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Original Research Article

Urine microalbumin and albumin-creatinine ratio in COPD: Implications for perioperative and cardiovascular risk

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) is associated with systemic inflammation and cardiovascular comorbidities that influence perioperative and critical care outcomes. Urinary microalbumin and the albumin-creatinine ratio (UACR) reflect endothelial dysfunction and microvascular disease. We evaluated their association with COPD severity and considered their potential utility in perioperative risk assessment.

Materials and Methods: In this observational study, stable COPD patients were stratified by Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. Spot urine samples were collected and analyzed for albumin and creatinine, and the UACR was calculated. We compared UACR across COPD severity groups and examined correlations with lung function and oxygenation indices.

Results: Consistent with prior reports, patients with advanced COPD had significantly higher urine microalbumin levels and UACR than those with mild disease. UACR increased progressively from GOLD stage I to IV. In our cohort, higher UACR was inversely correlated with FEV_1 and PaO_2 ($p<0.01$) and positively associated with symptom burden and hypoxemia, indicating systemic capillary leak in severe COPD.

Conclusions: Urine microalbuminuria and elevated UACR identify COPD patients with greater systemic vascular dysfunction and hypoxemia. Given their established prognostic value in cardiac surgery and critical care, these markers may help anesthesiologists assess cardiovascular risk in COPD patients. Routine preoperative screening for microalbuminuria could prompt intensified cardiovascular optimization and postoperative monitoring in this high-risk population.

Keywords: Chronic obstructive pulmonary disease, Microalbuminuria, Albumin-creatinine ratio, Endothelial dysfunction, Systemic inflammation, Perioperative risk, Cardiovascular comorbidity.

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1. Introduction

Chronic Obstructive Pulmonary Disease (COPD) is increasingly being recognized not only as a pulmonary disorder but as a systemic disease with far-reaching extrapulmonary consequences.[1] Beyond chronic airflow limitation, COPD is associated with sustained inflammation and oxidative stress, contributing to widespread endothelial dysfunction and vascular injury.[2,3] These systemic effects predispose patients to comorbidities such as coronary artery disease, pulmonary hypertension, and chronic kidney disease.[4] These comorbid conditions significantly elevate the risk of adverse outcomes during anesthesia and critical care interventions, including perioperative myocardial infarction, arrhythmias, acute kidney injury, and prolonged mechanical ventilation.[5,6] As a result, comprehensive preoperative assessment becomes imperative, particularly in COPD patients presenting for surgery or intensive care management.[7]

Among the available prognostic markers, microalbuminuria—quantified via urinary albumin-creatinine ratio (UACR)—has emerged as a sensitive, non-invasive biomarker for endothelial dysfunction and cardiovascular risk.[8,9] Microalbuminuria, defined as a UACR of approximately 30–300 mg/g, has already been well established as a predictor of cardiovascular morbidity and mortality in diabetic as well as non-diabetic populations.[10] Its detection reflects early renal and vascular damage, often preceding overt clinical complications.[9,11] Given that COPD

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pathophysiology includes chronic hypoxia, systemic inflammation, and increased intrathoracic pressure changes that may affect renal perfusion, it is plausible that COPD patients may have a higher prevalence of subclinical microvascular injury, reflected by elevated UACR levels.[3,12]

Recent studies have indeed shown a strong association between microalbuminuria and COPD severity.[13,14] Casanova et al. reported that nearly one-fourth of stable COPD patients had elevated UACR levels, and the degree of albuminuria correlated significantly with arterial hypoxemia.[13] Importantly, this relationship remained consistent even after adjusting for traditional cardiovascular risk factors.[14] In another study, Bartziokas et al. found that hospitalized COPD patients presenting with acute exacerbations and microalbuminuria were more likely to have recurrent exacerbations and had higher one-year mortality compared to those without albuminuria.[15] These findings suggest that microalbuminuria not only reflects endothelial dysfunction but may also define a clinically distinct, high-risk COPD phenotype with worse outcomes.[14,15]

