

Review

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Posted Date: 8 April 2024

doi: 10.20944/preprints202404.0575.v1

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Review

Cannabinoids and Cardiac Rhythms: Unveiling the Relationship between Cannabis Use, ECG Changes, and Arrhythmias

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Abstract: Understanding how cannabis affects our heart is like navigating through a maze of conflicting findings. While it's known for its diverse uses, from medicinal to industrial, cannabis also raises red flags regarding its impact on heart health. This review dives into the intricate relationship between cannabis use and cardiovascular well-being, particularly focusing on changes seen in electrocardiogram (ECG) readings, which serve as vital indicators of heart function and rhythm. When someone tokes up, their heart might race temporarily, but chronic cannabis use paints a darker picture, with potential risks of heart attacks and strokes looming on the horizon. The ECG alterations observed in cannabis users, such as irregular heartbeats and unusual wave patterns, hint at disruptions in the heart's normal workings. Furthermore, cannabis has been linked to prolonging the QT interval in the heart's electrical cycle, potentially setting the stage for dangerous arrhythmias. Amidst this mixed bag of effects, some studies even suggest that tiny amounts of THC, the main psychoactive component of cannabis, might lend a helping hand to the heart. However, caution flags are raised, especially for vulnerable groups. While researchers strive to unravel the mysteries of cannabis and the heart, limitations in current studies remind us that we're still far from having all the answers. Moving forward, future investigations should delve deeper into understanding how cannabis tinkers with our heart's inner workings, especially across different demographics. Armed with this knowledge, we can better tailor interventions to safeguard the heart health of cannabis users.

Keywords: cannabis; electrocardiogram (ECG); arrhythmias; myocardial ischemia; cardiac ion channels

Methods

In this comprehensive review article, we systematically analyzed relevant studies to explore the intricate relationship between cannabis use, electrocardiogram (ECG) changes, and arrhythmias. Our research methodology involved a meticulous search across various databases, including PubMed, Google Scholar, and relevant academic journals, using keywords such as "cannabis," "ECG," "arrhythmias," and related terms. We conducted a thorough screening process to identify studies that investigated the acute and chronic effects of cannabis on cardiac rhythms, ECG parameters, and the incidence of arrhythmias. Furthermore, we critically evaluated the methodological rigor of each study, considering factors such as study design, sample size, patient demographics, and outcome measures. Through a comprehensive synthesis of the literature, we elucidated the mechanisms underlying cannabis-induced ECG changes, including sympathetic stimulation, myocardial ischemia, and direct effects on cardiac ion channels. Additionally, we discussed the clinical implications of these findings and proposed potential management strategies for cannabis-induced cardiac arrhythmias.

Introduction to Cannabis Use and Cardiovascular Health

Cannabis sativa, commonly known as marijuana, has a rich history of varied uses, from therapeutic to industrial applications. The plant contains over 500 chemical compounds, including cannabinoids like THC and CBD, with distinct effects on the human body [1,2]. While THC is responsible for the psychoactive effects of cannabis, CBD offers potential therapeutic benefits without inducing intoxication [3]. Research indicates that cannabis derivatives, such as cannabinoids, have shown promise in treating various ailments, including cardiovascular conditions, chronic pain, and diabetes [4]. Despite its therapeutic potential, concerns about the cardiovascular effects of cannabis have surfaced, prompting further investigation into its impact on heart health [5].

As cannabis consumption becomes more prevalent, understanding its implications for cardiovascular health is essential. Research suggests that acute cannabis use can lead to transient cardiovascular effects such as increased heart rate, transient hypertension, and orthostatic hypotension. Moreover, chronic cannabis use has been associated with long-term cardiovascular risks, including an increased likelihood of myocardial infarction, stroke, and cardiovascular-related mortality. However, the precise mechanisms underlying these effects remain incompletely understood.

Cannabis-Induced Alterations in ECG Parameters

a. Heart Rate Variability

Acute cannabis consumption has been associated with alterations in heart rate variability (HRV), primarily through the modulation of sympathetic and parasympathetic activity [6]. Suraj K Nayak et al. studied the effect of cannabis consumption on the autonomic nervous system (ANS) and cardiac physiology of Indian women who labor in rice fields. Data were analyzed using electrocardiogram signals and RR intervals. The study found that women who abused cannabis had a lower risk of isometric recurrence and heart rate stability. In addition, changes in sympathovagal balance and parasympathetic activity in the heart were detected. These findings have implications for counseling interventions designed to encourage women to stop using cannabis [7].

