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Case Report

Hyperkalemic circulatory shock and cardiac arrest altered by therapeutic management: A case report

Rohit Mody[1]*, Debabrata Dash[2] and Bhavya Mody[3]

[1]Department of Cardiology, Max Super Speciality Hospital, Bathinda, Punjab, India.
[2]Department of Cardiology, Aster Hospital, Dubai, Al Quasis, UAE.
[3]Department of Medicine, Kasturba Medical College, Manipal, Karnataka, India.

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ABSTRACT

Hyperkalemia is one of the few potentially lethal electrolyte disturbances. Severe hyperkalemia (Serum potassium concentration > 6.5 mmol/L) occurs most commonly from renal failure or the release of potassium from cells and can cause circulatory shock, cardiac arrhythmias or cardiac arrest. Current BLS (Basic Life Support) and ACLS (Advanced Cardiovascular Life Support) protocol should be used to manage cardiac arrest associated with hyperkalemia. But early consideration should be given to using the selective method of therapeutic management in addition to standard ACLS protocols that can be provided rapidly, effectively in patients with cardiovascular instability. We describe here a case of chronic kidney disease and congestive heart failure who developed circulatory shock and eventually cardiac arrest due to hyperkalemia managed with Calcium Gluconate, Sodium Bicarbonate and Insulin along with standard advanced cardiovascular life support protocol.

Keywords: Potassium, hyperkalemia, acidosis, calcium, insulin, cardiac arrest.

*Corresponding author. Email: drmody_2k@yahoo.com. Tel: +91-9888925988.

INTRODUCTION

Hyperkalemia is the most common electrolyte disorder associated with cardiopulmonary arrest (McFadden, 2003). The first indication of hyperkalemia may be Electrocardiogram (ECG) changes. Almost all patients will have ECG abnormalities at serum potassium concentration higher than 6.5mmol/L (McFadden and Warren, 1997). There are several potential causes of hyperkalemia including renal failure, drugs, tissue breakdown, tumor lysis syndrome, hemolysis, metabolic acidosis, endocrine disorders like hypoaldosteronism and diet (Division of Data Services, 2001).

During hyperkalemic cardiac arrest, although there are no modifications to basic life support (BLS), it is controversial whether modifications to advanced cardiovascular life support (ACLS) with suitable therapeutic agents like:

- Calcium Chloride I/V bolus to protect heart first.
- Remove potassium from the body – Dialysis.
- Agents that shift potassium into cells:
  - Insulin + Dextrose
  - Sodium Bicarbonate

We hereby report a case of hyperkalemia in which the patient clinically was in circulatory shock and finally arrested. In this patient, we manage arrest with the usual ACLS protocol and combined this protocol with measures to reduce serum potassium levels immediately.

We describe here a case of chronic kidney disease and congestive heart failure who developed circulatory shock and eventually cardiac arrest due to hyperkalemia managed with Calcium Gluconate, Sodium Bicarbonate and Insulin along with standard advanced cardiovascular life support protocol.

The incidence of hyperkalemia in hospital patients ranges from 1.1% and 10% (Acker et al., 1998; Paice et al., 1983; Moore and Bailey, 1989; Einhorn et al., 2009; Conway et al., 2015). The incidence in the community varies dependent on the case-mix of the population studied. Studies in the general population report an

incidence of hyperkalemia (K+ >5.5 mmol/L) ranging from 2.3 to 7.2% in patients with an eGFR > 60 ml/min (Chang et al., 2016; Horne et al., 2019) and 2.9 to 40% in patients with an eGFR < 30 ml/min (Korgaonkar et al., 2010; Sarafidis et al., 2012; Turgutalp et al., 2016).

In-hospital mortality is significantly higher in patients with hyperkalemia (18.1%) compared to those with hypokalemia (5.0%) or normokalaemia (3.9%) (Conway et al., 2015). A U-shaped association between serum potassium and mortality has been shown in patients with ischaemic heart disease (Goyal et al., 2012), CKD (Korgaonkar et al., 2010; Hayes et al., 2012; Collins et al., 2017), and patients receiving long-term hemodialysis (HD) (Kovesdy et al., 2007). Patients with severe hyperkalemia (K+ > 6.5 mmol/L) are most at risk and in one report, the hospital mortality was 30.7% (An et al., 2012).

