

NEW DEFINITION OF MI: INDIAN PERSPECTIVES

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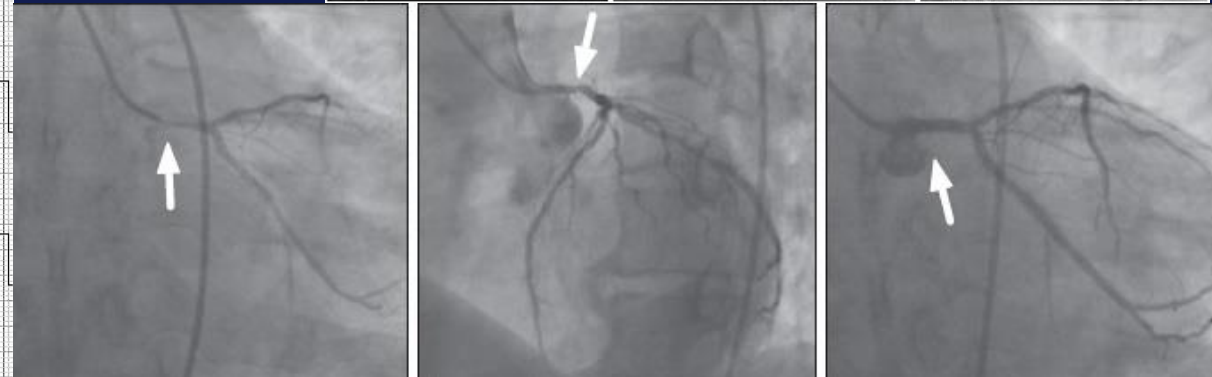
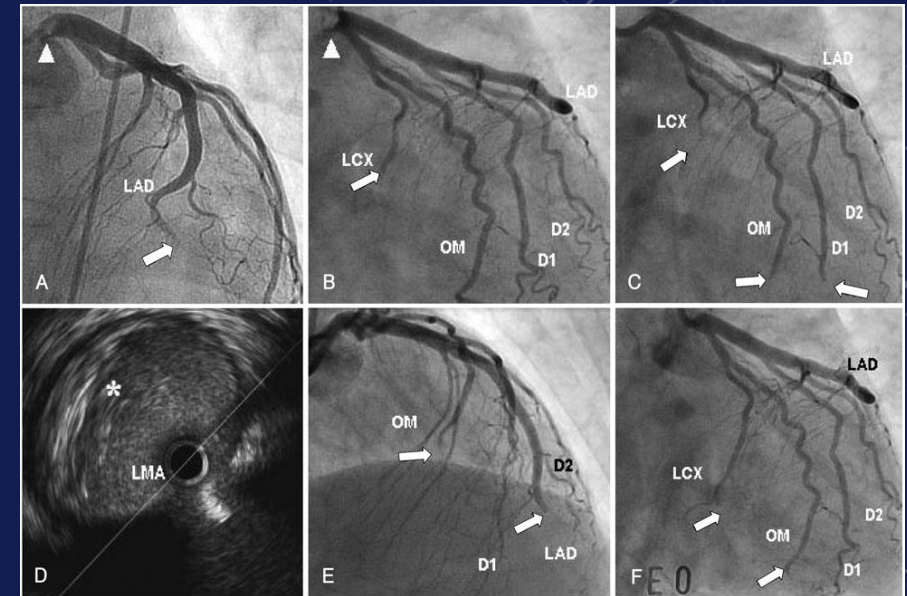
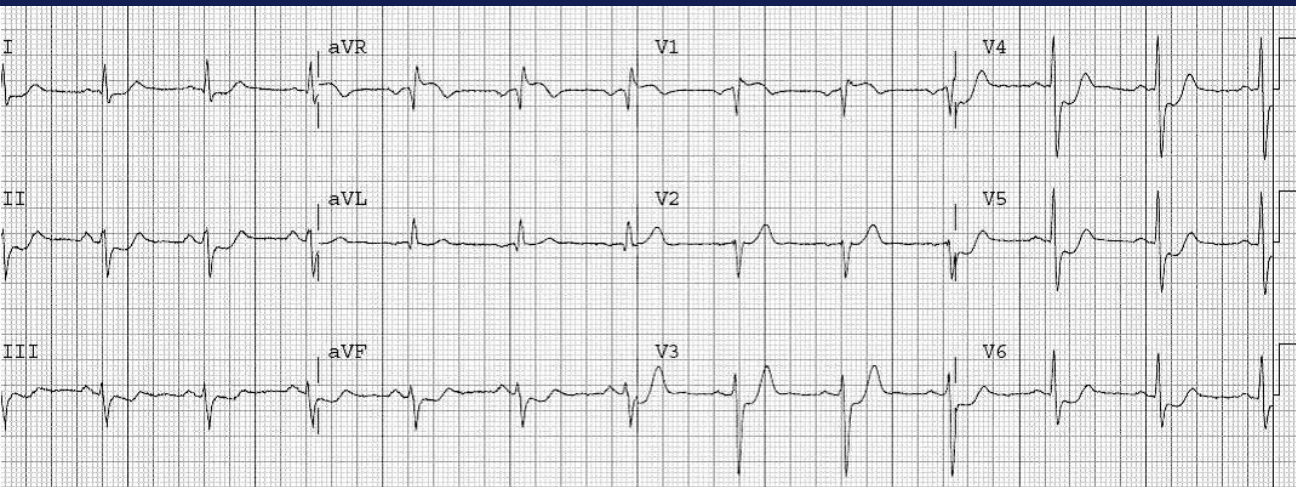
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43 yrs male smoker hypertensive non-diabetic dyslipidemic with family history of CAD and PTCA presented with acute chest pain with compressive feeling in chest and reached the PCI-able Cardiac Centre with in 120 minutes.

