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CASE REPORT

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Zolmitriptan-induced acute myocardial infarction

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Abstract

Triptans are an established treatment for acute migraine attacks. By activating 5HT1B/1D receptors they lead to vasoconstriction of the cerebral blood vessels which are dilated during migraine attacks. Moreover, they reduce secretion of vasoactive peptides and conduction of pain stimuli over the cerebral cortex. In up to 7% of cases of treatment with triptans, thoracic pain occurs, although this is mostly transient, mild and without lasting ischemia. We present the case of a 45 year-old woman with a history of migraine with visual aura since the age of 20. She had no history of diabetes mellitus, hypertension, smoking or any other risk factors for cardiovascular events before she was admitted to our emergency room with typical chest pain. An electrocardiogram revealed anterior myocardial infarction following her monthly dose of oral zolmitriptan. Catherization revealed a normal coronary arterial system. The laboratory indices for cardiac risk were within normal ranges. The patient was advised to avoid triptans permanently on being discharged. (Cardiol J 2012; 19, 1: 76–78)

Key words: zolmitriptan, acute myocardial infarction, side effect

Introduction

Triptans are an established treatment for acute migraine attacks. By activating 5HT1B/1D-receptors they lead to vasoconstriction of the cerebral blood vessels which are dilated during migraine attacks [1]. Moreover, they reduce secretion of vasoactive peptides and conduction of pain stimuli over the cerebral cortex [1]. Although triptans exert their therapeutic effect through cerebral vasoconstriction, studies have also documented vasoconstriction in the coronary, pulmonary, and systemic vasculature [2]. In up to 7% of cases of treatment with triptans, thoracic pain occurs, although this isusually transient, mild and without lasting ischemia. The incidence of myocardial infarction under triptans is estimated to be very low [3]. Most published case reports refer to myocardial infarction after the use of sumatriptan (subcutaneous [4, 5] oral [6–8] and nasal [9] application), whereas both stenotic [5, 8] and normal [6–9] coronary arteries could be found angiographically. Besides sumatriptan, acute coronary syndrome has been described after the use of zolmitriptan [10–12] and frovatriptan [13]. The mechanism for these events is thought to be related to vasoconstriction superimposed on a preexisting coronary lesion [14]. However, these side effects can also be seen in patients with angiographically normal coronary arteries.

Case report

This 45 year-old woman had a history of migraine with visual aura since she was 20. She aver-

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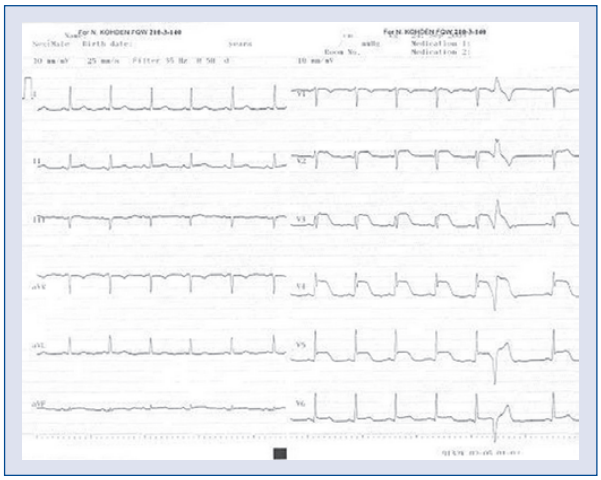

Figure 1. The electrocardiogram of the patient during her admission to the coronary care unit.

