e- ISSN – 3117-3527
Print ISSN - 3117-3519

International Journal of Obstetrics and Gynaecology Nursing

IJOGN

Journal homepage: www.mcmed.us/journal/ijogn

NEONATAL JAUNDICE: COMPREHENSIVE ASSESSMENT AND PHOTOTHERAPY-BASED MANAGEMENT IN CLINICAL PRACTICE

Aruljothi P*

Assistant Professor, Department of Community Health Nursing, Sri Balaji College of Nursing, Cheranmahadevi, Tirunelveli, Tamil Nadu, India

Article Info

Received 20/01/2026; Revised 08/02/2026; Accepted 27/02/2026

Corresponding Author

Aruljothi P

Email: mailto:aruljothipj@gmail.com

Abstract

Neonatal jaundice is a common clinical condition witnessed in the first few weeks of postnatal phase and with a significant proportion of term and preterm infants across the world. It is caused by the high serum bilirubin level caused by the accelerated red blood cell turnover, hepatic conjugation incompleteness, and high enterohepatic flow. Most of the cases are physiological and self-limiting but a subgroup can develop severe hyperbilirubinemia resulting in acute bilirubin encephalopathy and kernicterus unless timely detected and treated. Comprehensive assessment includes the systematic clinical examination, risk factors identification, objective determination of bilirubin concentration in total serum bilirubin estimation and transcutaneous bilirubinometry analyzed with age-specific nomograms. Unconjugated and conjugated hyperbilirubinemia require separation between the two in order to adopt the right actions. Phototherapy is the mainstay of therapy in cases of unconjugated hyperbilirubinemia, which acts via photochemical mechanisms to convert bilirubin to water soluble isomers that are excreted without hepatic conjugation. The development of light-emitting diode (LED) and the fiber-optic technologies has enhanced the efficiency and safety of treatment. The proper management also requires close attention on the nursing care, such as checking the temperature, hydration, bilirubin level, and timely observing complications, such as dehydration, temperature changes, and rebound hyperbilirubinemia. The evidence-based, multidisciplinary, and family-centered approach is critical in the prevention of neurologic sequelae and maximizing the results of neonatal care.

Keywords: Neonatal jaundice; Hyperbilirubinemia; Phototherapy; Bilirubin monitoring.

INTRODUCTION

Neonatal jaundice is considered as one of the most common clinical conditions in the initial postnatal care, which has a high rate of incidence in both term and preterm infants globally and has continued to be a point of serious concern in terms of neonatal assessment and emergency intervention procedures. It is defined by yellowish uncoloring of the skin, sclera and mucous membranes due to high levels of bilirubin in the serum which is medically known as hyperbilirubinemia. [1]Though neonatal jaundice is commonly regarded as a physiological transitional phenomenon linked with the process of adapting to extrauterine living conditions, this occurrence may escalate into hyperbilirubinemia and kernicterus which are both preventable but devastating neurological disorders. Pathogenesis of neonatal jaundice is complex, and it affects increased bilirubin synthesis through increased turnover of

red blood cells, the immaturity of hepatic conjugation system, increased enterohepatic circulation, and in some instances, underlying pathological conditions, including hemolytic disease, infection, metabolic defects, or genetic deficits of enzymes.[2] During the neonatal stage, the liver is incapable of conjugating unconjugated bilirubin to the water-soluble form, and excrete it, due to hepatic immaturity specifically, the reduced activity of uridine diphosphate glucuronosyltransferase (UGT1A1), which facilitates this process. Moreover, prematurity, low birth weight, birth trauma, polycythemia, difficulties in breastfeeding, and incompatibility of the maternal and fetal blood groups may be among the risk factors which may markedly affect the bilirubin kinetics and increase the risk of serious hyperbilirubinemia. Because bilirubin levels are dynamic during the first week of life, a complex process, systematic clinical examination along with objective measurement methods (transcutaneous bilirubinometry and total serum bilirubin estimation) using age-specific nomograms and evidence-based treatment thresholds will be required.[3] Infants at risk can be detected early in their lives and hence the early intervention can prevent the advancement to neurotoxicity. Phototherapy has been the mainstay of the management of unconjugated hyperbilirubinemia, and phototherapy is a noninvasive, safe and highly effective way of lowering serum bilirubin levels by converting bilirubin to water-soluble photoisomeric forms which can be excreted without hepatic conjugation. Improved light-emitting diode (LED) systems and fiber-optic equipment have enhanced the effectiveness of the treatment and reduced side effects because of the progress in phototherapy technology. Nonetheless, the best results are preconditioned not only by the technological availability but also by very scrupulous nursing care, constant control, thermoregulation, hydration support and education of parents.[4] The failure to identify in time and poor surveillance remain some of the causes of avoidable morbidity in resource-constrained environments, where the importance of standard screening procedures and capacity building in the health care profession cannot be overstated. Thus, an efficient method of neonatal jaundice includes proper risk identification, prompt laboratory analysis, the reasonable application of phototherapy, close attention to complications, and the family-centered practice of care. Healthcare providers can protect the health of newborns by reducing long-term neurological consequences and improving the overall perinatal outcomes through the combination of clinical expertise and evidence-based guidelines to detect the problem early and manage it adequately.[5]

