

## DIAGNOSTIC APPROCH ON NEONATAL JAUNDICE

Aman K. Bondre\*, Samiksha R. Onkar, Prof. Vishnudas K. Lokhande, Dr. Rahul S. Bijwar and Dr. Laxmikant N. Barde

Jagadambha Institute of Pharmacy and Research, Kalamb.

Article Received on  
28 October 2024,

Revised on 18 Nov. 2024,  
Accepted on 08 Dec. 2024

DOI: 10.20959/wjpr202424-34921



\*Corresponding Author

Aman K. Bondre

Jagadambha Institute of  
Pharmacy and Research,  
Kalamb.

## ABSTRACT

Jaundice is most common physical abnormality in the first week of life. A significant proportion of term and preterm infants develop neonatal. Neonatal Jaundice is Prevalent condition by yellow discoloration of the skin sclera and mucous membrane due to elevated total serum bilirubin level. 60% of term and 80% of preterm infants develop jaundice in a first week of life. Newborn jaundice occurs when baby has high level of bilirubin in blood. The liver helps break down the substance so it can be removed from the body in the stool. Production of bilirubin as a result of degradation of haem arising from normal red blood cells. Phototherapy is a simple and effective way to reduce the bilirubin level. Few babies rapidly rising bilirubin level which risk to develop Kernicterus. High serum bilirubin or rapidly rising bilirubin level is treated urgently to avoid neurotoxicity.

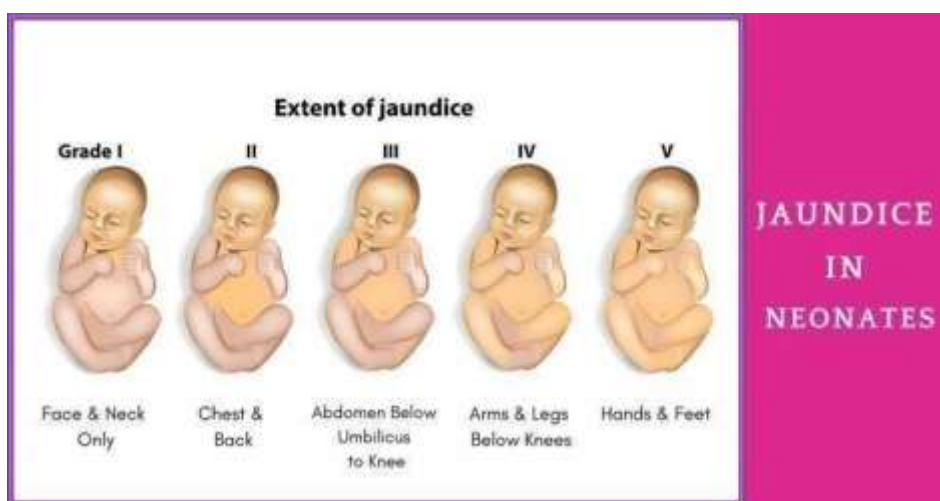
Implementation of neonatal jaundice care has been adversely affected by professional boundaries. Neonatal jaundice is common and usually benign early detection, prevents severe complications. Through physical examination, bilirubin level measurement, and risk assessment is critical. Treatment strategies include enhanced nutrition, phototherapy, exchange transfusion and intravenous immunoglobulin (IVIg), depending on the severity and underlying cause. Phototherapy remains the mainstay for treating hyperbilirubinemia, while exchange transfusion and IVIg are reserved for severe cases, particularly those involving immune-mediated haemolysis. **Background:** Neonatal jaundice is most common cause for intervention in newborn period. Transitory hyperbilirubinemia is present in almost all newborns. High serum level of bilirubin results in lethargy, poor feeding and Kernicterus of infant.

