



Association Between Gallstone Disease and Kidney Stone Disease: A Systematic Review and Meta-analysis

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Abstract

Background and aims: Gallstone disease (GSD) and kidney stone disease (KSD) have increased due to lifestyle in recent decades. This systematic review and meta-analysis aimed to investigate the association between these two diseases.

Methods: A comprehensive electronic database search was conducted before August 25, 2024. This systematic review and meta-analysis included observational studies. The meta-analysis employed a random-effects model to compute the overall summary estimates of the association between GSD and KSD using risk ratios with 95% confidence intervals (CIs) as the primary measure of the effect size. Heterogeneity was evaluated using chi-square tests, the I^2 statistic, and forest plots. Publication bias was assessed through Begg's and Egger's tests. A P value of less than 0.05 was considered statistically significant, and all analyses were performed using Stata 17 software.

Results: The meta-analysis included 9 studies encompassing 982 847 participants. The pooled analysis revealed a statistically significant association between GSD and KSD, with a risk of 1.78 (95% CI: 1.572.03, $P \leq 0.001$). Begg's and Egger's tests demonstrated no significant bias (Begg's test $P = 0.835$, Egger's test $P = 0.812$). Variables such as study year, sample size, mean age of participants, mean follow-up, and study quality as determined by the Newcastle-Ottawa Scale (NOS) were examined, but none could significantly impact heterogeneity ($P > 0.10$).

Conclusion: This systematic review and meta-analysis provide evidence of a significant association between GSD and KSD. Therefore, further investigation into the underlying mechanisms and potential risk factors is necessary.

Keywords: Kidney stone, Kidney calculi, Cholelithiasis, Gallstone disease

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Received: September 11, 2024

Accepted: October 22, 2024

ePublished: November 12, 2024



Introduction

Kidney stone disease (KSD) is increasing among the global population.¹ In addition to imposing a heavy financial burden on society and the healthcare system, this disease also disrupts people's quality of life.^{2,3} Pain, hematuria, urinary tract obstruction, and upper urinary tract infection are the most important symptoms of this disease that disrupt a person's daily life.⁴ KSD is a multifactorial disease in which several factors, such as unhealthy lifestyle (sedentary lifestyle and bad nutritional habits), age, gender, race, higher body mass index, ethnicity, family history of kidney stones, occupation, climate, geography of the place of residence, systemic diseases, diabetes, vascular disease, and chronic kidney disease, are involved in the risk of its occurrence.^{1,5-7} However, some mechanisms involved in the occurrence of KSD have yet to be identified, and this requires in-depth investigations that can play an essential

role in formulating prevention and treatment strategies.⁸

Gallstone disease (GSD) is another disease that has an increasing prevalence due to unhealthy diet, inactivity, and overweight.⁹ Approximately 6% of the world population is affected by GSD.¹⁰ In the United States, the prevalence of the disease has doubled over the past three decades, potentially due to the worsening of metabolic risk factors and the increased use of laparoscopic cholecystectomy.¹¹ Despite the majority of individuals with GSD having asymptomatic gallbladder stones, it can lead to severe pain in the right upper abdominal, nausea, vomiting, fever, chills, choledocholithiasis, cholangitis, cholecystitis, pancreatitis, and invasive surgery in affected patients.^{12,13}

Few studies have investigated this relationship. Therefore, a meta-analysis study evaluated the relationship between KSD with diabetes mellitus, hypertension, and GSD and reported that KSD was significantly

associated with increased risks of developing GSD, and there was a bidirectional relationship between KSD and GSD.¹⁴ Another review study demonstrated that GSD is independently associated with an increased risk of KSD.¹⁵

Although the pathogenesis of KSD is multifactorial, identifying the primary risk factors and comorbidities is crucial for understanding disease prevention and enhancing patient care.¹⁶ Considering the various complications of kidney and gallstones, it is necessary to fully understand the underlying factors in the occurrence of the disease to develop our knowledge of the multiple dimensions of these diseases. Considering that, to the best of our knowledge, no independent and comprehensive study has so far investigated the relationship between GSD and KSD, this study seeks to explore this association.

Materials and Methods

Database Selection, Search Terms, and Search Strategy

On August 25, 2024, several databases, including PubMed/MEDLINE, Scopus, Web of Science, and Embase, were systematically searched to identify studies relevant to our research objectives. The search strategy was developed using a combination of medical subject headings and frequently utilized keywords from previous literature. A comprehensive search was conducted using the search terms in [Supplementary file 1](#). The search process for this systematic review involved a rigorous examination of pertinent studies and prior review articles, with iterative refinement of the search queries to ensure exhaustive screening of all relevant publications. EndNote version 21.0.1 (released July 25, 2023, by Thomson Reuters) was employed to manage references and remove duplicate entries to maintain the integrity of the dataset.

Inclusion and Exclusion Criteria

Observational studies that adhered to specific criteria based on the **PECO framework** (population, exposure, control, and outcomes) were included in this study. The selected population comprised patients diagnosed with both GD and KSD. The exposure involved the presence of GSD and KSD, while the control group consisted of patients without either condition. Studies that did not conform to these criteria were excluded, including review articles, case series, case reports, abstract-only publications, conference poster presentations, unpublished study protocols, letters to the editor, in vivo and in vitro studies, and studies published in languages other than English.

