A PROSPECTIVE HOSPITAL BASED OBSERVATIONAL STUDY ON ELECTROLYTE CHANGES FOLLOWING PHOTOTHERAPY IN NEONATAL HYPERBILIRUBINEMIA

BY

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In partial fulfilment of the requirement or the degree of

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IN
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Under The Guidance Of

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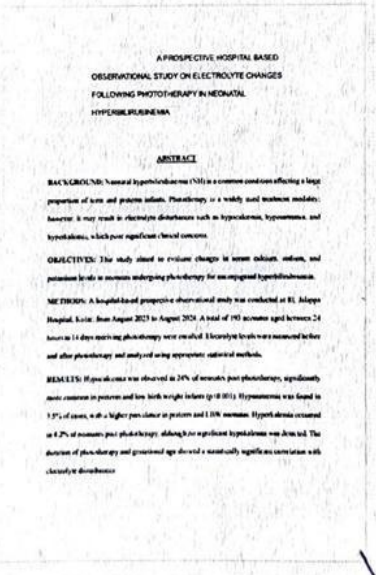

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ABSTRACT

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A PROSPECTIVE HOSPITAL BASED OBSERVATIONAL STUDY ON ELECTROLYTE CHANGES FOLLOWING PHOTOTHERAPY IN NEONATAL HYPERBILIRUBINEMIA ABSTRACT

BACKGROUND: Neonatal hyperbilirubinemia (NH) is a common condition affecting a large proportion of term and preterm infants. Phototherapy is a widely used treatment modality;

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LIST OF ABBREVIATIONS USED

RBCs	-	Red Blood Cells
GALD	-	Gestational Allo-immune Liver Disease
PT	-	Photo Therapy
DAT	-	Direct Antiglobulin Test
ESLD	-	End Stage Liver Disease
FTT	-	Failure To Thrive
GALT	-	Gut Associated Lymphoid Tissue
GGT	-	Gamma Glutamyl Transferase
UTI	-	Urinary Tract Infection
EBF	-	Exclusice Breast Feeding
BMJ	-	Breast Milk Jaundice
NH	-	Neonatal Hyperbilirubinemia
TSB	-	Total Serum Bilirubin
CHB	-	Conjugated Hyperbilirubinemia
UHB	-	Unconjugated Hyperbilirubinemia
HDN	-	Hemolytic Disease of the Newborn
G6PD	-	Glucose-6-Phosphate Dehydrogenase
BIND	-	Bilirubin-Induced Neurological Dysfunction
IVIG	-	Intravenous Immunoglobulin
UGT	-	Uridine Diphosphate Glucuronosyl Transferase

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A PROSPECTIVE HOSPITAL BASED OBSERVATIONAL STUDY ON ELECTROLYTE CHANGES FOLLOWING PHOTOTHERAPY IN NEONATAL HYPERBILIRUBINEMIA

ABSTRACT

BACKGROUND: Neonatal hyperbilirubinemia (NH) is a common condition affecting a large proportion of term and preterm infants. Phototherapy is a widely used treatment modality; however, it may result in electrolyte disturbances such as hypocalcemia, hyponatremia, and hyperkalemia, which pose significant clinical concerns.

OBJECTIVES: This study aimed to evaluate changes in serum calcium, sodium, and potassium levels in neonates undergoing phototherapy for unconjugated hyperbilirubinemia.

METHODS: A hospital-based prospective observational study was conducted at RL Jalappa Hospital, Kolar, from August 2023 to August 2024. A total of 193 neonates aged between 24 hours to 14 days receiving phototherapy were enrolled. Electrolyte levels were measured before and after phototherapy and analyzed using appropriate statistical methods.

RESULTS: Hypocalcemia was observed in 24% of neonates post-phototherapy, significantly more common in preterm and low birth weight infants ($p<0.001$). Hyponatremia was found in 3.5% of cases, with a higher prevalence in preterm and LBW neonates. Hyperkalemia occurred in 4.2% of neonates post-phototherapy, although no significant hypokalemia was detected. The duration of phototherapy and gestational age showed a statistically significant correlation with electrolyte disturbances.

CONCLUSION: Phototherapy in neonates is associated with significant electrolyte imbalances, particularly hypocalcemia and hyponatremia, especially in preterm and low birth weight infants. Routine monitoring of electrolytes during phototherapy is recommended to prevent complications.

KEYWORDS: Neonatal hyperbilirubinemia, phototherapy, hypocalcemia, hyponatremia, electrolyte imbalance, neonates.

INTRODUCTION

INTRODUCTION

Neonatal hyperbilirubinemia (NH) is considered as a more widespread prevalent aberrant physical manifestations in neonates. It affects around half of the neonates who were born at term. It also affects 80.0% of neonates born preterm through the first seven days of life. Elevated levels of unconjugated bilirubin in the blood lead to a yellowish discoloration of the skin , sclera. NH may be healthy or pathological, indicating either hepatic immaturity in bilirubin excretion or excessive bilirubin synthesis. It is the primary reason of readmission for term newborns discharged from the hospital. In the contemporary context of post-delivery hospital discharge, NH is thought to be the primary reason for the readmission of babies within the first seven days of life; it suggests the undeveloped excretory pathway for bilirubin in the liver. For doctors as well as parents, NH is a major concern.

Unconjugated hyperbilirubinemia in neonates left untreated leads to considerable morbidity due to its neurotoxic effects. Untreated, it leads to bilirubin encephalopathy and kernicterus as it has the capacity to traverse the blood-brain barrier. Therefore, early detection and intervention are crucial. Phototherapy presents side effects such as diarrhoea, ocular damage, dehydration, bronze baby syndrome, hyperthermia, and gonadal toxicity. One of the side effects related to electrolyte disturbance is hypocalcaemia. Hypocalcaemia can lead to terrible consequences such as irritability, jitteriness, convulsions, and breathlessness. Consequently, phototherapy-induced hypocalcaemia constitutes a considerable issue. Consequently, it is recommended that calcium administration be investigated for newborns requiring phototherapy.

Few studies have shown additional kinds of electrolyte imbalance in babies under phototherapy for NH treatment. Phototherapy could cause symptomatic hypocalcaemia and hyponatremia, which would need treatment. This study was conducted to investigate electrolyte imbalances in neonates undergoing phototherapy.

OBJECTIVES

OBJECTIVES:

1. To evaluate the alterations in electrolyte levels among neonates undergoing phototherapy for neonatal hyperbilirubinemia, at RL Jalappa Hospital,Kolar.
2. To determine the impact of phototherapy on Serum concentrations of Sodium, Potassium and Calcium .

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Neonatal jaundice is one of the medical indications that's seen as a result of increased total serum bilirubin (TSB), known as NH, which appears due to bilirubin accumulation in a newborn's skin. Neonatal jaundice is illustrated by yellowish pigmentation of the skin, sclerae, and mucous membranes. Jaundice the term, originated in France from "jaune", signifying yellow. Neonatal jaundice is a common clinical ailment during the initial 14 days of life and a significant reason for revisit to hospital post-delivery.[1] It affects around half of the neonates who were born at term. It also affects 80.0% of neonates born preterm through the first seven days of life.[2]

NH can present in two forms - physiological or pathological. There are 2 types of NH: "unconjugated hyperbilirubinemia (UHB) and conjugated hyperbilirubinemia (CHB)."

Once infant jaundice is diagnosed, one must first determine the cause of neonatal hyperbilirubinemia. Most babies with clinical jaundice have unconjugated hyperbilirubinemia as the cause. Ignoring CHB can cause bilirubin encephalopathy as well as other brain problems. Unconjugated bilirubin has bad influence on the CNS more in preterm neonates and those with inherited enzyme abnormalities.[3,4]

A transcutaneous assessing apparatus can be used to assess the bilirubin concentrations. Blood samples as such can also be used to assess bilirubin concentrations in order to detect unconjugated hyperbilirubinemia. Often, laboratory tests—including serum aminotransferase levels, prothrombin time, urine cultures, assessments for inborn metabolic disorders, and, in certain instances, imaging tests—reveal conjugated hyperbilirubinemia. Untreated, severe hyperbilirubinemia might cause bilirubin-induced neurological dysfunction (BIND) as well as acute and chronic bilirubin encephalopathy.[5] The main therapies for UHB are phototherapy exchange transfusions, intravenous immunoglobulin (IVIG).Though hyperbilirubinemia has been diagnosed and treated more effectively, it still poses a major sickness and death risk in newborns.[6]

Aetiology

The fundamental cause of neonatal jaundice is NH, that has two separate types – UHB and CHB, referred to as indirect and direct hyperbilirubinemia, respectively.

Unconjugated Hyperbilirubinemia

UHB is considered the predominant form. It may physiological or pathological. The Physiologic type of jaundice constitutes three-fourth of newborn hyperbilirubinemia and arises as a result of physiological change in newborn bilirubin metabolism. Normal serum bilirubin level less than 1 mg/dL. In newborns, typical levels of S. bilirubin are relatively elevated and vary with age. Term neonates also show a higher bilirubin load because of more RBC count and a shorter RBC lifetime. Decreased action of uridine diphosphate glucuronosyltransferase (UGT), which is required for the conjugation of bilirubin, stops metabolic bilirubin elimination. The UGT enzyme's activity level in a newborn is less. The activity is considered to be around one in 100^{th} of an adult.[7] Newborns are considered to have improved enterohepatic movement, which raises TSB levels even further. In full-term newborns, physiological jaundice often appears at day one of life , peaks between 48 - 96 hours, and then disappears in 14 to 21 days.[2] In contrast, pathological UHB manifests within the initial one day postnatally as the total serum bilirubin exceeds 2 standard deviation or rises by ≥5 mg/dL per 24 hours or >0.2 mg/dL per hour.[8] Unconjugated hyperbilirubinemia's aetiology may be classified into three separate mechanisms: heightened bilirubin creation, reduced bilirubin output, and additional variables.

Increase in Bilirubin Creation

Hemolysis can be caused by immune mechanisms, such as blood group mismatches like ABO or Rhesus incompatibility, or by non-immune factors, including structural defects in the red cell membrane and diseases involving brain parenchyma, polycythemia, and blood infections, may increase bilirubin production. Immune mediated destruction of red blood cells due to blood group incompatibility leads to hemolytic disease of the newborn (HDN).[9] In cases of ABO incompatibility, maternal immunoglobulin G (IgG) antibodies - specifically anti-A and anti-B can cross the

placenta and cause hemolysis in neonates with blood groups A, B, or AB , resulting in UHB. Although the direct antiglobulin test (DAT) is used for diagnosis , its effectiveness in predicting severe UHB is limited by its low sensitivity and predictive value . Though HDN manifests in only around 4% of affected newborns, approximately 15% of pregnancies have ABO incompatibility between mother and fetus.[10]

Rh incompatibility occurs when an Rh-negative mother develops an immune response to Rh-positive red blood cells, usually as a result of exposure during a prior pregnancy. This immune response leads to the production of antibodies targeting the Rh antigen. In the initial exposure, the mother generates IgM antibodies, which do not cross the placenta and therefore pose no threat to the fetus. However, in subsequent pregnancies with an Rh-positive fetus, the immune system produces IgG antibodies capable of crossing the placenta. These antibodies attack the fetal red blood cells, causing hemolysis. Because the Rh antigen strongly triggers the immune system, this condition can lead to hemolytic disease of the newborn (HDN), a serious disorder that often results in hydrops fetalis or elevated levels of unconjugated bilirubin in the newborn.. Identification of women prone to the development of Rh antibodies starts during pregnancy and helps to avoid newborn UHB brought on by immune-mediated hemolysis.[11]

Inheritable as an X-linked recessive disorder, glucose-6-phosphate dehydrogenase (G6PD) deficiency is a frequent red blood cell enzyme anomaly. G6PD helps shield red blood cells from oxidative stress by facilitating the conversion of NADP to its reduced form, NADPH . Exposure to oxidative stressors, including diseases, drugs and colours, results in the destruction of G6PD deficient RBCs, leading to anaemia and hyperbilirubinemia. Over 200 distinct mutations result in G6PD deficiency.[12] The indicators varies by variation, with some neonates potentially experiencing severe hyperbilirubinemia and bilirubin encephalopathy. A condition causing hemolysis, pyruvate kinase deficiency (PKD) could show as unknown hyperbilirubinemia (UHB) in newborns. Disrupting glycolysis and cellular energy generation, PKD is an autosomal recessive disease. PKD causes shorter lives for red blood cells, which causes haemolytic anaemia and unknown blood hemolysis.[13]

Conditions resulting in UHB as a result of RBC membrane abnormalities such as hereditary sphaerocytosis (HS) as well as hereditary elliptocytosis (HE). Hematological Syndrome (i.e., Minkowski Chauffard illness) is the predominant erythrocyte membrane abnormality resulting from alterations in erythrocyte membrane proteins.[14] Most cases are inherited as autosomal dominant (AD) traits and might show in the neonatal stage with UHB. [15] Fifteen Mutations in structural membrane proteins induce hereditary elliptocytosis, a sort of red blood cell membrane defect that traps elliptical-shaped red blood cells in the spleen, hence causing extravascular hemolysis and elevated total serum bilirubin. [16]

Additional risk factors for newborn unconjugated hyperbilirubinemia resulting from an increased bilirubin burden include etiologies causing RBC sequestration. Polycythemia is associated with several conditions such as intrauterine growth restriction (IUGR),infants of diabetic mother (IDM), large for gestational age (LGA) neonates, maternal smoking, high altitude pregnancies, twin-to-twin transfusion syndrome, and placental transfusion methods like delayed cord clamping and umbilical cord milking-all of which are linked to a heightened risk of UHB in newborns. Research indicates that both term and preterm newborns' neurodevelopmental results are improved by delay in clamping of the cord, which also decreases the incidence of postnatal anaemia.[17,18] Clamping the cord at a delayed time has become increasingly fashionable, however it may elevate the threat of HB.[19,20]

Reduced Bilirubin Clearance

Indirect hyperbilirubinemia typically arises due to reduced bilirubin clearance , often caused by either a deficiency or malfunction of the UGT enzyme. Conditions linked to insufficient UGT activity include Gilbert syndrome and Crigler -Najjar syndrome. Gilbert syndrome, in particular, is caused by mutation in the UGT1A1gene, leading to decreased production of the enzyme and resulting in elevated levels of unconjugated bilirubin. [21] Gilbert syndrome generally manifests as moderate jaundice throughout the periods of physiological stress, without the presence of hemolysis or liver impairment. Nonetheless, occurrences throughout the newborn interval are infrequent and typically linked to G6PD deficiency.[3,22] The

condition known as Crigler-Najjar (CN) syndrome type I is an Autosomal Recessive illness characterized by total lack of UGT action. Patients exhibit significant HB within the initial days of infancy, frequently resulting in encephalopathy. Those with CN syndrome type II have residual activity involving UGT enzymes; hence, those peoples total serum bilirubin are not so pronounced among those in individuals having type I form, and the occurrence of encephalopathy is infrequent.[23]

Misc Causes

Additional causes of UHG encompass congenital thyroid hormone deficiency, drugs, antibiotics, GI blockage, pyloric obstruction, breast-milk jaundice, and inadequate breast-feeding. Infants of diabetic mother have greater risk of developing UHB. Two other common causes of unconjugated hyperbilirubinemia in newborns are jaundice brought on by BF and breast milk. Breastfeeding jaundice appears in the 1st week of life , which causes dehydration and sometimes elevated salt.[7] Inadequate consumption reduces GI movements and the excretion of bilirubin in feces. In contrast, BMJ manifests late in the first 7 days postnatally, reaches its zenith during the fortnight, and often fades by 14 days, although it may remain for as long as three months. BMJ is infrequently pathological and correlates with sufficient consumption and healthy bulk up.[24,25,26]

Individuals with IDM frequently exhibit polycythemia, accompanied by a heightened prevalence of icterus.[27] UHB in congenital hypothyroidism is connected with diminished hepatic bilirubin absorption, compromised UGT activity, and reduced gut motility, whereas gastrointestinal obstruction enhances bilirubin recycling by increasing enterohepatic circulation. UHB in congenital thyroid hormone deficiency is connected with diminished bilirubin uptake by liver, compromised UGT activity, and reduced gut movements, whereas GI blockade enhances bilirubin recovery by increasing enterohepatic circulation.[28] Systemic infection may predispose a neonate to unconjugated hyperbilirubinemia by inducing oxygen damage to RBCc and elevating bilirubin levels.[29] Moreover, most of children with UHB exhibit a blend of 2 or more influencing variables, including as prematureness, a familial history of jaundice necessitating photo-therapy, Asian background, gender, and EBF.[(2)] Babies born preterm face a heightened threat of bilirubin encephalopathy as well as kernicterus,

necessitating vigilant watching; yet, there is inadequate information as well as a lack of agreement standards for addressing unconjugated hyperbilirubinemia in this population.[30,31] Due to the heightened threat of neurotoxicity, the bilirubin tolerance for commencing phototherapy is reduced compared to that for term newborns. Bilirubin functions as an antioxidant as well as may serve a physiological function in newborns.[32,33] Maintaining low bilirubin levels through vigorous therapy in babies born prematurely may diminish antioxidant levels and potentially exacerbate retinal damage. Furthermore, diminished antioxidant levels are correlated with chronic pulmonary illness and brain damage. Consequently, managing UHB in premature infants is difficult in the absence of proper guidelines with scientific support. The latest practice recommendations for the management of hyperbilirubinemia by the AAP in 2022 pertain exclusively to children above 35 weeks of gestation.[30,34]

Conjugated Hyperbilirubinemia (CHB)

"CHB, commonly known as newborn cholestasis, is defined by an increase in serum conjugated bilirubin levels exceeding 1.0 mg/dL, resulting from compromised hepatobiliary performance". Differentiating CHB from UHB is essential, as it necessitates immediate assessment and intervention.[35] The etiologies of CHB are numerous and generally categorized as follows:

Infectivity:

Congenital infections must be considered in the picture of newborn cholestasis, particularly when signs of these are present, such as growth restriction. Cytomegalovirus (CMV) is a prevalent congenital illness exhibiting several symptoms. The majority of infected neonates are asymptomatic; however, hepatomegaly and chronic hepatitis B may suggest hepatic involvement.[36] A meticulous examination of history, targeted serologies, and even culture outcomes facilitates diagnosis. Also urine and blood cultures are crucial to the investigative assessment, as UTI and blood infections may induce CHB in newborns. Furthermore, alterations in liver microcirculation, resulting from bacterial products and toxins, are considered potential mechanisms of cholestasis in people with urinary tract infections.[37]

Biliary flow obstruction:

Multiple causes are implicated in the flow obstruction. With incidence differences depending on location, biliary atresia (BA) is main etiology of CHB in newborns.[38] Taiwan, and US, the prevalence is found to be in excess.[39] The causes of biliary atresia (BA) remains poorly elucidated; nevertheless, genetic predispositions, viral infections, toxic exposures, chronic inflammation, and immune related injury to bile ducts also impact its development. The condition affects both intrahepatic as well as bile channels outside liver and often manifests 14 to 28 days postnatally with acholic stools and jaundice. Preliminary ultrasound assessment may indicate a missing gallbladder as well as a characteristic "triangular cord" indication, which is the remains of the extrahepatic bile duct.[40] Timely judgement is essential for optimizing efficacy of the "Kasai procedure" (i.e., hepatic portoenterostomy).[41] By any chance if surgery is postponed beyond 3 months, fewer than one fourth of patients are expected to counter, but surgery conducted before 2 months results in over two third achieving sufficient bile flow.[42] Choledochal cyst may result in biliary flow blockage. Choledochal cysts result in the dilation of both intrahepatic and extrahepatic bile ducts, which can be identified using ultrasonography, distinguishing them from the blocked channels characteristic of biliary atresia.[43] Neonatal sclerosing cholangitis (NSC) is an uncommon cholangiopathy that manifests in newborns with chronic hepatitis B, hepatosplenomegaly, acholic stools, and elevated functioning of GGT.[44] The chances of neonatal cholelithiasis is an uncommon condition that leads to considerable direct hyperbilirubinemia.[45]

Genetic:

Various genetic factors are discussed below which frequently lead to CHB.

