

RESEARCH ARTICLE

Available online at http://www.journalcra.com

International Journal of Current Research Vol. 13, Issue, 04, pp.17004-17014, April, 2021

DOI: https://doi.org/10.24941/ijcr.41119.04.2021

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

OPEN ACCESS

A CROSS-SECTIONAL STUDY OF MANAGEMENT OF NEONATAL JAUNDICE

*Chaudhary Veena

Assistant Professor, Department of Physiology, World College of Medical Sciences & Research and Hospital, Jhajjar, Haryana, India, Pin-124103

ARTICLE INFO

ABSTRACT

Article History: Received 25th January, 2021 Received in revised form 20th February, 2021 Accepted 19th March, 2021 Published online 24th April, 2021

Key Words:

(PT)-preterm, (AGA)-appropriate for gestational age, (SGA)-small for gestational age, (NW)-Normal weight -(2.5kg-3.99kg), (LBW)- low birth weight (1.5kg-2.499kg), (VLBW)- (1kg-1.499kg), (EONS)-early onset neonatal sepsis, (Inc)-ABO incompatibility, (TSH)-Thyroid Stimulating hormone, (IDM)-Infant of Diabetic Mother, (TSB)- Total Serum Bilirubin and (NNH)-Neonatal Hyperbilirubinemia. Objectives: In this study, our objectives were to facilitate early diagnosis and reduce subsequent complications of neonatal jaundice by appropriate treatment. Methods: The study was done as Random Sampling of neonates noticed to have deep yellow discolouration of whole body that were born in Rockland Hospital, Delhi, during the period 01.01.2012 till 07.08.2014. Results: In our crosssectional study of 661 neonates, the results showed that the prevalence of physiologic jaundice in neonates was 86.99% and the prevalence of pathologic indirect hyperbilirubinemia in neonates was 13.01%. Physiological jaundice in newborns was transient and dissipated without medical intervention. Sunbathing was advised in all cases of physiologic jaundice. Bilirubin count is also lowered through excretion — bowel movements and urination —so frequent and effective feedings were vital measures to decrease jaundice in infants. In pathologic jaundice cases when serum bilirubin levels were greater than 12 mg/dL, infant was treated with single surface or double surface phototherapy, depending on the infant's age and prematurity status. All the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis, so they were given antibiotics, phototherapy and other supportive treatment. No neonate with jaundice required exchange transfusion or liver transplantation. Conclusions: No neonate discharged from hospital was reported to have hydrops foetalis, kernicterus or cerebral palsy during admission or on follow up. Besides, the Neonatal Mortality Rate was zero, in our study. Thus, it is evident in our study that the morbidity and mortality are completely preventable in neonates by appropriate management of neonatal jaundice and associated risk factors.

Copyright © 2021. *Chaudhary Veena.* 2021. *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.*

Citation: Chaudhary Veena. "A cross-sectional study of management of neonatal jaundice", 2021. International Journal of Current Research, 13, (04), 17004-17014.

INTRODUCTION

The word 'jaundice' comes from the French word 'jaune', meaning 'yellow' and 'jaunisse' meaning "yellow disease". (11) The medical term for jaundice is icterus. The word 'icterus' comes from the Greek word 'ikteros'.(7) The origin of the word icterus is quite bizarre, coming from an ancient belief that jaundice could be cured from looking at the yellow bird icteria.(11) The term icterus is sometimes incorrectly used to refer to jaundice specifically of sclera.(7)(11)

*Corresponding author: Chaudhary Veena

(MD Physiology, DNB Paediatrics), Assistant Professor, Department of Physiology, World College of Medical Sciences & Research and Hospital, Jhajjar, Haryana, India, Pin-124103 Neonatal jaundice is a yellowish discolouration of mucous membranes and skin in a neonate (infant under 28 days of age), due to high bilirubin levels.(21) Other symptoms may include excess sleepiness or poor feeding. Complications may include seizures, cerebral palsy, or kernicterus.(21) Bilirubin was discovered by Rudolf Virchow in 1847.(17) The serum bilirubin level required to cause jaundice varies with skin tone and body region, but jaundice usually becomes visible on the sclera at a level of 2 to 3 mg/dL and on the face at about 4 to 5 mg/dL. With increasing bilirubin levels, jaundice seems to advance in a head-to-foot direction, appearing at the umbilicus at about 15 mg/dL and at the feet at about 20 mg/dL.(21) Under normal circumstance, the level of indirect bilirubin in umbilical cord serum is 1-3mg/dL and rises at a rate of

<5mg/dL/24hr; thus, jaundice becomes visible on the 2nd or 3rd day, usually peaking between the 2nd and 4th days at 5-6mg/dL and decreasing to less than 2mg/dL between the 5th and 7th days after birth. Jaundice associated with these changes is designated *physiologic jaundice* (non-pathologic unconjugated hyperbilirubinemia) and is believed to be the result of increased bilirubin production from breakdown of fetal red blood cell breakdown combined with transient limitation in conjugation of bilirubin by the immature neonatal liver. In premature infants, the rise in serum bilirubin tends to be the same or somewhat slower but of longer duration than in term infants. Peak levels of 8-12mg/dL are not usually reached until the 4th-7th day, and jaundice is infrequently observed after the 10th day, corresponding to the maturation of mechanisms for bilirubin metabolism and excretion.(13)(15)(21) In general, factors suggesting a pathologic jaundice a search to determine the cause of jaundice should be made if it appears in the 1st 24 hours after birth, serum bilirubin is rising at a rate faster than 5mg/dL/24hr, total serum bilirubin is >12mg/dL in a term infant (especially in the absence of risk factors) or 10-14mg/dL in a preterm infant, jaundice persists after 10-14 days after birth, or serum direct bilirubin fraction is >2mg/dL at any time. Other factors suggesting a *pathologic jaundice* (unconjugated or conjugated hyperbilirubinemia) are family history of hemolytic disease, pallor, hepatomegaly, splenomegaly, vomiting, lethargy, poor feeding, excessive weight loss, apnea, bradycardia, hypothermia, light coloured stools, dark urine positive for bilirubin, bleeding disorder, failure of phototherapy to lower the bilirubin level and signs of kernicterus.(15)(21) Persistent pathologic jaundice- that is, jaundice persisting beyond the first 14 days- is also seen in neonates, more commonly in breastfed babies.(13)

In young babies, unconjugated bilirubin (which is not carried by albumin) can penetrate the membrane that lies between the brain and the blood (the blood-brain barrier) because the blood-brain barrier has yet to develop fully, whereas more developed individuals with increased bilirubin in the blood are protected. Unconjugated bilirubin is potentially toxic to neural tissue (brain and spinal cord). The term kernicterus is used to denote the clinical features of acute or chronic bilirubin encephalopathy, as well as the yellow staining in the brain associated with the former. Kernicterus is also known to occur at lower levels of bilirubin in term babies who have risk factors, and in preterm babies.(13)(15) Kernicterus has been rising in recent years due to less time spent outdoors.(15) Jaundice and kernicterus are crucial global health issues, which must be addressed to reduce neonatal and child mortality globally and reach the sustainable development goals. The use of phototherapy was first discovered, accidentally, at Rochford Hospital in Essex, England, when a nurse, Sister Jean Ward, noticed that babies exposed to sunlight had reduced jaundice, and a pathologist, Dr. Perryman, who noticed that a vial of blood left in the sun, had turned green. Drs Cremer, Richards and Dobbs put together these observations,(6) leading to a landmark randomized clinical trial which was published in Pediatrics in 1958; it took another ten years for the practice to become established.(14)(18) There is currently no reliable evidence about whether home-based or hospital-based phototherapy is more effective for full term infants with jaundice.(19)

AIMS AND OBJECTIVES

In this study, the aim was to study the management of neonatal jaundice in a tertiary care hospital in Delhi, India. Our objectives were to facilitate early diagnosis and reduce subsequent complications of neonatal jaundice by appropriate treatment.

