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KIDNEY FUNCTION DISORDERS IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND OBESITY: DIAGNOSTIC AND THERAPEUTIC PERSPECTIVES

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Abstract

Chronic obstructive pulmonary disease (COPD) and obesity are common chronic conditions that frequently coexist and contribute to multisystem dysfunction. Among the less frequently discussed but clinically significant complications in such patients are kidney function disorders. The interaction between chronic hypoxia, systemic inflammation, oxidative stress, hemodynamic instability, insulin resistance, and metabolic abnormalities may accelerate renal impairment in this population. This article examines the mechanisms underlying kidney dysfunction in patients with COPD and obesity, highlights the main diagnostic approaches, and discusses current therapeutic perspectives. Early recognition of renal impairment in these patients is essential for improving prognosis, reducing complications, and optimizing multidisciplinary care.

Keywords

COPD, obesity, kidney dysfunction, chronic kidney disease, hypoxia, inflammation, renal diagnostics, multimorbidity, therapy, comorbidity

Introduction

Chronic obstructive pulmonary disease is a progressive inflammatory disorder of the airways characterized by persistent airflow limitation and systemic consequences beyond the lungs. Obesity, another major global health problem, is associated with metabolic, cardiovascular, and renal complications. The coexistence of COPD and obesity creates a complex clinical condition in which several organs and regulatory systems are simultaneously affected.

Kidney dysfunction in patients with COPD and obesity deserves special attention because renal impairment may develop gradually and remain clinically silent in the early stages. Chronic hypoxemia, hypercapnia, endothelial dysfunction, low-grade systemic inflammation, activation of the renin-angiotensin-aldosterone system, and obesity-related glomerular hyperfiltration contribute to structural and functional renal changes. These processes may result in reduced glomerular filtration rate, albuminuria, electrolyte imbalance, and progressive chronic kidney disease.

The aim of this article is to analyze the main mechanisms of kidney function disorders in patients with COPD and obesity and to summarize diagnostic and therapeutic approaches relevant to clinical practice.

Literature Review

Recent clinical and pathophysiological studies have shown that COPD should no longer be viewed as an isolated pulmonary disease. It is now recognized as a systemic disorder frequently associated with cardiovascular disease, skeletal muscle dysfunction, metabolic syndrome, and chronic kidney disease. Investigators have reported that reduced renal function is more common in patients with moderate to severe COPD than in the general population, especially in those with chronic hypoxemia and repeated exacerbations.

At the same time, obesity has been widely identified as an independent risk factor for renal injury. Increased body mass contributes to glomerular hyperfiltration, increased intraglomerular pressure, insulin resistance, dyslipidemia, and chronic inflammation. These processes may

eventually lead to obesity-related nephropathy. In obese individuals with COPD, renal damage may be aggravated by sleep-disordered breathing, arterial hypertension, and type 2 diabetes mellitus.

Several authors have emphasized that traditional assessment of kidney function may underestimate renal impairment in COPD patients due to reduced muscle mass and altered creatinine production. Therefore, more accurate biomarkers and combined diagnostic tools are increasingly recommended. Current literature also supports an integrated therapeutic approach aimed at correcting hypoxia, controlling body weight, reducing inflammation, managing comorbidities, and preventing nephrotoxic exposure.

Materials and Methods

This article is based on a narrative analytical review of clinical, pathophysiological, and therapeutic aspects of kidney dysfunction in patients with COPD and obesity. The discussion is structured around major pathogenic mechanisms, diagnostic criteria, and management strategies described in contemporary medical literature and clinical concepts. Special attention is given to the interaction between respiratory insufficiency, obesity-related metabolic disturbances, and renal hemodynamics.

The analysis includes the following components: evaluation of mechanisms linking COPD and obesity with renal impairment; identification of major laboratory and instrumental diagnostic methods; and review of therapeutic measures aimed at slowing progression of kidney dysfunction in multimorbid patients. A comparative and descriptive method was used to summarize the available evidence and formulate clinically relevant conclusions.

Results

The analysis demonstrates that kidney dysfunction in patients with COPD and obesity develops under the influence of several interrelated factors.

First, chronic hypoxia plays a major pathogenic role. Long-term oxygen deficiency causes renal vasoconstriction, endothelial dysfunction, activation of inflammatory mediators, and tubulointerstitial injury. Repeated episodes of hypoxemia during COPD exacerbations may worsen renal perfusion and accelerate nephron damage.

