Team Imaging Interpretation

Bonnie Ky, MD, MSCE

Panelists: Drs. Ana Barac, JB Durand, Jennifer Liu, and Dinesh Thavendiranathan
Case 1

- 55 year old female with left sided invasive ductal carcinoma (3cm; 4/10 lymph nodes+), ER-/PR-/HER2+

- Adjuvant chemotherapy (ddAC – doxorubicin and cyclophosphamide) → THP (docetaxel, trastuzumab, pertuzumab) → radiation therapy

- No cardiovascular disease or risk factors or medications

- No family history of cardiovascular disease
Baseline Assessment of Cardiac Function

3D LVEF 61%; GLS -21.6%; GCS -23%; BP 118/82
Echocardiogram after Completion of Doxorubicin

3D LVEF 51%; GLS -17.9%; GCS -20%; BP 132/84
Case 1 - Question

She is scheduled to start THP in 2 weeks. What would you recommend to the patient and oncologist?

- A. LVEF and strain are normal; continue with THP
- B. LVEF and strain are abnormal; hold one cycle; delay THP start for 3 weeks
- C. LVEF and strain are borderline normal; Consider starting ACE-I and/or beta blocker; continue with THP
- D. LVEF and strain are borderline normal; recommend docetaxel/trastuzumab only and not pertuzumab
Case 1 - Answer

• She is scheduled to start THP in 2 weeks. What would you recommend to the patient and oncologist?
  – A. LVEF and strain are normal; continue with THP
  – B. LVEF and strain are abnormal; hold one cycle; delay THP start for 3 weeks
  – C. LVEF and strain are borderline normal; Consider starting ACE-I and/or beta blocker; continue with THP
  – D. LVEF and strain are borderline normal; recommend docetaxel/trastuzumab only and not pertuzumab
Echocardiogram after 4 Cycles of THP

3D LVEF 46%; GLS -15.0%; GCS -17%; BP 133/78
Case 1 - Question

• What would you do at this point?
  – A. Continue with THP
  – B. Hold one cycle; no need for cardiac meds; recheck echocardiogram before restarting
  – C. Hold one cycle of THP; start or uptitrate ACE-I and/or beta blocker; check echocardiogram at 4 to 6 weeks
  – D. Hold one cycle and start diuretic; recheck echocardiogram before restarting
Case 1 - Answer

• What would you do at this point?
  – A. Continue with THP
  – B. Hold one cycle; no need for cardiac meds; recheck echocardiogram before restarting
  – C. Hold one cycle of THP; start or uptitrate ACE-I and/or beta blocker; check echocardiogram at 4 to 6 weeks
  – D. Hold one cycle and start diuretic; recheck echocardiogram before restarting
Case 2

• 78 year old female with metastatic bladder cancer, underwent 1 cycle of pembrolizumab

• Presents with signs and symptoms of heart failure
Case 2

E/A = 2.2  E’ = 4 cm/sec  GLS = -8.2%
Case 2 - Question

- How would you best describe the echocardiogram findings?
  - A. Severe LV dysfunction, LVEF 30%, with Grade III diastolic function
  - B. Moderate LV dysfunction, LVEF 40%, with Grade II diastolic function
  - C. Findings concerning for myocarditis
  - D. Findings concerning for amyloid cardiomyopathy
  - E. A and D
Case 2 - Answer

• How would you best describe the echocardiogram findings?
  – A. Severe LV dysfunction, LVEF 30%, with Grade III diastolic function
  – B. Moderate LV dysfunction, LVEF 40%, with Grade II diastolic function
  – C. Findings concerning for myocarditis
  – D. Findings concerning for amyloid cardiomyopathy
  – E. A and D
Case 2
Case 2 – Amyloidosis Workup

