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Biomarkers and Polysomnographic Parameters in Obstructive Sleep Apnea and OSA with Comorbidities: A Narrative Review

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Abstract: Obstructive sleep apnea (OSA) is a highly prevalent sleep-related breathing disorder that drives substantial cardiovascular, metabolic, and cerebrovascular morbidity through recurrent cycles of intermittent hypoxia, sleep fragmentation, oxidative stress, and systemic inflammation. Globally, OSA affects close to one billion adults, with roughly 425 million living with moderate-to-severe disease; prevalence estimates in India range from 3.7% to 21%, with about 5% of adults affected at a moderate-to-severe level. Despite this burden, the apnea-hypopnea index (AHI) — the mechanical, frequency-based metric that anchors current diagnostic and severity classification — correlates poorly with individual comorbidity risk and symptom burden, as it disregards event duration, desaturation depth, sleep-stage distribution, and cumulative hypoxic load. Major sleep and cardiovascular society guidelines have not yet incorporated inflammatory or REM-specific biomarkers into diagnostic or prognostic frameworks, even though expert consensus repeatedly flags this as an unmet need. This review synthesizes the rationale for and evidence behind a set of low-cost, routinely available blood-derived biomarkers — serum uric acid, fibrinogen, neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and the systemic inflammatory response index (SIRI) — as candidate correlates of polysomnographic severity in OSA, with particular attention to REM-specific indices and to patients with OSA-associated comorbidities such as hypertension, diabetes, and chronic obstructive pulmonary disease. We map the evidentiary, conceptual, methodological, measurement, population, temporal, and translational gaps that currently limit clinical adoption of these markers, particularly in resource-constrained and under-represented settings such as India. Addressing these gaps through adequately powered, stage-specific, and longitudinal studies could establish affordable biomarker panels as scalable adjuncts to AHI for risk stratification and could inform decision-support pathways for respiratory therapists and clinicians managing OSA in low- and middle-income countries.

Keywords: *Obstructive Sleep Apnea; Polysomnography; Biomarkers; Uric Acid; Fibrinogen; Neutrophil-To-Lymphocyte Ratio; Systemic Inflammatory Response Index; REM Sleep; Comorbidities.*

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I. INTRODUCTION

Obstructive sleep apnea (OSA) is a chronic, highly prevalent sleep-related breathing disorder marked by recurrent episodes of upper airway obstruction during sleep. These episodes produce intermittent hypoxemia, hypercapnia, and repeated sleep fragmentation, setting off a cascade of physiological derangements that extend well beyond the hours of sleep itself [1].

The scale of the problem is considerable. Global estimates place the prevalence of OSA at nearly one billion adults, with approximately 425 million affected by moderate-to-severe disease [1]. In India, reported prevalence ranges widely from

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3.7% to 21% depending on the population studied, with roughly 5% of adults estimated to have moderate-to-severe disease [2,3]. Despite this scale, OSA remains substantially underdiagnosed and undertreated, and this diagnostic gap contributes directly to the downstream cardiovascular and metabolic disease burden attributable to the condition [4].