All eligible publications' full texts were independently retrieved and reviewed to ensure thorough evaluation. In cases where discrepancies arose during the review process, these were resolved through discussion with a third team member to achieve a consensus. This rigorous screening process was implemented to maintain the accuracy and reliability of our study selection.

Screening Process and Full-Text Assessment

Two investigators independently screened the titles and

abstracts of the publications according to predefined inclusion and exclusion criteria. Studies that appeared to meet the inclusion criteria were identified, and their full-text articles were obtained for further evaluation. The same investigators then reviewed these full-text articles independently to confirm their eligibility. In cases where disagreements occurred, a third reviewer was consulted to resolve the discrepancies through discussion.

Data Extraction

Two investigators independently extracted data from the selected studies using the predefined eligibility criteria. Discussions and consultation with a third team member resolved any disagreements regarding study selection. Essential information, including the first author's name, publication year, study country, sample size, number of cases and controls, mean age, gender, mean body mass index, current smoking and alcohol consumption status, and presence of hypertension, was collected from each study. The other extracted data included follow-up duration and statistical data such as risk ratios (RR) or hazard ratios (HR) with 95% confidence intervals (CIs) for the association between GSD and KSD. All extracted data were systematically recorded in an Excel spreadsheet.

Quality Assessment

The Newcastle-Ottawa Scale (NOS) was utilized to assess the quality and potential bias of the observational studies included in our analysis. The NOS evaluates studies based on three main domains, namely, selection of study groups, comparability of groups, and ascertainment of either the exposure (for case-control studies) or the outcome (for cohort studies). Overall, each study was rated on a scale of nine points, and studies with scores of seven or higher indicated high quality.¹⁷

Reporting Guidelines

The researchers followed the 2020 Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure that the systematic review was reported accurately, transparently, and comprehensively.

Statistical Analysis and Data Synthesis

The meta-analysis employed RR as the primary metric to assess the association between GSD and KSD. The effect sizes for the relationship between exposure and outcome were reported using 95% CI and RR. Random-effect models were utilized to compute the overall summary estimates. Forest plots were generated to represent individual RR and the summary estimates visually. In addition, subgroup analyses were conducted based on pre-specified criteria, including geographical region (Europe, America, and Asia), study sample size (<100 000 vs. ≥ 100 000 participants), study period (≤ 2015 vs. >2015), study quality (moderate vs. good), and the mean age of participants (≤ 50 years vs. > 50 years). Heterogeneity

among the studies was evaluated using Cochran's Q test, with Chi-square and a significance level of $P < 0.1$, and further assessed using the I^2 statistic. Sensitivity analyses were performed to identify potential sources of statistical heterogeneity and to evaluate the robustness of the meta-analysis findings. The influence of individual studies on the overall summary estimates was examined by recalculating pooled estimates after sequentially excluding one study at a time. A meta-regression analysis was also conducted to explore the sources of variability in the observed effect sizes across studies. Publication and accumulation biases were assessed using Begg's and Egger's tests. All statistical analyses were conducted using Stata 17.0 (Stata LLC, College Station, TX, USA), with a significance threshold set at $P < 0.05$.

Results

Search Results

The PRISMA flowchart, which outlines the search strategy employed in this study, is illustrated in Figure 1. The initial electronic database search resulted in the retrieval of 682 titles and abstracts. Upon review, 48 of these articles were excluded due to duplication, where the same title appeared more than once. Further screening led to the exclusion of additional titles and abstracts for several reasons. Three studies were excluded because they did not include the indices relevant to our research focus.¹⁸⁻²⁰ Another two studies were removed from consideration as

they were categorized as case series or case reports, which did not meet the inclusion criteria for this analysis.^{21,22} Additionally, two studies were excluded because they were published in a language other than English, which was beyond the scope of this review.^{23,24} After screening the articles, 9 studies were finally included in this systematic review (Figure 1).

Characteristics of Selected Studies

Our analysis encompassed 7 studies exploring the association between GSD and KSD. These studies were conducted between 2010 and 2023 in various countries, including the United States, Taiwan, Sweden, and Korea. In total, these studies included 982 847 participants. Quality assessment categorized 2 studies as moderate and 5 as good quality (Tables 1 and 2).

The Association Between Gallstone and Kidney Stone Disease

The meta-analysis indicates a significant association between GSD and KSD. The risk ratio for KSD in patients with GSD was 1.78 (95% CI=1.57-2.03, $P \leq 0.001$), underscoring the link between GSD and KSD (Figure 2).

