Heart Sci J 2022; 3(3): 41-44

Contents list available at www.heartscience.ub.ac.id

Heart Science Journal

Journal Homepage : www.heartscience.ub.ac.id

Case Report

Cardiomyopathy and Frequent Monomorphic Premature Ventricular Complex (PVC): Which One Comes First?

Anita Suryo Santoso[1*], Djanggan Sargowo[2], Ardian Rizal[2]

[1] *Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*
[2] *Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia.*

ARTICLE INFO

Keywords:
Cardiomyopathy;
Premature Ventricular Complex;
Arrhythmia-Induced Cardiomyopathy;
Ablation.

ABSTRACT

Background: Non-ischemic dilated cardiomyopathy (NIDCM) may have ventricular arrhythmias. it causes 8-50% of cardiac deaths and arrhythmias can induce cardiomyopathy (AIC). where atrial or ventricular tachyarrhythmias or frequent ventricular ectopy lead to left ventricular dysfunction, leading to systolic heart failure.
Case Presentation: A 28-year-old male was admitted to the hospital with a chief complaint of chest discomfort. There were symptoms of dyspnea on effort and palpitation before. He had a history of alcoholics for more than five years. The ECG showed premature ventricular complex (PVC) bigeminy and recommended to have ambulatory Holter monitoring with the conclusion of frequent monomorphic right ventricle outflow tract (RVOT) origin PVC. Echocardiography indicated a decrease in left ventricular (LV) function, LV dilatation, and global hypokinetic. Cardiac magnetic resonance imaging (CMR) was performed, and there was no "edema" and myocardium fibrosis.
Conclusion: It is essential to analyze which comes first. Arrhythmia-induced cardiomyopathy (AIC) or cardiomyopathy-induced arrhythmia to have immediate treatment. From the collected data, we conclude that the frequent RVOT origin PVC-induced cardiomyopathy and catheter ablation is the definitive therapy

1. Introduction

Ventricular arrhythmia commonly occurs in patients with non-ischemic dilated cardiomyopathy (NIDCM). The prevalence was 60–87% of patients.[1] Its incidence increases with left ventricular (LV) function deterioration. Moreover, it accounts for 8–50% of cardiac death in that subset of patients. Besides, arrhythmia can induce cardiomyopathy (AIC), in which atrial or ventricular tachyarrhythmia or frequent ventricular ectopy results in LV dysfunction, leading to systolic HF. Identifying, stabilizing, and correcting the causes of cardiomyopathy can improve outcomes and quality of life.[2] Here, we reported a case about a young male with chest discomfort and frequent monomorphic RVOT origin PVC.

2. Case Illustration

A 28-year-old male was admitted to the hospital with chest discomfort while the patient was resting after breakfast. It radiated all over the chest and neck and lasted for 10 minutes. He was initially diagnosed with the acute coronary syndrome (ACS) and then given loading aspilet and clopidogrel. He also complained about similar symptoms three days before hospital admission that occurred intermittently. It is also relieved by rest. This symptom did not bother or limit him from doing his daily activities; thus, he did not seek treatment before. The patient also complained of easy fatigue and sometimes palpitation since two months ago while doing moderate to heavy activities, relieved by rest. He routinely controlled to a cardiologist two months before. The medication were Aspilet 1x 80 mg, Ramipril 1x 5 mg, bisoprolol 1x 2,5 mg and simvastatin 1x20 mg. He has been an active smoker since 15 years ago, about two packs per day, and alcoholism four bottles per week in 10 years. He showed normal hemodynamic and physical examination. The electrocardiogram (ECG) was performed with results of sinus rhythm, normal frontal axis, counterclockwise rotation horizontal axis, and no ST-T changes. The cardiac enzyme was normal. Then he was treated as stable angina pectoris.

On the second day, the patient complained about chest discomfort and shortness of breath if walking more than 5 meters. The ECG showed multiple premature ventricular complexes (PVC). The patient performed ambulatory Holter monitoring for 24 hours, with results of frequent monomorphic PVC RVOT origin (46%) with periodic nonsustained ventricular tachycardia (VT). The echocardiography showed LV dilatation with eccentric hypertrophy, Ejection Fraction (EF) 47.6%, and hypokinetic segment apical (basal), anteroseptal-inferior-anterior (basal-mid). To confirm the diagnosis, cardiac magnetic resonance imaging (CMR) was performed with results: non-ischemic dilated cardiomyopathy with mitral valve regurgitation,

*Corresponding author at: Brawijaya Cardiovascular Research Center, Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Malang, Indonesia
E-mail address: anita.surya.santoso@gmail.com (A. S. Santoso).