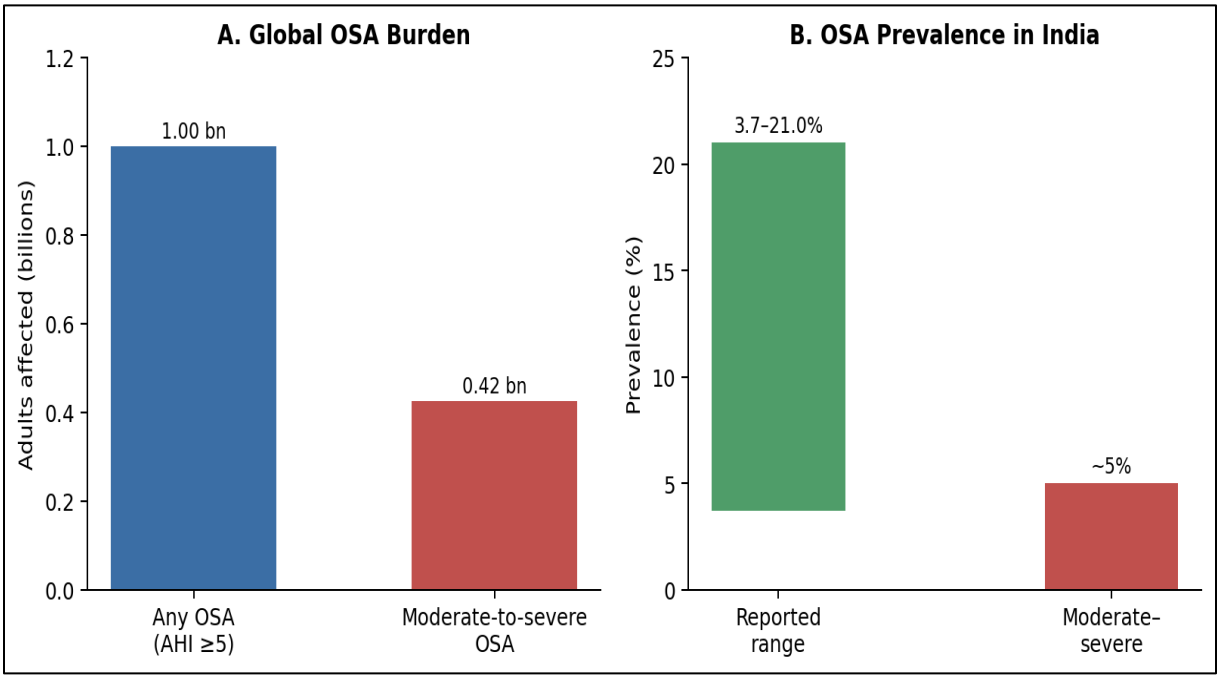

Fig 1. Global and Indian Estimates of OSA Prevalence, Illustrating the Overall Burden (Panel A) and the Wide Reported Prevalence Range in Indian Cohorts Alongside the Moderate-To-Severe Subset (Panel B). Data Drawn from Benjafield et al. [1], Suri et al. [2], and Udwadia et al. [3].

OSA is strongly and consistently linked to a cluster of comorbid conditions. It behaves as an independent predictor of both new-onset and treatment-resistant hypertension, contributes to insulin resistance and a higher prevalence of type 2 diabetes and metabolic syndrome, and raises the risk of coronary artery disease, arrhythmia, heart failure, sudden cardiac death, and stroke. Chronic kidney disease and non-alcoholic fatty liver disease are increasingly reported as additional end-organ consequences in patients with untreated disease [5–9].

Mechanistically, these complications arise through interconnected pathways. Recurrent cycles of intermittent hypoxia and reoxygenation generate reactive oxygen species and activate hypoxia-inducible factor-1α, driving oxidative stress; heightened sympathetic activity produces catecholamine surges and vascular remodeling; and endothelial dysfunction — marked by reduced nitric oxide availability and increased arterial stiffness — accelerates atherogenesis. Chronic systemic inflammation, evidenced by elevated hs-CRP, IL-6, IL-8, and TNF-α, sits at the center of this process, promoting vascular injury, metabolic disturbance, and progression of atherosclerosis [10–13].

Notably, major international guidelines have not caught up with this mechanistic understanding. The American Academy of Sleep Medicine and the International Classification of Sleep Disorders continue to define OSA on the basis of the apnea-hypopnea index (AHI) and oxygen desaturation thresholds alone [14,15]. The American Heart Association recognizes OSA as a cardiovascular risk factor but stops short of recommending biomarker-based stratification [16], and the European Respiratory Society's guidelines emphasize non-CPAP therapies without incorporating inflammatory or REM-specific markers [17]. Multiple expert statements have nonetheless flagged the need for biomarkers capable of improving clinical decision-making in OSA [18–21], and it is this evidence gap that motivates the present review.

Traditional inflammatory cytokines such as IL-6, IL-8, and TNF-α are well characterized in OSA, but their reliance on advanced immunoassays limits routine clinical use, particularly in resource-constrained health systems such as India's [12]. This review instead focuses on a set of low-cost, widely available biomarkers derived from routine hematological and biochemical testing — serum uric acid, fibrinogen, and complete blood count (CBC)-derived indices such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic inflammatory response index (SIRI) — that show promise as feasible, scalable correlates of OSA severity and comorbidity risk.

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II. HISTORICAL DEVELOPMENT OF THE EVIDENCE BASE

The recognition of OSA's systemic reach has evolved chronologically. Early epidemiological work established a strong, independent association between OSA and systemic hypertension [30,32]. Subsequent studies extended this to serious cardiovascular outcomes, including coronary artery disease and stroke [33,34]. The concept of a bidirectional relationship between OSA and metabolic syndrome — central obesity, hypertension, dyslipidemia, and insulin resistance — emerged next, prompting Somers and colleagues to coin the term "Syndrome Z" to capture this convergence [35,36]. Young et al. (2004) further quantified the prevalence and synergistic effects of these risk factors [37], and Yaggi et al. (2005) provided definitive evidence that OSA independently predicts stroke and transient ischemic attack [38]. Taken together, this body of work firmly positions OSA as a major contributor to the global non-communicable disease burden — one that demands prognostic tools reflecting its full systemic reach, rather than tools confined to respiratory event frequency alone.

