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CARDIOPULMONARY CONTINUUM: PATHOGENETIC INTERRELATIONS

Abstract. In the 21st century, clinical medicine faces new challenges due to the significant increase in the prevalence of chronic non-communicable diseases. Among the most significant are cardiovascular diseases, diabetes mellitus, and chronic obstructive pulmonary disease (COPD). These conditions greatly affect population health, leading to reduced quality of life, decreased functional capacity, and eventually loss of working ability. Modern medical practice shows that a single patient often presents with several chronic conditions simultaneously. Such a combination of different pathological states is referred to as comorbidity. Epidemiological data indicate that one of the most common combinations is the simultaneous presence of cardiovascular diseases and COPD. This combination significantly worsens the clinical course of disease, lowers patients' quality of life, and negatively affects long-term prognosis.

The relationship between cardiovascular and respiratory pathology has been termed the cardiopulmonary continuum. Currently, scientific literature actively discusses the shared mechanisms of development and progression of these diseases, as well as the similarity of risk factors that contribute to their formation. Among the most significant risk factors are smoking, excessive body weight, arterial hypertension, and metabolic disorders. Numerous clinical studies confirm that the combination of COPD with cardiovascular diseases is associated with a worse prognosis compared to the isolated course of these conditions. Therefore, COPD is considered not only a respiratory disease but also an independent risk factor for the development of cardiovascular complications and adverse outcomes in patients with comorbid pathology. The similarity of the pathogenesis mechanisms of these diseases is explained by a number of shared processes. Among them, particular importance is attributed to the activation of the renin–angiotensin–aldosterone system, the development of systemic inflammatory response, and endothelial dysfunction, which together contribute to the progression of both cardiovascular and respiratory pathology.

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КАРДІОПУЛЬМОНАЛЬНИЙ КОНТИНУУМ: ПАТОГЕНЕТИЧНІ ВЗАЄМОЗВ'ЯЗКИ

Анотація. У XXI столітті перед клінічною медициною постали нові виклики, зумовлені значним зростанням поширеності хронічних неінфекційних захворювань. До найбільш значних з них відносяться серцево-судинні захворювання, цукровий діабет та хронічне обструктивне захворювання легень (ХОЗЛ). Ці захворювання значно впливають на стан здоров'я населення, призводячи до погіршення якості життя пацієнтів, зниження функціональних можливостей і, надалі, до втрати працездатності. Сучасна медична практика свідчить, що в одного пацієнта нерідко виявляється кілька хронічних захворювань одночасно. Подібне поєднання різних патологічних станів сприймається як коморбідність. За даними епідеміологічних досліджень, однією з найпоширеніших комбінацій є одночасна наявність серцево-судинних захворювань та ХОЗЛ. Таке поєднання суттєво погіршує клінічний перебіг захворювань, знижує якість життя пацієнтів та негативно впливає на довгостроковий прогноз. Взаємозв'язок між патологією серцево-судинної та дихальної систем отримала назву кардіопульмонального континууму. В даний час у науковій літературі активно обговорюються загальні механізми розвитку та прогресування даних захворювань, а також схожість факторів ризику, що сприяють їхньому формуванню. До найбільш значущих факторів відносять куріння, надмірну масу тіла, артеріальну гіпертензію та метаболічні порушення. Численні клінічні дослідження підтверджують, що поєднання ХОЗЛ із серцево-судинними захворюваннями супроводжується більш несприятливим прогнозом порівняно з ізольованим перебігом цих патологій. У зв'язку з цим ХОЗЛ розглядається не лише як захворювання дихальної системи, а й як незалежний фактор ризику розвитку серцево-судинних ускладнень та погіршення наслідків у пацієнтів з комор бідною патологією. Подібність патогенетичних механізмів цих захворювань пояснюється низкою загальних процесів. Серед них особливе значення мають активація ренін-ангіотензин-альдостеронової системи, розвиток системної запальної реакції, а також порушення функції ендотелію судин, що в сукупності сприяє прогресу серцево-судинної, так і респіраторної патології.