https://doi.org/10.21776/ub.hsj.2022.003.03.8
Received 9 March 2022; Received in revised form 30 April 2022; Accepted 15 June 2022
Available online 30 July 2022

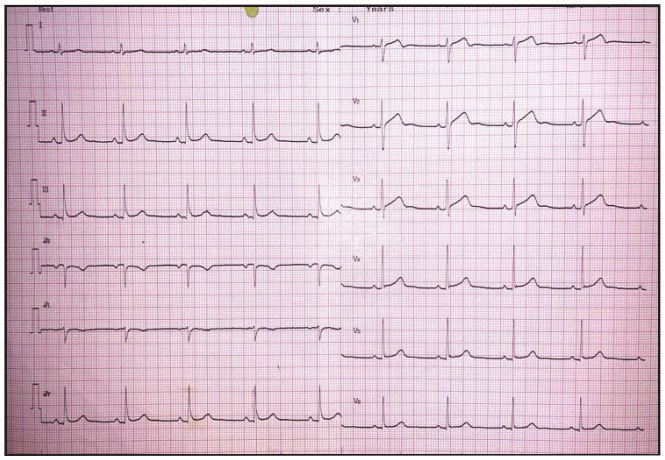

Figure 1. ECG in first-day hospitalization.

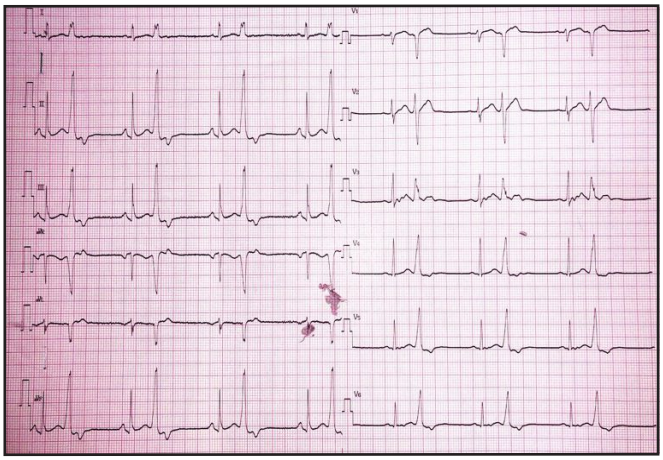

Figure 2. ECG on the second day of care in the ward.

global hypokinetic, a decrease of left ventricle function with EF 47%, without edema nor myocardial fibrosis. The diagnosis was changed to frequent PVC RVOT origin and heart failure (HF) stage C functional class II due to cardiomyopathy. The medications were stopped and were continued with ramipril 1x5mg and bisoprolol 2x 5 mg. The patient underwent a 2D catheter ablation in the next month, but there was difficulty in getting the source of PVC, so the procedure was ended. The patient was planned to have 3D catheter ablation.

3. Discussion

Cardiomyopathies are a heterogeneous group of myocardial diseases associated with mechanical or electrical dysfunction that usually exhibits inappropriate ventricular hypertrophy or dilatation.1 Dilated cardiomyopathy is defined by the presence of left ventricular dilatation and contractile dysfunction in the absence of abnormal loading conditions and severe coronary artery disease. It includes one of the most common causes of heart failure, with an estimated prevalence of 40 cases per 100000 individuals and an annual incidence of 7 cases per 100000 individuals.[1,2]

In this patient, there is suspicion of secondary etiology of cardiomyopathy by alcohol. Alcohol abuse accounts forG21–36% of dilated cardiomyopathy cases in high-income countries.[1,3] The relationship between alcohol intake and clinical heart failure is influenced by genetic, racial, and behavioral susceptibility factors. The diagnosis is based on a history of heavy alcohol intake (>80–100 g/day for more than five years), in combination with otherwise unexplained cardiomyopathy.[4]

Initial symptoms of heart failure exist in 80% of patients with dilated cardiomyopathy. These symptoms include orthopnea, ankle edema, fatigue after mild exertion, and excessive sweating.5 Sometimes, abdominal discomfort, nausea, anorexia, and cachexia symptoms can be prominent in advanced cases. Circulatory collapse is the most severe manifestation of congestive heart failure. Some individuals have palpitations and syncope. Thromboembolic events and rarely sudden death might be the initial symptom, particularly in infants. Physical symptoms can include peripheral and sacral edema, tachycardia, elevated jugular venous pressure, pulmonary crepitations, inferolateral displaced left ventricular apex beat, a gallop rhythm, and a mitral regurgitant murmur.[5,6]