Overview of Neonatal Jaundice

Neonatal jaundice is the typical clinical case which is experienced in the initial stages of neonatal life and is characterized by the yellow discoloration of the skin, scleras, and mucous membranes because of the high level of bilirubin in the blood. It occurs in around 60 percent of term-infants and up to 80 percent of preterm infants during the first week of life which is one of the most commonly encountered conditions in newborn care. Bilirubin is a metabolic product of normal red blood cell breakdown and in newborns, hyperthyroid erythrocyte turnover is coupled with the immaturity of liver functioning, that results in temporary hyperbilirubinemia. Placenta also aid in the removal of bilirubin during fetal life but when the infant is born, it is the liver of the neonate that assumes the workload.[6] Due to incomplete development of hepatic conjugation systems, especially the presence of uridine diphosphate glucuronosyltransferase (UGT1A1), there is the risk of accumulation of unconjugated bilirubin in the blood. The onset of jaundice is clinically cephalocaudal, with the first appearance of the condition on the face, followed by a downward extension to the trunk and limbs as increased levels of serum bilirubin increase. Neonatal jaundice in the majority of cases is physiological and develops after the first 24 hours in life, reaching its peak at the third to fifth day in term children and disappearing automatically in one to two weeks without causing any harm.[2,7] Nonetheless, jaundice when it starts within a 24-hour, increases at an alarming rate, lasts longer than two weeks, or is accompanied by elevated bilirubin is pathological and needs urgent assessment. Unconjugated hyperbilirubinemia is most severe as unconjugated bilirubin is lipid-soluble and may cross the immature blood-brain barrier that causes acute bilirubin encephalopathy and kernicterus, which is permanent neurological injury, such as hearing loss, cerebral palsy, and cognitive impairment. A number of risk factors cause an increase of the likelihood of significant hyperbilirubinemia, they comprise prematurity, low birth weight, bruising during delivery, hemolytic diseases, e.g., Rh or ABO incompatibility, glucose-6-phosphate dehydrogenase deficiency, sepsis, dehydration, and poor breastfeeding. Nutrition at the early age is very important in the elimination of bilirubin by facilitating bowel movements and lowering enterohepatic circulation[7,8]. The diagnosis of neonatal jaundice is made by close physical examination in natural light, review of feeding habits and urine and stool, and objective data on assessing the level of bilirubin through the use of the transcutaneous bilirubinometry or total serum bilirubin. Bilirubin levels are interpreted based on standardized nomograms that take into account the gestational and postnatal age in hours. When carefully monitored and treated especially by use of phototherapy when necessary, severe complications have greatly been reduced. In general, neonatal jaundice is a process of physiological transition in the majority of newborns, yet close monitoring and evidence-based treatment are vital factors to avoid negative

neurological progress and guarantee the maximum well-being of a neonate [2, 9].

Types of Hyperbilirubinemia

Hyperbilirubinemia in newborn infants is widely categorized according to the kind of bilirubin that is raised in the blood and knowledge of these forms is important in proper diagnosis, risk management and assessment. There are two major types known as unconjugated (indirect) hyperbilirubinemia and conjugated (direct) hyperbilirubinemia, which vary in the pathophysiology, clinical implications, and treatment methods. The most common type of hyperbilirubinemia that is experienced during neonatal stage is the unconjugated type, which is experienced when there is excess production, inability of the liver to take up the bilirubin, inability to conjugate, or increased enterohepatic circulation[10]. In infants, elevated red blood cell turnover and underdeveloped hepatic enzyme systems, especially low levels of uridine diphosphate glucuronosyltransferase (UGT1A1) make a contribution to this condition. Normal examples of benign unconjugated hyperbilirubinemia include physiological jaundice which occurs after the first twenty-four hours of life and self-limits. Nevertheless, severe unconjugated hyperbilirubinemia may be caused by pathological factors including hemolytic disease of the newborn (including Rh or ABO incompatibility), glucose-6-phosphate dehydrogenase deficiency, hereditary spherocytosis, sepsis, cephalohematoma, polycythemia, and prematurity, which necessitates an urgent treatment. The fact that unconjugated bilirubin is lipid-soluble and may penetrating immature bloodbrain barrier makes this type of bilirubin especially worrisome as the bilirubin may cause bilirubin-related neurologic dysfunction and kernicterus when excessive. Conjugated hyperbilirubinemia, or direct hyperbilirubinemia, on the contrary, is less frequent, yet never harmless in newborns[11]. It is an occurrence of the liver to excrete conjugated bilirubin impaired into the bile ducts causing build-up of water-soluble bilirubin in the blood. In contrast to unconjugated bilirubin, conjugated bilirubin does not enter the bloodbrain barrier thus it does not lead to kernicterus, but its presence is an indicator of hepatobiliary impairment or systemic illness. Among others, neonatal hepatitis, biliary atresia,galactosemia or tyrosinemia, congenital infections, total parenteral nutrition -induced cholestasis, and hereditary cholestatic syndromes are common causes. In clinical terms, conjugated hyperbilirubinemia can be characterized by pale or clay stool, dark urine, hepatomegaly, and inadequate weight gain, which necessitate an urgent diagnostic testing, which entails liver function analysis, radiographic examination, and metabolic screening. Sometimes, a mixed pattern of hyperbilirubinemia can be observed and in these cases, both unconjugated and conjugated fractions are increased. It is important to distinguish the two kinds, as the management approaches vary greatly; in severe cases of unconjugated hyperbilirubinemia, the condition is typically treated with phototherapy or exchange transfusion, whereas conjugated hyperbilirubinemia is not and instead, a severe cause of the condition should be identified and removed.[12] Correct laboratory testing of the total and direct bilirubin levels will help clinicians identify the type of bilirubin and treat it accordingly and in time to avoid complications and achieve positive results of neonatal outcomes.

