

Bidirectional ventricular tachycardia in rheumatic fever reactivation

Mohammad Iqbal*, Andy Sukmadja, Rosa Syafitri, Giky Karwiky and Chaerul Achmad

Department of Cardiology and Vascular Medicine, Universitas Padjadjaran, Jalan Eyckman 38, Bandung 40161, Indonesia.

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ABSTRACT

Bidirectional ventricular tachycardia (VT) is a rare rhythm disorder, usually described in digitalis intoxication, fulminant myocarditis, familial catecholaminergic polymorphic VT, familial hypokalemic periodic paralysis, and aconitine poisoning. This case describes a young male with rheumatic fever reactivation (RFR) with manifestation of myocarditis and bidirectional ventricular tachycardia (BVT). A myocarditis with BVT in RFR had not been reported before.

Keywords: Bidirectional ventricular tachycardia, rheumatic fever reactivation.

*Corresponding author. E-mail: dr_mohammadiqbal@yahoo.com. Tel: +6281310114128.

INTRODUCTION

Bidirectional ventricular tachycardia (BVT) is a rare rhythm disorder characterized by an alternating beat-to-beat of QRS axis in the frontal plane lead. BVT was initially described in 1922 as a manifestation of digitalis toxicity (Sabatini et al., 2014), It has also been reported in the setting of familial hypokalemic periodic paralysis (Stubbs, 1976), Anderson-Tawil Syndrome (Morita et al., 2007; Chakraborty et al., 2015), fulminant myocarditis (Berte et al., 2008), familial catecholaminergic polymorphic Ventricular Tachycardia (CPVT) (Leenhardt et al., 1995; Lee et al., 2009; Femenia et al., 2012), acute ischemia (Wase et al., 2014), aconitine poisoning (Sheth et al., 2015), Cardiac Sarcoidosis (Benjamin et al., 2017), and several other conditions which predispose cardiac myocytes to delayed after depolarizations (DADs) and triggered activity (Baher et al., 2011). BVT is thought to be associated with poor prognosis regardless of underlying etiology. Ventricular Tachycardia (VT) has rarely been reported in acute phase of rheumatic fever (Freed et al., 1985) and rheumatic heart disease (Shah et al., nd). However, BVT associated with carditis in rheumatic fever reactivation (RFR) had not been reported before.

CASE ILLUSTRATION

A 21 year-old man presented with palpitation and dyspnea was referred to our hospital. The patient had fever of 38.5°C, pharyngitis, and arthralgia on one week prior to admission. The patient had neither history of digoxin use nor family history of cardiac disease. Vital signs and physical examination on admission was unremarkable. The electrocardiogram (ECG) showed bidirectional VT (Figure 1). During hospitalization, ECG showed sinus rhythm with bigeminy premature ventricular contraction (PVC) and alternating beat to beat with prolonged PR interval (0.24 s) (Figure 2). Laboratory showed positive anti-streptolysin-O (ASTO) and elevated C-reactive protein (CRP) qualitatively, also mildly elevated Troponin T (0.18 ng/ml). Trans-thoracic echocardiogram (TTE) demonstrated dilated left ventricle, reduced ejection fraction (35%), global hypokinetic, trivial mitral regurgitation, trivial tricuspid regurgitation, and thickening and mild calcification of anterior tip of mitral valve leaflet (Figure 3).

The diagnosis of RFR was established based on WHO revised Jones criteria by presence of carditis (major

