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KIDNEY DYSFUNCTION IN COPD WITH COMORBID ARTERIAL HYPERTENSION: BIOMARKERS, CLINICAL PATTERNS, AND TREATMENT OPTIMIZATION

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a leading global cause of disability and mortality, increasingly recognized as a systemic condition with multiorgan involvement. When complicated by arterial hypertension (AH), COPD patients face substantially elevated risk of renal dysfunction, yet this triad remains underinvestigated.

Objective: To assess kidney dysfunction across COPD severity stages with and without comorbid AH, evaluate renal and vascular biomarkers, and investigate the efficacy of individualized therapy with Telsartan A (telmisartan + amlodipine) and eplerenone.

Methods: 291 COPD+AH patients and 66 COPD-only controls were examined with pulmonary function tests, 24-hour blood pressure monitoring, and biomarker panels (cystatin-C, eGFR, aldosterone, E-selectin, type IV collagen, TNF-α, CRP).

Results: COPD severity inversely correlated with eGFR and directly with all inflammatory and fibrotic markers; AH significantly worsened all renal

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parameters. Combined Telsartan A + eplerenone therapy improved FEV_1, eGFR, and reduced fibrotic/inflammatory markers.

Conclusions: AH accelerates renal dysfunction in COPD. Early aldosterone, type IV collagen, and E-selectin measurement enables subclinical nephropathy detection. Telsartan A + eplerenone provides meaningful renoprotective benefit.

Keywords: COPD, arterial hypertension, chronic kidney disease, eGFR, E-selectin, type IV collagen, aldosterone, telmisartan, eplerenone, comorbidity

Introduction

Chronic obstructive pulmonary disease (COPD) represents a major global public health challenge. Approximately 2.8 million people die from COPD annually, and WHO projections indicate it will rank among the top three leading causes of death by 2030 alongside cardiovascular and cerebrovascular diseases [**1,2**]. Population-based studies report COPD prevalence of up to 15.3% in Russian and Central Asian populations, with high rates of severe-stage disease requiring hospitalization [**3,4**].

Contemporary evidence has firmly established COPD not merely as a pulmonary condition but as a systemic disease. Structural and functional abnormalities in the bronchi, bronchioles, and alveoli trigger systemic chronic inflammation, driving endothelial dysfunction, microcirculatory disturbances, and multiorgan damage [**5,6,7**]. A large meta-analysis found that COPD patients carry a 2.46-fold higher risk of cardiovascular disease versus the general population [**11**].

Among the most clinically significant comorbidities, arterial hypertension (AH) frequently coexists with COPD. Studies report albuminuria in 46.7% of hypertensive patients alone, rising to 77.5% when COPD is also present, and the albumin-to-creatinine ratio is significantly higher in COPD+AH patients (65%) compared to AH alone (36.7%, p=0.029) [**30**]. Despite this evidence, early renal dysfunction in COPD-AH comorbidity remains underdiagnosed because its

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manifestations are masked by dominant respiratory and cardiovascular symptoms [**20,21**].

The pathophysiological link involves convergent mechanisms: systemic inflammation (TNF-α, IL-6, CRP) [**16,17**], oxidative stress, renin-angiotensin-aldosterone system (RAAS) overactivation, endothelial dysfunction (E-selectin elevation), progressive interstitial fibrosis (type IV collagen accumulation), and chronic hypoxemia-driven hemodynamic changes [**8,9,10**]. This study aimed to comprehensively characterize kidney dysfunction across COPD severity stages and evaluate optimized treatment with Telsartan A (telmisartan + amlodipine) and eplerenone [**34,35**].

3. Materials and Methods

3.1 Patient Population

The main group comprised 291 patients with COPD + arterial hypertension; the control group comprised 66 patients with COPD without AH. Patients were stratified by GOLD 2024 into severity stages II–IV (stage I excluded as non-hospitalized) [**1**] and by clinical COPD phenotype: bronchitic (n=117+22), emphysematous (n=55+15), and mixed (n=119+29). Stage II COPD+AH: n=18, mean age 62.5±2.7 years. Stage III: n=92, mean age 67.2±0.8 years. Stage IV: n=181, mean age 68.4±0.7 years.

