ISSN: 2181-3337 | SCIENTISTS.UZ VOLUME 5 ISSUE 7 JULY 2026

MATERNAL PRE-PREGNANCY BODY MASS INDEX AMONG TERM NEWBORNS WITH PROLONGED JAUNDICE: CLINICAL AND LABORATORY FINDINGS

Xudayberganov M[1], Sharipova S[2]
Head of the Department of Pediatrics and Neonatology,
Candidate of Medical Sciences, Associate Professor[1]
Assistant, Department of Pediatrics and Neonatology, Urgench State Medical Institute[2]

DOI: 10.67620/sai.57d0007

ABSTRACT

Background: Prolonged neonatal jaundice requires careful assessment because persistent unconjugated hyperbilirubinemia must be distinguished from conjugated hyperbilirubinemia and hepatobiliary disease. Maternal overweight and obesity may alter maternal and neonatal metabolic conditions, but evidence regarding prolonged jaundice remains limited. Objective: To describe maternal body mass index distribution and clinical–laboratory findings among term newborns with prolonged jaundice. Methods: This clinical and laboratory study included 140 full-term newborns with jaundice persisting beyond 14 days and total bilirubin above 85 µmol/L. Newborns were grouped according to maternal pre-pregnancy BMI. Maternal and neonatal hematological and biochemical indicators were assessed, and Spearman correlations were calculated. Results: Of 140 newborns, 100 (71.4%) were male. Maternal BMI categories were overweight in 57 cases (40.7%), class I–II obesity in 21 (15.0%), underweight in 25 (17.9%), and normal weight in 37 (26.4%). Maternal AST was higher in the overweight and obesity groups than in the normal-BMI group. Mean neonatal biochemical values did not differ significantly across groups. Within the overweight group, platelet count correlated positively with direct bilirubin (ρ=0.63), ALT (ρ=0.95), and AST (ρ=0.79). Conclusion: Overweight or obesity was present in 55.7% of mothers in this case series. The observed laboratory correlations are exploratory and support structured hepatobiliary assessment rather than causal inference..

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Keywords: *prolonged neonatal jaundice; maternal body mass index; overweight; obesity; hyperbilirubinemia; neonatal cholestasis; liver enzymes.*

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1. Introduction

The neonatal period, defined as the first 28 days of life, remains the most vulnerable stage of childhood. Global estimates indicate that 2.3 million children died during the first month of life in 2024, corresponding to approximately 6,200 neonatal deaths each day, and neonatal deaths accounted for 47% of all deaths before five years of age [11,12]. These figures provide a broad public-health context for improving recognition and management of conditions that may initially appear common or benign but can occasionally signal serious disease.

Jaundice is among the most frequent clinical findings in newborns. More than 80% of newborn infants develop visible jaundice during the first weeks after birth, usually because bilirubin production is high and hepatic conjugation and excretion are still maturing [1]. Most neonatal jaundice is transient and predominantly unconjugated. Nevertheless, severe unconjugated hyperbilirubinemia can cause acute bilirubin encephalopathy or kernicterus, while persistent jaundice can be the first sign of hemolysis, endocrine disease, infection, inherited metabolic disease, biliary obstruction, or another hepatobiliary disorder [1–5]. For this reason, clinical appearance alone is insufficient; bilirubin values must be interpreted according to the infant's age, gestational maturity, clinical status, and the direct or conjugated fraction.

Prolonged neonatal jaundice is commonly defined as jaundice persisting beyond 14 days in a term infant. International guidance emphasizes that infants who remain jaundiced at or after two weeks should undergo fractionated bilirubin measurement so that cholestasis is not missed [2–5]. The 2017 joint NASPGHAN–ESPGHAN guideline states that a direct bilirubin concentration above 1.0 mg/dL (17 μmol/L) warrants timely evaluation and, when appropriate, referral to pediatric gastroenterology or hepatology [3]. Timely recognition is particularly important because some causes, including biliary atresia, require rapid diagnostic and surgical decision-making. More recent reviews likewise recommend a stepwise evaluation that combines history, physical examination, total and direct bilirubin, liver enzymes, coagulation measures, targeted imaging, and metabolic or genetic testing when indicated [4,5].

