# "RISK FACTORS, SEVERITY AND OUTCOME AMONG NEONATES WITH THROMBOCYTOPENIA ADMITTED IN NEONATAL INTENSIVE CARE UNIT"

By

**Dr. ANKEM PRAVEEN**

**DISSERTATION SUBMITTED TO**

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

**IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF**

**DOCTOR OF MEDICINE**
**IN**
**PAEDIATRICS**

**Under the Guidance of**
**Dr. SUDHA REDDY V R**
**Professor and HOD,**
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**SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA**
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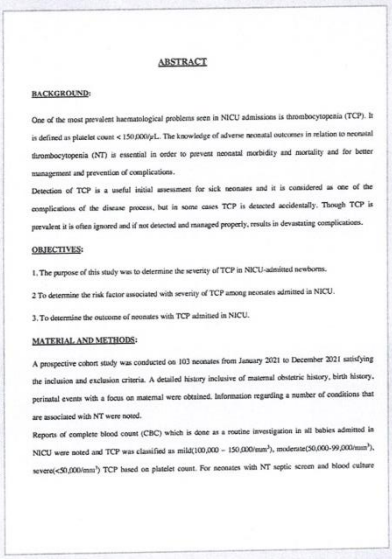

**ABSTRACT**

**BACKGROUND:**

One of the most prevalent haematological problems seen in NICU admissions is thrombocytopenia (TCP). It is defined as platelet count < 150,000/μL. The knowledge of adverse neonatal outcomes in relation to neonatal thrombocytopenia (NT) is essential in order to prevent neonatal morbidity and mortality and for better management and prevention of complications.

Detection of TCP is a useful initial assessment for sick neonates and it is considered as one of the complications of the disease process, but in some cases TCP is detected accidentally. Though TCP is prevalent it is often ignored and if not detected and managed properly, results in devastating complications.

**OBJECTIVES:**

1. The purpose of this study was to determine the severity of TCP in NICU-admitted newborns.

2 To determine the risk factor associated with severity of TCP among neonates admitted in NICU.

3. To determine the outcome of neonates with TCP admitted in NICU.

**MATERIAL AND METHODS:**

A prospective cohort study was conducted on 103 neonates from January 2021 to December 2021 satisfying the inclusion and exclusion criteria. A detailed history inclusive of maternal obstetric history, birth history, perinatal events with a focus on maternal were obtained. Information regarding a number of conditions that are associated with NT were noted.

Reports of complete blood count (CBC) which is done as a routine investigation in all babies admitted in NICU were noted and TCP was classified as mild(100,000 – 150,000/mm$^3$), moderate(50,000-99,000/mm$^3$), severe(<50,000/mm$^3$) TCP based on platelet count. For neonates with NT septic screen and blood culture

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# ACKNOWLEDGEMENT

I would like to thank God for giving me the opportunity, strength, courage and Blessings. I express my deep sense of gratitude and sincere thanks to my guide **Dr. Sudha Reddy.V.R.,** HOD and Professor, Department of Paediatrics, Sri Devaraj Urs Medical College, Kolar for her encouragement and guidance for successful completion by her constant support, wise constructive judgment the painstaking effort to weed out errors and advice.

I would like to express my deep sense of gratitude and humble thanks to Professors **Dr. Krishnappa J**, **Dr. K.N.V. Prasad**, yours passion for the subject always inspired me. Thanks for the guidance and encouragement.

I express my deep sense of gratitude and humble thanks to **Dr. Beere Gowda Y C**, Professor, for your advice and encouragement and support throughout post-graduation.

My heartfelt thanks to **Dr. Bhanuchand P, Dr. Narendra k k, Dr. James Daniel, Dr. Srikanth, Dr. Naveen kumar, Dr. Karthik** faculty in Department of Paediatrics for their guidance, encouragement and support.

I would like to thank my Senior residents **Dr. Abhinay, Dr. Abhilash** for their guidance and support.

I extend my sincere thanks to all seniors **Dr Srinadh, Dr Chinthana, Dr Akshatha, Dr Vidyashree, Dr Niranjan reddy, Dr Rajitha reddy, Dr Pravallika, Dr Sanjana reddy** 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 Jefrin Anto, Dr Chevva Prakash Reddy Dr Trisali Padala, Dr Bindu T, Dr Nikitha venkiteela, Dr Mathumitha T** for their support and love.

Heartfelt thanks to my juniors **Dr Jahnavi, Dr Kamalakar, Dr Saiteja, Dr Ramswaroop, Dr Rana**, **Dr Mouna, Dr Karthik.**

I would express my deepest gratitude to my beloved parents **Ankem Radhakrishna, Saraswathi A** for constantly believing in me and whose love, blessings and sacrifices made me the person what I am today. Especially my father who has supported me throughout.

I would like to thank my brothers **Ankem Kiran, Prakash P** for all their love and support. They have been my pillars throughout my post graduation and without whom this journey would have been impossible.

Special thanks to **Dr Likhitha Krishna** and **Monalisa M** for their love and constant support.

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 of NICU and the babies who were part of this study without whose support this study wouldn't have been possible.

**Date**:

**Place**: Kolar **Dr. Ankem Praveen**

# LIST OF ABBREVIATIONS

| GLOSSARY | ABBREVIATIONS |
|---|---|
| **Aptt** | Activated partial thromboplastin time |
| **BA** | Birth asphyxia |
| **BT** | Bleeding time |
| **CBC** | Complete blood count |
| **CT** | Clotting time |
| **DIC** | Disseminated intravascular coagulation |
| **E coli** | Escherichia coli |
| **EOT** | Early onset thrombocytopenia |
| **GDM** | Gestational diabetes mellitus |
| **GNB** | Gram negative bacilli |
| **GPC** | Gram positive cocci |
| **HIT** | Heparin induced thrombocytopenia |
| **IEM** | Inborn errors of metabolism |
| **IgG** | Immunoglobulin G |
| **INR** | International normalized ratio |
| **IUGR** | Intrauterine growth retardation |
| **IVH** | Intraventricular haemorrhage |
| **LBW** | Low birth weight |
| **LOT** | Late onset thrombocytopenia |

| | |
|---|---|
| **MAS** | Meconium aspiration syndrome |
| **MAS** | Meconium aspiration syndrome |
| **MSAF** | Meconium stained amniotic fluid |
| **NAT** | Neonatal alloimmune thrombocytopenia |
| **NEC** | Necrotizing enterocolitis |
| **NICU** | Neonatal intensive care unit |
| **NT** | Neonatal thrombocytopenia |
| **PIH** | Pregnancy induced hypertension |
| **PPH** | Persistent pulmonary hypertension |
| **PROM** | Premature rupture of membranes |
| **PT** | Prothrombin time |
| **RDS** | Respiratory distress syndrome |
| **SLE** | Systemic lupus erythematosus |
| **TCP** | Thrombocytopenia |
| **TLC** | Total leucocyte count |
| **WBC** | White blood cell |

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# ABSTRACT


**BACKGROUND:**

One of the most prevalent haematological problems seen in NICU admissions is thrombocytopenia (TCP). It is defined as platelet count < 150,000/µL. The knowledge of adverse neonatal outcomes in relation to neonatal thrombocytopenia (NT) is essential in order to prevent neonatal morbidity and mortality and for better management and prevention of complications.

Detection of TCP is a useful initial assessment for sick neonates and it is considered as one of the complications of the disease process, but in some cases TCP is detected accidentally. Though TCP is prevalent it is often ignored and if not detected and managed properly, results in devastating complications.

**OBJECTIVES:**

1. The purpose of this study was to determine the severity of TCP in NICU-admitted newborns.
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**MATERIAL AND METHODS:**

A prospective cohort study was conducted on 103 neonates from January 2021 to December 2021 satisfying the inclusion and exclusion criteria. A detailed history inclusive of maternal

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Reports of complete blood count (CBC) which is done as a routine investigation in all babies admitted in NICU were noted and TCP was classified as mild(100,000 – 150,000/$mm^3$), moderate(50,000-99,000/$mm^3$), severe(<50,000/$mm^3$) TCP based on platelet count. For neonates with NT septic screen and blood culture investigations were done. Other investigations such as coagulation profile, chest X-ray, neurosonogram (NSG) and computed tomography (CT) brain were performed whenever the need arose.

The various maternal and neonatal risk factors causing NT were assessed and a p value of $\leq$ 0.05 was considered to be significant, outcome of neonates with NT was studied and explored.

**RESULTS**:

Our study included 103 neonates who met the inclusion criteria. The present study showed an almost equal distribution among both males and females. Majority of neonates had mild TCP (68.0%). Moderate and severe TCP were present in 27.2% and 4.9% respectively. Various maternal risk factors were present such as PIH (56.3%), drug intake (35.0%), PROM (20.4%), anaemia (14.6%), GDM (8.7%) and Rh incompatibility (2.9%) even though non-significant. Majority of neonates (48.5%) had sepsis, 25.2% had RDS, 3.9% had DIC and 3.9% had NEC even though non-significant.

The mean duration of hospital stay in mild, moderate and severe TCP is 15.75 ± 8.10 days, 15.14 ± 11.78 days and 16.25 ± 3.30 days respectively. Platelet transfusion was done in 8.7% neonates. A mortality of 8.7% was seen in the study.

## CONCLUSION:

Thrombocytopenia (TCP) is a haematological condition affecting most of the neonates admitted to NICU and can have serious consequences. Mild TCP was most common than moderate and severe TCP. Preterm neonates had TCP more compare to term neonates. PIH and maternal drug intake were the commonest maternal risk factors. Sepsis and respiratory distress syndrome (RDS) were the commonest neonatal risk factors associated with TCP. Platelet transfusion was needed by most of the severe TCP cases. Although non-significant, the mean duration of stay in severe TCP was the highest. A mortality of 8.7% was noticed in the study.

# INTRODUCTION

# INTRODUCTION

One of the most prevalent haematological problems seen in NICU admissions is thrombocytopenia (TCP).[1] A platelet count of less than 150,000/µL is the clinical definition of neonatal thrombocytopenia (NT). There are two types of thrombocytopenia, early-onset (EOT) and late-onset (LOT), which manifest within the first 72 hours and after 72 hours of a newborn's life respectively. Mild TCP (platelet count of 100,000-150,000/µL), moderate TCP (50,000-99,000/µL), and severe TCP (<50,000/µL) are defined according to the severity of the disease.[1]

Sepsis, birth asphyxia (BA), premature birth, intrauterine growth retardation (IUGR), respiratory distress syndrome (RDS), hyperbilirubinemia, meconium aspiration syndrome (MAS), and low birth weight (LBW) are all risk factors for TCP in neonates.[2,3,4]

The most common cause of EOT is intrauterine growth restriction (IUGR), which is connected with prenatal conditions such maternal illness or placental insufficiency. In contrast, sepsis or necrotizing enterocolitis (NEC) are the root causes of LOT.[3]

TCP is usually diagnosed at birth or within the first 2-3 days after delivery in pregnancies complicated by prenatal hypertension, pre-eclampsia, and eclampsia syndrome, and usually resolves by day 10.

Only a small fraction of newborns with preeclampsia may develop severe or clinically significant thrombocytopenia, defined as a platelet count(<50,000/µL).[4]

Although NT is often mild, it can occasionally be severe and even fatal. A major risk is bleeding, especially bleeding in the brain (Intra cranial haemorrhage) or lungs (Pulmonary haemorrhage). Infants born prematurely or with extremely low birth weight are at a higher

risk for severe TCP and serious haemorrhage. The outcome is determined by the root cause.[3,5,6]

There has been a lot of study on the causes, clinical characteristics, and treatment of NT in NICUs over the past decade.[7-8]

It is unclear how much of an impact TCP has on neonatal outcomes because the topic has not been investigated previously. Neither has an article evaluated NT worth as a prognostic biomarker in infants with illness.

In order to reduce the risk of neonatal morbidity and mortality and improve the care and prevention of complications, it is crucial to have an understanding of the outcomes that might go wrong throughout neonatal development as a result of NT. The detection of TCP is an important first step in evaluating the health of newborns, as it is a known consequence of the underlying illness process. Despite TCP widespread presence, it is often overlooked, leading to the disastrous issues that arise when it is not recognised and addressed. [9]

We sought to characterise the risk factors, severity, and outcome of neonates with TCP admitted to our hospital's NICU due to the dearth of research from India and the rising incidence of this ailment.

# AIMS & OBJECTIVES

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1. The purpose of this study was to determine the severity of TCP in NICU-admitted newborns.
2. To determine the risk factor associated with severity of TCP among neonates admitted in NICU.
3. To determine the outcome of neonates with TCP admitted in NICU.

# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

One of the most prevalent haematological issues in newborn critical care units is TCP (platelet count <1,50,000/µL) (NICUs). It has been found that newborns hospitalised to neonatal critical care units had a substantially greater prevalence of TCP, ranging from 22 to 35%, than neonates generally, which varies from 1 to 5%. Preterm or unwell newborns in NICUs as well as those with exceptionally low birth weights are more likely to experience it[7]. Only 2% of healthy newborns have TCP at birth, and less than 3/1000 term infants have severe TCP (platelet count <50,000/µL)[8].

Low platelet counts in newborns, both preterm and term, are a frequent occurrence. TCP affects 0.7–4% of all newborns. Up to 22% of all neonates hospitalised to the NICUs experience low platelet counts, according to estimates. Regardless of the patient's age, TCP is defined as a platelet count less than 150,000/µL[10].

Approximately 8%–10% of all pregnancies are complicated by hypertensive problems[11]. Important maternal and neonatal morbidity and death are caused by hypertensive diseases. The most prevalent type of hypertension-related fatality is intracranial bleeding. The risk to the mother and foetus increases with a decreased platelet count[11].

Early-onset NT typically appears within the first 72 hours of birth and is associated with complications during pregnancy such as IUGR, maternal diabetes, maternal immune thrombocytopenic purpura (ITP), congenital infections, or neonatal alloimmune thrombocytopenia (NAT). Late-onset NT, which presents after the first 72 hours of life and is usually caused by NEC or sepsis, is frequently more severe and persistent than early-onset NT.[12].

**MEGAKARYOPOIESIS:**

Platelets, which are made from the cytoplasm of megakaryocytes, are small anucleate fragments that have a discoid form. "The production of megakaryocytes from stem cells is known as megakaryopoiesis, while the differentiation of megakaryocytes into platelets is known as thrombopoiesis (Fig.1). Platelet synthesis in a foetus begins in the yolk sac and, along with the rest of hematopoiesis, moves to the foetal liver and, ultimately, the bone marrow.[13,14.]

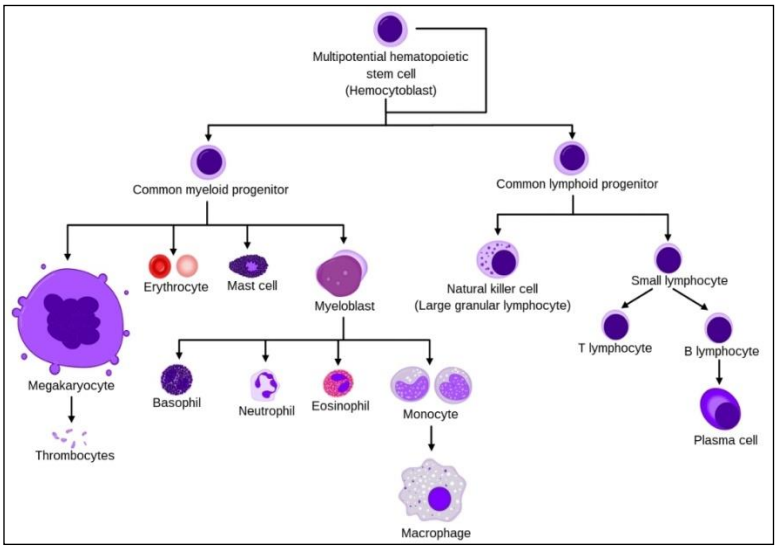


Figure No- 1: Flow diagram showing megakaryopoiesis through various steps

NT can result from a variety of diseases in newborns. Sepsis, birth asphyxia (BA), preterm, IUGR, hyperbilirubinemia, RDS, MAS, and LBW are the main causes of TCP in newborns. Bleeding signs rely on underlying conditions in addition to platelet numbers[15].

The two primary pathogenic mechanisms for NT are either enhanced destruction/sequestration or reduced platelet formation. The natural history of the TCP and the date of its development may frequently be used to anticipate the underlying aetiology of this problem[16]. Most cases of TCP are mild to moderate and improve without treatment.

Severe cases may result in intra ventricular haemorrhage (IVH), pulmonary haemorrhage, or death.[17].

**Neonatal thrombocytopenia(NT):**

Platelets are disc shaped with an average volume of 75/µm circulate in the blood and are 14 times smaller than erythrocytes.[18] Platelets are fragments of megakaryocytes that have been anucleated. When compared to the mean platelet count of 187,000/µL at 15 weeks of gestation, the average number of platelets in a foetus at 40 weeks of gestation is 274,000/µL. The data shows that while preterm infants often have lower platelet counts than full-term infants, they are still within the normal range for children and adults (between 150,000/µL and 450,000/µL). The following definition of TCP applies to both preterm and term infants: Platelet Count: <150,000/µL[14,19].

**Kinetic mechanisms of NT:**

As in adults, TCP in newborns can be caused by any one of three type of mechanisms: (i) decreased platelet generation, (ii) enhanced platelet breakdown, (iii) platelet sequestration (most commonly caused by hypersplenism), or (iv) a combination of these processes. The kinetic mechanism causing the TCP in the majority of affected neonates is unknown, because there are so many different disorders can cause TCP during neonatal period and in part because it is challenging to perform the "gold standard" tests on small, sick neonates[14].

