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Renal complications in eating disorders with metabolic risk factors: a systematic review

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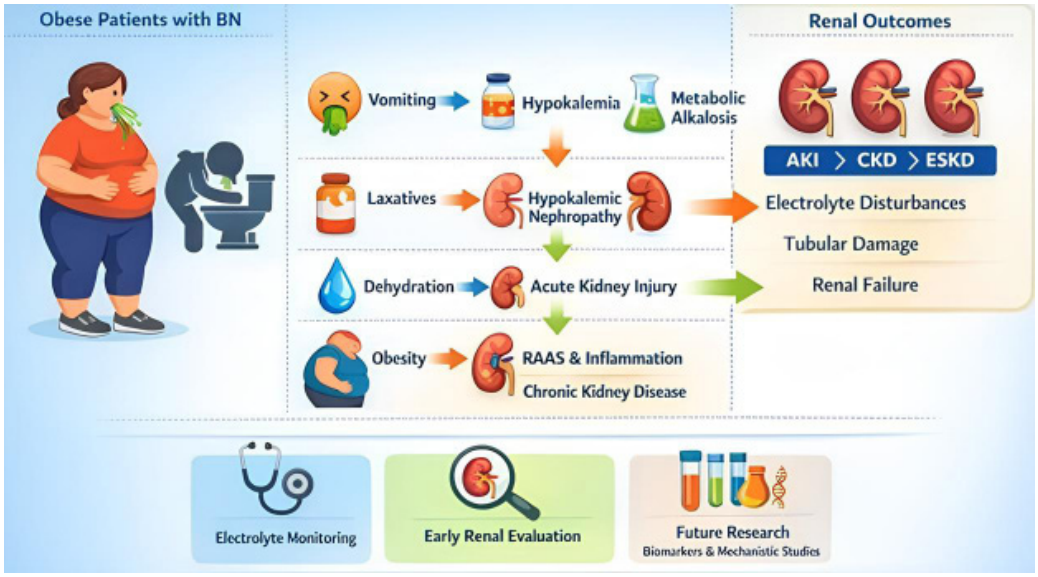
Highlights

- *Bulimia nervosa is associated with a spectrum of renal damage from electrolyte disturbances and AKI to CKD/ESKD.*
- *In the subgroup of BN in obese patients, the synergy of metabolic risks of obesity with purging could exacerbate kidney damage.*
- *The risk of chronic renal failure is exacerbated in obese patients with bulimia.*

Graphical Abstract



Abstract

Bulimia nervosa can have serious physiological and psychological effects in obese patients. Electrolyte disturbances, dehydration, and metabolic burden caused by bulimia nervosa can cause kidney injury. Furthermore, obesity, as an independent risk factor for kidney disease, may exacerbate renal complications in this population. The aim of the present systematic review was to evaluate renal disorders in patients with bulimia nervosa, with particular focus on obese individuals. A total of ten studies (including case reports and observational studies) published between 2001 and 2024 in PubMed, Scopus, Web of Science, and Cochrane Library were included. The results suggest that the most common pathway of renal injury in bulimia nervosa is chronic hypokalemia and dehydration, which may lead to hypokalemic nephropathy, tubulointerstitial nephritis, and acute or chronic kidney injury. Electrolyte and acid-base disturbances, along with decreased renal function, are frequent, and the risk of chronic kidney disease may be higher in obese patients. Limitations of the available evidence include the predominance of case reports and small observational studies, which restrict generalizability and preclude definitive causal conclusions. These findings highlight the need for cautious clinical monitoring, early renal assessment, and consideration of obesity as a compounding factor in managing patients with bulimia nervosa.

Keywords: Bulimia Nervosa. Renal Disorders. Obese Patients. Electrolytes. Renal Failure.

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INTRODUCTION

Obesity, as one of the significant health problems over the past few decades, is associated with an increased prevalence of chronic diseases as well as a wide range of physical and psychological consequences[1,2]. One of the less-considered aspects in this field is the occurrence of eating disorders, including bulimia nervosa, in obese patients, which can have significant effects on the individual's overall health[3]. Bulimia nervosa is characterized by unhealthy eating patterns, excessive overeating, and compensatory behaviors such as self-induced vomiting, which causes significant physiological and metabolic changes[4]. Among vital organs, the kidneys are most vulnerable to the consequences of this disorder, as electrolyte changes, dehydration, and metabolic overload can lead to severe renal dysfunction[5,6]. On the other hand, the presence of obesity as an independent risk factor for kidney disease can exacerbate the effects of bulimia nervosa and predispose to kidney failure, urinary stones, and impaired filtration function[7,8]. This is especially important when patients with obesity and bulimia nervosa simultaneously face intersecting psychological and physiological consequences, which complicate their diagnosis and treatment.

