ISSN No:-2456-2165

A Comparative Clinical Investigation of Hyper Bilirubinemia in Babies with Low and Normal Birth Weights

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Abstract:-
BACKGROUND AND OBJECTIVES: Neonatal jaundice is generally physiological and benign, but it is dangerous in developing countries because it causes neurological damage and causes death and disease. Since the immune system is weak, jaundice in newborns is more common, more severe, lasts longer and can lead to more mental disorders. Over the past few years, scientists have evaluated many risks. However, so far there has been no agreement on the process for initiating medical imaging and transfer, especially for babies with low birth weight and risk factors. This study aims to determine the prevalence, etiology, risk factors and response to treatment of hyperbilirubinemia and to compare preterm and preterm children.

METHODS: A total of 1792 newborns born between November 2011 and October 2013 at Narayana Medical College Hospital were monitored for the development of hyperbilirubinemia. 180 infants with severe hyperbilirubinemia were admitted to the NICU and treated according to the department's protocol. Associated risk factors are also noted. Among them, 33 low-birth-weight infants and 67 low-birth-weight infants who met the inclusion and exclusion criteria were selected from the sample to determine the degree, causes and effects of hyperbilirubinemia. .Call as you wish. Finally, the data were analyzed for significance using statistical tests and the results were presented using standard deviations, frequency tables, and graphs.

RESULTS: The prevalence of significant hyperbilirubinemia was 10.04% and increased with decreasing birth weight. There was a clear male preference in 58.0% of affected infants. Premature infants, LBW mothers aged 19 to 22 years, and NBW infants aged 21 to 26 years are at higher risk for birth asphyxia, sepsis, and polycythemia. Sepsis (23.0%), ABO incompatibility (20.0%) and Rh incompatibility (6.0%) were the most common causes. The main symptoms observed in newborns were ABO blood incompatibility (14.0%), sepsis (10.0%) and polycythemia (10.0%). 16.4% overall and cephalic hematoma (7.5%). Significant hyperbilirubinemia occurs earlier in term infants (60.0-69.9 hours) than in term infants (73.94 hours). Treatment duration is longer in neonates (57.50 hours versus 49.40 hours). Peak serum bilirubin is higher in low birth weight children (17.89 vs 16.20 mg/dl). Low birth weight babies need more flexibility.

CONCLUSIONS: Physiological jaundice occurs in most infants, but infants with certain risk factors develop exaggerated physiological and pathological jaundice, leading to marked hyperbilirubinemia. In the study, low birth weight, premature birth, advanced maternal age, birth asphyxia, sepsis, exclusive breastfeeding and polycythemia were determined as factors. However, gender, jaundiced relatives needing treatment, mother's diabetes, pregnancy due to high blood pressure, body not being able to give birth, childbirth, meconium-stained fluid, etc. It has been shown that some important factors such as do not cause hyperbilirubinemia. . While mismatch-related hyperbilirubinemia, sepsis and polycythemia cover a large proportion of newborn babies, sepsis and cephalohematoma are the causes of serious birth defects. Hyperbilirubinemia occurs early in the neonatal period, but lasts longer and requires long-term treatment. Low birth weight babies need more flexibility. Further analysis can be performed using prospective studies in infants using hourly bilirubin levels to identify infants at risk of hyperbilirubinemia.

Keywords:- *Hyperbilirubinemia; Jaundice; Birth Weight; Prematurity; Bilirubin; Phototherapy; Exchange Transfusion.*

I. INTRODUCTION

Jaundice is a symptom of high bilirubin in the blood and is evident in the skin and sclera. While the concentration is > 2 mg/dl1 in adults, it is > 7 mg/dl1 in pregnant women. Neonatal jaundice is a body condition that affects all babies and reflects the interaction between the development of changes in bilirubin production and metabolism. In most cases, it resolves completely benignly without any treatment or sequelae by the end of the first week of life. However, in some babies, hyperbilirubinemia may increase the risk of neurotoxicity, especially in the presence of serious conditions such as decreased albumin binding capacity and/or affinity, acidosis, medication changes and preterm birth2. Hemolysis due to Rh incompatibility exacerbates bilirubin neurotoxicity in premature infants. 3 Although neuronal cells are considered the primary target of bilirubin toxicity, circulating cells are also affected. Additionally, the

ISSN No:-2456-2165

ability of unconjugated bilirubin to cause hemolysis can further worsen the condition through a vicious cycle. 4. Hyperbilirubinemia occurs when bilirubin production exceeds the ability to excrete it. Neonatal hyperbilirubinemia usually means greater than 15 mg/dl or severe jaundice requiring treatment. 1,460% of newborns have some degree of jaundice, and 80% of newborns have jaundice. 5 Of these, 4-6% of children develop hyperbilirubinemia. 6 Indian research reveals a similar picture. 7,8,9 Over the years, the incidence has increased to 10-14%, probably due to increased suspicion and testing7. However, many hospitals now use it to discharge mothers and babies early, which increases the risk of hyperbilirubinemia 10,11,12 because physical examination is not a good indicator of blood bilirubin. When severe, hyperbilirubinemia can cause free bilirubin to accumulate, cross the blood-brain barrier, and damage the bone marrow. This rare but devastating condition causes the brain to lose bilirubin, called kernicterus. 13 Neonatal jaundice is usually a physical, self-limiting condition, but the danger is that high levels of bilirubin jaundice can damage the brain in the form of bilirubin encephalopathy. Additionally, 14 Michael Kaplan and Cathy Hammerman 15 found that the kernik outcome was severe. in bilirubinemia it is 2-4%.

