

## CASE REPORT

**BLOCKED CORONARY ARTERY DUE TO CORONARY SPASM TREATED WITH STENT INSERTION: A SUB-OPTIMAL RESULT?****Habib Rehman Khan, Sajid Aslam**

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Myocardial infarction (MI) is an extremely common cause of chest pain. MI can be acute with ST elevation (STEMI) or non-STEMI (NSTEMI). Coronary artery spasm can be severe enough to cause occlusion of the coronary arteries particularly with an exaggerated response in regions of coronary atheroma and plaque ulceration. It is not uncommon for coronary spasm to be mistaken with acute thrombotic occlusion of the coronary artery. We describe a case of a 42-year old man with known cardiac risk factors presents with chest pain for few hours to a tertiary centre hospital. A coronary angiogram showed occlusion of the right epicardial coronary artery with a severe spasm at the level of the atheroma. The initial coronary stent which was deformed because of intense pressure of spasm required another stent to be placed within the first stent. Diltiazem and Nitrates were started as secondary prevention treatment to reduce effect of coronary spasm. The patient made an uneventful recovery and was discharged home with no sequelae over the next 3 years follow up.

**Keywords:** Myocardial infarction; Coronary angiogram; Coronary spasm; Stent

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## INTRODUCTION

Myocardial infarction is a common acute cardiac presentation to cardiac centres in the developing and developed countries around the world. In majority of population, the underlying pathophysiology is rupture of a lipid plaque resulting in thrombotic occlusion of the coronary artery. In some cases, it can be coronary dissection, inflammatory conditions and intense coronary spasm.<sup>1-3</sup> The aetiology of coronary artery spasm is not entirely understood, although literature suggests alteration in endothelial function resulting in impaired nitric oxide release and cytokine release resulting in altered parasympathetic system response. Cigarette smoking and alcohol toxicity have been reported as independent risk factors for coronary artery spasm but the underlying mechanism behind the disease process is not yet defined.<sup>1-4</sup> Treatment depends on identifying coronary spasm and managing it with pharmacological therapies such as vasodilators and in severe cases of disrupted coronary flow, a coronary stent might be required to restore coronary flow.<sup>5,6</sup>

## CASE REPORT

A 42-year-old man was admitted to hospital with severe central crushing chest pain. The chest pain started at 05:15 in the morning, whilst he was making breakfast, and persisted whilst he drove to work. He had been getting intermittent chest pains over the preceding few days. There was no history of any significant medical illness apart from 45 pack-year smoking history. His brother had died in his 30's from a severe asthma attack, possibly also suffering from a myocardial infarction. On admission, his ECG showed minimal ST elevation in an inferior lead pattern, with the development of accompanying small Q waves. His ECG did not meet the criteria for primary coronary intervention and was treated

with dual anti-platelets, heparin and glycoprotein IIb/IIIa inhibitors. However, his chest pain continued and he was taken to the cardiac catheterisation lab. Coronary angiography via right femoral approach revealed no significant obstructive disease in the left coronary artery (LCA) system. The right coronary artery (RCA) was totally occluded at the junction of the proximal and middle thirds (figure-1). Coronary intervention was initiated using a 7 French Judkins right guide catheter with a balanced middle weight (BMW) guide wire. During insertion of the balloon a large degree of coronary spasm was observed (figure-2). The lesion was pre-dilated and a large thrombus burden was detected (figure-3). The right coronary artery was a large dominant vessel with minor distal disease. An FilterWire EZ™ was used for distal protection. A 3.5×20 mm Taxus™ Liberté™ bare metal stent was implanted. Interestingly, there was an intriguing appearance of systolic constriction of the stent at the site of the lesion (Figure-4). This was due to intense vascular spasm at this point of the right coronary artery. The severe spasm responded briefly with intra-coronary nitrates and was causing distortion of the stent. Therefore, a second 4×12 mm Taxus Liberté stent was deployed within the first segment of the RCA.

There was still some constriction observed (Figures-5 and 6) despite deployment of 2 stents but TIMI-3 flow was restored. The patient was stable at this point and the procedure completed. The patient remained stable over the next three days and was discharged on calcium channel antagonists, long acting oral nitrate along with dual antiplatelet therapy. At the subsequent follow up clinic appointment 5 months later, he had remained well and was back at work. His echocardiogram revealed preserved left ventricular function with minor regional wall motion abnormality in the inferior region. There were no hospital admissions for next 3 years on follow up.

