

Advanced Imaging of Pericardial Diseases

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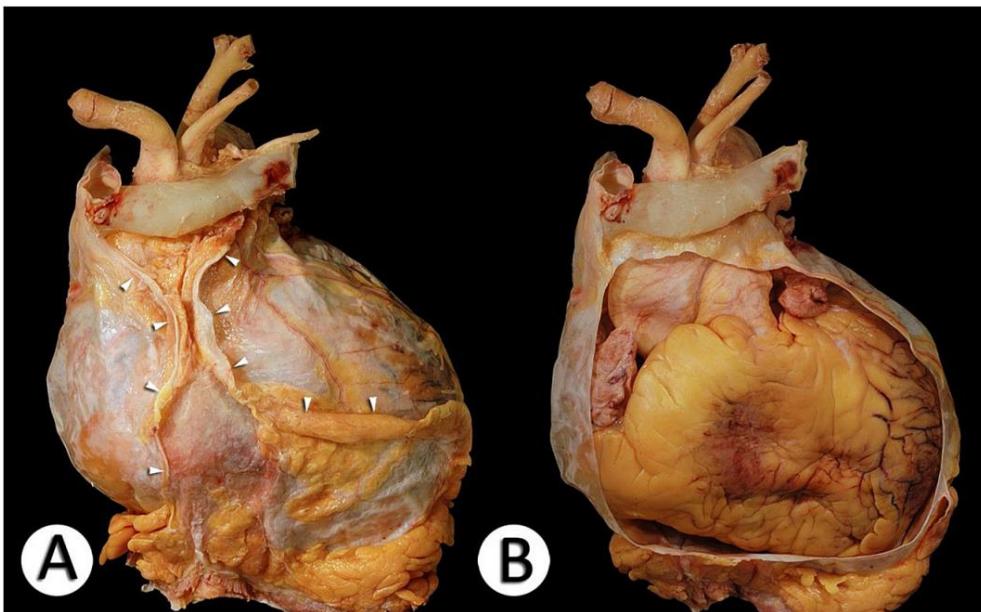


Pericardium

From Greek Πεπερί (around) & κάρδιον (heart)



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- Flask-shaped, double-walled sac containing the heart & the roots of the great vessels
- 2 layers: serous visceral layer & fibrous parietal layer
- Functions:
 - Facilitates cardiac chamber coupling & interaction
 - Lubricates (minimizes friction)
 - Mechanical barrier to infection

Intro to Pericardial Diseases

- Spectrum of disorders:
 - **Infectious & Inflammatory** (acute pericarditis)
 - **Thickening & scarring** (chronic constrictive pericarditis)
 - **Effusions** (to tamponade)
 - **Pericardial masses** (tumors, cysts, and diverticulum)
 - **Congenital processes**
- May be either isolated or part of a systemic disease
- Variable symptoms:
 - chest pain, shortness of breath, ascites, leg swelling, and hypotension
- Accurate diagnosis frequently requires integration of medical history, physical examination, MMI and invasive hemodynamic measurements

Echocardiography: First Line Test



- Readily available, cost effective and comprehensive assessment of pathology and corresponding physiology

Echocardiography

Main indications/advantages

- First-line diagnostic imaging test in the evaluation and follow-up of pericardial disease
- Widely available
- Low cost
- Safe
- Can be performed bedside or in hemodynamically unstable patients

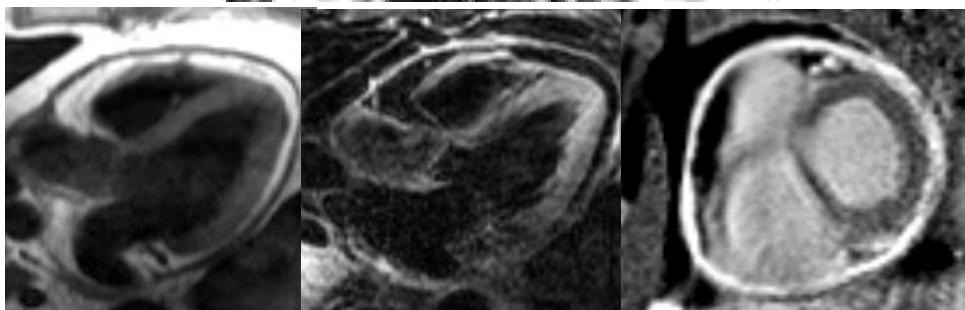
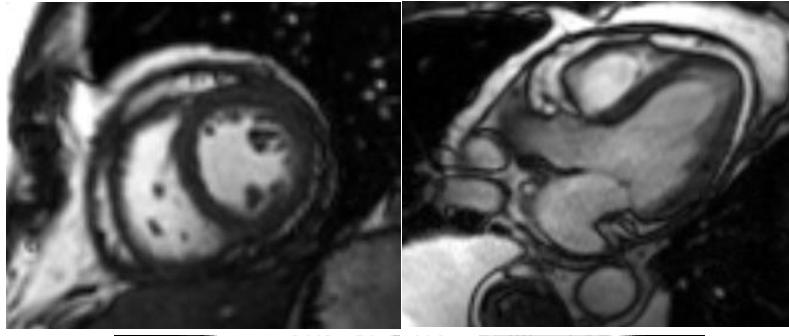
Main limitations/disadvantages

- Limited windows, narrow field of view
- Technical difficulties in case of obesity, obstructive lung disease or immediately post- cardiothoracic surgery
- Operator dependent
- Low signal-to-noise ratio of the pericardium
- Limited tissue characterization
- Loculated effusions

CMR: Complimentary Role in Evaluation & Management



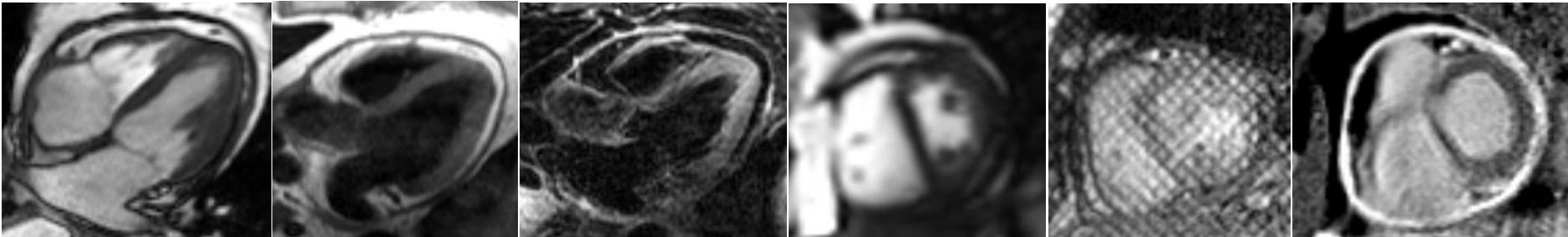
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- Advantages:
 - Large FOV
 - Excellent delineation of pericardial anatomy
 - Physiological information
 - Unique ability to evaluate for inflammation
 - Superior tissue characterization
- Disadvantages:
 - Time
 - CIED
 - Calcifications not seen
 - Use of Gad in GFR<30

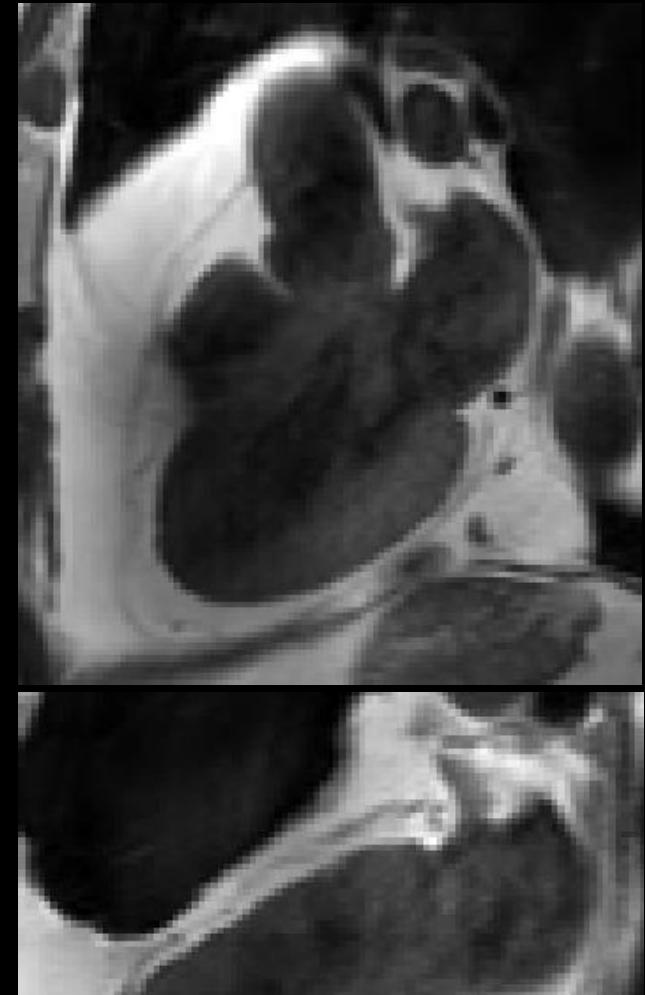
MRI Protocol: Evaluation of Pericardial Disease

- Bright-blood single shot SSFP & dark-blood HASTE *Thoracic anatomy*
- Bright-blood cine images (SSFP) *LV/RV volumes & function*
- Black-blood images (T1W FSE) *Pericardial morphology / thickness*
- Black-blood images (T2W FSE with fat sat) *Pericardial edema*
- Tagged cine images (T1W gradient-echo) *Epicardial / pericardial tethering*
- Real-time SSFP cine imaging *Ventricular interdependence*
- LGE images (PSIR) *Pericardial inflammation*



CMR: Normal Pericardium

T1 weighted spin echo sequences



Pericardial thickness (≥ 4 mm) is abnormal



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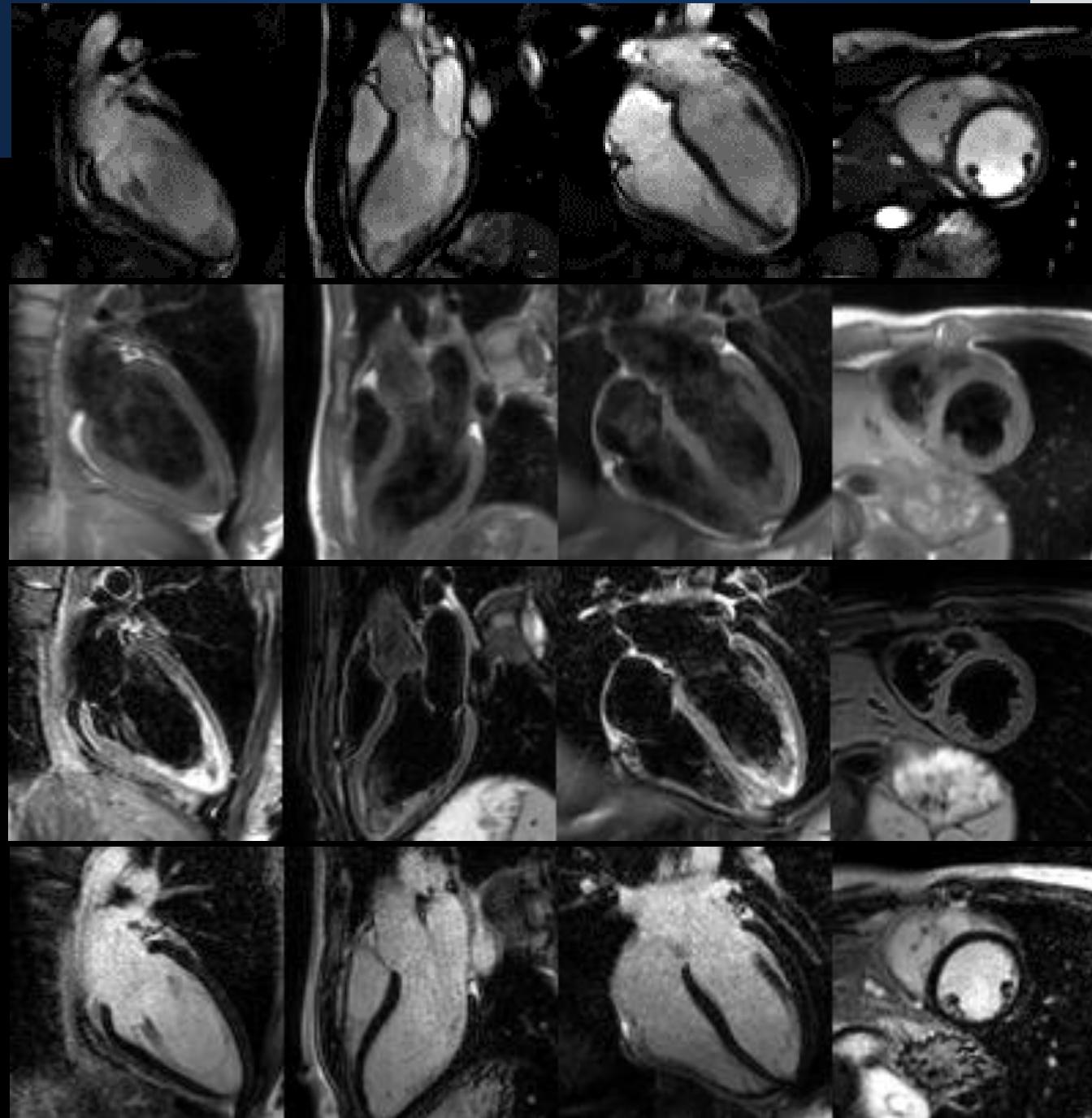
Cine SSFP

T1

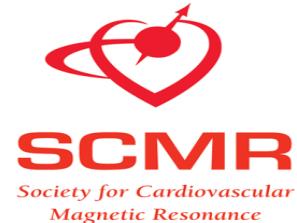
Normal
Pericardial

T2

LGE



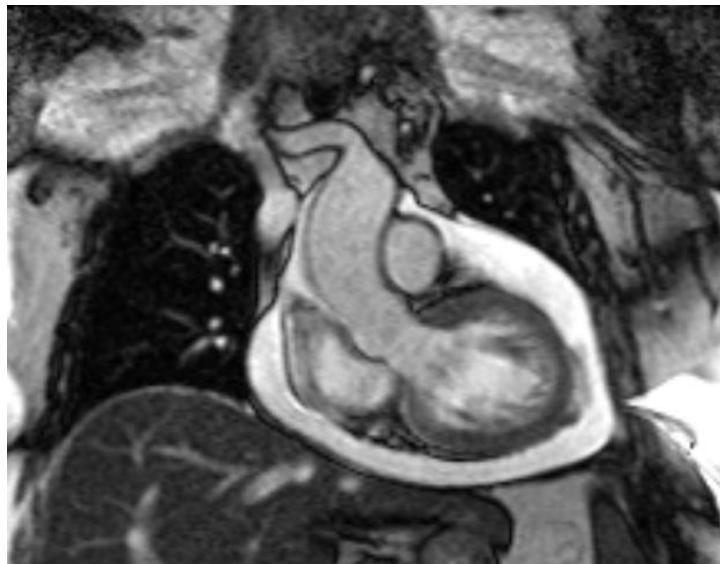
Pericardial Disease: Spectrum of Disorders



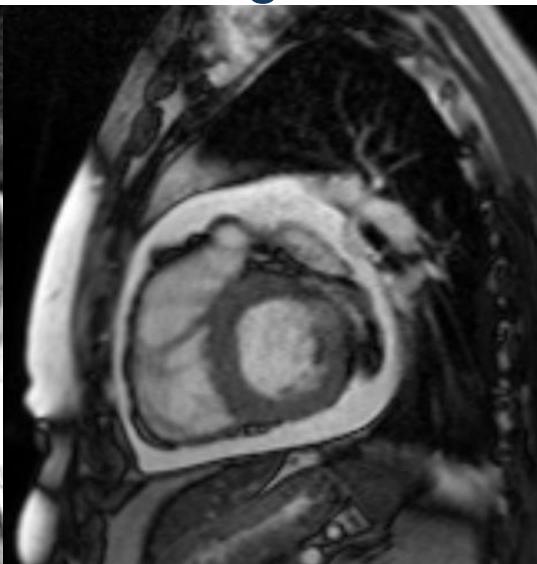
- Effusions (to tamponade)
- Infectious & Inflammatory (acute pericarditis)
- Thickening & scarring (chronic constrictive pericarditis)
- Pericardial masses (tumors, cysts, and diverticulum)
- Congenital processes (Complete/partial absence)

CMR: Pericardial Effusions

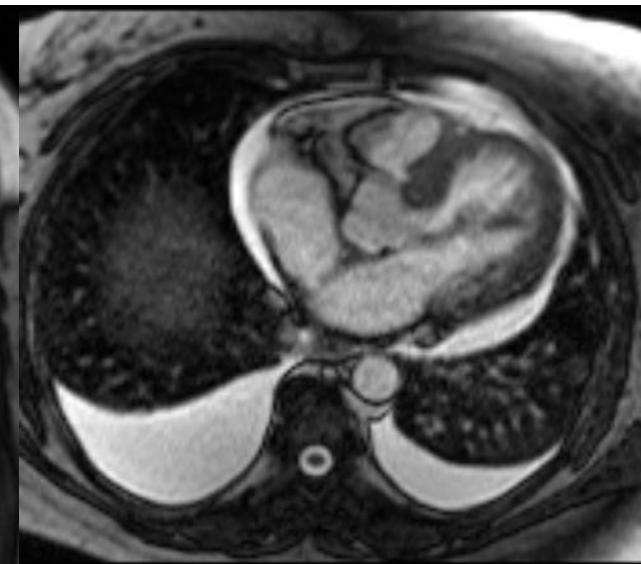
Coronal



Sagittal



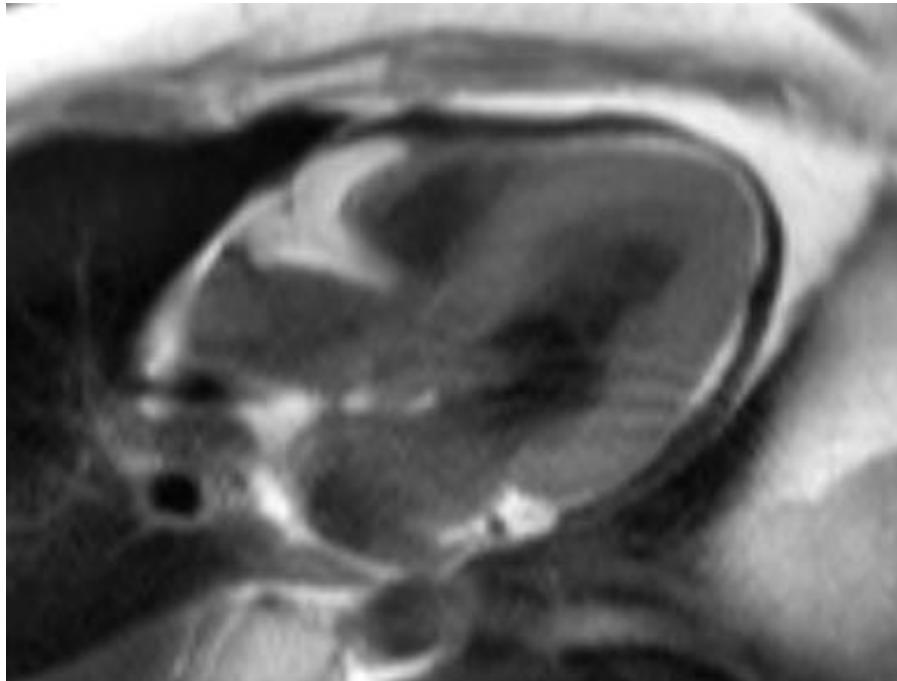
Axial



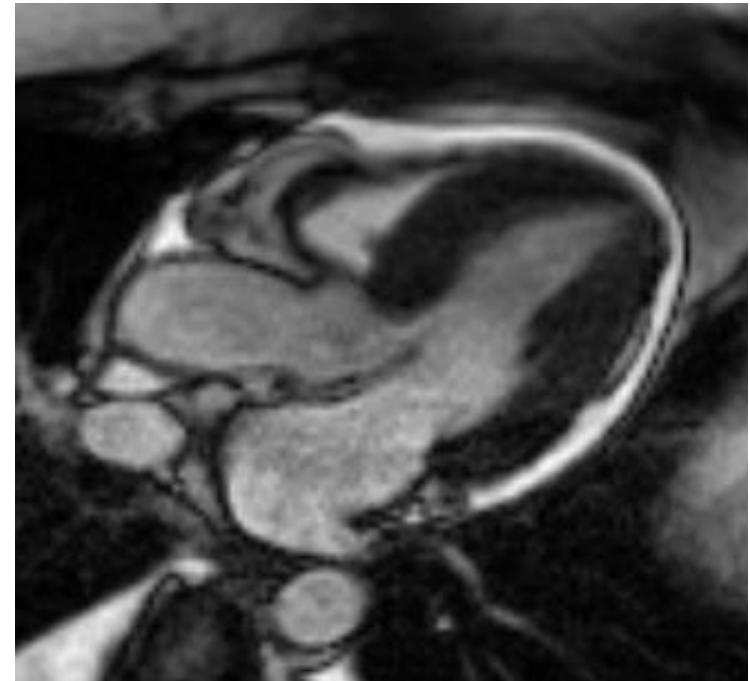
- Easily differentiates between pericardial effusions & pleural effusions & ascites
- Precisely delineates the size & distribution of fluid
- Enhanced sensitivity for loculated effusions
- Characterizes the fluid

