Medical : Jurnal Kesehatan Dan Kedokteran
Volume 3, Issue 2, July-December 2026
e-ISSN: 3064-6472 (online)
https://jurnal.sinesia.id/index.php/Medical-JKK/index

Therapeutic Efficacy And Clinical Outcomes Of Ursodeoxycholic Acid In Gallstone Disease And Metabolic Liver Disorders: A Narrative Review

Ihsanil Husna[1,2*], Feyza Desandra Ashila[3], Annisya M Safri[4], Hana Fadlina[2], Annisa Badriyyah Hakimah[5], Netti Suharti[6], Diana Laila Ramatillah[7], Yufri Aldi[8]
ihsanil.husna@umj.ac.id
[1]Doctoral Program in Pharmacy, Faculty of Pharmacy, Universitas Andalas, Padang, West Sumatra, Indonesia
[2] Department of Internal Medicine, Faculty of Medicine and Health Sciences, Universitas Muhammadiyah Jakarta, Jakarta, Indonesia
[3] Medical Student, Faculty of Medicine and Health Sciences, Universitas Muhammadiyah Jakarta, Jakarta, Indonesia
[4] Rumah Sakit Islam Jakarta Cempaka Putih, Central Jakarta, Jakarta, Indonesia
[5] Master's Program in Biomedical Sciences, Faculty of Medicine, Universitas Andalas, Padang, West Sumatera, Indonesia
[6] Department of Microbiology, Faculty of Medicine, Universitas Andalas, Padang, West Sumatra, Indonesia
[7] Faculty of Pharmacy, Universitas 17 Agustus 1945, Jakarta, Jakarta, Indonesia
[8] Faculty of Pharmacy, Universitas Andalas, Padang, West Sumatera, Indonesia

Info Article
|Submitted: 7 September 2026 **|Revised**: 24 September 2026 **|Accepted**: 4 November 2026
|Published: 4 November 2026
How to cite: Ihsanil Husna, etc., "Therapeutic Efficacy And Clinical Outcomes Of Ursodeoxycholic Acid In Gallstone Disease And Metabolic Liver Disorders: A Narrative Review", *Medical : Jurnal Kesehatan Dan Kedokteran*, Vol. 3, Issue 2, July-December, 2026, P. 362-375.

ABSTRACT

Ursodeoxycholic acid (UDCA) is a hydrophilic bile acid used in selected hepatobiliary disorders and cholesterol gallstone disease, with increasing interest in its cytoprotective, anti-inflammatory, and metabolic effects in metabolic dysfunction-associated steatotic liver disease (MASLD). This narrative review aimed to evaluate the therapeutic efficacy, clinical outcomes, safety, and limitations of UDCA in gallstone disease and metabolic liver disorders. Literature was identified from PubMed, ScienceDirect, and Google Scholar, prioritizing studies published between 2021 and 2026, while earlier landmark randomized trials were included when essential to assess dose, histological outcomes, or long-term efficacy. The evidence was analyzed qualitatively and synthesized thematically according to pharmacological mechanisms, gallstone prevention and dissolution, biochemical and histological liver outcomes, safety, dosing, and the potential dual role of UDCA in coexisting MASLD and gallstone disease. UDCA showed the greatest benefit in selected patients with small, radiolucent, cholesterol-rich, newly formed gallstones or biliary sludge and may reduce post-bariatric gallstone formation, biliary symptoms, hospital utilization, and the need for intervention. In metabolic liver disorders, UDCA consistently improved biochemical markers, particularly ALT, AST, and GGT, but these effects did not consistently translate into histological improvement or fibrosis regression. These findings indicate that UDCA has stronger evidence as a selective therapy for cholesterol gallstone disease than as a disease-modifying treatment for MASLD. Its role in metabolic liver disorders is best considered adjunctive, while direct evidence of simultaneous hepatic and biliary benefit remains limited.

Kata Kunci: *Ursodeoxycholic acid; Gallstone disease; Metabolic dysfunction-associated steatotic liver disease; Cholesterol gallstones; Bariatric surgery*

This work is licensed under CC BY-SA 4.0

Introduction

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly termed non-alcoholic fatty liver disease (NAFLD), is the most common chronic liver disorder and affects approximately 38% of the global adult population (EASL–EASD–EASO, 2024; Mali et al., 2025). MASLD is defined by hepatic steatosis accompanied by at least one cardiometabolic risk factor, including overweight or obesity, dysglycaemia, hypertension, or dyslipidaemia, in the absence of another identifiable cause (EASL–EASD–EASO, 2024). Its clinical spectrum ranges from metabolic dysfunction-associated steatotic liver to metabolic dysfunction-associated steatohepatitis (MASH), which is characterized by hepatocellular ballooning and lobular inflammation and may progress to fibrosis, cirrhosis, and hepatocellular carcinoma (Armandi & Bugianesi, 2024; EASL–EASD–EASO, 2024).

