Case report

The Crucial Role of Sequential Echocardiography in Spastic Angina Assessment

ABSTRACT

Background: Spastic angina, also known as Prinzmetal's angina cause a temporary contraction of the blood vessels, reducing blood flow to the heart muscle and leading to myocardial suffering. spastic angina poses a diagnostic problem, especially with the unavailability of the methyrgin test in some countries and the major risk of possible per- and post-test complications, up to and including death.
our case highlights the importance of obtaining echocardiography data both during the pericritical period and immediately after pain sedation in the diagnosis of spastic angina

Case presentation : This case report describes a 20-year-old patient with no specific risk factors or toxic habits, initially hospitalized for resting anginal chest pain, with a strictly normal clinical examination . The first hospitalization revealed an atypical ST-segment elevation, elevated troponin levels, and a normal echocardiogram. Despite normal results from an MRI prompted by suspected myocarditis, subsequent coronary angiography showed no anomalies. Upon readmission for recurrent anginal pain with initial hypotension, the per-critical phase exhibited atypical ST-segment elevation, positive troponins, and extensive kinetic disturbances on echocardiogram. Intense peri-umbilical pain and vomiting prompted an abdominal CT angiography with no anomalies detected. Treatment with a calcium channel blocker and anti-anginal medications provided relief. Post-critical ECG showed early repolarization, and follow-up echocardiograms indicated partial and then complete recovery of kinetic abnormalities. The diagnosis of spastic angina was confirmed, highlighting the importance of echocardiography during the pericritical period and after pain resolution.

Conclusion : This case raises the problem of the positive diagnosis of spastic angina, highlighting the value of two comparative echocardiograms by the same operator in diagnosing and monitoring spastic angina ,for rapid diagnosis and appropriate care to avoid 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 [1]. However, some manifestations of resting angina involve a completely different pathophysiological mechanism, specifically coronary spasm. Spastic angina, though a well-known clinical and pathophysiological entity for many years, often goes undiagnosed due to insufficient consideration and investigation. This condition can be potentially serious, leading to rhythm disturbances, including ventricular fibrillation and sudden death [2]. Coronary spasm results from endothelial dysfunction and/or hyperreactivity of smooth muscle cells triggered by endogenous

(acetylcholine, catecholamines, serotonin, histamine, etc.) or exogenous stimuli with vasoconstrictor effects (certain drugs like Sumatriptan, certain toxins like tobacco and cocaine) [3]. First described by Prinzmetal et al. in 1959, patients with spastic angina exhibit particularly loud anginal symptoms, occurring preferably at rest, in the early morning, or at night. Electrocardiogram (ECG) recordings during these critical episodes reveal ST-segment changes mimicking transmural myocardial infarction, normalizing with the alleviation of pain [4]. In cases where coronary angiography does not reveal significant stenosis, a methylergometrine provocation test can be performed [5], reliably diagnosing coronary spasm under secure conditions. The therapeutic foundations for spastic angina include lifestyle and dietary changes, with smoking cessation being crucial, and vasodilator treatment, primarily calcium channel blockers combined with nitrate derivatives [6].

2. CASE PRESENTATION

A 20-year-old patient, without specific risk factors or toxic habits, previously hospitalized in the cardiology intensive care unit for typical resting anginal chest pain.with a strictly normal clinical examination. Initial electrocardiogram showed an atypical ST-segment elevation in the lower and lateral areas without a mirror image, elevated troponin levels (2000 UI), and a normal echocardiogram. Subsequent MRI on the third day, prompted by suspected myocarditis, returned normal (figure 1). A coronary angiography (figure 2) was performed, yielding no anomalies . The patient was readmitted for similar anginal pain with initial hypotension (80/61 mmHg). The per-critical electrocardiogram showed an atypical ST-segment elevation in the inferior and lower lateral regions (figure 3), positive troponins (1300 UI), and kinetic disturbances extending to the anterolateral and inferoseptal walls with anterior wall akinesia at the per-critical echocardiogram, left ventricular ejection fraction (LVEF) at 15% by Simpson's biplane . This episode was accompanied by intense peri-umbilical pain and vomiting without associated gastrointestinal bleeding, prompting an abdominal CT angiography that revealed no anomalies. Initially treated with a calcium channel blocker and an anti-anginal, chest pain subsided after 3 hours, with a clinically adequate blood pressure of 123/72 mmHg. A post-critical ECG showed early repolarization with a clear regression of ST-segment elevation (figure 4). A

follow-up echocardiogram (H3) indicated partial recovery of kinetic abnormalities in the apical segments of the anterolateral, inferolateral, and anterior walls, LVEF at 30% by Simpson's biplane, The diagnosis of spastic angina was immediately made. A second echocardiographic control (H8) showed preserved LVEF and a complete recovery at the one month follow up with normalization pf the ejection fraction and the strain global longitudinal. (Figure 5). Treatment with a calcium channel blocker (verapamil 240 mg) and nitrate derivatives was maintained, a 1-year follow-up showed no episodes of angina.

