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Original Research Article

RISK-FACTOR PROFILE OF ADULTS WITH SEVERE ASTHMA IN A TERTIARY-CARE SETTING: A CROSS-SECTIONAL ANALYSIS

Dhanashree Mestry[1], Trupti Pisal[2], Preeti Gupta[2]

[1]*Junior Resident, Jagjivanram Hospital, Western Railway, Mumbai Central, India*
[2]*Senior DMO, Jagjivanram Hospital, Western Railway, Mumbai Central, India*

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Corresponding Author:
Dr. Preeti Gupta,
Senior DMO, Jagjivanram Hospital, Western Railway, Mumbai Central, India.
Email: drpreeti@gmail.com

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ABSTRACT

Background: Severe asthma imposes a disproportionate burden of morbidity and cost, yet its modifiable risk factors remain incompletely defined

Materials and Methods: We analysed 55 adults meeting Global Initiative for Asthma (GINA) Step 4/5 criteria at Jagjivan Ram Railway Hospital (July 2022 – June 2023). Sociodemographic data, anthropometry, comorbidities, exposures, spirometry, and biomarkers were captured. Associations between risk factors and airflow limitation (FEV_1 < 50 % predicted) were explored with χ^2 and t-tests (IBM SPSS 23).

Results: Mean age was 49.9 ± 16.0 years; 52.7 % were female. Overweight/obesity (BMI ≥ 23 kg m^{-2}) affected 60 % of patients, abdominal obesity 69 % of men and 93 % of women, and neck obesity 62 % and 79 %, respectively. Occupational exposure was present in 27.3 %, passive or active smoke exposure in 5.5 %, and pollutant/allergen exposure in 76.4 %. Hypertension (56 %), diabetes (40 %), and recurrent URTI (51 %) were common. Elevated absolute eosinophil count (AEC > 500 cells μL^{-1}) occurred in 61.8 % and IgE > 300 IU mL^{-1} in 54.5 %. FEV_1 averaged 44.6 ± 9.1 % predicted; lower FEV_1 was associated with BMI ≥ 30 ($p = 0.03$), AEC ≥ 500 ($p = 0.02$) and pollutant exposure ($p = 0.04$).

Conclusion: In this cohort, adiposity, eosinophilia, and environmental exposure were the principal modifiable correlates of severe airflow obstruction. Early identification and mitigation of these factors could prevent progression to refractory disease.

Keywords: Severe Asthma; risk factors; obesity; eosinophilia; environmental exposure.

INTRODUCTION

Asthma affects an estimated 272 million people worldwide, but only 3–10 % of these individuals develop severe disease that is refractory to high-dose inhaled corticosteroids (ICS) and additional controllers.[1] Although numerically few, these patients experience frequent exacerbations, accelerated lung-function decline, and markedly impaired quality of life, while consuming a disproportionate share of healthcare resources.[2] Consequently, prevention of progression to severe asthma and early correction of modifiable risk factors have become headline objectives in global and national respiratory strategies, including the 2024 update of the Global Initiative for Asthma (GINA).[3]

India alone contributes an estimated 34 million cases to the global asthma burden, and its urban megacities—Mumbai prominent among them—are witnessing a steep year-on-year rise driven by rapid motorisation, unregulated construction dust, and dietary westernisation. In such densely populated environments, residents encounter a unique cocktail of risk: ambient PM2.5 concentrations that routinely exceed WHO limits by five- to eight-fold, indoor biomass-smoke exposure from informal eateries, and high pre-monsoon humidity that favours perennial mould growth. These synergistic irritants act as a chronic inflammatory stimulus, shortening the interval between early-onset asthma and the development of fixed airflow obstruction.[4-8] Socio-economic barriers further delay specialist referral and