Physiological Fluid Readily Distinguished from Thickened Pericardium

T1 Spin Echo

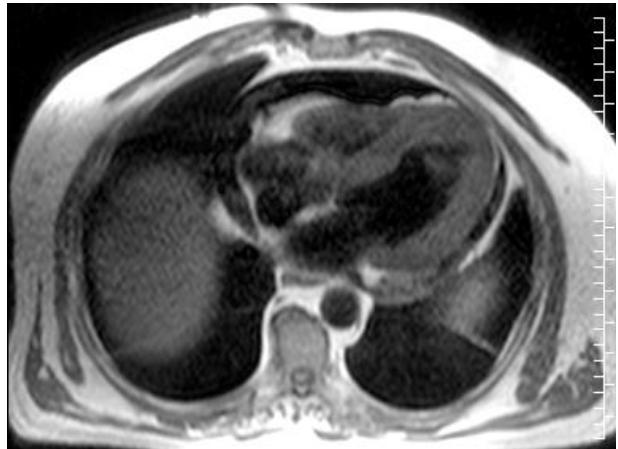


SSFP

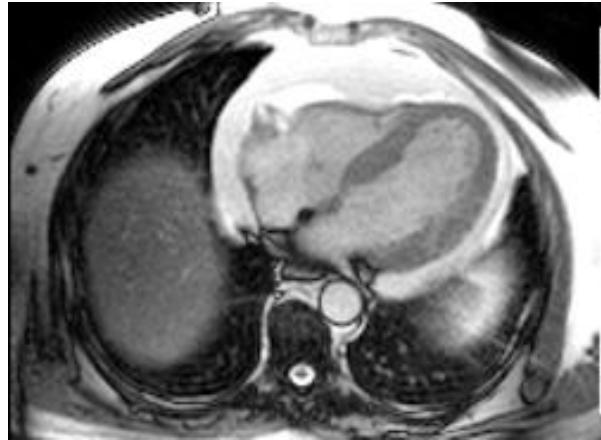


- Pericardium contains normally < 50 ml serous fluid
- Transudative fluid demonstrates high signal intensity on SSFP
- Easily distinguishes pericardial effusion from pericardial thickening

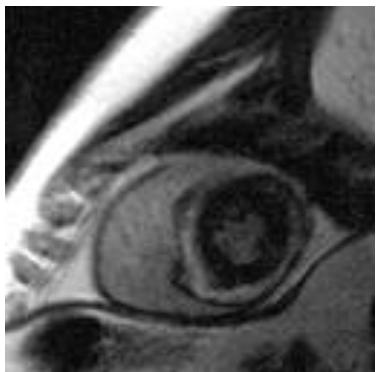
Pericardial Effusion Appearance



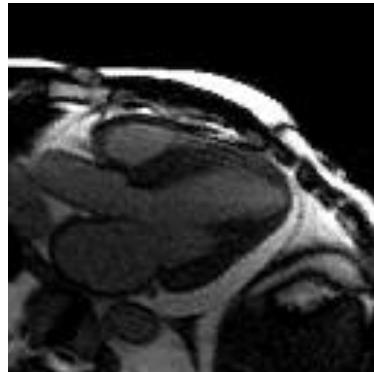
HASTE



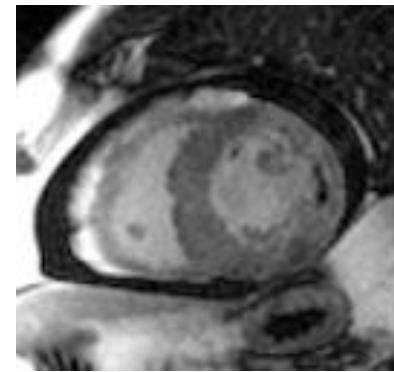
SSFP



IR
SEGMENTED



IR SINGLE
SHOT



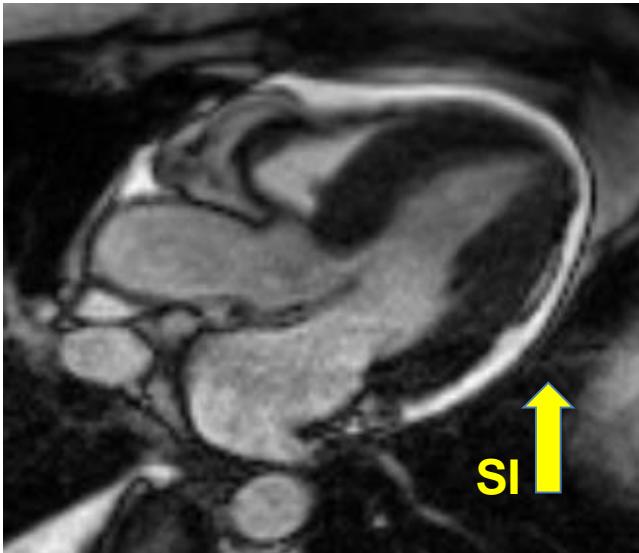
IR LONG TI

*** It is important to know what sequence is used and know what tissues like with each pulse sequence

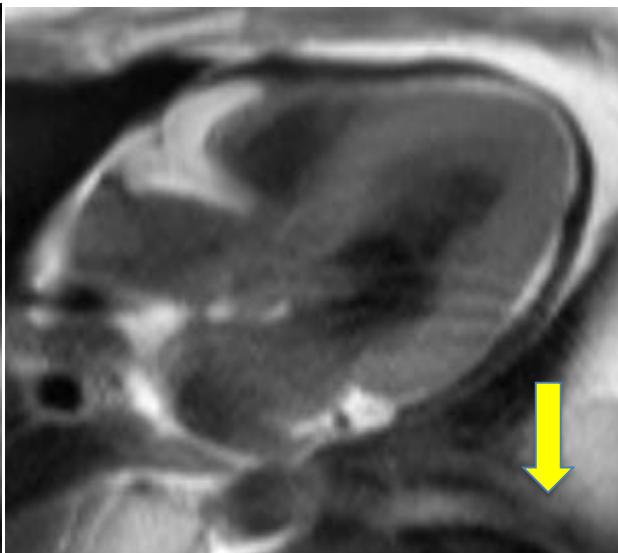
CMR: Composition / Characterization of Fluid

Transudate

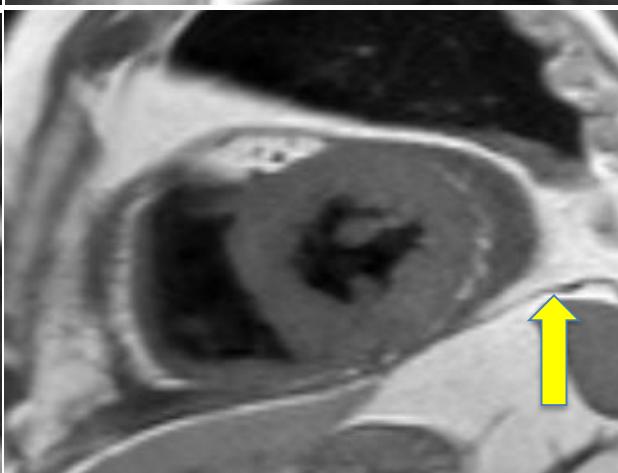
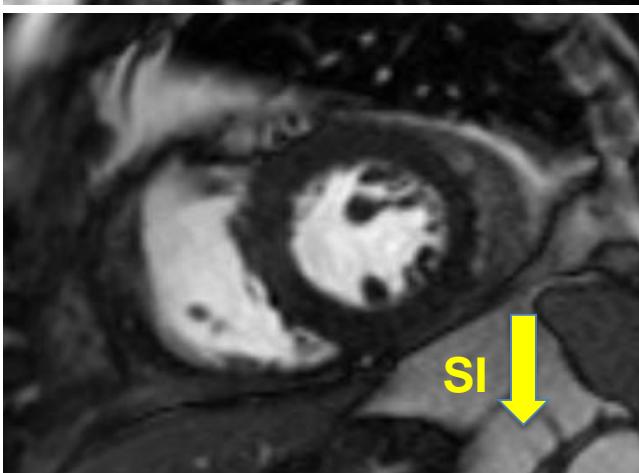
SSFP



T1 SE



Exudate



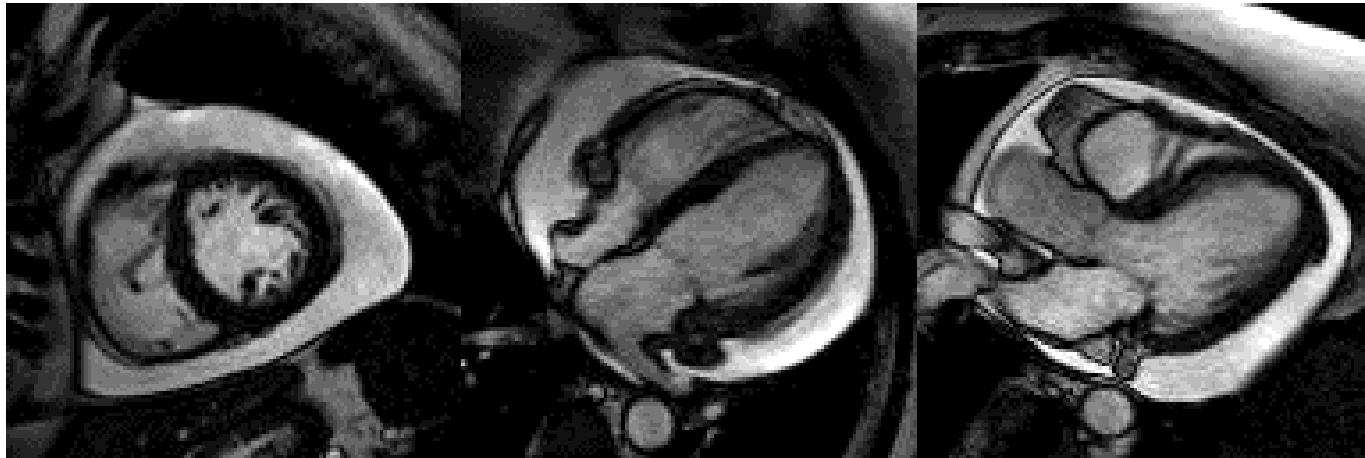
Cardiac Tamponade:

Physiology Depends on Etiology of Effusion
& Rate of Fluid Accumulation

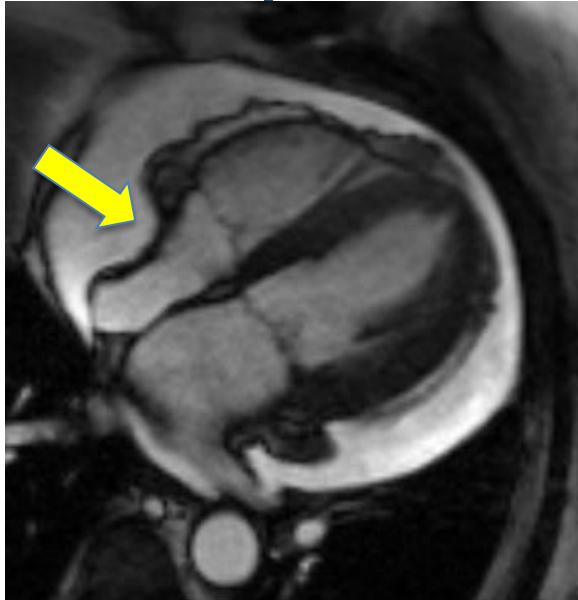


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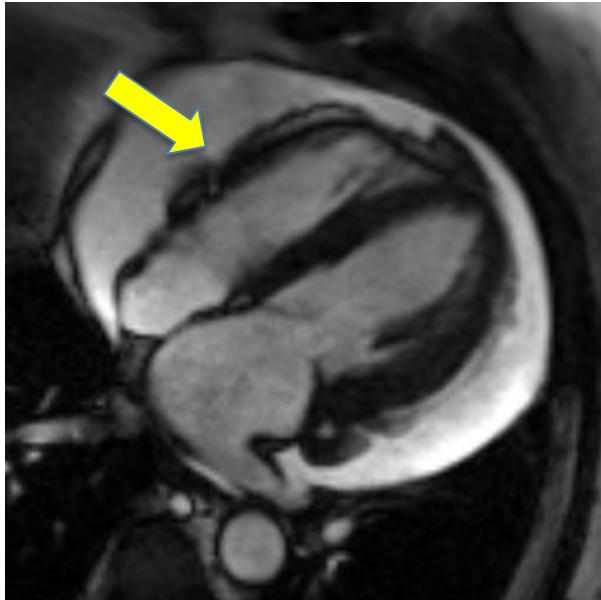
* Requires immediate communication to referring provider



Chamber Collapse:
RA Systole



Indirect signs:
Dilated IVC / hepatic veins



Pericardial Effusion: When to Consider Added Imaging

- Effusion in complex, loculated or clot present
- Localization and quantification are important
- Demonstrate other pathological conditions:
 - complex pleural effusions, lower lobe atelectasis, external masses abutting pericardium
- Differentiation of pericardial thickening from fluid
- Tissue characterization

Infectious & Inflammatory Pericardial Disease

48M with positional CP of recent onset.
PE was normal. ECG with early repolarization. Trop (-)
Ischemia work-up negative

3C



SA



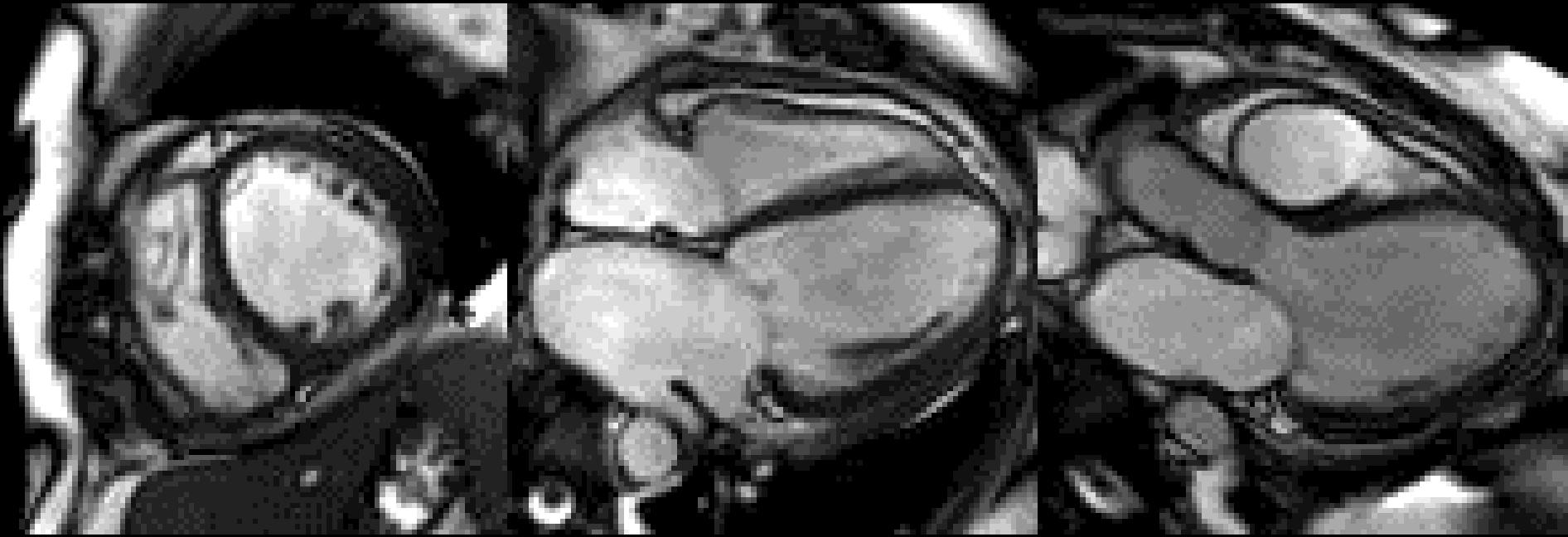
Echo 1st line but can not assess the pericardium !

Infectious & Inflammatory Pericardial Disease

Case 1

HOUSTON
Methodist[®]
DEBAKEY HEART &
VASCULAR CENTER

48M with positional CP of recent onset.
PE was normal. ECG with early repolarization. Trop (-)
Ischemia work-up negative



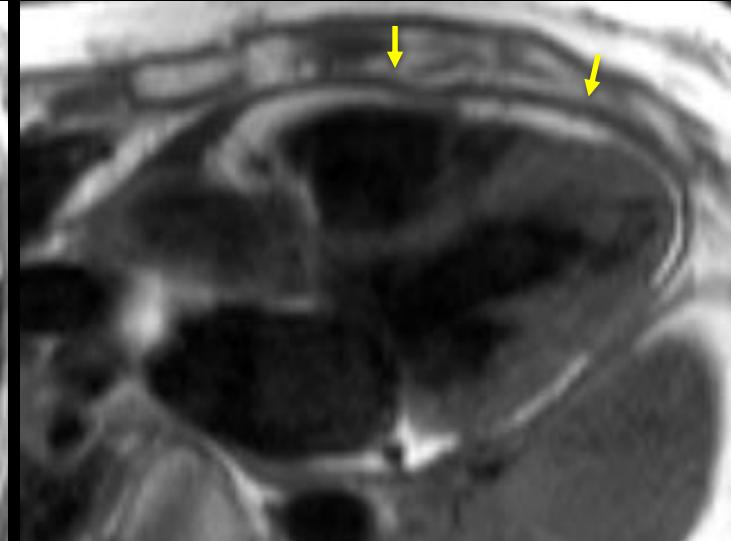
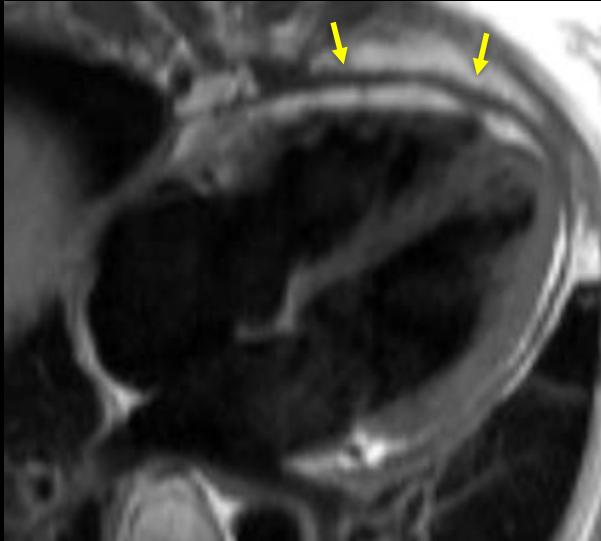
Supportive signs:
Increased thickness of the pericardium and pericardial effusion

