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Associations between Markers of Inflammation and Mitochondrial dysfunction with Continuous Glucose Monitoring (CGM) metrics in Diabetes Kidney Disease

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Objectives : Mitochondrial dysfunction and systemic inflammation contribute to progression of diabetic kidney disease (DKD). Recent studies showed that kidney injury molecule 1 (KIM-1) was implicated with in the pathogenesis of inflammation. We hypothesized increased glucose variability (GV), as compared to chronic hyperglycemia, may induce more profound mitochondrial dysfunction and inflammation.
Methods : Blinded continuous glucose monitoring (CGM) recordings, blood and urine samples were collected at baseline and the end of a 16-week randomized controlled trial (NCT0406155), which compared the effect of CGM versus self-monitoring of blood glucose in 65 type 2 diabetes patients with stage 3b to 5 DKD. Plasma tumor necrosis factor receptor family (TNFR1 and TNFR2) and KIM-1 were measured as surrogate of inflammation, while urinary supernatant mitochondrial DNA (mtDNA) level indicated the degree of mitochondrial dysfunction. For CGM metrics, GV was expressed as percentage of coefficient variation (CV); and glucose management indicator (GMI) was a measure of average glucose calculated from senor glucose.
Results : The mean age of the participants was 65.4±9.0 years and the estimated glomerular filtration rate (eGFR) was 26.1±9.6ml/min/1.73m^2. Plasma KIM-1 correlated positively with GMI (r = 0.306, p= 0.035) and negatively with time-in-range (TIR) 3.9–10mmol/L (r = -0.376, p= 0.01), but not with A1c. This remained significant after adjustment of baseline demographics and intervention arm. Urinary mtDNA showed modest but significant association with CV was in patients with eGFR<30 ml/min/1.73m^2 (r =0.373, p<0.05). However, there were no associations between CV with plasma TNFR family and KIM-1.
Conclusions : We found that plasma KIM-1 was independently associated with CGM metrics (GMI and TIR, but not CV). The relationship between GV and urinary mtDNA appeared more significant in patients with advanced DKD. These hypothesis-generating data warrant further studies to investigate the contribution of short-term glucose fluctuations towards systemic inflammation and mitochondrial dysfunction in the pathogenesis of DKD.