e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(7); 1349-1355

Original Research Article

Etiological Factors Associated with Chronic Maxillary Sinusitis: A Cross-Sectional Observational Study at a Tertiary Care Centre in Eastern India

Ritu[1], Rajesh Kumar Choudhary[2], Piyush Kumar[3], Renu Kumari[4]

[1]Junior Resident Academic (JRA-3), Department of Otorhinolaryngology (ENT), Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand, India

[2]Additional Professor, Department of Otorhinolaryngology (ENT), Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand, India

[3]Junior Resident Academic (JRA-3), Department of Otorhinolaryngology (ENT), Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand, India

[4]Junior Resident Academic (JRA-3), Department of Otorhinolaryngology (ENT), Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand, India

Received: 06-04-2026 / Revised: 22-05-2026 / Accepted: 25-06-2026

Corresponding Author: Dr. Ritu

Conflict of interest: Nil

Abstract

Background: Chronic maxillary sinusitis (CMS) is a multifactorial inflammatory disorder with significant quality-of-life burden affecting 146/1000 population in India.

Aim: To evaluate prevalence of etiological factors causing CMS in Eastern Indian population.

Methods: Cross-sectional observational study conducted at ENT OPD, RIMS, Ranchi over one year (n=127, age >17 years, symptoms >12 weeks). Consecutive sampling applied. Clinical history, SNOT-22, anterior/posterior rhinoscopy, diagnostic nasal endoscopy (Lund-Kennedy score), X-ray PNS and CT PNS (256-slice) were recorded. Bacteriology and socioeconomic assessment (Kuppuswamy) done. Analysis in Jamovi v2.6.19.

Results: Mean age 32.1±13.7 years, peak 21-30 years (41.7%), male predominance 61.4% (p=0.013). Nasal blockage (88.98%) and anterior discharge (81.89%) were commonest symptoms. Mean SNOT-22 23.5±19.9; 59.1% mild disease. Deviated nasal septum (41.7%) was commonest anatomical variation; 66.9% had multiple variations (p=0.027 association with severity). Allergy (56.7%), odontogenic causes (33.9%), environmental exposure (25.2%) and lower socioeconomic status (37.3% lower class) were major contributors.

Conclusion: CMS is multifactorial with anatomical, allergic, odontogenic and socioeconomic interplay requiring multidisciplinary evaluation and early intervention.

Keywords: Chronic maxillary sinusitis deviated nasal septum, SNOT-22, Lund-Kennedy, odontogenic sinusitis, anatomical variations, allergic rhinitis, socioeconomic status.

DOI: 10.25258/ijcpr.18.7.203

Introduction

Chronic maxillary sinusitis (CMS) is the most common healthcare complaint affecting 1 in 8 Indians, prevalence 146/1000 [1]. Defined as inflammation of maxillary sinus mucosa persisting >12 weeks with nasal blockage/discharge, facial pain/pressure and/or olfactory dysfunction plus endoscopic/CT evidence [2]. Maxillary sinus, first to develop (day 65-75 gestation) and largest paranasal sinus, is highly susceptible due to proximity to dentition and narrow ostium draining via osteomeatal complex (OMC) [3].

Pathophysiology involves OMC obstruction leading to hypoxia, mucociliary dysfunction, secretion stasis, biofilm formation and chronic inflammation [3,5,18].

Etiology is multifactorial: anatomical variations (septal deviation, concha bullosa, Haller cells) [4,15], bacteriology (S. aureus, anaerobes) [5,8], occupational/environmental pollutants [6,7,11], odontogenic [8,17], allergy [9,16], socioeconomic/lifestyle [10,14] and geographical factors [11]. This study evaluates etiological spectrum in Indian population to guide medical/surgical management.

Materials and Methods

Study Design: Cross-sectional observational study [2].

Study Settings: Department of ENT, Rajendra Institute of Medical Sciences (RIMS), Ranchi, Jharkhand – tertiary care teaching hospital serving urban and rural Eastern India. Duration: 1 year after IEC approval (Memo No. 03 IEC, RIMS, Dated 03.01.25).

Study Population: Clinically diagnosed CMS patients attending ENT OPD on consecutive sampling [1].

