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Overview of Coronary Artery Disease

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1. Introduction

Coronary artery disease (CAD) is a major cause of death and disability in developed countries. Although CAD mortality rates have declined over the past four decades in the United States (and elsewhere), CAD remains responsible for about one-third of all deaths in individuals over age 35 [1,2]. It has been estimated that nearly one-half of all middle-aged men and one-third of middle-aged women in the United States will develop some manifestation of CAD.

2. Prevalence

The 2010 Heart Disease and Stroke Statistics update of the American Heart Association reported that 17.6 million persons in the United States have CAD, including 8.5 million with myocardial infarction (MI) and 10.2 million with angina pectoris [2]. The reported prevalence increases with age for both women and men. In a 2009 report that used National Health and Nutrition Examination Survey (NHANES) data, MI prevalence was compared by sex in middle-aged individuals (35 to 54 years) during the 1998 to 1994 and 1999 to 2004 time periods [3]. Although MI prevalence was significantly greater in men than women in both time periods (2.5 versus 0.7 and 2.2 versus 1.0 respectively), there were trends toward a decrease in men and an increase in women. Data from NHANES (and other databases) that rely on self-reported MI and angina from health interviews probably underestimate the actual prevalence of advanced CAD.

This is likely as advanced occlusive coronary artery disease often exists with few symptoms or overt clinical manifestations. Silent ischemia, which is thought to account for 75 percent of all ischemic episodes [4], may be brought to light by electrocardiographic changes (ST segment depression) on an exercise test, ambulatory 24 hour electrocardiographic recording, or periodic routine electrocardiogram (ECG).

3. Global trends

Heart disease mortality has been declining in the United States and in regions where economies and health care systems are relatively advanced, but the experience is often quite different around the world [5]. Coronary artery disease is the number one cause of death in

adults from both low- and middle income countries as well as from high-income countries [5]. At the turn of the century, it was reported that CAD mortality was expected to increase approximately 29 percent in women and 48 percent in men in developed countries between 1990 and 2020. The corresponding estimated increases in developing countries were 120 percent in women and 137 percent in men [6].

The most dramatic increments in ischemic heart disease events on a percentage basis were forecast for the Middle East and Latin America. The experience in Asia is especially important because of the large populations involved. In India, CAD may not be largely explained by traditional risk factors [7]. In China, risk factor trends complement tracking of event rates. For example, the dramatic increase in CAD mortality in Beijing is attributable to greater cholesterol levels. The mean cholesterol level was 4.30 mmol/L (166 mg/dL) in 1984 and 5.33 mmol/L (206 mg/dL) only 15 years later [8]. In Latin America, declines in vascular disease rates have been less favorable than in the United States; unfavorable trends in physical activity, obesity, and smoking contribute to these differences [9].

International leaders have called for action plans to avert the projected global epidemic of ischemic heart disease in developing countries [10].

