



# EFFECTIVENESS OF MEDICAL TREATMENT FOR IDIOPATHIC FREQUENT PREMATURE VENTRICULAR COMPLEXES

İDYOPATİK SIK PREMATÜRE VENTRİKÜLER KOMPLEKSLERİN MEDİKAL TEDAVİSİNİN ETKİNLİĞİ



KEREM CAN YILMAZ

Medical Park Hospital, Bursa, Turkey

## ABSTRACT

**Introduction:** Premature ventricular complexes (PVCs) are common arrhythmias in the clinical practice with an estimated prevalence of 1 to 4% in the general population. Also catheter ablation is recommended for first line treatment in symptomatic idiopathic ventricular tachycardia but medical treatment is reasonable. The aim of this study is to demonstrate the success rate of combined medical therapy and the factors influencing it in patients presenting with frequent idiopathic premature ventricular complexes.

**Method:** We retrospectively analyzed 60 adult patients who visited the cardiology outpatient clinic between 2022 and 2025. We diagnosed idiopathic PVC via electrocardiography at hospital admission and underwent ambulatory 24-hour rhythm Holter monitoring. Patients were initially divided into two groups based on their response to drug therapy. The demographic data, PVC burden and counts on Holter monitoring, treatment duration, administered medications, and ECG characteristics of PVCs were compared between patients who responded to drug therapy and those who did not.

**Results:** There were two groups; Group 1 was drug-unresponsive group (n=32), Group 2 (n=28) was drug-responsive group. After first treatment period we changed and proceed additional medical therapy in drug-unresponsive group. There was significantly positive response to treatment in patients with a transition at V4 in PVCs. We showed that PVC count and burden was significantly higher then in the drug-responsive group, 3 of 32 patients had ablation after first treatment. In the second treatment group (n=29), more than an 80% reduction in PVC burden was observed in 14 of the 29 patients. Overall, 42 out of 60 patients (70%) showed a response to medical therapy.

**Conclusion:** As a result, combination therapy may be given to patients who are not considering interventional treatment for first option or who has not possibility to reach early for ablation.

**Keywords:** premature ventricular complex; medical treatment; antiarrhythmic drugs

## ÖZET

**Giriş:** Prematür ventriküler kompleksler (PVC'ler), genel popülasyonda yaklaşık %1 ila %4 arasında görülen yaygın aritmilerdir. Semptomatik idiyopatik ventriküler taşikardi hastalarında birinci basamak tedavi olarak kateter ablasyonu önerilmektedir, ancak medikal tedavi de makul bir seçenektir. Bu çalışmanın amacı, sık idiyopatik prematür ventriküler komplekslerle başvuran hastalarda kombine medikal tedavinin başarı oranını ve başarıyı etkileyen faktörleri göstermektir.

**Yöntem:** 2022 ile 2025 yılları arasında kardiyoloji polikliniğine başvuran 60 yetişkin hasta retrospektif olarak analiz edildi. İdiyopatik PVC tanısı, hastane kabulündeki elektrokardiyografi (EKG) ile konuldu ve hastalara 24 saatlik ritim holter monitörizasyonu yapıldı. Hastalar, ilaç tedavisine yanıtlarına göre başlangıçta iki gruba ayrıldı. Demografik veriler, Holter monitörizasyonundaki PVC yükü ve sayıları, tedavi süresi, uygulanan ilaçlar ve PVC'lerin EKG özellikleri, ilaç tedavisine yanıt veren ve vermeyen hastalar arasında karşılaştırıldı.

**Bulgular:** Çalışmadaki ilk iki grup; Grup 1, ilaçlara yanıt vermeyen grup (n=32), Grup 2 ise ilaçlara yanıt veren grup (n=28) olarak belirlendi. İlk tedavi döneminden sonra, ilaçlara yanıt vermeyen grupta tedavi değiştirildi ve ek medikal tedaviye devam edildi. PVC'lerde V4 geçişi olan hastalar, PVC sayısı ve yükü fazla olan hastalar ilaçlara yanıt veren grupta anlamlı olarak daha yüksek saptandı. İlk tedavi sonrası 32 hastanın 3'üne ablasyon uygulandı. İkinci tedavi grubundaki 29 hastanın 14'ünde PVC yükünde %80'den fazla azalma görüldü. Toplamda 60 hastanın 42'sinde (%70) medikal tedaviye yanıt olduğu görüldü.

