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J Nephrol Ther 2016, 6:5(Suppl)
http://dx.doi.org/10.4172/2161-0959.C1.031

Trisulfate disaccharide decreases calcium overload and protects liver injury secondary to liver ischemia/reperfusion

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Background: Ischemia and reperfusion (I/R) causes tissue damage and intracellular calcium levels are a factor of cell death. Sodium calcium exchanger (NCX) regulates calcium extrusion and trisulfate disaccharide (TD) acts on NCX decreasing intracellular calcium through the inhibition of the exchange inhibitory peptide (XIP).

Objectives: The aims of this research are to evaluate the TD effects in liver injury secondary to I/R in animals and ***in vitro*** action on cytosolic calcium of hepatocytes cultures under calcium overload.

Methods: In this study, Wistar rats submitted to partial liver ischemia were divided in to groups: Control: (n=10), surgical manipulation with no liver ischemia; Saline: (n=15), rats receiving IV saline before reperfusion; and TD: (n=15), rats receiving IV TD before reperfusion. Four hours after reperfusion, serum levels of AST, ALT, TNF-α, IL-6, and IL-10 were measured. Liver tissue samples were collected for mitochondrial function and malondialdehyde (MDA) content. Pulmonary vascular permeability and histologic parameters of liver were determined. TD effect on cytosolic calcium was evaluated in BRL3A hepatic rat cell cultures stimulated by thapsigargin pre and post treatment with TD.

Results: AST, ALT, cytokines, liver MDA, mitochondrial dysfunction and hepatic histologic injury scores were less in TD group when compared to Saline group ($p<0.05$) with no differences in pulmonary vascular permeability. In culture cells, TD diminished the intracellular calcium raise and prevented the calcium increase pre and after treatment with thapsigargin, respectively.

Conclusion: TD decreases liver cell damage, preserves mitochondrial function and increases hepatic tolerance to I/R injury by calcium extrusion in $Ca2^{+}$ overload situations.

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Substernal implantation of a subcutaneous implantable cardioverter-defibrillator in a patient with preexisting hemodialysis reliable outflow graft

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Central vein stenosis is a well-documented consequence of cardiovascular implantable electronic devices. In patients with advanced kidney disease, the need to preserve venous real estate is paramount. As such, entirely subcutaneous implantable cardioverter-defibrillators (S-ICDs), which require no venous leads, present an interesting area of development. At this time, the use of S-ICDs in renal failure patients remains controversial. We present a novel approach for implanting an S-ICD in a patient with end-stage renal disease, central venous stenosis and precordial hemodialysis graft obstructing the normal subcutaneous implant site. Implanting an S-ICD in this patient as normally indicated was presumed to carry a high risk of damage to either the lead or the pre-existing graft. The placement resulted in excellent sensing from all three vectors, showing neither T-waves nor artificial potentials. Defibrillation thresholds were tested by inducing ventricular fibrillation, and the first 70 joule shock effectively terminated the arrhythmia two times. The shock impedance was recorded as 68 ohms. There were no complications from the case and the patient was doing well at two-month follow-up.

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