Case Report

Suspected Cannabis Vaping–Induced Pericardial Effusion

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Abstract
Pericardial effusions refer to an excess accumulation of fluid within the pericardial cavity. The etiology is diverse, with most cases being idiopathic in nature. We report a case of suspected cannabis vaping–induced pericardial effusion in a 31-year-old South Asian patient, which was successfully managed with high-dose aspirin, colchicine therapy, and cannabis vaping cessation.

Key Clinical Message: Clinicians should be aware of the possible cardiovascular complications of cannabis vaping.

Keywords
pericardial effusion, cannabis, vaping

Introduction
Pericardial effusions refer to an excess accumulation of fluid within the pericardial cavity. The etiology is diverse, with most cases being idiopathic in nature.1-3 Cannabis is a widely used recreational and therapeutic drug with potentially adverse cardiovascular complications, including myopericarditis and myocarditis.4,5

Currently, to the authors’ knowledge, there are no case reports describing cannabis vaping and pericardial effusions. We report a case of suspected cannabis vaping–induced pericardial effusion in a 31-year-old South Asian patient, which was successfully managed with high-dose aspirin, colchicine therapy, and cannabis vaping cessation.

Case Report
A 31-year-old South Asian man with no significant medical history presented to the emergency department with a 1-week duration of worsening dyspnea. He did not report any angina, orthopnea, paroxysmal nocturnal dyspnea, or syncope. His social history revealed that he vaped large quantities of cannabis daily (half of a cartridge—1.5 mg, 95% tetrahydrocannabinol) or the prior 2 months without tobacco, alcohol, or illicit drug use. He did not have any recent travel history, nor did he have any pets. He did not display any antecedent viral symptoms and denied any sick contacts.

On admission to the emergency department, his vital signs included a blood pressure of 115/82 mmHg, a pulse of 88 beats per minute, and regular pulse oximetry of 98% on ambient air. On physical examination, he appeared comfortable without cardiopulmonary distress, with a mildly distended jugular venous pressure of 9 cm of water, and normal heart sounds without a pericardial friction rub on auscultation. He had no crackles upon auscultation of the lung fields, and no peripheral edema was noted.

His electrocardiogram revealed sinus rhythm, a rate of 83 beats per minute with small voltage complexes, and nonspecific ST-T changes such as T-wave inversions in leads V2, V6, II, III, and (aVF) (Figure 1). The chest radiograph did not indicate any acute cardiopulmonary disease. Pertinent diagnostic laboratory investigations revealed a normal troponin I 0.05 ng/mL (normal range: 0.0-0.08 ng/mL) and NT-pro-brain natriuretic peptide 105 pg/mL (normal range < 125 pg/mL). His complete blood count, renal, hepatic, and thyroid function tests were normal. A glycosylated hemoglobin and lipid panel were

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Received August 17, 2022. Revised October 17, 2022. Accepted November 2, 2022.

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also normal. A 2-dimensional transthoracic echocardiogram (2D-TTE) revealed a moderate circumferential pericardial effusion (“anterior” dimension of 13 mm, “posterior” dimension of 8 mm) with no impending tamponade physiology and preserved left ventricular function (Figure 2).

He was admitted to the cardiology ward and was administered high-dose aspirin (325 mg every 8 hours), colchicine (0.5 mg every 12 hours), and pantoprazole (40 mg every 12 hours). No oral or intravenous glucocorticoids were administered. Diagnostic pericardiocentesis was not performed as there was no evident tamponade physiology, and it was not deemed in the patient’s risk-benefit favor. During his ensuing 1-week hospitalization, he remained hemodynamically stable, and a repeat 2D-TTE indicated a significant interval improvement in the pericardial effusion (Figure 2). No pericardiocentesis or advanced thoracic imaging was performed.

Further detailed work-up included a negative interferon-γ release assay (QuantiFERON-TB Gold Plus), hepatitis panel, HIV, enzyme-linked immunosorbent assay (ELISA), RNA, polymerase chain reaction (PCR), and respiratory pathogen panel (BioFire) tests. Inflammatory markers (erythrocyte sedimentation rate and high-sensitivity C-reactive protein) were normal. A comprehensive rheumatologic and immunologic panel was unremarkable. Coronavirus 2019 PCR and rapid antigen tests were negative, in addition to blood cultures and urinalysis. He was extensively counseled with respect to the possibility of his cannabis vaping being implicated in the etiology of the pericardial effusion and subsequently discharged for routine outpatient follow-up with a surveillance 2D-TTE.

