

## CLINICAL VIGNETTE

# Vasospastic Angina vs. Myopericarditis: Unusual Presentation with Persistent ST Elevation and Chest Pain

Kayvan Kani, M.D., and Kamran Shamsa, M.D.

### Introduction

Prinzmetal (variant) angina is an uncommon cause of cardiac chest pain characterized by recurrent episodes of angina with transient ST-segment elevation that are related to focal spasm of an epicardial coronary artery due to yet unknown underlying physiopathology.<sup>1,2</sup> Cardiac biomarker levels, particularly troponin assays, are usually within normal range or mildly elevated. The key feature of variant angina is reversibility of coronary spasm with resolution of ST segment changes and anginal symptoms spontaneously or with nitroglycerin administration.<sup>1,2</sup>

We describe an interesting and uncommon presentation of a young man with chest pain, persistent ST-segment elevation, and highly elevated cardiac biomarkers consistent with ST elevation myocardial infarction (MI), without visible vasospasm on coronary angiogram.

### Case presentation

A 21-year-old male with past medical history of asthma and anxiety presented to the Emergency Department (ED) with chest pain for sixteen hours. The patient awakened from sleep early in the morning with chest pressure. The pain was severe (10/10), substernal, radiated to his left arm and back, and was associated with some dyspnea, diaphoresis, and nausea. His symptoms partially improved with analgesic medications but recurred severely in the evening, prompting him to present to the ED. He denied any upper respiratory symptoms, fever, chills, or myalgias but reported diarrhea for five days before symptom onset. He had been trying to stay hydrated by drinking water and Gatorade. There was no family history of early coronary disease, coagulation disorders, or other known risk factors for coronary artery disease. His mother was diagnosed with Wolff-Parkinson-White syndrome (WPW) and had catheter ablation in the past. He did not smoke nor take recreational drugs. Initial vital signs were stable. Physical examination was otherwise unremarkable apart from a slight tenderness in the left side of the chest. In the ED, he had ST-segment elevation in inferior leads (II, III, aVF), lateral leads (V4-V6), and 1 to 2 mm of downsloping ST-segment depression in V1 that was consistent with ST elevation myocardial infarction (Figure 1A). Creatinine kinase myocardial band (CK-MB) and troponin-I (cTnI) level returned positive at 80.5 ng/ml (normal range: less than 5.5

ng/ml) and 12.5 ng/ml (normal range; less than 0.1 ng/ml) respectively. He was started on aspirin and nitroglycerin and was immediately transferred to cardiac catheterization laboratory. Cardiac angiography revealed a dominant right coronary artery system without any atherosclerotic plaque, obstruction, or myocardial bridge. TIMI (Thrombolysis In Myocardial Infarction) angiographic flow was grade 3. Further studies including an echocardiogram showed a normal left ventricular function without wall motion abnormalities or pericardial effusion. Total white cell count, hemoglobin level, platelet count, C-reactive protein, erythrocyte sedimentation rate, thyroid stimulating hormone, electrolytes, and renal function were all within normal range. Urine toxicology screen returned normal as well.

Given the history and the normal findings of all subsequent studies, the most likely etiology of his anginal symptoms and elevated cardiac enzymes was considered to be vasospasm. He was transitioned off the nitroglycerin drip and started on oral isosorbide mononitrate, which was uptitrated after having another episode of chest pain after catheterization. Serial ECGs revealed 1 to 2 mm of persistent ST-segment elevation in the inferior and lateral leads for the entire 48-hour hospital stay (Figure 1B). His troponin-I level peaked to 28.2 ng/ml and down-trended appropriately. He was risk stratified, HgA1c was 5.2%, and total cholesterol was 113 mg/dl. In light of his clean coronaries, there was no need to initiate aspirin or a statin at the time. He was discharged home on daily isosorbide mononitrate and sublingual nitroglycerine as needed for chest pain. He remained hemodynamically stable with no recurrence of chest pain. He was seen two weeks after discharge, and repeat ECG revealed resolution of ST segment elevation with development of T wave inversions in the inferior and lateral leads (Figure 2). At 2 month follow-up, Gadolinium-enhanced magnetic resonance imaging showed a focal linear area of abnormal delayed hyper-enhancement in the inferior wall of the basal segments of the left ventricular myocardium, which was located within the epicardial and mid-myocardial region in a nonischemic territory without any associated regional wall motion abnormality (Figure 3). This finding was somewhat atypical for a myocarditis scar; however given the clinical history, the diagnosis of myopericarditis could not be ruled out.

