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Directorate General of Khoula Hospital

Pediatric and Neonatal Department

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Table of Content

Acronyms

NICU	Neonatal Intensive Care Unit
PNW	Post-Natal Ward
DGKH	Directorate General of Khoula Hospital
NH	Neonatal Hypoglycemia
EBM	Expressed Breast Milk
TFI	Total Fluid Intake
GFR	Glomerular Filtration Rate
GDH	Glutamate Dehydrogenase Hyperinsulinism.

1. Definitions:

- Transitional hypoglycemia: Brief period of hypoglycemia of less than 2.6 mmol/L within the first 72 hours post-birth. This period is characterized by hypoketotic hyperinsulinemia. Additionally, inappropriately large glycemic responses to glucagon and epinephrine suggest the absence of alternative fuels and the inappropriate preservation of glycogen in a newborn. A neonate above 48 hours should have a blood sugar reading above 3 mmol/L.
- Severe Hypoglycemia: Inability to consistently maintain pre-prandial blood glucose concentration (less than 2.8 mmol/L up to 72 hours and less than 3.3 mmol/L after 72 hrs of age) although being on more than 10–12 mg/kg/min glucose requirement.
- Persistent Hypoglycemia: blood glucose levels less than 3.3 mmol/L beyond the first 72 hours post-birth.

Chapter One:

1. Introduction

Hypoglycemia is a low plasma or blood glucose level in the neonate. In healthy-term neonates, there is a transient, asymptomatic, physiological fall in the blood glucose concentration with a nadir up to 1.4 mmol/L at 60–90 minutes after birth, without any symptoms later rising to levels less than 2 mmol/L by 4 hours. The decrease in glucose concentration soon after birth might stimulate physiological processes required for postnatal survival such as gluconeogenesis and glycogenolysis. It also enhances oxidative fat metabolism, stimulates appetite, and may help adapt to fast-feed cycles. Most neonates compensate for "physiologic" hypoglycemia by producing alternative fuels including ketone bodies, which are released from fat. Breastfed infants may tolerate lower blood sugar levels because of bioavailable alternate fuels like ketone. Clinically, significant neonatal hypoglycemia (NH) most commonly occurs in infants with impaired glycogenesis or ketogenesis, which may occur with excessive insulin production, altered counter-regulatory hormone production, an inadequate substrate supply, or a disorder of fatty acid oxidation. It occurs more frequently in infants who are small or large for gestational age, infants born to mothers who have diabetes, and late preterm infants. While there is agreement that recurrent (more than 3 episodes of less than 2.6 mmol/L) and severe hypoglycemia (less than 2 mmol/L) causes brain injury, the goals are to reduce neurologic impairment due to neonatal hypoglycemia while minimizing overtreatment of neonates with normal transitional low glucose concentrations. Screening protocols and intervention thresholds vary based on the neonate's age and maternal and neonatal risk factors (See appendix 1).

2. Purpose

The purposes of this guideline are to:

2.1 Standardize the approach to newborns who are at risk of hypoglycemia, and those who develop hypoglycemia.

2.2 Ensure early detection and timely intervention to reduce the risk of neurological damage.

2.3 Minimize unnecessary interventions for neonates with normal transitional low glucose level.

3. Scope

This guideline applies to all pediatricians and nurses working in NICU and managing newborns in both NICU & PNW at DGKH.

Chapter Two

4. Structure

It is the guideline of the DGKH to standardize the approach to newborns who are at risk of hypoglycemia, and those who develop hypoglycemia.

4.1 Screening:

Several different clinical thresholds for treatment have been suggested, but a blood glucose concentration less than 2.6 mmol/L is widely accepted as a target for treatment. Concentrations below this may be associated with altered brain function and delayed development. It is important to note that these thresholds are raised to 3.0 mmol/L in infants with suspected hyperinsulinism in the first 48 hours and 3.8 mmol/L after the first 48 hours. Additionally, Formula feeding is associated with increase in glucose concentration within 48 hours after birth compared with no milk, breastfeeding or expressed milk.

