MANAGEMENT OF ACUTE CORONARY SYNDROME

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ABSTRACT

The morbidity, mortality and economic consequences of Acute Coronary Syndrome (ACS) are not mutually exclusive. Often regarded as a silent killer, the hypernym constitutes a variety of ischemic heart diseases that all too often claim the lives of those with seemingly normal biology. Conveniently sorted into three conditions, with each characterized by distinct signs and symptoms, the syndrome was once regarded as a disease of the West. With detailed research and documentation, it has been asserted that it has become an endemic disease Africa has to contend with.
This article aims to present clinical guidelines in the management of ACS to health workers and students.

Keywords: Acute Coronary Syndrome, myocardial infarction, management.

INTRODUCTION

Ischemic heart disease (IHD) is described by the Joint International Society and Federation of Cardiology task force as myocardial impairment due to an imbalance between oxygen supply and demand of the myocardium.
Acute coronary syndrome (ACS) is an umbrella term that characterizes a constellation of clinical presentations that define a spectrum of ischemic heart diseases resulting from a sudden decreased flow of blood in the coronary arteries such that a part of the myocardium has difficulty functioning effectively or undergoes necrosis. It includes ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina (UA).[1]
Ischemia to heart muscles could be regional or global with single or recurrent episodes. Acute coronary syndrome is an emergency, and management must be optimal in all patients.1

EPIDEMIOLOGY

Cardiovascular diseases cause one-third of all deaths globally, of which 7.5 million deaths result from ischemic heart disease (IHD). Acute coronary syndromes (ACS) and sudden death cause the most IHD-related deaths, leading to 1.8 million deaths annually.[2,3]
The incidence of IHD and thus ACS increases with age. This occurs on average 7–10 years earlier in men compared with women. ACS occurs far more often in men below the age of 60 years, while the majority of patients over 75 years of age are women. On average, mortality occurs at younger ages in developing countries.[1]
It is projected that the significant burden of cardiovascular disease would shift to developing countries within this decade. Current projections suggest that by 2030, IHD will surpass HIV/AIDS as the primary cause of morbidity and mortality. Evidence that developing countries are experiencing an epidemiological transition with a new double burden of disease from non-communicable and communicable diseases is unfolding.[4]

AETIOLOGY

Atherosclerosis is the primary cause of acute coronary syndrome. These hemodynamically insignificant yet vulnerable lesions cause the formation of thrombus, which occludes the coronary arteries. Atheroma rupture is commonly found in 60% of patients compared to atheroma erosion, which constitutes 30%.[5]
The atheromatous lesion is composed of a large lipid core, calcium, numerous inflammatory cells, and a thin, fibrous cap. The dissolution of the fibrous cap is caused by the release of metalloproteinases from activated inflammatory cells. This event exposes thrombogenic material and promotes platelet activation and aggregation, activation of the coagulation cascade and vasoconstriction.

This process culminates in a thrombus formation within the coronary artery lumen and leads to varying degrees of vascular occlusion. Distal embolization may occur. Within 24 hours, spontaneous thrombolysis would have occurred in about 60% of patients.5 Regardless of thrombolysis, once obstruction ensues, tissue necrosis results. Other causes of acute coronary syndromes include coronary artery embolism, coronary spasm, coronary arterial embolism and coronary artery dissection.

The major determinants of a patient's clinical presentation and outcome are the severity and duration of the obstruction, the composite area of the myocardium affected, the demand by the myocardium, and compensation by the healthy heart muscle.[3]

PATHOPHYSIOLOGY

The acute coronary event reduces oxygen supply and increases oxygen demand by the myocardium and presents with features ranging from transient ischemia to infarction.[1]

The primary consequence of ischemia is myocardial dysfunction which presents with impaired contractility and relaxation of myocardial tissue, resulting in hypokinetic or akinetic segments; these segments may lead to paradoxical wall motion during systole. The composite area of the ischemic myocardium causes pathologies that vary from minimal heart failure to cardiogenic shock. A significant amount of myocardium must be affected to cause myocardial dysfunction.

Myocardial infarction, a permanent myocardial dysfunction, results from myocardial necrosis due to an abrupt reduction or cessation in myocardial perfusion. Myocardial infarction particularly affects the left ventricle but does not exclude other chambers of the heart. Necrosis of the interventricular septum may occur, leading to aneurysm, pseudoaneurysm, or rupture.

