Original Article

# The Many Faces of Arrhythmic Mitral Valve Prolapse: Case Series

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## Abstract

**Background:** Although it is frequently a benign condition, mitral valve prolapse (MVP) may be associated with an increased risk of ventricular arrhythmias (VA), a condition known as arrhythmic mitral valve prolapse (AMVP).

**Objectives:** To present diverse manifestations of AMVP by means of clinical cases that illustrate these patients' symptoms and electrocardiogram (ECG), 24-hour Holter, transthoracic echocardiogram (TTE), and cardiac magnetic resonance imaging (CMRI) findings and to discuss the decisions made in relation to different clinical outcomes.

**Methods:** This retrospective, descriptive, and observational study analyzed 5 patients with AMVP treated between 2019 and 2024, investigating markers of elevated risk for severe clinical outcomes, especially sudden cardiac death (SCD).

**Results:** Among the 5 patients evaluated, the most common clinical presentation was palpitations (100% of cases), followed by syncope (40%). Three patients (60%) had severe or very severe arrhythmia on 24-hour Holter monitoring, while 4 (80%) had mitral annular disjunction (MAD). Positive delayed enhancement was observed in 2 (40%) patients. In 2 cases (40%), implantable cardioverter-defibrillator (ICD) was indicated. In 1 case, mitral valve replacement was chosen, but the arrhythmias were not resolved; consequently, radiofrequency ablation was indicated. Although SCD has been described in the literature, no cases were observed in our sample.

**Conclusion:** AMVP may present distinct clinical manifestations, including severe outcomes. It is essential to identify risk markers for early diagnosis and treatment of this condition, with the goal of reducing SCD-related mortality in these patients.

**Keywords:** Mitral Valve Prolapse; Cardiac Arrhythmias; Cardiac Sudden Death; Ventricular Tachycardia.

## Introduction

Mitral valve prolapse (MVP) affects approximately 2% to 3% of the general population and is considered the most common valvular heart disease, especially in Western countries, with a slight predominance in the female sex.[1] Although the outcomes of MVP are mostly benign, there is growing evidence of a direct relationship with an increased risk of sudden cardiac death (SCD).[2,3] This condition is known as arrhythmic mitral valve prolapse (AMPV), and it is defined by the combination of MVP with frequent or complex ventricular arrhythmias (VA), in the absence of any other arrhythmic substrate.[4]

