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CASE REPORT

# Recurrent coronary spasm presenting as acute STEMI; To stent or not to stent?

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## Abstract

Coronary artery spasm is a relatively uncommon cause of ST segment elevation myocardial infarction. However, the clinical presentation and electrocardiographic findings are remarkably similar to STEMI secondary to plaque rupture.

Recurrent presentation with coronary vasospasm as STEMI presents a unique clinical dilemma especially if they present to different centers where the previous history is not well known. Management in the acute settings continue to pose significant challenges in these acutely unwell patients.

We describe and explore a similar clinical situation in which a patient with severe recurrent STEMI presentations was found to have coronary artery vasospasms.

The case was twice misinterpreted as having plaque rupture STEMI leading to percutaneous coronary intervention and then further difficulties in long term management in view of need for long term antiplatelet and suspected association of eosinophilia with coronary vasospasm which in turn can be caused by Aspirin.

## Keywords

Coronary Spam, ST elevation MI, Stent, ST Segment

## Introduction

Acute STEMI forms a major proportion of emergency medical presentations and can lead to significant mortality and morbidity if not treated in a timely manner.

One or more of the coronary arteries suddenly closing off is what causes an ST-elevation myocardial infarction. This abrupt cessation of blood flow is typically brought on by coronary artery plaque rupture, erosion, fissuring, or dissection. However, other factors, such as myocarditis, pericarditis, stress cardiomyopathy (Takotsubo), spontaneous coronary artery dissection, different channelopathies, and electrolyte abnormalities, can also result in ST-segment elevations[1]. Acute coronary vasospasm can also result in strikingly similar clinical and electrocardiographic presentation[2].

Coronary artery spasm can induce myocardial ischemia by causing transient and at times critical narrowing of epicardial coronary arteries. A single episode might involve one or more coronary arteries. These patients have generally good outcome[3].

While coronary artery spasm can present in a similar pattern, the management strategy should be different.

## Case presentation

A 45-year-old male was presented to our tertiary cardiac center through the primary percutaneous coronary intervention (PPCI) pathway with chest pain and ECG suggestive of anterior ST elevation MI (STEMI) in the early hours of the day. The limited information available suggested a recent presentation to another local PPCI center and possibility of a stent implantation. He was in cardiogenic shock and was taken direct to the cardiac Cath lab. His initial coronary angiogram showed a subtotal occlusion of LAD, severe lesion in proximal Cx and a patent stent in distal RCA (Figure 1a, 1b and 1c).

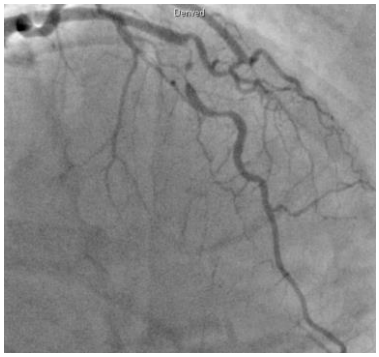

**Figure 1a: Severe stenosis in proximal/mid LAD.**

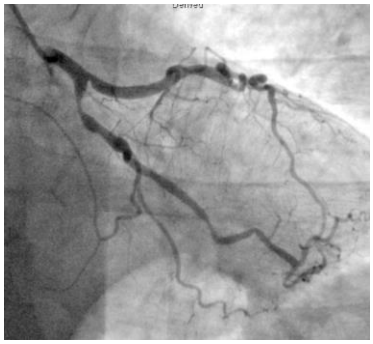

**Figure 1b: Severe stenosis in proximal Cx.**

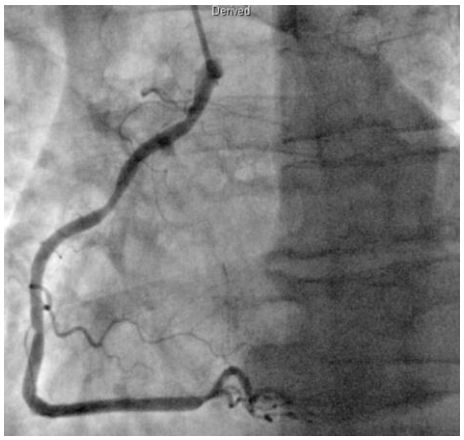

**Figure 1c: Patent stent in distal RCA.**

Patient remained very unwell with a blood pressure of 75/50. The LAD lesion was promptly treated with PPCI to LAD with a single DES, R Onyx 3 X 24 mm with excellent results (Figure 2) and dramatic improvement in the patient's clinical condition. However surprisingly the severe Cx artery lesion was no longer evident.

