

Seventy-five Year Old with Cocaine- induced Thrombus in a Drug Eluting Stent

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Citation

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Abstract

Cocaine abuse is widespread and is known to cause myocardial infarction by several different mechanisms. Compliance with medical instructions and medications is poor in patients using cocaine. We report a case of 75 year old cocaine user who developed late stent thrombosis. This patient who already had a DES with prior compliance with dual anti-platelet therapy developed an in-stent thrombus that was treated with a thrombectomy and new DES. Given the issue of compliance, and the increasing evidence of cocaine induced thrombus in native vessels and stents it is recommended that angioplasty alone should be the treatment of choice when intervention is required.

CASE REPORT

While cocaine has been used by over 11% Americans, it is very remarkable and unusual to see its use in a patient 75 years old. Cocaine is known to cause myocardial infarction and several different mechanisms are responsible.²

Regardless of the route of administration and the amount used, cocaine can cause increased oxygen demand by increasing wall tension, heart rate and contractility. Cocaine is also associated with thrombus formation caused by platelet aggregation. Patients with documented cocaine abuse and resultant myocardial infarctions have been reported and many patients have been treated with angioplasty and either bare metal or drug eluting stents. There are no prospective studies or evidence for which treatment is best. Compliance with anti-platelet medications is an issue, and it has been difficult to determine the role cocaine plays in the pathophysiology of the infarctions occurring in these patients. We present an unusual case of a 75 year old male with a prior drug eluting stent that had been placed two years prior to his current admission.

A 75 year old male with multiple risk factors for coronary artery disease had three previous drug eluting stents placed several years prior to this current admission. He had been compliant with dual anti-platelet therapy including both aspirin and clopidigrel for the recommended period time of 1 year. Inexplicably, he began to use crack cocaine and presented to the emergency room about one hour after having smoked cocaine with an Inferior STEMI. Urine was

positive for cocaine. Immediate cardiac catheterization was performed and a thrombus in a drug eluting stent to the right coronary artery was noted. A thrombectomy was done and a new bare metal stent was placed. The patient had an uneventful recovery and was discharged on dual anti-platelet therapy once again.

Drug eluting stents have reduced in stent stenosis significantly compared to bare-metal stents. However, problems with late in-stent thrombosis remain an issue which requires dual anti-platelet drug therapy with aspirin and clopidigrel for not less than one year. Our patient was compliant with his medications for the required one year following his initial stents. Thereafter, he inexplicably started smoking crack cocaine and developed a thrombus in a DES. Approximately 6% of patients presenting to the ER with cocaine associated chest pain have enzymatic evidence of myocardial infarction. About one half of the patients with cocaine-related myocardial infarction have no evidence of atherosclerotic coronary artery disease by subsequent angiography.² Most likely, this represents a supply and demand mismatch as a cause of the infarction. Cocaine may induce thrombus formation in the coronary arteries or in stents even in patients who appropriately take dual anti-platelet therapy for the recommended time, as evidenced by our patient. Enhanced platelet activation and aggregability, as well as an increase in the concentration of plasminogen-activator inhibitor which may promote thrombus formation have been previously reported³. Recently a report of 4

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patients⁴ with bare metal stents, and documented cocaine abuse, developed in stent thrombosis without re-stenosis. Non-compliance notwithstanding, it was felt that stent thrombosis could be ten times greater in patients using cocaine compared to non users⁵. We add to this cohort another patient with documented compliance with aspirin and clopidigrel, according to the current guidelines, in whom in-stent thrombosis also occurred. This would seem to suggest that thrombus formation secondary to cocaine is a likely cause of infarction especially in stents, regardless of the type of stent and regardless as to proper anti-platelet therapy or its duration of use. Given the number of patients with stents, cocaine usage and the issue of compliance, it seems reasonable to intervene when necessary with angioplasty only, obviating the need for anti-platelet therapy, or using bare metal stents, which shortens the time of anti-platelet therapy to only one month. Since a prospective study is not feasible, and not to increase the risk further of in stent

thrombosis with a drug eluting stent at this time it is our feeling that these patients should be treated with angioplasty only.

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