3.2 Investigations

All patients underwent: pulmonary function testing (SpO_2, FEV_1%, Tiffeneau index); 24-hour ambulatory BP monitoring (circadian profile classification as "dipper," "non-dipper," "night-peaker," or "over-dipper"); biochemical renal markers (cystatin-C, eGFR, creatinine) [**27,28**]; and inflammatory/fibrotic biomarker panels — serum aldosterone, type IV collagen (Elabscience COL4A1 ELISA Kit), E-selectin/CD62E (R&D Systems Quantikine ELISA), TNF-α (Mindray MR-96A), and CRP [**16,23,25**]. Statistical analyses used SPSS 26.0;

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group comparisons applied Mann-Whitney U or Kruskal-Wallis tests; categorical variables used Chi-square or Fisher tests; correlations were assessed by Pearson coefficient.

3.3 Treatment Protocol

All COPD+AH patients received individualized standard COPD therapy (per GOLD 2024 [**1**]) supplemented with Telsartan A (telmisartan 40–80 mg + amlodipine 5–10 mg) titrated to blood pressure targets, and eplerenone 25–50 mg/day for its anti-fibrotic and mineralocorticoid-blocking properties [**34,35,38**]. Parameters were re-evaluated after the full treatment course.

4. Results

4.1 Pulmonary Function

FEV_1 (% predicted) was significantly lower in COPD+AH patients at every severity stage compared to COPD-only controls: stage II (60.3±0.7% vs. 66.7±1.05%, $p<0.001$); stage III (34.5±0.4% vs. 41.1±1.02%, $p<0.001$); stage IV (20.7±0.26% vs. 27.1±3.2%, $p<0.05$). The Tiffeneau index mirrored this pattern at stages III and IV ($p<0.001$), confirming that comorbid AH worsens obstructive ventilatory impairment beyond COPD severity alone [**33**]. Across COPD clinical phenotypes (bronchitic, emphysematous, mixed), SpO_2, FEV_1, and FEV_1/FVC did not differ between AH and non-AH subgroups (all $p>0.05$), indicating phenotype-specific ventilatory patterns are driven by COPD pathophysiology rather than blood pressure status.

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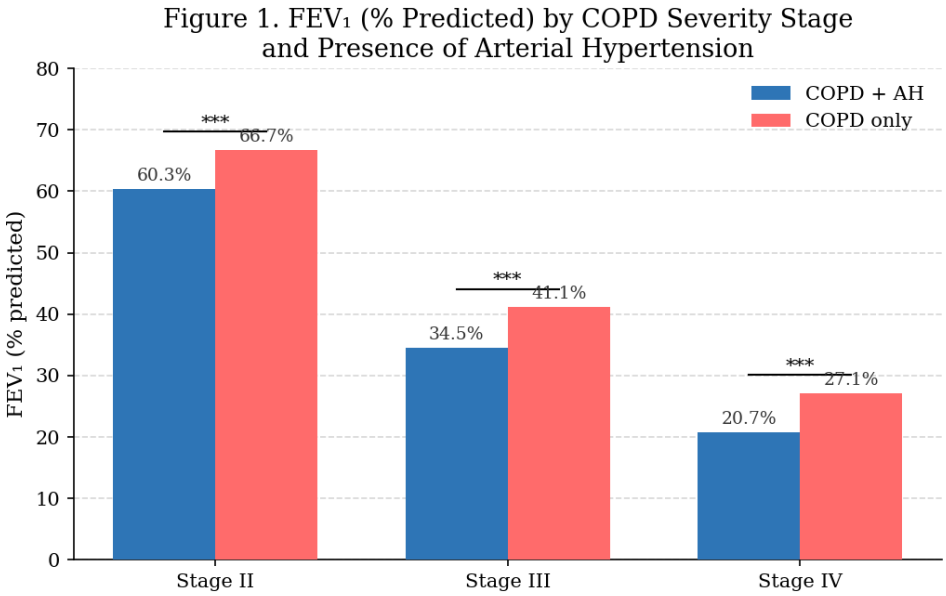

Figure 1. FEV₁ (% Predicted) by COPD Severity Stage and Presence of Arterial Hypertension (*** p<0.001)

4.2 Circadian Blood Pressure Profiles

24-hour blood pressure monitoring revealed progressive deterioration of circadian dipping with increasing COPD severity. The protective "dipper" phenotype was present in 61.1% (stage II), 15.2% (stage III), and only 11% (stage IV) of COPD+AH patients ($p<0.001$ between stages II–III and II–IV). The pathological "non-dipper" phenotype rose from 27.8% to 42.4% to 66.3% across stages (all inter-stage comparisons $p<0.05$ to $p<0.001$). The "night-peaker" profile, carrying the highest cardiovascular and renal risk, was present in 11.1%, 42.4%, and 22.7% across stages II, III, and IV, respectively. These findings demonstrate that progressive COPD severity disrupts normal nocturnal blood pressure dipping, amplifying sustained hemodynamic target-organ stress.

