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# The Crucial Role of Sequential Echocardiography in Spastic Angina Assessment

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***Authors' contributions***

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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***Case Report***

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## ABSTRACT

**Background:** Spastic angina, also known as Prinzmetal's angina, causes a temporary contraction of the blood vessels, reducing blood flow to the myocardium and leading to myocardial distress. Spastic angina poses a diagnostic problem, especially with the unavailability of the methyrgine test in some countries and the high risk of possible peri- and post-test complications, including death.
Our case highlights the importance of obtaining and comparing echocardiographic data both during the pericardial period and immediately after analgesia in the diagnosis of spasmodic angina without recourse to the methyrgin test.
**Case Presentation:** This case describes a 20-year-old patient, without particular risk factors or toxic habits, who was admitted to hospital because of anginal chest pain at rest, with normal cardiovascular auscultation. In the per-critical phase, echocardiography showed atypical ST-

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segment elevation, positive troponins and extensive kinetic disturbances. MRI for suspected myocarditis and coronary angiography for coronary lesions were performed without abnormalities. Post-critical echocardiograms showed partial recovery. Treatment with calcium channel blockers and anti-anginal drugs was started and brought relief, followed by complete recovery of the kinetic abnormalities on follow-up echocardiograms.
The diagnosis of spastic angina was confirmed, highlighting the importance of comparing echocardiograms performed per and post critically by the same operator.
**Conclusion:** This case raises the problem of the positive diagnosis of spastic angina and highlights the value of two comparative echocardiograms by the same operator in the diagnosis and monitoring of spastic angina, for rapid diagnosis and appropriate care to avoid a fatal complication.

*Keywords: Spastic Angina; echocardiography; chest pain; case report.*

## 1. INTRODUCTION

Acute coronary syndromes (ACS) are most commonly the result of rupture or erosion of inflammatory and lipid-laden atheromatous plaques (Virmani et al., 2006). However, some manifestations of resting angina involve an entirely different pathophysiological mechanism, namely coronary spasm.

Although spastic angina has been a recognised clinical and pathophysiological entity for many years, it often remains undiagnosed due to inadequate recognition and investigation, and its prevalence varies between countries, with a clear predominance in men.

This condition can be potentially serious, leading to arrhythmias including ventricular fibrillation and sudden death (Benamer & Millien, 2018).

Coronary spasm results from endothelial dysfunction and/or smooth muscle cell hyperreactivity triggered by endogenous (acetylcholine, catecholamines, serotonin, histamine, etc.) or exogenous vasoconstrictive stimuli (certain drugs such as sumatriptan, certain toxins such as tobacco and cocaine) (Lanza et al., 2011).

Spastic angina, first described by Prinzmetal et al. in 1959, is characterised by particularly severe anginal symptoms, which tend to occur at rest, early in the morning or at night.

Electrocardiogram (ECG) recordings during these critical episodes show ST segment changes mimicking transmural myocardial infarction, which normalise with pain relief (Prinzmetal et al., 1959).

In cases where coronary angiography shows no significant stenosis, a methylergometrine provocation test can be performed (Hayashi et al., 2003), which reliably diagnoses coronary spasm under safe conditions.

The therapeutic basis for spastic angina includes lifestyle and dietary changes, with smoking cessation being crucial, and vasodilator treatment, mainly calcium channel blockers combined with nitrate derivatives (Mishra, 2006).

## 2. CASE PRESENTATION

A 20-year-old patient, without specific risk factors or toxic habits, was admitted to the cardiac intensive care unit with typical angina pectoris at rest and normal cardiovascular auscultation.

The initial electrocardiogram showed an atypical ST-segment elevation in the inferior and lateral leads without inferior resolution (Fig. 1), high troponin levels (2000 IU) and kinetic disturbances extending to the anterolateral and inferoseptal walls with anterior wall akinesia on percutaneous echocardiography and a left ventricular ejection fraction (LVEF) of 15% on Simpson biplane.

An MRI (Fig. 2) for suspected myocarditis was normal, and a coronary angiogram (Fig. 3) was performed and showed no abnormalities.

Initially treated with a calcium channel blocker and an antianginal agent, the chest pain resolved after 3 hours with a clinically adequate blood pressure of 123/72 mmHg.

A post-critical ECG showed early repolarisation with clear resolution of ST-segment elevation (Fig. 4), and a follow-up echocardiogram (H3) showed partial recovery of kinetic abnormalities in the apical segments of the anterolateral, inferolateral and anterior walls, LVEF at 30% by Simpson's biplane.

