

**“CLINICAL PROFILE AND SHORT-TERM OUTCOMES
OF NEONATAL HYPOGLYCEMIA IN NEONATAL
INTENSIVE CARE UNIT IN A TERTIARY CARE
HOSPITAL - PROSPECTIVE OBSERVATIONAL
STUDY”**

**A DISSERTATION SUBMITTED TO MUHS FOR THE DEGREE
OF M.D. PAEDIATRIC**

[REDACTED]

[REDACTED]

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A DISSERTATION SUBMITTED

TO

[REDACTED]

[REDACTED]

M.D. PAEDIATRIC

BY

[REDACTED]

UNDER THE GUIDANCE OF

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AUGUST [REDACTED]

NAME OF COURSE	:	M.D. PAEDIATRIC
NAME OF SUBJECT	:	PAEDIATRIC
ADMISSION YEAR/ ACADEMIC YEAR	:	██████████

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ABSTRACT

Background: Neonatal hypoglycaemia is one of the most common metabolic disturbances encountered in newborns, particularly in those with identifiable risk factors. If not promptly recognized and managed, it can lead to adverse neurodevelopmental outcomes.

Aim and Objectives: [REDACTED]

[REDACTED]

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Methods: This prospective observational study was conducted in the NICU of a tertiary care hospital and included 241 neonates diagnosed with hypoglycaemia. Data were collected on demographic variables, maternal and neonatal risk factors, clinical features, glucose levels, timing of detection, treatment modalities, and short-term outcomes.

Results: [REDACTED]

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[REDACTED]

Conclusion:

[REDACTED]

[REDACTED]

[REDACTED]

[REDACTED]

Keywords: Neonatal hypoglycaemia, prematurity, gestational diabetes, blood glucose, newborn, NICU, short-term outcome

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INTRODUCTION

Neonatal Hypoglycaemia

Neonatal hypoglycaemia represents the most frequently encountered metabolic abnormality in the neonatal period. It refers to a condition wherein the glucose concentration in a newborn's blood falls below the physiologically acceptable threshold, potentially leading to adverse neurological outcomes if not identified and managed promptly(1). Despite its prevalence, controversies persist regarding the exact definition, screening methods, and management due to the complexity of neonatal metabolic adaptations and variability in clinical presentations(2).

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Neonatal hypoglycaemia is a frequently occurring and potentially serious metabolic condition in newborns. The lack of consensus regarding diagnostic thresholds and the nonspecific nature of clinical symptoms make it a challenging entity to manage. Nevertheless, the use of operational thresholds provides a valuable clinical guide, particularly in the absence of clear-cut symptoms(16). Early identification, timely intervention, and appropriate follow-up are essential in minimizing the risk of permanent neurological damage and ensuring healthy developmental outcomes. Ongoing research aimed at refining diagnostic criteria and understanding the long-term impact of neonatal hypoglycaemia is vital for improving care standards globally(17).

AIM AND OBJECTIVES

AIM:

[REDACTED]

[REDACTED]

[REDACTED]

OBJECTIVES:

[REDACTED]

[REDACTED]

REVIEW OF LITERATURE

Prevalence and Burden in NICUs

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Global Prevalence

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Prevalence in India

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Clinical Significance and Implications

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Immediate Clinical Burden

- **Increased NICU Admissions:** Hypoglycaemia is a major contributor to NICU admissions, especially in preterm infants and those with other metabolic or

endocrine disorders. The necessity for continuous monitoring and management in NICU settings contributes to overcrowding, especially in tertiary care hospitals(33).

- **Prolonged Hospitalization:** Neonates with persistent or severe hypoglycaemia may require extended stays in the NICU, leading to increased healthcare costs and longer periods of parental separation. Studies show that prolonged hospitalization due to hypoglycaemia-related complications can increase the length of NICU stays, with significant implications for family dynamics and hospital resource utilization(34).
- **Increased Use of Medical Resources:** The treatment of neonatal hypoglycaemia often involves the use of glucose supplementation methods such as intravenous glucose or buccal dextrose gel, which not only require medical interventions but also frequent monitoring, including blood glucose checks, which places further strain on hospital resources. Additionally, high-risk infants may require additional investigations, such as metabolic screenings, to rule out underlying causes like congenital hyperinsulinism, further increasing the burden on NICU services(35).

Long-Term Clinical Burden

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- **Neurodevelopmental Delays:** Children who experience significant hypoglycaemia during the neonatal period are at risk of cognitive delays, motor impairments, and learning disabilities, which may necessitate lifelong care and therapy. This not only affects the quality of life for the affected children but also imposes a significant financial and emotional burden on families(37).

- **Increased Healthcare Utilization:** Infants who suffer from severe or recurrent hypoglycaemia may experience long-term complications that require ongoing healthcare intervention, such as rehabilitation therapies or neurological assessments, which further contribute to the healthcare burden(38).
- **Social and Economic Burden:** Beyond medical costs, neonatal hypoglycaemia's long-term consequences can affect a child's educational performance and social integration, increasing societal costs related to healthcare, special education, and social services(38).

Economic Burden

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- **Healthcare Costs:** The direct healthcare costs of managing neonatal hypoglycaemia include the expenses associated with blood glucose monitoring, intravenous glucose therapy, and the provision of specialized care. In LMICs, where resources are often limited, these costs may be compounded by the need for extensive care and referral to tertiary centres(40).
- **Cost of Delayed Diagnosis and Complications:** If hypoglycaemia is not detected early or managed adequately, the long-term complications that arise from neurological damage can result in increased costs over time. Treating complications

like cerebral palsy, cognitive impairment, and epilepsy can add to the financial burden, both for healthcare systems and families(41).

- **Resource Allocation:** In NICUs, the frequent monitoring and treatment of neonates with hypoglycaemia demand significant personnel and infrastructure resources. The need for specialized care units, medical staff, and medical supplies further strains healthcare systems, particularly in areas with limited resources(42).

Impact on Healthcare Systems

Neonatal hypoglycaemia places considerable pressure on healthcare systems, particularly in resource-limited settings. The challenge lies not only in the acute management of hypoglycaemia but also in providing the necessary long-term follow-up care to prevent complications. Inadequate screening, delayed diagnoses, and insufficient resources to manage severe cases may increase the burden on NICUs and overall healthcare facilities(43).

■ **Lack of Standardized Protocols:** ■
■
■
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■

- **Training and Education:** Effective management of neonatal hypoglycaemia requires continuous training for healthcare professionals, which can strain hospital budgets, particularly in resource-constrained settings. Ensuring that NICU staff are well-versed in screening, diagnosis, and treatment of hypoglycaemia is crucial to reduce its burden and improve outcomes(45).

[REDACTED]

Definition of Neonatal Hypoglycaemia

[REDACTED]

Causes of Neonatal Hypoglycaemia in NICU

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Physiological Glucose Regulation in Neonates

Neonatal glucose homeostasis is governed by a finely balanced mechanism involving glucose production, storage, and utilization. Immediately after birth, the cessation of maternal glucose supply via the placenta necessitates a prompt metabolic transition. In utero, the fetus is in a relatively constant state of glucose availability. Postnatally, the neonate must adapt by initiating endogenous glucose production to meet energy demands, particularly for the brain, which consumes a substantial portion of total

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Table 1: Physiological Glucose Regulation in Neonates and Risk Factors for Hypoglycemia

Parameter	Normal Term Neonates	Preterm Infants	Small for Gestational Age (SGA)	Infants of Diabetic Mothers (IDM)
Glycogen Stores	Adequate hepatic stores	Reduced stores	Depleted due to intrauterine malnutrition	Usually adequate but rapidly depleted
Enzymatic Maturity	Fully developed	Immature enzymes for gluconeogenesis	Variable	Functional but suppressed by insulin
Ketogenesis	Prompt activation post-birth	Delayed	May be impaired	Suppressed due to hyperinsulinemia
Insulin Levels	Decline after birth	Normal or low	Normal	Elevated due to maternal hyperglycemia
Risk of Hypoglycemia	Low	High	High	High
Adaptation to Postnatal Life	Rapid and efficient	Delayed adaptation	Poor adaptation	Poor adaptation due to excess insulin

Importance of Glucose for Brain Development

Clinical Manifestations of Neonatal Hypoglycemia

Neonatal hypoglycemia is a common metabolic condition in newborns that often presents with subtle or nonspecific signs, making early diagnosis challenging. These manifestations can vary from mild and transient symptoms to more severe, life-threatening conditions, depending on the severity and duration of the hypoglycemia. Some neonates, especially those who are premature, low birth weight, or critically ill, may remain asymptomatic, which highlights the importance of routine glucose monitoring, particularly in high-risk infants. Below are the key clinical manifestations associated with neonatal hypoglycemia (55).

1. Jitteriness and Tremors

Jitteriness, or involuntary shaking of the neonate's body, is one of the first signs of neonatal hypoglycemia. This trembling can occur when glucose levels drop below the threshold needed for proper brain and muscle function. Jitteriness is often the most noticeable in neonates who are preterm or have low birth weight. It can be confused with normal reflexive movements, but its persistence or worsening in the absence of any external stimulus is a red flag for low glucose levels. Prompt intervention to correct glucose levels can help alleviate these tremors and prevent further complications (56)

2. Poor Feeding and Weak Suckling

Poor feeding, which may manifest as weak sucking, refusal to feed, or a failure to latch on, is a significant clinical manifestation of hypoglycemia in newborns. Neonates with low blood glucose often display lethargy and lack the energy required for effective feeding. Feeding difficulties can become a vicious cycle, as inadequate caloric intake contributes to further declines in blood glucose, worsening the infant's condition. Additionally, neonates who are preterm or

critically ill may have feeding difficulties due to other underlying health issues, such as gastrointestinal immaturity or respiratory distress, which makes the identification of hypoglycemia even more important.

3. Lethargy and Unresponsiveness

Lethargy, characterized by excessive sleepiness, reduced activity, and poor responsiveness to stimuli, is one of the more common and alarming signs of neonatal hypoglycemia. A lethargic infant may be hard to wake for feeding or may appear listless and weak when held. This condition is a result of insufficient glucose supply to the brain, leading to a lack of energy and decreased alertness. In more severe cases, lethargy may evolve into deep stupor or unresponsiveness, increasing the risk of brain injury if not promptly addressed.

