A REVIEW ON SERUM POTASSIUM ABMORMALITIES IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Abstract : Potassium plays an important role in cellular metabolism and normal neuromuscular function. Potassium balance has been a major role of kidney by increasing or decreasing the rate of potassium excretion. Serum potassium abnormalities is multifactorial. In renal impairment patients, the most common problem observed is hyperkalemia.The prevalence of hyperkalemia in chronic kidney disease is high. Hyperkalemia is a life-threatening severity that affects the cardiac conducting tissue leading to cardiac arrest. Therefore, early detection and monitoring of hyperkalaemia is necessary. It is important to treat hyperkalemia in emergency department. Various therapeutic approaches have been developed for the treatment of hyperkalemia in chronic kidney disease patients. The aim of the review is to identify the causes of hyperkalemia, differential diagnostic methods and various therapeutic approaches in chronic renal failure patients.

IndexTerms **- Hyperkalemia, chronic renal failure, cardiac arrest, potassium.**

INTRODUCTION

Potassium is the most important intracellular cation that helps the nerves to function and muscles to contract. Multifactorial effects can cause variation in potassium concentration such as insulin deficiency, uremia, acidosis, aldosterone deficiency, osmolarity disturbances (hyperglycemia)and renal failure.[1] Kidneys ability to intake potassium and excrete potassium may be affected by disease conditions such as heart failure, chronic kidney disease, diabetes mellitus and also drug therapy.[7]
In chronic kidney disease, hyperkalemia is caused due to kidney dysfunction on potassium homeostasis. Hyperkalemia is a condition which is defined as serum potassium concentration greater than 5.0mEq/L.[2] It is associated with fatigue and muscle weakness to the risk of death due to arrythmia and act as major driver to start dialysis in end stage renal disease (ESRD).

In maintenance hemodialysis, hyperkalemia or hypokalemia may occur due to changes in dietary intake and excretion of potassium. Hypokalemia is a relatively rare event comparing to hyperkalemia. Hyperkalemia primarily related to poor dietary compliance such as too much potassium intake, inadequate dialysis due to noncompliance or vascular access problems, medications such as ACEIs, potassium sparing diuretics, non-selective beta blockers, NSAIDs, and unfractionated heparin use in ESRD patients undergoing maintenance hemodialysis.[3-4]

PATHOPHYSIOLOGIC MECHANISM

Hyperkalemia can result due to the following reasons:

Excessive intake of potassium: In patients with GFR <15ml/min ,a slight increase in potassium intake can lead to hyperkalemia.

Decreased excretion

1)Renal insufficiency: With low urine flow which may cause low sodium transport to distal tubule lead to decreased excretion of potassium.

2)Medications: Combined treatment of spironolactone (potassium sparing diuretics) and ACE inhibitors in patients with renal impairment may lead to hyperkalemia.

3)Secondary hypoaldosteronism: urinary salt wasting leading to volume depletion and hypotension may also causes hyperkalemia.[9]

Shift of potassium from intracellular to extracellular compartment

1)Acidosis: In chronic renal failure, impaired ammonia excretion, decreased tubular reabsorption of bicarbonate and insufficient production of bicarbonate lead to acidosis which causes shift of potassium from intracellular to extracellular space.

2)Diabetic nephropathy: Decreased insulin levels cause accumulation of potassium in extracellular space.

3)Hyperosmolality: Conditions like hyperglycemia causes potassium to exit from cells.[10]

DIAGNOSIS

Hyperkalemia is clinically manifested as palpitations, nausea, muscle pain, or paraesthesia. It is assessed by kidney and heart function, urinary tract, hydration status and neurological evaluation. Electrocardiography (ECG) is also an important monitoring in patients with higher potassium level (>6.5mmol/L). ECG changes may present as peak T waves, QRS widening as well as depression of ST segment. Laboratory investigations includes urea, creatinine, potassium and others.[5]

