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Review Article

MANAGEMENT OF SEVERE HYPERKALEMIA

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Since hyperkalemia can result in cardiac arrest, specific therapy directed toward rapid reduction of serum potassium level is of utmost importance in preventing this potentially fatal condition. Because hyperkalemia is reversible, physicians managing patients with cardiac arrest should maintain a high index of suspicion for the presence of hyperkalemia as a cause of cardiac arrest. Many patients who experience hyperkalemic cardiac arrest do not have primary end-stage cardiac disease, therefore the opportunity for survival is greater in these patients. There are reports of patients who experienced hyperkalemic cardiac arrest with prolonged periods of asystole or ventricular fibrillation and survived with full neurologic recovery after appropriate management of hyperkalemia combined with the recommended cardiopulmonary resuscitative measures were instituted.

Severe hyperkalemia has been reported in a wide range of clinical settings (Table I) from the common, such as severe chronic renal failure (GFR < 15-20 ml/min) and diabetic ketoacidosis, to the more unusual, such as intravenous administration of potassium penicillin G (contains 1.6 meq $K/10^6$ units of penicillin).[1] Other causes include initiation of angiotensin converting-enzyme inhibitor (ACEI) therapy,[2] ingestion of excess amounts of KCl or potassium iodide (salt-substitute) in the presence of other predisposing factors (e.g., renal failure, congestive heart failure, ACEI),[3,4] relief of acute arterial occlusion,[5] administration of depolarizing neuromuscular blocking drugs (e.g., succinylcholine) during induction of anesthesia in patients with paraplegia/quadriplegia,[6-8] massive trauma or burns, rapid transfusion of stored blood,[9,10] therapy of leukemia or lymphoma,[11,12] or geophagia in patients with chronic renal failure (river bed clay contains as much as 1 meq potassium per gram).[13] Another cause of hyperkalemia is obstructive uropathy resulting in hyperkalemic distal renal tubular acidosis (voltage defect-d.RTA) and a hyporenin-hypoaldosterone or aldosterone-resistant state (type 4 RTA), conditions commonly present simultaneously.[14]

The hyperkalemia can actually worsen during the cardiac arrest, as a very large potassium efflux has been shown to occur during closed cardiopulmonary resuscitation.[15] This is thought to be due to ischemia-induced depletion of cellular adenosine triphosphate (ATP) stores which are required for normal functioning of Na,K-ATPase pump and maintenance of cellular ionic homeostasis. In the absence of ATP large quantities of potassium leak out of the cells along its concentration gradient.

Hyperkalemic cardiac arrest should be suspected when cardiac arrest occurs in a patient with severe renal insufficiency (GFR<15-20 ml/min). The probability of hyperkalemic cardiac arrest is even higher if the patient has been receiving potassium-sparing diuretics (e.g., triamterene, spironolactone, amiloride), potassium supplement, or any of the conditions mentioned earlier.

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Early EGG changes[16,17] which should alert the physician to the possibility of significant hyperkalemia include tenting of the T waves (K~ 5.5-6.0 mmol/L) best seen in the precordial leads and occasionally associated with some ST depression, prolonged PR interval, and widening of the QRS complex (K~ 6.0-7.0 mmol/L). Later changes include progressive flattening of the P wave with further widening of the QRS complex (K~7.0-7.5 mmol/L), absence of P wave and widened QRS complex merged with the T wave to produce a "sine wave" pattern (K~8.0 mmol/L). The latter pattern is frequently misinterpreted as ventricular tachycardia.

In addition to the classical sequence of events outlined above, virtually any arrhythmia or conduction disturbance may be seen in hyperkalemic patients. Various forms of fascicular and AV nodal block are particularly common. It is important to recognize that if hyperkalemia develops slowly (e.g., chronic renal failure), cardiac manifestations may be minimal or even absent despite serum potassium of 7.0-7.5 mmol/L. In rare cases, the ECG may be normal or near normal despite a marked elevation in the serum potassium to approximately 9 mmol/L.[18] In contrast, a rapid rise in serum potassium,[19,20] concomitant hypocalcemia,[21] hyponatremia,[22] or acidemia[19] may potentiate the cardiotoxic effects of hyperkalemia so that severe cardiac toxicity may be observed with serum potassium concentration of 6.0-6.5 mmol/L. Conversely, hypernatremia and hypercalcemia may counteract the membrane changes of hyperkalemia, thereby minimizing its cardiotoxicity.[18] Cardiac toxicity may develop without any premonitory changes in the ECG, and a perfectly normal EGG may degenerate directly to ventricular tachycardia or asystole, the terminal events in the course of hyperkalemic cardiotoxicity. During the hyperkalemic cardiac arrest, ECG usually shows either a bradycardia with wide QRS complex, a "sine wave" pattern, ventricular fibrillation, or asystole.