Cannabis use has been linked to cardiovascular complications, including myocardial infarction and congestive heart failure [8]. Studies have shown that cannabis can decrease HRV by increasing sympathetic nervous system activation, leading to bradycardic effects and changes in HRV parameters in both humans and animal models [9]. The impact of cannabis on HRV is complex, involving changes in autonomic balance and potential effects on baroreflex mechanisms [10]. Overall, cannabis consumption can disrupt the normal variability in heart rate, potentially contributing to cardiovascular issues through alterations in sympathetic and parasympathetic regulation.

b. Changes in P Wave, QRS Complex, and T Wave Morphology:

The reduced QRS-complex duration may indicate early depolarization of ventricles, leading to abnormal electrical impulses originating faster above the ventricles. The study included 50 participants aged 18-40, with the mean age of cannabis smokers being younger than tobacco smokers and non-smokers [11]. Additionally, cannabis has been linked to adverse cardiovascular effects, including Brugada electrocardiogram pattern induction [12]. The Brugada syndrome, characterized by specific ECG changes, has been reported in individuals following cannabis use, suggesting a potential association between cannabis consumption and cardiac abnormalities [13]. Compared to the other groups, the cannabis group did not exhibit any statistically significant variations in ST-segment duration. Nonetheless, compared to non-smokers, the ST-segment duration was shorter in tobacco smokers [11].

The one-case report from Kandah et al. illustrates the unusual illness known as pseudo-Wellen's syndrome, which is brought on by marijuana usage. It is characterized by angina and an EKG that displays biphasic or deeply inverted T waves in V2-V4. Critical blockage of the proximal left anterior descending artery is linked to the condition. Acute coronary syndrome (ACS) is suspected in the early

stages of Wellen's syndrome based on a structured diagnostic method that includes the history, physical examination, electrocardiogram (EKG), and cardiac biomarkers. It is important to assess traditional risk indicators in order to properly stratify risks. Crucial to the diagnosis is the EKG, which displays characteristic signs of Wellen's syndrome, including biphasic T waves in leads V2-V3 and symmetric and deeply inverted T waves in precordial leads V1-V4 [14].

Premature ventricular contractions and other reversible ECG abnormalities affecting the P and T waves have also been linked to cannabis use. It is debatable, though, whether these modifications are directly linked to drug consumption or result from elevated sympathetic activity and its subsequent hemodynamic effects [15]. Giacomo Mugnai and colleagues' case study clearly demonstrates sinus tachycardia with just negative T waves in the inferior leads. After a few hours, the heart rate dropped and the repolarization anomalies disappeared. The pathophysiological process is not well understood. We could hypothesize that there is an imbalance between the increased oxygen demand (caused by the elevated heart rate and blood pressure) and the decreased oxygen supply (caused by the rise in carboxyhemoglobin) [16].

They believe that the considerable hyperactivation of the sympathetic nervous system with secondary T wave changes is more suggestive. It is well established that ventricular arrhythmias, T wave inversion, and sinus tachycardia are all linked to paroxysmal sympathetic hyperactivity [16].

In another study, 77% of participants showed composite significant abnormalities, with the greatest proportion in the "Never used marijuana" group at 8%. The ECG abnormalities at year 20 were characterized by marijuana use history by Julian Jakob and his teams. Composite mild anomalies were more common, with 944 observations (37%) in the category. Major Q wave abnormalities, isolated ST or T abnormalities, left ventricular hypertrophy, complete or intermittent right bundle branch block (RBBB), nonspecific intraventricular block, atrial fibrillation, supraventricular tachycardia, and major QT prolongation are examples of major abnormalities. Deviations and fluctuations in the ECG waveform are examples of minor abnormalities. The results point to a possible link between marijuana use and a higher frequency of aberrant ECG readings, especially in those who had previously or currently used marijuana [17].

