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Vasospastic Angina Confirmed by Nitrate Challenge: A Case Report

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Abstract **Case Report**

A 61-year-old male smoker with hypertension presented with exertional chest pain and minor troponin elevation. ECG showed apico-lateral T wave inversions. A positive stress test led to coronary angiography, which revealed a subocclusion of the right coronary artery. After intracoronary nitrate injection, the lesion resolved completely, confirming coronary artery spasm. Vasospastic angina remains an underdiagnosed cause of chest pain and can mimic fixed coronary lesions, potentially leading to unnecessary interventions. Smoking is a key risk factor, and nitrate responsiveness is critical for diagnosis. This case highlights the importance of recognizing functional coronary obstruction and the role of nitrates in avoiding misdiagnosis.

Keywords: Vasospastic Angina, Coronary Artery Spasm, Prinzmetal Angina, Nitrate-Responsive Stenosis, Coronary Angiography.

INTRODUCTION

Vasospastic angina is a transient, reversible cause of myocardial ischemia resulting from focal or diffuse coronary artery spasm, often occurring in the absence of significant fixed atherosclerosis [1]. While classically presenting with rest angina and transient ST-segment elevation, its clinical spectrum includes exertional symptoms and dynamic ECG changes, sometimes mimicking acute coronary syndromes. The diagnosis requires a high index of suspicion, particularly in patients with normal or equivocal coronary angiography [2]. In such cases, intracoronary nitrate administration plays a pivotal role in differentiating dynamic vasoconstriction from true anatomical stenosis [3]. We report a case of exertional angina in a middle-aged smoker, where a subocclusive right coronary lesion resolved completely following nitrate injection, confirming the vasospastic nature of the presentation.

CASE REPORT

A 61-year-old North African male, with a 30 pack-year history of active smoking and long-standing arterial hypertension, presented to the Cardiology Department of the Military Hospital of Instruction Mohammed V in Rabat, Morocco, for exertional chest pain. The discomfort was brief, non-radiating, and reproducible with effort, raising suspicion for stable angina. He denied syncope, palpitations, or orthopnea. Physical examination was unremarkable, with normal blood pressure and heart sounds. A resting 12-lead ECG demonstrated sinus rhythm with isolated negative T waves in the apico-lateral leads. Laboratory tests revealed a slight elevation in high-sensitivity cardiac troponin T, exceeding the 99th percentile threshold without dynamic rise, suggesting minimal myocardial injury without clear evidence of infarction.

Given the intermediate pre-test probability of coronary artery disease and inconclusive resting ECG/troponin findings, a treadmill exercise stress test (Bruce protocol) was performed. The patient developed typical chest discomfort and 1.5 mm ST-segment depression in the inferolateral leads during stage II, leading to early test termination. This was interpreted as a clinically and electrically positive result for inducible myocardial ischemia.

Coronary angiography was promptly scheduled. The angiographic study revealed an apparent subocclusion of the mid-segment of the right coronary artery (RCA), initially raising concern for high-risk single-vessel obstructive disease. However, after administration of 200 µg of intracoronary nitroglycerin, the stenosis resolved completely with normalization of luminal diameter, confirming the diagnosis of dynamic coronary artery spasm (Figure 1A and 1B). The left

coronary arteries were free of significant stenosis or anatomical abnormalities.

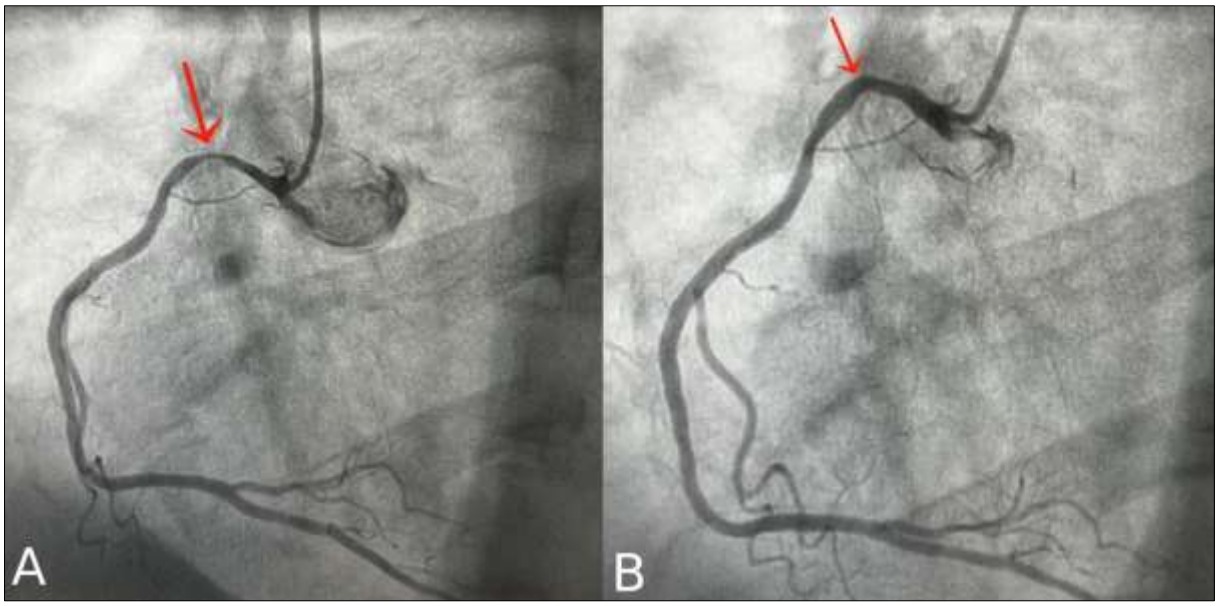

Figure 1: **(A) Subocclusion of the mid-right coronary artery before nitrate injection; (B) complete resolution after intracoronary nitroglycerin, confirming vasospastic angina**

The patient was initiated on long-acting calcium channel blockers and advised strict smoking cessation, which he accepted after structured counseling. He remained asymptomatic on follow-up with no recurrence of angina. This case underscores the importance of considering functional coronary disorders in the differential diagnosis of suspected ischemia, particularly in smokers or patients with angiographically ambiguous lesions.

