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Clinical Predictors of Ventricular Arrhythmias in Mitral Valve Prolapse

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ABSTRACT

Mitral valve prolapse (MVP), which affects approximately 2–3% of the population, is often benign but may be associated with a higher incidence of ventricular arrhythmias (VA) and sudden cardiac death (SCD). This study aimed to highlight the incidental detection of MVP among SCD cases and to investigate the associated histopathological features. In addition, we investigated the potential risk of VA in MVP patients by assessing heart rate variability (HRV) and heart rate turbulence (HRT). In 2021, two parallel investigations were conducted: a pathological examination of 225 individuals who experienced SCD, and a clinical evaluation of 50 MVP patients using 24-hour Holter monitoring. Among the SCD cases, MVP-like mitral valve changes were identified in eight subjects, along with fibrotic changes near the valve. The clinical study group was stratified into two categories based on age, clinical characteristics, and echocardiographic findings: group A: 23 individuals under 40 years with signs of myxomatous mitral valve degeneration; group B: 27 older adults with MVP and additional cardiovascular conditions. Holter monitoring in MVP patients revealed changes in HRV and HRT, which were frequently associated with the degree of mitral regurgitation (MR). These autonomic indicators may provide insight into the elevated risk of VA among this group of patients.

Keywords: Sudden cardiac death, Mitral valve prolapse, Myxomatous degeneration, Heart rate variability, Ventricular arrhythmia, Heart rate turbulence

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Introduction

Mitral valve prolapse (MVP) is recognized as a relatively frequent valvular abnormality, with an estimated prevalence of 2–3% in the general population [1–3]. It arises due to fibro-myxomatous degeneration, which leads to the thickening and elongation of one or both mitral valve (MV) leaflets. This structural alteration causes the leaflets to prolapse into the left atrium (LA) during systole, resulting in incomplete closure of the mitral valve and potential regurgitation [1]. MVP may occur as an isolated condition or in association with connective tissue disorders (CTDs) or other cardiovascular (CV) diseases [4–7]. A familial tendency is observed in 40–50% of cases, suggesting a genetic predisposition [1, 4, 5].

Clinically, MVP is classified into two major forms. The primary or non-syndromic variant occurs in the absence of identifiable connective tissue abnormalities, while the secondary or syndromic form is associated with systemic CTDs such as Marfan syndrome, Loeys-Dietz syndrome, or Ehlers-Danlos syndrome [1, 8]. Two-dimensional (2D) echocardiography remains the primary diagnostic tool for MVP, with the condition being confirmed by observing the asymmetric systolic displacement of the MV leaflets above the annular plane, typically described as a buckling motion [9].

Although MVP is typically benign, a subset of patients may develop complications, including ventricular arrhythmias (VA), severe mitral regurgitation (MR), heart failure, or even sudden cardiac death (SCD) [4, 10, 11]. The likelihood of developing VA can be assessed using 24-hour Holter monitoring, along with analysis of heart

rate variability (HRV) and heart rate turbulence (HRT), which provide insight into autonomic nervous system (ANS) function [4, 5, 12]. These measures have been extensively used in cardiovascular research to evaluate autonomic balance and to predict adverse outcomes in patients with heart disease [12–14]. A disruption in the balance between sympathetic and parasympathetic activity may increase the risk of VA and SCD [5, 14, 15].
The present study was conducted to assess the increased arrhythmic risk in MVP patients by evaluating HRV and HRT via 24-hour Holter monitoring. An additional aim was to identify morpho-pathological changes indicative of MVP in individuals who died from SCD [16–18].

