Echocardiography in Systemic Diseases

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DISCLOSURE

Relevant Financial Relationship(s)
None

Off Label Usage
None
Echo in Systemic Diseases

- Systemic diseases with secondary cardiac involvement are uncommon

But

- Echo can identify unique, characteristic features and echo may be the first clue to the underlying systemic illness
Cardiac Involvement in Systemic Diseases

- Autoimmune
- Endocrine
- Collagen Vascular Diseases
- Malignancy
- Amyloid/Infiltrative Diseases
- Radiation Induced Heart Disease
- Drug Induced Valvulopathy
Case

- 27 y/o female who presents with dyspnea, chest pain, and fatigue
  - NYHA class III
- Abnormal nuclear perfusion stress test led to coronary arteriography
  - Normal coronaries but LV gram suggestive of “Hypertrophic CM” (EF 75%)
- Elevated Sedimentation Rate
- Referred to Mayo Clinic → Echo performed
Apical 4 Chamber View
Diastolic Function

- MV Dec. Time = 105 msec
- MV Emax = 1.1 m/sec
- e’ = 0.04 m/sec
- E/e’ 28
What is the Diagnosis?

1. Hypertrophic Cardiomyopathy (Apical Variant)
2. Amyloidosis
3. Eosinophilic Endomyocardial Disease
4. LV Noncompaction
5. LV Myxoma
RV Biopsy (H&E Stain)
Hypereosinophilic Syndrome
Cardiac Manifestations

- Persistent increase in eosinophil count
  eosinophil count > 1500 cells/mm3
- CHF (dyspnea)
  - Restrictive Cardiomyopathy
  - Mitral regurgitation
- Systemic embolization
Eosinophilic Heart Disease

4 Stages:

1) Acute inflammatory myocarditis
2) Eosinophil rich thrombus deposition
   - Mediated by injured endothelium
3) Endocardial thickening
   - Valve involvement
4) Fibrosis

Hypereosinophilic Syndrome (HES)
Cardiac Involvement: 40-60% of patients

2-D Echo & Doppler Findings

- LV > RV inflow apical thrombo-obliteration, endocardial thickening
- Restrictive diastolic dysfunction
- Subvalvular thrombosis, leaflet entrapment MV > TV Leaflets; MR&TR

Ommen, Am J Cardiol 2000
Natural History Hypereosinophilic Syndrome

Myocarditis → Thrombus → Fibrosis

Image courtesy of Leslie Elvert RDCS
Basal LV Fibrosis with Mitral Posterior Leaflet Tethering

- Courtesy of Dr. Natesa Pandian
Eosinophilic Heart Disease
Contrast Helpful
Hypereosinophilic Syndrome

Treatment
• Medical therapy
  – Corticosteroids
  – Hydroxyurea
  – Interferon
  – CHF Meds
• Surgical Therapy
  – Palliative

Echo Differential Diagnosis
• Apical hypertrophic CM
• LV Noncompaction
• LV tumor
  – Myxoma
  – Papillary fibroelastoma
• Ischemic LV dysfunction with apical thrombus
Our Case:
TTE after 2 months of anticoagulation and 1 month of prednisone therapy
Patient with CREST Syndrome: Dyspnea and Edema

RVSP: 75 mmHg
Scleroderma and Pulmonary HTN

- PH present in 8-12% of scleroderma patients
  - Higher risk in CREST patients
- Accounts for 30% of deaths
- Screening for PH recommended
- RV dysfunction, cardiac index and pericardial effusion are markers of poor prognosis in PH
### Pericardial Involvement in Systemic Disease

