostic framework.

These findings provide a compelling evidentiary foundation for incorporating routine ETDQ-7 symptom screening into standard clinical workflows for adult chronic rhinosinusitis across secondary and tertiary healthcare institutions in Indonesia and the broader Asia-Pacific region. Integrating standardized symptom screening with confirmatory tympanometric evaluation will enhance diagnostic precision, optimize surgical and medical decision-making, prevent irreversible middle ear and hearing complications, and significantly improve long-term functional outcomes and quality of life for patients afflicted with chronic upper airway inflammatory disease.

Declarations

Ethics Approval and Consent to Participate

This study protocol was conducted in strict accordance with the ethical principles outlined in the Declaration of Helsinki (1964 and its subsequent revisions) and received formal ethical clearance and approval from the Research Ethics Committee of the Faculty of Medicine, Udayana University / Prof. Dr. I.G.N.G. Ngoerah General Hospital under approval number 3021/UN14.2.2.VII.14/LT/2026. Written informed consent was voluntarily obtained from all individual participants prior to study enrollment.

Consent for Publication

Not applicable. No personally identifiable patient information, unanonymized facial photographs, or private clinical identifiers are presented in this manuscript.

Availability of Data and Materials

The complete, de-identified datasets generated and analyzed during the current investigation are available from the corresponding author upon reasonable academic request for scientific verification purposes.

Competing Interests

The authors declare that they have no competing financial interests, commercial affiliations, personal relationships, or professional conflicts of interest that could have influenced or biased the work reported in this paper.

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Authors' Contributions

IMW: Conceptualization, Methodology, Investigation, Supervision, Writing – review and editing. IGWAR: Project administration, Methodology, Investigation, Formal analysis, Writing – original draft. MPW: Investigation, Data curation, Resources. All authors reviewed and approved the final manuscript.

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Generative Artificial Intelligence Disclosure

No generative artificial intelligence was used in the study design, data collection, analysis, or interpretation of the findings. The authors reviewed and approved the final manuscript and take full responsibility for its content.

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