Section: Paediatrics

Original Research Article

STUDY ON THE EFFECTIVENESS OF CORD BLOOD ALBUMIN AS A PREDICTOR OF NEONATAL JAUNDICE IN A TERTIARY CARE HOSPITAL IN EAST INDIA

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Received *: 04/06/2025*
Received in revised form *: 17/07/2025*
Accepted *: 05/08/2025*

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DOI:10.70034/ijmedph.2025.3.266

Source of Support: Nil,
Conflict of Interest: Nonedeclared

Int J Med Pub Health
2025; 15 (3); 1444-1449

ABSTRACT

Background: Neonatal Hyperbilirubinemia is the most common abnormal physical finding in 1st week of life, neonatal hyperbilirubinemia affects 60% of term and 80% of preterm neonates in 1st week of life. **Aim:** To estimate the development of Neonatal Hyperbilirubinemia at birth using cord blood serum albumin as a risk indicator.

Materials and Methods: Observational study performed on 200 healthy term newborns. Cord blood was collected from healthy term newborns delivered by cesarian section for cord serum album, in measurements. TSB were measured at 72-96 hours of life with serum sampling of peripheral venous blood. Babies were assessed clinically daily for Neonatal Hyperbilirubinemia and other complications.

Result: Study cohort is grouped into Group 1, Group 2 and Group 3 based on the Cord Serum Albumin level ≤ 2.8g/dl, 2.9-3.3g/dl and ≥ 3.4g/dl, respectively. In these groups, the newborns with total serum bilirubin level ≥17mg/dl after 72 hours requiring interventions like phototherapy or exchange transfusion are taken as neonatal hyperbilirubinemia. Statistical analysis was done for correlation of cord serum albumin with neonatal hyperbilirubinemia. It showed that cord serum albumin level ≤ 2.8g/dl is the critical for the newborn who developed neonatal hyperbilirubinemia. In cord serum albumin group ≥ 3.4g/dl, none of the newborns developed neonatal hyperbilirubinemia.

Conclusion: There is a correlation between Cord serum albumin level and neonatal hyperbilirubinemia in healthy term newborns. Cord serum albumin level of ≤2.8 g/dl can predict the development of neonatal hyperbilirubinemia.

Keywords: Cord Albumin, Neonatal Hyperbilirubinemia, Term Newborn, Serum bilirubin levels, Phototherapy.

INTRODUCTION

Neonatal Hyperbilirubinemia (NH) is the most common physical finding in the first week of life, affecting 60% of term and 80% of preterm infants. Caused by the accumulation of unconjugated bilirubin, it typically reflects a physiological transition from fetal to neonatal metabolism. However, severe cases may lead to serious complications such as cerebral palsy, hearing loss, and mental retardation. Early discharge practices, limited follow-up facilities—especially in India—and missed diagnosis contribute to increased readmission rates. Bilirubin is mainly cleared via the placenta in utero due to immature hepatic processing, with UDPGT activity developing slowly postnatally. Effective management includes early detection and phototherapy, which is cost-efficient and safer than exchange transfusion. Identifying high-risk neonates and implementing targeted follow-up programs can improve outcomes. Prioritizing awareness and practical neonatal care protocols is essential, particularly in resource-limited settings. Bilirubin is primarily produced from the breakdown of red blood cell hemoglobin (75%) and from early-labeled sources like ineffective erythropoiesis and tissue heme proteins (25%). Heme degradation involves two enzymatic

steps: first, heme oxygenase converts heme to biliverdin, releasing iron and carbon monoxide; second, biliverdin reductase reduces biliverdin to bilirubin. In plasma, bilirubin binds tightly to albumin, minimizing toxicity and preventing CNS entry.

Unbound bilirubin is taken up by hepatocytes and binds to ligandin (Y-protein), with phenobarbital enhancing ligandin levels. In the smooth endoplasmic reticulum, UDP-glucuronyl transferase conjugates bilirubin into water-soluble mono- and di-glucuronides. Conjugated bilirubin is excreted into bile, with excretion being the rate-limiting step. In the gut, β-glucuronidase can reverse conjugation, facilitating enterohepatic recycling unless gut bacteria convert bilirubin into non-reabsorbable urobilinoids.

