Updates Emergency Ischemic Chest Pain

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Abstract

Introduction: The hospital ED is one of the most important components of the health care system. There is an increase in numbers of chest pain patients visiting EDs leading to overcrowding, long waiting time, missed diagnostic cases and negative impact on patient satisfaction. Chest pain is one of the most common reasons people call for emergency medical help. Fortunately, chest pain was not always a signal for a heart attack. Often chest pain is unrelated to any heart problem. However, even if the chest pain the patient experience has nothing to do with his cardiovascular system, the problem may still be important and worth the time spent in an emergency room for evaluation. So, we aimed to update knowledge and review researches in the field of chest pain and reduction in waiting time, missed diagnostic cases and Overcome Crowdness Patients in emergency department.

Methods: Collection of all possible available data about the chest pain patients in the Emergency department. By many research questions to achieve these aims so, a midline literature search was performed with the keywords "critical care", "emergency medicine", "acute chest pain", "myocardial ischemia". All studies introduced that the myocardial ischemia is a serious pathology that face patients of the emergency and critical care departments. Literature search included an overview of recent definition, causes, pathophysiology, prophylactic and recent therapeutic strategies.

Results: Myocardial Ischemia means narrowing of Coronaries whatever it was transient or permanent, partial or complete, painful or silent, recurrent or firstly experienced, recordable or not. This, although chambers are full of blood, makes heart muscle blood supply decreases and if it continued, it may result in myocardium permanent damage (Myocardial infarction).

Conclusion: "Prevention is better than cure" Decline in death rates could be achieved by adopting a healthier lifestyle. That is why it is important for healthcare professionals to implement primary and secondary prevention. And there are many types emergency department of chest pain risk stratifications so any one of them should be applied e.g. TIMI or HEART score in daily emergency work.

Keywords: Emergency Department; Myocardial Ischemia; Management

Abbreviations

AA: Aortic Atresia; ALCAPA: Anomalous origin of the left coronary artery from the pulmonary artery; ARCAPA: Anomalous origin of the right coronary artery from the pulmonary artery; CHD: Congenital Heart Disease; CRUX: A point on the diaphragmatic surface of the

heart where the right atrio-ventricular groove, the left atrio-ventricular groove and the posterior inter-ventricular groove come together; DIAD: The Detection of Ischemia in Asymptomatic Diabetics Investigators; EDC: The Pittsburgh Epidemiology of Diabetes Complications; Endothelial dysfunction: Rhe vasoconstrictor forces overweigh the vasodilator one; Eurodiab: A multicenter, clinic-based study in Europe; FRS: Framingham Risk Score; IVS: Interventricular Septum; MACE: Major Adverse Cardiac Events; MRFIT: Multiple Risk Factor Intervention Trial; MS: Mitral Stenosis; PA: Pulmonary Atresia; SAD: Sagittal Abdominal Diameter; SHEP Trial: Systolic Hypertension in the Elderly Program Trial

Introduction and Rational

Every year emergency room doctors evaluate and treat millions of people for chest pain that may be cardiac or non-cardiac in origin. One of the most famous and Dangerous causes is Myocardial Ischemia [1].

Myocardial ischemia, also called cardiac ischemia, can damage heart muscle, reducing its ability to pump efficiently. A sudden, severe blockage of a coronary artery may lead to a heart attack. Myocardial ischemia may also cause serious abnormal heart rhythms [2].

Myocardial ischemia occurs when the blood flow through one or more of the blood vessels that leads to your heart (coronary arteries) is decreased. This decrease in blood flow leads to a decrease for oxygen your heart muscle (myocardium) receives. Myocardial ischemia may occur slowly as arteries become blocked over time, or it may occur quickly when an artery becomes blocked suddenly [1].

Painful sensations with ischemic episodes may be due, in part, to mechanical factors such as spasm and/or distention of cardiac vessels. The most common cause of ischemia is coronary artery stenosis due to arteriosclerosis. The lesions present with arteriosclerosis interfere with increases in blood flow necessary to meet increased cardiac demand, which may lead to pain. Spasms in the coronary arteries and vessels are commonly seen in patients with variant angina and Cardiac Syndrome X, but may also be important mechanisms in stable and unstable angina [2].

Some patients had chest pain due to other causes, in addition to angina. Patients as discomfort, tightness, or pressure in the chest typically describe angina with pain radiating to other areas such as the left arm, abdomen, or jaw. Anginal pain is generally associated with some level of exertion and often decreases with rest. In contrast, chest pain of other origins is usually more localized, variable and described as sharp or burning. Non-cardiac chest pain will not typically improve with rest. Determination of the cause of chest pain involves obtaining a careful medical history and diagnostic tests for ischemia such as electro-cardiography (ECG), stress testing, or coronary angiography. There are several types of angina. Stable angina refers to chest pain that is predictable, reproducible with exertion and relieved by rest or nitroglycerin. Unstable angina refers to a changing pattern of angina that is often present at rest and requires immediate medical attention. Variant angina usually occurs at rest or without stress, shows ST segment elevations on the ECG and is due to coronary artery spasm [3].

Approximately 6.5 million people in the United States suffer from angina, with 400,000 new cases diagnosed each year. The traditional medical model of disease does little to explain the variability seen in pain reporting among patients with angina--ranging from none to moderate to severe pain occurring several times per day. Research has found an inconsistent correlation between the severity of heart disease and the reported level of pain with angina [4,5].

Treatment for myocardial ischemia is directed at improving blood flow to the heart muscle and may include medications, a procedure to open blocked arteries or coronary artery bypass surgery. Making heart-healthy lifestyle choices is important in treating and preventing myocardial ischemia [6]. Shock is defined as circulatory insufficiency that creates an imbalance between tissue oxygen supply and oxy-

gen demand. The result of shock is global tissue hypoperfusion and is associated with a decreased venous oxygen content and metabolic acidosis (lactic acidosis) [4].

Aim of the Work

To update knowledge and review researches in the field of chest pain in the emergency department to be a reference for researchers.

Materials and Methods

Study design

This study was carried out as a systematic review. Collection of all possible available data about the chest pain patients in the Emergency department.

Materials

- Literatures from emergency medicine and intensive care textbooks.

- Published articles from famous emergency medicine and intensive care journals.

- Papers, abstracts and texts published on the internet concerned with the acute chest pain patients.

- Thesis and papers in Egyptian Universities.

Search strategy

A Medline literature search was performed with the keywords "critical care," "emergency medicine," "acute chest pain", "myocardial ischemia". All studies introduced that the myocardial ischemia is a serious pathology that face patients of the emergency and critical care departments. Literature search included an overview of recent definition, causes, pathophysiology, prophylactic and recent therapeutic strategies.

Medline (PubMed), Up to date, Blackwell-synergy, Elsevier, Oxford medicine library and e-medicine was searched using standardized methodological filter for identifying trials, which represent the most famous scientific sites on the internet.

The most famous paid evidence based web sites journals and internet sites that represent honest references to most of the Emergency medicine physicians and cardiologists will be searched as:

- *BMJ evidence based.*

- *Cochrane.org*

- *The journal of trauma*

- *The New England journal of medicine*

- *American Heart journal*

- *Coronary Health Care journal*

- *Southern medical journal*

- *The journal of critical care.*

Criteria for selecting those studies

- Initial screen to exclude studies not relevant to the review questions.

- Second screen determines which of the relevant studies are evidence based and of the highest quality to be included in the systematic review to be as free from bias as possible.

Inclusion criteria

- Different articles on the myocardial ischemia in chest pain unit and Emergency patients.

- Studies with appropriate research methodology according to the standards of critical appraisal was included.

- Any type of study design including:

    - Randomized controlled trials.

    - Open clinical trials.

    - Review articles.

    - Systematic review articles.

    - Meta-analysis.

Exclusion criteria

- All studies that are not relevant to Myocardial ischemia in emergency patients.

- Rejected articles, articles with poorly designed studies were also excluded.

Summarizing the results of relevant studies

The results of the best available published studies were summarized.

Study prepared by:

- Uses paid evidence based websites for searching about papers and texts.

- Used Microsoft Office Word documents 2010 in typing and preparation of the systematic review.

- Used Internet Explorer Browser version 7 for searching in the internet about papers, abstracts and texts.

Normal coronaries

Like all organs, the heart is made of tissue and requires a supply of oxygen and nutrients. Although its chambers are full of blood, the heart receives no nourishment from this blood. The heart receives its own supply of blood from an arteries network, called the coronary arteries [7].

The coronary arteries are distributed to bring adequate blood supply. Despite numerous variations, this rule is always met in healthy hearts. An analysis of the total distribution of the coronary arteries in the heart will show that no areas are left devoid of blood supply [8] (Figure 1).
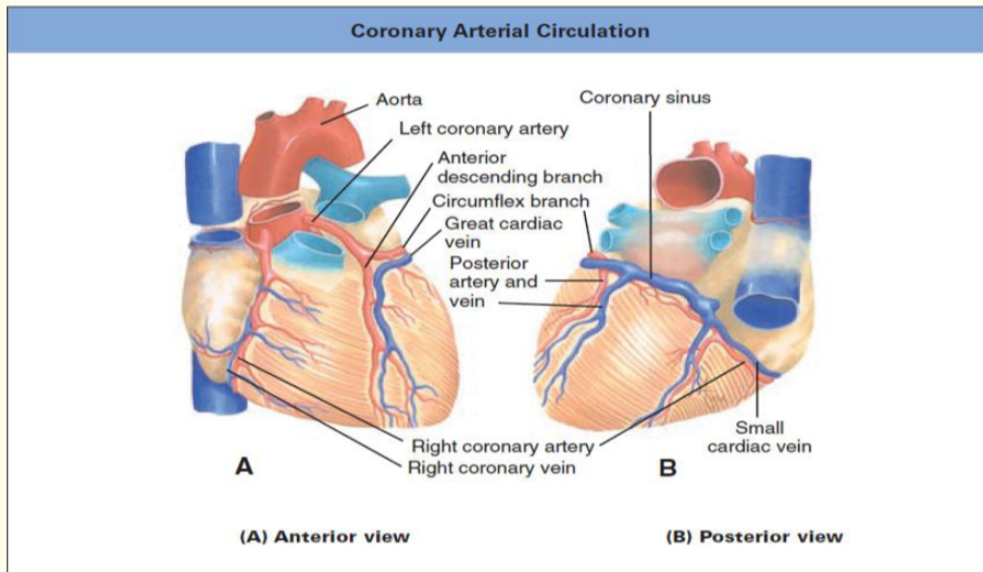
Figure 1: *Coronary artery anatomy.*

Left main coronary artery

The left main coronary artery arises from above the left portion of the aortic valve and then usually divides into two branches, known as the left anterior descending (LAD) and the circumflex (Circ) coronary arteries. In some patients, a third branch arises in between the LAD and the Circ. This is known as the ramus, intermediate, or optional diagonal coronary artery [7].