4. Apnea (Temporary Cessation of Breathing)

Apnea, or the temporary cessation of breathing for more than 20 seconds, is another potentially severe manifestation of neonatal hypoglycemia. This occurs due to a disruption in the regulation of respiratory drive by the brain, which relies on a stable glucose supply. In neonates, particularly those born prematurely, the lack of glucose can impair the brain's ability to regulate autonomic functions, leading to intermittent cessation of breathing. This condition requires immediate intervention to restore normal glucose levels and maintain oxygenation to the infant's vital organs.(57)

5. Seizures

Seizures are a serious complication of severe or prolonged neonatal hypoglycemia. Neonates may display abnormal body movements, such as jerking, twitching, or repetitive rhythmic movements. Seizures in hypoglycemic infants are indicative of significant brain dysfunction due to glucose deprivation. These seizures can have long-term consequences if not managed quickly, potentially leading to permanent

neurological damage. Seizures require urgent medical treatment, including glucose administration, and thorough evaluation to rule out other causes of seizure activity, such as infections or intracranial hemorrhage.(58)

6. Hypotonia (Decreased Muscle Tone)

Hypotonia, or a marked decrease in muscle tone, is another clinical sign seen in hypoglycemic neonates. Infants with hypotonia may appear “floppy” and lack the muscle strength needed to hold up their head or limbs. The decreased muscle tone results from the impaired function of the brain and neuromuscular system due to inadequate glucose supply. Hypotonia can interfere with the infant’s ability to engage in normal reflex activities, such as suckling, and may also affect their ability to perform voluntary movements.(59)

7. High-Pitched Cry

A high-pitched, shrill cry is sometimes heard in neonates with hypoglycemia. This type of cry is often described as being more intense and piercing than a typical infant cry. It is believed to be a result of neurological distress caused by low blood glucose levels. The high-pitched cry can indicate discomfort or a response to metabolic dysfunction, signaling the need for immediate evaluation and intervention to correct the hypoglycemia. If left untreated, this condition may evolve into more severe symptoms, including seizures or coma.(60)

8. Cyanosis and Hypothermia

In more severe or prolonged cases of neonatal hypoglycemia, the infant may show signs of cyanosis (a bluish tint to the skin) and hypothermia (low body temperature). Cyanosis occurs when there is inadequate oxygen delivery to tissues due to impaired circulatory function, which is common in critically ill infants. Hypothermia, especially in preterm or ill infants, results from the body’s inability

to generate sufficient heat due to glucose and energy deficiencies. Both cyanosis and hypothermia are indicators of severe metabolic compromise and require urgent medical attention to prevent further complications.

The clinical manifestations of neonatal hypoglycemia can range from mild and transient symptoms like jitteriness to severe and life-threatening conditions such as seizures and apnea. Since many of these symptoms overlap with other neonatal conditions, early detection through routine glucose monitoring is critical in at-risk neonates. Identifying hypoglycemia in its early stages allows for prompt intervention, which is essential in preventing long-term neurological damage and ensuring favorable outcomes(43).

Clinical Significance of Early Detection

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Importance of Early Detection

Neurological Protection: [REDACTED]

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Management of At-Risk Neonates:

- **Reduction of Neurodevelopmental Deficits:**

important in preventing more severe consequences like cerebral palsy or epilepsy(63).

Challenges of Detection

- **Asymptomatic Cases:** Many neonates, especially those who are asymptomatic, may not exhibit signs of hypoglycemia until it becomes severe. This makes early detection through routine screening of at-risk infants essential, as waiting for clinical signs to appear may lead to missed opportunities for intervention.
- **Nonspecific Symptoms:** When symptoms do occur, they are often nonspecific, including signs like jitteriness, lethargy, poor feeding, and apnea. These symptoms can overlap with other neonatal conditions such as sepsis or respiratory distress, making it difficult to diagnose hypoglycemia solely based on clinical presentation.
- **Diagnostic Tools:** The use of bedside glucose monitoring and other diagnostic tools for early screening has been shown to significantly improve the detection of

neonatal hypoglycemia. The application of screening protocols, particularly in high-risk groups, is crucial for identifying cases before symptoms become evident.

Benefits of Early Intervention

■ **Immediate Intervention:** Once hypoglycemia is detected, immediate interventions, such as oral feeding, IV glucose therapy, or addressing underlying causes (e.g., insulin overproduction in IDM infants), can be implemented to restore normal glucose levels and [REDACTED]

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■ **Management Guidelines:** Early detection and prompt intervention are emphasized in clinical guidelines, such as those set by the American Academy of Pediatrics (AAP) and the World Health Organization (WHO), which advocate for the

[illegible]

1. Early-Onset Hypoglycemia in High-Risk Infants

In neonates at high risk for hypoglycemia, such as those born prematurely, small for gestational age (SGA), or infants of diabetic mothers (IDM), hypoglycemia often manifests in the first few hours of life. These infants are at greater risk due to their underdeveloped metabolic systems, limited glycogen stores, and in some cases, hyperinsulinemia from maternal diabetes. Preterm infants, for example, have immature liver function and less glycogen available for conversion into glucose, which results in difficulty maintaining normal blood glucose levels. In addition, infants born to diabetic

mothers may experience persistent hypoglycemia because of elevated insulin levels that continue to affect glucose regulation after birth.

This early-onset hypoglycemia is often exacerbated by the stress of birth and delays in feeding. These infants require close monitoring to prevent prolonged hypoglycemia and associated complications. Timely feeding or intravenous (IV) glucose supplementation is essential to manage early hypoglycemia(66).

2. Physiological Hypoglycemia in Healthy Term Infants

[REDACTED]

3. Persistent Hypoglycemia Beyond the Early Hours

For neonates with risk factors such as being born preterm, SGA, IDM, or those who have experienced birth asphyxia or other perinatal stress, hypoglycemia may persist beyond the first few hours of life. Infants of diabetic mothers, for instance, may experience hypoglycemia for several hours or even days due to persistent hyperinsulinemia, which can impair glucose metabolism even after birth. These

neonates often require IV glucose administration or frequent feedings to maintain glucose homeostasis.

When hypoglycemia continues beyond 24 hours of life, further investigation is needed to rule out other causes, such as endocrine disorders (e.g., congenital hyperinsulinism, adrenal insufficiency), or metabolic conditions that affect glucose regulation. If the condition persists for 48 to 72 hours or longer, it raises concerns about a more serious underlying problem that needs prompt intervention(68).

4. Late-Onset Hypoglycemia

[REDACTED]

5. Investigating Persistent or Severe Hypoglycemia

For neonates who exhibit persistent or severe hypoglycemia beyond 48 to 72 hours, further investigation is essential to determine whether an underlying metabolic or endocrine disorder is present. Conditions such as congenital hyperinsulinism, adrenal

insufficiency, and inborn errors of metabolism may cause prolonged or recurrent hypoglycemia. A comprehensive diagnostic workup, including biochemical tests, genetic screenings, and endocrine evaluations, may be necessary to identify and treat the underlying cause.

Additionally, metabolic conditions such as glycogen storage diseases or disorders affecting fatty acid oxidation can present with hypoglycemia that doesn't resolve with initial glucose supplementation. These conditions require specialized care and treatment, often involving long-term management strategies.(70)

The age of presentation of neonatal hypoglycemia is influenced by a variety of factors, including the infant's gestational age, birth weight, and any perinatal stresses experienced. In high-risk infants, hypoglycemia often presents early, within the first few hours of life, and requires immediate intervention. While physiological hypoglycemia in term infants usually resolves on its own within the first few hours after birth, prolonged or persistent hypoglycemia is indicative of a more serious underlying issue. Neonates who experience hypoglycemia beyond 48 hours require thorough investigation to rule out metabolic or endocrine disorders. Early recognition and intervention are essential to prevent serious neurological damage and ensure the best possible outcomes for neonates with hypoglycemia(34).

Short-Term Outcome of Neonatal Hypoglycemia

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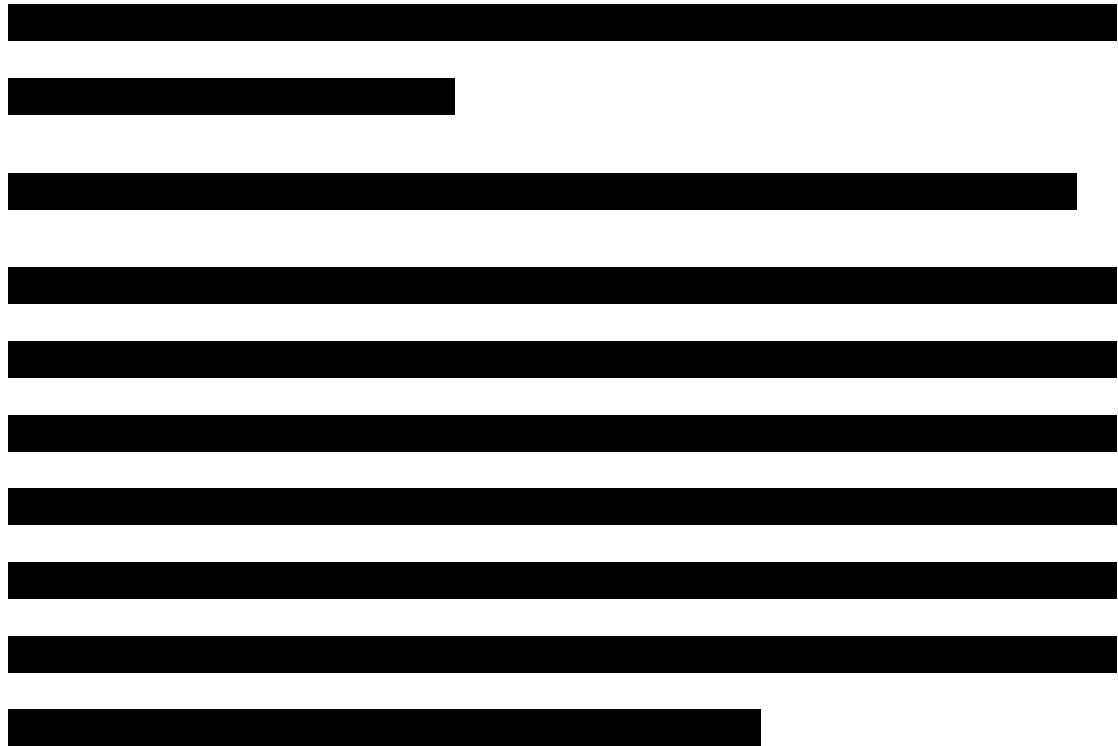
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1. The American Academy of Pediatrics (AAP) - 2011 Report

