References

1. Antman EM. ST-Segment Elevation Myocardial Infarction: Pathology, Pathophysiology, and Clinical Features In: Braunwald E, Bonow RO, Mann DL, Zipes DP, Libby P, editors. Braunwald's Heart Disease: a Textbook of Cardiovascular Medicine. 9th ed. Philadelphia: Elsevier Saunders; 2012. p.1087-110. [CrossRef]
2. Wijns W, Kolh P, Danchin N, Di Mario C, Falk V, Folliguet T, et al. Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS); European Association for Percutaneous Cardiovascular Interventions (EAPCI). Eur Heart J 2010; 31: 2501-55.
3. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, et al. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. J Am Coll Cardiol 2011; 58: e44-122. [CrossRef]
4. Vlaar PJ, Svilaas T, van der Horst IC, Diercks GF, Fokkema ML, de Smet BJ, et al. Cardiac death and reinfarction after 1 year in the Thrombus Aspiration during Percutaneous coronary intervention in Acute myocardial infarction Study (TAPAS): a 1-year follow-up study. Lancet 2008; 371: 1915-20. [CrossRef]
5. Svilaas T, Vlaar PJ, van der Horst IC, Diercks GF, de Smet BJ, van den Heuvel AF, et al. Thrombus aspiration during primary percutaneous coronary intervention. N Engl J Med 2008; 358: 557-67. [CrossRef]
6. Ho HH, Wong CP, Hau WK, Loh KK, Ong PJ. Intravascular ultrasound virtual histology imaging in acute ST-elevation myocardial infarction: A useful clinical tool during primary percutaneous coronary intervention. Acute Card Care 2011; 13: 245-7. [CrossRef]
7. Mirza A. Myocardial infarction resulting from nonatherosclerotic coronary artery diseases. Am J Emerg Med 2003; 21: 578-84. [CrossRef]
8. Dikmen M. Homosistein metabolizması ve hastalıklarla ilişkisi. Türkiye Klinikleri J Med Sci 2004; 24: 645-52.
9. Hoffman M. Hypothesis: hyperhomocysteinemia is an indicator of oxidant stress. Med Hypotheses 2011; 77: 1088-93. [CrossRef]
10. Brodie BR. Aspiration thrombectomy with primary PCI for STEMI: review of the data and current guidelines. J Invasive Cardiol 2010; 22(10 Suppl B): 2B-5B.

Address for Correspondence/Yazışma Adresi: Dr. Ali Çoner
Başkent Üniversitesi Tıp Fakültesi Hastanesi, Kardiyoloji Bölümü
Ankara-*Türkiye*
Phone: +90 312 212 68 68-1515 Fax: +90 312 223 73 33
E-mail: conerali@hotmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 17.12.2012

doi:10.5152/akd.2013.044

Cannabis smoking and sildenafil citrate induced acute coronary syndrome in a patient with myocardial bridge

Miyokart köprüsü olan bir hastada esrar içilmesi ve sildenafil sitrat ile indüklenen akut koroner sendrom

Introduction

Cannabis is a common substance of drug abuse among young adults because of its euphoric and addictive effects (1, 2). The pathophysiological effects of cannabis smoking and its relation to adverse cardiovascular events have been previously reported (1, 2). Sildenafil citrate is widely used as a primary pharmacological treatment of erectile dysfunction in men with and without underlying cardiovascular disease (3). However, the relative contribution of cannabis smoking when combined with sildenafil citrate in pathogenesis of acute coronary syndrome (ACS) is not well known.

We present here a case of cannabis smoking and sildenafil citrate induced ACS in a patient with myocardial bridge.

Case Report

A 42-year-old man presented to the emergency department with severe ongoing chest pain radiating to both arms followed by nausea and excessive sweating. The pain had started shortly after he had smoked two cannabis cigarettes with taking 50 mg sildenafil citrate and had engaged in sexual activity. Patient's history was normal except for smoking. Upon his visit to the emergency, the patient's blood pressure was 110/60 mmHg, with a rapid pulse of 130 beats/min. Initial electrocardiogram (ECG) showed sinus tachycardia and ST segment elevation in leads V1-V3 (Fig. 1A). Because the findings were thought to favor the ACS, the patient immediately underwent a coronary angiography. The left coronary angiogram revealed a myocardial bridging causing 100% systolic compression of mid-segment of left anterior descending artery with return to a normal caliber during diastole (Fig. 2) and right coronary angiogram showed hypoplastic coronary artery. Initial laboratory study revealed mildly elevated creatine kinase MB fraction with 7.1 ng/mL (normal range <5 ng/mL). The patient was started on aspirin, diltiazem, nitrate and lipid lowering agent and discharged home 4 days with disappearance of chest pain and ST elevation on ECG (Fig. 1B) after his cardiac catheterization. He remained asymptomatic and will be followed up regularly to determine whether abstinence from cannabis will prevent him from experiencing any future episodes of ACS.

