"A PROSPECTIVE STUDY TO DETERMINE ASSOCIATION BETWEEN WEIGHT LOSS AND NEONATAL HYPERBILIRUBINEMIA IN NEONATES"

By

Dr. ATLURI JAHNAVI

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTER, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

PEDIATRICS

Under the Guidance of

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JULY 2024

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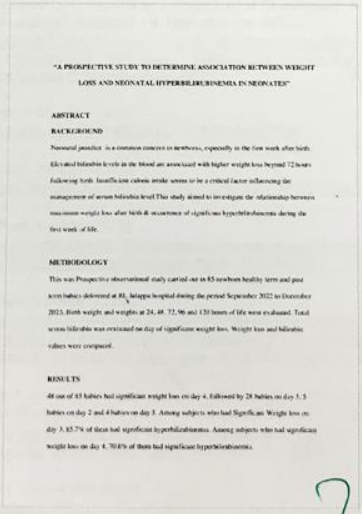

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ABSTRACT

BACKGROUND

Neonatal jaundice is a common concern in newborns, especially in the first week after birth. Elevated bilirubin levels in the blood are associated with higher weight loss beyond 72 hours following birth. Insufficient calorie intake seems to be a critical factor influencing the management of serum bilirubin level.This study aimed to investigate the relationship between maximum weight loss after birth & occurrence of significant hyperbilirubinemia during the first week of life.

METHODOLOGY

This was Prospective observational study carried out in 85 newborn healthy term and post term babies delivered at RL Jalappa hospital during the period September 2022 to December 2023. Birth weight and weights at 24, 48, 72, 96 and 120 hours of life were evaluated. Total serum bilirubin was evaluated on day of significant weight loss. Weight loss and bilirubin values were compared.

RESULTS

48 out of 85 babies had significant weight loss on day 4, followed by 28 babies on day 3, 5 babies on day 2 and 4 babies on day 5. Among subjects who had Significant Weight loss on day 3, 85.7% of them had significant hyperbilirubinemia. Among subjects who had significant weight loss on day 4, 70.8% of them had significant hyperbilirubinemia.

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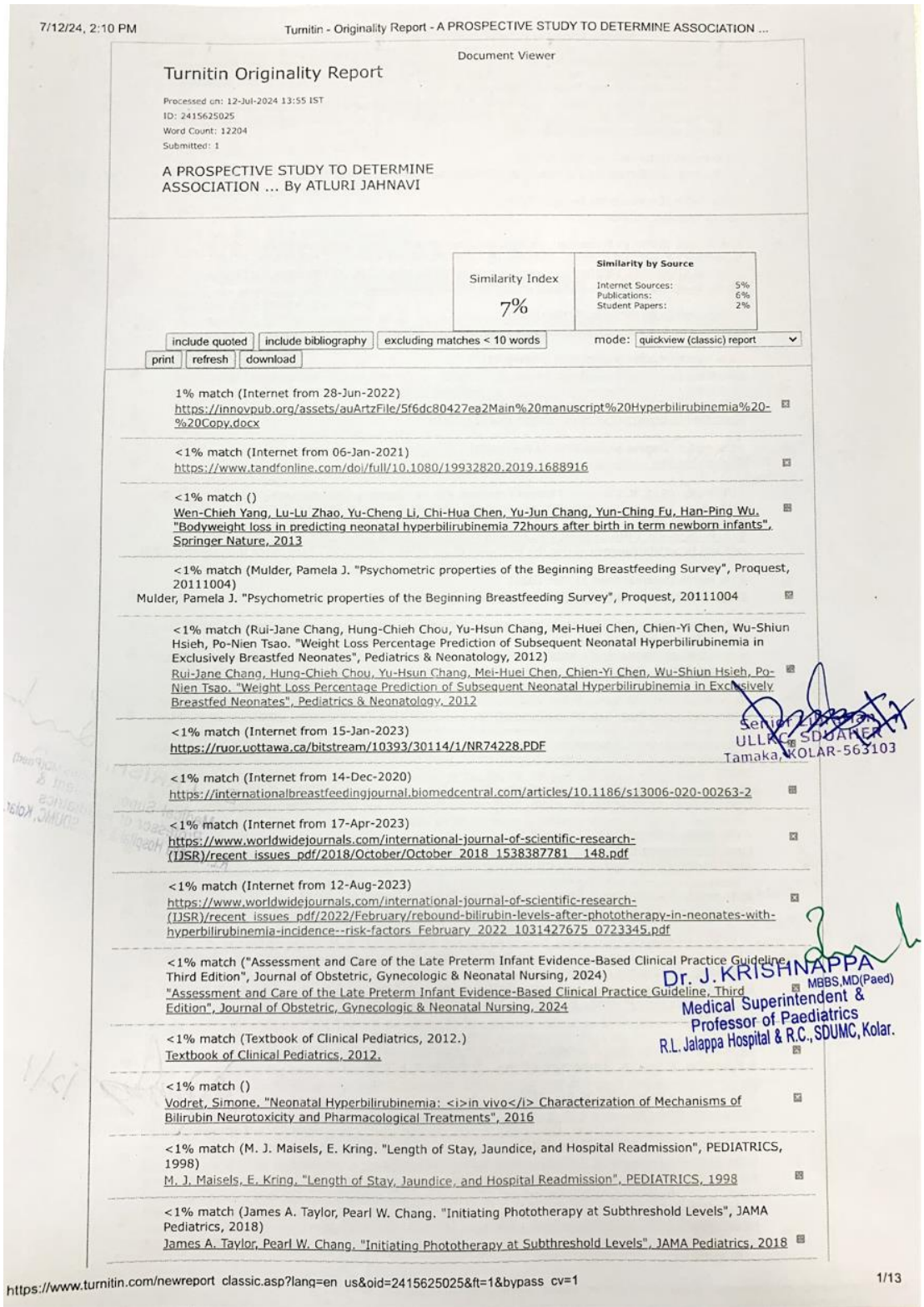

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"A PROSPECTIVE STUDY TO DETERMINE ASSOCIATION BETWEEN WEIGHT LOSS AND NEONATAL HYPERBILIRUBINEMIA IN NEONATES" ABSTRACT BACKGROUND Neonatal jaundice is a common concern in newborns, especially in the first week after birth. Elevated bilirubin levels in the blood are associated with higher weight loss beyond 72 hours following birth. Insufficient calorie intake seems to be a critical factor influencing the management of serum bilirubin level. This study aimed to investigate the relationship between maximum weight loss after birth & occurrence of significant hyperbilirubinemia during the first week of life. METHODOLOGY This was Prospective observational study carried out in 85 newborn healthy term and post term babies delivered at RL Jalappa hospital during the period September 2022 to December 2023. Birth weight and weights at 24, 48, 72, 96 and 120 hours of life were evaluated. Total serum bilirubin was evaluated on day of significant weight loss. Weight loss and bilirubin values were compared. RESULTS 48 out of 85 babies had significant weight loss on day 4, followed by 28 babies on day 3, 5 babies on day 2 and 4 babies on day 5. Among subjects who had Significant Weight loss on day 3, 85.7% of them had significant hyperbilirubinemia. Among subjects who had significant weight loss on day 4, 70.8% of them had significant hyperbilirubinemia. CONCLUSION Significant weight loss after birth may be a predictor for neonatal hyperbilirubinemia & serve as a helpful clinical indicator pointing towards necessary steps to be taken inorder to prevent worsening of this treatable condition which may otherwise lead to devastating complications INTRODUCTION In the present global setting, brief hospital stays following childbirth, it is critical to evaluate the variables linked to possibly avoidable reasons for readmissions of newborns. The most common reasons for readmission within the first weeks of life are feeding difficulties, sometimes accompanied by dehydration & hyperbilirubinemia especially in term babies, which are caused by inadequate oral intake. These conditions are highly correlated with one another.[1-4] For newborns, BWL of less than 10% are physiologically acceptable. According to a US Centers for Disease Control research, breastfed babies that develop faster in the first two months of life and then slow down until they are one year old are shorter and heavier than the "World Health Organization".[5] There is no reason to be concerned about an infant's development, even temporary BWL, if they are receiving proper breastfeeding. BWLs greater than 7% from birth weight, however, suggest potential issues with breastfeeding and call for immediate assessment.[6] Inadequate breast milk intake is the most prevalent cause of hypernatremia and severe weight loss. The second stage of lactogenesis, when enough milk is produced, starts in the first four days following birth. On the first day of life, a nursing baby may receive less than 100 milliliters of milk per day, but by the fourth day, milk production typically increases substantially to an average of 500 milliliters per day.7As a result, by the end of the first week, weight loss recovery is anticipated. The highest weight loss and recovery times were found to be, respectively, 2.7 and 8.3 days, according to MacDonald et al.[8] Excessive weight loss in neonates is associated with complications such as jaundice and dehydration, which can lead to renal failure, thrombosis, hypovolemic shock, and convulsions. Identifying the risk factors associated with excessive weight loss can help with the formulation of preventative measures. Jaundice impacts approximately 60% of full-term infants and 80% of premature infants among all morbidities occurring in the first week of life. It is common reason for readmission following hospital discharge following delivery. Increased generation from red cell breakdown, slower clearance by

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ACKNOWLEDGEMENT

*I would like to thank the **ALMIGHTY** for giving me the opportunity, strength and courage throughout the post-graduation and also to complete my dissertation.*

*I express my heartfelt and humble gratitude to my beloved guide **Dr.Krishnappa.J**, Professor, Department of Paediatrics who is constant source of encouragement and support throughout the post-graduation. His passion for the subject and patient care always inspired me.*

*I would like to express my deep sense of gratitude and humble thanks to **Dr.Sudha Reddy.V.R**, Professor and Head of the department for her constant guidance, support and encouragement.*

*I am grateful to **Dr. K.N. V. Prasad**, Professor, whose dedication to medicine and commitment to keeping us updated has been a constant source of inspiration. Thanks for sharing his knowledge with us daily and for his unwavering encouragement and support throughout my postgraduate studies.*

*My heartfelt thanks to all Associate professors **Dr.Narendra, Dr.Srikanth, Dr. James Daniel** for their practical tips, invaluable advice and constant encouragement.*

*My heartfelt thanks to all Assistant professors **Dr.Naveen Kumar, Dr.Chethan, Dr.Karthik, Dr.Abhishek, Dr.Bhanuchand, Dr.Niranjan, Dr.Vidya** for their practical tips, invaluable advice and constant encouragement.*

I would like to thank my Senior residents ***Dr.Jaishree, Dr.Pravallika, Dr.Parimala, Dr.Shrishail, Dr.Manasa*** *for their guidance and support.*

I extend my sincere thanks to my beloved senior ***Dr.Mathumitha.T,*** *for guiding me in patient care and supporting me throughout. I extend my sincere thanks to all seniors* ***Dr.Jefrin, Dr.Nikitha, Dr.Trisali, Dr.Bindu T, Dr. Praveen, Dr.Prakash*** *for sharing their immense knowledge. I am thankful for their valuable guidance and helping me with my dissertation.*

I would like to express my gratitude to my close friends ***Dr.Mouna, Dr.Saiteja, Dr.Kamalakar, Dr.Ramswaroop, Dr.Rana, Dr.Karthik*** *for their support and love.*

Heartfelt thanks to my juniors ***Dr.Bhargavi, Dr.Teja Manne, Dr.Varsha, Dr.Deepthi, Dr.Rohit****,* ***Dr.Deepak, Dr.Vishaka, Dr.Supreeth, Dr.Indra, Dr.Venkatesh, Dr.Pravallika, Dr.Ashwini, Dr.Akhila, Dr.Debashish.***

I would express my deepest gratitude to my beloved parents ***Atluri Ramesh, Atluri Jyothi*** *for constantly believing in me and whose love, blessings and sacrifices made me the person who I am today. They have been my pillars throughout my post graduation and without whom this journey would have been impossible. Especially my dad who is my best friend, my guide who teaches me morals and ethics constantly.*

Special thanks to my better half, ***Gowtham Yalavarthi,*** *Father in law* ***Mallikharjuna Rao,*** *Mother in law* ***Sri Lakshmi*** *for their love and constant support.*

I would like to thank my brother ***Atluri Hemanth,*** *for helping me in all editing work*

without whom this dissertation wouldn't have been possible.

*I would like to thank my paternal uncle **Atluri Venkataratnam**, for encouraging me to opt this profession, paternal aunt **Atluri Rani** for their constant support.*

*I would like to thank my cousins **Atluri Nithin,** who always encourages me and elevates my mood throughout this tough phase of post-graduation and **Atluri Lolini**, for constant support.*

*I would like to thank my cousins **Komma Sai Ram, Komma Renuka** for their support and my neice and nephew **Aarna** and **Avyan** for their love.*

*I would like to thank my best friend **Hema Latha,** who guides me and supports me in each and every task in my life.*

*I would like to thank **Mrs Gayathri, Mr Jagannath** who had helped me in the clerical work.*

*Lastly, I would like to express my gratitude to all my **interns** and **nurses Prema, Navya, Greyna, Mamatha, Priya, Asha, Leena** and the **children** who were part of this study without whose support this study wouldn't have been possible.*

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ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
TSB	Total Serum Bilirubin
BWL	Body Weight Loss
EHC	Enterohepatic Circulation
BIND	Bilirubin Induced Neurological Dysfunction
NWL	Neonatal Weight Loss
UCB	Unconjugated Bilirubin
CB	Conjugated Bilirubin
UGT1A1	UDP-glucuronosyl transferase 1A1
CNSI	Crigler-Najjar syndrome type I
CNSII	Crigler-Najjar syndrome type II
GS	Gilbert's syndrome
G6PD	Glucose 6-phosphate dehydrogenase
CNS	Central Nervous System

ROS	Reactive oxygen species
PT	Phototherapy
ET	Exchange transfusion
UDCA	Ursodeoxycholic acid
MNC	Minocycline
AAP	American Academy of Pediatrics
SBL	Serum bilirubin level
PPV	Positive Predictive Value
NPV	Negative Predictive Value
GA	Gestational age
LSCS	Lower segment cesarean section
NVD	Normal Vaginal Delivery
TCB	Transcutaneous Bilirubin
SDS	Standard Deviation Score
CI	Confidence Interval

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ABSTRACT

BACKGROUND

Neonatal hyperbilirubinemia is a frequently encountered issue during the early neonatal period, particularly within the first week of life. Significant levels of bilirubin in the blood are linked to increased weight loss after 72 hours post-birth. Inadequate caloric intake appears to play a significant role in the regulation of serum bilirubin levels.This study aimed to investigate the relationship between the maximum weight loss after birth and the occurrence of significant hyperbilirubinemia during the first week of life.

METHODOLOGY

This was a Prospective Observational study carried out in 85 newborn healthy term and post term babies delivered at RL Jalappa hospital during the period September 2022 to December 2023. Birth weight and weights at 24, 48, 72, 96 and 120 hours of life were assessed. Total serum bilirubin was assessed on day of significant weight loss. Weight loss and bilirubin values were compared.

RESULTS

48 out of 85 babies had significant weight loss on day 4, followed by 28 babies on day 3, 5 babies on day 2 and 4 babies on day 5. Among subjects who had Significant Weight loss on day 3, 85.7% of them had significant hyperbilirubinemia. Among subjects who had significant weight loss on day 4, 70.8% of them had significant hyperbilirubinemia.

CONCLUSION

Significant weight loss after birth may be a predictor for neonatal hyperbilirubinemia, and serve as a helpful clinical indicator pointing towards necessary steps to be taken inorder

to prevent worsening of this treatable condition which may otherwise lead to devastating complications.

Keywords: Hyperbilirubinemia, Significant weight loss, neonates

INTRODUCTION

INTRODUCTION

In the present global setting of brief hospital stays following childbirth, it is critical to evaluate the variables linked to possibly avoidable reasons for readmissions of newborns. The most frequent causes of readmission in the first two weeks of life are feeding issues with or without dehydration and hyperbilirubinemia, especially in term babies, which are caused by inadequate oral intake. These conditions are highly correlated with one another.[1,2,3,4] For newborns, BWL of less than 10% are physiologically acceptable. According to a US Centers for Disease Control research, breastfed babies that develop faster in the first two months of life and then slow down until they are one year old are shorter and heavier than the "World Health Organization".[5] There is no reason to be concerned about an infant's development, even temporary BWL, if they are receiving proper breastfeeding. BWLs greater than 7% from birth weight, however, suggest potential issues with breastfeeding and call for immediate assessment.[6]

Inadequate breast milk intake is the most prevalent cause of hypernatremia and severe weight loss. The second stage of lactogenesis, when enough milk is produced, starts in the first four days following birth. A nursing baby may get less than 100 milliliters of milk per day on the first day of life, but by the fourth day, milk supply has increased significantly to an average of 500 milliliters per day.[7] As a result, by the end of the first week, weight loss recovery is anticipated. The highest weight loss and recovery times were found to be, respectively, 2.7 and 8.3 days, according to MacDonald et al.[8]

Excessive weight loss in neonates is associated with complications such as jaundice and dehydration, which can lead to renal failure, thrombosis, hypovolemic

shock, and convulsions. Identifying the risk factors associated with excessive weight loss can help with the formulation of preventative measures. Of all the morbidities that occur in the first week of life, jaundice affects 60% of term and 80% of preterm babies. It is also the most common reason for readmission following hospital release following delivery. Increased generation from red cell breakdown, slower clearance by immature hepatic processes, and reabsorption by enterohepatic circulation (EHC) are the three mechanisms contributing to the elevated total serum bilirubin (TSB) content in neonates. In some infants, bilirubin-induced neurological dysfunction (BIND) can be brought on by elevated blood bilirubin levels. The majority of other times, jaundice is benign and doesn't need to be treated. Five to ten percent of them have clinically severe jaundice, which has to be treated to bring down TSB levels and avoid BIND.

