"A CROSS-SECTIONAL OBSERVATIONAL STUDY ON SERUM ELECTROLYTE ABNORMALITIES DURING AN EPISODE OF NEONATAL SEIZURE AT A TERTIARY CARE CENTRE IN KOLAR"

BY

Dr. DEEPAK MELASANGAM

DISSERTATION SUBMITTED TO
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In partial fulfilment of the requirement for the degree of

DOCTOR OF MEDICINE
IN
PAEDIATRICS

Under The Guidance Of

GUIDE - Dr. KRISHNAPPA.J

Professor and Head of unit

Department of Paediatrics

SDUMC, KOLAR

CO- GUIDE - Dr. SHASHIDHAR K.N
Professor
Department of Biochemistry

DEPARTMENT OF PAEDIATRICS
SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR, KARNATAKA-563103

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SIGNATURE OF THE CO-GUIDE

Dr. SHASHIDHAR K.N

PROFESSOR

DEPARTMENT OF BIOCHEMISTRY

SRI DEVARAJ URS MEDICAL COLLEGE,

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Dr. SUDHA REDDY VR

PROFESSOR AND HOD

DEPARTMENT OF PEDIATRICS

SDUMC, KOLAR

Dr. K.PRABHAKAR

PROFESSOR AND PRINCIPAL

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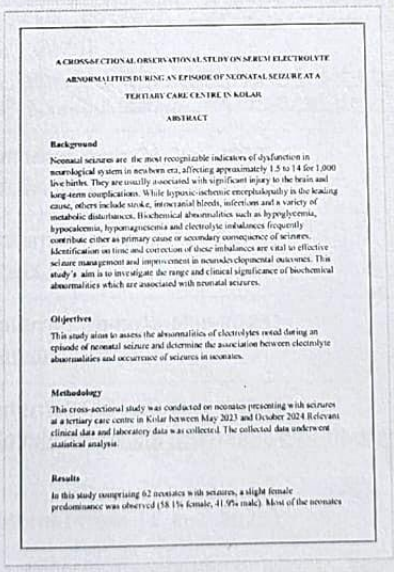

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ABSTRACT

Background

Neonatal seizures are the most recognizable indicators of dysfunction in neurological system in newborn era, affecting approximately 1.5 to 14 for 1,000 live births. They are usually associated with significant injury to the brain and long-term complications. While hypoxic-ischemic encephalopathy is the leading cause, others include stroke, intracranial bleeds, infections and a variety of metabolic disturbances. Biochemical abnormalities such as hypoglycemia, hypocalcemia, hypomagnesemia and electrolyte imbalances frequently contribute either as primary cause or secondary consequence of seizures. Identification on time and correction of these imbalances are vital to effective seizure management and improvement in neonates clopmental outcomes. This study's aim is to investigate the range and clinical significance of biochemical abnormalities which are associated with neonatal seizures.

Objectives

This study aims to assess the abnormalities of electrolytes noted during an episode of neonatal seizure and determine the association between electrolyte abnormalities and occurrence of seizures in neonates.

Methodology

This cross-sectional study was conducted on neonates presenting with seizures at a tertiary care centre in Kolar between May 2023 and October 2024.Relevant clinical data and laboratory data was collected. The collected data underwent statistical analysis.

Results

In this study comprising 62 neonates with seizures, a slight female predominance was observed (58.1% female, 41.9% male). Most of the neonates

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Medical Superintendent &
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Date: **DR. DEEPAK MELASANGAM**

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LIST OF ABBREVIATIONS USED

SIADH – Syndrome of inappropriate antidiuretic hormone secretion

EEG - Electroencephalogram

ICH – Intracranial hemorrhage

HIE – Hypoxic ischemic encephalopathy

TORCH – Toxoplasmosis, Others, Rubella, Cytomegalovirus and Herpes Simplex

FHR – Fetal heart rate

SDH – Subdural hemorrhage

SAH – Subarachnoid hemorrhage

IVH – Intraventricular hemorrhage

IPH – Intraparenchymal hemorrhage

GMH – Germinal matrix hemorrhage

CPAP – Continuous positive airway pressure

CSF – Cerebrospinal fluid

VLBW – Very low birth weight

PTH – Parathyroid hormone

IDM – Infant of diabetic mother

IV – Intravenous

GBS – Group B streptococci

CMV – Cytomegalovirus

HSV – Herpes simplex virus

CT – Computerised tomography

PVL – Periventricular leukomalacia

PET – Positron emission tomography

AED – Antiepileptic drug

RLJH – R L Jalappa Hospital

SPSS – Statistical Package for Social Sciences

NVD – Normal vaginal delivery

LSCS – Lower segment Cesarean section

AGA – Appropriate for gestational age

SGA – Small for gestational age

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Neonatal seizures are the most recognizable indicators of dysfunction in neurological system in newborn era, affecting approximately 1.5 to 14 for 1,000 live births. They are usually associated with significant injury to the brain and long-term complications. While hypoxic-ischemic encephalopathy is the leading cause, others include stroke, intracranial bleeds, infections and a variety of metabolic disturbances. Biochemical abnormalities such as hypoglycemia, hypocalcemia, hypomagnesemia and electrolyte imbalances frequently contribute either as primary cause or secondary consequence of seizures. Identification on time and correction of these imbalances are vital to effective seizure management and improvement in neurodevelopmental outcomes. This study's aim is to investigate the range and clinical significance of biochemical abnormalities which are associated with neonatal seizures.

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Methodology

This cross-sectional study was conducted on neonates presenting with seizures at a tertiary care centre in Kolar between May 2023 and October 2024.Relevant clinical data and laboratory data was collected. The collected data underwent statistical analysis.

Results

In this study comprising 62 neonates with seizures, a slight female predominance was observed (58.1% female, 41.9% male). Most of the neonates were term (72.6%) and delivered through cesarean section (53.2%). A significant proportion (40.3%) had low birth weight. Seizures usually began in the initial three days of life, especially on the second day (37%). Subtle type seizures were the most frequent type (50%), followed by generalized type tonic (29%) and multifocal type clonic seizures (11.3%). Electrolyte abnormalities were present in 66.1% of cases, with hypocalcemia (37.9%) and hypernatremia (31%) being the most common. Subtle type and generalized type tonic seizures were the most often associated with these disturbances. In total, nearly half (46.8%) of seizure cases showed a clear relation to electrolyte imbalances, highlighting the importance of routine biochemical evaluation in case of neonatal seizures.

Conclusion

In this study of neonates with seizures, almost half exhibited electrolyte imbalances, with hypocalcemia and hypernatremia being the most prevalent. While these disturbances are commonly linked to neonatal seizures, no significance in relationship was found between the type of seizure and electrolyte abnormalities. This highlights the importance of routine evaluation of serum electrolytes in all neonates with seizures, regardless of seizure classification. Prompt identification and correction of electrolyte disturbances are essential in improving clinical outcome and bring the risk of seizure recurrence down in this vulnerable population.

Keywords: Seizures, biochemical abnormalities, metabolic disturbances, hypocalcemia, hypernatremia

INTRODUCTION

INTRODUCTION

Seizures represent one of the commonest, easily identifiable indication of dysfunction in the neurological system in newborns.[1]They affect approximately 1.5 to 14 out of every 1,000 infants and are often linked to brain damage, which can lead to long-term complications.[2] Traditionally, neonatal seizures have been classified into different types, including multifocal clonic, focal clonic, myoclonic, tonic along with subtle seizures.[3] Hypoxic-ischemic injury is the primary cause behind neonatal seizures, with infarction of cerebrum and stroke being the next frequent causes, especially among otherwise healthy full-term infants without previous risk factors.[4] Such strokes usually involve the left middle cerebral artery and commonly lead to clonic seizures on the right side of the body. Additionally, intracranial bleeding accounts for 10–15% of cases, with intraventricular hemorrhage and periventricular hemorrhagic infarction being the predominant types in preterm infants, contributing to nearly 45% of seizures in this group.[5]

Infections of the central nervous system, whether occurring during birth or afterward, can also trigger seizures.[6] Biochemical imbalances frequently play a role, either as a primary cause or a secondary factor.[7] Some metabolic disturbances are temporary and easily treatable, while others stem from inherited disorders.[8] Infants who are born to diabetic mothers, small for gestational age infants, or those who have suffered birth asphyxia have an increased chance of developing hypoglycemia.[9] Late-onset hypocalcemia, frequently linked to formulas which are high in phosphate, is another recognized cause. This condition usually emerges within the first few days after birth and is often associated with hypoglycemia and hypomagnesemia, particularly in infants who have experienced trauma, hemolytic disease, or asphyxia.[10] Hypomagnesemia, defined by serum magnesium levels under 1.5 mg/100 mL cause seizures typically occurring between 2 and 4 weeks of age, and is frequently linked to secondary hypocalcemia.[11] Hypophosphatemia can arise from excessive phosphorus intake, impaired kidney function, or hypoparathyroidism.

Hyponatremia, resulting from fluid overload, kidney dysfunction, or SIADH, is a frequent complication of birth asphyxia and may make managing seizures more challenging.[12]

Neonatal seizures have multiple causes, including primary neurological conditions such as hypoxic-ischemic encephalopathy, central nervous system infections, intracranial hemorrhage, and congenital brain malformations; metabolic disturbances like glucose and electrolyte imbalances; and cryptogenic cases where no clear cause is identified.[13]

Timely diagnosis and management of the underlying pathology are crucial to prevent recurrent seizures, mitigate systemic complications, and reduce the risk of permanent neuronal damage. Biochemical abnormalities frequently accompany neonatal seizures, serving as either primary triggers or secondary manifestations.[14]

The prompt detection and correction of these metabolic disturbances are essential for effective seizure control and optimizing neurodevelopmental outcomes. The intention behind conducting this study was to explore range, clinical importance of biochemical abnormalities linked to neonatal seizure disorders.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

- To assess the abnormalities of electrolytes noted during an episode of neonatal seizure.
- To determine the association between electrolyte abnormalities and occurrence of seizures in neonates.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

SEIZURES:

A clinical seizure, characterized by sudden, abnormal burst of electrical activity in a cluster of neurons, causing a brief disturbance of neurological function. This presents as abnormal motor, sensory or autonomic responses, and may involve altered consciousness.

Seizures are characterized by clinical phenomena that occur alongside EEG seizure activity, making them distinctly epileptic. This definition includes sudden, clinical episodes that are common along with simultaneous EEG detected seizure activity.[15]

Seizures can be primarily detected through clinical means by direct observation. Recent studies that combined monitoring of pattern of EEG with clinical observation have highlighted few key considerations pertaining to seizures detected through clinical assessment in neonates. Firstly, certain motor and behavioural events previously labelled as seizures might not align with EEG-confirmed seizure activity, suggesting that their occurrence may have been overreported in earlier assessments. Secondly, a significant number of seizures detected through EEG show no visible motor or behavioural signs in neonates, implying that the true prevalence of neonatal seizures may have been underrecognized. [16]

Pathophysiology:

Seizures create an imbalance between the brain's energy supply and demand. While cerebral blood flow increases during seizures, it may not be suffice the brain's metabolic requirements.[17] Due to their lower cerebral metabolic rate and simpler neuronal networks, neonates are less susceptible to neuronal injury and cell death compared to adults. They also appear resistant to the toxic effects of glutamate. However, seizures can still inhibit brain growth, alter neuronal circuits, and increase

neuronal excitability. Recurrent seizures during early part of development can impair memory as well as spatial learning.[18] Status epilepticus as well as recurrent seizures can increase the brain's vulnerability to future seizure activity. Magnetic resonance spectroscopy studies have shown that seizures in immature rats can lead to metabolic disturbances and necrotic injury in the thalamus.[19]

INCIDENCE

Neonatal seizures occurrence can be approximated to 1.5 to 3.7 for 1,000 full- term live births, while their prevalence rises to 6–12% among infants who weigh less than 1500 grams at the time of birth.[20]

Types of Seizures:

Identifying seizures is a significant clinical challenge in newborns. These seizures are frequently challenging to recognize, potentially postponing identification of underlying condition and starting suitable plan of management.

According to Volpe JJ[21], there are four main types of seizures in neonates:

(1) Subtle, (2) Tonic, (3) Clonic and (4) Myoclonic.

	Electrographic Seizure	
Clinical seizure	Common	Uncommon
Subtle	+ [a]	
Clonic		
Focal	+	
Multifocal	+	
Tonic		
Focal	+	
Generalized		+
Myoclonic		
Focal,multifocal		+
Generalized	+	

[a] Only specific varieties of subtle seizures are commonly associated with simultaneous EEG seizure activity.

Neonatal Seizure- Types

1 . Subtle

Subtle seizures, the most frequent type in neonates, are especially common among preterm infants. While eye movements, apnoea, or limb movements (e.g., pedalling, boxing-like motions) may coincide with temporal-lobe EEG seizures in some cases, many such phenomena in term infants lack consistent EEG correlation. Apnoeic seizures, rarely epileptic, often accompany staring or mouthing but seldom cause bradycardia.[22] Caution is advised when diagnosing subtle seizures, especially in term infants without EEG confirmation.

2. Clonic Seizures

- **Focal:** Slow, rhythmic jerking of one body side (e.g., face/limbs), often post-ictally unconscious. Typically linked to focal pathology (e.g., stroke) but may occur in metabolic disorders.[23]
- **Multifocal:** Migratory jerks across limbs, usually non-Jacksonian. Both types strongly correlate with EEG seizures.

3. Tonic Seizures

- **Focal:** Sustained limb/trunk posturing, always EEG-associated.
- **Generalized (GTS):** Limb extension/flexion (e.g., "fencing posture"); 85% lack EEG correlation.

