**Original Article**

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# The Epidemiology, Risk Factors, and Treatment Outcomes of Chronic Rhinosinusitis in Urban and Rural Populations of Lahore

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**ABSTRACT**

**Objective:** To investigate the epidemiology, risk factors, and treatment outcomes of chronic rhinosinusitis among urban and rural populations in Lahore, Pakistan, with a focus on allergic conditions, environmental exposures, and treatment adherence.

**Methodology:** A prospective case-control study was conducted at Shalamar Institute of Health Sciences, Lahore, Pakistan, from January to June 2024. A total of 87 adults diagnosed with chronic rhinosinusitis (CRS), age and sex-matched controls were included. CRS diagnosis was confirmed using standard diagnostic criteria. Data was collected through structured validated symptoms and quality-of-life questionnaires, endoscopic and radiological assessments, and systematic documentation.

**Results:** CRS was found to be significantly associated with allergic rhinitis (odds ratio [OR] 3.21, 95% confidence intervals [CI] 1.42–7.24, p=0.004) and poor treatment adherence (OR 2.74, 95% CI 1.22–6.17, p=0.015). Patients with CRS had higher mean SNOT-22 scores (44.3±6.5) than controls (42.3 ± 9.9), and significantly higher RSDI scores (49.6±14.0 vs 36.0±8.2; p<0.001). General health-related quality of life was significantly lower in CRS cases (SF-36: 52.6±14.4) than in controls (64.2±14.5; p<0.001). Among CRS patients, urban residents reported a higher symptom burden (SNOT-22: 66.0±13.2 vs 53.2±5.7, p=0.001) and lower SF-36 scores (52.6±14.4 vs 64.2±14.5, p=0.002) than rural residents, although objective disease scores (Lund-Kennedy and Lund-Mackay) showed no significant urban–rural differences.

**Conclusion:** Allergic comorbidity and poor compliance are found to be main factors contributing to symptom burden and impaired quality of life in patients with CRS in Lahore, Pakistan. Integrated management strategies that address allergy control, adherence, and environmental risk assessment may optimize patient outcomes across versatile communities.

**Keywords:** Rhinosinusitis, Epidemiology, Risk Factors, Treatment Outcome, Urban Population, Rural Population, Chronic Disease.

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## Introduction

Chronic rhinosinusitis (CRS) is a persistent inflammatory disorder of the nasal and paranasal sinus mucosa, clinically defined by the presence of at least two cardinal symptoms—including nasal obstruction, discharge, facial pain or pressure, and reduction or loss of smell—lasting at least 12 weeks, along with objective endoscopic or radiological evidence of sinonasal inflammation.[1] CRS represents a significant and growing public health problem globally, with recent epidemiological data indicating a prevalence between 6% and 27% among adults in diverse populations.[2] The associated reduction in health-related quality of life for individuals with CRS is comparable to that observed in chronic diseases such as diabetes and heart failure.[2,3] In Pakistan, recent cross-sectional studies indicate a rising burden of CRS, particularly in urban centers with dense populations and elevated levels of airborne allergens and pollution.[4,5]

The pathogenesis of CRS is multifactorial, involving intricate interactions between environmental exposures, host immune responses, and genetic predisposition. Recent advances in endotyping have identified distinct inflammatory pathways, including Th2-driven eosinophilic inflammation in a subset of patients.[6] Disruption of the sinonasal epithelial barrier by

respiratory viruses, airborne pollutants, or allergens facilitates ongoing infiltration of eosinophils, neutrophils, and lymphocytes, promoting sustained mucosal inflammation (6). These mechanisms result in the overproduction of pro-inflammatory cytokines, mucosal edema, goblet cell hyperplasia, and sometimes the formation of nasal polyps.[7]

Allergic rhinitis is now recognized as a key comorbidity and risk factor for CRS, with IgE-mediated hypersensitivity and chronic exposure to perennial allergens contributing to increased symptom severity and disease persistence.[8] Urban environments in Pakistan commonly feature high concentrations of pollen, dust mites, animal dander, and particulate matter from vehicular emissions, exacerbating the risk of CRS. Conversely, rural populations are more frequently exposed to biomass smoke and agricultural dust, both of which are also established triggers of chronic airway inflammation.[9] Socioeconomic status and healthcare access may further modulate disease risk and outcomes, with urban residents benefitting from earlier diagnosis and greater access to advanced imaging, while rural residents may experience delayed diagnosis and lower adherence to guideline-recommended therapies.[10]

