

ST segment elevation myocardial infarction due to slow coronary flow occurring after cannabis consumption

Zawał serca z uniesieniem odcinka ST spowodowany zwolnieniem przepływu wieńcowego po zażyciu marihuany

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Abstract

Slow coronary flow (SCF) is an angiographic finding defined as the slow movement of contrast throughout the coronary lumen in the absence of epicardial coronary stenosis. It has been reported that SCF can on rare occasions cause ST elevated myocardial infarction (MI). Recent studies have shown that cannabis consumption can increase the risk of coronary heart disease and can trigger acute coronary syndromes, especially in young individuals without common risk factors. Here, we present a case of inferior MI in a patient who had consumed cannabis regularly over a long period and whose coronary angiography revealed SCF.

Key words: cannabis, slow coronary flow, ST-segment elevation myocardial infarction

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INTRODUCTION

Cannabis consumption is increasing, especially in the European Union. It has been proposed that cannabis is a risk factor for coronary heart disease (CHD) and can trigger acute coronary syndrome (ACS) [1]. ST segment elevation myocardial infarction (STEMI) associated with slow coronary flow is a rare condition [3]. Here, we present the case of a patient with STEMI following cannabis consumption. His coronary angiography showed normal anatomical coronary arteries, with prominent slow flow in the right coronary artery (RCA). We hypothesise that cannabis consumption was the main triggering factor for slow coronary flow and MI.

CASE REPORT

A 35 year-old man was admitted to the cardiology outpatient clinic complaining of chest pain of 12 hours' duration. Electrocardiography revealed pathological Q waves and 1 mm

ST elevation in inferior leads (Fig. 1). His past medical history was unremarkable. There was no family history of premature CHD. The patient smoked both cigarettes and cannabis. He had smoked tobacco for 20 years (30 cigarettes per day) and had smoked cannabis for ten years, (three joints per day). His chest pain had begun two hours after smoking cannabis. His physical examination did not reveal any specific findings. Blood pressure was 140/100 mm Hg and heart rate was 69/min. There was marked increase in cardiac markers (CK-MB: 82.9 ng/mL, troponin I: 14.1 ng/mL). He was hospitalised with a diagnosis of subacute inferior MI and coronary angiography was performed. Left main, left anterior descending (LAD) and circumflex (CX) arteries were normal. The RCA did not show any significant stenosis (Fig. 2). However, there was marked slowing of the coronary flow and delayed washout of contrast throughout lumen of RCA (Fig. 3). The TIMI frame count was within the normal range

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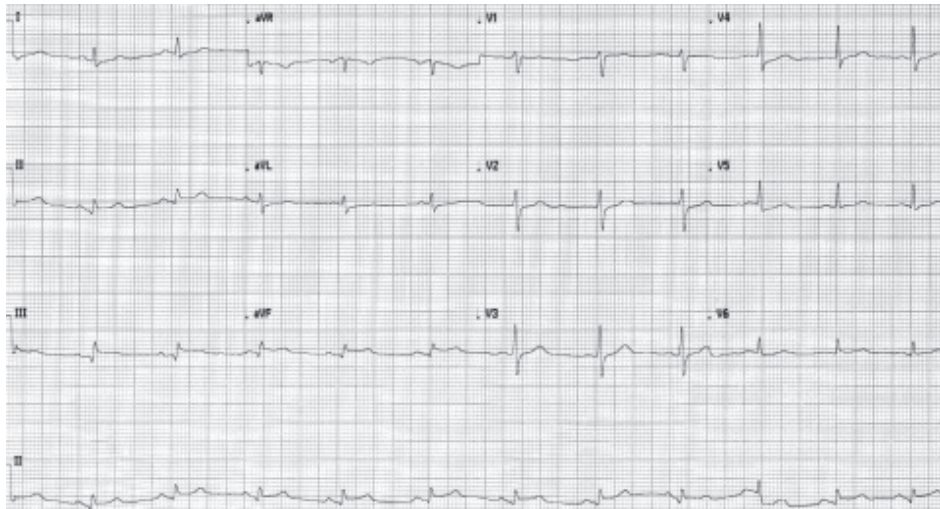


Figure 1. 12-lead ECG showing sinus rhythm and pathological Q waves with ST segment elevation in inferior leads

