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# A Case of Type 2 MI with Normal Coronary Arteries: Case Report & Literature Review

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**Abstract:**

***Clinicians have long recognized that acute myocardial infarction (MI) can occur in the absence of atherothrombosis . The Universal Definition of MI Global Taskforce introduced a classification system in 2007 (and reaffirmed in 2012) that defined type 2 MI (following standard diagnostic criteria) as MI occurring due to an imbalance in myocardial oxygen supply and/or demand not caused by atherosclerotic plaque disruption. Nevertheless, ambiguity remains regarding how to diagnose type 2 MI and how to distinguish it from both type 1 MI and myocardial injury.***

***Here we report a case of a 23 year old young woman attended to emergency department, with typical chest pain and shortness of breath for 6 hours, Diarrhoea for 2 days, and single time loss of consciousness for 5 minutes, 6 hours before attending to hospital. Cardiac enzymes were rising titres in subsequent samples, Serum Creatinine was also high. Echocardiography performed 36 hour later, showed no regional wall motion abnormality, coronary angiogram showed normal coronary arteries. So, a diagnosis of Myocardial Infarction (Type 2 MI) with Non Obstructive Coronary Artery (MINOCA) was made, and MINOCA was attributed to hypovolemic shock (resulting from Dirrrahoea), manifested as MI, Syncope and AKI.***

***Key Words: Type 2 MI, MINOCA, Hypovolemic Shock.***

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## Introduction:

The current (fourth) Universal Definition of MI Expert Consensus Document updates the definition of MI to accommodate the increased use of high-sensitivity cardiac troponin (hs-cTn). The criteria for **type 2 MI** includes detection of a rise and/or fall of cTn with at least one value above the 99th percentile and evidence of an imbalance between myocardial oxygen supply and demand unrelated to coronary thrombosis, requiring at least one of the following:

a. Symptoms of acute myocardial ischemia;

b. New ischemic ECG changes;

c. Development of pathological Q waves;

d. Imaging evidence of new loss of viable myocardium, or new regional wall motion abnormality in a pattern consistent with an ischemic etiology.[1]

Type 2 MI is considered as having "Many Faces". In the 2007 UDMI, CAD was required to diagnose type 2 MI,[3] whereas in the 2012 version, type 2 MI CAD was not required.[4]

In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anemia, respiratory failure, hypotension; the term 'MI type 2' is employed.

## Case Report:

A 23 Year old bangladeshi female, attended to emergency room with Chest pain and shortness of breath for 6 hours, Diarrhoea for 2 days, single time loss of consciousness (for 5 minutes) 6 hours back. The Chest pain was typical. Cardiac biomarkers were sent from ER (Emergency room), which came very high. Troponin I in first sample was "Above Assay"(Very high, above machine calculations); Second sample was 25000 pg/ml, Third sample was 8000 pg/ml. CK MB was 33 u/L in first sample, 56 u/L in second sample, and 36 u/L in third sample.

ECG done in ER showed, "T inversion in V1-V4".

