"PREVALENCE OF PHOTOTHERAPY INDUCED HYPOMAGNESEMIA IN TERM NEWBORNS WITH JAUNDICE"

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LIST OF ABBREVIATIONS

BERA	-	Brainstem Evoked Auditory Response
NMR	-	Nuclear Magnetic Resonance
G6PD	-	Glucose 6 phosphate deficiency
ICT	-	Indirect Coombs Test
DCT	-	Direct coomb test
NNF	-	National Neonatology Forum
PT	-	Phototherapy
PDA	-	Patent Ductus Arteriosus
MAP	-	Mean Arterial Pressure
DNA	-	Deoxyribonucleic Acid
NMDA	-	N- methyl –D-Aspartate
NVD	-	Normal Vaginal Delivery
LSCS	-	Lower segment Cesarian section
GA	-	Gestational age
RBC	-	Red blood cell
GIT	-	Gastro intestinal tract
CNS	-	Central Nervous System

ABSTRACT

Background and objectives

Jaundice is more prevalent during neonatal period and is found in around 60% of term neonates and 80% of preterm neonates. In early neonatal period readmission to hospital for neonatal jaundice is seen in around 6.5% of the babies. Phototherapy a commonly used and comparatively safer modality of treatment used to treat neonatal jaundice. Phototherapy can lead to few side effects like electrolyte imbalance like hypocalcaemia, hyponatremia, hypokalemia, dehydration, hyperthermia, feed intolerance, diarrhea and retinal damage. The main aim of this study is to know the correlation between serum magnesium in newborn with jaundice at initiation and after ending phototherapy to know prevalence of hypomagnesemia and to know the association of hypomagnesemia with duration of photherapy.

Materials and methods

One year observational study was conducted from September 2020 to September 2021 in the Department of Paediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 73 term newborns with hyperbilirubinemia requiring phototherapy were studied. Maternal and stillbirth data was collected in a structured proforma. Serum bilirubin level and magnesium level will be sent before starting phototherapy, 12 hour, 24hours and after stopping phototherapy. Serum calcium levels checked at admission.

Results

During the study period a total of 73 full term neonates with neonatal jaundice were enrolled. In our study 58.9% newborns were males and 41.1% were females, highest number of newborns were noted in the gestational age group of 38-39 weeeks

i.e 58.9% and lowest in 40-41 weeks i.e 19.1%. About 43.8% were born by normal vaginal delivery and 56.2% by LSCS. The study highlights that the calcium value at the time of admission among the neonates was within normal limit with mean value being 9.13.The newborns presented with jaundice at a mean age of 2.4days and the mean duration of phototherapy required was 30.16hours. It was observed that there is a statistically significant ($p < 0.001$) fall serum bilirubin values with fall in serum magnesium noted ($p < 0.001$.Blood levels of serum magnesium and bilirubin was done at admission, after 12 hours , after 24 hours and at stopping phototherapy .The prevalence of hypomagnesaemia after 24 hours of phototherapy was 2.7% and prevalence of hypomagnesaemia at stopping phototherapy was 8.2%. It was observed that prevalence of hypomagnesaemia increase with increase in duration of phototherapy ($p<0.002$).

Conclusion

The current study found a statistically significant decrease in magnesium level after completion of phototherapy in term neonates with jaundice. In our study the prevalence of hypomagnesaemia after stopping phototherapy was 8.2% but all of the neonates were asymptomatic i.e none of them had any symptoms of hypomagnesaemia like seizures, tachycardia, nystagmus, arrhythmia, fasciculation etc. This find hypothesis that an increase in level of extracellular magnesium is a defense mechanism that reduces the neurotoxic effect of bilirubin. Further studies are required to determine the values of magnesium treatment in the therapy of infants with high levels of bilirubin in order to reduce the toxic effects of bilirubin.

Keywords

Hyperbilirubinemia, Neonates, Hypomagnesemia, Phototherapy.

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INTRODUCTION

Neonatal jaundice is a common finding in neonates during the initial seven days of life. The yellowish discoloration of sclera and skin is because of deposition of unconjugated bilirubin.[1]

Jaundice is more prevalent during neonatal period and is found in around 60% of term neonates and 80% of preterm neonates [2]. In early neonatal period readmission to hospital for neonatal jaundice is seen in around 6.5% of the babies[3]. In significant number (4%) of term newborns, around 85% are readmitted for jaundice[4].

According to recommendation all the neonates leaving from hospital before completing 2 days of life should have a visit to hospital within 2 to 3 days of discharge to look for any clinical signs of jaundice [5].

Phototherapy a commonly used and comparatively safer modality of treatment used to treat neonatal jaundice. Phototherapy can lead to few side effects including electrolyte imbalance like hypocalcaemia, hyponatremia, hypokalemia, dehydration, hyperthermia, feed intolerance, diarrhea and retinal damage [7]. There are very few studies showing hypomagnesaemia as a side effect following treatment with phototherapy.

Magnesium is an abundant cation and second common intracellular cation in body[8]. It has multiple function in body like activation of around 300 enzymes, relaxation and contraction of muscles, neuronal activity, neurotransmitter release and cardiovascular activities[9].

The main aim of this study is to know the correlation between serum magnesium in newborn with jaundice at initiation and after ending phototherapy to know prevalence of hypomagnesaemia .There are many studies showing phototherapy leading to hypocalcaemia but there are paucity of Indian and western studies showing hypomagnesaemia in term neonates after phototherapy.

OBJECTIVES

Primary: To know the prevalence of hypomagnesaemia in term neonates with jaundice after receiving phototherapy

Secondary: To study the association of hypomagnesaemia with duration of phototherapy

REVIEW OF LITERATURE

A. HISTORY

Hippocrates made reference of jaundice in his literature as a serious disease seen among infants[10].

In 1913 for the first time neuronal and muscular dysfunction was documented in a child who survived severe neonatal hyperbilirubinemia[11].

Phototherapy was used a new treatment modality after its discovery 60 years ago, it was observed that it decreased the need for exchange transfusion and improved neurological outcome in neonates with jaundice[12].

In the year 1958 the very first use of artificial light source was made instead of sunlight and it was seen that it significantly reduced serum bilirubin levels . This promoted the first time use of 'cradle illumination' device and the result were reported in *The Lancet* [13].

B. Fetal bilirubin metabolism

The mechanism of excretion of unconjugated bilirubin synthesized during fetal life is through the placenta into the maternal circulation[14].

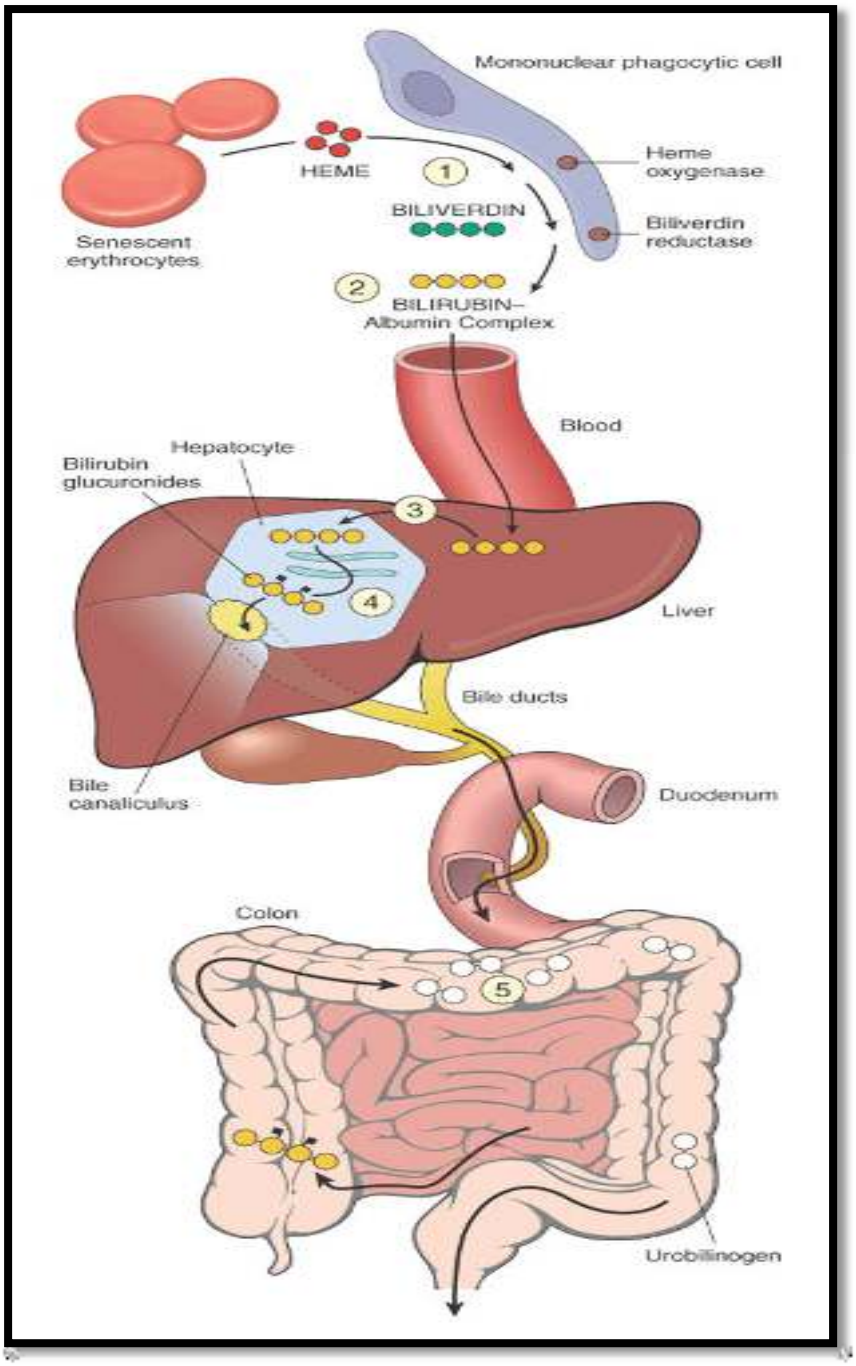


Fig 1 : Bilirubin metabolism[19]

Bilirubin metabolism [15]

There are various enzymatic reactions in body which are required for conversion of heme to bilirubin.

Bilirubin synthesis

The first and rate limiting step is formation of biliverdin from heme by enzymatic action of heme oxygenase [20].

Biliverdin reductase helps in synthesis of bilirubin from biliverdin[20].

The bilirubin in the plasma is attached to albumin, this bound form cannot enter the central nervous system so it is non-toxic[15].

Bilirubin uptake[15]

To cross hepatocyte cell membrane it should be disintegrated from albumin. This dissociated bilirubin will bind to Y protein (cytoplasmic ligand) in cytoplasm and it is transported to smooth endoplasmic reticulum. The concentration of ligand is increased by phenobarbitone.

Bilirubin conjugation[15]

The direct bilirubin is waters soluble, it is synthesized by enzymatic action of uridine diphosphate glucuronyl transferase. Both forms of conjugated bilirubin can be excreted against concentration gradient to bile duct.

Bilirubin excretion[15]

Conjugated bilirubin which contain large amount of bilirubin enters gastrointestinal tract and is ultimately exerted through stool.

Entero hepatic circulation of bilirubin[15]

The β-glucuronidase present in intestinal epithelium convert the conjugated bilirubin into unconjugated bilirubin which is reabsorbed from bowel.

Uses of bilirubin

The bilirubin has an antioxidant property it is one of its physiological role .It was studied in early 1950s that bilirubin protects against oxidation of vitamins like vitamin A and linoleic acid [16]. In late 1980s it was reported by Ames and colleagues that this antioxidant property of bilirubin is much more than that of vitamin E towards reactions like lipid peroxidation[17].

D. Causes of jaundice among neonates

Etiology

I. Physiologic jaundice[15]

i. Increased bilirubin production due to
    a. Decreased RBC lifespan among neonates (90 versus 120 days) and an increased RBC volume .
ii. Neonates will have more β- glucuronidase enzyme, less GI motility and decreased gut bacteria which leads to increased enterohepatic circulation.
iii. There is reduced binding capacity of ligandin to anions.
iv. Hepatic excretion of bilirubin is reduced.

Age based classification of causes of jaundice is important to decide treatment[20]

E. Complications of neonatal jaundice

The detrimental effects due to excess of bilirubin on CNS is called as Bilirubin encephalopathy. Kernicterus is referred to the characteristic neuropathological changes seen in particular areas of brain like basal ganglia, pons and cerebellum [11].

Bilirubin entry into brain

The main mechanism leading to damage to brain by unconjugated bilirubin is not well understood. Multiple hypotheses are being formulated to explain damage to brain by bilirubin toxicity [11].

One hypothesis states that, fat soluble nature of free bilirubin in blood will be in balance with bilirubin bound to albumin and it can enter the tissues. Thus, any imbalance in this homeostasis will elevate the free bilirubin within neuronal cells [11].

Second hypothesis is based on chemical nature of bilirubin .This theory correlates the pH and concentration of bound form of bilirubin with the bilirubin uptake rate by tissues. The hypothesis postulates that the low pH will increase the precipitation and uptake of bilirubin by the tissue [11].

Third hypothesis suggests that any damage to brain barrier will allow the entry of bound bilirubin into the brain[11].

Factors which increase the risk of jaundice are asyphixia, incompatibility, improper feeding, weight less for gestational age, any hematoma and preterm neonates[21].

Predictors of neuronal damage[11]

Brainstem Evoked Auditory Response (BERA)- is modality that can pick up damage caused by high bilirubin to brain because auditory pathway in newborn is more susceptible to damage from high levels of bilirubin. In a study conducted among 50 term neonates with moderate jaundice it was observed that ,latency period for BERA waves V and IV were longer than latency period observed among neonates with lower bilirubin levels[22].

