Medicine

Nephrology

5th year – lecture 7

الدكتور

مُحَمَّد يونس العطبي

الاختصاص الدقيق بأمراض وزرع الكلى

اختصاص الطب الباطني

MBChB, FICMS (Medicine), FICMS (Nephrology), CABMS (Nephrology)

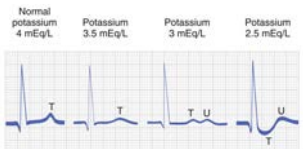
HYPOKALEMIA

Basic Information

- Serum K+ concentration less than 3.5 mEq/L
- Clinical presentations : Fatigue, myalgia, muscle cramps, constipation, proximal muscle weakness. If severe: arrhythmias , and rarely ascending paralysis with eventual respiratory impairment.
- Chronic long standing hypokalemia can causes: **(1)** hypokalaemic nephropathy) and interferes with the tubular response to ADH (acquired nephrogenic diabetes insipidus), resulting in polyuria and polydipsia. **(2)** Increasing BP(HT) . **(3)** glucose intolerance(DM).
- **Causes** :

1) **Redistribution hypokalemia (**Redistribution into cells**)** : Insulin excess B_2 –agonists as ventolin inhalor .
2) **Decrease K^+ intake**
3) **Excessive K^+ losses: GI loss :**Vomiting ,Diarrhoea .
   Renal loss (Urine K> 20mmol/day) : Check BP ➔**With normal blood pressure**: Diuretic therapy (loop and thiazide) ,Bartter's and Gitelman's syndromes , or Renal tubular acidosis (types 1 and 2) , ➔ **With hypertension** : primary Hyperaldosteronism (Conn's syndrome) , Cushing's syndrome, Liquorice/carbenoxolone , Liddle's syndrome Renovascular hypertension.

- **Diagnosis** : serum potassium , magnesium , bicarbonate, urine potassium , **ECG changes** are more predictive of clinical picture than serum [K^+] ➔ U waves most important (low amplitude wave following a T wave) ,➔ fattened or inverted T waves, depressed ST segment ,➔Prolongation of Q-T interval, P-R prolongation, wide QRS , Arrhythmias .**Treatment:** treat the cause , potassium repletion give KCL , K^+-sparing diuretics (spironolactone, amiloride , triamterene) can prevent renal K^+ loss . Restore Mg^{2+} in hypomagnesaemia.



Hyperkalaemia

Basic Information

- Serum K+ concentration greater than 5.5 mEq/L
- Clinical presentation : usually asymptomatic but may develop nausea, palpitations, muscle weakness, muscle stiffness, paresthesias, arefexia, ascending paralysis, and hypoventilation
- **Causes** :

1) **Redistribution hyperkalemia (**Redistribution out of cells**)** : Insulin deficiency ,β-blockers , Acidosis .
2) **increase K^+ intake :** Exogenous (diet, IV therapy) , Endogenous (haemolysis, rhabdomyolysis)
3) **Renal retention of K: check renal function ➔ renal failure :**AKI , CKD . **check serum aldosterone ➔With low aldosterone**: as Addison's disease ,Drugs (NSAIDs, ACE inhibitors, β-blockers, ciclosporin, prolonged heparin therapy) , **➔ With normal / high aldosterone**: as Tubular transport defects as Tubulointerstitial disease, Drugs (amiloride, spironolactone)

- **Diagnosis** : serum potassium , renal function test, bicarbonate, **ECG changes:** do not correlate well with serum [K^+] , Peaking of the T wave is an early ECG sign (tented T wave), Decreased amplitude of P waves and eventual absent of P waves, prolonged PR interval ,widening of QRS and eventual merging with T wave (**sine-wave pattern**) ,AV block , ventricular fibrillation, asystole . **Treatment: Acute therapy** is warranted if ECG changes are present or if patient is symptomatic hyperkalemia .

Treatment of Hyperkalemia

Mechanism	Therapy	Dose	Onset	Duration
Antagonize membrane effects	Calcium	Calcium gluconate, 10% solution, 10 ml IV over 10 min	1-3 min	30-60 min
Cellular potassium uptake	Insulin	Regular insulin, 10 U IV, with dextrose 50%, 50 ml, if plasma glucose <250 mg/dl	30 min	4-6 hr
	β_2-Adrenergic agonist	Nebulized albuterol, 10 mg	30 min	2-4 hr
Potassium removal	Sodium polystyrene sulfonate or calcium polystyrene sulfonate (calcium resonium)	30-60 g PO in 20% sorbitol *or* 30-60 g in water, per retention enema	1-2 hr	4-6 hr
	Hemodialysis	—	Immediate	Until dialysis completed

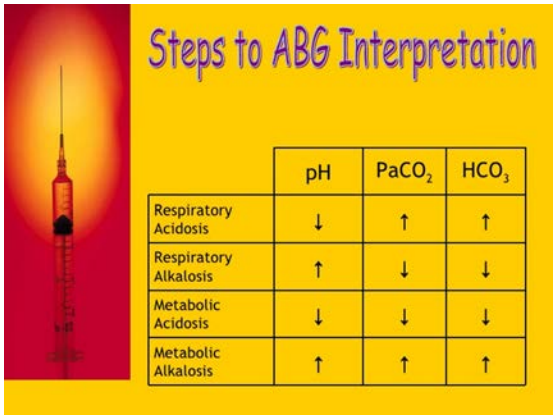
Chronic therapy : 1)**Enhance K^+ Removal from Body ➔ via urine** (preferred approach) :furosemide, if suspect aldosterone defciency GIVE fludrocortisones (synthetic mineralocorticoid). **➔via gastrointestinal tract** :cation-exchange resins: calcium resonium .Novel potassium binders (patiromer or sodium zirconium cyclosilicate) may be better tolerated

