IVABRADINE: BOON OR BANE??

Varsha Rakshit Prakash
Prakash Vadagenalli Sathyarayananarao

Introduction: Most of the drug-induced arrhythmias relate to prolongation of QT interval on the ECG, which can lead to polymorphic ventricular tachycardia (VT) and ‘Torsades de Pointes’ (TdP), which in turn may induce ventricular fibrillation (VF) and sudden death.

Methods: Objective: To present two cases of Ivabradine causing QT prolongation leading to a VT storm.

Result: Case report 1 A 70 year old lady, k/c/o IHD- status post PTCA presented with features of acute decompensated heart failure. ECG showed sinus tachycardia with LBBB, Echo showed Global hypokinesia of LV with severe LV systolic dysfunction (EF-30%). Patient was started on treatment for heart failure. In view of tachycardia, tablet Ivabradine was added. Soon after, patient developed episodes of polymorphic VT. Patient was started on anti arrhythmics and Intra Aortic Balloon Pump (IABP) support for the same, however the episodes of VT continued. In view of suspected drug induced polymorphic VT, tablet Ivabradine was stopped. Patient was also initiated on anti tachycardia pacing which was gradually reduced after 24 hours, following which patient had no further episodes of VT. Patient finally underwent CRT-D and was discharged in a stable condition.

Case report 2 A 45 year old lady, k/c/o dilated cardiomyopathy presented to the hospital with effort dyspnoea (NYHA class III) on enalapril, bisoprolol and spironolactone long term. ECG done showed sinus tachycardia and underlying LBBB. 2D echo showed dilated LA, LV, severe LV systolic dysfunction EF-25% with global hypokinesia. Patient underwent CRT-P. On the same day, patient was initiated on Tab Ivabradine 5mg BD in view of sinus tachycardia. Patient received 6 doses of the same. Two days later patient had sudden onset unresponsiveness. Monitor showed polymorphic VT, patient was defibrillated to sinus rhythm. Patient was put on IABP support & IV esmolol for the VT storm and Tab Ivabradine withdrawn. Patient continued to have recurrent episodes of polymorphic VT. A Bilateral cervical sympathectomy was planned. However, patient died of refractory cardiogenic shock.

Conclusion: Ivabradine is an If channel blocker, with a half life of 11 hours that leads to a reduction in the slope of the diastolic depolarization of the pacemaker action potential, thereby slowing the heart rate. Studies have shown that the hERG potassium channel inhibition by Ivabradine may contribute to QT prolongation and risk of TdP especially when taken with diuretics or other medicines that prolong the QT interval. In both the above cases, Ivabradine was co administered with diuretics which may have triggered the polymorphic VT. Ivabradine has been given a class IIa indication by the European Society of Cardiology for use in management of symptomatic patients with heart failure with a reduced ejection fraction (HFrEF). However, in view of its life threatening side effects as demonstrated above, it must be used judiciously.