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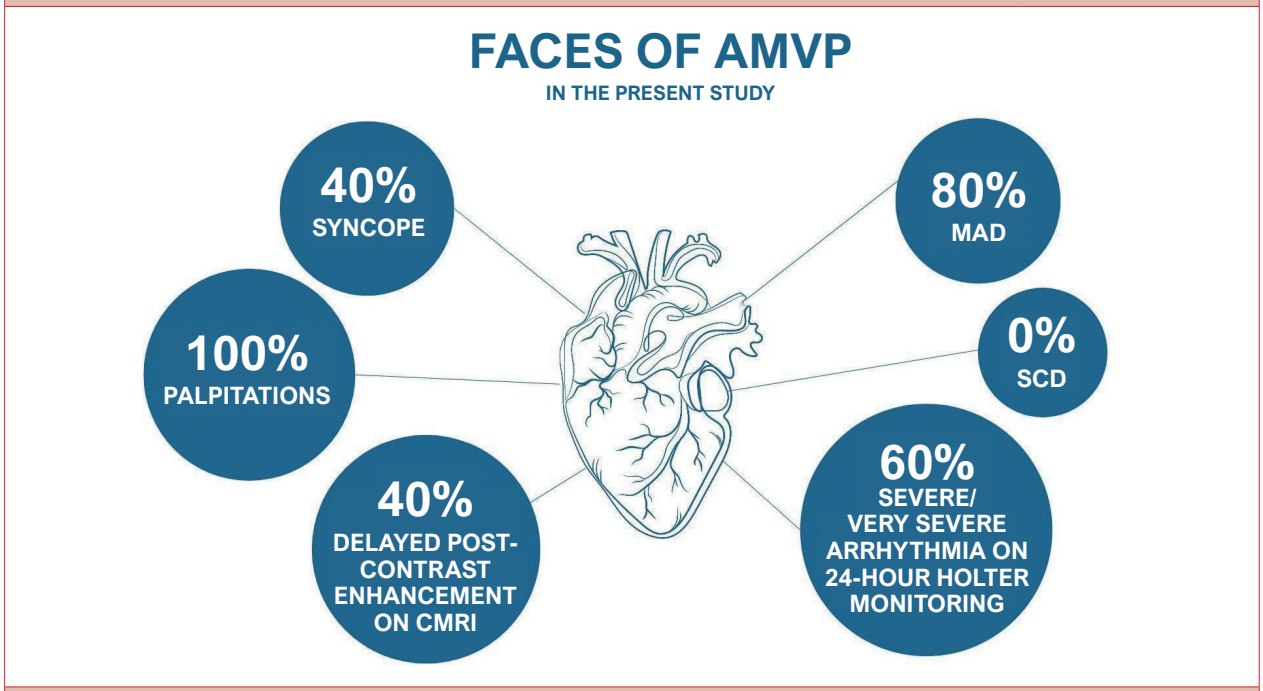
The fact that AMPV is known to be a heterogeneous condition makes it increasingly relevant to discuss, especially in the search for more robust data on its pathophysiology, risk stratification, and appropriate management.[4] In this scenario, studies have focused on determining the high-risk characteristics for the outcome of SCD, using clinical history and abnormalities on routine exams, for example, electrocardiogram (ECG), 24-hour Holter monitoring, transthoracic echocardiogram (TTE), and cardiac magnetic resonance imaging (CMRI).[1,5-7]

We present a series of 5 cases that illustrate the diverse clinical presentation of AMVP, discussing diagnostic and therapeutic challenges and reinforcing the importance of individualized management of these patients.

## Methodology

The objective of this observational, documentary, retrospective, and descriptive study was to portray the multiple possible presentations of AMVP. The study followed the recommendations of the Case Reports Guidelines (CARE).

**Central Illustration: The Many Faces of Arrhythmic Mitral Valve Prolapse: Case Series**

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*Source: the authors (2024). AMVP: arrhythmic mitral valve prolapse; CMRI: cardiac magnetic resonance imaging; MAD: mitral annular disjunction; SCD: sudden cardiac death.*

Analyses were conducted during the first half of 2024. The inclusion criteria involved adults over 18 years of age, from the state of Paraíba, Brazil, with AMVP. We evaluated 5 patients treated between September 2019 and March 2024. Data were collected by means of analysis of electronic medical records for 2 patients and the application of a questionnaire in a virtual environment for 3 patients, totaling 5 cases studied.

Data collection included medical history; electronic records of consultations; results of exams such as including ECG, CMRI, 24-hour Holter, coronary tomography angiography, TTE, and electrophysiological study; and patient management. All ethical aspects were strictly followed. A free and informed consent form was obtained, in accordance with the norms established by the Research Ethics Council and the Brazilian National Research Ethics Commission, and the study received approval from the Unipê Ethics and Research Committee (CAAE 78186124.9.0000.5176).

MVP was defined as a systolic displacement of the mitral leaflet into the left atrium (LA) of at least 2 mm from the mitral annular plane, on echocardiographic examination.[8] AMVP was defined by the presence of MVP (with or without mitral annular disjunction [MAD]), combined with frequent and/or complex VA in the absence of any other well-defined arrhythmic substrate.[4] Complex VA were defined as the presence of multifocal ventricular extrasystoles, paired ventricular extrasystoles, nonsustained ventricular tachycardia (NSVT), or R wave over T wave.[1] MAD was defined as a systolic separation between the ventricular myocardium and the mitral annulus supporting the posterior mitral leaflet.[9] For this study, a minimum cutoff value of 1 mm was considered to define the presence of MAD on CMRI.[7] CMRI was conducted in all 5 study participants, and it is the exam with the greatest accuracy in diagnosing this finding.