2) **dialysis** (renal failure, life threatening hyperkalemia unresponsive to therapy)

DISORDERS OF ACID–BASE BALANCE

Basic Information

- The concentration of hydrogen ions in both extracellular and intracellular compartments is extremely tightly controlled, and very small changes lead to major cell dysfunction.
- Normal values in midrange: normal concentration of HCO_3 = 24 mEq/L (range: 22-28 mEq/L) ,normal PCO_2 = 40 mmHg (range: 35-45 mmHg) , normal blood PH of 7.40 (range: 7.35-7.45)
- Simple approach to **arterial blood gas** (ABG) as following arrows methods :**ROME** (Respiratory opposite , Metabolic equal)



Metabolic Acidosis: occurs if the systemic PH falls <7.4 and is considered metabolic in origin if decrease bicarbonate [HCO_3^-] <24 mEq/L. With pure metabolic acidosis, compensation occurs through increasing ventilation and blowing off CO2 (decrease PCO_2 <40 mmHg).**Causes** according to **AG (anion gap)=Na – (bicarbonate +chloride)** , normal value 10-12

- **High AG metabolic acidosis:** lactic acidosis, ketoacidosis (as DKA) ,uremic toxins ,others as ethylene glycol poisoning ,methanol poisoning , salicylate (e.g. aspirin) overdose.
- **Normal AG Metabolic Acidosis (Hyperchloremic Metabolic Acidosis)** : either due to diarrhea (HCO_3^- loss from GI tract) , or renal tubular acidosis (RTA)

Treatment: treat the cause, use of bicarbonate ($NaHCO_3$) infusions is best reserved for situations where the underlying disorder cannot be readily corrected (note that lactate and ketoacid can be metabolized to HCO_3) and acidosis is severe (pH < 7.00). It can be taken orally as tablets or powder or given intravenously as a hypertonic bolus or an isotonic infusion, which can be created by adding 75 mmol $NaHCO_3$ to 500ml of 5% dextrose in water (D5W). Thus the bicarbonate deficit, in millimoles, can be estimated from the following formula: **Bicarbonate deficit** = 0.5× wt(kg) × (target HCO_3 –patient HCO_3) . When acute treatment is desired, 50% of the bicarbonate deficit should be replaced during the first 24 hours. Side effects of $NaHCO_3$: hypokalemia and fluid overload .

Metabolic alkalosis is characterised by an **increase** in the plasma bicarbonate concentration and the plasma pH . There is a compensatory **rise in PCO_2** due to hypoventilation associated with mortality as high as 45% . Causes of a metabolic alkalosis include:

Table. Chloride-responsive and -resistant causes of metabolic alkalosis

CHLORIDE-RESPONSIVE	CHLORIDE-RESISTANT
Vomiting	Cushing's disease
Aspiration	Renal artery stenosis
Diuretics	Diuretics
Zollinger-Ellison syndrome	Magnesium deficiency
Bicarbonate therapy	Renal failure and bicarbonate therapy
Potassium deficiency	

Clinical features: Patients with serum [HCO3] levels as high as 40 mmol/l are usually asymptomatic. With more severe metabolic alkalosis (serum [HCO3] >45 mmol/l), arterial Po_2 often falls to less than 50 mm Hg secondary to hypoventilation, and ionized calcium decreases (due to alkalemia). The adverse effect of most concern is hypokalemia, which increases the likelihood of cardiac arrhythmias in patients with underlying cardiac dysfunction.

Treatment

1. Saline sensitive metabolic alkalosis (most common): Metabolic alkalosis with hypovolaemia can be corrected by intravenous infusions of 0.9% saline with potassium supplements. This reverses the secondary hyperaldosteronism and allows the kidney to excrete the excess alkali in the urine
2. In metabolic alkalosis with normal or increased volume, treatment should focus on management of the underlying cause , replenish K^+ and Mg^{2+} deficits .Carbonic anhydrase inhibitor (e.g. acetazolamide) to facilitate loss of HCO_3^- in urine in metabolic alkalosis and heart failure.

MIXED ACID-BASE DISTURBANCES: defined as the simultaneous presence of two or more acid-base disorders.

1) If patient have metabolic acidosis (low PH, low HCO_3)➔ first calclutate anion gap ➔ 2nd calculate expected PCO_2 =15+ HCO_3 , if patient PCO_2 higher than expected that mean (**mixed metabolic acidosis with respiratory acidosis**) or if patient PCO2 lower than expected that mean (**mixed metabolic acidosis with respiratory alkalosis**).
2) If patient have metabolic alkalosis (high PH, high HCO_3) ➔ first calculate expected $PCO_2 = 40 + 0.7 \times (HCO_3 - 24)$, if patient PCO_2 higher than expected that mean (**mixed metabolic alkalosis with respiratory acidosis**) or if patient PCO_2 lower than expected that mean (**mixed metabolic alkalosis with respiratory alkalosis**).