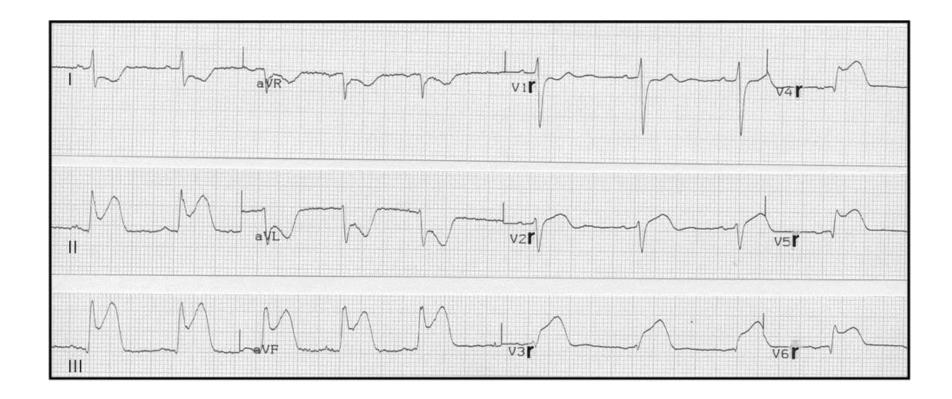
Acute heart failure

Clinical Case Scenario

• 60 –year old patient, with medical history of arterial hypertension on beta-blockers, heavy smoker, otherwise without comorbidities. In the morning he felt sudden retrosternal pressure and pain with propagation to the neck. Ambulance was called, 12-lead ecg was obtained.



• Immediately applied 250 mg ASA i.v., 7500 IU UFH i.v. and 4 ml (0,2 mg) of fentanyl i.v. for pain control. Patient immediately referred to the cathlab.

Antiplatelet therapy

Aspirin

- 162- to 325-mg load before procedure
- 81- to 325-mg daily maintenance dose (indefinite)*
- 81 mg daily is the preferred maintenance dose*

P2Y,, inhibitors

Loading doses

- . Clopidogrel: 600 mg as early as possible or at time of PCI
- Prasugrel: 60 mg as early as possible or at time of PCI
- Ticagrelor: 180 mg as early as possible or at time of PCI

Maintenance doses and duration of therapy

DES placed: Continue therapy for 1 y with:

- · Clopidogrel: 75 mg daily
- · Prasugrel: 10 mg daily
- Ticagrelor: 90 mg twice a day*

BMS† placed: Continue therapy for 1 y with:

- Clopidogrel: 75 mg daily
- Prasugrel: 10 mg daily
- Ticagrelor: 90 mg twice a day*

DES placed:

- Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 y
- Patients with STEMI with prior stroke or TIA: prasugrel

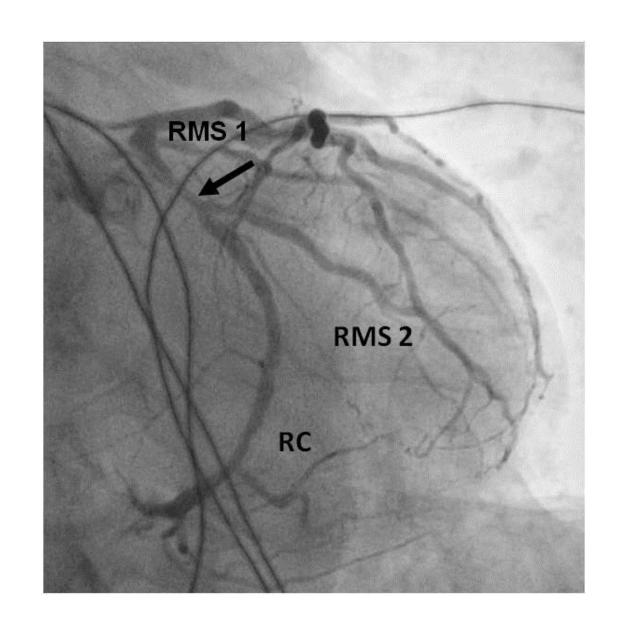
IV GP IIb/IIIa receptor antagonists in conjunction with UFH or bivalirudin in selected patients

