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Metabolic Syndrome as A Risk Factor for The Development of Kidney Dysfunction: A Comprehensive Systematic Review

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ABSTRACT

Background: The prevalence of metabolic syndrome increased by 35% in the United States starting in the 1980s, according to the Centers for Disease Control and Prevention (CDC). Changes in the structure and function of the kidneys have been related to metabolic syndrome. Reduced eGFR (estimated glomerular filtration rate) and proteinuria risk have been linked to metabolic syndrome and its various components. Moreover, numerous investigations revealed a correlation between reduced eGFR and metabolic syndrome. **The aim:** This study aims to investigate metabolic syndrome as a risk factor for the development of kidney dysfunction. **Methods:** By comparing itself to the standards set by the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020, this study was able to show that it met all of the requirements. So, the experts were able to make sure that the study was as up-to-date as it was possible to be. For this search approach, publications that came out between 2014 and 2024 were taken into account. Several different online reference sources, like Pubmed and ScienceDirect, were used to do this. It was decided not to take into account review pieces, works that had already been published, or works that were only half done. **Results:** In the PubMed database, the results of our search brought up 80 articles, whereas the results of our search on ScienceDirect brought up 164 articles. The results of the search conducted by title screening yielded a total of 17 articles for PubMed and 23 articles for ScienceDirect. We compiled a total of 20 papers, 14 of which came from PubMed and 6 of which came from ScienceDirect. We excluded 1 review article, 1 non-full text article, 1 article having insufficient outcomes, and 2 articles having ineligible subjects. In the end, we included fifteen research that met the criteria. **Conclusion:** Metabolic syndrome is a risk factor for the development of kidney dysfunction. Metabolic syndrome increases the incidences of CKD and proteinuria and decreases GFR. These findings suggest that it is important to screen for CKD in patients with metabolic syndrome.

Keywords: metabolic syndrome, kidney dysfunction, eGFR, association

INTRODUCTION

The syndrome metabolic also called Syndrome X, is a metabolic disorder characterized by abdominal obesity, hypertension, insulin resistance, and atherogenic dyslipidemia.[1,2] The health and expense of medical treatment of an individual are significantly impacted by metabolic syndrome.[3] The fundamental causes of syndrome metabolic syndrome are obesity, inactivity, genetic predisposition, diet, and sedentary life.[1,3] The Centers for Disease Control and Prevention (CDC) report that from the 1980s the prevalence of metabolic syndrome increased by 35% in the United States.[4] The prevalence of metabolic syndrome in persons 18 years of age and older is high in the United States[3]

According to Modified NCEP ATP III 2010 criteria diagnosed for metabolic syndrome are the presence of 3 or more of the following criteria: 1) fasting plasma glucose ≥100 mg/dL, 2) waist circumference Male: >102 cm; Female: >88 cm (Asian origin : Male: > 90 cm and Female: >80 cm), 3) blood pressure ≥130/≥85 mmHg or current use of antihypertensive drugs, 4) HDL cholesterol, Male: <40 mg/dL; Female <50 mg/dL, and 5) triglycerides ≥150 mg/dL.[5] The main cause of metabolic syndrome is excess weight. Insulin resistance is the result of tissue dysfunction and an accumulation of adipose tissue. The increased adipose tissue releases proinflammatory cytokines, including resistin, leptin, adiponectin, plasminogen activator inhibitor, and tumor necrosis factor, which negatively affects and modify insulin handling.[3] Insulin resistance leads to microvascular damage, which puts a patient at risk for vascular resistance, endothelial dysfunction, hypertension, and inflammation of the vessel walls. Damage to the endothelium can affect the body's equilibrium, leading to the development of hypertension and atherosclerotic disease.[3]

Nephrologists are paying more attention to the consequences of metabolic syndrome on the kidneys as its prevalence rises.[5] Metabolic syndrome has been linked to modifications in renal structure and function.[5] In clinical settings, determining the estimated glomerular filtration rate (eGFR) and looking for proteinuria (albuminuria) are the most practical methods of evaluating renal

function.[6] The normal for adult eGFR is 90 to 120 mL/minute.[6] Metabolic syndrome and its components were associated with decreased eGFR (estimated glomerular filtration rate) and the risk of proteinuria.[7,8] Several studies showed the association between metabolic syndrome and decreased eGFR. The involvement of each risk metabolic component should be considered when developing a preventive approach for kidney function decline. Qiu et al study found that the risk of decreased eGFR was found to be independently linked to metabolic syndrome, and the various components (abdominal obesity, elevated triglycerides, reduced HDL cholesterol, elevated fasting glucose, and elevated blood pressure) of metabolic syndrome have distinct effects on decreased eGFR.[7] The purpose of this study is to investigate metabolic syndrome as a risk factor for the development of kidney dysfunction.