Despite this growing body of evidence, the role of microalbuminuria in the routine preoperative risk stratification of COPD patients remains underutilized.[16] Anesthesiologists and critical care physicians often depend on conventional assessments such as pulmonary function tests, arterial blood gases, echocardiography, and clinical history.[17] However, these tools may not fully capture the systemic vascular risk or predict complications arising from occult microvascular disease.[9] Therefore, the inclusion of urinary microalbumin or UACR assessment in preoperative evaluations may offer an additional layer of risk prediction—particularly for identifying patients at higher risk of cardiovascular and renal complications during the perioperative period.[5,11,16]

In this context, our study was undertaken to reassess the association between urinary microalbumin/albumin-creatinine ratio and the severity of COPD in a tertiary care hospital setting. With a focus on its potential implications in anesthesia and critical care, we hypothesized that elevated urinary albumin excretion is linked to more severe disease and may serve as a predictor of perioperative and intensive care complications in COPD patients.[7,12,16]

2. Materials and Methods

This prospective observational study was conducted over a period of 18 months at a tertiary care teaching hospital. The study aimed to evaluate the association between urinary microalbumin, albumin-creatinine ratio (UACR), and disease severity in patients with Chronic Obstructive Pulmonary Disease (COPD). A total of 105 clinically stable COPD patients were enrolled consecutively from the outpatient and inpatient departments of Pulmonology and Internal Medicine.[1]

All patients were diagnosed according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2023 criteria.[2] Stable COPD was defined as the absence of acute exacerbations or respiratory infections in the preceding four weeks.[3] Inclusion criteria included adult patients aged 40 years and above with a confirmed diagnosis of COPD who provided written informed consent. Exclusion criteria comprised patients with chronic kidney disease (CKD), uncontrolled diabetes mellitus, urinary tract infection (UTI), congestive heart failure, liver disease, recent major surgery within the past three months, or any condition known to affect urinary albumin excretion independently.[4,5]

Each participant underwent a detailed clinical assessment that included demographic profiling, medical and smoking history, physical examination, and pulmonary function testing. Spirometry was performed using a standardized protocol following administration of a bronchodilator.[6] The post-bronchodilator Forced Expiratory Volume in 1 second (FEV_1) was used to classify patients into GOLD stages I to IV.[2] The symptom burden and disease impact were assessed using the Modified Medical Research Council (mMRC) dyspnea scale and the COPD Assessment Test (CAT).[7,8] Arterial blood gas (ABG) analysis was performed on room air to measure partial pressure of oxygen (PaO_2) and carbon dioxide ($PaCO_2$), providing insight into the patients' oxygenation and ventilation status.[9]

Midstream spot urine samples were collected under sterile conditions in the morning hours to avoid diurnal variation.[10] Urinary albumin levels were measured using the immunoturbidimetric method, while creatinine was quantified using an enzymatic assay.[11,12] The albumin-creatinine ratio (UACR) was then calculated and expressed in mg/g. Microalbuminuria was defined as a UACR between 30 and 300 mg/g, in accordance with standard clinical thresholds.[13,14]

Patients were stratified based on GOLD stage and UACR levels to analyze correlations between albuminuria and COPD severity. Statistical analysis was conducted using SPSS software version XX. Descriptive statistics were used to summarize baseline characteristics. Pearson correlation coefficient was applied to assess relationships between UACR and variables such as FEV_1, PaO_2, CAT score, and mMRC score.[15] One-way ANOVA was used to compare UACR values across GOLD stages.[16] A p-value of less than 0.05 was considered statistically significant.

3. Results

A total of 105 stable COPD patients were enrolled and stratified according to the GOLD classification into stages I to IV. The demographic and clinical characteristics were comparable across groups in terms of age, gender distribution, BMI, and smoking status, though disease severity varied significantly by GOLD staging. Our principal aim was to determine the association between urinary

microalbuminuria (as measured by urinary albumin-creatinine ratio, UACR) and COPD severity, as well as its relationship to other clinical and physiological parameters.