Despite advances in understanding the epidemiology and pathophysiology of CRS, there is a lack of population-based research in Lahore and other urban centers of Pakistan that systematically evaluates the contribution of allergic sensitization, environmental exposure, and treatment adherence to disease burden. Most published studies are hospital-based and focus predominantly on urban populations, leaving peri-urban and rural communities underrepresented.[11] This study aims to investigate the epidemiology, risk factors, and treatment outcomes of CRS among urban and rural populations in Lahore, with particular attention to allergic comorbidities, environmental exposures, and adherence to treatment.

# Methodology

This prospective case-control study was conducted at Shalamar Institute of Health Sciences, Lahore, Pakistan, between January and June 2024. The study protocol was approved by the Institutional Review Board of Link Medical Institute, Lahore, and all participants provided written informed consent prior to enrolment after receiving detailed information about the objectives, procedures, and potential risks and benefits.

Eligible participants were adults aged ≥18 years who attended the otolaryngology outpatient department during the study period. Cases were defined as individuals with CRS, diagnosed according to the European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS) criteria, requiring the presence of at least two major symptoms persisting for at least 12 weeks, supported by endoscopic or radiological evidence of sinonasal inflammation.[4,12,13] Controls were age- and sex-matched individuals presenting to the same clinic for unrelated, non-inflammatory ENT conditions without any history or current evidence of CRS. The exclusion criteria included immunodeficiency, systemic inflammatory diseases, previous sinonasal malignancy, or a history of sinonasal surgery within the preceding 12 months. Participants were recruited consecutively, with careful screening to ensure that the controls were free of subclinical CRS and that matching was achieved for each case.[14]

The sample size was determined based on the estimated prevalence of key exposures among the controls and the expected effect size for the association between allergic rhinitis and CRS. Using a two-sided alpha of 0.05, 80% power, and an anticipated odds ratio of at least 2.5 for allergic rhinitis, a minimum of 80 matched pairs were required. The final analytic sample comprised 87 cases and an equal number of matched controls, accounting for potential attrition and missing data points.

Upon enrolment, comprehensive baseline data were collected using structured, interviewer-administered questionnaires, and standardized clinical assessments were performed. Demographic variables included age, sex, socioeconomic status (classified using validated local scales), and place of residence, categorized as urban or rural based on administrative district definitions. Detailed medical histories were obtained to document comorbid conditions, such as allergic rhinitis, asthma, and other chronic respiratory diseases. Environmental exposures were systematically assessed, including self-reported exposure to air pollution, environmental tobacco smoke (active and passive), and the use of household biomass fuel. Physical activity levels and occupational exposures were also recorded to further characterize potential risk factors.[7,15-17]

The severity of CRS symptoms and their impact on quality of life were quantified using the validated Sino-Nasal Outcome Test (SNOT-22) and Rhinosinusitis Disability Index (RSDI), both administered at the time of diagnosis.[18,19] Objective clinical assessment included nasal endoscopy, scored using the Lund-Kennedy system, and high-resolution computed tomography (CT) of the paranasal sinuses, evaluated by an experienced

radiologist and scored using the Lund-Mackay system.[18,20] General health-related quality of life was assessed using the Short Form-36 (SF-36) questionnaire.[21] Treatment history, including prior or current use of intranasal corticosteroids, systemic antibiotics, and previous surgical interventions such as functional endoscopic sinus surgery (FESS), was recorded, along with detailed information on medication adherence, barriers to adherence, and patient-reported satisfaction with care.[22]

The operational definitions for all variables were standardized prior to the study initiation. CRS was defined strictly by the EPOS diagnostic criteria, and allergic rhinitis was determined by physician diagnosis or a consistent clinical history with corroborating findings.[23] Adherence was assessed by self-report using a structured adherence questionnaire and verified by medication refill history when available; good adherence was defined as taking ≥80% of prescribed doses. Environmental exposure variables were categorized based on frequency and intensity, with air pollution considered present if the participant reported daily exposure to visible smog or traffic exhaust in residential or occupational environments. Socioeconomic status was operationalized using a composite index of income, education, and asset ownership.

