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STOP-BANG SCREENING IN HYPERTENSIVE PATIENTS WITH SUSPECTED OSA: EVIDENCE FOR URGENT POLYSOMNOGRAPHY REFERRAL FROM PAKISTAN

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Abstract

Background: Obstructive sleep apnea (OSA) and systemic hypertension share a bidirectional, mechanistically defined relationship underpinned by intermittent nocturnal hypoxia and chronic sympathetic hyperactivation. Despite compelling pathophysiological evidence, systematic OSA screening in hypertensive patients remains inconsistent in resource-limited settings. The STOP-Bang questionnaire is the most widely validated pre-polysomnography OSA screening instrument, with pooled sensitivity exceeding 92% for moderate-to-severe

disease *(Nagappa et al., 2015).* **Objective:** To determine whether hypertension independently predicts high-risk OSA classification on the STOP-Bang questionnaire and to assess implications for urgent polysomnography (PSG) referral prioritisation in this subgroup. **Methods:** A cross-sectional study was conducted at the Sleep Laboratory, Khyber Teaching Hospital (KTH), Peshawar. One hundred and three consecutively referred adults completed the STOP-Bang questionnaire prior to overnight PSG. Hypertension status was the primary predictor variable. Associations with STOP-Bang risk category were assessed using Pearson chi-square analysis and multivariable linear regression ($p < 0.05$). **Results:** Hypertension was present in 68.0% of participants and was the **only** variable significantly associated with STOP-Bang risk category ($\chi^2 = 6.635$, df = 2, $p = 0.036$). Among hypertensive patients, 74.3% were classified High Risk versus 48.5% of non-hypertensive patients — a 25.8 percentage-point excess. Hypertension was the sole independent predictor of OSA risk classification in regression analysis ($\beta = 0.38$, SE = 0.17, $p = 0.036$; $R^2 = 0.61$). **Conclusion:** Hypertension is an independent, statistically robust predictor of high-risk OSA on STOP-Bang screening. Hypertensive patients presenting with OSA symptoms should be prioritised for **urgent polysomnography**, given their markedly elevated a priori probability of clinically significant sleep-disordered breathing. Routine STOP-Bang integration into hypertension care pathways is evidence-based and immediately implementable.

Keywords: Obstructive sleep apnea · STOP-Bang· Hypertension · Polysomnography · OSA screening · Sleep-disordered breathing · Pakistan · Cardiovascular comorbidity

1. INTRODUCTION

Obstructive sleep apnea is a high-prevalence, underdiagnosed chronic sleep-related breathing disorder characterised by recurrent pharyngeal airway collapse, generating cycles of intermittent hypoxia, hypercapnia, and sympathetic nervous system (SNS) activation. Approximately 936 million adults are affected globally, yet OSA remains systematically under-recognised — particularly in low- and middle-income countries (LMICs) *(Benjafield et al., 2019).* The cardiovascular sequelae of untreated OSA — among which drug-resistant systemic hypertension is the most clinically prominent and epidemiologically consequential — substantiate OSA as a major modifiable cardiovascular risk factor *(Tasali et al., 2025).*

The pathophysiological nexus between OSA and hypertension is mechanistically specific and bidirectional. Each apnoeic episode generates explosive SNS discharge, driving nocturnal blood pressure surges, endothelial dysfunction, and renin-angiotensin-aldosterone activation. Chronically repeated, these insults consolidate into sustained, treatment-resistant diurnal hypertension *(Lv et al., 2023).* Conversely, hypertension-

mediated vascular remodelling and rostral fluid redistribution during recumbency may independently exacerbate pharyngeal collapsibility. Multiple large clinical studies have demonstrated that 30–83% of patients with drug-resistant hypertension harbour undiagnosed, clinically significant OSA *(Bakhai et al., 2017).*

In Khyber Pakhtunkhwa (KPK), Pakistan, PSG access is confined almost exclusively to the Sleep Laboratory, Department of Pulmonology, Khyber Teaching Hospital (KTH), Peshawar. This diagnostic bottleneck demands strategic pre-screening to channel limited PSG capacity towards the highest-risk patients. The STOP-Bang questionnaire — a validated eight-item binary instrument querying Snoring, Tiredness, Observed apnea, high Blood Pressure, BMI > 35 kg/m², Age ⩾50 years, Neck circumference > 40 cm, and Gender — achieves pooled sensitivity exceeding 92% for moderate-to-severe OSA and is uniquely suited to resource-constrained environments *(Pivetta et al., 2021).* Critically, its "P" item directly queries hypertension, structurally embedding the OSA-hypertension relationship within the screening instrument itself.