4. Myoclonic Seizures

Rapid jerks (often flexor-dominant) rarely linked to EEG seizures. Focal (e.g., arm jerks), multifocal, or generalized;

FEW CAUSES OF NEONATAL SEIZURES:[24]

1. Hypoxic ischemic encephalopathy
2. Intracranial hemorrhage (ICH)
3. Central nervous system infections
4. Congenital malformations
5. Congenital malformations
6. Metabolic
7. Withdrawal from drugs and toxins
8. Benign nature neonatal seizures

Important etiologies and their time of onset are shown below**:**

1st day	Hypocalcemia , HIE , accidental injection of local agents of anaesthetics, pyridoxine dependency.
1 - 3 days	Hypoglycemia, , metabolic errors of inborn nature
4 to 7 days	TORCH infections, Meningitis, development malformations.
More than 7 days	Late - onset hypocalcemia, Late - onset meningitis

1. Hypoxic Ischemic Encephalopathy (HIE)

Hypoxic Ischemic Encephalopathy (HIE) is one of the primary causative of seizures in newborns. Perinatal asphyxia is reduced oxygen supply to fetal or neonate's brain, and this oxygen deprivation results in abnormal neurological signs after birth, the condition is termed HIE. This type of brain injury can arise from issues such as inadequate placental gas exchange, compromised blood flow due to umbilical cord compression, or postnatal complications like respiratory or cardiac failure. Severe oxygen deprivation during labor can lead to newborns with weakened heart and lung function, often reflected through less Apgar scores, which aggravates hypoxic - ischemic brain damage. Clinically, affected infants typically exhibit altered consciousness, abnormal muscle tone, impaired reflexes, forming recognizable syndrome.[25]

Risk factors - HIE:[26]

1. Chronic utero-placental insufficiency
2. Extended 2nd stage of labour (Greater than 120 mins)
3. Shoulder dystocia,
4. Extended & abnormal FHR
5. Macrosomia

Clinical Features:

Sarnat has classified the HIE in 3 distinct stage(s) according to clinical evaluation of newborn.[27]

Level of consciousness	Stage 1 (mild) Hyperalert	Stage 2 (moderate) Lethargic/obtunded	Stage 3 (severe) Stuporous
Neuromuscular control			
Muscular tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch	Overactive	Overactive	Decreased/absent
Segmental myoclonus	Present	Present	Absent
Complex reflexes			
Suck	Weak	Weak/absent	Absent
Moro	Strong	Weak	Absent
Oculovestibular	Normal	Overactive	Weak/absent
Tonic neck	Slight	Strong	Absent
Autonomic function			
Pupils	Mydriasis	Miosis	Variable
Heart rate	Tachycardia	Bradycardia	Variable
Bronchial/salivary secretions	Sparse	Profuse	Variable
Gastrointestinal motility	Normal/decreased	Increased/diarrhea	Variable
Seizures	None	Common/focal or multifocal	Uncommon
EEG	Normal/decreased	Early low voltage continuous delta and theta, later periodic, seizues focal 1–1.5 Hz spike-wave	Early periodic pattern with isopotential phases, later isopotential
Duration	<24 h	2–14 days	Hours–weeks

EEG, electroencephalogram.

Hypoxic-Ischemic Encephalopathy (HIE) presents with a broad range of clinical symptoms, varying in severity as mild to profound. Its most severe form, early phase form—typically lasting up to 12 hours after the injury—shows signs of brain dysfunction. During this time, affected newborns may appear unresponsive or comatose and exhibit irregular or periodic breathing patterns.[25]

Seizures—ranging from subtle to tonic or multifocal clonic types—are seen in around 50% of infants with moderate to severe asphyxia, usually occurring between 6 to 24 hours after the initial injury. Infants with more critical involvement may show worsening central nervous system function within 24 to 72 hours, progressing to deep coma, sustained apnoea, and signs of brainstem failure.[28]

Asphyxia can also cause injury to other organs, with the kidneys being particularly vulnerable. This often leads to acute tubular necrosis. Ongoing low urine output (less than 1 ml/kg/hour) strongly correlated with severity of HIE, is associated with poor prognosis in about 90% of cases.[29]

2. Intracranial Hemorrhage (ICH)

Intracranial bleeding (ICH) is major reason of seizures in newborn, with its incidence ranging from 2% to more than 30%, depending on the infant's gestational age and type of hemorrhage.[30]

Bleeding in the skull can varyingly occur in several locations:

(1) outside brain, such as in epidural, subdural or subarachnoid spaces;

(2) within brain tissue itself, affecting areas like the cerebrum or cerebellum; and (3) inside the brain's ventricles, often originating in subependymal germinal matrix or choroid plexus.

Frequency, causes, clinically distinct signs, diagnostic approaches, treatment options, and outcomes vary according to the hemorrhage's location, severity, and the infant's developmental maturity.

Diagnosis typically begins with clinical suspicion, particularly when a newborn exhibits seizures, irritability, decreased responsiveness, or focal neurological signs, and is confirmed through neuroimaging.

Treatment is tailored to the hemorrhage's size and location, as well as the baby's neurological condition. The main focus is on managing complications such as seizures and hydrocephalus following the bleed.

a) Subdural Hemorrhage (SDH)

- **Causes:** Trauma during delivery (e.g., breech presentation, instrumental delivery) ruptures bridging veins or dural sinuses (e.g., veins of Galen). Risk factors include macrosomia, thrombocytopenia, and coagulopathies.[31]

- **Symptoms:**
    - **Acute:** Brainstem compression (apnoea, coma) with posterior fossa SDH; seizures or irritability with convexity SDH.
    - **Chronic:** Subdural effusions may cause macrocephaly weeks later.

b) Subarachnoid Hemorrhage (SAH)[32]

- **Causes:** Rupture of leptomeningeal vessels; often asymptomatic or mild (e.g., bloody CSF). Distinguish from IVH extension in preterms.
- **Symptoms:** Seizures or irritability; rarely life-threatening.

c) Intraparenchymal Hemorrhage (IPH)

- **Causes:**
    - **Term infants:** Trauma or hypoxia.
    - **Preterms:** Cerebellar hemorrhage (5–10% at autopsy) or venous infarction.
- **Symptoms:** Focal signs (e.g., hemiparesis) in term infants; often silent in preterms.

d) Germinal Matrix/Intraventricular Hemorrhage (GMH/IVH)[33]

- **Causes (Preterms):** Fragile germinal matrix vessels affected by:
    - **Hemodynamic shifts** (e.g., fluctuating blood flow, hypertension).
    - **Coagulopathies** or venous congestion (e.g., high CPAP).

- **Complications:**

    1. **Hemorrhagic infarction** (venous obstruction → "fan-shaped" necrosis).

    2. **Hydrocephalus** (CSF obstruction or impaired absorption).

- **Symptoms:**

    - Often asymptomatic; seizures (tonic posturing, eye-rolling) may occur.

    - Hydrocephalus presents later (bulging fontanelle, macrocephaly)

3) <u>Metabolic disorders</u>

a) <u>Hypoglycemia</u>[34]

- Definition & Significance:

    - No universal threshold; typically < 40 mg/deciliter ~ (2.2 mmol/L) during the first 24 – 48 hours

    - Common in newborns due to transitional metabolic shifts; most cases are transient

    - Healthy term infants often maintain glucose via early feeding

- Clinical Concerns:

    - Transient hypoglycemia: Usually benign with prompt treatment

    - Persistent hypoglycemia: Suggests endocrine disorders (e.g., hyperinsulinemia) and may pose neurodevelopmental risks, though causality remains unclear

- Management:

    - Early breastfeeding/formula to stimulate endogenous glucose production

- IV dextrose for refractory cases (e.g., 10% dextrose bolus followed by infusion)
- Screening for endocrine/metabolic disorders if hypoglycemia persists

Hyperglycemia[35]

- Occurrence: Rare in term newborns but frequent in VLBW infants (<1500 g)
- Causes:
  - Immature insulin response, stress, or iatrogenic (excessive dextrose infusion)
- Risks: Osmotic diuresis, dehydration, worsened outcomes in prematurity
- Management:
  - Adjust dextrose infusions; consider insulin therapy if persistent (rarely needed)

b) <u>Hypocalcemia in Neonates</u>

Definition:

Neonatal hypocalcemia , defined by total serum calcium below 7 mg/deciliter or ionized calcium level less than 4 mg/deciliter ~ (1 mmol/L). In case of very low birth weight (VLBW) infants, ionized calcium levels ranging ~ 0.8 to 1 mmol/L frequently don't produce symptoms. However, term or near-term infants (>32 weeks gestation) may exhibit symptoms when ionized calcium falls below 1 mmol/L.[36]

Pathophysiology:

Calcium has a critical role in cellular and biochemical function. Disruptions in calcium levels are frequently observed in neonates.[37]

Hormonal Regulation:

Hormones like parathyroid hormone (PTH) as well and calcitriol (1,25-dihydroxyvitamin D) have crucial role in serum calcium balance. PTH is secreted in response to declining extracellular calcium, prompting calcium release from bones, enhanced renal calcium reabsorption, and increased synthesis of calcitriol. Calcitriol aids in gastrointestinal absorption of calcium and phosphate, and mobilization from bones.

Metabolism of Vitamin D:

Vitamin D produced in skin when it gets exposed to ultraviolet light or acquired from dietary sources. It is first converted in liver to 25 (OH) D, then further processed in kidneys into active 1,25 $(OH)_2$ D (calcitriol).[38]

Causes of Neonatal Hypocalcemia[39]

a) **Prematurity:** Reduced end-organ response to PTH despite adequate secretion.
b) **Infants of Diabetic Mothers (IDMs):** Incidence is 25–50%, especially with poor glycemic control. Mechanisms include elevated calcitonin, low PTH, altered vitamin D metabolism, and increased phosphate.
c) **Perinatal Asphyxia:** Often presents with low calcium and high phosphate due to stress and impaired intake.
d) **Congenital Conditions:** Absent or hypoplastic parathyroid glands in DiGeorge syndrome or Kenny-Caffey syndrome.
e) **Metabolic Disorders:** Pseudohypoparathyroidism, magnesium deficiency (including inborn errors), vitamin D deficiency (rare in early life), alkalosis, and bicarbonate use.
f) **Other Factors:** Citrate-containing blood transfusions, sepsis, phototherapy, and shock.

Late-Onset Hypocalcemia:

Usually occurring between the third and fifth day of life, particularly in term infants who are formula-fed. Often associated with hyperphosphatemia and sometimes maternal vitamin D deficiency.

Diagnosis[40]

Clinical Signs:

Symptoms range from subtle (jitteriness, stridor, increased reflexes) to severe (seizures, apnoea). Early onset is often silent, while late-onset may present with seizures.

History and Examination:

May include feeding history (e.g., exclusive breastfeeding or cow's milk use), abnormal movements, and lethargy. Physical findings may be minimal.

Laboratory Assessment:

Total calcium is divided into ionized (biologically active, ~50%), protein-bound (~40%), and anion-complexed (~10%). Ionized calcium is the preferred diagnostic measure.

Newborn calcium levels typically dip in the first 24–48 hours post-birth, then rise gradually.

Severe vitamin D deficiency (less than 10–12 ng/dL) should be suspected if clinical symptoms continue despite initial treatment.

Monitoring

For at-risk infants (e.g., VLBW, IDMs, birth asphyxia):

Measure ionised calcium levels - 12, 24, and 48 hours postnatally.

Assess serum phosphate and magnesium if at all hypocalcemia is detected.

Advanced hormone tests (PTH, 25 (OH) D, 1,25 $(OH)_2D$) are rarely required unless the condition is refractory to treatment.

Treatment[40]

Calcium Supplementation:

IV calcium (10% calcium gluconate) is standard for acute management. Careful monitoring is essential to avoid bradycardia or necrosis due to infusion complications.

Oral Options:

Calcium glubionate syrup is commonly used but may cause GI side effects. Continuous infusion may be needed for severe cases.

Preventive Use:

For critically ill newborns (e.g., with RDS, sepsis), calcium infusions help maintain normal levels.

c) Late-Onset with Hyperphosphatemia:[41]

Management includes switching to low-phosphate formulas (e.g., Similac PM 60/40), increasing calcium intake, and avoiding formulas high in phosphorus.

Vitamin D is generally unnecessary unless deficiency is proven.

d) Hypomagnesemia[42]

Low magnesium can impair PTH secretion and reduce its effectiveness, contributing to hypocalcemia.

Primary Hypomagnesemia: Rare autosomal recessive disorder presenting in the first weeks with seizures and tetany.

Transient Hypomagnesemia: Often resolves with calcium therapy.

e) Sodium Imbalances

Hyponatremia: Rarely causes neonatal seizures on its own but may occur with brain trauma, infection, or asphyxia.

Hypernatremia: Occasionally seen with adrenal disorders or inappropriate IV fluids.

4) Local Anesthetic Toxicity

Accidental fetal injection during maternal regional anaesthesia can lead to seizures soon after birth, mimicking asphyxia. Symptoms include low Apgar scores, fixed pupils, lack of eye movement, bradycardia, and hypotonia. Diagnosis is confirmed via blood/CSF anaesthetic levels.[43]

5) Additional Metabolic Causes

Inborn Error of Metabolism: Disorders affecting amino acids (or) organic acids metabolism frequently manifest with seizures, acidosis, and elevated ammonia levels.[44]

Pyridoxine Dependency: A rare type autosomal along with recessive condition causing seizures within hours of birth, responsive to IV pyridoxine.[45]

Glucose Transport Deficiency: Leads to seizures and developmental delays due to impaired brain glucose delivery, treatable with a ketogenic diet.[46]

6) Infections

Meningitis: About one-third of neonatal meningitis cases present with seizures. Early-onset types occur within 48 hours due to birth canal infection (e.g., GBS, E. coli, Listeria).

Late-Onset: Usually nosocomial (e.g., Staph aureus, Pseudomonas). Viral infections like CMV and HSV can also cause seizures, typically within the first few days.[47]

Neonatal Seizures- Diagnosis [48]

Accurate diagnosis of neonatal seizures often begins with a detailed prenatal and perinatal history and thorough physical examination. While advanced tests are useful, many causes can be identified with basic clinical assessments.

Initial Investigations:

Priority is given to identifying **treatable and life-threatening causes** such as:

- **Hypoglycemia**: Blood glucose <40 mg/dL.
- **Bacterial Meningitis**: Suggested by CSF findings (increased WBCs and protein, decreased glucose); confirmed by CSF culture.

Other essential blood tests include electrolytes (Na^+, K^+, Ca^{2+}, Mg^{2+}, PO_4^{3-}). Additional metabolic and imaging tests depend on clinical signs. **Focal seizures** often warrant cranial ultrasound or CT scan to detect localized brain injuries.

EEG (Electroencephalography):

- Mainly done in the **interictal** period.
- Useful to detect subtle seizures, assess brain background activity, and evaluate prognosis.
- Seizures may not show clinical signs; electrical seizures are often **brief (<2 minutes)** and **focal**, typically from temporal or central regions.
- Skilled interpretation is crucial due to age-specific EEG features.

Ultrasound (USG):

- Widely available and non-invasive.
- Helps detect **periventricular hemorrhage (PVH)** and **periventricular leukomalacia (PVL)**, especially in preterm infants.
- Hypoxic-ischemic injury appears as increase in echodensity, though distinguishing from hemorrhage can be difficult.

CT Scan:

- Valuable in diagnosing **intracranial hemorrhages** and **hypoxic-ischemic injuries**, especially in term infants.
- Best done between **2–4 days of life** to visualize early brain damage.
- Later scans may show atrophy or multicystic changes.
- Specific patterns (e.g., basal ganglia or parasagittal injuries) may aid diagnosis and prognosis.