The determinants of neonatal hyperbilirubinemia are heterogeneous. A meta-analysis identified exclusive breastfeeding, glucose-6-phosphate dehydrogenase deficiency, maternal–fetal ABO incompatibility, and preterm birth as important factors [10]. Large cohort work has also shown that hemolytic and nonhemolytic jaundice have different maternal and pregnancy risk profiles, with maternal obesity among the factors associated with nonhemolytic neonatal jaundice [9]. These findings indicate that maternal characteristics can contribute to the clinical context of neonatal jaundice, although the strength and mechanism of each association may differ across populations.

Maternal overweight and obesity are increasingly common. The World Health Organization estimated that 2.5 billion adults were overweight in 2022, including more than 890 million living with obesity [13]. In pregnancy, excess adiposity is associated with insulin resistance, chronic low-grade inflammation, altered lipid metabolism, nonalcoholic fatty liver disease, gestational diabetes, hypertensive disorders, and changes in placental function. These maternal conditions may affect neonatal metabolic adaptation directly or through associated obstetric and feeding factors. Maternal BMI is therefore a clinically accessible characteristic that may help identify newborns who merit closer surveillance, but it should not be interpreted in isolation.

Several studies have examined maternal weight and bilirubin outcomes. Rougée and colleagues reported an association between obstetric obesity and higher maternal and neonatal unconjugated bilirubin concentrations in a multiethnic sample [6]. Özdek and colleagues prospectively observed higher bilirubin values on postnatal days 5 and 15 among infants whose mothers gained more weight than recommended during pregnancy [7]. In a retrospective study of 3,594 healthy term newborns, Domosud and Kulik-Rechberger assessed maternal pre-pregnancy BMI and gestational weight gain in relation to early jaundice [8]. Although designs and outcomes differ, these studies support further investigation of maternal metabolic status as one component of neonatal jaundice assessment.

Important distinctions are required when interpreting a sample composed only of infants who already have prolonged jaundice. Such a sample can describe the maternal BMI distribution among cases and compare clinical or laboratory patterns between BMI-defined groups. It cannot, however, estimate the incidence of prolonged jaundice among all infants born to overweight or obese mothers unless the number and characteristics of all births are known. It also cannot establish that maternal BMI caused the jaundice.

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The present study is therefore framed as a descriptive clinical and laboratory analysis of term newborns with prolonged jaundice rather than a population-based risk study.

The objective of this study was to describe the distribution of maternal pre-pregnancy BMI among full-term newborns with prolonged jaundice, compare the available maternal and neonatal laboratory findings across BMI groups, examine reported correlations within the overweight group, and formulate a cautious, guideline-informed approach to further hepatobiliary assessment.

2. Materials and Methods

2.1 Study design and participants

A clinical and laboratory study was conducted in 140 full-term newborns with prolonged jaundice. The available study record defined prolonged jaundice as persistence of visible jaundice after the 14th day of life together with a serum total bilirubin concentration above 85 μmol/L. All included infants were born at term. The sample comprised 100 boys (71.4%) and 40 girls (28.6%).

Maternal pre-pregnancy body mass index was used to classify the mother–infant pairs into four groups. Group 1 included 57 newborns born to overweight mothers; group 2 included 21 newborns born to mothers with class I–II obesity; group 3 included 25 newborns born to underweight mothers; and group 4 included 37 newborns born to mothers with normal body weight. The BMI thresholds applied by the investigators were not specified in the source record; for interpretation, the categories correspond to the conventional adult BMI group labels used in clinical practice.

Because all participants had prolonged jaundice, the study was treated as a case series with internal comparisons by maternal BMI. The design did not include a control group of infants without prolonged jaundice or information on the maternal BMI distribution among all deliveries. Accordingly, analyses of group proportions were interpreted descriptively and were not used to calculate relative risk, odds ratios, incidence, or population prevalence.

