South Australian Paediatric Clinical Practice Guidelines

Managing Unexplained Hypoglycaemia in Children

(excluding diabetic and neonatal patients)

Note:

This guideline provides advice of a general nature. This statewide guideline has been prepared to promote and facilitate standardisation and consistency of practice, using a multidisciplinary approach. The guideline is based on a review of published evidence and expert opinion.

Information in this statewide guideline is current at the time of publication.

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Health practitioners in the South Australian public health sector are expected to review specific details of each patient and professionally assess the applicability of the relevant guideline to that clinical situation.

If for good clinical reasons, a decision is made to depart from the guideline, the responsible clinician must document in the patient's medical record, the decision made, by whom, and detailed reasons for the departure from the guideline.

This statewide guideline does not address all the elements of clinical practice and assumes that the individual clinicians are responsible for discussing care with consumers in an environment that is culturally appropriate and which enables respectful confidential discussion. This includes:

- The use of interpreter services where necessary,
- Advising consumers of their choice and ensuring informed consent is obtained,
- Providing care within scope of practice, meeting all legislative requirements and maintaining standards of professional conduct, and
- Documenting all care in accordance with mandatory and local requirements

Explanation of the aboriginal artwork:

The aboriginal artwork used symbolises the connection to country and the circle shape shows the strong relationships amongst families and the aboriginal culture. The horse shoe shape design shown in front of the generic statement symbolises a woman and those enclosing a smaller horse shoe shape depicts a pregnant woman. The smaller horse shoe shape in this instance represents the unborn child. The artwork shown before the specific statements within the document symbolises a footprint and demonstrates the need to move forward together in unison.

Cultural safety enhances clinical safety.

To secure the best health outcomes, clinicians must provide a culturally safe health care experience for Aboriginal children, young people and their families. Aboriginal children are born into strong kinship structures where roles and responsibilities are integral and woven into the social fabric of Aboriginal societies.

Australian Aboriginal culture is the oldest living culture in the world, yet Aboriginal people currently experience the poorest health outcomes when compared to non-Aboriginal Australians.

It remains a national disgrace that Australia has one of the highest youth suicide rates in the world. The over representation of Aboriginal children and young people in out of home care and juvenile detention and justice system is intolerable.

The cumulative effects of forced removal of Aboriginal children, poverty, exposure to violence, historical and transgenerational trauma, the ongoing effects of past and present systemic racism, culturally unsafe and discriminatory health services are all major contributors to the disparities in Aboriginal health outcomes.

Clinicians can secure positive long term health and wellbeing outcomes by making well informed clinical decisions based on cultural considerations.

The term 'Aboriginal' is used to refer to people who identify as Aboriginal, Torres Strait Islanders, or both Aboriginal and Torres Strait Islander. This is done because the people indigenous to South Australia are Aboriginal and we respect that many Aboriginal people prefer the term 'Aboriginal'. We also acknowledge and respect that many Aboriginal South Australians prefer to be known by their specific language group(s).

