Clinical Profile of Neonatal Hypoglycaemia in a Tertiary Care Centre in Mahabubnagar, Telangana

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ABSTRACT

BACKGROUND

Hypoglycaemia is one of the most common metabolic problems seen in neonatal intensive care units (NICU). Most cases of neonatal hypoglycaemia are transient and respond readily to treatment and are associated with excellent prognosis. Development of clinical signs and symptoms may be a late sign of hypoglycaemia. Persistent hypoglycaemia may result in possible neurologic sequelae. The purpose of this study was to assess the clinical pattern of hypoglycaemia in neonates admitted in special newborn care unit in Government General hospital, Mahabubnagar, Telangana and to also assess the influence of gestational age, birth weight, various comorbid conditions on blood glucose levels.

METHODS

This is an observational hospital-based study done in Government General Hospital, Mahabubnagar from June 2020 to May 2021. Neonates with hypoglycaemia (blood glucose < 45 mg/dl) at the time of admission are included in our study. Blood glucose values were monitored 2nd hourly on 1st day and 6th hourly thereafter. Following the detection of hypoglycaemia, the neonates were treated as per institutional protocol. Clinical features, laboratory parameters are studied and analysed.

RESULTS

Among the 99 neonates studied, 68 (68.7 %) were males and 31 (31.3 %) females; Term babies were 75 (75.7 %) and pre term babies were 24 (24.2 %). Low birth weight newborns (51.5 %) were more affected with hypoglycaemia compared to normal weight newborns (38.4 %). Among the 99 neonates studied, 96.9 % were treated and discharged. Average duration of stay was around 05 to 07 days.

CONCLUSIONS

Hypoglycaemia is most common condition in neonates. Routine screening should be done to all newborns at the time of admission. Timely intervention reduces long term neurological sequelae. Neonates presenting with dull activity, refusal to feed, vomiting, jitteriness, seizures must routinely undergo regular glucose monitoring. As the study shows, most hypoglycaemic neonates presented with those symptoms. Among the various comorbidities, hypoglycaemia occurred more in birth asphyxia and respiratory distress syndrome. So, it should be made mandatory to do glucose monitoring in these cases. Glucose monitoring should be made as a common screening method to prevent morbidity and mortality in neonatal intensive care units.

KEYWORDS

Hypoglycaemia, Pre-Term, Term, Low Birth Weight, Special New Born Care Unit, Small for Gestational Age, Large for Gestational Age

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DOI: 10.18410/jebmh/2021/589

How to Cite This Article:
Patta N, Kumar MJ, Sirazuddin M, et al. Clinical profile of neonatal hypoglycaemia in a tertiary care centre in Mahabubnagar, Telangana. J Evid Based Med Healthc 2021;8(35):3241-3246. DOI: 10.18410/jebmh/2021/589

Submission 25-06-2021,
Peer Review 03-07-2021,
Acceptance 05-08-2021,
Published 30-08-2021.

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BACKGROUND

One of the commonest metabolic problems encountered in neonates is hypoglycaemia. Overall incidence of neonatal hypoglycaemia is 1 to 5 per 1,000 live births and in high-risk neonates, it is up to 30 % and differs in various parts of the world. In preterm and intrauterine growth retarded neonates, the incidence is 15 % and 8 % in large for-gestational age infants particularly in infants of diabetic mothers.[1]

During intrauterine life, the main source of glucose to foetus is through placenta. When the umbilical cord is cut at birth, this supply of maternal glucose ceases abruptly. Hence, the neonate must maintain its own supply of glucose during periods of fasting and when feedings are interspersed intermittently.[2]

During the transition from continuous transplacental supply of glucose to the intermittent oral supply postnatally, episodes of hypoglycaemia can occur. The developmental immaturity of adaptive mechanisms like gluconeogenesis, glycogenolysis and ketogenesis may further accentuate the occurrence of hypoglycaemia. The main concern is the neurological injury due to hypoglycemia.[2]

