Research Paper

An Analysis of ECG and Troponin I in Patients Using Cannabis

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ABSTRACT

Background: Cannabis-derived substances like marijuana and hashish have various systemic effects, especially on the cardiovascular system. Due to conflicting reports about the cardiac complications of cannabis use, this study aimed to evaluate electrocardiographic (ECG) changes and serum troponin I levels in patients with cannabis intoxication.

Methods: This retrospective cross-sectional study reviewed medical records of patients admitted to the poisoning department at Khorshid Hospital in Isfahan, Iran, from 2011 to 2021, with documented cannabis use. After applying inclusion and exclusion criteria, 98 patients were included in the final analysis. Data on ECG findings, troponin I test results, demographic characteristics, and clinical history were collected and analyzed using SPSS version 25.

Results: Of the 98 patients studied, 37 (37.7%) had T-inversion. Nineteen patients (19.4%; 95% CI: 11.4–27.3%) showed changes associated with ACS, characterized by ST-elevation or ST-depression (with or without T-inversion or other abnormalities). The average age was 28.5 years, with most being male (83.7%), single (65.3%), having a history of substance abuse (74.5%), and co-ingesting other illegal drugs (62.2%).

Conclusion: The results of this study indicate that cannabis use may be linked to changes in ECGs associated with myocardial ischemic injuries. Due to the high incidence of abnormal cardiac findings in ECG and troponin I, evaluations are advised for patients presenting with cannabis intoxication to enable early detection of potential cardiac issues.

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Introduction

Various products, such as marijuana and hashish, are derived from the Cannabis sativa plant. These products can be ingested orally, smoked, and sometimes administered intravenously [1]. Historical evidence suggests that hashish was first used as a toxic substance in Egypt during the 11th century. Despite legal bans, the use of this plant has continued [2]. Recent research indicates that hashish is the most widely used drug among various substances [3-7]. It affects nearly all body systems and shares effects with alcohol, tranquilizers, opioids, and hallucinogens. Its functions include reducing anxiety, relieving pain, and acting as a psychoactive agent. It also increases appetite and produces multiple systemic effects [8]. Many observations regarding hashish's physiological impacts are attributed to delta-tetrahydrocannabinol (THC) [9]. THC acts as a mixed agonist (or antagonist) for two types of G-protein-coupled receptors called cannabinoid receptor 1 and 2 (CB1, CB2). CB1 receptors are primarily located in the central nervous system. However, they are also present in lesser amounts in various organs, including the heart, adrenal glands, fat tissue, liver, lungs, and presynaptic nerve endings. CB2 receptors are primarily found in the immune system [10]. It is believed that cannabis use raises heart rate and blood pressure by stimulating the sympathetic nervous system while reducing activity in the parasympathetic nervous system, leading to increased myocardial oxygen demand and decreased left ventricular ejection fraction, which can cause angina pectoris.

Additionally, there is a reduction in peripheral vascular resistance in skeletal muscles [9-11]. Even at high doses, sympathetic activity tends to decline, and parasympathetic activity increases, potentially causing bradycardia and hypotension that may result in dizziness, syncope, or presyncope [12, 13]. An autopsy study in Norway by Bachs and colleagues found THC was the sole substance detected after the sudden death of six young hashish users [14]. Westover and colleagues showed that hashish use, compared to tobacco and cocaine, likely has a lower link to ischemic heart attacks [15). Thomas also identified connections between marijuana use and myocardial infarction, sudden death, cardiomyopathy, stroke, transient ischemic attack, and arthritis [16). Recently, many cases of acute myocardial infarction following hashish smoking have been reported [1,17,18]. Other studies associate cannabis use with heart arrhythmias [19, 20] and stroke [21]. Some research suggests that cardiovascular problems may not be directly related to hashish use [22, 23]. Compared to other drugs, cannabis users often experience higher rates of coronary artery disease (CAD) and ST-elevation myocardial infarction (STEMI) [24].

