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Correlation between nasal polyps and serum creatinine – A preliminary observational study

Neehal Zuturu[1], Deviprasad Dosemane[1], Suraj Pai[2] & Meera Niranjan Khadilkar[1]*

[1]Department of Otorhinolaryngology – Head and Neck; & [2]Department of Cardiovascular and Thoracic Surgery, Kasturba Medical College Mangalore, Manipal Academy of Higher Education, Manipal-576 104, Karnataka, India

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Chronic rhinosinusitis (CRS) presents with prolonged sinonasal inflammation and may occur with (CRSwNP) or without (CRSsNP) nasal polyps. While creatinine changes have been studied in gastrointestinal polyps, their relation to nasal polyps remains unexplored. This study was undertaken to evaluate whether serum creatinine correlates with presence or severity of nasal polyps using Lund Mackay (LM) computed tomography (CT) score and Lund Kennedy (LK) endoscopy score in CRS patients. A retrospective observational study was conducted on adults diagnosed with chronic rhinosinusitis with and without nasal polyps, who underwent Functional Endoscopic Sinus Surgery (FESS). Clinical history and examination findings, pre-operative diagnostic nasal endoscopy and radiological findings, and intra-operative records were studied in 114 subjects. Median creatinine varied significantly ($P< 0.001$). In CRSsNP, LK DNE score showed a moderate negative correlation with creatinine ($p = 0.010$), but no correlation was found in CRSwNP. LM CT score did not correlate with creatinine in either group. Serum creatinine had poor predictive ability for polyps (AUC = 0.54, $p = 0.471$); at a ≥0.8 mg/dL cutoff, sensitivity was 72% and specificity 40%. No significant correlation could be observed between nasal polyps and serum creatinine levels and hence, creatinine cannot be reliably used to predict severity of nasal polyps.

Keywords: Kidney function tests, Nasal polyps, Paranasal sinus diseases, Rhinosinusitis

Chronic rhinosinusitis (CRS) refers to persistent inflammation of the nasal and paranasal sinus lining that continues for more than twelve weeks[1]. It is broadly classified into two forms: CRS with nasal polyps (CRSwNP) and CRS without nasal polyps (CRSsNP). Nasal polyps are described as benign, inflammatory outgrowths of sinonasal mucosa arising because of longstanding mucosal oedema[2]. As per cadaveric studies, nasal polyps occur in 5.5–20% cases, while symptomatic CRSwNP affects 2–3% of the population[3]. The severity of disease is usually evaluated using Lund-Mackay (LM) and Lund-Kennedy (LK) scores via imaging and diagnostic nasal endoscopy.

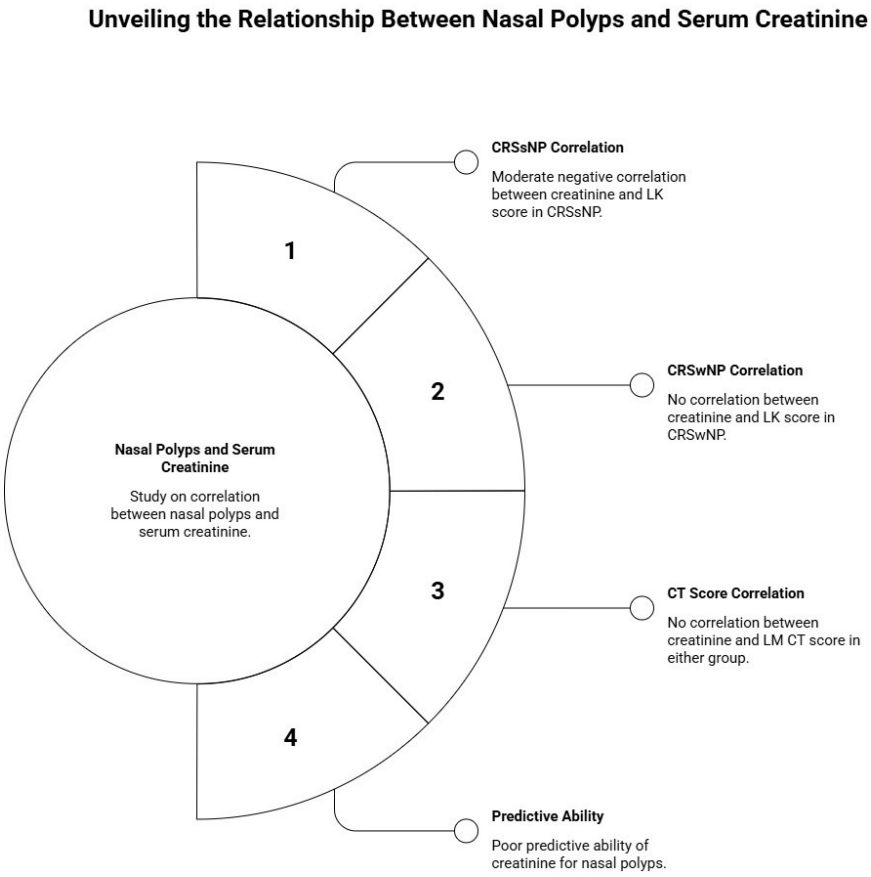
Recent research has shifted focus to the systemic manifestations of allergy and chronic inflammation, including prospective renal effects. Although colonic and colorectal polyps have shown some impact on serum creatinine, the relationship between nasal polyps and creatinine remains underexplored[4,5].

