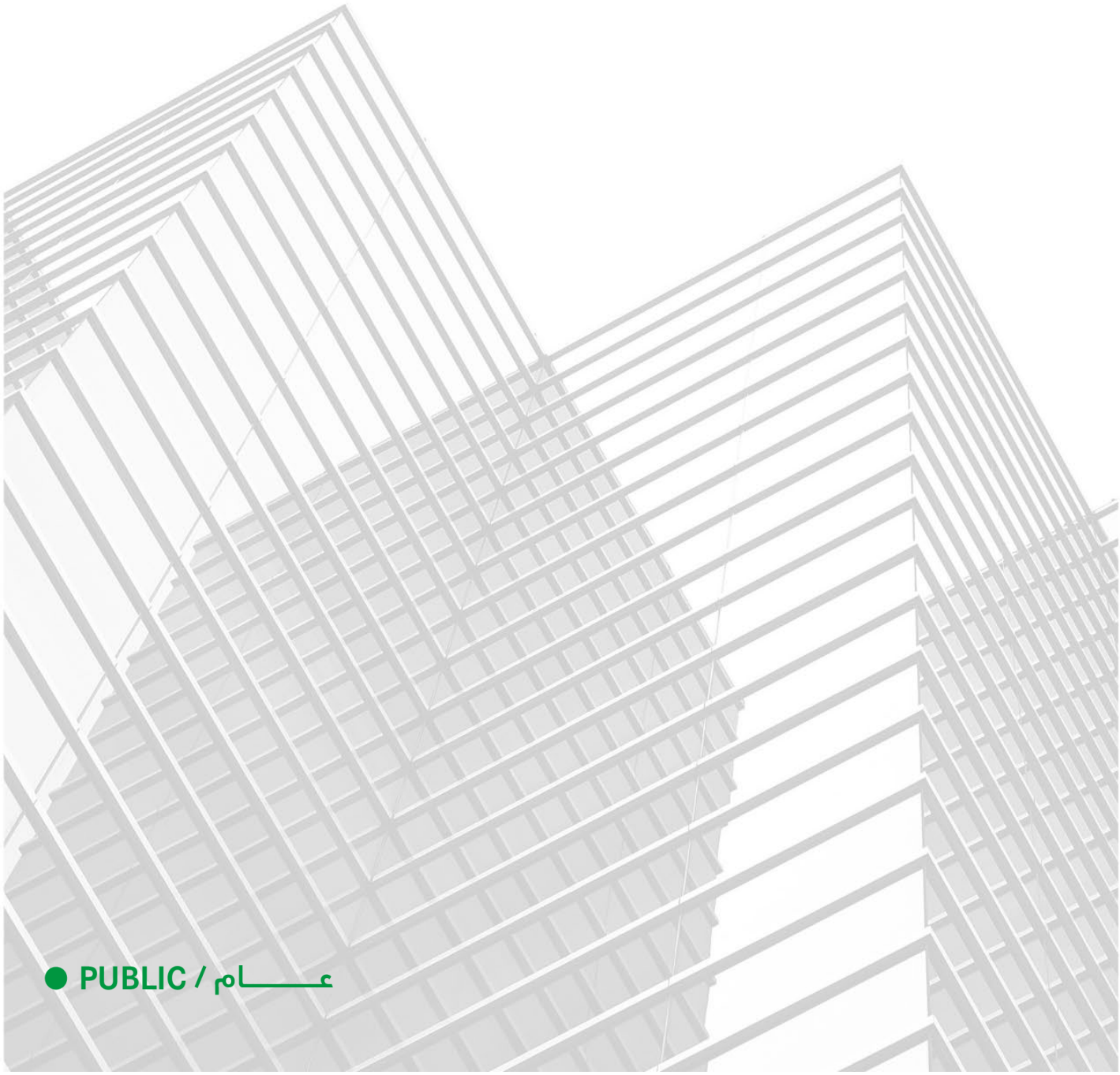
دائـــــرة الـصـــحـــة
DEPARTMENT OF HEALTH

