JMU
Jurnal Medika Udayana

ISSN: 2597-8012 JURNAL MEDIKA UDAYANA, VOL. 15 NO.3, MARET, 2026

Received: 2026-01-12 Revision: 2026-01-30 Accepted: 30-02-2026

THE CORRELATION BETWEEN BILIRUBIN LEVELS AND BIRTH WEIGHT IN NEONATES WITH NEONATAL JAUNDICE

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ABSTRACT

Background: Neonatal jaundice is a clinical condition caused by elevated bilirubin levels. Birth weight is often associated with the risk of neonatal jaundice due to liver function immaturity in low birth weight infants. However, research findings regarding the relationship between these two variables remain varied. This study aims to determine the relationship between total serum bilirubin levels and birth weight in neonates with neonatal jaundice at Ngoerah Hospital.
Methods: This research is an observational analytic study with a cross-sectional approach using medical record data of patients who met the inclusion criteria at Ngoerah Hospital.
Results: Descriptive analysis results showed that the mean birth weight of the sample was 2,859.38 grams and the mean total bilirubin level was 17.77 mg/dL. The Spearman bivariate test results showed no significant relationship between birth weight and total bilirubin levels (p=0.222; r=0.198). Multivariate analysis also confirmed that birth weight was not a significant predictor (p=0.318) after controlling for gestational age and gender variables.
Conclusions: There is no significant relationship between birth weight and total bilirubin levels in neonates with neonatal jaundice at Ngoerah Hospital. This result is likely influenced by the dominance of term infants in the sample and the presence of other, more dominant pathological factors.

Keyword : Neonatal jaundice, birth weight, total bilirubin levels, neonates.

INTRODUCTION

Neonatal jaundice is a clinical manifestation frequently encountered in neonates, characterized by yellow discoloration of the skin, sclera, and mucous membranes caused by elevated bilirubin levels in the blood. This condition is experienced by more than 50% of term infants and over 80% of preterm infants during the first week of life.[1] Globally, the Southeast Asia region ranks second after Africa in terms of the prevalence of neonatal jaundice.[2] Excessive accumulation of bilirubin, particularly the lipophilic unconjugated form, poses a significant health risk as it can penetrate the blood-brain barrier. This process potentially leads to bilirubin encephalopathy or kernicterus, resulting in permanent neurological damage such as hearing impairment, cerebral palsy, or mental retardation.[3]

Birth weight serves as a crucial indicator of neonatal health status. Infants with low birth weight (LBW) face a mortality risk up to 20 times higher compared to infants with normal birth weight.[4] Furthermore, LBW is associated with an increased risk of neonatal jaundice due to hepatic immaturity and diminished activity of the uridine diphosphate-glucuronosyltransferase (UGT1A1) enzyme.[5] This enzymatic limitation hampers the conjugation process, leading to the accumulation of unconjugated bilirubin in the blood. Conversely, infants with normal birth weight generally possess a more mature metabolic capacity, allowing for more efficient conjugation and excretion of bilirubin.

Previous research has identified a correlation between birth weight and total bilirubin levels; however, findings remain inconsistent across different regions. According to certain research, newborns with low birth weight are far more likely than those with normal birth weight to get neonatal jaundice.[6] However, other studies suggest that this relationship may be influenced or confounded by other factors such as gestational age, hemolysis, and sepsis. The variability in these findings indicates that birth weight might not act as a single determinant but rather interacts with various physiological and pathological factors.

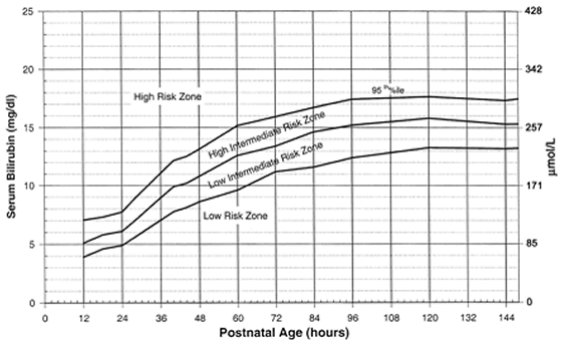
Given the variation in empirical evidence and the potential for severe complications if left unmanaged, there is a necessity for robust local data to strengthen the scientific basis for prevention, early detection, and management of neonatal jaundice in Indonesia. Therefore, further research is needed to investigate the specific relationship between these variables. This study aims to determine the relationship between total serum bilirubin levels and birth weight among neonates diagnosed with neonatal jaundice at Ngoerah Hospital.