- $^{99m}$Tc-PYP Scan $\rightarrow$ Grade 3 myocardial uptake

- AL amyloidosis labwork $\rightarrow$ normal serum immunofixation and free light chain ratio
<table>
<thead>
<tr>
<th>TYPE OF AMYLOIDOSIS</th>
<th>PRECURSOR PROTEIN</th>
<th>USUAL AGE AT ONSET</th>
<th>MAIN ORGANS INVOLVED</th>
<th>AVERAGE SURVIVAL TIME IN UNTREATED PATIENTS</th>
<th>SPECIFIC TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>AL (primary)</td>
<td>Abnormal light chains</td>
<td>50+</td>
<td>All except central nervous system; heart involved in 50% of cases</td>
<td>Noncardiac disease, 24 months; disease with HF &lt; 9 months</td>
<td>Chemotherapy aimed at plasma cells</td>
</tr>
<tr>
<td>Familial (ATTR)</td>
<td>Mutant TTR</td>
<td>20-70+ (dependent on mutation)</td>
<td>Peripheral and autonomic neuropathy; heart</td>
<td>7 to 10 years for neuropathy</td>
<td>Liver transplantation; Agents to stabilize TTR (tafamidis) or suppress its production</td>
</tr>
<tr>
<td>Senile systemic (SSA)</td>
<td>Wild-type TTR</td>
<td>70+</td>
<td>Heart</td>
<td>5 to 7 years</td>
<td>Agents to stabilize TTR (tafamidis) or suppress its production</td>
</tr>
<tr>
<td>Isolated atrial (IAA)</td>
<td>Atrial natriuretic peptide</td>
<td>Unknown</td>
<td>Cardiac atria (particularly in already diseased hearts)</td>
<td>No effect on survival</td>
<td>None needed</td>
</tr>
<tr>
<td>AA (secondary)</td>
<td>Serum amyloid A (SAA), inflammatory protein</td>
<td>Teens upward</td>
<td>Liver, kidney; heart rarely</td>
<td>10+ years</td>
<td>Treatment of underlying inflammatory condition</td>
</tr>
</tbody>
</table>

Potential Diagnostic Algorithm for ATTR Amyloidosis

Heart failure, syncope, or bradycardia, with echocardiogram and/or cardiac magnetic resonance imaging (CMR) suggesting/indicating cardiac amyloid

Bone scintigraphy with $^{99m}$Tc-DPD/HMDP/PYP

- Grade 0
- Grade 1
- Grade 2 to 3

Serum immunofixation + Urine immunofixation + serum free light chain assay (Freelite)
- Monoclonal protein present? Yes/No

Cardiac AL/ATTR amyloidosis unlikely

Review/request CMR

Need specialized assessment for Diagnosis: Histological confirmation and typing of amyloid

Cardiac amyloidosis

Variant ATTR amyloidosis

Wild-Type ATTR amyloidosis

Cardiac ATTR amyloidosis

TTR genotyping

Cardiac Structural and Functional Consequences of Amyloid Deposition by Cardiac Magnetic Resonance and Echocardiography and Their Prognostic Roles

- 322 subjects (133 AL, 189 ATTR) → CMR and echocardiography
- Amyloid burden defined by CMR-extracellular volume
- TAPSE and indexed Stroke Volume associated with mortality

Case 3

• 66 year old male with metastatic melanoma s/p 2 cycles of ipilimumab and nivolumab with worsening dyspnea. A chest CT reveals bilateral pulmonary emboli

• He is started on rivaroxaban, but after 1 month, has progressive dyspnea

• Chest CT demonstrates progressive clot burden
Case 3
Case 3 - Question

• What is the potential reason for lack of response to anticoagulation?
  – A. Echocardiogram and MRI suggest tumor/thrombus; anticoagulation alone will not resolve mass
  – B. The echocardiogram and MRI suggest thrombus; different anticoagulation regimen is needed
  – C. The echocardiogram and MRI suggest thrombus; longer treatment duration is needed
  – D. Cannot determine reason; more information needed
Case 3 - Answer

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  – C. The echocardiogram and MRI suggest thrombus; longer treatment duration is needed
  – D. Cannot determine reason; more information needed
### Characteristic CMR Findings - Cardiac Masses

<table>
<thead>
<tr>
<th>Mass</th>
<th>T1-weighted*</th>
<th>T2-weighted*</th>
<th>LGE</th>
<th>Perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyst</td>
<td>Low</td>
<td>High</td>
<td>No uptake</td>
<td>No</td>
</tr>
<tr>
<td>Lipoma</td>
<td>High</td>
<td>High</td>
<td>No uptake</td>
<td>No</td>
</tr>
<tr>
<td>Metastasis</td>
<td>Low</td>
<td>High</td>
<td>Heterogeneous</td>
<td>Yes</td>
</tr>
<tr>
<td>Thrombus</td>
<td>Low (high if acute)</td>
<td>Low (high if acute)</td>
<td>No uptake (dark)</td>
<td>No (dark)</td>
</tr>
</tbody>
</table>

