Martín-Hurtado AJ M, et al. https://doi.org/10.37551/S2254-28842024029

Metabolic syndrome in the renal patient. Literature review

Antonio José Martín-Hurtado[1], Carmen Morales-Cuenca[1], Raquel Espejo-Rodriguez[1], Rodolfo Crespo-Montero[1,2,3].

[1] Department of Nursing, Faculty of Medicine and Nursing, Universidad de Córdoba, Córdoba, Spain
[2] Nephrology Department, Hospital Universitario Reina Sofía de Córdoba, Córdoba, Spain
[3] Instituto Maimónides de Investigación Biomédica de Córdoba, Córdoba, Spain

Please cite this article in press as:
Martín-Hurtado AJ, Morales-Cuenca C, Espejo-Rodriguez R, Crespo-Montero R. Síndrome metabólico en el paciente renal. Revisión bibliográfica. Enferm Nefrol. 2024;27(4):284-93

Corresponding author:
Antonio José Martín Hurtado
antoniojnene@gmail.com

Reception: 07-29-24
Acceptance: 09-10-24
Publication: 12-30-24

ABSTRACT

Introduction: In recent years, chronic kidney disease has increased in prevalence worldwide, and the comorbidities associated with its onset, on numerous occasions related to the metabolic syndrome, accentuate its development. The diseases that trigger metabolic syndrome have acquired a worrying epidemiological character in today's society.

Objectives: Review and synthesise the scientific literature on metabolic syndrome in chronic renal patients.

Methodology: A literature review was carried out with a systematised search using the PubMed and Scopus databases. Articles were included that referenced the different components of metabolic syndrome in patients with renal pathology and whose publication was between 2019 and 2024 in English and Spanish.

Results: 18 articles were selected, including systematic reviews and observational studies. After analysing these, the following variables emerged: the alterations that make up the metabolic syndrome and its influence on chronic kidney disease, the appearance of the metabolic syndrome and its prevalence, and prevention measures.

Conclusions: There is evidence of a high correlation between the presence of diseases in the metabolic syndrome and the development of kidney disease. Arterial hypertension and diabetes mellitus stand out above obesity and dyslipidaemia. Prevention and early diagnosis are considered the best treatments.

Keywords: metabolic syndrome; nephropathy; renal treatment; obesity; diabetes mellitus; diabetic.

RESUMEN

Síndrome metabólico en el paciente renal. Revisión bibliográfica

Introducción: En los últimos años, la enfermedad renal crónica ha aumentado su prevalencia mundial, y las comorbilidades asociadas a su aparición, en numerosas ocasiones relacionadas con el síndrome metabólico, acentúan su desarrollo. Las enfermedades desencadenantes del síndrome metabólico han adquirido un preocupante carácter epidemiológico en la sociedad actual.

Objetivos: Revisar y sintetizar la bibliografía científica en relación el síndrome metabólico en el paciente renal crónico.

Metodología: Se ha realizado una revisión bibliográfica con búsqueda sistematizada a través de las bases de datos PubMed y Scopus. Se incluyeron artículos que referenciaran los diferentes componentes del síndrome metabólico en pacientes con patología renal, y cuya publicación estuviese comprendida entre 2019 y 2024 en inglés y español.

Resultados: Se seleccionaron 18 artículos entre los que se encuentran revisiones sistemáticas y estudios observacionales. Tras el análisis de estos, emergieron las siguientes temáticas: las alteraciones que conforman el síndrome metabólico y su influencia en la enfermedad renal crónica, la aparición del síndrome metabólico y su prevalencia, así como las medidas de prevención.

Conclusiones: Se evidencia una elevada correlación entre la existencia de enfermedades incluidas en el síndrome metabólico y el desarrollo de enfermedad renal. Entre ellos la hipertensión arterial y la diabetes mellitus destacan por encima de la obesidad y las dislipemias. La prevención y el diagnóstico precoz son considerados los mejores tratamientos.

Palabras clave: síndrome metabólico; nefropatía; tratamiento renal; obesidad; diabetes mellitus; diabético.

INTRODUCTION

Over recent decades, the prevalence of chronic kidney disease (CKD) has increased worldwide, affecting approximately 10–13% of the population. In Spain, according to data from the Registro Español de Enfermos Renales collected in 2022, the estimated incidence is 150.0 per million population (pmp), corresponding to 368 pre-emptive kidney transplant patients, 1,169 peritoneal dialysis patients, and 5,582 haemodialysis patients (a total of 7,119 individuals with kidney disease). Among these patients, diabetes mellitus (DM) as an underlying cause has increased to 22.0%[1]. It is estimated that 128 per 100,000 inhabitants worldwide die from this condition[2]. In addition, comorbidities associated with its development, such as obesity and arterial hypertension, have acquired a worrying epidemic character, making CKD a major public health problem.

