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Management of Neonatal Hyperglycemia Through Glucose Infusion Rate Adjustment in an Extremely Low Birth Weight Preterm Infant: A Case Report

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ABSTRACT

Background: Neonatal hyperglycemia is a common metabolic disturbance in preterm infants, particularly those with extremely low birth weight (ELBW). Immaturity of glucose homeostasis, critical illness, infection, and nutritional interventions may contribute to fluctuations in blood glucose levels. Appropriate glucose management is essential to prevent both hyperglycemia-related complications and inadequate nutritional intake. Case Presentation: We report the case of a female preterm infant born at 29 weeks and 4 days of gestation with a birth weight of 815 g (ELBW). The infant was admitted to the neonatal intensive care unit with severe respiratory distress and subsequently developed neonatal pneumonia and sepsis. Initial blood glucose was 44 mg/dL and increased to 128 mg/dL following a 10% dextrose bolus. Six hours after correction of the initial hypoglycemia, blood glucose increased to 201 mg/dL, indicating neonatal hyperglycemia. Glucose management was performed through serial blood glucose monitoring and individualized adjustment of the glucose infusion rate (GIR). The GIR was adjusted from 5.56 mg/kg/min on the first day to 6.94 mg/kg/min on the second day and 8.33 mg/kg/min on the third day. Blood glucose subsequently decreased to 122 mg/dL and 88 mg/dL, respectively, without insulin therapy. The infant received respiratory support, antibiotic therapy, nutritional management, and supportive care. During the subsequent hospitalization, respiratory status gradually improved. Endotracheal tube culture later confirmed ventilator-associated pneumonia caused by Serratia marcescens. Conclusion: This case highlights the dynamic nature of glucose dysregulation in an ELBW preterm infant with critical illness and infection. The transition from early hypoglycemia to hyperglycemia emphasizes the importance of serial glucose monitoring and individualized GIR adjustment. In this case, glucose levels were successfully controlled without insulin therapy, while maintaining adequate glucose delivery to support the nutritional requirements of a high-risk preterm infant. Integrated metabolic, nutritional, respiratory, and infectious monitoring is essential in the management of glucose disturbances in ELBW infants.

1. INTRODUCTION

Neonatal hyperglycemia is defined as a venous blood glucose concentration >125 mg/dL (6.9 mmol/L) or a capillary blood glucose concentration >150 mg/dL in neonates (Beardsall, 2021). This metabolic disorder is frequently encountered in neonatal intensive care units (NICUs), particularly among preterm infants and those with extremely low birth weight (ELBW), defined as a birth weight of <1,000 g. The incidence of hyperglycemia among preterm infants with very low birth weight may reach 20–80% (Angelis, Jaleel, and Brion, 2023), making it an important challenge in the metabolic management of high-risk neonates.

ELBW infants are particularly vulnerable to disturbances in glucose homeostasis because of immature hepatic and pancreatic function, limited insulin responses, and an exaggerated hormonal response to stress. These conditions may be further exacerbated by critical illness, infection, sepsis, and the administration of glucose-containing parenteral nutrition. At the same time, an adequate glucose supply must be maintained to meet the metabolic and growth

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requirements of preterm infants. Therefore, regulation of the glucose infusion rate (GIR) in ELBW infants requires a careful balance between preventing hypoglycemia and controlling hyperglycemia.

Neonatal hyperglycemia is not merely a laboratory abnormality but is associated with an increased risk of clinical complications. Persistent or severe hyperglycemia has been reported to be associated with an increased risk of intraventricular hemorrhage (IVH), retinopathy of prematurity (ROP), infection or sepsis, impaired growth, and adverse neurodevelopmental outcomes (Titchiner et al., 2024). However, these associations should be interpreted cautiously because ELBW infants commonly have multiple potential confounding factors, including organ immaturity, infection, respiratory disorders, and intensive care interventions.

Infection and systemic stress responses are important factors that may further impair glucose regulation in neonates. Increased concentrations of counter-regulatory hormones, including catecholamines and cortisol, during critical illness may increase glucose production and reduce insulin sensitivity. Consequently, pneumonia and sepsis in preterm infants may contribute to the development of hyperglycemia while simultaneously complicating the management of nutritional and glucose requirements.

