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Research Article

Neonatal Polycythemia in Infants of Gestational Diabetic Mothers

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ABSTRACT
Background & Objectives: Neonatal polycythemia (venous hematocrit 65%) is a well-recognized hematological complication in infants of gestational diabetic mothers (IGDMs). Fetal hyperinsulinemia and intrauterine hypoxia accelerate erythropoiesis, leading to increased red cell mass and blood hyperviscosity. This prospective observational study evaluated the incidence, hematological parameters, risk factors, and immediate clinical correlates of neonatal polycythemia in IGDMs admitted to the Neonatal Intensive Care Unit (NICU).
Methods: A hospital-based prospective cohort study was conducted in the NICUs attached to J.J.M. Medical College (Bapuji Child Health Institute, C.G. Hospital, and Women and Children Hospital, Davangere) over an 18-month period. A total of 105 live-born neonates of mothers with gestational diabetes mellitus (GDM) were enrolled. Central venous hematocrit was measured between 2 and 4 hours of life via microhematocrit centrifugation. Polycythemia was defined as a venous hematocrit 65%. Hematological parameters, timing of maternal GDM onset (early <24 weeks vs. late > 24 weeks), birth weight percentiles, and associated metabolic conditions were evaluated.
Results: The overall incidence of neonatal polycythemia was 27.6% (n=29/105). Polycythemia occurred in 30.0% (n=6/20) of infants born to mothers with early-onset GDM and in 27.1% (n=23/85) of those born to mothers with late-onset GDM. Large for gestational age (LGA) infants demonstrated a higher rate of polycythemia compared to appropriate for gestational age (AGA) infants. Polycythemic neonates showed a strong co-occurrence with hyperbilirubinemia (43.8% overall incidence) and hypoglycemia (64.8% overall incidence).
Conclusion: Neonatal polycythemia is a frequent hematological alteration in IGDMs, affecting over a quarter of live births. Mandatory venous hematocrit screening at 2 to 4 hours of life is essential in all IGDMs to facilitate early detection and prevent hyperviscosity-related microvascular complications.

Keywords: Infant of Diabetic Mother, Gestational Diabetes Mellitus, Neonatal Polycythemia, Venous Hematocrit, Hyperviscosity, Erythropoiesis.

INTRODUCTION

Gestational Diabetes Mellitus (GDM) complicates approximately 3% to 10% of pregnancies worldwide and represents a significant risk factor for fetal and neonatal morbidities. Among the diverse physiological alterations observed in infants of diabetic mothers (IDMs), neonatal polycythemia defined as a central venous hematocrit > 65%—is one of the most clinically critical hematological derangements.

The pathophysiology of polycythemia in diabetic pregnancies is rooted in the Pedersen hypothesis and chronic intrauterine hypoxia. Maternal hyperglycemia induces fetal hyperglycemia, which triggers beta-cell hyperplasia and fetal hyperinsulinemia. Hyperinsulinemia increases the fetal basal metabolic rate and tissue oxygen consumption. The resulting relative tissue hypoxia stimulates endogenous fetal erythropoietin (EPO) production by the liver and kidneys, driving bone marrow and extramedullary hyper-erythropoiesis. This process leads to elevated nucleated red blood cell (nRBC) counts and an expanded circulating red cell mass at birth.

As demonstrated by Mimouni et al. (1986) in

their prospective study, neonatal polycythemia occurs in nearly 30% of diabetic offspring compared to less than 6% of non-diabetic controls. Elevated venous hematocrit leads to exponential increases in blood viscosity once the hematocrit exceeds 65%, impairing microvascular perfusion across cerebral, renal, and gastrointestinal vascular beds. Furthermore, the degradation of an expanded erythrocyte volume significantly increases the bilirubin load, predisposing these infants to severe jaundice.

While maternal glycemic control is known to influence fetal growth, specific data regarding the incidence, hematological indicators, and timing of GDM onset in relation to polycythemia remain crucial for neonatal care. This study prospectively evaluated neonatal polycythemia in IGDMs to determine its incidence, association with GDM timing, and co-existing metabolic alterations.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational cohort study was conducted in the Neonatal Intensive Care Units (NICUs) attached to the Department of Pediatrics, J.J.M. Medical College, Davangere, Karnataka, India (Bapuji Child Health Institute and Research Centre, C.G. Hospital, and Women and Children Hospital) over an 18-month period (March 2021 to October 2022).

Eligibility Criteria

- **Inclusion Criteria:** All live-born neonates delivered to mothers diagnosed with GDM admitted to the NICU.
- **Exclusion Criteria:** Neonates with incomplete maternal antenatal records, stillbirths, severe major structural lethal anomalies, or lack of parental informed consent.

Hematological Protocol and Polycythemia Assessment

1. **Sampling:** Central venous blood samples were drawn from a peripheral vein between 2 and 4 hours of life without applying a tourniquet to avoid hemoconcentration artifacts.
2. **Hematocrit Determination:** Plain microhematocrit capillary tubes (74 mm length, 1.0 mm internal diameter) were filled to 75% capacity and centrifuged at 11,700 rpm for 5 minutes.
3. **Diagnostic Cutoff:** Polycythemia was defined as a central venous hematocrit > 65\%.
4. **Stratification & Monitoring:** Mothers were categorized by GDM onset timing: Early Onset (<24 weeks of gestation) vs. Late Onset (> 24 weeks). Neonates were categorized by intrauterine growth as Small for Gestational Age (SGA), Appropriate for Gestational Age (AGA), or Large for Gestational Age (LGA). Serial blood glucose and serum bilirubin levels were monitored to evaluate co-existing hypoglycemia (<40 mg/dL) and hyperbilirubinemia.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 22.0. Categorical data (polycythemia status, GDM timing, growth categories) were reported as frequencies and percentages. Continuous variables (venous hematocrit, gestational age) were expressed as mean \pm standard deviation. Between-group comparisons were made using the Chi-Square test (\chi^2) and Student's t-test. A p-value < 0.05 was considered statistically significant.

