■ ♣Sat-303 ■

Non-alcoholic fatty liver disease may affect coronary artery calcification in patient with CKD

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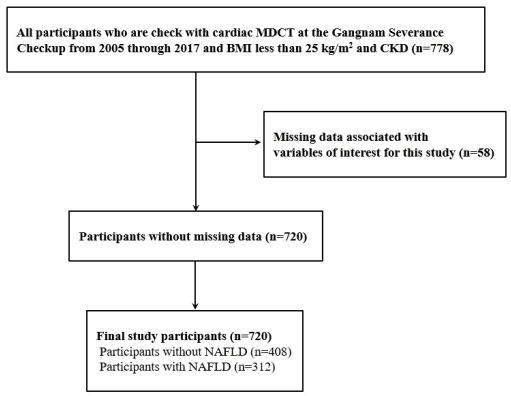
Background/Aims: Accumulating evidence supports an association between non-alcoholic fatty liver disease (NAFLD) and metabolic syndrome. Recently, the possible link between NAFLD and chronic kidney disease (CKD) has been reported. This study aimed to investigate whether the NAFLD is associated with coronary artery calcification (CAC) in the non-obse patients with CKD. **Methods:** Data from a total of 720 participants were provided from 2005 through 2017. CAC score was measured with computed tomography. Estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m2 or dipstick urine protein +1 or more was defined as CKD. To assess the central obesity, waist-to-hip ratio (WHR) was also measured. NAFLD was diagnosed in patients with evidence of liver steatosis on abdominal ultrasonography. **Results:** In univariate logistic regression model, the CAC was significantly higher in high waist hip ratio (W/H) and NAFLD. After adjusting for age, gender, systolic blood pressure, fasting plasma glucose, total cholesterol, WHR, eGFR, NAFLD was significantly associated with increased risk of CAC in non-obese CKD patients (OR=1.419, P=0.049). **Conclusions:** Our study suggested that NAFLD was significantly associated with CAC in the non-obese patients with CKD.

Fig 1. Diagram of participants enrollment



■ ♣Sat-304 ■

EFFECT OF OMEGA-3 FATTY ACID ON SIRT1/3 UP-REGULATION, PGC-1α ACTIVATION AND NRF1 PRODUCTION IN RATS

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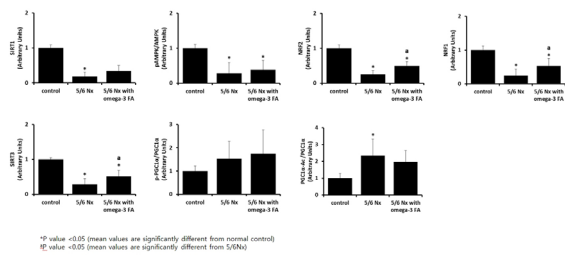
Background/Aims: Mitochondrial dysfunction is an important pathogenesis of renal tubular dysfunction and renal progression in acute kidney injury. Peroxisome proliferator-activated receptor gamma (PPARγ) coactivator-1alpha (PGC-1α) protects renal tubular cell by upregulating nuclear factor erythroid 2-related factor 2 (Nrf-2). Phosphorylation by AMPK and deacetylation by Sirtuin 1/3 (SIRT1 or SIRT3) are required for PGC-1α activation. However, this pathway was not evaluated in chronic kidney disease (CKD). The effect of omega-3 fatty acid (FA) on mediators related with mitochondrial dysfunction is not clear especially in kidney disease. The present study aimed to investigate whether omega-3 FA regulates mediators related with mitochondrial dysfunction in 5/6 nephrectomy rats. **Methods:** Male Sprague Dawley rats were divided into three groups: sham control (0.9% saline), 5/6 subtotal nephrectomy (Nx) (0.9% saline) and 5/6 Nx treated with omega-3 FA (300 mg/kg/day by gastric gavage) group. The expression of PGC-1α, phosphorylated (p) PGC-1α, acetylated PGC-1α, p-AMPK, SIRT1, SIRT3, nuclear respiratory factor (NRF) 1, mammalian target of rapamycin (mTOR), forkhead box class O (FoxO) 1/3, klotho, keap 1and Nrf2 were examined by western blot analysis. **Results:** Significant tubulo-interstitial changes such as tubular atrophy and dilatation were found in 5/6 Nx group compared to sham control. Omega-3 FA group showed attenuated these changes although serum creatinine was not significantly different. Compared with control group, PGC-1α, p-AMPK, SIRT1, SIRT3, NRF 1, mTOR, klotho and Nrf2 were significantly down-regulated in 5/6 Nx group and was recovered by omega-3 FA. The 5/6 Nx group significantly up-regulated keap 1, acetylated PGC-1α, FoxO1 and FoxO3a expression, which were significantly recovered by omega-3 FA. However, the expression of p-PGC-1α was not different among three groups.**Conclusions:** Significant changes of mediators related with mitochondrial dysfunction were found in CKD rat model. Omega-3 FA may mitigate mitochondrial dysfunction by up-regulating NRF 1 and Nrf2. This protective mechanism may be initiated by increased PGC-1α expression and deacetylation of PGC-1α, which was triggered SIRT1/3.



*P value <0.05 (mean values are significantly different from normal control)
†P value <0.05 (mean values are significantly different from 5/6Nx)