Normal electrical activity is altered once ischemia ensues. This results in ECG changes, namely, ST-T wave abnormalities-ST-segment elevation (injury current), ST-segment depression, T-wave inversion, pathologic Q wave and peaked T waves, conduction disturbances, and arrhythmias.[6]

CLASSIFICATION

Acute coronary syndrome includes:

1. Unstable angina
2. Non–ST-segment elevation myocardial infarction (NSTEMI)
3. ST-segment elevation myocardial infarction (STEMI).

They are distinguished based on symptoms, ECG findings, and cardiac marker levels, and they vary in approach to treatment and prognosis.

- Unstable angina is characterized when cardiac biomarkers do not meet the criteria for myocardial infarction (MI). It is often a prelude to myocardial infarction, arrhythmias or even sudden death.[7]

Infarction may be transmural or non-transmural. The depth of transmural necrosis is not clinically precisely determined; thus, infarcts are usually classified by the presence or absence of ST-segment elevation or Q waves on the ECG as STEMI or NSTEMI. Both types of MI may or may not produce Q waves on the ECG (Q wave MI, non-Q wave MI), therefore:

- Patients with acute chest pain and persistent (>20 min) ST-segment elevation (STEMI): It is reflective of a total coronary occlusion. Most of these patients will ultimately develop an ST-elevation MI (STEMI).

- Patients with acute chest pain but without persistent ST-segment elevation (NSTEMI): these patients rather exhibit persistent or transient ST-segment depression or T-wave inversion, broad T-waves, pseudonormalization of T waves, or no ECG changes at presentation. [7]

RISK FACTORS

Risk factors for ACS can be grouped into non-modifiable or modifiable.

The non-modifiable risk factors include increasing age, male sex, family history of cardiovascular diseases and race, while modifiable risk factors include hypertension, diabetes mellitus, smoking, dyslipidemia, abdominal obesity, excessive alcohol consumption, sedentary lifestyle and illicit drug use like amphetamine and cocaine.[3,4]

CLINICAL PRESENTATION

Signs and symptoms may vary and are based on the location and size of the lesion. 20-40% of patients typically present with severe chest pain, which often radiates to the left arm, neck, jaw, back, and epigastrium. Patients describe a feeling of tightness, pressure, or squeezing around the thoracic area. The pain may be associated with difficulty with breathing, weakness, dizziness, nausea, vomiting, palpitations, diaphoresis, fatigue, or syncope. These symptoms are not specific to ACS and may be attributed to gastrointestinal, neurological, pulmonary, or musculoskeletal diseases. Acute coronary syndrome can present with atypical symptoms or are infrequently asymptomatic, thereby requiring ECG, the elevation of cardiac biomarkers, or cardiac imaging for diagnosis. Women, diabetic and older patients usually present with atypical symptoms. [1]

COMPLICATIONS

Following the acute event, a myriad of complications can ensue. Complications of ACS can be grouped into acute and late complications. Acute complications include arrhythmias, conduction defects, cardiogenic shock, pericarditis, acute heart failure, mitral regurgitation, interventricular wall rupture. Late complications include postinfarction angina, recurrent ischemia, thromboembolism, Dressler's syndrome, recurrent arrhythmias, ventricular aneurysm, and pseudoaneurysm.

MANAGEMENT

A. **DIAGNOSIS**

Rapid and accurate diagnosis is critical in managing acute coronary syndrome because timely intervention and prompt treatment improve prognosis significantly. The initial goal is to alleviate cardiac ischemia.

Acute coronary syndromes should be considered in men and women above 35 years whose chief symptom is chest pain, excluding all other causes. The correct diagnosis of ACS involves history, risk factor analysis, initial and serial electrocardiogram review, and serial serum cardiac marker determination, thereby distinguishing between UA, NSTEMI, and STEMI.[8]

B. **INVESTIGATIONS**

Electrocardiogram

ECG is to be carried out and reviewed promptly. In emergencies, the ECG is the most precise investigation with a specificity of 90% and a sensitivity of 45%. [6] For STEMI, an initial ECG is usually diagnostic. It shows ST-segment elevation ≥ 1 mm in 2 or more connecting leads subtending the damaged area. ST-segment elevations may be subtle, and serial tracing may indicate the development of Q waves, which are, however, not necessary for diagnosis. NSTEMI is more challenging to interpret as they produce less striking, variable, and nonspecific degrees of ST-T wave abnormalities. In patients with transient ST-segment elevations, other pathologies such as pericarditis and Prinzmetal angina are possible differentials.

In situations where ECG reveals a J-point (the junction between ST-segment and QRS Complex) elevation in the limb leads, a myocardial infarction is to be suspected. 8 An ECG alone should not be used to rule out the possibility of ACS. [9,10]

Cardiac markers

Serum markers of myocardial cell injury are cardiac enzymes and cell contents. The troponins (cTn) are the most sensitive and specific and are markers of choice.