**Causes of NT:**

1. **Increased destruction**

**a. Maternally associated**

Common:

i) Immune mediated

- Alloimmune NT
- Maternal autoimmune disorders (ITP: immune thrombocytopenic purpura, systemic lupus erythematosus)

ii) Congenital infections

Uncommon:

- Maternal preclampsia
- Maternal drug intake (e.g. quinidine, certain anticonvulsants, certain diuretics)
- Rh incompatibility
- Placental abnormalities

**b. Not maternally associated**

Common :

- Infections
- Birth asphyxia (BA)
- Neonatal thrombosis (associated with indwelling catheters, coagulation abnormalities)

Uncommon:

- RDS
- Congenital heart disease
- Hemangiomas
- Hypersplenism

Rare:

- Wiskott–Aldrich syndrome
- Giant platelet syndromes (Bernard–Soulier, May–Hegglin, Mediterranean macrothrombocytopenia)
- Von Willebrand type IIb
- IEM

**2. Decreased production or bone marrow replacement**

Uncommon:

- Trisomy syndromes (13, 18)
- TCP with absent radii

Rare:

- Amegakaryocytic thrombocytopenia
- Fanconi's anemia, dyskeratosis congenita
- Congenital leukemia

**DIAGNOSIS OF NT (Fig-2) :-**

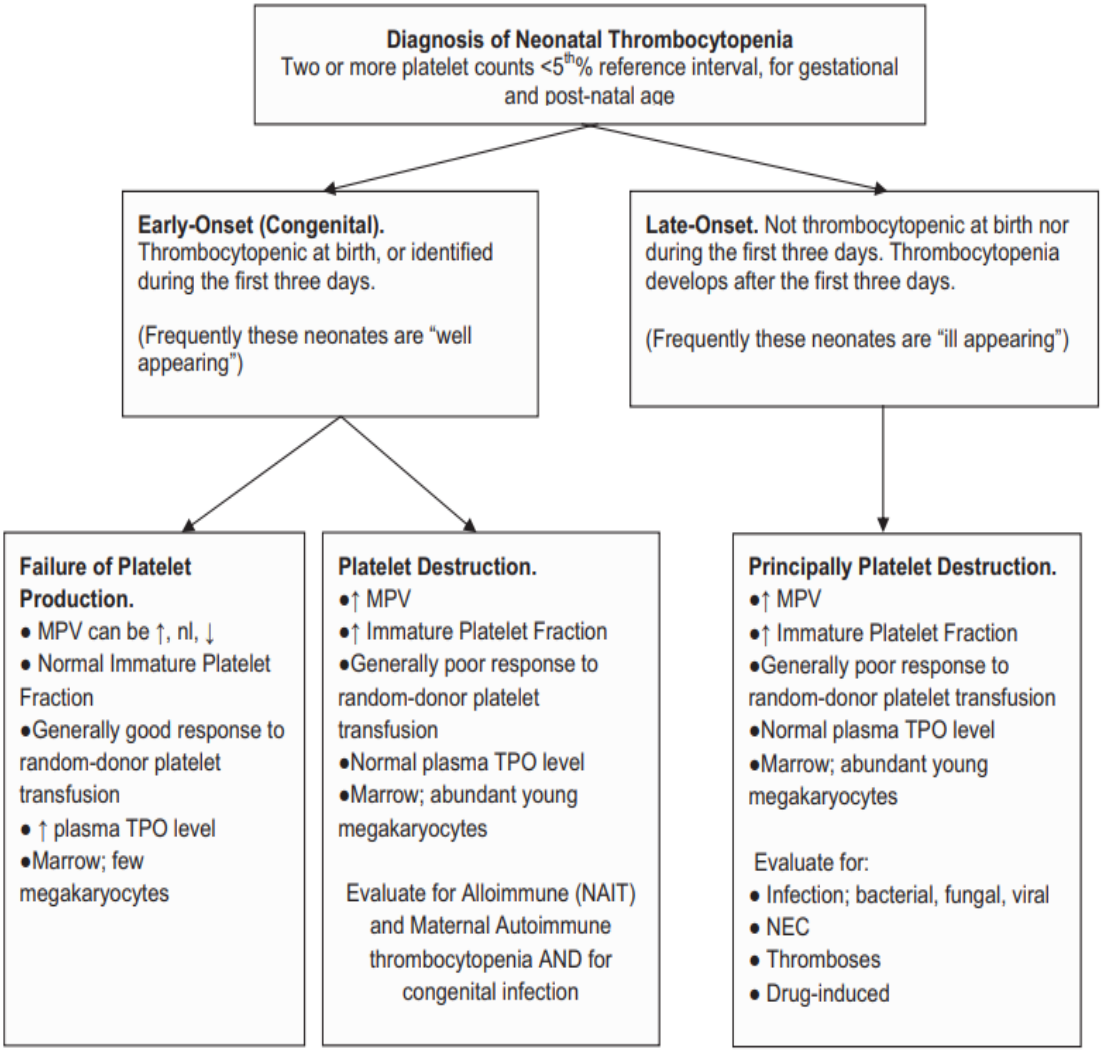


**Figure No-2: Flow chart for diagnosis of NT**

To assess TCP, a bone marrow examination focusing on indicators of megakaryocytopoiesis (megakaryocyte progenitors and megakaryocyte quantity, size, and ploidy distribution) and/or platelet kinetic measurements utilising autologous radiolabeled platelets would be preferable.[20] Unfortunately, the technological limitations of testing such a little, unwell infant have forced neonatologists to rely instead on indirect measurements of platelet production and consumption. Mean platelet volume, platelet-associated immunoglobulin G (IgG) levels, reticulated platelet counts, In-oxine-labeled platelet survival, and the number of circulating

megakaryocytes and their progenitor cells have all been measured to evaluate the kinetic mechanisms that lead to NT.[21,22].

Numerous studies have demonstrated that TCP, just like in older children and adults, occurs when the average foetal platelet count is >150,000/µL during the second trimester of pregnancy and remains steady after that. NT is divided into three categories: mild(100,000–150,000/µL), moderate(50,000- 100,000/µL) and severe(<50,000/µL) [8,14].

**Classification of NT related to the time of onset.**

EOT (≤72 hours)

1. Chronic fetal hypoxia (diabetes, PIH)
2. Asphyxia
3. Fetal/neonatal alloimmune thrombocytopenia
4. Viral infection (e.g HIV, enterovirus)
5. Renal vein thrombosis
6. Polycythemia
7. Chromosomal (Trisomy 13, 18, 21)
8. Bone marrow replacement (e.g., congenital leukemia)

LOT (>72 hours)

1. Acquired bacterial infection
2. NEC
3. Viral infection (e.g., herpes simplex virus, acquired cytomegalovirus)
4. Catheter-related thrombosis
5. HIT
6. Fanconi anemia

**Septicemia and TCP:**

Blood culture results that showed septicemia were strongly correlated with TCP. In the group of people with severe TCP, septicemia was 60% common.

Although it is one of the complications of the illness process and is regarded a valuable first screening for unwell newborns, there are instances where TCP is discovered unintentionally. Despite the fact that TCP is so common, it is frequently disregarded under the impression that it would go away on its own. However, if it is not identified and treated appropriately, it can have terrible side effects[23].

Hypertensive disorders are common in pregnancy, making them a common obstetric concern. Obstetricians and neonatologists face substantial challenges from these disorders since they are associated with a wide range of undesirable maternal outcomes and both immediate and long-term complications for newborns. Pregnancy-related hypertension, preeclampsia, and eclampsia may represent more than just a set of symptoms during pregnancy; they may also be part of a clinical syndrome involving serious vascular abnormalities in both the mother and the developing baby.[24].

**Evaluation of NT**

EOT or LOT, TCP severity, dysmorphic features, duration and clinical features (sick versus not sick), drugs administered, and transfusion response are all important factors to consider when evaluating a thrombocytopenic newborn in the NICU. Late-stage TCP is more likely to entail sepsis, NEC, thrombosis, and medications, whereas early-stage TCP should place special emphasis on the maternal history and prenatal events. It's important to remember that there's a lot of crossover between those two categories.

Newborns in the NICU may develop TCP during the first three days of life if they are exposed to a variety of pathogens, including those that cause NEC, sepsis, and the other disorders typically associated with late TCP. Some conditions often linked to early TCP can occasionally manifest 72 hours after birth[25].

**Risk of bleeding in neonates with thrombocytopenia**

In NICUs, 5-15% of newborns with severe TCP have major bleeding. IVH is the most significant and harmful bleeding occurrence. Pulmonary and gastrointestinal haemorrhage are other, less common bleeding incidents[26].

**Management of NT**

Establishing the aetiology of newborn TCP, delivering medicine according to the diagnosis, and providing supportive care are the main components of managing NT. For the majority of thrombocytopenic newborns, platelet transfusions are the only specific treatment now available. Due to a lack of conclusive information to inform transfusion choices, newborn platelet transfusion practises vary greatly around the globe.

To thrombocytopenic newborns, preventive or therapeutic platelet transfusions are administered to either lessen the risk of bleeding or halt active bleeding. Only 2% of the platelet transfusions performed in the NICU are due to thrombocytopenic haemorrhage; the remaining 98% are done as a preventative measure[27,28].

In 2012, Bolat et al. assessed the incidence of TCP and explored the relationship between the condition and the frequency of IVH grade 2 and fatality rates. Between 2009 and 2012, newborns in Turkey's NICU were the subjects of this study, which was conducted retrospectively. Among which 9.4% of the 2218 newborns assessed who

were thrombocytopenic developed it. Newborns who had TCP were more likely than infants who did not to have TCP to have IVH grade 2 symptoms.

Very severe instances of TCP had greater IVH grade 2 levels than did mild, moderate, and severe cases. The following conditions were major risk factors for death: birth weight 1500 g, gram-negative sepsis, extremely severe TCP, and platelet transfusion. According to the findings of this study, the prognosis for infants with TCP is not only dependent not just on platelet count but also on prenatal variables, sepsis, and reduced gestational age or birth weight[29].

Platelet counts, demographic information, and outcome data from newborns were collected by Christensen et al. in 2012, and template bleeding times were calculated. Reference ranges were created by removing results from newborns who had conditions that are linked with aberrant platelet counts. When a woman is between 23 and 25 weeks pregnant, a platelet count below the 5th percentile, or 100,000/μL, is regarded to be TCP. In 2.4% of neonates admitted to the NICU had severe TCP (platelet count <50,000/μL), most of whom had it because of environmental causes (bacterial and fungal sepsis, NEC and extracorporeal membrane oxygenation). Later gastrointestinal, pulmonary haemorrhages and IVH was not associated with platelet count. There was no correlation between having a low platelet count and increased mortality.[27].

In a hospital, Tirupati K. et al in 2016, examined the pattern, severity, and risk factors of newborn TCP on 200 neonates hospitalised to a hospital's NICU with TCP in a prospective observational research. Risk factors for both mothers and newborns were noted. Based on the level of TCP in the newborns, groups were created. The risk variables and the degree of TCP were compared. Based on the severity of their TCP,

200 newborns with the condition were split into three groups. Moderate to severe TCP affected 81% of infants.

PROM, anaemia and PIH were the most prevalent maternal predisposing variables. In this study 62.5% of infants had severe TCP and low birth weight. Among all neonates with TCP there are 44% of neonates with EOT and 56% of neonates with LOT. Sepsis and BA were the most prevalent neonatal risk factors, affecting 48.5% and 20% of newborns respectively. Early-onset NT was linked to birth hypoxia, whereas LOT was linked to sepsis. In ill newborns, severe TCP was suggested as a prognostic marker[2].

In 2016, Dahmane et al. conducted a retrospective analysis over a four-year period in the NICU. Included were all neonates who had at least one episode of TCP that was verified. IVH grade 2 in survivors were considered to have a poor prognosis. Among 808 neonates who were hospitalised, 12.4% had at least 1 episode of confirmed TCP, and 12 had two episodes. A total of 112 cases with TCP were recorded. In 74.1% of instances, TCP started within the first three days of life. 22.3% of people had mild TCP, 36.7% had moderate, and 41% had severe cases. The most frequent factor contributing to early TCP was IUGR.

The most typical reason of late TCP was nosocomial sepsis. It was discovered that platelet count, gestational age, birth weight, and the underlying reason all affect how thrombocytopenic neonates turn out. NT can be fatal, thus it is important to get the right diagnosis and use the right therapeutic and preventive measures to avoid any fatalities or neurological damage[30].

In a research by Sunil et al., in 2016, controls included 150 neonates with maternal problems such as gestational hypertension, preeclampsia, or eclampsia who were

delivered during the same time as the study's sample of 150 neonates with gestational hypertension. These infants cord blood was taken, and the platelet count was investigated. Infants born to women with prenatal hypertension, preeclampsia, or eclampsia syndrome were more likely to be premature and small for gestational age. Both the mothers and neonates of these instances had significantly lower platelet counts than controls. Platelet count in the newborn was affected by the degree of the mother's hypertension. Increases in both maternal systolic and diastolic blood pressure were associated with an increase in the incidence of TCP in the case group's neonates. Since these infants are more prone to develop TCP in the early postnatal period, they recommended that they be closely monitored and handled to avoid perinatal morbidity and mortality.[31].

With 60 infants, Krishna et al. conducted an observational study between October 2016 and September 2018. TCP was present in 10.9% of all NICU admissions. Of the sixty neonates diagnosed with TCP, 31 were born at full term and 29 were born prematurely. There were 29 premature babies, with 5% being extremely preterm, 8.3% being very preterm, and 35% being intermediate to late preterm. TCP ranged in severity from minimal in 32 neonates to moderate in 14, and severe in 14. There were a several main causes of TCP, but the most common were septicemia, birth asphyxia, and maternal factors. 6.6% of females had DIC, 3% had MAS, 3% had PIH, and 3% had NEC.[4]

Lethargy (33.3%) and poor feeding (35%) were the two most common symptoms among the 60 infants with TCP who were hospitalised. These symptoms were present in infants with mild, moderate, and severe TCP. Neonatal patients who were highly thrombocytopenic had a greater mortality risk. In this study, a low platelet count was an

independent risk factor for a bad outcome. It was suggested that lethargy and poor feeding can be utilised as a prognostic marker in newborns with TCP.[5]

Among 100 neonates who already had neonatal TCP or were developing it in the NICU participated in a prospective trial by Meena et al, in 2019 hundred newborns with TCP participated in the current study. Overall, mild TCP accounted for 46% of cases, moderate for 35%, and severe for 19%. About half (49%) of the infants were diagnosed with LOT, and about half (51%), with EOT. Anemia was the primary predisposing factor in the mother. In newborns, apnea was the most common symptom and sepsis was the most common cause of TCP.

Mortality was greatly impacted by sepsis, RDS, and NEC. Sepsis was the most frequent cause of death, followed by RDS and NEC. They suggested that it was crucial to spot at-risk newborns and start transfusion treatment in order to stop excessive bleeding and potentially serious morbidity[3].

Subjects of the study by Zama et al. were neonatal patients who underwent blood tests, that include CBC, platelet count, HB estimation, red cell index, and PCV; peripheral smear studies; blood cultures; bleeding time; clotting time; prothrombin time; and anti-platelet antibodies. TCP prevalence was found to be 28% in the sample. The research found that 11.2% of infants with TCP were significantly impacted. They deduced that TCP is a significant factor in septicemia being the leading cause of admission to the NICU.[6]

Ribiero conducted retrospective study in 2019 in neonates with TCP (platelet count <150,000/µL) in neonates hospitalised between January 1, 2008, and December 31, 2017. Newborns hospitalised within the first 72 hours of life were also not included if

they had TCP as a result of surgery. There were a total of 134 infants with TCP included in the study, with a prevalence of 3.3%. Of the 138 newborns analysed, 85 had EOT, 20 had LOT, 68 had severe TCP, and 66 did not.[32]

This study indicated that sepsis is a significant independent predictor of LOT. The results showed that sepsis brought on by gram-negative bacteria was a reliable predictor of severe TCP. The authors mentioned the importance of identifying risk factors, making an early diagnosis, and treating the underlying causes of neonatal TCP for successful treatment. This strong association between nosocomial sepsis caused by gram-negative pathogens and LOT and severe TCP makes sense the importance of managing this condition in NICUs.[33]

In 2021, Saber et al. extended the definition of NT to include term infants who met the requirements for it on two separate occasions. Age, weight, gestational age, birth mode, and history of systemic disorders such diabetes mellitus, pre-eclampsia, systemic lupus erythematosus (SLE), and ITP were all collected from the mothers. Newborn information such as gender, birth weight, diagnosis, type of respiratory support, CBC values, and outcome was also collected. In all, 55 term infants with NT met the inclusion criteria; among them, 29 had severe NT. The most common reason for NT was neonatal sepsis, followed by a postpartum illness. In cases where blood cultures came back positive, the most often isolated pathogens were Escherichia coli and Klebsiella. Cases of severe NT showed a higher mortality rate and required more platelet transfusions than those of mild/moderate NT with bleeding symptoms and pulmonary/intraventricular haemorrhage (IVH)[33].

# MATERIALS & METHODS

# MATERIAL AND METHODS

The present study was done to know the risk factors, severity and outcome among neonates admitted to the NICU with thrombocytopenia (TCP) in the Department of Paediatrics at R L Jalappa Hospital and Research centre (RLJH&RC), Kolar, Karnataka affiliated to Sri Devaraj Urs Medical College (SDUMC), a constituent college of Sri Devaraj Urs Academy of Higher Education and Research (SDUAHER).

**Source of Data:**

A total of 103 neonates with TCP admitted to the NICU of RLJH&RC between January 2021 and December 2021 were taken up for the study.

**Study Design:**

It is a 12 months prospective cohort study of neonates admitted to NICU with TCP.

**Sample size:**

Sample size was estimated by using the proportion of babies who had septicaemia as a cause for thrombocytopenia was 60% from the study by Reddy PK et al.[5] using the formula

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

$Z_{1-\alpha/2}$ = 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). in majority of studies p values are considered significant below 0.05 hence 1.96 is used in formula.

P = Expected proportion in population based on previous studies or pilot studies

d = Absolute error or precision.

P = 60% or 0.60

q = 40% or 0.40

d = 10% or 0.10

Using the above values at 95% Confidence level a sample size of 93 subjects with thrombocytopenia will be included in the study.

Considering 10% Nonresponse a sample size of 93+ 9.3 = 103 subjects will be included in the study.

**Inclusion criteria:**

1. All neonates admitted to NICU in RLJH&RC with TCP.

**Exclusion criteria:**

1. Neonates who were discharged against medical advice.
2. Neonates whose parents or guardians who did not agree to be a part of study.

**Method of collection of data:**

At admission to NICU the parents and / or the guardian were informed about the study and a written informed consent was taken from them. A detailed history inclusive of maternal history and obstetric history with a focus on history suggestive of bleeding and its type in the newborn or the mother was obtained as per the proforma.

All neonates fulfilling the inclusion criteria were included in the study. Information regarding a number of conditions that can be associated with neonatal thrombocytopenia (NT) was

recorded e.g., history of PIH, GDM, PROM, anaemia and SLE. Any consumption of drugs by the mother that could predispose to NT was documented.

Gestational age of all neonates was determined based on the New Ballard's scoring system till 14 days of life. [34]

The following investigations were sent for all neonates admitted to NICU :

1. Complete Blood Count (CBC)
2. Peripheral smear study

Reports of CBC which is done as a routine investigation in all babies admitted in NICU was noted.

**CBC**: Blood (2ml) was collected in sterile EDTA vacutainer tubes by venepuncture with all aseptic precautions and immediately transferred to Central Laboratory of RLJH&RC. CBC report was obtained from an automated hematology analyser and TCP was classified as mild(100,000-150,000/ µL), moderate(50,000-99,000/ µL) and severe(<50,000/ µL) based on platelet count- Table 1.

| **Group** | **Platelet count at admission** |
|---|---|
| I / Mild thrombocytopenia | **100,000- 150,000/** µL |
| II / Moderate thrombocytopenia | **50,000-99,000/** µL |
| III / Severe thrombocytopenia | **<50,000/** µL |

**Table 1:- Definition of various groups according to platelet count.**

Platelet counts were repeated every 24 hours in babies with severe TCP and every 48 hours in those with moderate and mild TCP until normal levels were achieved.

For neonates with NT the following investigations were sent:

1. Septic screen: If any two or more of the following parameters were present then the neonate was diagnosed as having probable sepsis.[35]
   - TLC < 5000/µL or more than 24,000/µL.
   - Absolute neutrophil count (ANC) - low count as per Monroe chart for term neonates and Mouzinho chart for very low birth weight neonates.
   - Immature or band cells to total neutrophil ratio- >0.2
   - Micro ESR- >10mm in 1$^{st}$ hour
   - CRP- >1mg/dL
2. Blood culture

Other investigations such as coagulation profile, chest X-ray, neurosonogram(NSG) and CT (computed tomography) brain was performed whenever the need arose.

**Blood culture:** Venous blood (2ml) was collected with all aseptic precautions in a bottle having specific media Mac Conkey Agar for aerobic and anaerobic organisms. Blood culture bottles were incubated at 37$^0$ C using automated BACT/ALERT equipment. Neonates with blood culture growing any organism within 48 hours or within 5 days of incubation was considered as culture proven sepsis (Definitive sepsis).[36]

**CRP**: Blood (2ml) was collected in a sterile vacutainer without anticoagulant by venepuncture and allowed to clot that separates serum. Quantitative determination of

CRP was done by latex turbidimetry using SPINREACT CRP- TURBILATEX and a value of more than 0.6mg/l was considered as abnormal.[6]

In selected cases, prothrombin time (PT), activated partial thromboplastin time (aPTT), INR were done as mentioned below:

**Coagulation profile:** A volume of 1.8 ml of venous blood was collected in a bottle containing 0.2 ml of 3.8% sodium citrate so that the ratio of blood and citrate is 9:1. In the present study PT, aPTT were done by automated coagulation analyzer. (Normal PT: 14-22 sec and Normal aPTT: 30-55 sec)

**The following definitions were used for the study purpose :**

**Premature rupture of membranes (PROM):** is defined as the disruption of fetal membranes before the beginning of labor, leading to spontaneous leakage of amniotic fluid.[37]

**Pregnancy induced hypertension (PIH):** is defined as maternal systolic blood pressure (SBP) >140 mm of Hg and diastolic blood pressure (DBP) >90 mm of Hg.[38]

**Gestational diabetes mellitus (GDM):** is defined as increased blood glucose levels recognised first during the time of pregnancy.