METHODS

Protocol

By following the rules provided by Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020, the author of this study made certain that it was up to par with the requirements. This is done to ensure that the conclusions drawn from the inquiry are accurate.

Criteria for Eligibility

For the purpose of this literature review, we investigate metabolic syndrome as a risk factor for the development of kidney dysfunction. It is possible to accomplish this by researching or investigating the incidence of CKD, the decrease in GFR, and proteinuria. As the primary purpose of this piece of writing, demonstrating the relevance of the difficulties that have been identified will take place throughout its entirety.

In order for researchers to take part in the study, they needed to fulfil the following requirements: 1) The paper needs to be written in English, and it needs to determine the association between metabolic syndrome and the development of

kidney dysfunction. In order for the manuscript to be considered for publication, it needs to meet both of these requirements. 2) The studied papers include several that were published after 2014, but before the time period that this systematic review deems to be relevant. Examples of studies that are not permitted include editorials, submissions that do not have a DOI, review articles that have already been published, and entries that are essentially identical to journal papers that have already been published.

Search Strategy

We used "metabolic syndrome"; "kidney dysfunction"; "eGFR"; and "association" as keywords. The search for studies to be included in the systematic review was carried out from February, 24th 2024 using the PubMed and ScienceDirect databases by inputting the words: (("metabolic syndrome"[MeSH Terms] OR ("metabolic"[All Fields] AND "syndrome"[All Fields]) OR "metabolic syndrome"[All Fields]) AND (("kidney"[MeSH Terms] OR "kidney"[All Fields] OR "kidneys"[All Fields] OR "kidney s"[All Fields]) AND ("dysfunctional"[All Fields] OR "dysfunctionals"[All Fields] OR "dysfunctioning"[All Fields] OR "dysfunctions"[All Fields] OR "physiopathology"[MeSH Subheading] OR "physiopathology"[All Fields] OR "dysfunction"[All Fields])) AND ("erbb receptors"[MeSH Terms] OR ("erbb"[All Fields] AND "receptors"[All Fields]) OR "erbb receptors"[All Fields] OR "egfr"[All Fields]) AND ("associate"[All Fields] OR "associated"[All Fields] OR "associates"[All Fields] OR "associating"[All Fields] OR "association"[MeSH Terms] OR "association"[All Fields] OR "associations"[All Fields])) AND (y_10[Filter]) AND (english[Filter]) used in searching the literature.

Data retrieval

After reading the abstract and the title of each study, the writers performed an examination to determine whether or not the study satisfied the inclusion criteria.

The writers then decided which previous research they wanted to utilise as sources for their article and selected those studies. After looking at a number of different research, which all seemed to point to the same trend, this conclusion was drawn. All submissions need to be written in English and can't have been seen anywhere else.