MRI:

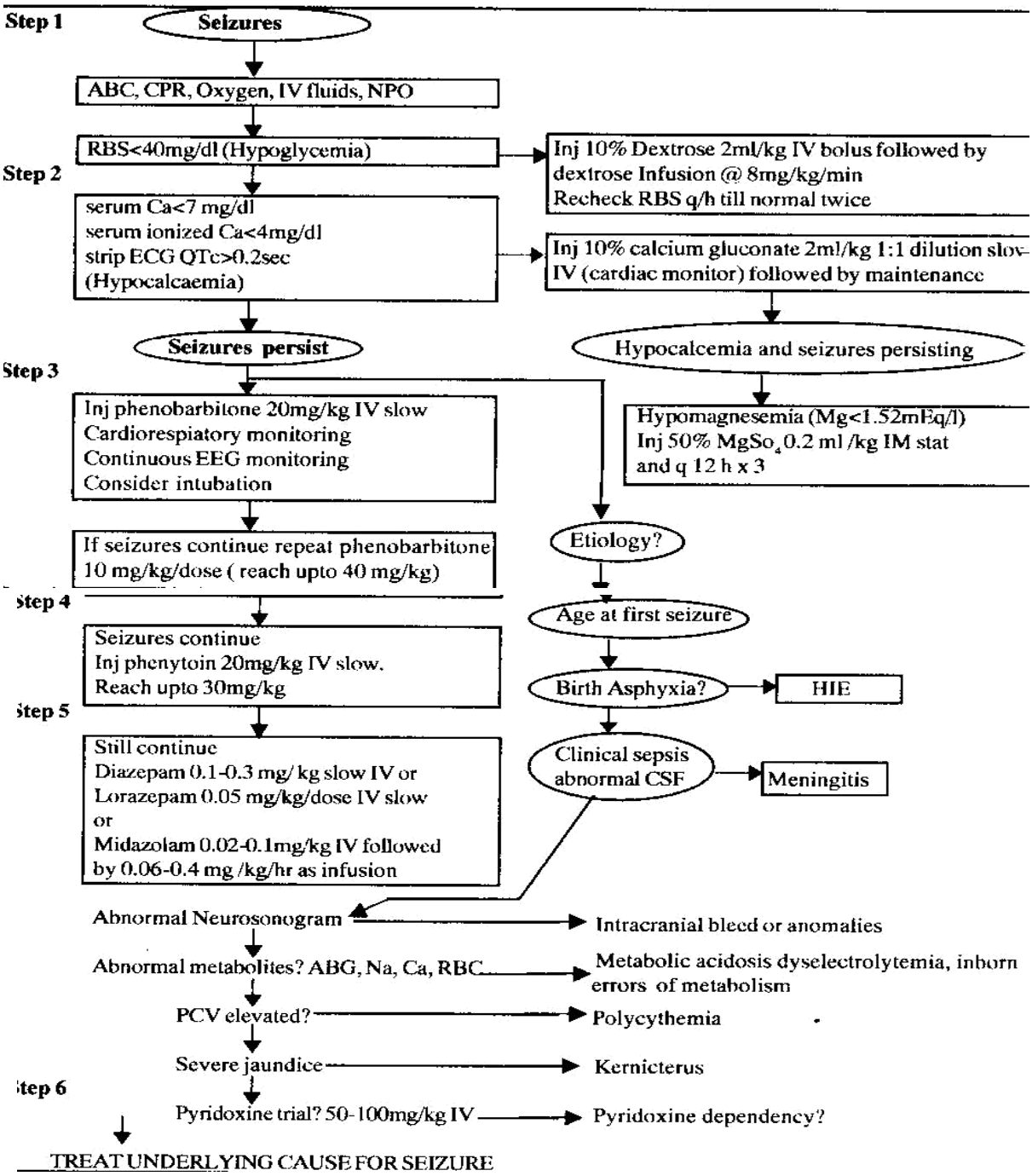
- It is more effective than CT at identifying subtle brain injuries.
- Can identify the **type and timing** of injury, offering valuable prognostic insight.

- Normal MRI in neonates with seizures usually suggests a **favorable outcome** with minimal neurological deficits.

PET Scan:

- **Not used routinely**, but helpful in research or select cases to evaluate **metabolic brain activity** in hypoxic-ischemic injury.

Neonatal Seizures - Management : First steps of management in neonatal seizure involve stabilizing vital signs, identifying and addressing any quickly reversible causes, and confirming the diagnosis through clinical evaluation or EEG. While starting targeted treatments for other manageable conditions is important, it should not postpone the timely administration of anticonvulsant medication.[49] Here, approach in management is shown below[50]


Step 1
Seizures
ABC, CPR, Oxygen, IV fluids, NPO
RBS<40mg/dl (Hypoglycemia)
Inj 10% Dextrose 2ml/kg IV bolus followed by dextrose Infusion @ 8mg/kg/min
Recheck RBS q/h till normal twice
Step 2
serum Ca<7 mg/dl
serum ionized Ca<4mg/dl
strip ECG QTc>0.2sec
(Hypocalcaemia)
Inj 10% calcium gluconate 2ml/kg 1:1 dilution slow IV (cardiac monitor) followed by maintenance
Seizures persist
Hypocalcemia and seizures persisting
Step 3
Inj phenobarbitone 20mg/kg IV slow
Cardiorespiatory monitoring
Continuous EEG monitoring
Consider intubation
Hypomagnesemia (Mg<1.52mEq/l)
Inj 50% MgSo4 0.2 ml /kg IM stat and q 12 h x 3
If seizures continue repeat phenobarbitone 10 mg/kg/dose (reach upto 40 mg/kg)
Etiology?
Step 4
Seizures continue
Inj phenytoin 20mg/kg IV slow.
Reach upto 30mg/kg
Age at first seizure
Birth Asphyxia?
HIE
Step 5
Still continue
Diazepam 0.1-0.3 mg/ kg slow IV or
Lorazepam 0.05 mg/kg/dose IV slow
or
Midazolam 0.02-0.1mg/kg IV followed by 0.06-0.4 mg /kg/hr as infusion
Clinical sepsis abnormal CSF
Meningitis
Abnormal Neurosonogram
Intracranial bleed or anomalies
Abnormal metabolites? ABG, Na, Ca, RBC
Metabolic acidosis dyselectrolytemia, inborn errors of metabolism
PCV elevated?
Polycythemia
Severe jaundice
Kernicterus
Step 6
Pyridoxine trial? 50-100mg/kg IV
Pyridoxine dependency?
TREAT UNDERLYING CAUSE FOR SEIZURE

STUDIES ON BIOCHEMICAL ABNORMALITIES IN NEONATAL SEIZURES

In 2024, Kyasa S et al., study reveals high frequency metabolic disturbances of neonates exhibiting seizures, with glucose and calcium deficiencies emerging as the predominant etiological factors. These observations underscore the critical need for standardized metabolic evaluation in seizure management protocols to facilitate rapid identification of reversible causes. Timely intervention for these imbalances, especially in early-onset seizure cases, may mitigate potential neurological sequelae and enhance prognostic outcomes. The findings advocate for the incorporation of comprehensive metabolic screening into routine neonatal seizure workups, while highlighting the necessity for additional investigations to assess longitudinal outcomes and refine therapeutic approaches.[51]

In 2024, Diggikar S et al in their study stated that some large observational studies with limited-quality data suggest a notable link between low blood sugar in newborns and later neurodevelopmental issues. However, more research with extended follow-up periods is crucial to establish clear threshold levels and assess the long-term effects beyond early childhood.[52]

In 2024, Spenard S et al conducted a study stating that Seizures during the neonatal period continue to represent a critical neurological emergency requiring admission to neonate intensive care units worldwide. Neonate seizures (NS) linked to heightened risk of neurodevelopmental disabilities and increased mortality.[53]

In 2024, Li H et al in their study stated that while rapid sodium correction was effective in our case, safety or effectiveness of prompt administration 3% hypertonic saline to treat seizure caused by severity of hyponatremia require in depth investigation through research.[54]

In 2022, Dr. Deepak Sharma, et. al. in their study noted that Birth asphyxia is significant risk factor for neonatal hypocalcaemia. Other associated risk factors include infants of diabetic mothers,

formula feeding, and neonates requiring bag-and-mask ventilation. Seizures were the most common clinical manifestation. Given this association, low level of calcium should be expected in an asphyxiated neonate as timely interventions are essential to prevent complications.[55]

In 2021, Falsaperla R et al reported This systematic review focuses on inborn error of metabolism (IEMs) that contribute to neonate seizures and epilepsy, specifically examining those not identified by expanded newborn screening (ENS) via tandem mass spectrometry. It outlines the associated clinical signs, biochemical markers, and recommended diagnostic approaches. Importantly, our findings suggest that infants who received only conventional anti-epileptic drugs (AEDs) experienced less favorable neurological outcomes than those treated with metabolic-specific therapies, such as vitamin B6 or the ketogenic diet. [56]

In 2021, Mitsiakos G et al in their study stated that Maternal lab tests showed significantly elevated calcium levels along with increased parathyroid hormone concentrations, attributed to a previously undetected parathyroid adenoma that had gone unnoticed during prenatal care.[57]

In 2020, Pisani F et al in their study elicted that Neonate electroclinical seizures are bundled with various risky factors, complications during pregnancy or the neonatal period, lesser Apgar scores, requirement for resuscitation at time of birth, moderate to severity of intraventricle hemorrhage (grades of II – IV) in preterms and perinatal asphyxia or hypoxic - ischemic encephalopathy (HIE) in terms. Due to these potential risks, continuous and careful observation for seizures is crucial throughout the neonatal period.[58]

In 2019, Pasi R et al in a study elicited , Hyponatremia was significantly prevalent in preterm neonates ($p = 0.020$). Additionally, mortality rates were notably higher among neonates with hyponatremia ($p = 0.003$). Furthermore, a strong association was observed between hyponatremia and Hypoxic Ischemic Encephalopathy (HIE), with a statistically significant correlation ($p < 0.001$).[59]

In 2016, Chen BB et al in a study observed Magnesium levels must be assessed when evaluating the underlying cause of seizures. In cases of hypomagnesemia and afebrile seizures, treatment should aim to keep magnesium levels above 0.65 mmol/L. While uncommon, genetic forms of hypomagnesemia should be taken into account after excluding more frequent causes.[60]

In 2023, Abdul Rahim et al in a study stated that Neonate seizures are among most common neurological disorders in newborn era and can lead to serious complications if not promptly managed. Timely and comprehensive evaluation, accurate diagnosis, and immediate intervention are essential to minimize the risk of long-term neurological harm. Identifying biochemical abnormalities can serve as an additional tool in determining the underlying cause of seizures.[61]

In 2016, Mishra et al in a study stated Focal - clonic and subtle seizures were most frequently observed type overall. Among primary metabolic seizures, hypocalcemia emerged as the most common biochemical disturbance. Such biochemical imbalances were also frequently linked with other underlying conditions, including birth asphyxia, intracranial hemorrhage, and meningitis. Therefore, identifying and addressing these abnormalities is crucial for effective seizure management.[62]

In 2014, Parvin et al in a study stated that EEG results show a significant statistical link with perinatal asphyxia, septicemia, and meningitis, but they do not correlate with the seizure types or patterns. [63]

MATERIALS & METHODS

MATERIALS AND METHODS

Data Source:

All neonates born at or admitted to RLJH who presented with neonatal seizures.

Study Design:

It's a hospital - based cross-sectional observational type study.

Study Period:

One year six months (May 2023 to October 2024) or till required sample size is met.

Method of collection of data:

This study started after obtaining ethical clearance from the institutional ethics committee as well as consent of the parents.

- All neonates within the inclusion criteria admitted in RLJH during mentioned study period was included in study.
- Following the acquisition of written informed consent from parents or caregivers, neonates were monitored for electrolyte abnormalities.

Inclusion Criteria:

All the term and preterm infants who present with seizures including all inborn, outborn neonates, whose parents or caregivers consent for participation in the study

Exclusion criteria:

- Neonates who had previously been treated with anticonvulsant medication or on ongoing anticonvulsant therapy will not be included in the study.

- Neonates with jitteriness.

Sample Size:

The sample size calculated was based on proportion of hypocalcemia (20%) observed in neonates with seizures, as reported in a study by Abdul Rahim et al., using the appropriate statistical formula

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

.$Z_{1-\alpha/2}$ = The standard normal variate (Z) is 1.96 for a 5% type I error ($P < 0.05$) and 2.58 for a 1% Type I error ($P < 0.01$). Since most studies consider P-values

below 0.05 as significant, a Z value of 1.96 was used in the sample size calculation formula.

P= prevalence based from previous studies

d is Absolute precision

P = 20% or 0.20

q = 80% or 0.80

d = 10% or 0.10

Using all above following values at 95% Confidence level , sample size of 62 subjects with neonatal seizure will include in this study.

Data Analysis :

Data will be recorded in Microsoft Excel and analyzed using SPSS version 22. Categorical variables will be expressed as frequencies and percentages, with the Chi-square test applied to

determine statistical significance. Continuous variables will present as means with standard deviation and the independent t-test will be used for assessing differences between means. A P-value less than 0.05 will regard as statistically significant.

Sampling Technique: Simple random sampling.

Graphical representation of data:

Microsoft Excel and Microsoft Word will be utilized to create various graphic representation of the data with bar charts and pie charts.

Methodology:

This study will be carried out at R.L. Jalappa Hospital, which is affiliated with Sri Devaraj Urs Medical College, a part of Sri Devaraj Urs Academy of Higher Education and Research.

Data from neonates presenting with neonatal seizures admitted to our hospital will be collected. Antenatal, perinatal and postnatal history will be collected and recorded in a predetermined proforma. Neonates admitted with seizures will be studied for electrolyte abnormalities.

From the present study, primary outcomes of interest are electrolyte abnormalities such as hyponatremia/hypernatremia, hypomagnesemia/hypermagnesemia, hypocalcemia/hypercalcemia in isolation or in combination in a neonate with seizures.

Serum sodium levels will be measured on venous blood sample and analysed using potentiometric method.[64,65] Hyponatremia is sodium value < 133 mmol/L in serum and hypernatremia is sodium value > 146 mmol/L in serum.[66,67]

Serum magnesium levels will be measured on venous blood sample and analysed using the Formazan dye method.[68] Hypomagnesemia is magnesium level in serum < 1.6 mg/dl and hypermagnesemia is magnesium level in serum > 3.1 mg/dl.[69]

Serum calcium levels will be measured on venous blood sample and analysed using the ARSENAZO-3 method.[70] Hypocalcemia is calcium level in serum < 7 mg/dl and hypercalcemia is calcium level in serum > 11 mg/dl.[71]

If any electrolyte abnormalities are detected, then that neonate will be considered as case of neonatal seizure due to electrolyte abnormalities and will be managed immediately according to institutional protocol.

