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Bradycardia And Ventricular Hyperexcitability Revealing Mitral Valve Prolapse With Severe Mitral Regurgitation Without Myocardial Fibrosis: A Case Report

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Abstract: Mitral valve prolapse (MVP) is a common valve abnormality in general population. Despite the general belief of a benign disorder, several articles since the 1980s report sudden cardiac death (SCD) in MVP patients, with a substantial percentage of asymptomatic young individuals. MVP is an underrated cause of arrhythmic SCD. The subset of patients with malignant MVP who may be at greater risk for SCD is characterized by young women with bi leaflet MVP, biphasic or inverted T waves in the inferior leads, and frequent complex ventricular ectopic activity with documented ventricular bigeminy or ventricular tachycardia (VT) and premature ventricular contractions (PVCs) configurations of outflow tract alternating with fascicular origin or papillary muscle. MVP is a common condition in the general population and is often encountered in asymptomatic individuals. Here, we report the case of a 52-year-old male presenting for ventricular bigeminy. Initial workup revealed a Barlow disease with mitral valve prolapse. The patient underwent mitral valve surgery with good outcomes.

Mitral valve prolapse is defined as abnormal bulging of the mitral valve leaflets into the left atrium during ventricular systole. Mitral valve prolapse is a common condition that is a risk factor for mitral regurgitation, congestive heart failure, arrhythmia, and endocarditis.

I. Introduction

Mitral valve prolapse (MVP) is one of the most common valvular heart diseases. Although MVP is generally considered benign, it can be associated with important complications, including sudden cardiac death (SCD), owing to ventricular arrhythmias (VAs). [1]. The incidence of sudden cardiac death due to ventricular tachyarrhythmias is two-fold in patients with mitral valve prolapse (MVP) compared to the general population, with the risk enhanced by mitral regurgitation (MR) [2]. In our case, we report a case of bradycardia and ventricular ectopy revealing mitral valve prolapse and severe mitral regurgitation requiring valve surgical replacement.

II. Case Report

We report the case of a 52-year-old male admitted to the cardiology department for a ventricular bigeminy. The patient had no cardiovascular modifiable risk factors. The patient consulted for palpitations. Physical exam revealed a slow heart rate and a systolic murmur of mitral regurgitation. EKG showed polymorphic premature ventricular contractions. Trans thoracic echocardiography showed severe mitral regurgitation with prolapse of segments P2 and A2 of the mitral valve. The left ventricle was dilated with an ejection fraction of 65%. The

rest of echocardiogram was normal, and there was no annular disjunction. Trans esophageal echocardiography confirmed the diagnosis of mitral valve prolapse and Barlow disease. Cardiac magnetic resonance imaging showed no fibrosis. The case was then discussed in a multidisciplinary team with cardiac surgeons. The final decision was surgical mitral valve replacement. It was performed with good outcomes, and the patient was discharged under anticoagulant therapy.

III. DISCUSSION

Mitral valve prolapse (MVP) is a common cardiac abnormality that affects 2%–3% of the general population. It is characterized by mitral valve leaflets that billow or prolapse into the left atrium. It can be diagnosed by auscultation of a mid-systolic click and mid to late systolic murmur. It is confirmed by two-dimensional echocardiography showing 2mm or more of leaflet prolapse into the left atrium in the long axis view. Although most cases of MVP are benign, a select subgroup may have life-threatening ventricular arrhythmias resulting in sudden cardiac death (SCD) [3].

Myxomatous degeneration is the most common cause of mitral prolapse in the United States and Europe, and progression of myxomatous mitral prolapse is the most common cause of mitral regurgitation that requires surgical treatment. Myxomatous degeneration appears to have genetic etiology. The genetics of myxomatous degeneration is complex and not fully worked out; it appears to be heterogeneous with multi-gene, multi-chromosomal autosomal dominance with incomplete penetrance. The molecular disorder of myxomatous degeneration appears to consist of a connective tissue disorder with altered extracellular matrix status and involves the action of matrix metalloproteinase, cysteine endoproteases, and tenomodulin [4].

The detection of ventricular arrhythmias (VAs) in subjects with MVP has been well-documented since the initial observations and was recently reconsidered and deeply investigated thanks to studies in which cardiac magnetic resonance (CMR) was used. However, the true prevalence of VAs is difficult to determine exactly because of the different MVP definitions, various populations studied, the type of VAs considered, and the eventual hemodynamical impairment, in the presence of significant mitral regurgitation that can affect the occurrence of VAs [5].

