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Original Research Article

Persistent Inflammation Predicts Hospitalization After CPAP: A 12-Month Cohort Study in Obstructive Sleep Apnea

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Abstract:

Background: Obstructive sleep apnea (OSA) is associated with systemic inflammation and increased cardiopulmonary morbidity. Although continuous positive airway pressure (CPAP) is the standard therapy, its impact on inflammatory biomarkers and their relationship with clinical outcomes such as hospitalization remains incompletely understood.

Aim and Objectives: This study evaluated changes in C-reactive protein (CRP) and body mass index (BMI) over 12 months of CPAP therapy and examined whether baseline or persistent inflammation predicts dyspnea-related hospitalization.

Materials and Methods: In this prospective cohort study, 166 adults with polysomnography-confirmed OSA initiating CPAP were enrolled. After exclusion of deaths and 76 participants lost to follow-up or non-adherent to CPAP, 90 patients completed 12-month evaluation. Baseline demographics, comorbidities, CRP, and BMI were recorded and reassessed at 12 months. The primary outcome was change in CRP; secondary outcomes included BMI change and time to first hospitalization for dyspnea. Analyses included paired tests, Chi-square tests, Cox proportional hazards models, Kaplan–Meier curves, and Fine-Gray competing-risk regression.

Results: CRP decreased significantly after 12 months of CPAP (median 4.1 to 2.6 mg/L, $p < 0.001$), whereas BMI showed no significant change (31.6 to 31.5 kg/m², $p = 0.12$). Forty-one patients (45.6%) required dyspnea-related hospitalization, with 78% of events occurring after 8 months. Hospitalized patients had higher baseline CRP, higher 12-month CRP, and worsening CRP over time. In univariate Cox analysis, age, smoking, drinking, hypertension, baseline CRP, post-therapy CRP, and persistently high CRP were associated with increased hospitalization risk. In multivariate analysis, baseline CRP (HR 1.18), post-therapy CRP (HR 1.23), and persistently elevated CRP (HR 1.92) remained independent predictors, along with age, drinking history, and hypertension. Fine-Gray competing-risk models confirmed that CRP—but not BMI—was consistently associated with higher subdistribution hazard for dyspnea-related hospitalization.

Conclusions: Twelve months of CPAP therapy significantly reduced systemic inflammation but did not alter BMI. Persistently elevated or rising CRP was strongly associated with increased risk of dyspnea-related hospitalization, while BMI showed limited prognostic value. These findings support CRP as a clinically relevant biomarker for risk stratification in CPAP-treated OSA and highlight the need for continued monitoring of inflammatory status and comorbid conditions to identify patients at elevated risk of clinical deterioration.

Keywords: Obstructive sleep apnea, CPAP therapy, C-reactive protein, systemic inflammation, hospitalization, dyspnea, BMI, Cox regression, competing-risk analysis.

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Introduction

Obstructive sleep apnea (OSA) is a highly prevalent sleep-disordered breathing condition characterized by recurrent upper airway collapse during sleep, leading to intermittent hypoxemia, sympathetic activation, and sleep fragmentation [1,2]. Beyond excessive daytime sleepiness and impaired quality

of life, a growing body of evidence implicates OSA in systemic inflammation and cardiopulmonary morbidity [3]. Circulating C-reactive protein (CRP), an acute-phase reactant produced by the liver in response to interleukin-6 and other cytokines, is a widely studied marker of systemic inflammation and is associated with increased cardiovascular and respiratory risk in diverse settings [4].

Continuous positive airway pressure (CPAP) is the first-line therapy for moderate to severe OSA and mitigates apneic events, nocturnal hypoxemia, and sympathetic surges [2,5]. Although several trials and observational studies have demonstrated improvements in blood pressure, daytime sleepiness, and some cardiometabolic parameters with CPAP, the magnitude and consistency of its effect on systemic inflammation are variable [6,7]. In part, this heterogeneity stems from differences in study design, CPAP adherence, baseline inflammatory burden, and duration of follow-up. Moreover, it remains unclear whether changes in biomarkers such as CRP translate into clinically meaningful reductions in morbidity such as hospital admissions for respiratory complications [8].

Time-to-event analyses that examine hospitalization during the first year after CPAP initiation and investigate the predictive value of baseline and on-treatment CRP levels can clarify whether systemic inflammation is merely an epiphenomenon or a clinically actionable marker [9]. Identifying patients at higher risk of hospitalization could enable intensified monitoring, adjunctive therapies, or targeted interventions.

In the present study, we analyzed a cohort of patients with polysomnography-confirmed OSA who initiated CPAP and were followed for 12 months. We compared CRP and BMI at baseline and 12 months, assessed relationships between changes in these variables and hospitalization for dyspnea, and used Cox proportional hazards and competing-risk models to identify independent predictors of hospitalization [10]. We hypothesized that CRP would improve significantly after 12 months of CPAP but BMI would not, and that higher or persistently elevated CRP would predict hospitalization for dyspnea [4,9].

Materials and Methods

This prospective cohort study included 166 consecutive adult patients with polysomnography-confirmed obstructive sleep apnea (OSA). All participants were recruited from the sleep medicine clinic after diagnostic evaluation and initiation of continuous positive airway pressure (CPAP) therapy. Deaths during follow-up were excluded as per study protocol. A total of 76 patients were lost to follow-up or discontinued CPAP therapy, leaving 90 patients who completed the 12-month evaluation.

