Intrahepatic Cholestasis of Pregnancy: A Narrative Review

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ABSTRACT

This narrative review synthesizes current literature on the pathogenesis and management of Intrahepatic Cholestasis of Pregnancy (ICP). ICP is a multifactorial liver disorder. It is characterized by intense pruritus and elevated serum bile acids (SBA) or liver enzyme levels. These symptoms typically emerge in the second or third trimester and resolve postpartum. The etiology involves a complex interplay of genetic, hormonal (e.g., elevated estrogen and progesterone), and environmental factors. This is supported by varied global prevalence (9.2-15.6% in South America versus 0.1-0.5% in Europe) and high recurrence rates (45–90%). Diagnosis requires excluding other hepatobiliary diseases. Pruritus must be present with a random peak SBA concentration of at least 10 µmol/L (or 19 µmol/L, depending on the guideline used).

Ursodeoxycholic acid (UDCA) at 10–20mg/kg/day is the first-line treatment. It reduces maternal symptoms and transaminase levels, though its effect on stillbirth is debated. Vitamin K supplementation is advised for a prolonged prothrombin time. Antenatal surveillance includes monitoring liver function and SBA every 1-2 weeks. Fetal monitoring, though necessary, has limited predictive value for sudden fetal death. The risk of stillbirth strongly correlates with SBA levels (3.44% for ≥ 100µmol/L).

Management focuses on risk-stratified delivery timing. If SBA is ≥ 100 µmol/L, delivery should occur at 35–37 weeks. An SBA between 40 and 99 µmol/L suggests delivery at 37 weeks. If SBA is < 40 µmol/L, pregnancy may continue to 39 weeks. Postpartum, SBA, and liver function should be reassessed at 6–8 weeks.

Keywords: Intrahepatic cholestasis of pregnancy; Serum bile acid; Stillbirth; Ursodeoxycholic acid

Gynecol Obstet Reprod Med 2026;32(1):000-000

Etiology and Clinical Features

Intrahepatic cholestasis of pregnancy (ICP) is a liver disorder that usually occurs in the second and third trimesters and resolves after delivery. ICP presents as severe pruritus and elevated serum bile acids or liver enzyme levels, without other systemic or liver diseases (1,2). If symptoms persist beyond 48 hours after birth, and clinical or lab findings do not normalize within 2–3 weeks, reconsider the ICP diagnosis (1).

ICP prevalence varies by region and ethnicity: 9.2-15.6% in South America, 1-2% in North America and Australia, and about 0.1-0.5% in Europe (3,4). ICP is more common in women with hepatitis C, gallstones, multiple gestations, assisted reproduction, and those over 35 (5). Recurrence ranges from 45% to 90% (6).

The etiology of ICP is multifactorial. It involves genetic, endocrine, and environmental influences (1). A higher prevalence among first-degree relatives, mutations in hepatobiliary transport proteins, and epigenetic modifications related to pregnancy hormones support a genetic basis (7). Rising estrogen and progesterone concentrations in late gestation are major etiologic factors (7). Women with a history of ICP may also develop pruritus during use of combined oral contraceptives (8).

Epidemiological studies show increased incidence during winter months. Seasonal factors, such as vitamin D and selenium deficiencies, may contribute to the pathogenesis. Elevated estrogen levels are thought to increase hepatic oxidative stress. In women with low selenium and glutathione

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Submitted for Publication: 12.10.2025 Revised for Publication: 26.01.2026 Accepted for Publication: 31.03.2026 Online Published: 02.04.2026

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	Doi:10.21613/GORM.2025.1715

How to cite this article:*Arica G. Yilmaz I. Buldum D. Barut A. Madazli R. Intrahepatic Cholestasis of Pregnancy: A Narrative Review. Gynecol Obstet Reprod Med. 2026; Articles in Press*

peroxidase levels, this oxidative injury cannot be compensated for. As a result, hepatocellular damage and reduced bile secretion develop.

Sulfated progesterone metabolites may inhibit farnesoid X receptor (FXR), thereby suppressing bile salt export pump activity and reducing bile acid transport. These effects may contribute to ICP (10,11).

ICP diagnosis comes after excluding other liver, biliary, skin, and systemic diseases that can cause pruritus or cholestasis in pregnancy. The main symptom is pruritus, present in about one-fourth of pregnancies (2). Key differentials are atopic eruption, polymorphic eruption, pemphigoid gestationis, and ICP.

In ICP, itching usually starts in the second half of pregnancy. In 80% of cases, it begins after the 30th week, but sometimes starts as early as 7–8 weeks. The itching is usually widespread, worse on the palms and soles, and stronger at night, often causing sleep loss. There are no original skin rashes, but scratches from itching can appear. The itching is likely caused by bile acids irritating nerves in the skin.

Sometimes, abdominal pain, nausea, vomiting, anorexia, dark urine, or steatorrhea can occur (6). Mild jaundice and higher direct bilirubin may be present (1). A deficiency of fat-soluble vitamins, such as vitamin K, can prolong prothrombin time and increase the risk of bleeding (6).

