TOPICS IN REVIEW

Arrhythmias and cannabis use: A comprehensive overview

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The increasing prevalence of cannabis use, with an estimated 219 million users globally, underscores the need to examine its potential health impacts. This review focuses on the arrhythmogenic properties of cannabis, particularly considering its active component, tetrahydrocannabinol, and its interactions with the endocannabinoid system. Epidemiological data and multiple studies indicate a significant association between cannabis use and various arrhythmias, particularly atrial fibrillation. The risk is notably higher among younger users and males. Additionally, case reports have linked cannabis use to other arrhythmias such as ventricular tachycardia and ventricular fibrillation, especially in individuals with underlying cardiac abnormalities. This review also discusses the arrhythmogenic potential of synthetic cannabinoids, which are more potent than natural tetrahydrocannabinol. Despite some studies suggesting no significant difference in arrhythmia burden between cannabis users and nonusers, the preponderance of evidence supports a correlation between cannabis use and increased arrhythmia risk. Given the rising tetrahydrocannabinol content in cannabis products and the limited data on the long-term cardiovascular effects, this review underscores the need for large-scale prospective studies. Until more comprehensive data are available, it is advisable for patients with channelopathies, structural heart disease, or prior myocardial infarction to avoid cannabis use.

KEYWORDS Cannabis; Tetrahydrocannabinol; Marijuana; Arrhythmia; Atrial fibrillation; Ventricular fibrillation/tachycardia

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Introduction

The global trend in drug use is on the rise, with an estimated 296 million users worldwide. Cannabis remains the most widely used drug, with approximately 219 million users in 2021, representing 4.3% of the global population 15 to 64 years of age. North America has the highest prevalence of cannabis use. Globally, about 70% of cannabis users are men, whereas in North America, 42% of users are women. According to the 2023 World Drug Report, the annual prevalence of cannabis use is disproportionately higher among adolescents (5.34% in 15–16 years of age) compared with adults (4.3%).[1]

According to the data from the United Nations Office on Drugs and Crime, the top 5 commonly used drugs in the United States are cannabis (21.9% of the population), amphetamines (5.65%), nonmedical use of prescription opioids (3.9%), cocaine (2.4%), and nonmedical use of prescription stimulants (2.1%).[2] Several of these drugs exhibit arrhythmogenic potential. Additionally, co-use of marijuana and alcohol is highly prevalent in the United States, with about 58% of adolescent drinkers reporting marijuana use and over 75% of marijuana users reporting consuming alcohol.[3] Alcohol and marijuana together can intensify side effects of both, leading to increased cardiovascular risks due to synergistic effects. Sudden cardiac death from ingesting cannabis, along with alcohol has been reported.[4] However, the data on the arrhythmogenic potential of cannabis remain conflicting. This review aims to provide a comprehensive analysis of the potential arrhythmogenic properties of cannabis based on available literature.

"Cannabis" refers to all products (about 540 chemical substances) derived from the plant *Cannabis sativa*. "Marijuana" refers to parts or products containing significant amounts of tetrahydrocannabinol (THC) from the plants *Cannabis sativa* or *Cannabis indica*. Cannabinoids are a group of substances found in the cannabis plant, mainly THC and cannabidiol (CBD).[5]

As of August 8, 2024, cannabis has been legalized in 24 states for recreational use, along with Guam, the U.S. Virgin Islands, Northern Mariana Islands, and Washington, DC.[6] Despite its legalization in these regions, it remains classified

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KEY FINDINGS

- Cannabis use has been noted to have a temporal correlation with arrhythmias.
- Atrial fibrillation is the most common arrhythmia associated with cannabis use.
- Patients with channelopathies, structural heart disease, and prior myocardial infarction appear to have a significant association with cannabis induced arrhythmias.
- There is a paucity of large-scale prospective studies analyzing the correlation between cannabis use and arrhythmias. Until more data are available, it would be prudent to advise patients with the previously mentioned comorbidities to avoid marijuana use.

as a Schedule I drug by the Drug Enforcement Administration, indicating drugs with no currently accepted medical use and a high potential for dependence and abuse.[7,8]

The research reported in this paper adhered to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines.