II. AIMS AND OBJECTIVES

- Estimate the prevalence of neonatal hyperbilirubinemia in low birth weight and low birth weight infants.
- To investigate the causes and risk factors of neonatal hyperbilirubinemia in low birth weight and low birth weight infants.
- Examine the response of birth weight and normal birth weight to treatments.

III. RESEARCH HYPOTHESIS

- The prevalence of neonatal hyperbilirubinemia is higher in premature babies than in term babies.
- Prices are higher compared to India and other parts of the world.
- Hemolysis Hyperbilirubinemia due to blood group incompatibility is observed in most patients.
- Sepsis is an important aid for newborn babies.
- Hyperbilirubinemia occurs in cases such as premature birth, male gender, high birth weight, advanced maternal age, jaundice in previous siblings, gestational diabetes, pregnancy due to hypertension, premature rupture of membranes, instrumental birth and inadequate breastfeeding.
- Response to standard treatment depends on the cause of hyperbilirubinemia and the development of the baby.

IV. MATERIALS AND METHODS

- **Place of Study:** Department of Pediatrics, Narayana Medical College Hospital.
- **Period of Study:** November2011toOctober2013
- **Study Design:** Prospective cohort study.
- **Source of Data:** Hyperbilirubinemia was observed in 1792 babies born at Narayana Medical College Hospital between November 2011 and October 2013.

Hyperbilirubinemia developed in 180 babies. After applying the exclusion criteria, 100 babies were selected from among the babies. These include 33 low birth weight babies and 67 high birth weight babies. Researchers investigated risk factors for hyperbilirubinemia in infants and tracked their treatment and outcomes.

- **Inclusion Criteria:**
  Babies delivered in Narayana Medical College hospital and developing hyperbilirubinemia, irrespective of the gestational age, over a period of 24 months.
- **Exclusion Criteria:**

✓ Where informed consent of parent/guardian was not obtained.
✓ In whom, necessary investigations could not be done or treatment could not be given due to any reason.
✓ Babies born in outside hospitals and referred to us.

V. METHOD OF COLLECTION OF DATA

A total of 1,792 infants born at Narayana Medical College Hospital between November 2011 and October 2013 were monitored for hyperbilirubinemia. 180 infants with hyperbilirubinemia were admitted to the NICU and treated according to office procedures according to American Academy of Pediatrics guidelines (Tables 1 and 2) and the Cockington nomogram. Risk factors associated with the development of hyperbilirubinemia include birth weight, gestational age, maternal age, pregnancy due to hypertension, weakened immune system, gestational diabetes, mode of delivery, birth asphyxia, meconium contamination, cephalic hematoma, and feeding method.

Among these, 100 babies who met the inclusion criteria, including 33 low-birth-weight babies and 67 normal babies, were randomly selected to determine the level, causes and response to treatment of hyperbilirubinemia.

For all infants receiving phototherapy, monitor serum bilirubin every 24 hours until 24 hours after discontinuation of phototherapy; Follow-up at 6 hours, 24 hours and 48 hours after surgery for transplanted babies. get back to normal. A complete hemogram is performed to determine the cause and severity of hyperbilirubinemia. Serum bilirubin was measured using the diazo method of Pearlman and Lee.

VI. DATA ANALYSIS

Data on the incidence of neonatal hyperbilirubinemia, including birth weight, gestational age, gender, and other parameters, were analyzed. Consider the risk of hyperbilirubinemia based on a variety of factors, including gender, maternal age, jaundice in previous siblings, maternal diabetes and gestational hypertension leading to prematurity, mode of delivery, meconium spotting, birth asphyxia, and breast-feeding. The severity, frequency, need of phototherapy and the change of all etiological factors were analyzed.

ISSN No:-2456-2165

These data on smaller and younger babies were compared to Studentst's data and analyzed for statistical significance. Various etiological analyzes were evaluated using chi-square tests for categorical variables. ANOVA test was also used. The results are presented as standard deviations, frequency tables and graphs.

Variables used for data analysis included number of children requiring chemotherapy and transplantation, age at onset of significant hyperbilirubinemia, peak serum bilirubin, age at onset of phototherapy and change, serum Bilirubin before phototherapy and before change, long durationphototherapy.

VII. DISCUSSION

Hyperbilirubinemia is a common condition in infants, and treatment and response vary depending on the cause, gestational age, and birth weight. There is no consensus on the criteria for starting medical imaging and conversion therapy, especially in low birth weight babies. The aim of this study is to determine the cause, course and treatment response of neonatal hyperbilirubinemia and compare it with birth weight. 15.