Albumin, a 69 kDa protein, makes up 60% of plasma protein. Synthesized endogenously from early fetal life, albumin does not cross the placenta. In neonates, albumin levels are low (~2.5 g/dL), rising to adult levels (~3.5 g/dL) gradually. Its role in neonatal hyperbilirubinemia is crucial—albumin binds circulating bilirubin, reducing free bilirubin and lowering risk of bilirubin-induced neurologic dysfunction. Albumin contributes 70–75% of plasma oncotic pressure and serves as a carrier for bilirubin, free fatty acids, calcium, cysteine, and drugs. It functions as an antioxidant and reflects liver synthetic capacity, though its 19–21 day half-life limits sensitivity to acute changes. In neonates, serum albumin levels rise with gestational age, averaging 3.1 g/dL at term. Postnatal increases prepare for oxidative stress and transport needs. Bilirubin binds albumin equimolarly (8.5 mg/g), and free bilirubin—neurotoxic and able to cross the blood-brain barrier—emerges when the bilirubin-to-albumin molar ratio exceeds 0.8, though exact toxicity thresholds remain undefined.

Etiology and Complications of Neonatal Hyperbilirubinemia

Neonatal hyperbilirubinemia arises from increased bilirubin production or impaired elimination. Physiologic jaundice is common in newborns due to elevated red cell mass, shorter RBC lifespan, increased ineffective erythropoiesis, and heightened enterohepatic circulation. Contributing factors include decreased ligandin-mediated uptake, impaired conjugation via reduced UDPG-T activity, and limited hepatic excretion.

Non-physiologic jaundice stems from:

- **Overproduction**: Blood group incompatibility (Rh/ABO), inherited RBC defects (e.g., spherocytosis, enzyme deficiencies), acquired hemolysis, polycythemia, gastrointestinal anomalies (e.g., pyloric stenosis), and swallowed maternal blood.
- **Under-secretion**: Metabolic/endocrine disorders (e.g., hypothyroidism, galactosemia), hereditary syndromes (e.g., Crigler-Najjar, Gilbert), and structural hepatobiliary defects (e.g., biliary atresia, choledochal cyst).
- **Mixed causes**: Sepsis, intrauterine infections (e.g., TORCH), and perinatal complications.
- **Uncertain mechanisms**: Breast milk jaundice and ethnic predispositions in East Asian and Indigenous populations.

Timing of jaundice onset offers diagnostic clues:

- 24–72 hours: Physiologic jaundice, sepsis, polycythemia, enhanced enterohepatic circulation.
- 72 hours: Neonatal hepatitis, hypothyroidism, Crigler-Najjar type II, Gilbert syndrome.

Complications include bilirubin encephalopathy and kernicterus, characterized by neurotoxic bilirubin deposition in CNS regions such as the basal ganglia and cerebellum. Pathogenesis involves unbound bilirubin crossing the blood-brain barrier, influenced by albumin binding, maturity of brain structures, and cellular metabolism. Lipophilic bilirubin may infiltrate tissues when plasma levels rise or albumin binding decreases, potentially leading to irreversible neuronal damage. Neonatal hyperbilirubinemia (NH) affects 60% of term and 80% of preterm infants during the first week of life. Three major hypotheses explain bilirubin neurotoxicity: precipitation of bilirubin acid in nerve membranes, increased tissue uptake due to low pH in acidotic infants, and permeability of a damaged blood-brain barrier. Recent studies highlight P-glycoprotein (P-gp), an ATP-dependent transport protein in the blood-brain barrier, as a key regulator preventing bilirubin's entry into the brain.

Risk factors for neurotoxicity include asphyxia, hyperthermia, sepsis, hypoalbuminemia, acidosis, calorie deprivation, prolonged hyperbilirubinemia, low birth weight, young gestational age, and excessive hemolysis. At the cellular level, bilirubin toxicity may disrupt neurotransmission, impair mitochondrial and membrane function, and interfere with enzyme activity.

Clinically, kernicterus progresses in phases: early (lethargy, poor feeding, hypotonia), intermediate (seizures, opisthotonos, apnea, hypertonia), and advanced (coma, death). Survivors may show chronic signs such as cerebral palsy, sensorineural hearing loss, dental dysplasia, and intellectual deficits.

Brainstem Evoked Auditory Response (BEAR) is a noninvasive, accurate test to detect bilirubin's early impact on auditory pathways, aiding decisions on exchange transfusion. Nuclear Magnetic Resonance (NMR) imaging and spectroscopy offer noninvasive tools to detect bilirubin-induced brain injury.

Kramer's dermal staining method assesses jaundice by observing skin color progression from face to soles, correlating to bilirubin levels. However, physical exam alone isn't reliable for serum bilirubin estimation. Key risk factors include prematurity, low birth weight, ABO/Rh

incompatibility, maternal diabetes, poor breastfeeding, bruising, and sibling history.