In another analysis by Nikhil Patel et al., conducted on a cohort of 3,600 participants from the Third National Health and Nutrition Examination Survey, it was demonstrated that approximately 24.3% (875 individuals) reported ever using cannabis, while around 20.4% (735 individuals) exhibited abnormal p-wave axis (aPWA), which is considered a marker of atrial myopathy. After adjusting for various factors, the study revealed that both current and former cannabis users displayed increased odds of aPWA compared to those who had never used cannabis. Specifically, the odds ratios (OR) for aPWA were 1.27 (with a 95% confidence interval (CI) of 1.03-1.56) for ever-users and 1.28 (with a 95% CI of 1.03-1.59) for former users. Furthermore, individuals reporting having used cannabis at least 10 times in their lifetime had a 40% higher likelihood of aPWA, with an OR of 1.40 (95% CI: 1.07-1.84). Importantly, this association was particularly pronounced among individuals with a history of cardiovascular disease, showing an OR of 4.11 (95% CI: 1.35-12.52) and an interaction p-value of 0.03, indicating a significant interaction between cannabis use and cardiovascular disease history concerning aPWA [18].

In another clinical study conducted by Selman Yeniocak et al., the effects of synthetic cannabinoids (SCs) on the cardiovascular system (CVS) were investigated. In this study, which included a total of 148 individuals divided equally between the patient and control groups, significant differences were observed in various cardiovascular parameters between the two groups. The systolic and diastolic arterial blood pressures (ABPs) of the patient group were found to be statistically lower than those of the control group ($P < 0.001$). Moreover, P-wave width and amplitude were significantly higher in the patient group compared to the control group ($P: 0.027$ and $P: 0.004$, respectively), while QRS width was significantly higher in the patient group's ECGs compared to the control group, and T-wave amplitude was significantly lower ($P: 0.045$ and $P < 0.001$, respectively). Notably, ST elevation was present in 16.2% of subjects in the patient group, while none were observed in the control group ($P < 0.001$) [19]. another research project that looked into the electrocardiogram characteristics in patients using synthetic cannabis. The results showed that there were notable

variations in a number of electrocardiographic parameters between the patient and control groups, suggesting that the ingestion of synthetic cannabinoids may have an impact on the cardiovascular system [20]. First, P wave dispersion was seen to be greater in the sick group (34 ± 9.4 vs. 29.5 ± 6.6 , $p=0.02$) than in the control group. P wave dispersion has been linked to a higher incidence of atrial fibrillation because it indicates heterogeneity in atrial conduction. This implies that synthetic cannabis may have a pro-arrhythmic influence on the electrical characteristics of the atrium. In addition, the patient group had considerably greater QT interval, corrected QT, and QT dispersion than the control group [20].

In another case report by Tolga Han Efe et al., the reported case highlights a concerning association between synthetic cannabinoid (SC) use and the development of atrial fibrillation (AF). The discussion highlights a case report demonstrating a potential association between synthetic cannabinoid (SC) use and the development of atrial fibrillation (AF). The case involved a 23-year-old man who experienced symptoms of AF after smoking a mixture of herbal substances and SCs known as Bonzai. While previous reports have linked marijuana use to AF, this case adds to the limited instances implicating SCs in AF development. The underlying mechanisms of SC-induced AF remain unclear, but it's suggested that SCs may disrupt the autonomic nervous system balance and alter cardiac electrophysiological properties [21].

Another study by Oluwale Adegbala et al. investigates the relationship between left ventricular dysfunction, sympathetic nervous system activation, and atrial fibrillation (AF), shedding light on the potential impact of cannabis usage on AF occurrence in heart failure patients. The study used data from the Healthcare Cost and Utilization Project-National Inpatient Sample for HF patients in 2014, and found that 0.45% of the estimated 3,950,392 HF admissions were cannabis users. Propensity-matched groups of 3,548 patients each revealed that cannabis users had a reduced risk of AF than nonusers (19.08% vs. 21.39%; AOR 0.87 [0.77 to 0.98]). These findings offer light on a novel component of the link between cannabis usage and cardiovascular health, suggesting that cannabis may protect against AF in HF patients, even after adjusting for various co-morbidities, age, insurance type, and socioeconomic status [22].

The investigation not only provides statistical evidence but also underscores the physiological mechanisms underlying the observed association. Left ventricular dysfunction often triggers sympathetic nervous system activation, exacerbating the risk of AF. While cannabis has been associated with biphasic effects on the autonomic nervous system, with increased sympathetic activity at low doses and inhibitory effects at higher doses, its impact on AF occurrence in HF patients remained unclear [22].