GUIDELINES FOR THE MANAGEMENT OF ANTIDIABETIC DRUG TOXICITY



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1.Guideline Purpose and Brief

1.1 With one in four suffering from diabetes in the United Arab Emirates, antidiabetic drugs are widely accessible and pose a constant risk of toxic exposure in children and adults. Among these drugs are insulin and oral hypoglycemic agents (OHA) such as biguanides, sulfonylureas (SU), and thiazolidinediones. Others, like the GLP-1 agonists, have accrued massive popularity due to their weight-loss properties (Please refer to the related DOH guidelines for the management of obesity) and add layers of risk due to their uncontrolled use. Similarly, current guidelines support the use of SGLT-2 inhibitors exclusively in diabetics. Mounting off-label use requires better awareness among healthcare professionals and the broader public.

1.2 With these guidelines, we aim to reduce the morbidity and mortality associated with antidiabetic drug toxicity by educating healthcare professionals on best practices to achieve:

- Better patient outcomes
- Lower healthcare costs
- Standardized patient care

2. Definitions and Abbreviations

No.	Term / Abbreviation	Definition
2.1	ABC	Airway, breathing, circulation
2.2	ADA	American Diabetes Association
2.3	AMS	Altered mental status
2.4	BGL	Blood glucose level
2.5	β-hCG	Beta human chorionic gonadotropin
2.6	CNS	Central nervous system
2.7	DOH	Department of Health
2.8	DPP-4	Dipeptidyl peptidase -4
2.9	ECG	Electrocardiogram
2.10	GI	Gastrointestinal
2.11	GLP-1	Glucagon-like peptide 1
2.12	IM	Intramuscular
2.13	IV	Intravenous
2.14	MOA	Mechanism of action
2.15	PDIS	Poison & Drug Information Service
2.16	PRN	Pro re nata (as needed)
2.17	SC	Subcutaneous
2.18	SGLT2	Sodium glucose co-transporter 2
2.19	SU	Sulfonylurea

3.Guideline Content

3.1 About these guidelines

3.1.1 These recommendations were formulated following an extensive review of current scientific evidence.

3.1.2 We offer clear guidance on toxic exposures to sulfonylureas and insulin and emphasize prompt recognition and correction of hypoglycemia, especially in pediatric populations. In contrast, while metformin exposure does not typically result in hypoglycemia, it can lead to lactic acidosis in certain clinical scenarios, usually amenable to hemodialysis. Finally, we outline key adverse effects related to the use of newer GLP-1 agonists and SGLT-2 inhibitors.

3.2 Purpose

3.2.1 The aim of these guidelines is to establish a consistent, evidence-based approach to diagnosing and treating antidiabetic toxicity, in both children and adults residing in Abu Dhabi. By empowering all healthcare providers with the latest evidence, DoH anticipates the following:

- Reduced morbidity and mortality
- Recognize high risk patients
- Improve the standard of care
- Lower healthcare costs

3.3 Scope

3.3.1 These guidelines apply to all cases of confirmed or suspected antidiabetic overdose or toxicity across all age groups in the Emirate of Abu Dhabi.

3.4 Key points

3.4.1 Sulfonylureas (SU) are the most important cause of hypoglycemic toxicity[1]

3.4.2 Octreotide is the antidote of choice for SU toxicity

3.4.3 Admission for at least twelve (12) hours must be recommended in cases of both intentional and nonintentional SU overdose

3.4.4 Metformin is not associated with hypoglycemia but can produce severe and unpredictable lactic acidosis in select patients

3.4.5 Management of insulin overdose centers around maintaining euglycemia

3.5 Recommendations for the diagnosis and management of antidiabetic drug toxicity

3.5.1 Consult PDIS

- PDIS should be contacted for all patients presenting with a suspected or confirmed antidiabetic/hypoglycemic overdose or toxicity. Call 800-424