**KEYWORD:** Neonatal, Bilirubin, kernicterus, Bilirubin encephalopathy, phototherapy, paediatrics, haematocrit.

## 1. INTRODUCTION

### 1.1 What are Jaundice

Jaundice is a skin and conjunctiva of newborn infants result when unconjugated bilirubin accumulate to level that make yellow colour visible our eye.<sup>[1,2]</sup> Approximately 60% of term of full term babies and 80% of premature babies.<sup>[3],[4]</sup> Neonatal Jaundice Neonatal jaundice is clinically characterized by yellowish coloration of skin sclera and mucous membrane and caused by high total serum bilirubin level.<sup>[5][6]</sup>



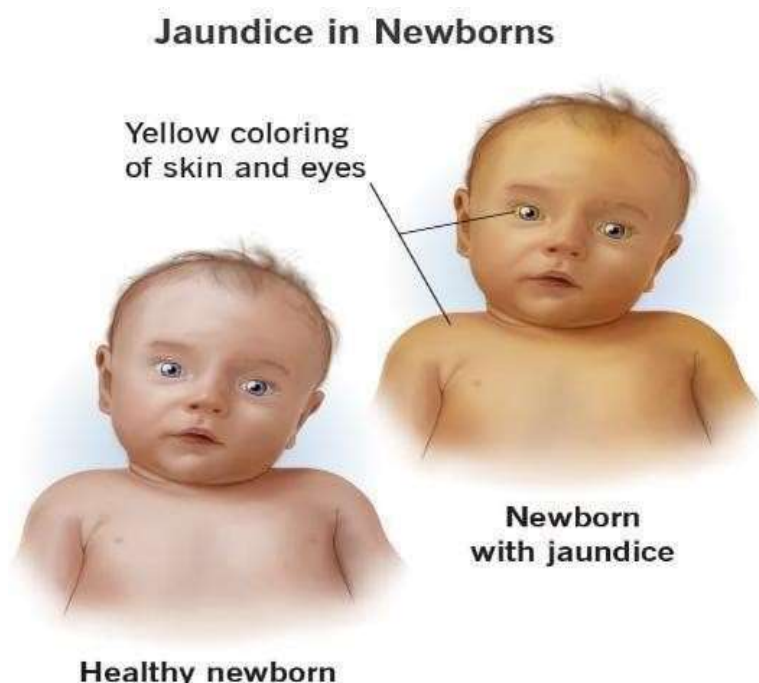
**Fig. No. 01: Newborn with Jaundice.**

Jaundice is thought to be visible at bilirubin in tissue including skin and mucous membrane. Newborn Jaundice can make the newborn sleepy and interfere with feeding.<sup>[7]</sup>

Neonatal jaundice can be best balance between production and elimination of bilirubin. Failure to recognize and manage neonatal jaundice could lead to bilirubin encephalopathy and newborn consequence.<sup>[8]</sup>

Phototherapy is widely used and accepted form of treatment for neonatal jaundice. It decreases serum bilirubin by converting fat soluble bilirubin into water soluble isomer.<sup>[9],[10],[11]</sup>

Breastfed newborn are more likely to develop jaundice<sup>[12]</sup>



**Fig. No. 02: Jaundice In Newborns.**

Hyper bilirubinaemia and Now it is widely used throughout the world. However, there are some cases which need exchange transfusion. With the declining incidence of Rhesus disease, ABO Incompatibility is said to be the commonest cause of haemolytic jaundice in the newborn, the outcome of which is comparatively good.<sup>[13]</sup>

A newborn has a bilirubin formation rate two to three times higher than that of an adult, largely Due to the high haematocrit and short life span of the newborn's red blood cells. Decreased bilirubin excretion is due to impaired ability of the neonatal liver to conjugate bilirubin and increased enterohepatic recirculation.<sup>[14]</sup>

## 1.2 HISTORY

### **Jaques Francois Eduoard Hervieux [1818-1900]**

In 1847 a thesis, on neonatal jaundice was submitted to the University of Paris which in many ways departed significantly in form and scope from the preceding works the same subject.



**Hervieux's Neuropathological observation**

Hervieux described brain Jaundice in 31 of 41 cases of neonatal jaundice. In all of these cases, clinical jaundice had been at its peak at the time of death, whereas in the 13 remaining cases, jaundice had only just appeared or was in the process of fading. He described intensity of brain jaundice as variable.

**Johannes Orth (1847-1923)**

The honor of having published the first description of pathoanatomical picture of kernicterus may belong to Johannes Orth.

**Orth's Description of kernicterus**

Orth work on neonatal jaundice was performed while he was still an assistant to Virchow. In his article, which primarily focused on pigment crystals in various organs, he described a term female infant who was born Nonicteric, but who became jaundiced soon after birth.

