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Editorial
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Type II Myocardial Infarction: A Critical Appraisal

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Editorial

The paramount importance of acute coronary thrombosis in the pathogenesis of acute Myocardial Infarction (MI) was clearly established in the 1980's through landmark pathologic and angiographic studies [1-3]. Thrombus formation on a ruptured or eroded atherosclerotic plaque was shown to cause nearly all ST elevation myocardial infarctions and the majority of non ST elevation myocardial infarctions. However, other than ECG criteria and symptoms, our laboratory diagnosis was rather crude and insensitive based mainly on creatinine kinase and its subfractions. With the development of troponin I and T assays, it became apparent that many situations caused an elevation of troponin without symptoms suggestive of acute MI. As the assays became even more sensitive, troponin elevation increased and in many hospitals such as ours, the number of consults tripled for a diagnosis of "Tropinemia".

In 2007, the first Universal Definition of myocardial infarction was published in an attempt to accurately define a MI [4]. This important document divided MI into five categories. We will concentrate on the definition and diagnosis of a Type II MI. There have been 3 subsequent publications of the Universal Definition of myocardial infarction since 2007. All define a type II myocardial infarction similarly. In general, a diagnosis of myocardial infarction requires a rise and/or fall in troponin with at least one value greater than the upper reference limit and clinical criteria either symptoms of acute myocardial ischemia, typical new ECG changes, or new wall motion abnormalities consistent with an ischemic etiology. In order to qualify as a type II myocardial infarction, there must also be an oxygen supply/demand imbalance with or without the presence of significant Coronary Disease (CAD). If angiography is performed, there should be no culprit lesion [5,6]. This distinguishes it from a Type I MI which is caused by thrombus formation on a culprit, ruptured or eroded plaque in an epicardial coronary artery.

According to the Universal Definition, Type II MI is caused by any process that decreases perfusion or increases oxygen demand. This would include some of the following scenarios. A reduced supply might be related to fixed coronary atherosclerosis without plaque rupture, or other epicardial reductions in supply such as coronary artery spasm, coronary artery dissection with or without intramural hematoma or coronary embolization. Other mechanisms that reduce oxygen supply might include severe bradyarrhythmias, respiratory failure with severe hypoxemia, severe anemia, hypotension or shock. Anything that significantly increases myocardial oxygen demand such as sustained tachyarrhythmias, severe hypertension with or without Left Ventricular Hypertrophy (LVH) might also potential mechanisms. Although not specifically emphasized, these supply/demand changes should be acute in most instances. For instance, a patient with severe chronic anemia would have a different hemodynamic and ischemic response than a patient who acutely drops the hemoglobin significantly due to an acute bleed. According to the definition, ischemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease [5]. So, what are our concerns?

The inclusion of an epicardial process such as coronary dissection, large vessel spasm or coronary embolization in the definition of a Type II MI: While these processes are examples of a supply/demand mismatch, aren`t all MIs (including Type I) supply/demand mismatches? The inclusion of epicardial and non epicardial causes is confusing and overlaps with the definition of a Type I MI. This is more than semantics as treatment strategies would likely differ in the presence of one of these epicardial causes. In general, the treatment of a Type II MI includes supportive measures such as volume adjustment, transfusions, and treatment of tachycardia, bradycardia, hypertension or hypotension [5]. The causes that only involve the epicardial coronary arteries as mentioned above would require different therapies including antiplatelet agents and possibly either anticoagulants, beta blockers, calcium channel antagonists or emergent angiography and percutaneous coronary intervention depending on the etiology.

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The definition of a Type II MI does not specify the degree of the supply/demand imbalance: While ischemic thresholds are not fixed and depend on the substrate of the patient (presence of CAD, LVH, reduced ejection fraction or valvular dysfunction), it is essential to include some parameters to guide the physician in diagnosis. It is not uncommon for patients seen in the emergency room with chest pain, anxiety or dyspnea to have an increased blood pressure or pulse rate even if no MI is present. Would this qualify as a supply/demand mismatch? The problem is the interpretation of the definition. The incidence of Type II MI in the literature varies by nearly 45 fold (1.6% to 71.2%) [7]. In a prospective study our group performed 10 years ago, the incidence of Type II MI was 29.6% [8]. Saaby et al., [9] utilizing specific supply/demand parameters for diagnosis found a similar incidence of Type II MI (26%). If a diagnosis has such a varied incidence, the criteria for its diagnosis must be too subjective.

The rise and or fall of troponin needed for MI diagnosis should be more specific: This comment is not necessarily applicable to only Type II MI but to the general diagnosis of MI in the Universal Definition. For years, our hospital has used a sensitive but not an ultrasensitive Tn I assay (ADVIA Tn I-Ultra with a 99th percentile of 0.04 ng/dL).

Should the rise and or fall depend on the baseline value? If a patient comes into our hospital with a value of 0.04 ng/dl which then increases to a peak of 0.05 ng/dl, would this be enough of a rise and/or fall to be consistent with an MI diagnosis or acute myocardial injury (depending if clinical criteria are present)? What about a baseline value of 1.0 ng/dl increasing to a peak of 1.05 ng/dl? Again, we believe that parameters should be specified for diagnosis and a percentage change possibly dependent on the baseline value be instituted. Presently, in the Universal Definition, only the definition of an MI after percutaneous coronary interventions (type 4a) states that the post procedure Tn should increase by >20%. While the sensitivity of the assay would be a critical factor, not every hospital utilizes the most ultrasensitive assay.

The definition of ischemia: In addition to troponin, Type II MI must have evidence of ischemia; otherwise, it is not classified as an MI. With a nearly 45 fold difference in the incidence of Type II MI, it is unlikely there is consistency in the literature in its definition. According to the Universal Definition of MI, ischemia is defined as typical but also atypical symptoms such as dyspnea or fatigue. It is well known that typical presentations are more specific for ischemia than atypical ones. If atypical presentations predominated in some studies as the only manifestation of ischemia, the incidence of Type II MI would be high. In this situation, heart failure exacerbations with dyspnea as the only ischemic manifestation with a little tachycardia and an increase in troponin might all be considered as infarctions. As previously mentioned, consistency in the incidence of MI across studies requires a greater uniformity of diagnostic criteria [10,11].

Conclusion

The present definition of Type II MI, based on clinical judgment by the clinician, is vague and subjective and largely responsible for the extremely wide incidence reported in the literature. While a Type II has been shown to increase short and long term mortality, who are the patients with the higher mortality? Greater consistency and specificity in the diagnosis of Type II MI are needed. Otherwise, the diagnosis may not be credible and comparisons of the incidence of Type II MI in the literature are useless.

Recently, an ICD code for Type II MI has been introduced and Type II MI has been included in the Hospital Readmission Reduction Program. It behooves us to get this diagnosis right! We suggest the elimination of epicardial etiologies (should be reclassified as Type I MI) and the development of specific parameters for the degree of supply/demand mismatch, ischemia and the delta change in troponin values. Clearly, more studies in the future are needed to develop evidence based guidelines for diagnosis and treatment.

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