3.5.2 Assess risk

3.5.2.1 Sulfonylureas

- MOA: Stimulate pancreatic insulin secretion
- Pediatric exposure to a single tablet may cause prolonged hypoglycemia.[1] Hypoglycemia may be delayed by up to 18 hours (e.g., modified release formulations)[1]
- Duration of activity, hepatic metabolism, and renal excretion of sulfonylurea drugs determine specific management of hypoglycemia[1]
- Risk factors for prolonged and sever hypoglycemia with sulfonylureas: age >65, polypharmacy, frequent hospitalizations, long-acting agents (e.g., glyburide), impaired drug clearance (kidney/hepatic dysfunction)[2]

Note: Interactions with other drugs may potentiate hypoglycemia (e.g., sulfonamides, propranolol, salicylates, valproic acid, MAOI)[2,3]

3.5.2.2 Metformin

- MOA: Decreases carbohydrate absorption from the gut, increases glucose uptake in peripheral tissues in the presence of insulin, and reduces hepatic gluconeogenesis[3,4]
- Small overdoses (≤10 g in adults, ≤1.7 g in pediatrics) may be asymptomatic or present with nausea and vomiting[2,4]
- Metformin-associated lactic acidosis (MALA) may occur with large overdoses, (metformin overdose does not typically cause hypoglycemia) co-morbid renal or cardiac failure, or in the presence of co-ingestants impairing renal function[2,4]

3.5.2.3 Thiazolidinediones

- MOA: Increase insulin sensitivity in peripheral tissue, thereby increasing glucose uptake; also reduce hepatic gluconeogenesis
- Limited reports in literature, none of which relate to hypoglycemia
- Accidental ingestion or deliberate overdose are unlikely to cause hypoglycemia

3.5.2.4 Insulin

- MOA: Promotes cellular uptake of glucose into skeletal and cardiac muscles and adipose tissue; also shifts potassium intracellularly[3]
- Based on formulation, onset of action and peak effect may be delayed by up to 18 hours; hypoglycemia can potentially last for days even with short acting insulin in geriatric patients and in cases of decreased renal function[5,3]
- Duration of action may be significantly prolonged with larger doses regardless of the type of insulin, especially with erratic absorption from subcutaneous administration[5]
- Poor correlation between insulin dose and severity of outcomes; however, timing of presentation correlates well with prognosis
- Insulin ingestion will not cause hypoglycemia because of enzymatic degradation in stomach[5]

3.5.2.5 GLP-1 analogues

- MOA: Stimulate insulin and inhibit glucagon secretion from pancreas[3,6]
- GI upset (nausea, vomiting, diarrhea) is common even in therapeutic range; pancreatitis reported but no causal relationship established[6]
- Hypoglycemia is uncommon[3,4,6]; however, it is reported at lower doses in diabetic patients compared to non-diabetics

3.5.2.6 DPP-4 inhibitors (gliptins)

- MOA: Similar to GLP-1 analogues; block enzyme which breaks down incretins such as GLP-1, increasing their circulating levels[3,4]
- Most cases are asymptomatic or develop only mild abdominal discomfort
- In a review of 650 patients, <0.5% developed hypoglycemia[3,7]

3.5.2.7 SGLT-2 inhibitors

- Commonly known as gliflozin or flozins
- MOA: Decrease glucose reabsorption by kidneys at level of proximal renal tubule (subsequently increasing urinary glucose excretion)[3,4,8]
- Most cases are asymptomatic or mildly symptomatic (nausea, vomiting, dizziness)

- Hypoglycemia is more likely in large intentional overdoses or accidental pediatric ingestions
- Hypotension with or without resultant acute kidney injury is possible due to osmotic diuresis and volume depletion[2,8]
- Associated with an increased risk of DKA even in the absence of hyperglycemia (i.e. euglycemic DKA)[3,8]. This risk is compounded in those taking canagliflozin and in type 1 diabetics[8]. Note that this adverse effect is common even at therapeutic doses

Refer to the list of relevant hypoglycemic vs non-hypoglycemic antidiabetic agents (Appendix I).

