

Mitral valve repair results in suppression of ventricular arrhythmias and normalization of repolarization abnormalities in mitral valve prolapse



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Introduction

Mitral valve prolapse (MVP) can be associated with ventricular arrhythmias (VA) and sudden cardiac death (SCD). Recently, VA and inferior T-wave inversions (TWI) have been identified as risk factors for SCD in MVP patients. We present a case of MVP with VA and inferior TWI that normalized following mitral valve repair.

Case report

A 39-year-old woman was referred to our outpatient cardiology clinic with severe mitral regurgitation (MR) secondary to MVP. Her family history was remarkable for sudden death in a 28-year-old brother, who was only found to have MVP on autopsy. On assessment, she was asymptomatic and her examination was compatible with severe MR, with no signs of heart failure or pulmonary hypertension. Her echocardiogram showed myxomatous mitral valve disease with bileaflet prolapse, severe (4+) MR, normal left ventricular (LV) size and ejection fraction, and normal pulmonary artery systolic pressure. Her electrocardiogram (ECG) showed normal sinus rhythm, inferior TWI, and frequent PVCs, which had a right bundle branch block–like pattern with transition in V₅ and a right inferior axis suggestive of an anterolateral papillary muscle origin (Figure 1). A Holter monitor revealed asymptomatic, frequent premature ventricular contractions (PVCs; 227 PVC/h) and 25 episodes of ventricular tachycardia (3–16 beats at 115–163 beats/min). She was started on bisoprolol 5 mg daily and a repeat Holter monitor continued to show frequent PVCs, although with a reduced frequency (128 PVC/h).

She remained asymptomatic with no changes in her echocardiogram until 5 years later, when she presented with atrial fibrillation and was admitted for urgent mitral valve repair.

KEYWORDS Mitral valve prolapse; Mitral regurgitation; Mitral valve repair; Sudden cardiac death; Ventricular arrhythmias (Heart Rhythm Case Reports 2018;4:191–194)

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Coronary angiography revealed normal coronaries. Her echocardiogram continued to show normal LV size and ejection fraction. She underwent an uncomplicated mitral valve repair and maze procedure. The repair consisted of a triangular resection of the posterior leaflet (mid scallop), leaflet plication, and placement of an annuloplasty band (CG future band, Medtronic, Minneapolis, MN). No reconstruction was performed on the subvalvular apparatus (Supplemental Videos 1A and 1B). Her ECG on postoperative day 1 revealed normalization of the TWIs in the inferior leads (Figure 2). She was discharged home on the same dose of bisoprolol. Her ECGs during her hospital stay and at her first postoperative outpatient visit showed no PVCs and a follow-up Holter monitor 6 months after surgery revealed a significant reduction of her PVC burden (25 PVC/h). The PVCs had similar morphology before and after surgery.

Discussion

MVP is a common valvular abnormality with a prevalence of 2%–3% in the general population. Although it is generally regarded as a benign condition, an association of MVP with SCD is accepted.

Efforts have been made to identify factors associated with SCD that can help risk-stratify patients with MVP. Clinical factors including female gender, NYHA class, and atrial fibrillation are reported to be associated with a higher risk of SCD.^{1,2} Other predictors of SCD include echocardiographic features, such as the LV ejection fraction, MR severity, bileaflet prolapse, redundant chordae, and longer leaflet length^{1–3}; and electrocardiographic features, such as TWIs and VA.²

Humphries and McKusick⁴ first described inferior TWIs in a patient with a late systolic murmur, a condition that was later described as MVP. Since then, inferior TWIs have been recognized to be associated with MVP,⁵ and recently they were found to be associated with a higher risk of SCD in patients with MVP.² The exact mechanism for TWIs is not clear. Early reports suggested papillary muscle ischemia as the cause; however, subsequent studies have not supported this hypothesis.⁶

KEY TEACHING POINTS

- Mitral valve repair results in suppression of ventricular arrhythmias and normalization of inferior T-wave inversions in mitral valve prolapse.
- Immediate normalization of T-wave inversions and suppression of ventricular arrhythmias post valve repair suggest a hemodynamic role in the pathogenesis of these findings.
- Surgical repair of mitral valve prolapse might have an important role in the prevention of sudden cardiac death.

There is no proven therapy to prevent SCD in patients with MVP. Beta-blockers are often used in patients with MVP and VA, although evidence is lacking. In 1 study of young adults with SCD in which MVP was the only identified cause, 21% of patients were receiving beta-blocker therapy at the time of the event, raising questions about their efficacy, notwithstanding the limitations of such analysis.⁷ Catheter ablation is effective in reducing appropriate implantable cardioverter-defibrillator shocks in MVP patients with recurrent PVC-triggered ventricular fibrillation.⁸ Whether surgical repair suppresses VA is controversial. There are isolated case reports of MVP patients with suppression of VA after surgical repair.^{9,10} Vaidya and colleagues¹¹ reported a case series in which surgical correction of bileaflet

MVP was associated with a reduction in VA and implantable cardioverter-defibrillator shocks.