The AAP guidelines emphasize the necessity of early detection of symptomatic and at-risk asymptomatic infants. These guidelines are particularly useful for establishing screening protocols to prevent prolonged hypoglycemia and its subsequent adverse outcomes. Here are the critical takeaways:

- **For Symptomatic Infants:**
 - If the glucose level drops below 40 mg/dL, intravenous (IV) glucose treatment is necessary.
- **For At-Risk Asymptomatic Infants:** Infants at higher risk include those who are:
 - Small-for-Gestational-Age (SGA)
 - Infants of Diabetic Mothers (IDM)
 - Large-for-Gestational-Age (LGA)

- Late Preterm (34-36 6/7 weeks gestation)

Screening Protocol for At-Risk Newborns:

- **First 4 Hours of Life:**
 - Initial glucose < 25 mg/dL: Provide feeding and then recheck glucose.
 - If repeat glucose remains < 25 mg/dL, administer IV glucose.
 - If glucose is between 25-40 mg/dL, feeding is sufficient and IV glucose may not be needed.
- **4 to 24 Hours of Life:**
 - Glucose < 35 mg/dL: Feed the infant and recheck glucose in 1 hour.
 - If the glucose is still < 35 mg/dL, start IV glucose.
 - If glucose levels are between 35-45 mg/dL, repeat feeding.
 - Target glucose should be >45 mg/dL to minimize risks.
- **After 48 Hours of Life:**
 - Plasma glucose should ideally exceed 60 mg/dL. This threshold ensures that transient hypoglycemia is avoided.

2. Pediatric Endocrine Society - 2015 Guidelines

The Pediatric Endocrine Society guidelines (2015) provide a more nuanced approach based on the infant's age and the presence of symptoms. These guidelines help ensure more precise glucose level thresholds based on clinical presentation(2).

- **For Symptomatic Infants:**

- If glucose < 40 mg/dL, treatment with IV glucose is immediate.
- **For Asymptomatic Infants at Risk:**
 - At-risk neonates include those who are SGA, IDM, LGA, or late preterm (34-36 weeks).

Screening Protocols for At-Risk Asymptomatic Infants:

- **First 4 Hours of Life:**
 - Initial Glucose: If < 25 mg/dL, feed the infant and then recheck glucose.
 - If repeat glucose is < 25 mg/dL, administer IV glucose.
 - If repeat glucose falls between 25-40 mg/dL, feeding may be sufficient.
- **4 to 24 Hours of Life:**
 - If glucose < 35 mg/dL, feed the infant and recheck glucose after 1 hour.
 - If glucose remains < 35 mg/dL, initiate IV glucose.
 - If glucose levels are between 35-45 mg/dL, repeat feeding and monitor.
 - The goal is to maintain glucose levels > 45 mg/dL to ensure adequate energy supply for the infant.
- **After 48 Hours of Life:**

- Plasma glucose should exceed 60 mg/dL to avoid persistent hypoglycemia, and to support the continued growth and metabolic stability of the neonate.

Key Considerations in Neonatal Hypoglycemia Management

1. Frequency of Monitoring:

- Neonates with known risk factors, including maternal diabetes, SGA, and preterm birth, require careful monitoring, particularly during the first 48-72 hours after birth, when glucose regulation mechanisms mature.

2. Feeding and IV Glucose Therapy:

- Feeding is the first-line treatment, especially for asymptomatic infants. If feeding fails to correct hypoglycemia, IV glucose should be provided to stabilize blood sugar levels.

3. Avoidance of Prolonged Hypoglycemia:

- Prolonged hypoglycemia is linked to neurodevelopmental impairments, so it is crucial to intervene early to prevent cerebral damage.

4. Accuracy of Point-of-Care Glucose Measurement:

- Point-of-care glucose testing using whole blood may not provide as accurate readings in the hypoglycemic range as plasma glucose measurements. Plasma glucose is considered more accurate for clinical decision-making.

The AAP (2011) and PES (2015) guidelines offer comprehensive frameworks for managing neonatal hypoglycemia, emphasizing early detection, prompt treatment, and

prevention of neurological damage. These protocols guide clinicians in addressing hypoglycemia, with individualized approaches based on infant age, clinical presentation, and underlying risk factors. Adhering to these protocols reduces the risk of long-term neurodevelopmental issues and helps ensure the well-being of high-risk neonates in the NICU(23).

Clinical Guidelines for Neonatal Hypoglycemia Management	
1	2
American Academy of Pediatrics (AAP) - 2011 • Symptomatic Infants: IV glucose if level <40 mg/dL • At-Risk Groups: SGA, IDM, LGA, Late Preterm (34-36 6/7 weeks) • First 4 Hours: Treat if <25 mg/dL • 4-24 Hours: Treat if <35 mg/dL, target >45 mg/dL • After 48 Hours: Target >60 mg/dL	Pediatric Endocrine Society (PES) - 2015 • Similar thresholds to AAP guidelines • More nuanced approach based on age and symptoms • First 4 Hours: Treat if <25 mg/dL • 4-24 Hours: Treat if <35 mg/dL, target >45 mg/dL • After 48 Hours: Target >60 mg/dL
Key Management Considerations	
• Frequent monitoring for at-risk infants (first 48-72 hours) • Feeding as first-line treatment for asymptomatic cases	• Early intervention to prevent cerebral damage • Plasma glucose measurements more accurate than point-of-care testing

Fig 1: Clinical Guidelines for Neonatal Hypoglycemia Management

Neurodevelopmental Sequelae of Prolonged Hypoglycemia

Prolonged neonatal hypoglycemia can result in severe and often irreversible neurodevelopmental sequelae, which are particularly concerning for neonates in high-risk categories such as preterm infants, those with intrauterine growth restriction (IUGR), or infants of diabetic mothers (IDM). These neonates are more vulnerable because their brains are still in critical stages of development, and an insufficient supply

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can be adversely affected, resulting in long-term motor deficits, including spasticity and

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Furthermore, longitudinal studies have shown that the recovery process for infants who experience hypoglycemia is largely dependent on the timeliness of intervention. Immediate interventions, including glucose administration, can lead to favorable outcomes with no long-term neurological effects. However, if hypoglycemia is left uncorrected for an extended period, the damage to neuronal structures becomes irreversible, leading to persistent developmental delays that affect motor, cognitive, and social functions.

The neurodevelopmental sequelae of prolonged neonatal hypoglycemia are substantial and can affect an infant’s entire life course. The risk of irreversible cognitive and motor impairments underscores the critical importance of early detection and treatment of hypoglycemia in neonates, particularly in those at high risk. With timely and effective intervention, the potential for long-term neurological damage can be minimized, highlighting the necessity of vigilant monitoring and proactive management in the neonatal period(17).

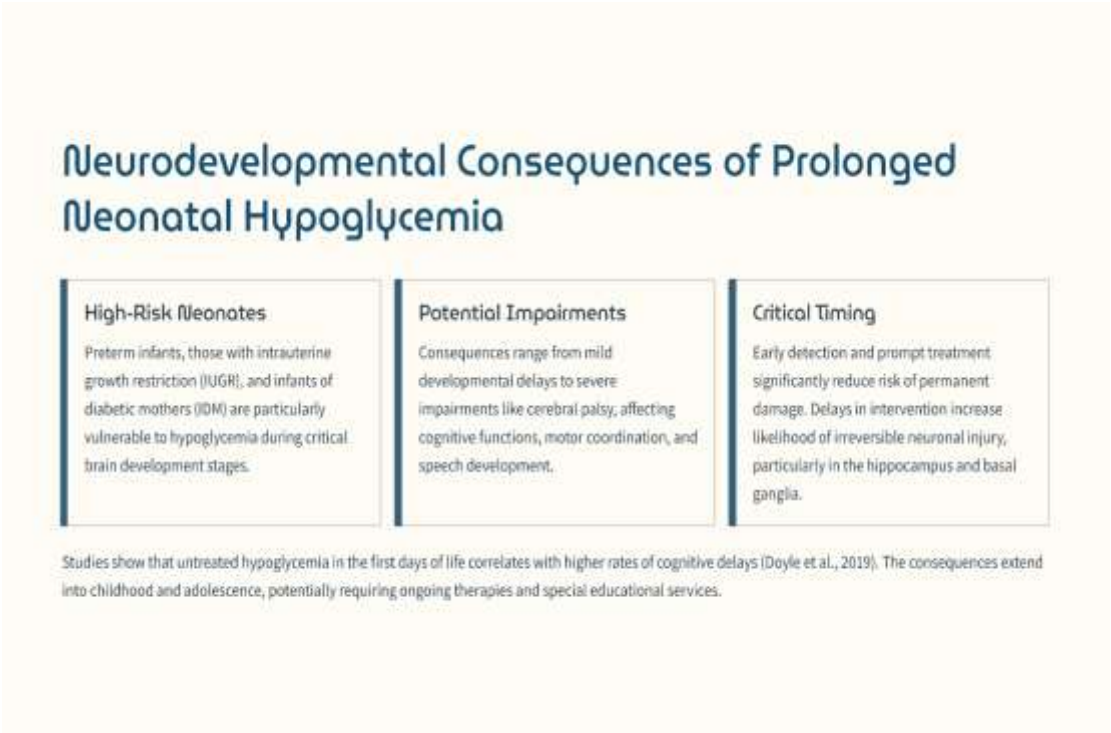


Fig 2: Summary of Neurodevelopmental Sequelae of Prolonged Hypoglycemia

Research Studies

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Chanie ES et al. (2023) [REDACTED]
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Gaps in Knowledge / Need for Study

Despite neonatal hypoglycemia being a prevalent condition in NICUs, there remain several gaps in knowledge that need to be addressed through focused research. While significant strides have been made in identifying the immediate risks associated with hypoglycemia, much is still unknown, particularly with regard to screening protocols, optimal glucose thresholds for intervention, and the long-term effects of transient hypoglycemia in neonates(67).