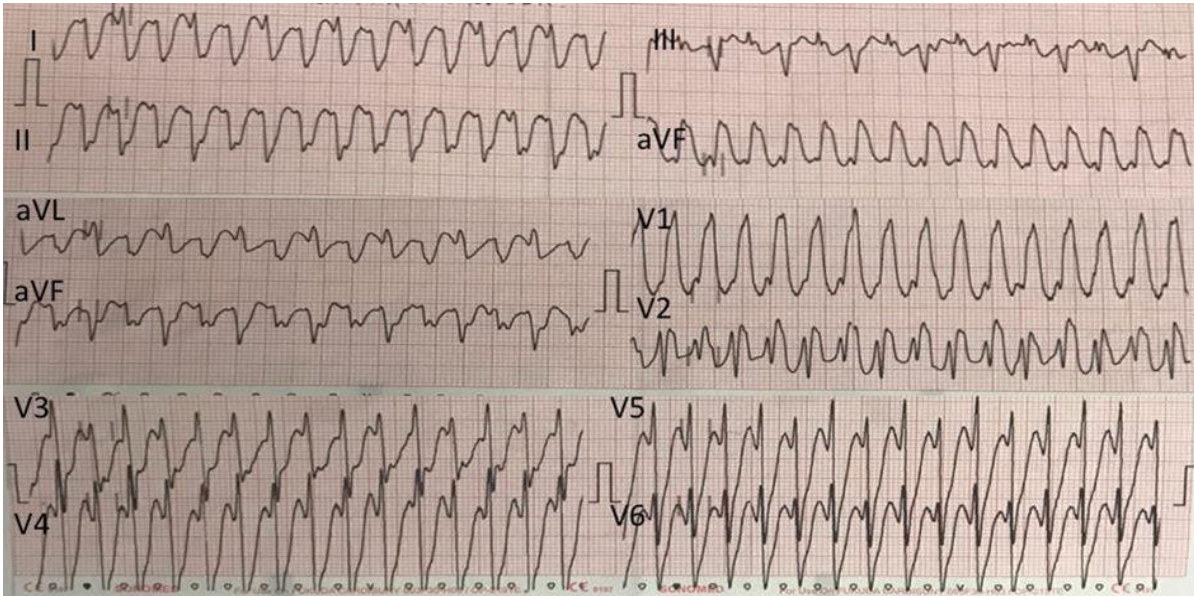


Figure 1. Alternating beat to beat during ventricular tachycardia.

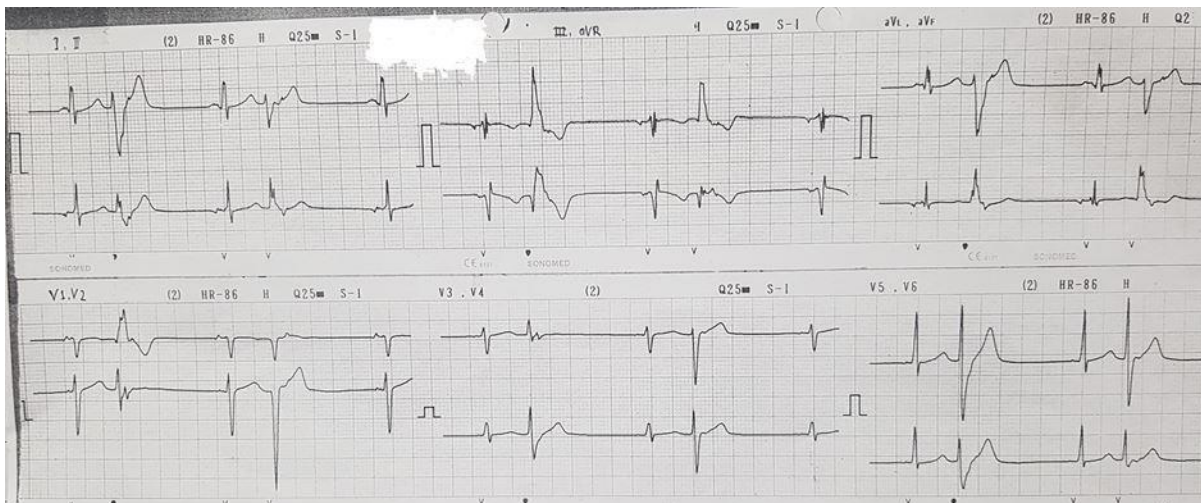


Figure 2. Sinus rhythm with bigeminy PVC alternating beat to beat with prolonged PR interval of 240 ms.

criteria), increased CRP, fever, and arthralgia (minor criteria) (Gewitz et al., 2015). Treatment with beta-blocker, angiotensin converting enzyme inhibitor, erythromycin, and high dose steroid was initiated. Patient was discharged without any VT or PVC. There are no symptoms of palpitation and heart failure during 3 months of follow up. Serial ECGs during follow up showed no VT and PVC. Ejection fraction improved $\geq 50\%$ after 3 months

DISCUSSION

Disturbance in cardiac conduction and rhythm are

common during acute phase of rheumatic fever, mostly manifested as atrioventricular conduction block, junctional tachycardia, and premature ventricular contraction. Immunologic mechanism has been implicated in development of acute carditis. Group A streptococci possess antigens that are immunologically related to heart tissue. Cross-reactive autoantibodies directed toward heart tissue were stimulated in certain patients after streptococcal infection. Immunologically mediated injury of electrically active tissue in the myocardium may have resulted in disturbances of impulse formation or conduction. Re-entry or increased automaticity of subsidiary ventricular pacemakers, and autonomic imbalance in sympathetic and