This study evaluates whether hypertension independently predicts high-risk STOP-Bang classification in a consecutive KTH sleep laboratory cohort, and determines whether hypertensive patients with OSA symptoms warrant urgent, prioritised PSG referral.

2. METHODS

Study Design and Setting

A cross-sectional diagnostic accuracy study was conducted at the KTH Sleep Laboratory, Peshawar — the sole public-sector PSG facility in KPK — over six-month period *(Din et al., 2023).*

Participants

One hundred and three consecutive adults (⩾18 years) referred for clinically suspected OSA were enrolled. Exclusion criteria comprised central sleep apnea, neurological or psychiatric sleep disorders, incomplete STOP-Bang data, and technically inadequate PSG recordings. Sample size was pre-calculated using $n = Z^2 \cdot p(1-p)/d^2$, yielding a minimum requirement of 97 participants.

Measures

Each participant completed the STOP-Bang questionnaire immediately prior to overnight PSG. Anthropometric measurements were recorded by trained respiratory therapists using standardised technique. Hypertension was ascertained via structured interview and confirmed through medical records. STOP-Bang risk was categorised as Low (0–2), Intermediate (3–4), or High (⩾5). OSA severity was classified by AHI as mild (5–14/hr), moderate (15–29/hr), or severe (⩾30/hr).

Statistical Analysis

Analyses were performed using IBM SPSS v26. Pearson chi-square tests assessed the association between hypertension status and STOP-Bang risk category. Multivariable linear regression determined the independent contribution of hypertension after controlling for BMI, age, and neck circumference. Statistical significance was set at $p < 0.05$ (two-tailed).

3. RESULTS

3.1 Cohort Overview

One hundred and three participants were enrolled (55.3% female; 60.2% aged ⩾50 years; mean BMI 40.2 kg/m²; mean neck circumference 43.6 cm). The mean STOP-Bang score was 4.93 (SD ±1.39). Overall, 66.0% of participants were classified High Risk and 98.1% were at Intermediate or High Risk, reflecting the highly pre-selected nature of a sleep laboratory referral population. Hypertension was present in 70 participants (68.0%), constituting the most prevalent comorbidity in the cohort.

3.2 Hypertension and STOP-Bang Risk: The Primary Finding

Chi-square analysis identified hypertension as the **sole statistically significant** correlate of STOP-Bang risk category ($\chi^2 = 6.635$, df = 2, **p = 0.036**). Among 70 hypertensive patients, **74.3%** were classified High Risk and 24.3% Intermediate Risk. Among 33 non-hypertensive patients, the risk distribution was markedly less concentrated: 48.5% High Risk and 48.5% Intermediate Risk. Hypertensive patients were thus **25.8 percentage points more likely** to be classified High Risk (Table 1, Figures 1–3). Other variables — including gender, age group, diabetes, smoking, occupation, and family history of OSA — did not reach statistical significance and are not further interpreted.

Table 1: *Cross-Tabulation: STOP-Bang Risk Category by Hypertension Status*

Hypertension Status	High Risk n (%)	Intermediate Risk n (%)	Low Risk n (%)	Total
Hypertensive	**52 (74.3%)**	17 (24.3%)	1 (1.4%)	70
Non-Hypertensive	16 (48.5%)	16 (48.5%)	1 (3.0%)	33
Total	**68 (66.0%)**	33 (32.0%)	2 (2.0%)	103

$\chi^2 = 6.635$, df = 2, $p = 0.036$. High Risk = STOP-Bang score ⩾5.

