Case Based Review of MDCT: Valves, function

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Disclosures

Consultant: GE-Healthcare

62 yo male referred for evaluation a dilated aortic root
What is the abnormality of the aortic root that is present?

1) dilatation of the aortic sinuses
2) aortic regurgitation
3) bicuspid aortic valve
4) rheumatic aortic valve

Which of these is NOT associated with bicuspid aortic valve?

1) ascending aortic dilatation
2) coarctation of the aorta
3) aortic stenosis and regurgitation
4) 10 fold increased rate of aortic dissection
5) 8% incidence in the population

- Normal aortic valve has 3 leaflets
- Bicuspid valve has fusion of 2 valve leaflets

http://www.med.yale.edu/intmed/cardio/echo_atlas/entities/aortic_stenosis_bicuspid.html

2 mm reconstructions through the aortic valve
Which of these is NOT associated with bicuspid aortic valve?

5) 8% incidence in the population

**Overall, 1-2% incidence in the population**

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**Bicuspid aortic valve**

1) Associated with dilatation of the ascending aorta.

• NOT necessarily due to hemodynamic consequences of the stenosis.

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**Bicuspid aortic valve**

1) Associated with dilatation of the ascending aorta.

• Aortic dilatation more rapid in bicuspid valves compared to normal valves with equivalent degrees of stenosis.

• Inherent structural abnormality in the aortic wall proposed (hereditary); shorter (6 month) follow-up intervals recommended)

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**Bicuspid Aortic Valves Are Associated With Aortic Dilatation Out of Proportion to Coexistent Valvular Lesions**


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**Bicuspid Ao valve – other associations**

*Coarctation* of the aorta:

• Bicuspid aortic valve present in 25-50% of patients with coarctation

*Aortic dissection*:

• 10 x increased rate compared to normal population
39-year old male with a 3-year history of highly symptomatic paroxysmal atrial fibrillation, for catheter ablation.

- Previously failed antiarrhythmic drug therapy with quinidine, and propafenone.
- CT angiography performed for assessment of pulmonary vein anatomy prior to EP ablation.

The purpose of pulmonary vein ablation in this setting (afib) is:

1) make the pulmonary veins smaller to decreases blood flow
2) create a continuous scar that blocks electrical conduction pathways from the pulmonary veins to the left atrium
3) temporarily stabilize the patient before definitive surgery

Key elements of a report for pulmonary vein analysis include:

- description of pulmonary vein size
- description of pulmonary vein variant anatomy
- evaluation for atrial clot
- description of incidental findings

http://www.sts.org/sections/patientinformation/arrhythmiasurgery/afib/

Analysis to include double oblique reformation of each pulmonary vein ostia

Pulmonary vein variant anatomy

Report long and short axis diameters, or areas
What anatomic variant is present for the left pulmonary veins that may affect the ablation procedure?

1) aneurysm of the left pulmonary vein
2) stenosis of the left pulmonary vein
3) common ostium for the left superior/ inferior veins
4) absent left superior vein
True statements regarding complications of pulmonary vein ablation:

1) Overall complication rate of 4%
2) Significant pulmonary vein stenosis is uncommon (0.4%)
3) Cardiac tamponade and cerebral embolus are complications
4) all of these


1011 patients
- Overall cx rate: 3.9%
- peripheral vascular cx: 1.2%
- pericardial effusion: 0.8%
- cardiac tamponade: 0.6%
- cerebral aneurysm: 0.5%
- significant pulmonary vein stenosis: 0.4%
Pre Post
Baseline Follow-up
CT scan, 1 week after HIFUS afib ablation procedure; chest pain, dysphagia
Esophageal perforation
Esophageal thickening
Left Atrial Appendage Evaluation
Which is true regarding LAA thrombus evaluation by CT prior to pulmonary vein ablation:
1) Incidence is up to >33% of afib patients
2) Sensitivity of gated MDCT is near 100%
3) Thrombus is identified by enhancement >30 HU above baseline
4) Incidence is ≤ 10%
Inferior pulmonary venous ostium
Which is true regarding LAA thrombus evaluation by CT prior to pulmonary vein ablation:

4) Incidence is ≤10%

- 1/178 patients had clot
- Johns Hopkins, 5/50 high risk patients
- routine anticoagulation, TEE screening account for low incidence rate

LA Clot:
- Clot enhancement not used for diagnosis
- Reported CT sensitivity: 40-100%

61 yo with chest pain 4 days post MI
- Referred to CT for evaluation of pulmonary embolism

Etiology of focal bulge LV wall:
1) subepicardial aneurysm
2) myocardial cleft, variant
3) rupture with false aneurysm
Etiology of focal bulge LV wall:

1) subepicardial aneurysm

In this setting, what is the reason for decreased cardiac attenuation on MDCT?

Reason for decreased cardiac attenuation (best answer):

1) acute myocardial infarction
2) inflammatory change due to pneumonia
3) transient perfusion, normal variant
4) artifact due to MDCT gating

Reason for decreased cardiac attenuation (best answer):

1) acute myocardial infarction

Appropriate management:

1) medical therapy alone
2) emergency cardiac catheterization
3) routine care
4) emergency cardiac surgery/repair
### Appropriate management

4) emergency cardiac surgery/repair
- Represents an impending myocardial rupture with high likelihood of death
- Lesion was patched at surgery

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### Acute Myocardial Infarction: Contrast-enhanced MDCT in a Porcine Model

Udo Hoffman, Ryan Milleva, Christian Enzweiler, Maros Ferencik, Scott Gulick, Jim Titus, Stephan Achenbach, Dylan Kwait, David Sosnovik, Thomas J. Brady


- Porcine AMI model (N=7)
- 4 slice MDCT scanning
- 3 hours post-coronary ligation – LAD or LADD
- CT Infarct size 17 ± 6 % similar to TTC 14 ± 6 %

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### Myocardial Viability by MDCT

Lower density in the area of infarction


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### LV infarct, thrombus

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### 1st pass perfusion abnormality

Circulation 2006 113:394

Albert C. Ludoo, MD; Marco A. S. Cordero, MD; PhD; Caterina Sitora, MD; Luciano C. Amado, MD; Richard T. George, MD; Assafia P. Zafirakis, MD; Ernst R. Schuder, MD; Veronica R. Fernandes, MD; M. B. Zieniszewski, MD; Nane Natanian, MD; Hosen H. Hafiz, MD; Natarese C. Wu, MD; J. M. H. Hess; MD; John A. C. Lima, MD
39 yo female with irregular heart rate, PVC’s

- ICD placed 2 years previously
- Multiple appropriate shocks
- CT scan to assess for cardiac anatomy and function

39 yo female, irregular heart rate, PVC’s

39 yo female, irregular heart rate, PVC’s

Which finding is NOT present:

1) Enlargement of the left ventricle
2) Enlargement of the right ventricle
3) Hypertrabeculation of the right ventricle
4) Dysfunction of the right ventricle

Which finding is NOT present:

1) Enlargement of the left ventricle
2) Enlargement of the right ventricle
3) Hypertrabeculation of the right ventricle
4) Dysfunction of the right ventricle
### Which of these does **NOT** cause RV enlargement in an adult?

1. ARVD/C  
2. Pulmonary hypertension  
3. Intracardiac L→R shunt  
4. Extracardiac L→R shunt  
5. Amyloidosis

### Which of these does **NOT** cause RV failure and enlargement in an adult?

5. Amyloidosis

Cardiac amyloidosis causes restrictive cardiomyopathy, left-sided symptoms usually predominate.