**Sonuç:** Sonuç olarak, girişimsel tedaviyi ilk seçenek olarak düşünmeyen veya ablasyona erken ulaşma imkânı olmayan hastalarda kombine ilaç tedavisi ilk seçenek olarak uygulanabilir.

**Anahtar kelimeler:** prematür ventriküler kompleks; medikal tedavi; antiaritmik ilaçlar

## INTRODUCTION

Premature ventricular complex (PVC): Premature occurrence of an abnormal QRS complex (duration typically  $\geq 120$  ms, corresponding T-wave typically broad and in the opposite direction of the major QRS deflection, no preceding P-wave) (1). Premature ventricular complexes (PVCs) are common arrhythmias in the clinical practice with an estimated prevalence of 1 to 4% in the general population on standard 12-lead electrocardiography (ECG) and between 40 and 75% of subjects on 24- to 48-h Holter monitoring (2).

However, very frequent PVCs have been associated with potentially reversible PVC-induced left ventricular dysfunction, or can be a sign of subclinical heart disease (3,4).

PVCs/ventricular tachycardia (VT) in patients without structural heart disease (SHD) are defined as idiopathic. In patients with presumed idiopathic PVCs/VT based on a negative history and normal physical examination, 12-lead ECG and transthoracic echocardiography are important first diagnostic steps to exclude underlying SHD (1). They often

**Corresponding Author:** Kerem Can Yılmaz, Fevzi Cakmak Caddesi, Kiraali, No:72 Medical Park Bursa Hospital , Cardiology Department  
**E-mail:** keremcny@hotmail.com  
**ORCID:** 0000-0003-3320-9508

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**Table 1.** Comparison of baseline characteristics between first drug groups

|                                                | Group 1 (n=32) | Group 2 (n=28) | p value      |
|------------------------------------------------|----------------|----------------|--------------|
| <b>Sex category (male) (%)</b>                 | 14 (43)        | 11 (39)        | 0.726        |
| <b>Age (years)</b>                             | 42.5 ± 11.5    | 45.8 ± 12.4    | 0.276        |
| <b>Hypertension (%)</b>                        | 10 (31)        | 9 (32)         | 0.941        |
| <b>Diabetes Mellitus (%)</b>                   | 1 (3)          | 2 (7)          | 0.476        |
| <b>Smoking (%)</b>                             | 14 (43)        | 9 (32)         | 0.356        |
| <b>Ejection Fraction (%)</b>                   | 59.5 ± 3.3     | 57.2 ± 9.3     | 0.229        |
| <b>Symptom at admission (%)</b>                | 24 (75)        | 22 (78)        | 0.744        |
| <b>Mitral valve insufficiency (%)</b>          | 9 (28)         | 14 (50)        | 0.216        |
| <b>PVC count at 1st holter monitoring</b>      | 11650 ± 6520   | 16271 ± 8010   | <b>0.019</b> |
| <b>PVC burden (%) at 1st holter monitoring</b> | 12.2 ± 6.7     | 17.2 ± 8.7     | <b>0.018</b> |
| <b>Treatment time (days)</b>                   | 61.8 ± 23.4    | 73.3 ± 24.5    | 0.069        |

Abb. PVC; premature ventricular complex

originate from common anatomical locations such as the right or left ventricular outflow tracts (RVOT/LVOT), aortic root, atrioventricular annuli, papillary muscles, or the Purkinje system (5). Automaticity, triggered activity (early and delayed-afterdepolarizations, reentry, purkinje related focal activity and ion channel mechanisms are part of pathophysiological mechanisms of PVCs (6).

Also catheter ablation is recommended for first line treatment in symptomatic idiopathic ventricular tachycardia (VT)/PVCs from the right ventricle outflow tract (RVOT) or the left fascicles, beta-blockers or non-dihydropyridine calcium channel blockers (CCBs) are indicated in symptomatic patients for medical treatment and other anti-arrhythmic drugs such as flecainide when they fail (1). However, in our country, due to the difficulty in accessing ablation and the disadvantage of its cost, follow-up with medical therapy for a certain period may be prioritized.