Clinical Examination did not reveal any abnormality.

ECG as below:



Trop-T +ve

Echo: Hypokinesia of the anterior wall with reduced diastolic compliance and LVEF 40%
Patient was given 180 mg Ticagrelor and ½ tablet of soluble Aspirin 325 mg

- **What are new?**
- **Cardiac Troponins**
- **Definitions**
- **Implications**
- **Conclusion & Take Home Message**

New Concept : Differentiation Of Myocardial Infarction From Myocardial INJURY

All Myocardial Infarctions are Myocardial Injuries
All Myocardial Injuries are not Myocardial Infarctions

$H_2O \approx cTn$

cTn: Cardiac Troponin → Gold Standard

Issues on Cardiac Troponin

99th percentile is the cut off point

So many variations: cTn-T , cTn-I, hscTn-T, hscTnI

Assays are not standardised. Values are different for different Labs

The 99th percentile of the overall population was 27 ng/L. Age and gender had a prominent influence on these values. **Tanza Zeller et al. Clin Chem Lab Med ,2014**

Concordance for paired cTnI and hs-cTnI measurements(n=1096)was verified using 99th percentiles for both genders (cTnI:30ng/L, hs-cTnI:25ng/L) and for men and women separately (hs-cTnI: M:34; F:16ng/L). **G.R Lee et al. Practical Laboratory Medicine 4 : 62–75,2016**

Controversies have been raised: Thomas Nestelberger et al : study published in the September 26, 2017, issue of the *Journal of the American College of Cardiology*..

In absence of accurate algorithm, adequate diagnostic accuracy using **clinical protocols and** currently established 99th percentile (gender-specific) and change (e.g. guideline-endorsed) criteria appears plausible

Relevance of cTn in Clinical Practice: Indian Perspective

Though cTn widely available in the Indian Metro cities, but not in the sub-metros. Misses the early birds.

CK-MB on the other hand is available universally . But again CK-MB miss late presnters.

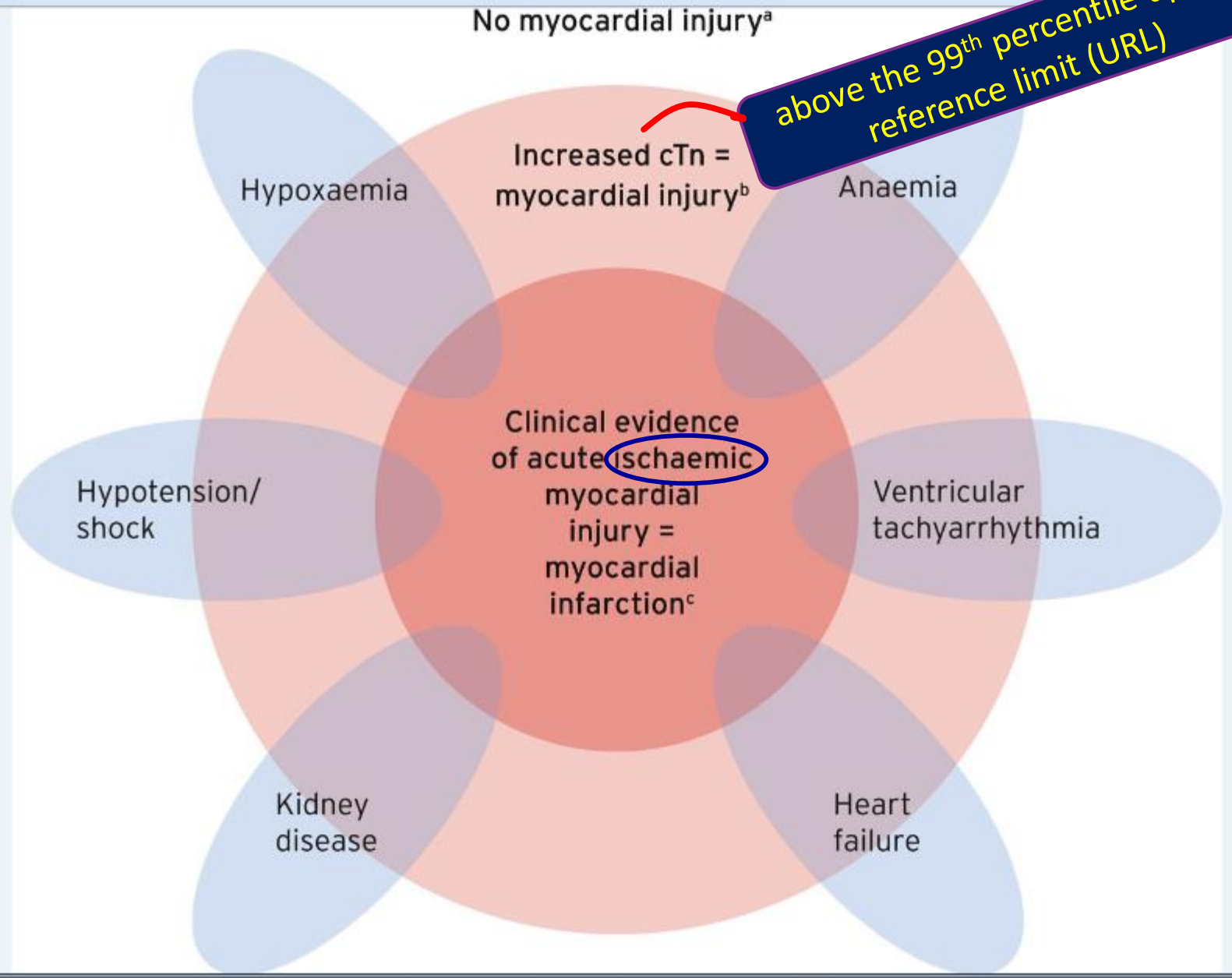
Repeated tests specially the pre-procedure analysis of cTn will be an extra-vaganza in India. Thus Type 4 MI will be always missed or mis-interpreted.

