Acute myocardial infarction in a young cocaine addict with normal coronaries: time to raise awareness among emergency physicians

Sir,

Cocaine is a popular drug for illicit use despite its high street price. In the recent past, its use in India is

increasing. Adverse cardiac events related to its abuse or intoxication including accelerated atherosclerosis, hypertension, dysrhythmias, vasospastic angina, rarely myocarditis, myocardial infarction (MI), and sudden death are primarily noted in the Western Countries. Data on Indian patients is sparse.

A 24-year male admitted to ICCU (intensive coronary care unit) with chest pain for preceding 3 h. Electrocardiogram (ECG) revealed extensive acute anteroseptal MI [Figure 1]. He was addicted to cocaine for preceding two months with last inhalation 1 h prior to chest pain initiation. On evaluation, no other coronary risk factors could be delineated except smoking. Qualitative Troponin-T tested positive and CPK-MB (creatinine phosphokinase MB fraction) value was 940 U/L. Thrombolysis was stopped prematurely for gum bleeding. Other management including benzodiazepine relieved his chest pain. Echocardiography revealed hypokinetic inter-ventricular septum and adjacent anterior wall at mid and basal cavity level with ejection-fraction 48% [Figure 2]. Coronary angiogram subsequently showed normal coronary arteries [Figure 3]. He was asymptomatic in subsequent follow-ups, but the wall motion abnormality in echocardiogram persisted.

MI after cocaine use is related to the block of re-uptake of norepinephrine that leads to α and β adrenergic effects. These include increased heart rate, blood pressure, and simultaneous coronary vasospasm with reduced oxygen delivery leading to myocardial ischemia.[1] Cocaine also activates platelets, increases platelets aggregability and potentiates thromboxane production, thus promoting thrombus formation.[2] The highest risk of coronary events is within 1st h of intake with no relation to the dose or route of administration.[3] Interestingly, anterior wall is involved in most cases.[4] Chest pain and ECG changes are common, even in absence of ischemia, related to high prevalence of hypertension among these patients, causing diagnostic difficulty.[4] Troponins are more sensitive and specific for myocardial injury than creatine kinase which may rise due to rhabdomyolysis.[5]

Treatment for chest pain and ECG changes after cocaine use include benzodiazepines, aspirin, and nitrates.[5] Benzodiazepines reduce blood pressure and heart rate and are recommended especially in patients with associated hypertension, tachycardia, or anxiety. Oxygen helps in limiting myocardial ischemia. Calcium channel and α blockers can be added next. Use of β blockers can be deleterious and should be avoided in acute stage, as they may worsen vasospasm by permitting unopposed stimulation of α receptors.

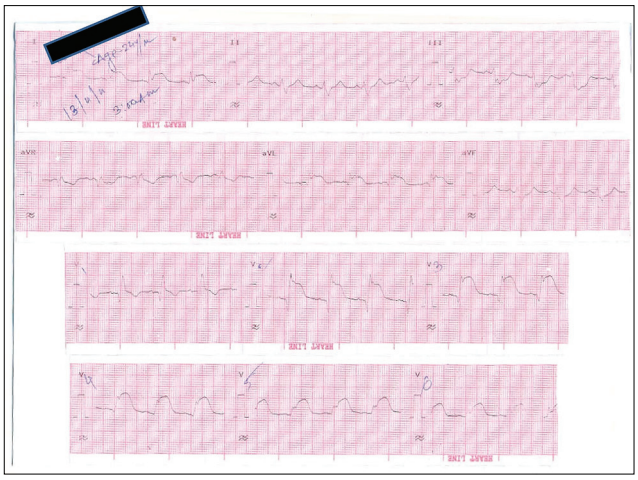

Figure 1: Electrocardiogram showing ST elevation in anteroseptal and lateral leads with reciprocal changes in inferior leads

