METABOLIC BIOMARKERS AND HEMODYNAMIC IMPAIRMENT IN ACUTE ISCHEMIC STROKE: AN INTEGRATED CLINICAL AND INSTRUMENTAL ANALYSIS

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Introduction

Ischemic stroke remains one of the leading causes of mortality and long-term disability worldwide. Increasing evidence suggests that disturbances in calcium–phosphorus homeostasis and lipid metabolism contribute to vascular calcification, endothelial dysfunction, and progression of atherosclerosis. However recent studies highlight the “vascular-bone axis” as a critical factor in stroke pathogenesis, where dysregulation of mineral homeostasis—specifically calcium and phosphorus—promotes medial arterial calcification and increases arterial stiffness. This process, coupled with dyslipidemia, accelerates the formation of unstable carotid plaques and impairs compensatory hemodynamic mechanisms. Furthermore, Vitamin D deficiency has been linked to impaired nitric oxide bioavailability and heightened neuro-inflammation, potentially exacerbating the severity of ischemic brain injury. Despite these insights, the synergistic predictive value of integrated biochemical and duplex-derived hemodynamic parameters in acute stroke management remains poorly defined.

Objective

To investigate the association between calcium–phosphorus metabolism disorders, lipid profile parameters, and clinical severity, neuroimaging findings, and brachiocephalic hemodynamic changes in patients with acute ischemic stroke.

Methods

This prospective study included 80 patients with acute ischemic stroke (mean age 64.2±8.4 years) and 20 healthy controls. Patients were categorized into two groups: Group I (n=40) with confirmed dysmetabolism (disturbed Ca-P and lipid profiles) and Group II (n=40) with normal metabolic parameters. Neurological deficit was quantified using the NIHSS scale. Laboratory markers included serum calcium (Ca), phosphorus (P), alkaline phosphatase (ALP), vitamin D (25(OH)D), and a full lipid panel (TC, LDL-C, HDL-C, TG). Instrumental diagnostics involved brain CT for infarct volume

assessment and duplex ultrasonography of brachiocephalic arteries to measure intima-media thickness (IMT) and blood flow velocities (Vps, Ved).

Statistical analysis comprised independent t-test, Mann–Whitney U-test, Pearson/Spearman correlation analysis, multiple linear regression, logistic regression, and ROC curve analysis. Odds ratios (OR) with 95% confidence intervals (CI) were calculated.

Results

Patients in Group I exhibited significantly higher NIHSS scores (14.2±3.1 vs. 9.4±2.5; $p<0.01$) and larger infarct volumes on CT compared to Group II. Strong positive correlations were identified between stroke severity and serum levels of calcium ($r=0.48$, $p<0.01$), phosphorus ($r=0.42$, $p<0.01$), and LDL-C ($r=0.51$, $p<0.001$). Conversely, vitamin D levels were inversely proportional to NIHSS scores ($r=-0.44$, $p<0.01$). Duplex scanning revealed a higher prevalence of unstable atherosclerotic plaques and increased IMT in Group I (1.24±0.12 mm vs. 0.98±0.08 mm; $p<0.05$). Multivariate logistic regression identified combined Ca-P and lipid disturbances as independent predictors of severe stroke (OR 3.2; 95% CI 1.8–5.6). ROC analysis for the integrated metabolic-hemodynamic model demonstrated high diagnostic accuracy with an AUC of 0.86.

Conclusions

Disturbances in calcium–phosphorus and lipid metabolism are significantly associated with increased stroke severity, greater atherosclerotic burden, and impaired brachiocephalic hemodynamics. Integrated biochemical and instrumental assessment improves risk stratification and may support the development of individualized therapeutic strategies in ischemic stroke.