RESULTS

Table 1: Gender distribution among study cases

Gender	Number	%
Male	26	41. 90%
Female	36	58.10%
Total	62	100 .00%

In this study, 62 neonates presenting with seizures were enrolled. Of these, 26 (41.90%) were male and 36 (58.10%) were female, indicating a modest female predominance. The female-to-male ratio was approximately 1.38:1.

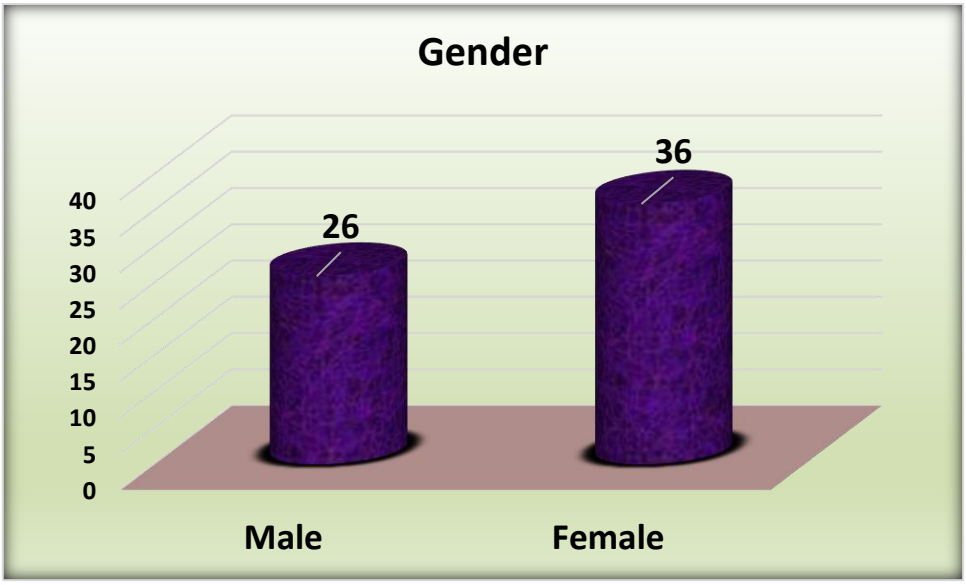


Figure 1: Gender distribution among the study cases

Table 2: Gestational Age among the study cases

Gestational Age	No. of cases	Percentage
Term	45	72.60%
Preterm	17	27.40%
Total	62	100.00%

Among the 62 neonates with seizures in this study, term infants accounted for the majority with 45 cases (72.60%), while 17 cases (27.40%) involved preterm infants. This indicates a higher prevalence of seizures among term neonates in the studied population

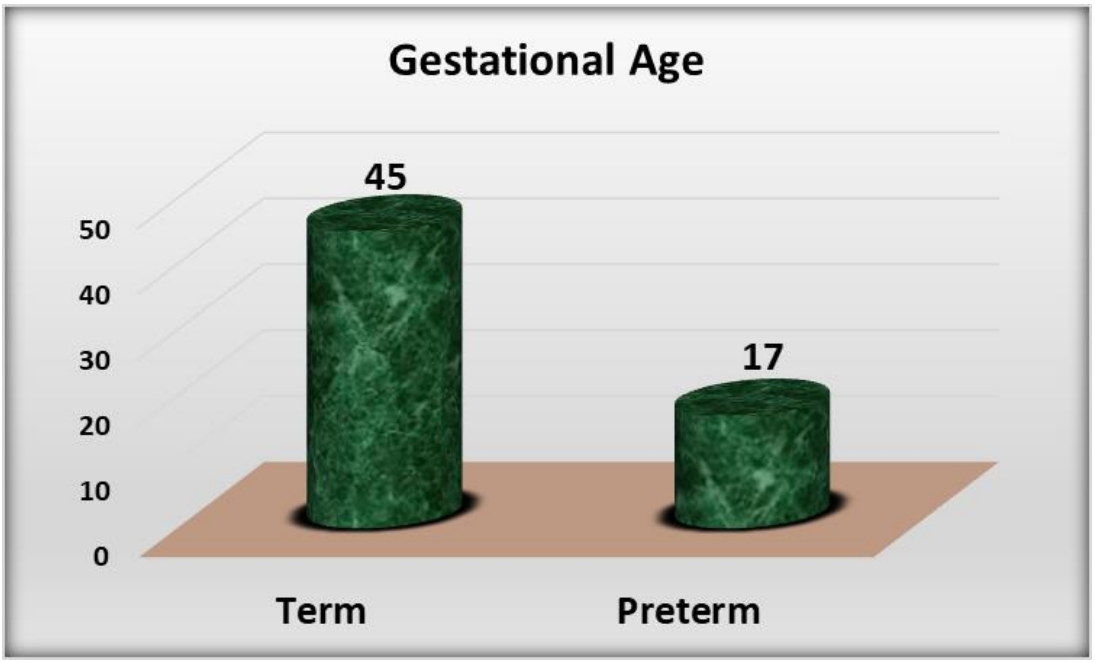


Figure 2: Gestational Age among the study cases

Table 3: Delivery type among the study cases

Delivery type	No. of cases	Percentage
Normal (NVD)	29	46.80%
LSCS	33	53.20%
Total	62	100.00%

Among the 62 neonates included in the study who presented with seizures, 29 (46.80%) were delivered through normal vaginal delivery (NVD), while a slightly higher proportion, 33 neonates (53.20%), were delivered via lower segment cesarean section (LSCS). This distribution indicates that neonate seizures were marginally frequently common in infants delivered through LSCS in this cohort. The mode of delivery can influence neonatal outcomes, especially in cases where LSCS is performed due to fetus in distress or complications that increase the risk.

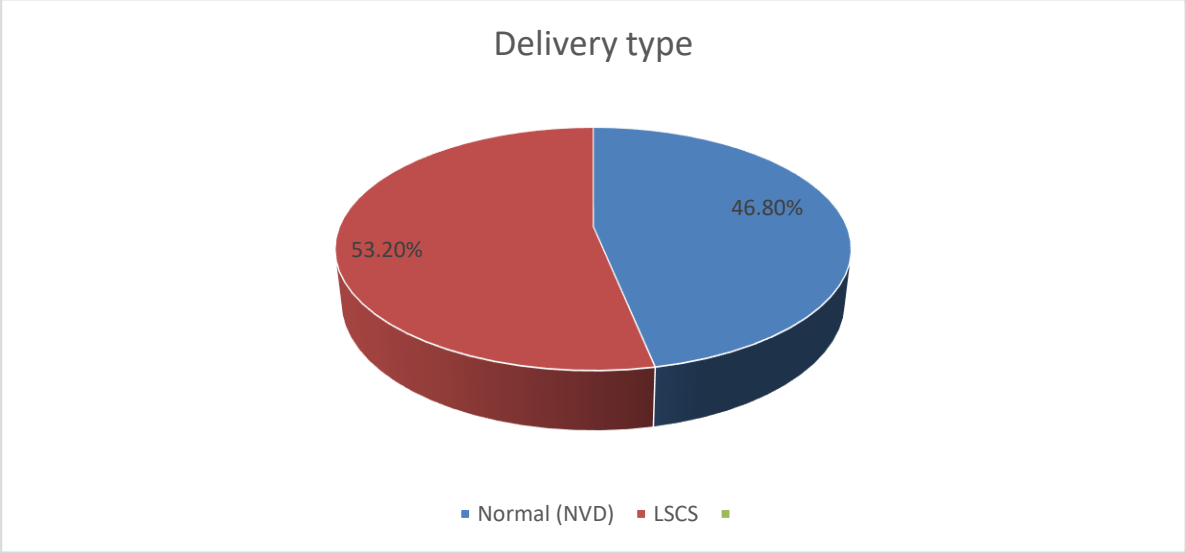


Figure 3 : Delivery type among the study cases

Table 4: Birth weight (kg) among the study cases

Birth weight (kg)	No. of cases	Percentage
Low Birth weight	25	40.30%
Normal Birth weight	37	59.70%
Total	62	100.00%

Out of the 62 neonates with seizures included in this study, 25 neonates (40.30%) had low weight-at-birth (< 2.5 kg), while 37 neonates (59.70%) had normal birth weight. This indicates that a significant proportion of seizure episodes occurred in neonates with low weight, which is recognized risk factor in various neonate complications

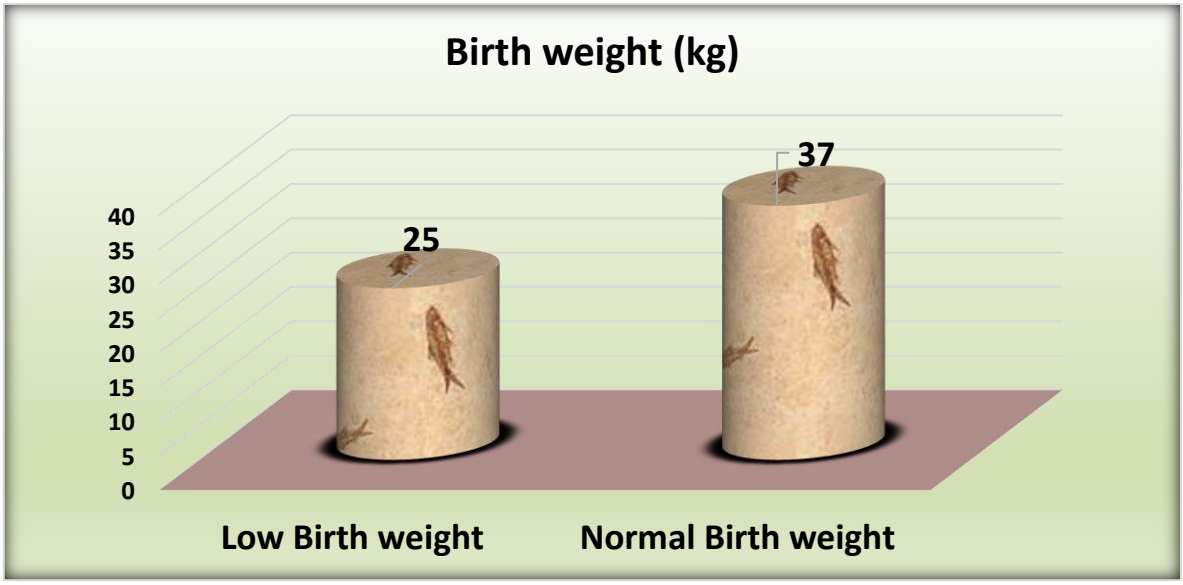


Figure 4: Birth weight (kg) among the study cases

Table 5 : The day of onset of neonatal seizures among the study cases

The day of onset of neonatal seizures	Number	%
1 day	18	29.00%
2 day	23	37.00%
3 day	8	12.90%
4 day	6	9.70%
5 day	3	4.90%
6 day	1	1.60%
7 day	0	0.00%
8-28 day	3	4.90%
Total	62	100.00%

In the present study, the onset of seizure occurred most frequently on **second day of life**, with **23 cases (37.00%)**, followed by **18 cases (29.00%)** on the **first day**. The third and fourth days accounted for **8 cases (12.90%)** and **6 cases (9.70%)**, respectively. Fewer cases were seen on the fifth and sixth days, with **3 cases (4.90%)** and **1 case (1.60%)** each. Notably, no seizures were recorded on the seventh day. Seizures occurring during the **late neonatal period (8–28 days)** were seen in **3 cases (4.90%)**.

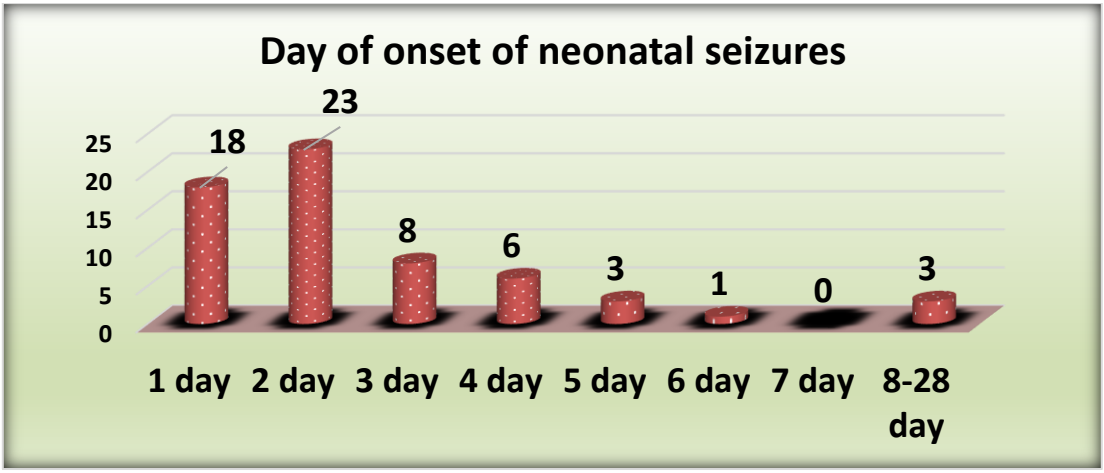


Figure 5: Day of onset of neonatal seizures among the study cases

Table 6: Types of seizure

Types of seizure	No. of cases	Percentage
Focal clonic	4	6.50%
Generalized tonic	18	29.00%
Multifocal clonic	7	11.30%
Subtle	31	50.00%
Mixed	2	3.20%
Total	62	100.00%

In the current study, subtle type seizures were most frequently observed type, accounts for 31 cases in total(50.00%). These are often difficult to detect clinically and may include eye deviation, lip smacking, or apnoea, underscoring the importance of careful observation in neonates with suspected neurological symptoms. Generalized type tonic seizures were second most commonly observed in 18 cases (29.00%), followed by multifocal - clonic seizures in 7 cases overall (11.30%). Focal type clonic seizures occurred in 4 neonates (6.50%), while mixed types of seizures were the least frequent, with only 2 cases (3.20%).

