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Case series - Acquired long QT syndrome in hospitalizedpatients

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Abstract :
Background:
Common causes for acquired long QT syndrome in hospitalized patients are drugs and electrolyte abnormalities.

Methods :
In this case series we report 4 cases of acquired long QT syndrome in hospitalized patients admitted for different diseases. Clinical characteristics , medications, electrolyte disturbances were analysed in all these patients .

Results :
In one patients the cause of long QT syndrome was due to amiodarone in patients with abnormal heart function, in one patient cause is due to amiodarone and hypokelemia,hypomagnesemia with abnormal heart function and in one patient the cause of long QT was due to parenteral quinine and in one patient cause of long QT syndrome was due to hypokalemia and hypomagnesemia.
Only one patient developed Torsades de Pointes and treated with intravenous magnesium. Remaining patients did not develop Torsades de Pointes because of early withdrawal of drug and correction of hypokalemia and hypomagnesemia.

Conclusions :
This case series highlights the risk of long QT syndrome in hospitalized patients receiving drugs which prolong QTc and electrolyte disturbances with normal and abnormal heart function. Early recognition of this abnormality and timely stopping of drugs and correction of electrolyte disturbances will help in preventing Torsades de Pointes.

Key words: Long QT syndrome,drugs,Torsades de Pointes,hypokalemia,hypomagnesemia.

Introduction:
Long QT [LQT] syndrome is a condition in which delayed repolarization of the heart predisposes patients to develop an often life threatening polymorphic ventricular tachycardia, known as Torsades de Pointes[TPD]. Acquired LQTS is often thought to be associated with treatment with QT prolonging medications, which most often exerts its effect via blockade of the delayed rectifier potassium current, a major repolarisation current in the heart. However multiple clinical risk factors have also been implicated in the potentiation of this arrhythmia, including electrolyte disturbances, congestive heart failure, bradycardia , female sex, digitalis therapy, anorexia nervosa and alcohol abuse(1-4).
As awareness of QT interval prolonging medications and patient risk factors for acquired LQTS rises, the often multifactorial nature of acquired LQT will be acknowledged well. In all of the cases of acquired LQTS presented in this series, there were>2 prominent risk factors for the development of LQTS, which

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always included at least one known QT prolonging medication and at least one electrolyte disturbance. This highlights the multi factorial nature of acquired LQTS and the importance of recognising risk factors and preventing consequences with early action.

CASE 1:

A 41 year old female patient was admitted for sub acute intestine obstruction. She had undergone exploratory laparotomy for release of adhesions of small intestine .She did not have any co morbidities, Her pre operative investigations were normal. During post operative period she developed stress induced cardiomyopathy with severe LV dysfunction and hypotension. She received asopressors and diuretics.Her blood pressure improved ,she developed atrial fibrillation for which she received parenteral amiodarone followed by oral amiodarone. Her rhythm converted to sinus rhythm. Subsequently she developed Long QT syndrome with ventricular bigemini with R on T phenomenon (fig1) .She had short episode of Torsades de pointes. She was treated with intravenous magnesium. Her investigations showed hypokalemia hypomagnesemia, which were corrected and amiodarone was stopped. Subsequently her QT interval became normal and she did not develop any further episodes of ventricular arrhythmias.

CASE 2 :

A 55 year old female patient was admitted for acute decompensated heart failure. Her investigation showed atrial fibrillation with rapid ventricular rate. Her 2D ECHO showed left ventricle hypertrophy with advanced diastolic dysfunction. She was diagnosed as heart failure with preserved ejection fraction. She was given amiodarone for conversion to sinus rhythm. She was given anticoagulants for stroke prevention.She was treated with intra venous furosemide for relief of pulmonary congestion.

Her rhythm converted to sinus rhythm.Amiodarone was changed to oral form 200mg two times a day. After 5 days her ECG showed prolonged QTc of 0.60 sec (fig2) .Her electrolyte were normal.Amiodarone was stopped.She was monitored for ventricular arrhythmias. After stopping amiodarone her QT interval became normal.She did not develop any ventricular arrhythmias.

CASE :3

A 32 Year old male patient was admitted with history of fever and altered sensorium. On investigations he was found to have falciparum malaria for which he was started on parenteral quinine therapy .His baseline ECG was normal and electrolytes were normal.

On 3rd day of quinine therapy his ECG showed prolonged QTc (0.76 sec) and T wave alternans with changing T wall polarity (fig3). His quinine was stopped and was given parenteral artesuanate.He was monitored continuously for arrhythmias.His history did not reveal history of sudden cardiac death in family.

His serum magnesium & potassium levels were normal .After stopping quinine for 72 hours his ECG became normal .He did not develop ventricular arrhythmias during hospital stay. He improved symptomatically & discharged from hospital.

