COMPLEMENT SYSTEM AS A BIOMARKET OF CARDIOVASCULAR DISEASES

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The diseases of the circulatory system occupy the first place in the structure of morbidity and mortality not only in the Republic of Belarus, but also around the world. In most cases, this is due to their untimely detection. Therefore, it is so important to detect the disease in the early stages and in the future to deal with the causes of its appearance. In this case, the complement system can play an important role.

Keywords: complement system, biomarkers, vascular remodeling, low-density lipoproteins.

In recent years, the ambiguity of the complement system in the pathogenesis of some cardiovascular diseases has been emphasized. It is noted that the complement system can participate in vascular remodeling. This occurs by the following mechanisms: from the initial protective response, which is aimed at removing cellular debris, to a potentially harmful role. Complement activation is the primary mediator linking the two main factors of pathological vascular remodeling, namely lipid/protein deposition and modification in the vessel wall and inflammatory response in the body. The processes underlying pathological vascular remodeling include lipid accumulation, cell proliferation, redox imbalance, proteolysis, leukocyte infiltration, cell death, and ultimately thrombosis.

Numerous studies have shown that complement components are present in both atherosclerotic plaques and abdominal aortic aneurysms [1,2]. Monocytes produce proteins of complement in response to the accumulation of cholesterol. Modified low-density lipoproteins can trigger the activation of the complement system. The interaction between coagulation and complement systems can also be an important trigger of complement activation in vascular tissues, so far as coagulation enzymes activate complement components and Vice versa. Thus, both local synthesis of some components and their absorption from plasma can contribute to increasing the level of complement in vascular tissues.

A number of studies have examined the potential role of complement proteins as diagnostic and predictive biomarkers of cardiovascular disease. Thus, C3a was associated with increased carotid artery thickness in a large cohort of subjects [3]. Elevated C3a levels were observed in a small cohort of patients with familial hypercholesterolemia. An elevated serum C3/C4 ratio has been proposed as a marker in acute coronary syndrome. Recently, a high level of C5b9 is an independent risk factor for acute ischemic stroke [2]. In addition, reduced C1q complement levels are associated with an increased risk of overall mortality after 10 years in diabetic patients referred for coronary angiography [4]. Hemolytic complement activity (CH50) is associated with subclinical atherosclerosis in patients with systemic lupus erythematosus [5].

In general, complement components are potential diagnostic and prognostic biomarkers of atherosclerosis, abdominal aortic aneurysm, and other circulatory diseases. However, whether they can be useful alone or in combination with other biomarkers for stratification of patients in clinical settings deserves further study.

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