Coronary artery disease

By

Dr. Sheerwan
Interventional Cardiologist

The most common cause of death worldwide.

Occult CAD is common in those who present with other forms of atherosclerotic vascular disease, such as intermittent claudication or stroke, and is an important cause of morbidity and mortality in these patients.

Pathogenesis

- Atherosclerosis : the most common cause
- Aortitis
- Vasculitis
- Autoimmune connective tissue diseases

Stable angina Ischaemia due to fixed atheromatous stenosis of one or more coronary arteries

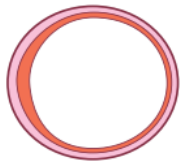
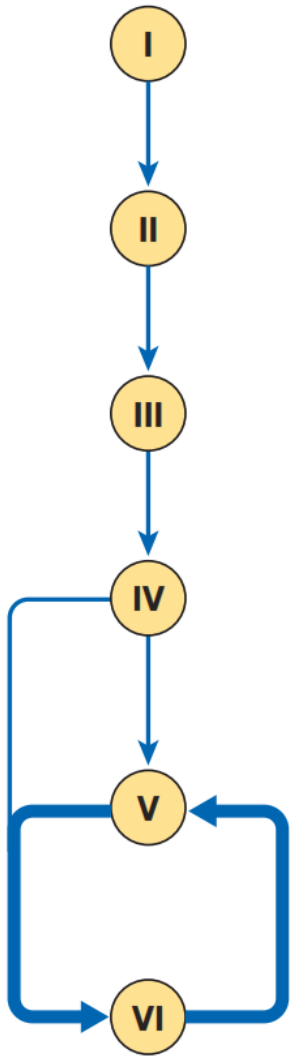
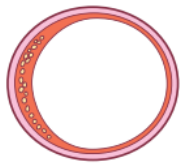
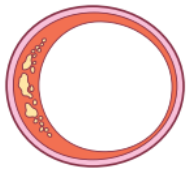
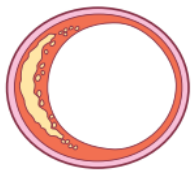
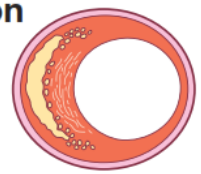
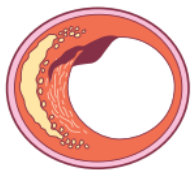
Unstable angina Ischaemia caused by dynamic obstruction of a coronary artery due to plaque rupture or erosion
with superimposed thrombosis

Myocardial infarction : Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture or erosion with superimposed thrombosis

Heart failure Myocardial dysfunction due to infarction or ischaemia

Arrhythmia Altered conduction due to ischaemia or infarction

Sudden death Ventricular arrhythmia, asystole or massive myocardial infarction

Nomenclature and main histology	Sequences in progression	Main growth mechanism	Earliest onset	Clinical correlation
Type I (initial) lesion Isolated macrophage foam cells 		Growth mainly by lipid accumulation	From first decade	Clinically silent
Type II (fatty streak) lesion Mainly intracellular lipid accumulation 				
Type III (intermediate) lesion Type II changes and small extracellular lipid pools 			From third decade	
Type IV (atheroma) lesion Type II changes and core of extracellular lipid 				Clinically silent or overt
Type V (fibroatheroma) lesion Lipid core and fibrotic layer, or multiple lipid cores and fibrotic layers, or mainly calcific, or mainly fibrotic 		Accelerated smooth muscle and collagen increase	From fourth decade	
Type VI (complicated) lesion Surface defect, haematoma-haemorrhage, thrombus 		Thrombosis, haematoma		

Many risk factors have been identified for atherosclerosis

Unknown factors account for up to 40%

- Age and sex

Pre-menopausal women have lower rates of disease than men, although the gender difference disappears after the menopause.

- Genetics

➢ Early-onset disease (ag< 50 in men and < 55 in women)
➢ Shared genetic, environmental and lifestyle factors.
➢ **Most common risk factors, such as hypertension,**
➢ **hyperlipidaemia and diabetes mellitus, are inherited in a polygenic manner.**

- Smoking : the most important modifiable risk factor
- Hypertension
- Hypercholesterolaemia
- Diabetes mellitus
- Physical activity
- Obesity
- Alcohol
- Diet
- Social deprivation

The effect of risk factors can be multiplicative rather than additive

.

Management

Primary prevention : aims to introduce lifestyle changes or therapeutic interventions to prevent CAD and other forms of atherosclerosis

Secondary prevention : involves initiating treatment in patients who already have had an event, with the aim of reducing therisk of subsequent events.

