SCIE DOI: 10.14744/scie.2025.83792
South. Clin. Ist. Euras. 2025;36(4):317-322

Original Article

Intrahepatic Cholestasis of Pregnancy and Neonatal Acidemia: A Retrospective Analysis of Perinatal Outcomes

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Submitted: 04.07.2025
Revised: 22.10.2025
Accepted: 04.11.2025

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Keywords: Bile acids; fetal cord blood pH; intrahepatic cholestasis of pregnancy; neonatal outcomes; perinatal morbidity.

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ABSTRACT

Objective: This study aimed to evaluate the incidence of neonatal acidemia and its association with fasting bile acid levels in pregnancies diagnosed with intrahepatic cholestasis.

Methods: This retrospective cohort study included pregnant women who delivered at the Department of Obstetrics and Gynecology of our institution between January 2020 and March 2025. Patients diagnosed with intrahepatic cholestasis of pregnancy (ICP) were enrolled.Maternal and neonatal data were collected from hospital records, including demographic characteristics, laboratory values, treatment regimens, delivery details, and neonatal outcomes. Statistical analysis was conducted using SPSS version 24.0.

Results: A total of 58 women with intrahepatic cholestasis of pregnancy (ICP) were included. Based on serum bile acid levels at diagnosis, patients were divided into two groups: <40 μmol/L (n=41) and ≥40 μmol/L (n=17). No significant differences were observed between the groups regarding maternal demographics, gestational age, birth weight, or history of ICP (p>0.05). Preterm birth rates were similar in both groups (48.8% vs. 47.1%, p=1.000). NICU admissions were more common in the ≥40 μmol/L group (70.6% vs. 53.7%), but not statistically significant (p=0.36). Incidences of neonatal jaundice, tachypnea, and respiratory distress syndrome showed no significant group differences (p>0.05). Neonatal acidemia, defined as umbilical cord pH <7.20 or <7.10, occurred at comparable rates across both bile acid groups, without statistically significant differences. The mean pH was 7.29±0.11 in the <40 μmol/L group and 7.28±0.11 in the ≥40 μmol/L group (p=0.856). No correlation was observed between maternal bile acid levels and neonatal umbilical cord pH (r=–0.02, p=0.884).

Conclusion: No significant association was found between maternal bile acid levels and neonatal acidemia. Further prospective studies are needed to clarify this relationship in intrahepatic cholestasis of pregnancy.

INTRODUCTION

Intrahepatic cholestasis of pregnancy is the most commonly diagnosed liver disorder during pregnancy, particularly in the second and third trimesters. The condition is characterized by widespread pruritus, which typically intensifies at night, along with elevated liver transaminase levels (AST, ALT) and increased serum bile acid concentrations.[1] The prevalence of ICP shows significant variation across different geographic regions and ethnic groups, with reported rates ranging from 0.4% to 10%.[2] A range of risk factors including metabolic and hepatobiliary conditions like gestational diabetes, obesity, hepatitis C, and a previous ICP diagnosis have been linked to the emergence of this disorder.[3]

The diagnosis is made on the basis of a serum bile acid level of ≥10 μmol/L in patients presenting with pruritus.[4] Some guidelines recommend ≥19 μmol/L as the diagnostic threshold; however, a level of ≥10 μmol/L is generally accepted.[5,6] While the collection of fasting samples is considered the optimal method for measuring serum bile acids, recent clinical guidelines also accept postprandial samples as valid for diagnostic purposes.[4,6]

The pathophysiology of intrahepatic cholestasis of pregnancy involves impaired bile acid transport across the hepatocellular membrane due to hormonal, genetic, and environmental factors. Elevated estrogen and progesterone metabolites are thought to inhibit bile acid excretion, leading to intrahepatic accumulation. This accumulation increases the permeability of the placental barrier and

may cause fetal cardiotoxicity, vasoconstriction of placental vessels, and arrhythmias, which together contribute to adverse fetal outcomes such as hypoxia and sudden intrauterine death.[7]

It has been reported that fetal complications are associated with serum bile acid levels. Consequently, the severity of the disease is categorized as mild (10-40 µmol/L), moderate (40-100 µmol/L), and severe (>100 µmol/L) based on the bile acid level. This classification system serves as a clinical guide, informing the monitoring of disease progression and the determination of delivery timing.[8]

Although elevated maternal bile acid levels in intrahepatic cholestasis of pregnancy have been linked to various neonatal complications such as meconium stained amniotic fluid and preterm birth, objective data on neonatal acidemia, particularly based on umbilical cord blood gas analysis, remain sparse and inconsistent.

