Cases You'll Never Forget

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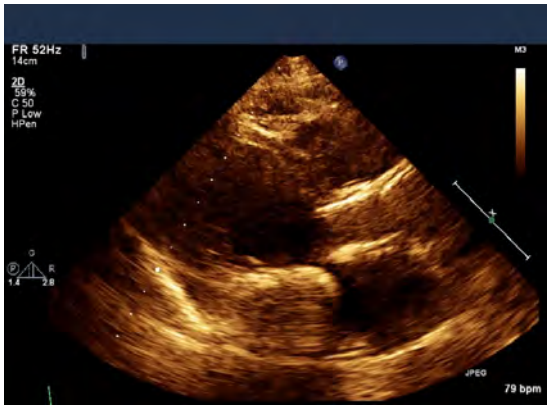
Caseous MAC

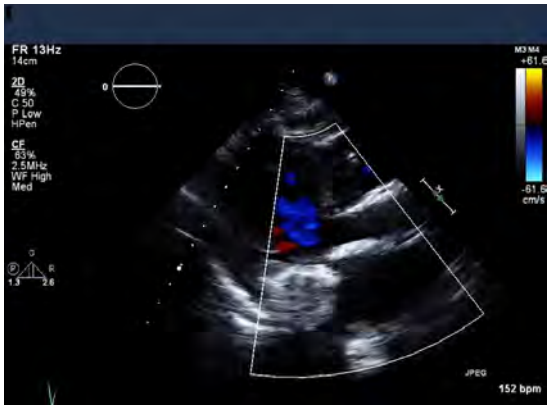
A 76 year-old woman with transcatheter aortic valve replacement and complete heart block with a dual-chamber permanent pacemaker had previously been diagnosed with severe caseating mitral annular calcification (MAC).

The mass was 3.4cm by cardiac magnetic resonance imaging in 2014, and had continued to grow over time causing mild mitral stenosis due to mass effect

2

Baseline TTE





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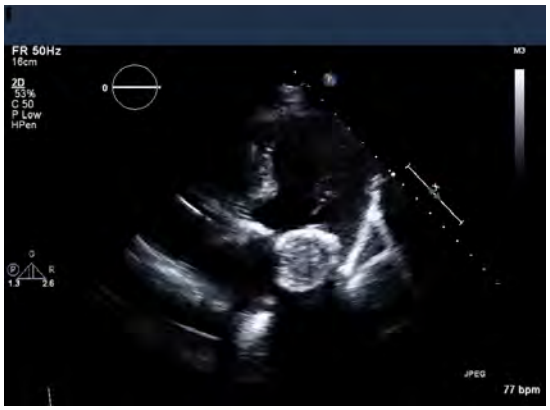
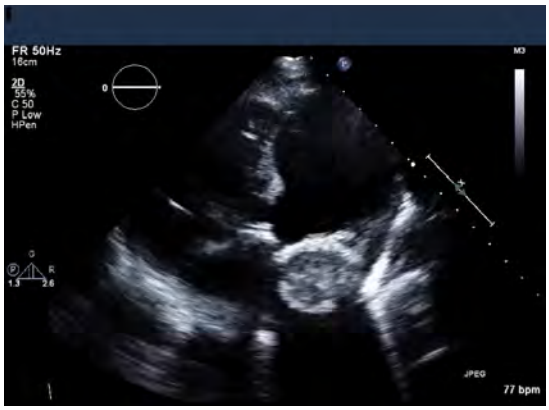
Baseline TTE





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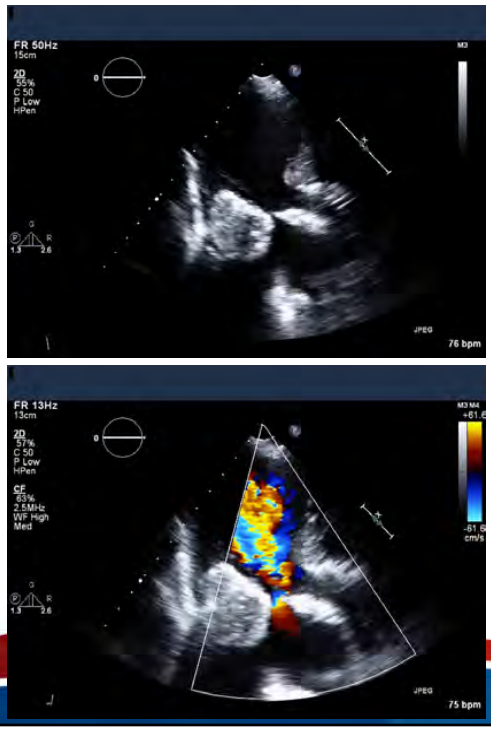