## Results

**Case 1:** A 42-year-old female patient complained of daily palpitations, without associated symptoms. She denied comorbidities or medication use. An ECG was performed, which showed sinus rhythm and inverted T waves in the inferior leads (Figure 1). A 24-hour Holter monitor identified 28% polymorphic VA, 58 of which were NSVT. At that time, her TTE showed MVP with 16-mm MAD, preserved ejection fraction, and slight enlargement of the LA. After initiating use of beta blockers, there was a reduction in VA density to 5% on a subsequent 24-hour Holter monitor. An exercise test was requested, which showed increased VA at peak exertion; coronary tomography angiography showed no significant lesions. CMRI confirmed important 15-mm MAD, but with an ejection fraction of 47% and delayed post-contrast enhancement in the lower portion of the mitral annulus, indicating fibrosis (Figure 2). Since the patient had multiple high-risk characteristics for SCD, electrophysiological study was requested for risk stratification; ventricular fibrillation was induced after the second cycle of extra-stimuli (Figure 3),

and the decision was made to implant an implantable cardioverter-defibrillator (ICD). After 1 year of follow-up, the patient continued without shocks from the device, taking beta blockers and amiodarone.

**Case 2:** A 63-year-old female patient complained of fatigue upon minimal effort, palpitations, and sporadic syncope without prodromes. The patient was hypertensive and was on losartan 50 mg, bisoprolol 5 mg, rosuvastatin 20 mg, escitalopram 10 mg, and clonazepam 0.5 mg. Her resting ECG was within normal limits, and the exercise test showed no abnormalities. Her TTE showed important LA dilation, moderate left ventricular dilation, and mitral valve with 8-mm MAD associated with myxomatous degeneration and leaflet prolapse. Based on these findings, a CMRI was requested, which revealed MAD and mitral annular fibrosis, demonstrated by the presence of delayed contrast enhancement in the mitral annular region near the P1 and P2 segments, in addition to mild mitral reflux and moderate LA enlargement. Her 24-hour Holter showed 1% polymorphic VA. In view of the condition with unexplained syncope, AMVP, delayed enhancement, and LA enlargement, the decision was made to implant an ICD.

**Case 3:** A 39-year-old female patient complained of palpitations and syncope episodes, dyspnea upon mild exertion, drowsiness, paroxysmal nocturnal dyspnea, and orthopnea. On physical examination, she had a holosystolic murmur in the mitral focus, grade 3+/6+. Her ECG showed sinus rhythm with ventricular bigeminy and extrasystole with left bundle branch block morphology, transition in V4 and inferior axis. TTE showed important mitral insufficiency, in addition to posterior mitral cusp prolapse. CMRI showed biatrial enlargement with ejection fraction of 68% and absence of delayed enhancement. The 24-hour Holter revealed 31% polymorphic VA, with a predominance of one of the morphologies, 70 being NSVT. Therefore, drug treatment with beta blockers and amiodarone was initiated, and a transesophageal echocardiogram was requested, which reclassified the mitral insufficiency as severe. Notwithstanding the reduction in VA density to