Lifestyle modification remains the cornerstone of MASLD management. International guidelines recommend sustained weight loss through caloric restriction, preferably within a Mediterranean dietary pattern, together with regular physical activity (Armandi & Bugianesi, 2024; EASL–EASD–EASO, 2024). Although these interventions may improve steatosis and inflammatory activity, long-term adherence is difficult, creating a need for effective pharmacological strategies. Resmetirom, a thyroid hormone receptor-β agonist, is among the few agents with evidence of MASH resolution and fibrosis improvement in registrational trials. Nevertheless, ursodeoxycholic acid (UDCA), a hydrophilic bile acid with cytoprotective, antioxidant, and choleretic properties, continues to attract interest because of its reported effects on liver enzymes and selected metabolic outcomes in MASLD (Amin et al., 2025; Armandi & Bugianesi, 2024; EASL–EASD–EASO, 2024; Nadinskaia et al., 2021).

MASLD also has a close epidemiological and pathophysiological relationship with gallstone disease (GSD). Insulin resistance, obesity, and dysregulated lipid metabolism contribute to both hepatic fat accumulation and cholesterol gallstone formation by promoting biliary cholesterol supersaturation and gallbladder hypomotility (Fujita et al., 2023; Mali et al., 2025). Population-based data indicate that MASLD is associated with higher odds of gallstones, with reported odds ratios ranging from 1.30 to 1.49, while approximately 41% of patients with symptomatic GSD may also meet diagnostic criteria for MASLD (Mali et al., 2025; Zhang et al., 2025). These findings support consideration of cardiometabolic assessment in patients presenting with gallstone disease.

Gallstones remain asymptomatic in most patients, but approximately 10%–20% may develop biliary colic, cholecystitis, pancreatitis, or other complications within 10 years (Fujita et al., 2023). Observation is generally appropriate for asymptomatic stones, whereas laparoscopic cholecystectomy remains the standard treatment for symptomatic disease. Rapid weight loss after bariatric surgery further

increases the risk of *de novo* gallstone formation, creating a specific setting in which preventive pharmacotherapy may be clinically relevant. UDCA has therefore been investigated both as prophylaxis after bariatric surgery and as an oral litholytic agent for selected cholesterol gallstones (Al-Huniti et al., 2023; Alrubaiaan et al., 2026; Hossain et al., 2023).

Despite its established biliary indications and reported biochemical benefits in MASLD, important uncertainties remain. In gallstone disease, therapeutic response varies according to stone composition, size, calcification, gallbladder function, and timing of treatment. In MASLD, reductions in ALT, AST, and GGT do not consistently translate into histological improvement or fibrosis regression. Furthermore, although MASLD and GSD frequently coexist and share common metabolic mechanisms, direct evidence that UDCA provides simultaneous clinically meaningful benefits for both conditions remains limited. This narrative review therefore aims to synthesize current evidence on the pharmacological mechanisms, efficacy, clinical outcomes, safety, dosing, and limitations of UDCA in gallstone disease and metabolic liver disorders, with particular attention to its potential role across the metabolic–biliary continuum.

Methods

Literature Search and Narrative Synthesis A structured literature search was conducted through PubMed, ScienceDirect, and Google Scholar, prioritizing publications from January 2021 to July 2026, while earlier landmark trials were included when they provided essential evidence on UDCA dose, histological outcomes, or long-term efficacy. Relevant studies were purposively selected, including randomized trials, cohort studies, systematic reviews, meta-analyses, guidelines, and selected mechanistic studies. The evidence was synthesized qualitatively according to major themes: mechanisms of action, gallstone prevention and dissolution, biochemical and histological outcomes in metabolic liver disorders, safety, dosing, and the potential dual role of UDCA in coexisting gallstone disease and MASLD.

Result and Discussion

Table 1. Summary of the therapeutic efficacy, clinical role, and limitations of UDCA

Clinical domain	Main findings	Patients most likely to benefit	Important limitations	Clinical interpretation
Post-bariatric gallstone prevention	UDCA reduces the incidence of de novo gallstones during rapid postoperative	Patients undergoing bariatric surgery, particularly during the period	Optimal dose and duration remain variable; adherence may be limited by	UDCA is supported as a preventive therapy in appropriately

