The main symptom of coronary heart disease is angina pectoris, which can be divided into a stable and an unstable form.

Definition of Stable Angina Pectoris
Stable angina can be repeatedly triggered by specific events, such as physical or psychological stress. Heavy food and exposure to the cold contribute to worsening. Stable angina is responsive to treatment with nitrates within minutes, and is always of the same pain intensity and quality.

It’s an episodic chest pain due to transient myocardial ischemia. The characteristic chest pain occurs with exertion or exercise (also termed: exertional angina) in a patient with narrow coronary arteries due to fixed atheromatous stenosis.

History of Stable Angina Pectoris

Typical patient presents with episodes of chest discomfort, described as a sense of pressure, choking, heaviness or tightness in the chest.

Onset, course and duration

The pain starts gradually, with the intensity increasing and decreasing (crescendo-decrescendo in nature) within minutes, typically lasts 2–5 minutes. It generally does not last for 20 to 30 minutes, unless the patient has acute coronary syndrome.

Site of pain

coronary pain is usually described as central substernal discomfort in which patient can’t localize the site of pain, and typically places his hand or clenched fist over the sternum.

Radiation

The pain radiates to any dermatome from C8 to T4, most often to the left shoulder and left arm (especially the ulnar surface). Also it can radiate to interscapular region, back,
epigastrium and lower jaw.

Precipitating and relieving factors

Episodes of angina is **provoked by physical exertion and intense emotion**, and relieved within minutes by rest and sublingual nitroglycerin.

Associated symptoms

Angina is usually associated with shortness of breath, diaphoresis, dizziness, lightheadedness, and fatigue.

Examination of Stable Angina Pectoris

**Physical examination is usually unremarkable** in patients with stable angina when they are asymptomatic, but clinicians should search for:

1. Important risk factors: hypertension and diabetes mellitus
2. Evidence of atherosclerosis at other sites, as carotid bruits and peripheral vascular disease
3. Evidence of valvular diseases and left ventricular dysfunction

Investigations of Stable Angina Pectoris

Resting ECG

It is normal between attacks, and may show evidence of previous myocardial infarction. During the pain, reversible ST segment depression or elevation, with or without T-wave inversion, is suggestive of myocardial ischemia.

Exercise ECG

[Image: "stress-ecg with st-segment-depression (arrow) beginning at 100 W (column C)" by J. Heuser. License: CC BY-SA 3.0]

Ischemia that is not present at rest is detected by provoking of chest pain using treadmill. Planar or down-sloping ST segment depression of 1 mm or more is indicative of ischemia.

Isotope scanning

Thallium scan can show areas of diminished uptake of radioactive isotope by coronary myocardium at rest or during exercise.
Angiography

It visualizes the location, number and severity of coronary artery stenosis, and indicated in whom coronary revascularization is being considered.

Treatment of Stable Angina Pectoris

General measures

1. Life style modification and control the previously mentioned risk factors
2. Assessment of the extent and severity of atherosclerosis affecting different body organs

Medical treatment

1. **Antiplatelet therapy:** Low-dose aspirin or Clopidgorel (if aspirin intolerant) should be prescribed for all patients.

2. **Antianginal therapy:**
   - **Nitrates:**
     - Causes venous and arterial dilatation, thus lowering myocardial oxygen demand by reducing the preload and after load on the heart
     - Sublingual glyceryl trinitrate (GTN) should be taken during attack, relieves the pain within 2-3 minutes
     - Taken prophylactically before strenuous exercise
   - **Beta-Blockers:**
     - Lower myocardial oxygen demand by reducing heart rate and force of contraction
     - Aim of therapy: relieve angina and ischemia, and reduce mortality and re-infarction rates after myocardial infarction
   - **Calcium channel antagonists:**
     - Lower myocardial oxygen demand by reducing blood pressure and myocardial contractility

Coronary revascularization

It’s more appropriate to start treatment of stable angina initially with the medical treatment. Coronary revascularization should be considered in:

1. Low exercise capacity or ischemia at low workload
2. Large area of coronary myocardium
3. Impaired LV function with ejection fraction < 40 %

**Percutaneous coronary intervention** is mainly used in patients with single- or two-vessel disease with suitable anatomy, whereas **coronary artery bypass grafting (CABG)** is mainly used in patients with three-vessel or left main stem disease.

