Acute myocardial infarction with normal coronary arteries in a young heroin user: A case report
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Summary
Substance abuse with cocaine or heroin are associated with multiple cardiovascular complications including acute myocardial infarction (AMI), left ventricular hypertrophy, fatal arrhythmias, sudden cardiac death and cardiomyopathy. We describe a case of AMI with normal coronary arteries in a 23-year old young man who collapsed within few minutes of sniffing heroin. He was documented to have ventricular tachycardia (VT) during admission which was converted to sinus rhythm after cardioversion. Subsequently he was found to have an acute anterior myocardial infarction with significant left ventricular dysfunction. Coronary angiography conducted later on was found to be normal.

Key words
AMI with normal coronary arteries, case report

Introduction
Cocaine-associated AMI is a well reported entity1-3. Heroin abuse and AMI has already been reported4. AMI with normal coronary arteries is a syndrome resulting from numerous conditions but the exact cause in a majority of the patients remains unknown. This type of myocardial infarction have been documented in pregnancy and among cigarette smokers and cocaine users5-7. Here the authors report a case of AMI in a young healthy male patient with history of substance abuse and his three year follow-up. This paper also discusses the possible pathological mechanisms underlying the pathogenesis of AMI with normal coronary arteries secondary to heroin abuse and current ideas on the management of cocaine or heroin induced AMI.

Case report
A 23 year old young man developed sudden onset of marked palpitation following few minutes of sniffing heroin. He also developed severe chest pain along with profuse sweating and within short time he collapsed. He was immediately brought to a coronary care unit. Electrocardiogram (ECG) revealed broad complex tachycardia suggestive of VT. As he was haemodynamically unstable VT was converted to sinus rhythm with cardioversion. ECG taken after cardioversion showed wide spread ST segment elevation in anterior leads with reciprocal ST depression in inferior leads. The diagnosis of AMI was made on ECG changes and cardiac enzyme profile. He was thrombolysed and his further hospital stay was uneventful. Echocardiogram was done one week after the acute event. Echo-Doppler study revealed dilated left ventricle with fairly good LV systolic function. Overall Left ventricular ejection fraction (LVEF) was 50%. Regional wall motion abnormalities were consistent with previous Septal and Apical myocardial infarction. Coronary angiogram conducted few weeks later was found to be normal. After counseling the patient quitted drug addiction and remains asymptomatic from cardiac point of view at three year follow-up.

Discussion
AMI is the most commonly reported cardiac...
consequences of cocaine misuse, usually occurring in men who are young, fit and healthy and who have minimal, if any, risk factors for coronary artery diseases. The mechanism by which cocaine induces AMI is largely not understood. Though cocaine induced AMI and arrhythmia are common heroin induced AMI is rare. But heroin induced AMI and cardiac decompensation have been reported in recent past. AMI occurring in young people with angiographically normal coronary arteries is well described but the pathophysiology of the condition remains unclear. The possible mechanisms causing AMI with normal coronary arteries are hypercoagulable states, coronary embolism, an imbalance between oxygen demand and supply, intense sympathetic stimulation, non-atherosclerotic coronary diseases, coronary trauma, coronary vasospasm, coronary thrombosis and endothelial dysfunction. It primarily affects younger individuals, and the clinical presentation is similar to that of AMI with coronary atherosclerosis. AMI with normal coronary arteries primarily affects younger persons and is distinctly rare in patients older than 50 years.

Our patient was young and his coronary angiogram did not show any significant coronary artery disease. Possibly he developed AMI complicated by VT due to severe coronary vasospasm. In AMI with normal coronary arteries, complications such as malignant arrhythmias, heart failure and hypotension are generally less common and prognosis is usually good. But our patient developed VT and was admitted in the hospital in collapsed state. AMI in patients with history of cocaine or heroin abuse are sometimes associated with significant coronary artery disease. In one series 90 patients with cocaine use presenting with AMI underwent coronary angiography. Significant disease (>50% stenosis) was present in 45 (50%) patients. AMI related to heroin use with significant coronary artery disease was previously reported in the medical literature. A young woman developed AMI due to an acute thrombosis of the left anterior descending artery induced by intravenous heroin use has been documented. So substance abusers may have AMI with significant coronary artery disease. The patient reported here was treated for AMI according to the standard guidelines. He was treated with thrombolysis, antiplatelet therapy, heparin, vasodilators and calcium channel blocker. At three year follow-up our patient was doing well. He quit drugs and is now asymptomatic from cardiac point of view. Recurrent infarction, post infarction angina, heart failure and sudden cardiac death are rare in this condition. Long term survival mainly depends on the residual left ventricular function. The reported case was documented to have reasonably good left ventricular systolic function at one year follow-up and we expect good long term survival is this particular case.

**Conclusion**
AMI can occur in young persons with normal coronary arteries and the diagnosis should be considered in young patients presenting with severe chest pain, particularly those abusing cocaine or heroin, so that reperfusion therapy can be initiated promptly. Thrombolytics, aspirin, nitrates and beta blockers should be
instituted as a standard therapy for AMI. Once normal coronary arteries are identified on subsequent coronary angiography, beta-blockers should be replaced by calcium channel blockers as coronary vasospasm appears to play a major role in the pathophysiology of this condition.

References