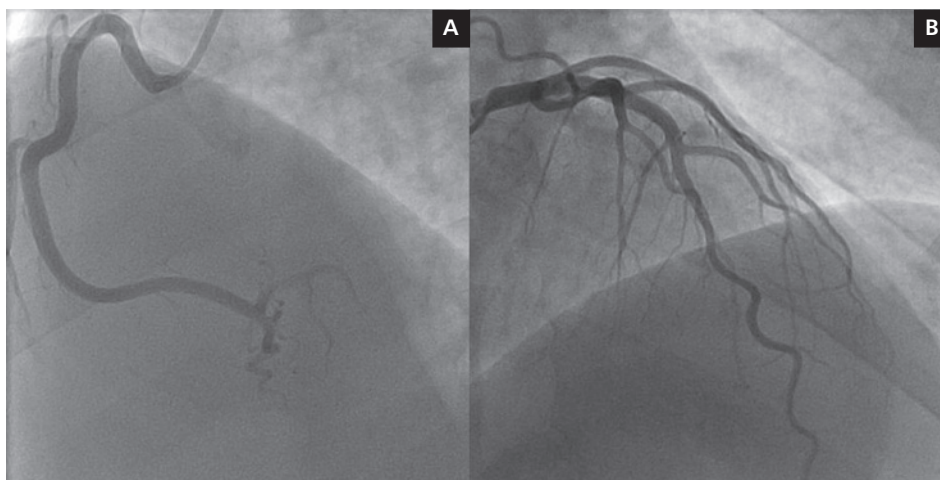


Figure 2. Left anterior oblique projection of right coronary artery (A); left anterior descending and circumflex arteries viewed in right cranial projection (B). In neither projection is there any significant stenosis or plaque formation

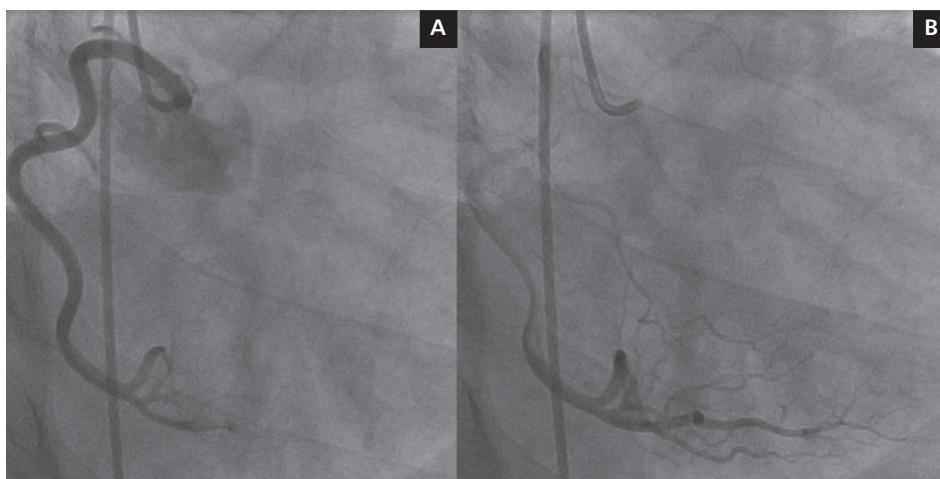


Figure 3. Right coronary artery viewed in the right anterior oblique view; filling (A) and wash-out phase (B)

for LAD and CX (32 and 25). The TIMI frame count for RCA was 50, which is abnormally high. Other laboratory findings: LDL cholesterol was 111 mg/dL, triglyceride was 75 mg/dL and glucose was 95 mg/dL. The patient was effectively treated with acetylsalicylic acid 300 mg, clopidogrel 75 mg, atorvastatine 80 mg, carvedilol 2×6.25 mg and enoxaparine, and was discharged 48 hours later.

DISCUSSION

Although there are nuclear figures regarding cannabis consumption in Turkey due to punitive government measures, it is widely used across the European Union. However, it is not unusual to detect a cannabis using patient with a detailed case history. Recently, a study performed in the Aegean region showed cannabis to be the most commonly used of the illicit drugs, substances and solvents [2]. The initial effects of cannabis occur via inhalation. Slight tachycardia and hypertension in the supine position, and prominent hypotension in the standing position, are its initial effects [4]. Cannabis increases cardiac output and carboxyhaemoglobin levels and thus can trigger myocardial ischaemia due to increased myocardial oxygen demand. Myocardial infarction in cannabis abusers is not uncommon [5, 6]. Patients described in the literature are usually young and free of classical risk factors. Angiographic data of such patients shows atherosclerosis, plaque rupture and thrombosis of coronary vessels. There has been no clear evidence of slow coronary flow in this patient group [1, 7]. It has also been shown that there is a five times greater risk of ACS within one hour of cannabis usage [8].