In healthy term newborns, hyperbilirubinemia is caused by an increase in unconjugated serum bilirubin during the first week of life.Excessive weight loss is a contributing factor to hyperbilirubinemia. A newborn may experience difficulties nursing if it loses more than 7% of its birth weight. Hyperbilirubinemia and feeding problems with or without dehydration-the most frequent causes of readmission in the first two weeks of life-have a strong link with one another because of inadequate oral intake, particularly in term neonates. Breastfeeding entirely from birth is advised for all healthy term neonates, according to numerous organizations. The enterohepatic circulation becomes more prone to bilirubin levels when healthy term infants who are exclusively breastfed and whose breastfeeding has not yet reached a satisfactory level at the time of discharge are more vulnerable to inadequate calorie intake, dehydration from decreased volume and frequency of breast milk, and secondary delayed gastrointestinal motility.[9,10]

There are two types of jaundice linked to breastfeeding: late breast milk jaundice and early breast feeding jaundice. Breastfeeding jaundice appears within the first week of

life, whereas breast milk jaundice peaks between days 10 and 21 of life and can last up to three months. Jaundice from breastfeeding was always assumed to be benign, but within the past 15 years, cases of kernicterus in infants who were solely or even partially breastfed have been reported.[11,12] Early discharge procedures combined with inadequate breastfeeding, which causes dehydration and/or malnutrition, may make this risk worse.[13] A key factor in the effectiveness of breastfeeding is lactation counseling.[12,13] The American Academy of Pediatrics advises that all breastfed babies should see a doctor within 3-5 days of age for a follow-up visit to determine body weight and whether jaundice is present, even in the event of an early release.

Breastfeeding exclusively can cause severe weight loss in infants, which can be made worse by hypernatremia and other illnesses. Starting to breastfeed as soon as possible after delivery and taking prompt action to address any concerns that may come up, like nipple troubles, delayed "coming in" of breast milk, poor breast attachment, and breast engorgement, will help promote successful breastfeeding. After nursing pairs are discharged from the hospital, it is critical to closely monitor them, with particular attention to the child's weight, in order to detect inadequate breastfeeding. The world health organization states that the first week of life-when the majority of baby fatalities occur-is when the largest gaps in infant care are typically seen. Moreover, many habits, including nursing and overcoming obstacles, start in the early stages of life and are linked to early life loss.[14] As a result, health care providers—particularly midwives and nurses—may be able to contribute significantly to prenatal care by recognizing the traits associated with postpartum depression and realizing that prevention is preferable to therapy. It was also proposed in a review study that doing observational research on NWL is more important than conducting clinical trials.[15]

Extreme hyperbilirubinemia patients have historically lost a large amount of weight, according to several research; however, few have examined this relationship separately in term infants who are otherwise healthy. This study sought to ascertain if weight loss in term and postterm newborns may serve as a predictor for neonatal hyperbilirubinemia.

OBJECTIVES

OBJECTIVES OF THE STUDY

To determine association between maximum weight loss percentage after birth and development of significant hyperbilirubinemia within 1st week of life.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Physiological weight loss refers to the weight reduction that occurs in almost all babies during their first few days of life. The primary cause is fluid decrease. A higher birth weight is associated with late cord clamping, resulting in a larger weight change. The consumption of adipose tissue by the babies as a source of energy also results in weight reduction.[15] Abrupt weight loss is linked to problems such hypoglycemia, dehydration, jaundice, and thrombosis, which can result in hypovolemic shock, renal failure, and convulsions.[16,17,18,19]

Previous research have demonstrated that primiparity and an older mother's age are risk factors for significant infant weight loss.[20,21] as a result of lactation starting later. It is anticipated that in the first postnatal period, idiopathic neonatal jaundice would manifest in 60–80% of healthy, full-term newborns.[22] Idiopathic newborn jaundice is associated with increased heme breakdown, immature liver function, low levels of intestinal flora, increased enterohepatic circulation of bilirubin, and inadequate intake.[23] Four to six completely wet diapers in a 24-hour period and the passing of three to four stools daily on the fourth day are signs of insufficient intake. Furthermore, by the third or fourth day, a baby who is receiving enough breast milk should transition from meconium to a mushy, mustard-colored feces. These evaluations assist in identifying breastfed babies who may be dehydrated owing to insufficient intake; nevertheless, because of individual variations, they are rather subjective. Body weight loss (BWL) %, when compared to other approaches, is an objective and practical instrument that may help determine whether treatments like supplemental nutrition should be taken into consideration.

Changes in the metabolism of bilirubin cause hyperbilirubinemia, or neonatal jaundice. High amounts of unconjugated bilirubin that persist unchecked can cause

bilirubin-induced neurological damage and, in the worst case scenario, kernicterus-related mortality. The fraction of unconjugated bilirubin (UCB) that is not bound to albumin (free bilirubin, Bf) increases when severe hyperbilirubinemia satisfies plasma albumin's bilirubin binding capacity. This lipophilic material penetrates the blood-brain barrier, accumulates within the brain, and damages neurons. The developing central nervous system affects many cellular functions, and coordinated interruption of these processes results in cellular harm. Long-term toxic bilirubin levels can cause a variety of neurological impairments in patients, including problems in motor, sensory, and cognitive processes. Even after extensive research, there are still many unanswered questions regarding the mechanics and causes of the disease's genesis.

According to earlier research, abnormal neonatal BWL is defined as 7% to 10% BWL by day 3 in infants who are exclusively breastfed.[24] On the other hand, perspectives about normal newborn BWL and the appropriate timing of supplementary feeding in order to prevent substantial hyperbilirubinemia are not in agreement. Therefore, the purpose of the prospective study was to ascertain if weight loss and newborn hyperbilirubinemia are related.

Breast feeding and weight loss

It is well known that breastfeeding benefits moms and families as well as newborns. Breastfeeding has been linked to an increased risk of severe and/or early infant hyperbilirubinemia, however.[25,26] It's unclear what mechanism is at play here. There have been theories put up regarding the mechanisms of insufficient calorie and/or fluid intake, decreased hepatic excretion of bilirubin, and enhanced intestinal absorption of bilirubin (enterohepatic circulation). A large-scale population investigation has

demonstrated that the pathophysiology of newborn hyperbilirubinemia is influenced by greater weight loss % rather than nursing in and of itself.[27]

In the nursery, inadequate breastfeeding is not unusual, especially for new moms. Hypernatremia, weight loss, and decreased urine production are examples of neonatal symptoms. Significant newborn hyperbilirubinemia is associated with higher weight loss and/or breastfeeding, according to a growing body of research. It is often advised to provide supplemental feeding for infants who are not receiving enough nursing care. But it is unclear what constitutes inadequate at this point. Previous studies have classified serious weight loss due to inadequate breastfeeding as occurring in 7% or more of BBW. A study by Rui-Jane Chang et al.[28] found that weight loss >8% of BBW after 48 hours (OR = 1.45; 95% CI = 1.06, 1.97) and >11% of BBW after 72 hours (OR = 2.01; 95% CI = 1.16, 3.46) are the best cut-off values for predicting the development of subsequent hyperbilirubinemia; the corresponding NPVs were 77.7% and 76.8%.

Bilirubin Metabolism

Bilirubin is the final result of heme catabolism in the intravascular compartment. Roughly 80% of bilirubin is produced by the reticulo-endothelial system breaking down erythrocyte haemoglobin; the remaining 20% is produced by myoglobin and other heme-containing proteins, like cytochromes, breaking down as well as inefficient erythropoiesis in the bone marrow. Heme is broken down by heme oxygenase into biliverdin, which is further broken down into UCB by biliverdin reductase. Water-insoluble, UCB binds to albumin in plasma and moves toward the liver. The enzyme UDP-glucuronosyl transferase 1A1 (UGT1A1) converts bilirubin to glucuronic acid in the hepatocyte endoplasmic reticulum.

Glucuronosylation increases bilirubin's solubility and is required for its elimination in the bile fluid, both of which help to limit the detrimental accumulation of UCB in tissues. Following conjugation, bilirubin is transferred from hepatocytes into the biliary fluid by active transporters like multidrug-associated resistance protein 2 (Mrp2). The bacterial flora in the small intestine uses β-glucuronidase to deconjugate bilirubin and then breaks it down into urobilinoids, which are mostly composed of urobilin and stercobilin, as well as their respective oxidation products, urobilinogen and stercobilin. Additionally, enterocytes take up a portion of the produced UCB and return it to the liver. Following hydrolyzation, a constant portion of bilirubin glucuronide is also released back into the plasma, where it can be reabsorbed by hepatocytes.

Bilirubin Toxicity and Disease

The buildup of UCB is caused by a change in the metabolism of bilirubin. Genes and non-genetic factors, or their combination, can cause hyperbilirubinemia. (Table 1 and 2).

Hereditary causes of Unconjugated Hyperbilirubinemia

Genetic diseases may be the source of elevated levels of UCB (Table 1). One in a million babies is born with the exceedingly rare recessive genetic condition known as Crigler-Najjar syndrome. Mutations in the UGT1A1 gene cause a lack of hepatic UGT1A1 activity, which is its defining feature. Inaction on the part of the patient may result in hyperbilirubinemia, severe brain damage, or even death. Despite the fact that there is a continuum of phenotypes, Crigler-Najjar syndrome is divided into two variants based on the severity of the symptoms: type I CNSI and type II CNSII.

When bilirubin glucuronides and UGT1A1 activity are completely absent from the bile, it results in hyperbilirubinemia in CNSI. If left untreated, this can lead to bilirubin neurotoxicity and death.

The existence of residual UGT1A1 enzyme activity separates CNSII, a milder version of the illness, from CNSI. This is caused by missense mutations in the gene, which lower the enzyme's production or substrate affinity. Patients with CNSII have low bilirubin glucuronoside levels in their bile because phenobarbital medication lowers bilirubin levels and activates the UGT1A1 gene.

The less severe hereditary variant of unconjugated hyperbilirubinemia is known as Gilbert's syndrome (GS). A mutation in the UGT1A1 gene's promoter may result in reduced enzyme expression in GS. Despite being milder, a variety of stressors can cause bilirubin levels in the GS condition to increase, increasing the patient's risk of high UCB levels.

Blood-related conditions like glucose 6-phosphate dehydrogenase (G6PD) deficiency and/or ABO and/or Rh incompatibility are examples of other hereditary causes. These disorders impact the metabolism of red blood cells, leading to heightened hemolysis and, as a result, worsening hyperbilirubinemia.

Table 1: Genetic cause of Hyperbilirubinemia Syndromes and related causes are listed

Syndrome	Causes
Crigler-Najjar Syndrome type I (CNSI)	Complete lack of UGT1A1 activity
Crigler-Najjar Syndrome type II (CNSII)	Important reduction of UGT1A1 activity
Gilbert's Syndrome (GS)	*UGT1 promoter polymorphism*
ABO incompatibility	Maternal IgG antibodies with specificity for the ABO blood group system pass through the placenta to the fatal circulation where they can cause haemolysis
Glucose 6-phosphate dehydrogenase (G6PD) deficiency	Low levels of G6PD, an enzyme involved in the metabolism of red blood cells, lead to haemolytic anaemia
Rh incompatibility	During birth, the mother may be exposed to the infant's blood, and this causes the development of antibodies, which may affect the health of subsequent Rh+ pregnancies

Non-genetic causes of unconjugated hyperbilirubinemia

In addition to genetic variables, a number of non-genetic factors can impact bilirubin metabolism (Table 2). Liver dysfunction can be caused by pathological situations such hypoxia, infection, sepsis, or hepatic disorders. These factors can affect the glucuronosylation system and raise the levels of bilirubin in the bloodstream. Another potential cause of hyperbilirubinemia is breastfeeding. Indeed, during the very early postnatal phases, when the expression of the liver enzyme is still extremely low, breast milk increases the amount of UCB by suppressing the intestine UGT1A1 expression. β-glucoronidase, which is present in breast milk, enhances the breakdown and absorption of bilirubin in the intestinal lumen. Furthermore, consuming insufficient amounts of breast milk exacerbates the disease by causing dehydration. Neonatal jaundice, one of the most common disorders in babies, is brought on by a delayed induction of UGT1A1 gene expression, which limits the ability of infants to conjugate. The co-occurrence of delayed UGT1A1 activity and other variables, like rapid fetal erythrocyte breakdown and/or inefficient serum albumin transport to the liver, can lead to acute hyperbilirubinemia.

Table 2 : Non-genetic cause of unconjugated hyperbilirubinemia

• Breast milk hepatitis Intestinal UGT1A1 expression is decreased by breast milk.
• Hypoxia.
• Infections.
• Hepatic disorders Liver dysfunction.
• Neonatal jaundice Delay in the UGT1A1 enzyme.

Bilirubin-induced neurological dysfunction (BIND)

Although jaundice is benign for the vast majority of newborns, preventative measures must be taken to minimize any potential neurodamage.

UCB concentrations can rise to potentially lethal amounts in newborn jaundice, beyond the binding capacity of albumin. Rekindled interest in bilirubin encephalopathy is a result of its increased prevalence in recent years.[29,30] Babies with excessively high UCB levels may later have BIND (bilirubin-induced neurological dysfunction) in the central nervous system if treatment is not given.[31]

BIND may be classified as mild, moderate, or severe according on the duration of bilirubin exposure and the associated symptoms. While the symptoms of mild to moderate BIND may be treated, severe BIND can cause permanent brain damage. Lethargy, ophthalmoplegia (ocular muscle paralysis), high-pitched screaming, opisthotonus (bowed body and rigid extremities or dystonia), convulsions, mental impairment, and frequently fatal kernicterus are the clinical indications of persistent bilirubin poisoning in babies. Bilirubin really targets particular areas of the developing brain, including the cerebellum, which includes granule and Purkinje neurons, as well as the basal ganglia, cochlear, and oculomotor nuclei. Autism, attention deficit problems, developmental delays, and isolated neural hearing loss have all been linked to even modest levels of UCB. In any event, persistently excessive bilirubin exposure may result in neurological aftereffects and may permanently affect the learning and memory of the newborn.

Brainstem auditory brainstem potentials, sometimes referred to as auditory brainstem response ABRs, and magnetic resonance imaging of the brain are used to determine the extent of BIND in babies because the auditory system is particularly susceptible to bilirubin toxicity. As a result, the response to auditory stimuli functions as a

reliable marker of brain activity. It is clear that treating and managing BIND would benefit from a full understanding of the neurological processes and molecular mechanisms producing bilirubin neurotoxicity.

"Kernicterus" is the term used to describe the permanent harm caused by extended exposure to elevated bilirubin levels. In low- and middle-income countries, on the other hand, the incidence can climb to 73 per 100,000 live births. In Western countries, the incidence of kernicterus in neonates with severe hyperbilirubinemia is 10 per 100,000 live births.[29] Preterm births occurring before the thirty-first week of pregnancy can cause a significant increase in instances, up to 1.8 per 1000 live births.[32] Given that the location of the selective damage relies on the neurodevelopmental age at the time of UCB exposure, a temporal window of CNS sensitivity to UCB toxicity has been proposed. The motor kernicterus subtype usually appears in children who are more than 34 weeks gestation, whereas the auditory kernicterus subtype is more common in neonates with peak levels of TB exposure at earlier gestational ages.[33]

Mechanisms of bilirubin neurotoxicity

1. Neurodegeneration

The general term describing the gradual loss of neuronal structures or functions, including death, is neurodegeneration. Neurodegeneration has a profound effect on the central nervous system because, once created, neuron renewal is strictly regulated. The etiology of neurodegeneration is frequently not fully understood, and the identification of the critical processes triggering the illnesses is still unknown despite the great efforts undertaken to clarify the consequences. Neurodegeneration can also be caused by elevated bilirubin levels. As previously stated, elevated levels of systemic UCB impact the developing central nervous system (CNS) because to genetic and non-genetic

modifications in bilirubin metabolism. Neonatal jaundice is a temporary condition that can cause neurotoxic amounts of bilirubin, which can damage neurons. Because bilirubin is able to adhere to cellular membranes, particularly those that are rich in myelin, it can be harmful to neurons.[34]

2. Oxidative stress

Reactive oxygen species (ROS, more generally, free radicals) are produced when antioxidant defenses are unbalanced, leading to oxidative stress. Chemical entities with unpaired electrons are called free radicals. These may originate from external electromagnetic radiation, chemical processes' byproducts, or the results of aerobic respiration. The free radicals become unstable due to the unpaired electrons, and once they reach a stable state, they transfer the unpaired electron to other molecules. A free radical chain reaction is produced when non-radical molecules interact with free radicals, passing along the instability of unpaired electrons to other molecules. Intense molecular instability has the potential to disrupt cellular homeostasis by impairing the body's defenses against this type of stress. The mitochondrial cytochrome oxidase in cells transfers electrons without releasing reactive oxygen species, preventing the overproduction of electrically unstable compounds. However, because oxidation may harm components like DNA, lipids, and proteins, oxidative stress is bad for cells. Because UCB contains cytoprotective and antioxidant qualities, slightly increased bilirubin concentrations are thought to be advantageous. Nevertheless, oxidative stress is brought on by elevated bilirubin levels. Studies conducted in vitro have demonstrated that oxidative stress is a key cause of bilirubin neurotoxicity. Excess ROS disrupts glutathione homeostasis and mitochondrial metabolism, which leads to cellular energy crisis, cytochrome c release, and Ca2+ disruption. Nervous cell death results from bilirubin-

induced excessive oxidative stress. As a result of neurons' loss of sensibility about the unbalanced metabolism of reactive oxygen production during neurodegeneration, oxidative stress is compromised, which compromises cell survival.

3. Neuroinflammation

The term "neuroinflammation" refers to the overall inflammatory condition affecting the brain and spinal cord. In addition to cytokines, chemokines, ROS, and secondary messengers, other factors that affect the degree of neuroinflammation include the injury type, the context, and the duration of the inflammatory stimulation.

Endothelial cells and CNS glia cells, including macrophages, oligodendrocytes, astrocytes, and microglia, are the primary providers of CNS immune surveillance. The goal of the CNS inflammatory response is to restore the compromised state that the stressor agents have caused. On the other hand, chronic stimulation of the neuroinflammatory response may exacerbate and magnify the injury by increasing the activation of glia cells.

Depending on their level of activation, migrating macrophages or microglia cells at the site of injury can either intensify the damage or heal it. Due to UCB neurotoxicity, there are more glia cells in the body, which activates important cellular and molecular components of neuroinflammation. This can lead to an aggravation of neurodamage and even cell death.

4. Autophagy

Organelles and proteins may be broken down in an organized manner by a cellular process called autophagy, which translates from the Greek to mean "self-eating." In actuality, autophagy is triggered during starving situations to supply extra energy and amino acids. In response to stressors, the basal autophagic activity is thought to function

as a cytoprotective mechanism, promoting survival. On the other hand, apoptosis is triggered when this process is not properly regulated, which leads to cell death. The cytoplasmic number of lysosomes is altered, which is harmful to the homeostasis of the cell, as a result of the ineffective clearance of the produced vesicles, the autophagosomes. Hydrolases break down the contents of the vesicles, while autophagosomes fuse with lysosomes to form autolysosomes. Further research is required to clarify the role of autophagy in the developing brain exposed to bilirubin, as this process has not yet been studied in vivo.