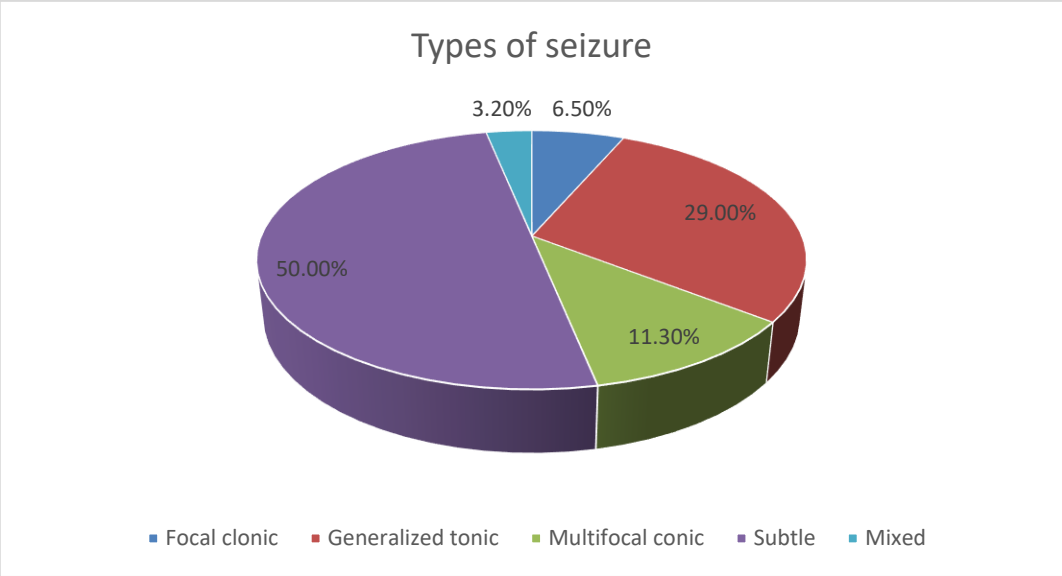


Figure 6: Types of seizure

Table 7: Electrolyte abnormalities among the study cases

Electrolyte abnormalities	No. of cases	Percentage
Yes	29	46.8%
No	33	53.2%
Total	62	100.00%

In this study, electrolyte abnormalities were identified in a significant proportion of neonates presenting with seizures. Out of the 62 cases, 41 neonates (66.10%) exhibited some form of electrolyte disturbance, while 21 cases (33.90%) showed no such abnormalities. This finding suggests that electrolyte imbalance is a common contributing factor in neonatal seizures and reinforces the importance of frequent biochemical assessment of all cases of neonate convulsions.

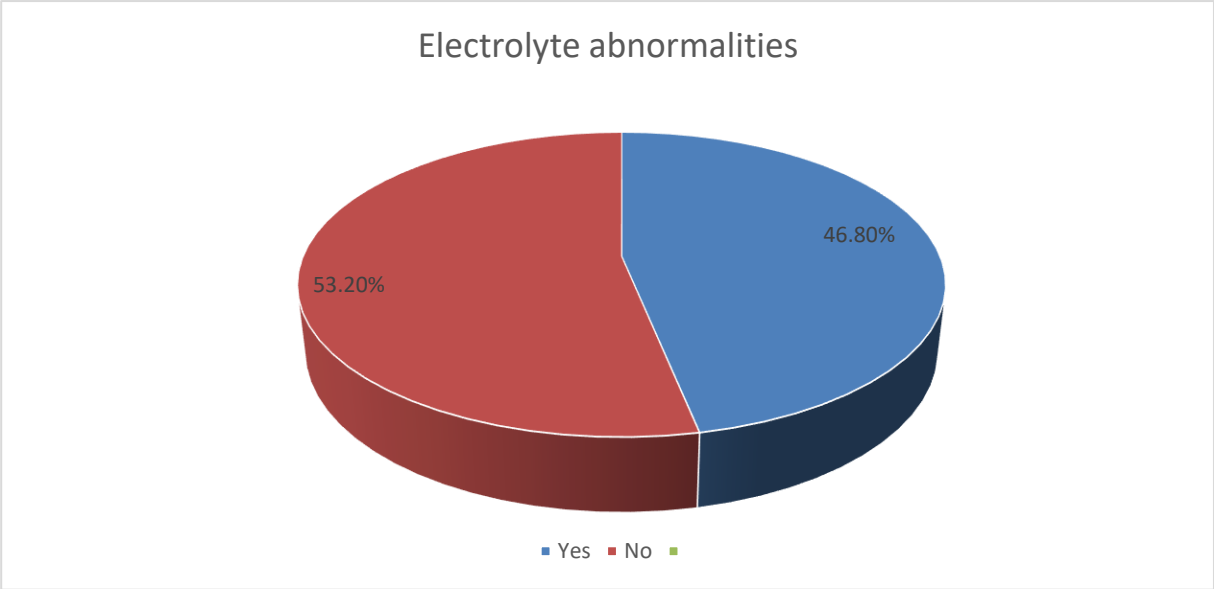


Figure 7 : Electrolyte abnormalities among the study cases

Table 8: Electrolyte abnormalities among the study cases

Electrolyte abnormalities	No. of cases (n=29)	Percentage
Hypocalcaemia	11	37.9%
Hypernatremia	9	31.0%
Hyponatremia	3	10.4%
Hypermagnesemia	2	6.9%
Hypercalcemia	3	10.4%
Hypomagnesemia	1	3.4%

Among the 62 neonates included in the study, 29 (46.8%) were found to have electrolyte abnormalities during seizure episodes. The most common abnormality observed was **hypocalcemia**, present in 11 cases (37.9%), indicating the critical role of calcium in neonatal neuronal stability and seizure threshold regulation. **Hypernatremia** was the second most frequent abnormality, seen in 9 cases (31.0%), followed by **hyponatremia** and **hypercalcemia**, each noted in 3 cases (10.4%). Less frequently, **hypermagnesemia** was seen in total 2 cases (6.9%) and **hypomagnesemia** in a total of 1 case (3.4%). These findings highlight that disturbances in calcium and sodium levels are the most prevalent electrolyte imbalances associated with neonate seizures, underscoring importance of timely biochemical screening and correction in affected neonates.

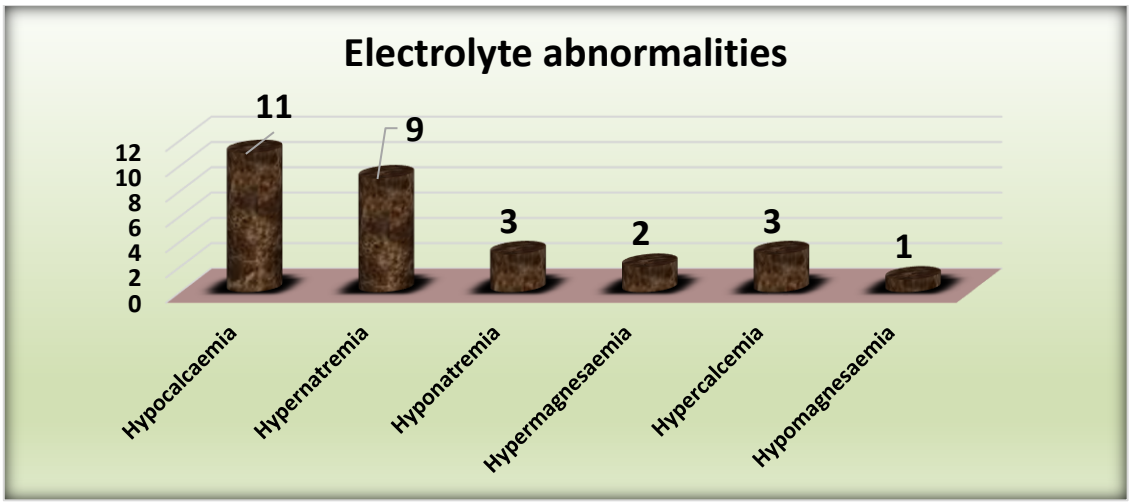


Figure 8: Electrolyte abnormalities among the study cases

Table 9: Types of seizure and Calcium levels

Types of seizure	Hypercalcemia	Hypocalcemia	Normal Calcium	Total
Focal clonic	0	1	3	4
Generalized tonic	2	3	13	18
Multifocal clonic	1	1	5	7
Subtle	0	5	26	31
Mixed	0	1	1	2
Total	3	11	48	62

In this study, hypocalcemia was observed in 11 neonates, most commonly associated with **subtle (5 cases)** and **generalized tonic seizures (3 cases)**. Hypercalcemia was seen in 3 cases, primarily with **generalized tonic** and **multifocal clonic** seizures. Among neonates with normal calcium levels, **subtle seizures** remained the most frequent (26 out of 48). These findings suggest a possible link between **calcium abnormalities** and specific seizure types, particularly **hypocalcemia** with **subtle type and generalized type tonic seizures**.

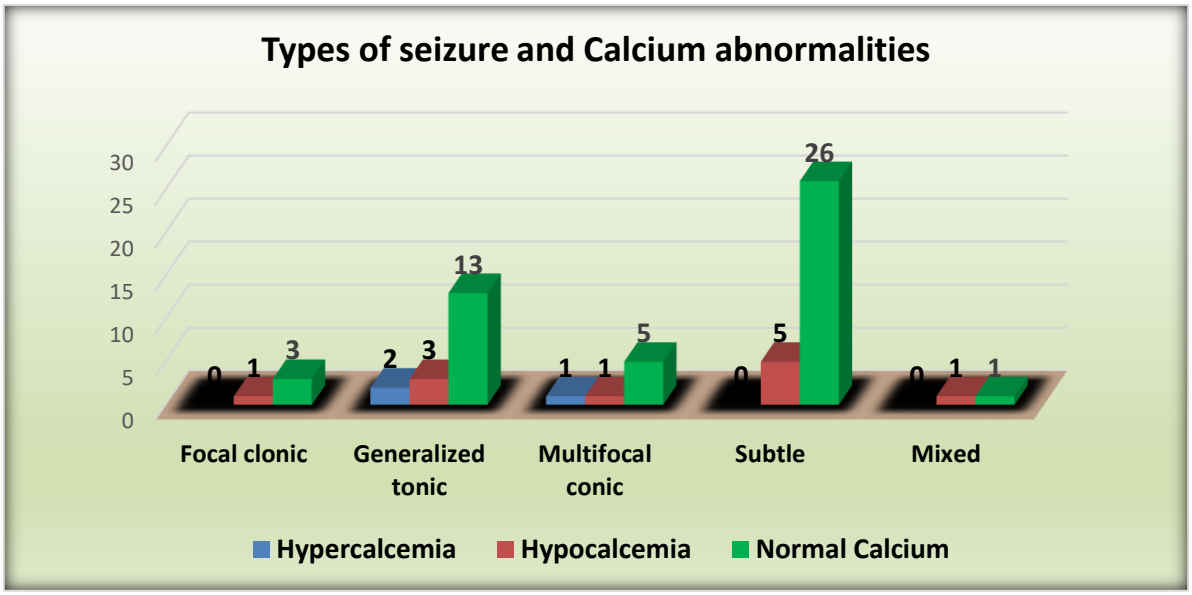


Figure 9: Types of seizure and Calcium levels

Table 10: Types of seizure and Magnesium levels

Types of seizure	Hypermagnesemia	Hypomagnesemia	Normal Magnesium	Total
Focal clonic	0	0	4	4
Generalized tonic	1	0	17	18
Multifocal clonic	0	0	7	7
Subtle	1	1	29	31
Mixed	0	0	2	2
Total	2	1	59	62

In this study, **hypermagnesemia** was observed in **2 neonates**, one each with **generalized tonic** and **subtle seizures**, while **hypomagnesemia** was seen in **1 neonate** with a **subtle seizure**. The vast majority (**59 out of 62 neonates**) had **normal magnesium levels**, among whom **subtle seizures (29 cases)** and **generalized tonic seizures (17 cases)** were most common. These findings indicate that **magnesium abnormalities were rare**, and **subtle seizures** were the most frequent type across all magnesium levels, suggesting a limited but possible role of magnesium imbalance in neonatal seizures.

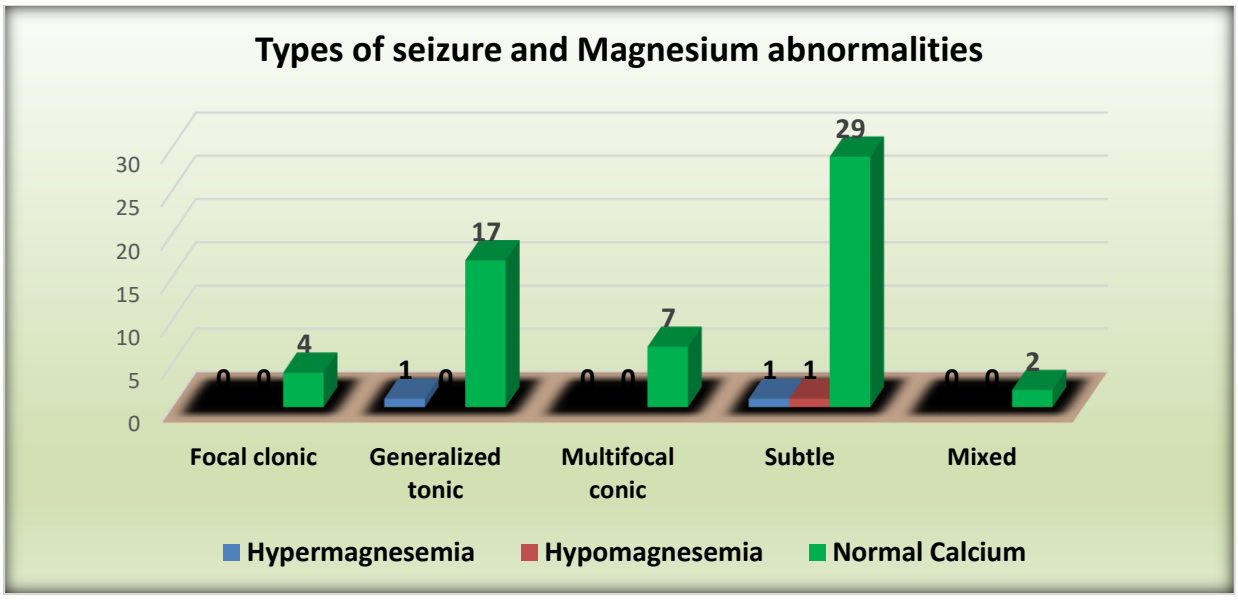


Figure 10: Types of seizure and Magnesium levels

Table 11: Types of seizure and Sodium levels

Types of seizure	Hypernatremia	Hyponatremia	Normal Sodium	Total
Focal clonic	0	0	4	4
Generalized tonic	2	1	15	18
Multifocal clonic	1	0	6	7
Subtle	5	2	24	31
Mixed	1	0	1	2
Total	9	3	50	62

In this present study, **hypernatremia** observed total in **9 neonates**, most commonly associated with **subtle seizures (5 cases)** and **generalized tonic seizures (2 cases). Hyponatremia** was noted in **3 cases**, predominantly seen with **subtle (2 cases)** and **generalized tonic seizures (1 case)**. Among the **50 neonates with normal sodium levels, subtle seizures (24 cases)** and **generalized tonic seizures (15 cases)** were the most frequent. These results suggest that **sodium imbalances**, particularly **hypernatremia,** may have a modest association with **subtle and generalized tonic seizures**, although **subtle seizures** remained the most common across all sodium levels.