	weight loss and may reduce gallstone-related interventions. Regimens of approximately 500–600 mg/day for 6–12 months are commonly supported, while 1,000 mg/day may offer greater short-term protection in some populations.	of rapid weight loss.	medication cost, nausea, and other gastrointestinal effects.	selected post-bariatric patients.
Gallstone dissolution	UDCA may dissolve cholesterol gallstones and biliary sludge, with higher success in stones measuring less than 5 mm and in newly formed stones.	Patients with small, radiolucent, non-calcified cholesterol stones, biliary sludge, and preserved gallbladder contractility who are unsuitable for or decline surgery.	Response is slower and less predictable for large, calcified, pigment, or long-standing stones. Evidence for combination therapy remains preliminary.	UDCA is a selective non-surgical option rather than a universal substitute for cholecystectomy.
Symptom and healthcare outcomes	Selected studies report reductions in dyspepsia, biliary colic, hospital visits, admissions, and the need for endoscopic or surgical intervention.	Patients with newly formed postoperative gallstones or sludge who meet criteria for medical treatment.	Evidence is heterogeneous, and improvement in symptoms is not consistently demonstrated across all populations.	Symptomatic benefits are possible but should not delay definitive surgery when clinically indicated.
Biochemical outcomes in MASLD	UDCA consistently reduces ALT, AST, GGT, and, in some studies, ALP. Improvements in FLI and lipid	Patients with biochemical liver abnormalities, particularly when a coexisting biliary indication is present.	Liver enzymes and FLI are surrogate outcomes and do not directly demonstrate resolution of	UDCA may provide biochemical stabilization and hepatoprotective support but is not an established

	parameters have also been reported.		steatohepatitis or fibrosis.	disease-modifying therapy for MASLD.
Hepatic steatosis and metabolic outcomes	Preclinical studies suggest effects on lipid oxidation, lipogenesis, oxidative stress, inflammation, gut-barrier integrity, and bile acid signalling. Clinical studies show selected improvements in metabolic and non-invasive indices.	Potentially patients with early metabolic liver dysfunction, although a definitive responsive phenotype has not been established.	Much of the mechanistic evidence is preclinical, and clinical surrogate improvements have not been consistently confirmed by histology.	Mechanistic plausibility is stronger than evidence for clinically meaningful structural liver recovery.
Histological and fibrosis outcomes	Standard-dose and high-dose UDCA have not consistently improved steatosis, necroinflammation, fibrosis, or global histological scores in randomized trials.	No clearly established subgroup for histological response.	Short-term changes in FibroTest or liver stiffness may reflect biochemical or measurement changes rather than true fibrosis regression.	Current evidence does not support UDCA as monotherapy for MASH resolution or fibrosis regression.
Coexisting MASLD and gallstone disease	UDCA may simultaneously improve selected biochemical liver markers and biliary outcomes through separate mechanisms.	Patients with MASLD and a clear biliary indication, such as small cholesterol stones or post-bariatric gallstone risk.	No robust trial has directly established a unified dual-disease benefit.	The potential dual role is plausible but should be considered adjunctive and individualized.
Safety and tolerability	UDCA is generally well tolerated; gastrointestinal symptoms, particularly diarrhoea and	Most appropriately selected patients without contraindications.	Higher doses may increase gastrointestinal intolerance, and long treatment duration can	UDCA has a favorable overall safety profile, but dose escalation should not be

	nausea, are the most common adverse effects.		reduce adherence.	assumed to improve efficacy.

Figure

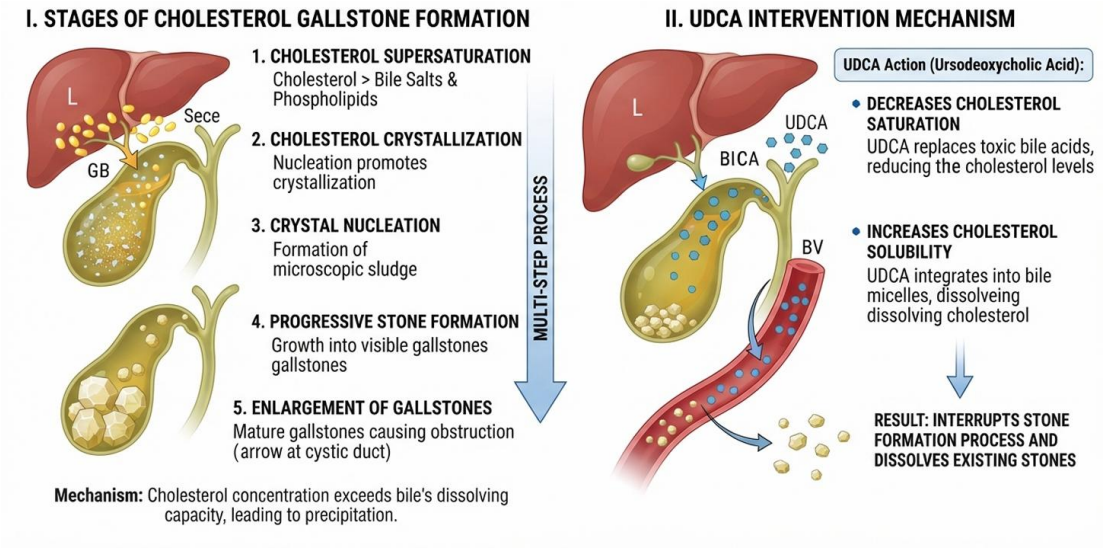


Figure 1. Stages of cholesterol gallstone formation and the mechanism of ursodeoxycholic acid intervention.