Standard therapeutic treatments for hyperbilirubinemia

A number of therapies have been suggested to reduce the chance of BIND. The goal of standard therapy is to lower hazardous UCB levels.

1 Phenobarbital treatment

The effectiveness of phenobarbital treatment in lowering plasma bilirubin levels has been the basis for the initial clinical difference between types I and II of Crigler-Najjar syndrome. The UGT1 gene is expressed more when phenobarbital is used. Phenobarbital transcriptionally induces the UGT1A1 gene in CNSII patients, increasing basal residual UGT1A1 activity and keeping bilirubin concentrations below the neurotoxicity threshold. On the other hand, as there are no indications of an increase in UGT1A1 activity, this medication has no effect on hyperbilirubinemia in CNSI patients. Since phototherapy's efficacy diminishes with age, these individuals receive temporary treatment with it. Thus far, liver transplantation has been the only curative therapy for CNSI.

2. Phototherapy

Intense phototherapy (PT) is the usual course of treatment for severe unconjugated hyperbilirubinemia. As UCB circulates in skin capillaries, light energy (emission range: 400–525 nm, peak emission: 450–460 nm) is absorbed by the protein. Without the necessity for liver conjugation, insoluble bilirubin can be eliminated into the bile or, at a slower rate, the urine thanks to this conversion into water-soluble photoisomers. PT is often quite successful in preventing transitory hyperbilirubinemia in healthy newborns due to the rapid development of the hepatic conjugation machinery. The prognosis of preterm at-risk newborns is affected by the time of physical therapy intervention. In fact, kernicterus or persistent post-icteric sequelae might be avoided by putting mechanisms in place to quickly and efficiently lower the elevated bilirubin burden before neurologic symptoms appeared.[35]

Preventive phototherapy may impact the rate of exchange transfusion and helps to maintain a lower blood bilirubin content. Even if physical therapy is effective in treating newborn jaundice, additional, potentially dangerous exchange transfusions may still be necessary.

The chronic unconjugated hyperbilirubinemia in Crigler-Najjar patients necessitates a daily phototherapy treatment duration of 12–14 hours. Nevertheless, as people age, a variety of variables, including thickening skin, increased pigmentation, and an increase in the body's surface to volume ratio, can impact how well PT treatments work. Consequently, the efficacy of PT is reduced as less blue light enters the capillaries. Patients with CNSI react to physical therapy (PT) only momentarily, and unless they have a liver transplant, they will always be at danger of brain injury.

3 Immunoglobulins

Hemolysis, a primary contributing factor to the development of jaundice, is brought on by blood incompatibility. When treating patients with hyperbilirubinemia brought on by Rh and/or ABO blood incompatibility, PT in conjunction with intravenous injections of immunoglobulins against immune-mediated hemolysis (such as antibodies against Rh or AB antigens) has been employed. It has been demonstrated that intravenous immunoglobulin injections greatly shortened the length of PT and eliminated the requirement for exchange transfusions in cases of baby jaundice.[36]

4 Exchange transfusion

Physical therapy (PT) is typically used to treat jaundice because of its high safety, ease of use, and adequate effectiveness. However, jaundiced children who do not react to PT or who are significantly hyperbilirubinemic at first presentation are treated with a more intrusive option, such as exchange transfusion (ET), in an effort to avoid or lessen bilirubin-induced brain damage. In this treatment, suitable fresh blood is used to partially replace the patient's blood (hyperbilirubinemic blood, in the case of newborn jaundice). Only at specialist centers is ET used, and there is a considerable danger of morbidity and death from vascular accidents, hypocalcemia, necrotizing enterocolitis, biochemical and haematological problems, and cardiac consequences.[37] The procedure's overall mortality rate varies greatly throughout centers, ranging from 0.3% to 0.7%, but in underdeveloped nations, it can as high as 17%.[38] It is anticipated that ET quickly and sufficiently reduces bilirubin levels. It's still unclear, though, if newer, less intrusive, and more effective therapies could successfully replace ET.

5. Experimental treatments

Conventional therapies are typically quite successful. However, in certain circumstances, an alternative strategy could be needed, combining routine therapy with experimental medications to enhance the prognosis of newborn hyperbilirubinemia. These experimental therapies can be categorized into two groups: those that provide neuroprotection without changing TB levels and those that aim to prevent the harmful buildup of UCB.

Orlistat and ursodeoxycholic acid

Through the intestinal mucosa, UCB can reabsorb itself in the intestinal lumen after diffusing from the blood compartment. Improving transmucosal bilirubin elimination efficiency could hasten UCB GI transit and be included in standard treatment. Ursodeoxycholic acid (UDCA) and orlistat are meant to encourage the excretion of UCB in the feces. Although the main purpose of orlistat is to stop the body from absorbing fats from food, UDCA also reduces the rate at which cholesterol molecules are absorbed by the gut. Orlistat plus UDCA therapy has been shown to lower plasma UCB concentrations by enhancing UCB-bile salt complex excretion in the stool. Both Crigler-Najjar patients and Gunn rats have shown improvement in bilirubin levels following this method's use.[39] More investigation is necessary to rule out the long-term adverse effects of orlistat and UDCA, as both have demonstrated hepatotoxic side effects. This raises concerns regarding the cost-effectiveness of these drugs.

Human serum albumin

In infants with severe hyperbilirubinemia, bilirubin encephalopathy does not always occur. The high TB concentrations may interact with a number of variables to either avoid or predispose to kernicterus. Human serum albumin (HSA) is one of these

components that is crucial to the bilirubin pathway. Actually, UCB travels to the liver in this form after binding to serum albumin. As long as the bilirubin is bound to albumin, UCB cannot cross the blood-brain barrier and enter the brain. The bilirubin-binding capacity of plasma albumin reaches saturation, and in lipophilic tissues, the unbound fraction (Bf) increases and accumulates. Theoretically, enhancing UCB's capacity to bind plasma should release bilirubin from tissues into plasma and halt neurodamage(Figure 2).

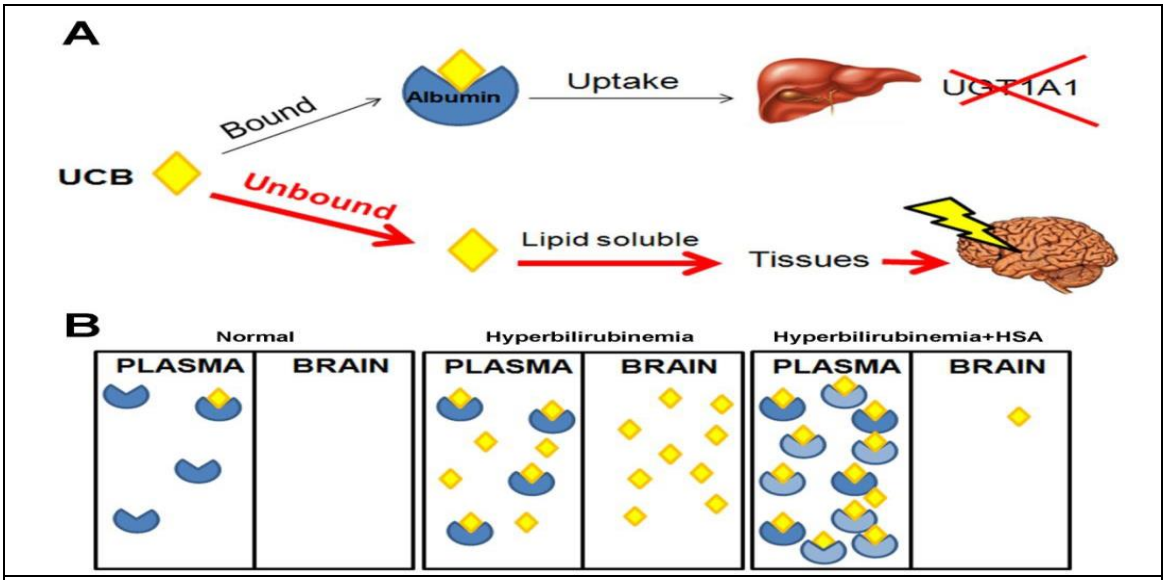

Figure 1: Reasons for administering the HSA. A) UCB's impact in the event of glucuronosylation deficiencies; B) UCB's mobilization from tissue to the newly supplied binding site in plasma is shown schematically.

Minocycline

MNC, a second-generation tetracycline, appears to have anti-inflammatory properties that are entirely different from its antibacterial properties. Because to MNC's anti-inflammatory properties, numerous neurodegenerative illnesses have been related to it, such as stroke, PD, and HD. The compound's capacity to inhibit the microglial inflammatory response is mostly to blame for this. MNCs inhibit inflammation by their effects on microglia, immune cell activation, and the resultant release of nitric oxide (NO), matrix metalloproteinases (MMPs), chemokines, and lipid mediators of inflammation. Additionally, pro-inflammatory cytokines are produced by astrocytes,

neutrophils, macrophages, and microglial cells. These cytokines include interleukin (IL) 1β (IL1β), IL6, and tumor necrosis factor α (TNF-α).

It has been shown that the neuroprotective properties of MNC are partially due to indirect activities in lowering glial (astrocytic/microglial) caspase 1 and inducible nitric oxide synthase (iNOS) activity, even if direct neuroprotective advantages have also been shown.[40]

Association between weight loss and neonatal hyperbilirubinemia

Within 72 hours of delivery, 63.6% of the neonates in the Pulmamidi et al (2021)[41] research showed signs of severe hyperbilirubinemia. 18.1% of breastfeeds demonstrated appropriate feeding, compared to 81.8% (45/55) who exhibited poor feeding. They found a correlation between birth weight decrease and marked hyperbilirubinemia. Neonatals with term gestation had increased weight loss and hyperbilirubinemia in 63.6% (35/55) of the cases. After 72 hours of delivery, severe hyperbilirubinemia of >12 mg/dl was linked to weight loss of more than 7%.

A total of 115 (33.5%) newborns in the Yang et al. (2013)[42] trial showed signs of severe hyperbilirubinemia 72 hours after delivery. Significant hyperbilirubinemia was shown to be statistically substantially connected with BWL percentages in the first three days following delivery, as well as 72 hours later. The BWL% on day three was a more reliable indicator of severe hyperbilirubinemia 72 hours after birth than it was on days one and two. Moreover, regardless of whether the baby was fed only breastmilk or a mix of formula and milk, the BWL for the first three days seemed to be influenced by the BWL for the previous day. This demonstrates that, with the appropriate care, newborns with a high BWL% on day 1 can still avoid more severe hyperbilirubinemia. It also highlights the significance of BWL percentage on days 2 and 3. In their investigation,

they discovered that there was no statistically significant correlation between breastfeeding and hyperbilirubinemia.

It was discovered in the Boskabadi et al. (2014)[43] study on breastfed term newborns that pathologic weight loss and hyperbilirubinemia are inhibited by frequent breastfeeding. Neonatal weight loss might exacerbate hyperbilirubinemia. They observed that infants with severe hyperbilirubinemia (>20 mg/dl) lost three times as much weight on average as those with mild hyperbilirubinemia (< 20 mg/dl). Therefore, it is advised to provide appropriate feeding therapies for newborns who have eating issues.

According to Chang et al. (2020),[16] hyperbilirubinemia was linked to newborns with lower gestational ages and higher percentages of weight loss. After 48 hours and 72 hours following delivery, they observed weight losses of around 8% and 11%, respectively. According to a prior study, infants with a BWL of greater than 7% had a 1.4-fold higher risk of jaundice in the early stages of their lives.[44]

In their research, Salas et al. (2009)[45] found that 5% of breastfed babies had a readmission due to hyperbilirubinemia. It is well known that the birth weight and gestational age are directly correlated. According to Salas et al.[45] 38% of their patients had considerable weight loss, and babies who had significant weight loss were much more likely to have severe hyperbilirubinemia (> 20 mg/dL).

Severe hyperbilirubinemia was defined as a bilirubin level more than 15 mg/dL from days 4 to 10 of life, and Huang et al. (2009)[44] found that 23.6% of their subjects experienced severe hyperbilirubinemia.

A prospective observational study by Rajeshkhanna et al. (2021)[41] on term and preterm babies weighing more than 2500 gms revealed that a weight loss of more than 7% within the first 72 hours of life could both be a useful clinical factor to prevent

significant hyperbilirubinemia within that time frame and potentially be a predictor of neonatal hyperbilirubinemia.

In a prospective cohort study on term newborns, Prachukthum et al. (2020)[46] found that term infants with neonatal hyperbilirubinemia experienced considerable weight loss during their two days in the hospital, necessitating readmission for phototherapy. As such, it was recommended that newborns with a BWL of 5% or above require close monitoring.

Twenty-eight (33%) of the 86 neonates with idiopathic hyperbilirubinemia in the Aylin Tarcan et al. (2005)[47] research showed significant weight loss. Twelve percent of the 86 jaundiced infants in total-nearly all of them were nursed exclusively-had significant weight loss in addition to hypernatremia. Risk factors for infants who were readmitted to the hospital due to jaundice within 14 days after birth were examined by Geiger et al. (2001).[48] It was discovered that 26.8% of the babies in this group had trouble eating, and 24.2% of them were dehydrated.

Maisels JM (1988) and Bertini G, (2001) examined the outcomes of newborns with bilirubin levels ≥12.9 mg/dL with those with bilirubin levels.[49] Oddie et al. (2001) found that 34 out of 904 neonates who were readmitted before 29 days of age had lost more than 10% of their body weight.[50] Breastfeeding jaundice is a condition of the first week of life, and the majority of instances were caused by failure to nurse in the days following delivery, according to two distinct investigations by Gartner LM et al (1999, 2001)[51,52]. Just one of the 904 newborns was admitted due to jaundice, but eight (0.9%) of them had serum Na levels greater than 150 mEq/L. They came to the conclusion that this issue exacerbates physiologic jaundice. Furthermore, a diet low in milk may cause meconium, which is high in bilirubin, to pass more slowly, increasing the absorption of bilirubin.

Whitmer DI et al. (1983)[53] looked at the connection between low calorie consumption and jaundice.[17] Even though the data did not prove that all breastfeeding newborns with weight loss and hypernatremia develop jaundice, they did show that children with this combination of problems may be readmitted to the hospital with worsened jaundice.

MATERIALS & METHODS

MATERIALS AND METHODS

Source of Data

All newborn healthy term and post term babies delivered at RL Jalappa hospital during the period of study and whose parents have consented to be a part of the study.

Study Design

A Prospective Observational study**.**

Study Period

September 2022 to December 2023.

Method of Collection of Data

Inclusion Criteria

All term and post term newborns who are clinically stable.

Exclusion Criteria

- Rh incompatibility
- Neonates of Diabetic mother
- Hypothyroid babies
- Babies born with congenital anomalies
- Babies born with genetic disorders
- Direct/conjugated bilirubinaemia
- Birth trauma- Cephalohematoma, Subgaleal haemorrhage, etc.
- Neonatal sepsis

Statistical Methods

A Microsoft Excel data sheet will be used to record the data, and SPSS version 22 software will be used for analysis. The representation of categorical data will take the shape of proportions and frequencies.

The chi-square significance test will be utilized. The continuous data will be described using the mean and standard deviation. The independent t test will be used as a significance test to ascertain the mean difference. P-values below 0.05 are considered statistically significant. Data visualization: A range of graph types were created using Microsoft Word and Excel.

A P value of 0.05 was considered statistically significant, meaning that the probability that the result is true, after all essential assumptions about the statistical test requirements were made.

Statistical software, Microsoft Excel and SPSS version 22 (IBM SPSS Statistics, Somers, NY, USA), were used for data analysis.

Methodology

All neonates fulfilling the inclusion criteria will be included in the study.

The following neonatal data were to be gathered: blood group, frequency of breastfeeding, signs and symptoms of hyperbilirubinemia, gender, gestational age, birth weight, and Direct Coombs test DCT (if applicable).

We will weigh the infant at birth as well as at 24, 48, 72, 96 and 120 hours of life. Newborns who were clinically icteric and whose TCB was found to be within the phototherapy range based on the AAP nomogram will have their total serum bilirubin evaluated.

Formula to Calculate Weight Loss

Weight Loss = (Birth Weight - Present Weight)*100 / Birth Weight

Total bilirubin level according to AAP nomogram risk chart will be taken as cut-off value for intervention. In each case, a correlation between the percentage of body weight lost and serum bilirubin will be performed. When determining whether a newborn delivered at a gestation of 35 weeks or more needs phototherapy or an exchange transfusion, the American Academy of Paediatrics (AAP) guidelines should be applied.

Two age-appropriate nomograms are offered by AAP: one for exchange transfusion and the other for phototherapy. Decisions are made using the TB value, and the direct portion shouldn't be subtracted from it. Risk factors include presence of isoimmune hemolytic disease, G6PD deficiency, other hemolytic conditions, sepsis, hypoalbuminemia (albumin < 3g/dl) and clinical instability in previous 24 hours.

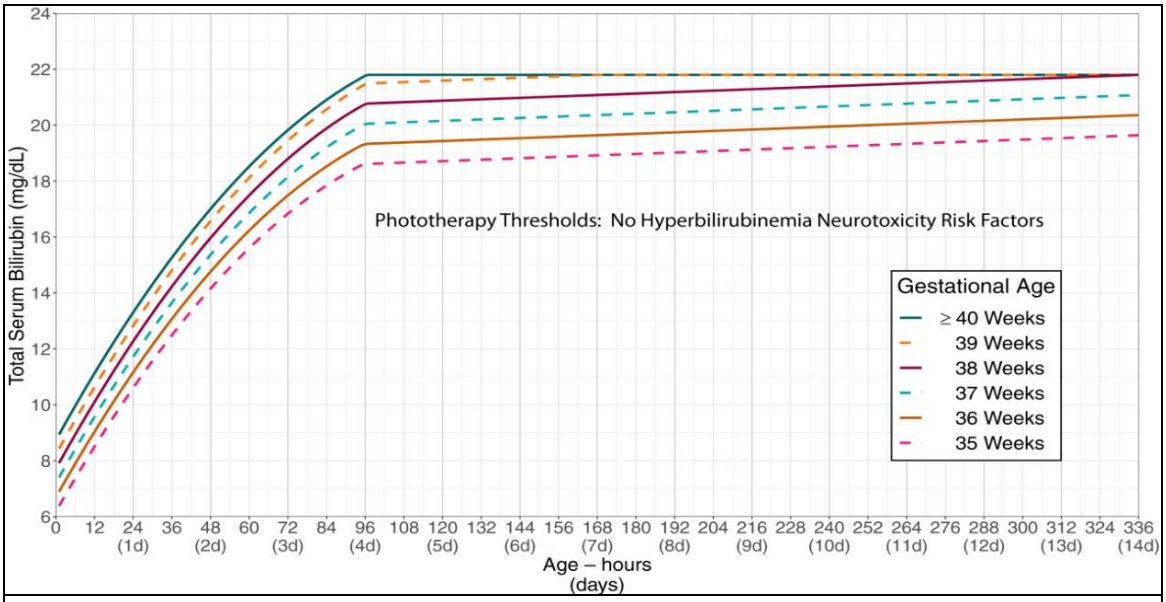

Figure 2: AAP nomogram for phototherapy in hospitalized infants of 35 or more weeks gestation without neurotoxicity risk factors

The weight of the newborns will be noted as follows: birth weight on day 1, which is 24 hours after delivery; day 2, which is 48 hours after delivery; day 3, which is 72

hours after delivery; day 4, which is 96 hours after delivery; and day 5, which is 120 hours after delivery, which is the date of birth. Calculations of percentages and ratios will be made when data is loaded into Excel sheets. Neonatal blood samples will be taken, and serum bilirubin levels will be estimated. Based on the AAP nomogram, the babies were separated into two groups: those with significant hyperbilirubinemia and those without.