Sample Size: The sample size was calculated using the formula, $n= [Z^2 \times p(1-p)]/E^2$

where p = 0.091, Z = 1.96 (95% confidence level), and E = 0.05 (absolute precision). The calculated sample size was 127.1, which was rounded to 127 participants.

Inclusion Criteria

- Age >17 years
- Symptoms suggestive of CMS >12 weeks [2].

Exclusion Criteria

Patients aged <17 years; duration of symptoms <12 weeks; systemic debilitating diseases (e.g., malignancy, liver cirrhosis, chronic kidney disease, or significant cardiac disease); immunocompromised status; previous sinonasal surgery; pregnancy; history of nasal trauma or facial fractures; involvement of paranasal sinuses other than the maxillary sinus; and patients with nasal masses or granulomatous diseases. [2]

Methodology

Detailed history (occupational, environmental, dental, allergy, lifestyle, sleep, Kuppuswamy SES, geography). SNOT-22 questionnaire (22 items 0-5; Mild <20, Moderate 21-50, Severe >50 per Toma & Hopkins) [12]. Examination: anterior/posterior rhinoscopy, diagnostic nasal endoscopy (4mm Karl Storz 0°/30°) after 4% lignocaine+adrenaline decongestion.

Lund-Kennedy score (polyps, discharge, edema 0-2/side, total 0-12) [13]. Imaging: CT nose-PNS (Siemens Dual Source 256-slice) for variations [15]. Bacteriology where applicable [5].

Statistical Analysis

Excel + Jamovi v2.6.19; mean±SD, frequency (%), t-test, Chi-square/Fisher's exact, $p<0.05$ significant [2].

Results

Table 1: Age Distribution (Mean 32.1±13.7, Median 27, Range 18-72) [2]

Age	N	%
≤20	22	17.3
21-30	53	41.7
31-40	23	18.1
41-50	13	10.2
51-60	9	7.1
>60	7	5.5

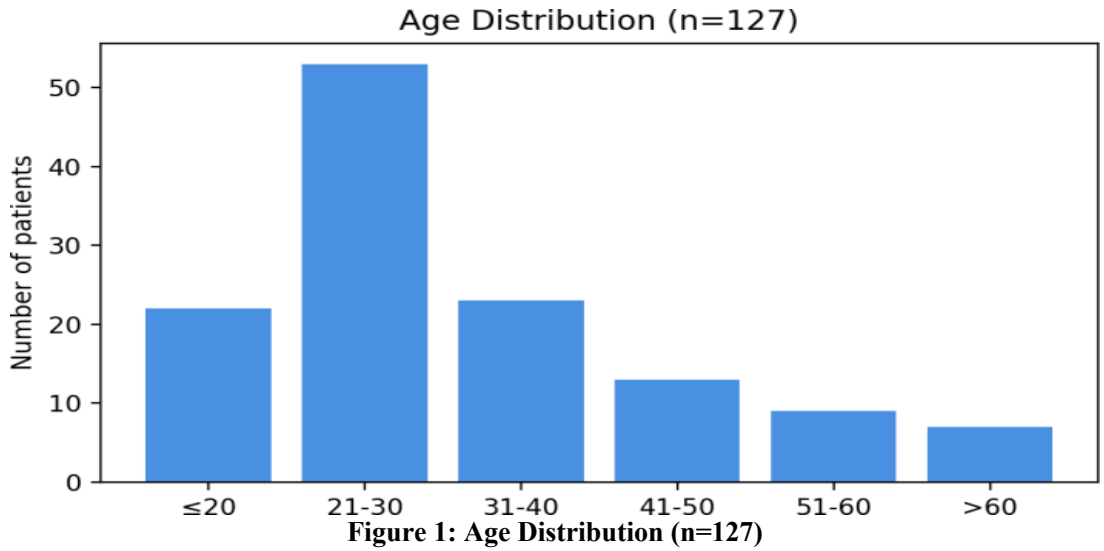

Figure 1: Age Distribution (n=127)

Table 2: Gender – Male predominance p=0.013 [6]

