



Both Wellens' Syndrome Types Post Hashish- Induced Acute Myocardial Infarction in A Young Cigarette Smoker, A Case Report

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Abstract

Acute myocardial infarction (STEMI) is unusual in young persons. In my present case was a young male presented with acute myocardial infarction after hashish (cannabis) smoking. The case was complicated with Wellens' syndrome. Heavy cigarette smoking was a possible *major predisposing* factor. An extremely rare to see both variants of Wellens' syndrome in the same case. The used study method for an abuse substance-induced in the current patient was a case study. The aim of the study directed towards the clinical observation of patient symptoms and complications after hashish smoking *and infarction* and how to deal with them?. Generally, hashish smoking implicated in *inducing acute myocardial infarction*. This helped with previous heavy cigarette smoking. So, why hashish smoking-induced *infarction*?. *Did* heavy cigarette smoking were a relevant predisposing factor?. *How* Both Wellens' syndrome types had happened?

Keywords

Hashish; Cannabis; Induced Acute Myocardial Infarction In A Young; Cigarette Smoker; Both Wellens' Syndrome

Abbreviations

CK-MB: Creatine Kinase MB
CPK: Creatine Phosphokinase
DES: Drug-Eluting Stent
ECG: Electrocardiographic
ICU: Intensive Care Unit
IV: Intravenous Infusion
LAD: Left Anterior Descending
LDH: Lactate Dehydrogenase
NSR: Normal Sinus Rhythm
NSTEMI: Non-ST-segment elevation myocardial infarction
STEMI: ST-Segment Elevation Myocardial Infarction
WS: Wellens' Syndrome

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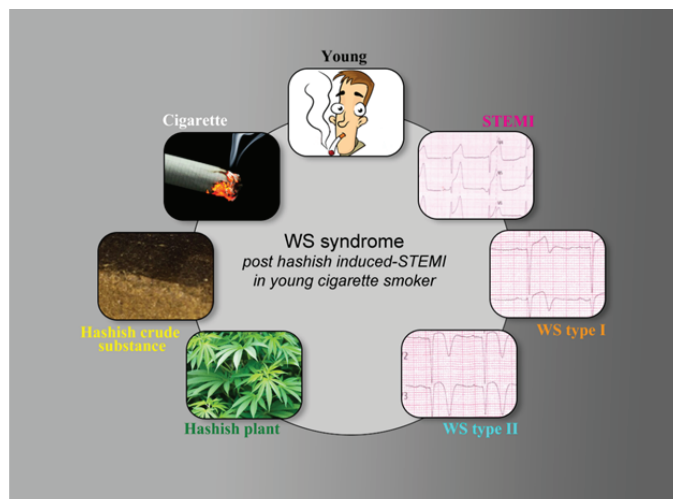
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Graphical Abstract

Introduction

Wellens' syndrome (WS) is a pattern of electrocardiographic T-wave changes associated with critical, proximal Left Anterior Descending (LAD) artery stenosis [1]. The WS is also referred to as LAD coronary T-wave syndrome [1]. Syndrome criteria include; T-wave changes plus a history of anginal chest pain without serum marker abnormalities; patients lack Q waves and significant ST-segment elevation; such patients show normal precordial R-wave progression [1]. The ECG changes occur during the pain-free period [2]. Typical ECG changes, either a biphasic T wave or deep symmetrical T wave inversions in the precordial leads, occur due to a critical LAD block [3]. WS is classified according to the ECG description into two types; type I: biphasic T waves in often in V2 & V3 but in type II: deep T wave inversion [2]. WS is not always an acute complication [3]. The course pattern may manifest itself persistently for weeks [3]. The Natural history of WS is an imminent anterior wall acute myocardial infarction [1,3]. T-wave changes in WS are typically observed in asymptomatic patients [3]. Although patients may well initially respond to medical management, they ultimately fare poorly with conservative therapy and require revascularization strategies [2]. Increased awareness of WS facilitates communication between ED physicians and cardiologists and significantly improves patient outcomes [3]. Once WS has been diagnosed, general treatment measures for NSTEMI including anti-ischemic therapy such as nitrates and beta-blockers in the absence of contraindications [3]. It is vital to identify this pattern and manage the patient by invasive revascularization at the earliest opportunity [2]. If left untreated or managed medically, the patient may develop an extensive myocardial infarction or sudden death [2,3].

