"SCREENING FOR BILIRUBIN INDUCED NEUROLOGICAL DYSFUNCTION (BIND) AMONG HYPERBILIRUBINEMIC NEONATES, A HOSPITAL BASED PROSPECTIVE STUDY."

BY

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IN

PEDIATRICS

UNDER THE GUIDANCE OF

Dr. S. V. PATIL, MD

PROFESSOR,

DEPARTMENT OF PEDIATRICS

SHRI B.M. PATIL MEDICAL COLLEGE, VIJAYAPUR

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SHRI.B.M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPUR.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled **"SCREENING FOR BILIRUBIN INDUCED NEUROLOGICAL DYSFUNCTION (BIND) AMONG HYPERBILIRUBINEMIC NEONATES, A HOSPITAL BASED PROSPECTIVE STUDY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr S V PATIL, M.D** Professor, Department of Paediatrics, Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapur.

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Dr. Shreyas Shridhar Vaidya

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ABBREVIATIONS

BIND	Bilirubin Induced Neurological Dysfunction
ABE	Acute bilirubin encephalopathy
BE	Bilirubin encephalopathy
NNJ	Neonatal Jaundice
TSB	Total serum bilirubin
UCB	Unconjugated bilirubin
SD	Standard deviation
AABR	Automated auditory brainstem response

ABSTRACT

Background: Hyperbilirubinemia is a common and often benign problem in neonates. Around 60% of term neonates and 80% of preterm neonates develop hyperbilirubinemia during neonatal period. Untreated unconjugated hyperbilirubinemia is potentially neurotoxic which may cause neonatal morbidity and mortality characterised by bilirubin induced neurological dysfunction (BIND), kernicterus and subsequently chorio-athetoid cerebral palsy.

Study justification: The local incidence of BIND is not known and BIND scoring criteria is not been adopted as standard for hyperbilirubinemic neonates' assessment. The study is set to estimate magnitude of the problem as it is easily preventable with simple measures.

Objectives: To determine Incidence of BIND and to assess the correlation of risk factors for BIND in neonates with hyperbilirubinemia.

Methods: A hospital based prospective observational study conducted in Shri B M Patil Medical College Hospital and Research center from December 2019 to June 2020. All neonates with gestational age >35 weeks presenting with hyperbilirubinemia were enrolled. A sample of 173 hyperbilirubinemic neonates were enrolled in the study. Serum Bilirubin level estimation was done if neonate was icteric. Criteria for Significant Hyperbilirubinemia were decided on the basis of American Academy of Pediatrics (AAP) guidelines. In case of significant hyperbilirubinemia BIND scoring was assessed. BIND scoring system was applied to detect changes in mental status, muscle tone, and cry pattern of significant hyperbilirubinemia neonates. In all BIND positive babies AABR was performed.

Results – Out of 173 neonates enrolled into the study, 80(46.2%) were females and 93 (53.8%) were males. The Mean age at admission was 70.3 ± 31 hours. The mean birth weight was 2665.8 grams ± 268.6 grams and mean weight at admission was 2183.1 grams ±259.2. grams. 83 (48%) mothers were primi parous and 90 (52%) mothers were multiparous. 125 (72.3%) neonates were term and 48 (27.7%) neonates were preterm. O positive (55.5%) was most common maternal blood group. A positive was most common baby's blood group.115 (66.5%) babies were delivered normal vaginally and 58 (33.5%) babies were delivered via LSCS. ABO incompatibility (38%) was most common maternal risk factor. Low intake of breast milk (64.7%) was most common neonatal risk factor. History of previous sibling receiving phototherapy was 22 (12.7%). Mean duration of history of jaundice 10.4 hours ± 7.9 hours. Mean total bilirubin was 14.1 mg/dl ± 3.2 mg/dl with maximum being 27mg/dl. Mean unconjugated bilirubin was 13.2 mg/dl ± 3.0 mg/dl with maximum being 26.3 mg/dl. Mean BIND score was 1 ± 0.7. Using BIND scoring criteria, the incidence of BIND was as follows according to severity, 48% had subtle acute bilirubin encephalopathy, acute bilirubin encephalopathy was 0% and chronic bilirubin encephalopathy were 0%. All BIND positive neonates passed AABR.

Conclusion – The incidence of bilirubin induced neurological dysfunction was 48% (Subtle BIND). We suggest the amalgamation of the BIND scoring system in all hyperbilirubinemic neonate's checklist at the time of admission.

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INTRODUCTION

Neonatal hyperbilirubinemia is usually benign problem in neonates and one of the leading causes of hospital readmission after birth. Around 60% of term neonates and 80% of preterm neonates develop jaundice in the 1st week of life, Untreated severe unconjugated hyperbilirubinemia is proved neurotoxic in neonates. Bilirubin induced neurological dysfunction (BIND) refers to clinical signs and symptoms associated with bilirubin neurotoxicity. It can either be acute or chronic. Extreme form of BIND is Kernicterus.[1] BIND refers to individuals with subtle neurodevelopmental disabilities without classical findings of kernicterus that appear to be due to bilirubin neurotoxicity.[2] In a recent review of the global burden of neonatal jaundice, South Asia was also reported to be the leading contributors to an estimated 1.1 million neonates with severe hyperbilirubinemia total bilirubin level (TB) >20 mg/dL worldwide[3]. In few developing countries, the incidence of severe hyperbilirubinemia is very high as it is in the developed world.[4.] In such areas, approximately 3% of neonates admitted to a hospital have signs and symptoms of BIND.[5]

Classic signs and symptoms of acute bilirubin encephalopathy (ABE) in the severely hyperbilirubinemic term neonates have been described by van Praagh, [6] Jones, [7] Volpe, [8] and Perlstein.[9] These include tone abnormalities such as hypotonia, hypertonia, retrocollis and opisthotonos, in association with varying degrees of drowsiness, lethargy, decreased feeding and irritability when described in terms of the neonate's muscle tone, mental status and cry pattern.

Kernicterus leads to devastating disability including athetoid cerebral palsy, speech and hearing impairment. These features represent the severe manifestations of BIND. The progression of acute bilirubin encephalopathy can be documented and provides a

idea for grading its severity.[10, 11, 12, 13,] A higher BIND score would be indicative of worsening signs and symptoms of acute neurotoxicity. The earliest signs and symptoms of ABE are non specific therefore may be easily missed . Moderate signs of ABE have been considered as a definitive signs of kernicterus and include beginning arching of the neck and trunk on stimulation, alternating with increasing lethargy, decreased feeding, unexplained irritability and usually accompanied by a shrill cry.

During the early phases, prompt and effective treatment could prevent chronic kernicterus sequelae. Advanced signs are progressive and marked by cessation of feeding, bicycling movements, inconsolable crying, irritability, inability to feed, fever, seizures and coma. Even with neonatal intensive care these late findings are threatening predictors of the probability of severe kernicterus complications.

OBJECTIVES

Objective of the study:

- To determine the incidence of bilirubin induced neurological dysfunction in hyperbilirubinemic neonates.

- To assess the correlation of Risk Factors for bilirubin induced neurological dysfunction in hyperbilirubinemic neonates.

REVIEW OF LITERATURE

The first description of kernicterus of the brain in neonates with jaundice was provided by Hervieux in 1847.[14,15.] The relation between the clinical encephalopathy associated with elevated total serum bilirubin (TSB) concentration and the gross pathologic changes seen as yellow staining of specific areas of the central nervous system was observed and described as early as 1875[16].

The terms bilirubin encephalopathy and kernicterus represent clinical and pathologic abnormalities respectively resulting from bilirubin toxicity in the central nervous system; to avoid confusion and encourage greater consistency in the literature, the American Academy of Paediatrics recommends that the word acute bilirubin encephalopathy be used to describe the acute manifestations of bilirubin toxicity which is non-permanent usually seen in the 1st week after birth only and the word kernicterus is reserved for chronic and permanent clinical sequel of bilirubin toxicity.[17]

Pathophysiology of bilirubin induced injury.

Bilirubin neurotoxicity is caused by free unconjugated bilirubin anion. This bilirubin anion binds to phospholipids (gangliosides) of neuronal plasma membrane causing injury. Intracellular bilirubin anion binds to the membrane phospholipids of sub cellular organelles causing: impaired energy metabolism, altered excitatory amino acid homeostasis and excitotoxic neuronal injury and cell death.[18].The blood– brain barrier has plays an important role in the protection of the neurons from bilirubin induced neuronal damage; however, its damage produces diffuse yellow staining, not the specific pattern of kernicterus.[19]According to recent study blood–brain barrier,

using ATP-dependent export by transporter molecules, acts as pump to clear free bilirubin from the brain and to maintain the concentration gradient of unconjugated bilirubin from plasma to cerebro spinal fluid (CSF).[20]

Necrosis and apoptosis caused by bilirubin damages brain tissue cells, either alone or in combination, neuro-anatomical distribution of bilirubin in neonates is dependent on the quantity(TSB and USB), duration of exposure to neuro toxic dose (free UCB), and the developmental timing of the exposure of sensitive brain tissue to free bilirubin. Bilirubin may also damage neurons by causing neuronal hyper excitability. Neuronal damage is via excitatory amino-acid neurotoxicity, or it may also have other membrane or neurotransmitter damaging effects. Finally, mitochondrial respiration and energy production are inhibited. Bilirubin has selective deposition in the basal ganglia, especially the globus pallidus and sub thalamic nucleus. Brainstem nuclei, most importantly the auditory (cochlear nucleus, inferior colliculus, superior olivary complex), oculomotor and vestibular nuclei are more vulnerable to bilirubin induced neuro toxicity.

Other most susceptible areas in developing brain are the cerebellum, especially the hippocampus, and the Purkinje cells. Movement disorders of dystonia and athetosis are clinically correlated with the basal ganglia lesions. Bilirubin damages of the auditory brainstem nuclei are associated with deafness, hearing loss, and a recently described entity known as auditory neuropathy (AN), also known as auditory dys-synchrony (AD). In brainstem oculomotor nuclei is affected. Leads to abnormalities which may results in strabismus and gaze palsies, especially paresis of up gaze.[21]

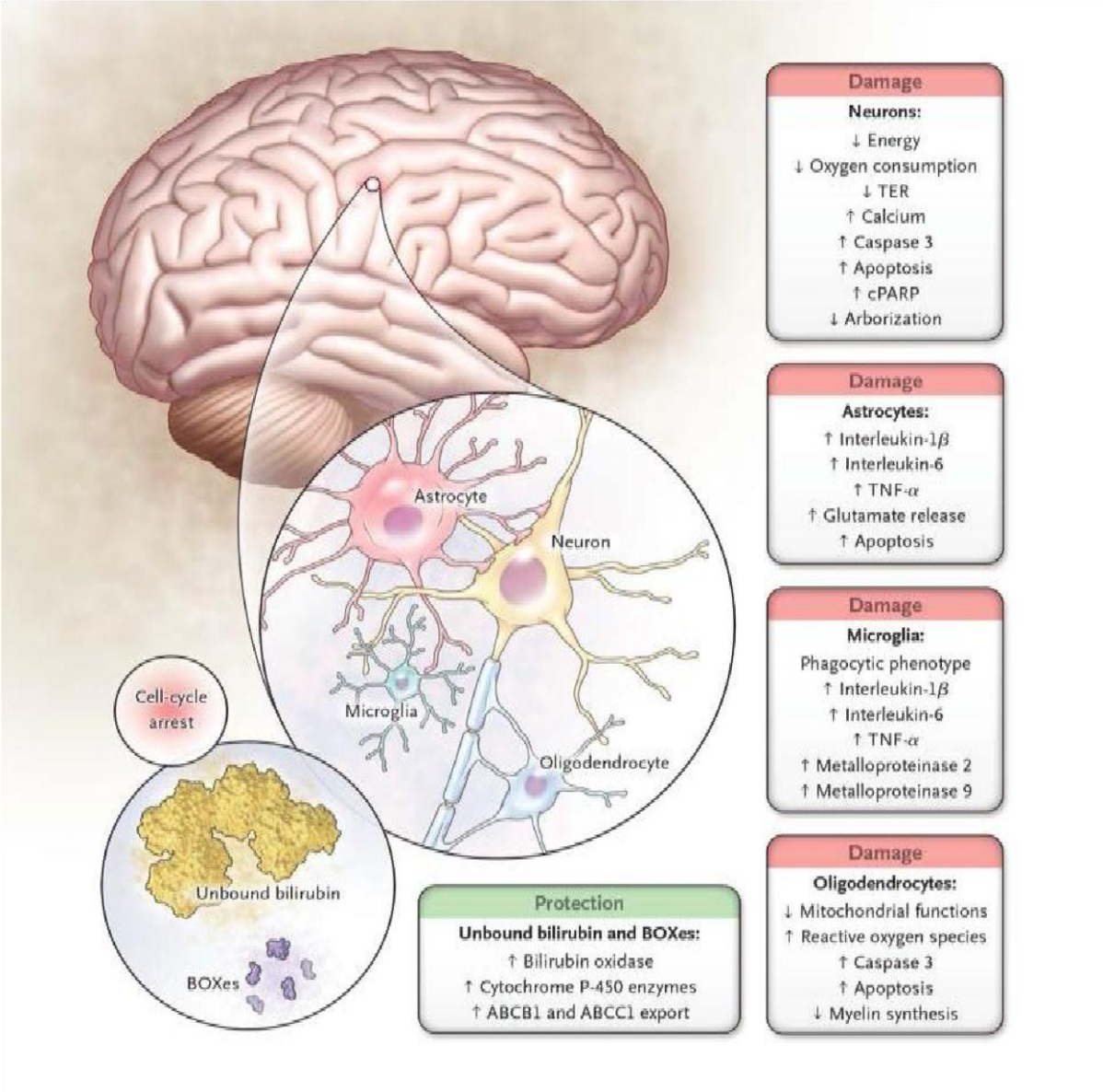

Figure 1. Cell types and metabolic processes affected by bilirubin in the CNS

Adapted from: Watchko J, Tiribelli C. Bilirubin-Induced Neurologic Damage - Mechanisms and Management Approaches. N Engl J Med .2013;369:2021-30. DOI: 10.1056/ NEJMra1308124 [34]

The main effects of bilirubin in neonates on neurons are decreased O_2 consumption and increased release of ca^{2+} and caspase 3, resulting in programmed cell death. There is also reduced dendritic and axonal arborization, which directly suggests impairment

of the intercellular exchange ability. A similar pattern is observed in oligodendrocytes, with increased apoptosis, impairment of the free radicals and oxidative stress (Redox), and decreased production of myelin. Microglial cells react to toxic injury associated with free unconjugated bilirubin by excessive release of proinflammatory cytokines and metalloproteinase activity as cells manifest the phagocytic phenotype. Similar proinflammatory pattern is observed in astrocytes, with enhanced release of glutamate and resultant programmed cell death. Simultaneously, neurons may reduce the intracellular concentration of bilirubin either by thrust out the pigment through the ABC transporters or by increasing the formation of the less toxic bilirubin oxidation products (BOXes) through bilirubin oxidase enzyme, cytochrome P-450 enzymes or both. These responses are protective, whereas all others result in cell damage; this confirms that once the intracellular concentration of bilirubin exceeds a toxic threshold (still to be defined), the polymorphic meta-bolic cascade leading to neurotoxicity ensues. The term cPARP denotes cleaved poly (adenosine diphosphate–ribose) polymerase, TNF-α tumor necrosis factor α, and TER transcellular resistance.[34]

The brain damage incurred by the toxicity of bilirubin is not always reversible. Nearly half of the neonates with moderate signs of ABE and almost all those with severe signs of ABE had persistent signs of bilirubin encephalopathy at the time of discharge or death. Eighty two percent of the group with moderate signs of ABE followed up had abnormal neuro-developmental outcomes at three months of age and some might maintain the state for their whole life[25]. It is too late for intervention in infant presenting with intermediate or advanced signs of ABE. Effective and prompt intervention during the early phase can prevent chronic kernicterus sequelae[26], thus

paediatricians should be familiar with the warning signs of ABE and ensure to intervene timely and effectively.

There is characteristic auditory finding in patients with bilirubin-induced neurological dysfunction is auditory neuropathy spectrum disorder, which is defined by abnormal auditory neuronal function (altered or missing auditory brainstem waveforms) in the presence of normal cochlear microphonics and otoacoustic emissions. The auditory system is particularly sensitive to the effects of bilirubin, ranging from subtle abnormalities in hearing and speech processing to complete deafness. Auditory pathway damage may occur at total serum/plasma bilirubin (TB) levels which were previously thought to be harmless, and may occur in the absence of other signs of classic kernicterus.

Auditory complications, a disabling neurological finding caused by bilirubin neurotoxicity, are typically characterized by varying degrees of auditory neuropathy (AN) / dys-synchrony (AD) ranging from central auditory processing difficulties with normal hearing to severe AN/AD with absent auditory brainstem responses, and possibly causing severe hearing loss and deafness [2]. In fact, the brainstem cochlear nuclei are said to be one of the first structures affected by elevated total bilirubin, followed by the auditory nerve [31,32]. Although the cochlea is not directly affected by elevated bilirubin levels, it is thought that damage to the cochlea may occur secondary to damage to the cochlear nucleus and/or auditory nerve [33].

BILIRUBIN INDUCED NEUROLOGICAL DYSFUNCTION.

The pilot kernicterus registry group in the U.S.A (1992-2004)[22] in an effort to provide a National reporting system proposed a BIND scoring system to grade the severity and progression of bilirubin induced neurological dysfunction among term and late term neonates. (Table 1.1)

TABLE A: BIND SCORING CRITERIA

CLINICAL SIGNS	BIND SCORE	ACUTE BILIRUBIN ENCEPHALOPHATHY
MENTAL STATUS		
Normal.	0	None
Sleepy, could be awaken	1	Subtle ABE
Lethargy and poor suck.	2	Moderate ABE
Semicoma, apnea and seizures.	3	Severe ABE
MUSLE TONE		
Normal	0	None
Mild to moderate hypotonic	1	Subtle ABE
Hypertonia arching of the back	2	Moderate ABE
Retrocolis, ophisthotonus	3	Severe ABE
CRY PATTERNS		
Normal	0	None
High pitched cry	1	Subtle ABE
Shrill difficult to console	2	Moderate ABE
Weak or absent cry	3	Severe ABE

INTERPRETATIONS

A BIND score of 7-9 represent advanced signs of ABE and urgent interventions needed to avoid further damage Score of 4-6 represent moderate signs of ABE and are easily reversible with prompt interventions.

Score of 1-3: consistent with subtle signs of ABE and are highly reversible.

