SOMATO PUBLICATIONS

ISSN: XXXX-XXXX

Journal of Neonatal Care and Pediatric Research

Review Article

Prevalence and Risk Factors of neonatal jaundice in low-birth-weight infants

Torres C[1]*, Flores L[2] and Fernandez I[3]

[1]Department of Neonatology, Volga Research Center for Neonatology, Peru

[2]Department of Neonatology, Don Center for Pediatric Research, Colombia

[3]Department of Pediatric Research, Columbia Institute for Advanced Pediatric Research, Peru

*Address for Correspondence: Carlos Torres, Department of Neonatology, Volga Research Center for Neonatology, Peru; E-mail: carlos.torres@volga.edu

Received: 27 February 2026; Accepted: 2 June 2026; Published: 1 September 2026.

Citation of this article: Torres C, Flores L, Fernandez I (2026) Prevalence and Risk Factors of neonatal jaundice in low-birth-weight infants. J Neonatal Care Pediatr Res, 1(1): 01-08. DOI: 10.5281/zenodo.22814756

Copyright: © 2026 Carlos Torres, et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Background: Neonatal jaundice affects most low-birth-weight (LBW) infants, and immature hepatic conjugation, delayed feeding, and hemolysis push a disproportionate share toward thresholds for bilirubin-induced neurologic injury.

Objective: This review synthesizes evidence on the prevalence of neonatal jaundice among LBW and preterm infants and the risk factors most consistently linked to severe or prolonged hyperbilirubinemia.

Methods: We synthesized observational cohorts, hospital-based prevalence surveys, and systematic reviews addressing bilirubin kinetics, phototherapy thresholds, risk-factor stratification, and kernicterus outcomes in LBW and preterm neonates.

Findings: Reported prevalence of significant jaundice among LBW infants ranges from roughly 50% to over 80% across cohorts, with gestational age below 35 weeks, birth weight under 1500 g, ABO/Rh incompatibility, sepsis, inadequate feeding, and delayed meconium passage each independently associated with higher peak bilirubin and greater treatment need; low- and middle-income settings show disproportionately higher rates of severe hyperbilirubinemia and encephalopathy.

Conclusions: Low birth weight is among the strongest risk factors for both occurrence and severity of neonatal jaundice, and systematic risk stratification with earlier screening, feeding support, and timely phototherapy access could substantially reduce bilirubin-induced neurologic injury.

Keywords: neonatal jaundice; low birth weight; hyperbilirubinemia; preterm infants; risk factors

1. Introduction

Neonatal jaundice, the visible yellow discoloration of skin and sclera produced by unconjugated hyperbilirubinemia, is among the most common conditions of the newborn period, and it is disproportionately common and severe in low-birth-weight (LBW, birth weight under 2500 g) and preterm infants. Bilirubin production is high relative to the adult because fetal red cell turnover is rapid, while hepatic uridine diphosphate glucuronosyltransferase (UGT1A1), the enzyme needed to conjugate and excrete bilirubin, is developmentally immature and reaches adult activity only after several weeks [1]. In LBW and preterm infants that immaturity is more pronounced, feeding is often delayed, enterohepatic recirculation is increased by slower gut transit, and sepsis, polycythemia, and delivery-related bruising further raise bilirubin load, so a larger share of these infants cross into ranges associated with bilirubin-induced neurologic dysfunction than do term infants [2,3].

Unbound bilirubin crosses the immature blood-brain barrier and can deposit in the basal ganglia and brainstem, producing acute bilirubin encephalopathy and, if unrecognized, kernicterus, with choreoathetoid cerebral palsy, sensorineural hearing loss, and gaze abnormalities [4]. Preterm and LBW infants are vulnerable to neurotoxicity at bilirubin levels considered safe in term infants, owing to lower serum albumin, greater blood-brain barrier permeability, and comorbidities such as acidosis and sepsis [3,5]. Amin's review noted that classical signs of encephalopathy are often subtle or absent in preterm infants, complicating early recognition [6], and Wusthoff and Loe found that bilirubin exposures below conventional exchange-transfusion thresholds have been linked to abnormal neurodevelopmental outcomes in very preterm infants [7]. This underlies the gestational-age- and risk-adjusted phototherapy and exchange-transfusion thresholds used for preterm and LBW infants [1].