*relative to myocardium; LGE = late gadolinium enhancement

Metastatic Disease to the Heart

- Occurs in 9.1 to 14.2% in patients with metastatic disease
- Most common: lung, breast, hematologic tumors, melanoma, renal cell carcinoma
- Rare: prostate cancer
- Mechanisms: direct extension, hematogenous, lymphatic or venous spread
- Features vary widely according to lesion etiology
# Primary Cardiac Lesions

<table>
<thead>
<tr>
<th>Benign neoplasms</th>
<th>Age</th>
<th>Location</th>
<th>Genetic Drivers</th>
<th>Malignant neoplasms</th>
<th>Age</th>
<th>Location</th>
<th>Genetic Drivers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Papillary fibroelastoma</td>
<td>Adult</td>
<td>Valves</td>
<td>KRAS</td>
<td>Undifferentiated pleomorphic sarcoma</td>
<td>Adult</td>
<td>Atria</td>
<td>MDM2</td>
</tr>
<tr>
<td>Myxoma</td>
<td>Adult</td>
<td>Atria</td>
<td>PRKAR1A</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rhabdomyoma</td>
<td>Pediatric</td>
<td>Ventricles</td>
<td>TSC1, TSC2</td>
<td>Angiosarcoma</td>
<td>Adult</td>
<td>Atria</td>
<td>Complex cytogenetics</td>
</tr>
<tr>
<td>Fibroma</td>
<td>Pediatric</td>
<td>Ventricles</td>
<td>PTCH1</td>
<td>Mesothelioma</td>
<td>Adult</td>
<td>Pericardium</td>
<td></td>
</tr>
<tr>
<td>Lipomatous hypertrophy of atrial septum</td>
<td>Adult</td>
<td>Atria</td>
<td>HMGA2</td>
<td>Lymphoma</td>
<td>Adult</td>
<td>Pericardium</td>
<td></td>
</tr>
<tr>
<td>Lipoma</td>
<td>Adult</td>
<td>Pericardium</td>
<td>HMGA2, TSC1, TSC2</td>
<td>Synovial sarcoma</td>
<td>Adult</td>
<td>Pericardium</td>
<td>SS18-SSX</td>
</tr>
<tr>
<td>Hemangioma</td>
<td>Adult</td>
<td>Ventricles</td>
<td></td>
<td>Rhabdomyosarcoma</td>
<td>Pediatric</td>
<td>Ventricles</td>
<td></td>
</tr>
<tr>
<td>Germ cell tumor</td>
<td>Pediatric</td>
<td>Pericardium</td>
<td></td>
<td>Liposarcoma</td>
<td>Adult</td>
<td>Ventricles</td>
<td></td>
</tr>
<tr>
<td>Histiocytoid cardiomyopathy</td>
<td>Pediatric</td>
<td>Ventricles</td>
<td>MT-CYB</td>
<td>Leiomyosarcoma</td>
<td>Adult</td>
<td>Vasculature</td>
<td>TPS3*</td>
</tr>
<tr>
<td>Inflammatory myofibroblastic tumor</td>
<td>Pediatric</td>
<td>Ventricles, valves</td>
<td>RET, VHL, SDH</td>
<td>Osteosarcoma</td>
<td>Adult</td>
<td>Atria</td>
<td>TPS3*</td>
</tr>
<tr>
<td>Paraganglioma</td>
<td>Adult</td>
<td>Atria</td>
<td></td>
<td>Myxofibrosarcoma</td>
<td>Adult</td>
<td>Atria</td>
<td>TPS3*</td>
</tr>
<tr>
<td>Granular cell tumor</td>
<td>Adult</td>
<td>Ventricles</td>
<td></td>
<td>Solitary fibrous tumor</td>
<td>Adult</td>
<td>Pericardium</td>
<td>STAT6</td>
</tr>
<tr>
<td>Epithelioid hemangioendothelioma</td>
<td>Adult</td>
<td>Ventricles</td>
<td>WWTRI-CAMTA1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamartoma of mature cardiac myocytes</td>
<td>Adult</td>
<td>Ventricles</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schwannoma</td>
<td>Adult</td>
<td>Atria</td>
<td></td>
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</tbody>
</table>

Case 4

- 52 year old male with BRAF V600E+ metastatic melanoma with lung and axillary node involvement
- No cardiovascular history or risk factors or medications
- Started on nivolumab; 1 week later, develops cellulitis; three weeks later, worsening fatigue, fever and dyspnea
- CT scan $\rightarrow$ moderate pericardial effusion; echocardiogram obtained
LA volume 36 ml/m²
TR velocity 3.3 m/sec
E/e’ 15
E’ lateral 5
E’ septal 7
GLS -23.8%
Case 4 - Question

What would your next steps be and why?

