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Case Report

Acute Coronary Syndrome Following Recent Cannabis Ingestion during Festive Occasion

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Abstract

Oral form of Cannabis (Bhang) is social norm in northern India often associated with festivities. Marijuana smoking is known to have hemodynamic consequences, which cannot be said about oral cannabis. Here we report a case of myocardial infarction by cannabis ingestion in 45 year old male who consumed cannabis for the first time and presented in emergency with chest pain and breathing difficulty.

Keywords: Cannabis;Myocardial Infarction; Angiography

Abbreviations

THC:Tetrahydrocannabinol; CB-1: Cannabinoid Receptor 1; ECG: Electrocardiogram; CVS: Cardiovascular System; PCI: Percutaneous Coronary Intervention; CVD: Cardiovascular Disease

Introduction

The Cannabis sativa plant contains 700 different chemical compounds, out of which principal psychoactive compound is Tetrahydrocannabinol (THC). THC acts via cannabinoid receptor-1 in central and peripheral nervous system and mediates adverse effects on the cardiovascular system through the same receptor [1,2]. The cardiovascular effects of marijuana depend on several factors like composition of the chemicals (the higher the THC content in the plant, the higher the likelihood of CB1 receptor mediated cardiovascular effects) and the route of administration (inhalation route can lead to rapid increases in plasma levels with more rapid decline, whereas oromucosal administration of marijuana extracts, such as nabiximols, or pure THC can result in lower, but more stable levels) [3]. Edible cannabis is thought to be safer than smoked cannabis in certain ways (reduced carcinogen exposure), titration of dose through oral administration, but it may result in stronger and more frequent adverse side effects resulting in panic, paranoia and performance impairment. This risk stems from slow and unpredictable absorption of orally administered cannabis [4,5]. While the role of inhaled cannabis is well established in cardiovascular diseases the same cannot be said for cannabis when ingested.We report a case of myocardial infarction induced by oral ingestion of cannabis in 45 year old male.

Case Presentation

45 year male, non hypertensive, non diabetic presented with complaints of retrosternal chest discomfort, heaving in nature, non radiating associated with perspiration for past 24 hours. The patient had a history of oral consumption of cannabis which was then followed by the presenting symptoms. Patient was anxious having profuse sweating. However, the patient had no history of addiction. On examination, Heart rate was-102/min and BP was 170/90 mm of hg with an increased Heart rate of 24/minute. Cardiovascular system examination revealed normal JVP, heart sounds were normally auscultated. Other systems were unremarkable. ECG showed ST elevation in limb leads 1 and aVL, precordial leads V2-V6 (Figure 1a). Patient had a history of having yearly medical check up for last 10 years and found to be normotensive, nondiabetic and normal lipid profile. Family history was negative for diabetes, dyslipidemia, hypertension cardiovascular, cerebrovascular disease. Qualitative troponin test showed a level more than 100 ngm/l. Urine Cannabinoid level came out to be more than 50 ng/ml. Echocardiography showed hypokinesia in mid anteroseptal and left apical region with left ventricular ejection fraction (LVEF) of 45% having with normal valves morphology, no pericardial effusion, no clot or vegetation. Serial CPK-MB came as 178 u/l, 104 u/l, and 80 u/ at 0,6 and 12 hours. Complete blood profile, serum electrolytes, kidney function test were within normal limit (Table 1) Thyroid profile, random blood sugar (RBS) and lipid profile were also within normal limits. Serial ECG showed gradual resolution ST-T changes over a period of 24 hours (Figure 1b). Thrombolysis was not done as he presented out of window period and his chest pain gradually disappeared. Immediate coronary PCI was not done as he was haemodynamically stable with no signs of heart failure and chest pain gradually relieving with medication. Coronary angiography was done on second day of admission which revealed normal coronaries (Figure 2a-2c).

Patient was admitted in intensive coronary care unit (ICCU) and managed conservatively with dual antiplatelets (Ecosprin 75 mg and Clopidogrel 75 mg), Inj Enoxaparin 60 mg subcutaneously, metoprolol 25 mg, Ramipril 2.5 mg and oral Nitroglycerin controlled release tablets 2.6 mg. Patient improved with conservative therapy and subsequently discharged on dual antiplatelets, ACE inhibitor and beta blocker.