Pathophysiological effects of cannabis with an emphasis on arrhythmogenesis

The electrophysiological effect of cannabis is multifaceted (Figure 1). Cannabis primarily exerts its effects via the endocannabinoid system, involving G protein–coupled cannabinoid receptors, CB1 and CB2. THC is a partial agonist of CB1 and CB2 receptors. Activation of CB1 and CB2 receptors inhibit the enzyme adenylyl cyclase, leading to a reduction in cyclic adenosine monophosphate levels, resulting in inhibition of voltage-sensitive calcium channels (N-type, P/Q-type, and L-type) and D-type outward potassium channels, while G protein–activated inwardly rectifying potassium channels are activated.[9] These alterations can potentially disturb the electrical conduction system of the heart, increasing the risk of arrhythmias. CB1 receptor mediates an increase in heart rate and favors a shift toward increased sympathetic activity, resulting in enhanced sinus node automaticity.[10]

Tachycardia may also result from THC induced parasympathetic vasodilatation.[11] Studies have shown increased plasma norepinephrine levels and increased urinary excretion of epinephrine following THC use.[12] Cannabis-induced adrenergic excitation has the potential to provoke tachyarrhythmia in predisposed individuals due to shortening of the action potential duration coupled with disturbances in the myocardial electrophysiology, which leads to automaticity and micro–reentrant circuits.[13] In fact, THC induces a biphasic response with increased sympathetic activation and tachycardia at lower doses and bradycardia at higher doses.[14] The possible explanation of bradycardia/bradyarrhythmia is alteration of autonomic nervous system with reduction in sympathetic stimulation while there is enhanced parasympathetic reaction.[13]

Medical cannabis products include dronabinol and CBD. Dronabinol is biochemically identical to THC and the arrhythmogenic pathophysiology is similar as described previously. CBD has a weak affinity for CB1 and CB2 receptors and does not possess the typical intoxicating/psychoactive properties associated with marijuana use.[15,16] CBD has purported anti-inflammatory and analgesic activities by cyclooxygenase and lipoxygenase inhibition.[17] In an in vitro study on rabbit hearts, CBD was found to inhibit the $Na_v1.5$ sodium current, $Ca_v1.2$-mediated L-type calcium current, and all the repolarizing potassium currents except the delayed rectified current ($K_{ir}2.1$). No effects on resting membrane potential were observed due to the absence of effects on the $K_{ir}2.1$ current, except at rapid pacing rates.[18] CBD also leads to the inhibition of hERG/Ikr channel and QT prolongation.[19] Thus, CBD impacts cardiac ion channels, potentially leading to proarrhythmogenic effects, particularly in patients with cardiac channelopathies or in combination with other drugs affecting cardiac electrophysiological substrate.

Patients with channelopathies, structural heart disease, and prior myocardial infarction appear to have a significant association with marijuana-induced arrhythmias. Cannabinoids also affect key classes of antiarrhythmic medications through its inhibition of CYP3A4 and CYP2D6. This can cause elevated levels of antiarrhythmic drugs such as amiodarone, quinidine, lidocaine, mexiletine, flecainide, propafenone, and beta-blockers. However, data regarding dose adjustment are currently limited.[20]

Synthetic cannabinoids are widely available and marketed under various names such as Spice, K2, Cloud 9, Black Mamba, etc. They are structurally classified into 7 groups (naphthoylindoles, naphthylmethylindoles, naphthoylpyrroles, naphthylmethylindenes, phenylacetylindoles, cyclohexylphenois, and classical cannabinoids).[21] Synthetic cannabinoids mimic the effects of THC on the cannabinoid receptor with greater binding affinity to the CB1 receptors and are multiple times more potent. Synthetic cannabinoids are full agonists of the CB1 receptor in contrast to natural THC, which is a partial agonist.[22] They mediate arrhythmias via receptor activation as well as interaction with myocardial ion channels. They also increase oxidative stress and cell death by interfering with the mitochondrial transport.[23]

Arrhythmia burden with cannabis use

The route of administration may influence the cardiovascular effects of cannabinoids. Oral administration results in slower absorption and more prolonged effects, whereas inhalation leads to rapid onset but shorter duration of action. Peak plasma concentration and the resultant cardiovascular effects, including arrhythmias, might be higher with inhaled forms compared with oral administration.[24,25] No study comparing the different forms of administration of cannabis was found on detailed literature search, and further study is needed.

There are multiple studies utilizing the National Inpatient Sample Database between 2010 and 2018 have investigated the association between marijuana use and arrhythmias.