### RV enlargement, differential diagnosis in the setting of suspected ARVD

- Normal variant (young age, <20)
- PAPVR
- Cardiac shunt or valve
- Other cardiomyopathy (rare)
- Pulmonary hypertension

### 2nd patient, same hx. Best diagnosis:

1. ARVD  
2. Pulmonary hypertension  
3. Intracardiac L→R shunt  
4. Extracardiac L→R shunt

### 2nd patient same hx and diagnosis:

1. ARVD  
   Arrhythmogenic right ventricular dysplasia

### ARVD CT findings

- Enlarged RV (RV/LV diameter > 1)  
- Relatively preserved LV  
- Very thin, atrophic RV wall  
- LV fatty replacement
CT findings for ARVD

- RV fatty wall replacement
- Relatively preserved LV
- Very thin, atrophic RV wall
- LV fatty replacement

Nonspecific CT findings for ARVD

- RV fatty wall replacement

Arrhythmogenic RV Dysplasia

- Fibrofatty infiltration of RV resulting in ventricular tachycardia
- Palpitations, syncope, sudden death
- Age 20-40.
- 30-50% cases are familial (autosomal dominant). MR screening of family members common, and likely to increase with improved genotyping.

Genes identified with mutations causing ARVD

<table>
<thead>
<tr>
<th>Gene</th>
<th>Protein</th>
<th>Inheritance</th>
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<tbody>
<tr>
<td>PKP2</td>
<td>plakophilin-2</td>
<td>dominant</td>
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“McKenna” Criteria:

2 major, 1 major + 2 minor, 4 minor*

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Major</th>
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<tbody>
<tr>
<td>Abnormal structure/function</td>
<td>• Severe dilatation and reduction of RV EF</td>
</tr>
<tr>
<td></td>
<td>• Localized RV aneurysms</td>
</tr>
<tr>
<td>ECG repolarization or depolarization abnormalities</td>
<td>• Severe segmental dilatation of the RV</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>QRS prolongation</td>
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<td>Family history</td>
<td>Confirmed at autopsy or surgery</td>
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Desmosome: mechanical cell coupling
35 yo female, recurrent palpitation and syncope

- Mother – sudden death at age 38
- 2 brothers: cardiac disease
- MDCT after 150 ml iodine contrast

Best diagnosis:
1) hypertrophic cardiomyopathy
2) anterior wall infarction
3) sarcoidosis
4) myocarditis

MRI, same patient

Best diagnosis:
1) hypertrophic cardiomyopathy
Current understanding of enhancement in hypertrophic cardiomyopathy

1) enhancement is associated with congestive failure
2) enhancement relates to better prognosis, fewer arrhythmias
3) enhancement corresponds to collagen deposition

Hypertrophic Cardiomyopathy

- abnormal muscle fibers, leading to thickened heart walls.
- *Myocardial disarray*: disorganized myocytes with fibrosis
- 1/500, 0.2% in the U.S.
- Chest pain, syncope, sudden death

Hypertrophic Cardiomyopathy (HOCM)

- Most common cause of sudden cardiac death <30 yrs old
- *(This case: death in the mother, both male siblings were affected)*

Symmetric Apical variant
Asymmetric septal hypertrophy

Systolic anterior motion (SAM) of the mitral valve at late systole, resulting in aortic outflow obstruction.

Hypertrophic cardiomyopathy

Delayed gadolinium enhancement correlates with collagen deposition but not myocardial disarray.¹

¹Moon et al, JACC, Volume 43, Issue 12, 16 June 2004, Pages 2260-2264

Hypertrophic CM: 80% show patchy intramural delayed enhancement

Cine

Delayed contrast

Late enhancement of the left ventricular wall in hypertrophic cardiomyopathy by ultrafast CT: a comparison with regional myocardial thickening


Saito H, Naito H, Takamiya M, Hamada S, Imakita S, Ohta M.

• 8 patients with HCM with ultrafast CT
• Late enhancement was patchy, on delayed phase
• Out of 48 LV segments, 21 had LE.
• Regional wall thickening in segments with LE was less segments without LE
Thank you

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