LITERATURE REVIEW

Neonatal Jaundice

Neonatal jaundice is a common clinical manifestation in neonates, characterized by yellow coloring of the skin, sclera, and mucous membranes caused by high amounts of bilirubin in the blood.[7] This illness is primarily caused by a buildup of unconjugated bilirubin, a yellow pigment generated by heme catabolism, specifically erythrocyte breakdown.[8] Clinically, jaundice occurs when total serum bilirubin (TSB) levels surpass 5-7 mg/dL, with a cephalocaudal distribution.[9] During the first week of life, up to 80% of preterm infants and more than half of term infants suffer from this prevalent illness.[1]

Neonatal jaundice is classified into physiological and pathological types based on its onset, duration, and severity.[10] Physiological jaundice is a non-pathological condition arising from a temporary imbalance between bilirubin production and elimination, often due to hepatic immaturity.[9] In term infants, it usually appears after the first 24 hours of life, peaks between the third and fifth days, and goes away on its own in 7 to 10 days.[7,11] On the other hand, pathological jaundice, which is defined by the onset of jaundice during the first 24 hours, a sharp rise in bilirubin levels (>5 mg/dL daily), or levels beyond the 95th percentile on the hour-specific nomogram, suggests an underlying illness.[12]



Gambar 1. Bhutani nomogram, an hour-specific nomogram for neonatal jaundice[13]

Major etiologies include hemolysis due to ABO or Rh incompatibility, G6PD deficiency, and sepsis.[12] The buildup of lipophilic unconjugated bilirubin can cross the blood-brain barrier and produce acute bilirubin encephalopathy or its chronic variant, kernicterus, which results in irreversible brain damage, if treatment is not received.[9]

Bilirubin Metabolism

The metabolism of bilirubin involves a complex series of steps starting from the breakdown of heme. In neonates, bilirubin production is approximately twice as high as in adults due to a shorter erythrocyte lifespan and a larger red blood cell mass. The heme oxygenase enzyme starts the process by breaking down heme into biliverdin, which is then reduced to unconjugated bilirubin. Because this type of bilirubin is hydrophobic and lipophilic, it must bind to albumin reversibly in order to be carried by the blood to the liver and avoid accumulating in tissues like the brain.[14,15]

Upon reaching the liver, bilirubin dissociates from albumin and enters the hepatocytes, where it undergoes conjugation catalyzed by the enzyme UGT1A1.[14,15] Water-soluble conjugated bilirubin, which is subsequently eliminated into the bile, is created by this process from poisonous unconjugated bilirubin. However, in neonates, this system is often inefficient due to immature UGT1A1 activity, which is only about 0.1–1% of adult levels.[16] Furthermore, once in the intestine, conjugated bilirubin can be deconjugated back into its unconjugated form by the enzyme beta-glucuronidase, which is highly active in neonates.[17,18] This allows bilirubin to be reabsorbed into the circulation, a process known as enterohepatic circulation, significantly contributing to the bilirubin load in newborns.

Bilirubin Level

In neonates, bilirubin production is approximately twice as high per kilogram of body weight compared to adults, primarily due to a shorter erythrocyte lifespan and a larger red blood cell mass.[19] Coupled with low activity of hepatic conjugation enzymes, this leads to the common phenomenon of physiological jaundice. In healthy term infants, jaundice typically appears 24–48 hours after birth, peaks between days 2 and 4, and subsides within 2 to 3 weeks.[9] Clinically, jaundice becomes visible on the skin and sclera when TSB levels reach approximately 5–7 mg/dL.