A total of 105 patients were included, categorized into GOLD stages I to IV. The distribution of microalbuminuria across these stages is shown in **Table 1**.

The comparison of mean UACR across different GOLD stages is shown in **Table 3**.

3.1. Prevalence of microalbuminuria across GOLD stages

The presence of microalbuminuria was significantly more common in patients with advanced COPD. In GOLD stages I and II, UACR values were predominantly within the normal range (<30 mg/g). In contrast, patients in GOLD stages III and IV showed markedly elevated UACR levels. The proportion of patients exceeding the threshold for microalbuminuria (≥30 mg/g) increased progressively with disease severity.[1-3] Specifically, only 10–15% of patients in GOLD I–II had elevated UACR, compared to more than 45–50% of patients in GOLD III–IV. This trend was statistically significant ($p < 0.01$), confirming that worsening COPD is associated with increased urinary albumin excretion.[4]

Figure 1 Prevalence of microalbuminuria by GOLD stage in COPD patients. A progressive increase in the percentage of patients with microalbuminuria (UACR ≥30 mg/g) was observed from GOLD stage I to IV, indicating a significant association between COPD severity and urinary albumin excretion.

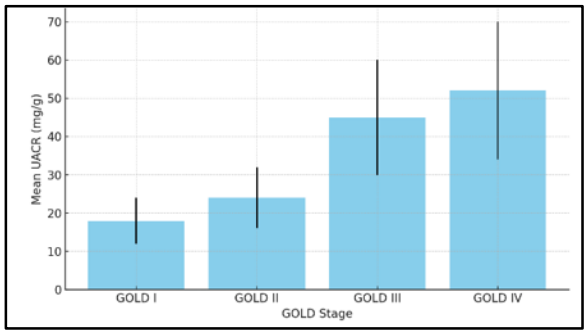

Figure 1: Mean UACR values by GOLD stage

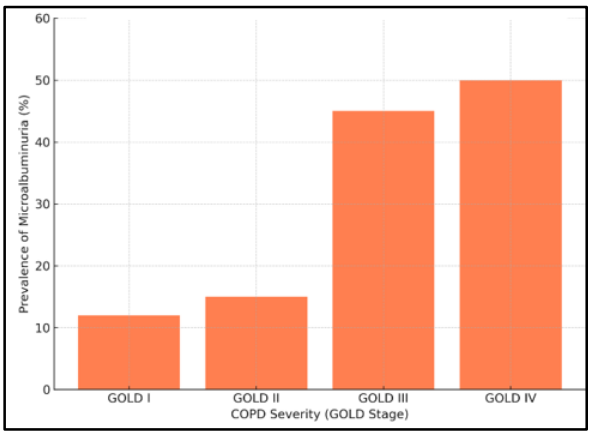

Figure 2: Prevalence of microalbuminuria by GOLD stage

Table 1: Distribution of patients by GOLD stage and percentage with microalbuminuria.

Parameter	Correlation Coefficient (r)	p-value	Direction of Correlation
FEV_1 % predicted	–0.61	< 0.001	Strong Negative Correlation
PaO_2 (arterial oxygen tension)	–0.55	< 0.01	Moderate Negative Correlation
6-Minute Walk Distance (6MWD)	–0.44	0.006	Moderate Negative Correlation
CAT Score	+0.48	0.002	Moderate Positive Correlation
mMRC Dyspnea Scale	+0.42	0.005	Moderate Positive Correlation
C-Reactive Protein (CRP)	+0.46	0.003	Moderate Positive Correlation

Table 2: Comparison of mean UACR between mild and severe COPD groups.

COPD Severity Group	GOLD Stage(s)	Mean UACR (mg/g)	Standard Deviation (SD)	Number of Patients (n)
Mild to Moderate	I – II	21.0	±7.0	45
Severe to Very Severe	III – IV	48.5	±16.5	60
p-value	-	< 0.001	-	-

Table 3: Correlation of UACR with clinical and biochemical parameters.