- Abciximab: 0.25-mg/kg IV bolus, then 0.125 mcg/kg/min (maximum 10 mcg/min)
- Tirofiban: (high-bolus dose): 25-mcg/kg IV bolus, then 0.15 mcg/kg/min
 - In patients with CrCl <30 mL/min, reduce infusion by 50%
- Eptifibatide: (double bolus): 180-mcg/kg IV bolus, then 2 mcg/kg/min; a second 180-mcg/kg bolus is administered 10 min after the first bolus
 - In patients with CrCl ≤50 mL/min, reduce infusion by 50%
 - Avoid in patients on hemodialysis
- · Pre-catheterization laboratory administration of IV GP IIb/IIIa receptor antagonist
- Intracoronary abciximab 0.25-mg/kg bolus

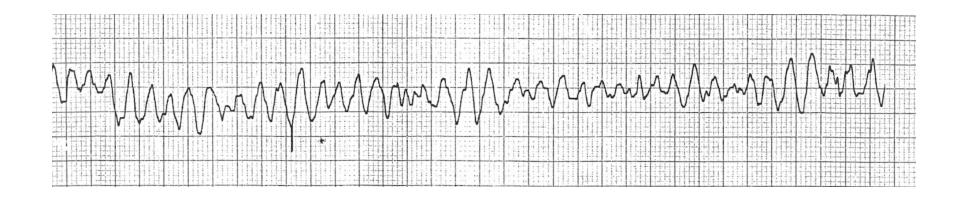
Anticoagulant therapy

UFH:

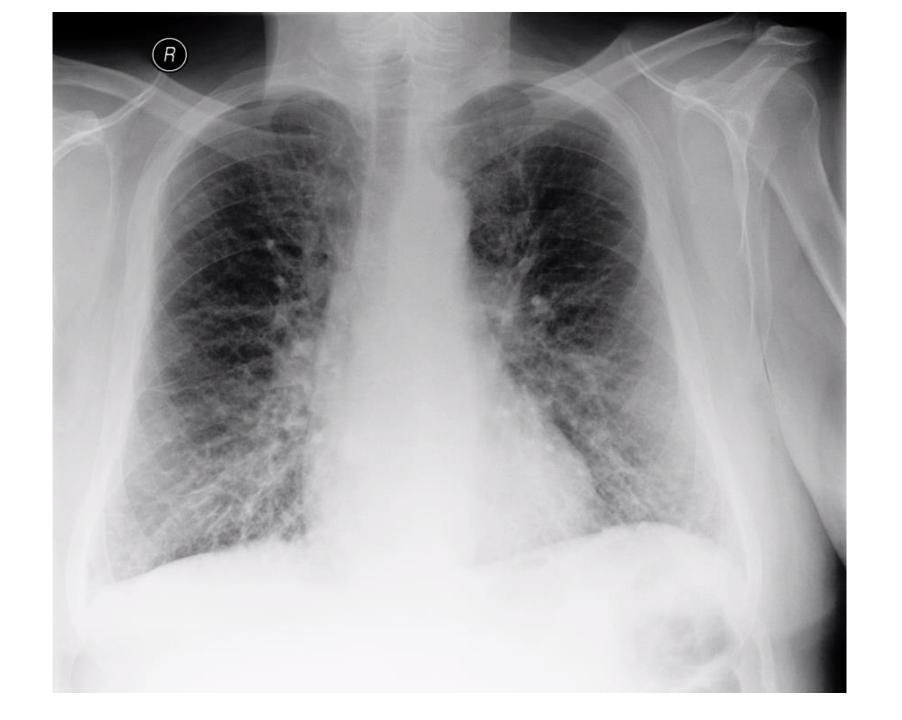
- . With GP llb/llla receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT\$
- . With no GP IIb/IIIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§



During coronarography:



- 2 biphasic 200 J discharges applied, sinus rhythm with palpable pulses restored. Afterwards consciousness regained.
- After 2 days the patient was hemodynamically stable, physiotherapy was started. Then he was transferred to a ward. The day before discharge cough with mild elevation of inflammatory markers was observed.