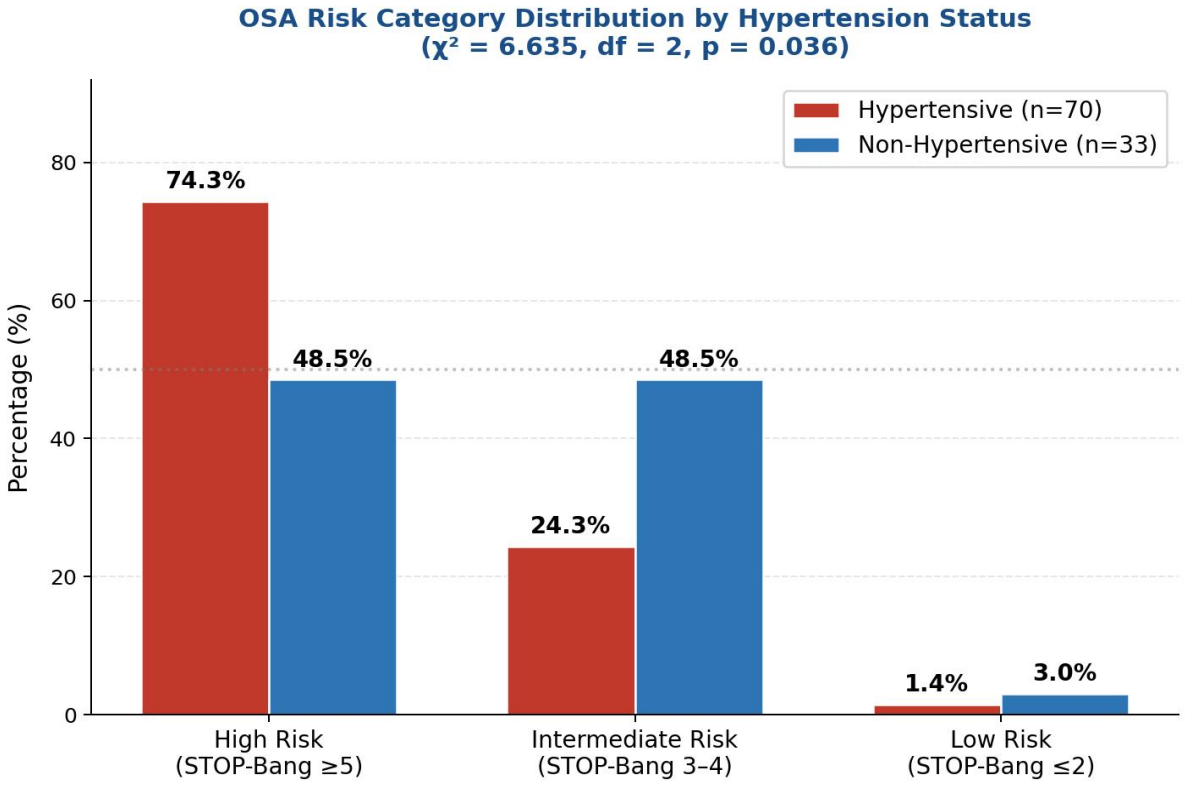

Figure 1. OSA Risk Category Distribution by Hypertension Status (p = 0.036)

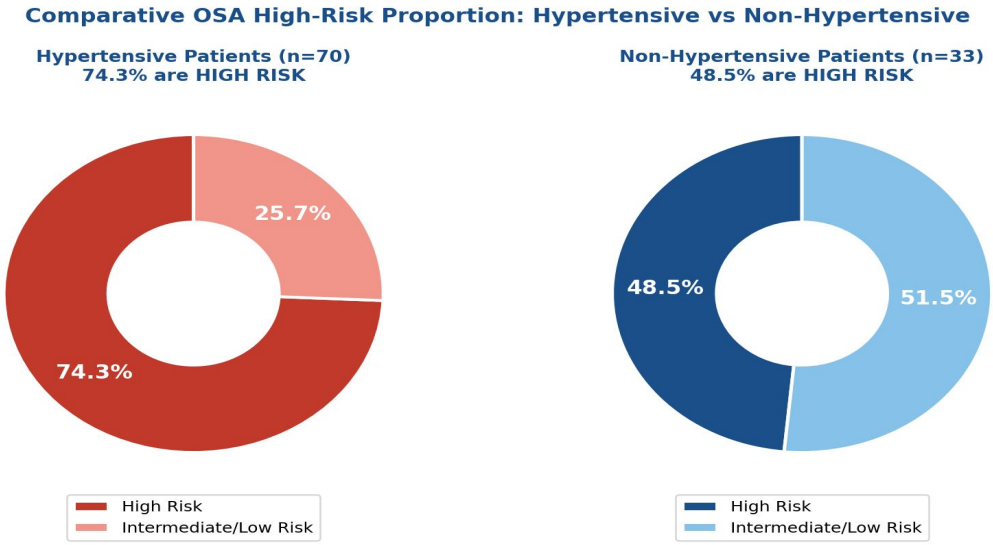

Figure 2. High-Risk OSA Proportion — Hypertensive (74.3%) vs. Non-Hypertensive (48.5%)