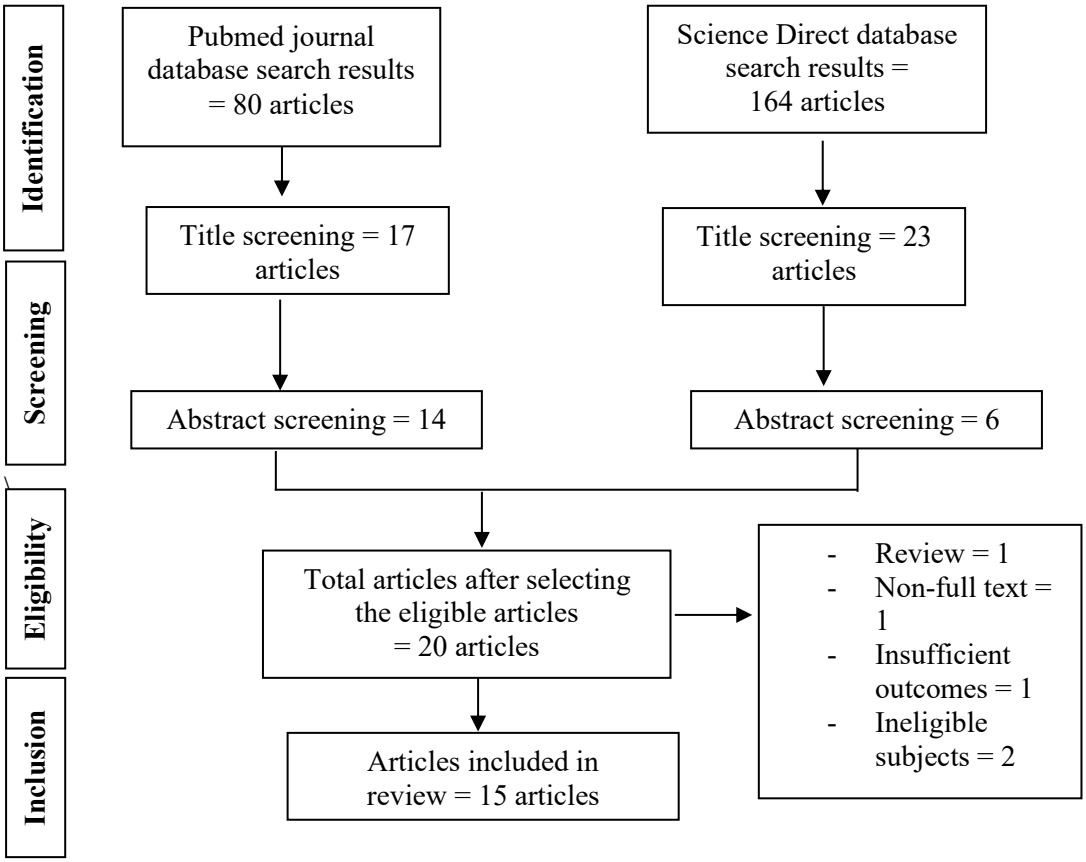

Figure 1. Article search flowchart

Only those papers that were able to satisfy all of the inclusion criteria were taken into consideration for the systematic review. This reduces the number of results to only those that are pertinent to the search. We do not take into consideration the conclusions of any study that does not satisfy our requirements. After this, the findings of the research will be analysed in great detail. The following pieces of information were uncovered as a result of the inquiry that was carried out for the purpose of this study: names, authors, publication dates, location, study activities, and parameters.

Quality Assessment and Data Synthesis

Each author did their own study on the research that was included in the publication's title and abstract before deciding on which publications to explore further. The next step will be to evaluate all of the articles that are suitable for inclusion in the review because they match the criteria set forth for that purpose in the review. After that, we'll determine which articles to include in the review depending on the findings that we've uncovered. This criteria is utilised in the process of selecting papers for further assessment to simplify the process as much as feasible when selecting papers to evaluate. Which earlier investigations were carried out, and what elements of those studies made it appropriate to include them in the review, are being discussed here

RESULT

In the PubMed database, the results of our search brought up 80 articles, whereas the results of our search on ScienceDirect brought up 164 articles. The results of the search conducted by title screening yielded a total of 17 articles for PubMed and 23 articles for ScienceDirect. We compiled a total of 20 papers, 14 of which came from PubMed and 6 of which came from ScienceDirect. We excluded 1 review article, 1 non-full text article, 1 article having insufficient outcomes, and 2 articles having ineligible subjects. In the end, we included fifteen research that met the criteria.

Table 1. The literature included in this study

Author	Origin	Method	Sample Size	Result
Chen, 2017[9]	China	Cross-sectional study	9,693 patients with MS	This study revealed that among Chinese urban populations, metabolic syndrome is prevalent and linked to CKD. The number of metabolic syndrome (MS) components and the risk of chronic kidney disease were correlated in a graded manner.
Guo, 2023[10]	China	Cross-sectional study	43 patients with MS	The results of this study showed that in individuals with CKD, there is a strong association between urine protein and eGFR as well as MS components. In order to minimize proteinuria and postpone the onset of CKD, early intervention and modification of MS components are crucial.