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Ключові слова: хронічна обструктивна хвороба легень; серцево-судинні захворювання; системне запалення; ендотеліальна дисфункція.

Problem Statement. The pandemic of chronic non-communicable diseases represents a global healthcare challenge with profound medical and socio-economic implications. Cardiovascular diseases (CVD), diabetes mellitus (DM), and chronic obstructive pulmonary disease (COPD) are among the conditions leading to a deterioration in the quality of life and, eventually, to functional impairment and disability [1, 17]. Notably, respiratory diseases, specifically COPD, are increasingly taking a leading position in the structure of overall morbidity.

Clinical practice frequently encounters the presence of multiple comor-bidities in a single patient. Recent research indicates that cardiovascular complications are the most common cause of death among patients with COPD [3, 7, 11]. A correlation has been established between the severity of bronchial obstruction—measured by the forced expiratory volume in 1 second (FEV1) – and CVD risk: the highest risk of cardiovascular mortality was observed in the group with the lowest FEV1 values [7, 9]. There is a robust evidence base demonstrating an unfavorable prognosis when COPD and CVD coexist [3, 11]. Currently, there is no definitive evidence regarding which condition is primary. It is highly probable that these chronic diseases develop concurrently in response to shared risk factors, such as smoking, arterial hypertension (AH), metabolic syndrome (MS), and diabetes mellitus [5, 7].

Objectives. The evidence regarding the coexistence of CVD and COPD has defined the objective of our study: to identify shared pathogenetic factors involved in the development and progression of these diseases.

The maintenance and progression of pathological processes in both CVD and COPD are rooted in pathogenetic pathways involving the synthesis of pro-inflam-matory cytokines, activation of the renin-angiotensin-aldosterone system (RAAS), and other mechanisms of cellular and humoral response. Below, we examine the specific role each of these mechanisms plays in the clinical overlap of COPD and CVD.

Systemic Inflammatory Response. The role of inflammatory mediators in the pathogenesis of CVD has been documented in numerous studies [10, 15, 16]. One of the most widely recognized markers of the inflammatory response is **C**-reactive protein (CRP), which exerts a pro-inflammatory effect on the vascular wall by increasing the production of other inflammatory mediators and adhesion molecules on the endothelial surface, thereby leading to the development of atherosclerosis [6, 13]. According to the Framingham Study, CRP levels of <1.1, 1.1–3.0, and >3.0 mg/L correlated with low, moderate, and high risks of cardiovascular events, respectively [6]. Inflammatory mediators are linked to key pathogenetic mechanisms of CVD development, such as reduced myocardial contractility, low cardiac output, endothelial dysfunction, and hypercoagulation [8, 13].

In COPD, the role of the inflammatory response was investigated in the NHANES III epidemiological study, which analyzed the association between the

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presence and severity of bronchial obstruction and CRP levels. Patients with severe bronchial obstruction (FEV1 <50% of predicted) were 2.18 times more likely to have elevated CRP (>2.2 mg/L) and 2.74 times more likely to have high CRP levels (>10 mg/L) compared to those without obstruction (after adjusting for age, sex, smoking, body mass index, and comorbidities). In patients with moderate obstruction (FEV1 50–80% of predicted), elevated and high CRP levels were found 1.41 and 1.56 times more frequently, respectively, than in those without obstruction. Similar patterns have been identified for other markers of systemic inflammation, such as serum fibrinogen, platelet counts, and white blood cell counts [11, 13].

Given the strong correlation between systemic inflammatory markers and COPD manifestations, several studies have suggested that COPD itself can trigger an inflammatory response [11, 14]. It is hypothesized that prolonged exposure to irritants leads to the development of an inflammatory response in the airways; however, this inflammation is not confined to the bronchi. Morphological studies of the pulmonary artery in COPD patients have revealed inflammatory infiltration within its wall, composed of the same cell types as the bronchial infiltrate. Furthermore, a greater degree of vascular wall infiltration correlates with a higher severity of endothelial dysfunction in these vessels [2, 14].