Materials and Methods

In the morphopathological arm of the study, 225 cases of sudden cardiac death (SCD) investigated during 2021 at the Forensic Department of our County were examined. Among these, 29 individuals were younger than 40 years old, while 196 were between 40 and 75 years old. Anatomopathological features suggestive of MVP were identified in three cases from the younger group and in five from the older group. The mitral valve leaflets were analyzed for structural abnormalities, and particular attention was given to the presence of fibrous plaques located on the postero-lateral wall of the left ventricle (LV). Tissue samples were collected from the leaflets, papillary muscles, and the adjacent endocardial surface, fixed in formalin, sectioned at 5 μm thickness, and stained with hematoxylin and eosin. All histological slides were reviewed by a trained pathologist [17–20].
For the clinical component, fifty patients previously diagnosed with MVP through 2D echocardiography were enrolled and classified into two distinct groups based on age, echocardiographic features, and clinical presentation. The first group consisted of 23 patients, including 10 men and 13 women aged between 18 and 39 years. These individuals presented MVP affecting one or both leaflets, accompanied by mitral regurgitation of varying severity. None had a confirmed diagnosis of Marfan syndrome. The second group included 27 patients, 14 men and 13 women, aged between 42 and 74 years, most of whom exhibited posterior leaflet involvement and concurrent mitral regurgitation.
All patients underwent a comprehensive cardiological evaluation that included a detailed physical examination, 12-lead electrocardiography (ECG), and 2D echocardiographic assessment. All ultrasound examinations were carried out by the same experienced cardiologist. MVP diagnosis was confirmed by demonstrating a displacement of more than 2 mm of one or both mitral valve leaflets into the LA during systole, along with leaflet thickening greater than 5 mm during diastole, as visualized in the parasternal long-axis and apical four-chamber views [9]. In addition to confirming MVP, the echocardiographic study also included measurement of left ventricular mass index (LVMI), left atrial volume index (LAVI), and ejection fraction (EF), calculated using the Simpson biplane method. Doppler echocardiography was used to evaluate the degree of mitral regurgitation [9].

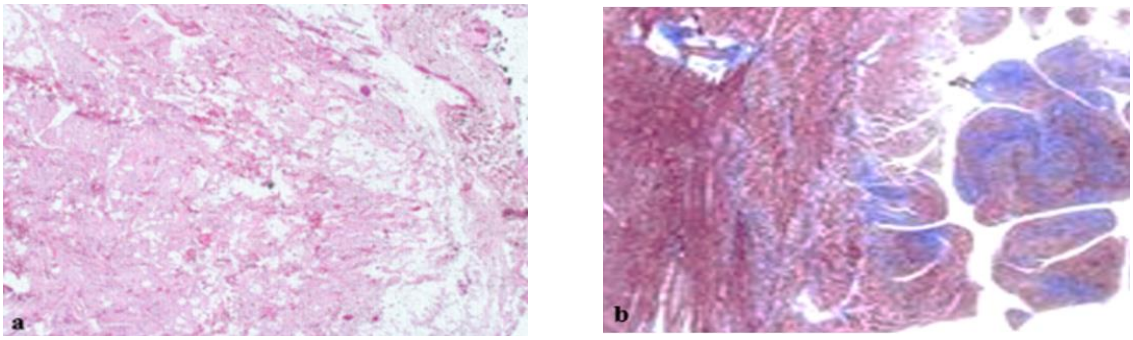
Holter Monitoring and Statistical Analysis

All participants underwent 24-hour ambulatory ECG monitoring using the Labtech Cardiospy Holter system. The presence and severity of ventricular arrhythmias (VA) were evaluated during this period. Analysis of heart rate variability (HRV) data was performed using the Nevrokard Long-Term aHRV software (version 5.0.0). HRV, which reflects spontaneous oscillations in heart rate and regular RR intervals, was assessed in the time domain by calculating the standard deviation of all normal-to-normal (NN) intervals (SDNN). Based on established thresholds, SDNN values below 50 ms were classified as indicative of poor health status, those between 50 and 100 ms as representing compromised health, and values exceeding 100 ms as reflecting healthy autonomic regulation [14].
To analyze heart rate turbulence (HRT), only patients with a minimum of six premature ventricular contractions (PVCs) were included. HRT evaluates fluctuations in sinus cycle length following isolated PVCs. Two parameters were measured: turbulence onset (TO), reflecting early sinus acceleration, and turbulence slope (TS), which corresponds to late sinus deceleration. In the literature, a TO value $< 0\%$ and a TS > 2.5 ms/RR interval are generally considered normal. According to risk stratification criteria, HRT findings were categorized as follows: Category 0 indicated normal TO and TS, category 1 indicated abnormality in either TO or TS, and category 2 indicated abnormalities in both parameters [14, 15].
For statistical analysis, SPSS software (version 25.0, IBM, Chicago, IL, USA) was employed. Continuous variables were expressed as mean ± standard deviation (SD) or as median with interquartile range (IQR), while categorical variables were summarized as counts and percentages. Given that the Shapiro-Wilk test demonstrated

a non-normal distribution of data, nonparametric statistical methods were used for further analysis. Comparisons of continuous variables between groups were carried out using the Mann–Whitney U test. The correlation between mitral regurgitation (MR) severity and HRT parameters was examined using Spearman's rank correlation coefficient. A P-value < 0.05 was considered statistically significant.
This study was approved by the Ethics Committee of our hospital (approval no. 4052/19.06.2020), and all enrolled patients provided written informed consent.

