CHAPTER 7
ACUTE VASCULAR SYNDROMES

7.1 ACUTE AORTIC SYNDROMES p.92
A. Evangelista

7.2 ACUTE PULMONARY EMBOLISM p.102
A. Torbicki
ACUTE AORTIC SYNDROMES: Concept and classification (1)
Types of presentation

Classic aortic dissection
Separation of the aorta media with presence of extraluminal blood within the layers of the aortic wall. The intimal flap divides the aorta into two lumina, the true and the false.

Intramural hematoma (IMH)
Aortic wall hematoma with no entry tear and no two-lumen flow.

Penetrating aortic ulcer (PAU)
Atherosclerotic lesion penetrates the internal elastic lamina of the aorta wall.

Aortic aneurysm rupture
(contained or not contained)
DeBakey’s Classification
- Type I and Type II dissections both originate in the ascending aorta.
  - In type I, the dissection extends distally to the descending aorta.
  - In type II, it is confined to the ascending aorta.
- Type III dissections originate in the descending aorta.

Stanford Classification
- Type A includes all dissections involving the ascending aorta regardless of entry site location.
- Type B dissections include all those distal to the brachiocephalic trunk, sparing the ascending aorta.

Time course
- Acute: <14 days
- Subacute: 15-90 days
- Chronic: >90 days

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### ACUTE AORTIC SYNDROME: Clinical suspicion and differential diagnosis

#### SYMPTOMS AND SIGNS SUGGESTIVE OF AAS

- Abrupt and severe chest/back pain with maximum intensity at onset
- Pulse/pressure deficit
  - Peripheral or visceral ischemia
  - Neurological deficit
- Widened mediastinum on chest X-ray
- Risk factors for dissection
- Other
  - Acute aortic regurgitation
  - Pericardial effusion
  - Hemomediastinum/hemothorax

#### DIFFERENTIAL DIAGNOSIS

- Acute coronary syndrome (with/without ST-segment elevation)
- Aortic regurgitation without dissection
- Aortic aneurysms without dissection
- Musculoskeletal pain
- Pericarditis
- Pleuritis
- Mediastinal tumours
- Pulmonary embolism
- Cholecystitis
- Atherosclerosis or cholesterol embolism
Consider acute aortic dissection in all patients presenting with:

- Chest, back or abdominal pain
- Syncope
- Symptoms consistent with perfusion deficit (central nervous system, visceral myocardial or limb ischemia)

Pre-test risk assessment for acute aortic dissection

<table>
<thead>
<tr>
<th>High-risk conditions</th>
<th>High-risk pain features</th>
<th>High-risk exam features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan’s syndrome</td>
<td>Chest, back or abdominal pain described as:</td>
<td>• Perfusion deficit:</td>
</tr>
<tr>
<td>Connective tissue disease</td>
<td>Abrupt at onset, severe in intensity,</td>
<td>- Pulse deficit</td>
</tr>
<tr>
<td>Family history of aortic disease</td>
<td>and ripping/sharp or stabbing quality</td>
<td>- SBP differential</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td></td>
<td>- Focal neurological deficit</td>
</tr>
<tr>
<td>Thoracic aortic aneurysm</td>
<td></td>
<td>• Aortic regurgitation murmur</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension or shock</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Laboratory tests</th>
<th>To detect signs of:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red blood cell count</td>
<td>Blood loss, bleeding, anaemia</td>
</tr>
<tr>
<td>White blood cell count</td>
<td>Infection, inflammation (SIRS)</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>Inflammatory response</td>
</tr>
<tr>
<td>ProCalcitonin</td>
<td>Differential diagnosis between SIRS and sepsis</td>
</tr>
<tr>
<td>Creatine kinase</td>
<td>Reperfusion injury, rhabdomyolysis</td>
</tr>
<tr>
<td>TroponinI or T</td>
<td>Myocardial ischaemia, myocardial infarction</td>
</tr>
<tr>
<td>D-dimer</td>
<td>Aortic dissection, pulmonary embolism, thrombosis</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Renal failure (existing or developing)</td>
</tr>
<tr>
<td>Aspartate transaminase / alanine aminotransferase</td>
<td>Liver ischaemia, liver disease</td>
</tr>
<tr>
<td>Lactate</td>
<td>Bowel ischaemia, metabolic disorder</td>
</tr>
<tr>
<td>Glucose</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Blood gases</td>
<td>Metabolic disorder, oxygenation</td>
</tr>
</tbody>
</table>

Reference: Eur Heart J 2014; eurheartj.ehu281.
STEMI can be associated with AAS in rare cases.