In the management of hyperkalemic cardiac arrest, the usual measures for the treatment of cardiac arrest (chest compression, airway management, atropine, epinephrine, cardiac pacing) should be administered concomitant with targeted treatment of the hyperkalemia, in particular, an immediate infusion of calcium gluconate. While epinephrine drives potassium into the cells and hence lowers serum potassium concentration within several minutes, calcium gluconate will immediately antagonize the cardiotoxicity of hyperkalemia.

Specific treatment of hyperkalemia includes measures to oppose the effects of hyperkalemia at the level of cell membrane, to reduce its plasma concentration by increasing its influx into the cells, and finally to remove it from the body pool. The first goal (membrane stabilization) is achieved by administration of calcium or hypertonic saline (if patient is hyponatremic). The second goal (transfer of potassium from the extracellular to the intracellular compartment) is achieved by administration of glucose and insulin, sodium bicarbonate, or β2 adrenergic agonists. The final goal (reduction of total body potassium) is achieved by hemodialysis/pertioneal dialysis, cation-exchange resin (Kayexalate) or diuretics.

Calcium infusion will immediately antagonize the cardiac effects of hyperkalemia, even in the normocalcemic patients.[16,17] This immediate protective effect is due to the fact that while hyperkalemia reduces the magnitude of the resting potential, calcium lowers the threshold potential, and as a result normalizes the membrane excitability.[24] Conversely, hypocalcemia raises the threshold potential and enhances the cardiotoxicity of hyperkalemia.[21] Thus, restoration of a normal serum calcium concentration in the latter group will have a dramatic effect to reverse the manifestations of cardiac toxicity. The beneficial effect of calcium begins within 1-3 minutes, but is relatively short lived (30-60 minutes). Infusion of calcium should be the first step in the management of severe potassium cardiotoxicity, as other temporizing measures (glucose plus insulin, $NaHCO_3$, etc.) may take up to 30-60 minutes to begin action. The usual dose is 10ml of 10% calcium gluconate solution infused over 2-3 minutes, and can be repeated after 5-10 minutes. Because hypercalcemia potentiates the cardiotoxicity of digitalis, calcium should be used only when absolutely necessary (loss of P waves or widened QRS complex) in patients taking digitalis. In this situation, it should be added to 100 ml of 5% dextrose in water and infused slowly over 20-30 minutes to permit a more evenly distribution throughout the extracellular space.

Since hyponatremia increases the cardiotoxicity of hyperkalemia, administration of *hypertonic saline* (50-100 meq of NaCl) to such patients may have a dramatic effect in reversing the electrocardiographic changes.[22] The beneficial effect of hypertonic saline is less predictable in patients with a normal serum sodium concentration. The onset of the beneficial effects is after 5-10 minutes and lasts for approximately 2 hours. Recently, sodium bicarbonate has largely replaced hypertonic saline because hyperkalemia is frequently associated with acidemia.

Sodium bicarbonate infusion shifts potassium from the extracellular into the intracellular space, thereby reducing serum potassium concentration. This is achieved by increasing blood pH and serum bicarbonate concentration. Increasing the pH exerts its effect independent of the change in serum bicarbonate.[25]

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Table I. Causes of hyperkalemia

1. Factitious
   a. Laboratory error
   b. Pseudohyperkalemia: In-vitro hemolysis, leukocytosis, thrombocytosis
2. Increased input / release
   a. Exogenous: High K diet, salt substitute, penicillin v-K
   b. Endogenous: Crush injury, tumor lysis, gastrointestinal bleed, catabolic states
3. Renal failure
   a. Acute
   b. Chronic (GFR<15-20 ml/min.)
4. Inadequate distal tubular delivery of Na
   a. Severe congestive heart failure
   b. Liver cirrhosis
   c. Severe volume depletion
5. Impaired renin-angiotensin system
   a. Addison's disease
   b. Primary hypoaldosteronism
   c. Primary hyporeninism
   d. Angiotensin deficiency or unresponsiveness
   e. Drugs: Heparin, ß-blockers, NSAID*, ACEI†,
6. Renal tubular disease (K secretory defect)
   a. Sickle cell disease
   b. Systemic lupus erythematosus
   c. Obstructive uropathy
   d. Amyloidosis
   e. Renal transplant
   f. Drugs: Spironolactone, triamterene, amiloride
7. Abnormal K distribution
   a. Acidemia
   b. Insulin deficiency, Hypertonicity (hyperglycemia, mannitol, etc.)
   d. Digitalis (decreases Na,K-ATPase activity)
   e. Succinylcholine
   f. Tissue damage
   g. Periodic paralysis
   h. Exercise
   i. ß-blockers (decrease catecholamine induced K uptake by muscle)
   j. Aldosterone deficiency

