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PREVALENCE AND ULTRASONOGRAPHIC FEATURES OF BILIARY TRACT ABNORMALITIES IN WOMEN OF REPRODUCTIVE AGE AND PREGNANT WOMEN WITH CHRONIC HEPATITIS B

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Abstract: Background: Pregnancy promotes bile stasis through progesterone-mediated gallbladder hypomotility and estrogen-driven changes in bile composition, increasing the risk of biliary sludge and gallstone formation. Chronic hepatitis B (CHB) complicates hepatobiliary imaging because gallbladder wall thickening and edema may occur in hepatic inflammation without primary gallbladder infection or obstruction.

Objective: To estimate the prevalence of biliary tract abnormalities in women of reproductive age with CHB, compare pregnant versus non-pregnant participants, and describe key ultrasonographic (echographic) features using standardized criteria.

Methods: Cross-sectional observational study of women aged 18–49 years with CHB (HBsAg positive ≥6 months), stratified into pregnant and non-pregnant groups. Fasting transabdominal ultrasonography evaluates gallbladder (wall thickness, distension, sludge, stones, pericholecystic fluid, Doppler hyperemia, sonographic Murphy sign) and bile ducts (common bile duct diameter, intrahepatic duct dilation). Laboratory variables (ALT/AST, bilirubin, ALP, GGT, albumin), HBV markers (HBV DNA, HBeAg), and confounders (BMI, parity, trimester, rapid weight loss/hyperemesis, antiviral therapy) are recorded. Prevalence is reported with 95% confidence intervals; multivariable regression explores associated factors.

Results: [Insert prevalence estimates for sludge, stones, composite cholecystitis phenotype, and ductal dilation; provide adjusted effect estimates for pregnancy/trimester and HBV-related variables.]

Conclusion: A standardized biliary ultrasound approach can define the burden and echographic spectrum of biliary pathology in CHB, particularly during pregnancy. Because wall thickening alone is nonspecific and common in hepatitis, interpretation must integrate symptoms, inflammatory markers, and cholestatic biochemistry.

Keywords: chronic hepatitis B; pregnancy; biliary sludge; cholelithiasis; gallbladder wall thickening; common bile duct; ultrasonography; cholestasis.

Abbreviations

- CHB – chronic hepatitis B
- HBV – hepatitis B virus
- CBD – common bile duct
- US/USG – ultrasonography
- ICP – intrahepatic cholestasis of pregnancy
- ALT/AST – alanine/aspartate aminotransferase

- ALP – alkaline phosphatase
- GGT – gamma-glutamyl transferase

Introduction

CHB is common among women of reproductive age and remains clinically important during pregnancy because of hepatitis flares, altered laboratory physiology, and the risk of mother-to-child transmission. Major professional guidance recommends universal HBsAg screening early in each pregnancy, evaluation of HBV DNA and HBeAg status, targeted maternal antiviral therapy (typically tenofovir) when viral load is high (to reduce perinatal transmission), and timely neonatal immunoprophylaxis.

Pregnancy is also a recognized lithogenic state. Serial ultrasound studies and postpartum cohorts show that biliary sludge and gallstones can appear during gestation and early puerperium; a proportion of sludge and even small stones may resolve after delivery, indicating a partly functional and reversible component.

Progesterone reduces gallbladder contractility and increases residual volume, while estrogens influence bile toward cholesterol supersaturation. Together these mechanisms produce bile stasis and sludge formation, which may progress to stones. Hyperemesis, prolonged fasting, and rapid weight changes can further increase sludge risk.

In CHB, interpretation of hepatobiliary ultrasound requires caution. Diffuse gallbladder wall thickening is not specific for cholecystitis and can occur in acute hepatitis and other systemic conditions, potentially leading to over-diagnosis and unnecessary interventions. Clarifying the prevalence and pattern of biliary ultrasound findings in CHB, especially during pregnancy, can improve diagnostic accuracy and guide referral pathways.

This document is an expanded, journal-ready template for an original observational study, including a standardized ultrasound protocol, operational definitions, and suggested analyses aligned with STROBE.