Publication Bias Assessment

Publication bias was assessed using Begg's and Egger's tests, which indicated no significant bias (Begg's test, $P = 0.835$, Egger's test, $P = 0.812$). The symmetrical funnel

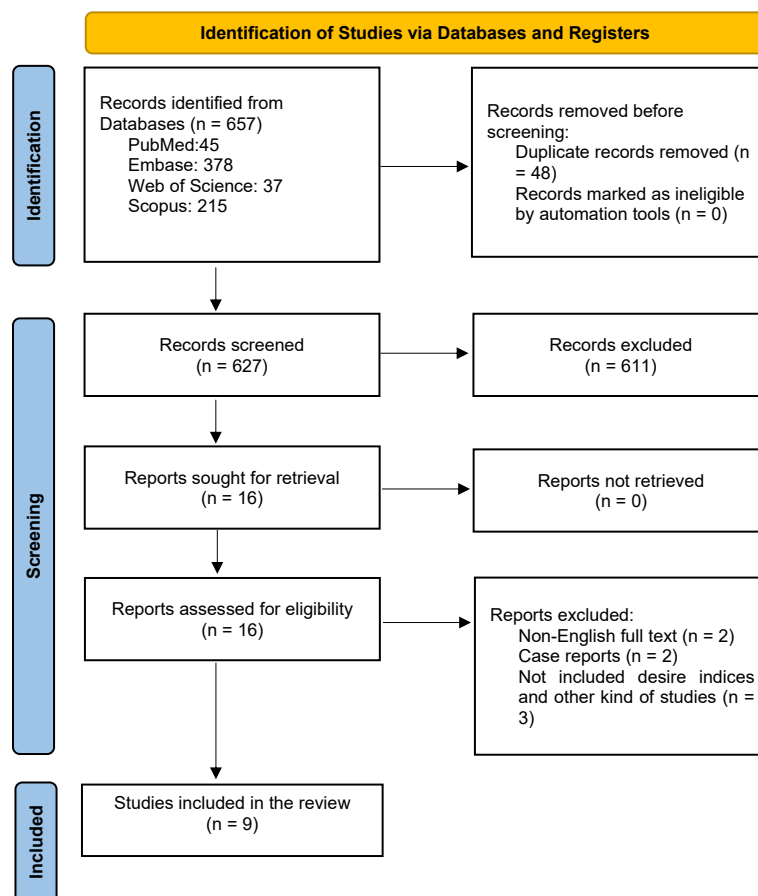


Figure 1. Flowchart for Including Studies in the Meta-analysis

Table 1. Characteristics of Included Studies

Lead Author	Year	Setting	Design	Sample Size	Number of Cases/Control	The Mean Age of Cases/Control	Male % of Cases/Control	Mean BMI of Cases/Control	Mean Follow-up Year	OR (95% CI)	NOS
Akoudad ²⁵	2010	USA	Prospective cohort study	12,161	974 (KSD)/ 11,187	60.4/60.0	66.3	28.3/ 28.5	10.8	HR: 1.41 (CI=0.78, 2.55)	8
Taylor, NHS I ²⁶	2011	USA	Prospective cohort study	121,700	6971 (GSD)/ 80,587	48/46	0	27/24	24	RR: 1.26 (CI=1.09, 1.44)	7
Taylor, NHS II ²⁶	2011	USA	Prospective cohort study	116,430	5694 (GSD)/ 102,530	38/36	0	29/25	14	RR: 1.32 (CI=1.14, 1.52)	7
Taylor, HPFS ²⁶	2011	USA	Prospective cohort study	51,529	2004 (GSD)/49,469	61/55	100	27/26	18	RR: 1.28 (CI=1.03, 2.57)	7
Li ²⁷	2014	Taiwan	Population-based retrospective cohort study	126,287	25 258 (GSD)/101029	55.9/55.4	45.5	NR	5	HR: 1.68 (CI=1.59, 1.77)	8
Hemminki ²⁸	2018	Sweden	Population-based cohort study	383,032	NA	51.77	43.8	NR	29	SIR for KSD 1.94 (CI=1.92-1.96) SIR for GSD 1.82 (CI=1.80—1.83)	7
Kim study I ²⁹	2019	Korea	Retrospective cohort study	10,355	20,711 (GSD)/82,844	54.8/54.8	48.4/ 48.4	NR	65.74	HRs of renal stones were 1.93 (95% CI=1.75–2.14) and 1.93 (95% CI=1.75–2.14) in the GSD group	8
Kim study II ²⁹	2019	Korea	Retrospective cohort study	118,075	23,615 (KSD)/ 94,460	46.1/46.1	64.6/64.6	NR	72.25	HRs of gallstones were 1.97 (95% CI=1.81–2.14) and 1.97 (95% CI=1.81–2.15) in the renal stone group	8
D'Amico ³⁰	2023	USA	Cohort	43,178	1063 (KSD)/ 42,115	38/38	0	31.1/30.2	10	OR 3.59 (95% CI=3.09–4.17)	8

Note. NHS: Nurse health study; HPFS: Health professional follow-up study; GSD: Gallstone; KSD: Kidney stone disease; NR: Not reported; NA: Not applicable; SIR: Standardized incidence ratio.

Table 2. Adjusted Variables in Included Studies

Lead Author	Year	Adjusted Variables
Akoudad ²⁵	2010	Age, gender, race, region, waist circumference, TG, HTN, DM, uric acid, and GSD
Taylor, NHS I ²⁶	2011	Age, BMI, use of thiazide diuretics, fluid intake, alcohol consumption, family history of KSD, HTN, DM, calcium supplement intake, use of animal protein, potassium, sodium, magnesium, sucrose, and caffeine
Taylor, NHS II ²⁶	2011	
Taylor, HPFS ²⁶	2011	
Li ²⁷	2014	Age, gender, HTN, hyperlipidemia, DM, UTI, liver cirrhosis, gout, Crohn's obesity, disease, and hyperparathyroidism
Hemminki ²⁸	2018	Age
Kim study I ²⁹	2019	Age, gender, income, region of residence, hypertension, diabetes, and dyslipidemia
Kim study II ²⁹		
D'Amico ³⁰	2023	Age, BMI, educational level, region, cigarette smoking, alcoholic consumption, Western dietary pattern, health insurance status, recent MD visit, DM, HTN, hyperlipidemia, and GSD

Note. TG: Triglycerides; HTN: Hypertension; DM: Diabetes mellitus; GSD: Gallstone; BMI: Body mass index; KSD: Kidney stone disease; UTI: Urinary tract infections.

plot (Figure 3) further supports the absence of bias.