Right coronary artery [1-5]

The right coronary artery (RCA) originates above the right portion of the aortic valve and runs in the right atrio-ventricular groove towards the crux.

The conus artery: It is considered the first branch of the RCA. In about 50% of patients, this vessel arises at the right coronary ostium or within a few millimeters of the RCA. It passes upward and anteriorly over the right ventricular outflow tract toward the LAD. Its primary importance is to serve as a source of collateral circulation in patients with LAD occlusion. In other 50% of patients, the conus artery arises from a small separate ostium in the right coronary sinus just above the right coronary ostium [6].

Distribution of the coronary arteries to specific regions of the left ventricular myocardium:

- **The left ventricle:** The diagonal branches of the LAD supply lateral wall of the left ventricle, the terminal portion of the LAD wraps around the apex to supply it and continue to supply the diaphragmatic surface of the left ventricle together with the PDA that anastomose with the termination of the LAD, sometimes when the RCA is dominant the PDA is large that continue in the posterior interventricular septum to the apex to supply all that area. The OM branches of the LCX supply the free posterior wall of the LV; hence the LV is mainly supplied by the left system [7].

- **Interventricular septum:** Usually the septum receives its blood supply from the anterior inter-ventricular arteries, however; there are two or three other arterial sources for the nourishment of the septum. The LAD supplies the anterior two thirds of the inter-ventricular septum, while the PDA supplies the posterior one third of the inter-ventricular septum [11].

- **Papillary muscles:** The antero-lateral papillary muscle receives dual blood supply from the diagonal branches of the LAD as well as the LCX, while the postero-medial muscle receives blood supply from the PDA.

- **The sinus node:** Receives blood supply from the sinus nodal artery of the RCA in 55% of cases and from the LCX in 35%, and the sinus node receives dual blood supply in 10% of cases [12].

- **The atrioventricular node**: Receives its blood supply from the atrio-ventricular branch of the PDA in 80% of cases, from the LCX in 10%, and dual blood supply is seen in the last 10% of cases. The presence of collateral circulation from the LAD makes the AVN more prone to ischemia in cases of LAD occlusion than the SAN [11].

- **His bundle and the bundle branches:** Receives blood supply from the AV nodal artery and the septal penetrating branches of the LAD. The anterior fascicle of the left bundle branch receives blood supply from the penetrating septal arteries of the LAD, while the posterior fascicle receives dual blood supply from the septal penetrating branches of the LAD as well as branches from the PDA [9].

- **The right ventricle:** It receives its main blood supply from the RCA that gives the anterior ventricular branch to the anterior wall of the right ventricle, the posterior ventricular branch to the diaphragmatic surface of the RV and the PDA that gives blood supply to the RV throughout its course. The anterior inter-ventricular branch gives blood supply to the adjacent parts of the RV while it runs in the anterior Interventricular groove [13].

Chest pain in emergency room

More than 6 million patients present with chest pain and suspected acute coronary syndrome (ACS) to emergency departments (EDs) across the U.S. annually. These patients require rapid and efficient triage to hospitalization versus discharge to maximize appropriate allocation of resources to the highest-risk patients who require timely life-saving therapy [5]. Most commonly repeated Causes are [6].

Causes of chest pain that are immediately life threatening:

- **Heart attack (acute myocardial infarction):** A heart attack occurs when blood flow to the arteries that supply the heart (coronary arteries) becomes blocked. With decreased blood flow, the muscle of the heart does not receive enough oxygen [6]. This can cause damage, deterioration, and death of the heart muscle.

- **Angina:** Angina is chest pain related to an imbalance between the oxygen demand of the heart and the amount of oxygen delivered via the blood. It is caused by blockage or narrowing of the blood vessels that supply blood to the heart. Angina differs from a heart attack in that arteries are not completely blocked, and it causes little or no permanent damage to the heart [8]. Stable angina occurs repetitively and predictably while patient is doing exercise and goes away with rest. Unstable angina results in unusual and unpredictable pain not relieved totally by rest, or pain that actually occurs at rest [9].

- **Aortic dissection:** The aorta is the main artery that supplies blood to the vital organs of the body, such as brain, heart, kidneys, lungs, and intestines. Dissection means a tear in the inner lining of the aorta. This can cause massive internal bleeding and interrupt blood flow to the vital organs [11].

- **Pulmonary embolism**: A pulmonary embolus is a blood clot in one of the major blood vessels that supplies lungs. It is a potentially life-threatening cause of chest pain but is not associated with the heart [14].

- **Spontaneous pneumothorax**: Often called a collapsed lung, this condition occurs when air enters the saclike space between the chest wall and the lung tissue. Normally, negative pressure in the chest cavity allows the lungs to expand. When a spontaneous pneumothorax occurs, air enters the chest cavity. When the pressure balance is lost, the lung is unable to re-expand. This cuts off the normal oxygen supply in the body [19].

- **Perforated viscus**: A perforated viscus is a hole or tear in the wall of any area of the gastrointestinal tract. This allows air to enter the abdominal cavity, which irritates the diaphragm, and can cause chest pain [22].

- **Cocaine-induced chest pain**: Cocaine causes the blood vessels in the body to constrict. This can decrease blood flow to the heart, causing chest pain. Cocaine also accelerates the progression of atherosclerosis, a risk factor for a heart attack [24].

Causes of chest pain that are not immediately life-threatening:

- **Acute pericarditis**: Means inflammation of the pericardium, which is the sac that covers the heart.

- **Mitral valve prolapse**: It is an abnormality of one of the heart valves in which the "leaves" of the valve bulge into the upper heart chamber during contraction. When this occurs, a small amount of blood flows backward in the heart. This may cause chest pain in some people [26].

- **Pneumonia**: Pneumonia is an infection of the lung tissue. Chest pain occurs because of inflammation to the lining of the lungs [27].

- **Disorders of the esophagus**: Chest pain from esophageal disorders can be an alarming symptom because it often mimics chest pain from a heart attack [28].

- **Acid reflux disease:** (Gastro-esophageal reflux disease, GERD, heartburn) occurs when acidic digestive juices flow backward from the stomach into the esophagus. Resulting heartburn is sometimes experienced as chest pain [29].

- **Esophagitis**: Esophagitis is an inflammation of the esophagus [30].

- **Esophageal spasm:** Esophageal spasm is defined as excessive, intensified, or uncoordinated contractions of smooth muscles of the esophagus [31].

- **Costochondritis**: An inflammation of the cartilage between the ribs. Pain is typically located in the mid-chest, with intermittently dull and sharp pain that may be increased with deep breaths, movement, and deep touch [38].

- **Herpes zoster**: Also known as shingles, this is a reactivation of the viral infection that causes chickenpox. With shingles, a rash occurs, usually only on one small part of the body [39]. The pain, often very severe, is usually confined to the area of the rash. The pain may precede the rash by 4 - 7 days. Risk factors include conditions in which the immune system is compromised, such as advanced age, HIV, or cancer [40]. Herpes zoster is highly contagious to people who have not had chickenpox or have not been vaccinated against chickenpox for the five days before and the five days after the appearance of the rash [27].

Chest pressure with dyspnea leads physicians to consider an acute coronary syndrome such as unstable angina or MI, but these symptoms also may represent chest wall pain or PE. Dyspnea is common in patients with heart failure, whereas dyspnea with fever is characteristic of pneumonia and bronchitis [37]. The usual descriptions of peptic ulcer disease and GERD include epigastric discomfort and retrosternal burning, but often it is difficult to distinguish clearly between classic "heart-burn" and classic "chest pressure". Although it often is thought that symptoms of anxiety can help distinguish pulmonary diseases from other causes of chest pain, this is not a consistent finding and should not be relied upon. There is enough overlap among clinical manifestations of different causes of chest pain to make "classic" symptoms unhelpful in differentiating among diagnoses and ruling out serious causes [46]. However, there are several validated clinical decision rules that combine groups of symptoms [22]. It is important to obtain a clear history of the onset and evolution of chest pain, with particular attention to details as location, quality, duration, and aggravating or alleviating factors. Certain key symptoms and clinical findings help rule in (or) out specific diagnoses [45].

Determining whether pain is (1) sub-sternal, (2) provoked by exertion, or (3) relieved by rest or nitroglycerin helps to clarify whether it is typical Anginal pain (has all three characteristics), atypical Anginal pain (has two characteristics), or non-angina pain (has one characteristic) [44]. Anginal chest pain has a high risk for CAD in all age groups; atypical Anginal chest pain carries intermediate risk for CAD in women older than 50 years and in all men; and non-anginal chest pain carries intermediate risk for CAD in women older than 60 years and men older than 40 years [14].

The likelihood of MI is higher if there is pain radiating to both arms [5], hypotension [6] an S3 gallop on physical examination or diaphoresis [9]. Other factors predicting MI include age greater than 60 years, male sex, and prior MI [15]. MI is less likely if pain is sharp or pleuritic. If the pain is reproducible by palpation of a specific tender area, the likelihood of MI decreases but chest wall pain may increase. A history of rheumatoid arthritis or osteoarthritis also increases the likelihood of chest wall pain [16].

The Rouan decision rule reliably predicts patients with chest pain and normal or nonspecific electrocardiogram (ECG) at higher risk for MI. However, because up to 3 percent of patients initially diagnosed with a non-cardiac cause of chest pain suffer death or MI within 30 days of presentation, patients with cardiac risk factors such as male sex, greater age, diabetes, hyperlipidemia, prior CAD, or heart failure warrant close follow-up [17] (Table 1).

