**Definition**

- **Epidemiology**
  Widespread in Western industrialized countries • Overall incidence increases with life expectancy • Prevalence approximately 4% • Present in approximately 5–10% of the male population • More common in males than in females (4:1).

- **Etiology, pathophysiology, pathogenesis**
  Endothelial damage from “atherogenic risk factors” • Formation of atheromatous plaques • Reduction of luminal diameter (becomes critical at 70% narrowing) • Diminished coronary flow reserve.
  **Risk factors:** Hyperlipoproteinemia, hypercholesterolemia, nicotine misuse, diabetes mellitus, arterial hypertension, obesity, familial disposition.

**Imaging Signs**

- **Modality of choice**
  Invasive coronary angiography.

- **Chest radiograph findings**
  Findings depend on the severity of the disease • Cardiopulmonary findings are initially normal • In severe cases LV enlargement, pulmonary venous congestion, and/or pleural effusion is seen.

- **Echocardiographic findings**
  (Stress-induced) LV dysfunction (regional hypo- or akinesia) • LV dilatation may precede dilatation of the LA, depending on disease severity • Secondary mitral insufficiency • Chronic congestion leads to pulmonary vein dilatation.

- **Nuclear medicine and PET findings**
  Detection and quantification of myocardial perfusion defects and dysfunction.

- **CT findings**
  Calcified plaques (calcium scoring) and soft plaques on multidetector CT angiography • Coronary stenoses • Signs of left-sided heart failure.

- **MRI findings**
  Same as echocardiography • MRA may show coronary stenoses • Decreased myocardial perfusion in response to pharmacologic stress (adenosine) • Myocardial infarction is detected by delayed contrast enhancement of the myocardial scar after gadolinium-DTPA administration (IR GE sequence).

- **Invasive diagnostic procedures**
  **Coronary angiography:** One or more stenotic coronary arteries • **IVUS:** More accurately delineates plaques and stenoses.
Fig. 1.1  P-A chest radiograph in CHD. Marked enlargement of the LV due to heart failure, and increased pulmonary vascular markings due to chronic pulmonary venous congestion. A goiter with tracheal narrowing was noted as an incidental finding.

Fig. 1.2  High-grade stenosis of the LCX shown by coronary angiography.
Fig. 1.3 Myocardial scintigraphy. An apical perfusion defect (arrow).

Fig. 1.4 Multi-detector CT. Diffuse sclerosis of the LCA.
Fig. 1.5  Coronary stent in a 65-year-old woman with known CHD. Curved MPR of a stent in segment 3 of the right coronary artery shows no morphologic evidence of in-stent restenosis.

Fig. 1.6a, b  Coronary plaque in a 56-year-old man with nonspecific chest discomfort. The patient had no ECG abnormalities and an echocardiogram also showed normal findings. Coronary status was investigated by cardiac CT. Two mildly stenosing mixed plaques (arrows) are seen in segments 6 and 8 of the LAD artery (a). Curved MPR of the right coronary artery shows moderately stenosing calcified plaques (white arrows) in segments 2 and 3 and a moderately stenosing soft plaque (black arrow) in segment 2 (b).
Coronary status in a 60-year-old woman with a familial disposition for CHD, current dyspnea, and non-specific ECG changes. Cardiac CT was done to exclude CHD, and VRT was used for three-dimensional cardiac imaging. This technique is useful for demonstrating findings (a), but the actual diagnosis should always rely on thin reconstructed slices or MPRs (see below). Curved MPRs of normal coronary vessels (b). Left panel: RCA; center panel: LAD; right panel: LCX.
Clinical Aspects

Typical presentation
Angina pectoris  • Exertional dyspnea  • Heart failure  • Cardiac arrhythmias.

Treatment options
Medical treatment for ischemic heart disease and cardiac insufficiency  • Interventional (PTCA, stent implantation) or surgical (aortocoronary bypass) myocardial revascularization.

Course and prognosis
Depend on the location and degree of coronary stenosis, myocardial ischemia, LV function (EF), and continued exposure to risk factors  • Complications: Arrhythmias, myocardial infarction, left-sided heart failure, sudden cardiac death.

What does the clinician want to know?
Number, location, and degree of coronary stenoses  • Signs of heart failure  • LV enlargement  • EF (important prognostic indicator!).