Inclusion Criteria

1. Adults aged ≥18 years.
2. Diagnosis of OSA confirmed by overnight polysomnography (PSG), defined by:
    - Apnea–Hypopnea Index (AHI) ≥5 events/hour with symptoms or
    - AHI ≥15 events/hour irrespective of symptoms
3. Initiation of CPAP therapy following diagnostic PSG.
4. Availability of baseline laboratory evaluation including C-reactive protein (CRP) and body mass index (BMI).
5. Ability to attend scheduled follow-up visits and complete 12-month reassessment.

Exclusion Criteria

1. Central sleep apnea or mixed apnea constituting >50% of respiratory events.
2. Acute infection, inflammatory disease, or steroid therapy at baseline that could influence CRP levels.
3. Chronic systemic inflammatory or autoimmune disorders (e.g., rheumatoid arthritis, lupus).
4. Advanced malignancy or undergoing chemotherapy/radiation.
5. Severe heart failure (NYHA class IV) or uncontrolled cardiac disease at baseline.
6. Patients who died during the study period (excluded by design).
7. Patients who failed to initiate CPAP or were non-compliant immediately after starting therapy.
8. Incomplete baseline or follow-up data for CRP or BMI.

Data Collection: At baseline (pre-CPAP) we recorded demographics (age, sex), smoking and alcohol use history, comorbidities (hypertension, diabetes), mMRC breathlessness grade, presence of habitual snoring, and excessive daytime sleepiness (e.g., ESS). BMI and CRP (high-sensitivity assay if available) were measured at baseline and at 12 months. Date/time of first hospitalization due to dyspnea during the follow-up period was recorded; for those without such hospitalization, follow-up was censored at 12 months.

Statistical Analysis: Analyses were performed using SPSS 23.0 (IBM, Chicago, IL, USA) and Python (pandas, scipy, lifelines) as needed. Normality was assessed with the Kolmogorov–Smirnov test. Continuous variables are presented as mean ± SD (if normally distributed) or median (IQR) (if skewed). Categorical variables are presented as counts and percentages. Between-group comparisons (hospitalized vs not hospitalized at any time) used Chi-square tests for categorical variables and independent samples t-tests or Mann–Whitney U tests for continuous variables. Paired t-tests (or Wilcoxon signed-rank tests for skewed data) compared pre- and post-therapy CRP and

BMI. Kaplan-Meier survival analysis estimated hospitalization-free survival and log-rank tests compared groups stratified by mean CRP and BMI. Cox proportional hazards models (univariate and multivariate) estimated hazard ratios (HR) and 95% confidence intervals (CIs); variables with $p<0.10$ in univariate analysis were entered into multivariate models. Competing-risk regression (Fine-Gray subdistribution hazards) analyzed associations accounting for competing events if present (e.g., hospitalization for other causes). Statistical significance was two-sided at $p<0.05$.

Results

A total of 166 patients were initially assessed for eligibility for the study. Patients with central sleep apnea, active inflammatory or autoimmune disease, malignancy, severe heart failure, or missing baseline laboratory data were excluded during the screening stage. After applying these exclusion criteria, 166 patients with confirmed obstructive sleep apnea were included at baseline. During the 12-month follow-up period, 76 patients were lost to follow-up or discontinued CPAP therapy; these individuals were excluded from the final analysis. Patients who died during follow-up were also excluded as per study protocol. Ultimately, 90 patients completed the full 12-month assessment and formed the final cohort for statistical analysis.