(One point for each clinical characteristic).	
Age greater than 60 years	
Diaphoresis NOTE: At no level of risk MI	
History of MI or angina can be completely ruled out	
Male sex	
Pain described as pressure	
Pain radiating to arm, shoulder, neck, or jaw	
Risk of MI (%)	**Score***
Up to 0.6	0
Up to 3.4	1
Up to 4.8	2
Up to 12.0	3
Up to 26.0	4

***Table 1**: Rouan decision rule for myocardial infarction clinical characteristics.*

Heart failure alone is an uncommon cause of chest pain, it may accompany acute coronary syndrome, valve disease or MI. Almost all patients with heart failure have exertional dyspnea, so the absence of exertional dyspnea is helpful in ruling out this diagnosis. Important diagnostic tests when evaluating for acute coronary syndrome in Emergency room include the 12-lead ECG and serum markers of myocardial damage [7] (Figure 2).
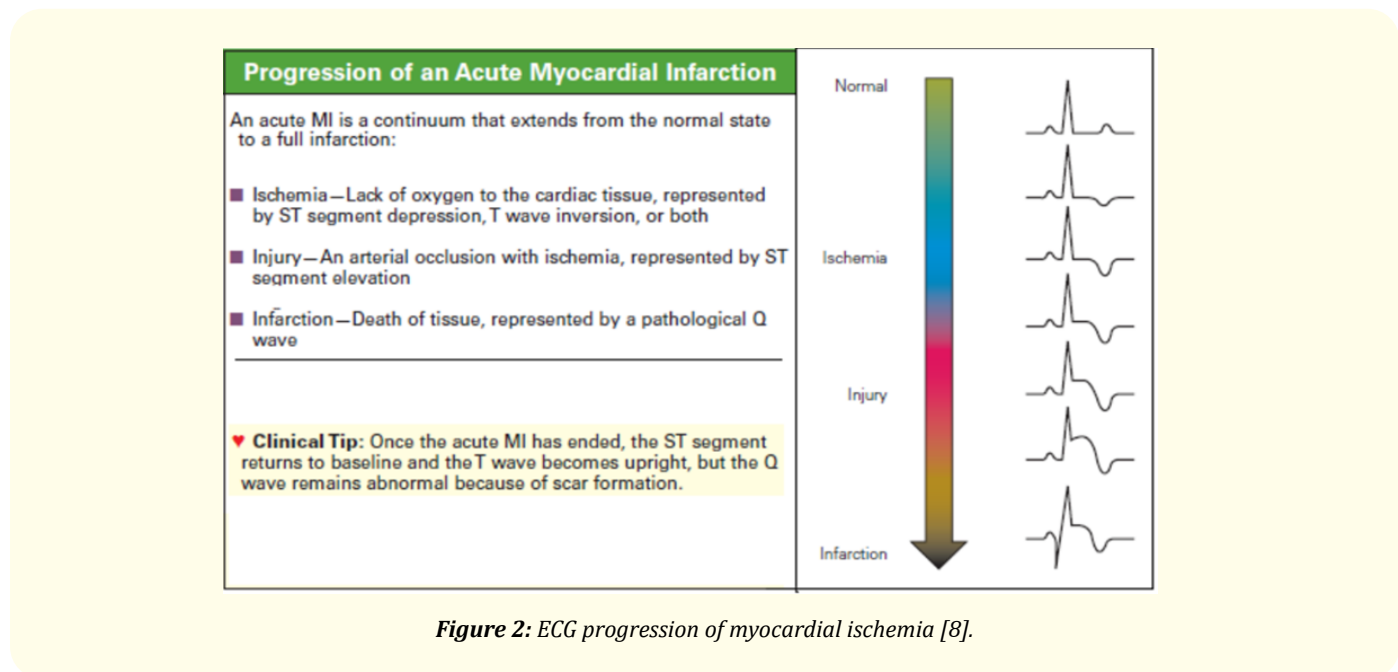

Figure 2: *ECG progression of myocardial ischemia [8].*

ECG findings that most strongly suggest MI are ST segment elevation, Q waves and conduction defect, especially if such findings are new compared with a previous ECG. New T-wave inversion increases the likelihood of MI. However, none of these findings is sensitive enough that its absence can exclude MI [56]. The most common markers of myocardial damage are creatine kinase, the MB isoenzyme of creatine kinase (CK-MB), troponin T, and troponin I. A CK-MB level greater than 6.0 ng per mL (6.0 mcg per L) within nine hours of presentation for emergency care modestly increases the likelihood of MI or death in the next 30 days [34].

Elevated levels of either troponin T (i.e. higher than 2 ng per mL [2 mcg per L]) at least eight hours from presentation or troponin I (i.e. higher than 1 ng per mL [1 mcg per L]) at least 6 hours from presentation support the diagnosis of MI or acute coronary syndrome and increase the likelihood of death or recurrent MI within 30 days [36]. A normal level of troponin T or troponin I between 6 and 72 hours after the onset of chest pain is strong evidence against MI and acute coronary syndrome, particularly if the ECG is normal or near normal [28].

In one study [29] of 773 patients who each presented to emergency departments with chest pain and had a normal ECG, researchers found that only 0.3 percent of those with a normal troponin I at six hours and 1.1 percent of those with a normal troponin T at six hours experienced acute MI or death in the 30 days following presentation. Thus, individuals with chest pain who have a history that indicates low risk of cardiovascular disease, a normal or near-normal ECG, and normal troponin levels can safely be evaluated as outpatients [37].

Patients at low risk usually do not need further testing unless there are other risk factors in their family or medical history that markedly increase their likelihood of CAD. Patients at intermediate risk for CAD who can exercise and have no left bundle branch block, preexcitation, or significant resting ST depression on their ECG can be evaluated with exercise stress ECG [27].

Patients with baseline ECG abnormalities should have perfusion imaging performed along with a stress ECG, and patients who cannot exercise may be evaluated with a pharmacologic stress or vasodilator test (e.g. dobutamine [Dobutrex], adenosine [Adenocard]). High risk patients for CAD should proceed directly to angiography, which allows definitive assessment of coronary artery anatomy for patients in whom other testing is non-diagnostic and for patients who could benefit from re-vascularization [30]. The exercise electrocardiography (ExECG) role in the ED among patients with intermediate or low risk for ACS has been tested in a number of studies [6,7].

On the basis of the collective data, it can be surmised that ExECG is a good and cost-effective test to triage patients with intermediate risk. There are, however, several limitations to ExECG: the percentage of patients who present to the ED and are unable to exercise has been reported to be as high as 35%, the rate of suboptimal exercise is largely unaccounted for in the published studies, the rate of false-positive tests is unacceptably high especially among women. Furthermore, stress imaging testing has been shown to have excellent accuracy and cost-benefit in stratifying patients in the ED [8].

Multiple imaging studies have been performed in the ED and CPU settings in patients with suspected ACS or those who were admitted with the diagnosis of acute myocardial infarction. Resting ECG have been reported to be 90% sensitive and 65% specific for the diagnosis of ACS or acute myocardial infarction [9].

A large randomized study of rest nuclear imaging in the ED showed that there was a 10% absolute reduction in hospitalization in patients with chest pain and non-diagnostic ECG when perfusion data was included in the decision-making [8].

In contrast echocardiography (CE) compared with a modified Thrombolysis in Myocardial Infarction (TIMI) score for triage of nearly 1,000 patients presenting with chest pain and non-diagnostic ECG. Both regional myocardial function and perfusion were analyzed by rest CE and related to early (in the first 24h), intermediate (at 30 days), and late (1 year) events. The comparison of CE was performed with the modified TIMI (mTIMI) score, which initially did not include serum troponin levels. Troponin levels became available during CPU stay after CE was performed.

The presence of normal regional function (RF) was most efficient in predicting lowest risk in the first 24h [23].

The lowest mTIMI score failed to identify approximately 4% of ACS (myocardial infarction), which is consistent with previous data. Further, CE also was able to classify patients with an intermediate mTIMI score into low risk (normal RF) and high risk (abnormal RF) for ACS. For a given RF finding, the presence of perfusion abnormality by CE was indicative of the highest risk whereas normal perfusion identified a very low risk (0.4%) for ACS. Even the subsequent complete TIMI score (which included the troponin level) failed to predict up to 5% of early events. These findings are a confirmation that clinical variable plus cardiac enzymes alone are insufficient to adequately triage low- and intermediate-risk patients in CPU, and the reliable discrimination would need an imaging test [23].

Which imaging test does one choose? In the case of nuclear imaging, the wealth of experience with myocardial perfusion imaging, the safety of adenosine or stress imaging, and the recent approach of using fatty acid imaging to identify ischemic memory offer tremendous advantages in the ED [11]. However, the ability to readily obtain information on left ventricular regional function and perfusion by rest CE at bedside is a significant advantage from a practical and universal applicability. Previous echocardiographic studies in the ED were per-

formed at a time when a significant proportion of transthoracic echocardiograms were suboptimal, thus limiting the clinical applicability of the approach [11]. The advent of CE and new transducer technology has had a tremendous positive impact on image quality, making transthoracic echocardiography interpretable in 90% of studies. Current transducer and equipment technology also allow myocardial perfusion imaging with little additional operator interaction [26]. The limitations of CE have to do with the traditional arguments of image quality and operator dependency of echocardiography. Contrast echocardiography has made the former problem a rarity and, with CE wall motion and thickening, can be appreciated even by a modestly trained eye. Interpretation of perfusion may be more challenging for the untrained eye. However, the growth of telemedicine means that the expert can both guide the acquisition and read the information in real time. These issues are not prohibitory to the application of CE or, for that matter, any imaging modality in the ED. Recent experimental data from work on targeted imaging using either radioisotopes or micro-bubbles add another exciting dimension to imaging in the ED [46].

For example, annexin-A5 can be bound to polymer micro-bubbles, which can then be insonated with ultrasound to image myocyte apoptosis in the infarcted myocardium [12]. It is also possible that the endothelial alterations during ischemic injury will also offer a convenient target for CE in ED in the future [13]. There are challenges to contend with implementation of imaging in the CPU protocol for triage. Patients with chest pain who present after-hours to the ED require additional resources that have to be specifically allocated, readily available, and carefully monitored (e.g., the quality of data obtained during "afterhours") [45]. The economics of imaging, whether it be rest or stress imaging in the CPU, is uncertain. Contrast echocardiography will certainly have more resource-based relative value units than standard non-contrast two-dimensional echocardiography but this would still favorably compare with resting or stress nuclear testing [44].