Second, systemic inflammation contributes to both pulmonary and renal injury. COPD is associated with increased circulating inflammatory cytokines, while obesity promotes chronic low-grade inflammation through adipose tissue-derived mediators. The combined inflammatory burden may impair glomerular filtration and promote fibrosis.

Third, obesity-related metabolic changes significantly affect kidney function. Hyperinsulinemia, insulin resistance, dyslipidemia, and arterial hypertension increase intraglomerular pressure and may lead to albuminuria and gradual decline in renal function. In many patients, the coexistence of metabolic syndrome intensifies this process.

Fourth, altered hemodynamics and comorbid cardiovascular disease worsen renal outcomes. Right heart dysfunction, pulmonary hypertension, and systemic hypertension may reduce effective renal blood flow. Fluid retention and venous congestion may additionally impair kidney function.

Fifth, pharmacological factors are important. Some patients with COPD and obesity are exposed to repeated courses of nonsteroidal anti-inflammatory drugs, certain antibiotics, diuretics, or radiographic contrast agents, all of which may increase the risk of renal injury in susceptible individuals.

The most common renal manifestations in these patients include decreased estimated glomerular filtration rate, microalbuminuria or proteinuria, electrolyte disorders, elevated serum urea and creatinine, and increased risk of acute kidney injury during exacerbations or hospitalization.

Discussion

The coexistence of COPD, obesity, and kidney dysfunction represents a clinically challenging triad. The relationship between these conditions is bidirectional and self-reinforcing. COPD-related hypoxia and systemic inflammation can promote renal injury, while kidney dysfunction may worsen acid-base balance, fluid regulation, and cardiovascular stability, thereby negatively influencing respiratory status. Obesity intensifies both processes through metabolic and hemodynamic pathways.

One of the main diagnostic difficulties is the underrecognition of chronic kidney disease in COPD patients. Serum creatinine alone may be misleading, especially in individuals with muscle wasting or sarcopenic obesity. Therefore, kidney assessment should include estimated glomerular filtration rate, urine albumin-to-creatinine ratio, serum electrolytes, blood pressure monitoring, and, when indicated, renal ultrasonography. Cystatin C may also provide additional value in selected patients because it is less dependent on muscle mass.

From a therapeutic perspective, management must be individualized and multidisciplinary. Optimal COPD treatment is essential because improved oxygenation and reduced exacerbation frequency may help preserve renal perfusion and limit inflammatory stress. In obese patients, body weight reduction through diet, physical rehabilitation, and behavioral support can improve insulin sensitivity, blood pressure, and renal hemodynamics. However, weight management should be gradual and supervised, especially in patients with severe respiratory limitation.

Control of cardiovascular and metabolic comorbidities is another cornerstone of therapy. Hypertension, diabetes, and dyslipidemia should be carefully managed to reduce renal and vascular injury. Nephrotoxic medications should be avoided whenever possible, and drug dosing should be adjusted according to renal function. During COPD exacerbations, close monitoring of hydration status, oxygenation, arterial blood gases, and renal markers is particularly important because acute kidney injury may develop rapidly under conditions of infection, dehydration, or hemodynamic instability.

Pulmonary rehabilitation and moderate physical activity may indirectly support kidney health by improving exercise tolerance, metabolic profile, and endothelial function. Nutritional correction is equally important, especially in patients with mixed obesity and muscle loss. In advanced cases, collaboration between pulmonologists, nephrologists, endocrinologists, cardiologists, and dietitians is necessary for optimal long-term management.

Conclusion

Kidney function disorders in patients with chronic obstructive pulmonary disease and obesity are an important but often underestimated clinical problem. Their development is associated with chronic hypoxia, systemic inflammation, oxidative stress, metabolic abnormalities, altered renal hemodynamics, and the burden of multiple comorbid conditions. These mechanisms interact and contribute to progressive renal impairment, especially in patients with severe COPD, obesity-related metabolic syndrome, or recurrent exacerbations.

Early detection of kidney dysfunction requires comprehensive assessment rather than reliance on serum creatinine alone. Estimated glomerular filtration rate, albuminuria testing, electrolyte analysis, and evaluation of cardiovascular and metabolic status are essential components of patient monitoring. Timely diagnosis allows clinicians to identify high-risk patients and prevent progression to advanced chronic kidney disease.

Therapeutic management should be multidisciplinary and focused on both pulmonary and renal protection. Optimization of COPD therapy, correction of hypoxemia, weight management, careful control of hypertension and diabetes, avoidance of nephrotoxic agents, and regular renal monitoring are key strategies. A personalized and integrated approach may improve outcomes, reduce complications, and enhance quality of life in this complex group of patients.

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