"CORD BLOOD BILIRUBIN AS AN EARLY PREDICTOR OF NEONATAL HYPERBILIRUBINEMIA"

By

DR. SOWMYA .R. MBBS

Dissertation submitted to

Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka

In partial fulfillment of the requirements for the degree of

M.D
IN
PAEDIATRICS

Under the guidance of
Dr.BEERE GOWDA .Y.C, M.D.
Professor & HOD Of Paediatrics

DEPARTMENT OF PAEDIATRICS
SRI DEVARAJ URS MEDICAL COLLEGE

KOLAR-563101

APRIL 2013

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Date :
Place : Kolar

Signature of the candidate
Dr. **SOWMYA.R**

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Date :
Place : Kolar

Signature of the Guide
DR.BEEREGOWDA.Y. C, MD,
Professor and HOD
Department of Paediatrics
Sri Devaraj Urs Medical College
Tamaka , Kolar.

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH , TAMAKA , KOLAR , KARNATAKA,

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Date :
Place : Kolar

Signature of the Co-Guide
Dr. N.Krishnamurthy, MD
Professor and HOD
Department of Biochemistry,
Sri Devaraj Urs Medical College
Tamaka , Kolar.

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Dr. BEEREGOWDA.Y.C M.D
Professor & HOD
Department of Paediatrics
SDUMC, Kolar.

Dr.M.B.Sanikop
Principal
SDUMC, Kolar.

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Signature of Member Secretary

ACKNOWLEDGEMENT

It is with great privilege, I express my most humble and profound gratitude to my Teacher and Guide **Dr. Beergowda.Y.C.,** Professor & H.O.D, Department of Pediatrics, Sri Devaraj Urs Academy Of Higher Education & Research Centre, for his guidance able supervision, valuable suggestions and constant encouragement throughout the study which facilitated the completion of this dissertation. He has made me realize that there is no substitution for hard work.

I express my gratitude to my eminent teacher and principal, **Dr. M.B.SaniKop,** Sri Devaraj Urs Medical College and Research Centre for permitting me to carry out this study, his constant support and encouragement.

I am thankful to **Dr. N.Krishnamurthy,** Professor & H.O.D, Department of Biochemistry, Sri Devaraj Urs Medical College and Research Centre for his guidance, able supervision, valuable suggestions and constant encouragement throughout the study which facilitated the completion of this dissertation.

I am thankful to **Dr. M.NarayanSwamy** Professor & H.O.D, Department of OBG, Sri Devaraj Urs Medical College and Research Centre, the **Faculty** and Staff of OBG Department for their constant support to complete my study.

I am extremely grateful to all my distinguished **Professors, Associate Professors, Assistant Professors, Senior Residents and Junior Residents** of the department of Pediatrics for their kind co-operation and guidance in conducting my study.

I thank my fellow **Post graduates** and **friends** for their support and cooperation.

I thank **Dr.K.P.Suresh,** Statistician, for providing me the statistical guidance.

I am extremely grateful to the technical staff for their valuable assistance during the study period.

Most important of all I shall be failing in my duties if I do not thank all the **MOTHERS AND BABIES** included in the study, without whom this study would not have been possible.

I express my deepest sense of gratitude to my parents **Mr & Mrs RaviKumar.Y.R. and Dr. Naveen.D.,Asst Prof, Dept of Orthopaedics**, for their encouragement, unabated inspiration and moral support during my career as a postgraduate.

I certainly owe my thanks to all the people who have helped me directly or indirectly in accomplishing this work.

Finally I thank god, for making all these wonderful people happen to me and pray for continued blessing and success.

Date:
Place:

Signature of the candidate
Dr. Sowmya.R.

ABBREVATIONS

AAP	:	American Academy of Pediatrics
AGA	:	Appropriate for Gestational Age
BEAR	:	Brain Evoked Auditory Response
BNR	:	Band Neutrophil Ratio
CB	:	Conjugated Bilirubin
CPD	:	Citrate Phosphate Dextrose
CS	:	Caesarean Section
DCT	:	Direct Coomb Test
DIC	:	Disseminated Intra Vascular Coagulation
ETCOc	:	End Tidal Carbon Monoxide
G6PD	:	Glucose-6-Phosphate Dehydrogenase
ICT	:	Indirect Coomb Test
P-gp	:	P-glycoprotein
PRBC	:	Packed Red Blood Cell
RBC	:	Red Blood Cell
STB	:	Serum Total Bilirubin
TcB	:	Trans Cutaneous Bilirubin
TSB	:	Total Serum Bilirubin
UCB	:	Unconjugated Bilirubin
UDPG-T	:	Uridine Di Phosphate Glucuronyl Transferase
VD	:	Vaginal Delivery

ABSTRACT

BACKGROUND

Neonatal hyperbilirubinemia (NH) is a cause of concern for the parents as well as for the pediatricians. Early discharge of healthy term newborns after delivery has become a common practice because of medical and social reasons as well as economic constraints. In significant number of babies, hyperbilirubinemia is the most common cause for readmission during the early neonatal period. Severe jaundice and even kernicterus can occur in some full term healthy newborns discharged early with no apparent early findings of haemolysis.

OBJECTIVE

To predict the risk of neonatal hyperbilirubinemia using cord blood bilirubin, in order to implement early treatment and there by minimize the risk of bilirubin dependent brain damage.

METHODS

This study was performed at the Department of Pediatrics of Sri Devaraj Urs Medical College & Research Hospital. Eligible healthy full-term newborns, 205 in number born at this hospital during 1-year period was prospectively enrolled in the study. cord blood was sent for bilirubin estimation at the time of birth. Serum Bilirubin estimation was done on day 3 of life.

RESULTS

Among 205 healthy term neonate who were enrolled into the study over the 1 year period, 4.39% were found to have significant jaundice (day 3 TSB >14mg/dl). Using CBB level of ≥2mg/dl as a cut off, neonatal hyperbilirubinemia can be predicted with sensitivity of 90%, specificity of 98.9%, positive predictive value of 81% and negative predictive value of 99.48%.

CONCLUSION

In the present study infants with neonatal hyperbilirubinemia (day 3 TSB >14mg/dl) had significantly higher levels of cord bilirubin (≥2mg/dl). So it can be concluded that cord blood bilirubin estimation is a non invasive and reliable investigation for early prediction of neonatal hyperbilirubinemia. So babies with cord blood bilirubin ≥2mg/dl defines, the risk group prone to develop significant hyperbilirubinemia . It also predicts dicision making regarding delay in discharge and frequent follow up for initial postnatal days and implementation of early treatment to minimize the risk of bilirubin dependent brain damage.

KEYWORDS:

Cord blood bilirubin, neonatal hyperbilirubinemia, Total serum bilirubin

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INTRODUCTION

Neonatal hyperbilirubinemia remains a public health concern for the parents as well as for the pediatricians as documented by recent reports of kernicterus in otherwise healthy term and near- term newborns. Kernicterus in such newborns is preventable, provided excessive hyperbilirubinemia for age is promptly identified and appropriately treated.[1,2]

With the intent to facilitate such identification and treatment, universal screening for severity of bilirubinemia before hospital discharge may predict that extraordinary segment of the neonatal population which is at risk for excessive hyperbilirubinemia during the first week after birth.[3]

Neonatal hyperbilirubinemia affects nearly 60% of term and 80% of preterm neonates during first week of life.[4] Early discharge of healthy term newborns after delivery has become a common practice because of medical and social reasons as well as economic constraints.[5,6] In significant number (6.5%) of babies, hyperbilirubinemia is the most common cause for readmission during the early neonatal period.[7] Up to 4% of term newborns who are readmitted to the hospital during their first week of life, approximately 85% are readmitted for jaundice.[8]

Severe jaundice and even kernicterus can occur in some full term healthy newborns discharged early with no apparent early findings of haemolysis.[9] It is difficult to predict which newborns are at increased risk for developing significant hyperbilirubinemia (Total Serum Bilirubin ≥15mg/dl).[10]

Thus, the recognition, follow-up, and early treatment of jaundice has become

more difficult as a result of earlier discharge from the hospital. The American Academy of Pediatrics (AAP) recommends that newborns discharged within 48 hours should have a follow-up visit after 2-3 days to detect significant jaundice and other problems.[11] This recommendation is not possible in our country due to limited follow up facilities in the community.

The potential risk of developing bilirubin encephalopathy or even kernicterus is high in babies with elevated serum biliruhin level. The sequalae could be serious as patients may develop athetosis, athetoid cerebral palsy, partial or complete high frequency sensorineural hearing loss, paralysis of upward gaze, dental dysplasia and intellectual deficits.[4,8] The treatment of severe neonatal jaundice by exchange transfusion is costly, associated with complications, time consuming and requires skilled manpower.[1]Early treatment of jaundice with phototherapy is effective, simple and cheap.[12,13]

The concept of prediction of jaundice offers an attractive option to pick up babies at risk of NH. A Total Serum Bilirubin level of >15 mg/dl is found in 3% of normal term babies.[14] The incidence of hyperbilirubinemia depends on regional variations, ethnic makeup of the population,[15,16] laboratory variability in the measurement of bilirubin, and the incidence of breastfeeding.[17] Predischarge hour specific bilirubin estimation, transcutaneous bilirubin measurement and ETCO measurement are some of the investigations done to predict the subsequent course of jaundice in newborn babies. [14]

There is an obvious need to develop simple predictive guidelines that will enable the physicians to predict or to identify which of the early discharged newborns will develop significant hyperbilirubinemia, and there by minimize the

risk of bilirubin dependent brain damage. The present study was conducted to evaluate the predictive value of cord bilirubin level for identifying term infants for subsequent hyperbilirubinemia.

OBJECTIVES

1. To predict the risk of neonatal hyper bilirubinemia using cord blood bilirubin, in order to implement early treatment and there by minimize the risk of bilirubin dependent brain damage.

REVIEW OF LITERATURE

HISTORY REVIEW

Hippocrates (460-370 BC) "Father of Medicine", made frequent references to jaundice as a serious disease. Greek Medicine was based on four humors – phlegm, yellow bile, blood and black bile.

Jaundice is a well known clinical entity in the Indian Medicine (Ayurveda).

Since the Vedic Era (1500 BC – 800 BC) this disease has been described. This has been mentioned among diseases in Atharvaveda. Ayurveda is based on "Tridosha theory of disease" – Vata (wind), Pitta (gall) and Kapha (mucus). Charaka Samhita (200AD) described one of the first references to skin icterus. Jaundice (kamale) is a specific condition, which arises due to aggravation of bile.[18]

Word "bile" is derived from latin bilis ("bile"). Word 'Bilirubin' and 'Biliverdin'-eans "red bile" and "green bile" Latinized. Icterus from Greek iketros, meaning "yellow colored", a word applied to a yellow bird as well. Word 'Jaundice'-from Old French jaundice, a word rooted in the Latin galbinus, meaning "greenish yellow", from galbus ("yellow").[19]

The first reference to jaundice in newborns is from a book published in the mid 15th century by Mettlinger, Germany entitled "Ein Regiment der jungen kinder" [Aurburg – 1473].[20]

In 1654, Panaroli reported apparent case of hemolytic disease of the newborn.[21] Erythroblastosis fetalis may well have been described in 1609 in France, a report by a midwife named Bourgeois described an hydropic infant girl died 15 min after birth with severe jaundice of the placenta and blood.[22]

Neonatal jaundice must have been noticed by caregivers through the centuries, but the scientific description and study of this phenomenon seem to have started in the last half of the 18th century.

In 1785 Jean Baptiste Thimote´e Baumes was awarded a prize from the University of Paris for his work describing the clinical course in 10 jaundiced infants.[23]

The first case was Baumes' own daughter, Justine. He believed that delayed meconium passage was a primary cause of neonatal jaundice, and espoused breast milk, particularly colostrum, from the infant's own mother as the best remedy for this problem. His thoughts on the reabsorption of bile from the duodenum seem to be confirmed by our present knowledge about the enterohepatic circulation of bilirubin.[23]

The work by Jaques Hervieux, which he defended for his doctor of medicine degree in 1847, was, in many respects, a landmark. Having dismissed most of the theories and work of his predecessors.[3] He had autopsied 44 jaundiced infants and apparently had clinical observations on many others. His descriptions of pathoanatomical findings were very detailed and systematic. A number of his clinical observations are still thought to be accurate today. [23]

His Clinical and Epidemiological Observations on Neonatal Jaundice are

1. The cause of neonatal jaundice is not known, but one can state that jaundice in the neonate is a manifestation of a recently established function that for a limited time exceeds its physiological limits.
2. Neonatal jaundice is a physiological condition.
3. Neonatal jaundice is, by itself, never fatal
4. Neonatal jaundice appears during the first 2 to 4 days of life and lasts for 1 to 2 weeks. It never reappears in the following months.

5. There is a cephalocaudal progression in the appearance of jaundice the extremities are always last to be affected. When jaundice disappears, the order is reversed.
6. Neonatal jaundice is very common approximately two thirds of all infants are affected. The prognosis in the absence of complicating conditions is benign.
7. Jaundice is not seen in foundlings who are wet-nursed, or in infants nursed by a woman who gave birth a long time ago.
8. The most frequent complicating conditions are scleredema, diarrhea, and thrush.
9. Treatment consists of combating the complicating conditions. Isolated neonatal jaundice does not need treatment.
10. In neonatal jaundice, the yellow colour is found throughout the tissues of the body, including the brain. Hervieux described brain jaundice in 31 of 44 cases of neonatal jaundice. In all of these cases, clinical jaundice had been at its peak at the time of death. He described the intensity of the brain jaundice as variable. Some brains were quite uniformly stained, while in other brains some regions were more heavily stained than others. It is noteworthy that he found the cerebrospinal fluid to be jaundiced in all cases. [23]

In 1847 Virchow isolated bilirubin crystals from hematomas and suggested that bilirubin was derived from blood.13 The relationship between the clinical encephalopathy associated with elevated serum bilirubin concentration and gross pathological changes seen as yellow staining of specific areas of the CNS was observed In 1847, Virchow suggested that the excessive destruction of red blood cells in the first week of life is the basic cause of jaundice.[24]

Orth an assistant to Virchow, in his article, which primarily focused on pigment crystals in various organs, he described a term female infant who was born nonicteric, but who became jaundiced soon after birth. The child died at 2 days of age with very pronounced jaundice, which was apparently her only sign/symptom. At autopsy all organs were found to be jaundiced, but with an underlying pallor that may perhaps point to the existence of anaemia. The brain was intensely yellow, but with much more intense staining of the basal ganglia, the wall of the third and fourth ventricles, the hippocampus, and the central parts of the cerebellum. On microscopic examination of the latter, the granular layer was found to be heavily stained. Orth also noted that although neurons of the basal ganglia were stained, the glial elements were not. [23]

Molisan demonstrated by transfusion experiments that erythrocytes of new born, breaks down twice as rapidly as compared to adults.[24]

In 1903, Schmorl coined the term 'Kernikterus' and described the pathology of the jaundice in the brain. He described his findings from the autopsies of 280 neonates, of whom 120 were jaundiced at the time of death. In the majority of these cases (114/120), he found the brain to be diffusely yellow. He noted that the intensity of the brain colour paralleled that of the face, which is often the most intensely jaundiced part of an infant's body, as also described by Hervieux. [23]

In the brains examined, the jaundiced nuclei were very sharply demarcated and, therefore, contrasted clearly with the colour of the surrounding tissue. Because of this sharp demarcation and the predilection for staining of the nuclei, Schmorl proposed the term kernicterus. He further suggested that the yellow colour was not simply attributable to saturation of the tissue with bile pigments, such as was the case (he believed) with eg, skin, but to binding of the bile pigments to

specific structural elements in the tissue. Microscopic examination of the tissue supported this hypothesis. Some neurons in the nuclei were strongly coloured, while others had a more pale yellow colour. These latter cells exhibited changes that suggested that they were in the process of dying. [23]

The first use of the term "Erythroblastosis fetalis" was by Rautmann in 1912 in reference to an hydropic still born. [25]

Halban in 1900 suggested that isoimmunization of the mother could be basis of erythroblastosis. [26]

Ottenberg in 1923 proposed that feto-maternal transfusion was etiologically responsible. [27] later Levine and Colleagues in 1941, demonstrated the role of Rh antibodies in the etiology of erythroblastosis fetalis. [28]

In 1907 Beneke, was the first to suggest that septicaemia might play an important role in icterus gravis neonatorum, and he theorized that the pigmentation of brain tissue was caused by a peculiar attraction of bile pigments to ganglion cells leading to their necrosis, damage to the ganglion cells by the bile salts which then became pigmented, or ischemic or the traumatic insult that allowed the cells to become pigmented.[19]

In 1913 Yippo, published a paper, "zeitschrifit der kinderheilunde", in which he described the yellowish discolouration of the newborn and umbilical cords. He also proposed a theory that functional immaturity of liver prevented the excretion of all bilirubin that is formed, permitting some to re-enter the circulating blood.[29]

As early as 1915, there were description of children who survived severe neonatal jaundice with resultant mental retardation and neuromuscular

dysfunction, with the jaundice being considered the causal agent (Guthrie, 1913; Spiller, 1915).[19]

Dutch Biochemists, Van Den Bergh and Muller in 1913, observed that serum from patients with haemolytic jaundice can be differentiated from the serum of patients with obstructive jaundice on the basis of chemical reactions. They observed that haemolytic serum did not react promptly with diazotised sulfanilic acid except in presence of alcohol while the other serum reacted in an aqueous solution.[19]

Diamond and colleagues in 1932, recognized that generalized edema of the fetus (hydrops fetalis), icterus gravis and congenital anemia of the newborn were all in fact a part of single condition which they termed "isoimmunization fetalis".[29]

In 1939, Landsteiner and Weiner, Levine and Stetson demonstrated the serological basis of maternal fetal blood group incompatibility and the identification of the Rh system of antigens.[30]

The first exchange transfusion in a newborn was performed in 1925 by Hart for treatment of erythroblastosis fetalis.[31] and in 1946, Wallerstein reported the successful exchange transfusion of three infants with erythroblastosis fetalis. [32]

Halbreent in 1944, coined the term "Icterus precox" for jaundice developed within 24 hours of birth. Allen et al in 1950 showed effectiveness of exchange transfusion as a protection from kernicterus.[33]

Crigler and Najjar in his publication made in 1952, described congenital familial non haemolytic jaundice with kernicterus and explained kernicterus as a process related more to elevated unconjugated bilirubin levels than to specific blood group incompatibilities or even hemolysis.[19]

Cremer and associates from Rochform hospital in Essex, published their report in 1958 regarding successful use of phototherapy for the treatment of neonatal jaundice. The initiator of phototherapy was a staff nurse of the above hospital, who noticed that babies whose uncovered parts were less yellow when compared to covered parts of body on exposure to sunlight.[34]

Bilirubin metabolism

Jaundice is the commonest abnormal physical finding during first week of life.

Between 25 to 50% of all term newborns and a higher percentage of premature newborns develop clinical jaundice.[14]

Sources of bilirubin

Bilirubin is derived from the breakdown of heme containing protein in the reticuloendothelial system.[14]

a. The major heme containing protein is red blood cell hemoglobin. This is the source of 75% of all bilirubin production.

b. The other 25% of bilirubin is called early labeled bilirubin. It is derived from hemoglobin released by ineffective erythropoiesis in the bone marrow, from otherheme containing proteins in tissues (ex: myoglobin, cytochromes, catalase, peroxidase) and from free heme.