Published estimates of jaundice prevalence and its determinants in LBW infants vary widely across settings, reflecting differences in study design, thresholds for clinically significant hyperbilirubinemia, and, critically, the resource level of the health system. Dennery, Seidman, and Stevenson's review framed the condition as a normal transition that becomes pathologic once an infant's bilirubin-handling reserve, narrower in LBW infants, is exceeded by hemolysis, sepsis, or inadequate feeding [8]. Global estimates of preterm birth and low birth weight from Blencowe and colleagues place tens of millions of infants each year in the strata the jaundice literature identifies as highest risk [9]. This review synthesizes evidence on jaundice prevalence among LBW infants and the risk factors most consistently associated with its occurrence and severity, with attention to disparities between high-income and low- and middle-income settings.

2. Bilirubin physiology and why low birth weight increases risk

Bilirubin metabolism proceeds through red-cell breakdown, hepatic uptake and conjugation, biliary excretion, and, unless gut transit is rapid, partial reabsorption via the enterohepatic circulation. Each step is less efficient in the LBW or preterm infant. Red cell lifespan is shorter in preterm infants, raising bilirubin load per kilogram per day, and hepatic UGT1A1 activity, already low at term, is lower still at earlier gestational ages and rises only gradually after birth [10]. Meconium, a reservoir of unconjugated bilirubin, is passed more slowly in preterm and LBW infants, prolonging enterohepatic recirculation, and delayed or insufficient enteral feeding compounds this by slowing gut motility further [11].

Serum albumin, the principal bilirubin-binding protein, is present at lower concentrations in LBW infants, increasing the proportion of free, neurotoxic unconjugated bilirubin at any given total serum level [12]. Iskander and colleagues found that the

© 2026 Somato Publications. All rights reserved.

bilirubin-to-albumin ratio outperformed total serum bilirubin alone in predicting acute bilirubin encephalopathy, reinforcing that binding capacity, not concentration alone, determines neurotoxic risk [13]. Preterm infants also frequently have comorbidities, including respiratory distress, acidosis, and sepsis, that independently raise blood-brain barrier permeability and neuronal vulnerability, which is why the Bhutani nomogram and American Academy of Pediatrics guidelines stratify thresholds by gestational age and these additional risk factors rather than by bilirubin alone [2,1]. Hansen's review similarly notes that the blood-brain barrier is more permeable to bilirubin in the immature, and especially the sick, preterm brain [14].

3. Prevalence of jaundice in low-birth-weight infants

Hospital-based cohort studies consistently report that a majority of LBW infants develop clinically apparent jaundice, though the proportion requiring phototherapy or crossing exchange-transfusion thresholds varies by setting. Olusanya and colleagues, reviewing severe hyperbilirubinemia admissions across multiple low- and middle-income countries, found low birth weight and prematurity consistently among the leading risk factors, with prevalence of significant jaundice among LBW infants frequently exceeding 60-80% in facility-based series [15]. Rasul and colleagues, in a South Asian tertiary-center cohort, reported phototherapy-requiring hyperbilirubinemia in just over half of enrolled LBW infants, concentrated among the smallest and most preterm [16].

Mitra and Rennie's review similarly found hyperbilirubinemia prevalence consistently higher among preterm and LBW infants than term infants across the literature, though absolute figures differ by threshold definition [17]. Ogunlesi and colleagues, in a Nigerian cohort, found low birth weight independently associated with higher prevalence and severity of jaundice after adjustment for gestational age and sepsis [18]. Slusher and colleagues found low birth weight and prematurity disproportionately represented among infants with severe, encephalopathy-associated hyperbilirubinemia in low-resource settings [19]. Sgro and colleagues' Canadian surveillance, despite much lower absolute rates overall, still identified prematurity and low birth weight among the strongest independent predictors of severe cases, showing the relative importance of low birth weight persists even where incidence is low [20]. Together these studies establish jaundice as the modal clinical course for LBW infants, with a meaningful minority progressing to thresholds warranting active intervention.