(A) Cholesterol gallstone formation begins with biliary cholesterol supersaturation, followed by cholesterol crystal nucleation, the development of microscopic biliary sludge, progressive stone formation, and enlargement into mature gallstones that may obstruct the cystic duct. (B) Ursodeoxycholic acid (UDCA) modifies the biliary environment by reducing cholesterol saturation and increasing cholesterol solubility within bile acid–phospholipid micelles. These effects may inhibit further crystal nucleation and stone growth and promote the gradual dissolution of selected cholesterol-rich gallstones. Note. L = liver; GB = gallbladder; UDCA = ursodeoxycholic acid.

Pharmacological Basis and Clinical Translation of UDCA

UDCA modifies the bile acid pool toward a more hydrophilic composition, reduces intestinal cholesterol absorption and biliary cholesterol secretion, and thereby lowers bile lithogenicity. These physicochemical effects explain its greater activity against biliary sludge and small, non-calcified cholesterol stones than against large or established stones (Lee et al., 2024; Stan et al., 2024). In a post-bariatric cohort, complete dissolution was reported in approximately 60% of treated patients, with a higher response among stones smaller than 5 mm (Buyuker et al., 2025). The difference between sludge, newly formed stones, and mature stones

suggests that stone organization, size, and treatment timing are more important determinants of response than exposure to UDCA alone.

Preclinical studies provide additional biological plausibility for hepatic effects. UDCA has been associated with activation of Nrf2/PPARγ, suppression of NF-κB signaling, increased CPT1A expression, reduced SREBP-1c expression, and improvements in intestinal tight-junction proteins and microbiota-related bile acid metabolism (Dong et al., 2026; Li et al., 2021). These pathways are consistent with antioxidant, anti-inflammatory, and lipid-regulatory activity. However, the mechanistic findings are predominantly derived from experimental models and should not be equated with disease modification in humans, because randomized clinical trials have not shown consistent global histological improvement (Leuschner et al., 2010; Lindor et al., 2004).

Therapeutic Outcomes in Gallstone Disease Prevention After Bariatric Surgery

Evidence is most consistent for prophylaxis during rapid weight loss after metabolic bariatric surgery. Meta-analyses of randomized trials reported a pooled risk ratio of approximately 0.33 for postoperative gallstone formation, corresponding to reductions of about 73% at 6 months and 67% at 12-24 months (Al-Huniti et al., 2023; Alrubaiaan et al., 2026). Observational data also suggest fewer ERCP procedures and fewer gallstone-related surgical or endoscopic interventions, although the reduction in cholecystectomy alone was not statistically significant in one cohort (Hossain et al., 2023). Thus, the preventive effect is supported more strongly for gallstone formation and composite biliary outcomes than for every individual intervention.

Dose comparisons are not entirely uniform. A study after sleeve gastrectomy favored 1,000 mg/day over 500 mg/day at 6 months, whereas meta-regression indicated substantial protection with 500-600 mg/day and did not identify baseline body mass index as a major modifier (Alrubaiaan et al., 2026; Fersahoğlu et al., 2022). These findings support regimen-specific use during the rapid weight-loss period rather than assuming that higher doses are universally superior.

Dissolution, Symptoms, and Predictors of Response

For oral litholysis, response is concentrated in carefully selected patients. Newly formed, radiolucent, non-calcified cholesterol stones, biliary sludge, preserved gallbladder function, and a diameter below 5 mm are associated with better outcomes, whereas pre-existing or larger stones respond less reliably (Buyuker et al., 2025; Demirpolat et al., 2025; Fujita et al., 2023). In patients who developed stones after sleeve gastrectomy, UDCA was associated with a lower subsequent cholecystectomy rate, but no significant reduction was observed for asymptomatic stones already present before surgery (Demirpolat et al., 2025). This

contrast supports early intervention during sludge or initial crystal formation rather than routine treatment of all established stones.

Clinical benefits may extend beyond radiological dissolution. Post-bariatric cohorts reported reductions in dyspepsia, biliary colic, hospital visits, and admissions, but these findings are derived mainly from observational populations and should not be generalized to all symptomatic gallstone disease (Buyuker et al., 2025; Hall et al., 2023). Laparoscopic cholecystectomy therefore remains the definitive treatment when symptoms or complications warrant surgery.