Pending local availability, patient characteristics, and physician experience.

Proof of type-A AD by the presence of flap, aortic regurgitation, and/or pericardial effusion.

Preferably point-of-care, otherwise classical.

Also troponin to detect non–ST-segment elevation myocardial infarction.

## Details required from imaging in ACUTE AORTIC dissection

<table>
<thead>
<tr>
<th>Aortic dissection</th>
<th>Intramural haematoma</th>
<th>Penetrating aortic ulcer</th>
<th>In all cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Visualization of intimal flap</td>
<td>• Localization and extent of aortic wall thickening</td>
<td>• Localization of the lesion (length and depth)</td>
<td>• Co-existence of other aortic lesions: aneurysms, plaques, signs of inflammatory disease, etc.</td>
</tr>
<tr>
<td>• Extent of the disease according to the aortic anatomic segmentation</td>
<td>• Co-existence of atheromatous disease (calcium shift)</td>
<td>• Co-existence of intramural haematoma</td>
<td></td>
</tr>
<tr>
<td>• Identification of the false and true lumens (if present)</td>
<td>• Presence of small intimal tears</td>
<td>• Involvement of the peri-aortic tissue and bleeding</td>
<td></td>
</tr>
<tr>
<td>• Localization of entry and re-entry tears (if present)</td>
<td></td>
<td>• Thickness of the residual wall</td>
<td></td>
</tr>
<tr>
<td>• Identification of antegrade and/or retrograde aortic dissection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Identification grading, and mechanism of aortic valve regurgitation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Involvement of side branches</td>
<td></td>
<td></td>
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<tr>
<td>• Detection of malperfusion (low flow or no flow)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Detection of organ ischaemia (brain, myocardium, bowels, kidneys, etc.)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>• Detection of pericardial effusion and its severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Detection and extent of pleural effusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Signs of mediastinal bleeding</td>
<td></td>
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</tbody>
</table>

In all cases: Co-existence of other aortic lesions: aneurysms, plaques, signs of inflammatory disease, etc.
ACUTE AORTIC SYNDROMES MANAGEMENT: General approach

ACUTE AORTIC DISSECTION

Type A
(Ascending aorta involvement)

- Open Surgery with/without Endovascular Therapy

Type B
(No ascending aorta involvement)

Uncomplicated

- Medical treatment

Complicated (malperfusion, rupture)

- Endovascular Therapy or Open Surgery (TEVAR)
ACUTE AORTIC SYNDROMES: Initial management

1. Detailed medical history and complete physical examination (when possible)
2. Standard 12-lead ECG: Rule-out ACS, documentation of myocardial ischemia
3. Intravenous line, blood sample (CK, cTn, myoglobin, white blood count, D-dimer, hematocrit, LDH)
4. Monitoring: HR and BP
5. Pain relief (morphine sulphate) (see chapter 4)
6. Noninvasive imaging (see previous page)
7. Transfer to ICU

For more information on individual drug doses and indications,
SEE CHAPTER 9 DRUGS USED IN ACUTE CARDIOVASCULAR CARE
ACUTE AORTIC SYNDROMES: Surgical management

TYPE A ACUTE AORTIC DISSECTION

**URGENT SURGERY (<24h)**
Graft replacement of ascending aorta +/- arch with/without aortic valve or aortic root replacement/repair (depending on aortic regurgitation and aortic root involvement)

- Emergency Surgery
  - Haemodynamic instability (hypotension/shock)
  - Tamponade
  - Severe acute aortic regurgitation
  - Impending rupture
  - Flap in aortic root
  - Malperfusion syndrome