The Pilot Registry group[22] followed up a hundred and twenty-five (125) patients with acute bilirubin encephalopathy who voluntarily reported to the registry to assess the root cause of their condition. Multiple providers at several sites managed this cohort of neonates for their new born jaundice and progressive hyperbilirubinemia. Clinical signs and symptoms of ABE, verbalized by care givers, were often inadequately elicited or noted and often not identified as an emergency. Clinical signs and symptoms of ABE were noted in 7 of 125 neonates with a subsequent diagnosis of kernicterus who were not re-examined and evaluated or treated for hyperbilirubinemia, although hyperbilirubinemia was noted at outpatient visits clinically. The remaining neonates, one hundred and eighteen had total serum bilirubin (TSB) levels >20mg per 100ml ($342\mu moll^{-1}$; range: 20.7 to 59.9mg per 100ml). There was no specific total serum bilirubin threshold which coincided with the onset of ABE and mortality was around 4% (5 of 125) in neonates readmitted at age less than one week. Progression of jaundice to hazardous levels and onset of neurological signs and symptoms were often not identified as neonatal care and medical supervision transitioned during the first week of life. Using the BIND scoring criteria, nine neonates with clinical signs and symptoms of ABE with a BIND scores less than four were identified. These neonates were labelled to have subtle or non-specific ABE. Severe sequelae were noted in 4 of 9 neonates. In contrast, among 91 of 116 neonates with advanced signs of ABE (BIND scores >6), nine neonates subsequently had no (n=3), mild (n=1) and moderate (n=5) post-icteric complications. The major underlying root cause for kernicterus was systems failure of services by multiple providers at multiple sites and inability to identify neonates who are at risk and manage severe hyperbilirubinemia in a timely manner.

Gamaleldin et al[24] described the importance of TSB level and other risk factors for neurotoxicity in predicting ABE in two hundred and forty nine (249) infants with total bilirubin levels of 25mg/dl (427umol/l) seen in Cairo university hospital over a period of twelve months. TSB values at admission ranged from 25mg (42 umol/l) to 76.4 mg/dl (1306.4 umol/l) and the threshold TSB level that identified ninety percent 90% of infants with bilirubin encephalopathy was 25mg/dl (434.3umol/l) when neurotoxicity risk factors were present. In contrast neurotoxicity was first seen at a total serum bilirubin level of 35.5mg/d l(607 umol/l) in one hundred and eleven infants without risk factors. Although the correlation between total serum bilirubin level and encephalopathy was poor, patients with hyperbilirubinemia resulting from Rhesus incompatibility developed BIND at an odds ratio of 48.6 and those with sepsis at an odds ratio of 20.6 as compared to other etiologies. At admission, forty-four neonates (18%) had moderate to severe signs of ABE (score 4 –9), 55 neonates had subtle signs of neurotoxicity (score 1–3), and150 neonates (60%) had no signs of ABE. Thirty-five neonates (14%) had signs of BE at discharge (9 [3.6%]) or at the time of death 26 [10.4%]) and all deaths were associated with signs of kernicterus[24].

In another study done in Iran to evaluate the prevalence of Bilirubin induced neurological dysfunction in term neonates with jaundice requiring exchange transfusion, one hundred and thirty three newborn were followed up over two years. The prevalence of BIND from this study was 48%, sixty four patients demonstrated signs and symptoms of acute bilirubin encephalopathy at the time of admission. Unsuccessful breast feeding was found to be a statistically significant risk factor for Bilirubin induced neurological dysfunction ($p= 0.001$)[27].

TABLE B: SUMMARISES BURDEN OF ABE FROM DIFFERENT SITES.

TITLE & AUTHORS	DESIGN	SAMPLE SIZE	BURDEN OF ABE
Risk factors for neurotoxicity in new born with severe hyperbilirubinemia. Iskandar I, Aborraya Sampson PD and Gamaleldin R et al .Cairo 2011[24].	Cross sectional study	N=249	40%.
ABE in term neonates requiring exchange transfusion. Seyedeh Khatami, P Ouya.Iran,2012	Cross sectional study	N=133	48%.

METHODOLOGY

Source of data

All hyperbilirubinemic neonates admitted (includes inborn and referred cases) in Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapur, fulfilling the inclusion and exclusion criteria.

173 cases of hyperbilirubinemia were studied in the span of 1.5 years.

Determination of sample size (n):

With 95% confidence level and margin of error of ±7.5%, a sample size of 173 subjects were enrolled into the study to determine the **"**Screening for bilirubin induced neurological dysfunction (BIND) among hyperbilirubinemic neonates, A hospital based prospective study."

By using the formula:

$$n = z^2 \frac{p(1-p)}{d^2}$$

where,

Z= statistic at 5% level of

significance d = margin of error

p = anticipated prevalence rate

(50%)

Statistical analysis for sample size determination

All characteristics will be summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) will be used. For categorical data, the number and percentage will be used in the data summaries and data will be analyzed by Chi square test for association, comparison of means using t test, ANOVA and diagrammatic

presentation.

Type of study

Prospective study

Selection criteria

Inclusion criteria:

All neonates with hyperbilirubinemia admitted to new born ward and NICU of Shri B.M. Patil Medical College, hospital and research centre and whose parents gave written informed consent.

Exclusion criteria:

1. Neonates with birth asphyxia defined as Apgar score of less than eight at five minutes.
2. Neonates with obvious central nervous system malformation like hydrocephalous.
3. Those with conjugated hyperbilirubinemia.
4. Neonates with clinical features of meningitis persistent convulsions, bulging fontanel and neck retraction.

Duration of study

December 2018 to June 2020 (1.5 years)

Method of study

A hospital based prospective study, a sample of 173 hyperbilirubinemic neonates were studied. Babies were assessed clinically for the development of icterus till they were discharged. Bilirubin level estimation was done if clinically icteric. Criteria for Significant Hyperbilirubinemia were decided on the basis of American Academy of Pediatrics (AAP) guidelines. In case of significant hyperbilirubinemia BIND scoring is noted. In all the BIND positive babies AABR was performed.

BIND SCORING CRITERIA

CLINICAL SIGNS	BIND SCORE	ACUTE BILIRUBIN ENCEPHALOPHATHY
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Score of 4-6 represent moderate signs of ABE and are easily reversible with prompt interventions.

Score of 1-3: consistent with subtle signs of ABE and are highly reversible.

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation. Chi-square (χ^2) test was used for association between two categorical variables.

The formula for the chi-square statistic used in the chi square test is:

$$\chi_c^2 = \sum \frac{(O_i - E_i)^2}{E_i}$$

The subscript "c" are the degrees of freedom. "O" is observed value and E is expected value. C= (number of rows-1)* (number of columns-1)

The difference of the means of analysis variables between two independent groups was tested by unpaired t test.

The t statistic to test whether the means are different can be calculated as follows:

$$t = \frac{(\overline{x_1} - \overline{x_2}) - (\mu_1 - \mu_2)}{\sqrt{\frac{{s_1}^2}{n_1} + \frac{{s_2}^2}{n_2}}}$$

where $\bar{x}_1$ = mean of sample 1

$\bar{x}_2$ = mean of sample 2

n_1 = number of subjects in sample 1

n_2 = number of subjects in sample 2

${s_1}^2$ = variance of sample 1 = $\frac{\Sigma(x_1 - \bar{x}_1)^2}{n_1}$

${s_2}^2$ = variance of sample 2 = $\frac{\Sigma(x_2 - \bar{x}_2)^2}{n_2}$

If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analysed using SPSS software v.23 (IBM Statistics, Chicago, USA) and Microsoft office 2007.

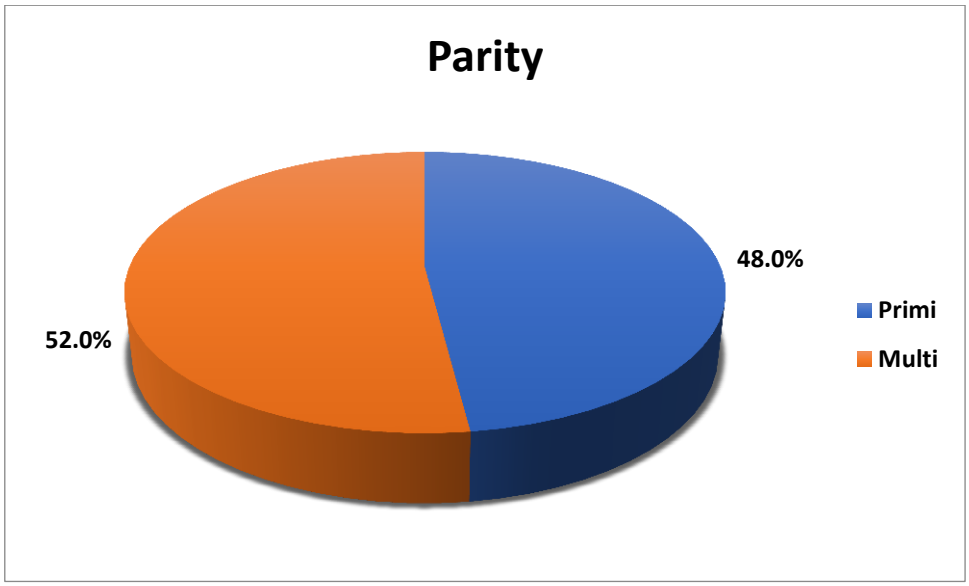
RESULTS

During the study period, a total of 173 hyperbilirubinemic neonates who met the inclusion criteria were enrolled into the study.

Table 1: Distribution of Cases according to Parity

Parity	N = 173	Percent
Primi	83	48
Multi	90	52
Total	173	100

Figure 1: Distribution of Cases according to Parity

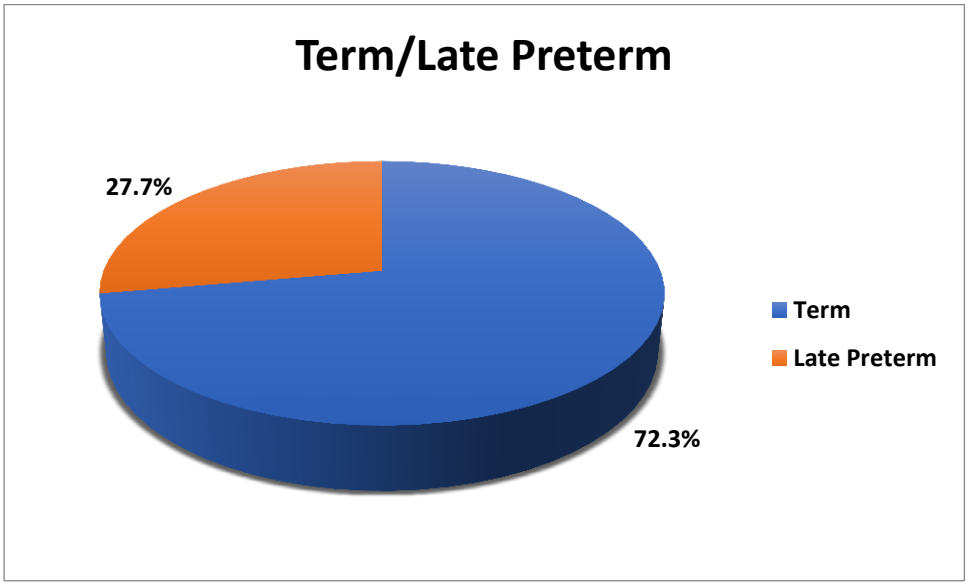


83 mothers were primi parous (48%) and 90 mothers were multiparous (52%).

Table 2: Distribution of Cases according to Term/Late Preterm

Term/Late Preterm		Percent
Term ≥ 37	125	72.3
Late Preterm ≥34 – 36^{+6}	48	27.7
Total	173	100

Figure2: Distribution of Cases according to Term/Late Preterm

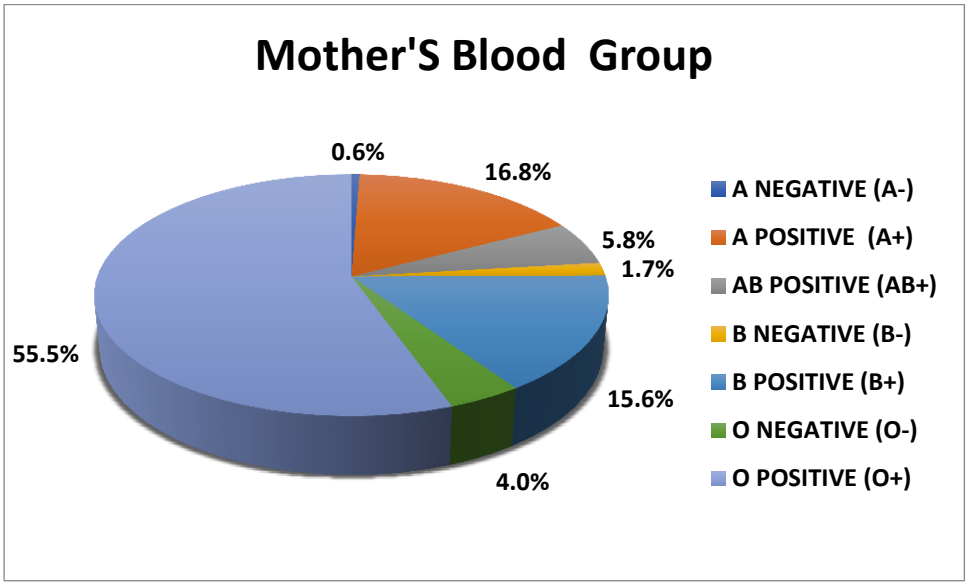


72.3 % of babies were term babies, and 27.7 % were late pre-term babies. (Table 2 and Figure 2).

Table 3: Distribution of Cases according to Mother's Blood Group

Mother's Blood Group	N =173	Percent
A NEGATIVE (A-)	1	0.6
A POSITIVE (A+)	29	16.8
AB POSITIVE (AB+)	10	5.8
B NEGATIVE (B-)	3	1.7
B POSITIVE (B+)	27	15.6
O NEGATIVE (O-)	7	4
O POSITIVE (O+)	96	55.5
Total	173	100

Figure 3: Distribution of Cases according to Mother's Blood Group

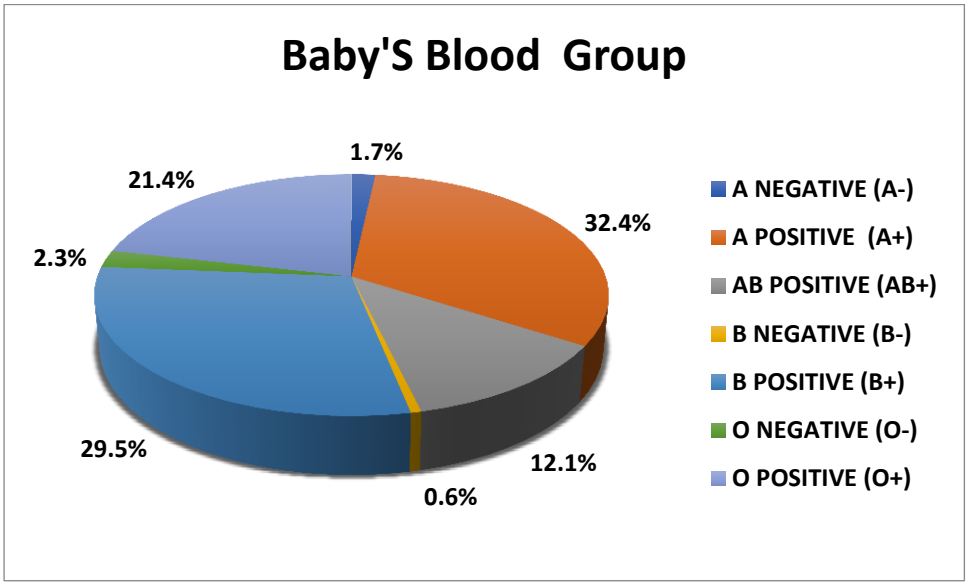


Most common mother's blood group among mothers was O positive.

Table 4: Distribution of Cases according to Baby's Blood Group

Baby's Blood Group	N = 173	Percent
A NEGATIVE (A-)	3	1.7
A POSITIVE (A+)	56	32.4
AB POSITIVE (AB+)	21	12.1
B NEGATIVE (B-)	1	0.6
B POSITIVE (B+)	51	29.5
O NEGATIVE (O-)	4	2.3
O POSITIVE (O+)	37	21.4
Total	173	100

Figure 4: Distribution of Cases according to Baby's Blood Group

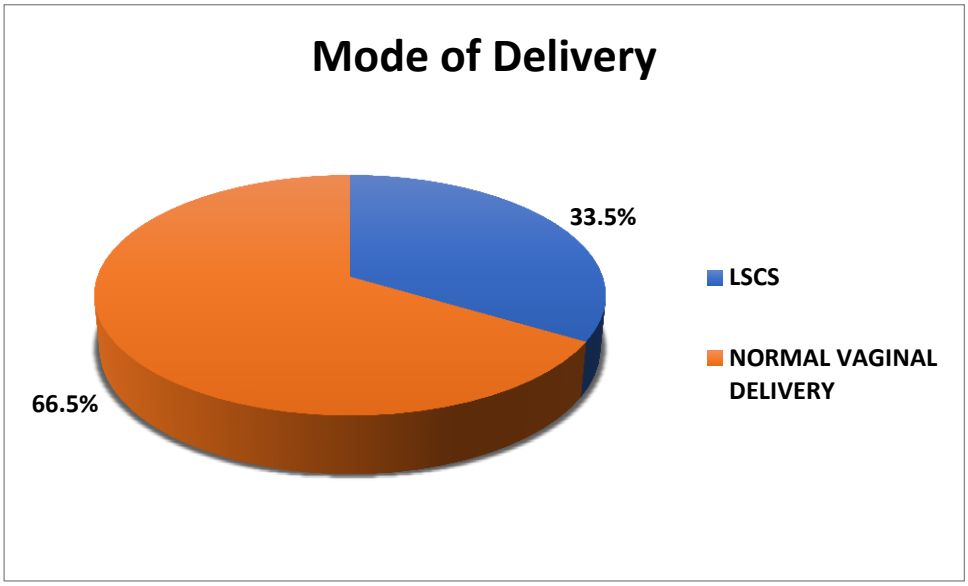


Among neonates B positive blood group was most common.

Table 5: Distribution of Cases according to Mode of Delivery

Mode of Delivery	N = 173	Percent
LSCS	58	33.5
Normal Vaginal Delivery	115	66.5
Total	173	100

Figure 5: Distribution of Cases according to Mode of Delivery

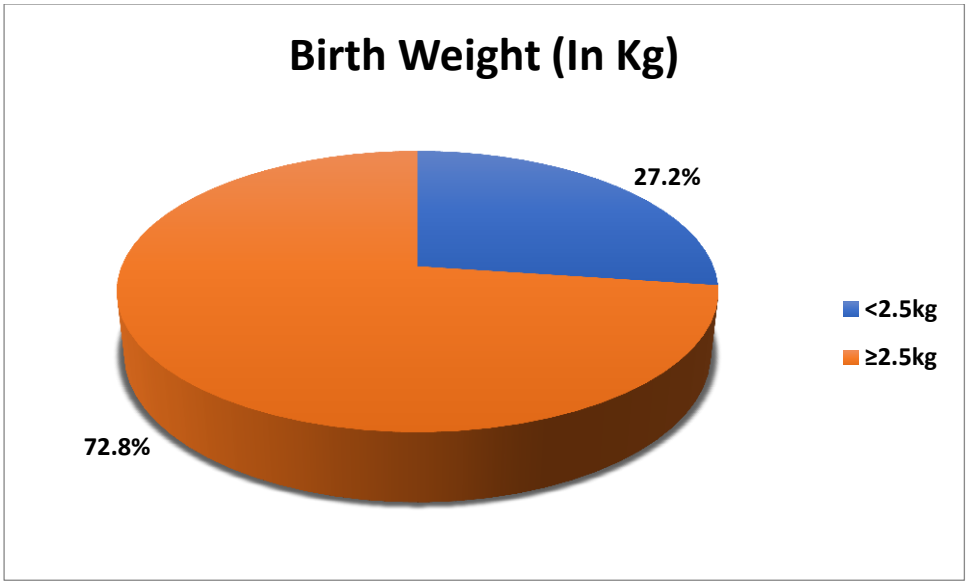


66.5 % neonates were delivered by normal vaginal delivery and 33.5% neonates by Caesarean section (Table 5 and Figure 5).