2.2 Clinical and laboratory assessment

Maternal assessment included complete blood count and biochemical blood testing. The recorded indicators were hemoglobin, erythrocyte count, platelet count, leukocyte count and differential, erythrocyte sedimentation rate, total protein, glucose, urea, creatinine, total bilirubin and bilirubin fractions, alanine aminotransferase (ALT), and aspartate aminotransferase (AST).

All newborns underwent complete blood count and biochemical testing. The neonatal biochemical panel included ALT, AST, total bilirubin, direct bilirubin, indirect bilirubin, urea, creatinine, calcium, total protein, and glucose. The study record did not provide assay manufacturers, analytical coefficients of variation, exact postnatal age at sampling, or reference intervals. Consequently, the present report focuses on the between-group patterns and correlations explicitly available in the source data rather than reconstructing unreported laboratory procedures.

For clinical interpretation, direct bilirubin was treated as the key screening measure for possible cholestasis, whereas ALT and AST were considered indicators of hepatocellular injury or stress rather than specific markers of biliary obstruction. Platelet count was interpreted as a nonspecific hematological measure that can vary with inflammatory, reactive, hematologic, and technical factors.

2.3 Statistical analysis

Absolute numbers and percentages were calculated for categorical variables. Continuous laboratory variables were summarized using means and the dispersion measures reported in the source manuscript. A two-sided significance threshold of $p<0.05$ was stated in the original analysis. Spearman rank correlation coefficients (ρ) were used to examine monotonic relationships among maternal BMI, neonatal bilirubin fractions, liver enzymes, and platelet count.

The correlation analysis within the overweight group was exploratory. Several pairwise correlations were examined, and no adjustment for multiple testing was reported. Therefore, the magnitude, direction,

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and clinical coherence of the correlations were considered alongside their p values. Correlation was not interpreted as evidence of causation, prediction, or a validated diagnostic threshold.

2.4 Ethical reporting

The source manuscript did not state the approving ethics committee, approval number, or the procedure used to obtain parental informed consent. These details must be supplied by the authors before submission to a journal that requires formal ethics reporting.

3. Results

3.1 Characteristics of the study sample

Among the 140 full-term newborns with prolonged jaundice, boys predominated, accounting for 100 cases (71.4%), while girls accounted for 40 cases (28.6%). The study record did not provide sex distributions for every maternal BMI group, although the overweight group contained 37 boys, representing 64.9% of that group.

The largest maternal BMI category was overweight: 57 newborns (40.7%) were born to overweight mothers. A further 21 infants (15.0%) were born to mothers with class I–II obesity. Thus, 78 of 140 infants (55.7%) in the case series had mothers classified as overweight or obese. Twenty-five infants (17.9%) were born to underweight mothers, and 37 (26.4%) were born to mothers with normal BMI. These percentages describe the composition of the prolonged-jaundice sample and should not be interpreted as the incidence of prolonged jaundice within each maternal BMI category.

Table 1. Study sample and distribution by maternal pre-pregnancy BMI

Characteristic / group	Definition in the study	n	% of total
Male newborns	Full-term newborns with prolonged jaundice	100	71.4
Female newborns	Full-term newborns with prolonged jaundice	40	28.6
Group 1	Maternal overweight	57	40.7
Group 2	Maternal class I–II obesity	21	15.0
Group 3	Maternal underweight	25	17.9
Group 4	Maternal normal BMI	37	26.4
Groups 1 + 2	Maternal overweight or obesity	78	55.7

Note. The table describes the composition of a case series. No denominator of all births was available for estimating BMI-specific incidence or risk.