Pathogenesis associated with CKD development is associated with the presence of chronic glomerulonephritis, chronic pyelonephritis, abuse of non-steroidal anti-inflammatory drugs, autoimmune diseases, congenital malformations, prolonged acute kidney injury, kidney transplantation, and metabolic syndrome[3]. The literature describes its most severe symptoms as being associated with advanced stages; in its early stages, patients are asymptomatic or present with non-specific symptoms, with lethargy and pruritus being the most common[4].

The definition of metabolic syndrome (MetS) varies internationally; therefore, prevalence estimates may differ because they depend on the inclusion of different parameters, as MetS represents a construct of multiple pathophysiological processes. Nevertheless, one universal feature is recognised: a high proportion of the population is affected. It occurs in 35% of adults in the United States. MetS is characterised by the presence of multiple conditions, including obesity (OB), hypertension (HTN), diabetes mellitus (DM), and dyslipidaemia (including elevated triglyceride levels and low HDL cholesterol)[5].

Each disease comprising MetS represents an independent health risk; thus, in their various combinations, they consolidate a significant health threat due to their associated pathophysiological mechanisms. These include insulin resistance, production of reactive oxygen species (ROS) such as free radicals, the development of oxidative stress (OS), and chronic inflammation[6].

Insulin resistance is the most widely accepted hypothesis underlying MetS. As insulin action deteriorates, glycogen synthesis and glucose transport decrease, while hepatic lipogenesis persists. In response to intracellular glucose deficiency, lipolysis is stimulated. Lipid accumulation may result from increased delivery of fatty acids to tissues[5].

In adipose tissue, which has endocrine and paracrine functions, adipocytes undergo hypertrophy and hyperplasia, inducing a hypoxic state, which may promote necrosis with macrophage infiltration and the release of adipokines such as interleukin-6 (IL-6), an inflammatory cytokine that plays an important role in the pathogenesis of T2DM[5].

Epidemiological studies associate MetS and CKD with the development of nephrolithiasis[6-8]. The apparent link between these conditions is oxidative stress. These comorbidities cannot be clearly categorised as either causal or consequential, as no consensus exists regarding the sequence of their development, thereby establishing a bidirectional relationship[7].

MetS is closely related to the development of atherosclerotic cardiovascular disease due to chronic inflammation and/or vascular endothelial dysfunction, among other mechanisms[5]. Another condition associated with MetS is chronic liver disease, present in 1 in 4 adults[9].

Regarding risk factors, these include dietary habits and physical inactivity (most evidently related to obesity), smoking, socioeconomic factors, alcohol consumption, and the use of medications such as antiretrovirals or antipsychotics. Non-modifiable risk factors include family history, advanced age, postmenopausal status, and certain ethnic groups (Latin American populations). Early intervention directly targets modifiable risk factors[5].

For these reasons, the present review was undertaken with the primary objective of examining and synthesising the existing scientific literature on MetS and its relationship with CKD.

METHODOLOGY

Study Design

We conducted a literature review with a systematic search of health sciences databases following the criteria and standardised recommendations of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement[10].

Search Strategy

PubMed and Scopus were selected as the databases for the search. The MeSH terms used were: *"Metabolic Syndrome"*, *"Kidney Disease"*, *"Kidney Treatment"*, *"Obesity"*, *"Diabetes*

Mellitus", and "*Diabet*". The final search strategy combined Boolean operators OR and AND as follows: "Metabolic Syndrome" AND "Obesity" AND "Diabetes Mellitus" OR "Diabet" AND "Kidney diseases" OR "kidney treatment".

Eligibility Criteria

During the investigation, certain criteria were stipulated and applied to the study.

Inclusion criteria:

- Articles published between 2019 and 2024.
- Original articles addressing CKD and MetS.
- Articles in English, Spanish, or Portuguese.

Exclusion criteria:

- Articles requiring subscription for full-text access.
- Paediatric and adolescent populations.

Quality Assessment

Methodological quality of the selected articles was assessed using the STROBE checklist[11] for observational studies and the CASPe tool[12] for systematic reviews.

Data Extraction

Extracted variables included author, year, country, study type, sample, outcomes, and quality of evidence.

Results Synthesis

Eighteen articles meeting the established criteria were selected. A thematic-categorical analysis of results and variables was performed.

RESULTS

Search Results

Database searches were conducted between February and April 2024. The selection process is illustrated in a PRISMA flow diagram **(figure 1)**.

Initially, 1,589 articles were identified. After applying eligibility criteria, 1,324 were excluded, leaving 265 articles. Four duplicates were removed, resulting in 261. Following title and abstract screening, 159 articles were excluded, leaving 102 for full-text review. Eighty-four were excluded after full reading, yielding a final selection of 18 articles.

Characteristics of Included Studies

Of the 18 studies, 10 were observational studies, 5 were systematic reviews (including 1 meta-analysis), 2 were narrative reviews, and 1 was a clinical practice guideline.

Table 1 illustrates the main characteristics of the articles, including author, country, year, study type, sample, objective, results, and quality criteria.