Systemic inflammatory markers in COPD are associated with typical indicators of disease severity, such as decreased FEV1, and virtually all mechanisms significant for CVD development: arterial stiffness, endothelial dysfunction, the degree of RAAS activation, and cardiac remodeling. Moreover, atherosclerotic plaques have been found to contain inflammatory infiltrates similar to those observed in the lungs of COPD patients [12]. The level of systemic inflammatory markers significantly correlates with the clinical severity of both COPD and CVD. A more pronounced inflammatory response in both conditions leads to poorer disease outcomes [6].

While the primary cause of systemic inflammatory response activation in the presence of comorbid COPD and heart failure (HF) remains impossible to pinpoint, many researchers believe it serves as a predictor for the development of both cardiovascular and respiratory complications.

RAAS Activation. Currently, the activation of the RAAS in CVD is extensively studied and recognized as one of the central mechanisms of pathogenesis. RAAS activation is linked to impaired renal perfusion occurring against a background of hypoperfusion and fluid retention. The effects of renin, angiotensin II, and aldosterone are associated with the development of vasoconstriction, endothelial dysfunction, cell proliferation, and vascular fibrosis. These mechanisms, in turn, lead to the progression of atherosclerosis, vascular remodeling, alterations in cardiac workload, and subsequent cardiac remodeling, forming the cornerstone of CVD pathogenesis. Consequently, RAAS blockers are currently the drugs of choice for the treatment of most cardiovascular conditions [10, 12].

The significance of RAAS activation in COPD is considerably less studied than in CVD. It is hypothesized that the mechanism of RAAS activation in COPD, similar

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to CVD, is associated with impaired renal perfusion triggered by hypoxia and the activation of the sympathetic nervous system. Some studies have demonstrated that plasma renin activity in COPD patients increases as early as the initial stages of the disease and correlates with the level of systemic inflammation [6, 11].

Thus, RAAS activation appears to be a shared pathogenetic mechanism in the development of both diseases, serving as a critical link between them.

Endothelial Dysfunction. The endothelium produces a variety of mediators that play a pivotal role in regulating vascular tone and vascular remodeling, including endothelin, angiotensin II, thromboxane A2, and prostaglandin F2α. Currently, endothelial dysfunction is extensively studied and regarded as a key link in the pathogenesis of cardiovascular diseases (CVD) [2].

Disruption of endothelial integrity leads to an imbalance between vaso-constrictive and vasodilatory mediators, anti- and procoagulant agents, as well as growth factors and their inhibitors [10, 14]. These alterations result in vasoconstriction, fibrosis of the vascular wall, impairment of the mechanical properties of arteries, and the development of atherosclerosis [3, 12].

The cumulative effect of all CVD risk factors leads to hypertrophy of the vascular muscular layer and remodeling of all layers of the vascular wall, resulting in increased arterial stiffness [11]. Due to the reduced elasticity of the vascular wall, there is an increase in pulse wave amplitude and arterial blood pressure, which subsequently increases cardiac afterload. The combination of these factors leads to hemodynamic disturbances and forms the pathogenetic basis of CVD.

Signs of endothelial dysfunction are detectable as early as the initial stages of COPD and are significantly correlated with blood oxygen saturation levels and bronchial obstruction indices. Markers of endothelial dysfunction (endothelin-1, vascular endothelial growth factor, nitric oxide) correlate with the degree of inflammation (levels of CRP, interleukin-6, and fibrinogen), and as inflammation subsides, the levels of these markers also decrease. Increased arterial stiffness has emerged as a systemic manifestation of COPD independent of endothelial dysfunction; while not age-related, it correlates with the severity of inflammation and the extent of lung damage [4, 6].

Cardiac Remodeling. In COPD, cardiac remodelingis driven by several pathogenetic mechanisms shared with CVD. The processes leading to the trans-formation into chronic corpulmonaleare based on hypoxemia and hypercapnia, endothelial dysfunction, the effects of systemic inflammatory response markers, polycythemia, and pulmonary hypertension (PH) [11]. These mechanisms stem from a primary ventilation-perfusion mismatch, leading to the destruction of lung parenchyma (resulting in emphysema) and alterations in the right heart chambers, with subsequent involvement of both the pulmonary and systemic circulations [15, 18].

