Primary PCI in a Patient of Inferior ST-segment Elevation Myocardial Infarction in the Time of Severe Hyperkalemia: A Case Report.

Deepak Natarajan, Chiranjib Deb, Vijeta Maheshwari,Mafooza Rashid, Betsheba Dinaker, Nirmalya Mukherjee.

Department of Interventional Cardiology, Moolchand MedCity, New Delhi.

Abstract

This case report describes the management of a middle-aged hypertensive male patient who presented with acute inferior ST-segment elevation myocardial infarction accompanied with severe hyperkalemia. His coronary angiography revealed a thread like right coronary artery along its entire course and normal left coronary artery system with patent coronary stents in the left anterior descending and left circumflex arteries. Subsequent to correction of hyperkalemia with intravenous calcium gluconate and regular insulin the patient underwent primary percutaneous coronary intervention (PPCI)of the right coronary artery with the deployment of a sirolimus eluting stent. The patient received overnight an infusion of tirofiban at half the usual dose between the diagnostic coronary angiogram and PPCI.

Key Words : *Hyperkalemia, ST-segment elevation myocardial infarction, calcium gluconate, primary percutaneous coronary intervention, regular insulin.*

INTRODUCTION

Severe hyperkalemia defined as serum potassium level greater than 6.5 mEq/L albeit not uncommon is a life threatening condition that if not treated immediately can be catastrophic. We describe the management of a middle aged male patient who presented with acute inferior ST-segment elevation myocardial infarction accompanied by severe hyperkalemia.

CASE REPORT

A 63 year old hypertensive male was admitted for severe intermittent retrosternal chest pain for the previous 10 hours. He had underwent percutaneous coronary intervention (PCI) 4 years ago when 2 bare metal stents had been deployed in mid left anterior descending (LAD) and left circumflex (LCX) arteries. He had been on 75 mgm of aspirin, 5 mgm of ramipril, sustained release 25 mgm metoprolol and 10 mgm of atorvostatin. He had however resumed smoking more than 15 cigarettes a day. He had on this occasion consumed more than a couple of diclofenac tablets at his chest pain onset. On admission to the ER he appeared disoriented and agitated, had a heart rate of 35 to 40 per minute, systolic blood pressure of 70 mm Hg and had 86% oxygen saturation breathing room air. There were basal crepitations while his respiration was 28 to 30 per minute. His 12 lead ECG revealed a sinus rate of 38 per minute, significant ST segment elevation in L2,L3 and AVF with ST segment depression in the precordial leads. There was also suggestion of peaking of T waves from V2 to V5 and absence of P waves (figure 1). The 2D echocardiogram demonstrated a normal sized left venticle with akinesia of the inferior wall and an ejection fraction of 50%.

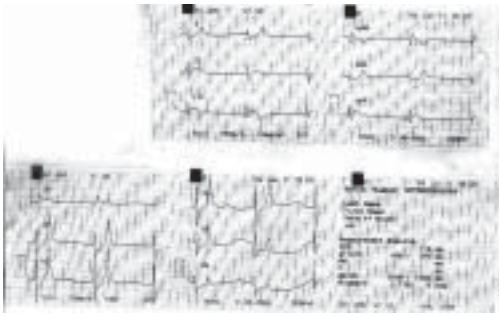


Figure 1: *Peaking of T waves from V2 to V5 and absence of P waves.*

A diagnosis of acute inferior ST elevation myocardial infarction (STEMI) with right ventricle infarction was made. Two litres of normal saline were rapidly infused intravenously and after an informed consent the patient was shifted to the catheterization laboratory for primary PCI. His coronary angiogram done from the right femoral route showed patent stents in the LAD and LCX arteries with no significant lesions in the left coronary system (figure 2). The right coronary artery (RCA) however was thread like in appearance . There was no change in the caliber of the RCA following 2 (50 mcg) boluses of intracoronary nitroglycerin (figure 3).

His laboratory investigations showed hemoglobin 12 gm %, total leukocytes 11,000 per cc, and platelets of 160,000 per cc. He had random sugar of 110 mgm % , blood urea 60 mgm %, serum creatinine 2.2 mgm %, serum potassium of 7.8 mEq/L, sodium 142 mEq/L and severe metabolic acidosos (pH 7.20, CO2 46 mmHg, O2 82 mmHg, HCO3-18 mEq/L, lactic acid 2.50 mEq/L). The CPK was 1715 units and CPK-MB was 102 units.