<table>
<thead>
<tr>
<th>SID</th>
<th>Estimated overall prevalence* (%)</th>
<th>Estimated frequency of pericardial involvement (%)</th>
<th>Type of pericardial involvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasculitis</td>
<td>&lt;10%</td>
<td>Rare (case reports)</td>
<td>Pericardial effusion, pericarditis</td>
</tr>
<tr>
<td>Takayasu arteritis</td>
<td>Rare</td>
<td>Rare (case reports)</td>
<td>Pericardial effusion, pericarditis</td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td>Rare</td>
<td>Rare (case reports)</td>
<td>Pericardial effusion, pericarditis</td>
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<tr>
<td>Polyarteritis nodosa</td>
<td>Rare</td>
<td>Rare (case reports, series)</td>
<td>Pericarditis</td>
</tr>
<tr>
<td>Kawasaki disease</td>
<td>&lt;5%</td>
<td>30%</td>
<td>Pericardial effusion, pericarditis</td>
</tr>
<tr>
<td>Churg–Strauss syndrome</td>
<td>&lt;5%</td>
<td>20–25%</td>
<td>Pericardial effusion, pericarditis</td>
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<tr>
<td>Wegener granulomatosis</td>
<td>&lt;5%</td>
<td>&lt;10%</td>
<td>Pericardial effusion, pericarditis</td>
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<tr>
<td>Connective tissue diseases</td>
<td>80–90%</td>
<td></td>
<td></td>
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<tr>
<td>Systemic lupus erythematosus</td>
<td>50–60%</td>
<td>&gt;50%</td>
<td>Pericardial effusion, pericarditis</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>20–30%</td>
<td>10–30%</td>
<td>Pericardial effusion (30%), pericarditis (10%)</td>
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<tr>
<td>Systemic sclerosis</td>
<td>5–10%</td>
<td>Symptomatic &lt;20%, overall &gt;60%</td>
<td>Pericardial effusion, pericarditis</td>
</tr>
<tr>
<td>Polymyositis and dermatomyositis</td>
<td>&lt;5%</td>
<td>&lt;10%</td>
<td>Pericarditis, pericardial effusion, cardiac tamponade (case reports)</td>
</tr>
<tr>
<td>Mixed connective tissue disease</td>
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<td></td>
<td>Pericarditis</td>
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<tr>
<td>Sjögren syndrome</td>
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<tr>
<td>Behçet’s disease</td>
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<tr>
<td>Granulomatous diseases</td>
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<td>Pericarditis</td>
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<tr>
<td>Sarcoidosis</td>
<td></td>
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<td>Pericarditis</td>
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<tr>
<td>Autoinflammatory diseases</td>
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<td>Pericarditis</td>
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<tr>
<td>Familial Mediterranean fever</td>
<td></td>
<td></td>
<td>Pericarditis</td>
</tr>
<tr>
<td>TNF receptor-1 associated periodic syndrome (TRAPS)</td>
<td></td>
<td></td>
<td>Pericarditis</td>
</tr>
</tbody>
</table>
33 Year Old Female → Multiple Strokes

- ANA positive and Antiphospholipid antibodies present
- Libman-Sacks endocarditis
Systemic Lupus Erythematosus
Cardiac Involvement

- Pericarditis (fluid ANA+)
  - 50-60% of cases
- Lupus anticoagulant
- Anticardiolipin or Antiphospholipid Abs
- Myocarditis
- Coronary arteritis
- Libman-Sacks (Marantic) vegetations
18 y.o. female with occipital stroke

- Lupus anticoagulant + antiphospholipid antibodies present
- Libman-Sacks endocarditis
Not only the mitral valve!
Antiphospholipid Syndrome

Diagnosis confirmed at surgery

- IgG and IgM Antiphospholipid antibody

- Importance of recognition
  - Unlikely repair
  - Choice of prosthesis
    - Avoid bioprosthesis if possible
  - Anticoagulation
Systemic Lupus Erythematosus Cardiac Involvement

- Pericarditis (fluid ANA+)
- Lupus anticoagulant
- Anticardiolipin antibodies
- Myocarditis
- Coronary arteritis
- Libman-Sacks (Marantic) vegetations

Courtesy of W Edwards MD
A 68-year-old man presents with fatigue and abdominal bloating. On cardiac exam, the jugular venous pressure revealed “CV” waves to angle of the jaw. An RV lift is present. There is a grade 2/6 pansystolic murmur at the lower sternal border that gets louder with inspiration. There is a soft systolic ejection murmur and diastolic murmur at the second left interspace. In addition, there is an enlarged and pulsatile liver. Images obtained from his TTE are shown.

Which of the following is the most likely diagnosis?