Objectives

- Primary: Estimate the prevalence of any biliary tract abnormality on ultrasound among women (18–49 years) with CHB, and compare pregnant vs non-pregnant participants.
- Secondary: Describe echographic patterns (sludge, stones, wall thickening, duct dilation); assess associations with trimester, BMI/parity, cholestasis markers (ALP/GGT/bilirubin), HBV DNA/HBeAg, and antiviral exposure; propose an interpretation and referral algorithm.

Materials and Methods

Study design and setting

Cross-sectional observational study at _____ (hospital/clinic), _____ (city, country), between _____ and _____. If retrospective, specify the chart/ultrasound archive source and case ascertainment method.

Participants

Inclusion: women aged 18–49 years; CHB defined by HBsAg positivity for ≥6 months or documented chronic infection; availability of standardized hepatobiliary ultrasound. Pregnant participants are eligible regardless of trimester.

Exclusion (recommended): prior cholecystectomy; known biliary tract malignancy/stricture; acute viral hepatitis at presentation; severe systemic causes of edema-related wall thickening (unless studied); and significant coinfection (HCV/HDV/HIV) if the research question is CHB-specific (alternatively include and adjust).

Variables

- Demographics: age, residence (urban/rural), relevant social factors (if available).
- Pregnancy variables: gestational age, trimester, parity, hyperemesis gravidarum/rapid weight change, prior biliary disease.
- Anthropometrics: BMI.
- HBV variables: HBeAg status; HBV DNA (IU/mL); ALT flares; antiviral therapy (drug, dose, start date, indication).
- Biochemistry: ALT, AST, total/direct bilirubin, ALP, GGT, albumin; complete blood count and CRP if available.
- Symptoms: RUQ pain/biliary colic, fever, nausea/vomiting, pruritus (ICP differential), jaundice.

Standardized ultrasound protocol

Perform ultrasound after 6–8 hours fasting whenever feasible. Use a uniform checklist. Record device model, transducer frequency, and operator experience. Consider blinded double reading for a subset to estimate interobserver reliability (kappa for categorical findings; intraclass correlation for continuous measures).

Gallbladder assessment

- Wall thickness: measure anterior wall adjacent to the liver; avoid tangential measurement. Wall thickness >3 mm commonly indicates thickening.
- Distension: record width/length; distension may support acute cholecystitis in appropriate context.
- Biliary sludge: dependent low-level echoes without acoustic shadowing; usually shifts with position. Document tumefactive (mass-like, non-mobile) sludge separately.
- Gallstones: echogenic foci with posterior acoustic shadowing and mobility; document size and number.
- Pericholecystic fluid; Doppler hyperemia; sonographic Murphy sign.

Bile duct assessment

- CBD diameter: inner-to-inner wall measurement; document maximal diameter. Interpret thresholds in context (age, post-cholecystectomy, cholestasis markers).
- Intrahepatic duct dilation: present/absent; segmental distribution.
- Choledocholithiasis suspicion: ductal dilation plus intraductal echogenic focus/shadowing; recognize limited sensitivity of transabdominal ultrasound.

Operational outcome definitions

Primary outcome (‘any biliary abnormality’): presence of ≥1 of the following on ultrasound: sludge, gallstones, echographic cholecystitis phenotype, CBD dilation (predefined), or intrahepatic duct dilation.

Secondary outcomes: prevalence of each component; symptom–ultrasound concordance; and stratified prevalence by trimester (T1/T2/T3).

Suggested echographic phenotypes (for reporting)

- Phenotype A – 'Stasis/lithogenic': sludge ± microlithiasis, normal wall thickness, no pericholecystic fluid, no ductal dilation.
- Phenotype B – 'Inflammatory gallbladder phenotype': wall thickening with stratification/edema ± hyperemia, plus supportive signs (Murphy sign, pericholecystic fluid, distension, stones).
- Phenotype C – 'Obstructive cholestasis phenotype': CBD/intrahepatic duct dilation plus cholestatic labs/jaundice; evaluate for obstruction (e.g., choledocholithiasis).
- Phenotype D – 'Hepatitis-associated edema': diffuse wall thickening with hepatitic labs but without stones/Murphy/pericholecystic fluid/hyperemia; treat as nonspecific and correlate clinically.