- According to a pulmonary consultation antibiotics were suggested. Otherwise he was deemed capable of discharge with subsequent visits at a local pulmonary physician. Medication was prescribed:
- Anopyrin 100mg 0-1-0

Clopidogrel 75mg 1-0-0

Concor COR 5mg 1-0-0

Prestarium NEO 5mg 1/2-0-0

Torvacard 40mg 0-0-1

Metformin 500mg 0-0-1

Euphylin 200mg 1-0-0

Klacid 500mg á 12 hod for 14 days

Biopron 2-0-0

- 3 days after discharge progressive increase of dyspnea. After another 2 days ambulance was called. At the scene respirátory failure was apparent, intubation was performed and the patient was transferred to the ICU.
- Ventilation: fully controlled, FiO2 0,9, Vt 450 ml, RR 25/min, I:E 1:1, PEEP 14, PIP 28
- Circulation: norepinephrine infusion at a rate of 1,5 mg/h

```
pH 7.27
pCO2 7.2
pO2 10.6
HCO3 24.3
BD- -3.3
sO2 0.934
```

Urea 8.8 mmol/l

Kreat. 122 umol/l

Na 140 mmol/l

K 6.5 mmol/l

Cl 107 mmol/l

Ca 2.08 mmol/l

Bi-celk. 6.9 umol/l

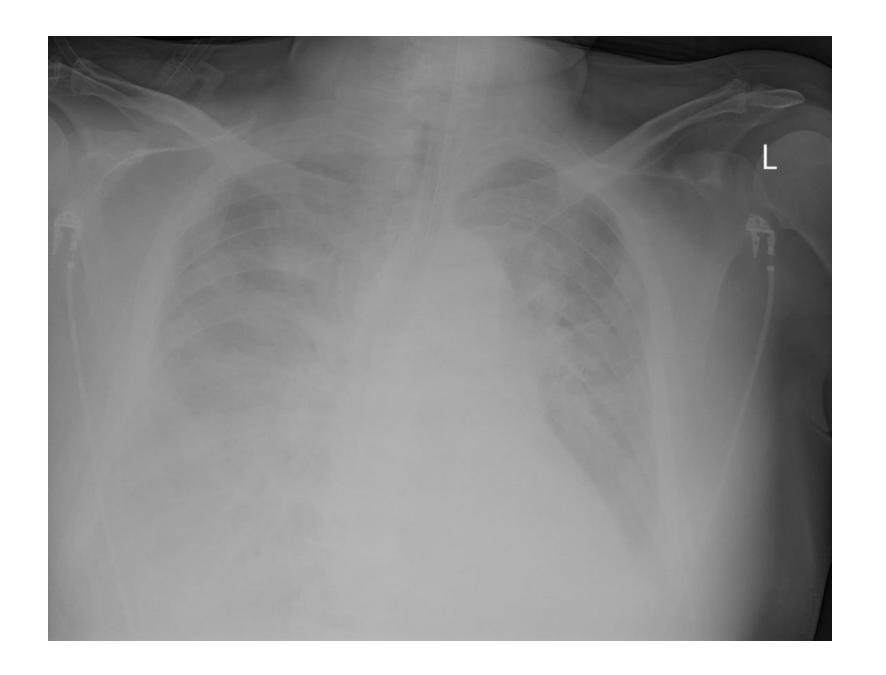
AMS 0.87 ukat/l

CB 60.8 g/l

Albumin 32.3 g/l

Glukóza 10.7 mmol/l

CRP 29.6 mg/l

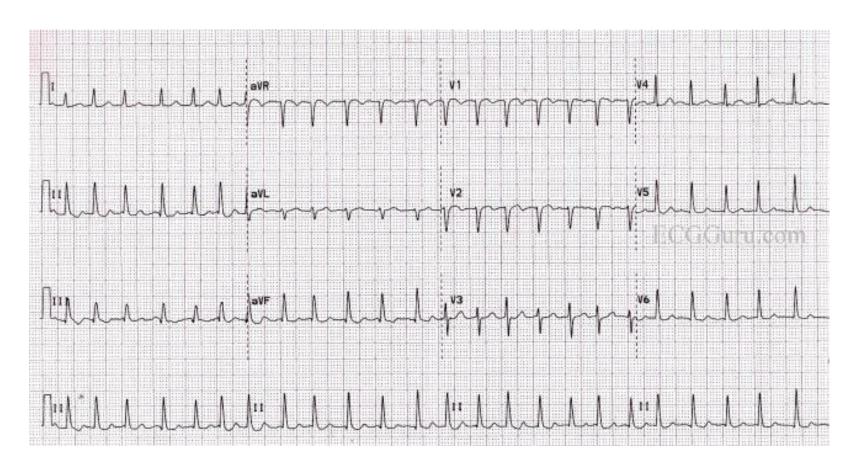


• Empirical ATB treatment was started (cefotaxime, claritromycine, oseltamivir), massive fluidothorax evacuated bilaterally.

