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# Cardiac Arrhythmias and Syncope

## A Case Study by Dr Jithin Sajeev

A 65-year-old man presented for cardiac Assessment. He is an avid cyclist and often rides his bike upwards of 400 kms a week without any cardiac symptoms. He had a long standing (>20-year) history of premature ventricular complexes and periods of non-sustained ventricular tachycardia. There was also a history of syncope and pre syncope.

The initial work up in his 40's included ECGs, echocardiograms, stress testing and electrophysiology studies. No abnormalities were noted at the time on diagnostic testing apart from premature ventricular complexes and short asymptomatic non sustained ventricular tachycardia on Holter monitor. He was treated symptomatically with beta blockade over the next decade.

He presented for further routine review and a repeat ECG showed marked abnormalities with inverted T waves and fractionated QRS complexes in lead V1 – V3. These features were concerning for possible **arrhythmogenic right ventricular cardiomyopathy (ARVC)**. Re-evaluation of cardiac risk undertaken. Echocardiography showed a dilated right ventricle with moderately reduced contraction.



Cardiac MRI revealed severely dilated right ventricle with severely reduced systolic function and fibrofatty replacement. These findings met the international task force definition for diagnosis of ARVC.

ARVC is a genetic defect of the intercellular junction called “desmosomes”. Most common presentation is with ventricular arrhythmias, ranging from premature ventricular complexes to ventricular tachycardia and fibrillation that lead to cardiac syncope or sudden cardiac death. It is associated with variable phenotypic expression and penetrance. Family history of sudden cardiac death or ARVC is reported in only 50% of patients. Pathogenesis is associated with rupture of desmosomes and resultant necrosis of myocytes with eventual replacement, with fibrofatty tissue.



**Heart Week**

**May 3 - 9, 2021**



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Over time there is progressive dysfunction of the myocardium which is accelerated by exercise. Diagnosis is obtained with aid of multiple modalities which can include histology (cardiac biopsy), cardiac imaging to assess structure and function, ECG abnormalities and familial or genetic history.

This patient was counselled to minimise his intensive exercise regime including cycling, as it leads to acceleration of myocyte loss and portends a higher risk for ventricular arrhythmias. Consideration was provided for cardiac defibrillator implantation due to the heightened risk for ventricular arrhythmias as recommended by international guidelines. The patients' adult children were advised to obtain cardiac assessment and a genetic assessment was organised to identify a genetic proband.

Patients with syncope and high-risk features should always undergo aggressive work up. High risk features include an abnormal ventricular structure or function, syncope during exertion, when supine, palpitations prior to syncopal events, family history of sudden cardiac death at a young age, systolic murmurs or coronary disease, ECG abnormalities include conduction abnormalities, persistent bradycardia < 40 bpm, ventricular arrhythmias.

Arrhythmogenic syncopal mechanisms may be associated with genetic abnormalities and family history should be obtained. Initial work up could be falsely reassuring, if undertaken during the early phase of a disorder. Periodic re-stratification of risk and pathogenesis should be undertaken with multimodality assessment.



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