Cannabis is the most widely used illegal substance in the World [4]. Its use is largely concentrated among young people (15- to 34-year-olds) [4]. It is derived from the plant Cannabis Sativa [5]. There are three main types of cannabis products: herb (marijuana), resin (hashish) and oil (hash oil) [6]. The most potent form of cannabis is cannabis oil, derived from the concentrated resin extract [6]. It may contain more than 60 percent of THC content [6]. 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 cannabis [4]. Delta-9-tetrahydrocannabinol (Δ -9-THC) and Cannabidiol (CBD), the two main ingredients of the cannabis sativa plant, have distinct symptomatic effects [4]. 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 potency [6]. Smoking cannabis is known to be a rare acutely trigger myocardial infarction [6,7]. Cannabis has been linked to dose-dependent way in inducing elevated rates of myocardial infarction [4]. One large study of 1,913 adults conducted in the United States found both a significant association between myocardial infarction and cannabis use and a dose-response effect [7]. Postulated mechanisms for this include complex interactions between increased oxygen demand (due to an increased heart rate and blood pressure), decreased oxygen supply (due to increase in carboxyhaemoglobin) and coronary vasospasm [5].

Cigarette smoking is a major, independent risk factor for coronary heart disease and acute myocardial infarction [8]. Cigarette smoking is an important determinant of acute coronary events [9]. The importance of cigarette smoking is confirmed as a cause of acute myocardial infarction [10]. 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 oxygen demand. Thereafter, smoking reduces the dimension of the coronary arteries and coronary blood flow [8].

Case Presentations

A 36-year-old married Egyptian male Police officer patient presented to the emergency room with acute severe retrosternal

chest pain radiating to the left arm in the early morning. He was a heavy smoker (60 cigarettes per day for 12 years). There was a recent history of two hashish cigarette smoking since about 45 minutes. The patient denied any history of cardiovascular disease or other relevant diseases. Upon examination, the patient appeared irritable, sweaty, and anxious. His vital signs were as follows: blood pressure: 100/70 mmHg, pulse rate: 68/minute; regular; temperature: 36.7°C, respiratory rate: 16/min, initial pulse oximetry: 94 %. No relevant local cardio-respiratory signs. The Patient was admitted in ICU and initially managed with O₂ inhalation using nasal cannula in the rate of 5 L/min, aspirin 4 oral tablet (75 mg), clopidogril 4 oral tablet (75 mg), streptokinase IVI (1.5 million units over 60 minutes) were added. Initial workup was an ECG tracing was showed NSR (VR;60 bpm) with ST-segment elevation myocardial infarction in anterior leads V1-6 with inferior reciprocal changes (II, III, aVF) (Figure 1A). Pethidine HCL (100 mg) was given for chest pain in intermittent doses. Chest pain was still compliant. Serial ECG tracings were taken for follow up. Laboratory work-up: Initial troponin I test was positive (958 ng/L), CPK (696 U/L), CK-MB (57 U/L), and LDH (667 U/L) Blood counts were normal except an elevated leukocytic count for (17300/mm³). The other biochemistry was normal. Echocardiography on the 2nd day of admission showed anterior hypokinesia with EF 50 % (Figure 2A). An emergent coronary angiography on the day of admission revealed critical proximal L.A.D artery thrombotic occlusion. Aspiration of LAD artery thrombus with DES stent placement it had happened (Figure 2B-2D). WS type I appeared on the 7th day but WS type I in the 5th day of admission. Coronary angiography was repeated after the appearance of WS type II with no abnormality detected. Patient continued on; captopril tablet (25 mg twice daily), aspirin tablet (75 mg, once daily), clopidogril tablet (75 mg, once daily), nitroglycerin retard capsule (2.5 mg twice daily), bisoprolol (5 mg, once daily), and atorvastatin (40 mg once daily) until discharged on the 3rd day.