Phototherapy was administered to the neonates in the significant hyperbilirubinemia group. When assessed, the relationships between the total bilirubin level and the birth weight loss (BWL) % within the first week were examined individually.

Hemolysis will be checked for and the reticulocyte count (relevant instances) will be calculated on smears stained with New Methylene Blue. Agglutination techniques will be used for both the direct and indirect Coombs test. Rh type and the blood groups of the mother and the newborn will be documented.

Sample Size

Sample size was estimated by using the proportion of significant neonatal hyperbilirubinemia was 13% from the study by Srisuwanet al.[7] using the formula.

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

$\mathbf{Z_{1-\alpha/2}}$ = is standard normal variate (at 5% type 1 error ($P<0.05$) it is 1.96 and at 1% type1 error ($P<0.01$) it is 2.58). As in majority of studies P values are considered significant below 0.05 hence 1.96 isused in formula.

P= Expected proportion in population based on previous studies orpilot studies.

d= Absolute error or precision.

P = 13% or 0.13.

q = 87% or 0.87.

d = 7.5% or 0.075.

The study will contain a sample size of 77 people based on the above values at the 95% Confidence level. The study will involve a minimum sample size of 77 + 7.7 = 85 people, accounting for 10% non response.

RESULTS

RESULTS AND ANALYSIS

Table 3 : Distribution based on gender

Gender	Number	Percentage
Male	49	57.6%
Female	36	42.4%

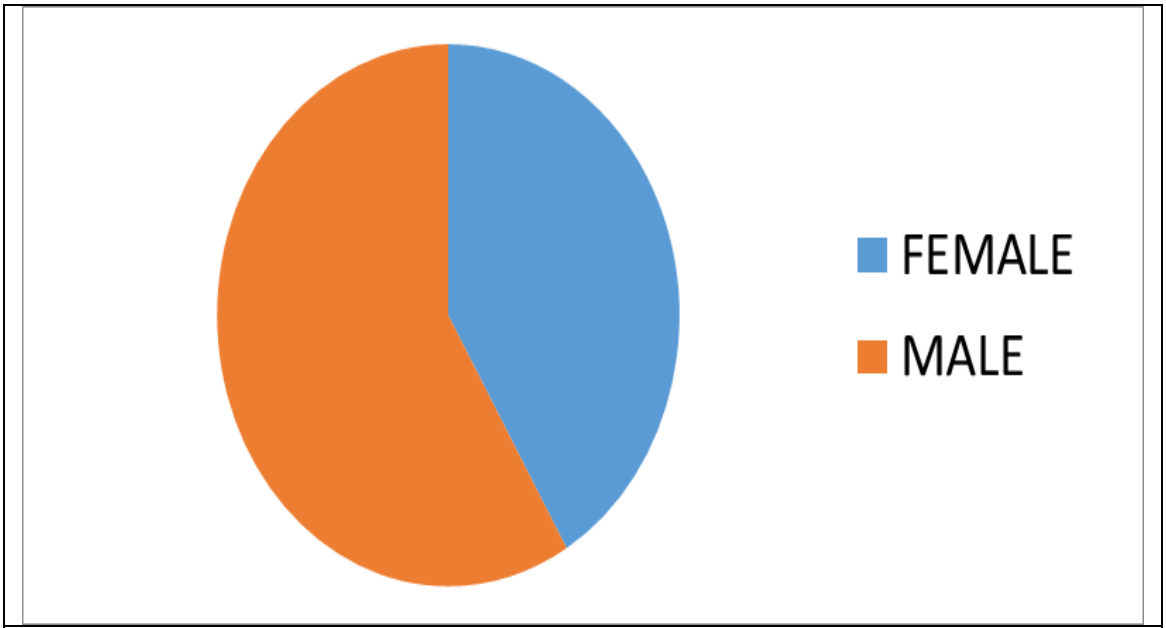

Figure 3

Out of the study population (n=85), 42.4% of the neonates were female and 57.6 % of the neonates were male.

Table 4 : **Distribution based on gestational age (n=85)**

Gestational age	Number	Percentage
Term	81	95.3%
Postterm	4	4.7%

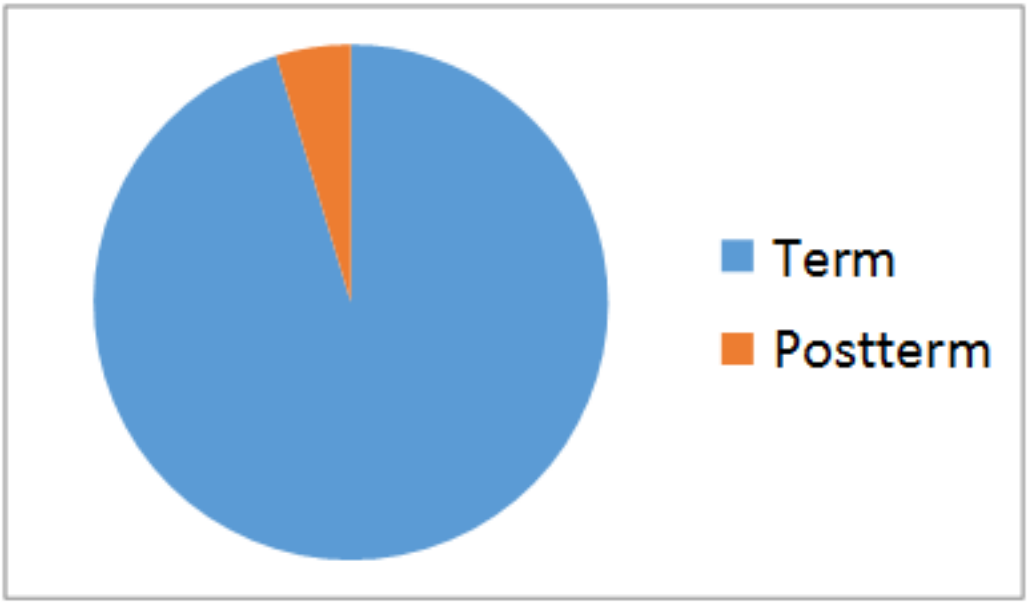

Figure 4

95.3% of the neonates were full term and 4.7% of the neonates were postterm.

Table 5 : Distribution based on mode of delivery (n=85)

Mode of delivery	Number	Percentage
NVD	24	28.2%
Instrumental	11	12.9%
LSCS	50	58.8%

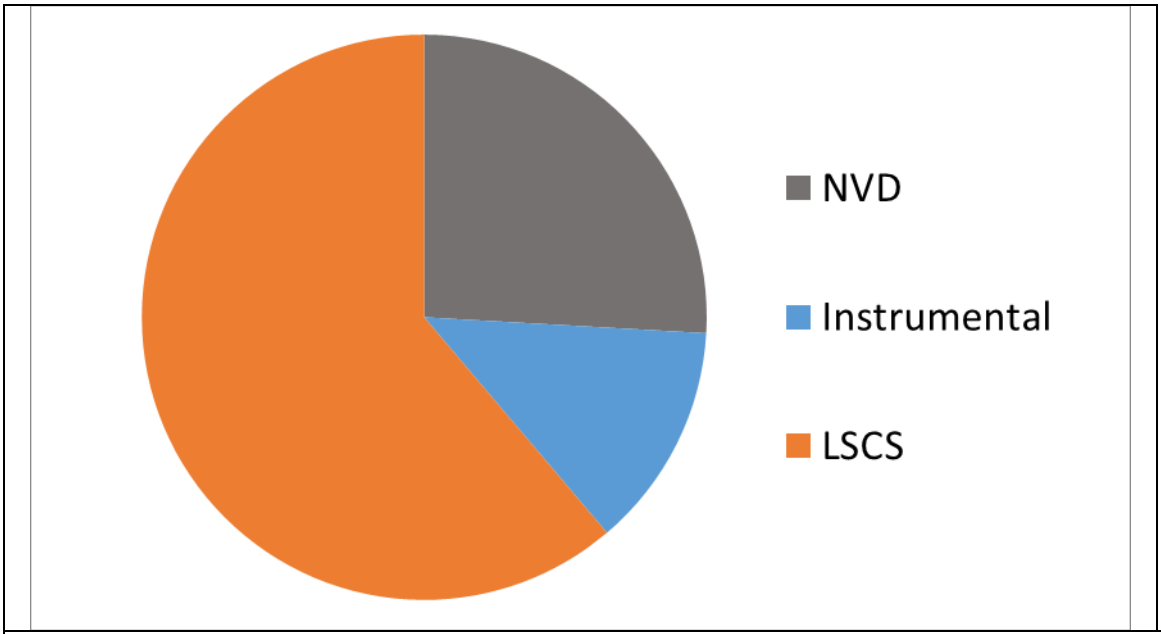

Figure 5

58.8% of the subjects were delivered through LSCS followed by 28.2% of the subjects delivered vaginally and 12.9% of the subjects had Instrumental delivery.

Table 6 : Distribution based on gravida (n=85)

Gravida	Number	Percentage
Primi	**42**	**49.4%**
Multi	**43**	**50.6%**

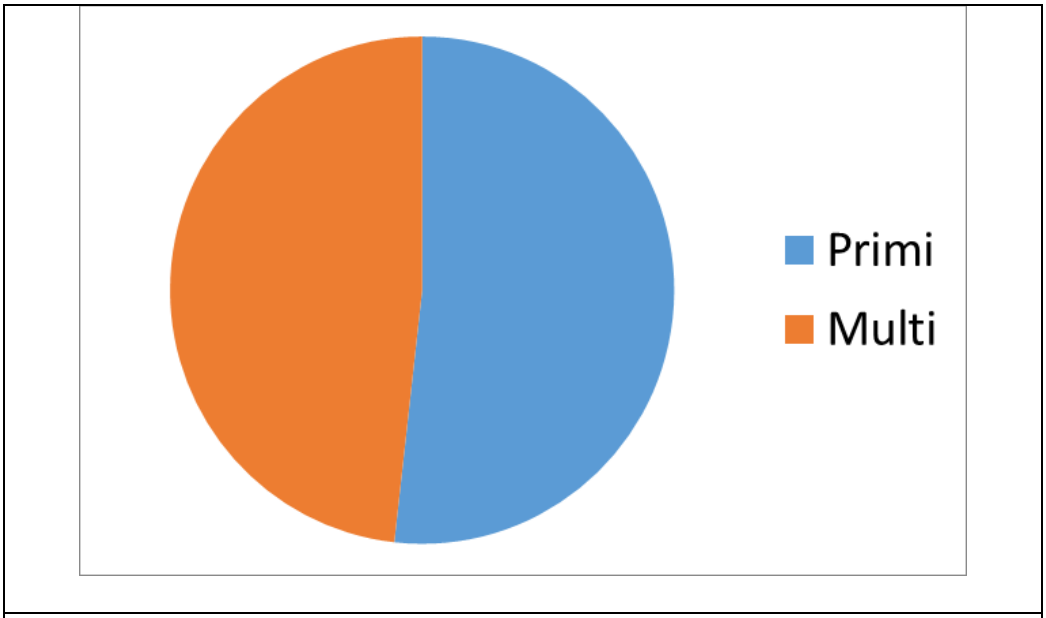

Figure 6

50.6% of the neonates were born to multigravida and 49.4% of the neonates were born to primigravida.

Table 7: Distribution based on S.Bilirubin in phototherapy range (n=85)

Serum Bilirubin	Number	Percentage
In Phototherapy range	63	74.1%
Not in Phototherapy range	22	25.9%

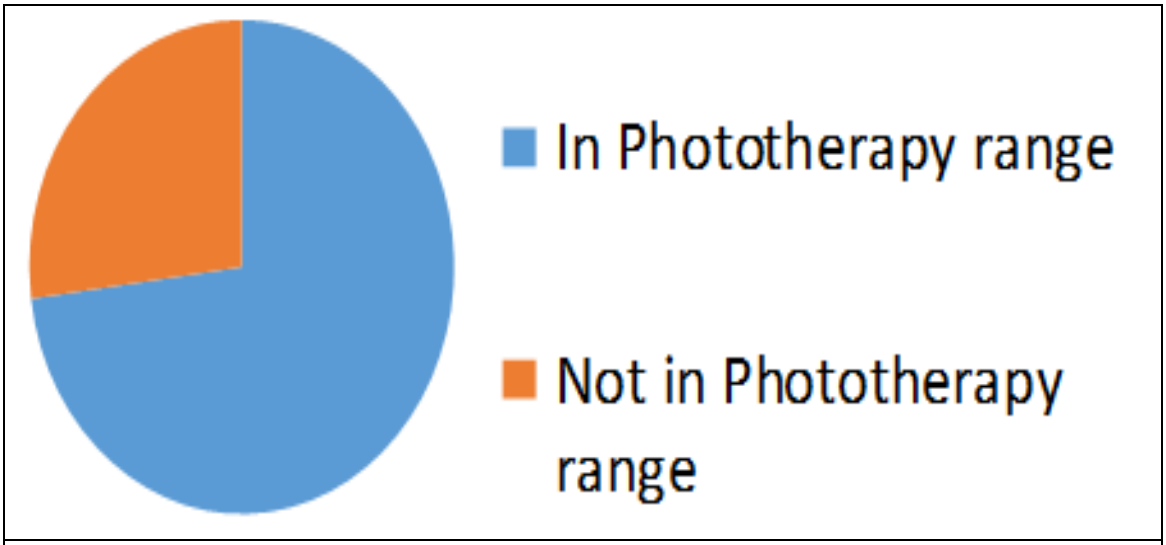

Figure 7

74.1% of the subjects had serum bilirubin in phototherapy range and 25.9% of the subjects didn't have serum bilirubin in phototherapy range.

Table 8 : Distribution of subjects according to significant hyperbilirubinemia and gravida

	Without significant hyperbilirubinemia		With significant hyperbilirubinemia	
	N	%	N	%
MULTI	14	32.6%	29	67.4%
PRIMI	8	19.0%	34	81.0%

Among the Multipara subjects significant hyperbilirubinemia was found in 67.4% and 32.6% didn't had significant hyperbilirubinemia. Among the primipara subjects significant hyperbilirubinemia was found in 81.0% and 19.0% didn't had significant hyperbilirubinemia. P Value 0.216, there was no statistically significant difference found between groups with respect to gravida.

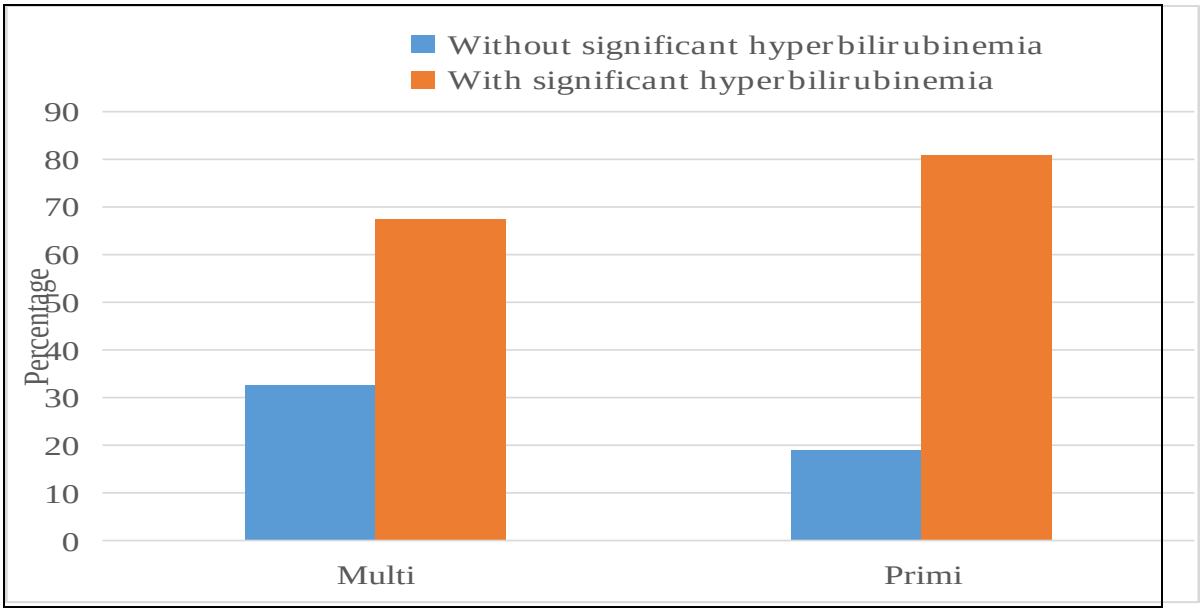

Fig 8: Graph showing Distribution of subjects according to significant hyperbilirubinemia and gravida

Table 9 : **Distribution of subjects according to significant hyperbilirubinemia and mode of delivery**

	Without significant hyperbilirubinemia		With significant hyperbilirubinemia	
	N	%	N	%
Instrumental	1	9.1%	10	90.9%
LSCS	7	14.0%	43	86.0%
NVD	14	58.3%	10	41.7%

Among subjects delivered through LSCS significant hyperbilirubinemia was found in 86.0% and 14.0% didn't have significant hyperbilirubinemia. Among subjects delivered through vaginally significant hyperbilirubinemia was found in 41.7%and 58.3% didn't had significant hyperbilirubinemia. Among subjects delivered through Instrumental significant hyperbilirubinemia was found in 90.9% and 9.1% didn't had significant hyperbilirubinemia. P Value <0.001, there was a statistically significant difference found between groups with respect to mode of delivery.