4.2 **The following should be followed regarding screening for Neonatal Hypoglycemia:**

a. Routine screening and monitoring of blood glucose concentration is not needed in healthy term newborn infants after an entirely normal pregnancy and delivery.
b. However, all babies at risk should be assessed for criteria for hypoglycemia monitoring prior to leaving a labor ward environment.
c. Hypoglycemia is most likely to occur in the first 24-48 hours of life. Therefore, it is quite safe to discharge these newborns if sugars persistently maintained > 2.6 mmol/L for 24 hours.
d. Monitoring 6 hourly (pre-feed) allows the lowest possible level to be identified. Three groups of neonates merit medical attention:
    i. Neonates with risk factors for hypoglycemia (see Appendix 1 & 2).
    ii. Infants with symptoms or signs of hypoglycemia (see Appendix 3).
    iii. Persistent or refractory hypoglycemia

4.3 Method of screening:

a. Point-of-care testing using reagent strips is rapid, bedside, convenient, and cost-effective.

b. Blood glucose measured by strip is 15% less than plasma glucose

c. In case of persistently low blood glucose, warranting an analysis of the critical sample, a laboratory sample for blood glucose estimation by using one of the laboratory enzymatic methods (glucose oxidase, hexokinase, or dehydrogenase method) should be sent.

d. A delay in testing of sample after collection can lead to a blood glucose reduction of up to 0.3 mmol/L every hour. This problem can be avoided by transporting the blood in tubes that contain a glycolytic inhibitor such as fluoride.

4.2 Prevention of neonatal hypoglycemia: The following should be followed in order to mitigate the risk of NH:

a. Skin-to-skin care.

b. Keep warm (warm clothes and hat).

c. Prior to feeds, check for signs of NH. (See appendix 3).

d. Promote breastfeeding within first hour of life.

e. Infants should continue breastfeeding on cue or not more than 3 hours between feeds.

f. If the baby is not on direct breast feeding, mother should continue to express milk at least 8-10 times per day (including at least once at night).

g. Until the baby is feeding effectively, support to resume breast milk feeds as soon as possible.

h. If the blood glucose level falls to below the acceptable threshold despite frequent breastfeeding together with the use of EBM, an alternative source of energy might be required. This may be achieved by giving infant formula.

i. If giving supplemental formula, in at risk neonates, a volume of 10-15 ml/Kg should be given per feed on day 1.

j. Do not give water or glucose water.

k. To raise blood glucose levels, slow feeding with breast milk or formula using a pump rather than bolus feeding may be considered, particularly when IV access is difficult.

l. Delaying the first bath has been found to reduce incidence of hypoglycemia.

4.4Initial management of neonatal hypoglycemia:

a. Management in the initial three days of life is initiated as per the chart (see Appendix 4).

b. Intravenous fluids management:

i. Target glucose infusion rate of 6 mg/Kg/min.

ii. Glucose infusion rate (mg/Kg/min) is calculated as following:

- Rate (ml/kg/day) / 144 x Glucose%.
- Rate (ml/Kg/day) x 0.007 x Glucose%.

iii. If fluid is restricted, use a higher concentration to maintain GIR of 6 mg/kg/min.

iv. If hypoglycemia persists, increase GIR by 2 mg/kg/min to reach 10-12 mg/kg/min.

i. When on IV fluids, check blood sugars initially every 3 hours until the target is maintained twice, and then every 6 hours.

ii. If severe or persistent neonatal hypoglycemia, refer to point **3.5**

4.4.1Feeding during hypoglycemia:

a. Keeping NPO until the blood sugar is stabilized for two readings.

b. Breastfeeding is usually not included (extra) in the TFI.

c. Bottle feeds might be started on small amounts (e.g. 5-10 ml) every 2 hours (as extra), and slowly to be included in the TFI when the blood sugar readings within normal range.

4.4.2Use of Dextrose gel:

a. Intrabuccal of 0.5 mL/kg of 40% dextrose gel can be used when available.

b. This dose provides 200 mg/kg of glucose, equivalent to an IV bolus of 2 mL/kg of D10 % solution.

c. Dextrose gel should be used with breastfeeding, expressed breast milk, or formula.

d. It should not be used in symptomatic patients.