4. Pathophysiology

4.1 Cellular level

Angina is caused by myocardial ischemia, which occurs whenever myocardial oxygen demand exceeds oxygen supply (Figure 1). Because oxygen delivery to the heart is closely
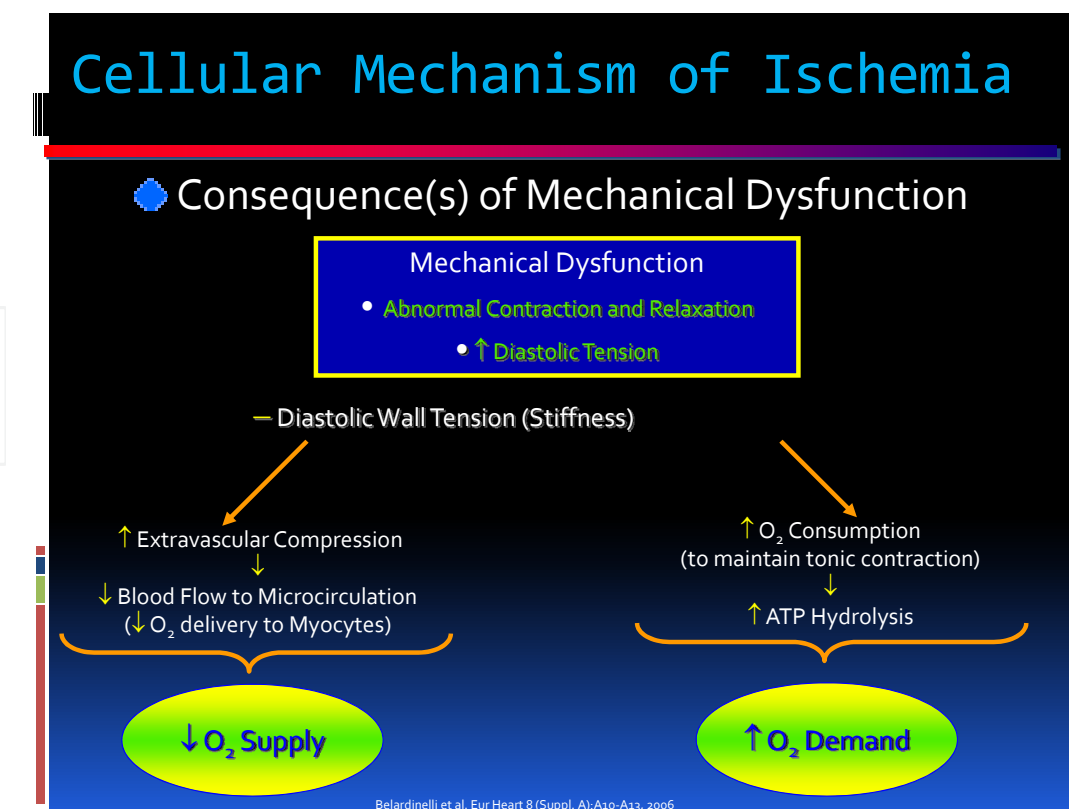

Fig. 1.

coupled to coronary blood flow, a sudden cessation of regional perfusion following a thrombotic coronary occlusion quickly leads to the cessation of aerobic metabolism, depletion of creatine phosphate, and the onset of anaerobic glycolysis. This is followed by the accumulation of tissue lactate, a progressive reduction in tissue ATP levels, and an accumulation of catabolites, including those of the adenine nucleotide pool. As ischemia continues, tissue acidosis develops and there is an efflux of potassium into the extracellular space. Subsequently, ATP levels fall below those required to maintain critical membrane function, resulting in the onset of myocyte death. Irreversible myocardial injury begins after 20 minutes of coronary occlusion in the absence of significant collaterals [11].

Irreversible injury begins in the subendocardium and progresses as a wave front over time, from the subendocardial layers to the subepicardial layers. This reflects the higher oxygen consumption in the subendocardium and the redistribution of collateral flow to the outer layers of the heart by the compressive determinants of flow at reduced coronary pressure. Factors that increase myocardial oxygen consumption (e.g., tachycardia) or reduce oxygen delivery (e.g., anemia, arterial hypotension) accelerate the progression of irreversible injury. In contrast, repetitive reversible ischemia or angina prior to an occlusion can reduce irreversible injury through preconditioning [12].