Pathophysiology

Pathophysiology of neonatal jaundice mostly revolves around the lack of balance between bilirubin production and elimination, which is especially susceptible to the neonatal changes through the intrauterine to extrauterine transition of life. Bilirubin is formed during degradation of hemoglobin released during the process of destroying senescent red blood cells. This process is magnified in neonates due to the fact that the mass of red blood cells per kilogram of body weight is higher and the erythrocyte lifespan is shorter (around 70-90 days versus 120 days in adults), which results in high bilirubin release. The resulting unconjugated bilirubin is lipid soluble and is transported by blood in the form of a complex with albumin.[13] Once born, the liver takes over the role of bilirubin metabolism which entails, hepatic uptake, conjugation and excretion. But the functional maturity of neonatal hepatocytes is low and the rate of the hydrolase uridine diphosphate glucuronosyltransferase (UGT1A1) that changes the unconjugated bilirubin to conjugated bilirubin water-soluble is highly diminished in the initial days of life. This enzymatic immaturity has the effect of inhibiting the ability of the liver to process bilirubin effectively leading to the build-up of bilirubin in the blood without a conjugation process. Moreover, the hepatic uptake proteins are not active and this further reduced clearance. Bilirubin is conjugated and then excreted in bile and is brought to the intestine. Late development of normal intestinal flora and slow gut motility in neonates increases enterohepatic circulation. The presence of beta-glucuronidase in the intestine of the newborn can deconjugate the bilirubin and the same is reabsorbed back into the bloodstream increasing the serum levels even more. This process can be made more difficult by factors related to breastfeeding, in particular, insufficient intake during the first days, which can postpone the passage of meconium and augment its reabsorption.[3] Other processes enhance the bilirubin build-up in a state of pathology. Accelerated red blood cell destruction occurs due to hemolytic conditions like Rh or ABO incompatibility, glucose-6-phosphate dehydrogenase deficiency, or hereditary spherocytosis which puts the liver significantly beyond its ability to handle bilirubin. Albumin-binding capacity may be decreased by hypoxia, acidosis, hypothermia, and some

drugs, which will elevate the percentage of free bilirubin in the bloodstream. Free unconjugated bilirubin is especially harmful as it may penetrate the immature blood-brain barrier and accumulate in the basal ganglia and nuclei of the brainstem causing bilirubin-induced neurologic dysfunction and in extreme cases, kernicterus. Premature birth has a contributive effect very low enzyme activity, low albumin concentration and high blood-brain barrier permeability also contributing to vulnerability. Therefore, neonatal jaundice is caused by a complicated combination of excessive production, defective conjugation, reduced excretion, and amplified reabsorption of bilirubin, and even neurotoxicity in case the serum concentrations exceed safe concentrations. Knowledge of these mechanisms is needed to identify them early, risk stratify, and promptly initiate therapeutic interventions to avoid the irreversible neurological damage.[8]