**Christian George Schmorl (1861 - 1932)**

To Christian Schmorl belongs the distinction of having coined term kernicterus (jaundice of the nuclei), which has subsequently been used both to describe a pathoanatomical picture seen at autopsy in those who died, as well as neurological syndrome in survivors of extreme jaundice.<sup>[15]</sup>

**2. ETIOLOGY**

Neonatal Jaundice is usually a Normal physiologic condition occurring during the transitional period afterbirth.

Early Jaundice - clinical jaundice within the First 24 hour of life is likely

To pathological and commonly as result of Isoimmunisation or other causes of significant

hemolysis Anti D-Prophylaxis is rhesus-negative mother can cause weakly positive DAT result as passive transfer of antibody.<sup>[16]</sup>

Bilirubin is formed from the catabolism of heme about 75% of bilirubin is derived from the breakdown of haemoglobin from aged red blood cell.

In first step heme is catalyzed by the membrane associated enzyme heme oxygenase (HO).<sup>[1]</sup>

There are at least two forms of enzyme: HO-1, the inducible form and HO-2 the constitutive form.<sup>[17]</sup>

The first enzymatic step requires molecular oxygen and NADPH donated from cytochrome P-450 system. Involves series of oxidation and reduction.<sup>[18,19,20]</sup>

- **Appearance** - The baby presents with yellowish appearance resulting from accumulation of bilirubin in skin, mucous membrane and conjunctiva or sclera.<sup>[21]</sup>
- **Clinical Significance** – Early onset with high peak level. Elevated conjugated bilirubin component.<sup>[22]</sup> Persists after the normal time for jaundice to resolve.<sup>[23]</sup>

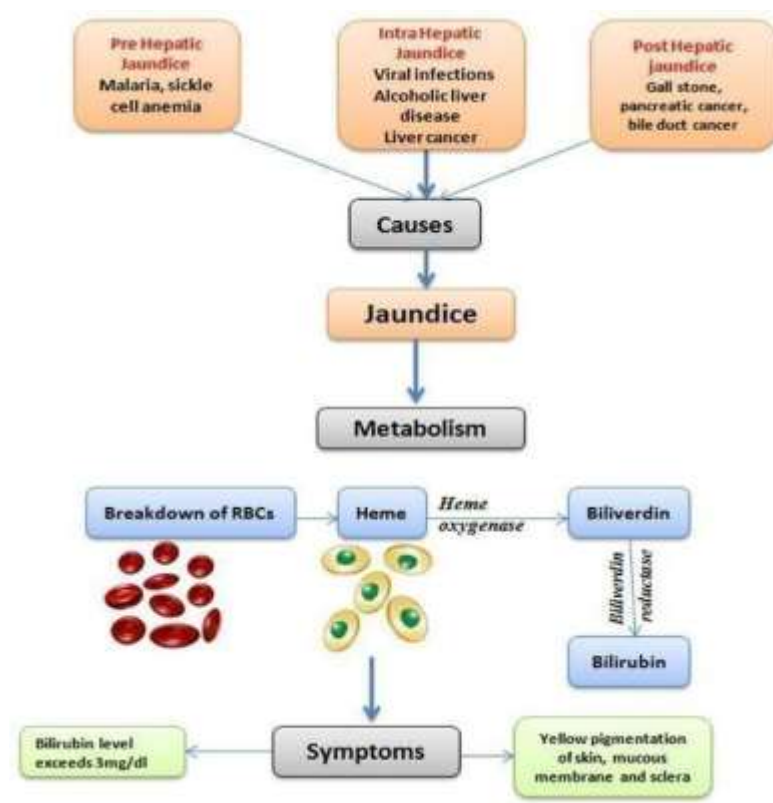


Fig. No. 03: Etiology of Neonatal Jaundice.<sup>[24]</sup>

### 3. Type of Neonatal Jaundice

Two type of Hyperbilirubinemia.

#### 3.1 Unconjugated Hyperbilirubinemia

Physiological jaundice account for 75% of newborn thy per bilirubinemia and caused by change bilirubinmetabolism in neonatal.<sup>[25]</sup>

Healthy adult have Normal total serum bilirubin. (TSB) / Level below 1mg/dl. In contract to neonateswhich TSB level are physiologically high.