Gender	Count	%
Male	78	61.4
Female	49	38.6

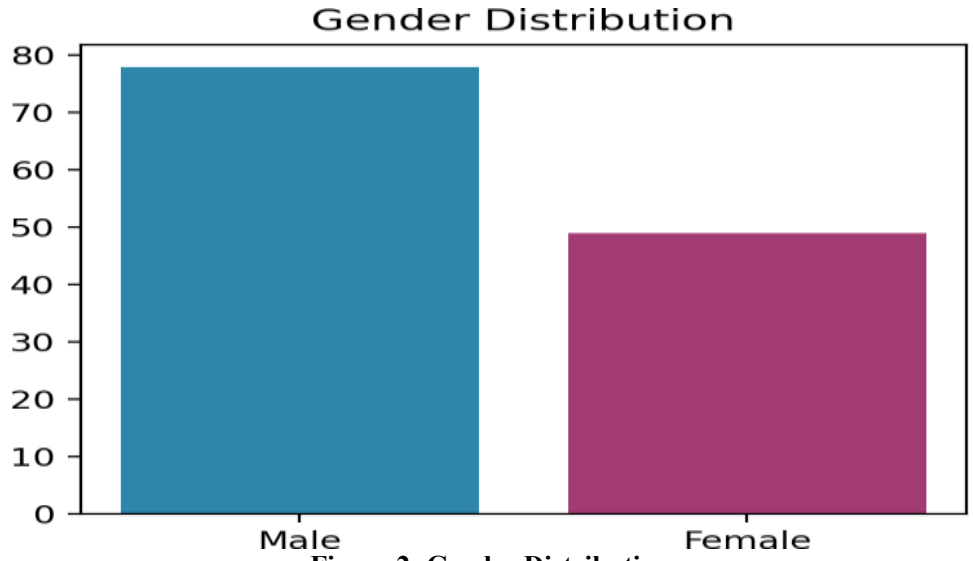

Figure 2: Gender Distribution

Table 3: Presenting Symptoms [3]

Symptom	N	%
Nasal blockage	113	88.98
Ant discharge	104	81.89
Postnasal drip	83	65.35
Headache	69	54.33
Facial pain	62	48.82
Sneezing	42	33.07
Smell loss	10	7.87
Bleed	5	3.94

