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THE RELATIONSHIP OF LIPID PROFILE INDICATORS WITH INFLAMMATORY MEDIATORS AND FIBRINOGEN IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract
Systemic lupus erythematosus (SLE) is an inflammatory rheumatic disease of autoimmune origin, which is more common in women. This disease involves a generalized inflammatory process that affects numerous internal organs and tissues. Patients with SLE also experience accelerated development of atherosclerosis and cardiovascular complications. The relationship between lipid metabolism disorders and inflammation plays a key role in this situation. This article analyzes the relationship between lipid metabolism disorders and inflammation in patients with systemic lupus erythematosus (SLE). Specifically, it examines the correlation of lipid profile parameters—total cholesterol, triglycerides, and high- and low-density lipoproteins—with acute-phase reactants such as C-reactive protein, ceruloplasmin, and fibrinogen. Based on an analysis of existing clinical and biochemical studies, the significance of these parameters and their potential use as prognostic markers are discussed.

Keywords: Systemic lupus erythematosus, dyslipidemia, C-reactive protein, fibrinogen, ceruloplasmin, inflammation, atherosclerosis, lipid profile.

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease belonging to a group of inflammatory systemic connective tissue diseases that leads to disruption of a number of metabolic processes in the body, including lipid

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metabolism. Dyslipidemias observed in patients with SLE are primarily caused by metabolic changes associated with inflammation. In medicine, this phenomenon is known as inflammation-related dyslipidemia.

In patients with SLE, total cholesterol levels can fluctuate. In some cases, they remain within the normal range, but more often, they tend to increase. This is especially evident during periods of high disease activity and with long-term treatment with steroids (glucocorticoids).

Glucocorticoids stimulate lipogenesis and disrupt glucose and lipid metabolism, which contributes to increased total cholesterol levels. Hypertriglyceridemia, or elevated triglyceride (TG) levels, is a common lipid metabolism disorder in SLE [1,5,9]. This phenomenon is primarily associated with the influence of cytokines produced during inflammation (e.g., TNF-α and IL-1). They stimulate increased synthesis of very low-density lipoproteins (VLDL) in the liver, inhibition of lipolysis, and the development of insulin resistance. As a result, TG levels increase, significantly increasing atherogenic risk. Low-density lipoproteins (LDL), or "bad" cholesterol, are one of the main atherogenic indicators of cardiovascular risk [8]. Patients with SLE often have elevated LDL levels, which is usually associated with impaired endothelial function due to inflammation, weakened antioxidant defenses, and lipid oxidation. High LDL levels contribute to the acceleration of atherosclerotic damage to vascular walls. High-density lipoproteins (HDL), or "good" cholesterol, perform an anti-atherogenic function and are involved in suppressing or controlling the immune response.

One of the main functions of HDL is the removal of excess cholesterol from the vessel walls and its transport to the liver. In SLE, especially during periods of high disease activity, HDL levels are significantly reduced. This limits the antioxidant and anti-inflammatory activity of HDL. As a result, atherogenic lipoproteins (LDL and ATDL) can accumulate on the vessel walls, contributing to the development of atherosclerosis. The development of dyslipidemia occurs due to interconnected pathological mechanisms occurring simultaneously [3,6]. First of all, a systemic inflammatory process leads to excessive production of inflammatory cytokines—interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and other biologically active molecules—which disrupts the normal regulation of lipid metabolism in the liver. As a result, very low-density