Huh, 2017[11]	Korea	Prospective cohort study	6,065 subjets	This 10-year follow-up cohort study unequivocally showed that individuals with MS are more likely to get acute CKD as well as a rapid drop in eGFR.
Kang, 2014[12]	Korea	Cross-sectional study	10,253,085 participants	This study verified MS as a potent and distinct risk factor for both men and women with CKD, as well as each of its component parts. Furthermore, we discovered a graded correlation between the risk for CKD and the quantity of MS components.

Kawamoto, 2019[13]	Japan	Prospective cohort study	419 patients with MS	This study suggested the reduction in eGFR was significantly correlated with MS. This study reveals that, among Japanese middle-aged and older people without CKD who live in communities, low HDL-C and increased HbA1c are more significant factors in the influence of MetS on eGFR reduction than obesity.
Kurata, 2016[14]	Japan	Cross-sectional study	18 patients with MS	The resultsshowed the correlations between increased prevalence of CKD in nonobese senior Japanese women and the existence of MS and the number of MS components. These correlations may be mediated by elevated blood pressure.
Li, 2014[15]	China	Prospective cohort study	394 patients with MS	The findings suggested that even in those without diabetes or hypertension, a Chinese rural cohort with normal renal function showed that MS was strongly linked to the likelihood of both rapid eGFR decline and renal function decline (RFD).
Ma, 2018[16]	China	Prospective cohort study	3,237 participants	This study discovered that in Chinese adults, CKD progressed more quickly due to both insulin resistance (IR) and MS.
Medeiros, 2017[17]	Brazil	Cross-sectional study	78 participants	This study showed a strong relationship between renal impairment and an increase in the number of MS components in young adult patients who had high WC and BMI.
Moustakim, 2021[18]	Morocco	Cross-sectional study	210 participants	This study demonstrated a separate link between MS and a higher risk of CKD and reduced eGFR.
Qiu, 2019[19]	USA	Cross-sectional study	33,300 participants	The results of this study showed that each component of MS has a varied influence on the decreased eGFR, but generally, the study population and several subgroups are linked to an increased risk of decreased eGFR due to MS.

Song, 2015[20]	Korea	Cross-sectional study	3,437 participants	This study showed that obesity predicts incident CKD regardless of metabolic health state, while MS predicts widespread CKD independent of weight status. Regardless of historical metabolic health and present weight status, incident CKD is also linked to current MS.
Stefansson, 2018[21]	Norway	Prospective cohort study	1,261 participants	This study suggested that for people in general, metabolic syndrome is an independent risk factor for a faster age-related GFR decline.
Wang, 2020[22]	China	Prospective cohort study	6,060 patients with MS	According to this research, MS and overweight/obesity independently predicted incidence of CKD in Chinese people. Those who met the criteria for both MS and overweight/obesity had the highest risk.
Zammit, 2015[23]	USA	Cross-sectional study	616 participants	These findings suggested that MS is linked to CKD in older persons without diabetes. The likelihood of developing CKD rose with the number of MS components, according to the results.

Incidences of Chronic Kidney Disease & Their Relation with Metabolic Syndrome

Thirteen of the fifteen identified studies suggested there was a significant association between metabolic syndrome and CKD development. Metabolic syndrome was the risk factor for the incidences of CKD.[9–18,20,22,23] Huh, et al. (2017)[11] showed that subjects with MS had a significant correlation with an elevated incidence of incident CKD (MS 373 (21.09%) vs 520 (12.10%) P = <.0001) when compared to subjects without MS. Ma, et al. (2018)[16] showed that for the total cohort, the incidence of CKD was 20.08 per 1000 person-years. Chen, et al. (2017)[9] showed that individuals with MS were 2.43 times more likely than non-MS participants to have CKD. Participants with MS had 1.99-fold higher odds of having CKD than those without MS, according to age- and gender-adjusted odds ratios ($p < 0.001$). Kurata, et al. (2016)[14] showed that women with MS were more likely to have CKD than women without it.