Primary prevention

- Do not smoke
- Take regular exercise (minimum of 20 mins, three times per week)
- Maintain an ‘ideal’ body weight
- Eat a mixed diet rich in fresh fruit and vegetables
- Aim to get no more than 10% of energy intake from saturated fat

Angina pectoris

A symptom complex caused by transient myocardial ischaemia occurs whenever there is an imbalance between myocardial oxygen supply and demand

Pathogenesis

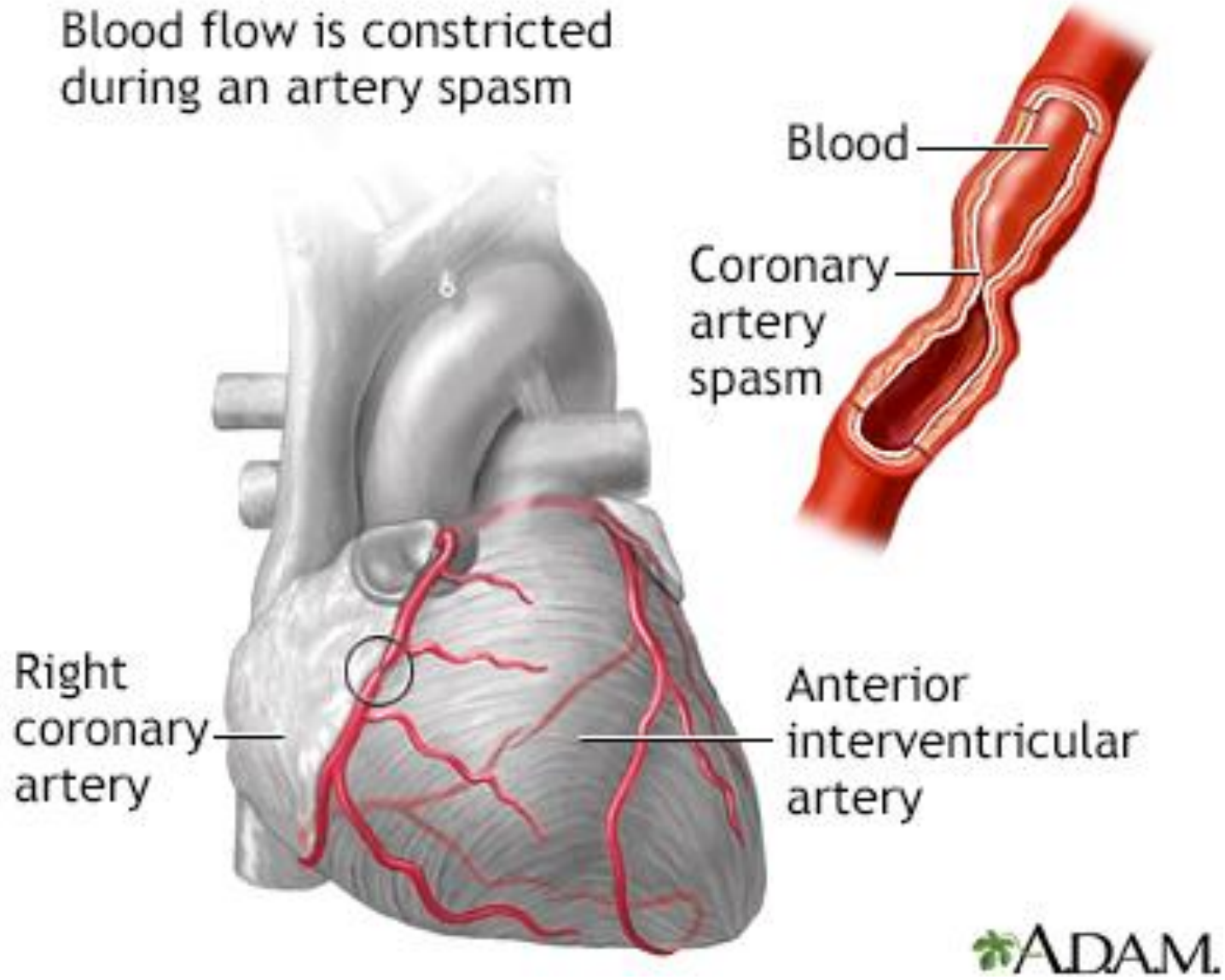
- Coronary atherosclerosis : the most common cause
- Aortic valve disease
- hypertrophic cardiomyopathy
- Vasculitis

- **Coronary artery spasm (prinzmetal angina)**

➢ Coexist with atherosclerosis
➢ isolated phenomenon in less than 1% of cases

- **Syndrome X**

➢ typical angina on effort
➢ objective evidence of myocardial ischaemia on stress testing
➢ normal coronary arteries on angiography is sometimes known as syndrome X.
➢ Many of these patients are women


Blood flow is constricted during an artery spasm
Blood
Coronary artery spasm
Right coronary artery
Anterior interventricular artery
A.D.A.M.