Based on previous studies reporting associations between bile acids and adverse fetal outcomes, we hypothesized that elevated maternal bile acid levels would correlate with neonatal acid-base disturbances, particularly acidemia.

This study aimed to assess perinatal outcomes in pregnancies complicated by intrahepatic cholestasis and to explore the relationship between maternal bile acid concentrations and neonatal acidemia.

MATERIALS AND METHODS

Study Design and Setting

The present study was designed as a retrospective cohort analysis. All women who delivered at the Department of Obstetrics and Gynecology of our institution between January 2020 and March 2025 were screened; those with a confirmed diagnosis of intrahepatic cholestasis of pregnancy (ICP) were included in the study. Approval for the study was provided by the Institutional Ethics Committee of Kartal Dr. Lütfi Kırdar City Hospital, and all procedures adhered to the ethical principles outlined in the Declaration of Helsinki (No: 2025/010.99/16/6, Date: 27/05/2025).

Patient Selection and Exclusion Criteria

ICP was diagnosed based on the presence of maternal pruritus and fasting serum bile acid levels ≥10 µmol/L, following the exclusion of other hepatic and dermatologic conditions. As per institutional protocol, all patients received ursodeoxycholic acid (UDCA) treatment.

Exclusion criteria included cases with incomplete neonatal outcome data or missing cord blood gas values, maternal age below 18 or above 45 years, and pregnancies complicated by fetal anomalies. As this was a retrospective study, informed consent was not required; however, patients with inaccessible or incomplete medical records were excluded from the analysis.

Data Collection

Maternal and neonatal data were retrospectively collected from the electronic medical records. The collected maternal variables included demographic characteristics such as maternal age, gravida and parity, as well as medical history including any comorbidities during pregnancy. Detailed information regarding the gestational week at the time of ICP diagnosis was recorded, along with laboratory parameters such as fasting serum bile acid levels at the time of diagnosis and again prior to delivery. Furthermore, an assessment was conducted to determine the presence of hepatic involvement. This assessment entailed the observation of liver enzyme levels, encompassing alanine aminotransferase (ALT) and aspartate aminotransferase (AST).

Information regarding the use of medical treatment, particularly ursodeoxycholic acid (UDCA), was documented in detail. The administered UDCA dose ranged between 10 and 15 mg/kg/day, divided into two or three doses per day. The duration of therapy was calculated from the date of diagnosis until delivery. Dose adjustments were made based on symptom severity and serum bile acid levels, in accordance with institutional protocol.

Abdominal ultrasound findings were evaluated for the presence of hepatobiliary abnormalities, including hepatomegaly, biliary sludge, gallstones, and intrahepatic ductal dilatation, as well as any other significant anatomical or pathological findings. Obstetric outcomes included mode of delivery, indication for cesarean delivery if applicable, and gestational age at the time of birth.

Neonatal outcomes included neonatal birth weight and sex, need for neonatal intensive care (NICU), duration of NICU stay, umbilical cord blood pH, and the presence of respiratory distress syndrome, tachypnea, or neonatal jaundice.

Statistical Analysis

For the purpose of all statistical analyses, the IBM SPSS Statistics for Mac version 24.0 software was employed. In this study, we utilized the Kolmogorov–Smirnov test and employed visual inspection of histograms to assess the normality of continuous variables. Variables with a parametric distribution were expressed as the mean (standard deviation), and comparisons between groups were performed using the independent samples t-test. In cases where the data were found to be non-normally distributed, a logarithmic transformation was first applied, and the parametric test was subsequently employed if the data were found to be normally distributed following this transformation. For variables that did not follow a normal distribution, the Mann-Whitney U test served as the appropriate statistical tool. These variables were described using the median and minimum–maximum values. Categorical comparisons were conducted via chi-squared or Fisher's exact tests, depending on the distribution of data in contingency tables. Results were deemed statistically significant when the p-value was below 0.05.

