Case Report

Refractory vasospasm of the left anterior descending artery causing hemodynamic instability after percutaneous coronary intervention

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ABSTRACT

Refractory coronary vasospastic angina is prone to fatal outcomes. Therefore, it is crucial that the most suitable treatment strategy be promptly elected so as to avert further cardiac complications. Here, authors present the clinical course of profound refractory coronary vasospastic angina in a 50-year-old man. Authors elected to manage the patient through stent implantation. Despite, a complication of perforation followed by refractory coronary vasospasm, authors elected to implant a second stent to relieve the patient from all symptoms.

Keywords: Coronary vasospasm, Drug-eluting stent, Percutaneous coronary intervention

INTRODUCTION

The phenomenon of coronary vasospasm developing post stent implantation is not an infrequent occurrence.[1] In such cases, sustained relief of vasospastic symptoms is vital, as recurrent episodes of ischemia can result in myocardial infarction (MI), arrhythmia, cardiogenic shock and sudden death.[2]

If the coronary vasospasm remains unresponsive to conventional anti-anginal drugs, this may necessitate treatment with either coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI). The vasospasm may reoccur after CABG surgery.[3]

However, latest generation drug-eluting stents (DES) have transformed the realm of interventional cardiology therefore making PCI a suitable therapeutic option.[4] Here, authors report a case of refractory vasospastic coronary angina successfully treated through stent implantation.

CASE REPORT

A 50-year-old recently diagnosed hypertensive male presented to the emergency room with angina (CCA-II) and associated perspiration. Emergency electrocardiogram (ECG) revealed T-wave inversion from leads V1-V6 (Figure 1). The patient was shifted to the coronary care unit. A loading dose of clopidogrel 600 mg, aspirin 300 mg, and rosuvastatin 40 mg were administered. Low-molecular weight heparin was also started. Troponin I levels (12.5 IU/L) were significantly elevated (normal range: 0.01-0.01IU/L). 2D echocardiography revealed no motion wall abnormalities with good biventricular function of 60% left ventricular ejection fraction (LVEF) and good diastolic function (Grade 1). In view of TIMI III score, the patient was taken for coronary angiography which revealed 90% thrombotic stenosis in mid-left anterior descending coronary artery (LAD) (Figure 2). A 3.5 x 24 mm second-generation DES was implanted in LAD. Immediately after post-dilation with a 3.5 x 15 mm Trek

NC balloon, the patient experienced severe chest pain. Check angiography revealed perforation draining into the pericardium with hypotension and cardiac tamponade (Figure 3).
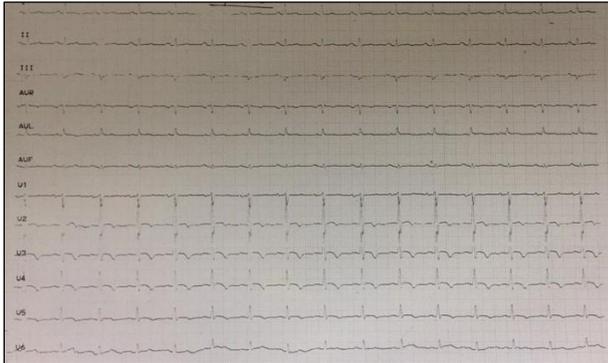

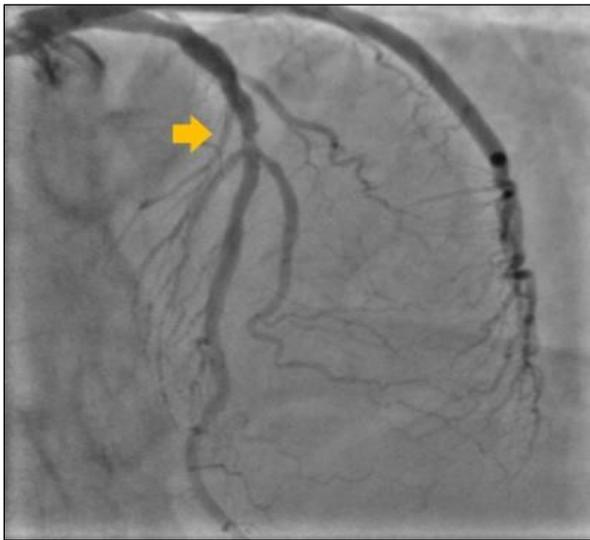**Figure 1: Electrocardiography on admission.**

Figure 2: In coronary angiography 90% thrombotic stenosis in mid-left anterior descending coronary artery.