Given the potentially serious consequences on public health as well as the economic and social burden resulting from the treatment of kidney diseases, a comprehensive and systematic review of the available evidence on the association between kidney disorders and bulimia nervosa in obese patients is necessary. present systematic review can identify clinical patterns, risk factors, and associated outcomes, and provide valuable guidance for designing preventive and therapeutic interventions in the fields of medicine, nursing, and healthcare. Despite the growing evidence linking eating disorders to renal dysfunction, there is still a lack of systematic synthesis focusing specifically on obese patients with bulimia nervosa. Present systematic review was designed according to a PICO framework to address the following question: in individuals with bulimia nervosa and obesity (P), how does the presence of metabolic risk factors (I/E), compared with individuals without obesity or metabolic risk factors (C), influence the occurrence and type of renal complications (O)?; therefore this review aims to summarize current evidence, identify mechanisms of renal injury, and highlight critical gaps for future research.

METHOD

Information sources and search strategy

Present study was a systematic review that was designed and conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines[9].

PubMed, Scopus, Web of Science, and Cochrane Library were among the international databases that were comprehensively searched. A combination of MeSH terms and keywords related to obesity, bulimia nervosa, renal disorders, renal disease, and eating disorders was used as search keywords.

The search strategy included the following MeSh terms: ((((((((((("Bulimia Nervosa"[Mesh]) OR ("Bulimia Nervosa/classification"[Mesh] OR "Bulimia Nervosa/complications"[Mesh] OR "Bulimia Nervosa/diagnosis"[Mesh] OR "Bulimia Nervosa/diagnostic imaging"[Mesh] OR "Bulimia Nervosa/diet therapy"[Mesh] OR "Bulimia Nervosa/drug therapy"[Mesh] OR "Bulimia Nervosa/epidemiology"[Mesh] OR "Bulimia Nervosa/etiology"[Mesh] OR "Bulimia Nervosa/history"[Mesh] OR "Bulimia Nervosa/metabolism"[Mesh] OR "Bulimia Nervosa/mortality"[Mesh] OR "Bulimia Nervosa/pathology"[Mesh] OR "Bulimia Nervosa/prevention and control"[Mesh] OR "Bulimia Nervosa/psychology"[Mesh] OR "Bulimia Nervosa/surgery"[Mesh] OR "Bulimia Nervosa/therapy"[Mesh])) OR "Feeding and Eating Disorders"[Mesh]) AND ("Obesity"[Mesh] OR "Obesity Management"[Mesh])) OR ("Obesity/diagnosis"[Mesh] OR "Obesity/diagnostic imaging"[Mesh] OR "Obesity/diet therapy"[Mesh] OR "Obesity/drug therapy"[Mesh] OR "Obesity/prevention and control"[Mesh] OR "Obesity/rehabilitation"[Mesh] OR "Obesity/surgery"[Mesh] OR "Obesity/therapy"[Mesh])) OR "Overweight"[Mesh]) OR "Cardiometabolic Risk Factors"[Mesh]) AND "Renal Agents"[Mesh]) OR "Kidney Diseases"[Mesh]) OR "Kidney Diseases/complications"[Mesh]) OR "Acute Kidney Injury"[Mesh]) OR "Acute Kidney Injury/complications"[Mesh]) OR "Renal Insufficiency, Chronic"[Mesh]) OR "Renal Insufficiency, Chronic/complications"[Mesh].

All databases were searched from 2000 to June 2024. Only articles published in English were considered eligible.

Inclusion and exclusion criteria

Eligible studies included original research articles that investigated renal complications in patients with eating disorders, including bulimia nervosa and related eating disorder subtypes, with or without the presence of obesity or other metabolic risk factors. Studies were required to report at least one renal outcome, such as acute kidney injury, chronic kidney disease, electrolyte disturbances, nephrolithiasis, or impaired renal function. Only studies published in English with full-text availability were included. Review articles, conference abstracts, editorials, letters to the editor, animal studies, and studies that did not report renal outcomes were excluded. Case reports were excluded from quantitative synthesis; however, due to the rarity of certain renal complications, relevant case reports were included in the qualitative synthesis.