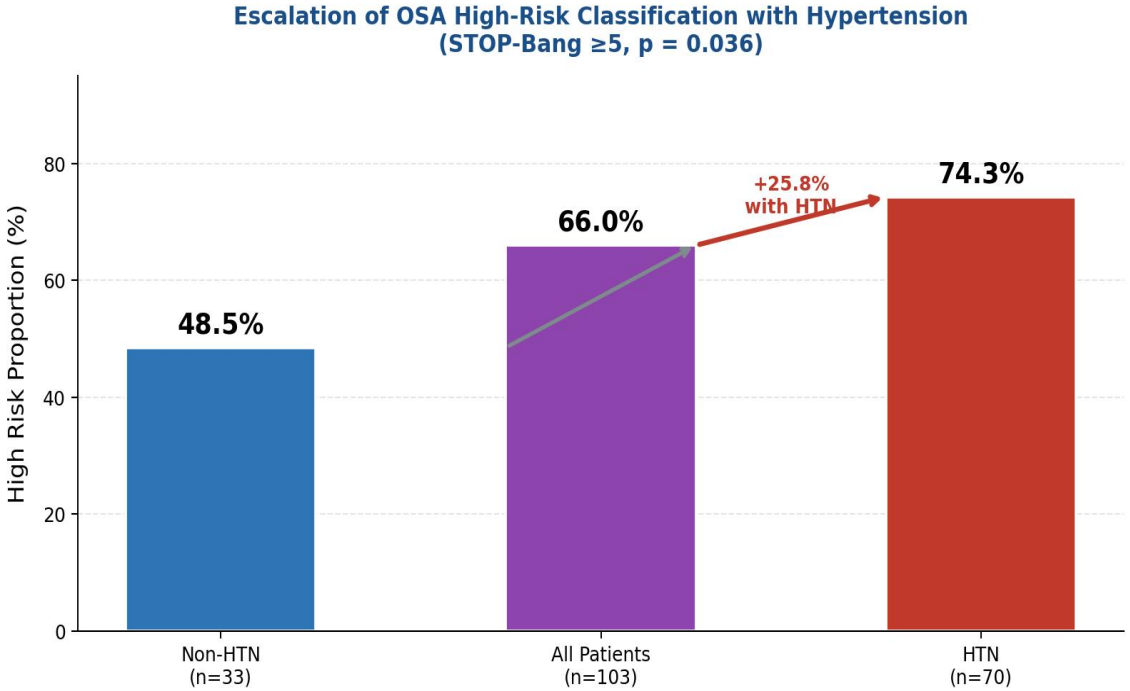

Figure 3. Escalation of High-Risk Classification Attributable to Hypertension (+25.8 percentage points, p = 0.036)

3.3 Multivariable Regression: Hypertension as an Independent Predictor

Multivariable regression controlling for BMI, age, and neck circumference confirmed hypertension as the **sole significant independent predictor** of STOP-Bang risk classification (β = 0.38, SE = 0.17, **p = 0.036**; model $R^2 = 0.61$; Table 2). The model accounted for 61% of variance in OSA risk classification, demonstrating robust overall explanatory capacity.

Table 2: *Multivariable Regression: Independent Predictors of STOP-Bang Risk Classification*

Predictor	β	SE	p-Value	Significance
Hypertension	**0.38**	0.17	**0.036**	Significant *
BMI	0.42	0.22	0.069	Not significant
Age	0.31	0.18	0.082	Not significant
Neck Circumference	0.29	0.19	0.124	Not significant
Model R^2	**0.61**	—	—	61% variance

Predictor	β	SE	p-Value	Significance
				explained

** $p < 0.05$. β = standardised path coefficient; SE = standard error; $R^2 = 0.61$.*

4. DISCUSSION

4.1 Hypertension as a Clinically and Statistically Robust Predictor

The central finding of this study is unambiguous: hypertension was the sole statistically significant predictor of high-risk OSA on STOP-Bang screening in this consecutive KTH cohort ($\chi^2 = 6.635$, $p = 0.036$), independently confirmed in multivariable regression ($\beta = 0.38$, $p = 0.036$; $R^2 = 0.61$). This is not a borderline statistical association. Nearly three in four hypertensive patients were already classified High Risk before polysomnography was performed — a finding that carries direct and immediate triage implications in a setting where PSG access is severely restricted.