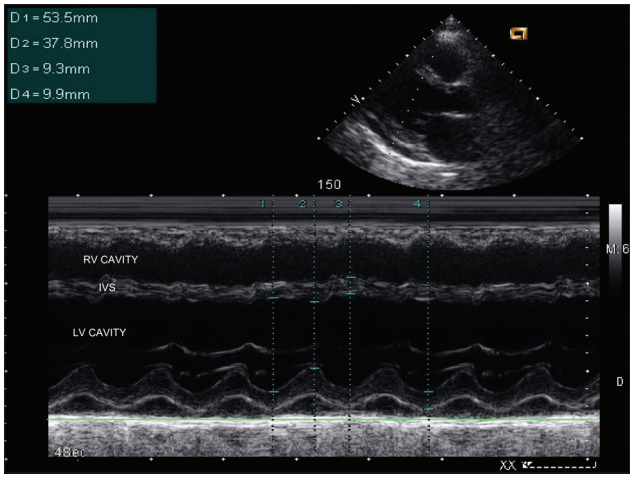

Figure 2: M-mode echocardiogram showing severely hypokinetic inter-ventricular septum. Right ventricle, left ventricle

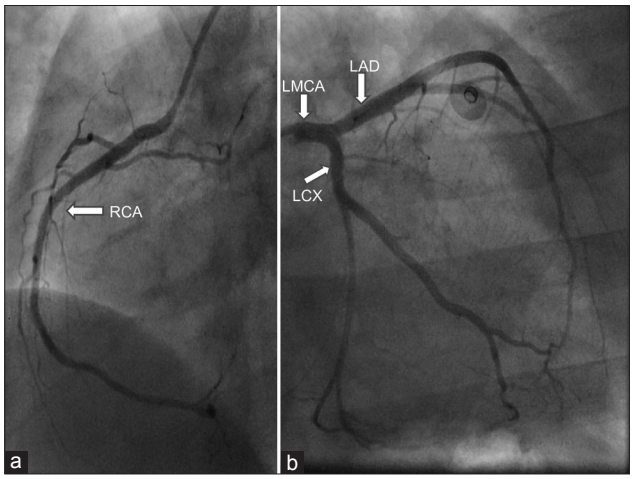

Figure 3: Coronary angiogram showing normal filling of (a) right and (b) left coronary arteries. Right coronary artery, left main coronary artery, left anterior descending artery, left circumflex artery

ST elevation myocardial infarction (STEMI) require immediate thrombolytic therapy or percutaneous coronary intervention; later preferred when available.[5] Since, platelets play important role in pathophysiology, GP IIB/IIIA (glycoprotein IIB/IIIA) platelet receptor inhibitors should be an integral part of acute intervention. Caution should be applied in thrombolytic therapy as cocaine users may have altered consciousness and sustained various injuries not obvious on initial evaluation.

More emergency physicians are likely to encounter this situation with increasing cocaine abuse in India. Cocaine addiction should be actively excluded in young patients with acute coronary syndrome.Qualitative determination of cocaine metabolites (benzoylecgonine) in urine may be performed in suspected patients. Long-term interventions include patient education and de-addiction.

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References

1. Hollander JE, Hoffman RS, Gennis P, Fairweather P, DiSano MJ, Schumb DA, *et al*. Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group. Acad Emerg Med 1994;1:330-9.
2. Togna G, Tempesta E, Togna AR, Dolci N, Cebo B, Caprino L. Platelet responsiveness and biosynthesis of thromboxane and prostacyclin in response to *in vitro* cocaine treatment. Haemostasis 1985;15:100-7.
3. Mittleman MA, Mintzer D, Maclure M, Tofler GH, Sherwood JB, Muller JE. Triggering of myocardial infarction by cocaine. Circulation 1999;99:2737-41.
4. Minor RL Jr, Scott BD, Brown DD, Winniford MD. Cocaine-induced myocardial infarction in patients with normal coronary arteries. Ann Intern Med 1991;115:797-806.
5. McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, *et al*. Management of cocaine-associated chest pain and myocardial infarction: A scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 2008;117:1897-907.

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