Hyperglycemia

William L. Lanier

Risk

- Incidence in USA: Can occur in virtually any anesthetized or critically ill pt
- Race with the highest prevalence: None

Perioperative Risks

- Dehydration resulting from osmotic diuresis
- Increased likelihood of neurologic injury following brain ischemia and perhaps traumatic brain injury and spinal cord injury
- Increased infection rate
- Diminished wound healing

Worry About

- Lyte abnormalities, particularly hypokalemia, while treating hyperglycemia.
- Hypoglycemia following insulin, resulting in insult to the CV system and CNS.
- Polyuria complicates assessment of fluid balance.

Overview

- Is not a disease.
- Typically produces adverse effects by three mechanisms: Increases in plasma osmolality, increases in postischemic tissue lactic acidosis, and inhibition of white blood cell function.
- In acute setting, blood glucose concentration can be estimated using indicator-impregnated strips or other point-of-care methodologies; confirmation can be made by mechanized techniques in a reference laboratory.

Etiology

- Results from DM (both insulin-requiring and non-insulin–requiring), other endocrinopathies (Cushing syndrome, acromegaly, obesity, pheochromocytoma), physiologic stress, drug administration (particularly corticosteroids), and glucose-containing fluid infusions

Usual Treatment

- Insulin.
- Isotonic IV crystalloid solutions to treat hypovolemia and dilute existing blood glucose.
- If possible, treat underlying cause (e.g., discontinue infusion of glucose-containing solutions, discontinue corticosteroids, reduce physiologic stress to pts).

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Dehydration in extreme cases		Dry mucosa in extreme cases	
CV	Mild positive inotropic effect with mild hyperglycemia Dehydration		Tachycardia, orthostatic hypotension	
GI		Polydipsia in extreme cases		
RENAL	Osmotically induced diuresis	Polyuria, urinary frequency		Elevated urine glucose
ENDO		See Etiology		Elevated blood glucose
HEME	Diminished WBC activity; changes in serum sodium concentrations			Serum sodium concentration decreases 1.6 mEq/L for each 100 mg/dL increase in glucose concentration
CNS			Altered consciousness, neurologic deficits	Plasma osmolality

Key References: Akhtar S, Barash PG, Inzucchi SE: Scientific principles of perioperative glucose regulation and control, *Anesth Analg* 110(2):478–497, 2010; Pasternak JJ, McGregor DG, Schroeder DR, et al: Hyperglycemia in patients undergoing cerebral aneurysm surgery: its association with long-term gross neurologic and neuropsychological function, *Mayo Clin Proc* 83(4):406–417, 2008.

Perioperative Implications

Preoperative Preparation

- Glucose reduction with insulin
- Hydration
- Normalization of lytes

Monitoring

- Blood glucose concentrations in all cases
- In severe cases, blood lytes, blood osmolality, and urine output

Airway

- Abnormality typically related to DM (reduced range of motion and abnormal atlanto-occipital contractions), acromegaly (distorted anatomy), or chronic corticosteroid use or Cushing syndrome (Cushingoid signs and symptoms, friable tissues)

Maintenance

- Maintain hydration.
- Insulin therapy.
- K+ replacement.

Extubation

- No special considerations, other than those related to underlying disease

Adjuvants

- Limit attempted reduction of blood glucose concentration to approximately 75 mg/dL/h to avoid problems with osmotic injury to brain and lyte disturbances.
- Monitor ECG during correction of profound hyperglycemia.

Postoperative Period

- Variations in physiologic stress, fluid administration, and drug usage make postop blood glucose concentrations difficult to predict and control.

Anticipated Problems/Concerns

- Increases in blood glucose concentrations by a mere 40 mg/dL may worsen outcome following cerebral ischemic insult. Hyperglycemia may also harm wound healing, increase infection rates, and worsen outcomes after myocardial infarction. In contrast, hypoglycemia resulting from excessive use of insulin may result in pt morbidity and mortality from neurologic and other causes, independent of ischemic events.
- Limb hypothermia or hypoperfusion will harm the accuracy of glucose measurements from skin-prick blood samples.
- Target blood glucose should be <180 mg/dL in most pts.

