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Original Research Article

Prevention of neonatal hypoglycaemia with oral dextrose gel among high-risk neonates born in tertiary care centre

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ABSTRACT

Background: Neonatal hypoglycemia, a significant cause of neurodevelopmental impairment, affects approximately 25% of neonates, particularly those at high risk, such as preterm, small for gestational age (SGA), large for gestational age (LGA), and infants of diabetic mothers (IDM). These infants may require intravenous dextrose therapy if blood glucose levels do not normalize with feeding alone. This study aimed to assess the effectiveness of oral 40% dextrose gel in preventing hypoglycemia, improving neurodevelopmental outcomes, and reducing hospital admissions and promoting early breastfeeding initiation.
Methods: This prospective study included neonates at risk for hypoglycemia, including preterm infants, SGA, LGA, and IDM, delivered at Gulbarga institute of medical sciences over 3 months. Neonates with endocrine disorders or sepsis were excluded. Each neonate in the study received 200 mg/kg (0.5 ml/kg) of oral dextrose gel, administered buccally. Capillary blood glucose levels were monitored every 6 hours for 72 hours.
Results: A total of 52 neonates were randomized into two groups: Group A (n=26) received oral dextrose gel, while group B (n=26) did not. In group A, 7.6% developed asymptomatic hypoglycemia, with 3.8% requiring intravenous dextrose. In group B, 26% had asymptomatic hypoglycemia, and 15% required intravenous dextrose. The differences in hypoglycemia incidence and intravenous dextrose use were not statistically significant (p=0.125 and p=0.323, respectively). Similarly, there was no significant difference in hospital stay duration (p=0.574).
Conclusions: Oral 40% dextrose gel did not significantly reduce hypoglycemia incidence or intravenous dextrose use compared to breastfeeding alone, nor did it affect the length of hospital stays.

Keywords: Oral dextrose gel, Neonatal hypoglycemia, Infant of diabetic mother

INTRODUCTION

Neonatal hypoglycemia is one of the most common metabolic disturbances in the neonatal period. 25% of neonates are at risk for developing neonatal hypoglycemia.[1] The causes of neonatal hypoglycemia are multifactorial, involving a variety of maternal, fetal, and neonatal factors. Some of the primary contributors to neonatal hypoglycemia include prematurity, where the infant's metabolic systems are not fully developed, and maternal conditions such as diabetes, which can lead to altered glucose regulation in the newborn. Additionally, intra-uterine growth restriction (IUGR) and congenital metabolic disorders, such as inborn errors of metabolism and endocrine-related issues like hyperinsulinism, further complicate glucose homeostasis in affected neonates. In healthy term neonates, there is typically a period of transient hypoglycemia during the initial few hours of life, with the lowest glucose levels occurring around the second hour after birth. This transient hypoglycemia is part of the normal physiological adaptation as the newborn shifts from a continuous maternal glucose

supply in utero to independent glucose production in the extrauterine environment. In most cases, this form of hypoglycemia is asymptomatic and self-resolving, requiring no specific medical intervention. However, the risk of significant hypoglycemia is notably higher in certain groups of neonates. These include preterm infants, especially those born before 35 weeks of gestational age, IDM who are often exposed to high levels of maternal glucose in utero, and infants who are either SGA or LGA. Preterm infants are particularly vulnerable due to immature metabolic and hormonal regulation, while IDM and LGA infants often experience hyperinsulinemia, which drives a rapid drop in blood glucose levels after birth.

Even asymptomatic episodes of hypoglycemia in at-risk neonates have been associated with long-term developmental consequences.[2] Studies have shown that untreated or recurrent hypoglycemia can lead to neurosensory impairment, including visual, motor, and cognitive deficits that manifest in later years.[3,4] In preterm neonates, repeated episodes of hypoglycemia have been linked to lower scores on the Bayley Scales of Infant and Toddler Development, which assess cognitive, motor, and language skills. Developmental delays and other cognitive impairments can significantly impact the quality of life and require long-term follow-up and intervention.

The management of neonatal hypoglycemia of both the Indian academy of pediatrics (IAP) and the American academy of pediatrics (AAP) guidelines emphasize breastfeeding or feeding with expressed breast milk and intravenous dextrose administration. IV dextrose treatment can lead to prolonged hospital stays and may delay the initiation of exclusive breastfeeding, which is critical for both mother-infant bonding and optimal nutritional outcomes. Prolonged hospital stays also contribute to increased healthcare costs and place additional stress on both the family and the healthcare system.