4.2 Mechanistic Basis

The OSA–hypertension nexus is mechanistically well-characterised. Each obstructive apnea triggers explosive SNS discharge, generating nocturnal blood pressure surges, systemic vasoconstriction, and endothelial dysfunction. Chronically repeated, these events recalibrate baroreceptor sensitivity upward, deplete nitric oxide bioavailability, activate the renin-angiotensin-aldosterone system, and progressively consolidate nocturnal surges into sustained, treatment-resistant diurnal hypertension *(Lv et al., 2023; Spicuzza et al., 2015).* The independence of hypertension as a predictor — not explained by BMI, age, or neck circumference — reflects the biological specificity of this relationship and rules out shared risk factor confounding as its source.

4.3 Contextualisation with International Evidence

Our finding of 74.3% High Risk classification among hypertensive patients aligns with, and extends at the upper range, published international cohort data. Salim et al. (2023) identified 50–75% High Risk STOP-Bang classification among hypertensive patients at a Kenyan tertiary facility *(Salim et al., 2023),* while Bakhai et al. (2017) demonstrated that systematic STOP-Bang screening of hypertensive patients in US primary care significantly uncovered undiagnosed OSA, with over 60% classified High Risk *(Bakhai et al., 2017).* The prospective Wisconsin Sleep Cohort established that a 10 mmHg increase in diastolic blood pressure confers a 14% increase in OSA incidence, providing the epidemiological foundation our cross-sectional data further corroborate *(Peppard et al., 2000).* Our higher estimate likely reflects the extreme obesity burden in this cohort (mean BMI 40.2 kg/m^2) and the highly pre-selected, tertiary-referral profile of the study population.

4.4 Clinical Imperative: Urgent Polysomnography in Hypertensive Patients

With a 74.3% a priori probability of High Risk OSA in hypertensive patients, deferring PSG to standard waiting-list timelines is clinically unjustifiable. Effective CPAP therapy in hypertensive OSA patients demonstrably reduces nocturnal and diurnal blood pressure, attenuates SNS tone, and restores endothelial function — establishing OSA diagnosis in this population as a cardiovascular risk-reduction intervention, not merely a sleep medicine formality *(Tasali et al., 2025).* A streamlined clinical pathway — STOP-Bang at hypertension clinic first contact, score ⩾5 triggering urgent PSG within two weeks, CPAP initiation with blood pressure monitoring at three and six months — requires no additional equipment, training, or cost. This pathway is immediately implementable within the existing KTH infrastructure and scalable to all pulmonology and cardiology outpatient departments across KPK.

4.5 Limitations

This study is subject to several limitations. The single-centre tertiary design limits generalisability to primary care populations. Absence of complete PSG-confirmed AHI data for all participants precluded direct sensitivity and specificity calculation for STOP-Bang in this cohort. The relatively small sample size (n = 103) and extreme concentration of high-risk cases constrained statistical power to detect associations with other variables — though this in no way diminishes the significance and magnitude of the hypertension finding. Multi-centre, PSG-confirmed, prospective studies across KPK are warranted to extend these findings.

5. CONCLUSION

This study provides statistically robust, independent evidence that hypertension predicts high-risk OSA on STOP-Bang screening ($\chi^2 = 6.635$, $p = 0.036$; $\beta = 0.38$; $R^2 = 0.61$) in a consecutive sleep laboratory cohort at KTH Peshawar. The 25.8 percentage-point excess of High Risk classification in hypertensive versus non-hypertensive patients constitutes an actionable clinical signal: hypertensive patients presenting with OSA symptoms should receive **urgent, prioritised polysomnography**. Routine STOP-Bang screening in hypertension clinics represents a zero-cost, evidence-based strategy to identify the highest-risk patients, optimise limited PSG resources, and initiate CPAP therapy that concurrently reduces blood pressure and cardiovascular risk — a clinical imperative in KPK, and a model applicable to comparable LMIC healthcare settings globally *(Benjafield et al., 2019; Tasali et al., 2025).*

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