



## EMC 370 Introduction to Medical Emergencies

### 23 Chest Pain

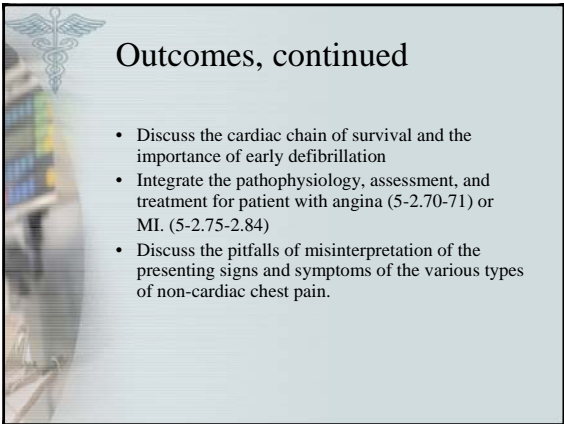
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## Outcomes

At the completion of this lecture, the learner will be able to:

- Describe cardiovascular disease incidence, morbidity, mortality and prevention strategies for reducing them. (5-2.1-2)
- Describe the epidemiology, morbidity, and mortality and pathophysiology of angina (5-2.65) and myocardial infarction. (5-2.72)
- Differentiate the characteristics of the pain of the angina and myocardial infarction. (5-2.77)



## Outcomes, continued

- Discuss the cardiac chain of survival and the importance of early defibrillation
- Integrate the pathophysiology, assessment, and treatment for patient with angina (5-2.70-71) or MI. (5-2.75-2.84)
- Discuss the pitfalls of misinterpretation of the presenting signs and symptoms of the various types of non-cardiac chest pain.



## Ischemic Heart Disease

- Leading cause of death in adults in US.
- 5 million ED visits annually due to symptoms compatible with acute chest pain.
- 40% of these rule in for ACS
- Total annual CAD cost: over \$100 billion
- CAD deaths annually: 600,000
- 60% of these die prior to hospital arrival
- great potential for improved mortality
- minimizing time from injury to treatment
- early defibrillation



## Early Defibrillation

- Depends on public education, first responders, and public access to equipment.
- Informed 1<sup>st</sup> responders
- In the future perhaps
  - informed citizens (even children)



## Sudden Cardiac Arrest

- Sudden Cardiac Arrest (SCA)
- more than 350,000 deaths / year
- usually the result of Ventricular Fibrillation
- The American Heart Association estimates as many as 100,000 deaths / year could be prevented through the widespread deployment of defibrillators.
- Survivors of Sudden Cardiac Arrest
  - excellent long-term prognosis
  - 60% of survivors live  $\geq$  five years



## SCA and AEDs

- Studies show cardiac victims' survivability decreases by about 10% every minute after a sudden cardiac arrest.
- Meaning rescuers have, at most, 8-10 minutes to defibrillate.
- Unfortunately, emergency response often takes 12 minutes or longer
- 90% of all cardiac arrests occur at work, home, or in a public setting.



## SCA and AED's


- In several studies, cardiac arrest was treated almost immediately with portable defibrillators:
- survival rates
  - at casinos
    - 53 percent
  - on airliners
    - 40 percent
- Both are far greater than overall rates in the US
  - a dismally low 2-5% survival rate
- According to the PADL (Public Access Defibrillation League ) Website [www.padl.org](http://www.padl.org)



## School Age

Could School Kids Do Better Than We Do?

- A study in 1999 showed sixth-grade students could follow the directions and took only 27 seconds longer to test-defibrillate than emergency medical technicians or paramedics.



## Dysrhythmias

- The prehospital phase is the period associated with the highest incidence of dysrhythmias.
- Treatment is indicated if the dysrhythmia:
  - exacerbates myocardial ischemia
  - Could potentially deteriorate into cardiac arrest
- Treatment guidelines will be discussed in Lecture 24.




## Assessment and Stabilization

- ABC's / COMEBIG
- Risks:
  - sudden death
  - worsening myocardial ischemia / infarct




## Lidocaine

- Prophylactic lidocaine is NO LONGER administered:
  - to decrease the incidence of ventricular ectopy
  - For primary ventricular fibrillation associated with acute MI
- Lidocaine may be harmful in the setting of an acute inferior wall MI with bradydysrhythmia.



### Clinical Risk Factors


- Classical risk factors are not critical at the present moment.
- Prognosis-based stratification
  - risk-based prehospital + ED decision making



### Classical Presentations


Substernal chest pressure, with

- N/V
- dyspnea
- diaphoresis
- radiation of pain
  - to medial aspect of one or both arms
  - to neck
  - to jaw




### Atypical Presentations

- Chest pain or discomfort that is **NOT** substernal chest pressure.
- With or without any of the other classical associated symptoms.
- Abdominal pain
- No pain or discomfort
- No associated symptoms



### Silent MI

- Silent MI accounts for approximately 20% of all MI's
- Vague complaints
- The following patients should be suspect for atypical/silent MI
  - Diabetes
  - Female
  - Hypertension
  - Alcoholism
  - Elderly



### Risk Factors for MI


- Male sex
- Increasing age
- Tobacco
- Hypertension
- Diabetes mellitus
- Hyperuricemia
- Hypercholesterolemia
- Obesity
- Prior history of ACS
- Family history of ACS