There is a burgeoning interest in studying the role of multislice computed tomography imaging of coronary anatomy in patients presenting to the CPU in the ED. Whether multi-slice computed tomography will cut into or complement CE or nuclear imaging remains to be seen. Cardiac magnetic resonance seems to be least practical because of the time it takes to obtain an image. Finally, the adverse clinical outcome and the fear of litigation of missed ACS in < 1% of patients have been strong deterrents to the implementation of any of the early diagnostic protocols in the ED [41].

This issue has been amplified by the uncertainty of marginal elevations of troponin in the setting of atypical symptoms and suspected ACS. All of this has further fueled the practice of watchful waiting in the CPU, with its attendant enormous cost burden. Imaging stress testing may alleviate some of this burden and arguably improve the efficiency and effectiveness of triage of patients in the CPU by bringing the cardiologist into play early in the process of clinical decision-making [42].

Therefore, it is imperative that we continue to find algorithms that may help to reliably identify the lowest-risk patients who may not even have coronary artery disease (CAD) because the economic burden of hospitalization of these patients is enormous. During the last decade, chest pain units (CPUs) in the ED were born out of such an effort to triage patients with intermediate and low-risk for ACS [5].

Typically, these units hold patients for as long as 12 h for clinical observation, during which time serial ECGs and cardiac enzymes are performed. A positive evaluation leads to hospitalization, whereas a negative evaluation leads to stress testing or discharge without stress testing [18].

Risk factors and pathophysiology

A risk factor for CAD is any characteristic or behavior that increases chances for developing coronary artery disease and its complications. Some risk factors can be controlled, or modified (hypertension, DM, cigarette smoking, excess weight.), but others cannot (age, sex, family history). Risk factors interact with each other, the more risk factors; the greater chances of developing coronary disease (Table 2).

Non modifiable risk factors	Modifiable risk factors
Advanced Age	Hypertension
Diabetes mellitus	
Gender (males are more susceptible)	Dyslipidemia
Cigarette smoking	
Race (African Americans tend to have higher blood pressure than other populations)	Excess weight
Metabolic syndrome	
Sedentary life style	
Family history	Stress
Oral contraceptives for women	

Table 2: *Risk factors for coronary artery disease [14].*

AHA/ACC consensus panel statement (1999) guide to preventive cardiology for women:

- Use of oral contraceptives is relatively contraindicated in women ≥ 35 years old who smoke.

- Women with a family history of premature heart disease are recommended to have a lipid analysis before starting oral contraceptives.

- Women with significant risk factors for diabetes are recommended to have glucose testing before starting hormonal contraceptives.

- If a woman develops hypertension while using oral contraceptives, she should be advised to stop taking them. Hormone replacement therapy (estrogen and progestin) also increases the risk for heart disease. The landmark Women's Health Initiative (WHI) found that a woman's risk of heart attack almost doubles during her first year of taking hormone replacement therapy (HRT) and levels off to an increased risk of 24% after about 6 years. Smoking dramatically increases the risk of myocardial infarction at the ages when the overall risk of this event begins to rise steeply. The combination of oral contraceptive pill use and smoking has a greater effect on risk than the simple addition of the two factors. Thus, oral contraceptive pills generally are not prescribed to smokers over 35 years of age. The risk of mortality from cardiovascular disease attributable to oral contraceptive pill use is up to 10 times higher in women 40 to 44 years of age than in women 20 to 24 years of age [9].

MI with normal coronaries

Chest pain may be associated with coronary arteries that appear "normal." Normal is defined here as no visible disease or luminal irregularities (less than 50%) as judged visually at coronary angiography. Myocardial infarction with 'normal' coronary arteries (MINCA) typically occurs in the under the age of 50 years. Normal angiography in patients with chest pain is five times more common in women than in men. Since coronary angiography developed, it has been recognized that 1 to 12% of patients may suffer from a myocardial infarction with angiographically normal coronary arteries (MINC) [10].

Usually there is no history of angina or previous myocardial infarction (MI), and risk factors for ischemic heart disease (IHD) may be absent. Symptoms and electrocardiographic (ECG) findings are similar to those of MI with angiographic coronary disease, though infarct sizes tend to be smaller. The proposed mechanisms for MINCA include coronary vasospasm, coronary thrombosis in situ or embolization from a distal source with spontaneous lysis, cocaine abuse, viral myocarditis, aortic dissection, hypercoagulable states, autoimmune vasculitis and carbon monoxide poisoning The leading cause of myocardial infarction in coronary artery disease patients is plaque rupture [12].

Most studies of MINC patients have shown that their cardiovascular risk profile is lower than that of patients with CAD. An endothelial dysfunction with a tendency toward increased vasomotor tone has also been implicated in the pathogenesis of MINCA. An interesting finding in another study was the significantly higher number of patients with febrile infections mainly of the upper airways, within 2 weeks prior to infarction in the MINC group [11].

Mortality rates are similar but morbidity is lower in myocardial infarction patients with absolutely normal coronary angiography compared with those with coronary artery stenosis. The only two independent factors predictive of poor outcome in myocardial infarction patients with normal coronary arteries are left ventricular dysfunction and diabetes. Patients with myocardial infarction and strictly normal vessels have very few ischemic events at follow-up and may be distinguished from both patients with non-significant lesions as well as those with minor angiographic irregularities. On the other hand, cardiac mortality correlates strongly and independently with a depressed ventricular function. Patients with acute MI and angiographically normal coronary arteries show a bimodal sex and age distribution: a younger age group, all men and uniformly strong cigarette smokers and an older group predominantly women with no significant association with cigarette smoking. Both groups seem to have a favorable prognosis.

Recent study concluded that young patients with myocardial infarction have good prognosis irrespective of the coronary anatomy, as patients with normal coronary angiograms had less risk factors and less frequent new ischaemic events. Q wave and non-Q wave infarctions have both been reported in patients with normal coronary arteries. Younger patients were predominantly men with a strong smoking history and a tendency to develop Q wave infarction. Older patients tended to be women who smoked less and who tended to develop non-Q wave infarcts. The overall prevalence rate of myocardial infarction with a normal coronary angiogram is low, approximately 3%, but appears to vary with age, with higher rates in young patients [13].

Coronary spasm

Coronary artery spasm is a temporary, abrupt, and focal (restricted to one location) contraction of the muscles in the wall of an artery in the heart, which constricts the artery. This spasm slows or stops blood flow through the artery. It has been proposed as a classic etiological factor of myocardial infarction with normal coronary arteries. Coronary spasm occurs most often from midnight to early morning when the patient is at rest and it is usually not induced by exercise in the daytime [14] (Figure 3).

Coronary vasospasm can produce myocardial infarction, most patients with MINCA do not have angina, and it usually affects the epicardial vessels. Certain angiotensin II type 1 receptor gene polymorphisms have been associated with an increased tendency to vasospasm in angiographically normal coronary arteries challenged with a potent vasoconstrictor. Cocaine use has been linked with MI in the absence of coronary artery disease. The proposed etiology is increased myocardial oxygen demand and paradoxical coronary vasospasm and thrombosis as a result of alpha-adrenergic action. This drug may increase concentration of plasminogen activator inhibitor, endothelial permeability to low-density lipoprotein, and platelet activation and agreeability. The fact that smokers show a decreased production
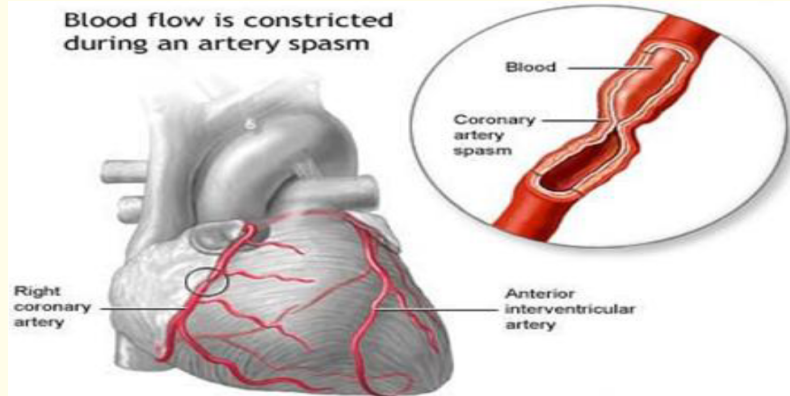
Figure 3: Blood flow in coronary artery spasm.

of nitric oxide which partly mediates endothelium-dependent vasodilatation, and because most of the patients with MINC are smokers, a pathophysiologic link may seem plausible [15].

Recanalization

In acute anterior wall myocardial infarction spontaneous coronary artery recanalization is associated with better global and segmental left ventricular systolic function, especially if the occlusion is of pre-septal localization, while collateral circulation is not related to better contractility. In acute inferior wall myocardial infarction one sees the reverse. Patency of the infarct-related artery before thrombolytic therapy or direct coronary artery angioplasty in the absence of heparin or aspirin has ranged from 9% to 28%, and similar prevalence rates have been demonstrated at 90 minutes during heparin infusion. Patients presenting with clinical features of spontaneous coronary reperfusion have a better prognosis and an excellent in-hospital outcome, with evidence of less myocardial damage than patients in whom reperfusion therapy was required to achieve TIMI-3 patency. The major threat to these patients is reinfarction due to reocclusion of the IRA since the large area of viable myocardium is potentially at risk [16].