CMR: Spin Echo

Case 1

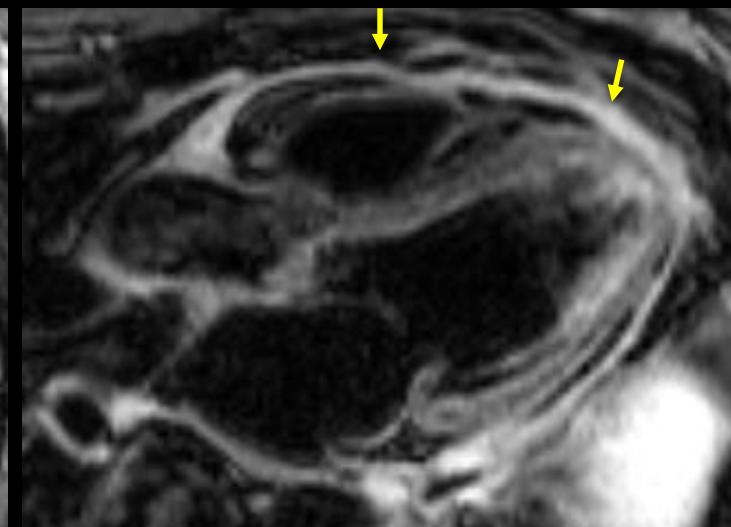
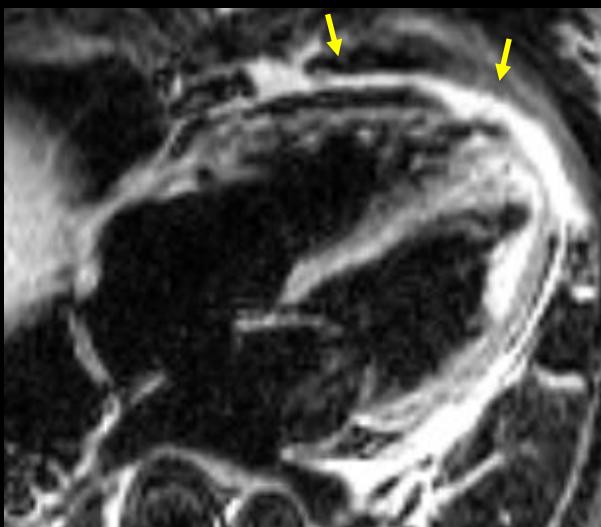
T1

(thickness)



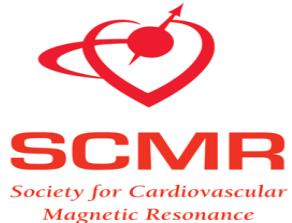
T2

(edema)



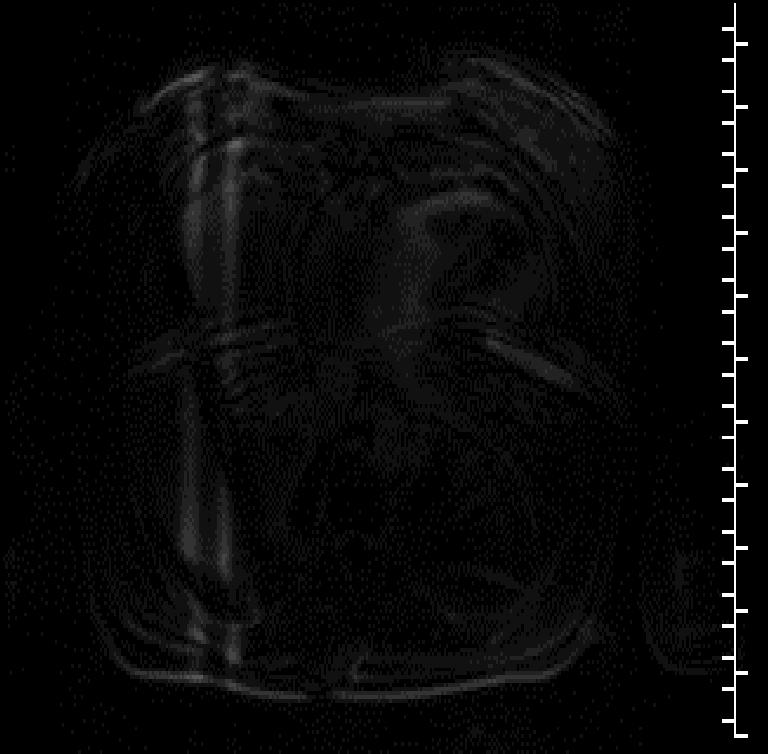
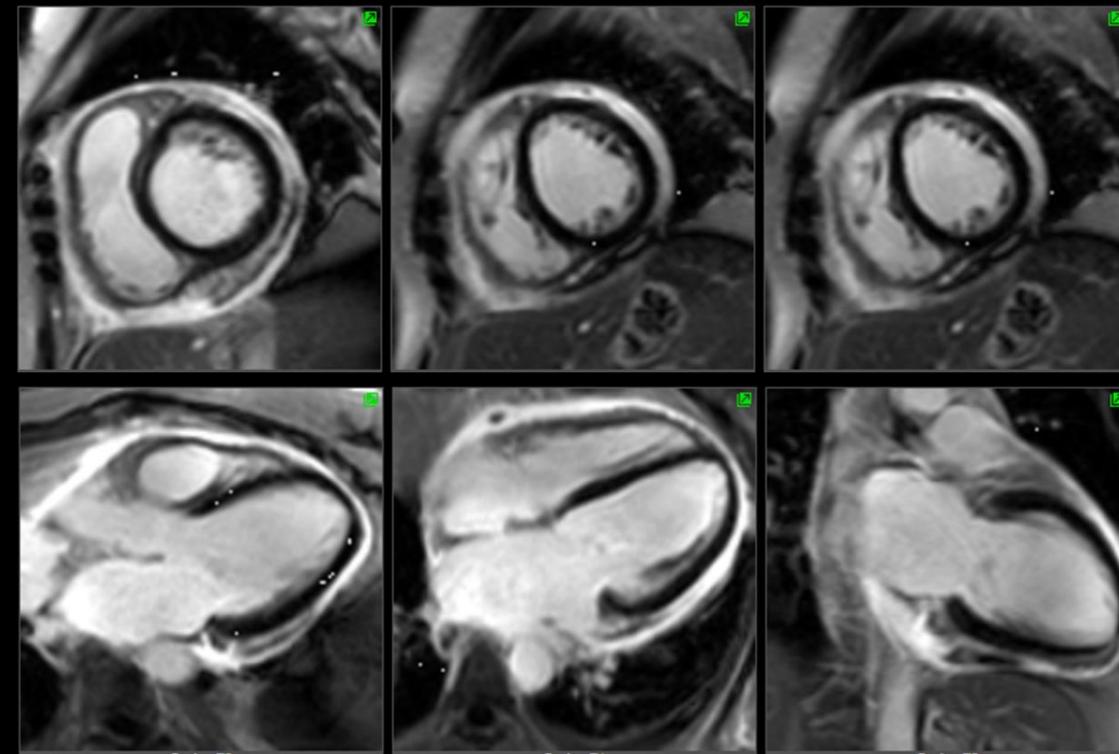
CMR: Post Gad Sequences

Case 1



LGE

Navigator gated 3D
inversion recovery



Acute Pericarditis:
Inflamed pericardium is enhanced by GBCA

Value of CMR in Pericarditis

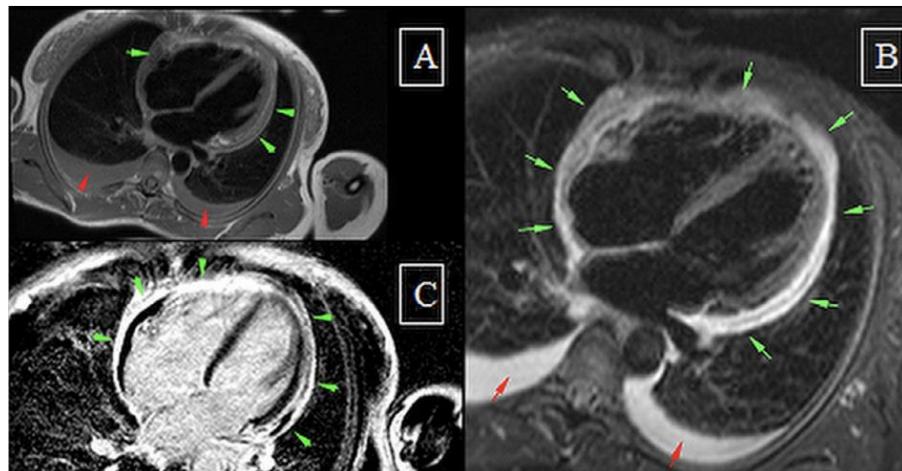
Pericarditis	Definition and diagnostic criteria
Acute	<p>Inflammatory pericardial syndrome to be diagnosed with at least 2 of the 4 following criteria:</p> <ul style="list-style-type: none">(1) pericarditic chest pain(2) pericardial rubs(3) new widespread ST-elevation or PR depression on ECG(4) pericardial effusion (new or worsening) <p>Additional supporting findings:</p> <ul style="list-style-type: none">- Elevation of markers of inflammation (i.e. C-reactive protein, erythrocyte sedimentation rate, and white blood cell count);- Evidence of pericardial inflammation by an imaging technique (CT, CMR).

CMR Findings Supporting Pericarditis

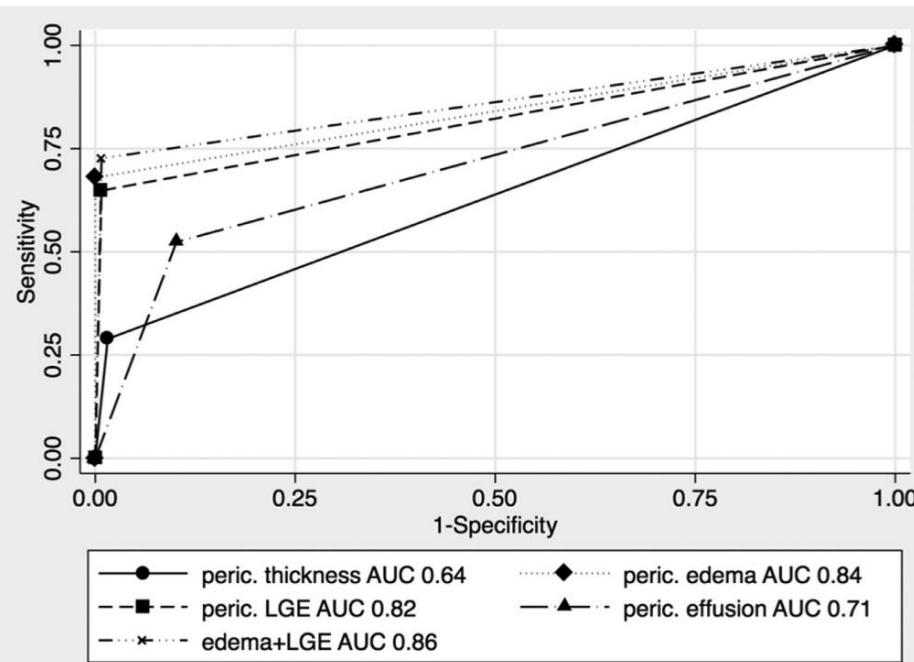


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- 1) Pericardial thickening,
- 2) Pericardial edema by STIR-T2w imaging,
- 3) Pericardial LGE
- 4) Pericardial effusion



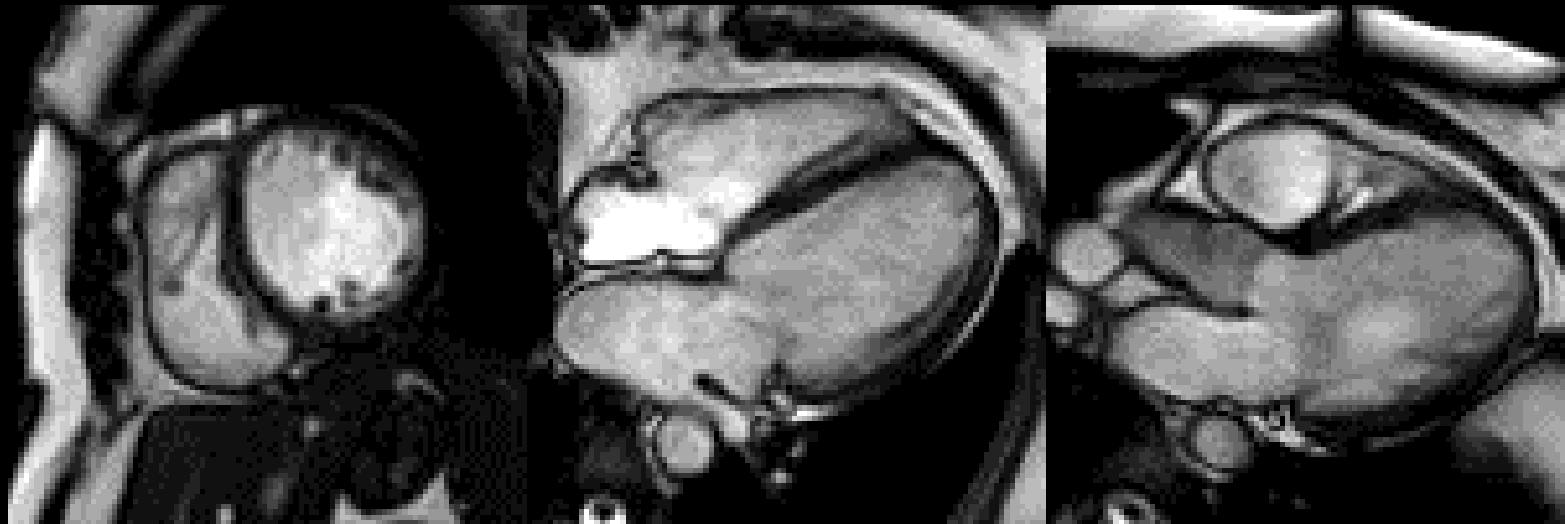
128 with idiopathic recurrent pericarditis



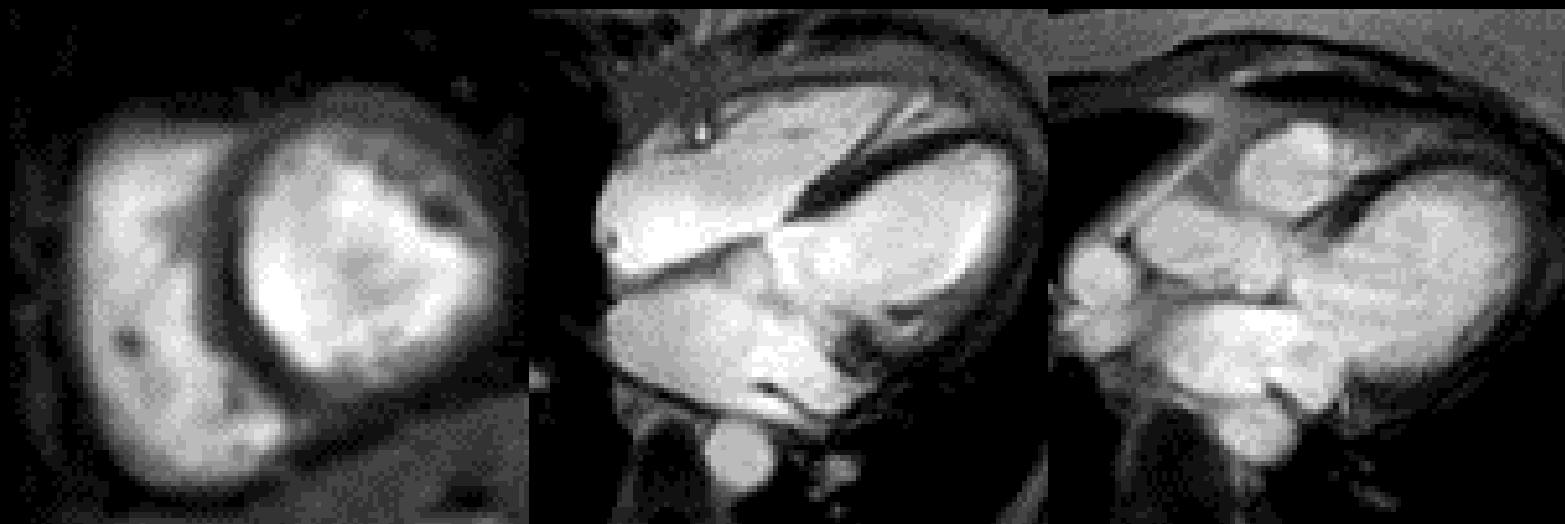
Imaging pericardial inflammation (thickness and LGE) may be a more reliable marker for diagnosis

