International Journal of Innovative Research in Medical Science (IJIRMS)
Volume 07, Issue 06, June 2022,
https://doi.org/10.23958/ijirms/vol07-i06/1430

Mini review

Type II Myocardial Infarction: What Do We Know?

Muneera Altaweel [*1,2], Sarah AlMukhaylid [2,3], Faisal AlAnazi [1], Abdullah AlRammadan [1], Ibrahim Altuwaim [1]

[1]King Abdulaziz Hospital, MNGHA, Al Ahsa 36363, Saudi Arabia
[2]King Abdullah International Medical Research Center (KAIMRC), Al Ahsa 36363, Saudi Arabia
[3]College of Applied Medical Sciences (CoAMS-A), King Saud Bin Abdulaziz University for Health Sciences / KAIMRC/ KAH, National Guard Health Affairs, Al-Ahsa, Saudi Arabia

*Corresponding author: Dr. Muneera AlTaweel; *Monaziz55@yahoo.com*

Received 13 June 2022; Accepted 22 June 2022; Published 25 June 2022

Introduction

Type I myocardial infarction (T1MI) is caused by acute atherosclerotic plaque disruption; in contrast, type 2 myocardial infarction (T2MI) is caused by changes in myocardial oxygen supply or demand in the absence of acute atherothrombosis [1]. T2MI was initially introduced as part of the universal definition of MI (UDMI). It refers to individuals with an increased cardiac troponin (cTn) level but no evidence of ischemic etiology. Most doctors are baffled by the word, and the definition remains inadequate. It's not TIMI with evidence-based therapy and guidelines, but rather peer review based. Is chest discomfort accompanied by decompensated heart failure, acute anemia, and Pulmonary embolism indicative of myocardial ischemia? Is transitory ST depression a marker of myocardial ischemia in individuals with fast AF, anemia, sepsis, or electrolyte disturbances? The answer to all these questions is unknown or is currently being determined. On the other hand, is it prudent to do coronary angiograms for all patients? While the answer is still uncertain, the response is emphatical NO. Most doctors use clinical judgment.

Fourth Universal Definition of Myocardial Infarction

The Fourth Universal Definition of MI (UDMI) recognizes five types of MI (Table 1) [1]. The UDMI defines myocardial injury based on the elevation of cTn concentration >99th percentile upper reference limit derived from a standard reference population and classified MI categories using a pathophysiological method, which resulted in more overlapping etiologies (Figure 2) [1]. TIMI is a primary coronary arterial event attributable to atherothrombotic plaque rupture or erosion. Type 2 MI occurs secondary to an acute imbalance in myocardial oxygen supply and demand without atherothrombosis. This imbalance may be attributable to reduced myocardial perfusion in the context of fixed coronary atherosclerosis (without plaque disruption), coronary artery spasm, microvascular dysfunction, coronary embolism, dissection, or systemic causes such as hypoxemia, anemia, hypotension, or bradyarrhythmia, or increased myocardial oxygen demand attributable to tachyarrhythmia or severe hypertension. The UDMI also identifies MI types 3 to 5 in the setting of sudden cardiac death without circulating biomarker evaluation or related to revascularization procedures.

Classification	Definition
Acute MI	Clinical evidence of acute myocardial injury as evident from detection of a rise and/ or fall of cTn values with at least one value >99th percentile URL and at least one of the following symptoms of myocardial ischemia: Symptoms of acute myocardial ischemia New ischemic ECG changes Development of pathological Q waves Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology Identification of a coronary thrombus by angiography or autopsy (not for type 2 MI)
Type 1 MI	MI caused by atherothrombotic coronary artery disease and usually precipitated by atherosclerotic plaque disruption (rupture or erosion)
Type 2 MI	MI caused by a mismatch between oxygen supply and demand by a pathophysiological mechanism other than coronary atherothrombosis (type 1 MI)
Acute nonischemic myocardial injury	Acute myocardial injury (rise and fall in biomarkers [cTn]) in the absence of a primary ischemic cause (ie, absence of MI)
Chronic myocardial injury	Chronic myocardial injury (cTn >99th percentile URL without an acute change)