16.39 Activities precipitating angina

Common

- Physical exertion
- Cold exposure
- Heavy meals
- Intense emotion

Uncommon

- Vivid dreams (nocturnal angina)
- Lying flat (decubitus angina)

Clinical features

Central chest pain, discomfort or breathlessness that is predictably precipitated by exertion or other forms of stress and relieved by rest

Physical examination

- **Other cause** : valve disease (particularly aortic)
- Risk factors : (hypertension, diabetes mellitus)
- Complications :left ventricular dysfunction (cardiomegaly, gallop rhythm)
- Manifestations of other arterial disease (carotid bruits, peripheral arterial disease)
- Exacerbating factors: (anaemia, thyrotoxicosis).

Investigations

- ECG
- CXR
- Echocardiography

Stress tests:

- exercise ECG
- Myocardial perfusion scanning
- Stress echocardiography

Angiography :

- CT coronary arteriography
- Coronary angiography

*

Management

- Careful assessment of the extent and severity of arterial disease
- Identification and treatment of risk factors
- Introduction of drug treatment for symptom control
- Identification of high-risk patients for treatment to improve life expectancy.

Drugs :

- Antiplatelet therapy : aspirin , Clopidogrel
- Lipid lowering drugs : statins

Antianginal drugs

- Nitrate
- Beta blockers : metoprolol , bisoprolol
- Calcium channel antagonist : amlodipine , diltiazim

Revascularisation

Other antianginal drugs:

Nicorandil : Potassium channel activators

Ivabradine: Sinus node blocker

Ranolazine: inhibits the late inward sodium current in coronary artery smooth muscle cells, with a secondary effect on calcium flux and vascular tone