Characteristic electrocardiographic (ECG) changes associated with hypokalemia include broad, flat T waves, ST depression, the appearance of U waves, QT interval prolongation, and finally ventricular arrhythmias leading to cardiac arrest. When serum [K] is less than 3.0mEq/L, generalized weakness can develop and serum [K] decreases to less than 2.5mEq/L, muscle necrosis and rhabdomyolysis can occur.[6]

Acute acidemia is a well-known cause of true hyperkalemia. Two mechanisms are involved: first, acute acidemia inhibits renal potassium secretion; second, as acidosis develops, hydrogen ions move into cells and this shift is associated with an efflux of potassium from cells.[8]

MANAGEMENT

Therapeutic management

It should be individualized on the basis of degree and cause of hyperkalemia. It should be guided by the clinical aspect and serial potassium measurements. Most therapies for hyperkalemia need transitory improvement by shifting potassium into intracellular space without removal of potassium.[11]

Insulin: It appears to have rapid action by shifting potassium into cells by stimulating the activity of sodium-potassium antiporter on cell membrane ,promoting the entry of sodium into cells by activation of Na+-K+ ATPase causing electrogenic influx of potassium.

IV dextrose : It is given to stimulate endogenous insulin production and thereby reduces serum potassium in hemodialysis patients.

IV calcium: It is indicated when the serum potassium is less than 6.5 mEq/L. Calcium increases the threshold potential by antagonising the effect of hyperkalemia .IV calcium can be given as 10ml of 10% of calcium gluconate over 2-3 min or as calcium chloride ,which contains 3 times the amount of calcium per 10 ml dose.[12-14]

Sodium bicarbonate: It works to shift potassium intracellularly but it is not considered as first line management ,boluses of 1 ml/kg of sodium bicarbonate 8.4% solution can be given.

Sodium zirconium cyclosilicate (ZS-9) : It is an insoluble non absorbed compound designed to capture potassium ions ,at initial dose of 1.25,2.5 ,5g or 10g times /day with meals for 48 hours.

Beta 2 agonist : Albuterol inhalation can be considered to lower potassium ,it stimulates NA+ -K+ ATPase ,which result in intracellular shift of potassium .Doses from 10-20 mg are given for hyperkalemia.

Diuretics : In patients with adequate kidney function, loop diuretics in combination with thiazide diuretics can be used for the excretion of potassium

Sodium polystyrene sulfonate (SPS): SPS is a cation exchange polymer that exchange sodium for potassium. It should be given as 15-30 gm by mouth with cathartics or as an enema. The rectal dose is 30-50g.

Dialysis: Hemodialysis is a method of removal of potassium when pharmacological therapies fails to eliminate potassium .Potassium could be decreased by >1 mEq/L within 60 minutes and by a total of 2 mEq/L within 180 minutes.[15]

Newer treatment options: Management of hyperkalemia has been added with the approval of a new drug in 2015. Patiromer is an option for chronic hyperkalemia in patients with CKD and on RAAS inhibitors especially those who benefit from continuation of therapy due to comorbid condition such as diabetes mellitus and heart failure. [16] It should be initiated at 8.5 gm daily, it is a powder for suspension in water for oral administration .

Suppportive Treatment : Dietary modifications which includes avoidance of coconut containing foods (such as coconut water ,coconut oil) ,canned foods etc. [17] Avoidance of medications such as potassium supplements,digoxin,ACE inhibitors or ARB inhibitors , amiloride, triamterene which worsens the risk for hyperkalemia.[18]

CONCLUSION

Serum potassium abnormality have been commonly observed among chronic renal failure patients ,in which hyperkalemia is the most silent and life threatening severity that may even lead to cardiac death.[19] Therefore effective and rapid diagnosis and management of hyperkalemia is clinically relevant and life saving. In order to that appropriate treatment should be implemented for moderate to severe hyperkalemia, combination of medication with different therapeutic approaches and in accordance with other co morbid conditions of the patient.[20]

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