Immediately after birth there occurs hormonal and metabolic adaptations which may ensure adequate glucose supply to neonate. In preterm and small for gestational age (SGA), this hormonal-metabolic adaptation will not take place as desired. Result being hypoglycaemia, which is more common in first seventy-two hours of life.[1]

Immediately after birth there occurs endocrine changes with a decline in plasma insulin levels and increase of catecholamine and glucagon. There will be endogenous production of glucose through gluconeogenesis, till exogenous nutritional supply started. Various adaptive mechanisms will occur such as hepatic glycogenolysis, lipolysis, and beta oxidation of fatty acids. The end result is production of ketone body and lactate, which are substrate for gluconeogenesis.

The hepatic glucose production in term new born is 4 - 6 mg/kg/min. The hepatic glycogen stores in newborn will last for 10 - 12 hours in immediate newborn period. Alanine, pyruvate, glycerol, and lactate are essential for gluconeogenesis. Lactate contributes to 30 % of gluconeogenesis whereas alanine, glycerol contributes 5 - 10 % in gluconeogenesis. Small for gestational age newborns have low glucose stores, suggesting inadequate gluconeogenesis. Rate of glucose generation and lipolysis are ineffective in small for gestational age (SGA) infants but gluconeogenesis from glycerol is efficient.[3]

Neurological development and intellectual functions are more affected due to hypoglycaemia in varying extent. Spasticity, ataxia and seizure disorder may occur in recurrent or prolonged hypoglycaemia. The extent of neurological injury depends on severity, recurrence, aetiology of hypoglycaemia in neonates.

Hypoglycaemia results in reduced adenosine tri phosphate (ATP) and creatine phosphate. This will impair the normal transmembrane concentration of gradient recovery for sodium and calcium ions. The net result is excessive calcium influx into the cells and the cellular phosphatase and protease are activated. This alters the mitochondrial metabolism and results in the formation of free radicals contributing to necrosis of neurons. This hypoglycaemic brain injury predominantly affects parieto-occipital regions causing cognitive, sensory, psychomotor, and behavioural deficits in children.[3]

In the neonatal period, euglycemia is essential for proper neurological development. So, with routine screening and early detection of hypoglycaemia we may prevent lot of neurologic sequalae due to hypoglycaemia. Various factors influence newborn blood glucose concentrations even in healthy term newborns, such as birth weight, gestational age, presence or absence of disease, perinatal complications, mode of delivery and feeding behaviour.[4]

In immediate neonatal period, gluconeogenesis is very important. Neonatal hypoglycaemia occurs due to impaired gluconeogenesis, brought about by excess insulin production, altered counter-regulatory hormone production. SGA (weigh less than 10th percentile) babies, large-for-gestational-age (LGA; weight more than 90th percentile) babies, IDMs (Infants of diabetic mothers) and preterm babies are more prone to this. Some doubt has been raised as to whether LGA babies who are not IDMs are truly at risk.[5]

Pre-term babies had three times increased risk of hypoglycaemia compared to term babies. Small-for-dates (SFDs) and large-for-dates (LFDs) babies were at increased risk of manifesting hypoglycaemia (7 and 10 times, respectively) as compared to the appropriate-for-dates (AFDs) babies. Approximately two-thirds of the hypoglycaemic babies will have one or more risk factors including birth asphyxia, diabetic mothers, respiratory distress and septicaemia. The most common symptom observed was lethargy, followed by jitteriness, respiratory abnormalities, hypotonia and seizures.[6]

The type of screening method we are using, the sample site, the operator technic, the associated comorbid condition will influence the blood glucose level in neonates. In special new born care units, we used glucose reagent strips most of the times to determine blood glucose level. There was a lot of variation between glucometer reading and laboratory report. So, the glucometer readings are of unproven reliability to determine hypoglycaemia in newborns. So, glucometer readings should be used only for screening purpose only, not for confirmed diagnosis. If there is hypoglycaemia with glucometer, a blood sample should be sent to laboratory for confirmation by glucose oxidase or glucose electrode method. Treatment should be started based on screening test report rather than waiting for laboratory report.