Non-pharmacological treatments

- Percutaneous coronary intervention
- Coronary artery bypass grafting

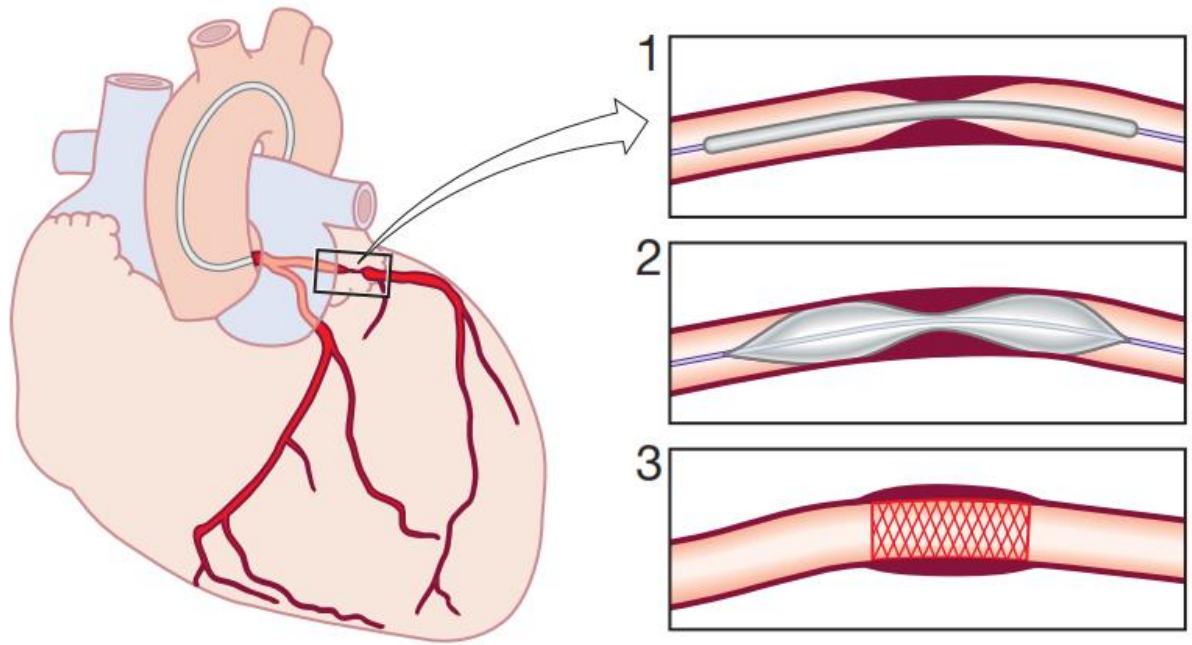
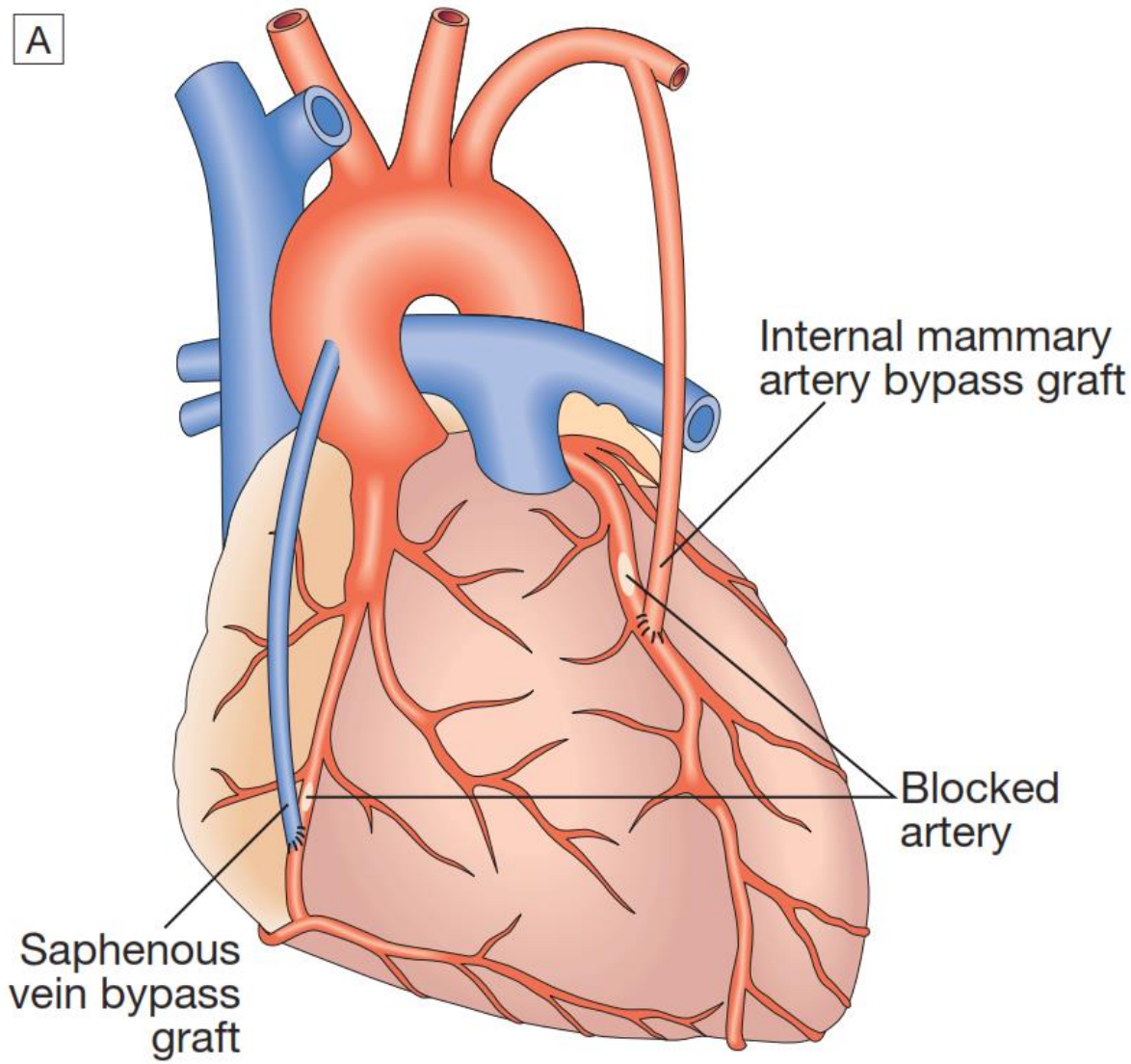


Fig. 16.59 Percutaneous coronary intervention. A guidewire is advanced from the radial (or femoral) artery to the coronary artery under radiographic control (1). A fine balloon catheter is then advanced over the guidewire to the stenotic coronary artery and the balloon is inflated to dilate the stenosis (2). When this has been achieved, a stent is usually placed at the site of the stenosis to maintain patency of the artery (3) (see text for more details).


A
Internal mammary artery bypass graft
Blocked artery
Saphenous vein bypass graft

Acute coronary syndrome

Unstable angina :

