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Metabolic syndrome alters inflammatory and membrane markers expression in human atrium cardiomyocytes

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Metabolic syndrome (MetS) is a cluster of various clinical cardiovascular risk factor and causes metabolic and structural cardiomyocytes damage. Our previous study showed that MetS increases cardiomyocytes stress chaperones (1). In this work we aimed to investigate if patients with MetS showed alteration of surface, mitochondria and inflammatory markers in atrium cardiomyocytes.

Atrium samples from MetS patients with stable angina, undergoing coronary artery bypass graft surgery were used. Samples from matched age subjects without MetS and no smokers, undergoing cardiac surgery for other reasons, were used as controls. The samples, obtained before cardioplegia, were fixed and processed for Caveolin 1 (Cav1), MURC, Citrate Synthase (CS), SIRT3, SOD1, IL6-10, iNOS and eNOS by immunohistochemistry. Compared to controls, in cardiomyocytes from MetS patients decreased the expression of Cav1, MURC, SIRT3, IL10 and eNOS, whereas increased the expression of CS, SOD1, IL6, iNOS.

Cardiomyocytes from MetS patients present an evident inflammatory chronic state, that alters the Cav1-MURC expression. These alterations could reduce the exchange functions of sarcolemma so impairing the contractile capacity associated with mitochondrial impairment. We speculate that these damages could induce antioxidant SOD1 over-expression to rescue the cells. All these results, first from human heart, suggest that MetS induces severe enzymatic disregulation in atrium cardiomyocytes that may predispose to cardiac surgery complications.

References

[1] Corsetti et al. (2012) Metabolic syndrome and chronic simvastatin therapy enhanced human cardiomyocytes stress before and after ischemia-reperfusion in cardio-pulmonary bypass patients. Int J Immunopathol Pharmacol 25(4): 1063-1074.

Key words

Metabolic syndrome, atrium, cardiomyocytes, inflammation, caveolin 1, human.