The management of neonatal hyperglycemia should take into account the severity of hyperglycemia, the clinical condition of the infant, energy requirements, and nutritional and fluid administration. Initial management generally includes evaluation and adjustment of glucose intake through regulation of the glucose infusion rate (GIR), accompanied by serial monitoring of blood glucose concentrations. In cases of persistent or severe hyperglycemia despite adjustment of glucose intake, intravenous insulin may be considered with close monitoring to avoid hypoglycemia (Parappil et al., 2022). The A.S.P.E.N. guidelines on parenteral nutrition in preterm infants also emphasize the importance of individualized glucose administration according to the clinical condition and metabolic requirements of the neonate (Angelika et al., 2023).

Although neonatal hyperglycemia in preterm infants has been widely reported, clinical reports describing the course of hyperglycemia in ELBW infants with concomitant infection and sepsis, including changes in glucose concentrations following correction of initial hypoglycemia and the response to GIR adjustment without insulin therapy, remain clinically relevant. Such cases illustrate the complexity of glucose management in ELBW infants who simultaneously require adequate nutritional support for growth and face metabolic stress associated with critical illness.

This case report describes the clinical course and management of hyperglycemia in a preterm infant born at a gestational age of 29 weeks and 4 days with a birth weight of 815 g (ELBW), who developed respiratory distress and neonatal infection. The case specifically describes the transition from hypoglycemia during the early postnatal period to hyperglycemia, serial glucose monitoring, and the response to GIR adjustment during NICU admission. This report aims to provide a clinical illustration of the challenges involved in maintaining glucose homeostasis in ELBW infants with comorbid conditions and to contribute to considerations for monitoring and managing glucose disturbances in high-risk neonates.

2. METHOD

Case Report

A female infant was born at Prof. Dr. Margono Soekarjo Regional General Hospital on May 4, 2026, at 09:53 Western Indonesia Time (WIB) by cesarean section to a 34-year-old mother, gravida 2, para 1, abortus 0 (G2P1A0), at a gestational age of 29 weeks and 4 days. Indications for termination of pregnancy included a history of previous cesarean section, fetal growth restriction, oligohydramnios, external hemorrhoids, and grade I obesity. At birth, the infant appeared premature, with a weak cry and decreased muscle tone. Following initial resuscitation, the infant had a heart rate of 130 beats/min and spontaneous respiration accompanied by chest wall retractions and grunting. Airway suctioning was performed, followed by endotracheal intubation using a 2.5-mm endotracheal tube (ETT) inserted to a depth of 6.8 cm and positive-pressure ventilation. After initial stabilization, the respiratory rate was 58 breaths/min, with an oxygen

saturation of 97%. The infant subsequently received vitamin K prophylaxis and gentamicin ophthalmic ointment.

Anthropometric measurements at birth were as follows: body weight, 815 g; body length, 31 cm; head circumference, 24 cm; chest circumference, 22 cm; abdominal circumference, 21 cm; and mid-upper arm circumference, 8 cm. Apgar scores at 1, 5, and 10 minutes were 5, 6, and 7, respectively, with a Downes score of 5. Based on these findings, the infant was diagnosed as a preterm infant with very low birth weight (VLBW), appropriate for gestational age, and severe respiratory distress. After stabilization in the delivery room, the infant was transferred to the neonatal intensive care unit (NICU) in a prewarmed incubator with ventilatory support through the ETT. Upon arrival at the NICU at 10:25 WIB, the infant appeared weak, with severe chest wall retractions and cyanosis that improved following oxygen administration. Mechanical ventilation was initiated using assist/control mandatory ventilation (A/CMV) mode with a peak inspiratory pressure (PIP) of 20 cmH_2O, inspiratory time (Ti) of 0.35 seconds, respiratory rate (RR) of 40 breaths/min, fraction of inspired oxygen (FiO_2) of 30%, and positive end-expiratory pressure (PEEP) of 6 cmH_2O. A 5-Fr orogastric tube (OGT) was inserted to a depth of 40 cm.

Physical examination revealed severe chest wall retractions without grunting, good bilateral air entry with vesicular breath sounds over both lung fields, regular heart sounds, a nondistended abdomen with normal peristalsis, and normal-appearing anus and external genitalia. The infant had not yet urinated or passed meconium. Gastric aspirate obtained through the OGT consisted of clear fluid. Given the clinical findings suggestive of respiratory distress syndrome, surfactant therapy was administered in two doses of 1.7 mL each at 10:45 WIB and 11:00 WIB. Central venous access was established through an umbilical venous catheter (UVC). On the first day of life, the fluid requirement was 80 mL/kg/day (65.2 mL/day), administered as 10% dextrose with a glucose infusion rate (GIR) of 5.56 mg/kg/min. The patient also received empirical antibiotic therapy consisting of ampicillin-sulbactam 50 mg every 12 hours and gentamicin 5 mg every 36 hours, as well as a loading dose of caffeine citrate 16 mg followed by a maintenance dose of 4 mg daily.