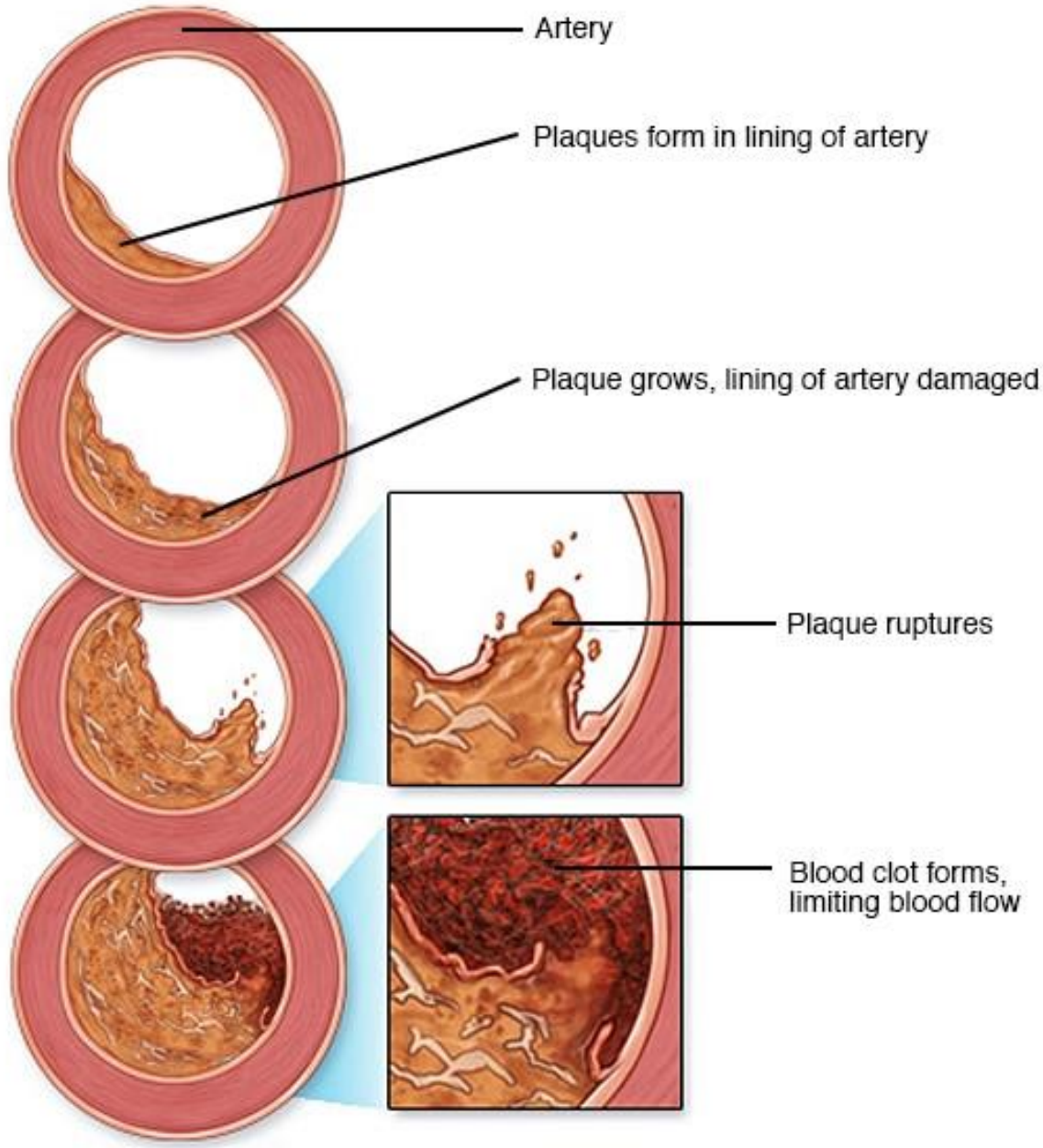
New-onset

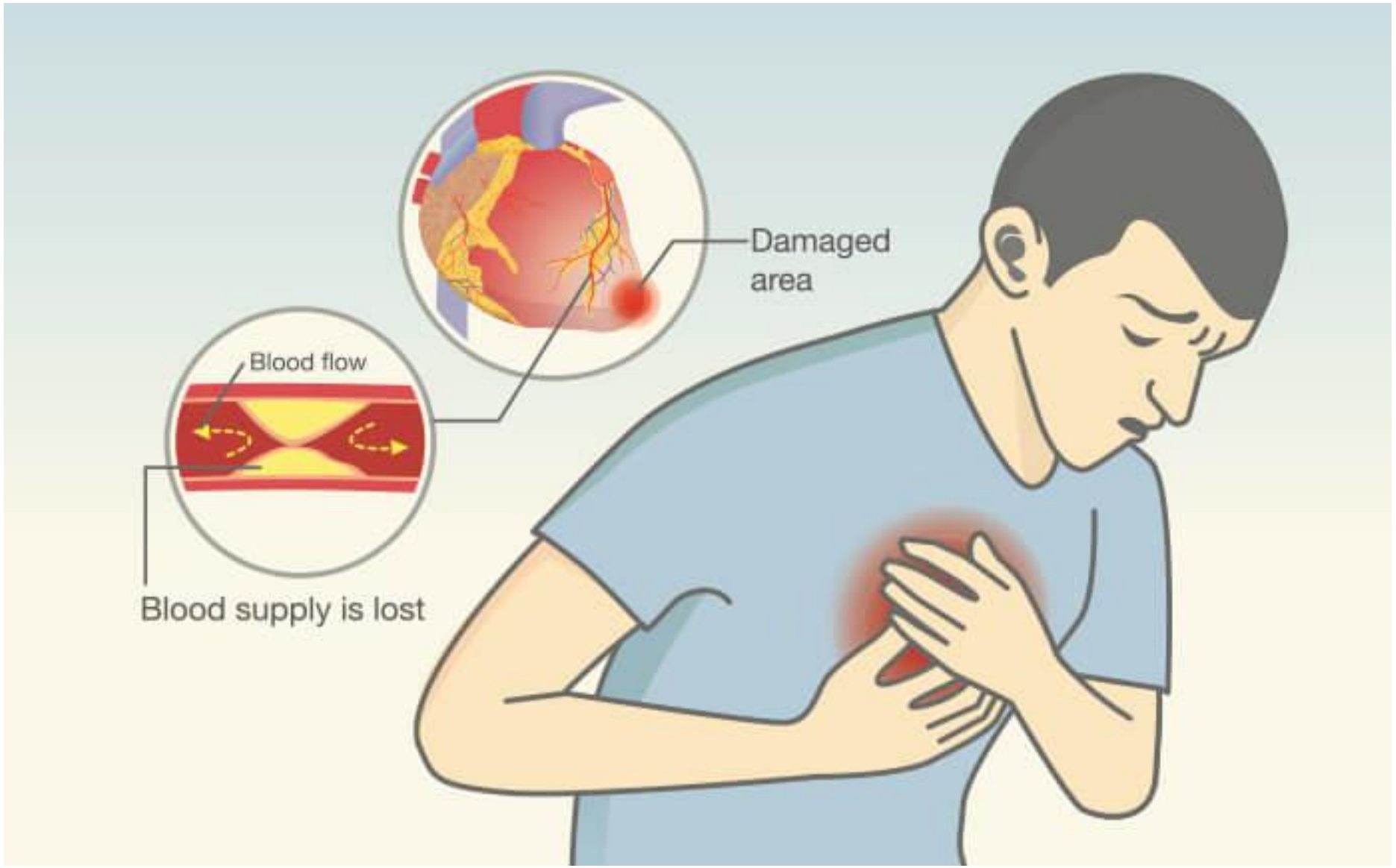
Or rapidly worsening angina (crescendo angina)

Or angina on minimal exertion

Or angina at rest in the absence of myocardial damage.

Myocardial infarction: there is evidence of myocardial necrosis.




Damaged
area
Blood flow
Blood supply is lost

16.49 Clinical features of acute coronary syndromes

Symptoms

- Prolonged cardiac pain: chest, throat, arms, epigastrium or back
- Anxiety and fear of impending death
- Nausea and vomiting
- Breathlessness
- Collapse/syncope

Physical signs

Signs of sympathetic activation

- Pallor
- Sweating
- Tachycardia

Signs of vagal activation

- Vomiting
- Bradycardia

Signs of impaired myocardial function

- Hypotension, oliguria, cold peripheries
- Narrow pulse pressure
- Raised jugular venous pressure
- Third heart sound
- Quiet first heart sound
- Diffuse apical impulse
- Lung crepitations