Lack of Consensus on Screening Protocols

[REDACTED]

Optimal Glucose Thresholds for Intervention

Another major gap in neonatal hypoglycemia research is the lack of a clear, universally accepted glucose threshold for initiating intervention. While operational thresholds such as a plasma glucose level below 45 mg/dL have been commonly used, there is ongoing debate regarding the appropriate level at which treatment should be initiated.

Different studies have proposed various thresholds, ranging from 40 mg/dL to 50 mg/dL, but no single value has been proven to be universally optimal for preventing both immediate harm and long-term neurodevelopmental sequelae. This uncertainty calls for research into the clinical significance of different glucose thresholds, particularly in relation to short-term outcomes in high-risk neonates(69).

Long-Term Impact of Transient Hypoglycemia

[REDACTED]

Limited Research on Indian Populations

In India, while neonatal hypoglycemia is a well-recognized issue, there is a lack of robust, localized research focusing on short-term outcomes and the unique risk factors prevalent in the Indian population. Much of the existing literature on hypoglycemia is from Western countries, where prevalence, screening practices, and clinical presentations may differ significantly from those in India. In India, there are high rates of intrauterine growth restriction (IUGR), low birth weight infants, and suboptimal early feeding practices, which may increase the risk of hypoglycemia. However, Indian

studies specifically examining the short-term clinical outcomes of neonatal hypoglycemia in the NICU setting—such as feeding issues, apnea, hypothermia, and extended NICU admissions—remain scarce. This lack of data hinders the development of localized, evidence-based protocols for the management of hypoglycemia in Indian NICUs(71).

Variability in Incidence, Clinical Presentations, and Outcomes Across Risk Groups

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Rationale

Neonatal hypoglycemia is a treatable condition if recognized early. Early detection and intervention can prevent significant long-term neurological and developmental complications. Hypoglycemia in newborns, particularly in high-risk infants such as those born prematurely, with intrauterine growth restriction, or to diabetic mothers, remains a common metabolic disturbance in neonatal intensive care units (NICUs). However, the clinical presentation of hypoglycemia can be subtle, and many neonates remain asymptomatic, which underscores the importance of proactive screening and timely treatment(77).

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MATERIAL AND METHODS

Study design: It was a Prospective Observational study

Place of study: Tertiary care hospital

Duration of study: 18 months Sample size:

Sample Size:

$$\text{Sample Size} = 1 + \frac{Z^2 \times p (1-P)}{e^2 N}$$

N = Population

size e = Margin of error

z = z score

p = standard deviation

Calculated sample size is 241

With prevalence 19.4 margin of error 5% and 95 %

Confidence interval.

	bsl	Treatment
Symptomatic	bsl <40 mg/dl	IV Dextrose
Asymptomatic Neonates (0–4 hrs of life)	bsl <25 mg/dl	Feeding was given and rechecked BSL after 1 hr; if BSL does not rise, IV Dextrose infusion was given
Asymptomatic Neonates (4–24 hrs of life)	bsl <35 mg/dl	Feeding was given and rechecked BSL after 1 hr; if BSL does not rise, IV Dextrose infusion was given
Asymptomatic Neonates (>24 hrs of life)	bsl <50 mg/dl	Feeding was given and rechecked BSL after 1 hr; if BSL does not rise, IV Dextrose infusion was given

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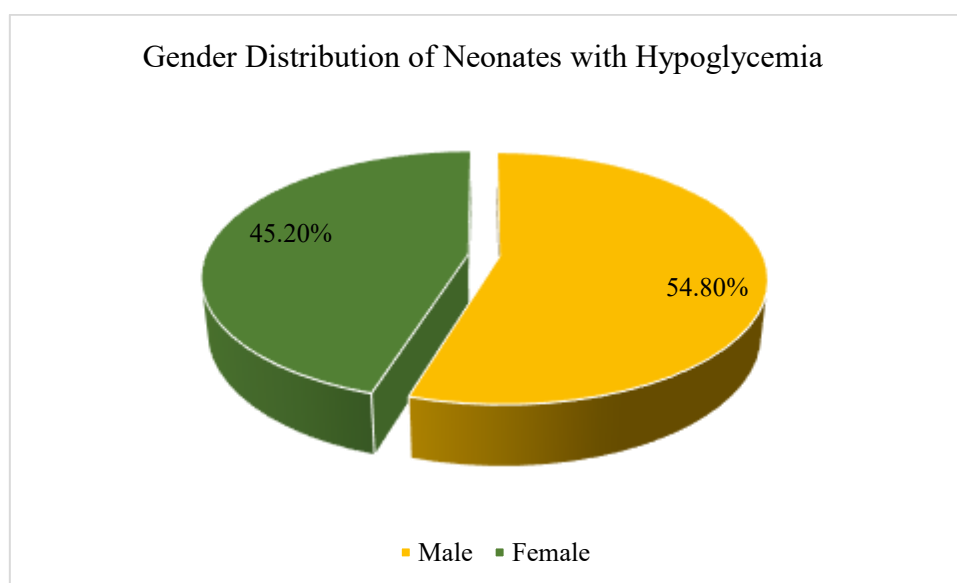
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RESULTS AND ANALYSIS

Table 2: Gender Distribution of Neonates with Hypoglycemia

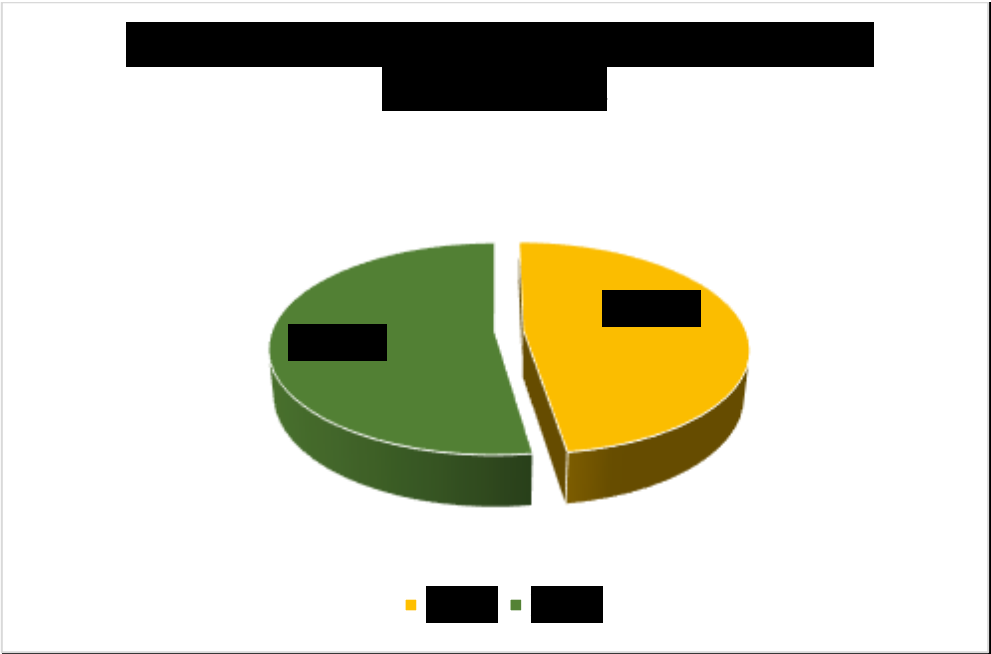
		n	%
Gender	Male	132	54.8%
	Female	109	45.2%
	Total	241	100.0%

Graph 1: Gender Distribution of Neonates with Hypoglycaemia



Among the 241 neonates with hypoglycemia, 132 (54.8%) were male and 109 (45.2%) were female, indicating a slight male predominance in the study population.