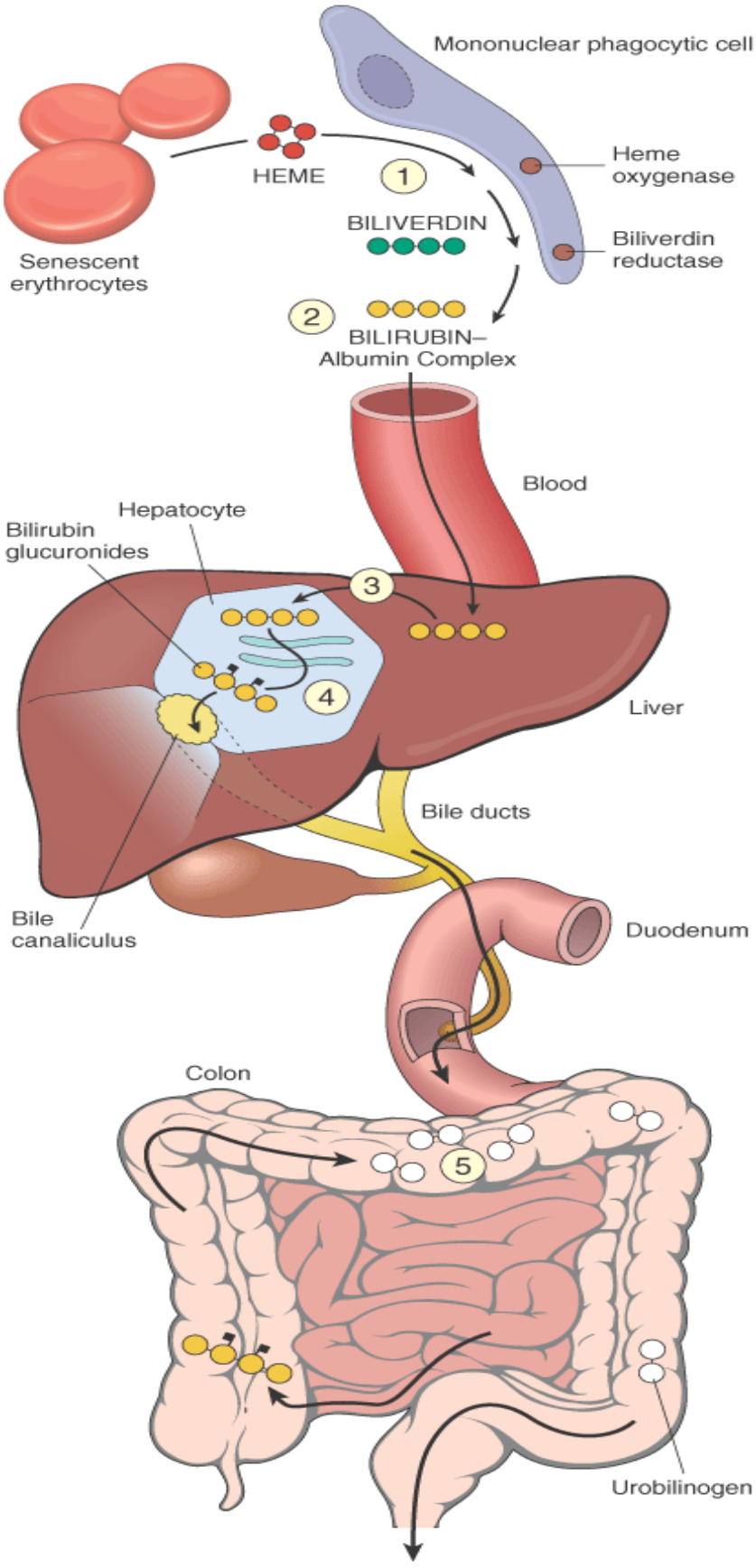


Fig. 1 : Bilirubin metabolism[35]

Bilirubin Metabolism[14]

The conversion of heme to bilirubin requires two closely linked enzymatic steps.

Bilirubin synthesis

1st step is conversion of heme to a linear tetrapyrrolebiliverdin ,1 molecule of ferrous ion and 1 mol of carbonmonoxide is released by enzyme Hemeoxygenase. It is the rate limiting step and upregulated during hemolysis.[36]

2nd step of bilirubin synthesis involves Biliverdinreductase found in cystosol of most cells. Biliverdin is converted to Bilirubin.[36]

Bilirubin transport in the plasma

Bilirubin in plasma is tightly bound to serum albumin, usually does not enter the central nervous system and is thought to be nontoxic.[14]

Bilirubin uptake

Non polar, fat, soluble bilirubin (dissociated from albumin) crosses the hepatocyte plasma membrane and is bound mainly to cytoplasmic ligandin (Y protein) for transport to the smooth endoplasmic reticulum. Phenobarbital increases the concentration of ligandin.[14]

Bilirubin conjugation

Unconjugated bilirubin (UCB) is converted to water soluble conjugated (direct) bilirubin (CB) in the smooth endoplasmic reticulum by uridinediphosphate glucuronyltransferase (UDPG-T). This enzyme is inducible by phenobarbital and catalyzes the formation of bilirubin monoglucuronide. Both mono and diglucuronide forms of conjugated bilirubin are able to be excreted into the bile canaliculi against a concentration gradient.[14]

Bilirubin excretion

Conjugated bilirubin in the biliary tree enters the gastrointestinal tract and is thus eliminated from the body in the stool, which contains large amount of bilirubin.

Excretion is considered to be the rate limiting step of overall bilirubin clearance from the plasma.[14]

Enterohepatic circulation of bilirubin

Conjugated bilirubin is not normally reabsorbed from the bowel unless it is converted back to unconjugated bilirubin by the intestinal enzyme β-glucuronidase. Intestinal bacteria can prevent the enterohepatic circulation by converting the conjugated bilirubin to urobilinoids, which are not substrates of β-glucuronidase.[14]

Fetal bilirubin metabolism

Most unconjugated bilirubin formed by the fetus is cleared by the placenta into the maternal circulation. Formation of conjugated bilirubin is limited in the fetus because of decreased fetal hepatic blood flow, decreased hepatic ligandin and decreased UDPG-T activity. The small amount of conjugated bilirubin excreted is usually hydrolyzed by β-glucuronidase and reabsorbed.[14]

Bilirubin is normally found in the amniotic fluid by 12 weeks gestation and is usually one by 37 weeks gestation. Increased amniotic fluid bilirubin is found in hemolytic disease of the newborn and in fetal intestinal obstruction below the bile ducts.[14]

Etiology of hyperbilirubinemia in newborn

Any process that increases the production or impairs the elimination of bilirubin can exacerbate the normally occurring physiologic jaundice in newborn.[14]

Etiology

I. Physiologic jaundice[14]

a. Increased bilirubin production due to-

Increased RBC volume per kilogram and decreased RBC survival (90 days versus 120 days) in infants.

- Increased ineffective erythropoiesis and increased turnover of non hemoglobin heme proteins.

b. Increased enterohepatic circulation due to high levels of intestinal β-glucuronidase enzyme, decreased intestinal bacteria, decreased gut motility.

c. Defective uptake of bilirubin from plasma due to decreased ligandin and binding ofligandin by other anions.

d. Defective conjugation due to decreased UDPG-T activity.

e. Decreased hepatic excretion of bilirubin.

II Non physiologic jaundice[14]

a. Over production

- Feto maternal blood group incompatibility.

- Hereditary Spherocytosis, Elliptocytosis, Stomatocytosis.

- Nonspherocytic hemolytic anemias.

- G6 PD deficiency and drugs.

- Pyruvate kinase deficiency.

- Other red cell enzyme deficiencies

- α - Thalassemia

- δ - β-Thalassemia

- Acquired hemolysis due to vitamin K, Nitrofurantoin, Sulfonamides, Antimalarials, Penicillin, Oxytocin, Bupivacaine or Infection.

- ❖ Extra vascular blood :Petechiae, hematomas, pulmonary, cerebral or occult hemorrhage.
- ❖ Polycythemia :Fetomaternal or fetofetal transfusion. Delayed clamping of the umbilical cord.
- ❖ Increased enterohepatic circulation
- ❖ Pyloric stenosis,
- ❖ Intestinal atresia or stenosis including annular pancreas,
- ❖ Hirschsprung disease,
- ❖ Meconium ileus or Meconium plug syndrome,
- ❖ Swallowed blood.

b. Undersecretion

- o Metabolic or endocrine conditions
- o Galactosemia
- o Familial Nonhemolytic jaundice (crigler-Najjar syndrome and Gilbert syndrome)
- o Hypothyroidism
- o Tyrosinosis
- o Hypermethioninemia
- o Drugs and Harmones – Novobiocin, Pregnanediol
- o Lucy – Driscoll syndrome
- o Infants of diabetic mothers
- o Prematurity, Hypopitutarism and Anencephaly.
- o Obstructive disorders
- o Biliary atresia
- o Dubin Johnson and Rotor syndrome

- Choledochal cyst
- Cystic fibrosis (inspissated bile)
- Tumor or band (extrinsic compression)
- Parenteral nutrition
- α1 – antitrypsin deficiency

c. Mixed

- Sepsis
- Intrauterine infections
- Toxoplasmosis
- Rubella
- CID
- Herpes simplex
- Syphilis, Hepatitis
- Respiratory distress syndrome
- Asphyxia
- Infant of diabetic mothers
- Severe erythroblastosisfetalis

d. Uncertain mechanism

- Breast milk jaundice
- Chinese, Japanese, Korean and American indian infants.

Causes of jaundice on the basis of age of onset[37]

Within 24 hours : Rh and ABO incompatibility, G6 PD and PK enzyme deficiency. Infections – Bacterial, Malarial, TORCH, Hereditary Spherocytosis, α-thalassemia,Administration of large amount of drugs – vitamin K, salicylates, sulfisoxazole etc. to the mother.

24 – 72 hours after birth : Physiologic jaundice, Blood group incompatibility, Polycythemia, Extra vascular bleed – cephalohematoma, subgaleal hemorrhage, breast feeding jaundice, neonatal sepsis, Increased enterohepatic circulation –intestinal obstruction.

After 72 hours of birth : Neonatal sepsis, Cephalohematoma, Neonatal hepatitis, Biliary atresia, Breast milk jaundice, Metabolic - Hypothyroidism, Hypopitutarism, Galactosemia, Tyrosinemia, Cystic Fibrosis, Hereditary fructosemia, Crigler - Najjar Syndrome, Gilbert Disease.

Complications of neonatal jaundice

Bilirubin encephalopathy refers to the clinical manifestations of the effects ofbilirubin on the central nervous system, where as kernicterus refers to the neuropathologic changes that are characterized by pigment deposition in specific areas of the CNS such as basal ganglia, pons and cerebellum.[19]

Bilirubin encephalopathy is a multifactorial process that requires a critical level of free bilirubin, access to the brain across the blood-brain barrier, and presence of susceptible nerve cells. The severity and duration of hyperbilirubinemia, the maturity of the structures involved, the binding capacity of albumin, the physiologic environment, and the cell membrane composition and metabolic state probably all are critical to the development of neurodysfunction.[4]

Entry of bilirubin into the brain

The mechanism by which uncojugated bilirubin enters the brain and damages it is unclear. Several hypotheses regarding entrance of bilirubin into the brain have been proposed.[19]

One hypothesis is the lipophilic nature of free bilirubin, in equilibrium with bound bilirubin, has access to tissues. Thus, any increase in the amount of free bilirubin or

reduction in the amount or binding capacity of albumin could increase the level of unbound bilirubin within the brain tissue, saturating membranes and causing precipitation of bilirubin acid within the nerve cell membrane.[19]

Second hypothesis is based on close examination of the chemical nature of bilirubin in solution and seeks to explain the increased risk in acidotic infants. In this theory, the rate of tissue uptake of bilirubin depends on both the concentration of albumin-bound bilirubin and the pH, with low pH enhancing precipitation and tissue uptake.[19]

Third theory suggests that bound bilirubin enters the brain mainly through a damaged blood-brain barrier.[19]

Recent studies suggest that unconjugated bilirubin is a substrate for Pglycoprotein (P-gp) and that the blood-brain barrier P-gp may play a role in limiting the passage of bilirubin into the CNS. P-gp is an ATP – dependent integral plasma membrane transport protein that translocates a wide range of substrates across biologic membranes.[19]

Factors that increase susceptibility to Neurotoxicity associated with Hyperbilirubinemia

Asphyxia, Hyperthermia, Septicemia, Hypoalbuminemia, Acidosis, Calorie deprivation, Prolonged Hyperbilirubinemia, Low birth weight, Young gestational age, Excessive hemolysis. [19]

Bilirubin toxicity at cellular level[19]

Four possible mechanisms have been proposed:

- Interruption of normal neurotransmission
- Mitochondrial dysfunction
- Cellular and intracellular membrane impairment
- Interference with enzyme activity

Clinical features[14]

Early : Lethargy, poor feeding, high pitched cry, hypotonia.

Intermediate : Irritability, opisthonous, seizures, apnea, oculogyric crisis, hypertonia , fever, retrocollis.

All infants who survive this phase develop chronic bilirubin encephalopathy (clinical diagnosis of kernicterus)

Advanced phase : Pronounced opisthonous, shrill cry, apnea, seizures, coma and death.

Chronic bilirubin encephalopathy (kernicterus)

It is marked by athetosis, athetoid cerebral palsy, partial or complete high Frequency sensorineural hearing loss, paralysis of upward gaze, dental dysplasia and intellectual deficits.[14]

Predicting Encephalopathy and Reversibility of damage[19]

Brainstem Evoked Auditory Response- Because auditory pathway of the newborn is particularly vulnerable to insult from the bilirubin, BEAR testing has been suggested as a tool that could identify or predict early effects of hyperbilirubinemia. Studies have shown increased bilirubin concentrations with changes in the amplitude and latency of these responses. BEAR is accurate and non invasive and assesses the functional status of the auditory nerve in the brainstem pathway.

BEAR testing could be used to screen hyperbilirubinemic full-term and premature infants for sensorineural hearing loss and could be incorporated into the assessment of need for exchange transfusions (Wennberg et al, 1982). [38]

Infant Cry Analysis - It has been shown that with moderately elevated ST Blevels, there is interference with neural conduction, as demonstrated by the BEAR, and changes in neural function in adjoining pathways, with resultanteffects on the vocal cords (increased tension on phonation). [19]

Nuclear Magnetic Resonance Techniques – Nuclear magnetic resonance (NMR)techniques, both imaging and spectroscopy, have been proposed as a rapid,noninvasive measure of impending or actual brain cell injury in the face of hyperbilirubinemia7 (Palmer and Smith, 1990).[39]

Evaluation, prediction and diagnosis of neonatal jaundice

Between 25% and 50% of all term newborns and a higher percentage of premature infants develop clinical jaundice. A serum bilirubin level of >15 mg/dl is found in 3% of normal term babies. Physical examination is not a suitable measure of serum bilirubin estimation. [14]

Kramer criterion

Visual assessment of serum bilirubin levels as suggested by Kramer (1969) , which relies on the cephalocaudal progression of jaundice with the raising serum bilirubin levels, head and neck,4 to 8 mg/dl ; upper trunk,5-12 mg/dl ; lower trunk and thighs,8 to 16 mg/dl ; palms and soles, greater than 15 mg/dl ; is now known to be fraught with error.[40]

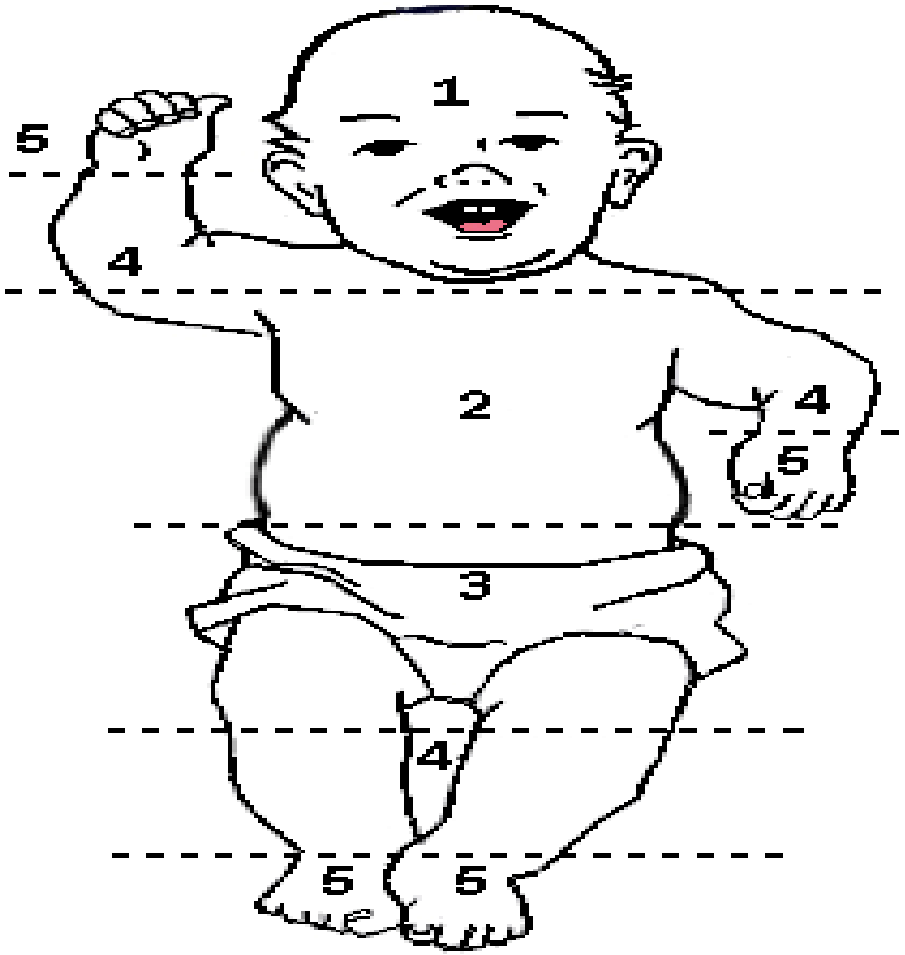


FIGURE – 2 KRAMER`S RULE[40]

Hour specific nomogram

Bhutani and colleagues (1999) generated a percentile based bilirubin nomogram using hour specific pre discharge STB levels from a racially diverse group of term healthy newborns with no ABO or Rh incompatibility who did not need phototherapy before 60 hours of age and of whom 60% were breastfed. Post discharge STB levels were measured by a hospital based bilirubin assay within 3 days after discharge. The risk for significant hyperbilirubinemia (STB greater than 17 mg/dl) for infants with a pre-discharge STB above the 95th percentile (high risk zone) was 57%,for infants with STB between the 75th and 95th percentiles (high intermediate risk) it was 13%, for infants with STB between the 40th and 75th percentiles (low intermediate risk zone) it was 2.1%, and for infants below 40th percentiles (low risk) it was 0. Limitations of this approach are the need for blood sampling and the cost of STB measurement.[41]

Transcutaneous bilirubin measurement (TcB)

Transcutaneous (TcB) is based on the measurement of light reflected from the skin. Many devices (Bilicheck ,Norcross ,Georgia) that measures the entire spectrum of visible light reflected from the skin has been shown to provide an accurate assessment of STB in term and near term newborn infants of diverse races and ethnicities. It is important to note that TcB measured is not the serum bilirubin but the amount of bilirubin that has moved into the tissues.[19]

End tidal carbon monoxide (ETCOc) measurement

The breakdown of Heme by the rate limiting enzyme HemeOxygenase leads to the formation of equimolar amounts of CO and Biliverdin.The measurement of CO in the exhaled breath in the newborn can be used as an index of heme degradation and bile production in vivo. It can help to distinguish between cases of increased bilirubin

production versus decreased elimination or impaired bilirubin conjugation.[19]

Risk factors for development of neonatal hyperbilirubinemia in infants of 35 or more weeks gestation[14]

- Major risk factors

- Pre-discharge TSB or TcB level in high risk zone
- Jaundice observed in the first 24 hours
- Blood group incompatibility with positive DCT or elevated ETCOc.
- Gestational age 35-36 weeks
- Previous sibling received phototherapy
- Cephalohematoma or significant bruising
- Exclusive breast feeding, particularly if nursing is not going well and weight loss in excessive.
- East Asian race.

Minor risk factors

- Pre discharge TCB or TcB level on the high intermediate risk zone
- Gestational age 37-38 weeks
- Jaundice observed before discharge
- Previous sibling with jaundice
- Macrosomic infant of a diabetic mother
- Maternal age ≥ 25 years
- Male gender

Decreased risk

- TSB or TcB level in low risk zone
- Gestational age ≥ 41 weeks
- Exclusive bottle feeding
- Black race
- Discharge from hospital after 72 hours.