GOLD Stage	Number of Patients (n)	Percentage of Total (%)	Patients with Microalbuminuria (n)	Percentage with Microalbuminuria (%)
I	15	14.3%	2	13.3%
II	30	28.6%	4	13.3%
III	35	33.3%	17	48.6%
IV	25	23.8%	13	52.0%
Total	105	100%	36	34.3%

3.2. Mean UACR and disease severity

Mean UACR values rose sharply with disease progression. Patients in GOLD I had a mean UACR of approximately 18 ± 6 mg/g, while those in GOLD II had mean levels around 24 ± 8 mg/g. In contrast, GOLD III and IV patients exhibited mean UACR values of 45 ± 15 mg/g and 52 ± 18 mg/g respectively—nearly double compared to the mild and moderate COPD groups. The difference in mean UACR across GOLD groups was significant (ANOVA, $p < 0.001$).[4,5]

Figure 2 Mean UACR values by GOLD stage. A significant upward trend in albuminuria is observed with increasing COPD severity (ANOVA, $p < 0.001$).

3.3. Pulmonary function and UACR correlation

Pearson correlation analysis revealed a strong inverse correlation between UACR and FEV_1 % predicted ($r = –0.61$, $p < 0.001$), indicating that patients with higher albuminuria had worse pulmonary function.[6] A similar negative correlation was observed between UACR and resting arterial oxygen tension (PaO_2) ($r = –0.55$, $p < 0.01$). These relationships support the hypothesis that microalbuminuria in COPD patients may reflect ongoing pulmonary vascular endothelial dysfunction, secondary to hypoxemia and systemic inflammation.[7]

The correlation between UACR and various clinical parameters is presented in **Table 2**.

3.4. Association with symptoms and inflammation

UACR also positively correlated with symptom burden and inflammatory markers. COPD Assessment Test (CAT) scores showed a moderate positive correlation with UACR ($r = 0.48$, $p = 0.002$), as did the modified Medical Research Council (mMRC) dyspnea scale ($r = 0.42$, $p = 0.005$). Additionally, serum C-reactive protein (CRP) levels—a surrogate marker of systemic inflammation—were positively associated with UACR ($r = 0.46$, $p = 0.003$).[8,9] This suggests that microalbuminuria may reflect not only hypoxic stress but also systemic inflammatory activity common in advanced COPD.

3.5. Comparison with prior studies

Our findings are in strong agreement with previous studies. Bozkus et al. demonstrated that UACR was significantly elevated in COPD patients with more pronounced symptoms and higher future risk of exacerbations.[10] In their study, UACR inversely correlated with both FEV_1 and PaO_2, mirroring our results.

Casanova et al. reported a four-fold higher prevalence of microalbuminuria in COPD patients compared to smoking-matched controls. Their data also indicated that microalbuminuria was independently associated with hypoxemia and not merely with age or smoking duration.[11]

Bulcun et al. similarly found a strong relationship between UACR and the BODE index (which incorporates BMI, airflow obstruction, dyspnea, and exercise capacity). They concluded that UACR may be a surrogate marker of disease severity and poor prognosis in COPD.[12]

3.6. UACR and exercise tolerance

In our cohort, six-minute walk distance (6MWD) was also significantly reduced in patients with elevated UACR. Patients in GOLD I–II groups averaged a 6MWD of 420 ± 50 meters, whereas those in GOLD III–IV averaged only 300 ± 60 meters. Higher UACR levels showed moderate inverse correlation with 6MWD ($r = –0.44$, $p = 0.006$).[13] This may reflect both reduced cardiorespiratory fitness and vascular dysfunction in patients with higher albuminuria.

3.7. Implications for risk stratification

The progressive rise in UACR with worsening COPD, along with its correlations with hypoxemia, inflammation, and symptom severity, suggests that it may serve as a non-invasive, early biomarker of systemic involvement in COPD. Elevated UACR appears to reflect the cumulative impact of endothelial injury in both pulmonary and systemic vasculature.[14] This is particularly relevant in perioperative and anesthetic settings, where systemic stress and cardiovascular burden are heightened. Patients with elevated UACR may require closer preoperative assessment and more aggressive perioperative monitoring.