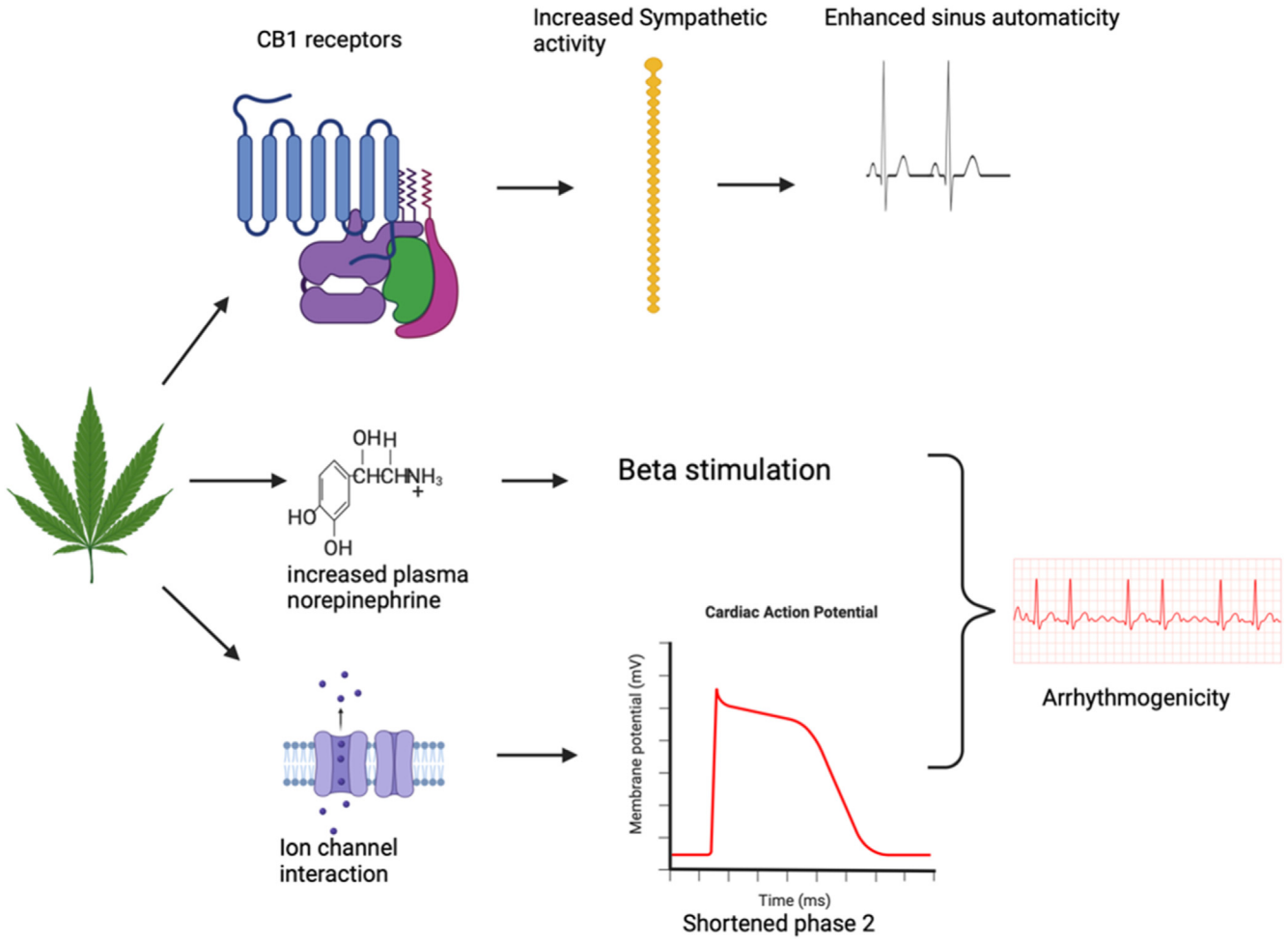

Figure 1 Pathophysiology of arrhythmia with cannabis use.