Discussion

Cannabis derived from the plant Cannabis sativa is a common drug of abuse among young adults because of its euphoric and addictive

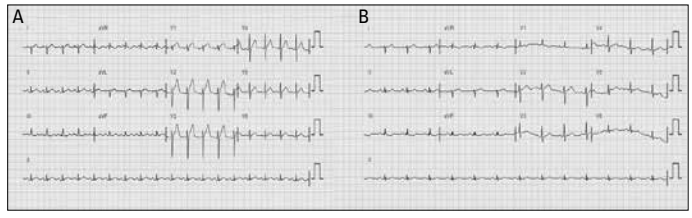

Figure 1. Initial electrocardiogram (ECG) showed sinus tachycardia and ST segment elevation in leads V1-V3 (A). There is no evidence of ST segment elevation on follow-up ECG (B)

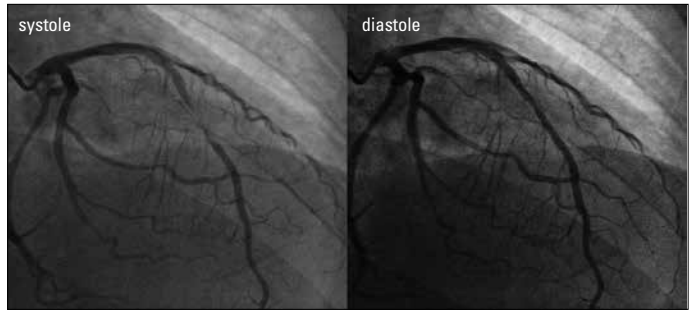

Figure 2. The left coronary angiogram revealed a myocardial bridging causing 100% systolic compression of mid-segment of left anterior descending artery with return to a normal caliber during diastole

effects (1, 2). The effects of cannabis are primarily mediated by the activation of cannabinoid receptors, which are present in brain, heart, blood vessels, spleen and immune system (1,2). Cannabis increases sympathetic activity while decreasing parasympathetic activity, producing tachycardia and increased myocardial contractility (4-6). Additionally, smoked cannabis is associated with an increase in carboxyhemoglobin, resulting in decreased oxygen-carrying capacity (4-6). Therefore, cannabis associated ACS may result from increased oxygen demand not met by a myocardial supply of oxygen (4-6). These adverse hemodynamic changes due to cannabis smoking may lead to plaque rupture in vulnerable individuals culminating in the ACS and sudden death (4-6). The ACS has also been reported in the presence of normal coronary arteries suggesting coronary vasospasm (4-6). Sildenafil citrate alone can cause mean peak reductions in systolic/diastolic blood pressure that are not dose related, whereas the heart rate is unchanged (3). Therefore, these adverse hemodynamic changes, particularly in association with aggravating factors such as decreased blood pressure due to sildenafil citrate may explain the occurrence of symptoms and myocardial ischemia in myocardial bridge.

Conclusion

Our case may suggest that coronary spasm in association with decreased blood pressure due to sildenafil citrate and myocardial bridge was the cause of the ACS in the absence of predisposing causes for thrombosis.

Acknowledgements
Authors want to thank Woo Jung Chun, MD, Ju Hyeon Oh, MD. for their help

Kyung Been Lee, Bong Gun Song, Gu Hyun Kang, Yong Hwan Park
Division of Cardiology, Cardiac and Vascular Center, Department of Medicine, Samsung Changwon Hospital, Sungkyunkwan University School of Medicine, Changwon-*Korea*