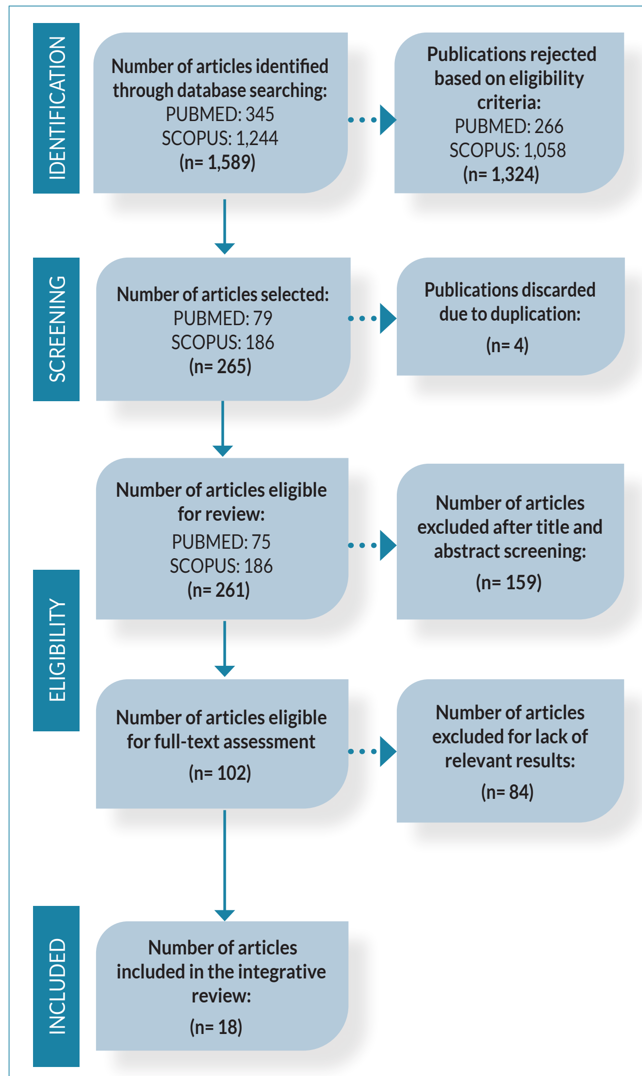

Figure 1. Search flowchart.

Analysis of variables

Prevalence

Bagasrawala et al. detected kidney disease in 1 in 5 patients studied (20.07%)[15]. Bikbov et al. estimated nearly 700 million individuals with CKD worldwide[18].

Park et al., in a population study of over 6 million participants, identified 16.38% with MetS; approximately 7% developed MetS and 5% recovered. Changes in MetS status were associated with altered CKD risk[21].

Fanaei et al. reported a MetS prevalence of 36.6%. During 18-year follow-up, 22.7% developed CKD. Incidence was 12.1/1,000/year in non-MetS individuals versus 27.4/1,000/year in MetS patients[27].

Martín-Hurtado AJ M, et al. https://doi.org/10.37551/S2254-28842024029

Comini et al. reported that among 788 individuals with HTN and/or DM, 65.4% had MetS, and among CKD patients, 75.2% had MetS[30].

Sociodemographic Variables

Most authors found no significant sex or age differences in MetS patients developing CKD[15-16]. However, conflicting findings exist.

Table 1. Characteristics of the studies included in the review.

AUTHOR, YEAR, COUNTRY	STUDY TYPE	SAMPLE	OBJECTIVE	RESULTS	QUALITY
Handelsman Y et al.; USA; 2023[13]	Clinical Practice Guideline.	Adults.	Development of recommendations for early diagnosis and management of comorbidities in patients with metabolic syndrome (MS).	Early intervention based on lifestyle modification, health education, and diet has been shown to reduce adverse outcomes and prolong life, whereas conventional treatment appears less effective and promotes sequential therapy, which is associated with adverse outcomes. Poor glycaemic control in diabetic patients increases the risk of developing nephropathy.	N/A
Martin-Moreno PL et al.; Spain; 2021[14]	Systematic review.	Kidney transplant recipients.	To investigate pre- and post-transplant obesity and post-transplant diabetes mellitus (PTDM).	Although controlling obesity could prevent CKD in 24.2% of men and 33.9% of women, pre- and post-transplant obesity does not demonstrate significant changes in renal function. However, increased physical activity in kidney transplant recipients is associated with a lower risk of PTDM.	CASPe (9/10)
Bagasrawala SI et al.; India; 2019[15]	Observational study.	568 obese Indian patients (BMI>33).	Prevalence of kidney disease in obese Indian population.	Metabolic syndrome is an independent risk factor for kidney disease. Among obese patients, only one fifth have kidney disease; however, associated comorbidities such as diabetes mellitus and hypertension play a more relevant role. Other parameters including age, weight, BMI, and body fat percentage were not statistically significant.	STROBE: (20/22)
Li H et al.; China; 2022[16]	Cross-sectional observational study.	570 CKD patients≥18 years, not on dialysis, from the Nephrology Department, Chinese PLA General Hospital Medical Centre.	To evaluate the ability to predict metabolic syndrome in CKD patients with and without T2DM.	The development of metabolic syndrome in obese individuals may be related to increased visceral adipose tissue, reduced subcutaneous tissue expansion, and metabolic changes. The visceral adiposity index stands out as a predictor of metabolic syndrome in CKD patients, particularly those with type 2 diabetes, outperforming other parameters such as BMI.	STROBE: (20/22)
Ammirati A.L.; Brazil; 2020[17]	Literature review.	CKD patients.	Compilation of strategies to reduce progression of CKD.	Strategies to slow CKD progression include blood pressure control, glycaemic control in diabetic patients, smoking cessation, and dietary management (especially protein intake restriction).	CASPE: (9/10)