Table 1: fourth universal definition of myocardial infraction

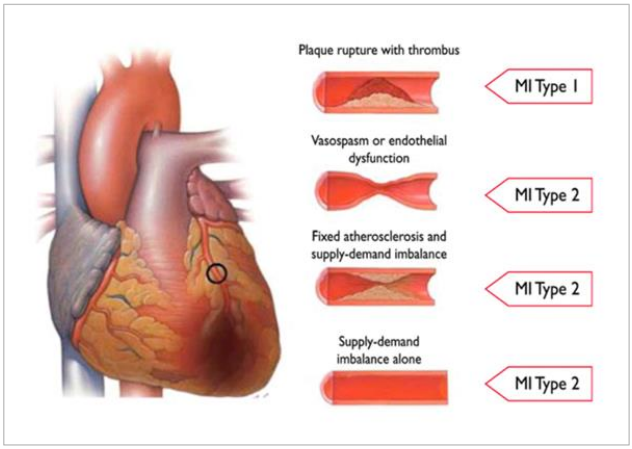

Figure 2: differentiating types of myocardial infraction (MI)

Type I Myocardial Infarction, Type II Myocardial Infarction, and Myocardial injury

T2MI is characterized by ischemia and another ailment, with or without coronary etiologies. T2MI is more prevalent in women (46%) than males (71%) and has an atypical presentation in women, the elderly, and diabetics, with less chest pain and more shortness of breath [2]. T2MI is currently on track with T1MI in terms of frequency. For patients with a rising and or falling cTn pattern, a diagnosis of acute MI is possible if ischemia is present [1]. The first steps include a careful history and physical examination, cTn measurements, and 12-lead ECGs, while imaging studies should be used selectively. cTn is highly specific for myocardial injury but does not differentiate between the etiologically [2,3]. T2MIs are often more minor events; there may not be ischemic electrocardiographic (ECG) findings or imaging abnormalities [3,4]. The ECG categorization of ST-segment elevation myocardial infarction (STEMI) and non-ST segment elevation myocardial infarction (NSTEMI) applies to T2MI as it does to T1MI. Among patients with acute MI with symptoms of myocardial ischemia, signs of ischemia on the ECG (ST-segment changes or the development of pathological Q waves), or evidence of a new regional wall motion abnormality, the diagnosis of acute MI is applied. STEMI occurs in 1% to 24% of T2MI patients, as determined by coronary imaging [5]. The more significant the ST-segment alteration is, the worse the consequences. Ischemic ECG and wall motion abnormalities are less standard in T2MI [3,4]. T2MI can occur with or without obstructive CAD and in patients with angiographically normal coronary arteries, such as spasm, embolism, endothelial dysfunction, or aortic dissection [6].

In the SWEDEHEART study, MI with nonobstructive coronary arteries occurred in 8% of MIs undergoing coronary angiography [7], of which 18% were T2MIs and 82% T1MIs. Among patients with T2MI who underwent coronary angiography, CAD was common. In the CASABLANCA research [8], where all patients with subsequent T2MI had angiography, nearly 60% had 50% obstruction in 2 vessels. In other studies, a bimodal distribution is observed with either no disease or severe CAD [9]. Nearly all investigations indicate that patients with T2MI manifest lower cTn values. Absolute concentrations and changes across serial measurements are greater with T1M1 than T2MI [10,11]. The CASABLANCA research involved 1251 individuals with coronary or peripheral angiography. T2MI patients reported a greater risk of future major adverse CV events (MACE) than those without T2MI after a median of 3.4 years of follow-up [8]. CASABLANCA acknowledges T2MI's clinical importance; this patient population is only now begun to be explored. Casablanca's T2MI rate was 12.2% using cTn, like the 7.1% reported in the Swedish sample hospitalized for acute MI. T2MI was more prevalent and recurrent than T1MI, despite aggressive medical therapy [8]. T2MI and myocardial damage can sometimes coexist; in the absence of ischemia, the diagnosis of acute myocardial injury is favored. In the TRITON-TIMI 38 trial, patients with T2MI had a nearly 3-fold increased risk for cardiovascular death [12]. In CASABLANCA, incident T2MI predicted all-cause and cardiovascular death and the composite of all-cause death, nonfatal MI, heart failure (HF), stroke, transient ischemic attack, peripheral arterial complication, and cardiac arrhythmia [8]. Studies focused only on patients with chest pain often exclude high-risk or critically ill patients, such as those with advanced renal disease, and suggest a more benign prognosis [12]. Raphael et al. identified individuals with T2MI and subsequent heart failure (HF) who required hospitalization in a big tertiary hospital. The majority of 359 (59%) had HF with preserved ejection fraction (HF-pEF), followed by HF with reduced ejection fraction (HF-rEF) 30%. Acute HF is the most prominent reason for hospitalization; 1/5 of T2MI patients will be readmitted for HF within a year [13]. After T2MI, strategies are needed to avoid HF [14]. Most patients with T2MI die from non-cardiovascular causes, as in studies of the critically ill where conjoint cardiovascular disease and critical illness increase death rates [18]. Nonetheless, studies with long-term follow-up indicate that cardiovascular mortality is common and explains 24% to 43% of deaths [8].