While most cases are physiological and benign, specific thresholds determine the need for intervention. According to American Academy of Pediatrics (AAP) guidelines, TSB levels up to 17–18mg/dL may be acceptable in healthy term infants without pathological signs.[9] On the other hand, a sharp increase in bilirubin (>5 mg/dL daily) denotes pathological jaundice that has to be assessed right away. Risk assessment is often performed using the Bhutani Nomogram, which maps bilirubin levels against the infant's age in hours to categorize them into low, intermediate, or high-risk zones.[9] Infants in the high-risk zone (above the 95th percentile) have a significant probability of developing severe neonatal jaundice and potential bilirubin encephalopathy, necessitating close monitoring or phototherapy.

Birth Weight

Birth weight is a fundamental anthropometric indicator used to assess the health status and maturity of a neonate. The World Health Organization (WHO) divides newborns into four groups according to their birth weight: normal birth weight (ranging from 2,500 to 3,999 grams), low birth weight (LBW: less than 2,500 grams), very low birth weight (VLBW: between 1,000 and 1,499 grams), and extremely low birth weight (ELBW: below 1,000 grams).[20] These

classifications are crucial for identifying infants vulnerable to neonatal complications.

Infants with LBW face significantly higher health risks compared to those with normal birth weight, including a neonatal mortality risk that is up to 20 times greater.[4] Physiological immaturity in LBW infants predisposes them to various complications such as asphyxia, hypoglycemia, and hypothermia. In the context of bilirubin metabolism, LBW is often associated with hepatic immaturity, leading to reduced conjugation capacity and a higher susceptibility to neonatal jaundice.[5]

The Relationship between Birth Weight and Neonatal Jaundice

The correlation between birth weight and bilirubin levels has been extensively studied, yet findings remain varied across different populations. A significant body of evidence suggests that LBW is a strong risk factor for neonatal jaundice. For instance, observational studies in Indonesia have reported that LBW infants have a significantly higher risk (OR ranging from 2.34 to 9.7) of developing neonatal jaundice compared to normal weight infants.[21,22] This association is theoretically supported by the fact that lower birth weight often correlates with organ immaturity, including the liver's limited ability to synthesize the UGT1A1 enzyme required for bilirubin conjugation.

However, there is contradicting research that suggests birth weight is not a significant independent predictor. According to certain research, gestational age frequently complicates the connection between birth weight and jaundice.[23,24] Birth weight frequently acts as a proxy for prematurity; thus, when gestational age is controlled in multivariate analyses, the statistical significance of birth weight often diminishes. Additionally, the "masking effect" of dominant pathological factors can obscure the relationship. In cases involving severe hemolysis (such as ABO incompatibility) or sepsis, the excessive production of bilirubin overwhelms the liver's capacity regardless of the infant's weight, rendering birth weight a non-significant factor in those specific cohorts.[25]

Methodological Contextualization of Confounding Variables

The relationship between birth weight and bilirubin levels is rarely a direct cause-and-effect interaction; rather, it is frequently obscured by various confounding variables that influence both birth weight and bilirubin metabolism. Methodologically, birth weight often acts as an intermediate variable for physiological immaturity, which is more accurately defined by gestational age. Although often co-occurring with LBW, gestational age is consistently identified as a stronger independent predictor of neonatal jaundice because it directly reflects the functional maturation of hepatic UGT1A1 enzyme capacity.[26,27] The inability of the liver to function optimally, whether due to structural changes as seen in chronic conditions or developmental immaturity, significantly impacts metabolic clearance.[28] Consequently, in multivariate models, the statistical significance of birth weight often diminishes once gestational age is included, suggesting that LBW frequently serves merely as a proxy for prematurity.[29]

Beyond physiological maturity, pathological and maternal factors also play a substantial role in modulating bilirubin levels independent of birth weight. Regardless of the infant's weight, hemolytic disorders including ABO incompatibility and glucose-6-phosphate dehydrogenase (G6PD) deficiency can significantly increase bilirubin generation by erythrocyte destruction, exceeding hepatic conjugation capability.[26] Additionally, maternal metabolic disorders like gestational diabetes can lead to fetal polycythemia and subsequent neonatal jaundice, while birth trauma (e.g., cephalohematoma) and specific delivery methods can increase the bilirubin load through extravascular blood degradation and enhanced enterohepatic circulation.[3,30,31]