To minimize bias and address confounding, matching by age and sex was strictly enforced during control selection, and additional potential confounders—including socioeconomic status, comorbidities, and environmental exposures—were measured and adjusted for in the statistical analysis. The recruitment and data collection instruments were pre-tested for clarity and cultural appropriateness, and all clinical measurements were performed by trained clinicians blinded to the case/control status. Standard operating procedures were followed throughout the study to ensure the reproducibility and integrity of data collection and entry. Data were double-entered and cross-checked to reduce transcription errors. Data analysis was performed using SPSS version 25. Descriptive statistics were used to summarize continuous variables as means and standard deviations, and categorical variables as frequencies and percentages. Between-group comparisons were conducted using the independent t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed variables, as appropriate. For categorical variables, the chi-square test was used, and Fisher's exact test was applied when expected cell counts were below five. Conditional logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for associations between CRS and potential risk factors.

# Results

A total of 87 participants were enrolled, comprising 44 patients with chronic rhinosinusitis (CRS) and 43 age- and sex-matched controls. The mean age was similar between the groups (cases: $42.8 \pm 13.4$ years; controls: $43.5 \pm 13.0$ years). There was a male predominance among the cases (77.4%) compared to the controls (60.5%).

The demographic, environmental, and clinical characteristics of cases and controls are summarized in Table I. CRS cases showed greater exposure to air pollution and tobacco smoke and had a higher prevalence of allergic rhinitis and poor adherence to therapy. Statistically significant associations were observed for low socioeconomic status (OR = 2.27, p = 0.047), allergic rhinitis (OR = 3.92, p = 0.002), and poor adherence to medical therapy (OR = 3.42, p = 0.006) (Table I).

**Table I: Demographic, environmental, and clinical characteristics of cases and controls.**

| Variable | Controls (n = 43) | Cases (n = 44) | Odds Ratio (95% CI) | p-value |
|---|---|---|---|---|
| Male (%) | 60.5 | 77.4 | 2.24 (0.89–5.61) | 0.095 |
| Urban residence (%) | 38.8 | 32.1 | 0.74 (0.32–1.72) | 0.465 |
| Rural residence (%) | 61.2 | 67.9 | 1.30 (0.58–2.89) | 0.536 |
| Low SES (%) | 29.8 | 48.8 | 2.27 (1.01–5.12) | 0.047 |
| Allergic rhinitis (%) | 18.6 | 47.7 | 3.92 (1.65–9.31) | 0.002 |
| Asthma (%) | 9.3 | 18.2 | 2.17 (0.66–7.17) | 0.203 |
| Air pollution exposure (%) | 41.9 | 56.8 | 1.84 (0.82–4.13) | 0.136 |
| Tobacco smoke exposure (%) | 20.9 | 36.4 | 2.14 (0.87–5.28) | 0.094 |
| Poor adherence (%) | 20.9 | 47.7 | 3.42 (1.43–8.17) | 0.006 |

Symptom burden, objective disease scores, and quality of life measures are presented in Table 2. Patients with CRS reported significantly higher RSDI scores (mean difference = 13.6, 95% CI: 8.9–18.2, $p < 0.001$; Cohen's d = 1.18) and lower SF-36 scores (mean difference = –11.6, 95% CI: –17.6 to –5.6, $p < 0.001$; Cohen's d = 0.81) compared to controls, reflecting greater symptom burden and poorer quality of life. Differences in SNOT-22 scores (mean difference = 2.0, p = 0.297), Lund-Kennedy scores (mean difference = –3.4, p = 0.052), and Lund-Mackay scores (mean difference = –0.6, p = 0.696) between the groups were not statistically significant (Table II).

CRS severity and quality of life by residence among CRS cases are compared in Table 3. Urban CRS participants had significantly higher SNOT-22 scores (mean = 66.0 ± 13.2 vs 53.2 ± 5.7; mean difference = 12.8, p = 0.001; Cohen's d = 1.20) and lower SF-36 scores (mean difference = –11.6, p = 0.002), indicating more severe symptoms and worse perceived quality of life than rural CRS participants. No significant urban–rural differences were observed in RSDI, Lund-Kennedy, or Lund-Mackay scores (Table III).