Kang, et al. (2014[12]) showed that compared to individuals without MS, those with MS showed a greater prevalence of CKD (10.48% vs. 4.91%, P,0.001). In total, 239,137 patients with CKD (n = 37.93%) had 40.04% abdominal obesity, 34.46% hypertriglyceridemia, 27.1% low HDL cholesterol, 62.74% high blood pressure, and 43.61% high fasting glucose. Of these, MS patient percentages were recorded. As the stage of CKD grew, so did the prevalence of MS. When compared to individuals without MS, those with MS showed a 1.884-fold higher OR (95% CI 1.867–1.902, P, 0.001) for proteinuria in multivariate logistic regression models. Song, et al. (2015)[20] showed that regardless of weight status, the risks for prevalent CKD rose in metabolically unhealthy categories when there was a difference between those without MS and obesity (odds ratio 4.19 (95 % CI 2.03–8.62) for non-obese with MS and odds ratio 4.63 (95 % CI 2.05–10.49) for those with MS and obesity). Wang, et al. (2020)[20] showed a 50% higher risk of CKD was linked to MS (OR 1.50, 95% CI 1.31-1.73). In individuals who were overweight or obese (OR 1.56, 95% CI 1.32e1.84) and those who weren't (OR 1.55, 95% CI 1.22-1.97), MS was linked to a greater risk of chronic kidney disease (CKD). Abdominal obesity, elevated TG, low HDL, and elevated blood pressure are among the elements of MS that have been independently linked to an increased risk of CKD.

Glomerular Filtration Rate

Twelve of the fifteen identified studies suggested metabolic syndrome was the risk factor for the decline of GFR.[9–13,15,16,18–21,23] Chen, et al. (2017)[9] showed that reduced renal function (eGFR < 60 mL/min/1.73 m2) was associated with MS. According to the unadjusted regression analysis, high blood pressure (OR 1.90, 95% CI 1.50–2.39), high triglycerides (OR 1.35, 95% CI 1.07–1.69), high fasting glucose (OR 3.26, 95% CI 2.50–4.26), and obesity (OR 1.64, 95% CI 1.31–2.07) were all linked to impaired renal function. Guo, et al. (2023)[10] showed that patients with varying stages of chronic kidney disease (CKD) exhibited variations in renal function and MS indexes. Additionally, eGFR was significantly negatively correlated with abdominal circumference, TG, SBP, DBP, and FPG (r = −0.526,

−0.412, −0.582, −0.396, −0.435, all $p < 0.05$) and significantly positively correlated with HDL-C ($r = 0.356$, $p < 0.05$) in the Pearson correlation analysis.

Qiu, et al. (2019)[19] showed that after controlling for age, gender, marital status, educational level, smoking status, alcohol consumption, physical activity, history of coronary artery disease, BMI, systolic blood pressure, FPG, triglycerides, and HDL cholesterol, the risk of decreased eGFR was positively associated with MS, abdominal obesity, elevated triglycerides, and elevated blood pressure, with adjusted ORs of 1.76 (95% CI 1.53–2.01; $p < 0.001$), 1.66 (95% CI 1.44–1.93; $p < 0.001$), 1.37 (95% CI 1.18–1.60; $p < 0.001$), and 1.92 (95% CI 1.57–2.35; $p < 0.001$), respectively. Song, et al. (2015)[20] showed that GFR was lower in those with MS compared to those without MS ($P < 0.001$) after adjusting for concurrent BMI and other covariates. In addition, Qiu, et al. (2019)[19] and Moustakim, et al. (2021)[18] showed that when compared to those without metabolic components, patients with more metabolic components had a greater chance of experiencing a decline in eGFR.

Ma, et al. (2018)[16] showed that of the 1394 people who had a little lower eGFR at baseline, 182 had CKD (incidence rate: 43.52 per 1000 person-years). In all, 33.28 person-years were affected by a modestly lowered eGFR. Kang, et al. (2014)[12] showed that the eGFR 60 mL/min/1.73 m2 (OR 1.364, 95% CI 1.355–1.373, P,0.001) and CKD (OR 1.526, 95% CI 1.518–1.535, P,0.001) were significantly influenced by the presence of MS in comparison to those without MS. Kawamoto, et al. (2019)[13] showed the annual eGFR decline rate in persons with MS significantly decreased with age ($r = -0.104$, $p = 0.035$). Li, et al. (2014)[15] showed following a median of 7.1 years, there was a 19.8% quick drop in eGFR. However, when evaluating the GFR decline >3 ml/min per 1.73 m^2/yr, Stefansson, et al. (2018)[21] showed that there was no significant difference in the GFR in those with metabolic syndrome was 47 (12.3%) vs 82 (9.3), $p = 0.11$.

