

## Clinical Vignette Sample from 2006

### BE STILL MY HEART! A SHOCKING DIAGNOSIS IN A 54 YEAR OLD MAN WITH NON-ISCHEMIC CARDIAC ARREST

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#### LEARNING OBJECTIVES:

1. Generate an appropriate differential diagnosis for non-ischemic causes of ventricular fibrillation.
2. Recognize Type I Brugada syndrome and understand the appropriate therapeutic options as well as the implications of the diagnosis.

#### CASE:

A 54 year old Caucasian man with schizophrenia, bipolar affective disorder, and dyslipidemia was observed by a bystander in a movie theater to suddenly collapse. CPR was initiated by a physician in the theater prior to the arrival of the paramedic team. The patient was noted to be pulseless at the onset of CPR and further, by the paramedics, to be in ventricular fibrillation. This rhythm resolved to normal sinus after three consecutive shocks with an automated external defibrillator. After transport to the hospital and stabilization, initial ECG was read as normal sinus rhythm with a right bundle branch block (RBBB). The patient had a peak CK of 3724 U/L and a peak troponin of 0.8 mcg/L. Coronary angiogram and echocardiography demonstrated no coronary artery disease or structural heart disease. Additional history revealed a report of two previous syncopal episodes of unknown origin. Medications were olanzapine and lamotrigine. The patient denied use of supplements or illicit drugs. Family history was negative for sudden death or heart disease. The patient was transferred to our institution for implantation of an automated implantable cardioverter-defibrillator (AICD). Further review of the ECG revealed a pattern consistent with Type I Brugada syndrome. The patient remained asymptomatic during his hospital stay, and an AICD was implanted prior to discharge. The patient planned to have his family members screened for Brugada syndrome.

#### DISCUSSION:

The vast majority of sudden cardiac deaths caused by ventricular fibrillation are due to myocardial infarction. The differential diagnosis of sudden cardiac death in the absence of structural heart disease includes drug-induced arrest/medication reaction, electrolyte abnormalities, long and short QT syndromes, Wolff-Parkinson-White syndrome, and Brugada syndrome. Brugada syndrome is uncommon, with a prevalence of less than 1% in epidemiological studies. The classic ECG pattern of Brugada is pseudo-RBBB with ST segment elevation in leads V1 through V3. Clinical manifestations are nine times more common in men than women and more common in Southeast Asian populations. The pathophysiology is related to a defective myocardial sodium channel gene (SCN5A) in most patients, with variably penetrant autosomal dominant inheritance. However, the syndrome is clearly heterogenous since the characteristic ECG findings can be seen with early right ventricular dysplasia, cocaine abuse, and certain psychotropic drugs. AICD implantation is the definitive treatment for Brugada syndrome, though one study suggests that administration of high-dose quinidine prevents arrhythmia while being both less expensive and less invasive. A proper diagnosis of Brugada syndrome will further allow the internist to screen the patient's family through regular ECG testing and to counsel avoidance of common medications that can precipitate a ventricular fibrillation pattern, including tricyclic anti-depressants and sodium channel blockers.