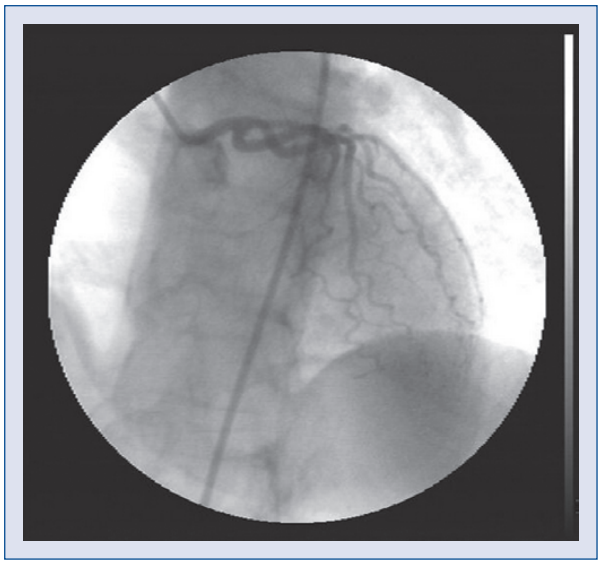

Figure 2. Normal coronary arteries in RAO caudal position in the coronary angiography.

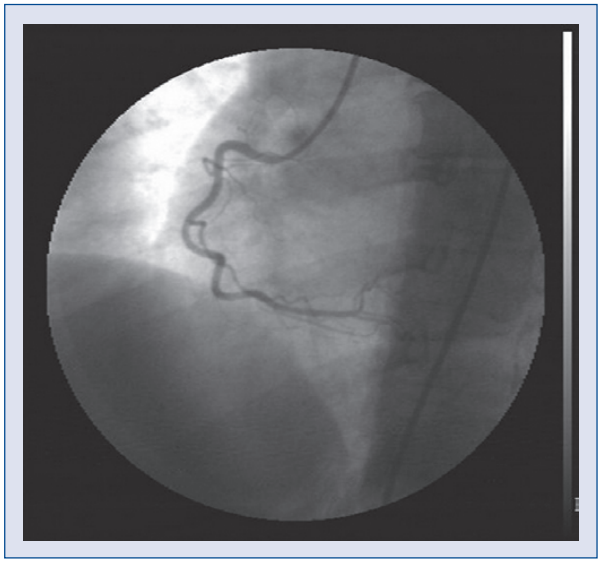

Figure 3. Normal right coronary artery on coronary angiogram.

aged one or two attacks per week and had been taking zolmitriptan for six years. On the day of admission, the patient had experienced a typical migraine attack and had taken 5 mg of zolmitriptan. Four hours later, she had a sudden onset chest pain, which she described as pressure localized to the retrosternal area. She repeated the same dose of zolmitriptan for cephalgia, while she was still having chest pain. She presented to the emergency room with ongoing chest pain, and a 12-lead electrocardiogram revealed ST-segment elevation in anterior precordial leads (Fig. 1). She had no history of diabetes mellitus, hypertension, smoking or other risk factors for cardiovascular events. She was admitted to the coronary care unit and a cardiac catheterization and additional laboratory studies were performed the following day. The catherization revealed a normal coronary arterial system (Figs. 2, 3). Laboratory indices for cardiac risk were within normal ranges. Cardiac enzymes and troponin-I were elevated. In her transthoracic echocardiogram, hypokinesia in the left ventricular anterior wall and apical part of the interventricular septum were reported, with an ejection fraction of 52%. The patient was given an angiotensin converting enzyme blocker and was advised to avoid triptans permanently when she was discharged. A control echocardiogram performed two months later revealed similar findings to that of the pre-discharge echocardiogram.

Discussion

Zolmitriptan, a 5HT1 agonist, belongs to a class of anti-migraine drugs that are known to cause vasoconstriction, precipitating chest pain in a significant proportion of patients, but rarely causing serious cardiovascular events. Zolmitriptan is an anti-migraine medication that shows its effect on the 5-hydroxytryptamine (5-HT) serotonin receptor. At first, this class of drugs was thought to affect solely cerebral vasculature by causing vasoconstriction. However, due to the presence of other 5-HT1-type receptors in coronary arteries, or varying degrees of receptor reactivities of these drugs, vasoconstriction on the coronary circulation has been seen and

has been widely documented *in-vivo* and *in-vitro* [2]. While almost all previously reported myocardial infarction, which were thought to be secondary to triptan use have occurred after administration of sumatriptan, recent reports involving tegaserod and zolmitriptan point to a possible class effect for these anti-migraine medications. This class effect is to be expected, as zolmitriptan has a similar mechanism of action as other triptans [14]. As mentioned previously, most cases reported have involved the use of sumatriptan. Hadjiloizou et al. [13] published a case of fravotriptan as a probable cause of vasoconstriction. Weder et al. [15] described for the first time an acute coronary syndrome in a 67 year-old female patient after oral intake of 2.5 mg naratriptan for migraine. Similarly to our case, three other cases of acute coronary syndrome with zolmitriptan have been reported [10–12], with one of the three--patients in question using zolmitriptan and citalopram concurrently.