Most hospitals do not practice this. No body will like the diagnosis: Post-procedure MI

SCAI definition

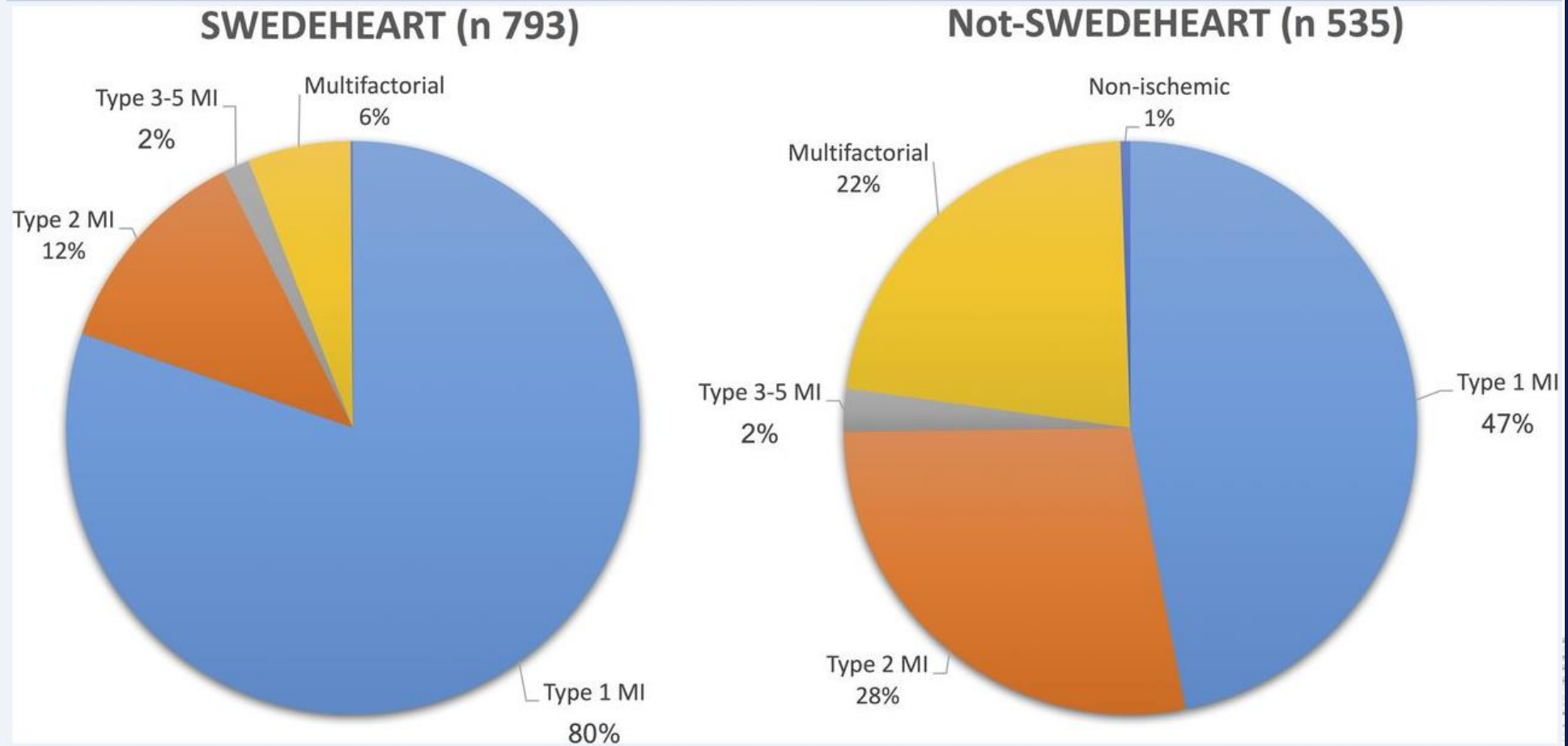
The definition proposed by the Society for Cardiovascular Angiography and Interventions (SCAI) relies on a >10 times increase of creatinine kinase (CK)–MB (5 times in the presence of new Q waves on electrocardiography) and aims to capture “clinically relevant” MIs, without specific focus on sensitivity (7).