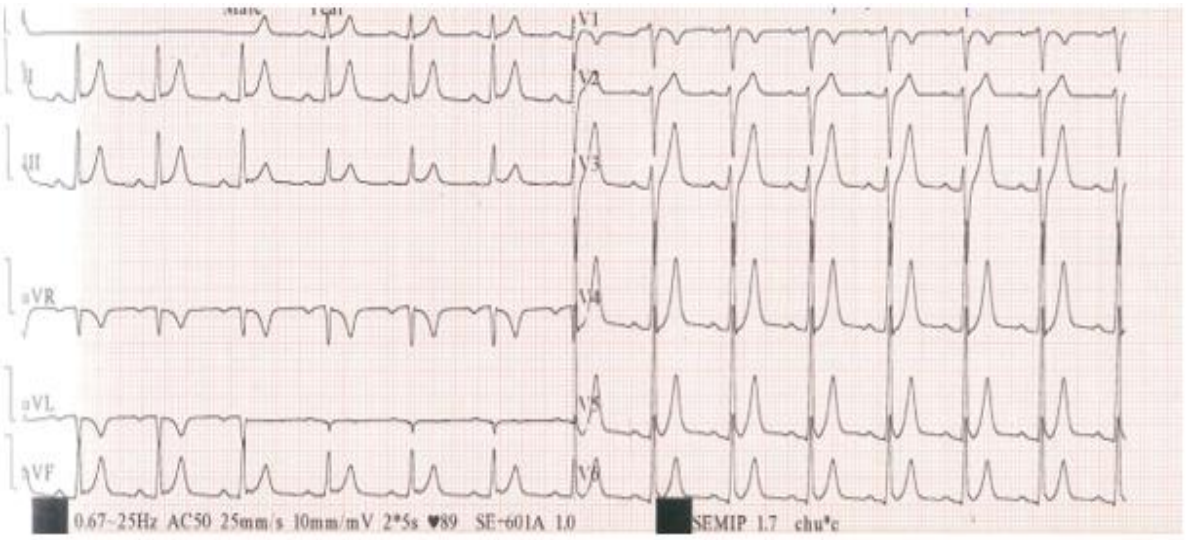


**Fig. 1. Per Critical Electrocardiogram Showing an Atypical St-Segment Elevation In The Inferior And Lower Lateral Regions**

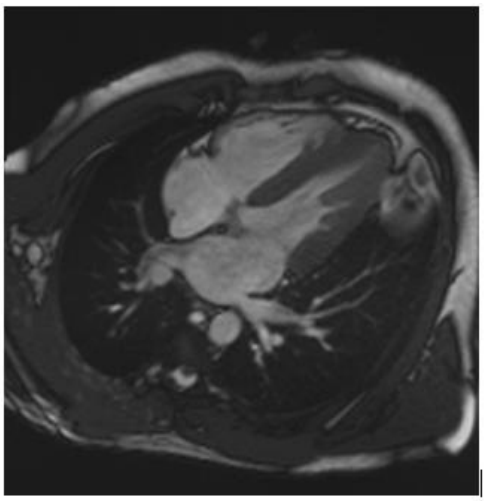

**Fig. 2. Cardiac MRI Without Abnormalities**

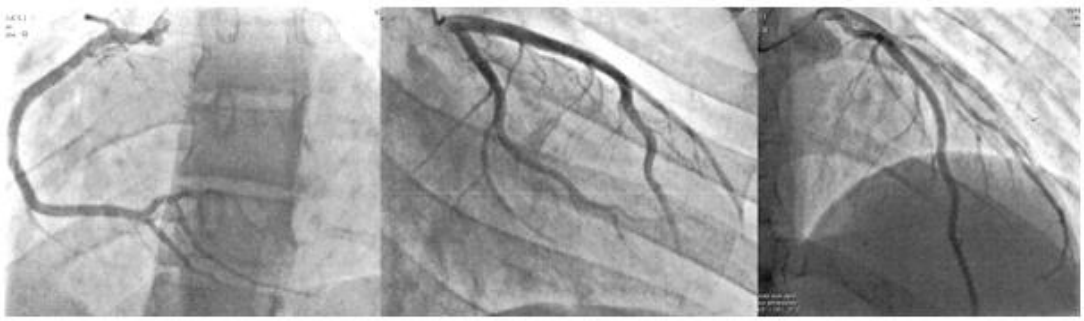

**Fig. 3. Coronary Angiography Without Abnormalities**

A diagnosis of spastic angina was immediately made. A second echocardiographic follow-up (H8) showed preserved LVEF and complete recovery at one month with normalisation of ejection fraction and global longitudinal strain (Fig. 5).

