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EC CARDIOLOGY
Case Report

Blocked Coronary Artery Due to Coronary Spasm Treated with Stent Insertion: A Sub-Optimal Result?

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Abstract

A 42-year-old man with history of active smoking and possible family history of premature ischemic heart disease presented with chest pain for few hours to hospital. Diagnosis of unstable acute coronary syndrome was made and he was taken to the catheterisation suite after he failed to improve on medical therapy. Coronary angiogram revealed occluded right coronary artery. He had angioplasty to his right coronary artery, and it was noted that he had severe spasm of his right coronary artery at the site of underlying atherosclerotic plaque treated with further coronary angioplasty. We show an interesting case of severe coronary spasm causing acute myocardial infarction and deformation of initial stent requiring further stent insertion to support the scaffold.

Keywords: *Coronary Artery; Coronary Spasm; ECG; ST elevation; Arterial spasm*

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 x 20mm 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 x 12mm Taxus Liberte 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.

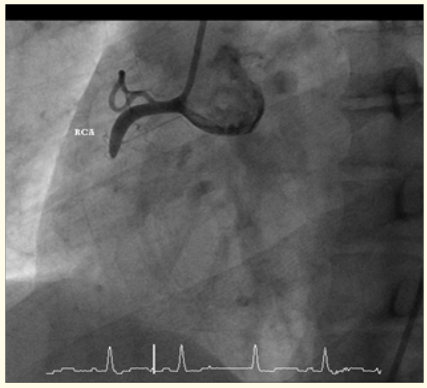

Figure 1: *Total occlusion of right coronary artery.*

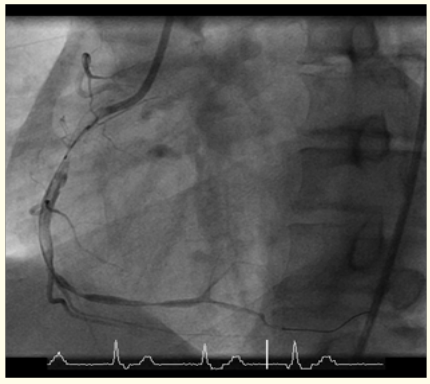

Figure 2: *Arterial spasm during balloon insertion.*

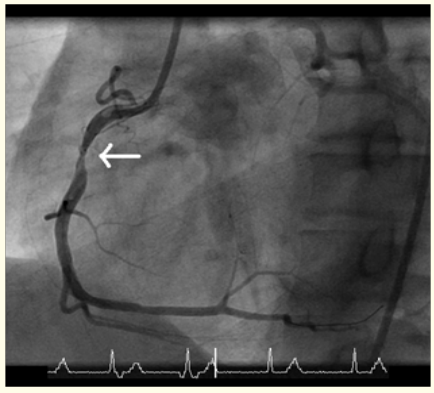

Figure 3: *Large thrombus burden observed.*

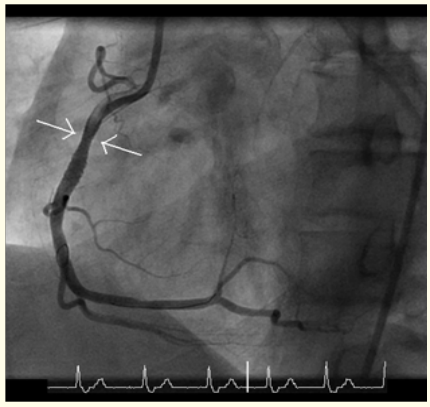

Figure 4: *Intense arterial spasm despite stent.*

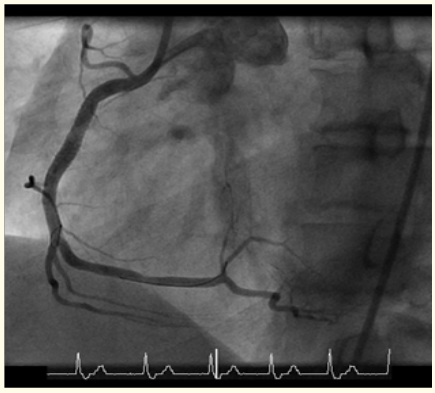

Figure 5: *Appearance after 2 stents.*

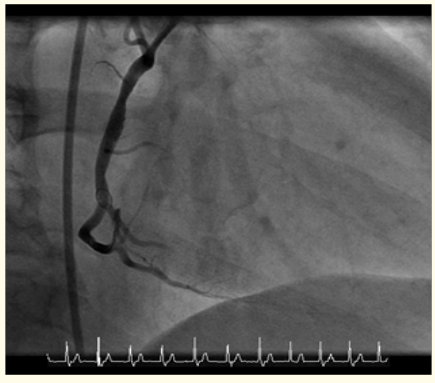

Figure 6: *TIMI 3 flow despite on going intense spasm.*

Discussion

Coronary spasm in both normal coronary arteries or in those with underlying atherosclerotic coronary artery obstruction is known as Prinzmetal angina [1]. Prinzmetal angina is a debilitating condition where patients experience symptoms at rest or with very minimal lev-

els of activity, especially in the mornings. Electrocardiography often show 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 [2]. 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 [3].

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 [4]. 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 [5].

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 utilised to treat severe cases, with varying degrees of success [6]. 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.

Conclusion

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.

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