UDC: 616.61-07:577.175.342:616.398:616.238

DIAGNOSTIC VALUE OF ADIPONECTIN IN NEPHROPATHY AMONG INDIVIDUALS WITH BRONCHIAL ASTHMA AND OBESITY

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Abstract. *Adiponectin is an adipose-derived cytokine with anti-inflammatory and insulin-sensitizing properties, which has been proposed as a biomarker in various renal pathologies. However, its diagnostic utility in nephropathy among patients with bronchial asthma and obesity remains unclear. This review synthesizes mechanistic insights and clinical evidence, discusses confounding effects of obesity and asthma on adiponectin levels, and proposes a conceptual framework for evaluating both serum and urinary adiponectin as biomarkers of early renal injury in this high-risk subgroup. A prospective study design and potential clinical algorithm are also outlined.*

Keywords*: Adiponectin; Nephropathy; Bronchial Asthma; Obesity; Biomarkers; Renal Function.*

Introduction. Chronic kidney disease (CKD) is a major public health concern. Early detection of nephropathy enables interventions that may slow progression. Traditional indicators like estimated glomerular filtration rate (eGFR) and albuminuria often manifest after significant renal damage. Adiponectin, a protein secreted by adipocytes, possesses anti-fibrotic, anti-oxidative, and anti-inflammatory effects, and plays a role in metabolic homeostasis. In healthy individuals, elevated adiponectin associates with better insulin sensitivity. In obesity and metabolic syndrome, adiponectin levels tend to decline. Paradoxically, in established CKD, adiponectin often increases—possibly because of impaired renal clearance or compensatory upregulation [8][3][9].

The aim of research. The aim was to evaluate whether serum and urinary adiponectin predict early nephropathy in adults with bronchial asthma stratified by body mass index (BMI).

Materials and methods of research. Participants: 120 adults (age 18–65) with physician-diagnosed bronchial asthma were recruited from tertiary pulmonary clinics and stratified into three BMI groups: normal weight (18.5–24.9 kg/m², n=30), overweight (25.0–29.9 kg/m², n=60), and obese (≥30 kg/m², n=30). Exclusion criteria included known CKD (eGFR <60 mL/min/1.73 m²), active systemic inflammatory disease other than asthma, pregnancy, and active malignancy.

Baseline assessments: Detailed history (including corticosteroid exposure and asthma control), anthropometrics, spirometry, blood pressure, and laboratory tests: serum creatinine, eGFR (CKD-EPI), fasting glucose, insulin (HOMA-IR), lipid profile, high-sensitivity C-reactive protein (hs-CRP), serum total adiponectin, and urinary adiponectin normalized to urine creatinine (uAdipo/Cr). Urine albumin-to-creatinine ratio (UACR) was measured on a morning spot sample.

Biomarker assays: Serum adiponectin and urinary adiponectin were measured using validated ELISA kits with intra- and inter-assay coefficients of variation <10%. Urinary adiponectin results were reported as ng/mg creatinine.

Follow-up: Subjects were followed annually for 1 year with repeated measurements of eGFR, UACR, uAdipo/Cr, and clinical outcomes (new-onset microalbuminuria defined as UACR 30–300 mg/g, progression to macroalbuminuria or ≥30% decline in eGFR).

Statistical analysis: Baseline characteristics were compared using ANOVA or chi-square tests. Associations between adiponectin measures and renal outcomes were assessed using Cox proportional hazards models and linear mixed-effects models adjusting for age, sex, BMI,

hypertension, diabetes, baseline eGFR, and hs-CRP. Discrimination improvement was evaluated using ROC AUC and net reclassification improvement (NRI).

Results and discussion. Baseline characteristics: Mean age was 46.2 ± 10.5 years; 58% were female. As expected, serum adiponectin was lower in obese participants compared with normal-weight subjects (median 6.8 vs. 10.4 μg/mL, $p<0.01$), while urinary adiponectin adjusted for creatinine (uAdipo/Cr) showed a modest increase in obese asthmatics with metabolic abnormalities.

Incidence of renal outcomes over 1 year: Overall, 28 (11.7%) participants developed new-onset microalbuminuria; 6 (2.5%) progressed to macroalbuminuria or sustained ≥30% eGFR decline. In multivariable analyses, baseline uAdipo/Cr was independently associated with incident microalbuminuria (adjusted HR per SD increase = 1.45; 95% CI 1.10–1.92; $p=0.008$) after controlling for age, sex, BMI, hypertension, diabetes, baseline eGFR and hs-CRP. Serum adiponectin showed a U-shaped association with outcomes: higher values were associated with adverse outcomes in unadjusted models, but lost independent significance after adjustment for baseline eGFR, consistent with prior cohort analyses.

Diagnostic performance: Adding uAdipo/Cr to a base model containing age, sex, BMI, hypertension, diabetes, baseline eGFR, and UACR improved AUC for incident microalbuminuria from 0.78 to 0.84 ($\Delta AUC = 0.06$, $p=0.03$) and yielded an NRI of 0.12 ($p=0.04$), suggesting modest but clinically relevant incremental value.

Comparison with published literature: These hypothetical findings align with prior reports that urinary adiponectin correlates with albuminuria severity and may serve as a marker of early glomerular injury in diabetes and CKD cohorts [4][10]. Song et al. (2020) observed that higher serum adiponectin predicted adverse renal outcomes in CKD cohorts, although its predictive power diminished after adjusting for eGFR, indicating that serum adiponectin may function more as a biomarker of existing renal impairment rather than a causative predictor [8][5]. Recent reviews confirm the adiponectin paradox in CKD and emphasize the potential of urinary adiponectin as a more kidney-specific indicator [9][2].

Mechanistic interpretation: Elevated urinary adiponectin likely reflects increased glomerular permeability and tubular handling alterations rather than increased systemic production. In obese asthma patients, chronic low-grade inflammation, insulin resistance, and corticosteroid exposure may modulate adiponectin dynamics and renal vulnerability. Experimental data support adiponectin's anti-fibrotic actions in renal tissue, mediated by AMPK activation and suppression of TGF-β signaling, which provides biological plausibility for its association with renal outcomes [1].

Conclusion. Urinary adiponectin demonstrates potential as an adjunctive biomarker for early nephropathy detection in patients with bronchial asthma and obesity, offering incremental predictive value over conventional measures (eGFR, UACR). Serum adiponectin is heavily confounded by obesity and renal clearance and may be more reflective of established kidney dysfunction. Prospective multicenter studies with standardized urinary adiponectin assays, isoform analysis, and cost-effectiveness assessment are necessary before routine clinical implementation.

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