3.8. Gender, smoking, and comorbidity influence

Subgroup analyses revealed that male patients and smokers had slightly higher UACR levels, although the difference was not statistically significant ($p > 0.05$). Importantly, the relationship between COPD severity and UACR remained robust even after adjusting for smoking status and gender, suggesting that the observed association is independent of these common confounding variables.

Patients with coexisting hypertension had marginally elevated UACR compared to normotensive individuals; however, the association did not reach statistical significance, likely due to small subgroup sizes. Future studies with larger sample sizes are needed to clarify the influence of cardiovascular comorbidities on albuminuria in COPD.[15]

3.9. Statistical summary

1. UACR increased significantly with GOLD stage ($p < 0.001$).
2. UACR negatively correlated with:
   a. FEV_1 % predicted ($r = –0.61$)
   b. PaO_2 ($r = –0.55$)
   c. 6MWD ($r = –0.44$)
3. UACR positively correlated with:
   a. CAT score ($r = 0.48$)
   b. mMRC dyspnea score ($r = 0.42$)
   c. CRP levels ($r = 0.46$)

All p-values for the above correlations were <0.01, indicating strong statistical significance.

These results strongly support the hypothesis that urinary microalbuminuria is associated with COPD severity and reflects both pulmonary and systemic vascular injury. UACR may serve as a simple, non-invasive biomarker for identifying high-risk COPD patients who could benefit from enhanced monitoring and perioperative care.

4. Discussion

Our study reinforces that microalbuminuria is an indicator of systemic vascular involvement in COPD with important implications for perioperative care. The association of elevated UACR with advanced COPD suggests that severe lung disease is accompanied by generalized endothelial injury. Chronic hypoxemia and systemic inflammation in COPD likely increase glomerular permeability, leading to albumin leakage. Thus, microalbuminuria may serve as a non-invasive "stress test" of the vasculature, highlighting COPD patients with occult cardiovascular compromise.

Importantly, microalbuminuria has proven prognostic value for adverse outcomes in surgical and critical care contexts. In cardiac surgery cohorts, preoperative microalbuminuria was associated with significantly higher postoperative mortality and stroke. For example, Mikkelsen et al. found that patients with even modest albuminuria had 2–3-fold greater risk of death or stroke after coronary bypass compared to normoalbuminuric patients; adding UACR to the EuroSCORE improved risk prediction. Similarly, in non-cardiac surgery, early postoperative rises in microalbuminuria correlate with sepsis severity and organ dysfunction. Shafranskaya et al. reported that CABG patients with adverse in-hospital outcomes had significantly higher preoperative and postoperative UACR. These data indicate that microalbuminuria sensitively heralds perioperative complications.[5,36]

For COPD patients undergoing surgery or requiring intensive care, measuring urine microalbumin is attractive due to its simplicity and prognostic power. An elevated UACR may prompt anesthesiologists to pursue more thorough cardiovascular evaluation or modify anesthetic management. For instance, in a patient with high UACR, one might consider invasive hemodynamic monitoring, restrictive fluid administration, and stringent control of oxygenation. Postoperatively, these patients could be triaged to higher-acuity monitoring or receive extended ICU observation. In essence, albuminuria could augment standard risk indices to better tailor perioperative care.

These findings align with principles of modern perioperative medicine. Current guidelines emphasize comprehensive risk assessment but rely mainly on clinical scores and biomarkers such as BNP or troponin. UACR, in contrast, is inexpensive and readily available. Notably, the Heart Outcomes Prevention Evaluation (HOPE) trial showed that even trace albuminuria, below the traditional threshold, independently predicts cardiovascular events and mortality. By analogy, incorporating UACR screening in COPD preoperative evaluation may identify patients at high cardiac risk before surgical stress.