Proteinuria

Two studies reported proteinuria as their outcome.[9,10] Chen, et al. (2017)[9] showed that high blood pressure (OR 2.78, 95% CI 2.27–3.42), high triglycerides

(OR 1.87, 95% CI 1.56–2.23), high fasting glucose (OR 304, 95% CI 2.46–3.77), and obesity (OR 1.57, 95% CI 1.30–1.89) were all linked to the presence of proteinuria in the unadjusted adjusted regression analysis. Following age and sex adjustments, proteinuria was linked to high blood pressure (OR 2.13, 95% CI 1,72–2.66), high triglycerides (OR 1.80, 95% CI 1.50–2.15), high fasting glucose (OR 2.40, 95% CI 1.92–3.00), and obesity (OR 1.35, 95% CI 1.12–1.63). Guo, et al. (2023)[10] showed that proteinuria showed a positive correlation ($r = 0.412, 0.362, 0.359, 0.647, 0.558$, all $p < 0.05$) with abdominal circumference, TG, SBP, DBP, and FPG, while there was a negative correlation ($r = -0.485$, $p < 0.05$) with HDL-C.

DISCUSSION

The incidence of MS has increased in recent years due to increased consumption of high-calorie foods and sugary drinks, leading to an increase in overweight and obesity. In 1989, Dr. Norman Kaplan referred to a “deadly quartet” of MS which is upper body obesity, diabetes, hypertension, and hypertriglyceridemia. Research has indicated that those with MS may also have a higher chance of developing renal dysfunction and microalbuminuria, which are symptoms of chronic kidney disease (CKD). Microalbuminuria is twice as common in patients with 1-2 MS symptoms as in those without the syndrome. MS was associated with decreased eGFR and proteinuria. Studies showed that MS is associated with an increased risk of acquiring chronic renal disease by 1.4 to 2.5 times (and up to 4.4 times in men) over time.[7,8,24,25]

The complex etiology of the MS associated with renal failure remains unclear. Several hemodynamic factors, including hypertension, glomerular hyperfiltration, insulin resistance, adipokine dysregulation, and the accumulation of free fatty acids and triglycerides in glomerular and tubulointerstitial cells influence acute and chronic functional and structural renal injury. Additionally, abdominal obesity with increased intra-abdominal pressure and insulin resistance play a role.[24]

MS caused by excessive energy intake, may trigger inflammatory and oxidative pathways, potentially blocking major anabolic signaling pathways like

insulin/IGF, and diverting energy from synthetic pathways. In vitro studies show IL-6 inhibits IGF-1 signaling pathways. Adipose tissue expansion in metabolic syndrome leads to the release of inflammatory cytokines, such as TNF-α, IL-6, and CRP. An experimental study in metabolic syndromes animals showed that Inflammatory macrophages and TNF-α infiltrate abdominal and peri-renal fat tissue, potentially affecting kidney function. Weight loss improves inflammatory and anti-inflammatory markers, and anti-inflammatory treatments reduce systemic and renal inflammation.[25]

There are likely multiple factors involved in the MS's origin and progression of CKD. Substantial experimental and clinical data has shown that one of the most important roles played by chronic inflammation or oxidative stress amplification in the complex and interconnected pathways leading to CKD development. Patients with MS frequently have activation of the RAAS (Renin-Angiotensin-Aldosterone System) despite increased extracellular fluid volume and sodium retention. Numerous theories have been proposed to account for the RAAS's activation: (1) sympathetic activation; (2) visceral fat-induced adipokine production in the RAAS; and (3) hemodynamic changes (disturbance of renal blood flow).[26,27]