- **It is subdivided into**

#### A. Increased bilirubin production

Haemolysis can be caused by Immune-mediated cred Non immune-mediated factor.<sup>[26]</sup>

Immune-mediated haemolysis - include blood group Incompatibilities such as ABO and RhesusIncompatibility.

#### B. Decrease Bilirubin clearance

Crigler -Najjar type I and II and Gilbert syndrome.<sup>[27]</sup>

#### C. Miscellaneous causes

Pathological hyperbilirubinemia in infant caused by various Factor as congenital hypothyroidism medicine like Swfa pharmaceutical ceftriaxone land penicillin intestinal obstruction, pyloric steNosis- breast milk Jaundice.<sup>[28]</sup>

In HDN Due to ABO incompatibility performed maternal Anti-A and Anti-B antibodies of immuNoglobulin and causes Haemolysis.<sup>[29,30]</sup>

Gilbert syndrome, Crigler -Najjar syndrome type-1 and classic Crigler -Najjar syndrome type 2 are three disorders caused abNormalities in UGT enzyme.

Breast Feeding and lactation jaundice dire causes of UHB in newborn.<sup>[31]</sup>

#### 3.2 Conjugated Hyperbilirubinemia

Conjugated hyperbilirubinemia also referred to as newborn cholestasis.<sup>[32]</sup>

DISH guishing cholestatic jaundice CHB from UHB is critical because is almost pathologic andwarrant prompt evaluation and treatment.

## 1. Causes of Neonatal cholestasis

### A. Obstruction of biliary Flow

Neonatal cholelithiasis, biliary atresia, neonatal sclerosing cholangitis.

Biliary atresia is a condition that blocks flow of bile from liver to small intestine in newborn, causing jaundice.

**B. Infection** - CMV, HIV, Rubella, herpes, virus syphilis, septicemia, UTI.

### C. Genetic Causes

- Bile acid synthesis disorder
- Galactosemia
- Tyrosinemia type-I<sup>[33]</sup>

## 4. CAUSES OF NEONATAL JAUNDICE

### 4.1 Pathological Jaundice

The early onset of jaundice is a risk factor for severe hyperbilirubinemia requiring immediate treatment.<sup>[34]</sup>

Pathological jaundice is the sum of bilirubin produced by mechanism plus any added Physiological mechanism of bilirubin by pathological insult Production.<sup>[35]</sup>

Haemolysis can cause blood extravasation, haemorrhage, (eg, cerebral) isoimmunisation.<sup>[36]</sup>

### 4.2 Physiological jaundice

In the first week of life most babies have total serum bilirubin that exceeds the upper limit Normal for adult.<sup>[37]</sup> Preterm infant exhibits a highest peak serum bilirubin Concentration occurring on day 3-6 and longer declining phase.<sup>[38]</sup>

High haemoglobin concentration in newborn infants - Immaturity of hepatic uptake, transport and conjugation system - Shorter lifespan for neonatal red blood cells compared to those of adults - Increased level of beta-glucuronidase in the gut. – releasing more unconjugated bilirubin to enter the enterohepatic circulation.<sup>[39]</sup>

### 4.3 Prolonged jaundice

Present in 15-40% of well breastfed babies 2 weeks of age and 9% of well breastfed Babies at 4 weeks of age.<sup>[40]</sup>



## 5. PATHOPHYSIOLOGY

The Normal destruction of circulating erythrocytes accounts for 75% of the daily bilirubin production in the newborn.<sup>[41]</sup>

Treatment for neonatal jaundice usually involves phototherapy to convert unconjugated bilirubin to a water-soluble form that can be excreted in the urine.<sup>[42]</sup>

Newborns have higher haemoglobin levels at birth, a shorter red blood cell life span, and a reduced conjugating ability of the newborn liver, which leads to higher total serum bilirubin levels than adults.

Hyperbilirubinemia leads to neurotoxicity by a number of mechanisms that include; Cellular death by interfering with DNA synthesis.

Disruption of protein synthesis and phosphorylation.

Impairment of nerve conduction (particularly the auditory nerve) and By inhibition of ion exchange and water transport in renal cells leading to neural swelling.