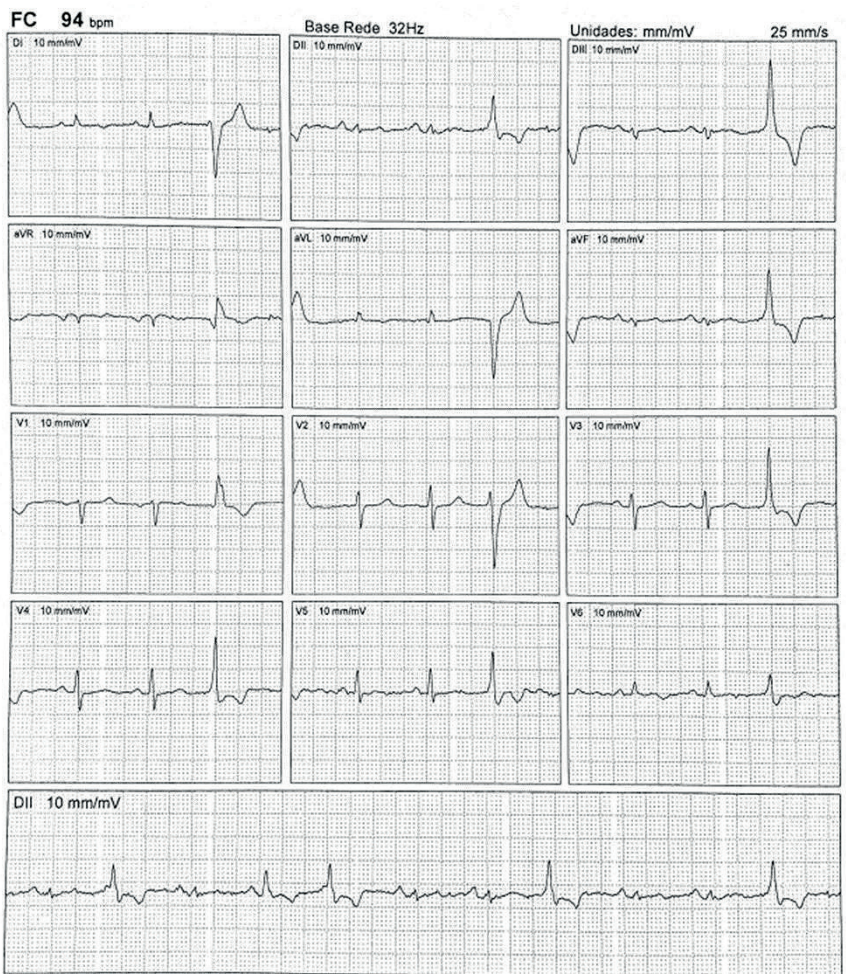


**Figure 1 –** *ECG showing sinus rhythm, T wave inversion in D1 and AVL, and monomorphic ventricular extrasystole with right bundle branch block morphology and possible location in the anterior mitral annulus. Source: the authors (2024). FC: Heart rate.*

14% on subsequent 24-hour Holter monitoring, the patient developed an episode of syncope without prodromes, and surgical mitral valve replacement with a mechanical prosthesis was indicated. In spite of this, the patient persisted with high VA burden, and radiofrequency ablation was performed in the arrhythmogenic focus, located in the anteroseptal portion of the right ventricular outflow tract. After 1 year and 7 months of follow-up, the patient remained asymptomatic.

**Case 4:** A 70-year-old female patient presented with complaints of palpitations and fatigue upon moderate exertion, which had begun approximately 10 years prior. She denied syncope and other associated symptoms. She

