

## Metabolic acidosis

Metabolic acidosis is primary reduction in bicarbonate ( $\text{HCO}_3^-$ ), typically with compensatory reduction in carbon dioxide partial pressure ( $\text{PCO}_2$ ); pH may be markedly low or slightly subnormal. Metabolic acidoses are categorized as high or normal anion gap based on the presence or absence of unmeasured anions in serum. Causes include accumulation of ketones and lactic acid, renal failure, and drug or toxin ingestion (high anion gap) and gastrointestinal or renal  $\text{HCO}_3^-$  loss (normal anion gap). Symptoms and signs in severe cases include nausea and vomiting, lethargy, and hyperpnea. Diagnosis is clinical and with arterial blood gas (ABG) and serum electrolyte measurement. The cause is treated; IV sodium bicarbonate may be indicated when pH is very low.

### Etiology

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Metabolic acidosis is acid accumulation due to

- Increased acid production or acid ingestion
- Decreased acid excretion
- Gastrointestinal or renal  $\text{HCO}_3^-$  loss

Acidemia (arterial pH < 7.35) results when acid load overwhelms respiratory compensation. Causes are classified by their effect on the anion gap

### High anion gap acidosis

The most common causes of a high anion gap metabolic acidosis are

- Ketoacidosis
- Lactic acidosis
- Renal failure
- Toxic ingestions

Ketoacidosis is a common complication of type 1 diabetes mellitus (see diabetic ketoacidosis), but it also occurs with chronic alcohol use disorder (see alcoholic ketoacidosis), undernutrition, and, to a lesser degree, fasting. In these conditions, the body converts from glucose metabolism to free fatty acid (FFA) metabolism; FFAs are converted by the liver into ketoacids, acetoacetic acid, and beta-hydroxybutyrate (all unmeasured anions). Ketoacidosis is also a rare manifestation of congenital isovaleric acidemia or congenital methylmalonic acidemia.

Lactic acidosis is the most common cause of metabolic acidosis in hospitalized patients. Lactate accumulation results from a combination of excess formation and decreased metabolism of lactate. Excess lactate production occurs during states of anaerobic metabolism. The most serious form occurs during the various types of shock. Decreased metabolism generally occurs with hepatocellular dysfunction from decreased liver perfusion or as a part of generalized shock. Diseases and drugs that impair mitochondrial function can cause lactic acidosis.

Renal failure causes high anion gap acidosis by decreased acid excretion and decreased  $\text{HCO}_3^-$  reabsorption. Accumulation of sulfates, phosphates, urate, and hippurate accounts for the high anion gap.

Toxins may have acidic metabolites or trigger lactic acidosis. Rhabdomyolysis is a rare cause of metabolic acidosis thought to be due to release of protons and anions directly from muscle.

### **Normal anion gap acidosis**

The most common causes of normal anion gap acidosis are

- Gastrointestinal (GI) or renal  $\text{HCO}_3^-$  loss
- Impaired renal acid excretion

Normal anion gap metabolic acidosis is also called hyperchloremic acidosis because the kidneys reabsorb chloride ( $\text{Cl}^-$ ) instead of reabsorbing  $\text{HCO}_3^-$ .

Many GI secretions are rich in  $\text{HCO}_3^-$  (eg, biliary, pancreatic, and intestinal fluids); loss due to diarrhea, tube drainage, or fistulas can cause acidosis. In ureterosigmoidostomy (insertion of ureters into the sigmoid colon after obstruction or cystectomy), the colon secretes and loses  $\text{HCO}_3^-$  in exchange for urinary chloride ( $\text{Cl}^-$ ) and absorbs urinary ammonium, which dissociates into ammonia ( $\text{NH}_3^+$ ) and hydrogen ion ( $\text{H}^+$ ). Ion-exchange resin uncommonly causes  $\text{HCO}_3^-$  loss by binding  $\text{HCO}_3^-$ .

The renal tubular acidoses impair either  $\text{H}^+$  secretion (types 1 and 4) or  $\text{HCO}_3^-$  absorption (type 2). Impaired acid excretion and a normal anion gap also occur in early renal failure, tubulointerstitial renal disease, and when carbonic anhydrase inhibitors (eg, acetazolamide) are taken.

### **Symptoms and Signs**

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Many people don't have symptoms, but some people may have:

- Confusion
- Fast heartbeat
- Feeling sick to your stomach
- Headache
- Long and deep breaths
- Not wanting to eat
- Vomiting

- Feeling tired
- Feeling weak

What problems can happen from metabolic acidosis?