Laboratory Findings: The most specific biochemical abnormality in ICP is an elevation in total serum bile acids (SBA)(6). However, the diagnostic threshold for serum bile acid concentration is debated, and various cut-off levels are used in clinical practice. Generally, a threshold of ≥10 µmol/L is used. Values over 15 µmol/L are considered confirmatory (1,2,5). For fasting measurements, 6–10 µmol/L is accepted as the upper limit of normal (5).

International societies have proposed differing thresholds for serum bile acid concentrations. The Royal College of Obstetricians and Gynecologists (RCOG) 2022 guideline notes that there is no single diagnostic test for ICP. However, a random peak serum bile acids (SBA) concentration ≥19 µmol/L with pruritus is considered diagnostic (12). Severity is classified by the South Australian Maternal and Neonatal Community of Practice (SAMNCP) as mild (19-39 µmol/L), severe (≥40 µmol/L), and very severe (≥100 µmol/L) (13). Similarly, the RCOG stratifies the disease into mild (19–39 µmol/L), moderate (40-99 µmol/L), and severe (≥100 µmol/L) (12). Diagnostic criteria by international societies are summarized in table I.

Doctors often obtain bile acid samples from fasting patients, but there is limited information on normal fasting bile acid levels by pregnancy week. Some studies show no big difference between fasting and non-fasting bile acid levels in people who are not pregnant. However, in one study of pregnant women, bile acid levels measured 1 and 3 hours after a meal were higher than fasting levels. More research is needed to know what is normal during pregnancy. Right now, guidelines advise random (not fasting) bile acid testing, which is easier for both patients and doctors.

Bile acids are cytotoxic end products of hepatic cholesterol metabolism. They are transported through the bile ducts to the gallbladder (5). In cholestasis, impaired excretion leads to elevated serum SBA concentrations. The rise in SBA may occur after the onset of pruritus. If levels are initially normal, they should be re-measured within 1-2 weeks (2,16).

In ICP, serum cholic acid rises, and chenodeoxycholic acid falls, increasing their ratio (17). Liver enzymes rise in 60-85% of cases, with ALT usually higher than AST and increasing up to 2-15 times normal (6,17). Alkaline phosphatase (ALP) is not specific since the placenta also produces it. Gamma-glutamyl transferase (GGT) is usually normal (6,17). Bilirubin is high in about 10% (5).

Maternal and Fetal Effects of ICP: ICP pregnancies have higher rates of gestational diabetes mellitus (GDM) and preeclampsia (16,18). Arafa and Donga found that the GDM risk is 2.19 times higher (95% CI 1.58-3.03), and the preeclampsia risk is 2.58 times higher (95% CI 2.37-2.81) than in normal pregnancies (19). A few women may have lasting liver problems after birth, so repeat bile acids and liver enzyme tests 6–8 weeks after delivery (16).

Women with a history of ICP have an increased long-term risk of hepatobiliary disease and should be counseled accordingly (16,17).

ICP is linked to more spontaneous preterm birth, meconium-stained amniotic fluid, abnormal cardiotocography

Table I. *Diagnostic criteria of intrahepatic cholestasis in pregnancy based on international societies*

Organization	Diagnostic Criteria
SMFM(2)	Pruritus + Total serum bile acid level >10 µmol/L (not clearly mentioned)
RCOG(12)	Pruritus + Random peak bile acid >19 µmol/L
FIGO(31)	Pruritus + Non-fasting serum bile acid >10 µmol/L
SAMNCP(13)	Pruritus + Non-fasting serum bile acid >19 µmol/L

RCOG: Royal college of obstetricians and gynecologists, FIGO: International federation of gynecology and obstetrics, SMFM: Society for maternal-fetal medicine, SAMNCP: South australia maternal and neonatal community of practice

(CTG), sudden fetal death, and neonatal respiratory distress syndrome (RDS) (16,17,20). Sudden, unexplained fetal deaths in otherwise normal fetuses are a major concern in ICP management.

The severity of maternal symptoms does not correlate with adverse perinatal outcomes (7,16,17). However, the risk of fetal death correlates with maternal serum bile acid levels(16,21). In a meta-analysis by Ovadia et al., stillbirth rates were 0.13%, 0.28%, and 3.44% for bile acid levels <40, 40-99, and ≥100 μmol/L, respectively (21).

The fetus begins to produce bile acids from the 12th gestational week onward, which normally pass across the placenta into the maternal circulation (5). In ICP, maternal bile acids cross to the fetus, causing toxic accumulation that impairs placental and fetal function (5). Bile acids can induce vasoconstriction of placental chorionic vessels, leading to acute anoxia and sudden death (5,16). They may also increase oxidative stress and apoptosis within the placenta (7).

They can also act directly on the fetal myocardium, causing arrhythmia and cardiac arrest(5,22). The incidence of RDS in neonates of ICP pregnancies is higher even after adjustment for gestational age (16,19). Bile acids may cause inflammation and surfactant dysfunction in the fetal lung, contributing to the development of RDS (23). In our study, we demonstrated that ICP diagnosed before 30 weeks of gestation was associated with adverse perinatal outcomes (20).

