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A Case Report

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# Musculoskeletal Chest Pain and Acute Myocardial Infarction with Psychological Stress Overlapping Using of Multiple Oral NSAIDs and Possible Coronary Spasm-Interpretation and Outcome

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## Abstract

***Rationale:*** *The musculoskeletal chest pain may be angina or a heart attack. Acute myocardial infarction is a serious cardiovascular disease. Psychosocial stress may be a risk factor for the acute coronary syndrome. Drug adverse effects may be critical and fatal. Oral non-steroidal anti-inflammatory drug use is associated with an increased risk of acute myocardial infarction. Coronary artery spasm is a cardiovascular disorder that has a remarkable role in the pathogenesis of stable angina, unstable angina, myocardial infarction, and sudden cardiac death.*

***Patient concerns:*** *An elderly male farmer patient presented to the emergency department with acute severe agonizing chest wall and epigastric pain with psychological stress post-ingested combination of intermittent irregular oral diclofenac, ibuprofen, and celecoxib tablets.*

***Diagnosis:*** *Acute inferior and possible associated posterior myocardial infarction post psychological stress overlapping using of multiple NSAIDs with probable coronary spasm.*

***Interventions:*** *Electrocardiography, streptokinase infusion, cardiac enzymes, and later echocardiography.*

***Outcomes:*** *clinical and electrocardiographic dramatic response to streptokinase infusion.*

***Lessons:*** *Understanding drug pharmacokinetics and pharmacodynamics is a pivotal step for all clinicians. Oral non-steroidal anti-inflammatory drugs and psychosocial stress should be considered in dealing with all diseases. A dramatic response for thrombolytic therapy in the presence of both inferior ST-segment elevation and posterior myocardial infarction affection carries a good prognosis. Coronary artery spasms may be interpretative suggested mechanisms in inducing acute myocardial infarction.*

**KeyWords:** Acute Myocardial Infarction, Coronary Artery Spasm, Ischemic Heart Disease, Musculoskeletal Chest Pain, Psychological Stress, Nsaids

## Abbreviations

CAS; Coronary artery spasm
ECG; Electrocardiogram
ED: Emergency department
ICU; Intensive care unit
IHD: ischemic heart disease
MI: myocardial infarctions
NSAID; Non-steroidal anti-inflammatory drug
O2: Oxygen
RV MI: right ventricular myocardial infarction
SCD; Sudden cardiac death
STEMI: ST-Elevation Myocardial Infarction
UA: Unstable angina
VR: Ventricular rate

## 1. Introduction

The musculoskeletal system may be a renowned source of chest pain. S, Tietze syndrome, costochondritis, chest wall syndrome, muscle tenderness, slipping rib, cervical angina, and segmental dysfunction of the cervical and thoracic spine are reported conditions to cause musculoskeletal chest pain [1]. The musculoskeletal chest pain may be angina or heart attack [2]. It is important to note that cardiopulmonary such as acute coronary syndrome and musculoskeletal chest pain syndromes can present similarly [3]. Early diagnosis of the musculoskeletal syndromes causing chest pain is an important key in the efficient differentiation and specific management of pain [4].

Psychosocial stress may be a risk factor for acute coronary syndrome, although the mechanisms underlying pathogenesis is still uncertain [5]. Oral non-steroidal anti-inflammatory drug (NSAID) use is associated with an increased risk of acute myocardial infarction (MI) according to a meta-analysis published in the BMJ. "Risk was greatest during the first month of NSAID use and with higher doses," report the researchers[6]. Higher doses of NSAIDs (celecoxib >200 mg, diclofenac >100 mg, ibuprofen >1,200 mg, naproxen >750 mg, and rofecoxib >25 mg) were associated with greater risks of MI. With prolonged use of NSAIDs, the risk of MI remained constantly elevated6. All NSAIDs are nearly accompanied by an increased risk of MI7. The onset of risk of acute MI mostly happens in the first week and the greatest occurs in the first month of treatment with higher doses [6,7].

**The Coronary Artery Spasm (CAS)** is a cardiovascular disorder applied to describe an abrupt, intense narrowing of an epicardial coronary artery that causes vessel occlusion or near-occlusion [8]. CAS plays an important role in the pathogenesis of ischemic heart disease (IHD), including stable angina (SA), unstable angina (UA), myocardial infarction (MI), and sudden cardiac death (SCD) [9]. So, malignant ventricular arrhythmias, acute MI, and SCD are essential serious sequels of obstructive CAS [9]. Emotional stress, anger, and fear are leading leading substratum for the attacks [10].