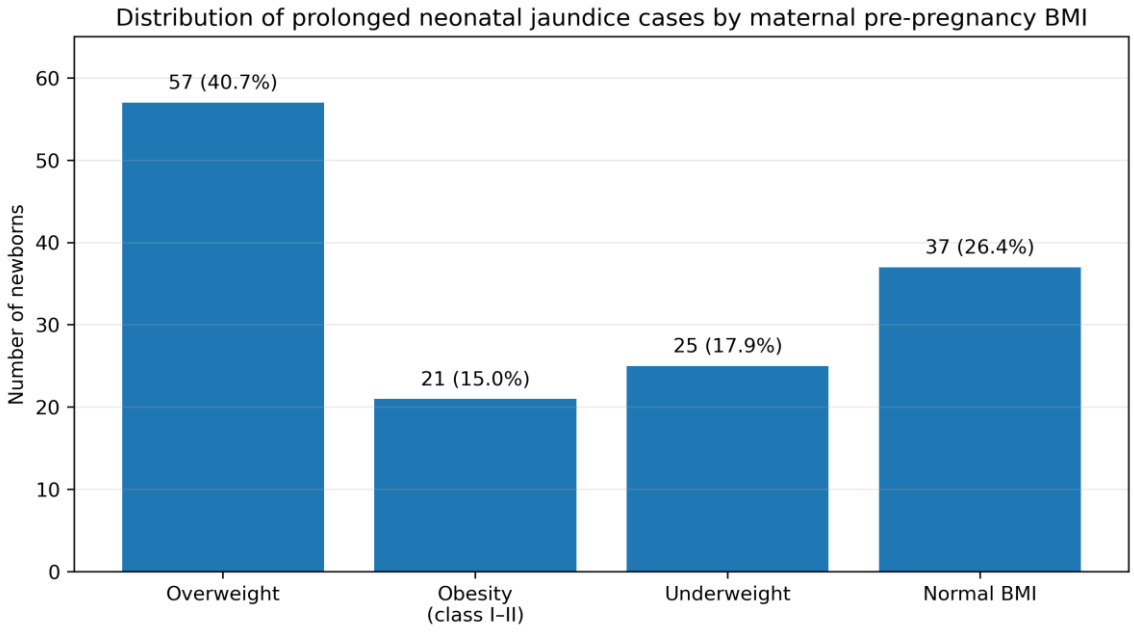


Chart 1. Distribution of prolonged neonatal jaundice cases by maternal pre-pregnancy BMI. Values are numbers and percentages of the 140 included newborns.

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3.2 Maternal laboratory findings

Most routinely measured maternal hematological indicators did not differ significantly between BMI groups in the source analysis. Lymphocyte measures were reported to vary among women with abnormal body weight, but the manuscript did not provide complete numerical estimates for each group. Without those estimates, the direction and clinical magnitude of the lymphocyte differences cannot be independently evaluated.

The clearest reported maternal biochemical difference involved AST. Mean AST activity was 35.3±5.6 U/L in mothers in the overweight group and 35.3±5.5 U/L in mothers with class I–II obesity. The corresponding mean among mothers with normal BMI was 17.7±1.4 U/L. The underweight-group value was not reported. The approximately twofold difference between the overweight/obesity groups and the normal-BMI group may be compatible with greater hepatic or metabolic stress, but AST is nonspecific and can be affected by hepatic, muscular, hemolytic, and pre-analytical factors. The finding therefore requires interpretation with ALT, clinical history, and other liver-related measures.

Table 2. Available maternal AST results by pre-pregnancy BMI group

Maternal BMI group	n	AST, mean ± reported dispersion (U/L)
Overweight	57	35.3 ± 5.6
Class I–II obesity	21	35.3 ± 5.5
Underweight	25	Not reported
Normal BMI	37	17.7 ± 1.4

Note. The source record did not specify whether the dispersion measure was a standard deviation or standard error and did not provide the underweight-group AST estimate.

3.3 Neonatal biochemical findings

The analysis reported no statistically significant between-group differences in neonatal ALT, AST, total bilirubin, direct bilirubin, indirect bilirubin, urea, creatinine, calcium, glucose, or total protein. This indicates that group means for the measured neonatal biochemical variables were broadly similar despite differences in maternal BMI category. It does not exclude clinically important abnormalities in individual infants, nor does it show equivalence between groups because equivalence testing and confidence intervals for group differences were not reported.

The absence of mean differences is relevant when interpreting the subsequent correlation analysis. A correlation observed within one BMI-defined group can coexist with similar group averages, because correlation concerns the joint ordering of two variables within individuals rather than the difference between group means. The results therefore address two distinct questions: whether average laboratory values differed by maternal BMI group, and whether specific neonatal markers moved together within the overweight group.