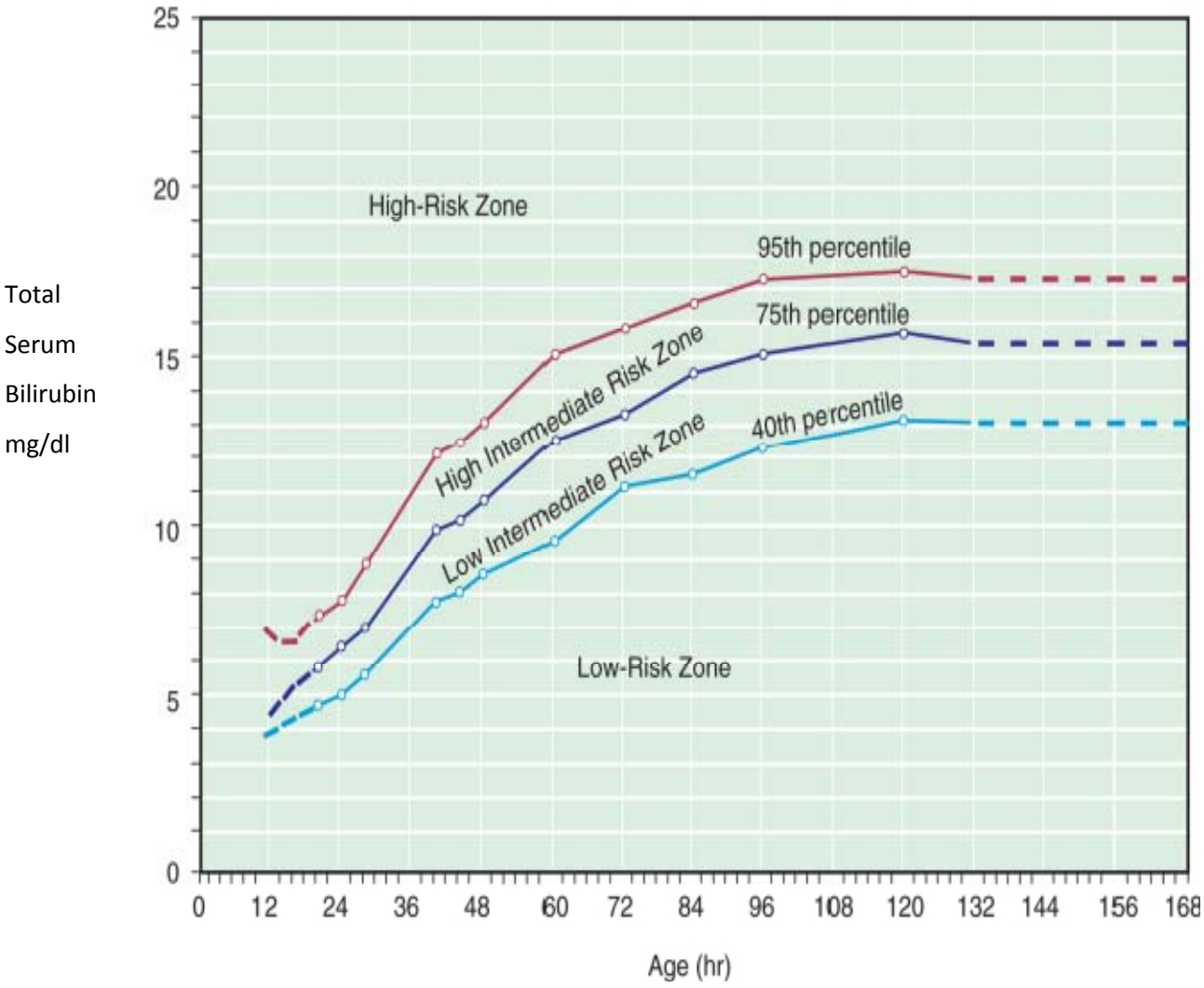


Fig 3 : Risk designation of term and near-term well newborns based on their hour-specific serum bilirubin values.

The high-risk zone is subdivided by the 95th percentile track. The intermediate at risk zone is subdivided into upper and lower risk zones by the 75th percentile track. The low-risk zone has been electively and statistically defined by the 40th percentile.[4]

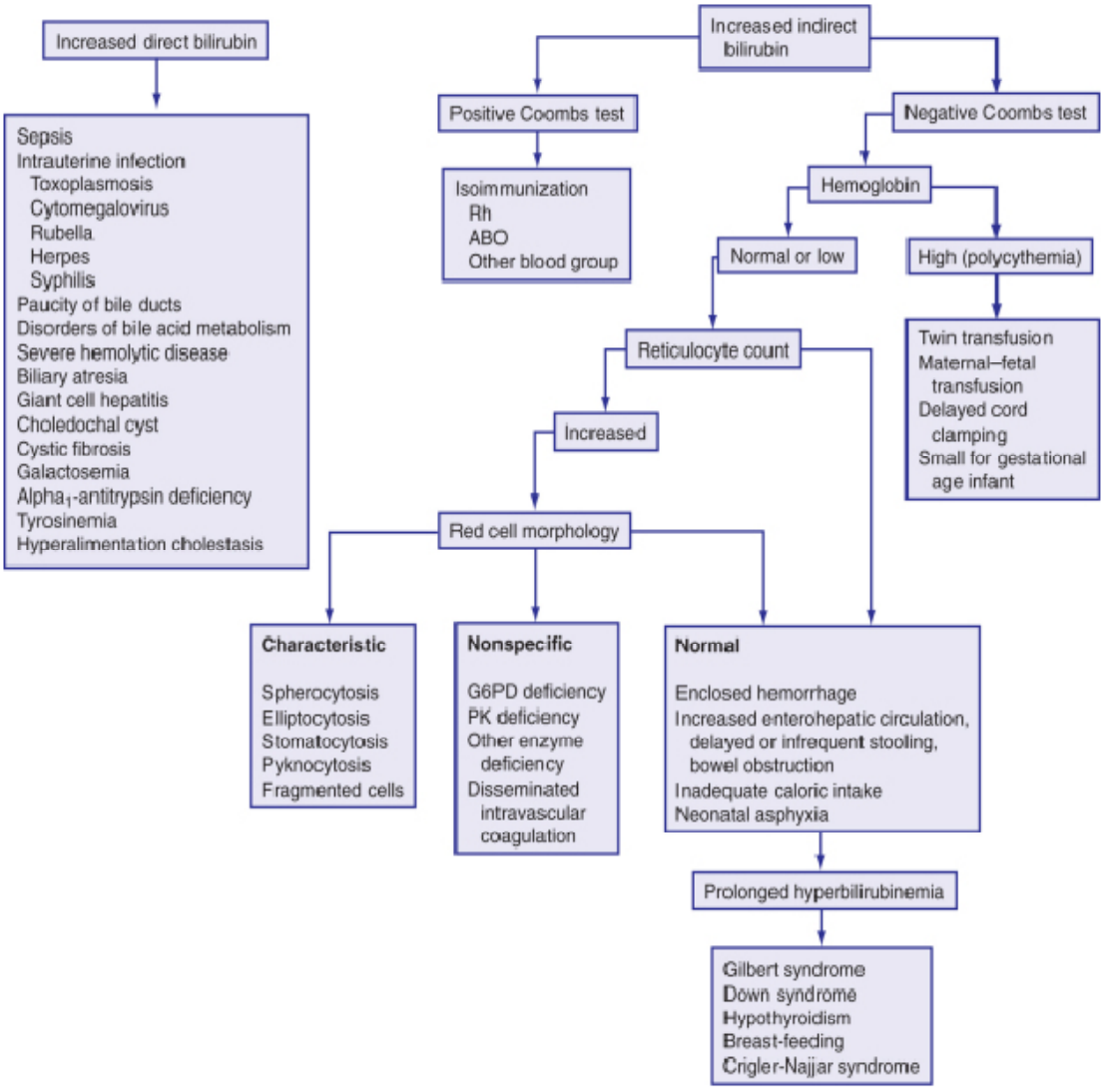


Fig 4 : Schematic approach to the diagnosis of neonatal jaundice [19]

Criterion for physiological jaundice [42]

- Type of bilirubin – Indirect bilirubin,
- Direct bilirubin never more than 2mg/dl or less than 15% of total bilirubin,
- Appearance - after 36 hours of age,
- Rate of rise of bilirubin – less than 5mg / dl/day,
- Severity of jaundice – Usually does not exceed 15 mg/dl,
- Natural course – Peak STB levels seen between 3rd – 5th days of life and 3rd – 7^{th} day in preterm and disappears by 2 weeks.
- Clinical condition – Healthy newborn.

Pathological jaundice is suspected in the newborn with [42]

- Clinical jaundice in the first 24 hours of life.
- STB > 15 mg/dl
- Rate of STB increase > 0.2 mg/dl/hr or 5mg/dl/day.
- Direct serum bilirubin > 2mg/dl or > 15% of total bilirubin
- Clinical jaundice persisting for > 2 weeks.

Guidelines for Phototherapy and Exchange transfusion in hospitalized infants of 35 or more weeks' gestation are depicted in Fig. 4 and Fig. 5 respectively. [11] (From American Academy of Pediatrics Subcommittee on Hyperbilirubinemia: Management of hyperbilibubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics 2004; 114:297-316).

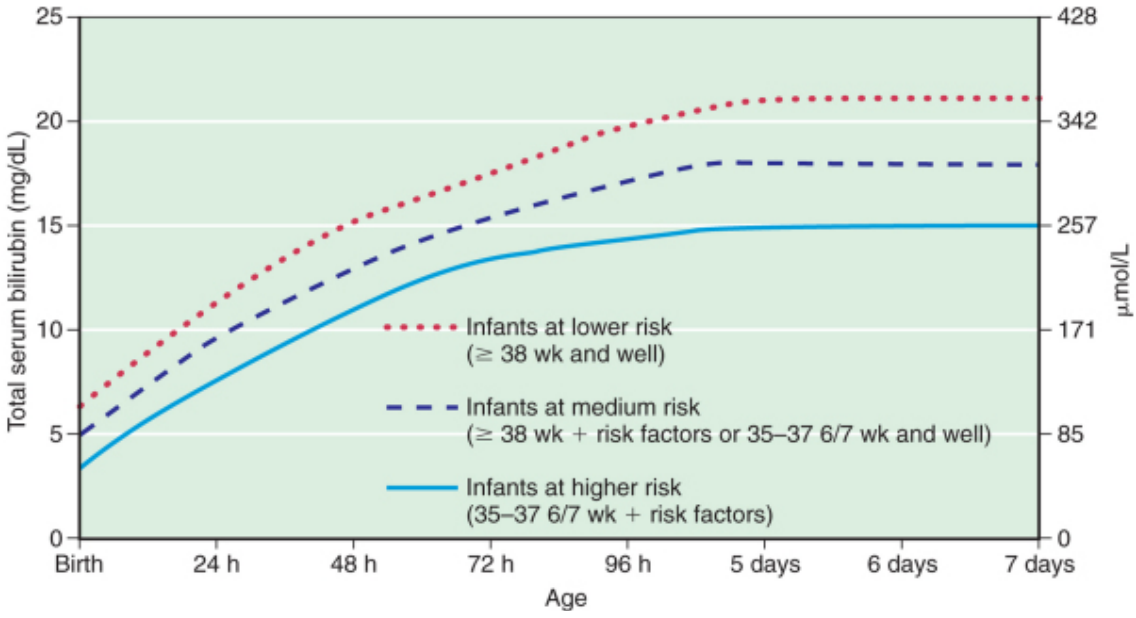


Fig. 4 : Guidelines for phototherapy in hospitalized infants of 35 or more weeks' gestation[4]

- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- Risk factors = isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin <3.0 g/dL (if measured).
- For well infants 35-37 6/7 wk can adjust TSB levels for intervention around the medium risk line. It is an option to intervene at lower TSB levels for infants closer to 35 wks and at higher TSB levels for those closer to 37 6/7 wk.
- It is an option to provide conventional phototherapy in hospital or at home at TSB levels 2-3 mg/dl (35-50mmol/L) below those shown but home phototherapy should not be used in any infant with risk factors.

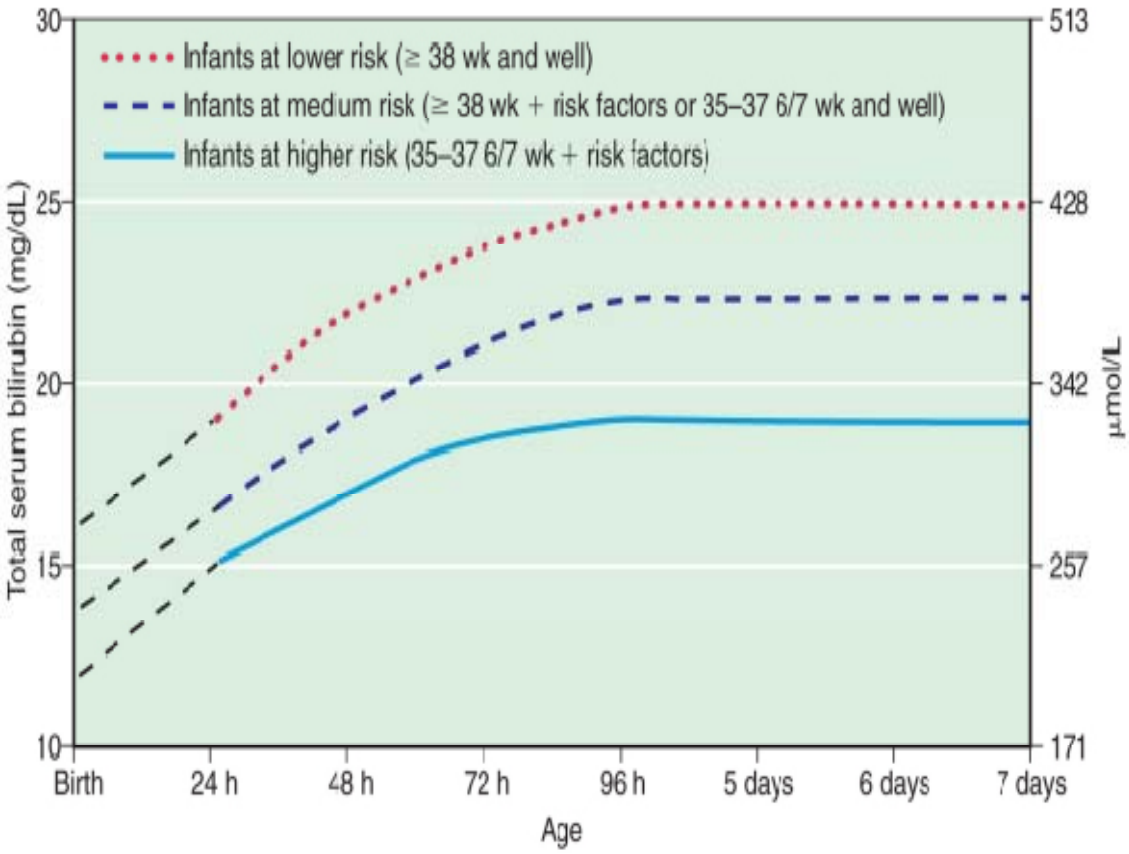


Fig. 5 : Guidelines for exchange transfusion in hospitalized infants of 35 or more weeks' gestation[4]

- The dashed lines for the first 24 hours indicate uncertainty due to a wide range of clinical circumstances and a range of responses to phototherapy.
- Immediate exchange transfusion is recommended if infant shows signs of acute bilirubin encephalopathy (hypertonia, arching, retrocollis, opisthotonos, fever, high pitched cry) or if TSB is ≥ 5 mg/dl (85 µmol/L) above these lines.
- Risk factors – isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis.
- Measure serum albumin and calculate B/A ratio.
- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin.
- If infant is well and 35-37 6/7 wk (median risk) can individualize TSB levels for exchange based on actual gestational age.

Laboratory evaluation[19]

I. Maternal: Blood grouping and Indirect Coombs Test (ICT) to test for Isoimmune hemolytic disease, Serology to rule out syphilis.

II. Infant :

- Total serum bilirubin and or Transcutaneous bilirubin.

- Blood grouping, Rh typing and Directcoomb test to test for isoimmune hemolytic disease.

- Hemoglobin and Hematocrit.

Anemia suggests hemolytic disease and large entrapped hemorrhage.

- Polycythemia cause jaundice.

- Reticulocyte count is elevated in hemolytic anemia.

- Red cell morphology – By peripheral blood smear

- Red cell fragmentation seen in disseminated intravascular coagulation (DIC)

- Spherocytes suggests ABO incompatibility or Hereditery Spherocytosis.

- Platelet count is decreased in infections.

- White blood cell count less than 50,000 cells/cumm or BNR> 0.2 suggest infection.

- Urine analysis for reducing substance to diagnose Galactosemia.

- Screening of G6 PD deficiency.

- Serum protein and albumin to estimate albumin binding capacity and reserve albumin binding site.

- pH

- Protein binding (2,4hydroxybenzeneazobenzoic acid (HABA), Salicylates)

These tests helps to measure the quantity of binding of bilirubin in the serum of jaundice infants.

i. Treatment of Neonatal Hyperbilirubinemia[37]

The aim of the therapy is to ensure that serum bilirubin is kept at a safe level and brain damage is prevented. Neonatal Hyperbilirubinemia is a medical emergency and delay in its management can lead to irreversible brain damage and death.

Preventive and suggestive measures

- Drugs known to aggravate jaundice or block the bilirubin binding sites on albumin should be withheld.
- Vitamin K in large doses should be avoided.
- Perinatal distress factors such as hypoxia, acidosis, hypothermia, hypoglycemia should be prevented or adequately managed.
- Use of phenolic detergents are avoided in nursery as they may enhance the jaundice in the babies.

Adequate feeding

Early feeding augments colonization of the gut and reduces the enterohepatic circulation. Effective evacuation of meconium is associated with elimination of conjugated bilirubin and stercobilin.

Pharmacological management

Phenobarbitone

Barbiturates have been shown to induce the maturation of microsomal enzymes, ligandin (Y-acceptor protein) and glucuronyltransferase (UDPG-T), thus improving the uptake, conjugation and excretion of bilirubin by the liver.

Phenobarbitone in a single dose of 10 mg/kg im or 5mg/kg/day in two divided doses orally for 3 days is indicated in cases of cord serum bilirubin level of > 2.5 mg/dl, early onset of jaundice due to any cause, difficult or instrumental delivery,Oxytoc in induced delivery with bruising and cephalohematoma.

Clofibrate

It is a potent enhancer of glucuronyltransferase. It is more efficacious but it is slow in its action and takes several days to show the beneficial effect.

Agar

It is a sea weed extensively used in processing of food. In dose of 250mg 6^{th} hourly orally it binds conjugated bilirubin in the gut and blocks the enterohepatic circulation. Its use is unpredictable and variable.

Cholestyramine

In dose of 1.5 mg/kg/day in 4 divided doses mixing in milk feeds has been shown to enhance fecal exertion of bilirubin and thus blocking enterohepatic circulation. Infant should be watched for constipation – intestinal obstruction and hypercholeremic acidosis.

Orotic acid

It is a metabolic precursor of uridinediphosphateglucuronic acid and thus promotes the conjugation of bilirubin. Its ability is limited and cost is prohibitive.

Tin-mesoporphyrin (SnMP)

Metalloporphyrins (Tin and Zinc) are structural analogs of heme and they inhibithemeoxygenase. It diminish the production of bile pigments by competitive inhibition. Hemeoxygenase is a rate limiting enzyme in heme metabolism. Tin mesoporphyrin (6 μmol/kg/single dose im) has been shown to significantly reduce bilirubin production. It is associated with high incidence of photosensitive skin reactions and potential risk of hepatic and renal toxicity.

Albumin infusion

When administered (1 gm/kg), half an hour before exchange transfusion it facilitates more effective removal of bilirubin and also improves the bilirubin binding capacity

of the baby. Use is avoided in babies with congestive cardiac failure because of risk of overloading the circulation. Rarely used due to exorbitant cost and risk of transmission of viral infections.

Inhibiting hemolysis[14]

IvIg (500-1gm/kg) used to reduce bilirubin levels in infants with Isoimmune Hemolytic disease. The immunoglobulins act by occupying the Fc receptors of reticuloendothelial cells, there by preventing them from taking up and lysing antibody coated Red Blood Cells.

Phototherapy[37]

Widely accepted, relatively safe and effective method for treatment of neonatalhyperbilirubinemia. Bilirubin absorbs light maximally at 420-460 nm and light sources with peak emissions in this range lower serum bilirubin levels by several mechanisms.

Photo oxidation

Photo oxidation of bilirubin into water soluble colorless form of bilirubin is very slow, ineffective.