A. Rheumatic heart disease
B. Carcinoid heart disease
C. Ebstein’s anomaly
D. Endocarditis
39 year old male with diarrhea, flushing and weight loss
Carcinoid Syndrome
Carcinoid: Echo Features

Tricuspid valve
- Thickened leaflets
- Retracted leaflets
- Fixed semi-open position

Pulmonary valve
- Thickened cusps
- Retracted and rigid
Severe (Torrential) Tricuspid Regurgitation

Systolic RV → RA pressure equalization

TR  CW Doppler

RV  RA

Courtesy of Dr. WK Freeman
Pulmonary Valve Involvement
Pulmonary Valve Involvement

Adapted from Mayo Image Data Base, William Edwards, MD
Carcinoid Tumors

- Arise from the GI tract
- Slowly growing
- Produce vasoactive substances
  - bradykinin
  - histamine
  - serotonin
  - prostaglandins
  - catecholamines
  - 5-HIAA
Carcinoid Heart Disease

- Carcinoid tumors: 1-2/100,000
- Carcinoid syndrome in 20-30%
- Deposition of a matrix-like material on the valves and endocardium of the right side of the heart
- Treatment of tumor does not cause regression of valve disease

Connolly HM. Curr Cardiol Rep. 2006
Carcinoid Heart Disease

Echo findings:

• Thickening and retraction of immobile tricuspid valve leaflets
• Severe tricuspid valve regurgitation
• May have similar findings in pulmonic valve
• Only 10-15% of cases involve left-sided valves
  – intra-cardiac shunt, primary bronchial carcinoid, primary gonadal carcinoid

Connolly HM. Curr Cardiol Rep. 2006
Carcinoid Syndrome: 3D TTE

Courtesy of Denisa Muraru, MD, PhD
Padua, Italy

Eur Heart J Cardiovasc Imaging 2012
Carcinoid Heart Disease

Over 50% of patients with Carcinoid Syndrome develop cardiac involvement

Flush

Wheezing

Diarrhea

Vasoactive Substances 5-HIAA
Outcome of Cardiac Surgery for Carcinoid Heart Disease

HEIDI M. CONNOLLY, MD, FACC, RICK A. NISHIMURA, MD, FACC, HUGH C. SMITH, MD, FACC, PATRICIA A. PELLIKKA, MD, FACC, CHARLES J. MULLANY, MD, LARRY K. KVOLE, MD

Rochester, Minnesota

Objectives. The hypothesis was that cardiac surgery for symptomatic carcinoid heart disease in conjunction with adjunctive therapy could improve the long-term outlook of patients with carcinoid heart disease.

Background. Patients with carcinoid heart disease have a dismal prognosis; most die of progressive right heart failure within 1 year after onset of symptoms. Improved therapies for the systemic manifestations of the carcinoid syndrome have resulted in symptomatic improvement and prolonged survival in patients without heart disease.

Methods. Twenty-six patients with symptomatic carcinoid heart disease underwent valvular surgery. Preoperative clinical, laboratory, Doppler echocardiographic and hemodynamic factors were evaluated. The survival of the surgical group was compared with that of a control group of 40 medically treated patients.

Results. There were nine perioperative deaths (35%), primarily from postoperative bleeding and right ventricular failure. Of the 17 surgical survivors, 8 were alive at a mean of 28 months of follow-up. The postoperative functional class of the eight surviving patients was substantially improved. Late deaths were primarily due to hepatic dysfunction caused by metastatic disease. The only predictor of operative mortality (p = 0.03) was low voltage on preoperative electrocardiography (limb lead voltage ≤ 5 mm). Predictors of late survival included a lower preoperative somatostatin requirement and a lower preoperative urinary 5-hydroxyindoleacetic acid level. There was a trend toward increased survival for the surgical group compared with the control group.

Conclusions. Because new therapies have improved survival in patients with the malignant carcinoid syndrome, cardiac involvement has become a major cause of morbidity and mortality. Valve surgery is the only definitive treatment. Although cardiac surgery carries a high perioperative mortality, marked symptomatic improvement occurs in survivors. Surgical intervention should therefore be considered when cardiac symptoms become severe.

