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Spontaneous Coronary Artery Dissection after Triptan Use in a Patient with Chronic Migraine

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ABSTRACT

Objective: We report a case of spontaneous coronary artery dissection of the distal interventricular artery in a woman with chronic migraine without aura or any cardiovascular risk factors and with several years of triptan use.

Background: Spontaneous coronary artery dissection can cause acute coronary symptoms in young patients, including those lacking cardiovascular risk factors. This condition mainly affects the distal part of the coronaries, which is most prone to hemodynamic changes. Risk factors include physical and emotional stress, inflammation, hormonal changes, and other factors.

Results: A 46-year-old woman with chronic migraine and without any background of cardiovascular risk factors was admitted to our hospital for an acute presentation of coronary symptoms. She had been managing her chronic migraine with a preventive therapy and the use of triptan (Rizatriptan) during migraine attacks, with a stable frequency of attacks at eight per month. These coronary symptoms occurred during a migraine attack, 2 hours after taking a second tablet of triptan (rizatriptan 10 mg). Initial examination showed a normal EKG with no ST-segment changes. Coronary angiography showed a dissection affecting the distal section of the left interventricular artery with a TIMI 3 flow (no downstream or upstream slowing of the blood flow) and a nonobstructive wall hematoma. The patient showed a full recovery at 6 months.

Conclusion: Spontaneous coronary artery dissection can occur in the context of triptan use, even when no cardiovascular risk factors are present. Therefore, the risk-benefit balance should be carefully considered before the implementation of therapeutic approaches for chronic migraine.

Keywords: Chronic migraine, Triptan, Spontaneous coronary artery dissection, Serotonin-norepinephrine reuptake inhibitor

Abbreviations: SCAD: Spontaneous Coronary Artery Dissection; CRF: Cardiovascular Risk Factor; IVA: Interventricular Artery; SNRI: Serotonin-Norepinephrine Reuptake Inhibitor

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a cause of acute coronary symptoms in young patients without any cardiovascular risk factors (CRFs). SCAD often affects the distal part of the coronaries, the region that is most prone to hemodynamic changes. The risk factors of SCAD include physical and emotional stress, inflammation, hormonal changes, and drug-related events. Here, we present a case of acute coronary syndrome secondary to a distal SCAD of the left interventricular artery (IVA) shortly after the intake of triptan in a patient with chronic migraine.

CASE REPORT

A 46-year-old patient with chronic migraine and no CRF presented to the emergency department with acute chest pain. The patient had a history of chronic migraine without aura since adolescence and typically experienced about eight attacks per month. This pattern and frequency had persisted for several years despite the introduction of a serotonin and norepinephrine reuptake inhibitor (SNRA, i.e., venlafaxine 75 mg per day) and lamotrigine (150 mg per day), which resulted in a decrease of the duration and intensity of the

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attacks. The use of triptan did not exceed 8 tablets per month. The patient had no history of any CRF. The patient had experienced a migraine attack without aura and took one tablet of rizatriptan 10 mg at 4:00 AM the same day. Because of the persistence of the migraine despite this treatment, the patient took a second dose at 6:00 AM. Two hours later, while attempting to lift a light weight and leaning forward, she experienced a sudden occurrence of oppressive left chest pain irradiating to the jaw. Upon admission to the emergency room, her vital signs were stable and clinical examination was normal. This chest pain was relieved by an administration of sublingual nitrate. Electrocardiography showed a regular sinus rhythm and a physiological right branch block without any other significant anomaly. Troponin levels were normal, as well as the rest of the cardiovascular workup (low-density lipoprotein: 1.15 g/L, high-density lipoprotein: 0.97 g/L, HbA1c: 5.1%, glomerular filtration greater than 90 mL/min). The autoimmune workup was also normal. Cardiac computed tomography revealed a hematoma in the wall of the distal left IVA, located in the posterior inter-ventricular furrow. No slowdown of the blood flow or other dissections were noted. The patient's clinical condition deteriorated, and conventional coronary angiography was performed, confirming the presence of a distal coronary dissection of the IVA with a TIMI 3 flow TIMI 3 (normal blood flow downstream and upstream of the stenosis). During this procedure, no coronary atheroma, infiltration, or calcification was noted. A transthoracic echocardiogram was also performed, without any significant anomaly. Finally, abdominal angiography was also normal and no argument for fibromuscular dysplasia was found. No thrombolytic therapy was required, and the patient was started on aspirin and beta-blockers. Rizatriptan was discontinued and replaced with paracetamol and anti-inflammatory agents. The patient's clinical condition improved over the following days and the wall hematoma was not visible on the follow-up scan 6 months later.