Slow coronary flow (SCF) is an angiographic finding defined as the slow movement of contrast throughout the coronary lumen in the absence of epicardial coronary stenosis. It is known that endothelial dysfunction and microvascular capillary disease are two main contributors to SCF. Although an association between SCF and ACS is a not infrequent finding, it has been reported that SCF in rare cases can cause STEMI [9]. In one study, ischaemia was documented in a patient with recurrent angina and SCF, using radionuclide via radionuclear scintigraphy [10]. The SCF is usually assessed via a visual TIMI score. For quantitative evaluation, the TIMI frame count is widely used, as proposed by Gibson et al. [11]. They found the average value of TIMI frame counts to be 36 ± 1 for LAD, 22.2 ± 4 for CX and 20.4 ± 3 for RCA. Yetkin et al. [12] examined 50 patients with MI SCF. They found a 36 ± 10 TIMI frame count for RCA. In our case, there was prominent and slow flow in RCA which seemed to be an infarct-related

artery. In our patient, TIMI grade 2a and a TIMI frame count of 50 were found, which is grossly abnormal.

Major causes of SCF are diabetes mellitus, hypertension, hyperlipidaemia and smoking. The effects of tobacco smoking on SCF progression have been well documented. However, such a correlation has not been shown for cannabis consumption [13]. Most cannabis abusers do not have the common risk factors for CHD, although their angiographic examinations show prominent atherosclerosis. This may indicate that cannabis can contribute to atherosclerosis progression, endothelial dysfunction and the formation of SCF. We believe that cannabis could be a major factor in SCF formation, separately from cigarette smoking.

References

1. Cappelli F, Lazzeri C, Gensini GF et al. Cannabis: a trigger for acute myocardial infarction? A case report. *J Cardiovasc Med (Hagerstown)*, 2008; 9: 725–728.
2. Aktas ÖE, Koçak A, Şenol E et al. Evaluation of narcotic and stimulant drugs abusers captured in Aegean region. *Türkiye Klinikleri J Foren Med*, 2006; 3: 13–14.
3. Uyarel H, Erden I, Cam N. Acute migraine attack, angina-like chest pain with documented ST-segment elevation and slow coronary flow. *Acta Cardiol*, 2005; 60: 221–223.
4. Korantzopoulos P, Liu T, Papaioannides D et al. Atrial fibrillation and marijuana smoking. *Int J Clin Pract*, 2008; 62: 308–313.
5. Kocabay G, Yildiz M, Duran NE et al. Acute inferior myocardial infarction due to cannabis smoking in a young man. *J Cardiovasc Med (Hagerstown)*, 2009; 10: 669–670.
6. Basnet S, Mander G, Nicolas R. Coronary vasospasm in an adolescent resulting from marijuana use. *Pediatr Cardiol*, 2009; 30: 543–545.
7. Dwivedi S, Kumar V, Aggarwal A. Cannabis smoking and acute coronary syndrome: two illustrative cases. *Int J Cardiol*, 2007; 128: e54–e57.
8. Mittleman MA, Lewis AR, Maclure M et al. Triggering myocardial infarction by marijuana. *Circulation*, 2001; 103: 2805–2809.
9. Li JJ, Wu YJ, Qin XW. Should slow coronary flow be considered as a coronary syndrome? *Med Hypotheses*, 2006; 66: 953–956.
10. Azzarelli S, Grasso C, Galassi AR et al. Coronary slow flow phenomenon: description of three cases evaluated with myocardial perfusion scintigraphy. *Ital Heart J*, 2005; 6: 341–344.
11. Gibson CM, Cannon CP, Daley WL et al. TIMI frame count: a quantitative method of assessing coronary artery flow. *Circulation*, 1996; 93: 879–888.
12. Yetkin E, Turhan H, Erbay AR et al. Increased thrombolysis in myocardial infarction frame count in patients with myocardial infarction and normal coronary arteriogram: a possible link between slow coronary flow and myocardial infarction. *Atherosclerosis*, 2005; 181: 193–199.
13. Erbay AR, Turhan H, Senen K et al. Documentation of slow coronary flow by the thrombolysis in myocardial infarction frame count in habitual smokers with angiographically normal coronary arteries. *Heart Vessels*, 2004; 19: 271–274.