Table 6: Distribution of Cases according to Birth Weight

Birth Weight (In Kg)	N = 173	Percent
<2.5kg	47	27.2
≥2.5kg	126	72.8
Total	173	100

Figure 6: Distribution of Cases according to Birth Weight

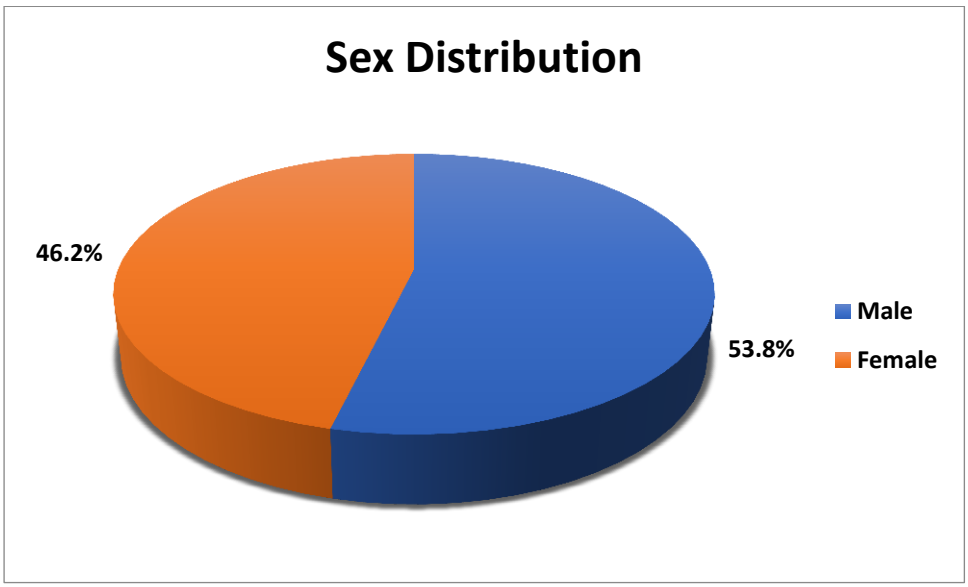


A majority of neonates (72.8%) had birth weight ≥ 2.5 Kg.

Table 7: Distribution of Cases according to Sex

Sex	N = 173	Percent
Male	93	53.8
Female	80	46.2
Total	173	100

Figure 7: Distribution of Cases according to Sex

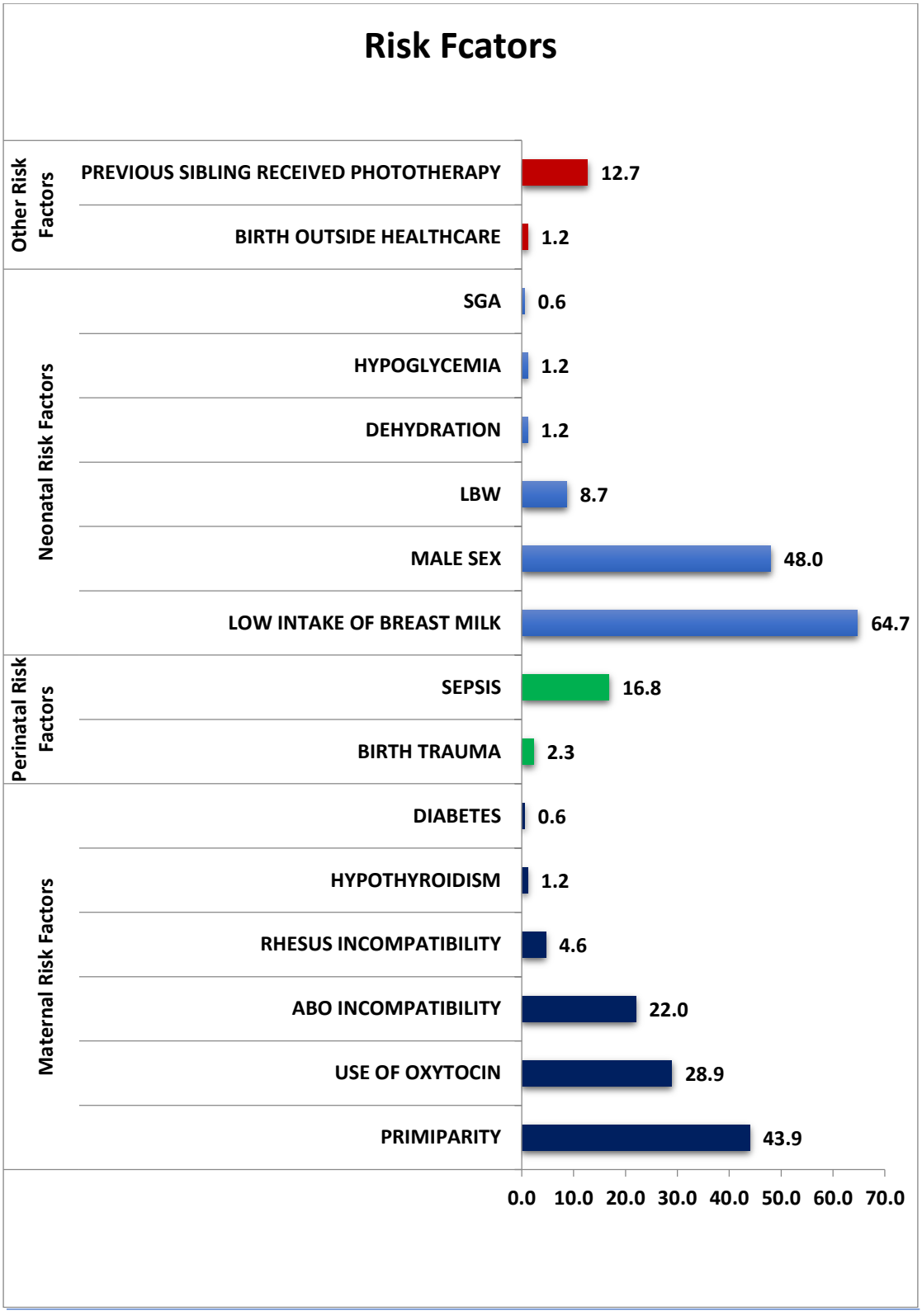


53.8 % neonates were males, and 46.2 % were females. The male: female ratio was found to be 1.16: 1 (Table 7 and Fig 7).

Table 8: Distribution of Cases according to Risk Factors

Maternal Risk Factors	**N = 173**	**Percent**
Primiparity	76	43.9
Use of oxytocin	50	28.9
ABO incompatibility	38	22.0
Rhesus incompatibility	8	4.6
Hypothyroidism	2	1.2
Diabetes	1	0.6
Perinatal Risk Factors	**N**	**Percent**
Birth trauma	4	2.3
Sepsis	29	16.8
Neonatal Risk Factors	**N**	**Percent**
Low intake of breast milk	112	64.7
Male sex	83	48.0
LBW	15	8.7
Dehydration	2	1.2
Hypoglycemia	2	1.2
SGA	1	0.6
Other Risk Factors	**N**	**Percent**
Birth outside healthcare	2	1.2
Previous sibling received phototherapy	22	12.7
Total	173	100

Figure 8: Distribution of Cases according to baseline Maternal and Neonatal Risk Factors



Among the maternal risk factors for hyperbilirubinemia, primiparity posed a major risk (43.9 %), followed by the use of oxytocin (28.9 %) and ABO incompatibility (22.0 %). Among the perinatal risk factors, 16.8 % neonates presented with sepsis and 2.3 % had birth trauma. Low intake of breast milk was a major neonatal risk factor (64.7%), followed by male sex (48%) and low birth weight (8.7%). Besides, 12.7 % infants had previous siblings who had received phototherapy. (Table 8)

Table 9: Descriptive Statistics of baseline characteristics.

Descriptive Statistics	**Range**	**Mean**	**SD**
Age (In Hours)	3-280	70.3	31.0
Birth Weight (In Grams)	2080-3720	2665.8	268.6
Admission weight (In Grams)	1980-3620	2183.1	259.2

The mean age of the neonates at admission in the study was 70.3 hours ± 31.0 hours, the range being 3 to 280 hours. Birth weight of the neonates ranged from 2080 grams to 3720 grams, the mean being 2665.8 grams. Weight of the neonates at the time of admission ranged from 1980 grams to 3620 grams, with a mean of 2183.1 grams ± 259.2 grams (Table 9).

Table 10: Descriptive Statistics of Icteric Cases

Descriptive Statistics	Range	Mean	SD
History of duration of jaundice.	4-76	10.4	7.9

The duration of having jaundice ranged from 4 -76 hours and mean duration was 10.4 hours ± 7.9 hours (Table 10).

Table 11: Descriptive Statistics of Bilirubin and BIND Score

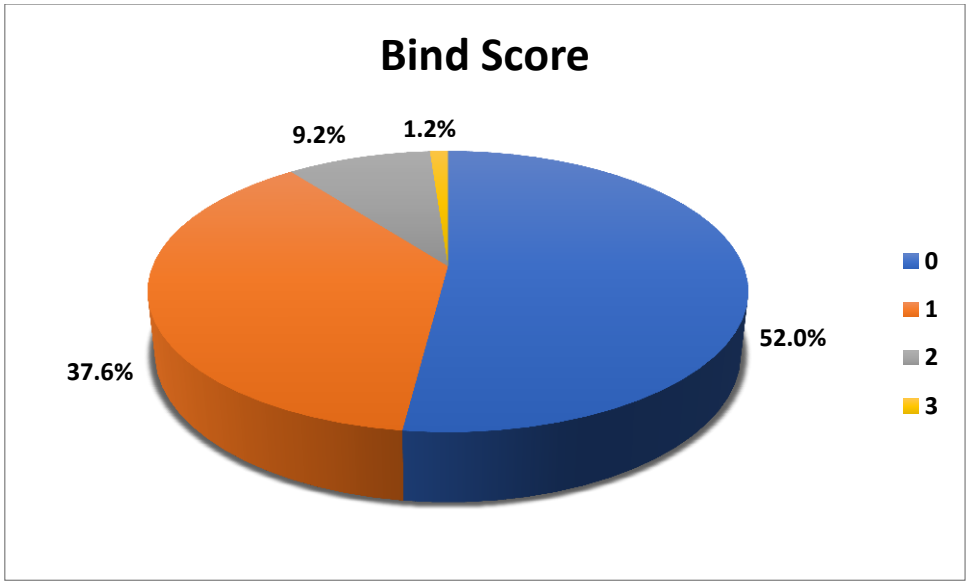
Descriptive Statistics	Range	Mean	SD
Highest Total Serum Bilirubin Value (TSB)	7.1-27	14.1	3.2
Highest Unconjugated Bilirubin Value (UCB)	7.0-26.3	13.2	3.0
BIND Score	0-3	1.6	0.7

The initial total bilirubin value of the study group ranged between 7.1 mg/dL to 27 mg/dL with a mean of 14.1 mg/dL ± 3.2 mg/dL. The initial value of unconjugated bilirubin ranged between 7.0 – 26.3 mg/dL with a mean value of 13.2 mg/dL ± 3.0 mg/dL of the study group. The BIND score ranged between 0 to 3, with a mean of 1.6 ± 0.7. (Table 11).

Table 12: Distribution of Cases according to BIND Score

BIND Score	N	Percent
0	90	52
1	65	37.6
2	16	9.2
3	2	1.2
Total	173	100

Figure 9: Distribution of Cases according to BIND Score

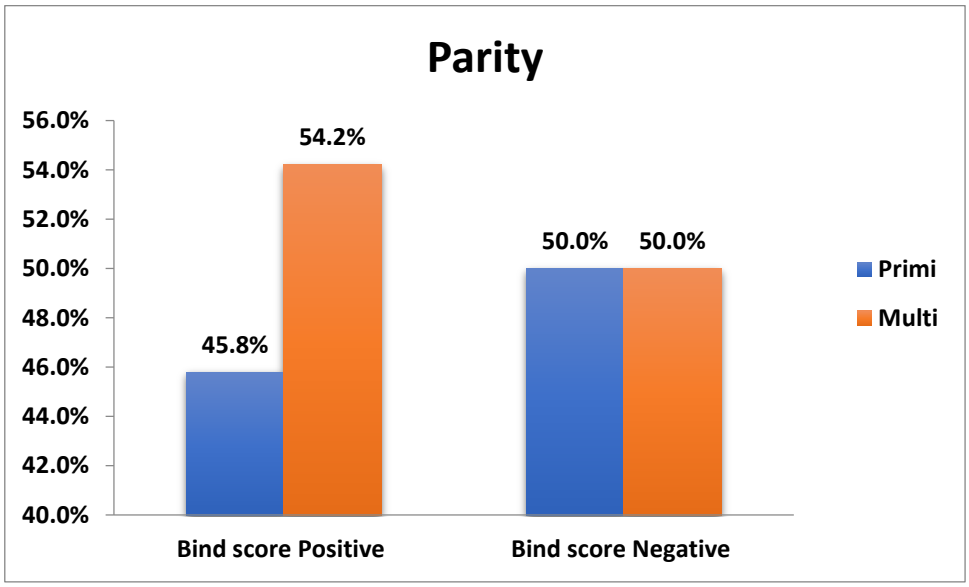


A major portion of neonates in the study group (52 %) had a BIND score of 0 and were considered negative. 37.6 % neonates had a BIND score of 1, 9.2% had a BIND score of 2 and 1.2 % has a BIND score of 3 and were considered positive(Table 12 and Figure 9).

Table 13: Distribution of Parity according to BIND Score

Parity	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
Primi	38	45.8%	45	50.0%	0.579
Multi	45	54.2%	45	50.0%	
Total	83	100.0%	90	100.0%	

Figure 10: Distribution of Parity according to BIND Score

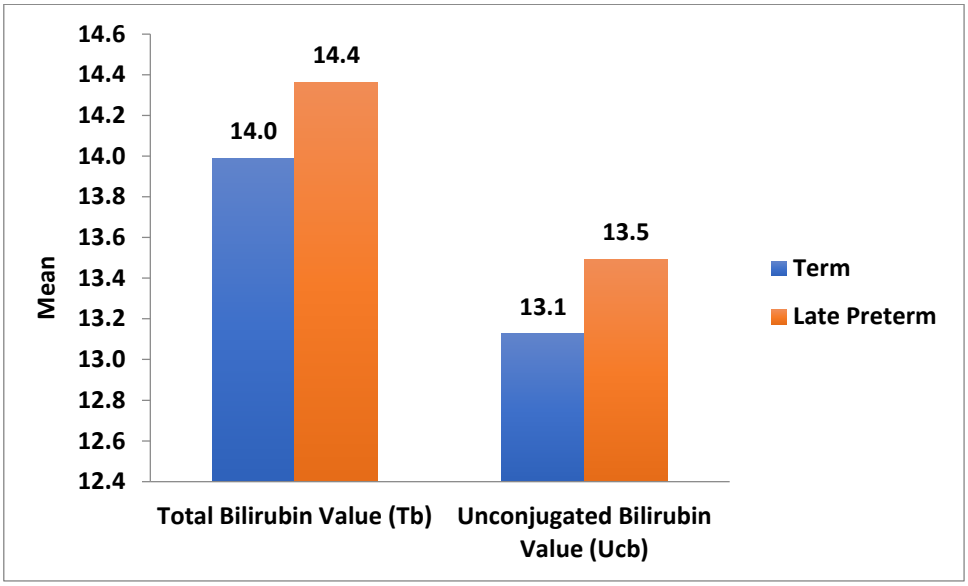


In the neonate group which had positive BIND score, neonates whose mothers were multiparous predominated as compared to neonates whose mothers were primiparous (54.2% vs. 45.8%). In the neonates who had a negative BIND score, the number of neonates whose mothers were primiparous and multiparous were equal. The difference in the two groups with respect to parity was not statistically significant (p=0.579).

Table 14: Distribution of Term and Late preterm according to bilirubin levels

Bilirubin	Term		Late Preterm		p value
	Mean	SD	Mean	SD	
Total Bilirubin Value (Tb)	14.0	3.2	14.4	3.3	0.492
Unconjugated Bilirubin Value (Ucb)	13.1	3.0	13.5	3.1	0.482

Figure 11: Distribution of Term and Late preterm according to bilirubin levels

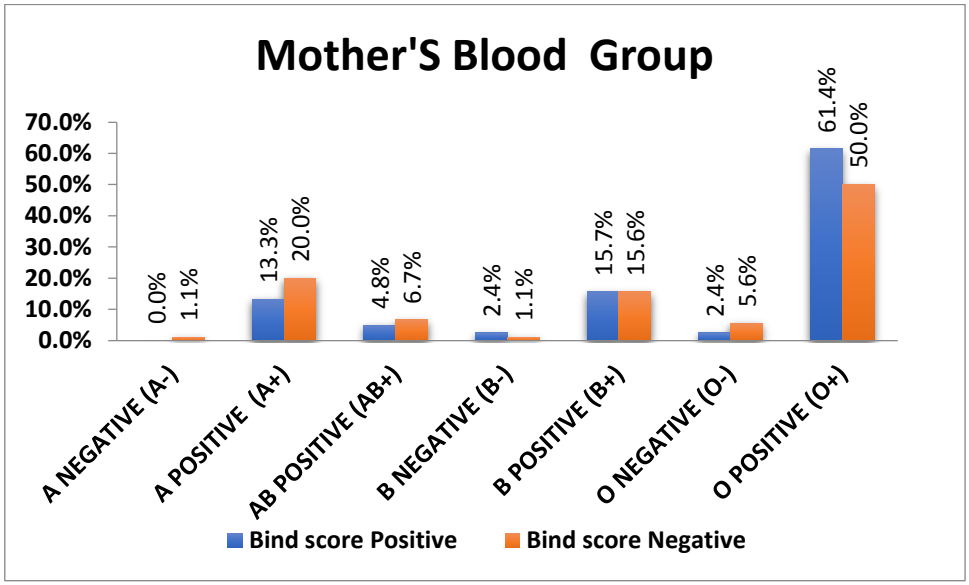


The late pre- term neonates had a mean Tb value slightly more than that of term neonates (14.4 mg/dl ± 3.2 mg/dl vs 14.0 mg/dl ± 3.2 mg/dl) . However, this difference was not statistically significant (p=0.492). The mean Ucb levels were also more in the late pre-term neonates as compared to term neonates (13.5 mg/dl ± 3.1 mg/dl vs 13.1 mg/dl ± 3.0 mg/dl). However, this difference was not statistically significant (p=0.482).