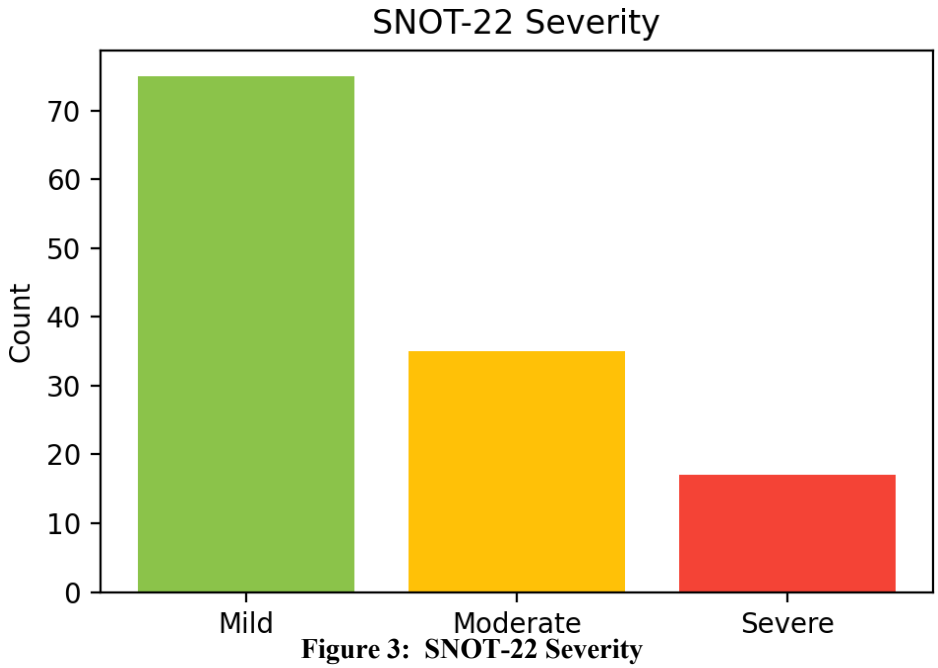

Figure 3: SNOT-22 Severity

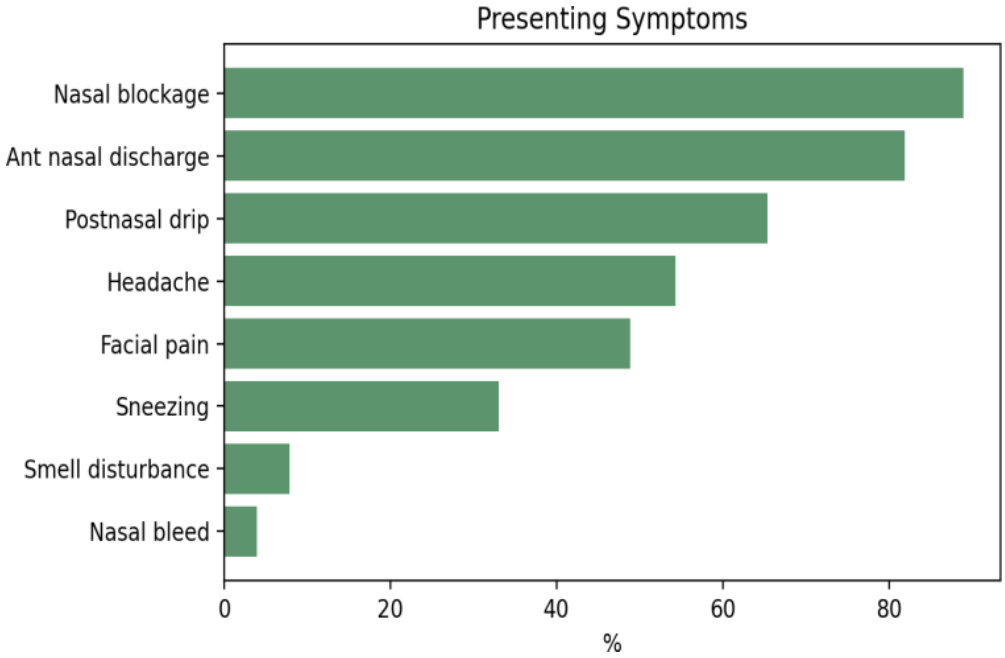

Figure 4: Presenting Symptoms

Table 4: SNOT-22 Severity Mean 23.5±19.9 [12]

Severity	N	%
Mild <20	75	59.1
Moderate 21-50	35	27.6
Severe >50	17	13.4

Table 5: Anatomical Variations on CT/DNE [4,15]

Variation	R	L	Bi	Total
DNS	26	22	5	53 (41.7%)
Agger nasi	10	8	2	20
Concha bullosa	8	5	2	15
Bulla ethmoidalis	5	3	1	9
Onodi	2	1	1	4
Haller	1	1	0	2

