MI Mechanical Complication Case Based

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Mechanical Complications

• Cardiogenic Shock
• Right Ventricular Infarction
• Papillary Muscle Rupture
• Ventricular Septal Defect
• Free Wall Rupture
• Conduction Abnormalities
Right Ventricular Infarction

Pathological studies evidence of RV infarction in 20 - 50% of inferior infarcts
Significant hypotension or cardiogenic shock resulting from RV infarction occurs in approximately 10% of inferior infarcts

Clinical Findings
Classic physical findings include: hypotension, elevated JVP, clear lung fields
Right Ventricular Infarction

In-Hospital Complications

Zehender, NEJM, 1993

RV infarct and implications on Mortality

Mehta, 2001
Diagnosis

- ECG: ST segment elevation in leads V₃ or V₄R (most specific test)
- Hemodynamics: elevated right atrial and RVED pressure (>12), normal to low pulmonary pressures, low (<15) PCWP, low C.O.
- Echocardiography: RV enlargement with depressed function in setting of inferior LV hypokinesis, + TR

Differential Diagnosis

- Several equally serious conditions can present with similar findings both on clinical exam and diagnostic tests, especially pulmonary embolus and pericardial tamponade.
- PE, constrictive pericarditis and tamponade may have similar hemodynamics
- PE may have similar echo features
- Remember V₃, V₄ R
Principles of Management

- Stabilization and reperfusion are the hallmarks of treatment. If patients can survive the initial 2-5 days, RV function typically improves.
- Hemodynamic monitoring and fluid administration to achieve PAWP of 15-18 mm Hg. If patients do not respond with an increase in C.O., dobutamine should be added. IABP may be necessary in some cases.

Principles of Management

- Avoid “pushing” fluids beyond above parameters. RV overdistention can cause RV MVO₂ and actually decrease C.O. by increasing intrapericardial pressure and limiting LV filling.
- Maintenance of AV synchrony is important to maintain RV filling.
Principles of Management

• Reperfusion therapy, particularly when patency of RV branches is achieved can dramatically restore RV function and overall hemodynamic stability
• Successful reperfusion has been associated with marked reduction in mortality (2% vs 58% in 1 study) and in-hospital complications

RV Function Following Infarction

Bowers, NEJM, 1998
Potential Reasons for Improvement in RV Function

• Favorable O₂ Supply / demand characteristics
• RCA - RA pressure differential
• Thin RV wall - ? direct perfusion from RV
• Low afterload
• Greater collateral potential
  – LAD
  – Conus

Conclusions

• RV infarction significantly increases the morbidity and mortality associated with inferior MI
• Cardiogenic shock from RV infarction has a lower mortality than that of cardiogenic shock from LV infarction
• Reperfusion therapy greatly improves in-hospital morbidity and mortality from RV infarction
• Prognosis of hospital survivors is excellent and RV function generally improves over time
Things to Remember About RV Infarction

• Beware of pseudo RV infarcts (PE, tamponade, constriction)
• $V_3/V_4R$ leads are the most specific for RV infarct
• Do what it takes to support hemodynamics
• Long term survival is good and RV function will improve if patient can get through the first few days

Acute Severe Mitral Regurgitation
MR patient

- 76 M with DM, CVA x2 - last 2012- able to walk with cane, but recently has not been walking due to SOB with minimal exertion for 4-6 weeks.
- Much more SOB for last few days - even at rest. One episode of mild chest pain for ~30 minutes before coming to the ER. NO prior episode of chest pain. Still feels quite SOB.
To OR

- Patient went to OR that night with LIMA to LAD and Mitral valve replacement
- Discharged but ended up in a nursing home for rehab

MR patient 2

- 66 WM with acute cardiogenic shock s/p MI
- Pt has history of dm, htn, hld presented with 3 days of CP and SOB. Also left shoulder pain, upper back pain. Presented to the ER and diagnosed with posterior MI, taken urgently to cath lab
Before the Cardiac Cath

- Patient did not look right
- HR 100-110
- SBP 90mmHg
- Pale
- Diaphoretic
- No murmur
Outcome

- Initially IABP, then Tandem Heart
- Stabilized
- Surgery next morning
- Doing well for 3 days
- Sudden drop in BP
- Opened bedside
- Myocardial Rupture

Incidence and Etiology

- Uncommon, <1% of total infarcts, but 5-10% of patients with cardiogenic shock
- Papillary muscle dysfunction
- Partial or complete tear of the papillary muscle
- Almost always associated with inferior infarction (~90%)
- Posterior papillary muscle usually responsible due to its single blood supply from the dominant coronary artery
### Papillary Muscle Rupture vs Dysfunction

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<th>Dysfunction N=16</th>
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<td>Hypertension</td>
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<td>Diagnosis to surgery (days)</td>
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Calvo, EHJ,1997

### Acute Mitral Regurgitation

#### Papillary Muscle Rupture vs Dysfunction

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<th>Rupture</th>
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<tr>
<td>2 vessel</td>
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<td>53%</td>
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<tr>
<td>3 vessel</td>
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<td>Mortality in hosp</td>
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<tr>
<td>Mortality f/u</td>
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Calvo, EHJ,1997
Papillary Muscle Rupture - Clinical Presentation

• Rupture occurs primarily within the first week following infarction (>75% of cases)

• Acute pulmonary edema is the most common presentation. Over 1/2 of patients in Mayo series had cardiogenic shock

• Murmur of MR variable, may be minimal or absent in low output states (18% of Mayo series had no murmur)

• Apical thrill rarely present (in contrast to VSD)
Papillary Muscle Rupture - Diagnosis

• Echocardiography is the diagnostic test of choice. If severe MR is found TEE should be performed to optimally visualize papillary muscles.

