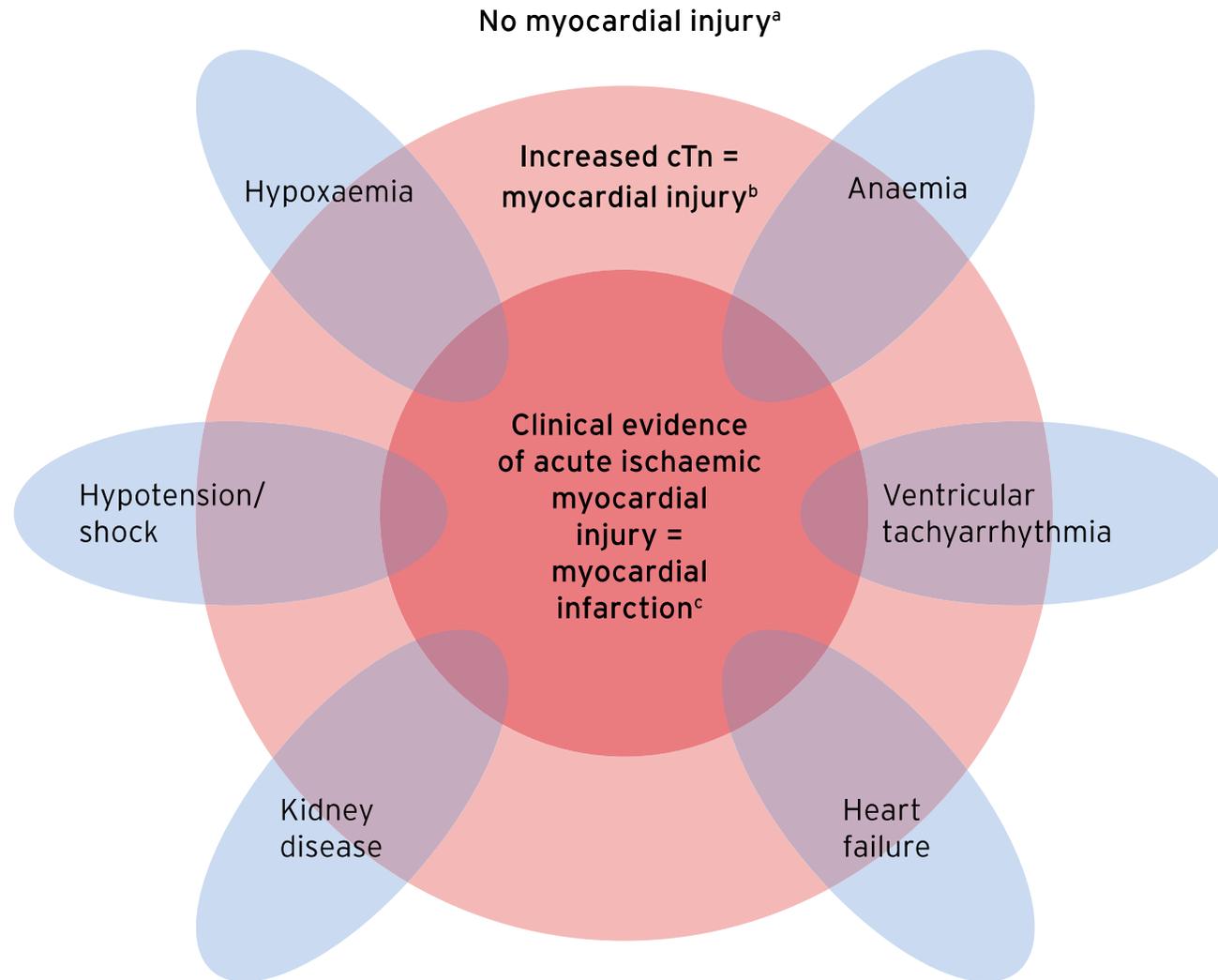# Έμφραγμα μυοκαρδίου στη ΜΕΘ

Κωνσταντίνος Τριανταφύλλου, MD, PhD
Επιμελητής Α΄

Καρδιολογικό Τμήμα Α΄ & Αιμοδυναμικό Εργαστήριο,
Γ.Ν.Α. "Ευαγγελισμός".

Spectrum of myocardial injury

No myocardial injury[a]

Increased cTn = myocardial injury[b]

Hypoxaemia

Anaemia

Hypotension/ shock

Ventricular tachyarrhythmia

Clinical evidence of acute ischaemic myocardial injury = myocardial infarction[c]

Kidney disease

Heart failure

Non-ischaemic myocardial injury or one of the MI subtypes?

