

Mitral valve prolapse mimicking an acute coronary syndrome

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Abstract

Mitral valve prolapse (MVP), described for the first time by JB Barlow in 1966 as the displacement of abnormally thickened mitral valve leaflets into the left atrium during systole, is the most frequently valvulopathy in clinical practice. Usually asymptomatic and predominantly in woman, MVP could associate atypical chest pain. Electrocardiogram (ECG) is normal in majority of cases but sometimes ST segment depression and negative T waves in inferior leads could occur. Previous ECG and the absence of coronary risk factors are the key of differential diagnosis for acute coronary syndrome. We present the case of a 48 year-old female known with MVP, hospitalized for persistent atypical anterior chest pain, ST-T segment depression and T-wave inversion in inferior and lateral leads suggesting an acute coronary syndrome. Patient's symptoms and ECG changes have disappeared 12 hours after admission under specific treatment. MDCT coronary angiography done after 1 month showed normal coronary arteries and the treatment was stopped. This case confirms that MVP diagnosis, a rare cause of ST segment depression, must be differentiated from an acute coronary syndrome.

Keywords: chest pain, ST segment, acute coronary syndrome, mitral valve prolapsed

Introduction

Abnormalities of the ST segment and T wave are common electrocardiographic findings [1]. ST-segment or T-wave changes may be secondary to abnormalities of depolarization (QRS voltage or duration) or so called primary repolarization abnormalities (unrelated to any QRS abnormality). Some of these conditions like acute coronary ischemia or acute pulmonary embolism are life

threatening, therefore hospitalization is mandatory. Here, we present the case of a 48 year-old female known with mitral valve prolapse (MVP) hospitalized for persistent atypical anterior chest pain, ST-T segment depression and T-wave inversion in inferior and lateral leads suggesting an acute coronary syndrome. Due to the various causes of these abnormalities like acute or chronic ischemia, pericarditis, myocarditis or drugs (digoxin, antiarrhythmic, or electrolyte abnormalities, particularly potassium abnormalities), the patient was hospitalized for further investigations.

There are many papers on differential diagnosis of ST-segment depression and T-wave inversion but only a few of these mentions that MVP could be incriminated [2-5].

Received: April 2016; Accepted after review: June 2016; Published: June 2016.

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Case report

A 48 year-old female, without cardiovascular risk factors or chronic treatment at home, with medical history of MVP, spasmophilia and chronic iron deficiency anemia, was admitted for chest discomfort at rest that evolved from burning sensation to retrosternal pain associated with palpitations. During the physical examination we found a mid-systolic click, followed by a late systolic murmur.

Cardiac biomarkers for necrosis and thyroid hormones were normal; a grade I chronic iron deficiency anemia (Hb=11.1 g/dL, serum iron=40 µg/dL, ferritin=5 ng/mL) and

mild hypocalcaemia (serum calcium=8.7 mg/dL) were found. No inflammatory syndrome, dyslipidemia (total cholesterol =188 mg/dL, triglycerides= 74 mg/dL, HDLc = 72 mg/dL, LDLc = 101 mg/dL), hepatic, renal or electrolyte abnormalities were detected.

The assessment of chest pain in our patient began with an ECG. Twelve leads surface ECG at admission showed sinus rhythm of 56/min, QRS axis of about +75°, PR=160 ms, horizontal ST segment depression of 0.5-1 mm and T-wave inversion in inferior and lateral leads (Figure 1). These changes remained stable for four hours and disappeared twelve hours later (Figure 2).