Meta-regression Analysis

A meta-regression analysis was conducted to explore potential sources of heterogeneity. Variables such as study year, sample size, mean age of participants, mean follow-up, and study quality as determined by the NOS underwent examination. However, none showed a significant impact on heterogeneity ($P > 0.10$, Table 3).

Sensitivity Analysis

Sensitivity analysis involved sequentially removing each study to assess the robustness of the meta-analysis results. The estimated RR remained stable, indicating the robustness of the findings (Table 4 and Figure 4).

Subgroup Analysis

A thorough subgroup analysis was conducted to explore factors contributing to heterogeneity. The strength of the association varies depending on the location, period, sample size, age of participants, follow-up duration, and quality of the study, with more recent, lower-, and higher-

quality studies tending to show more robust associations. Table 5 provides the results of the subgroup analysis.

Discussion

This meta-analysis investigated the association between GSD and KSD. The pooled analysis revealed a statistically significant association between GSD and KSD, with a relative risk of 1.78 (95% CI=1.57-2.03, $P \leq 0.001$). In a meta-analysis conducted on cohort studies by Lin et al, KSD was significantly associated with increased risks of developing GSD by 46%. Additionally, GSD was linked to a substantially higher risk of nephrolithiasis (RR=1.49, 95% CI=1.28-1.73). Thus, they found a bidirectional relationship between KSD and GSD.¹⁴ Another review article reported that the risk of developing cholesterol GSD and KSD is significantly elevated in individuals with obesity, metabolic syndrome, non-alcoholic fatty liver disease, and insulin resistance. Emerging evidence also suggests that GSD is independently associated with an increased risk of KSD.¹⁵

Multiple factors influence the cause of KSD. The primary kind of kidney stone is calcium oxalate, which is produced at Randall’s plaque on the surfaces of renal papillae.

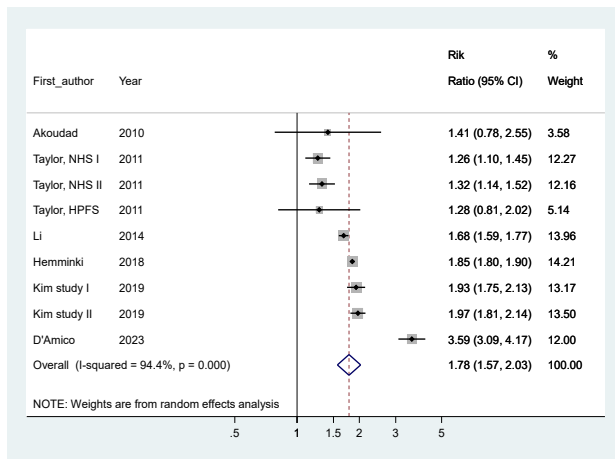


Figure 2. Forest Plot of the Relationship Between Gallstone and Kidney Stone Diseases