GDM is diagnosed when $\geq$1 value exceeds the criteria with One step oral glucose intolerance test (OGTT) with fasting blood glucose level 92 mg/dl, at 1 hour 180 mg/dl and at 2 hours 150 mg/dl.

or with two step glucose challenge test (GCT) when $\geq$2 values exceeds the criteria with fasting blood glucose level 90 mg/dl, at 1 hour165 mg/dl, at 2 hours 145 mg/dl and at 3 hours 125 mg/dl respectively.[39]

**Maternal anaemia:** is defined as a haemoglobin (Hb) level <11gm/dl during the period of pregnancy.[40]

**Systemic lupus erythematosus (SLE):** is diagnosed in an individual if at least 4 criteria which include at least 1 clinical and 1 immunological criterion were present based on systemic lupus international collaborating clinical criteria for classification of systemic lupus erythematosus (SLICC).[41]

Clinical manifestations:

1. Skin manifestations- malar rash, maculopapular rash
2. Oral or nasal ulcers
3. Non scarring alopecia
4. Synovitis in >2 joints
5. Serositis like pericarditis
6. Renal –urine protein creatinine ratio ≥0.5, RBC casts
7. Neurological – seizures, psychosis, neuropathies.
8. Haemolytic anaemia
9. Leukopenia (<4000/µL)
10. Thrombocytopenia ( platelet count <100,000/µL)

Immunological manifestations:

1. Anti nuclear antibodies (ANA)
2. Anti-ds DNA
3. Anti-smith antibody
4. Antiphospolipid antibody
5. Low serum compliments (C3 and C4)

6. Positive Direct coombs test (DCT) in absence of haemolytic anaemia.

**Maternal drug intake:** Drug induced immune thrombocytopenia is typically caused by platelet destruction from maternal drug dependent antibodies.

History of consumption of the following drugs were noted: quinine, quinidine, trimethoprim-sulfamethoxazole, pencillin, vancomycin, rifampin, carbamazepine, phenytoin, valproic acid, ceftriaxone, ibuprofen, oxaplatin, sumarin and GP IIb/IIIa inhibitors (abciximab, tirofiban) and heparin. [42]

**Rh incompatibility:** A Rh negative mother with Rh positive blood group neonate and presence of the following parameters in neonate-

- Anaemia.
- Unconjugated hyperbilirubinemia within 1$^{st}$ 24 hours of life.
- Elevated reticulocyte count (Normal values: 4-5% in term and 6-10% in preterm neonates).
- Positive Direct coombs test (DCT).[43]

**Neonatal factors**

**Respiratory distress syndrome (RDS):** is defined as presence of any two of the following features:

- Respiratory rate more than 60/minute
- Subcostal or intercostal retractions
- Expiratory grunt or groaning.[44]

**Birth asphyxia (BA):** according to guidelines of the American Academy of Paediatrics (AAP) and the American College of Obstetrics and Gynaecology (ACOG) following criteria were considered in diagnosing birth asphyxia.[45]

- Persistence of Apgar score of 0-3 for longer than 5 minutes.
- Profound metabolic or mixed academia (pH <7) in an umbilical artery blood sample.
- Neonatal neurological sequel (e.g., seizures, hypotonia and coma).
- Multi organ involvement.

**Persistent pulmonary hypertension (PPH):** Neonate presenting with respiratory distress, cyanosis and presenting with any of the following features is diagnosed with PPH.[46]

- Severe hypoxemia, usually a partial pressure of oxygen in arterial blood (PaO2)< 45 mm Hg with Fio2 of 1.0 and intermittent positive pressure ventilation, if necessary.
- If there is evident right to left or bidirectional haemodynamic shunting at ductus arteriosus (Normal PaO2 in post ductal blood is 7.5-15 mm Hg which is lower than post ductal PaO2) or at patent foramen ovale.
- Pulmonary arterial systolic pressure (PASP) more than 40 mm Hg.

**Congenital infections- TORCH (Toxoplasmosis, Other agents, Rubella, Cytomegalovirus, Herpes simplex virus):** Those with clinical features or laboratory evidence of TORCH infections.[47]

**Disseminated intravascular coagulation (DIC):** Neonate presenting with bleeding manifestations like petechiae, gastro intestinal haemorrhage, oozing from venipunture sites with Prolonged PT and aPTT (Normal PT: 14-22 sec and Normal aPTT: 35-55 sec), reduced fibrinogen (Normal: 150-300 mg/dl) and increased D-dimers forms the diagnostic criteria.[48]

**Necrotizing enterocolitis (NEC):** Neonate presenting with vomting, diarrhea, feed intolarence, abdominal distension, frank or occult blood in stools and systemic signs like apnea, bradycardia, lethargy, hypoglycemia and shock. It is classified and diagnosed based on Modified Bell's staging criteria.[49]

All the neonates with TCP were followed up till discharge or till death. On the day of discharge all the neonates underwent a detailed clinical examination to meet criteria for discharge.

**Satisfactory criteria for discharge included :**

- Resolution of acute problems.
- Baby accepting breast feed or paladai feeds .
- Adequate weight gain for a period of 3 consecutive days.
- Baby's weight more than or equal to 1.5 kg.
- No associated morbidity factors such as hypoxic ischemic encephalopathy, persistent seizures and intra cranial bleed.

**Statistical Analysis:**

Descriptive data are presented as number or percentages. Data was entered into Microsoft excel data sheet and was analysed using SPSS v23(IBM Corp.) version software.

**Normality of the continuous data**, was tested by Kolmogorov–Smirnov test and the Shapiro–Wilk test. Continuous data was represented as mean and standard deviation. **Independent t test** was used as test of significance to identify the mean difference between 2 quantitative variables. **Mann Whitney U test** was used as test of significance to identify the

median difference between 2 quantitative variables with Skewed distribution. **Kruskal Wallis test** was the test of significance to identify the mean difference between more than 2 groups for quantitative data with skewed distribution.

Categorical data was represented in the form of Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. **Fischer's exact test** was used as test of significance for qualitative data which did not fulfil the criteria for Chi-square test (2x2 tables only). **Yates correction** was applied wherever chi-square rules were not fulfilled (for 2x2 tables only).

Chi-square was used as test of significance. Continuous data was represented as mean and standard deviation. Independent t test was used as test of significance to identify the mean difference. P value <0.05 was considered as statistically significant.

**Graphical representation of data:** MS Excel and MS word were used to obtain various types of graphs such as bar diagram, Pie diagram, line diagram and Scatter plots.

**Chi-square test:**

$$X^2 = \sum (O - E)^2 / E$$

Where O is observed, E is expected

The dependent variable was the outcome which was classified into discharged (D) and expired (E). P-value (probability of the result) below 0.05 was considered significant.

**Statistical software:** MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data. EPI Info (CDC Atlanta), Open Epi, Med calc and Medley's desktop were used to estimate sample size, odds ratio and reference management in the study.

# RESULTS

# RESULTS

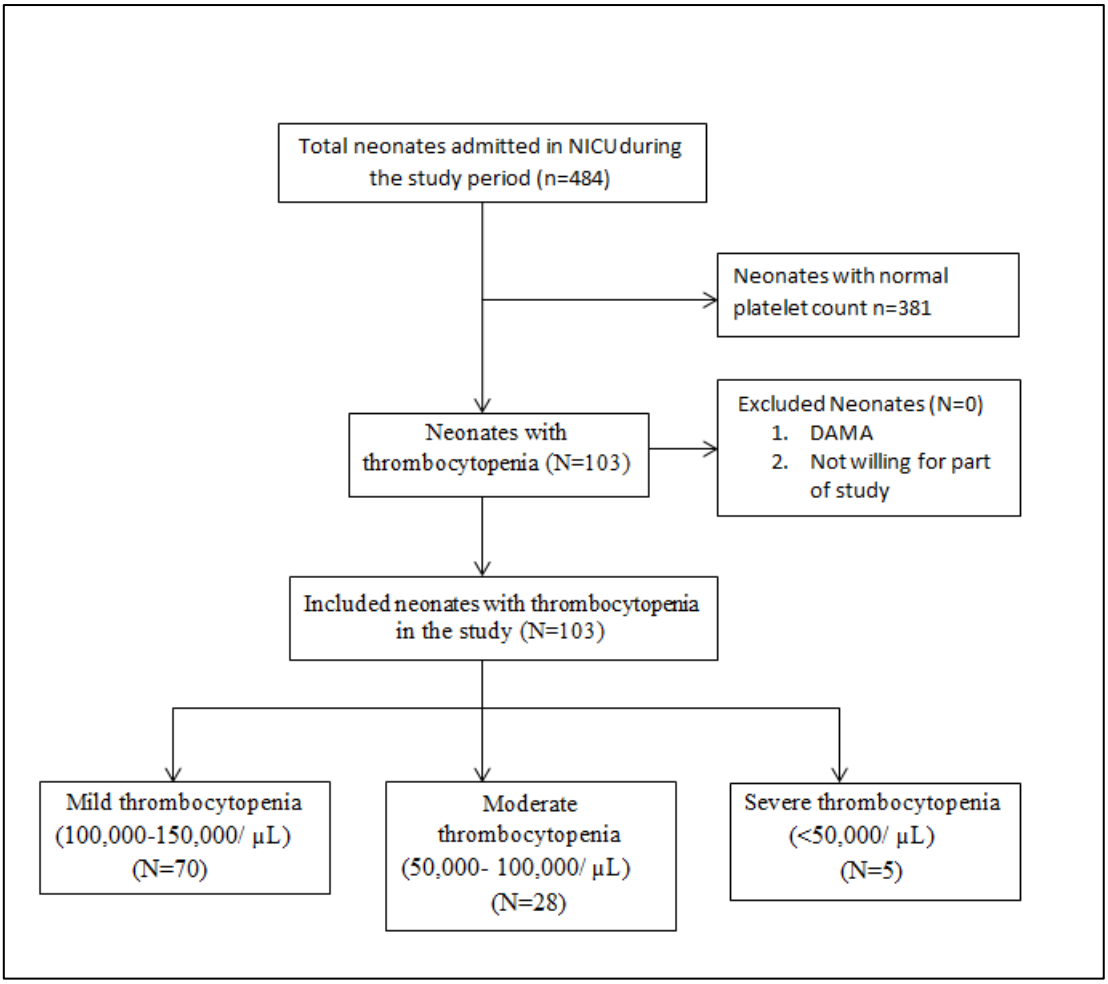


**Figure 3 : Flow diagram depicting the number of neonates included in the present study, along with severity of NT**

A total of 484 neonates were admitted to NICU during the study period. Among them 381 neonates with normal platelet count were excluded from the study.

A total of 103 neonates with TCP were included in our study as per the inclusion and exclusion criteria laid down. The subjects were divided into three groups based on their platelet counts as shown in figure 3.

**Table 2: Distribution of subjects based on severity of NT (N=103)**

| Groups | Description | No. of subjects | Percentage |
|---|---|---|---|
| Group I | Mild thrombocytopenia (100,000/µL- 150,000/ µL) | 70 | 68.0% |
| Group II | Moderate thrombocytopenia (50,000/µL-99,000/ µL) | 28 | 27.2% |
| Group III | Severe thrombocytopenia ( <50,000/ µL) | 5 | 4.9% |

Table 2 shows that majority (68.0%) of the neonates had mild TCP. Moderate and severe TCP were present in 27.2% and 4.9% respectively.

**Table 3: Distribution of subjects based on gender (N=103)**

| Gender | Frequency | Percentage |
|---|---|---|
| Male | 51 | 49.5% |
| Female | 52 | 50.5% |

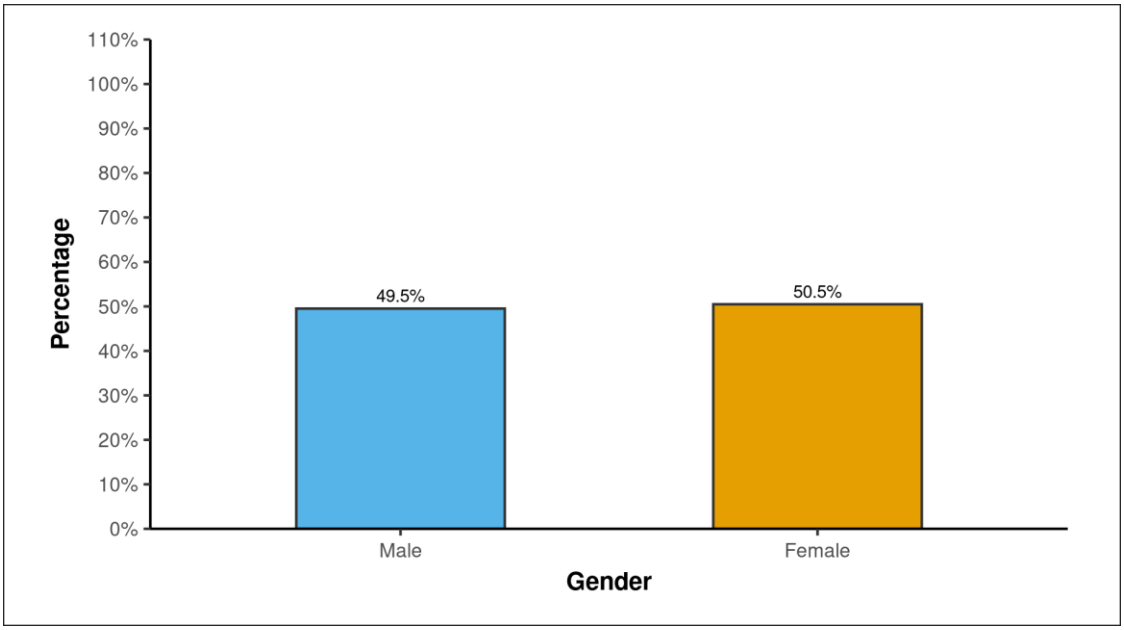


**Figure 4: Distribution of subjects based on gender**

Table 3 and figure 4 shows an almost equal distribution of female (50.5%) and male (49.5%) neonates.

**Table 4: Distribution of subjects based on mode of delivery (N=103)**

| Mode Of Delivery | Frequency | Percentage |
|---|---|---|
| Vaginal | 31 | 30.1% |
| Forceps | 1 | 1.0% |
| Caesarean | 71 | 68.9% |

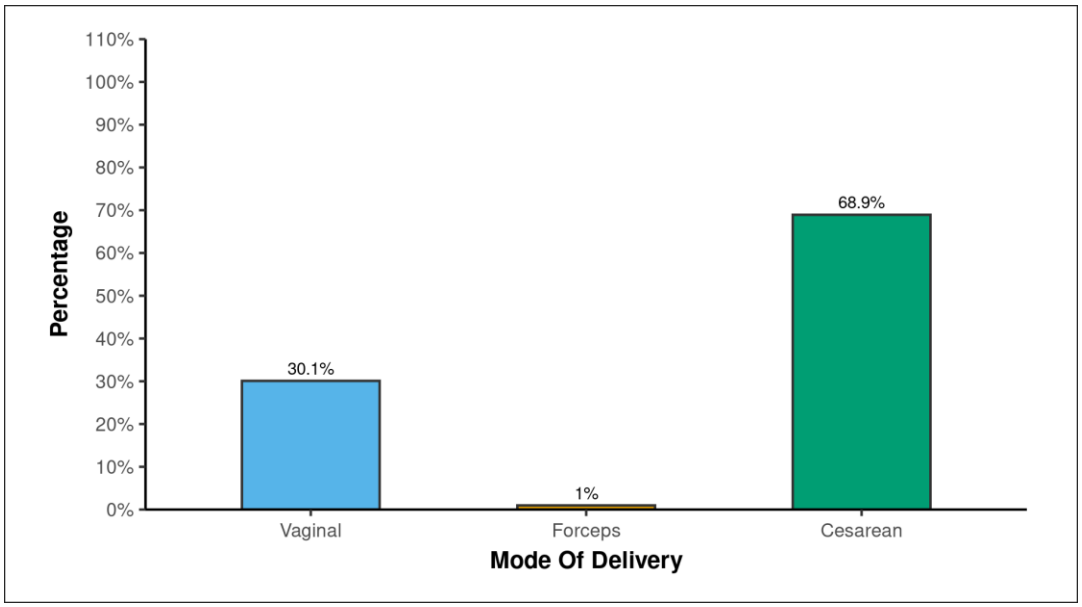


**Figure 5: Distribution of subjects based on mode of delivery**

Table 4 and figure 5 show that majority (68.9%) were born through caesarean section and 30.1% through vaginal mode of delivery. Only 1.0% neonates was born with assisted delivery using forceps.

**Table 5: Distribution of subjects based on gestational age**

| Gestational Age | Frequency | Percentage |
|---|---|---|
| Preterm (<37 Weeks) | 62 | 60.2% |
| Term (≥37 Weeks) | 41 | 39.8% |

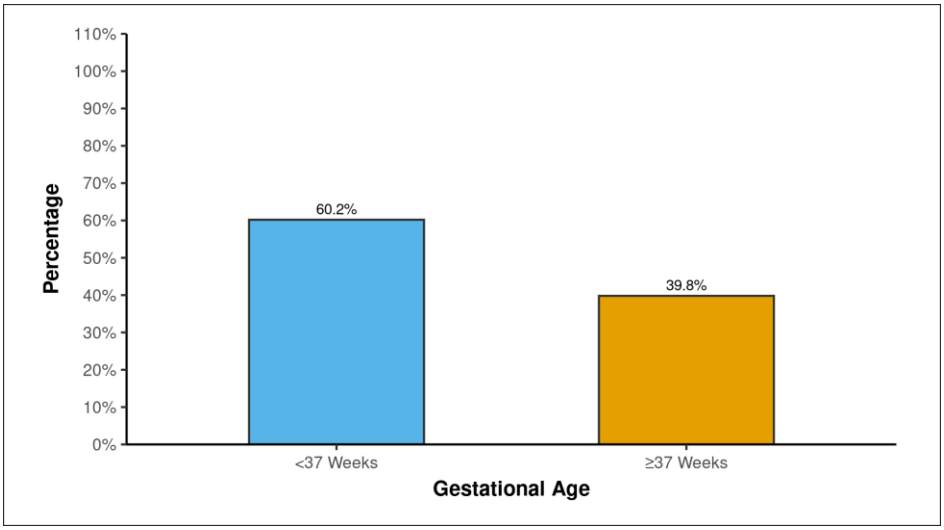


**Figure 6: Distribution of subjects based on gestational age**

Table 5 and figure 6 show that majority (60.2%) were preterm neonates with <37 weeks gestational age and (39.8%) were term neonates with ≥37 weeks of gestational age.