* NSAID = nonsteroidal anti-inflammatory drugs; † ACEI = angiotensin converting enzyme inhibitors

Bicarbonate infusion is particularly beneficial in patients who present with severe metabolic acidosis.[26] While acidemia due to inorganic metabolic acidosis results in a pronounced rise in the serum potassium level (0.5-1.2 mmol/L for each 0.1 unit change in pH), respiratory acidosis (0.1-0.3 mmol/L rise in K per each 0.1 unit change of pH) and organic metabolic acidosis (e.g., lactic acid and ß-hydroxybutyric acid) cause a much smaller rise in serum potassium level.[27-30] Sodium bicarbonate can be administered as a 50 meq bolus over 5-10 minutes. The onset of action in correcting hyperkalemia is after 5-10 minutes and the effect lasts approximately 2 hours. The dose can be repeated within 10-15 minutes. The major potential side effects of this modality are volume overload and precipitation of tetany or seizure in patients with preexisting hypocalcemia, due to further depression of the serum ionized calcium level.

Insulin lowers serum potassium concentration by stimulating its uptake by the cells, particularly muscle. This effect is via stimulation of Na,K-ATPase activity by insulin and is independent of glucose transport.[31] Glucose is generally co-administered with insulin to prevent hypoglycemia. Ten units of regular insulin is usually adm
insulin/5 grams of glucose) over a one hour period. Insulin alone would be sufficient if the patient is already hyperglycemic due to diabetes. It is emphasized that patients with hyperkalemia and diabetic ketoacidosis are generally total body potassium-depleted and are prone to develop hypokalemia as potassium moves back into the cells. The onset of action of insulin in reducing plasma potassium concentration is after 30-60 minutes and lasts for 4-6 hours. One may expect approximately 1 mmol/L decrease in plasma potassium concentration within 1-2 hours. Insulin/glucose infusion can be repeated as frequently as needed. The major side effects are hypo- or hyper- glycemia. Hyperglycemia should be avoided as it can exacerbate the hyperkalemia by increasing plasma tonicity and a solute drag effect.

ß 2-adrenergic agonists drive plasma potassium into the cells by stimulating Na,K-ATPase activity. Epinephrine released during the stress response as well as that exogenously administered during management of cardiac arrest drives potassium into the cells and hence decreases serum potassium concentration. Albuterol (salbutamol), a selective ß2-agonist, has also been shown to lower serum potassium concentration by 1-1.5 mmol/L within 30 minutes of its administration.[31-33] Albuterol can be administered either by intravenous (0.5 mg in 100 mg of a dextrose solution infused over 15 minutes) or nebulization (10-20 mg in 5 ml normal saline inhaled over 15 minutes) routes. The onset of action of albuterol is within 5-10 minutes, peak response is seen after 30-60 minutes of intravenous and 90 minutes of nebulization treatment, and the potassium lowering effect is sustained for 3-6 hours. The intravenous route should be preferred in patients requiring a more rapid decline in plasma potassium while nebulization treatment should be used in those with history of coronary artery disease.

Since the effects of calcium, hypertonic saline, insulin, sodium bicarbonate, and ß2-agonists are transient and potassium that is driven into the cells will re-enter plasma after a few to several hours, the above-

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Table II. Treatment of hyperkalemia

	Dose	Onset (min.)	Duration (hours)	Mechanism	Side effects
Calcium gluconate (10%)	10-20 ml, iv.	1-3	1-2	membrane stabilization	digoxin toxicity
Hypertonic saline (3%)*	50-100 meq, iv.	5-10	2	membrane stabilization	volume overload, hypernatremia
Insulin/ Glucose	1 unit regular insulin / 5 gram glucose, iv.	30-60	4-5	shift K into the cells	hyperglycemia, hypoglycemia
$NaHCO_3$	50-100 meq, iv.	5-10	2	shift K into the cells	volume overload, hypernatremia, tetany or seizure if hypocalcemic
ß2-Agonist (albuterol)	0.5 mg iv. or 10-20 mg nebulization over 15 min.	5-10	3-6	shift k into the cells	tachycardia, chest pain
Hemodialysis	3-4 hours	few	till dialysis ends	K removal	
Kayexalate	25-50 gram oral or per-rectum	30-60	4-6	K removal	volume overload, constipation
Diuretics		with start of diuresis	till diuresis ends	K removal	volume contraction