Symptoms frequently observed in hypoglycaemia are tremors, jitteriness, or irritability, seizures, coma, lethargy, apathy, poor feeding, vomiting, apnea, weak or high-pitched cry, cyanosis. Some infants may develop one or more symptoms, few neonates may not develop any symptoms. Clinical confirmation requires the following criteria for hypoglycaemia: serum glucose level < 45 at the time of symptoms and resolution of symptoms with correction of hypoglycaemia.

In hypoglycaemia, the main aim was to achieve euglycemia and to prevent further episodes of

hypoglycaemia. The various ways to manage the hypoglycaemia depend on aetiology causing the hypoglycaemia and clinical condition of the baby. Babies who have mild hypoglycaemia may be managed with enteral feeding either as breast feeding or formula feed. The feeding should be repeated every 2 hours and blood glucose monitoring should be done. If repeat blood glucose level is > 45, second hourly feeds continued while monitoring 6th hourly blood glucose for 24 hours. Breast-feeding is preferred oral feed if the child is able to suck. Expressed milk may be given if the child is not able to suck. Enteral feeding should be the first priority.

Few babies may need intravenous therapy. Babies who are not able to tolerate enteral feeds, who do not maintain euglycemia with feeds, who have very low blood glucose level may need IV therapy. IV cannula must be secured under aseptic conditions. Initial therapy is by giving bolus of 2 ml/kg 10 % dextrose over one minute and recheck blood glucose after 30 mins. If the baby is stable and able to feed, then shift to enteral feeds.

If there is persistent hypoglycaemia or recurrence of hypoglycaemia, shift to continuing therapy. Infusion of glucose at a rate of 6 to 8 mg of glucose/kg/minute. Recheck blood glucose levels hourly in between to see any recurrence. If there is no recurrence, then shift to enteral feeds and the glucose infusion tapered over time while maintaining euglycemia.

Some babies may need 12 to 15 mg of dextrose/kg/minute. The concentration of glucose and the rate of infusion are increased as required to achieve euglycemia. These higher concentrations must be infused in central line. Taper glucose infusion and concentration, monitoring blood glucose levels and wean off intravenous therapy, while shifting to enteral feeds. Few babies may not maintain euglycemia despite of above measures. In these babies, hydrocortisone may be given 10 mg/kg/day in two divided doses if the baby remains hypoglycaemic despite receiving glucose > 12 mg/kg/minute. In few cases, glucagon, diazoxide, epinephrine may be considered depending on condition of the baby and etiological causes.

For persistent hypoglycaemia, corticosteroids such as hydrocortisone at 5 - 15 mg/kg/day or prednisone at 2 mg/ kg/day is the preferred second line treatment, which reduces peripheral utilisation of blood glucose. Glucagon, produced by the α cells in the pancreas, is a counter regulatory hormone and initiates gluconeogenesis and glycogenolysis during hypoglycaemia, is helpful in raising blood glucose when infant has adequate glycogen stores.[7]

In persistent hypoglycaemia not responding to glucose infusion or corticosteroids, glucagon is administered at 30 mcg/kg or 300 mcg/kg/min infusion in neonates with adequate glycogen stores. This promotes glycogenolysis and gluconeogenesis. Glucagon is especially helpful for term infants, infant of diabetic mothers when short-term treatment is desirable like during transport of critically ill infants.

