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CASE REPORT
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CASE REPORT

COCAINE INDUCED CORONARY ARTERY SPASM LEADING TO ST ELEVATION MYOCARDIAL INFARCTION (STEMI)

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ABSTRACT:

Cocaine use may lead to acute myocardial infarction. Despite being expensive, it is most commonly used illicit street drug. Cocaine-induced infarction is particularly common in younger patients. Adversely affecting the cardiovascular system, it can lead to sudden cardiac death, ventricular arrhythmias and myocardial infarction. During acute event, coronary angiography may be normal or there may be evidence of coronary artery disease. Coronary artery spasm is reversible phenomena characterized by focal or diffuse constriction of coronary segment in one or more coronaries, restricting blood flow in coronaries, causing myocardial ischemia and thrombus formation.[1-2]

KEY WORDS: *cocaine, spasm, ST Elevation Myocardial Infarction, complications*

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INTRODUCTION:

Cocaine remains a popular drug for illicit use. Cocaine use, a leading cause of drug-related issues, accounts for 30% admission in emergency department. It has shown a recent surge over the last few years and has surpassed morphine use. By inducing tachycardia and increasing systolic blood pressure, cocaine use is linked to 25% of cases of myocardial infarction in patient age group 18-45 years. Also, by enhancing activities at alpha-1 adrenergic receptor, cocaine causes coronary vasospasm and endothelial injury, which in turn predisposes to thrombus formation and acute ST elevation myocardial infarction. Sudden cardiac death is thought to be results from cardiac Na channel blocking effect.[3]

CASE REPORT:

14-year-old young well built, class 9th O-level student enjoying high socioeconomic status, non-diabetic and normotensive, non-smoker with no history of pre mature coronary artery disease and sudden cardiac death in family, presented to us in emergency department with severe retro sternal chest pain, sudden onset chest, griping in nature, radiating to left arm for 45 minutes and was associated with sweating and dyspnea. It was partially relieved by rest and medications given at some peripheral hospital. The general physical and systemic examination was unremarkable except his heart rate was 104 beats per minute and blood pressure was 160/100 mmHg. Initially patient denied any kind of addiction. Once the routine workup was negative for causal of myocardial infarction, then on re-inquiring patient told that he is cocaine addict. He used to snort cocaine through nose for last six months. He suffered from chest pain for six hours after the last dose.

Pathology findings were as follows: Triglycerides = 102mg/dl, Cholestrol = 90mg/dl, HDL-Cholesterol = 28mg/dl, LDL-Cholesterol = 47mg/dl, T-Chol & H-Chol Ratio = 3.2 mg/dl, CRP = 24mg/l (raised), protein C: 85 IU/dL, protein S: 116 U/dL. Cardiac biomarkers were raised. Connective tissue profile showed negative ANA and RA Factor. Urine examination was normal with no proteinuria. Cocaine metabolites in urine and plasma were detected. Echocardiography showed good biventricular function and ejection fraction of LV was 60%. Coronary Angiography showed normal coronary arteries with sluggish flow and dominant right system.

Patient was treated with asprin, clopidogrel, benzodiazepine, statin, nitrates and heparin and became pain free after 4 to 6 hours.

Fig-1 shows ST elevation in II, III avF V5 and V6 with reciprocal changes in anterial leads and avL. Fig-2 and 3 shows there is progressive settling down of the ST elevation.

Fig-4 shows T inversion and II, III avF V5 and V6.

Fig -1 : First ECG done at periphery hospital.