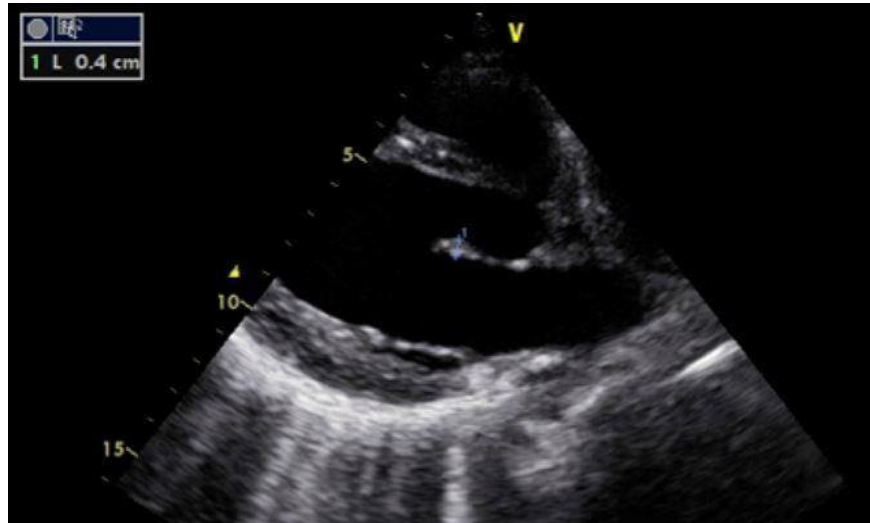


Figure 3. Parasternal long axis view showed thickening of the tip of anterior mitral leaflet (4 mm) and mild calcification.

parasympathetic tone may have precipitated arrhythmia (Freed et al., 1985). BVT had been reported previously in the case of fulminant and sub-acute myocarditis (Berte et al., 2008). VT had also been reported to be associated with acute phase of rheumatic fever and rheumatic heart disease (Freed et al., 1985; Shah et al., nd). This is the first reported case of BVT associated with subclinical carditis in RFR.

Mechanism of BVT is ectopic discharges by triggered activity mainly in the epicardium (Nam et al., 2005). BVT in myocarditis are preceded by multiple ectopic beats and increasing transmural dispersion which contribute as a substrate for VT. The triggered beat from the first site induced a triggered beat at the second site. The triggered beat from the second site reciprocally induced a triggered beat at the first site, and so forth. Bigeminy from two sites produced BVT. This ping pong mechanism of reciprocating bigeminy readily produces the characteristic electrocardiographic pattern of BVT, and its degeneration to polymorphic VT if additional sites develop bigeminy (Baheer et al., 2011). In our case, the ECG during hospitalization that showed PVC with alternating beat to beat (Figure 2) may explain why the patient had BVT.

According to the 1992 American Heart Association (AHA) revised Jones criteria statement, carditis, a major manifestation of acute phase of rheumatic fever or RFR has been considered to be a pancarditis and can involve the endocardium, myocardium, and pericardium. Clinical carditis remains universally accepted as a major manifestation in all populations. AHA Working group support the use of Echocardiography/Doppler testing to assess whether carditis is present in the absence of auscultatory findings, particularly in moderate- to high-risk populations and when Acute Rheumatic Fever is considered likely. Echocardiography/Doppler studies, however, can reveal mitral or aortic valvulitis. Prevalence

of subclinical carditis was 16.8%. Concept of subclinical carditis has become incorporated into other guidelines and consensus statements as a valid rheumatic fever major manifestation (Gewitz et al., 2015).

In our case, TTE showed thickening of anterior mitral leaflet tip and mild calcification (Figure 3) that may be a proof of preexisting rheumatic heart disease. Positive ASTO showed that RFR was an ongoing process. The diagnosis of RFR was established based on WHO revised Jones criteria¹⁶ by presence of myocarditis (one major criteria), fever, elevated CRP and arthralgia (two minor criteria), and positive ASTO.

CONCLUSION

Rheumatic fever reactivation remains an important cause of disease and death, especially in developing countries. Bidirectional VT during RFR with myocarditis has not been reported yet. Careful assessment of the BVT etiology is important to avoid misdiagnosis from other etiology.

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