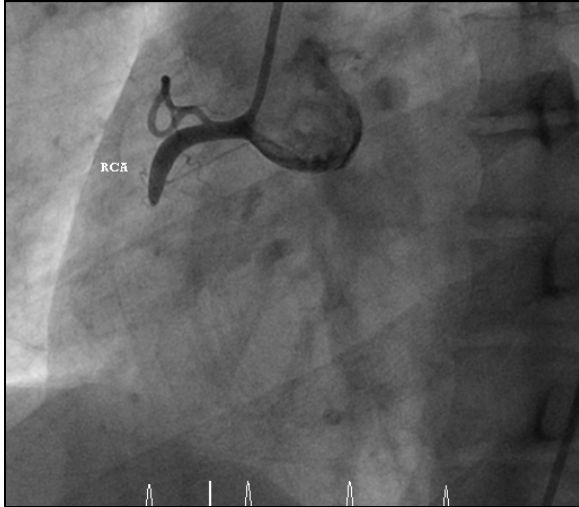


Figure-1: Total occlusion of right coronary artery

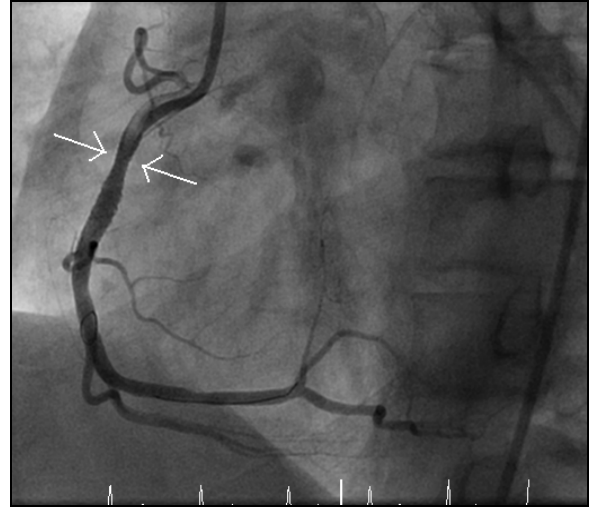


Figure-4: Intense arterial spasm despite stent.

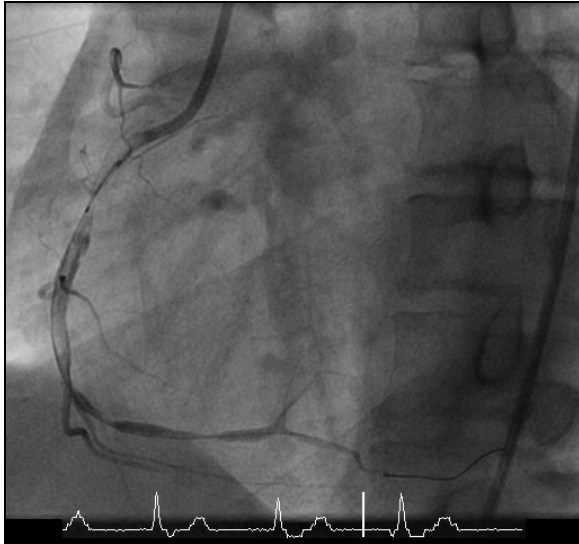


Figure-2: Arterial spasm during balloon insertion.

