CASE REPORT

Cocaine-induced very late stent thrombosis

Priyank Shah,¹ Rahul Vasudev,² Ahmad Hisham Abuarqoub,³ Faye Shamoon¹¹Department of Cardiology, New York Medical College at St Joseph's Regional Medical Center, Paterson, New Jersey, USA²Department of Internal Medicine, New York Medical College at St Joseph's Regional Medical Center, Paterson, New Jersey, USA³Department of Internal Medicine, Saint Michael's Medical Center, Newark, New Jersey, USA**Correspondence to**Dr Rahul Vasudev,
drahulvasudev@gmail.com

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SUMMARY

Cocaine misuse is a known cause of acute coronary syndrome (ACS). Management of these patients has always been a challenge due to medication compliance and eventual risk of stent thrombosis. However, even cocaine misusers who are compliant with dual antiplatelet therapy have been reported to have stent thrombosis. All cases of cocaine-induced stent thrombosis reported in the literature have occurred within first year of stent placement (acute, subacute or late). We report a first case of very late stent thrombosis in a 54-year-old active cocaine misuser who presented with ST segment elevation myocardial infarction, which was successfully managed with percutaneous transluminal coronary angioplasty. A review of all the reported cases of cocaine-induced stent thrombosis is also discussed. Given the high mortality associated with stent thrombosis, treatment option for cocaine misusers presenting with ACS should be conservative when possible. If percutaneous coronary intervention is needed, bare metal stent should be preferred.

BACKGROUND

Cocaine use is a well-documented and increasingly a common cause of acute coronary syndrome (ACS) in the USA.¹ The current guidelines for management of ischaemic pain after cocaine use recommend immediate coronary angiography in patients with persistent ST segment elevation despite nitrate and calcium antagonist therapy. Additionally percutaneous coronary intervention (PCI) is a class 1 recommendation in these patients if an occlusive thrombus is detected during angiography.^{1–2} But the potential adverse effects of continued cocaine use raises concern for patients who underwent PCI. Cocaine-induced stent thrombosis despite optimal dual antiplatelet therapy has been reported.^{3–8} Only survivors are reported in the relatively sparse literature, and therefore the true scale of the problem may be an underestimate. All reported cases of cocaine-induced stent thrombosis in the literature, thus far have either been acute, subacute or late stent thrombosis. We report a case of very late stent thrombosis (VLST) due to cocaine misuse in a 54-year-old patient. Also, a literature review of cocaine-induced stent thrombosis is discussed.

CASE PRESENTATION

A 54-year-old African-American man with medical history of coronary artery disease with stent placement in the left anterior descending (LAD) and diagonal artery (in 2010), ischaemic cardiomyopathy status postimplantable cardioverter defibrillator (ICD) placement (in 2012), hypertension, dyslipidaemia, well-differentiated rectal carcinoid tumour

(diagnosed July 2014), tobacco and cocaine misuse, came to the emergency room (ER) after an episode of sudden onset severe pressure like substernal chest pain radiating to his left arm in November 2014. He did not have any associated symptoms. The patient took ranolazine and sublingual nitroglycerine, which did not alleviate the pain, so he came to the ER. He also admitted to cocaine use 1 day prior to presentation. The patient stated that he was compliant to all his medications (aspirin, atorvastatin, lisinopril, carvedilol, ranolazine, sublingual nitroglycerine (as needed)). He took clopidogrel for more than 3 years after the stent was placed in 2010 but stopped taking it 6 months ago after discussing with his cardiologist. Previous stents were implanted at a different hospital and this information could not be obtained as the patient was unable to provide the hospital details.

On arrival to ER, his blood pressure was 126/84 mm Hg, heart rate was 80 bpm, temperature was 97.3°F, respiratory rate was 19/min and saturation was 100% on room air. His physical examination was unremarkable except for ICD felt in the left upper chest area. ECG revealed normal sinus rhythm with ST segment elevation in leads V2 to V5 (figure 1). The patient was taken to the cardiac catheterisation laboratory for ST segment elevation myocardial infarction (STEMI).