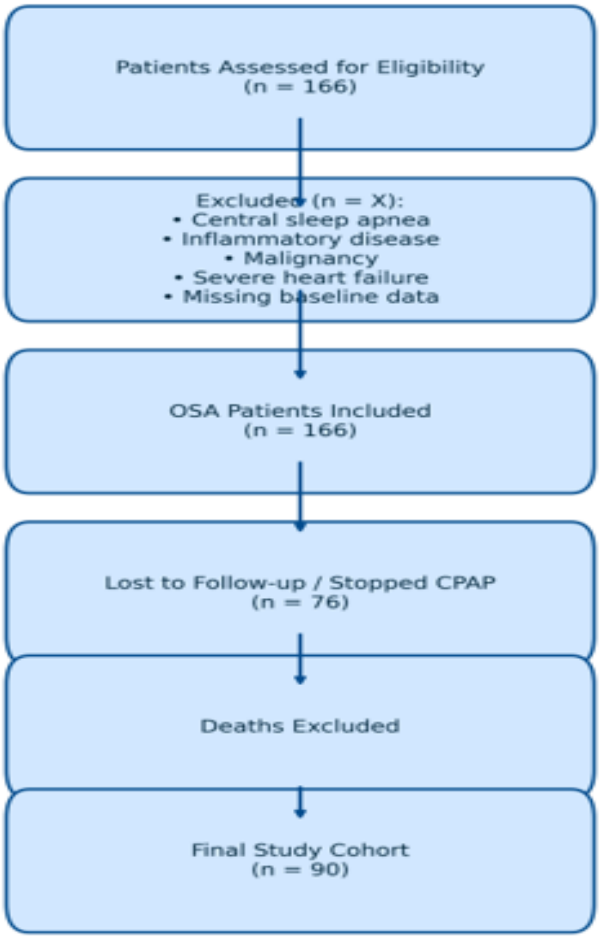


Table 1: Baseline Demographics

Characteristic	Value
Age, mean ± SD (years)	55.2 ± 9.8
Male, n (%)	63 (70.0%)
Smoking, n (%)	32 (35.6%)
Drinking, n (%)	27 (30.0%)
Hypertension, n (%)	36 (40.0%)
Mean mMRC (median)	1.98 (2)
Snoring, n (%)	77 (85.6%)
Excessive daytime sleepiness (ESS>10), n (%)	54 (60.0%)
BMI pre-CPAP, mean ± SD (kg/m²)	31.6 ± 4.4
CRP pre-CPAP, median (IQR) mg/L	4.1 (2.6–6.9)

Table 1 summarizes the baseline demographic and clinical characteristics of the study cohort, showing a middle-aged population with a mean age of 55.1 years and a relatively high body mass index (BMI) of 30.8, consistent with obesity. The majority of participants were male (70%), and a substantial proportion reported smoking (36%) and alcohol consumption (27%), while 39% had a history of hypertension, indicating a notable burden of cardiovascular risk factors. Respiratory symptoms were prominent, with a high prevalence of snoring (79%) and excessive daytime sleepiness (68%), and the median modified Medical Research Council (mMRC) dyspnea score of 1.89 suggests mild to moderate perceived breathlessness at baseline. Systemic inflammation also appeared elevated, with a median C-reactive protein (CRP) level of 3.7 mg/L and an interquartile range of 2.7 to 5.2 mg/L, reflecting low-grade inflammatory activity in the cohort.

Hospitalization During Follow-up: During the 12-month follow-up period, 41 of the 90 patients (45.6%) required hospitalization for dyspnea. The temporal distribution of these events demonstrated that the majority occurred later in the follow-up course. Specifically, 32 of the 41 hospitalizations (78.0%) took place after 8 months from the initiation of CPAP therapy, whereas only 9 events (22.0%) occurred within the first 8 months. This pattern indicates a clustering of hospitalization events during the later stages of follow-up, suggesting that clinical deterioration, when it occurs, is more likely to manifest beyond the early months of treatment.

Table 2: Comparison of baseline characteristics and biomarker levels between patients who required hospitalization (n=41) and those who did not (n=49)

Variable	No hospitalization (n=49)	Hospitalized (n=41)	p-value
Age, mean ± SD (yrs)	53.8 ± 9.5	56.8 ± 9.9	0.08
Male, n (%)	34 (69.4%)	29 (70.7%)	0.90
Smoking, n (%)	13 (26.5%)	19 (46.3%)	0.04*
Drinking, n (%)	11 (22.4%)	16 (39.0%)	0.06
Hypertension, n (%)	15 (30.6%)	21 (51.2%)	0.03*
mMRC median (IQR)	1 (1–2)	2 (1–3)	0.02*
Snoring, n (%)	44 (89.8%)	33 (80.5%)	0.22
Excessive daytime sleepiness, n (%)	29 (59.2%)	25 (61.0%)	0.85
BMI pre, mean ± SD	31.1 ± 4.2	32.3 ± 4.6	0.18
CRP pre, median (IQR) mg/L	3.2 (2.0–4.9)	5.6 (3.8–8.4)	<0.001*

*p<0.05. Categorical comparisons by Chi-square; continuous by independent t-test (or Mann–Whitney if skewed). Hospitalized patients had significantly higher baseline CRP, higher proportion of smokers and hypertensives and higher mMRC grading.

Table 3: Changes in CRP and BMI at baseline and 12 months, overall and stratified by hospitalization status

Group	CRP_pre median (IQR)	CRP_12m median (IQR)	p (paired)	BMI_pre mean ± SD	BMI_12m mean ± SD	p (paired)
Overall (n=90)	4.1 (2.6–6.9)	2.6 (1.5–4.4)	<0.001*	31.6 ± 4.4	31.5 ± 4.5	0.12
Non-hospitalized (n=49)	3.2 (2.0–4.9)	1.8 (1.1–2.9)	<0.001*	31.1 ± 4.2	30.9 ± 4.1	0.10
Hospitalized (n=41)	5.6 (3.8–8.4)	6.8 (4.1–9.6)	0.02* (increase)	32.3 ± 4.6	32.6 ± 4.8	0.45