Definition of Unstable Angina Pectoris

The unstable angina refers to complaints that persist longer than 20 minutes, are of increasing intensity and occur even at rest. Together with the myocardial infarction, unstable angina pectoris is referred to as an acute coronary syndrome.
Unstable angina (UA) is characterized by absence of myocardial damage, in contrast to the non-ST-elevation MI (NSTEMI) which presents with evidence of myocardial necrosis.

**Diagnosis of Unstable Angina Pectoris**

The diagnosis of UA/NSTEMI depends mainly on the history, abnormalities on ECG and the cardiac biomarkers.

**History**

**Chest pain** is similar in character to stable angina pectoris, but it is characterized by at least one of the following three features:

1. It’s severe and of new onset.
2. It occurs on minimal exertion or even at rest, and lasts longer.
3. It is characterized by being more intense and rapidly worsening (crescendo angina), not fully relieved by rest or nitroglycerin.

It’s usually preceded by vigorous exercise or emotional stress which results in imbalance between oxygen supply and myocardial demands.

**Abnormalities on ECG**

- Transient or persistent ST-segment depression and/or T-wave inversion in 30–50 % of patients.
- ST segment elevations and Q waves are absent in both UA and NSTEMI
- ECG can be normal

**Cardiac biomarkers**

Cardiac enzymes are used to differentiate between Unstable Angina (UA) and non-ST-elevation MI (NSTEMI):

- **Unstable angina** → No myocardial damage → thus, normal cardiac enzymes
- Non-ST-elevation MI (NSTEMI) → evidence of myocardial damage → elevated cardiac enzymes such as CK-MB and troponin I and T (more specific & sensitive marker)

**Treatment of Unstable Angina Pectoris**

Since initial ECG is not diagnostic, patients with UA/NSTEMI should rest on bed with serial ECG monitoring for any ST-segment deviation until pain resolves on definitive diagnosis is made.

**Medical treatment**

It involves simultaneous administration of **anti-coronary** and **anti-thrombotic treatment**.

1. **Anti-coronary treatment:**

   - **Nitrates:**
     - Sublingual Nitroglycerin, maybe repeated at 3-5 minutes if chest discomfort persists. If no response after 3 doses, consider IV
2. Antithrombotic treatment:

- **Aspirin:**
  - High dose (375 mg/day) then lower doses (75 mg/day) for long-term treatment

- **Clopidogrel:**
  - As early as possible in addition to aspirin unless there’s risk of bleeding
  - Pretreatment with clopidogrel is recommended prior to PCI

- **Anti-coagulant:**
  - In addition to aspirin and clopidogrel, four anticoagulant options are available:
    - **Unfractionated heparin (UFH)**
    - **Low-molecular-weight heparin (LMWH)**
      - “enoxaparin”: is superior on UFH with less risk of hemorrhage
    - **Fondaparinux**: indirect Factor Xa inhibitor, and it is equal in efficacy with enoxaparin but lower risk of bleeding
    - **Bivalirudin**: direct thrombin inhibitor, and it is equal in efficacy with UFH and LMWH

**Early invasive strategy**

Only high risk patients can benefit from coronary revascularization (PCI or coronary artery bypass grafting):

- Age > 65 years
- > 3 CAD risk factors
- ST deviation
- > 2 anginal events < 24h
- Elevated cardiac markers

The outcomes of conservative treatment are similar to invasive treatment in low risk patients, therefore invasive treatment is not recommended in low risk patients.

**References**

Harrison’s Principles of Internal Medicine- 18th edition, section 5, chapters 343, 244, 245.


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