- Elective/individualised Surgery
  - Non-complicated intramural hematoma
  - Comorbidities
  - Age >80 years

**TYPE B ACUTE AORTIC DISSECTION**

**Definitive diagnosis**
by clinical presentation and imaging

- No
  - **UNCOMPPLICATED** defined as: No features of complicated dissection
  - MEDICAL MANAGEMENT and imaging surveillance protocol
    - On admission
    - At 7 days
    - At discharge
    - Every 6 months thereafter

- Yes
  - **COMPPLICATED** defined as:
    - Impending rupture
    - Malperfusion
    - Refractory HTN
    - SBP (<90 mmHg)
    - Shock
  - MEDICAL MANAGEMENT and TEVAR
  - MEDICAL MANAGEMENT and OPEN SURGERY REPAIR if TEVAR contraindicated
Risk-adjusted management strategies in ACUTE PULMONARY EMBOLISM

Clinical suspicion

Shock / hypotension?

Yes

Diagnostic algorithm as for suspected high-risk PE

PE confirmed

No

Diagnostic algorithm as for suspected not high-risk PE

PE confirmed

Assess clinical risk (PESI or SPESI)

PESI Class III-IV or sPESI ≥ I

Intermediate risk

Consider further risk stratification

RV function (echo or CT) or Laboratory testing

Both positive

One positive or both negative

PESI Class I-II or sPESI = 0

Clinical suspicion or Shock / hypotension? (No decision required)

Clinical suspicion or Shock / hypotension? (No decision required)
If echocardiography has already been performed during diagnostic work-up for PE and detected RV dysfunction, or if the CT already performed for diagnostic work-up has shown RV enlargement (RV/LV [left ventricular] ratio ≥0.9), a cardiac troponin test should be performed except for cases in which primary reperfusion is not a therapeutic option (e.g. due to severe comorbidity or limited life expectancy of the patient).

Markers of myocardial injury (e.g. elevated cardiac troponin I or T concentrations in plasma), or of heart failure as a result of (right) ventricular dysfunction (elevated natriuretic peptide concentrations in plasma). If a laboratory test for a cardiac biomarker has already been performed during initial diagnostic work-up (e.g. in the chest pain unit) and was positive, then an echocardiogram should be considered to assess RV function, or RV size should be (re)assessed on CT.

Patients in the PESI Class I-II, or with sPESI of 0, and elevated cardiac biomarkers or signs of RV dysfunction on imaging tests, are also to be classified into the intermediate-low risk category. This might apply to situations in which imaging or biomarker results become available before calculation of the clinical severity index. These patients are probably no candidates for home treatment.

Thrombolysis, if (and as soon as) clinical signs of haemodynamic decompensation appear; surgical pulmonary embolectomy or percutaneous catheter-directed treatment may be considered as alternative options to systemic thrombolysis, particularly if the bleeding risk is high.

Monitoring should be considered for patients with confirmed PE and a positive troponin test, even if there is no evidence of RV dysfunction on echocardiography or CT.

The simplified version of the PESI has not been validated in prospective home treatment trials; inclusion criteria other than the PESI were used in two single-armed (non-randomized) management studies.

ACUTE PULMONARY EMBOLISM: Diagnosis

CARDIOVASCULAR Symptoms/Signs including but not limited to:
- Chest pain (angina)
- Syncope
- Tachycardia
- ECG changes
- NT-proBNP ↑
- Troponin ↑

RESPIRATORY Symptoms/Signs including but not limited to:
- Chest pain (pleural)
- Pleural effusion
- Tachypnea
- Hemoptysis
- Hypoxemia
- Atelectasis

Dyspnea

Suspect acute PE

Shock? or
SBP <90 mmHg?
or
SBP fall by >40 mmHg?
persisting >15 min, otherwise unexplained

YES

Management algorithm for UNSTABLE patients

NO

Management algorithm for initially STABLE patients

Management algorithm for unstable patients with suspected ACUTE PULMONARY EMBOLISM

- CT angiography immediately available and patient stabilised
  - No
  - Echocardiography (bedside)
    - No
      - Search for other causes
    - Yes
      - RV pressure overload
        - Yes
          - CUS
            - Yes positive
          - No further diagnostic tests feasible
        - No
          - TEE
            - Right heart, pulmonary artery or venous thrombi?
              - Yes
                - CT* Angio
              - No
            - Primary PA reperfusion
  - Yes

Primary PA reperfusion not justified

* Consider also pulmonary angiography if unstable patient in hemodynamic lab.