MATERIALS AND METHODS

Study Setting and Period of Study: The study was conducted in the Department of Paediatrics, Rockland Hospital at Qutab Institutional Area in Delhi, India during the period of 01 January 2012 to 07 August 2014. Presently, the Rockland Hospital is known as Medeor Hospital.

Study Design: The study was a Cross-sectional Study, conducted at the Department of Paediatrics, Rockland Hospital in Delhi.

Sample Size: For the present study, 661 neonates (645 singleton neonates and 16 twin neonates) were recorded and studied in Paediatrics Department in Rockland Hospital. These neonates were born alive to 653 mothers in Obstetrics and Gynaecology Department in Rockland Hospital, during the period 01.01.2012 till 07.08.2014.

Sampling Design: The study was done as Random Sampling of the neonates noticed to have deep yellow discolouration of whole body that were born in Rockland Hospital, Delhi. In this study, all the venous blood samples of neonates for Total Serum Bilirubin (TSB) and Direct Serum Bilirubin were collected during first 9 days of life, and in one case of persistent pathologic hyperbilirubinemia, the sample was again collected on Day 21 of life. These neonates were further investigated for hemoglobin estimation, hematocrit estimation, sepsis screen, Glucose-6-Phosphate Dehydrogenase (G6PD) activity and serum TSH levels. Besides, venous blood samples were collected for blood groups of about 516 neonates as well as blood groups of their mothers were also done.

Study Variables: Age and gender of neonate, physiologic jaundice or pathologic jaundice, tests of neonate (blood group of neonate and mother, total serum bilirubin level, indirect serum bilirubin level, direct serum bilirubin level, hemoglobin estimation, hematocrit estimation, Glucose-6-Phosphate Dehydrogenase (G6PD) activity and serum TSH level), maturity of neonate (term or preterm), weight of neonate (normal weight, low birth weight or very low birth weight), neonate with other risk factors (ABO or Rh incompatibility, anemia, polycythemia, early onset neonatal sepsis, congenital abnormality, infant of diabetic mother), method of treatment (sunlight or phototherapy or exchange transfusion), morbidity (hydrops fetalis, kernicterus or cerebral palsy) and mortality rate in a group of neonates born in Rockland Hospital, Delhi.

Inclusion Criteria/ Selection Criteria: Participants in the study eligible for inclusion were neonates of either gender, born alive in Rockland Hospital during the period 01.01.2012 till 07.08.2014. The mothers of these neonates were also included as participants after obtaining written consent from them. Neonates were included after obtaining proper informed written consent from their parent/guardian.

Intrauterine deaths were excluded from the study.

There were 7 neonates (3 males and 4 females) born in Rockland Hospital, Delhi and on Day 1 of life, these neonates were referred to the higher center. These 7 neonates were as follows:

- PT (28-29weeks) / Female /SGA/Very LBW
-) PT (29-30weeks) /Male/ SGA/LBW
- PT(31-32weeks)/Male/SGA/Extreme LBW (920grams)
- PT (32 weeks) / Female /SGA/ LBW
-) Term / Male / AGA
-) Term / Female / AGA
- J Term / Female / AGA (born by VAVD)

The further outcome of these 7 neonates is not known. These were born to 2 primigravida mothers and 5 multigravida mothers by emergency caesarean delivery in 6 cases and ventouse assisted vaginal delivery (VAVD) in one case. These 7 neonates were excluded from the study.

Study Characteristics: In this study, 661 neonates born alive in Rockland Hospital during the period 01.01.2012 till 07.08.2014, were recorded and studied. The demographic information, history, physical examination, investigations and management in the patient's questionnaire were recorded. Neonates that satisfied the inclusion criteria were selected and the neonates who did not meet the inclusion criteria were excluded.

Data Collection Methods and Tools: Neonates' history and investigations information was collected in questionnaires and the data was collected and reported, and then statistical analysis of data was performed using SPSS software. The calculations of odd's ratios were done using MedCalc statistical Software and calculations of P values were done using QuickCalcs-Graphpad Software.

Statistical Methods and Statistical Interpretation: The Chisquare test or Fisher's exact test was used to calculate the Two-tailed P values in our study. When presenting P values, it was helpful to use the asterisk rating system as well as quoting the P value:

 $\begin{array}{l} P < 0.05^{*} & , \mbox{it is statistically significant,} \\ P < 0.01^{**} & , \mbox{it is very statistically significant,} \\ P < 0.001^{***} & , \mbox{it is extremely statistically significant.} \end{array}$

RESULTS AND OBSERVATIONS

For the present cross-sectional study, 661 neonates (645 singleton neonates and 16 twin neonates) were recorded and studied in the Department of Paediatrics, Rockland Hospital at Qutab Institutional Area in Delhi, India during the period of 01 January 2012 to 07 August 2014. Presently, the Rockland Hospital is known as Medeor Hospital. These neonates were born alive to 653 mothers (42.88% primigravida and 57.12% multigravida) by caesarean delivery in 70.75% cases and by vaginal delivery in 29.25% cases, in Obstetrics and Gynaecology Department in Rockland Hospital, during the period 01.01.2012 till 07.08.2014.(4) Hence, more than 70% neonates were observed in hospital for more than 72 hours before discharge, and the remaining healthy neonates with no risk factors were discharged after 24 hours of observation in Rockland Hospital, Delhi. In our study, all the 661 neonates after discharge were followed up for progress of jaundice in

Paediatrics Out-Patient Department at Rockland Hospital, Delhi.

The various 645 singleton neonates (356 males and 289 females) were as follows:

Term, LGA and Macro	oso	mia-9 males and 3 females
Term, AGA and NW		- 299 males and 248 females
Term, SGA and LBW		- 12 males and 19 females
PT, AGA and NW	-	16 males and 07 females
PT, AGA and LBW	-	14 males and 10 females
PT, SGA and LBW	-	06 males and 01 female
PT, SGA and VLBW	-	00 male and 01 female

The various 16 twins (9 males and 7 females) were as follows:

Term, AGA and NW	-	1 male and 2 females
Term, SGA and LBW	-	4 males and 4 females
PT, AGA and NW	-	2 males only
PT, AGA and LBW	-	1 male and 1 female
PT, SGA and LBW	-	1 male only

Table 1. Table showing that the venous blood samples of 86 neonates with pathologic hyperbilirubinemia for serum bilirubin were collected during first 9 days of life, and in one case of persistent pathologic jaundice, the sample was again collected on Day 21 of life.

