DOWREGULATION OF RNA BINDING PROTEIN QKI6 MEDIATES SUSCEPTIBILITY OF MYOCARDIUM TO ISCHEMIA/REPERFUSION INJURY IN A TYPE 2 DIABETES MODEL, LEPTIN-DEFICIENCY MOUSE

Poster Contributions
Hall C
Sunday, March 30, 2014, 3:45 p.m.-4:30 p.m.

Session Title: Acute Coronary Syndromes: Basic II
Abstract Category: 2. Acute Coronary Syndromes: Basic
Presentation Number: 1224-223

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Backgrounds: When patients with diabetes develop myocardial infarction, they suffered from larger infarction than non-diabetic patients. Previous study of our lab showed that RNA-binding protein QKI inhibited ischemia/reperfusion-induced apoptosis in neonatal cardiomyocytes. The present study was designed to test whether QKI protein was involved in the increased susceptibility of myocardium in diabetic model.

Methods: This study was performed in 8-to-10-wk-old leptin-deficient obese (ob/ob) mice and compared with wild-type C57BL/6J (WT) mice. All animals underwent 30 min of coronary artery occlusion followed by 6 h of reperfusion. Adenovirus expressing QKI6 was constructed and injected into the anterior wall of heart to overexpress QKI6. Infarct size was determined after staining with triphenyltetrazolium chloride and Even's blue.

Results: There was not a significant difference in QKI5 expression between WT and ob/ob mice. While QKI6 was greatly downregulated in ob/ob mice, and QKI7 was significantly upregulated. Subjecting to ischemia/reperfusion, infarct size in ob/ob mouse heart was greatly larger than that in WT heart. The infarct size was significantly reduced by overexpressing QKI6 either in ob/ob or WT mice. There was no significant difference between these two QKI6-overexpressing groups, which suggested that the increased susceptibility to ischemia/reperfusion injury in ob/ob mice was greatly inhibited by QKI6 over-expression. Similar trends were observed in myocardial enzyme level observations as well.

Conclusion: QKI6 was downregulated in diabetic ob/ob transgenic mouse heart, which contributed to the increased susceptibility of the myocardium to ischemia/reperfusion injury.