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NEPHROLOGY - REVIEW

Obesity and glomerular filtration rate

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Abstract
Obesity has received considerable attention in general medicine and nephrology over the last few years. This condition increases the risk of metabolic syndrome, diabetes mellitus, hypertension, and dyslipidemia, which are the main risk factors for developing chronic kidney disease (CKD). Kidney damage caused by obesity can be explained by many mechanisms, such as sympathetic nervous and renin-angiotensin-aldosterone systems activation, mechanical stress, hormonal unbalance, as well as inflammatory cytokines production. Even though creatinine-based glomerular filtration rate (GFR) equations in obese individuals have been validated (Salazar-Corcoran and CKD-MCQ), changes in body weight after bariatric surgery (BS) leads to changes in creatininemia, affecting its reliability. Thus, an average between creatine and cystatin-based GFR equations would be more appropriate in this setting. Bariatric surgery can reverse diabetes mellitus and improve hypertension, which are the main causes of CKD. Conclusion: GFR can be affected by obesity and BS, and its value should be cautiously evaluated in this setting.

Keywords Obesity · Glomerular filtration rate · Bariatric surgery

Introduction

For the past several years, general medicine and nephrology have paid a great deal of attention to obesity, which is defined as a body mass index (BMI) ≥ 30 kg/m^2. Since 1980, obesity incidence has doubled in children and adults, which is now a pandemic that affects 40% of adult Americans. Moreover, the percentage of American individuals with extreme obesity (BMI 40 kg/m2) rose from 5.7% (2007–2008) to 7.7% (2015–2016) [1]

Chronic kidney disease (CKD), which consists of renal structure or function abnormalities present for > 3 months, affects more than 10% of the population worldwide and is strongly associated with accelerated cardiovascular morbimortality [2]. Obesity can aggravate the course of CKD, as it increases the risk of metabolic syndrome, diabetes mellitus (DM), hypertension, and dyslipidemia, which are the main risk factors for CKD development and progression. For this reason, the treatment of obesity is as important as the pharmacological treatment of the aforementioned comorbidities (DM, hypertension, etc.), and that of glomerular hyperfiltration per se (enalapril, dapagliflozin, etc.) in CKD patients [3–7].

Obesity-induced kidney damage can be explained by many mechanisms, as follows (Fig. 1): First, an excess of adipose tissue can activate both the sympathetic nervous and renin-angiotensin-aldosterone systems (RAAS), which promote glomerular hyperfiltration, as well as increased tubular sodium reabsorption in the proximal tubule and the thick ascending limb of the loop of Henle (TALH). Moreover, this phenomenon reduces sodium delivery to macula densa activating tubuloglomerular feedback, and increasing hyperfiltration. Aldosterone and angiotensin secreted by fat tissue are also implicated in the increased sodium reabsorption in obesity [8]. The above-mentioned mechanisms lead to an obesity-induced renal function reduction, and the appearance or worsening of hypertension, initiating

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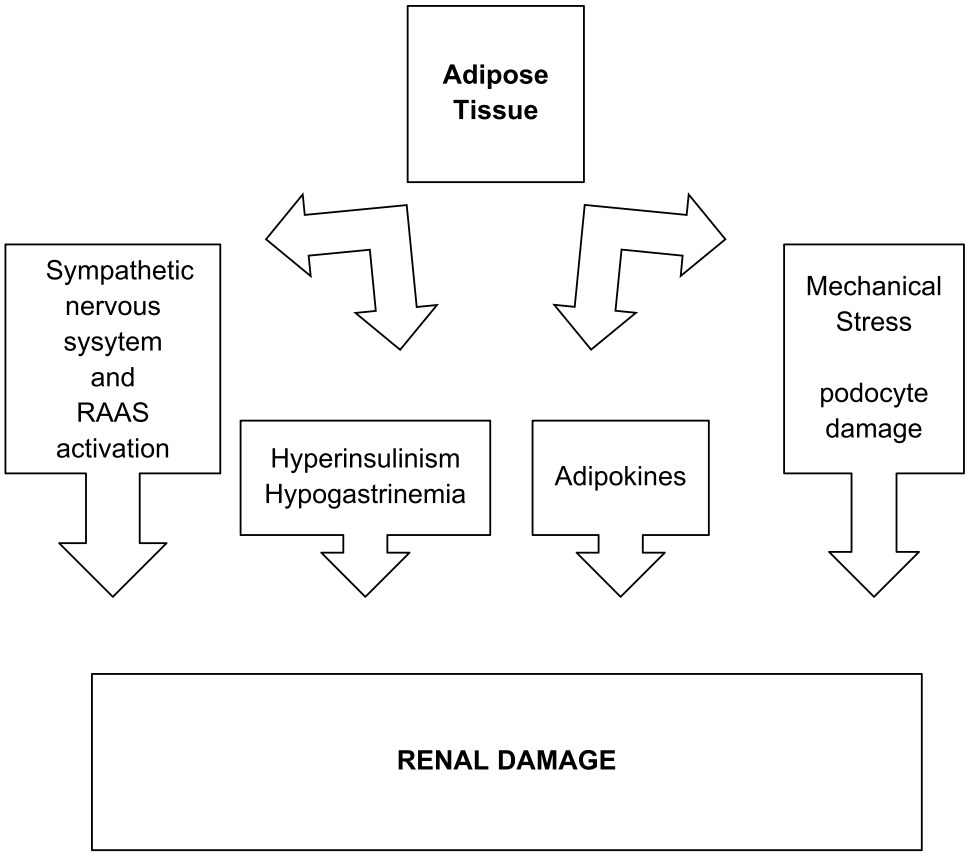