A preliminary randomized trial found that UDCA 600 mg/day combined with n-3 polyunsaturated fatty acids 3,000 mg/day produced higher radiological response and dissolution rates than UDCA alone (Lee et al., 2024). The complementary effects on biliary phospholipids and mucin provide a plausible explanation, but the preliminary design and limited evidence base require confirmation before combination therapy can be considered standard practice.

Therapeutic Outcomes in Metabolic Liver Disorders Biochemical and Metabolic Outcomes

Across systematic reviews and clinical studies, the most reproducible effect of UDCA in metabolic liver disorders is improvement in biochemical markers. Meta-analytic evidence indicates reductions in ALT, AST, GGT, and, in some studies, ALP, with larger ALT changes associated with longer treatment duration (Khurmatullina et al., 2026; Patel et al., 2024). A fixed dose of 300 mg/day improved ALT within 8 weeks, while 15 mg/kg/day for 6 months was associated with improvements in liver enzymes, the Fatty Liver Index, and lipid parameters (Chang et al., 2026; Nadinskaia et al., 2021). Because these outcomes are biochemical or surrogate measures, they indicate reduced biochemical activity but do not directly demonstrate resolution of steatohepatitis or fibrosis.

Some studies also reported lower carotid intima-media thickness and estimated cardiovascular risk, whereas vitamin E produced greater short-term transaminase reductions in one comparison (Amin et al., 2025; Nadinskaia et al., 2021). These observations suggest that UDCA may have broader metabolic effects, but cardiovascular event prevention and comparative superiority have not been established.

Dose and Histological Outcomes

Standard- and high-dose trials produced a consistent central finding: biochemical or localized inflammatory changes did not translate into global histological improvement. UDCA at 13-15 mg/kg/day for 2 years was not superior to placebo for steatosis, necroinflammation, or fibrosis, and increasing the dose to 23-28 mg/kg/day for 18 months did not improve overall histology, steatosis, or fibrosis (Leuschner et al., 2010; Lindor et al., 2004). High-dose therapy reduced lobular inflammation and GGT as secondary outcomes, but ALT and AST were not

significantly different from placebo at study completion (Leuschner et al., 2010). The absence of a clear histological dose-response relationship indicates that inadequate dose alone is unlikely to explain the negative structural outcomes.

Short-term reductions in FibroTest have been reported, while changes in liver stiffness were not statistically significant (Chang et al., 2026). FibroTest is partly influenced by biochemical components and therefore may change without true collagen regression. Similarly, CAP and liver stiffness measurement are useful for non-invasive assessment, but neither directly confirms treatment-related histological recovery (Cao et al., 2022). Current evidence consequently supports UDCA as an adjunct for biochemical improvement rather than monotherapy for MASH resolution or fibrosis regression.

Coexisting Gallstone Disease and MASLD

MASLD and gallstone disease share obesity, insulin resistance, dyslipidaemia, hypertension, and diabetes as major risk factors. Approximately 41% of patients with symptomatic gallstone disease met criteria for MASLD in one prospective study, and a population analysis found approximately 1.3-fold higher odds of gallstones among individuals with MASLD (Mali et al., 2025; Zhang et al., 2025). These studies demonstrate coexistence and association, not causality or a proven dual therapeutic effect.

UDCA could be clinically useful when a patient has both a valid biliary indication and biochemical liver abnormalities, because the drug may affect these outcomes through parallel mechanisms. Nevertheless, no robust trial has specifically shown that UDCA simultaneously modifies the natural history of both diseases. Its use in this setting should therefore remain individualized and adjunctive to weight management, dietary modification, physical activity, and treatment of cardiometabolic risk factors.

Safety, Dosing, and Overall Interpretation

UDCA is generally well tolerated, although gastrointestinal adverse effects are dose-related considerations. Standard-dose therapy was associated with more gastrointestinal events than placebo in one trial, and diarrhea occurred more frequently with high-dose treatment (Leuschner et al., 2010; Lindor et al., 2004). In post-bariatric practice, nausea and medication cost were important barriers to adherence (Hossain et al., 2023).

Dosing should follow the clinical objective: approximately 500-600 mg/day for 6-12 months has commonly been evaluated for post-bariatric prophylaxis; 600-1,000 mg/day has been studied for litholysis; and fixed or weight-based regimens have been evaluated for metabolic liver outcomes (Alrubaiaan et al., 2026; Buyuker et al., 2025; Chang et al., 2026; Lee et al., 2024). As summarized in Table 1, the evidence is strongest for post-bariatric prevention and selected cholesterol-stone dissolution, moderate for biochemical improvement in metabolic liver disease, and

insufficient for histological resolution or fibrosis regression. Dose escalation should not be assumed to improve efficacy and may reduce tolerability.