Configurationalphotoisomerization

Here E-isomers (4Z 15E, 4E 15E, 4E 15Z) which are more polar water soluble diazo negative compounds are produced. E isomers are nontoxic and after 8-12 hours of phototherapy they constitute about 25% of total serum bilirubin.

Structural isomerization

It is the production of stable water soluble structural isomers of bilirubin like lumirubin. These photocatabolites are readily excreted in bile, feces and to a lesser extent in urine. The conversion of bilirubin to lumirubin is irreversible and it cannot be reabsorbed. It is most important pathway for the lowering of serum bilirubin levels

and strongly related to the dose of phototherapy used in the range of 6 to 12 μw/cm2/nm.

Procedure of phototherapy

The narrow spectral blue light is most effective for phototherapy but it interferes with proper observation of the infant. White day light fluorescent lamps are quite effective and commonly used in our country. Blue and white tubes phototherapy unit are also available.

Nude infant is exposed to a portable or fixed light source kept at 45cm from the skin. Distance between the baby and phototherapy unit can be reduced to 15-20 cms to provide effective and more intensive phototherapy.

During phototherapy eyes must be shielded to prevent retinal damage and a diaper should be kept on to cover the genitals. For effective phototherapy, the minimal spectral irradiance or 'flux' of 4 to 6 μw/cm2/nm is available and maintained at the level of the infant's skin.

Side effects

- Passage of loose green stools because of transient lactose intolerance and irritant effect of photocatabolites causes increased colonic secretory losses
- Hyperthermia
- Irritability
- Dehydration
- Flea bite rash on the trunk or extremities
- Risk of opening up to PDA in preterm babies.
- Hypocalcemia due to secretion of melatonin from pineal gland
- Bronze baby syndrome – Infants with parenchymal liver disease with biliary

obstruction, due to excessive accumulation of bilifucin (Polymerized form of lumirubin) imparting brownish discoloration to the skin.

- Theoretically increased risk of skin malignancy later in life.

- Exposure to light may disturb the Circadian rhythm of the sex hormones thus having potential implications on onset of puberty and disturbances in future sex behavior.

Exchange transfusion[14]

There is no single reliable laboratory parameter that can predict with certainty the potentiality for development of brain damage due to bilirubin.[37]

Need for exchange transfusion is based on level of unconjugated serum bilirubin, gestational maturity, postnatal age, existence of or otherwise perinatal distress factors and the cause of jaundice.[37]

Choice of blood[14]

- O Rh negative blood in emergency situations.
- Fresh (<7 days old) type O cells with AB plasma to ensure that no anti A and anti B antibodies are present.
- In non immunehyperbilirubinemia, blood is typed and cross matched against the plasma and red cells of the infant. Exchange transfusion usually involve double the volume of the infant's blood and is known as a Two volume exchange.[160 ml/kg]. This replaces the 87% of infant's blood volume with new blood.

Technique

a) Exchange transfusion is done by push pull technique through the umbilical vein inserted only as far as required to permit the free blood exchange.

b) Isovolumetric exchange transfusion –Simultaneously pulling blood out of the umbilical artery and pushing new blood in the umbilical vein may be better tolerated in small sick or hydropic infants.

c) Exchange transfusion can be accomplished through central venous pressure line placed through the anticubital fossa or into the femoral vein through the saphenous vein and radial artery.

- In push pull method, blood is removed in aliquots that are tolerated by the infant.
- Usually 5ml for <1500gms, 10ml for infants 1500-2500gms, 15ml for 2500-3500gms and 20ml for >3.5kgs.
- The recommended time for the exchange transfusion is 1 hour.

Complications of Exchange transfusion

1. Hypocalcaemia and Hypomagnesemia : The citrated blood used binds ionic calcium and magnesium.
2. Hypoglycemia : High glucose content of CPD (300mg/dl) stimulates insulin secretion and causes hypoglycemia 1-2 hours after exchange.
3. Acid base balance: Citrate in CPD blood is metabolized to alkali resulting in latemetabolic alkalosis.
4. Hyperkalemia : Potassium levels may be greatly elevated in stored PRBC's.
5. Cardiovascular : Perforation of vessels, embolisation, vasospasm, thrombosis, infarction, arrhythmia, volume overload, arrest.
6. Bleeding : Thrombocytopenia, deficient clotting factors.

7. Infections : Bacteremia, hepatitis, CMV, HIV, West Nile virus and malaria.
8. Hemolysis :Hemoglobinemia, hemoglobinuria, and hyperkalemia caused by over heating of the blood have been reported.
9. Graft-versus-host disease. This is prevented by using irradiated blood.
10. Miscellaneous: Hypothermia, hyperthermia and possibly necrotizing enterocolitis.

PREDICTION OF NEONATAL HYPERBILIRUBINEMIA

Johnson et al reported that hospital stays, decreased vigilance in diagnosing jaundice and lack of physician compliance with current guidelines may account for re-emergence of severe hyperbilirubinemia and kernicterus.[43] Since 1991, there have been several articles published on re-emergence of kernicterus in full term infants.[1,44,45]

Over the past several decades there has been a shortening in the length of hospital stays for neonates and their mothers. Before World War II, the average length of hospital stay for women delivering vaginally was 7 to 10 days , while the average length of hospital stay for women delivering vaginally was 7 to 10 days , while in the postwar period this was shortened to 3 to 5 days. Data from the United States National Hospital Discharge Survey indicate that between 1970 and 1994, the average length of hospital stay for all deliveries continued to decrease. During this time period, the average length of stay for vaginal deliveries decreased from 3.9 to 2.0 days and the average length of caesarean section deliveries decreased from 7.8 to 3.9 days.[8]

An analysis of trend data indicated that in 1980, approximately 30% of all infants born in U.S Hospitals where discharged at 2 days or less, 30% were discharged at 3 days and remaining percent were discharged at 4 days or more. By 1993 30% of all infants born in hospitals were discharged at 1day, 38.5% at 2 days and 17% at 3 days and only 14.3% at 4 days or more.[8]

There is litany of problems that have been reported to be associated with or exacerbated by early discharge, missed new born screening hyperbilirubinemia, non initiation or premature cessation of breast feeding , feeding problems leading to dehydration and malnutrition, missed identification of congenital anomalies,

readmission and maternal postpartum cognitive deficits. [8]

Jaundice appears in 60% of term newborns and 80% of preterm infants by the first week of life. Upto 4% of term newborns who are readmitted to the hospital during their first week of life, approximately 85% are readmitted for jaundice. If left unrecognized and untreated, hyperbilirubinemia can have dire consequences. Overproduction and reduced removal of bilirubin may elevate serum bilirubin levels to toxic levels that presents the threat of brain damage (kernicterus). [8]

Kernicterus, resulting from the deposition of unconjugated bilirubin in brain cells, is clinically characterized by convulsions, opisthotonos, hypotonia, high pitched cry, and fever. Because of effective diagnosis and treatment, kernicterus has become a rare event. It is of concern, however that this rare event, while still infrequent, is becoming more common. Catz and colleagues recounted that in the United Stats, between 1991 and 1995, there were reports of 22 cases of term or near-term infants who had developed kernicterus after being discharged within 48 hours of birth. Of these cases 95% (n = 21) had been breastfed, 23% (n = 5) were glucose 6- phosphate dehydrogenase deficient, and others had identifiable risk factors such as bruising, ABO hemolytic disease , or other causes of hemolysis.[8]

The current list of identified risk factors to recognize infants who are likely to require treatment for hyperbilirubinemia is not adequate. While jaundice per se is not always preventable, nonetheless, early detection of threatening bilirubin levels permits initiation of phototherapy and prevents higher risk and higher cost exchange transfusion therapy or kernicterus. Early discharge complicates the ability to measure both the level of serum bilirubin and the rate of increase.[8]

Today, financial rather than family or medical consideration frequently influences

the decision about hospital discharge of the mother and infant after birth. Increasingly insurers are refusing payment for a hospital stay that extends beyond 24 hours after an uncomplicated vaginal delivery.[46] Early hospital discharge may be responsible for development of severe hyperbilirubinemia and kernicterus.[47] Hyperbilirubinemia is the commonest cause for readmission to the hospital after early discharge.[46-49]

In 1977, Risemberg et al. established a correlation between bilirubin levels in the umbilical cord blood and hyperbilirubinemia in newborns with ABO incompatibility. These researchers concluded that newborns presenting levels higher than 4 mg/100ml were a group at risk of developing severe hyperbilirubinemia and should be followed up and reassessed, since all of them presented serum bilirubin levels that were higher than 16 mg/100ml between 12 and 36 hours of life. In the present study, phototherapy was significantly associated with the presence of blood group incompatibility between mother and child, as well as with the unconjugated bilirubin level in cord blood. There was also a significant association between the unconjugated bilirubin in cord blood and the newborn's bilirubin level.[50]

In 1983 Palmer et al, presented a review of jaundiced newborn infants during the 10-year period to 1980. Included those whose serum bilirubin level was 9 mg/dl or more. Of 41,057 live births, 4,406 (10.7%) infants had hyperbilirubinemia. The most common (19.9;%) aetiological factor was prematurity, followed by ABO isoimmunisation 7.1%; sepsis 3.4%; Rhesus isoimmunisation 2.7%; bruising 2.2%; multifactorial 1.0% and glucose-6-phosphate dehydrogenase deficiency 0.5%. Treatment was not undertaken in 2,855 (64.7%) infants, but 1,419 (32.2%) received phototherapy alone, 122 (2.7%) infants received both exchange transfusion and phototherapy and 10 (0.2%) infants received exchange transfusion alone.

Of the infants requiring exchange transfusion 50.0% had Rhesus isoimmunisation, 28.0% ABO isoimmunisation, 10.6% jaundice of prematurity and the remainder were due to a variety of causes.[51]

In 1986 Rosenfeld J et analysed group of 108 full term newborns and reported that Infants with cord bilirubin levels less tha2.0 mg/dl have only a 4 percent chance of developing hyperbilirubinemia and a 1.4 percent chance of needing phototherapy. However, if serum cord bilirubin levels are more than 2.0 mg/dl, the infant has a 25 percent chance of developing subsequent hyperbilirubinemia.[52]

In 1989 Knudsen A et al in his study on unconjugated bilirubin concentration as a predictor of subsequent jaundice on 291 newborns found that if cord bilirubin was below 1.17 mg/dl, 2.9% became jaundiced as opposed to 85% if cord bilirubin was above 2.3 mg/dl. Furthermore, 57% of jaundiced infants with cord bilirubin above 2.3 mg/dl required phototherapy, but only 9% if cord bilirubin was 2.3 mg/dl or lower ($p< 0.003$). Since the ability of plasma to bind bilirubin in cord blood from jaundiced and non-jaundiced infants showed no significant differences, the increased cord bilirubin among infants who later became jaundiced is presumably caused by increased fetal bilirubin production or decreased removal of bilirubin from the fetal circulation.[53]

In 1991 Honket et al, Reported a 4-year experience (1986-1989) of neonatal jaundice. Babies who have received some form of treatment such as phototherapy are considered as cases of neonatal jaundice. However, the incidence of hyperbilirubinemia (defined as serum bilirubin level of 15 mg/dl or greater) fell from 3.23% to 2.11% of all live births. ABO Incompatibility, glucose-6-phosphate dehydrogenase (G6PD) deficiency and low birth weights (LBW) remain as the common aetiological factors of neonatal jaundice.[54]

In 1994, Rataj et al, investigated 800 healthy full-term newborns and reported that if cord bilirubin was under 1 mg% the jaundice occurred in 2.4% newborns, where as 89% of the infants with cord bilirubin above 2.5 mg% became jaundiced.[55]

Awasthi et al 1998, conducted a prospective cohort study of 274 neonates born in North India and predicted that occurrence of peak serum bilirubin level >15 mg/dl between second to fifth postnatal day by using serum bilirubin level measured between 18 to 24 hrs of life. The main outcome measures were hyperbilirubinemia and phototherapy. Hyperbilirubinemia was found in 12.8% babies using a cut-off >3.99 mg/dl with sensitivity and specificity of 67%. Using serum bilirubin levels estimated at 18-24 hrs of life as the "prediction test", approximately two-third of the neonates were test negative and had one in ten chances of readmission for treatment of hyperbilirubinemia, if discharged early.[56]

Alpay et al 2000, followed up a total of 498 healthy term newborns daily with serum total bilirubin measurements for the first 5 days of life, and cases with TSB of >17 mg/dl after 24 hours of life were defined to have significant hyperbilirubinemia. No newborns had a TSB level of >17 mg/dl in the first 72 hours of life. Sixty of 498 cases (12.05%) had significant hyperbilirubinemia after 72 hours of life, and these cases had significantly higher bilirubin levels than those who did not develop significant hyperbilirubinemia on each of the first 5 days' measurements. Of the 206 newborns who had a TSB level of >6 mg/dl in the first 24 hours, 54 (26.21%) developed significant hyperbilirubinemia, whereas only 6 of the 292 newborns (2.05%) who had a TSB level of <6 mg/dl on the first day developed significant hyperbilirubinemia. A mean TSB level of >6 mg/dl on the first day had the highest sensitivity (90%). At this critical serum bilirubin value, the

negative predictive value was very high (97.9%) and the positive predictive value was fairly low (26.2%). The use of the critical bilirubin level of 6 mg/dl in the first 24 hours of life can predict nearly all of the term newborns who will have significant hyperbilirubinemia and could determine all those who will require a phototherapy treatment later during the first days of life.[57]

Agarwal et al 2002, conducted a study on 220 infants. All infants were exclusively breastfed. Clinically detectable jaundice was present in 164(77%) and hyperbilirubinemia occurred in 22(10.3%) infants. Study predicted that infants with total serum bilirubin levels lesser than 6 mg/dl at 24 ± 6 hours would not develop hyperbilirubinemia.[58]

Suchonska et al 2004, investigated 187 healthy, full-term newborns in good general condition. Newborns with serological incompatibility were not included into the study. In 155 (83%) cases babies were born through normal vaginal delivery, in 32 (17%) by Caesarean section. The umbilical blood was taken immediately after delivery and the venous blood on the 3rd day of life to determine concentration of bilirubin. 3rd day Bilirubin values lower than 12.9 mg% were considered physiological. Hyperbilirubinemia was recognized when the concentration of bilirubin was over 12.9 mg%. Pearson test was used to estimate the correlation between bilirubin value in the umbilical blood and the venous blood. The mean value of total bilirubin in the umbilical blood was 1.30 mg% +/- 0.47 and in venous blood on the 3rd day of life 8.07 mg% +/- 3.08. No one with umbilical bilirubin concentration lower than 1 mg% developed hyperbilirubinemia. They concluded that concentration of bilirubin in the umbilical blood can be useful indicator of risk of icterus in newborns. Special care is needed for newborns whose concentration of bilirubin in umbilical blood is over 1 mg%.[59]

Bernaldo et al 2004, predicted that Blood incompatibility between mother and child was a predictor for the appearance of hyperbilirubinemia that required treatment. Considering a cut-off point of 2.0 mg/dl, 53% of the newborns who had greater unconjugated bilirubin levels in cord blood would reach levels requiring phototherapy by the third day of life. In addition, they also concluded that the presence of mother/child blood group incompatibility was statistically significant for the occurrence of unconjugated bilirubin serum levels that were indicative of phototherapy treatment during the same three-day period.[60]

In 2005, Kupfer et al investigated the predictive value of umbilical cord serum bilirubin (CBB) for the postnatal course of bilirubinaemia in healthy term and near- term newborns. Term appropriate-for-gestational-age (AGA; *n*=1100), small-for- gestational-age (SGA; *n*=163) and near-term infants (GA 34–36 wk; *n*=78) were included and separated according to their CBB levels, starting from <1.1 (group 1), 1.1–<1.7 (2), 1.7–2.3 (3) and >2.3 (4) mg/dl. The newborns were followed for at least 5 postnatal days, and CBB values were correlated with the development of hyperbilirubinemia and phototherapy (PT) treatment which showed a clear relation between CBB and the development of hyperbilirubinemia in all three patient populations. None of the 75 AGA patients of group 1 developed postnatal bilirubin values above 17.6 mg/dl, whereas 0.3, 3.4 and 8.6% of the patients in groups 2–4, respectively, did so. The frequency of phototherapy increased from 0% in group 1 up to 9.6% in group 4. For the prediction of further need of phototherapy using a CBB cut-off level of 1.7 mg/dl, they found a sensitivity of 90% and a negative predictive value of 99.1%, indicating that all patients with CBB values below 1.7 mg/dl (443/1100 or 40.2%) were at a very low risk of developing dangerous hyperbilirubinemia. Similar results were obtained

in SGA children with a sensitivity of 94.1% and a negative predictive value of 98.6%. In comparison to term newborns, they generally found higher bilirubin values in preterms. A total of 6.4% of preterm children developed bilirubin values over 17.6 mg/dl, compared with 3% of term children, and 47.4% of preterms had to be treated with phototherapy. Predicting the need of phototherapy by using a CBB cut-off level of 1.7 mg/dl revealed a sensitivity of 70.3% and a negative predictive value of 65.6%.[61]

In 2005, Amar Taksande et al., in a study on 200 healthy term neonates with gestation > 37 weeks, in the absence of significant illness or Rh hemolysis cord bilirubin was estimated by micromethod using calorimeterically using green filter with 540nm wavelength. Neonates were followed up clinically every 12 hrs till discharge and then after 72 hour total serum bilirubin (TSB) level was estimated again.

He concluded that increased cord blood bilirubin can be used as a predictor of the development of neonatal hyperbilirubinemia. Cord bilirubin level of >2mg/dl had the highest sensitivity (89.5%), and this critical bilirubin level had a very high (98.7%) negative predictive value and fairly low (38.6%) positive predictive value.[62]

Rostami et al 2005, on their study to identify healthy newborns at risk for developing significant hyperbilirubinemia by measuring bilirubin level in cord blood in 643 full term infants. Serum bilirubin level was obtained on umbilical cord serum and on day three of age. The total bilirubin ≥ 239μmo1/1 (14 mg/dl) was defined as significant hyperbilirubinemia. Data were analyzed using t-test, chi-square, and receiver operating characteristics (ROC) curve. Result showed mean and standard deviation of cord bilirubin level was 34.2± 15.9 μmol/1 (2.00 ±0.93 mg/dl). There was a statistically significant relation between use of oxytocin and subsequent significant hyperbilirubinemia ($p < 0.04$). 92.4% of neonates with cord bilirubin levels below

51.3 μmol/1 (3 mg/dl) did not develop significant hyperbilirubinemia. A cord serum bilirubin level above 51.3 μmol/1 is not a useful predictor of neonatal jaundice.

They concluded that cord serum bilirubin level cannot identify newborns with subsequent significant hyperbilirubinemia.[63]

Sun et al 2007, Investigated 523 healthy term newborns. The cord blood total serum bilirubin concentration and the serum albumin concentration were determined. The infants were aligned into four groups according to their CBB levels, starting from < 1.7 mg/dl (group 1); ≥1.7 mg/dl (group 2); ≥2.1 mg/dl (group 3); ≥2.4 mg/dl (group4). The frequency of hyperbilirubinemia and phototherapy (PT) were compared among the four groups. An analysis of CBB as a predictor of later development of jaundice was performed. The characteristics of the infants who became jaundiced (jaundiced group) were compared with the normal infants (non-jaundiced group). The frequency of patients with hyperbilirubinemia or phototherapy increased with increasing CBB levels. For the prediction of TCB ≥25 using a UCS bilirubin cut-off level, such as ≥2 mg/dl, a positive predictive value of 45.68% and sensitivity of 68.27% was found. It is significant to predict neonatal jaundice by CBB levels ($P < 0.001$). In the jaundiced group (TCB ≥25) CBB levels were significantly higher than those in the non-jaundiced group ($t = 10.96$, $P < 0.001$). No significant differences were found in the cord blood serum albumin concentration ($t = 2.38$, $P > 0.05$), the gestational age ($t = -0.90$, $P > 0.05$), and birth weight ($t = 0.10$, $P > 0.05$) between the jaundiced and non-jaundiced groups.[64]

In 2009, Rudy Satrya et al., in his prospective observational study on 88 health term newborns, Cord blood was collected for the total bilirubin, conjugated bilirubin, unconjugated bilirubin level measurement and blood group test. Measurements of

total bilirubin, conjugated bilirubin, and unconjugated bilirubin were repeated on the 5th day with serum sampling, or as soon as the newborn appeared to be jaundiced. Subjects were categorized into hyperbilirubinemia and non-hyperbilirubinemia newborns. There was a correlation between cord blood and the 5th day bilirubin level. By ROC analysis, cord blood bilirubin level of ≥2.54 mg/dl was determined to have high sensitivity (90.5%), specificity of 85%, and accuracy of 86.4%. He concluded there is a correlation between cord blood bilirubin level and hyperbilirubinemia in healthy term newborns. Cord blood bilirubin level at or greater than 2.54 mg/dl can predict the development of hyperbilirubinemia.[65]

Zakia Nahar et al 2009 carried a study on the value of umbilical cord blood bilirubin measurement in predicting the development of significant hyperbilirubinemia in healthy newborn. For this purpose 84 healthy newborn infants were enrolled and followed up for first 5 days of life. Study subjects were divided into two groups. Group-I consisted of 71 subjects, who did not develop significant hyperbilirubinemia (bilirubin <17mg/dl); Group-II consisted of 13 newborns, who developed significant hyperbilirubinemia (bilirubin >17mg/dl) during the follow up.