Correspondence: Dr. Deepak Natarajan, *Department of Interventional Cardiology, Moolchand MedCity, New Delhi.*
E-mail: *deepaknatarajan19@gmail.com*

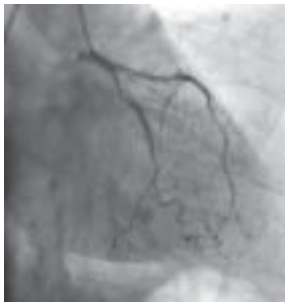


Figure 2: *Patent stents in the LAD and LCX arteries with no significant lesions in the left coronary system*

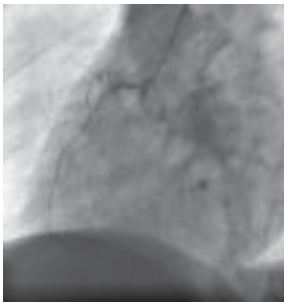


Figure 3: *The right coronary artery (RCA) however was thread like in appearance .*

The hyperkalemia was immediately treated with intravenous 10 ml of 10% calcium gluconate, 10 units of regular insulin in 100 ml of 50% dextrose intravenously and 50 mmol of intravenous sodium bicarbonate. This regimen was repeated twice more at 6 hour intervals.

In view of the severe hypekalemia it was decided to correct the electrolyte imbalance first and then proceed to PCI. The patient was given a bolus of 15 mcg/Kg of tirofiban followed by an infusion of (0.075 mcg/Kg/ minute) for 10 hours.

The next morning subsequent to positioning a temporary pacing lead in the right ventricle apex his RCA injection revealed a critical tight 90% proximal ulcerated stenosis (figure 4). His potassium had normalized to 4.1 mEq/L while his creatinine was 2.1 mgm%.The RCA was engaged by a 6 Fr JR guiding catheter and a 0.014 inch floppy wire was negotiated across the block. A 2.25 X 16 mm sirolimus stent was deployed at 16 atm subsequent to predilatation with a 1.5 X 10 mm balloon. Brisk antegrade TIMI 3 flow was achieved with no residual stenosis (Figures 5-6).

The patient was kept on ion exchange resins till discharge along with dual antiplatelet and statin thereapy.At discharge the potassium was 4.3 mEq/L, sodium 135 mEq/L, blood urea 61 mgm% and serum creatinine 1.8 mgm%.. The ECG on discharge exhibited sinus rhythm with fully resolved ST segment resolution and small Q waves in the inferior leads, without evidence of hyperkalemia. Segment resolution and small Q waves in the inferior leads, without evidence of hyperkalemia (Figure 7).

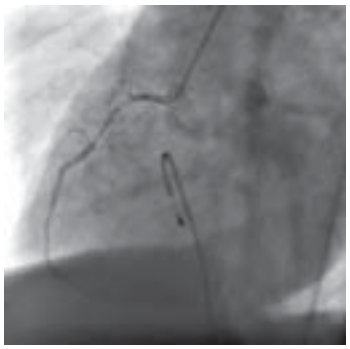
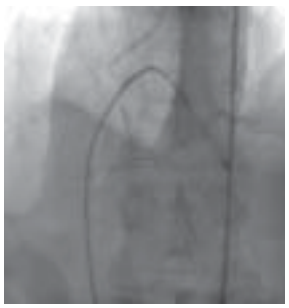
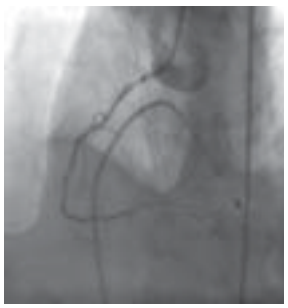


Figure 4: *RCA revealed a critical tight 90% proximal ulcerated stenosis.*





Figures 5-6: *Brisk antegrade TIMI 3 flow was achieved with no residual stenosis.*