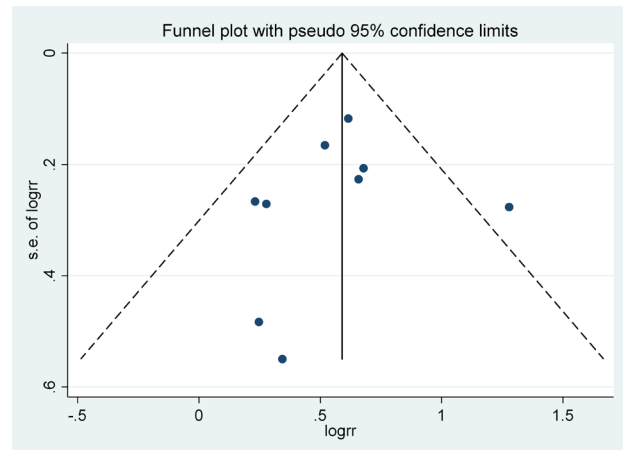


Figure 3. Funnel Plot for Evaluation of Publication Bias

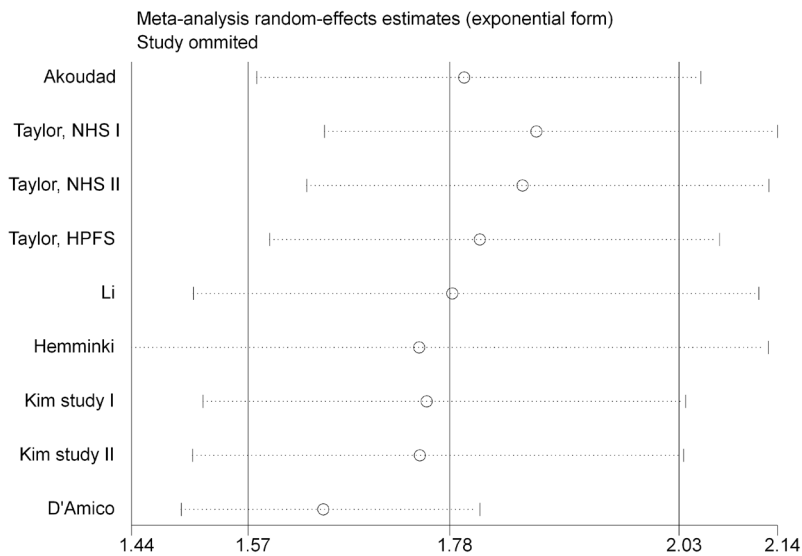


Figure 4. Sensitivity Analysis Plot for the Evaluation of the Relationship Between Gallstone and Kidney Stone Diseases

Table 3. Meta-regression Results in Studies Investigating the Relationship Between Gallstone and Kidney Stone Diseases

Meta-regression				Number of observations = 9		
REML estimate of between-study variance				Tau ² =0		
Residual variation due to heterogeneity				I-squared _{res} =0%		
The proportion of between-study variance explained				Adjusted R-squared=100%		
Joint test for all covariates				Model F (5, 3)=28.38		
With Knapp-Hartung modification				Prob>F=0.0099		
logrr	Coef.	Std. Err.	t	P > t	[95% CI]	
Year	0.046545	0.023585	1.97	0.143	-0.02851	0.121602
Sample size	-2.36E-07	7.44E-07	-0.32	0.772	-2.60E-06	2.13E-06
The mean age of cases control	-0.01183	0.009732	-1.22	0.311	-0.0428	0.019144
Mean follow-up (Year)	0.016937	0.019098	0.89	0.44	-0.04384	0.077715
NOS	0.411619	0.400967	1.03	0.38	-0.86444	1.687675
_cons	-95.9937	45.24809	-2.12	0.124	-239.993	48.00592

Note. REML: Restricted maximum likelihood; NOS: Newcastle-Ottawa Scale. * The significance level is $P \leq 0.1$. logrr: The natural logarithm of the risk ratio; Coef: Coefficient, Std. Err: Standard error; CI: Confidence interval; Cons: Constant.

Table 4. Results of Sensitivity Analysis for the Assessment of the Relationship Between Gallstone and Kidney Stone Diseases

Number	Publication First Author	Year	OR (95% CI)
1	Akoudad	2010	1.79 (1.57-2.05)
2	Taylor, NHS I	2011	1.87 (1.64-2.13)
3	Taylor, NHS II	2011	1.86 (1.62-2.12)
4	Taylor, HPFS	2011	1.81 (1.59-2.07)
5	Li	2014	1.78 (1.51-2.11)
6	Hemminki	2018	1.75 (1.44-2.12)
7	Kim study I	2019	1.75 (1.52-2.03)
8	Kim study II	2019	1.75 (1.50-2.03)
9	D'Amico	2023	1.64 (1.49-1.81)
Combined			1.78 (1.56-2.03)

Note. OR: Odds ratio; CI: Confidence interval.

Stone formation is intricate and arises from various physicochemical occurrences, such as oversaturation, nucleation, growth, aggregation, and the capture of urinary stone components within tubular cells.³¹ The formation of these stones is linked to decreased urine volume or increased excretion of substances that contribute to stone formation, such as calcium, oxalate, uric acid, cystine, xanthine, and phosphate. Calculi can also develop due to low urinary citrate levels or excessive urinary acidity.^{4,32,33} The ratio of parameters such as calcium/creatinine and uric acid/creatinine in patients with kidney stones is significantly higher than that in the control group. Moreover, in these studies, it has been shown that the ratio of oxalate to creatinine (oxalate/creatinine) significantly increases in people with urinary stones compared to the average population. In addition, the ratio of citrate to creatinine and magnesium to creatinine in the population with kidney stones are low.³⁴

Furthermore, the primary pathological mechanism underlying GSD is linked to the dysregulation of cholesterol and bile acid metabolism in the liver.^{35,36} Additionally, gut microbiota is essential for regulating bile acid composition, modulating the immune system, influencing

Table 5. Subgroup Analysis of the Association Between Gallstone and Kidney Stone Diseases

Characteristics		Number of Studies	OR (95% CI)	P Value
Study location	USA	5	1.63 (0.98-2.70)	0.058
	Europe	1	1.85 (1.80-1.90)	≤ 0.001
	Asia	3	1.85 (1.65-2.07)	≤ 0.001
Time period	2015 and before	5	1.41 (1.18-1.68)	≤ 0.001
	After 2015	4	2.21 (1.82-2.69)	≤ 0.001
Sample size	<100,000	3	2.14 (1.28-3.58)	0.004
	$\geq 100,000$	6	1.61 (1.44-1.81)	≤ 0.001
Mean age	≤ 50 years	4	1.85 (1.23-2.79)	0.003
	> 50 years	5	1.78 (1.65-1.92)	≤ 0.001
Follow-up	≤ 10 years	4	2.17 (1.70-2.76)	≤ 0.001
	> 10 years	5	1.43 (1.12-1.83)	0.004
Quality assessment	Good quality	5	2.09 (1.66-2.62)	≤ 0.001
	Moderate quality	4	1.43 (1.10-1.86)	0.007