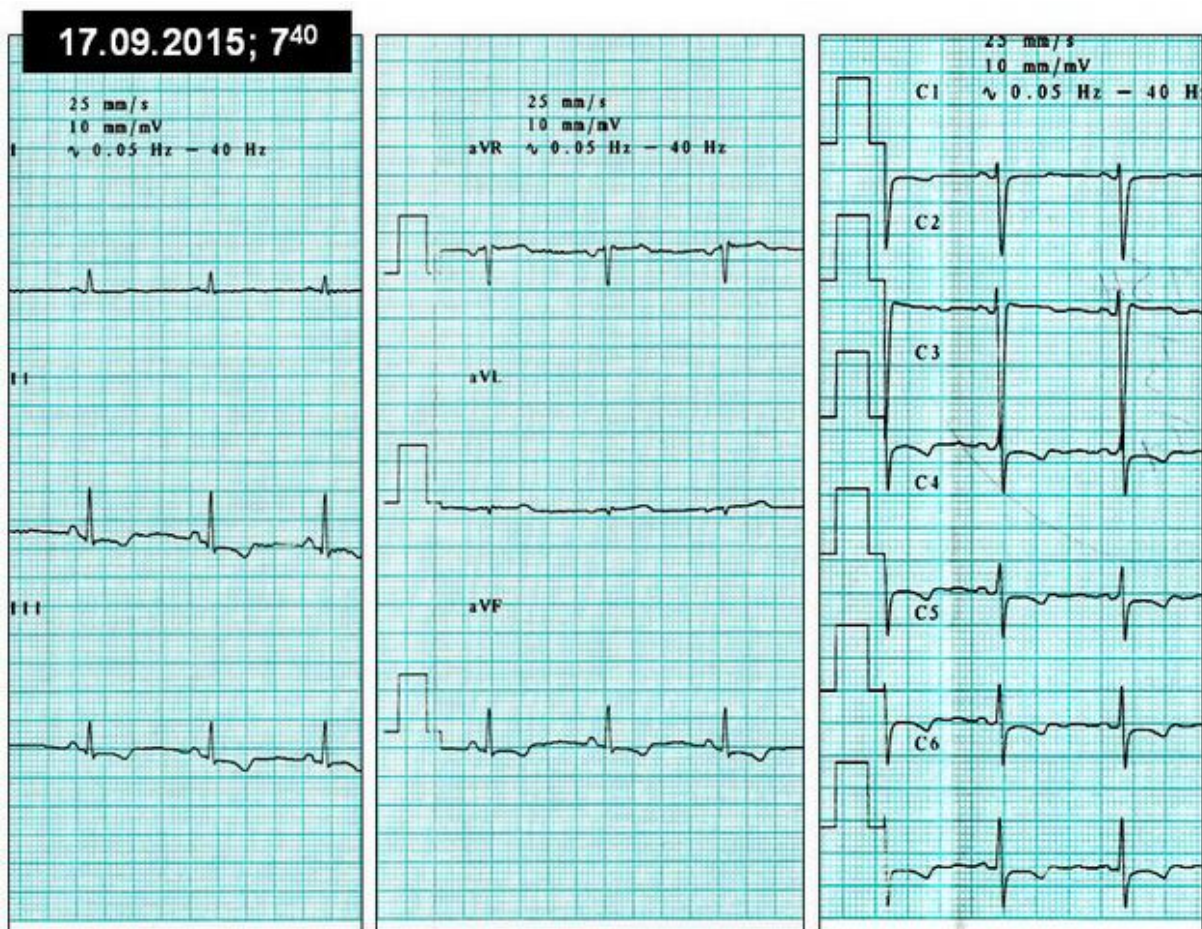


Fig. 1. The 12 leads ECG at the time of patient's admission.