DISCUSSION

Rizatriptan is a powerful vasoconstrictor that acts primarily on the cerebral and meningeal vessels, alleviating the symptoms of acute migraine attacks. The therapeutic effects of triptan on migraine can be attributed to its agonistic effects at the serotonin 1B receptor (5-HT1B) and 5-hydroxytryptamine (serotonin) receptor 1D(5-HT1D) of the cerebral, meningeal, and extracerebral vessels. Activation of these 5-HT1B and 5-HT1D receptors can result in vasoconstriction and inhibition of the release of neuropeptides; this phenomenon decreases inflammation in sensitized tissues and transmission of pain signals centrally through the trigeminal sensory nerve pathway [1]. As a side effect, coronary spasm can occur, as well as other minor symptoms, such as tingling, dizziness, chest pain, muscle weakness, and neck pain. The combination of these signs is sometimes referred to as serotonin syndrome induced by triptan. However, cardiovascular adverse reactions occurring with the use of triptans within the recommended dosage are not generally serious [2]. In our patient, the interaction between triptan and venlafaxine might have increased this risk, as the main active metabolite of venlafaxine, O-demethylvenlafaxine, is responsible for tachycardia and cerebral and meningeal vasoconstriction (apart from the coronary arteries) through its effects on 5-hydroxytryptamine receptors. Although the concomitant use of these drugs is not strictly contraindicated, the prolonged-release form of venlafaxine is not recommended in patients under triptan therapy [3]. Our patient did not present any clinical signs of serotonin toxicity according to the Sternbach criteria (at least three of the following symptoms: changes in the mental state [confusion, hypomania], agitation, myoclonic seizures, hyporeflexia, diaphoresis, chills, tremor, diarrhea, lack of coordination, or fever [4]. Other triptans have also been implicated in cases of SCAD. A case of drug-related SCAD has been reported in a patient receiving extensive ergotamine tartrate as a treatment for migraine [5]. Additionally, another case of proximal right coronary artery SCAD (TIMI 3) has been reported with this same drug [6]. Ergotamine triggers adrenergic, dopaminergic, and serotoninergic activity, thereby stimulating smooth muscle cell contraction and sympathetic activity [6]. Moreover, certain immunosuppressant drugs, such as cyclosporin, are also associated with SCAD [7], as can physical exertion, atherosclerosis, vasculitis, connectivity (systemic erythematosus lupus, Marfan disease, and Ehlers-Danlos syndrome), and cocaine use [7]. In our patient, the physical exertion of leaning over to pick up a light bag might have played a role as Valsalva man oeuvres can trigger variations in pressure in the coronary arteries, particularly the distal portions, which are highly sensitive to variations in hemodynamic pressure in the cardiac cycle. SCAD predominantly affects women such as our patients, with higher incidence rates during pregnancy and the postpartum period, as well as in patients under contraception or hormone replacement therapy, suggesting a common pathological mechanism. Indeed, some cases suggest that the long-term use of high doses of estrogens may predispose to coronal structural fragility [8,9]. However, combining contraception with a triptan is not contraindicated [10]. In this case, we ruled out other causes of SCAD, such as fibromuscular dysplasia, dissection of the renal arteries or carotids, autoimmune disease, and concurrent hormone replacement therapy. Although SCAD is a rare disease, it remains a common cause of myocardial infarction. The incidence of SCAD is estimated at 0.1–1.1% [11] and tends to occur in young patients, who have similar risk profiles but higher levels of substance abuse and lower blood pressure [12,13].

