

Obesity and the Kidney: Heavy Burden of the Epidemic



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Abstract

The 2017 world kidney day (WKD) theme was “Kidney Disease & Obesity” [1]. The choice of this topic underlines the increasing burden of both obesity and chronic kidney diseases (CKD) prevalence worldwide. In nephrology, the growing incidence of obesity, expose patients to special CKD known as obesity related glomerulopathy (ORG), a distinct entity including proteinuria, glomerular hypertrophy, progressive glomerulosclerosis and CKD. In this paper, we discuss the renal pathophysiology effects of obesity, the pathologic finding and the clinical features of ORG with some key points regarding the management of renal effect of obesity.

Introduction

The 2017 world kidney day theme was “Kidney Disease & Obesity” [1]. The choice of this topic underlines the increasing burden of both obesity and chronic kidney diseases (CKD) prevalence worldwide [1,2]. Over the past three decades, the prevalence of obesity (BMI ≥ 30 kg/m²) among adults aged 20-74 years has more than doubled, in the USA [2]. This increase occurred in men and women of all age groups and across diverse ethnicities [2-4]. By 2030, estimates predict that more than 50% of the US population will be obese [2-5]. In nephrology, the growing incidence of obesity, expose patients to special CKD known as obesity related glomerulopathy (ORG), a distinct entity including proteinuria, glomerular hypertrophy, progressive glomerulosclerosis and CKD [1-5].

Mechanisms of renal effects of obesity

The exact effect by which obesity may causes or worsens CKD is unclear. In the setting of obesity, the main kidney physiologic responses are increases in glomerular filtration rate and tubular reabsorption of sodium. Renin-angiotensin-aldosterone system (RAAS) and the renal sympathetic nervous system (RSNS) are also stimulated in this condition. All these physiologic reactions could lead to development of ORG. In addition, inflammatory adipokines produced by adipocytes might promote maladaptive cellular responses to glomerular hyperfiltration and albuminuria.

The pathologic concept of obesity-related glomerulopathy (ORG)

The first case reports of ORG were described in autopsy studies as a strange association between extreme obesity and the development of glomerular hypertrophy [1,2]. Renal biopsies are currently performed in obese patients. ORG is a distinct and authentic renal involvement of obesity. Based on detailed clinical-pathologic studies, the pathologic features of ORG include glomerulomegaly and a particular focal segmental glomerulosclerosis (FSGS) well known as perihilar variant of FSGS [2-6]. Several reports showed an important increase the ORG incidence from 0.2% to 2.0% in ten years (1990-2000). It is well admitted that ORG could be seen in all obesity grades [4-6].

Clinical features of ORG

The main clinical presentation of ORG includes isolated proteinuria with or without renal insufficiency. These renal manifestations are usually associated with hypertension (50-75%) and dyslipidaemia (70-80%) [1,2]. Proteinuria, assessed by semiquantitative dipstick (grade 1+ or more) or macroalbuminuria measurement (albumin: creatinine ratio >300 mg/g) is present in 4-10% of obese patients, especially in some Asian countries [1-7]. Nephrotic-range proteinuria (3 grams or more per day) is unusual. Full nephrotic syndrome is very rare in obese patients.

The clinical ORG evolution is characterized by slowly progressive proteinuria [2-8]. However, 10 to 33% of patients may develop progressive renal failure and end stage renal disease (ESRD) [6-10].

Management of renal effect of obesity

The treatment target in ORG is reduction of proteinuria and blood pressure [2]. RAAS blockade including angiotensin conversion enzyme (ACE) inhibitors or angiotensin-receptor blockers (ARBs) are well indicated. Weight reduction could reduce proteinuria as well [1,2].

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