## 2. Case Presentations

A 59-year-old married male, Farmer, heavy smoker, Egyptian patient presented to the emergency department (ED) with acute severe agonizing musculoskeletal chest wall and epigastric pain for about 3 hours. The patient gave a recent history of taking intermittent irregular oral diclofenac tablets (100 mg, BID), oral ibuprofen tablets (400 mg, TID), and oral celecoxib tablets (100 mg, BID) for about 60 days for low back pain. Vomiting, nausea, fatigue, tachypnea, profuse sweating, and dizziness were associated symptoms. The patient denied any history of cardiovascular disease or other relevant diseases. Upon examination, the patient appeared distressed, sweaty, and anxious. His vital signs were as follows: blood pressure of 110/70 mmHg, pulse rate of 58/minute; a regular, temperature of 36°C, respiratory rate of 25/min, and initial pulse oximetry of 95 %. No relevant local cardio-respiratory signs. Initial emergency ECG tracing on presentation showed sinus bradycardia of VR; 57 bpm with mild ST-segment elevations with small q-waves in inferior leads (II, III, and aVF) and reciprocal ST-segment depressions in I, aVL, and V2-6 leads. There is evidence of posterior myocardial infarction in V2 and 3 (Figure 1A). The patient was admitted to the intensive care unit (ICU) as an acute inferior with evidence of posterior myocardial infarction. He was initially managed with O2 inhalation using a nasal cannula at the rate of 5 L/min. Pethidine HCL (100 mg) was given for chest pain in intermittent doses as needed. Serial ECG tracings were taken. ECG Tracing was done on the ICU admission within 15 minutes of the above ECG tracing and just before given streptokinase showing sinus bradycardia of VR; 58 bpm with clear inferior ST-segment elevations, pathological Q-waves in inferior leads (II, III, and aVF), and reciprocal ST-segment depressions in I, aVL, and V1-6 leads. There is evidence of posterior myocardial infarction in V2 and 3 (Figure 1B). ECG Tracing is a right side ECG tracing that was done within 15 minutes of the above ECG tracing showing there is no evidence of right ventricular (RV) MI (V3R and V4R) (Figure 1C). Aspirin; 4 oral tablets (75 mg, then OD), clopidogrel; 4 oral tablets (75 mg, then OD), streptokinase IVI (1.5 million units), enoxaparin SC (60mg, BID), and atorvastatin (20 mg, OD) were given. ECG Tracing was done within 6 hours of ICU admission showing complete resolution of above ST-segment elevations and reciprocal ST-segment depressions but with still sinus bradycardia of VR; 58 bpm.

There is evidence of pathological Q-waves in inferior leads (II, III, and aVF) (Figure 1D). The patient became completely symptomatically free after streptokinase infusion. The initial CBC showed leukocytosis (14000/mm3). SGPT (18U/L) and SGOT (27U/L) were normal. Serum creatinine (1.5 mg/dl) and blood urea (59mg/dl) were slightly high. The troponin I test was positive (7ng/l). Ionized calcium was slightly low (1.0mmol/L). RBS was 86 mg/dl on admission. Later echocardiography showed inferior hypokinesia with EF 52 %. No other workup abnormality. The patient was continued on aspirin tablet (75 mg, OD), clopidogrel tablet (75 mg, OD), nitroglycerin retard capsule (2.5 mg, BID, started within 72 of presentation), enoxaparin SC (60mg, BID), captopril tablet (12.5 mg, OD), and atorvastatin (20 mg, OD) until discharged on the 5th day. Acute inferior and possible associated posterior myocardial infarction post psychological stress over the use of multiple NSAIDs with probable coronary spasm in the elderly patient was the most probable current diagnosis. The patient was advised to have future outpatient cardiovascular follow-ups.