We have learned from this clinical case the importance of obtaining echocardiography data both during the pericritical period and immediately after pain sedation. The partial or complete recovery of kinetic abnormalities allowed us to confidently conclude the diagnosis of Prinzmetal's angina. Therefore, we recommend performing two comparative echocardiograms conducted by the same operator, which can be of significant diagnostic value.

FIGURE 1. CARDIAC MRI WITHOUT ABNORMALITIES

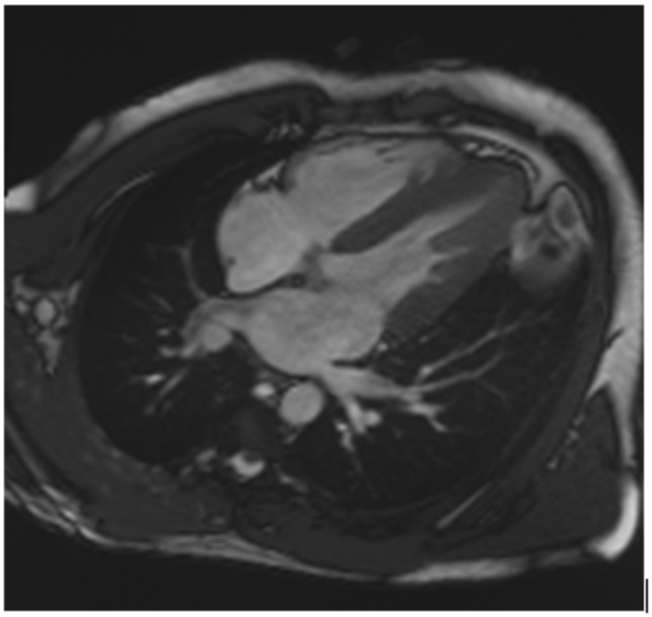


FIGURE 2. CORONARY ANGIOGRAPHYWITHOUT ABNORMALITIES

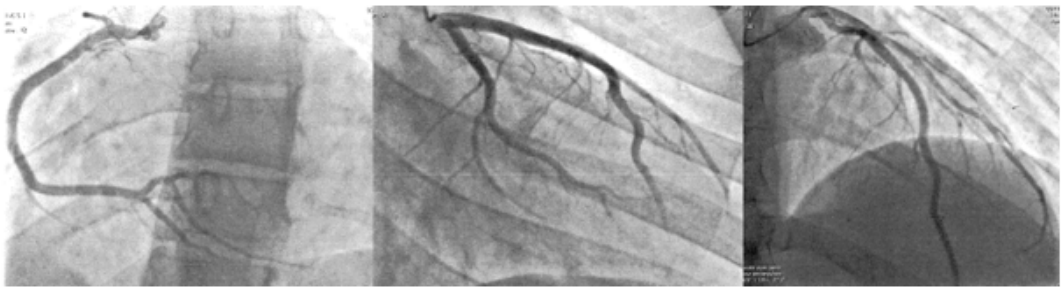


FIGURE 3. PER CRITICAL ELECTROCARDIOGRAM SHOWING AN ATYPICAL ST-SEGMENT ELEVATION IN THE INFERIOR AND LOWER LATERAL REGIONS