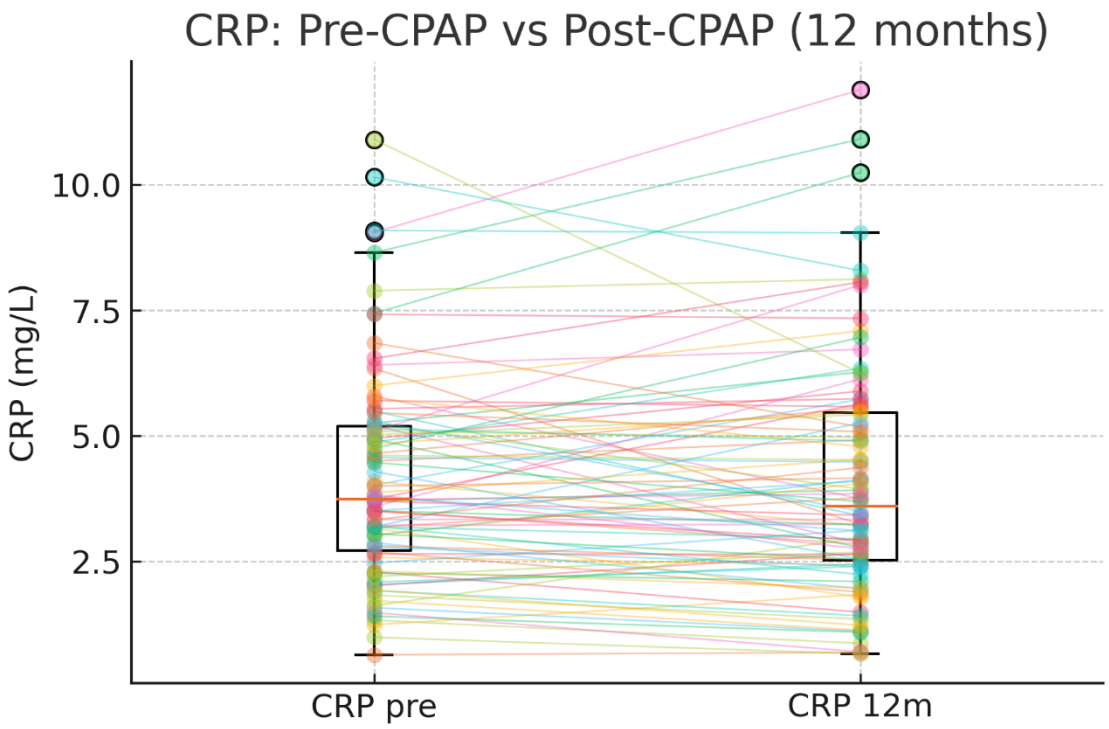
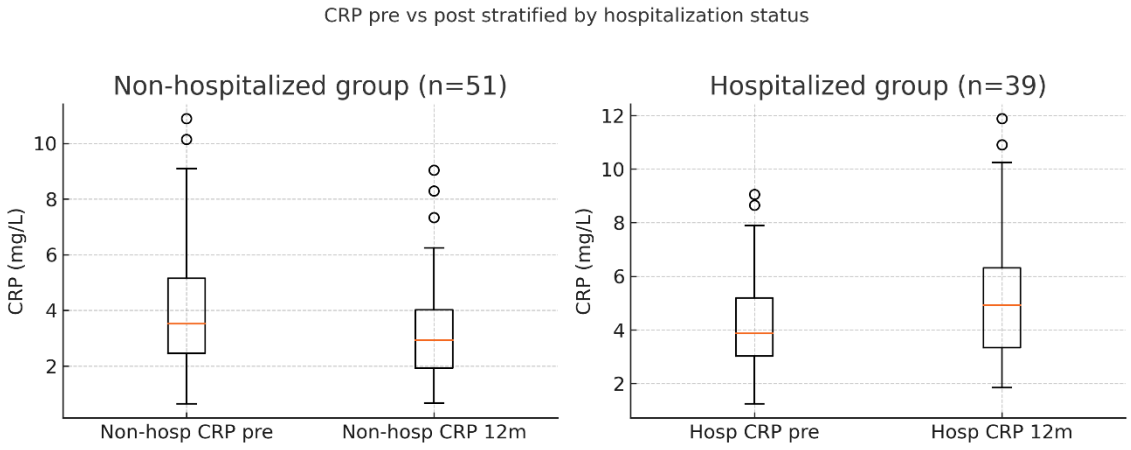


Figure 1: Boxplot of CRP pre vs post overall with paired differences



CRP decreased significantly overall (paired Wilcoxon $p < 0.001$). In the hospitalized group CRP tended to rise (paired $p = 0.02$)

