Xiao-Tao Ma, J Nephrol Ther 2017, 7:6 (Suppl)
DOI: 10.4172/2161-0959-C1-048

Et2Cit improved vascular calcification by Wnt/β-catenin pathway

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Vascular calcification is a risk factor for causing cardiovascular events and has a high prevalence among chronic kidney disease (CKD) patients. However, the effective method of treatment is still lack. Et_2Cit is expected to become one of the effective for treatment of vascular calcification. Vascular smooth muscle cells (VSMCs) were induced by a concentration of phosphorus (Pi) of 3.0 mmol/L and different concentrations of Et_2Cit and were subjected to cell calcification analyses, such as Alizarin red staining, Calcium Deposition, Alkaline phosphatase (ALP) Activity. Lithium chloride (LiCl) and dickkop1 (DKK1) were used to activate or inhibit Wnt/β-catenin pathways. The effect of Et2Cit on the Wnt/β-catenin pathway was measured using RT-PCR and Western blotting. Et_2Cit improved VSMC calcification that is induced by high Pi, up-regulated expression levels of VSMC markers and down-regulated levels of osteogenic markers. Et_2Cit decreased the Wnt/β-catenin pathway and β-catenin activity. These results suggest that Et_2Cit improved vascular calcification by the Wnt/β-catenin pathway in high phosphorus level-induced aortic calcification in CKD.

Biography

Xiao-Tao Ma has graduated from Xi 'an Jiaotong University, studying the treatment of chronic kidney diseases, especially in the prevention and therapy of vascular calcification.

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