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Cannabis Usage and Prothrombotic Milieu: A Case Report of 38 years Old Cannabis Addict with Multiple Thrombotic Events

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Abstract: A 38 Years Old male, Presented with AWMI, within a Window period of 10 hours. Patient is an alcoholic, smoker and a cannabis addict. Past history of Genito urinary TB; successfully completed anti tuberculous treatment. Patient was taken for coronary angiogram which showed LAD proximal total thrombotic occlusion. Primary angioplasty was done to LAD with Drug eluding stent. Post procedure he was evaluated for cause of young Mland Carotid artery Doppler was done. It showed Right Internal carotid artery thrombus. USG abdomen also showed small thrombosis in Abdominal aorta. Pt was started with antiplatelet and anticoagulant and he stopped cannabis post event, now stable post 4 months of follow up.

Keywords: Acute coronary syndrome, Cannabis, delta - 9 - Tetrahydrocannabinol

1. Introduction

Acute coronary syndrome is rarely caused by coronary artery disease in young patients, unless some other cardiovascular risk factors are present. Although Non atherosclerotic causes of ACS are rare, they need to be considered in young patients. Cannabis induced ACS is one of the important cause of myocardial infarction in young adults, delta - 9 - Tetrahydrocannabinol, the psychoactive component of cannabis increases the expression of glycoprotein IIb/IIIa on human platelets which effects the prothrombotic impact of cannabis. [1] This prothrombotic milieu can cause Hypercoagulability all over the vascular system. It is also affect Psychological, Physical and social wellbeing of the individual. Children are prone to develop somnolence, lethargy and rarely respiratory insufficiency. [2] It also affects Lung causing chronic bronchitis with inhaled use. Deterring patients from substance use by Peer network counselling, cognitive behavioral therapy Multidimensional family therapy is needed for Managing Cannabis use disorder. [3]

2. Case Presentation

A 38 years Old Male, Presented with complains of chest pain for 10 hours duration. Patient is a smoker, alcoholic and a cannabis addict. He smokes 10 - 12 cigarette / day, smoke cannabis every days around 10 packs with alcohol usage - 2 - 3 standard drink /week. His Vitals were stable and ECG showed ST elevation in anterior leads [Figure 1]. Patient was taken for Coronary angiogram which showed Proximal LAD total thrombotic occlusion [Figure 2]. LAD was wired and predilated with Minitrek balloon 1.2x6mm[at]14atm and Ryurei 2.5x15mm[at]12atm Pressure [Figure 3]. Stented with Xiencexpedition 3.5x23mm[at]16atm. Post procedure TIMI iii flow was established [Figure 4]. And post procedure patient was stable. Patient had Haemoglobin 18.2 g/dl and other routine investigation was normal - Including Platelet and WBC count. Patient underwent Carotid artery doppler which showed echogenic structure in Right Internal carotic artery. Hence Proceeded with CT angiogram of Intracranial and neck vasculature, which showed Noncontrast opacification of right internal carotid artery just after origin involving cervical and intracranial (petrous, cavernous, supraclinoid segments) with reformation of right middle cerebral artery [Figure 5]. USG abdomen was also taken which showed Echogenic small thrombus in Abdominal aorta. However there were no sign of Limb ischemia. Echocardiogram showed Moderate LV dysfunction with No clot In Left Ventricle. Serum Erythropoietin was normal and Jak 2 mutation was negative. ANA Profile was done which was negative. Other thrombophilic workup like protein C and Protein S mutation was done which was negative. Cannabis consumption with Coexisting Smoking was the Probable reason for hypercoagulability with coexisting lesion in Coronary artery. He was discharged with Rivaroxaban and dual antiplatelet agent. He Stopped Consuming cannabis and post 4 months follow up he is stable.

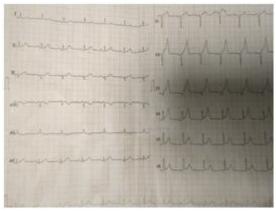


Figure 1: ECG suggestive of Anterior Wall Myocardial infarction

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Figure 2: Proximal LAD total thrombotic occlusion (LAO Caudal View)

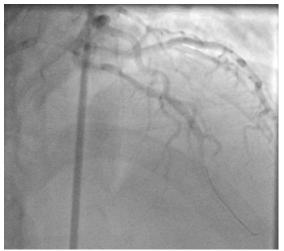


Figure 3: Balloon dilatation of Proximal LAD lesion



Figure 4: Post stenting - TIMI iii flow established

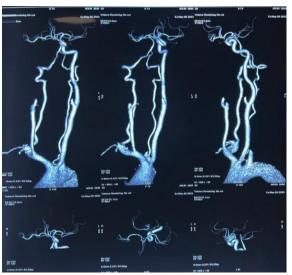


Figure 5: CT angiogram of neck vessel

3. Discussion

Illicit consumption of Cannabis is widespread globally. Despite the fact that Cannabis/Marijuana has decreased from its zenith in the late 1970s, the illicit use of this substance has remained alarmingly high especially among young adults. [4]

Cannabis acts via cannabinoid receptors CB1 that is highly Proatherogenic. These receptors are found in highest concentration in central/peripheral nervous system and cardiac muscle. Apart from Coronary vascular disease, they also cause stress Cardiomyopathy. Case reports of Myocarditits / Myopericarditis have also been reported with cannabis usage. [5] [6]

Cannabis and its products cause cardiovascular effects via various mechanism. Common Postulated causes include 1] Concurrent hazards - Patients who have Cannabis use also use tobacco products and alcohol2] Platelet activation 3] Oxidative stress 4] Sympathetic activation 5] Direct myocardial injury. [7] Although still the exact mechanism is not well understood, there is risk of endothelial disruption with cannabis usage leading to platelet aggregation and thrombus formation. There is also reported shorter clot formation time, marked increase in clot strength, speed of formation and thrombosis. [8] [9] Cannabis use also may cause Arteriopathy/ Juvenile peripheral obstructive arterial disease. [10] Similarly Smoking cannabis causes a net decrease in oxygen supply demand ratio which is due in part to an increase in blood carboxy Hemoglobin level [11] [12]

4. Conclusion

The increasing use of cannabis exposes a Major Risk of Hypercoagulable state. The Treating Doctor should suspect Cannabis induced MI in any young patients presenting with Myocardial infarction.

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