Age of	Neonates with	% Neonates with
baby	pathologic jaundice	pathologic jaundice
Day 1	01	0.15%
Day 2	05	0.76%
Day 3	20	3.03%
Day 4	12	1.81%
Day 5	17	2.57%
Day 6	15	2.27%
Day 7	09	1.36%
Day 8	05	0.76%
Day 9	02	0.30%
Day 21	01 Repeat neonate	
Total	86 neonates	13.01%

Total Serum Bilirubin was calculated by adding Serum Indirect Bilirubin and Serum Direct Bilirubin. Normal values of Bilirubin are as follows:

Total Serum Bilirubin = 0.3 to 1.0 mg/dL Serum Direct Bilirubin = 0.1 to 0.3 mg/dL Serum Indirect Bilirubin = 0.2 to 0.7 mg/dL

 Table 2. Table showing a comparison of prevalence of jaundice in a group of 661 neonates born in Rockland Hospital, Delhi

Age at which neonates	Physiologic	Pathologic	Total
were diagnosed	Jaundice	jaundice	
1-7 days	575	79	654
8-28days	000	07	007
Total	575	86	661

Hyperbilirubinemia is a higher-than-normal level of bilirubin in the blood. In our study, the results showed that the Total Serum Bilirubin was more than 2 mg/dL and the Serum Direct Bilirubin level was less than 2 mg/dL, in all the 661 neonates. Thus, in our study of 661 neonates, the results showed that almost all neonates had transient unconjugated

hyperbilirubinemia in the neonatal period. In our study, about 575 neonates had physiologic jaundice (non-pathologic

unconjugated hyperbilirubinemia) and about 86 neonates had pathologic indirect hyperbilirubinemia. In this study in Table 2, it is evident that almost all neonates with physiologic jaundice and about 90.70% (78) neonates with pathologic jaundice were diagnosed from Day 2 till Day 7 of life. Besides, one neonate was diagnosed with pathologic jaundice on Day 1 of life. In the Table 2, the two-tailed P value was less than 0.0001***, in the Fisher's exact test. By conventional criteria, the association between rows and columns was considered to be extremely statistically significant. Hence neonates should be routinely followed for progress of jaundice in the first week of life.

The various TSB levels of the 86 neonates with pathologic indirect hyperbilirubinemia were as follows:

12 mg/dL-12.99mg/dL- 10 neonates were as follows: Term/Male/AGA/NNH/EONS Term/Male/AGA/NNH/EONS/Inc Term/Male/AGA/NNH/EONS/Severe anemia /Inc Term/Female/LBW/SGA/NNH/EONS/Polycythemia Term/Male/AGA/NNH/Inc/(TSH on day2-11.19mIU/L) PT/Male/LBW/NNH PT/Twin/Male/LBW/SGA/NNH/EONS PT/Male/LBW/NNH/EONS PT/Female/LBW/NNH/EONS

13 mg/dL-13.99mg/dL- 8 neonates were as follows: Term/Female/AGA /NNH/EONS -3 neonates Term/Male/AGA/NNH/EONS/Inc Term/Female/AGA /NNH PT/Male/LBW/NNH/EONS PT/Female/VLBW/SGA/NNH/EONS/Polycythemia/ (TSH on day5-13.4 mIU/L)

14 mg/dL-14.99mg/dL- 8 neonates were as follows: Term/Male/AGA/NNH/EONS Term/Male/AGA/NNH/EONS Term/Female/AGA/NNH Term/Female/AGA/NNH/Inc Term/Female/AGA/NNH/EONS/Inc PT/Male/NW/AGA/NNH/EONS/Inc PT/Male/NW/NNH/EONS

15 mg/dL-15.99mg/dL- 13 neonates were as follows: Term/Male/AGA /NNH/EONS/IDM Term/Male/AGA /NNH/EONS/IDM Term/Male/AGA /NNH Term/Male/AGA /NNH Term/Female/AGA /NNH Term/Female/AGA/NNH/EONS Term/Female/AGA/NNH/EONS Term/Female/AGA/NNH PT/Male/NW/NNH/EONS PT/Male/LBW/NNH/EONS/VSD/Inc PT/Female/NW/NNH/EONS/Mild anemia/ (TSHday9-10.5 mIU/L) PT/Female/LBW/NNH/EONS

16 mg/dL-16.99mg/dL- 15 neonates were as follows: Term/Male/AGA/NNH/EONS -4 neonates Term/Male/AGA/NNH/EONS/Inc Term/Male/AGA/NNH/IDM/EONS/Inc/MMC (Sacral Meningomyelocele-size about 45 mm X 38 mm) Term/Male/AGA/NNH/IDM/EONS/Inc Term/Male/AGA/NNH/IDM Term/Male/AGA/NNH/Inc Term/Male/AGA/NNH Term/Female/AGA/NNH Term/Female/AGA/NNH/EONS/IDM Term/Female/LBW/SGA/NNH/EONS PT/Male/LBW/NNH/EONS/VSD/(TSHday9-10.5 mIU/L)/Inc PT/Female/LBW/SGA/NNH/EONS

17 mg/dL-17.99mg/dL- 10 neonates were as follows: Term/Male/AGA/NNH/EONS -2 neonates Term/Male/LGA/Macrosomia/NNH/IDM/Inc Term/Male/AGA/NNH Term/Female/AGA /NNH/IDM/Inc Term/Female/AGA /NNH/EONS Term/Female/AGA /NNH/EONS/Inc PT/Male/NW /NNH/EONS/Inc PT/Male/LBW /SGA/NNH/EONS/Polycythemia PT/Male/LBW/NNH/EONS/Left Congenital Talipes Equinovarus (CTEV) /Inc

18 mg/dL-18.99mg/dL-12 neonates were as follows:Term/Male/AGA/NNH/EONS/Inc-2 neonatesTerm/Female/AGA/NNH/EONS/Inc-2 neonatesTerm/Female/AGA/NNH/EONS-2 neonatesTerm/Male/AGA/NNH/EONS/(TSH on day 3-15.1 mIU/L)Term/Male/AGA/NNH/EONSTerm/Male/AGA/NNH/EONS/IDMTerm/Female/AGA/NNH/EONS/Inc/Mild anemiaTerm/Female/LBW/SGA/NNH/EONSPT/Male/NW/NNH/EONS

19 mg/dL-19.99mg/dL- 2 neonates were as follows: Term/Male/AGA/NNH/EONS PT/Female/NW/AGA/NNH/EONS/Polycythemia

20 mg/dL-20.99mg/dL- 1 neonate was as follows: Term/Female/AGA/NNH/EONS/Inc

21 mg/dL-21.99mg/dL- 1 neonate was as follows: Term/Female/AGA/NNH/EONS

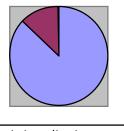
22 mg/dL-22.99mg/dL- 1 neonate was as follows: Term/Female/AGA /NNH/EONS/Inc

23 mg/dL-23.99mg/dL- 2 neonates were as follows: Term/Male/AGA/NNH/EONS/IDM/Inc PT/Male/LBW/NNH/EONS/Inc/(TSH on day6-11.6mIU/L)

24 mg/dL-24.99mg/dL-2 neonates were as follows: PT/Male/NW/NNH/EONS/IDM/Inc PT/Male/NW/AGA/NNH/EONS/Polycythemia (Day5-TSB-24.2 mg/dL, discharged after 3 days of phototherapy) and later again admitted for Persistent NNH (Day21-TSB-24.66 mg/dL, discharged after 3 days of phototherapy)

25 mg/dL-25.99mg/dL - 1 neonate was as follows: Term/Female/AGA/NNH/EONS/Inc In the present study, it is evident that the physiologic jaundice was seen in 86.99% (575) neonates {47.35% (313) males and

pathologic 39.64% (262)females} and indirect hyperbilirubinemia was seen in 13.01% (86) neonates {7.87% (52) males and 5.14% (34) females}. The two-tailed P value was less than 0.0001***, in the Chi-square test. By conventional criteria, this difference was considered to be extremely statistically significant. Among these 13.01% (86) neonates with pathologic indirect hyperbilirubinemia, about 9.68% (64) neonates had TSB between 12mg/dL to 17.99mg/dL and 3.33% (22) neonates had TSB between 18mg/dL to 26mg/dL. All the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis. In the present study, a late preterm neonate was again admitted on Day 21 of life, for persistent pathologic indirect hyperbilirubinemia.