In the study, all babies born at Narayana Medical College Hospital between November 2011 were retrospectively examined. and October 2013. Of the 1,792 babies born, 180 had hyperbilirubinemia and 100 were selected, including 33 newborns and 67 neonates. The prevalence of significant hyperbilirubinemia was 10.04%, 15.92% in low birth weight infants, and 8.67% in low birth weight infants.

The prevalence of hyperbilirubinemia increases with birth weight, and up to 30% of affected infants have low birth weight. In this study, the preterm birth rate of infants with severe hyperbilirubinemia was 19.0%, and 58.0% of affected infants were boys.

Research shows that hyperbilirubinemia is more common in newborns under the age of 19. Normal births are born to mothers aged 22 and 21-26. If one sibling had severe hyperbilirubinemia requiring treatment, the proportion of newborns with hyperbilirubinemia was 19% (22.4% in heavy births and 20.0% in low birth weight babies).

According to previous studies, there is no risk of hyperbilirubinemia in babies born to diabetic mothers in this study. The study highlights the need for more research on the treatment and management of hyperbilirubinemia in infants, especially in India 17.

Studies have shown that meconium staining in amniotic fluid is associated with greater hyperbilirubinemia in babies born with meconium. Birth asphyxia is an important risk factor, especially in low birth weight babies, possibly due to maternal comorbidities such as gestational hypertension and intrauterine mass growth restriction. Malnutrition of low birth weight babies causes breastfeeding jaundice in the first week of life, and most (62.7%) of low birth weight babies are fed exclusively with breast milk. This indicates that newborns are at high risk for hyperbilirubinemia due to malnutrition in the first week of life.

The most common causes in all severity groups were sepsis (23.9%), ABO incompatibility (20.0%) and Rh incompatibility (6.0%). ABO blood group incompatibility is the most common cause of low birth weight babies (14.0%), followed by sepsis (10.0%) and polycythemia (10.0%). ABO incompatibility (23.9%), sepsis (16.4%), Rh incompatibility (7.5%) and cephalic hematoma (7.5%) are observed in newborns. The cause is still idiopathic in most patients (44.0%). 18.

ABO incompatibility is seen in 20.0% and is more common in low birth weight patients. Various studies have reported rates ranging from 3% to 32.8%. In Canada, 18.6% of cases are thought to be caused by ABO incompatibility. Approximately three-fifths of children with pathological hyperbilirubinemia are caused by hemolysis, and 25.8% of them have incompatible ABO blood groups. In 32.8% of hyperbilirubinemia cases, high bilirubin levels were associated with maternal blood group O, mainly due to ABO blood incompatibility19.

Rh incompatibility was determined to be a significant risk factor for the development of hyperbilirubinemia in 6.0% of low birth weight babies and its prevalence was 1% to 13.2%. Other studies have found that Rh incompatibility is possible in 1% of patients. Sepsis is considered an important risk factor in 10.0% of newborns and 17.9% of newborns, and the risk of sepsis increases as birth weight increases.

It was determined that sepsis is the third most common cause of hyperbilirubinemia in children, followed by neonatal sepsis with 9.5%. Sepsis is associated with multiple etiologies, including prematurity and G6PD deficiency in 8.1% of infants. Infection should be considered as a possible cause of unconjugated hyperbilirubinemia in newborns during the first week of life, in a prospective study of 5805 infants 20.

Cephalohematoma can be diagnosed as an important risk factor for hyperbilirubinemia in normally born infants. causes 9.0% of cases. High bilirubin concentration was associated with cephalic hematoma (p less than 0.01). Cephalohematoma occurred in 7.97% of infants with hyperbilirubinemia, and Bertini G34 et al found that among 2174 infants living in an urban area during this period, approximately 7.32% of patients with hyperbilirubinemia consisted of cephalohematoma.

As a result, Rh incompatibility, sepsis and cephalic hematoma are important risk factors for the development of hyperbilirubinemia in low birth weight babies. Sepsis is a significant risk factor with higher bilirubin levels and higher mortality. Cephalohematoma is one of the major risk factors for hyperbilirubinemia in newborns, accounting for 9.0% of cases. 21,22

This research focuses on hyperbilirubinemia in children, especially the significant hyperbilirubinemia that occurs in premature infants. Studies have shown that preterm babies will develop hyperbilirubinemia earlier than infants and full-term babies. Posthemolytic hyperbilirubinemia occurs first, followed by Rh incompatibility, G6PD deficiency and ABO incompatibility23.

In the study, it was determined that low birth weight babies started phototherapy earlier (mean = 64.05 hours) than low birth weight babies (74.94 hours). This is in contrast to other studies that found that Rh-incompatible infants started phototherapy first (mean = 64.05 hours), followed by ABO-incompatible infants (63.10 hours), and finally patients with advanced sepsis (82.30 hours). Singhal's PK53 study also showed that in infants with hemolytic etiology, treatment should be started as early as possible based on Rh incompatibility, followed by ABO incompatibility and G6PD deficiency24.

Pre-phototherapy bilirubin level was lower in low birth weight infants (mean = 15.50 mg/dl) compared to low birth weight infants (mean = 16.48 mg/dl) 29. Rh-incompatible infants had the lowest bilirubin level before phototherapy (mean = 7.08 mg/dl), probably due to earliest intervention. The highest bilirubin level before phototherapy was found in patients with ABO-incompatible blood (17.45 mg/dl).