4.5 Approach to severe and persistent neonatal hypoglycemia:

a. In both severe and persistent neonatal hypoglycemia, hyperinsulinism (see Appendix 5) should be ruled out.

b. Critical samples to be collected before correction of hypoglycemia if:

i. blood sugar less than 2.8 mmol/L

ii. GIR more than 10-12 mg/Kg/min.

iii. Hypoglycemia persists for more than 48 hours.

c. Critical sample should include the following:

i. Confirmatory plasma glucose level.

ii. Insulin: Usually is undetectable. Level of more than 2mU/ml confirms hyperinsulinism.

iii. Growth hormone level (in selected cases): During hypoglycemia, the level is usually more than 10 ng/ml.

iv. Cortisol level: Usually more than 550 nmol/L during hypoglycemic event.

4.6 In addition to the above investigations, the following should be considered in non-conclusive results or when clinically suggestive:

a. Urine Ketone.

b. Urine-reducing substances: Positive in Galactosemia.

c. Lactate more than 2 mmol/L might be suggestive of Glycogen storage disease Type 1.

d. Metabolic screening including acylcarnitine profile to rule out fatty-acid-oxidation defect and organic academia.

e. Blood gas guides the approach to metabolic disorders.

f. Electrolytes might suggest congenital adrenal hyperplasia.

g. Ammonia – Elevated in Glutamate Dehydrogenase Hyperinsulinism (GDH).

h. Approach Persistent Neonatal Hypoglycemia, see Appendix 6& Appendix 7.

4.7 Management of severe and persistent Neonatal Hypoglycemia:

a. Initially, increase total fluids intake by 20 ml/Kg/day to the maximum allowed fluid intake (Maximum in the initial 24 hours is 100 ml/Kg/day). If still sugars are not controlled, increase the concentration of dextrose.

b. Concentrations greater than 12.5% must be administered using a central line.

c. If patients continue to experience hypoglycemia episodes (less than 3.3mmol/L) with a glucose infusion rate of approximately 20 mg/kg/min, additional therapeutic options may need to be explored.

d. It may be prudent to delay additional therapies as long as possible (up to 7 days of life) to allow the transient forms of hypoglycemia to resolve in order not to expose infants to unnecessary drug therapy.

e. The following medications might be considered:

4.7.1 Glucagon:

a. It stimulates gluconeogenesis and glycogenolysis.
b. It is given via IV bolus or infusion.
c. Intramuscular or subcutaneous injection can be considered if establishing IV access is difficult or not feasible.
d. Infusion of glucagon is preferred because it prevents an exaggerated stimulation of the pancreas due to a high glucose infusion rate and it does not interfere with the effective establishment of breastfeeding.
e. The glucose level rise within one hour upon administration and last, approximately, up to two hours.
f. Side effects include hyponatremia, thrombocytopenia and erythema necrolytic migrans.
g. Hypoglycemia non-responsive to glucagon may be provoked in glycogen storage disease.

4.7.2Doses of Glucagon:

a. Bolus (IV, IM or subcutaneously): 40 mcg/Kg (ranging from 200 mcg/kg to as low as 30 mcg/kg).
b. Infusion: 10-50 mcg/Kg/Hr

4.7.3Hydrocortisone:

a. It Stimulates gluconeogenesis and reduces glucose utilization in peripheral tissues.
b. It has a slower response than glucagon.
c. It may be preferred when:
   i. Hyponatremia is suspected.

ii. Infant is hypotensive.

iii. Evidence indicative of hypoadrenalism is present.

iv. Response to previously administered glucagon is insufficient.

d. Dose of hydrocortisone 4 mg/Kg/day which is divided in to 4-6 doses (Might be increased up to 15 mg/Kg/day in selected cases).

4.7.4 Diazoxide:

a. It is a potassium channel activator. It inhibits pancreatic insulin release.

b. It is used in cases of persistent hypoglycemia as long-term management, and to facilitate weaning from glucose infusion.

c. The action is within 1 hour of administration, with the duration of action of 8 hours, assuming normal renal function.

d. When effective, hypoglycemia normalizes within 2 to 4 days of therapy initiation. However, because of variations in kinetic parameters, a trial of 5 to 8 days is required before judging therapy a failure.

e. Dose: Typically initiated at 5 to 15 mg/ kg/day orally in 2 to 3 divided doses.

f. Side effects: The most commonly reported side effect associated with diazoxide use is hypertrichosis. It also causes sodium retention and fluid overload especially in babies with structural cardiac defects.

g. Failure to respond to a trial of diazoxide therapy would suggest dysfunction associated with the KATP receptor, at which point a trial of octreotide is suggested as it works more distally in the insulin secretory pathway.