Initial laboratory investigations included a complete blood count, random blood glucose (RBG), C-reactive protein (CRP), blood culture, blood gas analysis, and chest radiography. The initial RBG was 44 mg/dL, indicating neonatal hypoglycemia. The patient was immediately administered a 1.7-mL bolus of 10% dextrose, after which repeat testing showed an increase in blood glucose to 128 mg/dL. The other laboratory findings are presented in Table 1. Blood gas analysis showed a pH of 7.47, pO_2 of 45.8 mmHg, pCO_2 of 11.1 mmHg, base excess of −12.89 mmol/L, oxygen saturation of 94.3%, and an alveolar–arterial oxygen gradient ($AaDO_2$) of 152.3 mmHg, with an impression of partially compensated respiratory alkalosis.

Table 1. Laboratory Findings on Day 1 of Hospitalization

Parameter	**Value**
Hemoglobin	16.3 g/dL
White blood cell count	4,530/µL
Hematocrit	51%
Platelet count	121,000/µL
Blood group	B
Random blood glucose	128 mg/dL

Blood glucose monitoring demonstrated a significant increase in blood glucose levels. Six hours after correction of the initial hypoglycemia, the blood glucose concentration increased to 201 mg/dL, meeting the criteria for neonatal hyperglycemia. During the same period, transcutaneous bilirubin (TcB) measurements were obtained, as presented in Table 2. The TcB level at 18 hours of life was higher than the bilirubin treatment threshold (BFT); therefore, the patient received phototherapy for 36 hours.

Table 2. Transcutaneous Bilirubin (TcB) Measurements

Parameter	Value	Time
TcB	3.2	12 hours of life
BFT / BTT	4.6 / 7.01	12 hours of life
TcB	8.1	18 hours of life
BFT / BTT	4.6 / 7.8	18 hours of life

On the second day of hospitalization, the patient's clinical condition showed relative improvement. The patient remained weak, with mild chest retractions and no cyanosis. The Downes score decreased to 2. Mechanical ventilation was continued in A/CMV mode with a PIP of 15 cmH_2O, Ti of 0.35 seconds, RR of 40 breaths/min, FiO_2 of 30%, and PEEP of 6 cmH_2O. The OGT remained in place, with clear gastric residuals. The patient had begun to urinate and pass meconium. RBG was 122 mg/dL. Chest radiography showed findings consistent with pneumonia accompanied by hepatomegaly. The fluid requirement was increased to 100 mL/kg/day (81.5 mL/day), administered as 10% dextrose with a GIR of 6.94 mg/kg/min. Antibiotic therapy was continued according to the previous regimen.

On the third day of hospitalization, the patient's general condition remained stable, with mild chest retractions and no cyanosis. Ventilatory support was continued using the same settings as on the previous day. RBG decreased to 88 mg/dL without insulin intervention. Radiological evaluation continued to show pneumonia and hepatomegaly. The fluid requirement was increased to 120 mL/kg/day (97.8 mL/day), administered as 10% dextrose with a GIR of 8.33 mg/kg/min through the UVC. Antibiotic therapy was continued, and oral care with 0.2 mL of breast milk every 4 hours was provided when breast milk was available.

