



Neonatal Hypoglycemia

10

David H. Adamkin

Introduction

The management of low blood glucose concentrations in the first 48 h of life is one of the most frequently encountered issues the healthcare provider faces in the newborn nursery. The blood levels of glucose upon which we base our decision-making remain more a matter of expert opinion rather than being evidence-based. The data needed to establish a consensus opinion on blood glucose levels that should be treated in the newborn are still not definitive enough.

In fact, the lack of consensus has led to further confusion for the clinician as two pediatric organizations, the Committee on the Fetus and Newborn from the American Academy of Pediatrics (AAP) and the Pediatric Endocrine Society (PES), have recently provided expert opinions on the management of neonatal hypoglycemia that suggested different ranges of actionable blood glucose levels [1, 2]. The AAP guidance applies only to the first 24 h of life, and the PES strategy focuses on infants beyond 48 h of life with the emphasis on making certain we identify cases of persistent hypoglycemia before these infants are discharged. The two

organizations used different approaches to suggest the different ranges of glucose to act upon in the first 48 h of life.

In a recent editorial entitled “Imperfect Advice,” Adamkin and Polin contrast the approaches of the two organizations and offer suggestions on how to merge their advice for management [3]. The AAP statement addressed only the first 24 h of life [1]. This editorial recommended a glucose level > 45 mg/dL for 24–48 h, which was not covered in the AAP algorithm. This allows transition to the PES recommendations for blood glucose >60 mg /dL after 72–96 h of age [3]. In addition, the editorial addressed the main PES concern of not missing cases of persistent neonatal hypoglycemia, whereby infants with low blood glucose levels are either missed or infants treated for hypoglycemia the first day or two are discharged before they demonstrate levels >60 to 70 mg/dL through several normal feed-fast cycles before being discharged [3].

Management of glucose in the neonate involves management of several different and at times opposing, clinical issues. The clinician must diagnose and manage hypoglycemia, promote successful breastfeeding, avoid unnecessary admissions to the neonatal intensive care unit, and also prevent infants from reaching higher blood glucose levels that reflect glucose instability and untoward neurodevelopmental outcome [4].

D. H. Adamkin, MD
Division of Neonatal Medicine, Department of
Pediatrics, University of Louisville,
Louisville, KY, USA
e-mail: David.Adamkin@louisville.edu

Case Presentation

A 24-year-old primigravida, after an uncomplicated pregnancy, is admitted in labor at 40-week gestation. She has had perinatal screening tests including a negative screen for group B streptococcal (GBS) colonization at 36-week gestation. She also had normal screening blood glucose values early in pregnancy and no history of diabetes or abnormal glucose values at any time. Rupture of membranes occurred 2 h prior to delivery; Apgars were 8 and 9 at 1 and 5 min, respectively. The male infant is AGA, weighing 3200 g.

The mother has read about breastfeeding, spoken prenatally with a lactation consultant, and plans to exclusively breastfeed. She begins by breastfeeding the infant in the delivery room, described as “successful” in the record at 45 min of age. A point-of-care (POC) blood glucose is performed after the feeding, and it is 35 mg/dL. The nurse calls you to let you know about this level and if you want to do anything. Shortly thereafter, the mother is transferred to a postpartum room.

Should this term AGA infant with no history of maternal diabetes or other risk factors have been screened at all? Indications for screening for hypoglycemia include all of the following except:

1. Small for gestational.
2. Large for gestational age.
3. Exclusive breastfeeding.
4. Infant of a diabetic mother.
5. Late preterm infant.

You have your smartphone at your bedside and you pull up the “Glucose App, Sugar Wheel” (Fig. 10.1) [4], and you confirm what you thought: that this baby’s perinatal course met none of the risk factors that would lead to a screening blood glucose. Indications include large for gestational age, small for gestational age, infant of a diabetic mother, and late preterm infants (35–37 weeks of gestation). Some authors suggest that babies with perinatal asphyxia (Apgars ≤ 5 at 5 min) also be screened.

Answer: 3

At this time you:

1. Discontinue breastfeeding and start formula.
2. Supplement breastfeeding with formula.
3. Order 40% oral glucose gel.
4. Give IV D10W.
5. Continue breastfeeding.