The aim of this study is to demonstrate the success rate of combined medical therapy and the factors influencing it in patients presenting with frequent idiopathic premature ventricular complexes.

## METHODS

We retrospectively analyzed 60 adult patients who visited our cardiology outpatient clinic between 2022 and 2025. The study received institutional research ethics board approval from Mudanya University Ethical Committee (decision no: 2025-4/2, date: 30/07/2025). Patients provided informed consent.

PVCs/VT in patients without structural heart disease (SHD) are defined as idiopathic. First we diagnosed idiopathic PVC via electrocardiography (ECG) at hospital admission and underwent ambulatory 24-hour rhythm Holter monitoring. Patients were initially divided into two groups based on their response to drug therapy. The demographic data, PVC burden and counts on Holter monitoring, treatment duration, administered medications, and ECG characteristics of PVCs were compared between patients who responded to drug therapy and those who did not. Subsequently, the treatment of patients who did not respond to drug therapy was intensified, and the groups of patients who responded or did not respond to this enhanced therapy were designated as the third and fourth groups, respectively. Within these groups, demographic data, PVC burden and

**Table 2.** Comparison of transition leads in electrocardiogram between first drug groups

|               | Group 1 (n=32) | Group 2 (n=28) | p value      |
|---------------|----------------|----------------|--------------|
| <b>V1 (%)</b> | 7 (21)         | 1 (3)          | <b>0.033</b> |
| <b>V2 (%)</b> | 8 (25)         | 5 (17)         |              |
| <b>V3 (%)</b> | 10 (31)        | 6 (21)         |              |
| <b>V4 (%)</b> | 7 (21)         | 16 (57)        |              |

counts on the second Holter monitoring, ECG characteristics of PVCs, and the medications were compared.

Patients under 18 years old, prior history of PVC ablation, known coronary artery disease, heart failure, cardiac imaging demonstrating structural heart disease consisting of scar or wall motion abnormality, Brugada syndrome, arrhythmogenic right ventricular (RV) cardiomyopathy, or long QT syndrome were also excluded from the study. The demographic data of the patients, laboratory tests, echocardiography measurements, ambulatory rhythm Holter records, and ECG results were collected from the hospital database. Frequent idiopathic PVC was defined as ≥5% PVC on 24- hour ambulatory ECG (Holter) monitoring. The medical treatments received by the patients for idiopathic PVC were classified according to the Vaughan-Williams classification (7). Response to medical treatment was defined as ≥80% reduction in PVC burden with 24 hours of Holter monitoring at follow-up

The 12-lead ECG data were manually analyzed with regard to the normal beat and PVC. The PVC source was assessed using the 12-lead ECG recordings. The ECGs were taken at a speed of 25 mm/s, calibration of 10 mm/mV, and lowpass filter of 40 Hz. The origin of PVC was classified as transition zone from V1 to V4.

Statistics were conducted utilizing the Statistical Package for Social Sciences 21.0 for Windows (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to analyze the data. Continuous data with a normal distribution are expressed as the mean ± standard deviation, and categorical variables are expressed as number and percentage (%). Differences between categorical variables were determined by the chi-square test. Student's T-test or

**Table 3.** Comparison of medical treatment between first drug groups

|                                 | Group 1<br>(n=32) | Group 2<br>(n=28) | p<br>value |
|---------------------------------|-------------------|-------------------|------------|
| Metoprolol (%)                  | 15 (46)           | 10 (35)           | 0.236      |
| Metoprolol +<br>Propafenone (%) | 14 (43)           | 15 (53)           |            |
| Amiodarone (%)                  | 0                 | 1 (3)             |            |
| Nebivolol (%)                   | 1 (3)             | 0                 |            |
| Metoprolol +<br>Amiodarone (%)  | 0                 | 2 (6)             |            |
| Verapamile +<br>Propafenone (%) | 2 (6)             | 0                 |            |

the Mann-Whitney U test was utilized to compare continuous variables. Significance was assumed as  $p < 0.05$  value.