Alagille syndrome (ALGS) is an AD disorder. It results from alterations in the JAG1 or NOTCH2 genes, which causes deficiency of bile ducts which are interlobular.[46] With a high frequency, ALGS is the predominant etiology of familial intrahepatic cholestasis, but cholestatic hepatitis B (CHB) in individuals with ALGS may ameliorate with maturation.[35,47]Notable clinical characteristics encompass butterfly vertebrae, congenital cardiac anomalies, renal association and dysmorphic

traits. GGT levels are significantly raised, frequently reaching up to many times the normal amount.

Thosewith cystic fibrosis (CF) may intermittently exhibit cholestasis as a result of aberrant bile causing obstruction of bile ducts.[48] Especially in impoverished countries lacking access to newborn screening, neonatal cholestasis may serve as the initial indicator for detecting cystic fibrosis.

Alpha-1-antitrypsin deficiency is considered as the highest observed genetic etiology of bile obstruction and may present similarity to biliary atresia in newborns.[49] Similar to ALGS, cholestasis may ameliorate with advancing age.

Many inborn metabolic disorders are identified to induce bile obstruction in babies. Babies with galactosemia may demonstrate bile obstruction icterus, eye issues, liver enlargement, FTT, kidney damage, and E.coli infection following consumption of galactose from milk.[50]

A deficiency in the GALT enzyme leads to the accumulation of toxic galactose metabolites in various tissues and organs.The existence of chemicals that have the ability to reduce urine indicates galactosemia, although GALT function in hepatic or blood forming cells substantiates the diagnosis. Neonatal cholestasis is a clinical manifestation of hereditary tyrosinemia type 1, an AR condition developing due to lack of fumarylacetoacetate hydroxylase. Additional features of this condition include Fanconi syndrome affecting kidney, liver enlargement, coagulation abnormalities, and an elevated chance of hepatocellular cancer in elderly patients who didn't receive the treatment.[51]

Progressive familial intrahepatic cholestasis (PFIC) is a diverse collection of three genetic conditions. This condition affects the transfer of hepatobiliary contents, characterized by bile obstruction.[52] The two types including 1 and 2 typically occur during newborn period, whereas the third type emerges delayed in first year. Those afflicted by PFIC often progress to cirrhosis and ESLD in childhood. In analytical examinations, the GGT levels are standard in both types one and two, but increased in type 3.[53]

Aagenaes syndrome, or lymphedema cholestasis syndrome (LCS), is a rare inherited form of intrahepatic cholestasis characterized by neonatal biliary obstruction and swelling in the lower limbs. Aagenaes syndrome occurs as inherited as an AR condition, predominantly observed in people of the Norway or their ancestry.[54]

Dubin-Johnson syndrome (DJS) is an uncommon autosomal recessive disorder resulting from mutation in "ABCC2 gene", responsible for encoding and transporting non-biliary ion through liver. The distinctive characteristic of DJS is existence of a dark-hued liver and the presence in urine of the molecule known as coproporphyrin.[55]

Bile acid synthesis disorder (BASD) is caused by shortage of chemical essential for the alteration of cholesterol into components such as bile acids. BASDs are a rare etiology of cholestasis, yet numerous cases are amenable to medical intervention.

Miscellaneous:

Additional factors that may result in CHD encompass idiopathic newborn hepatitis, cholestasis produced by parenteral feeding, prenatal alloimmune hepatic illness, neonatal hemochraomatosis, and low blood pressure.

Parenteral nutrition-associated cholestasis (PNAC) is a significant iatrogenic cause of cholestasis in preterm infants receiving parenteral nutrition (PN). PNAC occurs in around 20% of infants undergoing parenteral nutrition for two weeks or more.[56] The duration of parenteral nutrition administration and intestinal breakdown are risk factors for parenteral nutrition-associated cholestasis.[57] Additional conditions, such as blood infections and necrotizing enterocolitis, may potentially exacerbate liver damage.[58]

Gestational alloimmune liver disease (GALD), accountable for newborn hemochromatosis, is an alloimmune condition described by intrahepatic and extrahepatic Fe accumulation, culminating in hepatic failure.[59,60] Idiopathic neonatal hepatitis refers to newborn cholestasis of unknown cause following comprehensive diagnostic assessment. Recent diagnostic methods facilitate more accurate diagnosis, resulting in a reduced number of neonatal cholestasis cases being categorized as "idiopathic."[42]

Epidemiology

UHB is commonly observed throughout the newborn phase. Around 80.0% of both full term and preterm newborns develop visible jaundice when their total serum bilirubin levels exceed five mg/dL.[2,61] However phototherapy is required in only approximately 10% of infants .[62] Physiological jaundice is the leading cause of clinical jaundice that appears after the first 24 hours of life, responsible for approximately fifty percent of cases.[63] Around 15% of breastfed newborns experience physiological UHB continuing over 21 days.[64]

Some newborns with jaundice show signs of pathological hyperbilirubinemia. Approximately 1 out of every 2,500 live babies will have severe hyperbilirubinemia, which is often defined as a bilirubin level more than 25 mg/dL. Most often identified causes are ABO incompatibility and G6PD deficiency.[65] The bilirubin levels of neonates of Asian heritage are higher than those of newborns of Black or White descent.[66,67] Babies born in mountainous areas or in countries bordering the Mediterranean Sea, especially Greece, are at a higher risk of developing neonatal jaundice.[68,69]

Acute bilirubin associated brain damage manifests in roughly 1 in 10,000 live births, whereas the incidence of chronic bilirubin associated brain damage is comparatively minimal, estimated at 1 in 50,000 to 100000 live births.[70] Nonetheless, developing countries indicate elevated incidences of kernicterus, a lasting neurological disorder.[71]

CHB is significantly rarer than UHB, occurring in around 1 in 2500 term newborns.[72] Biliary atresia is the predominant cause of obstructive jaundice in neonates, representing around one fourth to half of the cases, succeeded by infections and parenteral nutrition-induced biliary obstruction.[73] Approximately two third children with biliary atresia will necessitate liver transplant, making it the predominant rationale for paediatric liver transplants.[74]

Pathophysiology

Bilirubin is generated by the catabolic process of heme, a degradation byproduct of Hb, . The first step involves the conversion of heme into biliverdin,

through the action of the enzyme heme oxygenase , which also release iron and carbon monoxide in the process.[75,76]

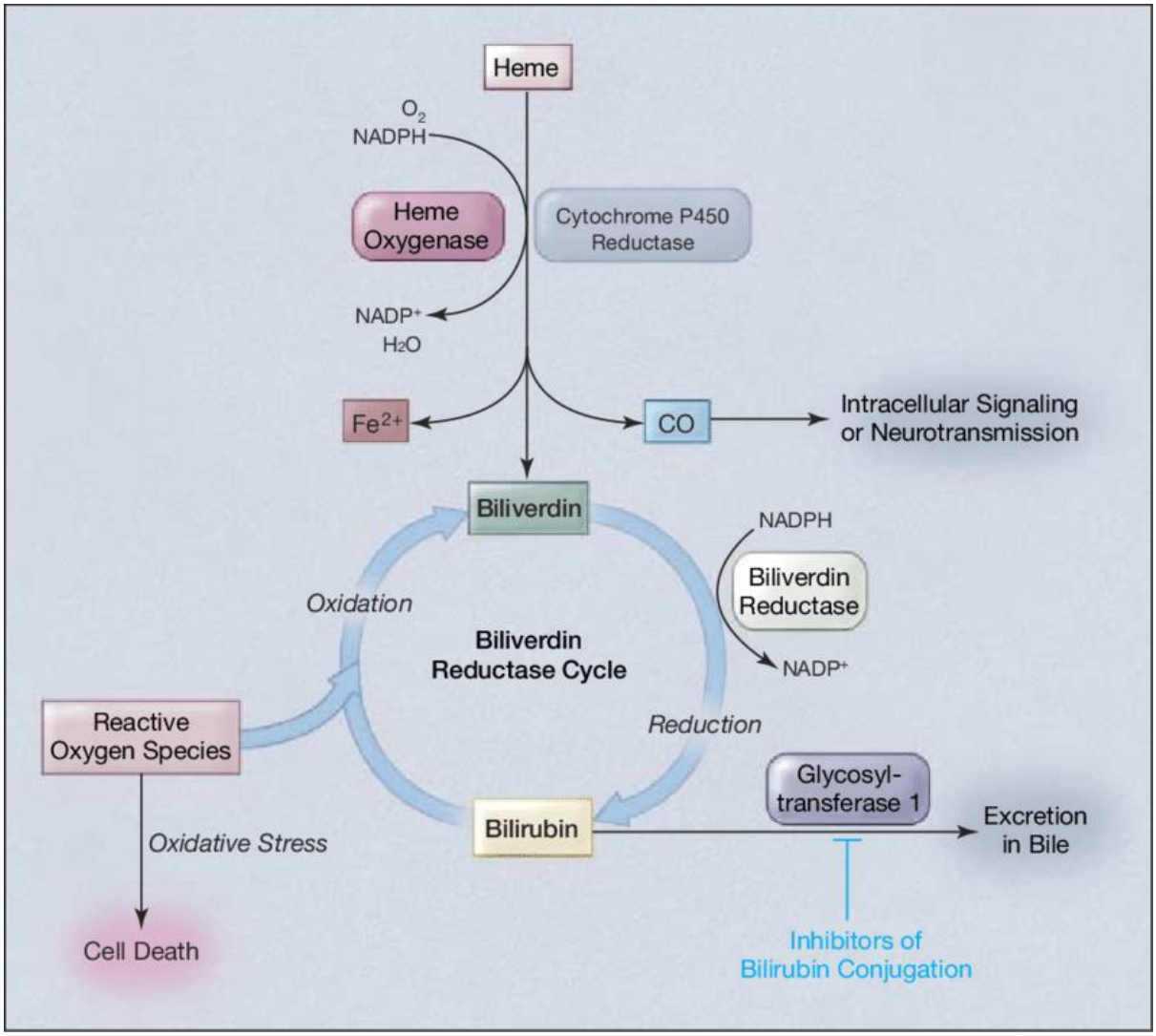


Figure 1: Bilirubin production and recycling.

Newborn display elevated bilirubin compared to adults, attributable to enhanced Hb at birth, a reduced red blood cell lifespan, and the neonatal liver's restricted conjugation capacity.[77] values of 5 to 6 mg/dL are common for healthy, full-term babies, in contrast to values below 1 mg/dL in adults. Pathologic jaundice in newborns is associated with several mechanisms , including increased produtction in the reticuloendothelial system, reduced uptake by the liver, insufficient conjugation , and heightened enterohepatic circulation. [75]

Unbound and unconjugated bilirubin may adhere to brainstem and other organs in extreme hyperbilirubinemia.[78,79,80]

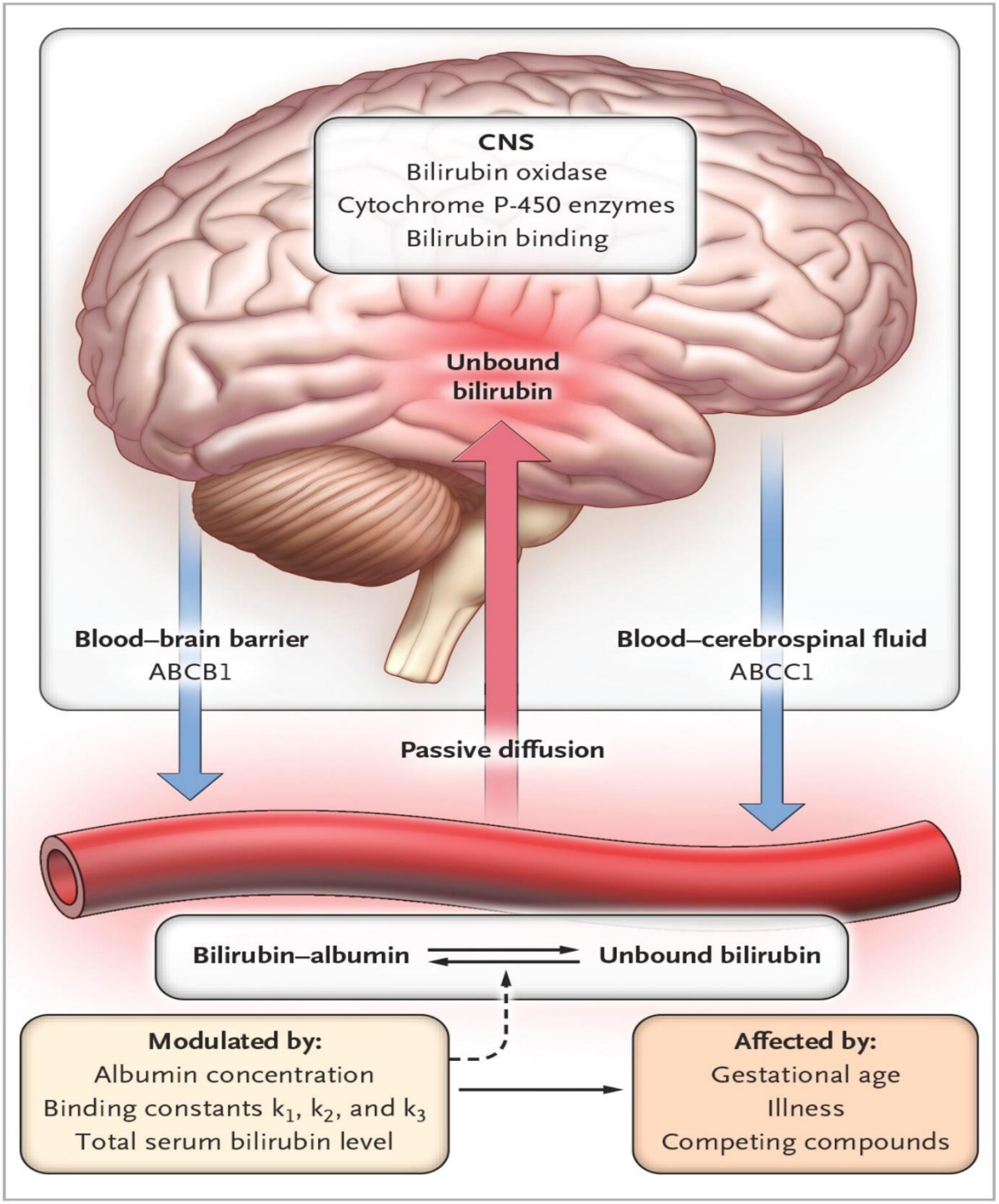


Figure 2: BIND mechanism

Clinical manifestations of bilirubin poisoning, including bilirubin encephalopathy and bilirubin-induced neurologic dysfunction (BIND), are associated with these routes. What happens to the brain depends on how long it's exposed to bilirubin and how much of it is in the brain.

Nonetheless, the TSB level has poor correlation with bilirubin harmfulness in the lack of blood destruction.[75] Preterm newborns exhibit heightened susceptibility to deleterious effects of unbound unconjugated bilirubin.[71]

CHB arises from dysfunctions in the various stages of bile salts and bilirubin metabolism, causing an accumulation of bile acids in the liver that promote bile duct proliferation and fibrosis.[81] Bile acid contributes to the inflammation and death of liver cells, resulting in liver cell damage and cirrhosis.[82] Insufficient bile secretion in cholestasis causes decreased absorption of fats and fat-soluble vitamins A, D, E, and K, potentially resulting in nutritional deficiencies and weight loss.[83]

Histopathology

The term kernicterus originated in the Germany - from term "kern," signifying core, which pertains to the portion of the brain representing the basal ganglia.

Histopathology shows - Yellow spotting of brain nuclei situated at deep location. Histopathological observations reveal nuclei exhibiting pyknosis, cytoplasmic vacuolation, and a reduction of Nissl material in neurons.[84]

The histopathologic characteristics of biliary atresia encompass -

- Enlargement of hepatic portal tracts accompanied by oedema,
- Fibrodysplasia,
- Bile duct expansion, and
- Occurrence of bile obstructions within the lumen of the duct.