restrict access to guideline-directed therapy. Simultaneously, indiscriminate over-the-counter availability of oral corticosteroids encourages self-medication, thereby fostering steroid-related comorbidities such as obesity, osteoporosis, and type 2 diabetes that further exacerbate airway dysfunction.[9] Seasonal viral epidemics—most notably influenza and the post-monsoon rise in respiratory syncytial virus—also precipitate severe exacerbations, underscoring the importance of vaccination in comprehensive risk management.[10] Collectively, these context-specific drivers underscore the need for granular local data rather than wholesale extrapolation from Western registries. Obesity itself has emerged as a potent yet modifiable driver of severe asthma. Excess adiposity imposes mechanical restraint on diaphragmatic excursion, promotes systemic low-grade inflammation, skews macrophage polarisation, and alters the pharmacokinetics of inhaled medications—all of which blunt the therapeutic response to ICS.[4] Environmental pollutants, occupational irritants, and perennial allergens additionally trigger persistent airway inflammation and remodelling via oxidative-stress pathways and epigenetic re-programming.[5] Comorbidities—particularly chronic rhinosinusitis, gastro-oesophageal reflux disease, and obstructive sleep apnoea—compound morbidity by perpetuating nocturnal symptoms, increasing systemic inflammatory load, and necessitating higher doses of systemic corticosteroids.[6] The therapeutic landscape has been transformed in the past decade by biologic agents targeting type-2 (T2) inflammatory cytokines and IgE. Monoclonal antibodies such as omalizumab, mepolizumab, benralizumab, and dupilumab have demonstrated the ability to halve exacerbation rates and reduce steroid dependence in carefully selected T2-high phenotypes.[7] Nevertheless, these therapies remain expensive, require cold-chain logistics, and have variable reimbursement in low- and middle-income countries. As a result, their uptake in India is limited to a small subset of patients able to access speciality centres, and the majority of adults with severe asthma continue to rely on broad-spectrum corticosteroids with attendant toxicity. Robust evidence on the relative contributions of obesity, pollutant exposure, allergen sensitisation, and systemic eosinophilia to severe asthma in Indian adults is scant; most national studies either pre-date the biologic era or focus on paediatric populations.[11] Further, heterogeneity in study design and case definitions hampers synthesis of available data. We therefore conducted a single-centre cross-sectional study to characterise the contemporary risk-factor landscape of severe asthma among adults attending a tertiary-care hospital in Mumbai and to identify attributes independently associated with worse lung function and healthcare utilisation. By doing so, we hope to generate region-specific evidence that can sharpen preventive strategies, guide phenotype-directed therapy, and support cost-effective allocation of biologics within resource-limited health systems.

MATERIALS AND METHODS

Design & setting: Cross-sectional study at Jagjivan Ram Railway Hospital, Western Railway, Mumbai.
Participants: Adults ≥ 18 years with physician-diagnosed asthma requiring GINA Step 4/5 therapy (high-dose ICS + ≥ 1 controller) or remodelled asthma (fixed obstruction). Exclusion: non-asthmatic obstruction, poor adherence or technique, refusal.
Variables & instruments: Structured questionnaire captured demographics, exposures, comorbidities, medication history. Anthropometry followed WHO STEPS. Spirometry (MasterScreen, Vyaire) conformed to ATS/ERS 2023; reversibility tested with 400 µg salbutamol + 80 µg ipratropium. Blood tests included AEC, total IgE (chemiluminescence). ENT evaluation and CT paranasal sinuses identified chronic rhinosinusitis.
Outcome measure: Severe airflow obstruction defined as post-bronchodilator FEV_1 < 50 % predicted.
Statistics: Categorical variables as frequency (%), quantitative as mean ± SD or median (IQR). Associations tested by χ^2/Fisher's exact or independent-sample t-test. $p < 0.05$ significant. IBM SPSS 23.0.

RESULTS

The cohort (n = 55) had median asthma duration 23 years (IQR 12–28). Mean BMI 26.4 ± 5.1 kg m^{-2}; 36 % pre-obese, 24 % obese. Comorbidity burden was high [Figure 1]. Patients with BMI ≥ 30 kg m^{-2} had lower mean FEV_1 (41.2 ± 7.9 %) versus BMI < 30 (46.8 ± 9.1 %, p = 0.03). Exposure to pollutants/allergens correlated with frequent exacerbations (mean 3.4 vs 1.8/year, p = 0.04). Elevated AEC ≥ 500 predicted hospitalisation (OR 2.3, 95 % CI 1.1–4.9). [Figure 2] depicts the inverse relation between BMI and FEV_1/FVC ratio.

Table 1: Age distribution

Age group (y)	n	%
18–30	7	12.7
31–45	14	25.5
46–60	17	30.9
> 60	17	30.9

Table 2: BMI categories

BMI class	n	%
< 18.5	4	7.3
18.5–22.9	11	20.0
23–24.9	7	12.7
25–29.9	20	36.4
≥ 30	13	23.6

Table 3: Major comorbidities

Condition	n	%
Hypertension	31	56.4
Diabetes mellitus	22	40.0
Recurrent URTI	28	50.9

Table 4: Treatment profile

Controller	n	%
ICS + LABA + LAMA	55	100
Oral/IV steroid bursts	34	61.8
Biologic (any)	15	27.3

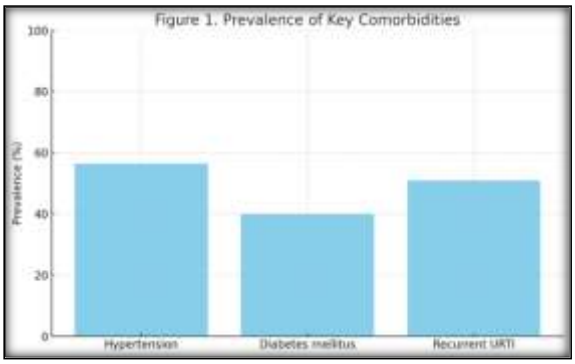

Figure 1: Prevalence of key comorbidities

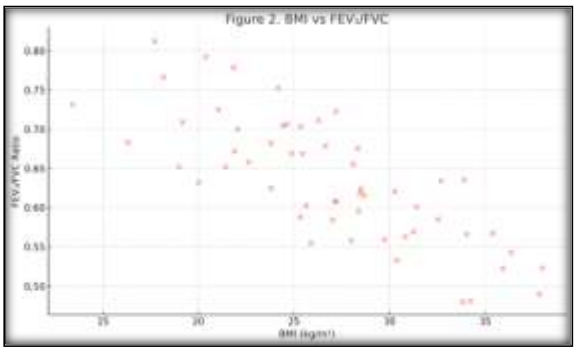

Figure 2. Scatterplot of BMI versus FEV_1/FVC (negative correlation, r = –0.42, p = 0.003).