Post Anti-Inflammatories

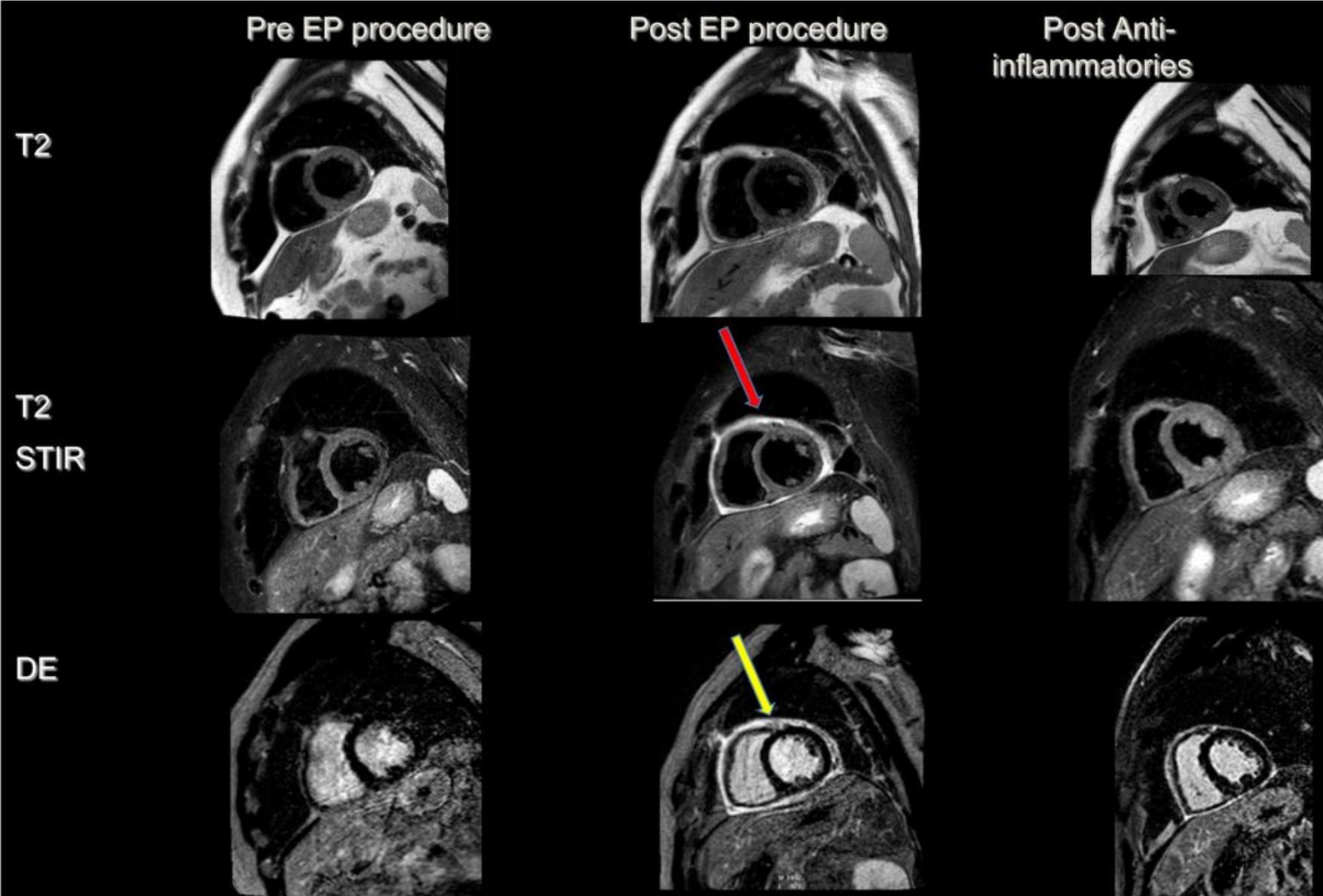
SSFP



LGE



Serial CMR Exams to Follow Pericardial Inflammation to Guide Therapy



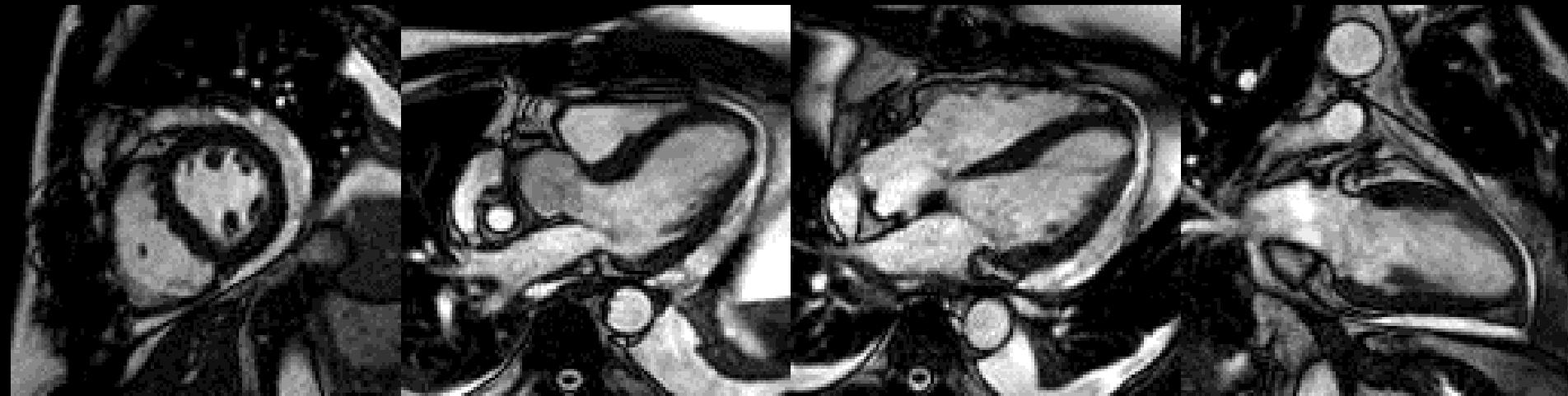


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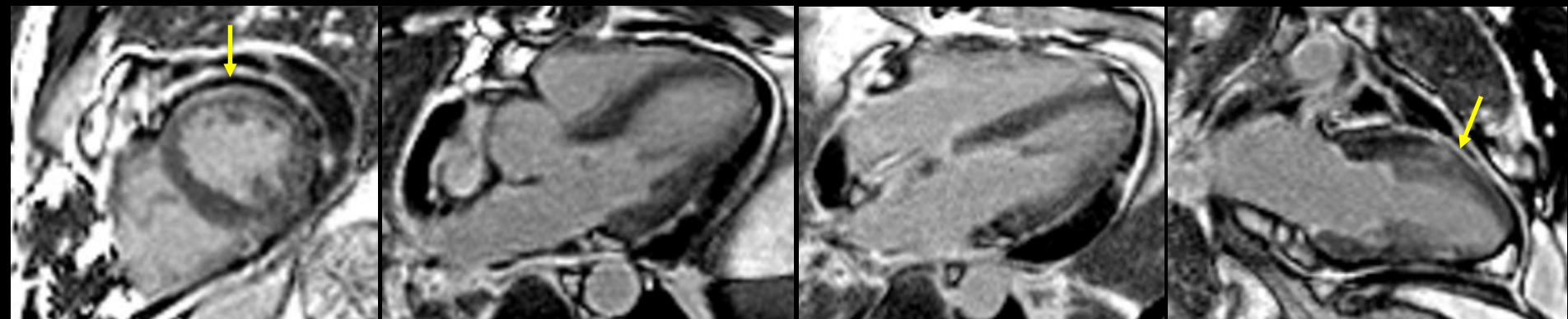
Myo-pericarditis

34M hx remote ASD closure presents with acute CP after viral prodrome.
Troponin (+). CTA normal.

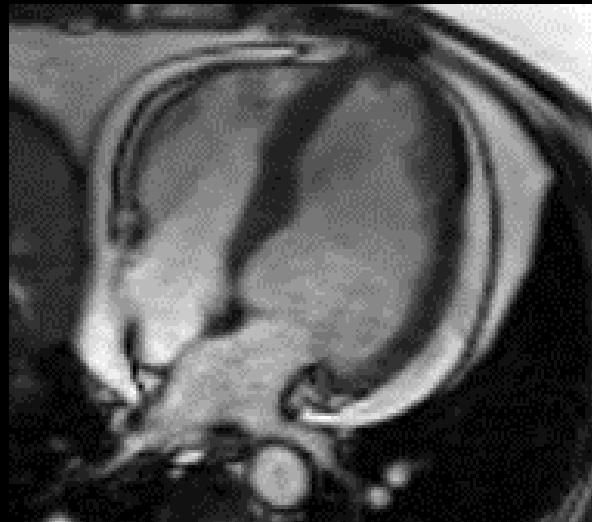
Cine SSFP: Pericardial effusion



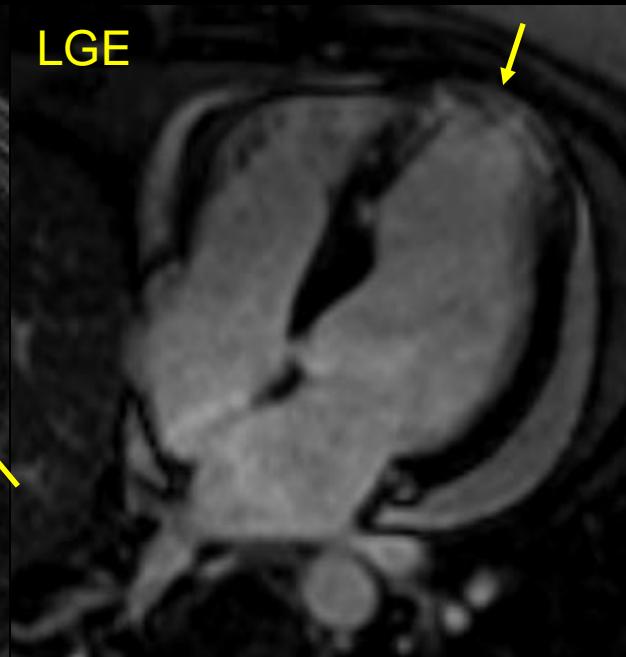
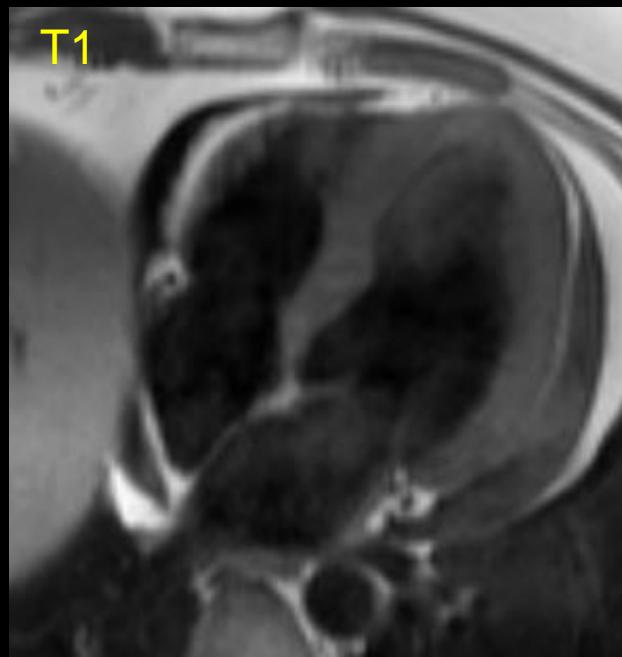
LGE PSIR: Pericardial uptake & epicardial scarring



Post-Infarct Pericarditis



55M 1 week s/p STEMI & PCA LAD
with positional CP. TTE new effusion



Acute Pericarditis: When to Consider Added Imaging

Clinical Features of Acute Pericarditis

- ◆ Inconclusive echo findings and ongoing clinical concern
- ◆ Failure to respond promptly to anti-inflammatory therapy
- ◆ Atypical clinical presentation
- ◆ Search for a specific cause (malignancy or TB)
- ◆ Suspicion of CP or effusive CP
- ◆ Associated trauma (penetrating injury, chest injury)
- ◆ Acute pericarditis in the setting of acute MI, neoplasm, lung or chest infection, or pancreatitis.

iodinated contrast

inflammation, detect small / loculated effusions or evaluate response to treatment

CT: if MRI is contra-indicated, or if suspicion for disease in adjacent anatomic structures

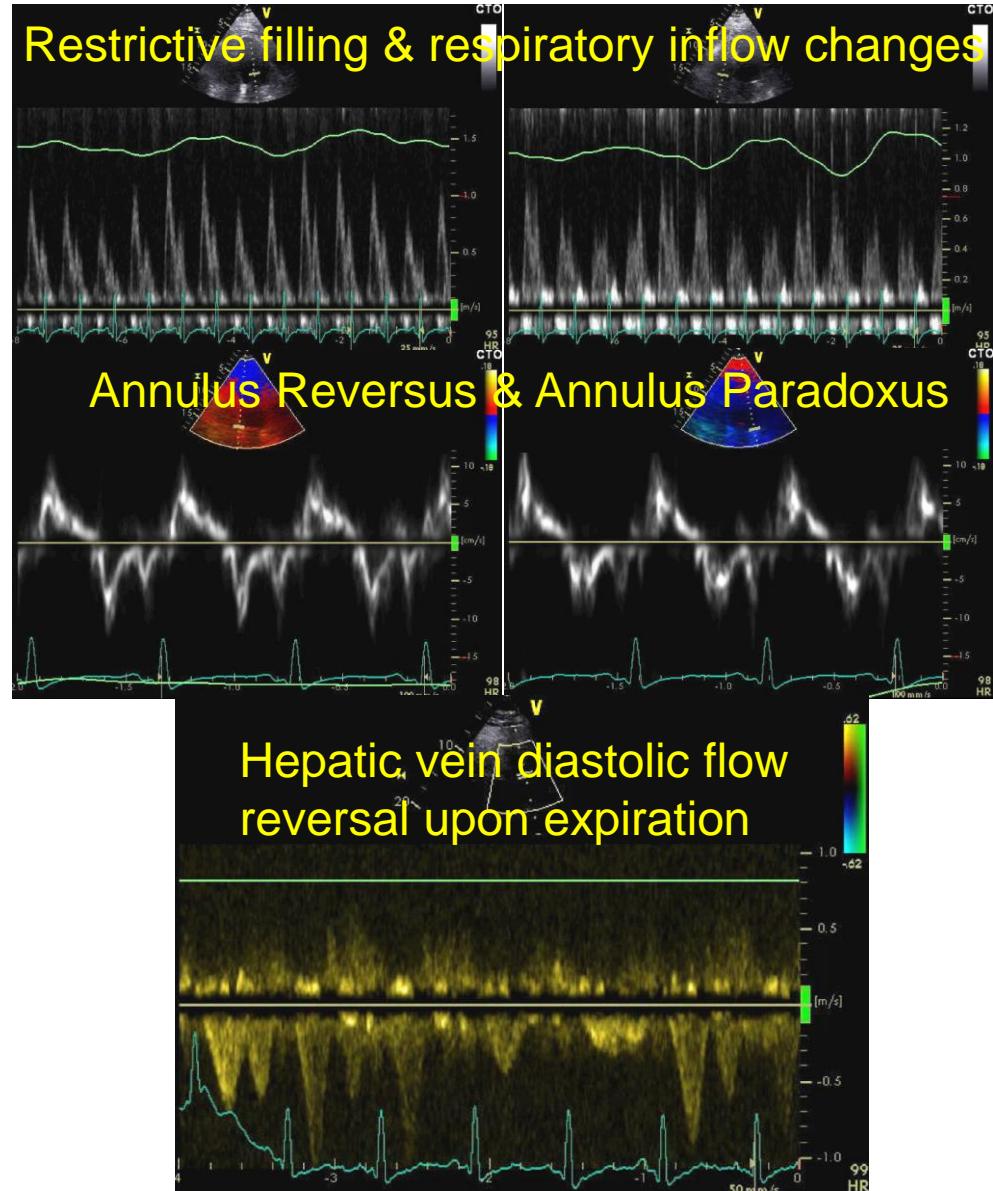
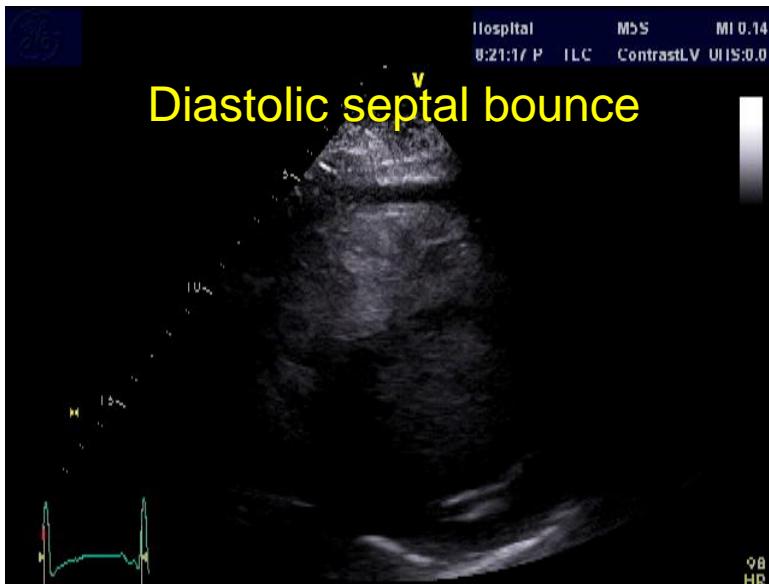
Thickening & Scarring of Pericardium

Case 2: Constrictive Pericarditis (CP)

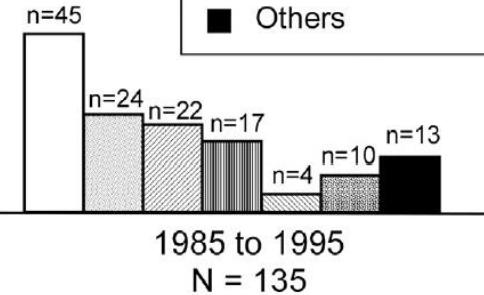
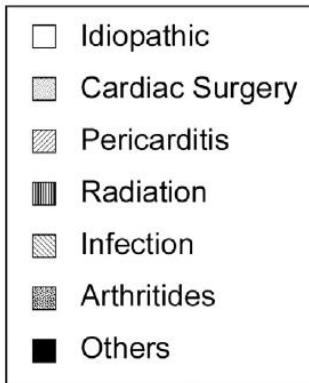


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- 47F PMHX of obesity, DM, HTN, AT3 deficiency, APLA positive, Factor V Leiden. Denies RA or SLE
- Presents to ED with 7 month history of progressive DOE, LE edema, fatigue and RUQ pain
- PE: +JVP (\uparrow with inspiration), \emptyset rub, +hepatomegally, 1-2+ LE edema
- Labs normal



Constrictive Pericarditis



- CP is a condition in which a thickened, scarred, inelastic, and often calcified noncompliant pericardium leads to:
 - Dissociation of intra-thoracic and intra- cardiac pressures
 - Ventricular interdependence/coupling
 - Elevation & equalization of cardiac filling pressures

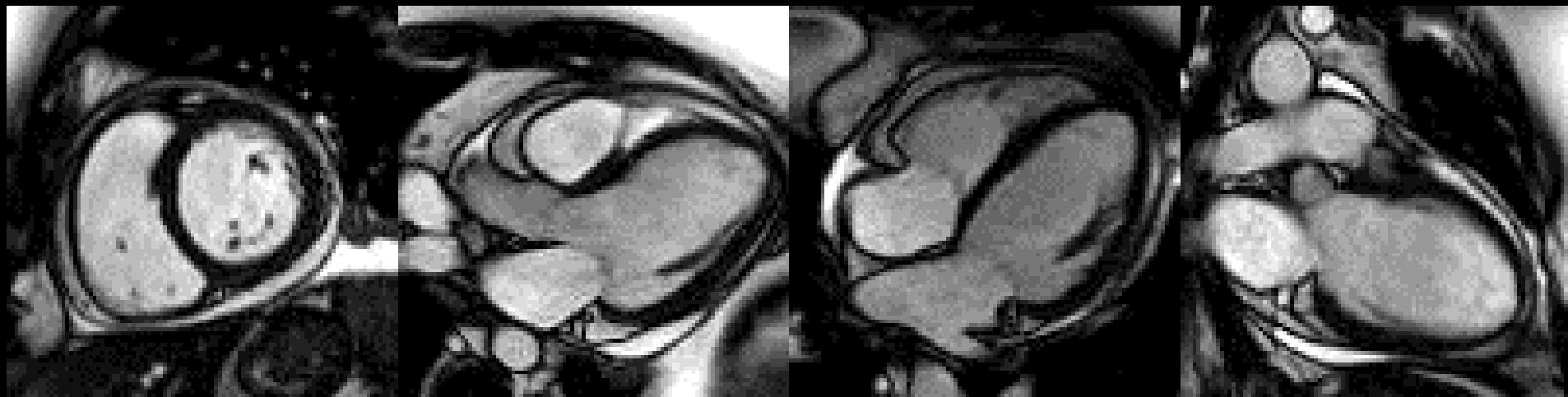
Table 8 Imaging findings of CP

Summary of echocardiographic findings

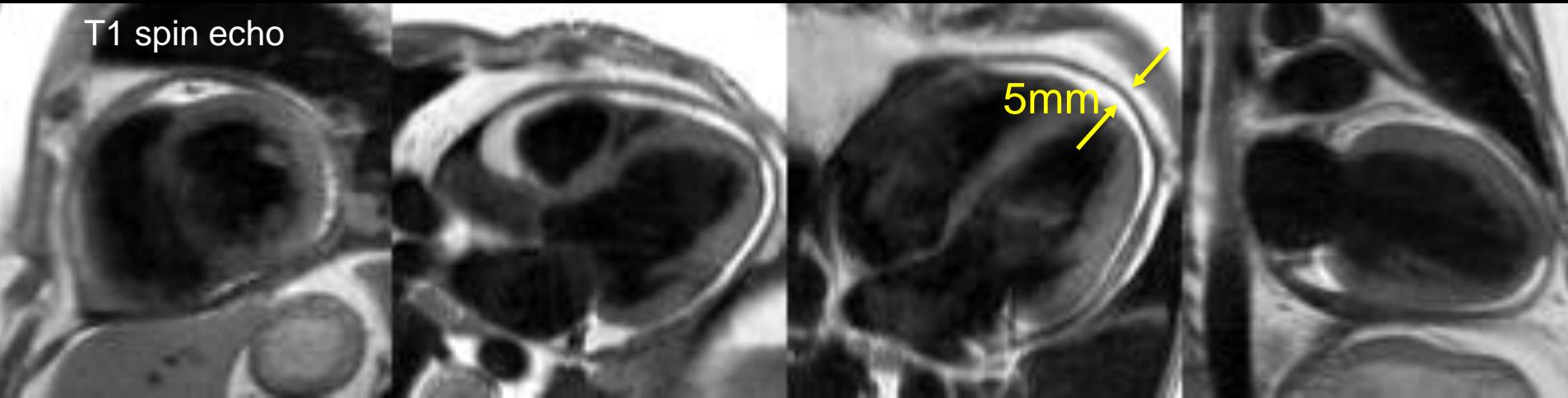
- M-mode: diastolic flattening of the LV posterior wall endocardium with little or absent respiratory movement, abrupt inspiratory posterior motion of the ventricular septum in early diastole with reciprocal changes in LV/RV dimensions throughout the respiratory cycle; premature opening of the pulmonary valve
- 2D: abrupt early diastolic LV and RV diastolic filling halt “diastolic checking”; inspiratory ventricular septal motion toward right ventricle (septal bounce); marked dilation and absent or diminished collapse of the IVC and hepatic veins
- Doppler: restrictive filling pattern of RV and LV diastolic filling; >25% fall in mitral inflow velocity and >40% increase in tricuspid velocity in the first beat after inspiration; opposite changes in expiration; low hepatic vein velocities; decreased expiratory diastolic hepatic vein velocities with large reversals
- Tissue Doppler: normal or increased mitral annular velocity (>7 cm/sec); annulus paradoxus; annulus reversus
- Color M-mode: normal or increased propagation velocity of early diastolic transmural flow

