



Cannabis consumption and myocardial infarction: a report of two cases

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Abstract

Background: Cannabis is the most widely consumed illicit substance in northern Morocco. An increasing body of literature relates acute cannabis use with major cardiovascular events, particularly acute myocardial infarction (AMI), in young adults without traditional coronary artery disease risk factors.

Case presentation: We report two cases of young men who developed ST-elevation myocardial infarction (STEMI) following active cannabis consumption. Case 1: a 29-year-old male who presented with atypical epigastric symptoms, developed cardiorespiratory arrest shortly after electrocardiogram (ECG) acquisition and died; post-mortem examination confirmed recent myocardial necrosis, although the causal link to cannabis remains presumptive in the absence of toxicological confirmation. Case 2: a 31-year-old male with combined cannabis and cocaine use who presented with a 7-hr history of epigastric pain; coronary angiography demonstrated multi-segment coronary thrombosis of the left anterior descending artery (LAD) without spasm or dissection, successfully managed by percutaneous coronary intervention (PCI).

Conclusions: These cases suggest that cannabis consumption may be a trigger for AMI, even in young adults without traditional cardiovascular risk factors. Coronary thrombosis was documented in Case 2, potentially aggravated by polydrug use. Atypical presentations, such as epigastric pain, should prompt active exclusion of AMI in young cannabis users. Larger prospective studies are needed to characterise the incidence, mechanisms and optimal management of cannabis-associated AMI in a better way.

Keywords

cannabis, myocardial infarction, coronary thrombosis, STEMI, tetrahydrocannabinol, young adults

Introduction

Cannabis (*Cannabis sativa*) is a widely consumed illicit psychoactive substance globally, with a remarkable prevalence in northern Morocco [1,2]. Its active constituent, delta-9-tetrahydrocannabinol (Δ -9-THC), exerts complex cardiovascular effects mediated through cannabinoid type 1 (CB1) and type 2 (CB2) receptors expressed in cardiac and vascular tissue [3].

A growing body of evidence associates acute cannabis consumption with the onset of acute myocardial infarction (AMI) in young adults without traditional cardiovascular risk factors [4]. Three principal pathophysiological mechanisms have been proposed: coronary vasospasm, coronary thrombosis and spontaneous coronary artery dissection (SCAD) [5]. Beyond AMI, cannabis use has been associated with other adverse cardiovascular outcomes, including ischaemic stroke, peripheral arterial disease and cannabis-induced cardiomyopathy [6]. These risks are compounded when cannabis is used in combination with other psychoactive substances such as cocaine, and may vary depending on THC concentration and route of administration.

We herein report two cases of STEMI in young male cannabis users, illustrating different pathophysiological mechanisms and clinical outcomes, and discuss the diagnostic and therapeutic implications.

Case reports

1. Case 1

A 29-year-old male, an active cannabis user, presented to the emergency department 1 hr after the sudden onset of acute epigastric pain, nausea and vomiting while at work. He had no prior medical history and no traditional cardiovascular risk factors. Clinical examination on arrival was unremarkable, with stable vital signs.

A 12-lead electrocardiogram (ECG) demonstrated ST-segment elevation in lead aVR associated with diffuse ST-segment depression in leads I, II, III, aVF and V2–V6 (Figure 1), a pattern consistent with global subendocardial ischaemia or critical left main/proximal left anterior descending (LAD) coronary artery occlusion, although other aetiologies of global subendocardial ischaemia cannot be excluded on ECG criteria alone.

Shortly after ECG acquisition, the patient experienced a sudden loss of consciousness and cardiorespiratory arrest. Resuscitation attempts were initiated immediately but proved unsuccessful. The patient was pronounced dead. Post-mortem examination confirmed recent myocardial necrosis, manifested as macroscopic myocardial hyperaemia in the antero-septal wall, consistent with an acute ischaemic event. The specific epicardial coronary artery segment involved could not be determined with certainty, and the presence or absence of underlying coronary atherosclerosis was not formally assessed histologically, as a complete autopsy was not performed due to restrictions imposed by the family. Toxicological confirmation of recent cannabis exposure (blood or urine THC levels) was not

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Figure 1 - 12-lead ECG of Case 1 showing ST-elevation in lead aVR with diffuse ST-depression in leads I, II, III, aVF and V2–V6, suggesting global subendocardial ischaemia or critical left main/proximal LAD occlusion. ECG, electrocardiogram; LAD, left anterior descending.

available. Accordingly, the causal attribution to cannabis in this case remains presumptive and constitutes a significant limitation.