The treatment of hyperkalemia is likely to evolve in the coming years with the availability of novel drugs and the development of new strategies to improve safety. Clinical decisions on when to treat and how aggressively to treat require a patient-centred approach guided by the clinical setting and rate of change in serum K+ level. Patients with moderate levels of hyperkalemia pose the greatest dilemma, especially when acuity is low, but warrant intervention to avoid deterioration. Severe hyperkalemia risks arrhythmias and cardiac arrest, therefore prompt recognition and intervention are required.

CASE PRESENTATION

Clinical presentation

A 65-year-old female arrived in the emergency department with the complaint of shortness of breath, nausea vomiting, and restlessness and generalized weakness. On physical examination, there was mild tachypnea; the patient was hypotensive with mild pitting edema - respiratory rate- 30/min, Blood pressure – [90/70] mmHg, Pulse – 45 beats/min. On auscultation, bilateral inspiratory crackles were heard at lung bases. The patient had not passed urine for 4 hours and catheterization yielded no output of urine. The patient was a known case of NIDDM (Non-Insulin Dependent Diabetes Mellitus) which was poorly controlled – HbA1C 8%. She was a known case of hypertension and Coronary artery disease (CAD). She underwent Percutaneous Coronary Intervention PCI), had a left ventricular ejection fraction (LVEF) of 40%. She was a known case of chronic kidney disease (CKD) with serum creatinine – 4.2 mg/dL.

Management

ECG revealed ST depression, Broad QRS waves, Sinus Bradycardia and Peaked T waves suggesting hyperkalemia (Figure 1). Arterial blood gas (ABG) revealed hyperkalemia and metabolic acidosis,(Figure 2). ECHO showed Regional Wall Motion Abnormalities (RWMA) in poster lateral wall with EF – 40%. An intravenous (IV) line was inserted; Calcium Gluconate 10 ml IV bolus was (given) in 5 minutes. Inotropic support was started with norepinephrine infusion and Noninvasive Ventilation (NIV) support was given. IV sodium bicarbonate 50 mmol/l bolus was given and the patient was shifted to Cath lab as there was substantial bradycardia with a heart rate of 45 beats per minute.

In the cath lab, an intra-arterial catheter was inserted which showed invasive blood pressure of 80/60 mm Hg. A temporary pacemaker was inserted. Inotropes were increased and IV furosemide was started at 2 ml/hour. The patient was shifted to the coronary care unit (CCU) and non-invasive ventilation [NIV] was initiated.

In CCU, Short-acting Insulin was given at 7 Units IV stat and IV Calcium Gluconate 10 ml IV bolus. But despite the above measures, the patient experienced sudden cardiac arrest and started gasping. ECG showed sine waves (Figure 3) and intra-arterial pressures dropped to 20/10 mmHg. Her sensorium was altered. Cardiac massage was started in accordance with BLS protocol. IV epinephrine 1 mg was repeated every 5 mins. Respiratory support with Ambu bag was initiated. Then suddenly ECG monitor revealed asystole Cardiac massage was continued and respiratory support was given with an Ambu bag. Calcium chloride 10 ml was repeated twice at 10 min intervals. IV sodium bicarbonate 50 mg was repeated every 10 min. Continuous infusion of sodium bicarbonate at 20 ml/h was initiated. Short-acting IV Insulin 5 units IV stat and infusion at 5 units/hour was started. Rhythm returned with the patient on pacing mode (Figure 4). There was an improvement in ABG parameters. BP increased to 160/90 mmHg. . Inotropes were tapered off; IV furosemide was started. Nebulization was done with salbutamol.