Fig. 1 Pathophysiologic mechanisms of obesity-induced renal damage

a cycle of kidney damage. The mechanisms that lead to sympathetic nervous activation are not completely understood, but several factors have been proposed as triggers, including hyperinsulinemia, hyperleptinemia, increased levels of fatty acids and angiotensin II, as well as baroreceptor reflex alterations [8, 9].

Second, sodium reabsorption is also stimulated in the proximal tubule and TALH by other unbalanced hormones such as hyperinsulinemia secondary to insulin resistance, and hypogastrinemia secondary to reduced soduretic gastrorenal axis activity in obese individuals [8].

Third, fat tissue itself is a site of production of several cytokines (adipokines), such as leptin, tumor necrosis factor α, and interleukin, which are responsible for an increase in renal inflammation, and oxidative stress [8]. Moreover, fat accumulation in podocytes damages them, leading to progressive albuminuria, oxidative stress, insulin resistance, and interstitial fibrosis [10]. Leptin increases the activity of the sympathetic nervous system and promotes fatty acid oxidation, increasing oxidative stress and proinflammatory cytokines secretion. Conversely, adiponectin is decreased in obese patients, and its low levels are associated with insulin resistance and impaired glucose and lipid metabolism, as well as podocyte damage [8]

Fourth, obesity induces glomerular hyperfiltration which increases capillarity tensile stress, and ultrafiltrate flow into Bowman's space that increases podocyte shear stress. These mechanical stresses lead to podocyte hypertrophy and glomerular basement membrane (GBM) lengthening. Since podocyte growth is limited, a mismatch develops between the GBM area and its area covered by podocyte feet, leading to podocyte damage, synechia formation, and segmental sclerosis. In addition, mechanical stress altered post-glomerular structures, resulting in tubular urinary space dilation, increased proximal tubular sodium reabsorption by hypertrophied cells and cytokines secretion which lead to tubulointerstitial inflammation, and fibrosis [11].

Finally, genetic factors, gestational diabetes, reduced renal mass (low birth weight, nephrectomy, chronic kidney disease), senescence, sleep apnea síndrome, pulmonary hypertension, non-alcoholic fatty liver disease, congenital anomalies of the kidney and urinary tract, as well as socioeconomic issues have been described as predisposing factors for obesity renal damage [11–13]. In the present article, the impact that obesity and its surgical treatment have on renal function is analyzed in detail.

Glomerular filtration rate markers in obese patients

Precise glomerular filtration rate (GFR) determination is crucial to evaluate disease progression, to monitor the renal

effect of an intervention, and to select the adequate drug dose in CKD patients. [14–16].

Gold standard methods for obtaining measured glomrular filtration rate (mGFR) are expensive, difficult to perform, and time consuming. These methods, which are based on the elimination of inulin, chromium-EDTA, or iohexol, are not usually used in clinical practice or in studies that include a large number of patients [10, 12]. Besides, Kittiskulnam et al. have documented that estimating GFR equations generally underestimated mGFR in obese subjects [17].

Serum creatinine is often used to assess GFR since it is an inexpensive and reliable renal function marker. The main determinant of creatinine production is skeletal muscle mass where creatinine is the final catabolite of its metabolism. Therefore, changes in body weight, particularly secondary to muscle mass reduction (sarcopenia), lead to changes in serum creatinine levels. This phenomenon could affect GFR estimation by equations based on serum creatinine, such as MDRD and CKD-EPI, since they could be biased. Regarding cystatin C as a glomerular filtration marker, even though it is less altered by muscle mass variation, it may be biased in people suffering from obesity or in those who have large variations in fat mass levels over time [16, 18].

It is worth mentioning that there are two creatinine-based equations that have been validated in an obese population: the Salazar–Corcoran equation (validated in an animal model and in humans) and the Quadratic Equation (MCQ) combined with CKD-EPI (CKD-MCQ) (validated in humans) (Table 1) [18].