## Discussion

In our case, the most likely etiology of symptoms was initially considered coronary vasospasm, considering the episodes of rest angina accompanied with ST elevation and the finding of normal coronary angiograms; however, the patient's persistent ST elevation, highly elevated cardiac enzymes, and precedent gastroenteritis raised the possibility of myopericarditis as a potential alternative diagnosis.

Cardiac biomarker elevation is present in a substantial number of patients presenting with variant angina and acute myopericarditis; however it is unusual, but not inconsistent, to detect elevated troponin levels to such an extent in either aforementioned conditions. No single diagnostic test definitively confirms the diagnosis of myocarditis. Cardiac magnetic resonance imaging (CMRI) is a useful diagnostic tool in patients with suspected myocarditis as it provides qualitative information on myocardial tissue characteristics. It could be a valuable addition to other methods particularly in diagnostically challenging patients with troponin-positive anginal pain and normal coronary arteries. CMRI findings in our patient were not very typical for a myocarditis scar, but given the clinical history, we were unable to rule out the diagnosis of myopericarditis. On the other hand, we were not able to confirm the diagnosis of vasospastic angina either.

Abnormal coronary vasomotion and endothelial dysfunction are implicated as the underlying mechanisms of coronary artery vasospasm. The observation that vasospastic episodes occur mostly from midnight to early morning, when vagal tone is highest, suggests a role for vagal activity as a trigger of spasm. Coronary vasospasm can be elicited by several provocative agents that trigger different receptors. Coronary angiography with selective intracoronary injection of acetylcholine in search of coronary spasm is considered a safe and reliable technique for diagnosis of variant angina as well as assessing coronary vasomotor function<sup>3</sup>; however, coronary vasospasm may be transient and not detected by angiogram.

Vasospastic angina and myopericarditis are common diagnoses in a patient presenting with acute coronary syndrome but normal coronary arteries. Diagnostic challenges may occasionally arise, especially in the setting of unusual presentations as in our patient. The application of timely diagnostic measures, long-term follow-up, and appropriate treatment remains the most effective strategy to ensure the correct diagnosis and treatment plan.

## Figures

**Figure 1:** ST-segment elevation in inferior leads (II, III, aVF), lateral leads (V4–V6), and 1 to 2 mm of downsloping ST-segment depression in V1 consistent with ST elevation myocardial infarction, (A) at the time of admission and (B) after 24 hours.

Figure 1A.