Table 15: Distribution of Mother's Blood Group according to BIND Score

Mother's Blood Group	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
A NEGATIVE (A-)	0	0.0%	1	1.1%	0.564
A POSITIVE (A+)	11	13.3%	18	20.0%	
AB POSITIVE (AB+)	4	4.8%	6	6.7%	
B NEGATIVE (B-)	2	2.4%	1	1.1%	
B POSITIVE (B+)	13	15.7%	14	15.6%	
O NEGATIVE (O-)	2	2.4%	5	5.6%	
O POSITIVE (O+)	51	61.4%	45	50.0%	
Total	83	100.0%	90	100.0%	

Figure12: Distribution of Mother's Blood Group according to BIND Score

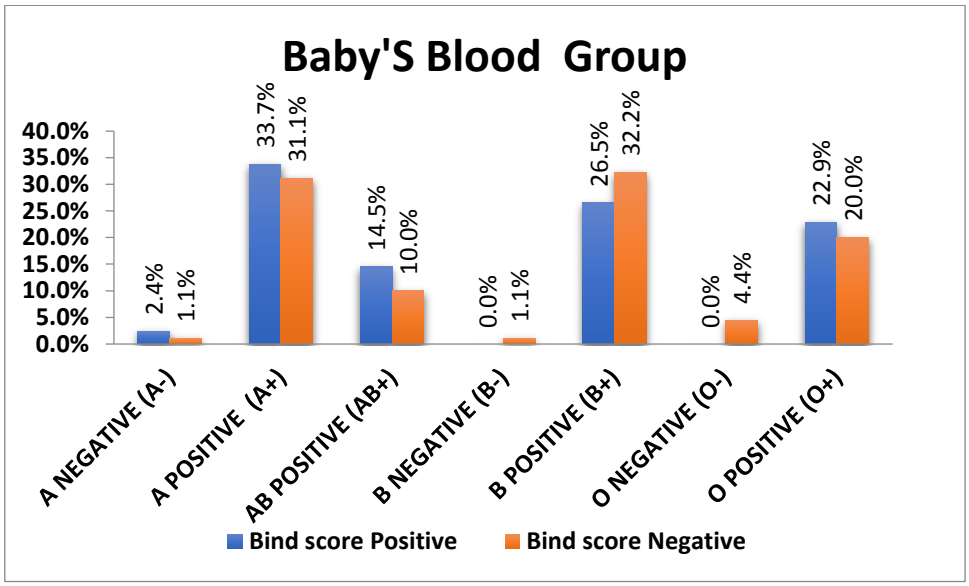


There was no significant difference between the BIND score positive and BIND score negative neonates with respect to their mothers having a particular blood group (p=0.564)

Table 16: Distribution of Baby's Blood Group according to BIND Score

Baby's Blood Group	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
A NEGATIVE (A-)	2	2.4%	1	1.1%	0.372
A POSITIVE (A+)	28	33.7%	28	31.1%	
AB POSITIVE (AB+)	12	14.5%	9	10.0%	
B NEGATIVE (B-)	0	0.0%	1	1.1%	
B POSITIVE (B+)	22	26.5%	29	32.2%	
O NEGATIVE (O-)	0	0.0%	4	4.4%	
O POSITIVE (O+)	19	22.9%	18	20.0%	
Total	83	100.0%	90	100.0%	

Figure 13: Distribution of Baby's Blood Group according to BIND Score

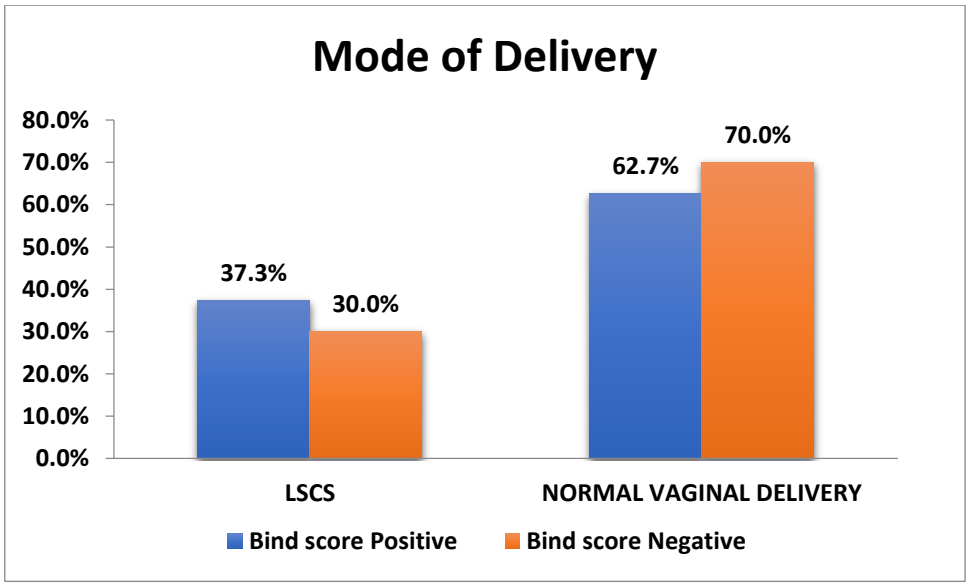


There was no significant difference between the BIND score positive and BIND score negative neonates with respect to having a particular blood group (p=0.372).

Table 17 : Distribution of Mode of Delivery according to BIND Score

Mode of Delivery	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
LSCS	31	37.3%	27	30.0%	0.306
Normal Vaginal Delivery	52	62.7%	63	70.0%	
Total	83	100.0%	90	100.0%	

Figure 14 : Distribution of Mode of Delivery according to BIND Score

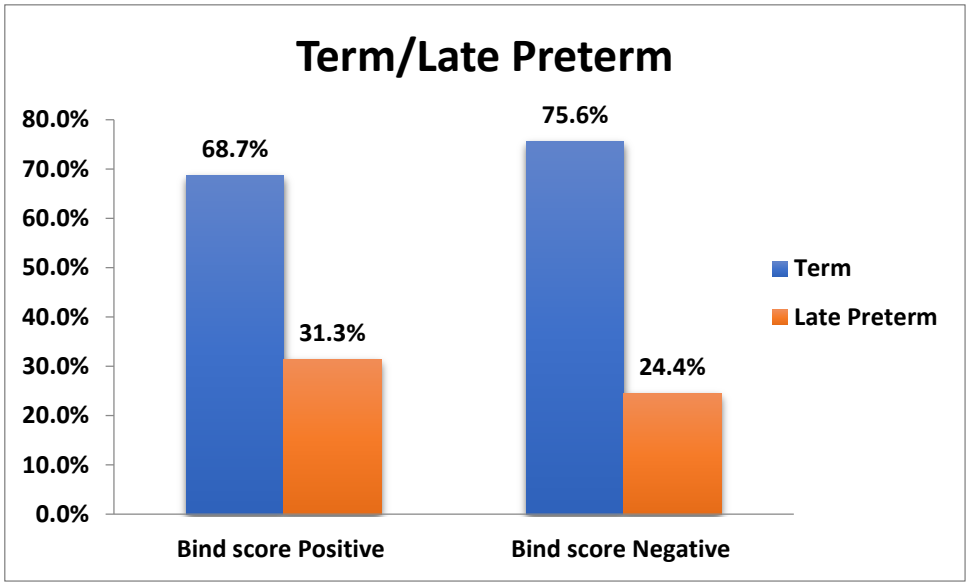


Among the neonates who had a positive BIND score, 62.7 % were delivered by normal vaginal delivery and 37.3 % by caesarean section. Among the neonates who had a negative BIND score, 70.0 % were delivered by normal delivery and 30.0 % by LSCS. Though the percentage of neonates who were delivered by caesarean section were more in the BIND positive group as compared to BIND negative groups, this difference was not statistically significant(p=0.306).

Table 18: Distribution of Term/Late Preterm according to BIND Score

Term/Late Preterm	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
Term	57	68.7%	68	75.6%	0.313
Late Preterm	26	31.3%	22	24.4%	
Total	83	100.0%	90	100.0%	

Figure 15: Distribution of Term/Late Preterm according to BIND Score

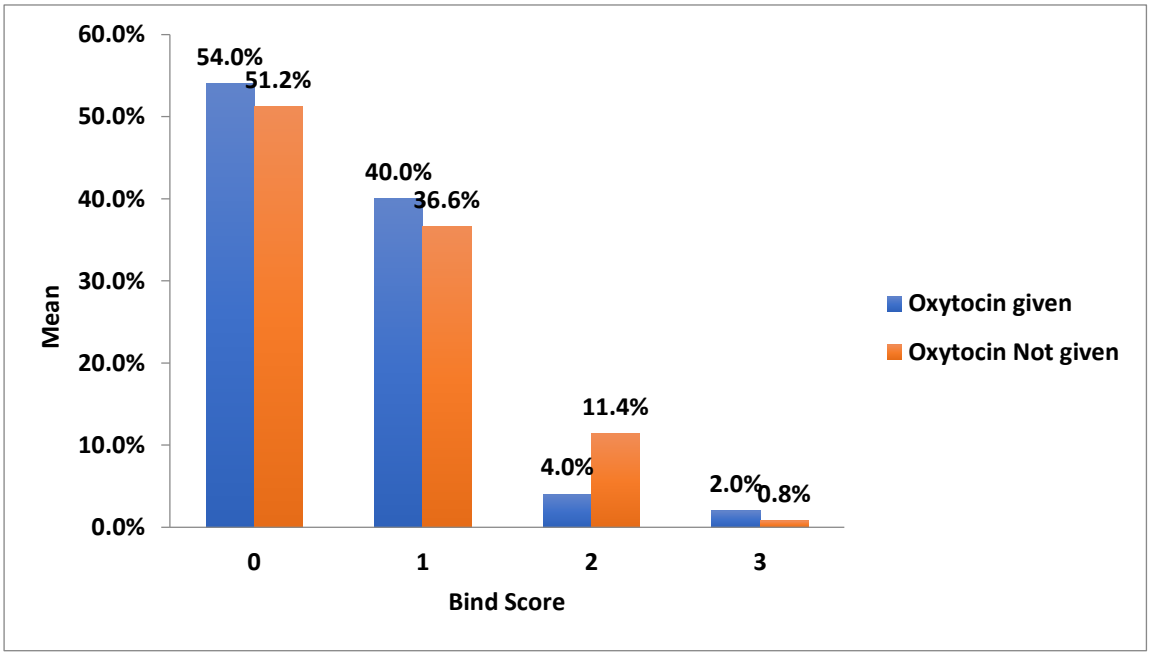


In the neonate group whose BIND score was positive, 68.7 % neonates were term neonates while 31.3 % were late pre term neonates. In the BIND score negative group, 75.6 % neonates were term neonates while 24.4% neonates were late pre term neonates. Though the percentage of late pre term neonates in the positive BIND group was more as compared to BIND negative group, this difference in distribution was not statistically significant (p=0.313) (Table 18 and Figure 15).

Table 19: Distribution on oxytocin used or not used according to BIND Score

Bind Score	Oxytocin given		Oxytocin Not given		p value
	N	%	N	%	
0	27	54.0%	63	51.2%	0.422
1	20	40.0%	45	36.6%	
2	2	4.0%	14	11.4%	
3	1	2.0%	1	0.8%	
Total	50	100.0%	123	100.0%	

Figure 16 : Distribution on oxytocin used or not used according to BIND Score

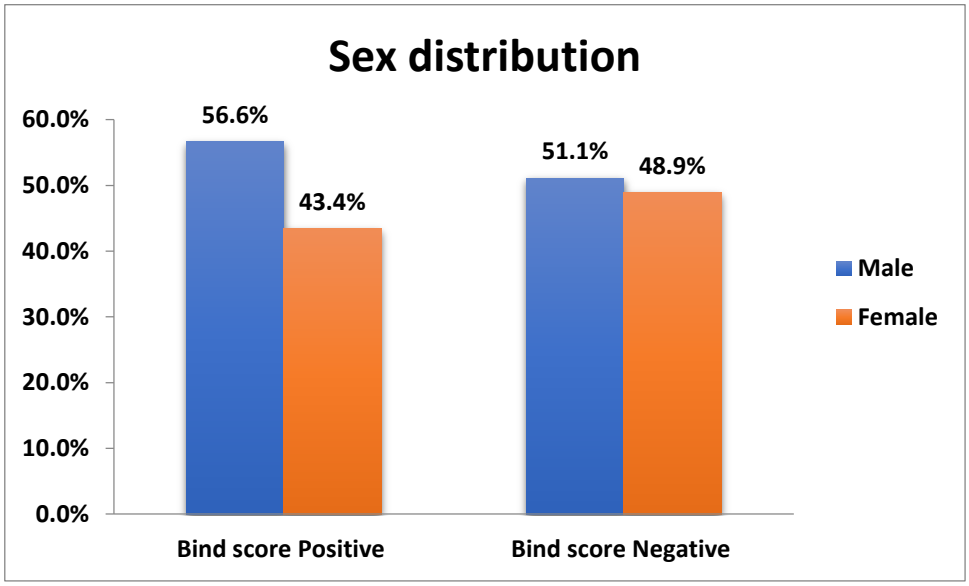


In the oxytocin group, 54 % of neonates had a BIND Score of 0, 40 % had a bind score of 1 and 4% had a BIND Score of 2 and 2% of neonates had a BIND Score of 3.Among the neonates whose mothers did not need oxytocin to induce labour, 51.2 % had a BIND score of 0,36.6% neonates had a BIND Score of 1, 11.4% had a BIND score of 2 and 0.8% neonates had a BIND Score of 3. There was no significant relationship between the use/non-use of oxytocin and BIND score (p=0.422).

Table 20: Distribution of Sex according to BIND Score

Sex	Bind score Positive		Bind score Negative		p value
	N	%	N	%	
Male	47	56.6%	46	51.1%	0.467
Female	36	43.4%	44	48.9%	
Total	83	100.0%	90	100.0%	

Figure 17: Distribution of Sex according to BIND Score

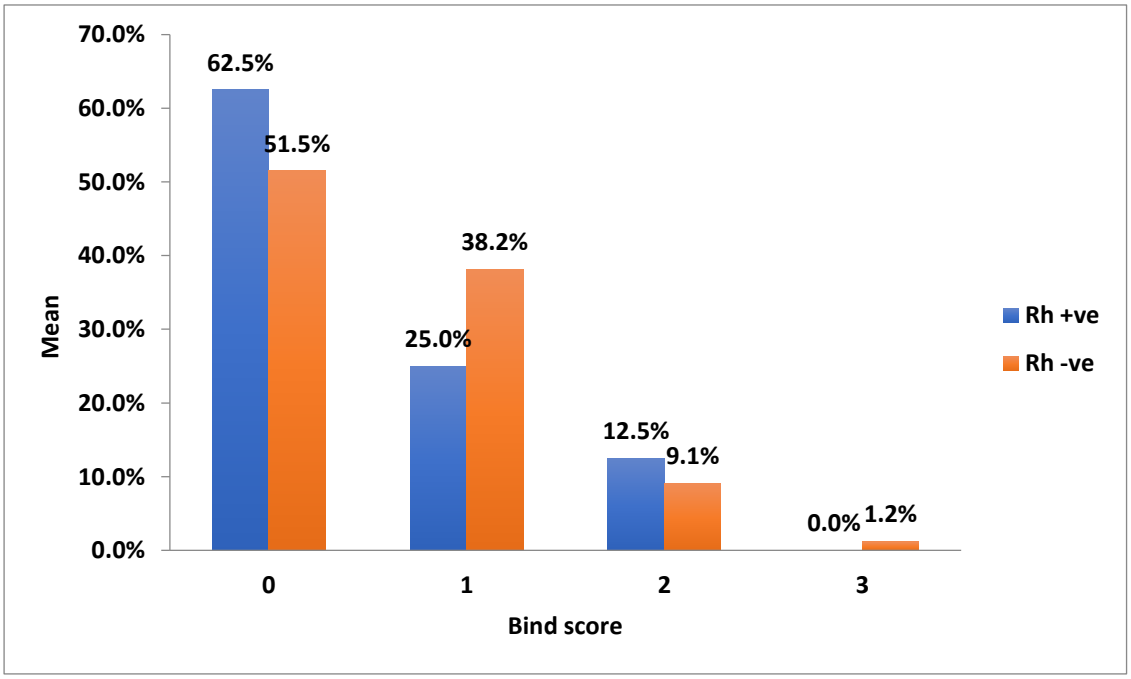


Among the neonates who had a positive BIND score, 56.6% were males and 43.4 % were females. In the BIND score negative group, 51.1 % were males and 48.9% were females. This distribution of sex among the two groups was not statistically significant (p=0.467).

Table 21: Distribution of Rh Status according to BIND Score

Bind Score	Rh +ve		Rh -ve		p value
	N	%	N	%	
0	5	62.5%	85	51.5%	0.868
1	2	25.0%	63	38.2%	
2	1	12.5%	15	9.1%	
3	0	0.0%	2	1.2%	
Total	8	100.0%	165	100.0%	

Figure 18: Distribution of Rh Status according to BIND Score

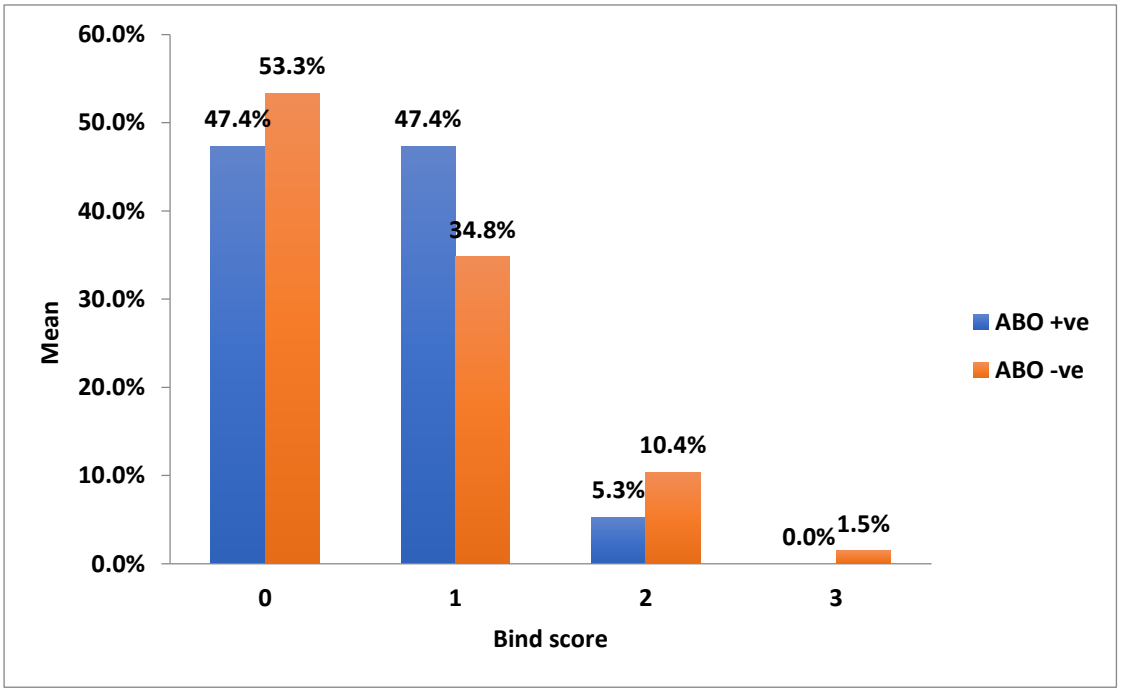


In the Rh+ve group, 62.5% neonates had a BIND score of 0, 25.0% of neonates had a BIND score of 1, 12.5 % of neonates had a BIND score of 2, and no neonate had a BIND score of 3. In the Rh –ve group, 51.5 % of neonates had a BIND score of 0, 38.2 % of neonates had a BIND score of 1, 9.1 % of neonates had a BIND score of 2, and 1.2 % of neonates had a BIND score of 3.