According to various study conducted on BERA and their use in clinical practice it has been concluded that BERA can be applied to screen neonates with hyperbilirubinemia for sensory neural hearing impairments and BERA can also be used to assess the requirement of exchange transfusion[23 ,24].

Nuclear Magnetic Resonance Techniques (NMR) –are noninvasive, rapid test used to measure injury to brain cells due to hyperbilirubinemia[25].

F. Evaluation and diagnosis of neonatal jaundice

Neonatal jaundice is common physiological problem seen in around 60% term and around 80% preterm newborns during first year of life.

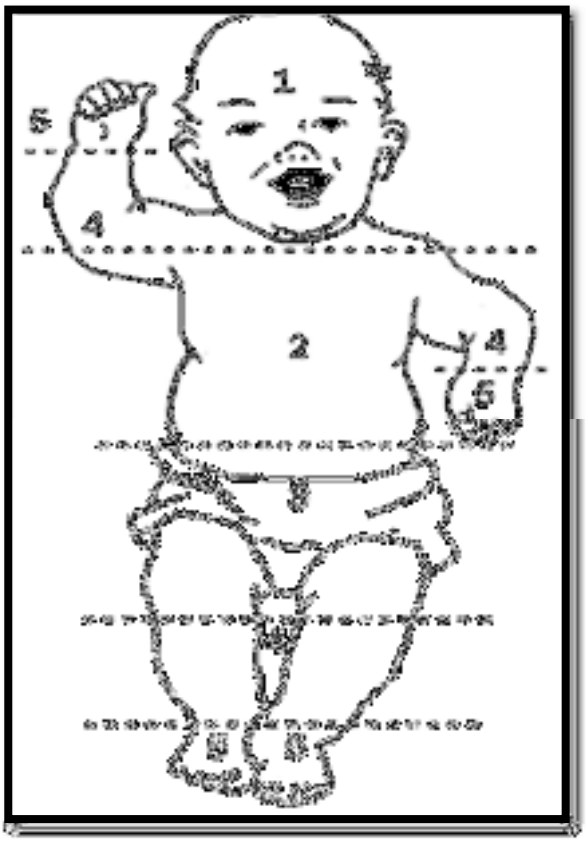


Fig 2 : Dermal zones according to Kramers rule

Table 1: Clinical assessment of jaundice based on Kramer's Dermal staining [26].

Area of body	Level of bilirubin(mg/dl)
Face	4-8
Upper trunk	5-12
Lower trunk & thighs	8-16
Arms & lower legs	11-18
Palms & soles	>15

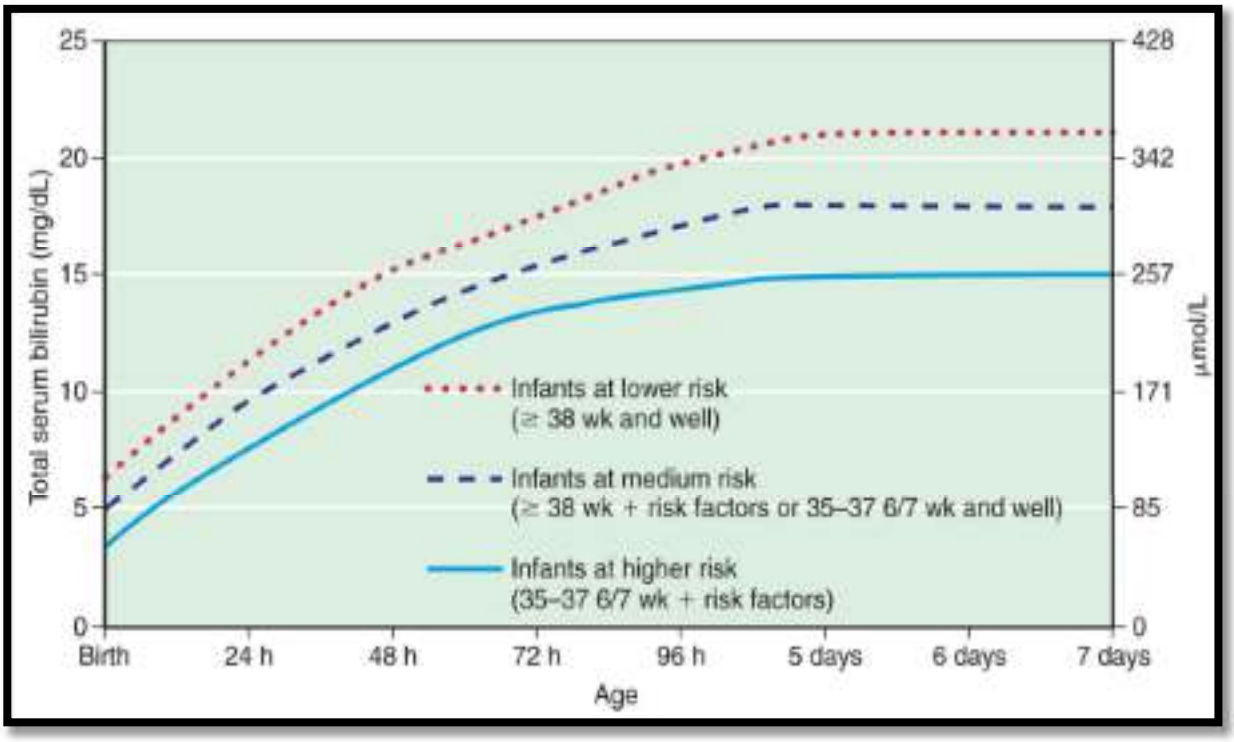


Fig 3: Graph to decide phototherapy in hospitalized neonates of ≥35 weeks gestation[18]

- Risk factors considered among newborns

➢ Alloimmune hemolytic disease,

➢ Deficiency of G6PD , asphyxia

➢ lethargy

➢ Instability in temperature

➢ sepsis, acidosis

➢ albumin <3.0 g/dL

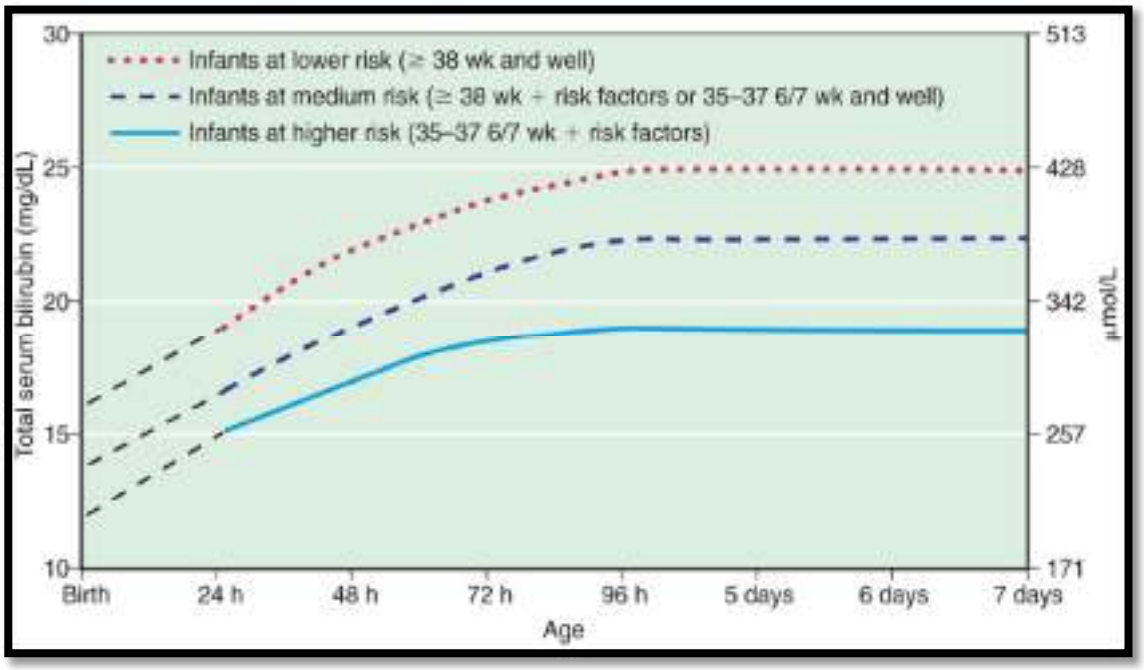


Fig 4: Graph to decide exchange transfusion among newborns ≥35 weeks [18]

G. **Laboratory Evaluation of Jaundiced Newborn**

Investigations are customized according to probable cause of hyperbilirubinemia based on clinical presentation. Investigation list as follows:

I. Maternal: Indirect Coombs Test (ICT) and Blood grouping of mother .

II. Neonates:

- Total and direct bilirubin.
- Blood group of newborn, Rhesus typing and Direct coomb test (DCT).

H. Treatment of Neonatal Hyperbilirubinemia[1]

Neonatal jaundice is one of the medical emergency and needs initiation of treatment without delay because any delay will cause permanent damage to brain.

There are various modalities to treat hyperbilirubinemia which include exchange transfusion, phototherapy and pharmacotherapy.

Jaundice treatment according to NNF guidelines[21].

1. Phototherapy is started based on total bilirubin value.
2. To initiate phototherapy gestational age is important that weight of newborn.
3. Age of the neonate should be considered in hours from birth.
4. Presence of conditions like asphyxia, hypoxia, hypercapnia, sepsis, any evidence of hemolysis and hypothermia indicates newborn is sick.

PHOTOTHERAPY (PT)

It was introduced in 1950s as a treatment modality of neonatal jaundice[13]

PT is one of the safe and effective modality for treatment of neonatal jaundice. There are various mechanism used in lowering the bilirubin level by PT.

Photo oxidation

It converts bilirubin into water soluble but this process of excretion of bilirubin is very time consuming.

Configurational photoisomerization

Bilirubin is converted to nontoxic, water soluble E-isomers. They are formed 8-12 hours after starting the treatment.

Structural isomerization

It is formation of lumirubin which is water soluble and structural isomers of bilirubin. This photocatabolites is removed from body by bile, stool and to a very less extent by urine. The process is irreversible and lumirubin cannot be reabsorbed into blood stream. It is most effective way to remove bilirubin from body. The dose of phototherapy needed to carry out this process is between 6 -12 $\mu w/cm^2/nm$.

Bilirubin absorbs light from the region of blue area with wavelength corresponding to 460nm. The degree of formation of photoproducts from bilirubin is mainly dependent on intensity of light used.[27]

Combination of 2 special blue and around 4-6 white fluorescent tubes to be used .This combination would deliver 12 $mw/cm^2/nm$.[27]

Bilirubin will absorb light and produces a transient state of bilirubin, these transient forms will combine with oxygen to release a colorless product of low molecular weight, they can produce lumirubin or any form of bilirubin isomer.

Lumirubin isomers will be removed by urine. Once in bile, configuration isomers revert spontaneously to the natural 4Z 15Z form of bilirubin [27,28].

Phototherapy procedure

The blue light with wavelength around 460nm is ideal for a good phototherapy. White fluorescent lights are effective, ideal and commonly used in India. Other phototherapy lights available are blue and white tube units.

Neonate is completely undress except for eye patch and diaper and kept at a distance of 45cm from light source. In case intensive phototherapy is needed the distance can be reduced to 15-20cm.

Phototherapy is said to be effective if spectral irradiance is between 4 to 6 µw/cm2/nm and this same intensity should be maintained on neontaes skin also.

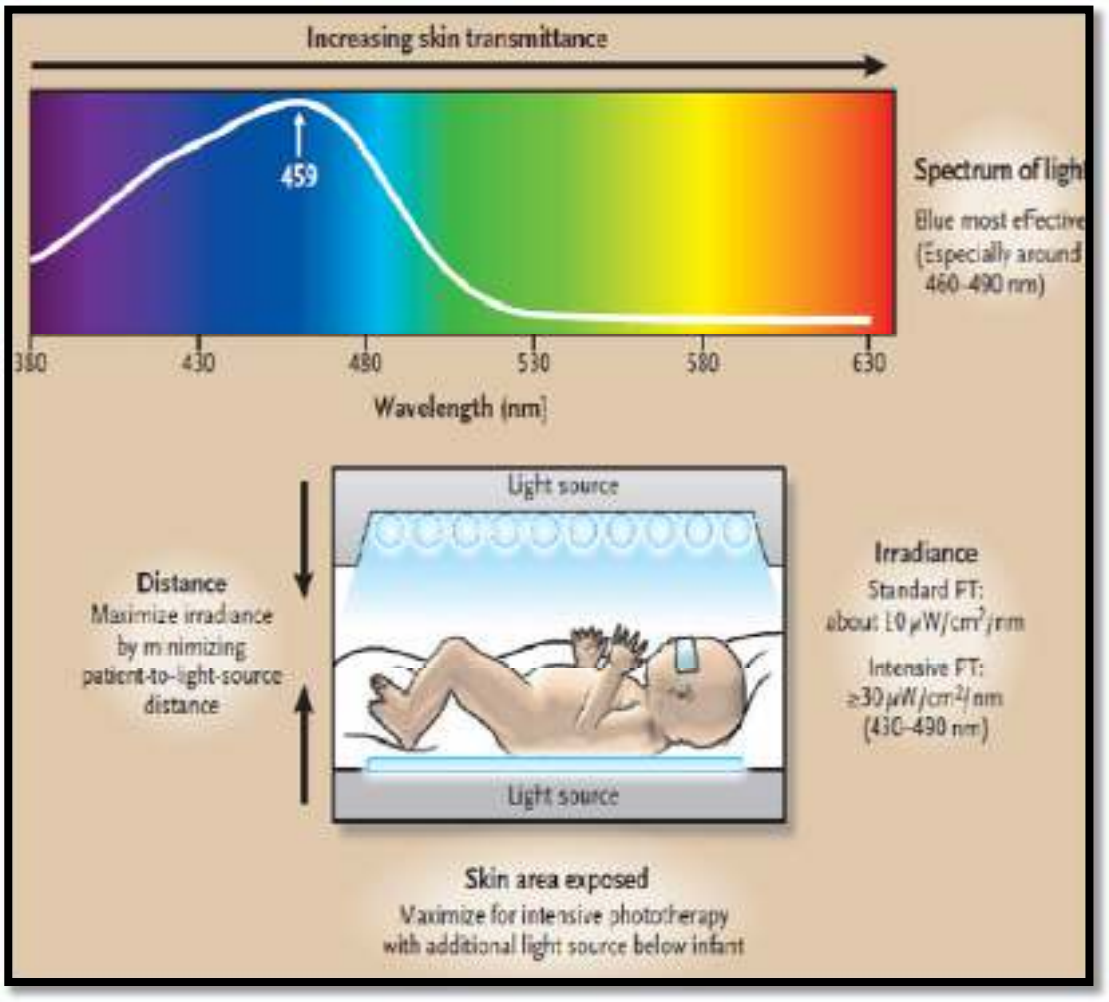


Fig 5 : Important factors for efficacy of phototherapy[5]