However they have not overlooked the importance of cTn-based definition

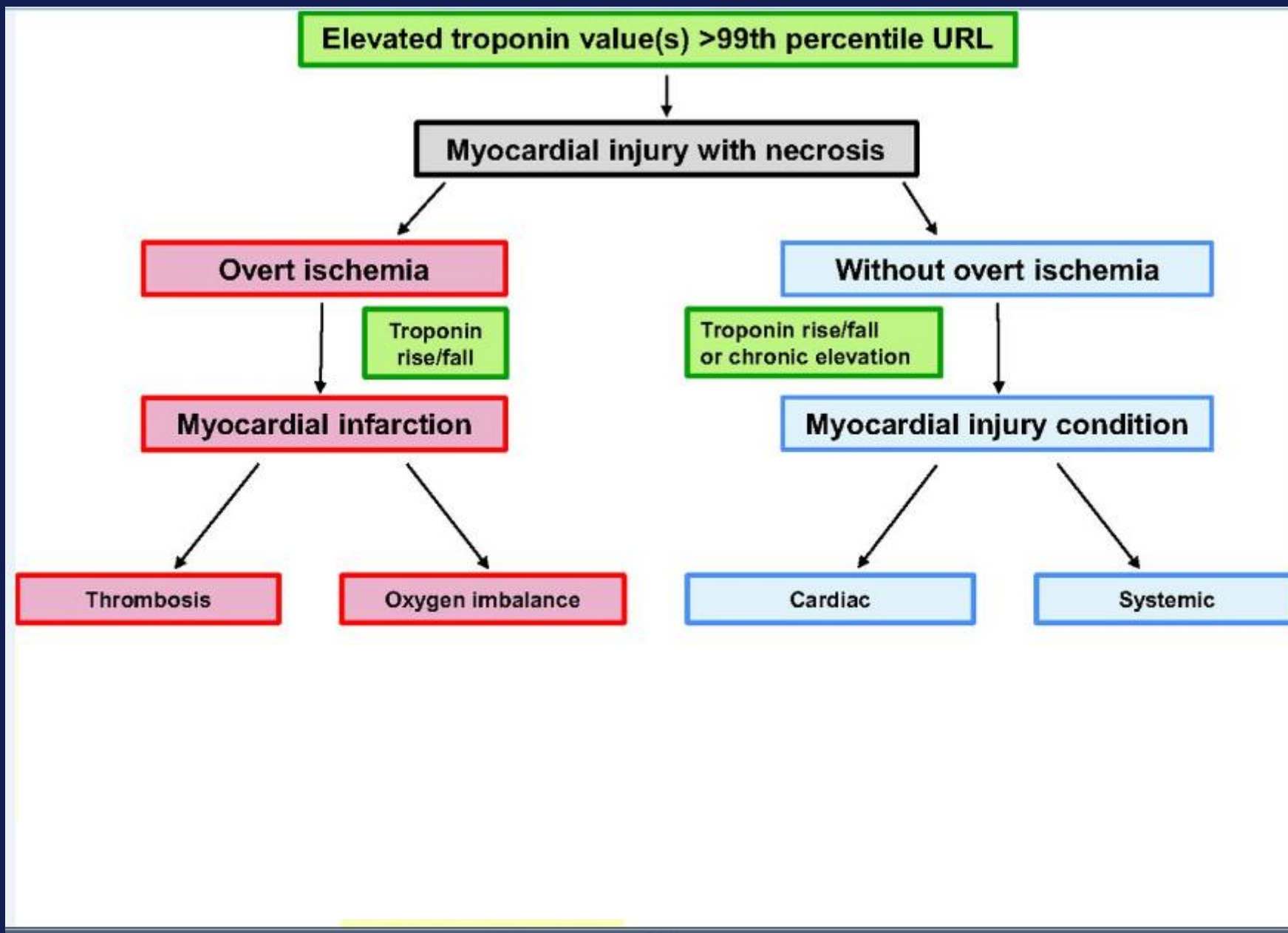


above the 99th percentile upper reference limit (URL)

Consequence of application of New Definition



Dramatically Increased the Diagnosis of MI



What Are Other New Aspects?

Differentiating MI from myocardial injury, electrical remodeling and arrhythmias, and the use of imaging as a Dx tool. ✓

Role of high-sensitivity cardiac troponin assays ✓

Imaging including echocardiography, radionuclide imaging, CMR and CTC angiography in the diagnosis of acute MI : to detect RWMA or loss of viable myocardium.