Differential Diagnosis

Coronary anomalies
– Anomalous origin of the coronary arteries
– Life-threatening variant: ALCA, most symptomatic during exercise

Syndrome X
– Angina pectoris
– Angiographically normal coronary arteries

Cardiomyopathies
– Impaired LV function
– Angiographically normal coronary arteries

Tips and Pitfalls

Suspicion of CHD warrants early investigation of coronary status and individualized risk assessment to assess prognosis and provide optimum treatment.

Selected References


**Definition**

- **Etiology**
  Variety of causes: Infectious • Autoimmune • Metabolic • Toxic • Neoplastic • Traumatic • Idiopathic • Idiopathic and infectious causes are responsible for approximately 80% of cases.

- **Pathophysiology, pathogenesis**
  Pericardial thickening and fibrous exudation during the acute phase (audible “pericardial rub,” pericarditis sicca) • Often accompanied by pericardial effusion (exudative pericarditis) • Myocardial involvement may occur (perimyocarditis) • Over time, pericarditis may lead to fibrous adhesion of the pericardial layers with regional constriction of the heart • Late sequelae may include calcifications (constrictive pericarditis).

**Imaging Signs**

- **Modality of choice**
  Echocardiography • MRI provides the highest sensitivity in equivocal cases.

- **Chest radiograph and CT findings**
  Often normal findings • May show signs of pericardial effusion • Pulmonary infiltrates and lymphomas in the setting of infection • CT may demonstrate pericardial thickening.

- **Echocardiographic findings**
  Pericardial effusion • Diastolic dysfunction due to constriction • Limited ability to evaluate pericardial morphology.

- **MRI findings**
  Same as Echocardiographic findings • Better visualization of the pericardium • Pericardial thickening and effusion • Contrast-enhanced imaging in acute inflammation (fat-saturated dark-blood T1-weighted TSE or IR GE sequence).

- **Invasive testing**
  May be appropriate in selected cases to exclude an acute coronary syndrome (see also Postinfarction Pericarditis and Dressler Syndrome).

**Clinical Aspects**

- **Typical presentation**
  Systemic inflammatory signs (fever, cough) • Retrosternal chest pain that improves on sitting up and leaning forward • ECG changes in 90% of patients • May take an asymptomatic course (e.g., in collagen diseases or uremia).

- **Treatment options**
  Steroidal and nonsteroidal anti-inflammatory agents • Aspirin • Antibiotics • Pericardial drainage for hemodynamically significant effusion or pericardial tamponade.
**Fig. 5.3** Acute pericarditis in a 37-year-old man. T1-weighted dark-blood TSE sequence in the four-chamber plane shows marked thickening and ill-defined margins of the pericardium (arrows). There is no pericardial effusion!

**Fig. 5.4** Contrast-enhanced IR GE sequence 15 min after administration of 0.2 mmol gadolinium-DTPA/kg. Four-chamber view shows marked enhancement of the pericardium (arrows).

**Fig. 5.5** Severe pericarditis secondary to a tonsillar abscess in a 20-year-old woman. Postcontrast CT shows pericardial effusion, marked pericardial enhancement (arrows), pleural effusions, and bilateral basal dystelectasis due to acute heart failure.
Course and prognosis
Usually has a good prognosis ● Recurrent pericarditis in 10–15% of cases ● Pericardial tamponade with acute heart failure is a rare but life-threatening complication.

What does the clinician want to know?
Pericardial effusion and thickening ● Inflammatory pericardial changes (MRI) ● Impairment of cardiac function.

Differential Diagnosis

Cardiac causes
– Acute coronary syndrome
– Myocardial infarction
– Myocarditis

Extracardiac causes
– Aortic dissection
– Pulmonary embolism
– Thoracic trauma

Chronic stage
– Constrictive pericarditis
– RCM

Tips and Pitfalls
Consider pericarditis in the DD of acute chest pain in patients who have a possibly corresponding history. The diagnosis should be established early by the clinical and laboratory findings and ECG. The initial workup should include echocardiography (pericardial effusion).

Selected References
Taylor AM, Dymarkowski S, Verbeken EK, Bogaert J. Detection of pericardial inflammation with late-enhancement cardiac magnetic resonance imaging: initial results. Eur Radiol. 2006; 16: 569–574
Definition

- Epidemiology
  Seven to nine percent of all congenital cardiac anomalies. More common in males than in females (2:1). Increased incidence in Turner syndrome.
- Etiology, pathophysiology, pathogenesis
  Underdevelopment of the left heart. No impairment of fetal circulation. Severe obstruction of the LV and LVOT. Oxygenated blood from pulmonary veins enters the right atrium through the foramen ovale. Dilatation of the right heart and pulmonary arteries. Systemic perfusion occurs through the PDA.

