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Letter to the Editor

Cannabis smoking and acute coronary syndrome: Two illustrative cases

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Abstract

Cannabis is a common substance of drug abuse among the young adults because of its euphoric and addictive effects. The pathophysiological effects of cannabis smoking and its relation to adverse cardiovascular events are well known. However, the relative contribution of cannabis smoking when combined with tobacco smoking to coronary artery disease is unclear and has not been well emphasized.

We describe two cases of acute coronary syndrome occurring in cannabis smokers who were tobacco smoker too. One, a 23 year old young boy who suffered from hypertension and acute coronary syndrome at a very young age and other, a 50 year old male admitted with acute coronary syndrome, developed asymptomatic dynamic electrocardiographic changes and had beta-blocker induced severe bronchospasm.

The modifiable nature of cannabis smoking and cigarette smoking, which often go hand in hand, needs no over emphasis. The cessation of twin smoking habits along with correction of other coronary artery disease risk factors is an important part of primary and secondary prevention.

Keywords: Cannabis; Twin smoking; Acute coronary syndrome; Bronchospasm; Beta blocker

1. Introduction

Cannabis is a common substance of drug abuse among young adolescents because of its sociocultural, psychotropic and addictive effects. The pathophysiological effects of cannabis smoking and its relation to adverse cardiovascular events in cannabis smokers have been previously reported [1]. However, the relative contribution of cannabis smoking when combined with tobacco smoking in pathogenesis of acute coronary syndrome (ACS) and its therapeutic implications is not well known. We describe two cases of ACS occurring in cannabis smokers who were also tobacco smokers.

2. Case 1

A 23 year old male, known hypertensive, presented with complaint of left sided chest pain for 3 days. He had left cigarette smoking 2 years back but admitted to smoking cannabis. He was also regularly drinking about 200 ml of alcohol everyday for the last 2 years. Physical examination including blood pressure was normal. ECG showed ST segment flattening/depression, and T wave inversion in leads II, III, aVF and V1–V6 (Fig. 1). He was treated as a case of ACS. Apart from elevated lipoprotein (a) levels of 58.3 mg/dl, he had otherwise normal renal, liver and lipid profile. Routine urine and abdominal ultrasound were normal. He made uneventful recovery. Patient refused undergoing coronary angiography despite repeated counselling. Alternatively, a stress myocardial perfusion scan performed 6 weeks after the acute event showed exercise induced reversible ischemia in the apical and apicoanterior walls. At

