Hypokalemia and Ventricular Arrhythmia In Acute Myocardial Infarction: A Retrospective Study (1992 - 1997)*

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Summary:

Hypokalemia is a cause of ventricular arrhythmia that can lead to ventricular tachycardia/ventricular fibrillation in patient suffering from Acute myocardial infarction. Study was conducted to find out any association between Hypokalemia and malignant arrhythmias in acute myocardial infarction. This retrospective study was conducted by screening of the charts of 2579 patients with newly diagnosed MI admitted during Jan. 1992 - 1997. Amongst 267 (10%) patients having malignant arrhythmia during the course of acute MI, 112 patients had hypokalemia. Whereas 164 were normokalemic. Cause of hypokalemia was diuretic therapy in 80 patients, diabetes in 18 patients and excessive vomiting in 10 patients. Incidence of VT and VF was different with various levels of S. Potassium. Amongst 600 patients suffering from hypokalemia, 21 patients out of 76 at S.K⁺ level of (2.0 - 2.5) m.eq/1, 48 patients out of 256 at level of (2.5 - 3.0) m.eq/1.43 out of 268 at level of (3.0 - 3.5) m.eq/1 had VT and VF in CCU. So the hypokalemia has significant effect in producing fatal arrhythmia in AMI, and Serum level is directly proportional to the incidence of VT/VF in this setup.

Disturbance of cardiac rhythm are common during acute MI, vast majority of post MI VI/VF occur with in the first 48 hrs. of MI, especially primary VF remains an important contributor to the risk of mortality during the 24 hours after MI, these early phases arrhythmias are probably largely a result of micro re-entry. Although other electrophysiologic mechanism, such as enhanced automaticity and triggered activity have been proposed in experimental model of MI. Convincing evidence of their role in human MI is not yet established. Important contributory factors include heightened adrenergic nervous system, tone, Hypokalemia. Hypomagnesemia, intracellular hypercalcemia, acidosis, free fatty acids production, lypolysis, free radical production from repurfusion from Ischaemic myocardium, the relative importance of these factors in the pathogenesis of arrhythmias during acute MI has not been established, nor has it been clearly shown that aggressive measures to correct these factors will reduce the frequency of arrhythmias and decrease the mortality from MI.

In an effort to find out an association of Hypokalemia for subsequent primary VF and VT during MI. We studied the charts of 2759 patients admitted in DMC CU LMC Hyderabad suffering from

acute Myocardial infarction and reviewed the finding of clinical, electrocardiographic and biochemistry data with particular attention to S. Potassium, S. Calcium and magnesium also in ECG localization of infarction, arrhythmias, Heart rate and QT interval.

Result:

In analysis of 2759 patients with MI 600 (22%) patients were found to have Hypokalemia and 276 (10%) patients were having VT in (110) and VF in (166). 112 patients had Hypokalemia of varying level out of the group of 276 patients who suffered arrhythmias and remaining 164 were Normokalemic (table-1) all the patients were in sinus rhythm or with occasional PVCS in base line ECG along with changes of MI, had normal calcium or Magnesium levels and did not receive any drug to potentiate arrhythmias. Cause of Hypokalemia was found due to:

- 1) Diuretic therapy in (15%) 80 patients.
- 2) Diabetes (3%) 18 patients.
- 3) Vomiting (2%) 10 patients.

In remaining 492 82% patients cause was unknown. (table-2).

The incidence of VT and VF was different with various levels of S. Potassium. Amongst 600 patients suffering from Hypokalemia 21 patients out of 76 with

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SK⁺ level of 2.0 - 2.5 m.eq/litre, 48 patients out of 256 with S.K⁺ (2.5 - 3.0) m.eq/l and 43 patients out of 258 patients with S.K⁺ (3.0 - 3.5) m.eq/l had VT/VF 258 patients with S.K⁺ (3.0 - 3.5) m.eq/l had VT/VF 258 patients with incidence of VT/VF as observed.

TABLE 1
Population studied

No. of patients	2759
Patients with Hypokalemia	600 (22%)
Patients with VT/VF	276 (10%)
Patients with Hypokalemia	112 (41%)
Patients with Normokalemia	164 (59%)

Discussion:

Clinical experience as well as observational data CCU population has identified Hypokalemia as an arrhythmogenic risk factor², VF in patient suffering macute myocardial infarction which may be result as chaemia and necrosis, as well as of altered machanic tone, Hypoxia, other electrolyte disturbances or result of concomitant therapies which take impact on the electrophysiological changes machanic with Ischaemia.

- Shortening of action potential.
- Depolarization of resting potential.
- 3 Slowing of rate of initial depolarization.
- Impaired conduction.

The molecular and cellular mechanism involve massium and calcium ions, the onset of myocardial massium is associated with larger increase in

TABLE 2

Cause of Hypokalemia

Diurenic therapy	90 (15%)
Distretic Dan magazidato	18 (3%)
Lase unknown	492 (82%)

partly because of the loss of inhibition of sensitive potassium channel, arrhythmias from either automaticity from single focus Elevated extra cellular potassium level extra depolarization which in turn inactivates and contributes to the reduced rate of action potential slowing conduction and entry arrhythmias. The current between and the potassium depolarized region can lead the potassium depolarized region can lead

So the arrhythmias may be enhanced by hypokalemias induced automaticity and facilitation of re-entry in patient suffering from MI with plasma K⁺ levels less than 3.2 m.eq/1 leads to ectopic complexes and malignant arrhythmias occur three times and the incidence generally vary with level of S. Potassium.

Epidemiological data suggest the incidence of VF and VT in acute MI may be decreasing in current era. Possibly due to aggressive attempts at infarct size reduction, correction of electrolytes deficit and greater use of Beta blocking agents. So the proper recognition of the cause of these malignant arrhythmias, e.g. hypokalemic and repletion of potassium will be helpful in decrease in hospital mortality.

TABLE 3

Level of Potassium and VT/VF

Serum K+ in mg%	Total No. of Patients with Hypokalemia	Patients with VT/VF	Percentage (%)
2.0 - 2.5	tinal ob 76 uction.	uptute Inte	27.6
2.5 - 3.0	256	48	18.7
3.0 - 3.5	268	43	16

Conclusion:

Hypokalemia in acute myocardial infarction should be taken seriously as it can be contributory factor in development of Fatal arrhythmias like VF/VT which can increase the mortality in this respect use of Diuretic carefully, management of excessive vomiting and replacement of potassium loss, as well as careful monitoring serum potassium during insulin therapy in DM should be performed to prevent fatal arrhythmias. Although other randomized clinical trail data, do not exist to confirm the benefits of repletion of potassium. It sounds in clinical practice to maintain S. Potassium level at greater than 4.0 m.eq/1 in patient with acute MI.

References:

- Paalo E. Pudda et al., QT interval and primary ventricular Fibrillation in acute MI. American Heart Journal Jan. 1981 volume 101 number 1.
- 2. Rayan et al. Management of acute MI and its complications JACC volume 28 No. 5 November 1996. 1328 428.
- Robert A et al. Comparative evaluation of new arrhythmic agents. American Heart Journal Dec. 1980 volume 100 No. 6 Part 2.
- Robert Roberts, MD, et al. Chapter 60 Pathophysiology of recognition and Rx of AMI. The Heart arteries and veins. Eighth edition.

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