3.5.3 Recognize clinical manifestations

Hypoglycemia[1,9]

- Autonomic: Tachycardia, diaphoresis
- CNS: Headache, agitation, tremors, drowsiness, AMS, coma, seizures

Lactic acidosis (often delayed)[4]

- Autonomic: Tachycardia, hypotension, dyspnea, shock
- CNS: AMS
- GI: Nausea, vomiting, diarrhea

3.5.4 Initial approach

3.5.4.1 Obtain a toxicologic history from patients, family members, and EMS personnel including:

- Drug name, formulation, and dose
- Time of overdose
- Nature of overdose (i.e., intentional or accidental)
- Presence of co-ingestants

3.5.4.2 Record a full set of vital signs (blood pressure, heart rate, respiratory rate, oxygen saturation, and body temperature).

3.5.4.3 Perform rapid bedside testing (glucose, ECG, and β-hCG for females of child-bearing age) and recognize clinically relevant hypoglycemia (see Appendix II)

3.5.4.4 Stabilize ABCs and assess for manifestations of toxicity (see under section 3.5.3).

3.5.4.5 Investigate based on clinical scenario and culprit drug:

- Serial BGL: Indicated in all cases of suspected hypoglycemia. Initially q30-60 mins until euglycemia maintained for 4 h, then q2-4 h
- Renal function & electrolytes (hypokalemia and hypomagnesemia): Required to assess complication risk, delayed or prolonged toxicity
- Blood gas +/- serum lactate level: To assess metformin toxicity
- Toxicologic panel (paracetamol, salicylate, and ethanol blood levels): For intentional overdoses to rule out co-ingestants

3.5.5 Management of sulfonylurea toxicity

3.5.5.1 Resuscitation: Refer to the initial approach.

- Standard supportive care (oxygen supplementation, etc.) as indicated
- Reversal of hypoglycemia
    - If BGL <60 mg/dL (<3.3 mmol/L), immediately administer IV dextrose bolus
        - Adult: 0.5-1 mL/kg of D50[9,3]
        - Peds: Use the 'rule of 50'(≤12 months: 5 mL/kg of D10; 1 - 8 years: 2 mL/kg of D25)[3,10]
    - If IV access cannot be established, consider glucagon IM or SC[3]

        - Adult: 1 mg
        - Peds: 0.03 mg/kg dose can be repeated every 15 mins if needed up to 3 total doses- max 1 mg/dose
        - Doses can be repeated q20 mins PRN [8]
    - Start octreotide infusion as soon as feasible (see 'Antidote' below)

- Maintenance of euglycemia
    - Check BGL q30-60 mins: Target 90-198 mg/dL (>5.5-11 mmol/L)
    - Consider judicious use of dextrose infusions until octreotide can be started if hypoglycemia not corrected by boluses
        - Adult: D10 at 100 mL/h
        - Peds: Maintenance fluids (0.9% NS + D5)
        - Note: Dextrose infusions are only a temporizing measure and are not recommended prophylactically (in the absence of hypoglycemia).[9] Their use is strongly associated with rebound hypoglycemia, and they should be discontinued as early as possible

    - In the unlikely event higher concentrations of dextrose are required, consider inserting a central line
        - Provide enteral feeding (i.e. oral complex carbohydrates) as tolerated [2,9]

- Monitor and replace potassium as needed[2]

3.5.5.2 Decontamination: Consider activated charcoal[2,4]

- Indications
    - Cooperative patient presenting within 2 h of ingestion (or 4 h for extended-release preparations)
    - Any intubated patient once the airway is secure.
- Dosage
        - Adult: 0.5-1 g/kg PO (up to 50 g)
        - Peds: 1 g/kg