DISCUSSION

Our findings highlight three actionable domains—obesity, eosinophilic inflammation, and environmental exposure—that collectively drive severe asthma in Indian adults. The overall picture is one of multifactorial vulnerability sustained by socio-environmental pressures and amplified by limited access to advanced therapy. The 60 % prevalence of overweight or obesity in our cohort mirrors recent urban-Indian surveillance reports and exceeds European severe-asthma registries.[8] Beyond mechanical load, adiposity promotes leptin-mediated neutrophilic inflammation that may reduce ICS pharmacodynamics and favour a T2-low, steroid-insensitive endotype.[4] Weight-reduction interventions—including structured nutrition programmes and bariatric surgery—have yielded clinically meaningful gains in asthma control and lung function, underscoring their relevance to this population.[9] Yet only 3.6 % of our participants reported regular exercise, highlighting an urgent need for behaviour-change counselling and community-based fitness initiatives. Integrating pulmonary rehabilitation into routine outpatient care could bridge this gap while concurrently addressing deconditioning and mood disturbance. Eosinophilia (62 %) and elevated IgE (55 %) signify a predominantly T2-high pattern that is theoretically amenable to anti-IL-5 or anti-IgE biologic therapy. However, only 27 % of eligible patients had accessed biologics, reflecting cost, inconsistent reimbursement, and limited availability in public hospitals. Real-world data show that biologics halve exacerbations and oral-steroid dependence, even in high-burden settings.[10] To translate this evidence into practice, national procurement policies could negotiate price caps, and pay-for-performance contracts might ensure cost-effectiveness. Pragmatic algorithms that combine peripheral eosinophil counts, serum IgE, and clinical surrogates (e.g., nasal polyps) offer an affordable first-pass screen for biologic eligibility and could be disseminated through primary-care networks. Nearly three-quarters of patients reported substantial pollutant or occupational exposure, consistent with the industrial milieu of Mumbai, where ambient $PM_{2.5}$ routinely exceeds WHO limits. Repeated irritant exposure accelerates airway remodelling and blunts bronchodilator responsiveness. Targeted counselling, employer-led engineering controls, and wider adoption of India's Clean Air Programme may mitigate risk at scale. At an individual level, portable particulate filters and N95 respirators have demonstrated short-term reductions in airway inflammation markers,[11] but their sustained effectiveness in severe asthma warrants further study. Hypertension (56 %), diabetes mellitus (40 %), and

recurrent URTI (51 %) were strikingly common. These conditions share inflammatory pathways with asthma and independently reduce lung-function reserve. Integrated disease-management models—in which pulmonologists, endocrinologists, and otolaryngologists share electronic care plans—could streamline optimisation of comorbidities and reduce polypharmacy.[12] Notably, 27 % of participants had osteoporosis, emphasising the collateral harm of chronic systemic-steroid exposure. A move toward steroid-sparing regimens, facilitated by biologics and optimised inhaler technique, is therefore imperative. Strengths of our study include comprehensive phenotyping, inclusion of hard-to-reach public-sector patients, and focus on an under-studied region. Limitations are the single-centre design, modest sample size, and reliance on peripheral eosinophil counts rather than sputum cytokine profiling. The cross-sectional nature precludes causal inference; however, the strong associations observed provide a rational basis for longitudinal investigation. Future work should incorporate digital inhaler monitoring and wearable pollution sensors to capture real-time interactions between adherence, exposure, and symptom burden. Our data argue for a multifaceted intervention bundle: (i) community-led weight-management programmes, (ii) affordable access to phenotype-matched biologics, (iii) occupational-health partnerships for exposure reduction, and (iv) integrated management of cardiometabolic and upper-airway comorbidities. Implementation science frameworks could evaluate scalability across India's diverse health-system tiers. Moreover, inclusion of severe-asthma modules in national non-communicable-disease platforms would enable systematic surveillance and resource allocation. In summary, obesity, type-2 inflammation, and environmental exposure emerge as synergistic, modifiable drivers of severe asthma in urban Indian adults. Addressing these domains through coordinated clinical and public-health strategies could attenuate the escalating burden of refractory disease.

CONCLUSION

Severe asthma in this tertiary-care cohort is characterised by high rates of adiposity, eosinophilia, and environmental exposure, each independently associated with worse lung function. Proactive weight management, reduction of pollutant exposure, and broader access to phenotype-guided biologic therapy could meaningfully reduce disease burden.

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