Table 3: Parity Distribution of Mothers of Neonates with Hypoglycaemia



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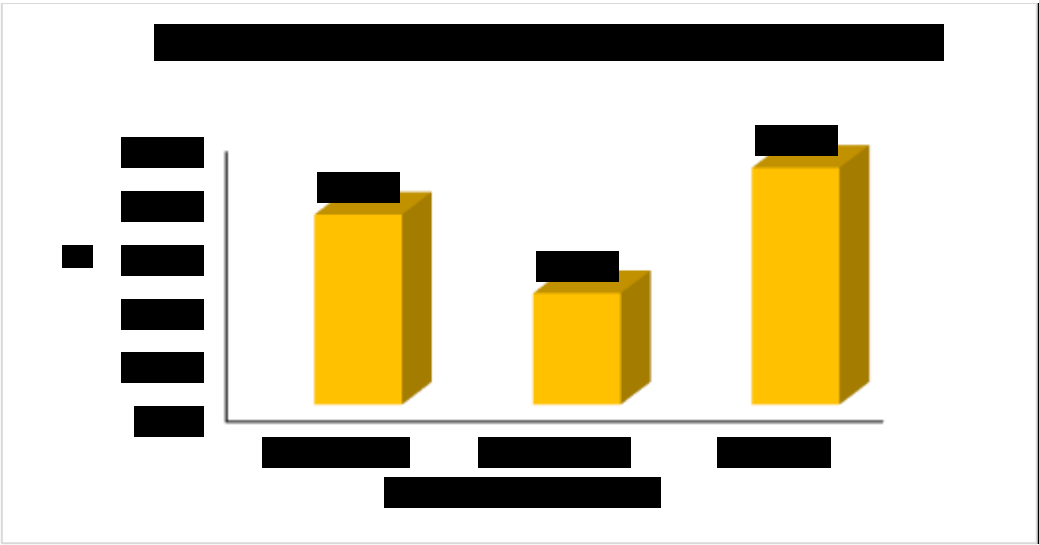
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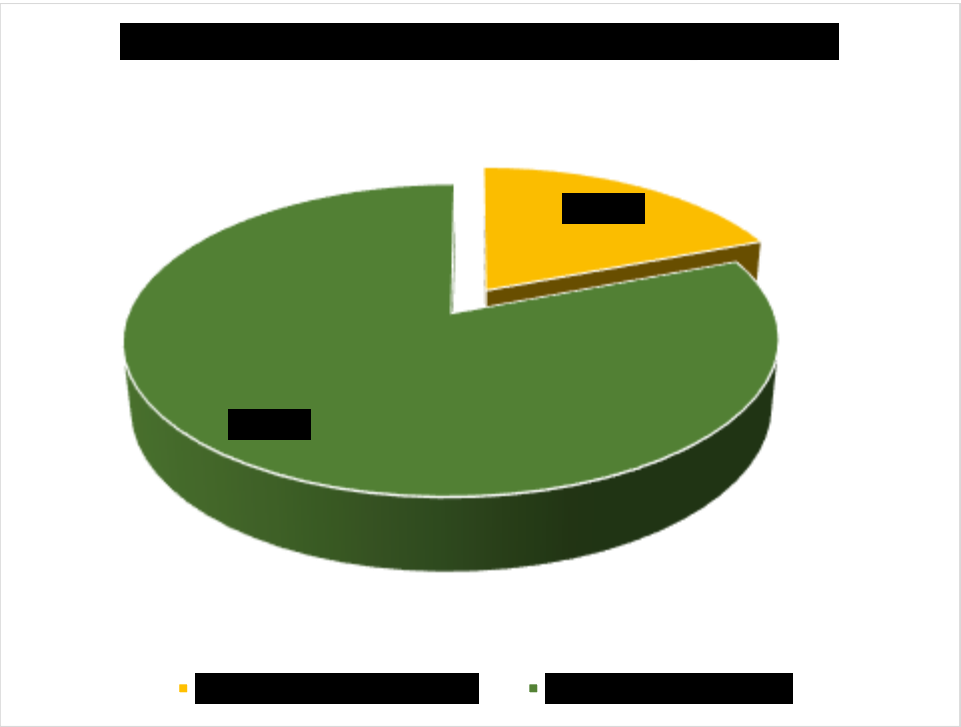
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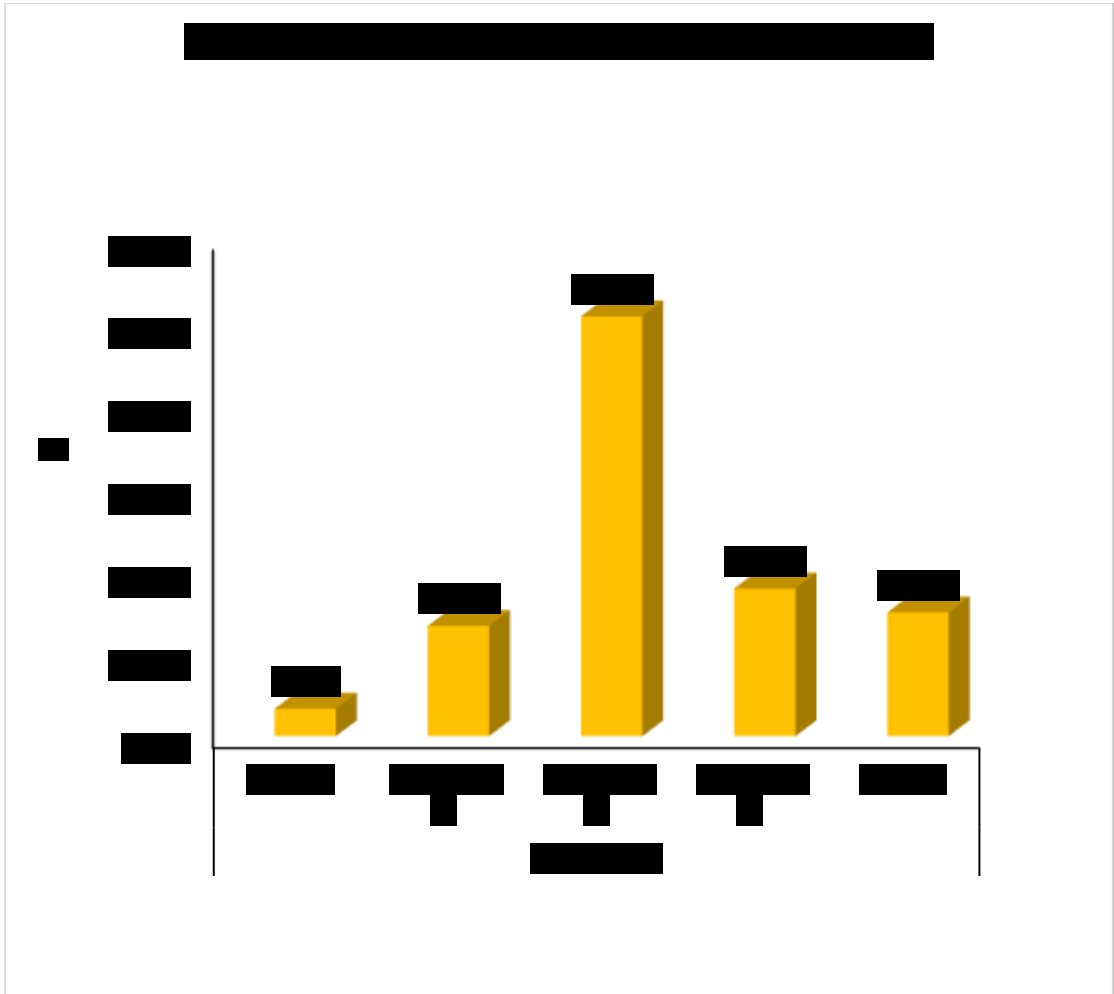
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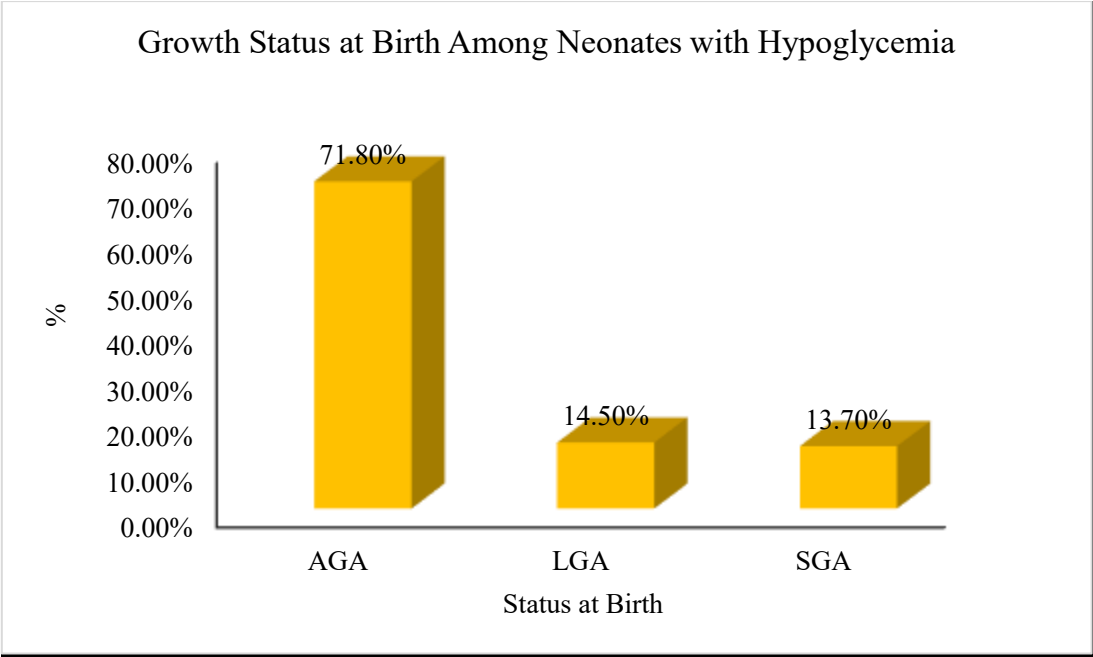
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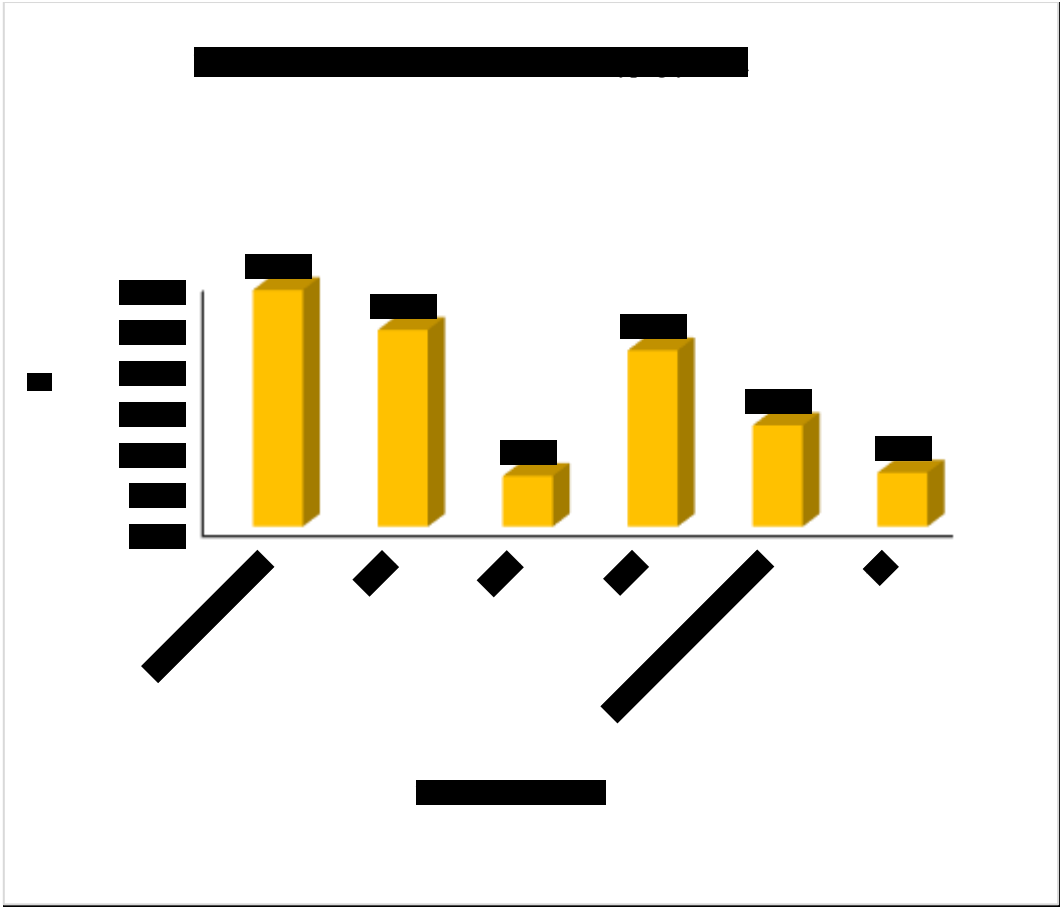
