

The Dramatic Reversal of Hashish-Induced Junctional Tachycardia and Unstable Angina with Standard Therapy in Heavy Smoker Patient

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ABSTRACT

Rationale: Smoking cannabis is known to be a rare acutely trigger acute coronary syndrome Cigarette smoking is a strong, independent risk factor for acute and chronic ischemic heart disease. Hashish smoking, unstable angina, and pass phenomenon are possible causes for junctional tachycardia.

Patient Concerns: A middle-aged married heavy smoker male patient presented in the emergency department with unstable angina within two hours post-two hashish cigarette.

Diagnosis: Hashish-induced junctional tachycardia and unstable angina was the most probable diagnosis.
Interventions: Electrocardiography, Echocardiography, and non-baric-oxygenation.

Lessons: Hashish smoking can be inducing unstable angina and junctional tachycardia, especially in the heavy smoker one. The major predisposing factor in hashish-induced acute myocardial infarction was a heavy cigarette smoking. So, How are you dealing with a heavy cigarette smoker patient presented with unstable angina after hashish cigarette smoking?

Outcomes: Dramatic reversal of Hashish-induced junctional tachycardia and unstable angina with standard anti-ischemic therapy in heavy cigarette smoker.

Keywords: Hashish-induced junctional tachycardia and unstable angina, hashish, cannabis, junctional tachycardia, unstable angina, cigarette smoker

Abbreviations

AVN: Atrioventricular node, ECG: Electrocardiography, ICU: Intensive care unit, MI: Myocardial infarction, NSR: Normal sinus rhythm, O₂: Oxygen, SAN: Sinoatrial node, THC: Tetrahydrocannabinol, VR: Ventricular rate

INTRODUCTION

Worldwide, cannabis is considered the most commonly utilized outlawed illegal substance¹. Its enormous usage is highly consumed in young people (15- to 34-year-olds)¹. However, the main source of cannabis is the Cannabis sativa plant². So, there are three major types of cannabis products: herb (marijuana), resin (hashish) and oil (hash oil)³. The most intense form of cannabis is cannabis oil, derived from the concentrated resin extract³. It may contain more than 60% of tetrahydrocannabinol (THC) content³. Different classes of chemicals, including nitrogenous compounds, amino acids, hydrocarbons, sugar, terpenes, and simple fatty acids, together contribute to the unique pharmacological and toxicological properties of

cannabis¹. Delta-9-tetrahydrocannabinol (Δ -9-THC) and Cannabidiol [CBD], the two main ingredients of the cannabis Sativa plant, have distinct symptomatic effects¹. As regards the United Nations Office on Drugs and Crime (UNODC), the amount of THC present in a cannabis sample is generally used as a measure of cannabis potency³. In humans, the acute effect of smoking cannabis usually manifests as an increase in heart rate with no significant change in blood pressure⁴. Of the negative effects of cannabis on the cardiovascular system including increased risk of acute coronary events, development of atrial ischemia, and increasing both heart rate and blood pressure⁵. Smoking cannabis is known to be a rare acutely trigger myocardial infarction (MI)^{3,6}. Cannabis

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has been linked to the dose-dependent way of inducing elevated rates of MI¹. One large study of 1,913 adults conducted in the United States found both a significant association between MI and cannabis use and a dose-response effect^{5,6}. Postulated mechanisms for this include complex interactions between increased oxygen (O₂) demand (due to increased heart rate and blood pressure), decreased O₂ supply (due to an increase in carboxyhemoglobin) and coronary vasospasm².