**Maternal risk factors:**

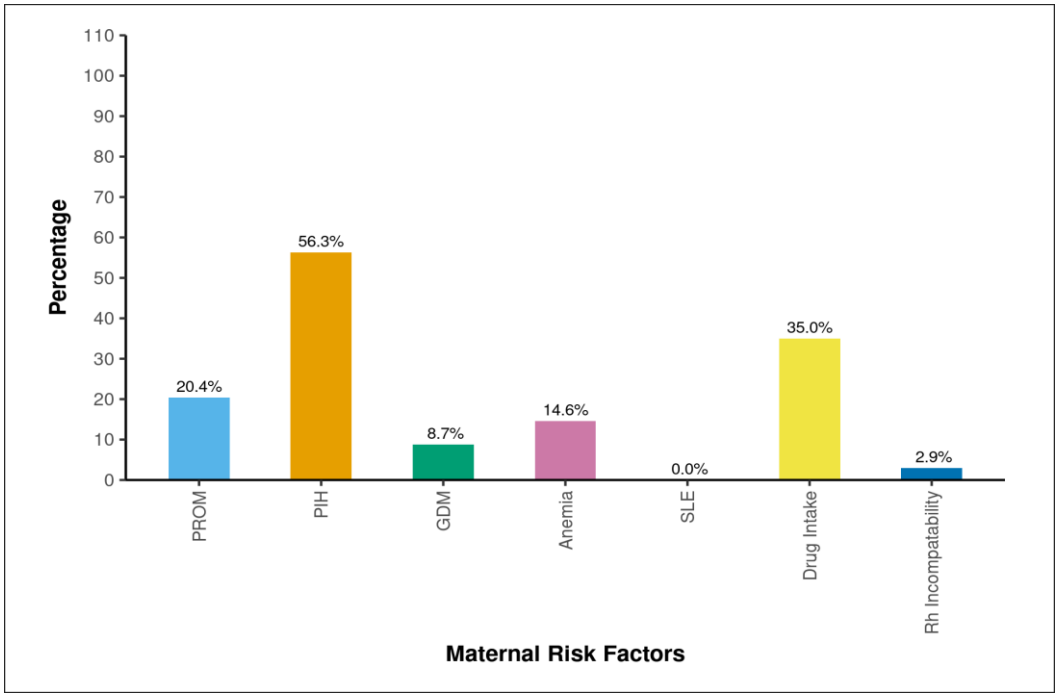


**Figure 7: Distribution of subjects based on maternal risk factors**

Among all neonates with TCP, 58 (56.3%) of their mothers had PIH and 36 (35.0%) of their mothers had drug intake. PROM was present in 21 (20.4%) and anaemia in 15 (14.6%). GDM and Rh incompatibility was present in 9 (8.7%) and 3 (2.9%) mothers respectively - Figure 7.

In some neonates with TCP, more than one maternal risk factor was found. History of both PIH and maternal drug intake was present in 23 mothers. GDM and maternal drug intake was present in 9 mothers. PIH and Rh incompatibility was present in 2 mothers. History of PROM and drug intake was present in 2 mothers. PIH, anaemia and drug intake history was present in 1 mother. PROM and Rh incompatibility was present in 1 mother as shown in Table 6.

**Table 6: Distribution of subjects based on multiple maternal risk factors**

| Maternal Risk Factors | Frequency | Percentage |
|---|---|---|
| PIH + Maternal drug intake | 23 | 60.5% |
| GDM + Maternal drug intake | 9 | 23.7% |
| PIH + Rh incompatibility | 2 | 5.3% |
| PROM + Maternal drug intake | 2 | 5.3% |
| PIH + Anaemia + Maternal drug intake | 1 | 2.6% |
| PROM + Rh incompatibility | 1 | 2.6% |

The various maternal risk factors associated with NT were studied and the significance of their association with TPN was analysed.

**Neonatal risk factors:**

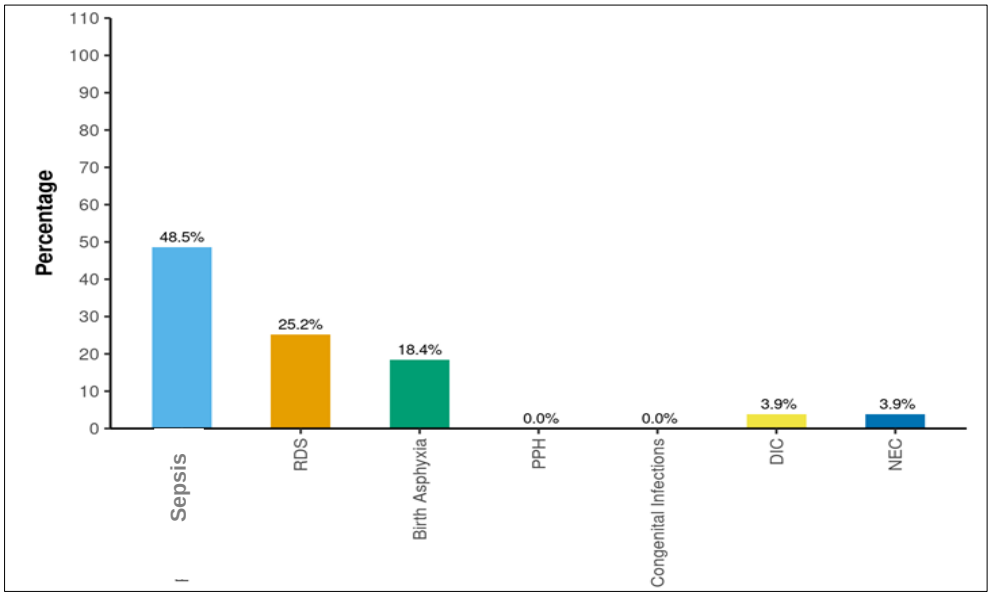


**Figure 8: Distribution of subjects based on neonatal risk factors**

Among neonates with TPN, 50 (48.5%) had sepsis and 26 (25.2%) had RDS. Birth Asphyxia was present in 19 (18.4%), DIC in 4 (3.9%) and NEC in 4 (3.9%) of neonates – Figure 8

**Maternal PROM:**

**Table 7: Association between PROM and NT (n=103)**

| Maternal Risk Factors: PROM | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Present (n=21) | 16 (76.2%) | 5 (23.8%) | 0 (0.0%) | 21 (100%) | 1.654 | 0.626 |
| Absent (n=82) | 54 (65.9%) | 23 (28.0%) | 5 (6.1%) | 82 (100%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 7 depicts the association between PROM and severity of NT. It was observed that among 21 neonates with PROM as maternal risk factor, 16 (76.2%) neonates had mild TPN while 5 (23.8%) had moderate TPN. However there was no significant difference between the various groups in terms of association of PROM ($\chi 2 = 1.654$, $p = 0.626$).

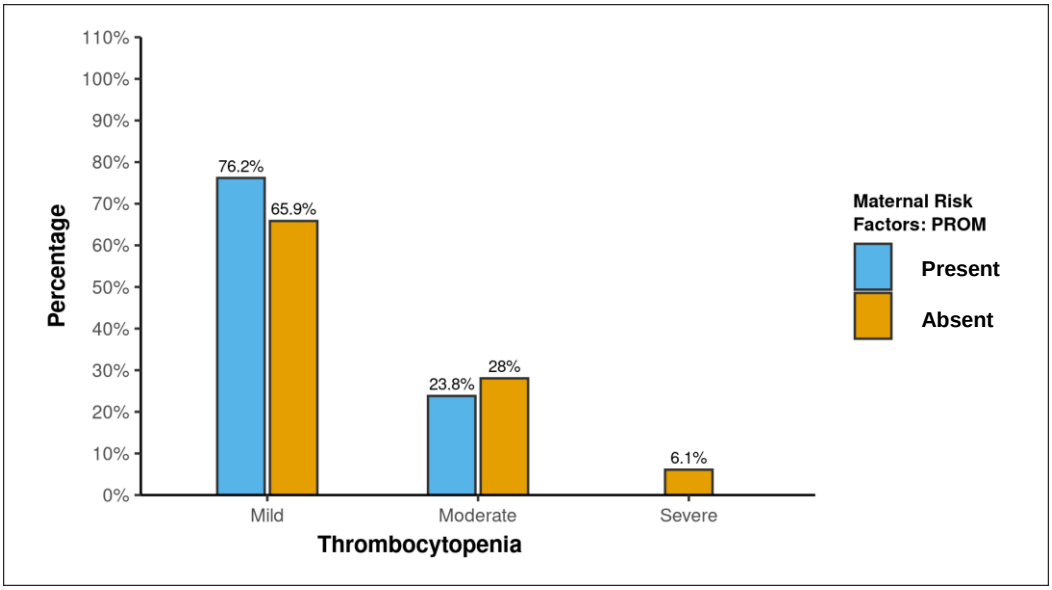


**Figure 9: Association between PROM and NT (n=103)**

**Maternal PIH:**

**Table 8: Association between PIH and NT (n=103)**

| Maternal Risk Factors: PIH | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Present (n=58) | 39 (67.2%) | 16 (27.6%) | 3 (5.2%) | 58 (100.0%) | 0.046 | 1.000 |
| Absent (n=45) | 31 (68.9%) | 12 (26.7%) | 2 (4.4%) | 45 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 8 depicts the association between PIH and severity of NT. It was observed that among 58 neonates with PIH as maternal risk factor, 39 (67.2%) had mild TPN. Moderate and severe TPN was present in 16 (27.6%) and 3 (5.2%) neonates respectively. However there was no significant difference between the various groups in terms of association of PIH ($\chi 2 = 0.046$, $p = 1.000$).

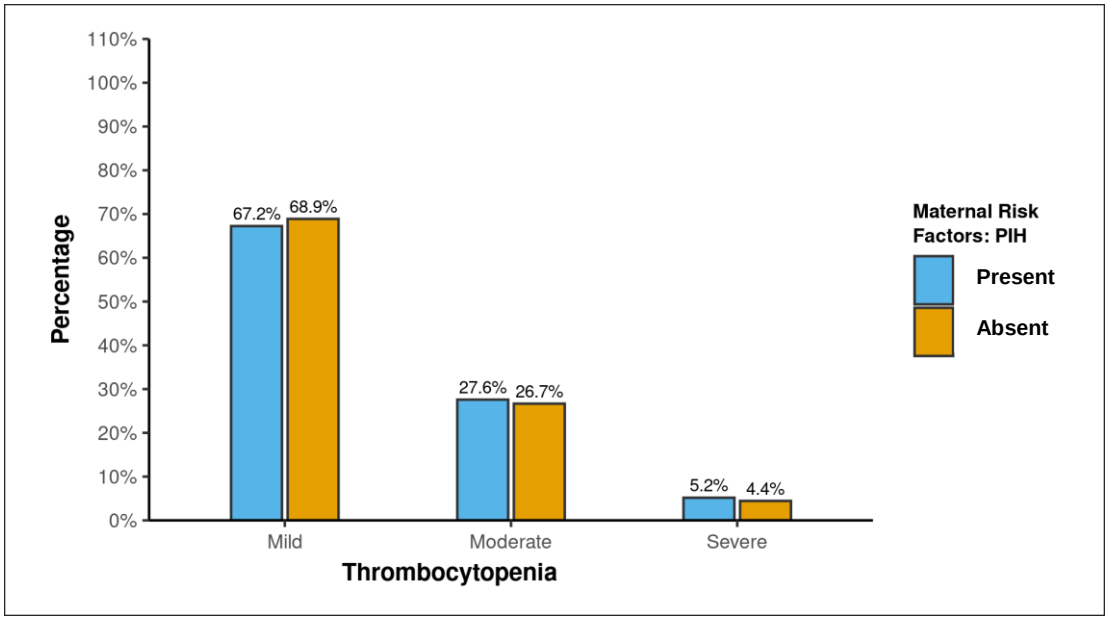


**Figure 10: Association between PIH and NT (N=103)**

**MATERNAL GESTATATIONAL DIABETES MELLITUS(GDM):**

**Table 9: Association between GDM and NT (N=103)**

| Maternal Risk Factors: GDM | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | χ2 | P Value |
| Present (n=9) | 5 (55.6%) | 2 (22.2%) | 2 (22.2%) | 9 (100.0%) | 6.441 | 0.082 |
| Absent (n=94) | 65 (69.1%) | 26 (27.7%) | 3 (3.2%) | 94 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 9 depicts the association between GDM and severity of NT. It was observed that among 9 neonates with GDM as maternal risk factor, 5 (55.6%) neonates had mild TPN while moderate and severe TPN was present in 2 (22.2%) neonates each. However there was no significant difference between the various groups in terms of association of GDM ($\chi2 = 6.441$, $p = 0.082$).

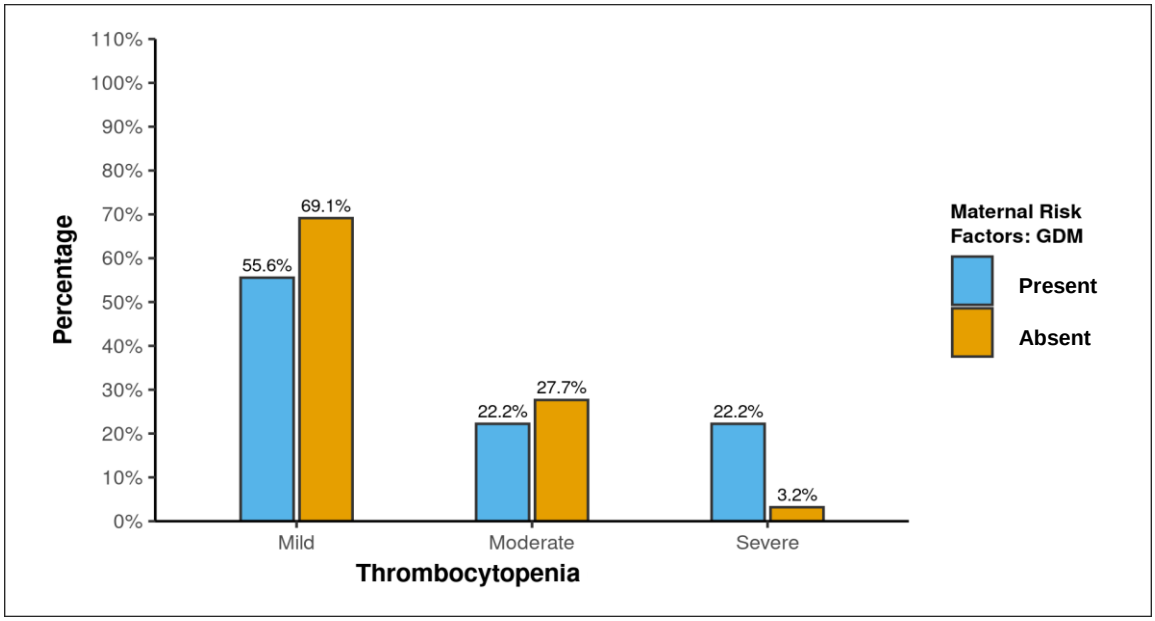


**Figure 11: Association between GDM and NT (n=103)**

**MATERNAL ANEMIA:**

**Table 10: Association between maternal anaemia and NT (n=103)**

| Maternal Risk Factors: Anaemia | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | χ2 | P Value |
| Present (n=15) | 10 (66.7%) | 5 (33.3%) | 0 (0.0%) | 15 (100.0%) | 1.101 | 0.792 |
| Absent (n=88) | 60 (68.2%) | 23 (26.1%) | 5 (5.7%) | 88 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 10 depicts the association between maternal anaemia and severity of NT. It was observed that among 15 neonates with maternal anaemia as risk factor, 10 (66.7%) neonates had mild TPN while 5 (33.3%) had moderate TPN. However there was no significant difference between the various groups in terms of association of Maternal Risk Factor anaemia ($\chi 2 = 1.101$, $p = 0.792$).

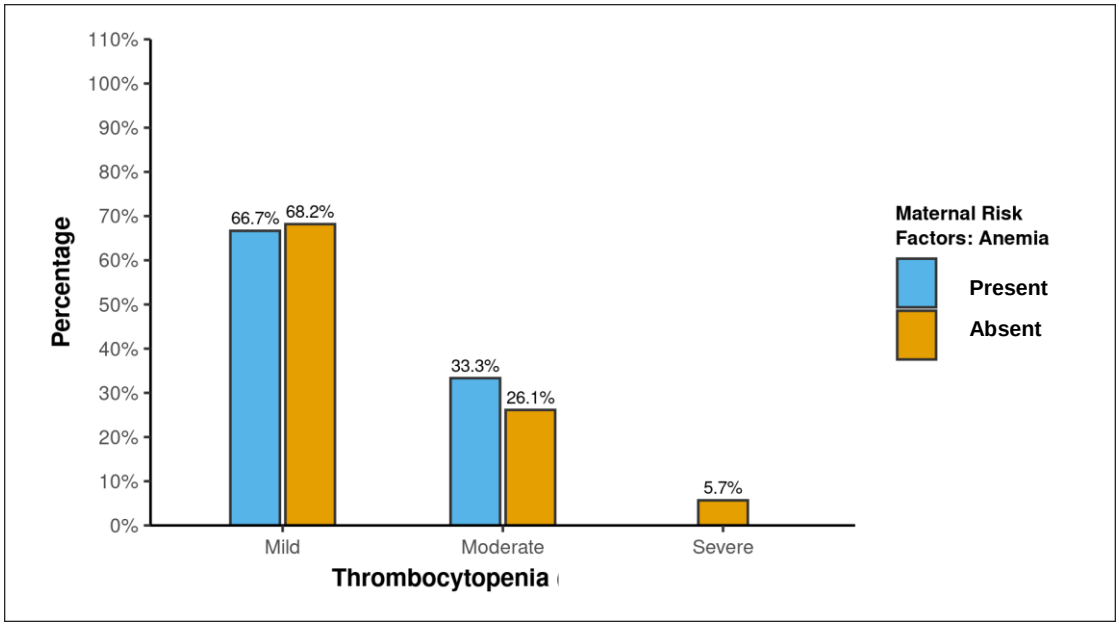


**Figure 12: Association between anaemia and NT (n=103)**

**Maternal drug intake:**

**Table 11: Association between maternal drug intake and NT (n=103)**

| Maternal Risk Factors: Drug Intake | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | χ2 | P Value |
| Present (n=36) | 22 (61.1%) | 11 (30.6%) | 3 (8.3%) | 36 (100.0%) | 1.993 | 0.379 |
| Absent (n=67) | 48 (71.6%) | 17 (25.4%) | 2 (3.0%) | 67 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 11 depicts the association between maternal drug intake and severity of NT. It was observed that among 36 neonates with maternal drug intake as risk factor, 22 (61.1%) had mild TPN while moderate and severe TPN was present in 11 (30.6%) and 3 (8.3%) respectively. However there was no significant difference between the various groups in terms of association of Maternal Risk Factor Drug Intake ($\chi2 = 1.993$, $p = 0.379$).

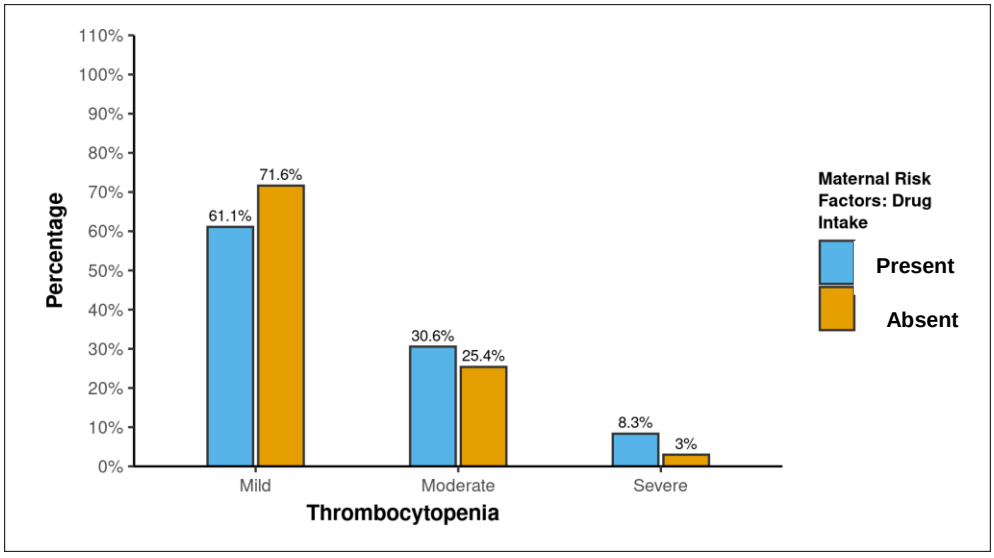


**Figure 13: Association between maternal drug intake and NT (n=103)**

**RH INCOMPATIBILITY:**

**Table 12: Association between Rh incompatibility and NT (n=103)**

| Maternal Risk Factors: Rh Incompatibility | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | χ2 | P Value |
| Present (n=3) | 2 (66.7%) | 0 (0.0%) | 1 (33.3%) | 3 (100.0%) | 6.003 | 0.159 |
| Absent (n=100) | 68 (68.0%) | 28 (28.0%) | 4 (4.0%) | 100 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 12 depicts the association between Rh incompatibility and severity of NT. It was observed that among 3 neonates with Rh incompatibility as risk factor, 2 (66.7%) had mild TPN while 1 (33.3%) had severe TPN. However there was no significant difference between the various groups in terms of distribution of Maternal Risk Factor: Rh Incompatibility ($\chi 2 = 6.003$, $p = 0.159$).

