Acute Coronary Syndrome: Risk stratification and management

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Highlights / Hoogtepunte

- What is cardiac chest pain?
- How do ECG-findings determine the immediate management of acute coronary syndromes (ACS)?
- What immediate therapy should the GP prescribe in the event of an ACS?

- Wat is kardiale borskaspyn?
- Hoe bepaal, EKG-bevindinge die onmiddellike hantering van 'n akute koronêre sindroom (AKS)?
- Watter terapie moet die algemene praktisyn voorskryf in AKS?

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1. DEFINITIONS

Acute severe prolonged chest pain of cardiac origin that occurs during rest is termed an acute coronary syndrome. An acute coronary syndrome may present as:

i. a new phenomenon: suddenly and unpredictably; or
ii. against a background of chronic stable angina.

An acute coronary syndrome describes three clinical entities: unstable angina, non-STEMI [Non-ST-Elevation Myocardial Infarct] or a STEMI [ST-Elevated Myocardial infarct]. They are classified together as an acute coronary syndrome probably because they share a common pathophysiology and their clinical presentation identical.

2. WHAT IS A CARDIAC CHEST PAIN?

The pain is usually typically in the centre of the chest, radiates to the neck, jaw, upper arms, even lower arms or back. The pain is typically dull, constricting, choking or "heavy" and patients describe it furthermore as squeezing, crushing, burning or aching (not sharp, stabbing, pricking). Patients can use an open hand or a clenched fist when describing the pain. Chest pain like this description, especially when present for longer than 20 minutes, is very indicative of an MI. Unfortunately, other respiratory, gastroenterological or musculo-skeletal diseases may provoke chest pain like this and need to be investigated only when an acute coronary syndrome has been excluded.

Associated features

The pain of acute coronary syndrome and massive pulmonary embolism and aortic dissection may have associated features: sweating, nausea, vomiting and breathlessness.

3. PATHOPHYSIOLOGY OF ACUTE CORONARY SYNDROME

The basic underlying pathology is coronary atherosclerosis in the majority of patients with atherosclerotic plaques. Pathologic studies have demonstrated that these plaques are soft consisting of a mass of lipids and a thin fibrous cap. Plaque rupture is associated with intense inflammatory changes induced by macrophages secreting proteolytic enzymes, cytokines and activating local Angiotensin II. The culprit lesion causing the symptom is usually:

i. complex ulcerated plaque; or
ii. fissured plaque.

In both cases of plaque rupture, there is a platelet-rich thrombus, which rapidly leads to further thrombosis, which will occlude the coronary artery lumen. The lipid-rich part of the plaque is highly thrombogenic. There may also be coronary artery spasm, induced by endothelial dysfunction, which may further occlude the lumen. This is a dynamic process causing episodes of myocardial ischaemia due to an abrupt reduction in coronary blood flow. This may eventually lead to a complete occlusion of the coronary artery. Thrombus formation may lead to fragmentation and distal thrombus emboli in the coronary vessels. Acute coronary syndrome can be viewed as a thrombotic disease of the coronary vessels.

4. DIAGNOSIS

An urgent resting 12-lead electrocardiogram is the first and most important test that is needed. The ECG may stratify the patient into the following groups:

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i. STEMI [ST-segment Elevation Myocardial Infarction]

With this diagnosis, consideration must be given to either thrombolysis or primary PCI [percutaneous coronary intervention]. Urgent primary PCI is the treatment of choice where it is available.

ii. NON-STEMI [NON-ST-Elevated Myocardial Infarction]

The ST-segments may be downsloping or horizontally depressed with or without T-wave inversion. Urgent anti-ischaemic and antithrombotic therapy are required and early referral necessary.

iii. Normal ECG

In both the NON-STEMI group and the normal ECG-group further risk stratification is required.

5. RISK STRATIFICATION

Clinical risk factors

Ongoing chest pain, pain that does not settle indicates important myocardial ischaemia and increases risk. Signs of heart failure (gallop, creps in lungs) progressively increase risk. Increasing age of the patient and diabetic patients are at increased risk. Severe hypertension in the acute phase that does not properly respond to morphine should, especially if systolic BP is ³ 160 mmHg, receive intravenous beta-blockers to reduce clinical risk.

ECG risk factors

When the ST-segments are raised [STEMI] or depressed [NON-STEMI], risk is increased compared to normal ST-segments or minor T-wave inversion. Risk increases with increasing degrees of atrio-ventricular heart block and risk is high with bundle branch block that develops with anterior infarction. Primary ventricular dysrhythmias in the first 48 hours are a high risk (no evidence that prophylactic lignocaine will prevent this) and could be related to ongoing ischaemia and or remodelling.

Metabolic risk factors

Diabetes mellitus or blood glucose on admission of ³ 11.1 mmol/*l* should receive a constant intravenous infusion of insulin to reduce the high associated risk. Increases in urea or creatinine on admission also confer a higher risk.

Cardiac markers

Any elevation in cardiac markers e.g. myoglobin (early), creatine kinase, CK-MB fraction or Troponin T or I will increase risk. Even minimal elevations are associated with increase in adverse events. Other cardiac markers such as C-reactive protein and B-type of natriuretic peptides are still under investigation to define their diagnostic role in the ACS.