Government of South Australia

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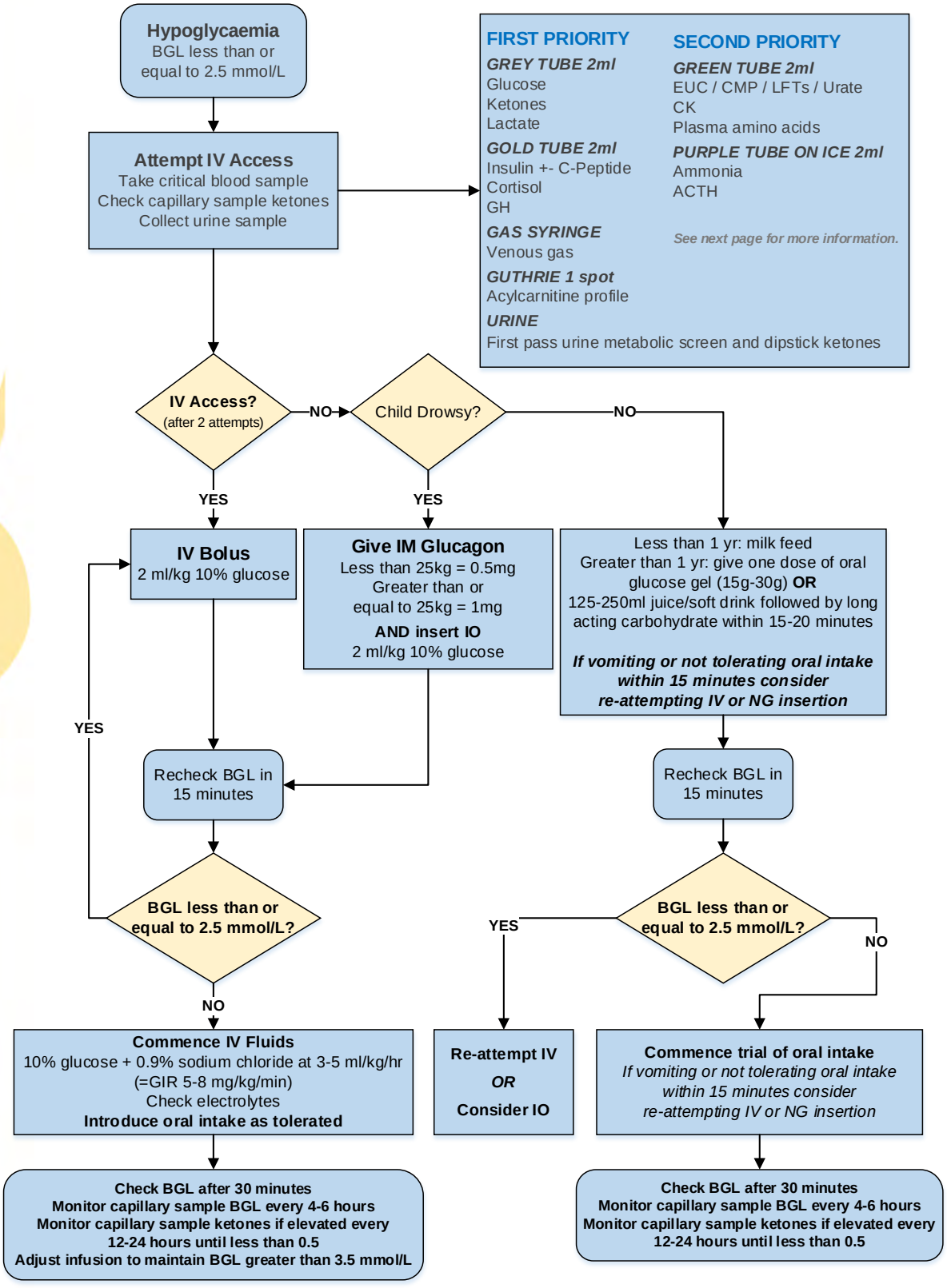
Flowchart



Purpose and Scope of PCPG

This guideline is intended to provide healthcare workers with a clear clinical approach for the initial assessment, investigation and management of infants or children who present with hypoglycaemia.

This guidelines excludes neonates, patients with diabetes or patients with a known metabolic disorder who have a special needs file. Please see relevant guidelines for these conditions.

Table of Contents

Important Points

- Hypoglycaemia is defined as blood glucose level (BGL) low enough to cause signs and symptoms of impaired brain function[1].
- A BGL less than or equal to 2.5 mmol/L requires investigation and treatment regardless of the presence of symptoms.
- If hypoglycaemia is not promptly recognised in children, they may develop symptoms and suffer permanent neurological sequelae[2].
- Investigations are important in the identification of an underlying diagnosis but should not delay management.
- A patient with a BGL less than or equal to 2.5 mmol/L OR a low BGL with altered conscious state or seizures requires prompt management with a bolus of 2ml/kg IV 10% glucose or IM glucagon.
- All presentations of hypoglycaemia, including those at rural and remote locations, should be discussed with an on call paediatrician at the appropriate Local Health Network.