New sections on TTS, MI with non-obstructive coronary arteries (MINOCA), CKD, Afib, regulatory perspectives on MI and silent or unrecognized MI ✓

integration of all data viewed in the context of the time horizon over which the suspected event unfolds.

Type 1: Occlusive/Non-occlusive by plaque rupture/erosion

Type 2: Stressor-induced myocardial O₂ demand-supply mismatch in CAD with out plaque rupture

Type 3: Post-death where suspicion is high on Ath. CAD

Type 4: Associated with PCI



Type 5: Associated with CABG

Enhanced Role of Various Modalities of Investigations

Highlighting **ECG** as an immense diagnostic parameter:

1. Consideration of new no-rate-related BBB with specific repolarisation pattern
2. ST-Elevation in aVR with specific repolarisation pattern as STEMI-equivalent
3. ECG detection of myocardial ischaemia in patients with ICDs, PPIs
4. Consideration of electrical remodeling (cardiac memory) in assessing repolarisation abnormalities with tachyarrhythmias, pacing and rate-related BBB

1. Enhanced role of **Cardiac MR** to define myocardial Injury

2. Enhanced role of **CT Coronary Angiography** in suspected MI

Imaging Modalities

- Echocardiography-Defining new RWMA when >20% Transmural thickness is affected
- Radionuclide Imaging: Viable Myocytes to be Imaged Directly
 - A. SPECT Tracers ^{201}Tl chloride, $^{99\text{m}}\text{Tc}$ sestamibi
 - B. PET Tracers ^{18}F FDG & ^{82}Rb ^{173}Lu
 - C. 4% of Myocardium= 5-10 gram of muscles

Cardiac MRI

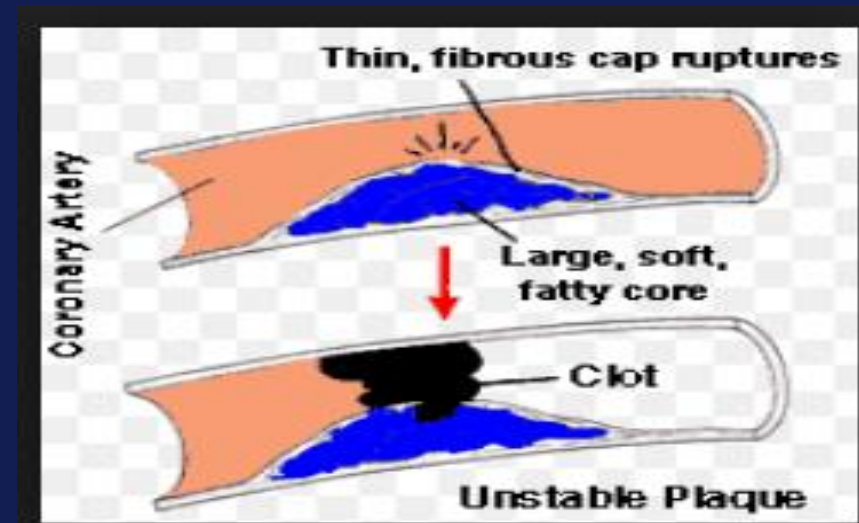
Prior or Silent or Un-recognised MI:

- 1. Abnormal Q-waves with or without symptoms in the absence of non-
ischaemic causes**
- 2. Imaging evidence of loss of VIABLE MYOCARDIUM in a pattern consistent
with ischaemic cause**
- 3. Patho-anatomical findings of a prior MI**

Criteria For Type 1 Myocardial Infarction

1. Sx suggestive of Myocard. ischaemia
2. Elevation of cTn values > 5X 99th percentile URL
3. New ECG ischaemic changes or LBBB
4. Angiographic findings of Coronary Artery occlusion or flow-limiting plaques
5. Imaging evidence of new loss of viable myocardium or new RWMA

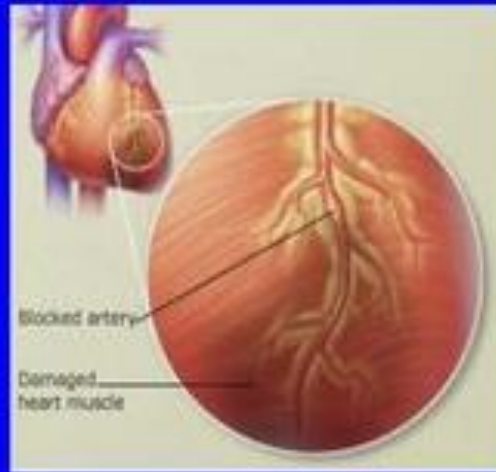
Type 1 myocardial infarction: Emphasis on the causal relationship of plaque disruption with coronary atherothrombosis