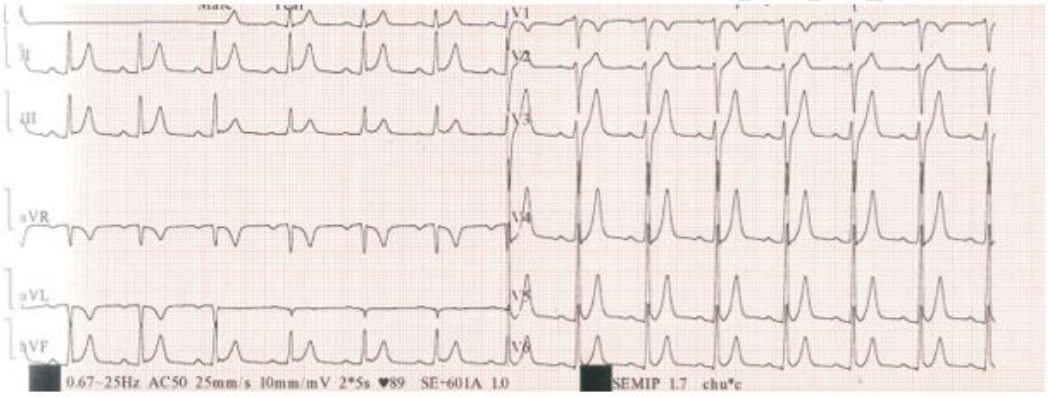


FIGURE 4. POST CRITICAL ECG SHOWING EARLY REPOLARIZATION WITH A CLEAR REGRESSION OF ST SEGMENT ELEVATION

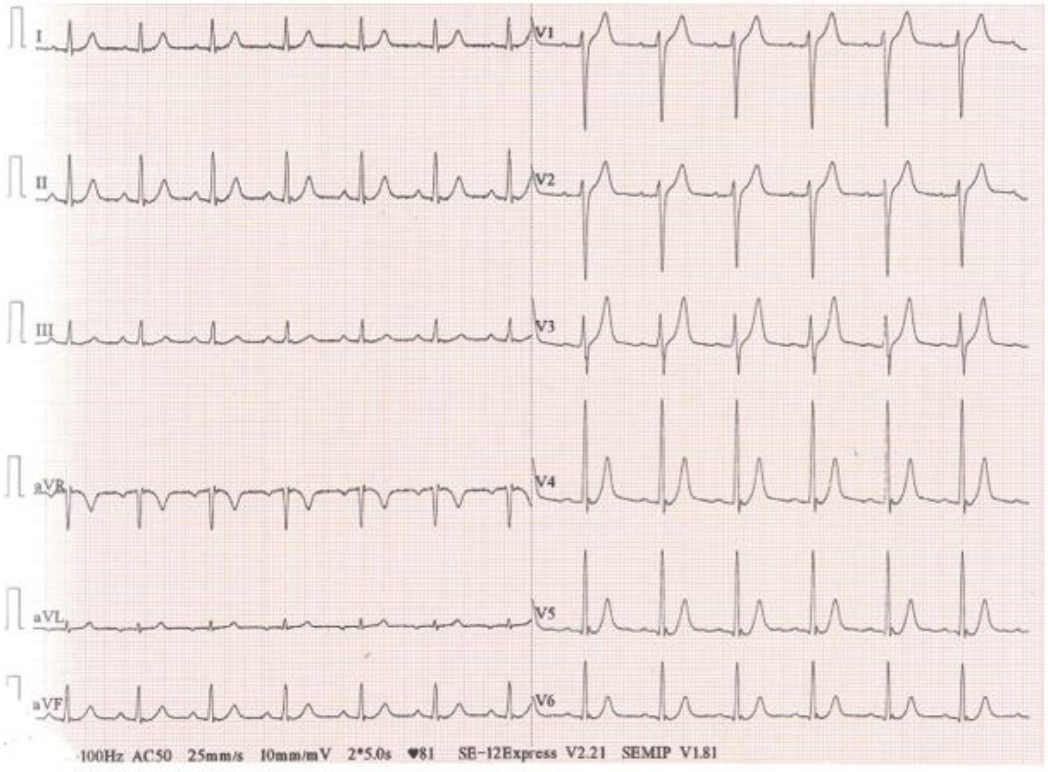
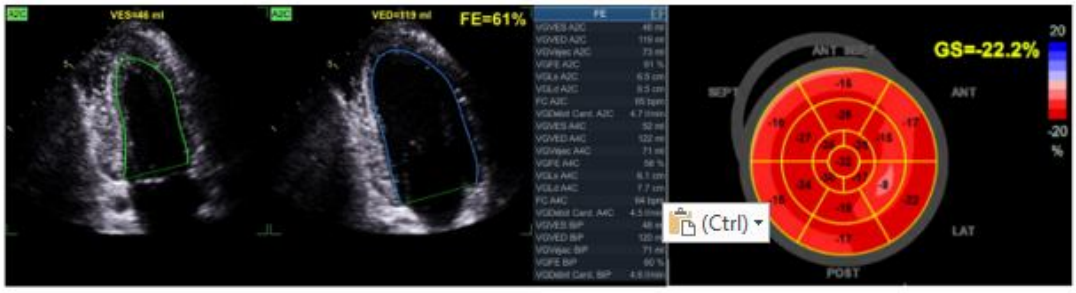


