Interesting presentation of a Broad complex tachycardia during the Head up tilt table test

khan Muhammad Taha
abdul baqi
leghari abid
khan Aamir hameed
Yawer Saeed

Introduction: Broad complex tachycardia (BCT) during head up tilt test (HUTT) is uncommon. Only one study reported incidence of BCT to be around 0.04%. However, patients with ischemic heart disease (IHD) can have ventricular tachycardia/fibrillation during HUTT with isoproterenol augmentation. We describe a case of BCT induced during HUTT without isoproterenol in a patient being evaluated for pre-syncope.

Methods: A 45 year old man with prior history of IHD and percutaneous intervention to LAD coronary artery 10 years ago presented with symptoms of pre-syncope. His recent echocardiogram showed left ventricular ejection fraction of 45-50%, no diastolic dysfunction or significant valvular abnormality. His echo and HUTT were done because of pre-syncopal symptoms as pre-operative evaluation for anal fistula surgery. During HUTT his ECG showed short RP tachycardia at rate of 139 beats/minute with broad QRS complexes but no change of axis. (Figure 1A) His investigations and his ECG traces during HUTT were reviewed and an Electrophysiological (EP) study was scheduled to discern type and origin of BCT as the differential were ventricular tachycardia (VT) and Antidromic atrioventricular reentry tachycardia (AVRT). EP study was performed with fluoroscopy guidance. During EP study ventricular pacing was decremental with central coronary sinus activation while atrial pacing showed no pre-excitation. Post-dobutamine augmentation, BCT was induced with leftward axis and transition between V1–V2 at a ventricular rate of 140beat/min. During BCT patient remained hemodynamically stable. BCT was confirmed as VT with AV dissociation with faster atrial pacing. Ventricular entrainment showed post pacing interval of 45 msec from mid ventricular septum and 110 msec from right ventricular apex. So it was concluded that VT focus was likely from mid-interventricular septum. Patient was counseled regarding VT and ICD therapy however patient refused ICD implantation. As ablation would have been close to His bundle and patient was also due for anal fistula surgery soon patient was kept on amoidarone. Post anal fistula surgery patient had persistent symptoms of pre-syncope. VT ablation was then planned however the 3D mapping identified the VT focus about 10mm away from the His bundle on the LV side.

Result: Parahisian VTs accounts for up to 10% of all idiopathic VTs and are defined by origin with in 10mm of His cloud or those with earliest recorded activation in presence of a His potential after mapping of all nearby structures. Such VTs are difficult to ablate as there is high risk for AV block. VT ablation in such cases requires a systematic approach with expertise along with 3D mapping.

Conclusion: This case highlights unusual occurrence of VT of parahisian origin during HUTT. The use of EP study to reliably diagnose the arrhythmia as VT and use of 3D mapping to identify parahisian region as the VT focus.