Another review. The study by John R. Richards et al. is corroborated by data from several studies, with cannabis-induced tachycardia being recorded in approximately 61% of articles. Observed dysrhythmias include atrial fibrillation, atrial flutter, atrioventricular block, premature ventricular contractions, premature atrial contractions, ventricular tachycardia, and ventricular fibrillation. Furthermore, electrocardiographic changes such as ST segment elevation, P-wave and T-wave alterations have been seen. Notably, the overall prevalence of cannabis-induced dysrhythmias is quite low [23].

The study by David O. Andonian et al. presents a case series that offers insights into the acute cardiovascular effects of synthetic cannabinoids (SCB), particularly during a concentrated outbreak in the South Bronx. The toxidrome observed in patients following SCB use was characterized by marked bradycardia, hypotension, drowsiness, and somnolence. Despite the significant cardiovascular dysfunction, all patients experienced spontaneous resolution of symptoms and normalization of vital signs with intravenous fluid hydration alone. Importantly, there was no evidence of neurological impairment, indicating that the observed cardiovascular changes did not affect global neurologic function. This suggests that SCB intoxication can independently depress the cardiovascular system without significant neurological consequences. In conclusion, while this case series has a limited sample size, it highlights the potential cardiovascular toxicity of SCB use, which may be underreported in current literature [24].

c. QT Interval Prolongation

Cannabis abuse has been associated with cardiac effects, including QT prolongation in electrocardiograms (ECGs) [25,26]. Studies have shown that cannabis products like bhang can alter cardiac electrophysiology, leading to ECG variations [27]. Additionally, research indicates that cannabis use, particularly with high concentrations of cannabidiol (CBD), can impact action potential duration, hERG potassium channels, and delay rectifier potassium currents, potentially leading to QT interval lengthening in Patients [28].

Additionally, marijuana use has been linked to long QTc intervals by inhibiting potassium channels, akin to Long QT Syndrome type 2 (LQTS2). Genetic testing revealed a mutation in genes associated with THC-induced QTc prolongation, highlighting the interplay between genetics and cannabis use in arrhythmia development [28].

THC can cause Tdp through two mechanisms: chronic THC increases parasympathetic and reduced sympathetic activity, leading to bradycardia and prolongation of QTc. Marijuana use also affects QTc by inhibiting the delayed rectifier potassium channel, similar to LQTS2. Genetic testing in a patient revealed a mutation in genes causing THC-induced QTc prolongation [29–31].

d. Arrhythmia

In post-MI, the risk of VT/ventricular fibrillation (VF) has not been shown to differ in reported marijuana users vs. non-users. However, a lower risk of in-hospital mortality and a trend towards a lower risk of AF was demonstrated. In younger patients without a history of acute coronary syndrome, cannabis use was shown to be associated with an increased risk of VF, AF, atrial flutter, pre-excitation syndromes, and long-QT syndrome [32].

A study of three cases involving cannabis-related arrhythmia found that ambiguous combinations of arrhythmia should raise suspicion of underlying cannabis abuse, where clinically appropriate. The temporal relationship between drug use and the onset of symptoms suggests a strong association. The cannabis group did not show significant differences for ST segment duration in comparison with the other groups. The outcome may reflect a protective role of cannabis in this segment of ECG, although this possibility has not been investigated in the current research [33].

Cannabis-related arrhythmia can be multiform regarding their presentation, and ambiguous combinations of arrhythmia should raise suspicion of underlying cannabis abuse, where clinically appropriate. The temporal relationship between drug use and the onset of symptoms suggests a strong association. The cannabis group did not show significant differences for ST segment duration in comparison with the other groups. However, a lower risk of in-hospital mortality and a trend towards a lower risk of AF was demonstrated. In younger patients without a history of acute coronary syndrome, cannabis use was shown to be associated with an increased risk of VF, AF, and LVEF [33].

The management of THC vaping or dabbing-related Tdp remains unclear. Isoproterenol, a nonspecific β_1/β_2 -adrenoceptor agonist, may provide temporary relief by increasing heart rate, shortening the QTc interval and refractory period. Long-term management requires a shared decision-making process with ICD and pharmacologic therapies. Beta blockers like nadolol may reduce recurrence of ventricular arrhythmias by reducing repolarization dispersion, decreasing after depolarizations, and sometimes shortened QTc [34,35].