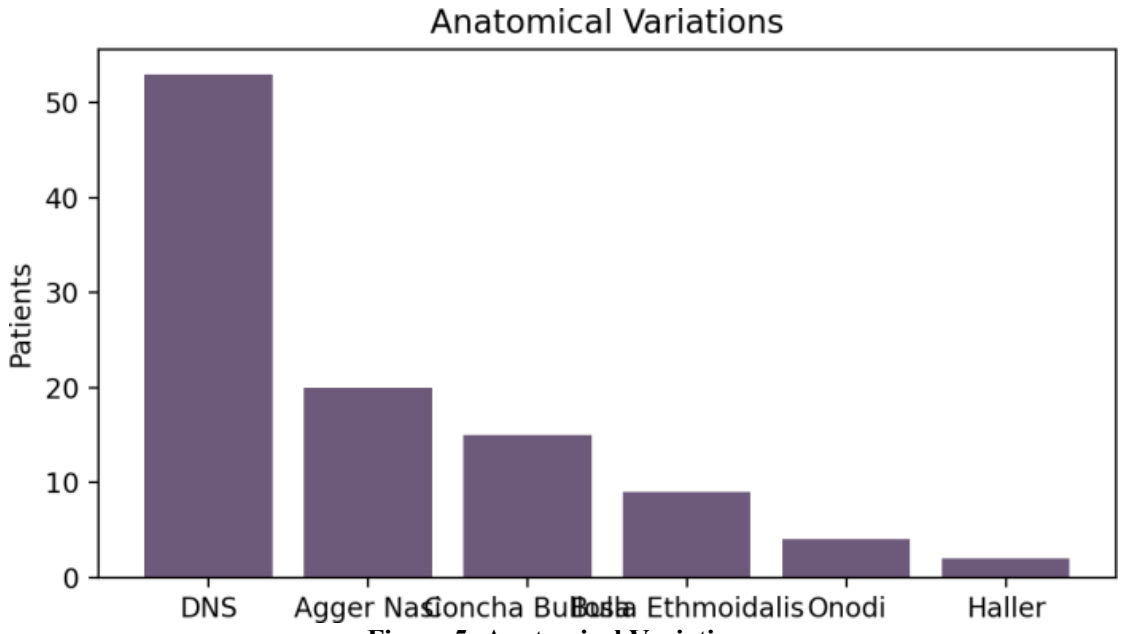

Figure 5: Anatomical Variations

Table 6: Number & Lateralization

Category	N	%
No variation	24	18.9
Single	18	14.2
>1 variations	85	66.9
Unilateral	31	24.4
Bilateral	72	56.7

Table 7: Lund-Kennedy Scores [13]

Finding	0 R/L	1 R/L	2 R/L	Positive R/L
Polyps	80/80	27/29	20/18	37.1/37.1%
Discharge	52/60	63/61	12/6	59.1/52.8%
Edema	53/62	68/61	6/4	58.3/51.2%

Table 8: Etiological Factors [8,9,11]

Factor	Yes	No
Allergy	72 (56.7%)	55 (43.3%)
Odontogenic	43 (33.9%)	84 (66.1%)
Environmental	32 (25.2%)	95 (74.8%)
Sleep disorder	40 (31.5%)	87 (68.5%)
Autoimmune	6 (4.7%)	121 (95.3%)
Rural/Urban	58 (45.7%)	69 (54.3%) urb

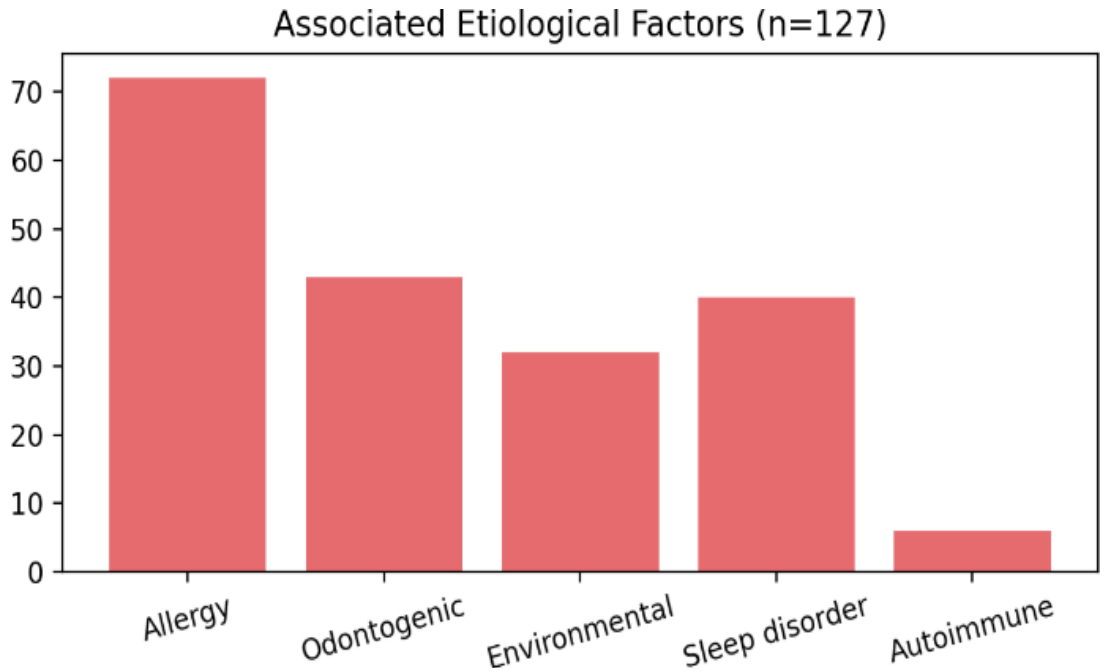

Figure 6: Associate Etiological Factors (n=127)

