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Obesity as a Risk Factor for the Development and Progression of Chronic Kidney Disease: A Systematic Review

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Abstract

***Objectives*:** To conduct a systematic review of existing literature to assess the relationship between obesity and CKD. ***Methods*:** A detailed computerized search of relevant databases was conducted to identify studies that met the inclusion criteria. The search encompassed PubMed, SCOPUS, Science Direct, Cochrane Library, and Web of Science to find pertinent research. ***Results*:** Our analysis included seven studies with a total of 21,191 obese patients and males comprised less than half of the participants, totaling 6675 (31.5%). The prevalence of CKD among obese individuals ranged from 7% to 48.9%. The results of this systematic review indicate a clear association between obesity and chronic kidney disease (CKD). Across multiple studies, obesity, particularly abdominal obesity, was found to significantly increase the risk of developing and progressing CKD, with higher body mass index (BMI) and waist-to-hip ratio (WHR) correlating with greater renal dysfunction. Key mechanisms involved include renal fat deposition, increased glomerular filtration rate (GFR) decline, and metabolic disturbances. ***Conclusion*:** This systematic review provides strong evidence that obesity is a significant risk factor for the development and progression of chronic kidney disease. The reviewed studies highlight the complex interplay between obesity, metabolic dysfunction, and renal impairment, underscoring the need for early identification and management of obesity in patients at risk for CKD.

Keywords: Obesity, Body Mass Index, Chronic Kidney Disease, Chronic Renal Failure, Systematic Review.

INTRODUCTION

Obesity is known to raise the risk of certain chronic diseases [1]. The global obesity epidemic has escalated into a public health catastrophe that affects many countries. In the United States, obesity is currently the second biggest cause of avoidable disease and death, trailing only smoking. There has been a growing interest in the involvement of the obesity pandemic in the risk of CKD [2, 3], owing in part to the concomitant significant rise in the incidence of end-stage renal disease (ESRD) [4], which has nearly doubled in the last decade. By 2010, the number of ESRD patients in the US is expected to reach 650,000, resulting in $28 billion in medical care costs [5].

Up to this point, no definitive conclusion has been established on whether obesity alone, without secondary metabolic disorders, leads to the onset of CKD or whether the link between MetS and CKD varies with weight fluctuation. The variability of obesity phenotypes, including the existence or absence of concurrent metabolic disorders, may explain the complexities of the link between BMI and CKD. Obesity is linked to CKD through multiple pathways, including enhanced glomerular filtration, adipose tissue inflammation, insulin resistance, and activation of the renin-angiotensin-aldosterone system (RAAS), which all contribute to kidney damage over time [6].

Obesity-related glomerulopathy is a separate entity that highlights the direct impact of increased body weight on renal structure and function [7]. Understanding obesity's role in CKD etiology is crucial for developing preventive and therapeutic measures to reduce the global burden of renal disease [8].

Despite the growing recognition of obesity as a key contributor to CKD, the complex interplay between

metabolic, hormonal, and inflammatory factors that drive kidney injury in obese individuals remains incompletely understood. Moreover, while weight loss interventions have shown promise in improving renal outcomes, there is a need for comprehensive data to establish effective clinical guidelines for the management of obesity in patients at risk for CKD. This systematic review aims to synthesize the current literature on the relationship between obesity and CKD, providing a detailed analysis of the underlying mechanisms, the role of obesity in CKD progression.

METHODS

This study conducted a systematic review following the guidelines set by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [9]. We performed an electronic search to identify relevant English-language articles that explored how the relationship between obesity and CKD. The search encompassed databases such as PubMed, Web of Science, SCOPUS, and Science Direct. Our search strategy included keywords related to CKD incidence among obese patients. Independently analyzing the search results, two reviewers chose eligible studies, retrieved data, and used appropriate assessment instruments to gauge the caliber of the included study.

Eligibility Criteria

Inclusion Criteria

1. Population: Obese adult patients (≥18 years).
2. Intervention: Studies that assess the incidence and interpretation of CKD.
3. Outcomes: Studies reporting on clinical outcomes, including but not limited to glomerular filtration rate (GFR) and BMI.
4. Study Design: Observational studies, randomized controlled trials (RCTs), cohort studies, and case-control studies published in peer-reviewed journals.
5. Language: English-language articles published.
6. Time Frame: Articles published within 2022-2024, or a specified relevant time frame defined by the researchers.

Exclusion Criteria

1. Population: Studies involving pediatric patients (<18 years) or patients with chronic conditions that significantly affect the kidney (e.g., diabetes).
2. Intervention: Studies that do not clearly discuss the incidence of CKD in obese patients.
3. Outcomes: Studies that do not report relevant outcomes.
4. Study Design: Case reports, qualitative studies, literature reviews, editorials, and studies with insufficient methodological quality.
5. Language: Studies not published in English.
6. Duplicated Data: Studies that have overlapping patient populations or outcomes reported in other included studies.