Management involves identifying high-risk infants, ensuring follow-up, encouraging exclusive breastfeeding, clinical evaluation, distinguishing physiological vs. pathological jaundice, and initiating appropriate interventions with parental counseling. Neonatal hyperbilirubinemia is a common condition characterized by elevated serum bilirubin levels. **Physiological jaundice** typically appears after 36 hours of life, with bilirubin levels peaking between the 3rd–5th day in term infants and resolving by two weeks. It rises at

Pathological jaundice is suspected if jaundice appears within the first 24 hours, TSB exceeds 15 mg/dl, rises >0.2 mg/dl/hour or >5 mg/dl/day, direct bilirubin >2 mg/dl or >15% of TSB, or persists beyond 2 weeks. Risk factors include iso-immune hemolysis, G6PD deficiency, sepsis, birth asphyxia, acidosis, and hypoalbuminemia.

According to **AAP 2004 guidelines**, phototherapy decisions are based on total bilirubin levels without subtracting the conjugated fraction. Phototherapy is initiated considering gestational age (35–37 6/7 weeks), infant's condition, and risk factors. Home phototherapy is discouraged for at-risk infants.

Exchange transfusion is indicated when bilirubin is dangerously high, especially with signs of acute bilirubin encephalopathy (e.g., hypertonia, retrocollis). It becomes mandatory if TSB exceeds guideline thresholds by ≥5 mg/dl. Monitoring includes serum albumin (B/A ratio), red cell morphology (spherocytes in ABO incompatibility, fragments in DIC), CBC parameters, and tests for G6PD and galactosemia.

Phototherapy, first introduced in the 1950s, reduces bilirubin via configurational and structural photoisomerization. Structural conversion to lumirubin is irreversible and facilitates excretion. Blue light (450–460 nm) is most effective; white light is commonly used in India. Distance from the light source influences intensity (15–45 cm optimal).

The **IAP NNF 2006 guidelines** emphasize treatment decisions based on TSB levels, gestational age, and infant condition. Prompt intervention is vital to prevent kernicterus and irreversible brain damage.

Phototherapy is the primary treatment for neonatal jaundice, requiring optimal spectral irradiance (4–6 μw/cm²/nm) at the infant's skin level. Key precautions include shielding the eyes to prevent retinal damage and covering the genitals. Breastfeeding is continued on demand, supplemented by extra IV fluids (10–20%) if needed. Adequate hydration is assessed via urine output, skin turgor, and weight. Temperature monitoring every 3–4 hours helps avoid hypo/hyperthermia. Neonates are weighed daily and total serum bilirubin (TSB) measured every 12 hours, or every 4–6 hours in severe cases. Adverse effects of phototherapy include dehydration, green stools (due to transient lactose intolerance and photo catabolites), hyperthermia, irritability, rash, PDA (in pre-terms), hypocalcemia, bronze baby syndrome, theoretical risk of skin malignancy, and circadian rhythm disruptions affecting puberty.

When phototherapy is insufficient, **exchange transfusion** is considered. It rapidly reduces serum bilirubin and replaces antibody-coated RBCs with unsensitized donor cells. Rh-negative blood or type O cells with AB plasma is preferred. A double volume exchange (160 ml/kg) replaces ~87% of the infant's blood. Techniques include push-pull through the umbilical vein, iso-volumetric exchange via the umbilical artery/vein, or central venous routes. Blood is withdrawn in aliquots based on weight (5–20 ml) over ~1 hour.

- Pharmacological adjuncts include:
- Phenobarbitone (10 mg/kg IM or 5 mg/kg/day orally) enhances bilirubin conjugation.
- Clofibrate boosts glucuronyl transferase but acts slowly.
- Agar (250 mg 6th hourly) binds bilirubin in the gut.
- Cholestyramine (1.5 mg/kg/day) promotes fecal bilirubin excretion.
- Orotic acid increases UDP-glucuronic acid synthesis but has limited use.
- Tin-mesoporphyrin inhibits heme oxygenase but carries toxicity risks.
- Albumin infusion (1 gm/kg) improves bilirubin removal but is costly and risky.
- IVIg (0.5–1 gm/kg) prevents RBC hemolysis in isoimmune conditions.