Of the enrolled subjects, 46 (55%) were male and rest 38(45%) were female; 64 (76%) were term babies and 20 (24%) were pre-term babies. Significantly higher percentage of pre-term babies developed hyperbilirubinemia. ROC(receiver operating characteristic) analysis demonstrates that the critical value ofcord blood bilirubin >2.5mg/dl had the high sensitivity (77%) and specificity (98.6%)to predict the newborn who would develop significant hyperbilirubinemia. At this level the negative predictive value was 96% and positive predictive value 91%.[66]

Randew S et al 2010, concluded that First day TSB estimation can serve as a reliable screening test for neonates at risk for subsequent hyperbilirubinemia. Neonates with the first day TSB <6.4 mg/dl have minimum risk of subsequent hyperbilirubinemia.[67]

METHODOLOGY

This study was conducted in R.L.Jalappa Hospital attached to Sri Devaraj Urs Medical College. Eligible healthy term newborns 205 in number born at this hospital during 1-year period (March 2011 to Feb 2012) were prospectively enrolled in the study.

INCLUSION CRITERIA

Healthy term, exclusively breastfed neonates

EXCLUSION CRITERIA

Any known obvious causes which lead to jaundice :

1. Any maternal illness.

2. Any maternal drugs.

3. Any birth injuries or infection.

4. ABO and Rh incompatibility and other hemolytic causes.

METHOD OF COLLECTION OF DATA

The demographic profile and relevant information of individual patient was collected by using structured Proforma by interviewing the mother and an informed consent was obtained. Gestational age was assessed by New Ballard score. CBB estimation and blood grouping and typing was done. Serum Bilirubin estimation was done 0n day 3 of life. All babies were followed up daily for the first five postnatal days.

LABORATORY INVESTIGATION:

1. Two(2) ml each of plain and EDTA cord blood sample was collected and subjected to following investigation

Blood group & type.

Total and direct serum bilirubin.

2. Two(2) ml each of plain and EDTA venous blood samples were collected from the baby on day 3 of life and after five days depending upon the clinical situation. These samples were subjected to following investigation.

Total and direct serum bilirubin & CBC.

Blood sample collected was stored away from light. The sample was refrigerated between 2 -8degree C till serum bilirubin estimation is done. Serum bilirubin estimation was done within 12 hours of collection of sample by Diazotized sulfanilic test.

This method for bilirubin estimation is based on principle that Bilirubin reacts with diazotised sulphanilic acid in acidic medium to form pink coloured azobilirubin with absorbance directly proportional to bilirubin concentration. Direct Bilirubin, being water soluble directly reacts in acidic medium. However indirect or unconjugated Bilirubin is solubilised using a surfactant and then it reacts similar to direct Bilirubin.

The main outcome of the study was inferred in terms of hyperbilirubinemia.

Serum bilirubin >14 mg/dl on day 3 of life (after 48 hrs of life) was taken as hyperbilirubinemia needing phototherapy and treatment is advised to all those full term healthy babies with serum bilirubin level of >14 mg/dl after 48 hours of life, as per the American academy of paediatrics practice parameter, 2004.

IAP-NNF also recommends considering phototherapy with neonatal serum bilirubin levels of >14 mg/dl after 48 hours of life.

Statistical Methods: Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. The following assumptions on

data is made, Assumptions: 1.Dependent variables should be normally distributed, 2.Samples drawn from the population should be random, Cases of the samples should be independent

Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. Pearson correlation between cord blood Bilirubin with Day3 bilirubin is performed to find the relationship.

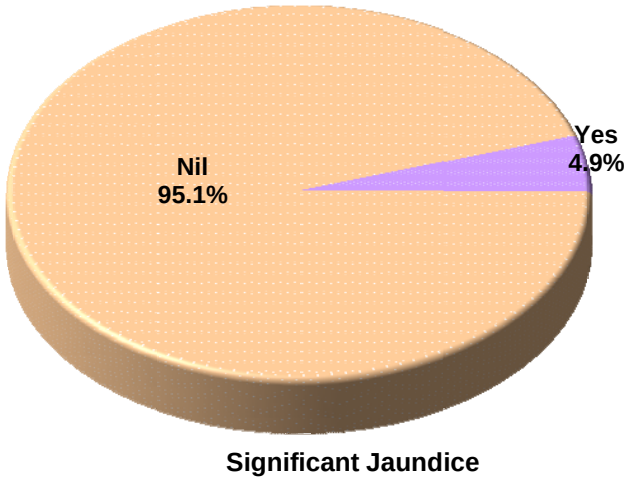
RESULTS

An observational correlation clinical study with 205 neonates was undertaken to study the correlation of cord blood Bilirubin and fetal and maternal outcome and To predict the risk of jaundice, in order to implement early treatment and there by minimize the risk of bilirubin dependent brain damage.

TABLE 1: STUDY POPULATION AND SIGNIFICANT JAUNDICE

Total	Significant Jaundice	
	Number	Percentage
205	10	4.9%

GRAPH 1: STUDY POPULATION AND SIGNIFICANT JAUNDICE



The incidence of significant hyperbilirubinemia in our study population is 4.9 %. Significant jaundice is defined as TSB >14 mg/ dl on day 3 of life

TABLE 2: GENDER DISTRIBUTION OF NEONATES STUDIED

Gender	Number of neonates	%
Male	116	56.6
Female	89	43.4
Total	205	100.0

GRAPH 2: GENDER DISTRIBUTION OF NEONATES STUDIED

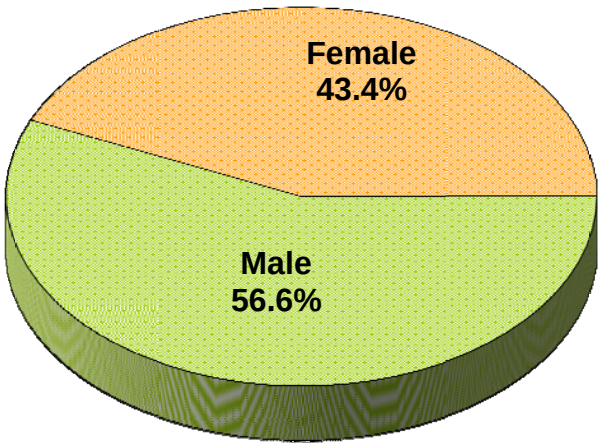
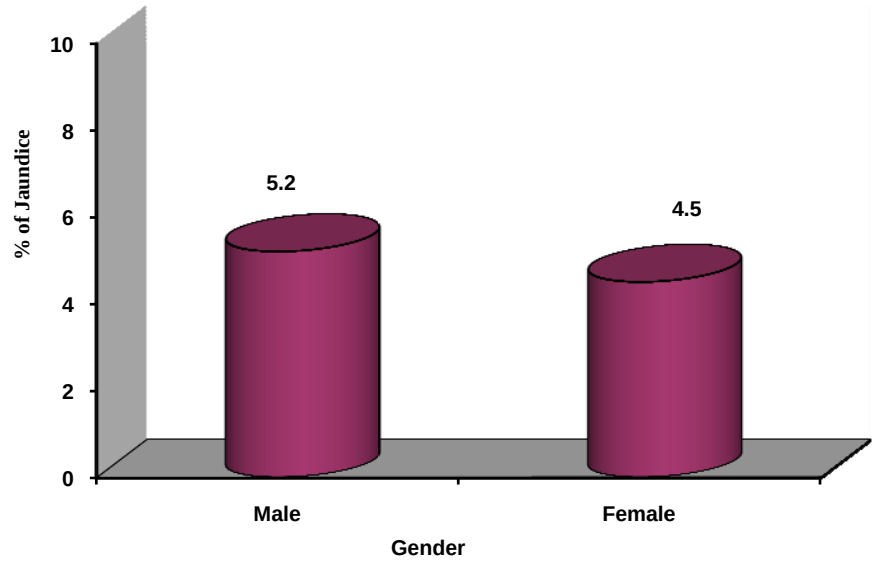


TABLE 3:ASSOCIATION OF GENDER OF NEONATES WITH INCIDENCE OF JAUNDICE

Neonates details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Gender of neonates				
Male	116	6	5.2	0.881
Female	89	4	4.5	0.861

GRAPH 3:ASSOCIATION OF GENDER OF NEONATES WITH INCIDENCE OF JAUNDICE

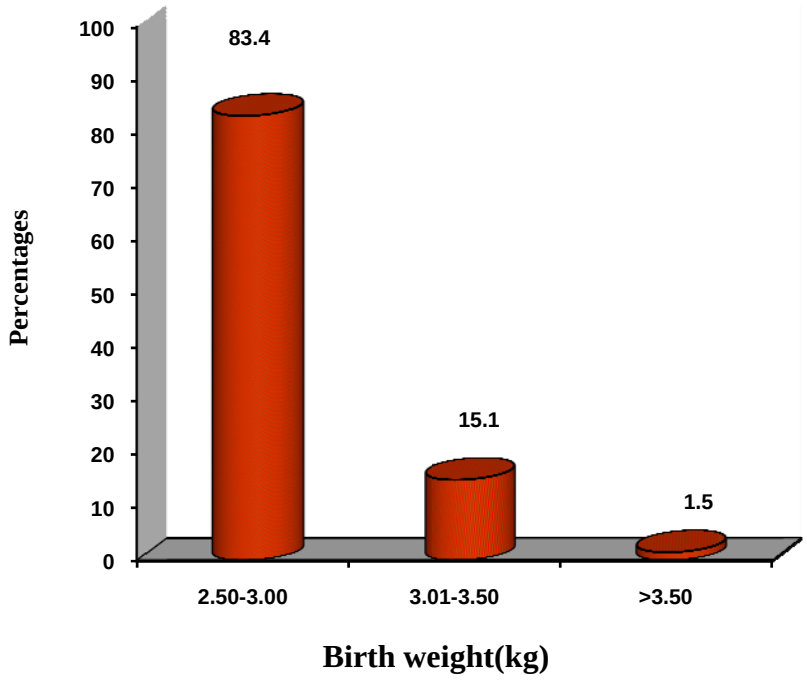


In the present study there is no significant difference in the serum bilirubin level of both the sexes. Hence the present study infers that the serum bilirubin level is independent of the sex of the newborn.

TABLE 4: BIRTH WEIGHT DISTRIBUTION OF NEONATES STUDIED

Birth weight(kg)	Number of neonates	%
2.50-3.00	171	83.4
3.01-3.50	31	15.1
>3.50	3	1.5
Total	205	100.0

GRAPH 4: BIRTH WEIGHT DISTRIBUTION OF NEONATES STUDIED



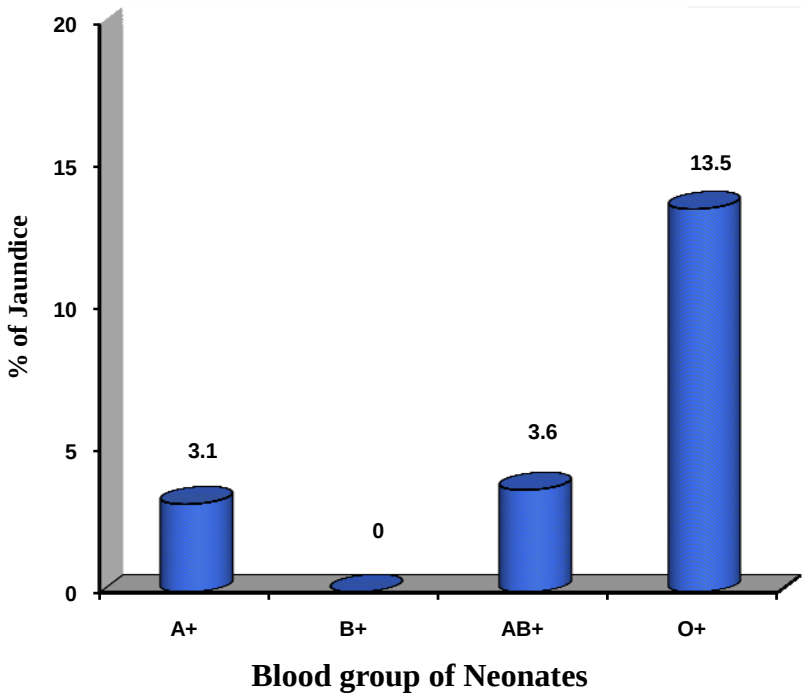
Mean ± SD: 2.86±0.27

The majority of the neonates born, around 83.4% had the birth weight within the range of 2.5-3.0 kg.

TABLE 5: ASSOCIATION OF NEONATE BLOOD GROUP WITH INCIDENCE OF JAUNDICE

Neonate Blood group	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
A+	64	2	3.1	0.505
B+	61	0	0.0	-
AB+	28	1	3.6	0.750
O+	52	7	13.5	0.004**
Total	**205**	**10**	**4.9**	**-**

GRAPH 5: ASSOCIATION OF NEONATE BLOOD GROUP WITH INCIDENCE OF JAUNDICE

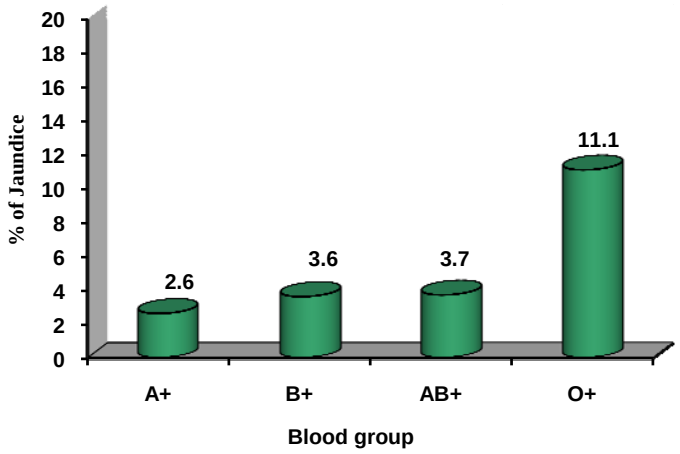


The present study shows significant relation (p value <0.004) between neonatal hyperbilirubinemia and O+ve blood group of the neonates(born to O +ve mother).

TABLE 6 : ASSOCIATION OF MATERNAL BLOOD GROUP WITH INCIDENCE OF JAUNDICE

Mother details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Blood group				
A+	77	2	2.6	0.349
B+	56	2	3.6	0.652
AB+	27	1	3.7	0.77
O+	45	5	11.1	0.054+
Total	205	10	4.9	-

GRAPH 6 : ASSOCIATION OF MATERNAL BLOOD GROUP WITH INCIDENCE OF JAUNDICE

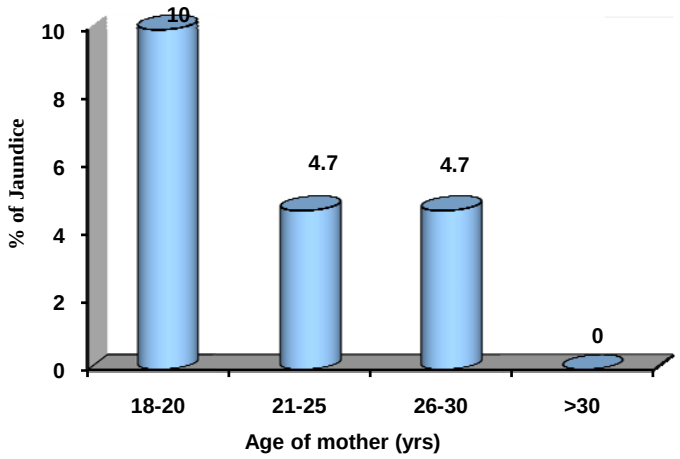


The predominant blood group among mothers was A+ve . There was significant relation between neonatal hyperbilirubinemia and babies(with O+ve blood group) born to O+ve mothers.

TABLE 7 : ASSOCIATION OF MATERNAL AGE WITH INCIDENCE OF JAUNDICE

Mother details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Age of mother (yrs)				
18-20	10	1	10.0	0.455
21-25	86	4	4.7	0.932
26-30	107	5	4.7	0.923
>30	2	0	0.0	-
Total	205	10	4.9	-

GRAPH 7 : ASSOCIATION OF MATERNAL AGE WITH INCIDENCE OF JAUNDICE

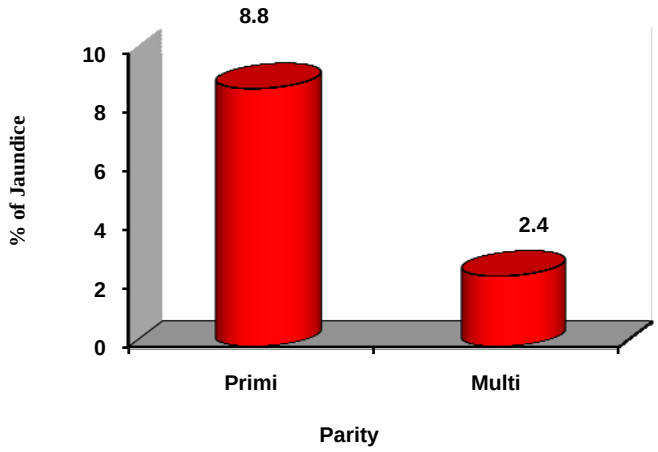


According to age of the mother, the study population was divided into 4 categories. The first category constitutes age group 18-20(4.9%), second category constitutes age group 21-25(42%), third category constitutes age group 26-30(52%), fourthcategory constitutes age group >30(1%). This implies neonatal hyperbilirubinemia is independent of maternal age.

TABLE 8 : ASSOCIATION OF MATERNAL PARITY WITH INCIDENCE OF JAUNDICE

Mother details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Parity				
Primi	80	7	8.8	0.106
Multi	125	3	2.4	0.195
Total	205	10	4.9	-

GRAPH 8 : ASSOCIATION OF MATERNAL PARITY WITH INCIDENCE OF JAUNDICE

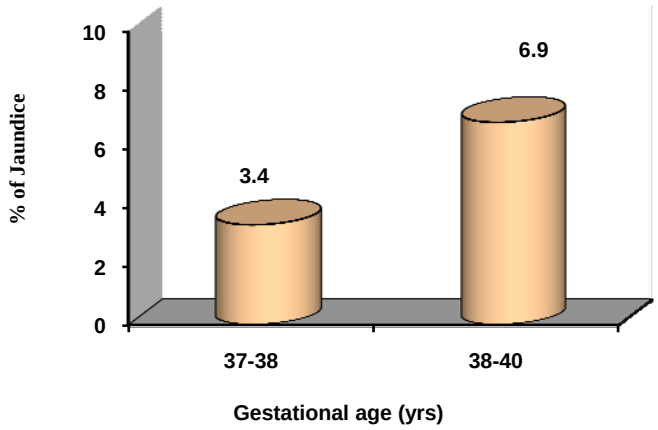


The majority of mothers were multipara accounting to 60%. There was no significant correlation between neonatal hyperbilirubinemia and parity.