Data Extraction

Rayyan (QCRI) was utilized to verify the accuracy of the search results [10]. The search produced titles and abstracts, which were assessed for relevance based on the established inclusion and exclusion criteria. The research team meticulously reviewed all studies meeting these criteria. Any disagreements were resolved through discussion and consensus. Key study data were systematically recorded using a predefined extraction form, including titles, authors, publication year, study location, participant demographics, gender distribution, follow-up duration, cut-off point for obesity diagnosis, GFR, BMI, prevalence of CKD, and main outcomes. An unbiased evaluation tool was created to assess the potential for bias in the included studies.

Data Synthesis Strategy

Summary tables were developed using information from relevant studies to provide a qualitative overview of the research findings and key elements. After completing data collection for the systematic review, the most effective method for utilizing the information from the included studies was determined.

Risk of Bias Assessment

The Joanna Briggs Institute (JBI) [11], critical evaluation criteria for studies reporting prevalence data were employed to assess the quality of the studies. This evaluation tool comprises nine questions, where a positive response earns a score of 1, and a negative, ambiguous, or irrelevant response receives a score of 0. Scores were categorized into quality levels as follows: below 4 for low quality, between 5 and 7 for moderate quality, and above 8 for high quality. Independent researchers assessed the quality of the papers, and any disagreements were resolved through discussion.

RESULTS

Systematic Search Outcomes

A thorough search of 2001 study papers yielded 1119 duplicates that were disregarded. After reviewing the titles and abstracts of 882 studies, 698 articles were rejected. Out of the 184 reports that were necessary, 6 were not found. 116 papers were excluded because the study results were inaccurate, 4 were editor's letters, and 6 were abstracts. 45 were disqualified for using the wrong demographic types. The qualifying requirements are met by the seven research publications that comprise this systematic review. A diagram illustrates the process by which the literature was selected in **Figure 1.**

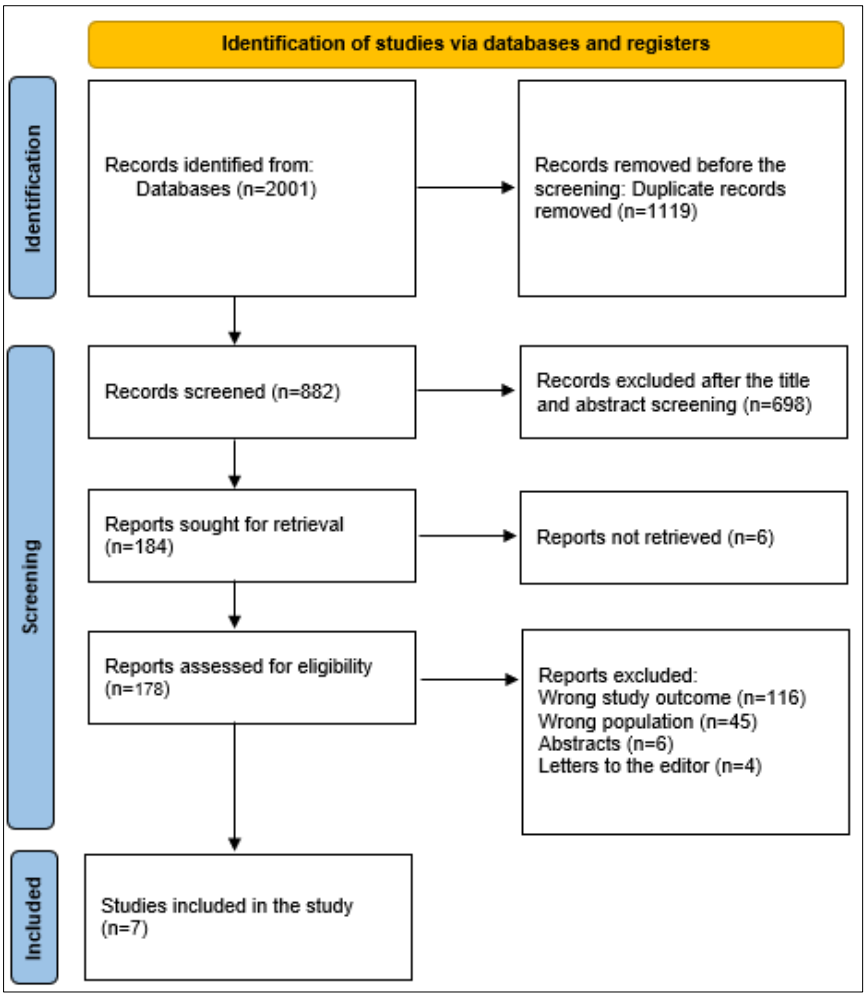

Figure 1: A PRISMA diagram is employed to encapsulate the research decisions.