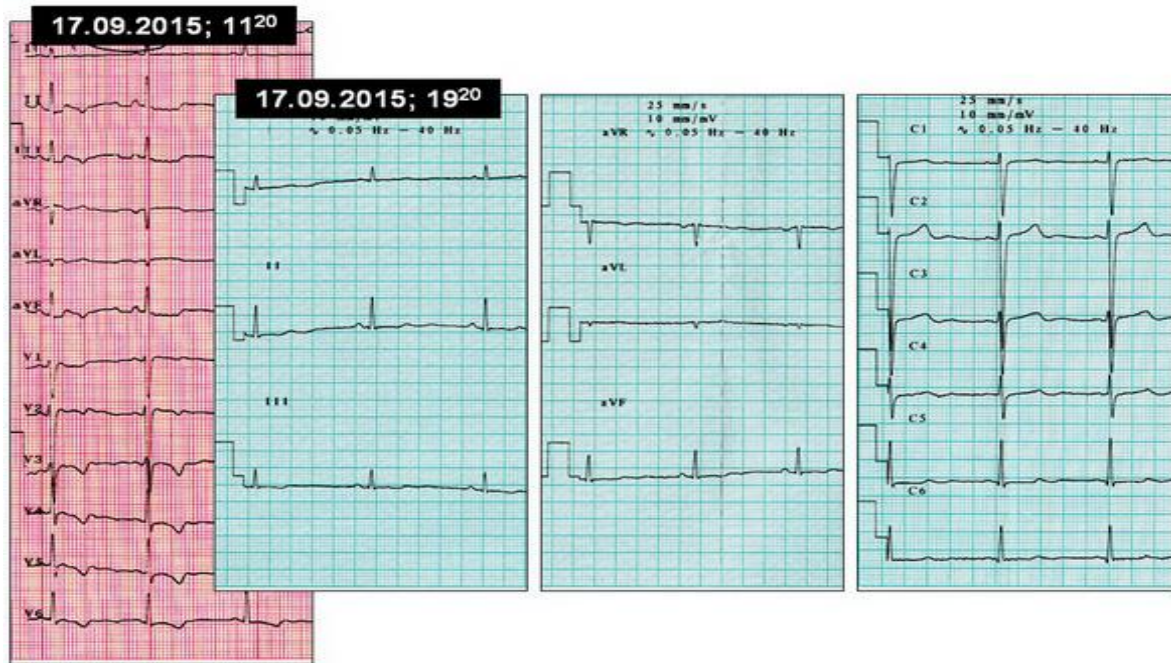


Fig. 2. Repeated 12 leads ECG after a few hours after admission.

Transthoracic bidimensional echocardiography has shown an abnormally thickened mitral valve leaflet (of 5 mm) with her displacement of 7 mm into the left atrium during systole (Figure 3), associated with mild

to moderate central mitral regurgitation at rest (vena contracta of 5.6 mm). Regional and global kinetic was normal, without any abnormalities of the other valves.

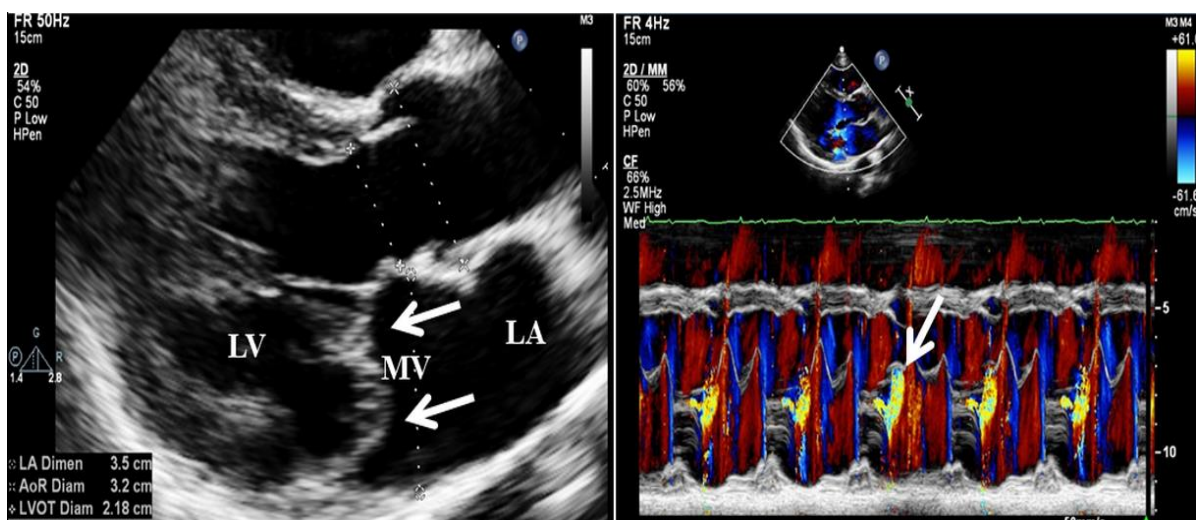


Fig. 3. Transthoracic echocardiography in parasternal long-axis view (left image), bi-dimensional and color M mode (right image), confirming the presence of a mitral valve prolapse, the displacement of abnormally thickened mitral valve leaflets into the left atrium during systole.

24 hour ECG Holter monitoring has shown stable sinus rhythm with an average of 67 beats per minute (minimum of 60 beats per minute and maximum 87 beats per minute), diffuse variable ST segment depressions and

negative T waves, rare and isolated supraventricular and ventricular ectopic beats, with uniform distribution between active and passive periods.