In recent years, there has been growing interest in the use of 40% oral dextrose gel as a simpler and less invasive alternative to IV dextrose for the management of hypoglycemia in at-risk neonates. Oral dextrose gel is a readily available, low-cost option that can be administered immediately after birth to neonates who are identified as being at risk for hypoglycemia, such as those born preterm, SGA, LGA, or to diabetic mothers. The gel is applied inside the infant's cheek (buccal mucosa), allowing for rapid absorption into the bloodstream. After administration of the gel, breastfeeding is initiated to support continued glucose regulation.

The aim of this study is to analyze whether the use of 40% oral dextrose gel in at-risk neonates is effective in reducing the incidence of neonatal hypoglycemia, minimizing the need for intravenous dextrose, and shortening the duration of hospital stay.

METHODS

We conducted a randomised controlled trial of 52 at-risk neonates (LGA, SGA, IDM, preterms <35 weeks of GA) delivered at Gulbarga institute of medical sciences. The trial is registered with clinical trial registry India - CTRI/2024/03/064726. These neonates were divided into 2 groups (Group A and B). In group A (n=26), the at-risk neonates were given 40% oral dextrose gel after birth and initiated breastfeeding, group B (n=26), at-risk neonates were initiated breastfeeding after birth without giving oral dextrose gel.

The study was conducted in duration of 3 months from July 2024-September 2024.The inclusion criteria of this study were neonates who were born preterm (<35 weeks), LGA, SGA, IDM, IUGR. We excluded neonates with sepsis and endocrine causes of hypoglycemia.

Group A neonates were administered a dose of 200 mg/kg (equivalent to 0.5 ml/kg) of 40% oral dextrose gel, which was carefully delivered into the buccal cavity using a 2 ml syringe. To monitor their glucose levels, capillary blood glucose was checked at 6-hour intervals using a bedside glucometer. Additionally, random blood sugar measurements were taken once daily throughout the first 72 hours of life to ensure comprehensive monitoring and assessment of the neonates blood glucose stability during this period.

Blood glucose levels of group B neonates without oral dextrose gel were also followed up 6th hourly using bedside glucometer. Since the cut-off for defining hypoglycemia is highly controversial and lack of consensus at present, we considered<45 mg/dL as hypoglycemia. Primary outcome is the incidence of hypoglycemia and the secondary outcome is the duration of hospital stay. Data analysis was performed using IBM SPSS windows version 26.

Categorical datas were compared between groups by using Chi-square test/Fischer's extract test. Statistical significance was determined as p=0.05. Convenient sampling was taken as we were unable to predict the number of at risk neonates.

RESULTS

Fifty two at-risk neonates were recruited between July 2024-September 2024. They were randomized into two groups, group A (n=26) and B (n=26) with matching baseline maternal and neonatal characteristics (Table 1). The Group A neonates received 40% oral dextrose gel after birth and initiated breastfeeding while the group B neonates were not given oral dextrose gel. Among group B neonates, 1 neonate developed sepsis was excluded from the study (Figure 1). The blood sugar levels were monitored in both the groups every 6th hourly using bedside glucometer with the intention to treat the analysis.