Results and Discussion

Morphopathological Observations
During 2021, a total of 225 sudden cardiac death (SCD) cases were evaluated at the Forensic Department of our County. Among the 29 deceased individuals under 40 years of age, and the 196 individuals aged between 40 and 75, MVP-related macroscopic changes of the mitral valve were identified in three and five cases, respectively. In the younger group, the majority showed posterior leaflet involvement (66%), while the remaining displayed bileaflet degeneration (33%). Leaflets appeared thickened with elongated chordae tendineae, consistent with myxomatous degeneration. In the older SCD victims, bileaflet MVP was present in 40% of cases, and isolated posterior leaflet prolapse in 60%. In all examined hearts, leaflet thickening, fibrotic elongation of chordae, and fibrous plaques along the adjacent posterolateral ventricular wall were observed.
Histopathological analysis revealed myxomatous infiltration with mucopolysaccharide deposition in the valve tissue of younger individuals (**Figure 1a**). In contrast, fibroelastic changes were more prominent in older patients. Disruption of collagen and fragmentation of elastic fibers were seen in both groups. Additionally, in six of the eight MVP cases (75%), focal endo-myocardial fibrotic changes were observed in the posterior leaflet region, neighboring ventricular wall, and at the summit of the papillary muscle. Hypertrophic cardiomyocytes with dysmorphic and irregularly shaped nuclei were also noted in these areas (**Figure 1b**).



a) b)

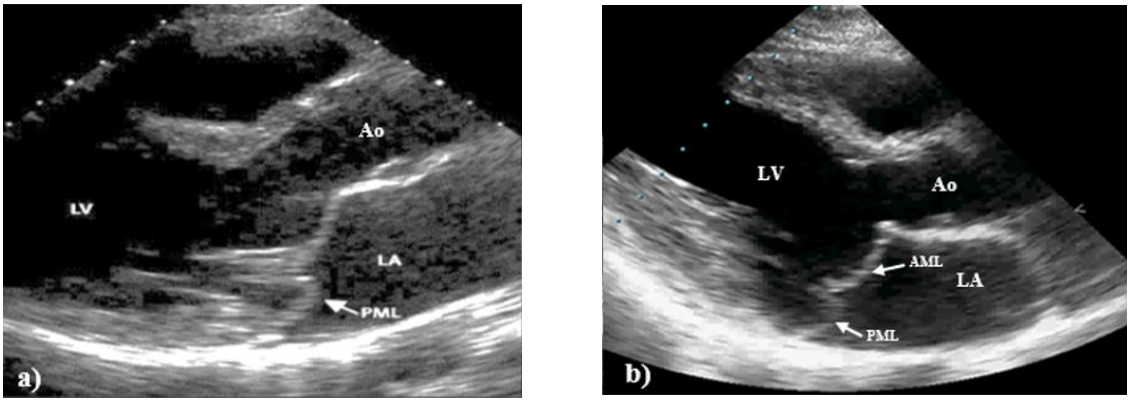
Figure 1. Histopathological features of mitral valve prolapse (MVP) demonstrating myxoid changes in the mitral valve (a), and focal fibrosis in the posterolateral left ventricular wall and papillary muscles (b).

Table 1. Results of clinical data, 2D echocardiography, and 24-hour Holter monitoring

Clinical and laboratory data	Group A (n = 23)	Group B (n = 27)	*P*-value
Gender: men/women[a]	10/13	14/13	0.555
Age[b]	27 (21-34)	61 (52-68)	< 0.001
Echocardiography:			
MVP of anterior leaflet[a]	2	3	0.951
MVP of the posterior leaflet[a]	12	15	0.811
MVP of both leaflets[a]	9	9	0.770
MR: Mild/Moderate/ Severe[a]	14/8/1	9/13/5	0.098
Hypo/a/dyskinesia[a]	-	21/6/4	-
LAVI[b]	33.2 (32.9-34.9)	37 (35.9-38)	< 0.001
LVMI[b]	93 (92-112)	99.2 (95.6-117)	< 0.001
EF (Simpson)[b]	65 (62-69)	51 (49-52)	< 0.001
24-hour ECG Holter monitoring:			
Mean HR[b]	76 (75-78)	63 (62-65)	< 0.001
PVC–isolated[a]	23	27	1