It is still unclear whether the mechanism of VA in MVP patients is due to mechanical, electrical, or hemodynamic factors. Valve leaflet traction on the papillary muscle causing myocardial stretch or leading to endocardial friction lesions are proposed as mechanisms for arrhythmia.⁷ Late gadolinium enhancement has been identified at the level of the papillary muscle and adjacent free wall in MVP patients and correlates with VA origin, supporting this mechanism.⁷ Alteration of the repolarization properties such as the QT interval, QT dispersion, and J-point elevation have been documented in MVP patients and may play an important role in arrhythmogenesis.¹² A series of histopathologic examinations of young patients with MVP and SCD showed evidence of underlying silent but potentially arrhythmogenic myocardial substrate, suggesting that a cardiomyopathic process may also play a role; however, these could be the cause of SCD independent of MVP.¹³ Autonomic dysfunction has also been proposed to play a role.¹⁴

Our case adds to the existing evidence that shows an association between surgical correction of MVP and suppression of VA. Furthermore, our case also shows normalization of TWIs after surgery, which supports the association between these changes and VA. It is interesting to note that the TWIs normalized on postoperative day 1, which suggests that the mechanism of these changes relates to hemodynamic rather than structural factors. Hemodynamic consequences from volume overload have been proposed as a risk factor for VA. In a study of 58 patients with MVP, moderate-to-severe MR was the only independent predictor of VA in a

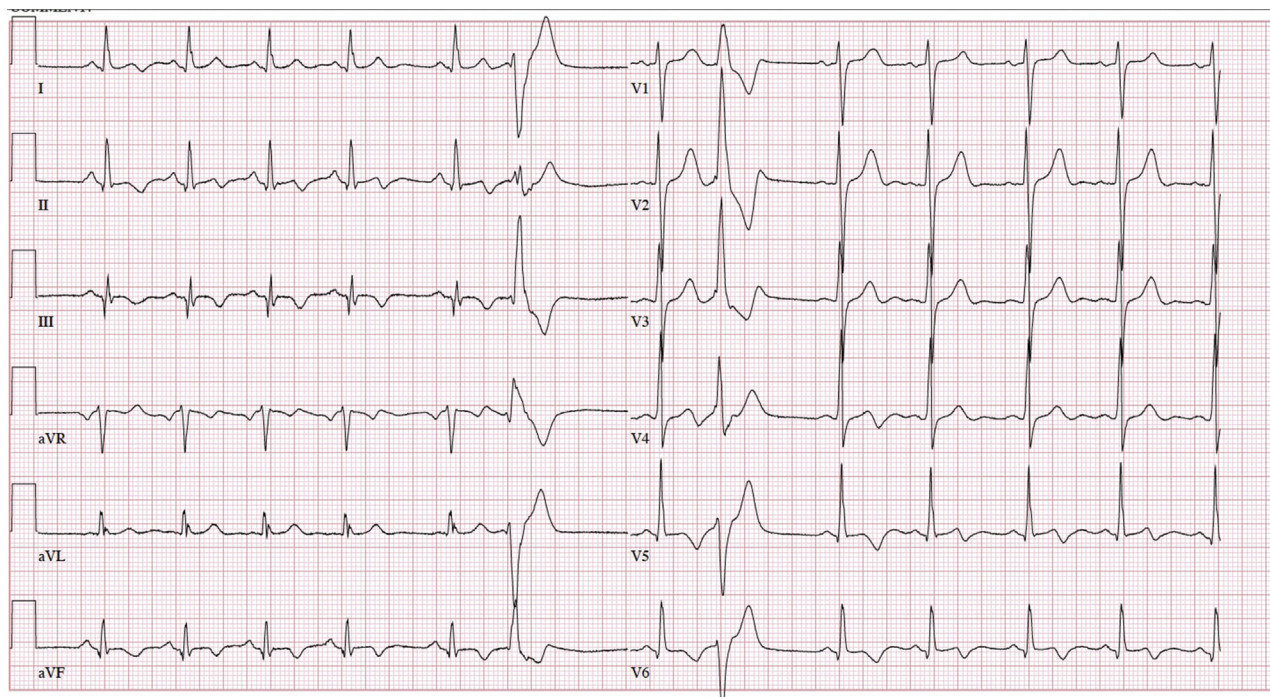


Figure 1 Baseline electrocardiogram showing frequent premature ventricular contractions and T-wave inversions in the inferior leads.