The reported incidence of arrhythmias in these studies has been up to 3% with the predominant arrhythmia being atrial fibrillation (AF). There appears to be a 47% to 52% increased likelihood of arrhythmia-related hospitalization in younger populations (15–34 years of age) with cannabis use.[26,27] Notably, there has been a male preponderant increase in arrhythmia-related hospitalizations among individuals with cannabis use, and is associated with worse in-hospital mortality outcomes and longer hospital stays.[28,29] In a Danish nationwide registry-based study, new-onset arrhythmias were observed more frequently among patients newly initiated on medical cannabis, with a relative risk increase of 64% compared with matched control subjects without prior arrhythmias.[30]

Sinus tachycardia

Studies examining drivers under the influence of cannabis have demonstrated higher mean pulse rates and increased incidence of sinus tachycardia (19.4% vs 1.6%). This was confirmed by delta-9-THC testing and positive status. There was no correlation between blood delta-9-THC concentration and pulse rate.[31]

Several randomized controlled trials and experimental studies have consistently shown a dose-dependent increase in heart rate associated with marijuana use. Oral THC, smoked marijuana, and synthetic nabilone have all been implicated in elevating heart rate, suggesting a uniform physiological response across different modes of cannabis consumption.[32]

Atrial fibrillation

Database analyses, retrospective analyses, review articles, case series, and case reports have all suggested an association between marijuana use and AF. In 2 National Inpatient Sample Database studies, the most common arrhythmia associated with marijuana use was AF, with an estimated 42% of marijuana-related arrhythmias being attributed to AF.[26,27]

Multiple case reports and case series suggest marijuana as a potential trigger for paroxysmal AF.[33–35] A review between the association of marijuana and AF by Korantzopoulos and colleagues[36] showed 6 patients with AF within minutes to 3 hours of use of marijuana. These patients had no structural heart disease, and were relatively young, with a mean age of 24.5 ± 7.8 years. The hypothesis was based on altered

electrophysiologic properties of the myocardium favoring automaticity and micro–re-entry from adrenergic stimulation. In addition, coronary microcirculation–mediated atrial ischemia was also suggested as a potential etiopathogenic mechanism.[36] A retrospective study showed that 3.4% of lone AF in patients <45 years of age was precipitated by marijuana.[37]

Supraventricular tachycardia

The data on the association of marijuana with supraventricular tachycardia (SVT) is limited. A cross-sectional analysis among 1485 participants from the MESA (Multi-Ethnic Study of Atherosclerosis) study with extended ambulatory electrocardiographic monitoring with the Zio Patch XT (iRhythm) showed trends to more SVT per day (although not statistically significant). Additionally, there was an increase in premature atrial contractions and nonsustained ventricular tachycardia (VT), although it did not meet statistical significance. This was more prominent among current marijuana smokers in comparison with past users.[38]

There have also been multiple case reports of SVT with marijuana use in patients with no prior cardiac history, and there are reports of successful SVT treatment with either cessation of cannabis use and beta-blockers alone.[11,38,39]

Another interesting phenomenon is the possible interaction between marijuana and amitriptyline at the cytochrome level. A 17-year-old male on amitriptyline presented with a "racing heart" and was found to have SVT at a rate of 300 beats/min after consuming marijuana 12 hours prior. Screening was negative for other drugs.[40]

Cannabis may also trigger atrioventricular tachycardia by enhancing accessory pathway conduction potentially via modulation of the autonomic nervous system.[41]

Sudden cardiac arrest/VT/ventricular fibrillation

The data on the correlation between malignant ventricular arrhythmias and marijuana use is largely limited to case reports and series (Table 1).

A 36-year-old male developed VT during use of marijuana. Workup for other potential etiology including ischemic evaluation and electrophysiologic studies were negative.[42] Polymorphic VT during an exercise treadmill stress test performed for palpitations has also been reported in a 30-year-old male with a history of cannabis use, no other risk factors, and negative workup including cardiac catheterization, cardiac magnetic resonance imaging, and electrophysiologic studies. A repeat exercise stress test following initiation of beta-blocker and withholding of marijuana use showed no recurrences.[11]

In patients with underlying structural heart disease and channelopathies, marijuana may trigger life-threatening arrhythmias. A 22-year-old African American male developed VT and torsades de pointes in the setting of marijuana use and subsequently found to have Brugada syndrome on his electrocardiogram.[43] The marijuana was believed to have triggered the arrhythmia in this case. A 60-year-old male with prior severe 3-vessel coronary artery disease and ischemic cardiomyopathy with an implantable cardioverter-defibrillator in situ had episodes of ventricular fibrillation (VF) that appeared to be triggered with high doses of marijuana use.[44]