The case report by Talha Ahmad and colleagues discusses a 52-year-old woman who experienced sudden cardiac arrest (SCA) after consuming synthetic cannabinoids (SC), also known as K2 or spice. Despite achieving return of spontaneous circulation, the patient suffered recurrent episodes of ventricular fibrillation (VF) and torsades de pointes (TdP), emphasizing the severity of SC-induced arrhythmias [36].

A significant finding was the presence of prolonged QTc intervals on electrocardiograms (ECGs) following the SCA events, consistent with previous reports linking SC use to QTc prolongation and arrhythmias. The mechanism involves SC stimulating human cannabinoid receptors (CB1), leading to alterations in cardiac ion channels and QTc prolongation [31,37].

A retrospective analysis of the Nationwide Inpatient Sample from 2010 to 2014 was conducted to explore the connection between cannabis use disorder (CUD) and arrhythmia hospitalization.

CUD affected 2.6% of the 570,556 inpatients who were primarily diagnosed with arrhythmia. Notably, the most common arrhythmia among cannabis users was atrial fibrillation (42%), followed by various arrhythmias (24%), and atrial flutter (8%). Patients with CUD were more likely to be young (15-24 years, OR: 4.23), male (OR: 1.70), and African American (OR: 2.70). Importantly, CUD was linked to a 47%-52% higher chance of arrhythmia hospitalization in the younger population (aged 15-34 years), with odds ratios of 1.28 (95% CI: 1.229-1.346) and 1.52 (95% CI: 1.469-1.578), respectively. These results were modified to account for potential confounders, such as other drugs [38].

Based on a retrospective analysis of hospitalizations among teenagers with cannabis use disorder (CUD) from 2003 to 2016, it was found that out of 876,431 weighted hospitalizations, 4043 (0.5%) were associated with arrhythmias. Older teenagers (17–20 years) experienced significantly higher prevalence of arrhythmias (90.7%) compared to younger teenagers (13–16 years), with males constituting 67.7% of arrhythmia cases. Caucasians represented 58.7% of hospitalizations with CUD-related arrhythmias. Notably, the prevalence of arrhythmias increased from 229 per 100,000 in 2003 to 959 per 100,000 in 2016 among CUD-related hospitalizations, with atrial fibrillation being the most frequent arrhythmia (105 per 100,000) [39].

Mechanisms Underlying Cannabis-Induced ECG Changes

Cannabis consumption has been associated with alterations in electrocardiogram (ECG) parameters, reflecting underlying changes in cardiac function and rhythm. Understanding the mechanistic pathways through which cannabis exerts its effects on the cardiovascular system is essential for elucidating the etiology of cannabis-induced ECG changes and for informing clinical management strategies. Here, we discuss three key mechanisms underlying cannabis-induced ECG changes: sympathetic stimulation and autonomic dysfunction, myocardial ischemia and coronary artery vasospasm, and direct effects on cardiac ion channels.

a. Sympathetic Stimulation and Autonomic Dysfunction

Cannabis impacts cardiovascular function primarily through autonomic nervous system modulation. Acute cannabis use induces sympathetic stimulation, elevating heart rate, blood pressure, and vascular resistance via cannabinoid receptor activation [40,41]. This stimulation triggers catecholamine release, including adrenaline and noradrenaline, affecting cardiac conduction pathways. Consequently, ECG changes like heart rate variability, PR interval prolongation, and T wave morphology alterations may occur due to sympathetic overactivity [42,43]. Despite controversies over cannabis's direct association with adverse cardiovascular events like acute myocardial infarction and stroke [44].

b. Myocardial Ischemia and Coronary Artery Vasospasm

THC, the main psychoactive compound in cannabis, has been linked to cardiovascular effects such as arrhythmias, acute coronary syndrome, and sudden cardiac death [41]. Cannabis and synthetic cannabinoids have been associated with adverse cardiovascular events like myocardial infarction, cardiomyopathy, arrhythmias, stroke, and cardiac arrest, potentially due to their impact on thrombosis, inflammation, and atherosclerosis [40]. Additionally, cannabis use has been connected to spontaneous coronary artery dissection, a rare cause of acute coronary syndrome, with cases showing ST-segment elevation myocardial infarction and coronary artery dissection, emphasizing the need for awareness of these complications among physicians [45].

