

## ST Elevation Myocardial Infarction due to Cocaine Induced Coronary Vasospasm

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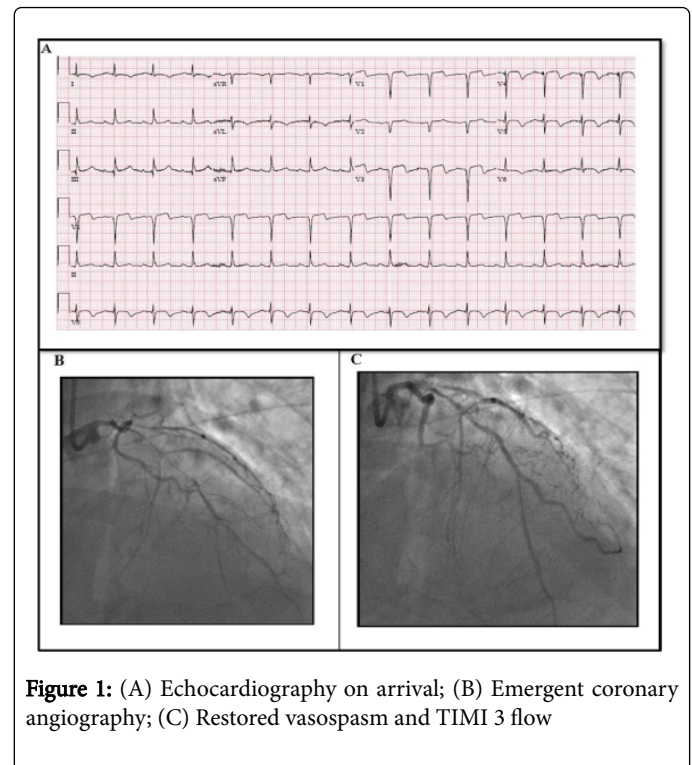
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Received date: September 24, 2015; Accepted date: September 28, 2015; Published date: September 30, 2015

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### Case Study

A 59-year-old man with medication-treated hypertension, non-insulin dependent diabetes mellitus and a current tobacco user, presented with substernal pressure-like chest pain of one hour duration, prior to arrival to the emergency room. Electrocardiography on arrival (Figure 1A) showed anterior ST segment elevations with subsequent anterior T-wave inversions. Initial troponin concentration was 72.58 ng/mL (reference range: 0.01-0.04 ng/mL). He underwent emergent coronary angiography, which revealed 50% mid-left anterior descending (LAD) artery stenosis with severe vasospasm involving the entire LAD segment with TIMI 1 flow down the artery (Figure 1B). Intra-coronary nitroglycerin administration resolved the vasospasm and TIMI 3 flow was restored (Figure 1C). Subsequent echocardiography showed a depressed left ventricular ejection fraction to 40% with wall motion abnormalities consistent with a LAD territory infarct. Urine toxicology screen showed evidence of cocaine and the patient eventually admitted to using cocaine prior to the onset of the chest pain. He was eventually discharged after being medicated with calcium channel blockers and nitrates. He was also started on carvedilol, aspirin, atorvastatin, lisinopril and spironolactone for management of his ischemic cardiomyopathy and concomitant atherosclerosis. Prior evidence suggests the safety of non-selective beta-blockade in the setting of cocaine use which was demonstrated in our case [1-4]. The patient was counseled extensively about abstinence from illegal drugs.



**Figure 1:** (A) Echocardiography on arrival; (B) Emergent coronary angiography; (C) Restored vasospasm and TIMI 3 flow

### References

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