

## Case report

## Marijuana-induced myocarditis in a 24-year-old man

Toktam Alirezaei<sup>1</sup>, Mohammad Kalateh Agha Mohammadi<sup>2</sup>, Rana Irilouzadian<sup>1,\*</sup>, Hamidreza Zarinparsa<sup>1</sup>

<sup>1</sup>Department of Cardiology, Shohadaye Tajrish Hospital, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran; <sup>2</sup>Department of Internal Medicine, Shohadaye Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

### Abstract

As marijuana, the most widely-used illicit drug in adolescents and adults, has some unknown side effects, marijuana abuse has become a public health concern. Also, marijuana affects different organs such as heart in its rate, rhythm and coronary flow; it eventually leads to events such as myocardial infarction and rarely myocarditis. A 24-year-old man without any medical history or cardiovascular risk factors presented with chest pain after marijuana consumption. Based on electrocardiogram, myocardial cytolysis and transthoracic echocardiography acute myocarditis diagnosis was established. A few days later, transthoracic echocardiography showed a small clot in apex with reduced left ventricle ejection fraction, in the absence of local akinesia. The patient was discharged with oral anticoagulant stable and without any symptoms. The myocarditis after marijuana abuse is rare. The physicians should include acute myocarditis in differential diagnosis of a patient with chest pain after using marijuana.

**Keywords:** *marijuana abuse; myocarditis; cannabis*

### Introduction

Marijuana is a plant-based drug which is consumed by approximately 4% of the world and 5.6% of the 15-year-old population globally. In the United States, these numbers are 14.5% of all ages and 20% of population aged 15 years old [1]. Approximately 30% of students are using marijuana at college entry which 8.5% of them had started the marijuana abuse at first year [2]. In 11 states of the United States, Canada and many European countries, weed consumption is legal or has been decriminalized. The most common way to use marijuana is by inhalation of the gray to green flowers and leaves of *Cannabis sativa* plant [3].

Marijuana contains more than 460 chemical materials such as  $\delta$ -9-tetrahydrocannabinol (THC) that all of its effects and side effects are still unknown [4]. Since marijuana has different effects on body, its abuse is a public health concern [5]. Marijuana affects cardiovascular system and causes irregularities in heart rhythm and rate (tachycardia and arrhythmias), changes in blood pressure (hypotension or hypertension), vasospasm, vasodilation, altered coronary flow to the heart and as a result, increased risk of myocardial infarction [6, 7]. Myocarditis, the inflammation of heart muscle, has various signs and symptoms, from nonspecific systemic symptoms (chest pain, fever, myalgia, palpitation and exertional dyspnea) to hemodynamic instability and sudden cardiac death [8, 9].

To the best of our knowledge, a few known cases of myocarditis after marijuana abuse have been reported to date [10-14]. Here, we

Received: May 2022; Accepted after review: June 2022; Published: June 2022.

\*Corresponding author: Rana Irilouzadian, Department of Cardiology, Shohadaye Tajrish Hospital, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran  
Email: [ranairilouzadian@gmail.com](mailto:ranairilouzadian@gmail.com)



reported a rare case of myocarditis after using marijuana in a young man.

### Case report

A 24-year-old man with no known medical history, who works as an apartment caretaker, presented to our clinic with chest pain after marijuana consumption. He had consumed marijuana approximately twice a week in the last three months. He didn't have any history of cigarettes, alcohol or any other illicit drug

consumption. He had no previous complaint of fever or respiratory symptoms and no history of trauma. Laboratory tests were performed and were within normal ranges but he refused any other medical care and went back home. He returned to our emergency department the next day because of ongoing pain. Our initial differential diagnoses included acute coronary syndrome, myocarditis due to viral infections and marijuana-induced myocarditis. His laboratory findings showed an elevation of cardiac markers such as creatinine kinase-MB (CK-MB) and troponin-I levels (Table 1).