Martín-Hurtado AJ M, et al. https://doi.org/10.37551/S2254-28842024029

AUTHOR, YEAR, COUNTRY	STUDY TYPE	SAMPLE	OBJECTIVE	RESULTS	QUALITY
Bikbov B et al.; United Kingdom; 2020[18]	Systematic review.	CKD patients.	To estimate the global burden of chronic kidney disease.	Worldwide prevalence of CKD was estimated at 697.5 million individuals, with 1.2 million deaths (projected to reach 2.2 million by 2040). Preventive efforts remain significantly lower than for other non-communicable diseases. Kidney health is closely linked to global health and prevention of diabetes, hypertension and obesity, although current outcomes remain suboptimal	CASPE: (10/10)
Fwu CW et al.; USA; 2024[19]	Cross-sectional observational study.	8,586 men and 8,420 women aged ≥20 years from the National Health and Nutrition Examination Survey.	To explore sex-specific and independent associations between non-diabetic metabolic syndrome, with and without obesity, and urinary incontinence (UI) and/or CKD.	Both male and female participants with obesity/metabolic syndrome had a higher incidence of UI or urgency UI.	STROBE: (20/22)
Varra FN et al.; Greece; 2024[20]	Systematic review.	Patients with OB.	To relate evidence linking obesity and chronic inflammation to the manifestation of metabolic dysfunctions.	Accumulated evidence indicates that obesity-related factors, including high-calorie diet, sedentary lifestyle, tumour microenvironment and dysregulation of the gut microbiota, exacerbate chronic tissue inflammation. Studies targeting obesity-associated inflammation show promising results regarding metabolic function and renal failure.	CASPE: (10/10)
Park S et al.; Korea; 2020[21]	Population-based observational study.	6,315,301 subjects: 4,537,869 without metabolic syndrome, 1,034,605 with chronic metabolic syndrome, 438,287 who developed metabolic syndrome and 304,540 who recovered from pre-existing metabolic syndrome	To study the relationship between reversal of metabolic syndrome and the probability of developing comorbidities	Development of metabolic syndrome was associated with a higher risk of CKD compared with those who never developed it. Recovery from metabolic syndrome was associated with lower CKD risk than persistent metabolic syndrome but higher than never having metabolic syndrome. Hypertension within metabolic syndrome was the strongest predictor of CKD.	STROBE: (22/22)
Sim R et al.; United Kingdom; 2022[22]	Systematic review and meta-analysis.	227,497 participants receiving different treatments: dopamine-2 agonists, DPP-4 inhibitors, thiazolidinediones, GLP-1 receptor agonists, insulin, SGLT2 inhibitors and sulfonylureas	To associate second-line antidiabetic therapy with renal (and cardiovascular) protection.	Analysis suggests that SGLT2 inhibitors, GLP-1 receptor agonists and sulfonylureas reduce the risk of renal outcomes. Patients treated with SGLT2 inhibitors had lower risk of acute kidney injury, serum creatinine doubling and slower decline in GFR.	CASPE: (10/10)