Side effects[29]

- Dehydration- the commonly used conventional phototherapy leads to change in thermal environment of neonates and may be reason for insensible water loss and dehydration with hypothermia or hyperthermia.
- Hyperthermia-high intensity PT lead to increased blood flow through tiny vessels of skin and muscles which causes increase in skin temperature.
- Bronze baby syndrome – it is due to excessive accumulation of bilifucin in skin which gives typical brownish color to skin of newborn with liver disease. The exact pathology for discoloration is not know but it usually resolves in few days after stopping phototherapy.
- Loose green stools-are caused due to irritant effect of photocatbolites on gut which leads to increased colonic secretions. Irrespective of bowel irregularities breastfeeding has to be continues and if required fluid resuscitation can be started.
- Bullous and purpuric eruptions- circulating levels of porphyrins in newborn causes development of bullous rashes. Rashes may develop within a day of starting PT .The rashes are self limiting within a period of one week even without any treatment.
- Electrolyte imbalance - De Curtis M, *et al. concluded* that absorption of electrolytes with water is inhibited in neonates receiving phototherapy mainly due to gut secretion[30].
- Hypocalcaemia - melatonin produced from pineal gland is altered due to altered circadian rhythm. Melatonin has a peak release during night times so it is called as hormone of darkness. Illumination of brain during phototherapy can reduce the melatonin levels, which in turn leads to hypocalcaemia. Other

action of melatonin on calcium metabolism in body are, it promotes bone formation, decreases bone resorption, increase activity of osteblasts, it decreases osteoclast cell activity.

- Other electrolyte imbalance seen are, hypomagnesaemia hypokalemia, hyponatremia etc
- Reopening of PDA among preterm neonates .Photons present in the cell will absorb the light during PT, it leads to activation of nitric oxide system which causes smooth muscles of aorta to get relaxed relaxation. PT causes changes in heart rate, heart output and also reduces the MAP .These cumulatively leads to opening up of PDA.
- Few studies theoretically postulates that PT increases the risk of skin cancers during adulthood. This was explained by theory that PT causes damage to DNA in cells by various mechanisms like oxidation injury to membrane .This ultimately leads to injury to DNA strands and will result in mutation.
- Early or delayed onset of puberty and altered sexual behavior in future is due to disturbance in circadian rhythm due to exposure to light. The altered circadian rhythm can be the cause of jitteriness, increased crying and altered heart rate among neonates.
- Damage to retina –the light from PT leads to photochemical changes in retina . Light will be absorbed by rhodopsin and it triggers lipid peroxidation and apoptosis of cells.

MAGNESIUM

Magnesium is a divalent cation and is the second common intracellular cation which has many necessary roles for normal functioning of body[31].

It has vital role in maintaining normal function of human body[30]-

- It is necessary for production of protein and nuclear material.
- It acts as cofactor for various enzymatic reaction and transporter system in body
- ATP and intracellular magnesium leads to formation of key complex and they have important role in various functions like as synthesis of protein , cell duplication and energy metabolism.
- It helps in contraction and relaxation of muscles, release of various neuronal transmitters for nerve activity and cardiac activity [31].

BODY CONTENT AND DISTRIBUTION[33]

The normal human body contains around 1000mmols of magnesium (22-26g). In that about 60% is within bones, of that 30% functions as a reservoir to normalize the serum levels of magnesium. Normal range of magnesium is 1.6-2.4mg/dl.

Skeletal muscles has 20%, soft tissues has 19 % and in extracellular fluid has less than 1 % of total body magnesium.

Around 20% of magnesium is bound to protein, ionized magnesium constitutes 65% and remaining of will form complex with various in body. 60-70% is bound to albumin and remaining 30% is associated with globulin.

Rate at which magnesium is transported through cell membrane differs from cell to cell, transport is more in liver, kidney, heart and less in skeletal muscle, brain and blood cells. Magnesium present within the cell is more in actively dividing cells it indicates that magnesium within cell is linked to metabolic function of cell[34].

Various organs are involved in hemostasis of body magnesium among them vital role is played by gastrointestinal tract, skeletal and renal system. Studies have shown that there is a correlation between magnesium level and severity of jaundicein neonates.

MAGNESIUM AND PHOTOTHERAPY

Magnesium along with all the functions mentioned above has an inhibitory action on NMDA receptors in body. The closure of NMDA receptors is mainly mediated by voltage dependent action of magnesium ions. It has been observed that there will be increase in serum magnesium levels in cases with neonatal jaundice.[35]

The various mechanisms used to explain the increase in magnesium following neonatal jaundice are to lysis of cells specially neurons and red blood cells that leads to release of intracellular magnesium .This increase in magnesium in cases of hyperbilirubinemia is attributed to a compensatory protective action of magnesium on neurons which help in improving the neuronal outcome .[36]

There are very few studies showing hypomagnesaemia as a side effect following phototherapy. The probable mechanism being the trans cranial illumination during phototherapy will leads to decreased levels of melatonin by inhibiting the pineal glands. Melatonin has inhibitory action on corticosterons. Corticosterone acts

on the bones and it increases the absorption of magnesium and calcium leading to hypomagnesaemia and hypocalcaemia .[37]

CLINICAL FEATURES SEEN IN HYPOMAGNESEMIA[31]

- Convulsions
- Involuntary muscle contractions
- Muscle weakness and cramps
- Fasciculation
- Nystagmus
- Atrial tachycardia, fibrillation
- Ventricular arrhythmia
- Hypocalcemia
- Hypokalemia
- Altered glucose hemostasis
- Osteoporosis

There are few studies showing positive correlation between phototherapy and hypomagnesemia. Sarici *et al.* depicted a correlation between magnesium levels and degree of jaundice in term neonates with jaundice[38].

Frargy et al conducted a study in hospital at Egypt for a duration of 1 year and study concluded that phototherapy can exponentially reduce levels of serum magnesium and bilirubin [36].A retrospective cross sectional study examined 143 newborns with hyberbilirubinemia in a medical center in Iran by Mosayebi et al ,concluded that there will be a significant decrease in magnesium levels in newborns with jaundice after receiving phototherapy irrespective of cause of the jaundice[39].

Karambin et al conducted a observational study in a hospital in Iran for a period of 1 year .It was concluded that magnesium level was significantly lower in neonates after receiving phototherapy compared with magnesium values before receiving phototherapy[40].Reddy et al conducted study in India for period of one and half year among 252 neonates and evaluated the electrolyte levels in newborns after phototherapy .It concluded that newborns undergoing PT for hyperbilirubinemia are at increased risk for developing electrolyte changes[41].

Study conducted by Shahriarpanah et al in Iran on neonates with hyperbilirubinemia who received phototherapy as a treatment modality for hyperbilirbinemia concluded that it can reduce the level of serum calcium and magnesium and also simultaneously increase the level of vitamin D in body[42].Bezboruah et al conducted a study in India on 206 neonates with neonatal hyperbilirubinemia for a period of 1 year and it was observed that phototherapy can lead to marked electrolyte dysfunction in newborns and premature ,birth weight less than expected newborns are at higher risk and these groups should be under continuous close monitoring[43].

Abdel-Raouf Khattab ,Raafat et al conducted a cross sectional study for 1 year in 100 neonates with neonatal jaundice at Cairo Egypt . The study concluded that mean serum levels of copper magnesium and calcium was significantly higher and mean levels of serum zinc was found to be less in neonatal jaundice. There was a statistically significant reduction in bilirubin, magnesium and calcium along with statistically significant increase in zinc and copper after phototherapy[44].

Hasan compared a group of neonates with hyperbilirubinemia with healthy infants and reported that there is increase in magnesium levels in icteric infants[45].

Choudhury and Borkotoki demonstrated a correlation between serum bilirubin and plasma magnesium concentration, this was explained by the mechanism of extracellular transfer of magnesium[46].

Gathwala et al studies the treatment with magnesium sulfate during postnatal period has a neuroprotective role against bilirubin toxicity[47].Mohsen et al concluded that there will be generalized injury to nerve cells and red blood cells leading to extracellular movement of magnesium .It leads to increase in serum magnesium during hyperbilirubinemia. This accentuates the neuroprotective role against the increasing serum bilirubin [48].

MATERIAL AND METHODS

Study design: Cross sectional study

Study duration: September 2020 to September 2021.

Place of study: KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Inclusion criteria:

1) Full term neonates with indirect hyperbilirubinemia requiring phototherapy

Exclusion criteria:

1. Preterm newborn
2. Conjugated hyperbilirubinemia
3. Jaundice within first 24 hours of life
4. History of administration of maternal magnesium sulphate
5. Neonates with history of birth asphyxia, obvious congenital malformation, sepsis neonatal hepatitis

Sample size:

The formula used for sample size calculation is,

$$n = \frac{2\sigma^2_{pool}(1-\rho)(Z_{\alpha/2}+Z_{\beta})^2}{(\mu_1 - \mu_2)^2}$$

Where μ1 is the mean of the variable before time point (pretest), μ2 is the mean of the variable after time point (post time) and $\sigma 2pool$ is the pooled variance,

for 95% $Z\alpha 2/$ value is 1.96 and for 90% power $Z\beta$ value is 1.28. Magnesium level (mg/dl) before and after phototherapy for subjects who suffered from jaundice with pathological hyperbilirubinemia is 2.3±0.7 and 2±0.5, respectively from below reference. $\sigma 2$ can be calculated from the below formula

$$\sigma^2 \text{pool} = \sqrt{s^2pre + s^2post - 2 \times \rho \times s_{pre} \times s_{post}}$$

For assumed ρ value as 0.3, from above formula, **minimum sample size required is 64 subjects**

Methodology:

Informed consent will be obtained from parents /guardian of neonates who meets the inclusion criteria and the neonates will be enrolled in the study. Patient data will be recorded in structured profoma. Serum bilirubin level and magnesium level will be sent before starting phototherapy, after 12 hour, after 24 hours and after stopping phototherapy. Serum calcium is checked at admission.

Patient data:

The following details were collected before enrolling a newborn for the study History including maternal comorbidities, mode of delivery, blood group, gestational age, neonatal birth weight, sex, history of birth asphyxia, APGAR scores at 1min and 5min

Laboratory investigation –

- Serum bilirubin using Diazo method
- Serum magnesium level using Xylidyl blue method

Statistical analysis:

Data were entered in MS-Excel and analysed in SPSS V25. Descriptive statistics were represented with percentages, Mean with SD. Shapiro wilk test was applied to find normality. Independent t-test were calculated. $P<0.05$ was considered as statistically significant

RESULTS

It is a hospital based cross-sectional study conducted for period of one year from September 2020 - September 2021 among the new-borns admitted under the Department of Paediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

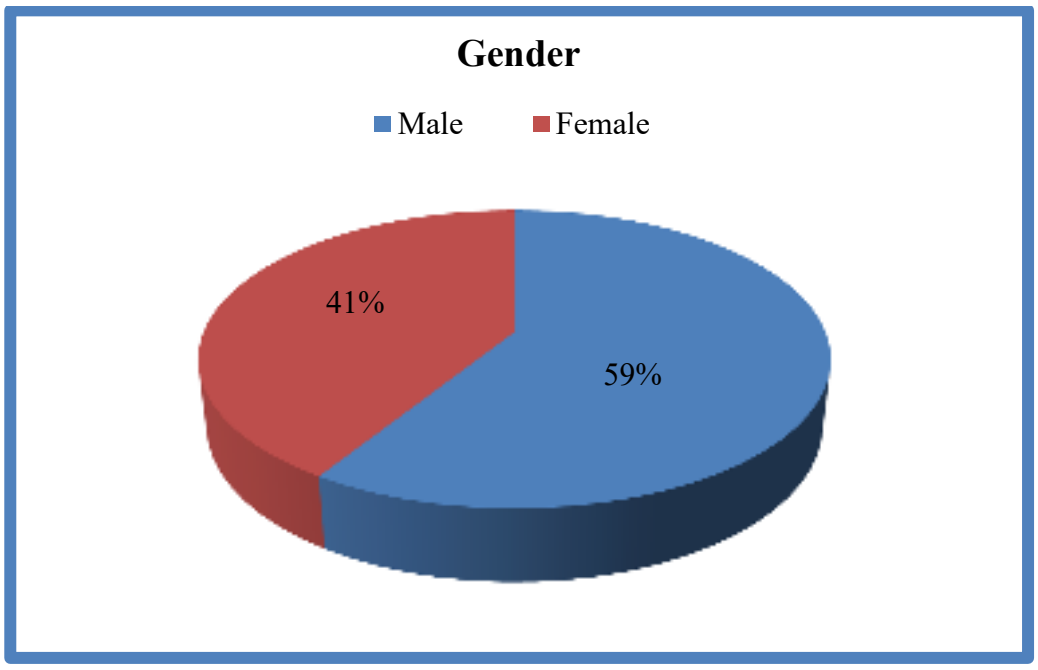
A total of 73 neonates satisfying the inclusion criteria were enrolled into our study.

