Obesity in CKD

Author:Dr.Khadijeh Makhdoomi
Address: Urmia University of Medical Sciences
makhdoomikhadijeh@yahoo.com

Obesity constitutes a strong risk factor for the development of chronic kidney disease.Obesity-associated nephropathy displays a continuum that evolves from glomerulomegaly to glomerulosclerosis and from mild to severe proteinuria in the absence of significant edema and hypoalbuminemia. The disease may well progress into end-stage renal disease unless weight management strategies are used. CKD and ESRD in obese individuals develop in an incremental fashion directly proportional to body mass index (BMI), independent of hypertension and diabetes comorbidities. Assessment of adiposity should include measurements of both body mass index and waist circumference. The prevalence of obesity, based on a body mass index of 30 kg/m^2 or greater, has increased substantially over the past 2 decades in Western societies. In the 9.4% who developed CKD, an elevated BMI accounted for an odds ratio (OR) of 1.23 for CKD. Corresponding to the degree of obesity in a directly proportional fashion, after accounting for confounders that include age, race, sex, smoking, dyslipidemia, and physical activity, obesity was found to increase CKD, for a period of 5 years, by 34% in obese individuals and 31% in overweight individuals compared with only 28% in non-obese hypertensive adults (OR 1.21 and 1.40, respectively),

Furthermore, the correlation between BMI and risk for ESRD the adjusted relative risks (RR) for ESRD for overweight, class I obesity, class II obesity, and extreme obesity were 1.87, 3.57, 6.12, and 7.07, respectively, after adjustments for baseline blood pressure and diabetes mellitus.

Besides skinfold thickness, waist–hip ratio, and body impedance analysis, the measures of abdominal obesity were shown to be stronger predictors of adverse clinical outcomes than BMI in CKD, although real advantages of these techniques in clinical practice in CKD have not been proven. In patients undergoing hemodialyis, however, body impedance analysis reflecting intracellular body composition but not BMI correlated well with frailty.

Obesity remains the number one preventable risk factor for chronic kidney disease because obesity largely mediates diabetes and hypertension, the 2 most common etiologies for end-stage kidney disease. However, obesity itself likely has independent effects on renal hemodynamics and individuals with a low number of nephrons are likely to be the most susceptible to these changes. Multiple mechanisms have been postulated whereby obesity directly impacts kidney disease including hyperfiltration, increased glomerular capillary wall tension, and podocyte stress. Weight loss reduces glomerular filtration rate and effective renal plasma

flow along with proteinuria, but these changes are most notable after bariatric surgery in adults with morbid obesity. Aside from adiposity itself, the high caloric intake that leads to obesity also may heighten chronic kidney disease risk.

The disease may evolve into impaired clearance, glomerulosclerosis, nephrotic-range proteinuria, and interstitial fibrosis that may develop into renal failure and global sclerosis. As such, depending on the extent of BMI and other comorbid conditions, obese patients may develop different stages of renal involvement on a spectrum anywhere from hyperfiltration to ESRD.

When weight loss reduced glomerular hyperfiltration, it also decreased proteinuria in patients with biopsy-proven ORG by up to 50% as early as 6 months into weight loss. Conversely, patients with increased BMI and no weight loss had an up to 29% increase in urine protein. When patients with diabetic and nondiabetic proteinuric nephropathies were randomized to follow either a low-calorie normoproteinic diet or their usual dietary intake, the mean weight loss in the diet group of approximately 4% was associated with a 31% reduction in proteinuria ($P < 0.005$).More interesting, in obese individuals with advanced diabetic nephropathy, a very-low-calorie ketogenic diet that reduced weight by 12% decreased albuminuria by 36%. Furthermore, these patients experienced statistically significant reductions in serum creatinine, cystatin C, fasting insulin and glucose levels, and insulin resistance.