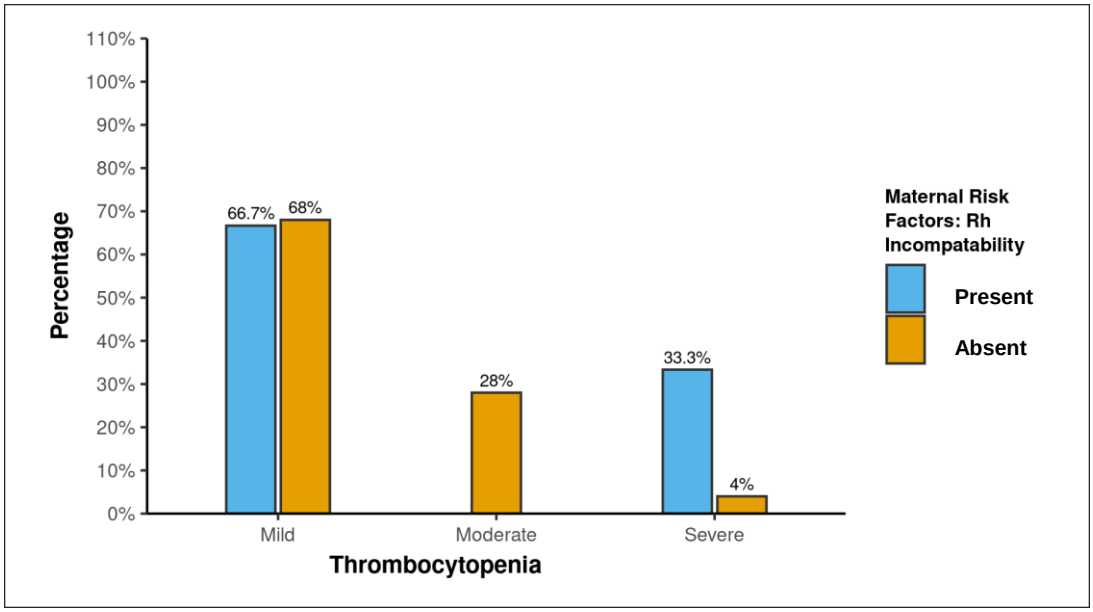


**Figure 14 : Association between Rh Incompatibility and NT(n=103)**

Other maternal factors such as mode of delivery and meconium stained liquor were not significantly associated with NT.

**Neonatal Risk Factors :**

**Probable sepsis and Culture proven sepsis:**

**Table 13: Association between probable sepsis, culture proven sepsis and NT (n=103)**

| Neonatal risk factor: Sepsis | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Probable sepsis (n=50) | 33 (66.0%) | 14 (28.0%) | 3 (6.0%) | 50 (100.0%) | 2.671 | 0.614 |
| Culture proven sepsis (n=15) | 9 (60.0%) | 6 (40.0%) | 0 (0.0%) | 15 (100.0%) | | |
| No sepsis (n=38) | 28 (73.7%) | 8 (21.0%) | 2 (5.3%) | 48 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 13 shows the association between probable sepsis, culture proven sepsis and severity of NT. It was observed that among 50 neonates with probable sepsis as neonatal risk factor, 33 (66.0%) neonates had mild TPN while moderate and severe TPN was present in 14 (28.0%) and 3 (6.0%) respectively. Among 15 neonates with culture proven sepsis, 9 (60.0%) neonates had mild TPN and 6 (40.0%) had moderate TPN. However there was no significant difference between the various groups in terms of association of probable sepsis ($\chi 2 = 2.671$, $p = 0.614$).

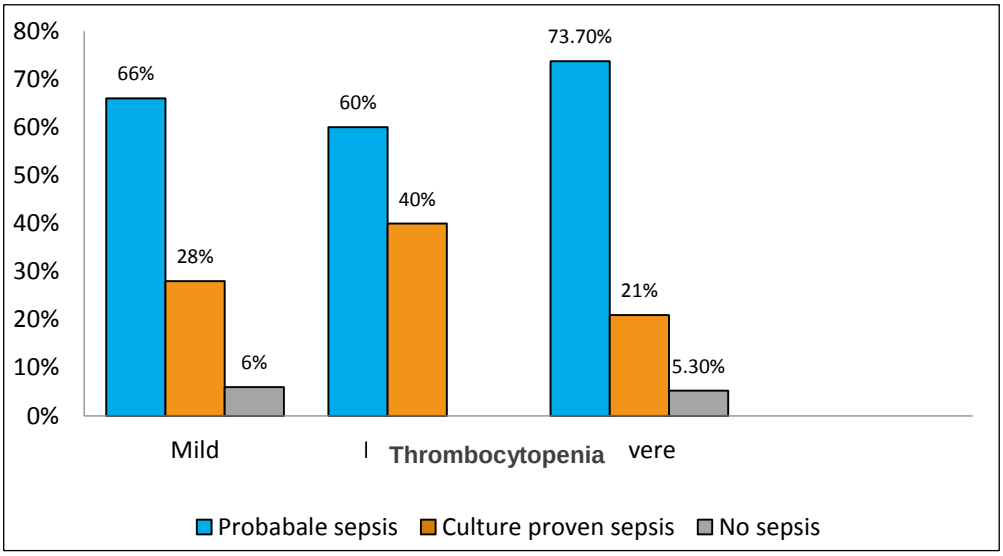


**Figure 15: Association between probable sepsis, culture proven sepsis and NT (n=103)**

**Table 14: Spectrum of organisms in culture proven sepsis among subjects with NT**

| Blood Culture | Thrombocytopenia | | | Fisher's Exact Test | |
|---|---|---|---|---|---|
| | Mild | Moderate | Total | $\chi 2$ | P Value |
| Non-Candida spp | 4 (66.7%) | 2 (33.3%) | 6 (100.0%) | 3.889 | 0.820 |
| Gram positive cocci (GPC) | 2 (66.7%) | 1 (33.3%) | 3 (100.0%) | | |
| Klebsiella spp | 1 (33.3%) | 2 (66.7%) | 3 (100.0%) | | |
| Candida spp | 0 (0.0%) | 1 (100.0%) | 1 (100.0%) | | |
| E coli | 1 (100.0%) | 0 (0.0%) | 1 (100.0%) | | |
| Gram negative bacilli (GNB) | 1 (100.0%) | 0 (0.0%) | 1 (100.0%) | | |
| Total | 9 | 6 | 15 | | |

Table 14 shows the spectrum of organisms in culture proven sepsis among subjects with NT. It was observed that culture proven sepsis was found in 15 neonates. Among 6 neonates with

culture proven non candida species positive, 4 (66.7%) neonates had mild and 2 (33.3%) had moderate TCP respectively. GPC was positive among 3 neonates those had mild and moderate TCP 2 (66.7%) and 1 (33.3%) neonates respectively. Among 3 neonates with klebsiella species positive, 1 (33.3%) neonate had mild TCP and 2 (66.7%) had moderate TCP. Candida species was positive among 1 (100.0%) neonate had moderate TCP. E coli and GNB was positive among neonates had mild TCP each one respectively.

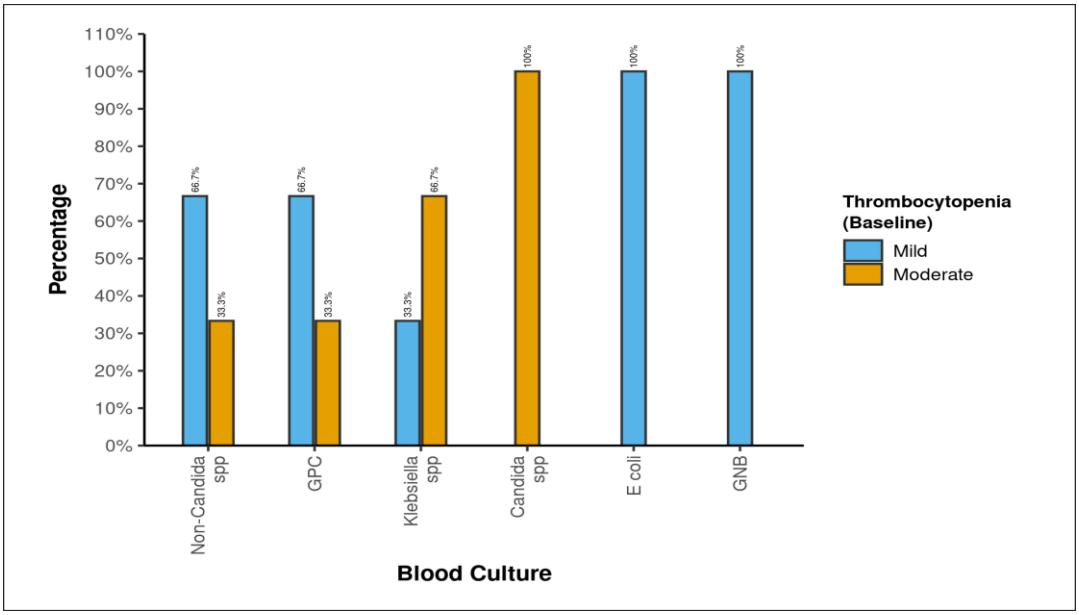


**Figure 16: Spectrum of organisms in culture proven sepsis among subjects with NT**

**RESPIRATORY DISTRESS SYNDROME(RDS):**

**Table 15: Association between RDS and NT (n=103)**

| Neonatal Morbidity: RDS | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | χ2 | P Value |
| Present (n=26) | 19 (73.1%) | 7 (26.9%) | 0 (0.0%) | 26 (100.0%) | 1.823 | 0.526 |
| Absent (n=77) | 51 (66.2%) | 21 (27.3%) | 5 (6.5%) | 77 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 15 depicts the association between RDS and severity of NT. It was observed that among 26 neonates with RDS, 19 (73.1%) neonates had mild TPN, while 7 (26.9%) had moderate TPN. However there was no significant difference between the various groups in terms of association of RDS ($\chi2 = 1.823$, $p = 0.526$).

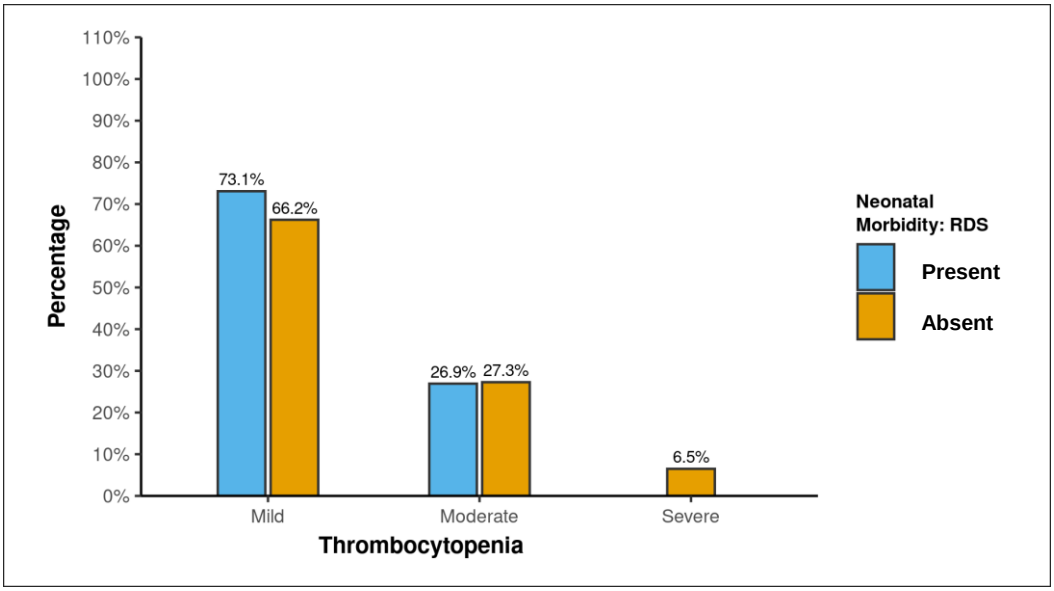


**Figure 17: Association between RDS and NT (n=103).**

**BIRTH ASPHYXIA (BA):**

**Table 16: Association between BA and NT (n=103)**

| Neonatal risk factor: Birth Asphyxia | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Present (n=19) | 14 (73.7%) | 3 (15.8%) | 2 (10.5%) | 19 (100.0%) | 2.769 | 0.205 |
| Absent (n=84) | 56 (66.7%) | 25 (29.8%) | 3 (3.6%) | 84 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 16 shows the association between BA and severity of NT. It was observed that among 19 neonates with BA, 14 (73.7%) neonates had mild TPN while moderate and severe TPN was present in 3 (15.8%) and 2 (10.5%) respectively. However there was no significant difference between the various groups in terms of association of BA. ($\chi 2 = 2.769$, $p = 0.205$).

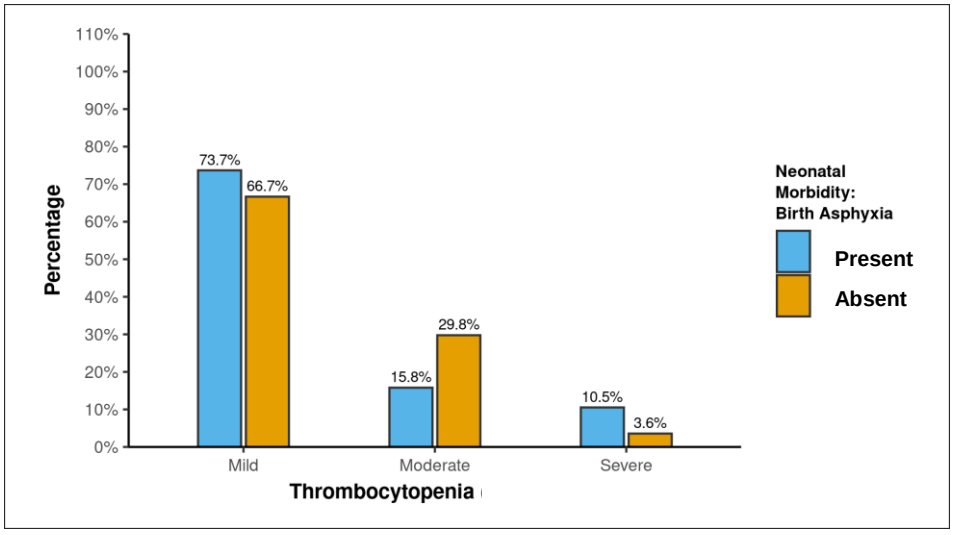


**Figure 18 : Association between BA and NT (n=103)**

**Disseminated intravascular coagulation(DIC):**

**Table 17: Association between DIC and NT (n=103)**

| Neonatal risk factor: DIC | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Present (n=4) | 2 (50.0%) | 2 (50.0%) | 0 (0.0%) | 4 (100.0%) | 1.196 | 0.446 |
| Absent (n=99) | 68 (68.7%) | 26 (26.3%) | 5 (5.1%) | 99 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 17 depicts the association between DIC and severity of NT. It was observed that among 4 neonates with DIC, 2 (50.0%) neonates had mild TPN, and 2 (50.0%) had moderate TPN. However there was no significant difference between the various groups in terms of association of DIC ($\chi 2 = 1.196$, $p = 0.446$).

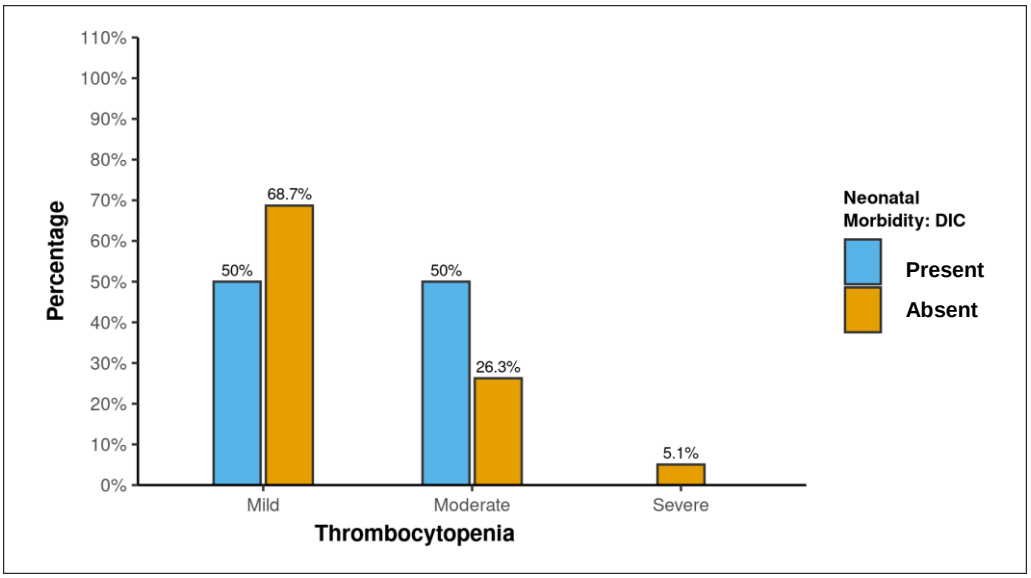


**Figure 19: Association between DIC and NT (n=103).**

**NECROTISING ENTEROCOLITIS(NEC):**

**Table 18: Association between NEC and NT (n=103)**

| Neonatal Morbidity: NEC | Thrombocytopenia | | | | Fisher's Exact Test | |
|---|---|---|---|---|---|---|
| | Mild | Moderate | Severe | Total | $\chi 2$ | P Value |
| Present (n=4) | 2 (50.0%) | 2 (50.0%) | 0 (0.0%) | 4 (100.0%) | 1.196 | 0.446 |
| Absent (n=99) | 68 (97.1%) | 26 (92.9%) | 5 (100.0%) | 99 (100.0%) | | |
| Total | 70 | 28 | 5 | 103 | | |

Table 18 shows the association between NEC and severity of NT. It was observed that among 4 neonates with NEC, 2 (50.0%) neonates had mild TPN, while 2 (50.0%) had moderate TPN. However there was no significant difference between the various groups in terms of association of NEC ($\chi 2 = 1.196$, $p = 0.446$).