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lipoprotein (VLDL) and triglyceride (TG) levels increase, while high-density lipoprotein (HDL) levels decrease. This condition is called "inflammatory-induced dyslipidemia," characterized by a shift in lipid balance toward an atherogenic direction compared to a healthy state. Furthermore, inflammation weakens the antioxidant and anti-atherogenic functions of HDL, altering its structure and making it less active. It is also important to note that glucocorticoids (prednisolone and similar drugs), widely used in the treatment of systemic lupus erythematosus, have a significant effect on lipid metabolism. They increase gluconeogenesis and the synthesis of very low-density lipoproteins (VLDL) in the liver, which leads to an increase in total cholesterol (TC) and triglycerides, and may also be accompanied by a decrease in HDL levels. Such hyperlipidemic conditions are often observed during treatment and are directly related to drug exposure. Another characteristic metabolic consequence of systemic lupus erythematosus (SLE) is the development of insulin resistance. As a result of inflammation and hormonal changes, cellular sensitivity to insulin decreases, leading to impaired lipid utilization in the body. The liver increases the synthesis of very low-density lipoproteins (VLDL) and increases triglyceride levels, while high-density lipoprotein (HDL) levels decrease. These mechanisms contribute to an increased risk of metabolic syndrome and atherosclerosis. In patients with SLE, fatigue, arthritis, myalgia, and other clinical manifestations lead to limited physical activity, which, in turn, leads to decreased energy expenditure and accumulation of fatty acids in the body. In the context of physical inactivity, lipid oxidation slows, contributing to increased lipid levels in the blood. Furthermore, an imbalance between inflammation and the antioxidant system leads to the development of oxidative stress. Reactive oxygen radicals cause oxidation of low-density lipoproteins (LDL), increasing their atherogenicity, and simultaneously reduce the functional activity of HDL [10]. Oxidative stress impairs the activity of antioxidant enzymes (ceruloplasmin, superoxide dismutase, and others), further exacerbating the pathological redistribution of lipids. All these pathogenic processes combine to create an atherogenic environment in the body, increasing the risk of cardiovascular disease. Therefore, regular monitoring of the lipid profile and analysis of inflammatory biomarkers is crucial for patients with systemic lupus erythematosus (SLE). These changes

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are often due to **cytokine exposure, hormonal therapy, decreased physical activity, and metabolic syndrome**. Therefore, assessment of patients' condition should be based not only on cholesterol levels but also on the severity of the inflammatory process.

In autoimmune and inflammatory diseases, such as systemic lupus erythematosus (SLE), the body produces a number of specific proteins in response to inflammatory activity. These proteins, known as acute-phase reactants, are synthesized primarily in the liver. Their levels are elevated by cytokines released by the immune system, particularly interleukin-6 (IL-6), interleukin-1 (IL-1), and tumor necrosis factor alpha (TNF-α). These proteins are early markers of inflammation, and their elevated levels are important for assessing disease activity, its severity, and the risk of cardiovascular complications [4]. C-reactive protein (CRP) is one of the most sensitive and widely used markers of inflammation. Its synthesis in the liver is sharply increased by interleukin-6. CRP binds to the phospholipids of damaged cell membranes in inflamed tissues, activating phagocytosis and maintaining the inflammatory response. In patients with SLE, CRP levels are often elevated, indicating disease activity. Furthermore, CRP promotes the activation of atherosclerotic processes by disrupting endothelial function and promoting the adhesion of lipoproteins to the vascular wall. These properties make CRP also an important biomarker of atherothrombosis [12].

Ceruloplasmin is a copper-transporting glycoprotein synthesized in the liver. It serves as an antioxidant: as an enzyme, it converts Fe^{2+} ions to Fe^{3+}, thereby limiting the formation of free radicals. During inflammation, ceruloplasmin levels increase because it helps counteract oxidative stress in tissues. In systemic lupus erythematosus (SLE), elevated ceruloplasmin levels indicate not only inflammation but also tissue damage and depletion of cellular antioxidant systems. In this situation, high ceruloplasmin levels can contribute to the development of atherosclerosis, as an imbalance in antioxidant balance leads to the oxidation of lipoproteins and, consequently, damage to vascular walls.

Fibrinogen is a key component of the blood coagulation system, its primary function being the formation of the fibrin network, which facilitates platelet aggregation. It is also an acute-phase reactant protein synthesized in the liver in

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response to inflammation. Elevated fibrinogen levels are frequently observed in systemic lupus erythematosus (SLE) and other autoimmune diseases. This condition leads to increased blood clotting, impaired microcirculation, and creates the preconditions for ischemic changes. Particularly high fibrinogen levels increase the tendency for blood clots to form in the cardiovascular system, which increases the risk of stroke, myocardial infarction, and other dangerous complications [1].