Multiple balloon dilatations were given with a 3.5 x 15 NC balloon, simultaneously pericardiocentesis was done by subxiphoid approach and 150 ml fluid was drained. A 5F pigtail was inserted and kept in the pericardial cavity. Check angiography after 15 minutes showed no leakage/oozing at the perforation site. 2D echocardiography revealed no pericardial effusion. The patient was shifted to the coronary care unit. Two hourly pericardial fluid aspiration was performed. The patient was hemodynamically stable between intervals with no aspirate through the pigtail and no pericardial effusion detected on 2D echocardiography. The patient was clinically and hemodynamically stable for the next 14 hours.

After 14 hours the patient experienced severe angina, bradycardia, hypotension and unresponsiveness. ECG

revealed giant ST-elevation in leads I, aVL, V1-V6. (Figure 4). He was immediately intubated and placed on ventilatory and inotropic support.
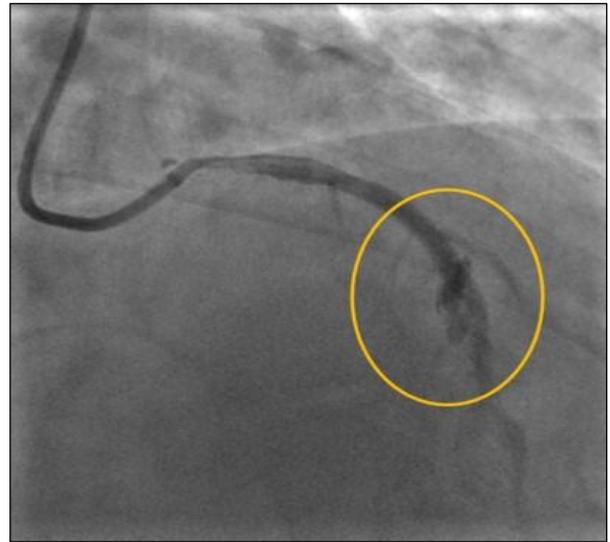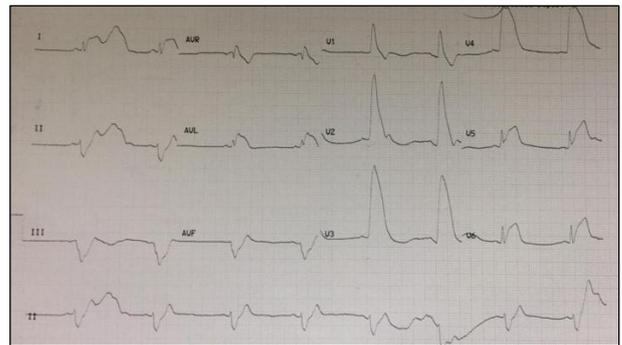

Figure 3: Coronary angiography perforation.

Figure 4: In electrocardiography acute myocardial infarction post first percutaneous coronary intervention.

Figure 5: Coronary angiography for left anterior descending artery proximal spasm.
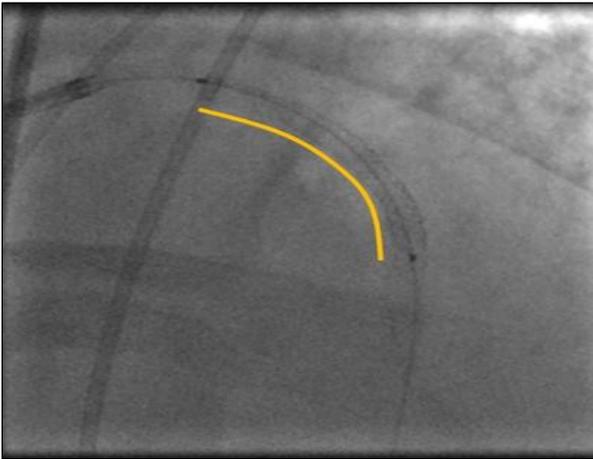
Figure 6: Coronary angiography deployed 3.5 x 37mm drug-eluting stent.

Figure 7: In coronary angiography post stent TIMI III flow.

He was immediately shifted to the cath lab. Echocardiographic screening revealed hypotension in LAD territory with depressed left ventricular systolic function (30% LVEF) and no pericardial effusion.