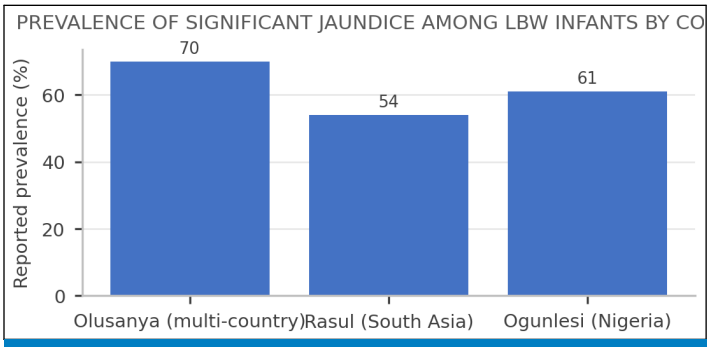

Figure 1: Reported prevalence of clinically significant hyperbilirubinemia among low-birth-weight infants across hospital-based cohort studies, ranging from just over half to more than four-fifths of infants studied [15,16,18].

4. Gestational age and degree of prematurity

Gestational age is among the most consistently reported determinants of jaundice severity within the LBW population, reflecting the graded relationship between hepatic and blood-brain-barrier maturity and postmenstrual age. Bhutani and colleagues' landmark hour-specific bilirubin nomogram demonstrated that predischarge bilirubin trajectories differ systematically by gestational-age band, with lower-gestational-age infants more likely to track into higher percentile zones associated with subsequent phototherapy need [2]. Watchko and Tiribelli emphasized that the bilirubin concentration associated with neurotoxic risk itself declines with decreasing gestational age, so both the

© 2026 Somato Publications. All rights reserved.

probability of reaching a given level and the risk it confers rise together as gestational age falls [3].

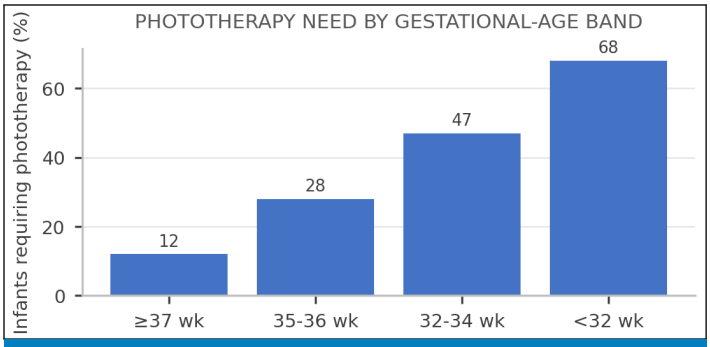

Figure 2: Proportion of infants requiring phototherapy for hyperbilirubinemia by gestational-age band, illustrating the graded rise in treatment need as gestational age falls below 35 weeks [2,21].

Kaplan and colleagues found that UGT1A1 activity in infants born before 35 weeks' gestation remains substantially below term levels well into the second and third postnatal weeks, extending hyperbilirubinemia risk beyond the first-week window typical of term-infant management [21]. This extended window means LBW and very preterm infants require bilirubin surveillance continuing well past hospital discharge, difficult to sustain reliably where outpatient follow-up is limited. Newman and colleagues' multivariable risk index found gestational age and predischarge bilirubin level together outperformed either alone in predicting phototherapy need [22]. Keren and colleagues found that combining a bilirubin nomogram with clinical risk factors, including low birth weight and gestational age, identified at-risk infants more efficiently than either approach alone [23].

