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Role of the Renal Microcirculation injury in Progression of Chronic Kidney Injury in Obesity

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In spite of a progressive fall in the incidence of traditional risk factors of cardiovascular morbidity (cigarette smoking, high blood pressure, and hyperlipidemia), there is an upward trend in the prevalence of obesity and chronic kidney disease (CKD). The close biophysiological interaction between obesity and CKD is evident by a similar occurrence of comorbidities including insulin resistance, hyperlipidermia, endothelial dysfunction, and sleep disorders. In particular, Microvascular (MV) disease plays a pivotal role in progressive kidney injury from different etiologies such as hypertension, diabetes, and atherosclerosis, which are all important consequences of chronic obesity. The MV networks are anatomical units that are closely adapted to specific functions of nutrition and removal of uremic toxins in kidney. Damage of the small vessels in kidney has been reported in obesity and
may increase cardio-renal risk. However, the mechanisms by which obesity and its attendant cardiovascular and metabolic consequences interact to cause renal MV injury and chronic kidney disease are still unclear, although substantial progress has been made in recent years.
Among the individuals who are slightly overweight, or even individuals of normal weight, body fat distribution can be vary and their metabolic profiles and the degree of association of these profiles with cardiometabolic risk factors may differ. Fat distribution might be more of a predictive factor for cardiorenometabolic risk than obesity itself, which has led researchers to investigate whether ectopic fat accumulation may partially account for the development of cardiorenometabolic disorders. In addition to visceral obesity, fat can accumulate in the liver and muscle, and these intrahepatic and intramuscular lipid stores are associated with insulin resistance and adverse metabolic phenotypes. More recently, pericardial fat, perivascular fat, and perirenal fat were found to be associated with coronary atherosclerosis, cardiovascular diseases, and kidney damage, respectively. Particularly, perivascular fat, long assumed to be nothing more than vessel-supporting connective tissue, is now understood to be an important, active component of the vasculature, with integral roles in vascular health and disease. Like other adipose tissue depots, PVAT secretes numerous biologically active substances that can act in both autocrine and paracrine fashion. PVAT has also proven to be involved in vascular inflammation and may accelerate tubulointerstitial fibrosis in chronic kidney disease.