Martín-Hurtado AJ M, et al. https://doi.org/10.37551/S2254-28842024029

AUTHOR, YEAR, COUNTRY	STUDY TYPE	SAMPLE	OBJECTIVE	RESULTS	QUALITY
Schwartz P et al.; Argentina; 2023[23]	Narrative review	Patients with OB.	To examine the relationship between obesity and glomerular filtration rate (GFR).	Bariatric surgery has a direct impact on obesity, which constitutes a risk factor for end-stage renal disease. It converts patients with ESRD into candidates for kidney transplantation. Its main benefit is a significant reduction in CKD risk factors such as HTN and DM.	CASPE: (9/10)
Scurt FG et al.; Germany; 2024[24]	Systematic review.	Patients with kidney disease.	To analyse the association between metabolic syndrome and CKD.	The risk profile of metabolic syndrome is associated with hyperuricaemia, nephrolithiasis and oxalate nephropathy. Renal damage due to direct kidney injury is associated with DM and HTN. Lifestyle improvement and optimisation of metabolic syndrome risk factors are essential components of management.	CASPe: (10/10)
Petramala L et al.; Italy; 2024[25]	Cross-sectional observational study.	772 hypertensive patients (407 men; 365 women; mean age 52.2±15.1 years).	To compare classifications in a large hypertensive population.	Visceral OB and metabolic syndrome have a major impact on overall health. In addition to directly affecting renal function, they contribute to systemic complications, activating pathophysiological mechanisms related to oxidative stress and endothelial dysfunction, leading to severe histological damage and increased proteinuria.	STROBE: (21/22)
Xu Q et al.; China; 2024[26]	Cohort observational study.	5,225 individuals from rural China divided into four groups according to metabolic syndrome status (never, previous abnormalities, new-onset, persistent).	To identify the association between changes in metabolic syndrome status and accelerated decline in renal function.	New-onset metabolic syndrome had the greatest impact on renal function. Hyperuricaemia was suggested as a risk marker for decline in GFR.	STROBE: (16/22)
Fanaei SM et al.; Iran; 2023[27]	Cohort observational study.	8,987 participants grouped as: (1) free of MS, (2) MS (DM+ HTN−), (3) MS+ (DM− HTN+), (4) MS+ (DM+ HTN+), (5) MS+ (DM− HTN−).	To investigate the association between CKD and metabolic syndrome over 18 years of follow-up.	The subgroup with diabetes and hypertension at baseline had the highest risk of CKD. Age was also a significant risk factor for CKD development.	STROBE: (19/22)
Moreira AD et al.; Brazil; 2022[28]	Cross-sectional observational study.	14,636 adults from ELSA-Brasil.	To investigate the association between adiposity indices and kidney disease.	Individuals with OB in any form (waist-to-hip ratio, hip circumference, waist-to-height ratio) were more likely to develop albuminuria.	STROBE: (21/22)

AUTHOR, YEAR, COUNTRY	STUDY TYPE	SAMPLE	OBJECTIVE	RESULTS	QUALITY
Chen IJ et al.; Taiwan; 2021[29]	Cross-sectional observational study.	400 individuals (141 men, 259 women) aged 50–90 years living in northern Taiwan.	To analyse the association between OB indices and CKD, stratified by sex.	Findings suggest sex acts as a modifier of CKD. In women, a significant association was observed between the visceral adiposity index (VAI) and CKD.	STROBE: (18/22)
Comini L de O et al.; Brazil; 2020[30]	Cross-sectional observational study.	788 individuals aged ≥18 years diagnosed with hypertension and/or diabetes mellitus followed in primary health care services in Viçosa, Brazil	To examine the association between metabolic syndrome and CKD in hypertensive/diabetic population with peripheral arterial disease.	Prevalence of metabolic syndrome among CKD patients was 75.2%. Results indicate that metabolic syndrome increases the probability of CKD. Peripheral arterial disease plays a strategic role in monitoring hypertensive and diabetic patients.	STROBE: (21/22)

A 2020 study suggested that in countries with lower socioeconomic indices, HTN and DM prevalence is higher and access to renal replacement therapy is more limited. Women show higher prevalence in KDIGO stages 1–3, while men exhibit higher mortality, suggesting faster disease progression in men[18].

Fwu et al. (2024) reported a link between non-diabetic MetS, obesity, and urinary incontinence as a precursor to CKD; prevalence was 56% in women vs 16% in men[19].

Varra et al. showed that over the past 50 years, MetS prevalence increased markedly in women, with little change in men[20].

Two studies suggested age contributes to the cumulative manifestation of MetS components and acts as a trigger[27-30].

Metabolic Syndrome Alterations

HTN is a major determinant of CKD within MetS[25,28]. Combined with DM, it markedly increases CKD risk[13,22,23,29]. Fanaei et al. demonstrated strong association between HTN and DM with CKD; absence of both yielded similar CKD prevalence as non-MetS individuals, highlighting HTN as particularly significant[27]. Conversely, Li et al. linked DM and insulin resistance to increased MetS prevalence among CKD patients[16]. The role of OB remains controversial. Several studies associate obesity or related markers (BMI, chronic inflammation) with increased CKD risk[18,23,30]. Schwartz et al. showed bariatric surgery improves MetS components and reduces CKD and ESRD risk[23]. Other studies link obesity with albuminuria and proteinuria[23,28].

However, other authors found no significant association between obesity and CKD prevalence[15,27,29]. Fanaei et al. showed waist circumference and low HDL were not associated with CKD when controlling for age, sex, and risk factors[27].

Consistent with the above-mentioned study, Bagasrawala et al. reported limited relevance of dyslipidaemia, particularly in older women[15]. In contrast, 2 studies identified visceral adiposity index as a stronger predictor of MetS than HTN and DM[16] and among women regardless of body weight[29].

Finally, the role of MetS as a whole in the development of CKD should be highlighted. The study conducted by Comini et al. (2020) reported that 75.2% of participants diagnosed with CKD also presented MetS; however, no association was demonstrated between the number of MetS components and disease onset[30]. In agreement with this finding, Park et al.confirmed the MetS–CKD association and added that patients who had recovered from MetS had a higher risk of developing CKD in the future than those who had never had MetS. Unsurprisingly, patients with active MetS showed the highest risk[27].

Only one article offered a different perspective regarding the sequence of disease onset. In this case, the Spanish study by Martín-Moreno et al. addressed post-transplant diabetes mellitus (PTDM), reporting that patients undergoing kidney transplantation are at risk of developing DM during the recovery period[14].

