Case report

Tramadol-Induced Hypertensive Crises and Myocardial Infarction with Possible Coronary Spasm in Smoker Patient

Yasser Mohammed Hassanain Elsayed

Critical Care Unit, Fraskour Central Hospital, Damietta Health Affairs, Damietta, Egypt

Received: 14 June 2019 / Accepted: 30 June 2019

Abstract

Rationale: Drug adverse effects are sometimes very serious and fatal. Hypertensive emergency and acute myocardial infarction had happened post-oral tramadol in a heavy smoker. Coronary artery spasm implicated in the pathogenesis of infarction. Patient concerns: A 45-year-old, heavy-smoker male patient presented to the emergency department with acute ST-segment elevation myocardial infarction post-ingested tramadol tablet. Diagnosis: Hypertensive emergency and acute myocardial infarction Interventions: Electrocardiography, intravenous nitroglycerin infusion, streptokinase infusion, cardiac enzymes, and later echocardiography. Outcomes: clinical and electrocardiographic dramatic response to both initial intravenous nitroglycerin infusion and later streptokinase infusion. Lessons: Myocardial infarction may be initially associated with negative troponin test and absent reciprocal changes. Tramadol may be inducing-acute ST-segment elevation myocardial infarction. So, how did tramadol-induced myocardial infarction and How to diagnose an infarction despite negative initial troponin test and absent reciprocal changes?

Keywords: Tramadol, Tramadol-induced hypertensive crises, Hypertensive emergency, Acute myocardial infarction, Possible coronary spasm in smoker patient

Introduction

Tramadol is an opioid analgesic and synthetic analog of codeine. Tramadol is a mu-opioid receptor agonist working at the central level. The analgesic effect and affinity of mu-opioid receptors for tramadol are 10-fold less than codeine\(^1\) Tramadol is a mostly given synthetic analgesic for both moderate and severe pain in primary circumference\(^1,2\)

Corresponding Author: Yasser Mohammed Hassanain Elsayed, MD, Critical Care Unit, Fraskour Central Hospital, Damietta Health Affairs, Egyptian Ministry of Health (MOH), Damietta, Egypt. E- mail: dryaser24@yahoo.com

DOI: 10.5455/ww.52835

This is an Open Access article under the terms of the Creative Commons Attribution-Non-Commercial 4.0 International License (https://creativecommons.org/licenses/by-nc/4.0/)
The initial dose is 25 mg and the maximum dose of 100 mg a day; adult dose should not exceed 400 mg a day. Tramadol is eminent as a less abuse substance and less associated respiratory depression. Serious possible side effects of tramadol are seizures, respiratory depression, serotonin syndrome, chest pain, dyspnea, tachycardia, and fainting. Other likely side effects of tramadol include; extreme drowsiness, swelling of the face, tongue, throat, or extremities, hallucinations, severe rash, thoughts of suicide, sleepiness, vomiting, itching, sweating, agitation, indigestion, dry mouth, diarrhea, tremor, loss of appetite, and sleeplessness. Gormel and his colleagues (2015) reported one case of tramadol induced Kounis syndrome with subsequent acute lateral myocardial infarction. It is hypothesized that vasospastic angina could be induced-myocardial infarction.

Hypertension is an extremely common cardiovascular and emergency problem, affecting about one billion individuals worldwide. At the same time, it causes an average of 7.1 million deaths a year around the world. Arterial hypertension is an essential independent risk factor for the development of cardiovascular disease and mortality in developed and developing countries. Approximately 1% of these patients will develop acute elevations in blood pressure at some point in their lifetime. Hypertensive crises (76% urgencies and 24% emergencies) represented 3% of all the patient visits, but 27% of all medical emergencies. Hypertensive crisis is defined as levels of systolic blood pressure >180 mmHg and/or levels of diastolic blood pressure >120 mmHg. Depending on whether there is damage to vital organs or not, we can distinguish between hypertensive emergency and hypertensive urgency. Hypertensive emergencies occur in up to 2% of patients with systemic hypertension. High blood pressure is an emergency modality. This situation is life-threatening conditions because of complicated outcome by acute injury to vital tissues, and can be presented with neurologic, renal, cardiovascular, microangiopathic and obstetric complications. When the blood pressure is too high, it can cause by hypertensive encephalopathy, hypertensive acute left ventricular relaxation associated with acute myocardial infarction or unstable angina, aortic dissection, subarachnoid hemorrhage, ischemic stroke, and severe pre-eclampsia or eclampsia. Hypertensive emergency is a modality with a severe increase in blood pressure without progressive dysfunction of vital organs. The most common symptoms are headache, dyspnea, nausea, vomiting, epistaxis, and pronounced anxiety. Immediate reduction in blood pressure is required only in patients with acute end-organ damage. Nitroglycerin as a potent venodilator reduces BP, decreasing preload and cardiac output Therefore, it is not an acceptable first choice for hypertensive emergencies except in patients with acute coronary ischemia.

The specific clinical situation may be absent and a non-specific elevation of serum levels of troponin I could be detectable. The 12-lead electrocardiogram (ECG) is an integral part of the pathognomonic workup of a patient with acute chest discomfort. This method is the easiest and most convenient tool to diagnose or rule out myocardial infarction. It may also be an indicator of response to treatment. However, do not rely on reciprocal changes to diagnose STEMI. Some cases may only have ST elevation with no reciprocal changes. Negative cardiac troponin result does not rule out angina or ischaemic heart disease. Coronary artery disease is present in at least a third of patients with low-risk clinical features and negative serum troponin I value throughout the first 12 hours of admission.