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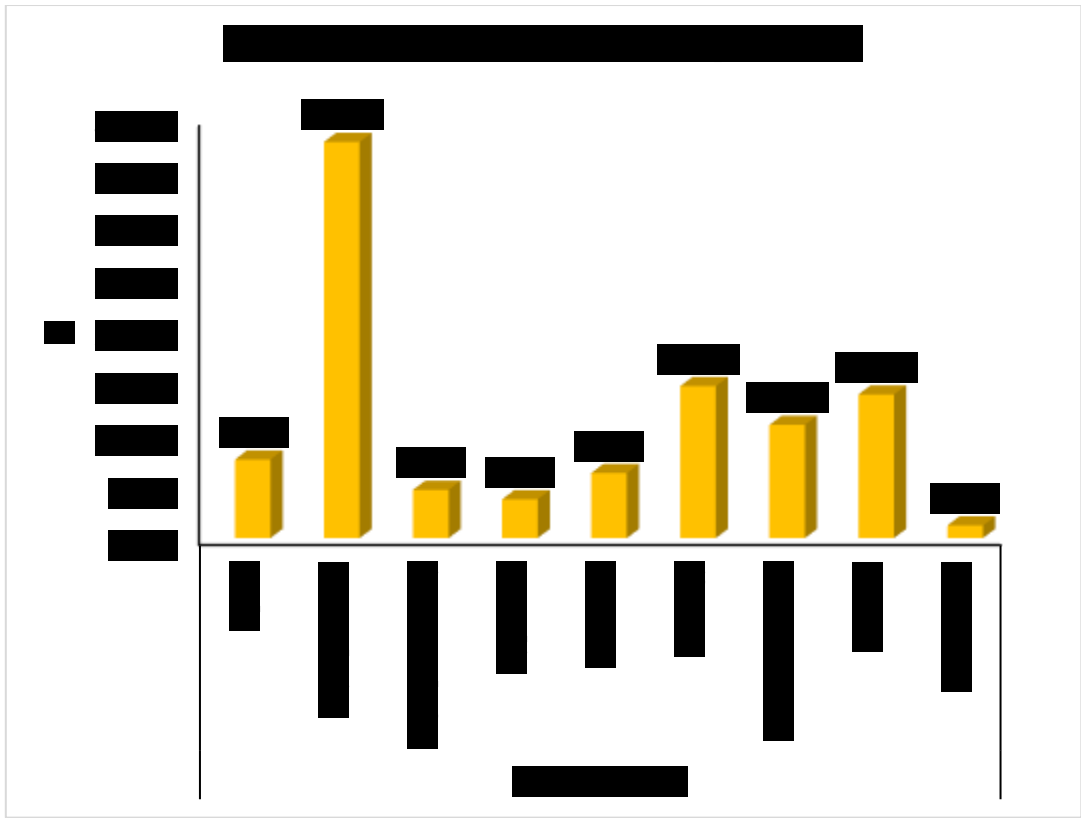
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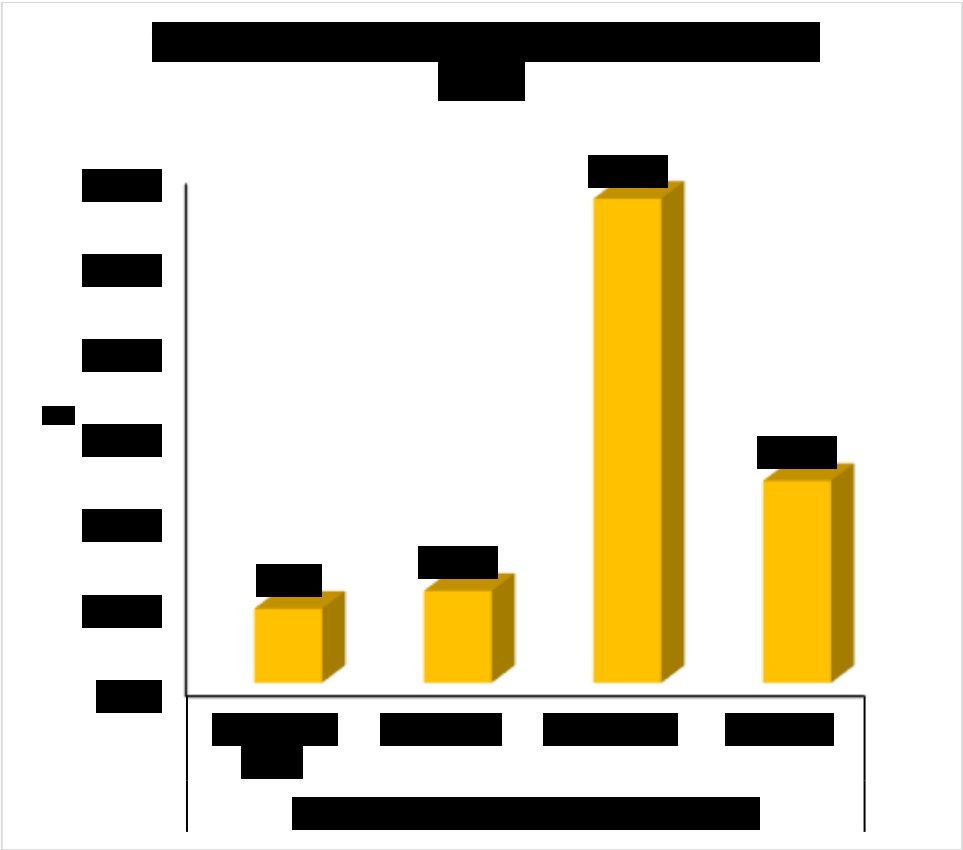
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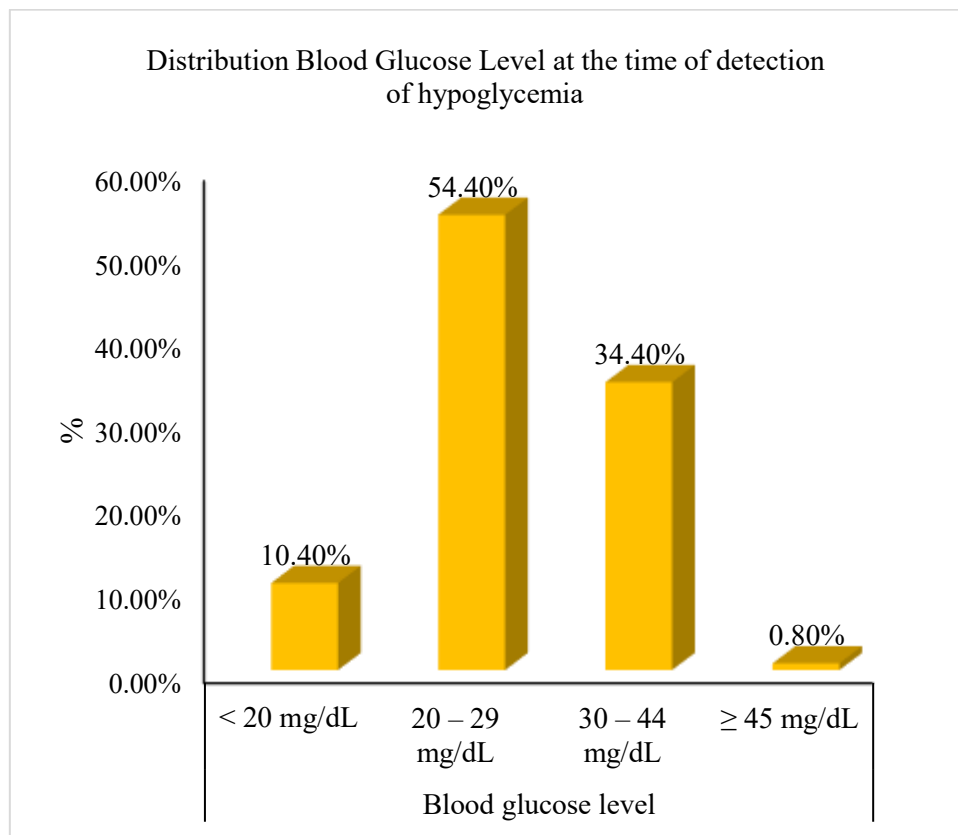
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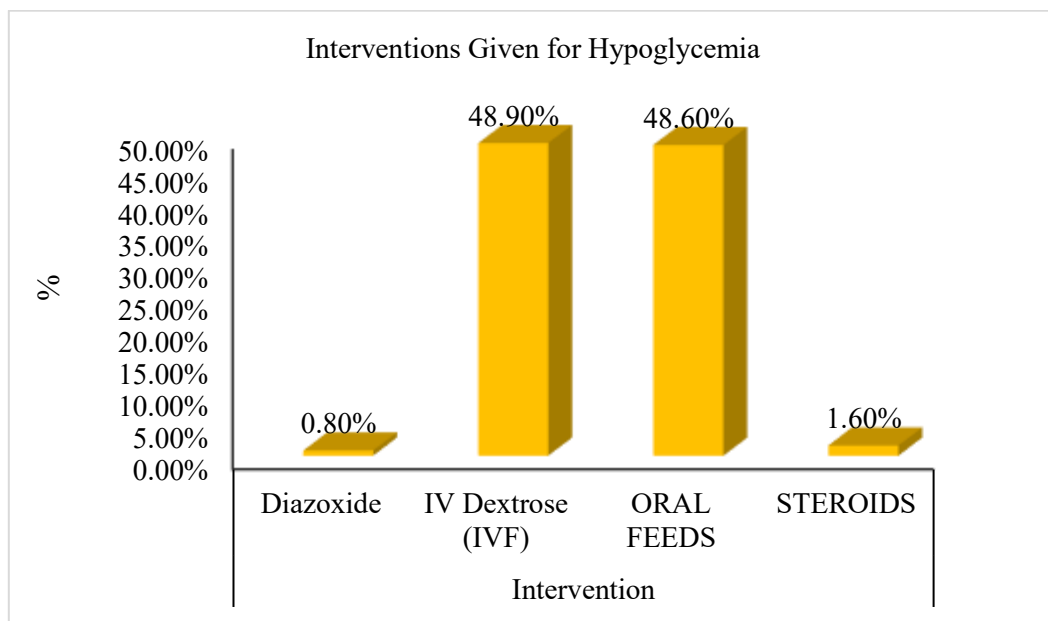
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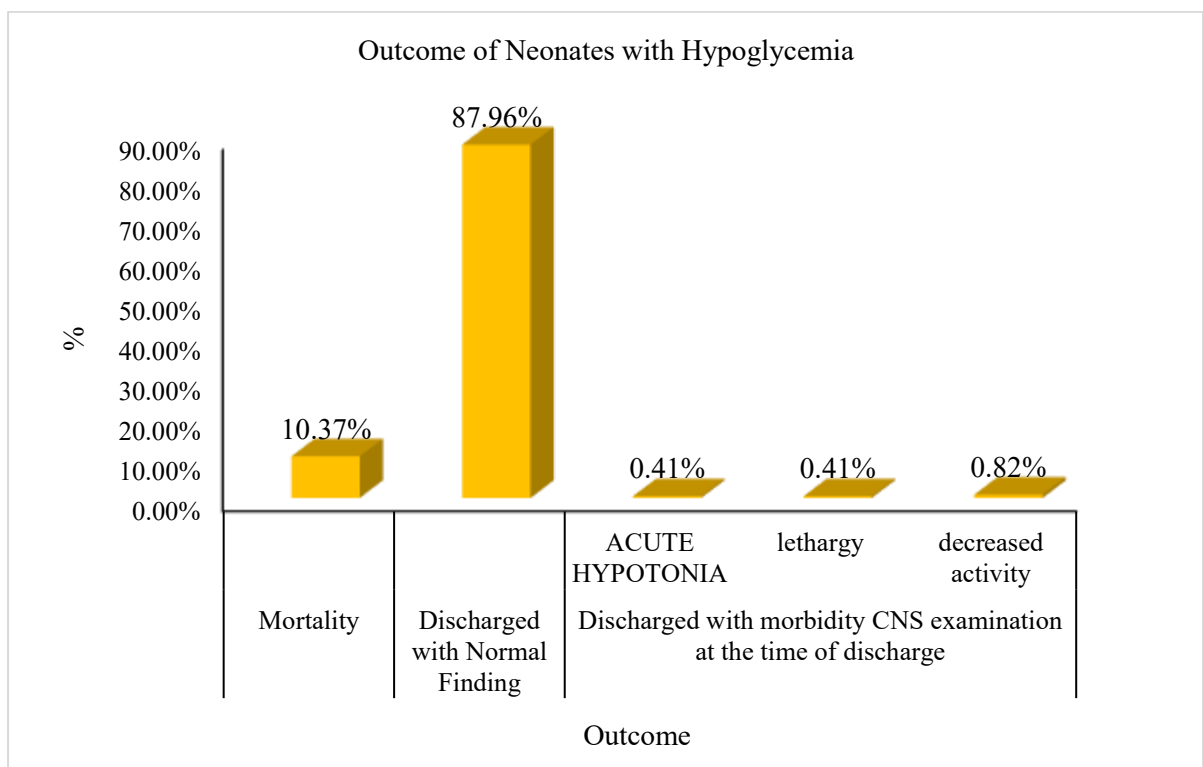
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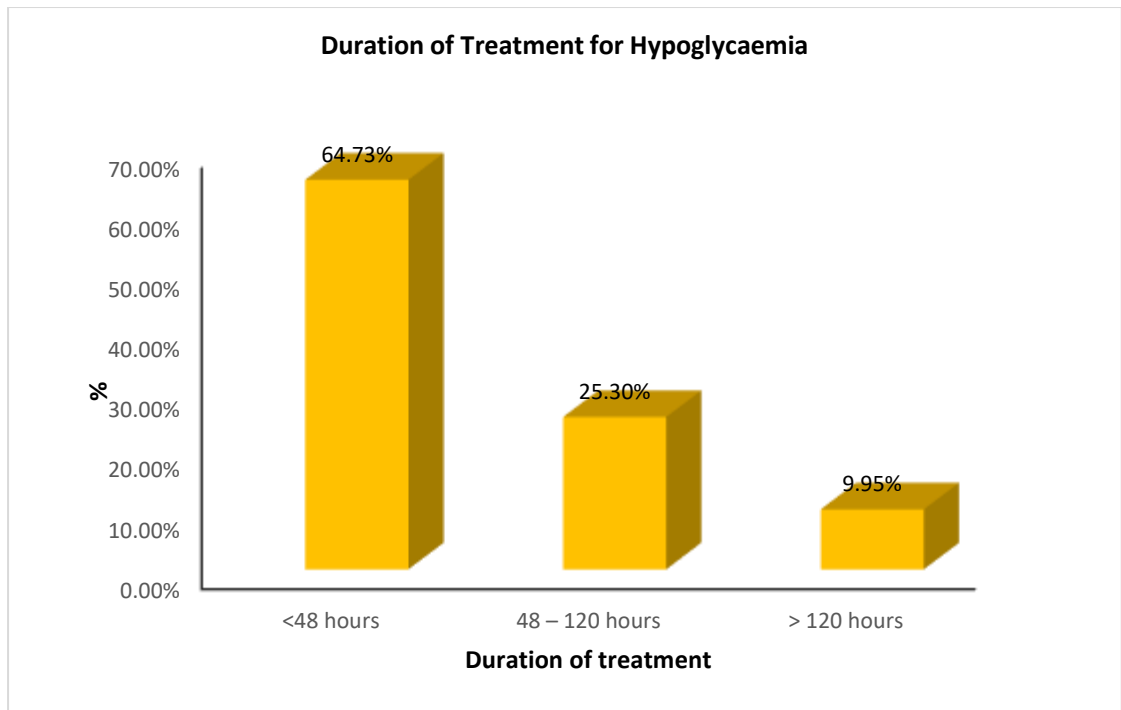
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DISCUSSION