The study by Park et al. stratified individuals according to the timing of MetS onset and suggested a lower glomerular filtration rate in individuals with persistent MetS during follow-up[21]. Conversely, the 2024 study by Xu Q et al., using the same classification criteria, proposed a lower glomerular filtration rate among patients with newly diagnosed MetS[26].

Preventive Measures

Detection, prevention, and optimisation of the different components of MetS constitute the most widely accepted strategy for CKD prevention[13,15-18,24-27,30]. Xu Q et al. further proposed serum uric acid as an independent predictor of

CKD, in addition to its established role in nephrolithiasis and gout[26].

Other authors advocate lifestyle modification, with particular emphasis on physical activity, dietary control, and cessation of smoking or other harmful substances[15,17,24,25]. Ammirati et al. specifically highlighted the importance of protein restriction in patients with CKD and recommended a multidisciplinary approach to manage related complications such as anaemia, mineral and bone disorders, and metabolic acidosis. They also proposed the establishment of a vaccination programme, with special attention to hepatitis B[17].

Only 3 authors suggested bariatric surgery as a therapeutic option to improve renal function and glomerular filtration rate, agreeing that it should be considered when conventional measures fail14,23,25

New-generation antidiabetic agents, such as SGLT-2 inhibitors and GLP-1 receptor agonists, were proposed by two authors[13,14]. Varra et al. further suggested improvement in renal function and insulin sensitivity with molecular-based therapy[20].

DISCUSSION

The objective of the present review was to examine the literature regarding the relationship between CKD and MetS, analysing their individual causes and reviewing approaches to prevention.

The results demonstrate a clear association between the pathologies leading to MetS and the development of kidney disease. Only one of the reviewed articles suggested a potential bidirectional relationship between these conditions, identifying post-transplant DM as a risk factor following kidney transplantation[14].

Most publications in this review identify HTN and/or DM as the principal MetS components associated with the development of CKD[13,22,23,29]. In agreement with these findings, a cross-sectional study conducted in Spain involving 2,659 patients associated the presence of HTN with increased risk of CKD[31]. Similarly, Yang H. et al. reported a strong association between HTN and CKD development and further linked MetS with hyperuricaemia[32].

Another Spanish study associated MetS components—obesity, HTN, hyperglycaemia, and dyslipidaemia—with vascular damage, which was proposed as a potential predictor of CKD[33].

Additional factors contributing to CKD risk include certain forms of dyslipidaemia such as visceral adiposity index and low HDL cholesterol, as well as obesity assessed using various metrics including BMI, waist–hip ratio, and waist–height ratio.

The role of OB remains controversial. Contrary to the preceding findings, several authors in this review considered obesity to be an insignificant factor in CKD development[15,27,29]. Kang SY et al. supported these conclusions, finding no difference in CKD progression risk or mortality between obese and non-obese individuals without MetS[34]. A large Korean study involving 130,504 participants confirmed the impact of MetS on CKD development but rejected BMI and waist circumference as independent determinants[35].

Conversely, other studies support OB as a relevant target for CKD prevention[18,23,30]. One Korean study associated obesity measured by waist circumference and waist-to-height ratio—but not BMI—with reduced renal function[36].Yang H. et al. likewise linked obesity to lower glomerular filtration rate and associated dyslipidaemia (elevated triglycerides) with CKD[32].

With respect to CKD prevention, most reviewed articles agree that prevention and control of MetS and its components constitute the primary preventive strategy[13,15,17,18,24-27,30]. Yang H. et al. further emphasised early intervention as a measure of substantial clinical value[32]. A Korean cross-sectional study that demonstrated a positive association between MetS presence, number of components, and increased risk of kidney cancer also advocated primary prevention of MetS components as treatment[37].

Consistent with earlier findings[15,24,25], 2 studies identified physical exercise as a renoprotective factor. One highlighted its antioxidant and anti-inflammatory effects, as well as its role in inducing autophagy[39]. Nataraj M et al. reported that exercise-based rehabilitation significantly improved renal function in patients with diabetes[40].

Study Limitations

This review presents several limitations. The main limitation is the exclusive use of the databases specified in the search strategy, which may have resulted in the exclusion of relevant studies indexed elsewhere.

Practical Considerations

Some lifestyle habits prevalent in modern society demonstrate low prioritisation of health preservation, which may partially explain the epidemic nature of MetS.

Nursing professionals play a critical role in the primary prevention of these conditions. Active engagement, implementation of screening programmes, and promotion of public awareness (including self-monitoring strategies) constitute powerful tools to reverse the rising incidence trend.

Only through modification of lifestyle behaviours and adoption of healthy habits can kidney disease be effectively prevented. Based on the findings of this review, a strong relationship exists between MetS progression and CKD development. Although the relative contribution of individual MetS components remains unclear, there is high consensus regarding the importance of HTN and DM. Obesity and

dyslipidaemia demonstrate inconsistent associations across studies and appear to represent weaker risk factors.