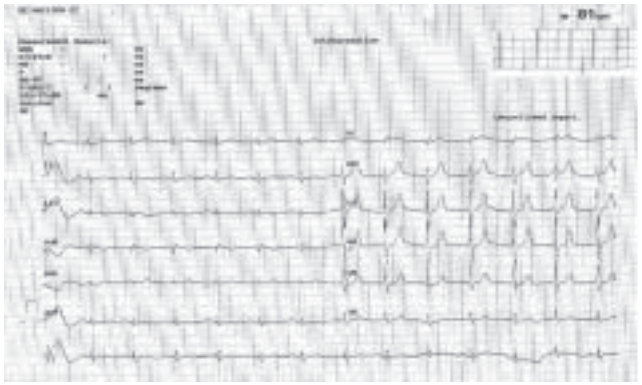


Figure 7: *ECG on discharge exhibited sinus rhythm with fully resolved ST segment resolution and small Q waves in the inferior leads, without evidence of hyperkalemia.*

DISCUSSION

This report describes the management of a patient with an acute inferior STEMI accompanied by severe hyperkalemia. Severe hyperkalemia (potassium more than 7.5 mEq/L) if left untreated carries a high mortality of more than 65%. It is imperative that hyperkalemia presenting with electrocadiographic changes or more than 6.5 mEq/L is rapidly corrected[1-4].

However in almost 50% of patients with serum potassium more than 6.5 mEq/L there may be no electrocardiographic changes[5].The other point to bear in mind for the clinician is that more than 60% patients presenting with severe hyperkalemia have an underlying renal impairment or have been on a single or a cocktail of potassium retaining medication.

The earliest ECG change observed with hyperkalemia are an absence of P wave, peaking and narrowing of T wave and shortening of the corrected QT interval. In cases of severe hyperkalemia other ECG manifestations seen are left and right bundle branch like widening of QRS complexes that can be distinguished from genuine bundle bundle branch block by the fact that in the latter the conduction delay is in the middle or the final stage whereas in hyperkalemia the widening is diffuse. In the final stages there is merging of the QRS complex and T wave producing a sinusoidal wave with QT interval prolongation . In some patients ST -segment elevation and depression can also

be seen mimicking an acute myocardial infarction. This is known as a " dialyzable injury current" [6-8].

Emergency treatment for severe hyperkalemia should be initiated immediately with intravenous infusion of calcium gluconate that begins to stabilizes the myocardial cell within 2-3 minutes but acts for only 30 to 60 minutes. Calcium chloride can also be used but as it is three times potent than calcium gluconate the dose has to be reduced. Calcium stabilizes the cell by maintaining the 15 mV gap between the resting membrane and threshold potentials . The normal resting membrane potential is made less negative from -90 mV to -80 mV. This is closer to the threshold potential of -75 mV and thereby makes it more vulnerable to lethal tachy-arrhythmias. Calcium may also speed up impulse formation in the sinoatrial and atrioventricular nodal cells reversing myocardial depression seen with hyperkalemia [9-10].

Serum potassium is next reduced with 10 units of regular insulin given intravenously.Insulin drives the excess potassium into the intracellular space by stimulating the Na-K ATPase pump in exchange with sodium in a 2:3 ratio. This effect is independent of glucose Therefore if a patient is already hyperglycemic extra dextrose need not be administered. Insulin begins to act in 30 to 60 minutes and reduces potassium by 0.5 to 1.0 mEq/L [11].

Nebulized salbutamol in high doses of 10 mgm has also been shown to reduce potassium levels by 0.62 to 0.98 mEq/L. Salbutomal has been seen to be safe and effective. It is therefore recommended to be employed as adjunctive treatment for patients with severe hyperkalemia. Sodium bicarbonate albeit capable of shifting potassium from extracellular to intracellular space by increasing blood pH is not recommended for routine usage because the effect is little and short lasting [12-13].

In the event of persisting hyperkalemia the quickest technique is to use hemodialysis. It is however rarely utilized because of its expense and invasive nature. The usual substitute is orally or rectally administered ion exchange resins that exchange potassium for sodium ions. Exchange resins decrease serum potassium by about 1 mEq/L in 24 hours.

This patient was on an angiotensin converting inhibitor, a long acting beta blocker, and upon that had consumed more than a couple diclofenac tablets. It is quite probable that these 3 drugs tilted the serum potassium to a dangerous level in this patient who also had impaired kidney function as suggested by his serum creatinine levels.