CMR Demonstrates Morphological Abnormalities

SSFP



T1 spin echo



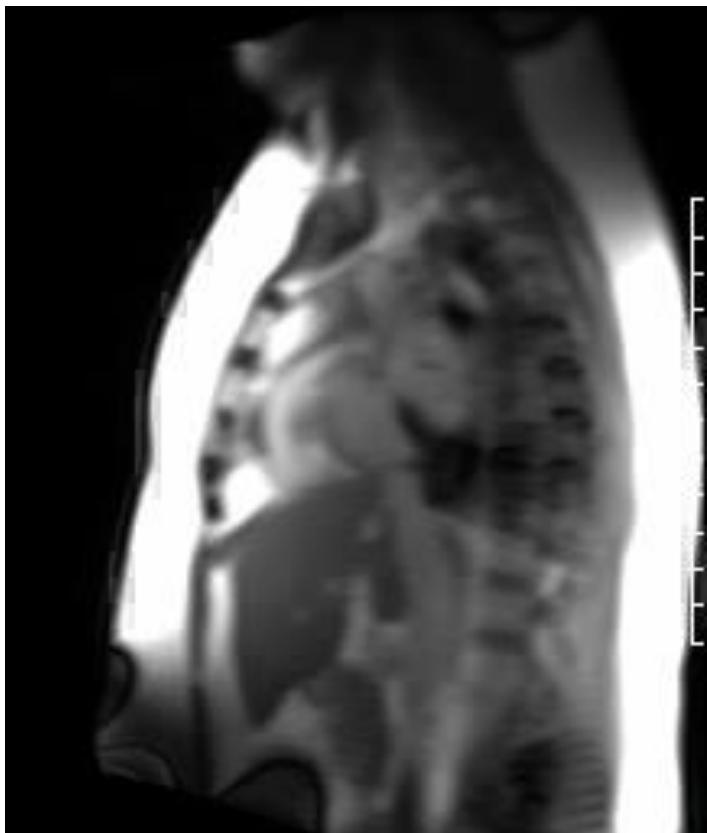
Thickened pericardium (>4 mm) in the proper clinical setting supports diagnosis of CP (>5–6 mm is highly specific)

Impaired RV/LV Diastolic Filling & Increased Venous Pressures

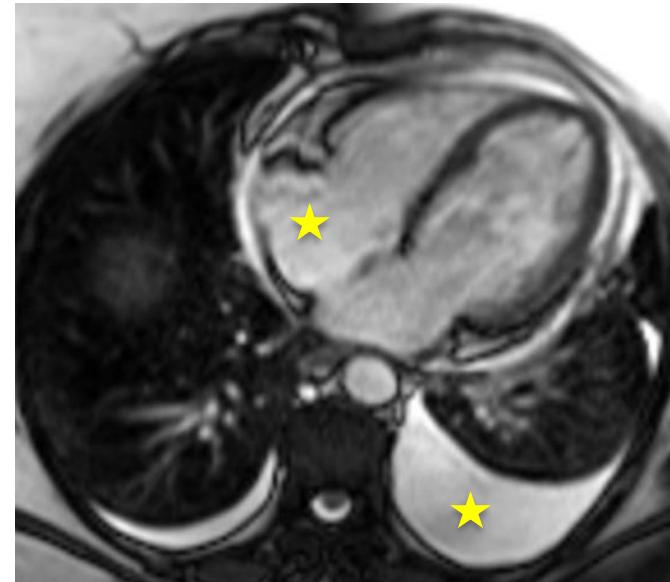


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IVC Plethora

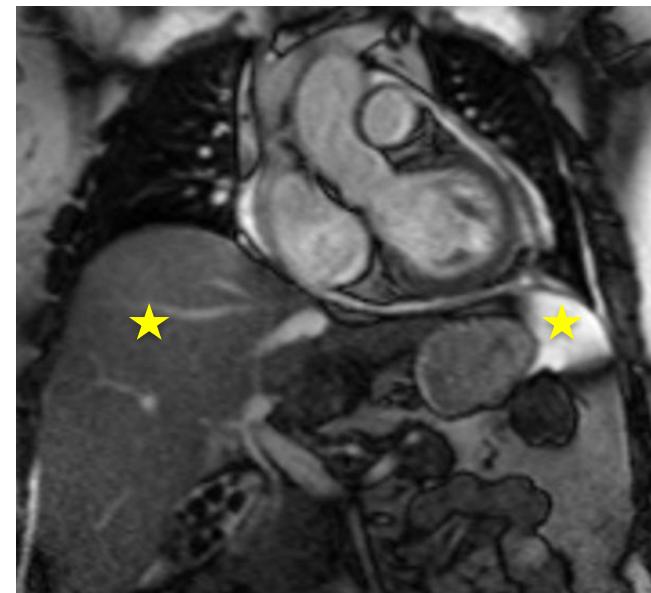


Enlarged RA



Pleural Effusion

Hepato-megally



Ascites

CMR Detects Functional & Hemodynamic Consequences of CP

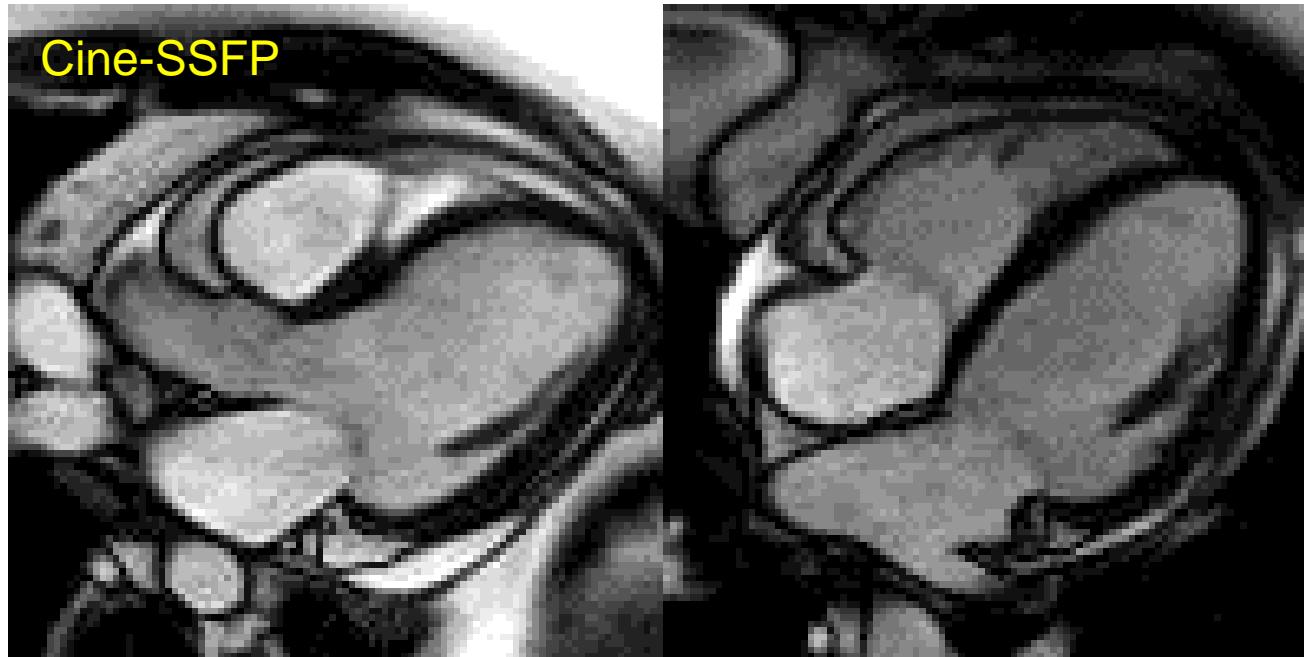


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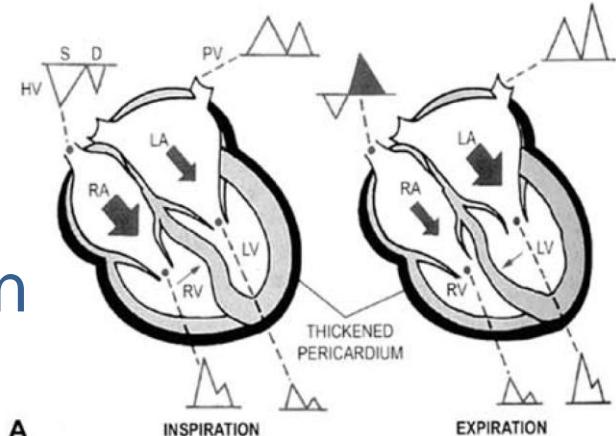
Increased ventricular coupling

Diastolic Septal Flattening / Inversion
“Septal Bounce / Shudder”

S-like
septal
motion



IVS bows
leftward
during
inspiration
in early
diastole

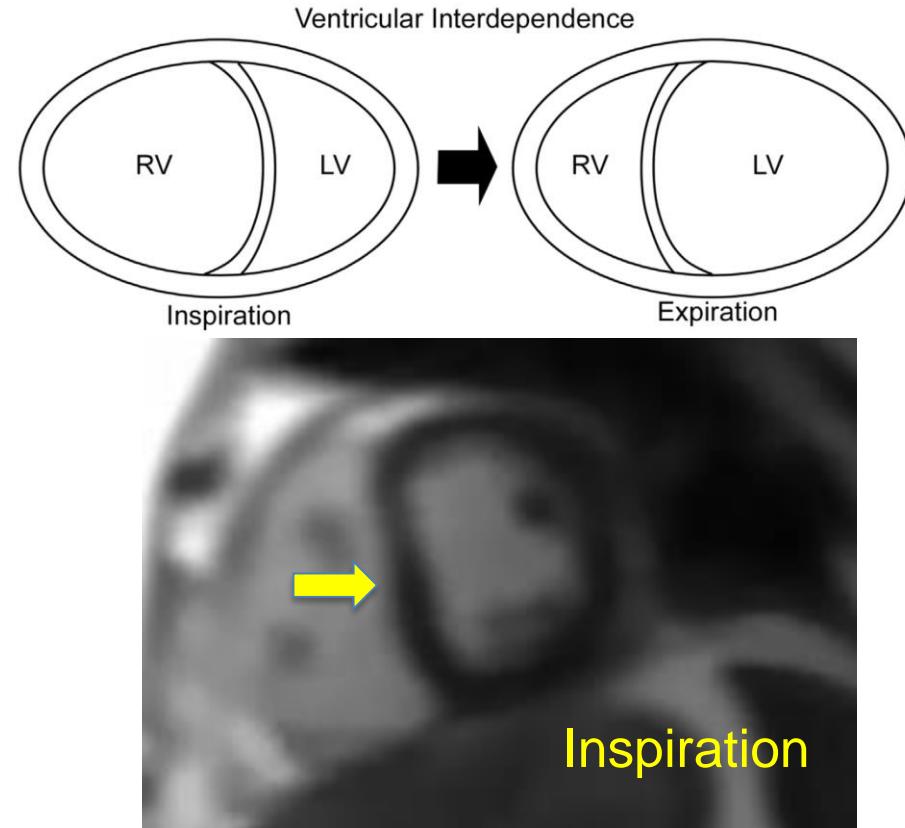
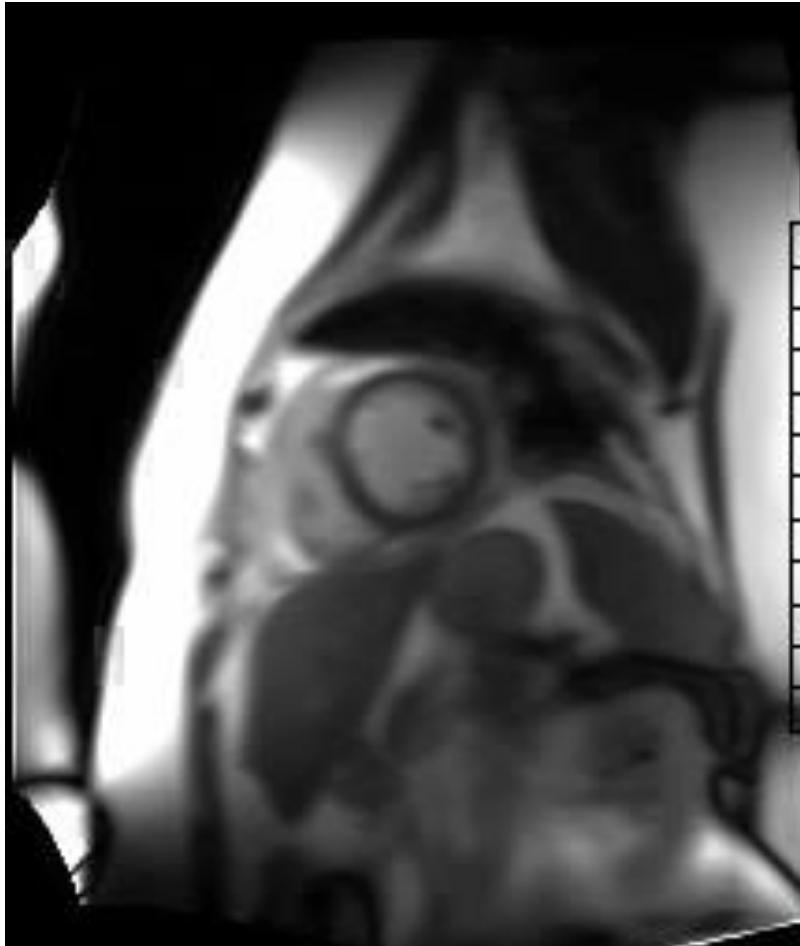




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Features of Constrictive Physiology: Respiratory Ventricular Interdependence

Real time Cine MR
during free breathing

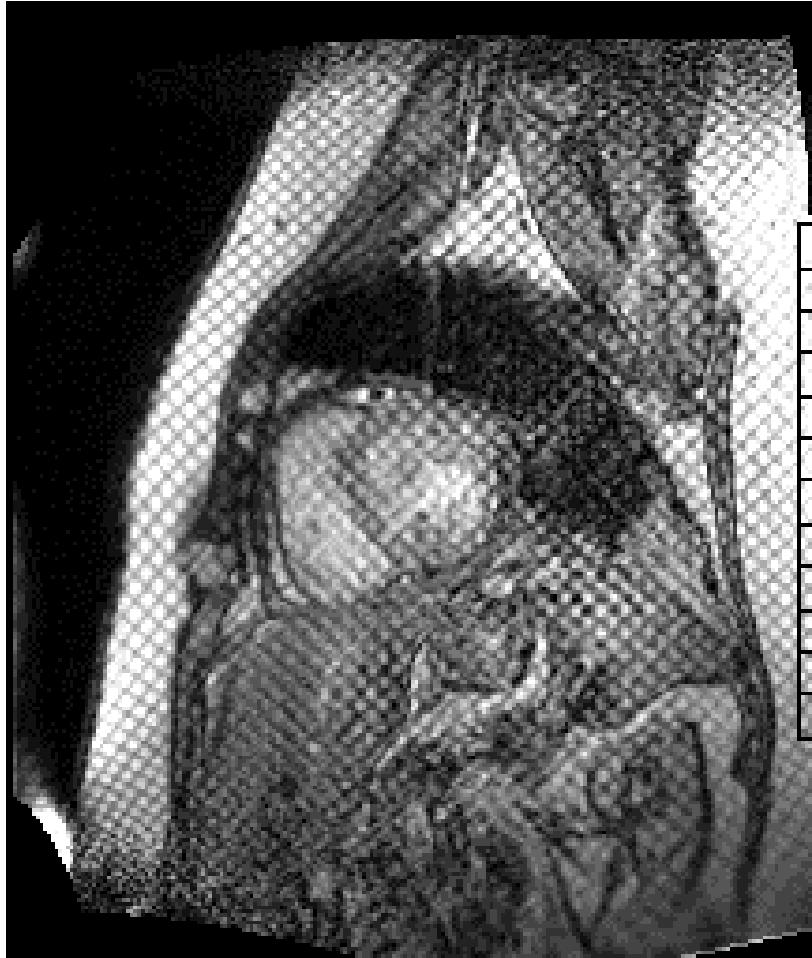
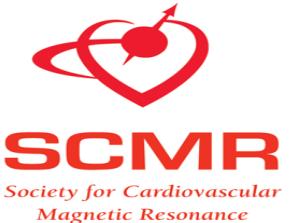


With ↑ systemic venous return to the right heart on inspiration, there is a shift of the ventricular septum toward the LV
(very specific finding)

CMR: Myocardial Tagging Sequences

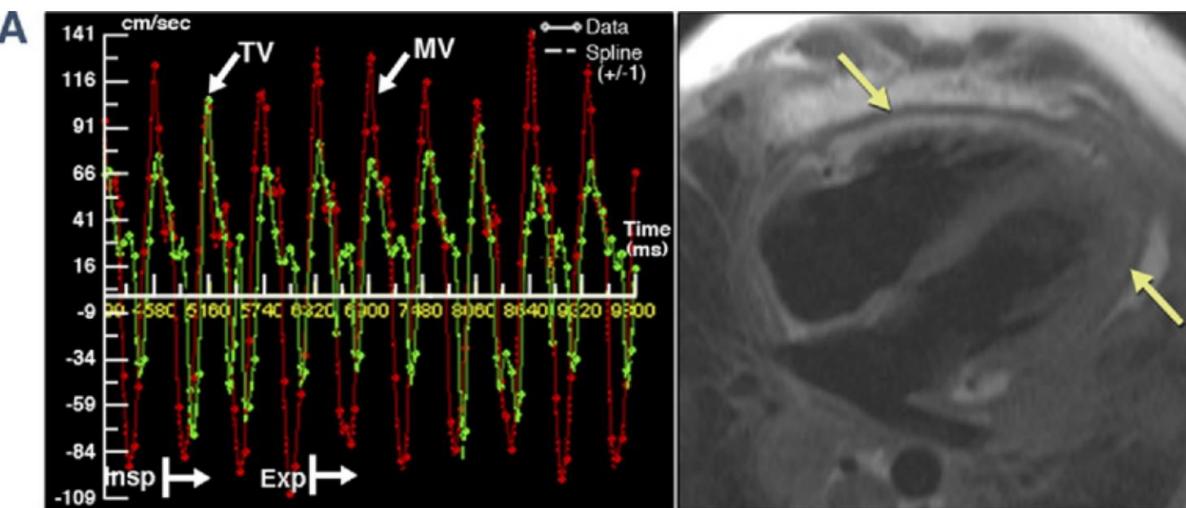
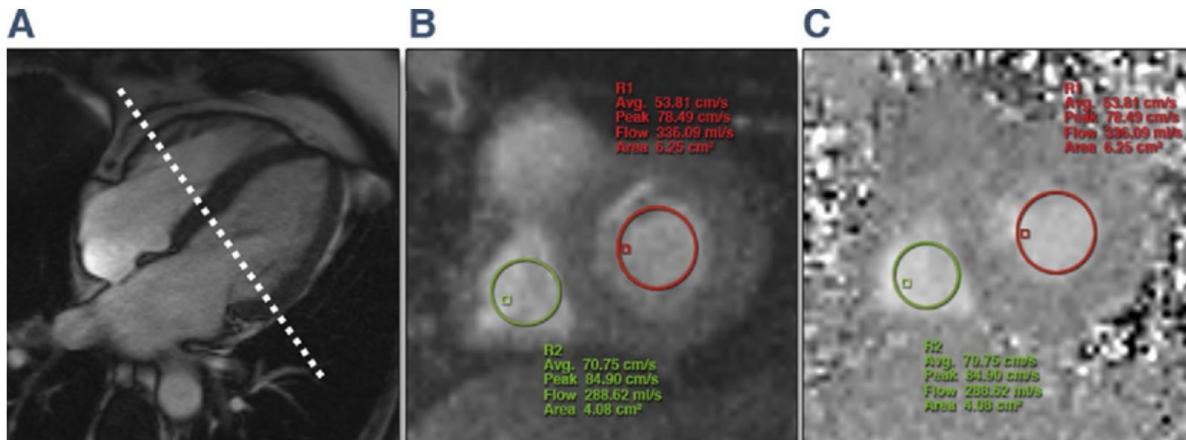
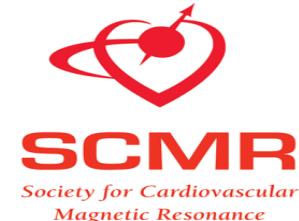
Demonstrate Pericardial-Myocardial Adherence

Case 2



Lack of grid deformation, indicates adherence & immobility of pericardial-myocardial interface

(RT-PC) Flow Measurement Reveal Accentuated & Discordant Respirophasic Changes in Mitral & Tricuspid Valve Inflow