4.2 Anatomical level

Acute coronary syndrome is usually caused by an unstable atherosclerotic plaque rupture with subsequent platelet-rich thrombus overlying the culprit lesion causing severe narrowing (Figure. 2). This abrupt decrease in blood supply often results in chest pain and ECG changes indicative of ischemia, and, if prolonged, results in myocardial necrosis and
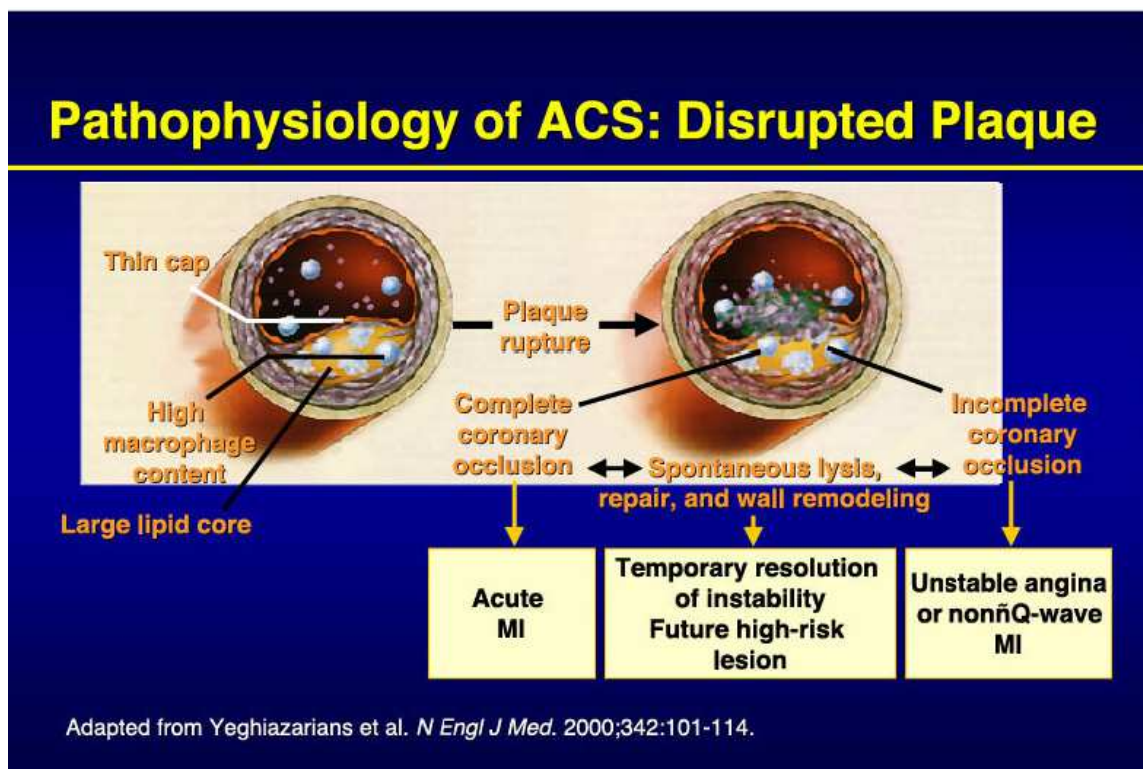

Fig. 2.

enzyme elevation. Less commonly, non ST elevation myocardial infarction is caused by diseases in which myocardial demand exceeds myocardial supply causing a similar clinical presentation. These diseases usually cause a hypermetabolic or high cardiac output state and include hyperthyroidism, anemia, fever, pheochromocytoma, hypertrophic cardiomyopathy, AV fistula and hypertensive urgency/emergency.

Whatever the mechanism of angina may be, patients usually develop the clinical manifestations in a sequence as described in Figure 3. Note that diastolic dysfunction is the earliest manifestation of ischemia, next to perfusion abnormalities which are seen in nuclear studies.