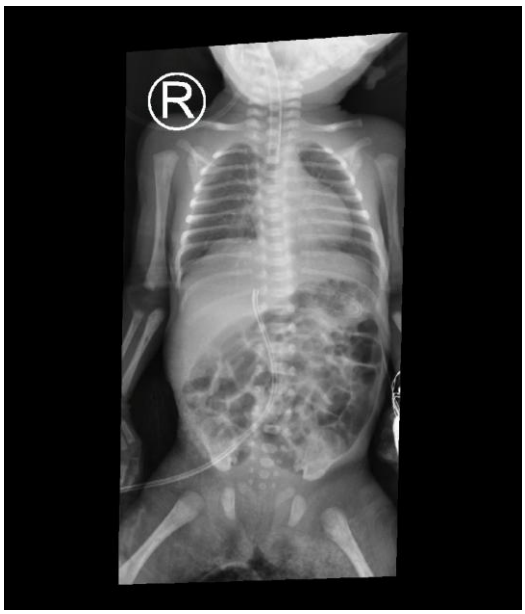

Figure 1. Babygram radiograph findings

By day 34 of hospitalization, corresponding to 33 days of age, the infant showed improvement in respiratory status, with a stable respiratory pattern and no increased requirement for ventilatory support. Hematological evaluation revealed normocytic normochromic anemia. In addition, endotracheal tube (ETT) culture confirmed ventilator-associated pneumonia (VAP) caused by Serratia marcescens, a finding consistent with the clinical course and the patient's existing risk factors.

3. RESULT AND DISCUSSION

Result

Serial blood glucose monitoring demonstrated a transition from initial neonatal hypoglycemia to hyperglycemia during the early postnatal period. On the first day of life, the initial blood glucose concentration was 44 mg/dL and increased to 128 mg/dL following administration of a 10% dextrose bolus. Six hours after correction of hypoglycemia, blood glucose increased to 201 mg/dL, meeting the criteria for neonatal hyperglycemia. The GIR was subsequently adjusted according to the patient's clinical condition and glucose concentrations. On the second day of hospitalization, the blood glucose concentration was 122 mg/dL, while the GIR was increased to 6.94 mg/kg/min. By the third day, blood glucose decreased to 88 mg/dL despite a further increase in GIR to 8.33 mg/kg/min, without the use of insulin.

During the early course of hospitalization, the patient's respiratory condition gradually improved. On the second day, the Downes score decreased from 5 to 2, with only mild chest retractions and no cyanosis. Mechanical ventilation was continued with stable settings, while chest radiography continued to demonstrate pneumonia and hepatomegaly. By day 34 of hospitalization, corresponding to 33 days of age, the infant demonstrated further improvement in respiratory status, with a stable respiratory pattern and no increased requirement for ventilatory support. Hematological evaluation at this stage showed normocytic normochromic anemia.

Microbiological evaluation later confirmed ventilator-associated pneumonia (VAP), with endotracheal tube culture yielding Serratia marcescens. The infant received empirical antibiotic therapy from the early hospitalization period, which was continued according to the clinical course. Overall, the glucose profile showed spontaneous normalization after adjustment of glucose administration, with no insulin therapy required, while respiratory status improved progressively during hospitalization. These findings illustrate the dynamic relationship between glucose requirements, critical illness, nutritional support, and clinical stabilization in an ELBW infant..

Discussion

This case report describes the management of neonatal hyperglycemia in a preterm ELBW infant born at a gestational age of 29 weeks and 4 days, complicated by bacterial neonatal pneumonia and early-onset sepsis, and provides a clinical reference for the management of metabolic complications in high-risk neonates in healthcare settings in Indonesia. Based on the time of onset, neonatal hyperglycemia can be classified as early-onset when it occurs within the first 48 hours of life and late-onset when it develops after 48 hours of life. The severity of hyperglycemia can be categorized as mild when the blood glucose concentration is >8.3 mmol/L (150 mg/dL), moderate when >10 mmol/L (180 mg/dL), and severe when >15 mmol/L (270 mg/dL) (Krishnan, Bhatt, and Seetharam, 2022).

From an epidemiological perspective, the incidence of neonatal hyperglycemia is closely associated with gestational age and birth weight. Estimates reported in the literature range from 45% to 80%, with the incidence being inversely related to birth weight among preterm infants. An incidence as high as approximately 80% has been reported in extremely preterm infants with a birth weight <750 g, approximately 45% in infants with a birth weight <1,000 g, and only approximately 2% in infants weighing >2,000 g (Angelis, Jaleel, and Brion, 2023). These findings are consistent with data reported by Böettger et al., indicating a negative correlation between gestational age and birth weight and the occurrence of hyperglycemic episodes. Across different studies, the incidence of neonatal hyperglycemia has ranged from 40% to 80%, partly due to differences in definitions and methodologies used to assess blood glucose concentrations (Kalogeropoulou, Iglesias-Platas, and Beardsall, 2023).

Neonatal hyperglycemia can arise through several mechanisms, including increased endogenous glucose production, reduced glucose uptake by peripheral tissues, and excessive exogenous glucose administration. In general, hyperglycemia results from excessive glucose production by the infant, excessive exogenous glucose infusion, and reduced glucose utilization capacity (Blanco and Kim, 2022).