Study selection process

In the first step, all retrieved records were entered into EndNote software, and duplicate studies were removed. Then, two researchers independently reviewed the titles and abstracts, and eligible articles were selected for full-text review; a third researcher resolved any disagreements.

Data extraction and quality assessment

Key information, including author names, year of publication, country of study, sample size, patient characteristics, type of renal disorder reported, and main results, was extracted. For this purpose, a standard data extraction form was designed and used.

The methodological quality of the included observational studies was assessed using the Newcastle–Ottawa Scale (NOS). This scale evaluates the risk of bias in non-randomized studies across three main domains: selection of study groups, comparability of groups, and assessment of outcomes. The total NOS score ranges from 0 to 9 points, with higher scores indicating better methodological quality and a lower risk of bias. ased on the total NOS score, studies were categorized as having low methodological quality and high risk of bias (0–3 points), moderate quality and moderate risk of bias (4–6 points), or high methodological quality and low risk of bias (7–9 points).

The methodological quality of included case reports was assessed using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Case Reports. This checklist is a validated tool specifically designed to evaluate the quality and risk of bias in case report studies. It consists of 8 key items assessing the clarity and completeness of clinical information, including patient demographics, clinical history, diagnostic assessment, intervention details, post-intervention outcomes, and the presence of clear takeaway lessons. Each item was rated as "Yes" (1 point), "No" (0 points), "Unclear" (0 points), or "Not applicable", resulting in a total possible score ranging from 0 to 8 points, with higher scores indicating better methodological quality. Case reports scoring 6–8 points were considered to have high methodological quality and low risk of bias, scores of 3–5 points indicated moderate quality, and scores of 0–2 points were classified as low quality and high risk of bias.

For included review studies, methodological quality was assessed descriptively rather than quantitatively. Instead, reviews were evaluated based on the clarity of objectives, comprehensiveness of literature coverage, transparency of data synthesis, and discussion of potential bias. Their quality is presented as a validity assessment, without assigning numerical scores.

Quality assessment was performed independently by two reviewers, and discrepancies were resolved through discussion.

RESULTS

The search identified ten primary studies investigating renal outcomes in patients with eating disorders, including bulimia nervosa (BN) and anorexia nervosa (AN), with or without obesity. These studies included case-control, cross-sectional, cohort, and case report designs, published until 2024 (Figure 1). A review of these studies revealed that the findings could be categorized into three main areas: Electrolyte and acid-base disorders, Acute and chronic kidney failure, and Association with metabolic diseases and risk factors (Table 1).

According to the present systematic review, the most common route of kidney damage in bulimia nervosa is chronic hypokalemia due to purging (induced vomiting/diuretic and malin abuse) and dehydration, leading to hypokalemic nephropathy, tubulointerstitial nephritis (TIN), acute kidney injury (AKI), and ultimately chronic kidney disease (CKD)[6]. Hyponatremia and hypokalemic metabolic alkalosis have also been reported in most studies. On the other hand, studies suggest that long-term hypokalemia can be associated with biopsychological changes including vacuolar tubulopathy and TIN, and in chronic bulimia nervosa patients, it can even lead to "end-stage renal disease/ESKD[10].