*Correspondence:
E-mail: meera.khadilkar@manipal.edu

This study aims to examine the correlation between serum creatinine and nasal polyps by comparing LM and LK scores with creatinine levels in patients with CRSsNP and CRSwNP, evaluating its utility as a marker of polyp severity.

Materials and Methods

This was a retrospective observational quantitative study, which included all patients aged 18 and above, with a diagnosis of chronic rhinosinusitis, both with and without nasal polyps, who underwent Functional Endoscopic Sinus Surgery (FESS). The study took place at a tertiary care centre in southern India. We collected data from patients who underwent treatment between January 2018 and September 2023. Ethical clearance was procured from the Institutional Ethics Committee. Inclusion criteria were adults (≥18 years) with chronic rhinosinusitis undergoing FESS. Exclusion criteria were patients with fungal sinusitis, paranasal sinus malignancy, pre-existing renal disease or muscular dystrophy, those who received contrast dyes for imaging, and patients taking nephrotoxic or creatinine-altering drugs (*e.g.*,



Graphical abstract

aminoglycosides, amphotericin B, cyclosporine, cimetidine, trimethoprim, pyrimethamine, phenacemide, dronedarone, probenecid, salicylates, antiretrovirals, selected chemotherapeutics, and tyrosine kinase inhibitors). All eligible patients meeting inclusion criteria within the study period were included through comprehensive institutional sampling. Clinical history, examination findings, diagnostic nasal endoscopy, radiology, and operative records were reviewed. LM CT and LK endoscopy scores were compared with serum creatinine levels[6,7]. Data analysis was completed using IBM SPSS for Windows, Version 25.0 (Armonk, NY: IBM Corp).

Results

There were 114 subjects in the present study. Ninety-nine patients underwent septoplasty and FESS combined, 14 underwent only FESS and 1 subject had undergone septoplasty, FESS, and tonsillectomy.

Epidemiology

With an average age of 35.52±11.39 years, the participants' ages varied from 18 to 65 years old (Table 1). Around 40% belonged to the 18 to 30 age range, and fewer than 3% were older than 60. The CRSwNP group had a greater median age (37 years) than the CRSsNP group (31 years), indicating a significant age difference ($p = 0.046$) between the two groups. The gender ratio of our participants was 2:1. About 59% of the participants had polyps (CRSwNP) while 41% of the participants did not (CRSsNP).

Clinical history

The most frequent symptoms were nasal obstruction (94.7%), followed by headache (66.7%), excessive sneezing (43%), rhinorrhoea (42.1%) and reduced sense of smell (35.1%). The distribution of facial pain ($p = <0.001$, moderate association) and hyposmia ($p = <0.001$, moderate association) differed significantly between the two groups. One-third of our subjects gave a history of allergy. Only 14.0% of the participants gave a history of comorbidities. The median duration of symptoms in months was 12.