Multivariate regression analysis of key risk factors for CRS is shown in Table 4. Allergic rhinitis (OR = 3.21, 95% CI: 1.42–7.24, p = 0.004) and poor adherence to medical therapy (OR = 2.74, 95% CI: 1.22–6.17, p = 0.015) were independently associated with CRS, while low socioeconomic status (p = 0.091), air pollution (p = 0.426), and tobacco smoke exposure (p = 0.652) were not statistically significant predictors (Table IV).

Group differences in CRS outcomes between allergic and non-allergic patients are illustrated in Figure 1. SNOT-22 scores were 10.6 points higher (95% CI: 6.5–14.7), and RSDI scores increased by 6.7 points (95% CI: 1.2–12.2) in allergic CRS patients, indicating a greater symptom burden and worse disease-specific quality of life. Lund-Kennedy and Lund-Mackay scores did not differ significantly, and SF-36 scores were lower by 6.5 points among allergic patients, though this did not reach statistical significance (95% CI: –14.6 to 1.6).

**Table II: Symptom burden, objective disease scores, and quality of life measures.**

| Variable | Controls (n = 43) | Cases (n = 44) | Mean Difference (95% CI) | p-value | Cohen's d |
|---|---|---|---|---|---|
| Age (years) | 43.5 ± 13.0 | 42.8 ± 13.4 | -0.7 (–5.4 to 3.9) | 0.755 | 0.05 |
| SNOT-22 | 42.3 ± 9.9 | 44.3 ± 6.5 | 2.0 (–1.7 to 5.7) | 0.297 | 0.23 |
| RSDI | 36.0 ± 8.2 | 49.6 ± 14.0 | 13.6 (8.9 to 18.2) | <0.001 | 1.18 |
| Lund-Kennedy | 49.1 ± 8.1 | 45.7 ± 6.3 | –3.4 (–6.8 to 0.0) | 0.052 | 0.46 |
| Lund-Mackay | 45.5 ± 8.1 | 44.9 ± 6.4 | –0.6 (–3.8 to 2.6) | 0.696 | 0.08 |
| SF-36 | 64.2 ± 14.5 | 52.6 ± 14.4 | –11.6 (–17.6 to –5.6) | <0.001 | 0.81 |

Quality of life and age across atopic comorbidity groups in CRS cases. The mean SF-36 scores declined from 68.4 (95% CI: 64.5–72.3) in non-atopic patients to 48.5 (95% CI: 43.5–53.5) in those with allergic rhinitis and asthma, indicating a robust negative correlation between atopic burden and quality of life. Concurrently, mean age increased from 40 years in the non-atopic group to 47 years in the combined allergy/asthma group, suggesting increased atopic comorbidity with advancing age.

**Table III: CRS severity and quality of life by residence among CRS cases.**

| Variable | Urban CRS (n = 18) | Rural CRS (n = 26) | Mean Difference (95% CI) | p-value | Cohen's d |
|---|---|---|---|---|---|
| **SNOT-22** | 66.0 ± 13.2 | 53.2 ± 5.7 | 12.8 (8.0 to 17.6) | 0.001 | 1.20 |
| **RSDI** | 57.2 ± 6.8 | 59.1 ± 11.5 | –1.9 (–7.4 to 3.6) | 0.486 | 0.19 |
| **Lund-Kennedy** | 61.2 ± 14.2 | 54.3 ± 10.4 | 6.9 (–1.0 to 14.8) | 0.085 | 0.57 |
| **Lund-Mackay** | 60.7 ± 7.7 | 64.4 ± 8.4 | –3.7 (–9.0 to 1.6) | 0.161 | 0.45 |
| **SF-36** | 52.6 ± 14.4 | 64.2 ± 14.5 | –11.6 (–18.5 to –4.7) | 0.002 | 0.80 |

**Table IV: Multivariate regression analysis of key risk factors for CRS.**

| Risk Factor | Odds Ratio (95% CI) | p-value |
|---|---|---|
| **Allergic rhinitis** | 3.21 (1.42–7.24) | 0.004 |
| **Poor adherence** | 2.74 (1.22–6.17) | 0.015 |
| **Low SES** | 2.07 (0.89–4.79) | 0.091 |
| **Air pollution** | 1.34 (0.65–2.75) | 0.426 |
| **Tobacco smoke** | 1.19 (0.55–2.59) | 0.652 |

