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Case Report

Rizatriptan-Induced Coronary Artery Spasm, Hypertension, and Sinus Tachycardia was Reversed with 5-Dinitrate

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Abstract

Rationale: Acute migraine is a common neurological emergency. Rizatriptan is a proven effective triptan for the acute management of migraine headache episode. Patient concerns: A 27-year-old single, the male patient presented in the emergency department with angina, hypertension, and sins tachycardia after ingested 20 mg of rizatriptan for an acute migraine attack. The case was admitted to the intensive care unit. Diagnosis: Rizatriptan-induced coronary artery spasm, hypertension, and sinus tachycardia was the suggested diagnosis. Interventions: Detailed physical examination, electrocardiography, and pulse oximetry assessment, and only managed with sublingual 5-dinitrate were the interventions for the current case. Outcomes: Dramatic termination of rizatriptan-induced above adverse effects to sublingual 5-dinitrate had happened. Lessons: Although the coronary artery spasm, hypertension, and sinus tachycardia were previously unknown occurrence post-oral 20 mg rizatriptan, all these adverse effects must be included in the differentiation workup of cases with unexplained and unusual acute symptoms. The novelty in this study was the dramatic response of the triple abnormalities; coronary artery spasm, hypertension, and sinus tachycardia with sublingual 5-dinitrate.

Keywords: Rizatriptan, Coronary Artery Spasm, Hypertension, Sinus Tachycardia, 5-Dinitrate

Introduction

Rizatriptan is a chemically selective 5-hydroxytryptamine1 subtype agonist (5-HT1B/1D). [1-3] However, according to the US Food and Drug Administration (FDA), rizatriptan is a proven effective triptan for the treatment of migraines in 1992. [1,3] The success of rizatriptan 5 and 10 mg in acute migraine has been distinctly reported.[4]

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Rapid relief of headache was the most common cause for a preference.[2] Rizatriptan possess immediate oral absorption and early onset of action[1,2,3,5] with approximately 90% absorbed from the gastrointestinal tract and 47% bioavailability via the first-pass metabolism.[4,5] Metabolism is the main route of rizatriptan excretion, but the renal excretion representing about 25% of total plasma clearance.[4] The drug plasma half-life is about 2–3 hours.[3,5] Monoamine oxidases are essential enzymes responsible for metabolizing rizatriptan.[1,3] The most common mechanism in the alleviation of migraine attacks is the stimulation of 5- hydroxytryptamine receptors, which decrease the brain blood flow responsible for migraine attacks.[1] Rizatriptan is quietly tolerated, with an overall incidence of adverse events and quality of life benefits similar to other triptans.[4] The recommended single adult dose is 5 mg.[6] The maximum recommended single dose is 10 mg. Rizatriptan 10 mg was more effective than rizatriptan 5 mg.[2] The choice of dose should be made on an individual basis, weighing the possible benefit of the 10 mg dose with the potential risk for increased adverse events.[6] Triptans are contraindicated in patients with ischemic heart disease, uncontrolled hypertension, a history of coronary vasospasm, and patients at high risk of asymptomatic coronary artery disease.[7,8] Regards rizatriptan acute toxicity, the approximate oral LD50 of rizatriptan was 700 mg/kg and 2227 mg/kg in mice and rats, respectively. The approximate IV LD50 values were 89 and 141 mg/kg in mice and rats, respectively.[6] Whatever, the summary of adverse events of rizatriptan for the treatment of acute migrainous attack, with specific adverse events incidence of 5% or more in patients receiving placebo, rizatriptan 5, or rizatriptan 10 mg were dizziness (4%, 6%, and 9%, respectively), sleepiness (4%, 5%, and 8%), nausea (4%, 5%, and 6%), and asthenia with fatigue (2%, 3%, and 5%).[5,9] In similar fashion, the most frequent adverse events in patients taking rizatriptan 5- or 10-mg tablet or wafer in comparative clinical trials included dizziness (5%-11% of patients across clinical trials), somnolence (4%-10%), asthenia or fatigue (2%-8%), dry mouth (2%-7%), nausea (2%-6%), and chest pain (1%-4%); these events were predominantly transient and mild or moderate in intensity.[5]