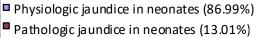


Figure 1. Pie diagram showing prevalence of jaundice in a group of 661 neonates born in Rockland Hospital, Delhi

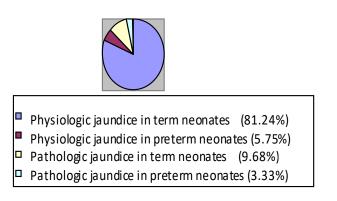


Figure 2. Pie diagram showing a comparison of prevalence of jaundice in term and preterm neonates in a group of 661 neonates born in Rockland Hospital, Delhi.

In the present study, it is evident that the physiologic jaundice was seen in 81.24% (537) term neonates (284 singleton males, 05 twin males, 6 twin females and 242 singleton females) and 5.75% (38) preterm neonates (21 singleton males, 13 singleton females, 1 twin female and 03 twin males). In the present study, it is also evident that the pathologic indirect hyperbilirubinemia was seen in 9.68% (64) term neonates (36 singleton males and 28 singleton females) and 3.33% (22) preterm neonates (15 singleton males, 6 singleton females and 01 twin male).

In the Table 3, the odd's ratio was 4.8577. The two-tailed P value was less than 0.0001***, in the Fisher's exact test. The association between rows (term and preterm neonates) and columns (neonates with physiologic jaundice and neonates with pathologic indirect hyperbilirubinemia) was considered to be extremely statistically significant. Thus, in our study, preterm gestation showed a strong association with pathologic indirect hyperbilirubinemia.

Table 3: Table showing a comparison of prevalence of jaundice in
term and preterm neonates in a group of 661 neonates born in
Rockland Hospital, Delhi

Neonates	Physiologic	Pathologic Indirect	Total
	Jaundice	Hyperbilirubinemia	
Term	537 (81.24%)	064 (09.68%)	601
Preterm	038 (05.75%)	022 (03.33%)	060
Total	575 (86.99%)	086 (13.01%)	661

Table 4. Table showing a comparison of prevalence of jaundice in neonates with weight 2.5kg and neonates with weight less than 2.5kg in a group of 661 neonates born in Rockland Hospital, Delhi.

Neonates	Physiologic Jaundice	Pathologic Indirect Hyperbilirubinemia	Total
Weight 2.5kg	517	070	587
Weight <2.5kg	058	016	074
Total	575	086	661

In the Table 4, the odd's ratio was 2.0374, the two-tailed P value equals 0.0268* in the Fisher's exact test. The association between rows (neonates with weight ≥ 2.5 kg and neonates with weight less than 2.5kg) and columns (neonates with physiologic jaundice and neonates with pathologic indirect hyperbilirubinemia) was considered to be statistically significant. Thus, in our study, low birth weight (LBW) and very low birth weight (VLBW) neonates showed a strong association with pathologic indirect hyperbilirubinemia.

Management of Neonatal Jaundice was done as follows:

- Physiological **j**aundice in newborns was transient and dissipated without medical intervention. Sunbathing was advised in all cases of physiologic jaundice.
- In pathologic jaundice cases when serum bilirubin levels were greater than 12 mg/dL, infant was treated with single surface or double surface phototherapy, depending on the infant's age and prematurity status. No neonate required exchange transfusion. No neonate needed liver transplantation. Bilirubin count is also lowered through excretion — bowel movements and urination —so frequent and effective feedings were vital measures to decrease jaundice in infants.

Management of Risk factors associated with Neonatal Jaundice is as follows:

Table 5. Table showing a comparison of prevalence of jaundice in neonates with early onset sepsis and neonates with no sepsis in a group of 661 neonates born in Rockland Hospital, Delhi.

Neonates	Physiologic	Pathologic Indirect	Total
	Jaundice	Hyperbilirubinemia	
No sepsis	447	017	464
Early onset	128	069	197
neonatal sepsis			
Total	575	086	661

In our study, the neonates with perinatal asphyxia, prolonged rupture of membranes (PROM), meconium stained amniotic fluid or neonates with sepsis screen positive (increased total leucocyte count, culture of blood or other body fluids positive for pathogenic bacteria growth, C Reactive protein (CRP) positive, etc.) were treated as early onset neonatal sepsis. In the Table 5, the odd's ratio was 14.1742 and the two-tailed P value was less than 0.0001*** in the Fisher's exact test. The

association between rows (neonates with no sepsis and neonates with early onset sepsis) and columns (neonates with physiologic jaundice and neonates with pathologic indirect hyperbilirubinemia) was considered to be extremely statistically significant. All the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis, in our study. Thus, in our study, early onset neonatal sepsis showed a strong association with pathologic indirect hyperbilirubinemia. All the neonates with sepsis were treated by appropriate antibiotics, in our study.

Neonatal polycythemia is defined as a central hemoglobin or hematocrit exceeding 2 standard deviations (SD) above the normal value for gestational and postnatal age. A full-term infant was considered to have polycythemia when the hemoglobin concentration was $\geq 22g/dL$ or haemocrit was $\geq 65\%$. (16)

In the Table 6, the odd's ratio was 7.0370. The two-tailed P value equals 0.0049**, in the Chi-square test. The association between rows (neonates with polycythemia and neonates with no polycythemia) and columns (neonates with physiologic with jaundice and neonates pathologic indirect hyperbilirubinemia) was considered to be very statistically significant. All the neonates with polycythemia were also associated with early onset neonatal sepsis, in our study. Thus, in our study, neonates with polycythemia showed a strong association with pathologic indirect hyperbilirubinemia. None of the neonates with polycythemia was treated by partial exchange in our study.

Table 6. Table showing a comparison of prevalence of jaundice in neonates with polycythemia and neonates with no polycythemia in Rockland Hospital, Delhi.

Neonates	Physiologi c Jaundice	Pathologic indirect hyperbilirubinemia	Total
No polycythemia	570	81	651
Polycythemia present	005	05	010
Total	575	86	661

 Table 7: Table showing a comparison of prevalence of jaundice in neonates born to mothers with no diabetes and neonates born to mothers with diabetes in Rockland Hospital, Delhi.

Mothers	Neonates with	Neonates with	Total
	Physiologic	Pathologic Indirect	
	Jaundice	Hyperbilirubinemia	
Mothers with	546	074	620
no diabetes			
Mothers with	029	012	041
diabetes			
Total	575	086	661

In the Table 7, the odd's ratio was 3.0531 and the two-tailed P value equals 0.0036** in the Fisher's exact test. The association between rows (mothers with no diabetes and mothers with diabetes) and columns (neonates with physiologic jaundice and neonates with pathologic indirect hyperbilirubinemia) was considered to be very statistically significant. About 8 neonates of mothers with diabetes were also associated with early onset neonatal sepsis, in our study. Thus, in our study, neonates of mothers with diabetes showed a strong association with pathologic indirect hyperbilirubinemia. All the neonates of diabetic mothers were closely monitored for hypoglycemia, in our study.

