Case Report

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Middle Cerebral Artery Infarction Associated with Sildenafil Citrate (Viagra) Use

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Abstract

İntroduction: Sildenafil is a phosphodiesterase 5 enzyme inhibitor used in the treatment of erectile dysfunction and pulmonary hypertension. The use of sildenafil with nitrates is well known to cause myocardial ischemia. However, patients with a history of hypotension, arrhythmia, previous myocardial infarction, or stroke should be given sildenafil carefully. A few cases have been reported in the literature related to ischemic stroke and transient ischemic attack due to sildenafil use.

Case report: We report a case of middle cerebral artery (MCA) infarction after two tablets sildenafil in a 57-year-old male patient with no underlying disease. In the diffusion MRI (magnetic resonance imaging) imaging of the patient, acute diffusion limitation was observed in the left frontal lobe and parietal lobe starting from the centrum semiovale level and also in the MCA supply area affecting the medial section of the temporal lobe. We believe that this infarction is caused by the development of severe hypotension due to alcohol use with sildenafil.

Conclusion: Not only availability of hypotension, history of MI and stroke, health care providers should also give attention prescribing sildenafil in the case of cigarette and alcohol use.

Key words: Sildenafil, ischemic stroke, hypotension

Introduction

Sildenafil citrate (Viagra; US Pharmaceutical Group, New York, NY, USA) is the drug of choice for most men with erectile dysfunction. The use of sildenafil with nitrates is well known to cause myocardial ischemia. However, patients with a history of hypotension, arrhythmia, previousmyocardial infarction, or stroke should be given sildenafil carefully. In both placebo-controlled and open-label studies, sildenafil was not associated with an increased risk of stroke[1]. These studies, however, excluded patients who had had myocardial infarction, lifethreatening

arrhythmia, or stroke in the previous 6 months—a population at very high risk for subsequent stroke. In a patient with no risk factor for a cerebrovascular event, we present MCA infarction after sildenafil and alcohol use.

Case Report

A 57-year-old, right-handed male patient came to the emergency room with a speech disorder, dropping of mouth, and inability to recognize his relatives for 2 days. He had no history of hypertension, diabetes, hyperlipidemia, heart disease or stroke. The patient had smoked 15 packs / year and used alcohol rarely.

The patient received two tablets sildenafil with alcohol 2 days ago. The relatives of the patient who think of alcohol as a cause of ill-consciousness did not bring the patient to the hospital on the first day. Since the patient did not improve his consciousness, his relatives brought him to the emergency room. In the physical examination, the patient was lethargic, did not have orientation and cooperation, or nuchal stiffness, his pupils were anisochoric, and myosis on his left eye was present. He had motor and sensory aphasia. The patient had hemiplegia on the right side.

His cranial tomography showed significant loss of density at the level of the left temporoparietal lobe.

MRI of the brain with diffusion-weighted imaging performed in the left frontoparietotemporal region, diffusion

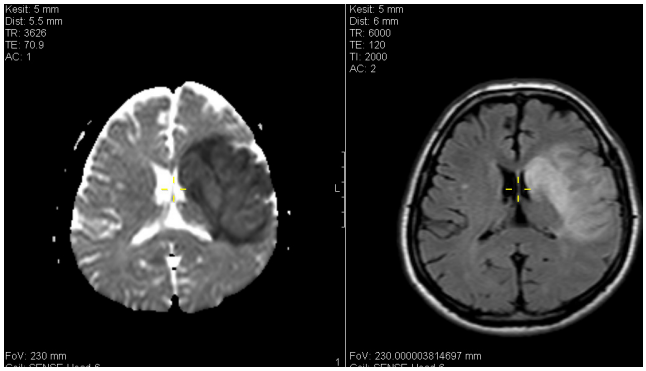

Figure 1: Cranial MRI image

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limitation was observed in the MCA supply area, consistent with acute infarction. In the evaluation of FLAIR images, the increase in signal area was noted (Fig. 1).

