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# Evaluation of glomerular hyper filtration in obesity: Which formula to use?

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## Abstract

**Introduction**: Obesity is a progressive chronic disease that is a renal risk factor. Renal hyperfiltration is an early stage in the development of chronic kidney disease (CKD). The objective of our study is to determine the prevalence of glomerular hyperfiltration in obese patients.

**Materials and Methods**: This is a prospective and descriptive study conducted over a period of 4 months. All patients with BMI over 30 kg/m2 who do not have diabetes, hypertension or another apparent cause of CKD.

**Results**: A total of 85 patients were included, the mean age was 41.96 years, with a female majority. The mean BMI was 38.24kg/m2 and the mean abdominal waist circumference was 114.57cm. Obesity was common in 55.88% and secondary in 44.11%. The prevalence of glomerular hyperfiltration was 85.18% by Cockcroft formula and 48.33% by MDRD formula.

**Discussion**: Obesity is a risk factor for CKD, which promotes increased renal blood flow by vasodilation of the afferent glomerular arteriole, resulting in glomerular hyperfiltration that leads to a change in the glomerular barrier, increasing the risk of developing CKD.

According to several studies, the Cockcroft formula is a better predictor of renal function in obese patients. A 51% increase in GFR in obese patients has been observed. In our study, the prevalence of glomerular hyperfiltration is 85.18% according to the Cockcroft formula.

**Conclusion**: Obesity is a risk factor for CKD that needs to be carefully considered.

**Keywords:** Obesity; Renal hyperfiltration; Cockcroft formula; CKD; Adipokines; Glomerular hypertrophy

## 1. Introduction

Obesity is a chronic progressive disease that can have adverse health consequences. The continuous increase in its prevalence is accompanied by an increase in the frequency of chronic diseases such as type 2 diabetes and cardiovascular diseases. Obesity is also an independent factor of renal risk that deserves to be taken into account. Renal hyper-filtration, defined by an abnormally high glomerular filtration rate (GFR), is an early stage in the development of chronic kidney disease (CKD).

The objective of our study is to determine the prevalence of glomerular hyperfiltration in obese patients

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## 2. Material and methods

This is a prospective and descriptive study conducted over a period of 4 months between March 2022 and July 2022.

All patients over 18 years of age, with BMI greater than 30kg/m2, with no diabetes, HTA or other apparent cause of chronic kidney disease were included. Socio-demographic data were collected. Anthropometric parameters were measured. The measurement of creatinine clearance was done by the formula MDRD and Cockcroft. Statistical analysis was done by software SPSS 26.

## 3. Results

A total of 85 patients were included. The average age was 41.96 14.58 years. The majority were women (89.4%). The average BMI was 38.24 6.71kg/m2. The average abdominal waist circumference was 114.57 12.44cm. Obesity was common in 55.88% (48 patients) and secondary in 44.11% (37 patients). Obesity was morbid in 34.11% of patients.

Vitamin D deficiency was present in 96.6%. Dyslipidemia was present in 25% of patients. Pre-diabetes was present in 47.8% of patients.

The prevalence of glomerular hyperfiltration is 48.33% according to the MDRD formula, and 85.18% according to the Cockcroft formula. Prevalence of kidney disease is 5.81% (Stage 2)