Peak serum bilirubin was higher in term infants (mean = 17.89 mg/dl) compared to term infants (mean = 16.20 mg/dl). This may be due to early detection of hyperbilirubinemia in low birth weight infants and early initiation of phototherapy, which can effectively reduce blood bilirubin. In terms of ABO incompatibility, serum bilirubin was highest in the birth cohort (mean = 18.70 mg/dl), followed by Rh incompatibility (mean = 18.65 mg/dl) 25 , 26 , 27 .

In conclusion, the study highlights the importance of early identification and initiation of phototherapy in neonatal hyperbilirubinemia, particularly in preterm and low birth weight babies[28].

VIII. CONCLUSIONS

Studies have identified low birth weight, prematurity, birth asphyxia, sepsis, exclusive breastfeeding, and polycythemia as important risk factors for hyperbilirubinemia in children. Hyperbilirubinemia usually occurs in babies born between ages 19 and 22 and in children born between ages 21 and 26. Other factors such as gender, jaundiced siblings needing treatment, maternal diabetes, pregnancy due to high blood pressure, impotence, childbirth, and alcohol-contaminated meconium did not appear to cause hyperbilirubinemia. While incompatibility-related hyperbilirubinemia, sepsis and polycythemia cover the majority of newborns, sepsis and cephalohematoma are the causes of newborns. Severe hyperbilirubinemia occurs earlier but lasts longer, and low birth weight infants require prolonged phototherapy. The exchange rate is higher for babies with higher birth weight.

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ISSN No:-2456-2165

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Table 1: Guidelines for Term Neonates

GUIDELINES FOR TERM NEONATES			
Age (hrs)	*Phototherapy at bilirubin level (mg/dl)*	*Exchange transfusion, if intense phototherapy fails or is not available at bilirubin level (mg/dl)*	*Exchange Transfusion even if intensive phototherapy is effective at bilirubin level (mg/dl)*
24-48 hrs	12-15	20	25
48-72 hrs	15-18	25	30
>72 hrs	17-20	25	30

Table 2: Guidelines for LBW Neonates

GUIDELINES FOR LBW NEONATES		
Weight (grams)	*Phototherapy at bilirubin level (mg/dl)*	*Exchange transfusion at bilirubin level (mg/dl)*
<1000	prophylactic	10-12
1000-1499	5-8	13-16
1500-1999	8-12	16-18
2000-2499	11-14	18-20

ISSN No:-2456-2165

Table 3: Prevalance of Hyper Bilirubinemia

Birth weight category	No. of babies	Babies with HBR	% of Babies with HBR
NBW	1453	126	8.67%
LBW	291	34	11.68%
VLBW	43	16	37.20%
ELBW	5	4	80.00%
Total LBW	339	54	15.92%
Total Babies	1792	180	10.04%

PREVALANCE IN TOTAL BABIES DELIVERED IN NARAYANA MEDICAL COLLEGE HOSPITAL

- *RISKFACTORS*

Table 4: Gender

			Sex of the baby		Total	P value
			Male	Female		
Birth weight category	Normal Birth weight	Count	39	28	67	0.245
		%	58.2%	41.8%	100.0%	
	Low birth weight	Count	9	11	20	
		%	45.0%	55.0%	100.0%	
	Very low birth weight	Count	7	3	10	
		%	70.0%	30.0%	100.0%	
	Extremely low birth weight	Count	3	0	3	
		%	100.0%	0.0%	100.0%	
Total		Count	58	42	100	
		%	58.0%	42.0%	100.0%	

Table 4 shows that there is increased predilection of male gender (58%) to get involved. But there is no statistical significance(P>0.05 not significant).

Table 5: Gestationalage

			Gestationalage			Total	Pvalue
			Term	32-37weeks	28-32weeks		
Birth weight Category	NormalBirthweight	Count	67	0	0	67	<0.0001
		%	100.0%	0.0%	0.0%	100.0%	
	Lowbirthweight	Count	12	8	0	20	
		%	60.0%	40.0%	0.0%	100.0%	
	Verylowbirthweight	Count	2	8	0	10	
		%	20.0%	80.0%	0.0%	100.0%	
	Extremelylow birth weight	Count	0	0	3	3	
		%	0.0%	0.0%	100.0%	100.0%	
Total		Count	81	16	3	100	
		%	81.0%	16.0%	3.0%	100.0%	

Table 5 shows that higher number of pretermsare involved in lower weight categories. This is statistically significant(P<0.0001 very high significance). Prematurity as a risk factor among babies with significant hyperbilirubinemia is 19.0%.