Results of Demographic and Clinical characteristics

Table 2: Gender of neonates

Gender	Frequency	%
Male	43	58.9
Female	30	41.1
Total	73	100.0

Fig 6 : Gender of neonates

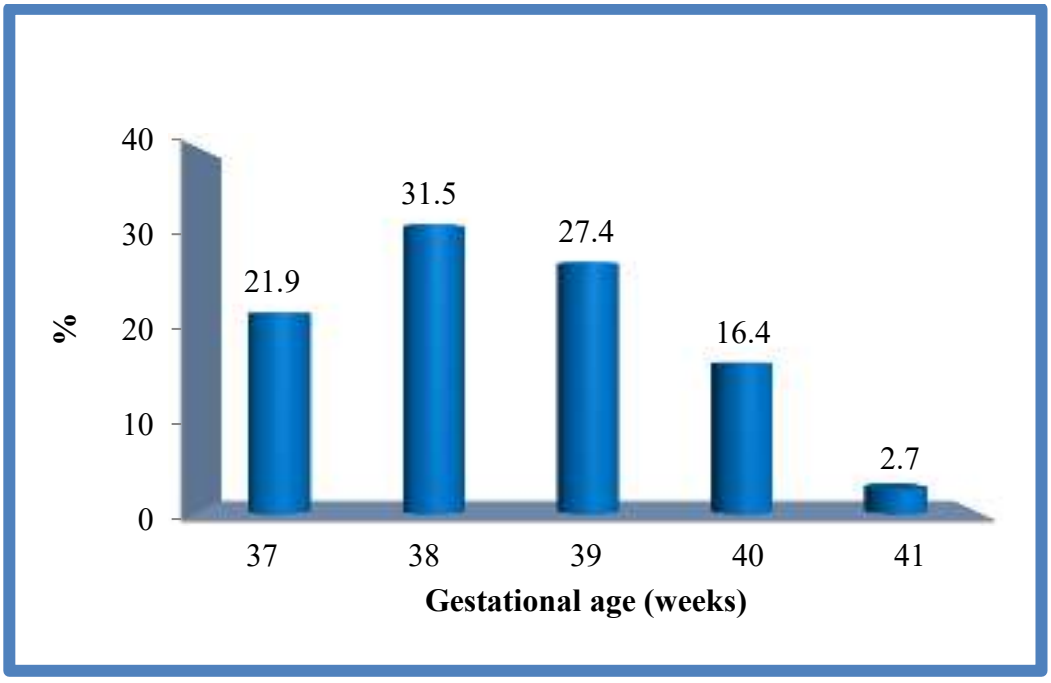


The table depict that gender distribution of newborns in study is 43(58.9 %) males and 30 were females (41.1%)

Table 3: Gestational age of newborns

Gestational age (weeks)	Frequency	%
37	16	21.9
38	23	31.5
39	20	27.4
40	12	16.4
41	2	2.7
Total	73	100.0

Fig 7 : Distribution of gestational age among newborns

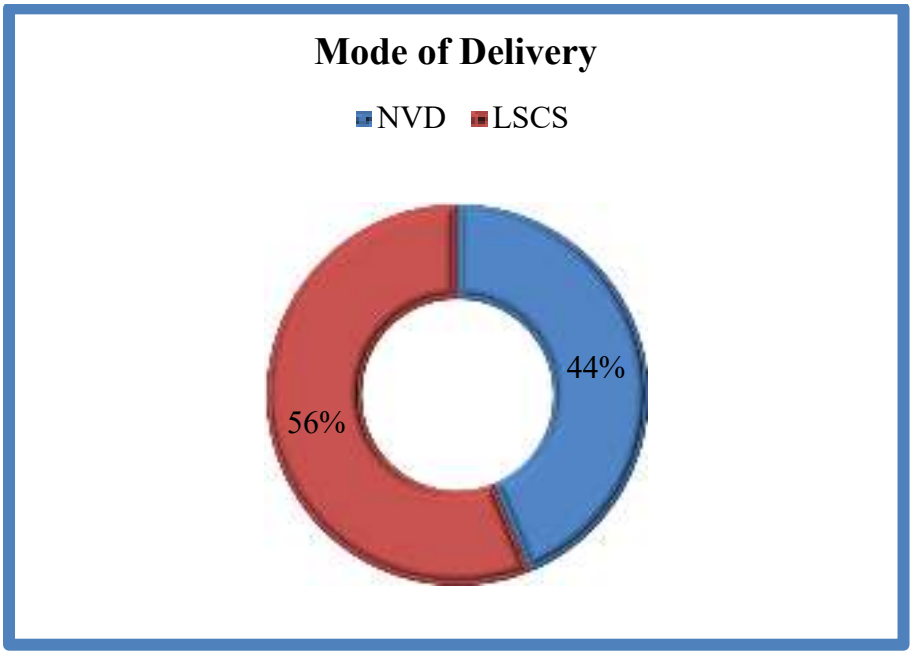


Majority of newborns who developed neonatal jaundice and required treatment belonged to gestational age of 38 weeks i.e 23 (31.5%)

Table 4 : Mode of delivery

Mode of Delivery	Frequency	%
NVD	32	43.8
LSCS	41	56.2
Total	73	100.0

Fig 8: Mode of delivery

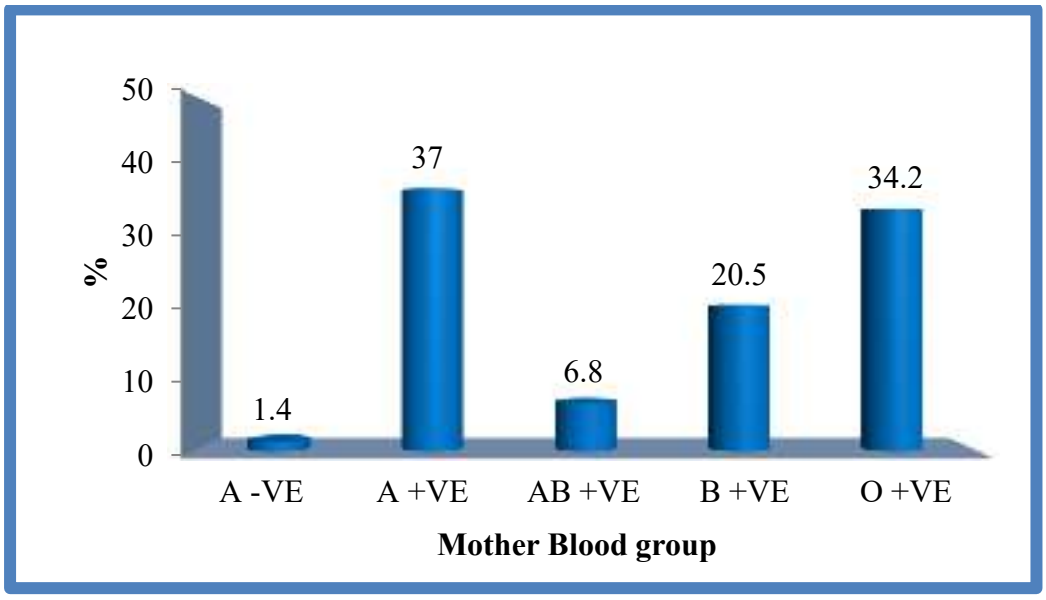


Majority of newborns in study group were born by LSCS constituting 41 out of 73 i.e 56.2%.

Table 5: Maternal blood group

Blood group	Frequency	%
A -VE	1	1.4
A +VE	27	37.0
AB +VE	5	6.8
B +VE	15	20.5
O +VE	25	34.2
Total	73	100.0

Fig 9: Maternal blood group



Majority of mothers had blood group A positive i.e 37% while second most common blood group being O positive i.e 34.2%

Table 6 : Antenatal magnesium sulphate

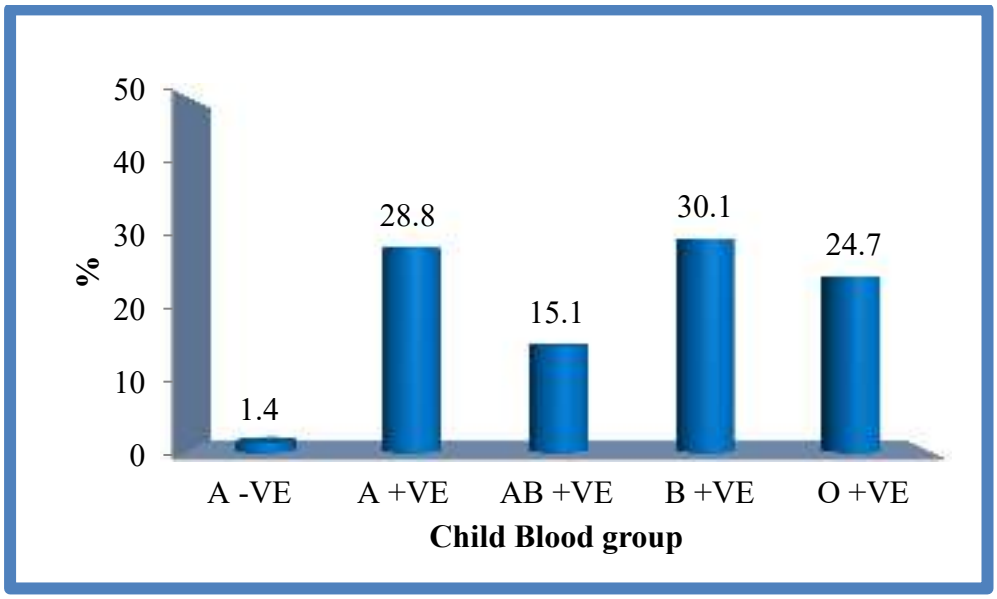
Antenatal magnesium sulphate	Frequency	%
NO	73	100.0

None of the mothers had received antenatal magnesium sulphate injection.

Table 7 : Neonatal blood group

Blood group	Frequency	%
A -VE	1	1.4
A +VE	21	28.8
AB +VE	11	15.1
B +VE	22	30.1
O +VE	18	24.7
Total	73	100.0

Fig 10 : Neonatal blood group



Distribution of neonatal blood group, out of 73 newborns 22 had B positive i.e 30.1% while second most common blood group being A positive i.e 28.8%

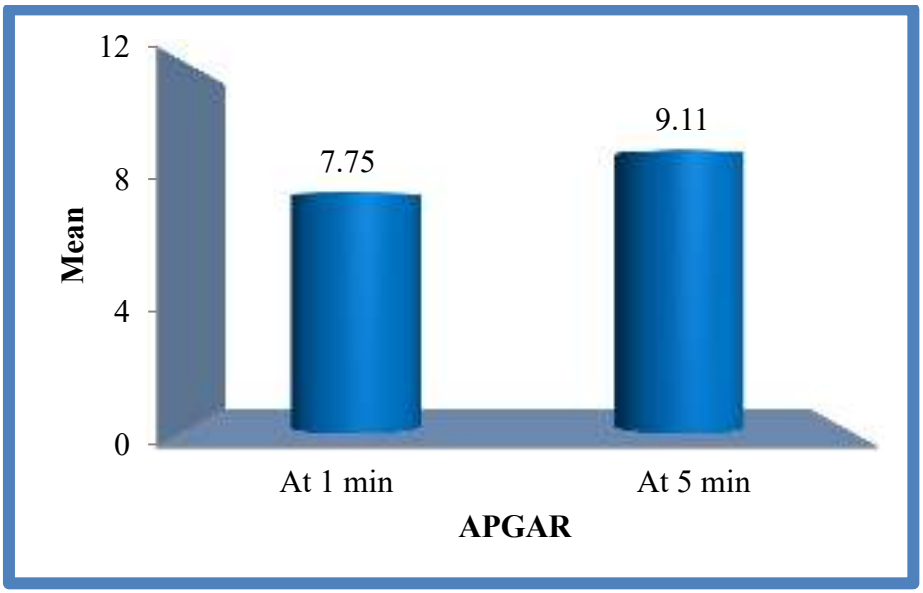
Table 8 : Demographic character of newborns in study group

Variable	N	Mean	SD
Gestational age(weeks)	73	38.47	1.09
Weight(kg)	73	2.68	0.26
APGAR at 1 min	73	7.75	0.68
APGAR at 5 min	73	9.11	0.61
Day of life at presentation	73	2.40	0.52

The mean gestational age (GA) of newborns enrolled in study was 38.47 weeks and mean weight of study population being 2.68kgs.