Enhanced elimination: N/A

3.5.5.3 Antidote: Octreotide opposes SU toxicity by inhibiting insulin secretion from the pancreas.

- Indication: Confirmed hypoglycemia in the setting of SU toxicity. Administer as early as possible during treatment
- Dosage
        - Adult: 50-100 mcg SC q6 h (preferred)[2,3] or may repeat IV q6 – 12 h if needed[2]
        - Peds: 1-1.25 mcg/kg/dose SC (maximum 50 mcg) q6 h; repeat as needed based upon BGL, however children generally only need a single dose[2]
- The SC route is preferred over IV except in cases where peripheral circulation may be compromised
- For IV administration, dilute in 50 mL NS or D5 and infuse over 15 – 30 mins followed by 1-2 mcg/kg/h up to 125 mcg/h. May also administer as IV push over 3 mins[2]
- If hypoglycemia recurs on therapy, correct with glucose and double the octreotide infusion rate
- Discontinue octreotide infusion when all the following criteria met:
    - Supplementary dextrose infusions ceased at least 4 h prior
    - No symptoms of hypoglycemia
    - Bedside BGL 45 mg/dL (>2.5 mmol/L) for more than 4 h[4]

3.5.5.4 Disposition

- Admission for 12-24 h is indicated for all patients with SU overdose to observe for delayed or prolonged hypoglycemia
- Consider discharge when:

- Normal BGL maintained for 12 h post-cessation of octreotide infusion and patients are on a normal diet
- Plasma insulin level (if available) is normal 6 h post-cessation
- Patient is asymptomatic and euglycemic

- Obtain psychiatric evaluation for intentional overdoses

3.5.6 Management of metformin toxicity

3.5.6.1 Resuscitation: Refer to the initial approach.

- Standard supportive care (oxygen supplementation, fluid resuscitation, endotracheal intubation, etc.) as indicated.
- Treat hypoglycemia as above; however, consider co-ingestants/other etiologies as metformin is unlikely to be the cause
- In critically ill patients, consider sodium bicarbonate (1-2 mmol/kg) as a temporizing measure until hemodialysis can be performed[3]
- In decompensating patients on therapeutic metformin, discontinue the offending agent and search for an underlying cause (sepsis, acute kidney injury, respiratory failure, etc.)

3.5.6.2 Decontamination: Consider activated charcoal for large overdoses only (see under section 3.5.5.2).

Enhanced elimination[2,4]: Hemodialysis removes both metformin and lactate, thereby correcting the metabolic acidosis, as well as preventing further lactate production.

- Indications: Decompensating patients on therapeutic metformin who develop lactic acidosis; unstable patients presenting in acute overdose with worsening acidosis

3.5.6.3 Antidote: N/A.

3.5.6.4 Disposition[4]

- Consider discharge when:
  - Asymptomatic patients following accidental exposure <10 g
  - Children following unintentional ingestion <1.7 g
- Consider admission when:
  - Doses exceeding those mentioned above (8-h observation)
  - Any patient with a history of metformin exposure presenting with lactic acidosis requiring critical care/hemodialysis
- Obtain psychiatric evaluation for intentional overdoses

3.5.7 Management of insulin toxicity

3.5.7.1 Resuscitation: Refer to the initial approach.

- Standard supportive care (oxygen supplementation, etc.) as indicated
- Reversal of hypoglycemia
  - Initial IV dextrose bolus (Refer to recommendation under section 3.5.5.1)
  - Commence dextrose infusion:
    - Adults: D10 at 100 mL/h
    - Peds: Maintenance fluids (0.9% NS + D5)
  - If hypoglycemia persists, treat with another bolus
  - If euglycemia cannot be maintained, consider central line insertion for more concentrated dextrose solutions
- Monitor and replace potassium as needed
- Provide enteral feeding (i.e., oral complex carbohydrates) as tolerated

3.5.7.2 Decontamination: N/A.

Enhanced elimination: N/A.