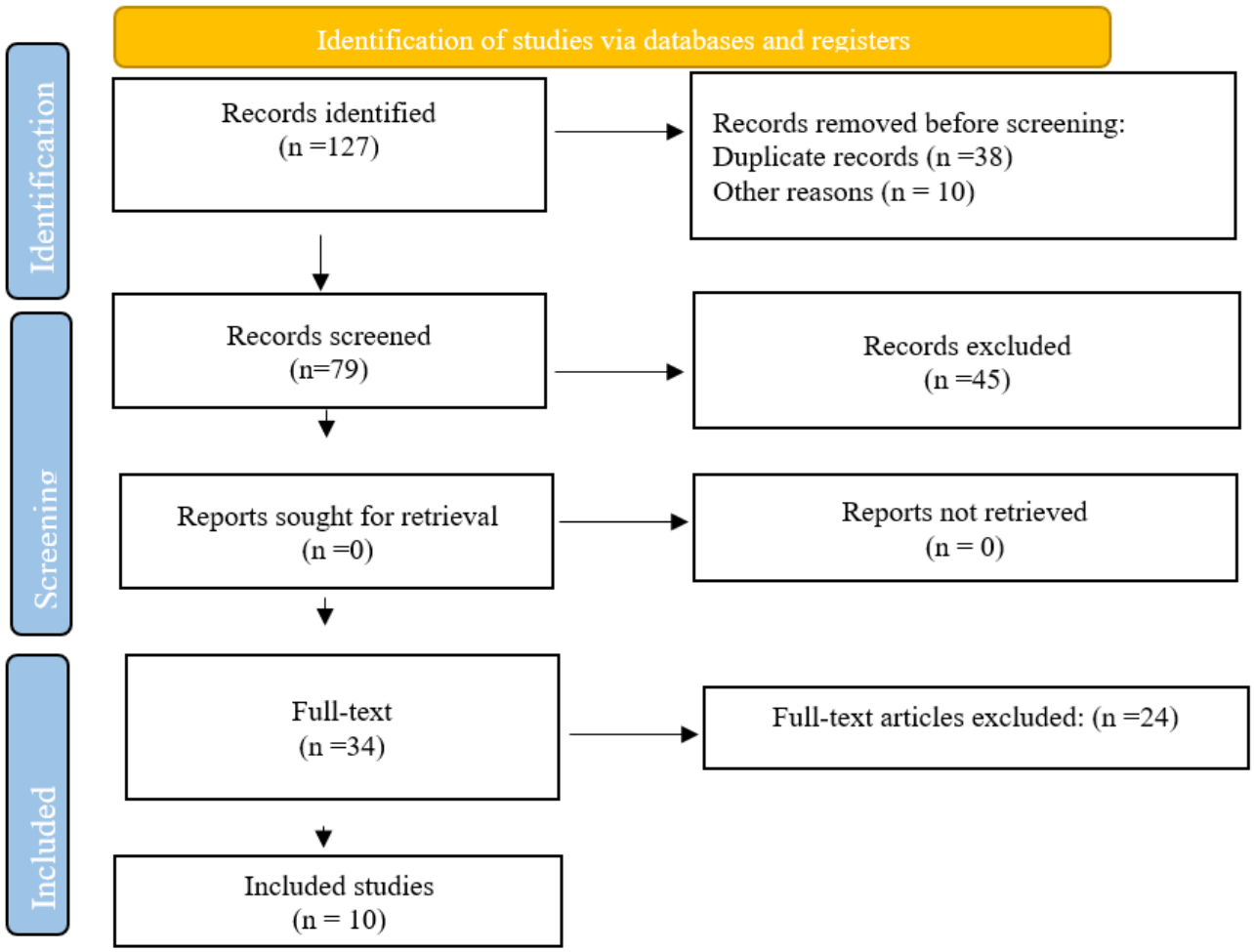

Figure 1 - PRISMA 2020 Flow Diagram.

On the other hand, tubulointerstitial patterns resulting from chronic hypokalemia have been reported in kidney biopsies; therefore, the importance of early diagnosis and cessation of purging behaviors is of great importance. Studies have shown that hypokalemia, hyponatremia, and acid-base disturbances are common in BN; severe hypokalemia can lead to cardiac arrhythmias and, at the renal level, hypokalemic nephropathy/TIN. Therefore, edema and rapid weight gain are observed after discontinuation of purging, requiring "prudent fluid/electrolyte management."

Electrolyte and acid-base disorders

In most cases and cross-sectional reports, patients with bulimia who engaged in repeated purging behaviors (intentional vomiting or use of laxatives and diuretics) developed hypochloremia, hypokalemia, and metabolic alkalosis. This condition has been identified as the most important predisposing factor for kidney damage[5,11,12]. In patients with BN, repeated purging behaviors (vomiting, laxatives, diuretics) led to hypokalemia, hypochloremia, and metabolic alkalosis, which were identified as the main predisposing factors for renal injury.

Acute and chronic kidney failure

Several case studies have reported the occurrence of acute renal failure in patients with bulimia nervosa and obesity, often due to severe dehydration and electrolyte imbalance. Some patients recovered after treatment and correction of fluids and electrolytes, but in several cases, renal failure progressed to chronic kidney disease (CKD)[13,14,15].

Association with metabolic diseases and risk factor

According to the reviewed studies, the coexistence of obesity and bulimia is directly related to increased blood pressure, type 2 diabetes, and metabolic syndrome. Also, having underlying diseases is considered an independent risk factor for kidney disease and can negatively affect kidney function[11,12]. Obesity in combination with BN was associated with higher prevalence of hypertension, type 2 diabetes, and metabolic syndrome. Underlying comorbidities were identified as independent risk factors for kidney disease and contributed to worse renal outcomes.