This unconjugated bilirubin is hydrophobic and is transported in circulation to the liver bound to albumin, where it is conjugated with glucuronic acid in the smooth endoplasmic reticulum by the enzyme uridine diphosphate-glucuronosyltransferase (UGT).<sup>[43]</sup> neurologic dysfunction (BIND) and bilirubin encephalopathy.<sup>[44]</sup> Deficient bile secretion in cholestasis results in malabsorption of fat and fat-soluble vitamins that often leads to failure to thrive with vitamin A, D, E, and K deficiencies.<sup>[45]</sup>

### 5.1 Diagnosis

Clinically, the differential diagnosis of neonatal jaundice varies during the first weeks of life. For instance, because virtually all neonates have decreased conjugation and excretion capabilities at birth, hyperbilirubinemia in the first 1–3 days of life almost always reflects an increase in bilirubin production.<sup>[46,47,48]</sup>

### 5.2 Clinical Assessment

This method is less accurate and more subjective in estimating jaundice.<sup>[49]</sup> parents should be counselled regarding benign nature of jaundice in most neonates, and for the need to be watchful and seek help if baby appears too yellow.<sup>[50]</sup>



### 5.3 Visual examination

Your baby will have a visual examination to look for signs of jaundice. They need to be undressed during this so their skin can be looked at under good, preferably natural, light.

### 5.4 Bilirubin Test

If it's thought your baby has jaundice, the level of bilirubin in their blood will need to be tested. A small device called a bilirubin Nometer, which shines light on to your baby's skin (it calculates the level of bilirubin by analysing how the light reflects off or is absorbed by the skin).

### 5.5 Ingram Icterometer

The instrument is pressed against the Nose and the yellow colour of the blanched skin is matched with the graded yellow lines and bilirubin level is assigned.<sup>[51,52]</sup>

### 5.6 Additional Test

These tests can include blood tests to determine the specific type of jaundice, such as a complete blood count (CBC) and blood group testing.<sup>[53,54]</sup>

### 5.7 Monitoring

This can be done through regular follow-up appointments and bilirubin level checks. Monitoring is important to ensure that the bilirubin levels do Not reach dangerous levels that could potentially harm the baby's brain.<sup>[55,56]</sup>

## 6. EPIDEMIOLOGY

In one study of all birth in India had significant neonatal jaundice. The incidence was almost three time higher in babies with low birth weight.

A few years ago it was Noted that 25% of newborn admission and readmission to hospitals in Pakistan were due to neonatal jaundice.<sup>[57][58]</sup>

However, the incidence of kernicterus is significantly higher in developing countries.<sup>[59]</sup> Conjugated hyperbilirubinemia is much less frequent than unconjugated hyperbilirubinemia and is almost always pathological.<sup>[60,61,62]</sup>

ABO incompatibility followed by G6PD deficiency is the most frequently identified cause identified.<sup>[63]</sup>

Newborns with Southeast and Far East Asian ancestry have higher recorded TSB levels than their White and African counterparts.<sup>[64,65][66]</sup>

### **6.1 Factors that influence the epidemiology of Neonatal jaundice**

A comparison of the different ethnic groups in Singapore in the 1960s found clinical NJ in 90% of Chinese infants in the 1st week of life compared to 70% in Malays, and 30% European infants.<sup>[67][68]</sup>

In a study in Nepal (n=18,985) Infants of Madhesi ethnicity (originating from the plains) had a decreased risk of jaundice compared to infants of Pahadi (originating from the hills) ethnicity [RR=0.21 (95% CI: 0.18–0.25)].<sup>[69]</sup>

The onset of NJ was on the 1st day of life in 12.8%, and between the 2nd–4th days in 72.4% of the cases.<sup>[70]</sup>

The timing of presentation of HB differed between ethnic groups. This occurred in 64% of Caucasians and 55% of Asians respectively Events during pregnancy.<sup>[71]</sup>

### **6.2 Maternal smoking**

Hardy and Mellit first suggested that maternal smoking reduced NJ, however the sample size was small and they did not control for other factors that might influence TSB level.<sup>[72]</sup>

Future studies need to control better for breast-feeding as a possible confounder, as smoking mothers may breastfeed less than Nonsmokers.<sup>[73]</sup>

### **6.3 Maternal Age and illness**

Thus, while some found the incidence to be increased in older mothers others found the highest risk in infants of younger mothers particularly in those <20 years of age.<sup>[74]</sup>