After admission, selective beta-blockers (Bisoprolol 5 mg once daily), statin (Atorvastatin 20 mg once daily), cytoprotective anti-ischemic agent (Trimetazidine 35 mg twice daily), anti-angina drug (Nicorandil 10 mg twice daily; with dual properties of a nitrate and K+ATP channel like), dual antiplatelet therapy (Aspirin 75 mg and Clopidogrel 75 mg once daily) and low molecular weight heparin (Enoxaparine 60 mg twice daily) were initiated. Cardiac biomarkers were negative and after 7 days the ECG abnormalities (horizontal ST segment depression and T-wave inversion in inferior and lateral leads) have disappeared (Figure 4). An exercise testing ECG performed

at 2 weeks after admission was negative, pretest probability according with current recommendation being low (14%). Although the patient showed a low probability for coronary artery disease she underwent a MDCT coronary angiography which has shown patent coronary arteries without any atherosclerotic lesion. The Agatston score on MDCT coronary angiography was 0, equivalent with low risk of coronary disease. The patient had a right coronary dominance, with all coronary arteries permeable (Figure 5), without any atherosclerotic lesion or significant stenosis.

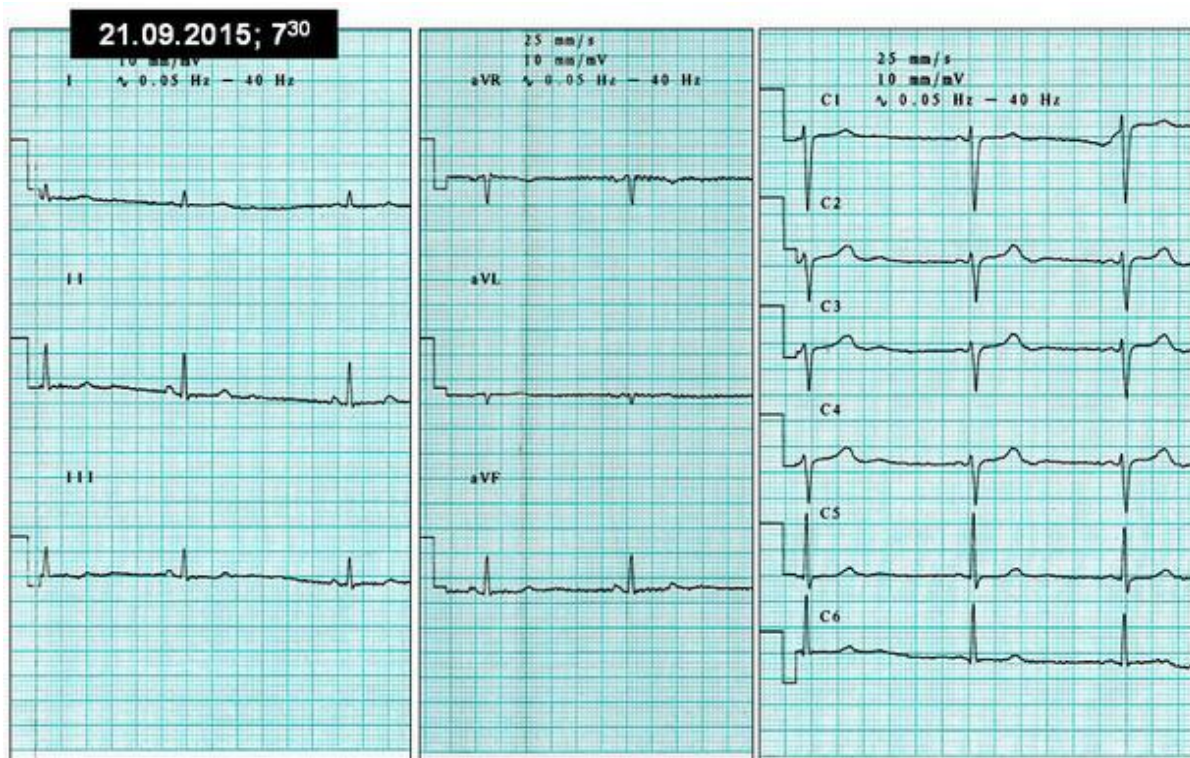


Fig. 4. Electrocardiogram of the patient at 1 week after admission.

The patient was discharged without any further treatment and we recommended, without any success, an investigation regarding the etiology of anemia at gastroenterology and gynecology departments. Four months later the patient was asymptomatic and the ECG was unchanged.

The final diagnosis in this case was MVP associated with moderate mitral regurgitation, isolated ectopic supraventricular and ventricular beats, grade I chronic iron deficiency anemia and mild hypocalcaemia.