## RESULTS

From 2022 to 2025 60 patients involved to our study with idiopathic PVCs over 5% burden. There were two groups; Group 1 was drug-unresponsive group ( $n=32$ ), Group 2 ( $n=28$ ) was drug-responsive group. After first treatment period we changed and proceed additional medical therapy in drug-unresponsive group. Then we analyzed group after giving second drug treatment group 3 and 4 for drug responsivity and unresponsivity. We showed all demographic data for Group 1 and 2 before first treatment in Table 1. Mean age, sex category, other risk factors, smoking, symptoms at admission, echocardiographic findings, treatment time were not statistically different between two groups. But in holter monitoring we showed that PVC count and burden was significantly higher then in the drug-responsive group,  $11650 \pm 6520$ ,  $16271 \pm 8010$ ;  $p=0.019$ ,  $12.2 \pm 6.7$ ,  $17.2 \pm 8.7$ ;  $p=0.018$  respectively.

There was significantly positive response to treatment in patients with a transition at V4 in PVCs (Table 2). 16 of 28 patients (57%) had transition at V4 ( $p=0.033$ ). We had 8 patients with a transition at V1 and just one of them was drug-responsive. Metoprolol and metoprolol plus propafenone were given most of the patients for first line therapy. There was no difference between drug groups ( $p=0.236$ ) (Table 3).

**Table 4.** Comparison of baseline characteristics between second drug groups

|                                         | Group 3 (n=14)  | Group 4 (n=15)  | p<br>value |
|-----------------------------------------|-----------------|-----------------|------------|
| Sex category (male)                     | 5 (35)          | 8 (53)          | 0.340      |
| Age (years)                             | $39.8 \pm 11.7$ | $44.6 \pm 11.8$ | 0.282      |
| Hypertension (%)                        | 4 (28)          | 6 (40)          | 0.518      |
| Diabetes Mellitus (%)                   | 0               | 1 (6)           | 0.326      |
| Smoking (%)                             | 4 (28)          | 8 (53)          | 0.176      |
| Ejection Fraction (%)                   | $59.8 \pm 3.6$  | $57.1 \pm 3.5$  | 0.559      |
| Symptom at admission (%)                | 12 (85)         | 10 (66)         | 0.231      |
| Mitral valve insufficiency (%)          | 3 (21)          | 5 (33)          | 0.561      |
| PVC count at 2nd holter monitoring      | $8250 \pm 2640$ | $9646 \pm 4790$ | 0.337      |
| PVC burden (%) at 2nd holter monitoring | $8.4 \pm 2.7$   | $10.1 \pm 4.7$  | 0.268      |

Abb. PVC; premature ventricular complex

After first treatment period, 3 patients underwent ablation for PVCs in drug-unresponsive group. The rest of unresponsive patients ( $n=29$ ) recieved another drug combination. There were no statistically significant difference for sex category, mean age, hypertension, diabetes, smoking, ejection fraction in echocardiography, semptoms at admission, PVC count at 2nd holter monitoring and PVC burden at 2nd holter monitoring. PVC count and burden were  $8250 \pm 2640$  vs  $9646 \pm 4790$ ,  $p=0.337$ , and  $8.4 \pm 2.7$  vs  $10.1 \pm 4.7$ ,  $p=0.268$  respectively (Table 4). On the contrary to first treatment groups, there was no difference between transition of PVCs ( $p=0.352$ ) (Table 5). There were 4 drug groups; metoprolol plus propafenone, metoprolol plus amiodarone, verapamile plus amiodarone and verapamile plus propafenone, there were no significance between drug groups ( $p=0.498$ ) (Table 6).

## DISCUSSION

PVCs are one of the most prevalent arrhythmias in clinical practice. Patients with idiopathic PVCs mostly have a good prognosis. However, symptoms may lead to a decrease in the quality of life and trigger cardiomyopathy in some patients in relation to the PVC burden (8,9).

In our study, 25 of 60 patients were given metoprolol, 29 of 60 patients were recieved metoprolol plus propafenone treatment (Table 3). Metoprolol plus propafenone therapy was the most successful treatment. 15 of 28 treated patients were treated with this regimen. We gave 29 patients metoprolol plus propafenone and 15 of 29 patients (51%) we saw over 80% reduction in PVCs. For metoprolol this ratio was 40%, 10 of 25 patients. In another study, Tang et al (10) compared flecainide, amiodarone and sotalol with beta-blockers and CCBs. They found complete PVC supression in one-third of patients with beta blockers and CCBs. In our study as a difference we used beta-blockers and CCBs with amiodarone and propafenone. As it was compatible with the literature beta blockers were 40% success in supression of PVCs and with additional drug Class I anti-arrhythmic propafenone success percentage was increased to 51% at first treatment. After second treatment (combination therapy) we had only 15 patients left unresponsive to drugs. We succeed over 80% supression in totally 42 patients, 28 from first treatment, and 14 from second treatment, so the total ratio in patients were 42/60 , 70%. In another study using