Type 1
Acute myocardial injury related to acute atherothrombotic coronary artery disease. It is usually precipitated by atherosclerotic plaque disruption that reduces blood supply to the myocardium.

Type 2
Acute myocardial injury related to an imbalance between myocardial oxygen supply and demand secondary to stressors unrelated to acute coronary atherothrombosis.

Type 3
Related to patients who suffer cardiac death, with symptoms suggestive of acute myocardial ischaemia accompanied by new ischaemic ECG changes and die before biomarker values could be obtained.
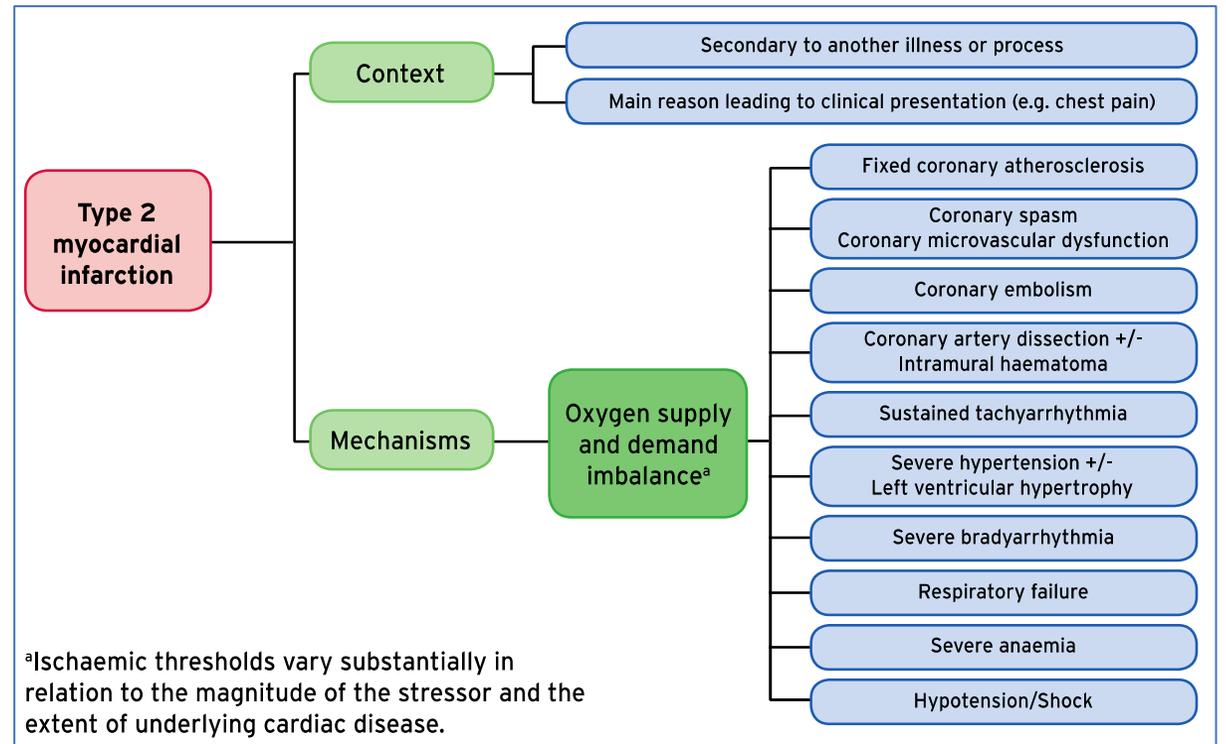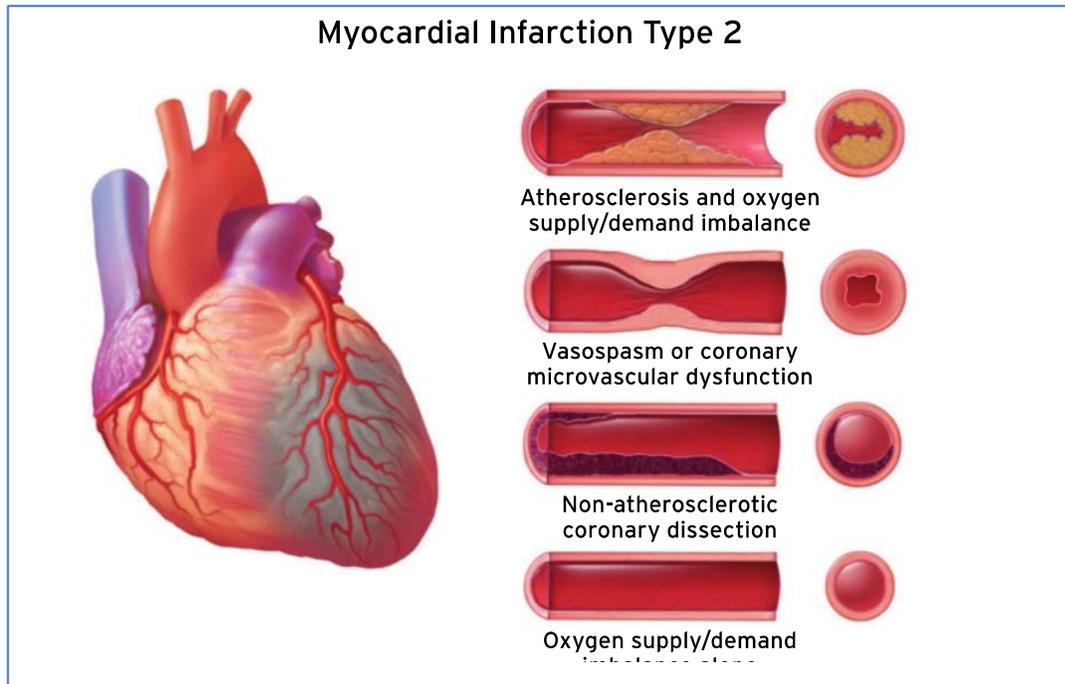
Type 4
a : PCI-related increases of cTn values >5 times the 99th percentile URL from a normal or if elevated, stable pre-procedural baseline. New myocardial ischaemia evidenced by ECG or imaging, or complications leading to reduced coronary blood flow are required.
b: acute myocardial ischaemic injury related to stent thrombosis
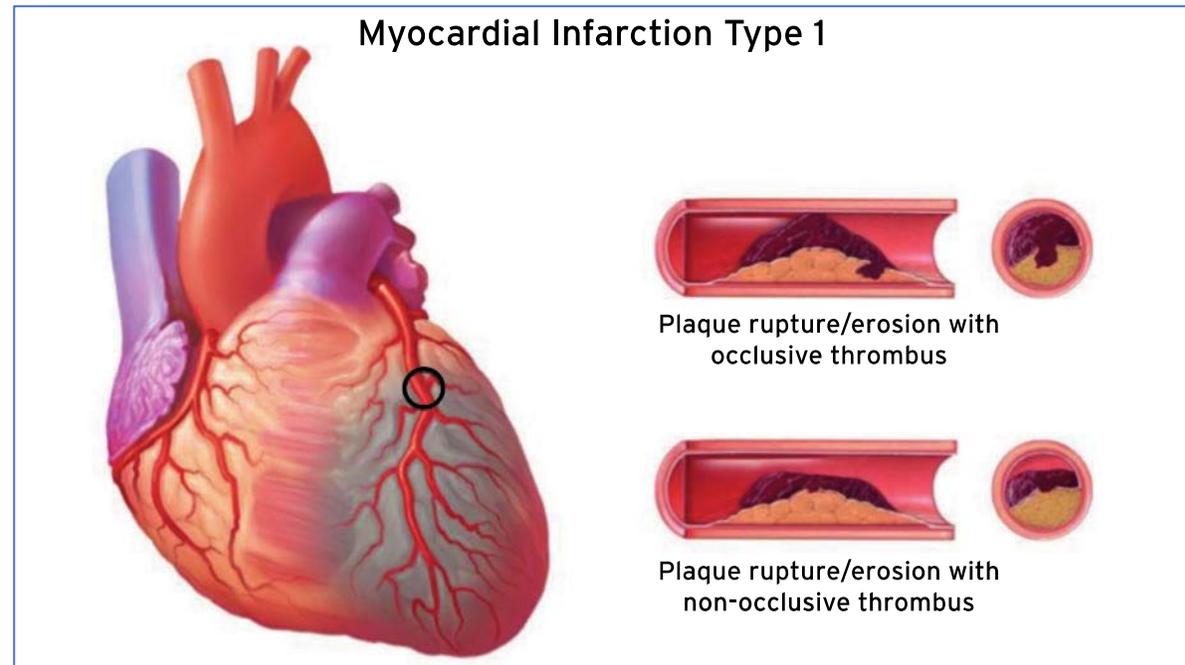c: acute myocardial ischaemic injury associated with restenosis.

