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Perspective

Peripheral Granulocytes and Dyslipidemia in People with Hyperlipidemia

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DESCRIPTION

Strong evidence suggests that the pro-inflammatory effects of Triglyceride-Rich Lipoproteins (TRLs) are at least largely responsible for their detrimental impact on the onset and progression of atherosclerosis. However, there has not yet been any research on how Hypertriglyceridemia (HTG) affects the subpopulation makeup of circulating neutrophils. This study looked at connections between circulating neutrophil subpopulation makeup and Triglyceride (TG) levels in middle-aged patients with dyslipidemia who did not yet have established Atherosclerotic Cardiovascular Illnesses (ASCVDs). The study included 91 individuals with dyslipidemia; including 22 (24.2%) patients with HTG Flow cytometry was used to phenotype neutrophil sub-populations (Navios 6/2, Beckman Coulter, USA). Conjugated monoclonal antibodies were employed to phenotype neutrophil subpopulations, including CD16, PE-Cyanine7 (Invitrogen, USA), CD11b-FITC (Beckman Coulter, USA), CD62L-PE (Beckman Coulter, USA), and CD184 (CXCR4)-PE-CF594 (BD Biosciences, USA) [1].

The quantity of circulating neutrophils with the phenotypes $CD16^{hi}CD11b^{hi}CD62L^{hi}$ and $CD16^{hi}CD11b^{lo}CD62L^{br}$ directly linked with TG levels. After doing a linear regression analysis, it was shown that there were statistically significant connections between TG levels and the neutrophil subpopulations $CD16^{hi}CD11b^{lo}CD62L^{br}$ and $CD16^{hi}CD11b^{br}CD62L^{lo}CXCR4^{hi}$. Up to 19.1% of the variation in the number of examined neutrophil subpopulations might be attributed to changes in TG levels [2].

Those with HTG showed significantly more neutrophils overall, neutrophils with $CD16^{hi}CD11b^{hi}CD62L^{hi}$ (mature neutrophils), and neutrophils with $CD16^{hi}CD11b^{lo}CD62L^{br}$ (immune-suppressive neutrophils) than patients with normal TG levels among middle-aged patients without known ASCVDs. Adjusted for the patients' sex and age, the TG level was linked to an increase in the number of $CD16^{hi}CD11b^{lo}CD62L^{br}$ and $CD16^{hi}CD11b^{br}CD62L^{lo}CXCR4^{hi}$ (ageing neutrophils) neutrophils.

A lipid condition that is quite frequent is Hypertriglyceridemia (HTG). Therefore, the incidence of HTG is 25% in the general population and over 30% in individuals receiving statin therapy, according to the US National Health and Nutrition Examination Surveys. HTG was found in 29.2% of Russians, according to the PROMETHEUS study (the Prevalence of Mixed Dyslipidemia and Severe Hypertriglyceridemia in the Russian Population). But in different patient categories, HTG is a factor that is independently linked to the emergence of unfavourable cardiovascular events.

Strong evidence suggests that the pro-inflammatory effects of triglyceride-rich lipoproteins (TRLs) are at least largely responsible for their detrimental impact on the onset and progression of atherosclerosis. Increased integrin expression (CD11b, CD11c, and CD18) on circulating monocytes as well as leukocytosis, neutrophilia, and monocytosis have all been linked to higher TRL levels. The development and maintenance of vessel wall inflammation are consequently influenced by this. The progression of atherosclerotic lesions is determined by monocytes and their offspring macrophages at all stages, according to current knowledge. However, neutrophils' significance in atherogenesis and their contribution to atherosclerosis have long been underappreciated. Neutrophil biology findings that led to a reevaluation of conventional wisdom on their roles and population heterogeneity have only recently resulted in a significant increase in the research of neutrophils in atherosclerosis. As a potential therapeutic target for the treatment of atherosclerosis, neutrophils are now being given consideration [3].

The importance of different subtypes of neutrophils may vary depending on the stage of atherosclerosis development, which may have practical implications. Dyslipidemia, a major risk factor for atherosclerosis, can change the makeup of the neutrophil subpopulation. However, little research has been done on how HTG affects the neutrophils that are in the blood. If neutrophils are incubated in a TRL-enriched emulsion, Alipour et al. demonstrated that there is a dose-dependent two- to three-fold increase in CD11b and CD66b expression.

HTG's impact on the subpopulation makeup of circulating neutrophils hasn't yet been researched, though. This study looked at connections between circulating neutrophil subpopulation makeup and Triglyceride (TG) levels in middle-

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aged patients with dyslipidemia who did not yet have established Atherosclerotic Cardiovascular Illnesses (ASCVDs). The study included patients with dyslipidemia without ASCVDs aged 40 to 64. Because there is a large increase in the risk of adverse cardiovascular events among patients over 40 compared to younger age groups, thorough cardiovascular risk assessment is advised for this group of patients according to several clinical standards. Patients with ASCVD were excluded from the study because the presence of ASCVDs associated with severe prolonged atherosclerosis may have a major impact on the makeup of the circulating innate immune cell pool itself [4].

Signed informed consent was a requirement for patient participation in the trial. The South Ural State Medical University Ethics Committee gave its approval to the study protocol. Total Cholesterol (TC) >4.9 mmol/L, low-density lipoprotein cholesterol (LDL-C) >3.0 mmol/L, TGs >1.7 mmol/L, and high-density lipoprotein cholesterol (HDL-C) 1.0 mmol/L in males or 1.2 mmol/L in women were the criteria for dyslipidemia. A rise in fasting TG >1.7 mmol/L was considered HTG [5].

Previously diagnosed ASCVDs (a history of cerebrovascular disease, coronary artery disease, peripheral artery disease, and coronary and peripheral artery revascularization), severe hepatic and renal dysfunctions (a decrease in Glomerular Filtration Rate (GFR) of more than 30 mL/min/1.73 m2), malignant neoplasms, established Chronic Inflammatory Diseases (CIDs), and acute inflammatory or infectious diseases were all used as exclusion criteria for the HTG.

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