Among non-modifiable risk factors, age shows a direct association with CKD development, whereas sex appears to exert less influence.

Prevention of MetS-related conditions therefore constitutes prevention of CKD itself. Early diagnosis combined with promotion of healthy lifestyles represents a cornerstone of effective disease management.

Conflicts of interest

Las autoras y autores declaran no tener ningún conflicto de interés.

Funding

Los autores declaran no haber recibido financiación alguna.

REFERENCES

1. Registro Español de Enfermos Renales (REER). [cited 21 May 2024]. Available from: https://www.ont.es/informacion-a-los-profesionales-4/registros-de-donacion-de-trasplantes-4-9/registro-espanol-de-enfermos-renales-4-9-4/.

2. García-Trabanino R, Hernández C, Rosa A, Domínguez Alonso J. Incidencia, mortalidad y prevalencia de enfermedad renal crónica terminal en la región del Bajo Lempa, El Salvador: 10 años de registro comunitario. Nefrologia 2016;36:517-22.

3. Martínez-Castelao A, Górriz JL, Bover J, Segura-de la Morena J, Cebollada J, Escalada J, et al. Documento de consenso para la detección y manejo de la enfermedad renal crónica. Aten Primaria 2014;46:501-19.

4. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024;105(4S):S117-314.

5. McCracken E, Monaghan M, Sreenivasan S. Pathophysiology of the metabolic syndrome. Clin Dermatol. 2018; 36:14-20.

6. Khan SR. Is oxidative stress, a link between nephrolithiasis and obesity, hypertension, diabetes, chronic kidney disease, metabolic syndrome? Urol Res. 2012;40: 95-112.

7. Lei X, Wen H, Xu Z. Higher oxidative balance score is associated with lower kidney stone disease in US adults: a population-based cross-sectional study. World J Urol. 2024;42(1):222.

8. Kim Y-J, Park MS, Kim W-T, Yun S-J, Kim W-J, Lee S-C. Hypertension influences recurrent stone formation in nonobese stone formers. Urology. 2011;77:1059-63.

9. Muzica CM, Sfarti C, Trifan A, Zenovia S, Cuciureanu T, Nastasa R et al. Nonalcoholic fatty liver disease and type 2 diabetes mellitus: A bidirectional relationship. Can J Gastroenterol Hepatol. 2020;2020:6638306.

10. Hutton B, Catalá-López F, Moher D. La extensión de la declaración PRISMA para revisiones sistemáticas que incorporan metaanálisis en red: PRISMA-NMA. Med Clin. (Barc) 2016;147:262-6.

11. STROBE [Internet]. [cited 22 May 2024]. Checklists. Disponible en: https://www.strobe-statement.org/chec-klists/.

12. Santamaría R. Programa de Habilidades en Lectura Crítica Español (CASPe). NefroPlus. 2017;9(1):100-1.

13. Handelsman Y, Butler J, Bakris GL, DeFronzo RA, Fonarow GC, Green JB et al. Early intervention and intensive management of patients with diabetes, cardiorenal, and metabolic diseases. J Diabetes Complications. 2023;37:108389.

14. Martín-Moreno PL, Shin H-S, Chandraker A. Obesity and post-transplant diabetes mellitus in kidney transplantation. J Clin Med. 2021;10:2497.

15. Bagasrawala SI, Sheth H, Shah H, Ansari R, Lakdawala M. Metabolic syndrome rather than obesity alone is more significant for kidney disease. Obes Surg. 2019;29:3478-83.

16. Li H, Wang Q, Ke J, Lin W, Luo Y, Yao J et al. Optimal obesity- and lipid-related indices for predicting metabolic syndrome in chronic kidney disease patients with and without Type 2 diabetes mellitus in China. Nutrients. 2022;14:1334.

17. Ammirati AL. Chronic kidney disease. Rev Assoc Med Bras 2020;66:S3-9.

18. Bikbov B, Purcell CA, Levey AS, Smith M, Abdoli A, Abebe M et al. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395:709-33.

19. Fwu C-W, Schulman IH, Lawrence JM, Kimmel PL, Eggers P, Norton J et al. Association of obesity, metabolic syndrome, and diabetes with urinary incontinence and chronic kidney disease: Analysis of the national health and nutrition examination survey, 2003-2020. J Urol. 2024;211:124-33.

20. Varra F-N, Varras M, Varra V-K, Theodosis-Nobelos P. Molecular and pathophysiological relationship between obesity and chronic inflammation in the manifestation of metabolic dysfunctions and their inflammation-mediating treatment options (Review). Mol Med Rep. 2024;29(6):95.

21. Park S, Lee S, Kim Y, Lee Y, Kang MW, Han K et al. Reduced risk for chronic kidney disease after recovery from metabolic syndrome: A nationwide population-based study. Kidney Res Clin Pract. 2020;39:180-91.

22. Sim R, Chong CW, Loganadan NK, Fong AYY, Navaravong L, Hussein Z et al. Comparative effectiveness of cardiovascular, renal and safety outcomes of second-line antidiabetic drugs use in people with type 2 diabetes: A systematic review and network meta-analysis of randomised controlled trials. Diabet Med. 2022;39(3):e14780.

