

Complex ventricular tachycardia coexistent with myocardial bridging

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ABSTRACT

Sustained monomorphic ventricular tachycardia is rarely a concomitant condition with myocardial bridging for which no evidence-based medical management has yet been certainly described. Here, in this case study, we present a case of malignant ventricular arrhythmia that may be associated with the myocardial bridge on the 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]. In this case study, we present a case of complex ventricular tachycardia (VT) in a patient with coronary MB.

CASE REPORT

A 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-QRScomplex 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 the advanced echocardiographic examination. Coronary angiography showed remarkable coronary compression in the middle segment of the left anterior descending artery (mid-LAD) during the systolic cardiac phase without significant coronary artery disease, indicating myocardial bridging (MB) (Fig. 2 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 could form the substrate for the sustained monomorphic VT. There were no further complaints and tachycardia with the 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 com-



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Correspondence: Sinan CERSIT, MD. Kosuyolu Kartal Kalp Egitim ve Arastirma Hastanesi, Kardiyoloji Klinigi, Istanbul, Turkey. Phone: +90 216 515 00 00 e-mail: sinancersit@hotmail.com © Copyright 2020 by Istanbul Provincial Directorate of Health - Available online at www.northclinist.com FIGURE 1. Wide-QRS-complex tachycardia compatible with sustained monomorphic ventricular tachycardia.

puted tomography. The patient's consent was obtained for this case report.

DISCUSSION

MB is an inborn abnormality that a certain segment of the epicardial coronary artery goes into the 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 may cause severe conditions, such as ischemia, acute coronary syndrome, arrhythmia (including supraventricular tachycardia and VT) and sudden cardiac death [3].

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

Sustained monomorphic VT is a potentially lifethreatening arrhythmia that 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.



FIGURE 2. (A) Coronary angiography in the posteroanterior cranial projection during systole (arrows). **(B)** Coronary angiography in the posteroanterior cranial projection during diastole.

VIDE01. Coronary angiography in the posteroanterior cranial projection during diastole and systole. There is dynamic compression of the midportion of the left anterior descending artery during systole.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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