Low-grade fever

Complications

- Mitral regurgitation
- Pericarditis

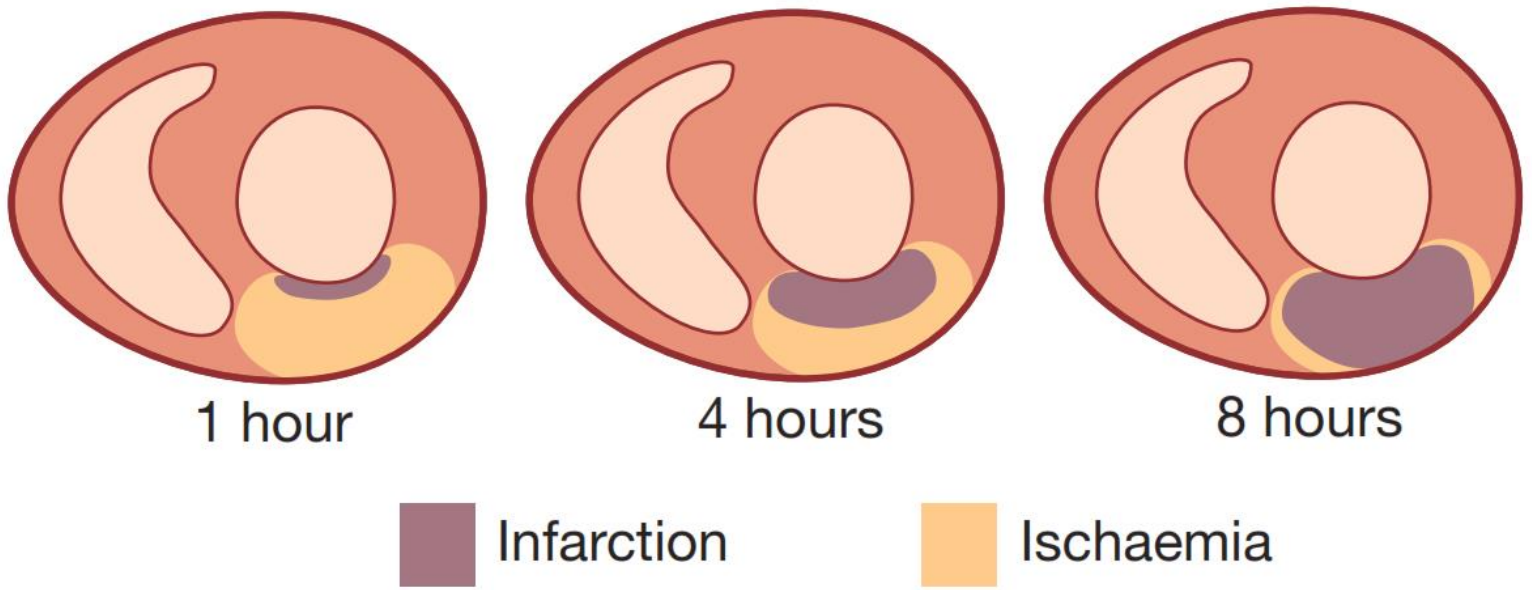


Fig. 16.63 The time course of myocardial infarction. The relative proportion of ischaemic, infarcting and infarcted tissue slowly changes over a period of 12 hours. In the early stages of myocardial infarction, a significant proportion of the myocardium in jeopardy is potentially salvageable.

The pain occurs in the same sites as angina but is usually more severe and lasts longer; it is often described as a tightness, heaviness or constriction in the chest.

Most patients are breathless

Painless or ‘silent’ MI may also occur and is particularly common in older patients or those with diabetes mellitus.

Syncope : by an arrhythmia or profound hypotension.

Sudden death

Complications:

- Heart failure
- Arrhythmias
- Recurrent angina
- Pericarditis
- Mural thrombus and embolism
- Mechanical complications : Papillary muscle rapture , free wall rapture , ventricular septal rapture
- Ventricular aneurysm

Investigations:

- ECG
- Echocardiography
- Cardiac biomarkers : serum troponin
- Chest X ray

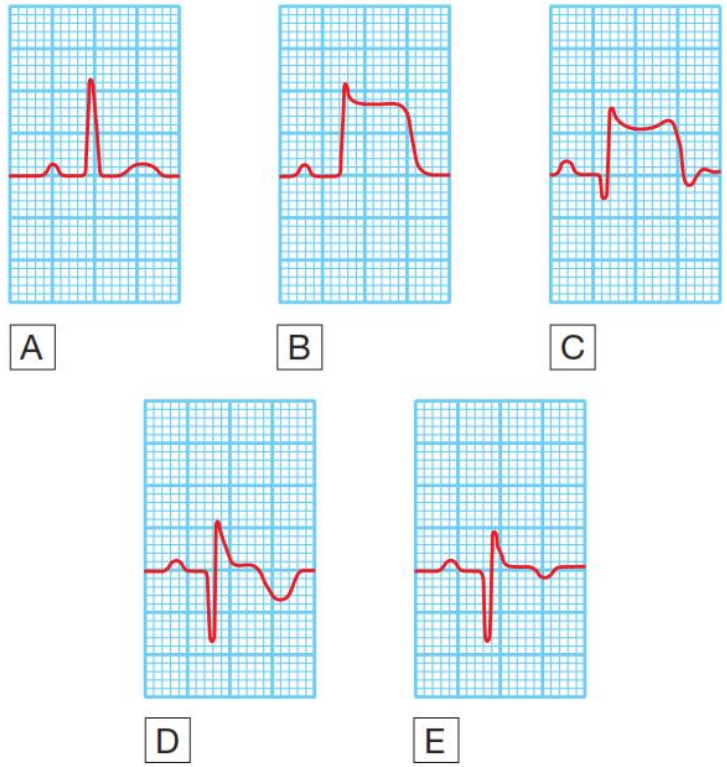


Fig. 16.65 The serial evolution of ECG changes in transmural myocardial infarction. A Normal ECG complex. B Acute ST elevation ('the current of injury'). C Progressive loss of the R wave, developing Q wave, resolution of the ST elevation and terminal T-wave inversion. D Deep Q wave and T-wave inversion. E Old or established infarct pattern; the Q wave tends to persist but the T-wave changes become less marked. The rate of evolution is very variable but, in general, stage B appears within minutes, stage C within hours, stage D within days and stage E after several weeks or months. This should be compared with the 12-lead ECGs in Figures 16.66–16.68.