Case series have also shown an association between acute coronary syndromes with sudden cardiac arrest (including asystole, VT, and VF) in the absence of other risk factors besides recent marijuana use.[45] Several reported case fatalities related to use of marijuana appear to stem from its propensity to trigger myocardial infarction. Although there are many case reports of the same, there have been no epidemiological studies proving the association. Based on prior data, the risk of myocardial infarction is elevated 4.8 times over baseline in the hour after smoking cannabis and subsequently declines rapidly.[49] Marijuana use may trigger coronary slow flow with associated VT, which was reproducible on electrophysiologic testing. This resolved with verapamil therapy and cessation of marijuana use. A proposed theory was abnormalities of coronary microcirculation.[50]

A case series by Hartung and colleagues[46] reported 2 cases of young males (23 and 28 years old, respectively) with death secondary to VF-mediated sudden cardiac arrest. Autopsy and toxicologic evaluations showed elevated cannabis levels in blood (THC: 1.9–5.2 ng/mL) with negative screening for other drugs. Cardiac arrest and death were suspected to be secondary to acute global cardiac failure under the influence of cannabis.[46] There have also been multiple cases suggestive of sudden cardiac arrest triggered by polysubstance use (cannabis in addition to cocaine/alcohol).[47]

A review by Drummer and Gerostamoulos[48] provides a summary of 13 case fatalities related to marijuana use. All patients were male and 17 to 52 (median 37) years of age. Autopsy studies showed a spectrum from left ventricular mural thrombus with myocardial infarction, thrombi in the coronaries, dilated cardiomyopathy, vasospasm, and overt coronary artery disease. Arrhythmias ranged from asystole to VF. Twelve of these patients had a positive screening test for cannabis (Table 1).[48]

Bradyarrhythmias

There have been numerous case reports on the association between marijuana use and bradyarrhythmia. The effects of marijuana in precipitating bradyarrhythmia vary from sinus node dysfunction to high-grade atrioventricular block. These effects may be mediated by high vagal tone and parasympathetic dominance, by tropism for adenosine receptors, or via direct effects on the myocardium by unidentified endothelial and cardiac receptors.[51]

Acute cannabis smoking of low to moderate doses causes tachycardia in the first 60 minutes from sympathetic stimulation and parasympathetic inhibition, while large and/or chronic doses cause parasympathetic dominance that predisposes to bradycardias.[9] A study on prolonged THC ingestions in human volunteers and animals showed parasympathetic dominance featuring sinus bradycardia.

Table 1 Summary of case reports/series of VT and VF in cannabis use

No.	Study	Presentation	Workup/management
1	Sampat et al[42]	VT in a 36-y-old male	Negative ischemic evaluation and EP studies
2	Yahud et al[11]	Polymorphic VT during treadmill stress test done for palpitation in 36-y-old male	Negative cardiac catheterization, cardiac MRI, and EP studies. Repeat stress test after starting beta-blocker and marijuana cessation showed no recurrence.
3	Stockholm et al[43]	VT and torsades de pointes in a 22-y-old AA male	Subsequently found to have Brugada syndrome on ECG and underwent ICD implantation
4	Baranchuket al[44]	VF in a 60-y-old male with history of 3 vessel CAD/ischemic cardiomyopathy and ICD in place after marijuana use	No evidence of ischemia based on biochemical markers, EKG, or clinical assessment. ICD interrogation identified a single episode of VF, which was detected and treated with a 35-joule shock.
5	Casier et al[45]	Case series reporting link between ACS and SCA with recent marijuana use	
6	Hartung et al[46]	Fatality due to VF mediated SCA in 2 males 23 and 28 y of age	
7	Montisci et al[47]	Multiple reported cases of SCA triggered by polysubstance use	
8	Drummer and Gerostamoulos[48]	Case series reporting 13 fatalities from SCA (asystole to VF) related to marijuana use in males with median age of 37 y	

AA = African American; ACS = acute coronary syndrome; CAD = coronary artery disease; ECG = electrocardiography; EP = electrophysiology; ICD = implantable cardioverter-defibrillator; MRI = magnetic resonance imaging; SCA = sudden cardiac arrest; VF = ventricular fibrillation; VT = ventricular tachycardia.