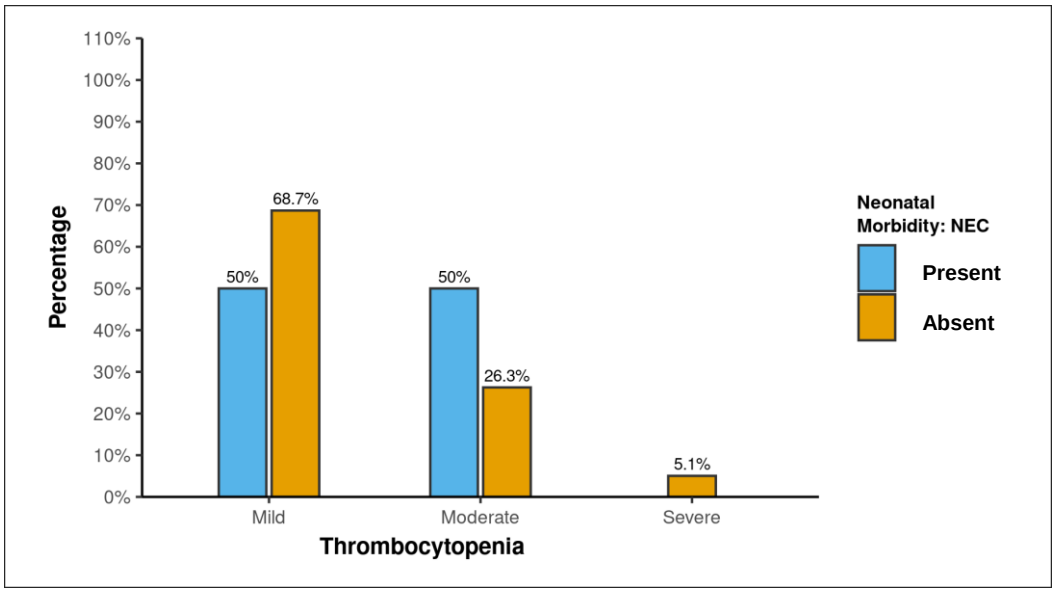


**Figure 20: Association between NEC and NT (n=103).**

There were no neonates with the neonatal risk factors like persistent pulmonary hypertension (PPH) and neonates with congenital infections in the present study.

**Outcome:**

**Platelet transfusion :**

**Table 19: Distribution of subjects based on platelet transfusion**

| Platelet Transfusion | Frequency | Percentage | 95% CI |
|---|---|---|---|
| Yes | 9 | 8.7% | 4.3% - 16.4% |
| No | 94 | 91.3% | 83.6% - 95.7% |

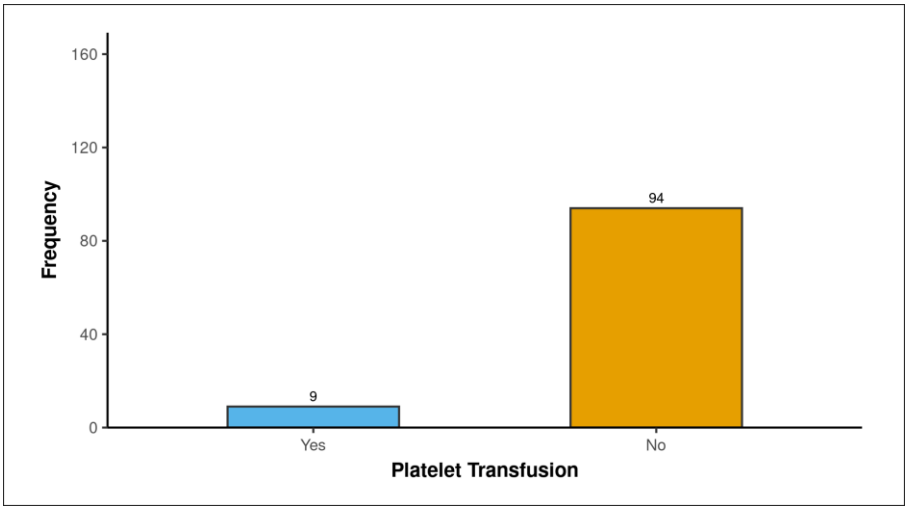


**Figure 21: Distribution of subjects based on platelet transfusion. (n=103)**

Table 19 and figure 21 shows the distribution of subjects based on need for platelet transfusion. It was observed that among all subjects platelet transfusion was administered to 9 (8.7%) neonates.

**Duration of hospital stay :**

**Table 20: Distribution of subjects based on NT and duration of hospital stay (discharged) (n=94).**

| Duration Of Stay (Days) | Thrombocytopenia | | | Kruskal Wallis Test | |
|---|---|---|---|---|---|
| | Mild | Moderate | Severe | $\chi 2$ | p value |
| Mean (SD) | 15.75 (8.10) | 15.14 (11.78) | 16.25 (3.30) | 2.950 | 0.229 |
| Median (IQR) | 15 (10-17.5) | 11.5 (10-14.75) | 16 (13.75-18.5) | | |
| Min - Max | 6 – 53 | 4 - 54 | 13 – 20 | | |

Table 20 shows the distribution of subjects based on NT and duration of hospital stay among discharged. It was observed that, neonates with severe TPN had more duration of hospital stay compare to neonates with mild and moderate TPN.

However there was no significant difference between the groups in terms of Duration of Stay (Days) ($\chi 2 = 2.950$, $p = 0.229$).

**Discharge/expired:**

**Table 21: Distribution of subjects based on outcome (discharge/expired) (n=103)**

| Outcome | Frequency | Percentage | 95% CI |
|---|---|---|---|
| Discharged | 94 | 91.3% | 83.6% - 95.7% |
| Expired | 9 | 8.7% | 4.3% - 16.4% |

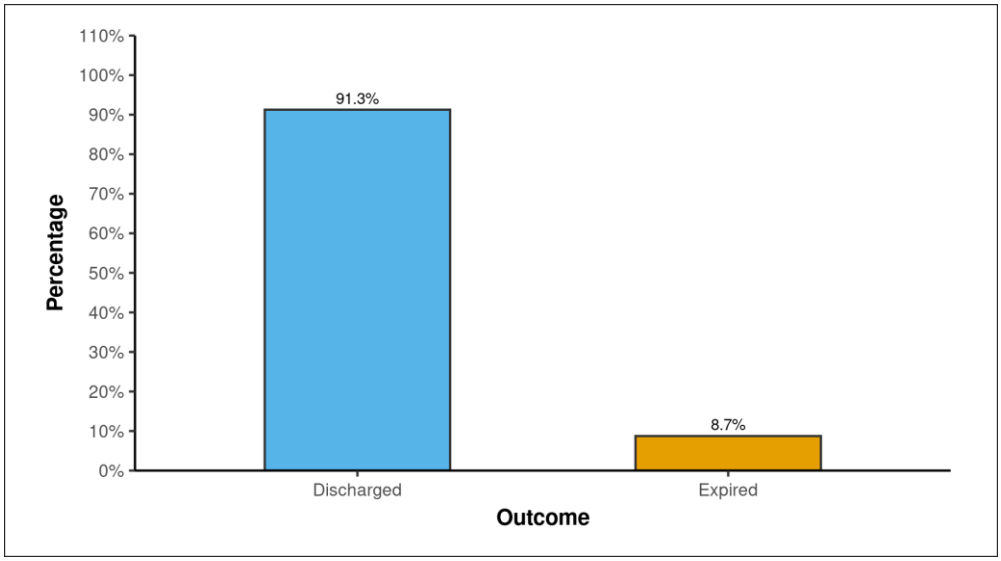


**Figure 22: Distribution of subjects based on Outcome (discharged/expired) (n=103)**

Table 21 and figure 22 shows the distribution of subjects based on outcome (discharged/expired). It was observed that among all subjects, 94 (91.3%) were discharged and 9 (8.7%) expired.

# DISCUSSION

# DISCUSSION

One of the most frequent haematological abnormalities seen in NICUs is NT (platelet count <150,000/µL). It can go unnoticed if not properly looked for. If it is not identified and adequately managed, it may lead to serious consequences[2]. The present study was a prospective cohort study conducted to analyse the risk factors, severity and outcome among the neonates suffering from TCP admitted in NICU.

The present study showed an almost equal distribution among both males and females. Contrastingly a male predominance 60% was seen in a study by **Reddy and Kondle**[5] and 56% in a study by **Tirupathi et al**[2]. The mean age of the participants in present study was 1.09 ± 0.47 days. In a study by **Sola et al**[9], 2 days was the median age of the participants.

In the present study it was seen that majority (68.9%) were born through Caesarean delivery followed by vaginal delivery (30.1%) and forceps delivery (1%). Similarly in a study by **Saber et al**[33], it was seen that 54.55% required a Caesarean section while 45.45% underwent a normal vaginal delivery. In the present study 60.2% of the participants had gestational age <37 weeks while the remaining 39.8% had ≥37 weeks with mean gestational age was 35.48 ± 2.93 weeks, whereas in a study by **Reddy and Kondle** 51.7% participants were term babies.[5]

Various maternal risk factors were present in neonates with TCP. Majority of neonates had maternal risk factor (56.3%) PIH, followed by maternal drug intake (35%), PROM (20.4%), anemia (14.6%), GDM (8.7%) and Rh Incompatibility (2.9%). None of the neonates had maternal risk factor SLE.

TCP was present in 56.3% of newborns who also had maternal PIH. PIH was the most prevalent maternal risk factor, affecting 13.5% of the newborns in a study by **Tirupathi et al**.[2] In their studies, **Meena et al**[3] and **Reddy and Kondle**[5] discovered that 19.0% and 18.3%

of newborns, respectively, had PIH. PIH can result in IUGR, is a known risk factor for NT, and is frequently a sign that a caesarean section is necessary. Although the precise pathophysiology of NT in PIH is uncertain, it is believed that the condition causes foetal hypoxia, which lowers foetal megakaryocytopoiesis and platelet production.[11] However in a study by **Meena et al**[3], anemia (48%) was the most common risk factor followed by PROM (30%), PIH (19%), oligohydramnios (2%) and eclampsia in (2%) cases.

In the present study the mean Apgar (5 Minutes) was 8.30 ± 1.00, whereas in a study by **Gunnink et al,**[16] a low Apgar score was seen in 12.6% subjects.

Meconium-stained amniotic fluid (MSAF) is problematic for both obstetricians and paediatricians because it increases the risk of caesarean delivery and increases perinatal morbidity and death. MSAF occurs between 12-20% of the time.[4] In the current study, 14.6% cases presented with meconium stained liquor, whereas in a study conducted by **Reddy and Kondle**[5] there were 6.6% cases developed meconium aspiration syndrome (MAS).

At admission mild, moderate and severe TCP was present in 68%, 27.2% and 4.9% respectively. Similar results were obtained in a study by **Meena et al**[3] mild, moderate and severe TCP was present in 46%, 35% and 19% respectively. In a study by **Reddy and Kondle**[5] 53.3% of participants had severe TCP, 23.3% had moderate TCP, and 23.3% had mild TCP. In study by **Zama et al** [6] mild TCP was more prevalent. Severe TCP was found in 65.6% of babies in a study by **Nandyal et al**[28].

In the blood culture done, majority cases (40%) showed the presence of Non-Candida species, followed by 20% had GPC, 20% had Klebsiella species, 6.7% had Candida species, 6.7% had E coli and 6.7% had GNB.

Among various neonatal risk factors causing NT, sepsis was the most commonest cause which was found in 50 (48.5%) neonates and was associated with mild TCP. The percentage of sepsis among neonates with TCP in **Tirupathi et al[2] study** was 48.5% and **Meena et al**[3] was 53% which were associated with severe TCP. Septicaemia leads to TCP due to decreased production and increased consumption of platelets resulting in severe TCP.

In the present study, majority (48.5%) had sepsis, 25.2% had RDS, 3.9% had DIC, 3.9% had NEC while no neonates had PPH or congenital infections. 60% of the infants in the research by **Reddy and Kondle[5]** had septicemia as the underlying cause of their TCP. In the study, 6.6% of babies developed DIC, 6.6% had MAS, and 5% had NEC. BA affected 23.3% of the babies. In 53% of instances in a study by **Meena et al**[3], sepsis was the most common factor contributing to NT, followed by RDS in 15%, Birth asphyxia in 11%, MAS in 10%, neonatal hyperbilirubinemia in 6%, and NEC in 5%.

This results were consistent with past studies that have identified sepsis as a possible risk factor for TCP in newborns admitted to the NICU.[2,3] **Reddy and Kondle[5]** study may have had a greater incidence of sepsis since it was conducted on preterm infants, who have weaker immune systems than term infants and are therefore more vulnerable to infection. BA was seen in 18.4% of the current cases. This result was similar to those of **Tirupathi et al** (20%), and **Zama** et al (24%) research.[2,6] Because of the decreased synthesis of platelets as well as the increased consumption of them, sepsis usually causes severe TCP. Prematurity is known to increase the risk of TCP because it reduces platelet production. Additionally, placental transfer of IgG from the mother to the growing foetus gets better with age but is slowed down in premature babies, rendering them more vulnerable to sepsis.[3]

In the current study, 17.5 % neonates had blood transfusion and 8.7% had platelet transfusion. Platelet transfusion was done in 40% neonates with severe TCP, where as 14.3%

and 4.3% in neonates with moderate and mild TCP respectively. In a research by **Reddy and Kondle**[5], 20% neonates needed platelet transfusion. **Meena et al**[3] revealed that in their research 5% neonates needed blood transfusion while 13% needed platelet transfusion and 27% were given FFP.

The mean duration of hospital stay was 14.92 ± 8.89 days. Although non-significant, the mean duration of hospital stay in mild, moderate and severe TCP is 15.75 ± 8.10 days, 15.14 ± 11.78 days and 16.25 ± 3.30 days respectively. **Reddy and Kondle**[5] observed a mean duration of hospital stay of 7, 9.5 and 16 days in mild, moderate and severe TCP respectively. The mean duration of hospital stay in a research by **Saber et al**[33] was slightly ($8.67 \pm 3.95$ days) as compared to the present study.

The fact that the babies in the severe TCP group expired before their sickness had fully developed may help to explain this. These findings conflicted with those of **Nandyal et al**.[23] who discovered a positive relationship between the length of stay and the severity of TCP and the quantity of subsequent platelet transfusions.

The outcome of this duration of hospital stay was that 91.3% were discharged while the remaining 8.7% expired. Out of the subjects that expired, 20 %, 21.4% and 2.9% had severe, moderate and mild TCP respectively. According to a research by **Reddy and Kondle**[5]**,** 16.6% of neonates with TCP were expired. Among them 80% of the neonates that expired had severe TCP, while 20% of the children had moderate TCP. The severe TCP neonates experienced a significant mortality rate. This may be associated with the more severe underlying condition or by the neonates increased vulnerability to problems in the severe TCP group. Additionally, in the study conducted by **Saber et al**[33] mortality was seen 10.9% neonates and around 34% according to **Meena et al**[3] .This may be because there are fewer cases of severe TCP (N=19) than of mild or moderate severity (N=71). It is possible to use

the correlation between high mortality and severe TCP as a prognostic indicator to evaluate the health of newborns.[3]

NT is typically attributed to impaired megakaryopoiesis, insufficient platelet synthesis, and accelerated platelet destruction. The time of the development of TCP can help identify the underlying cause of neonatal TCP to a greater extent". TCP occurs typically 72 hours after delivery and is caused by foetal hypoxia, as it does in babies delivered to moms who have gestational hypertension. Neonatal TCP caused by NEC and sepsis typically manifests after a few days of life and is severe. The danger of bleeding is much lower in foetal hypoxia than it is in sepsis. The necessity for a platelet transfusion varies depending on the cause and risk of bleeding.[32]

# CONCLUSION

# CONCLUSION

Thrombocytopenia (TCP) is a haematological condition affecting most of the neonates admitted to NICU and can have serious consequences. The present study makes an effort to identify the risk factors, severity and outcome of neonates with TCP in the admitted neonates. Among 103 neonates with TCP, majority (68.0%) had mild TCP. Moderate and severe TCP were present in 27.2% and 4.9% respectively. An almost equal distribution of males and female was seen in the current study.

Majority were born through Caesarean delivery (68.9%). Most of the participants had gestational age <37 weeks (60.2%). Various maternal risk factors such as PIH (56.3%), drug intake (35.0%), PROM (20.4%), anaemia (14.6%), GDM (8.7%) and Rh incompatibility (2.9%) were present. There was no significant association between maternal risk factors and severity of NT. Various neonatal risk factors such as Sepsis (48.5%), RDS (25.2%), BA (18.4%), DIC (3.95%) and NEC (3.9%) were present. There was no significant association between neonatal risk factors and severity of NT.

The mean duration of hospital stay in mild, moderate and severe TCP is 15.75 ± 8.10 days, 15.14 ± 11.78 days and 16.25 ± 3.30 days respectively. Platelet transfusion was given for 9 (8.7%) neonates. A mortality of 8.7% was noticed in the study.

# SUMMARY

# SUMMARY

The present study was a prospective cohort study that was conducted at RLJH & RC over a period of 1 year from January 2021 to December 2021 to evaluate the risk factors, severity and outcome among the neonates with TCP admitted in NICU.

A total of 103 neonates who fulfilled the inclusion criteria were included in the study. At admission, 68%, 27.2% and 4.9% presented with mild, moderate and severe TCP respectively.

The present study showed almost equal distribution among males and females. The mean age of the participants was 1.09 ± 0.47 days. Majority (68.9%) were born through caesarean delivery. The mean gestational age was 35.48 ± 2.93 weeks. In present study 60.2% of the participants had gestational age <37 weeks while the remaining 39.8% had ≥37 weeks. Various maternal risk factors such as PIH (56.3%), drug intake (35.0%), PROM (20.4%), anaemia (14.6%), GDM (8.7%) and Rh incompatibility (2.9%) were present. There was no significant association between maternal risk factors and severity of NT. Various neonatal risk factors such as Sepsis (48.5%), RDS (25.2%), BA (18.4%), DIC (3.95%) and NEC (3.9%) were present. There was no significant association between neonatal risk factors and severity of NT.

The mean duration of hospital stay in mild, moderate and severe TCP was 15.75 ± 8.10 days, 15.14 ± 11.78 days and 16.25 ± 3.30 days respectively. Platelet transfusion was given for 9 (8.7%) neonates. A mortality of 8.7% was noticed in the study.

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# ANNEXURES

# RISK FACTORS, SEVERITY AND OUTCOME AMONG NEONATES WITH THROMBOCYTOPENIA ADMITTED IN NEONATAL INTENSIVE CARE UNIT- A PROSPECTIVE COHORT STUDY

PROFORMA

NAME:

AGE:

IP NO:

UHID NO:

GENDER:

GESTATIONAL AGE:

ADDRESS:

PHONE NO:

DATE &TIME OF ADMISSION:

DATE &TIME OF DISCHARGE:

**MATERNAL HISTORY:**

Maternal Platelet count:

H/O PROM: YES/NO

Pregnancy induced hypertension: YES/NO

Gestational diabetes mellitus: YES/NO

Maternal Anemia (Hb<11 gm/dl): YES/NO

Systemic lupus erythematosus: YES/NO

Drugs : YES/NO IF YES, DETAILS----------

Any other significant maternal illness:

**BIRTH HISTORY:**

Mode of delivery: Normal vaginal delivery/ Cesarean delivery /Vacuum assisted vaginal delivery/Forceps delivery

Spontaneous/induced:

Meconium stained amniotic fluid: YES/NO

Apgar score: At 1minute: At 5minutes:

Significant perinatal events:

**NEONATAL FACTORS:**

1. FEATURES SUGGESTIVE OF SEPSIS

Respiratory distress: PRESENT / ABSENT

Hypoglycaemia: PRESENT / ABSENT

Lethargy: PRESENT / ABSENT

Excessive cry: PRESENT / ABSENT

Refusal of feeds: PRESENT / ABSENT

Temperature instability: PRESENT / ABSENT,

IF PRESENT DETAILS ---------

Seizures: PRESENT / ABSENT

2. FEATURES SUGGESTIVE OF BLEEDING:

External hemorrhage: PRESENT / ABSENT

IF PRESENT DETAILS ---------

Petechaie/purpura: PRESENT / ABSENT

Gastrointestinal bleed: PRESENT / ABSENT

Seizures: PRESENT / ABSENT

3. RESPIRATORY DISTRESS SYNDROME: YES / NO
4. BIRTH ASPHYXIA: YES / NO
5. PERSISTENT PULMONARY HYPERTENSION: YES / NO
6. CONGENITAL INFECTIONS: YES / NO
7. DISSEMINATED INTRAVASCULAR COAGULATION: YES / NO
8. NECROTISING ENTEROCOLITIS: YES / NO

**LABORATORY DETAILS:**

Haemoglobin:

Total leucocyte count:----------

Differential count: Neutrophils:

Lymphocytes:

Eosinophils:

Platelet count:

Absolute eosinophil count:

**If needed:**

CRP:

Coagulation profile: PT:

APTT:

INR:

Blood culture and sensitivity:

**If needed:**

CSF analysis:

Neurosonogram:

CT brain:

Direct coombs test:

**SEVERITY OF THROMBOCYTOPENIA:**

MILD: 100,000 – 150,000/$mm^3$

MODERATE: 50,000-99,000/$mm^3$

SEVERE: <50,000/$mm^3$

**OUTCOMES:**

H/O ANY BLOOD TRANSFUSION GIVEN: YES/NO

1. PRBC-No of units:
2. PLATELETS-No of units:

DURATION OF HOSPITAL:

1. NICU stay:
2. Mother's side:

OUTCOME:

1. Death/discharge:

**OTHER INTERVENTION:**

IV antibiotics: YES/NO IF YES, DETAILS----------

IV Immunoglobulin: YES/NO IF YES, DETAILS----------

Steroids: YES/NO IF YES, DETAILS----------

**PLATELET COUNT AT DISCHARGE:**

# INFORMED CONSENT FORM

I Mr./Mrs. _____ have been explained in my own understandable language, that I will be included in a study which is **RISK FACTORS, SEVERITY AND OUTCOME AMONG NEONATES WITH THROMBOCYTOPENIA ADMITTED IN NEONATAL INTENSIVE CARE UNIT- A PROSPECTIVE COHORT STUDY.**

I have been explained that my clinical findings, investigations, postoperative findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary, and I can withdraw from the study any time and this will not affect my relation with my doctor or the treatment for my ailment.