- A. Continue to follow patient, no additional diagnostic testing necessary at this point
- B. Prescribe oral diuretics, echocardiogram suggests elevated filling pressures with a preserved LVEF
- C. Order a cardiac MRI and check troponin levels, pericardial effusion could be a sign of myocarditis
Case 4 - Answer

• What would your next steps be and why?
  – A. Continue to follow patient, no additional diagnostic testing necessary at this point
  – B. Prescribe oral diuretics, echocardiogram suggests elevated filling pressures with a preserved LVEF
  – C. Order a cardiac MRI and check troponin levels, pericardial effusion could be a sign of myocarditis
At time of MRI:
Troponin 5 ng/L (ULN 14 ng/L)
Case 4 - Question

• What is the most likely diagnosis?
  – A. Myocarditis
  – B. Myocardial ischemia/coronary artery disease
  – C. Heart failure with preserved ejection fraction
  – D. Takotsubo cardiomyopathy
Case 4 - Answer

• What is the most likely diagnosis?
  – A. Myocarditis
  – B. Myocardial ischemia/coronary artery disease
  – C. Heart failure with preserved ejection fraction
  – D. Takotsubo cardiomyopathy
Immunotherapy Case Series: WHO

- VigiBase Database (WHO) Review: 101 cases of severe myocarditis with immune checkpoint inhibitors
- Median age: 69 years (range 20-90)
- Therapy: 57% anti-PD-1 monotherapy; 27% combination therapy (anti-PD-1/PD-L1 & anti-CTLA-4)
- Combination therapy with worse outcomes
- Timing: median onset 27 days (range 5-155); 76% in first week [only available for 1/3 of total cases]
- Concurrent AEs: Occurred in 42%; Death in 46%

Immunotherapy Case Series: FDA

• FDA Database review: 250 cases of myocarditis with immune checkpoint inhibitors between 2012-2017
• Increase in reports over time
• AEs: Pericardial effusion, pericarditis, and tamponade also observed; Death in 50%; mortality did not differ by clinical characteristics

Immunotherapy Cases Series: Multicenter Registry

- 35 cases from 8-center registry (2013-2017); compared to 964 controls without clinical myocarditis
- Single center incidence: 1.1% myocarditis; 0.52% MACE
- Median time to onset from first dose: 34 days (IQR 21-75); 81% within 3 months
- Diagnoses: 11 patients → cardiac biopsy/autopsy; 31 → CMR
- Treatment: steroids in 89% (IVIg, MMF, ATG and infliximab in < 4 cases)

Presentation of Immunotherapy Myocarditis

Potential Diagnostic Algorithm for Myocarditis

1. **Patient on immune checkpoint inhibitors (ICI) or prior ICI use**
   - Patient presenting with new cardiovascular (CV) symptoms
     - Electrocardiogram (ECG) and troponin test
       - Normal results
         - New ventricular arrhythmia or conduction system disease?
           - No (N)
             - Outpatient echo and NT-proBNP testing
           - Yes (Y)
             - Elevated troponin/abnormal EKG
               - If indeterminate troponin, retest to eliminate false result
               - Possible myocarditis: Admit patient
                 Stop ICI therapy; Urgent Cardiology/Cardio-Oncology consult;
                 Determine whether patient is stable or unstable to dictate treatment
       - Elevated results
### Typical Findings and CMR Criteria for Myocarditis

**MAIN**

<table>
<thead>
<tr>
<th>Diagnostic Targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial edema</td>
</tr>
</tbody>
</table>

**T2-based imaging**

- Regional high T2 SI
- Global T2 SI ratio ≥2.0 in T2W CMR images
- Regional or global increase of myocardial T2 relaxation time