Large cells with multiple nuclei and haematopoiesis are other abnormalities identified on microscopic studies of cholestatic liver tissues.[85] The prominence of hepatic erythropoiesis, while not diagnostic of any specific illness, is more commonly observed in cholestasis of infectious origin.[86] Among inherited causes of cholestasis , canalicular cholestatsis is frequently observed in patients with PFIC type 1. This form

of the condition is characterized by isolated periportal biliary metaplasia of hepatocytes and a notable absence of ductular proliferation. Hepatic architectural changes, inflammation, and prominent lobular and portal fibrosis are more common in PFIC type 2, despite similar histology.[53]

Examination findings

Evaluating a newborn with jaundice begins with obtaining a thorough medical history, encompassing puerperium problems, familial history, birth details, the beginning of related symptoms, sufficiency of breastfeeding, and maternal serological consequences. The color of stool and urine may indicate the type of jaundice.[8] AAP advocates for ocular examination of all babies for jaundice every half day from birth until discharge. Additionally, doctors must recognize danger signs for critical hyperbilirubinemia.[8] Important hazards in infants >35 weeks gestation comprise:[8]

- Near high normal bilirubin level before discharge
- Jaundice seen at first day
- ABO and Rh incompatibility
- Lower gestational age [87]
- Family history of phototherapy
- Traumatic birth
- EBF
- Genetic – Asian family

Newborns should be evaluated for jaundice in natural daylight.[88] Consequently, clinically severe jaundice must always be validated with bilirubin test or transcutaneous bilirubin (TcB) measurement. Pallor, petechiae, bruises, enlarged liver and spleen, loss of weight, and dehydration are some of the symptoms. It is important to check for signs of brain injury in all infants born with icterus, such as difficulty feeding, lethargy, disturbed sleep, abnormal tone, and epilepsy. In spite of this, kernicterus may cause no symptoms at all in as many as 15% of newborns.[75]

Investigations

Diagnostic Studies in UHB

The guidelines recommend assessing an infants bilirubin levels within the first 24 to 48 hours after birth.

Using -

- While the transcutaneous measurement device may reduce blood test frequency, its usefulness is less reliable in neonates with darker skin tone or after photo-therapy. One other drawback of the TcB is that it can't pick up on the direct bilirubin fraction, which is crucial for infant cholestasis diagnosis.
- "Blood samples for total serum bilirubin.[89,90,91.]
- Maternal and newborn blood types,
- Direct antibody test (DAT),
- Complete blood cell (CBC),
- Reticulocyte count,
- Blood smear, and
- G6PD testing.
- Serum albumin considered a proxy measure for free bilirubin.
- Free bilirubin is the portion responsible for bilirubin-induced toxicity.[92]
- The bilirubin-albumin ratio (B/A) ratio

Brain magnetic resonance imaging (MRI) findings have excellent sensitivity for bilirubin encephalopathy".[93]

Diagnostic Studies in Conjugated Hyperbilirubinemia

- "Serum aminotransferases - liver damage.
- Elevated alkaline phosphatase and GGT - biliary blockage.
- The prothrombin time (PT), international normalized ratio (INR), and serum albumin - liver synthesis and function.
- TORCH titres for intrauterine infections,
- urine cultures,

- viral cultures,
- serological titers,
- newborn screening,
- assessments for inborn errors of metabolism, alpha-1 antitrypsin phenotype, and
- genetic profiling"

Choledochal cysts, inspissated bile, gallstones, and biliary sludging may all be seen by hepatic ultrasonography. Biliary atresia is characterized by a triangle cord indication.[81] The Hepatobiliary scintigraphy is an additional technique employed to assess newborn cholestasis.[94] A liver biopsy is the definitive method for identifying newborn cholestasis. An competent pathologist's histopathologic interpretation will accurately diagnose 90% to 95% of cases, potentially averting needless procedures in individuals with IHC.[95]

Treatment of UHB

Phototherapy

Exchange transfusion.[8]

Phototherapy (PT)

PT is the main treatment for UHB. By lowering bilirubin levels to acceptable ranges, phototherapy lessens the likelihood of bilirubin toxicity and the need for other therapies. Infant GA, bilirubin measured at certain hours, and neurotoxicity risk factors determine the bilirubin threshold for PT.[8,34].Several factors increase the risk of bilirubin induced neurotoxicity and therefore lower the TSB threshold for initiating phototherapy , including: [8,34]

- Preterm
- Albumin level <3.0 g/dL
- Positive DAT
- G6PD deficiency or other blood destructive diseases

- Blood infection
- Deterioration of the condition of the child.[31]

The efficacy of PT depends on both the intensity and the wavelength of light used, in addition to the newborn's exposed skin. Augmenting the dosage of phototherapy can be accomplished by positioning PT components at a reliable space from the newborn and enhancing the quantity of components employed.

Bilirubin efficiently take in light within the blue-green spectrum. The fundamental process of phototherapy entails generating photoisomerization to transform bilirubin into lumirubin, which is efficiently eliminated in bile as well as urine.[96] During phototerapy , it is essential to expose the maximum possible body surface area of the neonate to the light surface, while shielding the eyes to prevent retinal injury and minimizing disruptions to ensure treatment effectiveness. Maintaining hydration is essential for sufficient urine production, as the majority of bilirubin is eliminated in the urine as lumirubin, produced throughout PT.[34]

Upon cessation of phototherapy, a rise in bilirubin levels, referred to as rebound bilirubin, may occur. This level is often inferior to the before treatment level and seldom necessitates the restart of PT.[97] Physical therapy is deemed safe; nonetheless, current findings indicate a potential correlation with lasting consequences, involving a minor threat of seizures. Nevertheless, no research has established causality.[98] Children undergoing PT may be at increased risk for solid organ cancers and nonlymphocytic leukemias, according to a number of studies.[99,100] The hostile effects of PT encompass spates, thirst, low calcium, eye impairment, oxidative stress in the blood .[101] Bronze baby syndrome is an illness illustrated by enhanced concentrations of conjugated bilirubin, which infrequently happens with PT, leading to uneven coloring of the skin, mucous membranes, and urine. Bronze baby syndrome often cures within several days after ceasing PT; however, the outcome is contingent upon the fundamental etiology of the CHB.[102,103]

Exchange Transfusion

During the 1940s, exchange transfusion (ET) emerged as inaugural effective management for jaundice.[104] Currently, exchange transfusion serves as the second-line

treatment, following the introduction of phototherapy in the 1950s.[105,106] This medication is indicated for newborn failure to react to PT or when the total serum bilirubin level reaches the limit for ET. The criteria for commencing exchange transfusion are determined by various parameters, including total serum bilirubin level and its rate of rise, newborn age , and the presence of threat for neurological problems.[34] ET swiftly eliminates bilirubin and antibodies that induce blood destruction from the newborn's bloodstream. The baby's blood is supplemented with donor blood that is a perfect match via a double-volume exchange transfusion. Given that the majority of bilirubin is extravascular, bilirubin level immediately after exchange transfusion is roughly two third of the pre-exchange; however, it subsequently rises to 70% to 80% of the pretreatment level due to equilibrium.[107,108]

Intravenous Immunoglobulin

(IVIG) is administered when immune-facilitated blood destruction leads to unconjugated hyperbilirubinemia, as it inhibits RBC destruction by binding to Fc receptors on RBCs. The guidelines advises IVIG combination for immune-facilitated blood destruction if the bilirubin stays inside two to three mg/dL of the ET limit regardless of aggressive PT.[109,110] The testimony indicating that IVIG diminishes necessity for ET is ambiguous. Nevertheless, IVIG is frequently employed in clinics to address severe UHB

Treatment of Conjugated Hyperbilirubinemia

The treatment of CHB is customized based on the underlying cause of the icterus. To attain optimal results, those who have biliary atresia necessitate a “Kasai procedure” in the initial 2 months of life to avert irreparable hepatic injury.[41,111] Infectious etiologies of bile obstruction are managed with targeted antibiotic therapies, while management with cholic acid and chenodeoxycholic acid repeatedly yields a therapeutic outcome for several bile acid synthesis disorders (BASDs). Patients with GALD demonstrate favourable responses to IVIG and double-volume exchange transfusion.(60) Cholestasis generated by parenteral nutrition is addressed with cyclic PN, minimizing disclosure length and commencing gastric feeds at the earliest opportunity.

Similar studies

In the research by Hakanson et al., during 1981, they studied rats that were subjected to white, fluorescent light. This resulted in a decrease in serum calcium levels in young rats. He demonstrated that the decrease in calcium levels was correlated along a decline in serum melatonin content. The result may be mitigated by protecting the occiput, decreasing steroid production, and administering exogenic melatonin. Hypocalcaemia can occur as a result of enhanced calcium absorption by bone following the diminished inhibitory impact of melatonin after transcranial illumination of the pineal gland.[112]

Zecca et al., in their study published in 1983, investigated 100 preterm neonates to ascertain the efficacy of Calcifediolo in preventing hypocalcemia induced by PT. The results indicate that Calcifediolo is ineffective in mitigating the rise of PT-induced low calcium in preterm newborns. Vitamin D is hence improbable to significantly influence the pathogenesis of hypocalcemia generated by phototherapy.[113]

In the research conducted by De Curtis et al., during 1989, thirty jaundiced neonates experiencing diarrhoea, undergoing phototherapy, were examined alongside 30 matched control infants to investigate the etiology of the looseness of the bowels. Fecal osmolality and electrolyte values were assessed, providing definitive confirmation that the looseness of the bowels originated from colonic discharge. Rectal concentration of H_20 and electrolytes was assessed in 10 jaundiced newborns undergoing phototherapy, 10 infants who had jaundice and not undergoing phototherapy, and 10 strong controls using a rectal dialysis bag. An additional cohort of eight jaundiced newborns was examined both during and after to phototherapy to record the reversal of ion transport alterations. The absorption of H_20, Nacl, and potassium was markedly reduced in patients undergoing PT compared to the control groups.[114]

In the Sethi et al. study, which was published in 1993, sixty infants with hyperbilirubinemia were incorporated into the study. In the study there were 20 preterm neonates in Group A and 20 full-term neonates in Group B. A control group

was established including ten infants from each cohort. The newborns in the study group received PT, whereas those in the control group did not get PT. The serum ca2+ levels of both groups were analyzed. Ninety percent of preterm neonates and three fourth of full-term neonates experienced low calcium levels following phototherapy. The study group exhibited a markedly significant decrease in both total and ionized calcium levels compared to the control group. Neonates undergoing phototherapy are advised to receive supplementary calcium to avert hypocalcemia.[115]

In the Jain et al study during 1998, they realized, 55.0% of before term newborns and thirty percent of full-term newborns with hyperbilirubinemia experienced hypocalcaemia following 2 days of PT. Among preterm newborns, one third exhibited jitteriness, whereas one fourth had irritability. Among full-term neonates, 50.0% exhibited jitteriness and 16.7% displayed irritability. Consequently, they determined that PT-induced hypocalcaemia is a considerable issue, warranting the consideration of calcium supplements for these infants.[116]

Yadav RK et al., during 2011, investigated the impacts of PT on 20 term and 20 before term hyperbilirubinemic newborns. Following 2 days of PT, a substantial decrease in Ca2+ levels was noted in two - third of term infants and 80% of preterm neonates. Calcium levels should be assessed in infants undergoing PT for over 2 days and maintained appropriately.[117]

Alizadeh - Taheri et al., during 2013, conducted a study, involving 147 term newborns. The study revealed a decrease in serum Ca2+ levels in 56.0% of the infants, with 7% experiencing significant hypocalcemia (p=0.03) after 2 days of PT.[118]

In their 2014 study, Arora et al., examined the neonatal unit at in Amritsar. After two days of phototherapy , hypocalcemia was observed in 43% of preterm and 56% of term newborns, with symptomatic cases being more common among preterm infants that their term counterparts.[119]

MATERIALS & METHODS

MATERIALS AND METHODS

Source of Data: Consecutive newborns with hyperbilirubinemia cases receiving phototherapy admitted in department of paediatrics, RL JALAPPA HOSPITAL, KOLAR

Study Design: Hospital based prospective observational clinical study.

Study Period: 1 year period from 2023 August to 2024 August

Inclusion Criteria:

1. All the neonates with unconjugated hyperbilirubinemia from 24 hours of life to 14 days of life receiving phototherapy.
2. Whose parents or caregivers are willing to give consent for the study.

Exclusion criteria:

- Neonates with conjugated bilirubinemia.
- Newborn with perinatal asphyxia.
- Baby of mother who had history of taking anti convulsants.
- Newborn fed with cow milk.
- Baby born to diabetic mother.
- Babies born with apparent major congenital anomaly.
- Baby with parents who are not willing to give consent.

Sample Size:

Sample size was calculated by using the proportion of hypocalcaemia in subjects who underwent phototherapy was 13.1% from the study by Thriupathi reddy et al. using the formula

$$\text{Sample Size} = \frac{Z_{1-\alpha/2}{}^2 P(1-P)}{d^2}$$

$Z_{1-\alpha/2}$ = is standard normal variate is 1.96

P = Expected proportion

d = precision

P = 13.1% or 0.131

q = 86.9% or 0.869

d = 5% or 0.05

n = 175 subjects

Considering 10% nonresponse rate 175 + 17.5 ≈ 193 subjects was included in the study.

Methods Of Collection of Data:

Prior written informed consent was taken for their cooperation to participate in the study. The patients were included in the study if they fulfilled the inclusion/exclusion criteria. Baseline data were stored from the patients along with pertinent clinical history and relevant lab investigations. We tested the newborns' venous blood for TB, DB,S/E , blood groups.

The Arsenazo technique was used to test calcium, the Diazo method to assess total and direct bilirubin, and an autoanalyzer (Erba EM 200 machine) to analyze electrolytes (Na, K). The antisera technique was used to evaluate the blood groups of neonates.

Both the first and second samples of electrolytes were taken at zero hours and forty-eight hours of PT, respectively, or upon termination of PT, whichever came first. As a control, the first sample was taken. In order to find out what happened to the electrolytes, researchers compared these two sample sets. We used appropriate statistical tests (Student's t-test) to tabulate and evaluate all of the data from the different groups. We compared the proportions using the chi-square test.

The American Academy of Pediatrics established guidelines for determining whether a child born at 35 weeks or more gestation required PT or an exchange

transfusion. The American Academy of Pediatrics has developed two age-specific nomograms - one to guide the initiation of phototherapy and the other for determining the need for exchange transfusion. Three separate categories of newborn risk were marked on the nomograms. Three groups were established: one for babies at lower risk (defined as 38 weeks or more with no risk factors), one for babies at medium risk (defined as 38 weeks or more with risk factors or 35 to 37 weeks without risk factors), and one for babies at higher risk (defined as 35 to 37 weeks with risk concerns).

Total bilirubin (TB) values were used to guide clinical decisions without subtracting the direct (conjugated) fraction. In medium risk neonate , phototherapy was initiated at TSB levels of 10mg/dl at 24 hours, 13mg/dl, at 48 hours, 15mg/dl at 72 hours and 18mg/dl at 96 hours or later. The cutoffs for lower-risk and higher-risk babies were about 2 mg/dL higher and 2 mg/dL lower, respectively, compared to the medium-risk infants. Hypoalbuminemia, sepsis, significant lethargy, acidosis, hypoxia, temperature instability, G6PD deficiency, and immune hemolytic anemia were among the risk factors.

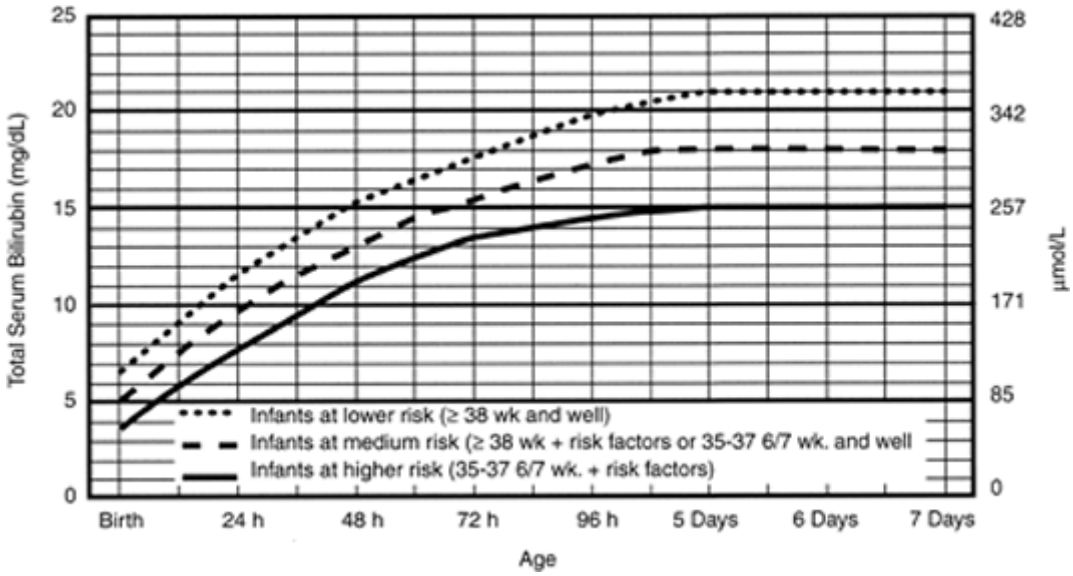


Figure 3 : AAP nomogram for phototherapy in hospitalized infants of 35 or more weeks gestation

STATISTICAL ANALYSIS

Microsoft 365 Excel and SPSS v27.0 were used for data collection and analysis, respectively. Statistical analysis was carried out using the Shapiro-Wilk test for normalcy. The findings were presented as means with standard deviations or medians with interquartile ranges, with frequency and percentage. Association between categorical variables were analyzed using the Chi-square test and Fisher's exact test , as appropriate . A t-test was used to determine the degree of association between the quantitative variables. Results with a P value less than 0.05 were deemed statistically significant, and all statistical analyses were conducted at a 5% level of significance.

