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Unexpected Cardiac Death in Pregnancy Associated with Mitral Valve Prolapse Case

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ABSTRACT

Background : Echocardiography frequently reveals mitral valve prolapse (MVP), which can be associated with symptoms ranging from a benign course to sometimes catastrophic sequelae, like heart failure, and infrequently, sudden cardiac death. In individuals with MVP, female sex, younger age, physiological or psychological stress, electrical instability, and structural alterations to the mitral apparatus appear to be risk factors for lethal ventricular arrhythmias. We present a rare instance of pregnant lady experiencing MVP-related cardiac arrest.

Case Report : The woman, 34, was taken to the hospital after going into cardiac arrest at home. She was eight weeks pregnant and had no prior history of heart problems. As she came at the Emergency Department, the 12-lead electrocardiogram showed premature ventricular complexes and sinus tachycardia. Her second heart arrest during her hospital stay was determined to be caused by torsades de pointes. Subsequent examinations demonstrated significant regurgitation of the mitral valve as a result of posterior leaflet prolapse and localized hypokinesis of the inferior wall and interventricular septum.

Conclusions : Mitral valve regurgitation is frequently associated with ventricular arrhythmia. It rarely has major repercussions, though. In order to prevent catastrophic outcomes, physicians must be aware of patients with MVP who exhibit indications of a prospective high-risk profile. Malignant arrhythmic mitral valve regurgitation can lead to sudden cardiac death.

KeyWords : Death, Sudden, Cardiac • Mitral Valve Prolapse • Pregnancy • Tachycardia, Ventricular

INTRODUCTION

Background

In the general population, mitral valve prolapse (MVP) accounts for 2% to 3% of echocardiographic findings [1]. Although MVP frequently has a benign course, complications such cerebral ischemia, endocarditis, and arrhythmias can be extremely concerning or even fatal [2]. One underestimates the danger of sudden cardiac mortality from MVP. In fact, we don't know anything about the underlying pathomechanisms. Many research have been conducted as a result to determine the high-risk profile of MVP patients. Nevertheless, because the majority of these investigations were retrospective, a causal link between MVP and sudden cardiac death could not be established [1-4].We describe the case of a pregnant patient who had an out-of-hospital cardiac arrest and was admitted to the hospital with a significant MVP that echocardiography verified.

Case Report

Without any previous suspicious symptoms, a 34-year-old lady unexpectedly passed out while she was getting ready for work. Her pregnancy status was G2P1001, she was eight weeks along, and she had no known history of heart problems. Apart from taking multivitamins during that time, her first pregnancy went smoothly. Furthermore, as far as her relatives knew, throughout her prenatal appointments, no abnormal cardiac abnormalities were found. She was safely brought to a nearby hospital by her husband, a pharmacist, who also provided her with urgent basic life support.A 12-lead electrocardiogram (ECG) revealed sinus tachycardia with premature ventricular complexes at the time of admission (Figure 1). Electrolyte imbalances or acidosis were not detected in arterial blood gas. Hemorrhagic stroke and high-risk pulmonary embolism were ruled out by computed tomography as the reasons of sudden cardiac death. Regretfully, the monitor caught a second cardiac arrest incident that was linked to torsades de pointes. After being revived, she was brought to our hospital for more investigations. For a thorough examination, the patient was admitted to the University Medical Center of Ho Chi Minh City's Interventional Cardiology Department's Intensive Cardiac Care Unit. Her patient's Glasgow coma

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score was 4 when she arrived.The cardiac apex was where the loudest 4/6 mid-systolic murmur could be detected. The thyroid and kidney functions of the patient were both normal. Liver enzyme levels and cardiac troponin T increased during the cardiac arrest before falling back into normal range.A twenty-four-hour Holter ECG showed sinus rhythm, bigeminy, couplets, extended QTc, short PR interval, and nonsustained ventricular tachycardia (Figure 2). Mitral valve chordae tendineae were seen on echocardiography and were in good condition. There was significant eccentric mitral valve regurgitation due to posterior leaflet P1 and P2 prolapse, resulting in vena contracta measuring 7.29 to 9.2 mm (Figure 3). Mitral annular displacement was not detected by a transthoracic echocardiography.

There was moderate dilation of the left atrium. Both hyperkinetic and maintained left ventricular systolic function were present. Nonetheless, reduced contractility was inhibited by the mid inferior wall, mid inferoseptum, and apical septum. Severe mitral regurgitation was established by cardiac magnetic resonance imaging (Figure 4). There was no sign of heart edema on T2-weighted sequences. The presence of fibrotic tissue was indicated by an increase in late gadolinium enhancement in the posterior wall's subendocardial layer (Figure 5). A panel for viral myocarditis was negative. We had to officially terminate the patient's pregnancy with her husband's consent due to her medical condition.More intrusive treatment was needed for subsequent care after two cardiac arrests. At the time, the patient was unresponsive, on mechanical breathing, and in danger of contracting pneumonia while in the hospital. Her neurological rehabilitation had an incredibly bad prognosis. A left heart catheterization was performed for the purpose of diagnosing coronary artery problems after the pregnancy was terminated. No stenosis or blockage was found on a coronary angiography (Figure 6). A surgical procedure to repair the mitral valve was necessary due to the severity of the regurgitation and the patient's complaints [5].