OUTCOME AND FOLLOW-UP

The patient regained consciousness, spontaneous respiration was attained, and BP returned to normal. She improved hemodynamically with slight tachypnea and bilateral crackles in lower lung fields on auscultation. Chest X-rays (CXR) showed bilateral pulmonary congestion. As the patient still had hypotension, IV normal saline was started at 200 ml/h along with IV furosemide [infusion]. Sodium Bicarbonate and Insulin were continued, Urine Output was 100 ml/h. On the second day, the patient was hemodynamically stable. The patient was still in fluid overload as shown by fine crackles on auscultation. The usual treatment for congestive cardiac failure (CHF) with diuretics and B-blockers was started. The dialysis was initiated, and 1.5-

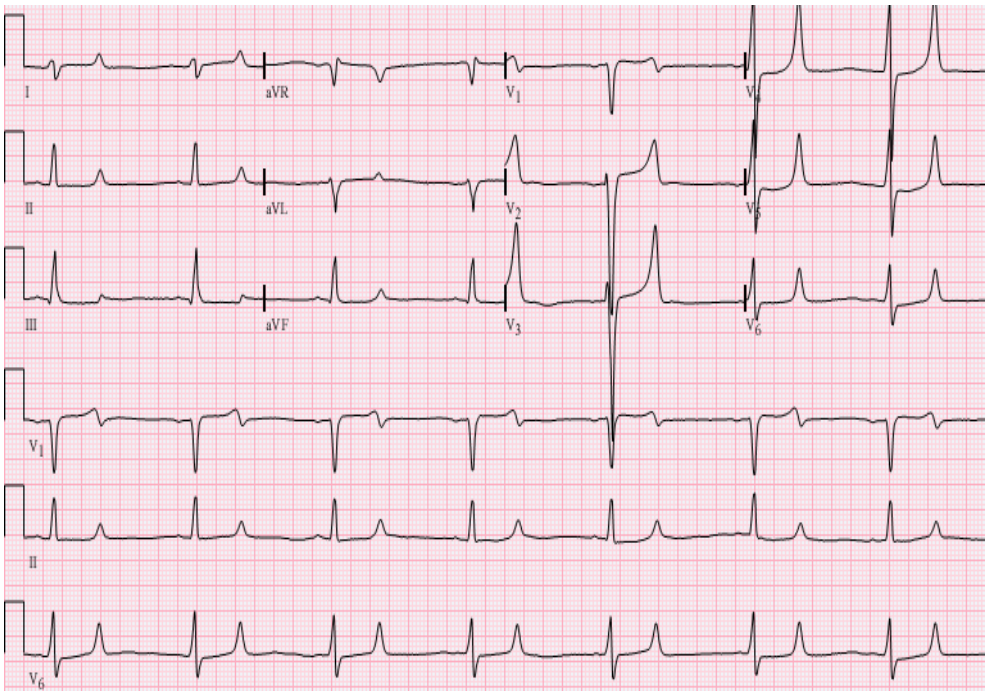

Figure 1. ECG of the patient on arrival to the ER showing ST depression, Broad QRS waves, Sinus Bradycardia and Peaked T waves.

Blood Gas Values			
↓ pH	7.018		[7.350 - 7.450]
pCO_2	35.7	mmHg	[35.0 - 45.0]
pO_2	97.9	mmHg	[83.0 - 108]
Oximetry Values			
↓ ctHb	5.8	g/dL	[12.0 - 16.0]
sO_2	95.8	%	[95.0 - 99.0]
FO_2Hb_e	95.0	%	[-]
$FHHb_e$	4.2	%	[-]
Electrolyte Values			
↑ cK^+	7.7	mmol/L	[3.5 - 4.5]
↓ cNa^+	134	mmol/L	[135 - 145]
cCa^{2+}	1.16	mmol/L	[1.15 - 1.29]
↑ cCl^-	112	mmol/L	[98 - 106]
Metabolite Values			
↑ $cGlu_c^n$	253	mg/dL	[70 - 105]
↑ cLac	6.5	mmol/L	[0.5 - 1.5]
Temperature Corrected Values			
pH(T)	7.018		
$pCO_2(T)$	35.7	mmHg	
$pO_2(T)$	97.9	mmHg	
Oxygen Status			
ctO_2e	8.0	Vol%	
$p50_e$	33.50	mmHg	
Acid Base Status			
$cBase(Ecf)_c$	-20.0	mmol/L	
$cHCO_3^-(P,st)_c$	9.0	mmol/L	

Notes

↑	Value(s) above reference range
↓	Value(s) below reference range
c	Calculated value(s)
e	Estimated value(s)

Figure 2. ABG on arrival.