Biomarkers such as C-reactive protein (CRP), ceruloplasmin, and fibrinogen are essential for assessing the inflammatory process in patients with systemic lupus erythematosus (SLE), as well as for the early detection of cardiovascular complications and predicting their risk. Regular monitoring of these biomarkers allows physicians to assess disease activity, monitor treatment effectiveness, and promptly take measures to prevent cardiovascular complications. Thus, these biomarkers have high diagnostic and prognostic value in the early detection of atherothrombosis, activation of blood clotting processes, and vascular damage [2].

In patients with systemic lupus erythematosus (SLE), there is a clear correlation between lipid metabolism disorders and inflammatory markers, which plays a significant role in the pathogenesis of the disease and the development of cardiovascular complications. Various clinical studies show that lipid profiles systematically change during inflammation. Firstly, there is a clear positive correlation between C-reactive protein (CRP) levels and triglyceride (TG) and very low-density lipoprotein (VLDL) levels. In other words, if CRP levels are elevated, TG and VLDL levels are also typically elevated. This reflects the direct negative impact of inflammation on lipid metabolism in the body. Furthermore, elevated CRP levels damage the vascular endothelium, promoting the accumulation of lipoproteins in the vascular lining, which, in turn, enhances atherogenic processes. Secondly, there is an inverse (negative) correlation between CRP and high-density lipoprotein (HDL) [11]. As inflammation increases, HDL levels tend to decrease. HDL is considered a protective factor against atherosclerosis, as it is involved in the removal of cholesterol from the vascular wall. A decrease in the concentration of these molecules leads to a weakening of the body's defenses against the cardiovascular system. Equally

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important is the condition associated with fibrinogen—a protein known not only for its role in blood clotting but also as a marker of inflammation. Patients with elevated fibrinogen levels typically also have high levels of triglycerides (TG) and very low-density lipoproteins (VLDL). This positive correlation indicates increased thrombogenicity (i.e., a tendency toward blood thickening) and an atherogenic state. In other words, high fibrinogen levels are associated with an increased risk of blood clots and accelerated atherosclerotic processes [7].

A relationship was also found between ceruloplasmin levels and dyslipidemia indicators. Ceruloplasmin is a protein with antioxidant properties, the level of which increases in inflammatory conditions. However, it can also indicate depletion of the body's antioxidant defense system. Elevated ceruloplasmin levels are typically accompanied by an increase in atherogenic lipids—very low-density lipoproteins (VLDL) and triglycerides (TG). This suggests that the combination of inflammation and oxidative stress increases the risk of atherosclerosis. Based on these correlations, it can be concluded that metabolic and inflammatory processes are closely interrelated in patients with systemic lupus erythematosus (SLE). Therefore, in these patients, it is important to simultaneously monitor both the lipid profile and inflammatory indicators, which allows for a more accurate risk assessment, effective disease management, and the prevention of cardiovascular complications [6]. Furthermore, a number of literature sources note that the combined analysis of lipid and inflammatory parameters in clinical practice opens up a number of important opportunities. Firstly, this approach enables the early detection of the risk of developing cardiovascular complications, particularly atherosclerosis and thrombosis. Elevated levels of dyslipidemia and inflammatory markers are considered the main pathogenetic factors for these complications, so preventive measures can be taken proactively based on these indicators. Secondly, the dynamics of biomarkers can be used to assess the degree of disease activity. Thus, elevated levels of inflammatory and lipid parameters indicate the active phase of the disease, while their normalization indicates remission [12]. This, in turn, allows the physician to individually adjust the treatment strategy. Thirdly, such indicators play an important role in therapeutic monitoring, that is, in assessing the effectiveness of the treatment. For example, if C-reactive protein levels decrease and the lipid profile improves while

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taking glucocorticoids, immunosuppressants, or lipid-lowering drugs, this indicates positive dynamics and the effectiveness of the treatment. Thus, these indicators have diagnostic and prognostic value in assessing the patient's condition, monitoring treatment results, and predicting potential complications.

Conclusion

In patients with systemic lupus erythematosus, clear correlations are observed between biochemical parameters related to lipid metabolism and inflammatory proteins. These correlations accelerate the development of cardiovascular complications and worsen the prognosis of the disease. Therefore, systematic assessment and monitoring of these parameters are essential for the effective management of patients with SLE. Comprehensive analysis of the lipid profile and inflammatory markers facilitates early diagnosis and targeted therapy in clinical practice.

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