Management of incessant junctional ectopic tachycardia presenting as severe left ventricular dysfunction with Ivabradine

Nitin Parashar
Nitin Naik

Introduction: Junctional ectopic tachycardia (JET) is an uncommon entity, seen in patients with structural heart disease or postcardiac surgery. It can rarely present in infancy in isolation as congenital JET. The management of this tachycardia remains very challenging for the cardiologists. Ivabradine is a new generation antiarrhythmic agent, acts by inhibiting funny current (If) in SA node, and has been recommended to treat coronary artery disease and congestive heart failure in certain situations. It has been used off-label in inappropriate sinus tachycardia and atrial fibrillation as well for the control of heart rate (HR). We treated one patient of severe left ventricular (LV) dysfunction due to incessant congenital JET with Ivabradine. This case is being presented here in view of rarity of arrhythmia and its successful management with non-invasive means.

Methods: A 4-year old boy presented with poor feeding and restlessness since late infancy. Later he developed easy fatigability, abdominal distension and shortness of breath. Parents also noticed abnormal pulsations in precordial area. He was found to have tachycardia, and received oral diuretics with some improvement in symptoms. Beta blockers and amiodarone had no effect on HR. His condition deteriorated in last one year with gross congestive heart failure (CHF). Physical examination revealed HR of 160 bpm with presence of S3, hepatomegaly and ascites. Electrocardiogram (ECG) of the patient showed narrow complex tachycardia with AV dissociation suggestive of JET (Image 1a). Echocardiogram revealed left ventricular ejection fraction (LVEF) of 15-20%. The attendants were unwilling for electrophysiological testing and radiofrequency ablation. Cautiously, patient was started on oral Ivabradine in the dose of 0.1 mg/kg/day divided in two doses along with a diuretic.

Result: Six hours after the initiation of Ivabradine, his HR decreased to 138 bpm; and further came down to 97 bpm in 24 hours. ECG showed junctional rhythm with intermittent sinus beats. His Holter monitoring showed normal sinus rhythm with intermittent junctional ectopic rhythms with a rate of 70 bpm. Burden of accelerated junctional rhythm had also decreased to 35%. Echocardiogram after 4 weeks revealed significant improvement in LVEF (35-40%). Then patient was continued on Ivabradine along with diuretic, beta blocker, ACE inhibitor and spironolactone. Follow up echocardiogram after 1 year showed normal LVEF (60-65%) and 24hr ECG monitoring showed intermittent junctional rhythm with adequate control of ventricular rate.

Conclusion: Our patient had severe LV dysfunction and heart failure caused by incessant JET. We believe that control of HR was due to the effect of ivabradine, previously resistant to beta blockers and amiodarone. Early detection and timely treatment of JET with ivabradine can control the HR along with improvement in LV function.