A spectrum of phenotypes and other disorders associated with arrhythmic cardiomyopathy genes includes LMNA, SCN5A, FLNC, RBM20, TTN, DES, PLN, TMEM43, DSP, DSG2, and DSC2. Truncating variants in the TTN gene (TTNtv) have been identified in 15-20% of patients with DCM and have been proposed to be the most genetic cause of DCM. There is increasing evidence, however, that the phenotypic manifestations of TTNtv are likely to be modified by additional genetic and acquired factors, such as alcohol and pregnancy.[6]

Alcoholic cardiomyopathy is associated with atrial and ventricular arrhythmias. Some literature admitted that myocardial fibrosis has a vital role in inducing ventricular arrhythmias in patients with non-ischemic dilated cardiomyopathy (NIDCM), such as ACM.[7] The LV remodeling process experience by patients with NIDCM is characterized by changes in the extracellular matrix, including the formation of fibrotic tissue. It may favor the genesis of reentry mechanisms and increase the chance of developing malignant ventricular arrhythmias. Alcohol induces cardiac arrhythmias by causing electrolyte disorders, QT prolongation, adrenergic hyperactivity, decreased heart rate variability, and baroreceptor sensitivity.[6,7]

Outflow tract tachycardia occurs with equal frequency in men and women. The mean age at diagnosis is 50 ± 15 years (range, 6 to 80 years). A benign course is observed in most patients, suggesting that the underlying pathophysiologic process is not progressive and that the tachycardia does not represent an early manifestation of occult cardiomyopathy.[7] These findings have also been confirmed during the long-term follow-up of patients with frequent root ectopic beats. In a small subset of patients with incessant and repetitive monomorphic RVOT tachycardia, secondary cardiomyopathy can develop. Also, those with frequent uniform ventricular ectopy, PVC-induced cardiomyopathy can develop. Typically, the PVCs comprise between 5% and 40% of all beats in affected persons and can originate from either the RVOT or the LVOT.[7,8] Ablation of the PVC focus reverses the cardiomyopathy and restores normal left ventricular function within 3 to 6 months. Therefore, not all outflow tract PVCs are benign. Accordingly, patients diagnosed with “idiopathic cardiomyopathy” should undergo ambulatory monitoring to assess their degree of PVC burden. In those with frequent uniform ectopy, ablation may be warranted. Ablation of the arrhythmogenic focus appears to eliminate recurrent episodes of malignant arrhythmia.[9]

Prognostic stratification is very important in this clinical setting, and detecting structural heart disease (SHD) is a crucial factor because it is the primary determinant of poor prognosis.[10] The clinical guidelines recommend routine transthoracic echocardiography (TTE) and coronary artery imaging in patients with malignant ventricular arrhythmia.[11] However, there is still uncertainty regarding the clinical role of cardiac magnetic resonance (CMR) because the spectrum and

prevalence of cardiac diseases identified by this imaging modality have not been established in these patients.[11,12] Tissue characterization with CMR imaging includes evaluation of irreversible tissue injury using late gadolinium enhancement (LGE) and identification of recent myocardial injury using T2-weighted "edema" imaging. The combined use of these established and validated techniques allows the detection of distinct patterns and distribution of myocardial injury that may help in determining the disease cause.[13] On the other hand, the extent of irreversible tissue injury assessed by LGE has been associated with the risk of arrhythmia in ischemic and non-ischemic patients. CMR has become a standard gold method for identifying cardiac morphology and functional characterization. It is possible to significantly increase the diagnostic power by the identification of the arrhythmogenic substrate.[14,15]

The big question is which one of the pathophysiologies of this patient, frequent PVC-induced cardiomyopathy or the cardiomyopathy that induced substrate for PVC? Why is it important to differentiate it? The treatment of each mechanism is different. This patient was in heart failure symptoms and showed ventricular arrhythmia. The ambulatory Holter monitoring showed frequent monomorphic RVOT origin PVC. The echocardiography indicated a decrease in LV function, LV dilatation with eccentric hypertrophy, and global hypokinetic motion. From the CMR, there was no evidence of "edema" or myocardium fibrosis. From the previous theory, we can conclude that frequent PVC-induced cardiomyopathy and catheter ablation is the definitive therapy.

4. Discussion

Identifying, stabilizing, and correcting the causes of cardiomyopathy improves outcomes and quality of life for patients in preexisting/without heart disease. Early recognition of the culprit arrhythmia to cardiomyopathy is paramount in providing treatment that improves symptoms, LVEF, and functional status. in this case, we reviewed the patient with diagnosed arrhythmia-induced cardiomyopathy, and 3D ablation was the definitive treatment.

5. Declarations

5.1. Ethics Approval and Consent to participate
Not applicable.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: ASS. Design: ASS. Control/supervision: DS, AR. Data collection/processing: ASS. Analysis/interpretation: AS. Literature review: DS, AR. Writing the article: ASS. Critical review: DS, AR. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

5.7 *Acknowledgements*
We thank to Brawijaya Cardiovascular Research Center.