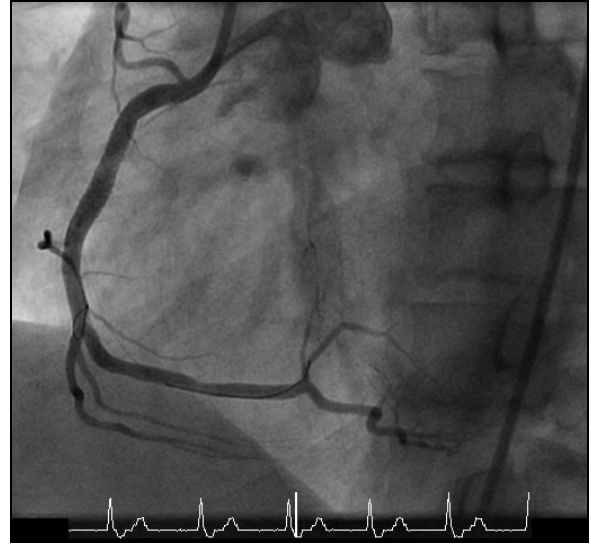


Figure-5: Appearance after 2 stents

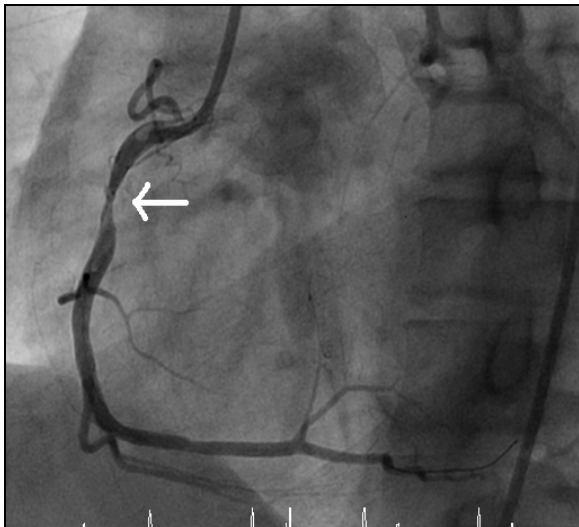


Figure-3: Large thrombus burden observed

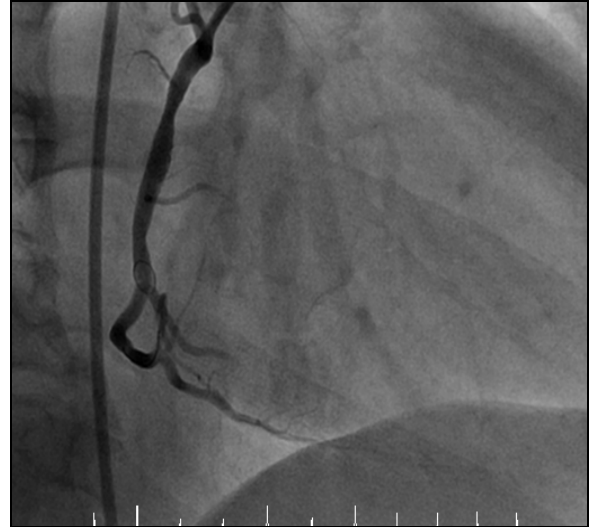


Figure-6: TIMI 3 flow despite ongoing intense spasm

## DISCUSSION

Coronary spasm in both normal coronary arteries or in those with underlying atherosclerotic coronary artery obstruction is known as Prinzmetal angina.<sup>1</sup> Prinzmetal angina is a debilitating condition where patients experience symptoms at rest or with very minimal levels of activity, especially in the mornings. Electrocardiography often shows dynamic ST and T wave changes, including ST segment elevation or depression. In atherosclerotic cases, the spasm usually occurs at or near the site of the disease.<sup>2</sup> Diagnosis of coronary vasospasm is based upon the clinical presentation alongside demonstration of epicardial coronary narrowing that can be reversed by vasodilators on coronary angiography.<sup>3</sup>

Cigarette smoking and alcohol toxicity have been reported as independent risk factors for coronary artery spasm but the underlying mechanism behind the disease process is not yet defined.<sup>4</sup> The leading theory suggests an abnormal response of the endothelium to activation of the parasympathetic nervous system. Under normal circumstances, stimulation of the parasympathetic nervous system leads to release of acetylcholine, which has two actions. Firstly, it causes direct vasoconstriction of the vessels; secondly, it promotes the production of nitric oxide. Nitric oxide usually causes relaxation of vascular smooth muscle, allowing vasodilatation. However, in abnormal endothelium there may be defective release of nitric oxide, giving the net result of unopposed vasoconstriction instead. Therefore, localized contraction of smooth muscle around an atherosclerotic plaque may lead to plaque rupture, platelet aggregation and thrombus formation.<sup>5</sup>

The mainstay of treatment is usually with vasodilators, such as nitrates and calcium channel blockers. However, there are very few trials that have assessed the efficacy of these treatments. There are reported incidences of percutaneous coronary intervention (PCI) being utilized to treat severe cases, with varying degrees of success.<sup>6</sup> However, PCI is not recommended routinely for coronary artery spasm because of the probability that spasm will occur in other parts of the coronary artery anatomy.

In this case report we have presented an occurrence of coronary artery spasm despite stent deployment. We are not aware of any previous cases that have demonstrated coronary artery spasm to such a severe degree. The debate still remains on whether stenting is enough to prevent epicardial narrowing of coronary arteries that are affected by severe spasm.

## REFERENCES

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