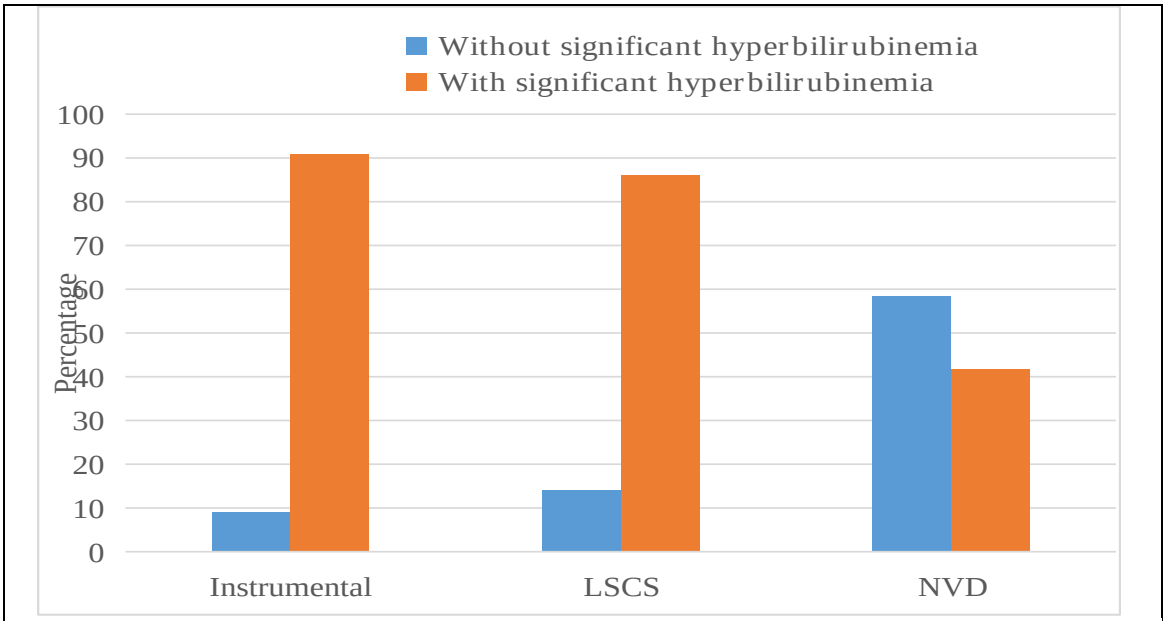

Fig 9: Graph showing Distribution of subjects according to significant hyperbilirubinemia and mode of delivery.

Table 10 : **Distribution of subjects according to day on which significant weight loss present**

	Frequency	Percent
Day 2	5	5.9
Day 3	28	32.9
Day 4	48	56.5
Day 5	4	4.7
Total	85	100.0

Majority of the subjects 56.5% had significant weight loss on day 4 followed by day 3 which was 32.9%, on day 2 it was 5.9% and on day 5 it was 4.7%.

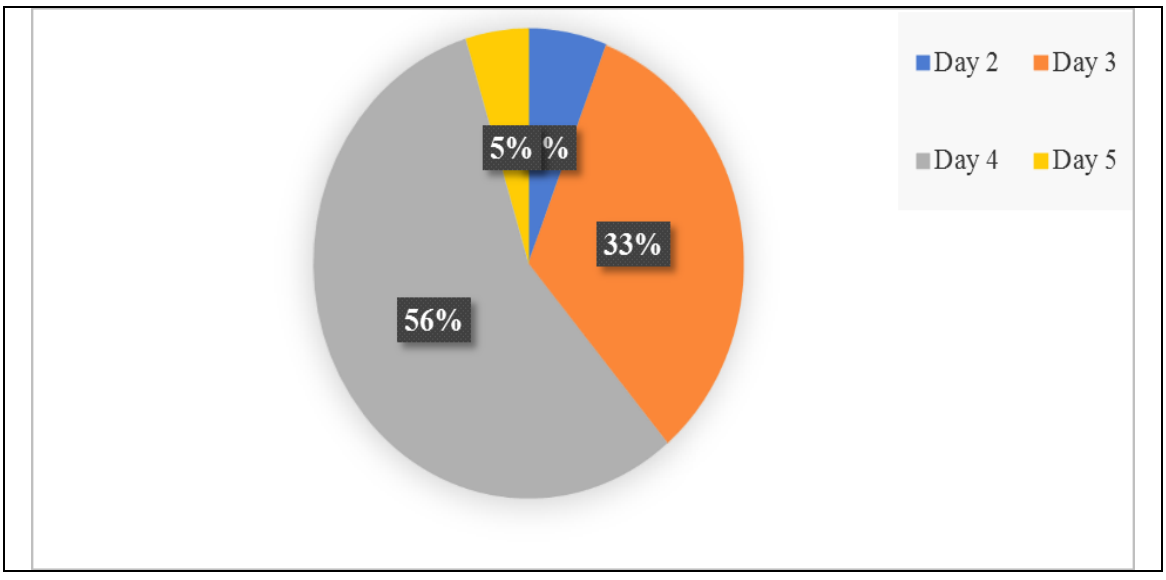

Fig 10: Graph showing Distribution of subjects according to day on which significant weight loss present

Table 11 :Day 2 Distribution of subjects according to Significant Weight loss and significant hyperbilirubinemia.

	Without significant hyperbilirubinemia		**With significant hyperbilirubinemia**	
	N	%	N	%
Significant weight loss	0	0%	5	100.0%

Among subjects who had Significant Weight loss on day 2, 100% of them had significant hyperbilirubinemia.

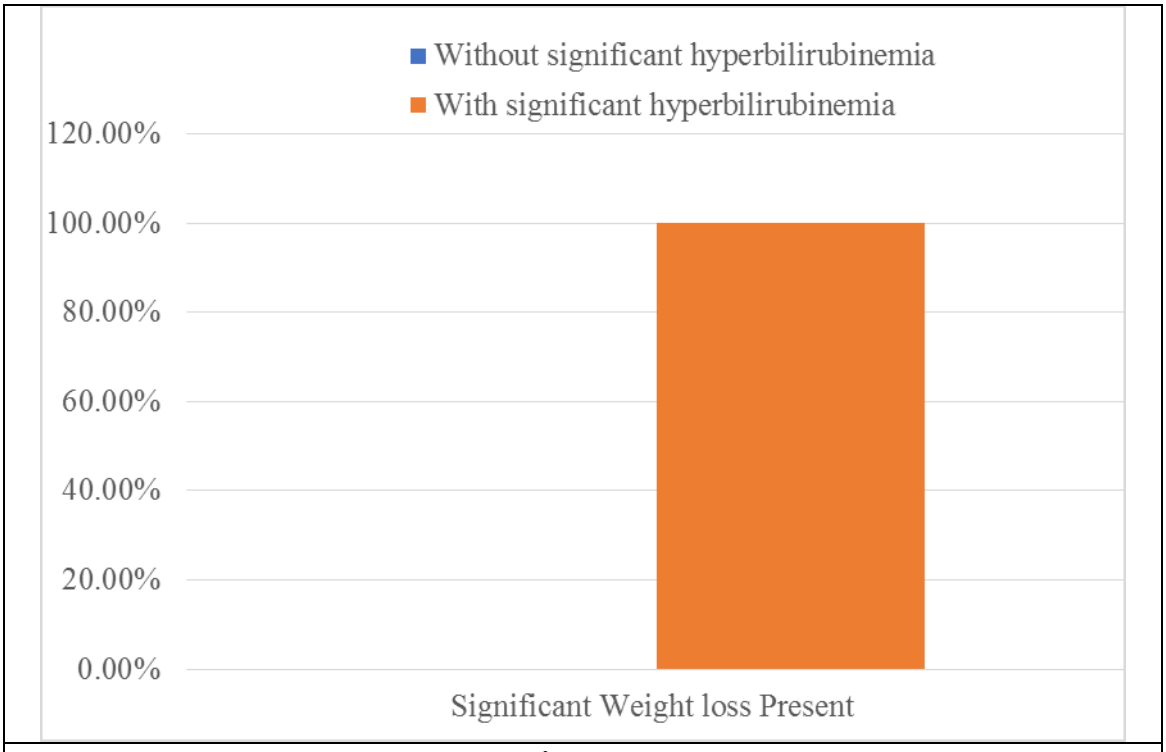

Figure 11

Table 12 : Day 3 Distribution of subjects according to Significant Weight loss and significant hyperbilirubinemia.

	Without significant hyperbilirubinemia		**With significant hyperbilirubinemia**	
	N	%	N	%
Significant weight loss	4	14.3%	24	85.7%

Among subjects who had Significant Weight loss on day 3, 85.7% of them had significant hyperbilirubinemia and 14.3% of them didn't had significant hyperbilirubinemia.

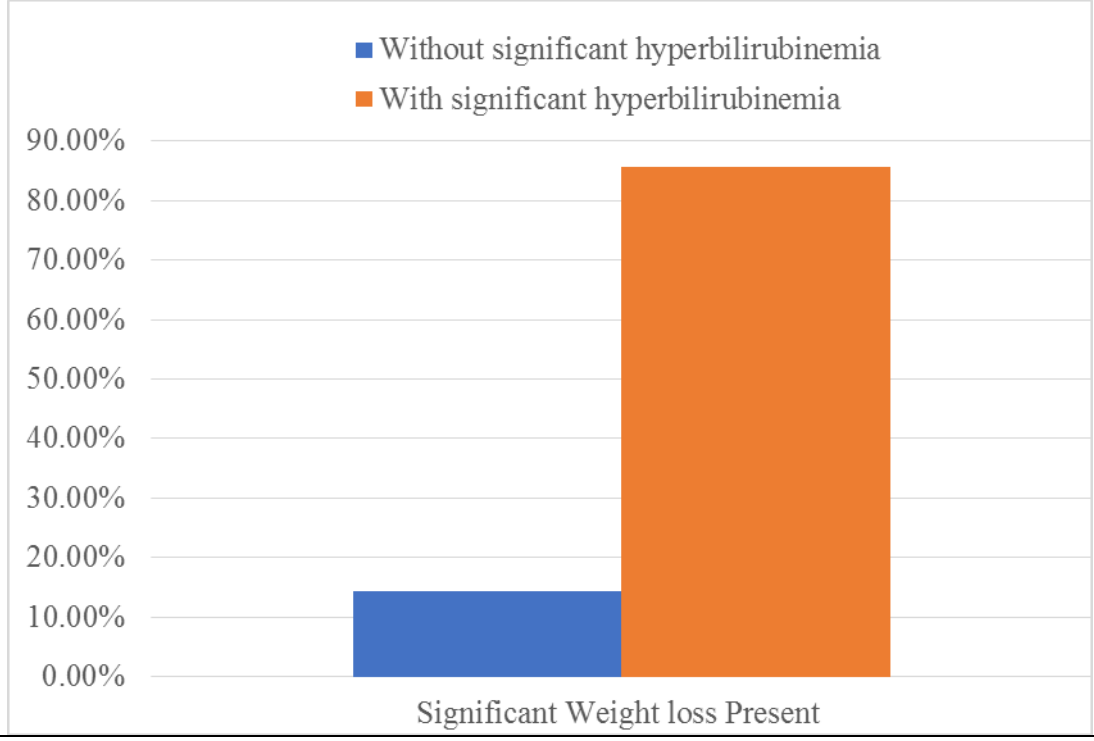

Figure 12

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Table 13 :On Day 4 Distribution of subjects according to Significant Weight loss and significant hyperbilirubinemia

	Without significant hyperbilirubinemia		**With significant hyperbilirubinemia**	
	N	%	N	%
Significant weight loss	14	29.2%	34	70.8%

Among subjects who had Significant Weight loss on day 4, 70.8% of them had significant hyperbilirubinemia and 29.2% of them didn't had significant hyperbilirubinemia.

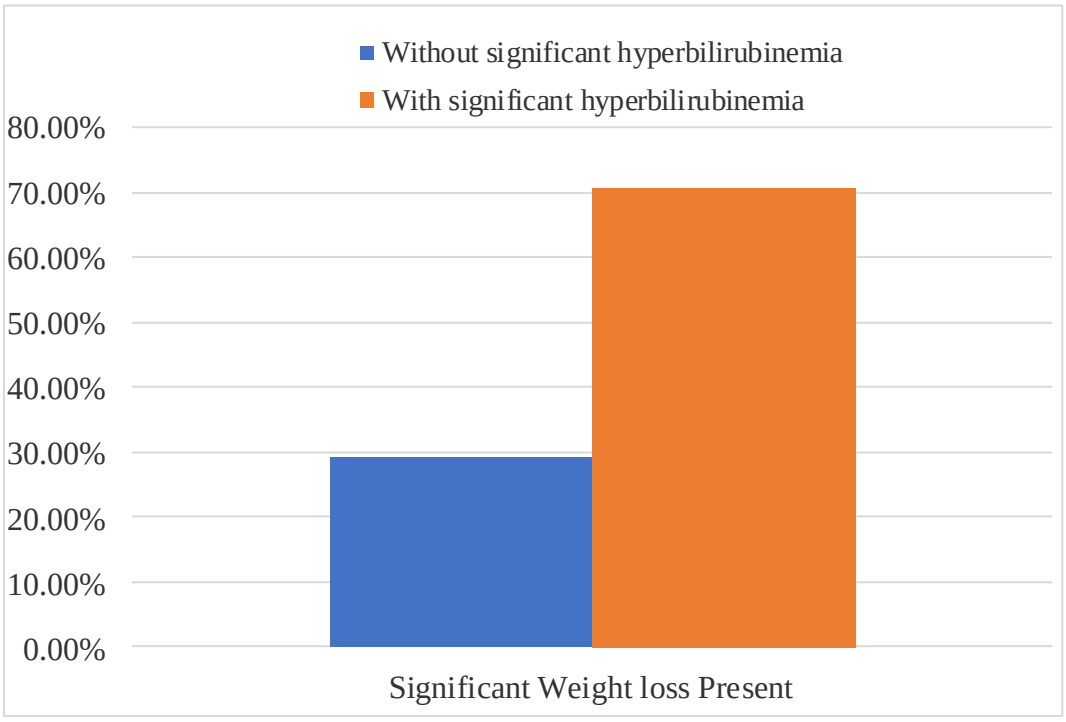

Figure 13

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Table 14 : On Day 5 Distribution of subjects according to Significant Weight loss and significant hyperbilirubinemia

	Without significant hyperbilirubinemia		**With significant hyperbilirubinemia**	
	N	%	N	%
Significant weight loss	4	100%	0	0%

Among subjects who had Significant Weight loss on day 5, 100% of them didn't had significant hyperbilirubinemia.

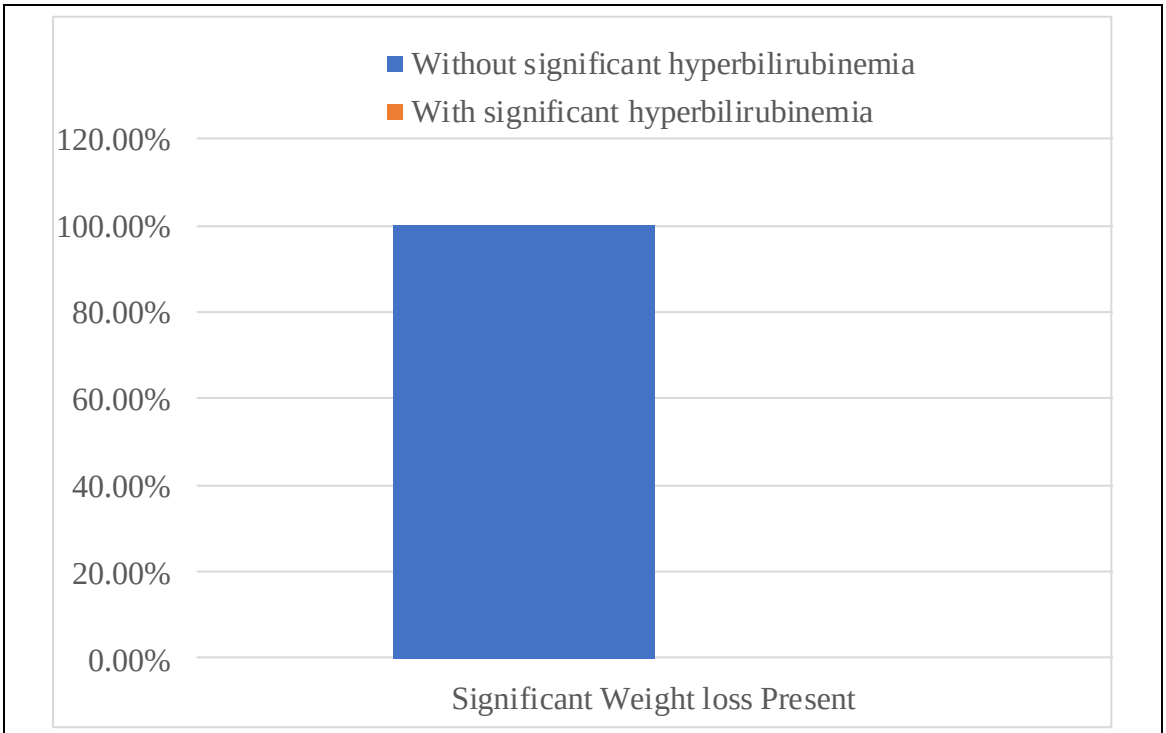

Figure 14

DISCUSSION

DISCUSSION

Incidences of neonatal jaundice vary from around 2 to 35% globally, and neurological impairment is linked to the harmful consequences of unconjugated hyperbilirubinemia.[47,48] Furthermore, a growing worry is the possibility of newborn jaundice brought on by excessive body weight loss (due to inadequate calorie intake).[49,50] Serum bilirubin levels (SBL) more than 12 mg/dL are considered hyperbilirubinemia,[51] whilst a total SBL greater than 5 mg/dL is considered newborn jaundice.[52] A high SBL can cause brain injury and encephalopathy, which can manifest as palsy of vertical gaze, central neural deafness, and choreoathetoid cerebral palsy.[53,54]

Hyperbilirubinemia is the most common problem in the early infant stage. Hyperbilirubinemia can cause encephalopathy (kernicterus), albeit this is not common.[55] Before tracking the bilirubin level in healthy term newborns, we must employ an objective indicator to identify hyperbilirubinemia since clinical jaundice manifests when the serum bilirubin level climbs above 5.6 mg/dl.[56,57] A rise in unconjugated serum bilirubin during the first week of life is the cause of hyperbilirubinemia in healthy term babies.[58,59] Dehydration from low-calorie and inadequate feeding during the first few days after delivery may be the cause of hyperbilirubinemia if additional explanations cannot be found. One risk factor for hyperbilirubinemia is weight loss.[59] A healthy term baby will typically lose 4–7% of their birth weight in the first few days of life.[60] The purpose of this study was to ascertain whether the greatest weight loss following delivery was associated with the onset of noticeable hyperbilirubinemia during the first week of life.

