Perioperative Implications

Preoperative Preparation

- Pts with metabolic acidosis may be hemodynamically unstable and demonstrate decreased responsiveness to inotropes and vasopressors.
- Consider postponing surgery until the underlying cause is corrected, unless treatment requires immediate surgical intervention.
- If surgery is urgent or emergent, consider ways to optimize the pt preop.

Intraoperative

- Invasive monitoring may be indicated, depending on the severity of illness.
- Goal for induction is hemodynamic stability.
- Inotropes and vasopressors should be readily available.
- Consider the need for pt to remain intubated postop.

Postoperative Period

- Pt may require postop ICU care and prolonged mechanical ventilation.

Anticipated Problems/Concerns

- Hemodynamic instability with decreased responsiveness to inotropes and vasopressors.
- Compensation for profound metabolic acidosis may lead to acute resp failure.
- Treatment with bicarbonate may paradoxically increase $PaCO_2$ and worsen intracellular acidosis and resp status.

Acidosis, Renal Tubular

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Risk

- Incidence in USA: Unknown
- Present in a variety of disease states, from mild to severe systemic illness

Perioperative Risks

- Hemodynamic instability (related to arteriolar vasodilation, acidosis, and decreased cardiac output)
- Hyperkalemia
- Insulin resistance and hyperglycemia
- Acute respiratory failure

Worry About

- Decreased responsiveness to vasopressors and inotropes
- Decreased activity of local anesthetic agents
- Arrhythmias

Overview

- RTA is a type of metabolic acidosis that is due to either abnormal bicarbonate loss or acid excretion by the kidneys in presence of a normal or near normal glomerular filtration rate.
- Results in non-anion gap metabolic acidosis.
- Metabolic acidosis not due to gastrointestinal bicarbonate loss or acute/chronic renal insufficiency.
- Related to either proximal tubule dysfunction of bicarbonate reabsorption, failure of distal tubule excretion of acid, or mineralocorticoid deficiency.
- Other findings may include recurrent nephrocalcinosis, growth retardation, and osteomalacia/rickets in children.
- Can be either inherited, transient, or acquired.

Etiology

- Distal RTA (type 1) is due to defective distal tubular H^+ secretion.
  - Clinical features include impairment of growth, polyuria, hypercalciuria, lithiasis, nephrocalcinosis, and K^+ depletion.
  - Acquired forms related to hypergammaglobulinemia, autoimmune disorders such as SLE or Sjögren syndrome, and pts with chronic liver disease.
  - Can be associated with sensorineural hearing loss.
- Proximal RTA (type 2) is due to defective proximal tubule reabsorption of bicarbonate.
  - Manifests as stunted growth in children.
  - Can be associated with Fanconi syndrome, and if so, can manifest with osteomalacia and rickets.
  - Other causes include medications and toxins such as acetazolamide, aminoglycoside antibiotics, expired tetracyclines, lead, cadmium, and mercury.
- Type 3 RTA is a combination of types 1 and 2.
  - Can be transient in pediatric pts with type 1 RTA.
  - Carbonic anhydrase II deficiency is an AutoR syndrome associated with osteoporosis, RTA, cerebral calcification, and mental retardation.
- Hyperkalemic RTA (type 4): Due to either mineralocorticoid deficiency or hormone resistance
  - Most frequently observed in children with hypo- or pseudohypoaldosteronism
  - Also found to be related with diabetic nephropathy, SLE, and AIDS nephropathy
  - Drug induced causes include COX inhibitors, ACE-I's, heparin, K retaining diuretics, trimethoprim, and others

Diagnosis

- Should be suspected anytime metabolic acidosis is accompanied with hyperchloremia and a normal plasma anion gap without evidence of gastrointestinal bicarbonate loss or acid ingestion
- Differential diagnosis (common distal causes of RTA):
  - Hypokalemic or normokalemic: Primary, hypercalcemia, renal transplant rejection, multiple myeloma, SLE, nephrocalcinosis, hepatic cirrhosis, amphotericin B, lithium, and toluene
  - Hyperkalemic: Sickle cell nephropathy, obstructive nephropathy, hypoaldosteronism, and SLE
- Common proximal causes of RTA: Primary, Fanconi syndrome, Wilson disease, metals (mercury, lead, and cadmium), early renal transplant, nephrotic syndrome, and amyloidosis
- Tests used to aid in diagnosis include:
  - CMP to assess plasma electrolytes, baseline kidney function, and plasma anion gap
  - Urine pH and urine anion gap
  - Urine osmol gap and urine PCO_2
  - Urine calcium and citrate excretion
  - Renin: aldosterone ratio
  - Oral administration of acidifying salt (typically with ammonium chloride loading) employed to assess for ammonium secretion; normal individuals achieve a urine pH of less than 5.5, whereas pts with distal RTA are unable to acidify urine
  - Furosemide test: PO or IV dose given to assess distal tubule acidification function
- Metabolic derangements associated with each type
  - Distal: Pt will present with hyperchloremic metabolic acidosis with a positive anion gap or an osmol gap <100 mmol/L. Diagnosis is supported by either normal or decreased plasma K^+ concentration and inability to lower urine pH <5.5 after either acidifying salt (ammonium chloride) loading or furosemide test.
  - Proximal: Pt will present with hyperchloremic metabolic acidosis, negative anion gap, or osmol gap above 100 mmol/L. Definitive diagnosis is made with presence of low urine pH at low plasma bicarb concentration and the presence of normal urine PCO_2 and a high urine bicarb excretion at normal plasma bicarb concentration. GI or renal loss, previous intake of acidifying salt, or excessive use of laxatives must be ruled out.
  - Hyperkalemic RTA: Should be considered if K^+ is increased with urine pH <5.5. Renin and aldosterone levels must be assessed. Hypoaldosteronism is the most common cause of hyperkalemic distal RTA.