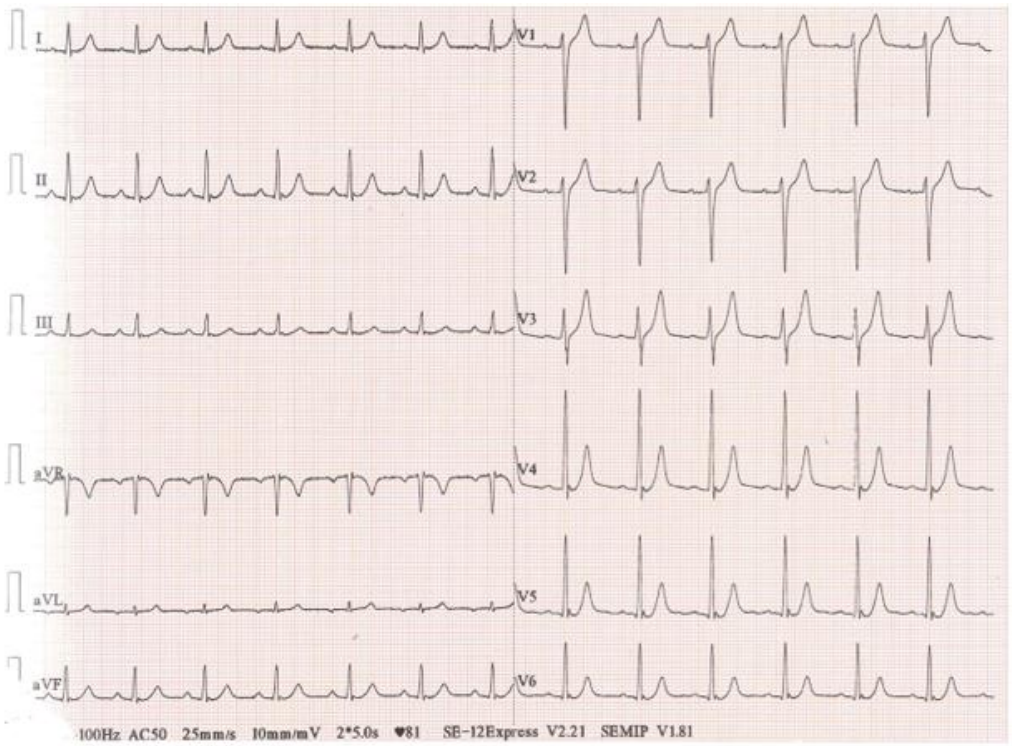


**Fig. 4. Post critical ECG showing early repolarization with a clear regression of st segment elevation**


**Fig. 5. Normalization of the ejection fraction and the strain global longitudinal at the 1 month follow up**

Treatment with a calcium channel blocker (verapamil 240 mg) and nitrate derivatives was maintained, and a 1-year follow-up showed no episodes of angina.

This clinical case has taught us the importance of obtaining echocardiographic data both during the pericritical period and immediately after analgesia.

The partial or complete recovery of the kinetic abnormalities allowed us to confidently conclude the diagnosis of Prinzmetal's angina. Therefore, we recommend that two comparative echocardiograms performed by the same operator, as they may be of significant diagnostic value.

## 3. DISCUSSION

Variant or Prinzmetal's angina differs from other forms in its pathophysiology, prognosis and treatment (Vandergoten et al., 1999). It is caused by a sudden reduction in the calibre of one or more coronary arteries due to significant vasoconstriction, and occurs predominantly in young men. It can cause severe transient myocardial ischaemia, often transmural.

Coronary artery spasm may be associated with a pre-existing atheromatous stenosis of 30% to 60%, or it may affect one or more coronary arteries with a normal angiographic appearance. In the first case, spasm is an aggravating factor that triggers angina, usually during exertion or exposure to cold, similar to classic angina. In the latter form, which is more common in young people, especially if they have other vasospastic abnormalities such as Raynaud's syndrome or are heavy smokers.

The prevalence of CAD varies widely between races and countries, but it remains the leading cause of ischaemic heart disease with non-obstructive coronary lesions (Hung et al., 2004). In fact, it is more common in men than in women (Takagi et al., 2013), in people aged between 40 and 70 years (D'Agate et al, 2002), and more common in Japanese (24.3%), followed by Taiwanese (19.3%) and Caucasian (7.5%) populations (Sueda et al., 2003).

Pain occurs at rest, often late at night, postprandially or after recovery from exercise. Painful episodes are intense, often lasting more than ten minutes, accompanied by diaphoresis and generally responsive to nitroglycerin (Bertrand et al., 1982).

