**INCESSANT VENTRICULAR TACHYCARDIA FOLLOWING PERMANENT HIS BUNDLE PACING**

Ulhas M Pandurangi  
Aishwarya S  
Kotti K  
Jaya Pradhap  
Radhika B  
Muthu Seenivasan  
Aparna Balaji  
Nagendran C  
Dinesh Seenivasalu  
Mahima P Manoj  
Nithin G  
Sailendri G  
Benjamin S  
Ravi Kumar  
Nirmala S  
Dasari Himaja  
Sandini S  
Swathi K

**Introduction:** Permanent His Bundle Pacing (PHBP) is emerging as the most physiological way of cardiac pacing. The issues related with acute performance of the pacing lead are being recognized. Occurrence of sustained ventricular tachycardia presumably due to myocardial and conduction tissue injury caused by active fixation lead at the His bundle has not been reported so far.

**Methods:** A 65-year-old female, rheumatic heart disease, mitral valve replacement (22mm OmnicarbonTM mechanical mitral prosthesis, 17 years back) normally functioning prosthetic valve, moderate aortic regurgitation and severe LV dysfunction (EF 30%, LVID(d) 65mm) underwent PHBP for syncope due to sick sinus disease with wide QRS ventricular escape rhythm (Fig1). The bradycardia was considered not due to any reversible causes. At a site (Fig2) where the His bundle lead (Medtronic SelectSecureTM 3830) recorded His bundle potential (Fig3), unipolar pacing (2.0V @1ms threshold) resulted in non-selective HBP with QRS duration of only 20ms more than the native QRS (Fig4). Post screwing (3 turns) bipolar HBP resulted in non-selective, preferential left bundle pacing (1.5V @1ms threshold) with further QRS widening (Fig5A). To achieve better paced QRS morphology it was decided to pace at a different site. Immediately after the lead was unscrewed, patient developed sustained monomorphic tachycardia of the morphology similar to the paced morphology (Fig5B). The tachycardia was hemodynamically unstable. Three attempts of external electrical cardioversion (100J-250J) failed. Adenosine (12mg IV) terminated the tachycardia for few seconds. Atrial and ventricular overdrive pacing including from the site of HBP (Fig6) could not terminate tachycardia. The diagnosis of ventricular tachycardia was established by these maneuvers. A single radiofrequency ablation lesion (40W, 50°C, 7F TherapyTM ablation catheter) at the site where the lead was screwed-in (Fig7 and 8) terminated the tachycardia in 5 seconds (Fig9). The ablation was continued for total 60 seconds. The tachycardia could not be induced by programmed atrial and ventricular stimulation even on isoprenaline.
The lead was finally screwed-in distal to the ablation site with satisfactory parameters (Fig10).

**Result**: During the 1st month follow-up patient remained asymptomatic. The injury caused by the HBP lead was still evident on the ECG (Fig11)- qRBBB and PR interval 240ms. A 24-hour Holter monitoring showed episodes of appropriate atrial and ventricular pacing and no sustained ventricular arrhythmias.

**Conclusion**: A sustained ventricular tachycardia responsive to adenosine but not to overdrive atrial or ventricular pacing can occur after unscrewing active fixation from the HBP site. The RFA at the fixation site cures the tachycardia.