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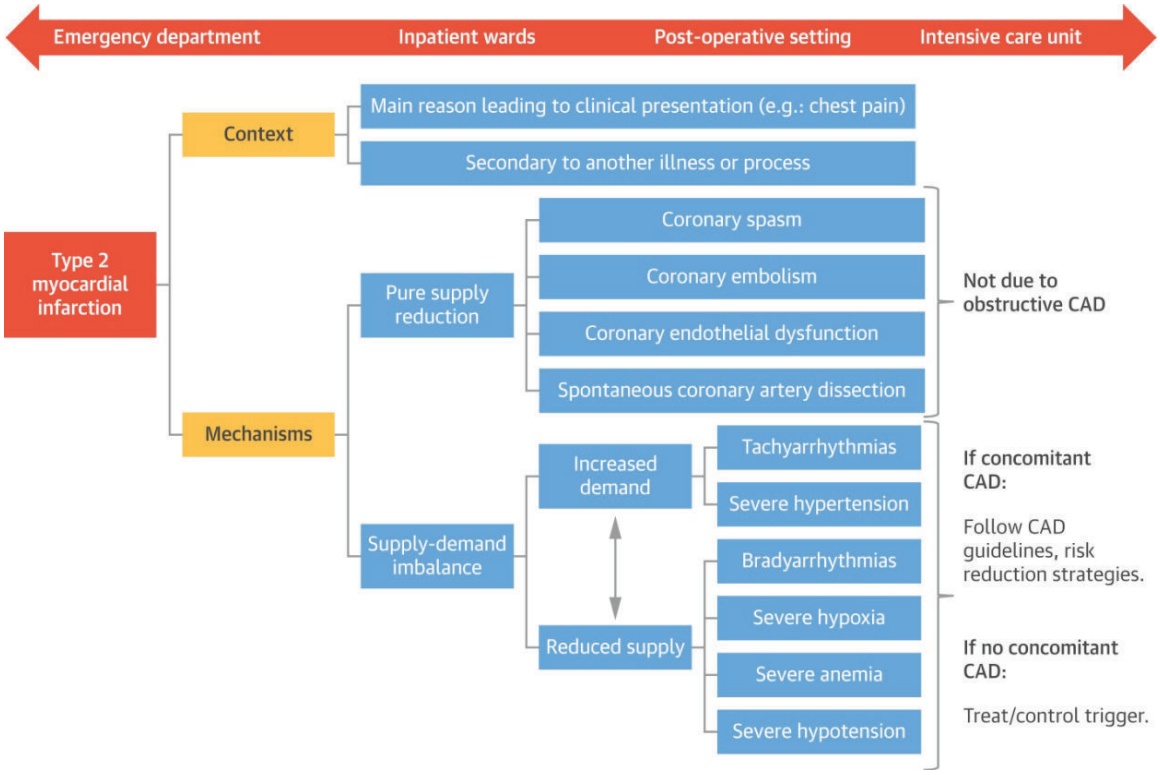


**Fig.-1:** *A Proposed framework by ACC for Type 2 MI*[2]

Color Doppler Echocardiography revealed, "No Regional Wall Motion Abnormality, EF(Ejection fraction) 59%, all chambers and valves are normal."

**Fig.-2:** *ECG: T inversion in V1-V4*

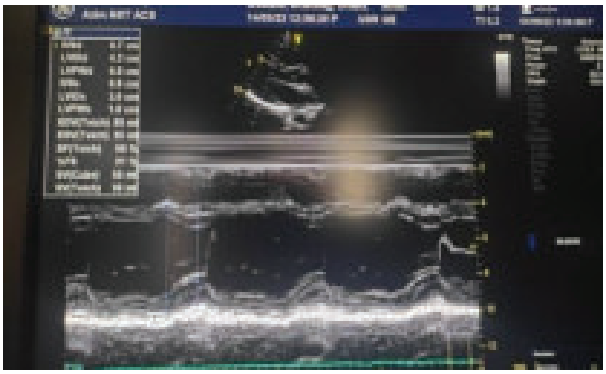

**Fig.-3:** *Echocardiography: No RWMA, EF 59%.*

CAG (Coronaryangiogram) revealed, "Normal epicardial coronary arteries"