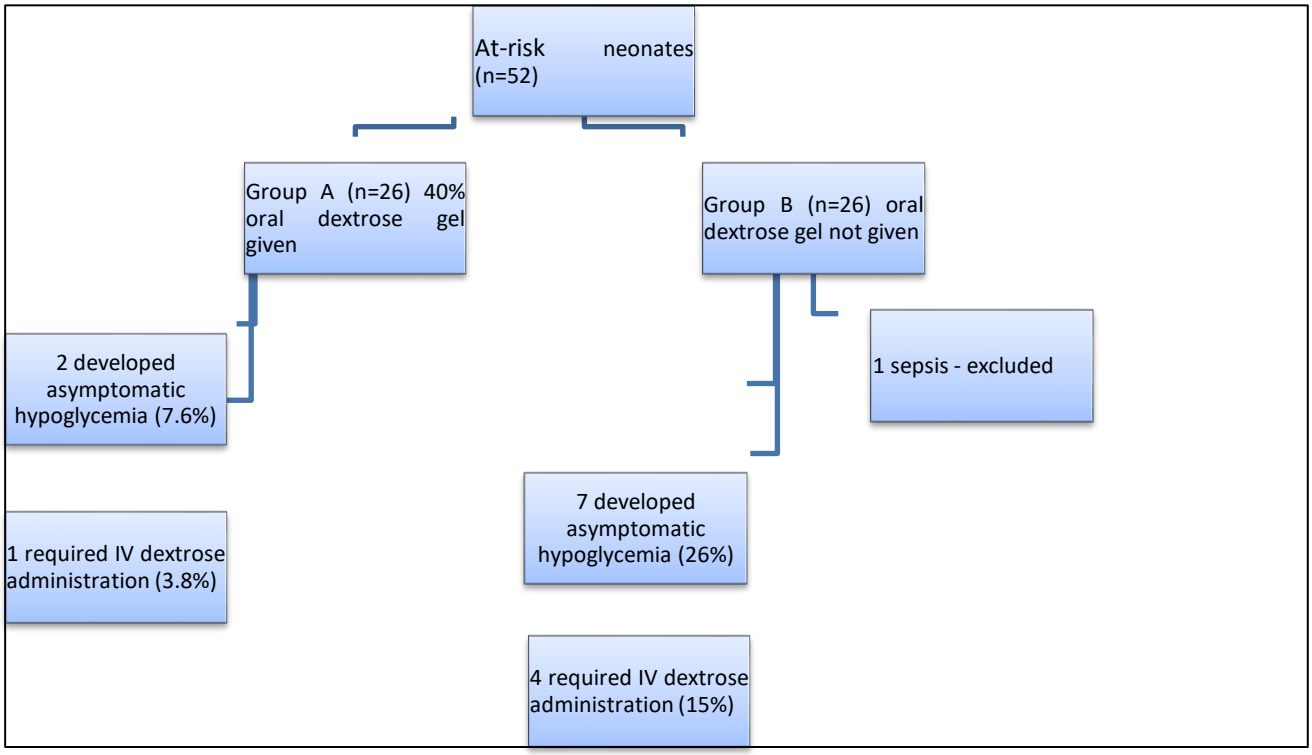

Figure 1: Flowchart.

Table 1: Maternal and neonatal baseline characteristics.

Maternal co-morbidities	Group A, (n=26)	Group B, (n=26)
Gestational hypertension	10	12
Anemia	6	8
Pre-eclampsia	4	3
Gestational diabetes	4	3
Overt DM	1	0
LSCS delivery	17	20
Birth weight (average)	2.2±0.8	2.8±1.2
Gestational age (in weeks)	35±1.4	36±0.6
LGA	4	3
SGA	6	8
IUGR	7	8
Preterms (<35 weeks)	9	7
IDM	2	1

Outcome

Among the neonates in group A (n=26) who received 40% oral dextrose gel, 2 (7.6%) developed asymptomatic hypoglycemia, with 1 (3.8%) requiring intravenous dextrose administration. In group B, which consisted of 25 neonates (excluding one with sepsis), 7 (26%) developed asymptomatic hypoglycemia, and 4 (15%) required intravenous dextrose administration. The p value for the difference in the incidence of asymptomatic hypoglycemia between group A and B was 0.125, indicating no statistically significant difference between the groups in the occurrence of hypoglycemia. Similarly, the p value for the difference in the requirement for intravenous dextrose administration between 2 groups was 0.323, suggesting that there is no statistically significant difference in need for intravenous dextrose between 2 groups. These findings imply that the use of oral dextrose gel did not result in a significant reduction in either incidence of hypoglycemia/need for intravenous treatment when compared to control group (Table 2).

The secondary outcome in terms of prolonged hospital stay (>1 week) in group A was 1 out of 26 i. e., 3.8% and in group B was 3 i. e., 12%. The p value for difference in prolonged hospital stay between group A (1 out of 26) and group B (3 out of 25) is 0.574. This indicates that there is no statistically significant difference in the rates of prolonged hospital stay between 2 groups.

Table 2: Primary and secondary outcomes.

Variables	Group A With oral dextrose gel, (n=26)	Group B Without oral dextrose gel, (n=25)	P value
Incidence of hypoglycemia	2 (7.6%)	7 (28%)	0.125
IV dextrose administration	1 (3.8%)	4 (15%)	0.323
Prolonged duration of hospital stays	1 (3.8%)	2 (12%)	0.574

DISCUSSION

This randomized controlled trial aimed to evaluate the efficacy of 40% oral dextrose gel in reducing the incidence of hypoglycemia, minimizing the need for intravenous dextrose, and shortening hospital stays in at-risk neonates. The study's findings revealed no statistically significant differences between neonates treated with oral dextrose gel (Group A) and those managed with breastfeeding alone (Group B) in terms of hypoglycemia incidence, intravenous dextrose administration, or hospital stay duration. These results are consistent with some earlier studies but differ from others in their assessment of the effectiveness of prophylactic oral dextrose gel.

For instance, a pivotal study by Harris et al reported that oral dextrose gel reduced the need for IV dextrose, although the effectiveness varied across different neonatal populations, particularly in preterm and low birth weight infants.[1] Similarly, Dawson et al found that prophylactic oral dextrose gel significantly reduced the incidence of hypoglycemia in at-risk neonates, particularly those with birth weights below the 10th percentile.[5] These findings highlight that the impact of oral dextrose gel may be more pronounced in specific subgroups of neonates, such as those with SGA or preterm infants. However, our study did not observe a statistically significant reduction in the incidence of hypoglycemia (p=0.125), which could be attributed to various factors, including the diversity in gestational ages and birth weights within our sample population.