References

1. Japp AG, Gulati A, Cook SA, Cowie MR, Prasad SK. The diagnosis and evaluation of dilated cardiomyopathy. Journal of the American College of Cardiology. 2016 Jun 28;67(25):2996-3010.

2. Centurión OA, Alderete JF, Torales JM, García LB, Scavenius KE, Miño LM. Myocardial fibrosis as a pathway of prediction of ventricular arrhythmias and sudden cardiac death in patients with non-ischemic dilated cardiomyopathy. Critical pathways in cardiology. 2019 Jun 1;18(2):89-97.

3. Sulaiman S, Yousef N, Mehanni MM, Sundararajan S, Wingert R, Wingert M, Mohammed A, Jahangir A. Burden of arrhythmia and electrophysiologic procedures in alcoholic cardiomyopathy hospitalizations. International journal of cardiology. 2020 Jan 28.

4. Gopinathannair R, Etheridge SP, Marchlinski FE, Spinale FG, Lakkireddy D, Olshansky B. Arrhythmia-induced cardiomyopathies: mechanisms, recognition, and management. Journal of the American College of Cardiology. 2015 Oct 13;66(15):1714-28.

5. Puntmann VO, Carr-White G, Jabbour A, Yu CY, Gebker R, Kelle S, Hinojar R, Doltra A, Varma N, Child N, Rogers T. T1-mapping and outcome in non-ischemic cardiomyopathy: all-cause mortality and heart failure. JACC: Cardiovascular Imaging. 2016 Jan 1;9(1):40-50.

6. Peters S, Kumar S, Elliott P, Kalman JM, Fatkin D. Arrhythmic genotypes in familial dilated cardiomyopathy: implications for genetic testing and clinical management. Heart, Lung and Circulation. 2019 Jan 1;28(1):31-8.

7. Vaseghi M, Hu TY, Tung R, Vergara P, Frankel DS, Di Biase L, Tedrow UB, Gornbein JA, Yu R, Mathuria N, Nakahara S. Outcomes of catheter ablation of ventricular tachycardia based on etiology in non-ischemic heart disease: an international ventricular tachycardia ablation center collaborative study. JACC: Clinical Electrophysiology. 2018 Sep 17;4(9):1141-50.

8. Zeppenfeld K. Ventricular tachycardia ablation in non-ischemic cardiomyopathy. JACC: Clinical Electrophysiology. 2018 Sep 17;4(9):1123-40.

9. Patel AR, Kramer CM. Role of cardiac magnetic resonance in the diagnosis and prognosis of non-ischemic cardiomyopathy. JACC: Cardiovascular Imaging. 2017 Oct 2;10(10 Part A):1180-93.

10. Ware JS, Amor-Salamanca A, Tayal U, Govind R, Serrano I, Salazar-Mendiguchía J, García-Pinilla JM, Pascual-Figal DA, Nuñez J, Guzzo-Merello G, Gonzalez-Vioque E. Genetic etiology for alcohol-induced cardiac toxicity. Journal of the American College of Cardiology. 2018 May 14;71(20):2293-302.

11. Dukkipati SR, Koruth JS, Choudry S, Miller MA, Whang W, Reddy VY. Catheter ablation of ventricular tachycardia in structural heart disease: indications, strategies, and outcomes—Part II. Journal of the American College of Cardiology. 2017 Dec 4;70(23):2924-41

12. Gopinathannair R, Etheridge SP, Marchlinski FE, Spinale FG, Lakkireddy D, Olshansky B. Arrhythmia-induced cardiomyopathies: mechanisms, recognition, and management. Journal of the American College of Cardiology. 2015 Oct 13;66(15):1714-28.

13. Disertori M, Masè M, Ravelli F. Myocardial fibrosis predicts ventricular tachyarrhythmias. Trends in cardiovascular medicine. 2017 Jul 1;27(5):363-72

14. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, Dubourg O, Kühl U, Maisch B, McKenna WJ, Monserrat L. Classification of the cardiomyopathies: a position statement from the

European Society Of Cardiology Working Group on Myocardial and Pericardial Diseases. European heart journal. 2008 Jan 1;29(2):270-6.

15. Muser D, Santangeli P, Castro SA, Pathak RK, Liang JJ, Hayashi T, Magnani S, Garcia FC, Hutchinson MD, Supple GG, Frankel DS. Long-term outcome after catheter ablation of ventricular tachycardia in patients with non-ischemic dilated cardiomyopathy. Circulation: Arrhythmia and Electrophysiology. 2016 Oct;9(10):e004328.