Abbreviations

AA	acetoacetate
ACTH	adrenocorticotropic hormone
BGL	blood glucose level
BOHB	beta-hydroxybutyrate
CK	creatine kinase
CMP	calcium, magnesium, phosphate
EUC	electrolytes, creatinine, urea
FAOD	fatty acid oxidation disorder
FFA	free fatty acids
GH	growth hormone
GIR	glucose infusion rate
GSD	glycogen storage disorder
IM	intramuscular
IO	intraosseous
IV	intravenous
LFTs	liver function test
NG	nasogastric

Definitions

Normoglycaemia	Normal fasting glucose is kept within a narrow physiological range of 3.5-5.5 mmol/L (other than in the first few days of life)[3].
Hypoglycaemia	Clinical hypoglycaemia is defined as BGL low enough to cause signs and symptoms of impaired brain function. The BGL threshold for signs of cognitive impairment may vary depending on the presence of alternative fuels such as ketones or lactate[1].
BGL	A BGL of **< 2.5 mmol/L** is generally regarded as the level at which prompt investigation and treatment should occur regardless of the presence of symptoms. Neurogenic symptoms generally occur at BGL < 3.0 mmol/L and cognitive function is impaired at BGL < 2.8 mmol/L[4].
Hypoglycaemia Associated Autonomic Failure	Previous exposure to an episode of hypoglycaemia can blunt neurogenic responses to subsequent hypoglycaemic episodes. This can persist for > 24 hours after a single episode of hypoglycaemia and longer after repeated episodes[1].

Critical Hypoglycaemia Investigations

TUBE TYPE		PHOTO	INVESTIGATIONS	VOLUME
FIRST PRIORITY INVESTIGATIONS - ***essential to be taken prior to treatment***				
Fluoro-oxalate	**Grey** pedi/ adult tube		Glucose Lactate B-hydroxybutyrate Acetoacetate Free fatty acids	2 ml *min 1.2ml*
Serum	**Gold** pedi-tube White OR Gold adult tube		Insulin / C-Peptide* Cortisol Growth Hormone	2 ml *min 1.5ml*
Gas syringe	Gas syringe or capillary tube (can be capillary)		Acid-Base	0.1ml
Guthrie Card	Newborn screening card (can be capillary)		Acylcarnitine Profile	1 spot
SECOND PRIORITY INVESTIGATIONS - ***can be taken prior or after treatment***				
Lithium Heparin	**Green** pedi/adult tube		Plasma amino acids EUC / CMP/ LFTS CK Urate	2 ml *min 1.1 ml*
EDTA	**Purple** pedi/adult tube		Ammonia **ON ICE, urgent to lab** ACTH **ON ICE**	2 ml *min 0.8ml*
FIRST URINE COLLECTION				
Collect the first pass urine (5-20ml) for urine amino acids / organics acids and dipstick urine ketones regardless of age and time since hypoglycaemia episode. *Urine can be collected via in and out catheterisation, clean catch or pedi-bag.* *Urine should not be collected with cotton balls.*				
CAPILLARY SAMPLE KETONES				
Measure capillary sample ketones on arrival, and if elevated, at regular intervals 12-24 hourly (in consultation with senior paediatric staff) to ensure resolution.				

Table 1: Critical Hypoglycaemia Investigations.

**C-Peptide if high clinical suspicion of hyperinsulinism or exogenous insulin.*

Background

Hypoglycaemia is not uncommon in children and requires prompt management to prevent brain injury. The brain has an obligate requirement for glucose but can use ketones or lactate as energy sources if required. Children have higher glucose utilisation rates per kg compared to adults due to their disproportionately larger brain size to body mass[1].

The maintenance of plasma glucose levels requires the complex interaction of multiple endocrine and metabolic pathways.

Glucose Homeostasis

- In the fed state:
  - After a meal, blood glucose levels rise and insulin is secreted from pancreatic B-cells
  - Insulin increases glucose uptake into skeletal muscle and adipose tissue
  - Insulin promotes glycogen synthesis and inhibits both glycogenolysis and gluconeogenesis.