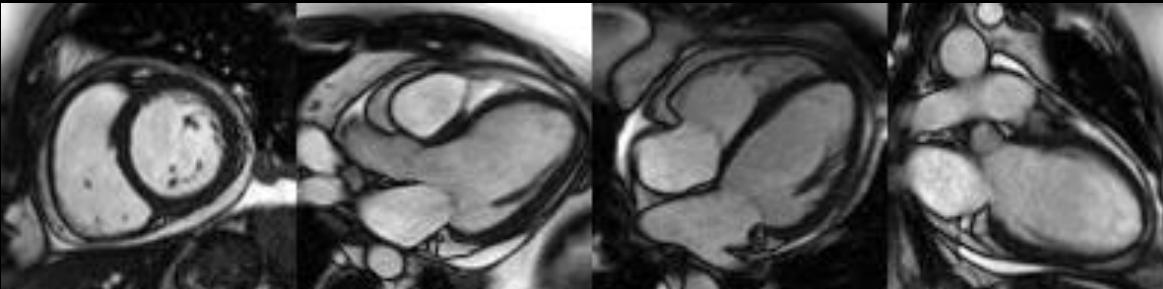


- Restrictive filling pattern
- Respiratory-related variation in cardiac filling
 - Enhanced RV filling on inspiration
 - Enhanced LV filling on expiration

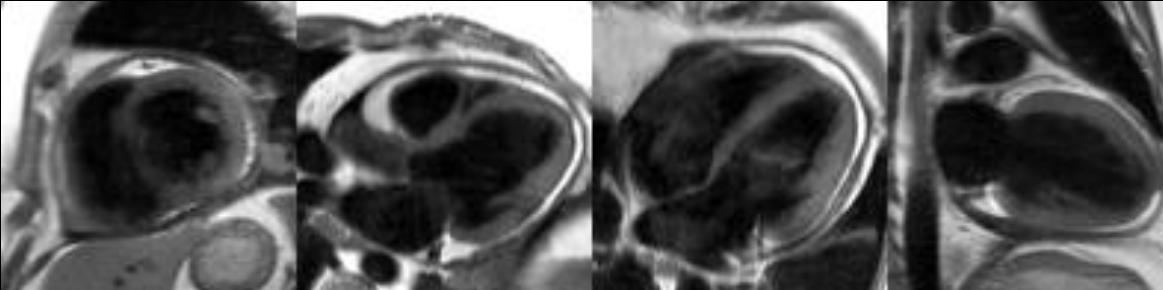
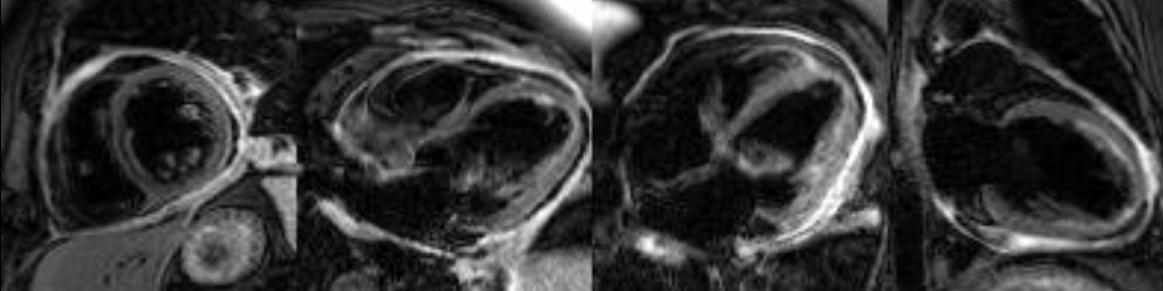
CMR Unique Ability:

Tissue Characterization

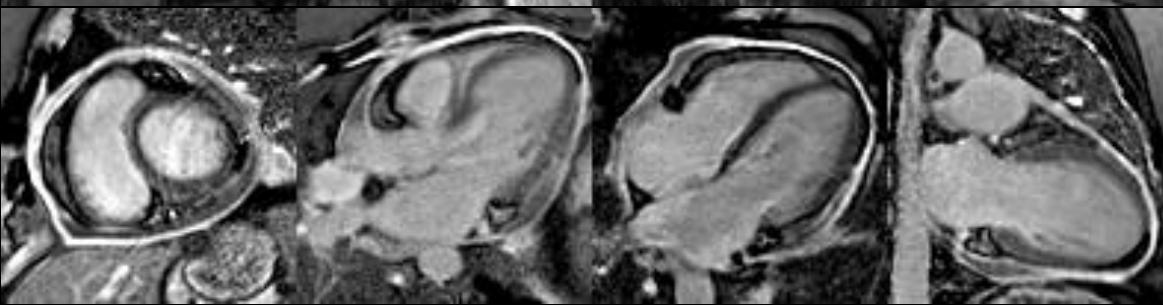
SSFP



T1

T2
“edema
imaging”

LGE



Surgical Findings Among CP Patients with Pericardial LGE



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Zurick. J. Am. Coll. Cardiol. Img. 2011;4;1180-1191

25 CP patients who underwent pericardectomy following CMR

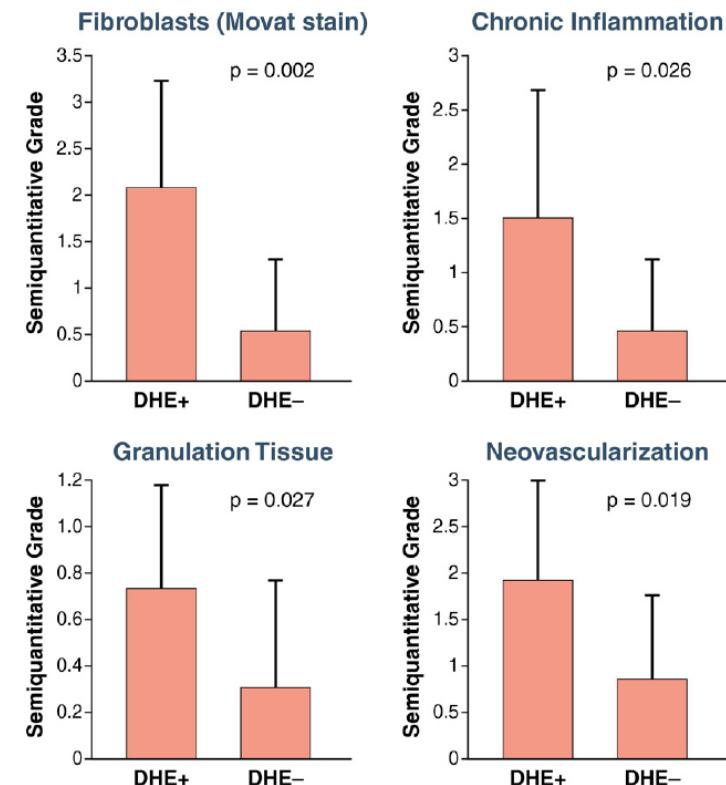
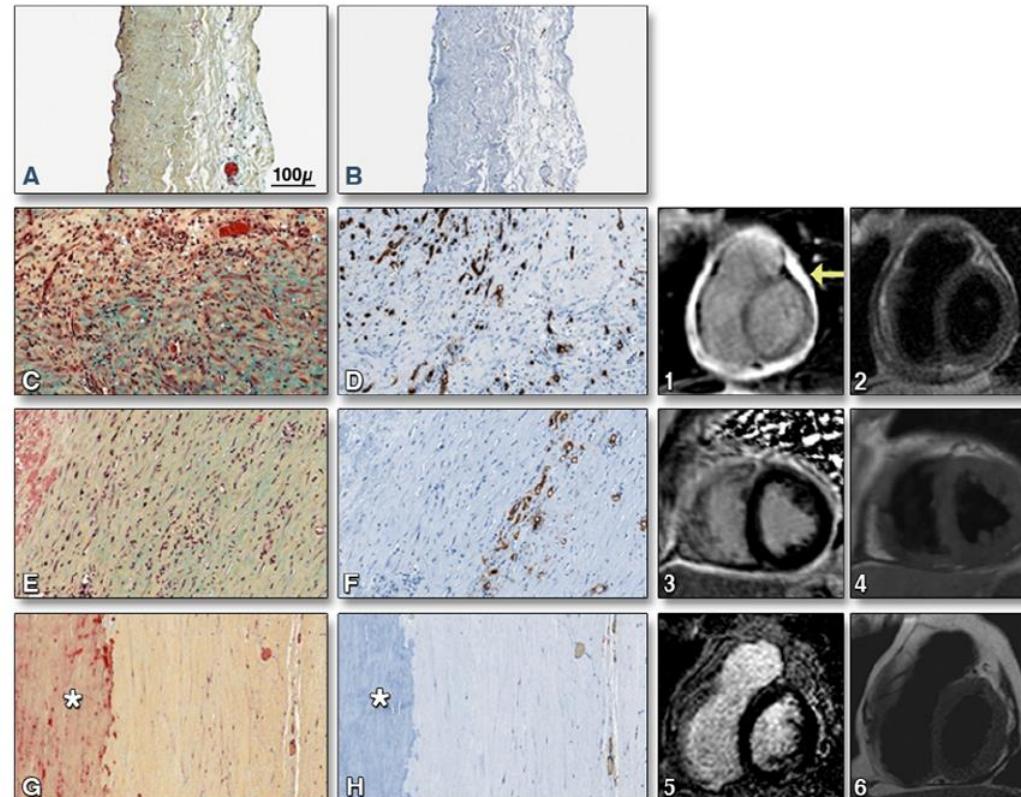
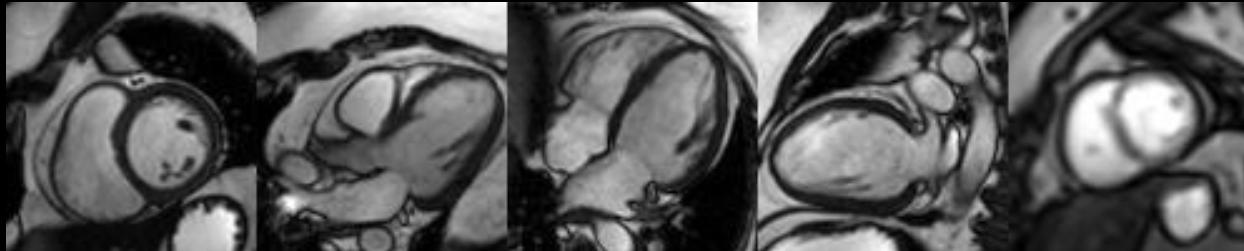


Figure 2. Surgical Pathology Semiquantitative Comparison Between DHE+ and DHE- Groups

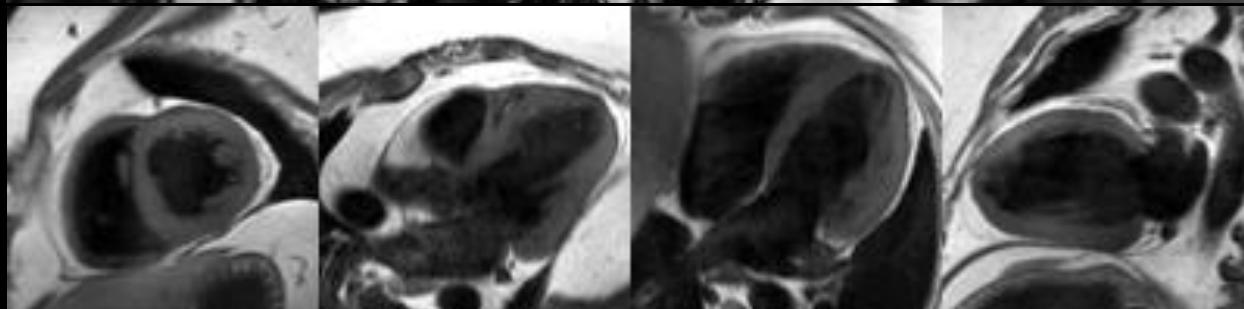
Pericardial LGE is correlated with ongoing, dynamic active *inflammatory* reaction