5. Clinical features

Coronary artery disease could be manifesting as a continuum from stable angina to acute coronary syndrome. Angina pectoris is a discomfort in the chest or adjacent areas caused by myocardial ischemia. It is usually brought on by exertion and is associated with a disturbance in myocardial function, without myocardial necrosis.
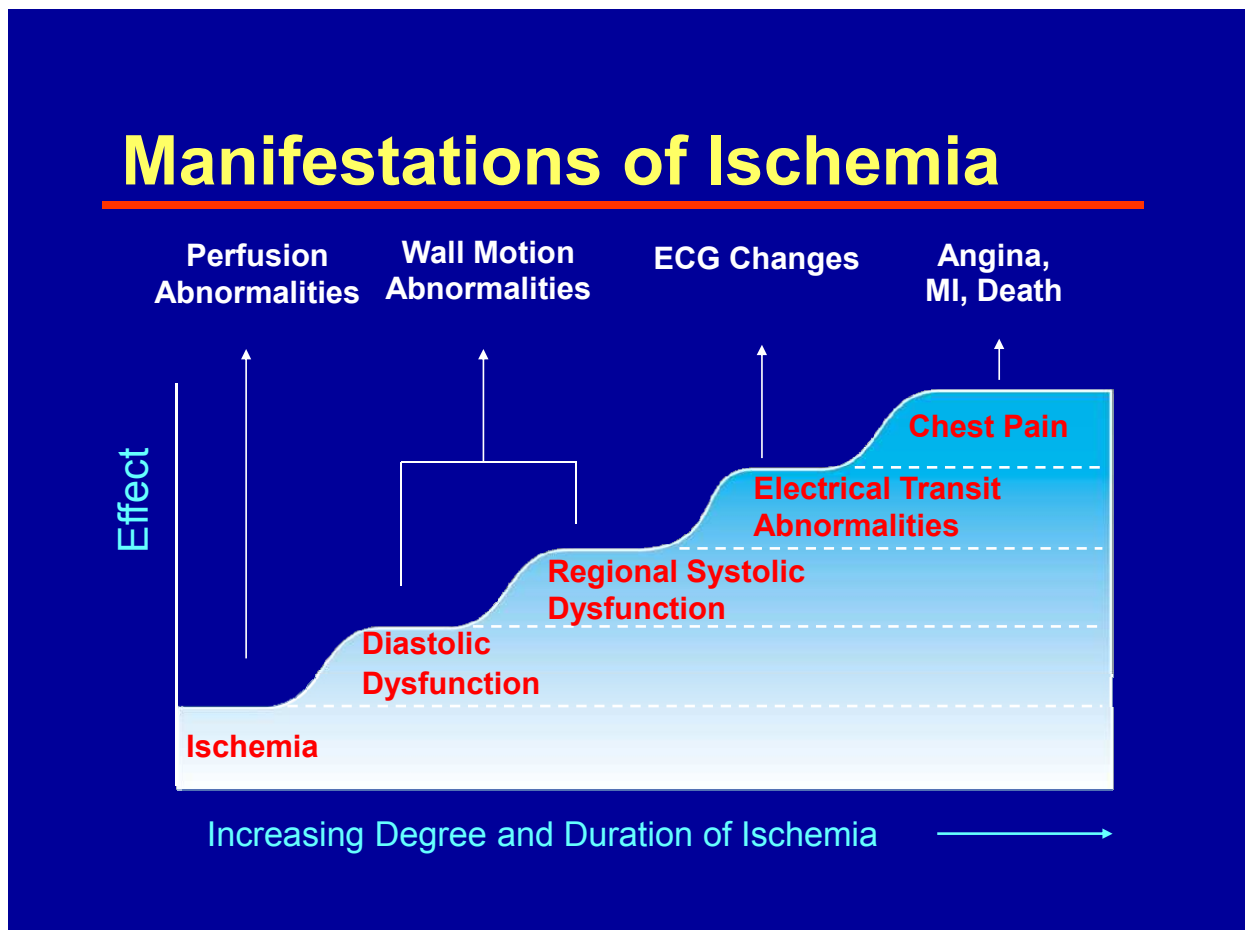

Fig. 3.

Heberden's initial description of angina as conveying a sense of "strangling and anxiety" is still remarkably pertinent. Other adjectives frequently used to describe this distress include viselike, constricting, suffocating, crushing, heavy, and squeezing. In other patients, the

quality of the sensation is more vague and described as a mild pressure-like discomfort, an uncomfortable numb sensation, or a burning sensation.

The site of the discomfort is usually retrosternal, but radiation is common and usually occurs down the ulnar surface of the left arm; the right arm and the outer surfaces of both arms may also be involved. Epigastric discomfort alone or in association with chest pressure is not uncommon. Anginal discomfort above the mandible or below the epigastrium is rare. Anginal equivalents (i.e., symptoms of myocardial ischemia other than angina), such as dyspnea, faintness, fatigue, and eructations, are common, particularly in the elderly.

A history of abnormal exertional dyspnea may be an early indicator of CAD even when angina is absent or no evidence of ischemic heart disease can be found on the electrocardiogram (ECG). Dyspnea at rest or with exertion may be a manifestation of severe ischemia, leading to increases in left ventricular (LV) filling pressure. Nocturnal angina should raise the suspicion of sleep apnea.

The term acute coronary syndrome (ACS) is applied to patients in whom there is a suspicion of myocardial ischemia. There are three types of ACS: ST elevation (formerly Q-wave) MI (STEMI), non-ST elevation (formerly non-Q wave) MI (NSTEMI), and unstable angina (UA). The first two are characterized by a typical rise and/or fall in biomarkers of myocyte injury.

For many years, the diagnosis of acute MI relied on the revised criteria established by the World Health Organization (WHO) in 1979. These criteria were epidemiological and aimed at specificity. A joint European Society of Cardiology (ESC) and American College of Cardiology (ACC) committee proposed a more clinically based definition of an acute, evolving, or recent MI in 2000 (13). In 2007 the Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Health Federation (ESC/ACCF/AHA/WHF) refined the 2000 criteria and defined acute MI as a clinical event consequent to the death of cardiac myocytes (myocardial necrosis) that is caused by ischemia (as opposed to other etiologies such as myocarditis or trauma) [14].