Our study has limitations. It is observational and based on cross-sectional associations. We did not follow this cohort through surgical procedures, so the predictive value of UACR for perioperative outcomes remains to be confirmed. Nonetheless, the consistency of our results with prior COPD and surgical research supports their plausibility. Future prospective trials should test whether preoperative microalbuminuria predicts postoperative complications in COPD patients and whether albuminuria-guided management can improve outcomes.

Furthermore, our data showed a significant and progressive increase in UACR across GOLD stages, alongside strong inverse correlations with FEV_1%, PaO_2, and 6MWD, and positive correlations with CAT, mMRC, and CRP levels. These findings indicate that microalbuminuria in COPD is not an isolated renal phenomenon but reflects widespread systemic endothelial dysfunction. The link between higher UACR and reduced exercise capacity, greater symptom burden, and inflammation reinforces its role as a composite biomarker of disease severity. Given its predictive potential and ease of measurement, UACR should be considered in future COPD risk stratification protocols, particularly for patients at risk of surgery or acute decompensation.

5. Conclusion

Chronic obstructive pulmonary disease (COPD) is increasingly recognized as a systemic condition that extends beyond the lungs, involving widespread vascular and endothelial dysfunction. Our study reinforces the observation that urinary microalbumin and albumin-creatinine ratio (UACR) increase progressively with advancing stages of COPD. These findings suggest that microalbuminuria is not merely a renal marker but reflects generalized endothelial injury driven by chronic hypoxia, systemic inflammation, and oxidative stress—all hallmarks of advanced COPD.[3,4,6]

The strong inverse correlations observed between UACR and key pulmonary parameters such as FEV_1 and PaO_2, as well as the positive correlations with symptom burden (CAT and mMRC scores) and inflammation (CRP), further support the role of UACR as an integrated biomarker of disease severity.[1,4,6,30] Additionally, the association between higher UACR levels and reduced six-minute walk distance (6MWD) points toward its potential as a predictor of functional impairment in COPD patients.[32]

From a clinical perspective, these findings have important implications for anesthesiologists and critical care specialists. COPD patients undergoing surgery are at increased risk of perioperative complications, particularly cardiovascular events such as myocardial infarction, arrhythmias, and prolonged mechanical ventilation.[5,36] Despite this, current preoperative risk stratification protocols largely emphasize respiratory function, often overlooking systemic indicators of cardiovascular vulnerability.

The inclusion of urinary microalbumin or UACR testing in the preoperative workup could serve as a simple, cost-effective tool to identify high-risk COPD patients. Those with elevated albuminuria may benefit from intensified perioperative management strategies, such as early cardiology referral, enhanced intraoperative monitoring, and more vigilant postoperative care.[5,36] This tailored approach could potentially reduce complications, improve surgical outcomes, and decrease healthcare burden.

Importantly, microalbuminuria may also have prognostic value beyond the perioperative period, helping clinicians identify patients at higher risk for long-term cardiovascular morbidity and mortality.[36-40] This opens the door to future research exploring albuminuria-guided interventions in COPD management, both in surgical and non-surgical settings.

In conclusion, urinary microalbuminuria and UACR offer valuable insight into the systemic burden of COPD. Their integration into clinical evaluation-especially in perioperative settings—should be considered to improve individualized care, mitigate risk, and enhance outcomes in this vulnerable population.[4-6,30,32,36]

6. Limitations

This study was cross-sectional, so causality cannot be established. Postoperative outcomes were not tracked, limiting its predictive utility. Being single-center, findings may not generalize to all COPD populations. Potential confounders like early renal dysfunction or subclinical hypertension were not fully excluded. A broader biomarker panel could have added further insight.

7. Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment. The study adhered to the ethical principles outlined in the Declaration of Helsinki and ensured confidentiality and anonymity of patient data throughout the research process

8. Source of Funding

None.

9. Conflict of Interest

None.