In the current study, out of 241 neonates diagnosed with hypoglycemia, 132 (54.8%) were male and 109 (45.2%) were female, indicating a slight male predominance. This pattern aligns with observations by Harris et al. (2012), who reported that male neonates were more frequently affected by neonatal hypoglycemia, likely due to delayed metabolic adaptation to extrauterine life and higher metabolic demands in male infants (30). Similarly, Harding et al. (2024) suggested that sex-based physiological immaturity, especially in glucose homeostasis and hormonal responses, may explain this male vulnerability (1). Giouleka et al. (2023) also noted that while not an independent risk factor, male sex often coexists with other conditions such as preterm birth and SGA that heighten the risk of hypoglycemia (5).

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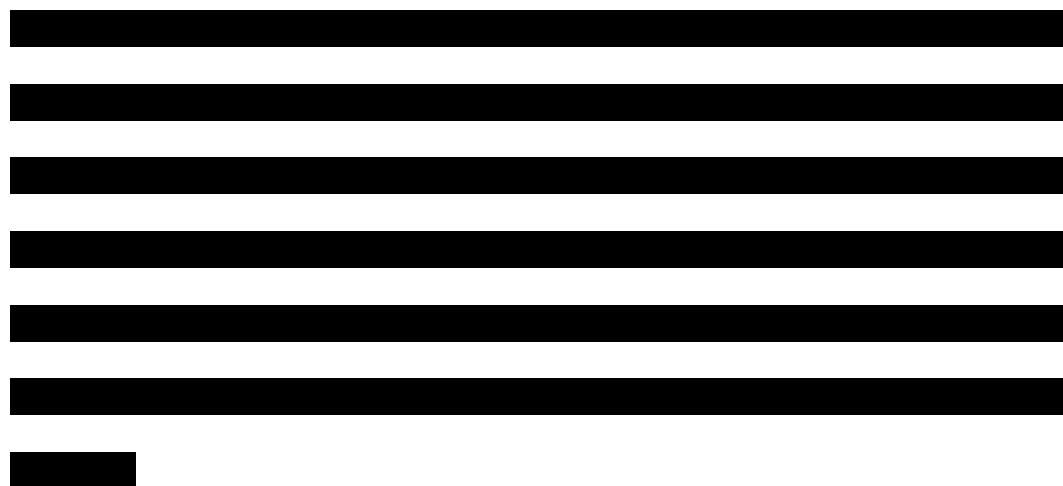
Limitations of the Study

1. **Single-Center Design:** The study was conducted in a single tertiary care hospital, which may limit the generalizability of findings to other healthcare settings or regions with differing practices and neonatal profiles.
2. **Short-Term Outcome Focus:** The study did not assess long-term neurodevelopmental outcomes, which are crucial for evaluating the true impact of neonatal hypoglycemia on cognitive and motor development.

3. **No Continuous Glucose Monitoring:** Intermittent blood glucose measurements were used, which might miss transient hypoglycemic episodes or underestimate glycemic variability.
4. **Lack of Detailed Neuroimaging or EEG Correlation:** Although some neonates presented with seizures or neurological signs, advanced imaging or electroencephalography (EEG) was not uniformly performed to document central nervous system involvement.
5. **Potential Confounding Factors Not Controlled:** Variables such as feeding practices, maternal BMI, socioeconomic status, and antenatal corticosteroid use were not adjusted for, which could have influenced neonatal glucose regulation.

CONCLUSION

This study provides a comprehensive overview of the clinical characteristics, risk factors, and short-term outcomes associated with neonatal hypoglycemia in a tertiary care NICU setting. The analysis revealed that conditions such as prematurity, low or high birth weight, small or large for gestational age status, and maternal disorders like gestational diabetes and pregnancy-induced hypertension significantly contributed to the occurrence of hypoglycemia.



These findings emphasize the critical importance of early screening, individualized monitoring, and timely treatment in at-risk neonates to ensure favorable early outcomes and reduce the risk of adverse neurological consequences.

SUMMARY

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[REDACTED] Most cases were managed effectively with intravenous dextrose or oral feeds, and the majority required a glucose delivery rate of 6–8 mg/kg/min. While most neonates recovered within 48 hours, a few required prolonged treatment or escalation of therapy.