Post-Natal Functional Factors

Following birth, the neonate’s physiological environment changes drastically, introducing functional factors such as sepsis, nutritional status, and microbiome development that further influence bilirubin metabolism. Neonatal sepsis is a potent confounder that exacerbates neonatal jaundice through multiple pathways, including non-immune hemolysis caused by bacterial toxins, hepatocellular dysfunction, and increased permeability of the blood-brain barrier.[32] This systemic inflammation increases the susceptibility of the brain to bilirubin toxicity, making sepsis a critical neurotoxicity risk factor even at lower serum bilirubin levels.[33]

Concurrent with infection risks, early nutritional intake is a critical determinant of bilirubin dynamics. Neonates experiencing significant weight loss (>7–10%) in the first few days of life, often due to inadequate breastfeeding, are at higher risk of jaundice.[26] Low caloric intake delays the passage of meconium, which is rich in bilirubin, thereby facilitating the reabsorption of unconjugated bilirubin via the enterohepatic circulation. This factor often interacts with birth weight, as LBW infants may exhibit weaker sucking reflexes, creating a dual confounding effect involving birth weight, nutritional intake, and bilirubin levels.

MATERIALS AND METHOD

This study examined the association between total serum bilirubin levels and birth weight using a quantitative analytical approach and a cross-sectional design. The research was conducted at RS Ngoerah (Prof. Dr. I.G.N.G. Ngoerah Hospital) in Denpasar. Secondary data was gathered from medical records between January and December of 2024.

This study included infants diagnosed with neonatal jaundice at RS Ngoerah. The whole population that fulfilled the eligibility requirements was included in the total sampling technique. Neonates with neonatal jaundice, term newborns (born at full term), and infants less than seven days were the inclusion criteria. Patients with incomplete medical record data met the exclusion criteria. Forty neonates in all were selected for analysis based on these criteria. Individuals with incomplete or absent medical records were not included. Forty neonates were chosen for investigation based on these criteria.

The variables in this study consisted of the independent variable, birth weight (measured in grams), and the dependent variable, total serum bilirubin levels (measured in mg/dL). Additionally, gestational age and gender were considered as confounding variables in the multivariate analysis. A structured checklist was used to retrieve data from medical records, and Microsoft Excel and the Statistical Package for the Social Sciences (SPSS) program were used for processing..

There were multiple phases to the data analyzing process. The mean, median, and standard deviation of the variables were described using univariate analysis. The Shapiro-Wilk test was used to test for normalcy because the sample size was smaller than 50 (N=40). The non-parametric Spearman's Rank Correlation test was used for bivariate analysis to ascertain the link between birth weight and bilirubin levels because the bilirubin data did not distribute normally ($p<0.001$). Furthermore, multivariate analysis using Linear Regression was performed after transforming the dependent variable (Log_TSB) to meet normality assumptions, controlling for gestational age and gender.

This study has been approved by the Research Ethics Committee of the Faculty of Medicine, Udayana University, with the Ethical Exemption Letter Number 1240/UN14.2.2.VII.14/LT/2025 and Protocol Number 2025.01.1.0467.

RESULT

The study included a total sample of 40 neonates diagnosed with neonatal jaundice at RS Ngoerah. A descriptive analysis was conducted to outline the demographic and clinical characteristics of the subjects, including gestational age (GA), birth weight (BW), and total serum bilirubin levels (TSB).

