CDI and Type 2 Myocardial Infarction

Deidre Barnett BSN RN CCDS

MedPartners HIM

OBJECTIVES

1. Understand the Clinical Documentation Improvement Specialists' responsibility to increase knowledge of all clinical conditions that reflect the most accurate SOI of the patient.
2. Understand the pathophysiology of type 2 MI.
2. Differentiate non ischemic vs. ischemic positive troponin.
3. Identify clinical terms and indicators for type 2 MI.
4. Provide an algorithm for decision making.

CDIS Role and Responsibility

Clinical Documentation Improvement (CDI) has become a very important part of healthcare today. Organizations rely on CDI to help meet the Centers for Medicare and Medicaid Services (CMS) coding guidelines. According to the ACDIS position paper outlining the CDIS roles and responsibilities, "CDIS must apply a broad clinical and coding knowledge base to discern relevant clinical conditions" (ACDIS Advisory Board, 2014). Nurse Clinical Documentation Improvement Specialists (CDIS) understand the clinical documentation with clear clinical indicators for many different diagnoses along with the treatments for these conditions. This knowledge allows the CDIS to help bridge the gap between physician documentation and what Health Information Management (HIM) coders are allowed to code based on that documentation (AHIMA, 2008). Most CDI programs start by identifying comorbid conditions that reflect a more accurate picture of the severity of illness (SOI) and risk of mortality of the patient. The benefits of this accurate picture are reflected in reimbursement, quality reporting, and physician profiling.

CDIS must seek to increase their knowledge of clinical conditions that reflect the SOI of the patient. Armed with this knowledge, the CDIS can formulate appropriate compliant queries to help the physician increase the accuracy of the record. A compliant query must include appropriate clinical indicators and treatment of the condition (AHIMA, 2008a).

Type 2 MI presents an opportunity to capture a major comorbid condition that more accurately describes a patient's SOI. Since the cause of Type 2 MI is not the same as Type 1 MI, CDIS must be able to recognize the clinical indicators and treatment plan that are unique to Type 2 MI. The Third Universal Definition of Myocardial Infarction was published in the American Journal of Cardiology in October of 2012 to further clarify the definitions of Myocardial Infarction with the ability to use the more sensitive biomarker of troponin to identify early cell death (Thygesen, et al., 2012). It further discusses the Type 2 MI that develops in patients with multiple comorbid conditions and critical illness. The Methodist Debakey Cardiovascular Journal published an algorithm that can be used as a handy guide to distinguish ischemic vs. non ischemic troponin elevations (Jneid, Alam, Virani, & Bozkurt, 2013). Specialists can now use this information to recognize this condition in seriously ill patients and query the physician appropriately if needed. Thus, the CDIS fulfills his/her responsibility and the organization's goal to accurately reflect the patient's severity of illness, the use of resources, and the quality of care provided.

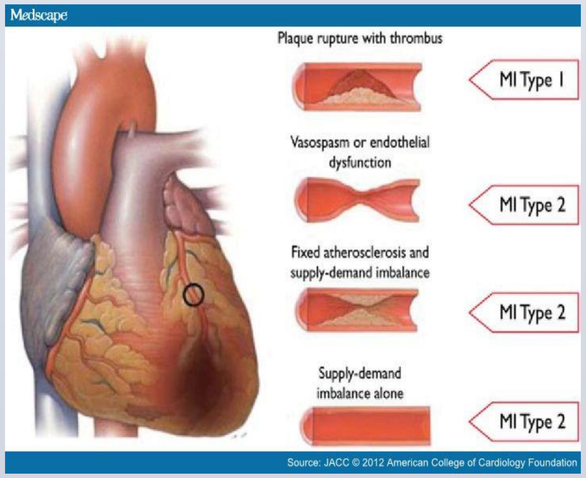
Pathophysiology of Type 2 MI

Onset of myocardial ischemia is the initial step in the development of MI and results from an imbalance between oxygen supply and demand.