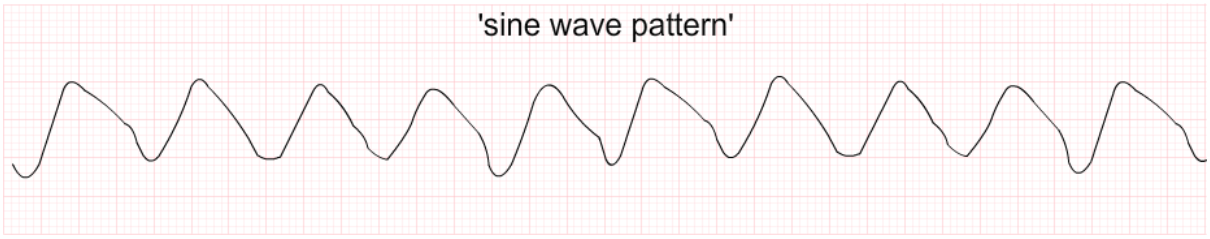

Figure 3. ECG showing sine waves.

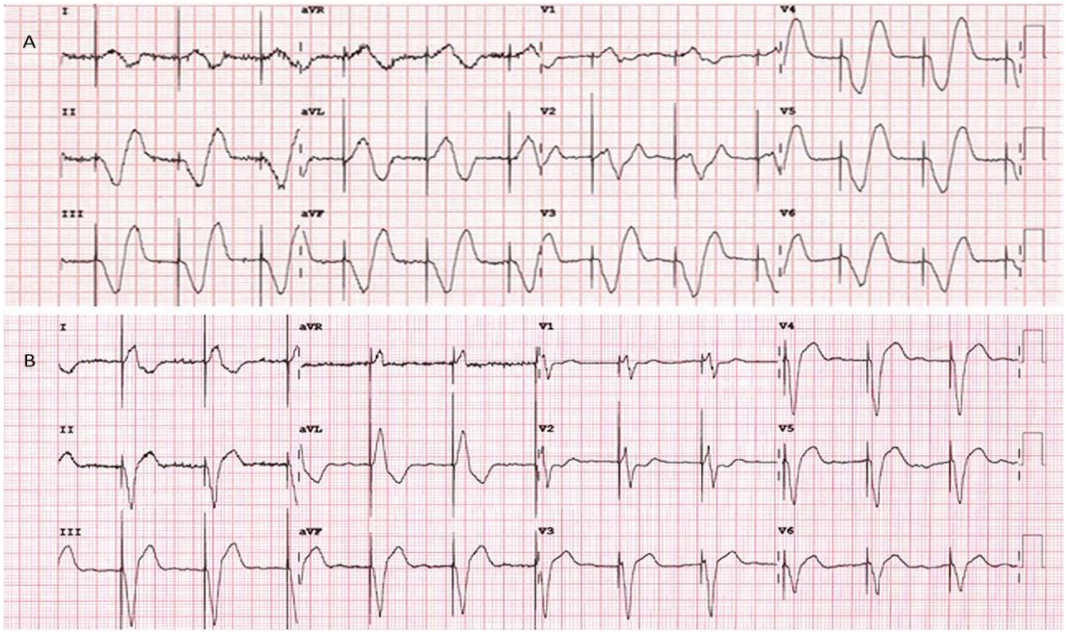

Figure 4. ECG of the patient after the revival of cardiac arrest on temporary pacing but still showing Tall T waves.