### **6.4 Maternal pharmacotherapy**

Pregnant women phenobarbital has been shown induce hepatic processing of bilirubin in the fetus In Malawi infants born to HIV-positive mothers who had received a 6-week course of nevirapine to reduce mother-to-infant virus transfer, were shown to have significantly reduced incidence of NJ compared to infants of mothers who were HIV negative and had not received nevirapine.<sup>[75]</sup>

### 6.5 Blood group incompatibility

Because bilirubin is the end product of haeme catabolism, increased breakdown of erythrocytes, as occurs in all kinds of haemolytic Anemia, increases bilirubin production causing neonatal jaundice.<sup>[76]</sup>

### 6.6 Birth weight

Low birth weight is also associated with increased risk for neonatal jaundice.<sup>[77,78]</sup>

### 6.7 Nutrition colouric intake, fluid

A proportion of breast-fed infants exhibit exaggerated and prolonged HB during the first days and weeks of life and breastfed infants had significantly greater need for PT than controls It may take from 1–4 months of slowly declining TSB levels before values Normalize.<sup>[79]</sup>

### 6.8 Polyglobulia / polycythemia

High haematocrit during the first days of life is associated with increased risk of NJ/HB However, late cord clamping had No effect on TSB course or the need for PT, though the haematocrit increased.<sup>[80]</sup>

## 7. RISK FACTORS OF NEONATAL JAUNDICE

1. **Genetic conditions:** Certain genetic disorders can affect the liver's ability to process bilirubin effectively.
2. **Infection during pregnancy or after birth:** Infections can disrupt liver function and contribute to jaundice.
3. **Difficulties with breastfeeding:** Inadequate milk intake can lead to dehydration and increased bilirubin levels.
4. **Maternal diabetes:** Babies born to diabetic mothers have a higher risk of developing jaundice.
5. **Rh disease:** Incompatibility between the mother's and baby's blood types can lead to jaundice.
6. **Male gender:** Boys are more likely to develop jaundice than girl.<sup>[81,82,83]</sup>

## 8. Clinical manifestation

- A. Bilirubin encephalopathy - Brown Urine ,Pale stool, Sleepiness
- B. Fever, High pitch cry, Poor Feeding -Massive enlargement of the liver and spleen.<sup>[84]</sup>
- C. However, if you Notice any of these signs or if the jaundice appears to be worsening.<sup>[85]</sup>

## 9. INVESTIGATION

### 9.1 Measurements of Bilirubin

A baby's TSB or TCB and gestation are good predictors of hyperbilirubinaemia risk. There is insufficient evidence available to support universal bilirubin screening to prevent chronic bilirubin encephalopathy and some evidence of harm.<sup>[86]</sup> If TCB is greater than 250micromol/L or less than 50 micro mol / L below threshold for phototherapy measure the TSB.<sup>[87,88]</sup> Clinical decision regarding treatment is based on TCB trend and Not one value.<sup>[89]</sup>

### 9.2 TCB Meter

Estimates bilirubin levels in the skin from wavelength patterns of light reflected from the skin and subcutaneous tissues.<sup>[90]</sup>

### 9.3 Precautions

Jaundice is prolonged or there is conjugated hyperbilirubinaemia Baby receiving phototherapy accuracy is unknown and may overestimate/underestimate level.<sup>[91]</sup>

### 9.4 Pathological Jaundice Investigation

A baby who is thriving and feeding well requires fewer investigations than an unwell baby who is Not thriving. history Check maternal antenatal screening for:  
ABO Rh D group - Red cell antibodies

### 9.5 Haematology

Appears excessively ruddy Has risk factors (e.g. maternal smoking, significant embryonic growth restriction, maternal diabetes) - ABO and Rh D—extended typing may be indicated if there are other maternal antibodies.