You look at the wheel, and you note that the initial feeding took place within the first hour of life and the level they obtained (which should have been 30 min after the first feed if the baby was at high risk) was at a level that is considered acceptable during the first 4 h of life. You advise continued breastfeeding and no further screens unless the infant shows signs of hypoglycemia.

Answer: 5

It is 5 am and the baby is breastfed again at 8 h of age. After the feeding, the nurse on routine assessment thinks the baby is slightly tremulous. She performs point-of-care (POC) glucose level, and it is 36 mg/dL. The nurse tells the mother that the baby should be supplemented with 1 oz of term formula to raise the blood glucose. The mother is very disappointed and asks the nurse to call you because her plan was to exclusively breastfeed the infant.

For some reason the next feeding wasn’t until 8 h of age. You recommended frequent feedings to promote glucose homeostasis and an optimal initiation of breastfeeding. The nurse called again at 8 h to report a POC of 36 mg/dL. On your wheel, this POC is just above the actionable level of 35 mg/dL for intravenous glucose (35 mg/dL for 4 to 24 h of age). Your choices include refeeding or IV glucose for levels 35–45 mg/dL. You should always confirm the POC with a plasma level from the lab and can wait to see if that value aligns with the POC. However, the nurse said that the infant was slightly tremulous.

At this time you:

1. Discontinue breastfeeding and start formula.
2. Supplement breastfeeding with formula.
3. Order 40% oral glucose gel.

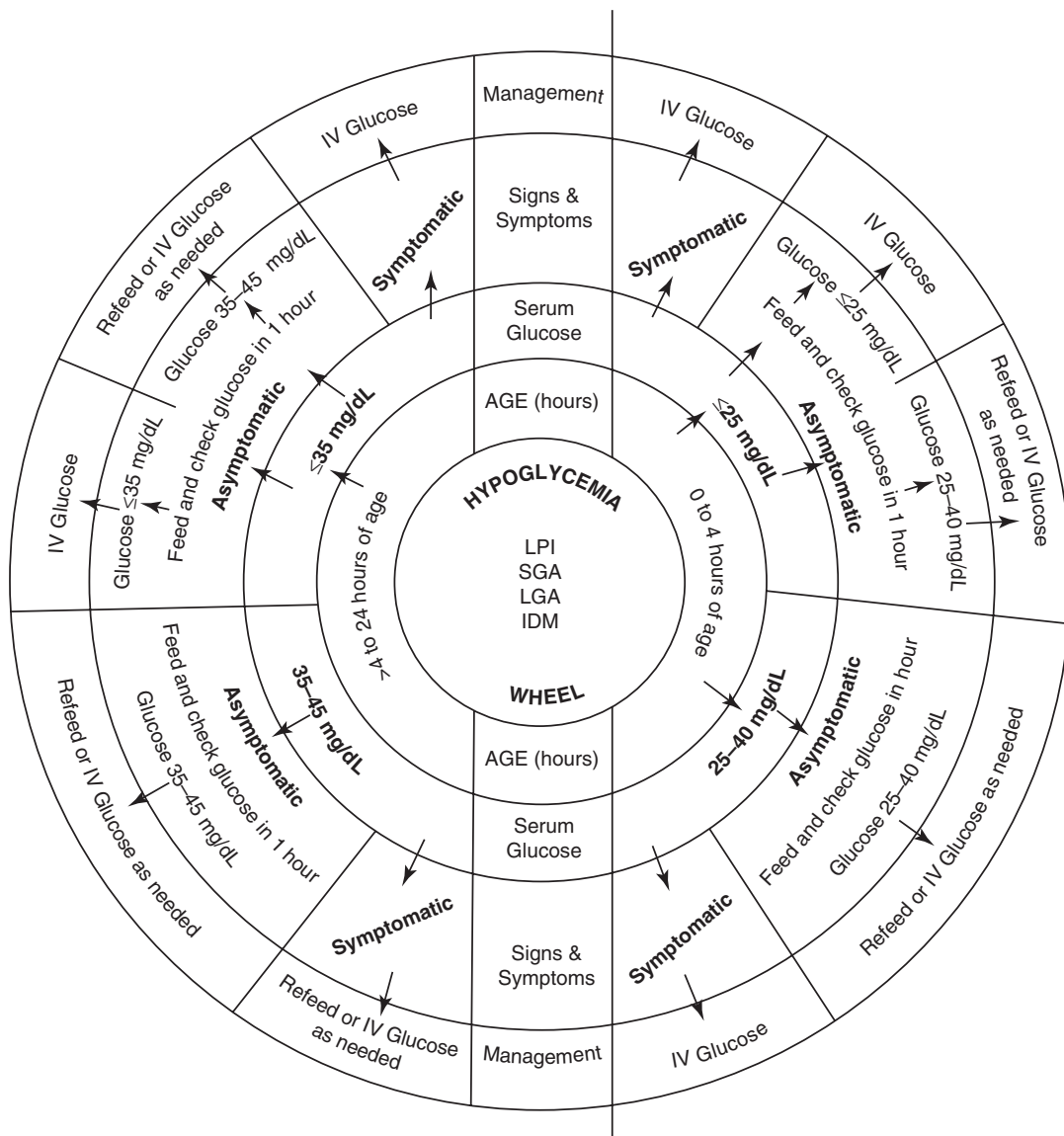


Fig. 10.1 The Sugar Wheel nomogram for the management of neonatal hypoglycemia in the first 24 h of life. Start at the center of the wheel, select the correct age and

glucose level, and then determine whether the patient is symptomatic or asymptomatic. The proper treatment can then be selected. (From Polin and Yoder [4]).

4. Give IV D10W.
5. Continue breastfeeding.

If this baby is truly jittery, then everything changes because the infant is symptomatic. The recommendation for the symptomatic infant below 40 mg/dL is immediate intravenous glucose, while you wait for the confirmatory laboratory plasma glucose. The clinical signs of

hypoglycemia are not specific and include a wide range of local or generalized manifestations that are common in sick neonates.

Your conundrum is whether this baby is truly symptomatic requiring IV glucose or is this normal newborn activity that would allow for feeding again and rechecking the POC glucose in 30 min? Remember this mother wanted exclusive breastfeeding for her infant. Do you have donor

milk for this infant if you choose to refeed? What about using dextrose gel to treat the glucose level to avoid the use of formula? The answers to these two questions are frequently “not yet.” Donor human milk as a supplement for the sluggishly feeding breastfed infant and the use of the new dextrose gel [5] may help to prevent babies with modest hypoglycemia from needing to go to the NICU for intravenous fluids. Current data suggest that the gel is safe and prevented admission to the NICU for intravenous fluids, but it did not improve neurodevelopmental outcome at 2 years of age [5, 6].

Therefore, formula might be the only choice, unless you believe the symptoms deserve intravenous treatment. If in your opinion the baby is truly “jittery,” then you would order a mini-bolus of D10W at 2 mL/kg and/or IV infusion at 5 to 8 mg/kg/min of D10W (80 mL/kg/d) to achieve a plasma glucose of 40 to 50 mg/dL. The “Sugar Wheel” offers you all these choices, and it is based on Fig. 10.2, the algorithm from the Clinical Report, Postnatal Glucose Homeostasis

in Late Preterm and Term Infants, published in Pediatrics in March 2011 from the AAP Committee on the Fetus and Newborn [1].

Could this infant simply have been left to continue breastfeeding? Yes, one of the choices was to continue simply breastfeeding this infant, monitoring POC glucose levels prior to feedings every 2 to 3 h because of the observation of this non-specific, ill-defined tremulousness that will probably resolve with further feedings and increased glucose levels. However, true jitteriness would require intravenous fluids.

Answer: 4 (If Truly Jittery); 5 (If No Clearly Defined Symptoms)

What is the value for plasma glucose at which a symptomatic infant should receive treatment?

1. 20 mg/dL.
2. 30 mg/dL.
3. 40 mg/dL.
4. 50 mg/dL.