3.4 Correlation analysis in the maternal-overweight group

Within the 57 mother–infant pairs in the overweight group, maternal BMI showed only weak or negligible correlations with neonatal biochemical indicators. The reported coefficients were $\rho=0.166$ for maternal BMI and total bilirubin, $\rho=0.131$ for maternal BMI and direct bilirubin, $\rho=-0.093$ for maternal BMI and indirect bilirubin, $\rho=0.014$ for maternal BMI and ALT, and $\rho=0.052$ for maternal BMI and AST. These values do not support a strong monotonic relationship between incremental BMI variation within the overweight category and the neonatal bilirubin or aminotransferase measures.

By contrast, neonatal platelet count was positively correlated with direct bilirubin ($\rho=0.63$), ALT ($\rho=0.95$), and AST ($\rho=0.79$); the source analysis reported $p<0.001$ for these three associations. Strong positive correlations were also reported among biochemical indicators: total bilirubin with direct bilirubin ($\rho=0.77$), direct bilirubin with ALT ($\rho=0.75$), direct bilirubin with AST ($\rho=0.62$), and ALT with AST ($\rho=0.84$). These findings describe a cluster of laboratory values that increased together among infants in the overweight-maternal-BMI group.

The very high platelet–ALT coefficient ($\rho=0.95$) deserves particular caution. Such a large correlation may represent a strong biological pattern, but it can also arise from a restricted sample, influential observations, duplicated measurements, data-entry errors, or a shared relationship with another

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unmeasured factor. A scatterplot review, influence analysis, and replication in an independent sample would be necessary before platelet count could be proposed as a diagnostic or prognostic marker.

Table 3. Reported Spearman correlations within the overweight maternal BMI group (n=57)

Variable 1	Variable 2	Spearman ρ	Reported significance
Maternal BMI	Total bilirubin	0.166	Not reported
Maternal BMI	Direct bilirubin	0.131	Not reported
Maternal BMI	Indirect bilirubin	−0.093	Not reported
Maternal BMI	ALT	0.014	Not reported
Maternal BMI	AST	0.052	Not reported
Platelet count	Direct bilirubin	0.63	p<0.001
Platelet count	ALT	0.95	p<0.001
Platelet count	AST	0.79	p<0.001
Total bilirubin	Direct bilirubin	0.77	Not reported
Direct bilirubin	ALT	0.75	Not reported
Direct bilirubin	AST	0.62	Not reported
ALT	AST	0.84	Not reported

Note. ALT=alanine aminotransferase; AST=aspartate aminotransferase. The source analysis did not report multiplicity-adjusted p values or confidence intervals for the correlations.

4. Discussion

This study describes maternal BMI categories and selected clinical–laboratory findings in 140 full-term newborns with prolonged jaundice. The largest single maternal BMI group was overweight, and overweight plus class I–II obesity accounted for 55.7% of the cases. Maternal AST values were higher in the overweight and obesity groups than in the normal-BMI group. Neonatal mean biochemical indicators did not differ significantly by maternal BMI category, but a set of strong within-group correlations was observed among platelet count, direct bilirubin, ALT, and AST in infants born to overweight mothers.

The case-series design is central to interpretation. The proportion of cases born to overweight or obese mothers cannot be compared with the proportion among all mothers who delivered during the same period because that denominator was not available. If overweight and obesity were similarly common in the underlying obstetric population, the observed 55.7% might simply reflect background maternal BMI distribution. Conversely, if those categories were uncommon among all deliveries, the case proportion might suggest overrepresentation. Without a non-jaundiced comparison group or population denominator, neither inference can be made reliably. The correct conclusion is therefore that overweight or obesity was common among mothers in this sample, not that it increased the incidence of prolonged jaundice by a quantified amount.