Note. OR: Odds ratio; CI: Confidence interval.

gene expression, and controlling gallbladder motility.³⁷⁻⁴¹ Cholesterol-supersaturated vesicles have the potential to coalesce and form complex, multilayered liquid-crystal structures, often referred to as liposomes. This aggregation occurs when the cholesterol concentration in the bile exceeds its solubility limits. Under normal conditions, these vesicles are kept in suspension within the bile. However, a decrease in gallbladder contractility, often due to impaired motility or other functional disorders, can disrupt this balance. As a result, the supersaturated liposomes may crystallize into solid cholesterol monocrystals. This transition from a liquid-crystal state to solid crystals can contribute to the incidence of GSD.⁴²

Therefore, common risk factors for GSD and KSD, such as age, genetics, nutrition, mobility, and overweight, can cause the simultaneous occurrence of these two diseases in individuals, and this can be widely used in preventive and therapeutic strategies.^{5-7,9}

In this study, significant heterogeneity was observed

across the included studies (94%). Considering the small number of studies included in this meta-analysis, if a variable had an important role, the corresponding *P* value was not significant.

Limitations of the Study

The limitations of the studies included in this systematic review and meta-analysis were among the limitations of this study. The failure to examine the type of stones formed, as well as the failure to examine all confounding and risk factors, also caused high heterogeneity in this study.

Conclusion

There was a statistically significant relationship between GSD and KSD. The analysis consistently demonstrated a positive correlation between these two conditions across various subgroups, including geographic location, period, sample size, age, follow-up duration, and study quality. The results underscore the importance of considering the potential co-occurrence of these conditions in clinical practice. Healthcare providers should be aware of this association, particularly when managing patients with a history of these diseases. Further research is warranted to explore the underlying mechanisms linking these two diseases and to identify potential risk factors. Understanding these connections could contribute to more effective prevention and management strategies for GSD and KSD.

Authors' Contribution

Conceptualization: Faramarz Beigi, Ghorbanali Rahimian.

Data curation: Saeid Heidari-Soureshjani.

Formal analysis: Saeid Heidari-Soureshjani.

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Resources: Ghorbanali Rahimian.

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Supervision: Faramarz Beigi.

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Writing—original draft: Faramarz Beigi, Alizamen Salehifard Jouneghani, Saeid Heidari-Soureshjani, Catherine MT Sherwin, Ghorbanali Rahimian.

Writing—review & editing: Faramarz Beigi, Alizamen Salehifard Jouneghani, Saeid Heidari-Soureshjani, Catherine MT Sherwin, Ghorbanali Rahimian.

Competing Interests

One of the authors of this article serves as a technical administrator for the journal. Nevertheless, the review and publication process for this article has been conducted in accordance with the same standards applied to all other submissions, thereby ensuring consistency and impartiality in the review process.

Ethical Approval

Not applicable.

Funding

Nil.

Supplementary Files

Supplementary file 1. Detailed search strategy tailored for each database (PubMed/MEDLINE, Scopus, Web of Science, and

Embase).