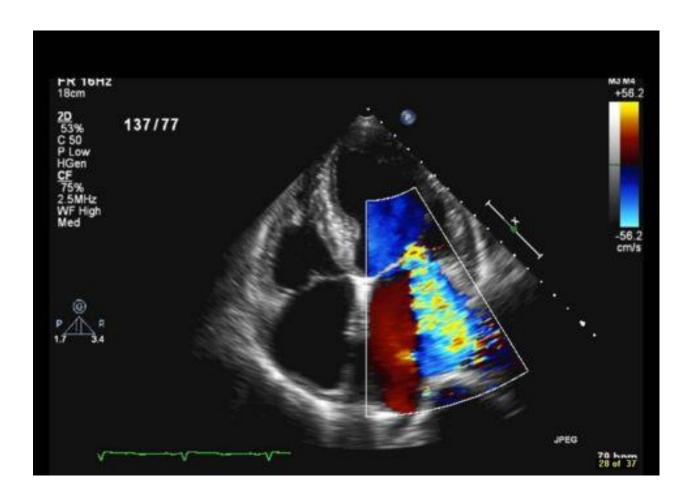
TProtein 24.6 g/l

TAlbumin 10.7 g/l

Several hours after admission:



- Amiodaron 300 mg with furher 900 mg/day applied, eventually very slow stabilisation of circulation, mild decrease of NE dose, very slight respiratory improvement.
- Sputum negative for bacteria and viruses.



- Echocardiographically Mi reg. 2-3+, EF of LV 60%.
- According to TEE partial rupture of chordae tendineae of posterior leaflet.
- Situation was reevaluated, acute heart failure with pulmonary oedema due to acute mitral regurgitation was established. The patient was referred to cardiothoracic surgery for Mi valve replacement.

Heart failure – pathophysiology:

Loss of myocardial contractility

- Partial, e.g. coronary artery disease
- Generalised, e.g. dilated cardiomyopathy, myocarditis

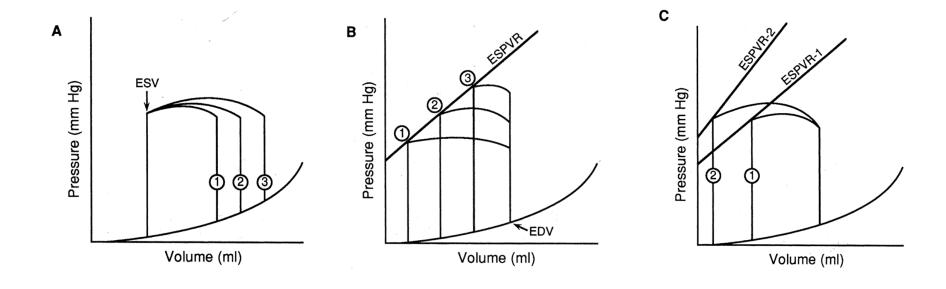
Impediments to cardiac filling and emptying

- Pressure, e.g. hypertension
- Volume, e.g. excessive intravascular volume
- Valvular disease
- Pericardial disease
- Restrictive disease

Abnormal organisation or signalling of cardiac contraction

- Tachyarrhythmias
- Bradyarrhythmias

Pressure – volume curve



Cathegories of heart failure

- Acute x chronic
- Right-sided x left-sided, bilateral
- Backward x forward (with low cardiac output)
- Systolic x diastolic

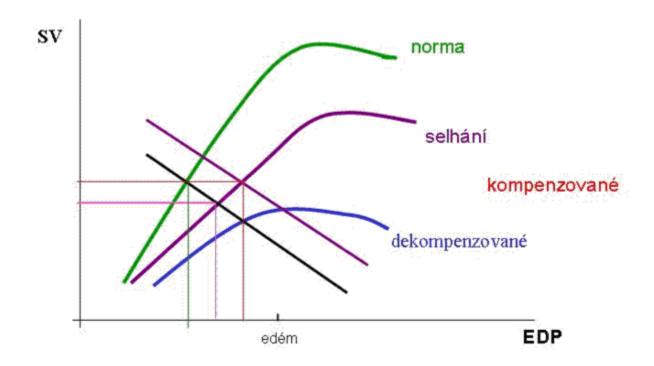
Diagnosis and evaluation

- History
- ECG
- Chest X-ray
- Laboratory
- Cardiac enzymes
- Natriuretic peptides
- Echocardiography