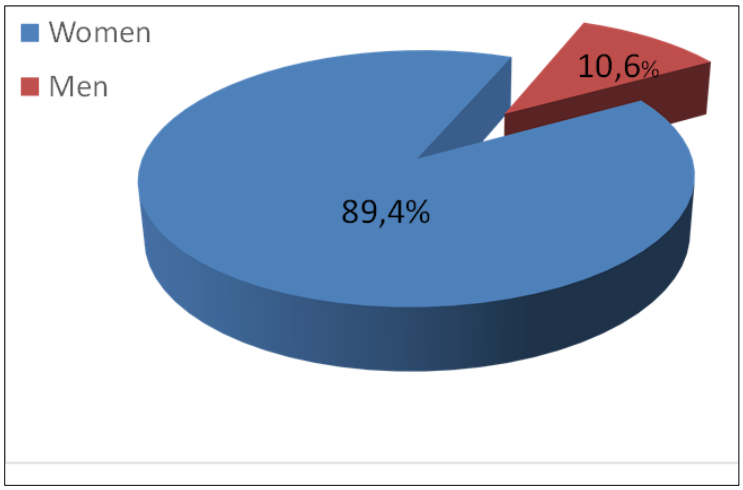


**Figure 1** Distribution of patients by sex

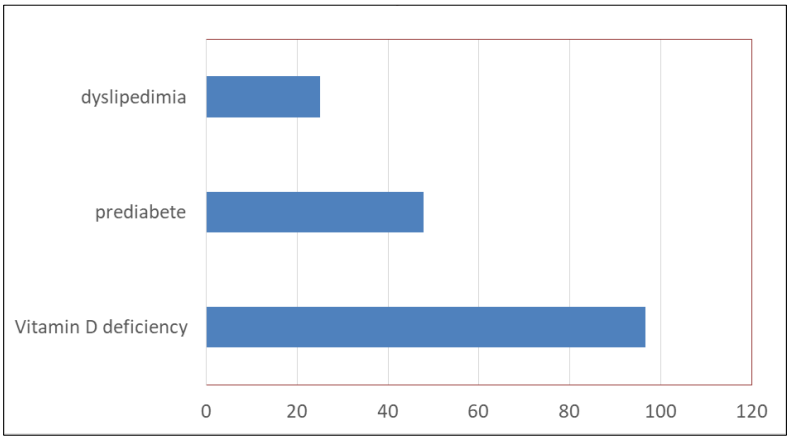


**Figure 2** The various biological abnormalities found in our patients

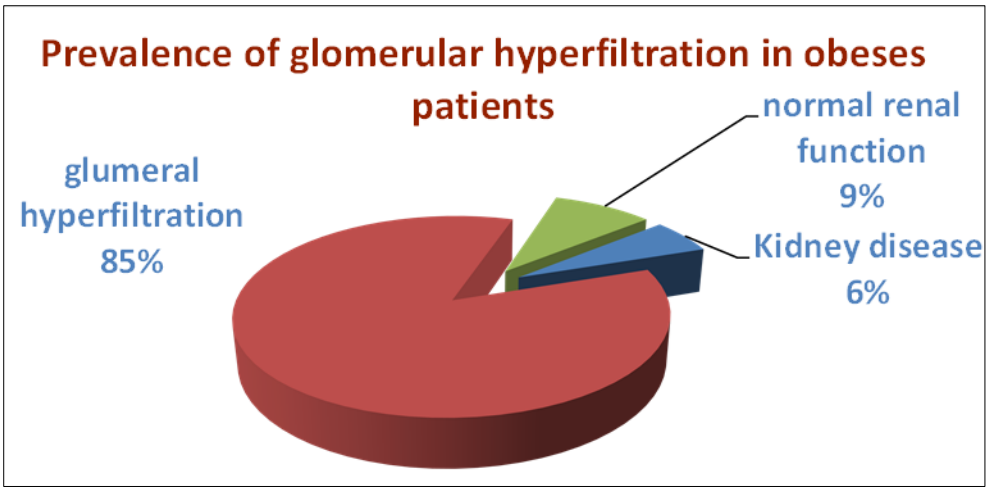


**Figure 3** Prevalence of glomerular hyper filtration in obeses patients

## 4. Discussion

Obesity has become a global epidemic, several studies show that it is a risk factor for the occurrence and progression of CKD, regardless of all other usual risk factors (type 2 diabetes, hypertension), and that increased body mass index is mainly associated with the onset of glomerulopathy. In the long term, the risk of terminal CKD is correlated with BMI, with a relative risk of 7 compared to normal weight subjects (1, 5, 7).