Percutaneous intervention was deferred in view of the severe hyperkalemia and the fact that the RCA initially despite intracoronary injections of nitroglycerine remained thread like. It may be hypothesized that in the setting of severe hyperkalemia the infarct related vessel goes into diffuse vaso-spasm.

Abciximab is the safest glycoprotein 2b/3a inhibitor(GPI) that can be administered in patients with significantly impaired glomerular filtration without any alteration in dosage. Tirofiban and eptifibatide can both be given as usual boluses but the infusion rate will need to be halved. Eptifibatide is contraindicated in patients on dialysis [14-16].

A tight almost ulcerated subtotal occlusion of the RCA was seen the following morning subsequent to 10 hours tirofiban (half dose) infusion and this was easily treated by percutaneous intervention deploying a sirolimus stent. The patient was subsequently managed on aspirin, clopidogrel, and atorvastatin.

CONCLUSION

This report underscores the fact that a patient presenting with acute STEMI accompanied with severe hyperkalemia requires immediate treatment of the raised potassium levels with calcium (because of its rapid stabilization of the myocardial cell) followed by insulin with or without glucose. Ion exchange resins are employed next to continue reducing serum potassium. Percutaneous coronary intervention is safe and effective subsequent to normalization of potassium serum levels. Adjunctive GPI's can be used with careful monitoring of dosage schedules.

REFERENCES

1. *Acker CG, Johnson JP, Palevsky PM,Greenberg A. Hyperkalemia in hospitalized patients: causes,adequacy of treatment, and results of an attempt to improve physician compliance with published therapy guidelines. Arch Intern Med 1998;158:917-24.*
2. *Greenberg A. Hyperkalemia: treatment options. Semin Nephrol 1998;18:46-57.*
3. *Weisberg LS. Management of severe hyperkalemia. Crit Care Med 2008; 36:3246-51.*
4. *Nyirenda MJ, Tang JI, Padfield PL, Seckl JR. Hyperkalemia. BMJ 2009; 339:1019-24.*
5. *Szerlip HM, Weiss J, Singer I.Profound hyperkalemia without electrocardiographic disturbances. Am J Kidney Dis 1986;7:461-5.*
6. *Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. Am J Emerg Med 2000;18:721-9.*
7. *Montague BT, Oullette JR, Buller GK. Retrospective review of the frequency of ECG changes in hyperkalemia. Clin J Am Soc nephrol 2008; 3: 324-30.*
8. *Zaikas A, Basagiannis C, Stiliadis I. Psuodoinfarction pattern in a patient with hyperkalemia, diabetic ketoacidosis and normal coronary vessels: A case report. J Med Case Report 2010;4:115.*
9. *Winkler AW, Hoff HE, Smith PK. Factors affecting the toxicity of potassium. Am J Physiol 1939;127:430-5.*
10. *Beeler GW, Reuter H. Membrane calcium current in ventricular myocardial fibres. J Physiol 1970; 207:191-209.*
11. *Blumberg A, Weidmann P, Shaw S, Gnadinger M. Effect of various therapeutic approaches on plasma potassium and major regulating factors in terminal renal failure. Am J Med 1998;85:507-12.*
12. *Blumberg A, Weidmann P, Ferrari P. Effect of prolonged bicarbonate administration on plasma potassium in terminal renal failure. Kidney Int 1992; 41:369-74.*
13. *Allon M. Copkney C. Albuterol and insulin for treatment of hyperkalemia in Hemodialysis patients. Kidney Int 1990;38:869-72.*
14. *Alexander KP, Chen AY, Roe MT,et al., for the CRUSADE Investigators. Excess dosing of antiplatelet and antithrombin agents in the treatment of non ST segment elevation acute coronary syndromes. JAMA 2005;294:3108-16.*
15. *Tsai TT, Maddox TM, Roe MT, et al., for the National Cardiovascular Data Registry. Contraindicated medication use in dialysis patients undergoing percutaneous coronary interventions. JAMA 2009; 302:2458-64.*
16. *Best PJ, Lennon R, Gersh BJ, et al. Safety of abciximab in patients with chronic renal insufficiency who are undergoing percutaneous coronary interventions. Am Heart J 2003;146: 345-50.*