Case 4:

A 40 year old male patient was admitted with post covid mucormycosis. He was operated for Rhino orbital mucormycosis .Post operatively he developed renal failure for which he received hemodialysis . During recovery phase his ECG showed prolong QTc of 0.56 sec.(|fig 4) His 2D echo cardiography was normal.He did not give any family history of sudden cardiac death. He did not receive any drug causing QT prolongation.His serum electrolytes showed low K and Mg levels. He received intra venous potassium and magnesium for correction of hypo magnesemia and hypokalemia.He was monitored in ICU for ventricular arrhythmias.After correction of electrolyte abnormalities his QT c interval became normal.He did not develop ventricular arrhythmias .

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Table 1 - Patient characteristics of 4 cases

Discussion:

Long QT syndrome is caused by congenital and acquired causes.The congenital form is familial disorder that can be associated with sensorineural deafness(Jervell and Lange-Nelsen syndrome,autosomal recessive)or normal hearing(Romano-Ward syndrome,autosomaldominant).Congenital LQTS is caused by inherited channelopathies created by mutations in one or more genes.(5,6)

Patients with acquired form may also have underlying genetic predisposition.Various causes for acquired LQTS are drugs such as Quinidine, procainamide,N-acetyl procainide,amiodarone,sotalol,disopyramide,phenothiazines,tricyclic antidepressants,erythromycin,pentamidine,some antimalarials,cisapride and probucol.

Other causes are electrolyte abnormalities (hypokalemia and hypomagnesemia),effects of liquid protein diet and starvation,central nervous system lesions,significant bradyarrhythmias,cardiac ganglionitis,and mitral valve prolapse.

Risk factors predisposing to acquired LQT and Torsades de pointes

Medication:

Antidepressants
Antipsychotics
Antibiotics
Amiodarone
Electolyte imbalance:
Hypokalemia
Hypomagnesemia
Hypocalcemia
Clinical risk factors :
Older age
Female gender
Alcohol abuse
Sleep apnea
Complete AV block
Dilated cardiomyopathy
Bradycardia .

Patients with congenital LQTS can initially have syncope,at times misdiagnosed as epilepsy,as a result of Torsades de Pointes.Sudden death can occur in this group of patients.Sudden cardiac death occurs in approximately 10% of paediatric patients with out preceding symptoms. Patients with LQTS at increased risk for SCD include those with family members who died suddenly at younger age and those who experienced syncope.

Stress testing can prolong the QT interval and produce T wave alternans,the later indicative of electrical instability.ECG should be obtained for all family members of patients with congenital LQTS.Premature ventricular stimulation electrically does not generally induce ventricular arrhythmias in this syndrome,and EP studies are not usually helpful in making diagnosis.

For patients with LQTS but not syncope,complex ventricular arrhythmias,family history of sudden cardiac death no treatment or treatment with beta blockers is generally recommended.

In asymptomatic patients with complex ventricular arrhythmias,a family history of early sudden cardiac death or a QTc interval of 500 msec or more,beta adreno receptor blockers such as nadolol at maximally tolerated doses are recommended.Implantation of a permanent pacemaker to prevent bradycardia or pauses that may predispose to the development TdP may be indicated.In patients with syncope or aborted sudden cardiac death ,an ICD is warranted.These patients should be managed

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with concomitant beta blockers.An ICD is benificial in these patients not simply because of its shocking capabilities,but also because of the ability to pace continually for prevention of bradycardia which induce TdP and algorhythms to prevent post-PVC pauses.Participation in competitive sports,previously contraindicated for patients with congenital LQTS ,has been liberalized.(7,8)

For patients with acquired LQTS stopping of drug is mandatory.Hypokalemia and hypomagnesemia should be corrected.If patient has Torsades de Pointes these patients should be treated with atrial or ventricular overdrive pacing and intravenous magnesium.

In hopitalised patients prolonged QT interval is caused by multiple factors.In case series of 11 patients reported by Genevieve C.Digby et al patients had multiple factors .In this case series all patients received drugs which prolong QT interval. All patients had hypokalemia and hypomagnesemia.In one patient dilated cardiomyopathy was present(9).In our case series of 4 patients 2 patients it is caused by amiodarone and one patient it is caused by parenteral quinine.In one patient it is caused by hypokalemia and hypomagnesemia.In our case series two patients had underlying heart disease.In one patient it caused by amiodarone ,hypokalemia and hypomagnesemia with low ejection fraction. In our case series only one patient had Torsades de Pointes which responded to intravenous magnesium.In remaining patients because of timely withdrawl of drugs responsible for it and correction of electrolyte disturbances we could prevent malignant ventricular arrhythmias.

Conclusions:

This case series highlights the multifactorial causes for development of prolonged QT interval in hospitaised patients. Early recognition of this abnormality on ECG and timely withdrawl of the drug and correction of electrolyte disturbances is very important for preventing lethal complications associated with prolonged QT interval.

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FIGURE LEGENDS :-

Fig1(case1) :ECG showing sinus rhythm with prolonged QT interval with ventricular bigemini with R on T phenomenon

Fig2(case2) :ECG showing sinus rhythm with left axis deviation and T wave inversion with prolonged QT interval

Fig 3(case 3) :ECG showing sinus rhythm with prolonged QT interval with T wave alternans

Fig4 (case 4) :ECG showing sinus rhythm with prolonged QT interval

Table 1 - Patient characteristics of 4 cases