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A CASE OF ACQUIRED LONG QT SYNDROME; CASE STUDY OF A 58YEAR OLD MALE WITH CORONARY ARTERY DISEASE

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ABSTRACT

58 years old Male known case of Inferior wall Myocardial infarction, Psychiatric illness on treatment presented with complaints of palpitations. ECG showed Long QTc with Torsade de pointes. On investigation patient had Hypokalemia. Correction of Hypokalemia with serum magnesium supplementation along with removal of the offending drug helped in recovery of the patient.

KEYWORDS: Acquired Long QT, Torsade de pointes, Hypokalemia.

1) INTRODUCTION

Long QT syndrome(LQTS) is a potentially life threatening cardiac arrhythmia characterized by delayed myocardial repolarization that produces QT prolongation and increased risk of Torsades des pointes- triggered syncope, seizures and sudden cardiac death.[1]

Acquired Long QT syndrome describes pathological excessive prolongation of the QT interval upon exposure to an environmental stressor with reversion back to normal following removal of the stressor in a structurally normal heart. The most common environmental stressor in acquired long QT syndrome is the drug therapy. Acquired long QT syndrome is an important issues for the clinician and a significant public health problem concerning the large number of drugs with adverse effects to potentially fatal outcome, the large number of patients exposed to these drugs and our inability to predict the risk for a given individual.[2]

2) CASE PRESENTATION

58 years old patient, Known case of Recent Inferior posterior wall Myocardial infarction, thrombolysed with Alteplase elsewhere and done elective PCI to LCX in our institute 2 months before presentation. Patient had Mild Left ventricular dysfunction and was started on Furosemide along with Aldactone. Patient was also a known case of Type 2 Diabetes mellitus on Oral hypoglycemic drugs; Psychiatric illness on treatment with Escitalopram and Olanzapine. Patient came after 2 months with history of palpitations. ECG taken showed Long QT with Torsades de pointes- QTc-640msec

(Figure 1) and his vitals were stable. Patient was started on IV magnesium 2 gram infusion for over 20 minutes. On investigating, his Serum Potassium was 2.6 and Serum Magnesium was 1.9 (normal limit- 1.7-2.2 mg/dl). He was started on Iv potassium – Infusion KCL at 10mmol/hour. Tablet Escitalopram, Olanzapine and Furosemide were withheld. His repeat ECG showed Ventricular bigeminy with Long QTc (Figure 2). He was continued on oral magnesium and potassium supplements. On discharge his potassium was 4.6 meq / L and QTc was 459 msec. He was discharged with Aripirazole as it had minimal effect on QTc After 2 week his ECG was taken which showed normal QTc (Figure 3).

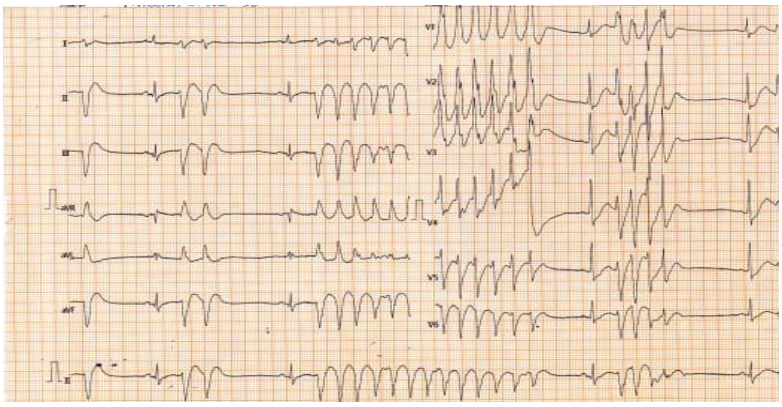

Figure 1: Long QTc(640msec) with Torsades de pointes.

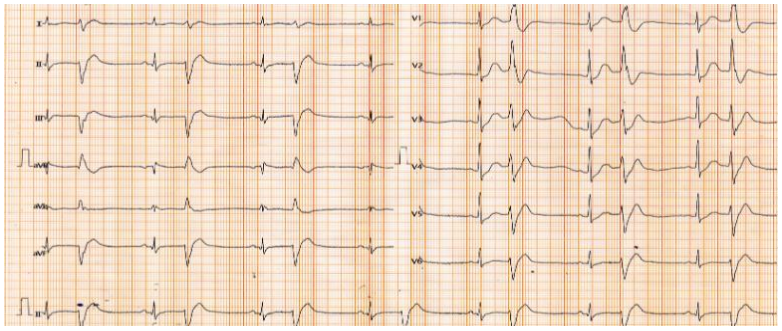

Figure 2.