### Diagnosis


A patient can still have an MI

- with no chest pain
- with a normal EKG
- with normal initial enzymes



## History


- Patients with a suspicious / high risk history and with a normal EKG and enzymes should always be transferred (and admitted) with a diagnosis of “rule out ACS”.
- typically, about 60% of these patients
  - will **not** have a diagnosis of “Acute MI” on discharge
  - about 40% **will** have a diagnosis of ACS or MI



## History


Careful history: suspicious / high risk

- Sensitivity (true positives / all those **with** CAD-ACS)
  - 90-95 %
- Specificity (true negatives / all those **without** CAD-ACS)
  - ~ 70 %




## Initial EKG and Cardiac Markers

- Sensitivity
  - 60-85 %
- Specificity
  - ~ 70 %




## Response to Medication

- Clinical improvement
- after nitroglycerin or an antacid
- does **NOT** rule out acute MI




## EKG Changes

- About one third of patients with an acute MI can have normal EKG.
- Another one-third may have non-specific changes




## EKG Criteria for MI

- ST segment elevation in 2 or more anatomically contiguous leads
- Precordial leads are contiguous even if they overlap divisions of the heart (e.g., V2 and V3 septal and anterior)
- $\geq 1$  mm in limb leads
- $\geq 2$  mm in precordial leads
- Chest pain when accompanied by ST segment elevation yields 90% probability of ACS (though not necessarily MI).




### EKG Changes - Probability

- Convex ST segment elevation
- With ST depression in the reciprocal leads
- Plus a history of acute chest pain is diagnostic of transmural infarction




### EKG Changes for MI

- Acute Lateral Wall MI
  - ST segment elevation in I, AVL, and V4-V6
  - with ST depression in III and AVF
- Acute Anterior Wall MI
  - ST segment elevation in I, AVL and V1-V3
  - with ST depression in II, III, and AVF
  - greater risk of dysrhythmias and pump failure




### EKG Changes for MI

- Acute Inferior Wall MI
  - ST segment elevation in II, III, and AVF
  - with ST depression in I, AVL, and V1-V4
- Acute Right Ventricular MI
  - ST segment elevation in II, III, AVF, V1 and right V4 (V4R)




### EKG Changes for MI

- Acute Posterior MI
  - ST segment depression in V1 and V2
  - tall R waves in V1 and V2



### ACS Complications


- Pain
- Evolution of infarct / ischemia
- Stable and unstable arrhythmias
- Bradycardia
- Sinus tachycardia
- Tachydysrhythmias
- CHF / cardiogenic shock
- Right Ventricular MI



### AV Blocks


- First degree AV block and Mobitz I second degree AV block
  - Generally due to increased vagal tone
  - Progression to complete AV block is rare
  - Usually associated with Inferior MI
  - Generally respond to medical therapy
- Mobitz II second degree AV block
  - generally due to destruction of infranodal conduction tissue
  - progression to complete AV block may occur suddenly
  - usually associated with anterior MI
  - pacemaker therapy is indicated






## Causes of Heart Failure

- Causes of left ventricular failure (LVF)
  - myocardial ischemia or infarction
  - tachydysrhythmias
  - overexertion / hypertension
  - drugs which impair cardiac function
- Causes of right ventricular failure (RVF)
  - Left ventricular failure
  - Pulmonary HTN / COPD
  - IMI with Right Ventricular Infarct



## CHF Signs and Symptoms

- Anxiety
- Tachypnea and tachycardia
- Pale, clammy skin
- Shortness of breath (LVF)
- Cough
- Rales and/or wheezing
  - present in LVF
  - NOT present in RVF
- Hepatojugular reflux → JVD (RVF)
- Peripheral edema (RVF)




## Early Reperfusion

- Depends on public and patient education
- Informed first responders
- Rapid in field determination of best reperfusion strategy




## Thrombolytic Therapy

- Numerous agents
  - Streptokinase
  - Tissue Plasminogen Activator (tPA) (Activase)
  - Antistreplase (APSAC) (Eminase)
  - Retaplast
  - Tenecteplase
- Goal
  - door to drug < 30 min



## Thrombolytic Therapy

- These agents convert plasminogen to plasmin (its active form) which, in turn, lyses the fibrin clot in the coronary.
- thrombolysis → reperfusion of coronary artery → reduced infarction size, and increased survival.




## Thrombolytic Therapy

- Should be initiated as quickly as possible in patients who meet selection criteria.
- < 40% of patients with acute MI




## Thrombolytic Therapy

- Agent availability and administration protocols vary from service to service and hospital to hospital.
- Familiarize yourself with local protocol(s).
- Inclusion criteria
  - Based on positive EKG



## Thrombolytic Contraindications

- Active internal bleeding
- History of CVA or TIA
- Neurosurgery or trauma to head or spine within last 2 months
- History of bleeding diathesis
- Severe, uncontrolled HTN
- Presence of intracranial pathology (aneurysm, tumor, AV-malformation)
- Pericarditis or dissecting aneurysm (BP in both arms)
- Trauma or surgery within last 2 weeks
- Pregnancy
- Previous allergic reaction to lytic agent



## Summary

We have discussed:

- The epidemiology, pathophysiology, assessment, complications and treatment of acute cardiac chest pain.
- The cardiac chain of survival and the importance of early defibrillation.
- The importance of early reperfusion therapy.