For arrhythmias involving ventricular regurgitation, targeted catheter ablation and implanted cardioverter efibrillation (ICD) are typically recommended in addition to surgical correction [5]. Her family, however, declined additional surgeries and invasive procedures due to her terrible neurological prognosis, only agreeing to an ICD implantation because it was a lifesaver. MVP may result in myocardial damage and signs of heart failure. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta blockers, aldosterone antagonists, and sodium-glucose co-transporter 2 inhibitors should be the cornerstones of guideline-directed medical therapy for heart failure [6].

Particularly, beta blockers can be used to treat ventricular arrhythmias and reverse left ventricular function in experimental mitral regurgitation [6]. The patient received an implanted ICD along with daily doses of metoprolol (25 mg) and lisinopril (10 mg). During her three weeks in the hospital, she got hospital-acquired pneumonia, which was treated with a course of meropenem. Three weeks following her discharge from the hospital, she experienced another episode of ventricular tachycardia, which the ICD successfully stopped at her routine monthly visit with us in the outpatient clinic.The patient remained healthy at the most recent checkup, which took place two years following her admission. Her transthoracic echocardiography showed no signs of left ventricular failure and remained nearly unchanged.

DISCUSSION

There was a strong suspicion that the young female patient had malignant arrhythmic MVP. We were unable to investigate the patient's and her family's cardiovascular histories. It's possible that the female patient had a mitral valve abnormality, but it wasn't adequately addressed during prenatal appointments. This patient would be eligible for additional evaluation by a cardiologist—which would enhance her prognosis—after a comprehensive physical examination, echocardiogram, and electrocardiogram.

The most prevalent cardiac defect in expectant mothers is MVP, a common valvular heart condition [7]. Since the patient did not have hypertension, thyroid issues, or myeloproliferative illnesses, it was improbable that she had infiltrative myocardiopathies. Furthermore, rather than infiltrative myocardiopathies, the primary cause of her echocardiographic abnormalities was assumed to be mitral regurgitation. There were no indications of bilateral atrial enlargement or pericardial thickening.

Except in cases of severe mitral regurgitation that result in cardiac failure, MVP is benign; nonetheless, some forms of MVP can also result in deadly malignant ventricular arrhythmias [7]. In their cohort investigation of single MVP patients, Essayagh et al. discovered that ventricular arrhythmias were common but infrequently led to catastrophic consequences [8]. After arrhythmogenic right ventricular dysplasia and coronary artery disease, MVP was the third most common cause of cardiac arrest in a prospective cohort study of Italian individuals under the age of 35 [4]. In those with MVP, the annual rate of sudden cardiac mortality is estimated to be between 0.2% and 0.4% [1]. Female patients with a median age of 30 and possible physiological or psychological stress, as well as frequent premature ventricular complexes or ventricular arrhythmias on Holter ECG, bileaflet MVP, and moderate mitral regurgitation on echocardiography, were proposed by Han et al. to have a high-risk profile [2].Studies examining the causal relationship between MVP and sudden cardiac death have suggested evidence of electrical instability and changing structure of the mitral apparatus, including left

ventricular fibrillation in the papillary muscles and inferobasal wall, mitral annulus disjunction, and systolic curling [1, 3]. Surgery is used to treat severe mitral regurgitation in most situations [5]. A lower risk of sudden cardiac death has been linked to correction of the flail leaflet mitral regurgitation [5].ICD installation is still advised as a class I guideline for people who suffer sudden cardiac death from ventricular tachycardia, even after surgical mitral regurgitation repair or replacement [5]. To reduce cardiac events, further research into a risk stratification model is required to decide who should receive more thorough diagnostic tests and main preventive treatments.

CONCLUSIONS

Heart diseases are the main causes of maternal mortality [9]. Significant alterations in hemodynamic and hormonal states during pregnancy may lead to myocardial infarction, peripartum myocardiopathy, and arrhythmias [10]. Given that our patient continued to be asymptomatic, it is probable that MVP had been present since her first pregnancy and had gone undiagnosed. Echocardiography is crucial for diagnosis, but it's also important to have more experience with complex MVP arrhythmias. As of right now, there are no guidelines for diagnosing or treating ventricular arrhythmias linked to MVP.Investigations using cardiac magnetic resonance and electrophysiology are restricted to specific situations. Due to a paucity of data from prospective and randomised control studies, prophylactic ICD implantation and surgical mitral valve repair or replacement are not generally recommended [1]. Beta-blockers continue to be the gold standard for treating ventricular arrhythmias, and implanting an ICD for secondary prevention is also advised [11].

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