Clinical Implications and Future Perspectives

UDCA may have a supportive role across the metabolic–biliary spectrum, although its biochemical benefits should be distinguished from histological efficacy. Meta-analytic evidence indicates that UDCA can reduce ALT, AST, and GGT, with longer treatment duration associated with greater ALT reduction (Khurmatullina et al., 2026). However, standard-dose UDCA at 13–15 mg/kg/day for 2 years was not superior to placebo in improving steatosis, necroinflammation, or fibrosis, and current EASL–EASD–EASO guidance therefore does not support UDCA as a primary treatment for MASH (EASL–EASD–EASO, 2024; Lindor et al., 2004). Lifestyle modification and cardiometabolic risk management remain central. In gallstone disease, UDCA may be considered for carefully selected patients with small, radiolucent cholesterol stones and preserved gallbladder function, but its use should be individualized according to stone characteristics, symptoms, operative risk, treatment duration, and patient preference rather than viewed as a universal alternative to cholecystectomy (Fujita et al., 2023; Hall et al., 2023).

Future research should refine patient selection through validated non-invasive tools and more clinically meaningful endpoints. The FAST score may help identify patients at risk of progressive steatohepatitis, but its performance varies across BMI and demographic subgroups, while CAP may be less reliable in severe obesity and LSM is generally more informative for advanced fibrosis (Cao et al., 2022; Woreta et al., 2022). The high prevalence of MASLD among patients with symptomatic gallstone disease supports targeted metabolic assessment, although universal screening requires further validation and cost-effectiveness data (Mali et al., 2025). Short-term reductions in ALT and FibroTest with fixed-dose UDCA justify further investigation but should not be interpreted as evidence of fibrosis regression. Future trials should therefore include longer follow-up, standardized clinical and patient-centered outcomes, validated imaging, and histological assessment where appropriate to identify subgroups most likely to benefit (Chang et al., 2026).

Conclusion

UDCA has clinically relevant but indication-dependent benefits in gallstone disease and metabolic liver disorders. Its strongest evidence supports the prevention of de novo gallstones after bariatric surgery and oral dissolution in carefully selected patients with small, radiolucent, non-calcified cholesterol stones, biliary sludge, and preserved gallbladder function, whereas larger, calcified, pigment, or pre-existing stones are less likely to respond. In metabolic liver

disorders, UDCA consistently improves biochemical markers such as ALT, AST, and GGT and may favorably affect selected lipid and non-invasive metabolic indices; however, these changes have not consistently translated into histological improvement or fibrosis regression. Therefore, UDCA should be regarded as a selective non-surgical therapy for cholesterol gallstone disease and an adjunctive hepatoprotective agent in MASLD rather than a definitive disease-modifying treatment. Although MASLD and gallstone disease frequently coexist, direct evidence supporting a unified dual therapeutic effect remains limited, and future studies should clarify optimal dosing, treatment duration, predictors of response, and clinically meaningful outcomes in *patients with both conditions.*

Acknowledgment

The authors would like to thank all individuals and institutions that supported the preparation of this manuscript.

References

Al-Huniti, M., Alsardi, Y., Odeh, A., Bdour, B., Hassanat, R., Aloun, A., Sha'ban, B. W., & Nseirat, S. M. (2023). Ursodeoxycholic Acid Prophylaxis and the Reduction of Gallstone Formation After Bariatric Surgery: An Updated Meta-Analysis of Randomized Controlled Trials. Cureus. https://doi.org/10.7759/cureus.50649https://doi.org/10.7759/cureus.50649

Alrubaiaan, A., Altunaib, F., AlMutairi, S. A., AlFadhly, N., & AlKandari, A. F. (2026). Efficacy of prophylactic ursodeoxycholic acid in preventing gallstone formation after metabolic bariatric surgery: An updated systematic review and meta-analysis of randomized controlled trials. BMC Surgery. https://doi.org/10.1186/s12893-026-03983-0

Amin, M., Amin, F., Ather, A., & Ahmad, S. (2025). Comparison of vitamin E and ursodeoxycholic acid in non-alcoholic steatohepatitis (NASH). Pakistan Journal of Intensive Care Medicine, 5(02), 128. https://doi.org/10.54112/pjicm.v5i02.128https://doi.org/10.54112/pjicm.v5i02.128

Armandi, A., & Bugianesi, E. (2024). Dietary and pharmacological treatment in patients with metabolic-dysfunction associated steatotic liver disease. European Journal of Internal Medicine, 122, 20–27. https://doi.org/10.1016/j.ejim.2024.01.005

Buyuker, F., Sermet, M., Ozsoy, M. S., Tosun, S., Ekinci, Ö., Baysal, H., & Alimoglu, O. (2025). The effect of ursodeoxycholic acid in dissolving gallstones formed after laparoscopic sleeve gastrectomy: Retrospective cohort study.