Table 8. Table showing a comparison of prevalence of jaundice in
neonates with serum TSH 10mIU/L and neonates with serum
TSH less than 10mIU/L in Rockland Hospital, Delhi.

Neonates with Serum	Physiologic	Pathologic	Total
TSH value after Day 1	Jaundice	jaundice	
Serum TSH<10mIU/L	196	79	275
Serum TSH 10mIU/L	084	07	091
Total	280	86	366

In our study, at birth, there was an acute increase of TSH with peak serum concentrations reaching 70-160mIU/L in few term and preterm infants. A rapid decline to less than 10mIU/L was seen in most neonates over the next 7 days, in our study. There was no case of congenital hypothyroidism, in our study. About 6 neonates with Serum TSH \geq 10mIU/L were also associated with early onset neonatal sepsis, in our study.

The serum TSH levels were less than 16mIU/mL in all the neonates with pathologic jaundice in our study. In the Table 8, the odd's ratio was 0.2068 and the two-tailed P value was less than 0.0001***, in the Fisher's exact test. The association between rows (neonates with serum TSH<10mIU/L and neonates with serum TSH \geq 10mIU/L) and columns (neonates with physiologic jaundice and neonates with pathologic jaundice) was considered to be extremely statistically significant. Thus, in our study, neonates with Serum TSH \geq 10mIU/L showed a strong association with pathologic indirect hyperbilirubinemia. None of the neonates with Serum TSH \geq 10mIU/L was given thyroxine therapy.

Table 9. Table showing a comparison of prevalence of jaundice in neonates with congenital abnormality and neonates with no congenital abnormality in a group of 661 neonates born in Rockland Hospital, Delhi.

Neonates	Physiologic Jaundice	Pathologic jaundice	Total
No congenital abnormality	570	82	652
Congenital abnormality present	005	04	009
Total	575	86	661

In the Table 9, the odd's ratio was 5.5609 and the two-tailed P value equals 0.0201* in the Fisher's exact test. The association between rows (neonates with congenital abnormality) and columns (neonates with physiologic jaundice and neonates with pathologic jaundice) was considered to be statistically significant. All the neonates with congenital abnormality were also associated with early onset neonatal sepsis, in our study. All the neonates with sepsis were treated by appropriate antibiotics, in our study.

Table 10. Table showing a comparison of prevalence of jaundice in neonates with compatible blood groups and neonates with incompatible blood groups in a group of 516 neonates born in Rockland Hospital, Delhi.

Neonates	Physiologic Jaundice	Pathologic Indirect Hyperbilirubinemia	Total
Compatible blood groups	284	056	340
ABO/Rh incompatibility	146	030	176
Total	430	086	516

In the Table 10, the odd's ratio was 1.0421 and the two-tailed P value equals 0.9011 in the Fisher's exact test. The association between rows (neonates with compatible blood groups and neonates with incompatible blood groups) and columns (neonates with physiologic jaundice and neonates with pathologic indirect hyperbilirubinemia) was considered to be not statistically significant.(5) There were about 145 neonates with physiologic jaundice, whose blood groups were not done, so these 145 neonates were excluded from this study. This study shows that the association of pathologic jaundice with neonates with ABO or Rh incompatibility was considered to be not statistically significant, in our study. The risk of initial sensitization of Rh-negative mothers was reduced to zero by the routine administration of Rh-immunoglobulin (RhoGAM) to all mothers at risk for Rh alloimmunization, since there was no case of pathologic jaundice with Rh incompatibility, in our study.

Table 11. Table showing a comparison of prevalence of jaundice in neonates with anemia and neonates with no anemia in Rockland Hospital, Delhi

Neonates	Physiologic Jaundice	Pathologic jaundice	Total
Anemia	015	03	018
No anemia	560	83	643
Total	575	86	661

In the Table 11, the odd's ratio was 1.3494 and the two-tailed P value equals 0.7184 in the Fisher's exact test. The association between rows (neonates with anemia and neonates with no anemia) and columns (neonates with physiologic jaundice) was considered to be not statistically significant, in this study. This shows that the association of pathologic jaundice with neonates with anemia was considered to be not statistically significant, in our study. The neonates with mild anemia were given oral iron therapy and there was one case of neonate with severe anemia who was given packed RBC transfusion, in our study.

Glucose-6-Phosphate Dehydrogenase (G6PD) activity: In the present study, the Glucose-6-Phosphate Dehydrogenase (G6PD) activity was normal in all the 86 neonates of Pathologic Indirect Hyperbilirubinemia and 214 neonates with physiologic jaundice. Remaining neonates with physiologic jaundice were not tested for Glucose-6-Phosphate Dehydrogenase (G6PD) activity. In our study, no neonate had a family history of G6PD deficiency. The two-tailed P value was less than 0.0001***, in the Chi-square test. By conventional criteria, this difference was considered to be extremely statistically significant.

Morbidity and mortality rate: In the present study, no neonate discharged from Rockland Hospital was reported to have hydrops foetalis, kernicterus or cerebral palsy during admission or on follow up. The two-tailed P value was less than 0.0001***, in the Chi-square test. By conventional criteria, this difference was considered to be extremely statistically significant. Besides, the neonatal mortality rate (NMR) was zero during the period 01.01.2012 till 07.08.2014, in the Paediatrics Department in Rockland Hospital, Delhi, India. The two-tailed P value was less than 0.0001***, in the Chi-square test. By conventional criteria, this difference was considered to be extremely statistically significant.

DISCUSSION

For the present cross-sectional study, 661 neonates (645 singleton neonates and 16 twin neonates) were recorded and studied in the Department of Paediatrics, Rockland Hospital at Qutab Institutional Area in Delhi, India during the period of 01 January 2012 to 07 August 2014. Presently, the Rockland Hospital is known as Medeor Hospital. These neonates were born alive to 653 mothers (42.88% primigravida and 57.12% multigravida) by caesarean delivery in 70.75% cases and by vaginal delivery in 29.25% cases, in Obstetrics and Gynaecology Department in Rockland Hospital, during the period 01.01.2012 till 07.08.2014.(4) In our cross-sectional study of 661 neonates, the results showed that almost all neonates had transient unconjugated hyperbilirubinemia in the neonatal period, with more than ninety percent affected during their first week of life. The prevalence of physiologic jaundice in neonates was 86.99% (81.24% in term neonates and 5.75% in preterm neonates) and the prevalence of pathologic indirect hyperbilirubinemia in neonates was 13.01% (9.68% in term neonates and 3.33% in preterm neonates). Among these 13.01% neonates with pathologic indirect hyperbilirubinemia, about 9.68% neonates had TSB between 12mg/dL to 17.99mg/dL and 3.33% neonates had TSB between 18mg/dL to 26mg/dL. Physiological jaundice in newborns was transient and dissipated without medical intervention. Sunbathing was advised in all cases of physiologic jaundice. Bilirubin count is also lowered through excretion - bowel movements and urination -so frequent and effective feedings were vital measures to decrease jaundice in infants. In pathologic jaundice cases when serum bilirubin levels were greater than 12 mg/dL, infant was treated with single surface or double surface phototherapy, on the infant's age and prematurity status. In our study, all the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis, so they were given antibiotics, phototherapy and other supportive treatment. No neonate with jaundice required exchange transfusion or liver transplantation. In our study, no neonate discharged from Rockland Hospital was reported to have hydrops foetalis, kernicterus or cerebral palsy during admission or on follow up. Besides, the Neonatal Mortality Rate was zero, in our study.