Screening and Management of Postnatal Glucose Homeostasis in Late Preterm and Term SGA, IDM/LGA Infants

[(LPT) Infants 34-36/7 weeks and SGA (screen 0-24 hrs): IDM and LGA≥34 weeks(screen 0-12 h)]

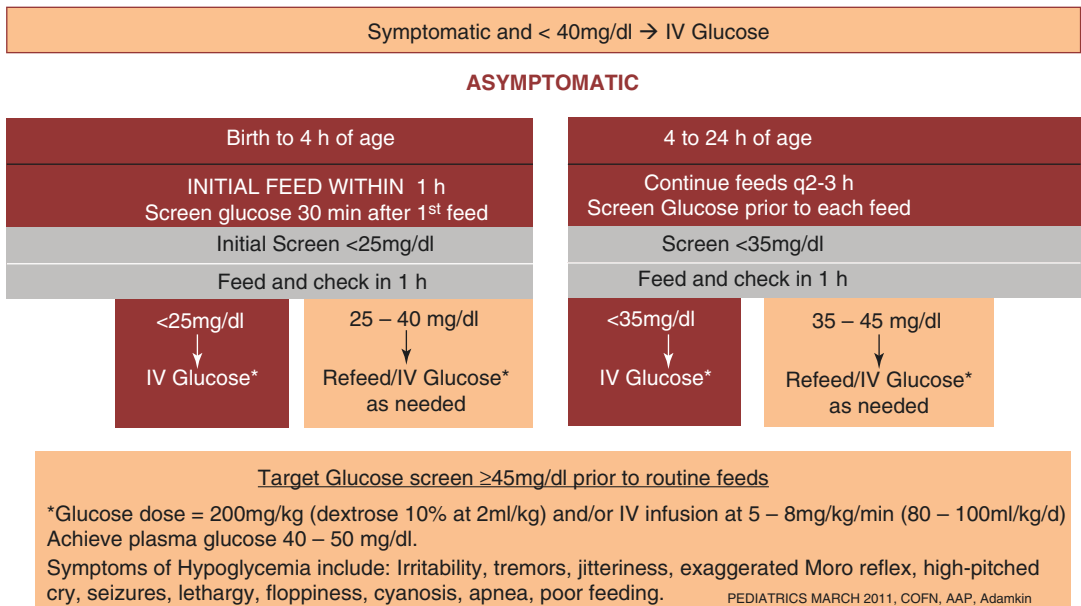


Fig. 10.2 Algorithm developed by the American Academy of Pediatrics for evaluation and treatment of neonates in the first 24 h of life. (Adamkin [1])

As alluded to above, the plasma glucose level that applies to symptomatic infants (signs included on the AAP algorithm and Sugar Wheel is <40 mg/dL [1, 4].

Answer: 3

Do infants that are exclusively breastfed have lower plasma glucose concentrations than those fed formula?

1. Yes
2. No

Infants who are exclusively breastfed tend to have lower blood glucose concentrations than those fed formula. Studies show a wide range of blood glucose values during the first 72 h of life for breastfeeding infants [7]. These same infants tend to have higher ketone levels, the principal alternate metabolic fuel for the brain. Therefore, studies indicate that breastfed term infants might have lower blood glucose concentrations and higher levels of ketone bodies than formula fed [7]. Still the same levels in the AAP algorithm should be applied to the breastfeeding infant.

Answer: 1

It is clear from this first case that the screening and management of postnatal glucose homeostasis in late preterm and term SGA, IDM/LGA infants (infant of a diabetic mother, large for gestational age) requires individual judgments and clinical choices based upon the risk and physical examination.

After birth, the normal newborn infant's plasma glucose concentration falls below levels that were prevalent in fetal life. In fact, at birth the blood glucose is about 70% of the maternal level. It falls rapidly to a nadir by 1 h of life to a value as low as 20 to 25 mg/dL [1]. These modest levels are common in healthy neonates and this fall is seen in all mammalian newborns. This is part of the normal transition to an extrauterine life. These levels are transient and the vast majority of infants are asymptomatic. Finally, the infant activates endocrine and metabolic transitions associated with successful adaptation.

When this adaptation fails, perhaps secondary to immaturity or illness, there is a limitation of substrate supply, which may disturb cerebral function and potentially result in neurologic sequelae [8]. A low plasma glucose may be indicative of this process but is not per se diagnostic. What is meant by "low"? How low is "too low"? At what glucose level does hypoglycemia lead to irreversible changes in brain structure or function?