Usual Treatment

- Focused on preventing pediatric growth restriction, nephrocalcinosis, and development of chronic renal failure in all ages.
- Treatment based on disease specific alkali replacement using either bicarbonate or citrate.
- Proximal RTA alkali supplementation usually needed until 3 to 5 y of age.
- Distal RTA is more likely to be permanent with treatment needed throughout life.
- Hyperkalemic RTA can be treated with fludrocortisone, furosemide, and alkali supplements if needed.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
NEURO	Altered mental status and seizures	Level of consciousness, delirium, somnolence nausea/vomiting, seizures, toxic ingestion	Obtunded, confused, somnolent	Toxicology screen, osmol gap, serum lytes
CV	Arteriolar vasodilation, hypotension, decreased response to vasopressors and inotropes, arrhythmias, hypocontractility	Signs of end-organ hypoperfusion	Tachycardia, hypotension, poor peripheral pulses, cold extremities, poor capillary refill	Invasive hemodynamic monitoring, ECHO, ECG
PULM	Hypoxemia, hyperventilation, respiratory failure	Tachypnea, dyspnea	Rapid and shallow breathing, accessory muscle use, hypoxia, and hypercarbia	CXR, ABG, pulse oximetry
RENAL	Oliguria, acute kidney injury, ATN	Urine output, chronic renal disease	Signs of hypo- or hypervolemia	UO, Cr, BUN, urine lytes, UA, serum lytes
GI		Nausea, vomiting, diarrhea, melena, abdominal pain	Abdominal pain to palpation	Serum lactate, radiographic imaging, upper/lower endoscopy
ID		Fever, rigors	Hyperthermia or hypothermia, signs of focal infection	WBC with differential, cultures, radiographic imaging
ENDO	Hyperglycemia, insulin resistance	DM, polyuria, polydipsia, hyperphagia	Signs of dehydration	Blood glucose, serum ketones

Key References: Morris CG, Low J: Metabolic acidosis in the critically ill: Part 1. Classification and pathophysiology and Part 2. Causes and treatment. *Anaesthesia* 63:294–301, 396–411, 2008; Laing CM, Unwin RJ: Renal tubular acidosis. *J Nephrol* 19(Suppl 9):S46–S52, 2006.

Perioperative Implications

Preoperative Preparation

- Carefully assess for electrolyte abnormalities, pH balance, volume, and kidney function via CMP, phosphorus, and calcium levels.
- Consider elective versus emergent surgery. If elective and found to have multiple metabolic derangements, consider postponement for appropriate medical management and if surgery is urgent or emergent, consider ways to optimize the pt preop.
- Remember that some forms of RTA can be associated with genetic or autoimmune syndromes such as Fanconi or Sjögren. Incorporate other syndrome specific clinical considerations for each case.

Monitoring

- Arterial line and/or central venous pressure can assist in assessment of volume status periop.

Intraoperative Preparation

- Pts with advanced renal pathogenesis should have anesthetic medications that avoid the kidney, including cis-atracurium with neuromuscular blockade requirement, and avoidance of morphine, which is conjugated to morphine 6-glucoronide and an active metabolite, and is eliminated through the kidney.
- Pts may be hemodynamically unstable, demonstrating decreased responsiveness to inotropes and vasopressors.
- Many pts with advanced renal pathogenesis have increased potassium levels, and, therefore, succinylcholine should be avoided as it can further increase potassium levels, leading to adverse effects such as cardiac arrest.

Postoperative Period

- Pt may require postop ICU care and prolonged mechanical ventilation.

Anticipated Problems/Concerns

- Hemodynamic instability with decreased responsiveness to inotropes and to vasopressors
- Compensation for profound metabolic acidosis, which may lead to acute respiratory failure
- Treatment with bicarbonate, which may paradoxically increase $PaCO_2$ and worsen intracellular acidosis and respiratory status

Acquired Immunodeficiency Syndrome

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Risk

- USA incidence of HIV: 50,000 per year
- USA prevalence of HIV: 1.2 million
  - Sub-Saharan Africa prevalence: 25.8 million in 2014
- USA prevalence of AIDS (HIV stages 3 and 4): 26,688
  - All but 8 were age 13 or older

Perioperative Risk

- Susceptibility to infection
- Drug interactions
- Occupational exposure/viral transmission

Overview

- AIDS is the clinical syndrome representing the late and more severe stages of infection with HIV.
- Once inside the host, HIV attaches and is internalized to CD4+ T4 helper lymphocytes. Viral RNA is transcribed into the cell's DNA allowing formation of viral progeny within the host. Host's CD4+ T4 helper lymphocytes become defective and unable to help fight opportunistic infections and neoplasms causing progressive immunocompromisation.
- In 2007 WHO published a clinical classification system for HIV/AIDS; stages 1 to 4 are based on clinical symptoms and presence of associated illnesses, and AIDS is defined by the presents of features of stages 3 and 4.

Etiology

- Transmission can occur when there is contact between HIV-infected blood or contaminated body fluids and open wounds, broken skin, or mucus membranes.
- During sexual encounters, delivery of a fetus, breastfeeding, inoculation by contaminated needles, and occupational exposure.

Usual Treatment

- Regimen of HAART.
  - Target different stages in HIV replication cycle.
- Pts may be taking additional drugs, both for treatment and for prophylaxis targeting associated illnesses, such as other antivirals, antifungals, antibiotics, and/or chemotherapy drugs.