References

1. Stamatelou K, Goldfarb DS. Epidemiology of kidney stones. *Healthcare (Basel)*. 2023;11(3):424. doi: [10.3390/healthcare11030424](https://doi.org/10.3390/healthcare11030424).
2. Cabo J, Gelikman DG, Hsi RS. The financial burden of nephrolithiasis and predictors of disease-specific financial toxicity. *Urology*. 2023;171:57-63. doi: [10.1016/j.urology.2022.08.053](https://doi.org/10.1016/j.urology.2022.08.053).
3. Vo AK, Somani BK, Ulvik Ø, Beisland C, Seitz C, Juliebø-Jones P. Measuring quality of life in patients with kidney stone disease: is it the future in endourology? *Curr Opin Urol*. 2024;34(2):91-7. doi: [10.1097/mou.0000000000001138](https://doi.org/10.1097/mou.0000000000001138).
4. Fan LL, Chen BH, Dai ZJ. The relation between gallstone disease and cardiovascular disease. *Sci Rep*. 2017;7(1):15104. doi: [10.1038/s41598-017-15430-5](https://doi.org/10.1038/s41598-017-15430-5).
5. Wang K, Ge J, Han W, Wang D, Zhao Y, Shen Y, et al. Risk factors for kidney stone disease recurrence: a comprehensive meta-analysis. *BMC Urol*. 2022;22(1):62. doi: [10.1186/s12894-022-01017-4](https://doi.org/10.1186/s12894-022-01017-4).
6. Qian X, Wan J, Xu J, Liu C, Zhong M, Zhang J, et al. Epidemiological trends of urolithiasis at the global, regional, and national levels: a population-based study. *Int J Clin Pract*. 2022;2022:6807203. doi: [10.1155/2022/6807203](https://doi.org/10.1155/2022/6807203).
7. Sui W, Hancock J, Asplin JR, Gould ER, Hsi RS. Nephrolithiasis and elevated urinary ammonium: a matched comparative study. *Urology*. 2020;144:77-82. doi: [10.1016/j.urology.2020.05.063](https://doi.org/10.1016/j.urology.2020.05.063).
8. Tamborino F, Cicchetti R, Mascitti M, Litterio G, Orsini A, Ferretti S, et al. Pathophysiology and main molecular mechanisms of urinary stone formation and recurrence. *Int J Mol Sci*. 2024;25(5):3075. doi: [10.3390/ijms25053075](https://doi.org/10.3390/ijms25053075).
9. Velkeniers B. Hormones after menopause? *Acta Clin Belg*. 2001;56(2):113-21. doi: [10.1179/acb.2001.019](https://doi.org/10.1179/acb.2001.019).
10. Wang X, Yu W, Jiang G, Li H, Li S, Xie L, et al. Global epidemiology of gallstones in the 21st century: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2024;22(8):1586-95. doi: [10.1016/j.cgh.2024.01.051](https://doi.org/10.1016/j.cgh.2024.01.051).
11. Unalp-Arida A, Ruhl CE. Increasing gallstone disease prevalence and associations with gallbladder and biliary tract mortality in the US. *Hepatology*. 2023;77(6):1882-95. doi: [10.1097/hep.0000000000000264](https://doi.org/10.1097/hep.0000000000000264).
12. Cariati A, Piromalli E. Limits and perspective of oral therapy with statins and aspirin for the prevention of symptomatic cholesterol gallstone disease. *Expert Opin Pharmacother*. 2012;13(9):1223-7. doi: [10.1517/14656566.2012.685161](https://doi.org/10.1517/14656566.2012.685161).
13. Wirth J, di Giuseppe R, Wientzek A, Katzke VA, Kloss M, Kaaks R, et al. Presence of gallstones and the risk of cardiovascular diseases: the EPIC-Germany cohort study. *Eur J Prev Cardiol*. 2015;22(3):326-34. doi: [10.1177/2047487313512218](https://doi.org/10.1177/2047487313512218).
14. Lin BB, Huang RH, Lin BL, Hong YK, Lin ME, He XJ. Associations between nephrolithiasis and diabetes mellitus, hypertension and gallstones: a meta-analysis of cohort studies. *Nephrology (Carlton)*. 2020;25(9):691-9. doi: [10.1111/nep.13740](https://doi.org/10.1111/nep.13740).
15. Ahmed MH, Barakat S, Almobarak AO. The association between renal stone disease and cholesterol gallstones: the easy to believe and not hard to retrieve theory of the metabolic syndrome. *Ren Fail*. 2014;36(6):957-62. doi: [10.3109/0886022x.2014.900424](https://doi.org/10.3109/0886022x.2014.900424).
16. Baddam A, Akuma O, Raj R, Akuma CM, Augustine SW, Sheikh Hanafi I, et al. Analysis of risk factors for cholelithiasis: a single-center retrospective study. *Cureus*. 2023;15(9):e46155. doi: [10.7759/cureus.46155](https://doi.org/10.7759/cureus.46155).
17. Peterson J, Welch V, Losos M, Tugwell PJ. The Newcastle-Ottawa scale (NOS) for Assessing the Quality of Nonrandomised Studies in Meta-Analyses. Ottawa: Ottawa Hospital Research

