Case Report

Terlipressin Induced Ventricular Tachycardia

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Abstract

Terlipressin, a vasopressin analogue, is commonly used to treat oesophageal variceal bleeding . Ventricular Tachycardia, a fatal arrhythmia due to Terlipressin, is a well known, but under reported complication. We report a case of broad complex Ventricular Tachycardia in a 53 year, male patient during treatment with Terlipressin for bleeding oesophageal varices, which reverted to normal sinus rhythm after withdrawal of drug and usage of anti arrhythmic drugs . Electrolyte disturbances, long QT interval as seen in alcoholic liver disease were found to be underlying causes for this.

Key Words: Ventricular tachycardia, Long QT, Hypokalemia, Terlipressin

Introduction

Terlipressin, a synthetic analog of triglycyl lysine vasopressin is effective in controlling bleeding varices[1-3]. The adverse effects of terlipressin include abdominal cramping, headache, arterial blood pressure elevation, acute myocardial infarction and arrhythmias.[2] Arrhythmias including ventricular tachycardia and bradycardia are known adverse effects of vasopressin, but are infrequently noticed with terlipressin. Hence, we are presenting this rare case of ventricular tachycardia following a therapeutic dose of terlipressin for oesophageal variceal bleeding, which reverted to normal sinus rhythm after withdrawal of drug and anti-arrhythmic drugs usage.

Case Report

A 53-year-old male, alcoholic for 30 years, presented with massive hematemesis. On admission his pulse rate was 114 beats/min and B.P was 130/90 mm of Hg. General Physical examination showed pallor but no signs of icterus, clubbing and lymphadenopathy. Abdominal examination showed enlarged liver with free fluid. His higher mental functions and cardiorespiratory status were normal. His laboratory findings include hemoglobin level of 10.5 g/dl, TLC 8900 cells/cu.mm, platelets -1,86,000, Total bilirubin 2.7mg/dl, direct 1.6, indirect 0.9, borderline liver enzymes and normal renal function tests. Serum electrolytes were Na-149meq/l, K-3.4 meq/l, Cl-118 meq/l. His ECG revealed prolonged QT interval (QTc) of 521msec. Abdominal ultrasonography demonstrates increased liver echogenicity and ascites. After stabilizing the patient, an emergency endoscopy was done which revealed bleeding oesophageal varices for which variceal band ligation was done. Post procedure patient was kept on I.V Fluids, Inj. Pantoprazole, Inj. Ondansetron and Inj. Terlipressin. Following 3rd dose of Terlipressin, after 20 minutes, patient developed asymptomatic Ventricular Tachycardia(VT), noticed on cardiac monitor (Figure 1). Cardioversion to normal rhythm was done with amiodarone bolus and infusion. Laboratory tests repeated after the episode revealed mild Hypokalemia (K-3.2 meq/l) but serum sodium, calcium and magnesium levels were normal. Electrolyte imbalance was corrected by intravenous replacement.