4.7.5 Hydrochlorothiazide:

a. It is a diuretic, which has a mechanism of action like the one of diazoxide.

b. It is usually used in conjunction with diazoxide to reduce its side effects.

4.7.6 Octreotide:

a. It is a pharmacological analog to natural somatostatin.

b. It is usually recommended for known or suspected cases of hyperinsulinism

c. Dose:

i. Intermittent dosing of octreotide should be administered for 1 to 2 hours following selected feeds. Doses may be given with every other feed.

ii. Dose starts subcutaneously at 5 mcg/kg/dose every 6 to 8 hours and is titrated up to 40 mcg/kg/day.

iii. If patients do not remain euglycemic with intermittent dosing, octreotide may be administered via continuous infusion in hypoglycemia.

d. Side effects: Transient malabsorption, reduction in growth, and occasional cholestasis is reported.

f. In resistant cases, babies might require a combination of Octreotide and Glucagon.

4.8 When medical therapy fails to maintain the glucose level in a safe range.

a. Surgical intervention is proposed for neonates with hyperinsulinemic hypoglycemia.

b. Nutritional therapy is advised especially for disorders of glycogen metabolism or hereditary fructose intolerance.

4.9 Preparation for discharge of neonates with Severe and Persistent Neonatal Hypoglycemia

a. Maintenance of glucose levels 3.8 mmol/L and more at 4 and 5 hours post feed should be documented before discharge is considered.

b. An underlying diagnosis for neonatal hypoglycemia should be ascertained and initiated on specific medical management in preparation for care at home.

c. Parents should be counseled regarding frequency of feeding, home blood glucose monitoring, medication delivery (if needed), or other treatment measures for hypoglycemia.

Chapter Three

5.Responsibilities :

5.1 **HOD of pediatrics and neonatology shall:**

5.1.1 Ensure that all pediatricians are aware of this guideline and emphasize the importance of following it.

5.1.2 Ensure that all pediatricians adhere to this guideline.

5.2 **Pediatricians shall:**

5.2.1 Follow and adhere to this guideline.

5.3 **The NICU nursing in- charge shall:**

5.3.1 Ensure that all NICU nursing staff are aware of these guideline

5.4 **Nursing staff shall:**

a) Adhere to this guideline.

Chapter Four:

5 Document history and version control table:

Version	Description	Author Name	Review Date
1	Initial release	Dr. Vidiya Ramdas	2027

6 References:

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7 Annexes:

7.1Appendix (1): Risk Factors for Neonatal Hypoglycemia

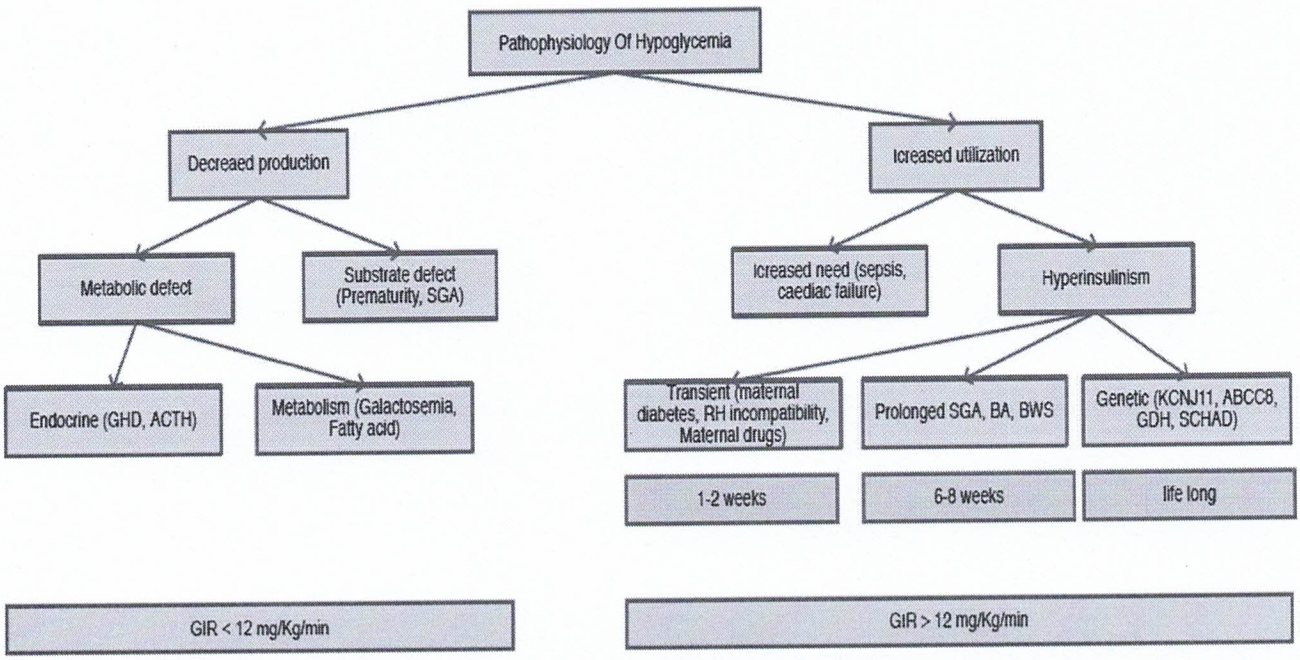
Newborn risk factors:
- Preterm <37 weeks' gestation - Infection - Asphyxia - Hypothermia - IUGR (<10th centile for gestational age) - Low birth weight (less than 2500 gm) - Macrosomia/Large for gestational age birth weight >4 kg (>90th centile for gestational age) - Cardio-Respiratory problems: e.g.: RDS, TTN, congenital heart disease - Polycythemia - Severe Rhesus disease - Midline defects - Endocrinopathies: Cortisol deficiency, Growth hormone deficiency, Hypothyroidism - Inborn errors of metabolism e.g., fatty acid oxidation disorders, glycogen storage diseases - Hyperinsulinism: Insulin-secreting tumors-Persistent Hyperinsulinemia of infancy, congenital hyperinsulinism - Beckwith Wiedemann Syndrome - Mosaic Turner syndrome, Costello syndromes
Maternal risk factors
- Maternal diabetes mellitus or impaired maternal glucose tolerance - Intrapartum administration of glucose - Maternal drugs: Oral hypoglycemic agent, beta-blockers such as Labetolol, Valproate