Resting electrocardiograms are usually normal or show repolarisation abnormalities similar to those seen in classic angina. Exercise ECGs may be normal or show changes similar to stable angina. During spasm and painful crises, the electrocardiogram will show sub-epicardial injury, characteristic of Prinzmetal's angina. This injury, which is often of considerable amplitude, disappears with the resolution of the spasm and does not show Q waves of necrosis. The electrical image reflects a transient acute transmural ischaemia resulting from a total blockage of flow in an arterial territory, often accompanied by ventricular hyperexcitability leading to premature beats or ventricular tachycardia salvoes (Schaffer & Cobb, 1975), the presence of which can be visualised during Holter monitoring.

Prinzmetal's angina is characterised by the disappearance of rhythm or conduction disturbances without treatment once the transient ischaemic episode has resolved.

Coronary angiography determines whether or not the coronary arteries are stenosed. If no significant stenosis is found, a methylergometrine provocation test can be performed during angiography to provoke a spasm and relieve it with an intracoronary injection of nitroglycerin.

The main treatment for spastic angina is smoking cessation and the administration of calcium channel blockers and/or nitrate derivatives. Non-selective beta-blockers are contraindicated as they may promote spasm.

The prognosis of this type of angina, especially if it occurs on healthy coronary arteries, is good, even if symptomatic spasm persists despite good medical treatment (Cobbe et al., 1996).

In most cases of spastic angina, the spasm is localised to a single arterial trunk, usually the right coronary artery (51% of cases), but it may also involve the left anterior descending (30% of cases) or circumflex (11% of cases) (Cardona et al., 2016). If the spasm is localised to a well-defined segment of the coronary arterial network, local mechanical treatment may be considered if the patient remains refractory to medical treatment.

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The use of angioplasty with stenting for spastic angina should be a very limited indication for refractory angina despite good medical therapy with documented focal spasm, given the relatively high risk of restenosis reported in the literature. Older studies have shown a high recurrence rate of severe arrhythmias in survivors of out-of-hospital cardiac arrest, which has a major impact on their prognosis (Beltrame et al., 2016; Bory et al., 1996).

The indication for sympathectomy or implantable cardiac defibrillator was limited to life-threatening situations defined by survivors of cardiac arrest and episodes of ventricular arrhythmia (Ahn et al., 2016).

In the case of ventricular arrhythmias (Ahn et al., 2016)., ICD placement should not be automatic, but should be conditional on the persistence of a positive spasm provocation test under maximal antispasmodic treatment, including at least two calcium channel blockers, nitrate derivatives and complete cessation of smoking.

The first 3 months after a CAS attack is the critical period with the highest risk of cardiovascular events (Takagi et al., 2013). Cardiac death occurs in 0 to 10% of patients with CAS (Matsuo et al., 2009) and recurrent vasospastic angina has been observed in 3.9 to 18.6% (Kaski et al., 1991). Risk and prognostic stratification scores are being developed by the Japanese Coronary Spasm Association to evaluate patients with CAS (Yokoi et al., 2010).

## 4. CONCLUSION

Spastic angina is a well-known and common pathology with significant morbidity and mortality, including a notable risk of sudden death.

The diagnosis is based on the association of clinical and electrocardiographic signs, confirmed by a methylergometrine provocation test.

Our case report demonstrates the importance of recognising the value of echocardiographic data both in the pericritical phase and immediately after analgesia.

The partial or complete recovery of kinetic disturbances could help in the diagnosis of Prinzmetal's angina. We therefore advocate the use of two comparative echocardiograms performed by the same operator, as this approach has considerable diagnostic value.
Smoking cessation is a crucial aspect of treatment, along with anti-spasmodic drug therapy consisting of calcium channel blockers and/or nitrate derivatives.

The risk of sudden death remains for several years after diagnosis.

For patients who remain symptomatic despite good medical management, coronary angioplasty or revascularisation surgery may be considered.

Implantable cardioverter defibrillator (ICD) implantation is controversial in patients with arrhythmias associated with spastic angina.

## DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

## ACKNOWLEDGEMENTS

We thank the patient for their approval of publishing this case report.

## AVAILABILITY OF DATA AND MATERIALS

The data of this case report includes the echocardiography film and all other patient's data that we had a consent to publish. These data are available from the corresponding author on reasonable request.

## CONSENT

Written informed consent was obtained from the parents for publication of this case report and accompanying images.

## ETHICAL APPROVAL

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

### COMPETING INTERESTS

Authors have declared that no competing interests exist.

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