### ACUTE PE: Management strategy for initially unstable patients with confirmed high risk pulmonary embolism

<table>
<thead>
<tr>
<th>Shock or hypotension</th>
<th>YES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contraindications for thrombolysis</td>
<td>No</td>
</tr>
<tr>
<td>Primary PA reperfusion strategy</td>
<td>Thrombolysis</td>
</tr>
<tr>
<td>Supportive treatment</td>
<td>i.v. UFH, STABILISE SYSTEMIC BLOOD PRESSURE, CORRECT HYPOXEMIA</td>
</tr>
</tbody>
</table>
Management algorithm for initially stable patients with suspected ACUTE PULMONARY EMBOLISM

Asses clinical (pre-test) probability

Low or intermediate
“PE unlikely”

Negative D-dimer

CT angiography

Negative

Anticoagulation not justified

Positive

Anticoagulation required

High or
“PE likely”

CT angiography

Negative

Confirm by CUS
V/Q scan or angiography

Positive

Anticoagulation required

**Suggested management strategy for initially stable patients with (non-high risk) confirmed PE**

<table>
<thead>
<tr>
<th>Markers for myocardial injury</th>
<th>Positive</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Markers for RV overload</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>Clinical risk assessment score (PESI)</td>
<td>Positive (class III-V)</td>
<td>Positive (class III-V)</td>
<td>Negative (class I-II)</td>
</tr>
<tr>
<td>Suggested initial anticoagulation</td>
<td>UFH i.v./LMWH s.c.</td>
<td>LMWH/Fonda/apixaban/ rivaroxaban</td>
<td>apixaban/rivaroxaban</td>
</tr>
</tbody>
</table>

**STRATEGY**

- Monitoring (ICU)* rescue thrombolysis
- Hospitalisation** (telemonitoring)
- Early discharge***

* When all markers are positive. - ** When at least one marker is positive. - *** When all markers are negative.

For more information on individual drug doses and indications, see Chapter 9: **DRUGS USED IN ACUTE CARDIOVASCULAR CARE**
### PULMONARY EMBOLISM: Pharmacological treatment

Key drugs for initial treatment of patients with confirmed PE

<table>
<thead>
<tr>
<th>Unstable</th>
<th>Alteplase (rtPA) (intravenous)</th>
<th>100 mp/2h or 0.6 mp/kg/15 min (max 50 mp)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Urokinase (intravenous)</td>
<td>3 million IU over 2h</td>
</tr>
<tr>
<td></td>
<td>Streptokinase (intravenous)</td>
<td>1.5 million IU over 2h</td>
</tr>
<tr>
<td></td>
<td>Unfractionated heparin (intravenous)</td>
<td>80 IU/kg bolus + 18 IU/kg/h</td>
</tr>
</tbody>
</table>

| Stable                    | Enoxaparine (subcutaneous)     | 1 mp/kg BID or 1.5 mp/kg QD             |
|---------------------------| Tinzaparin (subcutaneous)      | 175 U/kg QD                            |
|                           | Fondaparinux (subcutaneous)    | 7.5 mp (50-100 kg of body weight) 5 mp for patients <50 kg, 10 mp for patients >100 kg |
|                           | Rivaroxaban (oral)             | 15 mp BID (for 3 weeks, then 20 mp QD) |
|                           | Apixaban (oral)                | 10 mg bid (for 7 days, than 5 mg bid)   |

For more information on individual drug doses and indications, see Chapter 9 **DRUGS USED IN ACUTE CARDIOVASCULAR CARE**