Following references support our observations:

-) Almost all hyperbilirubinemia in the immediate neonatal period is unconjugated. Physiologic hyperbilirubinemia occurs in almost all neonates. Shorter neonatal RBC life span increases bilirubin production; deficient conjugation due to the deficiency of UGT decreases clearance; and low bacterial levels in the intestine combined with increased hydrolysis of conjugated bilirubin increase enterohepatic circulation. Bilirubin levels can rise up to 18 mg/dL by 3 to 4 days of life (7 days in Asian infants) and fall thereafter. Physiologic jaundice generally lasts less than seven days. The condition affects over half of babies in the first week of life. Of babies that are born early about 80% are affected.(21)
-) Transient neonatal jaundice is one of the most common conditions occurring in newborns (children under 28 days of age) with more than eighty percent affected during their first week of life.(17)
- Jaundice is observed during the 1st week after birth in approximately 60% of term infants and 80% of preterm infants.(13)(15)

-) The prevalence of neonatal jaundice in healthy term babies at National District Hospital in Bloemfontein was 55.2%. Although 52% of sampled infants had jaundice on the Bilicheck[®] meter, only 17% appeared clinically jaundiced. The consequence of a missed diagnosis and delayed treatment may cause serious morbidity (kernicterus).(3)
-) The incidence of neonatal hyperbilirubinemia in a retrospective study done in a tertiary care hospital was 13.47%. Preterm gestation showed a strong association with neonatal hyperbilirubinemia.(28)
-) The neonatal morbidity was studied in 7015 neonates born at the All India Institute of Medical Sciences Hospital, New Delhi. Neonatal hyperbilirubinemia occurred in 5.9 per cent, most of whom were premature.(29)
-) Hyperbilirubinemia is a common and, in most cases, benign problem in neonates. Overall, 6-7% of full term infants have indirect bilirubin levels >13mg/dL, and <3% have levels >15mg/dL.(15)
- A blood type incompatibility between the mother and baby is also a reason to track the newborn's jaundice more closely. This exists when a mother has the blood type O (and therefore has antibodies against A and B cells) and her newborn is of blood type A or B. This *may* cause the newborn's red blood cells to break down more quickly due to maternal antibodies that have leaked into the baby's bloodstream. A blood type incompatibility also exists if the mother has Rh (Rhesus) factor negative blood type and the newborn is Rh factor positive. This had been a common cause of severe neonatal jaundice, but is now very uncommon because Rh immune globulin (Rhogham) is given to mothers at risk before delivery.(8)
- Alloimune hemolytic disease from RhD antigen incompatibility is approximately 3 times more common among whites than among blacks, because of differences in Rh allele frequency. Indirect-reacting bilirubin content rises rapidly to high levels in the 1st 6-12 hour of life. The risk of initial sensitization of Rhnegative mothers has been reduced to less than 0.1% by the routine administration of Rh-immunoglobulin (RhoGAM) to all mothers at risk for Rh alloimmunization.(16)
- In about a third of all ABO incompatible pregnancies maternal IgG anti-A or IgG anti-B antibodies pass through the placenta to the fetal circulation leading to a weakly positive direct Coombs test for the neonate's blood. However, ABO HDN is generally mild and short-lived and only occasionally severe because: (1) IgG anti-A (or IgG anti-B) antibodies that enter the fetal circulation from the mother find A (or B) antigens on many different fetal cell types, leaving fewer antibodies available for binding onto fetal red blood cells. (2) Fetal RBC surface A and B antigens are not fully developed during gestation and so there are a smaller number of antigenic sites on fetal RBCs.(2)
- In a study, majority of new-borns with ABO incompatibility, developed hyperbilirubinemia between 3-5 days. It shows, hemolytic disease due to ABO incompatibility, becomes severe in presence of aggravating conditions or with risk factor. Hyperbilirubinemia due to ABO incompatibility resolves naturally in most cases (56%), as there is very

mild hemolysis. In cases, who required treatment, most of them were cured only by phototherapy (43%).(24)

- In a cross-sectional study, about 200 mothers and neonates were examined. Our findings depicted that mother's WBC, Hb, PLT, and gestational age were associated with jaundice (P < 0.05). Furthermore, there were significant relationships between different degrees of bilirubin with TSH, T4 levels and G6PD (P < 0.05). In fact, TSH, T4 levels and G6PD were found to be linked to neonatal hyperbilirubinemia. The risk factors for jaundice in our study population comprise some predisposing factors such as WBC, Hb, PLT, gestational age, TSH, and T4 levels, as well as G6PD. Neonates at risk of jaundice are linked to some maternal and neonatal factors that can provide necessary interventions to reduce the burden of the disease. Therefore, identification of associated factors can facilitate early diagnosis, and reduce subsequent complications.(20)
-) The tragedy of occurrence of kernicterus is compounded by the fact that, if newborn jaundice and neonatal hyperbilirubinemia are detected early, kernicterus is completely preventable. All newborn infants are at risk for newborn jaundice, which when unmonitored or untreated can progress to excessive bilirubin levels.(27)
-) In a retrospective study on 1020 patients admitted at a hospital during one year period 1st January 2012–31st December 2012, in 260 there was a diagnosis of indirect hyperbilirubinemia, associated pathology consisted of urinary tract infection in 15 cases, piodermatitis in 12, otitis media in 7, acute diarrhea in 14 cases and severe dehydration in 9 cases. Only one case complicated with kernicterus.(9)
-) Neurotoxicity is the major consequence of neonatal hyperbilirubinemia. An acute encephalopathy can be followed by a variety of neurologic impairments, including cerebral palsy and sensorimotor deficits; cognition is usually spared. Kernicterus is the most severe form of neurotoxicity. Although it is now rare, kernicterus still occurs and can nearly always be prevented.(21)
-) Common complications of preterm birth are high rates of respiratory distress syndrome, sepsis, periventricular leucomalacia, seizures, intraventricular hemorrhage, cerebral palsy, infections, pathologic jaundice, kernicterus, hypoxic ischemic encephalopathy, and visual and hearing problems. Complications of preterm birth were the leading cause of death in children younger than 5 years of age globally in 2016, accounting for approximately 16% of all deaths, and 35% of deaths among newborn babies. Preterm neonates who survive are at greater risk of a range of short-term and long-term morbidities.(32)
- Preterm birth is the most common cause of death among infants worldwide. Complications from preterm births resulted in 0.81 million deaths in 2015 down from 1.57 million in 1990.(10) The chance of survival at 22 weeks is about 6%, while at 23 weeks it is 26%, 24 weeks 55% and 25 weeks about 72%. The chances of survival without any long-term difficulties are lower.(13) Approximately 0.5% of births are extremely early periviable births, and these account for most of the deaths.(10)