III. LIMITATIONS OF THE APNEA-HYPOPNEA INDEX AS A SOLE SEVERITY METRIC

The AHI, derived from polysomnography (PSG), remains the diagnostic and severity-classification gold standard for OSA [39]. Yet the AHI is fundamentally a mechanical, frequency-based count: it treats every respiratory event as equivalent, regardless of its duration, the depth of the accompanying oxygen desaturation (nadir SpO_2), the degree of associated sleep fragmentation (arousal index), or the cumulative burden of chronic intermittent hypoxia (CIH) [40–42]. As a result, a patient with a moderate AHI but profound nocturnal hypoxemia and a high inflammatory load may carry a substantially greater risk of cardiovascular disease or metabolic syndrome than a patient with a numerically higher AHI but preserved oxygenation [43]. Mokhlesi et al. (2020) further showed that AHI correlates poorly with patient-reported excessive daytime sleepiness, underscoring its limitations as a universal proxy for disease impact [44]. These observations argue for biomarkers capable of capturing the downstream molecular consequences of CIH and inflammation — moving OSA classification beyond a purely mechanical count toward genuine physiological risk stratification [45].

IV. LOW-COST BIOMARKERS AS CANDIDATE CORRELATES OF OSA SEVERITY

An ideal complementary biomarker for OSA should be sensitive to OSA-induced pathology, correlate with end-organ damage, respond to treatment, and — critically — be inexpensive and easy to incorporate into routine practice [46]. Two biologically distinct but complementary marker sets meet these criteria: markers of oxidative stress and coagulation (uric acid and fibrinogen), and composite indices of systemic immune-inflammation (NLR and SIRI).

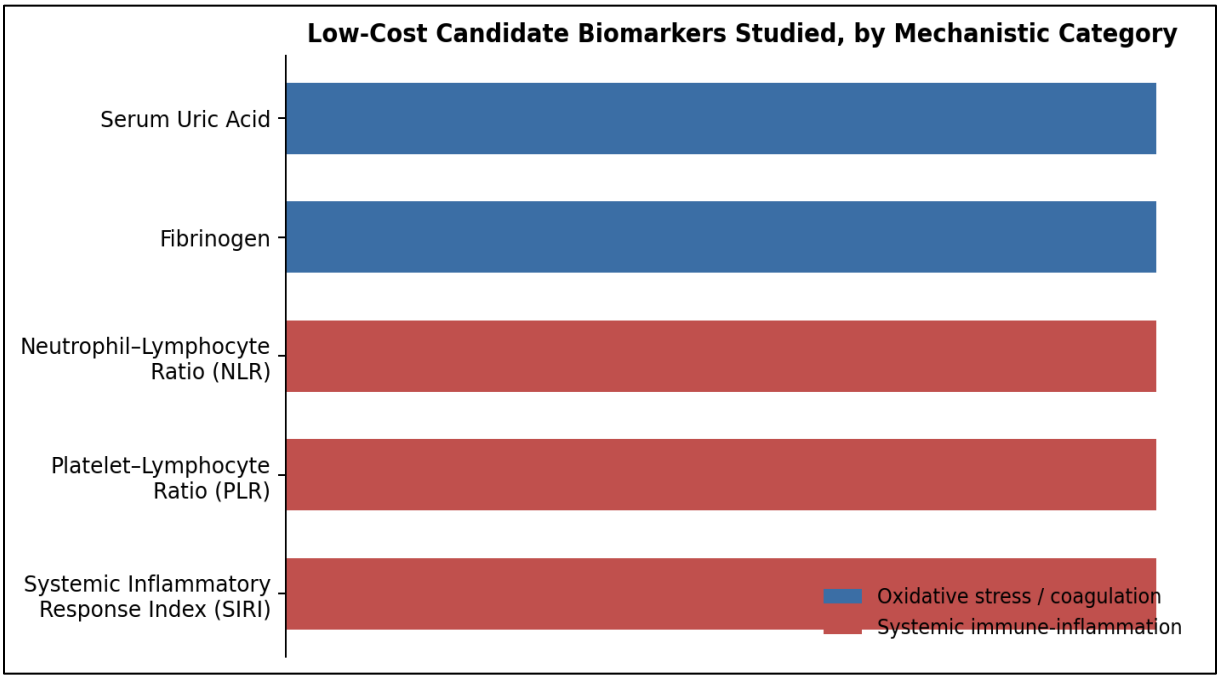

Fig 2. Candidate Low-Cost Biomarkers Evaluated in this Review, Grouped by their Principal Mechanistic Category.

