Impact of renal function on the underlying pathophysiology of coronary plaque composition in patients with type 2 diabetes mellitus

Both the progression of kidney disease and increased glycemic variability play important roles in the pathogenesis of coronary plaque formation via inflammatory pathways in patients with type 2 diabetes mellitus (T2DM). We prospectively enrolled 71 T2DM patients with 153 coronary artery lesions. Patients were divided into 2 groups according to their estimated glomerular filtration rate (eGFR) level: Group 1 (≥60 mL/min/1.73m², n = 40) and Group 2 (<60 mL/min/1.73m², n = 31). All patients underwent continuous glucose monitoring and blood test including serum tumor necrosis factor (TNF-α). In addition, gray-scale coronary intravascular ultrasound (IVUS) and iMap-IVUS were performed in the coronary lesions with < 50% luminal reduction. In Group 1, only the mean amplitude of glycemic excursions (MAGE) was independently associated with %Lipidic Volume (LV) (β = 0.477, p = 0.002). On the other hand, in Group 2, serum TNF-α was independently associated with %Fibrotic Volume (β = -0.471 and p = 0.007), %LV (β = 0.496 and p = 0.005) and %Necrotic Volume (β = 0.426 and p = 0.017). In T2DM patients, tissue characteristics of coronary plaques were associated with MAGE in patients with eGFR ≥60 mL/min/1.73m² and with serum TNF-α in those with eGFR <60 mL/min/1.73m².