(J Am Coll Cardiol 1995;25:410–6)
TEE
(4 chamber View)
Carcinoid Heart Disease
Carcinoid Tumor: Liver Metastases
58 yo man with pulmonary infiltrates and syncope
Cardiac Sarcoidosis

- Noncaseating granuloma
- Regional wall motion abnormalities in unusual distribution
- Heart block
- Sudden death

Courtesy William Edwards, MD
Sarcoidosis – Granulomas

- 11% right atrium
- 73% interventricular septum
- 46% right ventricular wall
- 7% left atrium
- 96% left ventricular free wall

Bargout R: Int J Cardio, 2004
Sarcoidosis – Echo features

Echo abnormalities are rare

- LV aneurysms
- LV dilatation
- LV scar RWMAs
- Mitral valve regurgitation
- Pulmonary hypertension

Cardiac Sarcoidosis (Echo)

58 yo woman with weight loss, tremor and HR of 125
Hyperthyroidism

- Atrial fibrillation
  - ↑ risk of systemic embolism
  - cardioversion after euthyroid
- Decreased Peripheral resistance
  - hypotension
- Exacerbation of underlying CAD
  - increased myocardial O2 demand
- Tachycardia induced cardiomyopathy
Tachycardia Mediated Cardiomyopathy

• 25% of patients w/ LV dysfunction & AF will have improved EF with rate control
• Usually *unaware* of rhythm
• Resting heart rate - poor indicator of overall rate control
• Consider in all pts with AF & LV dysfunction

2 Years after Cardioversion and Treatment of Hyperthyroidism
Hypothyroidism:
Large Pericardial Effusion
43 year old man
43 year old man with amyloidosis
What is the most likely Diagnosis?

- 19 year old male with an abnormal gait, cerebellar dysarthria, areflexia

1. HIV myocarditis
2. Friedrich’s Ataxia
3. Hypertrophic obstructive CM
4. Arrhythmogenic right ventricular cardiomyopathy
5. Cardiac amyloidosis
Friedrich’s Ataxia

• Rare AR neurodegenerative disorder
  – 1:50,000
• Ataxia, cerebellar dysarthria, areflexia
• Onset < 20 years, relentless course
• Echo features
  – Symmetrical hypertrophied LV
  – Prominent papillary muscles
  – Absence of SAM

Durr A: NEJM 1996
Mimickers of Amyloid

- Friedrich’s Ataxia
- Primary Hyperoxaluria
- Fabry’s Disease
- Hypertrophic cardiomyopathy
- Hydroxychloroquine-induced Cardiotoxicity
- Renal Failure
Primary Hyperoxaluria

- Rare metabolic disorder with autosomal recessive inheritance
- PHO type 1 (0.11 - 0.26 per 100,000 live births)
- Enzymatic defect resulting in enhanced conversion of glyoxalate to poorly soluble oxalate which is excreted in the urine
Fabry’s Disease

- Inherited X-linked recessive
- Lysosomal storage disease
- α-galactosidase A (α-Gal A) enzyme deficiency
- Intralysosomal accumulation of the glycosphingolipid globotriaosylceramide (GL-3)
- “Binary” appearance of walls on echo
Echocardiography showed in 83% of FC patients (95% of FC patients with LVH) a binary appearance of endocardial border absent in all HCM, hypertensive, and healthy subjects. The sensitivity and specificity of this echocardiographic feature in detecting Fabry patients in study population were 94% and 100%, respectively.
Hydroxychloroquine-induced Cardiotoxicity
Renal Failure
Infiltrative cardiomyopathies are characterized by the deposition of abnormal substances that cause the ventricular walls to become progressively rigid, thereby impeding ventricular filling. Some infiltrative cardiac diseases increase ventricular wall thickness, while others cause chamber enlargement with secondary wall thinning. Increased wall thickness, small ventricular volume, and occasional dynamic left ventricular outflow obstruction (e.g., amyloidosis) can outwardly appear similar to conditions with true myocyte hypertrophy (e.g., hypertrophic cardiomyopathy, hypertensive heart disease). Likewise, infiltrative disease that presents with a dilated left ventricle with global or regional wall motion abnormalities and aneurysm formation (e.g., sarcoidosis) may mimic ischemic cardiomyopathy. Low voltage (QRS complex was the sine qua non of infiltrative cardiomyopathy [e.g., cardiac amyloidosis). However, low voltage QRS complex is not a universal finding with the infiltrative cardiomyopathies. The clinical presentation, along with functional and morphologic features, often provides enough insight to establish a working diagnosis. In most circumstances, however, tissue or serologic evaluation is needed to validate or clarify the cardiac diagnosis and institute appropriate therapy. (J Am Coll Cardiol 2010;55:1769-79)
56 y/o Woman with a history of radiation therapy for Hodgkin’s lymphoma at age 14
Radiation Induced Cardiac Disease