RESULTS

This study involved a total of 58 pregnant women diagnosed with Intrahepatic Cholestasis of Pregnancy (ICP). Patients were categorized into two groups according to their serum bile acid levels at diagnosis: Group 1 (<40 µmol/L, n=41) and Group 2 (≥40 µmol/L, n=17). Maternal and perinatal characteristics of the two groups are presented in Table 1.

A statistically significant disparity was not observed between the groups with regard to maternal age, gravidity, parity, gestational age at diagnosis, gestational age at delivery, birth weight, or history of cholestasis in a previous pregnancy (p>0.05 for all). Alanine aminotransferase (ALT) levels were significantly higher in Group 2 than in Group 1 (197.2=127.6 vs. 121.2=121.3 U/L, p=0.045).

Although bile acid concentrations are presented as medians in Table 1 due to non-normal distribution, the mean (±SD) serum bile acid level for the entire cohort was 31.7=32.2 µmol/L. In subgroup analysis, the mean (±SD) values were 17.4=5.9 µmol/L in Group 1 and 66.0=42.9 µmol/L in Group 2.

Neonatal outcomes were compared between patients with bile acid levels <40 µmol/L (Group 1) and those with levels ≥40 µmol/L (Group 2). The rate of preterm birth was similar between Group 1 and Group 2 (48.8% vs. 47.1%, p=1.00). The rate of NICU admission was higher in Group 2 (70.6%) than in Group 1 (53.7%), but the difference was not statistically significant (p=0.23) (Table 2).

Neonatal jaundice was observed in 58.8% of Group 2 and 36.6% of Group 1 (p=0.15). Similarly, rates of tachypnea (36.6% vs. 29.4%, p=0.51) and respiratory distress syndrome (RDS) (9.8% vs. 11.8%, p=1.00) were comparable between groups, with no statistically significant differences observed in any of the evaluated neonatal outcomes (Table 2).

Neonatal acidemia was assessed using two different pH thresholds. When defined as a pH<7.20 (mild acidemia), it was observed in 14.6% of newborns in Group 1 and 17.6%

Table 1. Comparison of maternal and perinatal characteristics according to bile acid levels

	Group 1 (<40 µmol/L, n=41)	Group 2 (≥40 µmol/L, n=17)	p-value
Maternal age (years)	30.6 (5.4)	31.0 (7.0)	p=0.97*
Gravidity	3 (1-6)	2 (1-6)	p=0.51**
Parity	1 (0-4)	0 (0-3)	p=0.90**
ALT (U/L)	121.2±121.3	197.2±127.6	p=0.045***
AST (U/L)	82.6±92.4	168.3±185.2	p=0.085***
GA at diagnosis (weeks)	34 (26-38)	32 (18-38)	p=0.64**
Bile acid level at diagnosis (µmol/L)	18 (10.4-31.2)	52 (40-219)	p<0.001**
History of ICP in previous pregnancy (%)	7 (17.1)	3 (17.6)	p=0.95****
GA at delivery (weeks)	37 (27-39)	37 (32-40)	p=0.50**
Birth weight (g)	2842.3 (677.9)	2860.9 (628.5)	p=0.92***
Cesarean section (%)	22 (53.7)	8 (47.0)	p=0.48****

ALT: Alanine aminotransferase; AST = Aspartate aminotransferase; GA: Gestational age; ICP: Intrahepatic cholestasis of pregnancy; µmol/L: Micromoles per liter; g: grams. Data are presented as mean (SD) for parametric variables, median (min-max) for nonparametric variables, and frequency (percentage) for categorical variables. *Independent samples t-test after logarithmic transformation. **Mann-Whitney U test. ***Independent samples t-test. ****Chi-square test. p<0.05 was considered statistically significant.