- In the fasted state:
  - Insulin secretion is suppressed
  - Glucagon is secreted which promotes glucose production from glycogen in the liver by ***glycogenolysis***
  - Adreno-cortical axis is activated which increases cortisol, growth hormone and adrenaline levels which assist in regulating glucose levels
  - Glucose is produced by ***gluconeogenesis*** from amino acids, glycerol and lactate
  - Adipose tissue releases glycerol and free fatty acids
  - Free fatty acids are converted into ketones via ***fatty acid oxidation***
  - The brain can use ketones and lactate as its alternative energy source[4]

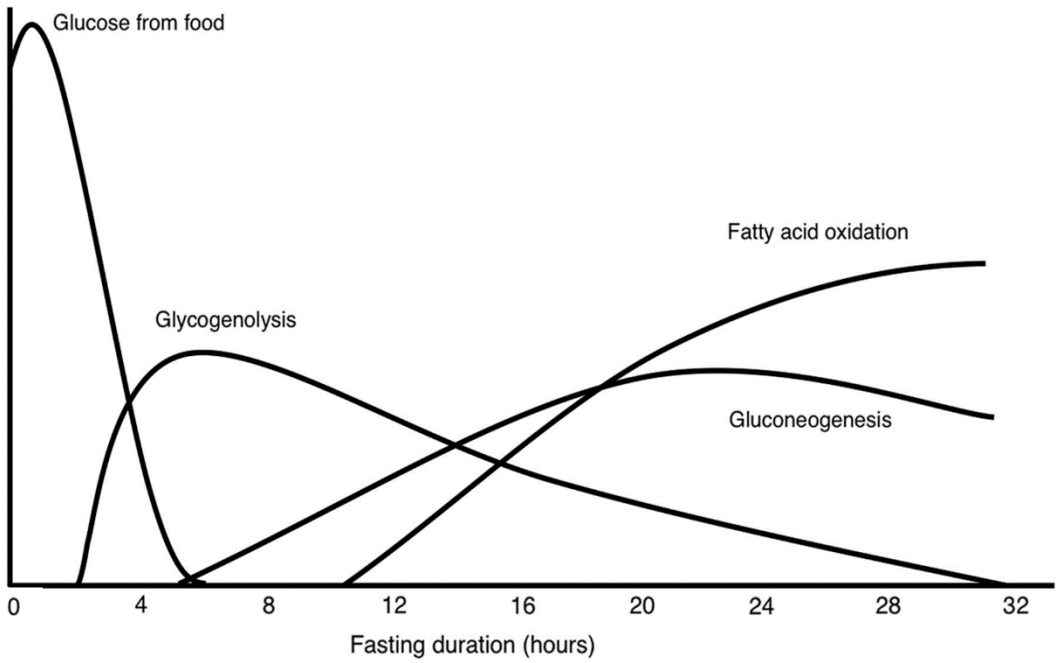


Figure 1: Energy sources during fasting[4]

Causes

Hypoglycaemia is a feature of many inherited metabolic and endocrine disorders. It can also occur in children who are otherwise well. Idiopathic Ketotic Hypoglycaemia is the most common cause of hypoglycaemia in healthy children between the ages of 18 months and 5 years of age and spontaneously resolves by 8-10 years of age. Idiopathic Ketotic Hypoglycaemia is a diagnosis of exclusion so it is important to consider other possible diagnoses.

When considering the differential diagnosis the questions to ask are:

- Is this ketotic or hypoketotic?
  - Hypoketotic: Hyperinsulinism, fatty acid oxidation disorders and some disorders of ketone metabolism
- Is the hypoglycaemia appropriate for the degree of fasting or 'metabolic stress'
- Does the child have any clinical features to suggest this is not idiopathic ketotic hypoglycaemia?[5]
- Could the child have sepsis?