Imaging Signs

- Modality of choice
  Echocardiography. MRI and invasive studies for postoperative care (e.g., after the Fontan procedure).
- Chest radiograph findings
  Cardiomegaly. Increased pulmonary venous markings. Possible congestion with interstitial edema. Narrow mediastinum.
- Echocardiographic findings
  Decreased aortic diameter (< 5 mm). Small LV. Dilatation of the right heart and pulmonary arteries. PDA. Duplex scan shows left-to-right shunt through the PFO. Echocardiography can be used to assess pressure relationships.
- CT and MRI findings
  Used mainly for postoperative evaluations. Patency of aortopulmonary (Bla-lock-Taussig) shunt and cavopulmonary (Glenn) shunt. Pulmonary arterial anatomy. MR flowmetry for evaluation of cardiac and shunt function.
- Invasive diagnostic procedures
  Can be used to detect coronary anomalies. Used mainly in postoperative follow-up. Flow visualization in the hypoplastic ascending aorta. Visualization of the connection of the pulmonary arteries through the PDA. Determination of postoperative pressure relationships.

Clinical Aspects

- Typical presentation
- Treatment options
  Prostaglandin E₁ to maintain ductal patency. With an interatrial defect small enough to maintain a pressure gradient, the ASD can be expanded by balloon dilatation (Rashkind atrioseptostomy). Palliative surgical intervention by the Norwood procedure. Heart transplantation is advocated at some centers.
- Course and prognosis
  Untreated newborns will die in a matter of days or weeks. Otherwise, the prognosis depends on the course and complications of the Fontan procedure.
Fig. 9.30  Diagrammatic illustration of HLHS, characterized by hypoplasia of the LA, LV, aortic valve, and ascending aorta (type I). Blood is oxygenated by mixing in the RA with a left-to-right shunt through the ASD. Systemic blood flow relies on a PDA.

Fig. 9.31  MR image in a 6-month-old boy with HLHS. T1-weighted TSE sequence in an axial plane demonstrates a hypoplastic LV (arrow), an ASD (arrowheads), and a single, hypertrophic RV.
What does the clinician want to know?

Degree of LV and aortic hypoplasia • Size of the PDA and ASD • Ventricular function • Tricuspid insufficiency • Coronary anomalies.

Differential Diagnosis

- Aortic stenosis, coarctation of the aorta, interrupted aortic arch
- Cardiomyopathy
  - LV pressure overload in a normally developed heart
  - Generally enlarged heart with normal morphology
  - Myocardial dysfunction
- Arteriovenous malformation
  - Morphologically normal heart with volume overload of all chambers

Tips and Pitfalls

If a diagnosis is not made immediately after birth, HLHS should be suspected in infants who show progressive cyanosis and rapid clinical deterioration • Echocardiography should be scheduled as soon as possible.

Selected References


Fig. 9.32a, b Same patient as in Fig. 9.31. Cine GRE sequence in an oblique coronal plane (a) and oblique sagittal plane (b). Note the hypertrophic single RV (arrow) and the functionally arterialized pulmonary artery (arrowhead) with a hypoplastic aorta. Blood is directed to the lungs through a Glenn or Fontan shunt (not shown).
Definition

Classification

Cross-sectional modalities for cardiac imaging utilize standard views, which are applied in a similar manner in echocardiography, MRI, and ECG-synchronized multidetector CT.

The standard views used are:
- Long-axis view of the RV or LV parallel to the interventricular septum.
- Four-chamber view.
- Short-axis view.
- RVOT and LVOT (so-called “three-chamber view”).

Fig. 11.4  T1-weighted MR image, TSE sequence. The line represents a longitudinal section through the left ventricle.
**Fig. 11.5** Anatomy of the left ventricle (from Schünke M et al. *Thieme Atlas of Anatomy. Neck and Internal Organs.* Stuttgart: Thieme; 2006).

**Fig. 11.6** Long-axis view of the left heart in a plane parallel to the septum. MR image, SSFP sequence in diastole.
* Left pulmonary artery
+ Aortic arch with origin of the left carotid artery
LA Left atrium
x Mitral valve
LV Left ventricle