In persistent hypoglycaemia, somatostatin is used when the hypoglycaemia is not responding to the above treatment. It inhibits insulin and growth hormone release. Octreotide, a long-acting analogue of endogenously occurring somatostatin which acts directly on the voltage gated calcium channels has an inhibitory effect on insulin release is used. It is used as a constant infusion at 3 – 10 mcg/kg/day, however, it is not currently FDA approved and there are concerns that it impedes neonatal growth.[7]

Comorbid conditions like prematurity, hypoxic-ischemic organ damage, sepsis may influence the prognosis and outcome of neonates with hypoglycaemia. Each of these can contribute to neurological damage in varying extent.

In hypoglycaemia, the cerebral electrical activity is decreased, with resultant membrane breakdown and release of free fatty acids, and the amino acid metabolism gets altered. Result will be excess production of glutamate. Glutamate being one of excitatory neurotransmitter in central nervous system, plays vital role in hypoglycaemic neurological injury.

Birth Asphyxia

In birth asphyxia, hypoglycaemia may lead to increased neurological damage in comparison to birth asphyxia alone. In hypoglycaemia, high energy phosphates will be reduced, and extracellular glutamate concentrations will be increased, there will be depletion of high energy phosphates, increased extra cellular glutamate concentrations, and inotropic glutamate receptors will get activated, resulting in increased intracellular sodium and calcium. During hypoxia, anaerobic glycolysis occurs which lead to decline of glucose in brain. Thus, both hypoglycaemia and hypoxia act synergistically resulting in neurological injury.

Severe hypoglycaemia in the newborn is associated with selective neuronal necrosis in multiple brain regions, including the superficial cortex, dentate gyrus, hippocampus, and caudate-putamen.[8]

Preterm Neonates

Hypoglycaemia and its associated complications are more common in preterm neonates because of limited glycogen and fat stores, inadequate gluconeogenesis. Preterm neonates have high metabolic demands because of relatively large brain size, and they will not be able to mount a counter-regulatory response to hypoglycaemia.

Preterm neonates have inadequate stores of glycogen and adipose tissues. Several enzymes involved in gluconeogenesis like phosphoenolpyruvate carboxykinase (PEPCK), glucose-6-phosphatase, fructose-1, 6-diphosphatase, and pyruvate carboxylase are in very low levels further depleting gluconeogenesis. So, the intra uterine growth restriction (IUGR) babies and preterm neonates are more prone to hypoglycaemia in immediate neonatal period.[7]

In SGA infants, there is a strong association between hypoglycaemia and small head circumference measured at 12 months, 18 months, and 5 years corrected age. The glial proliferation occurs during third trimester of intra uterine life and continues after birth. In preterm neonates, hypoglycaemia delays astrocyte proliferation. Besides occipital cortex, the sensorimotor cortex, thalamus, midbrain, brainstem and cerebellar vermis are more prone to hypoglycemicinjury.[7]

Neonatal hypoglycaemia is broadly divided into transient and persistent types, mainly based on the duration of hypoglycaemia.

Transient Hypoglycaemia - Types

Early Transitional - Adaptive Hypoglycaemia

During birth, few adaptation mechanisms occur like (↓glycogenolysis and gluconeogenesis and ↑insulin secretion). Examples of this type include infants of diabetic mothers, infants with hypothermia, asphyxiated babies and preterm babies.

Secondary Hypoglycaemia

Disorders of central nervous system, such as perinatal asphyxia, intracranial haemorrhage, and congenital anomalies may result in secondary hypoglycaemia. Other comorbid conditions which may result in hypoglycaemia include sepsis, cold injury, and congenital heart disease.

Classic Transient Neonatal Hypoglycaemia

This type of hypoglycaemia is typically seen in small for gestational age babies. Small for gestational age babies have inadequate stores of energy and defective gluconeogenesis. Most of these infants are symptomatic.