[REDACTED]

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ANNEXURE I

LIST OF ABBREVIATIONS

Abbreviation	Full Form
NICU	Neonatal Intensive Care Unit
mg/dL	Milligrams per deciliter
IUGR	Intrauterine Growth Restriction
SGA	Small for Gestational Age
LGA	Large for Gestational Age
IDM	Infant of Diabetic Mother
GIR	Glucose Infusion Rate
hrs	Hours
IV	Intravenous
SPSS	Statistical Package for the Social Sciences
IBM	International Business Machines
D10	10% Dextrose
Z	Z-score (standard normal distribution value)
e	Margin of Error
p	Prevalence or Proportion
N	Population Size
SD	Standard Deviation
%	Percent
FSL	Forensic Science Laboratory
PM	Postmortem
BT	Brought Dead
GDM	Gestational Diabetes Mellitus
PIH	Pregnancy-Induced Hypertension
PROM	Premature Rupture of Membranes
LSCS	Lower Segment Cesarean Section

ANNEXURE II

CASE RECORD FORM

S.no.

Patients Name.

Age.

Sex.

Date/time of birth. / / , . ,

Birth weight. Kg (s)

Date/time of Admission. / / , . .

[Redacted patient information]

Glucose infusion rates yes/no

Drugs yes/no

If yes (mention). Nifedipine/hydrocortisone/octreotide Outcome.

- * Discharge

- * Death

- * Morbidity at the time of discharge

ANNEXURE III

Informed Consent Form

Title of Research study: "Clinical Profile and short term outcomes of neonatal hypoglycemia in neonatal Intensive care unit in a tertiary care Hospital - prospective observational study."

Principal Investigator's Name:

Name of Parent Name : Name of Institution:

Date of Birth : Date:

Gender :

Address:

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For any queries you may contact:

MASTER CHART

Sr. No.	Gender	Parity	Maternal risk factors													
1	Female	Prim i	None													
2	Female	Prim i	None													
3	Male	Mult i	PIH													
4	Female	Prim i	Anemia													
5	Male	Mult i	PIH													
6	Male	Mult i	GDM													
7	Male	Prim i	PIH													
8	Female	Prim i	PIH													
9	Male	Prim i	PIH													
10	Male	Prim i	GDM													
11	Female	Prim i	PIH													
12	Female	Mult i	GDM													
13	Female	Mult i	None													
14	Male	Prim i	None													
15	Female	Mult i	PIH													
16	Female	Mult i	None													
17	Female	Prim i	None													
18	Male	Prim i	GDM													
19	Male	Prim i	None													
20	Male	Mult i	PIH													
21	Female	Mult i	None													
22	Male	Prim i	Anemia													
23	Male	Mult i	PIH													
24	Male	Mult i	PIH													
25	Male	Mult i	PROM													
26	Female	Mult i	PIH													
27	Female	Mult i	PIH													
28	Male	Mult i	PIH													
29	Female	Prim i	Anemia													
30	Female	Mult i	None													
31	Female	Prim i	None													
32	Female	Mult i	PIH													
33	Female	Mult i	Oligohyd rannios													
34	Female	Mult i	PIH													
35	Female	Prim i	PIH													

36	Male	Prim i	None													
37	Female	Mult i	PIH													
38	Female	Mult i	GDM													
39	Male	Prim i	PIH													
40	Male	Prim i	PIH													
41	Male	Prim i	PIH													
42	Male	Prim i	PIH													
43	Female	Prim i	PIH													
44	Male	Prim i	PIH													
45	Male	Mult i	PIH													
46	Female	Mult i	Anemia													
47	Female	Mult i	None													
48	Female	Prim i	None													
49	Male	Mult i	None													
50	Female	Prim i	PIH													
51	Female	Prim i	None													
52	Male	Mult i	PIH													
53	Male	Mult i	None													
54	Male	Prim i	PIH													
55	Male	Prim i	GDM													
56	Male	Prim i	None													
57	Female	Prim i	GDM													
58	Male	Prim i	GDM													
59	Male	Mult i	Oligohyd ramnios													
60	Male	Prim i	None													
61	Female	Prim i	GDM													
62	Female	Prim i	PIH													
63	Female	Mult i	PIH													
64	Male	Mult i	GDM													
65	Male	Mult i	Oligohyd ramnios													
66	Male	Prim i	None													
67	Female	Prim i	None													
68	Female	Prim i	PIH													
69	Female	Mult i	Anemia													
70	Male	Prim i	GDM													
71	Male	Mult i	GDM													
72	Female	Mult i	Anemia													
73	Female	Mult i	PIH													
74	Female	Mult i	None													
75	Female	Prim i	GDM													
76	Male	Mult i	None													

77	Male	Prim i	GDM														
78	Male	Multi i	GDM														
79	Male	Multi i	Anemia														
80	Male	Multi i	PIH														
81	Female	Prim i	PIH														
82	Male	Prim i	None														
83	Female	Multi i	None														
84	Female	Multi i	None														
85	Male	Prim i	PIH														
86	Female	Prim i	GDM														
87	Male	Multi i	PIH														
88	Male	Prim i	None														
89	Male	Prim i	None														
90	Male	Prim i	GDM														
91	Male	Prim i	GDM														
92	Female	Prim i	GDM														
93	Female	Prim i	PIH														
94	Male	Multi i	PIH														
95	Female	Multi i	None														
96	Male	Multi i	PIH														
97	Female	Prim i	GDM														
98	Female	Multi i	PIH														
99	Female	Prim i	None														
100	Male	Prim i	PIH														
101	Male	Multi i	PIH														
102	Female	Multi i	PIH														
103	Female	Prim i	None														
104	Male	Multi i	PIH														
105	Female	Multi i	None														
106	Female	Prim i	GDM														
107	Male	Prim i	GDM														
108	Male	Multi i	None														
109	Female	Prim i	None														
110	Male	Multi i	None														
111	Female	Multi i	GDM														
112	Female	Multi i	None														
113	Male	Prim i	GDM														
114	Male	Prim i	None														
115	Male	Multi i	None														
116	Male	Prim i	Oligohyd ramnios														
117	Female	Multi i	GDM														
118	Male	Multi i	Oligohyd ramnios														

119	Male	Multi	PIH													
120	Male	Multi	PIH													
121	Male	Prim	None													
122	Male	Multi	PIH													
123	Female	Multi	PIH													
124	Female	Prim	GDM													
125	Female	Multi	Anemia													
126	Female	Prim	PIH													
127	Female	Multi	None													
128	Male	Multi	PIH													
129	Male	Multi	PIH													
130	Male	Prim	GDM													
131	Female	Prim	None													
132	Female	Multi	None													
133	Female	Multi	GDM													
134	Female	Prim	None													
135	Male	Prim	PIH													
136	Male	Multi	GDM													
137	Female	Prim	Anemia													
138	Male	Multi	PIH													
139	Female	Prim	PIH													
140	Female	Multi	GDM													
141	Male	Multi	GDM													
142	Male	Multi	None													
143	Male	Prim	GDM													
144	Female	Prim	PIH													
145	Male	Prim	PIH													
146	Female	Prim	GDM													
147	Male	Prim	PIH													
148	Female	Prim	None													
149	Male	Multi	None													
150	Male	Prim	PIH													
151	Female	Prim	None													
152	Male	Prim	Anemia													
153	Female	Multi	PIH													
154	Male	Multi	None													
155	Male	Multi	PIH													
156	Female	Prim	None													
157	Male	Multi	None													
158	Male	Prim	None													
159	Female	Prim	GDM													
160	Male	Multi	GDM													

161	Male	Prim i	PIH														
162	Female	Mult i	PIH														
163	Female	Mult i	None														
164	Female	Mult i	GDM														
165	Male	Prim i	GDM														
166	Male	Prim i	GDM														
167	Male	Prim i	PIH														
168	Female	Prim i	GDM														
169	Male	Prim i	PIH														
170	Male	Prim i	PIH														
171	Male	Mult i	None														
172	Female	Prim i	GDM														
173	Female	Mult i	None														
174	Female	Prim i	None														
175	Male	Prim i	PIH														
176	Male	Prim i	PIH														
177	Female	Mult i	GDM														
178	Female	Prim i	PIH														
179	Male	Mult i	PIH														
180	Male	Prim i	None														
181	Female	Mult i	None														
182	Male	Mult i	PIH														
183	Male	Prim i	None														
184	Male	Prim i	None														
185	Male	Mult i	None														
186	Male	Prim i	None														
187	Female	Prim i	GDM														
188	Female	Prim i	None														
189	Female	Mult i	GDM														
190	Male	Mult i	PIH														
191	Male	Mult i	PIH														
192	Female	Prim i	PIH														
193	Female	Mult i	PIH														
194	Male	Prim i	GDM														
195	Female	Mult i	GDM														
196	Male	Prim i	PROM														
197	Female	Mult i	GDM														
198	Male	Prim i	GDM														
199	Female	Mult i	GDM														
200	Female	Mult i	PIH														
201	Female	Mult i	PIH														
202	Male	Prim i	GDM														

203	Male	Prim i	Oligohyd ramnios														
204	Female	Prim i	None														
205	Male	Multi i	GDM														
206	Female	Prim i	PIH														
207	Male	Prim i	None														
208	Male	Prim i	GDM														
209	Male	Multi i	None														
210	Male	Multi i	None														
211	Male	Multi i	GDM														
212	Male	Prim i	PIH														
213	Female	Multi i	GDM														
214	Male	Prim i	None														
215	Female	Prim i	GDM														
216	Male	Multi i	PIH														
217	Male	Prim i	PIH														
218	Male	Multi i	PIH														
219	Male	Multi i	GDM														
220	Male	Multi i	Anemia														
221	Male	Prim i	GDM														
222	Male	Prim i	None														
223	Female	Prim i	PIH														
224	Male	Multi i	GDM														
225	Female	Prim i	GDM														
226	Male	Multi i	GDM														
227	Male	Prim i	PIH														
228	Female	Multi i	PIH														
229	Female	Multi i	None														
230	Female	Multi i	PIH														
231	Male	Multi i	None														
232	Male	Multi i	PIH														
233	Female	Prim i	GDM														
234	Male	Prim i	PIH														
235	Male	Multi i	None														
236	Male	Prim i	GDM														
237	Female	Prim i	None														
238	Female	Prim i	None														
239	Male	Prim i	PIH														
240	Female	Prim i	GDM														
241	Male	Prim i	None														