Physiological Jaundice

Physiological jaundice is a widespread and generally harmless phenomenon that is found in newborns, which is a normal transitional phenomenon in the process of adapting to life outside the womb. It normally ensues within the first 24 hours of birth, mostly between second and third day of life, peaks between the 3rd to 5th day in term babies (and a little later in preterm babies), and is self-limiting throughout the course of one to two weeks without the need to resort to aggressive treatment. Nature-impaired ratio of elevated bilirubin synthesis over restricted ability of the immature neonatal liver to conjugate and excrete bilirubin effectively is the main cause of physiological jaundice.[12] The red blood cell mass and the length of the erythrocyte lifespan in newborns is relatively higher than in adult individuals, causing increased breakdown of fetal hemoglobin after birth. The result of this process is greater amounts of unconjugated bilirubin. Simultaneously, hepatic enzyme systems, especially uridine diphosphate glucuronosyltransferase (UGT1A1), are not properly matured at the beginning of neonatal stages, and lower the capacity of the liver to turn lipid-soluble unconjugated bilirubin into water-soluble conjugated bilirubin and excrete it. Also, the reduced hepatic uptake and clearance capacity further reduces bilirubin clearance. The other factor is that there is an increase of enterohepatic circulation due to the delayed intestinal motility and low colonization of the neonatal gut by the bacteria that enables the deconjugation of bilirubin by beta-glucuronidase and again reabsorption into the bloodstream.[14] Physiologically, jaundice is a yellowish tint that spreads cephalocaudally that is, starting on the face and descending down to the chest, abdomen and extremities as the bilirubin concentration increases. Physiological jaundice levels of total serum bilirubin are typically less than 12mg/dL in full-term babies and may be a little higher in preterm ones, but remain within safe ranges based on standard age-specific nomograms. The infant usually looks healthy, is feeding well, has normal vital signs and no indication of underlying illness or hemolysis. Lab test does not show any sign of blood group incompatibility, infection, or metabolic disorder. Excessive bilirubin build-up is prevented by adequate breastfeeding which eases the passage of stool and decreases enterohepatic circulation. Close observation of bilirubin levels, maintenance of appropriate feeding and hydration and that there is no evidence of progression that goes beyond physiological boundaries are the main management factors. Phototherapy can be thought of in case the bilirubin level is close to treatment levels, though the majority of cases should be left untreated.[2] It is crucial to distinguish physiological jaundice and pathological jaundice, with early developmental impact during the first 24 hours, a rapid increase in bilirubin, lasting longer than two weeks, or an exorbitantly high bilirubin level as some factors that could be indicative of underlying pathology that needs to be addressed immediately. In general, physiological jaundice indicates the physiological adaptations of the newborn which is safe and successfully resolved, without any long-term effects when closely observed and appropriate care is taken.

Table 1: Comparison Between Physiological and Pathological Jaundice

Feature	Physiological Jaundice	Pathological Jaundice
Onset	After 24 hours of life	Within first 24 hours
Peak Level	Day 3–5 (term infants)	Rapid rise (>5 mg/dL/day)
Duration	Resolves within 1–2 weeks	Persists >2 weeks (term infant)
Bilirubin Type	Mostly unconjugated	Unconjugated or conjugated
Serum Bilirubin Level	Usually <12 mg/dL (term)	Exceeds age-specific threshold
Clinical Condition	Infant otherwise healthy	May show lethargy, poor feeding, anemia
Common Causes	Physiological transition	Hemolysis, infection, metabolic disorders
Risk of Kernicterus	Low	High if untreated

Table 2: Indications and Monitoring Parameters During Phototherapy

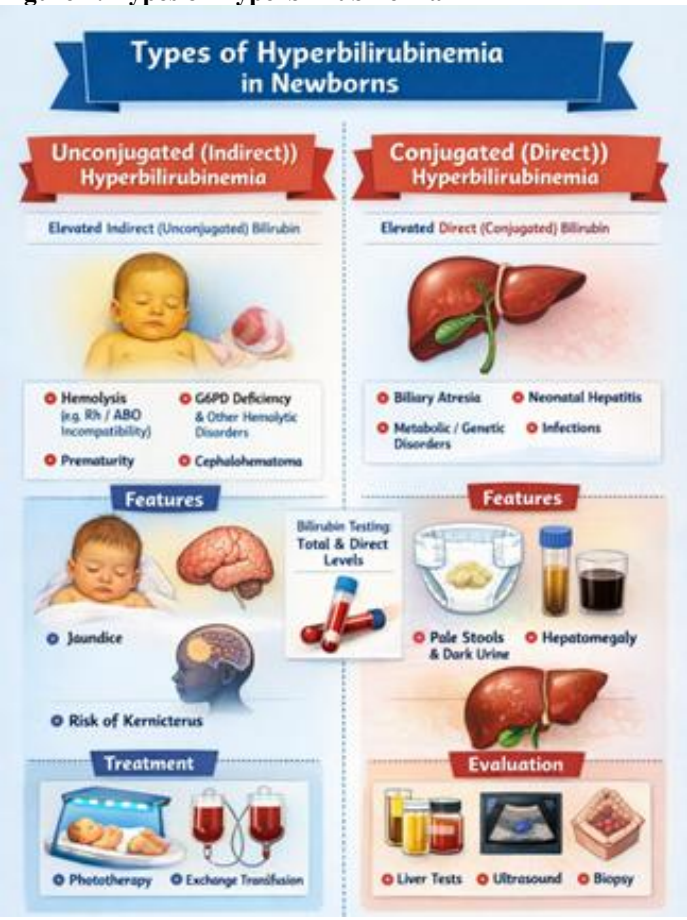
Parameter	Clinical Consideration	Nursing Responsibility

Initiation Criteria	Based on age-specific nomogram	Verify bilirubin threshold
Light Wavelength	430–490 nm (blue light)	Ensure correct device setting
Skin Exposure	Maximum surface area	Remove clothing except diaper
Eye Protection	Mandatory	Apply and check eye shields
Temperature	Risk of hyper/hypothermia	Monitor every 2–4 hours
Hydration	Increased insensible loss	Encourage feeding/IV fluids
Bilirubin Monitoring	Every 4–24 hours	Document and report trends
Rebound Monitoring	12–24 hrs after stopping	Schedule follow-up test

