

Acid Base Online Tutorial



Introduction
Pearls

Acid Base Physiology

Acid Base Abnormalities

Cases

◀ Back

Lactic Acidosis

Ketoacidosis

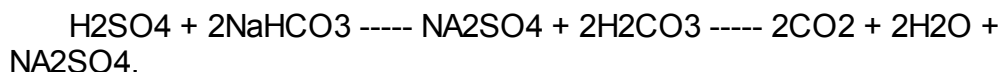
Renal Tubular
Acidosis

Uremia

Uremic Acidosis

Metabolic acidosis is a common complication of renal disease and results from an inability of the diseased kidney to excrete the daily dietary acid load. To understand the effects of acid base with progressive renal failure, it is first necessary to review the normal handling of acids.

The daily dietary acid load is primarily due to the generation of H₂SO₄ from the metabolism of sulphur containing amino acids. This acid is rapidly buffered by HCO₃⁻ and other buffers, leading to the formation of sodium sulphate salts.



To maintain the steady state, both the 2H⁺ and the SO₄²⁻ must be excreted in the urine. The excretion of H⁺ occurs via the excretion of titratable acids and more importantly, NH₄⁺. Whilst, the excretion of SO₄²⁻ anions depends on the capacity of the kidney to filter and reabsorb the anions.

In initial stages of chronic renal disease, as the number of functioning renal tubules are reduced, the tubular functions of the kidney are diminished and the kidney's ability to produce NH₄⁺ ions is affected, thus resulting in a reduction of hydrogen ions excreted and an increase in the amount of HCO₃⁻ ions excreted. The excretion of bicarbonate ions results in a reduction in plasma [HCO₃⁻] and thus metabolic acidosis. On the other hand, initially in CKD, ultrafiltration occurs and glomerular filtration rate reduces at a much slower pace than is the loss of tubular function. Therefore, the excretion of sulphate and other organic and inorganic acid anions is not affected as their filtration by the kidney is maintained. In addition, the kidneys lose the capacity to reabsorb these anions due to loss in tubular function leading to further anion excretion. To maintain electroneutrality, the kidneys retain Cl⁻ with each bicarbonate ion lost and thus early renal disease is associated with a hyperchloremic metabolic acidosis. The anion gap is not affected due to the continued excretion of organic acids by the kidneys.

In advanced kidney disease as GFR falls below 20ml/min, the kidneys capacity to filter the anions of organic acids is significantly diminished and thus there is retention of phosphates, sulphates, urate and hippurate anions in the plasma that significantly raise the anion gap resulting in an elevated anion gap metabolic acidosis.

To summarize: Early chronic kidney disease is associated with a hyperchloremic normal anion gap metabolic acidosis while end stage renal disease (uremia) is associated with an elevated anion gap metabolic acidosis.



In some cases of end stage renal disease, patients may also present with elevated anion and normal anion gap acidosis simultaneously.

The acidosis associated with renal disease is usually not severe and this is due to the increased buffering of retained H⁺ ions by bone. This process is manifested by the release of calcium salts from bone and their excretion in urine. This calcium loss over time can lead to osteopenia.

Treatment

To prevent bone loss and possible osteopenia, alkali therapy is generally recommended even for mild metabolic acidosis associated with chronic renal disease. It is assumed that alkali replacement will prevent the harmful effects of prolonged positive H⁺ balance, including the persistent buffering of H⁺ ions by bone.

Oral alkali is typically used to maintain the [HCO₃⁻] over 20 meq/L. This can be accomplished with relatively modest amounts of alkali (1.0 to 1.5 mEq/kg per day). Usually this is amount of new bicarbonate generated each day.

Therapies include sodium bicarbonate and Shohl (sodium citrate). Sodium citrate (Shohl solution) has been shown to enhance the absorption of aluminum from the gastrointestinal tract and should never be administered to patients receiving aluminum-containing antacids because of the risk of aluminum intoxication.

< PREVIOUS

NEXT >