liter fluid was removed in view of raised blood urea and creatinine. Pro-BNP was measured, and its values were as high as 7000 pg/ml. Post dialysis, the patient became hemodynamically stable and crackles were decreased with the absence of pedal edema. From the 2nd to 5th day, hemodialysis was advised every alternative day. Urea and creatinine were reduced after each dialysis. Serum potassium remained between 3.5 to 4.5 mmol/l. Management with diuretics, B-blockers, anti-platelet agents, nitrates and calcium channel blockers were continued (Figure 5). On the 5th day, ECG was significantly better (Figure 6) and the patient was discharged on this management and was advised maintenance hemodialysis by the nephrologist and follow up in cardiology OPD. She was followed up after 7 days. On examination, there was an overall clinical improvement with normalization of ECG changes. Her serum potassium was 4 mmol/L. The echo still showed LVEF of 35%. Urine output was good with urea – 120 mg/dl and creatinine – 4.5 mg/dl. She was advised to dialysis twice a week. Presently patient is on regular maintenance hemodialysis on an outpatient basis. A timeline depicting the overview of all the clinical events is given in Table 1.

DISCUSSION

Cardiac arrest in hyperkalemia occurs due to cardioplegia. Elevated potassium alters myocyte resting potential from –85 mV to that approaching –40 mV, inactivating fast sodium channels (Mount and Zandi-Nejad, 2011; Weisberg, 1993; De Oliveira et al., 2014; Robert et al., 2016). This blocks conduction of myocardial action potential inducing arrest. Patients with severe hyperkalemia may present with hemodynamic instability. Hyperkalemia may be more common than typically perceived especially in patients with circulatory shock, even for those with a history of chronic renal disease (Al Aseri, 2019). Severe hyperkalemia can lead to circulatory

Blood Gas Values			
↑ pH	7.469		[7.350 - 7.450]
?↑ pCO_2	53.2	mmHg	[35.0 - 45.0]
? pO_2	.....	mmHg	[83.0 - 108]
Oximetry Values			
↓ ctHb	5.0	g/dL	[12.0 - 16.0]
sO_2	98.4	%	[95.0 - 99.0]
FO_2Hb_e	97.6	%	[-]
$FHHb_e$	1.6	%	[-]
Electrolyte Values			
cK^+	4.3	mmol/L	[3.5 - 4.5]
↑ cNa^+	154	mmol/L	[135 - 145]
↓ cCa^{2+}	0.99	mmol/L	[1.15 - 1.29]
↑ cCl^-	106	mmol/L	[98 - 106]
Metabolite Values			
cGlu	89	mg/dL	[70 - 105]
↑ cLac	4.6	mmol/L	[0.5 - 1.5]
Temperature Corrected Values			
pH(T)	7.469		
$pCO_2(T)$	53.2	mmHg	
Acid Base Status			
? $cBase(Ecf)_c$	13.5	mmol/L	
? $cHCO_3^-(P,st)_c$	37.2	mmol/L	

Notes

Value(s) above reference range
Value(s) below reference range
Calculated value(s)
Estimated value(s)

pCO_2 0476 Measurement unstable
$pCO_2(T)$ 0476 Measurement unstable
pO_2 0476 Measurement unstable

Figure 5. ABG on Day 2.