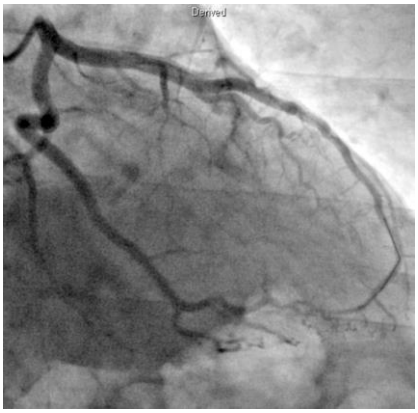

**Figure 2: Post PCI angiogram showing good results from LAD stent.**

Once the acute emergency was treated, the team was able to retrieve the angiographic images from our sister organization. This showed severe stenosis in RCA but unexpectedly completely normal left coronary system (Figure 3a, 3b).

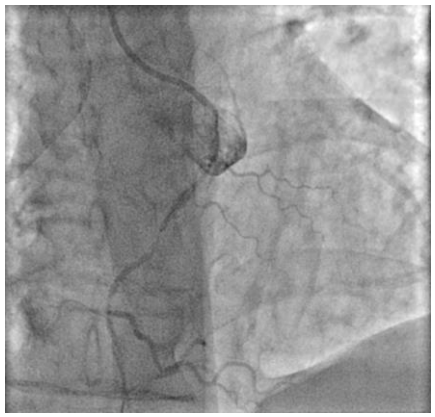

**Figure 3a: Severe lesion in the RCA.**

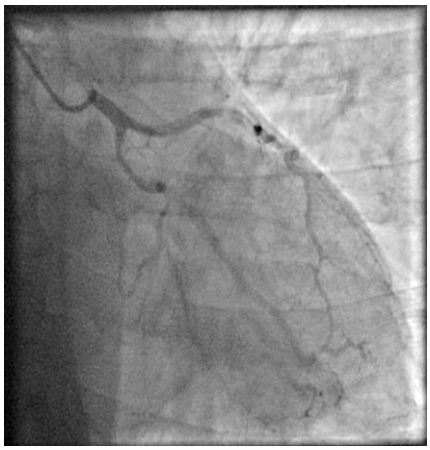

**Figure 3b: Normal left coronary system.**

During that initial presentation the distal RCA lesion was treated with a single DES implantation while the proximal RCA lesion had disappeared by the end of procedure (Figure 4).

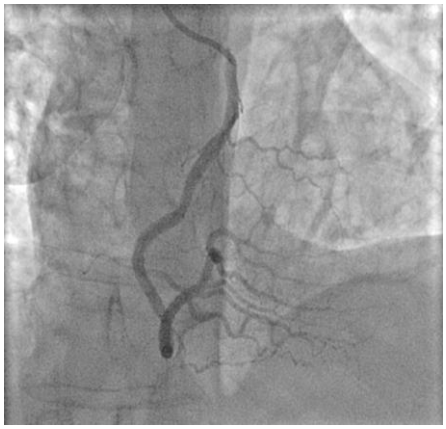

**Figure 4: Post PCI to distal RCA.**

With this knowledge of the 2 procedures a diagnosis of recurrent severe coronary artery spasm was made and clinically documented in patient's notes.

In a further 2 weeks' time, the patient again presented with inferior STEMI. An emergency coronary angiography showed severe ostial and proximal RCA narrowing (Figure 5).

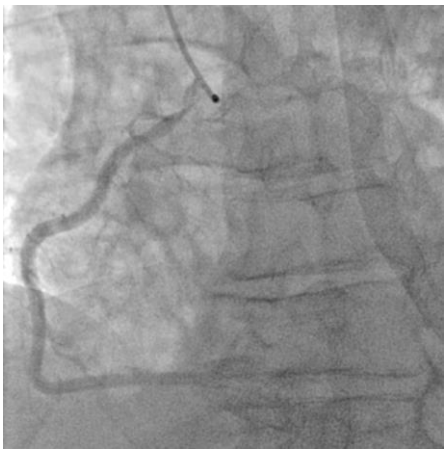

**Figure 5: Coronary angiography showing severe ostial/proximal RCA stenosis.**

With the prior knowledge of the patient condition, he was administered with intra coronary nitrates despite low blood pressure. This led to a dramatic improvement in patient's clinical condition with complete resolution of RCA stenosis (Figure 6).