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Here are some health problems that can happen if metabolic acidosis is not treated:

- Your kidney disease can get worse
- Bone loss (osteoporosis), which can lead to a higher chance of fractures in important bones like your hips or backbone.
- Muscle loss, because of less protein in your body.
- Endocrine disorders, which mean that glands that produce hormones in your body are not working like they should.
- Slowed growth in children
- Inflammation (swelling, redness, and pain)
- Amyloid accumulation, which is a build-up of protein in your body that can hurt your joints, organs and brain.
- Increased chance of death

### Diagnosis

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- Arterial blood gas (ABG) and serum electrolyte measurement
- Anion gap and delta gap calculated
- Winters formula for calculating compensatory changes
- Testing for cause

Recognition of metabolic acidosis and appropriate respiratory compensation are discussed in Diagnosis of Acid-Base Disorders. Determining the cause of metabolic acidosis begins with the anion gap.

The cause of an **elevated anion gap** may be clinically obvious (eg, hypovolemic shock, missed hemodialysis), but if not, blood testing should include

- BUN (blood urea nitrogen)
- Creatinine
- Glucose
- Lactate
- Possible toxins

Salicylate levels can be measured in most laboratories, but methanol and ethylene glycol frequently cannot; their presence may be suggested by presence of an osmolar gap.

Calculated serum osmolarity ( $2 [\text{sodium}] + [\text{glucose}]/18 + \text{BUN}/2.8 + \text{blood alcohol}/5$ , based on conventional units) is subtracted from measured osmolarity. A difference  $> 10$  implies the presence of an osmotically active substance, which, in the case of a high anion gap acidosis, is methanol or ethylene glycol. Although ingestion of ethanol may cause an osmolar gap and a mild acidosis, it should never be considered the sole cause of a significant metabolic acidosis. If the **anion gap is normal** and no cause is obvious (eg, marked diarrhea), urinary electrolytes are measured and the urinary anion gap is calculated as  $[\text{sodium}] + [\text{potassium}] - [\text{chloride}]$ . A normal urinary anion gap (including in patients with gastrointestinal losses) is 30 to 50 mEq/L (30 to 50 mmol/L) ; an elevation suggests renal  $\text{HCO}_3^-$  loss (evaluation of renal tubular acidosis is discussed elsewhere).

In addition, when metabolic acidosis is present, a **delta gap** is calculated to identify concomitant metabolic alkalosis, and Winters formula is applied to determine whether respiratory compensation is appropriate or reflects a second acid-base disorder.

#### **Treatment**

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## **Treatments for metabolic acidosis**

Treatment for metabolic acidosis works in three main ways:

- excreting or getting rid of excess acids
- buffering acids with a base to balance blood acidity
- preventing the body from making too many acids

Other kinds of treatment for metabolic acidosis include:

### **Respiratory compensation**

If you have respiratory acidosis, blood gas tests will show high carbon dioxide levels. Other tests to diagnose this kind of metabolic acidosis include breathing tests to show

how well the lungs are working, and a chest X-ray or CT scan to check for lung infection or blockage.

Respiratory treatments for metabolic acidosis include:

- bronchodilator medications (Ventolin inhaler)
- steroid drugs
- oxygen
- ventilation machine (CPAP or BiPaP)
- breathing machine (for severe cases)
- treatment to stop smoking

## **Metabolic compensation**

### **Diabetes treatment**

Resolving metabolic acidosis caused by untreated or uncontrolled diabetes includes treatment for diabetes. If you have diabetic ketoacidosis, your blood tests will show high blood sugar levels (hyperglycemia). Treatment includes balancing blood sugar levels to help the body remove and stop making acids:

- insulin
- diabetes medications
- fluids
- electrolytes (sodium, chloride, potassium)

Insulin treatment will only work if diabetes is causing the metabolic acidosis.

## **IV sodium bicarbonate**

Adding base to counter high acids levels treats some types of metabolic acidosis. Intravenous (IV) treatment with a base called sodium bicarbonate is one way to balance acids in the blood. It 's used to treat conditions that cause acidosis through bicarbonate (base) loss. This can happen due to some kidney conditions, diarrhea, and vomiting.

## **Hemodialysis**

Dialysis is a treatment for serious kidney disease or kidney failure. Blood tests for chronic kidney problems will show high levels of urea and other kinds of acid. A urine test can also show how well the kidneys are working.

Dialysis helps to remove extra acids and other wastes from the blood. In hemodialysis, a machine filters the blood and removes wastes and extra fluids. Peritoneal dialysis is a treatment that uses a solution inside your body to absorb wastes.

## **Other treatments for metabolic acidosis**

- Inotropes and other medications help improve heart function in conditions like low blood pressure and heart failure. This improves oxygen flow to the body and lowers blood acid levels. Blood pressure readings, blood tests, and an ECG (electrocardiogram) will show if a heart problem is causing the metabolic acidosis.
- Metabolic acidosis due to alcohol or drug poisoning is treated with detoxification. Some people may also need hemodialysis to clear out toxins. Blood tests including liver function tests will show an acid-base imbalance. A urine test and blood gas test can also show how serious the poisoning is.