Table 1. Characteristics of Research Subjects (N=40)

Variable	Mean ± SD	Median	Min - Max
GA (weeks)	37.80 ± 0.85	38	37 - 39
BW (g)	2,859.38 ± 524.33	2,895.0 0	1,760 - 3,900
TSB (mg/dL)	17.77±7.91	16.75	7.3 - 55.1

As presented in Table 1, the mean gestational age of the subjects was 37.80 ± 0.85 weeks, with a range of 37 to 39 weeks. This indicates that the sample predominantly consisted of term infants, fulfilling the study's inclusion criteria. The birth weight of the neonates showed significant variation, with a mean of 2,859.38 ± 524.33 grams. The lowest recorded birth weight was 1,760 grams, while the highest was 3,900 grams.

A total of 40 subjects were included in the study sample. Upon examining the demographic characteristics, the gender distribution within this cohort was observed to be relatively balanced. The group consisted of 19 male neonates, representing 47.5% of the total sample, and 21 female neonates, who made up the remaining 52.5%. With regard to the clinical findings, the primary dependent variable analyzed was the Total Serum Bilirubin (TSB). The results demonstrated a remarkably wide range of values across the subjects, indicating significant variability within the population. Specifically, the TSB concentrations spanned from a minimum observed value of 7.3 mg/dL to a maximum peak level of 55.1 mg/dL. Consequently, the mean TSB level for the entire group was recorded at 17.77 mg/dL, with a standard deviation of ± 7.91 mg/dL.

Before proceeding with the correlation analysis, a prerequisite normality test was conducted to determine the distribution of the data. The Shapiro-Wilk test was selected as the appropriate method because the sample size was less than 50 (N=40).

Table 2. Normality Test Results (Shapiro-Wilk)

Variable	Statistic	df	p-value	Distribution
BW	0.983	40	0.804	Normal
TSB	0.769	40	< 0.001	Not Normal
Log_TSB	0.965	40	0.246	Normal

The results, as detailed in Table 2, indicated that the data for Birth Weight were normally distributed, with a significance value of $p=0.804$ ($p > 0.05$). However, the data for TSB were not normally distributed, showing a significance value of $p<0.001$. Due to the non-normal distribution of the TSB data, the bivariate analysis was conducted using the non-parametric Spearman's Rank Correlation test.

For the purpose of multivariate analysis, which requires the dependent variable to be normally distributed, the TSB data underwent a natural logarithm transformation (Log_TSB). Subsequent testing confirmed that the transformed data (Log_TSB) followed a normal distribution ($p=0.246$), fulfilling the assumption for linear regression.

Table 3. Spearman's Correlation Test Results

Variables	r_s	p
BW vs. TSB	0.198	0.222

The hypothesis regarding the relationship between total serum bilirubin levels and birth weight was assessed using a bivariate analysis. The study found a correlation coefficient (r_s) of 0.198 using the Spearman's Rank Correlation test, suggesting a very weak positive link between the two variables.

The p-value found was 0.222. The result is not statistically significant since this value is greater than the significance level of $a = 0.05$. We conclude that there is no significant correlation between birth weight and total blood bilirubin levels in this study

group after rejecting the alternative hypothesis (H_a) based on this data.

A multivariate linear regression analysis was carried out to give a more thorough understanding of the variables affecting bilirubin levels. The purpose of this analysis was to assess the impact of birth weight while accounting for any confounding factors, particularly gender and gestational age.

The model summary indicated an R^2 value of 0.157, meaning that the combination of birth weight, gestational age, and gender could only explain 15.7% of the variance in bilirubin levels, while the remaining 84.3% was influenced by other unmeasured factors. The simultaneous ANOVA test showed an F-value of 2.227 with a significance of p=0.102, suggesting that these variables collectively did not have a significant effect on bilirubin levels.

Table 4. Multivariate Linear Regression Analysis Results

Variable	B	t	p-value
(Constant)	0.224	0.939	0.354
BW (g)	0	1.013	0.318
GA (weeks)	0.048	0.582	0.564
Gender	0.248	2.17	0.037

The partial test results (t-test) in Table 4 further confirmed the bivariate findings. Birth weight was found to be a non-significant predictor (p=0.318) even after adjustment. Similarly, gestational age did not show a significant association (p=0.564). Interestingly, gender was identified as a significant factor (p=0.037) contributing to the variation in bilirubin levels in this sample.