Socio demographics and Clinical Parameters of the Involved Participants and Studies

Table 1 summarizes the socio-demographic data from the research articles. Our analysis included seven studies with a total of 21,191 obese patients and males comprised less than half of the participants, totaling 6675 (31.5%). The studies consisted of four prospective cohort analyses [13-18], two cross-sectional studies [16, 17], and one case-control [12]. Geographically, three studies were conducted in China [12-17], and one each in Korea [13], the UK [15], Iran [16], and Morocco [18].

Table 2 shows the clinical parameters obesity is consistently linked with various negative outcomes related to CKD. Studies have shown that individuals with obesity, as defined by a BMI above 30 kg/m², have increased levels of renal fat, as observed through MRI. This accumulation of fat in and around the kidneys negatively impacts GFR and contributes to early kidney injury. Additionally, higher BMI and WHR were associated with a higher risk of CKD, with weight control, exercise, and proper food intake emerging as significant factors in mitigating this risk.

The prevalence of CKD in obese populations varied across studies but remained a prominent concern. The prevalence of CKD among obese individuals ranged from 7% [13] to 48.9% [15]. In some studies, it was observed that individuals with abdominal obesity were more likely to develop advanced kidney disease, suggesting that abdominal fat may play a role in modulating the relationship between metabolic-associated fatty liver disease (MAFLD) and CKD. However, one study found that the protective effect of obesity against CKD was only present in specific groups, particularly those with CKD alone, and not in individuals with comorbid conditions such as heart failure or diabetes.

Furthermore, other studies indicated that while overweight and obesity were related to CKD development, they were not consistently linked to increased risk. Certain populations, particularly older individuals with elevated BMI and other metabolic conditions such as hypercholesterolemia, were more susceptible to CKD, potentially due to hemodynamic changes at the glomerular level, activation of the renin-angiotensin-aldosterone system, and inflammatory processes.

Table 1: Sociodemographic parameters of the comprised research

Study ID	Study design	Country	Participants (n)	Mean age	Males (%)
Tang *et al*., 2024 [12]	Case-control	China	56	50.5	33 (58.9%)
Song *et al*., 2024 [13]	Prospective cohort	Korea	1,542	50.2 ± 8.2	42 (33.6%)
Cen *et al*., 2024 [14]	Prospective cohort	China	3790	43.2 ± 0.16	1206 (31.8%)
Al-Chalabi *et al*., 2024 [15]	Prospective cohort	UK	902	63	535 (59.3%)
Jam *et al*., 2022 [16]	Cross-sectional	Iran	14,463	47.3 ± 8.3	4701 (48.2%)
Chen *et al*., 2022 [17]	Cross-sectional	China	400	64.5 ± 8.5	141 (35.3%)
El Jadi *et al*., 2024 [18]	Prospective cohort	Morocco	38	36.8±10.7	17 (44.7%)

Table 2: Clinical parameters and outcomes of the comprised research

Study ID	Follow-up (years)	Obesity cut-off (kg/m^2)	BMI (kg/m^2)	GFR ($mL/min/l.73m^2$)	CKD prevalence (%)	Main outcomes	JBI
Tang *et al*., 2024 [12]	NR	≥28	30.1	96. ± 21.1	NR	Obese participants showed greater MR-renal fat biomarker levels assessed on Dixon-based MRI than non-obese participants. All MR-renal fat biomarkers exhibit a negative connection with eGFR, and perirenal fat thickness may have prognostic value for early kidney injury due to renal deposits of fat in obesity.	Moderate
Song *et al*., 2024 [13]	11.03 ± 4.2 y	≥ 25	NR	90.9 ± 15.7	190 (7%)	High BMI and WHR were linked to the incidence of CKD. Weight loss, exercise, and food control can all help to minimize fat tissue and obesity, hence protecting renal function in people who are in good health.	Moderate
Cen *et al*., 2024 [14]	30	≥ 30	32.9 ± 0.13	75.9 ± 0.4	1075 (28.4%)	Individuals with abdominal obesity were more probable to have advanced kidney disease. Abdominal obesity could modulate the link between MAFLD and CKD.	High
Al-Chalabi *et al*., 2024 [15]	22-97 m	≥ 30	NR	17.7-49.7	441 (48.9%)	Obesity could safeguard against negative results only in groups 1 (CKD alone) and 2 (CKD plus HF). This "protective" effect was not observed in patients with concurrent diabetes.	Moderate
Jam *et al*., 2022 [16]	5	≥ 30	27.5±4.6	<60	1058 (7.3%)	Overweight/obesity has been demonstrated to be independently but not substantially linked with an increased risk of CKD.	Moderate
Chen *et al*., 2022 [17]	NR	≥ 25	24.55 ± 3.57	96.3 ± 46.2	81 (20.3%)	NR	Moderate

