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# INTERRELATIONSHIP OF CARDIOMETABOLIC RISK FACTORS AND SYMPATHOADRENAL DYSREGULATION IN YOUNG PATIENTS WITH NEW-ONSET ANGINA PECTORIS

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**Background:** Premature coronary artery disease (CAD), initially manifesting as new-onset angina pectoris in young adults, represents an escalating clinical challenge. Chronic hyperactivation of the sympathoadrenal system (SAS) combined with metabolic derangements accelerates endothelial injury and coronary plaque instability. In modern clinical cardiology, increasing attention is directed toward subclinical neuroendocrine disturbances as independent predictors of coronary hemodynamic destabilization. Subtle variations in neurohumoral balance can trigger atherogenic dyslipidemia, exacerbate arterial stiffness, and accelerate coronary plaque progression. The exact mechanisms linking SAS functional status with specific cardiometabolic clusters require further clarification to improve prognostic stratification.

**Objective:** To investigate the pathogenetic interrelationship between cardiometabolic risk clusters and functional disturbances of the sympathoadrenal system in young individuals with newly diagnosed angina pectoris, and to establish a prognostic algorithm for clinical progression.

**Materials and Methods:** A prospective cohort study enrolled 90 young patients (aged 25–44 years; mean age 36.8 ± 4.6 years) with first-diagnosed stable or progressive angina pectoris, compared with 30 age- and sex-matched healthy controls. Cardiometabolic profiling included anthropometric indices (visceral adiposity index), lipid fractions (ApoB/ApoA1 ratio, LDL-C), glycemic status, and insulin resistance (HOMA-IR). Sympathoadrenal tone was evaluated via 24-hour urinary catecholamine excretion (epinephrine, norepinephrine, dopamine) and time/frequency-domain heart rate variability (HRV) parameters (SDNN, LF/HF ratio) using 24-hour Holter ECG monitoring. Statistical processing was executed with SPSS version 26.0, employing Student's t-test and Spearman correlation analysis ($p < 0.05$).

**Results and Conclusions**

Young patients with new-onset angina exhibited pronounced sympathetic predominance, evidenced by elevated 24-hour urinary norepinephrine excretion (68.4 ± 9.2 µg/24h vs. 34.1 ± 5.8 µg/24h in controls; $p < 0.001$) and an elevated LF/HF ratio (3.42 ± 0.61 vs. 1.48 ± 0.29; $p < 0.001$). Significant positive correlations were established between daily norepinephrine levels and HOMA-IR ($r = 0.69$; $p < 0.001$), visceral adiposity ($r = 0.58$; $p < 0.01$), and the ApoB/ApoA1 ratio ($r = 0.61$; $p < 0.001$). Urinary dopamine showed a significant negative correlation with norepinephrine ($r = -0.52$; $p < 0.01$), indicating depletion of dopaminergic reserve under chronic sympathetic stress. Multivariate logistic regression demonstrated that concurrent SAS hyperactivation (urinary norepinephrine > 65 µg/24h) and insulin resistance (HOMA-IR > 2.8) increased the 12-month risk of



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adverse coronary events (unstable angina progression or urgent revascularization) by 3.4-fold (OR = 3.42; 95% CI: 1.84–6.35; p < 0.001).
In conclusion, first-onset angina pectoris in young adults is driven by a synergistic neurohumoral-metabolic vicious cycle. Integrating catecholamine profiling and HRV spectral analysis with cardiometabolic indices enables early risk stratification and targeted preventive pharmacotherapy.

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