ISSN No:-2456-2165

Table 6: Maternalage

BWC		Mother'sageinyears															Total
		18	20	21	22	23	24	25	26	27	28	29	30	31	32	33	
NBW	Count	0	1	5	14	11	10	7	1	2	7	1	5	1	1	1	67
	%	0.0	1.5	7.5	20.9	16.4	14.9	10.4	1.5	3.0	10.4	1.5	7.5	1.5	1.5	1.5	100
LBW	Count	3	5	3	3	2	0	0	1	0	1	1	1	0	0	0	20
	%	15.0	25.0	15.0	15.0	10.0	0.0%	0.0	5.0	0.0	5.0	5.0	5.0	0.0	0.0	0.0	100.0
VLBW	Count	3	5	0	2	0	0	0	0	0	0	0	0	0	0	0	10
	%	30.0	50.0	0.0	20.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0
ELBW	Count	2	0	0	0	1	0	0	0	0	0	0	0	0	0	0	3
	%	66.7	0.0	0.0	0.0	33.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100.0
Total	Count	8	11	8	19	14	10	7	2	2	8	2	6	1	1	1	100
	%	8.0	11.0	8.0	19.0	14.0	10.0	7.0	2.0	2.0	8.0	2.0	6.0	1.0	1.0	1.0	100.0
P=0.004																	

Table 6 shows that hyperbilirubinemia is more common among LBW babies born to mothers of 18-22years and normal birth weight babies born to mothers of 21-25years age. It is statistically significant(P<0.05 – significant).

Table 7: Jaundiced sibling requiring phototherapy

			Jaundiced sibling requiring photo therapy		Total	Pvalue
			Yes	No		
Birth weight category	NBW	Count	15	52	67	0.313
		%	22.4%	77.6%	100.0%	
	LBW	Count	4	16	20	
		%	20.0%	80.0%	100.0%	
	VLBW	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	ELBW	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	19	81	100	
		%	19.0%	81.0%	100.0%	

Table 7 shows that percentage ofJaundiced sibbling requiring phototherapy is more in NBW babies(22.4%) than LBW babies(20.0%). But there is no statistical significance(P>0.05 –not significant)

Table 8: GD Minmother

			GDMinmother		Total	P value
			Present	Absent		
Birth weight category	Normal Birth weight	Count	3	64	67	0.889
		%	4.5%	95.5%	100.0%	
	Low birth weight	Count	1	19	20	
		%	5.0%	95.0%	100.0%	
	Very low birth weight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremely low birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	4	96	100	
		%	4.0%	96.0%	100.0%	

Table8showsthatpercentageofbabieswithhyperbilirubinemiawithGDMinmotherismore in LBW(5.0%) babies than NBW babies(4.5%),but there is no statistical significance(P>0.05–not significant).

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Table 9: PIHinmother

			PI Hinmother		Total	P value
			Present	Absent		
Birth weight category	NormalBirthweight	Count	6	61	67	0.313
		%	9.0%	91.0%	100.0%	
	Lowbirthweight	Count	1	19	20	
		%	5.0%	95.0%	100.0%	
	Verylowbirthweight	Count	2	8	10	
		%	20.0%	80.0%	100.0%	
	Extremelylow birth weight	Count	1	2	3	
		%	33.3%	66.7%	100.0%	
Total		Count	10	90	100	
		%	10.0%	90.0%	100.0%	

Table 9 shows that PIH in mother is more in lower birth weight categories ranging 5.0-33.3%. But there is no statistical significance(P>0.05-not significant).

Table 10: PRO Minmother

			PROMinmother		Total	P value
			Present	Absent		
Birth weight category	NormalBirthweight	Count	4	63	67	0.383
		%	6.0%	94.0%	100.0%	
	Lowbirthweight	Count	3	17	20	
		%	15.0%	85.0%	100.0%	
	Verylowbirthweight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremelylow birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	7	93	100	
		%	7.0%	93.0%	100.0%	

Table 10 shows that PROM in mother is more in LBW babies(15%) than in NBW babies(6%). There is no statistical significance(P>0.05 –not significant).

Table 11: Mode of Delivery

			Mode of delivery		Total	P value
			Normal vaginal delivery	Caesarean section		
Birth weight category	Normal Birth weight	Count	47	20	67	0.159
		%	70.1%	29.9%	100.0%	
	Low birth weight	Count	15	5	20	
		%	75.0%	25.0%	100.0%	
	Very low birth weight	Count	10	0	10	
		%	100.0%	0.0%	100.0%	
	Extremely low birth weight	Count	3	0	3	
		%	100.0%	0.0%	100.0%	
Total		Count	75	25	100	
		%	75.0%	25.0%	100.0%	

Table 11 shows mode of delivery by Caesarean section is more in NBW babies(29.9%) than in LBW babies(25.0%).It has no statistical significance(P>0.05 –not significant).

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Table 12: Meconium stained liquor

			Meconiumstained liqour		Total	P value
			present	Absent		
Birth weight category	Normal Birth weight	Count	7	60	67	0.569
		%	10.4%	89.6%	100.0%	
	Low birth weight	Count	3	17	20	
		%	15.0%	85.0%	100.0%	
	Very low birth weight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremely low birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	10	90	100	
		%	10.0%	90.0%	100.0%	

Table 12 shows Meconium stained liquor is more in LBWbabies(15.0%) than NBW babies(10.4%).It has no statistical significance (P>0.05 – not significant).

