Complications Post-Myocardial Infarctions
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Mechanical

1. Left Ventricular Dysfunction and Failure
   * Function of the ventricle is affected within seconds of occlusion of blood flow, and adverse remodeling can occur within the early course of an acute myocardial infarction (AMI) and continues over following months and years.
   * Results typically when the left coronary system is the culprit for ischemia
   * **Killip Classification**
     - **I** – no evidence of pulmonary congestion or shock
     - **II** – mild pulmonary congestion or an isolated S₃
     - **III** – pulmonary edema
     - **IV** – hypotension and evidence of shock
   * Management
     - Revascularization is ischemia is the primary cause of failure
     - Involves the use of diuretics to clear pulmonary congestion
     - Can involve the use of inotropic support to support systolic pressure with the goal of maintaining end-organ perfusion, and/or improve diuresis
     - Can involve the use of ACEI, if systolic blood pressure will tolerate, to afterload reduce the heart, especially with mitral insufficiency
     - Can involve the use of intra-aortic balloon pump therapy to support cardiac functioning, through increased coronary perfusion, and afterload reduction
     - The use of digoxin may have some added benefit by increasing inotropicity of the myocardium with lower filling pressures

2. Cardiogenic Shock
   * Characterized by hypotension and peripheral hypoperfusion, elevated ventricular filling pressure
   * Manifestations include mental obtundation or confusion, cold, clammy skin, and oliguria or anuria
   * When not secondary to a treatable cause such as arrhythmia, bradycardia, hypovolemia, or a mechanical defect → short term mortality is ~ 80% or greater
   * In autopsy’s AMI that involve larger that 40% of the myocardium are typically fatal
   * Management
     - Revascularization as ischemia is the primary cause of failure and time to treatment is critically important
Involves the use of inotropic support to support systolic pressure with the goal of maintaining end-organ perfusion, and/or improve diuresis

Involves the use of diuretics to clear pulmonary congestion

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3. Right Ventricular Infarction and Failure
   * Occurs with Inferior or Posterior infarctions where the right coronary or the left circumflex in a left dominant system is involved
   * Increases mortality by fivefold
   * Clinical triad include hypotension, elevated JVP with clear lung fields
   * On EKG, ST elevation in V4R had a sensitivity of 70%, with a specificity of 100%
   * Management
     ▪ Revascularization as ischemia is the primary cause of failure
     ▪ Optimize preload
     ▪ Can involve the use of inotropic support to maintain forward flow through right-side of heart with the goal of maintaining end-organ perfusion
     ▪ Can involve the use of ACEI, if systolic blood pressure will tolerate, to aid in myocardial remodeling
     ▪ The use of digoxin may have some added benefit by increasing inotropicity of the myocardium with lower filling pressures

4. Acute Mitral Regurgitation (MR)
   * Is an independent predictor of CV mortality in the postinfarction patient (relative risk of 2 – SAVE Trial)
   * Mild to moderate MR found in ~ 19% of postinfarction patients on left ventriculography due to ischemic contraction of papillary muscle
   * Severe MR results from necrosis of head of papillary muscle resulting in partial flail leaflets
   * Torrential MR results from complete rupture of head of papillary muscle secondary to necrosis which results in severe heart failure and shock (98/1190 – SHOCK Trial – more likely to be female, have NSTEMI, and have inferior or posterior than anterior infarction)
   * Clinically evident with loud pansystolic murmur, radiating to axilla, unless left ventricular function is depressed
   * Presence of shock/heart failure with preserved left ventricular function usually indicates mechanical complication present
• Left atrium may not be enlarged if acute, and will have large v waves on pulmonary capillary wedge pressure tracing
• Echocardiographic assessment is paramount
• Management
  ▪ Involves afterload reducing the left side of the heart with arterial dilators – pharmacologically with ACEI or IV nitrates if BP will tolerate or mechanically with Intra-aortic balloon pump therapy
  ▪ Revascularization is crucial with some evidence to support percutaneous coronary intervention as being superior over surgery due to high perioperative mortality (~27%) in the acute MI setting
  ▪ Severe MR due to valve disruption usually requires surgical correction