Table 9: Socioeconomic Status (Kuppuswamy) [10,14]

Class	N	%	P
Lower	47	37.3	0.006*
Lower-middle	45	35.7	0.002*
Upper-lower	17	13.5	<0.001*
Upper-middle	17	13.5	<0.001*

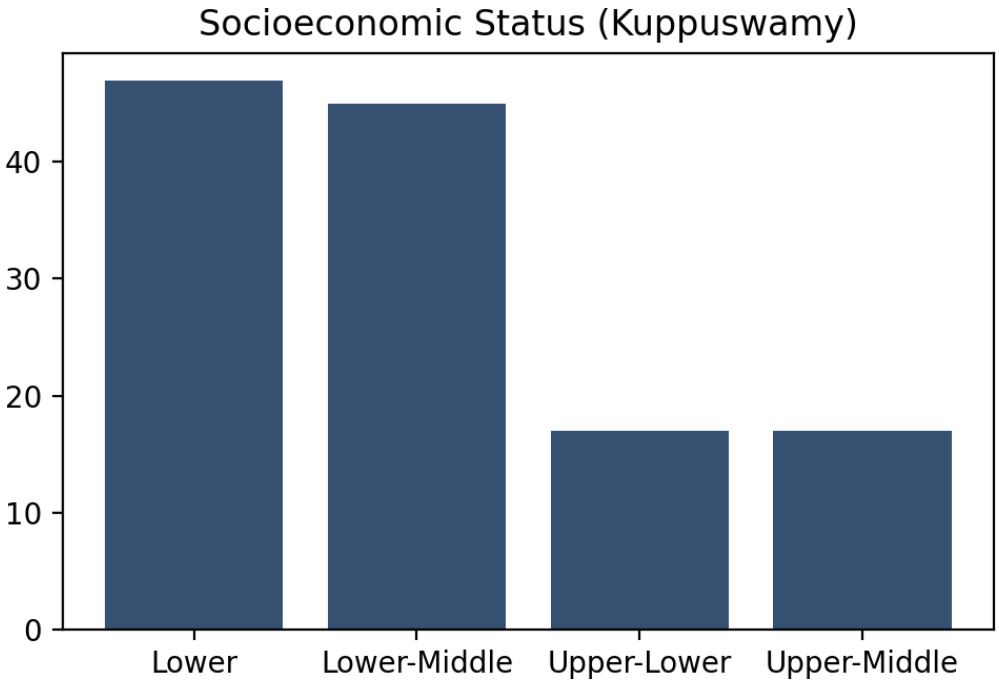

Figure 7: Socioeconomic Status (Kuppuswamy)

Table 10: Variation vs Severity – Fisher p=0.027* [4]

Severity	No var	Var present	Total
Mild	19	56	75
Moderate	5	30	35
Severe	0	17	17

Table 11: Bacteriology n=16 [5,8]

Organism	N	%
S. aureus	5	33.3
S. pneumonia	3	20.0
Klebsiella	2	13.3
Pseudomonas	2	13.3
E. coli	1	6.7
Peptostreptococcus	1	6.7
Mixed	1	6.7

Discussion

CMS peaked in 21-30y (41.7%) mean 32.1y, matching EPOS and Indian data showing young adult preponderance due to occupational allergen burden [2,11]. Male predominance (61.4%, p=0.013) mirrors Beule

[11] and Hastan et al. [7] due to outdoor dust/industrial exposure – farmers 12.6%, labourers 10.2% in our cohort [6]. Nasal obstruction 88.98% reflects OMC obstruction-hypoxia-mucociliary failure cycle [3]. SNOT-22 mean

23.5 with 13.4% severe shows heterogeneity needing individualized care [12]. Anatomical variations in 81.1%, multiple in 66.9%, DNS commonest 41.7%, followed by agger nasi and concha bullosa, consistent with Stammberger [4] and Bolger [15]. Significant association with severity (p=0.027) validates structural obstruction as primary driver, supporting routine CT-PNS and DNE [13,15]. Allergy 56.7% supports Settipane [9] and Benninger [16] – IgE-mediated edema obstructs OMC. Odontogenic 33.9% aligns with Brook [8] and Mehra [17] – close molar root proximity, anaerobic flora. Our bacteriology S. aureus 33.3% with mixed pattern supports biofilm chronicity [5,18].