The finding remains clinically relevant because previous studies provide a plausible basis for considering maternal weight status during neonatal assessment. Rougée et al. found that obstetric obesity was associated with higher maternal and neonatal unconjugated bilirubin concentrations and proposed reduced UGT1A1-mediated bilirubin clearance as a possible mechanism [6]. Özdek et al. reported higher postnatal bilirubin concentrations among term infants whose mothers gained more weight than recommended during pregnancy [7]. Domosud and Kulik-Rechberger also examined pre-pregnancy BMI and gestational weight gain in a large population of healthy term newborns [8]. A population-based analysis of hemolytic and nonhemolytic jaundice identified maternal obesity among the strongest maternal factors for nonhemolytic jaundice [9]. Together, these studies suggest that maternal adiposity or excessive gestational weight gain may be associated with bilirubin outcomes, while also showing that neonatal jaundice is multifactorial.

Several pathways could connect maternal metabolic status with neonatal jaundice, but the present data cannot distinguish among them. Maternal obesity is associated with insulin resistance, inflammation, altered placental nutrient transfer, higher rates of diabetes and operative delivery, and changes in breastfeeding initiation or neonatal weight loss. Each of these could influence bilirubin production, enterohepatic circulation, hydration, feeding, or hepatic conjugation. Maternal liver enzyme elevation

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could reflect steatotic liver disease or another source of metabolic stress, but AST alone is not specific. Mechanistic claims should therefore remain hypotheses until supported by better-characterized maternal metabolic, obstetric, placental, and neonatal data.

The elevated maternal AST values in the overweight and obesity groups were one of the clearest numerical differences. Their magnitude was approximately twice the reported mean in the normal-BMI group. Nevertheless, the clinical meaning is uncertain because complete ALT values, AST reference intervals, the dispersion measure, medication history, muscle injury, hemolysis indices, and diagnoses such as fatty liver disease were not provided. The result supports attention to maternal liver-related findings but does not establish maternal hepatic dysfunction or prove a pathway to neonatal jaundice.

The neonatal group means did not differ significantly across maternal BMI categories. This finding argues against a simple model in which maternal overweight or obesity uniformly increases the average bilirubin concentration or aminotransferase activity among all affected infants. It is possible that maternal BMI influences only a subgroup, that associated factors rather than BMI itself are responsible, or that the sample lacked power to detect modest differences. It is also possible that once prolonged jaundice is present, the range of neonatal laboratory values is driven more strongly by the infant's immediate clinical condition than by maternal BMI category.

The correlation cluster involving direct bilirubin, ALT, AST, and platelet count is potentially important but should be treated as exploratory. Direct bilirubin is relevant because conjugated hyperbilirubinemia is a marker of cholestasis and warrants prompt evaluation [3–5]. ALT and AST may rise with hepatocellular injury, although they do not identify the cause or severity of cholestasis by themselves. A positive relationship among these indicators can therefore be clinically coherent. Platelet count, however, is not a specific marker of neonatal cholestasis. Reactive thrombocytosis may occur with inflammation or recovery from illness, while thrombocytopenia can accompany severe liver disease, infection, or portal hypertension. The direction of the association may depend on age, disease stage, and underlying diagnosis.

The platelet–ALT correlation of $\rho=0.95$ is unusually high for independent clinical laboratory measurements. Before this association is used to support a screening algorithm, the raw data should be reviewed for outliers, repeated values, transcription errors, nonlinearity, and restricted ranges. Confidence intervals should be calculated, and the analysis should be repeated after robust or influence-sensitive checks. Because several correlations were tested, multiplicity should also be considered. Replication in a larger, prospectively enrolled sample is essential.

Clinical care should continue to follow established prolonged-jaundice pathways regardless of maternal BMI. NICE guidance recommends assessment of prolonged jaundice and measurement of conjugated bilirubin, with further tests guided by clinical suspicion [2]. The NASPGHAN–ESPGHAN guideline emphasizes total and direct bilirubin measurement after two weeks and timely specialist evaluation when direct bilirubin is elevated [3]. Contemporary reviews recommend assessment of stool and urine color, feeding and growth, hepatosplenomegaly, bleeding, infection, endocrine disease, metabolic disorders, and biliary obstruction, followed by targeted laboratory and imaging investigations [4,5]. Maternal overweight or obesity can be used as contextual information that may lower the threshold for careful review, but it should not replace these standard steps.