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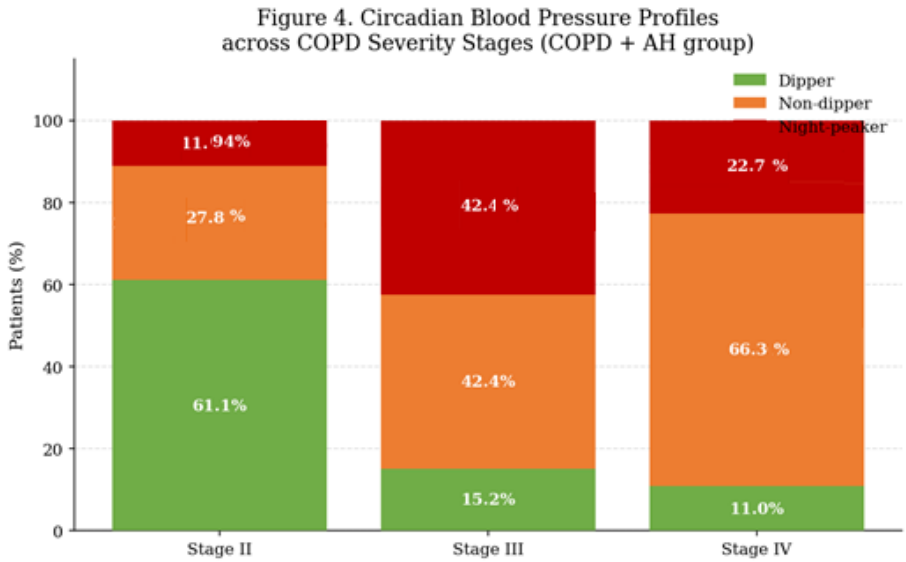

Figure 2. Circadian Blood Pressure Profiles across COPD Severity Stages (COPD + AH group)

4.3 Renal Function Parameters

Cystatin-C increased progressively with COPD severity and was significantly higher in COPD+AH vs. COPD-only at all stages: stage II (1.42±0.02 vs. 1.2±0.04 mg/L, $p<0.001$); stage III (1.74±0.01 vs. 1.53±0.04 mg/L, $p<0.001$); stage IV (2.02±0.02 vs. 1.88±0.03 mg/L, $p<0.001$). Corresponding eGFR was significantly lower in COPD+AH patients across all stages — notably reaching values below the chronic kidney disease threshold of 60 ml/min/1.73 m² at stages III and IV [**31**]. Serum creatinine only became significantly elevated at stage IV (115±0.87 vs. 108±1.7 µmol/L, $p<0.001$), underscoring cystatin-C's superiority as an early renal dysfunction marker [**27,28**]. The FEV_1–eGFR correlation weakened progressively across stages ($r=0.43 \rightarrow 0.30 \rightarrow 0.29$), reflecting the growing influence of hypoxemic, hemodynamic, and inflammatory factors on nephropathy at advanced stages [**20**].

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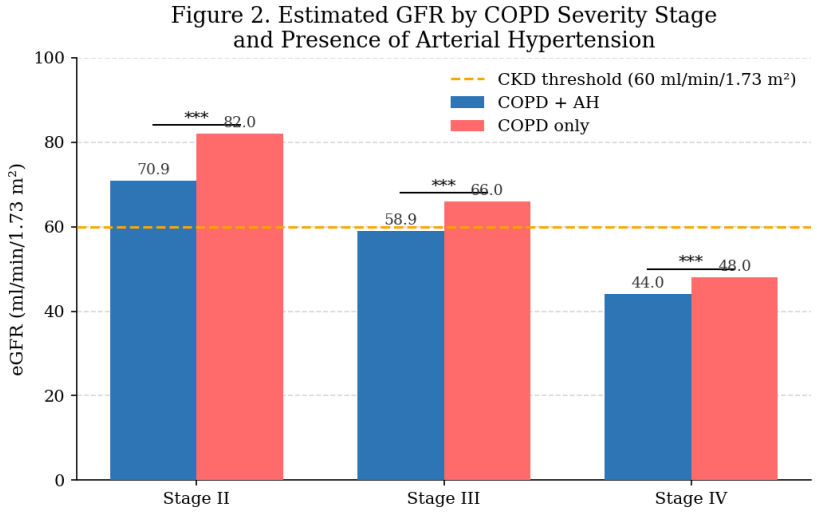

Figure 3. Estimated GFR by COPD Severity Stage and Presence of Arterial Hypertension. Orange dashed line = CKD threshold (60 ml/min/1.73 m²).