The average age of presentation of neonatal jaundice among the newborns enrolled in the study was 2.4days

Fig 11 : APGAR at 1 and 5 minutes after birth



This table shows that average APGAR score at 1 min of life was around 7 and average APGAR score at 5min of life is around 9

Table 9 : Phototherapy duration

Variable	N	Mean	SD
Phototherapy duration (hours)	73	30.16	5.18

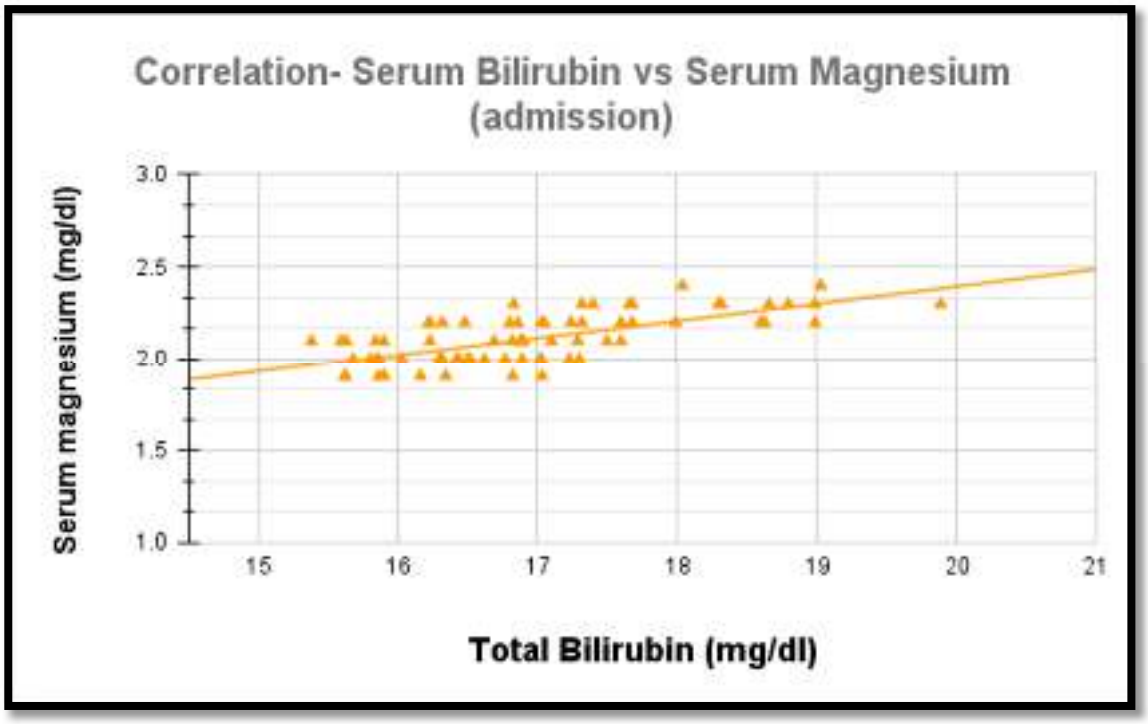
The mean duration of phototherapy received by new-borns with hyperbilirubinemia among the study population is 30.16 hours

Table 10: Calcium at admission

	N	Mean	SD
Calcium at Admission	73	9.13	0.26

Calcium levels were checked for all the neonates with hyperbilirubinemia before initiation of phototherapy .The mean serum calcium in study group was 9.13mg/dl

Fig 12: Correlation between admission serum bilirubin and serum magnesium

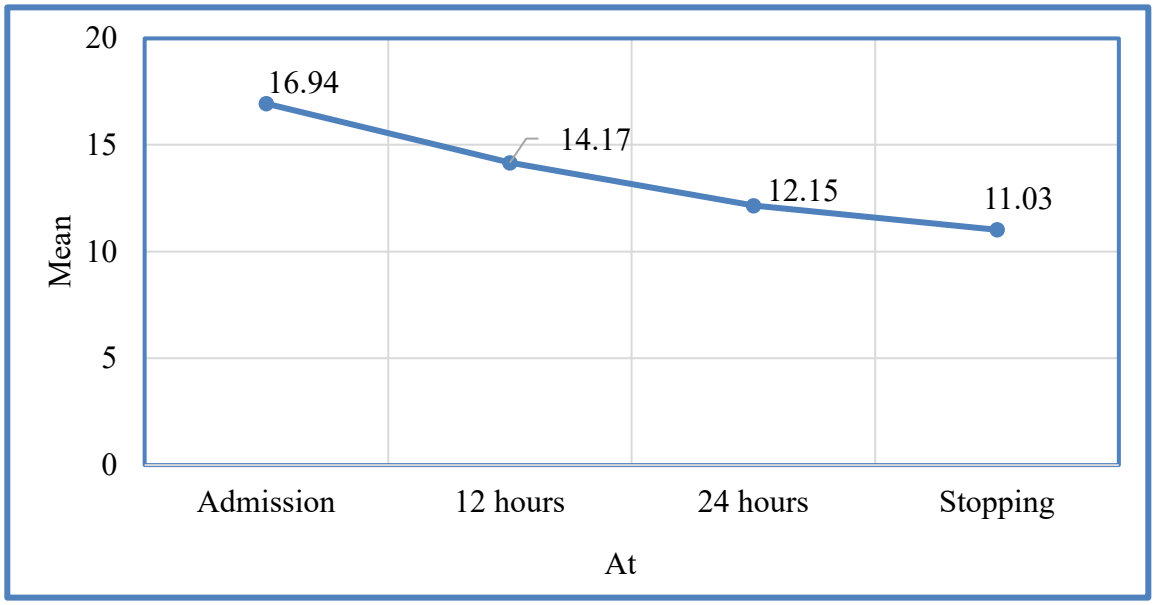


In present study it was observed that there is a positive correlation between levels of magnesium and bilirubin at admission (r= 0.6848 and p = <0.001)

Table 11 : Total bilirubin values at admission , 12hrs ,24 hrs and after stopping phototherapy

	N	Mean	SD	P-value
Total bilirubin at admission	73	16.94	0.99	**<0.001**
Total bilirubim after 12 Hours	73	14.17	1.19	
Total bilirubin after 24 Hours	73	12.15	1.02	
Total bilirubin after stopping	31	11.03	0.72	

Fig 13: Distribution of total bilirubin values at admission, 12hrs, 24 hrs and after stopping phototherapy

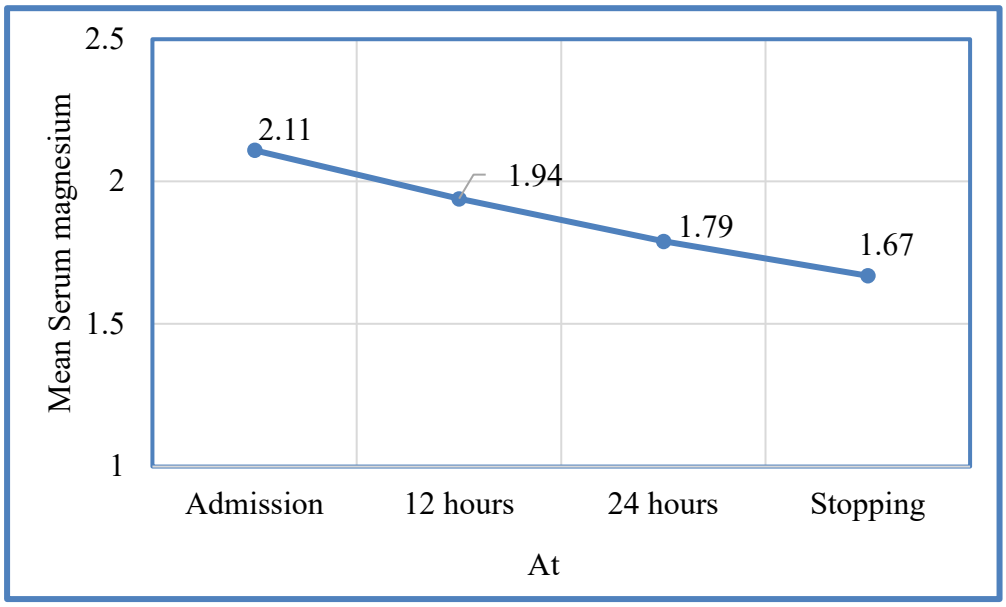


It was observed that the there was exponential fall in total bilirubin values in neonates receiving phototherapy for hyperbilirubinemia at admission , after 12hrs , after 24hrs and after stopping phototherapy with mean value of total bilirubin being 16.94, 14.17 12.15 and 11.03 respectively

Table 12 : Serum magnesium values at admission , 12hrs ,24 hrs and after stopping phototherapy

	N	Mean	SD	P-value
Serum magnesium at adm	73	2.11	0.13	**<0.001**
Serum magnesium after 12 hours	73	1.94	0.13	
Serum magnesium after 24 hours	73	1.79	0.13	
Serum magnesium after stopping	31	1.67	0.11	

Fig 14: Distribution of serum magnesium values at admission, 12hrs ,24 hrs and after stopping phototherapy



In our study it was observed that there was exponential decrease in the serum levels of magnesium among neonates who received phototherapy for neonatal jaundice. Mean serum magnesium levels at admission, 12 hours , 24 hours and after stopping phototherapy was 2.11 , 1.94, 1.79 and 1.67mg/dl respectively.

Table 13 : Serum magnesium levels at admission

Serum magnesium at admission	Frequency	%
Normal (1.6-2.4)	73	100.0

Table 14 : Serum magnesium at after 12 hours of phototherapy

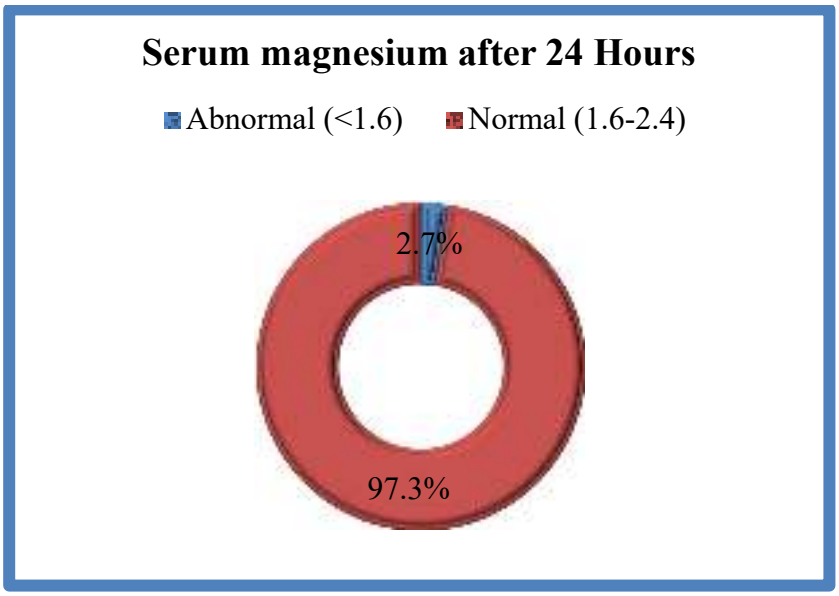
Serum magnesium after 12 Hours	Frequency	%
Normal (1.6-2.4)	73	100.0

Serum magnesium levels of all 73 neonates were within normal limit at the time of admission and after receiving 12 hours of PT.

Table 15 : Serum magnesium levels after 24hours of phototherapy

Serum magnesium after 24 Hours	Frequency	%
Abnormal (<1.6)	2	2.7
Normal (1.6-2.4)	71	97.3
Total	73	100.0

Fig 15 : Distribution of serum magnesium levels after 24hours of phototherapy

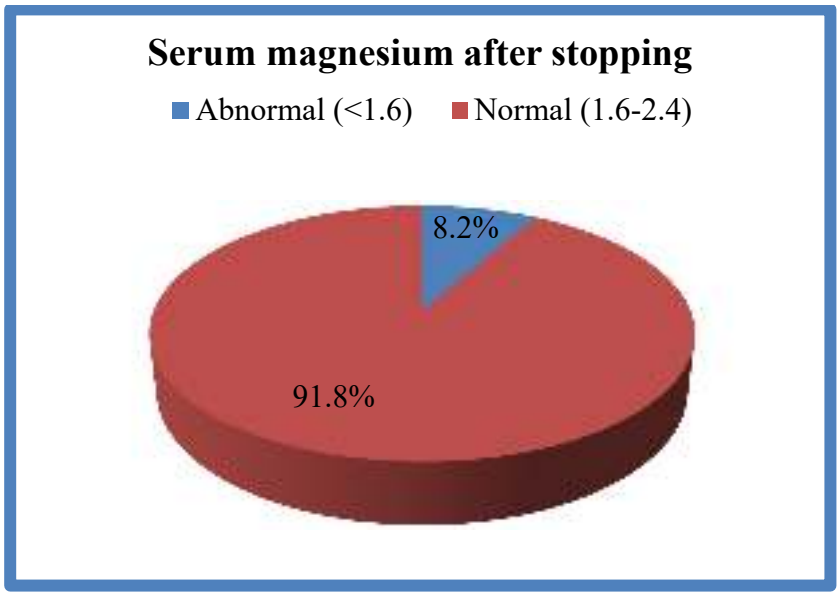


In our study it was observed that there was significant reduction in magnesium levels after 24 hours of phototherapy. Among 73 neonates in study group 2(2.7%) had serum magnesium values in hypomagnesemia range while rest of them had magnesium levels within normal limit.

Table 16 : Serum magnesium levels after stopping phototherapy

Serum magnesium after stopping	Frequency	%
Abnormal (<1.6)	6	8.2
Normal (1.6-2.4)	67	91.8
Total	73	100.0

Fig 16 : Distribution of serum magnesium levels after stopping phototherapy

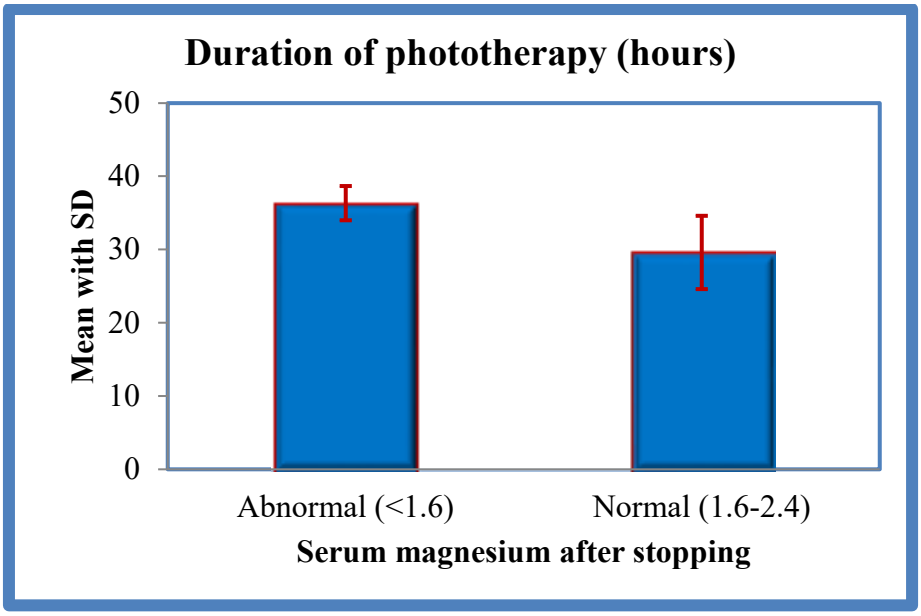


In our study it was observed that there was significant reduction in magnesium levels after stopping phototherapy. Among 73 neonates in study group 6(8.2%%) had serum magnesium values in hypomagnesemia range while rest of them had magnesium levels within normal limit.

