It’s All In The Tissue:
A Rare Case of Acute Cardiogenic Shock

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Laennec Young Clinician Presentation
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History of Present Illness

• 53 y/o F with a h/o hypothyroidism presented to the emergency department with progressive dyspnea + abdominal discomfort for ~ 2 weeks

• Started after traveling to Southwest + Niagara Falls

• Symptoms included body aches, congestion, non-productive cough and subjective fevers
Medical History

Medical/Surgical History
• Insomnia
• Hypothyroidism

Allergies
• None

Family History
• HTN and Hypothyroidism in Mother

Social History
• Works as marketing manager
• Married with 3 children
• Non-smoker, social alcohol use

Medications:
• Levothyroxine 25 mcg
Physical Examination

**Vitals:** 36.2  87/69 mmHg  HR 90  RR 22  96% on 3L

**General:** Mild distress, speaking full sentences

**Cardiovascular:** Regular rhythm, normal S1, 3/6 holosystolic murmur LLSB, +S3 gallop, no rub, JVP 14 cm with prominent V waves

**Pulm:** Bilateral inspiratory crackles b/l at both bases; diminished RLL breath sounds

**Abd:** Soft, non-tender, non-distended

**Neuro:** A and O x 3, no focal deficits

**MSK:**  Cool hands/feet; 1+ pitting edema from mid-shin downwards
Total Protein: 6.1 g/dL
Albumin: 2.9 g/dL
T.Bili: 0.6 mg/dL
AST: 850 u/L
ALT: 960 u/L
Lactic Acid: 3.1 mmol/L

Creatine Kinase: 459 u/L
Creatine Kinase-MB: 35.9 u/L
Troponin-T: 3.1 ng/mL
C-Reactive Protein: 87 mg/L
TSH: 1.68 mCU/ml
Electrocardiogram
Case Synthesis

Decompensated Heart Failure

- Subacute Presentation
- ? Cardiogenic Shock
- Autoimmune Disease
- Low Voltage EKG
- Urgent RHC/LHC
Differential Diagnosis of HF

- Toxins
- Genetic
- Metabolic/Endocrine
- Infiltrative
- Stress CM
- Ischemic
- Pregnancy
- Hypertension
- HCM
- Non-Compaction
- ARVC
- Infectious
- Infiltrative
- Inflammatory
Differential Diagnosis

Acute Cardiomyopathy

- Ischemic
- Sepsis
- Inflammatory
- Metabolic
**LHC/RHC**

- No CAD on coronary angiography

  - RA 19 mmHg
  - RV 33/15 mmHg
  - PA 32/19/25 mmHg
  - PCWP 25 mmHg
  - LVEDP 23 mmHg
  - BP: 81/61/71 mmHg
  - PA saturation 47%
  - FA saturation 99%
  - Fick CO: 2.6 L/min
  - Fick CI: 1.5 L/min/m²
  - SVR: 1600 dyn·sec/cm⁵
  - PVR 0.8 WU
  - PCWP 25 mmHg
  - PVR 0.8 WU

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**Endomyocardial Biopsy Performed**

**IABP Placed**
Clinical Course

- One-hour post cath, developed ventricular arrhythmias and progressive hypotension

- Biventricular Dysfunction (CVP/PCWP = 0.8)

- Ongoing hemodynamic compromise despite IABP

- CentriMag BiVAD Temporary Assist Device
Intraoperative TEE
Pathology

GIANT CELL MYOCARDITIS
Giant Cell Myocarditis

- Estimated incidence 22 cases/100,000

- Possible increased incidence among those with existing autoimmune diseases

- Differential includes sarcoidosis, eosinophilic myocarditis, infectious myocarditis, viral myocarditis

- Prognosis varies depending on fulminant versus non-fulminant variation
Patients with giant cell myocarditis have extremely ventricular compromise, or progress to dilated cardiomyopathy and logic evidence of myocardial inflammation. Failure to use beta-blocking outcome included New York Heart Association Class III or IV, pulmonary ejection fraction of less than 40%. Additional predictors of poor presentation with syncope, bundle branch block on electrocardiography, factors do appear to predict death or transplantation, including pre-predict adverse outcomes in viral myocarditis. Although many of documention transplant-free survival of 93% in 11 years. Those with nont myocarditis) have a surprisingly good prognosis, with one series patients with severe hemodynamic collapse at presentation (fulminating myocarditis may have a more varied outlook. Paradoxically, however, patients with more advanced cardiac dysfunction accompany myocarditis will recover in the majority of cases without long-term sequelae. Patients with acute myocarditis and mild cardiac involvement generally have a poor prognosis, with median survival of less than 6 months, and most patients will require transplantation to avoid succumbing to the disease. Poor prognosis, with median survival of less than 6 months, and most patients will require transplantation to avoid succumbing to the disease.
Immunosuppression in GCM

- Initiation of immunosuppression regimen (calcineurin inhibitors +/- azathioprine, steroids) are the mainstays of therapy; median survival improves with immunosuppression

- No robust RCT to guide therapy length or specific regimen; observational studies have demonstrated relapse upon cessation of immunosuppression

- Surveillance biopsies following recovery can guide titration of therapies

LT Cooper. *Am J Cardiol* 2008;102:1535–1539
Clinical Course

- Received pulsed dosed IV methylprednisone at time of BiVAD implant + steroid taper, followed by IVIG dosing and of Tacrolimus

- Remained on BiVAD support for 2 weeks with clinical

- Presence of arterial line pulsatility and aortic valve opening on adequate BiVAD support suggested possible recovery of native LV function
Reverse Ramp Study

3L Flow

1L Flow
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<th>LVAD Speed (RPM)</th>
<th>RVAD Speed (RPM)</th>
<th>LVAD Flow (L/Min)</th>
<th>RVAD Flow (L/min)</th>
<th>RA (mmHg)</th>
<th>Fick CI (L/min/ m²)</th>
<th>LVEDD (cm)</th>
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Clinical Course

• Reverse RAMP study showed evidence of myocardial recovery

• Decanulated from BiVAD support, transitioned to Milrinone → weaned off

• Discharged ~ 3 weeks following initial presentation, plan ongoing immunosuppression (tacrolimus and prednisone) with surveillance

• Patient back to work and running 10K races!
Follow-Up Cardiac MRI
Summary

• Narrow pulse pressure, clinical volume overload, lactic acidosis may suggest critical cardiogenic shock irrespective of non-invasive/invasive diagnostics

• Consider endomyocardial biopsy in a unexplained, acute and progressive cardiomyopathy

• Giant cell myocarditis often progresses to a fulminant course—anticipate need for early biventricular support

• Myocardial recovery can be assessed with step-wise reverse RAMP protocols
Thank You

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