LIPOPROTEINS AND THEIR RECEPTORS IN THE PATHOGENESIS OF CEREBROVASCULAR DISEASE: ASSOCIATIONS WITH STROKE RISK AND NEUROLOGICAL DEFICITS

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Introduction

Atherosclerosis is a leading cause of cardiovascular and cerebrovascular diseases, primarily driven by dyslipidemia and inflammatory processes. Despite the availability of lipid-lowering treatments, atherosclerosis continues to be a significant contributor to mortality worldwide. The pathogenesis of atherosclerosis involves the complex interaction of genetic, environmental, and inflammatory factors, adversely affecting lipid transport and metabolism. Emerging evidence suggests that lipoproteins such as lipoprotein A (Lp(a)) and their receptors play a crucial role in cerebrovascular accidents, particularly strokes. This study aims to assess the relationship between lipoproteins and their receptors in patients with acute cerebrovascular accidents and to evaluate their predictive value for stroke risk using Receiver Operating Characteristic (ROC) analysis.

Methods

This cross-sectional study was conducted at [Hospital Name] from [Start Date] to [End Date], involving 165 patients diagnosed with acute cerebrovascular accidents (ACVA). Comprehensive assessments were performed, including enzyme immunoassays for Lp(a) and lipoprotein-associated phospholipase A2 (Lp-PLA2). General blood tests, electrocardiography (ECG), and standard biochemical analyses were conducted. Biomarker measurements were performed using enzyme immunoassays for Lp(a) and Lp-PLA2 with standardized protocols. Data analysis included descriptive statistics, t-tests or Mann-Whitney U tests, and Pearson's or Spearman's correlation coefficients using IBM SPSS Statistics 19. A p-value of less than 0.05 was considered statistically significant.

Results

The study demonstrated a significant association between lipoprotein levels and the severity of neurological deficits in stroke patients. Lp(a) levels increased by 2.3 times ($P < 0.01$) in severe neurological deficits, while low-density lipoprotein receptor (LDLR) levels decreased by 2.2 times ($P < 0.01$) in these patients. Elevated levels of lectin-like oxidized LDL receptor (LOX-1) and Lp-PLA2 were also observed, correlating with the development of neurological deficits. A 1.8-fold decrease in thrombomodulin (TM) levels was also noted in patients with severe deficits. These findings highlight the critical role of lipid metabolism, thrombus formation, and endothelial dysfunction in the progression of cerebrovascular accidents.

Discussion

The findings underscore the role of dyslipidemia, particularly elevated Lp(a) and LOX-1 levels, in promoting atherosclerosis and cerebrovascular events. Lp(a) is known to accelerate plaque formation and promote thrombus development, contributing to stroke risk. Similarly, decreased LDLR levels are consistent with elevated plasma LDL concentrations, further increasing cardiovascular risk. The elevated levels of Lp-PLA2 and LOX-1 indicate ongoing inflammation and plaque instability in stroke patients. This study highlights the importance of early detection and targeted interventions to modulate lipoprotein levels and reduce inflammation in order to mitigate stroke risk. Future research should explore longitudinal designs to assess the predictive value of these biomarkers in stroke progression.