Hyperglycemic Hyperosmolar State

Jesse M. Raiten

Risk

- Elderly pts with DM, usually type II
- Debilitated pts who cannot care for themselves
- Chronically ill diabetic pts who experience exacerbation of an underlying comorbidity
- Incidence increased in African Americans, Hispanics, and Native Americans

Perioperative Risks

- Severe hypovolemia and hemodynamic instability
- Presence of diffuse organ system damage from poor glycemic control
- Altered mental status and increased risk of pulmon aspiration
- Periop stress causing further elevations in serum glucose

Worry About

- Cause of hyperglycemic hyperosmolar state.
- Volume status and potential hemodynamic instability.
- Electrolyte and acid-base abnormalities increase the risk of cardiac arrhythmias.

Overview

- Serious metabolic condition characterized by hyperglycemia, hyperosmolarity, and dehydration
- Is one of several potentially fatal states associated with poorly controlled DM
- Requires aggressive treatment and close electrolyte and hemodynamic monitoring

Etiology

- Inadequate insulin production and increased counter-regulatory hormone production (catecholamines, glucagon, cortisol) in the setting of an acute insult leads to severe hyperglycemia, dehydration, and electrolyte abnormalities.
- Triggering event may be infection, dehydration, CVA, inadequate dosing of insulin, silent myocardial

infarction, pancreatitis, or drug ingestion (drugs that affect carbohydrate metabolism).

Usual Treatment

- Aggressive volume resuscitation with isotonic fluids to re-establish end-organ perfusion
- Insulin replacement and correction of electrolyte abnormality (start dextrose-containing fluids when serum glucose approaches 250 mg/dL to help to prevent hypoglycemia and cerebral edema)
- Identify and treat underlying cause of hyperglycemic state
- Frequent evaluation of volume resuscitation and metabolic status in an ICU setting

Assessment Points

System	Effect	Assessment by Hx	PE	Test
NEURO	Altered mental status, obtundation, coma, seizures	Progression of mental status changes over days	Mental status exam, airway reflexes	Head CT, CSF culture
CV	Hypovolemia and shock	Polyuria progressing to anuria, sense of thirst, headaches, dry mouth	Orthostatic hypotension, tachycardia, dry mucous membranes	CVP, PAP, ECHO
PULM	Hyperventilation if severe metabolic acidosis, hypoventilation if brainstem malperfusion		Resp rate and pattern of ventilation	ABG
ENDO	Insulinopenia, hyperglycemia, hyperosmolarity	DM, recent infection or stress		Serum glucose and osmolarity
RENAL	Polyuria progressing to anuria, metabolic acidosis, electrolyte abnormalities			BUN and Cr, FEN_a, UO, ABG, lytes

Key References: Maletkovic J, Drexler A: Diabetic ketoacidosis and hyperglycemic hyperosmolar state, *Endocrinol Metab Clin North Am* 42(4):677–695, 2013; Pichardo-Lowden A, Gabbay RA: Management of hyperglycemia during the perioperative period, *Curr Diab Rep* 12(1):108–118, 2012.

Perioperative Implications

Monitoring and Intravenous Access

- Large-bore IV access for volume resuscitation, particularly before induction.
- Invasive monitoring including arterial line and CVP may be useful to guide volume replacement and allow for frequent glucose and electrolyte sampling.

Induction

- Aggressive volume resuscitation before induction.
- Rapid sequence induction if altered mental status and concern for aspiration.
- Limited use of succinylcholine if metabolic acidosis and hyperkalemia are present.
- Be prepared for exaggerated hemodynamic changes with induction despite adequate volume resuscitation.
- Smaller doses than usual of induction agent if pt is obtunded.