Table 17 : Correlation between mean duration of phototherapy received and serum levels of magnesium

Duration of phototherapy (hours)				P-value
Serum magnesium after stopping	N	Mean	SD	
Abnormal (<1.6)	6	36.33	2.34	**0.002**
Normal (1.6-2.4)	67	29.61	5.01	

Fig 17 : Correlation between mean duration of phototherapy received and serum levels of magnesium



In our study it was observed that the mean duration of phototherapy leading to development of hypomagnesemia (i.e serum magnesium level <1.6) among neonates receiving phototherapy was 36.33 hours.

DISCUSSION

Neonatal jaundice is one common condition encountered among neonates specially during the first seven days of life. The clinical signs like yellowish discoloration of skin and sclera should be picked up and to be evaluated accordingly. Phototherapy is one of the effective treatment modality used to reduce serum bilirubin levels. There are various side effects associated with phototherapy, electrolyte imbalance being one among them. This study was conducted to know prevalence of hypomagnesaemia among term neonates receiving phototherapy and any possible correlation between duration of phototherapy with severity of hypomagnesaemia.

One year cross sectional study was conducted from September 2020 to September 2021 in the department of pediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre Belagavi. A total of 73 neonates were enrolled.

In our study comprising of 73 newborns with hyperbilirubinemia 58.9% were males and 41.1% were females which was consistent with the epidemiological findings found in a study done in Turkey by Sarici et al[38] which showed a demography of 55.1% male and 44.8 % were females. Similarly another study done in Iran by Karambin et al[40] showed male to female incidence was 56.9% and 43.1% respectively. Though there is no gender predisposition for development of hyperbilirubinemia it is observed in our study that prevalence is slightly higher among males.

The overall prevalence of neonatal jaundice is more among preterm neonates than compared to term neonates. Our study group included only full term newborns similar to study conducted by Karambin et al[40] ,Eghbalian et al[49] and Taheri PA et

al[50] .In our study it was observed that the majority of neonates requiring phototherapy were with gestational age of 38 weeks which is consistent with findings seen in a study done at Egypt by El-Mazary et al [51].

In our study 43.8% of newborns were delivered by vaginal route and 56.2 % by caesarean section. Amar Taksande et al[52] in the year 2005 concluded that with a p value of 0.527 there was no association between the development of neonatal jaundice and the mode of delivery. Rudy Satrya et al[53] conducted study on 88 newborns with cut off neonatal hyperbilirubinemia of ≥14.9 mg/dl that there was no correlation between mode of delivery and neonatal hyperbilirubinemia.

The mean age at presentation of a neonatal with hyperbilirubinmia according to our study was 2.4 days, similar conclusion was made by Abdel -Raouf Khattab, Raafat et al[44] in a study conducted in Egypt that the mean age of presentation with hyperbilirubinemia was between 48 to 96 hours of life. Similar to our study Subhashini B et al[54] concluded that the mean age of presentation was 3.54 +/-1.6 days. In contrast to this there are few studies conducted by Karambin MM et al [40] and Mosayebi et al[39] in Iran concluded that mean age of presentation can go upto 6.15+/- 3.7 and 5.89+/-3.18 days respectively.

It is observed that mean duration of phototherapy received by neonates in our study group was 30.16 hours similar observation was made by Mosayebi Z et al[39] and Reddy et al[41] with mean duration being 2.47+/-0.68 days and 37.65+/-11.06 hours respectively. In contrast to these studies Eghbalian et al[49] concluded that mean duration of 3days with range of 1 to 7 days.

In our study we evaluated the serum calcium values of all 73 neonates at the time of admission and it was found that the mean calcium value was 9.13mg/dl. A study conducted by Fatemeh Haji Ebrahim Tehrain et al[55] among term neonates with jaundice also had similar average of 9.51mg/dl of serum calcium at admission. Various other studies supporting this results are Subhashini B et al[54] with mean calcium 8.84+/- 0.46, Gaafar MM et al[56] with mean calcium 9.63+/- 0.79 , Reddy et al[41] with mean calcium 9.16+/- 1.00. It was observed from thses studies that the mean calcium values among all the neonates before starting for hyperbilirubinemia was within normal range.

Estimation of the mean levels of bilirubin at admission 12 hours, 24 hours and at stopping phototherapy in our study showed a stastically significant (p-value <0.001) decline in serum bilirubin values, mean levels being 16.94 ,14.17, 12.15 and 11.03 respectively. Similar statistically significant decline in serum bilirubin was observed by Frargy et al[7] (p value <0.001) and Abdel-Raouf Khattab, Raafat et al [44](p value <0.001) . A study conducted by Bezboruah et al[43] in a study group of 206 neonates it was shown that there was a statistically significant reduction in serum bilirubin after phototherapy (p value <0.0001).

In our study it was observed that the mean magnesium value at admission among all the study population was within normal limit i.e 2.11mg/dl (p value < 0.001). As phototherapy was continued to treat the neonatal jaundice it was seen that there was a liner reduction in the serum magnesium values over period of time. Mean serum magnesium levels after 12 hrs, 24 hours and after stopping phototherapy was 1.94, 1.79 and 1.67 respectively. It was observed that prevalence of hypomagnesemia

after 24 hours of phototherapy was 2.7% and prevalence after stopping phototherapy was 8.2%.

The prevalence of hypomagnesemia was correlated with the duration of phototherapy received by the neonates and it was observed that the mean duration of phototherapy of 36.33 hours was seen in study population who developed hypomagnesemia where as a mean duration of phototherapy received by study group who had normal magnesium value at stopping phototherapy was 29.61 hours. These values are statistically significant with p value of <0.002. In our study it was observed that the severity of bilirubin level was consistent with proportionate higher levels of magnesium.

Hasan et al[45] conducted a comparative study among a group of healthy infants with neonates with hyperbilirubinemia and concluded that there was comparatively a higher level of serum magnesium value in jaundiced baby. In support to this study Choudhury and Borkotoki et al[46] conducted similar study and came to same conclusion and they attributed the results to intracellular shift of magnesium. In our study it was observed that there is a positive correlation between admission value of serum bilirubin and serum magnesium.

In agreement with our study Khosravi et al[35] reported that there will be a statistically significant decline in serum magnesium value in correlation with bilirubin value in newborns at starting and stopping phototherapy. Also Imani et al[57] conducted a study in 2012 among neonates with hyperbilirubinemia requiring phototherapy and came to similar conclusion. These studies also explained regarding the relation between serum magnesium and severity of hyperbilirubinrmia.

Mehta et al[58] suggested a possible mechanism of increased serum levels of magnesium during increasing toxicity by excessive bilirubin, it was speculated that magnesium has a neuroprotective role and its level increases to compensate the bilirubin toxicity. This conclusion help us to explain the finding in our study.

In a study by Mohsen et al[48] it was concluded that movement of magnesium out of cell due to cellular injury specially to red blood cells and neurons leads to increased magnesium levels in body and also concluded that the increased magnesium has some protective action on the neuronal cells.

Sarici et al[38] concluded that bilirubin has a special affinity to phospholipids of the cell membrane like NMDA receptors and they can even get deposited on neuronal membrane and cause their damage. Magnesium has antagonizing action against NMDA receptor and acts against the toxic effect of bilirubin on nerve cells[38].

The hypomagnesaemia seen in the neonates after stopping phototherapy was asymptomatic and didn't require any active intervention for the same.

Frargy et al[7] made some recommendations in their study in order to prevent hypomagnesaemia like covering the head during PT in prevent the light from reaching the pineal gland which in turn prevents the decrease in melatonin level and about possible role of magnesium oral supplements to prevent hypomagnesemia.

In our study we could conclude that there is a 8.2% prevalence of hypomagnesemia after receiving phototherapy among term neonates receiving phototherapy. It was also concluded that there is statistically significant correlation between hypomagnesemia and duration of phototherapy

LIMITATION AND SCOPE OF THE STUDY

- As the study included limited population from single centre, results cannot be extrapolated to the whole population. A study with large sample size from different geographical areas should be conducted to have reliable results.
- Studies are required to ascertain the need for therapy with magnesium in neonates with high bilirubin values.
- As majority of the data available is from developed nations hence, there is need for study across multiple settings in developing countries.

CONCLUSION

The prevalence of hypomagnesaemia in our study after stopping phototherapy was 8.2% but all of the neonates were asymptomatic i.e none of them had any symptoms of hypomagnesaemia like seizures, tachycardia, nystagmus, arrhythmia, fasciculation etc.

In our study it was observed that the mean duration of phototherapy leading to development of hypomagnesaemia (i.e serum magnesium level <1.6) among neonates receiving phototherapy was higher than those who didn't develop hypomagnesaemia.

Our study concluded that there is positive correlation between the serum levels of bilirubin and higher level of magnesium at admission suggesting that the bilirubin toxicity possibly leads to lysis of cells and causes outward movement of magnesium to extracellular space. There will be a statistically significant fall in serum levels of both bilirubin and magnesium when compared, before and after stopping phototherapy.

SUMMARY

One year cross sectional study was conducted from September 2020 to September 2021 in the Department of Pediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 73 term newborns with neonatal jaundice were included in the study. The salient findings of the study are summarized below:

- During the study period a total of 73 full term neonates with neonatal jaundice were enrolled in the study.
- In our study 58.9% newborns were males and 41.1% were females.
- In our study out of 73 newborns, highest number of newborns were noted in the gestational age group of 38-39 weeeks i.e 58.9% and lowest in 40-41 weeks i.e 19.1%. About 43.8% were born by normal vaginal delivery and 56.2% by LSCS.
- The study also showed that A positive was common blood group(37.0%) noted among mothers of these newborns followed by O positive (34.2%).Similar character studied among newborns showed that B positive(30.1%) was common blood group followed by A positive (28.8%).
- Our study showed that the mean gestational age and mean weight of presentation of newborns was 38.47 weeks and 2.68kg respectively.
- The study highlights that the calcium value at the time of admission among the neonates was within normal limit with mean value being 9.13.
- The newborns presented with jaundice at a mean age of 2.4days and the mean duration of phototherapy required was 30.16hours.
- In our study it was observed that there is a statistically significant ($p < 0.001$) fall serum bilirubin values with fall in serum magnesium noted ($p < 0.001$.Blood

levels of serum magnesium and bilirubin was done at admission, after 12 hours , after 24 hours and at stopping phototherapy.

- Our study also observed that the prevalence of hypomagnesaemia after 24 hours of phototherapy was 2.7% and prevalence of hypomagnesaemia at stopping phototherapy was 8.2%. It was observed that prevalence of hypomagnesaemia increase with increase in duration of phototherapy ($p<0.002$)
- In our study it was observed that thought the prevalence of hypomagnesaemia was 8.2% none of the newborns had any clinical symptoms of hypomagnesaemia like convulsions, involuntary muscle contractions, involuntary eye movements, tachycardia etc.

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ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

"PREVALENCE OF PHOTOTHERAPY INDUCED HYPOMAGNESEMIA IN TERM NEWBORNS WITH JAUNDICE"

Principal Investigator:

Guide:

You are hereby requested to involve yourself and your baby in the above said research to be conducted at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from September 2020 to September 2021 by me.

Introduction

Neonatal jaundice is a common finding in neonates during the initial seven days of life . The yellowish discoloration of sclera and skin is because of deposition of unconjugated bilirubin. Phototherapy a commonly used and comparatively safer modality of treatment used to treat neonatal jaundice. Phototherapy can lead to few side effects including electrolyte imbalance like hypocalcaemia, hyponatremia, hypokalemia, dehydration, hyperthermia etc. There are very few studies showing hypomagnesaemia as a side effect following treatment with phototherapy.

Voluntary participation

Your baby's participation in this study is your voluntary decision. Whether to participate or not to participate will not affect your current or future relationship with the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You

are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research**.**

Risk and benefits

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is your valuable contribution to medical research.

Withdrawal from the study

You can withdraw at any time from the study. There will be no penalty for withdrawal.

Privacy and Confidentiality

The only people who will know that you are a research participant are member of the research team. No information provided by you, during research will be disclosed to others without your written consent. When the results of the research are published or discussed in the conferences, no information will be disclosed that would reveal your identity. Any information obtained in connections with this study and that can be identified with you remain confidential and will be disclosed only with your permission.

Queries

If you have any queries you may contact

Dr._____

Post Graduate Student

Department of Pediatrics

JNMC, Belagavi-590010

Phone No.

Dr. _____

MD (Pediatrics),

Professor, Department of Pediatrics

JNMC, Belagavi-590010

Phone No.

If you have any questions about your rights or research participation you may contact

Dr. Roopa Bellad,

Chairperson, Ethical Committee

JNMC Belagavi-590010

Phone No.9448113403

You will be given a copy of this form for your information and to keep for your record

STATEMENT OF CONSENT

I hereby voluntarily agree for my baby participation in this study. I understand that even if I choose to allow my baby to take part in this study I have the liberty to withdraw at any time. My signature below indicates that I have read or have been told about this entire consent form including the risks and benefits and have had all my questions answered. I will be given a copy of this consent form.