Dissection

Spontaneous coronary artery dissection is a rare and often fatal cause of ischaemic heart disease occurring predominantly in young or middle aged otherwise healthy patients. It is mostly recognized at postmortem examination in young victims of sudden death. Aortic dissection and spontaneous coronary artery dissection can result in an MI with little evidence of coronary artery disease. As the dissection causes a "false lumen" which develops a hematoma that limits the flow of blood through the "true lumen," it leads to myocardial ischemia and infarction. While spontaneous coronary artery dissection (SCAD) is well recognized as a rare cause of chest pain, acute coronary syndrome, and sudden cardiac death, its optimal treatment is not established. SCAD is three times more likely to occur in women than in men and is often associated with per partum period, defined as the 9 months of pregnancy and up to 3 months postpartum. This was referred to the hormonal and hemodynamic changes that occur during pregnancy and the immediate postpartum period play a role, additionally, increased blood volume and cardiac output during this period increase the shear stress on the wall of the coronary vessel. About one in four female patients with spontaneous coronary artery dissection are in the per partum period. Contraceptives and the exceptional hormonal balances in the per partum period are supposed to weaken the arterial wall and to predispose it to rupture or dissection. Spon-

taneous coronary artery dissection has also been described in patients with Marfan's syndrome, with cocaine misuse, and after intense physical exercise [17].

Embolism

An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin. Most emboli are part of a dislodged thrombus (thromboembolism), but rare forms include fat droplets, air or nitrogen bubbles, atherosclerotic debris (cholesterol emboli), tumor, foreign bodies, the role of distal emboli in the etiology of MINCA is controversial. Theoretically valvular heart disease, endocarditis and mural thrombosis could predispose to embolic infarcts with subsequent re-canalization of the vessel lumen. In one study an increased incidence of mitral valve prolapse and mitral regurgitation was found in MINCA patients, but the results are hard to interpret because of small numbers and frequency of minor mitral valve prolapse in general population. Recently, prosthetic heart valves, dilated cardiomyopathy and invasive procedures like coronary angiography have increasingly been associated with embolic occlusion of coronary arteries. Acute myocardial infarction due to coronary thromboembolism from a left ventricular thrombus has been suspected in patients with dilated or aneurysmal left ventricles, but it has rarely been documented. Paradoxical embolism in a coronary artery is a recognized clinical entity, but it is rare and usually definitively established only at autopsy. Calcium embolization of the coronary artery was reported by Turcato E., *et al.* in 2000 to be the cause of myocardial infarction in two cases after percutaneous balloon mitral valvuloplasty [47].

Coronary aneurysm

Coronary artery aneurysm is an uncommon disease. It is defined as a coronary artery dilatation and exceeds the diameter of the normal adjacent segment or the diameter of the patient's largest coronary vessel by 1.5 - 2 times. The aneurysm can be divided into discrete aneurysms (localized dilatation, either saccular or fusiform) or ectasia (diffuse dilatation involving 50% of the artery). Coronary artery aneurysms are most commonly associated with atherosclerosis but also are reported with Kawasaki's disease, arteritis (polyarteritis nodosa, syphilis, systemic lupus erythematosus, and Takayasu's arteritis), mycoses, trauma, connective tissue disorders (Marfan's and Ehlers-Danlos syndromes), metastatic tumors, polycystic kidney disease, and percutaneous coronary interventions.

The pathophysiologic mechanisms that lead to development of these dilatations have not yet been clarified. Singh RB., *et al.* suggested there was an association between the chronic stimulation of endogenous nitric oxide, with consequent chronic stimulation of vascular relaxation, and the occurrence of ectatic areas in coronary arteries. Patients can present with a wide range of symptoms from being asymptomatic to sudden death. Complications include ischemia, myocardial infarction, fistula formation, spontaneous rupture, calcification, and distal embolization as a result of thrombus formation within the aneurysm. Rupture of a CAA can also cause acute myocardial infarction (AMI) and sudden cardiac death [48].

Hypercoagulability

One possible mechanism for MINCA is occlusion of the vessel lumen by thrombus that subsequently lyses rapidly. The risk of MI is above normal in people with raised plasma fibrinogen and plasminogen activator inhibitor 1. A raised homocysteine is thought to increase the concentrations of factor VII and thrombin which emphasis its role in MINCA. Moreover, Daniel Lesiège., *et al.* concluded that patients with MI, NCA and congenital coagulation disorder present a high risk of thrombosis recurrence under antiplatelet agent and recommended anticoagulation therapy in this situation. Nephrotic syndrome which is associated with proteinuria that results in the loss of low molecular weight proteins, which in turn alters the concentration and activity of coagulation factors as the liver tries to compensate for

the hypoalbuminaemic state, there is an increased synthesis of factors II, VII,VIII, X, XIII and fibrinogen resulting in raised blood levels [30]. Congenital coagulation abnormalities have been hypothesized as mechanisms of myocardial infarction with angiographically normal coronary arteries. Recently, a large multicenter study has found a higher prevalence rate of factor V Leiden, a newly described congenital coagulation disorder, in patients with myocardial infarction and normal coronary arteries, compared with myocardial infarction patients and coronary artery stenosis or healthy subjects. It has been shown that there is increased platelet consumption in young smokers without clinical evidence of coronary artery disease. This relation is presumably related to the mechanism of enhanced platelet aggregation and adhesion seen after smoking cigarettes that would be expected to increase the thrombotic risk in smokers with normal coronary arteries. It was observed in essential thrombocytosis and Polycycaemia Vera increase in the incidence of myocardial infarction with normal coronary arteries which explained by increase platelet activity beside increase platelet activation due to endothelial injury [49].

Microvascular angina

Myocardial infarction may result from occlusion of the small intramural vessels which causes are listed by Singh RB., *et al.* as follow:

- Hypertrophic cardiac disease.
- Friedriech's ataxia.
- Progressive muscle dystrophy.
- Progressive myotonic dystrophy.
- Collagen disease.
- Systemic lupus erythematosus.
- Scleroderma.
- Dermatomyositis.
- Diabetes mellitus.
- Hereditary connective tissue disorders.
- Marfan syndrome.
- Hurler' syndrome.
- Infiltrative disease.
- Amyloidosis.
- Neoplasm.
- Coagulopathy.
- Disseminated intravascular coagulation.
- Thrombotic Thrombocytopenic purpura.
- Whipple's disease [48].

Microvascular angina refers to all patients with angina, a normal angiogram, and evidence of impaired coronary microcirculation whether or not there are exercise induced ECG changes. 'Syndrome X'' describes patients with angina of exertion, a positive exercise ECG,

and a normal coronary angiogram, but excludes coronary artery spasm (Prinzmetal's angina). Also, in primary amyloidosis, obstructive intramural) coronary artery amyloid deposition can cause ischaemic symptoms. The epicardial coronary arteries are typically spared. Most patients with primary systemic amyloidosis and cardiac involvement have obstructive intramural coronary amyloidosis and associated microscopic changes of myocardial ischemia. Coronary artery affection by primary systemic amyloidosis is of bad prognosis as it is usually diagnosed during autopsy [19].

Myocardial bridging

Myocardial bridging is an anatomic variant not very common in humans, although not rare. It is characterized by the presence of one or more myocardial bundles that cross or surround a coronary artery segment. During systole the bridging causes a strangulation or constriction of the vessel, with subsequent normalization in diastole. The incidence of this anomaly is higher in women than in men. It is found in 5% - 86% in anatomic studies but only observed in 0.5% to 12% of patient undergoing coronary arteriography. The occurrence of acute myocardial infarction in myocardial bridging patients with normal coronary arteries is a very rare clinical finding. In patients with myocardial bridging and normal coronary arteries in coronary cine angiography and presenting compression of the coronary arteries during systole and decompression and normalization in diastole, the diagnosis of acute myocardial infarction must be consistently based on other associated data. The pain characteristics, electrocardiographic alterations in ST and T segments, early increase in CKMB, troponin and myosin levels should be assessed and valued [20].

Congenital anomalies

Overall, anomalies of the coronary arteries are rather rare, they may be seen with hemodynamic or myocardial perfusion abnormalities and may be manifested by sudden death.

Hemodynamically significant congenital Anomalies of coronary arteries are:

- Isolated/primary-without CHD.

- Anomalous origin of accessory coronary arteries from the pulmonary artery (ALCAPA, ARCAPA).

- Ectopic origin of the coronary arteries from aortic sinus.

- Absence of a coronary artery.

- Congenital coronary artery fistula.

- Secondary-with CHD.

- PA29+ IVS.

- AA31 + MS [21].

Myocardial ischemia is the consequence of several mechanisms limiting the blood flow in the anomalous CA, including the acute angle take-off from the aorta, the narrowed slit-like lumen with a potential for a flap like closure of the orifice, the proximal intramural course of the anomalous vessel within the aortic tunica media, which may further aggravate the obstruction, and the squeezing of the vessel along its course between the aorta and the pulmonary artery, particularly during exercise when there is an increased cardiac output with expansion of the great vessels. The origin of the left circumflex coronary artery (LCX) from the right sinus of Valsalva is one of the most

common anatomic variations of the coronary artery circulation that is associated with myocardial ischemia. Coronary anomalies have been implicated in chest pain, sudden death, cardiomyopathy, syncope, dyspnea, ventricular fibrillation, and myocardial infarction. It was found that there is a relationship between anomalous origin of left coronary artery from pulmonary artery [ALCAPA] and acute anterolateral myocardial infarction in newborns. Also if Coronary fistula cause significant coronary steal; that if occurred under resting conditions, it would cause myocardial infarction, hibernation, or resting chest pain. Left main coronary artery anomalies are rare but are also known as a cause of ischaemic heart disease, the magnitude of ischemic risk depends on the degree of angulation of the coronary artery after its origin from the aorta. This acute angulation; is often associated with a slit-like ostium that narrows with the aortic stretch with increasing cardiac output [24].

Hyperthyroidism

The cardiovascular system and thyroid hormone are closely related. The cardiovascular hemodynamic effects from the excess thyroid hormone result mainly from the direct action of T3 on cardiomyocytes, and indirect action through sympathetic effects such as an increase in sensitivity to catecholamine. The physiopathology mechanisms have not yet been totally explained. There is evidence that the thyroid hormone may affect the factors that determine the consumption of oxygen by the myocardium and that abnormalities in oxygen-hemoglobin dissociation could explain such a fact. Other possible mechanisms are: ischemia secondary to coronary vasospasm due to an unbalance in the autonomic cardiac innervation, a modification in the concentrations of thromboxane A, and prostacyclin in the coronary circulation, with insufficient vasodilatation to supply the metabolic demand [25].