A consistent definition of hypoglycemia does not exist for the first 2 days of life. The first neonates recognized as having significant hypoglycemia 60 years ago had seizures and blood glucose levels consistently below 20 to 25 mg/dL [9]. The abnormal signs cleared quickly after increasing the blood glucose level to >40 mg/dL. This is how "40" became a classic standard for defining hypoglycemia. Despite our enhanced understanding of metabolic disturbances and genetic disorders, we still do not have a precise complete definition of hypoglycemia. Because we don't have sufficient evidence to answer "how low is too low and for how long," we continue to rely on expert opinion and best available evidence.

Case Presentation

A term AGA infant was born after negative prenatal screens and an uneventful pregnancy to a 35-year-old gravida 2 woman. The mother also had normal prenatal glucose screens and no history of diabetes. Apgar scores were 5, 7, and 8 at 1, 5, and 10 min, respectively. The baby appeared cyanotic in the delivery room and received brief blow by oxygen.

At approximately 1 h of age, a bedside glucose was performed in the nursery when it was noted the baby had received oxygen in the delivery room. A POC glucose was obtained before the infant was sent out to the mother's room. The baby had normal vital signs and exam findings, and the oxygen saturation in room air was 97%. The POC glucose was 27 mg/dL. A stat specimen was also sent to the laboratory. The infant actually received a feeding of formula after this at approximately 1.5 h of age, and another POC was done 30 min after this feeding. As the nurse is calling you about what to do with this baby, the plasma

glucose from the lab is called to the nursery, and it was 37 mg/dL.

Should this baby have received the initial screening bedside POC glucose at 1 h of age before the feedings were initiated?

1. Yes
2. No

Cyanosis is a potential sign or symptom of hypoglycemia with established hypoglycemia (Refer to Signs and Symptoms of hypoglycemia in newborn infants in Hypoglycemia in Newborn Infants: Features Associated with Adverse Outcomes. Rozance PJ and Hay WH: Biol Neonate 2006;90;p 81.) [10]. However, brief cyanosis, associated with transition in the delivery room with rapid resolution, would not be. Apgar scores were not compatible with asphyxia. Therefore, this infant did not meet any of the risk factors to screen for hypoglycemia.

Answer: 2

POC screening tests are often:

1. Higher than values analyzed in the laboratory on plasma samples.
2. Lower than values analyzed in the laboratory on plasma samples.
3. The same as values analyzed in the laboratory on plasma samples.

Bedside screening values of POC glucose are not as accurate as plasma levels from the laboratory and usually are lower. This is particularly true at the lower levels of glucose where accurate data is needed the most [11]. Therefore we use the POC in order to treat glucose levels in symptomatic infants. In asymptomatic infants we can wait for the laboratory glucose for correlation with the bedside POC.

Answer: 2

What is the significance of the POC of 27 mg/dL at 1 h of age prior to feeding and the actual

plasma glucose from the lab of 37 mg/dL for the same sample?

1. It was drawn appropriately and should be acted on.
2. The POC value of 27 mg/dL must be treated with an IV glucose infusion.
3. The laboratory value is 37 mg/dL and the baby requires no treatment.

The bedside screen of 27 mg/dL at 1 h of age is typical of the nadir the glucose can reach at the lower centiles during transitional hypoglycemia and may not accurately reflect the true plasma glucose level. The AAP decided that it did not want to sample the nadir value to decide on management of hypoglycemia. Therefore, the screening was inappropriate. The POC value is low but if accurate is not abnormal since it represents a physiologic nadir. The 37 mg/dL plasma value is the most accurate, is normal, and also represents a physiologic nadir. To be consistent with WHO standards, all newborns should be fed by 1 h of age, and the screen should take place 30 min after the first feed [1].

Answer: 3

The repeat bedside POC at 2 h of age is 37 mg/dL. Is that an actionable level?

1. Yes
2. No

The repeat level of 37 mg/dL at 2 h of age should again be confirmed with a lab level because it is in the actionable range of 25–40 mg/dL. However, none of these levels were necessary in this infant who had no risk factors and was asymptomatic. The treatment for this infant is to let it be treated as a normal newborn and feed frequently.