Coronary artery spasm (CAS) is an abnormal contraction of an epicardial coronary artery inducing-myocardial ischemia.[10] CAS plays an a pivotal role in the pathogenesis of IHD, including stable angina, UA, MI, and SCD.[11] Most CAS is accompanying with ST-segment depression rather than ST-segment elevation on ECG.[12] CAS may associated with normal coronary arteries on angiographically as so-named 'variant of the variant.[13] CAS is a multifactorial disease.[14] The morbidity increases annually in lifestyle-linked diseases, such as hypertension, diabetes mellitus, and dyslipidemia.[15] Calcium antagonists are the cornerstone of medical treatment of CAS.[14] Coronary angiography is an essential workup for a definitive diagnosis.[14]

Episodes are usually shortly and quickly alleviated by the administration of nitrates. [16] However, nitrates causing vasodilation with dominant venous effects on large capacitance vessels. They also increase coronary collateral circulation, increase aortic compliance and conductance and blood flow to ischemic areas of the myocardium. In addition, nitrates mitigate anginal symptoms by direct impact on the coronary arteries, coronary collateral circulation, aortic compliance and conductance, and blood flow to ischemic areas of the myocardium.[17] The ECG abnormalities are temporary and reversible with nitrates.[18] Short-acting nitrates are helpful in acute ischemic heart disease.[19] CAS is yielding a dramatic response to the vasodilator action of nitrates.[20]

Case report

A 27-year-old single, carpenter, Egyptian male patient presented in the emergency department with acute severe chest pain and dyspnea. Profuse sweating and palpitation were the associated symptoms. The patient gave a recent history of attack of migraine headache pushing him to ingested two tablets of rizatriptan (10 mg) to reliving the headache. The patient denied the history of cardiovascular diseases, smoking, drugs or special habits or the same attack. Upon physical examination; generally, the patient was a dyspnea, tachycardic, severe sweaty, and anxious, with a regular heart rate of 135 bpm, blood pressure of 160/100 mmHg, respiratory rate of 18 bpm, the temperature of 36.9 °C, pulse oximeter of O2

saturation; 95% and tachycardia on heart auscultation. No more relevant clinical data were noted during the clinical examination. He was admitted in the ICU as angina. Urgent ECG was done that showing sinus tachycardia and ST-segment depressions in V3-6 leads (Figure 1). The second ECG tracing was taken within 15 minutes of ICU admission that showing sinus tachycardia and ST-segment depressions in V3-5 leads ± V6 (Figure 2 A). Sublingual 5-dinitrate tablet was given (5 mg). O2 inhalation was given (100%, by nasal cannula, 5L/min). The third ECG tracing was taken within 150 minutes of second ECG tracing showing normalization of previous ST-segment depressions (Figure 2 B). The only measured random blood sugar was 177 mg/dl. Troponin test was negative. Later echocardiography was normal. No more workup was done. The case was initially managed as vasospastic angina. Rizatriptan-induced coronary artery spasm, hypertension, and sinus tachycardia was the most probable diagnosis. The case discharged within 12 hours after controlling the chest pain, hypertension, tachycardia, and electrocardiographic coronary artery spasm with recommended outpatient clinic follow up.

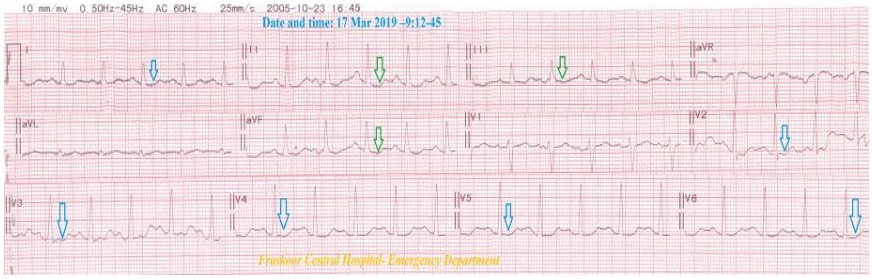

Figure 1. Urgent ECG tracing was taken in the emergency room showing sinus tachycardia and ST-segment depressions in II, III, aVF (green arrows), I, aVL, and V3-6 leads (blue arrows).