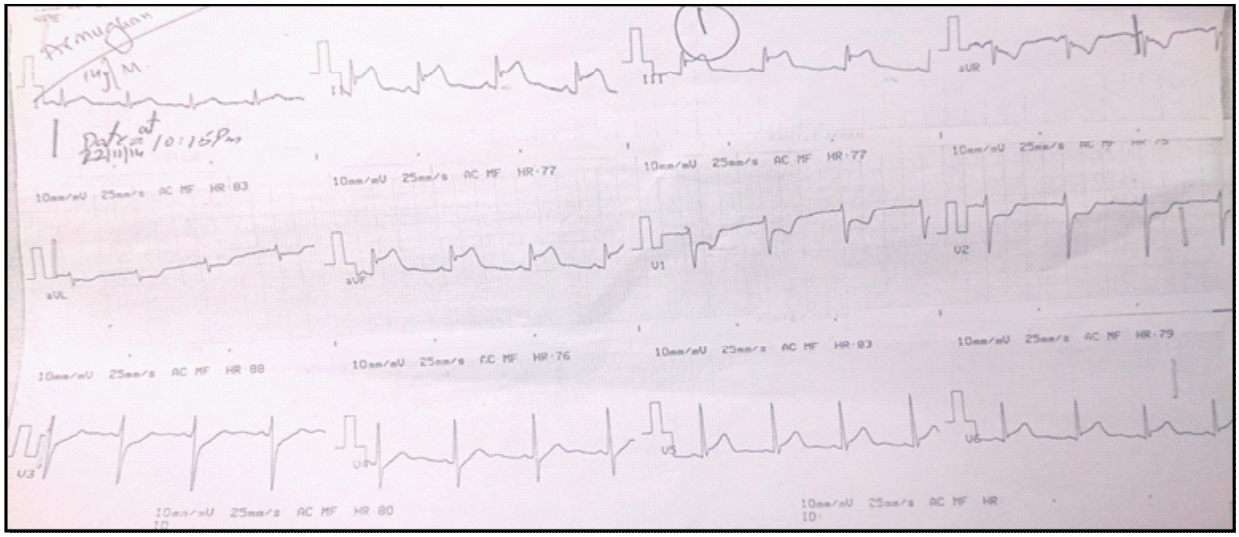
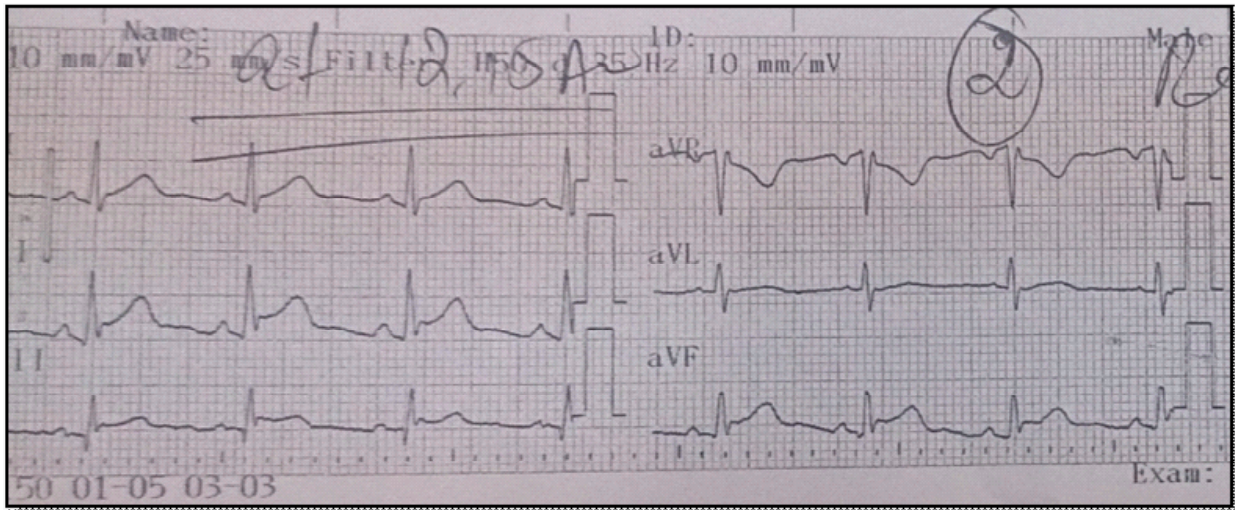


Fig - 2: ECGs done at Punjab Institute of Cardiology, Lahore.