Myocardial infarction in young adults

Acute myocardial infarction is rare in teenagers and young adults. The pathophysiology of their infarcts is varied but not usually due to athero-sclerotic plaque rupture except for those with genetically predetermined or familial hyperlipidemia. The incidence of CHD is declining in the UK in all age groups. The actual prevalence of the disease was found to be 0.5% in men and 0.18% in women between 35 and 44 years [24].

Causes for MI among patients aged less than 45 can be divided into four groups:

- Atheromatous CHD
- Non-atheromatous CHD
- Hypercoagulable states
- MI related to substance misuse.

The pathophysiology of myocardial infarction in the presence of "normal" coronary arteries remains unclear but can be explained on the basis of coronary artery thrombosis, embolization, spasm, or a combination of these processes. Proteinuria associated with the nephrotic syndrome results in the loss of low molecular weight proteins, which in turn alters the concentration and activity of coagulation factors Thus factors IX, XI, and XII are decreased due to urinary excretion, Increased synthesis of factors II, VII, VIII, X, XIII, and fibrinogen as a compensating mechanism resulting in rise in their blood levels. Antiphospholipid syndrome (Hughes' syndrome) Arterial and venous thrombosis is a prominent feature of this syndrome together with antiphospholipid antibodies and miscarriages of pregnancy, Antiphospholipid antibodies are associated with autoimmune diseases such as systemic lupus erythematosus, but when they occur in isolation, this is known as primary antiphospholipid syndrome. The main antiphospholipid antibodies implicated in thrombosis and atheroscle-

rosis are the anticardiolipin antibody, the lupus anticoagulant, and IgG antibodies against plasma-phospholipid binding proteins such as b2-glycoprotein I and prothrombin. Cardiac complications include myocardial infarctions and a high prevalence of valvular abnormalities of varying severity. Also, anticardiolipin antibody increases platelet adhesiveness. It is possible that the antiphospholipid antibodies predisposes to premature atherosclerosis compounding the risk for infarction with this syndrome. Coronary artery spasm is the predominant mechanism for myocardial infarction with use of cocaine. Cocaine has been associated with angina, myocardial infarction, tachyarrhythmia and Bradyarrhythmias, sudden cardiac death and myocardial contraction bands, which act as a substrate for arrhythmias [27].

The cardiac effects of cocaine are mediated via four main pathways

1. Increased myocardial oxygen demand due to an acute rise in systemic blood pressure and heart rate;
2. Coronary vasoconstriction caused by its a1-adrenergic properties and calcium dependent direct vasoconstriction;
3. Endothelial dysfunction which predisposes to vasoconstriction and thrombosis;
4. Promotion of arteriosclerosis [28].

Embolization of septic or non-septic vegetation from the aortic and mitral valves causing myocardial infarction has been reported. Myocardial bridging is associated with impeding blood flow during systole that can persist during diastole resulting in myocardial ischemia, which has been associated with myocardial infarction. Traditionally treatment involved surgical splitting of the band but there are now reports of successful treatment by stent implantation. Familial hyper-cholesterolaemia, is an autosomal dominant disorder clinically characterized by high serum cholesterol (low density lipoprotein fraction) concentrations, xanthomas, and premature atherosclerosis [29].

Various other lipid fractions and hyperhomocysteinaemia are implicated in premature atherosclerosis and myocardial infarction. Other risk factors include smoking, hypertension, insulin resistance, obesity, and a family history of premature cardiovascular events. Spontaneous coronary artery dissection is a rare cause of myocardial infarction. It is a condition with greater prevalence in young women, particularly in the per partum or early postpartum period. It also has been described in association with atherosclerotic plaque and in an idiopathic group of patients. The left anterior descending artery is often involved, but there are reports of multiple vessel involvement. Aneurysms, ectasia, and anomalous origin of coronary arteries are other causes of myocardial infarction in young adults [26].

In a series of patients who had their MI less than 45 years of age, 69% denied any chest pain before MI. The duration of symptoms was found to be less than a week in most of the patients. Evidence of significant coronary disease (mostly left anterior descending) was found in 93% of patients [30].

Summary

Presentation

Signs and symptoms

Symptoms: Dyspnea with typical or atypical crushing chest pain, sever or vary in intensity, persisting for 15 minutes or more often accompanied by sweating; may also have nausea, belching or vomiting. Resistant to analgesics or silent, relieved by rest, sublingual nitrates, morphia or not relieved. Patient may give history of similar attacks before and the way it relieved with.

Investigations

- ECG findings: No finding, ST segment elevation, Q-waves, and a conduction defect, if such findings are new compared with a previous ECG. New T-wave inversion also increases the likelihood of MI. None of these findings is sensitive enough that its absence can exclude MI.

- Elevated Cardiac Enzymes and Protein.

    - Creatine kinase (CK).

    - The MB isoenzyme of creatine kinase (CK-MB).

    - Troponin T and troponin I.

Prevention

Primary prevention (Risk factor screening)

Goal: Adults should know the levels and significance of risk factors as routinely assessed by their primary care provider.

- Begin risk factor assessment in adults at the age of 20.

- Update family history of coronary heart disease (CHD) regularly.

- Assess smoking status, diet, alcohol intake and physical activity at every routine evaluation.

- Record blood pressure (BP), body mass index (BMI), at each visit (at least every two years).

- Measure fasting serum lipoprotein profile (or total and HDL cholesterol if fasting is unavailable) and fasting blood glucose according to the person's risk for hyperlipidemia and diabetes, respectively (at least every five years; if risk factors are present, every two years).

Smoking

- Increasing the awareness of the danger of smoking among youth through media and campaigns opposing smoking that spreads to include children at schools as the age of smoking has declined to be less than 10 years.

- Encourage every smoker to quit.

- Warning against the danger of passive smoking.

Blood pressure control

Goal: Less than 120/80 mm Hg; for people who have been diagnosed with high blood pressure, the goal is less than 140/90 mm Hg; less than 130/80 mm Hg in people with renal (kidney) disease or diabetes.

- Promote healthy lifestyle modification. Advocate reducing weight; reducing sodium (salt) intake to less than 2300 mg a day; eating fruits, vegetables and low-fat dairy products; moderating alcohol intake; and at least 30 minutes of physical activity on most or all days of the week.

- For people with renal (kidney) disease or diabetes, start drug therapy if BP is 130 mm Hg or greater systolic or 80 mm Hg or greater diastolic.

- Start drug therapy for those with BP of 140/90 mm Hg or greater if BP goal is not achieved with lifestyle modifications. Add blood pressure medications, individualized to the patient's other requirements and characteristics (age, race or need for drugs with specific benefits).

Dietary intake

Goal: An overall healthy eating pattern.

- Advocate eating a variety of fruits, vegetables, grains, legumes, fat-free or low-fat dairy products, fish, poultry and lean meats.

- Match energy (calorie) intake with energy needs and make appropriate changes to achieve weight loss when needed.

- Modify food choices to reduce saturated and Tran's fats to less than 10 percent of calories, cholesterol to less than 300 mg per day, and Trans fats. (Tran's fats result from adding hydrogen to vegetable oils.) Substitute grains and unsaturated fats from fish, vegetables, legumes and nuts.

- Limit salt intake to less than 6 grams per day (2,300 mg of sodium).

Aspirin

Goal: Low-dose aspirin in people at higher risk of coronary heart disease (especially those with a 10-year CHD risk of 10 percent or greater).

- Benefits of reducing cardiovascular risk outweigh these risks in most patients with higher coronary risk.

- Consider 75 - 160 mg aspirin per day for people at higher risk (especially those with a 10-year CHD risk of 10 percent or greater).

Blood lipid management

- LDL cholesterol less than 160 mg/dL if no more than one risk factor is present.

- LDL cholesterol less than 130 mg/dL (less than 100 mg/dL is an option) if two or more risk factors are present and 10-year CHD risk is less than 20 percent.

- LDL cholesterol less than 100 mg/dL (less than 70 mg/dL is an option for very high-risk patients) if two or more risk factors are present or higher or if person has diabetes. Secondary goals (if LDL cholesterol is at goal range): If triglycerides are greater than 200– 499 mg/dL then use non-HDL cholesterol as a secondary goal.

- Non-HDL cholesterol less than 190 mg/dL for no more than one risk factor.

- Non-HDL cholesterol less than 160 mg/dL for two or more risk factors.

- Non-HDL cholesterol less than 130 mg/dL for diabetes or for two or more risk factors.

- If LDL cholesterol is above goal range:

    - Start therapeutic lifestyle changes diet to lower it: less than 7 percent of calories from saturated fat and less than 200 mg per day of dietary cholesterol.

    - If more LDL cholesterol lowering is needed, add dietary options (plant stanols/sterols not to exceed 2 g per day and/or soluble fiber 10 - 25 g per day); emphasize weight reduction and physical activity.

    - Rule out secondary causes of high LDL cholesterol (liver function tests, thyroid function tests, and urinalysis).

- After 3 months of TLC, consider LDL-lowering drug therapy if:

    - Two or more risk factors are present, and LDL cholesterol is 130 mg/dL or greater.

    - No more than one risk factor is present, and LDL cholesterol is 190 mg/dL or greater.

- Start drugs and advance dose to bring LDL cholesterol into range, usually with a statin, but also consider bile-acid-binding resin or niacin.

- If the LDL cholesterol goal is not achieved, consider combination drug therapy (statin plus resin or statin plus niacin).

- After LDL cholesterol goal has been reached, consider triglyceride level:

    - If triglycerides are 150–199 mg/dL, treat with therapeutic lifestyle changes (TLC).

    - If triglycerides are 200–499 mg/dL, treat high non-HDL cholesterol with TLC and, if needed, consider higher doses of statin or adding niacin or fibrate.

    - If triglycerides are 500 mg/dL or greater, treat with fibrate or niacin to reduce the risk of pancreatitis.

- If HDL cholesterol is less than 40 mg/dL in men and less than 50 mg/dL in women, start or intensify TLC. For higher-risk patients, consider drugs that raise HDL cholesterol (niacin, fibrates, statins).

Physical activity

Goal: At least 30 minutes of moderate-intensity physical activity on most, and preferably all, days of the week.