Additionally, a few hours after heavy use, vasospasm in the coronary arteries and death have been observed [1, 25, 26]. In contrast, combining tobacco and marijuana increases the risk of cardiovascular and respiratory diseases [27]. A study by Draz and colleagues found that combining cannabis with cigarette smoking might cause heart issues. They also noted that 87% of cannabis-using patients showed elevated ST-segments. Echocardiographic abnormalities, such as wall motion issues and ventricular systolic dysfunction, were present in all patients [28]. Cannabis-related myocardial infarction can occur due to several factors. First, cannabis-induced tachycardia, hypertension, and increased cardiac output stimulate the sympathetic nervous system through norepinephrine release, raising the heart's oxygen demand [29]. Second, oxygen transport capability decreases because of carboxyhemoglobin produced during cannabis use [30]. Other mechanisms include oxidative stress to cells, such as platelet activation, formation of oxidized low-density lipoprotein, and inflammatory responses triggered by free radicals during cannabis consumption [31]. Moreover, peripheral vascular reflexes may cause direct vasospasm [32]. Overall, cannabis use is linked to risky behaviors like high-calorie diets, cigarette smoking, and illegal drug use, all of which have harmful long-term effects on young adults [33]. A recent 2021 study by Shukla and colleagues shows that the duration of the complex QRS in cannabis smokers is shorter compared to cigarette smokers. They also suggest that cannabis may pose potential risks for both acute and chronic heart-related side effects, including those documented in the study.

Materials and Methods

Inclusion criteria

1- Use of cannabis and its derivatives, such as marijuana and hashish.

2- Age group of 45 years or younger.

3- Consciousness

4- Do not start CPR upon arriving at the hospital.

Exclusion criteria

1- Use of medication (drug) can lead to cardiovascular disorders.

2- Other causes of elevated troponin I (e.g., chronic kidney failure, subarachnoid hemorrhage, pulmonary embolism)

3- History of heart diseases, including cardiac infarction or WPW (Wolff-Parkinson-White syndrome)

4- Rhabdomyolysis

5- More than 20% of the file information is missing.

After obtaining the code of ethics from the ethics committee at Isfahan University of Medical Sciences (IR.MUI.MED.REC.1401.339) and permission from Isfahan Khorshid Hospital, we gathered information on patients' age, gender, residence (city or village), occupational status, marital status, education level, cigarette smoking, tobacco use, consumption of drugs or other stimuli, and underlying conditions such as dyslipidemia, diabetes, and hypertension. Their family history was not part of the study. If these data were not recorded in the patient files, we contacted patients via their mobile phones or spoke with their family members to collect the necessary information. During these calls, we obtained patients' consent, and the telephone operator assured us that their information would remain confidential. It is important to note that the positive cannabinoid test observed in the patient is from a urine sample. According to the hospital's standard protocol, urine is collected upon admission and before any treatment. If the patient is unable to produce urine, urinary catheterization is performed to facilitate urination. Urine samples are stored in a refrigerator at 4 degrees Celsius and tested for THC (tetrahydrocannabinol) within six hours, using a dipstick, to determine if the result is positive or negative. The ECG findings—such as ST elevation, ST depression, and T inversion—and Troponin I results (positive or negative) documented in the patient's records are collected and recorded. Finally, all data are entered into SPSS software (version 25). For statistical analysis, indices such as mean, standard deviation, frequency, and percentage are used. Patients were required to participate in the study if they used cannabis or similar substances. In fact, patients admitted to the poisoned ward with an acute history of cannabis use and symptoms of intoxication—such as mood changes, hallucinations, delirium, tachycardia, and other symptoms—who met additional criteria were included in the study, regardless of whether they initially showed signs of heart problems. Generally, an electrocardiogram was performed on all patients with a history of cannabis and related substance use, with some undergoing serial electrocardiograms, as mentioned in the text. It is important to note that patients with a history of cannabis and its derivatives use are referred to the poisoning center when they exhibit acute symptoms, which occur within less than 24 hours. In contrast, patients with complications from chronic cannabis use or those who used cannabis a few days ago are typically referred to psychiatric services. Given the retrospective and cross-sectional design of the study, we included cases specifically mentioning cannabis, its derivatives, and analogs as the cause of hospitalization, and patients did not report a history of other drug use or medical disorders.