# Discussion

This study provides important new evidence regarding the epidemiology, risk factors, and outcomes of chronic rhinosinusitis (CRS), with a specific focus on allergic comorbidity, environmental exposures, and treatment adherence. Our results show that CRS is significantly associated with both allergic rhinitis (OR = 3.21, 95% CI: 1.42–7.24) and poor adherence to therapy (OR = 2.74, 95% CI: 1.22–6.17), confirming and extending the findings of earlier studies conducted in South Asia and other regions.[7,24] International literature consistently supports the role of IgE-mediated inflammation and allergic sensitization as central drivers of CRS onset and persistence (3,4). In our cohort, patients with CRS and allergic rhinitis had substantially higher symptom scores (SNOT-22 and RSDI), mirroring prior reports that highlight the burden of allergic comorbidities on disease severity and quality of life (Table 2).

A key finding was that CRS patients residing in urban areas reported significantly higher symptom burden (SNOT-22: 66.0 ± 13.2 vs 53.2 ± 5.7, p = 0.001) and poorer health-related quality of life (SF-36: 52.6 ± 14.4 vs 64.2 ± 14.5, p = 0.002) than rural patients, despite no significant differences in objective disease scores (Lund-Kennedy and Lund-Mackay).[20] (Table 3). This pattern is

in line with large-scale international studies from Europe and China, where urban living—associated with higher levels of particulate pollution and allergen exposure—has been shown to exacerbate symptom reporting, even in the absence of greater mucosal disease.[24,25] The absence of a significant urban–rural difference in endoscopic or radiological scores in our sample further supports previous findings that subjective and objective disease severity may be disconnected, likely due to factors such as healthcare access, cultural context, and stress.[26]

Poor adherence to medical therapy emerged as an independent risk factor for CRS (Table 4), a finding consistent with guidelines recommending sustained use of intranasal corticosteroids and regular follow-up.[27] Non-adherence is widely recognized as a contributor to uncontrolled symptoms and increased morbidity in CRS, and our data reinforce the need for targeted patient education and support programs, as emphasized by recent interventional studies.[24,28]

Our use of effect sizes and confidence intervals ensures transparent and clinically meaningful interpretation. For example, the difference in RSDI scores between cases and controls was large (Cohen's d = 1.18), underscoring the real-world impact of CRS on patient wellbeing (Table 2). We did not find statistically significant differences in SNOT-22 or objective disease scores aligning with prior studies that emphasize the variability and multidimensional nature of CRS.[29] The robust association between allergic comorbidity, adherence, and patient-reported outcomes, but not structural findings, suggests that symptom management in CRS should be multidimensional and patient-centered.[30]

Our study's strengths include its prospective matched design, the use of validated clinical and patient-reported outcome measures, and careful adjustment for confounders. However, the relatively small sample size limits the precision of some estimates, and our results may not be fully generalizable to other regions or settings. In addition, reliance on self-reported adherence and environmental exposure may introduce some reporting bias, though efforts were made to corroborate these with objective data when possible.[31]

Overall, these findings reinforce the importance of integrating allergy management and adherence support into routine care for CRS patients in Pakistan and similar environments.[32] Urban patients may benefit from interventions aimed at reducing symptom burden through improved environmental control, patient education, and routine allergy screening.[33] Future multicenter research should focus on larger populations, incorporate objective measurement of environmental exposures, and explore the role of psychosocial and healthcare access factors in shaping patient outcomes. Our results add to the growing evidence that CRS is a complex, multifactorial disease in which allergic comorbidity and adherence, rather than objective disease markers alone, drive much of the clinical burden.[34]

# Conclusion

This study shows the importance of allergic comorbidity and treatment adherence in chronic rhinosinusitis in Lahore, Pakistan. While, environmental exposures and urban living are complex co-variants, it is allergy and adherence that have the strongest effect on patient outcomes. We found that patient-reported symptoms often do not match objective clinical assessments, especially in urban areas. This shows the need for a multidimensional approach to diagnosis and management. Integrating allergy screening, environmental risk assessment, and adherence support into routine care can improve outcomes. Future research should use larger, multicentre studies and focus on patient-centred results.

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