Environmental 25.2% as modifier per Larsen & Tos [6] and Hastan [7]. Lower SES predominance (L 37.3% p=0.006, LM 35.7% p=0.002) reflects Bhattacharyya [10] and Smith [14] – overcrowding, biomass fuel, delayed care. Sleep disorder 31.5% and sedentary 26.8% add

systemic inflammation [10]. Autoimmune rare 4.7% (GPA 2.4%) per Van Crombruggen [19] – uncommon but specific. Rural 45.7% vs urban 54.3% near-equal suggests referral bias and urban pollution impact [7,11]. Overall multifactorial interplay requires holistic multidisciplinary approach [2].

Conclusion

CMS in Eastern India affects young productive males predominantly. Anatomical variations (DNS, agger nasi, concha bullosa) present in >80% and significantly correlate with SNOT-22 severity, representing major etiological factor. Allergy (56.7%) and odontogenic infection (33.9%) are key co-factors; environmental, lifestyle and sleep act as modifiers. Lower socioeconomic status significantly associated, highlighting social determinants. Management requires comprehensive evaluation: early CT/DNE, correction of anatomical/dental factors, allergy control, culture-guided antibiotics targeting S. aureus and anaerobes, and socioeconomic-environmental improvement. Provides region-specific data for prevention and future longitudinal studies.

Limitations

Single-Centre, n=127, exclusion of masses/previous surgery, subjective Lund-Kennedy, no longitudinal follow-up.

References

1. Grover G, et al. Epidemiology of chronic rhinosinusitis in India: prevalence 146/1000. Indian J Otolaryngol Head Neck Surg. 2020;72:34-38.
2. Fokkens WJ, Lund VJ, Hopkins C, et al. European Position Paper on Rhinosinusitis and Nasal Polyps 2020. Rhinology. 2020;58(Suppl S29):1-464.
3. Cohen NA. Sinonasal mucociliary clearance in health and disease. Ann Otol Rhinol Laryngol Suppl. 2006;196:20-26.
4. Stammberger H. Functional endoscopic sinus surgery: The Messerklinger technique. BC Decker; 1991.
5. Sanclement JA, et al. Bacterial biofilms in surgical specimens of CRS. Am J Rhinol. 2005;19(4):351-357.
6. Larsen K, Tos M. Occupational exposure and chronic sinusitis. Acta Otolaryngol. 1991;111:560-565.
7. Hastan D,et al. Chronic rhinosinusitis in Europe—an underestimated disease. Allergy. 2011;66(9):1216-1223.
8. Brook I. Sinusitis of odontogenic origin. Otolaryngol Head Neck Surg. 2006;135(3):349-355.
9. Settipane GA. Epidemiology of nasal polyps and allergy. Allergy Asthma Proc. 1996;17:231-236.
10. Bhattacharyya N. Socioeconomic and comorbidity burden of adult rhinosinusitis. Laryngoscope. 2009;119:1090-1095.
11. Beule A. Epidemiology of chronic rhinosinusitis. Laryngorhinootologie. 2015;94(Suppl1):S176-S200.
12. Toma S, Hopkins C. Stratification of SNOT-22 scores into mild, moderate or severe. Clin Otolaryngol. 2016;41:665-669.
13. Lund VJ, Kennedy DW. Staging for rhinosinusitis. Otolaryngol Head Neck Surg. 1997;117:S35-S40.
14. Smith KA, et al. Socioeconomic status and CRS severity. Int Forum Allergy Rhinol. 2013;3:619-624.
15. Bolger WE, Butzin CA, Parsons DS. Paranasal sinus bony anatomic variations and mucosal abnormalities. Laryngoscope. 1991;101:56-64.
16. Benninger MS, et al. Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, pathophysiology. Otolaryngol Head Neck Surg. 2003;129:S1-S32.
17. Mehra P, Murad H. Maxillary sinus disease of odontogenic origin. Otolaryngol Clin North Am. 2004;37:347-364.
18. Psaltis AJ, et al. The role of bacterial biofilms in chronic rhinosinusitis. Otolaryngol Head Neck Surg. 2007;137:917-921.