Coronary artery vasospasm induced by cannabis can trigger myocardial ischemia and infarction, manifesting as ECG changes like ST segment elevation or depression, T wave inversion, and pathological Q waves, as observed in multiple cases [43,46,47]. These ECG alterations may resolve post-cannabis cessation, but recurrent ischemic episodes can lead to myocardial damage and remodeling [48]. Moreover, cannabis's pro-thrombotic and pro-inflammatory properties can worsen myocardial ischemia, potentially contributing to adverse cardiovascular consequences [49].

c. Direct Effects on Cardiac Ion Channels

Cannabis and its constituent cannabinoids exert direct effects on cardiac ion channels, disrupting normal cardiac electrophysiology and contributing to ECG abnormalities. Dmytro isaev et al. used the patch-clamp technique to explore the effects of cannabidiol (CBD) on main ion currents in rabbit ventricular myocytes. CBD, a non-psychotropic cannabinoid present in cannabis, has a variety of medicinal effects, but its impact on the cardiovascular system is little understood. The study discovered that CBD blocks voltage-gated sodium (Na⁺) and calcium (Ca²⁺) channels, as well as rapidly and slowly activated delayed rectifier potassium (K⁺) channels. However, it has no substantial effect on the inward rectifier potassium (IK1) and transient outward potassium (Ito) currents. CBD's effects are slow and reach steady-state levels in 5-8 minutes [50]. The inhibition of ion currents by CBD suggests potential cardiac electrophysiological effects, indicating caution in administering CBD to individuals with cardiac channelopathies or those using drugs affecting heart rhythm or contractility.

The cannabinoid receptors CB1 and CB2 are abundantly expressed in the central nervous system (CNS) and peripheral tissues, including the cardiovascular system. CB1 receptors are mainly found in pre-synaptic axon terminals of neurons, while CB2 receptors are prevalent on immune cells and in cardiovascular tissues. Both receptors signal through G-protein coupling, affecting various cellular processes. Additionally, GPR55, another cannabinoid-sensitive receptor, plays a role in cardiovascular homeostasis and disease by influencing intracellular signaling cascades. Endocannabinoids like AEA and 2-AG act as primary ligands for cannabinoid receptors, modulating physiological and pathological conditions through autocrine and paracrine mechanisms [51–55].

Cannabinoids exert their effects on cardiovascular function through modulation of various receptors including cannabinoid receptors (such as CB1), TRP receptors, PPAR receptors, GPR receptors, monoamine receptors, and adenosine receptors, which regulate vascular tone. While endocannabinoids typically induce vasodilation, exogenous cannabinoids can induce unpredictable changes in coronary artery diameter. Additionally, cannabinoids can increase sympathetic tone, leading to elevated myocardial oxygen demand and reduced diastolic coronary perfusion time, predisposing individuals to arrhythmias. These effects are further compounded by increased sinoatrial node activity and alterations in cardiac ion channel function, potentially resulting in ventricular tachyarrhythmias [56].

In contrast to earlier studies examining the effects of long-term cannabis use, research in animals suggests that giving them extremely small amounts of THC might actually protect their hearts. This study showed that when mice were given a tiny dose of THC (0.002 mg/kg) two hours and 48 hours before being induced with a heart attack, it helped partially restore their heart function. Interestingly, this effect wasn't seen in mice treated only with a mixture of ethanol, cremophor, and saline, which was used as a carrier for THC. Moreover, the mice given the ultra-low THC dose had smaller heart attacks, lower levels of troponin T in their blood, less tissue damage, and fewer immune cells infiltrating their heart tissue [57].

In conclusion, cannabis-induced ECG changes are mediated by a complex interplay of sympathetic stimulation, myocardial ischemia, and direct effects on cardiac ion channels. Understanding these mechanisms is crucial for identifying individuals at risk of adverse cardiovascular events related to cannabis use and for implementing targeted interventions to mitigate these risks.

Management Strategies for Cannabis-Induced Cardiac Arrhythmias

Cannabis use has been associated with various cardiac arrhythmias, including atrial fibrillation, atrioventricular block, and ventricular tachycardia [32,33,58]. Managing these cannabis-induced arrhythmias typically involves supportive care alongside targeted pharmacological interventions.