Table 2. Neonatal outcomes according to bile acid groups

	Group 1<40 µmol/L (n=41)	Group 2 ≥40 µmol/L (n=17)	p-value
Preterm birth n (%)	20 (48.8)	8 (47.1)	1.00
NICU admission n (%)	22 (53.7)	12 (70.6)	0.23
Jaundice n (%)	15 (36.6)	10 (58.8)	0.15
Tachypnea n (%)	15 (36.6)	5 (29.4)	0.51
RDS n (%)	4 (9.8)	2 (11.8)	1.00
Mild acidemia (pH<7.20) n (%)	6 (14.6)	3 (17.6)	1.00
Severe acidemia (pH<7.10) n (%)	2 (4.9)	2 (11.8)	0.70

NICU: Neonatal Intensive Care Unit; RDS: Respiratory Distress Syndrome; n: Number of cases. Categorical variables are presented as frequency (percentage) and compared using the Chi-square test. p<0.05 is considered statistically significant. No statistically significant differences were found.

Table 3. Comparison of umbilical cord blood gas parameters according to bile acid levels

	Group 1<40 µmol/L (n=41)	Group 2 ≥40 µmol/L (n=17)	p-value
pH	7.30 (6.80-7.43)	7.28 (7.06-7.44)	0.797*
pCO_2 (mmHg)	48.91 (13.81)	50.33 (11.84)	0.591**
pO_2 (mmHg)	33.63 (11.17)	30.82 (11.88)	0.410***
HCO_3 (mmol/L)	20.80 (9-27.4)	21.60 (7-27.6)	0.885*
Base Excess (BE)	3.50 (0.1-13)	4.20 (0.3-9.3)	0.771*

pCO_2: Partial pressure of carbon dioxide; pO_2: Partial pressure of oxygen; HCO_3^-: Bicarbonate; BE: Base excess; SD: Standard deviation. Data are presented as mean (SD) for parametric variables, median (min–max) for nonparametric variables. *Mann-Whitney U test. **Independent samples t-test after logarithmic transformation. ***Independent samples t-test. p<0.05 was considered statistically significant.

in Group 2 (p=1.00). Using a stricter definition (pH<7.10, severe acidemia), the rates were 4.9% and 11.8% for Group 1 and Group 2, respectively (p=0.70). In both definitions, the differences between the bile acid groups were not statistically significant (Table 2).

The comparison of umbilical cord blood gas parameters between the two bile acid groups is presented in Table 3. No statistically significant differences were observed in any of the measured parameters, including pH, pCO_2, pO_2, bicarbonate (HCO_3^-), or base excess (BE), between Group 1 (<40 µmol/L) and Group 2 (≥40 µmol/L). Median cord blood pH values were 7.30 and 7.28 in Group 1 and Group 2, respectively (p=0.797). Similarly, other gas parameters showed comparable distributions across groups, suggesting no association between bile acid levels and cord blood gas status at birth.

Additionally, Pearson correlation analysis showed no statistically significant relationship between maternal bile acid levels and neonatal cord pH (r=–0.02, p=0.884).

DISCUSSION

Intrahepatic cholestasis of pregnancy is one of the most common liver-related disorders occurring during pregnancy. Serum bile acid levels are widely accepted as the primary parameter for both diagnosis and monitoring of the condition. In the evaluation of ICP severity, bile acid levels are classified as mild if between 10-40 µmol/L, moderate if between 40-100 µmol/L and severe if above 100 µmol/L.[5,6,9] In the present study, due to the distribution of the number of patients, the grouping was made in two categories: <40 µmol/L and ≥40 µmol/L. However, the limited number of patients with concentrations ≥100 µmol/L precluded triple classification and constrained the statistical power of subgroup analyses.