One of the most common iatrogenic causes is parenteral nutrition. Parenteral nutrition is frequently used in preterm and critically ill infants in neonatal units. VLBW infants require higher fluid intake because of increased caloric requirements, substantial insensible fluid losses, and diuresis related to renal immaturity, which may increase the risk of iatrogenic hyperglycemia (Angelika et al., 2023). In addition, excessive lipid infusion in neonates may contribute to hyperglycemia by reducing peripheral glucose utilization and impairing the ability of insulin to suppress hepatic glucose production (Krishnan, Bhatt, and Seetharam, 2022).

Sepsis is another important non-iatrogenic cause that should be considered. Neonatal sepsis may manifest with hyperglycemia as a nonspecific clinical finding. In this setting, hyperglycemia may result from reduced insulin secretion, decreased peripheral glucose utilization, and a stress response mediated by counter-regulatory hormones (Krishnan, Bhatt, and Seetharam, 2022). Preterm infants are also reported to have a higher incidence of hyperglycemia associated with fungal sepsis (Angelis, Jaleel, and Brion, 2023).

Medications are also significant etiological factors. Glucocorticoids can induce hyperglycemia by increasing insulin resistance and gluconeogenesis while reducing insulin production. Methylxanthines increase catecholamine secretion, whereas phenytoin may suppress insulin release or cause insulin insensitivity. Beta-adrenergic agents, including dopamine, epinephrine, and norepinephrine, can influence hepatic and muscular glycogenolysis and gluconeogenesis (Gomella, Cunningham, and Eyal, 2020). In addition, maternal use of diazoxide may cause transient neonatal hyperglycemia because the drug can cross the placenta and suppress insulin secretion (Blanco and Kim, 2022).

Physiological stress also contributes to the development of neonatal hyperglycemia. Increased concentrations of stress hormones, such as epinephrine and norepinephrine, whether triggered by physiological stress or administered intravenously to support cardiac output, can inhibit both insulin secretion and insulin action. Circulating epinephrine and norepinephrine concentrations may also increase two- to six-fold during dopamine and dobutamine infusions (Gomella, Cunningham, and Eyal, 2020). Understanding the pathophysiology of neonatal hyperglycemia requires consideration of glucose homeostasis during fetal and neonatal life. Glucose is the primary energy substrate during fetal development and the neonatal period. After birth, glucose becomes a major energy substrate for the central nervous system. During fetal life, glucose homeostasis is entirely dependent on placental glucose transfer, with no significant endogenous glucose production occurring in the fetus (Blanco and Kim, 2022).

At birth, a sudden change occurs in the source of glucose supply. Maternal glucose delivery is abruptly interrupted when the umbilical cord is clamped. During the first hours of life, newborns maintain blood glucose concentrations through the activation of glycogenolysis and gluconeogenesis mediated by complex hormonal interactions. After birth, glucagon, epinephrine, growth hormone, and cortisol concentrations increase, promoting an increase in serum glucose through glycogenolysis, while the accompanying suppression of insulin levels helps maintain stable plasma glucose concentrations (Blanco and Kim, 2022).

In preterm infants, these regulatory mechanisms are immature, making them more susceptible to disturbances in glucose homeostasis. Although the mechanisms underlying hyperglycemia in preterm infants are not yet fully understood, they are thought to involve an inadequate insulin response to glucose, failure to suppress gluconeogenesis or endogenous glucose production in response to intravenous glucose administration, and reduced expression of glucose transporters (Angelis, Jaleel, and Brion, 2023).

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fully understood, they are thought to involve an inadequate insulin response to glucose, failure to suppress gluconeogenesis or endogenous glucose production in response to intravenous glucose administration, and reduced glucose transporter levels (Angelis, Jaleel, and Brion, 2023).

Pancreatic immaturity also plays an important role. Glucose metabolism dysregulation in preterm infants is multifactorial and includes inadequate insulin production, limited glycogen stores, and possible insulin resistance. In critically ill neonates, reduced insulin production and decreased insulin sensitivity may occur because of immature or less responsive peripheral insulin receptors. Increased secretion of counter-regulatory hormones, such as epinephrine and cortisol, in response to physiological stress also contributes to hyperglycemia (Angelis, Jaleel, and Brion, 2023).