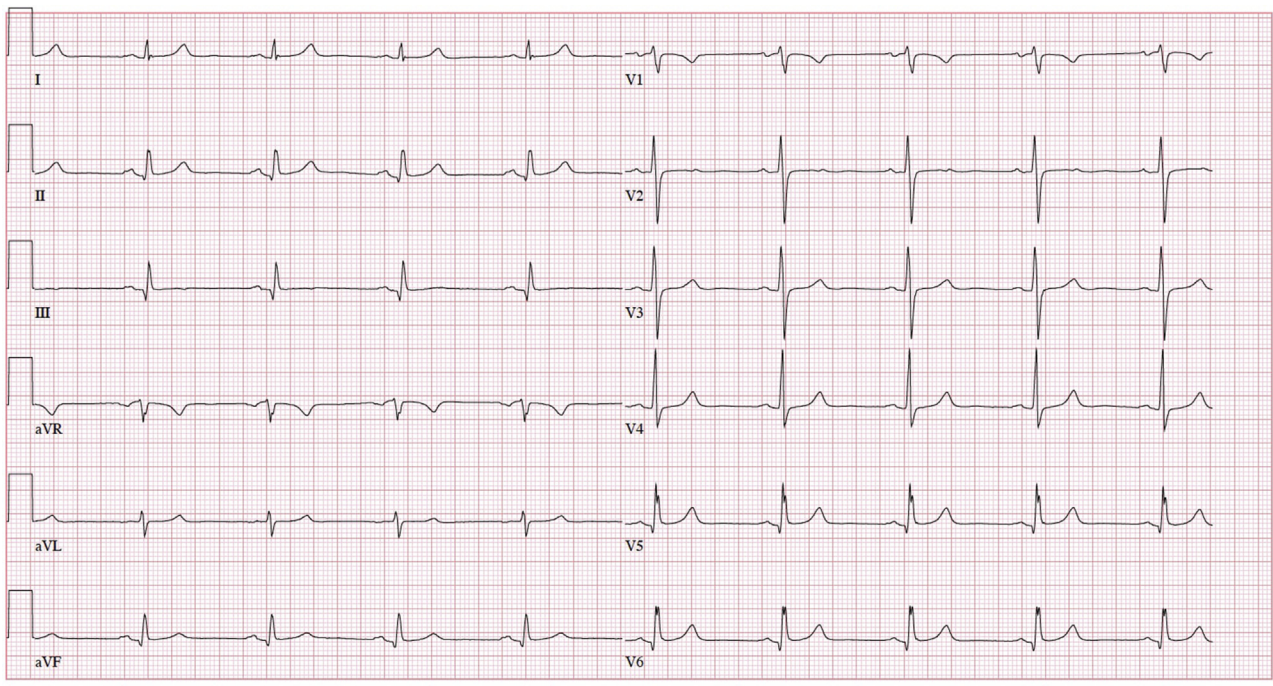


Figure 2 Postoperative electrocardiogram showing normalization of inferior T-wave inversions.

multivariable logistic regression analysis.³ Our case supports this finding. Cardiac magnetic resonance imaging was not done on our patient, and therefore it is not clear if the presence or absence of late gadolinium enhancement may be an important predictor of the subsequent normalization of TWI or suppression of VA following mitral valve repair.

While inverted T waves can be caused by “T-wave memory,” we do not believe that this was the cause in our case. It is generally accepted that inverted T waves due to T-wave memory follow the direction of the QRS complex during the preceding episode of abnormal ventricular activation. In our case, the direction of the PVC’s QRS complex in the inferior leads was positive and thus would not explain the inverted T wave in the inferior leads. Second, T-wave memory affects both inferior and precordial leads, which is different from the localized changes observed in the inferior leads in our case. Third, the immediate normalization of TWIs post mitral valve repair suggests that it was related to the mitral valve abnormalities rather than T-wave memory.

Our patient had mild shortening of the QRS complex duration immediately after surgery. We believe that this is likely a hemodynamic effect of mitral valve repair on reducing LV volume. QRS duration has been shown to correlate with LV end-diastolic volume.¹⁵ Differences in heart rate can also impact QRS duration; however, the QRS duration remained short on subsequent ECGs with faster heart rates, making this explanation unlikely.

Conclusion

We report a case of MVP with electrocardiographic risk factors for SCD, including VA and TWIs in the inferior leads. Mitral valve repair resulted in immediate normalization of

the TWIs and suppression of VA, suggesting a hemodynamic role in the pathogenesis of these findings.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2018.02.012>.

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