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Association of Obesity with Outcomes in Patients with Chronic Kidney Disease

Rameen Aijaz, Bushra Jamil

ABSTRACT

Objective: The present study was designed to clarify the impact of obesity with outcomes among hospitalized patients with chronic kidney disease (CKD).

Study design and setting: A 6 months prospective cohort study from From April to September 2024, which took place in the Department of Medicine, Aga Khan University Hospital, Karachi Pakistan.

Methodology: The patients were enrolled from inpatient and outpatient medical wards of AKUH and 200 hospitalized CKD patients were recruited and divided equally into obese (BMI >30 kg/m²) and non-obese (BMI <30 kg/m²) groups. Baseline characteristics were contrasted, and 30-day in-hospital mortality was assessed. We conducted subgroup analyses for age, sex, diabetes and hypertension. p-values were computed by SPSS version 25 and the results of = 0.05 determined significance.

Results: Compared with non-obese patients, obese patients were younger, more likely to be male, and had a higher mean estimated glomerular filtration rate (eGFR). Diabetes occurred less frequently in the obese group (21% vs 47%) and hypertension was present in both groups. Obese patients had a 7% 30-day mortality compared to 3% for non-obese patients [RR = 2.33 (p =.20)]. The risk of mortality was higher in women, younger individuals and those with diabetes but the associations did not reach statistical significance.

Conclusions: Obesity was significantly associated with increased short-term mortality in hospitalized CKD patients, especially for women and diabetics.

Keywords: Body Mass Index, Diabetes Mellitus, Hypertension, Kidney Disease, Mortality, Obesity

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INTRODUCTION

Chronic kidney disease (CKD) is characterised by change in renal parenchyma, functional or structural that lasts for more than three months and whose estimated glomerular filtration rate (eGFR) is less than 60 ml/min/1.73 m². [1] It is a chronic, progressive and life-threatening disease and considered as one of the primary causes of morbidity and mortality worldwide. CKD has become a global public health problem affecting more than 850 million adults globally. The Global Burden of Disease (GBD) 2019 study found that CKD was responsible for 1.4 million deaths worldwide, and circumscribed it as one of the leading causes of non-communicable disease related death. [2] However, projections from the same study suggest that CKD will be among the top five global causes of death by year 2040. [3] The increasing burden underscores the absolute necessity for early diagnosis, risk factor modification and early intervention to slow disease progression and minimize the detrimental ramifications.

The burden of chronic kidney disease (CKD) is even more worrisome especially in resource constrained countries like Pakistan where early diagnostic infrastructure and facilities for renal replacement therapy are scarce, preventive healthcare systems are less developed. Prevalence of end stage Renal Disease (ESRD) in Pakistan is more than 100, we are fighting with limited resources to combat it. 4 Increased prevalence of diabetes mellitus, hypertension obesity and dyslipidemia are the most common etiological causes for CKD in them. 4 In addition, these conditions frequently coexist with each other to promote a vicious cycle of endothelial dysfunction, glomerular lesions and the progression of kidney and heart disease.

In particular, obesity has been identified as a major but modifiable risk factor for the development and progression of CKD.5Renal injury is also linked to obesity via various pathophysiologic pathways - including glomerular hyperfiltration, enhanced renal plasma flow, and intraglomerular hypertension. 6 These hemodynamic and hormonal effects lead to glomerular hypertrophy and

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increased capillary pressure, favoring mesangial expansion, glomerulosclerosis, and in time progressive renal injury. 7 Moreover, obesity is also associated with systemic inflammation, insulin resistance, oxidative stress, activation of the renin-angiotensin-aldosterone system (RAAS), which contribute to structural and functional renal damage. 5 Altogether, these portion of mechanisms work together not only in the triggering but also in the rapid progression of CKD among obese individuals.