- J Survival rates have greatly improved in recent years for infants of borderline viability; however, these infants remain at risk of developing a wide array of complications, not only in the neonatal unit, but also in the long term. Morbidity is inversely related to gestational age; however, there is no gestational age, whollv including term that is exempt. Neurodevelopmental disabilities and recurrent health problems take a toll in early childhood. Subsequently hidden disabilities such as school difficulties and behavioural problems become apparent and persist into adolescence.(25)
- By pathologic criteria, kernicterus develops in 30% of infants (all gestational ages) with untreated hemolytic disease and bilirubin levels >25-30 mg/dL. The incidence at autopsy in hyperbilirubinemic preterm infants is 2-16%.(15)
- Babies with neonatal jaundice may be treated with colored light called phototherapy, which works by changing trans-bilirubin into the water-soluble cisbilirubin isomer.(14)(30)(33) The phototherapy involved is not ultraviolet light therapy but rather a specific frequency of blue light. The light can be applied with overhead lamps, which means that the baby's eyes need to be covered, or with a device called a biliblanket, which sits under the baby's clothing close to its skin.(14)
- Alternative therapy: Acupuncture, and traditional Chinese medicine do not work and should not be used.(13)
-) The bilirubin levels for initiative of phototherapy vary depends on the age and health status of the newborn. However, any newborn with a total serum bilirubin greater than 21 mg/dL should receive phototherapy.(1)
-) There is currently no reliable evidence about whether home-based or hospital-based phototherapy is more effective for full term infants with jaundice.(30)
- Jaundice in newborns is usually transient and dissipates without medical intervention. In cases when serum bilirubin levels are greater than 4–21 mg/dL (68-360 μ mol/L), infant may be treated with phototherapy or exchanged transfusion depending on the infant's age and prematurity status. A Bili light is often the tool used for early treatment, which often consists of exposing the baby to intensive phototherapy. Sunbathing is effective treatment, and has the advantage of ultra-violet-B, which promotes vitamin D production.(26)
-) Bilirubin count is also lowered through excretion bowel movements and urination —so frequent and effective feedings are vital measures to decrease jaundice in infants.(22)
-) Much like with phototherapy the level at which exchange transfusion should occur depends on the health status and age of the newborn. It should however be used for any newborn with a total serum bilirubin of greater than 428 µmol/l (25 mg/dL).(1)(33)

Following references don't support our observations:

-) Bilirubin in LBW infants is significantly higher in males when compared with females.(31)
-) Globally over 100,000 late-preterm and term babies die each year as a result of jaundice.(23)

-) Neonatal mortality rate of India fell gradually from 85.2 deaths per thousand live births in 1969 to 22.7 deaths per thousand live births in 2018.(12)
-) In the present study, the neonatal mortality rate was zero.(4)

SUMMARY

In this study, the aim was to study the management of neonatal jaundice in a tertiary care hospital in Delhi, India. Our objectives were to facilitate early diagnosis and reduce subsequent complications of neonatal jaundice by appropriate treatment. The study was a cross-sectional study, conducted at the Department of Paediatrics, Rockland Hospital in Delhi. For the present study, 661 neonates (645 singleton neonates and 16 twin neonates) were recorded and studied in Paediatrics Department in Rockland Hospital. These neonates were born alive to 653 mothers in Obstetrics and Gynaecology Department in Rockland Hospital, during the period 01.01.2012 till 07.08.2014. Presently, the Rockland Hospital is known as Medeor Hospital. The study was done as Random Sampling of the neonates noticed to have deep yellow discolouration of whole body that were born in Rockland Hospital, Delhi. In this study, all the venous blood samples of neonates for Total Serum Bilirubin (TSB) and Direct Serum Bilirubin were collected during first 9 days of life, and in one case of persistent pathologic hyperbilirubinemia, the sample was again collected on Day 21 of life. These neonates were further investigated for hemoglobin estimation, hematocrit estimation, sepsis screen, Glucose-6-Phosphate Dehydrogenase (G6PD) activity and serum TSH levels. Besides, venous blood samples were collected for blood groups of about 516 neonates as well as blood groups of their mothers were also done. Participants that satisfied the inclusion criteria were selected and the participants who did not meet the inclusion criteria were excluded. Neonates' history, investigations and management information was collected in patient's questionnaires and the data was collected and reported, and then statistical analysis of data was performed using SPSS software. The calculations of odd's ratios were done using MedCalc statistical Software and calculations of P values were done using QuickCalcs-Graphpad Software. The Chi-square test or Fisher's exact test was used to calculate the Two-tailed P values in our study.

In our cross-sectional study of 661 neonates (601 term neonates and 60 preterm neonates), the results showed that almost all neonates had transient unconjugated hyperbilirubinemia in the neonatal period, with more than ninety percent affected during their first week of life. The prevalence of physiologic jaundice in neonates was 86.99% (81.24% in term neonates and 5.75% in preterm neonates) and the prevalence of pathologic indirect hyperbilirubinemia in neonates was 13.01% (9.68% in term neonates and 3.33% in preterm neonates). Among these 13.01% neonates with pathologic indirect hyperbilirubinemia, about 9.68% neonates had TSB between 12mg/dL to 17.99mg/dL and 3.33% neonates had TSB between 18mg/dL to 26mg/dL. Physiological jaundice in newborns was transient and dissipated without medical intervention. Sunbathing was advised in all cases of physiologic jaundice. Bilirubin count is also lowered through excretion - bowel movements and urination -so frequent and effective feedings were vital measures to decrease jaundice in infants. In pathologic jaundice cases when serum bilirubin levels were greater than

12 mg/dL, infant was treated with single surface or double surface phototherapy, depending on the infant's age and prematurity status. In our study, all the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis, so they were given antibiotics, phototherapy and other supportive treatment. No neonate with jaundice required exchange transfusion or liver transplantation. In our study, no neonate discharged from Rockland Hospital was reported to have hydrops foetalis, kernicterus or cerebral palsy during admission or on follow up. Besides, the Neonatal Mortality Rate was zero, in our study.

CONCLUSIONS

From this cross-sectional study of 661 neonates (601 term neonates and 60 preterm neonates), it is concluded that almost all neonates had transient unconjugated hyperbilirubinemia in the neonatal period, with more than ninety percent affected during their first week of life. The prevalence of physiologic jaundice in neonates was 86.99% and the prevalence of pathologic indirect hyperbilirubinemia in neonates was 13.01%. Among these 13.01% neonates with pathologic indirect hyperbilirubinemia, about 9.68% neonates had TSB between 12mg/dL to 17.99mg/dL and 3.33% neonates had TSB between 18mg/dL to 26mg/dL. Physiological jaundice in newborns was transient and dissipated without medical intervention. Sunbathing was advised in all cases of physiologic jaundice. Bilirubin count is also lowered through excretion — bowel movements and urination —so frequent and effective feedings were vital measures to decrease jaundice in infants. In pathologic jaundice cases when serum bilirubin levels were greater than 12 mg/dL, infant was treated with single surface or double surface phototherapy, depending on the infant's age and prematurity status. In our study, all the neonates with TSB more than 18mg/dL were also associated with early onset neonatal sepsis, so they were given antibiotics, phototherapy and other supportive treatment. No neonate with jaundice required exchange transfusion or liver transplantation. In our study, no neonate discharged from Rockland Hospital was reported to have hydrops foetalis, kernicterus or cerebral palsy during admission or on follow up. Besides, the Neonatal Mortality Rate was zero, in our study. Thus, it is evident in our study that the morbidity and mortality in neonates are completely preventable by appropriate management of neonatal jaundice and associated risk factors.