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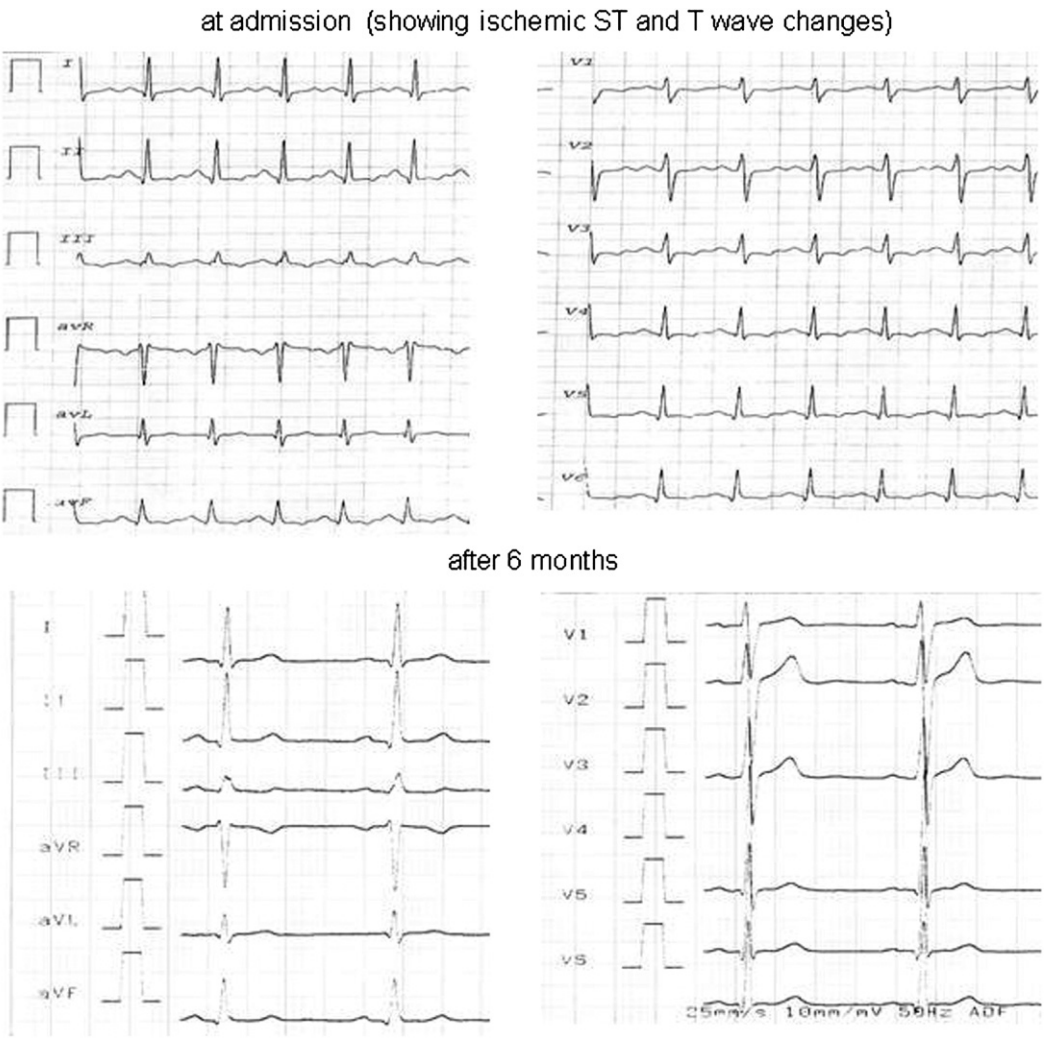

Fig. 1. ECG of patient in case 1 at admission (showing ischemic ST and T wave changes).

his last follow-up visit, he was no longer smoking cannabis; asymptomatic and regularly taking medicines as prescribed for secondary prevention of CAD.

3. Case 2

A 50 year old male presented with acute onset severe retrosternal chest pain, sweating and vomiting. He was a cigarette smoker and a regular cannabis smoker since his early teens. There were no other identifiable coronary risk factors. He looked older than his chronological age, otherwise unremarkable. Admission electrocardiogram showed ST segment elevation in leads V2–V5 and aVL (Fig. 2). Creatine kinase (MB) level was raised up to 685 U/L. A diagnosis of acute anteroseptal and lateral myocardial infarction (MI) was made. Thrombolytic therapy along with standard treatment for ACS was administered. Pain subsided within next 3 h but he developed severe bronchospasm after first dose of beta-blocker, metoprolol. Beta-blocker therapy was omitted. His subsequent stay in the hospital was completely asymptomatic. However, his successive ECGs showed dynamic ST segment and T wave changes in addition to gradual evolution to Q waves in leads V3–4 (Fig. 2). He denied smoking cigarette or cannabis during hospital stay. The patient could not afford coronary angiography due to lack of financial resources. The facilities for cardiac catheterization and urine toxicology screen for cannabis were not available at our institution. Patient responded to conventional therapy very well.

4. Discussion

Cannabis is derived from the plant *Cannabis sativa*. It is a common drug of abuse in both urban and rural population in India. Several preparations of this plant e.g. bhang, marijuana, charas, ganja, hashish etc. are either smoked or taken orally by people. The effects of cannabis are primarily mediated by the activation of cannabinoid receptors, which are present in brain, heart, blood vessels, spleen and immune system [1]. Cannabis is rapidly absorbed through the lungs. Smoking cannabis produces supraventricular and ventricular contractions and reversible electrocardiographic P, T waves