Case 2: Hypokalaemia in Metabolic Acidosis

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Case History

Analyte	Result	Reference Interval
Sodium	138 mmol/L	135 – 145
Potassium	1.51 mmol/L	3.5 – 5.1
Chloride	107.6 mmol/L	94 – 110
Urea nitrogen	13.1 mg/dL	7-18.7
Creatinine	0.7 mg/dL	0.57-1.11
e-GFR	111.9	≥90
Arterial blood gas		
pH	7.33	7.35-7.45
pCO2	30 mmHg	35.00-45.00
pO2	75 mmHg	80.00-100.00
Bicarbonate	15.8 mmol/L	22.00-26.00
Potassium	1.2 mmol/L	3.5-5.1

Questions

1. What is the most critical result?
2. What possibilities could you offer to explain the potassium result?
3. What are the causes of hypokalaemic in metabolic acidosis?
4. Are there any other tests you might perform to clarify the likely cause of the low potassium result?

Discussion

Question 1

The most critical result is the potassium level. Very low potassium levels (less than 2.5 mmol/L) can be life-threatening and require immediate medical attention. Significant muscle weakness occurs at serum potassium levels below 2.5 mmol/L. It is an ascending pattern affecting the lower extremities, progressing to involve the trunk and upper extremities and potentially progressing to paralysis. Affected muscles can include respiratory muscles, which can lead to respiratory failure and death. Severe hypokalaemia can also lead to various cardiac dysrhythmias.

Question 2

Hypokalaemia can be caused by an intracellular shift of potassium, a decrease in potassium intake, and an increase in potassium output. Some causes of hypokalaemia can usually be determined through patient history, such as hypokalaemia due to vomiting, diarrhea, or diuretic use. However, in some cases, the cause may not be apparent, and establishing a diagnosis can be challenging (1). The cause of transient hypokalaemia may be due to a shift of potassium into the cells, while ongoing hypokalaemia may be due to insufficient potassium intake or excessive potassium loss. Excessive potassium loss can result from disorders in the kidneys or outside the kidneys (1, 2).

Question 3

The combination of hypokalemia and normal anion gap metabolic acidosis can be caused by renal and extrarenal losses of potassium and bicarbonate. The most common extrarenal causes are from the gastrointestinal tract in the presence of diarrhea and vomiting and the use of diuretics. Renal causes of hypokalaemia in a normal anion gap metabolic acidosis can be due to renal tubular acidosis, diabetic ketoacidosis, use of medications such as amphotericin B and acetazolamide. The approach to hypokalemia patients with metabolic acidosis can use a flowchart based on Figure 1.

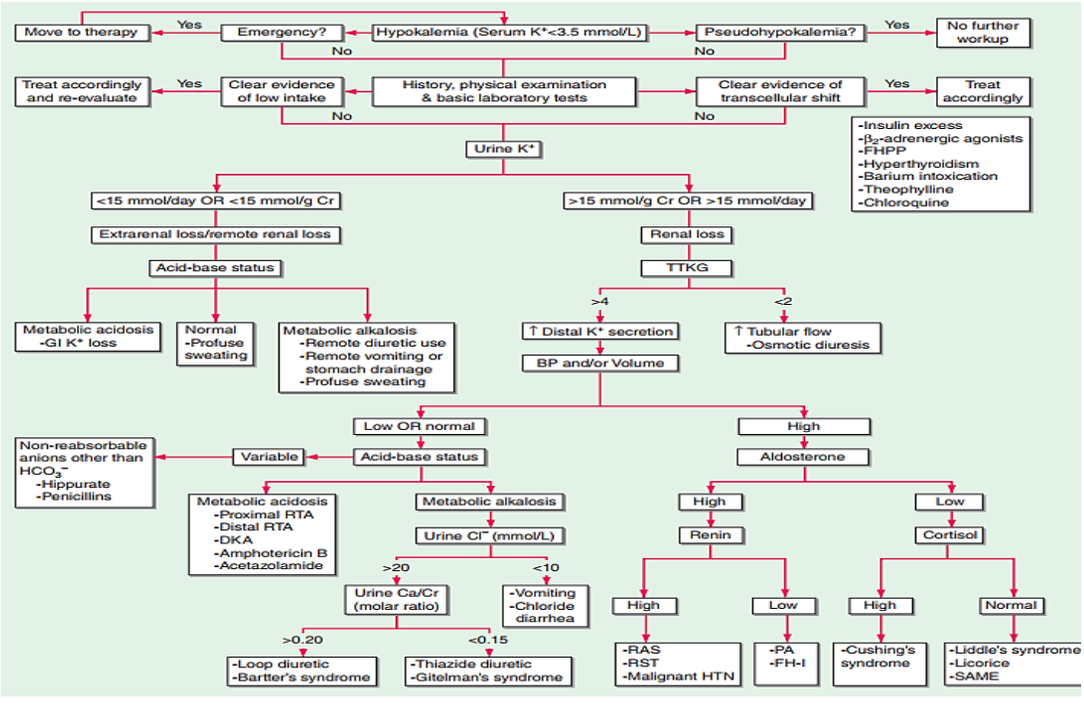

Figure 1. Approach to patients with hypokalaemia and metabolic acid-base disorders (1).

Question 4

The approach to differentiate between renal and extra-renal causes of hypokalemia can be made by measuring potassium excretion in a 24-hour urine sample or looking at random urine potassium concentration values. If the urine potassium concentration exceeds 15 mmol/day, this suggests a renal disorder as the cause. Conversely, if the urine potassium concentration is less than 15 mmol/day, there may be an extra-renal cause (1).

Assessment of potassium excretion in urine can also be done using Trans-tubular Potassium Concentration Gradient (TTKG) calculation. The trans-tubular Potassium concentration Gradient provides an estimation of potassium levels in the tubular fluid, especially at the end of the cortical collecting duct. The cortical collecting duct is the last place where potassium levels in urine are determined. The TTKG formula used is based on several references: (3, 4).

$$\mathbf{TTKG} = \frac{\mathbf{K^+(urine)x\ Osmolality\ (serum)}}{\mathbf{K^+\ (serum)x\ Osmolality\ (urine)}}$$

The following diagnostic steps performed in patients with hypokalemia are blood pressure checks and assessment of acid-base status to narrow down the differential diagnosis.

If renal tubular acidosis is suspected, additional laboratory tests, such as plasma anion gap, are performed. The plasma anion gap is the calculated result between cations and anions in plasma (including sodium, potassium, chloride, and bicarbonate) (5). Examination of urine NH4+ excretion can be used as the next step to establish the diagnosis of possible RTA. NH4+ excretion tends to be high in extrarenal loss and low in RTA. Although clinical laboratories do not routinely measure urine NH4+ levels, estimation of NH4+ excretion can be done by calculating the anion gap of urine (AGU) (1). After calculating the urine anion gap to narrow down the differential diagnosis, urine pH is examined (6, 7).

References

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