RESULTS

RESULTS

Table 1: Gender distribution of the study population

Gender	Number	Percentage
Male	106	54.9 %
Female	87	45.1 %
Total	193	100.0

Figure 4: Graph representing gender distribution

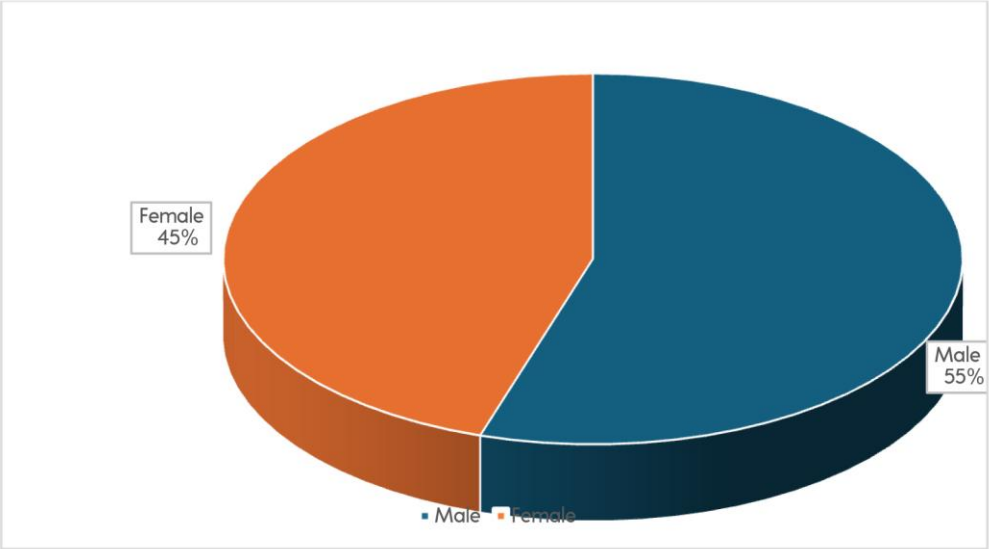


Table 2 : Distribution based on gestational age (n=193)

Gestational Age	Male	Female	Total (%)
< 37 weeks	24	23	47 (%)
37 – 40 weeks	66	48	114 (%)
> 40 weeks	16	16	32 (%)

Figure 5 : Graph representing gestational age distribution

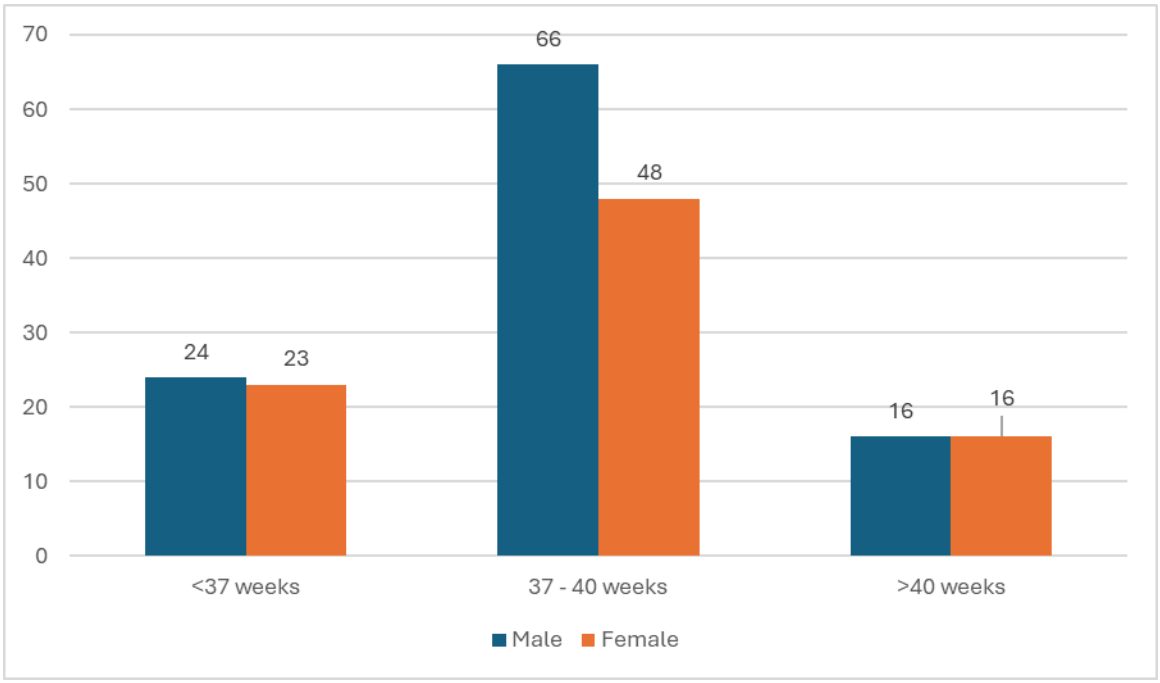


Table 3: Distribution based on weight (n=193)

Weight	Male	Female	Total (%)
LBW (<2.5 kg)	49	39	88 (42.2 %)
Normal weight	57	48	105 (57.7 %)

Figure 6: Graph representing weight distribution of study population

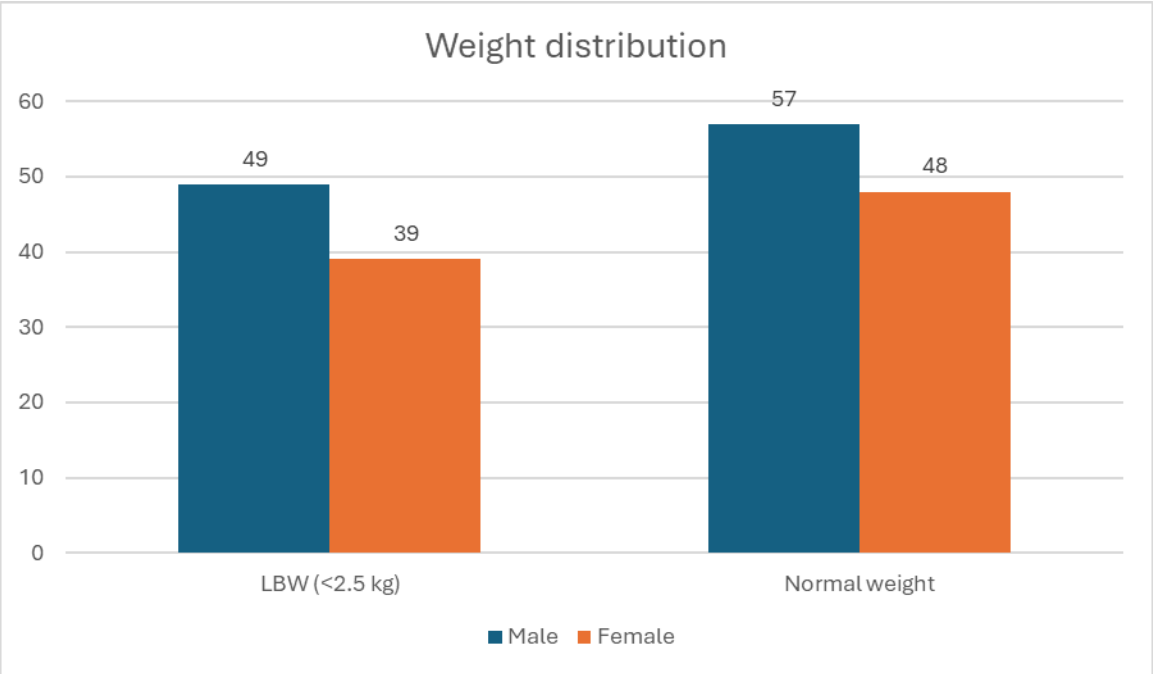


Table 4: Distribution based on phototherapy duration (n=193)

Phototherapy duration	NUMBER
< 24 Hrs	44 (22.5 %)
24 – 48 Hrs	137 (71.1 %)
> 48 Hrs	12 (6.3 %)

Figure 7 : Graph representing duration of PT distribution of study population

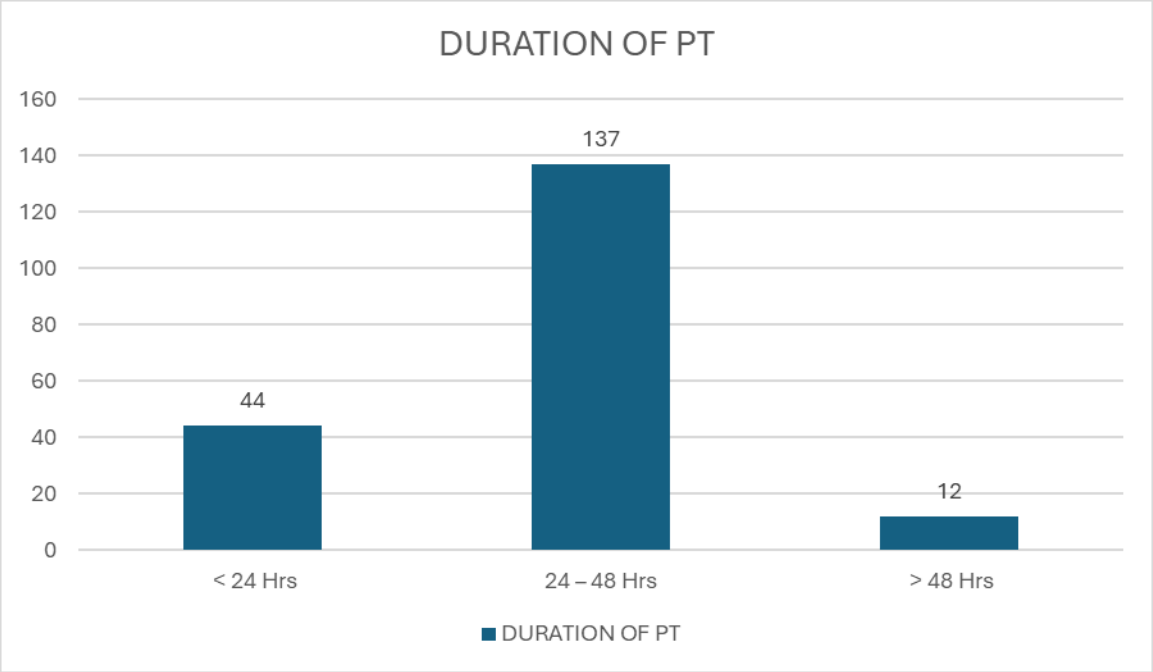


Table 5: PRE-Phototherapy calcium values according to weight (n =193)

Calcium	LBW (n =88)	Normal wt (n=105)	Total (n=193)	P value
< 8	9 (10 %)	2 (2.4%)	11 (5.6%)	0.048*
8 - 11	79 (90 %)	103 (97.5%)	182 (94.4%)	

Figure 8: Graph representing PRE-Phototherapy calcium values according to weight of study population

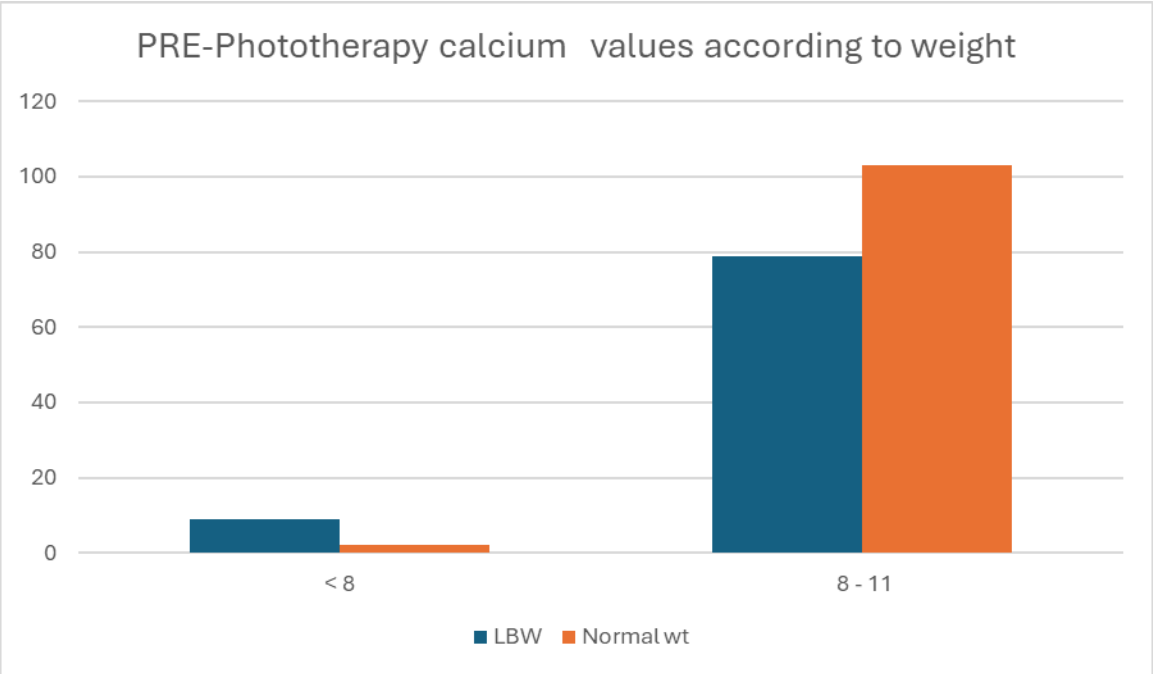


Table 6 : PRE-Phototherapy Sodium values according to weight (n =193)

Sodium	LBW	Normal wt	Total	P value
< 135	3 (3.3 %)	0	3 (1.4%)	0.245
135 -145	60 (66.6%)	92 (87.8 %)	152 (78.8%)	
> 145	25 (30 %)	13 (12.2 %)	38 (19.7%)	

Figure 9: Graph representing PRE-Phototherapy sodium values according to weight of study population

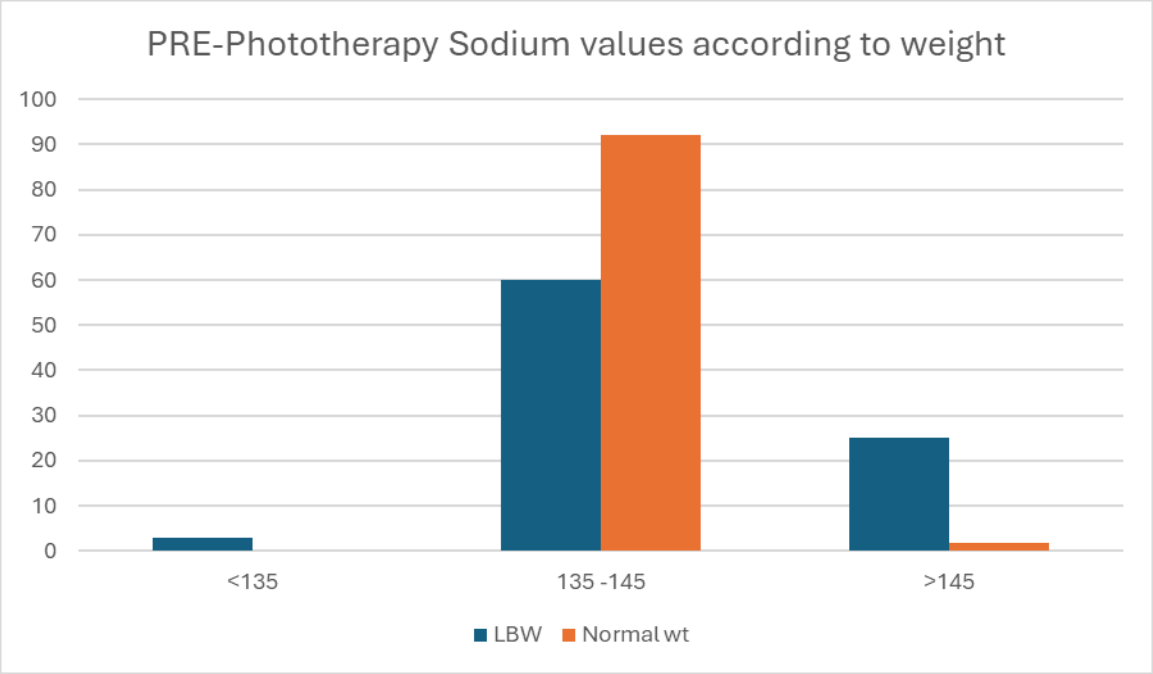


Table 7: PRE-Phototherapy Potassium values according to weight (n =193)

Potassium	LBW	Normal wt	Total	P value
< 3.5	0	0	0	<0.001
3.5 – 5.5	88	105	193 (100%)	
> 5.5	0	0	0	

Figure 10: Graph representing PRE-Phototherapy potassium values according to weight of study population

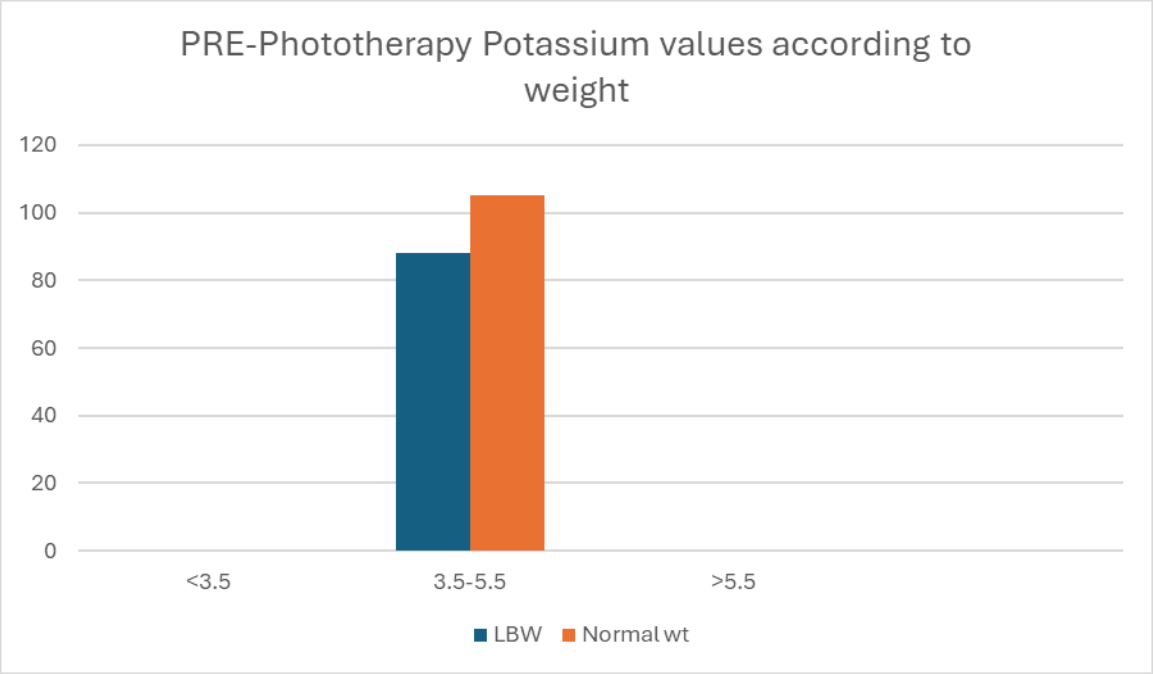


Table 8: Correlation of post phototherapy calcium with weight

Calcium	LBW (n=88)	Normal wt (n=105)	Total (n=193)	P value
< 8	32 (36.6%)	14 (14.6%)	46 (24%)	<0.001
8 - 11	56 (63.3%)	91 (85.4%)	147 (76%)	