Finally, even though there is no agreement on which is the best GFR equation to be used across the life span in obese individuals, and cystatin C-based GFR equations showed to be less biased than creatinine-based ones, it seems that the accurate way to estimate GFR was to obtain estimated glomerular filtration rate (eGFR) by calculating an average between creatinine and cystatin C-based GFR equations in this population [19–23].

Indexing vs not indexing GFR in obese patients

The concept of indexing GFR to some body parameters, such as body surface área (BSA) or extracellular fluid volume (ECV), in severe obese individuals is currently controversial, since it was documented that an absolute mGFR value was more reliable than an indexed one (by BSA or ECV), as such adjustment leads to a significant GFR underestimation in severe obese population [1, 24–26].

In this sense, it has been reported that comparison of indexed and non-indexed eGFR (applying equations based on creatinine, cystatin, or both) with indexed and non-indexed mGFR (obtained using iohexol clearance), before and after BS, found that the most accurate way to estimate GFR was to obtain non-indexed eGFR by calculating an average between creatinine and cystatin C based GFR equations ([1, 27].

Since Cockcroft–Gault equation is still used in some clinical settings, such as oncologic drug dosification, to calculate renal function applying this equation in severe obese patients, it should use the patient's lean weight instead of their theoretical or actual weight for calculation [24, 28].

Therefore, the most reliable way to evaluate GFR in severely obese individuals is to measure it using a non-creatinine-based method (iohexol, etc.), and expressing it without indexing it. However, if eGFR should be obtained using a creatinine-based equation, such as MDRD or CKD-EPI, this should be performed by de-indexing it as follows [18]:

MDRDd (deindexed) = MDRD × patient body surface/1.73.

CKD-EPId (deindexed) = CKD-EPI × patient body surface/1.73.

Glomerular filtration rate markers after weight reduction in obese patients

Glomerular hypertrophy, and adaptive focal segmental glomerulosclerosis are the most relevant histopathological manifestations of obese-related renal glomerulopathy. Although renin-angiotensin-aldosterone blockade is effective in controlling proteinuria and hyperfiltration in the short term, the effect of diet-mediated weight loss or bariatric surgery (BS) has more consistent antiproteinuric effects and is much more effective in reversing hyperfiltration [15]. Many studies and meta-analyses have shown that obesity surgery reduces albuminuria and proteinuria, as well as has a positive effect on renal damage. However, studies on obesity have often measured renal function using glomerular

Table 1 creatinine-based equations validated in obese individuals

Equation name	Equation formula
Mayo clinic quadratic (MCQ)	MCQ = exp (1.911 + 5.249/CrS [mg/dl] – 2.114/CrS [mg/dl] – 0.00686 × age (years) – 0.205 if a woman)
Salazar-Corcoran	Creatinine Clearance (man) = [137 − age] × [0.285 × weight] + (12.1 × height) (51 × SCr)
	Creatinine clearance (woman) = [146 − age] × [0.287 × weight] + (9.74 × height) (60 × SCr)

CrS serum creatinine

Table 2 Renal function evaluation pre- and post-bariatric surgery (BS)

Studies	Main finding
Chang et al. (2020)	Indexed and non-indexed eGFR scr-scys can be less biased than indexed or non-indexed eGFRscr or eGFRscys
Chuah et al. (2018)	mGFR (51Cr-EDTA) and eGFR (CKD-EPI scys, scys-scr, and CC equations) did not change after BS. Conversely, eGFR (MDRD and CKD-EPI screat equations) improved after BS
von Scholten (2018)	After large weight loss, absolute mGFR was reduced while mGFR adjusted for BSA was unchanged; and eGFR scr overestimated renal function while eGFR scyst was unaffected
Rothberg et al. (2020)	eGFR indexed to actual BSA showed hyperfiltration which improved after weight loss eGFR scyst indexed to actual BSA provided a more accurate renal function evaluation
Grangeon-Chapon et al. (2021)	In CKD-obese patients treated with BS, mGFR scaled to EFV could be preferred to evaluate the effect of BS on renal function
Friedman et al. (2014)	In severe obese patients with normal renal function CKD-EPI scr-cyst equation best predicted mGFR in both pre- and post-BS

eGFR estimated glomerular filtration rate, *scr* serum creatinine, *scys* serum cystatin, *mGFR* measured glomerular filtration rate, *CC* Cockcroft Gault, *BSA* body surface área, *EFV* extracellular fluid volume, *CKD* chronic kidney disease