Treatment:

- Analgesia: morphine sulphate

- Antiplatelet : aspirin , clopidogrel or Ticagrelor

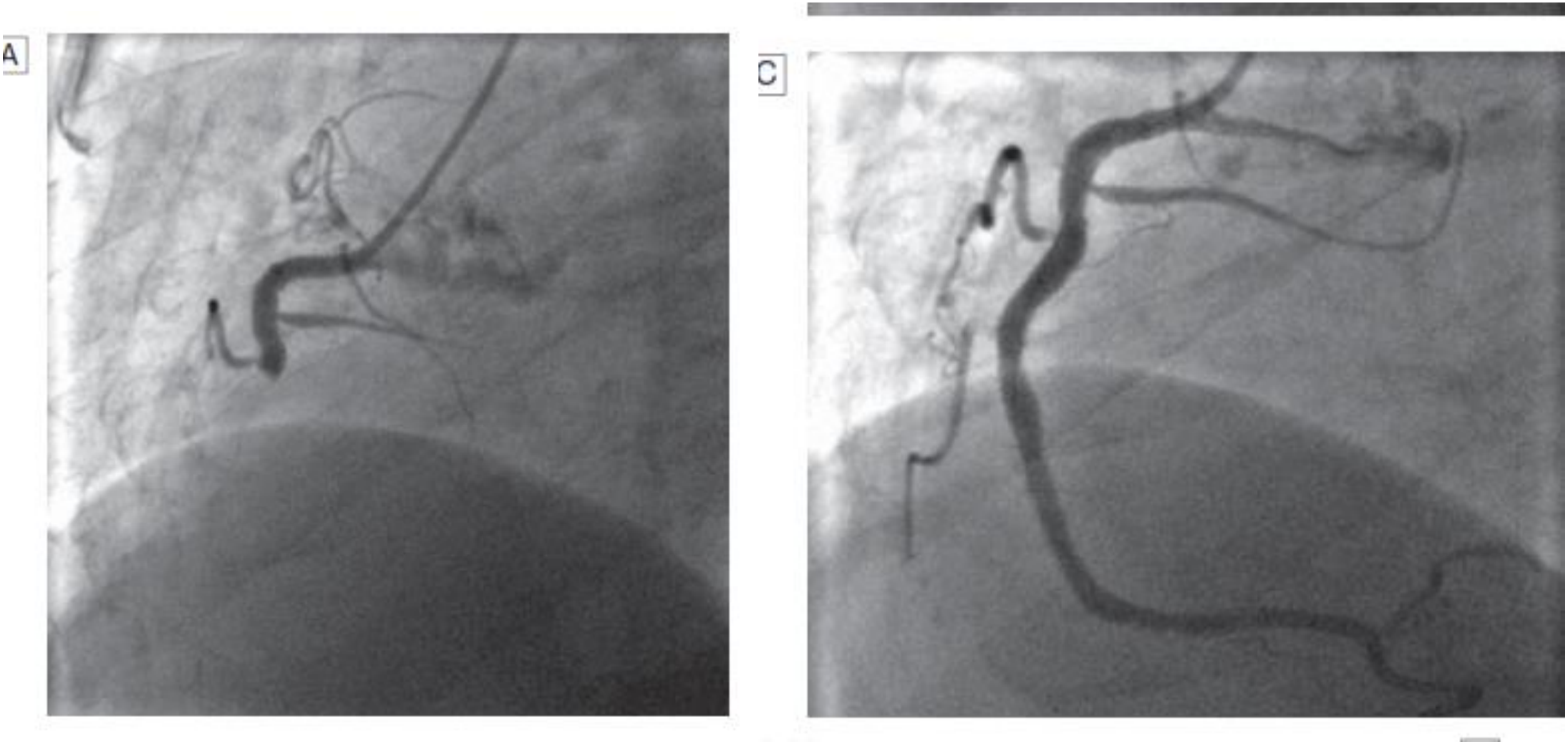
- Reperfusion :
  - Revascularization : PCI (percutaneous coronary intervention) or CABG
  - IV fibrinolytic agents (for ST elevation myocardial infarction only)

- Anticoagulation ; Unfractionated heparin or LMW heparin

- Lipid lowering drugs : statin

- Antianginal drugs : Beta blockers , calcium channel blockers , nitrate

- ACEI : angiotensin converting enzyme inhibitors


A
C

- Heart failure management
- Risk factors management : smoking , DM , HT
- Rehabilitation

THANKS