Conclusion

Type II myocardial infarction is caused by myocardial oxygen supply and demand mismatch without acute atherothrombotic plaque disruption. T2MI can potentially be hazardous with confounding short- and long-term prognoses. A noncoronary trigger usually causes a heterogeneous entity. Individualized therapies should be tailored to specific conditions for those with T2MI caused by supply-demand mismatch. Also, a consensus with regards to diagnosis is required. Imaging may help distinguish MI from injury.

Ethics approval and consent to participate:

Not applicable

List of abbreviations

Type 1 myocardial infarction (T1MI)
Type 2 myocardial infarction (T2MI)
Universal definition of Myocardial infarction (UDMI)
Cardiac troponin (cTn)
Electrocardiogram (ECG)
ST-segment elevation myocardial infarction (STEMI)
non-ST segment elevation myocardial infarction (NSTEMI)

References

[1] Thygesen K, Alpert JS, Jaffe AS, et al., "for the Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction". Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol 2018;72: 2231–64.

[2] Lippi G, Sanchis-Gomar F, Cervellin G. "Chest pain, dyspnea, and other symptoms in patients with type 1 and 2 myocardial infarction. A literature review". Int J Cardiol 2016;215:20–2.

[3] Sandoval Y, Smith SW, Sexter A, Schulz K, Apple FS. "Use objective evidence of myocardial ischemia to facilitate the diagnostic and prognostic distinction between type 2 myocardial infarction and myocardial injury". Eur Heart J Acute Cardiovasc Care 2018 Jul 1

[4] Smilowitz NR, Weiss MC, Mauricio R, et al. "Provoking conditions, management and outcomes of type 2 myocardial infarction and myocardial necrosis". Int J Cardiol 2016;218:196–201.

[5] Sandoval Y, Thygesen K. "Myocardial infarction type 2 and myocardial injury". Clin Chem 2017;63: 101–7.

[6] Ong P, Athanasiadis A, Hill S, etal. "Coronary artery spasm as a frequent cause of acute coronary syndrome: The CASPAR (Coronary Artery Spasm in Patients with Acute Coronary Syndrome) Study". J Am Coll Cardiol 2008;52:523–7.

[7] Baron T, Hambraeus K, Sundström J, et al. "for the TOTAL-AMI Study Group. Impact on long-term mortality of obstructive coronary artery disease and classification of myocardial infarction". Am J Med 2016; 129:398–406.

[8] Gaggin HK, Liu Y, Lyass A, et al. "Incident type 2 myocardial infarction in a cohort of patients undergoing

coronary or peripheral arterial angiography". Circulation 2017;135:116–27.

[9] Saaby L, Poulsen TS, Hosbond S, et al. "Classification of myocardial infarction: frequency and features of type 2 myocardial infarction". Am J Med 2013;126:789–97.

[10] Greenslade JH, Adikari T, Mueller C, et al. "Characteristics and occurrence of type 2 myocardial infarction in emergency department patients: a prospective study". Emerg Med J 2018;35: 169–75.

[11] Nestelberger T, Boeddinghaus J, Badertscher P, et al. "Effect of definition on inci- dence and prognosis of type 2 myocardial infarc- tion". J Am Coll Cardiol 2017;70:1558–68.

[12] Neumann JT, Sorensen NA, Rubsamen N, et al. "Discrimination of patients with type 2 myocardial infarction". Eur Heart J 2017;38:3514–20.

[13] Raphael, C. E., Roger, V. L., Sandoval, et al. "Incidence, Trends, and Outcomes of Type 2 Myocardial Infarction in a Community Cohort". Circulation 2020;141(6), 454–463.

[14] McCarthy CP, Jones-O'Connor M, Olshan DS, et al. "The Intersection of Type 2 Myocardial Infarction and Heart Failure". J Am Heart Assoc. 2021;10(17):e020849.

[15] Babuin L, Vasile VC, Rio Perez JA, et al. "Elevated cardiac troponin is an independent risk factor for short- and long-term mortality in medical intensive care unit patients". Crit Care Med 2008;36:759–65.