Table 3: Complications of Hyperbilirubinemia and Phototherapy

Category	Complication	Clinical Signs	Management
Neurological	Acute bilirubin encephalopathy	High-pitched cry, hypotonia, arching	Immediate escalation, possible exchange transfusion
Neurological	Kernicterus	Permanent motor/hearing deficits	Prevention is key
Fluid Balance	Dehydration	Weight loss, reduced urine output	Increase feeding/IV fluids
Temperature	Hyperthermia	Flushed skin, tachycardia	Adjust environment/light distance
Skin	Rash, tanning	Erythema	Continue monitoring
Rare	Bronze baby syndrome	Grayish-brown skin	Reassess therapy
Gastrointestinal	Loose stools	Increased stool frequency	Monitor hydration

Figure 1: Types of Hyperbilirubinemia



Pathological Jaundice

Pathological neonatal period jaundice is characterized as hyperbilirubinemia, which occurs due to a disease process and is highly dangerous in the case of severe complications, unless detected and addressed on time. In contrast to physiological jaundice, which occurs after 24 hours of age and has a predictable self-limiting course, pathological jaundice occurs within the first 24 hours of life, increases rapidly (more than 5 mg/dl/day) or has a serum bilirubin level that exceeds the recommended levels depending on the gestational age of the infant and the number of postnatal hours. It can also continue to exist after more than two weeks in term infants indicating that there is still pathology.[7] Hemolysis is by far the most frequent cause of pathological jaundice in which excessive destruction of red blood cells causes the overproduction of unconjugated bilirubin that exceeds the conjugating ability of the liver. The etiology that is well-known is hemolytic disease of the newborn related to Rh incompatibility or ABO incompatibility, where maternal antibodies pass through the placenta and destroy the fetal erythrocytes. The other causes of hemolytic are glucose-6-phosphate dehydrogenase deficiency, hereditary spherocytosis and other red cell membrane or enzyme defects. Some infections like neonatal sepsis may also have the same effect of affecting hepatic functioning and elevating bilirubin levels.[7,15] Also, birth trauma, cephalohematoma, polycythemia, as well as internal hemorrhage are associated with the elevated bilirubin production. Prematurity also increases the susceptibility because of immature hepatic enzymes and low albumin-

binding capacity. Conjugated (direct) hyperbilirubinemia might be present in some cases, which implies dysfunction of hepatobiliary. Conjugated pathological jaundice is caused by biliary atresia, neonatal hepatitis, metabolic disorders (galactosemia or tyrosinemia), congenital infection and cholestatic syndromes. Pathological jaundice can be clinically characterized by intense yellow discolouration of the palms and soles, poor feeding, lethargy, high pitch cry, hypotonia or hypertonia and severe cases can also include evidence of acute bilirubin encephalopathy including arching of the back (opisthotonus) and seizures. Pale and dark urine and stools can be indicative of conjugated hyperbilirubinemia. Laboratory testing normally involves total and direct serum bilirubin, blood grouping and Coombs test, complete blood count, reticulocyte count, peripheral smear, liver function testing and screening of metabolic or infectious etiologies. Early diagnosis is very important since high bilirubin concentrations of unconjugated bilirubin may penetrate the immature blood -brain barrier and accumulate in the brain tissues causing kernicterus, which is a physical cause of irreversible brain damage, which is preventable. Management is based on the severity and etiology and may need intense phototherapy, intravenous immunoglobulin in immune-mediated hemolysis, managing underlying infection or metabolic disorder and in severe cases exchange transfusion. Morbidity needs to be prevented by timely interventions, careful monitoring, and early diagnosis to achieve positive neonatal outcomes.

Serum Bilirubin Measurement

The measurement of serum bilirubin is one important part in the evaluation and management of neonatal jaundice as it offers objective and reliable means of ascertaining span of hyperbilirubinemia and is used to make decisions on how to treat it. Although it may be the case that clinical assessment by means of visual inspection may indicate the presence of jaundice, it is usually subjective and unreliable especially in preterm infants or infants with darker skin complexion. Thus, the accurate diagnosis and risk stratification of the bilirubin levels is impossible without quantitative measurements. Total Serum Bilirubin (TSB) estimation is the gold standard method which is acquired by studying a sample of blood in the laboratory. TSB is a reflection of the sum of the unconjugated (indirect) and conjugated (direct) bilirubin.[5,16] Fractionated bilirubin measurement is done in situations when one wonders whether the situation involves cholestasis or hepatobiliary disease to differentiate between direct and indirect components. High unconjugated bilirubin is linked with physiological or haemolytic factors, whereas high direct bilirubin indicates that of a pathological nature, e.g., biliary atresia or hepatitis neonatum. TcB has also been demonstrated to be a useful and noninvasive screening technique to supplement serum testing. TcB machines determine bilirubin concentration by analyzing the skin color and are especially effective in standard screening prior to the release of a patient. Nevertheless, high levels of TcB should be proved by a serum test, particularly where the levels are close to treatment levels.[17,18] Bilirubin level interpretation does not rely on absolute values alone but should incorporate the gestational age of the infant, birth weight, and postnatal hours of the child besides the existence of risk factors like prematurity, hemolysis, sepsis, or asphyxia. The determination of the levels falling into low risk zone, intermediate risk zone, and high risk zone and the decision of whether phototherapy or exchange transfusion is necessary is done using standardized nomograms, like hour-specific bilirubin charts. Serial measurements are usually necessary to follow trends, especially in infants under phototherapy or with rising bilirubin levels[19]. Timely intervention is achieved through close observation and complications like acute bilirubin encephalopathy are avoided. Safe neonatal care is comprised of proper techniques of sampling, proper documentation and timely reporting of key values. In such a way, the measurement of serum bilirubin is a key in evidence-based management of neonatal jaundice as it allows early identification, making a valid therapeutic choice, and preventing neurological consequences in the long term.

