

Vasospastic angina on coronary angiography

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DESCRIPTION

A 47-year-old woman with no prior medical history presented to an outside facility with chest pain. She was transferred to our facility for a ST-elevation myocardial infarction after an initial ECG revealed ST-elevations in leads V2–V5. Her coronary angiography showed severe coronary vasospasm of the mid-left anterior descending artery that resolved with intracoronary nitroglycerin and mild myocardial bridging. Her cardiac biomarkers were negative, a transthoracic echocardiogram was unremarkable, and her electrolytes were normal. Further history revealed non-exertional chest pressure with radiation to her right shoulder that occurred intermittently over the past 3 months for a few minutes at a time. These episodes often occurred during periods of stress and had improved with nitroglycerin. She had no smoking history. After the left heart catheterisation, she was started on amlodipine 5 mg daily to reduce coronary vasospasm. She did not have a recurrence of chest pain and her blood pressure was controlled. She had new T wave inversions in leads V4–V6 but was asymptomatic (figure 1 and video 1).

Vasospastic angina, otherwise known as Prinzmetal or variant angina, has been defined as nitrate-response angina that occurs at rest, coincides with transient ischaemic ECG changes, and coronary artery spasm that either resolves spontaneously or can be induced with provocative testing.¹

The exact cause of this phenomenon is unclear, but it is associated with smoking history and migraines, neither of which this patient had. Other exacerbating factors, include stress, alcohol, cocaine and ergot alkaloids.² It most frequently occurs between the ages of 50 and 60 years, and has male predominance.³ Myocardial bridging has been associated with an increased propensity for vasospasm. This has been seen in studies involving more successful provocation of coronary spasm in patients with myocardial bridging.⁴ Myocardial bridging has been described in the context of vasospastic angina.⁵

The classic clinical presentation includes non-exertional chest pain resembling classic angina, but

more prolonged and severe, typically between the evening and morning hours. Coronary angiography should be considered to rule obstructive coronary artery disease, particularly in this case in which vasospastic angina is more of a diagnosis of exclusion.⁶ The diagnosis of vasospastic angina can be challenging given the transient nature of coronary vasospasm. The gold standard of diagnosing coronary vasospasm is to perform provocative testing with stimuli such as hyperventilation or with agents such as acetylcholine or ergonovine.² However, objective evidence of coronary vasospasm was seen on this patient's coronary angiography, including response to nitroglycerin during the procedure.

The management of vasospastic angina includes eliminating risk factors, such as smoking, stress and beta-blockers. Calcium channel blockers (CCB), either dihydropyridine or non-dihydropyridine, are considered first-line therapy. Nitrates are adjunctive therapy for angina refractory to CCB.²

This case describes a diagnostically challenging clinical entity. Clinical suspicion based on history should be the starting point for evaluating these patients. Vasospasm is often a proposed diagnosis in patients presenting with angina who have angiographically normal coronary arteries or mild, non-obstructive coronary artery disease. Coronary spasm can be divided into focal and diffuse. Focal coronary spasm is associated with vasospastic angina, which comprises approximately 15% of patients with coronary vasomotor disorders. This contrasts with diffuse coronary spasm, which is thought to be related to microvascular spasm.^{7,8} Focal spasm carries a worse prognosis than diffuse.⁹

Vasospastic angina is a rare but important cause of myocardial ischaemia. Its identification is often delayed and if left untreated can lead to myocardial infarction, cardiac dysrhythmia and death.

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REFERENCES

- 1 Beltrame JF, Crea F, Kaski JC, et al. International standardization of diagnostic criteria for vasospastic angina. *Eur Heart J* 2017;38:2565–8.



Figure 1 Normal sinus rhythm with ST-elevations in leads V2–V5 without pathological Q waves. This pattern suggests transmural ischaemia without infarction.

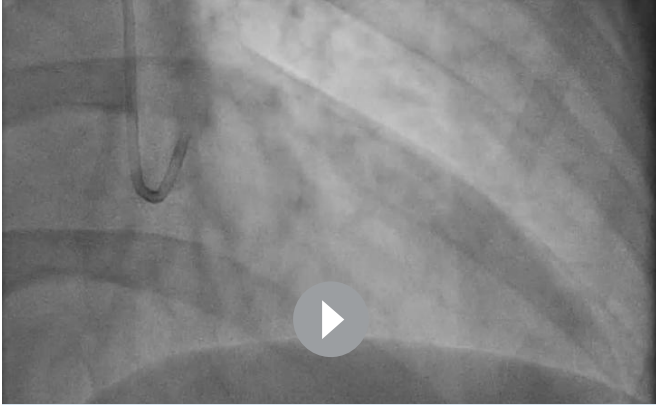


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Learning points

► Vasospastic, otherwise known as Prinzmetal or variant angina, is



- 2 Picard F, Sayah N, Spagnoli V, *et al.* Vasospastic angina: a literature review of current evidence. *Arch Cardiovasc Dis* 2019;112:44–55.
- 3 Lanza GA, Crea F. Vasospastic angina. *J Card Prac* 2003;2.
- 4 Teragawa H, Oshita C, Ueda T. The myocardial bridge: potential influences on the coronary artery vasculature. *Clin Med Insights Cardiol* 2019;13:117954681984649.
- 5 Angelini P, Uribe C, Lozano P. Differential local spasticity in myocardial bridges. *Tex Heart Inst J* 2012;39:384–8.
- 6 Thamwiwat A, Levine GN. Chest Pains and Angina. In: *Cardiology secrets*. Philadelphia, PA: Elsevier, 2018: 125–34.
- 7 Kinlay S. Coronary artery spasm as a cause of angina. *Circulation* 2014;129:1717–9.
- 8 Martínez Pereyra V, Hubert A, Seitz A, *et al.* Epicardial and microvascular coronary spasm in the same patient?-acetylcholine testing pointing towards a common pathophysiological background. *Coron Artery Dis* 2020;31:398–9.
- 9 Sato K, Kaikita K, Nakayama N, *et al.* Coronary vasomotor response to intracoronary acetylcholine injection, clinical features, and long-term prognosis in 873 consecutive patients with coronary spasm: analysis of a single-center study over 20 years. *J Am Heart Assoc* 2013;2:e000227.

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