Maintenance

- Closely follow serum glucose, lytes, and acid-base status.
- Continue volume resuscitation until UO is adequate and hemodynamics have stabilized.

Emergence

- Assessment of airway reflexes and ability to protect airway before tracheal extubation.
- Ensure metabolic and lyte status is corrected and pt meets the usual criteria for extubation

Postoperative Period

- Continued insulin therapy and observation for worsening hyperglycemia due to surgical stress response

Anticipated Problems/Concerns

- Comorbidities and diffuse end-organ damage increase morbidity and mortality of pts with HHS, particularly periop.

Hyperkalemia

Alan David Kaye | Mark R. Jones | Rachel J. Kaye

Risk

- Any pt with plasma K^+ concentration <5.5 mEq/L

Perioperative Risks

- Muscle weakness and paralysis
- Cardiac conduction system abnormalities
- CV collapse:
  - Peaked T waves (6–7 mEq/L)
  - ST depression
  - Prolonged P-R interval and widened QRS (10–12 mEq/L)
  - Ventricular fibrillation or asystole

Worry About

- Adverse effects are likely to accompany acute increases in K^+; chronic increases are better tolerated.
- Depolarizing muscle relaxants, especially if given to pts with burns, spinal cord transection, catatonia with immobility, or muscle trauma.
- Digitalis toxicity.
- Acidosis.

Overview

- Condition that can be due to increased total body K^+ content or alterations in distribution between intracellular and extracellular sites

Etiology

- Diminished renal excretion
- Acute oliguric renal failure
- Chronic renal failure
- Addison disease
- Hyporeninemic hypoaldosteronism
- Medications: Potassium-sparing diuretics, NSAIDs, heparin, K^+-containing antibiotics
- RAASi: Incidence of hyperkalemia <2% with RAASi monotherapy: Increased to 5% in pts receiving dual-agent RAASi therapy, and to 5%–10% when dual therapy was administered to pts with CKD
- Ingestion of K^+-rich foods, salt substitutes in pts with renal insufficiency
- Transcellular shifts
- Acidosis: Respiratory or metabolic
- Cell destruction: Trauma, burns, rhabdomyolysis, hemolysis, tumor lysis, or reperfusion of ischemic limb or organ
- Hyperkalemic periodic paralysis
- Diabetic hyperglycemia
- Depolarizing muscle relaxant causing K^+ release, especially in pts with burns, spinal cord transection, catatonia with immobility, muscle trauma, or denervating muscle
- Massive transfusion, particularly with irradiated blood
- Factitious hyperkalemia
- Tourniquet method of drawing blood
- Hemolysis of drawn blood due to delay in chemical determination

Usual Treatment

- Promote transfer of K^+ from ECF to ICF.
- Glucose and insulin: 25–50 g glucose with 10–20 units regular insulin/70 kg.
- Sodium bicarbonate: 50–100 mEq/70 kg.
- Hyperventilation: with each pH change of 0.1, there is an inverse change in K^+ of 0.5 mEq/L (goal $PaCO_2$ 25–30 mm Hg).
- Enhance K^+ elimination: Diuretics, exchange resins (Kayexalate), dialysis.
- Antagonism of cardiac effects: Ca^{++} gluconate—10–30 mL of a 10% solution over 10–20 min/70 kg counteracts cardiac effects.
- 50–250 mL hypertonic saline (3%–5%): Stabilizes membrane potential; effective only in hyponatremic pts.
- β_2-receptor agonists: 10–20 mg aerosol (nebulized) or 0.5 mg in 100 mL of 5% dextrose in water (IV) redistributes K^+ from ECF to ICF; use with caution in pts with CAD.
- New treatments: K^+ binders (patiromer and sodium zirconium cyclosilicate [ZS-9]) increase fecal potassium excretion; may offer better predictability, tolerability, and safety for pts with hyperkalemia; importantly, these new K^+ binders may allow the continued use of such medications as RAASis