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Possible role of the PI3K/Akt signaling pathway in the dystrophin expression in myocardium from septic mice

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The cell signaling pathways related to PI3K play an important role in the pathogenesis of diseases with inflammatory component, such as cancer, myocardial infarction, atherosclerosis, ischemia-reperfusion injury and sepsis. Recent evidence indicates that the PI3K/Akt signaling pathway may represent a compensatory mechanism in order to limit the pro-apoptotic and pro-inflammatory events in response to sepsis. This pathway has also been reported as a regulator of cardiomyocyte contractility, which could be related to expression of the structural glycoproteins, mainly dystrophin that confers structural stability to the myofiber sarcolemma and transmits force between sarcomeres and cell membrane to the extracellular matrix. Severe sepsis was induced by cecal ligation and puncture (CLP) in mice and the LY294002 (5mg/kg) was used 1 hour before surgery to inhibit the PI3K/Akt signaling. The survival rate was higher in CLP plus LY294002 group as compared to only CLP and controls. Histopathologycal analysis showed foci of disorganized miofibrils and increased membrane permeability, more evident in CLP plus LY294002 group as compared to only CLP and controls. We observed decreased myocardial dystrophin expression in septic mice as compared to controls, but this dysthophin loss was more pronounced in myocardium from septic mice treated with LY294002. The initial evidences suggest that PI3K/AKT signaling may be involved in these myocardial structural changes and could be implicated in sepsis-induced cardiac depression and represents an innovative approach to study the intrinsic mechanism of cardiac abnormalities caused by sepsis, which can provide highly significant contribution in this specific area of knowledge.

Biography

Erica C. Campos graduated in Physiotherapy and since 2004 develops her researches at Laboratory of Cellular and Molecular Cardiology of Department of Pathology at the Ribeirão Preto Medical School, University of São Paulo where she concluded her Master Science and Ph.D. degrees. Her studies emphasize mainly models of cardiac injury induced by isoproterenol and doxorubicin. Currently she is a Postdoctoral fellow in the same University and she is studying the mechanisms of cardiac dysfunction in the septic cardiomyopathy model.

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