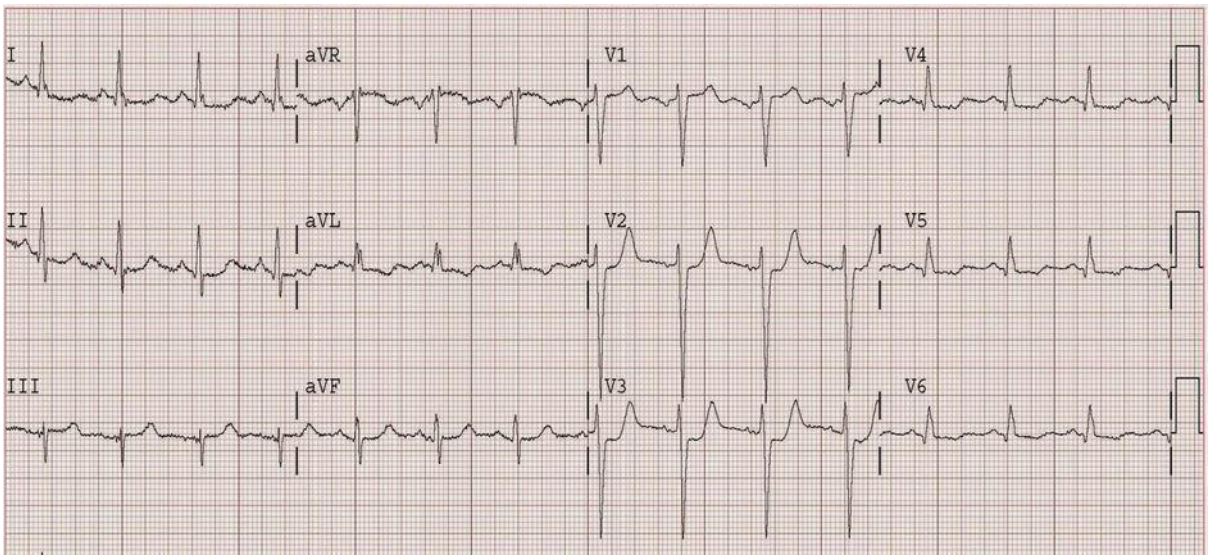

Figure 6. ECG on 5th Day.

shock and should be considered in the differential diagnosis in the workup of shock in ED (Fleet et al., 2012). The serum potassium does not always predict ECG changes or the degree of cardiotoxicity and

Table 1. Timeline.

Time	Place	Physical examination	Investigations and results	Interventions done
DAY 1				
5 PM	Arrival in the ER	●Vitals: BP – 90/60 RR – 30/min Pulse – 50/min	●ECG revealed ST depression, Broad QRS m and Peaked T waves. ●Serum Creatinine – 4.2mg/dL ●ABG - Serum K+ 7.7 mmol/l, Ca2+ - 1.16 mmol/l, Na+ - 134 mmol/l, Glucose – 250 mg/dl, Lactate 6.6 mmol/l, Bicarbonate – 9 mmol/l, pH 7.018, SO2 – 90% ●ECHO showed RWMA in posterolateral wall with EF– 40%	●IV line inserted ●Calcium Gluconate 10 ml IV bolus in 5 minutes. ●Ionotropic support started with norepinephrine infusion ●NIV support
5.10 PM	ER	●Vitals unchanged		●Same measure continued ●Ionotropic support increased ●IV sodium bicarbonate 50 mmol/l bolus given. ●Patient shifted to Cath lab
5.20 PM	Cath Lab	●Intra-arterial catheter inserted which showed invasive blood pressure – 80/60 mmHg		●Temporary pacemaker inserted.
5.40 PM	Cath Lab			●Inotropes increased ●Furosemide started at 2ml/hour ●The patient shifted to CCU for NIV support
6 PM	CCU	●Blood glucose – 300 mg/dl		●Short-acting Insulin is given – 7 Units IV stat ●IV calcium Gluconate – 10 ml IV bolus. ●Inotropes increased
6.15 PM	CCU	●Patient had a sudden cardiac arrest ●Patient was gasping.	●ECG shoed Sine-waves ●Intra-arterial pressures dropped – 20/10	●Cardiac massage was given under BLS protocol. ●IV epinephrine 1 mg repeated after every 5 mins. ●Respiratory support with Ambu bag
6.20 PM	CCU		●ECG monitor showed a straight line with no pressures.	●Cardiac massage continued. ●Calcium Chloride 10 ml repeated twice at 10 mins intervals. ●IV sodium bicarbonate 50 mg repeated every 10 minutes ●Continuous infusion of sodium bicarb at 20 ml/h. ●Short acting IV Insulin 5 units IV stat and Infusion at 5 units/Hour

Table 1. Continues.