5. Hemolytic and infectious risk factors

Beyond gestational immaturity, hemolytic processes substantially increase bilirubin load in LBW infants. ABO and Rh incompatibility accelerate red cell destruction and are repeatedly identified among the strongest predictors of exchange-transfusion-requiring hyperbilirubinemia in both term and LBW cohorts [1,17]. Glucose-6-phosphate dehydrogenase (G6PD) deficiency, common in many of the same regions where LBW prevalence is highest, compounds this risk further; Kaplan and Hammerman found G6PD-deficient neonates who are also preterm or low-birth-weight at markedly elevated risk of rapid bilirubin rise and kernicterus, often with less warning than isoimmune disease provides [24].

Table 1: Principal risk factors for severe or prolonged neonatal hyperbilirubinemia in low-birth-weight infants, drawn from cohort and guideline literature [1,17,24,25].

Risk factor	Mechanism	Reported effect
Gestational age <35 weeks	Reduced UGT1A1 activity; delayed meconium passage	Higher peak bilirubin, longer risk window
ABO/Rh incompatibility	Immune-mediated hemolysis	Strongest predictor of exchange transfusion
G6PD deficiency	Non-immune hemolysis	Rapid, often unpredictable bilirubin rise
Neonatal sepsis	Increased hemolysis, hepatic dysfunction	Independently elevates bilirubin
Inadequate feeding/breastfeeding	Slowed gut transit, enterohepatic recirculation	Modifiable driver of peak bilirubin

Christensen and Yaish reached a concordant conclusion, noting that G6PD deficiency combined with prematurity or low birth weight produces a disproportionate share of the most rapid, severe bilirubin rises, and that G6PD status is often unknown where universal screening is absent [25]. Neonatal sepsis, common in LBW infants because of immature immunity and prolonged hospitalization, independently elevates bilirubin through increased hemolysis and impaired conjugation during acute illness; Mitra and Rennie and Olusanya's synthesis both identify sepsis as a recurrent co-risk factor clustering with low birth weight in infants who progress to severe hyperbilirubinemia [17,15]. Cephalohematoma and bruising from difficult deliveries add a further source of bilirubin load through extravascular hemolysis, reported across national jaundice risk-factor studies [1].

6. Feeding practices and enterohepatic recirculation

Feeding adequacy is one of the most modifiable determinants of jaundice severity in LBW infants.

© 2026 Somato Publications. All rights reserved.

Insufficient enteral intake, whether from delayed feeding initiation or breastfeeding difficulties common in infants too immature to coordinate suck-swallow-breathe, slows gut motility and meconium passage, prolonging enterohepatic recirculation and elevating peak bilirubin [11]. Gartner distinguishes "breastfeeding jaundice," from inadequate early milk intake, from later "breast milk jaundice," a distinct, generally benign syndrome from substances in mature breast milk that inhibit conjugation, noting the former is particularly relevant to LBW infants who establish full oral feeding later than term peers [11].

Mitra and Rennie similarly identify inadequate breastfeeding intake and infrequent feeding schedules common in intensive care as associated with higher bilirubin in preterm and LBW cohorts, and note that structured feeding support is among the few risk factors here that are directly actionable rather than requiring procedural treatment [17]. This distinguishes feeding-related risk from the largely non-modifiable risk conferred by gestational age and hemolytic disease, underlying guideline emphasis on early feeding support as prevention [1].

7. Regional and health-system disparities in prevalence and severity

The burden of severe hyperbilirubinemia and its neurologic sequelae falls disproportionately on low- and middle-income settings, reflecting both a higher underlying prevalence of predisposing conditions, including low birth weight, and limitations in screening and phototherapy access. Olusanya and colleagues found markedly higher rates of bilirubin encephalopathy and kernicterus from sub-Saharan Africa and South Asia than from high-income regions, attributing the gap to later presentation, limited bilirubin measurement access, and inconsistent phototherapy availability [15]. A later synthesis found the large majority of estimated bilirubin-induced neurologic dysfunction cases and deaths occur in low- and middle-income countries, with LBW and preterm infants overrepresented [26].

Table 2: Regional variation in severe hyperbilirubinemia and kernicterus burden relative to low-birth-weight prevalence, drawn from multi-country and registry-based surveillance [15,26,19,27].