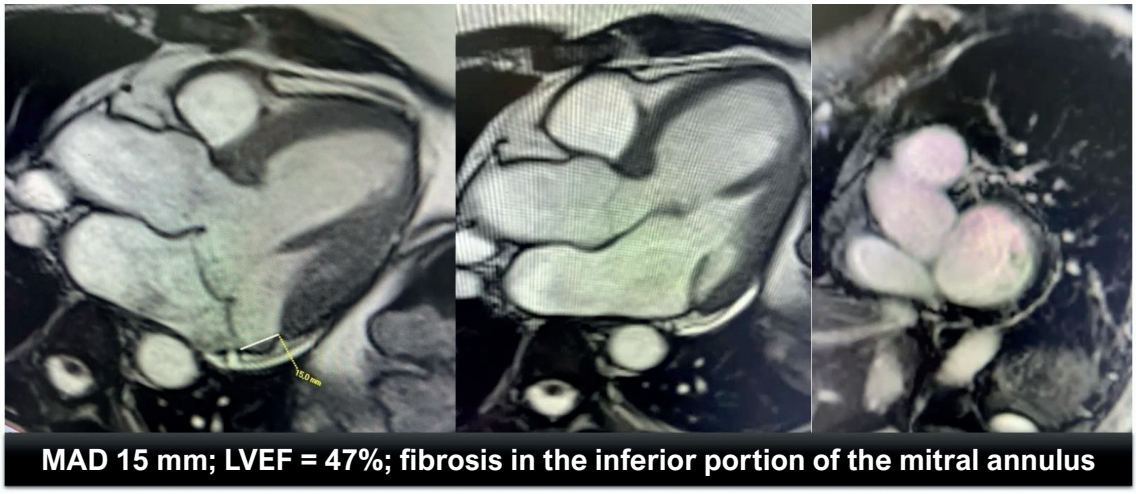


**Figure 2 –** *CMRI showing 15-mm MAD and fibrosis in the inferior portion of the mitral annulus. Source: the authors (2024). LVEF: left ventricular ejection fraction; MAD: mitral annular disjunction.*

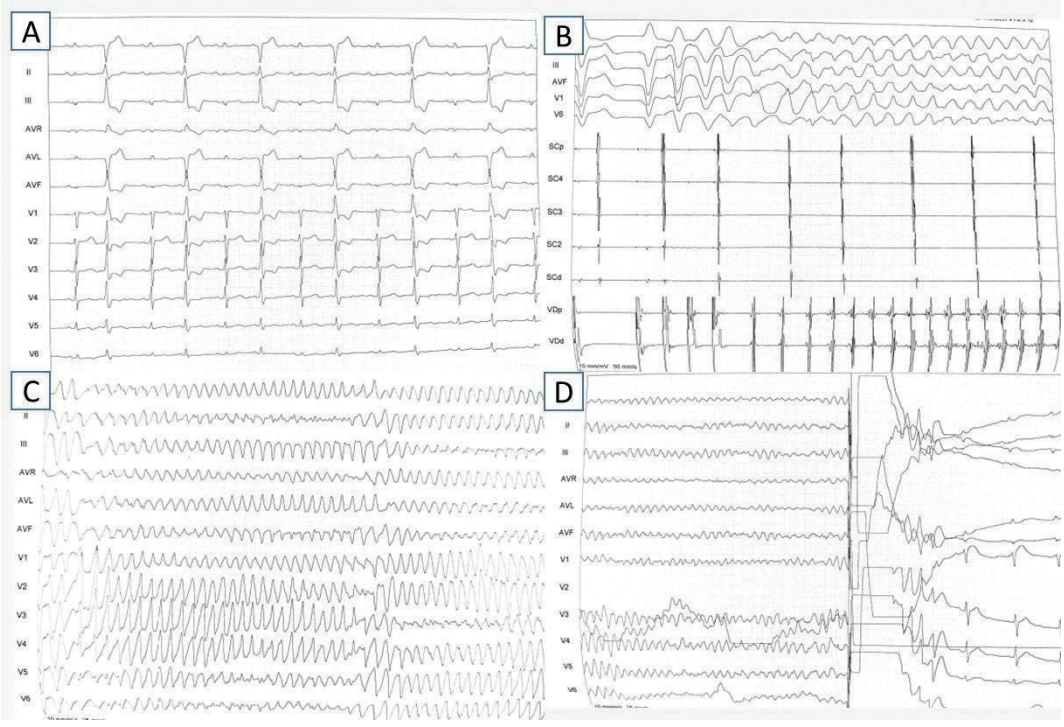


**Figure 3 –** *Induced ventricular fibrillation on electrophysiological study.*

had been diagnosed with systemic arterial hypertension, glaucoma, prediabetes, and takotsubo syndrome; she was taking acetylsalicylic acid 100 mg/day, rosuvastatin 5 mg/day, olmesartan 20 mg/day, metoprolol 25 mg twice a day, and metformin 500 mg 3 times daily. Her 24-hour Holter revealed 21% polymorphic VA, but without NSVT; her TTE identified the presence of mild mitral reflux. A CMRI was requested, which revealed myxomatous degeneration of the mitral valve, anterior leaflet prolapse, and mild MAD (< 5 mm) in the inferior and inferolateral portion, with minimal regurgitation, in addition to tricuspid valve with myxomatous degeneration, septal leaflet prolapse, and mild reflux. Radiofrequency ablation of the arrhythmogenic focus was indicated, but no arrhythmias were present during the procedure; thus, the decision was made to begin optimized drug treatment with amiodarone 100 mg/day and metoprolol 50 mg twice a day. After 7 months of follow-up, the patient remained asymptomatic.