It was reported in a cohort of middle-aged 50-year-old men that the prevalence of microalbuminuria increased from 9.5% in the normal build group to (BMI < 25) 18.3% in the overweight group and 29.3% in obesity. Indeed, subjects with abdominal obesity are more likely to develop microalbuminuria. And this risk was significantly higher in subjects with glomerular hyperfiltration (2,5).

According to several studies, the Cockcroft formula is a better predictor of kidney function in obese patients (2, 12). A 51% increase in GFR in obese patients was objectified. In our study, the prevalence of glomerular hyperfiltration is 85.18% according to the Cockcroft formula (figure 3).

The physiopathological mechanisms of CKD linked to obesity involve glomerular hemodynamic changes as well as activation of many cytokines and growth factors through hyperlipidemia, hyperinsulinemia and increased leptin (5,9).

With regard to hemodynamic changes, obesity promotes an increase in renal blood flow by predominant vasodilation of the related glomerular arteriole, which causes glomerular hyperfiltration. This directly changes the phenotype of glomerular cells, promotes an increase in intraglomerular pressure, and causes a change in the glomerular barrier, which increases the risk of developing CKD (3,4,10).

In individuals affected by obesity, hyperfiltration and compensatory glomerular hypertrophy occur to meet the increased metabolic demand caused by weight gain, which is predicted by the appearance of less certainly reversible lesions, which may involve mesangial expansion, paramesareal deposition, thickening of the glomerular basal membrane, fusion of pedicels as in nephrotic syndrome and finally focal glomerulosclerosis. These lesions are also called obesity-related glomerulopathy (ORG) (5,6).

Adipokines, particularly leptin and adiponectin, have also been implicated in the genesis of obesity-related kidney damage. Leptin has been shown to increase proteinuria and synthesis of extracellular matrix proteins and, therefore, the development of focal and segmental glomerulosis. It causes the proliferation of glomerular and mesangial endothelial cells, increases the expression and secretion of TGFb and its receptor (subtype II) and induces the production of type I and IV collagen. As the kidney is the main site of leptin degradation, the development of nephropathy changes this function and increases leptinemia. The establishment of this vicious circle leads to worsening nephropathy (6,11).

Obesity is associated with a low-grade inflammatory state, oxidative stress, abnormal lipid metabolism, activation of the renin-angiotensin-aldosterone system and increased insulin production, including accumulation of ectopic lipids

and increased deposition of renal sinus fat. These different effects lead to specific pathological changes in the kidney that may be responsible for the higher risk of CKD seen in observational studies (7,9).

Obesity is associated with a number of risk factors that contribute to the increased incidence and prevalence of nephrolithiasis. Higher body weight is associated with lower urinary pH and increased urinary excretion of oxalate, uric acid, sodium and phosphate. Diets rich in protein and sodium can lead to more acidic urine and a decrease in urinary citrate, which also contributes to the risk of kidney stones. Insulin resistance characteristic of obesity can also predispose to nephrolithiasis by its impact on the tubular Na-H exchanger and the formation of ammonia and the promotion of an acidic medium (1,5,8).

The mechanisms behind the increased risk of kidney cancer observed in obese individuals are less well characterized. Insulin resistance, and the resulting chronic hyperinsulinemia, and increased production of insulin-like growth factor-1 and many complex secondary humoral effects may stimulate the growth of various tumor cell types. More recently, the endocrine functions of adipose tissue, its effects on immunity, and the generation of an inflammatory milieu with complex effects on cancers have emerged as additional explications (8,10).

## 5. Conclusion

Obesity is a risk factor for the CKD and deserves careful consideration in light of its increasing prevalence in the general population.

Reducing body weight is a therapeutic measure to prevent the onset of end-stage chronic kidney disease.

## Compliance with ethical standards

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*Disclosure of conflict of interest*

The authors declare no conflict of interests.

*Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

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