I have been explained about the interventions needed possible benefits and adversities due to interventions, in my own understandable language.

I have understood that all my details found during the study are kept confidential and while publishing or sharing of the findings, my details will be masked.

I have principal investigator mobile number for enquiries.

I in my sound mind give full consent to be added in the part of this study.

Signature of the patient:

Name:

Signature of the witness:

Name:

Relation to patient:

Date:

Place:

## PATIENT INFORMATION SHEET

**STUDY TITLE: RISK FACTORS, SEVERITY AND OUTCOME AMONG NEONATES WITH THROMBOCYTOPENIA ADMITTED IN NEONATAL INTENSIVE CARE UNIT- A PROSPECTIVE COHORT STUDY**

Principal investigator: Dr Ankem Praveen / Dr. Sudha Reddy V R

I Dr. Ankem Praveen , Post graduate student in Department of Paediatrics at Sri Devraj Urs Medical College, will be conducting a study titled **RISK FACTORS, SEVERITY AND OUTCOME AMONG NEONATES WITH THROMBOCYTOPENIA ADMITTED IN NEONATAL INTENSIVE CARE UNIT- A PROSPECTIVE COHORT STUDY,** my dissertation under the guidance of Dr Sudha Reddy V R , Professor and Head the of Department of Paediatrics. The participants of this study i.e. include 103 neonates who are with thrombocytopenia, admitted in the neonatal intensive care unit.

You will not be paid any financial compensation for the participation of your neonate in this research project including investigations.

The investigations performed in neonatal intensive care unit (NICU) as part of management- CBC, and other investigations will be performed as and when required- CRP, Blood culture and sensitivity, Renal function tests, Serum electrolytes, Coagulation profile, Chest X-ray, Neurosonogram, CT Brain.

All the data will be kept confidential and will be used only for research purpose by this institution. You are free to provide consent for the participation of your child in this study. You can also withdraw your child from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

Name and Signature of the Principal Investigator
Dr. Ankem Praveen
Date-

## ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ನಾನು ಶ್ರೀ / ಶ್ರೀ. _____ ಅನ್ನು ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಇದು ನವಜಾತ ಶಿಶು ತೀವ್ರ ನಿಗಾ ಘಟಕ ಯುನಿಟ್‌ನಲ್ಲಿ ಒಪ್ಪಿಕೊಂಡಿರುವ ಥ್ರಂಬೋಸೈಟೋಪೆನಿಯಾದೊಂದಿಗೆ ನವಜಾತ ಶಿಶುಗಳಲ್ಲಿ ಅಪಾಯಕಾರಿ ಅಂಶಗಳು, ತೀವ್ರತೆ ಮತ್ತು ಹೊರಹೊಮ್ಮುವಿಕೆ- ಒಂದು ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲಾಗುವುದು.

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಆವಿಷ್ಕಾರಗಳು, ತನಿಖೆಗಳು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಗೆ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ನನ್ನ ಸ್ವಂತ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ, ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಕಾರಣದಿಂದಾಗಿ ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಪ್ರತಿಕೂಲತೆಗಳ ಅಗತ್ಯವಿರುವ ಮಧ್ಯಸ್ಥಿಕೆಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಕಂಡುಬರುವ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗಿದೆ ಮತ್ತು ಸಂಶೋಧನೆಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಅಥವಾ ಹಂಚಿಕೊಳ್ಳುವಾಗ, ನನ್ನ ವಿವರಗಳನ್ನು ಮರೆಮಾಚಲಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ವಿಚಾರಣೆಗಾಗಿ ನನ್ನ ಬಳಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ ಇದೆ.

ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ನನ್ನ ಸಂಪೂರ್ಣ ಮನಸ್ಸಿನಲ್ಲಿ ನಾನು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ:

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ:

ಹೆಸರು:

ರೋಗಿಗೆ ಸಂಬಂಧ:

ದಿನಾಂಕ:

ಸ್ಥಳ:

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ನವಜಾತ ಶಿಶು ತೀವ್ರ ನಿಗಾ ಘಟಕ ಯುನಿಟ್‌ನಲ್ಲಿ ಒಪ್ಪಿಕೊಂಡಿರುವ ಥ್ರಂಬೋಸೈಟೋಪೆನಿಯಾದೊಂದಿಗೆ ನವಜಾತ ಶಿಶುಗಳಲ್ಲಿ ಅಪಾಯಕಾರಿ ಅಂಶಗಳು, ತೀವ್ರತೆ ಮತ್ತು ಹೊರಹೊಮ್ಮುವಿಕೆ- ಒಂದು ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಅಂಕೇಮ್ ಪ್ರವೀಣ್ / ಡಾ.ಸುಧಾ ರೆಡ್ಡಿ ವಿ ಆರ್

ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜಿನ ಪೀಡಿಯಾಟ್ರಿಕ್ಸ್ ವಿಭಾಗದ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ ಡಾ. ಅಂಕೆಮ್ ಪ್ರವೀಣ್, ನಿಯೋನಾಟಲ್ ಇಂಟರೆಂಟ್ ಕೋರ್ಟ್ನಲ್ಲಿನ ಥ್ರೊಂಬೊಸೈಟೊಪೆನಿಯಾ ಅಡ್ಮಿಟ್ಡ್ ನಿಯೋನೇಟ್ಸ್, ರಿಸ್ಕ್ ಫ್ಯಾಕ್ಟರ್ಸ್, ಸೆವೆರಿಟಿ ಮತ್ತು ಹೊರಗಿನ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ. ಪೀಡಿಯಾಟ್ರಿಕ್ಸ್ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕ ಮತ್ತು ಮುಖ್ಯಸ್ಥ ಡಾ.ಸುಧಾ ರೆಡ್ಡಿ ವಿ.ಆರ್ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ. ಈ ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವವರು ಅಂದರೆ, ನವಜಾತ ಶಿಶುವಿನ ತೀವ್ರ ನಿಗಾ ಘಟಕದಲ್ಲಿ ದಾಖಲಾದ ಥ್ರಂಬೋಸೈಟೋಪೆನಿಯಾದ 103 ನಿಯೋನೇಟ್‌ಗಳನ್ನು ಒಳಗೊಂಡಿದೆ.

ತನಿಖೆಗಳು ಸೇರಿದಂತೆ ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ನಿಮ್ಮ ನವಜಾತ ಶಿಶುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪರಿಹಾರವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ನಿರ್ವಹಣೆಯ ಭಾಗವಾಗಿ ನವಜಾತ ತೀವ್ರ ನಿಗಾ ಘಟಕದಲ್ಲಿ (ಎನ್‌ಐಸಿಯು) ನಡೆಸಲಾದ ತನಿಖೆಗಳು ಮತ್ತು ಸಿಬಿಪಿ, ಮತ್ತು ಇತರ ತನಿಖೆಗಳನ್ನು ಅಗತ್ಯವಿದ್ದಾಗ ಮತ್ತು ಸಿಆರ್‌ಪಿ, ರಕ್ತ ಸಂಸ್ಕೃತಿ ಮತ್ತು ಸೂಕ್ಷ್ಮತೆ, ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳು, ಸೀರಮ್ ವಿದ್ಯುದ್ವಿಚ್ ಛೇದ್ಯಗಳು, ಹೆಪ್ಪುಗಟ್ಟುವಿಕೆ ಪ್ರೊಫೈಲ್, ಎದೆಯ ಎಕ್ಸರೆ , ನ್ಯೂರೋಸೊನೊಗ್ರಾಮ್, ಸಿಟಿ ಬ್ರೈನ್.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನೀವು ಒಪ್ಪಿಗೆ ನೀಡಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವನ್ನು ಅಧ್ಯಯನದಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ನೀವು ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸುವುದರಿಂದ ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ನೀವು ಪೂರ್ವಾಗ್ರಹ ಮಾಡುವುದಿಲ್ಲ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ಡಾ.ಅಂಕೇಮ್ ಪ್ರವೀಣ್

ದಿನಾಂಕ-

## KEY TO MASTER CHART

| GLOSSARY | ABBREVIATIONS |
|---|---|
| **Ab** | Absent |
| **Abn** | Abnormal |
| **Age** | Age in days |
| **APGAR(5 MIN)** | APGAR AT 5 MINUTES |
| **BC** | Blood culture |
| **BT** | Blood transfusion |
| **CD** | Caesarean delivery |
| **CPC** | Change in platelet count |
| **CS** | Candida species |
| **D** | Discharge |
| **DHS** | Duration of hospital stay |
| **E** | Expired |
| **E COLI** | Escherichia coli |
| **F** | Female |
| **FD** | Forceps delivery |
| **GA** | Gestational age in weeks |
| **GNB** | Gram negative bacilli |
| **GPC** | Gram positive cocci |
| **KS** | Klebsiella species |
| **M** | Male |
| **Mi** | Mild |
| **Md** | Moderate |
| **MOD** | Mode of delivery |
| **MRF : ANEMIA** | Maternal risk factor anemia |
| **: DI** | Drug intake by mother that causes neonatal thrombocytopenia |
| **: GDM** | Gestational diabetes mellitus |
| **: PIH** | Pregnancy induced hypertension |
| **: PROM** | Premature rupture of membranes |
| **: RH.I** | Rhesus incompatibility |
| **: SLE** | Systemic lupus erythematosus |

| | |
|---|---|
| **N** | NO |
| **NCS** | Non candida species |
| **NR** | Normal |
| **NRF : BA** | Neonatal risk factor birth asphyxia |
| **: CI** | Congenital infections |
| **: DIC** | Disseminated intravascular coagulation |
| **: NEC** | Necrotizing enterocolitis |
| **: PPH** | Persistent pulmonary hypertension |
| **: RDS** | Respiratory distress syndrome |
| **: SEP** | Sepsis |
| **PCA** | Platelet count at admission |
| **PCD** | Platelet count at discharge |
| **PL.T** | Platelet transfusion |
| **PT/APTT** | Prothrombin time/Activated partial thromboplastin time |
| **S** | Severe |
| **SI.No** | Serial number |
| **TLC** | Total leukocyte count |
| **TN(ADMISSION)** | Thrombocytopenia at admission |
| **TN(DISCHARGE)** | Thrombocytopenia at discharge |
| **W** | Weeks |
| **Y** | Yes |

# MASTER CHART

| Sl.No | Name | Age (Days) | Gender | MOD | GA (Weeks) | GA | MRF: PROM | MRF: PIH | MRF: GDM | MRF: ANEMIA | MRF: SLE | MRF: D.I | MRF: RH.I | APGAR (5 MIN) | MSL | PCA (Lacs) | TN (ADMISSION) | TLC ($x10^3/mm^3$) | NRF: SEP | NRF: RDS | NRF: BA | NRF: PPH | NRF: CI | NRF: DIC | NRF: NEC | BC | PT/APTT(Abn) | BT | PLT | DHS (Days) | PCD (Lacs) | TN (Discharge) | CPC | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Venkatalakshmi | 2 | F | VD | 38 | ≥37 W | N | Y | N | N | N | Y | N | 9 | N | 1.37 | Mi | 9.5 | Y | N | N | N | N | N | N | | N | N | N | 6 | 1.84 | Ab | 0.47 | D |
| 2 | Khusboo | 1 | M | VD | 34 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.12 | Mi | 10.7 | Y | N | N | N | N | N | N | | N | N | N | 6 | 1.6 | Ab | 0.48 | D |
| 3 | Sravani | 1 | F | VD | 35 | <37 W | Y | N | N | N | N | N | N | 9 | N | 0.86 | Md | 9.8 | N | Y | N | N | N | N | N | | N | Y | N | 41 | 1.68 | Ab | 0.82 | D |
| 4 | Prathiba Deepa | 1 | F | FD | 34 | <37 W | N | N | Y | N | N | Y | N | 8 | N | 1.17 | Mi | 25 | N | Y | N | N | N | N | N | | N | N | N | 7 | 1.68 | Ab | 0.51 | D |
| 5 | Deepa Twin 1 | 1 | M | CD | 36 | <37 W | Y | N | N | N | N | N | N | 9 | N | 1.21 | Mi | 8.14 | Y | N | N | N | N | N | N | | N | N | N | 24 | 2.38 | Ab | 1.17 | D |
| 6 | Archana | 1 | F | CD | 31 | <37 W | Y | N | N | N | N | N | N | 9 | N | 0.94 | Md | 9.3 | Y | N | N | N | N | N | N | | N | N | N | 10 | 1.89 | Ab | 0.95 | D |
| 7 | Mubeena | 3 | F | CD | 39 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 0.69 | Md | 4.7 | Y | N | N | N | N | N | N | | N | N | N | 10 | 1.03 | Mi | 0.34 | D |
| 8 | Shabreen | 1 | F | CD | 28 | <37 W | N | Y | N | N | N | N | N | 8 | N | 0.98 | Md | 9.6 | N | Y | N | N | N | N | N | | N | N | N | 4 | 3.33 | Ab | 2.35 | D |
| 9 | Manjula | 1 | F | CD | 36 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.32 | Mi | 12.9 | N | Y | N | N | N | N | N | | N | N | N | 15 | 2.6 | Ab | 1.28 | D |
| 10 | Haritha | 1 | M | VD | 36 | <37 W | N | N | N | Y | N | N | N | 9 | N | 1.37 | Mi | 11.6 | Y | N | N | N | N | N | N | NCS | N | Y | N | 16 | 1.79 | Ab | 0.42 | D |
| 11 | Sushma | 1 | M | CD | 33 | <37 W | N | Y | N | N | N | Y | N | 8 | N | 1.47 | Mi | 5.2 | Y | N | N | N | N | N | N | NCS | N | N | N | 16 | 2.32 | Ab | 0.85 | D |
| 12 | Pruthvi | 5 | M | CD | 36 | <37 W | N | Y | N | N | N | N | N | 7 | N | 0.57 | Md | 9.1 | Y | N | N | N | N | N | N | | N | N | N | 12 | 2.37 | Ab | 1.8 | D |
| 13 | Asha Rani | 1 | M | CD | 33 | <37 W | Y | N | N | N | N | N | N | 9 | N | 1.15 | Mi | 6.6 | N | Y | N | N | N | N | N | | N | N | N | 23 | 4.3 | Ab | 3.15 | D |
| 14 | Sirisha B.A | 1 | M | CD | 38 | ≥37 W | N | N | N | Y | N | N | N | 9 | Y | 1.06 | Mi | 9.9 | Y | N | N | N | N | N | N | | N | N | N | 12 | 1.7 | Ab | 0.64 | D |
| 15 | Nethra | 1 | M | VD | 30 | <37 W | Y | N | N | N | N | N | N | 8 | N | 1.37 | Mi | 7.5 | Y | N | N | N | N | N | N | GPC | N | N | N | 26 | 2.6 | Ab | 1.23 | D |
| 16 | Hemavath | 1 | M | CD | 39 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 1.45 | Mi | 8.2 | Y | N | N | N | N | N | N | | N | N | N | 15 | 2.1 | Ab | 0.65 | D |
| 17 | Shyamala | 1 | M | VD | 36 | <37 W | N | Y | N | N | N | Y | N | 8 | N | 0.85 | Md | 7.1 | N | Y | N | N | N | N | N | | N | N | N | 11 | 1.57 | Ab | 0.72 | D |
| 18 | Hemavathi.P | 1 | M | VD | 32 | <37 W | Y | N | N | N | N | N | N | 8 | N | 1.36 | Mi | 5.3 | N | Y | N | N | N | N | N | | N | N | N | 15 | 1.47 | Mi | 0.11 | D |
| 19 | Dhanalakshmi | 1 | M | CD | 38 | ≥37 W | Y | N | N | N | N | N | N | 8 | N | 1.44 | Mi | 14.9 | N | N | Y | N | N | N | N | | N | N | N | 14 | 2.27 | Ab | 0.83 | D |
| 20 | Pooja.C | 1 | M | CD | 36 | <37 W | Y | N | N | N | N | N | N | 8 | Y | 1.36 | Mi | 11.3 | N | N | Y | N | N | N | N | | N | N | N | 8 | 1.79 | Ab | 0.43 | D |
| 21 | Deepika.K | 1 | M | VD | 34 | <37 W | Y | N | N | N | N | Y | N | 8 | N | 1.05 | Mi | 2.1 | Y | N | N | N | N | N | N | | N | N | N | 16 | 1.91 | Ab | 0.86 | D |
| 22 | Raziya Rabbni | 1 | M | CD | 36 | <37 W | N | N | Y | N | N | Y | N | 9 | N | 0.94 | Md | 12.9 | Y | N | N | N | N | N | N | | N | N | N | 7 | 1.26 | Mi | 0.32 | D |
| 23 | Latha | 1 | F | CD | 39 | ≥37 W | Y | N | N | N | N | N | N | 8 | Y | 1.41 | Mi | 44.6 | N | N | N | N | N | Y | N | GNB | Y | Y | Y | 5 | 0.08 | S | -1.33 | E |
| 24 | Reshma Begum | 1 | F | VD | 32 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.42 | Mi | 7.4 | N | Y | N | N | N | N | N | | N | N | N | 13 | 1.81 | Ab | 0.39 | D |
| 25 | Ruksar Fathima | 1 | F | CD | 30 | <37 W | N | Y | N | N | N | N | N | 7 | N | 1.17 | Mi | 13.3 | N | N | Y | N | N | N | N | | N | Y | Y | 3 | 0.41 | S | -0.76 | E |
| 26 | Sunitha Bai | 1 | F | CD | 37 | ≥37 W | N | Y | N | N | N | Y | N | 6 | N | 1.35 | Mi | 21.1 | N | N | Y | N | N | N | N | | N | N | N | 8 | 1.5 | Ab | 0.15 | D |
| 27 | Chaitra N.G | 1 | F | CD | 38 | ≥37 W | N | Y | N | N | N | Y | N | 9 | N | 0.93 | Md | 6.3 | Y | N | N | N | N | N | N | | N | N | N | 15 | 1.77 | Ab | 0.84 | D |
| 28 | Kavya | 1 | F | CD | 39 | ≥37 W | N | Y | N | N | N | Y | N | 9 | N | 0.96 | Md | 12.1 | Y | N | N | N | N | N | N | | N | N | N | 12 | 1.38 | Mi | 0.42 | D |
| 29 | Sindhu.N | 1 | F | CD | 39 | ≥37 W | N | N | N | Y | N | N | N | 7 | N | 1.41 | Mi | 4.1 | N | N | Y | N | N | N | N | | N | N | N | 14 | 1.11 | Mi | -0.3 | D |
| 30 | Divya Shree M | 1 | M | CD | 29 | <37 W | N | Y | N | N | N | N | N | 6 | N | 0.98 | Md | 4.3 | N | N | Y | N | N | N | N | | N | Y | Y | 54 | 3.92 | Ab | 2.94 | D |
| 31 | Arathi | 1 | M | VD | 29 | <37 W | N | Y | N | N | N | N | N | 7 | N | 1.45 | Mi | 8.4 | N | Y | N | N | N | N | N | | N | Y | N | 29 | 2.03 | Ab | 0.58 | D |
| 32 | Gayathri | 1 | F | CD | 39 | ≥37 W | Y | N | N | N | N | N | N | 6 | N | 1.21 | Mi | 27.5 | N | N | Y | N | N | N | N | | N | Y | N | 31 | 3.29 | Ab | 2.08 | D |
| 33 | Geethanjali | 1 | F | CD | 38 | ≥37 W | N | N | Y | N | N | Y | N | 9 | N | 1.43 | Mi | 20.8 | Y | N | N | N | N | N | N | | N | N | N | 15 | 2.53 | Ab | 1.1 | D |
| 34 | Shilpa | 1 | M | CD | 40 | ≥37 W | N | Y | N | N | N | N | N | 9 | N | 1.19 | Mi | 11.8 | Y | N | N | N | N | N | N | | N | N | N | 15 | 3.35 | Ab | 2.16 | D |
| 35 | Lavanya Twin 1 | 1 | F | CD | 38 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 0.69 | Md | 13.8 | Y | N | N | N | N | N | N | NCS | N | N | N | 15 | 2.11 | Ab | 1.42 | D |
| 36 | Mamatha K | 1 | M | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | 9 | Y | 1.12 | Mi | 24.9 | Y | N | N | N | N | N | N | NCS | N | N | N | 16 | 1.31 | Mi | 0.19 | D |
| 37 | Bhavani V | 1 | F | CD | 33 | <37 W | N | N | N | N | N | Y | N | 8 | N | 0.61 | Md | 32.1 | N | Y | N | N | N | N | N | | N | N | N | 23 | 3.8 | Ab | 3.19 | D |
| 38 | Naziya | 1 | M | CD | 37 | ≥37 W | N | Y | N | N | N | N | Y | 6 | N | 1.29 | Mi | 12.9 | N | N | Y | N | N | N | N | | N | N | N | 13 | 1.7 | Ab | 0.41 | D |
| 39 | Lakshmidevi | 2 | F | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | | N | 1.37 | Mi | 14.3 | N | N | N | N | N | N | Y | | N | N | N | 11 | 1.84 | Ab | 0.47 | D |
| 40 | Samyuktha | 1 | F | CD | 32 | <37 W | N | Y | N | N | N | Y | N | 6 | N | 1.21 | Mi | 17.5 | N | N | Y | N | N | N | N | | N | Y | N | 31 | 4.68 | Ab | 3.47 | D |
| 41 | Lakshmi | 1 | M | VD | 34 | <37 W | Y | N | N | N | N | N | N | 9 | N | 1.26 | Mi | 10.6 | N | Y | N | N | N | N | N | | N | N | N | 7 | 1.54 | Ab | 0.28 | D |
| 42 | Divya | 1 | F | VD | 34 | <37 W | N | N | Y | N | N | Y | N | 8 | N | 1.14 | Mi | 25 | N | Y | N | N | N | N | N | | N | N | N | 8 | 1.62 | Ab | 0.48 | D |
| 43 | Shabana Taj | 1 | M | CD | 34 | <37 W | Y | N | N | N | N | N | N | 8 | N | 1.31 | Mi | 6.3 | Y | N | N | N | N | N | N | | N | N | N | 15 | 2.47 | Ab | 1.16 | D |
| 44 | Ramya | 1 | M | CD | 38 | ≥37 W | Y | N | N | N | N | N | N | 9 | Y | 1.36 | Mi | 24.9 | Y | N | N | N | N | N | N | NCS | N | N | N | 14 | 2.24 | Ab | 0.88 | D |
| 45 | Nayana | 1 | M | CD | 35 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.02 | Mi | 11.6 | Y | N | N | N | N | N | N | | N | Y | N | 26 | 5.06 | Ab | 4.04 | D |
| 46 | Prema | 1 | F | CD | 38 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 1.17 | Mi | 11.8 | Y | N | N | N | N | N | N | | N | N | N | 8 | 1.84 | Ab | 0.67 | D |
| 47 | Lahari | 1 | F | CD | 39 | ≥37 W | N | Y | N | N | N | N | N | 9 | N | 0.78 | Md | 13.8 | Y | N | N | N | N | N | N | NCS | N | N | N | 11 | 2.11 | Ab | 1.33 | D |
| 48 | Chinna | 1 | F | CD | 35 | <37 W | Y | N | N | N | N | N | N | 9 | N | 0.7 | Md | 17.2 | Y | N | N | N | N | N | N | KS | N | Y | N | 26 | 1.53 | Ab | 0.83 | D |
| 49 | Husna Taj | 1 | F | VD | 38 | ≥37 W | N | Y | N | N | N | N | N | 9 | Y | 1.39 | Mi | 26 | Y | N | N | N | N | N | N | | N | N | N | 17 | 1.53 | Ab | 0.14 | D |
| 50 | Swetha Sree | 1 | F | VD | 32 | <37 W | N | Y | N | N | N | Y | N | 8 | N | 0.66 | Md | 15.2 | N | N | N | N | N | Y | N | | Y | Y | Y | 6 | 0.52 | Md | -0.14 | E |
| 51 | Anjali | 1 | F | CD | 30 | <37 W | N | Y | N | Y | N | Y | N | 8 | N | 0.72 | Md | 18.2 | N | Y | N | N | N | N | N | | N | Y | Y | 3 | 0.48 | S | -0.24 | E |
| 52 | Varalakshmi | 1 | F | VD | 38 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 1.19 | Mi | 7.6 | Y | N | N | N | N | N | N | | N | N | N | 22 | 1.04 | Mi | -0.15 | D |
| 53 | Sandya | 1 | F | VD | 34 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.21 | Mi | 18 | Y | N | N | N | N | N | N | GPC | N | N | N | 26 | 1.64 | Ab | 0.43 | D |