5. Ventricular Septal Rupture
• Occurs ~ in 2% of acute MI’s and is determined by location of MI and has implications for surgery
  ▪ Anterior infarction rupture is usually direct perforation over apical region
  ▪ Inferior infarction rupture occurs in posterior or basalar region with complex, serpiginous defects
• Usually occurs anywhere from 2 to 7 days
• Mortality high even with surgical repair (43 to 59%)
• Clinically, acute signs of right and left heart failure, with loud pansystolic murmur at LSB (may sound like MR), may be unimpressive with depressed left ventricular function, large proportion will have systolic thrill at LSB
• Echocardiographic assessment is important
• Mortality increases with time, generally 1% per hour
• Management
  ▪ Surgical repair early offers overall reduced patient mortality, although increase in operative mortality

6. Free Wall Rupture
• Almost uniformly fatal and accounts for 10 to 20% of inhospital deaths
• More common in older patients (> 70 years → 86% - GISSI Trial)
• Survival can occur when pericardium ‘bound down’ and limits extent of blood tracking into pericardium
• Fibrinolytic therapy can increase risk of rupture and accelerate timing to within 24 to 48 hours
• Predisposing factors include being female, having anterior MI’s, hypertension on admission, lack of reperfusion, and persistent ST elevation
• Presents as sudden collapse, PEA, and failure to respond to CV resuscitation
* May have a small warning or ‘sentinel’ rupture with symptoms of chest discomfort, impending doom, and intermittent bradycardia
* Management
  ▪ If surgical repair untaken operative mortality very high

**Pericardial**

7. **Pericarditis**
* 25% of patients with Q wave infarcts and 9% with non-Q wave infarcts will develop pericarditis, more common if there is no reperfusion
* Occurs usually within first week
* Rub may be present and only found in about half of patients with typical symptoms OR may be transient with no symptoms, but is not required for diagnosis
* Management
  ▪ If asymptomatic, no specific treatment
  ▪ If symptomatic, high dose aspirin, may use NSAID’s, but probable increased risk of rupture due to inhibition of inflammation/repair

8. **Pericardial Effusion/Tamponade**
* Effusion can be detected on echo in a quarter of acute Q wave MI’s, usually serous fluid
* Tamponade occurs rarely, and is a clinical diagnosis based on the presence of a pulsus paradoxus, and hemodynamic compromise
* Management
  ▪ If symptomatic → mechanical drainage with echo guidance depending on location and size of effusion

9. **Dessler’s Syndrome**
* Occurs 2 to 11 weeks after acute MI
* Characterized by prolonged or recurrent pleuritic chest pain, pericardial friction rub, pulmonary infiltrates, or small pulmonary effusion, and elevated ESR
* Probably due to an autoimmune reaction
* Management
  ▪ Similar to management of pericarditis

**Electrical**

10. **Accelerated Idioventricular Rhythm**
* Reperfusion arrhythmia which occurs frequently following fibrinolytic therapy
* Characterized by LBBB morphology with rate less than 120 beats/minute
* Typically, self-terminating and asymptomatic, thereby not requiring intervention

11. **Ventricular Fibrillation/Sustained Ventricular Tachycardia (VF/VT)**
* Marker of adverse prognosis if occurs after first 24 hours postinfarction and is a strong predictor of 6-week mortality (hazard ratio of 6.10 – GISSI 3)
* Risk factors include older age, hypertension, diabetes mellitus, previous MI, and not receiving fibrinolytic therapy
* Frequency of VF has markedly decreased since the ‘70’s likely due earlier and more aggressive reperfusion therapy with fibrinolytic therapy and now with early percutaneous coronary intervention, use of β-Blocker therapy, and admission of lower risk patients to the CCU with more aggressive management of electrolyte imbalances and heart failure
* Prognosis of VF depends on clinical state
  * Occurring in presence of hemodynamic compromise has hospital mortality of 80%
  * Patients surviving early inhospital VF complicating MI due to acute ischemia experience no adverse effects on long term survival following discharge
* Management
  * Lidocaine is effective, but does not improve mortality, with possible adverse effects with increased risk of asystole
  * Low serum potassium is associated with higher risk of VF ⇒ Aim to keep serum K+ > 4.0 mmol/L
  * Cardioversion/Defibrillation if hemodynamically compromised