REFERENCES

- American Academy of Pediatrics Subcommittee on Hyperbilirubinemia July 2004. "Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation". Pediatrics. 114 1 : 297–316. doi:10.1542/peds.114.1.297. PMID 15231951.
- Bethesda DL 2005. "Hemolytic disease of the newborn". Blood Groups and Red Cell Antigens. National Center for Biotechnology Information.
- Brits H, Adendorff J, Huisamen D, et al. 2018. The prevalence of neonatal jaundice and risk factors in healthy term neonates at National District Hospital in Bloemfontein. Afr J Prim Health Care Fam Med. 2018;10 1 :e1-e6. Published 2018 Apr 12. doi:10.4102/ phcfm. v10i1.1582
- 4. Chaudhary Veena. 2020. "Prevalence of sex ratio, preterm birth rate, low birth weight rate, twin birth rate, congenital

abnormalities, caesarean delivery rate, morbidity and mortality rate in neonates born in a tertiary care hospital", International Journal of Current Research, 12, 05, 11373-11380.

- 5. Chaudhary Veena. 2020. "A study of effect of ABO and Rh incompatibility on anemia and jaundice in neonates born in a tertiary care hospital", International Journal of Current Research, 12, 07, 12457-12466.
- Cremer, RJ; Perryman, PW; Richards, DH 24 May 1958. "Influence of light on the hyperbilirubinaemia of infants". Lancet. 1 (7030): 1094–7. doi:10.1016/s0140-6736 58 91849-x. PMID 13550936.
- "Definition of Icterus". MedicineNet.com. 2011. Archived from the original on 7 August 2012. Retrieved 3 February 2013.
- 8. Dennis Clements, October 01, 2013. Newborn Jaundice. The DukeHealth.org archives.
- 9. Dobrin C, Davidescu D, Burca R, et al. 2014. PO-0674 Incidence Of Indirect Hyperbilirubinemia At Newborn, Associated Pathology And The Role Of Phototherapy Archives of Disease in Childhood 2014; 99:A474-A475.
- GBD 2013 Mortality and Causes of Death Collaborators January 2015. "Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013". Lancet. 385 9963 : 117–71. doi:10.1016/S0140-6736 14 61682-2. PMC 4340604. PMID 25530442.
- 11. Icterus | Define Icterus at Dictionary.com Archived 2010-12-31 at the Wayback Machine. Dictionary.reference.com. Retrieved on 2013-12- 47.
- 12. India Neonatal mortality rate, 1960-2018 knoema.com
- "Jaundice in newborn babies under 28 days | Guidance and guidelines". NICE. October 2016. Retrieved 11 December 2017.
- 14. Jones, Clay 9 May 2014. "Separating Fact from Fiction in the Not-So-Normal Newborn Nursery: Newborn Jaundice". Science-Based Medicine.
- 15. Kliegman RM, Goyal NK, Shaughnessy EE, et al. 2020. Jaundice and hyperbilirubinemia in newborn. Nelson Textbook of Paediatrics 21st edition; 2020:953-961.
- Kliegman RM, Omar Niss, Russel E. Ware, et al. 2020. Hemolytic disease of the fetus and newborn. Nelson Textbook of Paediatrics 21st edition; 2020:967-971.
- Lightner, David A 2013. "Early Scientific Investigations". Bilirubin: Jekyll and Hyde Pigment of Life. Progress in the Chemistry of Organic Natural Products. 98. pp. 9–179. doi:10.1007/978-3-7091-1637-1_2. ISBN 978-3-7091-1636-4.
- Lucey, J; Ferriero, M; Hewitt, J June 1968. "Prevention of hyperbilirubinemia of prematurity by phototherapy". Pediatrics. 41 6:1047–54. PMID 5652916.
- Malwade, US; Jardine, LA 10 June 2014. "Home-versus hospital-based phototherapy for the treatment of nonhaemolytic jaundice in infants at more than 37 weeks' gestation". The Cochrane Database of Systematic Reviews 6: CD010212. doi:10.1002/14651858. CD010212.pub2. PMID 24913724.
- Mojtahedi SY, Izadi A, Seirafi G, Khedmat L, Tavakolizadeh R. Risk Factors Associated with Neonatal Jaundice: A Cross-Sectional Study from Iran. Open Access Maced J Med Sci. 2018;6 8 :1387-1393. Published 2018 Aug 11. doi:10.3889/oamjms.2018.319

- "Neonatal Hyperbilirubinemia". Merck Manuals Professional Edition. August 2015. Retrieved 11 December 2017.
- O'Keefe L May 2001. "Increased vigilance needed to prevent kernicterus in newborns". American Academy of Pediatrics. 18 5: 231. Archived from the original on 2007-09-27.
- Olusanya, BO; Teeple, S; Kassebaum, NJ February 2018. "The Contribution of Neonatal Jaundice to Global Child Mortality: Findings From the GBD 2016 Study". Pediatrics. 141 2 : e20171471. doi: 10.1542/ peds.2017-1471. PMID 29305393.
- Patel AS, Desai DA, Patel RA. 2017. Association of ABO and Rh incompatibility with neonatal hyperbilirubinaemia. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2017 Apr; 6 4 :1368-1375
- Saigal S, Doyle LW January 2008. "An overview of mortality and sequelae of preterm birth from infancy to adulthood". Lancet. 371 9608: 261–9. doi:10.1016/S0140-6736 08 60136 -1. PMID 18207020.
- Salih FM December 2001. "Can sunlight replace phototherapy units in the treatment of neonatal jaundice? An in vitro study". Photodermatology, Photoimmunology and Photomedicine. 17 6: 272–7. doi:10.1034/j.1600-0781. 2001. 170605 .x. PMID 11722753.
- Schwoebel Ann, Bhutani Vinod, Johnston Lois. 2004. Kernicterus: A "Never-Event" In Healthy Term and Near-Term Newborns. Newborn and Infant Nursing Reviews NAINR . 2004; 4 4 :201-210.

- Shetty, Anil and Kumar, Binoop. 2014. A study of neonatal hyperbilirubinemia in a tertiary care hospital. International Journal of Medical Science and Public Health. 3. 1. 10.5455/ijmsph.2014.010820141.
- 29. Singh M, Deorari AK, Khajuria RC, Paul VK. 1991. A four year study on neonatal morbidity in a New Delhi hospital. Indian J Med Res. 1991;94:186-192.
- Stokowski LA December 2006. "Fundamentals of phototherapy for neonatal jaundice". Adv Neonatal Care.
 6 6: 303–12. doi:10.1016/j.adnc.2006.08.004. PMID 17208161. S2CID 31233601.
- Tioseco JA, Aly H, Milner J, Patel K, El-Mohandes AA.Does gender affect neonatal hyperbilirubinemia in low-birth-weight infants?.Pediatr Crit Care Med.2005;6 2 :171-174. doi:10.1097/01.PCC.0000154961.37833.79
- 32. Vogel JP, et al. 2018. The global epidemiology of preterm birth, Best Practice and Research Clinical Obstetrics and Gynaecology 2018 , https://doi.org/10.1016/j.bpobgyn.2018.04.00
- Wolkoff, Allan W. 2012 . "Chapter 303: The Hyperbilirubinemias". In Longo, Dan L.; Kasper, Dennis L. eds. . Harrison's principles of internal medicine 18th ed. . New York: McGraw-Hill. ISBN 978-0071748896