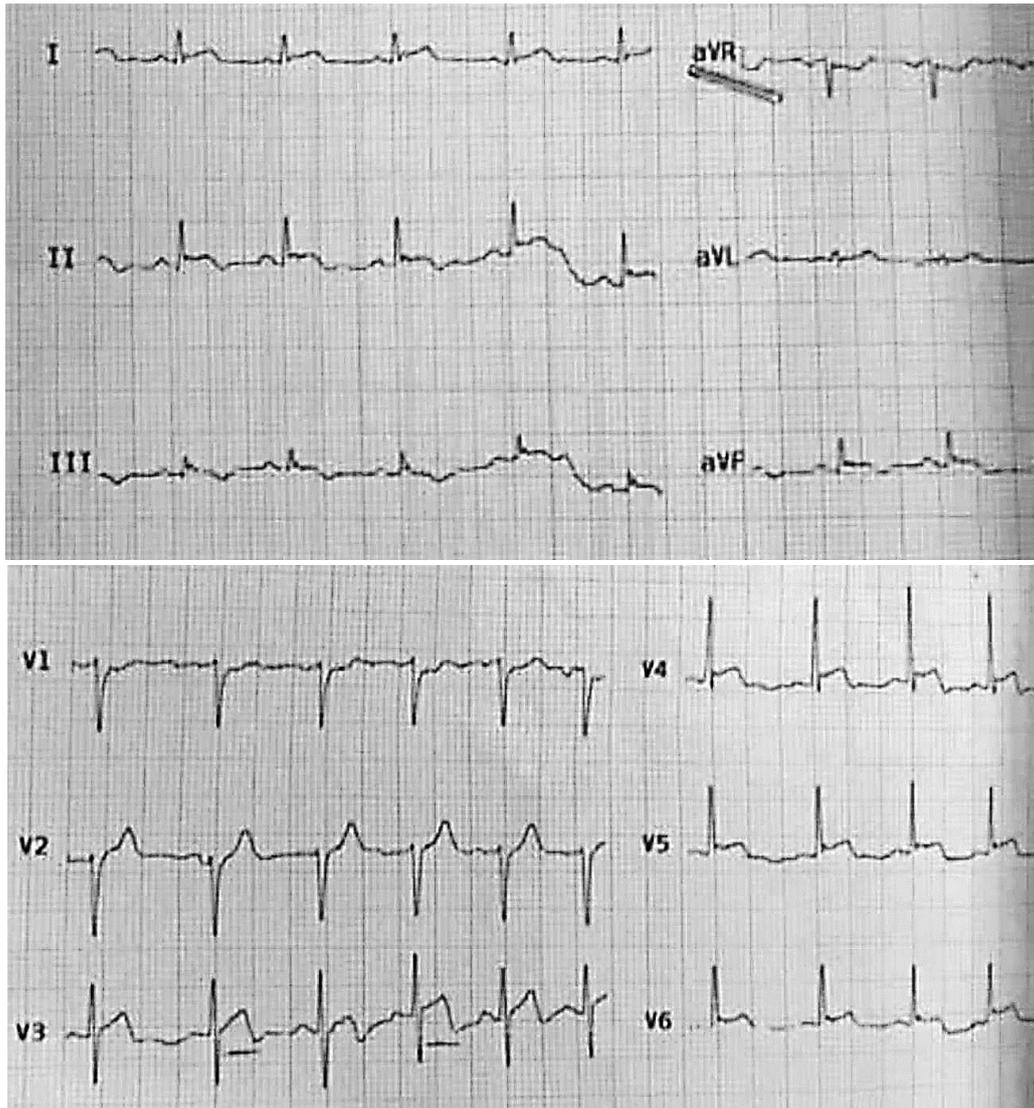
**Table 1.** Laboratory test results showed the elevation of troponin and CKMB

Lab Test	Result	Reference Range
BUN (mg/dL)	12	6-24
Cr (mg/dL)	0.76	0.5-1.5
K (Potassium), (mg/dL)	3.9	3.5-5.0
Na (Sodium), (mg/dL)	136	135-150
WBC (x 10 <sup>9</sup> /L)	9.9	4-11
Hb (g/dL)	15.3	12.5-16
Plt (x 10 <sup>9</sup> /L)	162	140-450
CK (U/L)	573	24-195
CK-MB (U/L)	47	1-24
Troponin I (ng/mL)	6.75	>0.12