Preventive measures include avoiding bilirubin-displacing drugs, phenolic nursery detergents, and excess vitamin K. Managing perinatal distress and initiating early feeding help reduce enterohepatic circulation and support meconium clearance. Timely follow-up post-discharge is critical as significant jaundice may present in up to 60% of term and 80% of preterm neonates, potentially causing bilirubin-induced brain damage if missed.

Objectives

Primary Objective: To evaluate the predictive value of cord blood serum albumin (CSA) levels for the development of significant neonatal hyperbilirubinemia requiring therapeutic interventions such as phototherapy or exchange transfusion.

Secondary Objectives:

- To analyze the association between different CSA level ranges and the severity of neonatal hyperbilirubinemia.
- To estimate the proportion of neonates at birth who may require treatment for hyperbilirubinemia based on their CSA levels.
- To identify a critical CSA threshold that may serve as an early biomarker for high-risk neonates.
- To contribute to the optimization of early screening protocols and intervention strategies for neonatal jaundice management.

Review of Literature

Jaundice was first described in Ayurveda (1500–800 BC) as a bile imbalance. Greek medicine linked it to yellow bile. Early neonatal cases appeared in 15th-century Europe. Scientific breakthroughs from Virchow to Crigler-Najjar clarified bilirubin's role, while discoveries in immunology and transfusion revealed causes of erythroblastosis fetalis and led to lifesaving treatments

Zakia Nahar et al. (2009) conducted a study on 84 healthy newborns to assess the predictive value of cord blood bilirubin for hyperbilirubinemia: **Group I (71 infants)**: No significant hyperbilirubinemia, **Group II (13 infants)**: Developed hyperbilirubinemia (>17 mg/dL) **Results**: Sensitivity: 77%, Specificity: 98.6%,Positive Predictive Value: 91%, Negative Predictive Value: 96%. **Conclusion**: Cord bilirubin >2.5 mg/dL is a reliable early predictor, particularly relevant for preterm infants.

Risemberg et al. (1977) Studied newborns with ABO incompatibility. Found that **cord blood bilirubin >4 mg/100 ml** predicted **serum bilirubin >16 mg/100 ml** within 12–36 hours. Recommends follow-up for early intervention.

Rosenfeld (1986) Analyzed 108 full-term infants had only **4% risk** of jaundice vs. **25%** in >2 mg/100 ml group. Higher bilirubin levels were associated with increased phototherapy needs.

Sao Paulo et al. Found that **cord blood bilirubin ≥2.0 mg/dl** indicated a **53% probability** of requiring phototherapy.

Bhutani et al. (1999) Developed a **percentile-based nomogram** for risk stratification based on pre-discharge TSB. High risk (>95th percentile): **57% chance** of significant hyperbilirubinemia. Low risk (**0% chance**. Supported universal pre-discharge TSB screening to reduce kernicterus risk.

Transcutaneous Bilirubinometry (Bilicheck):

Spectrophotometric device used for non-invasive bilirubin estimation.

Effective within bilirubin range.

Values above 75th percentile on the nomogram indicate high-risk status

Newman et al. Combined **clinical risk factors** with **early serum bilirubin** improved predictive value over bilirubin alone.

Stevenson et al. Attempted using **End Tidal Carbon Monoxide (ETCO)**. While not improving nomogram accuracy, combining ETCO with early TSB helped distinguish between increased bilirubin production (e.g., hemolysis) vs. decreased elimination (e.g., conjugation defect).

Suchanda Sahu (2011): Grouped neonates by cord albumin levels: **82% developed jaundice**. 3.4 g/dl: None developed jaundice. Cord albumin shown to predict risk stratification.

Trivedi et al. (2013): Included **605 term neonates**; **33.88% developed hyperbilirubinemia**. **Cord albumin** seen in **53.53%** of affected babies. Albumin and bilirubin together enhanced prediction.

Meena et al. (2015): Group A (**95.5% jaundice**, high phototherapy & exchange transfusion rates. Group C (>3.4 g/dl): **36.6% jaundice**, low intervention requirement. Significant association ($p < 0.001$).

Reshad M et al. (2016): Evaluated term & preterm neonates. Albumin ≤2.8 g/dl showed high sensitivity. Albumin ≥3.4 g/dl deemed protective.

Aiyappa GKC et al. (2017): Cord albumin had **71.8% sensitivity**, **65.1% specificity**. Higher jaundice rate and intervention need in Group 2 (

Pradeep Kumar et al. (2017): Studied 50 neonates; Group I (albumin **85.71% icterus at 24–48 hrs**, all needed phototherapy.