If any of the above is present the patient falls into the high-risk category and further invasive intervention should be considered. If all of the above are normal, then the patient has a low risk and should have an early stress ECG.

6. THERAPEUTIC MANAGEMENT OF ACS

I. Anti-ischaemic therapy

The aim of this treatment is to diminish or abort myocardial ischaemia.

Nitrates

Nitrates dilate veins (therefore reduce pre-load) and, in higher doses, dilate arterioles (therefore reduce after load) and, as a consequence of these two actions, oxygen demand of the myocardium is reduced. Nitrate-dose is titrated until symptoms of pain disappear or haemodynamic parameters, tachycardia or low blood pressure, develop. The major drawback of nitrates is tolerance.

Beta-blockers:

Beta-blockers lower heart rate, lower blood pressure and reduce myocardial contractility, thus reducing oxygen demand. They reduce early infarction mortality probably by preventing malignant ventricular dysrhythmias by preventing re-infarction and even possibly by preventing myocardial rupture.

II. Anti-thrombotic therapy

A. Anti-platelet therapy

Anti-platelet therapy is the most effective strategy and aspirin plays a critical role.

i. **An Aspirin** should be chewed and swallowed immediately. Aspirin inhibits cyclo-oxygenase in the platelets for the duration of the platelet lifespan (7-10 days) and platelets are unable to produce thromboxane A_2 and therefore unable to aggregate. Aspirin can reduce events by 50 percent as compared to placebo.

ii. **Clopidogrel** inhibits the ADP-receptor on the platelet surface rendering the platelet unable to aggregate. The use of these agents during a coronary intervention (e.g. stenting) is highly effective. Both aspirin and clopidogrel benefit patients across the range of risk.

iii. **Glycoprotein IIb/IIIa-receptor antagonist.** These agents inhibit the fibrinogen receptor on the platelet, which is the final common pathway of platelet aggregation. These agents are highly effective in intervention cardiology. They may have a place in the management of the acute coronary syndromes especially when the cardiac markers (e.g. Troponin T/I) are raised.

B. Thrombolysis:

Thrombolytic therapy should only be used for a STEMI as the final occlusion of the coronary artery is caused by a blood clot. Streptokinase 1.5 million units in a short infusion intravenous over 30-60 minutes is necessary. Alternatively, Alteplase 100 mg given over 90 minutes I.V. can be used. Newer agents are coming on the market. Absolute contraindications like uncontrolled HT, previous stroke, previous surgery, previous bleeding or allergy to streptokinase should be adhered to.

C. Anticoagulants

i. Unfractionated Heparin

Heparin exerts its antithrombotic effect by stimulating antithrombin-III, making it an indirect thrombin inhibitor. The effect of heparin is measured as a prolongation of the activated partial thromboplastin time (aPTT). The therapeutic range is an aPTT of 60-90 seconds.

ii. Low-molecular weight Heparin: [L.M.W.]

This type of heparin has anti-Xa-activity

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and is more predictable in its response, does not need aPTT-monitoring, and can be given subcutaneously. Mortality reduction is slightly better than with unfractionated heparin.

Unfractionated or LMW-heparin should be added to the treatment regime of all patients with the ACS.

III. Plaque-stabilisation therapy

Percutaneous intervention is an effective method to achieve short-term plaque stabilisation. Long-term aspirin with or without warfarin (INR 2-3], or high-dose statins, or combination of aspirin with clopidogrel, will reduce further events, probably by stabilising plaques.

IV. Secondary prevention

All patient categories should, after definitive therapy, e.g. invasive interventions on medically treated, be started on secondary prevention therapy:

- Aspirin: life-long.
- Angiotensin-converting enzyme-inhibitor.
- Beta-blocker.
- Statin: Life-long.
- No smoking: Life-long.

Dietary and physical rehabilitation, including exercise, should be instituted.

7. SUMMARY:

Rapid evaluation and risk stratification of cardiac chest pain is essential. Life-saving treatment like aspirin, beta-blockers and heparin should be used aggressively. Early referral of high-risk patients is necessary. For more detailed management, please obtain and use the South African Guidelines on the acute coronary syndrome [SAMJ 3 October 2001].❐

Please refer to the CPD Questionnaire on page 51.

References:

1. Management of acute coronary syndromes: Clinical guidelines of SAMA. *SAMJ* 3 Oct 2001; Vol 91:879-892.
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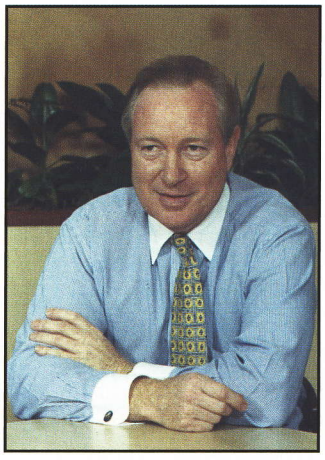

The Board of the Professional Provident Society Group is pleased to announce the appointment of Mr. Mike Jackson as chief executive of PPS Insurance.

Mr. Jackson brings 30 year's experience in all aspects of the financial services sector to the Group and will commence duties on 29 September 2003.

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