Figure 1 presents a practical workflow based on the study's focus and current guidance. It begins with clinical confirmation of persistent jaundice and proceeds to fractionated bilirubin and core laboratory assessment. Infants without conjugated hyperbilirubinemia or clinical red flags may be managed according to the likely benign or noncholestatic cause with planned follow-up. Infants with elevated direct bilirubin, pale stools, dark urine, hepatomegaly, poor growth, coagulopathy, infection signs, or other red flags require urgent hepatobiliary investigation and referral. The workflow is a proposed clinical organization tool and was not prospectively validated in this study.

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The study has several strengths. It includes a defined sample of 140 full-term newborns with prolonged jaundice, applies the same maternal BMI grouping across the cohort, assesses both maternal and neonatal laboratory parameters, and examines bilirubin fractions rather than total bilirubin alone. The reporting of correlation coefficients provides information about relationships that may be obscured by group means. The study also addresses a clinically relevant question in a setting where maternal metabolic disorders are becoming more common.

The limitations are substantial. First, the case-only design prevents estimation of risk, incidence, or causal effects. Second, recruitment dates, clinical setting, sampling method, and exclusion criteria were not reported in the available record. Third, the analysis did not account for major neonatal jaundice determinants such as ABO or Rh incompatibility, glucose-6-phosphate dehydrogenase deficiency, breastfeeding pattern, postnatal weight loss, cephalohematoma, infection, thyroid disease, phototherapy, delivery mode, or age at laboratory sampling. Fourth, maternal gestational weight gain, diabetes, hypertension, and detailed liver assessment were not included in the reported analysis. Fifth, complete numerical results and confidence intervals were unavailable for many laboratory comparisons. Sixth, the exploratory correlation analysis involved multiple tests without reported adjustment, and the very high coefficients require raw-data verification. Finally, ethics approval and consent information must be added before submission.

Future research should use a prospective cohort design that enrolls all eligible term births and follows infants beyond 14 days. Maternal pre-pregnancy BMI and gestational weight gain should be measured using standardized definitions. Important confounders should be recorded, including maternal diabetes, hypertension, medications, delivery mode, infant sex, gestational age, birth weight, bruising, blood-group incompatibility, G6PD status, feeding, weight loss, and treatment. The primary outcome should distinguish prolonged unconjugated jaundice from conjugated hyperbilirubinemia or confirmed cholestasis. Multivariable models could then estimate whether maternal BMI independently predicts prolonged jaundice after adjustment for these factors. External validation would be required before any maternal-BMI-based clinical score is used.