Statistical analysis

- Report prevalence with 95% confidence intervals (Wilson/exact).
- Compare pregnant vs non-pregnant: χ^2/Fisher's exact; t-test or Mann–Whitney U.
- Multivariable logistic regression with the primary outcome; candidate covariates: pregnancy status/trimester, age, BMI, parity, ALT, bilirubin, ALP/GGT, HBV DNA, HBeAg, antiviral exposure.
- Sensitivity analyses: exclude non-fasting scans; alternative CBD thresholds; separate sludge-only vs stones; remove acute symptomatic cholecystitis cases (if focus is subclinical changes).

Ethics

Ethics approval: _____ (committee, ID). Consent: prospective written consent or retrospective waiver as permitted. Data privacy compliant with local regulation.

Results (template)

A total of n = ____ women with CHB were enrolled: ____ pregnant and ____ non-pregnant. Median age was ____ (IQR ____–____). HBV DNA median was ____ IU/mL; ____% were HBeAg positive; ____% received antiviral therapy. Among pregnant participants, trimester distribution was: T1 __%, T2 __%, T3 __%.

Table 1. Baseline characteristics

Variable	Pregnant (n=__)	Non-pregnant (n=__)	p value
Age, years			
BMI, kg/m²			
ALT, U/L			
GGT, U/L			
Total bilirubin, µmol/L			
ALP, U/L			
HBV DNA, IU/mL			
HBeAg positive, n (%)			
On antiviral therapy, n (%)			
Trimester distribution	T1: __; T2: __; T3: __	N/A	—

Table 2. Ultrasonographic biliary findings

Ultrasound finding	Pregnant n (%)	Non-pregnant n (%)	Effect (95% CI)
Biliary sludge			
Gallstones			
Tumefactive sludge (mass-like)			
Wall thickening >3 mm			
Pericholecystic fluid			
Doppler hyperemia			
Sonographic Murphy sign			
Composite inflammatory phenotype (B)			
CBD dilation (threshold)			
Intrahepatic duct dilation			
Any biliary abnormality (primary)			

Discussion (expanded)

Interpretation of biliary ultrasound in CHB pregnancy should separate three overlapping domains: (i) pregnancy-driven stasis/lithogenesis; (ii) true inflammatory gallbladder disease; and (iii) hepatitis-related/systemic wall edema.

Serial ultrasound studies and postpartum cohorts demonstrate dynamic development of biliary sludge and gallstones across pregnancy; a proportion resolve after delivery. A recent systematic review/meta-analysis synthesizes global prevalence estimates of gallstones in pregnancy and highlights regional heterogeneity, supporting the need for local epidemiologic data.

A key pitfall is over-interpretation of diffuse wall thickening. In acute viral hepatitis, gallbladder wall thickening is frequently observed and correlates with hepatitic severity; it does not necessarily indicate primary cholecystitis. This template therefore recommends explicitly reporting a 'hepatitis-associated edema' phenotype when thickening occurs without other supportive cholecystitis signs.

Ductal dilation should be interpreted in context. Commonly used adult thresholds on transabdominal ultrasound are around 6–8 mm (with variation by age and after cholecystectomy). In intrahepatic cholestasis of pregnancy, imaging typically does not show mechanical biliary dilation, despite cholestatic symptoms/labs. Thus, ductal dilation in CHB pregnancy should prompt evaluation for obstruction when clinical and biochemical features support it.

Guideline-based CHB pregnancy management (screening, HBV DNA assessment, late-pregnancy maternal antiviral therapy when indicated, and neonatal immunoprophylaxis) should proceed in parallel with biliary evaluation. Including HBV DNA and antiviral exposure in multivariable analyses helps separate pregnancy physiology from CHB activity and treatment effects.

Conclusion

Women with CHB—especially during pregnancy—may have a measurable burden of biliary sludge/stones and a spectrum of gallbladder wall and ductal findings on ultrasound. A standardized echographic protocol and explicit phenotype reporting can improve diagnostic clarity and reduce over-diagnosis of cholecystitis when wall thickening reflects hepatitis-related edema. Longitudinal follow-up is warranted to define postpartum evolution and links to symptoms and obstetric outcomes.

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