Pushpanjali et al. (2018): Studied 105 babies. Group A (**80.9% jaundice**, 71.4% phototherapy. Group C (>3.3 g/dl): Only **26% jaundice**, minimal intervention. Recommends albumin-based risk stratification before discharge.

MATERIALS AND METHODS

Conducted at Hi-Tech Medical College and Hospital, Bhubaneswar.

- Duration: March 2023 – February 2025.
- Included 200 randomly selected term neonates.
- Ethical approval obtained from the Institutional Ethical Committee.

Inclusion & Exclusion Criteria

Inclusion:

- Term neonates of either gender.
- Birth weight ≥2.5 kg.
- APGAR score ≥7/10 at 1 minute.
- Delivered via normal or Caesarean section.

Exclusion

- Preterm birth, Rh incompatibility.
- Neonatal sepsis, respiratory distress, birth asphyxia.
- Instrumental delivery.
- Meconium-stained amniotic fluid.
- Jaundice within the first 24 hours.

Data Collection Procedure

1. Parental informed consent obtained prior to participation.
2. Demographics and maternal history were recorded through structured proforma.
3. Gestational age assessed via New Ballard Score (if LMP was uncertain).
4. Cord Serum Albumin sampled at birth.
5. Total Serum Bilirubin (TSB) assessed between 72–96 hours of life.
6. Daily follow-up for the first 4 days to monitor Neonatal Hyperbilirubinemia (NH).

Laboratory Investigations

Cord Blood (2 ml):

- Collected immediately post-delivery from placental side.
- Assessed for Cord Serum Albumin via auto-analyzer (ERBA XL640200).
- Venous Blood (72–96 hrs):

- Evaluated for:
  - Total and Direct Bilirubin (Diazotized sulfonic acid spectrometry method).
  - Blood group (Antisera-based agglutination technique).
- Samples stored at 2–8°C, protected from light, processed within 12 hours.

Outcome & Analysis

- Neonatal Hyperbilirubinemia defined as TSB ≥17 mg/dL after 72 hours.
- Treatment decisions guided by AAP 2004 guidelines and IAP-NNF criteria.
- Statistical analysis done using SPSS v24, employing chi-square tests.

RESULTS

Demographics and Delivery Details

- **Gender distribution**: Of the 200 newborns, 61.4% were female and 38.6% male.
- **Mode of delivery**: The majority (61.4%) were born via vaginal delivery, with 38.6% born through cesarean section.
- **Maternal weight**: Most mothers weighed between 60–70 kg (41.1%) and 70–80 kg (32.7%) near delivery.
- **Oxytocin use**: Oxytocin was administered in 60% of deliveries (120 out of 200 cases).
- **Maternal blood groups**: Blood group O was the most common (45%), followed by B (25%) and A (22%).

Neonatal Birth Profiles

- **Birth weight**: All newborns weighed ≥2.5 kg, with 84% between 2.5–3.0 kg. Mean birth weight was 2.73 kg.
- **Cord serum albumin (CSA) levels**: The cohort was categorized into:
  - Group 1 (≤2.8 g/dL): 46.5%
  - Group 2 (2.9–3.3 g/dL): 30%
  - Group 3 (≥3.4 g/dL): 23.5%
- **Newborn blood groups**: B+ (47%) was the most prevalent, followed by A+ (30%) and O+ (19%).

Neonatal Hyperbilirubinemia (NH) Findings

- **Total serum bilirubin (TSB)**: Measured at 72–96 hours postnatally:
  - 82% had TSB between 10–14 mg/dL
  - 11.5% had ≥17 mg/dL
- **Phototherapy requirement**:
- 25 newborns (12%) required phototherapy.
- No newborn required exchange transfusion.

Correlation Analyses

- **CSA vs Gender**: No statistically significant correlation (P = 0.352).
- **CSA vs Birth weight**: No significant relationship (P = 0.84).
- **CSA vs Maternal weight**: No significant correlation (P = 0.084).
- **CSA vs Oxytocin administration**: No significant association (P = 0.137).
- **CSA vs Phototherapy need**: Highly significant (P < 0.001):
- 26.6% of Group 1 (low CSA) required phototherapy
- Only 1.6% of Group 2 and none in Group 3 required it.