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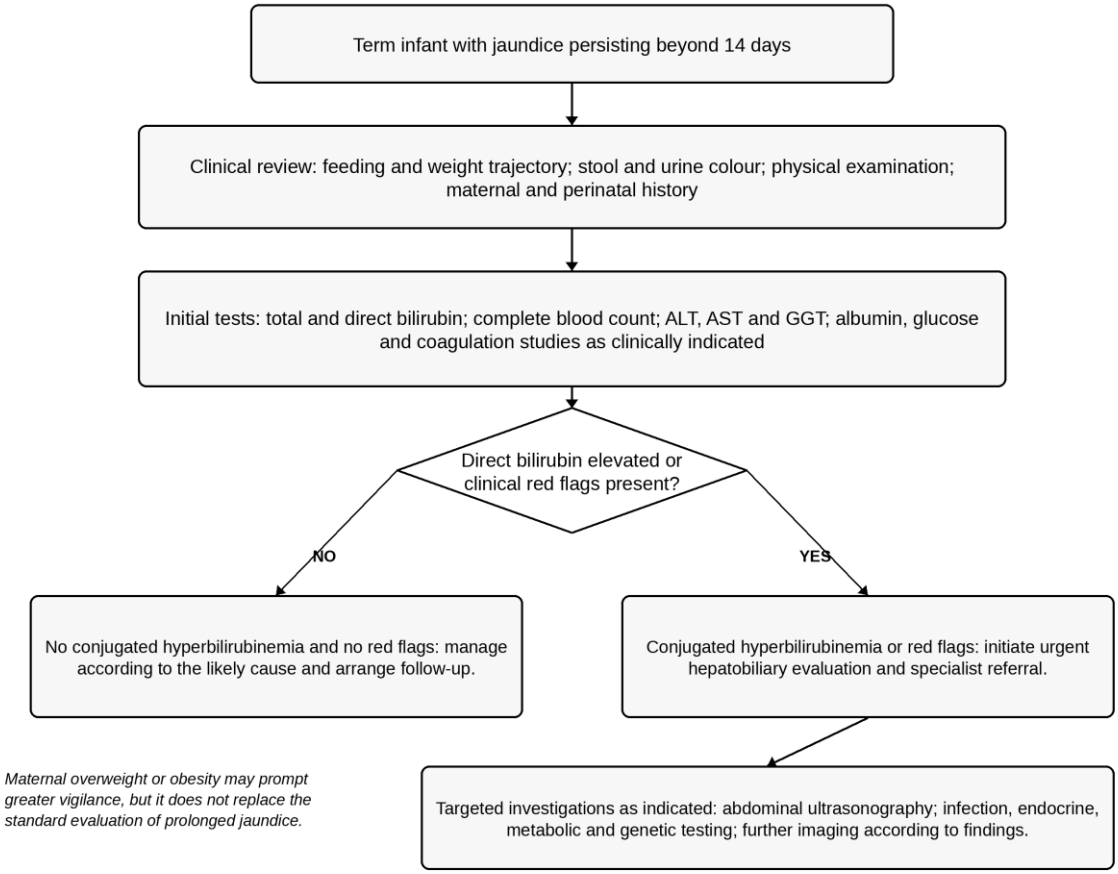


Figure 1. Guideline-informed clinical evaluation pathway for a term infant with prolonged jaundice. The pathway is an original synthesis for this manuscript and was not validated as a diagnostic algorithm in the present study.

Table 4. Interpretation of the main findings and appropriate level of inference

Observed finding	Supported interpretation	Interpretation not supported by the design
55.7% of cases had mothers with overweight or obesity	Overweight/obesity was common among mothers in this case series	Maternal overweight/obesity increased population incidence or caused prolonged jaundice
Maternal AST was higher in overweight and obesity groups	Maternal BMI groups differed in reported AST values	AST proves fatty liver disease or a causal maternal–neonatal mechanism
No significant differences in neonatal group means	Average measured neonatal biochemical values were similar across BMI groups	The groups are clinically equivalent or individual abnormalities are absent
Strong platelet, bilirubin, ALT, and AST correlations	Markers co-varied within the overweight group	Platelet count is a validated diagnostic biomarker or predictor of cholestasis

5. Conclusion

Among 140 full-term newborns with prolonged jaundice, 40.7% were born to overweight mothers and 15.0% to mothers with class I–II obesity; together these groups accounted for 55.7% of the case series. Maternal AST was higher in the overweight and obesity groups than in the normal-BMI group, whereas the available neonatal biochemical means did not differ significantly across maternal BMI categories.

Within the overweight group, platelet count showed strong positive correlations with direct bilirubin, ALT, and AST, and several bilirubin and aminotransferase measures were also strongly correlated. These findings may indicate a coordinated laboratory pattern in a subgroup of infants, but they remain exploratory and require raw-data verification, adjustment for confounding, and independent replication.

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Maternal BMI can be considered contextual information during assessment of prolonged neonatal jaundice, but the present design does not establish increased risk or causality. Every term infant with jaundice persisting beyond 14 days should receive clinically appropriate assessment, including fractionated bilirubin and timely evaluation for cholestasis when direct bilirubin is elevated or red flags are present.

Declarations

Ethics approval and consent to participate. The approving committee, approval number, and parental consent procedure were not included in the source manuscript and must be supplied by the authors before submission.

Conflict of interest. The authors should insert their conflict-of-interest declaration.

Funding. The authors should state whether the study received specific funding.

Data availability. The authors should state whether de-identified data are available and under what conditions.

Author contributions. The authors should provide contribution statements that reflect their actual roles.

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