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Metabolomic characterization of coronary artery diseases and key role of N-Acetyl-neuraminic acid

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Statement of the Problem: Artery disease (CAD) remains a leading cause of mortality worldwide. The current clinical diagnosis differentiates among the types of CAD mainly dependents on coronary angiography. High cost limit it to a select population. On one hand, a sizable portion of individuals who underwent invasive angiography had been shown to have normal coronary arteries. On the other hand, episodes of myocardial ischemia or infarction are possible after atypical symptoms in some patients with CAD, especially in patients who are elderly or have diabetes. Additionally, the transition from coronary stability to instability is less well understood. The purpose of this study was to characterize different types of CAD by metabolomics and assess the diagnostic value of metabolomics-based biomarkers in different types of CAD. Meanwhile, translate metabolomic-derived biomarkers to disease mechanisms.

Methodology & Theoretical Orientation: A cohort of 2,324 patients who underwent coronary angiography from four independent centers was studied. Plasma metabolomic profiles were determined by liquid chromatography–quadrupole time-of-flight mass spectrometry and were analyzed by multivariate statistics. Significant differential metabolites were identified by cross comparisons with and within CAD groups including normal coronary artery (NCA), nonobstructive coronary atherosclerosis (NOCA), stable angina (SA), unstable angina (UA), and acute myocardial infarction (AMI).

Findings: We characterized the metabolomic profiling of CAD and screened out potential markers that can be used for diagnosis and predication clinic types of CAD. N-Acetyl-neuraminic acid (Neu5Ac) was highly elevated in plasma during CAD progression.

Conclusion & Significance: Plasma metabolomics are powerful for characterizing metabolic disturbances for CAD. Differences in small-molecule metabolites may reflect underlying CAD and serve as biomarkers for CAD progression. Functional metabolomics identified a key role for N-Acetyl-neuraminic acid in CAD and targeting neuraminidase-1 by pharmacological inhibitors oseltamivir and zanamivir represent an unrecognized therapeutic intervention for CAD.

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