Figure 11: Graph representing correlation of post phototherapy calcium with weight of study population

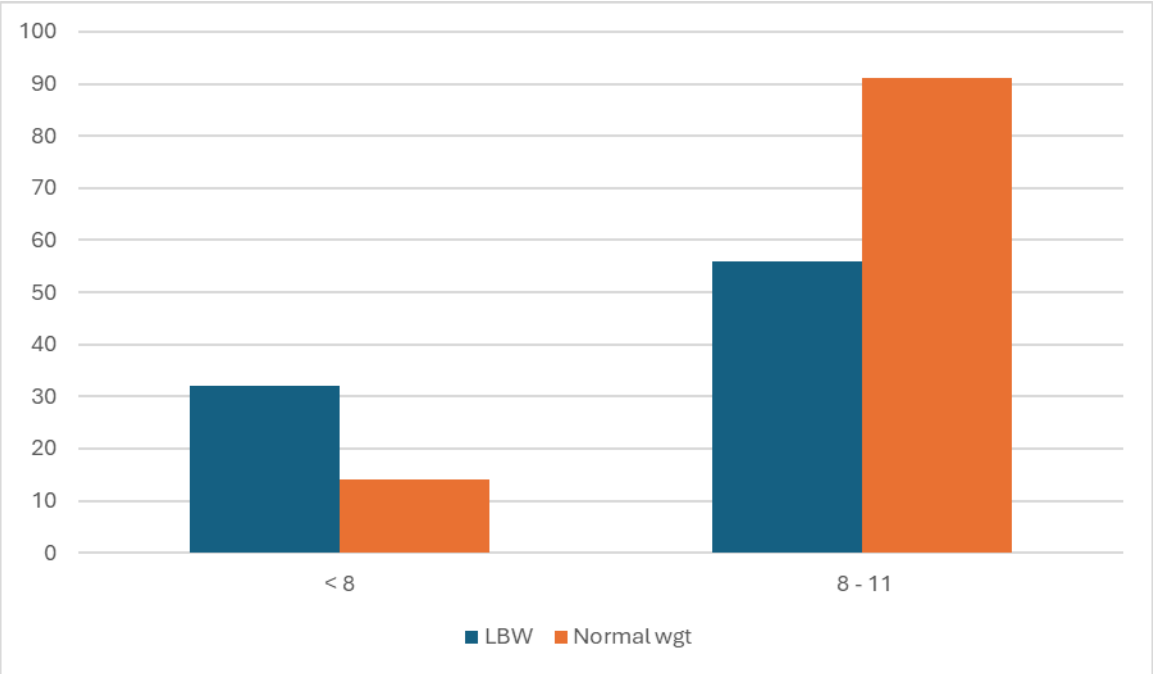


Table 9: Association between post phototherapy sodium and weight

Sodium	LBW	Normal	Total	P value
< 135	6 (8.4%)	0	6 (3.5%)	<0.001
135 -145	50 (53.3 %)	96 (91.5%)	146 (75.4%)	
>145	32 (38.3 %)	9(8.5%)	41 (21.2%)	

Figure 12: Graph representing post phototherapy sodium and weight of study population

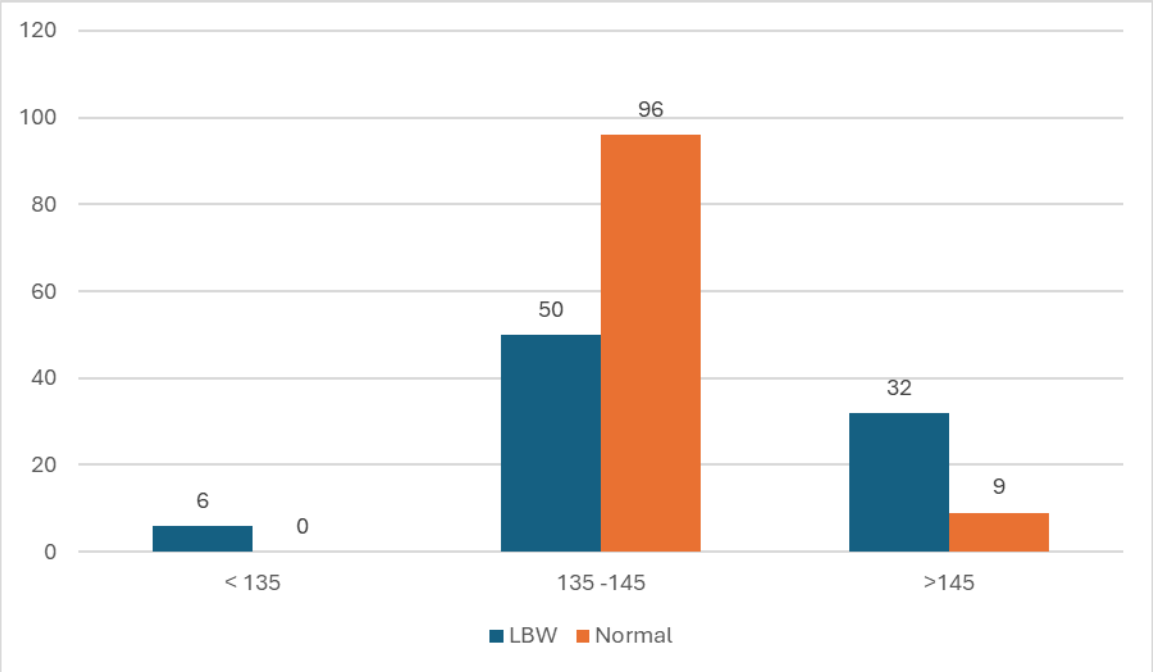


Table 10: Association of post phototherapy potassium and weight

Potassium	LBW	Normal	Total	P value
< 3.5	0	0	0	<0.001*
3.5 – 5.5	85 (96.6 %)	100 (95.3%)	185 (95.8%)	
> 5.5	3 (3.4%)	5 (4.8%)	8 (4.2%)	

Figure 13: Graph representing correlation of post phototherapy potassium with weight of study population

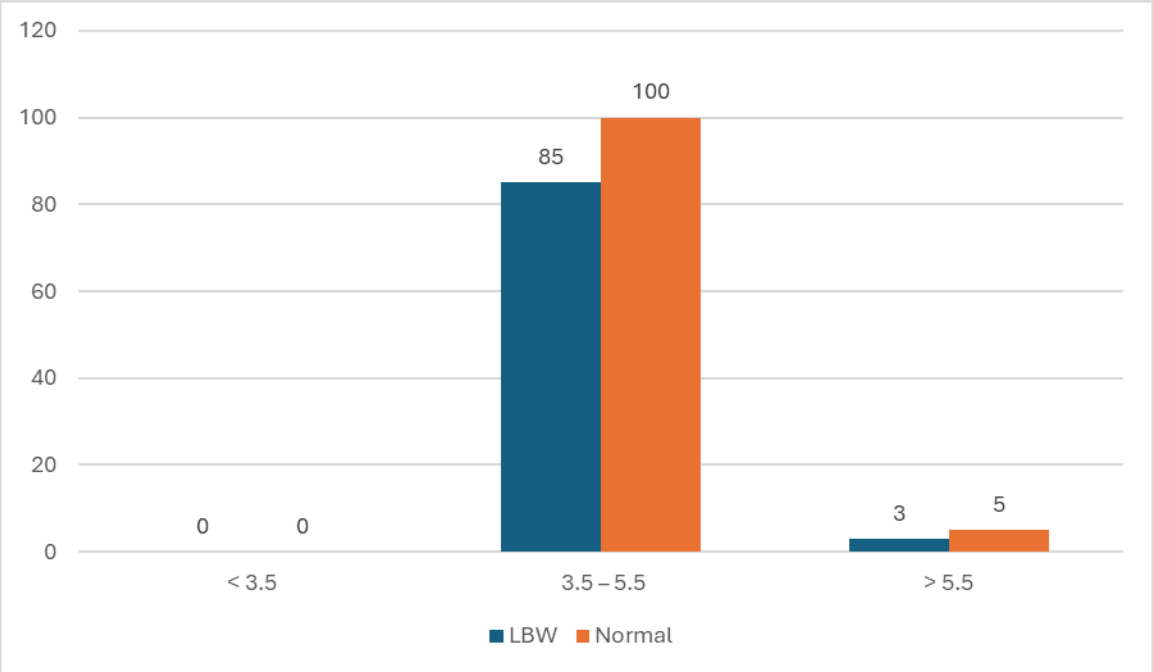


Table 11: Association of post phototherapy calcium with gestational age

Calcium	< 37 weeks (n=47)	37-40 weeks (n=114)	40 weeks (n =32)	Total (n=193)	P value
< 8	29 (61.2%)	16 (13.6%)	0	45 (24%)	<0.001
8 - 11	18 (38.8 %)	98 (86.4 %)	32(100%)	148 (76%)	

Figure 14: Graph representing association of post phototherapy calcium with gestational age of study population

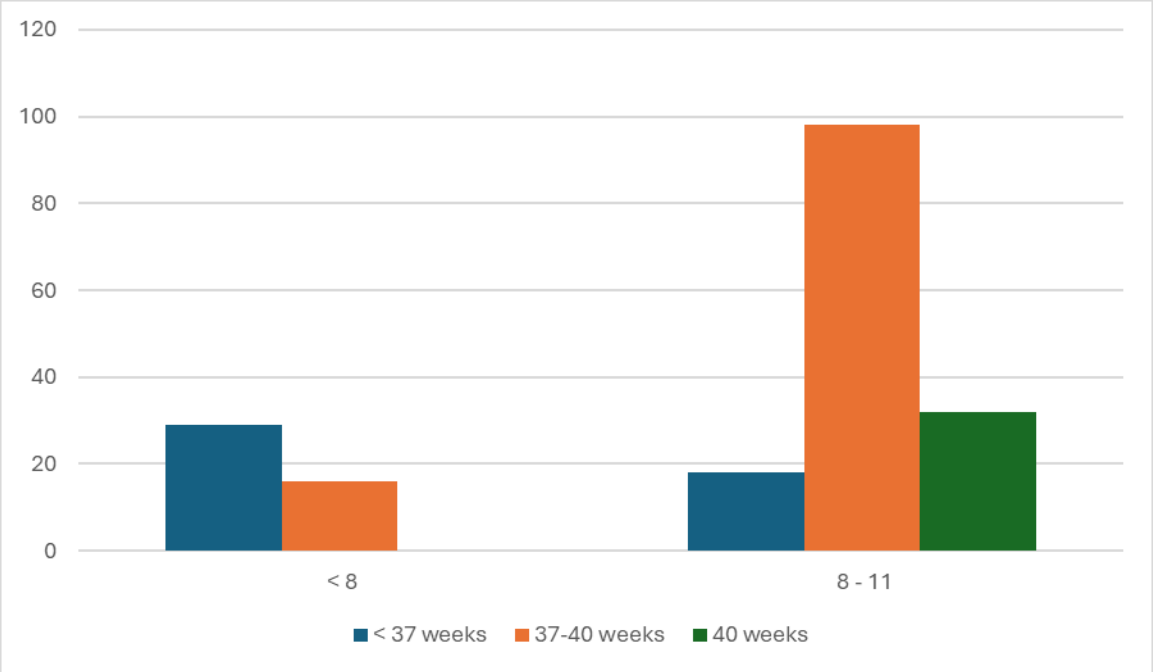


Table 12: Association of post phototherapy sodium and gestational age

Sodium	< 37 weeks	37-40 weeks	> 40 weeks	Total	P value
< 135	4 (8.4%)	3 (2.3%)	0	7 (3.5%)	<0.001
135 – 145	34 (72.3%)	104 (89.7 %)	4 (11.2%)	142 (75.4%)	
> 145	9 (19.4%)	7 (8%)	28 (88.8%)	44 (21.2%)	

Figure 15: Graph representing association of post phototherapy sodium and gestational age of study population

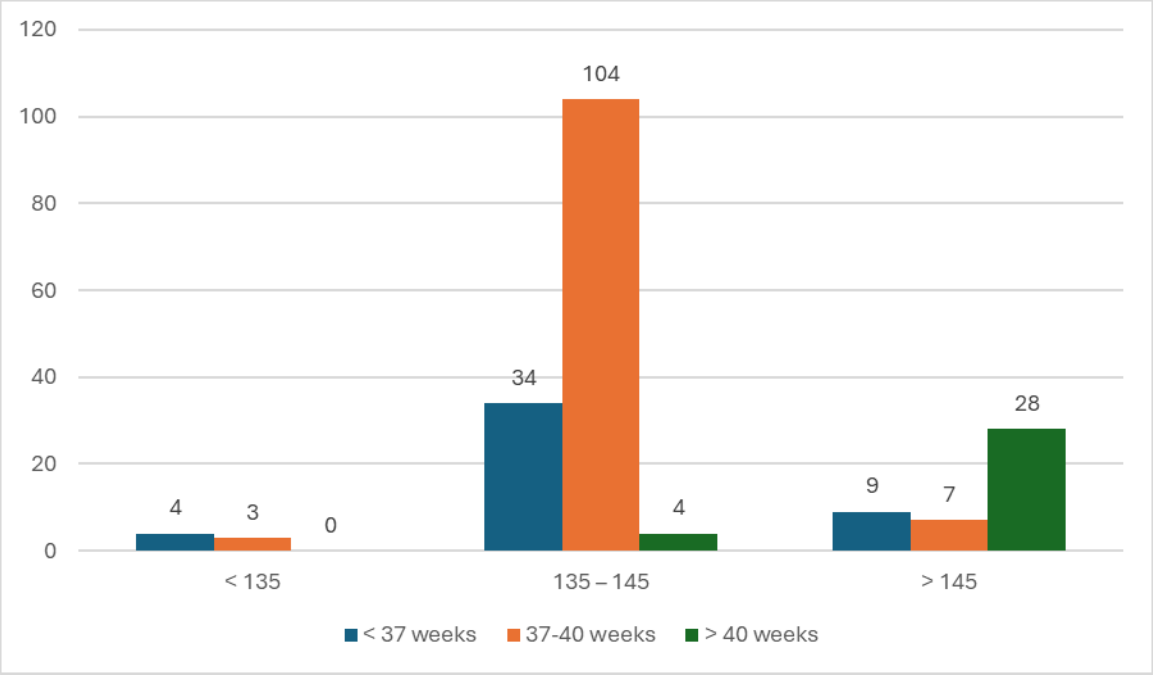


Table 13: Correlation of post phototherapy potassium with gestational age

Potassium	< 37 weeks	37-40 weeks	> 40 weeks	Total	P value
< 3.5	0	0	0	0	<0.001
3.5 – 5.5	43(91.6%)	111(97.7%)	30 (94.4%)	184 (95.8%)	
> 5.5	4 (8.4%)	3 (2.3%)	2 (5.6%)	9 (4.3%)	

Figure 16: Graph representing correlation of post phototherapy potassium with gestational age of study population

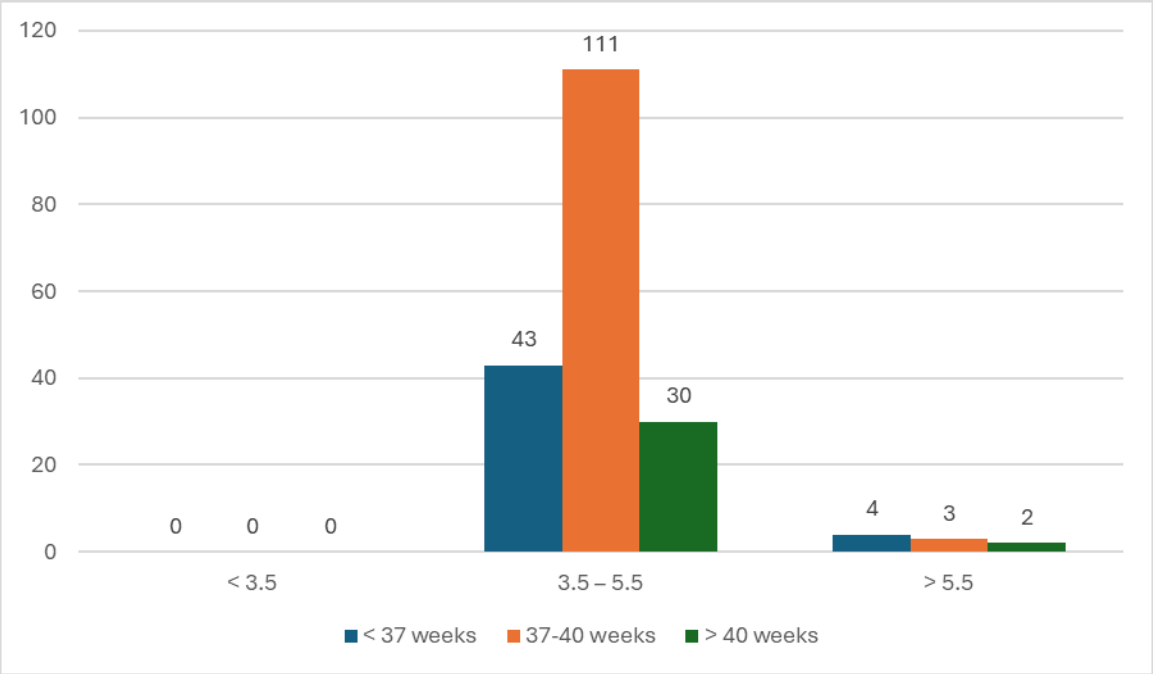


Table 14: Association of post phototherapy calcium in relation to duration of phototherapy

Calcium	< 24 hours (n=44)	24-48 hours (n=137)	> 48 hours (n=12)	Total (n=193)	P value
< 8	4 (9.3%)	38 (27.8 %)	4 (33.4 %)	46 (24%)	0.104
8 - 11	40 (90.7%)	99 (72.2 %)	8 (66.6 %)	147 (76%)	

