A RELATIONSHIP BETWEEN BODY MASS INDEX AND CHRONIC KIDNEY DISEASE - A SURVEY

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ABSTRACT

Aim: To find the relationship body mass index (BMI) to Chronic Kidney Disease
Objective: To explore the relationship of Body mass index and the prevalence of Chronic Kidney Disease. And also to improve the early evaluation and management of risk factors associated with it.
Background: Overweight and obesity have become global epidemics and it has been suggested that they can lead to impaired kidney function. An increase in body fat is often associated with an increased risk of the metabolic diseases such as chronic kidney disease, diabetes mellitus, hypertension and dyslipidemia. Body mass index is the primary focus of any obesity treatment. The relationship between the body weight and metabolic diseases helps in a better understanding of the pathophysiological process.
Reason: To help improved evaluation and management of risk factors associated with Chronic Kidney Disease.

INTRODUCTION

Obesity defined by elevated body mass index (BMI) has been regarded as a cardiovascular risk factor in the general population. Obesity is also associated with increased risk of incident Chronic kidney disease. Negative effects of obesity include those effects mediated by conditions caused or worsened by it, such as diabetes mellitus (DM) or hypertension, and direct adverse metabolic effects, such as inflammation, increased synthesis of apolipoprotein B and very LDLs, increased production of insulin, and insulin resistance.

The deleterious effects of obesity on human health are systemic in nature, and kidney dysfunction is now recognized as a relevant health risk posed by obesity. Diabetes and hypertension are established pathways whereby fat excess may induce renal damage but the risk for chronic kidney disease (CKD) in obesity is largely independent of these comorbidities. Several population-based, observational studies showed obesity, defined by BMI, as an independent risk factor in the development of CKD and end stage kidney disease (ESKD). In other studies, overweight, obesity and increased central fat distribution have been associated with reduced estimated glomerular filtration rate (eGFR) and microalbuminuria. The most widely recognized strategy for characterizing obesity depends on BMI (WHO) considers a BMI in the vicinity of 20 and 25 kg/m² as ordinary weight, a BMI in the vicinity of 25 and 30 kg/m² as overweight, and a BMI of >30 kg/m² as fat. It should be emphasized that population norms of BMI could be different based on ethnic and racial background. Although BMI is easy to calculate and used in many nutritional guidelines, this metric is a poor estimate of fat mass distribution, especially in CKD.

MATERIALS AND METHODS

The study included a sample of 50 patients who were admitted in the inpatient ward of general medicine department in Saveetha medical college & Hospitals. The height and weight of 50 patients who were diagnosed as chronic kidney disease were recorded and their respective BMI were calculated. The calculated BMI was then analyzed statistically.
RESULTS
The recorded data were statistically analyzed and the results were formulated. According to the survey among the chronic kidney disease patients, 52% of the patients were present in the category of 25-30 BMI whereas 36% of the patients were present in the category of above 30 BMI. 12% of the patients were present in the category of 19-25 BMI. (Fig 1) (Fig 2)

DISCUSSION
Few studies have evaluated BMI as a potential risk factor in the development of kidney disease, and available results are in favor for it. Similarly, studies of BMI and CKD showed variable results, depending on study design and method of measuring. Also, the exact role of increased body size in the development of CKD independent of confounding factors is unclear. In a cross-sectional analysis, morbid obesity was related to CKD, but these associations were mediated by the effects of diabetes and hypertension. The studies of Beddu et al. [14] demonstrated that the survival advantage of high BMI among CKD patients on long-term dialysis was limited to those with normal or increased muscle mass. Patients with high BMI and high body fat had increased all-cause and cardiovascular mortality.

Pathophysiological studies have revealed that, among patients with CKD, obesity is not an innocuous bystander and may directly or indirectly damage the kidney. Evidence for the direct damaging effect of obesity is the following. Because of heightened sympathetic activity, high levels of angiotensin II, and hyperinsulinemia, obesity is often accompanied by glomerular hyperfiltration and increased proximal tubular sodium resorption. Enhanced proximal salt reabsorption determines a reduced delivery of sodium to the macula densa. This then causes afferent vasoconstriction and enhanced renin activation. As a result of high local angiotensin II levels, the efferent arteriole is constricted in the obese, and glomerulomegaly and focal glomerulosclerosis ensue. Evidence is emerging that fat cells may trigger inflammation in the kidney indirectly by producing inflammatory cytokines, which may further aggravate renal dysfunction. [15]

CONCLUSION
In conclusion, our study has apparently shown a significant association between BMI and CKD, independent of other potential mediators such as hypertension, diabetes, high cholesterol level and insulin resistance. Further analyses are necessary to confirm the pathogenesis of obesity-related CKD.

References