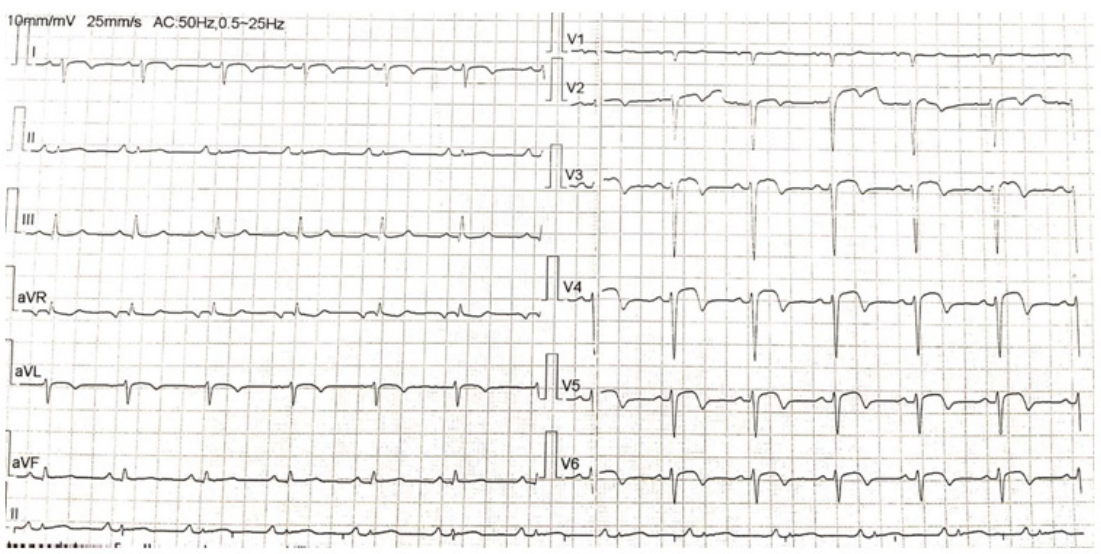

Figure 1

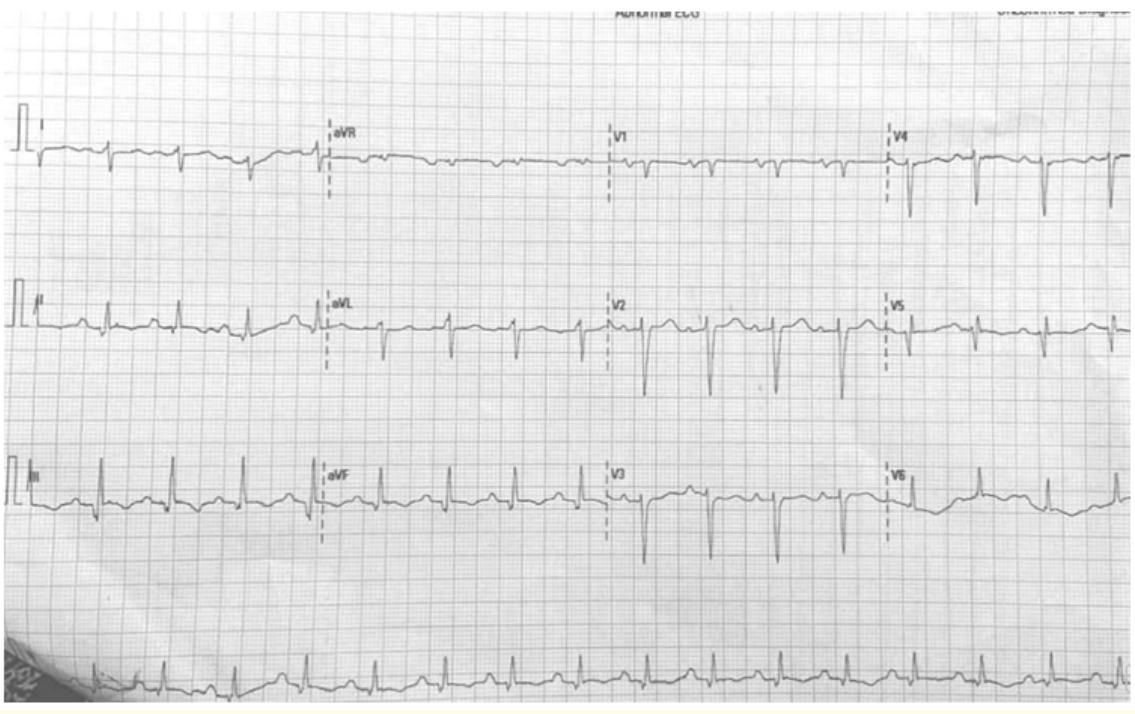

Figure 1b

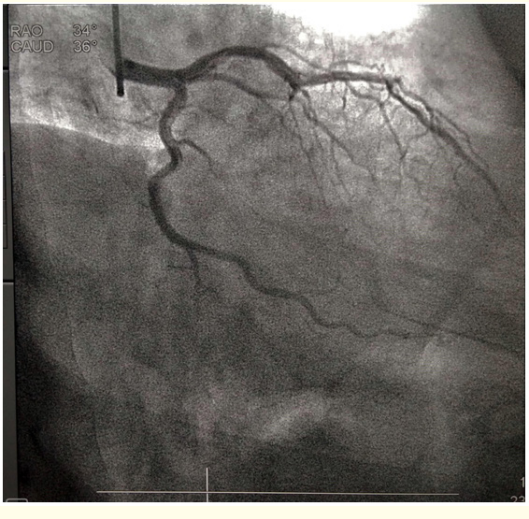

Figure 2a

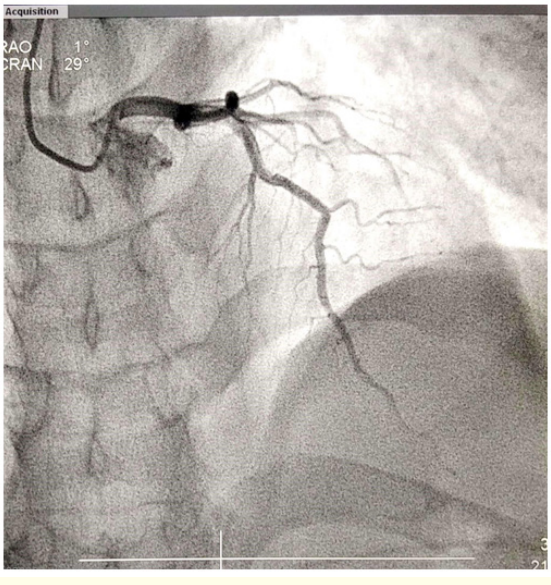

Figure 2b

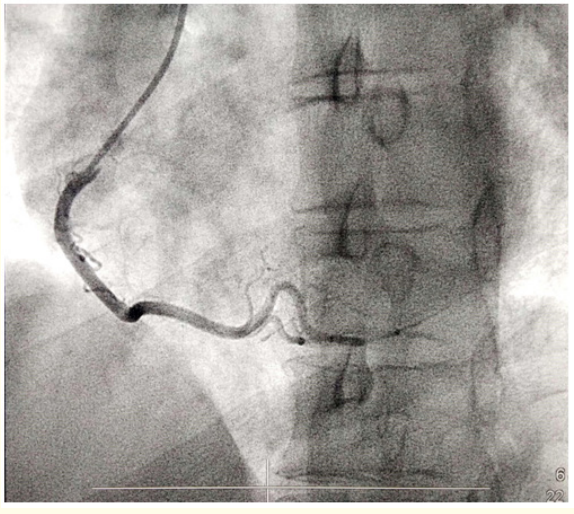

Figure 2c

Hb	16 gm%
TLC	8600/cumm
Plateletcount	280000/cumm
Serum urea	27 mg/dl
Serum creatinine	1.2 mg/dl
Total cholesterol	160 mg/dl
LDL	108.3 mg/dl
Sodium	143 mg/dl
Pottasium	4.3 mg/dl
Triglyceride	93 mg/dl
HDL	38.8 mg/dl
Mg	2.6 mg/dl
CPKMB	178, 104, 80 U/L
HBA1C	5.6%
Urine Canabinoid	> 50 ng/ml
T3	0.93 ng/ml
T4	11.21 mcg/dl
TSH	0.967 IU/ml

Table 1: Baseline investigations.