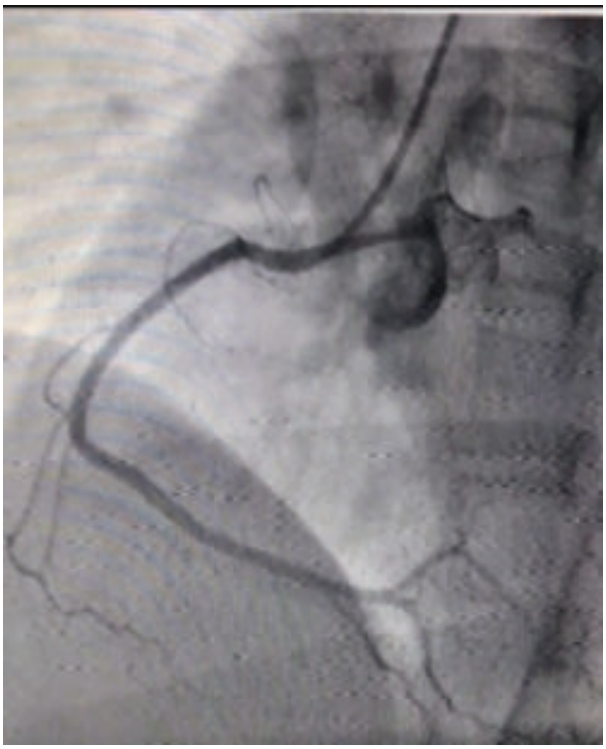

**Fig.-4:** *RCA: Normal & Disease free*

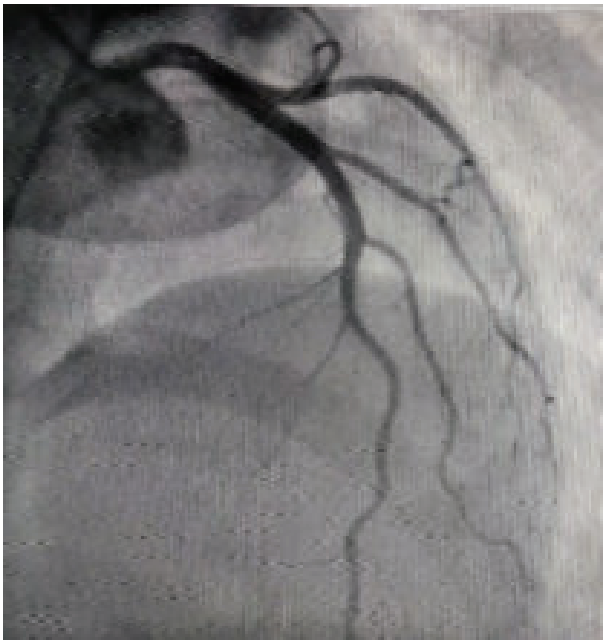

**Fig.-5:** *LAD & LCX: Normal & Disease free*

**Discussion and Literature review:**

Diagnosing type 2 MI requires fulfilling the following criteria[5]:

- An elevated but changing troponin value
- Clinical conditions known to increase the oxygen demand or decrease the oxygen supply like tachycardia
- Absence of symptoms and/or signs indicating other nonischemic causes of troponin elevations like myocarditis.
- Clinical features inconsistent with type 1 acute MI.

Though much attention is not given to this deadly disease, Type 2 MI occurs more frequently than type 1 MI, Type 2 MI is common in hospitalised patients, on average accounting for 10–20% of MIs. Type 2 MI patients may be asymptomatic, might have minimal, if any, ECG changes like, ST elevation, is more common in type 1 MI but can occur in type 2 MI in 5% of patients . The volume of involved cardiomyocytes is localised in type 1 MI to the territory supplied distal to the plaque event, whereas in type 2 MI we hypothesise that it may be a more global ischaemic phenomenon. The most important differentiator is the presence/absence of factors that may disturb the oxygen supply–demand balance. In the absence of any of these factors, type 2 MI cannot be diagnosed. More recently, the term MI with non-obstructive coronary arteries (MINOCA) has been used in the literature, including in an ESC position paper, referring to lesions with <50% stenosis. It is an exclusion diagnosis, encompassing type 2 MI, takotsubo syndrome etc

In a recent review, type 2 MI comprised 10.5% of MINOCA. The initial management should be to reverse the triggering factors. The well-established evidence base for anti-platelets and anticoagulants in type 1 MI has not been shown to be of benefit in type 2 MI.

Given that by definition a clinical diagnosis of type 2 MI means that the clinician believes there has not been an acute atherothrombotic event, we hypothesise that dual antiplatelets and anticoagulants are not likely to be beneficial. The role of invasive coronary angiography with or without PCI, as well as CT coronary angiography, is not well-defined in the type 2 MI population. Furthermore, IVUS and OCT have been little used.

Patients with T2MI have similar or higher all-cause mortality than patients with T1M1 .In part, because many studies include critically ill patients with comorbidities. They are at high risk for cardiovascular mortality and major adverse cardiovascular events. In the TRITON-TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel-Thrombolysis In Myocardial Infarction 38) trial, patients with T2MI had a nearly 3-fold increased risk for cardiovascular death[6]. In CASABLANCA, incident T2MI predicted all-cause and cardiovascular death, as well as the composite of all-cause death, nonfatal MI, heart failure (HF), stroke, transient ischemic attack, peripheral arterial complication, and cardiac arrhythmia.[7]

Conclusion:

The heterogenic nature of Type 2 Myocardial Infarction is a big challenge. Though type 1 myocardial infarction remains to be common, Type 2 myocardial infarction has not been given much importance. We highlight the need of vigilance and prompt diagnosis of type 2 myocardial infarction, to reduce the ongoing mortality of this deadly 'many face' disease.

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