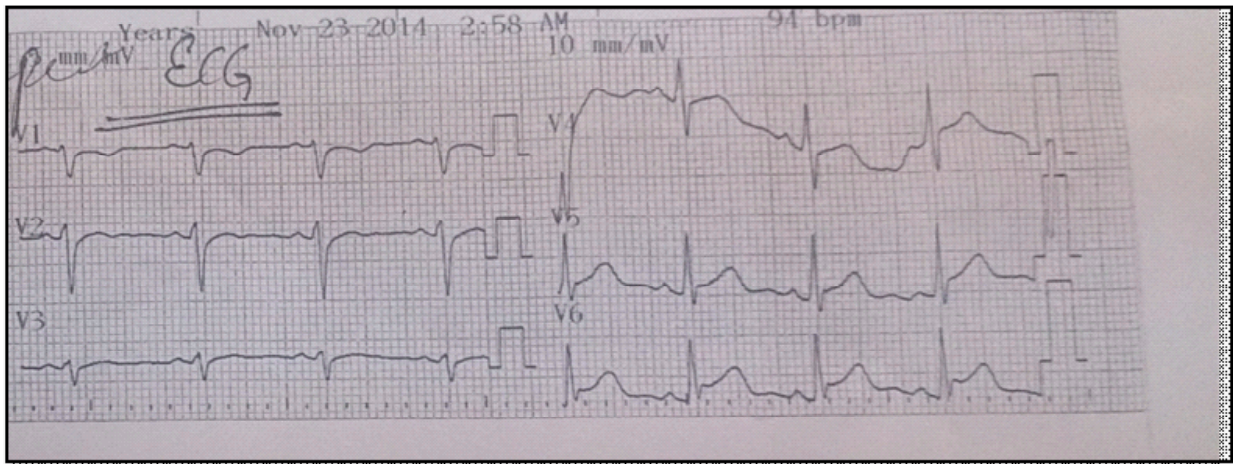


Fig-3: As this patient was pain free and ST segments were significantly settled down, the patient was not thrombolysed and heparin infusion was started. Serial ECGs were done which are as follows:

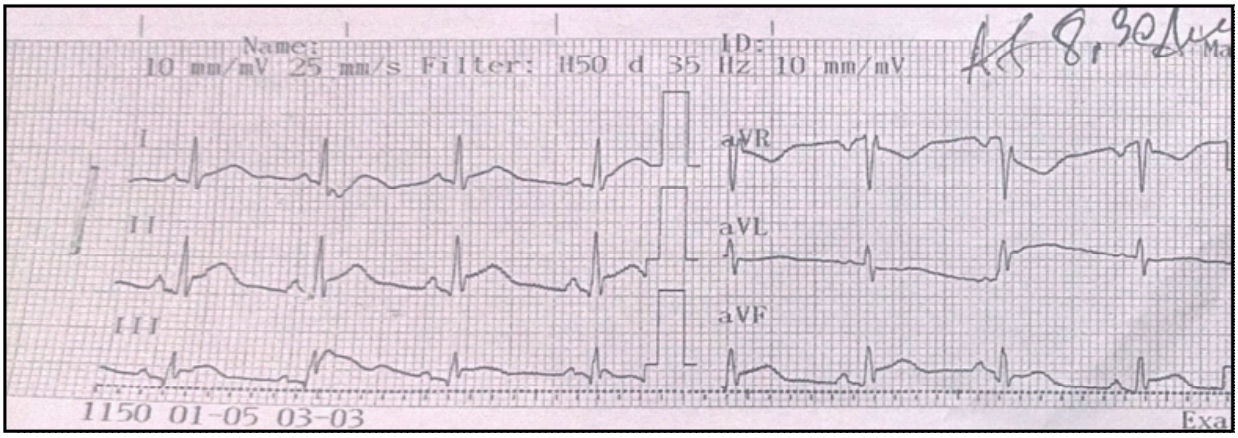
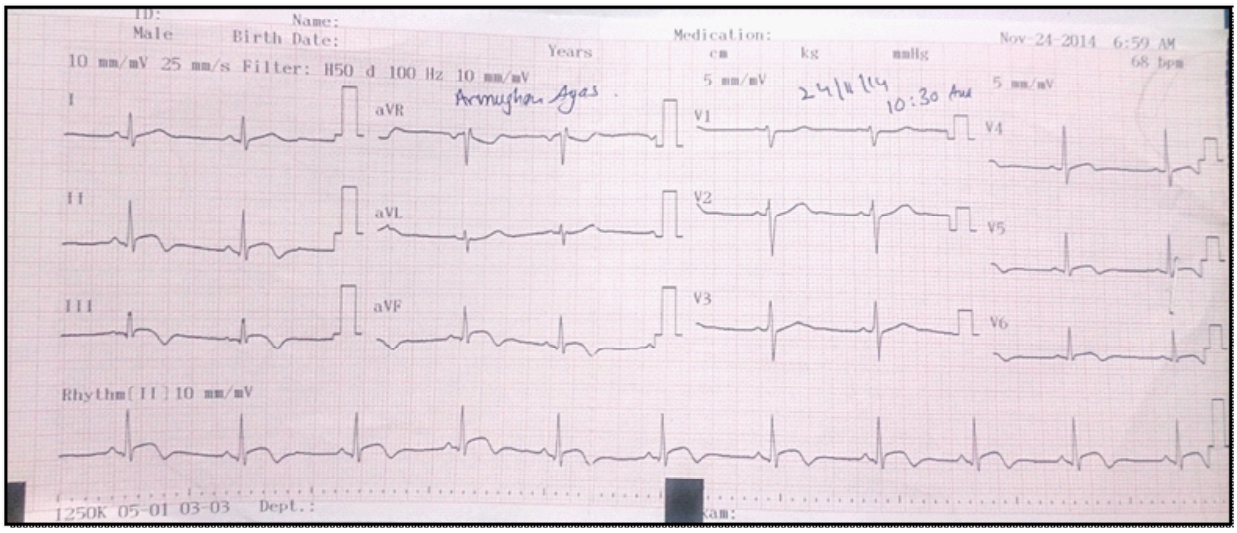


Fig - 4: Shows T inversion and II, III avF V5 and V6.



DISCUSSION:

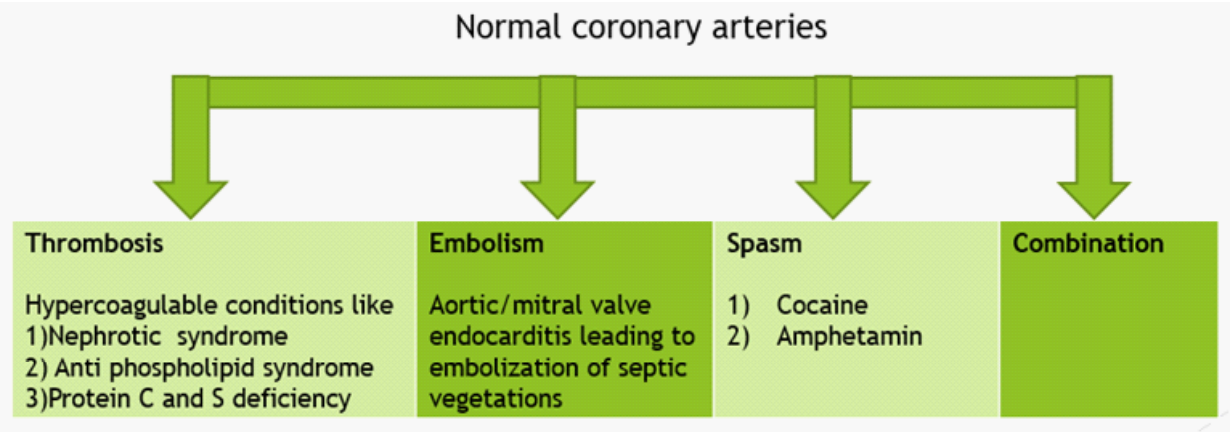
Cocaine is a naturally occurring stimulant that, with continued use, produces an addictive syndrome. It is sold as a crystalline white powder, which varies in purity and is often diluted with procaine, dry milk, or talcum powder. It is usually inhaled, but may be injected intravenously. [4]