Principles of therapy

- Elimination of the precipitating factor revascularisation, arrhytmia management, valve replacement...
- Decrease of myocardial O₂ consumption beta-blockers, antipyretics, mech. ventilation
- Increase of O₂ delivery to the tissues oxygen, vasodilators, optimalisation of preload, decrease of afterload

Frank – Starling curve

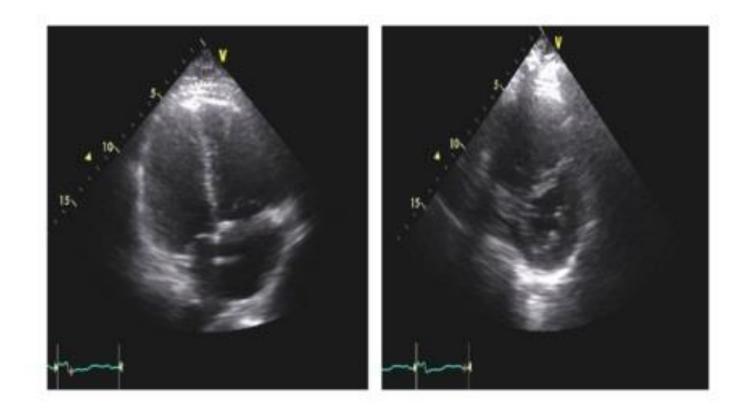


Acute congestive left-heart failure

- Pulmonary oedema
- Diastolic failure, hypertension crisis in many cases
- Oxygen
- Diuretic (furosemid 20 125 mg i.v.)
- Vasodilators (nitroglycerin sublingually, ISDN i.v.)
- Opioid (for example morphin 2-5 mg i.v.)
- NIV, invasive MV

Acute right-heart failure

- Acute increase of RV aterload
- Decreasing the afterload of RV (sildenafil, prostacycline, inhalation NO)
- Increasing afterload of LV (vasopressor)
- Reducing preload of RV diuretic



Cardiogenic shock

- Systolic pressure < 90 mm Hg for at least 30 minutes or need of vasopressors
- Congestion in pulmonary circulation or elevated filling pressures of LV
- Signs of organ hypoperfusion:
 - mental alteration
 - oliguria
 - serum lactate elevation
 - capillary refill time above 2 seconds, cyanosis

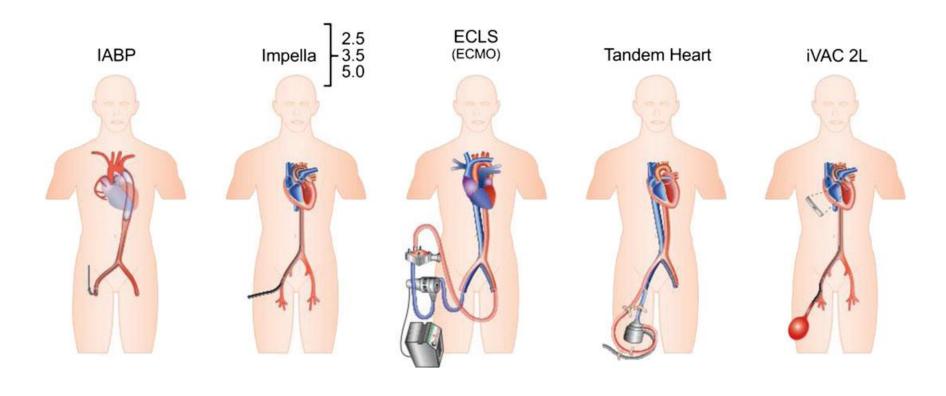
Causes

- Acute myocardial infarction
- Acute mitral regurgitation
- Ventricular septum defect
- Free ventricular wall rupture
- Acute myokarditis
- Arrhythmias

Support therapy

- Fluids
- Vasopressors (noradrenaline x dopamine)
- Inotropes (dobutamine, PDE inhibitors, levosimendan)
- Organ support (CRRT...)

Mechanical circulatory support



Thanks for Your attention