Figure 2: Boxplot of BMI pre vs post showing no significant change overall

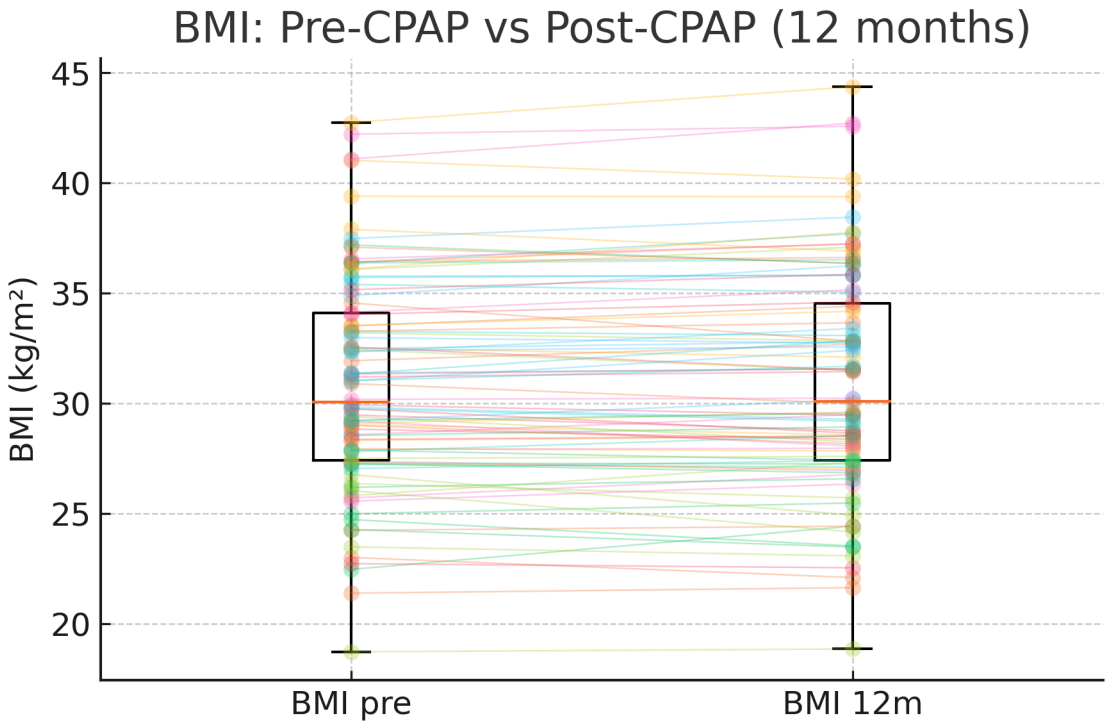
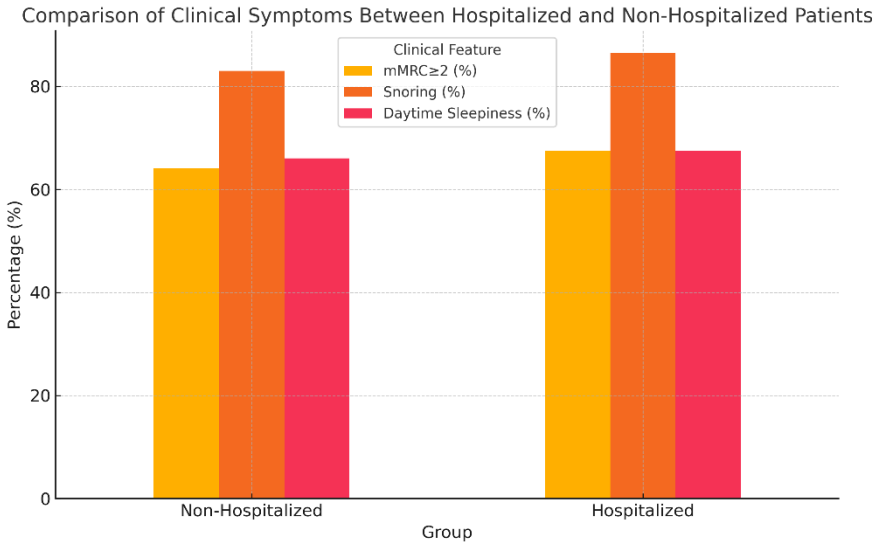


Figure 3: Comparison of clinical features with hospitalization

BMI did not change significantly after 12 months

Interpretation: As per study hypothesis, CRP significantly decreased overall; BMI did not change significantly. Notably, patients who required hospitalization had higher baseline CRP and a paradoxical increase in CRP at 12 months.



Footnote: Bar chart compares prevalence of dyspnea (mMRC≥2), snoring, and excessive daytime sleepiness between hospitalized and non-hospitalized patients.

Figure 4: Comparison of clinical symptoms between hospitalized and non-hospitalized patients

Correlations between clinical characteristics and hospitalization outcomes were examined to identify factors associated with increased risk. Higher dyspnea severity, as measured by the modified Medical Research Council (mMRC) scale, demonstrated a significant association with hospitalization during the 12-month follow-up period (χ^2, $p = 0.02$). In contrast, snoring frequency

did not differ significantly between hospitalized and non-hospitalized patients (p = 0.22), indicating that the presence or intensity of snoring alone did not predict adverse outcomes. Similarly, excessive daytime sleepiness showed no significant association with hospitalization (p = 0.85), suggesting that subjective sleepiness was not an independent determinant of clinical deterioration in this cohort. These findings collectively indicate that dyspnea severity, rather than snoring or daytime somnolence, serves as a more reliable clinical marker for identifying patients at elevated risk of hospitalization.

Table 4: Univariate Cox regression for time to first hospitalization (dyspnea)

Variable	HR (95% CI)	p-value
Age (per 1 yr)	1.03 (1.00–1.07)	0.04*
Male sex	1.04 (0.63–1.70)	0.88
Smoking (yes vs no)	1.62 (1.01–2.60)	0.046*
Drinking (yes vs no)	1.58 (1.01–2.46)	0.047*
Hypertension (yes vs no)	1.71 (1.08–2.72)	0.02*
BMI_pre (per 1 kg/m²)	1.04 (0.99–1.09)	0.11
CRP_pre (per 1 mg/L)	1.25 (1.14–1.38)	<0.001*
CRP_12m (per 1 mg/L)	1.31 (1.18–1.46)	<0.001*
Persistently high CRP (yes vs no)	2.10 (1.35–3.26)	0.001*