Coronary angiography revealed patent mid-LAD stent with no leakage from the perforation site but significant spasm of proximal LAD from the ostium with distal TIMI II flow (Figure 5). The spasm was refractory to intracoronary nicorandil, nitroglycerine adenosine and nitroprusside. Due to refractiveness of the spasm and hemodynamic instability, the decision to stent proximal LAD was made and PCI with a 3.5 x 37 mm second-generation DES was performed along with post dilatation (Figure 6). TIMI III flow was achieved (Figure 7). The patient required ventilatory and inotropic support for the next 72 hours with aggressive critical care management for acidosis, dyselectrolytemia, and minor bleeding. However, LVEF persistently remained in the range of 30-35%. The patient was shifted to intermediate care after 96 hours. There was improvement in LVEF to 40-45% prior to discharge. ECG showed evolved anterior wall myocardial infarction at the time of discharge (Figure 8).
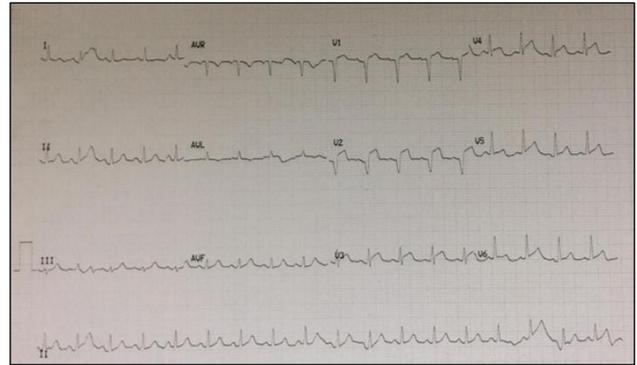

Figure 8: Electrocardiography evolved anterior wall myocardial infarction at the time of discharge.

DISCUSSION

Intracoronary nitroglycerin is the conventional treatment of coronary vasospasms. However, in many cases the vasospasm fails to respond to medical therapy, presenting as refractory vasospasm, as observed in our patient. In the present unusual case, post stent implantation, the patient suffered perforation along with hypotension and cardiac tamponade. Pericardiocentesis was performed and hemodynamically stability was achieved, however the patient suffered coronary vasospasm in proximal LAD. Due to the lack of response with medical therapy, authors opted to place a coronary stent at the site of the refractory coronary vasospasm.

Current literature, evidences treatment of coronary vasospasms with PCI as a controversial topic owing to reports of both successful and unsuccessful experiences. Azar et al, concluded inefficiency of stenting due to development of diffuse vasospasm involving the entire left coronary circulation post bare-metal stent (BMS) implantation.[5] Shibutani et al. suggested subacute stent thrombosis secondary to DES-induced coronary vasospasm whilst warning against stent-edge spasm post stent implantation.[6] Kapoor et al, reported cardiac arrest and multivessel spasm of the entire right and left coronary circulatory systems post PCI.[1] The review by Brott et al, highlighted 13 cases of spasm arising post stent placement, two of which culminated into death from cardiogenic shock and intracranial bleeding after aggressive anticoagulation.[2] Two other multivessel diffuse spasms necessitated intervention with intra-aortic balloon pump placement due to severe ischemia or shock.

However, not all cases have resulted in unfavorable outcomes. Brott et al, and Jeong et al, documented successful treatment of coronary vasospasm through stenting. Furthermore, Khan et al, reported an interesting case of stent deformation due to high vasospastic pressure.[2,3,7] As reported in this case, their rescue strategy also involved the implantation of a second stent. In their case, the second stent was implanted within the first stent. Hokimoto et al, have published real-world data on prevalence of cardiovascular events related to coronary

spasm following PCI according to stent type.[8] Their findings have revealed 0.4% prevalence with second-generation DES as compared to 2.9% and 3.2% for BMS and first-generation DES, respectively. These findings indicate second-generation DES to be safer and more efficacious regarding cardiac events induced by coronary vasospasm. This data may to some extent explain the conflicting outcomes with PCI. In our patient, authors had opted for second-generation DES both times.

Coronary vasospasm is not a benign entity and can have a fatal outcome. A correct and timely diagnosis of refractory coronary vasospasm will reduce patient exposure to inappropriate therapies as well as the risks to Major Adverse Cardiac Events (MACE). Furthermore, it is of paramount importance to select the most suitable treatment strategy that will avert serious sequela, including possible long-term morbidity and mortality.

CONCLUSION

Authors conclude that coronary stenting is the only option to refractory coronary spasm especially in cases of proximal vessel involvement in patients with hemodynamic instability, depressed left ventricular systolic function and primary percutaneous coronary intervention.

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