CAUSES OF HYPOGLYCAEMIA	
ENDOCRINE	Hyperinsulinism - congenital, insulinoma Adrenal Insufficiency Growth Hormone deficiency Hypopituitarism Dumping Syndrome Hypothyroidism (very rarely)
METABOLIC	Glycogen Storage Disorder Fatty Acid Oxidation Disorders Disorders of Gluconeogenesis Disorders of carbohydrate metabolism Disorders of ketone metabolism Organic acidaemias Mitochondrial respiratory chain defects
SYSTEMIC	Idiopathic Ketotic Hypoglycaemia Illness - sepsis, gastroenteritis Starvation / Malnutrition Eating Disorders Liver Disease
TOXINS	Medications - insulin, sulphonylureas, salicylates, beta-blockers, chemotherapy Alcohol Methanol

Table 2: Causes of Hypoglycaemia[1,3,6]

Assessment

Given the wide differential diagnosis, a thorough history and examination is important to identify other precipitating causes that need further investigation.

CLINICAL FEATURES OF HYPOGLYCAEMIA	
AUTONOMIC FEATURES	**NEUROGLYCOPAENIC FEATURES**
Adrenergic Response > Palpitations > Tremor > Anxiety	> Lethargy > Irritability > Weakness > Confusion > Headaches > Dizziness > Hypothermia > Seizure > Coma
Cholinergic Response > Sweating > Hunger > Nausea/Vomiting > Abdominal pain	

Table 3: Clinical Features of Hypoclycaemia [1,2]

History

History of event:

- Acute illness: vomiting, diarrhoea, fever
- Diet History
  - Time from last meal (event in fed or fasted state)
  - What meal was eaten
    - Milk products (galactosaemia)
    - Fructose (hereditary fructose intolerance)
    - Protein (amino acid or organic acid disorders)
- Ingestion: ingestion of alcohol, oral hypoglycaemic agents, aspirin, beta blocker
- History of intercurrent illness
- History of feeding and overnight fasting when well

Past Medical History

- Perinatal history: birth weight, gestational age, neonatal hypoglycaemia
- Past history of hypoglycaemia or episodes that may have be missed or diagnosed as other conditions (e.g seizure disorder)
- Previous gastric surgery, fundoplication (postprandial hypoglycaemia)

Family history

- Affected family members with hypoglycaemic episodes
- Unexplained or sudden deaths in infants or children
- Consanguinity (parents share common ancestry)[1,2]

Examination

- Conscious State
- Vital signs: tachypnoea may be due to metabolic acidosis in some inborn errors of metabolism, fever /hypothermia in sepsis
- Growth: Assessment of weight and length on growth centiles to estimate growth trajectory
  - Short stature: growth hormone deficiency as clue to possible hypopituitarism
  - Underweight for age: may be at risk of ketotic hypoglycaemia
- Midline facial defects: hypopituitarism
- Hepatomegaly: Glycogen storage disorder, defects in gluconeogenesis, galactosaemia
- Hyperpigmentation: adrenal insufficiency
- Hemihypertrophy, macroglossia, omphalocele: Beckwith Wiedemann[1,2]

Investigations

Critical investigations should be taken in patients presenting with their first episode of hypoglycaemia with a BGL less than or equal to 2.5 mmol/L, a severe episode of hypoglycaemia or any patient with unexplained hypoglycaemia who shows signs/symptoms of impaired brain function.

A capillary glucometer reading is unreliable at low readings; hence it is important to confirm that true (laboratory) BGL is less than or equal to 2.5 mmol/L. However, treatment should not be delayed while waiting for laboratory glucose[1].

Investigations should ideally be taken prior to the treatment of hypoglycemia unless it will cause significant delay to management.

If a patient is normoglycemic after treatment prior to presentation, all investigations should be completed in a timely manner and labelled as “post treatment” for ease of interpretation.

Please see Table 1 for more information on critical investigations.

First Priority Investigations

In order of priority:

- Glucose
- Insulin / C-Peptide (C-peptide if high clinical suspicion of hyperinsulinism or exogenous insulin)
- Ketones (acetoacetate, B-hydroxybutyrate)
- Cortisol
- Acid Base (including lactate)
- Growth Hormone
- Acylcarnitine profile
- Urine metabolic screen (the first urine passed after the episode of hypoglycaemia is the CRITICAL SAMPLE for measurement of organic acids and ketones)
- Urine collected (avoid cotton ball collection as can interfere with organic acid and amino acid analysis).