23. Schwartz P, Capotondo MM, Quaintenne M, Musso-Enz GM, Aroca-Martinez G, Musso CG. Obesity and glomerular filtration rate. Int Urol Nephrol. 2023;56:1663-8.

24. Scurt FG, Ganz MJ, Herzog C, Bose K, Mertens PR, Chatzikyrkou C. Association of metabolic syndrome and chronic kidney disease. Obes Rev. 2024;25(1):1-16.

25. Petramala L, Gigante A, Sarlo F, Servello A, Circosta F, Marino L et al. Relevance of obesity-related organ damage and metabolic syndrome classification in cardiovascular and renal risk stratification in patients with essential hypertension. Front Cardiovasc Med. 2024;28;11:1369090.

26. Xu Q, Fan X, Chen G, Ma J, Ye W, Ai S et al. New-onset metabolic syndrome is associated with accelerated renal function decline partially through elevated uric acid: an epidemiological cohort study. Front Endocrinol (Lausanne). 2024;15:1328404.

27. Fanaei SM, Mehran L, Amouzegar A, Masoumi S, Amouzegar A, Azizi F. The impact of metabolic syndrome on chronic kidney disease development. Insights from a big prospective study. Eur J Clin Invest. 2023;53(4):e13945.

28. Moreira AD, Velasquez-Melendez G, Ladeira RM, da Silva Junior GB, Fonseca M de J, Barreto SM. Association between adiposity indexes and kidney disease: Findings from the longitudinal study of adult health (Elsa-Brazil). J Am Nutr Assoc. 2022;41:275-80.

29. Chen I-J, Hsu L-T, Lu M-C, Chen Y-J, Tsou M-T, Chen J-Y. Gender differences in the association between obesity indices and chronic kidney disease among middle-aged and elderly Taiwanese population: A community-based cross-sectional study. Front Endocrinol (Lausanne). 2021; 12.

30. Comini L de O, de Oliveira LC, Borges LD, Dias HH, Batistelli CRS, da Silva LS et al. Individual and combined components of metabolic syndrome with chronic kidney disease in individuals with hypertension and/or diabetes mellitus accompanied by primary health care. Diabetes Metab Syndr Obes. 2020;13:71-80.

31. Crespo JJ, Piñeiro L, Otero A, Castiñeira C, Ríos MT, Regueiro A et al. Administration-time-dependent effects of hypertension treatment on ambulatory blood pressure in patients with chronic kidney disease. Chronobiol Int. 2013;30:159-75.

32. Yang H, Cui Z, Quan Z. Effects of metabolic syndrome and its components on chronic kidney disease and renal function: A two-sample Mendelian randomization study. Metab Syndr Relat Disord. 2024;22:114-22.

33. Rodríguez-Rodríguez R, Hornum M, Rodríguez Rodríguez AE, Bevc S, Trevisani F, Fernández G et al. Renal disease in metabolic syndrome: The hidden role of intrarenal ischemia. Kidney Int Rep. 2024;9:1419-28.

34. Kang SY, Lee YH, Jeong SJ, Kim JS, Jeong KH, Hwang HS. How obesity and metabolic syndrome affect cardiovascular events, progression to kidney failure and all-cause mortality in chronic kidney disease. Nephrol Dial Transplant. 2024;39:778-87.

35. Yang J, Lee HS, Lim CY, et al. Impact of obesity on renal function in elderly Korean adults: a national population-based cohort study. Kidney Research and Clinical Practice. 2024;j.krcp23.187.

36. Oh H, Quan SA, Jeong J-Y, Jang S-N, Lee JE, Kim D-H. Waist circumference, not body mass index, is associated with renal function decline in Korean population: Hallym aging study. PLoS One. 2013;8:e59071.

37. Wang L, Du H, Sheng C, Dai H, Chen K. Association between metabolic syndrome and kidney cancer risk: a prospective cohort study. Lipids Health Dis. 2024;17;23(1)142.

38. Perreault L, Pan Q, Aroda VR, Barrett-Connor E, Dabelea D, Dagogo-Jack S et al. Exploring residual risk for diabetes and microvascular disease in the Diabetes Prevention Program Outcomes Study (DPPOS). Diabet Med. 2017;34:1747-55.

39. Malheiro LFL, Fernandes MM, Oliveira CA, Barcelos I de S, Fernandes AJV, Silva BS et al. Renoprotective mechanisms of exercise training against acute and chronic renal diseases – A perspective based on experimental studies. Life Sci. 2024;346:122628.

40. Nataraj M, Maiya GA, Nagaraju SP, Shastry BA, Shivashankara KN, Shetty S et al. Effect of exercise-based rehabilitation on functional capacity and renal function in type 2 diabetes mellitus with nephropathy: a randomized controlled trial. Int Urol Nephrol. 2024;56(8):2671-82.

This is an open access article distributed under a Creative Commons licence.
https://creativecommons.org/licenses/by-nc/4.0/