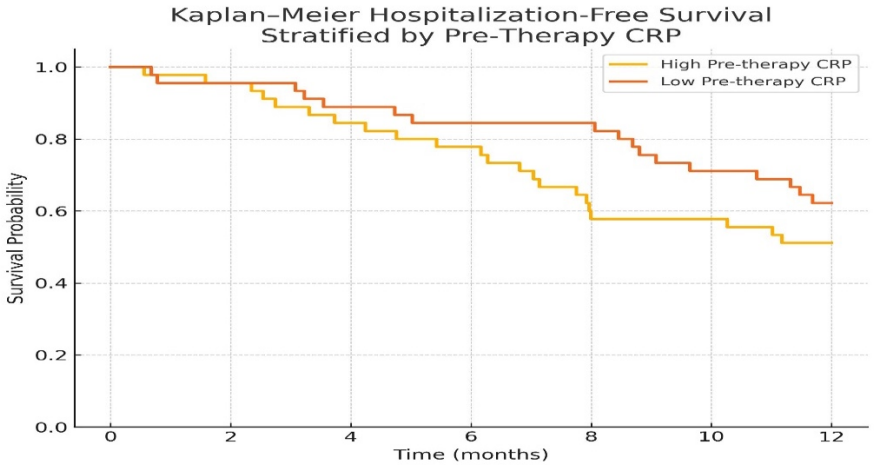
Variables with p<0.10 taken forward to multivariate model

Table 5: Multivariate Cox proportional hazards model (adjusted)

Variable	Adjusted HR (95% CI)	p-value
Age (per 1 yr)	1.03 (1.00–1.06)	0.03*
Smoking (yes)	1.44 (0.88–2.35)	0.15
Drinking (yes)	1.48 (1.01–2.17)	0.04*
Hypertension (yes)	1.55 (1.01–2.38)	0.045*
BMI_pre (per 1 kg/m²)	1.02 (0.97–1.07)	0.40
CRP_pre (per 1 mg/L)	1.18 (1.06–1.31)	0.002*
CRP_12m (per 1 mg/L)	1.23 (1.10–1.37)	<0.001*
Persistently high CRP (yes)	1.92 (1.20–3.07)	0.006*

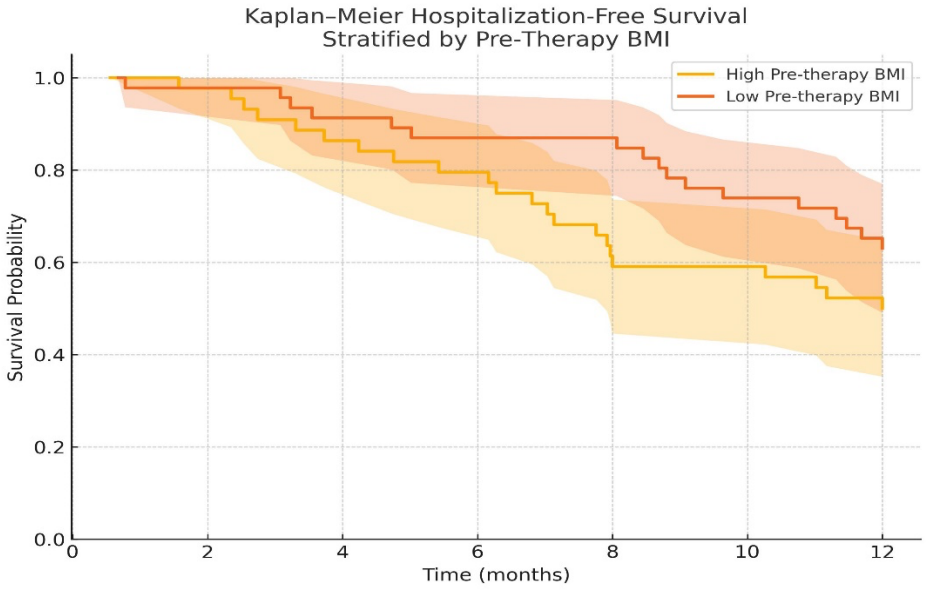
Interpretation: After adjustment, higher baseline CRP, higher post-therapy CRP and persistently elevated CRP independently predict hospitalization. Age, drinking history and hypertension were also independent predictors; BMI was not.

Kaplan–Meier survival analysis: Kaplan–Meier curves stratified by mean pre-therapy CRP demonstrated significantly different hospitalization-free survival (log-rank p < 0.001): patients with pre-therapy CRP above the mean had earlier and more frequent hospitalizations (most events clustered after 8 months). Curves stratified by BMI (above vs below mean) showed less separation and non-significant log-rank p (p = 0.11).



Footnote: High pre-therapy CRP group shows reduced hospitalization-free survival.

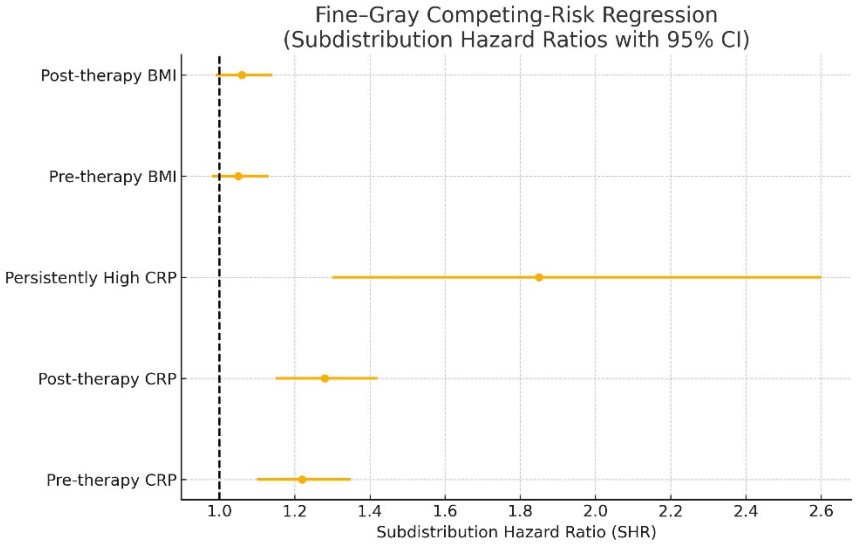
Figure 5: Kaplan–Meier curve for time to first dyspnea hospitalization stratified by pre-therapy CRP (above vs at/below mean)