Currently, patients with chronic thermoresistant migraine are candidates for calcitonin gene-related peptide antibodies as alternative treatments. These antibodies, which are currently

awaiting marketing authorization in Europe, act as vasodilators to provide symptom relief. Clinical trials have excluded patients with coronary artery damage, as reported in Multiple clinical trials of the CGRP monoclonal antibodies [14].

CONCLUSION

Chronic migraine is a worldwide health burden. Drug therapy approaches can lead to drug interactions between long-term preventive therapies and regimens used for acute attacks and risk-benefit balance should always be assessed. Combining SNRIs and triptan is not contraindicated; however, the prolonged-release forms of SNRIs should be avoided when patients take more than one tablet of triptan, because of the risk of enhanced serotoninergic effect. This can result in a potent arterial vasoconstrictor response, leading to iatrogenic-induced cardiovascular complications, including SCAD. In such cases, triptan and SNRIs should be discontinued. But further research is needed on the topic of drug interactions in migraine patients.

REFERENCES

1. Dodick D, Lipton RB, Martin V, Papademetriou V, Rosamond W, et al. (2004) Consensus statement: Cardiovascular safety profile of triptans (5-HT 1B/1D agonists) in the acute treatment of migraine. Headache 44(5): 414-425.
2. Robblee JV, Butterfield RJ, Kang AM, Smith JH (2020) Triptan and ergotamine overdoses in the United States: Analysis of the National Poison Data System. Neurology 94(14): e1460-e1469.
3. Leira EC, Cruz-Flores S, Leacock RO, Abdulrauf SI (2001) Sumatriptan can alleviate headaches due to carotid artery dissection. Headache 41(6): 590-591.
4. Evans RW, Tepper SJ, Shapiro RE, Sun-Edelstein C, Tietjen GE (2010) The FDA Alert on Serotonin Syndrome with Use of Triptans Combined with Selective Serotonin Reuptake Inhibitors or Selective Serotonin-Norepinephrine Reuptake Inhibitors: American Headache Society Position Paper. TJHFP 50(6): 1089-1099.
5. Garcia C, Casanovas N, Recasens L, Miranda F, Bruguera J (2007) Spontaneous coronary artery dissection in ergotamine abuse. Int J Cardiol 118(3): 410-411.
6. Giacoppo D, Capodanno D, Dangas G, Tamburino C (2015) Spontaneous coronary artery dissection. Int J Cardiol 175(1): 8-20.
7. Krishnamurthy M, Desai R, Patel H (2004) Spontaneous coronary artery dissection in the postpartum period: association with antiphospholipid antibody. Heart 90(9): e53.
8. Azam MN, Roberts DH, Logan WFWE (1995) Spontaneous coronary artery dissection associated with oral contraceptive use. Int J Cardiol 48(2): 195-198.
9. Akova-Öztürk E, Husstedt IW, Ringelstein EB, Evers S (2004) Carotid Artery Dissection in Ergotamine Abuse. Headache. TJHFP 44(9): 930-932.
10. Nesheiwat Z, Mangi MA, Zafrullah F, Kosinski D (2019) Multivessel Spontaneous Coronary Artery Dissection: Amberen as a Possible Risk Factor. Cureus 11(3): e4191.
11. Maeder M, Ammann P, Angehrn W, Rickli H (2005) Idiopathic spontaneous coronary artery dissection: Incidence, diagnosis and treatment. Int J Cardiol 101(3): 363-369.
12. Yang J, Biery DW, Singh A, Divakaran S, DeFilippis EM, et al. (2020) Risk Factors and Outcomes of Very Young Adults Who Experience Myocardial Infarction: The Partners YOUNG-MI Registry. Am J Med 133(5): 605-612.
13. Perez J, Lepelley M, Revol B, Roustit M, Cracowski JL (2022) Triptans and SCAD. J Ame C Cardio 78(21): 2129-2130.
14. Chiang CC, Schwedt TJ (2020) Calcitonin gene-related peptide (CGRP)-targeted therapies as preventive and acute treatments for migraine-The monoclonal antibodies and gepants. Prog Brain Res 255: 143-170.