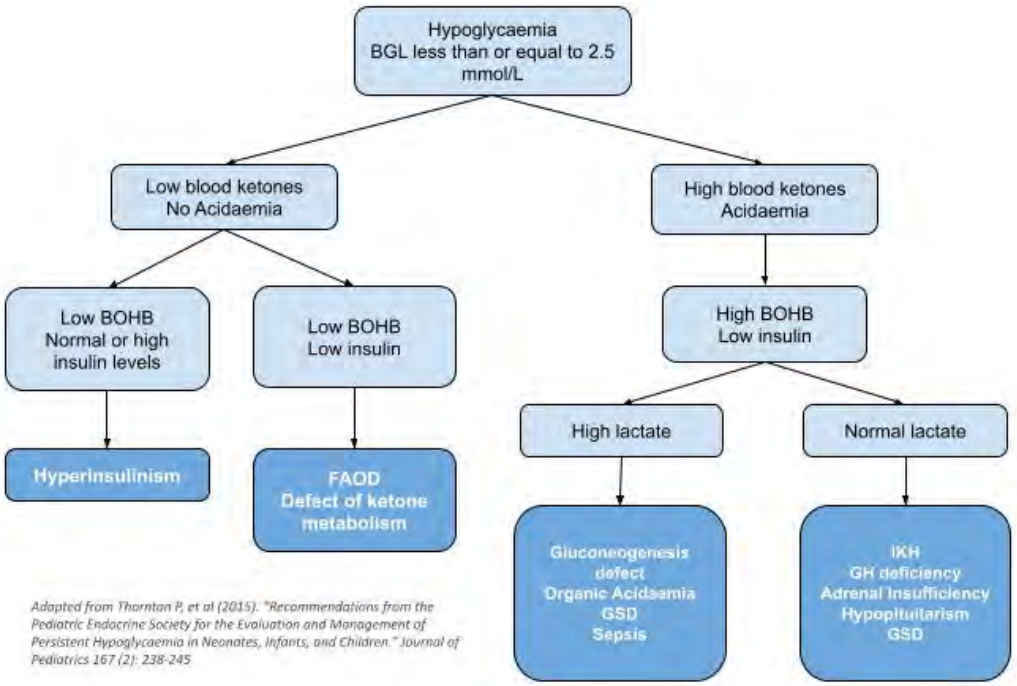
Second Priority Investigations

In order of priority:

- EUC / LFTS / CMP / Urate
- CK
- Plasma amino acids
- Ammonia
- ACTH

Note: Free fatty acids are no longer required in acute unexplained hypoglycaemia as they have limited diagnostic value and will not be processed in South Australian laboratories from 2022.

Interpretation of Investigations



Adapted from Thornton P, et al (2015). "Recommendations from the Pediatric Endocrine Society for the Evaluation and Management of Persistent Hypoglycaemia in Neonates, Infants, and Children." Journal of Pediatrics 167 (2): 238-245

