

Post MI Complications and Cardiogenic Shock



Case Presentation

- **A 62 yo man presented to the ER complaining of chest pain and dyspnea for 4 weeks.**
- **Severe substernal intermittent pain and pressure with diaphoresis, palpitations, progressive dyspnea, and fever.**
- **His PCP started Abx treatment for suspected pneumonia**
- **Continued to worsen and he came to the ER where an EKG was consistent with a prior inferolateral MI.**

Case Presentation

- **PMHx :**
 - HTN
- **PSHx :**
 - B/L Carpal tunnel repairs, T&A, fractured ankle repair
- **FHx**
 - Mother : deceased, Alzheimers.
 - Father : MI at 50 yo, deceased, at 90 from a “massive stroke”
 - Brother : 4 vessel CABG
- **SocHx:**
 - 1/2 pack a day x 3 years, quit 2 years ago
 - Drinks one alcoholic beverage a week
 - Employed as a mechanic
- **Medications :**
 - Norvasc 5mg daily, Aleve prn
- **NKDA**

Case Presentation

- ROS :
 - + Fevers, weight loss, exertional dyspnea
- Vital Signs :
 - T: 97° P: 119 BP 118/84 R: 18

Case Presentation

- Physical Exam :
 - Gen:
 - AAOx3 – **Mild Respiratory distress**
 - Neck:
 - No JVD or hepatojugular reflux
 - Cardiovascular:
 - **Distant heart sounds**, No murmurs, rubs or gallops
 - Pulmonary:
 - **Decreased bibasilar with slight rales at the left lower lung base**
 - Abd:
 - Soft, nontender
 - Extremities:
 - No Edema. Palpable pulses

Laboratory Findings

Troponin:

**0.59 > 0.56 > 0.55
> 0.53**

Myoglobin:

52 > 67 > 57 > 43

ProBNP: 8550

Mg: 2.0

LDH: 241

Na: 134

K : 3.4

Cl: 97

CO2: 23

BUN: 13

Cre: 0.9

Glucose: 122

WBC: 7.80

Hg: 16.2

Hct: 48.4

Plt: 313,000

ABG: 7.47/29.0/59.9/88.4/21.1

Case Presentation

- **EKG:**
 - Sinus tachycardia with an inferolateral myocardial infarction with poor R-wave progression anteriorly and some nonspecific ST-T wave changes
- **Bedside Echocardiogram:**
 - Left Ventricular Ejection Fraction of 20%
 - Anterior apical aneurysm
 - Lateral akinesis and inferior akinesis
- **CXR:**
 - Perihilar congestion, pulmonary edema pattern and small bilateral pleural effusion. Suspect acute CHF
 - **His dyspnea improved with lasix**