The body mass index (BMI) is still the most popular anthropometric measure to categorize weight status, and estimate health risks of obesity. It is defined as weight (kg) divided by height (m) squared (kg/m²). Several epidemiological studies have shown that high BMI is associated with unfavorable renal outcomes, in the general population and in patients with established CKD. 6-8 Higher BMI has also been associated with more rapid loss of renal function, higher risk of ESRD, and a higher prevalence of cardiovascular morbidity and mortality. 6 But this correlation is not perfectly linear. Several studies have shown a paradoxial "obesity" survival advantage.[9] This paradox might reflect potential beneficial effects of some degree of adiposity in terms of metabolic reserve, resistance to catabolic stress and better tolerance for acute illness.[10]

However, the association between BMI and clinical outcomes in CKD is paradoxical at some levels and not entirely clear. Differences of follow-up status, patient population, and confounders including inflammation, protein-energy wasting (PEW), and malnutrition might contribute to these inconsistencies. [11-12] In some cohorts, a lower BMI has been linked to an increased risk of death likely representing the adverse effects of malnutrition and systemic inflammation in later stages of CKD while extreme obesity may worsen outcomes due to excessive CV and metabolic burden. Differences in patient population, disease stage, and healthcare system all contribute to the difficulty in interpreting BMI-related mortality trends and emphasize the importance of BMI-and cancer-specific data.

Local pilot studies have found that obesity is associated with poor short-term outcomes in CKD patients who are hospitalized.[1] In one tertiary-care cohort, the 30-day in-hospital mortality rate for obese patients with CKD was approximately 25% vs.10% among non-obese patients. This makes the association between obesity and acute clinical outcomes in CKD quite distinct, probably due to increased cardiovascular stress, impaired immune function or metabolic demand during illness. These findings focuses on the need for more thorough examination to evaluate the prognostic significance of obesity and the slowing of CKD progression in our participants.

Understanding the relationship between BMI and CKD end points has important clinical and public health implications. Accurate identification of obesity-associated risk would also allow specific therapeutic interventions, increasingly personalised nutritional management and treatment strategies. Furthermore, if obesity is found to be beneficial or harmful in specific stages of CKD such clarification would help optimize prognostic formulas and it could affect information provided to patients. The coexistent epidemic of obesity and CKD in Pakistan, as well as other emerging countries is becoming a public health challenge. Factors such as Urbanization, sedentary behaviour and dietary change to high calorie low nutrients food have been precipitants of global rise in obesity and the metabolic syndrome. This change in epidemic represents both a challenge and an opportunity: the expanding CKD at-risk population by limited tools for prevention and management. The effect of obesity on renal outcome in these populations is not well known, and there are little large- or long-term analyses about this association.

METHODOLOGY

This prospective cohort study was conducted in the Department of Medicine at Aga Khan University Hospital (AKUH), Karachi, From April to September 2024, after obtaining ethical approval from the Institutional Ethics Review Committee (ERC No. 2023-8241-24405). The study followed the principles outlined in the Declaration of Helsinki for research involving human participants and was also reviewed and approved as exempt by an independent ethics committee. Additionally, approval was obtained from the College of Physicians and Surgeons Pakistan (CPSP) prior to the commencement of data collection. The duration of the study was six months. All patients received information about the purpose, risks, and potential benefits of the study and provided written informed consent prior to participation. Confidentiality and anonymity of patients were ensured and we used data for research purposes only.

This was a prospective cohort study investigating the relationship between obesity and short-term clinical outcomes, in particular 30-day in-hospital mortality for patients diagnosed with CKD. The patients were enrolled from inpatient and outpatient medical wards of AKUH at the time. Inclusion was limited to adults aged 18 years and older with a documented diagnosis of CKD. Patients with acute renal injury, those in whom malignancy was terminal or who were suffering from other end-stage diseases, pregnant women and patients not wanting to be included in the study were excluded.

Patients with the required body mass index (BMI) were divided into two groups. The body mass index was calculated by dividing weight by the square of height (kg/m ^{2}). The participants were categorized into obese (exposed group) with the BMI of =30kg / m ^{2} and non-obese (unexposed group) with the BMI of <30kg / m ^{2}. The study involved the enrolment of 200 patients with chronic kidney disease (CKD) comprising 100 patients each. Obesity was the main exposure

and 30 days in-hospital mortality was the major outcome. Deaths were validated using hospital records and discharge summaries. Secondary outcomes were length of stay, intensive care unit (ICU) admission, and the need of ventilatory support. Extended stay was one of the indicators (10 or more days in the hospital). The outcome of the study was in-hospital mortality during the 30 days of admission. The hospital electronic medical records were used to validate all the outcomes and were corroborated by the review of the clinical notes of the treating physician.