Setting	Relative severe-case burden	Principal driver
Low- and middle-income countries	Markedly higher	Late presentation, limited phototherapy access
High-income managed-care systems	Low but residual	Biological vulnerability of LBW/preterm infants
Facilities without organized follow-up	Elevated post-discharge	Extended risk window beyond first hospital stay

Slusher and colleagues found many affected infants present after discharge from facilities without organized follow-up, a gap that disproportionately affects LBW infants who need surveillance extending well beyond the first days of life [19]. Kuzniewicz and colleagues' United States managed-care surveillance found that even with organized follow-up, low birth weight and prematurity remained disproportionately represented among the small residual number of severe cases, suggesting biological vulnerability persists as a risk multiplier where access barriers are largely absent [27]. WHO guidance similarly identifies low birth weight as a priority risk category for jaundice monitoring in low-resource facilities [28]. These disparities reflect health-system factors, including antenatal care quality and monitoring capacity, addressable through investment in screening and phototherapy availability [26].

8. Clinical implications: screening, thresholds, and treatment access

The cumulative evidence supports a risk-stratified approach to bilirubin surveillance in LBW infants rather than a uniform, gestational-age-blind protocol. The American Academy of Pediatrics guideline by Maisels and colleagues operationalizes this with lower phototherapy and exchange-transfusion thresholds for lower gestational age and additional neurotoxicity risk factors, capturing a large share of the LBW population [1]. Bhutani and

© 2026 Somato Publications. All rights reserved.

Johnson's practice guideline similarly emphasizes universal predischarge bilirubin assessment on gestational-age-adjusted nomograms and structured follow-up scheduling to catch infants whose risk window extends past the first hospital stay [5].

Cost-effective, accessible phototherapy is central to translating risk-factor knowledge into reduced neurologic injury in resource-limited settings. The AHRQ evidence report by Ip and colleagues found that while phototherapy reliably lowers bilirubin across gestational-age strata, evidence guiding thresholds in the smallest preterm infants remained comparatively sparse [29]. Rennie and colleagues' summary of the NICE guideline emphasizes structured risk-factor assessment at birth, including birth weight and gestational age, as the entry point for tiered monitoring and treatment [30]. Slusher and Olusanya both argue that combining risk-stratification tools with reliable phototherapy access and caregiver education offers the most direct path to narrowing the prevalence and severity gap between settings [19,26]. Low birth weight is thus not merely one risk factor among many but a central axis around which gestational immaturity, hemolysis, sepsis, and feeding inadequacy cluster and interact.

9. Conclusion

Neonatal jaundice is the near-universal clinical experience of low-birth-weight infants, and a substantial minority progress to hyperbilirubinemia severe enough to warrant phototherapy or exchange transfusion, with prevalence in hospital-based LBW cohorts ranging from roughly half to more than four-fifths of infants studied [15,16]. The physiological basis, reduced hepatic conjugation, lower serum albumin, delayed meconium passage, and comorbidity with sepsis and hemolytic disease, is well characterized, and gestational age, ABO/Rh incompatibility, G6PD deficiency, sepsis, and inadequate feeding emerge repeatedly as the principal modifiers of occurrence and severity [2,1,17]. The disproportionate burden observed in low- and middle-income settings reflects health-system limitations rather than differences in biology, and represents the most tractable target for reducing this largely preventable cause of neonatal neurologic injury [26,19]. Gestational-age-adjusted risk stratification at birth, structured post-discharge surveillance, feeding support, and reliable phototherapy access together constitute an evidence-based strategy for reducing both the prevalence and severity of neonatal jaundice in this vulnerable population [5,3].