The criteria used to define MI differ somewhat depending upon the particular clinical circumstance of the patient: those suspected of acute MI based upon their presentation, those undergoing either coronary artery bypass graft surgery or percutaneous intervention, or those who have sustained sudden unexpected, cardiac arrest with or without death [14].

For patients who have undergone recent revascularization or who have sustained cardiac arrest or death, the criteria for the diagnosis of MI are given in detail in the Table 1.

For all other patients in whom there is a suspicion of MI, a typical rise and/or gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis, with at least one of the following is required:

- Ischemic symptoms
- Development of pathologic Q waves on the ECG
- ECG changes indicative of ischemia (ST segment elevation or depression)
- Imaging evidence of new loss of viable myocardium or a new regional wall motion abnormality.

In addition, pathologic findings (generally at autopsy) of an acute MI are accepted criteria.

The joint task force [14] further refined the definition of MI by developing a clinical classification according to the assumed proximate cause of the myocardial ischemia.

Patulous aneurysmal dilation involving most of the length of a major epicardial coronary artery is present in approximately 1 to 3 percent of patients with obstructive CAD at autopsy or angiography. This angiographic lesion does not appear to affect symptoms, survival, or incidence of MI. Most coronary artery ectasia and/or aneurysms are caused by coronary atherosclerosis (50 percent), and the rest are caused by congenital anomalies and inflammatory diseases, such as Kawasaki disease.

Type 1
Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

Type 2
Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension

Type 3
Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

Type 4a
Myocardial infarction associated with PCI

Type 4b
Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

Type 5
Myocardial infarction associated with CABG

Table 1. Types of Myocardial Infarction

6. Diagnosis

Evaluation of new onset chest pain in stable individuals should begin with the consideration of imminently life-threatening causes (including acute coronary syndrome, pulmonary embolus, aortic dissection, pneumothorax, and esophageal rupture). This is usually accomplished using clinical judgement, along with ECG testing, and less frequently exercise testing, other noninvasive testing, or invasive angiography.

This is being discussed in detail by other authors in various chapters. nce a life-threatening etiology has been excluded, attempts should be made to identify the specific cause of symptoms and begin treatment. A diagnostic pattern will frequently emerge, based upon the patient's risk factors, description of the pain, and associated symptoms.

Standard clinical characteristics routinely obtained during the initial medical evaluation of patients with UA/NSTEMI can be used to construct a simple classification system that is predictive of risk for death and cardiac ischemic events. The TIMI (Thrombolysis in Myocardial Infarction) risk score (Table 2) includes variables that can be easily ascertained when a patient with UA/NSTEMI presents to the medical care system. The variables used to construct the score were based on observations from prior studies of risk stratification and incorporate demographic and historical features of the patient, measures of the tempo and acuity of the presenting illness, and indicators of the extent of myocardial ischemia and necrosis.

Since patients with an acute coronary syndrome are at increased risk of death and nonfatal cardiac events, clinicians must assess prognosis on an individual basis to formulate plans for evaluation and treatment. The TIMI risk score for UA/NSTEMI is a simple prognostication

TIMI Risk Score	All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 d After Randomization, %
0–1	4.7
2	8.3
3	13.2
4	19.9
5	26.2
6–7	40.9

The TIMI risk score is determined by the sum of the presence of 7 variables at admission; 1 point is given for each of the following variables:

- age 65 y or older;
- at least 3 risk factors for CAD;
- prior coronary stenosis of 50% or more;
- ST-segment deviation on ECG presentation;
- at least 2 anginal events in prior 24 h;
- use of aspirin in prior 7 d;
- elevated serum cardiac biomarkers.

Prior coronary stenosis of 50% or more remained relatively insensitive to missing information and remained a significant predictor of events.

Table 2. TIMI Risk Score for Unstable Angina/Non-ST Elevation MI

scheme that enables a clinician to categorize a patient's risk of risk of death and ischemic events at the critical initial evaluation. A promising clinical application of this score is identification of a patient for whom new antithrombotic therapies would be especially effective.

While ECG manifestations are an important component of diagnosis, one should be aware of the pitfalls of the ECG. Tables 3, 4 and 5 describe the ECG manifestations of ischemic disease.