To the best of our knowledge, our report represents the fourth case of acute coronary syndrome related to zolmitriptan. Normal coronary arteries were shown angiographically, so a vasospastic mechanism was considered. Although mostly seen in diseased coronary arteries, this side effect of triptans can also be recognized in normal coronary arteries. Our patient was advised to avoid triptans, but they can be the only course of treatment for some migraine patients. As pointed out by Evans et al. [14], physicians should attempt to use other abortive therapies in migraine patients with any cardiovascular risk factors. In those patients for whom no other effective options exist for migraine therapy, specific counseling regarding symptoms of myocardial ischemia should be given by physicians when prescribing these medications.

Conflict of interest: none declared

References

1. Tfelt-Hansen P, De Vries P, Saxena PR. Triptans in migraine: A comparative review of pharmacology, pharmacokinetics and efficacy. Drugs, 2000; 60: 1259–1287.
2. MacIntyre PD, Bhargava B, Hogg KJ, Gemmill JD, Hillis WS. Effect of subcutaneous sumatriptan, a selective 5HT1 agonist on the systemic, pulmonary, and coronary circulation. Circulation, 1993; 87: 401–405.
3. Dodick D, Lipton RB, Martin V et al. Triptan Cardiovascular Safety Expert Panel Consensus Statement: Cardiovascular safety profile of triptans (5HT1B/1D-agonists) in the acute treatment of migraine. Headache, 2004; 44: 414–425.
4. Ottervanger JP, Paalman HJ, Boxma GL, Stricker BH. Transmural myocardial infarction with sumatriptan. Lancet, 1993; 341: 861–862.
5. Anghileri E, Toso V, Perini F. Acute myocardial infarction after sumatriptan administration for cluster headache. Neurol Sci, 2006; 26: 456–459.
6. Weir RA, Oldroyd KG, Walters MR. Myocardial infarction after oral sumatriptan use in a woman with normal coronary arteries. Am J Med, 2007; 120: e7–e8.
7. O'Connor P, Gladstone P. Oral sumatriptan-associated transmural myocardial infarction. Neurology 1995; 45: 2274–2276.
8. Hack JB. Oral sumatriptan-induced myocardial infarction. J Toxicol Clin Toxicol, 2004; 42: 309–311.
9. Erbilen E, Ozhan H, Akdemir R, Yazici M. A case of myocardial infarction with sumatriptan use. Pediatr Cardiol, 2005; 26: 464–466.
10. Pacheco-Coronado R, McMullan PW, Galbut BH, Galbut EJ, Snell J, Schaer GL, Kavinsky CJ. Myocardial infarction after taking zolmitriptan. Yale J Biol Med, 2005; 78: 147–150.
11. Mikhail GW, Airoldi F, Colombo A. Acute myocardial infarction following the use of antimigraine therapy. J Invasive Cardiol, 2004; 16: 602–603.
12. Acikel S, Dogan M, Sari M, Kilic H, Akdemir R. Prinzmetal--variant angina in a patient using zolmitriptan and citalopram. Am J Emerg Med, 2010; 28: 257.e3–257.e6.
13. Hadjiloizou N, Rakhit R. Frovatriptan, a probable cause of coronary vasoconstriction. Int J Clin Pract, 2008; 62: 1121–1122.
14. Evans RW, Martin V. Assessing cardiac risk prior to use of triptans. Headache, 2000; 40: 599–602.
15. Weder CR, Schneemann M. Triptans and troponin: A case report. Orphanet J Rare Dis, 2009; 4: 15.