The involved nomenclature⁷ to understand the type of junctional rhythms (JR) is dependent on their rate. They are classified as follows: **1.** Junctional bradycardia: Ventricular rate <40 bpm. **2.** Junction escape rhythm: Ventricular rate 40-60 bpm. **3.** Accelerated junctional rhythm (AJR): Ventricular rate (VR) of 60-100 bpm. **4.** Junctional tachycardia: Ventricular rate >100 bpm⁷. If there is a blockage for the sinoatrial node (SAN) electrical activity is blocked or is less than the automaticity of the atrioventricular node (AVN)/His Bundle a JR starts. Numerous conditions and medications can lead to a diseased SAN and lead to the AVN/His Bundle to take over due to the higher automaticity of the ectopic pacemaker^{7,8}. Junctional tachycardia may also be due to ischemia of the AVN, especially with acute inferior infarction involving the posterior descending artery, the origin of the AV nodal artery branch⁸. This rhythm may occur in persons of any age. Junctional rhythms, which are common in younger⁸. The following causes implicated in inducing junctional tachycardia⁷: **toxic and pharmacologic:** e.g., cannabinoids, opioids, clonidine, reserpine, cimetidine, lithium, amitriptyline, radiation therapy, antiarrhythmics class I to IV (e.g., beta-blockers, calcium channel blockers, adenosine, and digoxin), ivabradine, and isoproterenol infusion, **cardiovascular:** e.g., acute and chronic coronary artery disease, rheumatic fever, repair of congenital heart disease, pericarditis, myocarditis, sick sinus syndrome, inherited channelopathy, **endocrinal and metabolic:** e.g., hypothyroidism, anorexia nervosa, sleep apnea, hypoxia, hyperkalemia, amyloidosis, and Lyme disease, **central:** e.g., neuromuscular disorder, x-linked muscular dystrophy, intracranial hypertension, **autonomic:** vasovagal simulation (endotracheal suctioning), carotid sinus hypersensitivity, and **traumatic:** e.g., recent cardiac surgery and chest trauma^{7,8}. Junctional tachycardia may also be

due to the onset of acute coronary syndrome⁹. Prognosis is good⁸. Complications of junctional rhythm are usually limited to symptoms such as dizziness, dyspnea, or presyncope⁸.

Cigarette smoking is a strong, independent risk factor for acute and chronic ischemic heart disease¹⁰. Cigarette smoking is an important determinant of acute coronary events¹¹. The importance of cigarette smoking is confirmed as a cause of acute coronary syndrome¹². The pathogenesis for the adverse effect of cigarette smoking on the coronary arterial circulation is complex and multi-factorial. Smoking increases both heart rate and blood pressure, thereby augmenting myocardial O₂ demand. Thereafter, smoking reduces the dimension of the coronary arteries and coronary blood flow¹⁰.

CASE PRESENTATION

A 43-year-old married, driver, Egyptian male patient presented in the emergency department with acute severe chest pain and palpitation. The chest pain was anginal. Profuse sweating was the associated symptoms. The patient gave a recent history of smoking two hashish cigarettes. He was a heavy cigarette smoker (40-60 cigarette per day for 17 years) The patient denied the history of cardiovascular diseases, smoking, drugs or special habits or the same attack. Upon physical examination; generally, the patient was tachycardic, sweaty, and anxious, with a regular heart rate of 110 bpm, blood pressure of 140/80 mmHg, respiratory rate of 16 bpm, the temperature of 36.4 °C, pulse oximeter of O₂ saturation; 97% and tachycardia on heart auscultation. No more relevant clinical data were noted during the clinical examination. He was admitted to the ICU as unstable angina. Urgent ECG was done showing junctional tachycardia (**Figure 1A**). The second ECG tracing was taken within 3 minutes of the first one. The junctional tachycardia was rapidly replaced with sinus tachycardia with the appearance of straight ST-segment depressions in V4-6 leads (**Figure 1B**). The patient was initially managed in the ICU with 100% O₂ inhalation using nasal cannula at the rate of 5 L/min. Aspirin; four oral tablet (75 mg), clopidogrel; four oral tablet (75 mg), bisoprolol; one oral tablet (5 mg), enoxaparin; twice SC daily (60 mg), and atorvastatin; one oral night tablet (40 mg) were urgently given. Pethidine HCL (100 mg) was given for chest pain in intermittent doses as needed. The third ECG tracing was taken within 75 minutes of the

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first ECG tracing showing normalization of above ST-segment depressions and return to normal sinus rhythm (NSR) (**Figure 2**). Clinical improvement with the disappearance of chest pain had happened. The only measured random blood sugar was 121 mg/dl. The troponin test was positive (87 ng /L). Later echocardiography was mild anterolateral hypokinesia with EF 55%. No more workup was done. The case was initially managed as unstable angina. Hashish-induced junctional tachycardia and unstable

angina was the most probable diagnosis. The patient was continued; aspirin tablet (75 mg, once daily), clopidogrel tablet (75 mg, once daily), nitroglycerin retard capsule (2.5 mg twice daily), enoxaparin; (60 mg twice SC daily), and atorvastatin; (40 mg one oral night tablet) until discharged. The patient discharged within 36 hours after controlling the chest pain, tachycardia, and serial electrocardiographic normalization with recommended outpatient clinic follow up.