2. Case 2

A 31-year-old male with a history of heavy cannabis abuse and occasional cocaine use presented to the emergency department with a 7-hr history of epigastric pain. He had no documented history of hypertension, diabetes, or dyslipidaemia. Cocaine use was specifically addressed during history-taking; the patient reported only occasional past use, with the last episode reported more than 1 week prior to presentation. Formal toxicological screening was not performed at our centre; while acute cocaine intoxication was not clinically suspected, its synergistic prothrombotic and vasoconstrictive contribution cannot be entirely excluded and is acknowledged as an important confounding factor. Neither case provided precise quantitative data on cannabis use frequency, quantity, route of administration, or THC concentration, precluding any dose-response inference.

The 12-lead ECG revealed ST-segment elevation in the septo-apical (V2–V5) and high lateral (I, aVL) territories (Figure 2), consistent with proximal LAD occlusion. The patient was transferred urgently for coronary angiography.

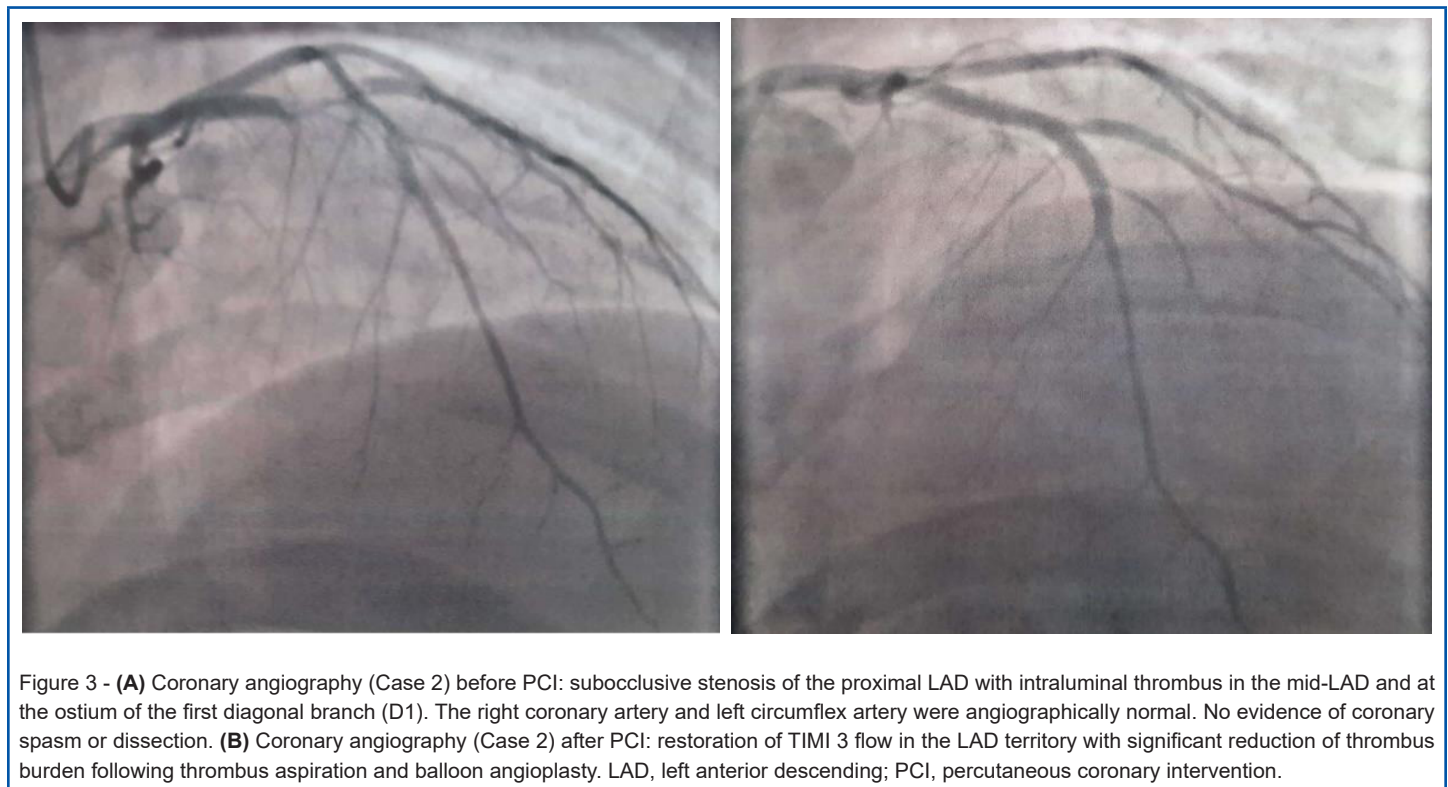
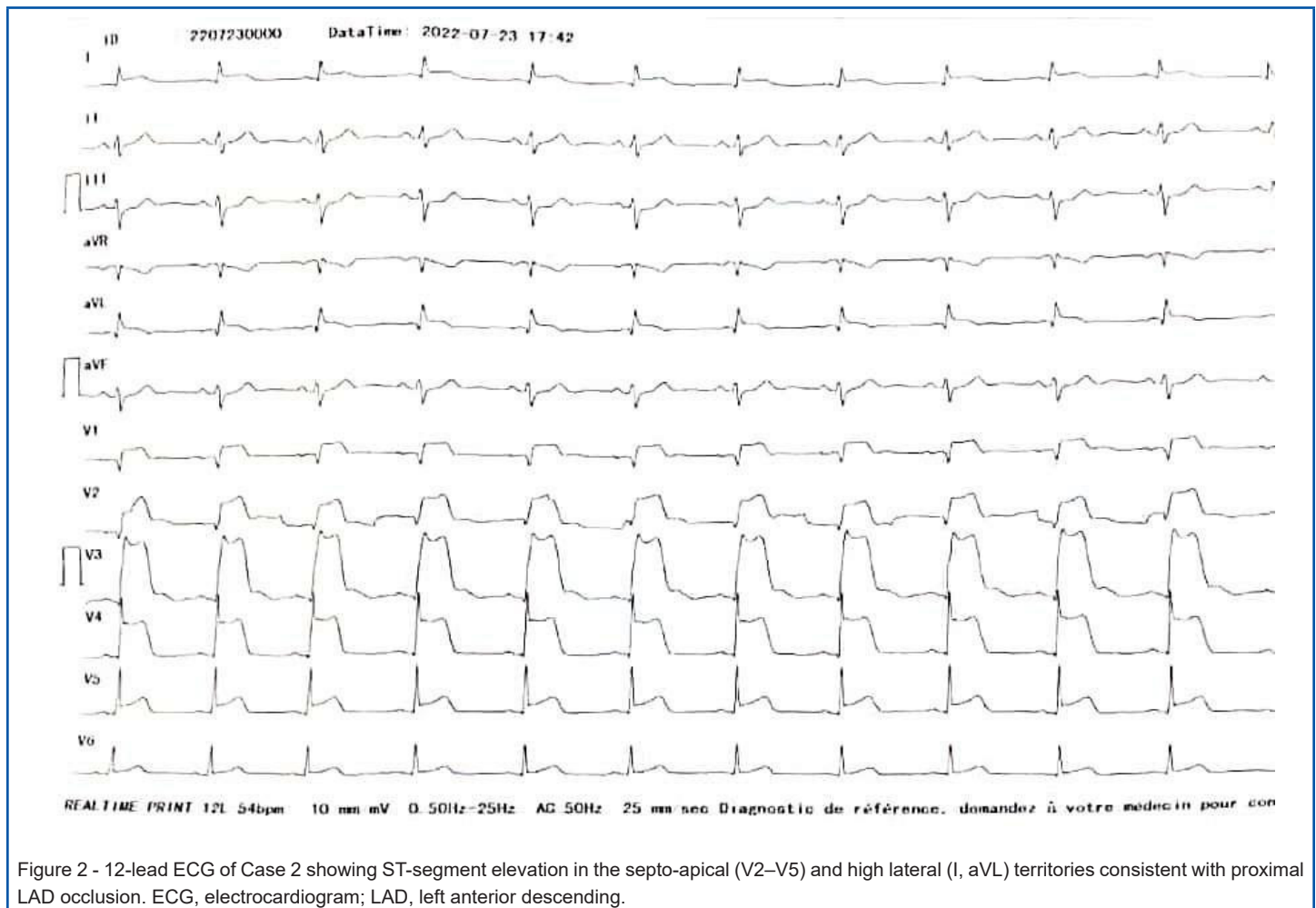
Coronary angiography demonstrated: (1) subocclusive stenosis of the proximal LAD; (2) intraluminal thrombus in the mid-LAD; and (3) subocclusive stenosis with intraluminal thrombus at the ostium of the first diagonal branch (D1) (Figure 3A). No evidence of coronary

