

**Case Report** 

# A RARE COMPLICATION: CORONARY ARTERY SPASM AFTER DRUG ELUTING STENT IMPLANTATION DURING PERCUTANEOUS INTERVENTION LEADING TO ST ELEVATION MYOCARDIAL INFARCTION( STEMI).

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## Author's Contribution

### All authors declare no conflict of interest.

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## ABSTRACT:

Coronary artery spasm is define as constriction of a coronary artery segment which may be focal or diffuse, may bein single or more vessels which is reversible and causes myocardial ischemia by restricting coronary blood flow, leading to thrombus formation. we present a case of coronary artery spasm after drug eluting stent implantation during percutaneous intervention (PCI) leading to ST Elevation Myocardial Infarction (STEMI), which is a rare complication. I

KEYWORDS: Percutaneous coronary intervention, spasm, stent, ST Elevation Myocardial Infarction, complications

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#### **INTRODUCTION:**

ercutaneous coronary intervention (PCI) is considered as an invasive modality for treatment of coronary artery disease.<sup>1</sup> PCI can be usually performed successfully without any complications but few complications can be encountered. Although these complications occur in 1-2% of patients and could be fatal.<sup>2</sup> Coronary artery spasm is an important cause of chest pain. Chest pain after successful PCI constitute a considerable problem and some time fatal if caused by myocardial infarction. Such pain constitute either residual coronary stenosis, acute occlusion, spasm or myocardial infarction. The management of such kind of complications involve repeat coronary angiography and some additional interventions. We present a case report "Coronary artery spasm after drug eluting stent implantation during percutaneous coronary intervention (PCI) leading to ST Elevation Myocardial Infarction (STEMI)" which is a rare complication.<sup>3</sup>

### CASE REPORT

A 65 year old female non diabetic, normotensive non somker presented to emergency department(ED) with two days history of retrosternal chest pain, sudden in onset ,severe in intensity, radiating to left arm and jaw but not to interscapular region, partially relieved with rest, associated with sweating and was not related to food intake, body movements and respiration.

On relevant physical examination patient has average built and height with pulse 60/min ,regular , B.P 130/80 mm of Hg and Respiratory rate 16/ min. Systemic examination was unremarkable.

Her ECG done which showed ST-T changes in lead III and AVF as in Fig.

Troponin i level was raised 1.29 ng/dl

Other relevant investigations were within normal limit.

Patient was loaded with Clopidogrel , aspirin, heparin and then on maintenance dose of these medications. Patient symptomatically improved with these medications after 4 hours. Patient was subjected to coronary angiography through right femoral approach which showed tight focal proximal stenosis and normal other LAD and ICX.

PCI to RCA was done with 3X12 Everolimus –Eluting Stent deployed at 14 atm with final TIMI III flow and post procedure ACT was 540s.Whole procedure was complications free.

After two hours post PCI patient developed chest pain with sweating, relevant examination was

unremarkable with no femoral access site complications. Serial ECGs done with no significant ST T changes in first ECGs but 2nd and 3rd ECG showed ST elevation in inferior leads

Patient was shifted to cath lab where her check injection was done through right ulnar approach, which showed patent stent followed by severe diffuse spasm at stent outlet with intra luminal





Post stent implantation







Post PCI ECG.







haziness and thrombus in proximal PDA, as shown in fig.

It was decided to put patient on iv nitroglycerine for 12 hours and tirofiban (aggrastat) infusion according to body weight for 24 hours, she became asymptomatic after 3 hours followed by serial ECGs. After nitroglycerine infusion for 12 hours she was put on oral nitroglycerine and amlodopine





along with other medications.

After 3 days she was subjected to another check injection through right femoral approach which showed patent stent followed by mild mid disease with distal TIMI III flow. There was no evidence of intra luminal haziness or thrombus.

#### DISCUSSION

Although drug eluting stent(DES) reduces restenosis more effectively than bare metal stents (BMS) after percutaneous coronary intervention(PCI), there are variety of DES related complications such as coronary artery spasm.<sup>4</sup> The degree of vasoconstriction during a spasm ranges from clinically undetectable to complete occlusion. Coronary spasm is most often seen clinically in patients above 50 years old and decreases as age advances . Female smokers suffer most frequently from coronary artery spasms. The first clinical suggestion is that DES implantation can induce new-onset stent-edge spasm. <sup>5</sup> Some reports have suggested endothelial dysfunction and enhanced vascular

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smooth muscle contractility with the involvement of the Rho-kinase pathway play an important role in DES induced coronary artery spasm. Coronary vasoconstrictive responses are more at the edges of coronary artery segments implanted with DES compared with BMS.<sup>6</sup> It is possible that repeated, broad and long stent-edge spasm results in new cardiac events i.e myocardial infarction and death was reported in some patients with severe multivessel, non-intervention-related vascular spasm after DES implantation, adjunctive medical treatment is critical for these patients Inability to administer to adjunctive medical treatment for DES related coronary artery spasm will promote coagulation system and thrombus formation which may result acute ST Elevation Myocardial Infarction(STEMI),. One can give intra coronary nitrates, iv nitrates and tirofiban. Long term administration of vasodilating drugs such as calcium channel blockers and nitrates are useful for coronary vasospasm.<sup>7</sup>

The second clinical suggestion is that stent-edge spasm may have an important role in the development of acute and sub acute Stent thrombosis. There are two possible reasons for this phenomenon. The first is that coronary vasospasm promotes coagulation and may lead to thrombus formation. Some studies focused on the relationship between coronary vasospasm and thrombus formation have observed accelerated platelet and coagulation activities induced by coronary artery spasms which

may result in acute myocardial infarction. Several cases in which coronary vasospasm led to coronary thrombosis and myocardial infarction have also been reported, supporting a potential mechanism underlying the phenomenon of the stent-edge spasm-related acute and sub acute stent thrombosis (ST). The second possible reason for the phenomenon of acute and sub acute ST secondary to stent-edge spasm is few ST risk factors like the CYP2C19 polymorphism, non or weak responsive to Clopidogrel, factors related to devices, patients, lesions, and procedure have been suggested in the development of ST. Therefore, we believe that coronary vasospasm became apparent after DES implantation and that stent-edge spasm should have been treated with anti vasoconstrictive drugs to control and prevent the occurrence of acute and sub acute ST.<sup>8</sup>

#### CONCLUSION:

Coronary artery spasm after DES implantation leading to ST elevation myocardial infarction can be acutely treated with intra coronary nitrates, intra coronary tirofiban, intra venous nitrates and tirofiban. Long term management includes oral nitrates and calcium channel blockers along with other routine medications.

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