- Pancarditis: pericardial, myocardial, endocardial/valvular (fibroelastosis)
- Acute pericarditis during therapy
- Delayed pericarditis: constriction, pericardial effusion
- Cardiomyopathy: diastolic/systolic dysfunction
- CAD: intimal proliferation, endothelial dysfunction
- Conduction system defects
Radiation Induced Cardiac Disease
Risk Factors

- Total radiation dose
- Younger age during radiation therapy
- Higher percentage anteroposterior vs. tangential beam trajectory
- Anthracycline therapy: cardiomyopathy and valvular disease
- Smoking, hyperlipidemia, DM: CAD

Radiation Therapy for Hodgkin’s Lymphoma Cardiovascular Effects in 404 Patients  
(Treated 1962-1998)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Incidence</th>
<th>Median Time After Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary Artery Disease</td>
<td>10.4%</td>
<td>9 Yrs</td>
</tr>
<tr>
<td>Carotid ± Subclavian Disease</td>
<td>7.4%</td>
<td>17 Yrs</td>
</tr>
<tr>
<td>Significant Valvular Disease</td>
<td>6.2%</td>
<td>22 Yrs</td>
</tr>
</tbody>
</table>

Hull MC, et al. JAMA 2003; 290:2831
Radiation Therapy for Hodgkin’s Lymphoma Clinically Significant Valvular Disease

- Aortic Stenosis (48%)
- Mitral Regurgitation (28%)
- Mitral Stenosis (10%)
- Tricuspid Regurgitation (10%)
- Aortic Regurgitation (4%)

Hull MC, et al. JAMA 2003; 290:2831
Drug-Induced Valvular Disease
Echocardiographic Findings

- Thickening and retraction of valve leaflets or cusps
  - No commissural fusion
  - Reduced mobility, restricted closure coaptation
- Thickened, fused, shortened MV/TV chordal support apparatus
- Variable regurgitation, rarely significant stenosis

Mimics Rheumatic Valve Disease
Ergot Induced Valve Disease
**MDMA (3,4-Methylenedioxymethamphetamine) Echo Findings with “Ecstasy” Abuse**

<table>
<thead>
<tr>
<th></th>
<th>MDMA Users (n=33)</th>
<th>Controls (n=29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of use</td>
<td>6.1 ± 3.4 yrs</td>
<td>0</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>24.3 ± 3.1</td>
<td>25.6 ± 3.1</td>
</tr>
<tr>
<td>MR ≥ Grade 2/4</td>
<td>4 (14%)</td>
<td>0</td>
</tr>
<tr>
<td>Restricted MV motion</td>
<td>7 (24%)</td>
<td>0</td>
</tr>
<tr>
<td>TR ≥ Grade 2/4</td>
<td>13 (45%)</td>
<td>0</td>
</tr>
<tr>
<td>Restricted TV motion</td>
<td>7 (24%)</td>
<td>0</td>
</tr>
<tr>
<td>AR ≥ Grade 1/4</td>
<td>4 (14%)</td>
<td>0</td>
</tr>
</tbody>
</table>

- Prevalence of MDMA abuse: 0.4 – 6% worldwide

Droogmans S, et al. Am J Cardiol 2007; 100: 1442
A 60 year old male farmer is referred for evaluation of dyspnea

- NYHA Class III symptoms
- PMH: Type 2 DM
- Abnormal LFT’s
- Physical Exam:
  - 110/70 mmHg, HR 70 BPM
  - S3 gallop
  - Bronze skin
Normal sinus rhythm
Premature atrial complexes
Left bundle branch block
with secondary ST–T abnormalities
When compared with ECG of 16-AUG-2007 16:16,
Premature atrial complexes are now present
and T waves have changed

Technician ID: 582

Referred by: 10650
Confirmed By: STEPHEN HAMMILL MD
Apical 4 Chamber View
Coronary Angiography
Coronary Angiography
What would you recommend next to help establish the diagnosis?

