The Pathology of Acute Myocardial Infarction

Patrick J Gallagher  MD PhD
Centre for Medical Education
University of Bristol
Scope of Presentation

• Incidence, classification and treatment of acute myocardial infarction
• The classical pathology of acute myocardial infarction
• The contemporary pathology of acute myocardial infarction
• Post operative myocardial infarction: Recent pathophysiological advances
Classification of Acute Myocardial Infarction

- Clinical/physiological classification on basis of ventricular function
- Biochemical confirmation from troponin estimations
- ECG classification on basis of ST segment changes into NSTEMIs and STEMIs
- Pathological distinction between transmural and subendocardial infarcts
Incidence of Acute Myocardial Infarction

- A small increase both STEMIIs and NSTEMIIs in the late 1990s
- A progressive and sustained decrease in incidence of STEMIIs subsequently
- Progressive reduction in acute mortality
- Progressive increase in prevalence of cardiac failure
Age- and Sex-Adjusted Incidence Rates of Acute Myocardial Infarction, 1999 to 2008.

Classical Appearances

- Confluent area of clearly defined coagulative necrosis
- Surrounding zone of hyperaemia
- Coronary artery thrombosis
- Adherent mural thrombus
- Myocardial rupture with PEA arrest
- Now rarely seen except in out of hospital deaths after “missed” myocardial infarcts
Myocardial Reperfusion

• Early and successful myocardial reperfusion with thrombolytic drugs or primary cardiac intervention (PCI) is the ideal management strategy for AMI

• In the short and long term the pattern of cardiac pathology is very different to conventional textbook descriptions
Current Appearances

- Successful reperfusion associated with haemorrhage into infarcted tissue
- Spotty ill defined areas of necrosis
- Incidence of mural thrombus and rupture much reduced
- Small residual thrombi after thrombolysis
- Stents in situ after percutaneous interventions
Reperfusion Injury

- Prompt restoration of coronary blood flow preserves myocardium
- Histological changes include haemorrhage, contraction band necrosis, endothelial swelling and vascular plugging
- In a small proportion of cases reperfusion is associated with paradoxical death of myocytes, so called *lethal reperfusion injury*
Myocardial Infarction after Surgery

- The infarcts are often small and usually close to an area of healed infarction
- Coronary thrombosis is rare
- Critical ischaemia is the result of tachycardia and coronary narrowing
- The infarcts are usually haemorrhagic, possibly because of reperfusion when heart rate declines
Ischaemia after Vascular Surgery

- 185 patients, 70% male, mean age 66yrs
- 66 had transient ischaemic events with ST segment depression
- 12 (6.5%) had a myocardial infarct
- Mean duration of ischaemia was 225 mins. Most had a post operative tachycardia
- All infarcts were non Q wave/NSTEMIs, diagnosed biochemically

*Landesberg et al JACC 2001;37:1839-45*
PeriOperative Ischaemic Evaluation (POISE study)

- 8351 patients, 190 centres, 23 countries
- Four post operative cardiac biomarkers and a range of clinical, ECG and imaging
- 415 (5.0%) had a perioperative MI
- Only 34.7% had ischaemic symptoms, only 12.3% had Q waves
- 30 day mortality 11.6% in those with MIs but 2.2% in remainder

Devereaux et al Annals of Internal Medicine 2011;154:523
Risk Factors for Perioperative MI

• Post operative increase heart rate
• History of stroke
• Major vascular or orthopaedic surgery
• Creatinine > 175µmol/l
• Increasing age
• Emergency or urgent surgery
• Serious bleeding (≥ 2 units transfused)
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Summary Points

- New treatments have improved the survival after myocardial infarction
- These are associated with a range of new pathological changes
- All pathologists must be able to identify and evaluate coronary stents
- Post operative myocardial infarction must be identified both clinically and at autopsy