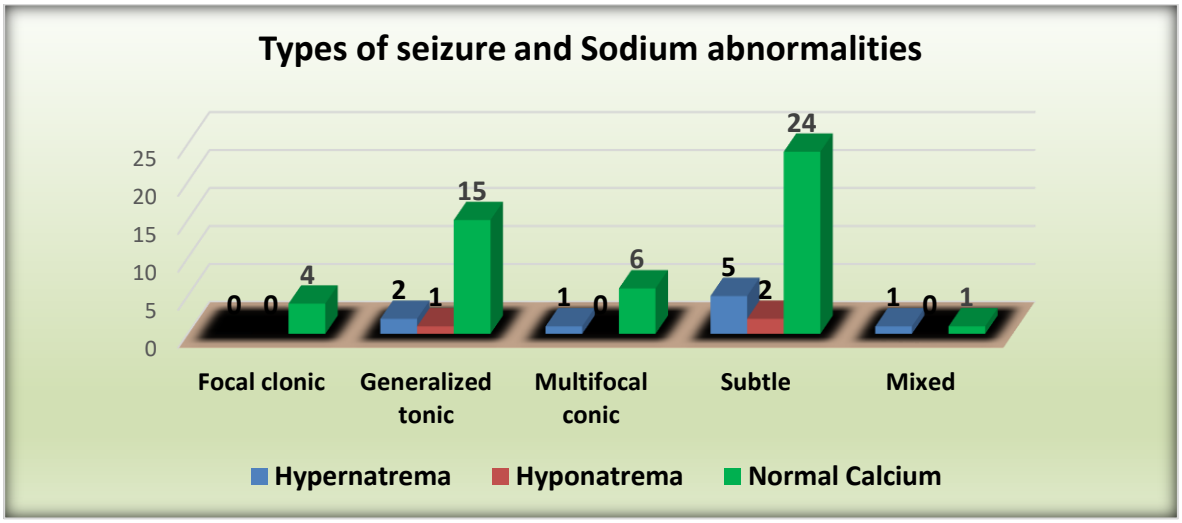


Figure 11: Types of seizure and Sodium levels

Table 12: Electrolyte Abnormalities among seizure cases

Seizure cases	No. of cases	Percentage
Electrolyte Abnormalities among seizure cases	29	46.8%
without Electrolyte Abnormalities among seizure cases	33	53.2%
Total cases	62	100.00%

In this present study involving 62 neonates presenting with complaints of seizures, **electrolyte abnormalities identified in 29 cases (46.8%)**, while **33 cases (53.2%)** had **no detectable electrolyte disturbances**. This suggests that **electrolyte imbalance is a significant contributing factor** in nearly half of all neonatal seizure episodes. Among the abnormalities, disturbances in calcium, sodium, and magnesium levels were most frequently noted.

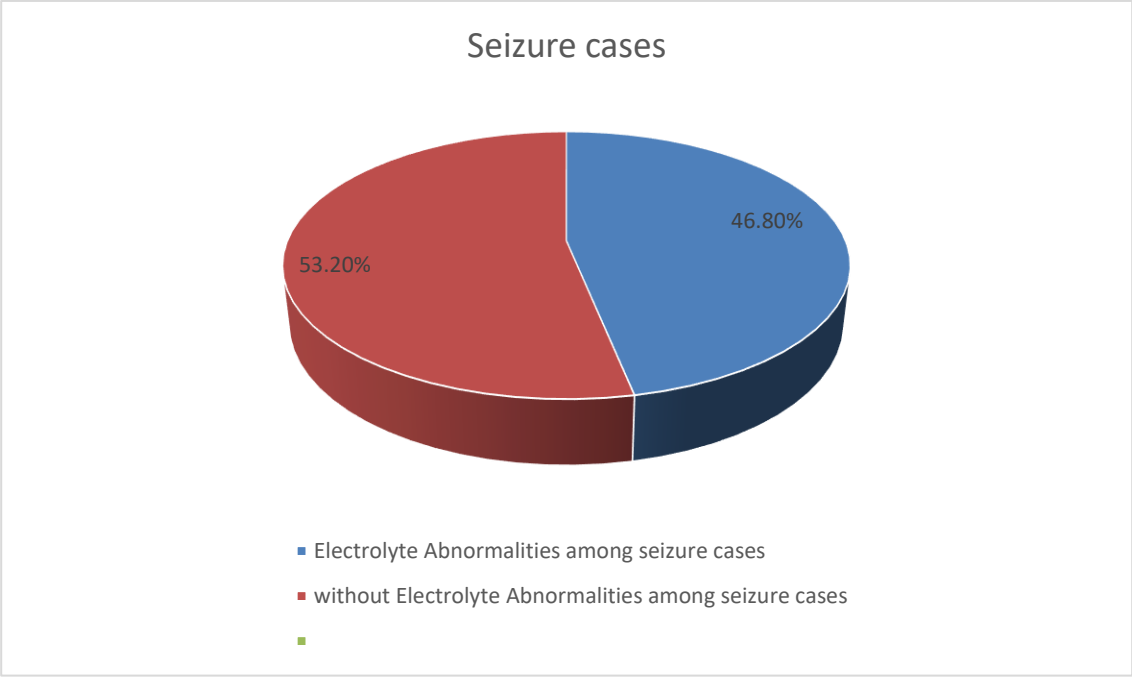


Figure 12: Electrolyte Abnormalities among seizure cases

Table 13: Types of seizure and Electrolyte Abnormalities

Types of seizure	Electrolyte Abnormalities		Total
	Yes	No	
Focal clonic	1	3	4
Generalized tonic	9	9	18
Multifocal conic	3	4	7
Subtle	14	17	31
Mixed	2	0	2
Total	29	33	62

p=0.527 (p>0.05 not significant)

Among the 62 neonates with seizures, **29 cases (46.8%)** were associated with **electrolyte abnormalities**, while **33 cases (53.2%)** had **normal electrolyte levels**. **Subtle seizures** were the most common type overall (31 cases), with **14 of these (45.2%)** showing electrolyte disturbances. **Generalized tonic seizures** were the next most frequent (18 cases), equally distributed between those with and without abnormalities (9 each). **Multifocal clonic seizures** showed electrolyte disturbances in **3 out of 7 cases (42.9%)**, while **focal clonic seizures** had only **1 out of 4 cases (25%)** with an abnormality. Interestingly, **both cases of mixed seizures (100%)** were associated with electrolyte imbalances. These findings indicate that **electrolyte abnormalities occur across all seizure types**, with a relatively higher proportion in generalized, subtle, and mixed forms, highlighting the importance of electrolyte assessment regardless of seizure presentation.

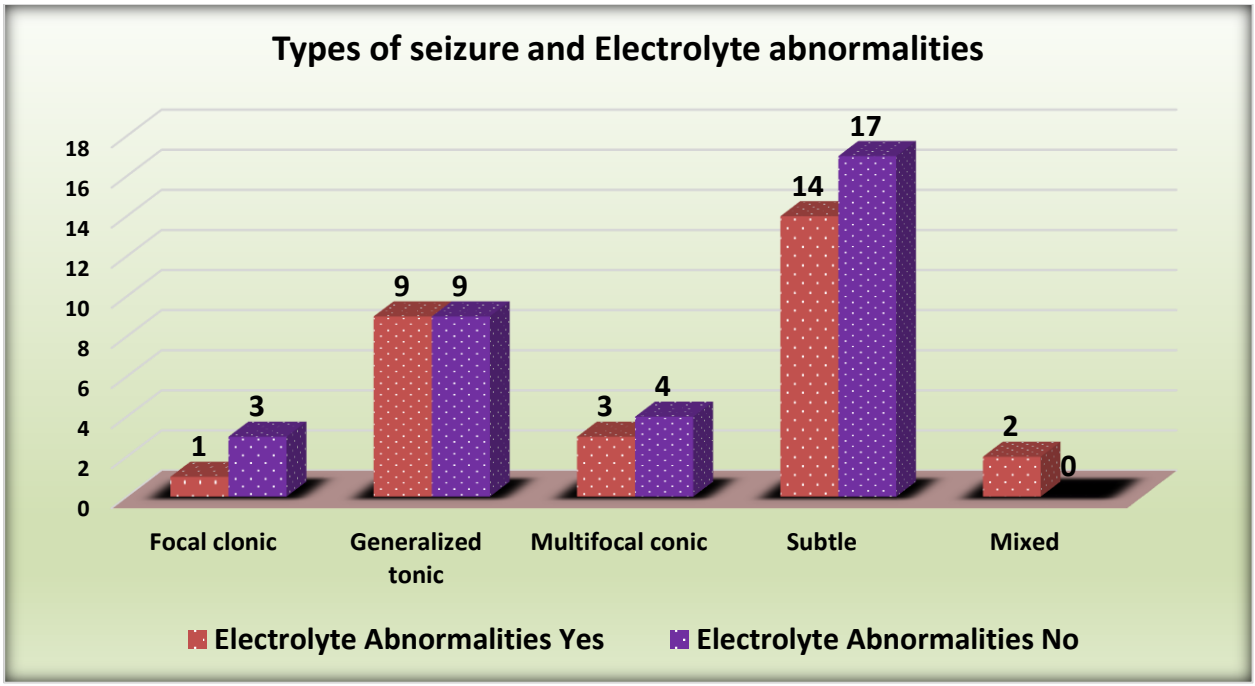


Figure 13: Types of seizure and Electrolyte Abnormalities

DISCUSSION

DISCUSSION

1. GENDER DISTRIBUTION AMONG STUDY PARTICIPANTS

In the current study, 62 neonates presenting with seizures were included, with 26 (41.9%) males and 36 (58.1%) females, showing a slightly higher proportion of female neonates. The female-to-male ratio was approximately 1.38:1.

In contrast, study by Deepak Sharma et al. reports that out of the enrolled neonates, 143 were male and 93 were female. Among those diagnosed with hypocalcemia, 23 (56.1%) were males and 18 (43.9%) were females.[55]

Different results reported in study done by Kyasa Srinivas et al., where about 40 (52.6%) of the participants were in the male categort and 36 (47.4%) were female. [51]

A study by Falsaperla et al. showed contrasting results, with seven male and six female patients.[56]

In contrast, Pasi et al. found that males (48 out of 76) were 1.7 times more numerous than females (28 out of 76). [59]

Abdul Rahim et al. reported contrasting findings, with male participants comprising 39 (55.71%) and female participants accounting for 31 (44.29%).[61]

Mishra et al. observed contrasting results, with males representing 60% (n=60) and females 40% (n=40) of the neonates experiencing convulsions in the hospital.[62]

2. GESTATIONAL AGE AMONG THE STUDY CASES

In this study, out of the 62 neonates who experienced seizures, the majority were term neonates, comprising 45 cases (72.60%), while the remaining 17 cases (27.40%) were preterm

neonates. This pattern indicates that, within this study group, neonate seizures are common in term infants in comparison to preterm infants.

Comparable results were reported by Kyasa Srinivas et al., where 54 (71.1%) of the infants were full-term and 22 (28.9%) were preterm based on gestational age. [51]

Similar findings were observed in study by Pasi et al., when 64 neonates were full-term, 10 were preterm, and 2 were post-term. The mean of gestational age in the neonates was 37.86 ± 2.71 weeks, ranging from 24 to 43 weeks.[59]

Abdul Rahim et al. reported similar findings, with 19 preterm births (27.14%) with 51 term births (72.86%) and no post-term births among their study participants.[61]

Similarity in results were found in study by Mishra et al., where 35% of infants were preterm , 65% were full-term based off on gestation age. [62]

3. DELIVERY TYPE AMONG THE STUDY CASES

Among the 62 neonates included in the study who presented with seizures, 29 (46.80%) were delivered through normal vaginal delivery (NVD), while a slightly higher proportion, 33 neonates (53.20%), were delivered via lower segment cesarean section (LSCS). This distribution indicates that neonate seizures were marginally common in infants delivered through LSCS in this cohort. The mode of delivery can influence neonatal outcomes, especially in cases where LSCS is performed due to distress in fetus or complications that may increase risk of neonatal seizures.

Abdul Rahim et al. reported similar findings, with delivery methods comprising 2.86% by forceps, 48.57% by lower segment cesarean section (LSCS), and 48.57% by normal vaginal delivery. [61]

By contrast, Mishra et al. reported delivery methods as 28% by cesarean section, 24% by operative vaginal delivery, and 48% by routine vaginal delivery.[62]

4. BIRTH WEIGHT (KG) AMONG THE STUDY CASES

Out of the 62 neonates with seizures included in this study, 25 neonates (40.30%) had lowest birth weight (less than 2.5 kg), while 37 neonates (59.70%) had normal birth weight. This indicates that a significant proportion of seizure episodes occurred in neonates with low weight at birth, which is recognized risk factor for various neonate complications.

Similar results observed in study by Kyasa Srinivas et al., with 49 (64.5%) infants having a normal birth weight and 27 (35.5%) classified as low birth weight.[51]

Similar findings reported by Pasi et al., when most neonates, 61 (80.3%), were classified as Appropriate for the Gestational Age (AGA), while 15 ~ (19.7%) were Small for the Gestational Age (SGA). [59]

Similar findings reported by Abdul Rahim et al., when the birth weights ranged from 0.99 kg to 4.16 kg, with a mean of 2.56 kg (95% CI: 2.40 to 2.72). Within study population, 31 newborns had low birth weight (below 2.5 kg), while 39 had normal weight (2.5 kg or above).[61]

Similar findings were observed in study done by Mishra et al., when 68% of neonates were appropriate for the gestational age, 6% were large for the gestational age, and 26% were small for the gestational age. [62]

5. DAY OF ONSET OF NEONATAL SEIZURES AMONG STUDY CASES

In the present study, the onset of seizures occurred most frequently on the **second day of life**, with **23 cases (37.00%)**, followed by **18 cases (29.00%)** on the **first day**. The third and fourth days accounted for **8 cases (12.90%)** and **6 cases (9.70%)**, respectively. Fewer cases

were seen on the fifth and sixth days, with **3 cases (4.90%)** and **1 case (1.60%)** each. Notably, no seizures were recorded on the seventh day. Seizures occurring during the **late neonatal period (8–28 days)** were seen in **3 cases (4.90%)**.

