Cardiac MRI: Role in diagnosis or exclusion of heart disease

Dr. Matthew Barrett, Consultant Cardiology and Cardiovascular Imaging
Beacon Hospital
Current challenges in cardiology

- People living longer
  - Greater incidence of cardiovascular disease, heart failure

- Greater availability of devices / advanced interventions
  - More people surviving acute events to develop chronic cardiovascular disease
  - Greater impetus to use advanced therapy appropriately
    - Revascularisation of chronic total coronary artery occlusion
    - Implantation of ICD / Cardiac resynchronisation device

- Better medical therapies
The traditional toolbox

- ECG
- Holter
- Stress ECG
- Echocardiography
- Invasive angiography
The traditional toolbox

- ECG
- Holter
- Stress ECG
- Echocardiography
- Invasive angiography
Echocardiogram

- Used as standard part of cardiac assessment
- First used in 1953 for generation of simple M-Mode images
- Advanced with use of 2-D, 3-D, doppler and strain imaging
- Fast bedside global assessment of myocardial function, valve function and a basic idea of intracardiac haemodynamics
BUT
Limitations

- Generation of high quality echocardiographic images dependent on many variables
  - Patient body habitus
  - Patient positioning
  - Ability to tolerate pressure from probe
  - Operator experience
  - Operator technique
  - Absence of significant pulmonary disease

- Lacks ability to characterise tissue
  - Scar
  - Oedema
  - Dead wall vs stunned wall
Structures not visualised by echo

- Proximal coronary arteries
- Left atrial appendage
- Pulmonary veins
- Branch pulmonary arteries
Further limitations

- Without ability to characterise tissue, echocardiography lacks sufficient discriminatory power to clarify aetiology of heart failure.
- Also lacks the ability to identify disease phenotypes where myocardial thickness, wall motion or valves are unaffected.
What can we tell from echo vs CMR?

**Echo**
- Degree of chest tenderness
- LVEF
- RWMA
- Wall thickness
- Valves
- Shunt

**Cardiac MRI**
- Fibrosis/scar
- Viability
- Myocardial perfusion
- Quantitative RV function
- Proximal coronary anatomy
Clinical scenarios where echo struggles
Not all LVH is created equal

- Hypertensive heart
- Athletic heart
- ‘Normal’ ageing – sigmoid septal hypertrophy
- Hypertrophic cardiomyopathy
- Cardiac amyloid
- Cardiac sarcoid
- Anderson-Fabry disease
Not all cardiomyopathies are created equal

- Ischaemic cardiomyopathy
- Dilated cardiomyopathy (DCM)
- Hypertrophic cardiomyopathy (HCM)
- Infiltrative cardiomyopathy
- Myocarditis
- Left ventricular non-compaction (LVNC)
- Arrhythmogenic right ventricular cardiomyopathy (ARVC)
Not all coronary disease is created equal

- Accurate quantification of LVEF – could device therapy be required in addition to revascularisation?
- Extent of myocardial fibrosis and likelihood to respond to revascularisation – is stenting worthwhile or are we resupplying blood to dead meat?
- Is there an alternative process? CAD may be a bystander
  - Angiogram is an anatomical test – the presence of stenosis and the absence of adequate myocardial perfusion are not necessarily the same thing
- Chronic total occlusion of a coronary artery – is the muscle supplied by the artery still viable?
Gadolinium Contrast

- Allow differentiation of normal myocardium from fibrosis or infarction
- Gadolinium unable to cross intact cell membrane – rapid ‘washout’
- Delayed MRI scanning after Gd administration identifies areas of disarray of intact cell membranes (acute necrosis) or expanded extracellular matrix (fibrosis)
The value of scar imaging

Qualitative

• Scar location and character gives clues as to underlying diagnosis
• Presence vs absence of scar a major predictor of mortality

Quantitative

• Greater scar burden – greater concern re: ventricular arrhythmias, sudden death etc
Differentiating the ‘normal’ abnormalities from ‘abnormal’ abnormalities
Common echo findings

- Slightly dilated atria or ventricles
  - Early heart failure?
  - Dilated cardiomyopathy?
  - Athletic remodelling?

- Slightly thickened LV or RV wall
  - Hypertensive?
  - Athletic heart?
  - Hypertrophic cardiomyopathy?
  - Infiltrative disease?