Table 22: Distribution of ABO blood group Status according to BIND Score

Bind Score	ABO +ve		ABO -ve		p value
	N	%	N	%	
0	18	47.4%	72	53.3%	0.416
1	18	47.4%	47	34.8%	
2	2	5.3%	14	10.4%	
3	0	0.0%	2	1.5%	
Total	38	100.0%	135	100.0%	

Figure 19: Distribution of ABO blood group Status according to BIND Score



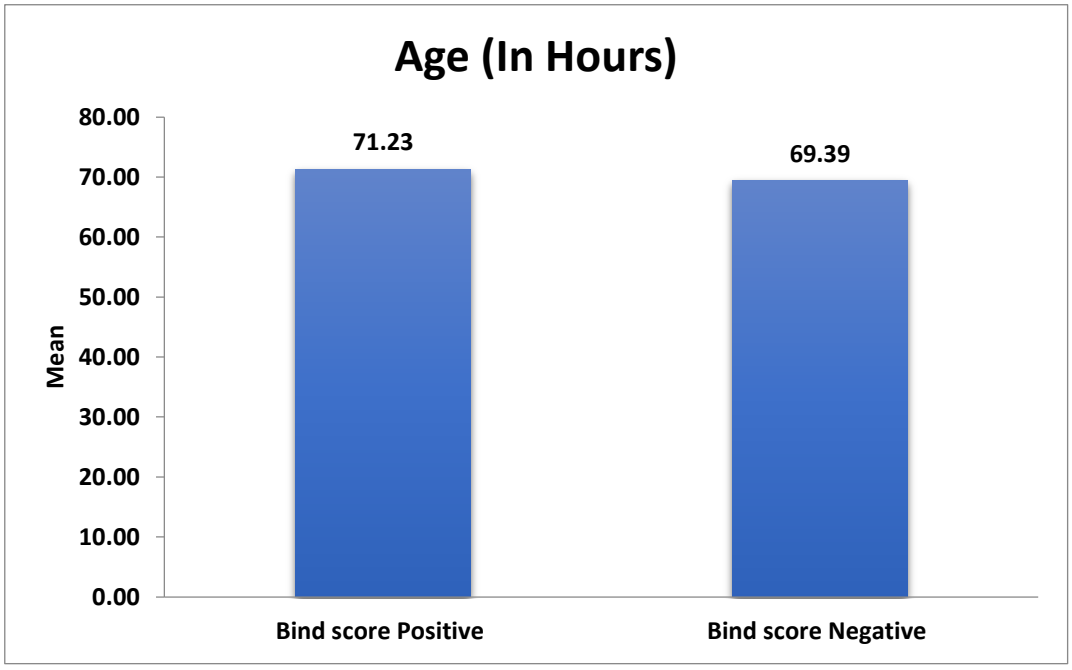
In the ABO+ve group, 47.4% neonates had a BIND score of 0, and an equal number of neonates had a BIND score of 1, 5.3 % of neonates had a BIND score of 2, and no neonate had a BIND score of 3. In the ABO –ve group, 53.3 % of neonates had a BIND score of 0, 34.8 % of neonates had a BIND score of 1, 10.4 % of neonates had a BIND score of 2, and 1.5 % of neonates had a BIND score of 3.

Table 23: Comparison of Mean Parameters according to BIND Score

Parameters	Bind score Positive		Bind score Negative		p value
	Mean	SD	Mean	SD	
Age (In Hours)	71.23	26.42	69.39	34.84	0.698
Birth Weight (In Grams)	2663.87	290.39	2667.51	248.55	0.929

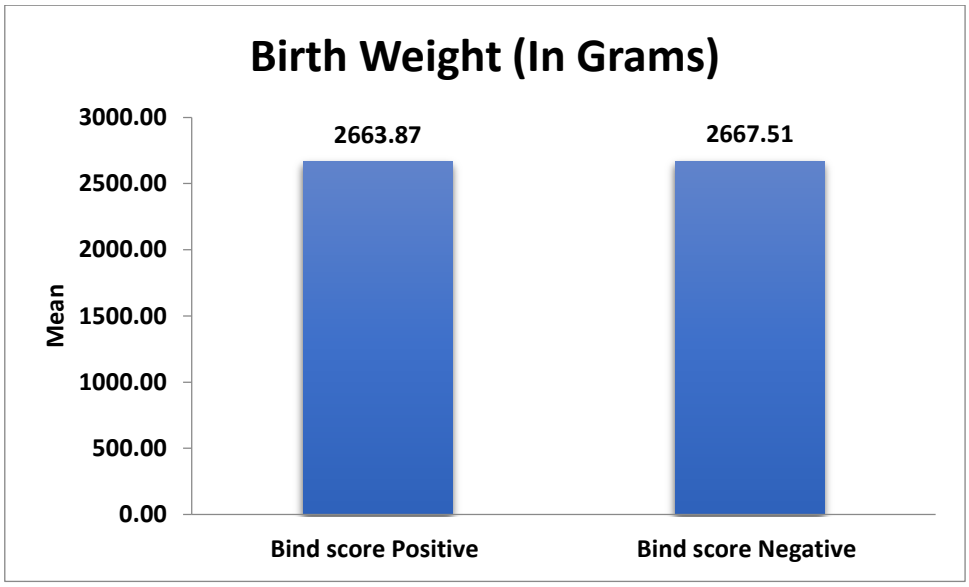
Note: * significant at 5% level of significance (p<0.05)

Figure 20.1: Comparison of Age according to BIND Score



The mean age of neonates who had a positive BIND score was 71.23 hours ± 26.42 hours, while that of neonates having a negative BIND score was 69.39 hours ± 34.84 hours. The difference between the two groups was **not statistically significant** (p=0.698) .

Figure 20.2: Comparison of birth weight according to BIND Score

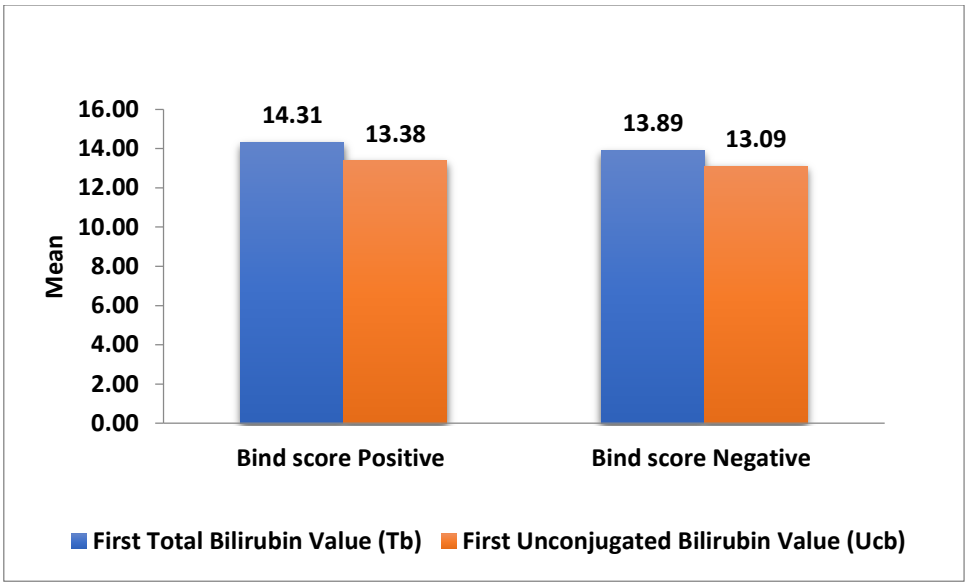


Neonates who had a positive BIND score had a mean birth weight of 2663.87 grams ± 290.39 grams and neonates having negative BIND score had mean birth weight of 2667.51 grams ± 248.55 grams. The difference between the two groups was **not statistically significant** (p=0.929).

Table 24: Comparison of Mean Bilirubin Parameters according to BIND Score

Parameters	Bind score Positive		Bind score Negative		p value
	Mean	SD	Mean	SD	
First Total Bilirubin Value (Tb)	14.31	3.91	13.89	2.37	0.399
First Unconjugated Bilirubin Value (Ucb)	13.38	3.70	13.09	2.31	0.538

Figure 21: Comparison of Mean Bilirubin Parameters according to BIND Score



The mean initial total bilirubin value of the neonate group who had a positive BIND score was more than that of the group who had a negative BIND score (14.31mg/dL ± 3.91 mg/dL vs. 13.89 mg/dL ± 2.37 mg/dL). However, this difference was **not statistically significant** (p=0.399). Similarly, the initial unconjugated bilirubin value was higher in the neonates who had a positive BIND score as compared to the neonates who had negative BIND score (13.38 mg/dL ± 3.70 mg/dL vs. 13.09 mg/dL ± 2.31 mg/dL). However, this difference **was not statistically significant** (p=0.538).

DISCUSSION

Injury caused by deposition of free unconjugated bilirubin in the neurons leads to permanent injury in neonates. Neonatal hyperbilirubinemia is one of the most prevalent clinical problems observed during the 1st week of life affecting approximately 60% of term and 80% of preterm neonates. Pathophysiology of the jaundice is the same in term and preterm neonates, but premature babies are at a higher risk of developing hyperbilirubinemia and BIND. High bilirubin level may cause neurological impairment even in term neonates. Approximately 5-10% of them have clinically significant hyperbilirubinemia which may lead to bilirubin induced neurological dysfunction if untreated (Rennie, 2010) [36].

In our study, neonates who had multiparous mothers outnumbered neonates who had primiparous mothers (52 % vs 48 %) (Table 1 and Figure 1). Jamir et al [37] reported 44.3 % primi births. There are conflicting views on the correlation of parity with hyperbilirubinemia. According to Rochjati [38], parity > 4 is one of the risk factors in pregnant women as it increases the risk of occurrence of complications of pregnancy, fetal growth disorders, asphyxia and prematurity. The occurrence of antepartum haemorrhage, impaired placenta in pregnancy grandemultipara causes the disruption of transportation of food and oxygen from the mother to the fetus leading to a low birth weight baby. Winkjosastro[39] (2008) also report that a high parity results in a baby that is born with low birth weight and consequently hyperbilirubinemia.

It is well documented that there is an increased risk for significant hyperbilirubinemia with decreasing gestational age. Late pre-term infants account for a large number of hospital readmissions for management of jaundice and hyperbilirubinemia [38]. Premature infants are known to be especially susceptible to bilirubin neurotoxicity,

with kernicterus reported following TB levels far lower than the threshold expected in term neonates [17]. Similarly, among extremely pre-term neonates, unconjugated bilirubin (UB) is proportional to gestational age, meaning that the most premature infants have the highest UB, even for similar TB levels. In our study, 72.3 % babies were term babies, and 27.7% were late pre-term babies. (Table 2 and Figure 2). Jamir et al [37] reported 64% term neonates in their study.

In our study, 55.5% neonates had mothers whose blood group was O positive (most common), 16.8% neonates had mothers whose blood group was A positive, which is second most common (Table 3 and Figure 3)

In our study, 32.4 % neonates had A positive blood group (most common), 29.5 % had B positive blood group which is second most common (Table 4 and Figure 4).
In our study 66.5 % neonates were delivered by normal vaginal delivery and 33.5% neonates by Caesarean section (Table 5 and Figure 5). A study by Jamir et al [37] reported that 84 % neonates were delivered by normal vaginal delivery in their study (n=150). Nepal D [40] et al reported 87.7 % neonates were delivered by spontaneous vaginal delivery, 8.2 % by caesarean section and 4.1 % by instrumental aid.

Majority of the neonates in our study (72.8 %) had birth weight more than 2.5 kg (Table 6 and Figure 6). Jamir et al [37] reported that 51.3 % of neonates had adequate birth weight.

In our study, 53.8 % neonates were males, and 46.2 % were females. The male: female ratio was found to be 1.16: 1 (Table 7 and Fig 7). In a retrospective study conducted Nepal D [40] in 2009, male babies (72.6%) outnumbered female babies (1), as in our study.

In our study, among the maternal risk factors for hyperbilirubinemia, primiparity posed a major risk (43.9 %), followed by the use of oxytocin (28.9 %) and ABO incompatibility (22.0 %). Among the perinatal risk factors, 16.8 % neonates presented with sepsis and 2.3 % had birth trauma. Low intake of breast milk was a major neonatal risk factor (64.7%), followed by male sex (48%) and low birth weight (8.7%). Besides, 12.7 % infants had previous siblings who had received phototherapy. (Table 8 and Figure 8). Patel et al.[41] have reported the incidence of ABO incompatibility in their study to be 13.79% and that of Rh incompatibility to be 1.37%. Jamir [37] et al have reported that the most common etiological factor for pathological neonatal hyperbilirubinemia was due to deficiency of enzyme G6PD (12%). This was followed by prematurity (8.7%) and sepsis (5.3%). The other common causes included breast milk jaundice (4%), Rh isoimmunisation (2%), ABO incompatibility (1.3%) and hypothyroidism (1.3%). Lesser common causes were in neonates of diabetic mothers (0.7%) and cephalhematoma (0.7%).

Arun Babu [42] et al have reported that Rh incompatibility was an independent predictor of abnormal development in babies with neonatal jaundice. Sumangala Devi and Bindu [43] found that there was a statistically significant relationship between hyperbilirubinemia and Low Birth Weight (LBW), pre-term delivery, Pre-term premature rupture of the membranes (PPROM), breast feeding, neonatal infection, instrumental delivery and presence of Gestational Diabetes Mellitus and Intra Uterine Growth Restriction (IUGR).

Mala Kumar et al [44] reported that ABO/Rh incompatibility (odds ratio 4.00; 95% CI: 1.13–14, p = 0.030), a positive Coomb's test (odds ratio 5.7; 95% CI: 1.53–21.4, p = 0.0096), culture-proven sepsis (odds ratio 16; 95% CI: 0.82–312, p = 0.067), were found to be significant risk factors for development of ABE.

The pooled data from a meta-analysis of 13 studies by Olusanya [45] from low and middle income countries showed that ABO incompatibility (OR, 4.01; 95% CI:2.44-6.61), Rhesus hemolytic disease (OR, 20.63; 95% CI:3.95-107.65), G6PD deficiency (OR, 8.01; 95% CI:2.09-30.69), UGT1A1 polymorphisms (OR, 4.92; 95% CI:1.30-18.62), sepsis (OR, 9.15; 95% CI:2.78-30.10) placed infants at increased risk of severe hyperbilirubinemia or bilirubin induced neurologic dysfunctions.

In our study, the mean age of the neonates at admission was 70.3 hours ± 31.0 hours, the range being 3 to 280 hours (Table 9). In a study by Nepal D et al.[40] 34, 4.1 % neonates were admitted to hospital within 24 hours of birth, 27.4% were admitted within 24-72 hours of delivery, 54.8 % neonates were admitted to hospital at the age of 72 hours to 168 hours, and 13.7% were admitted after 168 hours (7 Days).

In our study, the birth weight of the neonates ranged from 2080 grams to 3720 grams, the mean being 2665.8 grams ± 268.6 grams(Table 9). A study by Sumangala Devi and Bindu[43] reported that low birth weight had a statistically significant relationship with hyperbilirubinemia.

In our study, the weight of the neonates at the time of admission ranged from 1980 grams to 3620 grams, with a mean of 2283 grams ± 259.2 grams (Table 9).

In our study, the duration of having icterus ranged from 4 -76 hours and mean duration was 10.4 hours ± 7.9 hours (Table 10). Sharma et al.[46] also reported that early onset of jaundice within 24 hours was significantly associated with adverse neurodevelopmental outcomes (DQ≤70) at 3 and 12 months of age.

The serum bilirubin level varies with birth weight, gestational age, chronological age and internal milieu of the body. Jamir et al [37] concluded that severe unconjugated

hyperbilirubinemia is potentially neurotoxic and conjugated hyperbilirubinemia is a harbinger of underlying severe illness.

In our study, the initial total bilirubin value of the study group ranged between 7.1 mg/dL to 27 mg/dL with a mean of 14.1 mg/dL ± 3.2 mg/dL. The initial value of unconjugated bilirubin ranged between 7.0 – 26.3 mg/dL with a mean value of 13.2 mg/dL ± 3.0 mg/dL of the study group. The BIND score ranged between 0 to 3, with a mean of 1 ± 0.7(Table 11). Arun Babu et al.[42] reported that a peak serum bilirubin level of ≥ 22mg/dl was an independent predictor of abnormal development in babies with neonatal jaundice.

The BIND score is used to grade the severity and progression of bilirubin induced neurological dysfunction among term and late-term neonates. Lower values indicate normalcy. Higher values indicate increasing severity. In our study, a BIND score of 0 was considered negative, while a BIND score of 1 or more was deemed to be positive. A major portion of neonates in the study group (52 %) had a BIND score of 0 and were considered negative. 37.6 % neonates had a BIND score of 1, 9.2 % had a BIND score of 2 and 1.2 % has a BIND score of 3 and were considered positive(Table 12 and Figure 12). So, the incidence of BIND was 48 % in our study.

In our study, in the neonate group which had positive BIND score, neonates whose mothers were multiparous predominated as compared to neonates whose mothers were primiparous (54.2% vs. 45.8%). In the neonates who had a negative BIND score, the number of neonates whose mothers were primiparous and multiparous were equal. The difference in the two groups with respect to parity was not statistically significant (p=0.579) (Table 13 and Figure 13). Our study is in line with a study by Astutik and Yuliawatib [47] in Indonesia who reported that there is no relationship

between the maternal parity (p = 0,084; POR = 0,204 95% CI 0,040-1,031) and the incidence of jaundice.