**T1-based imaging**

- Regional or global increase of native myocardial T1 relaxation time or ECV
- Areas with high SI in a nonischemic distribution pattern in LGE images

<table>
<thead>
<tr>
<th>Diagnostic Targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 - edema (intra or extra-cellular), hyperemia/capillary leak, necrosis, fibrosis</td>
</tr>
<tr>
<td>EGE - hyperemia, capillary leak</td>
</tr>
<tr>
<td>LGE - necrosis, fibrosis, (extracellular acute edema)</td>
</tr>
<tr>
<td>ECV - edema (extracellular), hyperemia/capillary leak, necrosis, fibrosis</td>
</tr>
</tbody>
</table>

**SUPPORTIVE**

<table>
<thead>
<tr>
<th>Diagnostic Targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV dysfunction</td>
</tr>
</tbody>
</table>

- Pericardial effusion in cine CMR images
- High signal intensity of the pericardium in LGE images, T1-mapping or T2-mapping
- T1 mapping or T2 mapping
- Systolic LV wall motion abnormality in cine CMR images

**Diagnostic Targets**

- Pericardial inflammation

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Case 5

- 66 year old male with hypertension and dyslipidemia presents with worsening lower extremity edema, dyspnea on exertion, fatigue and anorexia

- Murmur on exam, echocardiogram obtained
Case 5 - Question

• What is the most likely diagnosis?
  – A. Moderate tricuspid regurgitation secondary to RV annular dilation
  – B. Severe tricuspid regurgitation secondary to carcinoid disease
  – C. Moderate tricuspid regurgitation secondary to marantic endocarditis
Case 5 - Answer

• What is the most likely diagnosis?
  – A. Moderate tricuspid regurgitation secondary to RV annular dilation
  – B. Severe tricuspid regurgitation secondary to carcinoid disease
  – C. Moderate tricuspid regurgitation secondary to marantic endocarditis
Carcinoid

• Low grade neuroendocrine tumors that arise in the GI tract, lung, or gonads
• Carcinoid syndrome (flushing, diarrhea, cramping, wheezing) from vasoactive substances released by tumor (serotonin)
• 50% of carcinoid patients with cardiac involvement
• CV complications primarily associated with right sided structures; 10 to 15% have left sided disease
Carcinoid

- Imaging reveals thickened, restricted leaflets and papillary muscles and chords
- Fibrotic plaques observed on valves and subvalvular apparatus
- Treatment includes chemotherapy, tumor resection, somatostatin analogs
Case 6

• 71 year old female with invasive thymic squamous cell carcinoma, involving pericardium and diaphragm

• Treated with carboplatin, paclitaxel and then proton RT

• Suffered from disease progression and started on pembrolizumab

• Notes fatigue; labs after the 3rd infusion of pembrolizumab
  – Tn 0.052 (range <0.015 ng/ml)
Case 6

- Treated with steroids until troponin normalizes
- Disease progression, and pembrolizumab stopped
- Sunitinib started, and patient reports worsening fatigue
Case 6
Case 6 - Question

• What does the CMR demonstrate?
  – A. Moderately decreased LVEF with normal RV function
  – B. Nonspecific enhancement of the inferior and inferolateral walls
  – C. Both A and B
  – D. None of the above
Case 6 - Answer

- What does the CMR demonstrate?
  - A. Moderately decreased LVEF with normal RV function
  - B. Nonspecific enhancement of the inferior and inferolateral walls
  - C. Both A and B
  - D. None of the above
Case 7

- 55 year old female with chronic dyspnea on exertion has newly diagnosed right sided breast cancer
  - Stage IIIA (T3N2M0); Tumor 6.1cm

- Oncologist recommends neoadjuvant chemotherapy with dose dense doxorubicin (240mg/m^2) with cyclophosphamide, followed by paclitaxel

- Pre-treatment echocardiogram is obtained
Case 7

LVIDd 6.3cm  LVIDs 4.6cm  (BSA 1.8m²)
AV peak gradient: 27 mmHg
Mean gradient: 13 mmHg
AV VTI: 54 cm
LVOT VTI: 30 cm
AR PHT: 205 msec
AR slope: 6.6 m/sec²
Case 7 - Question