Differentiating between pathological and physiological jaundice is important since the former has a complex mechanism.[61] Breastfeeding-associated jaundice, often referred to as breast "nonfeeding" jaundice or breastfeeding-related jaundice, is caused by

an increase in enterohepatic bilirubin circulation and is linked to nursing during the second and fourth days following delivery.[61,62] Although the exact process is yet unknown, it is believed to be connected to a decrease in caloric intake that causes an increase in bilirubin's enterohepatic circulation. Reduced calorie consumption may also result in a decrease in the amount of unconjugated serum bilirubin cleared, which might be another underlying cause.[61]

This study examined the relationship between weight loss and neonatal hyperbilirubinemia in 85 newborns. The findings of the present study and those of similar research available in the literature are as follows.

42.4% of the 85 individuals in this study were female, and 57.3 percent of the subjects were male. 58.1% (32/55) of the participants in the Rajeshkhanna Pulmamidi et al[41] research were men, while 41.8% (23/55) were women. 64.6% of the 79 newborns in Salas et al.'s[15] research were male.

Gender is one of the risk factors for NWL, according to the findings of the Haseli A et al. research. Gender was one of the predictors of NWL in the first 24 hours following birth in a study by Jane (2010).[64] among their study, however, the kind of breastfeeding was linked to the gender of the mother since, among newborns breastfed, the female to male ratio was 118 to 100, but among infants given formula, it was 52 to 100. As such, the sort of nursing a female newborn receives influences her WL. Because of this, medical professionals need to be aware of WL in breastfeeding female newborns, particularly those who were delivered by cesarean section.

There is a connection between significant hyperbilirubinemia and birth weight decrease. On day 4, most of the newborns in our research had lost a substantial amount of weight, and of those, 70.8% had a significant amount of hyperbilirubinemia. Seventy-two hours after delivery, 63.6% of the neonates in the Chang RJ study[16] had severe

hyperbilirubinemia. A total of 115 (33.5%) newborns in the Yang et al. study[42] showed signs of severe hyperbilirubinemia 72 hours after delivery. Similar weight losses of around 8% and 11%, respectively, were reported by Chang et al.[16] following 48 and 72 hours of delivery. After 72 hours of life, 219 newborns (25.1%) showed evidence of severe hyperbilirubinemia. Comparing those with future hyperbilirubinemia to those without, the former showed lower gestational ages, greater BW loss percentages on the second and third days of life, and higher maximum BW loss percentages. There was no discernible difference in the distribution of newborn gender, BBW, or delivery mode between these two groups. Only a high maximum BW loss percentage and a short gestational age were found to be substantially linked with eventual hyperbilirubinemia by multiple logistic regression.

We examined the BW loss percentages on the second and third days of life independently in order to determine the best cut-off values for the prediction of ensuing neonatal hyperbilirubinemia. We computed the relative odds ratios (OR) for the development of hyperbilirubinemia as well as its present. Neonatals with BW loss percentages more than 8% on the second day were much more likely to develop neonatal hyperbilirubinemia [OR Z 1.45; 95% confidence interval (95% CI) Z 1.06, 1.97; p Z 0.019]. For the BW loss percentage of 8% on the second day, the positive predictive value (PPV) was 29.3%, the negative predictive value (NPV) was 77.7%, the sensitivity was 46.6%, and the specificity was 62.4%. Neonatal hyperbilirubinemia that developed later in life was substantially correlated with a BW loss percentage on the third day of life between 6% and 11%. The best nourishment for newborns is breast milk, and neonatal hyperbilirubinemia is rarely fatal or complex, hence Rajeshkhanna Pulmamidi et al.[41] propose using the larger BW loss % as the cut-off value for predicting eventual hyperbilirubinemia. The following data were obtained using the cut-off value of 11% BW

loss percentage at 3 days old (OR Z 2.01; 95% CI Z 1.16, 3.46; p Z 0.012): PPV = 37.7%, NPV = 76.8%, sensitivity = 11.7%, and specificity = 93.8%.

Previous studies have classified serious weight loss due to inadequate breastfeeding as occurring in 7% or more of BBW.[65] Supplementary feeding was administered in our nursery to breastfed newborns who had lost more than 10% of their body weight, unless their parents had insisted on exclusive nursing. However, these norms are not empirical. According to the current research, weight loss of >8% of BBW after 48 hours (OR Z 1.45; 95% CI Z 1.06, 1.97) and >11% of BBW after 72 hours (OR Z 2.01; 95% CI Z 1.16, 3.46) are the best cut-off values for prediction of impending hyperbilirubinemia; NPVs were 77.7% and 76.8%, respectively.

The weight of newborns is also impacted by the practice of breastfeeding. 81.8% (45/55) of the research participants had insufficient breastfeeding, whereas 18.1% demonstrated appropriate breastfeeding. In their investigation, Yang et al. discovered that there was no statistically significant correlation between breastfeeding and hyperbilirubinemia. In their study, Salas et al.[15] found that 5% of breastfed babies had a readmission due to hyperbilirubinemia.

Haseli A et al.'s study[63] found that breastfeeding raises WL ≥ 5% and WL ≥ 7% by 7 and 3 times, respectively. Because the mean WL of newborns fed baby formula (2.78 ± 1.65%) was lower than that of infants exclusively breastfed (4.89 ± 2.86%), this variable was the greatest predictor of NWL. In this aspect, McDonald's statistics were similarly identical (6.6% versus 3.5%).[66] The current study's results are in line with the majority of other research's findings.[8,35,67] Davanzo et al., on the other hand, showed that newborns given breast milk had a mean WL of 6.3% compared to 7.5% for formula-fed infants.[68]

The American Academy of Pediatrics (AAP) recommendations state that a weight gain (WL) of greater than 7% within 3 to 5 days of delivery may be a sign of potential breastfeeding issues.[69] Notwithstanding the established advantages of breastfeeding, babies who are nursed frequently experience dehydration and inadequate weight gain following delivery. These illnesses are frequently avoidable, and if detected in time, they won't harm babies in the long run.[70,71] Since the amount of milk given to breastfed newborns determines their WL.[72] Hospitals that prioritize breastfeeding should take breastfeeding protocols and practices seriously. They should also provide training to moms and implement other policies that support effective breastfeeding. These steps might lower newborn NWL if they are taken before to delivery. The late initiation of formula feeding following a time of breast milk deprivation was another factor contributing to nonverbal lactation (NWL) in babies given formula in the current research.[16]

It is well known that the birth weight and gestational age are directly correlated. Ninety-one (95.3%) of the neonates in this research were term newborns; only 4.7% of the patients were postterm. While neonates with lower gestational age and greater weight loss percentage were also linked to hyperbilirubinemia in the Chang et al. study[16], 63.6% (35/55) of the neonates with term gestation showed greater weight loss and were also linked to hyperbilirubinemia in the Rajeshkhanna Pulmamidi et al.[41] study. Prachukthum S. et al.[73] The GA of infants with hyperbilirubinemia was 38.4±1 weeks, whereas the GA of infants in the non-hyperbilirubinemia group was 38.8±0.9 weeks, indicating a significant difference in age ($p<0.01$).

In a research by Haseli A et al.[63] 982 babies' medical records were examined. The first group showed greater rates of breastfeeding (90.35% compared 52.98%), jaundice (32.77% versus 29.08%), nulliparity (37.86% versus 22.49%), and cesarean

section (67.21% versus 18.96%) when comparing factors between infants who had lost more than 5% of their birth weight and other infants. Compared to baby formula (2.78 ± 1.65%), the mean WL of exclusively breastfed babies (4.89 ± 2.86%) was greater. The multivariate analysis (logistic regression) results indicated that the following risk factors were associated with NWL of more than 5%: breastfeeding, cesarean section, female gender, mother's inexperience with breastfeeding, weight over 4000 grams, and jaundice. On the other hand, the only risk factors associated with NWL of more than 7% were breastfeeding, cesarean section, and female gender. This is somewhat in line with Merry and Montgomery's findings, which showed that babies had lost, on average, 4% to 9.8% of their body weight.[74] According to a Marchini et al. study, babies lost three to six percent of their birth weight in the first four days after delivery.[75] According to Martinez et al.'s research, the average WL within three to five days following delivery was 5.09 ± 2.89.[76] Their study's conclusions are in line with those of the majority of earlier research, which revealed a mean NWL of 3% to 7%.[77-79]

In this study, after 72 hours of delivery, 63.6% (35/55) of the participants had severe hyperbilirubinemia of >12 mg/dl and had lost more than 7% of their body weight. Salas et al.[45] found that substantial weight reduction occurred in 38% of their patients. There was a considerably higher frequency of severe hyperbilirubinemia (> 20 mg/dL) in babies who had experienced significant weight loss. According to research by Boskabadi et al., babies with severe hyperbilirubinemia (>20 mg/dl) lose weight on average three times more than those with mild hyperbilirubinemia (< 20 mg/dl).[43] Significant hyperbilirubinemia developed in 23.6% of their patients, and Huang et al.[44] classified substantial hyperbilirubinemia as a bilirubin level of more than 15 mg/dL between days 4 and 10 of life.

At a median age of 4.7 days, breastfed term babies with hyperbilirubinemia were readmitted. Sixty-six percent (64.6%) of these newborns were male. At admission, the average TSB level was 18.6 ± 3.0 mg/dL, with a range of 15.1 to 31.3 mg/dL. There was a notable decrease in weight in thirty (38%) of the newborns with hyperbilirubinemia who had to be readmitted. Over ten percent of these individuals experienced weight loss. Compared to neonates with hyperbilirubinemia plus severe weight loss, infants with hyperbilirubinemia alone had substantially lower mean total serum binding behavior (TSB) levels (18.0 vs. 19.5 mg/dl; $p < 0.05$). A greater incidence of severe hyperbilirubinemia in babies was linked to significant weight loss (46.7% vs. 18.4%; $p < 0.05$). Babies who lost a significant amount of weight had a four-fold increased risk of having severe hyperbilirubinemia (OR: 3.9; 95% CI: 1.4-10.8; $p < 0.05$) compared to those who lost a tolerable amount of weight. More than 10% weight loss in infants put them at risk (OR: 4.2; 95% CI: 1.4-12.7; $p < 0.05$).

About 25% of neonates who were breastfed experienced weight loss of more than 5% during the first 24 hours of life.[80] Weight loss of more than 12% was observed in around one-third of nursing term newborns readmitted for hyperbilirubinemia (mean TSB level of 22.8 mg/dL).[81] Furthermore, compared to bottle-fed infants, breastfed babies with substantial hyperbilirubinemia (> 12.9 mg/dL) lost more weight at birth (6.9% vs. 4.2%).[4] After 72 hours of life, a higher rate of weight loss (8.0% vs. 6.4%) was likewise associated with significant hyperbilirubinemia.[8] When it comes to controlling serum bilirubin, fasting and low-calorie meals appear to be more effective than breastfeeding alone.[8,4] We discovered a statistically significant difference between babies with severe and moderate hyperbilirubinemia in the percentage of weight loss (8.8% vs. 5.9%, respectively). Moreover, our study revealed that a considerable degree of weight loss was

also seen by about 60% of newborns with severe hyperbilirubinemia who were readmitted.

In the current study, 58.8% of the patients were delivered by laparoscopic surgery, followed by vaginal birth in 28.2% of the subjects and instrumental delivery in 12.9% of the subjects. There is little and sometimes contradicting information available about the impact of vaginal or cesarean delivery on neonatal hyperbilirubinemia.[82–86] Birth type was found to have no bearing on the development of newborn hyperbilirubinemia in earlier research.[5] According to another study, cesarean sections decreased the chance that neonates will experience jaundice;[82-83] nonetheless, compared to cesarean sections, vaginal births had a lower risk factor for neonatal hyperbilirubinemia. Other reports state that infants delivered vaginally have a decreased risk of jaundice.[85–86]

A total of 170 infants with jaundice were included in the research by Ali Naghipour et al.[87] of whom 60.1% were males and 39.9% were girls. 39.8% of the patients were born vaginally, whereas 60.2% underwent a cesarean section. The method of delivery and hyperbilirubinemia (10-14.9 mg/dl) were significantly correlated (P = 0.01). In 41.4% of instances, the jaundice initially emerged between days 4 and 7 after birth, while in 25% of patients, it was noticed during the first 24 hours of life. 288 people in the research by Ozdemirci, S. et al.[88] met the requirements for inclusion while still in the newborn stage. There were 131 (22.6%) newborns with hyperbilirubinemia in the cesarean birth group compared to 157 (16.8%) in the vaginal delivery group. Hyperbilirubinemia was much more common in the group of newborns who underwent cesarean sections (p = 0.01).

Regarding the mode of delivery, 74 (27.5%) of the 269 neonates born by Lower Segment Caesarean Section (LSCS) exhibited hyperbilirubinaemia, according to research by Vijay Baburao Sonawane et al.[89] Another variable linked to NWL in the Haseli A et

al.[63] research was cesarean section. This is in line with research by Kagler[90] and Davanzo,[91] who found a link between weight loss during childbirth and cesarean sections. It has been demonstrated that the kind of delivery affects NWL independent of the newborn feeding method. Less than ideal nutrition following a C-section, according to researchers, may result in higher levels of NWL. In actuality, CS has the opposite effect on when and how long a mother breastfeeds, particularly after an emergency cesarean section. This is true even though "natural" CS is promoted and special breastfeeding education programs are offered. Lower breastfeeding rates may also be the result of a mother not fully recovering from surgery.[92]

49.4% of the participants in the current research were primipara and 50.6% of the subjects were multipara. In the Vijay Baburao Sonawane et al.[89] study, blood total bilirubin levels were higher in 95 out of 500 babies than the suggested acceptable limit (13 mg/dL at 72 hours of life). Thus, in their investigation, the prevalence of hyperbilirubinaemia was 19%. Forty (42.1%) of the ninety-five newborns with hyperbilirubinemia were born to primigravida women, and forty (47.3%) to multigravida mothers. Out of the 95 instances of newborn hyperbilirubinemia, 21 neonates (19.5%) were delivered vaginally normally, and 74 neonates (70.5%) were delivered via Lower Segment Caesarean Section (LSCS). The results of the study showed that there was no significant relationship (p-value=0.8) between the neonates' blood total bilirubin levels and the parity of the mother. Of the ninety-five newborns, forty (42.1%) were born to primigravida mothers, and fifty-five (57.9%) to multigravida moms.

The Haseli A et al[63] study did not find a significant correlation between WL and parity; nevertheless, WL was reported to be 5% to 7% in newborns whose mothers had never breastfed. Some study has indicated that the number of pregnancies has a contradictory effect as a predictor of NWL. WL and nulliparity are positively correlated,

notwithstanding Patricia et al.'s inability to detect any connection between the number of pregnancies and NWL.[93, 94] The number of pregnancies overall as well as the mother's nursing experience were evaluated in the current study. The findings suggested that a mother's capacity to nurse her child appears to be more important than the quantity of pregnancies in NWL in the initial postpartum period. Mothers who are unable to nurse their babies well during their first pregnancy may face difficulties in their subsequent pregnancies.

Of the trial participants, 74.1% had bilirubin levels that could be treated with phototherapy, and 25.9% did not have serum bilirubin levels in the phototherapy range. Zaitsu et al. found a weak connection (adjusted β 0.14; 95% CI 0.11–0.17) between bilirubin levels and maximal body-weight reduction.[95] Despite this, the correlation remained clinically negligible. Maximum body weight loss and total serum bilirubin levels ≥15 mg/dL were linked to an elevated likelihood of phototherapy treatment (adjusted RR 1.27; 95% CI 1.04–1.54), despite the low absolute cumulative incidence of baby jaundice and excess body weight loss. Indeed, at approximately 4% of the maximal bodyweight decrease, the risk of phototherapy and total blood bilirubin levels ≥15 mg/dL were statistically significant.

The average birth weight on the third day was 3119 ± 352 g, and the average BWL percentage was 7.07 ± 2.82%, citing Yang WC's research [42]. After a 72-hour period from delivery, the average TSB level was 13.39 ±3.12 mg/dL. Furthermore, 72 hours after birth, TSB levels were greater than 15 mg/dL in 115 neonates (33.5%). Particularly for the BWL percentages on days two and three, there was a strong connection ($p < 0.001$) between the BWL percentages during the first three days after birth and substantial hyperbilirubinemia 72 hours later. Following a 72-hour period, the group with non-significant hyperbilirubinemia had an average total serum binding (TSB)

level of 11.67 ± 2.21 mg/dl, whereas the group with substantial hyperbilirubinemia had an average level of 16.8 ± 1.36 mg/dl. There was no obvious correlation seen between significant hyperbilirubinemia and any specific feeding or delivery method seventy-two hours after birth.

Within 72 hours of delivery, there was a statistically significant correlation between the BWL percentages in the first three days postpartum and severe hyperbilirubinemia. Specifically, compared to days 1 and 2, the BWL% on day 3 was a more accurate predictor of severe hyperbilirubinemia 72 hours after birth. Furthermore, the BWL for the first three days appeared to be impacted by the BWL for the day before, irrespective of whether the baby was fed exclusively breastmilk or a combination of formula and milk. This shows that neonates with a high BWL% on day 1 can still be spared more serious hyperbilirubinemia if given the right care. Additionally, it highlights the significance of the BWL percentage on days two and three. The neonates whose BWL percentages were less than 0.4% on day 1, less than 2.9% on day 2, and less than 0.6% on day 3 did not exhibit significant hyperbilirubinemia 72 hours after birth. Babies with BWL percentages greater than 10.2% on day 1, more than 10.9% on day 2, and more than 11.3% on day 3 had significant hyperbilirubinemia 72 hours after birth. Moreover, it appears that 7.60% by day 2 and 8.15% by day 3 were the optimal cutoff values for predicting hyperbilirubinemia 72 hours following delivery. As of day 3, 8.4 ± 2.54% of the healthy, term babies with substantial hyperbilirubinemia had BWL percentage, whereas the non-significant hyperbilirubinemia group had 6.4 ± 2.73%.