A systematic review and meta-analysis by Olusanya et al [45] reported that primiparity (OR, 1.59; 95% CI:1.26-2.00) had increased risk of BIND. Scrafford et al [48] reported primiparity to be a significant risk factor for neonatal jaundice after adjusting for multiple variables.

In our study, the late pre- term neonates had a mean Tb value slightly more than that of term neonates (14.4 mg/dl ± 3.2 mg/dl vs 14.0 mg/dl ± 3.2 mg/dl). However, this difference was not statistically significant (p=0.492). The mean Ucb levels were also more in the late pre-term neonates as compared to term neonates (13.5 mg/dl ± 3.1 mg/dl vs 13.1 mg/dl ± 3.0 mg/dl). However, this difference was not statistically significant (p=0.482) (Table 14 and Figure 14).

A study by Aboelreesh et al.[49] reported that preterm infants had a statistically significant lower mean TSB value as compared with term infants (18.5mg/dl ± 0.7mg/dl vs 22.8 mg/dl ± 3.1 mg/dl, p<0.001. The direct serum bilirubin values were also lower in the preterm group as compared to term group, but not significant (0.5 ± 0.3 vs 0.6 ±0.6 , p=0.23).

ABO incompatibility occurs in 'A' and 'B' blood group babies of O positive mothers. These babies are reported to be at high risk of severe hyperbilirubinemia. There was no significant difference between the BIND score positive and BIND score negative neonates with respect to their mothers having a particular blood group (p=0.564), in our study (Table 15 and Figure 15).

In our study, there was no significant difference between the BIND score positive and BIND score negative neonates with respect to having a particular blood group (p=0.372) (Table 16 and Figure 16).

A recent study by Khurana et al [50] reported that late pre-term and term neonates with and without ABO incompatibility had similar bilirubin levels and no increased risk of significant hyperbilirubinemia. In a Turkish study, Akgul et al [51] aimed to evaluate the effect of fetal-neonatal blood group on the severity of haemolysis and jaundice due to maternal-fetal ABO incompatibility. The study concluded that blood type has no effect on the severity of the hemolytic jaundice in ABO incompatibility. The result from our study is in line with these studies. A study by Kalakheti et al [52] reported that neonate with ABO incompatibility had two times higher chances of having hyperbilirubinemia than those babies with O '+ve' blood group.

There are conflicting views regarding the relation of mode of delivery and hyperbilirubinemia. Unnecessary interventions during delivery such as excessive use of oxytocin during labor, assisted deliveries and caesarean section are also considered as the risk factors [53,54.] In the present study, among the neonates who had a positive BIND score, 62.7 % were delivered by normal vaginal delivery and 37.3 % by caesarean section. Among the neonates who had a negative BIND score, 70.0 % were delivered by normal delivery and 30.0 % by LSCS. Though the percentage of neonates who were delivered by caesarean section were more in the BIND positive group as compared to BIND negative groups, this difference was not statistically significant (p=0.306) (Table 17 and Figure 17). Similar to our study, Boskabadi et al.[51] found no significant relationship between the mode of delivery and the incidence of jaundice. A Turkish study by Bilgin et al.[56] also did not observe a statistically

significant difference between 24 hours' and 48 hours' bilirubin levels of infants born vaginally or with caesarean section.

However, in an Indonesian study by Wijaya et al [57], caesarean section was associated with an increased risk of neonatal jaundice, which might be induced by maternal anaesthesia, especially bupivacaine. Ozdemirci et al.[58] also reported that compared with vaginal births, caesarean births led to higher rates of neonatal hyperbilirubinemia. According to a study by Tamook et al[59], the prevalence of jaundice was higher among neonates born by caesarean section, compared to those who were naturally delivered. An Indian study by Gupta et al.[60] also reported that on day 3, neonatal serum bilirubin is statistically significantly increased in neonates delivered by oxytocin-induced or Caesarean sections. In the case of vaginal delivery, neonates are stressed before birth which causes them to induce conjugative enzymes before vaginal delivery, thereby leading to lower levels of unconjugated bilirubin. Also, newborns delivered by caesarean section are breast-fed relatively infrequently during the first 48 hours of life than those born by vaginal delivery, possibly leading to hyperbilirubinemia.

An Iranian study by Yazdiha et al [61] indicated that caesarean delivery was not a risk factor for hospitalisation due to icterus during the first week after delivery. In some other studies, lower bilirubin levels after caesarean section are reported and are supposedly explained by placental transfusion or timing of cord clamping[56].

In contrast, Chang et al. [62] reported that bilirubin level was higher among naturally delivered neonates, compared to those born by caesarean section. An Iranian study by Garosi et al. [63] reported that the mean total bilirubin level was significantly higher in newborns delivered vaginally (17.3±3.5 mg/dl), compared to cases born by caesarean section (16.1±3.9 mg/dl) (P=0.02). Mala Kumar et al.[44] also reported that normal

vaginal delivery (odds ratio 5.5; 95% CI: 1.1–27.4, p = 0.037) was a significant risk factor for the development of ABE.

In the present study, among the neonate group whose BIND score was positive, 68.7 % neonates were term neonates while 31.3 % were late pre term neonates. In the BIND score negative group, 75.6 % neonates were term neonates while 24.4% neonates were late pre term neonates. Though the percentage of late pre term neonates in the positive BIND group was more as compared to BIND negative group, this difference in distribution was not statistically significant (p=0.313) (Table 18 and Figure 15).

However, a meta analysis by Olusanya[45] reported that low gestational age (OR, 1.71; 95% CI:1.40-2.11) was a risk factor for BIND. Sumangala Devi and Bindu[43] also reported that there was a statistically significant relationship between pre-term delivery, Pre-term premature rupture of the membranes (PPROM), and hyperbilirubinemia. A study by Aboelreesh et al.[49] reported that preterm neonates had a BIND score significantly higher than full term neonates (5.3 ± 1.8 vs 3.7 ± 1.96, p< 0.001). The study further revealed that 52% preterm neonates had advanced ABE based on the BIND score as compared to 12 % in the full term neonates, which was statistically significant (p<0.001). 36% of full term neonates had a mild ABE based on BIND score as compared to16% preterm infants who had mild ABE. 52% full term infants reported moderate ABE while 32% of preterm infants reported moderate ABE in the study.

Although oxytocin is widely accepted as a safe and effective initiator of uterine contractions, some studies have reported an association between oxytocic drugs and neonatal hyperbilirubinemia. Mechanisms that have been proposed to explain the

higher incidence of neonatal hyperbilirubinemia and oxytocin administration are trauma to the fetal erythrocytes as a result of uterine activation, vasoconstrictive effect of oxytocin on uterine blood vessels, alterations in erythrocyte deformability due to the anti-diuretic activity of oxytocin and hyponatremia caused by the administration of large quantities of electrolyte-free diluents for oxytocin infusion [64].

In our study, oxytocin was used to induce labour in 28.9 % cases (n=50). Of these, 54 % of neonates had a BIND Score of 0, 40 % had a bind score of 1 and 4% had a BIND Score of 2 and 2% of neonates had a BIND Score of 3.Among the neonates whose mothers did not need oxytocin to induce labour, 51.2 % had a BIND score of 0,36.6% neonates had a BIND Score of 1, 11.4% had a BIND score of 2 and 0.8% neonates had a BIND Score of 3 (Table 19 and Figure 16). There was no significant relationship between the use/non-use of oxytocin and BIND score (p=0.422). Similar to our study, Taneja S et al [64] also found no correlation of maternal oxytocin infusion and hyperbilirubinemia in neonates. In contrast, Garosi et al [63] reported that the mean total and direct bilirubin levels were higher in neonates delivered using oxytocin (0.4±.1 and 17.99±0.4, respectively), compared to those without oxytocin induction (0.383±0.1 and 16.2±0.28, respectively) (P=0.001). In a study in Baroda, India, Patel et al.[41] reported that use of oxytocin during vaginal delivery for labor induction or reinforcement may influence the positive relationship between the severity of jaundice and oxytocin use.

Among the neonates who had a positive BIND score, 56.6% were males and 43.4 % were females. In the BIND score negative group, 51.1 % were males and 48.9% were females. This distribution of sex among the two groups was not statistically significant (p=0.467) (Table 20and Figure 17). In contrast to our study, an Iranian study by Garosi et al [63] reported that the mean total and direct bilirubin levels were

higher among female newborns (0.397 ± 0.013 and 17.2 ± 0.29, respectively), compared to the male neonates (0.379 ±0.22 and 15.9 ± 0.37, respectively) (P=0.005 and P=0.02, respectively).

In our study, in the Rh+ve group, 62.5% neonates had a BIND score of 0, 25.0% of neonates had a BIND score of 1, 12.5 % of neonates had a BIND score of 2, and no neonate had a BIND score of 3. In the Rh –ve group, 51.5 % of neonates had a BIND score of 0, 38.2 % of neonates had a BIND score of 1, 9.1 % of neonates had a BIND score of 2, and 1.2 % of neonates had a BIND score of 3(Table 21 and Figure 18). There was no significant relationship between the Rh +ve/ Rh –ve group and BIND score (p=0.868). A study by Patel et al.[41] reported that in Rh incompatibility group, 10% new-born developed kernicterus.

In our study, in the ABO +ve group, 47.4% neonates had a BIND score of 0, and an equal number of neonates had a BIND score of 1, 5.3 % of neonates had a BIND score of 2, and no neonate had a BIND score of 3. In the ABO –ve group, 53.3 % of neonates had a BIND score of 0, 34.8 % of neonates had a BIND score of 1, 10.4 % of neonates had a BIND score of 2, and 1.5 % of neonates had a BIND score of 3(Table 22 and Figure 19). There was no significant relationship between the ABO +ve and ABO –ve group and BIND score (p=0.416). Akgul et al.[51] also reported that blood type has no effect on the severity of the hemolytic jaundice in ABO incompatibility. However, a study by Patel et al.[41] reported that in ABO incompatible group, 0.5 % new-born developed kernicterus.

The mean age of neonates who had a positive BIND score was 71.23 hours ± 26.42 hours, while that of neonates having a negative BIND score was 69.39 hours ± 34.84

hours. The difference between the two groups was **not statistically significant** (p=0.698) (Table 23 and Figure 20.1).

Neonates who had a positive BIND score had a mean birth weight of 2663.87 grams ± 290.39 grams and neonates having negative BIND score had mean birth weight of 2667.51 grams ± 248.55 grams. The difference between the two groups was **not statistically significant** (p=0.929) (Table 23 and Figure 20.2). Gamaleldin et al.[24] reported that low admission weight (OR: 0.83 per 100 g) increased the risk for bilirubin encephalopathy, especially when other risk factors were present. In an Indian study, Mala Kumar [44] et al reported that lower weight on admission (2254.68 g ± 417 g vs 2481.75 g ± 369 g; p = 0.0195), found to be a significant risk factor for the development of ABE. Jamir et al [37] reported that there was no significant difference in the mean serum bilirubin values between neonates of different birth weights and maturity.

The mean initial total bilirubin value of the neonate group who had a positive BIND score was more than that of the group who had a negative BIND score (14.31mg/dL ± 3.91 mg/dL vs. 13.89 mg/dL ± 2.37 mg/dL). However, this difference was **not statistically significant** (p=0.399) (Table 24 and Figure 21).

In a study in Cairo by Gamaleldin [24] et al., the admission TSB values ranged from 25 to 76.4 mg/dL. Forty-four neonates had moderate or severe ABE at admission; 35 of 249 infants (14%) had evidence of BE at the time of discharge or death. TSB levels correlated poorly with the presence or absence of ABE or BE in these patients. The threshold TSB level that identified 90% of infants with ABE/BE was 25.4 mg/dL when neurotoxicity risk factors were present. In contrast, neurotoxicity was first observed at a TSB level of >31.5 mg/dL in 111 infants without risk factors. The study concluded that neonates without risk factors for neurotoxicity had a higher tolerance

for hyperbilirubinemia than recognised in management guidelines. The great variation in response to TSB indicated that biological factors other than TSB values were important in the pathogenesis of BE. Based on their meta-analysis, Olusanya [45] et al. reported that high total serum bilirubin levels (OR, 1.46; 95% CI:1.10-1.92) were a risk factor for severe hyperbilirubinemia or BIND. In a study in Egypt, El Houchi et al.[66] reported that all infants with severe acute bilirubin encephalopathy (BIND scores 7-9) either died or suffered residual neurologic and auditory impairment. Of 24 cases with moderate encephalopathy (BIND 4-6), 15 (62.5%) resolved following aggressive intervention and were normal at follow-up. Three of 73 infants with mild encephalopathy (BIND scores 1-3) but severe jaundice (TSB ranging 33.5-38 mg/dL; 573-650 μmol/L) had residual neurologic and/or auditory impairment. A BIND score ≥4 had a specificity of 87.3% and a sensitivity of 97.4% for predicting poor neurologic outcomes (receiver operating characteristic analysis). BIND scores trended higher with severe hyperbilirubinemia (r^2 = 0.54, P < .005), but 5/39 (13%) infants with TSB ≥36.5 mg/dL (624 μmol/L) had BIND scores ≤3, and normal outcomes at 3-5 months. The study concluded that BIND score could be used to evaluate the severity of acute bilirubin encephalopathy and predict residual neurologic and hearing dysfunction[66].

Similarly, the initial unconjugated bilirubin value was higher in the neonates who had a positive BIND score as compared to the neonates who had negative BIND score (13.38 mg/dL ± 3.70 mg/dL vs. 13.09 mg/dL ± 2.31 mg/dL). However, this difference **was not statistically significant** (p=0.538) (Table 23 and Figure 23).

CONCLUSION

Neonatal hyperbilirubinemia is very common neonatal problem in day to day practice. Most neonates associated with high risk factors present with significantly elevated bilirubin levels posing a risk of Bilirubin Induced Neurological Dysfunction. The Incidence of bilirubin induced neurological dysfunction (subtle) was 48% in our study. Early detection of BIND by incorporating BIND scoring system will identify sick neonates with bilirubin toxicity prompting early intervention in the form of Intensive Phototherapy along with fluid therapy thereby preventing further complication of bilirubin toxicity. Corroboration of this scoring system could offer a much-needed practice to determine the actual extent of neonatal bilirubin encephalopathy related morbidity and mortality and thereby reduce or eliminate this preventable morbidity which has long-term, tragic consequences for the neonates, their families and their communities.

RECOMMENDATION

The amalgamation of the BIND scoring criteria in the neonatal checklist at the time of admission would help in categorizing the severity of BIND and early interventions for those presenting with manifestations of BIND by eliminating this preventable morbidity which has long-term, tragic consequences for the neonates, their families and their communities.

SUMMARY

This prospective hospital based observational study was conducted during November 2018 – May 2020. During this study period 173 cases of hyperbilirubinemic neonates were included in the study. In case of significant hyperbilirubinemia BIND scoring was noted and AABR was performed.

In our study we made the following observations:

- The incidence of BIND was 48 % in our study.
- Neonates who had multiparous mothers outnumbered neonates who had primiparous mothers (52 % vs 48 %).
- 55.5% neonates had mothers whose blood group was O positive (most common), 16.8% neonates had mothers whose blood group was A positive, which is second most common.
- 32.4 % neonates had A positive blood group (most common), 29.5 % had B positive blood group which is second most common.
- 66.5 % neonates were delivered by normal vaginal delivery and 33.5% neonates by Caesarean section.
- Majority of the neonates in our study (72.8 %) had birth weight more than 2.5 kg.
- 53.8 % neonates were males, and 46.2 % were females. The male: female ratio was found to be 1.16: 1.
- Among the maternal risk factors for hyperbilirubinemia, primiparity posed a major risk (43.9 %), followed by the use of oxytocin (28.9 %) and ABO incompatibility (22.0 %).

- Among the perinatal risk factors, 16.8 % neonates presented with sepsis and 2.3 % had birth trauma.
- Low intake of breast milk was a major neonatal risk factor (64.7%), followed by male sex (48%) and low birth weight (8.7%). Besides, 12.7 % infants had previous siblings who had received phototherapy.
- The mean age of the neonates at admission was 70.3 hours ± 31.0 hours, the range being 3 to 280 hours.
- The birth weight of the neonates ranged from 2080 grams to 3720 grams, the mean being 2665.8 grams ± 268.6 grams.
- The weight of the neonates at the time of admission ranged from 1980 grams to 3620 grams, with a mean of 2183.1 grams ± 259.2 grams.
- The history of duration of jaundice ranged from 4 -76 hours and mean duration was 10.4 hours ± 7.9 hours.
- The initial total bilirubin value of the study group ranged between 7.1 mg/dL to 27 mg/dL with a mean of 14.1 mg/dL ± 3.2 mg/dL. The initial value of unconjugated bilirubin ranged between 7.0 – 26.3 mg/dL with a mean value of 13.2 mg/dL ± 3.0 mg/dL of the study group.
- The BIND score ranged between 0 to 3, with a mean of 1.1 ± 0.7.
- In the positive BIND score group, neonates whose mothers were multiparous predominated as compared to neonates whose mothers were primiparous (54.2% vs. 45.8%). In the neonates who had a negative BIND score, the number of neonates whose mothers were primiparous and multiparous were equal. The difference in the two groups with respect to parity was not statistically significant (p=0.579).

- The late pre- term neonates had a mean Tb value slightly more than that of term neonates (14.4 mg/dl ± 3.2 mg/dl vs 14.0 mg/dl ± 3.2 mg/dl). However, this difference was not statistically significant (p=0.492). The mean Ucb levels were also more in the late pre-term neonates as compared to term neonates (13.5 mg/dl ± 3.1 mg/dl vs 13.1 mg/dl ± 3.0 mg/dl). However, this difference was not statistically significant (p=0.482).
- There was no significant difference between the BIND score positive and BIND score negative neonates with respect to their mothers having a particular blood group (p=0.564).
- There was no significant difference between the BIND score positive and BIND score negative neonates with respect to having a particular blood group (p=0.372).
- Among the neonates who had a positive BIND score, 62.7 % were delivered by normal vaginal delivery and 37.3 % by caesarean section. Among the neonates who had a negative BIND score, 70.0 % were delivered by normal delivery and 30.0 % by LSCS. Though the percentage of neonates who were delivered by caesarean section were more in the BIND positive group as compared to BIND negative groups, this difference was not statistically significant (p=0.306).
- Among the neonate group whose BIND score was positive, 68.7 % neonates were term neonates while 31.3 % were late pre term neonates. In the BIND score negative group, 75.6 % neonates were term neonates while 24.4% neonates were late pre term neonates. Though the percentage of late pre term neonates in the positive BIND group was more as compared to BIND

negative group, this difference in distribution was not statistically significant (p=0.313).