Results

In this retrospective cross-sectional study, we examined electrocardiographic (ECG) changes and serum troponin I levels in 98 patients treated for cannabis intoxication at Khorshid Hospital from 2011 to 2021. The clinical and demographic data of these patients were initially reviewed. The population primarily consisted of young individuals, with an average age of 28.58 years (standard deviation 6.07, median 28 years, mode 28 years, and an age range of 19-44 years). The study primarily included males: approximately 82 males (82.7%) and 16 females (16.3%). This reflects typical patterns seen in substance use. Regarding marital status, 34 patients (34.7%) were married, and 64 (65.3%) were single, which may suggest that these differences are related to behavioral and social factors. Only two patients (2.0%) were from rural areas, while nearly all others (96-98.0%) were from urban areas. Intentional poisoning accounted for 49 cases (50.0%), unintentional poisoning for 14 cases (14.3%), and undetermined poisoning for 35 cases (35.7%). These cases indicate that poisoning involved both intentional and unintentional exposures. Cannabis was the sole drug involved in poisoning for 37 patients (37.8%), while in 61 patients (62.2%), poisoning involved cannabis along with other substances. The most common substances detected in urine toxicology tests were morphine, amphetamine, and benzodiazepines. Eighty cases (81.6%) involved exposure to marijuana, hashish, or grass, while the remaining 18 patients (18.4%) were poisoned by cannabis and its derivatives. An overview of the baseline clinical and demographic characteristics is provided in Table 1.

Overall, 45 individuals (45.9%) had normal ECGs, while 53 patients (54.1%) showed abnormalities. The most common finding was T-wave inversion, observed in 37 patients (37.7%), either alone or in conjunction with other changes. Nineteen patients (19.4%; 95% CI: 11.4–27.3%) had alterations related to ACS, characterized by ST-elevation or ST-depression, with or without T-inversion or other abnormalities. This suggests that this group is at higher risk for ischemic

Table 1. Demographic and Clinical Characteristics of Patients at Baseline (N=98)

Characteristic	*N (%) or Mean (SD)
Age (years)	
Mean (SD)	28.58 (6.07)
Median (Mode)	28 (28)
Range	19–44
Gender	
Male	82 (83.7)
Female	16 (16.3)
Marital Status	
Single	64 (65.3)
Married	34 (34.7)
Residence	
Urban	96 (98.0)
Rural	2 (2.0)
Occupation	
Freelance	58 (59.2)
Unemployed	24 (24.5)
Employed	15 (15.3)
Student	1 (1.0)
Education	
Unknown	39 (39.8)
Diploma	32 (32.7)
Below Diploma	13 (13.3)
Bachelor's	11 (11.2)
Master's	3 (3.1)
History of Addiction	
Yes	73 (74.5)
No	20 (20.4)
Unclear	5 (5.1)
History of Suicide	
Yes	12 (12.2)
No	85 (86.7)
Unclear	1 (1.0)
History of Previous Poisoning	
Yes	6 (6.1)
No	91 (92.9)
Unclear	1 (1.0)
Type of Poisoning	
Intentional	49 (50.0)
Unintentional	14 (14.3)
Unknown	35 (35.7)
Poisoning Agent	
Cannabis Alone	37 (37.8)
Cannabis + Other Drugs	61 (62.2)
Entry Criteria	
Cannabis and Derivatives	18 (18.4)
Marijuana/Hashish/Grass	80 (81.6)

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*Note: Percentages are rounded to one decimal place. SD = Standard Deviation.