References

1. Ashton CH. Pharmacology and effects of cannabis: a brief review. Br J Psychiatry 2001; 178: 101-6. [CrossRef]
2. Caldicott DG, Holmes J, Roberts-Thomson KC, Mahar L. Keep off the grass: marijuana use and acute cardiovascular events. Eur J Emerg Med 2005; 12: 236-44. [CrossRef]
3. Jackson G, Montorsi P, Cheitlin MD. Cardiovascular safety of sildenafil citrate (Viagra): an updated perspective. Urology 2006; 68: 47-60. [CrossRef]
4. Bachs L, Morland H. Acute cardiovascular fatalities following cannabis use. Forensic Sci Int 2001; 124: 200-3. [CrossRef]
5. Fisher BA, Ghuran A, Vadamalai V, Antonios TF. Cardiovascular complications induced by cannabis smoking: a case report and review of the literature. Emerg Med J 2005; 22: 679-80. [CrossRef]
6. Lindsay AC, Foale RA, Warren O, Henry JA. Cannabis as a precipitant of cardiovascular emergencies. Int J Cardiol 2005; 104: 230-2. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Bong Gun Song
Division of Cardiology, Cardiac and Vascular Center
Department of Medicine, Samsung Changwon Hospital
Sungkyunkwan University School of Medicine
#50, Changwon, 630-723-*Korea*
Phone: 82-55-602-6327 Fax: 82-55-290-6654
E-mail: aerok111@hanmail.net
Available Online Date/Çevrimiçi Yayın Tarihi: 17.12.2012

doi:10.5152/akd.2013.045

A case of radial arteriovenous fistula during coronary angiography

Koroner anjiyografi esnasında oluşan radiyal arteriyovenöz fistül

Introduction

An arteriovenous fistula (AVF) is a complication after transradial approach and only three cases have been reported (1-3). All these cases were treated surgically.

We report a case of radial AVF, which developed during transradial coronary angiography and closed spontaneously during 2 months of follow- up period.

Case Report

A 62-year-old woman was referred to our hospital due to acute coronary syndrome for coronary angiography. Right radial approach was the route for coronary angiography. After the completion of the left coronary system angiography, we were unable to selectively engage the right coronary system with the 5F right Judkins catheter. At the first hand, we were satisfied with the unselective right coronary views. However, the patient`s clinical condition changed our opinion and we intended to use the 5F right Amplatz catheter. We introduced the 0035 guidewire again and tried to advance the wire. However, the patient became severely painful. An angiogram of the sheath revealed an AVF between the right radial artery and the adjacent vein opening up to the cephalic vein (Fig. 1, 2. Video 1, 2. See corresponding video/movie images at www.anakarder.com). The procedure was discontinued. After the consultation with the vascular surgeon and radiology consultant, it was agreed upon that the patient should be followed up with a vascular ultrasound. One week after the procedure, the right upper vascular ultrasound scan also revealed the AVF between the proximal right radial artery and the adjacent vein opening up to the cephalic vein. The peak systolic and diastolic flow rate in the fistula tract was measured as 53 cm/sec and 17 cm/sec, respectively (Fig. 3). Two months later the vascular ultrasound showed no sign of the fistula (Fig. 4).

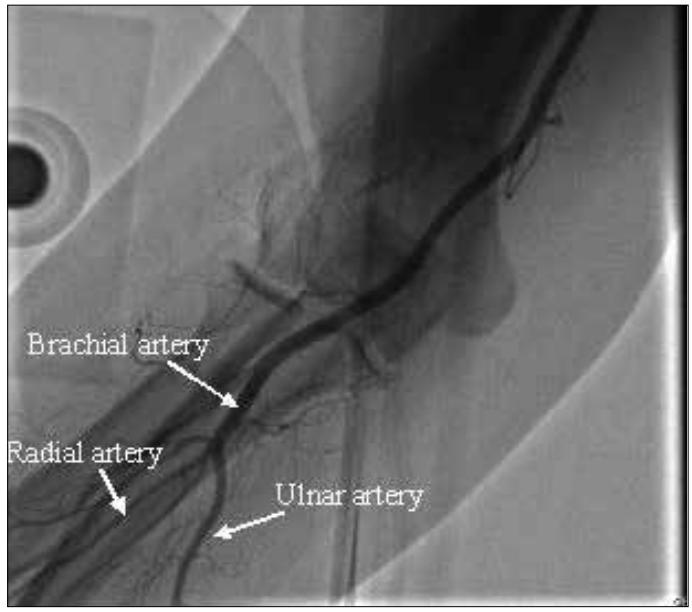

Figure 1. Normal anatomy of the forearm arteries before angiography