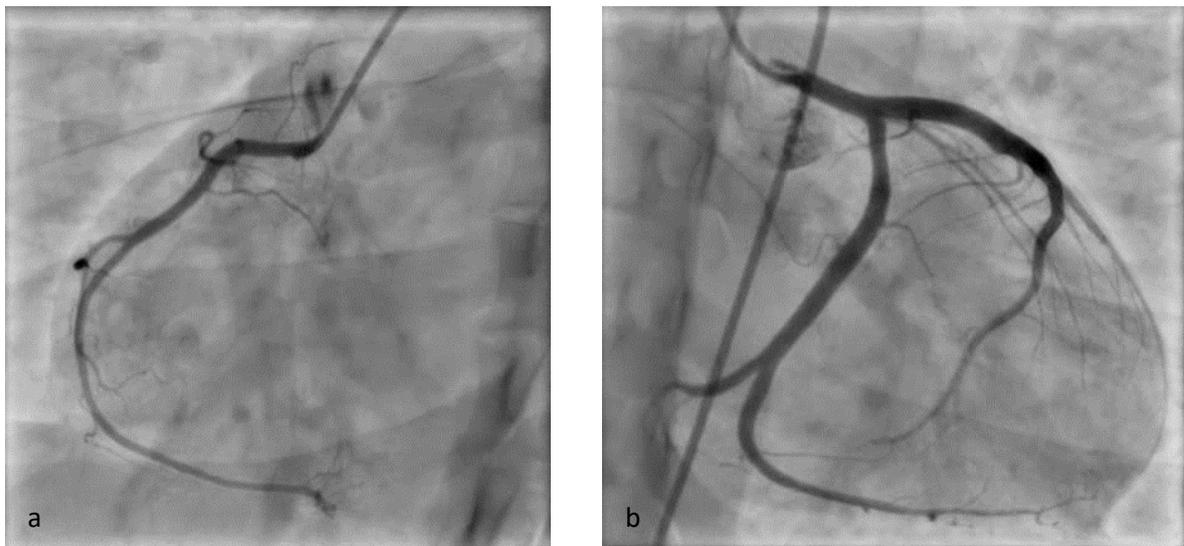
*Abbreviations:* BUN, blood urea nitrogen; Cr, creatinine; Plt, platelet count; Hb, hemoglobin; WBC, white blood cell count; CK-MB, creatinine kinase MB; CK, creatinine phosphokinase

Initial electrocardiogram (ECG) was obtained and showed ST-segment elevation in inferior and lateral leads with biphasic T wave in inferior leads and ST segment sublevel in aVR lead (Figure 1). First transthoracic echocardiography demonstrated normal left ventricle (LV) size and reduced LVEF (35%) with global myocardial hypokinesia and grade II diastolic dysfunction. Then, he was transferred to a heart center for further evaluation. Due to progressive chest pain, he became a candidate for emergency coronary angiography. Based on coronary angiogram (Figure 2), the patient had no coronary obstruction, myocardial bridge or spasm.

Three days later, follow-up echocardiography revealed smoky pattern in LV and a small, 13\*10 mm clot in apex (Figure 3) in the absence of local akinesia. During the follow-up, the electrocardiogram changed (Figure 4) with modification in the same territories, in addition being suggested the damage of the pericardium. For confirmation of myocarditis, cardiac MRI was also requested but the patient refused to undergo MRI. Oral anticoagulant, angiotensin receptor blocker and beta blocker were prescribed and the patient was discharged without any symptoms and in a good clinical condition. Counseling on quitting marijuana was conducted.



**Fig. 1.** Initial electrocardiogram with ST elevation in inferior (with biphasic T wave) and V3 to V6 leads and also, ST segment sublevel in aVR lead