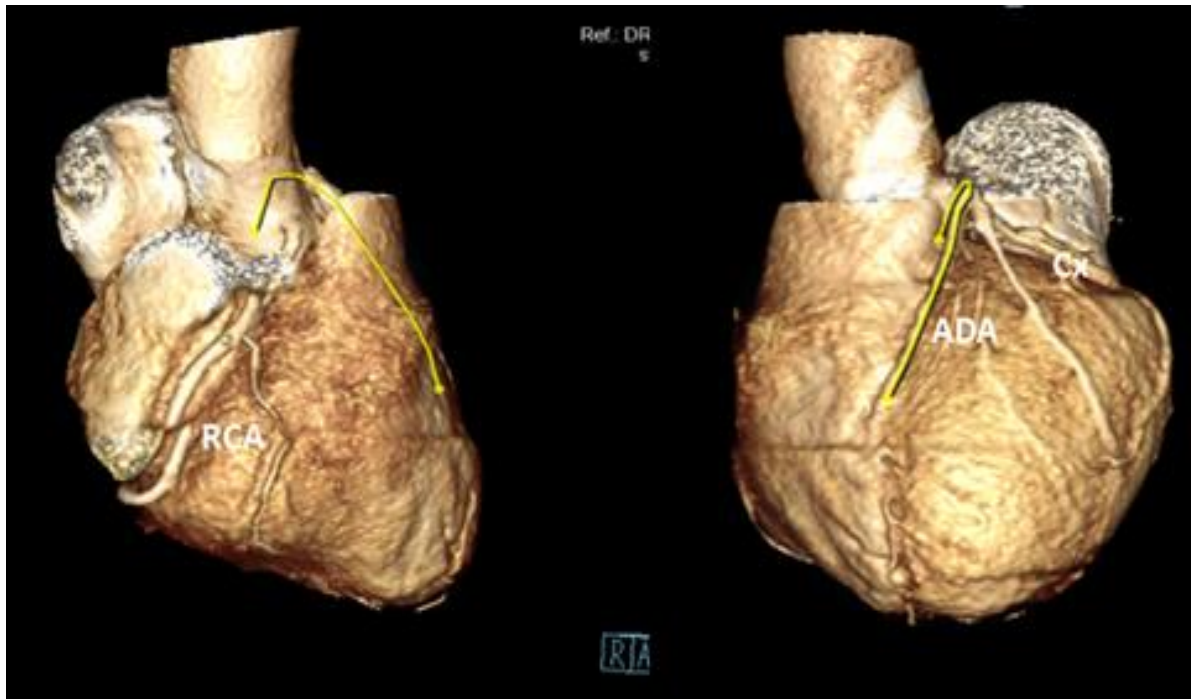


Fig. 5. MDCT coronary angiography has shown that all coronary arteries were permeable (left image: RCA – right coronary artery; right image: ADA-anterior descending artery and Cx - circumflex artery).

Discussion

The patient is a middle-aged, relatively healthy woman, known with chronic iron deficiency anemia and MVP. She didn't follow any chronic treatment at home and she did not presented any cardiovascular risk factors. The chest pain described by the patient is atypical for angina pectoris, but what raises our suspicion of diagnosis is the ECG which reveals ST-T changes, which according to the guidelines, can be interpreted as unstable angina.

Differential diagnosis in this case has to be performed with acute coronary syndrome and ionic disorders like hypocalcemia and hypomagnesaemia. Cardiac biomarkers and echocardiography were against an acute coronary syndrome. Other counterarguments were the absence of coronary risk factors, and the atypical chest pain. Also, in young patient coronary spasm related to hypocalcemia is one of the mechanisms involved in the chest pain occurrence [6]. The main changes induced by hypocalcaemia are QT interval prolongation, diffuse negative T waves and ST segment elevation. For our patient, ECG

changes were not suggestive for hypocalcaemia. However, it is unusual that a mild hypocalcaemia can induce symptoms and ECG changes only in inferior leads [7].

Another ionic disorder like hypomagnesaemia might be incriminated, being well known that hypomagnesaemia is frequent in women and present in more than 85% of cases with MVP; in addition, hypomagnesaemia is associated with chest pain and palpitations.