Langenbeck's Archives of Surgery, 410(1), 91. https://doi.org/10.1007/s00423-025-03656-1

Cao, Y., Xiang, L., Qi, F., Zhang, Y., Chen, Y., & Zhou, X. (2022). Accuracy of controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) for assessing steatosis and fibrosis in non-alcoholic fatty liver disease: A systematic review and meta-analysis. eClinicalMedicine, 51, 101547. https://doi.org/10.1016/j.eclinm.2022.101547

Chang, Y., Cho, Y. K., Kim, Y. S., Kim, S.-E., Cheon, G. J., Kim, J. H., Yang, H., Kim, W., Ahn, S. B., Yoon, E. L., Cheong, J. Y., Lee, J.-W., Kim, M. Y., Kim, H. J., Lee, S. H., Cho, E. Y., Choi, N. R., Lee, H. W., Kim, K. M., … Jang, J. Y. (2026). Standard-Dose Ursodeoxycholic Acid Improves Biochemical Liver Function and Fibrosis in Chronic Liver Disease: Multicenter, Randomized, Double-Blind, Placebo-Controlled Trial. Journal of Korean Medical Science, 41(19), e89. https://doi.org/10.3346/jkms.2026.41.e89

Demirpolat, M. T., Celikkaya, M. O., Ertekin, S. C., Basak, F., & Sisik, A. (2025). Efficacy of ursodeoxycholic acid in reducing the necessity of cholecystectomy due to pre-existing and subsequently formed gallstones in patients who underwent laparoscopic sleeve gastrectomy. BMC Surgery, 25(1), 30. https://doi.org/10.1186/s12893-025-02767-2

Dong, X., Sun, W., Xu, H., Xie, Y., He, J., Liu, X., Liu, X., Ali, A., Chen, M., Zhang, L., Wu, L., & Shao, K. (2026). Ursodeoxycholic acid alleviates high-fat diet-induced liver injury by modulating gut microbiota-mediated bile acid metabolism: An integrated microbiota-metabolomics analysis. Frontiers in Nutrition, 13, 1714100. https://doi.org/10.3389/fnut.2026.1714100

European Association for the Study of the Liver (EASL), European Association for the Study of Diabetes (EASD), & European Association for the Study of Obesity (EASO). (2024). EASL-EASD-EASO Clinical Practice Guidelines on the Management of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). Obesity Facts, 17(4), 374–444. https://doi.org/10.1159/000539371

Fersahoğlu, M., Çakmak, A., Çiyiltepe, H., Esen Bulut, N., Fersahoğlu, A., Karip, A., Ergin, A., Bilgili, A., & Memişoğlu, K. (2022). The effect of different doses of ursodeoxycholic acid on gallstone formation after sleeve gastrectomy. Journal of Istanbul Faculty of Medicine / İstanbul Tıp Fakültesi Dergisi, 85(1), 29–34. https://doi.org/10.26650/IUITFD.948141https://doi.org/10.26650/IUITFD.948141

Fujita, N., Yasuda, I., Endo, I., Isayama, H., Iwashita, T., Ueki, T., Uemura, K., Umezawa, A., Katanuma, A., Katayose, Y., Suzuki, Y., Shoda, J., Tsuyuguchi, T., Wakai, T., Inui, K., Unno, M., Takeyama, Y., Itoi, T., Koike, K., & Mochida, S. (2023). Evidence-based clinical practice guidelines for cholelithiasis 2021.

Journal of Gastroenterology, 58(9), 801–833. https://doi.org/10.1007/s00535-023-02014-6

Hall, L., Halle-Smith, J., Evans, R., Toogood, G., Wiggins, T., Markar, S. R., Kapoulas, S., Super, P., Tucker, O., & McKay, S. C. (2023). Ursodeoxycholic acid in the management of symptomatic gallstone disease: Systematic review and clinician survey. BJS Open, 7(2), zrac152. https://doi.org/10.1093/bjsopen/zrac152

Hossain, I., Brodie, J., O'Brien, E., Tedman-Aucoin, K., Lawlor, D., Murphy, R., Twells, L., Pace, D., Evans, B., & Ellsmere, J. (2023). Ursodeoxycholic acid for prevention of gallstone disease after laparoscopic sleeve gastrectomy: An Atlantic Canada perspective. Surgical Endoscopy, 37(7), 5236–5240. https://doi.org/10.1007/s00464-023-10015-y

Khurmatullina, A. R., Andreev, D. N., Maev, I. V., Zaborovsky, A. V., Kucheryavyy, Y. A., Beliy, P. A., & Sokolov, P. S. (2026). Efficacy of ursodeoxycholic acid in metabolic dysfunction-associated steatotic liver disease: An umbrella review of meta-analyses on liver enzymes. Frontiers in Medicine, 13, 1771830. https://doi.org/10.3389/fmed.2026.1771830

