Role of non-invasive imaging in the evaluation of athlete's heart: differentiating physiological from pathological changes

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The Athlete’s Heart

“...morphological and electrical remodeling which occurs to varying extents dependent upon the sporting discipline.”


22% Harvard University Athletes had echo evidence of physiologic, exercise-induced cardiac remodeling.

RWT = \((2 \times \text{PWT})/\text{LVEDd}\)
“Athlete’s Heart”: Sport-specific EICR

Characteristic Adaptations:
- Mild to moderate Eccentric LVH & RV dilation
- Biatrial enlargement
- Normal to slightly reduced resting LVEF
- Normal or enhanced early diastolic function
- Normal or enhanced LV twisting/untwisting
“Athlete’s Heart”: Sport-specific EICR

Characteristic Adaptations:
• Mild concentric LVH but no RV remodeling
• Normal to mildly enlarged LA size
• Normal to hyperdynamic resting LVEF
• Normal to slightly reduced early LV diastolic function
• Compensatory increase in Late LV diastolic function
“Athlete’s Heart”: Sport-specific EICR

Normal “Pre-training” Cardiac Structure and Function

Endurance Training

Strength Training

RV Dilation +/- Mild RVH
Eccentric LV Hypertrophy

RV No Δ
Concentric LV Hypertrophy

Common Causes of Sudden Cardiac Death in Young Athletes

Structural Cardiac Abnormalities
- Hypertrophic cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy
- Congenital coronary artery anomalies
- Marfan syndrome
- Mitral valve prolapse/Aortic stenosis

Electrical Cardiac Abnormalities
- Wolff Parkinson White syndrome
- Congenital long QT syndrome
- Brugada syndrome
- Catecholaminergic polymorphic ventricular tachycardia

Acquired Cardiac Abnormalities
- Infection (myocarditis)
- Trauma (commotio cordis)
- Toxicity (illicit/performance enhancing drugs)
- Environment (hypo/hyperthermia)

Comparison of Causes of SCD in Athletes

CMP → Athlete’s Heart → “Grey Zone”
Impact of Ethnicity

Myocardial Thickening

Physiologic Concentric LVH

HCM
Hypertensive CMP
Non-compaction
Infiltrative CMP
Pelliccia. Progress in Cardiovascular Diseases. 2012


18% BA
4% WA
Myocardial Mechanics

Caselli. JASE 2015: 236-244

Afonso. BMJ 2012

<table>
<thead>
<tr>
<th></th>
<th>Between HCM and AT-LVH*</th>
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<tbody>
<tr>
<td></td>
<td>Area (95% CI)</td>
</tr>
<tr>
<td>Septal wall thickness (mm)</td>
<td>1.000 (0.998 to 1.001)</td>
</tr>
<tr>
<td>LV posterior wall thickness (mm)</td>
<td>0.908 (0.843 to 0.972)</td>
</tr>
<tr>
<td>Indexed LA dimension (cm/m²)</td>
<td>0.921 (0.861 to 0.981)</td>
</tr>
<tr>
<td>LV fractional shortening (%)</td>
<td>0.714 (0.580 to 0.848)</td>
</tr>
<tr>
<td>ThDI</td>
<td>0.952 (0.909 to 0.995)</td>
</tr>
<tr>
<td>Tissue Doppler imaging:</td>
<td></td>
</tr>
<tr>
<td>S' wave (cm/s)</td>
<td>0.912 (0.897 to 0.937)</td>
</tr>
<tr>
<td>E' wave (cm/s)</td>
<td>0.995 (1.005 to 0.984)</td>
</tr>
<tr>
<td>A' wave (cm/s)</td>
<td>0.666 (0.796 to 0.535)</td>
</tr>
<tr>
<td>GLS-avg (%)</td>
<td>0.920 (0.862 to 0.978)</td>
</tr>
<tr>
<td>SDI</td>
<td>0.890 (0.818 to 0.961)</td>
</tr>
</tbody>
</table>
LVH Regression with Detraining

Eccentric LVH

\[\downarrow 15\% (1.9\text{mm})\]

Concentric LVH

Weiner J Am Coll Cardiol. 2012

Pelliccia. Circulation 2002
Value of cMRI

A

RV

LV

B

RV

LV

C

RV

LV

*
Respective contribution of cardiovascular exams

14 athletes (9%) with normal hearts on TTE
Physiological vs. Pathological

- Eccentric or concentric LVH without regional variability
- Asymmetrical or regional hypertrophy is abnormal
- Wall thickness > 15mm is pathological entire proven otherwise
- Abnormal diastolic parameters are helpful
- Quantifying functional capacity ($VO_{2\text{max}}$) can be useful
- Additive value of Cardiac MRI (especially with an abnormal ECG)
- Detraining may be required
Left Chamber Dilation

Physiologic
Eccentric LVH

Familial DCM
Idiopathic DCM
Toxic DCM
Valvular Heart Disease

Mean 55.5±4.3mm

14%
LV adaptations in World-Class Pro Cyclists

LVIDd >60mm (51%)

EF 40-52% (7%)

Chamber Enlargement Response to Detraining

Average ↓7% (4mm)

Pelliccia. Circulation 2002
Physiology vs. Pathology

- Eccentric LVH vs Dilated Cavity
- Concomitant RV & LA enlargement
- Normal to supranormal myocardial mechanics
- Exercise testing
  - Stress echo
  - CPET
- Absolute cutoffs especially with detraining less useful
Right Chamber Dilation

Physiologic RV Dilation

ARVC
RV Afterload States

RV 5.5cm
LV 4.5cm
RV Size in Athletes

28% had values greater than the proposed “major criteria” for ARVC

# Table 2 RV functional parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD (range)</th>
<th>ASE normal value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV ε (%)</td>
<td>-27 ± 6 (-18 to -41)</td>
<td>-18 to -39</td>
</tr>
<tr>
<td>RV SRS' (sec⁻¹)</td>
<td>-1.53 ± 0.43 (-0.75 to -2.63)</td>
<td>-0.7 to -2.54</td>
</tr>
<tr>
<td>RV SRE' (sec⁻¹)</td>
<td>2.00 ± 0.61 (0.87 to 3.76)</td>
<td>NA</td>
</tr>
<tr>
<td>RV SRA' (sec⁻¹)</td>
<td>1.25 ± 0.56 (0.28 to 2.88)</td>
<td>NA</td>
</tr>
<tr>
<td>RV FAC (%)</td>
<td>47 ± 7 (35 to 61)</td>
<td>35 to 63</td>
</tr>
<tr>
<td>RV S' (cm/sec)</td>
<td>11 ± 1.3 (7 to 14)</td>
<td>&gt;6</td>
</tr>
<tr>
<td>RV E' (cm/sec)</td>
<td>-10 ± 2.1 (-6 to -17)</td>
<td>NA</td>
</tr>
</tbody>
</table>

FAC, Fractional area change; NA, not available.

*Rudski et al.⁹
Physiological vs. Pathological

- Associated with LV remodeling (absolute dimensions not helpful)
- Normal to low normal function
- Normal to supranormal RV mechanics
- Exercise testing
  - Stress echo
  - CPET
- Not associated with functional or structural changes of ARVC
- cMRI can be helpful
Pathology

- Symptoms
- Family History
- ECG
- LVH Type/distribution
- Arrhythmias
- Peak VO2
- Stress EF
- Late Gado Enhancement
- Δdeconditioning
Thank You!
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