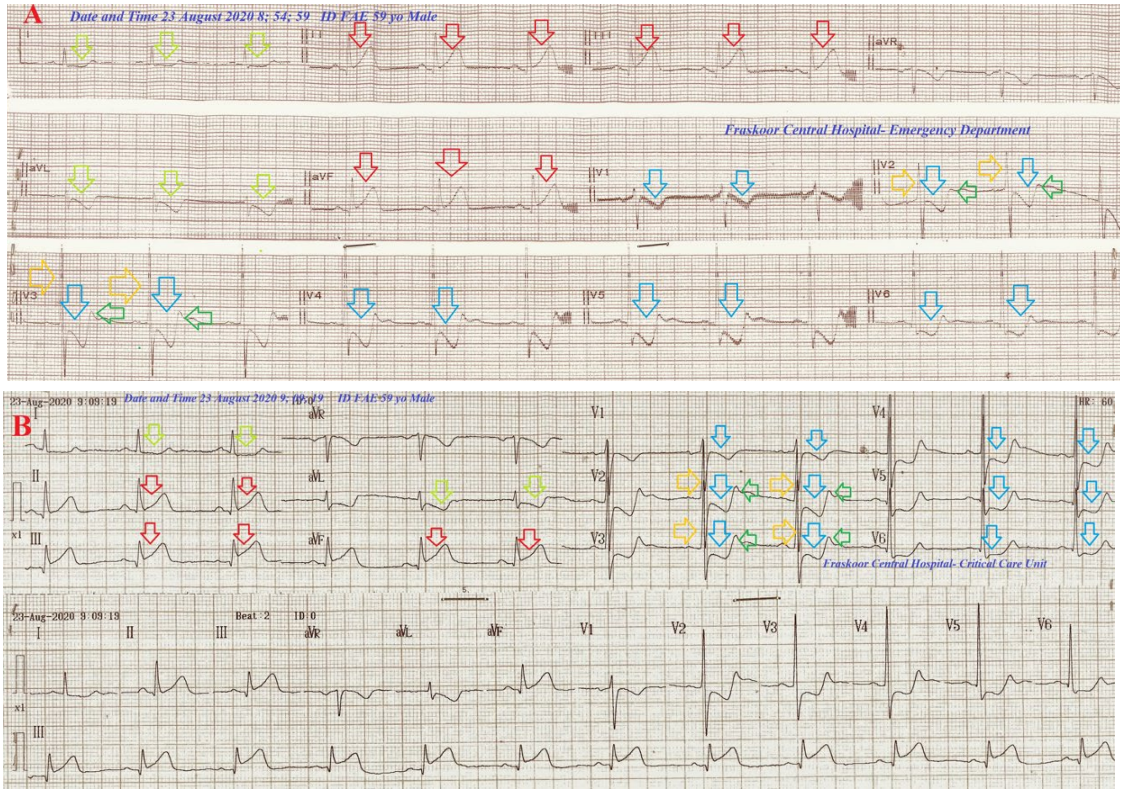
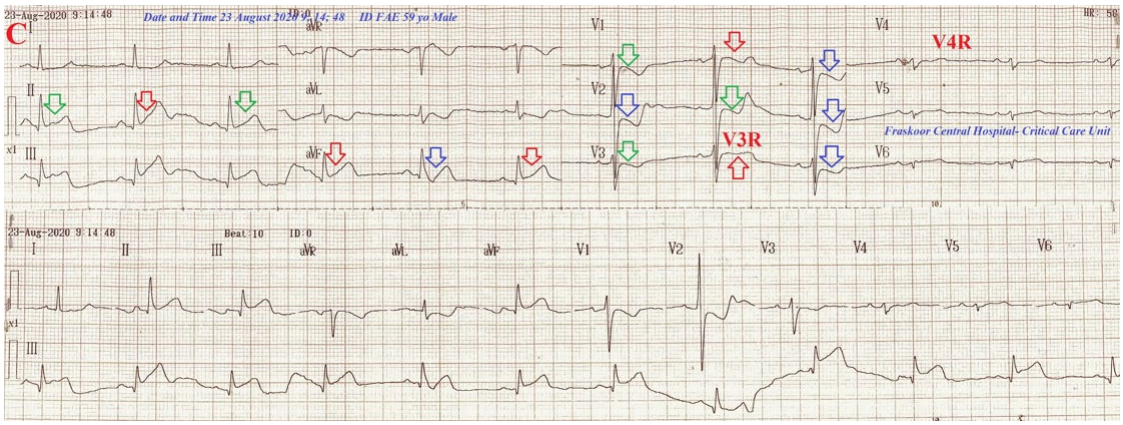



**Figure 1:** Serial ECG tracings were done A-tracing was done on the initial emergency presentation showing sinus bradycardia of VR; 57 bpm with mild ST-segment elevations (red arrows) with small q-waves in inferior leads (II, III, and aVF) and reciprocal ST-segment depressions in I, aVL, and V2-6 leads (lime and blue arrows). There is evidence of posterior MI in V2 and 3 (orange, green, and blue arrows). B-tracing was done on the ICU admission within 15 minutes of the above ECG tracing and just before given streptokinase showing sinus bradycardia of VR; 58 bpm with clear inferior ST-segment elevations (red arrows), pathological Q-waves in inferior leads (II, III, and aVF), and reciprocal ST-segment depressions in I, aVL, and V1-6 leads (lime and blue arrows). There is evidence of posterior MI in V2 and 3 (orange, green, and blue arrows).