Supportive Care

The initial step in managing cannabis-induced arrhythmias is discontinuing cannabis use. This cessation is critical as ongoing use can perpetuate the arrhythmia and hinder effective treatment

[32][58]. Patients should receive guidance to abstain from cannabis and be educated about the potential cardiovascular risks it poses.

Pharmacological Interventions

Depending on the specific arrhythmia, different pharmacological agents may be employed:

Atrial Fibrillation

For cannabis-induced atrial fibrillation, beta-blockers or calcium channel blockers can be utilized to control the ventricular rate [32]. Antiarrhythmic drugs like amiodarone or flecainide may also be considered to restore normal sinus rhythm.

Atrioventricular Block

In cases of cannabis-induced atrioventricular block, temporary pacing might be necessary to maintain sufficient cardiac output, followed by the use of chronotropic agents such as isoproterenol or epinephrine to increase heart rate [58]. If the block persists, a permanent pacemaker may be required.

Ventricular Tachycardia

For cannabis-induced ventricular tachycardia, antiarrhythmic medications like amiodarone or lidocaine may be administered to terminate the arrhythmia and prevent its recurrence [32]. In severe instances, cardioversion or defibrillation may be warranted.

Discussion:

It's imperative to tailor the management of cannabis-induced arrhythmias to each patient's clinical presentation and underlying medical conditions. Close monitoring and follow-up are crucial to ensure effective management of these potentially life-threatening cardiac complications.

Discussion

Understanding the impact of cannabis on our hearts is like navigating a maze filled with twists and turns, both fascinating and perplexing. In this journey, we delve into the realm of scientific discoveries to unravel the intricate relationship between cannabis use and its effects on our heart rhythms, as depicted in electrocardiogram (ECG) readings.

Think about it as walking a path where each step reveals a new discovery. We're not just exploring theories; we're delving into how these findings affect real people – perhaps your neighbor who enjoys an occasional smoke or your friend managing a heart condition.

As we sift through the research findings, one thing becomes clear: education is key. Imagine the impact of knowing the potential risks of cannabis on your heart health. It's about empowering individuals to make informed choices about their well-being, armed with knowledge that can shape their future.

Yet, amid the uncertainty, there's room for optimism. The review suggests ways to intervene, offering hope for those grappling with cannabis-related heart issues. Picture someone taking the first steps towards quitting, supported by resources and guided by professionals, on a journey towards better heart health.

When it comes to treatment, the review presents a spectrum of options, from medications to devices, each offering a lifeline to those whose heart rhythms have been disrupted by cannabis. It's about restoring a sense of normalcy, envisioning a future where hearts beat steadily once again.

But like any journey, there are challenges along the way. The review points out the need for more research, urging scientists to explore uncharted territories and shed light on the mysteries that still linger. It's a call to action, inviting collaboration and innovation to pave the way for a healthier tomorrow.

Suggestions for Future Studies:

Let's follow the stories of individuals over time to understand how cannabis impacts their hearts, shedding light on both the short-term and long-term effects.

Dive deeper into the science behind cannabis and heart rhythms, unraveling the complex interactions between cannabinoids and our cardiac cells.

Let's ensure our research reflects the diversity of our communities, considering factors like age, gender, and background to capture the full picture of cannabis's effects on our hearts.

Explore new ways to help those struggling with cannabis-related heart issues, from cessation programs to innovative treatments, offering hope and support along the way.

Limitations

Our journey is filled with twists and turns, with studies taking different paths and using different methods. This diversity can make it challenging to draw clear conclusions that apply to everyone.

While we uncover many connections between cannabis and heart health, we can't always say one causes the other. It's like trying to piece together a puzzle without all the pieces – there's still more to learn.

Some studies may have biases that influence their findings, like who they include or how they collect data. These biases can muddy the waters and make it harder to trust the results.

We're still missing pieces of the puzzle, especially when it comes to certain groups of people, like older adults or those with existing heart conditions. As we continue our journey, let's make sure no one gets left behind.

In essence, our exploration of cannabis and heart health is a journey of discovery, filled with twists, turns, and new insights waiting to be uncovered. As we navigate this terrain, let's keep our hearts open to new possibilities and our minds open to the lessons that lie ahead.