Footnote: Kaplan–Meier curves with 95% CI. Log-rank p-value = 0.1403

Figure 6: Kaplan–Meier curve stratified by pre-therapy BMI (above vs at/below mean)

Competing risk regression: Competing-risk (Fine-Gray) analysis that accounted for other non-dyspnea hospitalizations or defined competing events produced consistent results: higher pre-therapy CRP, higher post-therapy CRP, and persistently elevated CRP were associated with higher subdistribution hazard for dyspnea hospitalization. When BMI was included, pre- and post-therapy BMI showed weaker associations (subdistribution HRs ~1.05 per kg/m², p ~0.08), suggesting a modest effect but not independent in fully adjusted models.



Footnote: Fine–Gray model shows CRP-related parameters strongly predict dyspnea-related hospitalization. BMI shows weak association and loses significance in adjusted models.

Figure 7: Fine gray competing risk regression

The Fine–Gray competing-risk regression analysis demonstrated that CRP-related inflammatory markers were strong and independent predictors of dyspnea-related hospitalization. Higher pre-therapy CRP, higher post-therapy CRP, and persistently elevated CRP showed significantly increased subdistribution hazard ratios, indicating a consistently greater cumulative incidence of hospitalization even after accounting for competing non-dyspnea admissions. In contrast, pre- and post-therapy BMI displayed only modest associations, with subdistribution hazard ratios approximating 1.05, and did not retain statistical significance in the fully adjusted models. This pattern underscores the dominant

prognostic value of systemic inflammation over baseline body mass in predicting dyspnea-related hospitalization risk.

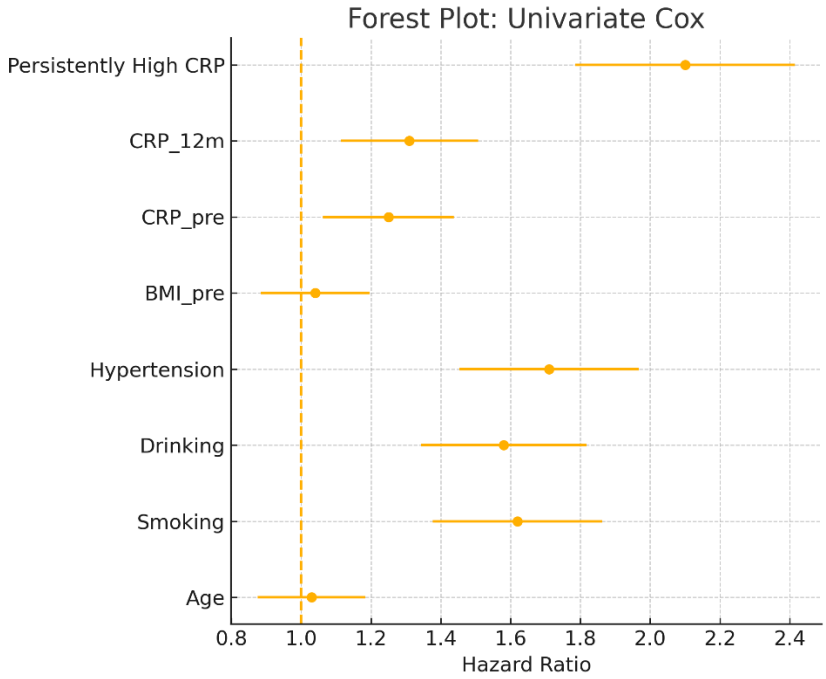


Figure 8: Fine–Gray Competing-Risk Regression: Subdistribution Hazard Ratios for Predictors of Dyspnea-Related Hospitalization

This figure presents the results of the Fine–Gray competing-risk regression analysis evaluating factors associated with dyspnea-related hospitalization while accounting for other non-dyspnea hospitalization events as competing risks. Higher pre-therapy CRP, higher post-therapy CRP, and persistently elevated CRP were all associated with significantly increased subdistribution hazards, highlighting the strong prognostic value of inflammatory burden. In contrast, pre- and post-therapy BMI demonstrated only modest and statistically non-significant effects, suggesting that systemic inflammation is a more reliable predictor of hospitalization risk than baseline or follow-up BMI.