- Institute; 2011. p. 1-12.
18. Weikert C, Weikert S, Schulze MB, Pischon T, Fritsche A, Bergmann MM, et al. Presence of gallstones or kidney stones and risk of type 2 diabetes. *Am J Epidemiol.* 2010;171(4):447-54. doi: [10.1093/aje/kwp411](https://doi.org/10.1093/aje/kwp411).
 19. Beksac K, Tanacan A, Cagan M, Dönmez HG, Fadiloglu E, Unal C, et al. Relationship of cholelithiasis and urolithiasis with methylenetetrahydrofolate reductase polymorphisms. *J Invest Surg.* 2021;34(10):1104-7. doi: [10.1080/08941939.2020.1742402](https://doi.org/10.1080/08941939.2020.1742402).
 20. Andersson H, Bosaeus I, Fasth S, Hellberg R, Hultén L. Cholelithiasis and urolithiasis in Crohn's disease. *Scand J Gastroenterol.* 1987;22(2):253-6. doi: [10.3109/00365528708991889](https://doi.org/10.3109/00365528708991889).
 21. Blickman JG, Herrin JT, Cleveland RH, Jaramillo D. Coexisting nephrolithiasis and cholelithiasis in premature infants. *Pediatr Radiol.* 1991;21(5):363-4. doi: [10.1007/bf02011489](https://doi.org/10.1007/bf02011489).
 22. Ramey SL, Williams JL. Nephrolithiasis and cholelithiasis in a premature infant. *J Clin Ultrasound.* 1986;14(3):203-6. doi: [10.1002/jcu.1870140309](https://doi.org/10.1002/jcu.1870140309).
 23. Grosse H. [Frequency, localization and associated disorders in urinary calculi. Analysis of 1671 autopsies in urolithiasis]. *Z Urol Nephrol.* 1990;83(9):469-74. [German].
 24. Topchiashvili ZA, Penin VA. [Relationship between cholelithiasis and urolithiasis]. *Vestn Khir Im I I Grek.* 1968;100(2):73-5. [Russian].
 25. Akoudad S, Szklo M, McAdams MA, Fulop T, Anderson CA, Coresh J, et al. Correlates of kidney stone disease differ by race in a multi-ethnic middle-aged population: the ARIC study. *Prev Med.* 2010;51(5):416-20. doi: [10.1016/j.ypmed.2010.08.011](https://doi.org/10.1016/j.ypmed.2010.08.011).
 26. Taylor EN, Chan AT, Giovannucci EL, Curhan GC. Cholelithiasis and the risk of nephrolithiasis. *J Urol.* 2011;186(5):1882-7. doi: [10.1016/j.juro.2011.06.067](https://doi.org/10.1016/j.juro.2011.06.067).
 27. Li CH, Sung FC, Wang YC, Lin D, Kao CH. Gallstones increase the risk of developing renal stones: a nationwide population-based retrospective cohort study. *QJM.* 2014;107(6):451-7. doi: [10.1093/qjmed/hcu017](https://doi.org/10.1093/qjmed/hcu017).
 28. Hemminki K, Hemminki O, Koskinen AIM, Försti A, Sundquist K, Sundquist J, et al. Familial risks in and between stone diseases: sialolithiasis, urolithiasis and cholelithiasis in the population of Sweden. *BMC Nephrol.* 2018;19(1):158. doi: [10.1186/s12882-018-0945-y](https://doi.org/10.1186/s12882-018-0945-y).
 29. Kim SY, Song CM, Lim H, Lim MS, Bang W, Choi HG. Bidirectional association between gallstones and renal stones: two longitudinal follow-up studies using a national sample cohort. *Sci Rep.* 2019;9(1):2620. doi: [10.1038/s41598-019-38964-2](https://doi.org/10.1038/s41598-019-38964-2).
 30. D'Amico M, Wason S, Cozier YC. Correlates of nephrolithiasis in US black women: data from the black women's health study. *Urolithiasis.* 2023;51(1):29. doi: [10.1007/s00240-022-01391-6](https://doi.org/10.1007/s00240-022-01391-6).
 31. Alelign T, Petros B. Kidney stone disease: an update on current concepts. *Adv Urol.* 2018;2018:3068365. doi: [10.1155/2018/3068365](https://doi.org/10.1155/2018/3068365).
 32. Shastri S, Patel J, Sambandam KK, Lederer ED. Kidney stone pathophysiology, evaluation and management: core curriculum 2023. *Am J Kidney Dis.* 2023;82(5):617-34. doi: [10.1053/j.ajkd.2023.03.017](https://doi.org/10.1053/j.ajkd.2023.03.017).
 33. Han H, Segal AM, Seifter JL, Dwyer JT. Nutritional management of kidney stones (nephrolithiasis). *Clin Nutr Res.* 2015;4(3):137-52. doi: [10.7762/cnr.2015.4.3.137](https://doi.org/10.7762/cnr.2015.4.3.137).
 34. Jawalekar SL, Kulkarni UJ, Surve VT, Bhutay A. Evaluation of different urinary constituent ratios in renal stone formers. *Ann Biol Res.* 2010;1(3):50-5.
 35. Cortés VA, Barrera F, Nervi F. Pathophysiological connections between gallstone disease, insulin resistance, and obesity. *Obes Rev.* 2020;21(4):e12983. doi: [10.1111/obr.12983](https://doi.org/10.1111/obr.12983).
 36. Xie C, Huang W, Young RL, Jones KL, Horowitz M, Rayner CK, et al. Role of bile acids in the regulation of food intake, and their dysregulation in metabolic disease. *Nutrients.* 2021;13(4):1104. doi: [10.3390/nu13041104](https://doi.org/10.3390/nu13041104).
 37. Wang DQ, Cohen DE, Carey MC. Biliary lipids and cholesterol gallstone disease. *J Lipid Res.* 2009;50(Suppl):S406-11. doi: [10.1194/jlr.R800075-JLR200](https://doi.org/10.1194/jlr.R800075-JLR200).
 38. Grigor'eva IN, Romanova TI. Gallstone Disease and Microbiome. *Microorganisms.* 2020;8(6):835. doi: [10.3390/microorganisms8060835](https://doi.org/10.3390/microorganisms8060835).
 39. Larabi AB, Masson HL, Bäumlér AJ. Bile acids as modulators of gut microbiota composition and function. *Gut Microbes.* 2023;15(1):2172671. doi: [10.1080/19490976.2023.2172671](https://doi.org/10.1080/19490976.2023.2172671).
 40. Cai J, Sun L, Gonzalez FJ. Gut microbiota-derived bile acids in intestinal immunity, inflammation, and tumorigenesis. *Cell Host Microbe.* 2022;30(3):289-300. doi: [10.1016/j.chom.2022.02.004](https://doi.org/10.1016/j.chom.2022.02.004).
 41. Staley C, Weingarden AR, Khoruts A, Sadowsky MJ. Interaction of gut microbiota with bile acid metabolism and its influence on disease states. *Appl Microbiol Biotechnol.* 2017;101(1):47-64. doi: [10.1007/s00253-016-8006-6](https://doi.org/10.1007/s00253-016-8006-6).
 42. Reshetnyak VI. Concept of the pathogenesis and treatment of cholelithiasis. *World J Hepatol.* 2012;4(2):18-34. doi: [10.4254/wjh.v4.i2.18](https://doi.org/10.4254/wjh.v4.i2.18).