- If a patient has suspected cardiovascular, respiratory, metabolic, orthopedic or neurological disorders, or is middle-aged or older and sedentary, he or she should consult a physician before starting a vigorous exercise program.

- Moderate-intensity activities (40 to 60 percent of maximum capacity) are equivalent to a brisk walk (15 - 20 minutes per mile).

- Vigorous-intensity activities (more than 60 percent of maximum capacity) offer added benefits.

- Recommend resistance training with eight to 10 different exercises, 1 - 2 sets per exercise, and 10 - 15 repetitions at moderate intensity on two or more days per week.

- Include flexibility training and an increase in daily lifestyle activities to round out the regimen.

Weight management

Goal: Achieve and maintain desirable weight (body mass index 18.5 - 24.9 kg/m^2). When a person's BMI is 25 kg/m^2 or higher, the waist measurement goal is less than 40 inches for men, and less than 35 inches for women:

- Start a weight-management program through restricting calories in diet and increasing caloric expenditure (exercise) as appropriate.

- For overweight or obese persons, reduce body weight by 10 percent in the first year of therapy.

Diabetes management

Goal: HbA1c of less than 7 percent:

- Start appropriate therapy to achieve near-normal fasting plasma glucose or as indicated by near-normal HbA1c. The first step is diet and exercise.

- Second-step therapy is usually oral hypoglycemic drugs: sulfonylureas and/or metformin with ancillary acarbose and thiazolidinedione. Third-step therapy is insulin.

- Treat other risk factors more aggressively. For example, change BP goal to less than 130/80 mm Hg for patients with high blood pressure, and LDL cholesterol goal to less than 100 mg/dL or lower.

Secondary prevention: Identifying and treating people with established disease and those at very high risk of developing cardiovascular disease.

- Treating and rehabilitating patients who've had a heart attack or stroke to prevent another cardiovascular or cerebrovascular event.

What can secondary prevention achieve?

- Extend overall survival.

- Improve quality of life.

- Decrease need for interventional procedures such as angioplasty and bypass grafting.

- Reduce the incidence of subsequent heart attack (myocardial infarction).

- Heart or stroke patients can do this to help lower their risk of recurring disease:

- An assessment of fasting lipid profile.

- 30 - 60 minutes physical activity, preferably daily, or at least five days / week.

- Weight adjustment to the ideal, by sticking to a diet and exercise program.

- Checking blood pressure regularly. Adjustment by medication. Weight control, physical activity, modifying sodium (salt) intake.

- Considering aspirin intake daily or another medication.

- Nicotine replacement methods and formal programs to help quitting smoking.

Smoking

Goal: Complete cessation.

Intervention recommendations

- Ask about tobacco use status at every visit.

- Advise patient and family members to quit.

- Assess the tobacco user's willingness to quit.

- Assist by counseling and developing a plan for quitting.

- Arrange follow-up, referral to special programs, or pharmacotherapy (including nicotine replacement and bupropion).

- Urge avoidance of exposure to environmental tobacco smoke at work and home.

Blood pressure control

Intervention recommendations

- For all patients, initiate or maintain lifestyle modification (weight control, increased physical activity, alcohol moderation, sodium reduction, and emphasis on increased consumption of fresh fruits, vegetables and low-fat dairy products).

- For patients with blood pressure 140/90 mm Hg or greater (or 130/80 mm Hg or greater for individuals with chronic kidney disease or diabetes): As tolerated, add blood pressure medication, initially treating with beta blockers and/or ACE inhibitors, with addition of other drugs such as thiazides as needed to achieve goal blood pressure.

Lipid management

- Start dietary therapy. Reduce intake of saturated fat (to less than 7 percent of calories) *trans*-fatty acids, and cholesterol (to less than 200 mg dietary cholesterol per day).

- Adding plant stanol/sterols (2 grams/day) and viscous fiber (more than 10 grams/day) will further lower LDL-C.

- Promote daily physical activity and weight management

    - Encourage increased intake of omega-3 fatty acids in the form of fish or in capsule form (1 gram/day) for risk reduction. For treating elevated triglycerides, higher doses are usually necessary for risk reduction.

For lipid management

Assess fasting lipid profile in all patients, and within 24 hours of hospitalization for those with an acute cardiovascular or coronary event. If patients are hospitalized, initiate lipid-lowering medication before discharge as follows:

- If baseline LDL-C is 100 mg/dL or greater, initiate LDL-lowering therapy (typically with a statin).

- If on-treatment LDL-C is 100 mg/dL or greater, intensify LDL-lowering drug therapy (may require LDL-lowering drug combination [statin + ezetimibe, bile acid sequestrant, or niacin*]).

- If baseline LDL-C is 70 to 100 mg/dL, it is reasonable to treat to LDL-C less than 70 mg/dL.

- If triglycerides are 200 to 499 mg/dL, non-HDL-C# should be less than 130 mg/dL, and further reduction of non-HDL-C to less than 100 mg/dL is reasonable.

Therapeutic options to reduce non-HDL-C are:

- More intense LDL-C–lowering therapy, or

- Niacin* (after LDL-C–lowering therapy), or

- Fibrate therapy# (after LDL-C–lowering therapy)

- If triglycerides are 500 mg/dL or greater, therapeutic options to prevent pancreatitis are fibrate or niacin* before LDL-lowering therapy; and treat LDL-C to goal after triglyceride-lowering therapy. Achieve non-HDL-C to less than 130 mg/dL if possible. Patients with very high triglycerides should not consume alcohol.

*Dietary supplement niacin must not be used as a substitute for prescription niacin. It should not be used for cholesterol lowering because of potentially very serious side effects. #Non-HDL cholesterol is total cholesterol minus HDL cholesterol.

Physical activity

Goal: 30 minutes, 7 days per week (minimum goal, 5 days per week).

Intervention recommendations

- For all patients, assess risk with a physical activity history and/or exercise test, to guide prescription.

- For all patients, encourage minimum of 30 to 60 minutes of moderate-intensity aerobic activity, such as brisk walking, on most, preferably all, days of the week, supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening, and household work).

- Encourage resistance training two days per week.

- Advice medically supervised programs for high-risk patients (e.g., recent acute coronary syndrome or revascularization, heart failure).

Weight management

Goal: Body mass index (BMI) 18.5 - 24.9 kg/m^2. Waist circumference less than 40 inches in men and less than 35 inches in women.

Intervention recommendations

- Calculate BMI and/or waist circumference on each visit and consistently encourage weight maintenance/reduction through appropriate balance of physical activity, caloric intake and formal behavioral programs when indicated to maintain/achieve a BMI between 18.5 and 24.9 kg/m^2.

- If waist circumference (measured horizontally at the iliac crest) is 35 inches or greater in women and 40 inches or greater in men, initiate lifestyle changes and consider treatment strategies for metabolic syndrome as indicated.

- The initial goal of weight loss therapy should be to reduce body weight by approximately 10 percent from baseline.

- Further weight loss can be attempted if indicated through further assessment.

*A BMI of 18.5 to 24.9 is considered as normal body weight. People with a BMI of 25–29.9 are considered overweight, while people with a BMI of 30 or greater are considered obese.

Diabetes management

- Initiate lifestyle and pharmacotherapy to achieve near normal HbA1c.

- Begin vigorous modification of risk factors (e.g., physical activity, weight management and blood pressure control and cholesterol management as recommended above).

- Coordinate diabetes care with patient's primary care physician or endocrinologist.

Antiplatelet agents/anticoagulants

- Start aspirin at 75 to 162 mg/d and continue indefinitely in all patients unless contraindicated.

- Patients undergoing coronary artery bypass grafting, aspirin should be started within 48 hours after surgery reduces saphenous vein graft closure. Dosing regimens ranging from 100 to 325 mg/d appear to be efficacious. Doses higher than 162 mg/d can be continued for up to one year.

- Start and continue clopidogrel 75 mg/d in combination with aspirin for up to 12 months in patients after acute coronary syndrome or percutaneous coronary intervention with stent placement (one month or more for bare metal stent, three months or more for sirolimus-eluting stent, and six months or more for paclitaxel-eluting stent).

- Patients who have undergone percutaneous coronary intervention with stent placement should initially receive higher-dose aspirin at 325 mg/d for one month for bare metal stent, three months for sirolimus-eluting stent, and six months for paclitaxel-eluting stent.

- Manage warfarin to international normalized ratio 2.0 to 3.0 for paroxysmal or chronic atrial fibrillation or flutter, and in post–myocardial infarction patients when clinically indicated (e.g. atrial fibrillation, left ventricular thrombus).

- Use of warfarin in conjunction with aspirin and/or clopidogrel is associated with increased risk of bleeding and should be monitored closely.

Renin-angiotensin-aldosterone system blockers

Intervention recommendations

Angiotensin-converting enzyme (ACE) inhibitors:

- Start and continue indefinitely in all patients with left ventricular ejection fraction of 40 percent or less and in those with hypertension, diabetes or chronic kidney disease, unless contraindicated.

- Consider for all other patients.

- Among lower-risk patients with normal left ventricular ejection fraction in whom cardiovascular risk factors are well controlled and revascularization has been performed, use of ACE inhibitors may be considered optional.

Angiotensin receptor blockers

- Use in patients who are intolerant of ACE inhibitors and have heart failure or have had a myocardial infarction with left ventricular ejection fraction of 40 percent or less.

- Consider in other patients who are ACE-inhibitor intolerant.

- Consider use in combination with ACE inhibitors in systolic-dysfunction heart failure.

Aldosterone blockade

- Use in post-myocardial infarction patients who do not have significant kidney dysfunction or elevated serum potassium, who are already receiving therapeutic doses of an ACE inhibitor and beta blocker, have a left ventricular ejection fraction of 40 percent or less, and have either diabetes or heart failure.

Beta blockers

- Start and continue indefinitely in all patients who have had myocardial infarction, acute coronary syndrome, or left ventricular dysfunction with or without heart failure symptoms, unless contraindicated.

- Consider chronic therapy for all other patients with coronary or other vascular disease or diabetes unless contraindicated.