All investigations were performed in accordance with institutional clinical protocols. Informed written consent was obtained from the patient for both the procedure and publication. Ethical approval for the publication of this case report was granted by the local ethics committee of the Military Hospital of Instruction Mohammed V. Patient anonymity was maintained in compliance with the Declaration of Helsinki.

DISCUSSION

Coronary vasospastic angina (VSA) is increasingly recognized as an important cause of myocardial ischemia, even in patients without significant fixed stenoses [4]. Large registries suggest a substantial fraction of "angina" patients have a vasospastic component, for example, surveys in Japan report that up to 40% of anginal patients exhibit VSA, and is more commonly documented in men – a difference largely explained by higher tobacco exposure in males [4]. The prevalence varies by geography: East Asian cohorts show rates in the 19–24% range, whereas Western populations have rates on the order of ~7–8% [5]. Our patient – a 61-year-old hypertensive man with a long history of heavy smoking – fits the classic demographic profile for VSA. His constellation of risk factors (age, gender, smoking, hypertension) is well aligned with those seen in contemporary VSA series.

Cigarette smoking, in particular, is the single most important trigger and risk factor for epicardial coronary spasm [6]. Tobacco exposure promotes endothelial dysfunction and vascular smooth-muscle hyper-reactivity, priming the coronaries for exaggerated constriction [6]. In contrast, the role of hypertension is less well-defined, although chronic hypertension can further impair endothelial function and amplify vasomotor tone [7]. The pathophysiology of VSA involves transient, reversible hypercontractility of coronary smooth muscle (e.g. via Rho-kinase pathways) superimposed on impaired endothelium-mediated vasodilation. These abnormalities can produce focal (segmental) or diffuse spasm of an epicardial artery [4]. Clinically, spasm may be precipitated by autonomic fluctuations, circadian factors (often in the early morning), cold exposure, or other stimuli, and can recur episodically in patients with otherwise unobstructed coronaries.

In typical cases, VSA causes rest angina (often nocturnal or early morning) with transient ischemic ECG changes [2]. Prinzmetal's original description emphasized transient ST-segment elevation during such episodes, and indeed ST-elevation in the corresponding leads is often seen when a major coronary segment is fully occluded by spasm [2]. However, partial or sub-total spasm may instead produce ST-segment depression. Notably, exercise-induced VSA – as in our patient – is

relatively uncommon but well-described [8]. Although exertional symptoms raise suspicion for fixed atherosclerosis, a vasospastic mechanism must still be considered. In our patient, the acute chest pain occurred with exertion, which highlights that the clinical spectrum of VSA can overlap with typical angina. Key diagnostic clues came from angiography: during catheterization the right coronary artery appeared sub-totally occluded, yet this lesion completely resolved after intracoronary nitroglycerin. In practice, an angiographic "stenosis" that reverses with nitroglycerin is pathognomonic of spasm [9]. Indeed, diagnostic criteria for definite VSA emphasize nitrate-responsive angina with transient ischemic ECG changes (or provoked spasm) [9]. When uncertainty remains, invasive provocation testing with acetylcholine or ergonovine is considered the gold-standard to confirm VSA [4]. Such testing can unmask spasm in vessels that appear normal at baseline and should be performed at experienced centers in patients with high clinical suspicion.

Once diagnosed, management of VSA focuses on preventing spasm and relieving acute attacks [10]. Acute therapy is based on rapid vasodilation – sublingual or intravenous nitrates promptly relieve spasm. For chronic management, calcium-channel blockers (both dihydropyridine and non-dihydropyridine agents) are first-line because they directly inhibit smooth-muscle constriction [7]. Long-acting nitrates (e.g. isosorbide mononitrate) are often added for additional protection. In fact, conventional pharmacotherapy of VSA relies on calcium channel blockers and nitrates. In refractory cases (notably in Japan), Rho-kinase inhibitors (e.g. fasudil) or nicorandil may be used, and high-dose calcium-channel blockers with or without combination therapy are employed. By contrast, β-blockers – especially nonselective agents – should generally be avoided in VSA, since blocking β2-receptors can leave unopposed α-mediated vasoconstriction [1]. Equally important is aggressive lifestyle modification. Smoking cessation is essential, as cigarette use is by far the most potent and modifiable precipitant of spasm. Other risk factors (e.g. uncontrolled hypertension) should be optimized to improve endothelial function. Patients should also be counseled to recognize symptoms early and carry nitroglycerin for prompt relief [6].

Conclusion

Vasospastic angina represents a distinct and potentially reversible form of myocardial ischemia that can mimic obstructive coronary artery disease both clinically and angiographically. Transient coronary narrowing with full resolution following nitrate administration remains a critical diagnostic clue, particularly in patients with compatible risk profiles such as active smoking and hypertension. Accurate recognition of this entity enables the initiation of targeted vasodilator therapy and risk factor control, which are essential for preventing recurrence and improving long-term outcomes.

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