Type 5
CABG-related increases of cTn values >10 times 99th percentile URL from a normal or if elevated, stable pre-procedural baseline. New myocardial ischaemia or new loss of myocardial viability is required.
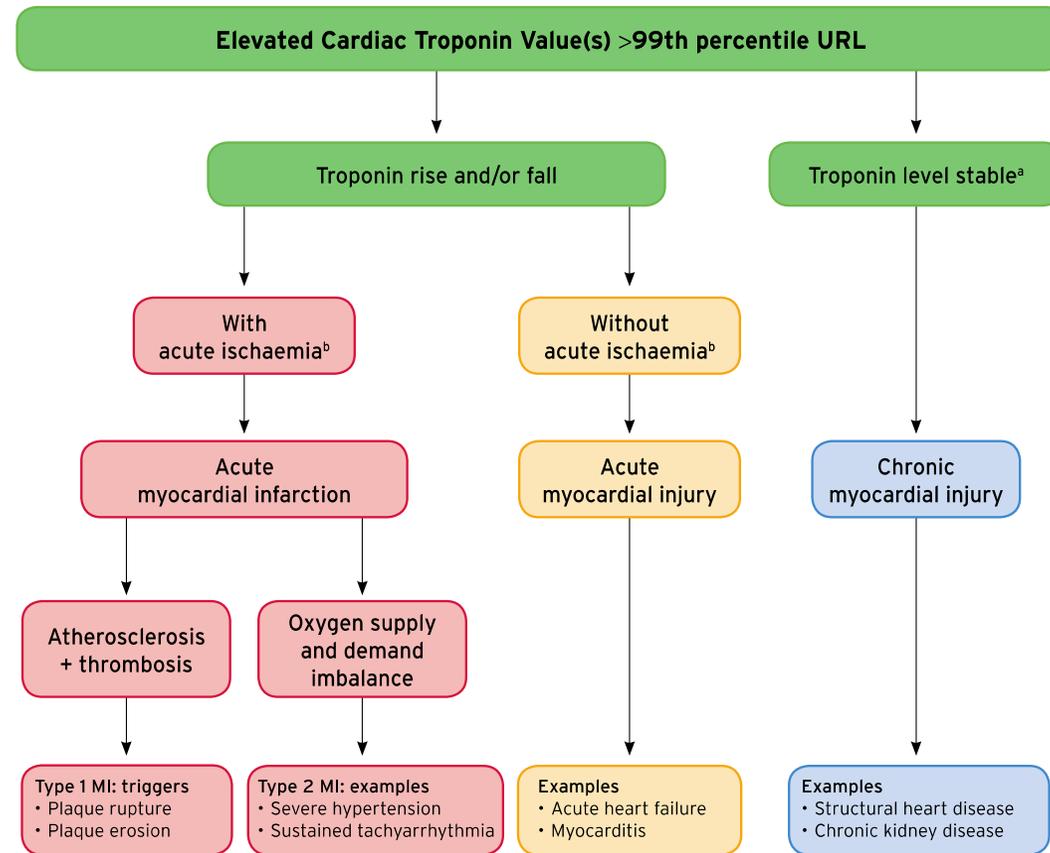
Myocardial Infarction Type 2

Atherosclerosis and oxygen supply/demand imbalance

Vasospasm or coronary microvascular dysfunction

Non-atherosclerotic coronary dissection

Oxygen supply/demand imbalance alone

Type 2 myocardial infarction

Context
- Secondary to another illness or process
- Main reason leading to clinical presentation (e.g. chest pain)

Mechanisms — **Oxygen supply and demand imbalance[a]**
- Fixed coronary atherosclerosis
- Coronary spasm / Coronary microvascular dysfunction
- Coronary embolism
- Coronary artery dissection +/- Intramural haematoma
- Sustained tachyarrhythmia
- Severe hypertension +/- Left ventricular hypertrophy
- Severe bradyarrhythmia
- Respiratory failure
- Severe anaemia
- Hypotension/Shock

[a]Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.

Thygesen K, Alpert JS, Jaffe AS, et al.
Fourth universal definition of myocardial infarction (2018).
*Eur Heart J 2019;40:237-69.*

Myocardial Infarction Type 1

Plaque rupture/erosion with
occlusive thrombus

Plaque rupture/erosion with
non-occlusive thrombus

Thygesen K, Alpert JS, Jaffe AS, et al.
Fourth universal definition of myocardial infarction (2018).
Eur Heart J 2019;40:237-69.