vasospasm or SCAD was identified. The right coronary artery and left circumflex artery were angiographically normal.

Primary PCI was performed with glycoprotein (GP) IIb/IIIa inhibitor infusion and manual thrombus aspiration, followed by balloon angioplasty of the proximal LAD lesion. No stent was implanted, given the absence of angiographic evidence of underlying atherosclerotic plaque. Intravascular imaging (IVUS or OCT) was unavailable at our centre at the time of the procedure, which hindered the ability to characterise the underlying pathophysiology fully. The aspirated thrombus material was not sent for histopathological analysis, representing a further limitation. Post-procedural angiography demonstrated restoration of TIMI 3 flow in the LAD territory with a significant reduction in thrombus burden (Figure 3B).

The patient was discharged on dual antiplatelet therapy (aspirin 100 mg and ticagrelor) in accordance with current ESC guidelines for STEMI management [7]. Low-dose rivaroxaban (2.5 mg BID) was added, given the high thrombus burden and evidence of benefit in the ACS setting from the ATLAS ACS 2–TIMI 51 trial [8]. We acknowledge that this triple antithrombotic combination carries an increased haemorrhagic risk and that the applicability of the ATLAS ACS 2–TIMI 51 trial findings to a young patient with drug-induced thrombosis and angiographically normal coronary arteries requires careful individual risk-benefit assessment.

Following discharge, the patient was enrolled in a structured cannabis cessation and cardiovascular rehabilitation programme. At 1-year follow-up, the patient reported complete cannabis abstinence



and remained asymptomatic with no recurrent cardiovascular events. Antiplatelet therapy was de-escalated at 12 months, with continuation of aspirin 100 mg and rivaroxaban 2.5 mg BID as a long-term antithrombotic strategy.

Discussion

Cannabis is an increasingly recognised trigger of AMI in young individuals [4,5]. Three mechanisms have been described in the literature:

(1) Coronary vasospasm: CB1 receptor stimulation by Δ -9-THC induces endothelial dysfunction and paradoxical vasoconstriction, leading to focal or diffuse coronary spasm [3,5]. This mechanism predominates in cases with angiographically normal coronary arteries.

(2) Coronary thrombosis: Δ -9-THC exerts a potent prothrombotic effect by upregulating GP IIb/IIIa expression on platelets, enhancing platelet aggregation and promoting thrombotic occlusion of non-atherosclerotic coronary arteries [9]. This mechanism was illustrated in Case 2, where angiography revealed multi-segment thrombosis involving the proximal and mid-LAD and the ostium of the first diagonal branch, without underlying atherosclerosis, spasm, or dissection. When GP IIb/IIIa inhibitor infusion was administered alone, it proved insufficient, requiring additional thrombus aspiration, underscoring the severity of the prothrombotic state. The concomitant cocaine use in Case 2 introduces a critical confounding factor: cocaine is a well-established and potent trigger of coronary vasospasm and thrombosis [10]. Whether the observed thrombosis should be attributed to cannabis, cocaine or their synergistic interaction cannot be determined with certainty from clinical or angiographic data alone, and this ambiguity is acknowledged as a major limitation.

(3) SCAD: Cannabis-associated haemodynamic stress – including tachycardia and blood pressure fluctuations – may predispose to SCAD, a mechanism distinct from classical atherothrombotic disease [5,11]. Neither SCAD nor coronary spasm was documented in the two presented cases.

Case 1 illustrates the potentially fatal nature of cannabis-related AMI, even in young individuals without prior cardiac history. The atypical presentation – epigastric pain, nausea and vomiting rather than classic chest pain – highlights the diagnostic challenge. Clinicians must maintain heightened vigilance for AMI in young patients presenting with atypical gastrointestinal symptoms, particularly when cannabis use is identified. However, without toxicological confirmation of recent cannabis exposure, the causal link in Case 1 remains presumptive.

Case 2 illustrates the management of drug-induced AMI with a heavy thrombus burden. While standard STEMI protocols were followed [7], the incremental benefit of low-dose rivaroxaban in drug-induced AMI without underlying atherosclerosis warrants further study [8].

Regarding optimal post-acute management, no dedicated guidelines exist for cannabis-related AMI. Patient counselling

regarding cannabis cessation is paramount, as cannabis use substantially increases the risk of recurrent cardiovascular events, and abstinence should be strongly encouraged in all affected patients.

Conclusions

Our two cases illustrate that cannabis consumption may trigger AMI even in young adults without traditional cardiovascular risk factors, with coronary thrombosis being the documented mechanism in the surviving patient. The fatal outcome in Case 1 underscores the potentially lethal nature of cannabis-related cardiac events, even in the absence of toxicologically confirmed cannabis exposure or definitive coronary pathology at autopsy. Atypical presentations, including epigastric pain, nausea and vomiting, should prompt active exclusion of AMI, particularly in young cannabis users. Cannabis use history should be systematically elicited in all young patients presenting with unexplained ACS. Patient counselling regarding cannabis cessation and enrolment in rehabilitation programmes should be integrated into post-discharge care. Larger prospective studies are needed to characterise in a better way the true incidence, underlying mechanisms, dose-response relationships and optimal antithrombotic management of cannabis-associated AMI, particularly in the context of polydrug use.

Conflicts of Interest

The authors have each completed the International Committee of Medical Journal Editors Form for uniform Disclosure of Potential Conflicts of Interest. No authors have any potential conflict of interest to disclose.

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Ethics statement

The study was approved by an institutional medical research ethics committee and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the next of kin of the deceased patient (Case 1) and from the patient in Case 2, in accordance with the Declaration of Helsinki.

Consent to Participate

All participants were provided an informed written consent to participate in the study in accordance with the code of ethics of the World Medical Association (Declaration of Helsinki) for experiments on humans.

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