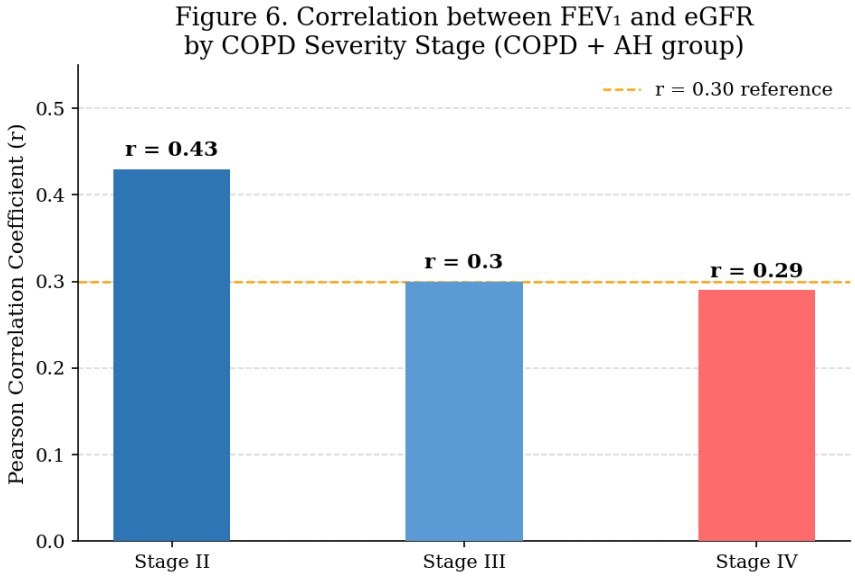

Figure 4. Pearson Correlation Coefficient (r) between FEV₁ and eGFR by COPD Severity Stage (COPD + AH group), showing progressive weakening with disease advancement.

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4.4 Inflammatory and Fibrotic Biomarkers

Aldosterone, type IV collagen, E-selectin, and TNF-α all rose with COPD severity and were significantly higher in COPD+AH than COPD-only at every stage (all $p<0.001$). Aldosterone levels at stage IV reached 315±3.08 pg/mL in the COPD+AH group vs. 290±5.1 pg/mL in controls, confirming intense RAAS activation driving both hypertension and renal fibrogenesis [**32**]. Type IV collagen peaked at 218±1.93 ng/mL (COPD+AH, stage IV), reflecting accelerating nephrosclerosis. E-selectin at 92±0.83 ng/mL (COPD+AH, stage IV) confirms compound vascular endothelial inflammation [**11,17**]. TNF-α at 14.8±0.15 pg/mL (stage IV, COPD+AH) confirms sustained systemic inflammatory burden [**16,18**].

Figure 3. Key Biomarker Levels by COPD Severity Stage and Presence of Arterial Hypertension