The prevalence of hypoxemia in COPD patients can only be estimated tentatively. Hypoxemia in COPD leads to a deterioration in the quality of life and exercise tolerance, increases the workload on skeletal muscles, and raises the risk of

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mortality. The main contribution to the progression of hypoxemia in COPD patients is made by the disruption of ventilation-perfusion ratios due to airflow limitation and the emphysematous destruction of the pulmonary capillary bed architecture [2]. Increasing hypoxemia and elevated pressure in the pulmonary circulation lead to impaired systolic function of the right chambers, as well as dilation of the right atrium and ventricle due to rising pulmonary artery pressure. The development of corpulmonaleas a result of cardiac remodeling in COPD significantly worsens the prognosis [4, 17].

Another mechanism leading to cardiac remodeling is endothelial dysfunction. The development of endothelial dysfunction underlies the mechanisms of coronary vessel damage and the progression of CVD, specifically heart failure (HF). In 30–40% of patients, the clinical presentation of HF is caused by impaired diastolic heart function and maladaptive remodeling. It has been proven that exercise tolerance and quality of life in patients with chronic HF correlate more strongly with restrictive diastolic dysfunction than with left ventricular systolic function. The pathogenesis of HF with preserved ejection fraction (HFpEF) differs from that of HF with reduced ejection fraction (HFrEF) and may be linked to the activation of systemic inflammatory components. This subsequently leads to endothelial dysfunction, increased arterial stiffness, and fibrosis of the cardiac tissue [11, 18]. The primary pathogenetic mechanism by which COPD affects the structural and functional state of hemodynamics is the development of pulmonary hypertension (PH) resulting from reflexive vasoconstriction in response to hypoxia (the Euler–Liljestrand reflex).

Progressive PH leads to right ventricular hypertrophy, the formation of corpulmonale, and, as a logical consequence, right-sided heart failure. PH is most closely associated with right ventricular (RV) systolic dysfunction and indicates advanced stages of lung disease. Changes in intracardiac hemodynamic parameters are detectable even in the early stages of COPD, prior to the development of significant PH. Furthermore, therapy aimed at correcting PH has minimal impact on subsequent cardiac remodeling.

In summary, patients with COPD and pre-existing CVD face a poorer prognosis compared to those without COPD. There is a heightened risk of new cardiovascular events, as mortality in these patients is primarily driven by cardiovascular causes [11]. The coexistence of COPD and CVD, compounded by shared risk factors, significantly impacts both prognosis and patient quality of life. It can be hypothesized that COPD serves as an independent predictor of adverse outcomes, thereby influencing the pathogenesis and clinical course of CVD [18]. Consequently, patients presenting with this comorbidity require a distinct risk stratification approach, as their risk of cardiovascular catastrophes may be substantially higher.

Beyond the mutual aggravation of these conditions through the summation of cardiovascular risk, they share several key components in their cytokine and vascular pathogenesis. At certain stages of progression, both COPD and CVD involve the activation of the RAAS and systemic inflammatory responses, as well as the development of hypoxemia and impaired trophism across various organs and systems.

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Vascular pathology is characterized by escalating endothelial dysfunction, subsequent arterial wall alterations, and cardiac remodeling. Despite certain differences, these pathogenetic milestones manifest similarly in both diseases, leading to the progression of COPD and CVD and a critical decline in cardiac contractile function.

The presence of shared pathogenetic pathways in patients with COPD and CVD may facilitate the development of new risk assessment criteria based on the severity of impairment and the involvement of inflammatory markers, the RAAS, and endothelial dysfunction indices. For patients with COPD who have existing CVD or associated risk factors, it is essential to re-evaluate strategies for preventing cardiovascular complications, particularly in light of existing clinical guidelines for cardiovascular prophylaxis. Modification of current risk factors and secondary prevention of CVD should be initiated in such patients as early as possible.

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