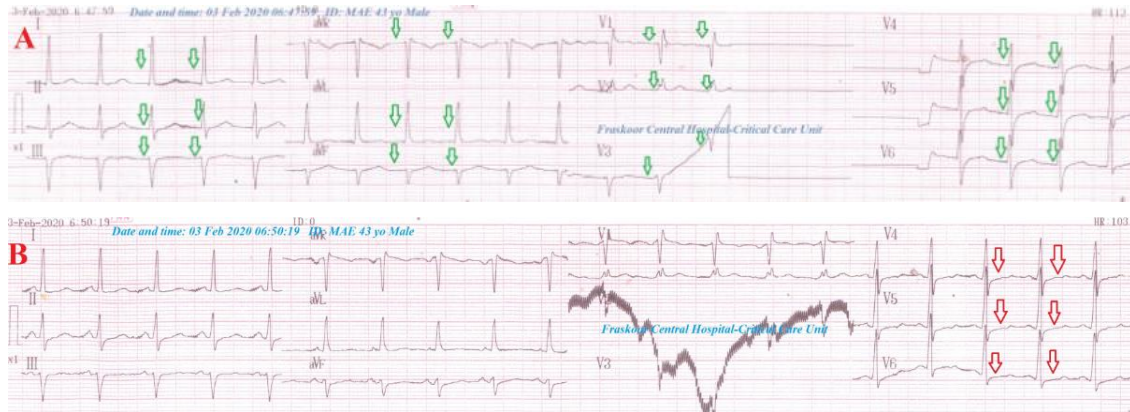


Figure1. Urgent ECG tracing was taken in the ICU showing evidence of junctional tachycardia with rapid regular VR, and absent “P-wave” (green arrows) (**tracing 1A**). Sinus tachycardia (VR; 100) with straight ST-segment depressions in V4-6 leads (red arrows) have rapidly appeared (**tracing 1B**). There are incomplete right bundle branch block and artifact in lead V3.

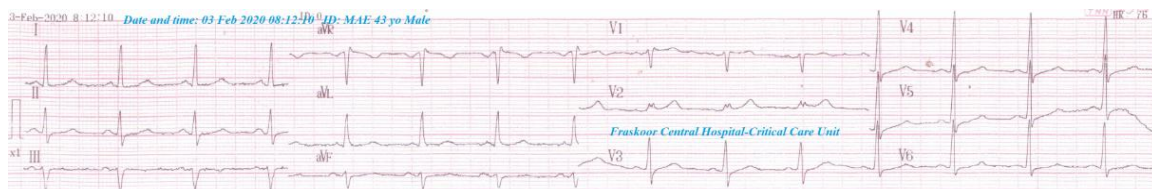


Figure2. ECG tracing was taken within 75 minutes of the first ECG tracing showing normalization of above ST-segment depressions and return to normal sinus rhythm (NSR with VR; 74)

DISCUSSION

- **Overview;** A middle-aged married heavy cigarette smoker male patient presented in the emergency department with unstable angina within two hours post-two hashish cigarette.
- **The primary objective** for the current case was the presence of unstable angina in the heavy smoker male patient within two hours post-two hashish cigarette.
- **The secondary objective** for the case study was; How are you dealing with a heavy cigarette smoker patient presented with unstable angina after two hashish cigarette smoking?
- **Limitations of the study;** there were no known limitations for the case study.

- I can't compare the study case with another one due to there was no publicized similar cases.
- Cession of cigarette smoking was strongly advised. Future planning for coronary angiography was **recommended**.

CONCLUSIONS

- Hashish smoking can be inducing unstable angina and junctional tachycardia, especially in the heavy cigarette smoker patient.
- The major predisposing factor in hashish-induced acute myocardial infarction was a heavy cigarette smoking.
- Unstable angina and pass phenomenon are possible other mechanisms for junctional tachycardia.

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- Thrombotic, coronary vasospasm, and hypoxia were suggested mechanisms for unstable angina.

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