filtration rate (GFR) equations based on creatinine (MDRD or CKD-EPI). Since these patients experience a reduction in muscle mass associated with weight loss after the procedure, these equations could overestimate the patient's GFR value. Therefore the reduction in creatinine observed in these patients could falsely suggest an improvement in renal function [14]. Chuah et al. evaluated renal function in CKD patients using 51Cr-EDTA, and observing that GFR remained stable 12 months after BS [7]. This result contrasts with the significant renal function improvement observed when GFR was assessed by applying creatinine-based GFR equations, such as MDRD or CKD-EPI [12]. Then, GFR was evaluated in a prospective observational study that included 27 adults with BMI $\geq$ 35 kg/m^2, before and after undergoing BS. GFR was measured (mGFR) using plasma iohexol clearance and compared to eGFR applying CKD-EPI equation based on creatinine, cystatin C, or both. The result was that an average of the equations based on creatinine and cystatin C were less biased than those based on creatinine or cystatin alone, and this might be due to opposite biases in GFR estimation that creatinine and cystatin have. Serum creatinine values are directly related to muscle mass, which decreases between 20 and 25% after obesity surgery. On the other hand, cystatin C has been associated with inflammation, which is common in severe obesity [1]. In addition, it was also observed that GFR equations based on creatinine overestimated post-BS renal function, while those based on creatinine and cystatin C levels at the same time were more accurate [16].

Since it was relevant to determine the impact that BS had on renal health, and for this purpose was essential to identify the best method to estimate GFR, it was decided to perform an original study where GFR was measured by iohexol clearance before and after BS, and this mGFR was compared to eGFR by applying MDRD and CPD-EPI equations base on creatinine, cystatin C or both. Observing that, in obese subjects with normal renal function undergoing obesity surgery, serum cystatin C was more strongly correlated with mGFR than serum creatinine, and the CKD-EPI creatinine-cystatin C equation better predicted mGFR before and after surgery [29]. In a prospective study, Rothberg et al. reported that when eGFR was indexed to actual BSA, many individuals evidenced renal hyperfiltration, which improved after weight loss. They also found that estimating equations based on cystatin C and indexed to actual BSA can provide a more accurate renal function assessment [20].

In a prospective intervention study, investigating the effect of large weight loss after gastric bypass surgery on mGFR (51Cr-EDTA plasma clearance), and on eGFR (using both creatinine and cystatin C), they found an absolute reduction in mGFR which may reflect the resolution of glomerular hyperfiltration, whereas body surface área (BSA) corrected mGFR was unaltered [16]. Billeter et al. performed a prospective cohort study in mild obese insulin-dependent patients, which showed that their creatinine and cystatin C clearances notably improved, and albuminuria decreased after BS. The fact that both creatinine and cystatin C clearances improved confirms that renal function at least stabilized postoperatively and in some cases even improved [4].

It is worth mentioning that minor pharmaceutically induced weight reduction did not induce any change in serum creatinine levels and, consequently, creatinine-based equations (MDRD and CKD-EPI) can be applied in this population [16] (Table 2).

Renal and cardiovascular benefits of bariatric surgery (BS)

Significant obesity increases the risk of end-stage renal disease (ESRD), and more than one-fifth of CKD patients in the United States have a body mass index of 35 kg/m^2.

Therefore, BS can prevent ESRD progression or allow some ESRD patients who suffer from severe obesity to become candidates for kidney transplantation. Kun Li et al. revealed that BS could remarkably reduce urinary albumin and protein excretion in obese patients [3, 30, 31]. In addition, BS can also achieve sustained weight loss, improve blood pressure, reduce dyslipidemia, hyperglycemia, and even result in DM remission. As obesity significantly raises the risk of DM and hypertension, the two most prominent causes of ESRD, it is crucial to consider how BS affects CKD risk factors beyond the post-BS GFR improvement. After Roux en Y gastric bypass and laparoscopic adjustable gastric banding, DM type II was shown to be in remission in 66.7 and 28.6% of patients, respectively. Besides, BS produced better glycemic control than medical treatment alone for weight reduction. Finally, post-BS albuminuria, proteinuria, and cardiovascular events are reduced [32–35]. The studies that evaluated the impact of obesity surgery on renal health have mostly measured renal function using eGFR. It deserves mentioning that muscle mass and serum creatinine reduction that accompanies post-BS weight loss, may falsely show an improvement in kidney function [7], although post-BS there was a statistically significant reduction in hyperfiltration, albuminuria, and proteinuria [14]. Finally, a renal health threat post-BS is hyperoxaluria, and the increased incidence of oxalate stones, induced by post-BS fat malabsorption [33].

Conclusion

Measured and estimated glomerular filtration rate (GFR) should not be indexed to body surface área in obese individuals, since this may underestimate their GFR. Even though there are creatinine-based (GFR) equations which were validated in obesity, changes in body weight after bariatric surgery lead to changes in creatininemia, affecting its reliability. Thus, an average between creatinine and cystatin-based GFR equations would be more appropriate in these settings.

Declarations

Conflict of interest All the authors declare that they have no conflict of interest.

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