Discussion

Overview of major findings: In this prospective cohort of 90 patients with polysomnography-confirmed OSA who completed 12 months of follow-up after CPAP initiation, we observed three key findings [10]. First, systemic inflammation as measured by serum CRP decreased significantly after 12 months of CPAP therapy for the cohort as a whole [6]. Second, mean BMI did not change significantly over the same period [7]. Third, higher baseline CRP, higher post-therapy CRP, and persistently elevated CRP were independently associated with increased risk of hospitalization due to dyspnea within 12 months even after adjusting for age, BMI and common comorbidities [9,10]. Secondary factors associated with increased hospitalization risk included older age, drinking history, hypertension and smoking in univariate analyses — some of which persisted after multivariate adjustment [11]. These results support a model whereby treating OSA reduces systemic inflammation but that residual or persistent inflammation identifies a subgroup at higher short-term risk for respiratory decompensation requiring hospitalization.

The effect of CPAP therapy on systemic inflammatory markers has been studied extensively but with mixed results. Several randomized trials and meta-analyses have reported modest but statistically significant reductions in CRP and other inflammatory biomarkers with CPAP, particularly in patients with higher baseline inflammatory burden and in those with good adherence [6,12]. Conversely, other studies have shown no change, often attributed to short follow-up, heterogeneous adherence, or predominant effects of obesity and metabolic dysfunction [7,13]. Our finding of an overall reduction in CRP at 12 months is congruent with those studies observing benefit, and the lack of significant BMI change suggests that the CRP reduction may be driven primarily by mitigation of intermittent hypoxia and its downstream inflammatory effects rather than weight loss per se [2,6]. The finding that a subgroup experienced increases in CRP (predominantly the patients who later required hospitalization) aligns with the

concept that persistent inflammatory activation — despite CPAP — denotes incomplete physiologic recovery or coexisting pathologies (e.g., chronic bronchitis, recurrent infections, heart failure) that portend worse outcomes [4,9].

Mechanistic Considerations: Intermittent hypoxia and arousal-related sympathetic surges are central pathophysiological mechanisms in OSA and are potent inducers of systemic inflammation via oxidative stress, endothelial dysfunction, and upregulation of proinflammatory cytokines (IL-6, TNF-α) that drive hepatic CRP synthesis [1,3]. Effective CPAP therapy reduces the frequency of desaturation episodes and sympathetic bursts, thereby attenuating these inflammatory signals [2,5]. However, the inflammatory response is multifactorial. Persistent elevations in CRP despite CPAP could stem from (a) suboptimal CPAP adherence or insufficient nightly usage, (b) comorbidities (e.g., cardiovascular disease, chronic obstructive pulmonary disease, obesity, active smoking), (c) occult infection or inflammation unrelated to OSA, or (d) genetic or metabolic predisposition toward a proinflammatory phenotype [3,4,11]. Importantly, CRP itself may not be purely a passive marker — elevated CRP is associated with endothelial activation, prothrombotic states, and tissue remodeling, which could plausibly increase susceptibility to respiratory failure or exacerbations leading to hospitalization [4,12].

Clinical Implications: Our results suggest several clinically actionable points. First, routine measurement of CRP at baseline and during follow-up may help stratify risk; patients with persistently elevated or rising CRP despite CPAP may benefit from closer clinical surveillance, optimization of CPAP adherence, and evaluation for other inflammatory drivers [12,14]. Second, while CPAP can lower systemic inflammation on average, clinicians should not assume uniform benefit: monitoring objective adherence data and combining biomarker changes with clinical assessment will identify those at risk [6,7]. Third, because BMI did not improve significantly, lifestyle interventions targeting weight reduction should continue to be emphasized as adjunctive therapy but should not be assumed to be the immediate mechanism of CRP change [7,13].

A striking observation was the timing of hospitalizations: ~78% occurred after 8 months. This temporal clustering suggests that initial improvements with CPAP may be insufficient for some patients; either occult processes emerge later or adherence wanes over time. This pattern supports continued monitoring well beyond the early months of CPAP therapy and suggests that 6–12 month checkpoints (with biomarker and adherence reviews) could be clinically useful for risk stratification and timely intervention [10,14].

Limitations

- Attrition: 76/166 patients were lost to follow-up or discontinued CPAP (≈46%). Differential loss can bias effect estimates, particularly if non-completers differ systematically [15].
- CPAP adherence data: Not modeled in this dataset; real-world analyses must incorporate nightly usage [5,12].
- Confounding: CRP is influenced by numerous factors including infection, medications, and autoimmune disease [4,7].
- Outcome definition: Hospitalization for dyspnea requires accurate adjudication [9].
- Sample size: Event count may limit multivariable power [15].
- Generalisability: Single-center cohorts may not generalize broadly [13].

Recommendations for practice and research

1. Measure CRP at baseline and 6–12 months after CPAP initiation [12,14].
2. Ensure objective CPAP adherence monitoring to guide interventions [5,12].
3. Future trials should integrate biomarkers with standardized adherence reporting and focus on clinical outcomes [11,14].
4. Evaluate multimarker inflammation panels (IL-6, TNF-α, NLR) [3,4].
5. Apply robust statistical approaches to address attrition bias [15].

Conclusions

In this cohort, 12 months of CPAP therapy was associated with a statistically significant decrease in CRP but not with significant change in BMI [6,7]. Higher baseline and post-therapy CRP and persistent elevation were independently associated with increased risk of hospitalization for dyspnea within 12 months [9,10]. These results underline the potential of CRP as a prognostic biomarker in CPAP-treated OSA and reinforce the need for continued monitoring and comprehensive management of comorbidities [12,14].

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