**Table 5.** Comparison of transition leads in electrocardiogram between second drug groups

|        | Group 3 (n=14) | Group 4 (n=15) | p value |
|--------|----------------|----------------|---------|
| V1 (%) | 2 (14)         | 5 (33)         | 0.352   |
| V2 (%) | 5 (35)         | 3 (20)         |         |
| V3 (%) | 3 (21)         | 5 (33)         |         |
| V4 (%) | 4 (28)         | 2 (13)         |         |

flecainide that guideline recommended, Kojic et al (11) compared flecainide vs sotalol vs propafenone head-to-head. They found that flecainide was superior to other drugs with 55.6 % in total or near total suppression in PVCs and 64% over 80%. As a contrast to our study they found propafenone less effective than sotalol (11% vs 20%). Other trials only use one drug for suppression of PVCs, but unlike to other studies we used combination therapy and it seemed more effective than when they used alone. In our study flecainide was not used, due to difficulties in obtaining the drug in our country. But we showed same efficacy with combination and stepwise therapy with other drugs.

No significant differences were observed between groups 3 and 4 in terms of PVC count or burden, the administered drug classes, or PVC transition among patients who responded to treatment. This may also be attributed to the small sample size. Although verapamil plus propafenone or amiodarone combinations appeared to be more successful in these patients in percentage terms, the number of patients receiving these treatments was very small.

Undoubtedly, radiofrequency catheter ablation is more effective than drug therapy. It has 80 to 95% procedural success rate in a study depending on its origin (12,13). Although the success of ablation is evident, it has been demonstrated that drug therapy should not be disregarded due to factors such as limited access to three-dimensional mapping for all patients, the fact that ablation is performed only in certain centers, and associated costs. Unlike other studies, in our patient group, those with a PVC QRS transition at V4 showed a markedly higher response to drug therapy compared to other groups. In another study Atici et al (14) observed that PVCs with multiple morphologies responded better to treatment. In the same study they also reported that RVOT-originating PVCs showed better response to medical therapy although the difference was not statistically significant ( $p=0.088$ ) (14). This may be attributable to the small sample size or may suggest that combination therapy, administered in a manner not previously applied, could be more effective for RVOT extrasystoles. As demonstrated in some other studies, the greater the number of PVCs, the better the response to drug therapy. The similarly high ejection fraction values of the patients in our study do not allow for a meaningful comparison of EF.

## CONCLUSION

The distinction of our study from other studies was in demonstrating the effect of combination drug therapy and the first study to evaluate the response of patients who did not respond to initial treatment after a second therapy. As a result, combination therapy may be given to patients who are not considering interventional treatment for first option or who has not possibility to reach early for ablation.

**Table 6.** Comparison of medical treatment between second drug groups

| Drugs                        | Group 3 (n=14) | Group 4 (n=15) | p value |
|------------------------------|----------------|----------------|---------|
| Metoprolol + Propafenone (%) | 8 (57)         | 7 (46)         | 0.498   |
| Metoprolol + Amiodarone (%)  | 4 (28)         | 3 (20)         |         |
| Verapamil + Amiodarone (%)   | 2 (14)         | 3 (20)         |         |
| Verapamil + Propafenone (%)  | 0              | 2 (13)         |         |

The limitations of the study include the small sample size, the non-random selection of drug groups based on physician preference, and the generally high utilization of certain drugs in treatment.

**Ethics Committee Approval:** The study received institutional research ethics board approval from Mudanya University Ethical Committee (decision no: 2025-4/2, date: 30/07/2025).

**Informed Consent:** Patients provided informed consent.

**Authorship Contributions:** Concept – KCY.; Design – KCY.; Supervision – KCY.; Materials – KCY.; Data collection &/or processing – KCY.; Analysis and/or interpretation – KCY.; Literature search – KCY.; Writing – KCY.; Critical review – KCY.

**Conflict of Interest:** No conflict of interest was declared.

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