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Ischemic Heart Disease

MICRORNA-137 AND MICRORNA-106B ARE NOVEL MYOCARDIAL ISCHEMIA/INFLAMMATION-DEPENDENT BIOMARKERS WITH HIGH DIAGNOSTIC SENSITIVITY AND SPECIFICITY FOR ACUTE CORONARY SYNDROME (ACS)

Oral Contributions
B402
Sunday, April 7, 2024, 10:34 a.m.-10:44 a.m.

Session Title: Highlighted Original Research: Ischemic Heart Disease and the Year in Review
Abstract Category: 22. Ischemic Heart Disease: Clinical Science
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Authors: Salwa A. Elgebaly, W. Frank Peacock, IV, Michael A. Azrin, Emerson C. Perin, Nashwa El-Khazragy, Robert Fuller, Don Kreutzer, Robert H. Christenson, Nour Heart, Inc., Vienna, VA, USA, University of Connecticut School of Medicine, Farmington, CT, USA

Background: Although troponin is able to detect myocardial necrosis, there is a need for biomarkers that could identify acute myocardial ischemia without necrosis in a population with suspected Acute Coronary Syndrome (ACS). Here we characterize the gene expression levels of two disease-specific microRNA (miR) regulators that are linked to ACS pathology; one for myocardial ischemia (miR-137) and another for inflammation (miR-106b). Bioinformatics analysis confirmed the specificity of miR-137 & miR-106b in ACS by the absence of their upregulation in non-ischemic patients with comorbidities.

Methods: Using a quantitative PCR assay, expression levels of miR-137 & miR-106b and high sensitivity troponin were measured in serum from healthy subjects (n=16) (baseline), non-ischemic patients with comorbidities (n=37) (controls), & ischemic subjects confirmed by coronary angiography to have unstable angina (UA, n=73) or acute myocardial infarction (AMI, n=86).

Results: MiR-137 & miR-106b are significantly expressed in UA and AMI patients, but not in healthy & controls (Figure 1). With confidence interval of 95%, they showed sensitivity of >98% and specificity of >97% for classifying controls (n=37) & ACS (n=159) groups. Troponin is elevated in AMI, not UA.

Conclusion: The novel ischemia/inflammation-dependent miR-137 & miR-106b are promising biomarkers that could identify a population of patients with ischemia, but without injury or infarction, thus potentially improving the treatment algorithms.

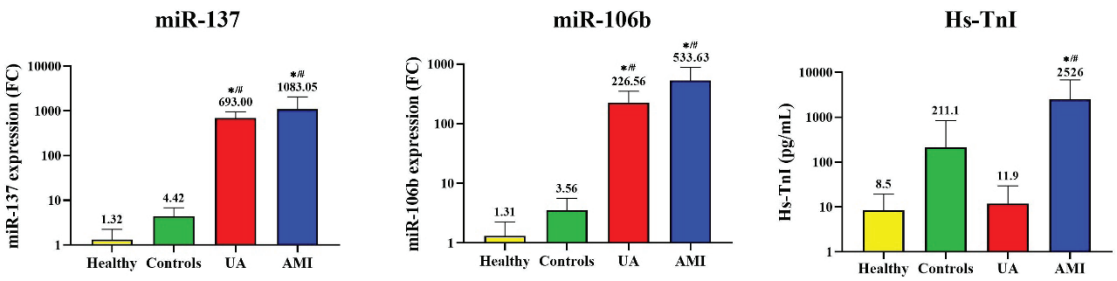

Figure 1: Bar chart illustrating gene expression levels of miR-137 and miR-106b in healthy subjects (n=16), non-ischemic (7 symptomatic and 30 asymptomatic) controls with comorbidities (n=37), and ACS patients (UA, n=73 and AMI, n=86), as well as levels of high sensitivity troponin I (hs-TnI) in healthy subjects (n=16), non-ischemic (negative stress imaging), symptomatic controls with comorbidities (n=7), UA patients (n=73), and AMI patients (n=16). Data is presented as mean and SE of the mean. FC: Fold change, *: Significant difference compared to healthy group ($p<0.001$), and #: Significant difference compared to control group ($p<0.001$).