Discussion and Conclusion

There is a growing body of evidence that demonstrates an association between marijuana use and adverse cardiovascular disease events. The available literature proposes that marijuana adversely affects the CVS through three different possible mechanisms Cannabis-induced arteritis, Vasospasms, Platelet aggregation [6-8]. Reversible arterial vasospasm is considered the most common cause of marijuana-induced vascular events [6]. While, the underlying mechanisms for this are unclear, numerous case reports have associated THC with coronary vasospasm-induced cardiomyopathy [9,11]. While cannabis has been shown to be pro-thrombotic, little is known on the mechanism of THC-induced platelet aggregation [6]. In 2004, Deusch., *et al*. demonstrated the presence of CB1 and CB2 receptors on platelet cell membranes [12]. Furthermore, it was also shown that the expression of glycoprotein IIb-IIIa and P-selectin on platelet membranes increases during CB1 activation in a dose-dependent manner [12]. The available literature shows research only on inhaled cannabinoids and there effects on various systems (Table 1). Vandrey R.,*et al*. concluded that whole blood cannabinoid concentrations significantly correlates with subjective drug effects. Correlations between blood cannabinoids and between oral fluid and all pharmacodynamic outcomes is either non-significant or not orderly by dose. Quantitative levels of cannabinoids in whole blood and oral fluid is low compared with levels observed following inhalation of cannabis. As there is no literature associating coronary artery disease to ingested cannabis we can relate inhaled cannabis causing coronary artery disease as similar mechanism.

In our patient there was no comorbidity and no family history of cardiovascular disease or any risk factor and patient was having yearly medical check up. The temporal association of chest pain within hours of consuming cannabis associates cannabis as a possible cause for myocardial infarction. The mechanism of myocardial infarction in our patient can be ascribed to Cannabis induced coronary vasospasm as ECG changes subsided in follow up ECGs and normal coronary angiogram. The role of inhaled cannabis is well explained by various case reports, but what is interesting in our patient is that he was non addict and had myocardial infarction during first time exposure to oral consumption. In India oral cannabis (Bhang) is generally considered as less toxic form of cannabis and widely consumed during times of festivals. However, in our patient this form of cannabis was not that safe and had serious cardiovascular complications. Further studies are required to elucidate its role and dose response relationship.

Bibliography

1. Izzo AA., *et al*. "Non-psychotropic plant cannabinoids: new therapeutic opportunities from an ancient herb". *Trends in Pharmacological Sciences* 30 (2009): 515-527.

2. Radwan MM., *et al*. "Isolation and pharmacological evaluation of minor cannabinoids from high-potency *Cannabis sativa*". *Journal of Natural Products* 78 (2015): 1271-1276.

3. Pal Pacher., *et al*. "Cardiovascular effects of marijuana and synthetic cannabinoids: the good, the bad, and the ugly". *Nature Reviews Cardiology* 14 (2017): 566.

4. Hudak M., *et al*. "Edible cannabis-induced psychosis: intoxication and beyond". *American Journal of Psychiatry* 172 (2015): 911-912.

5. Favrat B., *et al*. "Two cases of "cannabis acute psychosis" following the administration of oral cannabis". *BMC Psychiatry* 5 (2005): 17.

6. Hodcroft CJ., *et al*. "Cannabis-associated myocardial infarction in a young man with normal coronary arteries". *Journal of Emergency Medicine* 47 (2014): 277-281.

7. Dahdouh Z., *et al*. "Cannabis and coronary thrombosis: What is the role of platelets?" *Platelets* 23 (2014): 243-245.

8. Ducros A., *et al*. "The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients". *Brain* 130 (2007): 3091-3101.

9. Gunawardena MD., *et al*. "Myocardial infarction following cannabis induced coronary vasospasm". *BMJ Case Report* (2014).

10. Basnet S., *et al*. "Coronary vasospasm in an adolescent resulting from marijuana use". *Pediatric Cardiology* 30 (2009): 543-545.

11. El Menyar AA. "Drug-induced myocardial infarction secondary to coronary artery spasm in teenagers and young adults". *Journal of Postgraduate Medicine* 52 (2006): 51-56.

12. Deusch E., *et al*. "The procoagulatory effects of delta-9-tetrahydrocannabinol in human platelets". *Anesthesia and Analgesia* 99 (2004): 1127-1130.