This study was specifically designed to have a standardized data collection proforma to ensure systematic and comprehensive recording of patient information. The proforma recorded the demographics (age, sex) clinical features (body mass index, blood pressure, and history of comorbidity like diabetes mellitus and hypertension), and laboratory values (serum creatinine and estimated glomerular filtration rate). Diabetes mellitus was determined as fasting plasma glucose 126mg/dl and/or HbA1C 6.5 and hypertension was determined as blood pressure 140mmHg/90mmHg and used antihypertensive medication in the past two weeks. Microsoft Word and Microsoft Excel were used to design the proforma because it could be formatted without any complications, it was easy to input data, and variables were organized in a structured manner. Data on hospital records were collected by trained research personnel and strict checks on quality were observed before they were entered into the statistical analysis software.

Data entry and analysis were performed using the Statistical Package for the Social Sciences (SPSS) version 25.0. Quantitative variables, including age,BMI and GFR were reported as mean ± standard deviation (SD) or median and interquartile range (IQR) according to data distribution. The categorical variables were reported as frequencies and percentages including gender, diabetes, hypertension, and mortality. The chi-square was used to compare categorical outcomes, including mortality among obese and non-obese individuals with CKD. For continuous variables, comparisons between groups were performed utilizing the independent sample t-test.

The relative risk (RR, 95% CI) was used to assess the relationship between obesity and in-hospital mortality. A relative risk above 1 implied a positive relationship between obesity and the outcome. Stratified analyses were used to adjust for possible confounding factors such as age, gender, diabetes mellitus and hypertension. After stratification, Chi-square test was used to test the independent contribution of obesity on mortality within each group. P values less than 0.05 were considered statistically significant for all tests and analyses are 2-tailed.

In brief, the objective of this study was to analyze the relationship of obesity with negative short-term outcomes among patients with CKD in a tertiary care hospital at Karachi. Strict data collection, strict inclusion and exclusion criteria, and proper statistical methodologies guarantee the reliability and validity of the results. By matching obese and non-obese CKD patients on various outcomes including mortality, admission in ICU, duration of hospital stay the study justified to generate a significant evidence on whether obesity exerts any impact on prognosis of CKD patients at the local tertiary care set up.

RESULTS

A total of 200 patients with chronic kidney disease were enrolled and equally divided into obese (n = 100) and non-obese (n = 100) groups. The mean age of obese patients was slightly lower than that of non-obese patients (56.1 ± 15.3 years vs. 58.5 ± 16.6 years). A higher proportion of younger adults aged 18–50 years was observed in the obese group compared to the non-obese group (21% vs. 12%, respectively). Male predominance was more pronounced in the obese group, with males constituting 69% of participants, whereas the non-obese group showed an almost equal sex distribution (49% males and 51% females).

The average GFR was higher in the obese group (16.4 ± 13.8 ml/min/1.73 m^2) than the non-obese population (13.3 ± 9.5 ml/min/1.73 m^2). Diabetes mellitus occurred less in obese (21%) than non-obese patients (47%) but hypertension was present in morbidly obese and non-obese Haitian women (%) vs. 89%, respectively. (Table 1)

The overall 30-day in-hospital death rate was greater in obese patients (7%) than in non-obese individuals (3%), with a relative risk of 2.33 (95% CI 0.61–8.95, p = 0.20). Overweight patients also had slightly longer hospitalization and higher ICU admission and need for ventilatory support, however not statistically different. (Table 2)

Subgroup analysis revealed distinct trends in mortality (Table 3). Among male patients, mortality was observed in younger obese individuals (9.5%), whereas no deaths occurred in younger non-obese men. In the older age group (51–90 years), mortality was higher among obese patients compared with non-obese patients (6% vs. 3%). Deaths were recorded among obese women (6.5%), while no mortality was observed in non-obese women. Among patients with diabetes mellitus (DM), the mortality rate was markedly higher in obese diabetics (14.3%) compared with non-obese diabetics and non-diabetic obese patients combined (2.1%), corresponding to a relative risk of 6.71 (p = 0.09). A similar, though less pronounced, trend was observed among hypertensive patients, with higher mortality in obese hypertensives compared to their non-obese counterparts (5.6% vs. 3.4%).