Serum creatinine levels

Serum creatinine levels in the present study ranged between 0.53-1.36 mg/dL. Average serum creatinine

Table 1 — Demographic data

Parameters	CRSwNP (n = 67)	CRSsNP (n = 47)	p-value
Age (years)***	37.30 ± 11.43	32.98 ± 10.94	0.046[1]
Age***			0.035[2]
18-30 Years	23 (34.3%)	23 (48.9%)	
31-40 Years	18 (26.9%)	15 (31.9%)	
41-50 Years	17 (25.4%)	3 (6.4%)	
51-60 Years	6 (9.0%)	6 (12.8%)	
61-70 Years	3 (4.5%)	0 (0.0%)	
Gender			0.893[3]
Male	45 (67.2%)	31 (66.0%)	
Female	22 (32.8%)	16 (34.0%)	
Clinical features			
Nasal obstruction	65 (97.0%)	43 (91.5%)	0.228[2]
Headache	42 (62.7%)	34 (72.3%)	0.282[3]
Facial pain***	8 (11.9%)	20 (42.6%)	<0.001[3]
Reduced smell***	33 (49.3%)	7 (14.9%)	<0.001[3]
Postnasal drip	10 (14.9%)	12 (25.5%)	0.158[3]
Epistaxis	9 (13.4%)	5 (10.6%)	0.655[3]
Rhinorrhoea	30 (44.8%)	18 (38.3%)	0.490[3]
Excessive sneezing	32 (47.8%)	17 (36.2%)	0.218[3]
Snoring	9 (13.4%)	5 (10.6%)	0.655[3]
Duration of symptoms (months)	29.27 ± 53.57	25.96 ± 39.03	0.814[1]
History of allergy	27 (40.3%)	11 (23.4%)	0.060[3]
Comorbidities	12 (17.9%)	4 (8.5%)	0.155[3]
Serum creatinine (mg/dL)	0.89 ± 0.19	0.86 ± 0.19	0.471[1]
LM DNE Score***	3.52 ± 2.00	2.30 ± 1.44	0.002[1]
LK CT Score***	11.27 ± 6.83	6.83 ± 4.42	<0.001[1]
Surgery performed***			<0.001[2]
Septoplasty + FESS	53 (79.1%)	46 (97.9%)	
FESS	14 (20.9%)	0 (0.0%)	
Septoplasty + FESS + tonsillectomy	0 (0.0%)	1 (2.1%)	

***Significant at p<0.05, 1 Wilcoxon-Mann-Whitney U Test, 2 Fisher's Exact Test, 3 Chi-Square Test, CRSwNP chronic rhinosinusitis with nasal polyps, CRSsNP chronic rhinosinusitis without nasal polyps, LM Lund Mackay, DNE Diagnostic Nasal Endoscopy, LK Lund Kennedy, CT computed tomography, FESS functional endoscopic sinus surgery

was 0.88±0.19 mg/dL; median serum creatinine varied significantly (p = <0.001) *i.e*. 0.95 mg/dL in men and 0.7 mg/dL in women.

Lund Mackay and Lund Kennedy scores

LM CT score showed a significant difference (W = 2186.500, p = <0.001) between the two groups, median LM CT score in CRSwNP being greater (Table 2).

In a similar vein, the LM surgery score showed significant difference between the groups (W = 2317.000, p = <0.001), median LM surgery score in CSwNP being greater.

A statistically significant moderate negative correlation (rho = -0.37, p = 0.010) was noted between LK DNE Score and serum creatinine (mg/dL) in the CRSsNP group. For every 1 unit increase in LK Score, serum creatinine decreased by 0.05 mg/dL. Conversely, for every mg/dL increase in serum creatinine (mg/dL), LK Score decreased by 2.64. However, there was no statistically significant correlation between LK Score and serum creatinine (mg/dL) in CRSwNP group (rho = -0.09, p = 0.478). LM score and serum creatinine (mg/dL) did not significantly correlate in either CRSwNP group (r = 0.08, p = 0.536) or CRSsNP group (rho = -0.24, p = 0.103) (Fig. 1).

Serum creatinine (mg/dL) had an area under ROC curve of 0.54 in predicting the presence of polyps. There was no statistically significant difference (p = 0.471). At a cutoff of serum creatinine (mg/dL) ≥0.8, it predicts the presence of polyps with a sensitivity of 72% and a specificity of 40% (Table 3, and Fig. 2).