ST elevation
New ST elevation at the J-point in two contiguous leads with the cut-off points: ≥ 0.2 mV in men or ≥ 0.15 mV in women in leads V_2–V_3 and/or ≥ 0.1 mV in other leads

ST depression and T-wave changes
New horizontal or down-sloping ST depression ≥ 0.05 mV in two contiguous leads; and/or T inversion ≥ 0.1 mV in two contiguous leads with prominent R-wave or R/S ratio >1

Table 3. ECG manifestations of acute myocardial ischemia (in absence of LVH and LBBB)

Any Q-wave in leads V_2–V_3 ≥ 0.02 s or QS complex in leads V_2 and V_3

Q-wave ≥ 0.03 s and ≥ 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V_4–V_6 in any two leads of a contiguous lead grouping (I, aVL, V_6; V_4–V_6; II, III, and aVF)[a]

R-wave ≥ 0.04 s in V_1–V_2 and R/S ≥ 1 with a concordant positive T-wave in the absence of a conduction defect

[a]The same criteria are used for supplemental leads V_7–V_9, and for the Cabrera frontal plane lead grouping.

Table 4. ECG changes associated with prior myocardial infarction

False positives
   Benign early repolarization
   LBBB
   Pre-excitation
   Brugada syndrome
   Peri-/myocarditis
   Pulmonary embolism
   Subarachnoid haemorrhage
   Metabolic disturbances such as hyperkalaemia
   Failure to recognize normal limits for J-point displacement
   Lead transposition or use of modified Mason–Likar
   configuration[24]
   Cholecystitis
False negatives
   Prior myocardial infarction with Q-waves and/or persistent
       ST elevation
   Paced rhythm
   LBBB

Table 5. Common ECG pitfalls in diagnosing myocardial infarction

7. Management

Patients with an initial ECG reading that reveals new or presumably new ST segment depression and/or T wave inversion, although not considered candidates for fibrinolytic therapy, should be treated as though they are suffering from MI without ST elevation or unstable angina (a distinction to be made subsequently after scrutiny of serial ECGs and serum cardiac marker measurements). In patients with a clinical history suggestive of STEMI and an initial nondiagnostic ECG reading (i.e., no ST segment deviation or T wave inversion), serial tracings should be obtained while the patients are being evaluated in the emergency department.

Emergency department staff can be alerted to the sudden development of ST segment elevation by periodic visual inspection of the bedside ECG monitor, by continuous ST segment recording, or by auditory alarms when the ST segment deviation exceeds programmed limits. Decision aids such as computer-based diagnostic algorithms, identification of high-risk clinical indicators, rapid determination of cardiac serum markers, two-dimensional echocardiographic screening for regional wall motion abnormalities, and myocardial perfusion imaging have greatest clinical utility when the ECG reading is nondiagnostic. In an effort to improve the cost-effectiveness of care of patients with a chest pain syndrome, nondiagnostic ECG reading,

and low suspicion of MI but in whom the diagnosis has not been entirely excluded, many medical centers have developed critical pathways that involve a coronary observation unit with a goal of ruling out MI in less than 12 hours.

Once the diagnosis of coronary artery disease is made, the management depends upon the severity of the underlying severity of the disease. The treating physician has to diagnose the clinical scenario appropriately as the treatment largely depends of the initial diagnosis. Stable coronary artery disease could be managed conservatively whereas acute coronary syndrome has to be managed very aggressively. Select patients have to be considered for early invasive strategy too.

The prehospital care of patients with suspected STEMI is a crucial element bearing directly on the likelihood of survival. Most deaths associated with STEMI occur within the first hour of its onset and are usually caused by ventricular fibrillation. Accordingly, the importance of the immediate implementation of definitive resuscitative efforts and of rapidly transporting the patient to a hospital cannot be overemphasized.

