Amiodarone in junctional ectopic tachycardia: A word of caution!

Dear Editor,

Junctional ectopic tachycardia (JET) is a supraventricular tachycardia and constitutes the commonest arrhythmia following congenital cardiac surgery with an incidence ranging from 2-11.2%.[1] The resultant tachycardia and atrioventricular (AV) dyssynchrony can result in significant hemodynamic compromise. Therefore, rate control is paramount in such a scenario. Despite being self-limiting, it is challenging to control the heart rate in patients with refractory JET necessitating the administration of a range of antiarrhythmics. We report a patient with refractory JET post-intracardiac repair (ICR) for Tetralogy of Fallot wherein the patient developed multiple episodes of polymorphic ventricular tachycardia (VT) while receiving an amiodarone infusion for rate control in JET.

Multiple predisposing factors for post-operative JET such as young age, surgery in the proximity to AV node, cardiopulmonary bypass (CPB) duration >90 minutes, hypomagnesemia, hyperthermia, and co-existing anemia.[1] The index patient demonstrated a majority of the aforementioned factors (9 months age, ICR procedure, 110 minutes CPB duration, and hyperthermia) and therefore, enhanced susceptibility to JET. JET was diagnosed on a

12-lead electrocardiogram (ECG) as a heart rate (HR) of 160-180/minute with a narrow-complex tachycardia and AV dissociation [Figure 1].

An institutional protocol of JET management was followed, which comprised of hyperthermia control, sedation to attenuate the adrenergic surge, maintaining euvolemia, correcting dyselectrolytenemia, magnesium supplementation, and optimal inotrope infusion dosage (as inotropic stimulation predisposes to JET). The present patient manifested moderate right ventricular dysfunction on echocardiography, which necessitated adrenaline 0.05 μg/Kg/min and dobutamine 5 μg/kg/min infusions. Following rate control failure, the patient was digitalized. As JET was still refractory, 5 μg/kg intravenous (IV) amiodarone bolus over 20 minutes was followed by 10 μg/kg/min infusion achieving an HR of 120-140/minute and stable hemodynamics, with a mean arterial pressure ranging from 65-75 mmHg. On the second postoperative day, the patient developed episodes of polymorphic VT (4-6) while receiving amiodarone infusion for the preceding 12 hours. The potassium level in blood gas analysis was 4 mmol/L. These episodes were managed with defibrillation as they were associated with hemodynamic compromise. On a subsequent meticulous evaluation of the ECG with regards to the tachycardia correction (Bazett's formula), QTc (corrected QT interval = QT/√RR = 300/√0.36 = 300/0.6 = 500 msec), was found to be prolonged despite normal QT (300 msec). In this context, amiodarone infusion was believed to be the cause of QTc prolongation as there was no co-existing hypocalcemia. Considering the observation that there were no new episodes of torsades de pointes (TdP) after cessation of amiodarone infusion with a resultant normalization of QTc interval 48 hours after the termination of drug infusion, the existing temporal relation attributed the amiodarone-induced QTc prolongation as the most likely cause of TdP.

Amiodarone is a broad-spectrum class III anti-arrhythmic agent, which is conventionally presumed to exhibit the low potential of causing QTc prolongation and drug-induced TdP (<0.5%).[2] However, in contrast to this notion, certain studies have reported the incidence of hemodynamically significant TdP after IV amiodarone therapy to be around 1.5%, elucidating that the incidence might be underestimated based on studies evaluating chronic oral dosing.[3] Some other researchers have elaborated on the fact that the risk involved with the proarrhythmic potential of amiodarone is often underrated.[4] Tong *et al*. implicated amiodarone as the cause of drug-induced TdP in as high as 54% in their case series.[5] The studies have revealed a higher incidence in the female sex, co-existing hypokalemia, ventricular dysfunction, and digoxin pre-treatment.[6] The peculiarity of drug-induced

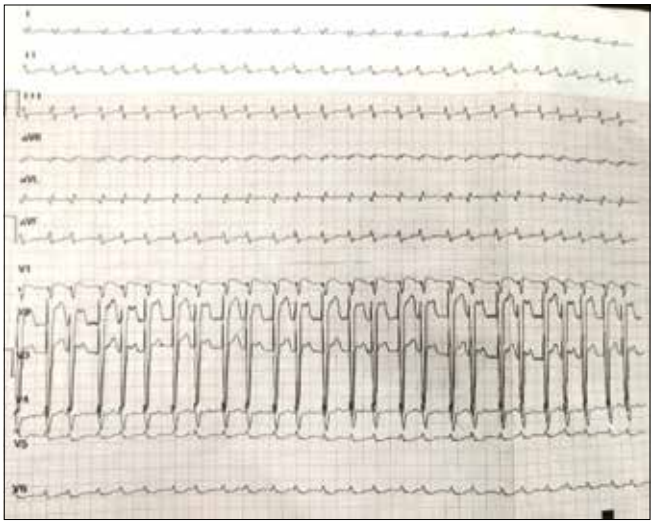

Figure 1: The 12-lead postoperative electrocardiogram depicting JET (narrow complex tachycardia)

TdP is the prolongation of QTc in the background of a normal QTc before drug initiation, pause dependent onset of polymorphic VT culminating as ventricular fibrillation and subsequent normalization of QTc almost 48-72 h post drug withdrawal as evident in the index patient.[7]

To conclude, IV amiodarone therapy in JET can precipitate drug-induced TdP in a predisposed clinical setting. The index patient highlights the importance of vigilant monitoring and surveillance for early detection of QTc prolongation in the background of anti-arrhythmic therapy as the conventional antiarrhythmics have proarrhythmogenic potential. Moreover, the present case also elucidates the importance of the on-going search for the inclusion of more selective and safer drug therapies to the modern antiarrhythmic armamentarium.

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Conflicts of interest

There are no conflicts of interest.

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