7.2 Appendix (2): Pathophysiological Classification of Neonatal Hypoglycemia

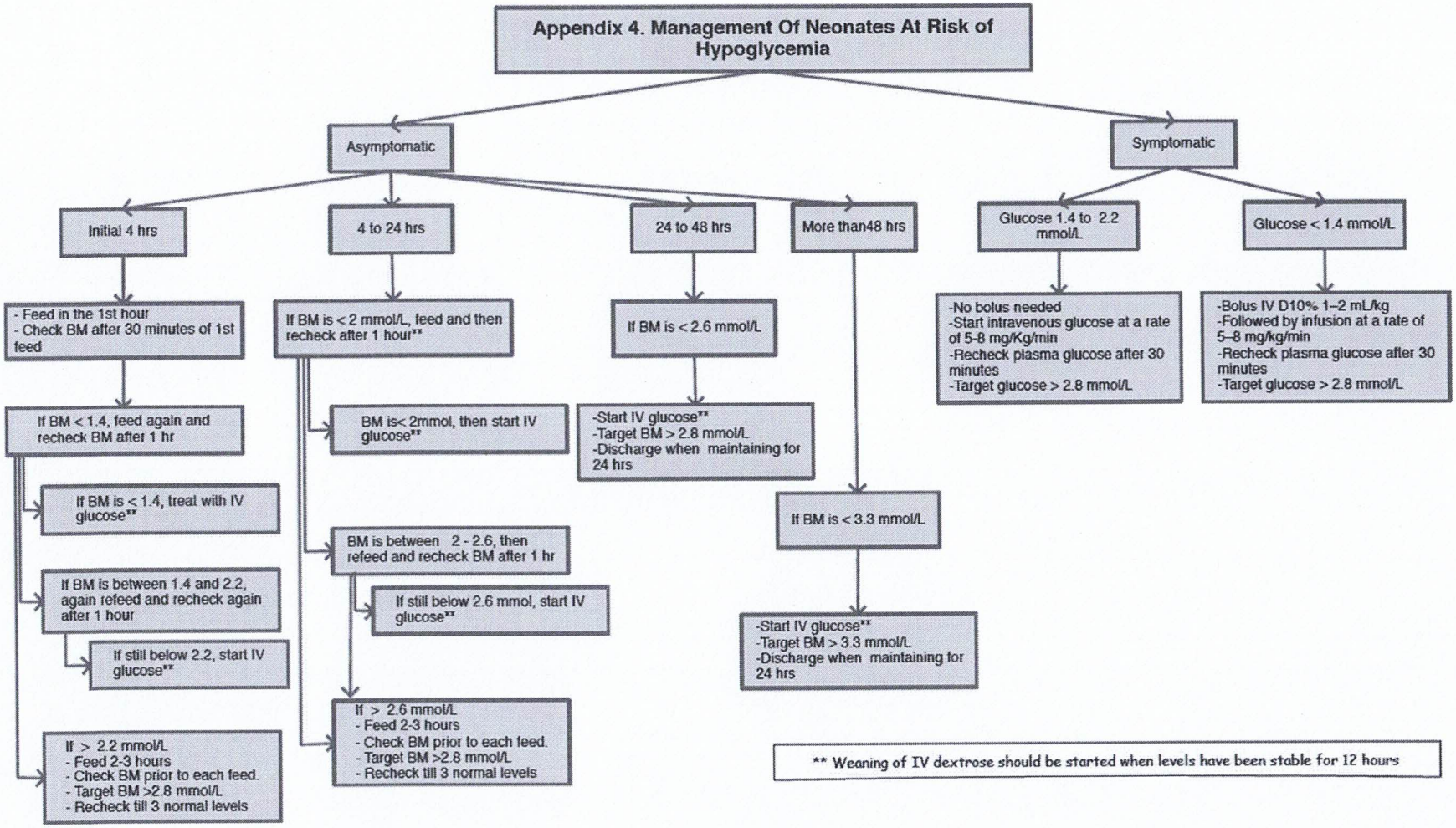
Decreased Production/Stores	Increased Utilization
- Poor glucose storage/supply/production 1. Poor oral intake (or delayed feeding) 2. Prematurity 3. Intra-uterine growth restriction - Inborn errors of metabolism 1. Endocrine abnormalities 2. Growth hormone deficiency 3. Cortisol deficiency	- Infant of diabetic mother - Large for gestational age - Sepsis - Asphyxia - Hypothermia - Polycythemia - Hyperinsulinemia - Maternal beta blockers - Oral hypoglycemic

7.3 Appendix (3): Clinical Signs of Neonatal Hypoglycemia

Adrenergic	Neuroglycopenic
- Tachypnea - Tachycardia - Poor suck - Poor feeding - Pallor - Temperature instability	- Lethargy and hypotonia - Apnea or irregular breathing -Cyanosis -Seizures