Major components of the delay from the onset of symptoms consistent with acute myocardial infarction (MI) to reperfusion include the following: (1) the time for the patient to recognize the seriousness of the problem and seek medical attention; (2) prehospital evaluation, treatment, and transportation; (3) the time for diagnostic measures and initiation of treatment in the hospital (e.g., "door-to-needle" time for patients receiving a thrombolytic agent and "door-to-balloon" time for patients undergoing a catheter-based reperfusion strategy); and (4) the time from initiation of treatment to restoration of flow (Figure 4). Thrombolytic therapy is vital for early reperfusion and this has been discussed in other chapters.
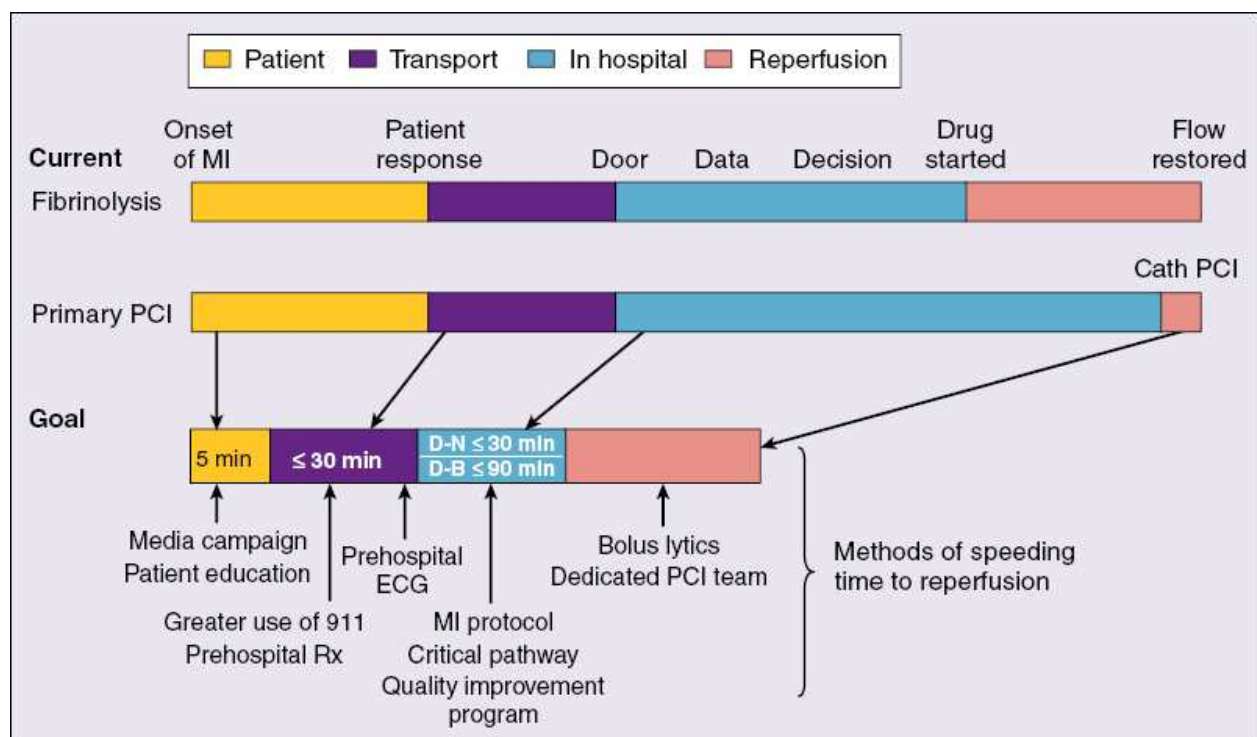
(Modified from Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction]. Circulation 110:e82, 2004.)

Fig. 4.

Adjunctive medical therapy is a critical component of therapy for STEMI and confers benefit in addition to that gained by reperfusion therapies, regardless of method of reperfusion (Table 6).

Ancillary therapy can be used to facilitate and enhance coronary reperfusion or to limit the consequences of myocardial ischemia. Early, aggressive use of antiplatelet therapies, such as aspirin, and clopidogrel, confers significant additional mortality benefit when given as adjuncts to thrombolysis or PCI. Beta-blockers, angiotensin-converting enzyme inhibitors in appropriately selected patients, and 3-hydroxy-3-methylglutaryl CoA reductase inhibitors (statins) have all been shown to reduce the risk of cardiovascular events and mortality in patients who have STEMI. Therapies such as nitroglycerin and morphine have no mortality benefit but may improve symptoms and reduce ischemic burden. Calcium-channel blockers and prophylactic anti-arrhythmic drug therapy (lidocaine) may increase mortality. The suggested benefit of metabolic modulation at the myocyte level with electrolytes, glucose, and insulin seen in small, early trials has not been reproduced in larger, randomized studies.