Table 2 — Distribution of LM DNE score and LK CT score and correlation with serum creatinine levels

Scoring	CRSwNP	CRSsNP	Wilcoxon-Mann Whitney U Test	p-value
LM DNE Score				
Mean (SD)	3.52 (2.00)	2.30 (1.44)	2114.500	0.002
Median (IQR)	3 (2-5)	2 (1.5-4)		
Min – Max	0 – 8	0 – 5		
LK CT Score				
Mean (SD)	11.27 (6.83)	6.83 (4.42)	2186.500	<0.001
Median (IQR)	10 (6-16)	6 (3-9.5)		
Min – Max	1 – 24	1 – 20		
Correlation with serum creatinine	Spearman Correlation Coefficient		p-value	
CRSwNP				
LK DNE score	-0.09		0.478	
LM CT score	0.08		0.536	
CRSsNP				
LK DNE score	-0.37		0.010	
LM CT score	-0.24		0.103	

SD standard deviation, IQR interquartile range, LK Lund Kennedy, DNE Diagnostic Nasal Endoscopy, LM Lund Mackay, CT computed tomography, CRSwNP chronic rhinosinusitis with nasal polyps, CRSsNP chronic rhinosinusitis without nasal polyps

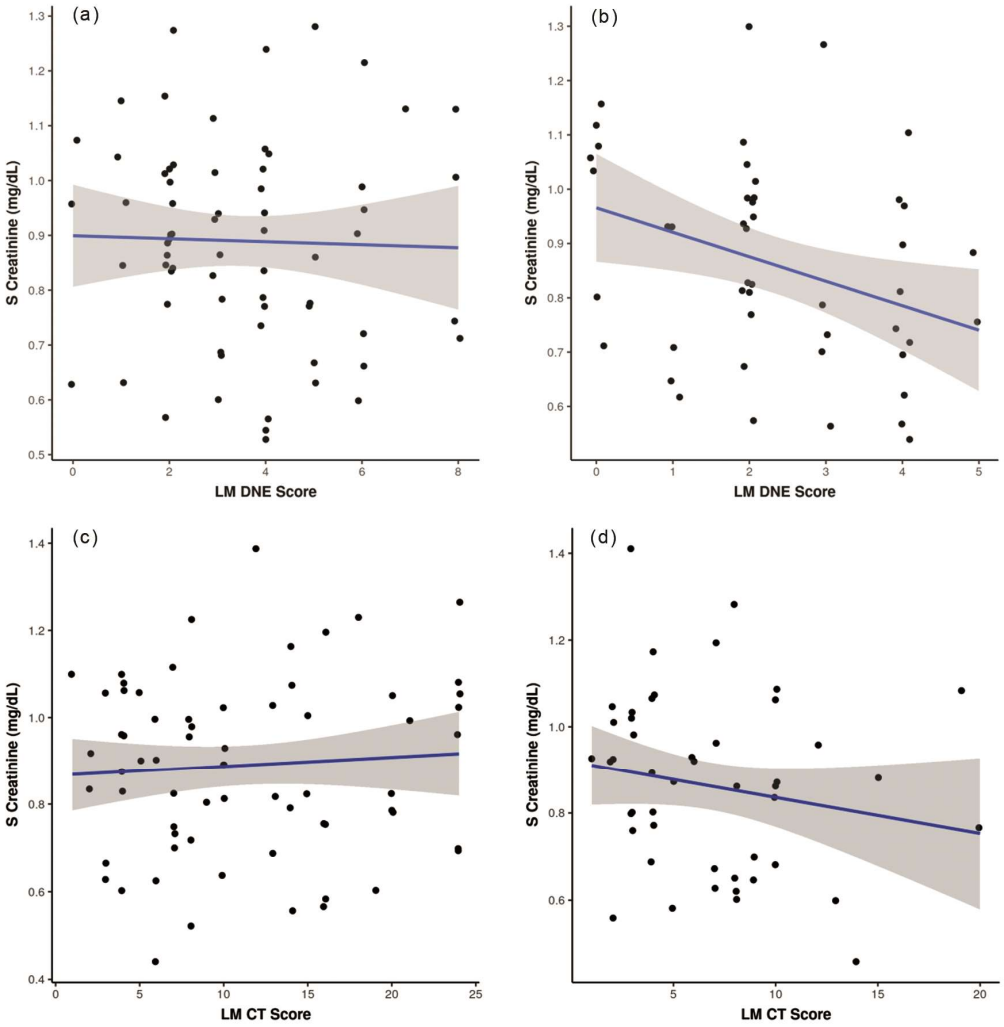


Fig. 1 — Distribution of LM CT Score and LK DNE Score with serum creatinine levels