Coronary artery spasm (CAS) is strong vasoconstriction of coronary arteries that causes total or partial artery blockage. CAS with transient ST-segment elevation can occur in diseased coronary arteries as Prinzmetal's variant angina. It may also occur in angiographically normal coronary arteries as so-called variant of the variant. CAS plays an important role in the pathogenesis of ischemic heart disease, including stable angina, unstable angina, myocardial infarction, and sudden death. Approximately, hypertension and hypercholesterolemia are the commonest causes of coronary artery spasm. Smoking is a major risk factor for vasospastic angina. Calcium antagonists are the main therapeutic option. Coronary angiography and provocative testing are essential tests for the definitive diagnosis of coronary artery spasm.
Case presentations

A 45-year-old married heavy-smoker Egyptian male driver patient presented to the emergency department with acute severe agonizing chest pain. Chest pain was a teary compressive referred to the back. There was a past history of hypertension on furosemide tablet (40 mg once daily), and captopril tablet (25 mg twice daily). The patient gave a recent history of taken oral tramadol tablet (50 mg) since about 2 hours. The patient denied any history of cardiovascular disease or other relevant diseases. Upon examination, the patient appeared irritable, sweaty, and anxious. His vital signs were as follows: blood pressure of 200/130 mmHg, pulse rate of 100/minute; regular, the temperature of 36.9°C, respiratory rate of 18/min, and initial pulse oximetry of 97 %. No relevant local cardio-respiratory signs. The patient was admitted in ICU and initially managed with O2 inhalation using nasal cannula in the rate of 5 L/min and sublingual isosorbide dinitrate tablet (5 mg), sublingual captopril tablet (25 mg). ECG tracing was the initial workup that showed sinus tachycardia (VR;100 bpm) with ST-segment elevation myocardial infarction in anterior leads V2-6 (Figure 1 A). Intravenous nitroglycerin infusion (5 µg/min with intermittent titration) was given. Serial ECG tracings were taken. Pethidine HCL (100 mg) was given for chest pain in intermittent doses. Chest pain was still compliant. Bisoprolol oral tablet (5 mg) was added. No significant change in ECG within 45 minutes of first ECG tracing (Figure A-C). After 45 minutes of first ECG tracing (Figure D-E) ST-segment elevation myocardial infarction appeared extended in (I, aVL, aVF) leads (Figure 2 C). Blood pressure was controlled within two hours of admission (150/90 mmHg). But, chest pain was still compliant. Aspirin 4 oral tablet (75 mg), clopidogrel 4 oral tablet (75 mg), streptokinase IVI (1.5 million units over 60 minutes) were added. ECG tracing was taken within 12 hours of streptokinase infusion. ST-segment elevation started to regress pathological Q wave started to develop (Figure 2 F). The patient became complete symptomatically free after streptokinase infusion. Chest pain had absolutely disappeared. Initial troponin I test was negative. Repeated troponin I became positive within 6 hours was positive (311 ng/Liter). RBS was 237 mg/dl on admission. Later echocardiography showed anterior hypokinesia with EF 57 %. No other workup abnormality. The patient was continued on; captopril tablet (25 mg twice daily), aspirin tablet (75 mg, once daily), clopidogrel tablet (75 mg, once daily), nitroglycerin retard capsule (2.5 mg twice daily), and atorvastatin (40 mg once daily) until discharged on the 5th day.

Figure 1. Serial ECG tracings in ICU. Tracing A; Initial presentation showing acute STEMI. ST-segment elevation in V2-6 carry the criteria of acute STEMI (Red arrows), no ST-segment elevation in I, aVL leads (Blue arrows), no reciprocal changes in inferior leads (II, III, aVF) (Green arrows). Tracing B, and C; is nearly the same as tracing A, but; vibration artifacts in III, aVL leads in Tracing B (Multiple small black arrows).
Discussion
In my case; an acute myocardial infarction and hypertensive emergency post-oral tramadol had happened. ST-segment elevation carries the criteria of acute STEMI. CAS is often implicated suggested mechanism in inducing acute STEMI. Smoking was a major risk factor. Negative initial troponin test with absent reciprocal changes does not rule out the diagnosis of STEMI. Delayed disappearance of acute chest pain after streptokinase therapy indicates severe coronary artery occlusion. The severity of acute chest pain and its character me implicate aortic dissection in the differential diagnosis. So, bisoprolol as a β-blocker therapy was added. PVCs post-streptokinase therapy may indicate the success of coronary artery reperfusion. I can’t compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

• Study question; How did tramadol-induced myocardial infarction? and How to diagnose an infarction despite negative initial troponin test and absent reciprocal changes?
• The primary objective; for my case study was clearing the existence of the hypertensive emergency and acute myocardial infarction post-oral tramadol in a heavy smoker.
• The secondary objective; for the case study was IV controlling of hypertensive emergency with nitroglycerin and subsequent streptokinase therapy infusion for acute myocardial infarction
• Limitations of the study; There are no known limitations to the study.
It is recommended to widening the research in clearing the effect of tramadol in inducing-acute myocardial infarction and hypertensive crises. Also, it is recommended to avoid tramadol in the presence of any risk factor for coronary artery spasm such as smoking.

Conclusions
Tramadol may inducing-acute ST-segment elevation myocardial infarction. CAS is often implicated suggested mechanism in inducing acute STMI. Smoking was a major risk factor. the priority of management for any physician
should be directed to identifying the etiology and to know the drug adverse effects. Recommendations and cautious on using tramadol especially in smokers and the ischemic patient will be advised.

**Conflicts of interest:**
There are no conflicts of interest.

**Acknowledgment:**
I wish to thank nurses of the critical care unit and emergency department who make extra ECG copy for helping me.

**References**