Cardiac troponins, Creatinine Kinase-MB (CK-MB) isoenzyme, and Creatinine Kinase (CK) are the most desired cardiac enzymes used in ACS diagnosis in that order.[11] CK is the least desired as its serum level is also elevated in non-cardiac conditions like trauma, hyperthyroidism, hyperthermia and renal insufficiency.9 CK-MB is more cardiac-specific than CK and is helpful in the early diagnosis of ACS. CK-MB is usually detected in serum about 4-6 hours immediately after ischemia. It peaks in an average of 12-24 hours and normalizes in 2-3 days. CK-MB does not accurately predict the degree of the infarct. A combined serum assay for all of these three cardiac enzymes is probably most useful, especially in determining the extent of the infarct. Elevated levels of CK-MB and cardiac troponins suggest an acute myocardial infarct.[9]

Coronary angiography

If possible, emergency coronary angiography and percutaneous coronary intervention (PCI) are promptly carried out within minutes after acute myocardial infarction. Angiography is an urgent procedure in patients with STEMI, continuous chest pain, and complications. With uncomplicated NSTEMI or unstable angina, angiography need not be urgent but should be carried out to identify possible lesions.[6]

Other tests

Routine laboratory tests identify nonspecific abnormalities compatible with tissue necrosis (increased ESR, moderately elevated WBC count with a shift to the left, haemogram). A lipid profile obtained within 24 hours is essential.[6] Stress test imaging may be done in patients with symptoms but non-diagnostic ECGs. Right heart catheterization is not routinely advised and is performed on patients with severe complications.[9]

C. TREATMENT

Patients with presumed ACS are managed with clopidogrel, aspirin, heparin, nitrates, statins and opioids. Oxygen is administered when oxygen saturation is reduced.[12] For STEMI patients, on confirmation of diagnosis, risk stratification and reperfusion therapy should commence. Injectable thrombolytics are administered and possibly, coronary angioplasty of identified lesions. Several drug trials show that thrombolysis reduces mortality by 29% in STEMI patients treated within 6 hours after the commencement of symptoms, primarily chest pain.[13]

The earlier reperfusion is commenced, the better the prognosis.

Empirical treatment with aspirin, another platelet inhibitor, low-molecular-weight heparin, intravenous nitroglycerin, and opioids is employed to manage unstable angina. Statin usage within 14 days of ACS diagnosis has been shown to reduce the risk of further ACS.[14]

Surgeries for ACS include the following coronary revascularization procedures: Percutaneous coronary intervention (PCI) (angioplasty and stenting) and coronary artery bypass graft (CABG) surgery.

D. PROGNOSIS

Certain prognostic risk factors among patients with ACS include demographics and some clinical characteristics: Increase in age, patients with prior ACS, patients with associated peripheral and cerebrovascular diseases and patients with complications have a more dire prognosis.

The Thrombolysis In Myocardial Infarction (TIMI) risk score, developed by the independent TIMI study group, can prognosticate patients with UA/NSTEMI.[15] It classifies a patient's risk of death, repeat ischemic events, and serves as a basis for therapy.

The Global Registry of Acute Coronary Events (GRACE) uses clinical presentations and biomarker characteristics as a basis for prognosis. The nine factors, including

age, heart failure, peripheral vascular disease, Killip class, elevated cardiac enzymes, cardiac arrest on admission, serum creatinine concentration, systolic blood pressure, and ST-segment deviation, can predict repeat MI, death or both.[16]

The Killip classification system is used in individuals who have suffered from an acute myocardial infarction. Physical examination and possible development of heart failure predict and stratify the risk of mortality. Individuals with a high Killip class are more likely to die within the first 30 days after the acute event.[17]
Biomarkers for prognosis include Natriuretic peptide, both types B and N, and Monocyte chemo attractive protein (MCP)-1.

PREVENTION

Primary prevention of ACS begins with a healthy lifestyle and appropriate medication to lower risk factors. A balanced diet with larger portions of vegetables and fruit, 150 mins of moderate aerobic activity or 75 mins of vigorous exercise per week is recommended.[18] Adhering strictly to the management plan in patients with hypertension, hypercholesterolemia and diabetes mellitus remain highly essential.

SUMMARY

Educating the public and advocacy on the risk factors and varied presentation of ACS is essential to achieve a reduction in mortality and morbidity. Primary prevention is still the backbone of countering most diseases.

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