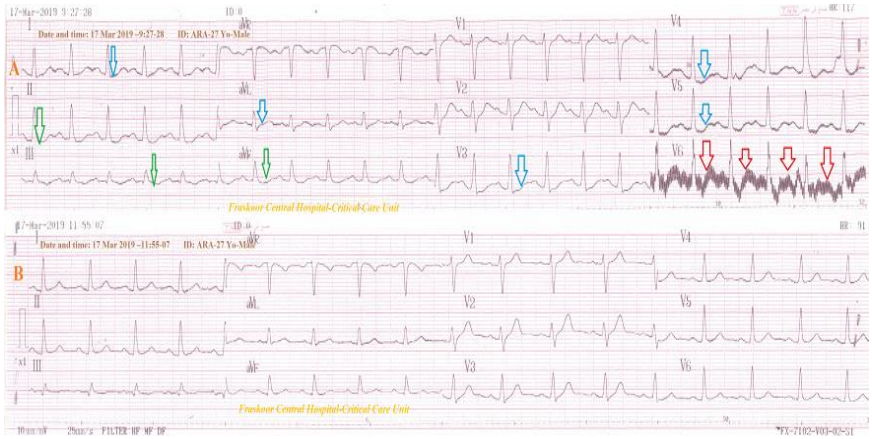

Figure 2. Serial ECG tracings. A tracing was taken within 15 minutes of ICU admission that showing sinus tachycardia and ST-segment depressions in V3-5 leads (blue arrows) ± V6 (author can't decide about ischemic change in V6 due to artifact) (red arrows). B tracing showing normalization of the above ST-segment depressions.

Discussion

The Overview of the case results included in the occurrence of coronary artery spasm, hypertension, and sinus tachycardia post oral intake of rizatriptan for reliving the acute migraine headache attack. We can't compare the current case study to other studies due to absent of similar condition. The primary objective for my case study was clinical an appearance of electrocardiographic coronary artery spasm and sinus tachycardia, and clinical hypertension post oral intake of rizatriptan. The secondary objective was the clinical improvement of patient symptoms with no further sequels and signifying the higher suspicion of rizatriptan-induced adverse effects. Although the coronary artery spasm, hypertension, and sinus tachycardia were previously unknown occurrence post-oral 20 mg rizatriptan, all these adverse effects should be included in the differential diagnosis workup of patients with unexplained and unusual acute symptoms. The novelty in this study

was the dramatic response of the triple abnormalities; coronary artery spasm, hypertension, and sinus tachycardia with sublingual 5-dinitrate. Serious chest pain including; acute coronary syndrome, aortic dissection, acute pericarditis, and pulmonary embolism often implicated in the differential diagnosis. This study was an observational retrospective case report. It is recommended to expect that rizatriptan may be inducing these adverse effects of coronary artery spasm, hypertension, and sinus tachycardia. Also, it is recommended to avoid rizatriptan in suspected ischemic heart disease, uncontrolled hypertension, a history of coronary vasospasm, and patients at high risk of asymptomatic coronary artery disease.

Conclusion

Rizatriptan may be inducing coronary artery spasm, hypertension, and sinus tachycardia. Refreshment the dramatic role of sublingual 5-dinitrate in reliving the triple abnormalities; coronary artery spasm, hypertension, and sinus tachycardia.

Author contributions

YE conceived of the study and participated in its design and coordination as well as helped to draft the manuscript; also read and approved the final manuscript.

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Conflict of interest

All authors declare that they have no conflict of interest.

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