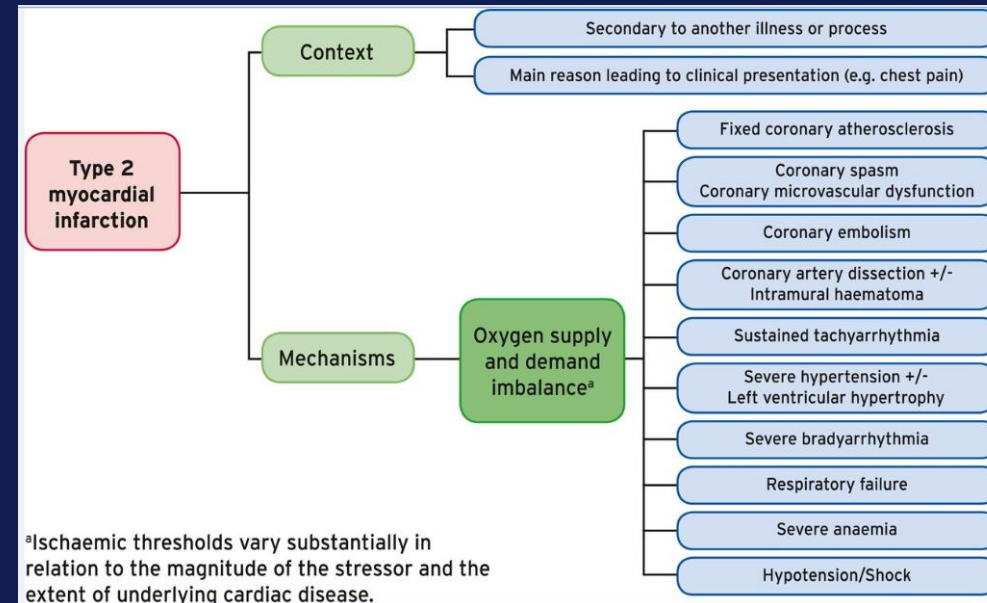
Criteria For Type 2 Myocardial Infarction

Type 2 myocardial infarction: Set by demand and supply imbalance unrelated to thrombosis;

- Type 2 myocardial infarction: Release of coronary artery disease. Different from Type 2 myocardial infarction



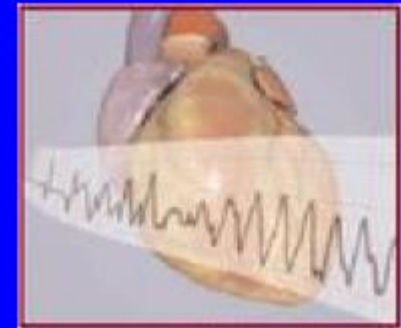
Emphasis on the settings causing O₂ demand-supply mismatch with NO/
Insignificant CAD without any relevance to prognosis & Rx



Criteria For Type 3 Myocardial Infarction

Diffrentiating between presumably ischaemia-driven event and SCD of non-ischaemic origin

- Patients who suffer cardiac death, with symptoms suggestive of myocardial ischaemia accompanied by presumed new ischaemic ECG changes like LBBB or ventricular fibrillation, but die before blood samples for biomarkers can be obtained, or before increases in cardiac biomarkers can be identified or myocardial infarction detected by autopsy examination.



Criteria For Type 4a Myocardial Infarction

1. Sx suggestive of Myocard. ischaemia
2. Elevation of cTn values > **5X 99th** percentile URL in normal baseline and > **20X 99th percentile** if baseline is elevated and stable or falling
3. New ECG ischaemic changes or LBBB
4. Angiographic findings of procedural complications like loss of patency of a major Coronary Artery or side branch with slow/no-flow or embolisation
5. Imaging evidence of new loss of viable myocardium or new RWMA

Emphasis on distinction between procedure-related (PCI) Myocardial Injury or Myocardial Infarction



Criteria For Type 4b Myocardial Infarction

1. ECG Evidence of Myocardial infarction
2. Rise & fall of Bio-markers of at least one value of cTn > 99th percentile URL
3. Stent thrombosis detected by CAG/autopsy



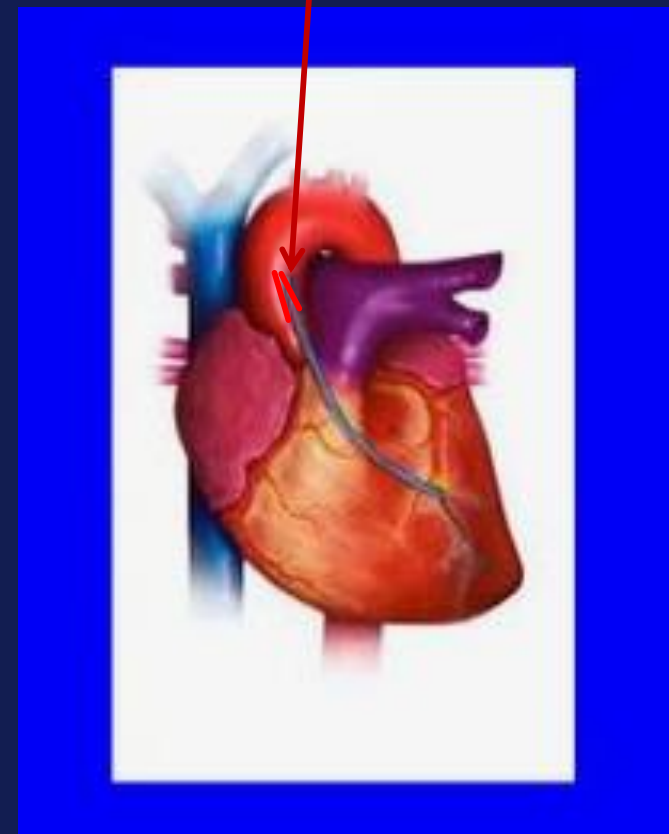
Criteria For Type 4c Myocardial Infarction