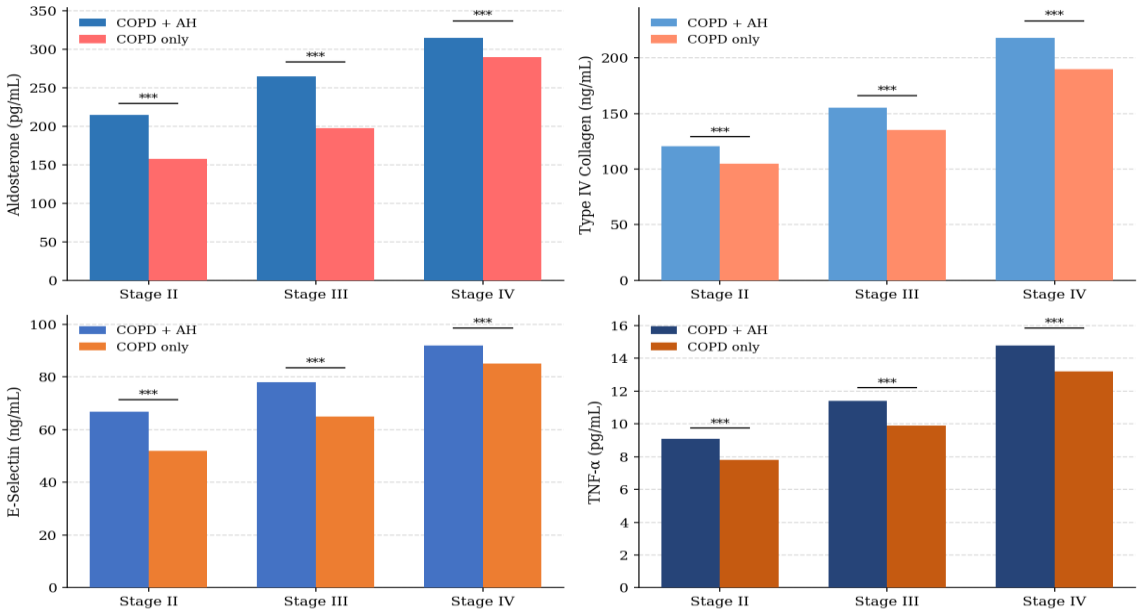

Figure 5. Key Biomarker Levels (Aldosterone, Type IV Collagen, E-Selectin, TNF-α) by COPD Severity Stage and Presence of Arterial Hypertension (all inter-group comparisons $p<0.001$).

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5. Treatment Outcomes

Individualized complex treatment with Telsartan A and eplerenone produced clinically and statistically significant improvements across all three COPD severity stages [**34,35,38**]. FEV_1 improved at stage II (60.3±0.7% → 63.8±0.8%, $p<0.01$), stage III (34.5±0.4% → 37.3±0.4%, $p<0.001$), and stage IV (20.7±0.26% → 22.3±0.3%, $p<0.001$). eGFR improved at stage II (70.1±1.4 → 78.9±1.5 ml/min/1.73 m², $p<0.01$), stage III (58.9±0.7 → 68.2±0.8, $p<0.001$), and stage IV (44±0.3 → 49.7±0.4, $p<0.001$). Type IV collagen decreased significantly at all stages, with the most pronounced reduction at stage IV (218.5±1.9 → 196.6±1.7 ng/mL, $p<0.001$). TNF-α also fell significantly at every stage (stage IV: 14.8±0.1 → 12.6±0.1 pg/mL, $p<0.001$).

Figure 5. Changes in Key Parameters Before and After Treatment with Telsartan A + Eplerenone

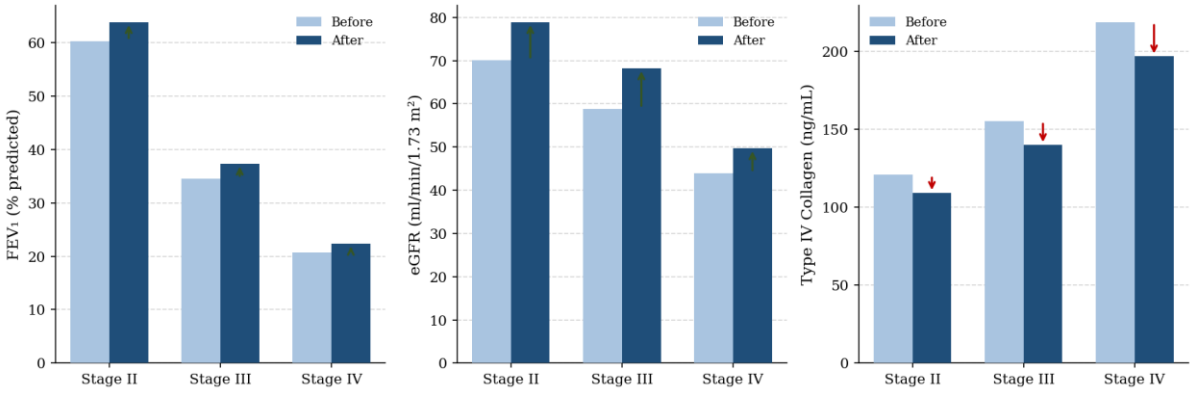

Figure 6. FEV_1, eGFR, and Type IV Collagen Before and After Treatment with Telsartan A + Eplerenone across COPD Severity Stages.