**Fig. 2.** Coronary angiogram showing normal right (a) and left (b) coronary arteries

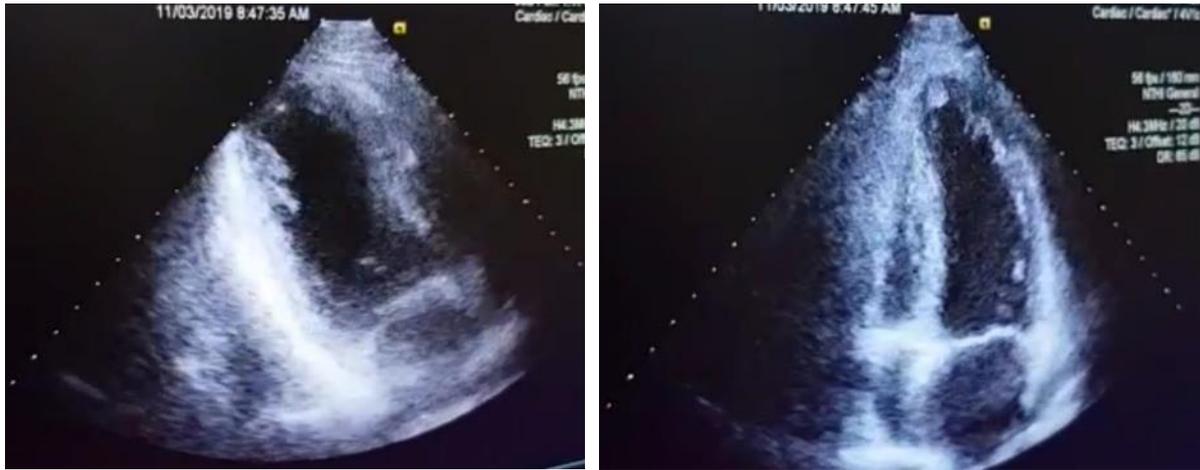


Fig. 3. Follow-up echocardiogram showing a small clot in cardiac apex.