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Table 1 - Patient demographics and renal outcomes.

Author (Year)	Study Design	Sample size	Mean BMI (kg/m²)	Population / Focus	Disorder / Obesity Status	Renal Out-come Re-ported	Quality Assessment Tool	Score / Risk of Bias
Gurevich *et al.* (2021)[6]	Case–control	1	28.5	Adolescents with anorex-ia nervosa (AN)	BN / Obese	36.8% had impaired kidney func-tion (eGFR < 90 mL/min/1.73 m²)	NOS	7 (Low risk)
Burlaka & Mityuryayeva (2024)[15]	Case report	1	14	14-year-old with eating disorder (ED)	BN / Obese	Acute kid-ney injury requiring ICU (AKI)	JBI	7 (High quality)
Puckett (2023)[5]	Review (in--cludes data)	2	NR	Eating disor-ders, esp. BN + AN-BP	BN / Mixed	Hypokalemic nephropathy, CKD from purging be-ha-viors	Narrative assessment	Descriptive only
Miyahara *et al.* (2024)[10]	Retrospective cohort	1	NR	Patients with AN (2005–2023)	AN-BP / Obese	Decreased creati-nine-eGFR, corre--lation with FT3 and dehydration markers	NOS	8 (Low risk)
Stheneur *et al.* (2024)[13]	Multi-center cross-sec-tional	45	17.8	Hospitalized AN patients	AN / Non--obese	62% had eGFR of 60–90, 17.8% had 45–60, 2% <45; low BMI predicts renal im-pairment	NOS	7 (Low risk)
Yasuhara *et al.* (2005)[14]	Case report	120	16.5	Longstand-ing BN pa-tient	AN / Non--obese	End-stage kidney dis-ease (ESKD) due to hypokalemic nephropathy	JBI	6 (Moder-ate quality)
Arora *et al.* (2012)[16]	Case report (2 cases)	68	17.2	ED patients with ARB and purging	AN / Non--obese	ARB-induced acute kidney injury (AKI)	JBI	7 (High quality)
Khatri *et al.* (2023)[12]	Case report	45	17.8	AN-BP pa-tient with ESRD on hemodialysis	AN / Non--obese	ESRD likely from hypokalemic nephropathy se-condary to laxative abuse	JBI	6 (Moder-ate quality)
Miyahara *et al.* (2024)[10]	Retrospective cohort (same as above)	39	13	AN patients	Mixed	(same as above)	NOS	8 (Low risk)
Karger (2011)[11]	Pathophysio-log-ic review	NR	NR	ED including BN	Mixed	Binge/purge beha-vior impairs renal function even shor-t-term	Narrative assessment	Descriptive only

Quality assessment

Overall, the majority of included studies demonstrated moderate to high methodological quality, supporting the reliability of the synthesized evidence on renal complications in patients with eating disorders and metabolic risk factors (Table 2).

A mechanistic summary of renal complications in patients with eating disorders is presented in Table 1. The main pathways of kidney injury include chronic hypokalemia due to purging behaviors (vomiting, laxative/diuretic abuse), dehydration leading to acute kidney injury, and obesity-related metabolic inflammation contributing to chronic kidney disease. This table highlights the interaction between type of eating disorder, obesity status, and renal outcomes, providing a clear overview of the physiologic mechanisms underlying kidney injury in this population.

Table 2 - Mechanistic Summary Table – Renal Complications in Eating Disorders.

Mechanism	Disorder	Obesity Status	Renal effect	Supporting Studies
Vomiting / Purg-ing	BN	Obese	Hypokalemia → Metabolic alkalosis → Hypokalemic nephropathy	Burlaka & Mityuryayeva (2024)[15], Yasuhara *et al.* (2005)[14], Arora *et al.* (2012)[16]
Laxative / Diuret-ic abuse	BN / AN-BP	Obese / Non-obese	Hypokalemic nephropa-thy, Tubulointerstitial nephritis (TIN), AKI	Khatri *et al.* (2023)[12], Mi-yahara *et al.* (2024)[10]
Dehydration / Volume depletion	BN / AN	Obese / Non-obese	Acute kidney injury (AKI), decreased eGFR	Miyahara *et al.* (2024)[10], Stheneur *et al.* (2024)[13]
Chronic hypoka-lemia	BN	Obese	Chronic kidney disease (CKD), End-stage renal disease (ESRD)	Yasuhara *et al.* (2005)[14], Puckett (2023)[5]
Obesity-related inflammation	BN / AN	Obese	CKD, hypertension, met-abolic syndrome, nephropathy	Stheneur *et al.* (2024)[13], Puckett (2023)[5]
Drug-induced nephrotoxicity (ARB)	BN / ED	Mixed	Acute kidney injury (AKI)	Arora *et al.* (2012)[16]