TABLE 9 : ASSOCIATION OF MATERNAL GESTATIONAL AGE WITH INCIDENCE OF JAUNDICE

Mother details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Gestational age (yrs)				
37-38	119	4	3.4	0.448
38-40	86	6	6.9	0.390
Total	205	10	4.9	-

GRAPH 9 : ASSOCIATION OF MATERNAL GESTATIONAL AGE WITH INCIDENCE OF JAUNDICE

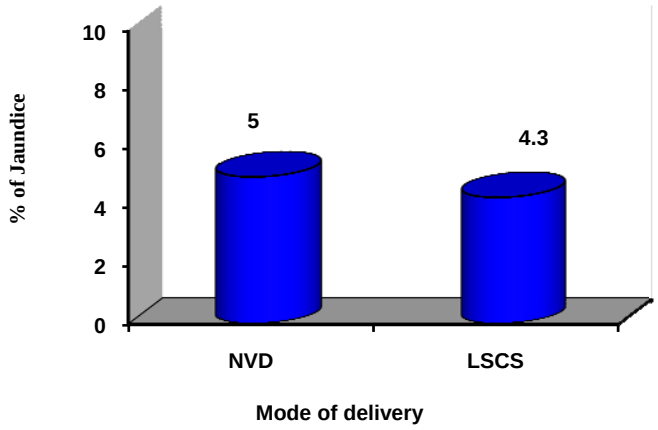


The majority of babies were born at 37-38 weeks of gestation (58%). There was no significant correlation between neonatal hyperbilirubinemia and gestational age.

TABLE 10 : ASSOCIATION OF MODE OF DELIVERY WITH INCIDENCE OF JAUNDICE

Mother details	Number of patients	Incidence of Jaundice (on day 3)	% of Jaundice	P value
Mode of delivery				
NVD	159	8	5.0	0.953
LSCS	46	2	4.3	0.851
Total	205	10	4.9	-

GRAPH 10 : ASSOCIATION OF MODE OF DELIVERY WITH INCIDENCE OF JAUNDICE



The majority of babies were born of normal vaginal delivery (77.6%) . There was no significant correlation between neonatal hyperbilirubinemia and mode of delivery.

TABLE 11 : LEVELS OF BILIRUBIN (CORD BLOOD) OF NEONATES STUDIED

Cord blood : Bilirubin	Number of neonates (n=205)	%	Mean ± SD
Total bilirubin			
<1.0	26	12.7	1.42±0.56
1.0-1.9	168	81.9	
2.0-2.9	5	2.4	
≥3.0	6	2.9	

Mean cord blood bilirubin was 1.42mg/dl.

GRAPH 11 : LEVELS OF BILIRUBIN (CORD BLOOD) OF NEONATES STUDIED

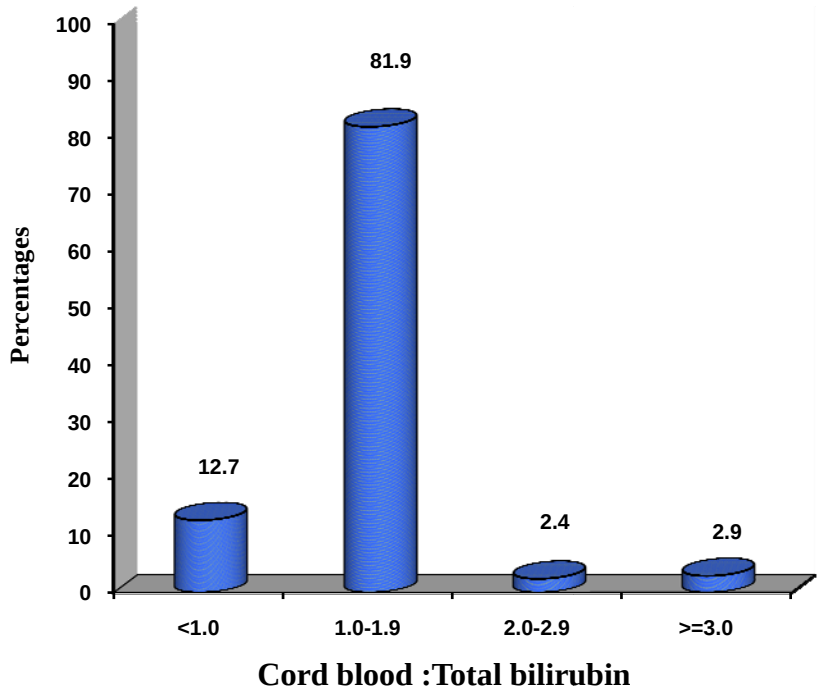


TABLE 12 : LEVELS OF BILIRUBIN (ON DAY 3) OF NEONATES STUDIED

Bilirubin	Number of neonates (n=205)	%	Mean ± SD
Total bilirubin			
8.0-10.0	49	23.9	11.66±2.16
10.0-14.0	146	71.2	
>14.0	10	4.9	

Mean day 3 Total serum bilirubin was 11.66mg/dl.

GRAPH 12 : LEVELS OF BILIRUBIN (ON DAY 3) OF NEONATES STUDIED

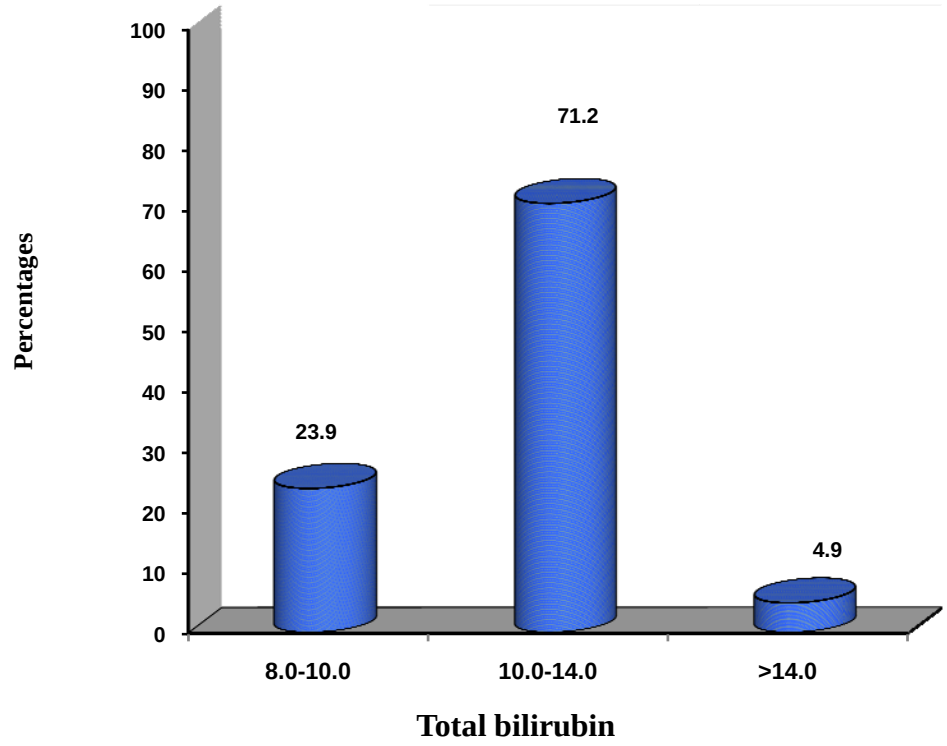
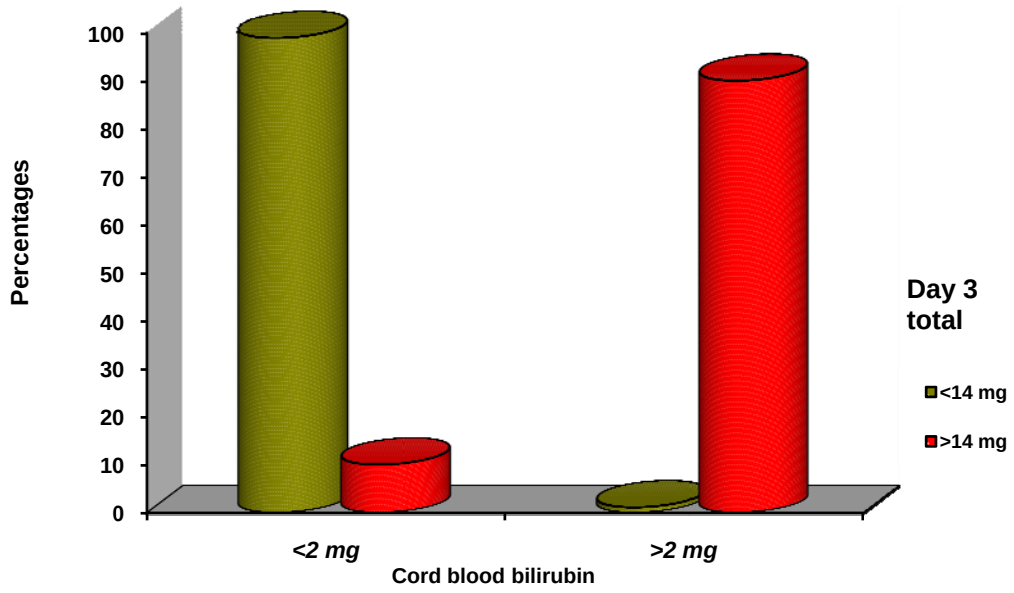


TABLE 13 : CORRELATION OF CORD BLOOD BILIRUBIN WITH DAY3 TOTAL BILIRUBIN

Cord blood Bilirubin	Day 3 total bilirubin		P value
	<14 mg	>14 mg	
<2 mg	193(98.9%)	1(10.0%)	<0.001**
>2 mg	2(1.1%)	9(90.0%)	
Total	195(100.0%)	10(100.0%)	

GRAPH 13: CORRELATION OF CORD BLOOD BILIRUBIN WITH DAY3 TOTAL BILIRUBIN



The present study infers that cord serum bilirubin levels of the babies with neonatal hyperbilirubinemia (>14mg/dl) is significantly higher (CBB>2mg/dl) than the babies without hyperbilirubinemia.

TABLE 14 : CORRELATION OF CORD BLOOD BILIRUBIN WITH DAY3 TOTAL BILIRUBIN

Cord blood Bilirubin	Day 3 total bilirubin		P value
	<14 mg	>14 mg	
<2 mg	193	1	<0.001**
>2 mg	2	9	
True Positive	True Negative	False Positive	False Negative
9	193	2	1
Sensitivity %	Specificity %	PPV %	NPV%
90.00	98.97	81.82	99.48

In the present study probability that a neonate with cord bilirubin higher than 2 mg/dl would later develop hyperbilirubinemia (Positive Predictive Values) was 81% The negative predictive value of the cord bilirubin lower or equal to 2 mg/dl was 99.48%. If a neonate becomes hyperbilirubinemic, the probability that the cord bilirubin was higher than 2 mg/dl was 90% (Sensitivity). The probability that the cord bilirubin was lower or equal to 2 mg/dl was 98.97% (Specificity) in a Non hyperbilirubinemic neonate.

DISCUSSION

Our study hypothesis was that a high serum bilirubin level at birth would also predict a high peak later in life. Our aim was to quantify the relationship between Cord blood bilirubin with peak serum bilirubin levels of the first three days. We chose cord blood estimation for initial serum bilirubin estimation because it is a non invasive way and the results are available within few hours after birth.

The potential risk of developing bilirubin encephalopathy or even kernicterus is high in babies with elevated serum biliruhin level. Kernicterus in new-borns is preventable, provided excessive hyperbilirubinemia for age is promptly identified and appropriately treated.

With the intent to facilitate such identification and treatment, universal screening for severity of bilirubinemia before hospital discharge may predict that extraordinary segment of the neonatal population which is at risk for excessive hyperbilirubinemia during the first week after birth.

Currently we do not have a reliable method of anticipating such levels of hyperbilirubinemia. It is possible that closer, and more frequent, follow up after birth and discharge from the hospital might prevent some of these unfortunate outcomes, but rare, sporadic cases of kernicterus may not be preventable unless we adopt an approach to surveillance of the newborn that is substantially more rigorous than has been practiced. The feasibility, costs, risks and benefits of such an approach need to be determined.

To address this issue AAP recommends that follow up should be provided to all neonates discharged less than 48 hours after birth by a health care professional in

an office, clinic, or at home within 2 to 3 days of discharge. Compliance with this advice may not be easy however, particularly in rural or lower socioeconomic areas, and given the rarity of kernicterus, it will be very difficult, if not possible to document the benefits of this policy.

Umbilical cord blood collection is not associated with any pain. Furthermore, most important is that the data are available immediately after birth. The babies at risk for developing hyperbilirubinemia can be detected at birth in a non invasive way if the neonate leaves the hospital within the first few postnatal days. The use of Cord blood bilirubin values may help to predict infants with low risk for hyperbilirubinemia and minimise an unnecessary prolongation of hospitalization.

Keeping these factors in consideration our study was conducted on term healthy neonates with non-haemolytic jaundice. The outcome was hyperbilirubinemia. We have considered peak serum bilirubin level >14 mg/dl on day 3 of life as significant hyperbilirubinemia since specific treatment is considered at or above this level.

TABLE NO 15 : COMPARISON STUDIES ON THE PREDICTIVE ABILITY OF CORD BLOOD BILIRUBIN LEVEL AND THE NEONATAL HYPERBILIRUBINEMIA.

Studies	Cut off Cord STB (mg/dl)	Cut off neonatal hyperbilirubinemia (mg/dl)	Sensitivity	Specificity	PPV	NPV	P Value
Present	≥2	≥15	90%	98.97%	81.82%	99.48%	<0.001
Knudsen[53] (1989)	≥2.35	≥15	13%	99%	85%	72%	<0.001
Amar Taksande et al[62] (2005)	≥2	≥17	89.5%	85%	38.8%	98.7%	0.0000
Sun et al 64 (2007)	≥2	≥17	68%		45%		<0.001
Zakia Nahar et al[66] (2009)	≥2.5	≥17	77%	98.6%		96%	<0.05
Rudy et al[65](2009)	≥2.54	≥12.9	90.5%	85%			0.001

The incidence of hyperbilirubinemia in the Present study is 4.9%

In the present study probability that a neonate with cord bilirubin higher than 2 mg/dl would later develop hyperbilirubinemia (Positive Predictive Values) was 81% The negative predictive value of the cord bilirubin lower or equal to 2 mg/dl was 99.48%. If a neonate becomes hyperbilirubinemic, the probability that the cord bilirubin was higher than 2 mg/dl was 90% (Sensitivity). The probability that the cord bilirubin was lower or equal to 2 mg/dl was 98.97% (Specificity) in a Non hyperbilirubinemic neonate.

Knudsen[53] (1989), established that if the cord bilirubin was below 20 µmol/l, 2.9% became jaundiced, as opposed to 85% if the cord bilirubin was above 40 µmol/l. Furthermore, 57% of jaundiced infants with cord bilirubin above 40 µmol/l required phototherapy, but only 9% if the cord bilirubin was 40 µmol/l or lower (0.008) in correlation with the present study.

Amar Taksande et al[62] (2005), showed that the cord bilirubin level >2mg/dl has a sensitivity 89.5%, specificity 85%, negative predictive value of 98.7% and positive predictive value of 38.8% in correlation with the present study.

Zakia Nahar et al[66] (2009), showed that the cord bilirubin level ≥2.5mg/dl has a sensitivity 77%, specificity 98.6%, with negative predictive value of 96% in correlation with the present study.

Sun et al[64] (2007), Rudy Satrya et al[65] (2009) studies are in correlation with the present study.

CONCLUSION

Neonatal bilirubinemia is the one of the most common reasons for readmission and at risk for development of kernicterus. Early identification of babies at risk will help in better management of these babies.

In our present study neonates with significant hyperbilirubinemia(>14mg/dl) on day 3 of life had significantly elevated levels of cord blood bilirubin(≥2mg/dl). There was no significant correlation between neonatal hyperbilirubinemia and birth weight or sex of the baby. There was also no significant association between neonatal hyperbilirubinemia and gestational age, parity or mode of delivery. But there was significant relation between neonatal hyperbilirubinemia and babies born to mothers with O+ve blood group.

From our study it can be concluded that cord blood bilirubin estimation is a non invasive and reliable investigation for early prediction of neonatal hyperbilirubinemia. So babies with cord blood bilirubin ≥2mg/dl defines the risk group prone to develop significant hyperbilirubinemia . It also predicts dicision making regarding delay in discharge and frequent follow up for initial postnatal days and implementation of early treatment to minimize the risk of bilirubin dependent brain damage.

SUMMARY

Eligible healthy term newborns, 205 in number born at R.L.Jalappa Hospital attached to Sri Devaraj Urs Medical College, during 1-year period (March 2011 to Feb 2012) were prospectively enrolled in the study.

Healthy term neonates with hospital stay of upto 5 days were selected for the study. Neonates were followed from birth to 5th postnatal day. Informed written consent was taken from the parents. All the babies were followed up daily for the development of jaundice during postnatal visits. Cord blood was collected at birth and bilirubin estimation was done within 12 hours of collection of the blood. 3rd day bilirubin estimation was done. Peripheral venous blood was collected for estimation of bilirubin.

Main outcome of the study was inferred in terms of neonatal hyperbilirubinemia of >14mg/dl as per the American academy of paediatrics practice guidelines 2004 and IAP-NNF recommendations.

There was no significant correlation between neonatal hyperbilirubinemia and birth weight or sex of the baby. There was also no significant association between neonatal hyperbilirubinemia and gestational age, parity or mode of delivery. But there was significant relation between neonatal hyperbilirubinemia and babies(with O+ve blood group) born to mothers with O+ve blood group.

In the present study a neonate with Cord blood bilirubin level of $\geq$ 2mg/dl has a sensitivity of 90% and specificity of 98.97%, positive predictive value 81% and negative predictive value of 99.48% in predicting the risk of neonatal hyperbilirubinemia.

It is recommended to have cord blood bilirubin estimation of all healthy term babies delivered in an institution to prevent the dangerous consequences of neonatal hyperbilirubinemia like Kernicterus. This can reduce the morbidity and mortality due to hyperbilirubinemia.

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PROFORMA

A PROSPECTIVE STUDY OF CORD BLOOD BILIRUBIN IN HEALTHY TERM NEWBORN AS AN EARLY PREDICTOR OF NEONATAL HYPERBILIRUBINEMIA.

Name of mother : Age of mother :

Address :

Blood Group :

Obstretic score

Gestational age (weeks): LMP : EDD :

Mode of Delivery:

The Baby:

Name: I.P.No. : Sex:

DOB : TOB :

APGAR SCORE 1min: 5min:

Birth Weight (gm/kg) : Blood Group :

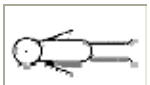
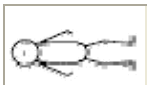
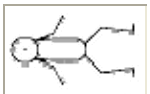
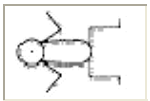
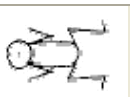
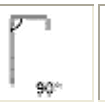
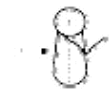
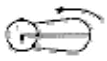
Cord blood bilirubin levels _____ mg/dl

Serum bilirubin level at third day of life _____mg/dl

(If done early specify the age in hours).