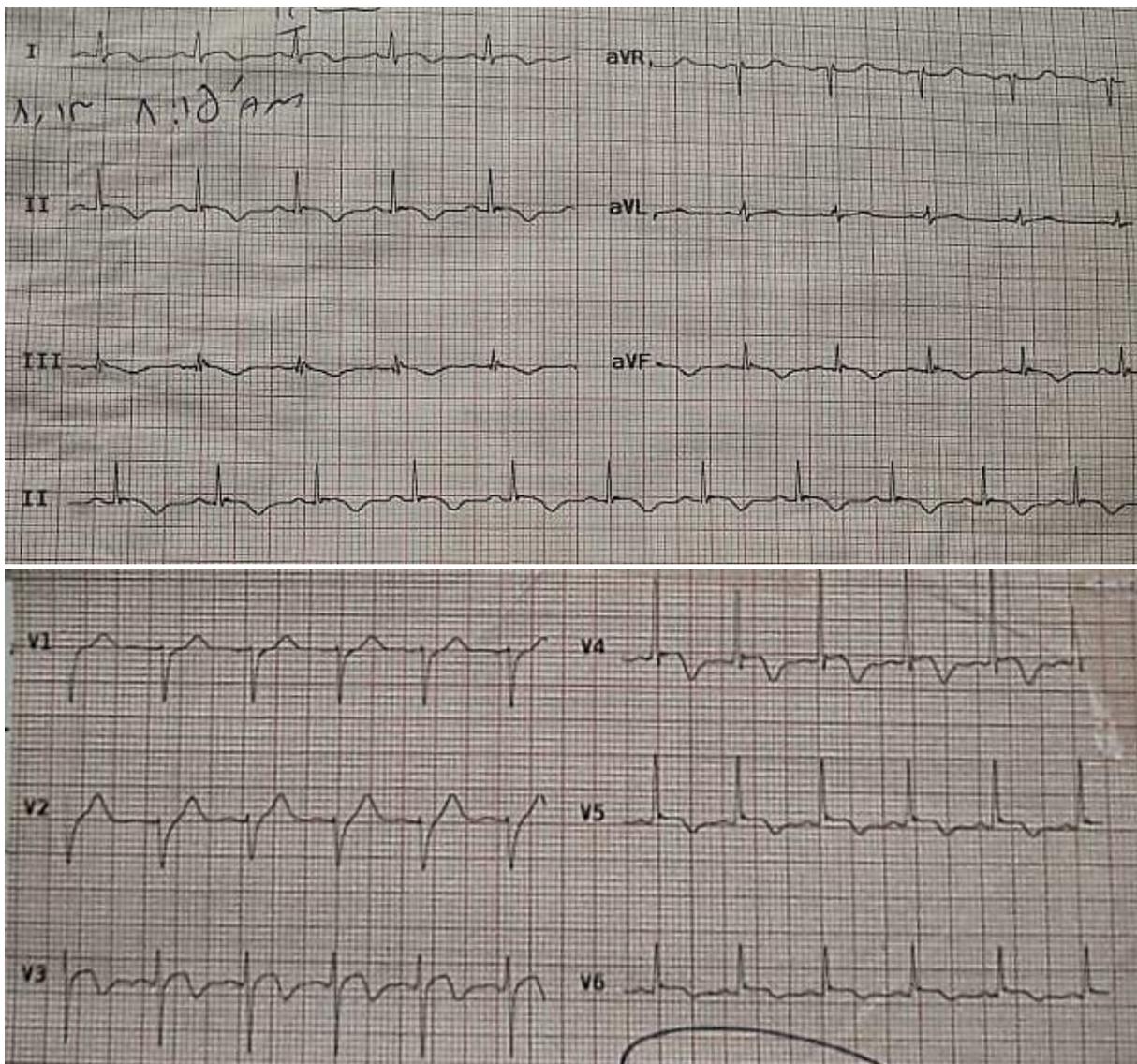


Fig. 4. The electrocardiogram during the follow-up showing the evolution of the changes in the same territories.

## Discussions

Marijuana, the most common drug of abuse, is one of the *Cannabis sativa* L plants family which contains tetrahydrocannabinol (THC), cannabidiol (CBD) and cannabinol (CBN) and has different effects on animals and human [15]. Marijuana has different receptors in body tissues and may affect the cardiovascular system by platelet aggregation, coronary spasms and marijuana-induced arteritis and lead to increased heart rate, systolic and diastolic blood pressures, sympathetic nervous system hyperactivity, myocardial infarctions (MIs) in young patients and coronary arterial vasospasm [16, 17].

Myocarditis is diagnosed by patient's clinical presentation and meeting the diagnostic criteria based on different imaging and lab tests [17]. The etiology of myocarditis is viruses, bacteria, fungi, protozoa, microbial toxins and etc. [18]. The pathogenesis of myocarditis after marijuana abuse is not recognized yet; marijuana may contain contaminants, heavy metals and pesticides that can affect the myocardial tissue [5] which warrants more researches in this context. The serum level of troponin and CK or CK-MB in myocarditis will increase as a result of myocardial injury [19], similar to our patient's elevated CK-MB and troponin. Since increased cardiac enzymes, ECG changes and chest pain can mimic MI, this may result in many unnecessary cardiac catheterizations or even fibrinolytic therapies in non-PCI capable centers. Although fibrinolytic therapy or coronary angiography in this setting makes sense but if we take a glance on

marijuana-induced myocarditis, we can prevent these procedures and their complications. The serum troponin levels have a direct relation to adverse outcomes in adult patients presenting with acute myocarditis [20].

## Conclusions

The unfamiliar and rare association between marijuana and myocarditis may have extended impact due to its widespread use globally. Nowadays, different forms of cannabis are used in various industries such as pharmaceutical, beauty and cosmetics and food. This raises the concern for rare side effects of this product as we discussed one in this report and warrants further researches regarding the pathophysiology of these side effects. We also suggest that specialists consider myocarditis in a patient who presents with chest pain after consuming marijuana.

## Competing interests

The authors of the present study declare that none of the authors have any conflict of interest.

## Consent for publication

A written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy and all of the authors declare that confidentiality of the patient was respected.

## Funding/Support

There is no funding to present study.

## References

1. World drug report 2021. Vienna: UN, Office on Drugs and Crime; 2021 [https://www.unodc.org/unodc/en/data-and-analysis/wdr2021.html available at 06/20/2022]
2. Suerken CK, Reboussin BA, Sutfin EL, Wagoner KG, Spangler J, Wolfson M. Prevalence of marijuana use at college entry and risk factors for initiation during freshman year. *Addict Behav.* 2014; 39(1):302-307. doi: 10.1016/j.addbeh.2013.10.018
3. Volkow ND, Baler RD, Compton WM, Weiss SR. Adverse health effects of marijuana use. *N Engl J Med.* 2014; 370(23):2219-2227. doi: 10.1056/NEJMra1402309
4. Amar MB. Cannabinoids in medicine: A review of their therapeutic potential. *J Ethnopharmacol.* 2006; 105(1-2):1-25. doi: 10.1016/j.jep.2006.02.001
5. McLaren J, Swift W, Dillon P, Allsop S. Cannabis potency and contamination: a review of the literature. *Addiction.* 2008; 103(7):1100-1109. doi: 10.1111/j.1360-0443.2008.02230.x
6. Mach F, Montecucco F, Steffens S. Cannabinoid receptors in acute and chronic