The impact of intrahepatic cholestasis on obstetric outcomes has been extensively documented in the extant literature. The most frequently reported complications include preterm labour, meconium-stained amniotic fluid, fetal distress, fetal asphyxia and intrauterine fetal death. These findings suggest that the disease may have serious effects on fetal well-being.[10,11]

In the study conducted by Brouwers et al.,[12] it was reported that bile acid may increase the risk of preterm labour by causing toxic effects on placental membranes. It has been hypothesised that this mechanism may be responsible for the early degeneration of amniotic membranes and the acceleration of cervical maturation. In our study, preterm labour occurred in 48.3% of 58 patients diagnosed with intrahepatic cholestasis. This rate is significantly higher compared with the frequency of preterm labour of approximately 11% reported in the general obstetric population.[13] The present study is consistent with earlier research[12,14] and demonstrates that intrahepatic cholestasis is a high-risk condition for preterm delivery. In a subgroup analysis, a higher rate of preterm delivery was observed in pregnant women with serum bile acid levels ≥40 µmol/L. However, this difference was not statistically significant. This may be attributable to the inability of the analyses to attain sufficient statistical power due to the constrained sample size.

The necessity for the utilisation of the Neonatal Intensive Care Unit (NICU) is a prevalent neonatal outcome in pregnancies accompanied by cholestasis and prematurity. The development of this condition is hypothesised to be facilitated by fetal distress and respiratory system problems. In the present study, the necessity for NICU was found to be elevated in the group with a serum bile acid level of 40 µmol/L and above. However, this discrepancy was not statistically significant.

In this study cohort, neonatal jaundice occurred in 44.6% of newborns, and tachypnea was identified in 35.7% of cases. While the incidence of jaundice was consistent with the overall neonatal population, the prevalence of tachypnea was found to be higher than the averages reported in existing studies.[15,16] However, later subgroup analyses did not show a statistically significant link between these neonatal complications and serum bile acid levels. Therefore, these results should be interpreted cautiously, and further research with larger sample sizes is needed to explore possible associations.

Another important issue pertaining to ICP is fetal acidosis and postnatal acid-base balance. In the present study, neonatal acidosis was objectively evaluated by umbilical

cord blood gas analysis. Acidemia, defined as a pH below 7.20, was identified in 15.5% of cases, with more severe acidemia (pH <7.10) present in 6.9%. However, no significant relationship was found between bile acid level and pH value. In a prospective case-control study, Futterman et al.[17] investigated troponin I levels to assess fetal myocardial injury potentially associated with sudden fetal death. Troponin I levels were found to be significantly elevated in the group with intrahepatic cholestasis of pregnancy. In their secondary analysis, cord blood pH and base excess were assessed to investigate neonatal acidosis. Consistent with our results, the study found no meaningful differences between the ICP and control groups. The results indicate that maternal bile acids might exert minimal influence on the acid-base balance of the fetus. It has been hypothesised that the protective effect of the placental barrier, fetal compensatory mechanisms, or treatments such as UDCA, may weaken this relationship.

Specifically, the placenta may limit the transplacental passage of toxic bile acids through active transport systems such as the bile salt export pump, thereby reducing fetal exposure.[18] Additionally, the fetal acid–base balance may be buffered by endogenous compensatory mechanisms including bicarbonate and protein buffering systems.[19] Recent investigations such as Tosun et al.[20] have explored endoplasmic reticulum stress–related biomarkers as novel predictors of placental dysfunction in ICP, providing new insights beyond conventional bile acid measurement. UDCA treatment, commonly used in intrahepatic cholestasis of pregnancy, has been reported to improve certain aspects of placental function, which may help support fetal well-being and contribute to a more stable intrauterine environment.[21] These factors, alone or in combination, may contribute to the lack of a clear association between maternal bile acid levels and neonatal acidosis observed in this study.

Clinically, our findings suggest that while bile acid levels remain a key diagnostic marker in ICP, they may not directly reflect the degree of fetal acid-base impairment. This suggests that fetal risk in cholestasis of pregnancy is multifactorial. In addition to bile acid concentration, many factors such as placental perfusion, maternal liver function, and response to treatment also contribute to fetal risks. Therefore, when assessing perinatal risk, bile acid measurement should be interpreted in conjunction with other maternal and fetal parameters.

The main limitations of our study are its small sample size, the low number of patients in the group with high bile acid levels, and its single-center, retrospective design. These factors may limit the generalizability of our findings and constrain our ability to establish causal relationships. In addition, due to the retrospective nature of the study, certain confounding variables such as genetic predisposition and medication compliance could not be fully controlled. The small number of patients with severe cholestasis also reduced the statistical power for subgroup analyses. Therefore, multicenter prospective studies with larger patient populations are needed to validate and expand upon these findings.