Table 1: Cord Albumin levels relation with Phototherapy

	Cord Albumin levels			
Phototherapy	**≤ 2.8**	**2.9-3.3**	**≥ 3.4**	**Total**
No	69(73.4%)	59(98.4%)	47(100%)	177(87.1%)
Yes	24(26.6%)	1(1.6%)	0(0%)	25(12.9%)
Total	93(100%)	60(100%)	47(100%)	200(100%)

Clinical Variable Correlation with Phototherapy

- **Gender**: No significant difference (P = 0.408).
- **Mode of delivery**: No association (P = 0.679).
- **Oxytocin use**: Not statistically significant (P = 0.557).
- **CSA level**: Statistically significant (P < 0.001) correlation with NH requiring phototherapy.
- CSA level is the only variable significantly associated with the need for phototherapy, highlighting its potential role in predicting NH severity.
- Other clinical variables—including gender, birth weight, maternal weight, delivery mode, and oxytocin administration—showed no significant impact on NH development or treatment needs.

DISCUSSION

Neonatal hyperbilirubinemia (NH) is a significant cause of readmission among term newborns. While kernicterus—the chronic sequelae of acute bilirubin encephalopathy—is rare, its incidence is unknown and its consequences irreversible. Defining a physiological bilirubin threshold may be misleading and potentially dangerous. Timely detection and intervention are crucial, especially in early-discharged infants.

This study aimed to assess the role of **cord serum albumin (CSA)** levels in predicting risk for NH. Several predictive studies using cord bilirubin levels show varied opinions, whereas albumin—being a bilirubin-binding protein—holds potential as a reliable risk marker.

Sex of Newborns

The present study included 200 newborns: 77 males and 123 females. Out of these, 25 developed NH (≥17mg/dl). Statistical analysis (p = 0.352) showed no significant association between sex and NH. These findings align with studies by Amar Taksande et al. (2005) and Rostami et al. (2005), both indicating no gender predilection.

However, other studies like Maisal et al. (1998) and Rudy Satrya et al. (2009) observed a higher risk of NH in males. Conversely, Trivedi et al. (2013) also reported increased NH among male infants.

Mode of Delivery

Among 123 vaginal deliveries, 15 cases developed NH; among 77 cesarean deliveries, 11 developed NH. The association was statistically insignificant (p = 0.679), showing no correlation between delivery mode and NH. This result mirrors findings from Amar Taksande et al. (2005), Rostami et al. (2005), and Rudy Satrya et al. (2009).

Oxytocin Induction of Labour

Of the 120 mothers receiving oxytocin, 18 neonates developed NH, while 8 of the 80 non-induced cases developed NH. No significant association was found (p > 0.05). These outcomes are consistent with studies by Rostami et al. (2005) and Amar Taksande et al. (2005). Oral E et al. (2003) concluded that oxytocin infusion doesn't impact NH unless used for induction.

Incidence Rates Compared with Other Studies

The present study noted a NH incidence rate of 12.87%. Other comparable studies include:

Table 2: Comparison with other studies

Study	Year	Cases	Incidence (%)
Palmer *et al.*	1983	41057	10.70
Awasthi *et al.*	1998	274	12.80
Agarwal *et al.*	2002	213	10.30
Trivedi *et al.*	2013	605	33.80
Pushpanjali *et al.*	2018	105	25.00

The rate in this study aligns closely with multiple previous reports, suggesting consistent patterns across populations.

Cord Serum Albumin (CSA) Levels and NH

Newborns were grouped by CSA levels:

- **Group 1**: ≤2.8 g/dl → 24 out of 25 NH cases (96.1%)
- **Group 2**: 2.9–3.3 g/dl → 1 case
- **Group 3**: ≥3.4 g/dl → No cases

Statistical significance was noted (p < 0.001), indicating a strong inverse relationship between CSA and NH. These findings correlate with studies by Sahu et al. (2011) and Trivedi et al. (2013).

CONCLUSION

Neonatal hyperbilirubinemia affects 5–10% of healthy term neonates, with jaundice accounting for ~85% of early readmissions.

Cord serum albumin ≤2.8 g/dL significantly correlates with hyperbilirubinemia (≥17 mg/dL), indicating increased phototherapy risks.

CSA level is the only variable significantly associated with the need for phototherapy, highlighting its potential role in predicting NH severity.

Other clinical variables—including gender, birth weight, maternal weight, delivery mode, and oxytocin administration—showed no significant impact on NH development or treatment needs.

Identifying high-risk neonates at birth can guide discharge decisions and improve early management.

Limitations

Only full-term healthy newborns were studied; follow-up lasted 5 days post-delivery, potentially missing delayed peak bilirubin levels.

Study focused solely on the predictive value of cord serum albumin without assessing other risk factors.

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