7 PM	CCU	●Rhythm returned with patient on pacing mode.	●Repeat ABG showed: Serum K+ 6.5 mmol/l, Ca2+ - 1.15 mmol/l, Na+ - 132 mmol/l, Glucose – 177 mg/dl, Lactate 2.4 mmol/l, Bicarbonate – 20 mmol/l, pH 7.2, SO2 – 97%	
7.15 PM	CCU	BP – 160/90		●Inotropes tapered off ●Furosemide started ●Nebulization started with salbutamol.
7.30 PM	CCU	●Pt regained consciousness; Spontaneous respiration attained ●BP – 140/90	●Repeat ABG was sent.	IV NS started 200 ml/h NaHco3 continued Insulin Continued.
8.30 PM		●Patient stable ●Slight tachypnea with bilateral crackles in lower lung fields.	●CXR showed b/l pulmonary congestion ●Pro-BNP – 7000 pg/ml	●Patient continued on IV Furosemide ●Urine Output - 100 ml/h
DAY 2				
8 AM			●Serum Creatinine – 4.5mg/dL ●Serum Urea – 180 mg/dl	●Dialysis initiated – 1.5-liter fluid removed
2 PM		Patient stable post-dialysis	EF – 40%	●Patient managed medically
DAY 3				
10 AM				●Dialysis is initiated every alternative day. ●For CHF – Diuretics, B-blockers
DAY 5				
10 AM	●Patient discharged			●Follow up – on dialysis

Abbreviations: ER – Emergency Room, BP – Blood Pressure, RR – Respiratory Rate, ABG- Arterial blood gases, RWMA – Regional wall motion abnormalities, EF- Ejection fraction, NIV – Non- Invasive Ventilation, CCU – Cardiac Care Unit.

profound hyperkalemia may occur without ECG manifestations. Severe bradycardia can be a manifestation of hyperkalemia. In our patient, the cause of hyperkalemia was multifactorial. There have been significant recent advances in our understanding of the mechanisms that maintain potassium homeostasis and the clinical consequences of hyperkalemia. Accordingly, the major risk factors for hyperkalemia are renal failure, diabetes mellitus, adrenal disease, and the use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers or potassium-sparing diuretics. Hyperkalemia is associated with an increased risk of death, and this is only in part explicable by hyperkalemia-induced cardiac arrhythmia. In addition to its well-established effects on cardiac excitability, hyperkalemia could also contribute to peripheral neuropathy and cause renal tubular acidosis. Hyperkalemia—or the fear of hyperkalemia—contributes to the underprescription of potentially beneficial medications, particularly in heart failure (Hunter and Bailey, 2019).

The patient had slightly symptomatic uremia and marked elevation in BUN and Creatinine indicating that his renal insufficiency had been slowly progressive over a long period.

The patient's hyperkalemia was secondary to chronic renal failure possibly complicated by hyperkalemic distal