CT angiography ther was a plaque in the left Common Carotid Artery distal segment causing 50% stenosis. The left Internal Carotid Artery (ICA) was occluded, plaque was present at the right ICA proximal leading to critical stenosis. The left ICA shows retrograde filling and no contrast enhancement was observed at the MCA level. The patient was not given a tPA (Tissue Plasminogen Activator) and thrombectomy could not be performed because the patient admitted 24 hours later.

TPA and thrombectomy could not be performed because the patient admitted 24 hours later. Clexane 2* 60 mg, aspirin, plavix treatments were started.

His thyroid functions, creatine kinase (CK) and CK-myocardial band (MB) levels, Protein C and S were in normal range. Folate (5,5µ/L), B12 (152 µ/L) . Total cholesterol: 201 mg / dL Triglyceride: 321 mg / dL HDL: 33 mg / dL LDL: 104 mg / dL.

EKG showed normal sinus rhythm and a transthoracic echocardiogram did not reveal thrombus, vegetations, or other source of embolus, EF was measured as 55-60%. Valve pathology was not detected. After 6 days, brain magnetic resonance imaging revealed acute diffusion restriction on the left frontal lobe and parietal lobe starting from the centrum semiovale level, as well as the MCA supply area affecting the medial segment of the temporal lobe. Approximately 15 days of motor weakness and motor aphasia persisted and there was no improvement in right hemiplegia. The blood pressure was between 120/80 and 140/90 mmHg to date.

Discussion

Sildenafil is a phosphodiesterase inhibitor used in the treatment of sexual dysfunction and pulmonary hypertension. The mechanism of action is the inhibition of cyclic guanosine monophosphate (cGMP) -specific phosphodiesterase type 5 (PDE5). Increased cGMP increases the effect of nitric oxide (NO)[2]. Since there are relatively high levels of PDE5 in the human corpus cavernosum and in vascular, visceral and tracheal smooth muscles, NO leads to vasodilatation not only in the corpus cavernosum but also in systemic vessels. Systolic blood pressure decreases by 8-10 mmHg in patients due to systemic vasodilatation[3-4]. Sildenafil causes hyperemic nasal congestion, redness, headache and reduces pulmonary blood flow. Ischemic stroke due to sildenafil use is reported only a few times in the literature[5]. The etiology of vascular ischemia after sildenafil use is still uncertain. Several reports assume that hypotension, cardioembolism, or hypercoagulation is responsible. Although sildenafil has no direct effects on platelet function, it modestly potentates the inhibitory effect of the NO donor sodium nitroprusside on ADP-induced platelet aggregation ex vivo. According to this mechanism of action, adverse bleeding episodes are the major concern so, it is unlikely that sildenafil induces a hypercoagulable state[6-8]. When sildenafil is used with alcohol, the vasodilator effect increases and the tension decreases more. In our patient with symptomatic cerebrovascular disease, we think that the use of sildenafil together with alcohol can significantly reduce blood pressure and cause stroke. Another possible mechanism may be related to an increase in sympathetic activity.

Sildenafil-induced arterial vasodilatation has been shown to produce an increased pressure gradient on the left ventricular outflow tract, and pre-existing cardiomyopathy may be prone to produce atrial fibrillation[8]. It is believed that cardiovascular complications may occur with or before sexual activity[9-10].

Because sexual activity, sildenafil, drug and alcohol interactions or underlying cardiovascular disease are all miscible, it will be more difficult to determine the exact cause of the mechanism of action. It is known that sildenafil should be administered with caution to patients with hypotension, previous myocardial infarction, stroke or arrhythmia. Our experience with unilateral MCA region infarction due to sildenafil use suggests that sildenafil should be prescribed with caution in patients without risk factors.

Conclusion

Not only availability of hypotension, history of MI and stroke, health care providers should also give attention prescribing sildenafil in the case of cigarette and alcohol use

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