- Risk Factors:
- 1. Maternal illness Yes/No
- 2. Maternal drugs Yes/No
- 3. Family history of haemolytic diseases.
- 4. Birth injuries Yes/No

NEUROMUSCULAR MATURITY

SIGN	SCORE							SIGN SCORE
	-1	0	1	2	3	4	5	
Posture								
Square Window								
Arm Recoil								
Popliteal Angle								
Scarf Sign								
Heel To Ear								
TOTAL NEUROMUSCULAR SCORE								

PHYSICAL MATURITY

SIGN	SCORE							SIGN SCORE
	-1	0	1	2	3	4	5	
Skin	Sticky, friable, transparent	gelatinous, red, translucent	smooth pink, visible veins	superficial peeling &/or rash, few veins	cracking, pale areas, rare veins	parchment, deep cracking, no vessels	leathery, cracked, wrinkled	
Lanugo	none	sparse	abundant	thinning	bald areas	mostly bald		
Plantar Surface	heel-toe 40-50mm: -1 <40mm: -2	>50 mm no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases over entire sole		
Breast	imperceptable	barely perceptable	flat areola no bud	stippled areola 1-2 mm bud	raised areola 3-4 mm bud	full areola 5-10 mm bud		
Eye / Ear	lids fused loosely: -1 tightly: -2	lids open pinna flat stays folded	sl. curved pinna; soft; slow recoil	well-curved pinna; soft but ready recoil	formed & firm instant recoil	thick cartilage ear stiff		
Genitals (Male)	scrotum flat, smooth	scrotum empty, faint rugae	testes in upper canal, rare rugae	testes descending, few rugae	testes down, good rugae	testes pendulous, deep rugae		
Genitals (Female)	clitoris prominent & labia flat	prominent clitoris & small labia minora	prominent clitoris & enlarging minora	majora&minora equally prominent	majora large, minora small	majora cover clitoris &minora		
TOTAL PHYSICAL MATURITY SCORE								

MATURITY RATING

TOTAL SCORE (NEUROMUSCULAR + PHYSICAL)	WEEKS
-10	20
-5	22
0	24
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

KEY TO MASTER CHART

1. DOB – Date Of Birth
2. TOB – Time Of Birth
3. TB – Total Bilirubin
4. DB – Direct Bilirubin
5. PT – Phototherapy
6. NVD – Normal Vaginal Delivery
7. LSCS – Lower Segment Caesarean Section

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
1	B/O Savtha	680027	1/3/2011	8.20 AM	M	7:10	9:10	2.70	B+	1.5	0.5	14	1.2		B+	24	G1	38	NVD	0
2	B/O Aruna	681066	1/3/2011	11.45AM	M	7:10	9:10	3.50	A+	1.3	0.4	11	0.4		B+	21	G1	37	LSCS	0
3	B/O Usha	681331	3/3/2011	3.25AM	F	8:10	9:10	2.60	AB+	1.6	0.4	11	0.6		B+	25	G2P1L1	38	NVD	0
4	B/O Sultana	681630	4/3/2011	1.45AM	F	8:10	9:10	2.50	O+	1.4	0.5	14	1.1		O+	23	G3P1L1A1	38	NVD	0
5	B/O Mamatha	682283	6/3/2011	3.30PM	F	7:10	9:10	3.30	A+	1.2	0.4	11	0.8		A+	29	G1	38	NVD	0
6	B/O Lakshmi	682971	8/3/2011	4.10PM	M	7:10	9:10	2.50	O+	1.6	0.7	13	1		O+	25	G3P2L1D1	37	NVD	0
7	B/O Puspa	682997	8/3/2011	11.05PM	M	7:10	9:10	3.50	B+	1.6	0.4	10	0.8		B+	21	G1	38	LSCS	0
8	B/O Sumathi	683439	10/3/2011	7.45AM	M	7:10	9:10	2.70	B+	1.1	0.5	14	0.7		B+	26	G3P2L2	39	LSCS	0
9	B/O Sowmya	683992	11/3/2011	10.15AM	F	7:10	9:10	3.50	AB+	2.1	0.6	12	0.6		B+	29	G2P1L1	39	LSCS	0
10	B/O Deepa	685389	18/3/2011	8.25AM	M	7:10	9:10	2.75	B+	1.5	0.4	9	0.4		B+	22	G1	38	NVD	0
11	**B/O Ramya**	**686654**	**21/3/2011**	**10.50AM**	**M**	**8:10**	**9:10**	**2.50**	**O+**	**2.5**	**0.8**	**19**	**1.2**	**PT**	**O+**	**22**	**G1**	**39**	**NVD**	**0**
12	B/O Saraswati	687818	26/3/2011	12.05AM	F	7:10	9:10	2.70	AB+	0.8	0.4	9	0.7		B+	22	G1	39	NVD	0
13	B/O Lavanaya	689119	8/4/2011	9.50AM	F	7:10	9:10	3.50	A+	1	0.5	14	0.7		A+	31	G3P1L1A1	40	LSCS	0
14	B/O Srimathi	691788	11/412011	5.55AM	F	7:10	9:10	2.70	A+	1.1	0.5	11	0.8		A+	25	G2P1L1	40	NVD	0
15	B/O Prema	692115	12/4/2011	4.30AM	F	8:10	9:10	2.75	A+	1.8	0.4	14	0.6		A+	25	G2P1L1	38	NVD	0
16	B/O Sabana	693477	16/4/2011	3.25PM	M	7:10	9:10	3.30	B+	1.4	0.4	8	0.6		B+	20	G3P2L1D1	38	NVD	0
17	**B/O Pareneeta**	**693510**	**18/4/2011**	**7.20AM**	**F**	**7:10**	**8:10**	**3.00**	**AB+**	**3.3**	**0.4**	**19**	**1**	**PT**	**B+**	**27**	**GI**	**37**	**NVD**	**0**
18	B/O Rajalakshmi	693581	18/4/2011	12.35PM	F	8:10	9:10	2.80	B+	1.5	0.4	10	0.4		A+	25	G2P1L1	39	LSCS	**0**
19	B/O Manjula	693610	18/4/2011	6.40PM	F	8:10	9:10	3.50	O+	1.2	0.6	14	1		O+	27	G3P2L1D1	39	NVD	0
20	B/O Sunanda	693811	18/4/2011	7.40PM	M	8:10	9:10	2.75	O+	1.1	0.4	9.4	0.5		O+	28	G2P1L1	40	NVD	0
21	B/O Vijayalakshmi	694839	21/4/2011	6.15AM	F	8:10	9:10	2.70	A+	1.6	0.4	13	0.7		A+	25	G1	40	NVD	0
22	B/O Shashi	694889	22/4/2011	8.45PM	M	7:10	9:10	3.50	B+	1.1	0.7	12	0.7		B+	20	G2A1	40	NVD	0
23	B/O Sarvatunisa	695469	26/4/2011	12.30PM	M	7:10	9:10	2.50	B+	0.9	0.1	12	0.5		B+	28	G2PIL1	38	LSCS	0
24	**B/O Roopa**	**697349**	**1/5/2011**	**11.55AM**	**M**	**7:10**	**9:10**	**2.60**	**O+**	**2.2**	**0.4**	**17**	**1.2**	**PT**	**O+**	**23**	**G2A1**	**38**	**NVD**	**0**
25	B/O Nandini	697670	5/5/2011	11.10AM	F	7:10	9:10	3.15	B+	1.6	0.5	11	0.7		AB+	23	G2P1L1	40	LSCS	0
26	B/O Reshma	698686	6/5/2011	6.30AM	M	7:10	9:10	2.80	AB+	0.8	0.4	8	0.7		B+	24	G1	37	NVD	0
27	B/O Roopa	699699	9/5/2011	6.45AM	M	7:10	9:10	3.00	O+	1.1	0.4	12	0.4		O+	25	G2P1L1	40	NVD	0
28	B/O Sunita	700654	13/5/2011	1.20AM	M	7:10	9:10	3.25	AB+	1.3	0.6	13	0.6		B+	19	G1	39	NVD	0
29	B/O Geetha	701410	14/5/2011	3.40AM	M	7:10	9:10	3.00	O+	1.4	O.2	11	0.6		O+	21	G1	38	NVD	0
30	B/O Hemavathi	703591	24/5/2011	6.30AM	M	7:10	9:10	2.75	O+	1.2	0.4	12	0.6		O+	20	G1	38	LSCS	0
31	B/O Manjula	705816	1/6/2011	1.30PM	F	8:10	9:10	3.20	AB+	1.1	0.4	13	0.6		B+	19	G1	39	NVD	0
32	**B/O Azzara Begum**	**706677**	**2/6/2011**	**3.00PM**	**M**	**8:10**	**9:10**	**3.20**	**O+**	**3.8**	**1.5**	**20**	**1.3**	**PT**	**O+**	**28**	**G3P1L1A1**	**39**	**LSCS**	**0**
33	B/O Nalini	706948	4/6/2011	9.10PM	F	7:10	9:10	3.15	O+	1.8	0.6	16	0.6		O+	18	G1	40	NVD	0
34	B/O Mangamma	706278	5/6/2011	2.30AM	M	7:10	9:10	2.80	A+	1.8	0.6	9	0.4		B+	24	G2P1L1	38	NVD	0
35	B/O Richa singh	707296	6/6/2011	1.30PM	F	7:10	9:10	2.70	B+	1.2	0.4	11	0.8		AB+	27	G2P1L1	38	NVD	0

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
36	B/O Priya	709269	13/6/2011	1.15AM	F	7:10	9:10	3.00	AB+	1.5	0.5	12	0.8		A+	24	G1	38	NVD	0
37	B/O Anitha	709319	14/6/2011	4.10AM	F	7:10	9:10	3.00	B+	1.5	0.4	11	0.4		A+	24	G1	38	NVD	0
38	B/O Shashikala	709919	15/6/2011	9.00AM	F	7:10	9:10	2.70	B+	1.5	0.7	10	0.5		B+	26	G2P1L1	37	LSCS	0
39	B/O Shaziya	710227	16/6/2011	5.30AM	F	7:10	9:10	2.60	O+	1.9	0.5	13	0.6		O+	28	G2P1L1	38	NVD	0
40	B/O Uma Devi	710243	17/6/2011	7.15PM	M	7:10	9:10	2.80	A+	0.3	0.9	11	0.8		B+	30	G3P2L2	38	NVD	0
41	B/O Manjula	710833	18/6/2011	11AM	M	7:10	9:10	2.90	A+	1	O.4	11	0.6		A+	24	G1	38	NVD	0
42	B/O Narayanamma	710774	18/6/2011	10.15PM	M	7:10	9:10	3.00	O+	1.2	0.4	9	0.5		O+	25	G1	38	NVD	0
43	B/O Pavithra	710554	21/6/2011	2.10PM	M	7:10	9:10	2.60	A+	1.5	0.5	11	0.8		A+	27	G2P1L1	39	NVD	0
44	B/O Navaneetha	711507	22/6/2011	12.40PM	M	8:10	9:10	2.60	O+	1.8	0.6	11	0.8		O+	28	G2P2L2	38	NVD	0
45	B/O Sandya	712342	24/6/2011	8.30PM	M	7:10	9:10	3.00	AB+	1.8	0.6	12	0.6		a+	24	G1	39	NVD	0
46	B/O Swetha	712593	25/6/2011	11PM	M	7:10	9:10	2.60	A+	1.8	0.8	13	0.5		B+	24	G1	39	NVD	0
47	B/O Nikitha	712636	26/6/2011	2AM	F	7:10	9:10	2.60	O+	1.9	0.8	11	0.5		O+	28	G3P2L2	38	LSCS	0
48	B/O S udha	713161	28/6/2011	3.15AM	F	8:10	9:10	2.80	A+	1	0.5	12	0.6		AB+	26	G2P1L1	38	LSCS	0
49	B/O Faheen	713579	29/6/2011	11.30PM	M	7:10	9:10	2.80	B+	0.9	0.3	10	0.4		AB+	28	G2P1L1	38	NVD	0
50	B/O Sampangiyamma	714229	1/7/2011	5.45AM	M	7:10	8:10	3.00	B+	1.8	0.8	13	0.5		AB+	30	G3P2L2	38	NVD	0
51	B/O Mubarak	714240	1/7/2011	7.30AM	F	7:10	9:10	3.00	O+	1.5	0.8	12	0.5		O+	22	G1	39	NVD	**0**
52	B/O Sudharani	714241	1/7/2011	12.10AM	F	8:10	9:10	3.00	B+	1.1	0.4	11	0.8		B+	28	G2P1L1	39	LSCS	0
53	B/O Sulochana	714627	3/7/2011	2.35PM	M	8:10	9:10	3.00	B+	0.9	0.4	11	0.8		A+	29	G2P1L1	38	LSCS	0
54	B/O Sumithra	713660	3/7/2011	10.10PM	F	8:10	9:10	2.90	O+	1	0.6	10	0.6		O+	29	G2P1L1	40	LSCS	0
55	B/O Nagaveni	715362	4/7/2011	2.20AM	M	7:10	9:10	2.70	A+	1	0.5	9	0.5		AB+	28	G3P2L2	40	NVD	0
56	B/O Lalitha	714646	4/7/2011	5.40 PM	F	7:10	9:10	2.60	AB+	1.5	0.4	13	0.6		A+	21	G1	38	NVD	0
57	B/O Vinutha	715442	4/7/2011	8PM	M	7:10	9:10	2.60	B+	1.2	0.8	12	0.4		AB+	32	G2P1L1	38	LSCS	0
58	B/O Sumithramma	714927	4/7/2011	10.10PM	F	7:10	9:10	2.70	A+	0.9	0.4	11	0.3		A+	27	G2P1L1	39	NVD	0
59	B/O Bhargavi	715062	5/7/2011	5.10AM	M	7:10	9:10	2.60	A+	1.6	0.6	12	0.6		B+	26	G2P1L1	38	NVD	0
60	B/O Rukhmani	717294	12/7/2011	1.50AM	M	7:10	9:10	2.60	B+	1.1	0.8	11	1.1		AB+	28	G2P1L1	38	NVD	0
61	B/O Swarna	717043	12/7/2011	4.25pm	F	7:10	9:10	3.50	AB+	1.8	0.6	13	0.6		A+	29	G2P1L1	38	NVD	0
62	B/O Bhagyamma	718200	15/7/2011	1.20AM	M	7:10	9:10	3.00	AB+	1.5	0,8	13	0.6		A+	30	G2P2L2	38	NVD	0
63	B/O Saritha	703591	16/7/2011	8.15PM	M	7:10	9:10	2.60	B+	1.2	0.6	14	0.8		B+	24	G1	39	NVD	0
64	B/O Prema	718248	18/7/2011	11.10AM	M	8:10	9:10	2.60	O+	1	0.4	9	0.6		O+	28	G2A1L0	39	NVD	0
65	B/O Ramya	719248	20/7/2011	8PM	M	8:10	9:10	2.60	A+	1.1	0.4	13	0.5		A+	29	G2P1L1	38	NVD	0
66	B/O Suma	719888	21/7/2011	10.40PM	F	7:10	9:10	3.30	O+	1.2	0.4	13	0.6		O+	25	G1	39	LSCS	0
67	B/O Bhaghya	718831	21/7/11	7.10 PM	F	7:10	9:10	2.60	AB+	0.8	0.4	9	0.5		A+	30	G2P1L1	38	NVD	0
68	B/O CHIATRA	720148	22/7/11	7.30 AM	M	7:10	9:10	2.60	B+	1	0.5	9	0.8		AB+	23	G1	38	NVD	0
69	B/O SUMALATHA	720202	23/7/11	6.40 AM	M	7:10	9:10	3.00	O+	1.2	0.5	13	0.5		O+	26	G2P2L1	39	LSCS	0
70	B/O SASHIKALA	721947	28/7/11	8.10 PM	F	7:10	9:10	3.50	B+	0.8	0.4	11	0.5		B+	25	G1	38	NVD	0

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
71	B/O ANITHA	722242	29/7/11	2.00 PM	M	7:10	9:10	2.90	O+	1.2	0.8	13	0.5		O+	25	G1	39	LSCS	0
72	B/O VEENA	722511	29/7/11	6.30 PM	F	7:10	9:10	3.00	O+	1.3	0.6	13	0.6		O+	26	G2A1L0	38	NVD	0
73	YESHODA	722500	30/7/11	1.40 AM	M	7:10	9:10	2.60	A+	1.2	0.5	11	0.6		B+	24	G1	38	NVD	0
74	B/O BHARTHI	722599	1/8/2011	2.00 AM	M	7:10	9:10	2.60	A+	1.9	0.4	11	0.6		A+	26	G2P1L1	39	NVD	0
75	B/O SUJATHAMMA	722943	1/8/2011	10.30 AM	F	7:10	9:10	3.00	A+	1.8	0.8	13	0.8		A+	30	G3P1L1A1	39	LSCS	0
76	B/O NASEEN TAJ	723301	2/8/2011	8.20 AM	M	7:10	9:10	2.90	B+	1.9	0.3	13	0.8		B+	28	G2P1L1	38	LSCS	0
77	B/O SHALEENI	723699	4/8/2011	2.20 AM	F	7:10	9:10	2.80	B+	1.8	0.4	11	0.8		A+	30	G2P1L1	38	NVD	0
78	B/O ANITHA	724306	5/8/2011	12.30 AM	M	7:10	9:10	2.80	A+	1	0.4	11	0.5		A+	28	G2P1L1	39	NVD	0
79	B/O SHIVAMMA	725309	10/8/2011	6.10 AM	F	7:10	9:10	2.60	A+	1.8	0.3	10	0.6		AB+	28	G3P2L2	38	NVD	0
80	B/O LALITHA	725527	10/8/2011	10.00 PM	F	7:10	9:10	2.60	B+	1.5	0.5	11	0.8		B+	26	G2P1L1	38	LSCS	0
81	B/O Amaravathy	726056	12/8/2011	3.20PM	F	7:10	9:10	2.60	B+	1.2	0.5	9	0.6		A+	24	G1	39	NVD	0
82	B/O Indrani	726429	14/8/2011	11.10AM	M	7:10	9:10	2.90	A+	1.2	0.4	11	0.4		B+	29	G2P1L1	38	NVD	0
83	B/O Gayathri	726457	15/8/2011	9.50AM	M	7:10	9:10	2.80	A+	0.8	0.4	9	0.5		A+	29	G2P1L1	39	LSCS	0
84	B/O Nethravathy	727314	17/8/2011	7.25AM	F	7:10	9:10	2.90	O+	1.1	0.5	11	0.8		O+	28	G2P1L1	39	NVD	0
85	B/O Sameen taj	727875	19/8/2011	7.50PM	M	7:10	9:10	2.80	B+	1.8	0.8	11	0.8		AB+	26	G2P1L1	39	NVD	0
86	B/O Bibi ayesha	727929	19/8/2011	9.30PM	M	7:10	9:10	3.00	O+	0.9	0.4	10	0.8		O+	24	G1	39	NVD	0
87	B/O Mamatha	727017	21/8/2011	5.10PM	M	7:10	9:10	2.90	A+	1.2	0.5	13	0.8		AB+	22	G1	39	NVD	0
88	B/Osheela	728961	22/8/2011	7.15PM	M	7:10	9:10	2.60	B+	1.3	0.5	13	0.6		B+	23	G1	39	NVD	0
89	B/O Veena	728350	22/8/2011	9.45pm	M	7:10	9:10	3.00	A+	1.6	0.4	9	0.5		A+	23	G1	39	NVD	0
90	B/O Eshwaramma	729355	25/8/2011	2AM	F	7:10	9:10	2.75	AB+	1.1	0.3	12	0.6		A+	29	G3P2L2	38	NVD	0
91	B/O Shaziyakanum	729885	26/8/2011	6.40AM	F	7:10	9:10	3.00	B+	0.9	0.4	11	0.4		B+	29	G2P1L1	38	NVD	0
92	B/O Vanitha	729839	27/8/2011	3.40PM	M	7:10	9:10	3.10	B+	0.8	0.4	13	0.5		A+	28	G2P1L1	39	LSCS	0
93	B/O Kavitha	731578	2/9/2011	6AM	M	7:10	9:10	2.70	B+	1.9	0.6	8	0.6		B+	26	G2P1L1	38	NVD	0
94	B/O Munirathnamma	732769	6/9/2011	1.10AM	F	7:10	9:10	2.67	O+	0.8	0.6	11	0.8		O+	29	G3P2L2	38	NVD	0
95	B/O Sujatha	733174	7/9/2011	3.30PM	M	7:10	9:10	3.00	B+	1.5	0.4	8	0.6		AB+	22	G1	40	NVD	0
96	B/O Rukminiyamma	734149	11/9/2011	5.30PM	F	7:10	9:10	2.60	O+	1.8	0.6	12	0.4		O+	29	G3P2L2	38	NVD	0
97	B/O Huzna Taj	735262	14/9/2011	8.10PM	M	7:10	8:10	2.80	B+	1.5	0.6	11	0.3		A+	29	G3P2L2	38	NVD	0
98	B/O Ammu	736364	18/9/2011	6.30PM	M	7:10	8:10	2.80	O+	1.8	0.5	13	0.6		O+	22	G1	39	LSCS	0
99	B/O Sharda	736420	19/9/2011	3.05AM	F	7:10	8:10	2.80	B+	1.2	0.8	13	0.4		AB+	24	G2P1L1	38	NVD	0
100	B/O Savitha	738122	22/9/2011	8.20PM	M	7:10	8:10	2.60	B+	1.9	0.4	12	0.6		AB+	21	G1	40	LSCS	0
101	B/O Shamala	738162	23/9/2011	3.20AM	M	7:10	8:10	2.65	A+	1	0.3	8	0.4		A+	24	G2P1L1	38	NVD	0
102	B/O Prema	738146	23/9/2011	4.50AM	M	7:10	8:10	2.70	A+	1.4	0.4	12	0.6		A+	27	G2P1L1	38	LSCS	0
103	B/O Shahtaz	737888	23/9/2011	1.10AM	F	7:10	8:10	2.60	B+	1.6	0.6	9	0.8		B+	29	G3P1L1A1	38	NVD	0
104	B/O Manjula	737881	23/9/2011	8.20PM	M	7:10	8:10	3.25	A+	1.5	0.3	11	0.6		A+	28	G1	39	NVD	0
105	B/O Manjula	737889	23/9/2011	9.40PM	F	7:10	8:10	3.30	A+	1.8	0.6	13	0.8		AB+	22	G1	39	NVD	0