DISCUSSION

The primary finding of this study demonstrates that birth weight is not significantly associated with total serum bilirubin levels in neonates diagnosed with neonatal jaundice. This conclusion is supported by the bivariate analysis results (r_s = 0.198, p = 0.222) and further confirmed by the multivariate linear regression model (p = 0.318) after controlling for gestational age and gender. Consequently, the alternative hypothesis (H_a) is rejected.

This result deviates from a number of earlier observational studies that found LBW to be a substantial risk factor for newborn jaundice. Due to hepatic immaturity, LBW was linked to a 2–9-fold increase in risk in those studies.[21,22] However, the non-significant result in this current study suggests that within this specific population, birth weight does not act as a standalone determinant of bilirubin dynamics. Several physiological and methodological factors may explain this discrepancy.

First, the dominance of gestational age over birth weight regarding hepatic maturity plays a crucial role. Physiologically, the capacity for bilirubin conjugation is dependent on the activity of the hepatic enzyme UGT1A1, which correlates more strongly with gestational age than with somatic growth (birth weight).[5,34] The descriptive data of this study reveals that the sample consisted predominantly of term infants, with a mean gestational age of 37.80 ± 0.85 weeks. In term infants, hepatic function is generally sufficient to handle physiological bilirubin loads, even if the infant is small for gestational age (SGA). Therefore, in a cohort with homogenous gestational maturity, the variation in birth weight becomes less relevant to bilirubin metabolism .

Second, the results point to a potential "masking effect" caused by dominant pathological conditions. The study recorded extreme total serum bilirubin values reaching up to 55.1 mg/dL, with a non-normal distribution. Such extreme levels strongly suggest the presence of severe underlying pathologies, such as sepsis, ABO/Rh incompatibility, or G6PD deficiency, which were not fully captured in the dataset.[25] In cases of massive hemolysis or systemic infection, the rate of bilirubin production far exceeds the liver's conjugation capacity, regardless of the infant's birth weight. Under these pathological circumstances, the influence of birth weight becomes negligible compared to the magnitude of the underlying disease process.

Interestingly, the multivariate analysis identified gender as a significant predictor (p=0.037) contributing to bilirubin variance. This aligns with literature suggesting that male infants may have a higher predisposition to neonatal jaundice, potentially linked to genetic factors like G6PD deficiency, which is X-linked, although this specific diagnosis was not verified in all subjects.[35]

Several limitations of this study must be acknowledged to contextualize the findings. First, the use of secondary data from medical records limited the researcher's ability to verify clinical details and resulted in the exclusion of potential subjects due to incomplete data. Second, the relatively small sample size (N=40) resulted in a wide confidence interval and reduced statistical power, increasing the risk of Type II errors (failing to detect a relationship that might exist) . Third, critical confounding variables such as maternal-fetal blood incompatibility (ABO/Rh) and G6PD deficiency status could not be consistently controlled in the multivariate model due to data unavailability. The inability to isolate these pathological factors likely contributed to the "masking effect" observed in the results.

CONCLUSION AND SUGGESTION

This study concludes that there is no statistically significant relationship between birth weight and total serum bilirubin levels in neonates diagnosed with neonatal jaundice at Ngoerah Hospital (p = 0.222; r = 0.198). Multivariate analysis further confirmed that birth weight is not a significant independent predictor (p = 0.318) after controlling for gestational age and gender. These findings suggest that birth weight does not serve as a primary determinant of bilirubin dynamics in this specific population, likely due to the predominance of term infants and the potential masking effect of unmeasured pathological confounders. Consequently, future research should utilize a prospective design with a larger sample size and strictly control for hemolytic status (ABO/Rh incompatibility and G6PD deficiency) and sepsis to isolate the specific effect of birth weight. Clinically, healthcare professionals are advised not to rely solely on birth weight for risk prediction but to prioritize comprehensive assessments focusing on gestational age and signs of neurotoxicity, while hospitals are encouraged to improve the completeness of medical records regarding blood types and comorbidities to support accurate future analysis..

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