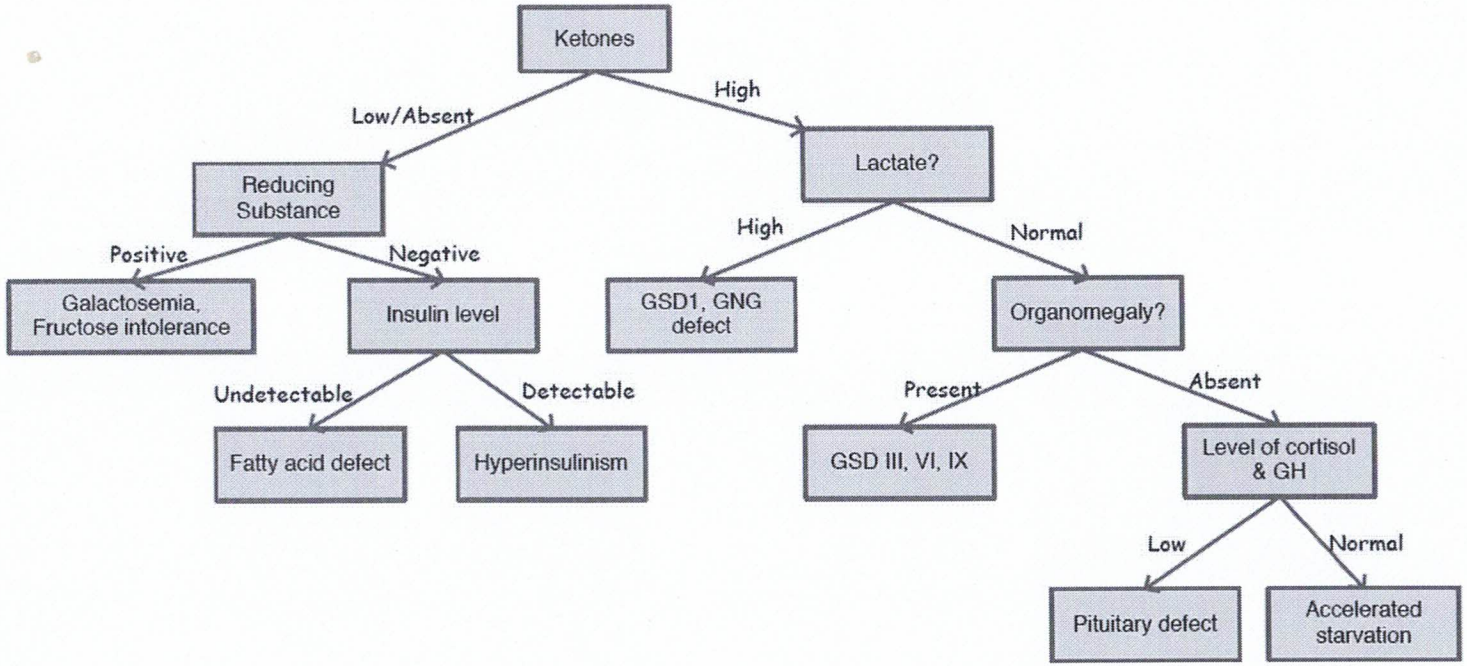
Appendix 4. Management Of Neonates At Risk of Hypoglycemia



8.5 Appendix (5): Classification of Hyperinsulinism

Transient	- Associated with: maternal diabetes, maternal intake of drugs,& RH incompatibility - resolve by 1-2 weeks
Prolonged	- Associated with: SGA, preterm, LGA, birth asphyxia (due to stress related increased pancreatic insulin production), & BWS - Resolved by 6-8 weeks
Persistent	- Due to K channel defects (KCNJ11 / ABCC8 / GDH/ S) - Usually macrosomic - Severe & life long - Resistant to diazoxide

8.6 Appendix (6): Approach to Persistent Neonatal Hypoglycemia



8.7 Appendix (7): Tips to Approach to Persistent Neonatal Hypoglycemia

Category	Feature/s	Differential
Clinical Signs	SGA	Hyperinsulinism
	Macrosomia	- Antenatal insulin excess - Beckwith Weidman Syndrome - Potassium channel mutations
	Normal birth weight	- GCK -GDH - Focal form of hyperinsulinism
	Organomegaly	- Galactosemia - Glycogen storage disorders - Hyperinsulinism - BWS
	ambiguous genitalia	DSD
	Omphalocele	BWS
	Cataract	Galactosemia
	Cholestasis	- Hypopituitarism - Galactosemia
	Onset after feed	- Organic academia - Galactosemia
	Nystagmus	Septo-optic dysplasia associated with HESX 1 defect
	Midline defects, Micropenis, cholestasis	- Multiple pituitary hormone defects - Adrenal hyperplasia congenita - DAX 1 defect
	Hyperpigmentation, hyponatremia, hypokalemia, metabolic acidosis	- Adrenal insufficiency - Congenital adrenal Hyperplasia
Investigations	Ketone –ve Urine reducing Substance +ve	Galactosemia (Cataract)
	Ketone –ve Urine reducing Substance -ve	- Hyperinsulinism (GIR >12 mg/Kg/min) - FAO defect (GIR not high)
	Ketone +ve Normal Lactate	Multiple pituitary hormone deficiency (No organomegaly, micropenis, midline defects, cholestasis)
	Ketone +ve High lactate	Glycogen storage disease (Presentation is rare in neonatal period, Organomegaly)
GIR	Normal (reduced insulin production)	- SGA - Premature neonate - Multiple pituitary hormone deficiency
	High (Increased utilization)	Hyperinsulinism