Table 3 — Serum creatinine as a diagnostic test

Parameter	Value (95% CI)
Cutoff by ROC	≥ 0.8 (p = 0.471)
AUROC	0.54 (0.431 - 0.649)
Sensitivity	71.6% (59-82)
Specificity	40.4% (26-56)
Positive Predictive Value	63.2% (51-74)
Negative Predictive Value	50.0% (33-67)
Diagnostic Accuracy	58.8% (49-68)
Positive Likelihood Ratio	1.2 (0.91-1.59)
Negative Likelihood Ratio	0.7 (0.42-1.17)
Yuden Index	12.1
Diagnostic Odds Ratio	1.71 (0.78-3.77) (p = 0.178)

CI confidence interval, AUROC area under ROC curv

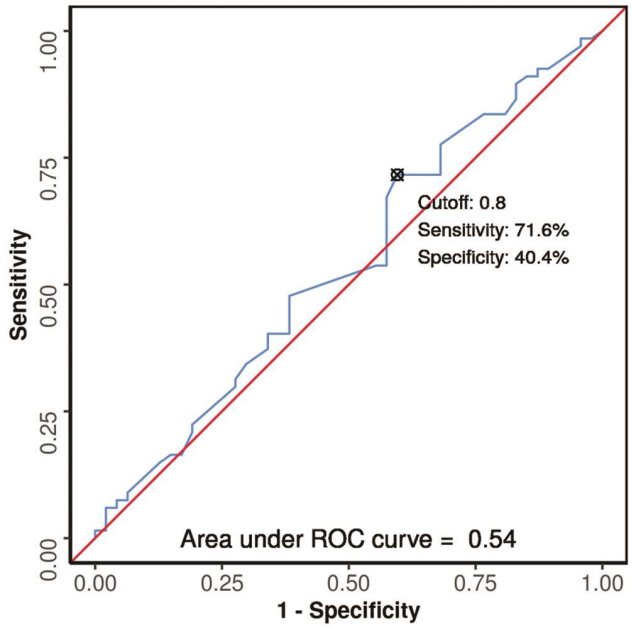


Fig. 2 — Serum creatinine as a diagnostic test

Discussion

The relationship between chronic kidney disease (CKD) and CRS has become an area of growing research interest. Epidemiological evidence suggests a possible systemic link, as proved in a longitudinal cohort study where CKD patients displayed a significantly greater risk of developing CRS (adjusted hazard ratio 1.28), sparking debate on common pathways of pathogenesis and highlighting the need to assess the systemic manifestations of localized airway disease[8]. Adegbiji *et al*. noted that many people living with chronic kidney disease reported troublesome nasal symptoms, particularly a feeling of nasal blockage and excessive nasal secretions These changes were thought to result from uremic deposits in the nasal mucosa, which may lead to irritation, oedema, and subsequently, rhinosinusitis. In their study, rhinosinusitis was detected in 30% of CKD patients, and nasal polyps were present in 1.9% of cases[9]. These findings suggest that impaired renal function may cause sinonasal inflammation; this indicates that there may be a possible link between kidney disease and upper airway pathology.

Work on systemic biomarkers has raised the question of whether mucosal polyps in different organs might have some link with kidney function. Yang *et al*. found that patients with colorectal polyps had higher serum creatinine (p=0.002), and Chao *et al*. reported a similar rise in creatinine among those with non-adenomatous colorectal polyps compared with controls ($P< 0.05$)[4,5]. In otolaryngology, Kronbichler and colleagues reported that a small proportion of patients with nasal polyps, around 5%, also had a primary renal disorder, and this association remained even after adjustment[10]. Taken together, these reports suggest that polyp disease in the colon and the sinonasal tract may sometimes be accompanied by mild changes in basic laboratory markers such as creatinine. It is still unclear whether this reflects a true biological link, shared risk factors, or just the downstream effect of long-standing inflammation.

Considering these prior reports, we examined whether serum creatinine—a standard measure of kidney function—correlates with nasal polyp presence or severity in CRS patients. We looked specifically at creatinine levels against Lund-Mackay CT scores and Lund-Kennedy endoscopic grades. In our cohort, creatinine showed no link to either polyps or overall disease burden. This differs from Sinha *et al*., who found higher creatinine in sinonasal polyposis patients (0.94 mg/dL) versus controls (0.80 mg/dL; p=0.002), with an adjusted odds ratio of 3.36 (95% CI: 1.42–7.97; p=0.006)[11]. Methodological differences, patient profiles, or sample sizes likely account for the discrepancy between studies. Biologically, our negative findings fit with nasal polyposis being mainly a local type-2 inflammation issue. Cytokines like IL-4, IL-5, and IL-13 fuel tissue remodelling and polyp growth right at the site, without much systemic spillover to affect renal markers like creatinine[1,3].