➢ *Markers of Oxidative Stress and Coagulation*

Serum uric acid, the end-product of purine metabolism, is generated through CIH-induced activation of xanthine oxidase, making it a simple and inexpensive proxy for the degree of oxidative insult [47,48]. Rodriguez-Colinas et al. (2016) demonstrated its correlation with AHI severity, and its

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independent association with hypertension and metabolic syndrome adds further value for risk stratification [49,50]. Fibrinogen, a major acute-phase reactant and coagulation protein, offers an accessible readout of systemic inflammation and pro-thrombotic tendency [52]. CIH and inflammation in OSA are known to raise circulating fibrinogen, reflecting haemostatic dysfunction and a heightened risk of thrombotic events such as stroke, as confirmed by von Känel et al. (2014) [53,54].

➢ *Markers of Systemic Immune-Inflammation*

Composite indices derived from a standard complete blood count capture the multi-cellular character of OSA-related inflammation more fully than any single cytokine. The neutrophil-to-lymphocyte ratio (NLR) — the absolute neutrophil count divided by the absolute lymphocyte count — offers a simple readout of the balance between pro-inflammatory activation (neutrophilia) and immuno-regulatory suppression (lymphopenia) [56]. Elevated NLR has been consistently linked to greater OSA severity and independently predicts adverse cardiovascular outcomes across multiple chronic diseases, an elevation attributed to the endothelial activation and stress-hormone release triggered by CIH [57,58]. The systemic inflammatory response index (SIRI), a newer composite that additionally incorporates monocyte counts — key drivers of chronic inflammation and atherosclerosis in OSA — has shown stronger predictive value for severe OSA than NLR alone in recent work, including a 2025 retrospective analysis, positioning it as a promising tool for early recognition and referral [59–61]. Combining these markers (uric acid, fibrinogen, NLR, and SIRI) with existing PSG data offers a pathway toward more comprehensive, biologically grounded risk assessment for patients with OSA and its associated comorbidities.

V. SYNTHESIS OF RESEARCH GAPS

Despite growing interest, the literature connecting low-cost biomarkers to polysomnographic severity remains fragmented across several dimensions.

➢ *Evidence Gap*

Most existing work has centered on expensive or specialized assays such as IL-6, TNF-α, or CRP, leaving affordable markers — uric acid, fibrinogen, NLR, PLR, and SIRI — comparatively underexplored, particularly against PSG metrics beyond overall AHI, including REM-specific hypoxic burden and sleep fragmentation.

➢ *Conceptual Gap*

The mechanistic links between OSA-specific sleep-stage disturbances and biomarker signatures remain poorly defined. It is unclear how REM-predominant disease, apnea duration, or desaturation morphology specifically shape biomarker alterations, and the differential biomarker–PSG relationships in overlap syndromes (OSA with COPD, diabetes, or hypertension) have not been well characterized.

➢ *Methodological Gap*

Existing studies are frequently cross-sectional, single-center, and underpowered, and rely on AHI as the sole severity index. Confounders such as obesity, metabolic syndrome, smoking, and medication use are often inadequately controlled, and single-timepoint (typically morning) blood sampling leaves diurnal variation in biomarker expression unexplored.

➢ *Measurement Gap*

Reliance on AHI as the dominant PSG metric overlooks more sensitive indices — REM-AHI, REM-ODI, hypoxic burden, desaturation area index, and arousal index — and biomarker cut-offs are rarely calibrated against sleep-stage- or event-specific PSG loads, weakening the signal-to-noise ratio needed to establish clinically meaningful thresholds.

➢ *Population Gap*

Most cohorts are Western and male-dominated, with limited representation of women, older adults, and patients from low- and middle-income countries. Indian populations remain under-represented despite a high combined burden of OSA and related comorbidities such as COPD, diabetes, and hypertension, limiting the external validity of existing findings to diverse socio-economic contexts.

➢ *Temporal Gap*

The predominantly cross-sectional nature of existing studies leaves prognostic and longitudinal questions unanswered — whether baseline biomarkers predict worsening PSG severity or comorbidity onset, and whether these markers track improvements in REM-specific indices or hypoxic burden with CPAP or BiPAP therapy.