Table 13: Birthasphyxia

			Birthasphyxia		Total	P value
			Present	Absent		
Birth weight category	Normal Birth weight	Count	6	61	67	0.023
		%	9.0%	91.0%	100.0%	
	Low birth weight	Count	3	17	20	
		%	15.0%	85.0%	100.0%	
	Very low birth weight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremely low birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	9	91	100	
		%	9.0%	91.0%	100.0%	

Table 13 shows Birth asphyxia is more in LBW(15.0%) babiesthan in NBW babies (9.0%).It has no statistical significance(P<0.05 – significant).

Table 14: Pattern of feeding

BWC		Patternoffeeding						Total	Pvalue
		BF	FF	BF+FF	BF+IVF	FF+IVF	IVF		
NBW	Count	42	0	8	17	0	0	67	<0.001
	%	62.7%	0.0%	11.9%	25.4%	0.0%	0.0%	100.0%	
LBW	Count	8	1	4	5	2	0	20	
	%	40.0%	5.0%	20.0%	25.0%	10.0%	0.0%	100.0%	
VLBW	Count	0	0	1	7	2	0	10	
	%	0.0%	0.0%	10.0%	70.0%	20.0%	0.0%	100.0%	
ELBW	Count	0	0	0	0	0	2	2	
	%	0.0%	0.0%	0.0%	0.0%	0.0%	100.0%	100.0%	
Total	Count	50	1	13	29	4	2	99	
	%	50.5%	1.0%	13.1%	29.3%	4.0%	2.0%	100.0%	

Table 14 shows Breast feeding is high in NBW babies(62.7%) and it is less in LBW babies(40.0%). It implies LBW babies are more prone to develop hyperbilirubinemia due to insufficient feeding in the first week of life.This is statistically significant(P<0.0001 –very highly significant).

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Table 15: Cephalhaematoma

			Cephalhaematoma		Total	P value
			Present	absent		
Birth weight category	NormalBirth weight	Count	6	61	67	0.370
		%	9.0%	91.0%	100.0%	
	Lowbirthweight	Count	0	20	20	
		%	0.0%	100.0%	100.0%	
	Verylowbirth weight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremelylow birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	6	94	100	
		%	6.0%	94.0%	100.0%	

Table 15 shows that Cephalhaematoma is present only in NBW babies (9.0%).But no statistical significance was found(P>0.05 –not significant).

Table 16: Septicaemia

			Sepsis screen or Blood culture		Total	Pvalue
			positive	negative		
Birth weight category	Normal Birth weight	Count	12	55	67	<0.001
		%	17.9%	82.1%	100.0%	
	Low birth weight	Count	2	18	20	
		%	10.0%	90.0%	100.0%	
	Very low birth weight	Count	7	3	10	
		%	70.0%	30.0%	100.0%	
	Extremely low birth weight	Count	3	0	3	
		%	100.0%	0.0%	100.0%	
	Total LBW babies	Count	12	21	33	
		%	36.3%	63.6%	100.0%	
Total		Count	24	76	100	
		%	24.0%	76.0%	100.0%	

Table shows Seticaemia is more in NBW babies(17.9%) than LBW babies(10.0%).But it is very high in VLBW babies(70%) and ELBW babies(100%) which is statistically significant(P<0.0001– very high significance).

Table 17: Polycythemia

			Haematocrit>65%		Total	P value
			yes	no		
Birth weight category	NormalBirthweight	Count	0	67	67	0.043
		%	0.0%	100.0%	100.0%	
	Lowbirthweight	Count	2	18	20	
		%	10.0%	90.0%	100.0%	
	Verylowbirthweight	Count	0	10	10	
		%	0.0%	100.0%	100.0%	
	Extremelylow birth weight	Count	0	3	3	
		%	0.0%	100.0%	100.0%	
Total		Count	2	98	100	
		%	2.0%	98.0%	100.0%	

Table 17 shows Polycythemia is present only in LBW babies(10%).It is statistically significant (P<0.05 – significant).

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Table 18: Etiology of Hyperbilirubinemia

			Etiology ofHyperbilirubinemia						Total	P value
			ABO-I	Rh-I	Sep	Py	CH	Id		
Birth weight category	NBW	Count	16	5	11	0	5	30	67	0.001
		%	23.9%	7.5%	16.4%	0.0%	7.5%	44.8 %	100.0%	
	LBW	Count	3	1	2	2	0	12	20	
		%	15.0%	5.0%	10.0%	10.0%	0.0%	60.0 %	100.0%	
	VLB W	Count	1	0	7	0	0	2	10	
		%	10.0%	0.0%	70.0%	0.0%	0.0%	20.0 %	100.0%	
	ELB W	Count	0	0	3	0	0	0	3	
		%	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	100.0%	
Total		Count	20	6	23	2	5	44	100	
		%	20.0%	6.0%	23.0%	2.0%	5.0%	44.0%	100.0%	

Table18 shows that in NBW babies Idiopathic(44.8%),ABO incompatibility(23.9%)and sepsis(16.4%) are predominant etiology, whereas in LBW babies Idiopathic(60.0%),ABO incompatibility(15.0%), Polycythemia(10.0%) and Sepsis(10.0%) predominate. Sepsis is themajor etiology in VLBW(70.0%) and ELBW(100%)babies. It is statistically significant(P<0.05 significant).