Title: Cannabis-Induced Alterations in ECG Parameters: Insights from Clinical Studies.

Study	Authors	Findings
Nayak et al.[7]	Suraj K Nayak	Cannabis consumption linked to lower risk of isometric recurrence and heart rate stability, changes in sympathovagal balance, and parasympathetic activity.
Brown & Patel [8]	Brown C, Patel N	Cannabis associated with cardiovascular complications, decreased HRV, increased sympathetic nervous system activation, leading to bradycardia, and alterations in HRV parameters.
Lee & Kim [13]	Lee S, Kim M	Cannabis use linked to Brugada syndrome, characterized by specific ECG changes, suggesting association between cannabis consumption and cardiac abnormalities.
Jakob et al. [17]	Julian Jakob	Marijuana use associated with higher frequency of aberrant ECG readings, especially major Q wave abnormalities, ST or T abnormalities, left ventricular hypertrophy, bundle branch block, atrial fibrillation, and QT prolongation.
Patel et al. [18]	Nikhil Patel	Cannabis users displayed increased odds of abnormal p-wave axis, marker of atrial myopathy, with pronounced association among individuals with history of cardiovascular disease.
Yeniocak et al. [19]	Selman Yeniocak	Synthetic cannabinoids linked to lower arterial blood pressures, wider P-wave, QRS duration, and lower T-wave amplitude, and ST elevation, suggesting significant cardiovascular impact.
Richards et al. [23]	John R. Richards	Cannabis-induced tachycardia observed in 61% of cases, with dysrhythmias including atrial fibrillation, atrial flutter, ventricular tachycardia, and ST segment elevation, albeit with low overall prevalence.
Andonian et al. [24]	David O. Andonian	Synthetic cannabinoids induce marked bradycardia, hypotension, without significant neurological impairment, suggesting independent cardiovascular toxicity, highlighting potential underreporting in literature.

Study	Authors	Findings
Efe et al. [21]	Tolga Han Efe	Synthetic cannabinoid use potentially linked to atrial fibrillation, suggesting disruption of autonomic nervous system balance and alteration of cardiac electrophysiological properties.
Adegbala et al. [22]	Oluwole Adegbala	Cannabis usage associated with reduced risk of atrial fibrillation in heart failure patients, possibly mediated by protection against left ventricular dysfunction and sympathetic nervous system activation.
Kandah et al. [14]	Kandah et al.	Pseudo-Wellen's syndrome linked to marijuana usage, characterized by angina and EKG changes including biphasic or deeply inverted T waves, suggesting critical blockage of proximal left anterior descending artery.

Title: Cannabis-Induced Arrhythmias: Clinical Insights.

Study	Findings	References
Post-MI Risk Analysis	No significant difference in VT/VF risk between marijuana users and non-users post-MI. Lower in-hospital mortality and trend towards lower AF risk. Increased risk of VF, AF, atrial flutter, pre-excitation syndromes, and long-QT syndrome in younger patients without ACS history.	[32]
Cannabis-Related Arrhythmia	Ambiguous arrhythmia combinations may indicate underlying cannabis abuse. Lower risk of in-hospital mortality and trend towards lower AF risk. No significant difference in ST segment duration. Increased VF, AF, and LVEF risk in young patients without ACS history.	[33]
THC Vaping-Related Tdp Management	Unclear management; Isoproterenol may provide temporary relief by increasing heart rate and shortening QTc interval. Long-term management with ICD and nadolol may reduce ventricular arrhythmia recurrence.	[34,35]
Synthetic Cannabinoid-Induced Arrhythmia	SC-induced SCA, VF, and TdP in a 52-year-old woman. Prolonged QTc intervals observed post-SCA events. Mechanism involves SC stimulating CB1 receptors, altering cardiac ion channels, and prolonging QTc.	[31,36,37]
CUD and Arrhythmia Hospitalization	CUD linked to 47%-52% higher chance of arrhythmia hospitalization in young population (15-34 years), primarily with atrial fibrillation. Younger age, male gender, and African American ethnicity associated with CUD-related arrhythmia.	[38]
Teenage CUD-Related Arrhythmia Hospitalizations	Prevalence of arrhythmias higher among older teenagers (17–20 years) with CUD. Males and Caucasians more affected. Prevalence of arrhythmias increasing over time, with atrial fibrillation most frequent.	[39]

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