Mechanism of Sudden Cardiac Death in Brugada Syndrome : From the Clinical Point of View

Kenzo Hirao, M.D.

Tokyo Medical and Dental University
Tokyo, JAPAN

The mechanism of Brugada syndrome (BrS) has traditionally been proposed to be exclusively linked to repolarization abnormalities. Phase 2 reentry could be responsible for PVC triggering ventricular fibrillation (VF). However, recent clinical studies showed the evidence of depolarization abnormalities. Conduction abnormalities may serve as a substrate for VF in BrS patients.

Conduction delay (CD) can be detected by such clinical findings as follows; 1) Intraventricular CD as right BBB in ECG, 2) His-ventricular CD as the H-V prolongation in His electrogram, 3) Ventricular CD in a small mass as late potential in signal averaged ECG, 4) Ventricular CD in a large mass as fragmented QRS, 5) CD in the RV outflow tract epicardial region demonstrated by conus brach recording, and 6) CD in the RV epicardial region demonstrated by delayed and fragmented electrograms recorded by epicardial catheter mapping.

We experienced a 38 y/o male with VF due to BrS and J-waves who suffered from VF storm under intensive drug therapy. Epicardial RV mapping showed delayed and fragmented ventricular potentials, to which catheter ablation was performed and was effective to control VF.

The mechanism of VF in a patient with BrS is linked to depolarization abnormalities, which may be a substrate of VF due to ventricular CD caused by anatomical or functional abnormalities. The substrate of VF in BrS can be efficiently modified by catheter ablation, which may be promising alternative therapy in BrS patients.