INVESTIGATION	INTERPRETATION
Insulin / C-Peptide	Inappropriately normal or raised insulin during hypoglycaemia may indicate hyperinsulinism. Given the short half life of insulin (5-6 mins) C-peptide may be useful in determining hyperinsulinism (C-peptide will be high) or if there has been exogenous insulin administration (C-peptide will be low).
Ketones	Hypoketosis may indicate hyperinsulinism, fatty acid oxidation disorder (FAOD) or disorder of ketone metabolism B-Hydroxybutyrate (BOHB) is the ketone measured in capillary sample blood testing. Acetoacetate is the ketone measured in urine.
Blood gas	Metabolic acidosis may indicate ketoacidosis, lactic acidosis or organic acidaemia.
Lactate	High lactate may indicate sepsis, glycogen storage disorder, gluconeogenesis disorders, mitochondrial disorder or may occur following hypoglycaemic seizure
Cortisol	Should be elevated in context of stress response to hypoglycaemia. Inappropriately normal or low in context of hypoglycaemic episode may indicate hypopituitarism or adrenal insufficiency. Cortisol response is commonly blunted in hyperinsulinism also.
Growth Hormone	Inappropriately low or low normal may indicate isolated GH deficiency or hypopituitarism.
Acylcarnitine profile	Specific acylcarnitine species will be raised in fatty acid oxidation disorder or organic acidaemia A specific pattern of acylcarnitines will be observed as a normal response to fasting or metabolic stress
Electrolytes	Abnormal electrolytes may occur in adrenal insufficiency or dehydration
Liver function tests	Abnormal in liver disease or some metabolic disorders such as glycogen storage disorders
CK	May be elevated in fatty acid oxidation disorders or glycogen storage disorders
Urate	May be elevated in glycogen storage disorders
Ammonia	Increased in liver disease, organic acidaemia, hyperinsulinism-hyperammonaemia (HIHA) disorder
ACTH	Inappropriately low in hypopituitarism or inappropriately high (without appropriate cortisol response) Addison's Disease (primary adrenal insufficiency)
Plasma amino acids	Raised ketogenic (branched chain) and gluconeogenic amino acids are seen in Idiopathic Ketotic Hypoglycaemia. Generalised low plasma amino acids are noted in patients with poor reserves. Specific amino acid patterns will be present in some metabolic disorders.
Urine Organic Acids	Ketone bodies and dicarboxylic acids are seen in the normal response to fasting hypoglycaemia. Specific organic acids will be present in some metabolic disorders at the time of hypoglycaemia.

Table 4: Interpretation of Investigations[3,7,]

Management

Initial Treatment

IV access

- IV bolus 10% glucose 2ml/kg
- Recheck capillary BGL at 15 minute intervals until BGL stable ≥ 2.5 mmol/L

No IV access

- If child awake, not drowsy:
  - < 1 year: give milk feed
  - > 1 year give oral glucose gel (15-30g) or 125-250ml of juice/soft drink followed by complex carbohydrate (1 slice of bread or banana)
- Child drowsy:
  - IM glucagon (≤ 25kg: 0.5mg, ≥ 25mg: 1mg)

  AND
  - Insert intraosseous needle and give 10% glucose 2ml/kg

Recheck BGL within 15 minutes

If following initial treatment child is unable to recommence feeds, is vomiting or remains unwell:

- Start IV glucose infusion using 10% glucose / 0.9% sodium chloride at a rate of 3-5 ml/kg/hr (equivalent to giving 5-8 mg/kg/min glucose)
  - *A 5% glucose infusion is usually NOT sufficient to maintain BGL or clear ketones*
- If IV access is unable to be obtained and there are no contraindications (i.e. drowsy or encephalopathic), can consider nasogastric insertion and administration of feeds (breast milk, formula, polyjoule, 10% glucose) at maintenance rates.
- Recheck BGL at 30 minute intervals until BGL stable > 3.5 mmol/L

If BGL remains below 2.5 mmol/L

- Repeat bolus of 10% glucose 2ml/kg
- Increase IV glucose infusion rate to 6ml/kg/hr 10% glucose / 0.9% sodium chloride (10 mg/kg/min glucose)
- If BGLs remain low, increase the concentration of glucose in IV fluids - note for glucose concentrations > 12.5% central access is required and should be discussed with paediatric metabolic or endocrine specialists.
- Monitor electrolytes regularly

To make 10% glucose + 0.9% sodium chloride

Use 1L bag of 5% glucose + 0.9% sodium chloride, withdraw 100ml of fluid from the bag and discard. Inject 100ml of 50% glucose into the bag and mix well.