One potential explanation for the lack of significant findings in our study could be the relatively small sample size, which may have lacked sufficient power to detect meaningful differences. Bremner et al emphasized that larger trials with more homogenous cohorts are needed to better understand the effects of prophylactic dextrose gel.[6] Moreover, the study's short follow-up period of 72 hours may not have captured the long-term effects of the intervention, as the benefits of early glucose supplementation could extend beyond the immediate neonatal period, influencing later neurodevelopmental outcomes.

Furthermore, the threshold used to define hypoglycemia in our study (<45 mg/dL) is still a subject of debate. Brosnan et al discussed that defining hypoglycemia strictly by blood glucose levels may not fully reflect the clinical impact of low glucose on neonates, especially in cases where hypoglycemia is asymptomatic.[7] In our cohort, while oral dextrose gel was administered, some neonates still experienced transient asymptomatic hypoglycemia, which suggests that factors beyond glucose supplementation, such as the neonate's ability to metabolize glucose effectively and their overall energy balance, may play a role in the preventing hypoglycemia.

The absence of a significant reduction in hospital stays between the two groups in our study (p=0.574) is also consistent with findings from McKinlay et al who observed no substantial difference in hospital stay durations between infants treated with oral dextrose gel and those managed with routine care.[8] This could be related to the complexity of neonatal care, where many factors, such as the neonate's general health, feeding capacity, and the presence of other comorbidities, contribute to the duration of hospital stays.

The transition from intrauterine to extrauterine life is particularly challenging for preterm and low-birth-weight infants, who often struggle with glucose regulation. As Froehlich et al noted, these infants are at a higher risk of persistent hypoglycemia due to their limited glycogen stores and immature metabolic pathways.[9] While oral dextrose gel has been shown to provide a temporary glucose boost, it may not address the underlying metabolic challenges faced by these vulnerable populations. Miller et al found that combining glucose gel with more intensive nutritional support, such as early and frequent breastfeeding, may offer better outcomes for these high-risk infants.[10]

Our study suggests that oral dextrose gel, while potentially helpful in some cases, may not be a comprehensive solution for preventing hypoglycemia in all at-risk neonates. The lack of significant benefit in both hypoglycemia reduction and hospital stay duration highlights the need for further research. Future studies should focus on larger, multi-center trials with longer follow-up periods, homogeneous populations, and more defined glucose thresholds to better assess the long-term benefits of prophylactic glucose supplementation.

In conclusion, although oral dextrose gel has shown promise in some studies, our findings indicate that it did not significantly reduce the incidence of hypoglycemia or the need for intravenous dextrose treatment compared to breastfeeding alone in our cohort. Given the complexity of neonatal glucose regulation, additional interventions beyond glucose gel may be necessary to optimize outcomes for at-risk neonates.

Clinical implications

Despite the lack of statistical significance, the lower proportion of neonates requiring intravenous dextrose in the oral dextrose gel group (3.8%) compared to the control group (15%) suggests that oral dextrose gel may have a clinical benefit in reducing the need for more invasive treatments. While not definitive, this finding may encourage clinicians to consider oral dextrose gel as an adjunct to breastfeeding in the management of at-risk neonates, particularly in settings where intravenous access is challenging or invasive interventions are undesirable.

Further research with larger sample sizes and longer follow-up periods is needed to evaluate the long-term effects of oral dextrose gel on neonatal outcomes, including neurodevelopmental status and the risk of recurrent hypoglycemia. Additionally, studies comparing different dosing regimens, timing of administration, and combination therapies (e.g., oral dextrose gel plus enhanced breastfeeding support) could provide a more comprehensive understanding of the most effective strategies for managing neonatal hypoglycemia.

Limitations

The primary limitation of this study is its small sample size, which may limit the generalizability of the results. The study was conducted at a single centre, and neonates were followed only for the first 72 hours of life, making it difficult to assess long-term outcomes such as neurodevelopmental impact or recurrence of hypoglycemia. Furthermore, the relatively short hospital stays in both groups may have limited our ability to detect significant differences in hospital stay duration between the two groups.

CONCLUSION

In conclusion, while this study did not find statistically significant differences between the use of 40% oral dextrose gel and standard breastfeeding practices in reducing hypoglycemia incidence or the need for intravenous dextrose, the findings suggest a potential clinical benefit. Larger-scale studies are needed to confirm these results and to further explore the role of oral dextrose gel in neonatal hypoglycemia management.

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