- Mild reduction in LV function
  - Early stage of a progressive cardiomyopathy or just related to bundle branch block / dyssynchrony / previous surgery?
Common ECG / holter findings

- Brief NSVT / AIVR / couplets
- Frequent ectopics
- LBBB
- Nonspecific t-wave inversion
- Syncope or presyncope without definitive ECG changes
Raised troponin without explanation

- ‘Troponinitis’?
- Inflammatory process without frank myocardial dysfunction?
- Cardio-embolic event?
- Angiographic underestimation of disease or ‘flush occlusion’ of a branch coronary artery?
- Infiltrative disease?
Who comes to CMR

- HCM vs HTN/athlete
- Mild HCM vs Athletic Remodelling
- Mild DCM vs Athletic Remodelling
- Abnormal ECG Normal ECHO
- ARVC vs Dilated RV due to exercise
- Troponin Event Arrhythmias Syncope
- LVH
- Ventricular ectopy Normal echo and ECG
- Syncope
Sometimes Echo is enough
The ‘Barn Door’ case

- Asymmetric septal hypertrophy >20mm
- Obvious systolic anterior motion of mitral valve
- Marked LVOT pressure gradient
- Family history of HCM or genetic diagnosis of disease-causing sarcomeric mutation
- Classic ECG changes
The ‘Barn Door’ case

+ DIAGNOSIS OF HCM EASY
For everything else, there’s CMR
The diagnostic challenge

- Young, fit individual
- Concentric LVH 14-17mm
- No SAM / LVOTO
- Lateral repolarisation abnormalities on ECG
- No family hx or risk factors (SCD, CAD, HCM)
- Asymptomatic
Athlete’s Heart

- Challenging condition to confidently diagnose
- Generally a diagnosis of exclusion
- Features
  - Hypertrophy – can mimic HCM
  - Chamber dilatation – can mimic DCM
  - Hypertrabeculation – can mimic LVNC

- CMR CRITICAL TO DIFFERENTIATE
Sample Cases
Case 1

- 41 year old athlete - possible sarcoid
- Vague palpitations, no syncope
- Negative endomyocardial biopsy
- Normal echo and normal 24 hr holter.
Case 2

• 57 yr old male
• Lifelong high level athlete (endurance)
• VF arrest
• Normal angiogram
• Normal echocardiogram
• Mild troponin elevation – ‘was it just the arrest that caused the rise?’
Case 3

- 42 yr old GP.
- Runs 30-50K a week.
- 3 week history of reduced exercise tolerance with mild SOB. Nil else.
- Normal ECG.
- Echo suggestive of reduced RV function – difficult to characterise fully – mildly reduced TAPSE
Case 4

- 56 year old man
- Chronic total occlusion of LAD and RCA
- Reduced LV function – 25% by echo

Questions about next treatment step:
- Complex PCI of both vessels
- Insert ICD
- Transplant
So who needs a CMR?
CMR

Diagnosis

Prognosis

Guide Tx
Diagnosis

- Unexplained LVH
  - No clear history of hypertension, advanced age
  - Young, otherwise healthy

- Unexplained reduction in LV function
  - No clear history of ischaemia, valvulopathy

- Unexplained LBBB, arrhythmia

- Suspicion of familial cardiomyopathy, sudden cardiac death etc

- Systemic conditions – sarcoid, amyloid, haemochromatosis etc – is the heart involved?
Prognosis

- Degree of fibrosis
  - Should an ICD be considered earlier than normal due to severity of scarring?
  - Should transplant be considered rather than standard heart failure therapy?
- Severity of reduced EF – echo images only get you so far
- Likelihood to respond to treatment
- Resolution of scar or improvement in EF over time
Treatment

- Does this person need a device?
  - Is the EF truly below 35% or is the echo inaccurate?
  - Is there minimal scar burden, suggesting we ‘push on’ with medical therapy before considering device?

- Will this person benefit from revascularisation?
  - Is the myocardium ‘dead meat’?
  - Is there a specific perfusion defect – eg LAD territory - that might make surgery preferable to PCI?

- Is the patient’s chest pain truly angina?
  - Is there a perfusion defect or is coronary disease incidental?
Classic Cardiology

Symptoms of chest pain / SOB

History and examination

ECG – nothing too impressive but not 100% normal

Angiogram – moderate non-obstructive disease

Holter – brief episodes of ectopy

‘Medical therapy’

Echocardiogram – mild LVH, normal EF, slightly big LA

Still not better

Desperation
- Respiratory OPD
- Try steps 1-8 again
Modern Cardiology

- Symptoms of chest pain / SOB
  - History and examination
    - ECG / holter as required
    - CMR
      - Cardiac dx made
      - Cardiac dx outruled
Thank you