Glucose Infusion Rate (GIR)

- Calculating the amount of glucose required (GIR) to maintain normoglycaemia is helpful when considering the diagnosis
- Most infants/children will be normoglycaemic on an initial GIR of 5-8 mg/kg/min
- Infants or children who are hyperinsulinaemic OR have minimal glycogen stores may need a high glucose infusion rate to maintain normoglycaemia
- Infants with congenital hyperinsulinism usually require a GIR greater than 10mg/kg/min[3]

GIR = (% of glucose infusion X rate of infusion (ml/hr)) / (Body weight in kg x 6)

Ongoing Management

All patients with unexplained hypoglycaemia needing investigation require a period of observation and discussion with a paediatrician. Admission to an inpatient service is usually required but observation in the emergency department or extended emergency care unit can be considered in patients with a previous history of idiopathic ketotic hypoglycaemia who are well and tolerating oral intake.

Admission to General Paediatric Ward required when:

- Hypoglycaemia is unexplained
- Red flags on history or examination
- Acidosis
- Not tolerating oral intake / vomiting
- Requiring IV fluid therapy or multiple IV boluses
- Not returned to baseline conscious state

All children who do not require admission require a trial of fluids and observation for at least four hours in the emergency department or extended emergency care unit. These children include well children, who are tolerating oral intake AND have a history of idiopathic ketotic hypoglycaemia.

- Appropriate oral fluids: 100% apple juice, 10% glucose polymer (Polyjoule)
- Unsuitable oral fluids: Hydralyte ice blocks / rehydration solution (1.6% glucose)

However, consider re-attempting IV insertion and admission under the General Paediatrics team if the child does not tolerate oral fluids, is vomiting or initial investigations are abnormal.

During admission to ward:

- Continue 10% glucose + 0.9% sodium chloride infusion
- Administer ondansetron for children over 6 months of age with nausea and vomiting
- Encourage oral fluids and diet, preferably with foods containing carbohydrates
- Once tolerating diet, ketones <0.5 and BGL stabilised > 3.5 , wean and discontinue IV fluids

Monitoring

- BGL should be checked 15 minutes after initial treatment
- BGL should be checked 30 minutes after commencement of intravenous fluids
- Capillary sample BGL should be monitored every 4-6 hours following stabilisation of BGLs to aim for BGL above 3.5
- Capillary sample ketones should be monitored every 12-24 hours if elevated, aiming for clearance of ketones (<0.5)

*A controlled fast can be dangerous and should never be done in the acute setting

Discharge Disposition

Patients can be discharged when:

- Initial investigations have been reviewed and no significant diagnoses identified
  - Acid-base, lactate, EUC, CMP, LFTs
- They are eating and drinking normally
- Normal conscious state
- Ketones <0.5 and BGL >3.5 following cessation of IV therapy for at least 4 hours

Discharge Education

- Recommend child having food prior to bed to prevent fasting for longer than 12 hours
- If unwell with > 2 vomits or lethargy to have a low threshold for presentation to the emergency department.
- If appropriate, provide with hypoglycaemia handout.

Follow Up

Follow up for presumed Idiopathic Ketotic Hypoglycaemia
All patients should be followed up, either face to face or virtual with the local General Paediatric team within 3 months, to review blood and urine results and to decide if a referral to Metabolic or Endocrine Units is necessary

Refer to Metabolic Unit:

- Recurrent unexplained hypoglycaemia
- Severe hypoglycaemia (BGL < 0.5)
- Lactic acidaemia
- Concerning features on history or examination (hepatomegaly)

Refer to Endocrine Unit when

- Concerning features on history or examination for hormone deficiency (short stature, midline defects)
- Laboratory findings consistent with an inappropriate hormone response to hypoglycaemia (cortisol, growth hormone, insulin, ACTH)

References

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Acknowledgements

The South Australian Child and Adolescent Health Community of Practice gratefully acknowledge the contribution of clinicians and other stakeholders who participated throughout the guideline development process particularly:

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Next review due: 04/08/2027

ISBN number: 978-1-76083-568-2

CGSQ reference: PCPG006

Policy history: Is this a new policy (V1)? **Y / N**

Does this policy amend or update and existing policy? **Y / N**

If so, which version?

Does this policy replace another policy with a different title? **Y / N**

If so, which policy (title)?

Approval Date	Version	Who approved New/Revised Version	Reason for Change
24/08/22	V1.0	Domain Custodian, Clinical Governance, Safety and Quality	Original version.