• Right heart catheterization typically shows large “V” waves on the PAWP tracing

• Differential diagnosis: VSD, infarct extension with cardiogenic shock
Papillary Muscle Rupture - Diagnosis

LV, LA pressure in PM rupture

Management

• Majority of patients require mechanical ventilation
• IABP, nitroprusside and / or dobutamine for acute stabilization
• Emergent coronary angiography
• Mitral valve repair / replacement + CABG ASAP!! Surgical delay = increased mortality
Things to Remember About Acute, Severe MR

- Associated with inferior infarcts (90%)
- Posteromedial Papillary muscle usually involved
- High % single vessel disease, good EF
- Murmur may not be impressive
- Pulmonary edema, Cardiogenic shock most common presentation
- Echo, TEE best diagnostic test
- Early surgery with CABG
- Excellent long term survival in hospital survivors

Postinfarction Ventricular Septal Defect
Ventricular Septal Defect

Incidence

• Uncommon, <1% of total infarcts, but 2-5% of patients with cardiogenic shock

Anatomic Features

• ~ 55% due to inferior infarction, ~ 45% due to anterior infarction
• Inferior infarct VSD’s are located in the posterobasal region of the septum, anterior VSD’s in the apical septum
• Conduction abnormalities common (~1/3 in 1 series)
Ventricular Septal Defect

Patient Characteristics

• Mean age >65 in virtually all series
• Hypertension present in ≥50% of patients
• Typically first infarct, most patients have no antecedent angina
• High percentage of single vessel disease
• VSD usually occurs within the first week after MI, approximately 50% in the first 48 hours

Ventricular Septal Defect

Clinical Presentation

• Like acute MR most patients develop acute onset of biventricular failure or cardiogenic shock (~ 50/50)
• Classically, patients have a new holosystolic murmur and a precordial thrill
• Magnitude of L → R shunt (and characteristics of murmur) inversely proportional to size of infarct and directly related to residual LV function
Ventricular Septal Defect

Diagnosis and Management

- Echo/Doppler is the best diagnostic tool
- “Step up” in RV saturation characteristic. Large “V” waves often seen on PAWP tracing - can be confused with MR
- IABP, dobutamine, nitroprusside for acute stabilization
- Mortality 100% without surgery
- Timing of surgery remains somewhat controversial

Ventricular Septal Defect

Long Term Outcome

- Hospital survivors have very favorable long term outcome.
- 1 year survival rates are approximately 70%
- 7-10 actuarial survival 60 - 65%
- Most patients NYHA Class 1 or 2
Ventricular Septal Defect

Things to Remember About Postinfarction VSD’s

• Approximate equal distribution between anterior and inferior infarcts
• High percentage of single vessel disease, first time infarcts
• Similar presentation to acute MR but pulmonary edema less prominent
• Degree of shunt inversely proportional to size of infarct
• Right heart cath data can be misleading → Echo

Postinfarction Free Wall Rupture
• 68 year old man with history of hypertension, diabetes, smoking who was brought to the ER by the EMS after being found lethargic
• Called perfusionist
• Placed ECMO
• CV surgery

Autopsy
• Acute/subacute myocardial infarction of the posterior left ventricle with wall rupture
• Hemopericardium (150ml) with clot formation
• Cardiomegaly (453g) with biventricular hypertrophy
• Coronary artery atherosclerosis, moderate, s/p stent placement
• Small aneurysms of iliac arteries
Free Wall Rupture

- “Classic” patient: elderly (>70) female with hypertension
- Usually presents as a catastrophic event - EMD due to tamponade. Syncope and shock also common.
- Time course for rupture, prevalence of single vessel disease similar to papillary muscle rupture (PMR) and VSD
- Point of rupture typically at the juncture of normal and infarcted myocardium
- Unlike PMR and VSD, circumflex is often culprit vessel (~ 40% in 1 series)

Free Wall Rupture

- Primary PTCA and early thrombolysis may reduce the incidence of rupture
- Late thrombolysis may increase the incidence of rupture
- *Rarely*, rupture is a subacute process. With prompt diagnosis and surgery these patients may be salvageable
- Echo is the diagnostic modality of choice. Any pericardial effusion in a patient with sudden hemodynamic compromise should suggest the diagnosis. Effusions with echo dense structures (clot) characteristic
Free Wall Rupture

- Mortality in patients who make it to surgery: 33%
- Long term outlook for surgical survivors is good: 13/16 alive (mean f/u 30 mo), 11 NYHA class 1

(Purcaro, et al, AJC, 1997)

Thank you