Figure 17: Graph representing association of post phototherapy calcium in relation to duration of phototherapy of study population

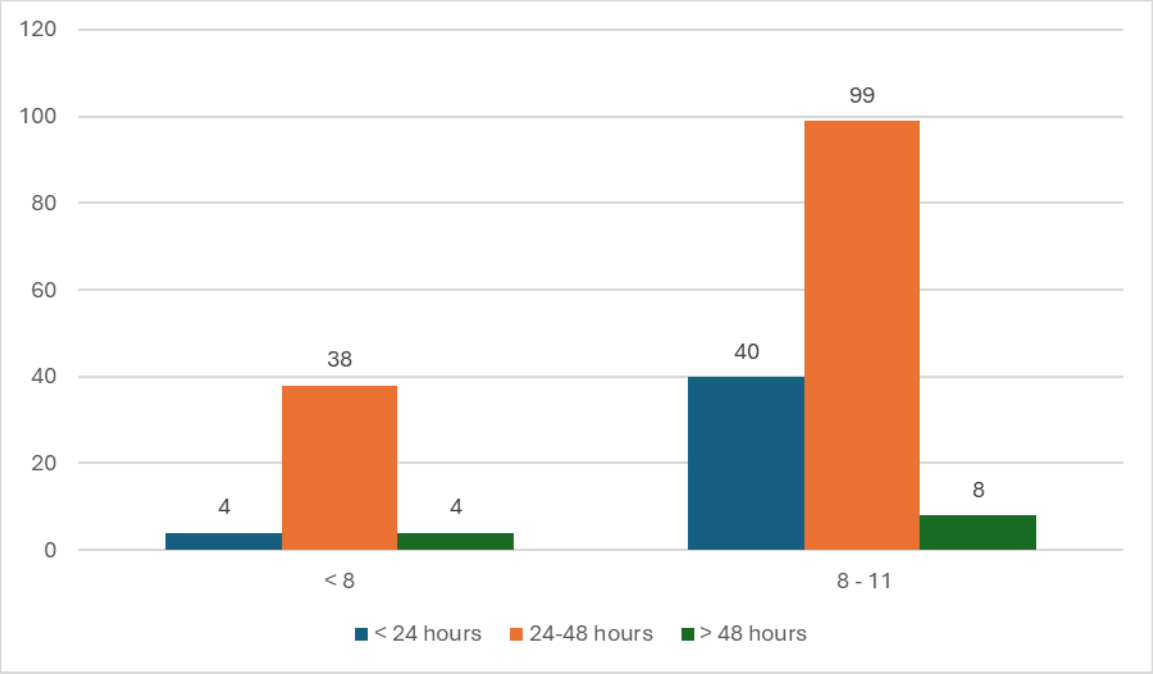


Table 15: Association of post phototherapy sodium and duration of phototherapy

Sodium	< 24 hours	24-48 hours	> 48 hours	Total	P value
< 135	0	5 (4.9%)	0	5 (3.5%)	<0.001
135 – 145	38 (87.5%)	106 (77.3%)	2 (11.2%)	146 (75.4%)	
> 145	6 (12.5%)	26 (17.8%)	10 (88.8%)	42 (21.2%)	

Figure 18: Graph representing association of post phototherapy sodium and duration of phototherapy of study population

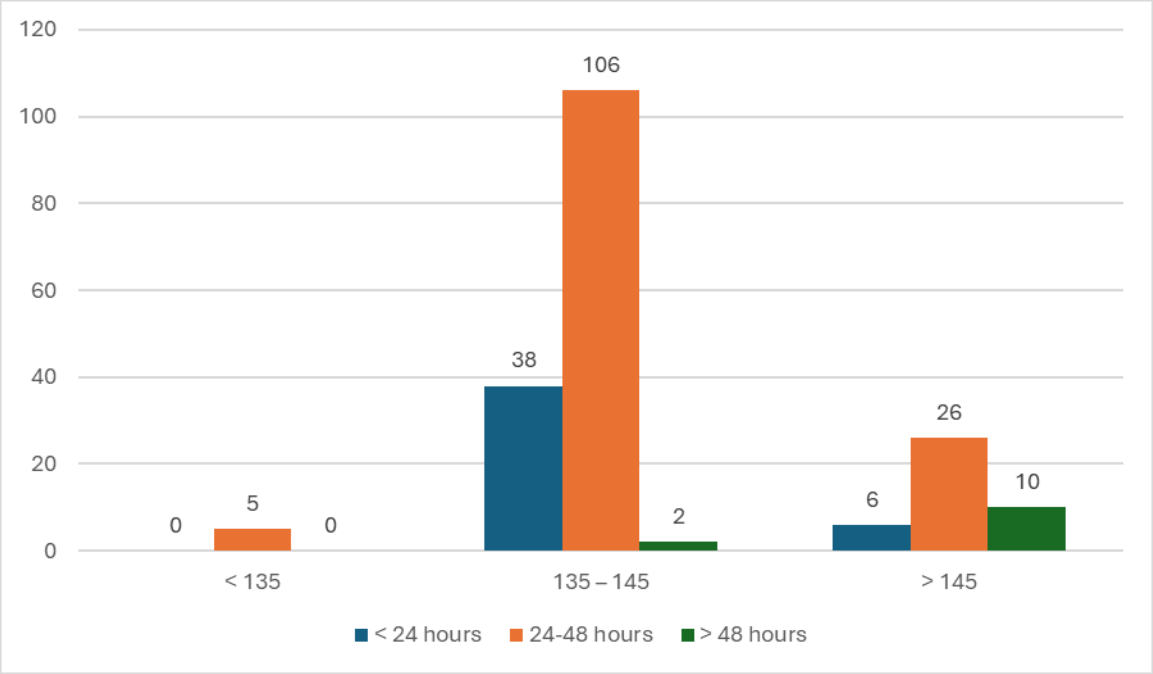
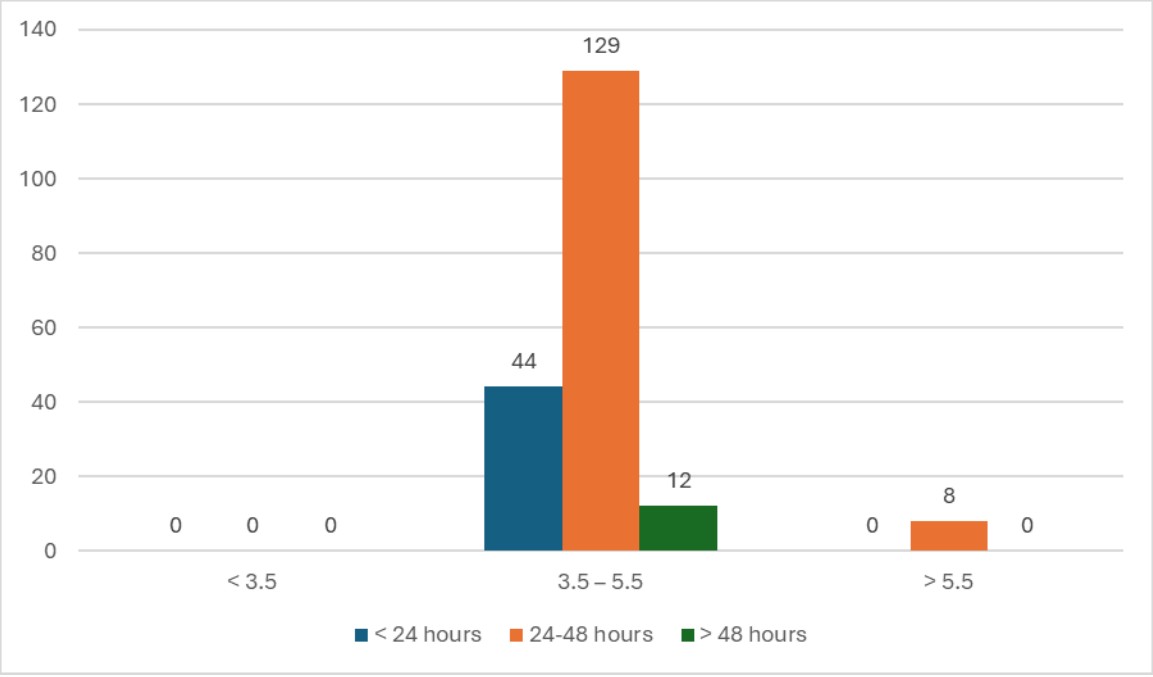


Table 16: Association of post phototherapy potassium and duration of phototherapy

Potassium	< 24 hours	24-48 hours	> 48 hours	Total	P value
< 3.5	0	0	0	0	<0.001
3.5 – 5.5	44 (100 %)	129 (94 %)	12(100 %)	185 (95.8%)	
> 5.5	0	8 (6 %)	0	8 (4.3%)	

Figure 19: Graph representing association of post phototherapy potassium in relation to duration of phototherapy of study population



DISCUSSION

DISCUSSION

This study was conducted among 193 newborns who underwent phototherapy for unconjugated hyperbilirubinemia from 24 hours of life to 14 days of life. The electrolyte changes were studied before and after phototherapy

Table 17: Gender distribution in various studies

		This study	Reddy et al.(120)	Alizadeh-Taheri et al(118)	Karamifar et al(121)
Gender	Males	54.9%	59.1%	50.1%	62.9

This study found that males made up the majority of the participants who underwent phototherapy, accounting for 54.9% of the total, in comparison to females. Men made up 59.1% of the participants in the research carried out by Reddy et al., 50.1% of the participants in the research carried out by Alizadeh-Taheri et al., and 62.9% of the participants in the research carried out by Karamifar et al.

Table 18: Gestational age distribution in various studies

		This study	Reddy et al.(120)	Yadav et al(117)	Arora et al.(119)	Karamifar et al(121)
Gestational age	37 – 40 weeks	59.1%	77.0%	60.0%	54.0%	59.4%

In this particular study, the bulk of the participants were full-term babies, which accounted for 59.1% of the total. When compared to the percentage that was found in the Karamifar et al trial, which was 59.4%, the percentage that was found in the Reddy et al study was 77.0%. In the study conducted by Arora et al., full-term babies made up 54.0% of the individuals. 60.0% of the babies in the Yadav et al. trial were born at full term.

Table 19: Low birth weight distribution in various studies

		This study	Reddy et al.(120)
Low birth weight	<2.5Kg	42.2%	23%

In our study, 42.2% of infants were born with a low birth weight, compared to 23% reported in the study by Reddy et al.,

Table 20: Calcium – post phototherapy distribution in various studies

		This study	Reddy et al.(120)	Yadav et al(117)	Alizadeh-Taheri et al(118)	Arora et al.(119)	Karamifar et al(121)
Calcium – post phototherapy	<8 g/dl	24.0%	<7g/dl - 13.1%	73.3%	56.0%	50.0%	14.4%

It was shown that 5.6% of the participants in this study had hypocalcemia prior to receiving phototherapy, but after receiving PT, the incidence of low calcium levels jumped to 24.0%. The incidence of post-PT hypocalcemia was 13.0% in the study that was carried out by Reddy et al., but the incidence of low calcium level was on the higher side in the study that was carried out by Yadav et al., with 73.3% of the participants having the same condition. The incidence of hypocalcaemia was found to be 56.0% in the study conducted by Alizadeh-Taheri and colleagues, while the incidence of hypocalcaemia was found to be 50.0% in the study conducted by Arora and colleagues. According to the findings of the study conducted by Karamifar et al., the incidence of hypocalcemia following phototherapy was reduced by 14.4%.

Table 21: Sodium – post phototherapy distribution in various studies

		This study	Reddy et al.(120)
Sodium – post phototherapy	<135	3.5%	6.0%

According to the findings of this research, 78.8 percent of the babies had normal sodium levels, and just 1.4% of them had hyponatremia prior to the phototherapy session. Following the completion of phototherapy, the rate of hyponatremia stood at 3.5%. Nearly twice as many cases were reported prior to the implementation of phototherapy. In the research carried out by Reddy and colleagues, the percentage of patients who experienced hyponatremia after phototherapy was 6.0%.

Table 22: Potassium – post phototherapy distribution in various studies

		This study	Reddy et al.(120)
Potassium – post phototherapy	<3.5 and > 5.5	0.0 and 4.2%	<3.5 - 0.4%

Before beginning the phototherapy sessions, none of the neonates who participated in this trial had hypokalemia or hyperkalemia conditions. After phototherapy, around 4.2% of neonates were found to have hyperkalemia. The results of our analysis did not reveal any instances of hypokalemia. Following phototherapy, there were 0.4% of cases of hypokalemia that were documented in the study that was conducted by Reddy and colleagues.

Phototherapy was associated with a significant overall increase in the incidence of hypocalcemia. Notably, low birth weight infants exhibited a significantly higher incidence of hypocalcemia compared to those with normal birth weight ($p < 0.001$), with the difference in calcium levels between the two groups also reaching statistical significance ($p = 0.048$). This study demonstrated that calcium levels varied significantly based on birth weight, with LBW infants tending to have lower calcium levels. Similarly , in the study by Reddy et al., hypocalcemia following phototherapy was more prevalent among LBW infants (36.2%) compared to normal birth weight infants (6.2%). Therefore, it may be deduced that newborns with a LBW were at a greater risk of hypocalcemia after receiving phototherapy than babies with a normal birth weight.

The levels of calcium in neonates who were born prematurely, those born at term, and those born after term were shown to be significantly different after phototherapy ($p<0.001$). Neonatal patients who were born prematurely were more likely to have hypocalcemia than those who were born at term. According to the research conducted by Reddy and colleagues, there was a statistically significant difference in the incidence of hypocalcemia following phototherapy intervention between preterm and late infants. Following phototherapy, the occurrence of hypocalcemia was found to be significantly higher in before term newborns (41.2%) compared to term newborns (6.2%) on average. As a result, it may be deduced that preterm infants suffered from a higher probability of hypocalcemia after receiving phototherapy than term infants. The study by Yadav et al. found no significant difference in serum calcium levels between the study and control groups prior to phototherapy. After 48 hours of phototherapy , a significant reduction in serum calcium levels was observed among preterm infants in the study group ($p<0.0001$) .Term neonates also exhibited a statistically significant decline in calcium levels following phototherapy ($p<0.005$). In the study by Arora et al., hypocalcemia was detected in almost half of the preterm infants (43%) and 30 of 54 term newborns (56%) after 2 days of continuous PT.

Following phototherapy, serum sodium levels changed, and our research revealed that there were substantial changes between preterm, term, and post-term newborns in terms of sodium production. The statistical significance of the difference was also found to be less than 0.001.0. The investigation that was conducted by Reddy and colleagues also revealed results that were substantially comparable. Preterm neonates had a higher incidence of hyponatremia following phototherapy (17.6%) compared to term neonates (3.1%), who had a comparatively lower incidence. Based on this, it can be deduced that preterm newborns were more likely to experience hyponatremia after receiving phototherapy than term babies.

In our study the variation in potassium levels with respect to gestational age was not statistically significant . Reddy et al, study also couldn’t find any significant difference in the potassium levels based on gestational age.

CONCLUSION

CONCLUSION

Among the 193 samples of neonates who received phototherapy, incidence of hyponatremia was 3.5% . This was notably higher in preterm (8.4%) and LBW (8.4%) compared to term newborns (2.3%) and those with normal birth weight.

Incidence of hypocalcemia is also significantly varied between gestational age of the newborns and weight of the newborns. The incidence of hypocalcemia was 24.0% in this study. Preterm neonates and low birth weight babies tend to have higher incidence of hypocalcemia.

After phototherapy, around 4.2% of neonates were found to have hyperkalemia. The results of our analysis did not reveal any instances of hypokalemia.

LIMITATIONS

LIMITATIONS

- Further follow ups were not done for the neonates post discharge from hospital.
- It is a single centre study and a multicentre study can reveal more information on the same topic
- Time limitations restricted the further follow up and collecting more samples for the study.

SUMMARY

SUMMARY

This study was conducted among 193 newborns who underwent phototherapy for unconjugated hyperbilirubinemia from 24 hours of life to 14 days of life. The electrolyte changes were studied before and after phototherapy

- This study found that males made up the majority of the participants who underwent phototherapy, accounting for 54.9% of the total, in comparison to females.
- In this particular study, the bulk of the participants were full-term babies, which accounted for 59.1% of the total.
- In our study, 42.2% of infants were born with a low birth weight
- It was shown that 5.6% of the participants in this study had hypocalcemia prior to receiving phototherapy, but after receiving phototherapy, the incidence of hypocalcemia jumped to 24.0%.
- According to the findings of this research, 78.8 percent of the babies had normal sodium levels, and just 1.4% of them had hyponatremia prior to the phototherapy session. Following the completion of phototherapy, the rate of hyponatremia stood at 3.5%.
- Before beginning the phototherapy sessions, none of the neonates who participated in this trial had hypokalemia or hyperkalemia conditions. After phototherapy, around 4.2% of neonates were found to have hyperkalemia.
- the incidence of hypocalcemia in LBW babies was significantly higher compared to the incidence in normal weight babies ($p < 0.001$).
- The levels of calcium in neonates who were born prematurely, those born at term, and those born after term were shown to be significantly different after phototherapy ($p<0.001$)
- There were substantial changes between preterm, term, and post-term newborns in terms of sodium production
- The change in potassium levels based on gestational age was not statistically significant .