➢ *Translational Gap*

No validated clinical pathway currently integrates affordable biomarker testing with PSG triage in resource-limited settings. Cost-effectiveness analyses in the Indian context are scarce, and clinicians and respiratory therapists lack simple decision-support tools for patient stratification, limiting the scalability of biomarker-informed screening.

VI. TOWARD ADDRESSING THE GAPS: A PROPOSED STUDY FRAMEWORK

A cross-sectional, observational study is proposed to test whether low-cost biomarkers (uric acid, fibrinogen, NLR, PLR, and SIRI) associate with PSG parameters — with particular attention to REM-specific indices — in adults with OSA alone versus OSA with comorbidities (COPD, diabetes, hypertension). Based on an anticipated correlation coefficient of $r = 0.35$ between biomarkers and PSG severity (drawn from Topuz et al. [62] and Shahul et al. [63]), a minimum of 62 participants would be required for 80% power at 5% significance; accounting for an expected 15% attrition from incomplete PSG data, a target enrollment of 72 participants is proposed, split evenly between the two clinical groups.

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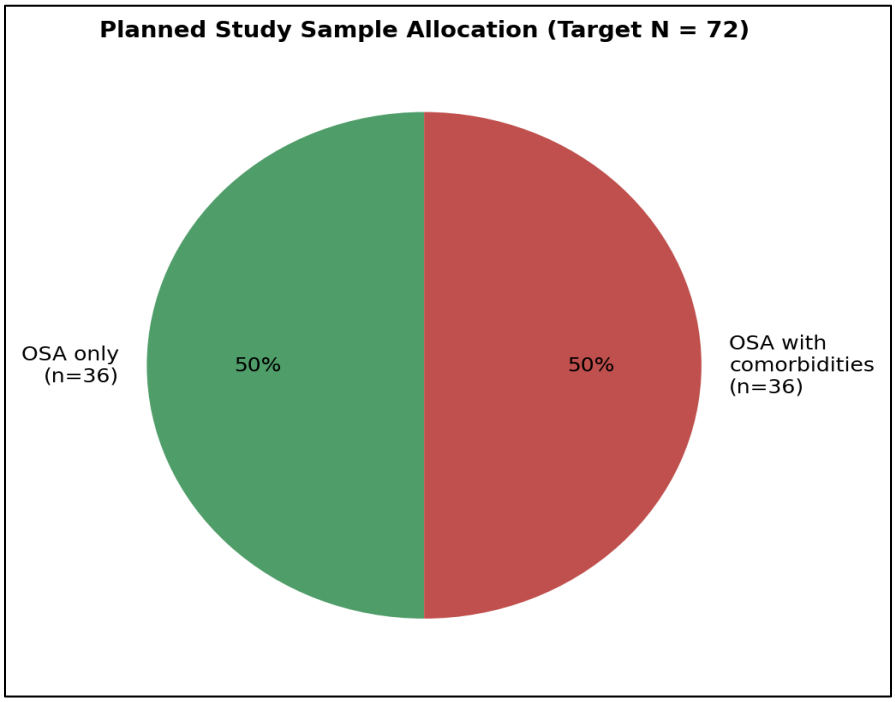

Fig 3. Proposed Sample Allocation for a Study Designed to Test Biomarker–PSG Associations in OSA with and without Comorbidities.

Such a design — combining full-night, in-lab PSG with fasting biomarker assays, standardized covariate capture, and multivariable regression adjusted for age, sex, BMI, smoking, and comorbidities — would directly address the evidence, conceptual, methodological, measurement, population, temporal, and translational gaps outlined above, while remaining feasible in a resource-constrained Indian tertiary-care setting.

VII. CONCLUSION AND FUTURE PERSPECTIVES

OSA imposes a large and growing global and Indian health burden, driven by mechanisms — intermittent hypoxia, oxidative stress, and systemic inflammation — that the AHI alone cannot capture. Low-cost, routinely available biomarkers such as uric acid, fibrinogen, NLR, PLR, and SIRI offer a biologically plausible and pragmatically feasible complement to PSG-based severity classification, particularly for identifying REM-specific hypoxic burden and comorbidity risk. Realizing this potential will require adequately powered, longitudinal studies in diverse, under-represented populations — including Indian cohorts with high rates of OSA-comorbidity overlap — coupled with efforts to translate validated biomarker thresholds into simple, decision-support pathways for frontline clinicians and respiratory therapists. Such work could ultimately enable scalable, affordable risk stratification for OSA in low- and middle-income healthcare settings.

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