Signature of the authorized representative/ parent: _____

Date: _____

Name: _____

Relation to the Subject: _____

Signature of the witness: _____

Date: _____

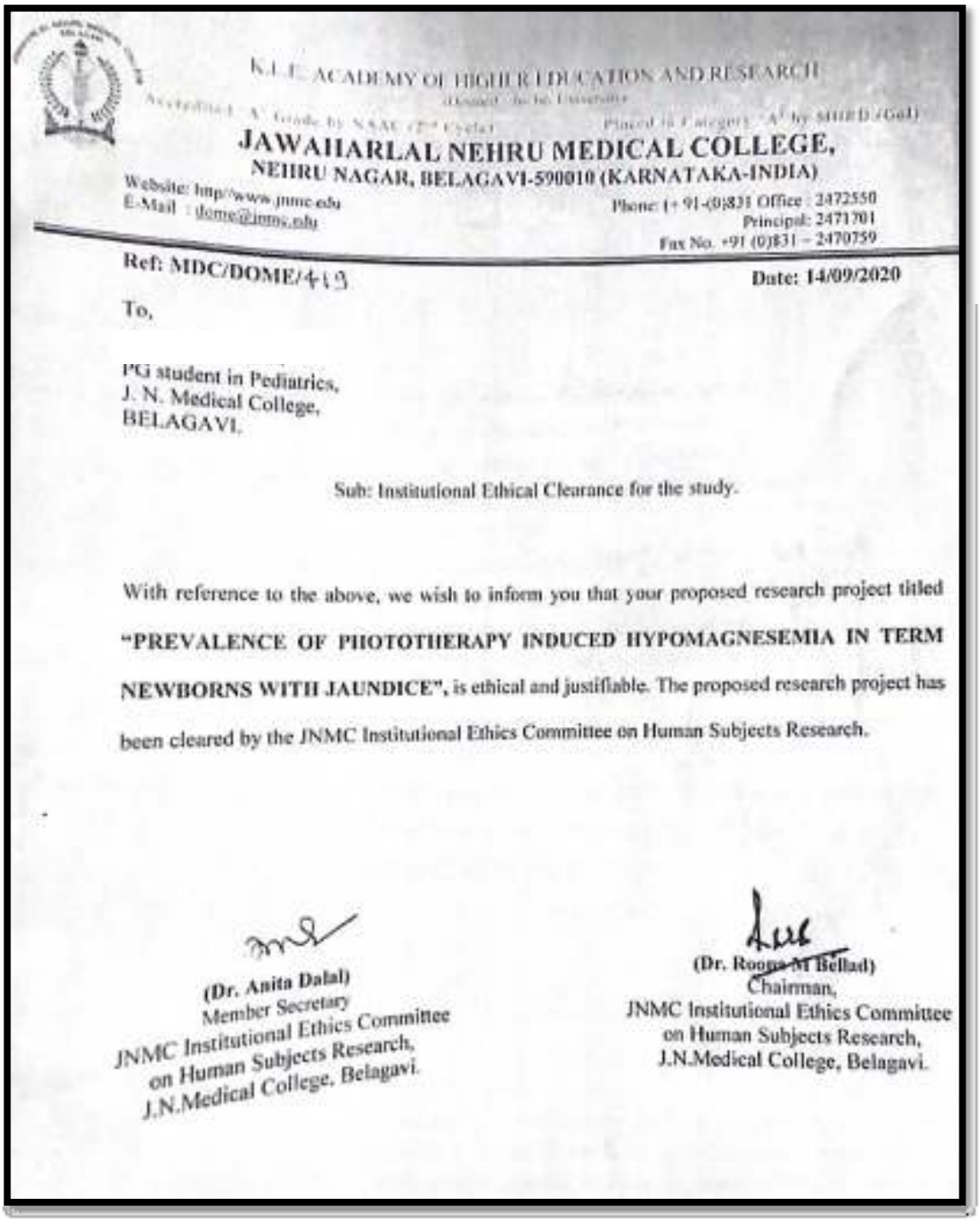
Name: _____

Signature of investigator: _____

Date: _____

Name: _____

ANNEXURES II – INSTITUTIONAL ETHICAL CLEARANCE



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed-to-be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http://www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/413 **Date: 14/09/2020**

To,

PG student in Pediatrics,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF PHOTOTHERAPY INDUCED HYPOMAGNESEMIA IN TERM NEWBORNS WITH JAUNDICE"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE III - PROFORMA

"PREVALENCE OF PHOTOTHERAPY INDUCED HYPOMAGNESEMIA IN TERM NEWBORNS WITH JAUNDICE"

Sl.no

1) Name-

2) Date of birth-

3) Gender-

4) Gestational age-

5) Mode of delivery-LSCS/Normal/Ventouse/Forceps

Indication for LSCS-

Maternal detail

6)Obstetric history-

7)Comorbidity-

8)Antenatal magnesium sulphate- Yes/No

9)Resuscitation-

10)APGAR score 1st min- 5th min-

11)Day of life at presentation-

12)Duration of phototherapy- hrs

13)Investigation

	At admission	At 12hrs	At 24hrs	After stopping
Serum bilirubin				
Serum magnesemia				
Baby blood group				
Calcium				

ANNEXURE IV - KEY TO MASTERCHART

CODING

GENDER

M- Male (1)

F-Female (2)

METHOD OF DELIVERY

NVD-Normal vaginal delivery (1)

LSCS- Lower segment caesarean section (2)

OBSTRETIC HISTORY

G- Gravida

P-Paraity

MATERNAL COMORBIDITIES

HTN- Hypertension

GDM- Gesatational diabetes mellitus

RESUCITATION

CIAB- Cried Immediately After Birth

Sl No	Name	DOB	Gender	Gestational age (weeks)		Mode of Delivery	Maternal details									Investigations												
																			At admission				After 12 hours			After 24 hours			After Stopping		
					Weight (kg)		Obstetric history	Comorbidity	Blood group	Antenatal magnesium sulphate	Resuscitation	APGAR at 1 min	APGAR at 5 min	Day of life at presentati	Duration of photother	Blood group	Calcium	total bilirubi	direct bilirubi	Serum magnesium	total bilirubim	direct bilirubi	Serum magnesium	Total bilirubi	direct bilirubi	Serum magnesium	Total bilirubi	direct bilirubi	Serum magnesium
1	B/O RENUKA PRAKASH	27-08-2020	1	38	2.4	2	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	8	2	28	O POSITIVE	9.1	15.8	0.36	2	13.42	0.38	1.8	11	0.28	1.7			
2	B/O RATNA NAIK	03-09-2020	1	38	2.36	2	G3P2L1D1	GESTATIONAL DM , HYPOTHYROIDISM	B POSITIVE	NO	CIAB	8	9	2	28	B POSITIVE	8.8	15.9	0.3	1.9	12.68	0.45	1.8	11.63	0.65	1.8			
3	B/O RENUKA	06-09-2020	1	38	2.5	2	G2P21		O POSITIVE	NO	CIAB	7	8	2	30	A POSITIVE	8.9	16.23	0.48	2.2	14.35	0.75	1.9	13.64	0.54	1.8	11.5	0.32	1.7
4	B/O SOUJANYA	07-09-2020	2	37	2.6	1	PRIMIGRAVIDA	HYPOTHYROIDISM	B POSITIVE	NO	CIAB	7	9	2	24	B POSITIVE	9	15.38	0.9	2.1	14.62	0.65	2	11.3	0.89	1.8			
5	B/O DEEPA PATIL	09-09-2020	1	37	2.8	1	G2P1L1	GESTATIONAL HTN	A POSITIVE	NO	CIAB	7	9	3	36	A POSITIVE	8.4	17.3	0.94	2	15.32	0.55	1.8	14.98	0.99	1.6			
6	B/O SAROJA	09-09-2020	2	38	2.9	2	G2P1L1	HYPOTHYROIDISM	B POSITIVE	NO	CIAB	7	9	2	28	AB POSITIVE	9.1	16.32	0.48	2.2	13.64	0.91	2.1	11.58	0.58	2.1			
7	B/O SUNANDHI JADHAV	11-09-2020	1	39	2.45	1	PRIMIGRAVIDA	GESTATIONAL HTN	A POSITIVE	NO	CIAB	7	9	2	26	A POSITIVE	9	15.62	0.93	1.9	14.8	0.69	1.9	11.53	0.86	1.7			
8	B/O LAXMI NIRANJAN	13-09-2020	2	40	3	2	G3P2L2	GESTATIONAL DM	B POSITIVE	NO	CIAB	7	9	2	28	AB POSITIVE	9.1	15.68	0.38	2	14.68	0.93	1.8	11.48	0.37	1.7			
9	B/O ANNAPURNA	15-09-2020	1	38	2.6	1	PRIMIGRAVIDA		B POSITIVE	NO	CIAB	8	9	2	28	AB POSITIVE	8.9	15.63	0.92	2.1	13.62	0.95	1.9	12.6	0.94	1.9			
10	B/O ANURADHA LINGAPPA	18-09-2020	1	40	2.5	2	G2P1L1		O POSITIVE	NO	CIAB	8	9	2	38	O POSITIVE	8.8	19.89	0.48	2.3	13.68	0.94	2.1	10.6	0.84	2	9.63	0.85	1.9
11	B/O TEJASWINI	22-09-2020	1	39	3.1	1	PRIMIGRAVIDA		O POSITIVE	NO	CIAB	8	9	3	36	O POSITIVE	9.2	16.82	0.93	2.1	14.84	0.54	2.1	13.4	0.82	1.9	11.32	0.93	1.8
12	B/O VIDYA	26-09-2020	2	38	2.7	1	PRIMIGRAVID	HYPOTHYROIDISM	A POSITIVE	NO	CIAB	7	8	3	32	O POSITIVE	9.2	16.3	0.64	2	12.87	0.67	1.6	11.4	0.64	1.5			
13	B/O YANKAVVA MANJAPPA	28-09-2020	1	40	2.3	1	G4P3L3	ANAEMIA	O POSITIVE	NO	CIAB	8	9	2	38	B POSITIVE	9.4	16.31	0.49	2	15.02	0.88	1.7	12.2	0.39	1.6	10.48	0.99	1.5
14	B/O SIDDHI GANESH	28-09-2020	2	37	2.9	1	G3P1L0A1		B POSITIVE	NO	CIAB	7	8	3	28	B POSITIVE	9.1	16.16	0.51	1.9	13.44	0.94	1.8	11.32	0.79	1.6			
15	B/O SAVITHA BABASAB	29-09-2020	2	38	2.5	2	PRIMIGRAVIDA	HYPOTHYROIDISM	A POSITIVE	NO	CIAB	6	8	3	28	B POSITIVE	9.6	17.99	0.51	2.2	14.01	0.58	1.8	11.4	0.77	1.8			
16	B/O DEEPALI LAXMAN	29-09-2020	1	37	2.8	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	8	2	24	A POSITIVE	9.4	17.5	0.44	2.1	13.77	0.67	1.8	11.86	0.85	1.8			
17	B/O SUNANDHA MALVALI	30-09-2020	1	39	2.4	1	PRIMIGRAVIDA	GESTATIONAL HYPERTENSION	B POSITIVE	NO	CIAB	8	9	3	24	B POSITIVE	9	17.32	0.38	2.2	15.87	0.85	2.1	12.32	0.54	1.8			
18	B/O SHAMSHAD	06-10-2020	2	39	2.6	1	PRIMIGRAVIDA	RH NEGATIVE PREGNANCY	A NEGATIVE	NO	CIAB	7	9	3	38	A NEGATIVE	9.3	16.89	0.48	2	16.84	0.56	1.8	13.64	0.58	1.7	10.82	0.34	1.5
19	B/O CHAITRA HIREMATH	29-10-2020	1	40	2.8	1	G2A1	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	2	28	A POSITIVE	9.2	16.34	0.48	1.9	12.9	85	1.7	11.68	0.98	1.5			
20	B/O KIRTI MANI	29-10-2020	1	37	2.4	2	G3P1L1A1	GESTATIONAL HTN	O POSITIVE	NO	CIAB	7	9	3	34	O POSITIVE	9.3	16.8	0.5	2.2	14.78	0.68	2	12.48	0.46	1.7	11.5	0.88	1.6
21	B/O SAVITHA ANIL	31-10-2020	1	38	2.4	1	G4P2L2A1	ECLAMPSIA	B POSITIVE	NO	CIAB	8	10	2	24	AB POSITIVE	9.4	17.03	0.32	2.2	13.62	0.99	1.9	11.8	0.84	1.8			
22	B/O VITHATAI GURAV	04-11-2020	1	39	3.1	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	9	2	30	O POSITIVE	9.1	16.88	0.23	2.1	12.1	0.46	2	11.58	0.68	1.9	10.4	0.65	1.7
23	B/O SHILPA NILAJAGI	04-11-2020	2	37	3.2	2	G3P1L1A1	GESTATIONAL DM	A POSITIVE	NO	CIAB	8	9	2	32	O POSITIVE	9.2	18.6	1.01	2.2	13.82	0.92	2.1	12.7	0.78	1.7	11.48	0.97	1.6
24	B/O LAXMI NAIK	05-11-2020	2	37	3	2	G3P2L2	HYPOTHYROIDISM	AB POSITIVE	NO	CIAB	7	9	3	34	B POSITIVE	9.6	18.99	0.9	2.2	13.92	0.8	2.1	12.32	0.69	1.9	10.08	0.64	1.7
25	B/O SUNITHA SHEKAR	05-11-2020	2	41	2.9	2	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	8	9	4	32	A POSITIVE	8.4	17.68	0.94	2.2	14.68	1	2	13.6	0.32	1.8	11.4	0.48	1.6
26	B/O SATTEVVA PATIL	09-11-2020	2	37	2.7	2	G3P2L1D1	GESTATIONAL HTN	A POSITIVE	NO	CIAB	8	9	3	38	O POSITIVE	9	17.29	0.22	2.1	15.28	0.38	2	13.4	0.65	1.9	11.8	0.78	1.8
27	B/O DEEPA KATAGI	10-11-2020	1	41	2.4	2	PRIMIGRAVIDA	GESTATIONAL DM , HYPOTHYROIDISM	A POSITIVE	NO	CIAB	8	10	3	32	A POSITIVE	9.6	17.66	1.01	2.3	14.7	0.87	2	12.68	0.84	1.8	10.4	0.85	1.7
28	B/O SANGEETHA SARAPNA	10-11-2020	2	39	2.9	2	PRIMIGRAVIA		O POSITIVE	NO	CIAB	8	9	3	24	A POSITIVE	9.2	18.32	0.99	2.3	13.84	0.64	2.1	10.4	0.65	1.8			
29	B/O VANDANA GANESH	12-11-2020	2	38	3.1	2	G3P1L1		A POSITIVE	NO	CIAB	9	10	2	32	AB POSITIVE	9.2	15.86	0.42	2	13.42	0.4	1.8	12.08	0.61	1.6	10.1	0.78	1.5
30	B/O GEETHA HIREMATH	13-11-2020	1	38	3	2	G2P1L1		AB POSITIVE	NO	CIAB	8	9	2	24	A POSITIVE	9.1	18.8	0.33	2.3	13.07	0.48	2.1	11.6	0.56	1.8			
31	B/O KAVERI PATIL	14-11-2020	2	38	2.8	2	PRIMIGRAVIA		O POSITIVE	NO	CIAB	8	9	3	24	B POSITIVE	9.1	16.44	0.83	2	13.1	0.63	1.8	11.5	0.45	1.7			
32	B/O SAVITHA BASAPPA	15-11-2020	2	40	2.9	2	G3P1L1A1		O POSITIVE	NO	CIAB	8	9	2	28	B POSITIVE	9	15.59	0.45	2.1	14.07	0.58	2.1	11.8	0.55	1.8			
33	B/O SWETHA SUBASH	15-11-2020	1	40	2.7	2	PRIMIGRAVIDA		B POSITIVE	NO	CIAB	8	10	3	34	AB POSITIVE	9	16.5	0.94	2	14.64	0.86	1.9	12.5	0.89	1.8	11.28	0.42	1.7
34	B/O SAVITHRI SHIVANAND	17-11-2020	2	38	2.9	2	G2P1L1		O POSITIVE	NO	CIAB	8	10	2	38	O POSITIVE	9.4	17.24	0.96	2.2	14.82	0.68	2.1	12.4	0.87	1.8	10.24	0.54	1.6
35	B/O LAXMI PATIL	19-11-2020	2	39	2.4	1	G5P3L3A1		A POSITIVE	NO	CIAB	9	10	2	38	A POSITIVE	9	18.66	0.88	2.3	13.74	0.38	2.1	12.23	0.85	2.1	11.25	0.48	1.7
36	B/O DEEPTHI RAJU	19-11-2020	2	40	2.6	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	9	2	28	O POSITIVE	8.9	17.6	0.86	2.1	12.81	0.45	2	10.23	0.56	1.9			
37	B/O GOUSABI SHANU	19-11-2020	2	39	2.3	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	9	10	3	38	A POSITIVE	8.9	16.42	0.89	2	14.61	0.32	1.9	13.15	0.59	1.8	12.5	0.67	1.6
38	B/O RADHA BIRADHAR	20-11-2020	1	39	2.7	1	G2P1L1		O POSITIVE	NO	CIAB	8	10	2	22	O POSITIVE	9	16.77	1.01	2	14.14	0.36	1.9	11.6	0.42	1.7			
39	B/O SHILA PARASU	20-11-2020	1	38	2.5	2	PRIMIGRAVIDA		B POSITIVE	NO	CIAB	8	9	3	22	AB POSITIVE	9.6	18.99	0.93	2.3	12.65	0.37	2.1	10.4	0.59	2			
40	B/O ANJUM BEPARI	21-11-2020	1	38	2.4	2	PRIMIGRAVIDA	GESTATIONAL HTN	O POSITIVE	NO	CIAB	9	10	3	24	O POSITIVE	9.6	16.03	0.3	2	13.36	0.48	1.8	11.64	0.83	1.7			
41	B/O BHAGYASHREE PRAVEEN	22-11-2020	2	39	2.3	1	G2A1	GESTATIONAL HTN	A POSITIVE	NO	CIAB	9	10	2	22	B POSITIVE	9	16.88	0.92	2.1	14.86	0.93	1.8	12.65	0.75	1.6			
42	B/O ANJALI SAWANT	23-11-2020	2	39	2.9	2	PRIMIGRAVIDA	PLACENTA PREVIA	A POSITIVE	NO	CIAB	7	9	3	38	A POSITIVE	9.1	15.9	0.26	2.1	13.32	0.4	2	12.8	0.36	1.9	11.5	0.85	1.8
43	B/O ASHWINI SAJJAN	24-11-2020	1	38	2.6	2	PRIMIGRAVIDA	GESTATIONAL HTN	B POSITIVE	NO	CIAB	9	10	2	24	B POSITIVE	9	15.86	0.83	2	12.55	0.65	1.9	10.4	0.68	1.7			
44	B/O BHAGYASHREE RAJ	29-11-2020	2	38	2.8	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	9	2	38	B POSITIVE	9.8	16.48	0.55	2.2	13.5	0.39	2.2	12.63	0.93	2	11.05	0.94	1.8
45	B/O SUDHA ARYAN	30-11-2020	1	37	2.9	2	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	9	2	24	O POSITIVE	9.1	15.84	0.87	2.1	11.51	0.52	2	10.63	0.82	2			

