THE ROLE OF LIPOPROTEINS AND RECEPTORS IN THE PATHOGENESIS OF ACUTE CEREBROVASCULAR ACCIDENTS: BIOMARKER ANALYSIS AND STROKE RISK EVALUATION

Nargiza Vakhabova

Tashkent medical academy, neurology department

Introduction

Atherosclerosis is a significant contributor to cerebrovascular disease, primarily driven by dyslipidemia and inflammatory processes. These factors promote the formation of atheromatous plaques, which can lead to cerebrovascular events such as stroke. Lipoproteins, including lipoprotein A (Lp(a)), and their receptors play a critical role in this process. Despite advancements in lipid-lowering therapies, the pathogenesis of atherosclerosis remains poorly understood, and effective stroke prevention strategies are limited. This study aims to assess the relationship between lipoproteins and their receptors in patients with acute cerebrovascular accidents (ACVA) and to evaluate their predictive value for stroke risk.

Methods

This cross-sectional study involved 165 patients diagnosed with ACVA, including those with ischemic stroke and transient ischemic attacks, between [2022] and [2024]. Patients underwent comprehensive biomarker assessments, including enzyme immunoassays for Lp(a) and lipoprotein-associated phospholipase A2 (Lp-PLA2). Biomarkers were measured using standardized protocols (Elabscience Human Lipoprotein Lp-a ELISA Kit and Lipoprotein A2 Phospholipase ELISA Kit). General blood tests and lipid profiles were also performed. Statistical analyses were conducted using IBM SPSS Statistics 19, with independent t-tests and Pearson's correlation coefficients. Significance was set at $P < 0.05$.

Results

The study revealed significant associations between lipoprotein levels and the severity of neurological deficits in stroke patients. Lp(a) levels increased by 2.3 times in patients with severe neurological deficits (206.5±23.3 mg/ml, $P < 0.01$) compared to those with mild deficits. LDL receptor (LDLR) levels decreased by 2.2 times (0.240±0.01 ng/ml, $P < 0.01$) in severe cases. Lectin-like oxidized LDL receptor (LOX-1) levels were significantly elevated (0.300±0.11 ng/ml, $P < 0.01$), highlighting its role in plaque instability and inflammation. Thrombomodulin (TM) levels were reduced by 1.8 times (5.5±0.18 mg/l, $P < 0.01$) in severe cases. Lp-PLA2 levels were also elevated in patients with severe neurological deficits, with a 1.2-fold increase ($P < 0.05$).

Discussion

The study findings underscore the role of dyslipidemia, particularly elevated Lp(a) and LOX-1 levels, in the progression of atherosclerosis and cerebrovascular events. Lp(a) has been shown to promote thrombus formation and plaque development, contributing to increased stroke risk. Decreased LDLR levels, which impede LDL clearance from the bloodstream, further elevate cardiovascular disease risk. Elevated Lp-PLA2 levels were associated with heightened inflammation, suggesting that these biomarkers could serve as potential targets for therapeutic interventions. Early detection of lipid dysregulation and targeted treatments aimed at modulating lipoproteins may reduce the risk of stroke in high-risk populations.