

Malignant Arrhythmia in a 40-Year-Old Male

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ABSTRACT

Importance ~~Traditionally~~While MVP has traditionally been considered a benign condition, recent studies have found that MVP has arrhythmogenic potential, with autopsy and clinical studies reporting ~~the condition that MVP is~~MVP as an ~~important underestimated and~~ underdiagnosed cause of arrhythmic sudden death and idiopathic out-of-hospital cardiac arrest.

~~The objective of this study was~~**Objective:** to describe a case of mitral valve prolapse in a 40-year-old male.

Design, Setting, and Participants This case report describes a 40-year-old ~~white~~White male with MVP who presented to the emergency ~~room~~department.

Interventions The patient was promptly ~~given~~administered an intravenous bolus of ~~Cordarone~~Cordarone, 300 mg, and started on an amiodarone infusion.

Main Outcomes and Measures ~~The main outcomes were~~Electrophysiology, coronary angiography, and minithoracotomy and surgical mitral valve repair results.

Results In this ~~41-year-old~~41-year-old ~~white~~White man, electrophysiologic study performed with voltage mapping revealed scars and double potentials that extended from the tip to the base of the posterior papillary muscles, extending to 2 cm toward the septum. There was inducible VT with the RBBB morphologic features as described above. Coronary angiography revealed that the patient’s coronary arteries were ~~normal~~unremarkable. Intraoperative open minithoracotomy and surgical mitral valve repair examination revealed visible scarring over the posterior papillary muscles, around which basal

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circumferential cryoablation was performed. Atrial fibrillation cryoablation and left atrial appendage plication were completed conjunctively. Three weeks after mitral valve repair, a subcutaneous ICD was implanted. At outpatient visits up to 6 months, the patient remained in sinus rhythm and free of symptomology.

Conclusions and Relevance Myocardial stretching insult is attributable to abnormal traction, increased systolic velocity, and excursion of papillary muscles. Furthermore, mechanical stretching also directly induces altered membrane depolarization indicative of ventricular arrhythmias. There is thus a theoretical electrophysiologic basis for MVP intervention for arrhythmia control, although results from surgical intervention has been variable.

Case Report: A 40-year-old ~~Caucasian~~ ~~caucasian~~ White male with a chief ~~complaint~~ ~~concern~~ of a heart valve problem presented acutely to the emergency ~~room~~ ~~department~~ with onset of palpitations and diaphoresis, preceded by several days of flulike symptoms. He was afebrile and alert, with a blood pressure of 98/62 mm Hg. The electrocardiogram showed ~~-a wide complex~~ wide complex tachycardia at a rate of 218/min with right bundle branch block (RBBB) morphologic features and northwest frontal axis. He was positive for Brugada's and Josephson's signs. There was evidence of fusion beats with intermediate Q-R-S morphologic features and width (first and 17th beats along lead 2).

The patient was promptly given an intravenous bolus of Cordarone, 300 mg, by the emergency services staff but soon became hemodynamically unstable. Defibrillation was initiated but aborted because of spontaneous conversion to a narrow complex rhythm. A subsequent electrocardiogram showed atrial fibrillation with inferior biphasic T-wave abnormalities. A physical revealed a soft S 1 sound and pan-systolic murmur at the apex radiating to the left axilla. There was no midsystolic click. Echocardiography revealed anterior mitral valve prolapse (MVP) with severe posteriorly directed mitral regurgitation (MR) and dilated left atrium. The left ventricle (LV) was mildly dilated with an ejection

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fraction of 0.53. Subsequent telemetry monitoring showed recurrent ventricular ectopic beats of similar RBBB morphologic features. The patient was started on an amiodarone infusion.

The patient's diagnosis was malignant arrhythmic MVP syndrome. Magnetic resonance imaging revealed late gadolinium enhancement in the posterior papillary muscles and midinferior LV wall that was suspicious for MVP-induced scarring.

The clinical challenge was formulation of a treatment strategy to control symptoms, improve survival and function, and prevent arrhythmia recurrence. The evidence of LV systolic dysfunction (ejection fraction <.60) in the context of severe primary MR is a class I indication (level of evidence B) for mitral valve surgery in the American Heart Association/American College of Cardiology guidelines for management of valvular heart disease, and atrial fibrillation is a class IIa indication (level of evidence B). Furthermore, intraoperative arrhythmia ablation can be performed conjunctively. Although percutaneous catheter ablation of ventricular tachycardia (VT) (and/or atrial fibrillation) is a reasonable option, this does not address the patient's need for mitral valve surgeries. Radiofrequency ablation of clinically dominant ventricular ectopic foci is efficacious in alleviating symptoms and reducing appropriate implantable cardioverter-defibrillator (ICD) shocks in a small group of arrhythmic MVP patients, but randomized trial data on hard clinical end points has yet to emerge. Our-The patient with structural heart disease and spontaneous sustained VT, regardless of hemodynamic stability, should also receive an ICD to prevent passing-away-death from a sudden cardiac event; the implantation can be performed after mitral valve surgery.

The future treatment plan-strategy was surgical mitral valve repair with intraoperative arrhythmia ablation, followed by device therapy. Electrophysiologic study performed with voltage mapping revealed scars and double potentials that extended from the tip to the base of the posterior papillary muscles, extending to 2

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cm toward the septum. There was inducible VT with the RBBB morphologic features as described above. Coronary angiography revealed that the patient's coronary arteries were normal. The patient underwent open minithoracotomy and surgical mitral valve repair. Intraoperative examination revealed visible scarring over the posterior papillary muscles, around which basal circumferential cryoablation was performed. Atrial fibrillation cryoablation and left atrial appendage plication were completed conjunctively. Three weeks after mitral valve repair, a subcutaneous ICD was implanted. At outpatient visits up to 6 months, the patient remained in sinus rhythm and free of symptomatology.

Discussion: Traditionally considered a benign condition, MVP has recently been reported as having~~recent studies have found that MVP has~~ arrhythmogenic potential, with autopsy and clinical studies reporting that MVP is an ~~important~~ underestimated and underdiagnosed cause of arrhythmic sudden death and idiopathic out-of-hospital cardiac arrest. Of importance, ventricular arrhythmic risk is independent of LV function or severity of MR.

Malignant MVP syndrome is typically encountered in young women with bileaflet MVP and a midsystolic click, which were absent in this case; however, the combination of MVP, a polymorphic or RBBB-type VT of LV origin with prevailing baseline inferior repolarization abnormalities, and frequent outflow tract ventricular ectopic activity should prompt the ~~provider~~ medical team to consider this unifying diagnosis.

Malignant MVP syndrome is effected by multifactorial mechanisms of electrical instability. Previous studies report that most patients with arrhythmic MVP have late gadolinium enhancement in the papillary muscles and inferobasal LV wall, suggesting scarring-related reentrant electrical instability.

These findings suggest a myocardial stretching insult attributable to abnormal traction, increased systolic velocity and excursion of papillary muscles. Furthermore, mechanical stretching also directly induces altered membrane

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depolarization indicative of ventricular arrhythmias. There is, thus, a theoretical electrophysiologic basis for MVP intervention for arrhythmia control, although results from surgical intervention ~~has~~have been variable.