Myocardial Infarction Secondary to an Ischemic Imbalance

(MI Type 2)

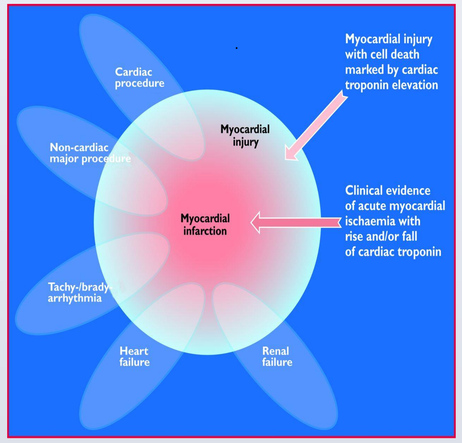
In instances of myocardial injury with necrosis, where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, the term 'MI type 2' is employed. In critically ill patients, or in patients undergoing major (non-cardiac) surgery, elevated values of cardiac biomarkers may appear, due to the direct toxic effects of endogenous or exogenous high circulating catecholamine levels. Also coronary vasospasm and/or endothelial dysfunction have the potential to cause MI (Thygesen, et al., 2012).



Clinical Indicators and Treatment

- **A cTn level >99th percentile of the URL is considered elevated and is the cut-off level for a diagnosis of MI.**
- It is important to distinguish acute causes of cTn elevation, **which require a rise and/or fall of cTn values**, from chronic elevations that tend not to change acutely.

This illustration shows various clinical entities: for example, renal failure, heart failure, tachy- or brad arrhythmia, cardiac or non-cardiac procedures that can be associated with myocardial injury with cell death marked by cardiac troponin elevation.



Thygesen K et al. *Circulation*. 2012;126:2020-2035

Signs of Myocardial ischemia:

- History of CAD
- History of MI
- EKG changes
  - new LBBB
  - presence of Q waves
- Pain in the chest, upper extremities, mandible, or epigastric region
- Dyspnea and fatigue

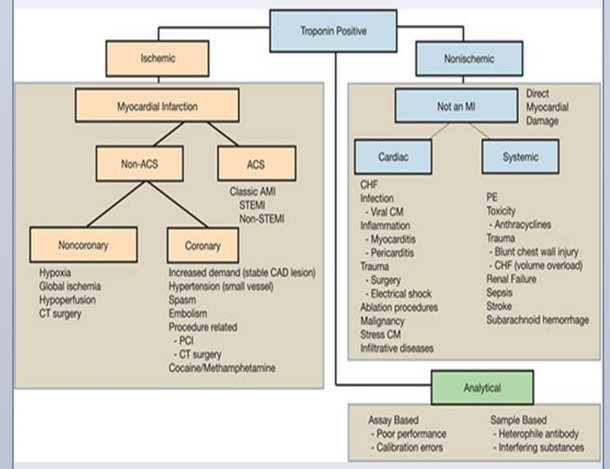
*However, there may be no symptoms at all (other than elevated troponin levels) in women, the elderly, diabetics, or post-operative and critically ill patients.

Treatment of Type 2 MI

Treatment of Type 2 MI is based on treatment of the underlying cause of the ischemia e.g. hypoxia, hypovolemia/hemorrhage, hypertension. Therefore typical treatments for AMI (Type 1) may not apply.

When to QUERY

The table below is a useful tool to help distinguish between ischemic and non-ischemic reasons for elevated troponin levels.



Conceptual model for clinical distribution of elevated troponin. Adapted from Newby et al.6 ACS: acute coronary syndrome; AMI: acute myocardial infarction; CAD: coronary artery disease; CHF: congestive heart failure; CM: cardiomyopathy; CT: cardiothoracic; PCI: percutaneous coronary intervention; PE: pulmonary embolism; STEMI: ST-segment elevation myocardial infarction.

In addition to the clinical indicators and underlying etiologies discussed, look for these buzz words for Non ACS myocardial infarction:

- Supply demand mismatch
- Demand ischemia
- Coronary spasm
- Hypoxic ischemia
- Cocaine or methamphetamine use

References

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