Persistent Hypoglycaemia

Persistent hypoglycaemia is defined as persistent low plasma glucose concentrations beyond the first 7 days of life. Hyperinsulinism is the most common cause of persistent hypoglycaemia in neonates. There will be inappropriate secretion of insulin in the presence of low plasma glucose. It is the most common cause of persistent hypoglycaemia in the neonates. Other less common causes are defects in gluconeogenesis, glycogenolysis, galactosemia, and hereditary fructose intolerance.[9]

Objective

The objective of this study is to assess the clinical pattern of hypoglycaemia in neonates admitted in special newborn care unit in Government General hospital, Mahabubnagar, Telangana and to also assess the influence of gestational age, birth weight and various comorbid conditions on blood glucose levels.

METHODS

This is a descriptive, observational, record-based study. Hospital records of all newborns, who were hospitalized in special newborn care unit of Government General Hospital, Mahabubnagar from June 2020 to May 2021 were reviewed. Neonates with hypoglycaemia (blood glucose < 45 mg/dl) at the time of admission were included in our study. Blood glucose values were monitored 2nd hourly on 1st day and 6th hourly thereafter. Following the detection of hypoglycaemia, the neonates were treated as per institutional protocol. Clinical features, laboratory parameters were studied and analysed.

Inclusion Criteria

All newborns asymptomatic or symptomatic, whose blood glucose level was < 45 mg/dl at the time of presentation at special newborn care unit.

Exclusion Criteria

- Babies above 28 days of life.
- Euglycemic newborns at admission.
- Neonates presented with other comorbid conditions without hypoglycaemia.
- Neonates with congenital malformations and endocrine deficiencies were excluded from the study.

The study was conducted over 12 months involving 99 neonates who were admitted with hypoglycaemia at special newborn care unit. Blood glucose values were monitored 2nd hourly on 1st day and 6th hourly thereafter. Blood glucose value of less than 45 mg/dl was defined as hypoglycemia.[10]

Blood glucose monitoring was done using rapid glucose test strip mounted on glucometer device. The right or left heel of neonate was disinfected using cotton wool and methylated spirit. The disinfected site was allowed to air dry. Sterile lancet was used to prick heel. Glucose test strip mounted on glucometer was used to make contact with blood from puncture site and the displayed blood sugar noted accordingly. Following the detection of hypoglycaemia, the neonates were treated as per institutional protocol.

Statistical Analysis

Data was compiled and tabulated by using standard appropriate statistical technique, which includes numbers and percentages.

RESULTS

In our study, total number of babies were 99. Among them, maximum number of hypoglycaemic neonates were term babies 75 (75.75 %), preterm constitute 24 (24.2 %). Majority were males 68 (68.7 %) remaining 31 (31.3 %) constituted female babies. (Table 1)

Gestational Age	Male	Female	Total
Pre-Term	14	10	24 (24.2 %)
Term	54	21	75 (75.75 %)
Total	**68**	**31**	**99**

Table 1. Gestational Age and Sex Distribution of Hypoglycaemic Neonates

Clinical Features	Number of Patients
Vomiting, refusal to feed	22
Lethargy, jitteriness, dull activity	34
Seizures	10
Fever	07
Respiratory distress	13
Neonatal jaundice	02
Birth Asphyxia	10

Table 2. Clinical Profile of Cases

In our study, 22 babies presented with refusal to feed and vomiting, 34 babies presented with lethargy, jitteriness and dull activity. 10 babies presented with active seizures. 07 babies with fever had hypoglycaemia at presentation.

10 babies with birth asphyxia and 13 babies with respiratory distress syndrome had hypoglycaemia. 01 case of confirmed Covid positive baby and 02 cases of neonatal jaundice had hypoglycaemia at admission.