**Case 5:** A 65-year-old male patient presented symptoms of palpitations and dyspnea for 3 months. He denied syncope. He had been diagnosed with systemic arterial hypertension and dyslipidemia and was taking tamsulosin 0.4 mg/day and rosuvastatin 20 mg/day. His ECG revealed sinus rhythm with end conduction delay. His 24-hour Holter showed 3% polymorphic VA, without NSVT. TTE showed a mitral valve with mildly thickened cusps and posterior leaflet prolapse. CMRI, in turn, demonstrated mild mitral insufficiency, with a 5-mm MAD. Bisoprolol 5 mg/day was prescribed, resulting in a significant reduction in arrhythmic density and control of symptoms.

## Discussion

This study is one of the first case series published in Brazil to portray the vast myriad presentations of AMVP, highlighting different risks of SCD and the difficulty in adopting scientifically-based conducts due to the low prevalence and scarce literature on the condition. It is worth noting that all participants in this study were treated in the private health system, which to a certain extent facilitated management of these patients, given that it was necessary to perform high-cost exams and procedures.

History of fainting or syncope, abnormalities on Holter monitoring (polymorphic VA), ECG (T wave inversion and prolonged QT interval), TTE (bileaflet prolapse, myxomatous degeneration, and MAD), and CMRI (delayed gadolinium enhancement in the annular region and papillary muscles, indicating fibrosis) (Figure 4), are considered high-risk markers for SCD in patients with AMVP.[4]

The patient described in case 1 did not have significant clinical symptoms; however, she exhibited abnormalities on ECG, TTE, and CMRI suggestive of a high risk for the development of SCD. Given these findings, electrophysiological study was indicated, which culminated in the implantation of an ICD.

Performing electrophysiological study to stratify the risk of SCD in cases of AMVP is still controversial. A meta-analysis carried out in patients with AMVP observed that this exam can be useful in mapping VA in patients with some risk markers, such as female sex, T wave inversion on ECG, polymorphic ventricular extrasystoles, MAD and double leaflet MVP on TTE, and delayed enhancement in the inferior left ventricular wall and papillary muscle on CMRI,[10] which were present in this patient.

With respect to ICD, it has been shown to be the indicated therapy for patients with AMVP at high risk of SCD, as was the case of patients 1 and 2. In the case of the latter, there was a clinical history of unexplained syncope, suggestive of cardiogenic etiology, which, in conjunction with the high-risk markers (syncope, bileaflet prolapse, MAD, myxomatous degeneration, and delayed enhancement in the mitral annular region), led us to decide not to request electrophysiological study, but rather to promptly indicate ICD implantation. The use of ICD as primary prevention should be considered in patients with AMVP if it is associated with high-risk phenotypes for ventricular tachycardia.[4]

In case 3, the patient presented MVP with important mitral insufficiency, in addition to complex VA, with frequent NSVT and syncope of cardiogenic etiology. Although beta blockers and amiodarone reduced the frequency of VA, the symptoms initially persisted. The patient underwent mitral valve replacement with a mechanical prosthesis in an attempt to correct the mitral insufficiency and reduce the arrhythmic burden; however, the patient persisted with high VA density, requiring radiofrequency ablation. Regarding this therapeutic choice, studies have provided evidence of its ability to reduce the burden of ventricular ectopy, prevent ICD shocks, and improve systolic function, and it is routinely indicated for cases of frequent symptomatic ventricular ectopy, sustained ventricular fibrillation/tachycardia, and/or cases refractory to antiarrhythmic drug therapy, provided that the arrhythmogenic focus has been correctly delineated.[5]

In case 4, the patient complained of palpitation, and examination showed MVP associated with MAD; in this case, drug treatment was initially chosen. Data are relatively limited regarding the efficacy of antiarrhythmic drugs for VA specifically related to MVP. Nonetheless, due to their relative safety, the use of these medications continues to be the first line of treatment for cases of AMVP.[6] In the absence of expected control, new possible forms of management have been considered, such as radiofrequency ablation. As described in case 3, the patient was indicated for the procedure, but the absence of VA induction during electrophysiological study meant that radiofrequency ablation was not effective, and we decided to optimize drug treatment to control the condition.