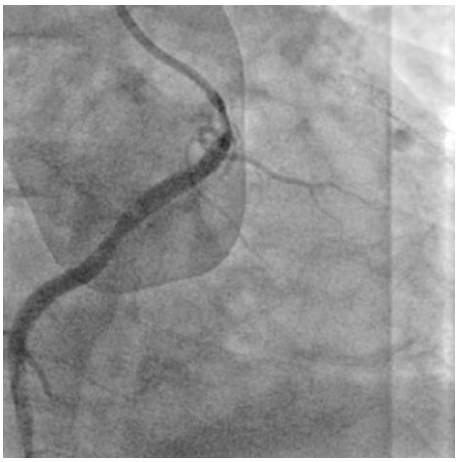

**Figure 6: Complete resolution of RCA lesion after injection of nitrates.**

## Management & Results

With rare presentation and relative lack of evidence in this field, management of coronary spasm presenting as acute STEMI poses a unique challenge. There have only been a few cases of recurrent coronary artery vasospasm reported in literature, and a review of these cases do highlight the difficulty that clinicians face in diagnosis and patients' management presenting with acute STEMI secondary to coronary vasospasm[4,5]. In one extreme case reported by Ahooja et al, the patient ended up having a coronary artery bypass grafting (CABG) following presentation with STEMI secondary to coronary vasospasm[6]. Another case reported by Zhang et all, the patient with multiple coronary artery stenosis on coronary angiography following a presentation with STEMI was planned to have CABG only for the ECG changes to resolve following administration of nitrates and resolution of coronary artery stenosis on repeat coronary angiography[7]. Majority of these cases reported had the first presentation to the hospital with STEMI without any history of coronary artery disease. In contrast, the case reported in our review had

recurrent presentations with STEMI and been acutely and severely unwell.

## Discussion

Our understanding of this acutely life threatening but potentially benign and completely reversible condition remains limited. This is despite the fact that coronary vasospasm as a cause for myocardial ischemia has been studied for a long time. In 1959, Dr. Myron Prinzmetal was the first person to distinguish it as a separate entity, in his article "A variant form of angina pectoris"[8] from the classic Heberden's angina described in 1772[9,10].

### Risk factors and triggers

Three major risk factors for CAS include high-sensitivity C-reactive protein (hs-CRP), smoking, and age[11]. Some studies have suggested that these risk factors may be gender specific, older men who smoke are at increased risk of developing coronary spasm[12,13,14].

The administration of pharmacological agents such as cocaine[15], sympathomimetic agents (epinephrine, norepinephrine, etc.), beta-blocking agents (propranolol, etc.), parasympathomimetic agents (methacholine, pilocarpine, etc.), and ergot alkaloids are examples of the trigger factors for coronary artery spasm[16,17]. Physical or mental stress[18], electrolyte deficiency[19] (ergonovine, ergotamine, etc.). By generating vasoconstrictor chemicals including thromboxane and serotonin, both of which have been linked to CAS, activated platelets may cause CAS[20,21].

### Eosinophilia, coronary spasm and role of Aspirin

Previous investigations have shown that coronary artery spasm was occasionally linked with allergic reactions, such as asthma, in addition to the typical risk factors for vasospasm, such as smoking[22,23] or hyper-susceptibility to specific drugs. For these people, allergen testing may be useful in preventing pointless provocations. Our patient had never abused illegal drugs or had asthma.

This finding can lead to a difficult dilemma regarding use of aspirin in patients with STEMI, as potentially use of Aspirin can lead to vasospasm.

Aspirin-exacerbated respiratory disease (AERD) has been documented and studied[24]. These exacerbations have been noted to be associated with high blood eosinophil count which in turn is associated with coronary artery vasoconstriction[25,26]. In extreme cases, coronary vasospasm can cause complete coronary occlusion leading to acute coronary syndrome and myocardial infarction[27,28] or even fulminant heart failure[29].

## Conclusion

Coronary artery vasospasm presenting as acute STEMI remains relatively uncommon, its timely diagnosis has important implications both for the acute and long-term management. In the acute setting, an accurate diagnosis will prevent the patient been subjected to unnecessary coronary intervention and in extreme case CABG. In the long-term management calcium channel blockers rather than B Blockers appear to be of more benefit. Even more importantly, there are suggestions that avoiding the use of Aspirin might be beneficial. Use of aspirin may make coronary artery vasospasm caused by eosinophilia worse. These individuals who come with ischemic chest pain symptoms should be checked for eosinophilia since prompt corticosteroid treatment can save lives.

Although the prognosis is generally considered to be good, but this depends on accurate and prompt diagnosis in the first place. In summary, as the etiology of acute STEMI secondary to coronary vasospasm is different, the management approach should also be different. A high level of suspicion on the part of attending clinicians and emergency responders may help in early identification and institution of more effective and appropriate management. More work clearly is needed in this

very complicated and rather less understood aspect of acute medical and cardiac emergency.

## Acknowledgment

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