Lee, S. Y., Jang, S. I., Cho, J. H., Do, M. Y., Lee, S. Y., Choi, A., Lee, H. S., Yang, J., & Lee, D. K. (2024). Gallstone Dissolution Effects of Combination Therapy with n-3 Polyunsaturated Fatty Acids and Ursodeoxycholic Acid: A Randomized, Prospective, Preliminary Clinical Trial. Gut and Liver, 18(6), 1069–1079. https://doi.org/10.5009/gnl230494

Leuschner, U. F. H., Lindenthal, B., Herrmann, G., Arnold, J. C., Rössle, M., Cordes, H., Zeuzem, S., Hein, J., & Berg, T. (2010). High-dose ursodeoxycholic acid therapy for nonalcoholic steatohepatitis: A double-blind, randomized, placebo-controlled trial†. Hepatology, 52(2), 472–479. https://doi.org/10.1002/hep.23727

Li, H., Wang, Q., Chen, P., Zhou, C., Zhang, X., & Chen, L. (2021). Ursodeoxycholic Acid Treatment Restores Gut Microbiota and Alleviates Liver Inflammation in Non-Alcoholic Steatohepatitic Mouse Model. Frontiers in Pharmacology, 12, 788558. https://doi.org/10.3389/fphar.2021.788558

Lindor, K. D., Kowdley, K. V., Heathcote, J. E., Harrison, E. M., Jorgensen, R., Angulo, P., Lymp, J. F., Burgart, L., & Colin, P. (2004). Ursodeoxycholic acid for treatment of nonalcoholic steatohepatitis: Results of a randomized trial. Hepatology, 39(3), 770–778. https://doi.org/10.1002/hep.20092

Mali, D., Gupta, S., A.K.Kaundal, Sharma, B., & Bodh, V. (2025). Prevalence and Severity of Metabolic Dysfunction-Associated Steatotic Liver Disease Among Patients with Gallstone Disease: A Prospective Hospital-Based Study. International Academic Research Journal of Surgery, 05(01), 1–6. https://doi.org/10.47310/iarjs.2025.v05i01.021

Nadinskaia, M., Maevskaya, M., Ivashkin, V., Kodzoeva, K., Pirogova, I., Chesnokov, E., Nersesov, A., Kaibullayeva, J., Konysbekova, A., Raissova, A., Khamrabaeva, F., & Zueva, E. (2021). Ursodeoxycholic acid as a means of preventing atherosclerosis, steatosis and liver fibrosis in patients with nonalcoholic fatty liver disease. World Journal of Gastroenterology, 27(10), 959–975. https://doi.org/10.3748/wjg.v27.i10.959

Patel, V. S., Mahmood, S. F., Bhatt, K. H., Khemkar, R. M., Jariwala, D. R., Harris, B., George, M. M., Kurudamannil, R. A., Anyagwa, O. E., Tak, R. S., & Kassem, M. (2024). Ursodeoxycholic Acid\'s Effectiveness in the Management of Nonalcoholic Fatty Liver Disease: A Systematic Review and Meta-analysis. Euroasian Journal of Hepato-Gastroenterology, 14(1), 92–98. https://doi.org/10.5005/jp-journals-10018-1434

Stan, S. I., Biciuşcă, V., Clenciu, D., Mitrea, A., Boldeanu, M.-V., Durand, P., & Dănoiu, S. (2024). The therapeutic mechanisms and beneficial effects of ursodeoxycholic acid in the treatment of nonalcoholic fatty liver disease: A systematic review. Medicine and Pharmacy Reports, 97(1), 12–25. https://doi.org/10.15386/mpr-2629https://doi.org/10.15386/mpr-2629

Woreta, T. A., Van Natta, M. L., Lazo, M., Krishnan, A., Neuschwander-Tetri, B. A., Loomba, R., Mae Diehl, A., Abdelmalek, M. F., Chalasani, N., Gawrieh, S., Dasarathy, S., Vuppalanchi, R., Siddiqui, M. S., Kowdley, K. V., McCullough, A., Terrault, N. A., Behling, C., Kleiner, D. E., Fishbein, M., … for the NASH Clinical Research Network. (2022). Validation of the accuracy of the FASTTM score for detecting patients with at-risk nonalcoholic steatohepatitis (NASH) in a North American cohort and comparison to other non-invasive algorithms. PLOS ONE, 17(4), e0266859. https://doi.org/10.1371/journal.pone.0266859

Zhang, Y., Zhang, J., Yu, D., Tu, F., Liu, J., Han, B., Li, B., Yuan, Y., Chen, C., & Zhou, M. (2025). Association between metabolic dysfunction associated steatotic liver disease and gallstones in the US population using propensity score matching. Scientific Reports, 15(1), 910. https://doi.org/10.1038/s41598-025-85218-5