12. **Post-infarction Ventricular Premature Beats and Non-sustained Ventricular Tachycardia (PVB/NSVT)**
* Ventricular ectopics are common post MI
* No specific therapy is required ⇒ Antiarrhythmics associated with increase in mortality; except amiodarone
* The significance of NSVT is controversial
* Treatment of these have failed to improve survival
* Frequent runs of NSVT should have evaluation by an Electrophysiologist
* Use of AICD has been shown to effective over pharmacological therapy to improve survival
13. **Atrial Fibrillation (AF)**
   - Relatively common postinfarction with 2.5% presenting with AF; and 7.9% developing AF during hospitalization
   - Underlying causes are heart failure, pericarditis, atrial ischemia, and elevated LVEDP leading to atrial stretch
   - **Management**
     - Amiodarone has been shown to be more effective than digoxin alone in achieving conversion to sinus rhythm
     - In a non-randomized trial, the combination of Amiodarone and digoxin was superior to Amiodarone alone in restoring and maintaining sinus rhythm

14. **Heart Block and Conduction Disturbances**
   - Sinus Bradycardia occurs frequently in the acute MI setting, particularly inferior infarctions
   - AV Blocks, including first, second, or third degree are also common
   - 1st degree usually results from medications that are given that slow the AV node conduction
   - 2nd degree is classified as either Type I (Wenckebach) usually associated with an inferior MI and rarely necessitates transvenous pacing, but responds well to Atropine if symptomatic; or as Type II which is typically located below the His bundle and can easily progress to complete AV Block requiring transvenous pacing
   - Complete AV Block in patients with inferior or anterior infarction are usually secondary to ischemia to both the SA and AV nodes. In anterior infarctions the block may occur suddenly, with only a 1st degree block as a warning sign. Whereas in inferior infarctions there may be 1st, or 2nd degree blocks, and junctional or ventricular escape rhythms that are asymptomatic
   - Intraventricular Conduction disturbances can occur in up to 10% of MI patients and can be uni, bi, or trifasicular blocks. Newly acquired bi or trifasicular blocks are more common in large anterior infarcts and carry a greater risk of asystole.
   - **Management**
     - Transvenous pacing indicated in persistent high grade AV blocks or advanced conduction disturbances with symptoms

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**Late**

15. **Postinfarction Angina and Myocardial Ischemia**
   - 20% of patients will develop postinfarction angina and those with early postinfarction angina are 10X more likely to extend the territory of infarction and have a worse long-term prognosis
   - **Management**
16. **Cardiac Thromboembolism**
   - Develop in up to 40% of patients with large anterior transmural MI’s
   - If left untreated, up to 15% of thrombi will dislodge and result in symptomatic embolic event
   - More common within first few months following infarction
   - Management
     - Involves anticoagulation with warfarin for 6 months if there is a documented intracavitary thrombi; and 3 months prophylactically

17. **Left Ventricular Aneurysm**
   - Develops following a large transmural anterior infarct
   - Can often be palpated as a dyskinetic region adjacent to the apical impulse, may also auscultate a S3 and observe signs of heart failure
   - A non-specific EKG marker is persistent ST elevation that persists weeks following infarction
   - Ventricular tachycardia is commonly associated with an aneurysm
   - Increases risk of thromboembolism (~5%), with the risk of being greatest within the first few weeks
   - Predisposes to ventricular arrhythmias

18. **Pseudoaneurysm**
   - Rare
   - Myocardial rupture is sealed off by surrounding adherent pericardium
   - May progressively get larger, but maintains a narrow neck
   - Most common symptoms are heart failure, chest pain, and dyspnea, but very few report symptoms (< 10%)

19. **Psychosocial Complications**
   - ~20 – 50% have anxiety, depression, denial, hostility, and feel socially isolated
   - 15 – 20% of patients hospitalized may have a major depressive disorder
   - Compound effects result from lifestyle and socioeconomic factors and can result in poor adherence to therapies which subsequently lead to poor outcomes
   - Management
     - Cardiac Rehabilitation