10. References

1. Maisels, M.J., Bhutani, V.K., Bogen, D., Newman, T.B., Stark, A.R., Watchko, J.F. (2009) Hyperbilirubinemia in the newborn infant >/=35 weeks' gestation: an update with clarifications. Pediatrics, 124(4): 1193-1198. DOI: 10.1542/peds.2009-0329

2. Bhutani, V.K., Johnson, L., Sivieri, E.M. (1999) Predischarge screening for severe neonatal hyperbilirubinemia identifies infants who need phototherapy. Pediatrics, 103(1): 6-14. DOI: 10.1542/peds.103.1.6

3. Watchko, J.F., Tiribelli, C. (2013) Bilirubin-induced neurologic damage--mechanisms and management approaches. New England Journal of Medicine, 369(21): 2021-2030. DOI: 10.1056/NEJMra1308124

4. Shapiro, S.M. (2005) Definition of the clinical spectrum of kernicterus and bilirubin-induced neurologic dysfunction (BIND). Journal of Perinatology, 25(1): 54-59. DOI: 10.1038/sj.jp.7211157

5. Bhutani, V.K., Johnson, L.H., Keren, R. (2004) Diagnosis and management of hyperbilirubinemia in the term neonate: for a safer first week. Pediatric Clinics of North America, 51(4): 843-861. DOI: 10.1016/j.pcl.2004.03.011

6. Amin, S.B. (2011) Clinical assessment of bilirubin-induced neurotoxicity in premature infants. Seminars in Perinatology, 35(3): 135-141. DOI:

© 2026 Somato Publications. All rights reserved.

10.1053/j.semperi.2011.02.007

7. Wusthoff, C.J., Loe, I.M. (2015) Impact of bilirubin-induced neurologic dysfunction on neurodevelopmental outcomes. Seminars in Fetal and Neonatal Medicine, 20(1): 52-57. DOI: 10.1016/j.siny.2014.12.003

8. Dennery, P.A., Seidman, D.S., Stevenson, D.K. (2001) Neonatal hyperbilirubinemia. New England Journal of Medicine, 344(8): 581-590. DOI: 10.1056/NEJM200102223440807

9. Blencowe, H., Cousens, S., Oestergaard, M.Z., Chou, D., Moller, A.B., Narwal, R., et al. (2012) National, regional, and worldwide estimates of preterm birth rates in the year 2010 with time trends since 1990 for selected countries. Lancet, 379(9832): 2162-2172. DOI: 10.1016/S0140-6736(12)60820-4

10. Kaplan, M., Wong, R.J., Sibley, E., Stevenson, D.K. (2011) Neonatal jaundice and disorders of bilirubin metabolism. In: Christensen, R.D., Fanaroff and Martin's Neonatal-Perinatal Medicine, 9th ed. Elsevier, Philadelphia.

11. Gartner, L.M. (2001) Breastfeeding and jaundice. Journal of Perinatology, 21(Suppl 1): S25-S29. DOI: 10.1038/sj.jp.7210629

12. Ahlfors, C.E. (2001) Predicting bilirubin neurotoxicity in jaundiced newborns: Free bilirubin versus bilirubin-albumin binding. Current Opinion in Pediatrics, 13(2): 129-133. DOI: 10.1097/00008480-200104000-00006

13. Iskander, I., Gamaleldin, R., El Houchi, S., El Shenawy, A., Seoud, I., El Gharbawi, N., et al. (2014) Serum bilirubin and bilirubin/albumin ratio as predictors of bilirubin encephalopathy. Pediatrics, 134(5): e1330-e1339. DOI: 10.1542/peds.2013-1764

14. Hansen, T.W.R. (2011) Pathophysiology of kernicterus. Seminars in Fetal and Neonatal Medicine, 15(3): 174-179.

15. Olusanya, B.O., Ogunlesi, T.A., Slusher, T.M. (2015) Why is kernicterus still a major cause of death and disability in low-income and middle-income countries? Archives of Disease in Childhood, 99(12): 1117-1121. DOI: 10.1136/archdischild-2013-305506

16. Rasul, C.H., Hasan, M.A., Yasmin, F. (2010) Outcome of neonatal hyperbilirubinemia in a tertiary care hospital in Bangladesh. Malaysian Journal of Medical Sciences, 17(2): 40-44.