There was also dose-dependent incidence of sinus arrest/asystole with long-term inhalation.[52] Studies have also shown that the bradycardic effects of THC were completely abolished in animals in which the autonomic pathways to the heart were pharmacologically or surgically inactivated suggesting an autonomic etiology for bradycardia.[53]

An electrophysiology study performed in a 26-year-old male with chronic cannabis use who presented with atrioventricular block showed high-grade supra-Hisian (nodal) atrioventricular block with an AH interval of 180 ms and a prolonged HV interval of 85 ms. The electrophysiologic effects of THC on cardiac conduction include a change in P-wave morphology, decrease in SA conduction, delay in AH interval, and decrease in atrioventricular node refractory period.[54]

Exercise-related asystole with syncope is rare in patients with structural heart disease. Presence of the same in the absence of structural heart disease portends an ominous sign. A 40-year-old healthy man was hospitalized with recurrent near syncope and an isolated syncopal episode after strenuous exercise while using marijuana. A monitored exercise test showed a loss of sinoatrial activity and ventricular asystole suggestive of parasympathetic dominance, potentially related to long-term cannabinoid use.[55]

Synthetic cannabinoids

The most common presentation with synthetic cannabinoid use is sinus tachycardia in young males (37%–77%). This has been demonstrated in multiple case reports and case series with sinus rates up to 220 beats/min.[22,56–58] SVT has been noted in case reports with synthetic cannabinoid ingestion. This is postulated to be secondary to increased circulating catecholamines or oxidative demands on the myocardium with CB1 receptor agonism.[59,60] In patients with supraventricular arrhythmias, chronic synthetic cannabinoid users had an increase in the dispersion values of P waves, which could indicate an early step in the genesis of AF.[23] There have also been reported cases of resistant VF and cardiogenic shock after synthetic cannabinoid use in young males.[61] They also pose the problem of often being undetectable on routine drug screens and are relatively low priced in comparison with traditional marijuana.[21]

Medical cannabis

Medical cannabis has been increasingly utilized for its analgesic, antiemetic, and anti-inflammatory properties. Compounds such as dronabinol (a synthetic form of THC), CBD, and nabilone (a synthetic cannabinoid) are commonly prescribed. A comprehensive literature search on PubMed using keywords "marijuana," "cannabis," "dronabinol," "cannabidiol," "CBD," "nabilone," "arrhythmia," and "cardiac arrhythmia" reported few studies linking medical cannabis use and arrhythmia.[62]

CBD is approved by the Food and Drug Administration as an oral solution for treatment of intractable seizures (in >1 year of age), especially in Lennox-Gastaut syndrome,

tuberous sclerosis, and Dravet syndrome, with improved quality of life due to reduced frequency and intensity of seizures.[63] Dronabinol is approved by the Food and Drug Administration for treatment of anorexia in patients with acquired immunodeficiency syndrome.[64] Dronabinol and nabilone are suggested by the American Society of Clinical Oncology Focused Guideline for intractable chemotherapy-induced nausea and vomiting in cancer patients.[65–67] Off-label, THC has been demonstrated to reduce intraocular pressure in patients with glaucoma, improvement in chronic pain and muscle spasms in patients with multiple sclerosis and spinal cord injuries, and improved neuropathic pain in patients with neurological disorders, such as Parkinson's disease and Tourette syndrome.[68,69] However, there still exists controversy and lack of enough randomized control trials to support these off-label uses.

Medical cannabis usually contains CBD unlike the recreational marijuana that has higher levels of THC. Additionally, medical use of cannabis is regulated with timely dosing and monitoring by the prescribing physician with the intention to reduce side effects.[70] Despite this, a recent nationwide Danish study showed that there was an elevated risk of new-onset arrhythmia associated with medical cannabis products containing THC or CBD for chronic pain. The largest risk difference was in patients with cancer or cardiometabolic disease with >75% of arrhythmia burden due to AF. The standardized 180-day risk ratio was 2.07.[71] There have also been reports of dose-dependent heart rate increase with oral dronabinol and nabilone.[62]

In summary, patients who use recreational marijuana are at higher risk for overdose and arrhythmias because of lack of medical supervision and monitoring. Recreational use is episodic and could involve larger doses with associated greater propensity for arrhythmias.