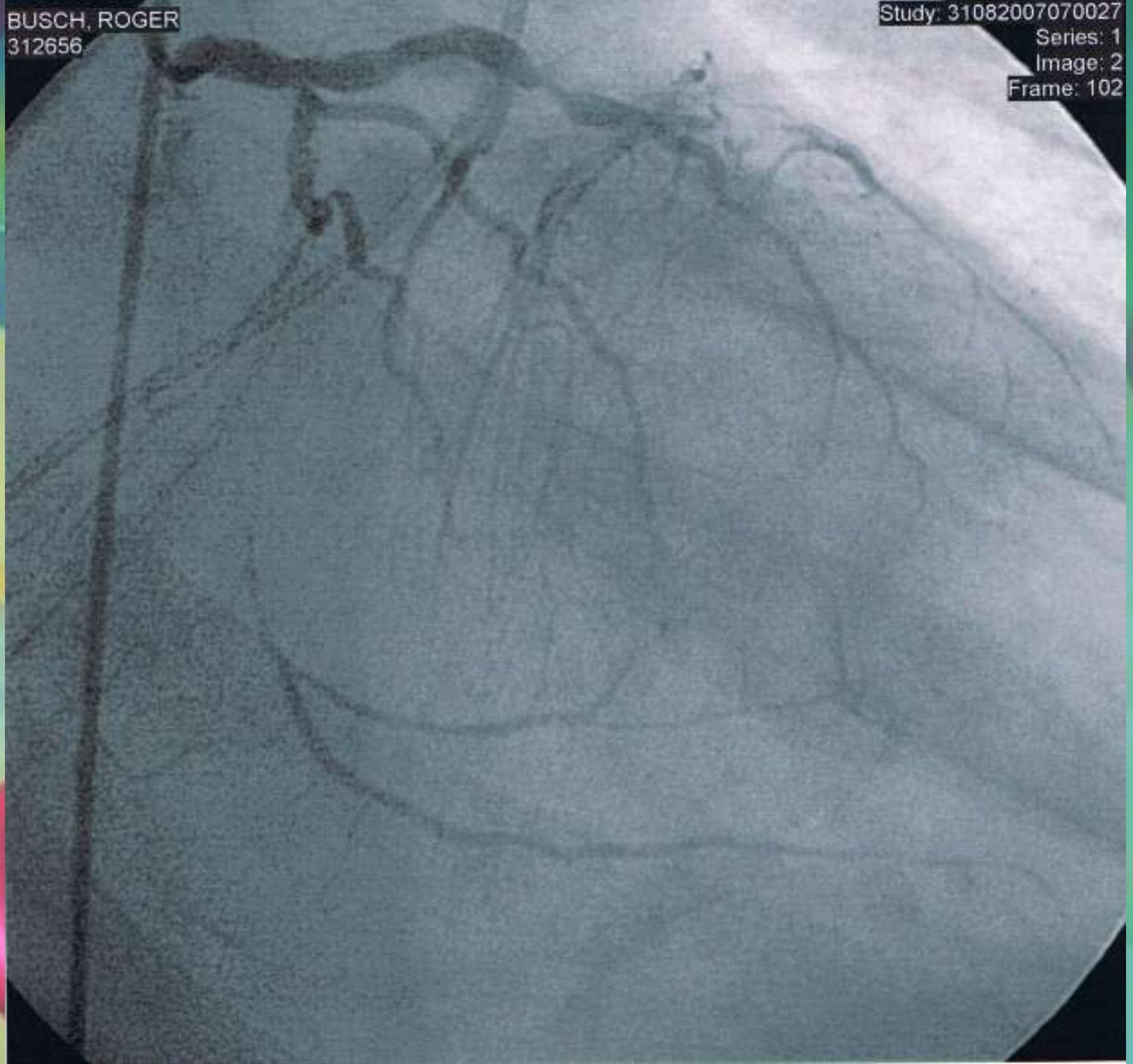
Cardiology Consult

PLAN:

- 1. We will transfer the patient to ICU secondary to his noted ischemic cardiomyopathy with ejection fraction of 20% with an elevated heart rate and low blood pressure consistent with borderline shock.**
- 2. We will hold any beta blockers at this time due to his elevated heart rate compensating to keep elevated cardiac output.**
- 3. We will start the patient on aspirin, an ACE inhibitor, Lovenox and a statin.**
- 4. We will proceed with a left and right heart catheterization in the morning.**