17. Mitra, S., Rennie, J. (2017) Neonatal jaundice: aetiology, diagnosis and treatment. British Journal of Hospital Medicine, 78(12): 699-704. DOI: 10.12968/hmed.2017.78.12.699

18. Ogunlesi, T.A., Dedeke, I.O., Adekanmbi, A.F., Fetuga, M.B., Ogunfowora, O.B. (2007) The incidence and outcome of bilirubin encephalopathy in Nigeria: a bi-centre study. Nigerian Journal of Medicine, 16(4): 354-359. DOI: 10.4314/njm.v16i4.37337

19. Slusher, T.M., Zamora, T.G., Appiah, D., Stanke, J.U., Strand, M.A., Lee, B.W., et al. (2017) Burden of severe neonatal jaundice: a systematic review and meta-analysis. BMJ Paediatrics Open, 1(1): e000105. DOI: 10.1136/bmjpo-2017-000105

20. Sgro, M., Campbell, D., Shah, V. (2006) Incidence and causes of severe neonatal hyperbilirubinemia in Canada. Canadian Medical Association Journal, 175(6): 587-590. DOI: 10.1503/cmaj.060328

21. Kaplan, M., Muraca, M., Vreman, H.J., Hammerman, C., Vilei, M.T., Rubaltelli, F.F., et al. (2002) Imbalance between production and conjugation of bilirubin: a fundamental concept in the mechanism of neonatal jaundice. Pediatrics, 110(4): e47.

22. Newman, T.B., Xiong, B., Gonzales, V.M., Escobar, G.J. (2000) Prediction and prevention of extreme neonatal hyperbilirubinemia in a mature health maintenance organization. Archives of Pediatrics and Adolescent Medicine, 154(11): 1140-1147. DOI: 10.1001/archpedi.154.11.1140

23. Keren, R., Luan, X., Friedman, S., Saddlemire, S., Cnaan, A.,

© 2026 Somato Publications. All rights reserved.

Bhutani, V.K. (2008) A comparison of alternative risk-assessment strategies for predicting significant neonatal hyperbilirubinemia in term and near-term infants. Pediatrics, 121(1): e170-e179. DOI: 10.1542/peds.2006-3499

24. Kaplan, M., Hammerman, C. (2004) Glucose-6-phosphate dehydrogenase deficiency: a hidden risk for kernicterus. Seminars in Perinatology, 28(5): 356-364. DOI: 10.1053/j.semperi.2004.09.001

25. Christensen, R.D., Yaish, H.M. (2013) Hemolytic disorders causing severe neonatal hyperbilirubinemia. Clinics in Perinatology, 42(3): 515-527. DOI: 10.1016/j.clp.2015.04.007

26. Olusanya, B.O., Kaplan, M., Hansen, T.W.R. (2018) Neonatal hyperbilirubinaemia: a global perspective. Lancet Child and Adolescent Health, 2(8): 610-620. DOI: 10.1016/S2352-4642(18)30139-1

27. Kuzniewicz, M.W., Wickremasinghe, A.C., Wu, Y.W., McCulloch, C.E., Walsh, E.M., Wi, S., et al. (2014) Incidence, etiology, and outcomes of hazardous hyperbilirubinemia in newborns. Pediatrics, 134(3): 504-509. DOI: 10.1542/peds.2014-0987

28. World Health Organization (2013) Pocket Book of Hospital Care for Children: Guidelines for the Management of Common Childhood Illnesses, 2nd ed. WHO, Geneva.

29. Ip, S., Chung, M., Kulig, J., O'Brien, R., Sege, R., Glicken, S., et al. (2004) An evidence-based review of important issues concerning neonatal hyperbilirubinemia. Pediatrics, 114(1): e130-e153. DOI: 10.1542/peds.114.1.e130

30. Rennie, J., Burman-Roy, S., Murphy, M.S. (2016) Neonatal jaundice: summary of NICE guidance. BMJ, 340: c2409. DOI: 10.1136/bmj.c2409

© 2026 Somato Publications. All rights reserved.