events. For example, eight patients (8.2%) had isolated ST-elevation; one of these patients had a negative troponin test, and the follow-up ECG showed T-wave inversion. Two patients (2.0%) had only ST-depression and were discharged. Three patients (3.1%) experienced ST-elevation with T-inversion, two (2.0%) had ST-depression with T-inversion, and one of these patients had two negative troponin I tests; a follow-up ECG showed T-inversion and sinus tachycardia. Four patients (4.1%) exhibited T-inversion with sinus bradycardia or hyperacute T-waves; two patients (2.0%) had ST-elevation, T-inversion, and sinus tachycardia or tall T-waves. Five patients (5.1%) showed isolated abnormalities such as hyperacute T-waves, poor R-wave progression, or sinus tachycardia; one was followed by an ECG indicating features of complete ACS (ST-elevation, ST-depression, T-inversion). One patient (1.0%) with ST-depression, wide QRS, and prolonged QT had a positive troponin test and died. Seven patients (7.1%) had second ECGs; two of these were normal, one showed isolated T-inversion, and four had changes associated with ACS. The third ECG (1.0%) revealed T-inversion. Overall, 80 patients (81.6%) were discharged by a doctor, 15 (15.3%) left AMA, and one patient (1.0%) was transferred to the psychiatric ward. Table 2 summarizes the initial ECG findings, follow-ups, and outcomes.

Table 3 summarizes the second ECG results, along with links to the initial ECGs, troponin results, and outcomes.

The final results of the study are shown in Table 4. Most patients performed well overall; however, one death occurred among the complicated cases, and no deaths were observed in the non-ACS groups.

Discussion

A retrospective cross-sectional study assessed ECG changes and serum troponin I levels related to cardiotoxicity in 98 patients who experienced cannabis poisoning after admission to Khorshid Hospital's poisoning ward in Isfahan, Iran, from 2011 to 2021. The study found that 54.1% of patients (53 out of 98) showed ECG changes, mainly indicative of acute coronary syndrome (ACS). T-wave inversions were seen in 37.7% of patients. These findings support previous research linking cannabis use to heart problems. Fourteen patients (14.3%) displayed ST-elevation on their ECGs during their first, second, or third tests, suggesting possible myocardial ischemia. These results are consistent with Draz et al., who reported higher rates of ST-elevation myocardial infarction (STEMI) among cannabis-positive patients compared to controls without substance use. In their cohort of 138 young men with acute myocardial infarction (AMI), 47.8% of cannabis-positive patients showed ischemic wall motion abnormalities, and none had normal coronary arteries, highlighting a strong link between cannabis use and coronary ischemia [28]. Unlike this study, which involved only male patients with confirmed AMI, our research included both genders and a wider range of cannabis intoxication cases, which may explain the lower prevalence of ACS findings (19.4% vs. 47.8%). Still, the presence of ST-elevation supports the idea that cannabis can trigger acute ischemic events, possibly through mechanisms

Table 2. Summary of Initial ECG Results, Follow-Up ECGs, and Patient Outcomes (N=98).

ECG Finding Category	N (%)	Follow-up ECG (N; Key Changes)	Outcomes (Discharged / AMA / Transferred / Death)
Normal	45 (45.9)	2; 1 T-Inversion, 1 Normal	35 / 9 / 1 / 0
Isolated T-Inversion	25 (25.5)	2; 1 Persistent T-Inversion, 1 Normal	19 / 5 / 1 / 0
Isolated ST-Elevation	8 (8.2)	1; ST-Elevation + T-Inversion	7 / 1 / 0 / 0
Isolated ST-Depression	2 (2.0)	None	2 / 0 / 0 / 0
ST-Elevation + T-Inversion	3 (3.1)	None	3 / 0 / 0 / 0
ST-Depression + T-Inversion	2 (2.0)	1; T-Inversion + Sinus Tachycardia	2 / 0 / 0 / 0
ST-Elevation + ST-Depression + T-Inversion	1 (1.0)	None	1 / 0 / 0 / 0
T-Inversion + Other (e.g., Bradycardia/Tall-T)	4 (4.1)	None	4 / 0 / 0 / 0
ST-Elevation + T-Inversion + Other (e.g., Tachycardia/Tall-T)	2 (2.0)	None	2 / 0 / 0 / 0
ST-Depression + Other (Wide QRS/Long QT)	1 (1.0)	None	0 / 0 / 0 / 1
Isolated Other (e.g., Tall-T/poor R-Progression/Tachycardia)	5 (5.1)	1; ST-Elevation + ST-Depression + T-Inversion	5 / 0 / 0 / 0

*Note: AMA = Against Medical Advice. Percentages are based on the total unless stated otherwise.

