Case Report

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Ventricular Tachyarrhythmia after Sildenafil Intake: A Case Report

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Received: 27 December 2016 **Revised:** 19 January 2017 **Accepted:** 08 February 2017

ARTICLE INFO

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Keywords:
Sildenafil;
Ventricular arrhythmia;
Acute;
Myocardial infarction

ABSTRACT

This study reports the occurrence of ventricular tachyarrhythmia after sildenafil (Viagra®) intake. A 60-year-old man was admitted to the hospital with ventricular arrhythmia after sildenafil (Viagra®) intake. The echocardiography was normal, but in angiography, mild atherosclerotic plaques were seen. Comprehensive clinical trials are needed to prove any correlation between sildenafil intake and acute myocardial infarction.

Citation: Iranpoor D. **Ventricular Tachyarrhythmia after Sildenafil Intake: A Case Report**. Case Rep Clin Pract 2017; 2(1): 13-5.

Introduction

For about 2 decades, sildenafil has been used as an anti-erectile dysfunction agent, but several case reports have indicated that sildenafil was related to some cardiovascular adverse events (1, 2). Sildenafil is a –phosphodiesterase-5 (PDE-5) inhibitor which consequently breaks down 3', 5'-cyclic guanosine monophosphate (cGMP). The increase of cGMP relaxes the vascular smooth cells of the corpora cavernosa; moreover, it dilates pulmonary and systemic vessels (3, 4). In this case report, we present the case of a 60-year-old male patient with ventricular tachyarrhythmia after sildenafil intake.

Case Report

A 60-year-old man, without a history of any cardiac diseases and cardiac risk factors such as smoking, dyslipidemia, and hypertension, was admitted to the emergency ward of Bushehr Heart Center Hospital. The chief complaints were chest pain, palpitation, and weakness following Viagra® intake (25 mg, one tablet) 2 hours before admission. On examination, he was conscious, the heart rate was 200/minute, blood pressure was 80/50 mmHg, and respiratory rate was

18/minute. In cardiac examination, S1 and S2 were normal and no heart murmur was heard; moreover, the lung was clear with no rales and wheezing. The electrocardiography showed wide complex tachycardia with nearly 200 beats/minute [sustained ventricular tachycardia (VT)] (Figure 1).

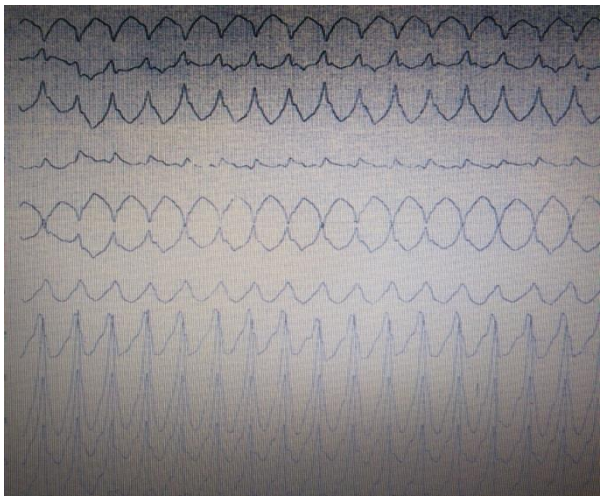

Figure 1. The electrocardiography of the patient showing ventricular tachycardia with a pulse rate of about 180-200 bpm

Due to the unstable hemodynamic condition and precollapsed state, VT was terminated by 50 Joule synchronized DC shock, and then amiodarone drip 0.5 mg intravenous maintenance was administered for 24 hours after admission. Then, the patient was transferred to the coronary care unit (CCU) for more monitoring and assessment. In the CCU, the serum level of electrolytes, such as Na/K/Ca/Mg, was measured that was within the normal range. Moreover, cardiac troponin/blood urea nitrogen/creatinine and complete blood count were measured that were also within the normal range. Echocardiography was performed for the patient and showed that the size and volume of heart chambers were normal. Ejection fraction and valvular function were normal without any abnormality in the wall motion of the right and left ventricles. However, in angiography, he showed mild atherosclerotic plaques without significant stenosis and thrombosis. In ventriculography, size and motion of the heart were normal, and no abnormality in mitral and aortic valves was detected. The patient was discharged after 2 days with amiodarone 200 mg (tablet) twice/day and recommended to an outpatient visit in the next week.

Discussion

In this case report, we present the case of a 60-year-old man with ventricular arrhythmia following sildenafil intake. In line with our findings, a case report in 2007 by Rasmussen presented a 41-year-old man with VT, 90 minutes after sildenafil intake. Similar to our case, the patient in that report did not show any family history of heart diseases and history of cardiopulmonary symptoms. The VT with the frequency of 220/minute was detected in the patient at admission (5). Moreover, another experimental study in pig revealed VT after administration of sildenafil with nitric oxide (6). The mechanism responsible for VT after sildenafil intake is not clear; however, a study in guinea pig heart indicated that the rapid component of the delayed rectifier potassium current is blocked by sildenafil citrate (7). Although these studies reported VT after sildenafil intake, it is well established that PDE-5 inhibitors are helpful for the treatment of those patients with erectile dysfunction and stable cardiovascular disease (Class-I, level of evidence A) (8). It is difficult to interpret these results in light of the potential relationship between sildenafil and VT because these findings have been presented by studies with small sample size and case reports. Hence, more randomized clinical trials with large series are required to confirm any possible correlation between sildenafil and acute myocardial infarction.

As a conclusion, more clinical trials are required to approve any correlation between sildenafil intakes with VT. It is recommended that people with erectile dysfunction should be fully examined before sildenafil consumption.

Conflict of Interests

Authors have no conflict of interests.

Acknowledgments

I would like to thank the patient for his contribution to the maintenance of our patient record without which this project would have been impossible.

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