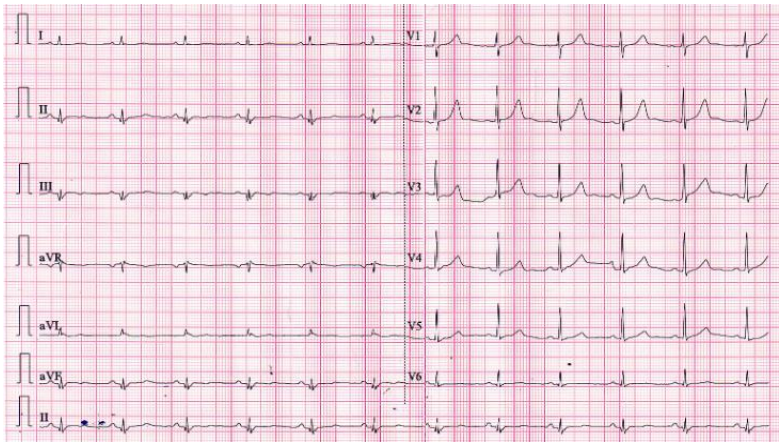

Figure 3.

3) DISCUSSION

The QT interval on the surface ECG is a representation of repolarization time in the ventricle. QT intervals in humans vary as a function of age, sex, heart rate, heart disease, and drugs and are generally less than 480 msec. "Acquired long QT syndrome" describes not one end of a physiologic spectrum, but rather pathologic QT interval prolongation, generally to greater than 550–600 msec, upon exposure to an environmental stressor and reversion back to normal following withdrawal of the stressor. When QT intervals are markedly prolonged in this fashion, the polymorphic ventricular tachycardia torsade de pointes becomes a real risk; torsade de pointes can be self-limited or can degenerate to fatal arrhythmias such as ventricular fibrillation. It is the potential for torsade de pointes and sudden death that has generated such attention to acquired long QT syndrome.[3][4]

The acquired causes of LongQT include drugs, electrolyte imbalance such as hypokalemia and hypomagnesemia, marked bradycardia, cocaine, organophosphorus compounds, subarachnoid hemorrhage, myocardial ischemia, etc. However drug Induced Long QT syndrome is the most predominant cause.[5][6] Common among them includes antihistamines (terfenadine and astemizole), gastrointestinal agents (cisapride), antipsychotics (sertindole), and urologic agents (terodiline).[5][6]

QT interval on the surface electrocardiogram represents the summation of action potentials in ventricular myocytes. QT prolongation entails action potential prolongation, that results from an increase in inward current (e.g., through sodium or calcium channels) or a decrease in outward current (e.g., through potassium channels). Myocardial repolarization is primarily mediated by efflux of potassium ions. Two subtypes of the delayed rectifier potassium current, IKr (rapid) and IKs (slow), are predominantly responsible for repolarization. The two currents have different activation, inactivation, and deactivation characteristics, different sensitivities to blocking drugs, different rate, and catecholamine sensitivity and were later found to be the result of expression of different genes.[7][8]

The hallmark mechanism of acquired LQTS and TdP is the blockade of IKr by specific drugs.[9] Ikr blockade causes a delay in phase 3 rapid repolarization of the action potential, which is reflected by QT prolongation. Prolonged repolarization can cause early afterdepolarizations (EADs) due to activation of inward depolarizing currents (L-type calcium channels or sodium-calcium exchange current), that appear as depolarizing oscillations in membrane voltage during phases 2 and 3 of the action potential. EADs that reach threshold voltage can cause a ventricular extrasystole preceded by a long QT interval on the surface ECG. On the other hand, dispersion of refractoriness due to heterogeneity in ventricular repolarization can create zones of unidirectional block. Repetitive extrasystoles, unidirectional block and zones of slow conduction can lead to reentry and TdP.[10]

In clinical practice, the adverse effects of QT prolonging drugs can be prevented by not exceeding the recommended dose, restricting the dose in patients with preexisting heart disease or other risk factors and avoiding concomitant administration of drugs that inhibit drug metabolism or excretion, prolong the QT, or produce hypokalaemia. The potassium level should be checked regularly when the patient takes potassium-wasting diuretics. If the patient develops TdP, the offending drug should be stopped and electrolyte abnormalities corrected. When prescribing a QT prolonging drug, patients should be warned of the problems associated with its use. A card listing risk factors (including other drugs that prolong QT), precautions and contraindications for co-prescriptions could be given to the patient[11]. The treatment of Long QT syndrome is Magnesium Intravenous even if the serum Magnesium values are within normal range.

4) CONCLUSION

Frequent Monitoring of Patients with ECG who are on QT prolonging drugs should be done. Additional precautions should be taken for such patients when they are on Drugs causing Electrolyte imbalance.

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