Table 3. Summary of Second ECG Findings, Initial ECG Context, Troponin I Results, and Patient Outcomes (N=7).

Patient Group / Finding	N (%)	Initial ECG	Troponin I (First Round)	Key Second ECG Changes	Outcome
Minimal changes	2 (28.6)	minimal	1 Negative (both rounds); 1 Not Tested	1 T-Inversion + Sinus Tachycardia; 1 ST-Elevation	Discharged
Normal Second ECG	2 (28.6)	1 Normal; 1 T-Inversion	Not Tested	Normal (1 with normal third ECG)	Discharged
Isolated T-Inversion	1 (14.3)	T-Inversion	Not Tested	T-Inversion	Transferred
ST-Depression + T-Inversion	1 (14.3)	Normal	Not Tested	ST-Depression + T-Inversion	Discharged AMA
ACS Triad (ST-Elevation + ST-Depression + T-Inversion)	1 (14.3)	Sinus Tachycardia	Not Tested	ST-Elevation + ST-Depression + T-Inversion	Discharged

*AMA = Against Medical Advice, ACS = Acute Coronary Syndrome.

such as increased myocardial oxygen demand from tachycardia or coronary vasospasm [9, 29]. The occurrence of T-wave inversion (37.7%) is notable. Shukla et al. conducted a cross-sectional comparison of ECG parameters among cannabis users, tobacco smokers, and non-smokers, reporting a shorter QRS complex duration in cannabis users compared to tobacco smokers, with no significant difference in ST-segment duration [34]. Interestingly, our study included one patient with a wide QRS and a prolonged QT interval, contrasting with Shukla et al.'s finding of a shorter QRS duration. This difference might be due to variations in study groups (acute versus chronic use) or confounding factors such as co-ingestion of other drugs, especially since 62.2% of our patients were poly-substance users. Additionally, other ECG abnormalities like sinus tachycardia, sinus bradycardia, tall T-waves, and poor R-progression in 12 patients suggest that cannabis can cause various electrophysiological disturbances beyond ischemic changes, possibly mediated through effects on the autonomic nervous system [12]. In a case study, Mittleman et al. identified a 4.8-fold increased risk of AMI within 60 minutes of cannabis exposure, with the risk decreasing rapidly afterward [29]. This temporal link supports our findings, as the acute presentation of our patients (hours after drug use) can explain the observed ECG abnormalities consistent with ischemia. The predominance of male patients (83.7%) and a history of substance abuse (74.5%) align with Mittleman et al.'s observation that cannabis users are mainly male and often current smokers—both well-known cardiovascular risk factors [29]. However, our study did not specify the exact timing of cannabis consumption for each patient. The cardiovascular responses to cannabis mainly result from activation of cannabinoid receptors CB1 and CB2, which modulate sympathetic and parasympathetic nervous system

Table 4. Summary of ECG Subgroups, Troponin I Results, Follow-up ECGs, and Patient Outcomes (N=98)

Sub-Group	N (%)	Second ECG (N; Key Changes)	First Troponin I	Second Troponin I	Outcome
ST-Elevation	8 (8.2)	1; ST-Elevation & T-Invert	1 Negative (12.5%)	-	Discharged
Only ST-Depression	2 (2.0)	-	-	-	Discharged
ST-Elevation & T-Invert	3 (3.1)	-	-	-	Discharged
ST-Depression & T-Invert	2 (2.0)	1; Sinus Tachycardia & T-Invert	1 Negative	1 Negative	Discharged/transferred
ST-Elevation + ST-Depression + T-Invert	1 (1.0)	-	-	-	Discharged
ST-Depression & Other Findings	1 (1.0)	-	-	-	Death
ST-Elevation & T-Invert & Other Findings	2 (2.0)	-	-	-	Discharged
Normal ECG	45 (45.9)	2; 1 T-Invert & ST-Depression, 1 Normal	1 Positive	-	Discharged
Only T-Invert	25 (25.5)	2; 1 Normal, 1 T-Invert	1 Negative	-	Discharged
T-Invert & Other Findings	4 (4.1)	-	-	-	Discharged
Other Findings	5 (5.1)	1; ST-Elevation, ST-Depression, T-Invert	-	-	Discharged