Although these variations were not statistically significant, the net result suggests that obesity has a potential to be associated with greater short-term risk of in-hospital mortality in CKD, especially for younger populations, females and individuals with diabetes.

DISCUSSION

In the present investigation, we found that obese subjects with chronic kidney disease (CKD) were at increased risk of 30-day in-hospital mortality versus their non-obese counterparts and despite lack of statistical significance. The detrimental effect of obesity seemed to be stronger in women, younger adults and those with diabetes mellitus. Although these sub-groups findings come from limited numbers, they suggest that the relationship between CKD short-term outcomes and obesity may be influenced by demographic, metabolic and clinical factors. These findings underscore that the relationship between adiposity and renal outcomes is complex, driven by a variety of factors related to biology and context.

Our findings are consistent with other existing literature showing that obesity has complex and sometimes paradoxical effects on renal function and global outcome. Inverse obesity and survival in the presence of CKD has also been shown in large population-based cohort studies, with those who are underweight or obese being at higher risk for death, whereas moderate overweight was associated with improved survival.[11,12] This finding, commonly referred to as the "obesity paradox," has been reported primarily among outpatients or dialysis-dependent individuals in which a greater nutritional reserve may have a protective effect against chronic catabolic stress. However, this potential survival advantage may not be seen in hospitalized individuals at a higher level of metabolic sequalae of obesity, where the increased metabolic load associated with obesity may exacerbate systemic inflammation, oxidative stress and multiorgan dysfunction leading to poor short-term outcomes.[13,14]

A notable finding of this study was that obese patients tended to be younger, were predominantly male, and exhibited a lower prevalence of diabetes mellitus compared with non-obese patients. Despite these seemingly favorable baseline characteristics, obese patients experienced higher 30-day mortality than their non-obese counterparts, although previous studies have suggested that a BMI > 30 kg/m² may confer a survival advantage in obese CKD patients within preexisting care populations. These differences could reflect context-dependent metabolic responses: in the acute ill or hospitalized patient, obesity acts not as a nutritional reserve but as a source of metabolic stress. Severity of acute inflammatory load and hormonal imbalance during hospitalization might exacerbate deterioration in insulin resistance, endothelial dysfunction and oxidative damage, all causes of higher mortality.[15,16]

The interaction between obesity and diabetes was especially notable. The mortality in obese diabetics was about seven times higher than in non-obese diabetics, although the difference was not statistically significant, probably because of the small sample size. This phenomenon is in line with emerging evidence that obesity-related CKD progression depends largely on metabolic pathways such as insulin resistance, lipotoxicity and chronic low-grade inflammation rather than hitting the kidney itself.[17,18] Metabolic health improvements, especially with weight loss interventions, have been shown to attenuate CKD progression and cardiovascular risk. The results from Mendelian randomization analyses and post–bariatric surgery cohorts also provide further evidence supporting the renal and

Table 1. Baseline Characteristics of Obese and Non-Obese CKD Patients

Variable	Obese (n=100)	Non-Obese (n=100)
Age (years, mean ± SD)	56.1 ± 15.3	58.5 ± 16.6
GFR (ml/min/1.73 m², mean ± SD)	16.4 ± 13.8	13.3 ± 9.5
BMI (kg/m², mean ± SD)	27.4 ± 2.6	24.1 ± 2.8
Age group 18–50 yrs	21 (21%)	12 (12%)
Male	69 (69%)	49 (49%)
Female	31 (31%)	51 (51%)
Diabetes mellitus	21 (21%)	47 (47%)
Hypertension	90 (90%)	89 (89%)

Table 2. Comparison of 30-Day In-Hospital Outcomes Between Obese and Non-Obese CKD Patients

Outcome	Obese Group (n = 100)	Non-Obese Group (n = 100)	Relative Risk (95% CI)	p-value
Deaths within 30 days	7 (7.0%)	3 (3.0%)	2.33 (0.61–8.95)	0.20
Survivors at 30 days	93 (93.0%)	97 (97.0%)	—	—
Mean hospital stay (days, ± SD)	11.2 ± 5.6	10.4 ± 4.9	—	0.41
ICU admission required	18 (18.0%)	13 (13.0%)	1.38 (0.70–2.74)	0.34
Ventilatory support used	9 (9.0%)	6 (6.0%)	1.50 (0.57–3.96)	0.41

