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Case Report

Myocardial Infarction in Non-Obstructive Coronary Artery (MINOCA) in a Young Girl with Multidrug Poisoning

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Abstract

A 24 year young girl was admitted through the emergency room with chest pain, abdominal discomfort & sleeping tendency after taking some unprescribed medications. She had no risk factors for cardiovascular disease. On query she gives a history of taking multiple medications available at her room due to some emotional outbreak. Her relatives give a history of taking Paracetamol 1000 mg, Cephradine 2500 mg, Propranolol 100 mg, Bilastine 160 mg & Hydroxyzine 200 mg. None of these drugs are proven to cause Myocardial Infarction at this dosage. Initial ECG showed Sinus Bradycardia which can be explained by taking Propranolol. But 2 hours after admission she developed severe chest pain & at that time ECG showed gross ST depression. Troponin-i was found raised. After initial management Coronary Angiogram was done which revealed Normal Epicardial Coronaries. Patient was managed symptomatically & improved completely.

Keywords: *MINOCA, Young Girl,* Multidrug Poisoning

Introduction

The definition of MINOCA groups several conditions that share the absence of obstructive coronary arteries [1]. In MINOCA, despite angiographic evidence revealing normal or nearly normal coronary arteries (stenosis <50%), patients present with the similar clinicalsigns & symptoms of MI. Almost 1–14% of AMI occur in the absence of obstructive CAD [2-3]. As treatment modalities are different from Myocardial Infarction to MINOCA, proper understanding of the pathophysiology is crucial for appropriate evaluation and management. However, there is no previous literature based on Randomised Control Trial suggesting drug toxicity as a direct risk factor leads to the symptoms figure 1. In this case report, we found a young lady with no previous illness developed bradycardia followed by significant ST change in ECG along with raised troponin-I level after taking multiple drugs at nontoxic dose. A coronary angiography was carried out immediately, however no signs of obstruction

Were detected, hence a suspicion towards drug induced MINOCA was raised.

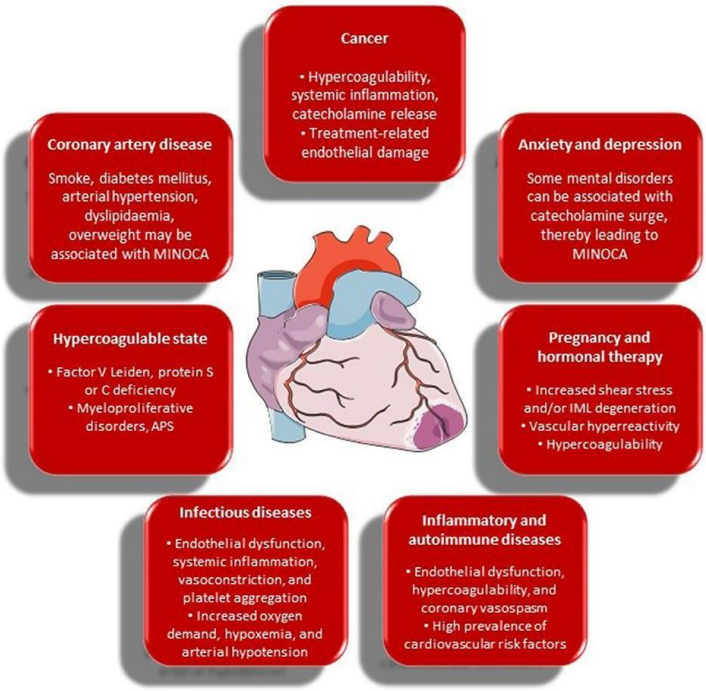

Figure 1. Risk factors and comorbidities of MINOCA [1].

Case Presentation

A 24 years young lady presented with the complaints of central chest pain, abdominal discomfort, and excessive sleeping tendency. Upon further inquiry, she reported ingesting multiple unprescribed medications in response to an emotional outbreak. She did not have any significant past medical illness and denied having any of the chronic medical conditions. She is not alcoholic and does not smoke. Further probing on psychiatric illness and recreational drug abuse were found insignificant. Upon initial examination, the patient appeared distressed and exhibited signs of discomfort. Vital signs revealed sinus bradycardia on cardiac monitor, which was attributed to the ingestion of Propranolol. However, two hours after admission, she developed severe chest pain, which resembled the characteristic of cardiac type chest pain.

Patient's initial ECG showed sinus bradycardia Figure 2a, likely secondary to Propranololingestion. However, the subsequent ECG after 2 hours exhibited gross ST-T changes in most leads, correlating with the onset of severe chest pain Figure 2b. Laboratory investigations revealed elevated levels of Troponin-i- 44.6 ng/mL, indicating myocardial damage. Routine blood investigations reveal nothing significant. A quick Echocardiography was arranged, but found no structural & functional abnormality. Coronary angiogram was performed to assess coronary artery patency, which surprisingly revealed normal epicardial coronaries, ruling out traditional ischemic etiologies Figure 3.

