Acid-Base balance

Learning objectives

Mechanisms and importance of strict acid base balance-1

Types and causes of acid base disturbance-2

Management of acid base defect-3

Acid- base balance:

Arterial blood PH **(7.36-7.44)= 7.40**

H^+ conc. range **(36 – 44 nmol/L).**

* This tight haemostatic mechanism is important for the function of many PH-sensitive enzymes.

* The most important buffer system is the carbonic acid/ bicarbonate buffer system.

* Bicarbonate conc. in ECF **(21-28 mmol/L).**

C.a

$Co_2 + H_2O \leftrightarrow H_2Co_3 \leftrightarrow H^+ + HCo_3^-$

The Co_2 source is the lung and HCO_3^- from the kidneys.

PH= 6.1 + log[Hco3]/0.03*pco2

*The above called Henderson – Hasselbalch equation.

*Respiratory compensation for acid- base disturbance can occur quickly, due to alternation in ventilatory drive mediated through PH changes in the brain stem. Acidosis lead to ventilation and reduce PCO_2 and hence the PH. Conversely systemic alkalosis leads to inhibition of ventilation (although this is limited by the development of hypoxia).

*Acidosis due to chronic respiratory or metabolic (non-renal) cause enhance urinary excretion of acid, and increasing the plasma bicarbonate.

C/F:

1-Tissue malfunction due to disturbed PH (such as altered cardiac and CNS function such as hypotension, headache, lethargy, drowsiness, CNS depression).

2- Secondary changes in respiration (Kuassmaul respiration).

3-Clinical picture of the underlying cause e.g. D.M. or lung disease.

* These clinical presentation only become evident when the venous plasma bicarbonate concentration is noted to be abnormal, or when a full blood gas analysis shows abnormalities in PH, PCO_2 or bicarbonate.

* In metabolic disturbance, respiratory compensation is almost

immediate. So that the predicted compensatory change in PCO_2 is achieved soon after the onset of the metabolic disturbance.

* In respiratory disorders, a small initial change in bicarbonate occurs as a result of chemical buffering of CO_2, largely within red blood cell, but the kidney achieves further compensatory changes in bicarbonate concentration as a result of long term adjustments in acid secretory capacity, requiring days to weeks.

Metabolic acidosis:

Causes

A-Normal anion gap (where mineral acid"HCl" accumulates, or where there is a primary loss of bicarbonate buffer from the ECF, there is no addition to the plasma of a new acidic anion [$(Na^+ + K^+)$ – $(Cl^- + HCO_3^-)$]. The plasma chloride increases to replace the depleted bicarbonate levels.

* This gap normally around 15 mmol/L.

1- Inorganic acid addition: the therapeutic infusion or poisoning with NH_4Cl, HCl.

2- G.I base loss: diarrhea, small bowel fistula, urinary diversion procedure.

3- Renal tubular acidosis(RTA):

A- Proximal RTA (type 2): congenital e.g Fancon's syndrome, Wilson's disease, paraproteinaemia e.g myeloma, Amyloidosis, hyperparathyroidism, heavy metal toxicity, drugs (carbonic anhydrase inhibitor).

B- Classical distal RTA (type 1): congenital, hyperglobulinaemia, autoimmune connective tissue diseases e.g SLE. Toxins and drugs (amphotericin).

C- Hyperkalaemic distal RTA (type 4): hypoaldosteronism, obstructive uropathy, drugs e.g amiloride, spironolactone, renal transplant rejection.

B-Increased anion gap:

* Endogenous acid load: diabetic ketoacidosis, starvation ketosis, lactic acidosis, renal failure.

* Exogenous acid load: aspirin, methanol, ethylene glycol poisoning.

(normal maximal level of plasma lactate 2mmol/L):

1- Type 1 lactic acidosis: due to tissue hypoxia as in patients with circulatory failure and shock.

2- Type 2 lactic acidosis: as in liver disease, drugs and toxins such as metformin.

* In renal tubular acidosis, there is a hyperchloraemic (normal anion gap) acidosis with no evidence of G.I disturbance, and the urine PH is inappropriately (greater than 5.5 in the presence of systemic acidosis).

In proximal RTA there is defect in bicarbonate reabsorption.

In distal RTA there is defect in acid secretion.

Proximal RTA associated with urinary wasting of amino acids, phosphate, and glucose "fanconies syndrome" as well as bicarbonate and potassium.

Management:

1- Sodium and water replacement.

2-Intravenous bicarbonate for severe acidosis PH < 7.15, tissue dysfunction, and when the underlying disorders cannot be readily corrected, because rapid correction lead to hypokalemia and hypocalcaemia.

Metabolic Alkalosis:

* Characterized by an increase in the plasma bicarbonate concentration and the plasma PH.

•There is compensatory rise in PCO_2 due to hypoventilation. The causes are best classified by the accompanying disturbance of ECF volume.

1- Diuretic drugs (other than carbonic anhydrase inhibitors, and potassium – sparing drugs).

2-Normovolaemic or hypervolaemic metabolic alkalosis such as corticosteroid excess as in primary hyperaldosteronism (Conn's syndrome), Cushing syndrome, corticosteroid therapy, and overuse of antiacid salts.

Management:

1-Isotonic sodium chloride.

2-Potassium replacement.

3-In metabolic alkalosis with hypervolaemia treating the underlying cause especially removal of the mineralocorticoid activity.

Respiratory acidosis:

Increase CO_2 with compensatory increase in plasma bicarbonate this can arise from lesions of the neuromuscular pathway, it lead to drawzness.

Management ▭ correction of the causative factors where possible, external ventilatory support.

:Respiratory alkalosis

hyperventilation with reduced PCO_2 and increase in plasma PH. Renal compensation occurs if the condition is sustained leading to reduces tubular acid secretion and falls in plasma bicarbonate. It occur in anxiety, assisted ventilation, pregnancy, pulmonary embolism, chronic liver disease, certain drugs such as salicylate. Clinical features (include agitation, digital tingling, .trousseau's sign, and Chvostek's sign and tetany or seizures)