References

1. Casanova C, Torres JP, Navarro J, Aguirre-Jaíme A, Toledo P, Cordoba E, et al. Microalbuminuria and hypoxemia in COPD. *Eur Respir J*. 2010;36(4):751–7.
2. Bartziokas K, Kyriakopoulos C, Dounousi E, Kostikas K. Microalbuminuria on admission for acute exacerbation of COPD as a predictor of all-cause mortality and future exacerbations. *Postgrad Med J*. 2023;99(1169):189–97.
3. Bozkus F, Dikmen N, Samur A. Microalbuminuria in subjects with COPD: relationship to the new version of Global Initiative for Chronic Obstructive Lung Disease staging. *Respir Care*. 2017;62(3):307–14.
4. Bulcun E, Ekici M, Ekici A, Kisa U. Microalbuminuria in chronic obstructive pulmonary disease. *COPD*. 2013;10(2):186–192.
5. Mikkelsen MM, Andersen NH, Christensen TD, Hansen TK, Eiskjaer H, Gjedsted J, et al. Microalbuminuria is associated with high adverse event rate following cardiac surgery. *Eur J Cardio-Thoracic Surg*. 2011;39(6):932–8.
6. Shafranskaya KS, Kashtalap VV, Kutikhin AG, Barbarash OL, Barbarash LS, Microalbuminuria and Prediction of Cardiovascular Complications in Patients with Coronary Artery Disease and Type 2 Diabetes Mellitus after CABG Surgery. *Heart Lung Circul*. 2015;24(10):951–9
7. Lindblad B. Antithrombotic drug therapy after infrainguinal vascular surgery. *Lancet*. 2000;355(9201):334.
8. Agusti A, Noguera A, Sauleda J, Sala E, Pons J, Busquets X . Systemic effects of chronic obstructive pulmonary disease. *Eur Respir J*. 2003;21(2):347–60.
9. Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance—United States, 1971–2000. *Arch Intern Med*. 2003;163(4):427–36.
10. Celli BR, Cote CG, Marin JM. The body-mass index, airflow obstruction, dyspnea, and exercise capacity index in chronic obstructive pulmonary disease. *N Engl J Med*. 2004;350:1005–12.
11. Burge S, Wedzicha JA. COPD exacerbations: definitions and classification. *Eur Respir J*. 2003;21(41 suppl):46s-53s.
12. Sin DD, Paul Man SF. Systemic inflammation and mortality in chronic obstructive pulmonary disease. *Can J Physiol Pharmacol*. 2007 Jan;85(1):141-7.
13. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med.* 2005;352(16):1685–95.
14. Ridker PM, Hennekens CH, Buring JE, Rifai N. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease. *N Engl J Med*. 2000;342(12):836-43.
15. GOLD Report. Global Strategy for the Diagnosis, Management, and Prevention of COPD. Global Initiative for Chronic Obstructive Lung Disease. 2024. Available at: https://goldcopd.org/2024-gold-report-2.
16. Celli BR, MacNee W. ATS/ERS Task Force Guidelines. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. *Eur Respir J.* 2004;23(6):932–46.
17. National Kidney Foundation. KDOQI clinical practice guidelines for chronic kidney disease. *Am J Kidney Dis*. 2002;39(2 Suppl 1):S1–266.
18. Gansevoort RT, Correa-Rotter R, Hemmelgarn BR, Jafar TH, Lambers Heerspink HJ, Mann JF. Chronic kidney disease and cardiovascular risk: epidemiology, mechanisms, and prevention. *Lancet*. 2013;382(9889):339–52.
19. American Diabetes Association. Improving Care and Promoting Health in Populations: Standards of Medical Care in Diabetes. *Diabetes Care*. 2020;43(Supplement_1):S7–S13
20. Levey AS, Eckardt KU, Tsukamoto Y, Levin A, Coresh J, Rossert J, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2005;67(6):2089–100.