• What are the major echocardiographic findings?
  – A. Heavy trabeculation concerning for noncompaction cardiomyopathy
  – B. Moderate to severe aortic regurgitation secondary to bicuspid aortic valve, dilated LV with LVEF of 50 to 55%
  – C. Normal LV size, increased LV wall thickness, LVEF 55%
  – D. Moderate aortic regurgitation, moderate aortic stenosis, calcified and trileaflet aortic valve, LVEF 50 to 55%
Case 7 - Answer

• What are the major echocardiographic findings?
  – A. Heavy trabeculation concerning for noncompaction cardiomyopathy
  – B. Moderate to severe aortic regurgitation secondary to bicuspid aortic valve, dilated LV with LVEF of 50 to 55%
  – C. Normal LV size, increased LV wall thickness, LVEF 55%
  – D. Moderate aortic regurgitation, moderate aortic stenosis, calcified and trileaflet aortic valve, LVEF 50 to 55%
Bicuspid Aortic Valve

- Affects 0.5-2% of adults
- Associated conditions: aortic regurgitation, aortic stenosis, aortic dilation, coarctation
- Aortic regurgitation common, in 47-64% at mean age of 45 years
- Aortic stenosis in 12-37% (moderate/severe) at mean age of 54 years
- Mixed valvular disease associated with worse prognosis

Bicuspid Aortic Valve – Sievers Classification

<table>
<thead>
<tr>
<th>main category: number of raphes</th>
<th>0 raphe - Type 0</th>
<th>1 raphe - Type 1</th>
<th>2 raphes - Type 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>21 (7)</td>
<td>269 (88)</td>
<td>14 (5)</td>
</tr>
</tbody>
</table>

| 1. subcategory: spatial position of cusps in Type 0 and raphes in Types 1 and 2 |
|-------------------------------|-----------------|-----------------|------------------|
| lat                           | 13 (4)          | 216 (71)        | 14 (5)           |
| ap                            | 7 (2)           | 45 (15)         |                  |
| L - R                         |                 | 8 (3)           |                  |
| N - L                         |                 |                 |                  |
| L - R / R - N                 |                 |                 |                  |

<table>
<thead>
<tr>
<th>2. subcategory: Valvular Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
</tr>
<tr>
<td>S</td>
</tr>
<tr>
<td>(I + S)</td>
</tr>
<tr>
<td>No</td>
</tr>
</tbody>
</table>

Case 8

• 55 year old male with relapsed AML s/p stem cell transplant, hypertension, dyslipidemia presents with complaints of sharp, pleuritic chest pain

• EKG and echocardiogram obtained
Case 8
Case 8 – Question

What next step is likely to be of greatest diagnostic or therapeutic yield?

- A. Stress test
- B. Transesophageal echocardiogram
- C. Cardiac MRI
- D. Start anticoagulation and repeat echocardiogram in 5 days
Case 8 – Answer

- What next step is likely to be of greatest diagnostic or therapeutic yield?
  - A. Stress test
  - B. Transesophageal echocardiogram
  - C. Cardiac MRI
  - D. Start anticoagulation and repeat echocardiogram in 5 days
Case 8
Case 8 – Question

• What does the cardiac MRI show?
  – A. Transmural enhancement of the anteroseptum, distal inferior wall, and apex with associated LV thrombus
  – B. RV apical thickening with peripheral hyperenhancement, central hypoenhancement, and RV thrombus
  – C. RV infarct and thrombus; LV infarct and thrombus
  – D. Both A and B
Case 8 – Answer

• What does the cardiac MRI show?
  – A. Transmural enhancement of the anteroseptum, distal inferior wall, and apex with associated LV thrombus
  – B. RV apical thickening with peripheral hyperenhancement, central hypoenhancement, and RV thrombus
  – C. RV infarct and thrombus; LV infarct and thrombus
  – D. Both A and B
Hypereosinophilic Syndromes

• Hypereosinophilia rare; organ damage secondary to eosinophilic overproduction and infiltration
  – Primary (neoplastic – stem cell, myeloid or eosinophilic disorders > 1500 cells/ml)
  – Secondary (reactive, allergy, parasitic infections)
  – Idiopathic
  – Organs: skin, lung, GI, heart, and brain
  – CV: restrictive cardiomyopathy, extensive and obliterative cavity thrombus, myocardial inflammation and necrosis
  – Three phases: acute, intermediate, fibrotic

THANK YOU!