The patients with cocaine induced chest pain should have done ECG, chest x-ray, in addition to ACS, the differential diagnosis are aortic dissection which was present in some case series (>20%) patients had a recent history of drug use and dissection.[5,6]

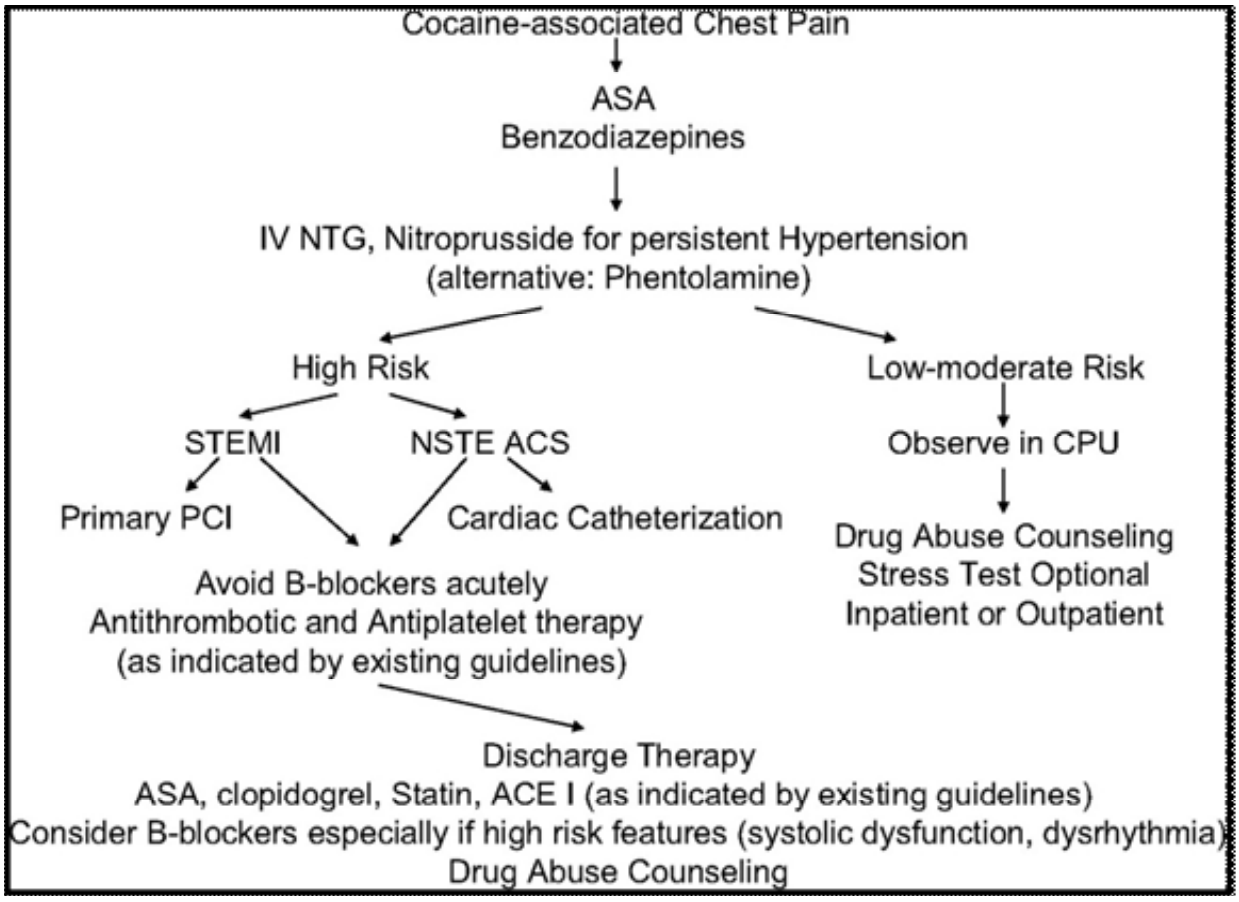
Cocaine has very diverse effect on cardiovascular system, with various mechanisms that can cause myocardial ischemia resulting in acute myocardial infarction. The major mechanism is related to increased circulating norepinephrine secondary to inhibited reuptake for which cardiovascular sympathetic response increases and needs high demand of myocardial oxygen while myocardial perfusion decreases due to coronary vasospasm.[7]