Answer: 2

The AAP and the PES have provided us with expert opinion about the management of neonatal

hypoglycemia. The PES used neuroendocrine and metabolic data to demonstrate that the first 48 h can be characterized as a transitional hyperinsulinemia with low ketone levels, inappropriate preservation of glycogen, and mean glucose levels of 55–65 mg/dL. By 72 h, levels rise to those observed in older children and adults of >70 mg/dL. The 55–65 mg/dL range is the same level at which adults and older children demonstrate neurogenic symptoms, and this range defines the critical recommendation from the PES for the first 48 h of life [2].

The AAP relied on analyses of the lower range of glucose that occurs during the establishment of postnatal glucose homeostasis and advised actionable ranges of 25–40 mg/dL for the first 4 h of age and 35–45 mg/dL from 4 to 24 h of age. Adamkin and Polin, in a commentary in the *Journal of Pediatrics*, advised using a level of >45 mg/dL for infants between 24 and 48 h of age [3].

By 72 h of age, levels rise to >60–70 mg/dL, similar to older children and adults. The AAP recommendations and the Adamkin and Polin editorial for treatment below or in the actionable ranges for the first 48 h are based on the individual risk assessment and examination of the infant [1, 3]. The AAP believed that these levels provided a margin of safety and flexibility to identify those infants who require intravenous dextrose and those with borderline levels that need to be followed beyond 48 h to prevent missing cases of persistent hypoglycemia. It was thought that this approach would promote successful breastfeeding and avoid unnecessary admission to the NICU in contrast to the PES recommendation of plasma glucose >50 mg/dl for the first 48 h. However, over time the two organizations' recommendations have come closer together. The commitment to try and identify cases of persistent hypoglycemia prior to discharge has been accepted by the AAP, including the "fasting challenge" for some infants with borderline blood glucose levels after 48 h of life and who had been treated with IV dextrose or maintained lower levels of glucose over the first days of life [2].

Case Presentation

A term male infant is born to a 21-year-old primigravida who had no prenatal care. Because of failure to progress, a cesarean section was performed. Apgar scores were 6 and 7 at 1 and 5 min, respectively. The infant weighed 4200 g, and length and head circumference plotted around the 75th percentile on the growth chart. At 6 h of age, the infant appeared lethargic, feeding poorly at the breast and very jittery. A bedside POC was done and was 10 mg/dL. A laboratory specimen was also sent simultaneously.

When should this infant have been screened for hypoglycemia?

1. Immediately after birth.
2. After the first feed (within the first hour of life).
3. 2 h.
4. 4 h.
5. 6 h.

This macrosomic infant with asymmetric macrosomia (suggesting birth to a diabetic mother) should have been screened at the first sign of symptoms and, if asymptomatic, screened 30 min after the first feeding, within 1 h after delivery. Relevant conditions in the pathogenesis of neonatal hypoglycemia include those that cause excess utilization of glucose, such as hyperinsulinism in infants of diabetic mothers, LGA, SGA, islet cell, or other endocrine pathologies. Both IDM and LGA term infants are considered high risk and should be screened according to the algorithm.

Answer: 2

What is the appropriate treatment of this baby?

1. Refeed with breast milk.
2. Refeed with formula.
3. Refeed with D10W.
4. Treat with continuous IV infusion.

5. Treat with a bolus of D10W at 2 mL/kg and IV infusion at 5–8 mg/k/min of D10W (80 mL/k/d).

While the AAP algorithm states that for the asymptomatic baby with low glucose, another feeding could be attempted, this is a severely symptomatic infant that should be treated with intravenous dextrose (bolus followed by continuous infusion) and monitored closely.