| Sl.No | Name | Age (Days) | Gender | MOD | GA (Weeks) | GA | MRF: PROM | MRF: PIH | MRF: GDM | MRF: ANEMIA | MRF: SLE | MRF: D.I | MRF: RH.I | APGAR (5 MIN) | MSL | PCA (Lacs) | TN (ADMISSION) | TLC ($x10^3/mm^3$) | NRF: SEP | NRF: RDS | NRF: BA | NRF: PPH | NRF: CI | NRF: DIC | NRF: NEC | BC | PT/APTT(Abn) | BT | PLT | DHS (Days) | PCD (Lacs) | TN (Discharge) | CPC | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 54 | Zaheed Taj | 1 | F | CD | 36 | <37 W | N | N | Y | N | N | Y | N | 9 | N | 0.11 | S | 7 | Y | N | N | N | N | N | N | | N | N | Y | 18 | 2.75 | Ab | 2.64 | D |
| 55 | Varalakshmi G | 1 | M | CD | 30 | <37 W | N | Y | N | N | N | N | N | 9 | N | 0.94 | Md | 14.2 | N | Y | N | N | N | N | N | | N | N | N | 3 | 1.04 | Mi | 0.1 | E |
| 56 | Leela | 1 | M | CD | 35 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 0.84 | Md | 8.24 | Y | N | N | N | N | N | N | CS | N | Y | Y | 16 | 0.64 | Md | -0.2 | E |
| 57 | Bi Bi Ameena Kouser | 1 | M | CD | 39 | ≥37 W | N | Y | N | N | N | N | N | 9 | N | 1.34 | Mi | 7.3 | Y | N | N | N | N | N | N | | N | N | N | 9 | 1.45 | Mi | 0.11 | D |
| 58 | Pushpa | 1 | F | CD | 38 | ≥37 W | N | N | Y | N | N | Y | N | 9 | N | 1.3 | Mi | 12.3 | N | N | N | N | N | N | Y | | N | N | N | 8 | 1.59 | Ab | 0.29 | D |
| 59 | Divya | 1 | M | VD | 34 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.26 | Mi | 14.2 | N | Y | N | N | N | N | N | | N | N | N | 14 | 1.51 | Ab | 0.25 | D |
| 60 | Asha B V | 1 | F | VD | 34 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.37 | Mi | 14.6 | N | Y | N | N | N | N | N | | N | N | N | 23 | 2.37 | Ab | 1 | D |
| 61 | Bhargavi | 1 | M | VD | 38 | ≥37 W | Y | N | N | N | N | N | N | 8 | N | 1.23 | Mi | 13.1 | Y | N | N | N | N | N | N | | N | N | N | 6 | 2.12 | Ab | 0.89 | D |
| 62 | Kavya Kadam Twin 1 | 1 | F | CD | 35 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.31 | Mi | 11 | N | Y | N | N | N | N | N | | N | N | N | 19 | 4.44 | Ab | 3.13 | D |
| 63 | Kavya Kadam Twin 2 | 1 | F | CD | 35 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.26 | Mi | 8.2 | Y | N | N | N | N | N | N | KS | N | N | N | 19 | 1.88 | Ab | 0.62 | D |
| 64 | Zabi Pasha | 1 | F | VD | 42 | ≥37 W | N | N | N | Y | N | N | N | 7 | Y | 1.22 | Mi | 26.3 | N | N | Y | N | N | N | N | | N | N | N | 7 | 0.75 | Md | -0.47 | D |
| 65 | Rumana | 1 | M | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | 9 | N | 1.42 | Mi | 18.9 | Y | N | N | N | N | N | N | | N | N | N | 10 | 2.5 | Ab | 1.08 | D |
| 66 | Prema V | 1 | F | VD | 30 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.16 | Mi | 13 | N | Y | N | N | N | N | N | | N | N | N | 36 | 2.9 | Ab | 1.74 | D |
| 67 | Naveena N | 2 | M | VD | 36 | <37 W | N | N | Y | N | N | Y | N | | N | 0.28 | S | 7.6 | Y | N | N | N | N | N | N | | N | N | Y | 14 | 1.13 | Mi | 0.85 | D |
| 68 | Amala Devi Twin 1 | 1 | M | CD | 33 | <37 W | Y | N | N | N | N | N | Y | 8 | N | 1.47 | Mi | 6.8 | N | Y | N | N | N | N | N | | N | N | N | 16 | 2.13 | Ab | 0.66 | D |
| 69 | Arshiya | 1 | F | CD | 35 | <37 W | N | Y | N | N | N | N | N | 8 | N | 1.12 | Mi | 13.7 | N | N | Y | N | N | N | N | | N | N | N | 9 | 2.68 | Ab | 1.56 | D |
| 70 | Zaiba Siddiqa | 1 | M | CD | 35 | <37 W | N | N | Y | N | N | Y | N | 9 | N | 0.94 | Md | 10.8 | N | N | N | N | N | N | Y | | N | N | N | 11 | 5.18 | Ab | 4.24 | D |
| 71 | Vedha | 1 | M | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | 9 | N | 1.14 | Mi | 8.8 | Y | N | N | N | N | N | N | | N | N | N | 10 | 2.39 | Ab | 1.25 | D |
| 72 | Pooja | 1 | F | CD | 39 | ≥37 W | N | Y | N | N | N | N | N | 8 | N | 1.13 | Mi | 11 | Y | N | N | N | N | N | N | | N | N | N | 8 | 1.56 | Ab | 0.43 | D |
| 73 | Rukmani | 1 | M | VD | 39 | ≥37 W | N | Y | N | N | N | N | N | 7 | Y | 1.16 | Mi | 15.9 | N | N | Y | N | N | N | N | | N | Y | N | 15 | 2.18 | Ab | 1.02 | D |
| 74 | Mala N | 1 | F | CD | 35 | <37 W | N | Y | N | N | N | Y | N | 7 | N | 1.42 | Mi | 47.7 | N | N | N | N | N | Y | N | | Y | Y | N | 21 | 1.19 | Mi | -0.23 | D |
| 75 | Dhanalakshmi M | 1 | F | CD | 32 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.15 | Mi | 18.6 | Y | N | N | N | N | N | N | | N | N | N | 13 | 1.4 | Mi | 0.25 | D |
| 76 | Keerthi | 1 | M | CD | 36 | <37 W | N | Y | N | N | N | Y | N | 8 | Y | 1.4 | Mi | 20.9 | N | Y | N | N | N | N | N | | N | N | N | 10 | 0.97 | Md | -0.43 | D |
| 77 | Ranjitha | 1 | F | CD | 35 | <37 W | N | N | N | Y | N | N | N | 9 | N | 1.02 | Mi | 10 | Y | N | N | N | N | N | N | | N | N | N | 10 | 1.36 | Mi | 0.34 | D |
| 78 | Kalyani | 1 | F | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | 9 | Y | 1.4 | Mi | 17.7 | Y | N | N | N | N | N | N | | N | N | N | 14 | 1.36 | Mi | -0.04 | D |
| 79 | Anitha | 1 | F | CD | 38 | ≥37 W | N | Y | N | N | N | N | N | 7 | N | 1.11 | Mi | 11 | N | N | Y | N | N | N | N | | N | N | N | 11 | 0.93 | Md | -0.18 | D |
| 80 | Manjunath | 1 | M | VD | 38 | ≥37 W | N | Y | N | N | N | N | N | 7 | Y | 0.73 | Md | 11.1 | N | N | Y | N | N | N | N | | N | N | N | 5 | 1.38 | Mi | 0.65 | D |
| 81 | Nilima | 1 | M | CD | 33 | <37 W | N | Y | N | N | N | N | N | 7 | N | 0.78 | Md | 12.6 | N | N | Y | N | N | N | N | | N | N | N | 14 | 1.76 | Ab | 0.98 | D |
| 82 | Roopa K | 1 | M | VD | 34 | <37 W | Y | N | N | N | N | N | N | 9 | N | 1.32 | Mi | 11.2 | Y | N | N | N | N | N | N | | N | N | N | 10 | 1.56 | Ab | 0.24 | D |
| 83 | Shylamma | 1 | M | CD | 40 | ≥37 W | N | Y | N | N | N | N | N | 9 | Y | 1.14 | Mi | 18.7 | Y | N | N | N | N | N | N | | N | N | N | 16 | 0.63 | Md | -0.51 | D |
| 84 | Yellamma | 1 | M | CD | 36 | <37 W | Y | N | N | N | N | Y | N | 7 | Y | 1.45 | Mi | 20.9 | N | Y | N | N | N | N | N | | N | N | N | 10 | 0.97 | Md | -0.48 | D |
| 85 | Monika | 1 | F | CD | 35 | <37 W | N | Y | N | N | N | N | N | 9 | N | 0.93 | Md | 13.2 | N | Y | N | N | N | N | N | | N | N | N | 13 | 1.3 | Mi | 0.37 | D |
| 86 | Ruksar | 1 | F | VD | 33 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.21 | Mi | 18 | N | Y | N | N | N | N | N | | N | N | N | 17 | 1.13 | Mi | -0.08 | D |
| 87 | Deepa N | 1 | M | VD | 33 | <37 W | N | N | N | Y | N | N | N | 9 | N | 1.22 | Mi | 9.4 | N | Y | N | N | N | N | N | | N | N | N | 16 | 1.71 | Ab | 0.49 | D |
| 88 | Zareena Taj | 1 | F | CD | 32 | <37 W | N | Y | N | N | N | Y | N | 9 | Y | 1.25 | Mi | 8.9 | Y | N | N | N | N | N | N | | N | N | N | 21 | 1.51 | Ab | 0.26 | D |
| 89 | Gangaratna | 1 | M | CD | 36 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 0.9 | Md | 23.3 | Y | N | N | N | N | N | N | | N | N | N | 10 | 2.25 | Ab | 1.35 | D |
| 90 | Amreena | 1 | M | VD | 39 | ≥37 W | N | Y | N | N | N | N | Y | 9 | N | 0.49 | S | 8.7 | Y | N | N | N | N | N | N | | N | N | N | 13 | 1.73 | Ab | 1.24 | D |
| 91 | Bhavya | 1 | M | VD | 38 | ≥37 W | N | N | N | Y | N | N | N | 9 | N | 0.68 | Md | 12.8 | Y | N | N | N | N | N | N | | N | N | N | 13 | 1.38 | Mi | 0.7 | D |
| 92 | Tina | 1 | M | CD | 38 | ≥37 W | Y | N | N | N | N | N | N | 9 | N | 0.68 | Md | 16.2 | N | N | N | N | N | N | Y | | N | N | N | 7 | 1.58 | Ab | 0.9 | D |
| 93 | Mangala | 1 | M | VD | 33 | <37 W | Y | N | N | N | N | N | N | 9 | N | 0.64 | Md | 11.4 | N | N | N | N | N | Y | N | | Y | Y | N | 12 | 0.58 | Md | -0.06 | E |
| 94 | Ranjitha | 1 | F | VD | 37 | ≥37 W | N | N | N | Y | N | N | N | 8 | N | 0.88 | Md | 12.2 | Y | N | N | N | N | N | N | GPC | N | N | N | 9 | 1.62 | Ab | 0.74 | D |
| 95 | Lalitha | 1 | M | CD | 38 | ≥37 W | N | Y | N | N | N | Y | N | 9 | N | 0.72 | Md | 12.6 | Y | N | N | N | N | N | N | KS | N | N | N | 12 | 0.41 | S | -0.31 | E |
| 96 | Shymala | 1 | M | CD | 38 | ≥37 W | N | Y | N | N | N | Y | N | 5 | N | 1.25 | Mi | 14.4 | N | N | Y | N | N | N | N | | N | Y | N | 23 | 4.39 | Ab | 3.14 | D |
| 97 | Swetha | 1 | F | CD | 34 | <37 W | N | Y | N | N | N | Y | N | 7 | N | 0.3 | S | 4.8 | N | N | Y | N | N | N | N | | N | N | N | 20 | 1.89 | Ab | 1.59 | D |
| 98 | Susmitha | 1 | F | VD | 32 | <37 W | N | Y | N | N | N | N | N | 6 | N | 0.42 | S | 12.6 | N | N | Y | N | N | N | N | | N | N | N | 8 | 0.36 | S | -0.06 | E |
| 99 | Latha A | 1 | M | CD | 38 | ≥37 W | N | N | Y | N | N | Y | N | 9 | N | 1.47 | Mi | 12.6 | Y | N | N | N | N | N | N | E COLI | N | N | N | 17 | 1.61 | Ab | 0.14 | D |
| 100 | Sameena Begum | 1 | F | CD | 32 | <37 W | N | Y | N | N | N | N | N | 6 | N | 1.4 | Mi | 5.7 | N | N | Y | N | N | N | N | | N | Y | Y | 53 | 1.27 | Mi | -0.13 | D |
| 101 | Bharathi Twin 1 | 1 | M | CD | 34 | <37 W | N | Y | N | N | N | Y | N | 9 | N | 1.4 | Mi | 13.1 | Y | N | N | N | N | N | N | | N | N | N | 14 | 2.41 | Ab | 1.01 | D |
| 102 | Vanitha S | 1 | F | CD | 35 | <37 W | N | N | N | Y | N | N | N | 9 | Y | 1.06 | Mi | 12.4 | N | Y | N | N | N | N | N | | N | N | N | 11 | 1.42 | Mi | 0.36 | D |
| 103 | Asma | 1 | F | CD | 33 | <37 W | N | Y | N | N | N | N | N | 9 | N | 1.4 | Mi | 11.4 | N | Y | N | N | N | N | N | | N | N | N | 17 | 1.89 | Ab | 0.49 | D |