DISCUSSION

Based on the findings of the present systematic review, in obese patients with bulimia nervosa, behaviors such as self-induced vomiting and use of laxatives can lead to chronic hypokalemia, metabolic alkalosis, and dehydration, which can damage the kidneys and progress from AKI to CKD and ESKD[12,14,17]. The study showed that long-term BN leads to ESKD. This pattern has also been observed in recent reports that chronic laxative abuse ultimately leads to ESKD[14], emphasizing the role of long-term electrolyte pathology in the development of end-stage renal failure[18]. Studies have shown that in adolescents hospitalized with anorexia nervosa, 36.8% were reported to have impaired renal function (eGFR <90)[6]; and in cohorts/series of anorexia nervosa patients, a decline in eGFR and its association with markers of dehydration and hormonal status have been reported[10]. A multicenter study of 2024 also showed that low BMI is an independent predictor of renal dysfunction in hospitalized anorexia nervosa patients[13]. Although these are not directly bulimia nervosa, they share physiological mechanisms (dehydration, electrolyte pathology, tubulopathy) with bulimia nervosa, suggesting that "eating disorders" generally provide the basis for kidney damage.

Studies have shown that the interaction of bulimia nervosa with obesity can enhance the risk of CKD. Obesity increases the risk of CKD through pathways such as hyperactivity of the renin-angiotensin system, adipokines-mediated inflammation, and changes in glomerular hemodynamics[12,19]. Several reports have shown that medications such as angiotensin receptor blockers can precipitate or exacerbate AKI in the setting of dehydration/purging[20]. The combination of purging-induced dehydration, chronic hypokalemia, and hyperactivation of the renin-angiotensin-aldosterone system (RAAS) may synergistically accelerate kidney injury, highlighting the complex pathophysiology in obese BN patients. Obesity contributes to renal injury not only via hemodynamic changes but also through inflammatory mechanisms, including adipokine dysregulation, insulin resistance, and oxidative stress, which can exacerbate kidney damage in patients with bulimia nervosa. These findings should be taken seriously in the clinical care of bulimia nervosa patients with obesity and hypertension.

Despite the overall convergence of evidence, standardization of renal outcomes is still inadequate. Creatinine-based eGFR can be misleading in patients with severe changes in muscle mass; more recent studies have emphasized the use of cystatin C and composite indices to more accurately estimate renal function[10,13].

Important research gaps related to the aim of the present study were the lack of BN-specific cohorts (especially in the obese subgroup) with adequate sample size and long-term follow-up, the absence of standardized renal outcomes, inadequate control of confounders, underreporting of laxative/diuretic abuse, and uncertainty in estimating the dose-response of renal injury. Bulimia nervosa is often underdiagnosed in obese individuals, which can lead to misclassification in studies and underestimation of renal risk in this population. It is important to distinguish between reversible functional kidney injury, which may recover with fluid/electrolyte correction, and irreversible structural nephropathy, such as hypokalemic nephropathy and tubulointerstitial fibrosis, which can progress to CKD or ESKD[21,22,23].

The present study had limitations, including design heterogeneity, publication bias, predominant reliance on eGFR creatinine, insufficient information on purging intensity/duration and actual laxative dose, and limited generalizability to the "bulimia nervosa + obesity" subgroup.

CONCLUSION

Bulimia nervosa is associated with a spectrum of renal damage from electrolyte disturbances and AKI to CKD/ESKD. Available evidence emphasizes the importance of early identification, electrolyte monitoring, and multidisciplinary intervention. In the subgroup of BN in obese patients, the synergy of metabolic risks of obesity with purging could exacerbate kidney damage. Direct clinical implications include routine electrolyte monitoring and early renal evaluation. From a public policy perspective, integration between nephrology and psychiatry is recommended to improve patient outcomes. Proposals for future research include prospective cohort studies, investigation of novel biomarkers such as cystatin C and NGAL, and mechanistic studies to elucidate pathways of renal injury. Still, dedicated longitudinal studies are needed to quantify this risk accurately.

CRediT author statement

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All authors have read and agreed to the published version of the manuscript.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the workreported in this paper.

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