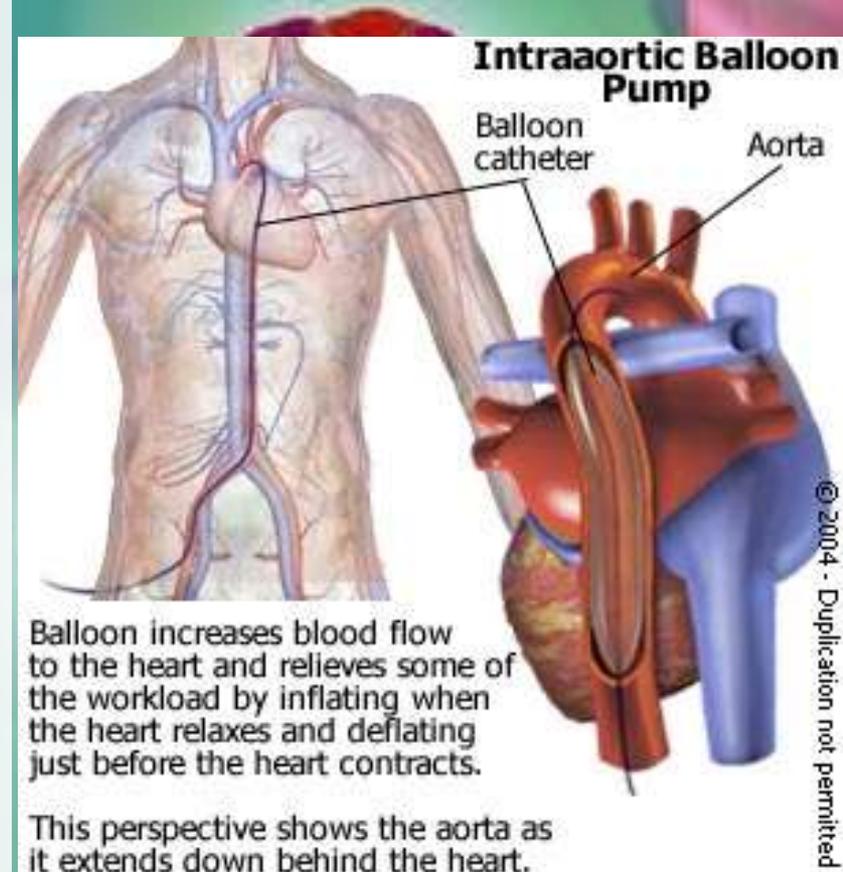
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Case Presentation

- Cardiac Catheterization
 - 100% occlusion of the LAD
 - 100% occlusion left circumflex
 - 90% RCA stenosis
 - Severe LV dysfunction with an EF of 10-15%
 - IABP placed
- Consulted CT Surgery for CABG evaluation



CT Surgery Consult

- Questioned the viability of the lateral walls
 - If minimal viability, then CABG would not improve his LV function
- Suggested transfer to TGH for either:
 - 1. High risk PCI to RCA
 - 2. high risk CABG with LV assist as a bridge to transplant

Transfer to TGH

- Pt was transferred to TGH for evaluation of possible heart transplant and/or CABG.
- Rumor has it that the Thallium Scan there showed:
 - Practically NO viable myocardium in 2/3 of the anterior wall and apex
 - 50-60% viable myocardium in the septum and inferior lateral wall
 - Anterior basal segment and basal inferior and lateral segments are viable
 - Global hypokinesis in the visible segments of the myocardium
 - LV EF is markedly decreased
- As of yesterday, he was being pre-op'd for a Cardiac Transplant

Recap

- MI 4 weeks ago
- Left Ventricle decimated, EF of 10-20%, aneurysm formation
- Shock caused by inadequate cardiac output
- Awaiting Cardiac Transplant

Post MI Complications

“ACT RAPID”

- **A**rrhythmias
- **C**ongestive Heart Failure
- **T**amponade / **T**hromboembolic disorder
- **R**upture (Ventricle, septum, papillary muscle)
- **A**neurysm (Ventricle)
- **P**ericarditis
- **I**nfection
- **D**eath / **D**ressler's Syndrome

Post MI Complications

- **Arrhythmic**
 - **Ischemic**
 - **Mechanical**
 - **Embolic**
 - **Inflammatory / Immunologic**
- 

Arrhythmic Complications

- **Dysrhythmia is the most common complication after acute MI. It is related to the formation of re-entry circuits at the confluence of the necrotic and viable myocardium.**
 - **PVCs ~ 90%**
 - **Supraventricular ~ 10%**
 - **Bradyarrhythmias – common with inferior MI**
 - **Complete AV Block ~ 20% with RV infarct**
 - **Vfib ~ 2 - 4%**