Answer: 5

Case Presentation

A 32-year-old gravida 2 delivers a male infant by cesarean section because of failure to progress, a fetal heart rate tracing with fetal distress, and meconium-stained amniotic fluid. Fetal macrosomia was suspected during the pregnancy and the mother had been monitored for hyperglycemia and was treated with insulin for diabetes during the pregnancy. Apgar scores were 4, 6, and 8 at 1, 5, and 10 min, respectively. The infant's birthweight was 4.6 kg. The infant was breastfed in the delivery room and was screened with a bedside POC glucose 30 min after the completion of the breastfeeding. The baby's examination revealed macrosomia, but he was in no distress and was described as quiet but with normal reflexes, tone, and color. The first screening POC glucose was 15 mg/dL; a plasma glucose sent from the same sample was 18 mg/dL. The nurse called the pediatrician and asked what to do next. The decision was to refeed since the baby was asymptomatic, but clearly, concerns were raised about the maternal diabetes and the baby's macrosomia. The baby was refeed, and 30 min later the POC was 20 mg/dL and plasma glucose from the laboratory was 23 mg/dL.

Was it the right decision to refeed this baby after the initial POC of 15 and confirmed with an 18 mg/dL from the lab?

1. Yes
2. No

It was the right decision to refeed after the initial feed when the baby had a POC of 15 mg/dL, which the lab confirmed to be low at 18 mg/dL. Despite a low blood glucose, some babies like the one presented may respond and can be managed enterally. The macrosomia suggests it is unlikely this infant will be manageable with only breastfeedings and supplements, but if the baby looks well and is asymptomatic, this approach can be attempted.

Answer: 1

What do you do next after the second phone call with the laboratory glucose of 20 mg/dL in a baby that is still described as non-distressed but sleepy?

1. Refeed with breast milk.
2. Refeed with formula.
3. Gavage with D10W.
4. Administer 40% glucose gel to the oral mucosa.
5. Treat with a bolus of D10W at 2 mL/kg and/or IV infusion at 5 to 8 mg/k/min of D10W (80 mL/k/d).

This baby needs to go to a site that can provide intravenous fluids and monitor glucose very carefully, particularly if this infant requires hypertonic dextrose for persistent hypoglycemia.

Answer: 5

The baby is transferred to the tertiary care center, and despite increasing IV rates, the infant still has glucose levels below 40 mg/dL at a glucose infusion rate of 16 mg/kg/min. Unfortunately, this infant has a difficult course attaining a normal glucose despite very high glucose infusion rates and is treated with steroids and then octreotide for persistent hypoglycemia. An endocrine work-up for severe hypoglycemia would be necessary if the hypoglycemia persists despite vigorous treatment with intravenous glucose.

Infants with persistent hypoglycemia or those with inadequate response to treatment need

Table 10.1 Causes of recurrent or persistent hypoglycemia [10]

| Hormone deficiencies | |
|---|--|
| Multiple endocrine deficiency or congenital hypopituitarism | Anterior pituitary “aplasia” |
| | Congenital optic nerve hypoplasia |
| Primary endocrine deficiency | Isolated growth hormone deficiency |
| | Adrenogenital syndrome |
| | Adrenal hemorrhage |
| Hormone excess with hyperinsulinism | Beckwith-Wiedemann syndrome |
| | Hereditary defects of pancreatic islet cells |
| Hereditary deficits in carbohydrate metabolism | Glycogen storage disease |
| | Fructose intolerance |
| | Galactosemia |
| | Glycogen synthase deficiency |
| | Fructose, 1–6 diphosphatase deficiency |
| Hereditary deficits in amino acid metabolism | Maple syrup urine disease |
| | Propionic acidemia |
| | Methylmalonic acidemia |
| | Methylmalonic acidemia |
| | Tyrosinosis |
| | 3-OH-3-methylglutaryl-CoA lyase deficiency |
| Hereditary defects in fatty acid metabolism | Acyl-CoA dehydrogenase—medium, long chain |
| | Deficiency |
| | Mitochondrial β -oxidation and degradation defects |

From Cornblath and Ichord [12]

further evaluation. Recurrent or persistent hypoglycemia (Table 10.1) [12] is defined as a condition that [1] requires infusions of large amounts of glucose (>12 to 16 mg/kg/min) to maintain normoglycemia or [2] persists or recurs beyond the first 7 to 14 days of life. These infants require specific diagnostic determinations such as a rapid trial of therapeutic/diagnostic agents to determine the cause and therapy [2].

Hyperinsulinism, hypopituitarism, and fatty acid oxidation disorders are probably the most common of these rarer causes of neonatal hypoglycemia that should be diagnosed before discharge.

