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P160 -Protective effect of 1,25-dihydroxyvitamin D3 against contrast-induced nephropathy in diabetic rats through modulation of inflammatory responses, oxidative stress.

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αContrast-induced nephropathy (CIN) remains a leading cause of iatrogenic acute kidney injury, and is associated with increased morbidity and mortality, Diabetes is an important predisposing factor for CIN, espicelly in these renal functional impairment patients. The aim of this study was to investigate the effects of 1,25-dihydroxyvitamin D3 against contrast-induced nephropathy in diabetic rats and explore its underlying mechanisms. Diabetes was induced in Male Sprague Dawley rats by a single dose of streptozotocin (60 mg/kg i.v.). Animals were then divided into control group, diabetic group, diabetic CI-AKI group and 1,25-(OH)2D3 group, CI-AKI rats injected diatrizoate (10 ml/kg, 3g I(iodine)/10 ml) via tail vein. And examine the serum creatinine (SCr), blood urea nitrogen (BUN) and blood glucose under anesthetization. Malondialdehyde (MDA), and superoxide dismutase (SOD) were determined to evaluate the oxidative status in the renal tissue. Compared with control group, the level of MDA and the activities of SOD were significantly higher in DM group. by contrast with diabetic CI-AKI group, the activities of SOD decreased and the level of MDA increased.and compared with 1,25-(OH)2D3 group, the activities of SOD decreased and the level of MDA increased much more. Futhermore, Compared with diabetic group, The expression levels of the TNF-α, IL-1β, IL-33,IL-17, IL-23 in diabetic group and 1,25-(OH)2D3 group, were different at each time point ($P<0.05$), Compared with diabetic CI-AKI group, The expression levels of the above inflammatory factors in the 1,25-(OH)2D3 group were different at various time points ($P<0.05$). and NF-κB proteins has the same phenome. Our result suggested that NF-κB Pathway, oxidative stress, inflammatory responses play a partly important role in CI-AKI in the diabetic rats. 1,25-(OH)2D3attenuated both inflammatory processes and inhibited oxidative stress and the NF-κB pathway against CIN in diabetic rats. project supported by Shang Dong Provincial Nature Fund Joint Special Fund Project, No ZR2018LH006：: corresponding author: Xiangling Li E-mail: lixiangling163@163.com