Myocardial scarring represents a well-recognized risk factor for malignant VAs in different clinical settings. In patients with MVP, fibrosis in the setting of left ventricular (LV) remodeling due to valve regurgitation is a well-known phenomenon. LGE specifically concentrated in the LV infero-basal wall suggests a crucial role of prolapse-induced mechanical forces on the myocardium in generating an arrhythmogenic substrate and triggering life-threatening arrhythmias. However, not all patients with MVP and a history of cardiac arrest have bileaflet MVP with focal myocardial fibrosis, suggesting that LV fibrosis alone cannot explain the pathogenesis of VAs in all patients. Indeed, our patient showed the same feature, ventricular arrhythmia with no associated fibrosis in CMR [5].

IV. Conclusion

Subjects with MVP experience ventricular arrhythmias more often including premature ventricular contractions, couplets, and non-sustained ventricular tachycardia compared to subjects without MVP. Our case confirms the importance of looking for valvular heart disease in front of ventricular bigeminy, even without fibrotic scar on MRI.

Competing interests

The authors declare that they have no competing interest

Figures

Figure 1: EKG of patient showing polymorphic ventricular premature contractions

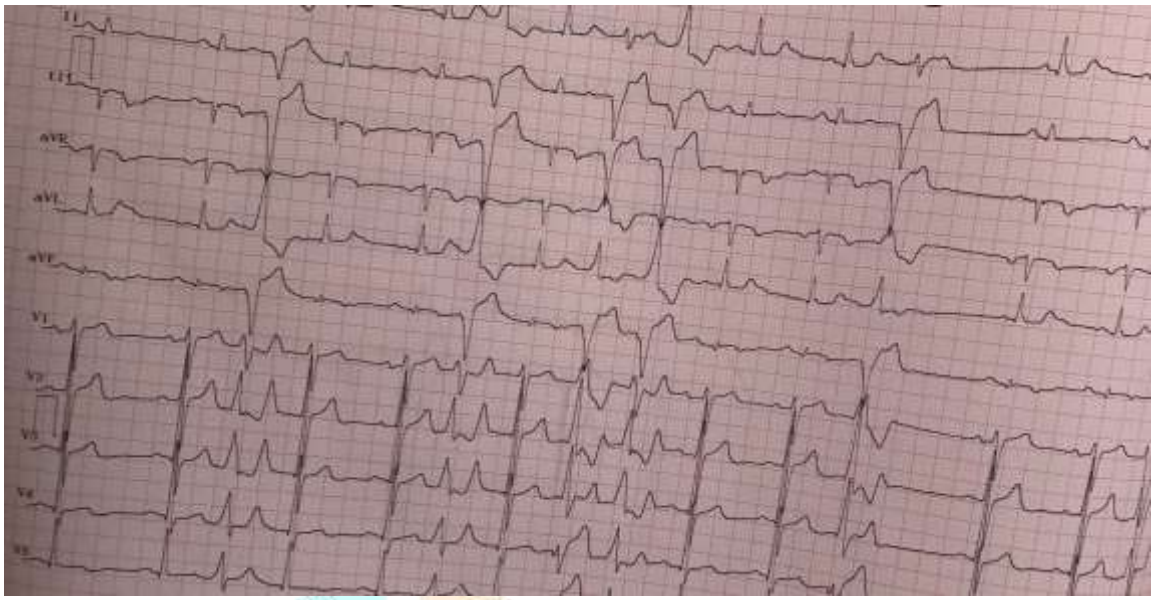
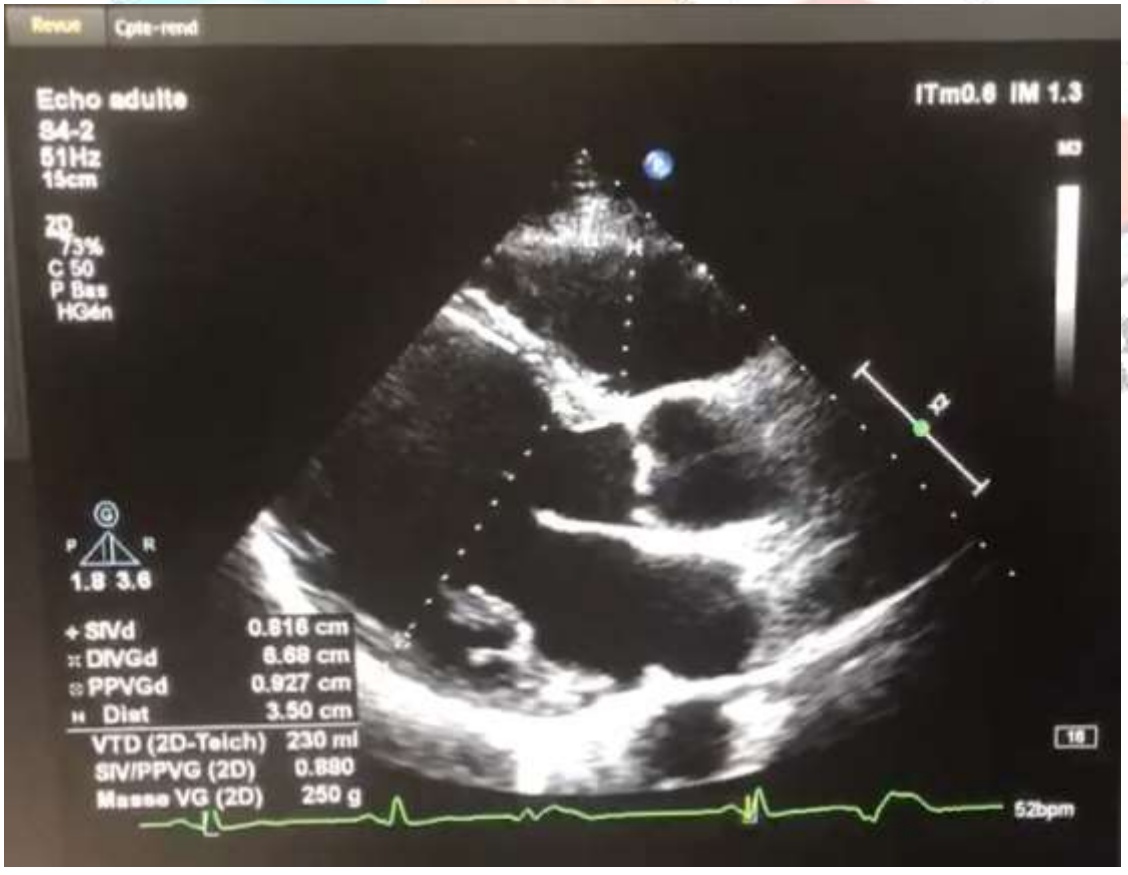
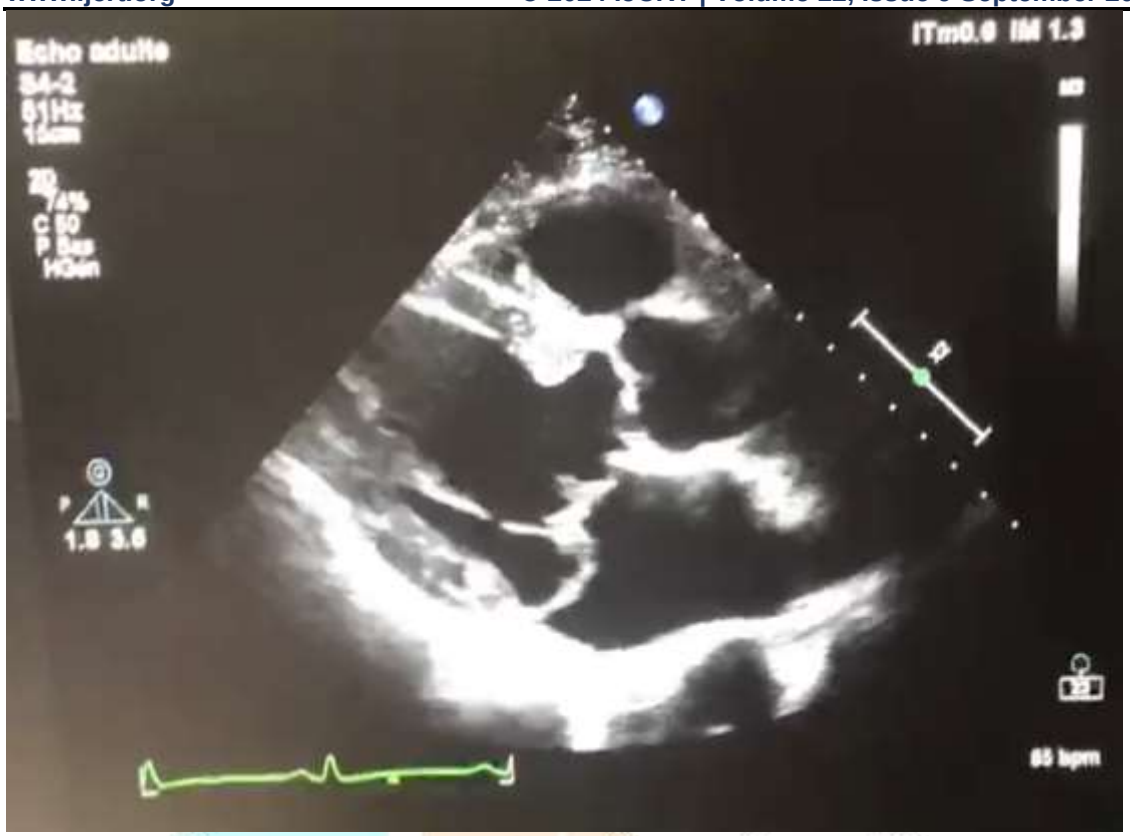


Figure 2: Transthoracic echocardiography showing a dilated left ventricle and a mitral valve prolapse





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