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ANNEXURE

INFORMED CONSENT FORM

Date:

I, Mr/Mrs _____ have been explained in my own vernacular language that my child will be included in **"A PROSPECTIVE HOSPITAL BASED OBSERVATIONAL STUDY ON ELECTROLYTE CHANGES FOLLOWING PHOTOTHERAPY IN NEONATAL HYPERBILIRUBINEMIA"** hereby I give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters. The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow my child as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo the procedure, undergo investigations, and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose, the operation / procedure, etc may be video graphed or photographed. All the expenses needed for the study will be bored by principal investigator. All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

_____

(Signature/Thumb impression & Name of the Guardian)

(Relation with patient)

Witness:

(Signature & Name of Research person /doctor)

ಮಾಹಿತಿಯುಕ್ತ ಸಮ್ಮತಿ ನಮೂನೆ

ದಿನಾಂಕ:

ನನಗೆ, ಶ್ರೀ/ಶ್ರೀಮತಿ _____ ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ನನ್ನ ಮಗುವನ್ನು "ನವಜಾತ ಹೈಪರ್ಬಿಲಿರುಬಿನೆಮಿಯಾದಲ್ಲಿ ಫೋಟೊಥೆರಪಿ ನಂತರ ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಬದಲಾವಣೆಗಳ ಕುರಿತು ನಿರೀಕ್ಷಿತ ಆಸ್ಪತ್ರೆ ಆಧಾರಿತ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ "ದಲ್ಲಿ ಸೇರಿಸಲಾಗುವುದು ಎಂದು ನಾನು ಈ ಮೂಲಕ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಇದರಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ . ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ . ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಸಿಕ್ಕಿದೆ . ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ . ನನ್ನ ಮಗುವನ್ನು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಲು ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ . ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು , ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು , ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಸಮ್ಮತಿಸುತ್ತೇನೆ . ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ, ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊ ಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು . ಅಧ್ಯಯನಕ್ಕೆ ಬೇಕಾದ ಎಲ್ಲಾ ವೆಚ್ಚಗಳನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಬಳಸಬಹುದು . ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಉಂಟಾಗುವ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

(ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು ಮತ್ತು ರಕ್ಷಕರ ಹೆಸರು)

(ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ)

ಸಾಕ್ಷಿ:

(ಸಹಿ ಮತ್ತು ಸಂಶೋಧನಾ ವ್ಯಕ್ತಿ/ವೈದ್ಯರ ಹೆಸರು)

PATIENT INFORMATION SHEET

Principal investigator: Dr. NANDANURI VARSHA REDDY

I Dr. VARSHA REDDY Post graduate student in Department at Sri Devraj Urs Medical College, will be conducting a study titled **"A PROSPECTIVE HOSPITAL BASED OBSERVATIONAL STUDY ON ELECTROLYTE CHANGES FOLLOWING PHOTOTHERAPY IN NEONATAL HYPERBILIRUBINEMIA"** for my dissertation under the guidance of Dr. KRISHNAPPA. J, Professor of Department of Paediatrics. You will not be paid any financial compensation for the participation of your child in this research project. All the expenses needed for the study will be bored by principal investigator. All the data will be kept confidential and will be used only for research purpose by this institution. You are free to provide consent for the participation of your child in this study. You can also withdraw your child from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator

Date-

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ನಂದನೂರಿ ವರ್ಷಾ ರೆಡ್ಡಿ

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿನಿ ಡಾ. ವರ್ಷಾ ರೆಡ್ಡಿ, ನನ್ನ ಪ್ರಬಂಧಕ್ಕಾಗಿ "ನವಜಾತ ಶಿಶುಗಳ ಹೈಪರ್ಬಿಲಿರುಬಿನೆಮಿಯಾದಲ್ಲಿ ಫೋಟೊಥೆರಪಿ ನಂತರ ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಬದಲಾವಣೆಗಳ ಕುರಿತು ನಿರೀಕ್ಷಿತ ಆಸ್ಪತ್ರೆ ಆಧಾರಿತ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ. ಮಕ್ಕಳ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕರಾದ ಡಾ. ಕೃಷ್ಣಪ್ಪ ಜೆ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ. ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪರಿಹಾರವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ. ಅಧ್ಯಯನಕ್ಕೆ ಅಗತ್ಯವಿರುವ ಎಲ್ಲಾ ವೆಚ್ಚಗಳನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನೀವು ಒಪ್ಪಿಗೆ ನೀಡಲು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವನ್ನು ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿನ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ನಿಮ್ಮನ್ನು ಹಾನಿಗೊಳಿಸುವುದಿಲ್ಲ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-

PROFORMA

Name :

Date of Birth:

Sex:

Gestational age:

Term / preterm:

Informant :

Birth weight :

Address: ……...Telephone No……

MATERNAL HISTORY -

Obstetric score -

Mother Blood Group :

Maternal risk factors (If any)

Mode of delivery -

BABY DETAILS -

Baby Blood Group :

Total serum bilirubin level:

Sample collected at _____ hrs. of life.

Threshold value according to AAP nomogram -

Whether in phototherapy range - Yes / No

	Pre phototherapy	Post phototherapy	DIFFERENCE
Sodium			
Potassium			
Calcium			
Total bilirubin			
Direct bilirubin			

MASTER CHART

MASTER CHART

SL NO	NAME OF THE PATIENT	SEX	UHID NO	GESTATIONAL AGE	BABY WEIGHT	MOTHER BLOOD GROUP	BABY BLOOD GROUP	DURATION OF PHOTOTHERAPY	SERUM TOTAL BILIRUBIN	PRE PT NA	PRE PT K	PRE PT CA	POST TB	POST PT NA	POST PT K	POST PT CA	
		MALE 1/ FEMALE 2		LPT:1, TERM:2, POSTTERM :3	LBW:1 , NORMAL :2			<24 HRS:1, 24-48HRS :2 , >48 HRS:3		LOW:1, NORMAL:2, HIGH:3	LOW:1, NORMAL:2, HIGH:3	LOW:1, NORMAL:2, HIGH:3		LOW:1, NORMAL:2, HIGH:3	LOW:1, NORMAL:2, HIGH:3	LOW:1, NORMAL:2, HIGH:3	
1	B/O MALLIKA	MALE	228252	TERM	LBW	B POSITIVE	A POSITIVE	24-48HRS	16	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	LOW	
2	B/O PADMA	FEMALE	228448	LPT	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	LOW	
3	B/O SAWTHI	FEMALE	228461	PT	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	14.4	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
4	B/O PRIYANKA	MALE	229867	TERM	LBW	A POSITIVE	A POSITIVE	<24HRS	18.4	HIGH	NORMAL	NORMAL	11	HIGH	NORMAL	NORMAL	
5	B/O AFREEN ANJUM	MALE	232357	LPT	LBW	A POSITIVE	A POSITIVE	24-48HRS	19.2	LOW	NORMAL	NORMAL	12	NORMAL	HIGH	LOW	
6	B/O BHANU	FEMALE	232392	PT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
7	B/O SARITHA	MALE	232574	TERM	LBW	A POSITIVE	A POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL	
8	B/O KRISHNAVENI	MALE	233929	TERM	LBW	B POSITIVE	B POSITIVE	24-48HRS	18.6	HIGH	NORMAL	NORMAL	9	HIGH	NORMAL	NORMAL	
9	B/O ASHWINI	MALE	233923	TERM	NORMAL	A POSITIVE	B POSITIVE	>48HRS	21	NORMAL	NORMAL	NORMAL	6	NORMAL	NORMAL	NORMAL	
10	B/O PRIYANKA	MALE	233939	LPT	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	19	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
11	B/O SHARADHA	FEMALE	235415	TERM	LBW	A POSITIVE	A POSITIVE	<24HRS	18.8	NORMAL	NORMAL	LOW	10	HIGH	NORMAL	NORMAL	
12	B/O NOOORAIN TAJ	MALE	235800	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
13	B/O VANDHAN	MALE	236780	TERM	LBW	O POSITIVE	B POSITIVE	<24HRS	18	NORMAL	NORMAL	NORMAL	13	HIGH	NORMAL	NORMAL	

	A															
14	B/O ARATHI	FEMALE	236946	LPT	LBW	B POSITIVE	A POSITIVE	24-48HRS	20.6	NORMAL	NORMAL	NORMAL	11	LOW	NORMAL	LOW
15	B/O PAVITHRA	FEMALE	237159	TERM	NORMAL	A POSITIVE	O POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL
16	B/O RAMYA	MALE	241099	TERM	LBW	O POSITIVE	B POSITIVE	<24HRS	20.8	HIGH	NORMAL	NORMAL	13	NORMAL	NORMAL	LOW
17	B/O CHANDHANA	FEMALE	241128	TERM	LBW	B POSITIVE	A POSITIVE	24-48HRS	20.4	NORMAL	NORMAL	NORMAL	12	HIGH	NORMAL	NORMAL
18	B/O MONISHA	MALE	242575	LPT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	17	NORMAL	NORMAL	LOW	11	NORMAL	NORMAL	NORMAL
19	B/O RANJITHA	MALE	242664	LPT	LBW	O POSITIVE	O POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	NORMAL	13	HIGH	HIGH	LOW
20	B/O SOWMYA	MALE	243840	TERM	LBW	O POSITIVE	O POSITIVE	>48HRS	22	HIGH	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL
21	B/O MASUDHA	MALE	243991	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	20.4	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	LOW
22	B/O GAYATHRI	MALE	245353	TERM	NORMAL	O POSITIVE	O POSITIVE	<24HRS	19.4	NORMAL	NORMAL	NORMAL	13	NORMAL	NORMAL	NORMAL
23	B/O AMREEN ANJUM	FEMALE	245459	LPT	LBW	A POSITIVE	O POSITIVE	24-48HRS	20.2	NORMAL	NORMAL	NORMAL	14	LOW	NORMAL	LOW
24	B/O CHANDRIKA	FEMALE	246355	PT	LBW	O POSITIVE	A POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	12	HIGH	NORMAL	NORMAL
25	B/O LAKSHMI	FEMALE	346429	TERM	NORMAL	A POSITIVE	B POSITIVE	>48HRS	19.4	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL
26	B/O SURYA BANU	MALE	249307	LPT	LBW	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	LOW
27	B/O RANJITHA	MALE	249262	PT	LBW	O POSITIVE	O POSITIVE	24-48HRS	18.4	NORMAL	NORMAL	NORMAL	8.4	HIGH	NORMAL	NORMAL
28	B/O PREMA	FEMALE	250741	LPT	NORMAL	B POSITIVE	O POSITIVE	<24HRS	20.4	NORMAL	NORMAL	NORMAL	12.5	NORMAL	NORMAL	NORMAL
29	B/O ARCHANA	FEMALE	250768	TERM	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL
30	B/O MOUNIKA	MALE	250785	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	12	HIGH	NORMAL	LOW	12.5	NORMAL	NORMAL	NORMAL
31	B/O SIREESHA	MALE	252416	LPT	LBW	A POSITIVE	O POSITIVE	>48HRS	21.4	LOW	NORMAL	NORMAL	14	HIGH	NORMAL	LOW
32	B/O SHWETHA	FEMALE	253432	TERM	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	22	HIGH	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL
33	B/O UMMETHANI	MALE	254918	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	0.6	NORMAL	NORMAL	NORMAL

34	B/O PRIYADHARSHINI	MALE	258289	TERM	LBW	B POSITIVE	O POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	12.3	HIGH	NORMAL	NORMAL	
35	B/O SHILPA	FEMALE	258270	LPT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	21.4	NORMAL	NORMAL	NORMAL	12.3	NORMAL	HIGH	NORMAL	
36	B/O PAVITHRA	FEMALE	258355	TERM	LBW	O POSITIVE	A POSITIVE	>48HRS	21.6	NORMAL	NORMAL	NORMAL	11.2	NORMAL	NORMAL	LOW	
37	B/O VARALSKMI	MALE	258375	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	17	NORMAL	NORMAL	NORMAL	11.3	NORMAL	NORMAL	NORMAL	
38	B/O SONIA	MALE	259620	TERM	LBW	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	10.6	HIGH	NORMAL	NORMAL	
39	B/O VARALSKAMI	MALE	259479	LPT	LBW	O POSITIVE	A POSITIVE	<24HRS	21.2	NORMAL	NORMAL	NORMAL	11.6	HIGH	NORMAL	NORMAL	
40	B/O MUSKAN	MALE	261121	TERM	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	18.8	NORMAL	NORMAL	NORMAL	11.3	NORMAL	NORMAL	NORMAL	
41	B/O MAJEERA	FEMALE	262302	PT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	15.6	HIGH	NORMAL	NORMAL	
42	B/O MUBEEN TAJ	FEMALE	258133	TERM	LBW	A POSITIVE	A POSITIVE	>48HRS	14	HIGH	NORMAL	NORMAL	12.5	NORMAL	NORMAL	NORMAL	
43	B/O RANJITHA	MALE	264927	PT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	19.4	HIGH	NORMAL	NORMAL	11.8	NORMAL	NORMAL	NORMAL	
44	B/O SHALINI	MALE	286094	TERM	LBW	A POSITIVE	A POSITIVE	<24HRS	17	NORMAL	NORMAL	NORMAL	10.8	NORMAL	NORMAL	NORMAL	
45	B/O NAGIMA TAJ	MALE	283572	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	15	NORMAL	NORMAL	LOW	8.6	HIGH	NORMAL	NORMAL	
46	B/O KALAVATHO	FEMALE	283576	LPT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	6.8	NORMAL	NORMAL	LOW	
47	B/O SUBHA	FEMALE	284870	TERM	LBW	O POSITIVE	B POSITIVE	>48HRS	21	NORMAL	NORMAL	NORMAL	8.9	HIGH	NORMAL	NORMAL	
48	B/O ASHABHAI	MALE	283532	LPT	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	20.6	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	LOW	
49	B/O NAGMA TAJ	MALE	283572	TERM	LBW	O POSITIVE	O POSITIVE	<24HRS	12	NORMAL	NORMAL	NORMAL	9	HIGH	NORMAL	LOW	
50	B/O SUMIYA TAJ	MALE	284441	TERM	LBW	AB POSITIVE	A POSITIVE	24-48HRS	17	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
51	B/O PREMA	FEMALE	284642	PT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
52	B/O SIREESHA	FEMALE	284433	TERM	LBW	O POSITIVE	O POSITIVE	>48HRS	19	NORMAL	NORMAL	LOW	0.2	HIGH	NORMAL	NORMAL	

53	B/O NABEENA	MALE	284868	PT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	18.2	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
54	B/O AYESHA	MALE	283494	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	12	HIGH	NORMAL	NORMAL	15	NORMAL	HIGH	NORMAL	
55	B/O NAMEERA	MALE	284775	LPT	LBW	B POSITIVE	B POSITIVE	24-48HRS	15	HIGH	NORMAL	NORMAL	12	LOW	NORMAL	LOW	
56	B/O MUSRATH	MALE	287701	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	19	NORMAL	NORMAL	NORMAL	14	NORMAL	NORMAL	NORMAL	
57	B/O SUNITHA	FEMALE	286181	TERM	NORMAL	B POSITIVE	O POSITIVE	<24HRS	19	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
58	B/O NAGMA TAJ	FEMALE	286139	LPT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	19	NORMAL	NORMAL	NORMAL	10.2	NORMAL	NORMAL	LOW	
59	B/O MANJULA	FEMALE	286133	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	14.4	NORMAL	NORMAL	NORMAL	8.6	NORMAL	NORMAL	NORMAL	
60	B/O DIVYASREE	MALE	282463	LPT	NORMAL	AB POSITIVE	B POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
61	B/O RENUSREE	MALE	286215	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	19.4	NORMAL	NORMAL	NORMAL	.	HIGH	NORMAL	NORMAL	
62	B/O SUMITGHRA	FEMALE	287965	TERM	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	15	NORMAL	HIGH	NORMAL	
63	B/O THASMIA	FEMALE	289035	LPT	LBW	B POSITIVE	O POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	12	HIGH	NORMAL	LOW	
64	B/O VARALAKSHMI	FEMALE	287623	LPT	LBW	O POSITIVE	A POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	LOW	8	HIGH	NORMAL	LOW	
65	B/O MEHERTAJ	FEMALE	289026	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	12.5	NORMAL	NORMAL	NORMAL	
66	B/O HAJIRA BHANU	MALE	284866	TERM	LBW	O POSITIVE	A POSITIVE	<24HRS	22	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
67	B/O PAVITHRA	MALE	287835	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
68	B/O KHATHIJA KUBRA	MALE	287842	TERM	LBW	A POSITIVE	B POSITIVE	24-48HRS	19.2	HIGH	NORMAL	NORMAL	14	LOW	NORMAL	NORMAL	
69	B/O KAVTHA	FEMALE	287840	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	19.2	NORMAL	NORMAL	NORMAL	11.6	NORMAL	NORMAL	NORMAL	
70	B/O PAVITHRA	MALE	287881	LPT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	21.6	NORMAL	NORMAL	NORMAL	8.6	NORMAL	NORMAL	LOW	
71	B/O ASHA	FEMALE	287641	TERM	LBW	O POSITIVE	B POSITIVE	<24HRS	14	HIGH	NORMAL	NORMAL	8.8	NORMAL	NORMAL	LOW	
72	B/O RABIYA	MALE	287764	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	10.2	NORMAL	NORMAL	NORMAL	
73	B/O	MALE	297601	TERM	LBW	O POSITIVE	B POSITIVE	24-48HRS	14.4	NORMAL	NORMAL	NORMAL	12	HIGH	NORMAL	NORMAL	

	RAMADEVI																
74	B/O SUSHMITHA	MALE	290623	TERM	LBW	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	LOW	
75	B/O PREMA	FEMALE	296024	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	8.6	NORMAL	NORMAL	NORMAL	
76	B/O SOWNDHRAYA	MALE	296742	TERM	NORMAL	A POSITIVE	A POSITIVE	>48HRS	16	NORMAL	NORMAL	NORMAL	10.2	NORMAL	NORMAL	NORMAL	
77	B/O KALAVATHI	FEMALE	296174	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	14.4	NORMAL	NORMAL	NORMAL	8.6	NORMAL	NORMAL	NORMAL	
78	B/O PAVITHRA	MALE	267721	LPT	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	14	HIGH	NORMAL	NORMAL	11.3	NORMAL	NORMAL	LOW	
79	B/O KASTURI	MALE	288671	TERM	NORMAL	A POSITIVE	B POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	8.9	NORMAL	NORMAL	NORMAL	
80	B/O BHAVANI	MALE	294901	TERM	LBW	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	7.4	NORMAL	NORMAL	NORMAL	
81	B/O AYESHA	FEMALE	294991	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	5.6	NORMAL	NORMAL	NORMAL	
82	B/O SWAPNA	MALE	294024	LPT	LBW	B POSITIVE	O POSITIVE	<24HRS	14	HIGH	NORMAL	NORMAL	8.9	HIGH	NORMAL	NORMAL	
83	B/O DEEPA	MALE	294021	PT	LBW	A POSITIVE	O POSITIVE	24-48HRS	12.4	NORMAL	NORMAL	NORMAL	5.6	NORMAL	NORMAL	NORMAL	
84	B/O VARALSKAMI	MALE	294481	TERM	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	8.6	NORMAL	NORMAL	NORMAL	
85	B/O LAKSHMI	FEMALE	294682	LPT	NORMAL	A POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	9.5	NORMAL	NORMAL	NORMAL	
86	B/O KAVITHA	FEMALE	294666	LPT	LBW	B POSITIVE	A POSITIVE	24-48HRS	16	NORMAL	NORMAL	NORMAL	8.8	HIGH	NORMAL	LOW	
87	B/O SHI[PA	FEMALE	296141	TERM	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	9.6	NORMAL	NORMAL	NORMAL	
88	B/O ARBIN TAJ	MALE	297741	TERM	LBW	O POSITIVE	O POSITIVE	<24HRS	14.4	NORMAL	NORMAL	NORMAL	8	HIGH	NORMAL	NORMAL	
89	B/O JANAKI	MALE	300833	TERM	LBW	A POSITIVE	A POSITIVE	24-48HRS	18.4	HIGH	NORMAL	NORMAL	11	HIGH	NORMAL	NORMAL	
90	B/O MALA	MALE	300765	LPT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	19.2	HIGH	NORMAL	NORMAL	11.2	NORMAL	HIGH	NORMAL	
91	B/O CHANDRIKA	MALE	300839	LPT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	8.9	NORMAL	NORMAL	LOW	
92	B/O UMAVATHI	FEMALE	302270	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL	