A model for interpreting myocardial injury.

Thygesen K, Alpert JS, Jaffe AS, et al.
Fourth universal definition of myocardial infarction (2018).
Eur Heart J 2019;40:237-69.
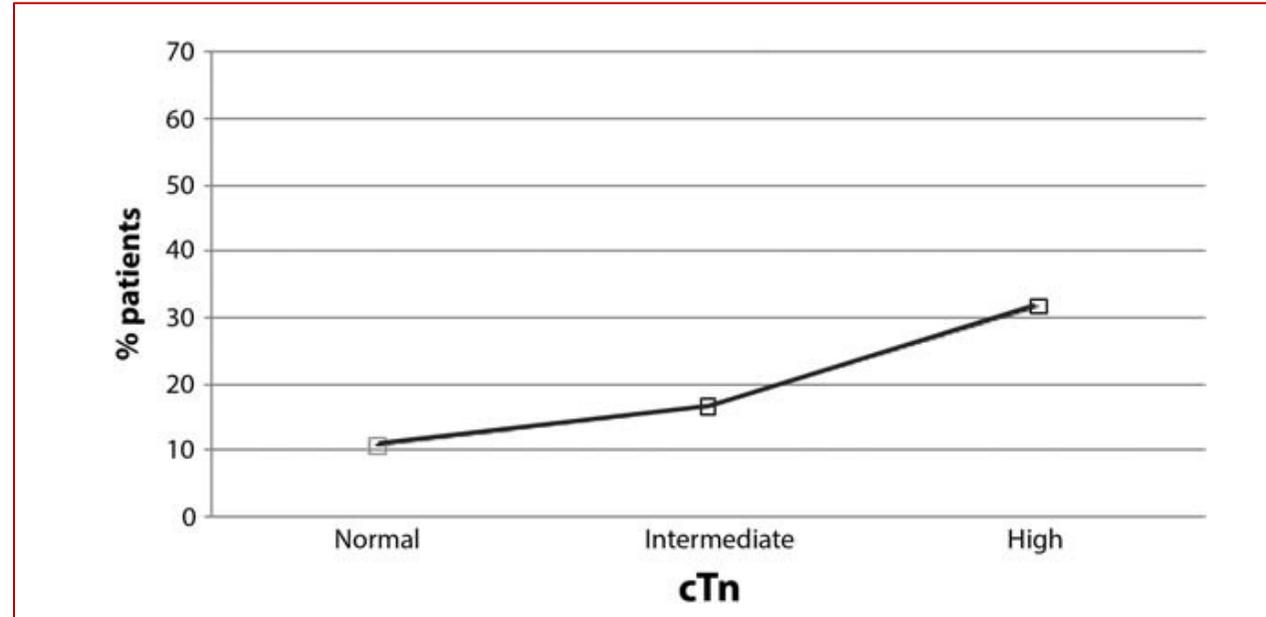
Relationship between 30-day mortality and troponin level.

➢ Patients with no or intermediate Tn elevation had a significantly lower associated risk of death if treated with statins.

➢ Tn positive patients had a significantly lower associated risk of death if treated with aspirin and/or β-blockers.

Myocardial infarction in intensive care units: A systematic review of diagnosis and treatment

Iain Carroll[1], Thomas Mount[2] and Dougal Atkinson[2]

19 papers

(a) Three papers considered the ability of ECG monitoring to diagnose MI.
(b) Eight papers compared the relationship of cardiac enzymes to clinical (symptom and ECG) findings.
(c) Three papers considered the use of echocardiography in diagnosis.
(d) Three considered the role of angiography in diagnosis.
(e) Two considered autopsy findings.
(f) Four considered treatment modalities available.

Suggested strategy for monitoring and diagnosis of MI in the ICU

Monitoring on leads V2, V3 or V4
Daily 12-lead ECG or 12-lead monitoring.
Avoidance of using automated ECG interpretation.

Daily troponin measurement in all patients.
Evaluation of ECG by 2 clinicians considering and enzyme results.

Use of echocardiography to identify RWMA, especially when great Tn increase.

If there are no echocardiographic abnormalities, angiography is unlikely to reveal a treatable lesion.

Role of interventional cardiologist

Understand the clinical context of the ICU patient before cath (benefit vs risk)

Decide when to cath

Take precautions to avoid cath related complications

Decide about revascularization (benefit vs risk, strategy, timing)

Ευχαριστώ πολύ για την προσοχή σας