1. Cardiac Endomyocardial Biopsy
2. Cardiac MRI
3. Cardiac CT
4. Dobutamine Stress Echo
Cardiac Cine-MRI
Contrast MRI: No delayed Hyperenhancement
60 year old male farmer with Type 2 DM, bronze skin, and abnormal LFT’s. What is the most likely diagnosis?

a. Cardiac hemochromatosis
b. Cardiac amyloidosis
c. Cardiac sarcoidosis
d. Fabry’s Disease
e. Carcinoid syndrome
Hemochromatosis

- ↑ total body iron – intracellular deposits in heart, liver, pituitary, pancreas, gonads, skin
Iron-Overload Cardiomyopathy: Pathophysiology, Diagnosis, and Treatment

COLM J. MURPHY, MD, FRCPC, AND GAVIN Y. OUDIT, MD, PhD, FRCPC

Edmonton, Alberta, Canada

ABSTRACT

Background: The prevalence of primary (hereditary) hemochromatosis and secondary iron overload (hemochromatosis) is reaching epidemic levels worldwide. Iron-overload leads to excessive iron deposition in a wide variety of tissues, including the heart and endocrine tissues.

Methods and Results: Iron-overload cardiomyopathy is the primary determinant of survival in patients with secondary iron overload, while also being a leading cause of morbidity and mortality in patients with primary hemochromatosis. Iron-induced cardiovascular injury also occurs in acute iron toxicosis (iron poisoning), myocardial ischemia-reperfusion injury, cardiomyopathy associated with Friedreich ataxia, and vascular dysfunction. The mainstay therapies for iron overload associated with primary hemochromatosis and secondary iron overload is phlebotomy and iron chelation therapy, respectively. L-type Ca²⁺ channels provide a high-capacity pathway for ferrous (Fe²⁺) uptake into cardiomyocytes in iron-overload conditions; calcium channel blockers may represent a new therapeutic tool to reduce the toxic effects of excess iron.

Conclusions: Iron-overload cardiomyopathy is an important and potentially reversible cause of heart failure at an international scale and involves diastolic dysfunction, increased susceptibility to arrhythmias and a late-stage dilated cardiomyopathy. The early diagnosis of iron-overload cardiomyopathy is critical since the cardiac dysfunction is reversible if effective therapy is introduced before the onset of overt heart failure. (J Cardiac Fail 2010;16:888–900)

Key Words: Cardiomyopathy, hemochromatosis, oxidative stress, anemia, cardiac MRI, echocardiography.
Hemochromatosis

- Think of this when DCM seen in setting of hepatic dysfunction; diabetes, tanned skin
- Diagnosis is critical, since reversible
  - Males 9:1
  - 2-3/1000 population
  - Ferritin usually > 500, transferrin > 50%
- Normal wall thickness
- Arrhythmias, conduction abnormalities

Intracellular iron
- directly toxic to myocytes

Courtesy of William Edwards, MD
26 year old with Hemochromatosis
After Tx with Deferoxamine
The evaluation of the T2* relaxation time is an excellent noninvasive correlate of myocardial iron deposition and is a useful technique to follow response to iron-chelation therapy.

Myocardial T2* has been shown to have no relation to serum ferritin and liver iron overload.

T2* relaxation time predicts CHF and Arrhythmias.

Circulation 2009;120:1961-8
Take Home Points

• The Iron Heart is a weak heart…
• Hemochromatosis may be a cause of idiopathic dilated cardiomyopathy
  – Reversible with treatment
• Cardiac MRI (T2 relaxation time) is important in helping to establish diagnosis and monitoring treatment effects
Circulation Function and Treatment in β-Thalassemia Major: A Consensus Statement From the American Heart Association


on behalf of the American Heart Association Committee on Heart Failure and Transplantation of the Council on Clinical Cardiology and Council on Cardiovascular Radiology and Imaging

Circulation. 2013;128:281-308; originally published online June 17, 2013;
28 year old male with hemophilia and dyspnea
HIV and Cardiac Disease

• Clinical cardiac involvement - 10% AIDS
  – Myocarditis (50% at autopsy)
  – Ventricular arrhythmias
  – Heart failure (DCM)
  – Pericarditis and effusions
  – Infectious or malignant invasion
  – Diastolic dysfunction
  – Pulmonary Hypertension?

Conclusions:
Systemic Diseases and the Echo Boards

- Carcinoid Syndrome
- Hypereosinophilic endomyocardial disease
- Sarcoidosis
- Systemic Lupus Erythematosus
- Scleroderma/Crest: Pulm Hypertension
- Amyloidosis
- Hyper or Hypothyroidism
- Radiation Heart Disease
- Drug Induced Valve Disease
- Hemochromatosis
Thank You!
mankad.sunil@mayo.edu

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