- Oxytocin was used to induce labour in 28.9 % cases (n=50). Of these, 54 % of neonates had a BIND Score of 0, 40 % had a bind score of 1 and 4% had a BIND Score of 2 and 2% of neonates had a BIND Score of 3.Among the neonates whose mothers did not need oxytocin to induce labour, 51.2 % had a BIND score of 0,36.6% neonates had a BIND Score of 1, 11.4% had a BIND score of 2 and 0.8% neonates had a BIND Score of 3. There was no significant relationship between the use/non-use of oxytocin and BIND score (p=0.422).
- Among the neonates who had a positive BIND score, 56.6% were males and 43.4 % were females. In the BIND score negative group, 51.1 % were males and 48.9% were females. This distribution of sex among the two groups was not statistically significant (p=0.467).
- In the ABO group, 47.4% neonates had a BIND score of 0, and an equal number of neonates had a BIND score of 1, 5.3 % of neonates had a BIND score of 2, and no neonate had a BIND score of 3. In the ABO –ve group, 53.3 % of neonates had a BIND score of 0, 34.8 % of neonates had a BIND score of 1, 10.4 % of neonates had a BIND score of 2, and 1.5 % of neonates had a BIND score of 3(Table 22 and Figure 19). There was no significant relationship between the ABO +ve and ABO -ve group and BIND score (p=0.416).
- The mean age of neonates who had a positive BIND score was 71.23 hours ± 26.42 hours, while that of neonates having a negative BIND score was 69.39

hours ± 34.84 hours. The difference between the two groups was not statistically significant (p=0.698).

- Neonates who had a positive BIND score had a mean birth weight of 2663.87 grams ± 290.39 grams and neonates having negative BIND score had mean birth weight of 2667.51 grams ± 248.55 grams. The difference between the two groups was not statistically significant (p=0.929).
- The mean initial total bilirubin value of the neonate group who had a positive BIND score was more than that of the group who had a negative BIND score (14.31mg/dL ± 3.91 mg/dL vs. 13.89 mg/dL ± 2.37 mg/dL). However, this difference was not statistically significant (p=0.399).
- The initial unconjugated bilirubin value was higher in the neonates who had a positive BIND score as compared to the neonates who had negative BIND score (13.38 mg/dL ± 3.70 mg/dL vs. 13.09 mg/dL ± 2.31 mg/dL). However, this difference was not statistically significant (p=0.538).
- All BIND positive babies passed AABR test. There was no auditory involvement.

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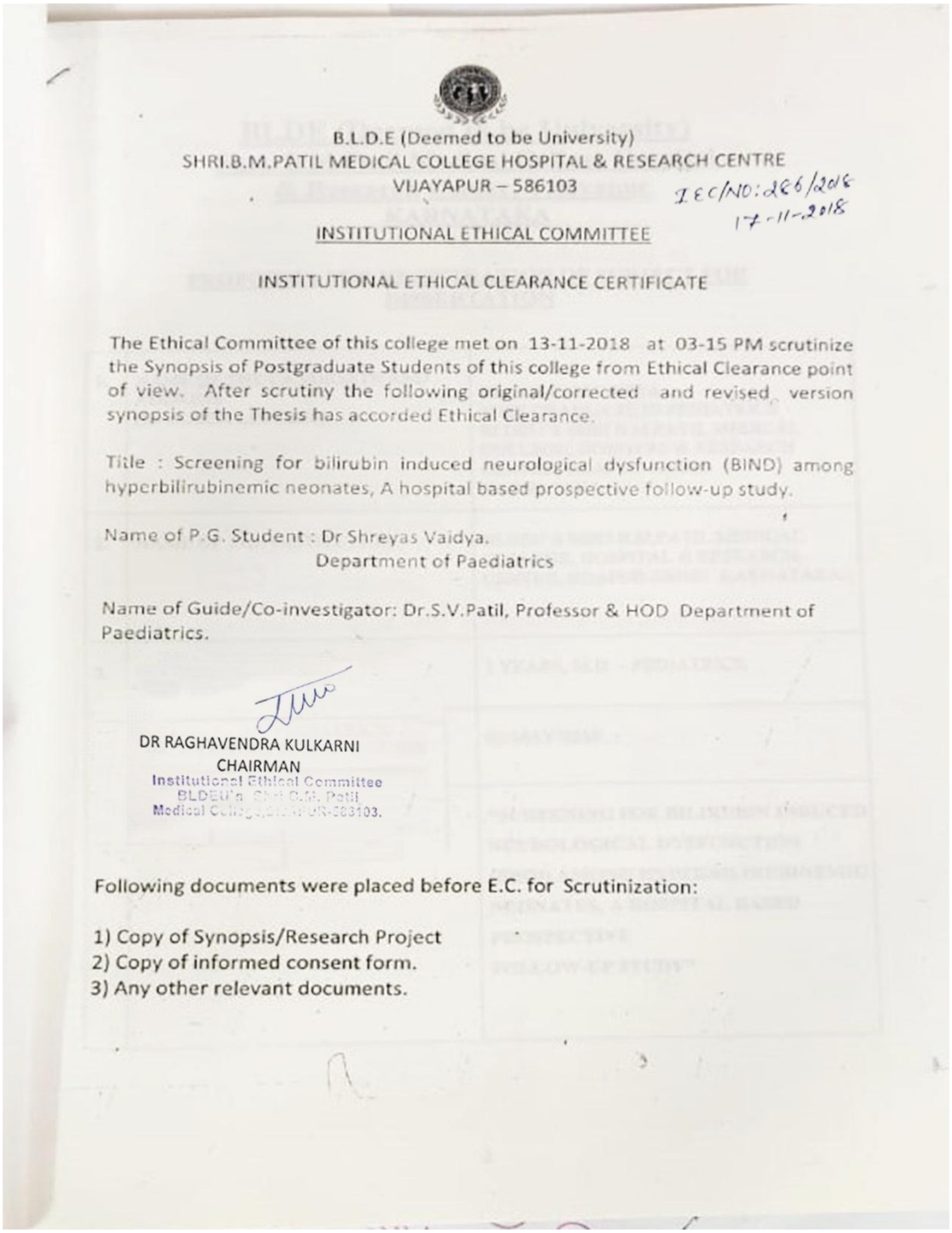
ANNEXURES

ETHICAL CLEARANCE CETIFICATE



B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO:286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : Screening for bilirubin induced neurological dysfunction (BIND) among hyperbilirubinemic neonates, A hospital based prospective follow-up study.

Name of P.G. Student : Dr Shreyas Vaidya.
Department of Paediatrics

Name of Guide/Co-investigator: Dr.S.V.Patil, Professor & HOD Department of Paediatrics.

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College, VIJAYAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

CONSENT FORM

BLDEA's Shri B.M. PATIL Medical College, Hospital & Research Centre, Bijapur-586103.

TITLE OF THE PROJECT	**:**	**SCREENING FOR BILIRUBIN INDUCED NEUROLOGICAL DYSFUNCTION (BIND) AMONG HYPERBILIRUBINEMIC NEONATES, A HOSPITAL BASED PROSPECTIVE STUDY**
GUIDE	**:**	**DR. S.V PATIL, MD** PROFESSOR DEPARTMENT OF PEDIATRICS
PG STUDENT	**:**	**Dr. SHREYAS VAIDYA** PG DEPARTMENT OF PEDIATRICS (MD PEDIATRICS)

PURPOSE OF RESEARCH:

I have been informed that this study will help in screening for bilirubin induced neurological dysfunction among hyperbilirubinemic neonates.

PROCEDURE:

I am aware that in addition to routine care received, I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations which will help the investigator in this study

BENEFITS:

I understand that participation in the study will help the investigator to help in the early detection of neonatal hyperbilirubinemia and bilirubin induced neurological dysfunction.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time; Dr Shreyas Vaidya at the department of paediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr Shreyas Vaidya may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to the baby resulting directly from participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the baby. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr Shreyas Vaidya Date

(Investigator)

PROFORMA

Name –

IP no –

DOB - Age in days – Birth weight - Admission Weight –

Sex –

Address –

Obstetric score –

Mother's blood group - Baby's Blood group –

APGAR

1min –

5min –

High risk Factors –

Maternal –

Primiparity / Teenage Pregnancy / Diabetes / Rhesus incompatibility / ABO incompatibility / Use of oxytocin.

Perinatal –

Mode of delivery –
Birth trauma / Birth Asphyxia / Delayed cord clamping / Congenital Infections –

CMV/Syphilis / Sepsis

Neonatal –

Male sex / Prematurity / LBW / SGA

Polycythaemia / hypoglycaemia / low intake of breast milk / dehydration / weight loss

Others –

Previous sibling received phototherapy or exchange transfusion.

Birth outside of the health care.

H/o duration of jaundice – (in hours)

GENERAL PHYSICAL EXAMINATION:

Birth weight…….gms

HR: RR: HC:

CFT: TEMP: LENGTH:

SYSTEMIC EXAMINATION:

CVS:

RESPIRATORY SYSTEM:

GASTRO – INTESTINAL SYSTEM:

CNS:

BIND scoring –

CLINICAL SIGNS	BIND SCORE	ACUTE BILIRUBIN ENCEPHALOPHATHY
MENTAL STATUS		
Normal.	0	None
Sleepy, could be awaken	1	Subtle ABE
Lethargy and poor suck.	2	Moderate ABE
Semicoma, apnea and seizures.	3	Severe ABE
MUSLE TONE		
Normal	0	None
Mild to moderate hypotonic	1	Subtle ABE
Hypertonia arching of the back	2	Moderate ABE
Retrocolis,, ophisthotonus	3	Severe ABE
CRY PATTERNS		
Normal	0	None
High pitched cry	1	Subtle ABE
Shrill difficult to console	2	Moderate ABE
Weak or absent cry	3	Severe ABE

AABR – PASS/FAIL

KEY TO MASTER CHART

BW – Birth Weight

AW – Admission weight

OS – Obstetric Score

LPT – Late Preterm

MBG – Mother's blood group

BBG – Babies blood group

HDOJ – History of duration of jaundice

HC – Head circumference

TB – Total bilirubin

UCB – Unconjugated bilirubin

BIND – Bilirubin induced neurological dysfunction

MASTER CHART

S.NO	AGE (IN HOURS)	BW (IN GRAMS)	AW (IN GRAMS)	SEX	OS	Term/LPT	MBG	BBG	APGAR at 1MIN	APGAR at 5MIN
1	70	2620	2405	Male	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	7	9
2	9	3420	3450	Male	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
3	49	2650	2540	Male	Primi	LPT	A POSITIVE (A+)	A POSITIVE (A+)	7	9
4	95	3200	3210	Female	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
5	70	3120	2980	Female	Multi	term	O NEGATIVE (O-)	O POSITIVE (O+)	7	9
6	115	3145	3042	Female	Multi	term	AB POSITIVE (AB+)	B POSITIVE (B+)	8	9
7	120	3720	3460	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
8	20	2611	2510	Female	Primi	lpt	O NEGATIVE (O-)	A POSITIVE (A+)	8	9
9	45	3250	3160	Male	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
10	70	2414	2320	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
11	72	2414	2320	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
12	74	2568	2320	Female	Multi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
13	78	2568	2500	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
14	78	2580	2500	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
15	38	2568	2500	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
16	76	2610	2585	Female	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
17	76	2680	2520	Female	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
18	100	2312	2270	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
19	78	2472	2410	Female	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
20	98	2620	2468	Female	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
21	76	2620	2468	Female	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
22	78	2680	2520	Female	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
23	80	2680	2520	Male	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
24	70	2561	2480	Male	Multi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
25	69	2560	2480	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
26	70	2480	2360	Male	Multi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
27	41	2380	2300	Male	Multi	LPT	A POSITIVE (A+)	AB POSITIVE (AB+)	8	9
28	89	2560	2500	Female	Primi	LPT	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
29	45	2380	2300	Female	Primi	LPT	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
30	69	2340	2200	Male	Primi	LPT	B POSITIVE (B+)	B POSITIVE (B+)	7	9
31	46	2480	2360	Male	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
32	46	2480	2360	Female	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
33	70	2850	2760	Female	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	7	9
34	70	2560	2500	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
35	68	2480	2400	Female	Multi	term	AB POSITIVE (AB+)	B POSITIVE (B+)	7	9
36	70	2780	2600	Female	Multi	term	B NEGATIVE (B-)	AB POSITIVE (AB+)	7	9
37	48	2480	2400	Male	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
38	46	2780	2620	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
39	26	2568	2480	Female	Primi	term	O NEGATIVE (O-)	A POSITIVE (A+)	8	9
40	49	2600	2580	Male	Multi	term	B POSITIVE (B+)	AB POSITIVE (AB+)	7	9
41	48	2940	2700	Female	Primi	term	A POSITIVE (A+)	O POSITIVE (O+)	7	9
42	76	2480	2360	Male	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
43	50	2480	2280	Female	Primi	LPT	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
44	51	2680	2511	Male	Primi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
45	59	2568	2428	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9

46	54	2480	2300	Male	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
47	80	2680	2580	Male	Primi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
48	99	2876	2346	Male	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
49	76	3120	2854	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
50	75	2420	2380	Female	Primi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
51	22	2680	2520	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
52	78	2680	2490	Female	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
53	50	2688	2428	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
54	46	2564	2510	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
55	140	2280	2102	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	8
56	76	2380	2108	Female	Multi	LPT	O NEGATIVE (O-)	O NEGATIVE (O-)	8	8
57	69	2340	2260	Male	Multi	LPT	O POSITIVE (O+)	O NEGATIVE (O-)	8	8
58	50	2480	2380	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
59	70	2640	2610	Female	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
60	56	2680	2520	Male	Primi	term	AB POSITIVE (AB+)	A POSITIVE (A+)	8	9
61	88	2580	2470	Male	Primi	term	AB POSITIVE (AB+)	A POSITIVE (A+)	8	9
62	50	2320	2162	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	9	8
63	77	2460	2360	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
64	32	3180	3180	Male	Primi	term	A POSITIVE (A+)	AB POSITIVE (AB+)	8	9
65	76	2640	2530	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
66	68	2568	2430	Female	Primi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
67	76	2650	2590	Female	Primi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
68	74	2600	2580	Female	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
69	75	2600	2520	Male	Primi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	7	9
70	70	2700	2589	Male	Primi	term	AB POSITIVE (AB+)	B POSITIVE (B+)	7	9
71	68	2640	2390	Male	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
72	94	2480	2260	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	7	9
73	122	2780	2698	Male	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	7	9
74	70	2390	2260	Male	Multi	term	B NEGATIVE (B-)	B NEGATIVE (B-)	7	9
75	68	2649	2540	Male	Multi	term	B POSITIVE (B+)	O POSITIVE (O+)	7	9
76	70	2480	2360	Male	Primi	LPT	B POSITIVE (B+)	O POSITIVE (O+)	8	9
77	68	2900	2760	Female	Primi	term	AB POSITIVE (AB+)	B POSITIVE (B+)	8	9
78	46	2840	2700	Male	Multi	term	AB POSITIVE (AB+)	A POSITIVE (A+)	7	9
79	51	2416	2380	Male	Primi	term	O NEGATIVE (O-)	A POSITIVE (A+)	8	9
80	70	2368	2230	Male	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
81	49	2380	2220	Male	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
82	70	2672	2614	Male	Primi	term	O NEGATIVE (O-)	B POSITIVE (B+)	8	9
83	74	2710	2666	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
84	68	2414	2374	Female	Multi	LPT	A POSITIVE (A+)	O POSITIVE (O+)	8	9
85	94	2683	2520	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
86	68	2628	2538	Male	Multi	LPT	A POSITIVE (A+)	O POSITIVE (O+)	8	9
87	47	2610	2580	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
88	50	2568	2432	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
89	69	2680	2468	Male	Multi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
90	50	2440	2410	Male	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
91	70	2568	2420	Female	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
92	18	2614	2614	Male	Multi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
93	45	2080	1980	Male	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
94	46	2800	2650	Male	Primi	term	O POSITIVE (O+)	A POSITIVE	7	9

								(A+)		
95	71	3000	2800	Female	Multi	term	B POSITIVE (B+)	O POSITIVE (O+)	7	9
96	110	3000	2600	Female	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
97	25	2600	2550	Female	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
98	76	2800	2400	Male	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	7	9
99	49	2800	2500	Female	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	7	9
100	76	3000	2500	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
101	78	2600	2500	Female	Primi	LPT	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
102	78	3620	3580	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	8	9
103	86	3620	3548	Male	Multi	term	B POSITIVE (B+)	O POSITIVE (O+)	8	9
104	77	2380	2330	Male	Multi	LPT	A POSITIVE (A+)	AB POSITIVE (AB+)	8	9
105	77	2380	2220	Male	Primi	LPT	A POSITIVE (A+)	O POSITIVE (O+)	8	9
106	77	2380	2200	Female	Primi	LPT	A POSITIVE (A+)	O POSITIVE (O+)	8	9
107	86	2568	2482	Male	Primi	LPT	O POSITIVE (O+)	A NEGATIVE (A-)	8	9
108	78	2568	2482	Female	Primi	LPT	O POSITIVE (O+)	A NEGATIVE (A-)	8	9
109	76	2418	2350	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
110	50	2418	2350	Female	Primi	term	O POSITIVE (O+)	A NEGATIVE (A-)	8	9
111	76	2568	2420	Female	Multi	term	AB POSITIVE (AB+)	A POSITIVE (A+)	8	9
112	50	2568	2420	Male	Multi	term	AB POSITIVE (AB+)	A POSITIVE (A+)	8	9
113	78	2618	2584	Female	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
114	78	2560	2420	Female	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
115	77	2560	2420	Male	Primi	term	O POSITIVE (O+)	AB POSITIVE (AB+)	8	9
116	121	2468	2320	Male	Multi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
117	50	2468	2320	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
118	50	2568	2462	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
119	50	2564	2480	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
120	50	2610	2522	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
121	76	2680	2520	Male	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
122	26	2311	2300	Male	Multi	term	O NEGATIVE (O-)	B POSITIVE (B+)	8	9
123	70	2568	2468	Male	Multi	term	A POSITIVE (A+)	B POSITIVE (B+)	8	9
124	40	2620	2280	Female	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
125	40	2568	2500	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
126	45	2480	2368	Female	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
127	108	2280	2200	Male	Primi	LPT	O POSITIVE (O+)	A POSITIVE (A+)	8	9
128	90	3020	2980	Male	Primi	term	B POSITIVE (B+)	O POSITIVE (O+)	8	9
129	50	2480	2420	Female	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
130	70	2640	2580	Male	Multi	LPT	AB POSITIVE (AB+)	B POSITIVE (B+)	7	9
131	70	2650	2480	Female	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
132	70	2340	2150	Male	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
133	100	2680	2550	Female	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
134	108	2840	2650	Male	Primi	LPT	B NEGATIVE (B-)	B POSITIVE (B+)	7	9
135	90	2780	2540	Male	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	7	9
136	107	2650	2500	Female	Primi	LPT	B POSITIVE (B+)	B POSITIVE (B+)	7	9
137	109	2860	2750	Female	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
138	70	2780	2640	Female	Multi	LPT	B POSITIVE (B+)	B POSITIVE (B+)	7	9
139	68	2650	2500	Male	Primi	term	B POSITIVE (B+)	AB POSITIVE (AB+)	7	9
140	46	3200	2700	Male	Multi	term	B POSITIVE (B+)	A POSITIVE (A+)	7	9
141	118	3500	3100	Male	Primi	term	O POSITIVE (O+)	O POSITIVE (O+)	7	9
142	94	2800	2300	Female	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9