46	B/O SUNITHA PATTU	02-12-2020	1	40	3.1	1	G3P2L1D1 WITH CEPHALIC	HYPOTHYROIDISM	A POSITIVE	NO	CIAB	8	9	2	38	O POSITIVE	9.4	16.83	0.91	2.3	12.05	0.78	2	11.16	0.58	1.9	10.32	0.74	1.7
47	B/O ASHWINI SAJJAN	03-12-2020	1	38	2.9	2	PRIMIGRAVIDA		B POSITIVE	NO	CIAB	8	9	2	34	AB POSITIVE	9.3	16.22	0.92	2.2	13.61	0.75	1.9	12.77	0.51	1.8	10.68	0.36	1.6
48	B/O ANJALI PARIT	04-12-2020	1	40	2.4	1	G2P1L1		O POSITIVE	NO	CIAB	8	10	2	24	B POSITIVE	8.9	17.6	0.92	2.2	13.62	0.89	2	12.5	0.82	2			
49	B/O SONALI SUNIL	06-12-2020	1	40	2.6	2	PRIMIGRAVIDA		O POSITIVE	NO	CIAB	9	10	3	36	O POSITIVE	9.3	19.03	0.93	2.4	13.78	0.51	2	11.8	0.84	1.8	10.89	0.64	1.7
50	B/O BIBIAYESHA IMRAN	10-12-2020	1	39	2.8	2	G4P3L3		A POSITIVE	NO	CIAB	9	10	2	28	O POSITIVE	9.1	18.3	0.93	2.3	12.7	0.55	2	11.05	0.45	1.8			
51	B/O USHA	23-12-2020	2	40	2.4	2	PRIMIGRAVIDA	GESTATIONAL HTN	B POSITIVE	NO	CIAB	9	10	2	24	B POSITIVE	9.3	16.69	0.96	2.1	14.82	0.64	2	12.7	0.53	1.8			
52	B/O VISHALAXMI RAV	24-12-2020	1	37	2.6	2	G2P1L0		A POSITIVE	NO	CIAB	7	9	2	28	AB POSITIVE	9.4	15.62	0.83	1.9	11.1	0.88	1.8	10.64	0.89	1.6			
53	B/O NAGARATNA	25-12-2020	2	39	2.8	1	PRIMIGRAVIDA	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	3	26	O POSITIVE	9	16.32	0.62	2	12.64	0.94	1.9	10.84	0.63	1.8			
54	B/O LAXMI SHANKAR	26-12-2020	2	37	2.3	2	G2P1L0D1	GESTATIONAL DM	O POSITIVE	NO	CIAB	8	9	2	28	A POSITIVE	9.3	17.1	0.94	2.1	13.97	0.44	1.9	10.3	0.97	1.8			
55	B/O LAXMI GANGAPPA	29-12-2020	1	38	2.8	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	8	9	3	38	A POSITIVE	9	18.63	0.93	2.2	16.26	0.68	2.1	14.58	0.93	2.1	12.68	0.64	1.8
56	B/O BIBIAYESHA	02-01-2021	1	39	2.9	2	PRIMIGRAVIDA		O POSITIVE	NO	CIAB	7	9	2	36	B POSITIVE	8.8	16.82	0.92	1.9	15.68	0.88	1.8	13.58	0.48	1.7	10.02	0.81	1.5
57	B/O ALFIYA TOUSIK	05-01-2021	2	37	2.5	1	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	7	8	2	32	A POSITIVE	9.2	17.4	0.93	2.3	14.32	0.45	2.1	12.56	0.61	1.8	11.5	0.77	1.7
58	B/O NAGARATNA	08-01-2021	1	37	2.4	1	G2P1L1	GESTATIONAL HTN	A POSITIVE	NO	CIAB	7	9	2	26	B POSITIVE	9.1	16.3	0.63	2	14.62	0.78	1.8	11.89	0.94	1.7			
59	B/O NUTHAN	09-01-2021	2	39	2.2	2	G2P1L1	GESTATIONAL THROMBOCYTOPENIA	O POSITIVE	NO	CIAB	7	10	2	30	A POSITIVE	9	16.62	0.83	2	14.88	0.69	1.8	12.4	0.68	1.8			
60	B/O PRAMITKAUR	13-01-2021	1	40	2.3	1	PRIMIGRAVIDA	GESTATIONAL HTN	O POSITIVE	NO	CIAB	8	10	3	28	O POSITIVE	9.2	17.32	0.68	2.3	15.44	0.59	2	12.05	0.81	1.8			
61	B/O MANJULA	22-01-2021	1	39	3.1	1	G3P2L2		B POSITIVE	NO	CIAB	8	9	3	28	AB POSITIVE	9.6	17.03	0.89	1.9	15.36	0.44	1.8	12.01	0.64	1.6			
62	B/O SAVITHRI	02-02-2021	1	37	2.9	2	PRIMIGRAVIDA	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	8	2	36	B POSITIVE	9.1	17.23	0.84	2	15.68	0.94	1.8	14.04	0.62	1.6	11.8	0.92	1.5
63	B/O BHARATHI	04-02-2021	2	37	2.4	2	PRIMIGRAVIDA	GESTATIONAL HTN	AB POSITIVE	NO	CIAB	8	9	2	34	B POSITIVE	8.6	16.9	0.93	2.1	14	0.83	1.9	13.66	0.48	1.8	10.68	0.32	1.7
64	B/O BHIAVVA	05-02-2021	1	39	2.4	2	G2P2L0	GESTATIONAL DM	A POSITIVE	NO	CIAB	8	9	2	38	AB POITIVE	8.8	15.86	0.92	1.9	14.92	0.64	1.7	12.48	0.28	1.7	10.5	0.51	1.5
65	B/O VEENA HAYAL	05-02-2021	1	37	2.3	2	PRIMIGRAVIDA	GESTATIONAL HTN,HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	2	28	A POSITIVE	9	16.52	0.93	2	15.92	0.59	1.8	12.1	0.44	1.7			
66	B/O SUNITHA KIRAN	08-02-2021	1	38	2.8	2	G2P1L1	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	3	32	A POSITIVE	9.1	16.86	0.92	2.2	15.82	0.99	2.1	12.86	0.97	1.9	11.56	0.84	1.8
67	B/O ASHWINI PATIL	25-02-2021	1	38	3.1	1	PRIMIGRAVIDA	GESTATIONAL DM	AB POSITIVE	NO	CIAB	7	8	3	30	A POSITIVE	9.4	17.02	0.98	2	14.68	0.58	1.8	12.8	0.48	1.7	10.8	0.94	1.7
68	B/O SHOBHA PATIL	01-03-2021	1	39	3	1	G2P1L1		B POSITIVE	NO	CIAB	8	9	2	24	B POSITIVE	9.1	15.84	0.92	2	13.08	0.43	1.9	12.62	0.44	1.7			
69	B/O RUKSAR	07-03-2021	2	38	2.8	2	G2P1L1	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	2	28	A POSITIVE	9.1	18.04	0.82	2.4	14.63	0.82	2.1	12.2	0.86	2			
70	B/O SHARADHA INGALE	23-03-2021	1	38	2.6	1	PRIMIGRAVIDA		AB POSITIVE	NO	CIAB	7	8	3	24	B POSITIVE	9.4	17.68	0.42	2.3	16.84	0.92	2.1	12.09	0.85	1.8			
71	B/O FATHIMA SHAIKH	25-03-2021	1	38	2.5	2	PRIMIGRAVIDA		A POSITIVE	NO	CIAB	8	10	3	38	B POSITIVE	9	17.03	0.71	2	15.82	0.68	2	14.05	0.56	1.9	11.62	0.75	1.7
72	B/O NIKITA PATIL	26-03-2021	1	39	2.6	1	PRIMIGRAVIDA	HYPOTHYROIDISM	O POSITIVE	NO	CIAB	8	9	2	28	B POSITIVE	8.9	16.23	0.88	2.1	15.32	0.42	2	12.5	0.49	1.8			
73	B/O SEEMA	29-03-2021	2	39	2.8	2	PRIMIGRAVIDA		O POSITIVE	NO	CIAB	8	9	2	30	A POSITIVE	8.9	17.05	0.48	2.2	15.62	0.38	2	11.9	0.68	1.7			