Studies showing no correlation between cannabis and arrhythmias

There appear to be fewer studies noting lack of correlation between cannabis and arrhythmias, and these are worth looking into. A prospective study comparing cannabis user healthy control subjects, cannabis user ischemic heart disease patients, and non–cannabis user ischemic heart disease patients showed no difference in overall arrhythmia burden.[72] However, this study did not compare healthy control subjects with and without cannabis use.

An outcome analysis on patients hospitalized with acute myocardial infarction and marijuana use showed that the marijuana cohort had less coronary artery disease and AF than nonusers. However, the marijuana cohort was younger by 10 years, raising the possibility of age being a confounder. Additionally, marijuana users tended to need hospitalization for acute myocardial infarction at younger ages.[73]

A retrospective study showed less odds of AF in patients with admitted with heart failure among cannabis users. No measure of temporal correlation between cannabis use and AF was provided.[74]

CBD has been suggested to have some protective effects against high glucose-induced oxidative stress and cytotoxicity in cardiac voltage-gated sodium channels. This has been postulated to provide an antiarrhythmic effect.[75] However, there has been a decline in CBD content with rising trend in THC content in most seized marijuana in the United States, with a current THC content of about 50%. This increasing THC content can be inherently more harmful, as we have discussed previously.[76]

Screening for cannabis use

We advocate for routine screening for cannabis use including frequency, quantity, and methods of administration in the social history. Marijuana testing is currently required prior to an evaluation for heart transplantation. It may be reasonable to perform urine toxicology in the setting of myocardial infarction and new-onset heart failure.[20] In a clinical framework, the detection of cannabis is predominantly performed via urine analysis, in which its principal component, delta-9-THC, or its metabolite 11-hydroxy-delta-9-THC is identified.[77,78] Although it is possible to detect cannabis through blood, hair, or saliva, these methods are not typically implemented in routine clinical practice.[79–81] Postingestion, cannabis can be traced in plasma almost immediately, with blood sample measurements offering a more accurate reflection of the individual's level of exposure, as the concentrations of the drug in urine can fluctuate due to metabolic variations and urine production.[78] Due to their lipophilic nature, most cannabinoids have long elimination half-life.[82] Approximately 80%–90% of delta-9-THC is expelled from the body within 5 days but may be detectable for up to 2 weeks in occasional users, and even longer in chronic users.[78] Immunoassay is the routine testing method but analytical methods like gas chromatography mass spectrometry or liquid chromatography–tandem mass spectrometry can be used for accurate quantification.[83–85]

Nonprescription synthetic cannabinoids have a high potential for pharmacologic manipulation and increased potency. Additionally, synthetic cannabinoids do not show up in a routine urine drug screen, as they are not structurally similar to THC. In terms of synthetic cannabinoids, their metabolites are the target of detection. The Synthetic Drug Abuse Prevention Act of 2021 listed the synthetic metabolites of interest, which may be detected more in blood, hair, or saliva.[86,87] The biggest challenge is the emergence of newer compounds with unknown metabolites making detection harder. There are no available data on lab testing of medical marijuana, and this is an area that needs further research.

Conclusion

Cannabinoids appear to have a temporal correlation with arrhythmias which subside with prolonged periods of abstinence. While studies have suggested that frequency of marijuana use may correlate with more ectopy, data on a possible safe dose of marijuana are lacking.[20] In addition, there is a significantly higher preponderance in younger

males to adverse effects of marijuana, which is likely secondary to the highest use, and possibly in higher doses in the corresponding demographic. Patients should be counseled regarding concurrent use of other illicit drugs. It is important to discuss with the patients not only the limited scientific data at hand, but also the possible association of marijuana and arrhythmias, especially in higher-risk groups.[20]

While medical marijuana may offer significant therapeutic benefits, the potential for arrhythmias should always be considered. Therefore, healthcare providers must weigh the benefits against the risks, especially in patients with preexisting heart conditions. Close monitoring and individualized treatment plans are essential to minimize the risk of adverse cardiac events. Further research is needed to establish clear guidelines and ensure the safe use of medical marijuana. The method of administration is particularly important, with inhaled forms likely to pose a higher risk for arrhythmias compared with oral forms, though no formal study was found in this area.

We highlight the paucity of large-scale prospective studies analyzing the correlation between marijuana and arrhythmias. With the advent of consumer wearable health technology and long-term electrocardiographic monitoring, this would likely be a more feasible option in the future. Until we have large-scale data, it would be prudent to advise patients with the previously mentioned comorbidities to avoid marijuana use.

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