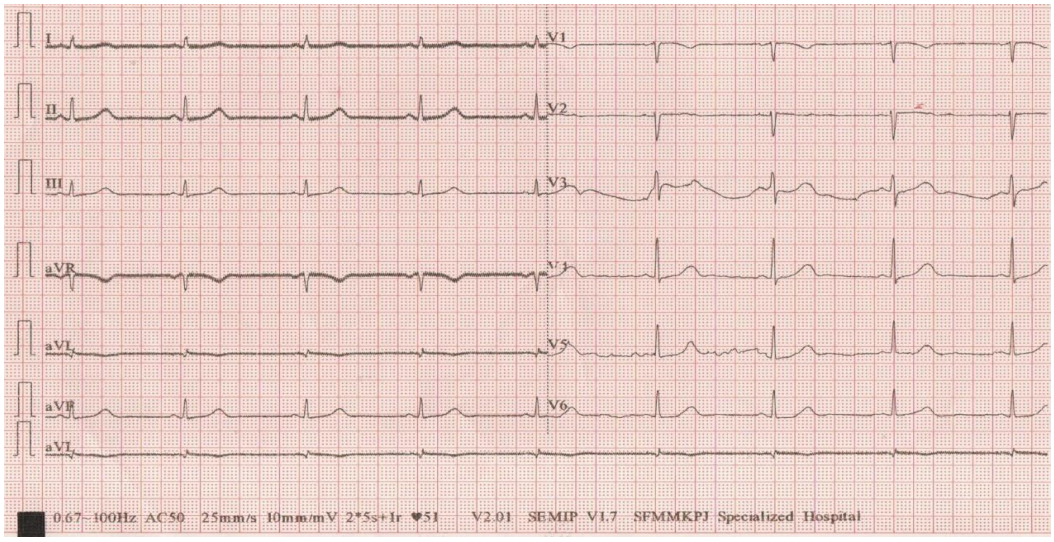

Figure 2a. ECG during initial assessment showing Sinus Bradycardia.

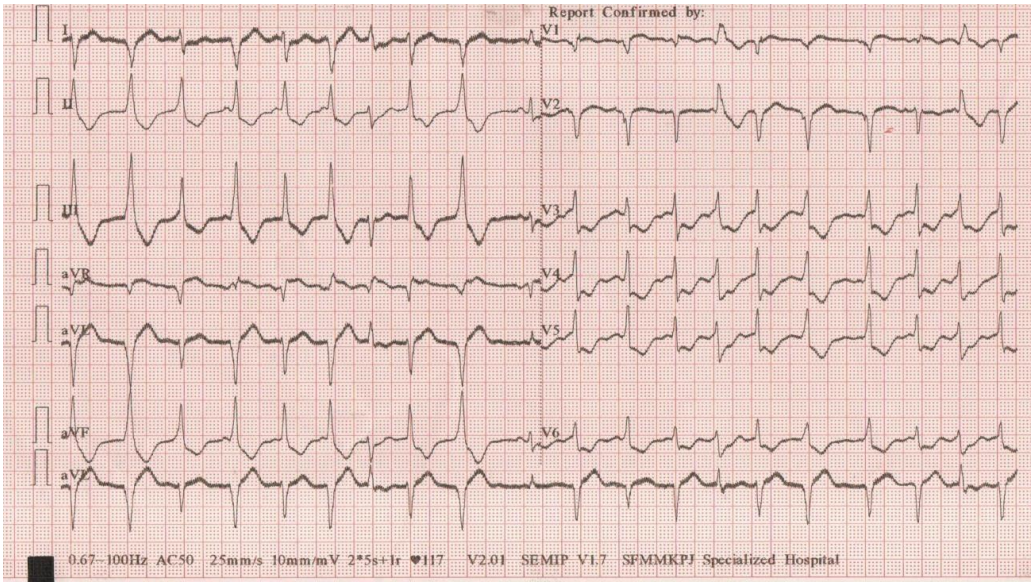

Figure 2b. ECG after 2 hours showing gross ST-T Changes.