RESULTS

Population Characteristics

A total of 105 IGDMs were evaluated. The mean gestational age was 37.54 \pm 1.80 weeks. Mothers were categorized into early-onset GDM (19.0\%, n=20) and late-onset GDM (81.0%, n=85). Growth assessment revealed 53.3% (n=56) AGA, 33.3% (n=35) LGA, and 13.3% (n=14) SGA infants.

Incidence of Neonatal Polycythemia

Neonatal polycythemia (venous hematocrit \ge 65\%) was identified in **29 of 105 infants**, yielding an overall incidence of **27.6%**.

Polycythemia Status in IGDMs (N=105)

Polycythemic (Hct >= 65%) : 27.6% (29)
Non-Polycythemic : 72.4% (76)

Polycythemia by Timing of GDM Onset and Growth Category

When stratified by maternal GDM onset, polycythemia occurred in 30.0% (n=6/20) of neonates in the early-onset GDM group compared to 27.1% (n=23/85) in the late-onset GDM group. Although polycythemia was slightly higher in the early-onset group, the difference was not statistically significant (p =

0.792).
Polycythemia rates were higher among LGA infants (34.3\%, n=12/35) compared to AGA infants (25.0\%, n=14/56).

Table 1: Polycythemia Distribution according to Maternal GDM Timing and Neonatal Parameters

Parameter	Subgroup	Total (N=105)	Polycythemic (n=29)	Non-Polycythemic (n=76)	p-value
GDM Onset Timing	Early Onset (<24 wks)	20	6 (30.0%)	14 (70.0%)	0.792
	Late Onset (> 24 wks)	85	23 (27.1%)	62 (72.9%)	
Growth Category	Large for GA (LGA)	35	12 (34.3%)	23 (65.7%)	0.312
	Appropriate for GA (AGA)	56	14 (25.0%)	42 (75.0%)	
	Small for GA (SGA)	14	3 (21.4%)	11 (78.6%)	
Gestational Age	Preterm (<37 wks)	20	5 (25.0%)	15 (75.0%)	0.781
	Term (\ge 37 wks)	85	24 (28.2%)	61 (71.8%)	

Associated Metabolic and Hematological Derangements

Polycythemic neonates demonstrated a high co-occurrence of metabolic complications. Among polycythemic infants (n=29), 72.4% experienced hypoglycemia (compared to 61.8% in non-polycythemic infants), and 58.6% developed hyperbilirubinemia requiring phototherapy.

Table 2: Co-occurrence of Metabolic Alterations with Polycythemia

Associated Condition	Overall Cohort (N=105)	Polycythemic Group (n=29)	Non-Polycythemic Group (n=76)
Hypoglycemia (<40 mg/dL)	68 (64.8%)	21 (72.4%)	47 (61.8%)
Hyperbilirubinemia	46 (43.8%)	17 (58.6%)	29 (38.2%)
Hypocalcemia (<7.0 mg/dL)	37 (35.2%)	11 (37.9%)	26 (34.2%)

DISCUSSION

Neonatal polycythemia is a major hematological adaptation to the altered intrauterine environment of diabetic pregnancies. In this prospective study, the observed 27.6% incidence of polycythemia in IGDMs closely replicates the classic findings of Mimouni et al. (1986), who documented a 29.4% incidence of venous hematocrit > 65% among infants of diabetic mothers.

Pathophysiological Mechanisms

The development of polycythemia in IGDMs is primarily driven by active intrauterine hyper-erythropoiesis rather than passive intrapartum maternal-fetal blood transfusion. Persistent maternal hyperglycemia leads to fetal hyperinsulinemia, elevating fetal oxygen consumption. This relative tissue hypoxia stimulates fetal erythropoietin production in both the renal and hepatic tissue, driving erythroid progenitor proliferation. Consequently, neonates exhibit increased red cell volume, elevated reticulocyte counts, and heightened circulating nucleated red blood cells at birth.

Clinical Significance and Metabolic Links

Polycythemia significantly increases blood viscosity, leading to sluggish microvascular flow and compromised tissue oxygenation. In our study, polycythemic infants showed a high rate of concurrent hyperbilirubinemia (58.6%). This association is explained by the expanded erythrocyte mass; as these erythrocytes undergo normal neonatal clearance, the hemoglobin breakdown overloads hepatic conjugation pathways.

Additionally, the co-occurrence of hypoglycemia (72.4% in polycythemic

neonates) underscores the increased glucose consumption by an expanded erythrocyte volume, compounded by hyperinsulinemia-mediated suppression of hepatic glucose output.

Screening and Management Implications

Because physical examination alone is unreliable for identifying hyperviscosity, central venous hematocrit sampling at 2 to 4 hours of life remains the gold standard. Early screening permits timely interventions, such as partial exchange transfusion or fluid management, preventing microvascular complications such as renal vein thrombosis or necrotizing enterocolitis.

CONCLUSION

Neonatal polycythemia occurs in 27.6% of infants born to gestational diabetic mothers. Driven by fetal hyperinsulinemia and intrauterine hypoxia, polycythemia frequently co-occurs with hyperbilirubinemia and hypoglycemia. Central venous hematocrit screening between 2 and 4 hours of life should be routinely performed in all IGDMs to enable early detection and management.

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