Coronary angiography showed stent thrombosis of stents in the mid-LAD and second diagonal (figure 2, video 1). There was also about 60% stenosis in the first obtuse marginal. Aspiration thrombectomy was performed multiple times followed by balloon angioplasty (maximum inflation up to 20 atmospheres) of the mid-LAD and second diagonal with no residual stenosis and TIMI-3 flow (figure 3, video 2). Optical coherence tomography (OCT)/Intravascular ultrasound was not performed given that the patient presented as STEMI, high pressure inflation was carried out postprocedure to ensure stent apposition.

The patient developed accelerated idioventricular rhythm transiently during the procedure, most likely indicating reperfusion arrhythmia. Left ventriculogram showed severe hypokinesia of mid-distal anterior, anteroapical and inferoapical segments of the left ventricle with ejection fraction of about 35%. Eptifibatide bolus was given, infusion was started and he was transferred to coronary intensive care unit. Eptifibatide was continued for 18 hours. Echocardiography showed reduced ejection fraction of 30–35% with severe hypokinesia of mid-distal anterior, anteroapical and inferoapical segments of the left ventricle.

Ticagrelor was added to his medication regimen and other medications except carvedilol were continued.

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Figure 1 ECG on arrival showing sinus rhythm with ST segment elevation in leads V2–V5.

OUTCOME AND FOLLOW-UP

The patient had an uncomplicated hospital course. He was counselled in detail regarding complications associated with persistent use of cocaine and reinforced to stop using it. He was also counselled on smoking cessation and was discharged home in stable condition on day 3. The patient was seen once postprocedure in outpatient clinic and latter followed up with cardiology team near his home as our centre was not convenient for him.

DISCUSSION

We did an extensive literature review on cocaine-induced stent thrombosis. We searched PubMed for this literature review using search terms ‘cocaine and stent thrombosis’, ‘cocaine and myocardial infarction’, and ‘cocaine and acute MI’. All the case reports and major case studies on cocaine misusers who presented with stent thrombosis so far were reviewed. We abstracted all the relevant data from these studies. None of these cases presented with VLST.

Cocaine is the most commonly misused drug by patients presenting to the ER. It accounts for 40% of ER visits in the USA.⁹

The national survey of drug use and health estimated 1.9 million active cocaine misusers in the USA in 2008.¹⁰ Stent thrombosis is serious concern with cocaine use. The risk of stent thrombosis is 10 times higher in cocaine misusers compared with non-misusers.^{5 11} Almost 15–30% of patients with stent thrombosis die within 30 days of the event, reflecting the importance to understand the risk of stent thrombosis in cocaine misusers.¹¹

A total of 16 cases of cocaine-induced stent thrombosis have been published so far in the English literature. Table 1 summarises all the reported cases of cocaine-induced stent thrombosis.^{3–8} Stent thrombosis in all these cases was either early (acute/subacute) or late, but none of them were VLST as described in our case. Among these reported cases of cocaine-induced stent thrombosis, 13 (81.25%) of them presented with STEMI, while the rest n=3 (18.75%) presented

Figure 2 Coronary angiogram in right anterior oblique cranial view showing stent thrombosis and 100% occlusion of mid-left anterior descending and second diagonal (as shown by the arrow).

Video 1 Video of coronary angiogram in right anterior oblique cranial view showing stent thrombosis and 100% occlusion of mid-left anterior descending and second diagonal.

Figure 3 Coronary angiogram in right anterior oblique cranial view after aspiration thrombectomy and balloon angioplasty showing re-established flow in left anterior descending and second diagonal (as shown by the arrow).

Video 2 Video showing coronary angiogram in right anterior oblique cranial view after aspiration thrombectomy and balloon angioplasty re-established flow in left anterior descending and second diagonal.

with non-STEMI. A total of seven patients (43.75%) presented as early stent thrombosis and the remaining nine patients (56.25%) presented as late stent thrombosis. Out of the 16 patients, only 1 patient was treated conservatively, 1 had coronary artery bypass graft (CABG) and the rest were managed with percutaneous transluminal coronary angioplasty (PTCA) and/or stent. Balloon angioplasty is almost always the treatment of choice for cocaine-induced stent thrombosis. In our literature review, 10/16 (62.5%) patients were treated with PTCA alone. Of all the 16 patients, only 1 patient (the patient who underwent CABG in Singh *et al*⁵ case study) died in the postoperative period.