The patient reported in case 5 did not present significant clinical findings; however, he reported the presence of palpitation, the most common presentation of the arrhythmic syndrome of MAD.[7] Regarding the control of VA, the patient in question showed a significant reduction in arrhythmic density and symptom control with the use of bisoprolol, although the suppression of ventricular extrasystoles by means of therapy with antiarrhythmic drugs in a small study was not effective.[6]

This study was able to identify various faces of AMVP, for example, the presence of patients between 39 and 70 years old, especially women, who had varied clinical findings such as dyspnea, syncope, and palpitations. Radiological findings were also diverse among patients, but some were repeated more frequently, such as structural changes in the mitral area, including MAD, myxomatous degeneration, and fibrosis (Figure 5).

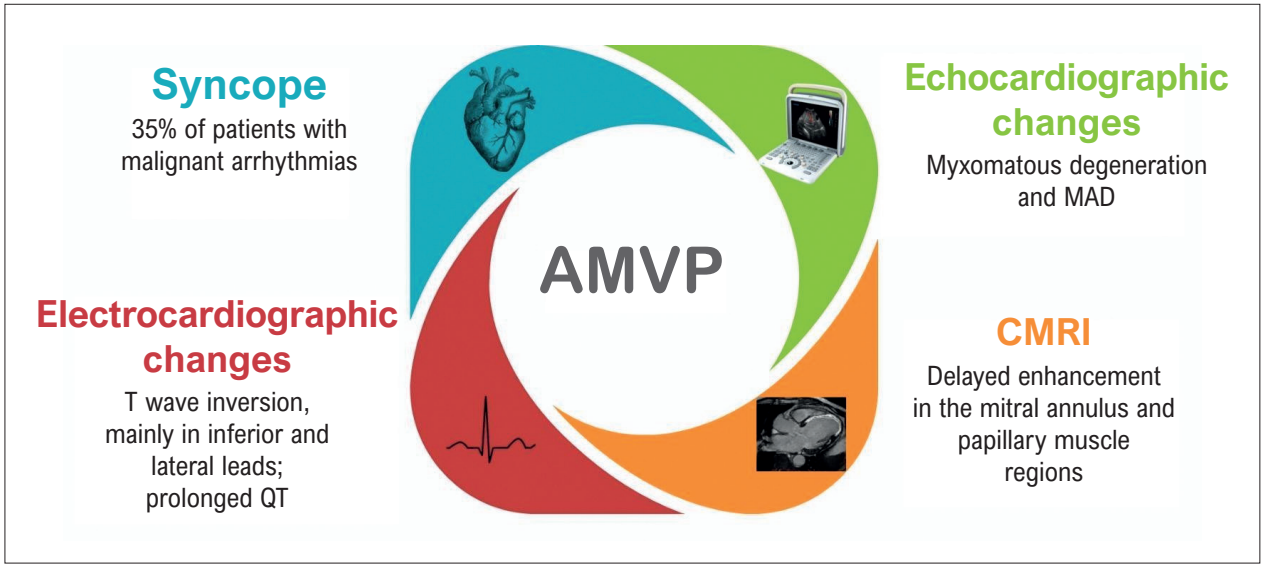


**Figure 4 –** *Markers of high risk for SCD. Source: the authors (2024). AMVP: arrhythmic mitral valve prolapse; CMRI: cardiac magnetic resonance imaging; MAD: mitral annular disjunction.*

It is worth underscoring that the importance and, at the same time, the challenge of risk stratification in MVP is in identifying high-risk patients within a large population of low-risk patients.[11]