One month after admission, a MDCT coronary angiography was performed; this has an established role in the management of patients with an uncertain diagnosis of coronary heart disease [8]. Although no significant stenosis could be found, a possible coronary microcirculation abnormality should not be ruled out. This aspect can be taken into consideration in the case of patients with typical chest pain, with changes on the ECG or stress tests which all suggest myocardial ischemia, but without any hemodynamic significantly coronary artery stenosis. It is known that the microcirculation dysfunction occurs within 5 to 10 years earlier in women than in men.

The authors consider that the final diagnosis for our patient is MVP mimicking an acute coronary syndrome.

MVP has nonspecific symptoms like palpitations, atypical chest pain, anxiety or phobia [9, 11]. Our patient had atypical chest pain and palpitations. Palpitations are the expression of ventricular ectopic beats or supraventricular arrhythmias [3, 9]. Chest pain in these patients is variable described: usually located in the retrosternal area, prolonged, uncorrelated with exertion and it can rarely mimic pectoris angina. Chest pain causes are not known. There are different theories which involve excessive stress on papillary muscle with consecutive ischemia and dysfunction of adjacent ventricular myocardium [10].

ECG changes in patients with MVP are ST segment depression and T wave inversion in inferior leads, as in our patient's case. The possible mechanisms involved in ECG changes are papillary muscles ischemia, vasospasm and microvascular perfusion defects [11]. In our case these mechanisms might be triggered by the anemia, hypocalcaemia, or undetectable hypomagnesaemia. Stress test at exertion is frequently associated with false positive results, especially in women with normal coronary artery.

In most cases, MVP evolution is benign. Complications rate is 2% per year, with similar life duration as in normal individuals. Several long-term prognostic studies suggest that complications occur most commonly in patients with a mitral systolic murmur, those with thickened redundant mitral valve leaflets, and those with increased left ventricular or left atrial size, especially in men older than 45 years. The most important complication is severe mitral regurgitation due to the myxomatous degeneration or chordae rupture. Other complications related to MVP are: sudden cardiac death, arrhythmias, pulmonary hypertension, infectious endocarditis, cerebrovascular embolism. Supraventricular and ventricular arrhythmias are related to MVP complications. Ventricular arrhythmias occur in 34% of patients with MVP, ventricular ectopic beats being most frequently encountered (66%). Ionic disorders are trigger factors for ventricular arrhythmias and sudden cardiac

death. For our patient, ventricular ectopic beats were triggered by hypocalcaemia and anemia. Sudden death is a rare complication of MVP occurring in 2% of known cases during long-term follow-up (366–373), with annual mortality rates of 1% per year. It is probably due to the malignant arrhythmias and spontaneously primary chordae rupture. Predictive risk factors for sudden cardiac death are significant mitral regurgitation, redundant chordae and reduced left ventricular systolic function and cusps thickness [11, 12]. Our patient has low risk for severe complications, because she has none of above mentioned predictive risk factors.

Asymptomatic patients with MVP and mild mitral regurgitation should be monitored every three years. High risk patients should be followed annually [9]. Short term prognosis for this patient could be influenced by the ventricular and supraventricular arrhythmia especially in the presence of spasmophilia and anemia, therefore these abnormalities should be corrected.

On long term, it is mandatory to follow the evolution of the mitral valve regurgitation and its consequences over the left atrium, left ventricular function, and over pulmonary hypertension occurrence. On long term, our patient should be clinically and ECG Holter monitored annually and every three years by echocardiography examination. Symptoms occurrence could change patient's follow-up.

Surgical treatment is recommended in symptomatic patients with MVP associated with severe mitral regurgitation or asymptomatic patients but with left ventricular dilatation (end-diastolic diameter >45 mm) or reduced left ventricular ejection fraction. Severe mitral regurgitation associated with atrial fibrillation and pulmonary hypertension has class II indication for surgical therapy [11]. Our patient has no indication for surgical treatment.

Conclusion

In conclusion, we must note that MVP can mimic an acute coronary syndrome. Between these two pathologies, the differentiation can be made with the help of clinical information,

ECG, echocardiography and cardiac biomarkers. Sometimes, another investigation like MDCT coronary angiography is necessary for a correct diagnosis. An appropriate diagnosis and treatment can be lifesaving in these situations. This case highlights that ECG interpretation could sometimes provide unexpected surprises. Therefore, an integrative approach is needed, with the correlation of all clinical and laboratory

diagnosis elements, in order to manage successfully diagnosis process.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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