Study ID	Follow-up (years)	Obesity cut-off (kg/m^2)	BMI (kg/m^2)	GFR ($mL/min/l.73m^2$)	CKD prevalence (%)	Main outcomes	JBI
El Jadi *et al*., 2024 [18]	3 m	≥ 30	37.4 ± 2.2	107.8 ± 5.9	4 (10.5%)	Obesity is associated with chronic renal disease. This kidney risk is caused by reactions among hemodynamic parameters at the glomerular threshold, stimulation of the renin-angiotensin-aldosterone network and the autonomic nervous system, and the presence of pro-inflammatory and pro-fibrosing agents. The elderly obese individuals who had elevated BMI, hypercholesterolemia, and hyperLDLemia were the most affected.	High

DISCUSSION

Our review reported a prevalence of CKD among obese individuals ranged from 7% [13], to 48.9% [15]. The results of this systematic review indicate a clear association between obesity and CKD. Across multiple studies, obesity, particularly abdominal obesity, was found to significantly increase the risk of developing and progressing CKD, with higher body mass index (BMI) and metabolically healthy with normal weight correlating with greater renal dysfunction. Key mechanisms involved include renal fat deposition, increased GFR decline, and metabolic disturbances. Similarly, Wang *et al.,* found that obesity raises the risk of KD in people in general, and this link appears to be higher in women than in men. Obesity has a negative impact on the progression of KD in patients with renal disorders [19]. But in contrast, Zang *et al.,* found that individuals with aberrant metabolic status are much more likely to develop CKD, regardless of BMI. For metabolically healthy people, the risk of CKD increases as BMI rises, and obese people are at a much greater risk [20].

In terms of whether an appropriate pattern of higher weight contributes to the occurrence of CKD, our current study found that only MHOW persons had an obvious increased risk for CKD. Existing research suggests that excess visceral adipose tissue can cause a feedback loop in which obesity-induced losses in kidney function contribute to the onset of hypertension by activating the sympathetic nerve and renin-angiotensin systems [21]. Computed tomography and MRI data also indicated that metabolically healthy obese people had a lower percentage of visceral adiposity and the amount of liver fat [22, 23].

The findings from this review have important implications for clinical practice. Given the strong association between obesity and CKD, healthcare providers should prioritize the identification and management of obesity in patients at risk for renal impairment. Weight loss interventions, including diet modification, exercise, and possibly bariatric surgery, should be considered as part of a comprehensive strategy to prevent CKD or slow its progression in obese individuals. Additionally, regular monitoring of kidney function in obese patients may help identify early signs of CKD, allowing for timely interventions to prevent further renal damage. Importantly, these interventions should be tailored to the individual patient, taking into account factors such as age, comorbid conditions, and overall health status.

Strengths and Limitations

One of the primary strengths of this review is the comprehensive search strategy employed to identify relevant studies across multiple databases. This approach allowed for the inclusion of a diverse range of studies, enhancing the generalizability of the findings. Furthermore, the large sample size included in the review strengthens the conclusions, as it enables more robust analyses of the association between obesity and CKD. The inclusion of studies with long-term follow-up periods also adds depth to the analysis, allowing for an assessment of the long-term effects of obesity on CKD progression.

Despite its strengths, this review has several limitations that warrant consideration. First, the studies included in the review vary widely in terms of study design, population characteristics, and definitions of obesity and CKD, which may introduce heterogeneity in the results. Second, many of the studies relied on self-reported data for BMI and other obesity-related measures, which could result in misclassification bias. Additionally, the observational nature of the included

studies precludes the ability to establish a definitive causal relationship between obesity and CKD. Confounding factors such as physical activity, diet, and socioeconomic status may also play a role in the observed associations but were not consistently controlled for across studies.

CONCLUSION

This systematic review provides strong evidence that obesity is a significant risk factor for the development and progression of chronic kidney disease. The reviewed studies highlight the complex interplay between obesity, metabolic dysfunction, and renal impairment, underscoring the need for early identification and management of obesity in patients at risk for CKD. While weight loss interventions offer promise in mitigating CKD risk, further research is needed to refine treatment strategies and improve outcomes for obese individuals with CKD. Addressing obesity as part of a comprehensive approach to kidney health could have a profound impact on reducing the global burden of CKD and improving patient outcomes.

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