3.5.7.3 Antidote: Octreotide may be considered in large overdoses/refractory cases. Consult PDIS for further advice.

3.5.7.4 Disposition

- Consider discharge for patients who are well at 6 h with a normal BGL
- Consider observation for at least 18 h for asymptomatic patients with an intentionally administered large quantity of insulin (especially long-acting)
- Admit patients requiring dextrose infusion to a critical care unit (HDU/ICU)
- Obtain psychiatric evaluation for intentional overdoses

3.5.8 Management of other antidiabetic toxicities

3.5.8.1 Thiazolidinediones

- Consider a conservative approach with an 8-h observation period, monitoring for potential albeit unlikely hypoglycemia

3.5.8.2 GLP-1 analogues, DDP-4 inhibitors

- Consider a short observation period of 4-6 h as symptoms are unlikely to manifest beyond this period
- Symptomatic treatment as needed (e.g., anti-emetics, hydration)
- Correct hypoglycemia, if any (see under section 3.5.5.1)

3.5.8.3 SGLT-2 inhibitors

- Consider a short observation period of 4-6 h as symptoms are unlikely to manifest beyond this period
- Obtain serum and urine ketones in any patient on SLGT-2 inhibitors presenting with abdominal pain, fatigue, vomiting and discontinue drug until symptoms resolve
- Supportive treatment as needed, including fluid resuscitation for hypovolemia or hypotension
- Note that the associated AKI usually improves with time; however, dialysis may be necessary in severe renal impairment
- Correct hypoglycemia, if any (see under section 3.5.5.1)

Appendix I: List of relevant hypoglycemic and non-hypoglycemic agents

Agent	Duration	Comment
Likely to cause hypoglycemia		
SU – 1st generation		Toxicity depends on agent, dose, and co-ingestants/drug interactions.
Acetohexamide	12 – 18 h	
Chlorpropamide	24 – 72 h	
Tolazamide	10 – 24 h	
Tolbutamide	6 – 12 h	
SU – 2nd generation		
Glimepiride	24 h	
Glipizide	16 – 24 h	
Glyburide	18 – 24 h	
Insulin		Deliberate SC injection can cause prolonged hypoglycemia. Oral administration is generally benign.
Rapid Acting		
Aspart	3 – 5h	
Glulisine	<5 h	
Lispro	<5 h	
Short Acting		
Regular	5 – 8 h	
Intermediate Acting		
Lente	18 – 24 h	
NPH	18 – 24 h	
Long Acting		
Detemir	24 h	
Glargine	24 h	
Ultralente	20 – 36 h	
Unlikely to cause hypoglycemia		
Thiazolidinediones		Liver toxicity at therapeutic doses. Caution in congestive heart failure.
Pioglitazone	16 – 24 h	
Rosiglitazone	12 – 24 h	
DDP-4 inhibitors		GI symptoms are more likely than hypoglycemia.
Sitagliptin	24 h	
Saxagliptin		
Linagliptin		
Alogliptin		
SGLT-2 inhibitors		Euglycemic DKA. Hypotension due to volume depletion.
Canagliflozin	24 h	
Dapagliflozin		
Empagliflozin		
Variable effect on BGL (but low likelihood)		
Biguanides		Lactic acidosis.
Metformin	12 – 24 h	
GLP-1 analogues		Exenatide available as ER, lasting as long as 10 weeks. Semaglutide available as PO & SC.
Exenatide	6 – 8 h	
Liraglutide	24 h	
Dulaglutide	1 week	
Albiglutide	1 week	
Semaglutide	2 weeks	

Appendix II: ADA classification of hypoglycemia[11,12,13,15]

Level	Description
Level 1	BGL 54 – 70 mg/dL (3.0 – 3.9 mmol/L)
Level 2	BGL <54 mg/dL (<3.0 mmol/L)
Level 3	Any BGL associated with altered mental status requiring assistance for correction of hypoglycemia