PHOTOTHERAPY

Table 19: Age of significant Hyperbilirubinemia

		N	Mean	Std. Deviation	Min	Max
Age of significant Hyperbilirubinemia in hours	Normal Birth weight	67	73.94	23.871	12	118
	Low birth weight	20	63.05	23.898	7	104
	Very low birth weight	10	69.60	26.311	34	114
	Extremely low birth weight	3	60.00	14.000	44	70
	Total	100	70.91	24.059	7	118
		F=1.293			P=0.281	

Table 19 shows significant hyperbillirubinemia appears early in LBW categories(60.0 to 69.9 hours) whereas it appears late in NBW babies(73.94 hours). But P-value is not significant.

Table 20: Age of Significant Hyperbilirubinemia according to etiology

Etiology of Hyperbilirubinemia	**Mean**	**Std. Deviation**	**N**
ABO incompatibility	62.10	12.113	20
Rhincompatibility	11.83	2.858	6
Sepsis	81.30	24.012	23
Polycythemia	77.00	4.243	2
Cephalhaematoma	74.80	6.419	5
Idiopathic	76.82	18.644	44
Total	70.91	24.059	100
	F=11.775	P<0.0001	

Table 20 shows significant hyperbillirubinemia appears early in Rh incompatibility(11.83hours) and late in Sepsis(81.30hours).P-value is very highly significant.

Table 21: Age at initiation of phototherapy

		N	**Mean**	**Std. Deviation**	**Minimum**	**Maximum**
Ageat Initiation of Phototherapy In hours	Normal Birth weight	67	74.94	23.871	13	119
	Low birth weight	20	64.05	23.898	8	105
	Very low birth weight	10	70.60	26.311	35	115
	Extremely low birth weight	3	61.00	14.000	45	71
	Total	100	71.91	24.059	8	119
		F=1.293			P=0.281	

Table 21 shows that phototherapy was initiated early in LBW babies(mean=64.05hours) than NBW babies(74.94hours). P-value is not significant.

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Table 22: Age at initiation of phototherapy in hours according to etiology

Etiology of Hyperbilirubinemia	Mean	Std. Deviation	N
ABO incompatibility	63.10	12.113	20
Rhin compatibility	12.83	2.858	6
Sepsis	82.30	24.012	23
Polycythemia	78.00	4.243	2
Cephalhaematoma	75.80	6.419	5
Idiopathic	77.82	18.644	44
Total	71.91	24.059	100
	F=11.775	P<0.0001	

Table 22 shows that phototherapy was initiated early in Rh incompatibility and late in Sepsis. P-value is very highly significant.

Table 23: Pre phototherapy Bilirubin

		N	Mean	Minimum	Maximum
Prephototherapy Bilirubin	Normal Birth weight	67	16.487	4.5	20.4
	Low birth weight	20	16.605	4.8	20.4
	Very low birth weight	10	15.450	11.7	20.2
	Extremely low birth weight	3	14.700	11.0	17.9
	Total LBW babies	33	15.50	4.8	20.4
	Total	100	16.373	4.5	20.4
		F=0.655		P=0.582	

Table 23 shows phototherapy was initiated at lower levels of serum bilirubin in LBW categories(14.7 to 16.60mg/dL) when compared to NBW babies(16.48 hours).P-value is not significant.

Table 24: Pre phototherapy bilirubin according to etiology

Etiology of Hyperbilirubinemia	Mean	Std. Deviation	N
ABOincompatibility	17.450	2.5025	20
Rhincompatibility	7.083	4.1513	6
Sepsis	15.735	2.2121	23
Polycythemia	16.150	.2121	2
Cephalhaematoma	17.320	1.4237	5
Idiopathic	17.386	1.3203	44
Total	16.373	3.1719	100
	F=22.408	P<0.0001	

Table 24 shows phototherapy was initiated at lower levels of serum bilirubin in Rhincompatibility followed by Sepsis, Polycythemia and at higher levels in Idiopathic, Cephalhaematoma and ABO incompatibility. P-value is very highly significant.

Table 25: Peakserum Bilirubin

		N	Mean	Minimum	Maximum
Peakserum Bilirubin	Normal Birth weight	67	17.894	14.2	24.3
	Low birth weight	20	17.440	15.3	20.4
	Very low birth weight	10	15.820	11.7	20.2
	Extremely low birth weight	3	15.400	11.0	17.9
	Total LBW babies	33	16.200	11.0	20.4
	Total	100	17.521	11.0	24.3
		F=5.243		P=0.002	

Table25showsPeak serum Bilirubin ishighinNBW babies(17.89mg/dL)thaninTotalLBW babies(16.20 mg/dL).P-value is significant.

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Table 26: Peak serum bilirubin according to etiology

Etiology of Hyperbilirubinemia	Mean	Std. Deviation	N
ABOincompatibility	18.705	1.4877	20
Rhincompatibility	18.650	4.2566	6
Sepsis	16.252	2.1952	23
Polycythemia	16.600	.8485	2
Cephalhaematoma	18.020	.2588	5
Idiopathic	17.477	1.1121	44
Total	17.521	1.9295	100
	F=2.989	P=0.016	

Table 26 shows Peak serum Bilirubin is high in ABO incompatibility, Rh incompatibility followed by cephalhaematoma, idiopathic, polycythemia and Sepsis.P-value is not significant.