Mechanisms and clinical effects of adjunctive therapies for patients who have ST elevation myocardial infarction

Agent	Mechanism	Clinical Effect
Adjunctive therapies for AMI		
Aspirin	Antiplatelet	Improve survival
		Decrease reinfarction, CVA
Thienopyridines (clopidogrel, ticlopidine)	Antiplatelet	Recommended in aspirin-allergic patients
		Decrease death, MI, CVA in NSTE ACS
Glycoprotein IIb/IIIa inhibitors	Antiplatelet	Decrease MI, ischemic complications following primary PCI
		Decrease death or MI in high-risk NSTE ACS
Unfractionated heparin	Antithrombin	Decrease death and MI in prefibrinolytic era
Low molecular weight heparins	Antithrombin	Reduce cardiac events in NSTE ACS versus unfractionated heparin
Direct thrombin inhibitors	Antithrombin	Recommended in heparin-induced thrombocytopenia
Beta-brockers	Decrease myocardial oxygen demand ($\downarrow$HR, $\downarrow$BP)	Improve survival
		Reduce infract size, ventricular arrhythmias, recurrent ischemia
Adjunctive therapies for AMI		
ACE Inhibitors	Vasodilator (BP)	Improve survival
	Prevent LV remodeling	Decrease heart failure. LV dysfunction
IV nitroglycerine	Venous, arterial, coronary vasodilator ($\downarrow$BP, $\downarrow$preload)	No effect on survival
		Decrease recurrent ischemia
HMG CoA Reductase Inhibitors	Lipid lowering	Decrease future CV death and MI
	Anti-inflammatory	May decrease early ischemic events
Magnesium	Myocardial protective	Therapy for torsades depointes
	Anti-arrhythmic	May improve reperfusion outcomes
Calcium-channel blocker	Decrease myocardial oxygen demand ($\downarrow$HR, $\downarrow$BP)	No survival benefit
		Possible use in beta-blocker–intolerant patients without CHF or LV dysfunction
Warfarin	Oral anticoagulant	Reduced embolic risk with atrial fibrilliation. LV thrombus or dysfunction
Glucose-insulin potassium infusion	Metabolic modulator	May improve intracellular myocardial energy stores and outcome

Abbreviations: ACS, acute coronary syndrome; BP, blood pressure; CHF, congestive heart failure; CV, cardiovascular; CVA, cerebrovascular accident; HR, heart rate; IV, intravenous; LV, left ventricular; MI, myocardial infarction; NSTE, non–ST-segment elevation; PCI, percutaneous coronary intervention.

Adapted from Sura AC. et al.

Table 6.

8. Conclusion

Although improvements in the management of patients who have STEMI have led to a decline in acute and long-term fatality rates, reperfusion and ancillary therapies remain underused. Several initiatives (eg. Get With the Guidelines in USA) are designed to improve adherence to guidelines and access to appropriate reperfusion therapies. To date, clinical advancement is judged on achieving and maintaining epicardial artery patency. New fibrinolytics and combination therapies will continue to evolve. There will be technological advances and improvement in operator skills for PCI. The ultimate goal for the management of STEMI remains unchanged: to open occluded arteries quickly in carefully screened patients and in a cost-effective manner.

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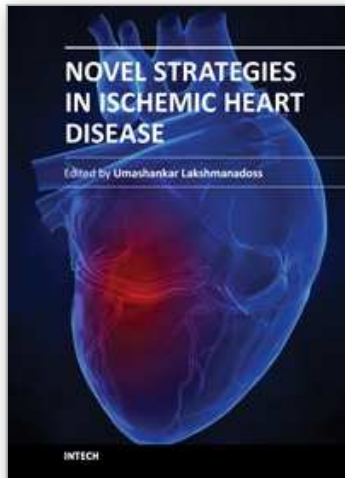
Novel Strategies in Ischemic Heart Disease

Edited by Dr. Umashankar Lakshmanadoss

The first edition of this book will provide a comprehensive overview of ischemic heart disease, including epidemiology, risk factors, pathogenesis, clinical presentation, diagnostic tests, differential diagnosis, treatment, complications and prognosis. Also discussed are current treatment options, protocols and diagnostic procedures, as well as the latest advances in the field. The book will serve as a cutting-edge point of reference for the basic or clinical researcher, and any clinician involved in the diagnosis and management of ischemic heart disease. This book is essentially designed to fill the vital gap existing between these practices, to provide a textbook that is substantial and readable, compact and reasonably comprehensive, and to provide an excellent blend of "basics to bedside and beyond" in the field of ischemic heart disease. The book also covers the future novel treatment strategies, focusing on the basic scientific and clinical aspects of the diagnosis and management of ischemic heart disease.

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