Hyperglycemia subsequently has a direct effect on serum osmolality. For every 1 mmol/L (18 mg/dL) increase in blood glucose concentration, serum osmolality increases by approximately 1 mOsm/L. When serum glucose reaches approximately 22.2 mmol/L (400 mg/dL), serum osmolality may exceed 300 mOsm/L, potentially causing rapid shifts of intracellular water in the brain and increasing the susceptibility of the brain to cerebral hemorrhage (Gomella, Cunningham, and Eyal, 2020). Neonatal hyperglycemia is associated with a broad range of clinical complications, ranging from clinically manageable dehydration to life-threatening conditions. Hyperglycemia can cause osmotic diuresis, potentially resulting in polyuria and dehydration. A glucose infusion rate (GIR) >15 mg/kg/min may increase excessive lipogenesis, which rarely produces overt clinical manifestations but may be detected through elevations in liver enzymes (Krishnan, Bhatt, and Seetharam, 2022).

Neurological complications are a major concern, particularly in extremely preterm infants. In VLBW infants, a blood glucose concentration >10 mmol/L during the first 28 days of life has been associated with more than a twofold increase in 28-day mortality. In addition, hyperglycemia during the first 24 hours of life has been associated with reduced cerebral white matter structure on magnetic resonance imaging (MRI). Hyperglycemia has also been identified as a predisposing factor for severe intraventricular hemorrhage and increased mortality in VLBW infants (Beardsall, 2021).

Long-term complications have also been documented. Hyperglycemia has been associated with late-onset sepsis, necrotizing enterocolitis, retinopathy of prematurity, adverse neurodevelopmental outcomes, and prolonged hospitalization (Titchiner et al., 2024). Hyperglycemia may also cause electrolyte disturbances through increased sodium loss in neonates with glycosuria, as well as impaired creatinine clearance associated with acute kidney injury (Krishnan, Bhatt, and Seetharam, 2022). The management of neonatal hyperglycemia includes both conservative and medical approaches. Conservative management primarily focuses on reducing the GIR toward the target range, minimizing precipitating factors, providing adequate supportive care to reduce physiological stress, and ensuring sufficient caloric intake (Parappil et al., 2022).

Several important principles should be considered in management. Dextrose concentrations <5% and a GIR below 4 mg/kg/min are not recommended for the management of hyperglycemia because reducing glucose infusion to excessively low levels may substantially decrease caloric intake and impair infant growth. Conversely, a GIR >15 mg/kg/min should be avoided because it may promote excessive lipogenesis and result in abnormalities in liver enzyme levels (Krishnan, Bhatt, and Seetharam, 2022).

Several genetic syndromes and chromosomal abnormalities have been associated with neonatal hyperglycemia. Hyperglycemia has been observed in conditions such as 46,XX deletion of chromosome 13q, chromosome 6q24 abnormalities involving paternal uniparental disomy, Wolcott–Rallison syndrome, DEND (Developmental delay, Epilepsy, and Neonatal Diabetes) syndrome, Rogers syndrome, and IPEX (Immune dysregulation, Polyendocrinopathy, Enteropathy, X-linked) syndrome. Most cases present as isolated neonatal diabetes, whereas others are associated with various extrapancreatic clinical abnormalities. The identification of neonatal diabetes mellitus (NDM) requires a high degree of clinical awareness because its management differs from that of transient neonatal hyperglycemia and has important genetic implications for the patient's family (Gomella, Cunningham, and Eyal, 2020).

4. CONCLUSION

Neonatal hyperglycemia is a common metabolic complication in preterm infants with extremely low birth weight, particularly in critically ill infants experiencing physiological stress and infection. In this case, blood glucose increased to 201 mg/dL within the first 24 hours of life after an initial episode of hypoglycemia and was subsequently controlled through serial glucose monitoring and adjustment of glucose intake without the need for insulin therapy. This clinical course highlights the importance of individualized glucose monitoring and adjustment of the glucose infusion rate (GIR) in high-risk preterm infants.

The patient's clinical condition, characterized by respiratory distress syndrome, pneumonia, sepsis, and subsequent ventilator-associated pneumonia, indicates that glucose dysregulation in preterm infants is multifactorial. Therefore, regular metabolic monitoring should be integrated with respiratory, nutritional, hematological, and infection monitoring throughout NICU care. This case emphasizes the importance of early detection and close monitoring of changes in blood glucose concentrations to prevent complications associated with both hyperglycemia and hypoglycemia in high-risk neonates.

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