FIGURE 5. NORMALIZATION OF THE EJECTION FRACTION AND THE STRAIN GLOBAL LONGITUDINAL AT THE 1 MONTH FOLLOW UP



3. DISCUSSION

Variant or Prinzmetal's angina is distinguished from other forms by its pathophysiology, prognosis, and treatment [7]. It is caused by a sudden reduction in the caliber of one or more coronary arteries due to significant vasoconstriction, occurring preferably in young males. It can cause serious transient myocardial ischemia, often transmural. Coronary spasm can occur in conjunction with a pre-existing atheromatous stenosis of 30 to 60%, or it may affect one or more coronary arteries with a normal appearance on angiography. In the former case, spasm is an exacerbating factor triggering angina, usually during exertion or exposure to cold, similar to classic angina. In the latter form, which is more common in young individuals, especially if they exhibit other vasospastic anomalies such as Raynaud's syndrome or are heavy smokers, pain occurs at rest, often in the late night, postprandial, or after physical exertion recovery. Painful episodes are intense, often lasting beyond ten minutes, accompanied by sweating, and generally responsive to nitroglycerin [8]. Resting electrocardiograms are usually normal or show repolarization disorders, similar to classic angina. Exercise electrocardiograms may be normal or present modifications comparable to stable angina. During spasm and painful crises, the electrocardiogram will reveal sub-epicardial injury, characteristic of Prinzmetal's angina. This injury, often of significant amplitude, disappears with the resolution of the spasm, showing no Q waves of necrosis. The electrical image reflects transient acute transmural ischemia, resulting from a total interruption of flow in an arterial territory, often accompanied by ventricular hyperexcitability, leading to premature beats or ventricular tachycardia salvoes [9]. Atrioventricular block can also occur. What characterizes Prinzmetal's angina is the disappearance of rhythm or conduction disturbances without treatment as soon as the transient ischemic episode regresses. The presence of these disturbances can be visualized during Holter monitoring. Coronary angiography determines whether the coronaries are stenosed or not. If no significant stenosis is found, a methylergometrine provocation test can be performed during angiography to provoke spasm and relieve it by intracoronary nitroglycerin injection. Spastic angina is primarily treated by 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 coronaries, is good, even if symptomatic spasm persists despite well-conducted medical treatment [12]. In most cases of spastic angina, the spasm is localized to a single arterial trunk, usually the right coronary artery (51% of cases), but it can also involve the left anterior descending (30% of cases) or circumflex (11% of cases) [14]. When spasm is localized to a well-defined segment of the coronary arterial network, local mechanical treatment may be considered if the patient remains refractory to medical treatment. The use of angioplasty with stent placement for spastic angina should be a very limited indication for refractory angina despite well-conducted medical treatment with documented focal spasm, considering the relatively high risk of restenosis reported in the literature. Older studies have shown high recurrence rates of severe rhythm disturbances in survivors of out-of-hospital cardiac arrest, strongly affecting their prognosis [15,16].
Consequently, indications for implantable cardioverter-defibrillator (ICD) placement in this context have been greatly expanded. However, ICD placement should not be automatic but conditioned by the persistence of positive spasm provocation test under maximum antispasmodic treatment, including at least two calcium channel

blockers, nitratederivatives, and complete smoking cessation. Ventricular arrhythmias during spasm are rather rare (1.5% of cases) and exceptionally lead to sudden death [13].

4. CONCLUSION

Spastic angina is a well-known and common pathology with significant morbidity and mortality, including a notable risk of sudden death. Diagnosis relies on the association of clinical and electrocardiographic signs, confirmed by a methylergometrine provocation test. Our case report showed the importance of realization the significance of incorporating echocardiographic data during both the pericritical phase and immediately following pain sedation. The partial or complete restoration of kinetic disturbances could help to diagnose Prinzmetal's angina. Consequently, we advocate for the implementation of two comparative echocardiograms conducted by the same operator, as this approach holds substantial diagnostic merit. Smoking cessation is a crucial aspect of treatment, along with antispasmodic drug therapy consisting of calcium channel blockers and/or nitrate derivatives. The risk of sudden death remains present for several years after diagnosis. For patients whoremain symptomatic despite well-conducted medical treatment, coronary angioplasty or revascularization surgery can be considered [17]. Implantation of an implantable cardioverter-defibrillator (ICD) is controversial for patients presenting with arrhythmias concomitant with spastic angina.

CONSENT : PATIENT CONSENT WAS OBTAINED PRIOR TO THE STUDY

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