renal tubular acidosis. The patient presented in the ED in a state of shock and from ECG changes hyperkalemia was suspected. Despite the emergency measures, the patient's condition regressed to cardiac arrest because of the patient being in severe renal insufficiency and even oliguria. The presence of CAD with LV dysfunction probably increased the susceptibility to hemodynamic instability and even cardiac arrest. The earliest changes were seen in our patient when K+ levels were 7.2 mmol/l. Also, the patient was hemodynamically unstable. In addition to the classical sequence of events outlined above and the table, virtually any arrhythmia or disturbance can be seen in hyperkalemic patients. Our patient went into cardiac arrest developed sinewave ECG changes, which were unresponsive to defibrillation attempts and pacing. The patient ultimately developed cardiac arrest and asystole. Our patient's ECG passed through a sequence of bradycardia to sine waves to asystole in a matter of several minutes. Most emergency physicians currently use variable guidelines for the management of hyperkalemic cardiac arrest. The 2010 AHA guidelines for Cardiopulmonary resuscitation (CPR) and emergency cardiovascular care in the management of severe cardiac arrest due to hyperkalemia recommends the use of ACLS modifications in form of IV calcium gluconate, sodium bicarbonate, glucose +/- insulin, diuresis, nebulized salbuterol, sodium polystyrene and dialysis. But in the state when cardiac arrest occurs secondary to hyperkalemia, it may be reasonable to administer adjuvant IV therapy as outlined above or cardiotoxicity in addition to standard ACLS (Class 2B, LOE C) (Vanden Hoek et al., 2010). The recent guidelines for and emergency cardiac care' (American Heart Association, 1992) address the entity of hyperkalemic arrest in the following fashion. "When hyperkalemia, hypocalcemia or calcium channel blocker toxicity is present, use of Calcium is probably helpful (Class 2A)." The guidelines also recommend the use of sodium bicarbonate in hyperkalemic cardiac arrest as follows "In certain circumstances such as in patients with preexisting metabolic acidosis, hyperkalemia or tricyclic antidepressant or phenobarbital overdose, sodium bicarbonate is beneficial." These guidelines provide no detailed systematic approach to the subset of patients who may present with cardiac arrest. But in our patient, during the management of hyperkalemic cardiac arrest, chest compression, airway management, atropine, epinephrine and cardiac pacing were administered with the targeted treatment of hyperkalemia. Specific treatment of hyperkalemia including measures to oppose the effect of hyperkalemia at the cell membrane level, to reduce its plasma concentration by increasing its influx into the cells, and finally, to remove it from the body pool. The first goal of membrane stabilization was achieved by the administration of calcium and sodium bicarbonate (hypertonic saline). The second goal – a shift of potassium from extracellular space to intracellular space – was achieved by administration of insulin, sodium bicarbonate or B2 adrenergic agonist. And the final goal – reduction of total body potassium, was achieved by hemodialysis.

Conclusion

Our case beautifully illustrates that hyperkalemia can not only present with hyperkalemic cardiac arrest but also can present with hemodynamic instability. The hemodynamic instability persisted even after reversion to sinus rhythm and when volume loss was repleted. In a patient coming to ED with hemodynamic instability, hyperkalemia should always be thought of as a differential diagnosis. In the event of cardiac arrest due to hyperkalemia, the BLS protocol remains the same but ACLS protocol the incorporation of IV calcium gluconate/ calcium chloride, IV sodium bicarbonate, IV insulin +/- glucose, and finally, the dialysis, can assist in [recovery]. Although in literature, the role of these therapeutic measures administered during resuscitation is not firmly established, our case is an example where these therapeutic measures were effective not only in aborting the arrest but also in restoring hemodynamic stability (Vanden Hoek et al., 2010; American Heart Association, 1992). However, the 2020 Hyperkalemia Renal Association guideline incorporates these measures in their guidelines (Alfonzo et al., 2020).

LEARNING POINTS/TAKE HOME MESSAGES

- Our case beautifully illustrates that hyperkalemia can not only present with hyperkalemic cardiac arrest but also can present with hemodynamic instability.
- In a patient coming to ER with hemodynamic instability, hyperkalemia should always be thought of as a differential diagnosis.
- In the event of Cardiac arrest due to hyperkalemia, the BLS protocol remains the same but ACLS protocol needs modification and the incorporation of IV calcium gluconate/ Calcium chloride, IV Sodium Bicarbonate, IV insulin +/- Glucose and finally, the dialysis, can assist in recovery.
- Although in literature, the role of these therapeutic measures is not firmly established in the case of resuscitation during hyperkalemic cardiac arrest, our case is an example where these therapeutic measures were effective not only in aborting the arrest but also in restoring hemodynamic stability.

Ethics approval and consent to participate

Ethical approval was not required since it is an accepted procedure.

Competing interests

The authors declare that they have no competing interests.

Consent for publication

Written consent has been obtained to publish the case report from the guardian. The consent copy is available with the authors and ready to be submitted if required.

Conflict of interest

No financial support has been taken from anyone.

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