**CARDIAC ELECTROPHYSIOLOGY, INJURY BIOMARKERS AND MYOCARDIAL VIABILITY IN VARIOUS TYPES OF ISCHEMIC INSULTS**

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Objective: To compare the effects of various types of ischemic insults caused by increasing volumes of coronary microemboli or occlusion/reperfusion on cardiac electrophysiology, injury biomarkers and viability.

Background: Coronary microembolization occurs spontaneously in acute coronary syndromes and iatrogenically during percutaneous coronary interventions and other systemic diseases, such as valvular, endocarditis, cardiomyopathy, mural thrombus, arrhythmias, hypertension, diabetes, systemic lupus erythematosus and sickle cell, also cause coronary embolization.

Methods: A catheter was placed in LAD artery of 27 pigs. Two microemboli volumes (16 and 32 cubic-mm) were delivered distal to the 2nd diagonal LAD branch. At the same location the LAD was occluded for 90min/reperfused. Electrophysiological changes were continuously monitored after interventions using Holter monitor. Blood samples were obtained before, 1 and 3 days after embolization for measuring creatine-kinase MB and troponin I. Histochemical/histopathological stains and semi-automatic 3SD threshold method were used to characterize and measure damaged myocardium.

Results: The extent of myocardial damage was 6.3±0.7% (16cubic-mm), 8.8±0.5% (32cubic-mm) and 15±2.1% LV mass (coronary occlusion/reperfusion). Creatine-kinase and troponin I were significantly higher after microembolization and LAD occlusion/reperfusion compared with baseline and correlated with extent of myocardial damage. The injury biomarkers were higher in animals subjected to LAD occlusion/reperfusion than microemboli. Holter monitor showed ventricular and supraventricular ectopic activities in all animals up to 3 days after interventions.

Conclusions: This study demonstrates myocardial response to various ischemic insults resulting from vascular and extravascular sources. Electrophysiological abnormalities and release of cardiac injury biomarkers depend on the severity of 1) myocardial damage and 2) inflammation.