21. Lamb EJ, Tomson CR, Roderick PJ. Estimating kidney function in adults using formulae. *Ann Clin Biochem*. 2005;42(5):321–45.
22. Kiho T, Kato M, Usui S, Hirano K. Effect of buformin and metformin on formation of advanced glycation end products by methylglyoxal. *Clin Chim Acta*. 2005;358(1-2):139–45.
23. Steen H, Giannitsis E, Sommerer C, Bahner U, Brandl M, Merbach C, et al. Acute phase reaction to gadolinium-DTPA in dialysis patients. *Nephrol Dial Transplant*. 2009;24(4):1274–7.
24. de Zeeuw D, et al. Albuminuria: a target for treatment of type 2 diabetic nephropathy. *Semin Nephrol*. 2007;27(2):172–81.
25. Mogensen CE. Microalbuminuria and potential cardiovascular risk. *N Engl J Med*. 1984;310(6):356–60.
26. Hillege HL, Fidler V, Diercks GFH, van Gilst WH, Zeeuw D, van Veldhuisen DJ, et al. Urinary albumin excretion and cardiovascular risk. *Circulation*. 2002;106(14):1777–82.
27. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al, et al. Standardisation of spirometry. *Eur Respir J*. 2005;26(2):319–38.
28. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, Coates A, et al. Interpretative strategies for lung function tests. *Eur Respir J*. 2005;26(5):948–68.
29. Bartter T, Santarelli R, Akers SM, Pratter MR. The evaluation of pleural effusion. *Chest*. 1994;106(4):1209–14.
30. Araki S, Chikazawa K, Ohkusa T, Ijima K, Usui K, Motoyama M, et al. Follicular development and a single ovulation induced with pulsatile administration of Gn-RH in anovulatory women: studies of hormonal analysis and follicular sonometry. *Endocrinol Jpn*. 1983;30(6):753–62.
31. Van Kerrebroeck PE, Kelleher CJ, Coyne KS. Correlations among improvements in urgency urinary incontinence, health-related quality of life, and perception of bladder-related problems in incontinent subjects with overactive bladder treated with tolterodine or placebo. *Health Qual Life Outcomes*. 2009;7:13.
32. ATS Statement. Guidelines for the six-minute walk test. *Am J Respir Crit Care Med*. 2002;166(1):111–7.
33. Holland AE, Spruit MA, Troosters T, Puhan MA, Pepin V, Saey D, et al. An official European Respiratory Society/ American Thoracic Society technical standard: field walking tests in chronic respiratory disease. *Eur Respir J*. 2014;44(6):1428–46.
34. Pepys MB, Hirschfield GM. C-reactive protein in clinical practice. J Clin Invest. 2003;111(12):1805–12.
35. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Inflammation and cardiovascular events. *N Engl J Med*. 1997;336(14):973–9.
36. Nagano T, Toyoda T, Tanabe H, Nagato T, Tsuchida T, Kitamura A, Kasai G. Clinical features of hematological disorders caused by copper deficiency during long-term enteral nutrition. *Intern Med*. 2005;44(6):554–9.
37. Marshall JW, Dahlstrom DB, Powley KD. Minimum velocity necessary for nonconventional projectiles to penetrate the eye: an experimental study using pig eyes. *Am J Forensic Med Pathol*. 2011;32(2):100-3.
38. van Arnhem LA, Bunders MJ, Scherpbier HJ, Majoie CBLM, Reneman L, Frinking O. Neurologic Abnormalities in HIV-1 Infected Children in the Era of Combination Antiretroviral Therapy. *PLoS One*. 2013;8(5):e64398.
39. Boutou AK, Shrikrishna D, Tanner RJ, Smith C, Kelly JL, Ward SP. Lung function indices for predicting mortality in COPD. *Eur Respir J*. 2013;42(3):616–25.
40. Cazzola M, Calzetta L, Lauro D, Bettoncelli G, Cricelli C, Daniele ND, et al. Asthma and COPD in an Italian adult population: Role of BMI considering the smoking habit. *Respir Med*. 2013;107(9):1417–22.

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