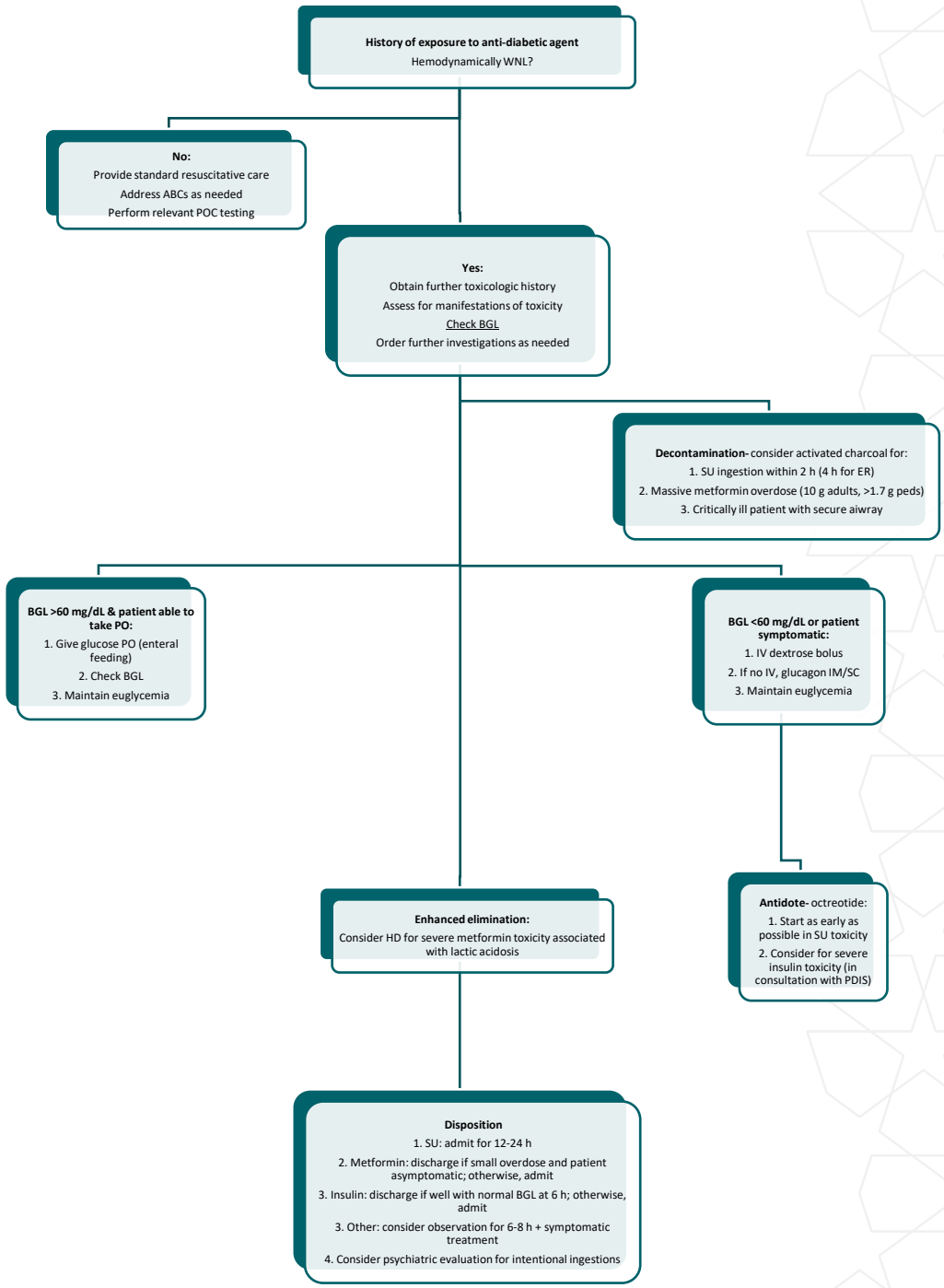
Non-diabetics

The threshold at which symptoms start to develop is between 50 and 70 mg/dL (2.8 – 3.9 mmol/L), references vary. Again, if patients are symptomatic at a level higher than this, consider prompt treatment.

Pediatrics

A level below 70 mg/dL (3.9 mmol/L) is a trigger for correction, <54 mg/dL (3.0 mmol/L) is clinically serious, and any reading associated with significant cognitive deficit is considered severe hypoglycemia.

Appendix III: Management algorithm



4.Relevant References Documents

No.	Reference Date	Reference Name	Relation Explanation / Coding / Publication Links
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