Stent thrombosis in all studies occurred more in cocaine misusers but this could be attributed to poor compliance to medications and instructions. However, there have been reported cases of stent thrombosis in cocaine misusers despite compliance to the dual antiplatelet regimen. The mechanism of cocaine-induced stent thrombosis is multifactorial. Cocaine exposure causes platelet activation, platelet containing microaggregate formation, modulation of thromboxane levels and possibly impaired stent endothelialisation.^{12–14} Long-term cocaine use is also known to be associated with high basal levels of circulating platelets that have undergone α -granule release. Cocaine also increases plasminogen activator inhibitor activity, fibrinogen and von Willebrand factor levels.

VLST after drug eluting-stent (DES) placement is still a concern. Although infrequent, it is a catastrophic complication, which can lead to myocardial infarction and sudden cardiac death. The mechanism of VLST in DES is unclear. In a report studying VLST in sirolimus-eluting stents, three different mechanisms were explained using coronary angiography (CAS) and OCT.¹⁵ CAS and OCT showed uncovered and malapposed stent struts at the culprit lesion. Late stent malapposition is caused by stent underexpansion and thrombus resolution or by positive vessel remodelling.¹⁵ Delayed neointimal coverage over stent struts and late stent malapposition are thought to be associated with VLST in patients treated with DES.¹⁵ Mechanism behind VLST in cocaine users who has DES is still unexplained. However, it is possible that our patient had underlying nidus (malapposed stent due to positive remodelling and/or delayed neointimal coverage) and cocaine use triggered stent thrombosis since he presented a day after cocaine use.

Table 1 Summarising case studies and case reports of cocaine-induced stent thrombosis

Case study/ case report	Number of patients	Number of CA	CA with ST	Number of patients in control group	ST in non-CA/ control	BMS in CA with ST	CA with ST compliant to DAPT	ST (AC/SA/LA)	Clinical picture at ST	Outcome in CA with ST(PTCA/ CABG/MED)
McKee (CS)	71	71	5 (7.6%)	3216	0.6%	3	3	SA—2 LA—3	NSTEMI—1 STEMI—4	PTCA—5
Karlsson (CS)	247	12	4 (33%)	235	2(0.85%)	4	4	SA—1 LA—3	STEMI—4	PTCA—2 PTCA with stent—2
Singh (CS)	1125	81	4 (5%)	1044	6(0.57%)	4	0	AC—1 SA—1 LA—2	NSTEMI—2 STEMI—2	PTCA—2 CABG—1 MED—1
Makaryus <i>et al</i> (CR)	1	1	1	NA	NA	1	1	AC	STEMI	PTCA with stent
Morhaf Ibrahim (CR)	1	1	1	NA	NA	1	1	SA	STEMI	PTCA
Chait (CR)	1	1	1	NA	NA	0	1	LA	STEMI	PTCA with stent

AC, acute; BMS, bare metal stent; CA, cocaine misusers; CABG, coronary artery bypass graft; CR, case report; CS, case study; DAPT, dual antiplatelet therapy; LA, late; MED, medical management; NA, not applicable; NSTEMI, non-ST elevation myocardial infarction; PTCA, percutaneous transluminal coronary angioplasty; SA, subacute; ST, in stent thrombosis; STEMI, ST elevation myocardial infarction.

Learning points

- ▶ Stent thrombosis is associated with significantly increased morbidity and mortality.
- ▶ The risk of stent thrombosis is 10 times higher in cocaine misusers compared with non-misusers. The presentation in most cases is either early or late stent thrombosis. However, very late stent thrombosis can occur as described in our case.
- ▶ Also patients with cocaine-induced stent thrombosis usually present as ST segment elevation myocardial infarction. The treatment option in cocaine misusers presenting with acute coronary syndrome should be conservative when possible. In these patients thrombus aspiration and/or GPIIb/IIIa inhibitors as the preferred treatment of choice.

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