Phototherapy

Phototherapy is the most effective and the main modality of treatment in the management of unconjugated hyperbilirubinemia in neonates since it reduces greatly the chance of bilirubin-induced neurological injury in case the treatment is initiated at the right time. It is a painless, noninvasive and low-cost treatment that operates under the principle of exposing the skin of the infant to certain wavelengths of light which are usually in the blue spectrum of between 430 to 490 nanometers. This energy of light penetrates the skin and converts the unconjugated bilirubin that is stored in the subcutaneous tissues into water-soluble photoisomers which are excreted in the bile and urine without hepatic conjugation.[20,21] The efficacy of phototherapy varies with a number of factors such as the wavelength and intensity (irradiance) of the source of light, the size of area of the infants skin being exposed, the distance between the light and the infant, and the length of exposure. Light-emitting diode (LED) phototherapy units are favored as they have high irradiance, are energy efficient, produce little heat, and have a long lifespan in comparison to traditional fluorescent lamps. Fiber-optic phototherapy machines, including biliblankets, are even more flexible by creating the opportunity to be treated the whole day and time, and promoting parental bonding and breastfeeding.[22] Phototherapy is commonly started at the

age-specific levels of serum bilirubin according to the standardized nomograms that take into consideration the gestational age and the risk factors. In the course of treatment, the infant remains unclothed with the exception of a diaper to maximize the coverage of the skin and protective eye shields are used to avoid retinal injury. Proper hydration and regular feeding are promoted to help in increasing bilirubin excretion via urine and stool. Periodic monitoring of the serum bilirubin levels is used to evaluate treatment response and decide whether or not to continue or increase therapy. [21]Despite the overall safety of phototherapy, there are minimal side effects including loose stools, mild skin rash and increased insensible water loss as well as unstable temperatures. Uncommon complications are the bronze baby syndrome among infants with cholestasis. Phototherapy is stopped when the bilirubin levels reach safe levels, and rebound bilirubin levels can be re-examined to verify that things are stable. In general, phototherapy is the mainstay of the management of neonatal jaundice in terms of its effectiveness, safety profile, and the ability to avoid some severe outcomes like acute bilirubin encephalopathy and kernicterus when applied based on evidence-based guidelines.

Mechanism of Action

Phototherapy of neonatal jaundice works via the fact that certain wavelengths of light can be used to break up the unconjugated bilirubin into water-soluble structures which can be eliminated without being hepatically conjugated. Unconjugated bilirubin is lipid-soluble and rigidly bound to albumin in bloodstream but with increase levels, the non-bounded bilirubin is able to cross the immature blood-brain barrier and accumulate in nervous tissue, causing bilirubin-induced neural dysfunction.[21,23] Phototherapy makes use of blue light within the wavelengths of about 430 490 nanometers; this is the wavelength that the bilirubin absorption spectrum reaches its peak. In the event that the skin of the infant is exposed to such light, the bilirubin molecules of the dermal and subcutaneous tissues absorb the light energy and change their structure and configuration through photochemical reactions. Structural isomerization is the most significant reaction in this process that converts native bilirubin to lumirubin, a water-soluble isomer and can be quickly released in bile and urine without conjugation in the liver. This is an irreversible process that is thought to be the main route that causes the rapid decrease in serum bilirubin during phototherapy. [2]The other process is that of configurational isomerization in which bilirubin is converted into less toxic and more polar isomers that circulate in the bloodstream and are more readily excreted in the liver. Also, there is a small pathway called photo-oxidation and the results are small colorless polar molecules which are excreted via the urine. The photochemical processes rely on the intensity of light (irradiance), the surface area of the skin is exposed to, the period of treatment, and the distance between the light source and the infant. The higher the exposure to the light of the skin, the higher the bilirubin to be converted into light, thus improving the level of therapeutic efficacy.[20] Since phototherapy works on bilirubin that is in the skin and capillaries that are located at the surface, constant exposure is significant to maintain low levels of serum. Notably, phototherapy does not have any influence on the cause of hyperbilirubinemia, instead, it increases the speed of bilirubin removal and eliminates the toxic effect. Phototherapy is also a cornerstone intervention in the management of jaundice in infants and helps to prevent acute bilirubin encephalopathy and kernicterus because it quickly decreases the levels of unconjugated bilirubin.[12,24]