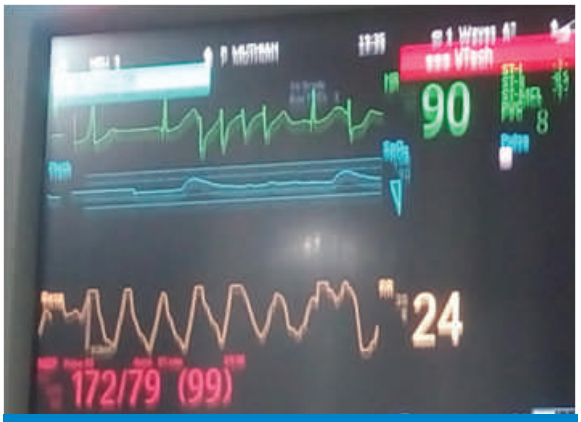

Fig 1 - Monomorphic Ventricular Tachycardia

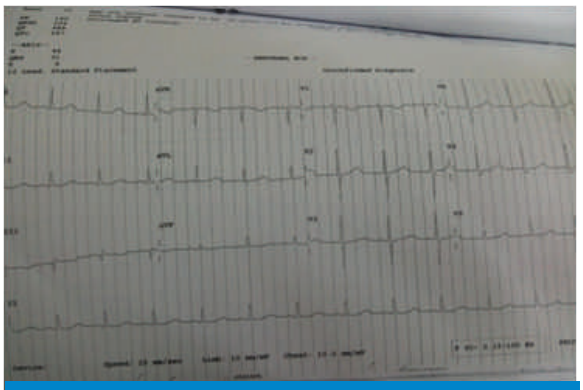

Fig 2 - Baseline ECG with Prolonged QT

ECG taken 1 hr before showed sinus tachycardia, 109 beats/ min and QTc prolonged to 547msec (Figure 2). Post cardioversion ECG revealed prolonged QTc persisting for the next 5 days (Figure 4). BP readings were 156/86 mmHg at 07.30 pm and 170/100 mmHg at 08.00pm. Corresponding pulse rates were 58 beats/min and 70 beats/min, but before the episode of arrhythmia, heart rate decreased to 52 beats/min. A myocardial infarction was ruled out with normal 2D echo and Cardiac panel study. Terlipressin was immediately discontinued and he was kept on injectable octreotide and amiodarone. Patient improved gradually and there were no further episodes of VT.

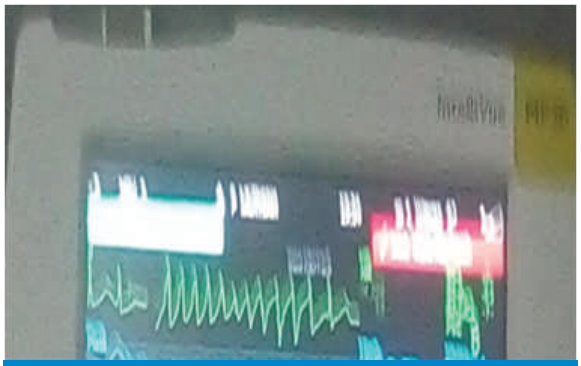

Fig 3 - VT on Cardiac Monitor

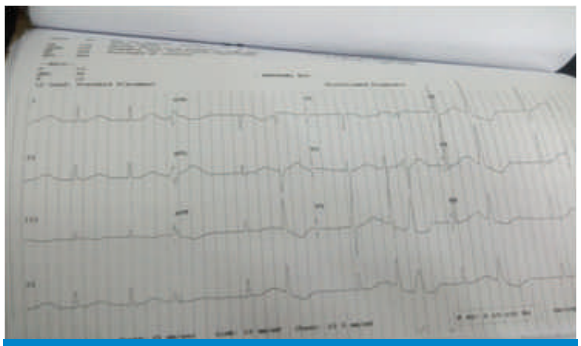

Fig 4 - Post Cardio version ECG

Discussion

A prolonged QT interval, is the most common ECG finding in patients with liver cirrhosis.[4,5] Prolongation of the QT interval predisposes the patients to polymorphic ventricular tachycardia called Torsades de pointes[6]. Delayed repolarization of cardiac muscle cells, because of potassium channel irregularities and sympathoadrenergic overactivity will lead to QT interval prolongation[7]. Major factors responsible for QT interval prolongation in cirrhosis are in Table 1[7].

Most patients who develop drug-induced arrhythmias have underlying risk factors[8,9]. Hypokalemia and hypomagnesemia, are the most important factors responsible for drug-induced long QT syndrome.

In above case he had mild prolongation of the QTc and with terlipressin, it progressed to an episode of VT. Here, the decompensated liver and the electrolyte imbalance were mainly responsible. Patient had hypokalemia, which caused VT in a patient with compromised liver function and terlipressin administration.

Conclusion

The major factor leading to pro-arrhythmogenic action of vasopressin and terlipressin is electrolyte imbalance. The present case shows the need for careful use of intravenous terlipressin and meticulous care of patients during treatment, with cardiac monitoring and correcting electrolytes, and limiting its use to proven variceal bleeding.

Factors	Examples
Decompensated liver	High Child Pugh class, High MELD score, GI Bleeding
Blood parameters	Electrolytes, Creatinine, Aldosterone , Nor epinephrine .
Fluid overload	Left ventricular end Diastolic parameters
Coronary events	Risk factor- Elder Age , DM , Smoking & Alcholism
Drugs	Terlipressin, Erythromycin.

Table 1 - Factors affecting QT prolongation in cirrhosis patients7

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