- complications of atherosclerosis. *Br J Pharmacol.* 2008; 153(2):290-298.
7. Kattoor A, Mehta J: Marijuana and coronary heart disease. American College of Cardiology [https://www.acc.org/latest-in-cardiology/articles/2016/09/22/08/58/marijuana-and-coronary-heart-disease available at 06/20/2022]
  8. Feldman AM, McNamara D. Myocarditis. *N Engl J Med.* 2000; 343(19):1388-1398. doi: 10.1056/NEJM200011093431908
  9. Fabre A, Sheppard MN. Sudden adult death syndrome and other non-ischaemic causes of sudden cardiac death. *Heart.* 2006; 92(3):316-320. doi: 10.1136/hrt.2004.045518
  10. Kariyanna PT, Jayarangaiah A, Singh N, et al. Marijuana induced myocarditis: a new entity of toxic myocarditis. *Am J Med Case Rep.* 2018; 6(9):169-172. doi: 10.12691/ajmcr-6-9-1
  11. Leontiadis E, Morshuis M, Arusoglu L, Cobaugh D, Koerfer R, El-Banayosy A. Thoratec left ventricular assist device removal after toxic myocarditis. *Ann Thorac Surg.* 2008; 86(6):1982-1985. doi: 10.1016/j.athoracsur.2008.04.092
  12. Rodríguez-Castro CE, Alkhateeb H, Elfar A, Saifuddin F, Abbas A, Siddiqui T. Recurrent myopericarditis as a complication of marijuana use. *Am J Case Rep.* 2014; 15:60-62. doi: 10.12659/AJCR.889808
  13. Tournebize J, Gibaja V, Puskarczyk E, Popovic B, Kahn J-P. Myocarditis and cannabis: An unusual association. *Toxicol Anal Clin.* 2016; 28(3):236. doi: 10.1016/j.toxac.2016.05.005
  14. Nappe TM, Hoyte CO. Pediatric death due to myocarditis after exposure to cannabis. *Clin Pract Cases Emerg Med.* 2017; 1(3):166-170. doi: 10.5811/cpcem.2017.1.33240
  15. Yamamoto I, Watanabe K, Matsunaga T, Kimura T, Funahashi T, Yoshimura H. Pharmacology and toxicology of major constituents of marijuana—on the metabolic activation of cannabinoids and its mechanism. *Toxin Rev.* 2003; 22(4):577-589. doi: 10.1081/TXR-120026915
  16. Franz CA, Frishman WH. Marijuana use and cardiovascular disease. *Cardiol Rev.* 2016; 24(4):158-162. doi: 10.1097/CRD.000000000000103
  17. Tso M, Kushneriuk DJ, Bree TL, Nosib SS. Incidental acute ST elevation due to cannabis-induced myocarditis after a mechanical fall. *CJC Open.* 2021; 3(10):1303-1306. doi: 10.1016/j.cjco.2021.05.008
  18. Magnani JW, Dec GW. Myocarditis: current trends in diagnosis and treatment. *Circulation.* 2006; 113(6):876-890. doi: 10.1161/CIRCULATIONAHA.105.584532
  19. Smith SC, Ladenson JH, Mason JW, Jaffe AS. Elevations of cardiac troponin I associated with myocarditis: experimental and clinical correlates. *Circulation.* 1997; 95(1):163-168. PMID: 8994432.
  20. Nishii M, Inomata T, Takehana H, et al. Serum levels of interleukin-10 on admission as a prognostic predictor of human fulminant myocarditis. *J Am Coll Cardiol.* 2004; 44(6):1292-1297. doi: 10.1016/j.jacc.2004.01.055