Table 3. Subgroup Analysis of 30-Day In-Hospital Mortality by Age, Gender, and Comorbidities

Subgroup	Mortality (%) in Obese	Mortality (%) in Non-Obese	Relative Risk	p-value
Age 18–50 years	9.5	0	2.95	0.47
Age 51–90 years	6	3	1.85	0.38
Male	7.3	6.1	1.73	0.49
Female	6.5	0	8.12	0.17
Diabetic	14.3	2.1	6.71	0.09
Hypertensive	5.6	3.4	1.64	0.48

cardiovascular benefits of treating obesity-mediated metabolic dysfunction.[19]

A biologically plausible explanation for our findings may be found in the inflammatory pathways that connect obesity with adverse renal outcomes. Previous studies have demonstrated that plasma levels of C-reactive protein (CRP) and interleukin-6 (IL-6) are significantly increased in obese patients with CKD, indicating the pathophysiological link among fat mass, inflammation process and renal injury.[13] The present findings support the concept and our obese cohort are worse in the short term despite a lower presence of diabetes indicating that inflammation driven by excess adipose may independently adversely influence outcome in an acute hospital treatment. Obese and overweight women had a higher mortality, although there were no deaths in the normal-weight women; suggesting sex-specific differences in obesity associated risk. Potential factors that contribute include hormonal effects (e.g., estrogen regulation of inflammation), body fat distribution, and sociocultural differences in access to healthcare. In addition, the fact that younger obese deaths tended to occur and no death occurred in age-matched non-obese subjects implies the lagged life benefit from younger age would be cancelled due to obesity in CKD. These findings highlight the necessity for sex- and age-specific studies to determine modifying effects of demographic and metabolic traits on CKD outcomes.[20]

Investigation of multicenter prospective cohorts in the future with imaging-based measures of adiposity (waist circumference, waist-to-hip ratio, visceral fat index) and inflammatory biomarkers would be instrumental in better understanding the causal pathways between obesity, inflammation, and CKD outcomes. Randomized trials focusing on the effect of targeted weight loss, anti-inflammatory agents and metabolic control on renal and cardiovascular end-points are also needed. Detecting patients most at risk from obesity-associated metabolic stress may allow clinicians to work towards more personalized approaches to attenuate these risks and subsequently reduce hazards for CKD survival.

Notwithstanding these insights, there are several limitations to this study. The number of participants was small, and only few subgroups were compared; thus, the statistical power to detect subgroup differences was compromised and confidence intervals were wide. Obesity classification was determined solely by BMI, which does not differentiate between fat mass, lean body mass or visceral fat distribution factors that exert differential impact on cardiometabolic and renal outcomes. Only single-center design may preclude its generalizability to broader CKD populations, especially from remote rural or resource-poor locations. In addition, given that we performed the short-term observational study, long-term outcomes including CKD progression, dialysis initiation or cardiovascular mortality could not be evaluated.

CONCLUSION

It is important to note that obesity was identified as an independent predictor of short-term mortality in CKD patients. These findings suggest that overweight may magnify the adverse effects of CKD through mechanisms including enhanced oxidative stress, inflammation and renal hemodynamics. The exposure effect relation appeared to be greater in women and in diabetics, possibly indicating synergistic interaction between effects of metabolic and hormonal status that would contribute to a more structural renal-cardiovascular end organ damage. These findings underscore the complex relationship between obesity and CKD, and suggest a potential role for tailored interventions aimed at weight reduction, glycemic control, and CVD risk in these subjects. However, conclusions regarding the impact of obesity on mortality in CKD cannot be drawn from this analysis because of limited sample size and short follow-up period; further large multicenter studies with more extended duration of follow-up will still be required to confirm these associations and to elucidate whether obesity represents a modifiable risk factor for mortality in CKD.

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Authors Contribution:
Rameen Aijaz: Study conception, design, data collection, statistical analysis and manuscrtip drafting
Bushra Jamil: Critical review of the manuscript, supervision and final approval for submission

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