Contrast Findings were noted in a study by Kyasa Srinivas et al, the onset of seizures occurred during First 24 Hours was found in 18(23.7%), during 1- 3 days in 37(48.7%), during 4- 7 days in 15(19.7%) and after 1 week in 6(7.9%). [51]

A study done by Pasi et al. reports that a majority of neonates, 85.5% (n=65), had developed seizures within the initial three days of life. Among these, 63% (n=41) were hyponatremic, with 35 (53.8%) having serum sodium levels between 120 - 133 mmol/L and 6 (9.2%) experiencing severity of hyponatremia. In contrast, only 25% of cases with late-onset seizures were hyponatremic. [59]

Abdul Rahim et al. reported similar findings, noting that 20% patients experience their first seizure within the 24 hours of birth. This followed 31% between 24 and 72 hours (days 1 to 3), 14% between 4 days and 1 week (days 4 to 7) and 7.14% (5 patients) after more than one week (over 7 days).[61]

6. TYPES OF SEIZURE

In the current study, subtle type of seizures were most frequently observed type, accounting to 31 cases in total (50.00%). These are often difficult to detect clinically and may include eye deviation, lip smacking, or apnoea, underscoring the importance of careful observation in neonates with suspected neurological symptoms. Generalized type tonic seizures were the second most common, observable in 18 cases (29.00%), followed by multifocal type clonic seizures in 7 cases total(11.30%). Focal clonic seizures occurred in 4 neonates (6.50%), while mixed types of seizures were the least frequent, with only 2 cases (3.20%).

Comparable findings reported by Kyasa Srinivas et al., with subtle type seizures observed in 42 neonates (55.3%), tonic seizures in 24 neonates (31.6%), and clonic seizures in 10 neonates (13.2%). [51]

Similar findings reported by Pasi et al., when a higher proportion of neonates with subtle type seizures were hyponatremic (64.3%), followed by 54.3% among those with focal seizures. [59]

Similarity in results were observed in study by Abdul Rahim et al., where subtle seizures occurred in 43 infants (61.43%), tonic seizures in 18 infants (25.71%), and clonic seizures in 9 infants (12.86%) within the study population. [61]

In study by Mishra et al., distribution of seizure types included focal clonic in 30% of cases, multifocal clonic in 17%, subtle seizures in 28%, and tonic seizures in 25% of neonates. [62]

Parvin et al. reported similar findings, with tonic seizures observed in 45.1% of cases, subtle seizures in 35.3%, clonic seizures in 15.7%, and mixed seizure types in 3.9% of the neonates. [63]

7. PRESENCE OF ELECTROLYTE ABNORMALITIES AMONG THE STUDY CASES

In this study, electrolyte abnormalities were identified in a significant proportion of neonates presenting with seizures. Out of the 62 cases, 41 neonates (66.10%) exhibited some form of electrolyte disturbance, while 21 cases (33.90%) showed no such abnormalities. This observation indicates that electrolyte disturbances are a frequent underlying cause of neonatal seizures, highlighting the necessity of routine biochemical evaluations in all neonates presenting with convulsions.

Comparable findings were reported by Pasi et al., where metabolic abnormalities were identified in 66 neonates (86.8%). Among them, 11 had isolated metabolic disturbances, while 55 presented with metabolic issues alongside other comorbid conditions. [63]

8. DISTRIBUTION OF ELECTROLYTE ABNORMALITIES AMONG THE STUDY CASES

Among the 62 neonates included in the study, 29 (46.8%) were found to have electrolyte abnormalities during seizure episodes. The most common abnormality observed was **hypocalcemia**, present in 11 cases (37.9%), indicating the critical role of calcium in neonatal neuronal stability and seizure threshold regulation. **Hypernatremia** was the second most frequent abnormality, seen in 9 cases (31.0%), followed by **hyponatremia** and **hypercalcemia**, each noted in 3 cases (10.4%). Less frequently, **hypermagnesemia** was seen among 2 cases (6.9%) and the abnormality **hypomagnesemia** in 1 case (3.4%). These findings highlight that disturbances in calcium and sodium levels are the most prevalent electrolyte imbalances associated with neonate seizures, underscoring importance of timely biochemical screening and correction in affected neonates.

Findings were similar in a study by Kyasa Srinivas et al, distribution of biochemical abnormalities were found to be **hypocalcemia** in 19(25%), hyponatremia in 9(11.8%), hypoglycemia in 26(34.2%), hypomagnesemia in 7(9.2%). [51]

Similar findings were reported by Pasi et al., where hyponatremia and hypocalcemia were frequently observed in cases of multifactorial or mixed seizures, whereas hypoglycemia (noted in 10 cases) was the most prevalent metabolic disturbance in primary metabolic seizures. [59]

Similar observations were made in study by Abdul Rahim et al., involving 70 neonates. Hypoglycemia, identified in 17 infants (24.29%), while 14 (20%) had hypocalcemia. Hyponatremia was seen in 6 cases (8.57%), and hypomagnesemia in 3 (4.29%). Additionally,

hypernatremia was present in 3 neonates (4.29%). Combined electrolyte disturbances were also noted: 2 infants (2.86%) had both hypoglycemia and hypocalcemia, and 1 infant (1.43%) had both hypocalcemia and hypomagnesemia. [61]

9. TYPES OF SEIZURE AND CALCIUM LEVELS

In this study, hypocalcemia was observed in 11 neonates, most commonly associated with **subtle (5 cases)** and **generalized tonic seizures (3 cases)**. Hypercalcemia was seen in 3 cases, primarily with **generalized tonic** and **multifocal clonic** seizures. Among neonates with normal calcium levels, **subtle seizures** remained the most frequent (26 out of 48). These findings suggest a possible link between **calcium abnormalities** and specific seizure types, particularly **hypocalcemia** with **subtle type and generalized type tonic seizures**.

Similarity in results were observed in study by Pasi et al., where hypocalcemia was most frequently identified biochemical abnormality, affecting 49 neonates (64.5%). In contrast, 22 infants (28.9%) had normal serum calcium levels, while 5 cases (6.6%) showed hypercalcemia, with serum calcium levels exceeding 1.23 mmol/L.[59]

10. TYPES OF SEIZURE AND MAGNESIUM LEVELS

In this study, **hypermagnesemia** was observed in **2 neonates**, one each with **generalized tonic** and **subtle seizures**, while **hypomagnesemia** was seen in **1 neonate** with a **subtle seizure**. The vast majority (**59 out of 62 neonates**) had **normal magnesium levels**, among whom **subtle seizures (29 cases)** and **generalized tonic seizures (17 cases)** were most common. These findings indicate that **magnesium abnormalities were rare**, and **subtle seizures** were the most frequent type across all magnesium levels, suggesting a limited but possible role of magnesium imbalance in neonatal seizures.

11. TYPES OF SEIZURE AND SODIUM LEVELS

In present study, **hypernatremia** was observable in total **9 neonates**, most commonly associated with **subtle seizures (5 cases)** and **generalized tonic seizures (2 cases). Hyponatremia** was noted in **3 cases**, predominantly seen with **subtle (2 cases)** and **generalized tonic seizures (1 case)**. Among the **50 neonates with normal sodium levels, subtle seizures (24 cases)** and **generalized tonic seizures (15 cases)** were the most frequent. These results suggest that **sodium imbalances**, particularly **hypernatremia,** may have a modest association with **subtle and generalized tonic seizures**, although **subtle seizures** remained the most common across all sodium levels.

Similar findings as reported by Pasi et al., when a higher proportion of neonates with subtle seizures were hyponatremic (64.3%), followed in number by 54.3% in those with focal seizures. Among neonates with severe hyponatremia, 50% (4 out of 8) died. In contrast, 16.2% (6 out of 37) of those with serum sodium levels between 120 - 133 mmol/L expired, while 96.8% (30 out of 31) of normal sodium level neonates were successfully discharged after receiving treatment. The study finds significant association between hyponatremia and poor outcomes, such as death, in neonatal seizures (p=0.003). [59]

12. ELECTROLYTE ABNORMALITIES AMONG SEIZURE CASES

In present study involving 62 neonates presenting with complaints of seizures, **electrolyte abnormalities were identified in 29 cases (46.8%)**, while **33 cases (53.2%)** had **no detectable electrolyte disturbances**. This suggests that **electrolyte imbalance is a significant contributing factor** in nearly half of all neonatal seizure episodes. Among the abnormalities, disturbances in calcium, sodium, and magnesium levels were most frequently noted.

Similar findings were observed in a study by Kyasa S et al., which reported a highly prevalent biochemical abnormalities among neonates with seizures, along with hypoglycemia affecting 39 infants (43.8%) and hypocalcemia observed in 28 infants (35.4%) as the leading contributors. [51]

13. TYPES OF SEIZURE AND ELECTROLYTE ABNORMALITIES

Among the 62 neonates with seizures, **29 cases (46.8%)** were associated with **electrolyte abnormalities**, while **33 cases (53.2%)** had **normal electrolyte levels**. **Subtle seizures** were the most common type overall (31 cases), with **14 of these (45.2%)** showing electrolyte disturbances. **Generalized tonic seizures** were the next most frequent (18 cases), equally distributed between those with and without abnormalities (9 each). **Multifocal clonic seizures** showed electrolyte disturbances in **3 out of 7 cases (42.9%)**, while **focal clonic seizures** had only **1 out of 4 cases (25%)** with an abnormality. Interestingly, **both cases of mixed seizures (100%)** were associated with electrolyte imbalances. These findings indicate that **electrolyte abnormalities occur across all seizure types**, with a relatively higher proportion in generalized, subtle, and mixed forms, highlighting the importance of electrolyte assessment regardless of seizure presentation.

SUMMARY

SUMMARY

This cross-sectional observational study included 62 neonates who presented with seizures to a tertiary care center in Kolar. The objective was to learn regarding serum electrolyte abnormalities during neonatal seizures and their association with seizure occurrence and characteristics.

Demographics and Clinical Profile:

In the study group, females were slightly more numerous, accounting for 58.1%. The majority of neonates were full-term (72.6%), and over half (53.2%) were delivered through cesarean section. An incidence of Low birth weight (under 2.5 kgs) noted in 40.3% of the infants. Most seizures began within first two days of extrauterine life, with highest occurrence on day 2 (37%). Subtle type seizures were predominant type, seen in 50% of studied cases, followed in number by generalized tonic seizures at 29%.

Electrolyte Abnormalities:

Electrolyte disturbances were identified in 46.8% of neonates with seizures. Hypocalcemia was the most frequent abnormality (37.9%), followed by hypernatremia (31%). Other abnormalities included hypercalcemia, hyponatremia, hypermagnesemia, and hypomagnesemia but were less common.

Correlation Between Seizure Types and Electrolyte Abnormalities:

Although electrolyte abnormalities were present across all seizure types, statistical analysis revealed no significance in association between the seizure type and the presence or absence of electrolyte disturbances ($p = 0.527$). This suggests that electrolyte imbalances occur irrespective of seizure classification.

Additional Findings:

- Calcium abnormalities were more common in generalized tonic and subtle seizures.
- Sodium imbalances (hypernatremia and hyponatremia) were frequently noted in subtle seizures.
- Magnesium abnormalities were rare and not significantly associated with any seizure type.

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CONCLUSION

CONCLUSION

In this study of neonates with seizures, almost half exhibited electrolyte imbalances, with hypocalcemia and hypernatremia being the most prevalent. While these disturbances are commonly linked to neonatal seizures, no significance in relationship was found between the type of seizure and electrolyte abnormalities. This highlights the importance of routine evaluation of serum electrolytes in all neonates with seizures, regardless of seizure classification. Prompt identification and correction of electrolyte disturbances are essential in improving clinical outcome and bring the risk of seizure recurrence down in this vulnerable population.

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ANNEXURE

PATIENT INFORMATION SHEET

A CROSS-SECTIONAL OBSERVATIONAL STUDY ON SERUM ELECTROLYTE ABNORMALITIES DURING AN EPISODE OF NEONATAL SEIZURE AT A TERTIARY CARE CENTRE IN KOLAR

Principal Investigator: DR. DEEPAK MELASANGAM

I, Dr.Deepak Melasangam, Post-graduate student in Department of Paediatrics at Sri Devaraj Urs Medical College, will be conducting a study titled "A CROSS-SECTIONAL OBSERVATIONAL STUDY ON SERUM ELECTROLYTE ABNORMALITIES DURING AN EPISODE OF NEONATAL SEIZURE AT A TERTIARY CARE CENTRE IN KOLAR", for my dissertation under the guidance of Dr.Krishnappa , Professor in the Department of Paediatrics. The participants of this study include neonates presenting with seizures, including both intramural and extramural neonates. Neonates will be monitored for electrolyte abnormalities during seizure episodes. The potentiometric method will be used to estimate serum sodium and potassium levels. The Formazan dye method will be used for estimating serum magnesium levels. The ARSENAZO-3 method will be used to measure the levels of serum calcium. You will not be paid any financial compensation for the participation of your child in this research project. The financial expenditure if required for tests will be taken care of by principal investigator.