Conclusion

This study found no evidence to suggest that there is a significant association between maternal serum bile acid levels and neonatal acidemia. Although our findings suggest no direct relationship, the limited number of patients with high bile acid levels may have restricted the power to detect such an association. Therefore, larger-scale, prospective studies are needed to better clarify this potential relationship and its clinical implications.

Ethics Committee Approval

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of Kartal Dr. Lütfi Kırdar City Hospital (Date: 27.05.2025, Decision No: 2025/010.99/16/6).

Informed Consent

Not required due to the retrospective design of the study.

Peer-review

Externally peer-reviewed.

Authorship Contributions

Concept: A.Y., D.K.; Design: A.Y.; Supervision: D.K.; Funding: A.Y.; Materials: A.Y.; Data collection and/or processing: D.K.; Analysis and/or interpretation: D.K.; Literature search: A.Y., D.K.; Writing: A.Y., D.K.; Critical review: A.Y.

Conflict of Interest

None declared.

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Gebeliğin İntrahepatik Kolestazı ve Yenidoğan Asidemisi: Perinatal Sonuçların Retrospektif Analizi

Amaç: Çalışmamızın amacı intrahepatik kolestaz tanısı almış gebeliklerde yenidoğan asidemisi insidansını saptamak ve bunun açlık safra asidi düzeyleri ile ilişkisini değerlendirmektir.

Gereç ve Yöntem: Kurumumuz Kadın Hastalıkları ve Doğum Kliniği'nde Ocak 2020 ile Mart 2025 tarihleri arasında doğum yapan ve intrahepatik kolestaz (İHK) tanısı konan gebelerin dosyaları retrospektif olarak tarandı ve çalışmamıza dahil edildi. Anne ve yenidoğana ait veriler; demografik özellikler, laboratuvar değerleri, tedavi protokolleri, doğum bilgileri ve yenidoğan sonuçları hastane kayıtlarından toplanmıştır. İstatistiksel analizler SPSS 24.0 programı kullanılarak yapıldı.

Bulgular: Toplam 58 İHK tanılı gebe çalışmaya dahil edilmiştir. Hastalar, serum safra asidi düzeylerine göre <40 µmol/L (n=41) ve ≥40 µmol/L (n=17) olmak üzere iki gruba ayrılmıştır. Gruplar arasında maternal demografik özellikler, gebelik haftası, doğum ağırlığı ve önceki kolestaz öyküsü açısından anlamlı fark bulunmamıştır (p>0.05). Preterm doğum oranları her iki grupta benzer bulunmuştur (%48.8 vs. %47.1; p=1.000). Yenidoğan yoğun bakım ünitesine (YYBÜ) yatış oranı ≥40 µmol/L grubunda daha yüksek saptanmakla birlikte (%70.6 vs. %53.7), bu fark istatistiksel olarak anlamlı değildir (p=0.36). Yenidoğan sarılığı, takipne ve solunum sıkıntısı sendromu insidansları da gruplar arasında benzer bulunmuştur (p>0.05). Yenidoğan asidemisi (göbek kordonu pH <7.20 ya da <7.10 olarak tanımlanan olgular) her iki grupta benzer oranlarda gözlenmiş, gruplar arasında anlamlı fark saptanmamıştır. Ortalama pH değerleri <40 µmol/L grubunda 7.29±0.11≥40 µmol/L grubunda ise 7.28±0.11 olarak hesaplanmıştır (p=0.856). Maternal serum safra asidi düzeyi ile göbek kordonu pH değeri arasında anlamlı bir korelasyon bulunmamıştır (r=–0.02; p=0.884).

Sonuç: Gebelik kolestazı olan hasta grubunda, maternal safra asidi düzeyleri ile yenidoğan asidemisi arasında anlamlı bir ilişki saptanmamıştır. Bu ilişkinin daha iyi anlaşılabilmesi için ileriye dönük çalışmalara ihtiyaç vardır.

Anahtar Sözcükler: FGL-2 düzeyi; nonalkolik steatohepatit; prognostik önem.