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
106	**B/O Sowmya**	**738221**	**24/9/2011**	**5.30AM**	**F**	**7:10**	**8:10**	**2.60**	**O+**	**3.5**	**1.2**	**19**	**0.8**	**PT**	**O+**	**21**	**G1**	**38**	**NVD**	**0**
107	B/O Shabreen Taj	738461	24/9/2011	2.15PM	F	7:10	8:10	2.60	O+	1.7	0.8	11	0.5		O+	29	G3P1A1L1	38	LSCS	0
108	B/O Taseena	738549	24/9/2011	4.35PM	F	7:10	8:10	2.55	O+	1.2	0.8	13	0.4		O+	30	G2P1L1	38	NVD	0
109	B/O Kruti	738602	25/9/2011	8.25AM	F	7:10	8:10	2.60	A+	1.4	0.8	13	0.6		A+	23	G1	38	NVD	0
110	B/O Pushpa	738523	26/9/2011	6.50AM	M	7:10	8:10	2.60	AB+	1.1	0.6	13	0.8		A+	24	G2P1L1	38	NVD	0
111	B/O Shilpa	739921	29/9/2011	5.10AM	F	7:10	8:10	2.70	B+	1.8	0.8	11	0.5		B+	28	G1	39	NVD	0
112	B/O Vanajakshi	740561	1/10/2011	1.40AM	M	7:10	8:10	2.80	AB+	1	0.5	8	0.4		B+	30	G3P2L2	37	NVD	0
113	B/O Rathnamma	740585	1/10/2011	10.35PM	F	7:10	8:10	3.00	A+	1.1	0.4	11	0.8		A+	28	G2P1L1	38	NVD	0
114	B/O Gangaratna	742410	5/10/2011	4.30AM	M	7:10	8:10	3.20	O+	1.5	0.5	13	0.6		A+	29	G2P1LOD1	38	LSCS	0
115	B/O Usha	741334	5/10/2011	4:35PM	F	7:10	8:10	2.60	A+	1.8	0.6	12	0.5		A+	20	G1	38	NVD	0
116	B/Okanyamma	741566	6/10/2011	7:25PM	F	7:10	8:10	2.90	O+	1.8	0.2	13	0.6		O+	29	G2P1L1	38	LSCS	0
117	B/O Nagaveni	742106	7/10/2011	6.38PM	M	7:10	8:10	3.10	B+	1.8	0.4	12	0.6		AB+	22	G1	39	NVD	0
118	B/O Dhakshayini	742445	8/10/2011	7.10AM	F	7:10	8:10	3.20	O+	1.9	0.8	14	0.6		O+	28	G3P2L2	37	NVD	0
119	B/O Lalitha	742857	10/10/2011	8.50PM	F	7:10	8:10	2.60	A+	1	0.6	11	0.4		A+	25	G2P1L1	38	NVD	0
120	B/O Triveni	742813	10/10/2011	10.30PM	F	7:10	8:10	3.20	AB+	0.8	0.4	10	0.6		A+	26	G2P1L1	38	NVD	0
121	B/O Gayathri	743207	11/10/2011	12.40PM	F	7:10	9:10	3.20	A+	1.9	0.8	13	0.5		B+	28	G2P1L1	38	NVD	0
122	B/O Nandini	743215	12/10/2011	1.40PM	M	7:10	9:10	2.60	A+	0.7	0.4	12	0.5		A+	24	G1	39	NVD	0
123	B/O Sujata	745409	12/10/2011	5.10PM	F	7:10	9:10	2.60	A+	1.4	0.6	11	0.8		A+	26	G2P1L1	38	NVD	0
124	B/O Swayeema	744171	14/10/2011	11.40PM	F	7:10	9:10	2.60	B+	1.1	0.8	13	0.6		B+	30	G2P2L2	38	NVD	0
125	B/O Shabana	744525	16/10/2011	12.45AM	M	7:10	9:10	2.70	A+	1.8	0.6	11	0.9		A+	28	G2P1L1	38	NVD	0
126	B/O Shahnaz Hussain	744592	16/10/2011	9.55PM	M	7:10	9:10	3.40	AB+	1.6	0.4	13	0.7		A+	21	G1	39	NVD	0
127	B/O Thippakka	744919	17/10/2011	12.15AM	M	7:10	9:10	3.20	B+	1.4	0.6	10	0.5		AB+	23	G1	39	NVD	0
128	B/O Anjum	745539	19/10/2011	9.40AM	M	7:10	9:10	2.70	A+	1.4	0.6	14	0.6		B+	24	G1	39	NVD	0
129	B/O Shashikala	744556	19/10/2011	8.20PM	M	7:10	9:10	3.20	AB+	1.1	0.6	11	0.8		B+	26	G1	39	LSCS	0
130	B/O Meena	747104	26/10/2011	6.20AM	M	7:10	9:10	2.80	A+	1.9	0.6	13	0.4		AB+	28	G2P1L1	38	NVD	0
131	B/O Prema	747112	26/10/2011	7.20AM	M	7:10	9:10	2.90	O+	0.9	0.4	11	0.5		A+	28	G2P1L1	39	NVD	0
132	B/O Kavitha	748193	28/10/2011	1.10AM	F	7:10	9:10	2.60	AB+	1.1	0.4	13	0.4		B+	24	G1	39	NVD	0
133	B/O Surekha	747867	28/10/2011	10.10AM	F	7:10	9:10	2.80	A+	1.9	0.7	13	0.4		A+	21	G1	39	NVD	0
134	**B/O Usha**	**747882**	**28/10/2011**	**10.10PM**	**M**	**7:10**	**9:10**	**2.80**	**A+**	**2.8**	**0.9**	**17**	**1.2**	**PT**	**A+**	**27**	**G2P1L1**	**39**	**NVD**	**0**
135	B/O Manjula	748174	29/10/2011	8.35AM	M	7:10	9:10	2.60	O+	1	0.4	10	0.6		O+	30	G3P2L2	38	NVD	0
136	B/O Sharmala Devi	749489	3/11/2011	2.20PM	M	7:10	9:10	3.00	B+	1.5	0.8	11	0.6		A+	22	G1	39	NVD	0
137	B/O Kumudhini	749450	4/11/2011	3.10AM	M	7:10	9:10	2.60	A+	1.1	0.8	13	0.5		AB+	28	G2P1L1	38	LSCS	0
138	B/O Varalakshmi	750165	5/11/2011	4.40AM	M	7:10	9:10	3.50	A+	1.4	0.4	11	0.6		A+	28	G2P1L1	38	LSCS	0
139	B/O Salma Taj	750225	6/11/2011	7.50PM	M	7:10	9:10	2.80	O+	1.9	0.6	13	0.5		O+	22	G1	39	NVD	0
140	B/O Manjula	750845	8/11/2011	7.40PM	M	7:10	9:10	3.00	O+	0.8	0.3	11	0.6		O+	24	G2P1L1	38	NVD	0

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
141	B/O Bharthi	751196	9/11/2011	8.20PM	F	7:10	9:10	2.70	A+	1	0.5	9	0.4		B+	22	G1	39	NVD	0
142	B/O Madhuri	752072	11/11/2011	11.10PM	M	7:10	9:10	3.20	B+	1.4	0.6	13	0.4		AB+	28	G2P1L1	38	NVD	0
143	B/O Sathaya	751853	12/11/2011	1.20AM	M	7:10	9:10	3.00	AB+	0.8	0.4	11	0.4		A+	21	G1	39	NVD	0
144	**B/O Varalakshmi**	**752815**	**16/11/2011**	**7.35PM**	**F**	**7:10**	**9:10**	**2.60**	**O+**	**4.2**	**1**	**19**	**1.2**	**PT**	**A+**	**29**	**G4P3L2D1**	**38**	**NVD**	**0**
145	B/O Manjula	753835	18/11/2011	7.40PM	F	7:10	9:10	2.80	O+	1.1	0.5	11	0.6		O+	21	G1	39	NVD	0
146	B/O Suma	754733	20/11/2011	9.40AM	M	7:10	9:10	2.90	A+	1	0.3	8	0.4		A+	25	G1	39	NVD	0
147	B/O Aruna	755079	22/11/2011	2.40PM	M	7:10	9:10	2.60	O+	1.1	0.5	11	0.4		A+	27	G2P1L1	38	LSCS	0
148	B/O Komalamma	755755	25/11/2011	4.40AM	F	7:10	9:10	3.20	B+	1.8	0.8	13	0.4		AB+	29	G3P2L2	38	NVD	0
149	B/O Chowdamma	796087	26/11/2011	5.10AM	F	7:10	9:10	2.70	A+	1.5	0.4	12	0.6		A+	30	G3P2L2	37	NVD	0
150	**B/O Vinoda**	**765464**	**29/11/2011**	**8.40PM**	**M**	**7:10**	**9:10**	**3.00**	**O+**	**3.9**	**1.4**	**18**	**1.5**	**PT**	**B+**	**25**	**G1**	**39**	**LSCS**	**0**
151	B/O Roja	757539	1/12/2011	12.40AM	F	**7:10**	**9:10**	2.60	A+	0.9	0.4	10	0.6		A+	21	G1	39	NVD	0
152	B/0 Ramadevi	757711	1/12/2011	1PM	M	**7:10**	**9:10**	3.00	B+	1.1	0.4	14	0.6		A+	29	G2P1L1	38	NVD	0
153	B/O Vijaylakshmi	758171	3/12/2011	2.40AM	M	**7:10**	**9:10**	2.50	O+	0.8	0.4	11	0.6		O+	30	G3P2L2	38	NVD	0
154	B/O Rajeshwari	758557	4/12/2011	5.15AM	F	**7:10**	**9:10**	3.80	A+	1	0.4	10	1		B+	25	G2P1L1	38	NVD	0
155	B/O Lakshmi	758507	4/12/2011	3.40pm	F	**7:10**	**9:10**	2.60	B+	1	0.6	8	0.8		B+	28	G3P2L2	38	NVD	0
156	B/O Reshma	759170	6/12/2011	11.10AM	M	**7:10**	**9:10**	3.00	A+	1.4	0.4	11	0.5		A+	22	G1	39	NVD	0
157	B/O Vasundra	759122	6/12/2011	12.10pm	m	**7:10**	**9:10**	3.00	B+	1.9	0.6	12	0.6		AB+	28	G2P1L1	38	LSCS	0
158	B/O Shabina	759517	7/12/2011	6.40AM	F	**7:10**	**9:10**	2.60	A+	1	0.6	11	0.4		A+	29	G2P1L1	38	NVD	0
159	B/O Sudha	795855	8/12/2011	7.10AM	M	**7:10**	**9:10**	3.00	B+	1.1	0.5	11	0.6		B+	25	G2P1L1	38	LSCS	0
160	B/O Reshmi	761706	15/12/2011	4.10AM	F	**7:10**	**9:10**	3.80	B+	1.3	0.6	13	0.5		A+	27	G2P1L1	38	NVD	0
161	B/O Nouheena	762024	16/2/2011/	10.40PM	M	7:10	9:10	2.80	O+	1.4	0.5	12	0.6		O+	28	G2P1L1	38	NVD	0
162	B/O Pankaj	763106	21/12/2011	1.40AM	F	7:10	9:10	3.00	AB+	0.8	0.4	10	0.6		A+	24	G1	39	NVD	0
163	B/O Mangalagowri	763411	21/12/2011	4PM	M	7:10	9:10	2.90	A+	1.4	0.6	11	1		A+	28	G2P1L1	38	NVD	0
164	B/O Kalishma	763710	23/12/2011	1.45AM	F	7:10	9:10	2.70	A+	1	0.6	12	0.8		B+	21	G1	39	NVD	0
165	B/O Chandrika	783708	23/12/2011	3.40PM	M	7:10	9:10	2.90	AB+	1.6	0.7	12	0.8		B+	21	G1	39	NVD	0
166	B/O Manjula	764338	25/12/2011	3.30PM	M	7:10	9:10	2.60	AB+	1	0.4	8	0.6		A+	28	G2P1L1	38	NVD	0
167	B/O Ayesha taj	764343	26/12/2011	5am	M	7:10	9:10	2.70	B+	1.9	0.8	13	0.6		B+	25	G2P1L1	37	LSCS	0
168	B/O Sunitha	764989	28/12/2011	7.10AM	M	7:10	9:10	3.00	B+	1.8	0.6	12	0.5		B+	25	G1	38	NVD	0
169	B/O Divya	765316	28/12/2011	7.20PM	M	7:10	9:10	3.00	0+	1.1	0.6	10	0.8		O+	21	G1	39	LSCS	0
170	B/O Syeda nadiya	766209	1/1/2012	12.45PM	M	7:10	9:10	2.90	A+	1.7	0.7	13	0.8		A+	25	G2P1L1	38	LSCS	0
171	B/O Lakshmidevi	766519	2/1/2012	7.30AM	F	7:10	9:10	3.00	B+	0.9	0.5	11	0.6		A+	21	G1	39	NVD	0
172	B/O Suma	766918	4/1/2012	3.50AM	M	7:10	9:10	3.40	O+	1.8	0.6	13	0.8		O+	24	G2P1L1	38	LSCS	0
173	B/O Malathy	767830	6/1/2012	7.40PM	M	7:10	9:10	3.00	AB+	1.6	0.5	11	0.6		A+	20	G1	39	NVD	0
174	B/O Anusuyamma	767857	7/1/2012	6.30AM	M	7:10	9:10	2.60	B+	1.6	0.6	11	0.5		B+	28	G2P1L1	38	NVD	0
175	B/O Rajalakshmi	768295	8/1/2012	6.25AM	M	7:10	9:10	3.00	O+	1.3	0.6	12	0.5		A+	26	G3P2	38	NVD	0

Sl N0.	Name	Ip No.	DOB	TOB	Sex	Apgar Score		Birth Weight in Kgs	Blood Gorup	Bilirubin				Treatment	Mothers details					
										Cord Bl		Day 3								
						1"	5"			TB	DB	TB	DB		Blood Group	Age	Obs Score	Gest. Age	Delivery Mode	Risk factors
176	B/O Sangeetha	768224	8/1/2012	11.30PM	F	7:10	9:10	3.60	AB+	0.8	0.3	9	0.5		B+	28	G2P1L1	38	NVD	0
177	B/O Manjula	768682	9/1/2012	7.50pm	F	7:10	9:10	2.60	A+	1.1	0.5	9	0.6		A+	23	G2P1L1	39	NVD	0
178	B/O Suma	770083	15/1/2012	12.10AM	F	7:10	9:10	2.70	B+	1.1	0.6	11	0.4		A+	26	G1	39	NVD	0
179	B/O Aruna	770377	16/1/2012	3AM	F	7:10	9:10	2.80	A+	1.1	0.5	10	0.9		A+	27	G2P1L1	38	NVD	0
180	**B/O Sumithra**	**770950**	**17/1/2012**	**7.10AM**	**F**	**7:10**	**9:10**	**3.00**	**B+**	**2.1**	**0.9**	**13**	**0.6**		**B+**	**22**	**G1**	**39**	**NVD**	**0**
181	B/O Thriveni	771635	20/1/2012	10.20AM	F	7:10	9:10	2.70	B+	1	0.4	11	0.8		A+	29	G2P1L1	38	LSCS	0
182	B/O Varalakshmi	771370	20/1/2012	10.35PM	F	7:10	9:10	3.00	B+	1.9	0.6	13	0.6		AB+	29	G2P1L1	38	NVD	0
183	B/O Anitha	773528	20/1/2012	7.10PM	M	7:10	9:10	2.80	A+	1	0.4	8	0.4		B+	28	G3P2L2	38	NVD	0
184	B/O Shoba	772663	24/1/2012	1.40PM	M	7:10	9:10	2.60	A+	1.1	0.5	11	0.6		A+	26	G2P1L1	38	NVD	0
185	B/O Asharani	772645	24/1/2012	4.40PM	M	7:10	9:10	2.90	O+	1	0.4	11	0.8		O+	28	G2P1L1	38	NVD	0
186	B/O Seema	773786	28/1/2012	12.25AM	M	7:10	9:10	3.00	A+	1.4	0.7	9	0.6		A+	24	G1	39	NVD	0
187	B/O Manjula	774650	1/2/2012	3.50AM	F	7:10	9:10	2.80	O+	1.1	0.8	11	0.6		O+	21	G1	39	NVD	0
188	B/O Vijayamma	776176	5/2/2012	4.30AM	F	7:10	9:10	2.90	A+	0.9	0.4	9	0.6		A+	28	G2P1L1	38	LSCS	0
189	B/O Kanthamma	776217	7/2/2012	3.20PM	M	7:10	9:10	3.00	AB+	1	0.5	10	0.7		A+	29	G3P2L2	38	NVD	0
190	B/O Kousar taj	776543	8/2/2012	8.10AM	M	7:10	9:10	2.90	B+	1	0.5	11	0.6		B+	28	G2P1L1	38	NVD	0
191	B/O Prema	776544	8/2/2012	8.40AM	M	7:10	9:10	2.80	B+	1	0.4	8	0.4		AB+	20	G1	39	NVD	0
192	**B/O Deepa**	**777500**	**10/2/2012**	**10.10AM**	**M**	**7:10**	**9:10**	**2.70**	**A+**	**3.8**	**1.1**	**17**	**1.1**	**PT**	**AB+**	**26**	**G1**	**39**	**NVD**	**0**
193	B/Ochaithra	777859	11/2/2012	3.20PM	M	7:10	9:10	2.80	B+	1.9	0.9	13	0.6		B+	26	G1	39	NVD	0
194	B/O Mala	777949	12/2/2012	3.10PM	M	7:10	9:10	2.60	O+	1.5	0.4	13	0.8		O+	23	G1	39	NVD	0
195	B/O Pavithra	778399	14/2/2012	4.15AM	M	7:10	9:10	3.00	B+	1.9	0.8	13	0.9		A+	28	G1	38	NVD	0
196	B/O Reshmabegum	778401	14/2/2012	9.10AM	F	7:10	9:10	2.60	A+	1	0.5	9	0.8		B+	23	G2P1L1	38	LSCS	0
197	B/O Nazeembegum	778675	14/2/2012	2.40PM	F	7:10	9:10	2.60	B+	1.1	0.5	11	0.9		A+	26	G3P2L2	38	NVD	0
198	B/O Kalavathy	778866	14/2/2012	9.10PM	M	7:10	9:10	2.60	A+	1.9	0.9	14	0.6		A+	21	G1	39	NVD	0
199	B/O Raziya	779316	16/2/2012	5.50PM	M	7:10	9:10	2.90	O+	1	0.5	8	0.6		B+	28	G3P2L2	38	NVD	0
200	B/O Padmamma	780183	20/2/2012	10.20PM	M	7:10	9:10	3.00	B+	1.9	0.8	10	0.9		B+	20	G1	39	NVD	0
201	B/O Shalini	780869	23/2/2012	1.30AM	F	7:10	9:10	2.90	O+	1.1	0.5	10	0.5		O+	23	G1	39	NVD	0
202	B/O Amaravathy	780866	23/2/2012	2.30PM	F	7:10	9:10	2.60	A+	1.8	0.4	13	0.8		A+	28	G2P1L1	38	LSCS	0
203	B/O Anitha	781479	25/2/2012	5.20AM	M	7:10	9:10	2.90	A+	1.8	0.8	13	0.6		AB+	21	G1	39	NVD	0
204	B/O Rathnamma	781728	25/2/2012	4.40PM	F	7:10	9:10	2.60	AB+	1	0.8	11	0.6		A+	29	G2P1L1	38	NVD	0
205	B/O Manjula	781817	26/2/2012	2.10AM	F	7:10	9:10	2.60	O+	0.9	0.4	11	0.8		O+	25	G2P1L1	30	NVD	0