This result is consistent with earlier research that found 25% of 874 newborns with hyperbilirubinemia had a BWL of 8.96 ± 1.99% and 33% of 86 neonates with hyperbilirubinemia had a severe BWL of greater than 10%.[65,66] The BWL percentage cutoff values, which were 7.60% by day 2, 8.15% by day 3, and 4.48% on day 1 were

found to be helpful in predicting severe hyperbilirubinemia 72 hours after delivery by the researchers.

K. S. Indriyani and others.[96] A linear regression analysis revealed a relationship between the total serum bilirubin level and the percentage of birth weight gained or lost on the seventh day after delivery (regression model y=65.12+0.59 X, p=0.007) and the third day after birth (regression model y=6.52+0.53 X, p<0.001). The results of a logistic regression study involving associated factors and hyperbilirubinemia indicated that the only significant link between the two was the percentage of birth weight decrease on the third day (OR 38(95% CI 2.29) p=0.011).

CONCLUSION

CONCLUSION

In our study, majority of the newborns had significant weight loss on day 4 of life, among which 70% of them developed significant hyperbilirubinemia indicating that significant weight loss increases the risk of hyperbilirubinemia.

The study revealed that significant weight loss is an important risk factor for development of hyperbilirubinemia in the newborn period.

Significant weight loss upon delivery may be a sign of newborn hyperbilirubinemia and a useful clinical signal indicating what has to be done to keep this curable illness from getting worse and causing life-threatening consequences.

However, the development of significant weight loss should be studied based on the feeding practices like exclusive breast feeding and formula milk feeding among a larger newborn population to further strengthen the objective of our study.

LIMITATIONS

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1) In our study, there was no inclusion of feeding practises like breast feeding and formula milk feeding to look for significant weight loss.
2) The percentage of development of weight loss among neonates varied with each day of life in different newborns like one newborn developed significant weight loss on day 2 of life whereas the other developed on day 4 of life.
3) In our study there was no equal distribution of sexuality among the babies to correlate with significant weight loss.
4) Daily serum bilirubin levels by blood sampling couldn't be done in our study.
5) It is a relatively smaller and hospital-based study, hence larger study is required to confirm the results.

SUMMARY

SUMMARY

- This Prospective Observational Study was conducted in RL Jalappa hospital from September 2022 to December 2023.

- 85 neonates satisfying the inclusion and exclusion criteria were included in the study.

- The study was conducted in the way as explained in the methodology. Birth weight and weights at 24, 48, 72, 96 and 120 hours of life were assessed. Total serum bilirubin was assessed on day of significant weight loss. Weight loss and bilirubin values were compared.

- 74.1% of the subjects had serum bilirubin in phototherapy range and 25.9% of the subjects didn't have serum bilirubin in phototherapy range.

- Majority of the neonates 56.5% had significant weight loss on day 4 followed by day 3 which was 32.9%, on day 2 it was 5.9% and on day 5 it was 4.7%.

- Among subjects who had Significant Weight loss on day 3, 85.7% of them had significant hyperbilirubinemia.

- Among subjects who had Significant Weight loss on day 4, 70.8% of them had significant hyperbilirubinemia

- Among subjects delivered through LSCS significant hyperbilirubinemia was found in 86.0%. Among subjects delivered through vaginally significant hyperbilirubinemia was found in 41.7%. Among subjects delivered through Instrumental significant hyperbilirubinemia was found in 90.9%. P Value <0.001, a statistically significant difference regarding the mode of delivery was discovered between the groups.

- The study revealed that significant weight loss is an important risk factor for development of hyperbilirubinemia in the newborn period.

- Significant weight loss upon delivery may be a sign of newborn hyperbilirubinemia and a useful clinical signal indicating what has to be done to keep this curable illness from getting worse and causing life-threatening consequences.

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ANNEXURE

ANNEXURE -A

PROFORMA

Name : Date of Birth :

Gestational age :

Sex :

Informant :

Birth weight :

Address:……..Telephone No……

Mother Blood Group :Baby Blood Group :

Time	Weight	Wt loss %	TCB
At birth			
Day 1 (24hrs)			
Day 2 (48hrs)			
Day 3 (72hrs)			
Day 4 (96hrs)			
Day 5 (120hrs)			

Total serum bilirubin level :

Sample collected at_____hrs of life. Threshold value according to AAP nomogram -Whether in phototherapy range - Yes / No

ANNEXURE-B

INFORMED CONSENT FORM

Date:

I, Mr/Mrs_____,have been explained in my own vernacular language that my child will be included in **A PROSPECTIVE STUDY TO DETERMINE ASSOCIATION BETWEEN WEIGHT LOSS AND NEONATAL HYPERBILIRUBINEMIA IN NEONATES,** 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 bore 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 & Name of Pt. Attendant) (Signature/Thumb impression &

Name of Patient/Guardian)

(Relation with patient)Witness:

_____

(Signature & Name of Researchperson/doctor)

ಮಾಹಿತಿಯುತಸಮ್ಮತಿನಮೂನೆ

ದಿನಾಂಕ :

ನಾನು,

_____,

"ನೀನೇಟ್‌ನಲ್ಲಿ ತೂಕ ನಷ್ಟ ಮತ್ತು ನವಜಾತ ಶಿಶುವಿನ ಹೈಪರ್ಬಿಲಿರುಬಿನೆಮಿಯಾ ನಡುವಿನ ಸಂಬಂಧವನ್ನು ನಿರ್ಧರಿಸಲು ಒಂದು ಪ್ರಾಸ್ಪೆಕ್ಟಿವ್ ಸ್ಟಡಿ ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇನೆ"
ಹೆಮಟೊಲಾಜಿಕಲ್ಮತ್ತುಕ್ಲಿನಿಕಲ್ನಿಯತಾಂಕಗಳಅವಲೋಕನಗಳನ್ನುದಾಖಲಿಸಲುನಾನುಯಾವುದೇಬಲಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆನನ್ನಮಾನ್ಯಲಿಖಿತತಿಳುವಳಿಕೆಯನ್ನುನೀಡುತ್ತೇನೆ.
ಒಳಗೊಂಡಿರುವಸ್ವಭಾವಮತ್ತುಅಪಾಯಗಳನ್ನುನನಗೆವಿವರಿಸಲಾಗಿದೆ, ನನ್ನತೃಪ್ತಿ.
ನಡೆಸುತ್ತಿರುವಅಧ್ಯಯನದಬಗ್ಗೆನನಗೆವಿವರವಾಗಿವಿವರಿಸಲಾಗಿದೆ.
ನಾನುರೋಗಿಯಮಾಹಿತಿಹಾಳೆಯನ್ನುಓದಿದ್ದೇನೆಮತ್ತುಯಾವುದೇಪ್ರಶ್ನೆಯನ್ನುಕೇಳಲುನನಗೆಅವಕಾಶವಿದೆ.
ನಾನುಕೇಳಿದಯಾವುದೇಪ್ರಶ್ನೆಗೆನನ್ನತೃಪ್ತಿಗೆಉತ್ತರಿಸಲಾಗಿದೆ
.ನನ್ನಮಗುವನ್ನುಈಸಂಶೋಧನೆಯಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವಂತೆಅನುಮತಿಸಲುನಾನುಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದಒಪ್ಪಿಗೆಯನ್ನುನೀಡುತ್ತೇನೆ. ನಾನುಇತಿಹಾಸವನ್ನುಒದಗಿಸಲು, ದೈಹಿಕಪರೀಕ್ಷೆಗೆಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆಒಳಗಾಗಲು,
ತನಿಖೆಗೆಒಳಗಾಗಲುಮತ್ತುಅದರಫಲಿತಾಂಶಗಳುಮತ್ತುದಾಖಲೆಗಳನ್ನುಇತ್ಯಾದಿಗಳನ್ನುವೈದ್ಯರು / ಸಂಸ್ಥೆಇತ್ಯಾದಿಗಳಿಗೆಒದಗಿಸಲುನಾನುಈಮೂಲಕಒಪ್ಪಿಗೆನೀಡುತ್ತೇನೆ.
ಶೈಕ್ಷಣಿಕಮತ್ತುವೈಜ್ಞಾನಿಕಉದ್ದೇಶಕ್ಕಾಗಿಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ,ಅಧ್ಯಯನಕ್ಕೆಅಗತ್ಯವಿರುವಎಲ್ಲಾವೆಚ್ಚಗಳನ್ನುಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿವಹಿಸುತ್ತಾರೆ.ಇತ್ಯಾದಿಗಳನ್ನುವೀಡಿಯೊಗ್ರಾಫ್ಅಥವಾಛಾಯಾಚಿತ್ರಮಾಡಬಹುದು.
ಎಲ್ಲಾಡೇಟಾವನ್ನುಪ್ರಕಟಿಸಬಹುದುಅಥವಾಯಾವುದೇಶೈಕ್ಷಣಿಕಉದ್ದೇಶಕ್ಕಾಗಿಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದಸಮಯದಲ್ಲಿಯಾವುದೇಅಹಿತಕರಪರಿಣಾಮಗಳಿಗೆನಾನುವೈದ್ಯರು / ಸಂಸ್ಥೆಇತ್ಯಾದಿಗಳನ್ನುಜವಾಬ್ದಾರರನ್ನಾಗಿಮಾಡುವುದಿಲ್ಲ.

_____ _____

(ರೋಗಿಯಪರಿಚಾರಕನಸಹಿಮತ್ತುಹೆಸರು) (ಸಹಿ/ಹೆಬ್ಬೆರಳಿನಗುರುತು& ರೋಗಿಯ/ರಕ್ಷಕನಹೆಸರು)

(ರೋಗಿಯೊಂದಿಗೆಸಂಬಂಧ)

ಸಾಕ್ಷಿ :

_____

(ಸಂಶೋಧನಾವ್ಯಕ್ತಿ/ವೈದ್ಯರಸಹಿಮತ್ತುಹೆಸರು)

ANNEXURE-C

PATIENT INFORMATION SHEET

Principal investigator: Dr JAHNAVI

I Dr. JAHNAVI, Post graduate student in Department at Sri Devaraj Urs Medical College, will be conducting a study titled **"A PROSPECTIVE STUDY TO DETERMINE ASSOCIATION BETWEEN WEIGHT LOSS AND NEONATAL HYPERBILIRUBINEMIA IN NEONATES**

" 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 bore 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 careat this institution.

Name and Signature of the Principal InvestigatorContact number : 9010047418

Date-

ರೋಗಿಯಮಾಹಿತಿಹಾಳೆ

ಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿ: ಡಾಜಾಹ್ನವಿ

ನಾನುಡಾ.ಜಾಹ್ನವಿ, ಶ್ರೀದೇವರಾಜ್ಅಸ್ಪೈರ್ಯಕೀಯಕಾಲೇಜಿನಲ್ಲಿವಿಭಾಗದಸ್ನಾತಕೋತ್ತರವಿದ್ಯಾರ್ಥಿನಿ, ಎಂಬಶೀರ್ಷಿಕೆಯಅಧ್ಯಯನವನ್ನುನಡೆಸಲಿದ್ದೇನೆ

"ನೀನೇಟ್‌ನಲ್ಲಿ ತೂಕ ನಷ್ಟ ಮತ್ತು ನವಜಾತ ಶಿಶುವಿನ ಹೈಪರ್ಬಿಲಿರುಬಿನೆಮಿಯಾ ನಡುವಿನ ಸಂಬಂಧವನ್ನು ನಿರ್ಧರಿಸಲು ಒಂದು ಪ್ರಾಸ್ಪೆಕ್ಟಿವ್ ಸ್ಟಡಿ ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇನೆ"

ನನ್ನಪ್ರಬಂಧಕ್ಕಾಗಿಮಾರ್ಗದರ್ಶನದಲ್ಲಿಡಾ. ಕೃಷ್ಣಪ್ಪ. ಜೆ, ಪೀಡಿಯಾಟ್ರಿಕ್ಸ್ವಿಭಾಗದಪ್ರಾಧ್ಯಾಪಕ.

ಈಸಂಶೋಧನಾಯೋಜನೆಯಲ್ಲಿನಿಮ್ಮಮಗುವಿನಭಾಗವಹಿಸುವಿಕೆಗಾಗಿನಿಮಗೆಯಾವುದೇಹಣಕಾಸಿನಪರಿಹಾರವನ್ನುಪಾವತಿಸಲಾಗುವುದಿಲ್ಲ.ಅಧ್ಯಯನಕ್ಕೆಅಗತ್ಯವಿರುವಎಲ್ಲಾವೆಚ್ಚಗಳನ್ನುಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿವಹಿಸುತ್ತಾರೆ.

ಎಲ್ಲಾಡೇಟಾವನ್ನುಗೌಪ್ಯವಾಗಿಇರಿಸಲಾಗುತ್ತದೆಮತ್ತುಈಸಂಸ್ಥೆಯಿಂದಸಂಶೋಧನಾಉದ್ದೇಶಕ್ಕಾಗಿಮಾತ್ರಬಳಸಲಾಗುತ್ತದೆ. ಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಮಗುವಿನಭಾಗವಹಿಸುವಿಕೆಗೆಒಪ್ಪಿಗೆನೀಡಲುನೀವುಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ಯಾವುದೇಕಾರಣಗಳನ್ನುನೀಡದೆನೀವುಯಾವುದೇಸಮಯದಲ್ಲಿನಿಮ್ಮಮಗುವನ್ನುಅಧ್ಯಯನದಿಂದಹಿಂಪಡೆಯಬಹುದು.

ಭಾಗವಹಿಸಲುನಿಮ್ಮನಿರಾಕರಣೆಯುಈಸಂಸ್ಥೆಯಲ್ಲಿಯಾವುದೇಪ್ರಸ್ತುತಅಥವಾಭವಿಷ್ಯದಕಾಳಜಿಗೆನಿಮ್ಮನ್ನುಪೂರ್ವಾಗ್ರಹಮಾಡುವುದಿಲ್ಲ.

ಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿಯಹೆಸರುಮತ್ತುಸಹಿ

ಸಂಪರ್ಕಸಂಖ್ಯೆ : 9010047418

ದಿನಾಂಕ

MASTER CHART

SL NO	GESTATIONAL AGE	GRAVIDA	MODE OF DELIVERY	SEX	MOTHER BGT	BABY BGT	BIRTH WEIGHT	DAY 1 WEIGHT	DAY 1 WEIGHT LOSS PERCENTAGE	DAY 1 TCB	DAY 2 WEIGHT	DAY 2 WEIGHT LOSS PERCENTAGE	DAY 2 TCB	DAY 3 WEIGHT	DAY 3 WEIGHT LOSS PERCENTAGE	DAY 3 TCB	DAY 4 WEIGHT	DAY 4 WEIGHT LOSS PERCENTAGE	DAY 4 TCB	DAY 5 WEIGHT	DAY 5 WEIGHT LOSS PERCENTAGE	DAY 5 TCB	DAY OF SAMPLE COLLECTION	SERUM BILIRUBIN	THRESHOLD VALUE ACCORDING TO AAP NOMOGRAM	WHETHER IN PHOTOTHERAPY RANGE
1	37+2	MULTI	NVD	F	B POSITIVE	A POSITIVE	2.58	2.52	2.3	4	2.4	6.9	9	2.32	10	14	2.3	10.8	16	2.32	10	11	4	16	20	NO
2	38+1	PRIMI	NVD	F	B POSITIVE	B POSITIVE	2.5	2.42	3.2	3	2.4	4	6	2.36	5.6	9	2.3	8	14	2.3	8	11	4	15	20.8	NO
3	39+3	PRIMI	NVD	F	O POSITIVE	O POSITIVE	3.22	3.04	5.4	4	3	6.8	9	2.9	9.9	15	2.92	9.3	11	2.96	8	8	3	14.4	19.5	NO
4	37+2	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	2.52	2.46	2.3	4	2.34	7.1	11	2.26	10.3	19	2.24	11.1	15	2.26	10.3	12	3	18.4	18	YES
5	38+1	MULTI	LSCS	F	A POSITIVE	A POSITIVE	3	2.82	6	6	2.7	10	10	2.66	11.3	18	2.7	10	12	2.74	8.6	7	3	19.2	18.8	YES
6	38	PRIMI	LSCS	M	O POSITIVE	B POSITIVE	3.06	2.94	3.9	4	2.8	8.4	10	2.74	10.4	15	2.7	11.7	19	2.74	10.4	14	4	21	20.8	YES
7	37+1	MULTI	LSCS	M	A POSITIVE	A POSITIVE	2.82	2.76	2.1	3	2.64	6.3	9	2.56	9.2	13	2.54	9.9	19	2.56	9.2	13	4	20	20	YES
8	37+3	PRIMI	LSCS	M	B POSITIVE	B POSITIVE	2.52	2.46	2.3	4	2.34	7.1	11	2.26	10.3	19	2.24	11.1	15	2.26	10.3	11	3	18.6	18	YES
9	38	PRIMI	INSTRUMENTAL	M	A POSITIVE	B POSITIVE	3.4	3.28	3.6	4	3.14	7.6	7	3.08	9.4	13	3.02	11.1	18	3.08	9.4	12	4	21	20.8	YES
10	37+4	MULTI	LSCS	M	B POSITIVE	O POSITIVE	3.18	3.06	3.7	5	2.9	8.8	10	2.84	10.6	18	2.8	11.9	14	2.84	10.6	12	3	19	18	YES
11	37	PRIMI	NVD	M	A POSITIVE	A POSITIVE	2.52	2.46	2.3	4	2.34	7.1	11	2.26	10.3	19	2.24	11.1	15	2.26	10.3	11	3	18.8	18	YES
12	38+2	PRIMI	LSCS	M	O POSITIVE	A POSITIVE	3.06	2.94	3.9	4	2.8	8.4	10	2.74	10.4	15	2.7	11.7	19	2.74	10.4	14	4	21	20.8	YES
13	39+1	PRIMI	INSTRUMENTAL	F	O POSITIVE	B POSITIVE	3.16	3.04	3.7	5	2.9	8.2	10	2.84	10.1	14	2.8	11.3	20	2.84	10.1	15	4	22	21.5	YES
14	37+3	PRIMI	LSCS	F	B POSITIVE	A POSITIVE	2.62	2.56	2.2	4	2.44	6.8	7	2.36	9.9	13	2.34	10.6	18	2.36	9.9	12	4	20.6	20	YES
15	37+6	PRIMI	LSCS	M	A POSITIVE	O POSITIVE	3.4	3.3	2.9	4	3.14	7.6	7	3.08	9.4	13	3.02	11.1	18	3.08	9.4	12	4	21.2	20.8	YES
16	37+3	PRIMI	LSCS	F	O POSITIVE	B POSITIVE	2.62	2.56	2.2	4	2.44	6.8	7	2.36	9.9	13	2.34	10.6	18	2.36	9.9	12	4	20.8	20	YES
17	37+3	PRIMI	LSCS	F	B POSITIVE	A POSITIVE	2.62	2.56	2.2	4	2.44	6.8	7	2.36	9.9	13	2.34	10.6	18	2.36	9.9	12	4	20.4	20	YES
18	39+2	PRIMI	INSTRUMENTAL	M	O POSITIVE	A POSITIVE	2.94	2.84	3.4	8	2.7	8.1	18	2.64	10.2	15	2.64	10.2	10	2.7	8.1	7	2	17	16.5	YES
19	38+2	MULTI	LSCS	M	O POSITIVE	O POSITIVE	2.84	2.74	3.5	3	2.66	6.3	7	2.6	9.1	12	2.52	11.2	17	2.6	9.1	10	4	21.2	20.8	YES
20	38+2	MULTI	LSCS	M	O POSITIVE	O POSITIVE	2.88	2.78	3.4	4	2.66	7.6	9	2.6	9.7	14	2.54	11.8	19	2.6	9.7	12	4	22	20.8	YES
21	38+5	MULTI	NVD	F	O POSITIVE	B POSITIVE	2.78	2.6	6.4	5	2.5	10	10	2.48	10.7	17	2.48	10.7	11	2.5	10	5	3	20.4	19.2	YES
22	38	MULTI	NVD	M	O POSITIVE	O POSITIVE	2.3	2.12	7.8	5	2.1	8.2	12	2.02	12	19	2.02	12	10	2.1	8.2	6	3	19.4	18.8	YES
23	37	MULTI	LSCS	M	A POSITIVE	O POSITIVE	2.82	2.76	2.1	4	2.64	6.3	8	2.56	9.2	13	2.54	9.9	19	2.56	9.2	11	4	20.2	20	YES
24	39+3	PRIMI	LSCS	M	O POSITIVE	A POSITIVE	2.94	2.84	3.4	6	2.7	8.1	18	2.64	10.2	15	2.64	10.2	10	2.7	8.1	7	2	17	16.5	YES
25	37+3	MULTI	NVD	F	A POSITIVE	B POSITIVE	2.58	2.38	7.7	5	2.32	10	10	2.3	10.8	17	2.32	10	11	2.36	8.5	6	3	19.4	18.5	YES
26	42+2	MULTI	LSCS	M	AB POSITIVE	B POSITIVE	3.12	3.04	2.5	6	2.98	4.4	10	2.86	8.3	20	2.84	8.9	18	2.86	8.3	14	3	20	19.8	YES
27	37+2	PRIMI	LSCS	M	O POSITIVE	O POSITIVE	2.52	2.46	2.3	4	2.34	7.1	11	2.26	10.3	19	2.24	11.1	12	2.26	10.3	8	3	18.4	18	YES
28	37+1	MULTI	NVD	M	B POSITIVE	O POSITIVE	2.82	2.76	2.1	4	2.64	6.3	9	2.56	9.2	13	2.54	9.9	19	2.56	9.2	12	4	20.4	20	YES
29	42+2	MULTI	LSCS	M	AB POSITIVE	B POSITIVE	3.12	3.04	2.5	7	2.98	4.4	10	2.86	8.3	20	2.84	8.9	16	2.86	8.3	11	3	20	19.8	YES
30	39+4	PRIMI	NVD	F	O POSITIVE	O POSITIVE	2.7	2.54	5.9	4	2.5	7.4	7	2.48	9.2	10	2.42	10.3	12	2.48	9.2	9	4	12	21.7	NO
31	38	PRIMI	LSCS	M	A POSITIVE	O POSITIVE	3.4	3.28	3.6	4	3.14	7.6	7	3.08	9.4	13	3.02	11.1	18	3.08	9.4	12	4	21.4	20.8	YES
32	39	PRIMI	NVD	F	B POSITIVE	O POSITIVE	3.16	3.04	3.7	5	2.9	8.2	10	2.84	10.1	14	2.8	11.3	20	2.84	10.1	15	4	22	21.5	YES
33	39+1	PRIMI	LSCS	F	O POSITIVE	A POSITIVE	3.16	3.04	3.7	5	2.9	8.2	10	2.84	10.1	14	2.8	11.3	20	2.84	10.1	15	4	22	21.5	YES
34	37+5	MULTI	INSTRUMENTAL	M	B POSITIVE	O POSITIVE	3.18	3.06	7	5	2.9	8.8	10	2.84	10.6	18	2.8	11.9	14	2.84	10.6	12	3	20	18	YES
35	37+3	MULTI	INSTRUMENTAL	M	O POSITIVE	A POSITIVE	3.5	3.4	2.8	3	3.34	4.5	7	3.26	6.8	12	3.16	9.7	18	3.2	8.5	11	4	21.4	20.4	YES
36	38+5	MULTI	LSCS	M	O POSITIVE	A POSITIVE	3.46	3.34	3.4	4	3.2	7.5	10	3.14	9.2	13	3.1	10.4	19	3.14	9.2	12	4	21.6	20.8	YES

SL NO	GESTATIONAL AGE	GRAVIDA	MODE OF DELIVERY	SEX	MOTHER BGT	BABY BGT	BIRTH WEIGHT	DAY 1 WEIGHT	DAY 1 WEIGHT LOSS PERCENTAGE	DAY 1 TCB	DAY 2 WEIGHT	DAY 2 WEIGHT LOSS PERCENTAGE	DAY 2 TCB	DAY 3 WEIGHT	DAY 3 WEIGHT LOSS PERCENTAGE	DAY 3 TCB	DAY 4 WEIGHT	DAY 4 WEIGHT LOSS PERCENTAGE	DAY 4 TCB	DAY 5 WEIGHT	DAY 5 WEIGHT LOSS PERCENTAGE	DAY 5 TCB	DAY OF SAMPLE COLLECTION	SERUM BILIRUBIN	THRESHOLD VALUE ACCORDING TO AAP NOMOGRAM	WHETHER IN PHOTOTHERAPY RANGE
37	39+2	PRIMI	LSCS	M	O POSITIVE	B POSITIVE	2.94	2.84	3.4	8	2.7	8.1	18	2.64	10.2	15	2.64	10.2	10	2.7	8.1	7	2	17	16.5	YES
38	42+3	MULTI	LSCS	M	AB POSITIVE	B POSITIVE	3.12	3.04	2.5	7	2.98	4.4	10	2.86	8.3	20	2.84	8.9	16	2.86	8.3	10	3	20	19.8	YES
39	38+1	PRIMI	LSCS	M	O POSITIVE	A POSITIVE	3.06	2.94	3.9	4	2.8	8.4	10	2.74	10.4	15	2.7	11.7	19	2.74	10.4	13	4	21.2	20.8	YES
40	37+4	MULTI	LSCS	M	B POSITIVE	O POSITIVE	3.18	3.06	3.7	5	2.9	8.8	10	2.84	10.6	18	2.8	11.9	14	2.84	10.6	12	3	18.8	18	YES
41	40	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	3.62	3.52	2.7	4	3.34	7	9	3.3	8	14	3.24	10.4	19	3.24	10.4	13	4	22	21.8	YES
42	38+5	MULTI	NVD	F	A POSITIVE	A POSITIVE	2.48	2.44	1.6	4	2.4	3	7	2.34	5.6	10	2.3	7.2	12	2.26	8.8	16	5	14	21	NO
43	38	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	3.14	3	4.6	6	2.82	10.1	10	2.8	10.8	18	2.82	10	12	2.86	8.9	7	3	19.4	18.8	YES
44	39+2	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	2.94	2.84	3.4	8	2.7	8.1	18	2.64	10.2	15	2.64	10.2	10	2.7	8.1	7	2	17	16.5	YES
45	39+2	MULTI	LSCS	F	O POSITIVE	O POSITIVE	2.52	2.34	7.1	4	2.26	10.3	9	2.2	12.6	15	2.22	11.9	11	2.26	10.3	8	3	15	19.5	NO
46	37+3	PRIMI	LSCS	F	O POSITIVE	A POSITIVE	3.3	3.14	4.8	4	3.1	6	10	3	9	2	2.9	12	18	3	9	12	4	22	20	YES
47	38+5	MULTI	NVD	M	O POSITIVE	B POSITIVE	3.46	3.34	3.4	4	3.2	7.5	10	3.14	9.2	13	3.1	10.4	19	3.14	9.2	12	4	21	20.8	YES
48	37+3	PRIMI	LSCS	F	B POSITIVE	B POSITIVE	2.62	2.56	2.2	4	2.44	6.8	7	2.36	9.9	13	2.34	10.6	18	2.36	9.9	12	4	20.6	20	YES
49	39+4	PRIMI	LSCS	M	O POSITIVE	O POSITIVE	4.66	4.54	2.5	3	4.44	4.7	6	4.36	6.4	9	4.2	9.8	10	4.26	8.5	3	4	12	21.7	NO
50	37+2	MULTI	NVD	F	AB POSITIVE	A POSITIVE	2.58	2.52	2.3	4	2.4	6.9	9	2.32	10	14	2.3	10.8	16	2.32	10	11	4	17	20	NO
51	38+4	MULTI	LSCS	M	O POSITIVE	B POSITIVE	2.9	2.7	6.8	5	2.62	9.6	10	2.6	10.3	17	2.6	10.3	11	2.62	9.6	5	3	21	19.2	YES
52	38+2	MULTI	NVD	M	O POSITIVE	O POSITIVE	2.9	2.72	6.2	5	2.66	8.2	12	2.6	10.3	19	2.66	8.2	10	2.68	7.5	6	3	19	18.8	YES
53	37+2	PRIMI	LSCS	F	O POSITIVE	A POSITIVE	2.5	2.34	6.4	6	2.2	12	20	2.2	12	18	2.16	13.6	10	2.2	12	7	2	21.2	15	YES
54	39+5	PRIMI	LSCS	M	O POSITIVE	O POSITIVE	2.7	2.54	5.9	4	2.5	7.4	7	2.48	9.2	10	2.42	10.3	12	2.48	9.2	9	4	12	21.7	NO
55	40+2	MULTI	INSTRUMENTAL	F	B POSITIVE	B POSITIVE	3.5	3.42	2.28	3	3.38	3.42	6	3.3	5.7	10	3.2	8.5	16	3.2	8.5	11	4	15	21.8	NO
56	38+1	PRIMI	INSTRUMENTAL	F	O POSITIVE	O POSITIVE	2.9	2.72	6.2	5	2.66	8.2	12	2.6	10.3	19	2.66	8.2	10	2.68	7.5	6	3	19	18.8	YES
57	37+4	MULTI	LSCS	M	B POSITIVE	O POSITIVE	3.18	3.06	3.7	5	2.9	8.8	10	2.84	10.6	18	2.8	11.9	13	2.84	10.6	11	3	19	18	YES
58	40	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	3.62	3.52	2.7	4	3.34	7	9	3.3	8	14	3.24	10.4	19	3.24	10.4	13	4	22	21.8	YES
59	39	MULTI	LSCS	F	O POSITIVE	O POSITIVE	3.22	3.04	5.4	4	3	6.8	9	2.9	9.9	15	2.92	9.3	11	2.96	8	8	3	14.4	19.5	NO
60	42+3	MULTI	INSTRUMENTAL	M	AB POSITIVE	B POSITIVE	3.12	3.04	2.5	5	2.98	4.4	10	2.86	8.3	20	2.84	8.9	16	2.86	8.3	11	3	20	19.8	YES
61	38+2	MULTI	LSCS	F	O POSITIVE	O POSITIVE	2.3	2.12	7.8	5	2.1	8.2	12	2.02	12	19	2.02	12	10	2.1	8.2	6	3	19.4	18.8	YES
62	37+2	MULTI	NVD	F	AB POSITIVE	B POSITIVE	2.58	2.52	2.3	4	2.4	6.9	9	2.32	10	14	2.3	10.8	16	2.32	10	11	4	17	20	NO
63	37	MULTI	LSCS	M	B POSITIVE	O POSITIVE	2.82	2.76	2.1	4	2.64	6.3	9	2.56	9.2	13	2.54	9.9	19	2.56	9.2	13	4	20	20	YES
64	38	PRIMI	LSCS	M	O POSITIVE	A POSITIVE	3.06	2.96	3.2	4	2.8	8.4	10	2.74	10.4	15	2.7	11.7	19	2.74	10.4	14	4	21.2	20.8	YES
65	40+1	PRIMI	INSTRUMENTAL	M	A POSITIVE	A POSITIVE	3.62	3.52	2.7	4	3.34	7	9	3.3	8	14	3.24	10.4	19	3.34	10.4	13	4	22	21.8	YES
66	39+1	PRIMI	LSCS	F	O POSITIVE	A POSITIVE	3.16	3.04	3.7	5	2.9	8.2	10	2.84	10.1	14	2.8	11.3	20	2.84	10.1	15	4	22	21.5	YES
67	37+4	MULTI	INSTRUMENTAL	M	O POSITIVE	A POSITIVE	3.38	3.24	4.1	3	3.18	5.9	7	3.1	8.2	12	3.04	10	18	3.1	8.2	11	4	21	20.4	YES
68	37+5	MULTI	NVD	F	A POSITIVE	B POSITIVE	2.6	2.44	6.1	5	2.34	10	10	2.3	11.5	17	2.34	10	11	2.38	8.4	6	3	19.2	18.5	YES
69	38	PRIMI	LSCS	M	A POSITIVE	A POSITIVE	3	2.82	6	6	2.7	10	10	2.66	11.3	18	2.7	10	12	2.74	8.6	7	3	19.2	18.8	YES
70	38+5	MULTI	INSTRUMENTAL	M	O POSITIVE	B POSITIVE	3.46	3.34	3.4	4	3.2	7.5	10	3.14	9.2	13	3.1	10.4	19	3.14	9.2	12	4	21.6	20.8	YES
71	38+1	PRIMI	NVD	F	O POSITIVE	B POSITIVE	2.5	2.42	3.2	4	2.4	4	6	2.36	5.6	9	2.3	8	16	2.3	8	13	4	14	20.8	NO
72	39+1	PRIMI	NVD	M	O POSITIVE	O POSITIVE	2.52	2.34	7.1	4	2.26	10.3	9	2.2	12.6	15	2.22	11.9	11	2.26	10.3	8	3	14	19.5	NO

SL NO	GESTATIONAL AGE	GRAVIDA	MODE OF DELIVERY	SEX	MOTHER BGT	BABY BGT	BIRTH WEIGHT	DAY 1 WEIGHT	DAY 1 WEIGHT LOSS PERCENTAGE	DAY 1 TCB	DAY 2 WEIGHT	DAY 2 WEIGHT LOSS PERCENTAGE	DAY 2 TCB	DAY 3 WEIGHT	DAY 3 WEIGHT LOSS PERCENTAGE	DAY 3 TCB	DAY 4 WEIGHT	DAY 4 WEIGHT LOSS PERCENTAGE	DAY 4 TCB	DAY 5 WEIGHT	DAY 5 WEIGHT LOSS PERCENTAGE	DAY 5 TCB	DAY OF SAMPLE COLLECTION	SERUM BILIRUBIN	THRESHOLD VALUE ACCORDING TO AAP NOMOGRAM	WHETHER IN PHOTOTHERAPY RANGE
73	38+5	MULTI	LSCS	M	O POSITIVE	B POSITIVE	2.78	2.6	6.4	5	2.5	10	10	2.48	10.7	17	2.48	10.7	11	2.5	10	5	3	20.4	19.2	YES
74	40	MULTI	NVD	F	B POSITIVE	B POSITIVE	3.5	3.42	2.2	3	3.38	3.42	6	3.3	5.7	10	3.2	8.5	16	3.2	8.5	11	4	15	21.8	NO
75	40+2	MULTI	NVD	F	A POSITIVE	A POSITIVE	3.5	3.42	2.28	3	3.38	3.42	6	3.3	5.7	10	3.2	8.5	16	3.2	8.5	11	4	15	21.8	NO
76	38+5	MULTI	NVD	F	A POSITIVE	A POSITIVE	2.48	2.44	1.6	3	2.4	3	7	2.34	5.6	10	2.3	7.2	12	2.26	8.8	16	5	16	21	NO
77	38+5	MULTI	LSCS	M	O POSITIVE	B POSITIVE	3.46	3.34	3.4	4	3.2	7.5	10	3.14	9.2	13	3.1	10.4	19	3.14	9.2	12	4	21.4	20.8	YES
78	38+4	MULTI	NVD	F	B POSITIVE	B POSITIVE	2.48	2.44	1.6	3	2.4	3	7	2.34	5.6	10	2.3	7.2	12	2.24	9.6	16	5	14	21	NO
79	40+1	PRIMI	LSCS	M	A POSITIVE	B POSITIVE	3.62	3.52	2.7	4	3.34	7	9	3.3	8	14	3.24	10	19	3.24	10.4	13	4	22	21.8	YES
80	38+2	PRIMI	LSCS	F	B POSITIVE	B POSITIVE	2.5	2.42	3.2	3	2.4	4	6	2.36	5.6	9	2.3	8	14	2.3	8	11	4	15	20.8	NO
81	38+3	MULTI	LSCS	F	A POSITIVE	A POSITIVE	2.48	2.44	1.6	3	2.4	3	7	2.34	5.6	10	2.3	7.2	12	2.26	8.8	16	5	14	21	NO
82	38+1	PRIMI	NVD	F	B POSITIVE	O POSITIVE	2.5	2.42	3.2	4	2.4	4	6	2.36	5.6	9	2.3	8	15	2.3	8	11	4	14	20.8	YES
83	38	PRIMI	LSCS	M	A POSITIVE	O POSITIVE	3.4	3.28	3.6	4	3.14	7.6	7	3.08	9.4	13	3.02	11.1	18	3.08	9.4	12	4	21	20.8	YES
84	37+3	MULTI	LSCS	F	B POSITIVE	B POSITIVE	2.78	2.72	2	3	2.6	6.4	8	2.52	9.3	12	2.5	10	14	2.52	9.3	10	4	15	20	NO
85	39+2	MULTI	NVD	F	A POSITIVE	B POSITIVE	3.5	3.42	2.28	3	3.38	3.42	6	3.3	5.7	10	3.2	8.5	16	3.2	8.5	11	4	15	21.5	NO