iv = intravenous

* hypertonic saline should be considered only in the hyponatremic hyperkalemic patients

mentioned modalities should only serve as temporizing measures until more definitive therapy can be instituted to decrease the total body potassium content. Nevertheless, these temporizing measures can usually reduce plasma potassium enough to alleviate the immediate life-threatening cardiotoxicity that the patient is facing.

Although in certain clinical situations, one temporizing modality may seem more appropriate than the other (e.g., sodium bicarbonate infusion in severe acidemia, insulin in diabetic hyperglycemia or ketoacidosis, calcium infusion with wide QRS complex or life-threatening arrhythmias, etc.), during life-threatening situations, all of the above modalities should be employed promptly and simultaneously. In patients with end stage renal failure, life threatening hyperkalemia can be temporarily managed (till definitive therapy which is removal of potassium by hemodialysis is instituted) with insulin / glucose and ß2-adrenergic agonists, however, the efficiency of sodium bicarbonate infusion in lowering plasma potassium in this clinical setting has been inconsistent.[31-35] In the case of hyperkalemic cardiac arrest it seems logical to continue with the resuscitation and the above-mentioned modalities until serum potassium is less than 6.0 mmol/L and the patient has normal serum sodium and calcium levels, before a decision is made to withdraw support.

After the patient's cardiovascular status stabilizes, the following infusion may be started to maintain serum potassium at a safe level while measures to remove excess potassium from the body are being implicated. A mixture of 1000 ml of 10% dextrose in normal saline or water (based on presence or absence of hyponatremia) plus 100 meq of sodium bicarbonate and 20 units of regular insulin, administered initially at the rapid rate of 500 ml in the first 30 minutes with the remainder given over the next 2-3 hours.

To remove excess potassium from the body one can administer diuretics (loop or thiazide-type), cation-exchange resin (sodium polystyrene sulfonate), or use dialysis. Among these measures, *dialysis* offers the most immediate means of removing plasma potassium. Dialysis is particularly important in patients with acute

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renal failure who are hypercatabolic, in whom cell breakdown can result in the release of large quantities of potassium into the extracellular fluid. Hemodialysis is preferred because the rate of potassium removal is many times faster than with peritoneal dialysis.[36] Adding no potassium to the dialysis solution, one can remove approximately 40 meq of potassium during the first hour of hemodialysis. The serum potassium concentration will begin to fall within minutes of starting dialysis, and the hypokalemic effect will last as long as dialysis is continued. The major disadvantage of this modality is the time required to prepare the equipment and to obtain a vascular access.

Another effective modality to remove potassium from the body is administration of a cation-exchange resin. Sodium polystyrene sulfonate (*Kayexalate*) is the major cation-exchange resin used. This resin absorbs potassium and to some degree calcium and magnesium in exchange for Na. Each gram of resin may bind as much as 1 meq of potassium and release approximately 2 meq of sodium. It can be administered either orally or via retention enema. In either case it should be mixed with sorbitol to prevent constipation. It usually takes 1-2 hours to see the onset of Kayexalate action. On average, 50 grams of Kayexalate will lower the serum potassium concentration by 0.5-1 mmol/L over 4-6 hours.

Diuretics (loop diuretics [furosemide, bumetanide, ethacrynic acid], or thiazide diuretics) can be used in patients with preserved renal function and are particularly beneficial in the setting of volume overload. Diuretics increase distal nephron delivery of salt which makes more sodium available for exchange with potassium and hence increase potassium excretion.

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Notice of Correction

The following typographical errors occurred in the paper by Roodpeyma S, Shahmohammadi A: "Supravalvar aortic stenosis in children: report of 13 cases" Med J Islam Repub Iran 6(4): 241-4, 1993.

- in line 9 of the abstract membranous type should be "15%" instead of "5%"
- in line 4 of paragraph 4 of Introduction the words "mandible" and "overhanging" should be separated by the word "and".
- in line 2 of paragraph 5, "100" should be "1000".
- in line 1 of Material and Methods, "21 years between 1975" should be "15 years between 1975 and 1990".
- in line 8 of results" the patients" should read" three patients".

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