93	B/O SMITHA	MALE	302267	PT	NORMAL	B POSITIVE	B POSITIVE	<24HRS	18.6	NORMAL	NORMAL	NORMAL	7	NORMAL	NORMAL	NORMAL	
94	B/O ROOPA	MALE	302264	TERM	LBW	A POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	9.6	NORMAL	NORMAL	NORMAL	
95	B/O ARSHITHA	MALE	306874	TERM	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	19	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL	
96	B/OSUJATHA	FEMALE	306927	LPT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	18.8	HIGH	NORMAL	NORMAL	7.5	NORMAL	NORMAL	LOW	
97	B/O GEETHA	FEMALE	302260	TERM	LBW	O POSITIVE	A POSITIVE	<24HRS	21	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	NORMAL	
98	B/O NIRMALA	MALE	306873	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL	
99	B/O ARCHITHA	FEMALE	306874	TERM	NORMAL	B POSITIVE	A POSITIVE	24-48HRS	20.6	NORMAL	NORMAL	NORMAL	7	NORMAL	NORMAL	NORMAL	
100	B/O SUJATHA	MALE	306927	PT	NORMAL	A POSITIVE	O POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	NORMAL	6.5	NORMAL	NORMAL	NORMAL	
101	B/O GAYATHRI	MALE	308019	TERM	NORMAL	O POSITIVE	B POSITIVE	<24HRS	20.8	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
102	B/O SUWARNA	FEMALE	309685	TERM	NORMAL	B POSITIVE	A POSITIVE	24-48HRS	20.4	NORMAL	NORMAL	LOW	18	NORMAL	NORMAL	LOW	
103	B/O RAMYA	FEMALE	314128	TERM	LBW	O POSITIVE	A POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	12	HIGH	NORMAL	NORMAL	
104	B/O KULFAM	MALE	311787	LPT	LBW	O POSITIVE	O POSITIVE	<24HRS	21.2	NORMAL	NORMAL	NORMAL	14	HIGH	NORMAL	NORMAL	
105	B/OMUBEEN	FEMALE	314134	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
106	B/O LAVNAYA	FEMALE	314100	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	20.4	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
107	B/O SAHANA	MALE	315307	TERM	NORMAL	O POSITIVE	O POSITIVE	<24HRS	19.4	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
108	B/O UMAVATHI	MALE	319619	PT	NORMAL	A POSITIVE	O POSITIVE	24-48HRS	20.2	NORMAL	NORMAL	NORMAL	15	NORMAL	NORMAL	NORMAL	
109	B/O BHABYAGMMA	FEMALE	315335	LPT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	17	NORMAL	NORMAL	NORMAL	14	NORMAL	NORMAL	NORMAL	
110	B/P KAVYA	FEMALE	319677	TERM	NORMAL	A POSITIVE	B POSITIVE	24-48HRS	19.4	HIGH	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
111	B/O SAVITHA	MALE	322920	LPT	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	NORMAL	
112	B/O ANUSHA	MALE	324114	TERM	NORMAL	O POSITIVE	O POSITIVE	<24HRS	18.4	NORMAL	NORMAL	NORMAL	7	NORMAL	NORMAL	NORMAL	
113	B/O DHARSHINI	FEMALE	324082	TERM	NORMAL	B POSITIVE	O POSITIVE	<24HRS	20.4	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
114	B/O DIVYASRE	FEMALE	323394	TERM	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	LOW	

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115	B/O ARUNAMMA	FEMALE	324763	LPT	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	12	NORMAL	NORMAL	NORMAL	6	NORMAL	NORMAL	LOW	
116	B/O RAKSHITHA	MALE	327531	PT	NORMAL	A POSITIVE	O POSITIVE	>48HRS	21.4	NORMAL	NORMAL	NORMAL	15	NORMAL	NORMAL	NORMAL	
117	B/O SUSHMA	MALE	327606	TERM	LBW	B POSITIVE	O POSITIVE	24-48HRS	22	HIGH	NORMAL	NORMAL	15	NORMAL	NORMAL	NORMAL	
118	B/O VEENA	MALE	332597	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	15	NORMAL	NORMAL	NORMAL	
119	B/O MARIYA	FEMALE	333383	TERM	NORMAL	B POSITIVE	O POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
120	B/O RADHA	FEMALE	334308	TERM	LBW	O POSITIVE	A POSITIVE	24-48HRS	21.4	NORMAL	NORMAL	LOW	12	HIGH	NORMAL	LOW	
121	B/O SHABREEN	MALE	335626	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	21.6	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	
122	B/O NANDHINI	MALE	336494	LPT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	12	NORMAL	NORMAL	LOW	
123	B/O KOUSER	FEMALE	339794	PT	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
124	B/O ROOPA	MALE	339858	TERM	LBW	O POSITIVE	A POSITIVE	<24HRS	21.2	HIGH	NORMAL	NORMAL	14	NORMAL	NORMAL	NORMAL	
125	B/O SHRUTHI	MALE	340224	TERM	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	18.8	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
126	B/O BINDHU	FEMALE	339341	LPT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
127	B/O SOUBHAGYA	MALE	340222	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
128	B/O KUMARI	MALE	343810	TERM	NORMAL	A POSITIVE	A POSITIVE	<24HRS	19.4	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
129	B/O SOWNDHARYA	MALE	344823	PT	LBW	A POSITIVE	A POSITIVE	24-48HRS	17	NORMAL	NORMAL	NORMAL	9	HIGH	NORMAL	NORMAL	
130	B/O DIVYARANI	FEMALE	346318	LPT	LBW	O POSITIVE	O POSITIVE	24-48HRS	15	HIGH	NORMAL	NORMAL	9	NORMAL	NORMAL	LOW	
131	B/O AMREEN TAJ	FEMALE	343292	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
132	B/O MOUNIKA	MALE	347237	TERM	LBW	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	14	NORMAL	NORMAL	LOW	
133	B/O NAZIYA	MALE	346411	LPT	NORMAL	B POSITIVE	B POSITIVE	<24HRS	20.6	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
13	B/O	FEMALE	347236	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	12	HIGH	NORMAL	NORMAL	10	NORMAL	NORMAL	NORMAL	

4	MOUNISHA																
135	B/O VINUTHA	FEMALE	347812	TERM	NORMAL	AB POSITIVE	A POSITIVE	24-48HRS	17	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	LOW	
136	B/O SHYAMALA	FEMALE	347781	TERM	LBW	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	15	LOW	NORMAL	NORMAL	
137	B/O ANITHA	FEMALE	351846	TERM	NORMAL	O POSITIVE	O POSITIVE	<24HRS	19	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
138	B/O NANDHINI	FEMALE	356128	TERM	LBW	O POSITIVE	A POSITIVE	24-48HRS	21.2	HIGH	NORMAL	NORMAL	15	NORMAL	NORMAL	NORMAL	
139	B/O RESHMA	FEMALE	354611	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	12	NORMAL	NORMAL	NORMAL	8	HIGH	NORMAL	NORMAL	
140	B/O ARATHI	FEMALE	356697	TERM	NORMAL	B POSITIVE	B POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	16.2	NORMAL	NORMAL	NORMAL	
141	B/O JANMESHA	FEMALE	359879	TERM	LBW	O POSITIVE	O POSITIVE	24-48HRS	19	HIGH	NORMAL	NORMAL	11	NORMAL	NORMAL	LOW	
142	B/O PAVITHRA	FEMALE	356124	TERM	NORMAL	B POSITIVE	O POSITIVE	<24HRS	19	NORMAL	NORMAL	NORMAL	14	NORMAL	NORMAL	NORMAL	
143	B/O VEDHAVATHI	FEMALE	523848	TERM	LBW	B POSITIVE	A POSITIVE	<24HRS	16	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
144	B/O BHANUMATHI	MALE	523547	TERM	LBW	B POSITIVE	B POSITIVE	<24HRS	15	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
145	B/O HEMAVATHI	FEMALE	522470	PT	LBW	O POSITIVE	O POSITIVE	24-48HRS	14.4	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	LOW	
146	B/O ARBINA	MALE	524095	TERM	LBW	A POSITIVE	A POSITIVE	<24HRS	18.4	HIGH	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
147	B/O LALITHA	FEMALE	523789	TERM	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	19.2	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
148	B/O SUPRITHA	MALE	524799	LPT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
149	B/O BHAVANI	FEMALE	524077	PT	LBW	A POSITIVE	A POSITIVE	<24HRS	20	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	LOW	
150	B/O DEEPA	MALE	525792	TERM	LBW	B POSITIVE	B POSITIVE	24-48HRS	18.6	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	NORMAL	
151	B/O KAVITHA	MALE	526003	PT	LBW	A POSITIVE	B POSITIVE	24-48HRS	21	HIGH	NORMAL	NORMAL	6	NORMAL	NORMAL	NORMAL	
152	B/O ANUJA	MALE	506082	TERM	NORMAL	B POSITIVE	O POSITIVE	<24HRS	19	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
153	B/O NANDINI	FEMALE	526517	TERM	LBW	A POSITIVE	A POSITIVE	24-48HRS	18.8	NORMAL	NORMAL	NORMAL	10	NORMAL	NORMAL	LOW	
154	B/O LAVANYA	MALE	526545	PT	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	

155	B/O PALLAVI BAI	FEMALE	526702	LPT	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	18	NORMAL	NORMAL	NORMAL	13	HIGH	NORMAL	NORMAL	
156	B/O SHRAVANI	FEMALE	527270	PT	LBW	B POSITIVE	A POSITIVE	<24HRS	20.6	NORMAL	NORMAL	NORMAL	11	NORMAL	NORMAL	NORMAL	
157	B/O NANDINI	MALE	527277	LPT	NORMAL	A POSITIVE	O POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
158	B/O MUNIRAT NAMMA	FEMALE	527400	LPT	LBW	O POSITIVE	B POSITIVE	24-48HRS	20.8	NORMAL	NORMAL	LOW	13	NORMAL	NORMAL	NORMAL	
159	B/O MOUNIKA	MALE	525420	PT	NORMAL	B POSITIVE	A POSITIVE	24-48HRS	20.4	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	NORMAL	
160	B/O SABA AFREEN	FEMALE	524146	PT	LBW	O POSITIVE	A POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	11	NORMAL	NORMAL	LOW	
161	B/O SUSHMA	FEMALE	528145	PT	LBW	O POSITIVE	O POSITIVE	24-48HRS	21.2	NORMAL	NORMAL	NORMAL	13	NORMAL	NORMAL	NORMAL	
162	B/O SUKANYA	MALE	528689	TERM	LBW	O POSITIVE	O POSITIVE	<24HRS	22	NORMAL	NORMAL	NORMAL	12	NORMAL	NORMAL	LOW	
163	B/O SHIRISHA	FEMALE	528698	LPT	LBW	O POSITIVE	B POSITIVE	24-48HRS	20.4	LOW	NORMAL	LOW	12	NORMAL	NORMAL	LOW	
164	B/O SANGEETHA	MALE	529004	PT	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	19.4	NORMAL	NORMAL	NORMAL	13	NORMAL	NORMAL	NORMAL	
165	B/O KEERTHI	FEMALE	529232	PT	NORMAL	A POSITIVE	O POSITIVE	24-48HRS	20.2	NORMAL	NORMAL	NORMAL	14	HIGH	NORMAL	NORMAL	
166	B/O MADHAVI	FEMALE	530048	PT	LBW	O POSITIVE	A POSITIVE	<24HRS	17	NORMAL	NORMAL	NORMAL	12	NORMAL	HIGH	NORMAL	
167	B/O HEMAVATHI	MALE	528394	TERM	LBW	A POSITIVE	B POSITIVE	24-48HRS	19.4	NORMAL	NORMAL	NORMAL	8	HIGH	NORMAL	NORMAL	
168	B/O SARASWATHI	FEMALE	528361	LPT	LBW	AB POSITIVE	B POSITIVE	24-48HRS	20	HIGH	NORMAL	NORMAL	9	NORMAL	NORMAL	LOW	
169	B/O M USHA	MALE	530588	LPT	NORMAL	O POSITIVE	O POSITIVE	<24HRS	18.4	NORMAL	NORMAL	NORMAL	8.4	NORMAL	NORMAL	NORMAL	
170	B/O NAYANA	MALE	530595	TERM	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	20.4	HIGH	NORMAL	NORMAL	12.5	NORMAL	NORMAL	NORMAL	
171	B/O PRIYADHARSHINI	MALE	530594	PT	LBW	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	12	LOW	NORMAL	NORMAL	
172	B/O SAKAMMA	MALE	528502	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	12	NORMAL	NORMAL	NORMAL	12.5	HIGH	NORMAL	NORMAL	
173	B/O LAKSHMI	FEMALE	531225	PT	NORMAL	A POSITIVE	O POSITIVE	>48HRS	21.4	NORMAL	NORMAL	NORMAL	14	NORMAL	HIGH	NORMAL	
174	B/O ASMA BEGUM	MALE	531399	PT	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	22	HIGH	NORMAL	NORMAL	12	HIGH	NORMAL	NORMAL	

175	B/O MALA	MALE	531932	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	0.6	NORMAL	NORMAL	NORMAL	
176	B/O LASKMI KUMARI	MALE	531985	LPT	NORMAL	B POSITIVE	O POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	12.3	HIGH	NORMAL	NORMAL	
177	B/O AMBIKA	FEMALE	532720	LPT	LBW	O POSITIVE	A POSITIVE	>48HRS	21.4	NORMAL	NORMAL	NORMAL	12.3	NORMAL	NORMAL	LOW	
178	B/O VEENA	MALE	532994	TERM	LBW	O POSITIVE	A POSITIVE	<24HRS	21.6	NORMAL	NORMAL	NORMAL	11.2	NORMAL	NORMAL	NORMAL	
179	B/O KAVYA	FEMALE	533275	TERM	LBW	O POSITIVE	B POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	11.3	NORMAL	NORMAL	LOW	
180	B/O YASMEEEN	FEMALE	534247	PT	NORMAL	AB POSITIVE	B POSITIVE	24-48HRS	20	NORMAL	NORMAL	NORMAL	10.6	NORMAL	NORMAL	NORMAL	
181	B/O SARALAMMA	MALE	534446	TERM	NORMAL	O POSITIVE	A POSITIVE	24-48HRS	21.2	HIGH	NORMAL	NORMAL	11.6	HIGH	NORMAL	NORMAL	
182	B/O JEEVITHA	FEMALE	534857	TERM	LBW	B POSITIVE	O POSITIVE	<24HRS	18.8	NORMAL	NORMAL	LOW	11.3	HIGH	NORMAL	NORMAL	
183	B/O SINDHU PRIYA	MALE	535418	PT	NORMAL	A POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	15.6	NORMAL	NORMAL	NORMAL	
184	B/O NAIMA	FEMALE	535453	LPT	LBW	A POSITIVE	A POSITIVE	24-48HRS	14	NORMAL	NORMAL	NORMAL	12.5	NORMAL	NORMAL	NORMAL	
185	B/O GAGANESHWARI	MALE	535459	TERM	LBW	A POSITIVE	A POSITIVE	24-48HRS	19.4	NORMAL	NORMAL	NORMAL	11.8	NORMAL	NORMAL	LOW	
186	B/O FIRDOSE	MALE	536602	LPT	LBW	A POSITIVE	A POSITIVE	<24HRS	17	HIGH	NORMAL	NORMAL	10.8	NORMAL	NORMAL	NORMAL	
187	B/O PRIYANKA	MALE	536617	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	8.6	HIGH	NORMAL	NORMAL	
188	B/O SHOBARANI	FEMALE	536046	PT	LBW	O POSITIVE	A POSITIVE	24-48HRS	22	NORMAL	NORMAL	NORMAL	6.8	HIGH	NORMAL	LOW	
189	B/O ANNAPOORNA	MALE	537398	TERM	LBW	O POSITIVE	B POSITIVE	24-48HRS	21	NORMAL	NORMAL	NORMAL	8.9	NORMAL	NORMAL	LOW	
190	B/O KALPANA	FEMALE	538177	TERM	NORMAL	B POSITIVE	B POSITIVE	<24HRS	20.6	NORMAL	NORMAL	NORMAL	9	NORMAL	NORMAL	NORMAL	
191	B/O ASHWINI	MALE	536605	TERM	NORMAL	O POSITIVE	O POSITIVE	24-48HRS	15	NORMAL	NORMAL	NORMAL	8	NORMAL	NORMAL	NORMAL	
192	B/O AMBIKA	FEMALE	538622	LPT	LBW	AB POSITIVE	A POSITIVE	24-48HRS	17	HIGH	NORMAL	NORMAL	6.5	NORMAL	NORMAL	NORMAL	
193	B/O SHILPA	MALE	542320	TERM	NORMAL	O POSITIVE	B POSITIVE	24-48HRS	16	NORMAL	NORMAL	NORMAL	9.9	HIGH	NORMAL	NORMAL	