### 9.6 Infection

Investigate for congenital infections e.g. clinical signs of suggestive history, severe jaundice, elevated conjugated bilirubin thrombocytopenia.<sup>[92]</sup>

### 9.7 Prolong jaundice investigation

The most common cause of prolonged jaundice is breast milk jaundice occurring in up to 30% wellbreastfeeding babies.<sup>[93,94]</sup>

Progression of early Jaundice – History. Weight gain, Feeding, Thyroid Function test<sup>[95]</sup>  
Recurrent or new presentation of jaundice - Microscopy and culture urinary tract infection is a

potential cause of prolonged jaundice, CMV, Reducing substances—present Galactosemia, Genetic, Family History, RBC metabolism disorder, Test for Glucuronosyltransferase deficiency disorder test for red cell membrane disorder.<sup>[96]</sup>

### 9.8 Other investigations may include

Reducing substance in urine test to screen for galactosemia, Blood gas measurements to assess the risk of bilirubin CNS toxicity Hepatobiliary scintigraphy to assess the function of the biliary tract.<sup>[97]</sup>

## 10. MANAGEMENT / TREATMENT

### 10.1 Medication

- **Phenobarbital** Phenobarbital increases the conjugation and excretion of bilirubin. It reduces serum bilirubin levels by at least 25%. The drug functions by means of phenobarbital-responsive enhancer module that stimulates the gene for UGT 1A1 to induce production of bilirubin- conjugating enzyme.
- **Salicylates** salicylates and other analgesics should be avoided in newborns with significant jaundice because they compete with bilirubin for binding sites on albumin.
- **Ibuprofen** Ibuprofen should be used with caution in premature infants with significant jaundice because it can displace bilirubin from albumin binding sites. This could increase the risk of bilirubin encephalopathy.
- **Furosemide** furosemide, a sulphonamide diuretic, has been recommended for use in the newborn infant, a study was made of its effect on the bilirubin-binding capacity of albumin. Furosemide was compared to Sulfisoxazole a known displacer of bilirubin.<sup>[98,99]</sup>

### 10.2 Nutrition

- **Breastfeeding** - Breastfed babies are more prone to developing prolonged jaundice than formula fed babies if there is inadequate milk production - Encourage breastfeeding—baby may need to feed 8–12 times per day - Offer breastfeeding support - Routine supplementary feeds Not recommended even if having phototherapy—Most newborns with jaundice can continue breastfeeding.<sup>[100]</sup>
- **Hydration** -It's important to maintain Normal hydration for a jaundiced newborn. This can be done by encouraging breastfeeding, providing additional oral fluids, or administering fluids intravenously

- **Intravenous Fluid**-Intravenous (IV) fluids are often used to treat neonatal jaundice in infants who can't be fed orally.

### 10.2.1 Some other things to consider include

- **Hyponatremia**-This is when serum sodium levels are less than 130 mEq/L. It can be caused by excessive free water intake or inadequate sodium intake. - Water or glucose water supplementation this is Not recommended because it can interfere with breastfeeding and may cause hyponatremia.<sup>[101]</sup>

## 10.3 Exchange Transfusion

Exchange transfusion was pioneered by Wallerstein and Diamond in 1940.

In cases where phototherapy fails to achieve the desired results, a complete blood transfusion may be deemed necessary.

Exchange transfusion (ET) was the first successful treatment ever used for jaundice and is now the second-line treatment for severe unconjugated hyperbilirubinemia.<sup>[102]</sup>

Absence of bilirubin in the fresh blood leads to a rapid decrease in the total bilirubin level in the baby's blood.<sup>[103]</sup>

TSB levels immediately following ET is about 60% of the pre-exchange level that later increase to 70 to 80% of pre-exchange levels as a result of equilibrium with an extravascular moiety of bilirubin. During ET, vitals should be monitored closely, and TSB, CBC, serum calcium, glucose, and electrolytes need to be checked following procedure- ➡

Exchange transfusion should be performed in a specialized unit having intensive care facilities and appropriate expertise. Double volume (2 x 80 ml/kg), fresh (<7 days old), having a haematocrit 45-50% need to be used.<sup>[104]</sup>

- **Supplementation**

Probiotics supplementation therapy may be an effective and safe treatment option for pathological neonatal jaundice.

- **Ferrous Sulphate**

Use with caution in any baby who has a haemolytic condition (endogenous iron stores may be high Not low) Undertake iron studies before commencing treatment to confirm iron deficiency and absence of iron overload.<sup>[105,106]</sup>

- **Folic Acid**

Where there has been a high red cell turnover. Dose: 50–100 microgram/kg/day Commence from seven days of age.<sup>[108],[107]</sup>

#### 10.4 Phototherapy

The discovery of light as a therapy for neonatal hyperbilirubinemia was made in 1958 by a nurse in UK who observed remarkable disappearance of jaundice on exposing the baby to sunlight.