Serum creatinine is also a poor indicator of broader systemic or inflammatory activity. Although it is a reliable and widely accepted measure of kidney function, it lacks the sensitivity to pick up the subtle

physiological changes seen in disorders like CRS, where the inflammatory process is largely restricted to the sinonasal mucosa. Previous studies have similarly noted that creatinine is not a reliable marker when there is no overt kidney disease[12,13].

Recently, CRS research has focused on markers that more accurately reflect the underlying disease. Indicators such as serum IgE, tissue eosinophil counts, and local cytokine profiles have been found to correlate more closely with specific CRS endotypes and treatment outcomes[1]. In addition, systemic markers like C-reactive protein (CRP), the eosinophil-to- lymphocyte ratio (ELR), and the neutrophil-to-lymphocyte ratio (NLR) have been studied for their potential to indicate disease severity and risk of recurrence. Even so, these routinely available markers often fall short in clearly distinguishing between CRS subtypes or in accurately representing inflammation that is largely confined to the sinonasal mucosa[8].

The lack of a clear association in our study may partly reflect how the cohort was selected. Because we excluded patients with established renal disease or conditions known to influence creatinine levels, the study group became quite uniform, leaving little variation in creatinine values to examine. While this strengthened internal validity, it may also have reduced the chance of detecting small but meaningful differences that might appear in a more diverse or higher-risk population. Differences in study design, patient characteristics, and exclusion criteria across published work also help explain why previous studies have reported inconsistent findings regarding the usefulness of serum creatinine as a biomarker in CRS.

In our study, we assessed whether serum creatinine could help distinguish different CRS phenotypes, using a cut-off value of ≥0.8 mg/dL. In our study, the receiver operating characteristic (ROC) curve for serum creatinine gave an area under the curve (AUC) of 0.54, which is only marginally better than chance. Sensitivity was acceptable at 71.6%, but specificity was low at 40.4%, resulting in many false positives. Taken together, the likelihood ratios and diagnostic odds ratio were close to 1 and did not reach statistical significance, indicating that creatinine had no real diagnostic value for CRS or nasal polyposis in this cohort. This contrasts with the findings of Sinha *et al*., who reported higher creatinine levels in patients with sinonasal polyposis and described creatinine as an independent predictor with good discrimination (AUC 0.803)[11]. Differences in the study populations, inclusion criteria, or statistical models could explain why the two sets of results do not agree. When placed alongside the wider literature, our data are more in line with the view that creatinine is primarily a test of kidney function and does not reliably mirror inflammatory activity in CRS[12,13]. Larger, methodologically consistent studies will be needed before creatinine can be considered a useful biomarker in this setting.

Our study has certain limitations. Because of its retrospective design, we had little control over confounding factors and cannot draw any causal conclusions. We also included only patients who had FESS, which may have skewed the sample toward those with worse or more hard-to-treat CRS; patients managed medically alone were excluded. The number of patients was relatively small, partly due to fewer surgeries during the COVID-19 pandemic. Finally, we looked at serum creatinine alone and did not measure other renal markers or markers of systemic inflammation.

Future work should address these gaps with large, multicentre prospective studies across different patient groups. Longer follow-up periods—measuring kidney function, inflammatory markers, and changes in polyp size over time—might pick up mild systemic effects that short-term studies miss. Testing a wider array of biomarkers beyond serum creatinine, including local tissue markers and systemic inflammatory and metabolic measures, would give a clearer picture of how CRS affects the body overall. It would also be worth exploring shared biological pathways, such as chronic inflammation, immune dysregulation, or oxidative stress, since identifying these could point to new diagnostic targets or treatment options. In the end, this work could help guide more targeted, individualised treatment based on biomarkers and help reduce unnecessary systemic testing.

Conclusion

We found no evidence that serum creatinine can reliably predict whether someone has nasal polyps or how severe their chronic rhinosinusitis is, even though some earlier studies hinted at a link between kidney function and polyps in other parts of the body. Our results fit with the idea that nasal polyposis is mainly driven by local inflammation in the nose and sinuses. To clarify whether there really are any systemic effects, future studies need to focus on

finding biomarkers that are both sensitive and specific for rhinosinusitis. Better biomarkers could help determine if systemic involvement occurs, guide treatment choices, and ultimately improve outcomes for patients with chronic rhinosinusitis and nasal polyps.

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Conflict of interest

All authors declare no conflict of interest.

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