Systematized[a]	8	13	0.167
Unsustained/sustained VT[a]	1	6	0.199
SDNN[b]	107 (102-109)	97 (52-102)	< 0.001
HRT Category 0[a]	20	7	< 0.001
Category 1[a]	2	11	0.011
Category 2[a]	1	9	0.013

Note: BMI: body mass index; MVP: mitral valve prolapse; MR: mitral regurgitation; LAVI: left atrial volume index; LVMI: left ventricular mass index; EF: ejection fraction; HR: heart rate; PVC: premature ventricular contractions; VT: ventricular tachycardia; SDNN: standard deviation of normal-normal RR intervals; HRT: heart rate turbulence.



a) b)

Figure 2. a) 2D echocardiography of posterior leaflet prolapse, and b) bileaflet prolapse; LA = left atrium, LV = left ventricle, Ao = aorta, PML = posterior mitral leaflet, and AML = anterior mitral leaflet

Clinical Study

Group A consisted of 23 patients (10 males and 13 females), with a median age of 27 years (range: 21–34 years), who were diagnosed with MVP based on 2D echocardiographic evaluation (**Figure 2**). Their diagnostic and laboratory characteristics are summarized in **Table 1**. Among these individuals, posterior leaflet prolapse was the most commonly observed presentation (52.17%), followed by prolapse of both leaflets (**Figures 2a** and **2b**). Most patients had mild mitral regurgitation (MR), recorded in over 60% of the group. Within this group, 20 patients exhibited SDNN values greater than 100 ms and were categorized under HRT category 0. These cases were primarily associated with mild MR; only 6 patients displayed moderate MR. Two patients had SDNN values in the intermediate range (50–100 ms) and belonged to HRT category 1; both had moderate MR. One individual, with severe MR resulting from bileaflet prolapse, showed a markedly reduced SDNN (< 50 ms) and was placed in HRT category 2. A moderate, statistically significant correlation was found between MR severity and HRT category (Spearman r = 0.553; P = 0.006).

Group B was composed of 27 patients (14 males and 13 females), with a median age of 61 years (range: 52–68 years), also diagnosed with MVP. The majority had coexisting cardiovascular conditions (22 out of 27). Similar to group A, prolapse of the posterior mitral leaflet was predominant, followed by bileaflet prolapse. MR was notably more severe in this group, with two-thirds of patients exhibiting moderate to severe MR. Compared to group A, group B had significantly elevated left atrial volume index (LAVI) and left ventricular mass index (LVMI), along with a lower ejection fraction (EF), all showing a P-value < 0.001 (see **Table 1**).

Within group B, 9 patients maintained normal SDNN values; among them, 4 individuals (2 of whom had moderate MR) were classified under HRT category 1. Twelve patients had intermediate SDNN values (50–100 ms), with HRT categories distributed as follows: 2 in category 0, 7 in category 1, and 3 in category 2. In this subset, 2 had mild MR, 9 had moderate, and 1 had severe MR. The remaining 6 patients exhibited SDNN values < 50 ms and belonged to HRT category 2; 4 of these had severe MR. A strong positive correlation was observed between MR severity and HRT classification (r = 0.777; P = 0.001), as per Spearman's analysis.

Parallel to the clinical investigation, autopsy findings from 225 individuals who died from sudden cardiac death (SCD) in 2021 were reviewed at the County's Forensic Institute. In 8 cases (3.55%), MVP was identified. Microscopic evaluation revealed age-related histological differences: in younger individuals, prominent myxomatous degeneration was primarily located in the middle scallop, resembling classic Barlow's pathology,

whereas older individuals showed fibroelastic remodeling with chordal elongation or rupture and localized fibrotic plaques.

These outcomes are consistent with previous research indicating that mitral valves removed during surgery in MVP patients with advanced MR tend to show an expanded valve area, dilated annular structures, and elongated or ruptured chordae tendineae [1, 5]. Modern classification distinguishes Barlow-type MVP (leaflets thicker than 5 mm) from fibroelastic deficiency (with segmental thickening less than 5 mm). However, it remains uncertain whether these represent different genetic entities or variations within a shared pathological spectrum.