Previous studies have shown that cocaine use may cause acute coronary occlusion by inducing coronary vasospasm and activating platelets leading to thrombus formation. Revascularization through Primary coronary intervention, performed by experienced team, remains the preferred modality of treatment. Thrombolytic are the suitable alternatives if primary coronary intervention is not available. Beta blockers may enhance coronary vasospasm or may elevate blood pressure paradoxically, should be used with cautions.[8,9]

Causes of Myocardial Infarction in Teenage and young



Management



American Heart Association recommendations for cocaine-associated chest pain
McCord J et al. Circulation. 2008;117:1897-1907

CONCLUSION:

Cocaine-induced myocardial infarction in patients with normal coronary arteries probably involves adrenergically mediated increases in myocardial oxygen consumption, severe endothelial injury leading to vasoconstriction of epicardial arteries and coronary thrombosis.

References:

1. Gil Botero, E., Medina Ramírez, C., Rodríguez Padilla, R., Paredes Rosero, M., & Crispín Amorocho, G. P. Reporte de drogas Colombia. 2017; 3: 224.
2. Hsue PY, Salinas CL, Bolger AF, Benowitz NL, Waters DD. Acute aortic dissection related to crack cocaine. Circulation. 2002;105: 1592-1595.
3. Kushman SO, Storrow AB, Liu T, Gibler WB. Cocaine-associated chest pain in a chest pain center. Am J Cardiol. 2000;85:394-396, A10
4. Terminology and Information On Drugs (second edition). Laboratory and Scientific Section United Nations Office on Drugs and Crime. United Nations New York, 2003.
5. Hsue PY, Salinas CL, Bolger AF, Benowitz NL, Waters DD. Acute aortic dissection related to crack cocaine. Circulation. 2002;105: 1592-1595.
6. Eagle KA, Isselbacher EM, DeSanctis RW; International Registry for Aortic Dissection (IRAD) Investigators. Cocaine-related aortic dissection in perspective. Circulation. 2002;105:1529-1530.
7. Whitby, L. G., Hertting, G., & Axelrod, J. Effect of cocaine on the disposition of noradrenaline labelled with tritium. Nature, 187(4737), 604
8. Hollander JE, Hoffman RS, Gennis P, Fairweather P, Feldman JA, Fish SS, DiSano MJ, Schumb DA, Dyer S. Cocaine-associated chest pain: one-year follow-up. Acad Emerg Med. 1995;2:179-184.
9. Hollander JE, Hoffman RS. Cocaine-induced myocardial infarction: an analysis and review of the literature. J Emerg Med. 1992;10:169-177.