1. In-stent re-stenosis
2. All criteria of Type 4b

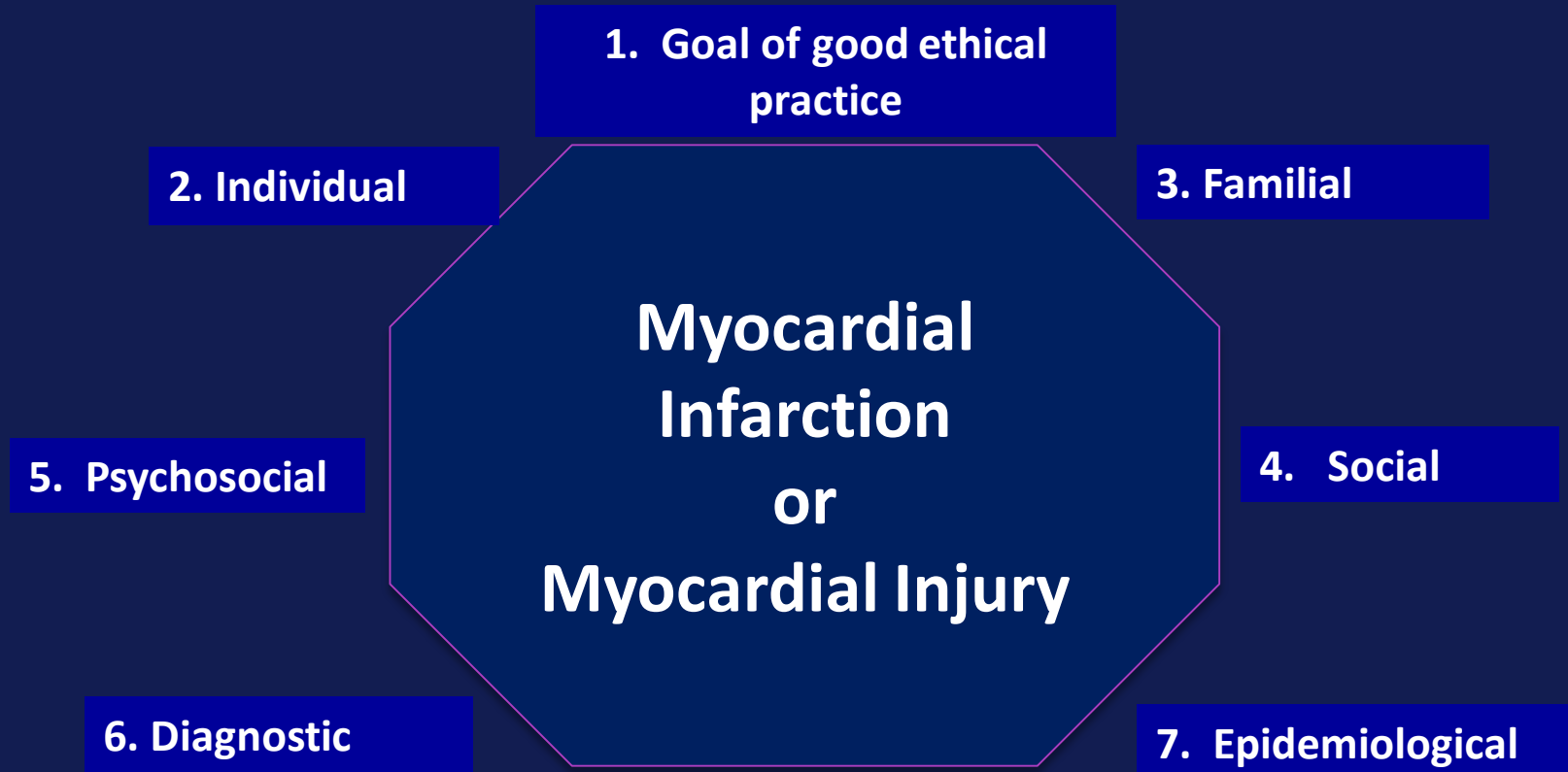
Criteria For Type 5 Myocardial Infarction

Emphasis on distinction between procedure-related (CABG) Myocardial Injury or Myocardial Infarction