Our histopathological findings mirrored traditional observations, showing myxoid infiltration with mucopolysaccharide buildup, breakdown of collagen, and fragmentation of elastin in the mitral valve and associated chordae [1]. Additional pathological features—such as fibrosis of the valve leaflets and localized endocardial damage—could be linked to the development of ventricular arrhythmias (VA) [21, 22]. It is hypothesized that the repeated mechanical stress on papillary muscles from prolapsing mitral leaflets may lead to structural changes that increase the likelihood of arrhythmias [5, 21, 22]. Although increased arrhythmogenic activity has been reported in MVP patients with papillary muscle scarring on Holter monitoring, the broader clinical implications are still being explored [21, 23].

This clinical evaluation included 50 patients diagnosed with MVP confirmed via 2D echocardiography [9]. Prolapse of the posterior leaflet was most common, with bileaflet involvement occurring less frequently. Anterior leaflet prolapse was rare. All patients had MR of varying severity.

Diagnosing mitral valve prolapse (MVP) through echocardiography remains challenging. MVP manifests in two primary forms: the classic type, frequently found in younger individuals and linked to myxomatous changes in the valve leaflets, and a second type, commonly observed in older adults, often accompanied by coronary artery disease and worsening mitral regurgitation (MR). When MVP is detected solely in the apical 4-chamber view, it may be misleading—this is frequently due to the complex, saddle-shaped three-dimensional configuration of the mitral valve (MV) annulus, which includes several scallops [9]. The integration of 3D echocardiography into clinical workflows has greatly improved diagnostic accuracy by allowing detailed visualization of the MV's structure and providing clearer identification of each scallop.

In terms of heart rate variability (HRV) and heart rate turbulence (HRT) analysis within our study groups, group A included only a few patients with abnormal findings: two with moderate MR exhibited mildly reduced SDNN, while one patient with severe MR had significantly decreased SDNN and was classified in HRT category 2. In contrast, pathological SDNN values were identified in two-thirds (66.66%) of group B participants. Moreover, HRT abnormalities were present in 92.59% of group B cases, particularly among those with moderate or severe MR. Statistical analysis demonstrated a robust correlation between HRT parameters and MR severity in this group ($r = 0.777$; $P < 0.001$). When considering all patients collectively, a strong overall relationship between MR severity and HRT was confirmed ($r = 0.715$; $P = 0.001$). These findings support the hypothesis that MVP is associated with an autonomic nervous system (ANS) imbalance, marked by elevated sympathetic nervous activity [5, 6].

It's worth noting that this investigation was carried out during the COVID-19 pandemic. Although we intended to primarily include patients diagnosed with MVP before 2020, some participants may have contracted SARS-CoV-2. The virus is known to have widespread systemic effects [24, 25], including disruptions to the ANS, potentially affecting both HRV and HRT.

Alterations in HRV and HRT among MVP patients—particularly those with MR—remain a topic of ongoing research. Numerous studies have reported decreased parasympathetic tone and heightened sympathetic activity in such individuals, resulting in reduced HRV [14, 15]. Turcher Y emphasized a correlation between increasing MR severity and elevated arrhythmic risk [26]. Conversely, Van der Wall did not observe significant associations between HRV, HRT, and the incidence of ventricular arrhythmias (VA) [12]. Nevertheless, several other investigations have documented notable HRT alterations in MVP patients when compared to healthy controls [5, 10, 27–30], likely reflecting ANS dysfunction rather than direct hemodynamic consequences. In our study, the pronounced HRT changes in group B may be attributable to the older age of participants, the presence of ischemic heart disease, and reduced left ventricular ejection fraction (EF).

Although MVP is not typically considered a highly disabling condition, particularly in younger individuals, the frequent occurrence of arrhythmias and the looming threat of sudden cardiac events can negatively affect patients' psychological well-being. Many patients experience significant anxiety or depressive symptoms, often warranting psychiatric consultation [31, 32].

Conclusion

In MVP patients, the tendency to develop ventricular arrhythmias appears to be closely linked to disturbances in autonomic balance, particularly involving heightened sympathetic activity. This risk can be evaluated through HRV and HRT analysis, which correlate with the severity of MR. Furthermore, the progression of fibrotic changes near the mitral valve may also play a role in arrhythmogenesis.

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Ethics Statement: None

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