Types of Phototherapy Units

Phototherapy units that are applied in the treatment of neonatal hyperbilirubinemia differ in the modes of delivery of the therapeutic blue light that delivers optimum light at wavelengths of 430 to 490 nanometers to facilitate breakdown of bilirubin. Conventional or traditional phototherapy units use fluorescent lamps which are usually blue or special white tubes and placed over the infant in an open bassinet or incubator. They have been in use over many decades and are efficient in situations where there is sufficient irradiance and exposure of the skin; they produce more heat, need frequent replacement of the bulbs and may be less efficient in use than newer systems.[25] Over the last few years, there has been a shift towards the new light-emitting diode (LED) phototherapy units to be used in most of the neonatal care facilities because of their high-performing features. LED lights are of high intensity, narrow spectrum blue light with a consistent irradiance, low heat generation, reduced energy, and increased life. They are small, are robust as well as frequently come with a variable intensity control that enables clinicians to adjust therapy to the degree of hyperbilirubinemia severity. Another important type is fiber-optic phototherapy systems also known as biliblankets or phototherapy pads.[12] These devices have fiber-optic cables that transmit the blue light using a flexible pad which can be wrapped around or under the infant. Fiber-optic systems are especially useful since they provide an opportunity of more frequent interaction between parents, breastfeeding, and could be applied together with overhead phototherapy to provide intensive treatment. Intensive or double-surface phototherapy units incorporate overhead LED or fluorescent lights with extra sources of light which are placed under or around the baby and bring the maximum exposure of the skin, and the rate of bilirubin release is maximized. It is advisable that such systems be used when there are high bilirubin levels or

where bilirubin levels are rising at very high rates to prevent exchange transfusion. A few of the modern units have included irradiance control systems in order to achieve maximum therapeutic output.[20] A choice of a phototherapy unit is determined by the severity of clinical conditions, the resources available and institutional guidelines. The effectiveness is dependent on the type applied and sufficient exposure of the skin, appropriate positioning, and close observation irrespective of the type applied. In general, the development of phototherapy technologies has increased the efficiency, safety and comfort of treatment, which is one of the factors of enhanced neonatal outcomes in the management of hyperbilirubinemia.[4,6].

CONCLUSION

Neonatal jaundice is a common condition that requires careful, evidence-based management to ensure safe outcomes. While most cases are physiological, the risk of severe hyperbilirubinemia necessitates early identification, accurate bilirubin assessment, and differentiation from pathological causes. Phototherapy remains the primary and effective treatment, supported by advances such as LED and fiber-optic systems. However, successful management also depends on comprehensive nursing care, including monitoring hydration, temperature, and potential complications. Early detection, appropriate intervention, and family-centered care are essential to prevent serious outcomes like acute bilirubin encephalopathy and kernicterus. A multidisciplinary approach integrating clinical guidelines, technology, and vigilant care can significantly improve neonatal health outcomes and reduce long-term complications.