Obesity increases renal blood flow, GFR, glomerular pressure, and filtration fraction, leading to increased urinary albumin excretion and glomerulosclerotic damage. The first sign of renal injury is progressive proteinuria, which may precede GFR decline. Obesity-related glomerulopathy is a microscopically visible pathology. Features of renal biopsies that are more common in obese persons with normal renal function than in non-obese individuals include enlarged mesangial matrix, podocyte hypertrophy, mesangial cell proliferation, and glomerulomegaly. Obesity promotes modifications that, even in a nephron with normal capacity, are typical of the lower nephron number associated with CKD from other causes ultimately leading to renal failure. The histopathology showed that obesity causes a continuum of glomerular changes, from ultrastructural changes to clinical nephropathy and hyperfiltration-induced glomerulomegaly. This leads to mechanical stretching of podocytes, eventually resulting in focal and segmental glomerulosclerosis, or obesity-related glomerulopathy (ORG), causing proteinuria

and reduced GFR. Obesity activates the RAAS and sympathetic nervous system, leading to kidney dysfunction and hypertension. Obesity-induced CKD is triggered by increased free fatty acid levels, leading to lipotoxicity in renal tubular cells and inflammation. This leads to insulin resistance, endothelial dysfunction, and blood pressure elevation in individuals with abdominal obesity.[24,26]

Similar to this study, the study by Xie et al (2019) also found that MS was associated with CKD. An unadjusted analysis of the study showed that the components of MS, elevated triglycerides, elevated fasting blood glucose, and elevated waist circumference were associated with CKD, but reduced HDL cholesterol and hypertension grades were not associated with CKD. After adjusting the age and gender, the study showed that components that are elevated waist circumferences, hypertension grades, elevated triglycerides, and elevated fasting blood glucose were strongly associated with CKD but not with reduced HDL cholesterol. After adjustment, the association between high TG and elevated FBG and hypertension grades persisted even after additional adjustments for age, gender, antihypertensive medication, and statin therapy. This study also showed that triglycerides were independent of diabetes and hypertension grades associated with CKD. [28] There is evidence that high cholesterol contributes significantly to the decline of kidney function, either by hastening kidney artery atherosclerosis or by having deleterious effects on mesangial cells through lipids.[29] Dyslipidemia likely damages kidneys by accelerating the process of glomerulosclerosis. Renal insufficiency is linked to elevated blood pressure and hyperglycemia through oxidative stress, inflammation, vascular endothelial dysfunction, and advanced glycation end products. Dyslipidemia likely damages kidneys by accelerating the process of glomerulosclerosis.[8]

Supporting this study result, the study by Hu et al (2017) found that MS is associated with an increased risk for a mildly reduced eGFR in the middle-aged and elderly Chinese population. Participants with MS had a 1.29-fold increased odds ratio for mildly reduced eGFR compared to those without MS after adjusting for potential confounders.[30] Same as the Hu et al (2019) study found that the odds ratio

incidence of CKD and rapid decline of eGFR were significantly higher in MS than the non-MS group.[31]

The meta-analysis study by Rashidbeygi et al (2019) revealed that there was an association between MS, its components, and the risk of proteinuria or albuminuria, but there was no significant relationship between decreased HDL cholesterol with albuminuria. CKD is indicated by proteinuria and albuminuria. Proteinuria is brought on by either insufficient tubular reabsorption of the filtered proteins or a shift in glomerular permeability, which leads to elevated glomerular filtration of plasma proteins. The recent meta-analysis discovered that fat distribution is important in both albuminuria and proteinuria, with abdominal obesity—rather than overall obesity—being a risk factor for both conditions. Insulin resistance is caused by several factors, including raised glucagon levels, obesity-related inflammation, and increased glomerular hyperfiltration. Insulin resistance and inflammation are associated with endothelial dysfunction, reduced generation of endothelial nitric oxide, and worsening renal hemodynamic performance. These variables bring on hypertension, podocyte destruction, and proteinuria/albuminuria. Additionally, LDL cholesterol, hypertriglyceridemia, sodium retention, activation of the sympathetic nervous system, decreased levels of Na+ and K+ ATPase activity, and elevated GFR are associated with insulin resistance. These conditions can lead to compromised mitochondria and renal cell damage.[8]

CONCLUSION

Metabolic syndrome is a risk factor for the development of kidney dysfunction. Metabolic syndrome increases the incidences of CKD and proteinuria and decreases GFR. These findings suggest that it is important to screen for CKD in patients with metabolic syndrome.

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