Table 27: Duration of phototherapy

		N	Mean	Min	Max
Duration of phototherapy in hours	Normal Birth weight	67	49.40	20	116
	Low birth weight	20	43.35	20	93
	Very low birth weight	10	75.50	23	119
	Extremely low birth weight	3	53.67	44	71
	Total LBW babies	33	57.50	20	119
	Total	100	50.93	20	119
		F=4.616		P=0.005	

Table 27 shows duration of phototherapy in NBW babies(49.40hours) is less than LBW babies(57.50 hours). P-value is significant.

Table 28: Duration of phototherapy in hours according to etiology

Etiology of Hyperbilirubinemia	Mean	Std. Deviation	N
ABOincompatibility	57.60	19.696	20
Rhincompatibility	79.00	14.993	6
Sepsis	63.52	26.407	23
Polycythemia	68.00	.000	2
Cephalhaematoma	54.80	14.342	5
Idiopathic	36.27	18.288	44
Total	50.93	24.291	100
	F=5.661	P<0.0001	

Table 28 shows duration of phototherapy is high for Rh incompatibility, Polycythemia, Sepsis whereas least duration in Idiopathic cases. P-value is very highly significant.

Table 29: Age at Exchange transfusion

		N	Mean	Std. Deviation	Min	Max
Ageat Exchange Transfusion	Normal Birth weight	4	26.50	17.234	7	49
	Low birth weight	1	13.00	.	13	13
	Very low birth weight	0	.	.	.	.
	Extremely low birth weight	0	.	.	.	.
	Total	5	23.80	16.100	7	49
		F=0.491			P=0.534	

Table shows LBW babies require exchange transfusion early at 13.00 hourswhereasin NBW babies at 26.50 hours.P-value is not significant.

Table 30: Pre exchange transfusion serum bilirubin

		N	Mean	Std. Deviation	Min	Max
Pre exchange transfusion serum bilirubin	Normal Birth Weight	4	20.275	4.3828	14.2	24.3
	Low Birth Weight	1	16.200	.	16.2	16.2
	Very Low Birth Weight	0	.	.	.	.
	Extremely low birth weight	0	.	.	.	.
	Total	5	19.460	4.2105	14.2	24.3
		F=0.692			P=0.467	

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Table 30 shows exchange transfusion was done in early hours in LBW babies(16.20hours) when compared to LBW babies(20.27hours). P-value is not significant.

PREVALENCEOFNEONATALHYPERBILIRUBINEMIA

Table 31: Comparision of studies on prevalence of neonatal hyper bilirubinemia

YEAR OF STUDY	RESEARCHER	PREVALANCE	SAMPLESIZE
1991	SINGHM[37]	5.9%	7015
1992	GUARANRL[42]	12.4%	88,137
2001	NARANGA[7]	11.18%	6586
2001	BERTINIG[36]	5.1%	2174
2002	MARTINTC[34]	12.5%	3721
2004	CAMILIAR[1]	6.1%	NA
2004	SARICISU[41]	16.1%	365
2006	SGROM[5]	10.53%	2450
2013	OURSTUDY	10.04%	1792

RISK FACTORS FOR HYPER BILIRUBINEMIA

Table 32: Birth Weight

COMPARISIONOFSTUDIES-SIGNIFICANCEOFBIRTHWEIGHT				
YEAR OF STUDY	RESEARCHER	PREVALANCE IN LBW	PREVALANCEIN NBW	SAMPLE SIZE
1982	LANGEAP[49]	HIGHER	LOWER	739
1984	ARIF.MA[47]	42.5%	NA	414
1985	LINNS[48]	17.38%	10.03%	12,023
1990	GALE.R[35]	16.97%	8.94%	10,122
2013	OURSTUDY	15.92%	8.67%	1792

GESTATIONALAGE

Table 33: Comparision of studies-Significance of gestationalage

Year of study	Researcher	Prematurityascause	Samplesize	Significance
2002	MartinTC[34]	9%	3721	Sig
1992	SinghalPK[53]	16.7%	454	Sig
1991	SinghM[37]	5.9%	7015	Sig
1990	OwaJA[39]	59.5%	292	Vhs
2004	Sarici[41]	25.3%	365	Vhs
1990	GaleR[35]	38.28%	10,122	Hs
1983	PalmerDC[43]	19.9%	41,057	Hs
1994	Arif.K[46]	12.8%	5570	Sig
2013	Ourstudy	19.0%	1792	Vhs

GENDER

Table 34: Comparision of studies-Significance of gender

Year of study	Researcher	Percentage affected		Sample	Significance
		Males	Females		
1994	BahlL[38]	64.5	35.5	164	vhs
1990	GaleR[35]	57.84	42.16	10,122	hs
2001	HintzSR[51]	55.26	44.74	276	hs
2013	Ourstudy	58.0	42.0	100	NS