Treatment
Initial measures

- Oral nitrates and Aspirin
- ECG within 5 minutes of arrival
- History and examination including BP both arms
- IV cannula

- Oxygen if Sa0$_2$ < 98% (unless history of COPD), pain relief with GTN, morphine or diamorphine, metoclopramide if pain still present

- FBC, coagulation, U+E, glucose, lipids, LFT, troponin, CK, CXR

- Consider other diagnoses e.g. PE, aortic dissection, pneumothorax.

Risk stratification

Calculate TIMI risk score.

Low risk (TIMI risk score 0-2)

If the TIMI risk score is low and troponin is not elevated, aim for early discharge:

- Normal ECG, age < 40 and 0 - 1 risk factors (DM, smoking, FH premature CAD, HTN, hypercholesterolemia, PVD) - consider alternative diagnoses. GP follow-up

- Normal ECG, age > 40 or ≥ 2 more risk factors - arrange early exercise test. Refer to cardiac assessment and Discharge with aspirin 75 mg and GTN spray with advice to return if recurrent symptoms

- Non-diagnostic (known pre-existing ECG abnormalities) or uninterpretable ECG (e.g. bundle branch block, LVH) - refer to cardiac assessment If angina is suspected. Discharge with aspirin 75 mg od and GTN spray with advice to return if recurrent symptoms.

Moderate - High risk (TIMI risk score 3 - 7)

If the patient has ECG or cardiac marker evidence of an ACS or if in the opinion of the admitting physician this is felt to be likely, treatment should be initiated immediately on admission.

Treatment should consist of:

- Aspirin 300 mg then 75 mg od.

- Clopidogrel 300 mg then 75 mg od.

- Fondaparinux 2.5 mg od s/c (see guideline WAHT-CAR-042).

- Beta blocker (e.g. bisoprolol 2.5 - 5 mg od) titrated till HR<60 bpm.

- If beta blocker contra-indicated diltiazem may be used though rate control is less effective.

- Statin - the default for ACS cases is Atorvastatin 80 mg od. Use simvastatin 40 mg od if there are concerns regarding tolerability of high dose statin therapy.

- ACE inhibitor (e.g. ramipril 1.25 mg bd titrated to 5 mg bd) started the day after admission if BP > 100 and creatinine < 200.

- IV nitrates if still in pain or ECG evidence of ischemia.

Additional therapies

- IIbIIIa inhibitors - In the highest risk patients, or if there is evidence of recurrent chest pain with dynamic ECG changes (especially ST depression), use glycoprotein IIbIIIa inhibitor infusion

- Omega-3 fatty acids - Omacor 1 gm od improves prognosis when started within 3 months of a myocardial infarction, predominantly by reducing sudden cardiac death.

- Aldosterone antagonists: If heart failure with LV impairment present, consider spironolactone 25 - 50 mg od or eplerenone 25 - 50 mg od. Contra-indicated in hyperkalaemia or renal failure (Cr>200 μmol/l). Monitor potassium.

Nursing

- Transfer high risk ACS patients (TIMI risk 5 - 7) to CCU.

- Manage moderate risk ACS patients initially on MAU with ECG monitoring if no cardiology bed available, but aim to transfer to Laurel 1/CCU as soon as possible.

Cardiac catheterisation

- All patients at high or moderate risk with an elevated troponin or dynamic ST depression >1mm should undergo in-patient coronary angiography and revascularisation unless there are contra-indications. Refer to cardiology/cardiology assessment sisters within 24 hours.

- Even in the absence of an elevated troponin or dynamic ST changes, patients with a TIMI risk score 3-7 may still be best managed by in-patient coronary angiography. Refer to cardiology assessment within 24 hours.

- Emergency cardiac catheterisation may be required if there are on-going or recurrent symptoms with dynamic ST changes or haemodynamic instability. Consult with a cardiologist.

Echocardiography

- Echocardiography should be performed in all patients after MI to assess LV function. If severe LV impairment (EF < 35%), consider Holter monitoring after 3 weeks to look for non-sustained VT (≥ 3 beats at rate > 120) - consider referral for VT stimulation study and possible ICD

- If EF < 30% and QRS > 120 ms, consider referral for ICD.

Glucose control

- Intensive glucose control offers benefits in patients admitted with MI.

- IV insulin and glucose in all patients with STEMI and admission glucose > 11.0 mmol/l for 24 - 48 hours. Contact dialectologist when patients started on insulin.

- For patients known to have diabetes not treated with insulin, a period of insulin treatment is advised. Convert to s/c insulin (e.g. Novomix 30 bd regime) after 24 - 48 hours.

- For patients known to have diabetes treated with insulin, convert to usual s/c insulin after 24 - 48 hours and monitor control.

- For patients not known to have diabetes, stop infusion after 24-48 hours and monitor blood glucose. Contact dialectologist who will arrange a glucose tolerance test if glucose control is satisfactory.

Recommendations

"Prevention is better than cure"

Risk assessment

Goal: Adults should know the levels and significance of risk factors as routinely assessed by their primary care provider.

Smoking

Increasing the awareness, encourage every smoker to quit.

Blood pressure control

Goal: Less than 120/80 mm Hg in non-hypertensive and within the accepted range in hypertensive and diabetics.

Dietary intake

Goal: An overall healthy eating pattern.

Aspirin

Goal: Low-dose aspirin in people at higher risk of coronary heart disease.

Blood lipid management

Keeping LDL, HDL and Cholesterol in levels of the safe zone

Rule out secondary causes of high LDL cholesterol (liver function tests, thyroid function tests, and urinalysis).

Physical activity

Goal: At least 30 minutes of moderate-intensity physical activity on most, and preferably all, days of the week.

Weight management

Goal: Achieve and maintain desirable weight (body mass index 18.5 - 24.9 kg/m^2).

Diabetes management

Goal: HbA1c of less than 7 percent.

Secondary prevention: Identifying and treating people with established disease and those at very high risk of developing cardiovascular disease.

Smoking

Goal: Complete cessation.

Blood pressure control

Control blood pressure via lifestyle modification and medications.

Lipid management

Dietary therapy. Reduce intake of saturated fat, *trans*-fatty acids, and cholesterol, medications may be used.

Physical activity

Goal: 30 minutes, 7 days per week (minimum 5 days/week) if not contraindicated.

Advice medically supervised programs for high-risk patients (e.g. recent acute coronary syndrome or revascularization, heart failure).

Weight management

Goal: Body mass index (BMI) 18.5 - 24.9 kg/m^2. Waist circumference less than 40 inches in men and less than 35 inches in women.

Diabetes management

Initiate lifestyle and pharmacotherapy to achieve near normal HbA1c.

Antiplatelet agents/anticoagulants

- Start aspirin at 75 to 162 mg/d and continue unless contraindicated.

- Manage warfarin to international normalized ratio 2.0 to 3.0 for paroxysmal or chronic AF or flutter, and in post-myocardial infarction patients when clinically indicated (e.g. atrial fibrillation, left ventricular thrombus).

- Use of warfarin in conjunction with aspirin and/or clopidogrel is associated with increased risk of bleeding and should be monitored closely.

Renin-angiotensin-aldosterone system blockers

- For all other patients but for lower-risk patients are low risk and after recanalization, it is optional.

Angiotensin receptor blockers

- Use in ACE inhibitors intolerant having heart failure or myocardial infarction with left ventricular ejection fraction of 40 percent or less. Or it may be used in combination with ACE inhibitors in systolic-dysfunction heart failure.

Aldosterone blockade

- Use in post-myocardial infarction patients who do not have significant kidney dysfunction or elevated serum potassium, who are already receiving therapeutic doses of an ACE inhibitor and beta blocker, have a left ventricular ejection fraction of 40 percent or less, and have either diabetes or heart failure.

Beta blockers

- Start and continue indefinitely in all patients who have had myocardial infarction, acute coronary syndrome, or left ventricular dysfunction with or without heart failure symptoms, unless contraindicated.

- Consider chronic therapy for all other patients with coronary or other vascular disease or diabetes unless contraindicated.

Treatment

Initial measures

- Oral nitrates and Aspirin

- ECG within 5 minutes of arrival

- History and examination including BP both arms

- IV cannula

- Oxygen if $Sa0_2$ < 98% (unless history of COPD), pain relief with GTN, morphine or diamorphine, metoclopramide if pain still present

- FBC, coagulation, U+E, glucose, lipids, LFT, troponin, CK, CXR

- Consider other diagnoses e.g. PE, aortic dissection, pneumothorax.

Risk stratification: Calculate TIMI risk score.

Low risk (TIMI risk score 0 - 2)

If troponin is not elevated, aim for early discharge.

Moderate - High Risk (TIMI risk score 3 - 7)

If ECG or cardiac marker evidence of an ACS or if in the opinion of the admitting physician this is felt to be likely, emergency treatment should be initiated immediately on admission as mentioned above, admit and consult Cardiologist.

If troponin elevated or dynamic ST depression >1mm should undergo in-patient coronary angiography and revascularization unless there are contra-indications. Refer to cardiology/cardiology assessment sisters within 24h.

Emergency cardiac catheterization may be required if there are on-going or recurrent symptoms with dynamic ST changes or hemo-dynamic instability. Consult with a cardiologist.

Echocardiography

Echocardiography is needed in all patients after MI to assess LV function.

Glucose control

Intensive glucose control offers benefits in patients admitted with MI.

Other recommendations

1. Trying to prevent the incidence of myocardial ischemia by proper awareness and life style modification.

2. Early detection of risky patient via risk stratification and early proper management and treatment.

3. Being updated by knowing and following the most recent approved international guidelines in treating myocardial ischemia.

4. Doing more and more studies and researches about myocardial ischemia in chest pain patient in emergency department and sharing the results to help other physicians in helping patients.

Conclusion

"Prevention is better than cure" Decline in death rates could be achieved by adopting a healthier lifestyle. That's why it's important for healthcare professionals to implement primary and secondary prevention.

The Initial Measures in Emergency Department Included: Oral nitrates and Aspirin, ECG within 5 minutes of arrival, History and examination including BP both arms, IV cannula, Oxygen if SaO_2 < 98% (unless history of COPD), pain relief with GTN, morphine or diamorphine, metoclopramide if pain still present, FBC, coagulation, U+E, glucose, lipids, LFT, troponin, CK, CXR and Consider other diagnoses e.g. PE, aortic dissection, pneumothorax.

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