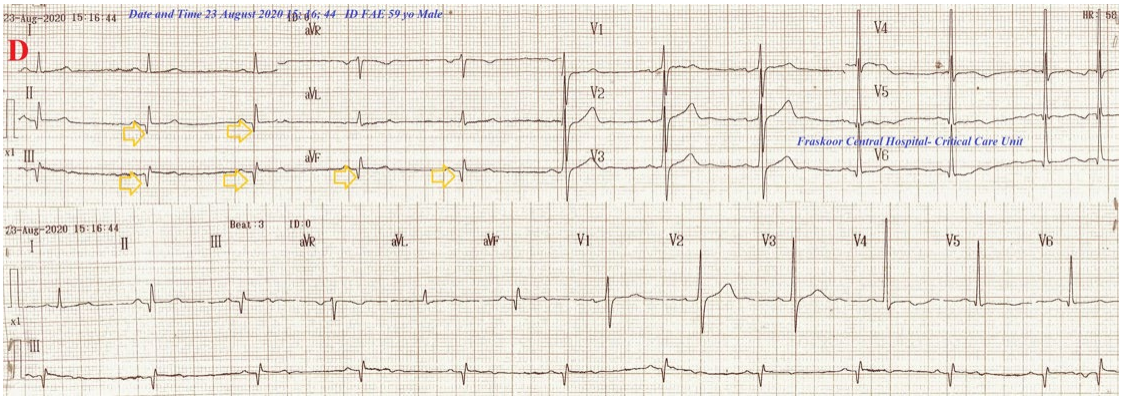



**Figure 1- C:** Tracing; a right-side ECG tracing was done within 15 minutes of the above ECG tracing showing there is no evidence of RV MI (V3R and V4R). There are both Wavy triple sign of hypocalcemia in V2 and V3R leads (Yasser's sign; red, green, and blue arrows) and Wavy double of hypocalcemia in II, aVF, and V2 leads (Yasser's sign; red with green, red with blue, and green with blue arrows). D. Tracing was done within 6 hours of the initial ECG tracing and within 6 hours of given streptokinase showing resolution of above ST-segment elevations and reciprocal ST-segment depressions. There are still pathological Q-waves (golden arrows) in inferior leads (II, III, and aVF) and snus bradycardia of VR; 58 bpm.

## 3. Discussion

• Overview: An elderly male farmer patient presented to the ED with acute severe agonizing chest wall and epigastric pain post-ingested combination of intermittent irregular oral diclofenac, ibuprofen, and celecoxib tablets.

• The primary objective of the current case study was the existence of acute inferior and possible associated posterior myocardial infarction after psychological stress over the use of multiple NSAIDs in an elder patient.

• The secondary objective of the case study is how to manage this patient in the ICU?

• The taken intermittent irregular doses of oral diclofenac, ibuprofen, and celecoxib tablets for more than one month with overlapping have a probability to cause acute STEMI. Potentiating or synergism for the three drugs is a possible interpretation.

• Avoidance of B-blockers due to the presence of bradycardia and vasodilators such as nitrates and captopril was done in the first 48 hours to avoid possible happening severe hypotension, shock, and death that can be happening with inferior STEMI.

• There are borderline sinus bradycardia is common with inferior MI.

• The absence of shock, heart block, and RV with inferior STEMI may be good prognostic findings.

• There is an excellent response for thrombolytic therapy.

• Coronary artery spasm is implicated in both pathogenesis and differential diagnosis.

• The only limitation of the current study was the unavailability of cable of posterior ECG leads (V7-9) for confirmation of posterior MI.

• I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

## 4. Conclusions

• Understanding drug pharmacokinetics and pharmacodynamics is a pivotal step for all clinicians.

• Oral non-steroidal anti-inflammatory drugs and psychosocial stress should be considered in dealing with all diseases.

• A dramatic response for thrombolytic therapy in the presence of both inferior ST-segment elevation and posterior myocardial infarction affection carries a good prognosis.

• Coronary artery spasms may be interpretative suggested mechanisms in inducing acute myocardial infarction.

## Conflicts of interest:

• There are no conflicts of interest.

## Acknowledgment

• I wish to thank the nurses team of the critical care unit who make extra-ECG copies for helping me. I want to thank my wife to save time and improving the conditions for supporting me.

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