6. Discussion

This study provides comprehensive evidence that comorbid arterial hypertension substantially accelerates renal dysfunction in COPD patients through converging pathophysiological mechanisms. COPD-driven systemic inflammation —

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evidenced by markedly elevated TNF-α, CRP, and E-selectin — extends injurious effects to the renal microvasculature, promoting glomerular barrier disruption and nephrosclerosis [**16,17,18**]. The compounding of AH further amplifies E-selectin levels, confirming synergistic vascular endothelial activation.

RAAS overactivation plays a central role [**32**]. Aldosterone elevation was proportional to COPD severity and further amplified by AH, driving sodium retention, oxidative stress, and direct cardiac and renal fibrosis via type IV collagen deposition in basement membranes. Significant reductions in both aldosterone and type IV collagen following eplerenone therapy confirm the causal role of mineralocorticoid receptor activation in this fibrogenic pathway [**34,38**].

The progressive loss of nocturnal blood pressure dipping — with "non-dipper" and "night-peaker" phenotypes dominating in advanced COPD stages — sustains 24-hour hemodynamic load on the kidneys, eliminating overnight reparative phases and compounding inflammatory nephropathy. This circadian disruption represents an independent mechanism of renal injury superimposed on the inflammatory and fibrotic pathways.

Cystatin-C proved superior to creatinine as an early GFR marker [**27,28**], detecting significant glomerular decline at stages II and III when creatinine remained within acceptable limits. The weakening FEV_1–eGFR correlation across stages reflects the progressive shift from ventilation-driven to hemodynamic- and inflammation-driven nephropathy at advanced disease [**20,24**] — an important consideration for clinical staging and treatment intensity decisions.

The treatment results support combining RAAS blockade (telmisartan/eplerenone) with calcium channel antagonism (amlodipine) [**34,35**]. Telmisartan's ARB action with PPAR-γ agonism provides anti-inflammatory effects beyond simple RAAS blockade. Eplerenone's selective mineralocorticoid receptor blockade directly targets aldosterone-driven fibrogenesis identified in this study [**38**]. Together, these agents address multiple pathophysiological nodes

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simultaneously, explaining the broad-based improvements observed across pulmonary, renal, inflammatory, and fibrotic parameters [**34,35,38**].

7. Conclusions

Based on this research, the following principal conclusions are drawn:

1. Comorbid arterial hypertension significantly worsens FEV_1 at all COPD severity stages (stage II: 60.3±0.7% vs. 66.7±1.05%; stage III: 34.5±0.4% vs. 41.1±1.02%; stage IV: 20.7±0.26% vs. 27.1±3.2%; all $p \leq 0.05$).
2. eGFR is significantly reduced and E-selectin significantly elevated in COPD+AH across all stages (stage II: 66.8±2.2 vs. 52.02±2.5 ng/mL; stage IV: 92±0.83 vs. 84.99±1.9 ng/mL; all $p<0.001$), confirming compound endothelial dysfunction and nephrosclerosis.
3. Aldosterone, TNF-α, and type IV collagen rise progressively with COPD severity and are significantly higher in COPD+AH, confirming additive RAAS activation, systemic inflammation, and fibrosis as drivers of kidney damage.
4. Circadian blood pressure dipping deteriorates progressively: the "dipper" phenotype falls from 61.1% (stage II) to 11% (stage IV), while "non-dipper" and "night-peaker" profiles increase substantially, sustaining persistent hemodynamic renal stress.
5. The FEV_1–eGFR correlation weakens with advancing severity ($r=0.43 \rightarrow 0.30 \rightarrow 0.29$), reflecting the growing contribution of hypoxemia and hemodynamic factors to nephropathy at advanced disease.
6. Individualized Telsartan A + eplerenone therapy significantly improved FEV_1, eGFR, type IV collagen, aldosterone, E-selectin, and TNF-α at all COPD severity stages, confirming its renoprotective, anti-inflammatory, and anti-fibrotic value.

8. Clinical Recommendations

Measurement of serum aldosterone, type IV collagen, and E-selectin is recommended in all COPD patients with arterial hypertension as an early

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screening strategy for subclinical renal pathology, enabling intervention before clinically manifest chronic kidney disease.

Individualized standard COPD therapy should be supplemented with Telsartan A (telmisartan + amlodipine) dosed according to blood pressure targets and eplerenone 25–50 mg/day in COPD-AH comorbidity. This combination stabilizes renal structural and functional parameters and mitigates the systemic inflammatory burden across all COPD severity stages.

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