143	70	2368	2280	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
144	107	2620	2580	Male	Multi	term	A POSITIVE (A+)	AB POSITIVE (AB+)	8	9
145	140	2560	2430	Male	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
146	46	2850	2560	Female	Primi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
147	143	2800	2600	Female	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	7	9
148	144	3240	3100	Female	Multi	term	B POSITIVE (B+)	O POSITIVE (O+)	7	9
149	28	2655	2550	Male	Multi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
150	49	2326	2200	Female	Primi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	8	9
151	50	2568	4490	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
152	80	2568	2360	Male	Multi	term	O POSITIVE (O+)	B POSITIVE (B+)	8	9
153	60	2160	2080	Female	Primi	LPT	O POSITIVE (O+)	O POSITIVE (O+)	8	9
154	29	2600	2542	Male	Multi	term	A NEGATIVE (A-)	O POSITIVE (O+)	8	9
155	280	3028	2800	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
156	80	2680	2880	Male	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9
157	23	2960	2720	Female	Primi	LPT	O POSITIVE (O+)	O POSITIVE (O+)	7	9
158	94	2800	2680	Male	Primi	LPT	A POSITIVE (A+)	AB POSITIVE (AB+)	8	9
159	23	2580	2480	Male	Primi	term	O POSITIVE (O+)	O NEGATIVE (O-)	8	9
160	48	2580	2480	Male	Primi	term	O POSITIVE (O+)	O NEGATIVE (O-)	8	9
161	70	3000	2860	Male	Multi	term	B POSITIVE (B+)	O POSITIVE (O+)	8	9
162	47	3200	2920	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	9	9
163	50	2860	2720	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
164	46	2720	2620	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	8	9
165	116	3020	2980	Male	Multi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
166	3	2720	2628	Female	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	8	9
167	80	2480	2360	Female	Multi	term	A POSITIVE (A+)	O POSITIVE (O+)	8	9
168	8	2860	2680	Female	Primi	term	O POSITIVE (O+)	O POSITIVE (O+)	8	9
169	126	3000	2890	Female	Multi	term	A POSITIVE (A+)	A POSITIVE (A+)	7	9
170	80	2800	2470	Female	Primi	term	O POSITIVE (O+)	B POSITIVE (B+)	7	9
171	144	2800	2680	Male	Multi	LPT	O POSITIVE (O+)	B POSITIVE (B+)	7	9
172	75	3000	2860	Male	Primi	term	B POSITIVE (B+)	B POSITIVE (B+)	7	9
173	90	2628	2520	Female	Primi	term	O POSITIVE (O+)	A POSITIVE (A+)	8	9

S.NO	MATERNAL RISK FACTORS	MODE OF DELIVERY	PERINATAL RISK FACTORS	NEONATAL RISK FACTORS	OTHER RISK FACTORS
1		LSCS		MALE SEX	
2	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
3	PRIMIPARITY	LSCS		MALE SEX, PREMATURITY	
4		LSCS			PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
5	DCT	LSCS			PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
6		LSCS			
7		LSCS		MALE SEX	
8	PRIMIPARITY, RHESUS INCOMPATIBILITY	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	
9	PRIMIPARITY, ABO INCOMPATIBILITY	LSCS		MALE SEX	
10	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LBW, LOW INTAKE OF BREAST MILK	
11	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
12		LSCS		LOW INTAKE OF BREAST MILK	
13	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
14	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
15	USE OF OXYTOCIN	NORMAL VAGINAL		LOW INTAKE OF BREAST	

		DELIVERY		MILK	
16	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
17	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
18	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
19	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
20	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
21	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	
22	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	
23	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
24	ABO INCOMPATIBILITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
25	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
26	ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
27	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
28	PRIMIPARITY, ABO INCOMPATIBILITY	LSCS			
29	PRIMIPARITY, ABO INCOMPATIBILITY	LSCS			
30	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX	
31	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
32	DIABETES	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
33	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY			
34	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX	
35	HYPOTHYROIDISM	NORMAL VAGINAL DELIVERY			
36	HYPOTHYROIDISM	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
37		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
38		LSCS			
39	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
40		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
41	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
42	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
43	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
44	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX	
45	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY			
46	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
47	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
48	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK, WEIGHT LOSS	BIRTH OUTSIDE HEALTHCARE
49	ABO INCOMPATIBILITY	LSCS			
50	PRIMIPARITY	NORMAL VAGINAL DELIVERY			
51		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
52	PRIMIPARITY, RHESUS INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
53	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
54	ABO INCOMPATIBILITY	LSCS	SEPSIS	MALE SEX	
55	ABO INCOMPATIBILITY	LSCS	SEPSIS	MALE SEX, LBW	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
56		LSCS	SEPSIS	LBW, RDS	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
57		LSCS	SEPSIS)	LBW, RDS	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION

58	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
59	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX	
60	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX	
61	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX	
62	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
63	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
64	PRIMIPARITY	LSCS		MALE SEX	
65	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY			PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
66	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	
67	PRIMIPARITY	NORMAL VAGINAL DELIVERY			
68	PRIMIPARITY	NORMAL VAGINAL DELIVERY			
69	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX	
70	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
71		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK, DEHYDRATION	
72	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
73	PRIMIPARITY, ABO INCOMPATIBILITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
74		LSCS		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
75		NORMAL VAGINAL DELIVERY		MALE SEX	
76	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
77	PRIMIPARITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
78		LSCS		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
79	PRIMIPARITY, RHESUS INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
80	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
81	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	LSCS	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
82	PRIMIPARITY, RHESUS INCOMPATIBILITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	LSCS	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
83	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
84		NORMAL VAGINAL DELIVERY	BIRTH TRAUMA	LOW INTAKE OF BREAST MILK	
85	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		HYPOGLYCEMIA, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
86		NORMAL VAGINAL DELIVERY	BIRTH TRAUMA	MALE SEX, LOW INTAKE OF BREAST MILK	
87		LSCS		LOW INTAKE OF BREAST MILK	
88		LSCS		LOW INTAKE OF BREAST MILK	
89	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
90	PRIMIPARITY	NORMAL VAGINAL DELIVERY	BIRTH TRAUMA	LOW INTAKE OF BREAST MILK	
91	PRIMIPARITY	NORMAL VAGINAL DELIVERY	BIRTH TRAUMA	LOW INTAKE OF BREAST MILK	
92	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, HYPOGLYCEMIA, LOW INTAKE OF BREAST MILK, DEHYDRATION	
93		NORMAL VAGINAL DELIVERY		LBW, SGA, LOW INTAKE OF BREAST MILK	
94	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
95		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
96		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
97		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
98	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	

99	ABO INCOMPATIBILITY	LSCS		LOW INTAKE OF BREAST MILK	
100	PRIMIPARITY	LSCS		MALE SEX	
101	PRIMIPARITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
102	PRIMIPARITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
103		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
104	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
105	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
106	PRIMIPARITY	LSCS		LBW, LOW INTAKE OF BREAST MILK	
107	PRIMIPARITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
108	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
109	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
110	ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
111		LSCS		LOW INTAKE OF BREAST MILK	
112	RHESUS INCOMPATIBILITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
113		LSCS	SEPSIS	LOW INTAKE OF BREAST MILK	
114	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
115	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
116	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LBW	
117	PRIMIPARITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
118	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
119	PRIMIPARITY, ABO INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
120	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
121	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
122	RHESUS INCOMPATIBILITY, USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LBW, LOW INTAKE OF BREAST MILK	
123	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
124	USE OF OXYTOCIN	LSCS		LOW INTAKE OF BREAST MILK	
125	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
126	ABO INCOMPATIBILITY	LSCS	SEPSIS	LBW, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
127	PRIMIPARITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
128	PRIMIPARITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
129	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY	SEPSIS	LBW, LOW INTAKE OF BREAST MILK	
130		LSCS		MALE SEX	
131		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
132		NORMAL VAGINAL DELIVERY		MALE SEX, LBW, SGA, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
133		NORMAL VAGINAL DELIVERY		LOW INTAKE OF BREAST MILK	
134	PRIMIPARITY, RHESUS INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX	
135	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
136	PRIMIPARITY	NORMAL VAGINAL DELIVERY			
137		NORMAL VAGINAL DELIVERY			
138		LSCS		LOW INTAKE OF BREAST MILK, DEHYDRATION	
139	PRIMIPARITY	LSCS		MALE SEX	
140		LSCS		DEHYDRATION	
141	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX	
142		NORMAL VAGINAL DELIVERY			BIRTH OUTSIDE HEALTHCARE
143	PRIMIPARITY	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
144		NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
145		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
146	PRIMIPARITY	LSCS			

147		NORMAL VAGINAL DELIVERY			
148		NORMAL VAGINAL DELIVERY			
149		NORMAL VAGINAL DELIVERY			
150	PRIMIPARITY	LSCS		LBW, LOW INTAKE OF BREAST MILK	
151	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	LOW INTAKE OF BREAST MILK	
152	USE OF OXYTOCIN	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
153	PRIMIPARITY	NORMAL VAGINAL DELIVERY		LBW, LOW INTAKE OF BREAST MILK	
154	RHESUS INCOMPATIBILITY	LSCS		MALE SEX	PREVIOUS SIBLING RECEIVED PHOTOTHERAPY OR EXCHANGE TRANSFUSION
155		LSCS		LOW INTAKE OF BREAST MILK	
156	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
157	PRIMIPARITY	LSCS		LOW INTAKE OF BREAST MILK	
158	PRIMIPARITY	NORMAL VAGINAL DELIVERY		MALE SEX, LOW INTAKE OF BREAST MILK	
159	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
160	PRIMIPARITY	NORMAL VAGINAL DELIVERY	SEPSIS	MALE SEX, LOW INTAKE OF BREAST MILK	
161		LSCS		MALE SEX, DEHYDRATION	
162	PRIMIPARITY	LSCS		MALE SEX, LOW INTAKE OF BREAST MILK, DEHYDRATION	
163		LSCS		LOW INTAKE OF BREAST MILK, DEHYDRATION	
164	PRIMIPARITY	LSCS		MALE SEX	
165		LSCS		MALE SEX, LOW INTAKE OF BREAST MILK	
166	PRIMIPARITY	NORMAL VAGINAL DELIVERY			
167		LSCS			
168	PRIMIPARITY	LSCS			
169		NORMAL VAGINAL DELIVERY			
170	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY			
171	ABO INCOMPATIBILITY	LSCS			
172	PRIMIPARITY	LSCS		MALE SEX	
173	PRIMIPARITY, ABO INCOMPATIBILITY	NORMAL VAGINAL DELIVERY		PREMATURITY	

S.NO	DHOJ	HC (IN CM)	LENGTH (IN CM)	TB	UCB	BIND SCORE
1	5	35	49	13.6	12.2	3
2	8	35.2	48.2	12.4	9.7	1
3	4	36	48.6	6.5	6	2
4	8	35.2	48.6	16	15.6	2
5	10	34.2	50.1	13.6	13.1	2
6	8	35.2	47.8	12.9	11.9	2
7	12	34.5	49.2	13.5	12.4	1
8	8	36.3	50.3	12	11.6	0
9	8	34.2	48.6	10.8	10.6	0
10	10	36.5	50.1	10.6	10	1
11	6	36.5	50.1	14.7	13.7	0
12	10	37.6	50	12.1	11.6	1
13	12	34.6	48.9	14.7	14	1
14	6	35.2	48.5	9.4	9	1
15	12	34.6	48.9	12.5	12	1
16	6	34.2	49.2	15.7	15	0
17	6	34.2	49.2	10.6	10	0
18	6	36.2	46.2	12	11.4	0
19	6	36.2	48.8	8.1	7.5	1
20	6	36.2	48.6	15.5	15	1
21	6	36.2	48.6	12.1	11.6	1
22	12	35.2	50.2	15.5	15	1
23	8	35.2	50.2	15	14.6	1
24	6	34.2	49.3	13	12.2	0
25	12	34.2	49.2	12.9	12	0
26	6	34.2	50.3	12.7	12.3	1
27	10	34.6	48.6	14.4	14	1
28	6	35.2	48.2	13.5	12.9	1
29	10	35.2	48.2	12.7	12.1	1
30	10	32	49.5	12.3	12	0
31	12	34.6	48.2	12.5	12	0
32	12	34.6	48.2	13.7	13	0
33	6	33	49	14.9	14	1
34	8	33	49	13.9	13.6	0
35	8	33	49	14	13.5	0
36	8	33	49	13.7	13	1
37	8	35.6	48.6	9.9	9.5	1
38	10	34.8	49.2	10.3	9.8	0

39	10	34.6	50.2	7.4	7	0
40	5	32.4	50	11.8	11	0
41	6	35	50	10.6	10	0
42	6	36.3	50.3	17	16	0
43	10	36.3	48.8	20.6	19.6	1
44	6	34.2	48.6	18.9	16.6	1
45	12	35.2	48.6	15.2	13	0
46	12	34.2	48.6	11.7	10.7	1
47	6	34.8	48.6	12.9	12.4	0
48	48	33	51	14.7	13.7	0
49	6	34.2	52.2	14.3	13.5	0
50	12	34.5	50.2	15.2	14.6	1
51	8	35.2	48.6	20.3	19	0
52	10	35.2	48.6	14	13.1	0
53	6	34.8	48.6	14.4	13.3	0
54	12	38.2	51.3	13.6	13	1
55	12	38.2	51.3	18.1	15.1	2
56	6	36.3	48.3	13.8	13	0
57	6	36.3	48.3	15.6	15.2	0
58	12	36.3	50	10.4	9.8	1
59	12	36.3	50	16.6	16	1
60	7	38.3	50.1	10.2	9.7	0
61	8	38.3	50.1	16.3	15.5	0
62	8	35.8	50	11.9	10	0
63	9	35.8	50	12.6	11.5	0
64	6	36	51	16.7	15.2	0
65	10	34.5	49.2	15.2	14.7	0
66	12	34.6	48.6	14.7	14.2	0
67	8	34	49	13.6	13	0
68	8	33.4	49.5	13	12.6	0
69	8	34	49.5	13.9	13	0
70	12	33	49	14.8	14	1
71	10	33.6	49	27	26.3	2
72	8	33	49.3	16.9	16	0
73	10	33.5	50	24.1	23.6	1
74	14	34	50	15	14.5	0
75	10	32.5	49	13.7	13	0
76	10	33	51	16.1	15.8	0
77	8	33.6	48.5	14	13.5	0
78	7	34	49	14.2	13.7	0
79	12	34.6	51.2	2.4	2	0
80	12	34.6	51.2	11.4	11	0
81	6	35.2	39.6	10	9.5	1
82	6	35.2	39.6	2.4	2	1
83	12	35.2	48.8	17.9	17	2
84	10	36.2	48.6	16	15.5	1
85	12	35.2	48.8	15.7	14.5	2
86	10	36.2	48.6	12.3	11.9	1
87	6	36.8	48.6	11.4	11	1
88	6	36.8	48.6	16.1	13.8	1
89	10	36.8	51.2	18.1	17.5	1
90	6	36.3	51.2	13.3	12.8	1
91	6	36.3	51.2	11.5	11	1
92	10	36.8	51.2	26.5	23.5	3
93	6	34.2	48.6	7.9	7.3	1
94	6	33	48	19	18	1
95	24	33	48	21.8	21.2	1
96	24	33	43	13.4	10.2	0
97	8	35	44	13.4	13	0
98	24	33	50	16.8	16.1	0
99	48	33	46	16.8	16.2	0
100	76	33	48	13.2	12.8	0
101	10	35	48	14.8	14.3	0
102	10	35	48	15.2	14.7	1
103	12	36	51	16.2	15.6	0
104	6	36	50	12.2	11	1
105	10	35	51	14.5	14	0
106	10	35	48	11.9	10.5	0
107	6	36	51	12.7	12	1
108	6	36	51	14.9	9.7	0
109	12	36	51	18.6	18	0
110	6	36	50	12.3	11.8	1
111	10	36	51	14.7	14	1
112	10	36	50	16.9	16	2
113	10	36	51	16.6	16.2	2
114	12	36	50	16.1	8	1
115	12	36	51	16.1	15	1
116	12	36	51	14.3	13.8	1
117	6	36	49	14.3	13.8	1
118	6	36	51	13.6	12.8	0
119	6	36	50	11	10	0
120	10	36	51	13.6	13	0
121	10	36	49	14.1	13.6	0
122	6	35	50	2.2	1.6	0
123	6	36.2	47.8	14.6	14.2	0
124	6	37.2	48.6	11.8	11	1
125	10	37.8	48.6	8.8	8.3	1
126	10	37.2	51.3	16.5	16	0
127	8	38.2	46.3	14.2	13.8	1
128	12	35	50	16	15.3	1
129	10	35.2	48.6	18.8	18.6	0
130	10	33	49	16.9	16	0
131	8	32.5	49	13.6	13	0
132	10	33	50	17.5	17	1

133	8	33	51	14.2	13.6	0
134	8	33	51	17	16.5	1
135	10	34	49.5	14.6	14	1
136	10	33	49	13.3	12.8	1
137	10	33	50	10	9.5	0
138	8	33	51	19.4	17.1	2
139	8	33	50	14.6	14	0
140	24	32	50	12.2	11.7	1
141	8	35	50	16	15.6	0
142	24	33	47	13.4	12	0
143	6	36	50	14.5	13	1
144	10	36	48.6	16	15.4	2
145	8	33	49.6	9.1	8.1	2
146	5	34	48	8.5	7.8	2
147	40	33	48	11.7	10.7	0
148	24	33	48	19	15	1
149	6	35.2	44.4	7.1	6.1	1
150	8	34.2	48.6	13.1	12.5	0
151	10	34.2	48.2	17.3	15.5	0
152	6	34.2	48.6	12.5	12	0
153	6	34.2	46.8	8.5	8	1
154	6	34.2	48.2	14.2	13.8	0
155	10	34.2	48.6	12.6	12	0
156	6	36.2	50	12.9	11.9	1
157	12	34.2	48.6	16.3	15	0
158	12	34.8	48.6	16.2	15.6	0
159	10	34.6	48.6	15.3	14.7	0
160	10	34.6	48.6	15.1	14.3	0
161	8	37	49	12.6	10.8	1
162	10	35.6	50	15	13.8	1
163	8	35.2	51.2	14.5	13.5	0
164	10	36	53	15.6	14	2
165	12	35.6	51	11.5	10	0
166	6	34.5	47.2	12.6	11.8	0
167	8	35.6	50	16.2	15.8	0
168	10	36.2	50	16.8	15.2	0
169	12	33	46	10.8	10.4	0
170	24	33	47	13.4	13	0
171	12	33	46	14.1	13.7	0
172	12	33	48	16.2	13.2	0
173	12	36.2	50.6	19.8	18.5	2