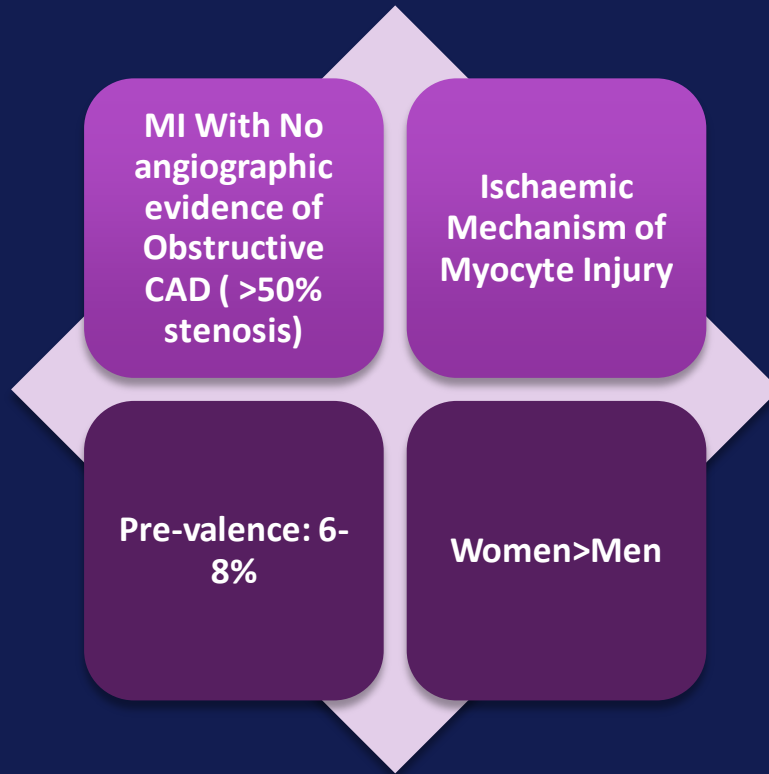
1. Elevation of cTn values $> 10X$ 99th percentile URL in case of baseline value < 99 th percentile
2. New pathologic Q-wave or new LBBB
3. Angiographic documentation of new graft or new native coronary artery occlusion
4. Imaging evidence of new loss of viable myocardium or new RWMA



Implications



MINOCA

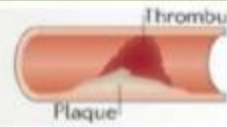


NSTEMI > STEMI

Potential mechanisms of MINOCA

It is a Clinical Syndrome and not a final diagnosis!

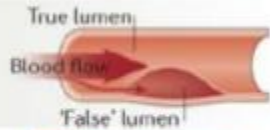
Plaque disruption



Vasospasm



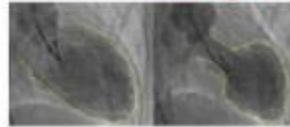
Dissection



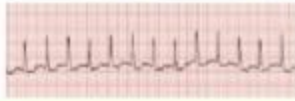
Thromboembolism



Microvascular dysfunction



Supply/demand mismatch



MINOCA:

A case study of a 55-year-old woman with an anterior STEMI presentation.

MINOCA - 'a working diagnosis'

Myocardial Infarction with Non-Obstructive Coronary Arteries

Differential diagnosis work up

Thrombophilia screen

No coagulation abnormalities detected

Cardiac Magnetic Resonance (CMR) imaging



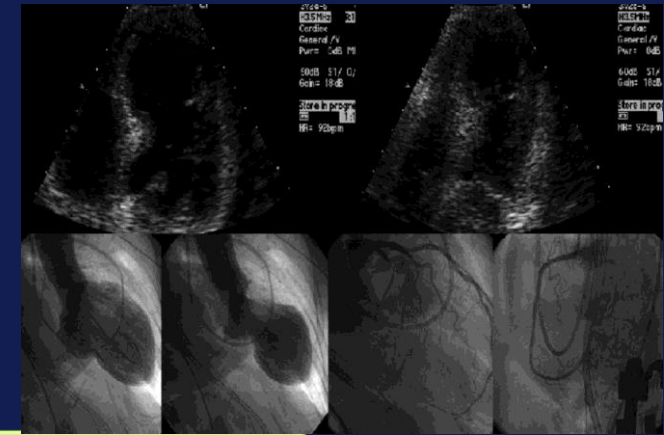
Subendocardial enhancement

Intravascular ultrasound imaging

Provocation spasm test

Final Diagnosis
Anterior Subendocardial Myocardial infarct

TAKO TSUBO SYNDROME (TTS)



Intense Emotional/Physical Stress

In-patient: 4-5%>. Cardiogenic shock, VSR, malignant arrhythmias

cTn: Modest transient elevation

BNP/NT-BNP elevated

Recovery of ventricular function 3-6 months

Post-Menopausal

ECG: Widespread ST-T elevation : prolonged QTc

Echo: Apical ballooning- no correlation with Wall Motion and single CAD

CAG: Mostly normal. 15% CAD

Catecholamines X

Conclusion and Take Home Message

It is an important, elaborate and improvised evidence-based definition. But in many cases the diagnosis can not be offered at the time of admission. And thus it is retrospective in nature in a good no. of cases

cTn is a big issue specially in Indian scenario bcz of the availability factor as well as the cost.

Even if MRI is not done routinely either pre- and post-PCI & is not standard of care of Therapy, this limitation does not affect the applicability of the definition

Most core laboratory-identified complications are not reported by the local investigators, which suggests ACL assessment may be required to ensure a systematic application of the Fourth universal definition of MI type 4a criteria.

Thanks one and all