Ischemic Complications

- Patients with infarct extension or post MI angina usually have continuous or recurrent chest pain, with protracted elevation in creatine kinase (CK) and occasional new ECG changes. CK-MB is a more useful marker for tracking ongoing infarction than troponins, given their shorter half-life. Rising and falling CK-MB levels suggest infarct expansion or recurrent infarction. **Elevations of CK-MB greater than or equal to 50% more than a previous nadir are diagnostic for reinfarction.**

Mechanical Complications

- **Papillary Muscle Rupture : 13 hours post MI**
 - Acute Mitral Regurgitation
- **Ventricular Septal Rupture : 2-5 days post MI**
- **Free Wall Rupture : within 2 weeks post MI**

- **Left Ventricular Failure and Cardiogenic Shock**
- **Right Ventricular Failure**
- **Ventricular Aneurysm**

Embolic Complications

- **The incidence of clinically evident systemic embolism after MI is less than 2%. This figure increases in patients with anterior wall MIs. The overall incidence of mural thrombus after MI is approximately 20%. Large anterior MI may be associated with mural thrombus in as many as 60% of patients**

Inflammatory and Immunologic Complications

- **Pericarditis : 1-8 weeks post MI in 10%**
 - Postural Chest Pain
 - Aspirin is the therapy of choice for post-MI pericarditis in doses of 650 mg every 4 to 6 hours. NSAIDS and corticosteroids should be avoided for 4 weeks after the acute event. These agents may interfere with myocardial healing and contribute to infarct expansion.
- **Dressler's Syndrome : weeks to months in 1-3% of post MI patients**
 - Postcardiac injury syndrome (PCIS)

Dressler's Syndrome

- **The syndrome consists of a persistent low-grade fever, chest pain (usually pleuritic in nature), a pericardial friction rub, and /or a pericardial effusion. The symptoms tend to occur after a few weeks or even months after infarction and tend to subside in a few days. Signs include elevated ESR.**

Question

- A 74-year-old woman dies two days after complaining of severe chest pain. The results of EKG, echo, and laboratory studies are consistent with a clinical diagnosis of myocardial infarction. Postmortem histologic examination of the heart demonstrates an area of ischemic necrosis infiltrated by neutrophils. No infarcts were found in the brain, kidneys, or spleen. Which of the following was the most likely complication leading to this patient's death?
 - A. Arrhythmia ★
 - B. Fibrinous pericarditis
 - C. Mural thrombosis
 - D. Ventricular aneurysm
 - E. Ventricular rupture

Answer

The correct answer is A. During the first few days after a myocardial infarction, the heart is particularly susceptible to arrhythmia, which is the most common cause of death in the early stages.

Fibrinous pericarditis (B) is a frequent manifestation of a transmural infarct. The Necrotic muscle incites an acute inflammatory reaction that involves the visceral pericardium and results in fibrin deposition.

Mural thrombosis (C) develops commonly on the endothelial surface of infarcted myocardium. Thromboembolism may lead to brain infarcts and could be fatal.

Ventricular aneurysm (D) is a late complication, developing weeks after infarction when the necrotic myocardium is replaced by scar.

Ventricular rupture (E) most commonly occurs between the 5th and 8th day post MI

Trivia you'll never need to know

- **The IABP's developers thought that private insurance companies would not stand for such a radical new therapy, and approached the military, asking them to keep an eye out for IABP candidates in military hospitals. One day, the call came. A retired general was hospitalized in Walter Reed Army Medical Center. He had a history of multiple infarcts and was now end-stage. The IABP scientists were all ready to go, until they found out the general was former President Dwight D. Eisenhower. They declined the opportunity because they worried that if the IABP failed in him, it would permanently ruin the future prospects of the device.**