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Metabolic Acidosis

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Mini Review

Primarily it is the reduction of [HCO3-] concentration. There is a reduced serum [HCO3-] concentration with normal or appropriate PaCO2 reduction.

Base Deficiency takes two forms [1]:

A. Loss of HCO3- (diare etc.)

B. Titration of HCO3- with noncarbonic acids.

To distinguish these two etiological factors, "ANION GAP" is determined.

Anion gap = Major plasma cations - major plasma anions

= ([Na+] + [K +]) + ([HCO3 -] + [Cl-])

= (140 + 5) + (25 + 105) = 11-19 mmol / L

In reality, anion gap is not formed to maintain electronneutrality. In other words, the sum of all anions is equal to the sum of all cations. Unmeasured predominant cations (X) = Ca +, Mg +, gamma globulin

Unmeasured anions (Y) = albumin, phosphate, sulfate, lactate, weak acid salts all anions = all cations measurable anions + immeasurable anions = measurable anions + immeasurable anions

HCO3+ Cl- + Y = Na + K + X

(Na + K) - (HCO3- + Cl) = Y-X

Anion Gap = Y-X

Anion gap = anions not measured - non-measured cations Causes that increase the non-measured anions or reduce the non-measured cations increase the anion gap.

Anion gap = anions not measured - non-measured cations

Plasma albumin is responsible for the largest fraction of anion gap (11 mEq / L). Decreased anion gap is usually due to hypoalbuminemia and severe hemodilution. Anion gap corrected to hypoalbuminemia: anion gap can be calculated lower than expected due to hypoalbuminemia (eg salicylate intoxication + hypoalbuminemia). Adjusted Anion Gap = Detected Anion Gap + 2.5 (normal albumin level-detected albumin level)

Metabolic Acidosis with High Anion Gap [1]

When nonvolatil acids increase, they dissociate and give H + ions and their anions increase in the environment. When reacting with the H + HCO3- formed and producing CO2, its corresponding anion (conjugated base) accumulates. Consequently, the anion accumulates in the extracellular fluid and replaces HCO3- titrated with H + (anion gap).

Increased anion gap (> 12 mmol / L) (Normochloremic metabolic acidosis)

a. **Increased endogenous nonvolatil acid production**:

Lactic acidosis,

Diabetic ketoacidosis,

Mapple syrup,

Methylmalonic aciduria,

Starvation

b. **Insufficiency in endogenous nonvolatil acid**

excretion:

kidney orphan (GFR <20 ml / min)

c. Uptake of exogenous nonvolatil acids:

Salicylate toxicity

Methanol toxicity, formic acid

Ethylene glycol, glycolic acid

Blood transfusion (ACD)

d. Excess organic salt treatment:

Ringer lactate

Carbenicillin

High dose penicillin

Na acetate

e. Dehydration

Lactic Acidose [1]

In the increased anion gap, which cannot be explained by uremia or ketones, lactic acidosis is suspected.

Type A: why is insufficient tissue oxygenation. Type B: concerns abnormalities in prüvat metabolism. Lactathemia showing normal metabolic acidosis with normal anion gap can be detected in intensive care patients. This paradox is due to hypoalbuminemia, hyperchloremia, mixed acid-base disorder in this patient group. In starvation, a moderate ketoacidosis occurs with renal leakage of NaCl, K, Ca, phosphate, Mg. Organic acid filtration deteriorates when GFR is 20ml / min. Metabolic acidosis with Normal Anion Gap. It is typically associated with hyperchloremia. Plasma Cl- rises to replace HCO3- loss. Hyperchloremic acidosis is usually due to loss of HCO3 from GIS (diarrhea etc.) or renal leakage (renal tubular acidosis).

Normal Anion Gap (Hyperchloremic Metabolic Acidosis) [2]

Low Serum K:

a. HCO3- loss from GIS

b. Carbonic anhydrase inhibition (acetozolamide)

c. Urea diversion

d. Renal tubular acidosis

e. Long ileal loop

Serum K: N or higher:

i. Ammonium chloride

ii. Arginine chloride

iii. HCl

iv. Dilution (excessive application of bicarbonate-free liquids)

v. Obstructive uropathy

vi. Kr. Pyelonephritis

In acetozolamide areas and renal tubular acidosis; Renal loss of HCO3- increased due to the inability to absorb HCO3- from the kidney or lack of sufficient H + secretion in the form of titratable acid. Rapid extracellular volume loading with poor or normal saline from HCO3- causes dilutionary hyperchloremic acidosis; The general scenario is to use normal saline for resuscitation in trauma cases. Cationic amino acid solutions contain chloride as anion for their cations. Infusion of amino acid solutions can cause hyperchloremic metabolic acidosis.

Treatment in Metabolic Acidosis [3]

The primary cause should be treated with manifest acidemia. PaCO2 is targeted as 30 mm Hg, despite this compensation, if pH <7.2, alkaline treatment is indicated. When acidosis is normal anion gap, alkaline treatment is more inevitable. In High Anion Gap, as some of the unmeasured anions are converted to bicarbonate, partial recovery of acidosis is achieved. The need for treatment in kr acidosis (uremia, RTA) depends on the general condition of the patient and the symptoms of acidosis, beyond arterial pH.

Bicarbonate Treatment [3]

The amount of bicarbonate required depends on the degree of acidemia and the amount of total body fluid that the bicarbonate will disperse. Half of the deficit is given acutely, the other half is given at 8-12 hours

Example: 70kg of patients, [HCO3 -] = 14 mEq / L

What is the amount of [HCO3-] to be given acutely?

A. [HCO3-] deficit = normal value-current value

[HCO3-] gap = 24-14 = 10 mEq / L

B. Distribution volume = Total body weight x 0.5

Distribution volume = 70 x 0.5 = 35 L

C. Dose = Clear x Distribution x 0.5 = 10 x 35 x 0.5 = 175 mEq

Can be used in BE to calculate the required bicarbonate

NaHCO3 = BE x 30% x body weight

Salicylate intoxication,

pH = 7.32, PaCO2 = 30 mmHg, [HCO-3] = 15 meq / L

Purpose of treatment: If I aim to raise the pH to 7.45, to which level should I raise the plasma bicarbonate?

[H +] = 24 x (PCO2 / [HCO-3])

[H +] = 36 neq / L at pH = 7.45

36 = 24 x (30 / [HCO-3])

[HCO-3] = 20 meq / L

Since the reaction of bicarbonate with H + will form H2O and CO2, HCO-3 should be applied very carefully to patients with impaired respiratory function. HCO3- administration in type A lactic acidosis (hypoperfusion): controversial due to hypernatremia, hyperosmolarity and CSF acidification.

In this case can be used:

a. Carbicab: It is a mixture of 0.3M NaHCO3 and 0.3M Na carbonate. Offered as an alternative that does not produce CO2

b. THAM: It is stated that it can buffer metabolic and respiratory acids and decreases PCO2 as pH rises.

c. Dichloroacetate: not a buffer. It lowers the lactic acid level by stimulating the pruvant dehydrogenase, which converts the private into acetyl CoA.

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