6 mo Post Inflammatory Therapy Tissue Characteristics

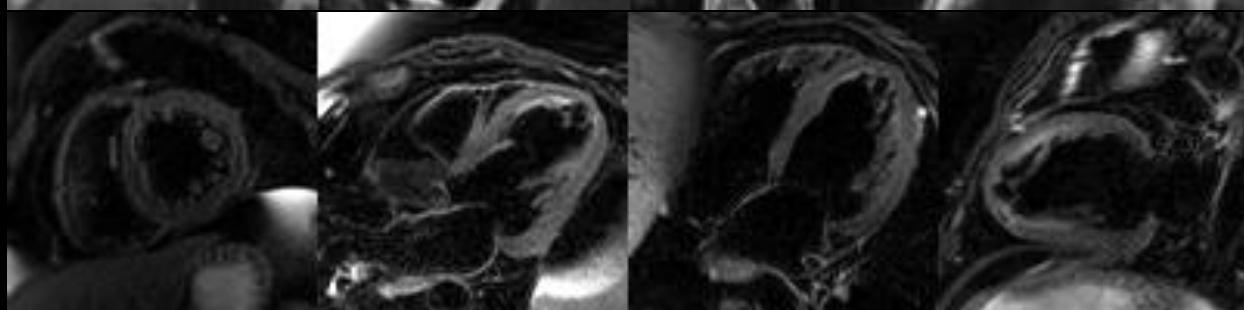
SSFP



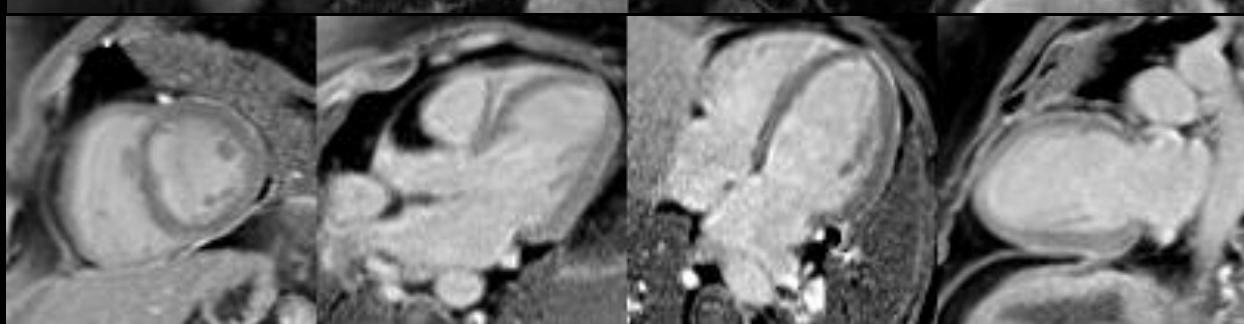
T1



T2

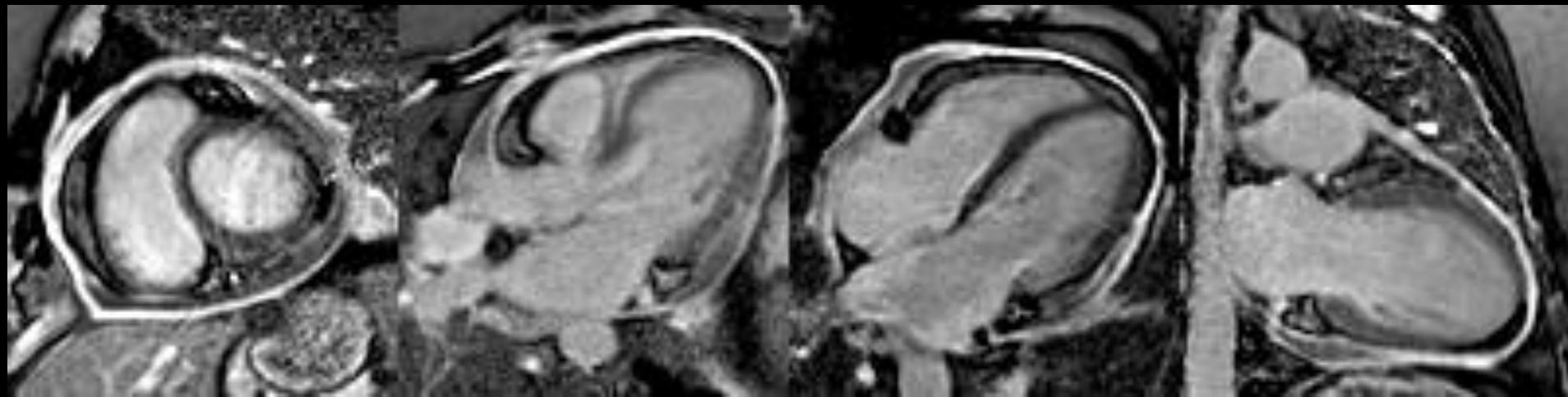


LGE

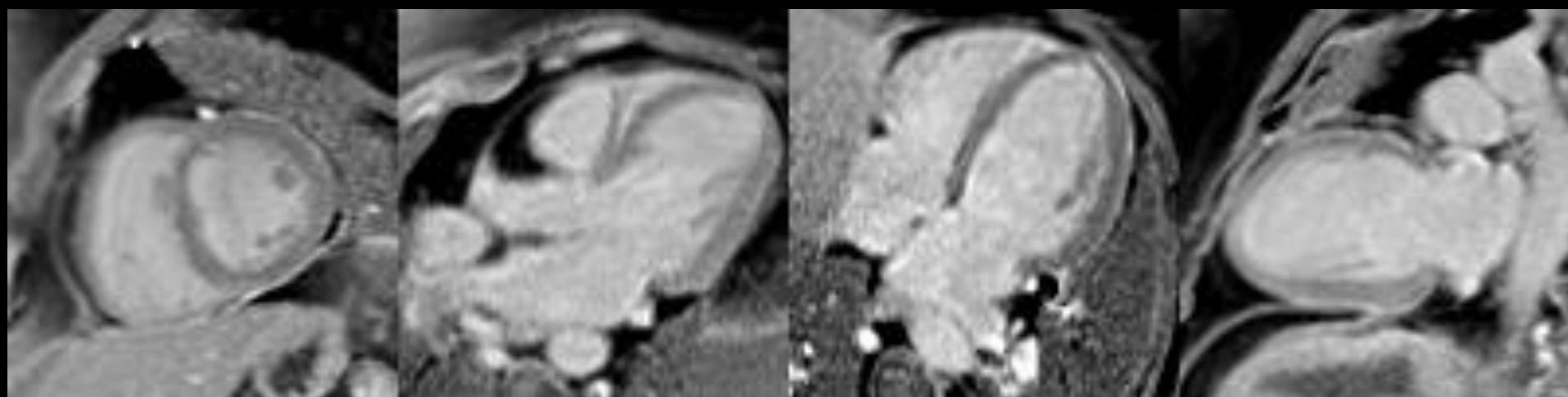


LGE Comparison

Pre-Therapy

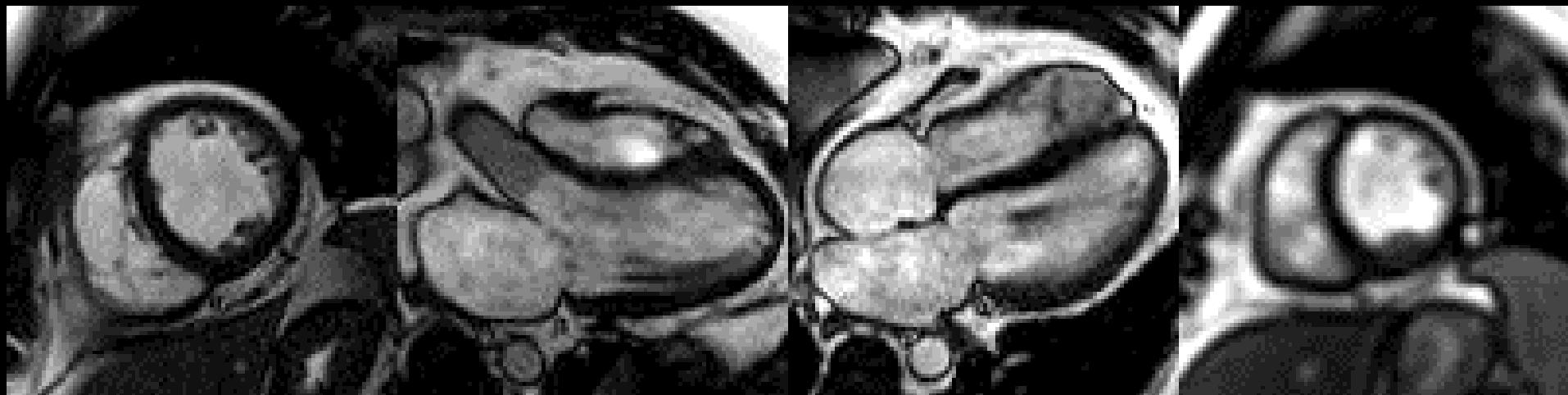


Post-Therapy

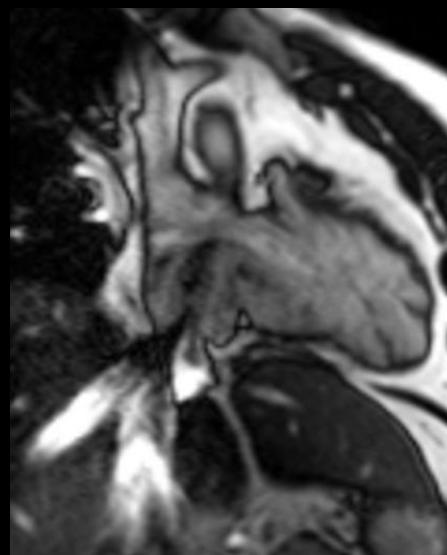


Case 3: Constrictive Pericarditis

62M with Afib, RUQ discomfort and LE edema of several years duration

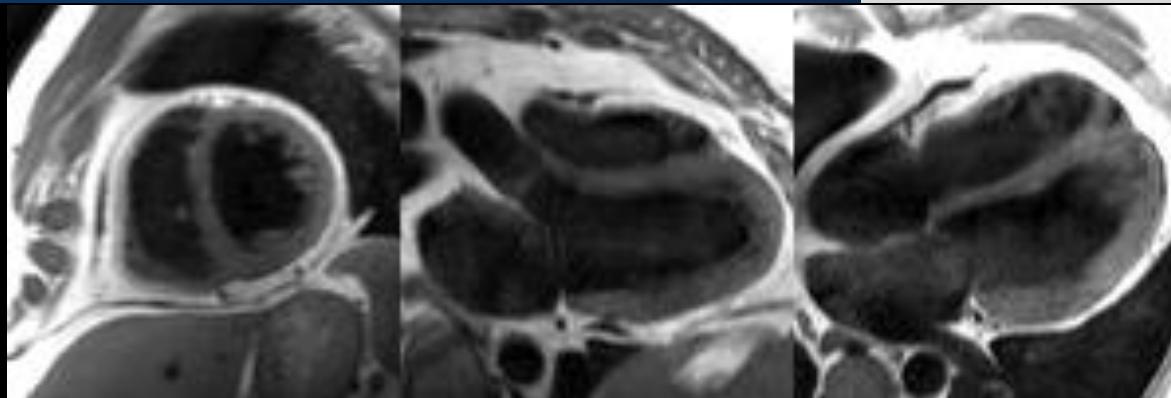


Morphological &
physiological findings
for constriction

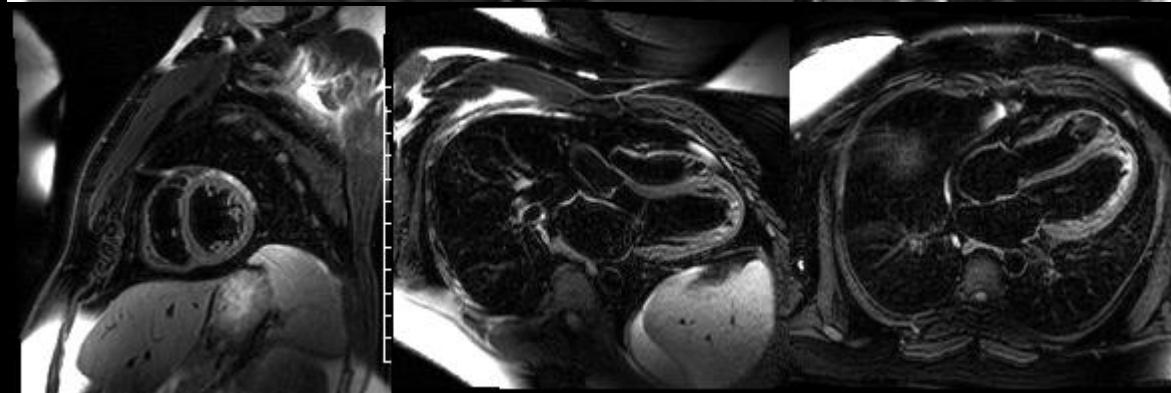


Case 3: Tissue Characteristics

T1



T2



LGE



Non-Contrast CT: End Stage



- Pericardial calcification
- Pericardial thickness of >4 mm (diffuse or localized),
- Narrowing and tubular deformation of the RV,
- Normal or small ventricular size,
- Straightening of the IVS.
- Signs of impaired diastolic filling of the RV:
 - dilatation of the IVC, hepatic veins, and RA,
 - hepatosplenomegaly,
 - ascites, and pleural effusions.

Pericardial calcification represents end stage of fibrosis and inflammation

CP is Reversible in Some Patients

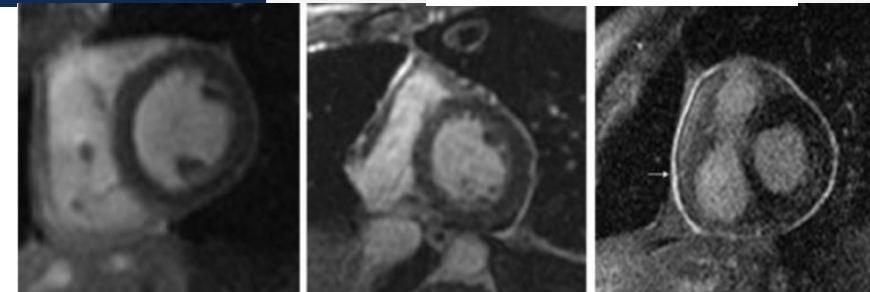
- LGE is Independently Valuable -



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Table 3. Baseline CMR Features in Reversible and Persistent Constriction

	Reversible CP (n=14)	Persistent CP (n=15)	P*
LVEF, %	56±3	56±3	0.94
RVEF, %	49±2	46±2	0.45
Abnormal septal motion, %	71	93	0.12
Septal flattening, %	71	73	0.91
RV and/or LV deformation, %	21	13	0.79
Focal compression, %	14	7	0.50
Myocardial LGE, %	7	7	1.0
IVC dilatation, %	50	73	0.20
Pericardial effusion, %	64	53	0.40
Complex pericardial effusion, %	29	20	0.79
Pleural effusion, %	64	60	0.81
Thick pericardium, %	71	73	1.0
Pericardial thickness by SSFP, mm	4±1	3±1	0.21
Pericardial thickness by FSE, mm	4±1	4±1	0.76
Pericardial LGE thickness, mm	4±1	2±1	0.001
No pericardial LGE, %	0	27 (n=4)	0.002
Mild pericardial LGE, %	7 (n=1)	40 (n=6)	
Moderate pericardial LGE, %	93% { 43 (n=6) }	33 (n=5)	
Severe pericardial LGE, %	50 (n=7)	0	



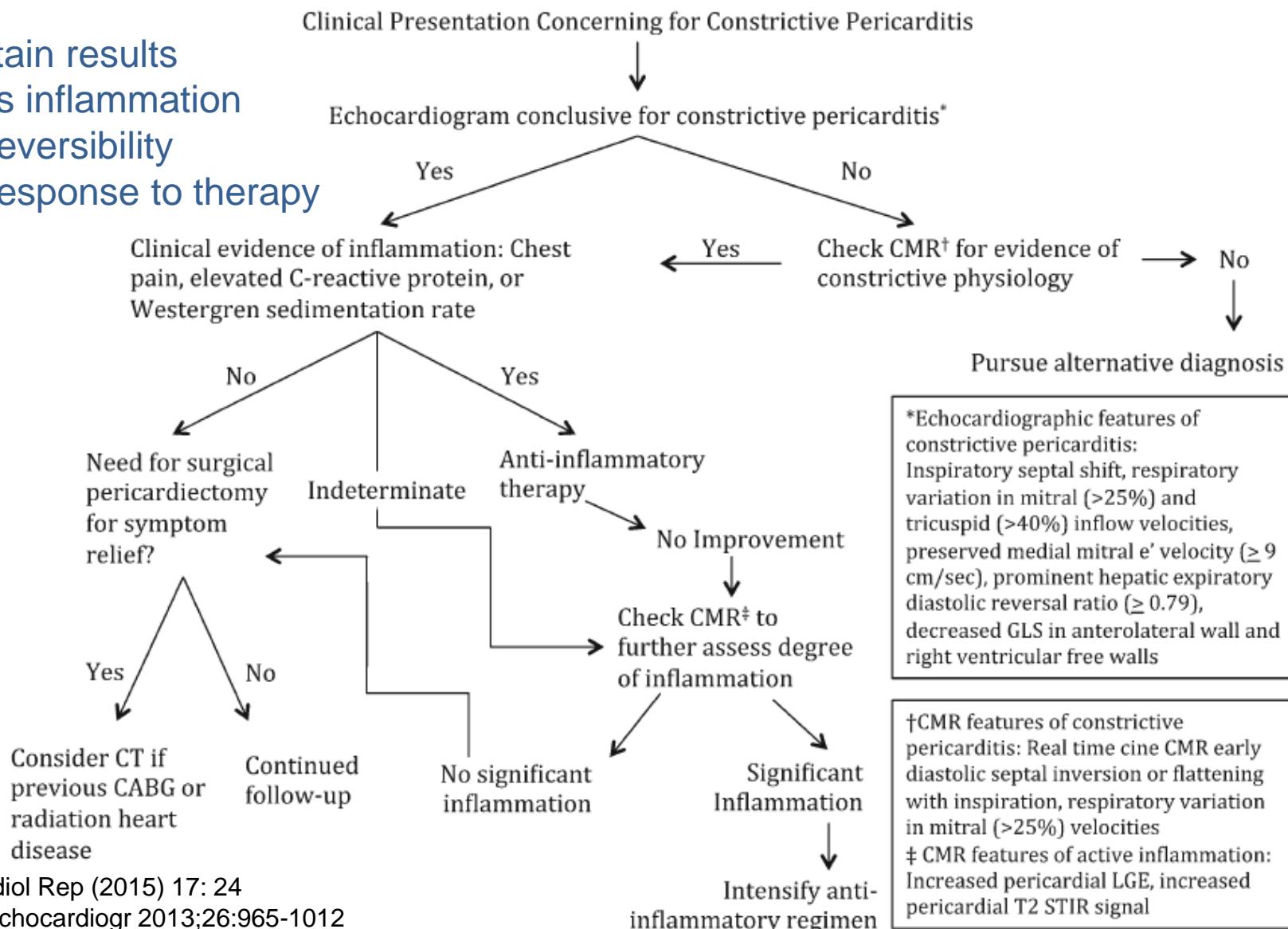
- All patients with reversible constrictive pericarditis had pericardial LGE
 - 93% patients had moderate or severe pericardial LGE

Not All Patients with CP Need to Undergo Pericardectomy

When to Consider Added Imaging in Chronic Pericarditis

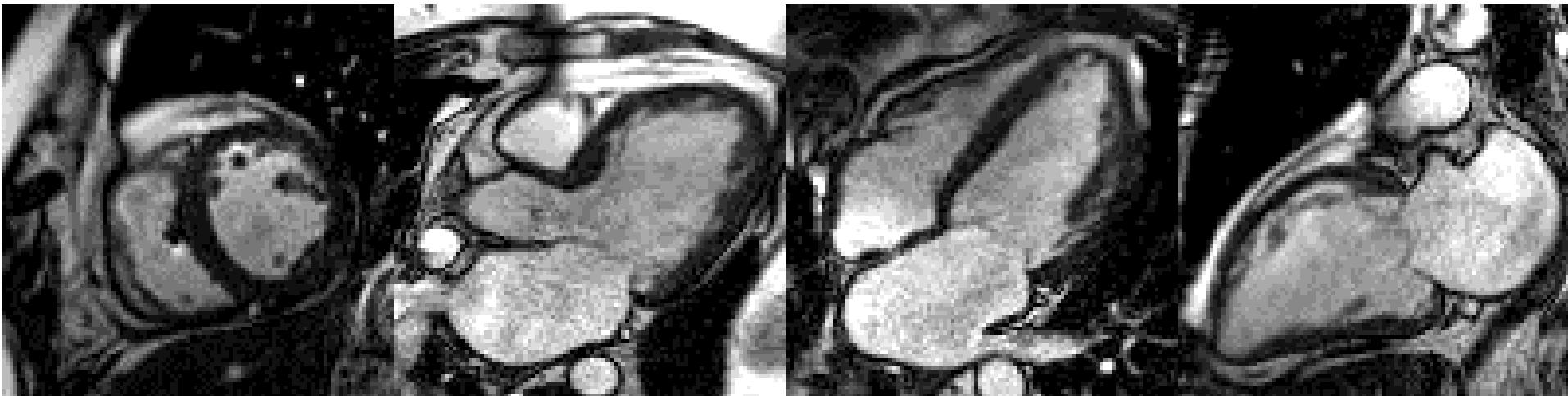
CMR:

- Uncertain results
- Assess inflammation
 - Reversibility
 - Response to therapy



Case 5: Constriction?

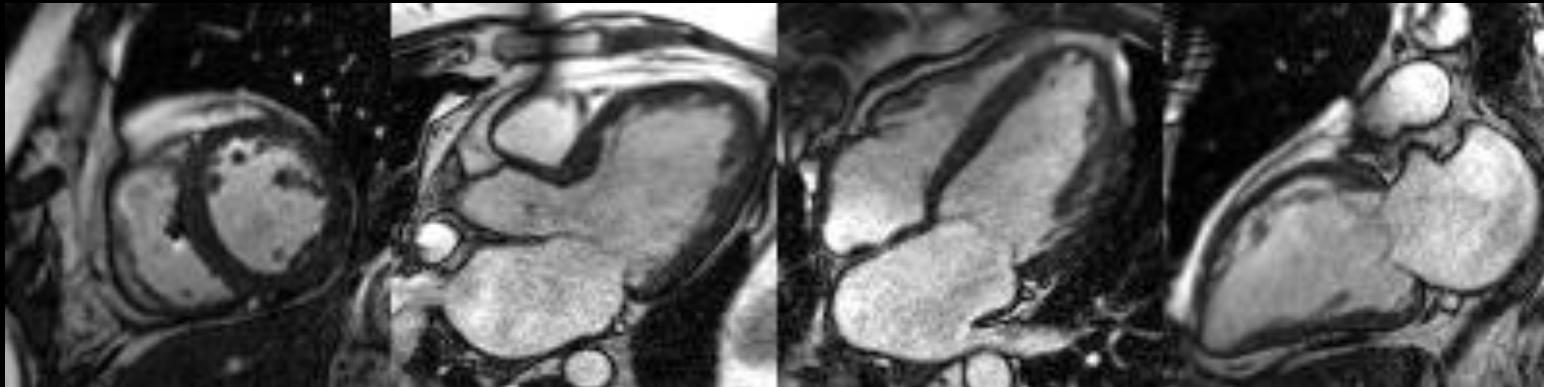
65 NICMP with RHF sxs (recurrent pleural & pericardial effusions)
Referred to CMR for pericardial evaluation



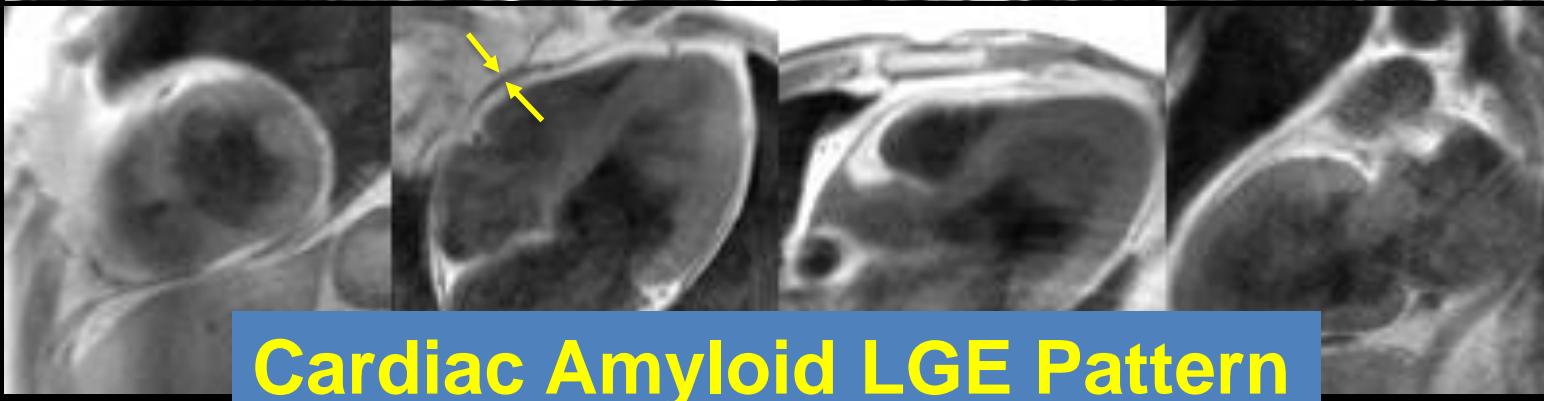
Tissue Characteristics:

Differentiating Constriction vs Restriction

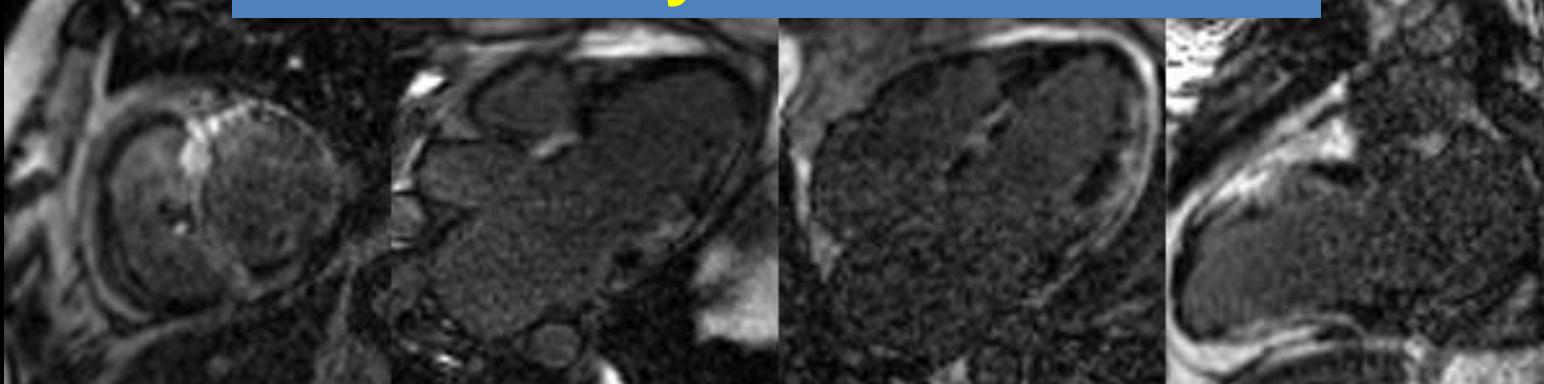
SSFP



T1

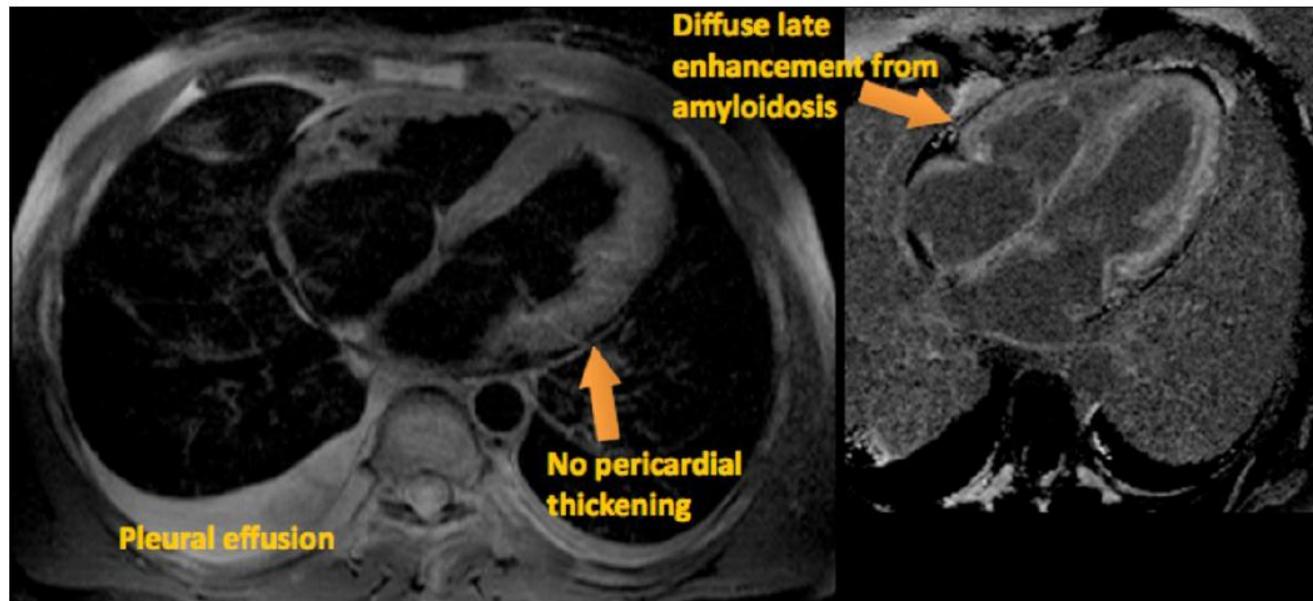


LGE



Constriction vs Restriction

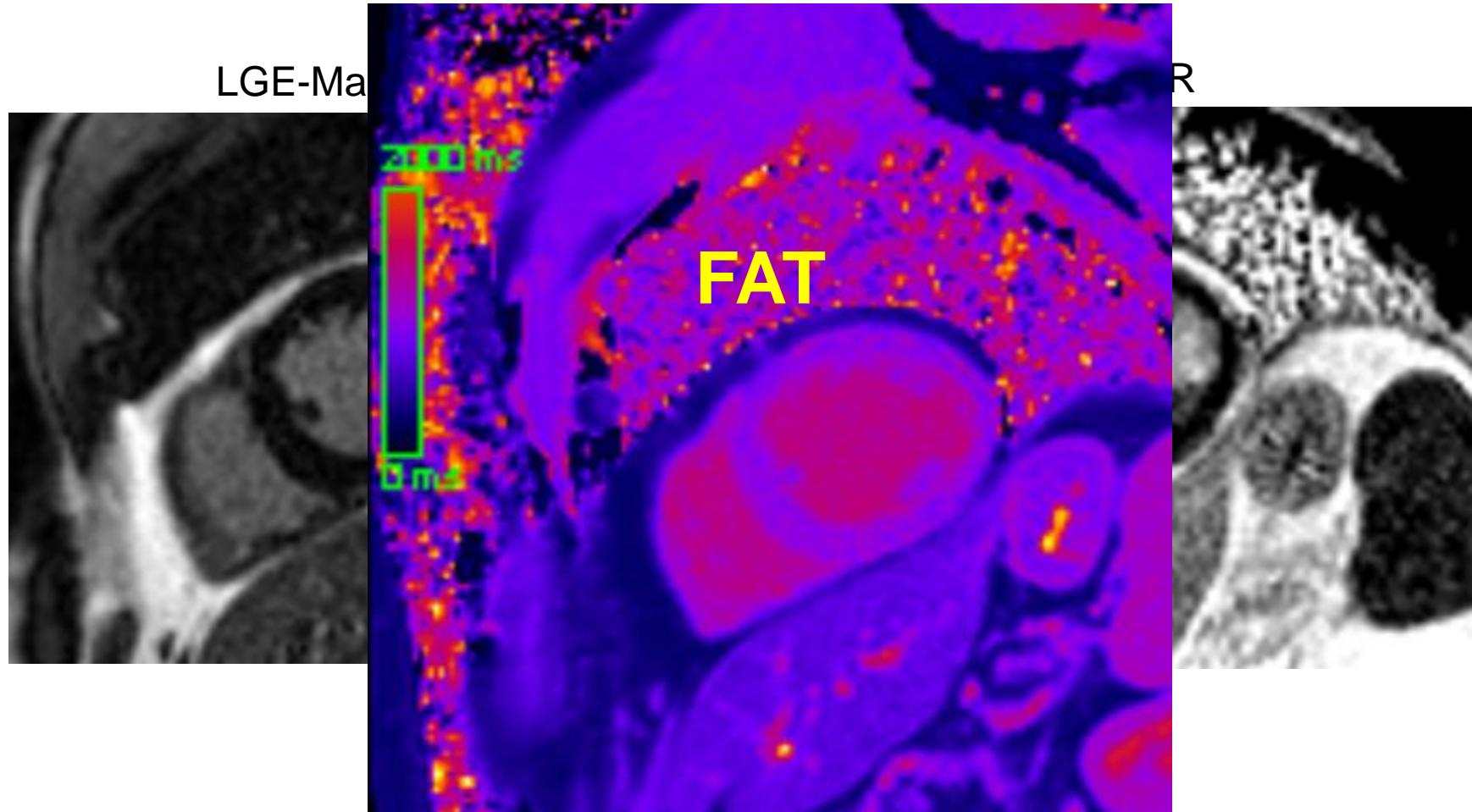
	Constriction	Restriction
Equal right-left side filling pressures	Present	Left at least 3-5 mm Hg > right
Respiratory variation in left-right pressures/flows	Exaggerated	Normal
Ventricular wall thickness	Normal	Usually increased
Atrial size	Possible LA enlargement	Biatrial enlargement
Septal "bounce"	Present	Absent
Pericardial thickness	Increased	Normal



Comparison of CMR Findings

	Acute Pericarditis	Constrictive Pericarditis	Restrictive Cardiomyopathy	Pericardial Effusion
T1W	Enhancement of thickened pericardium	Fibrotic and/or calcified pericardium has low signal intensity unless there is residual inflammation	Normal pericardial thickness and signal	Transudate: low intensity signal Exudative: High intensity signal
T2W	High intensity signal in the pericardial tissue	Fibrotic and/or calcified pericardium has low signal intensity unless there is residual inflammation	Normal pericardial thickness and signal	Transudate: High intensity signal Exudate: low intensity signal
LGE	High intensity signal	No LGE unless there is residual inflammation	Variable depending on the underlying disease but should have LGE in the pericardium	LGE in the case of acute pericarditis
RF tagging		Loss of the normal slippage of the outer pericardium over the epicardial surface during the cardiac cycle	Normal	
SSFP	Thickened pericardial layers (>4 mm), variable amount of pericardial fluid, septal bounce may occur due to decreased pericardial compliance	May have thickened pericardial layers (>4 mm), Septal bounce and respiratory variation in septal excursion	Normal pericardial thickness < 3mm	Pericardial width > 4mm regarded as abnormal amount of fluid
Phase encoding velocimetry	No specific findings unless there is pericardial effusion associated with tamponade physiology	Restrictive filling pattern of RV and LV diastolic filling; >25% fall in mitral inflow velocity and >40% increase in tricuspid velocity in the first beat after inspiration; opposite changes in expiration	May have restrictive filling pattern but no respiratory variation of flow across the mitral and tricuspid valve	In the case of tamponade Restrictive filling pattern of RV and LV diastolic filling; >25% fall in mitral inflow velocity and >40% increase in tricuspid velocity in the first beat after inspiration; opposite changes in expiration

Hyperenhancement?



Pericardial Masses

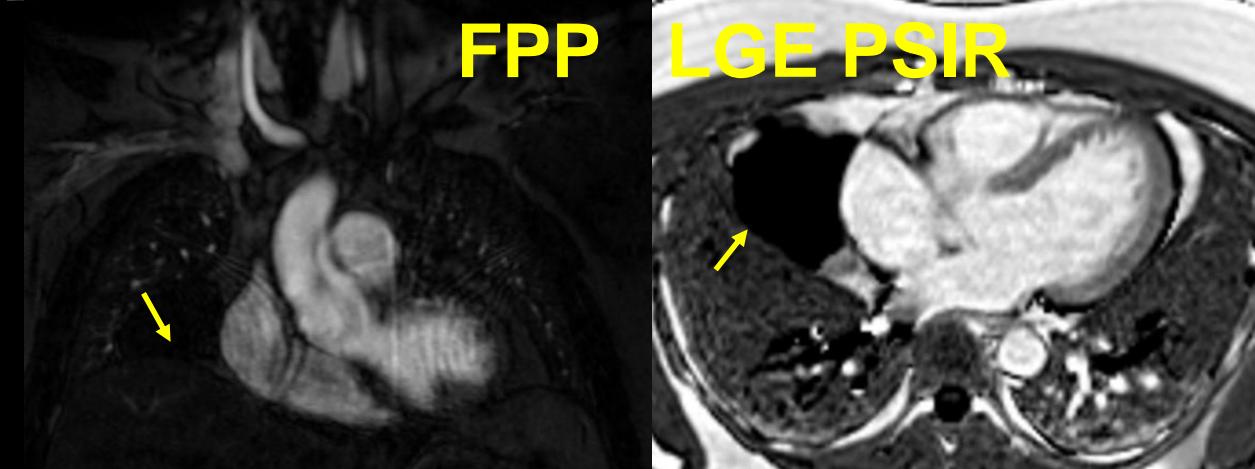
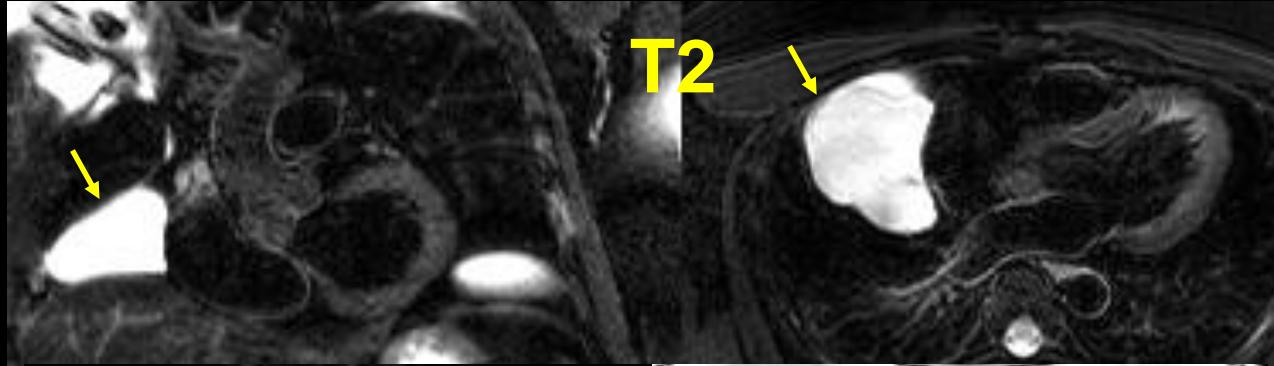
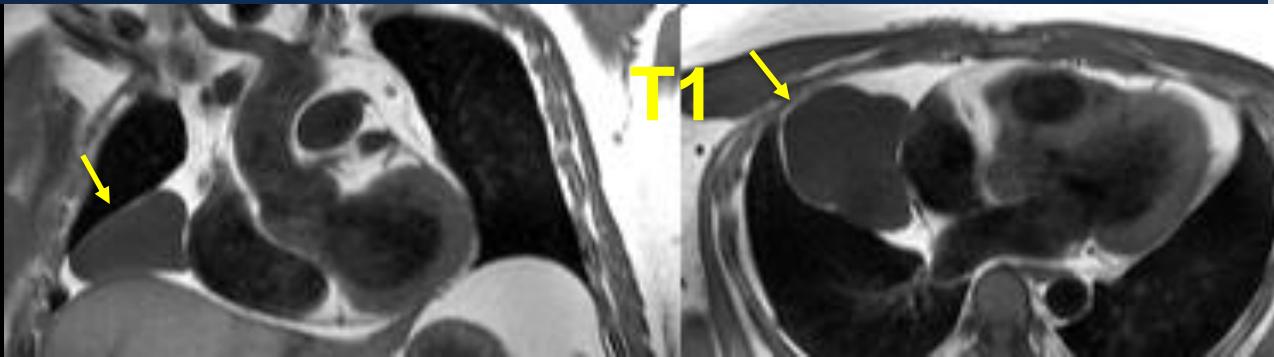
Table 2
Differential Diagnosis of Pericardial Masses

Cyst	
Hematoma	
Acute	
Subacute	
Chronic	
Metastatic neoplasm	<u>(Vast majority!)</u>
Carcinoma of lung or breast	
Lymphoma	
Melanoma	
Primary neoplasm	
Benign (lipoma, teratoma, fibroma, hemangioma)	
Malignant (mesothelioma, lymphoma, sarcoma, liposarcoma)	

- CMR provides:
 - Accurate description of the pericardial abnormalities
 - Relationship to the surrounding structures
 - *Tissue characterization*
 - Establishment of the differential diagnosis
 - Assessment of complications

Benign Pericardial Cyst

Tissue Characteristics



- Smooth-bordered, encapsulated fluid-filled cysts
- Right > left costophrenic angle
- Have signal characteristics of water
- Majority

CMR:

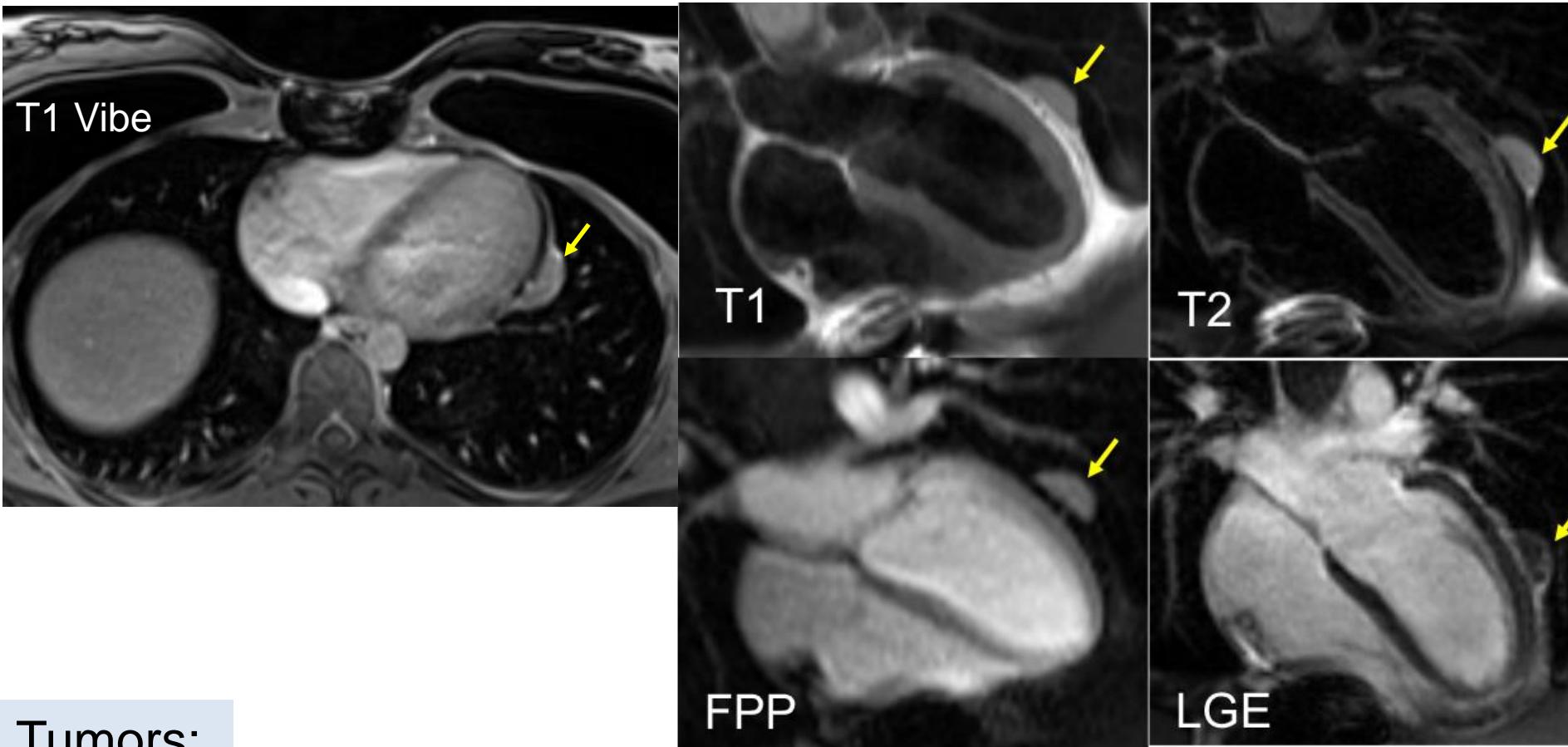
- low or intermediate signal T1
- homogeneous high intensity on T2
- No contrast uptake

Tumors Demonstrate Contrast Uptake



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32F with hx of prior pelvic mass removal

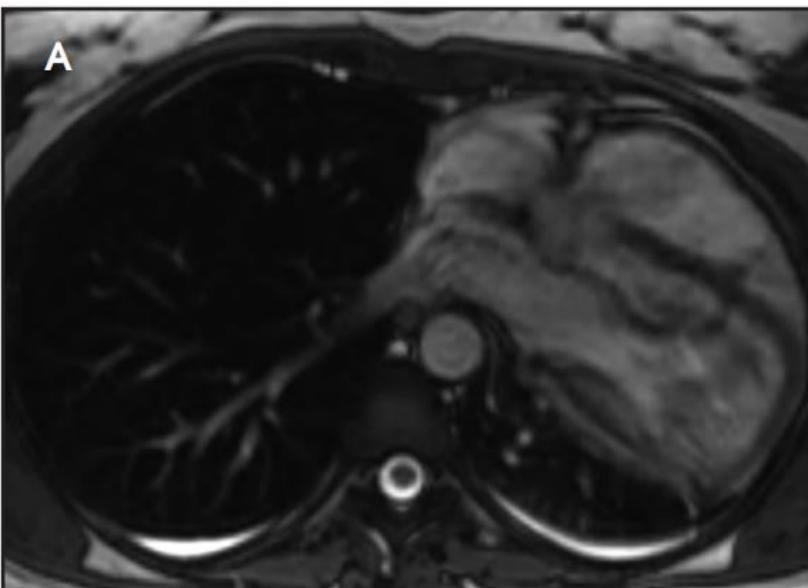


Tumors:

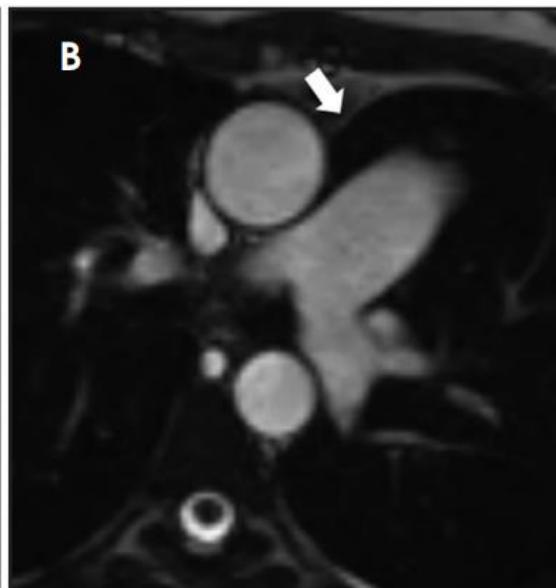
Hyperintense on T2 weighted sequences & demonstrate Gad uptake

Congenital Absence of the Pericardium

Complete

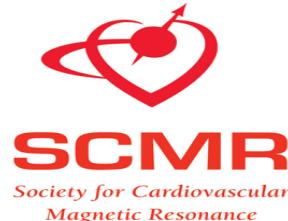


Partial



Appropriate Use Criteria

MMI in the Assessment of Cardiac Structure & Function



Indication	TTE (With or Without 3D; With Contrast as Needed)	TEE (With or Without 3D)	Stress Echo*	Strain/Strain Rate Imaging by Speckle or Tissue Doppler	F-18 FDG PET	Tc-99m PYP	MPI (SPECT/PET)	CMR	CT†	ANG	RVG
Other											
40. Suspected pericardial diseases	9 (A)	4 (M)	1 (R)	5 (M)	1 (R)	1 (R)	1 (R)	7 (A)	7 (A)	1 (R)	1 (R)

CMR Conclusions

- Powerful complementary tool to
 - Determine pericardial morphology
 - Assess physiological function / hemodynamics
 - Superior tissue characterization, except calcification
- Unique ability to detect pericardial inflammation and assess for reversibility and monitor response to treatment
- First choice for tissue characterization of cardiac masses