**Fig. No. 04: Babies Under Phototherapy.**

Is the use of visible light for the treatment of hyperbilirubinemia in the newborn. The dose of phototherapy is a key factor in how quickly it works; dose in turn is determined by the wavelength of the light, the intensity of the light (irradiance), the distance between the light and the baby, and the body surface area exposed to the light.<sup>[109]</sup>

The outcome of phototherapy is improved in our resource limited settings by optimizing factors that affect the efficiency of phototherapy. These include:

- **Spectrum of light**

Light penetrates the skin well and is absorbed maximally by bilirubin in blue green spectrum,



but a combination of white and blue light is used for convenience of examination of the baby during phototherapy without removing the baby from the cot.

- **Spectral irradiance**

Spectral irradiance should be measured for quality checks from time to time. It serves two objectives

- a) Reminder to change bulbs/light source when their life is near expiry and
- b) To help staff optimize irradiance for the baby

- **Spectral Power**

The greater the surface area irradiated, the more bilirubin molecules will be impacted by light.<sup>[110]</sup>

- **Duration of phototherapy**

The higher the total serum bilirubin, the more rapid is the decline with phototherapy. Evidence suggests that phototherapy can be stopped once total serum bilirubin falls by 50  $\mu$  mol/litre (3 mg/dl) below the phototherapy range on a time specific graph.<sup>[111]</sup>

- **Care of an infant under phototherapy**

While an infant is receiving phototherapy, breast feeding needs to be continued every 2 to 3 hours.

If there is a difficulty in establishment of breast feeds and baby has lost >10% of birth weight, breast feeding should be supplemented with either a formula or expressed breast milk by a naso or an orogastric tube.<sup>[112]</sup>

## 10.5 Care during phototherapy

**Table No. 01.**

Clinical Care	<ul style="list-style-type: none"> <li>- If possible do Not separate mother and baby during phototherapy</li> <li>o Provide information to support the woman and family during treatment and/or phototherapy</li> <li>- Nurse baby with only a nappy and fold down to exposure maximum skin surface area<sup>[113]</sup></li> <li>- If baby has loose stools, consider the use protective barrier creams on buttocks.</li> <li>- Use eye protection</li> <li>o Lubricating eye drops may be indicated.</li> </ul>
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	<ul style="list-style-type: none"> <li>○ Monitor for eye discharge, conjunctivitis and eye protection placement</li> <li>- Continuously observe baby</li> <li>- Monitor baby's temperature:</li> </ul>
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## 10.6 Phototherapy in home

Table No. 02.

Inclusion	Discuss with parent their motivation, abilities and understanding of safer sleeping principle. Unconjugated hyper bilirubinaemia
Exclusion	<ul style="list-style-type: none"> <li>- Poor feeding</li> <li>- Temperature instability</li> <li>- Asphyxia/acidosis</li> <li>- Lethargy</li> </ul>
Parent Information	<ul style="list-style-type: none"> <li>- Safer infant sleeping principle</li> <li>- when seek advice</li> <li>- Temperature management and monitoring<sup>[114]</sup></li> </ul>



Fig. No. 05: Phototherapy in Home.

## 11. OTHER TREATMENT

Intravenous gamma globulin has been shown to significantly reduce the need for exchange transfusion in ABO and Rh haemolytic disease. In cases, when total serum bilirubin is rising despite intensive phototherapy, IVIG (0.5-1 gm/kg) over a period of 4 hrs may prove helpful (evidence quality B; benefits exceed harms.<sup>[115,116]</sup>

## 12. CONCLUSION

Neonatal jaundice is relatively common and characterised by hyperbilirubinemia.

Neonatal jaundice is common and usually benign, early detection and appropriate management are vital to prevent severe complications. Continued research and education are essential to optimize outcome for affected infants. Babies diagnosed with both unconjugated jaundice and conjugated jaundice should be evaluated and treated in collaboration with physician who has liver experience.

Neonatal jaundice is the most common condition needing medical attention in the neonatal period. The majority of these cases present with unconjugated hyperbilirubinaemia and most infants respond well to phototherapy when the bilirubin level reaches the treatment threshold. Infants with risk factors.

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