Discussion

The pericardium is a fibroelastic sac that surrounds the heart and normally contains a thin layer of fluid. A pericardial effusion is considered present when the accumulated fluid within the sac exceeds the small amount that is normally present. There is a diverse spectrum of etiologies for pericardial effusions, including neoplasia, infections, inflammatory conditions, systemic diseases, drugs, and toxins, and in many cases, no specific cause is ascertained; it is either deemed idiopathic or presumed viral.\(^1\)\(^2\)\(^3\)

Cannabis vaping is increasing in popularity, and its use has nearly doubled in the last decade.\(^6\) Vaping devices can be divided into dab pens or vaporizers. In both devices, the desired substance, contained in a reservoir or cartridge, is heated in a vaporization chamber to produce an aerosol and then inhaled via a mouthpiece. Dab pens use cannabis concentrates referred to as butane hash oil or butane honey oil. Vaporizers can utilize dried or liquid forms of cannabis as well as cannabis concentrates.\(^7\) There is still much to be discovered about the adverse effects of vaping cannabis, but thus far, there

Figure 1. The patient’s electrocardiogram indicating sinus rhythm, a rate of 83 beats per minute with small voltage complexes, and nonspecific ST-T changes such as T-wave inversions in leads V₅-V₆, II, III, and aVF.
have been reports of E-cigarette or vaping-associated lung injury (EVALI) occurring in users. The known cardiovascular effects of cannabis use include tachycardia, hypertension, and orthostatic hypotension, and in rare cases, it can precipitate acute coronary syndromes. There have been case reports of myopericarditis and myocarditis in cannabis users, although no causal link has been established, and it remains ambiguous whether cannabis or contaminants of the substance may have played a role in the pathophysiology.

Our patient reported daily vaping of dried cannabis herb for the prior 2 months (half of a cartridge—1.5 mg, 95% tetrahydrocannabinol). Our tentative differential diagnosis suspected that the patient developed pericarditis with an associated effusion, despite the normal inflammatory markers. The patient’s symptomatology also coincided with the onset of his new habit, and the pericardial effusion resolved soon after cannabis cessation, alluding to a temporal link that may have been confounded by his 4-week therapeutic course. It is entirely plausible that routine vaping of cannabis was implicated in the development of the pericardial effusion; however, it remains unknown whether other constituents or contaminants could have contributed. The sale of cannabis is not regulated in Trinidad and Tobago; thus, the drug’s purity is not assured.

Our patient also underwent comprehensive diagnostic testing, including for rheumatologic disease, which ultimately proved unrevealing. As aforementioned, diagnostic pericardiocentesis was deferred as the patient did not display overt tamponade physiology, and the anterior effusion was 13 mm and was considered in the risk-benefit analysis. Most idiopathic pericardial effusions are presumed to be viral in origin, and an in-depth work-up of viral causes is usually not performed. However, an extensive assay for common respiratory pathogens (coronavirus, Enterovirus, rhinovirus, influenza, parainfluenza, respiratory syncytial viruses, Bordetella, Mycoplasma, and chlamydia) was negative in this case, although it does not entirely exclude an atypical pathogen. Ideally, further advanced cardiac imaging with cardiac computed tomography (CCT) or magnetic resonance imaging (cMRI) could have been performed; however, the patient swiftly responded to the conventional therapies of high-dose aspirin and colchicine evidenced by significant improvement.
interval echocardiographic resolution between 1 week and 1 month, respectively.

**Conclusion**

We report a case of suspected cannabis vaping-induced pericardial effusion in a 31-year-old South Asian patient, which was successfully managed with high-dose aspirin, colchicine therapy, and cannabis vaping cessation. Clinicians should be aware of the possible cardiovascular complications of cannabis vaping.

**Author Contributions**

All authors contributed equally to writing the manuscript, and all authors read and approved the final manuscript.

**Declaration of Conflicting Interests**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

**Funding**

The author(s) received no financial support for the research, authorship, and/or publication of this article.

**Ethics Approval**

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Informed Consent**

The patient has provided both verbal and written informed consent to have the details and images of his case published, and institutional approval was not required for publication.

**Data Sharing**

All available data can be obtained by contacting the corresponding author.

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