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

Name of the Principal Investigator – Dr. Deepak Melasangam

Contact number: 9966198015

Date-

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಕೋಲಾರದ ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ ನವಜಾತ ಶಿಶುಗಳ ರೋಗಗ್ರಸ್ತವಾಗುವಿಕೆಗಳ ಸಂಚಿಕೆಯಲ್ಲಿ ಸೀರಮ್ ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಅಸಹಜತೆಗಳ ಕುರಿತು ಅಡ್ಡ-ವಿಭಾಗೀಯ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಿಆರ್. ದೀಪಕ್ ಮೇಳಸಂಗಮ್

ನಾನು ಡಾ.ದೀಪಕ್ ಮೇಳಸಂಗಮ್, ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ಪೀಡಿಯಾಟ್ರಿಕ್ಸ್ ವಿಭಾಗದ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ, "ಇತ್ತೀಚಿನ ದಿನಗಳಲ್ಲಿ ಸೀರಮ್ ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಅಸಹಜತೆಗಳ ಕುರಿತು ಒಂದು ಅಡ್ಡ-ವಿಭಾಗೀಯ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇನೆ , ಮಕ್ಕಳ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕರಾದ ಡಾ.ಕೃಷ್ಣಪ್ಪ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನನ್ನ ಪ್ರಬಂಧಕ್ಕಾಗಿ. ಈ ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವವರು ಇಂಟ್ರಾಮುರಲ್ ಮತ್ತು ಎಕ್ಸ್ಟ್ರಾಮುರಲ್ ನವಜಾತ ಶಿಶುಗಳನ್ನು ಒಳಗೊಂಡಂತೆ ರೋಗಗ್ರಸ್ತವಾಗುವಿಕೆಗಳೊಂದಿಗೆ ಪ್ರಸ್ತುತಪಡಿಸುವ ನವಜಾತ ಶಿಶುಗಳನ್ನು ಒಳಗೊಂಡಿರುತ್ತಾರೆ. ರೋಗಗ್ರಸ್ತವಾಗುವಿಕೆ ಸಂಚಿಕೆಗಳ ಸಮಯದಲ್ಲಿ ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಅಸಹಜತೆಗಳಿಗಾಗಿ ನವಜಾತ ಶಿಶುಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ. ಪೊಟೆನ್ಟಿಯೊಮೆಟ್ರಿಕ್ ವಿಧಾನವನ್ನು ಸೀರಮ್ ಸೋಡಿಯಂ ಮತ್ತು ಪೊಟ್ಯಾಸಿಯಮ್ ಮಟ್ಟವನ್ನು ಅಂದಾಜು ಮಾಡಲು ಬಳಸಲಾಗುತ್ತದೆ. ಸೀರಮ್ ಮೆಗ್ನೀಸಿಯಮ್ ಮಟ್ಟವನ್ನು ಅಂದಾಜು ಮಾಡಲು ಫಾರ್ಮಾಜಾನ್ ಡೈ ವಿಧಾನವನ್ನು ಬಳಸಲಾಗುತ್ತದೆ. ARSENAZO-3 ವಿಧಾನವನ್ನು ಸೀರಮ್ ಕ್ಯಾಲ್ಸಿಯಂ ಮಟ್ಟವನ್ನು ಅಳೆಯಲು ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪರಿಹಾರವನ್ನು ಪಾವತಿಸಲಾಗುವುದಿಲ್ಲ. ಪರೀಕ್ಷೆಗಳಿಗೆ ಅಗತ್ಯವಿದ್ದಲ್ಲಿ ಹಣಕಾಸಿನ ವೆಚ್ಚವನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಗಳು ನೋಡಿಕೊಳ್ಳುತ್ತಾರೆ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಈ ಸಂಸ್ಥೆಯಿಂದ ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ಒಪ್ಪಿಗೆ ನೀಡಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಮಗುವನ್ನು ಅಧ್ಯಯನದಿಂದ ಹಿಂಪಡೆಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಕಾಳಜಿಗೆ ನಿಮ್ಮನ್ನು ಪೂರ್ವಾಗ್ರಹ ಮಾಡುವುದಿಲ್ಲ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು : ಡಿಆರ್. ದೀಪಕ್ ಮೇಳಸಂಗಮ್

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ :9966198015

ದಿನಾಂಕ-

INFORMED CONSENT FORM

Date:

I, Mr/Mrs _____, have been explained in my own vernacular language that my child will be included in "A CROSS-SECTIONAL OBSERVATIONAL STUDY ON SERUM ELECTROLYTE ABNORMALITIES DURING AN EPISODE OF NEONATAL SEIZURE AT A TERTIARY CARE CENTRE IN KOLAR", hereby I give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters .The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow my child as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

Signature & Name of Patient Attendant/Mother

Signature & Name of Researcher/Doctor

Relation with patient

Witness:

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ದಿನಾಂಕ:

ನಾನು, ಶ್ರೀ/ಶ್ರೀಮತಿ _____, ನನ್ನ ಮಗುವನ್ನು "ಕೋಲಾರ್‌ನ ತೃತೀಯ ಆರೈಕೆ ಕೇಂದ್ರದಲ್ಲಿ ನವಜಾತ ವಶಪಡಿಸಿಕೊಳ್ಳುವಿಕೆಯ ಪ್ರಸಂಗದ ಸಮಯದಲ್ಲಿ ಸೀರಮ್ ವಿದ್ಯುದ್ವಿಚ್ ೇದ್ಯ ವೈಪರೀತ್ಯಗಳ ಬಗ್ಗೆ ಅಡ್ಡ-ವಿಭಾಗದ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುವುದು ಎಂದು ನನ್ನ ಸ್ವಂತ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ" ಹೆಮಟೊಲಾಜಿಕಲ್ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯವಾದ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಿ. ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ನನ್ನ ಮಗುವನ್ನು ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಂತೆ ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

ರೋಗಿಯ ಸಹಿ ಮತ್ತು ಹೆಸರು
ಪರಿಚಾರಕ/ತಾಯಿ

ಸಹಿ ಮತ್ತು ಹೆಸರು
ಸಂಶೋಧಕ/ವೈದ್ಯ

ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ
ಸಾಕ್ಷಿ :

PROFORMA

Name: IP NO.:
Age: DOB:
Sex: DOA:
Address: DOD:
Informant:

CHIEF COMPLAINTS:

History of Seizures since:

Started on _____day of birth
Duration of Seizures:
Type of Seizures a) Subtle b) Generalised Tonic
c) Focal clonic d) Multifocal clonic
e) Myoclonic f) Mixed
How often seizures occur :
Consciousness during attack and in between seizures

History of Fever Yes/No
History of Refusal of Feeds Yes/No
History of Excessive Cry Yes/No
History of Vomiting Yes/No

FAMILY HISTORY:

History of consanguinity :
History of birth order of child :
History of Similar complaints in other siblings:
History of Neurological disorder in other sibling:

ANTENATAL HISTORY:

Age of mother:
Parity of mother:

History of present illness:

Maternal High Risk factors:
Duration of Labour : 1st Stage 2nd Stage 3rd Stage

BIRTH HISTORY:

Place of delivery Hospital or Home
Gestation: Term/ Preterm/ Post-term
Foetal growth: AFD/SFD/LFD
LBW: Yes/No
Type of delivery: Normal/Caesarean/ forceps/ Assisted
Apgar Score 1 minute 5 minute 10th minute
Cried immediately after birth/ Any Resuscitation done
Meconium stained/ Foul smelling liquor/ PROM Yes/No
Umbilical Catheterization/ bleeding Yes/No
History of Birth injury: Yes/No
History of Local anaesthesia injection Yes/No

POST NATAL HISTORY:

Activity of the baby:
Breast feeding started after:
History of difficulty in feeding:
History of Jaundice- onset/ duration/ treatment received:

GENERAL PHYSICAL EXAMINATION:

General appearance and posture of the baby:

Any external congenital anomaly Yes/No

Head -Size and Shape
-Hair distribution
-Anterior fontanelle
-Posterior fontanelle
-Caput/ Cephal hematoma

Eyes

Cyanosis Yes/No

Icterus Yes/No

Dehydration Yes/No

Skin: Petechiae/ Purpura/ Pustule/ Vesicles/ hypo or hyperpigmented spots/ hemangioma

Umbilicus: Redness/ Induration/ Discharge/ Normal/ if fallen on which day.

Anthropometry

-Head Circumference
-Chest circumference
-Length
-US:LS Ratio
-Birth Weight gms.:
-Present weight:

Vital Signs:

-Heart rate: /min
-Respiratory rate: /min
-Blood pressure: mm Hg
-Capillary Refilling time: sec
-Temperature:

Gestational age : _____ Weeks.

SYSTEMIC EXAMINATION

1.Central Nervous System:

- Higher mental functions
- Cranial nerves
- Motor examination: Tone
  Power
  Involuntary movements
- Reflexes - Superficial: Abdominal
  Cremasteric
  Deep: Biceps
  Triceps
  Knee
  Ankle
- Neonatal Reflexes: Moro's
  Sucking/rooting
  Palmar grasp
  Tonic neck reflex

2.Skull/ Spine:

3.Abdominal Examination:

4.Respiratory System
5.Cardiovascular System:

Working Diagnosis:

Day of seizure: Etiology:

INVESTIGATIONS:
Haemoglobin -
PCV -
Total count -
Differential Count:

- Neutrophils –
- Lymphocytes -
- Monocytes -
- Eosinophils –
- Basophils -

Peripheral smear study -

CRP:

Blood culture -

- Organism:
- Sensitivity:

Blood Glucose:

Serum electrolytes:

- Sodium
- Potassium
- Calcium
- Magnesium

CSF Analysis:

- Color
- Protein
- Sugar
- Total count
- Culture sensitivity

Chest X-ray:

USG Cranium:

EEG:

MRI BRAIN:

Final diagnosis:

Treatment given:
Remarks:

MASTER CHART

Name	Gender	Gestational age	Delivery type	Birth weight	Day of onset	Electrolyte abnormality	Seizure type	Fever	Refusal of feeds	Vomiting	Family history	Antenatal history	Birth history	Postnatal history	Anatomical abnormalities	Anthropometry	Vitals	CVS	CNS	P/A	RS	CBC	CRP	CSF Analysis	NSG	GRBS	Culture
B/O Bhagyalakshmi	Male	Preterm	NVD	Low	1	Hypocalcemia	Focal clonic	No	No	No	No	Not significant	PROM	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Bhavani	Male	Term	LSCS	Normal	3	Hypernatremia	Generalised tonic	Yes	No	No	No	Significant	PROM	Not significant	No	Normal	Normal	ASD	Normal	No abnormalities	Respiratory distress	Anemia	Negative	Normal	Normal	Normal	No growth
B/O Najma	Female	Preterm	NVD	Low	2	Hypocalcemia	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Anemia	Negative	Normal	Normal	Normal	No growth
B/O Bharathi	Female	Term	NVD	Normal	1	Hypernatremia	Subtle	Yes	Yes	Yes	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Suman	Male	Term	LSCS	Normal	2	Hypermagnesemia	Generalised tonic	No	No	No	No	Significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Varsha	Female	Preterm	NVD	Low	2	Hypocalcemia	Multifocal clonic	No	No	No	No	Not significant	PROM	Not significant	No	Normal	Unstable	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Swathi	Female	Term	LSCS	Normal	1	No	Generalised tonic	No	No	No	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
B/O Banumathi	Female	Term	LSCS	Normal	3	Hypercalcemia	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Microcephaly	Normal	Tiny PDA	Poor suck	No abnormalities	No distress	Thrombocytopenia	24 mg/dl	Normal	PVL	Normal	Klebsiella
B/O Asma	Female	Preterm	NVD	Low	1	Hypocalcemia	Generalised tonic	No	No	No	No	Significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Mary	Male	Term	LSCS	Normal	2	No	Subtle	No	Yes	Yes	No	Not significant	PROM	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	Respiratory distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
B/O Saroja	Female	Term	NVD	Low	1	Hypermagnesemia	Subtle	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Anuradha	Male	Term	LSCS	Normal	2	Hypercalcemia	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	12 mg/dl	Normal	PVL	Normal	Enterobacter
B/O Damini	Male	Preterm	NVD	Low	2	Hypocalcemia	Generalised tonic	No	No	No	No	Significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Swetha	Male	Term	LSCS	Normal	2	No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Lakshmi	Female	Term	LSCS	Low	1	No	Generalised tonic	No	No	Yes	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
B/O Jamuna	Female	Term	NVD	Normal	3	Hypocalcemia	Subtle	No	Yes	No	No	Not significant	Not significant	Not significant	No	Microcephaly	Normal	Normal	Poor suck	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Mohammadi	Male	Preterm	NVD	Low	2	No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	VSD	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Shalini	Female	Term	LSCS	Normal	1	No	Multifocal clonic	No	No	No	No	Not significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	Respiratory distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Gunavathi	Female	Term	LSCS	Normal	3	Hypocalcemia	Subtle	No	No	Yes	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Rani	Male	Preterm	NVD	Normal	2	Hyponatremia	Generalised tonic	No	No	No	No	Significant	PROM	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Kouser	Female	Term	LSCS	Normal	1	No	Generalised tonic	No	Yes	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Poor suck	No abnormalities	No distress	Normal	Negative	Normal	PVL	Normal	No growth
B/O Rajeshwari	Female	Term	LSCS	Normal	4	Hypocalcemia	Subtle	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Aishwarya	Male	Preterm	NVD	Low	3	No	Subtle	No	No	No	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Ramya	Female	Term	LSCS	Normal	2	No	Generalised tonic	No	No	No	No	Not significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
B/O Balu	Male	Term	LSCS	Normal	1	Hypocalcemia	Subtle	No	No	Yes	No	Not significant	Not significant	Not significant	No	Normal	Normal	ASD	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Sarika	Female	Term	LSCS	Normal	4	No	Generalised tonic	No	No	No	No	Significant	PROM	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
B/O Junaid	Male	Preterm	NVD	Low	2	Hypocalcemia	Subtle	No	Yes	No	No	Not significant	Not significant	Jaundice	No	Microcephaly	Unstable	Normal	Normal	No abnormalities	Respiratory distress		Negative	Normal	Normal	Normal	No growth
B/O Sangamithra	Female	Term	LSCS	Normal	2	No	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	Respiratory distress	Thrombocytopenia	48 mg/dl	Normal	PVL	Normal	Pseudomonas
B/O Karuna	Female	Term	LSCS	Normal	3	Hypercalcemia	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Anemia	Negative	Normal	Normal	Normal	No growth
B/O Roja	Male	Term	NVD	Low	1	No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	12 mg/dl	Normal	PVL	Normal	No growth
B/O Saritha	Female	Term	LSCS	Normal	2	Hypernatremia	Generalised tonic	Yes	No	No	No	Not significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth

Electrolyte abnormality	Seizure type	Fever	Refusal of feeds	Vomiting	Family history	Antenatal history	Birth history	Postnatal history	Anatomical abnormalities	Anthropometry	Vitals	CVS	CNS	P/A	RS	CBC	CRP	CSF Analysis	NSG	GRBS	Culture
No	Subtle	No	No	No	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Subtle	No	Yes	Yes	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
Hypernatremia	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	VSD	Poor suck	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
No	Focal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
Hypernatremia	Subtle	Yes	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	MSAF	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
Hypocalcemia	Mixed	No	No	Yes	No	Significant	PROM	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	24 mg/dl	Normal	PVL	Normal	E.Coli
No	Generalised tonic	No	Yes	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
Hyponatremia	Subtle	No	No	No	No	Not significant	PROM	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
Hypernatremia	Subtle	No	No	No	No	Significant	PROM	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	ASD	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
Hypomagnesemia	Subtle	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Unstable	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	12 mg/dl	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	Yes	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Anemia	Negative	Normal	Normal	Normal	No growth
Hypernatremia	Subtle	Yes	Yes	No	No	Not significant	MSAF	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
No	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Thrombocytopenia	24 mg/dl	Normal	PVL	Hypoglycemia	Klebsiella
No	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Subtle	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
Hypernatremia	Subtle	Yes	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Tiny PDA	Poor suck	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Subtle	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Multifocal clonic	No	No	Yes	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Multifocal clonic	No	No	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
Hyponatremia	Subtle	No	Yes	No	No	Not significant	Not significant	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Generalised tonic	No	No	No	No	Not significant	Not significant	Not significant	No	Microcephaly	Normal	Small PFO	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
No	Generalised tonic	No	No	No	No	Not significant	MSAF	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Hypoglycemia	No growth
Hypernatremia	Mixed	No	No	Yes	No	Not significant	Not significant	Jaundice	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth
No	Subtle	No	No	No	No	Significant	PROM	Not significant	No	Normal	Normal	Normal	Normal	No abnormalities	No distress	Normal	Negative	Normal	Normal	Normal	No growth