In relation to MAD, echocardiography studies indicate a minimum limit of 5 mm.[12] However, on CMRI, the length of MAD can vary from 1 mm to over 15 mm.[7,13] This variability in length makes diagnosis challenging, since the lack of a clear diagnostic criterion can lead to overdiagnosis, posing challenges to clinical assessment and appropriate patient management. Nonetheless, although the exact required length remains unclear, research has shown that disjunctions smaller than 5 mm are generally considered incidental findings, while disjunctions greater than 8.5 mm have been associated with more unfavorable prognosis.[14,15]

Our study has some limitations. The variability of clinical manifestations and responses to proposed treatments in a small sample makes it difficult to find patterns that could serve as recommendations for other patients with AMVP. The small number of patients included may also favor biases that limit the extrapolation of some of the findings, requiring future studies with larger samples. Data collection performed in medical records makes the study vulnerable to recording errors and omission of information, while collection performed by applying a questionnaire may introduce recall bias. Moreover, the study may not reflect disease progression and the long-term effects of treatments, since the analyses were performed in the first half of 2024, and patient follow-up is still ongoing.

## Conclusion

AMVP can manifest with diverse clinical presentations, including unfavorable outcomes such as SCD. In clinical practice, this diagnosis can often be challenging, requiring extensive clinical assessment and high-cost diagnostic tests. Furthermore, the management and treatment of this condition have yet to be well established in the literature, requiring further, more comprehensive studies. In this context, the identification of risk markers, such as MAD and the presence of VA, is essential for the early diagnosis and treatment of these patients.

## Author Contributions

Conception and design of the research, analysis and interpretation of the data and writing of the manuscript: Barbosa YEL, Evangelista IWQF, Sousa JMR, Jales LCL, Viegas ELM, Athayde GAT; acquisition of data: Barbosa YEL, Evangelista IWQF, Sousa JMR, Jales LCL, Viegas ELM, Moura DMC, Pereira RAR, Athayde GAT; statistical analysis: Barbosa YEL, Athayde GAT; critical revision of the manuscript for intellectual content: Moura DMC, Targueta GP, Resende LA, Pereira RAR, Athayde GAT.

### Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

### Sources of Funding

There were no external funding sources for this study.

### Study Association

This study is not associated with any thesis or dissertation work.

### Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Centro Universitário de João Pessoa (UNIPÊ) under the protocol number 78186124.9.0000.5176. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

| | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|---|---|---|---|---|---|
| **Sex and age** | Female, 42 | Female, 63 | Female, 39 | Female, 70 | Male, 65 |
| **Symptoms** | Palpitations | Dyspnea, palpitations, syncope | Dyspnea, palpitations, syncope, drowsiness, orthopnea, paroxysmal nocturnal | Palpitations and dyspnea | Palpitations and dyspnea |
| **ECG** | T wave inversion and ventricular extrasystoles | Normal | Ventricular bigeminy and extrasystole with LBBB | — | End conduction delay |
| **24-hour Holter** | 28% VA | 1% VA | 31% VA | 21% VA | 3% VA |
| **TTE** | MVP, enlarged LA | MVP, MI | MVP, MI | Mild mitral reflux | MVP and thickened posterior cusp |
| **CMRI** | MAD 15 mm, MA fibrosis | MAD 8 mm, MA fibrosis | Biatrial enlargement | MVP, MAD < 5 mm, MV myxomatous degeneration | MAD 5 mm, MI |

**Figure 5 –** *Various faces of AMVP in the present study. Source: the authors (2024). CMRI: cardiac magnetic resonance imaging; ECG: electrocardiogram; LA: left atrium; LBBB: left bundle branch block; MA: mitral annulus; MAD: mitral annular disjunction; MI: mitral insufficiency; MV: mitral valve; MVP: mitral valve prolapse; TTE: transthoracic echocardiogram; TV: tricuspid valve; TVP: tricuspid valve prolapse; VA: ventricular arrhythmia.*

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