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*Note: ACS = Acute Coronary Syndrome (ST-elevation/ST-depression ± T-inversion/other).

functions [9, 10]. The combination of tachycardia and hypertension appears about 15 minutes after peak THC plasma levels and increases myocardial oxygen demand, potentially triggering ischemic events in vulnerable individuals [9, 11]. Furthermore, cannabis smoking may reduce oxygen-carrying capacity via carboxyhemoglobin formation and induce coronary vasospasm, both contributing to ischemic risk [31, 33]. The T-wave inversion and other ECG changes observed in our study may reflect these pathophysiological processes, along with oxidative stress and platelet activation caused by cannabis [31]. The concurrent use of other substances, such as tobacco or additional drugs (62.2% of our cohort), could intensify these effects, as studies show increased cardiovascular risk with combined tobacco and cannabis use [27]. Our findings support routine ECG and troponin I testing in patients presenting with cannabis intoxication, especially those showing sympathomimetic symptoms like tachycardia and hypertension, which could indicate tissue hypoxia from respiratory depression. The detection of T-wave inversion in 37.7% of patients and ACS-related findings in 19.4% highlights the importance of close monitoring for cardiovascular complications. ECG, being a quick and inexpensive tool, can facilitate early detection of ischemic changes, as shown by Shukla et al. [34]. Repeating ECG and troponin tests should be considered for patients with inconclusive initial results, as some later developed ACS features upon serial assessment. Sinus tachycardia and bradycardia were observed in our study, while Harding et al. reported premature atrial contractions, episodes of supraventricular tachycardia, premature ventricular contractions, and non-sustained ventricular tachycardia (NSVT) [35]. Data from 434,104 American adults indicate that daily cannabis use increases the adjusted odds ratios (aORs) for cardiovascular outcomes, including myocardial infarction (aOR 1.25), stroke (aOR 1.42), and combined CAD, MI, and stroke (aOR 1.28) [36]. Our findings support these results by documenting ECG abnormalities related to ACS, further emphasizing cannabis's potential harmful cardiovascular effects during acute use. The higher odds ratios among non-smokers and younger individuals highlight the significance of our findings in a predominantly young male population. Finally, studies on biomarkers like creatinine, BUN, and BNP, as discussed by Jean-Louis et al. [37], could help identify additional risk factors for cardiovascular side effects in cannabis users.

Several limitations are present. Troponin I, an important biomarker for detecting heart muscle injury, was rarely used during the trial. Only five patients (5.1% of 98) underwent testing, and one patient (1.0% overall; 20% of those tested) showed a positive result, indicating potential heart damage. The remaining 93 patients (94.9%) did not undergo initial troponin testing, and repeat testing was even less frequent, with only one patient (1.0% overall) being tested again. The fact that over 62% of patients used multiple substances introduces confounding factors that could influence ECG results; for example, amphetamines may affect the electrocardiogram. The lack of specific timing details regarding cannabis use before symptom onset

prevents establishing a direct temporal connection between cannabis consumption and ECG changes. Since the study was conducted at a single institution, its findings have a limited scope. Exploring the dose-response relationship between cannabis use and cardiovascular effects, along with the impact of different methods of administration (e.g., smoking versus oral), would provide clearer insights into the underlying mechanisms.

Conclusion

Our research indicates that the most common ECG changes associated with cannabis use are T-wave inversion and ST elevation, with 54.1% of patients showing ECG abnormalities. The average age of patients was 28.5 years, and all were male. In most cases, poisoning resulted from the use of marijuana, hashish, and grass (rather than cannabis or its derivatives), and many had also used other illegal drugs.

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Conflicts of Interest

The authors report there are no competing interests to declare.

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