REFERENCES

1. Van der Geest, B. A. M., De Mol, M. J. S., Barendse, I. S. A., De Graaf, J. P., Bertens, L. C. M., Poley, M. J., et al. (2022). Assessment, management, and incidence of neonatal jaundice in healthy neonates cared for in primary care: A prospective cohort study. *Scientific Reports, 12*.
2. Hegyi, T., & Kleinfeld, A. (2021). Neonatal hyperbilirubinemia and the role of unbound bilirubin. *Journal of Maternal-Fetal & Neonatal Medicine, 35*, 9201–9207.
3. Yang, H., Lin, F., Chen, Z.-K., Zhang, L., Xu, J.-X., Wu, Y.-H., et al. (2021). UGT1A1 mutation association with increased bilirubin levels and severity of unconjugated hyperbilirubinemia in ABO incompatible newborns of China. *BMC Pediatrics, 21*.
4. Sarathy, L., Chou, J. H., Romano-Clarke, G., Darci, K. A., & Lerou, P. H. (2024). Bilirubin measurement and phototherapy use after the AAP 2022 newborn hyperbilirubinemia guideline. *Pediatrics, 153*.
5. Hulzebos, C. V., Vitek, L., Coda Zabetta, C. D., Dvořák, A., Schenk, P., Van der Hagen, E. A. E., et al. (2021). Screening methods for neonatal hyperbilirubinemia: Benefits, limitations, requirements, and novel developments. *Pediatric Research, 90*, 272–276.
6. Neonatology Society, Pediatrics Society. (2025). Guidelines for the diagnosis and treatment of common neonatal diseases in primary healthcare institutions: Neonatal jaundice (2025). *Chinese Journal of Contemporary Pediatrics, 27*.
7. Obeagu, E. I., & Obeagu, G. U. (2024). Antioxidants and the prevention of neonatal jaundice: A narrative review. *International Journal of Medical Science and Pharmaceutical Research, 10*, 28–34.
8. Zaman, B. U., Lone, P. A., Irtika, I., & Mir, N. Y. (2024). Clinico-etiological profile of neonates with jaundice in a tertiary care hospital of Northernmost India. *International Journal of Contemporary Pediatrics, 11*, 566–570.
9. Coquery, S. S., Georges, A., Cortey, A., Floch, C., Avran, D., Gatbois, E., et al. (2022). Discharge of newborns with risk factors of severe hyperbilirubinemia: Description of a hospital at home-based care monitoring and phototherapy. *European Journal of Pediatrics, 181*, 3075–3084.
10. Wang, Y., Wei, R., Zhao, W., & Zhao, C. (2023). Bilirubin removal by polymeric adsorbents for hyperbilirubinemia therapy. *Macromolecular Bioscience, 23*, 2200567.
11. Qian, S., Kumar, P., & Testai, F. D. (2022). Bilirubin encephalopathy. *Current Neurology and Neuroscience Reports, 22*, 343–353.
12. Inamori, G., Kamoto, U., Nakamura, F., Isoda, Y., Uozumi, A., Matsuda, R., et al. (2021). Neonatal wearable device for colorimetry-based real-time detection of jaundice with simultaneous sensing of vitals. *Science Advances, 7*.
13. Hansen, T. W. R., Wong, R. J., & Stevenson, D. K. (2020). Molecular physiology and pathophysiology of bilirubin handling by the blood, liver, intestine, and brain in the newborn. *Physiological Reviews, 100*, 1291–1346.
14. Akagawa, S., Akagawa, Y., Yamanouchi, S., Teramoto, Y., Yasuda, M., Fujishiro, S., et al. (2021). Association of neonatal jaundice with gut dysbiosis characterized by decreased Bifidobacteriales. *Metabolites, 11*, 887.
15. Zahed Pasha, Y., Alizadeh-Tabari, S., Zahed Pasha, E., & Zamani, M. (2020). Etiology and therapeutic management of neonatal jaundice in Iran: A systematic review and meta-analysis. *World Journal of Pediatrics, 16*, 480–493.
16. Aune, A., Vartdal, G., Bergseng, H., Randeberg, L. L., & Darj, E. (2020). Bilirubin estimates from smartphone images of

newborn infants' skin correlated highly to serum bilirubin levels. *Acta Paediatrica, 109*, 2532–2538.
17. Candel-Pau, J., Maya-Enero, S., Garcia-Garcia, J., Duran-Jordà, X., & López-Vílchez, M. Á. (2024). Transcutaneous bilirubin reliability during and after phototherapy depending on skin color. *European Journal of Pediatrics, 183*, 2819–2830.
18. Maya-Enero, S., Candel-Pau, J., Garcia-Garcia, J., Duran-Jordà, X., & López-Vílchez, M. Á. (2021). Reliability of transcutaneous bilirubin determination based on skin color determined by a neonatal skin color scale. *European Journal of Pediatrics, 180*, 607–616.
19. Zhao, Y., Zhang, W., & Tian, X. (2022). Analysis of risk factors of early intraventricular hemorrhage in very-low-birth-weight premature infants: A single-center retrospective study. *BMC Pregnancy and Childbirth, 22*.
20. Bhutani, V. K., Wong, R. J., Turkewitz, D., Rauch, D. A., Mowitz, M. E., Barfield, W. D., et al. (2024). Phototherapy to prevent severe neonatal hyperbilirubinemia in the newborn infant 35 or more weeks of gestation: Technical report. *Pediatrics, 154*.
21. Wang, J., Guo, G., Li, A., Cai, W.-Q., & Wang, X. (2021). Challenges of phototherapy for neonatal hyperbilirubinemia (Review). *Experimental and Therapeutic Medicine, 21*.
22. Choi, S., Jeon, Y., Kwon, J. H., Ihm, C., Kim, S. Y., & Choi, K. C. (2022). Wearable photomedicine for neonatal jaundice treatment using blue organic light-emitting diodes (OLEDs): Toward textile-based wearable phototherapeutics. *Advanced Science, 9*, 2204622.
23. Demirel, H. N., Ozumut, S. S., & Ovalı, H. F. (2024). Continuous versus intermittent phototherapy in treatment of neonatal jaundice: A randomized controlled trial. *European Journal of Pediatrics, 183*, 3389–3396.
24. Daggle, L., Sharma, N., Setiady, I., & Leonard, K. (2024). Management of neonatal hyperbilirubinemia: Shedding light on the American Academy of Pediatrics 2022 clinical practice guideline revision. *Pediatric Annals, 53*.
25. Van der Schoor, L. W. E., Verkade, H. J., Bertolini, A., De Wit, S., Mennillo, E., Rettenmeier, E., et al. (2021). Potential of therapeutic bile acids in the treatment of neonatal hyperbilirubinemia. *Scientific Reports, 11*.