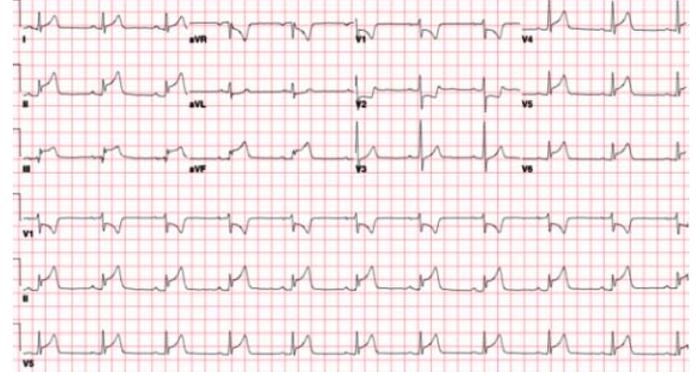
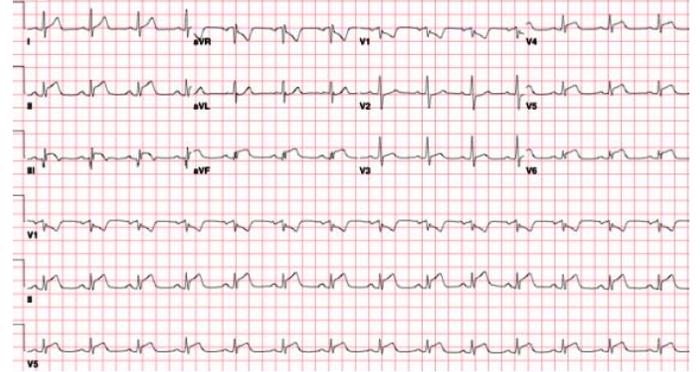
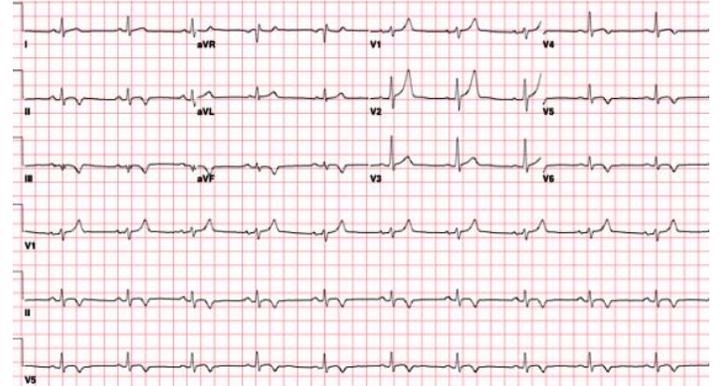


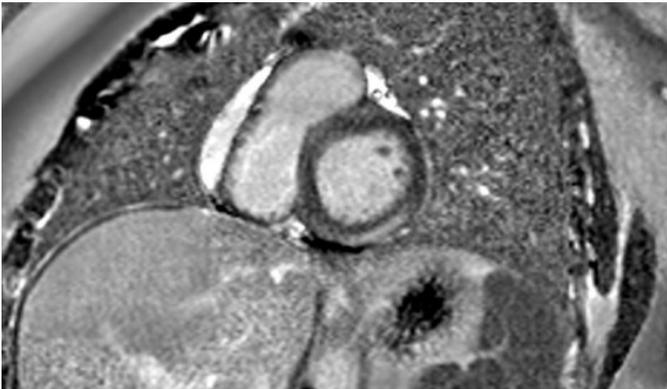
Figure 1B.



**Figure 2:** Resolution of ST segment elevation with development of T wave inversions in the inferior (II, III, aVF), lateral leads (V4–V6), and 2 weeks later.



**Figure 3:** Gadolinium-enhanced magnetic resonance imaging for detection and quantification of myocardial fibrosis: a focal linear area of abnormal delayed hyper-enhancement in the inferior wall of the basal segments of the left ventricular myocardium located within the epicardial and mid myocardial region in a non-ischemic territory, 2 months later.



## REFERENCES

1. **Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N.** Angina pectoris. I. A variant form of angina pectoris; preliminary report. *Am J Med.* 1959 Sep;27:375-88. PubMed PMID: 14434946.
2. **Prinzmetal M, Ekmekci A, Kennamer R, Kwoczynski JK, Shubin H, Toyoshima H.** Variant form of angina pectoris, previously undelineated syndrome. *JAMA.* 1960 Dec 3;174:1794-800. PubMed PMID: 13738072.
3. **Ong P, Athanasiadis A, Borgulya G, Vokshi I, Bastiaenen R, Kubik S, Hill S, Schäufele T, Mahrholdt H, Kaski JC, Sechtem U.** Clinical usefulness, angiographic characteristics, and safety evaluation of intracoronary acetylcholine provocation testing among 921 consecutive white patients with unobstructed coronary arteries. *Circulation.* 2014 Apr 29;129(17):1723-30. doi: 10.1161/CIRCULATIONAHA.113.004096. Epub 2014 Feb 26. PubMed PMID: 24573349.

Submitted March 24, 2015