Birth Weight	Number of Neonates
Large for age neonates (> 3.5 kg)	09 (9 %)
Normal birth weight (2.5 - 3.5 kg)	38 (38.4 %)
Low birth weight (< 2.5 kg)	51 (51.5 %)
Very low birth weight (< 1.5 kg)	01 (1 %)

Table 3. Distribution According to Birth Weight

In our study, 38 (38.4 %) babies had normal birth weight and 51 (51.5 %) babies had low birth weight, very low birth weight – 1 (1 %), large for gestational age – 09 (9 %). (Table 4)

Cured or relieved of symptoms	96
Deaths (due to sepsis etc)	03
Average duration of admission	05 to 07 days

Table 4. Outcomes- Observed

Among the 99 babies studied, 96.9 % babies were treated and discharged. Average duration of admission was around 05 to 07 days. 3 % mortality noted (with other comorbidities such as very low birth weight, sepsis rather than hypoglycaemia).

DISCUSSION

Glucose is essential for the newborn brain. Prolonged hypoglycaemia may cause long term neurological damage. Various hormones play key role in glucose maintenance. Blood glucose is lowered by insulin, whereas glucagon, epinephrine and cortisol are the hormones that maintain euglycemia. Pancreas, adrenal glands and brain regulate the release of these hormones.

In all the neonates, immediately after birth there will be initial fall in glucose over the first 2 hours of life. Over the next 96 hours, the glucose level gradually rises. Compared to formula-fed neonates, breastfed neonates will have slightly lower glucose and higher ketone bodies.

In present study of neonatal hypoglycaemia, among 99 neonates, majority were males (68.68 %). Similar results were observed in study of Manjunatha Babu R, et al. and study by Patel P, GogoiPR.[11,12]

In our study maximum number of neonates were term 75 (75.75 %), similar to study of Abolodje Efe, Ozhe Sunday et al. In contrast, study of Manjunatha Babu R et al. where majority of hypoglycaemic neonates were preterm.[13,11]

In our study, 34 babies presented with dull activity, jitteriness and lethargy; 22 babies presented with vomiting and refusal to feed; 10 babies presented with seizures. Ten birth asphyxia babies, 13 respiratory distress babies, 07 fever babies presented with hypoglycaemia at the time of presentation. In study of PL Kashyap et al. the common symptoms were poor feeding (66.67 %), lethargy (19.05 %), jitteriness (4.17 %), irritability (2.5 %), hypotonia (0.83 %), and cyanosis (0.83 %). Among the comorbid conditions, birth asphyxia was present in 8 (6.67 %), sepsis in 9 (7.5 %), polycythaemia in 3 (2.5 %) and shock in 1 (0.83 %).[14]

In this study, majority of the babies were low birth weight (LBW) (51.5) % followed by neonates with normal birth weight (38.4 %) where as in study of VSSY Murty et al. there is a very low incidence of hypoglycaemia in LBW newborn babies. In study of A. De et al. similar finding of low birthweight babies prone to develop hypoglycaemia was found.[15,16.]

Among the 99 babies studied, 96.9 % babies were treated and discharged. The 3.0 % mortality was due to various complications like sepsis etc. Average duration of admission was around 05 to 07 days.

CONCLUSIONS

Hypoglycaemia is most common condition in neonates. Routine screening should be done to all newborns at the time of admission. Timely intervention reduces long term neurological sequelae.

In our study, term neonates, and low birth weight neonates got more affected with hypoglycaemia. So, term and low birth weight neonates are more prone to hypoglycaemia.

Neonates presenting with dull activity, refusal to feed, vomiting, jitteriness, seizures must routinely undergo regular glucose monitoring. As in my study, most hypoglycaemic neonates presented with those symptoms.

Among the various comorbidities, hypoglycaemia was more common in birth asphyxia and respiratory distress. So, it should be made mandatory to do glucose monitoring in these cases. Glucose monitoring should be made as a common screening method to prevent morbidity and mortality.

Data sharing statement provided by the authors is available with the full text of this article at jebmh.com.
Financial or other competing interests: None.
Disclosure forms provided by the authors are available with the full text of this article at jebmh.com.
My sincere thanks to all my colleagues for their great effort. And my special thanks to our nursing staff for their kind cooperation. Special thanks to our Head of Department, Paediatrics for encouraging to do the study.

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