Discussion

- Overview of the case results included in the occurrence of acute myocardial infarction (STEMI) in a young person
- I can't compare the current case study to other studies due to absent of similar condition
- Study question here; Why acute STEMI had happened in young person?. And why hashish smoking-induced infarction?. Did

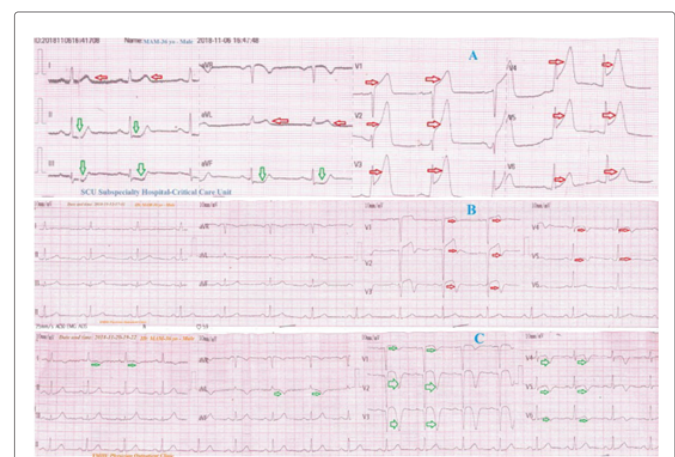


Figure 1: Serial ECG tracings.

Tracing A; Initial presentation showing acute STEMI. ST-segment elevation in V1-6, I, aVL. Carry the criteria of acute STEMI (Red arrows) with inferior reciprocal changes (II, III, aVF) (green arrows). Tracing B; on the 7th day of the presentation showing biphasic T wave in the precordial leads; more clearly in V2-3. Tracing C; on the 14th day of the presentation showing deep symmetrical T wave inversions in the precordial leads; more clearly in V2-3. (Green arrows).

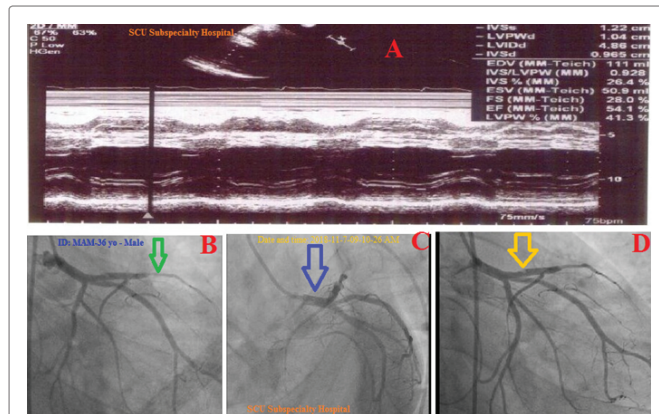


Figure 2: M-Mode Echocardiography showing borderline EF.

A; Coronary angiography; B; showing critical narrowing of LAD (Green arrow), C; on the aspiration of thrombus (Blue arrow), and D; showing DES of LAD (Yellow arrow).

heavy cigarette smoking were a relevant predisposing factor? How Both WS types had happened?

- The primary objective for my case study was clinical appearance of hashish smoking-induced acute STEMI but an associated cigarette smoking was a major predisposing factor. In addition to the clinical presence of both WS types carry significant prognostic value. But, the secondary objective was the clinical improvement of patient symptoms with no further sequels.

Limitations of the study

There are no known limitations of the study.

Recommendations

It is recommended to expect the happening of acute STEMI after hashish smoking even in a young patients. The risk higher if the patient was a heavy cigarette smoker. Also, the appearance of WS was a complication occurred within the first two weeks of STEMI presentation.

Conclusions

- Hashish smoking can induce acute STEMI, even in the young patient
- The major predisposing factor in hashish-induced acute myocardial infarction was a heavy cigarette smoking.
- Thrombotic and coronary vasospasm were suggested mechanism for acute STEMI
- The appearance of WS; type I, then type II indicates the usual sequence for LAD critical stenosis and not separate types. A further broad study will be recommended

- The occurrence of WS after coronary angiography not only meaning LAD critical stenosis but may also be implicated in development of the disease

Conflicts of Interest

There are no conflicts of interest.

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