Complex ventricular tachycardia coexistent with myocardial bridging

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ABSTRACT

Sustained monomorphic ventricular tachycardia is rarely concomitant condition with myocardial bridging for which no evidence-based medical management has yet been certainly described. Herein is presented a case of malignant ventricular arrhythmia that may be associated with myocardial bridge on coronary artery. The clinical management and medical treatment of the patient are discussed.

Keywords: Coronary angiography; myocardial bridging; sustained monomorphic ventricular tachycardia.

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Myocardial bridging (MB) is an inborn disorder defined by systolic compression of coronary artery lumen that normalizes during diastole. It may cause several manifestations such as angina, arrhythmia and sudden death [1]. We present a case of complex ventricular tachycardia (VT) in a patient with coronary MB.

CASE REPORT

Forty-eight years-old man presented to the emergency department due to sudden onset of palpitations and dizziness. He had no coronary risk factors and prior history of cardiac disease. Heart rate was 155 beats/minute, and blood pressure was 118/63 mm Hg. His physical examination was unremarkable. The electrocardiogram (ECG) demonstrated a wide-QRS-complex tachycardia compatible with a sustained monomorphic ventricular tachycardia (VT), which terminated spontaneously (Fig. 1). The next ECG revealed normal sinus rhythm. Laboratory analysis including troponin was normal and there were no segmental wall-motion abnormalities in advanced echocardiographic examination. Coronary angiography showed remarkable coronary compression in the middle segment of left anterior descending artery (mid-LAD) during systolic cardiac phase without significant coronary artery disease, indicating myocardial bridging (MB) (Figs. 2, 3 and Video 1). A relationship was suspected between VT and MB after excluding other possible causes of VT. Cardiac magnetic resonance imaging (MRI) did not demonstrate zones of scar that

![Figure 1. Wide-QRS-complex tachycardia compatible with a sustained monomorphic ventricular tachycardia](image-url)
could form the substrate for the sustained monomorphic VT. There was no further complaints and tachycardia with initiation of medical therapy with 100 mg metoprolol succinate. One month after this event, the patient was able to perform 10.1 METS on a Bruce protocol with a blunted maximal heart rate of 115 beats/minute on metoprolol and no perfusion defect was detected by single-photon emission computed tomography.

DISCUSSION

MB is an inborn abnormality that certain segment of epicardial coronary artery goes into heart muscle band. MB is reported angiographically in 1.22% to 15% of patients and is usually localized to mid-LAD [1, 2]. Although MB has been generally considered a benign disease, it causes serious conditions such as ischemia, acute coronary syndrome, arrhythmia (including supraventricular tachycardia and VT) and sudden cardiac death [3].

Delay in duration of ventricular relaxation due to prolongation of the MB contraction, especially with episodes of tachycardia, may cause to impair diastolic coronary perfusion. This pathologic condition may contribute to VT associated with MB [4].

Sustained monomorphic VT is a potentially life-threatening arrhythmia which should be urgently evaluated for ischemic heart disease under the guidance of multimodality imaging including advanced echocardiography, conventional coronary angiography and cardiac MRI [5].

Medical management with negative inotropic and chronotropic agents is considered as first-line therapy. Beta blockers decrease in contractility and compression of the coronary arteries by lowering the heart rate and increasing diastolic interval. Thus, these agents are generally beneficial although they have not been studied in randomized controlled trials [6].

CONCLUSION

MB may be a cause of VT although it is generally accepted as a benign anomaly.

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REFERENCES