An underlying metabolic or hormonal etiology should be suspected when hypoglycemia is

of unusual severity or occurs in an otherwise low-risk infant. Some clues to a possible underlying metabolic-hormonal disorder include:

- Symptomatic hypoglycemia in a healthy, well-grown term infant
- Hypoglycemia with seizures or abnormalities of consciousness
- Persistent or recurrent hypoglycemia
- Hypoglycemia with other abnormalities (midline defects, micropenis, exophthalmos, labile thermoregulation)
- Hypoglycemia requiring greater than 10 mg/kg/min of glucose
- Family history of sudden infant deaths or developmental delays

Clinical Pearls

1. Neonates at risk for hypoglycemia (LGA, IDM, SGA, late preterm) should be screened in the first hour of life (after a feed) for hypoglycemia.
2. Newborns displaying symptoms that could be caused by hypoglycemia need to be responded to immediately. The correct diagnosis must be determined and treatment initiated immediately if hypoglycemia is found.
3. The American Academy of Pediatrics (AAP) algorithm can be used to monitor and treat neonatal hypoglycemia in the first 24 h of life.
4. The recommendations of the Pediatric Endocrine Society (PES) discuss glucose levels after 48 h of life and were developed to heighten awareness of persistent hypoglycemic syndromes in infants with low levels of glucose that do not normalize.
5. There is a gap (24–48 h) not covered by either the AAP guideline or PES recommendations. Maintaining a glucose level > 45 mg/dL from 24 to 48 h of life is recommended.
6. It is critical to identify those with persistent hypoglycemic syndromes to pre-

vent neurologic injury. It is possible that low glucose levels seen in the first 48 h of life may herald metabolic disorders.

7. Neonates diagnosed with hypoglycemia need to achieve a glucose level of >60 mg/dL after 48 h, and levels must be maintained through several fast-feed cycles.

References

1. Adamkin D. Committee on fetus and newborn. Postnatal glucose homeostasis in late-preterm and term infants. *Pediatr*. 2011;127(3):575–9.
2. Thornton PS, Stanley CA, DeLeon DD, Harris D, Haymon MW, Hussain K, et al. Recommendations from the pediatric Endocrine Society for evaluation and management of persistent hypoglycemia in neonate, infants, and children. *J Pediatr*. 2015;167:238–45.
3. Adamkin D, Polin R. Imperfect advice: neonatal hypoglycemia. *J Pediatr*. 2016;176:195–6.
4. Adamkin D. Hypoglycemia. In: Polin R, Yoder M, editors. *Workbook in practical neonatology*. 5th ed. New York: Saunders; 2014. p. 47.
5. Harris DL, Weston PJ, Signal M, Chase JG, Harding JE. Dextrose gel for neonatal hypoglycaemia (the sugar babies study): a randomized, double-blind, placebo-controlled trial. *Lancet*. 2013;382:2077–83.
6. Harris DL, Alsweiler JM, Ansell JM, Gamble GD, Thompson B, Wouldes TA, et al. Outcome at 2 years after dextrose gel treatment for neonatal hypoglycemia: follow-up of a randomized trial. *J Pediatr*. 2016;170:54–9.
7. Hoseth E, Jorgensen A, Ebbesen F, Moeller M. Blood glucose levels in a population of healthy, breastfed, term infants of appropriate size for gestational age. *Arch Dis Child Fetal Neonatal Ed*. 2000;83(2):F117–9.
8. Cornblath M, Hawdon JM, Williams AF, Aynsley-Green A, Ward-Platt MP, Schwartz R, et al. Controversies regarding definition of neonatal hypoglycemia: suggested operational thresholds. *Pediatrics*. 2000;105(5):1141–5.
9. Cornblath M. Reminiscence of a 50 year adventure. *NeoReviews*. 2006;90(2):74–86.
10. Rozance P, Hay W. Hypoglycemia in newborn infants: features associated with adverse outcomes. *Biol Neonate*. 2006;90:74–86.
11. Altimier L, Roberts W. One touch II hospital system for neonates: correlation with serum glucose values. *Neonatal Netw*. 1996;15(2):15–8.
12. Cornblath M, Ichord R. Hypoglycemia in the neonate. *Semin Perinatol*. 2000;24:136–49.