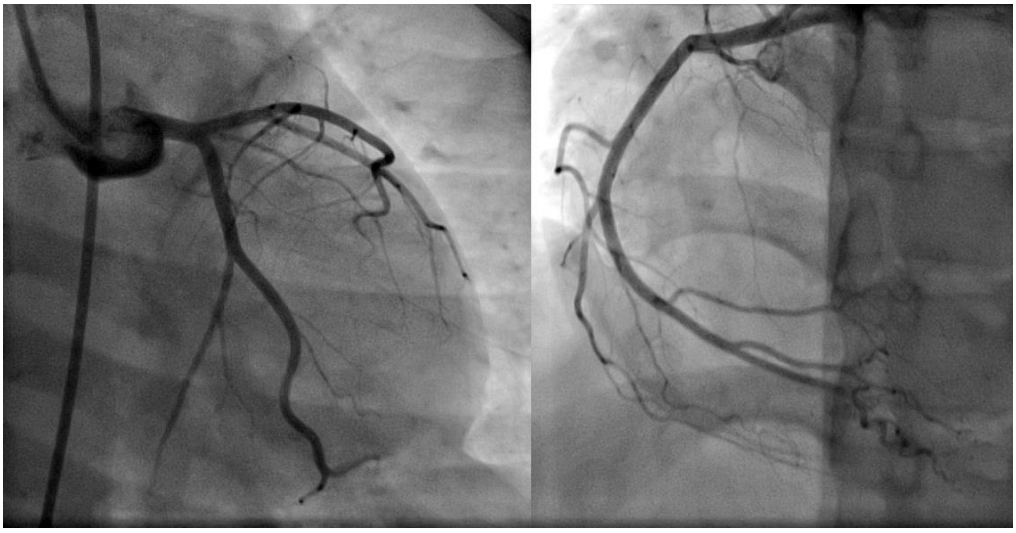

Figure 3. Coronary Angiogram reveals normal epicardial coronaries

The patient's symptoms and laboratory findings were consistent with myocardial infarction (MI) despite the absence of traditional risk factors and obstruction in coronary arteries. The etiology of MI in this case was attributed to the ingestion of multiple unprescribed medications, including Paracetamol 1000 mg, Cephradine 2500 mg, Propranolol 100 mg,Bilastine 160 mg, and Hydroxyzine 200 mg. Although none of these medications are typically associated with MI at these dosages, their combined ingestion likely led to myocardial damage.

The patient was managed symptomatically, with a focus on pain relief, hemodynamic stabilization, and cardiac monitoring. Given the absence of obstructive coronary disease, traditional reperfusion strategies such as thrombolysis or percutaneous

coronary intervention were deemed unnecessary. Instead, supportive measures such as fluid management, analgesia, antiemetic, anti-ulcers and close cardiac monitoring were instituted. With time, the patient's symptoms resolved completely, and she demonstrated clinical improvement. The patient was advised against self-medication and educated on the potential risks associated with unprescribed drug ingestion. Follow-up appointments were scheduled to monitor cardiac function and ensure ongoing adherence to prescribed medications and lifestyle modifications to mitigate cardiovascular risk factors. Additionally, psychiatric evaluation and counseling were recommended to address underlying emotional issues contributing to the patient's self-medication behavior. Her family members are also counseled.

Discussion

Here we have discussed a young female patient, who has been diagnosed as MINOCA following taking a few unprescribed medications those are not causing vasospasm, ulceration and obstruction in the epicardial coronary arteries. Despite having significant ST abnormalities in ECG and raised Trop-I level, Echocardiography and Coronary Angiographic findings were not suggestive of MI (Invasive provocative coronary vasospasm testing could not be performed due to the acuteness of the situation). Besides, the patient didn’t have any relevant past medical history which is related to hypercoagulable state and coronary artery disease. Therefore, multidrug poisoning was held responsible for her chest pain, ECG changes and raised troponin level. Moreover, she responded to symptomatic treatment and ECG and Troponin level settled on its ownwithout any intervention.

As per previous study and reports a few drugs are responsible for MI by causing epicardial vasospasm and toxicity to myocardiocytes. Some chemotherapeutics [4], antimigraine drugs (triptans) [5], antibiotics (amoxicillin with clavulanic acid, cefuroxime) [6,7] bromocriptine, β-blockers, pseudoephedrine [8], acetylsalicylic acid (ASA), steroidal and non-steroidal anti-inflammatory drugs [9] are attributed commonly and we know their pathophysiology. But here we did not find any signs of obstruction and signs of toxic myocarditis in CAG and Echocardiography, despite having all features of MI in this patient. So we believe this is a case of MINOCA and a thorough study is needed to find out the drug related risk factors and their pathophysiological background to facilitate the management approach and avoid irrational invasive procedures.

Conclusions

This case report underscores the significance of considering medication- induced myocardial infarction (MI) as a differential diagnosis, especially in young patients with atypical presentations following multidrug abuse. The absence of traditional cardiovascular risk factors and normal coronary arteries on angiogram posed diagnostic challenges, highlighting the importance of a comprehensive evaluation in

such cases. Recognizing medication-related cardiovascular complications is essential for appropriate management and prevention of adverse outcomes. The management of medication- induced MINOCA requires a multifaceted approach, including symptom relief, hemodynamic stabilization, psychological support, and patient education on the risks of self-medication. Further research is needed to elucidate the underlying mechanisms of medication-induced myocardial injury and to develop targeted interventions to mitigate cardiovascular risks associated with polypharmacy and self-medication.

Conflict of Interest: None.

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