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Case Report

Acute ST-elevation myocardial infraction after use of oral sumatriptan: a rare presentation

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ABSTRACT

Sumatriptan is an agonist of 5-hydroxytryptamine type-1 (5HT1) receptors that is widely used as a migraine abortant; however, there have been studies showing angina, coronary vasospasm, and even myocardial infraction in patients with predisposing cardiac risk factors. We present the case of a female patient with no cardiovascular risk factor who developed ST-elevation myocardial infraction 30 minutes after ingesting oral sumatriptan for her migraine.

Keywords: Sumatriptan, Migraine, Myocardial infraction

INTRODUCTION

Sumatriptan is an agonist of 5-hydroxytryptamine type-1 (5HT1) receptors that is widely used in the treatment of migraine. It improves migraines symptoms through intracranial vasoconstriction, mediated by inhibition of neuroinflammatory peptide release.¹ Sumatriptan and other 5-HT1 receptor agonists cause a sensation of chest tightness without electrocardiographic abnormalities in 3% to 5% of patients, and more rarely might provoke myocardial ischemia or cardiac dysrhythmia.²⁻⁴

Thrombolytic myocardial infraction after triptan use is rare, and in all reported cases to date, it has occurred after parenteral administration or in patients with atherosclerotic coronary artery disease.⁵⁻⁷ Here, we present the case of a female who developed ST-elevation myocardial infraction 30 minutes after ingesting sumatriptan for her migrainous symptoms. Triptan-induced vasospasm and infraction must be considered in patients with recent migraine treatment, even in those without cardiac risk factors.

CASE REPORT

A 45-year-old female presented to a community health center emergency department after having abrupt onset of severe retrosternal chest pain approximately 30 minutes after oral ingestion of 50 mg sumatriptan advised by family physician for her migraine. She had a past medical history of migraine for which she took naproxen and domperidone. She had no history of coronary artery disease (CAD), diabetes mellitus, pulmonary disorders, tobacco abuse, cocaine use, or any recent illness or injury. She did not take exogenous estrogen nor had any family history of heart disease.

The electrocardiogram at presentation revealed an acute anterior ST-elevation myocardial infraction. The presence of headache delayed the decision to thrombolysis. She was given sublingual nitrate and referred to a tertiary center for consideration of primary angioplasty. During patient transport, her pain gradually improved after use of sublingual nitrate.

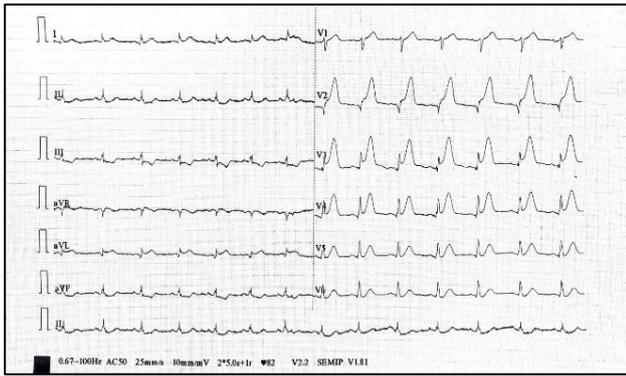


Figure 1: First Electrocardiogram (ECG) at presentation after use of oral sumatriptan.

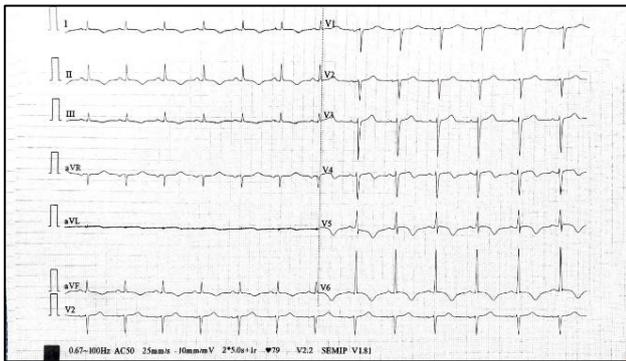


Figure 2: Second ECG done at cardiac center after use of sublingual nitrate.



Figure 3: Transthoracic echocardiography- apical 4 chamber view showing dilated LV apex.

Once she arrived to the cardiac center, her chest pain had nearly resolved and she had re-perfused electrocardiographically, with a resultant Q-wave anterior myocardial infarction. Transthoracic echocardiography revealed severely impaired left ventricle function with an akinetic anteroapical segment. Early coronary angiography revealed a right dominant coronary circulation with normal epicardial coronaries. The patient was treated with standard secondary preventive therapies and advised to permanently avoid triptan use.

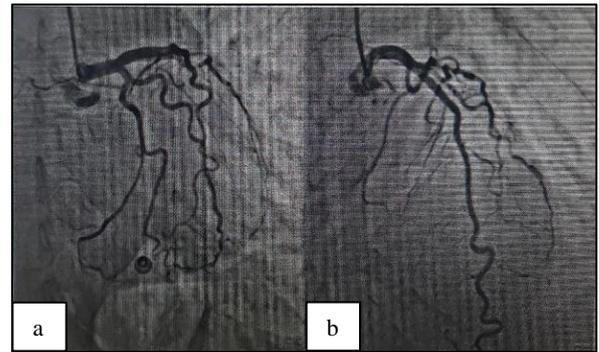


Figure 4: Coronary angiogram (a) RAO caudal view, (b) AP cranial view showing normal coronary arteries.

DISCUSSION

Sumatriptan belongs to the anti-migraine medication class called the triptans, which targets the 5-hydroxytryptamine (5-HT₁) serotonin receptor in vascular smooth muscle. Initially, these medications were believed to abort migraines by targeting the vasoconstricting 5-HT₁ receptors solely in the cerebral vasculature.⁸ Coronary circulation was believed to possess only serotonin 5-HT₂ receptors, ensuring that coronary vasoconstriction would be avoided in triptan use. Despite this, there have been studies showing vasoconstrictive effects in the coronary circulation with the injectable form of these medications.⁹ There have been few reports of patients having myocardial ischemia or infarction with the oral form of sumatriptan, as in this case, with even fewer showing coronary angiographic evidence of coronary spasm.¹⁰⁻¹²

Earlier reports have described triptan-related myocardial infarction in the context of atheromatous coronary artery disease, after parenteral triptan use, or vasospastic disease.⁵⁻⁷ Patients with coronary artery disease may be more susceptible to the vasomotor effect of sumatriptan and related drugs through increased expression of coronary hydroxytryptamine receptors, and triptan therapy is not recommended in these individuals.¹³ To our knowledge this is the first report of myocardial infarction after oral triptan use in a patient with normal coronary arteries from India.

CONCLUSION

Our case suggests that normal cardiac evaluation does not guarantee safety, so family physician should counsel the patient about the potential cardiovascular risk of sumatriptan, even if there is no prior history of CAD. If there are cardiac risk factors, this medication should be avoided, or first attempted under close medical supervision.

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Ethical approval: Not required

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