Treatment: The first-line treatment for ICP is ursodeoxycholic acid (UDCA)(1). UDCA is a natural bile acid constituting 1-3% of bile acids in healthy individuals (24). It is safe for use during pregnancy, with no known maternal or fetal adverse effects (24). UDCA has been shown to reduce pruritus, serum transaminase, and bile acid levels (16).

A meta-analysis by Kong et al. demonstrated that UDCA significantly reduced the risks of preterm birth, fetal distress, low 5-minute Apgar scores, neonatal RDS, and NICU admission, with relative risk reductions of 44-67% (25). Another meta-analysis by Ovadia et al. found no significant reduction in stillbirth rates (0.7% in treated vs 0.6% in untreated pregnancies; aOR 1.04, 95% CI 0.35–3.07) (26).

Nevertheless, UDCA is recommended to improve maternal and perinatal outcomes (27,28). Although it decreases total serum bile acid levels, a recent study showed that even after normalization of serum bile acids, the cholic/chenodeoxycholic acid ratio in maternal and cord blood plasma remained elevated (28).

The recommended dosage is 10-20 mg/kg/day for mild disease, 250 mg × 3/day or 300 mg × 2/day; for severe cases, 500 mg × 3/day or 600 mg × 2/day (1). Other agents used include rifampicin, cholestyramine, hydroxyzine, S-adenosylmethionine, phenobarbital, dexamethasone, and activated charcoal(3,16). None is as effective as UDCA in improving symptoms or biochemical parameters (3,30).

RCOG recommends vitamin K supplementation (5-10 mg/day)in cases with prolonged prothrombin time (12), though universal prophylaxis is not yet agreed upon (1).

There is limited evidence regarding the efficacy of topical emollients for pruritus relief, but they may be recommended due to their favorable safety profile and symptom-relieving effects (12). Antihistamines such as chlorphenamine can also be considered to improve comfort at night due to their sedative side effects (12).

Management of Pregnancy: Guidelines recommend monitoring liver enzymes every 1–2 weeks and evaluating coagulation parameters, especially in women with elevated enzymes (1). The Society for Maternal-Fetal Medicine (SMFM)advises screening for hepatitis C in ICP pregnancies (2). SAMNC recommends hospital admission for patients with bile acids >40 μmol/L or ALT >200 IU/L (9).

Fetal growth and well-being should be monitored closely; however, the efficacy of CTG and Doppler studies in preventing adverse outcomes or sudden fetal loss remains uncertain (1). ICP is not associated with fetal growth restriction, and birth weights are comparable to those of uncomplicated pregnancies. Antenatal surveillance tests (e.g., CTG and Doppler studies), which are valuable for assessing fetal acidosis and well-being in conditions such as placental insufficiency, offer limited prediction for fetal death associated with ICP(12).

Current guidelines recommend delivery at 37–38 weeks (1,2). Timing should be individualized after discussing risks with the family. Delivery at 37 weeks is appropriate for bile acids > 40 μmol/L; if bile acids are < 40 μmol/L and no additional obstetric indications exist, the pregnancy may continue to 39 weeks (2,16). For bile acids ≥ 100 μmol/L, the risk of fetal death is high, and delivery between 35–37 weeks may be considered (1,2,16). Recommendations from international organizations on delivery timing based on bile acid levels are summarized in table II.

There is no consensus on monitoring SBA levels after the initiation of UDCA treatment. However, repeated SBA measurements can be considered in patients with unresolved clinical and laboratory findings of ICP. RCOG recommends repeating SBA measurements weekly as delivery approaches in cases of mild and moderate ICP to guide a reconsideration of delivery timings should SBA levels increase (12). Conversely, a decrease in SBA levels following UDCA treatment should not be used to determine the timing of delivery.

ICP itself is not an indication for cesarean section; induction of labor may be performed with continuous electronic fetal monitoring (1). Cesarean section rates are, however, higher among ICP pregnancies.

Table II: *Recommendations for delivery timing based on bile acid levels from international societies*

Organization	Criteria	Recommended Gestational
Week for Delivery		
ACOG(32)	Total bile acid levels <100 μmol/L	360/7–390/7 weeks or at diagnosis
SMFM(2)	Total bile acid levels ≥100 μmol/L Delivery before 36 weeks if necessary	360/7 weeks or at diagnosis
RCOG(12)	Random peak bile acid 19–39 μmol/L Random peak bile acid 40–99 μmol/L Random peak bile acid ≥100 μmol/L	by 40 weeks 38–39 weeks 35–36 weeks
FIGO(31)	Non-fasting serum bile acid 10–39 μmol/L Non-fasting serum bile acid 40–99 μmol/L Non-fasting serum bile acid ≥100 μmol/L	37–39 weeks 36–39 weeks (closer to 36 weeks) 35–36 weeks

Competing Interests: The authors declare that they have no competing interests.

Conflict of Interest: No conflict of interest is declared by the authors.

Authors' contributions: DB, IY, and AB participated in the literature search. GA and RM contributed to the writing of the paper and draft revision. All authors have read and approved the final manuscript.

Acknowledgment: This study has been published on TMFTP under the Expert Opinions category.

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