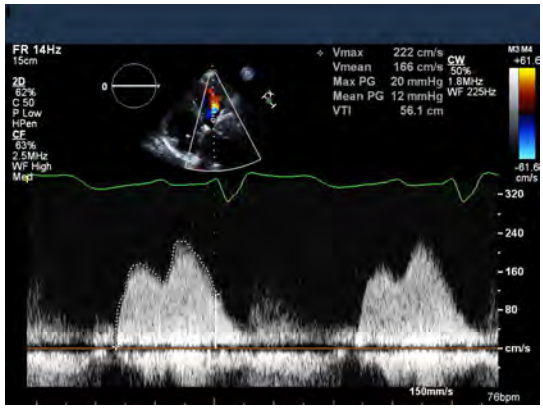

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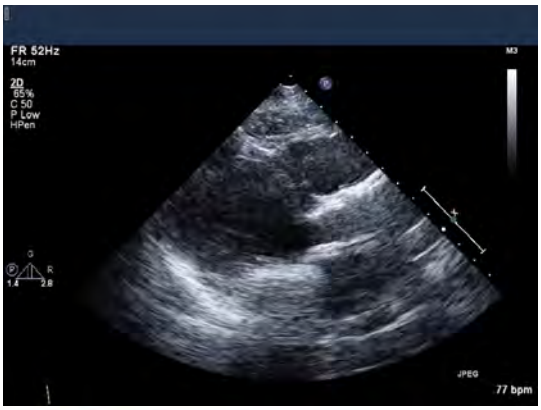
Following a stroke, a repeat TTE

Baseline



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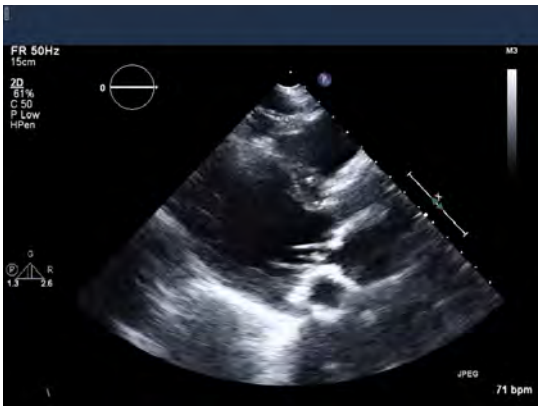

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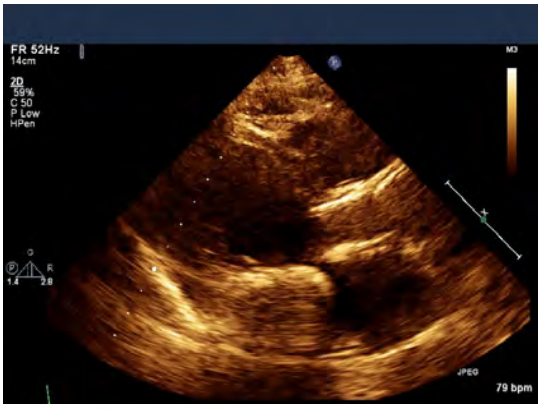
Baseline



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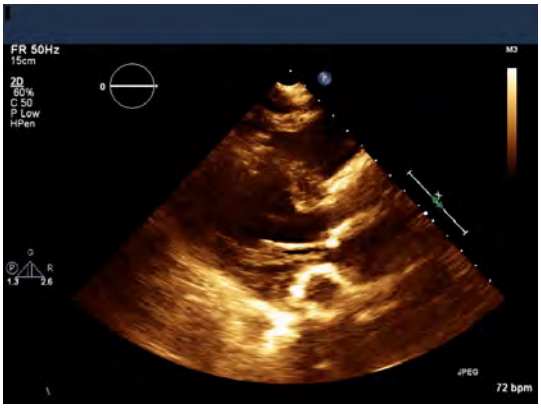

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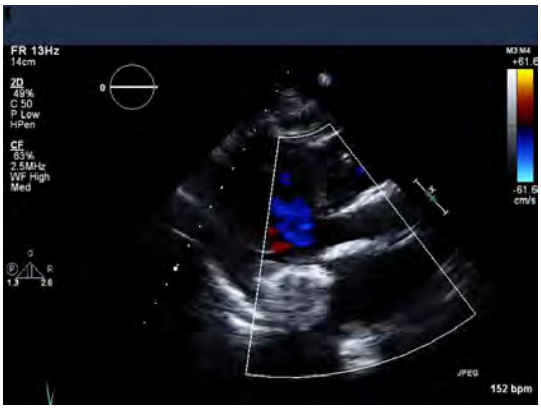
Baseline



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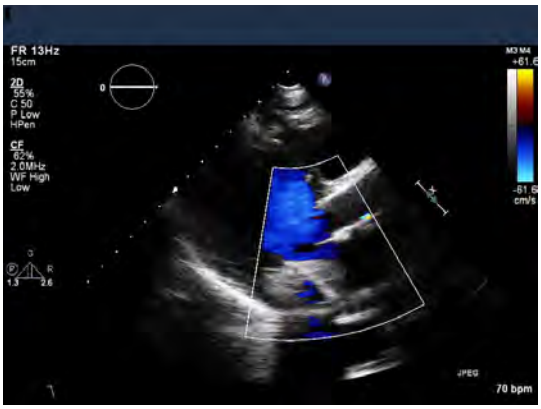

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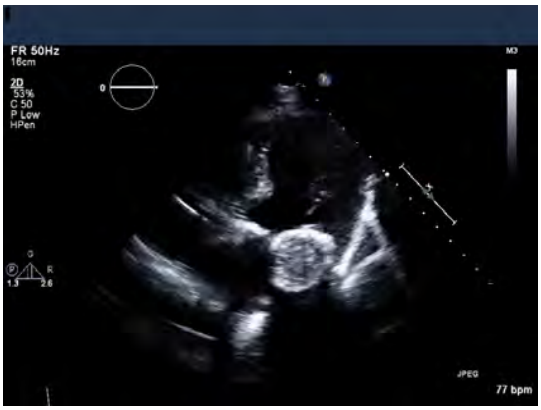

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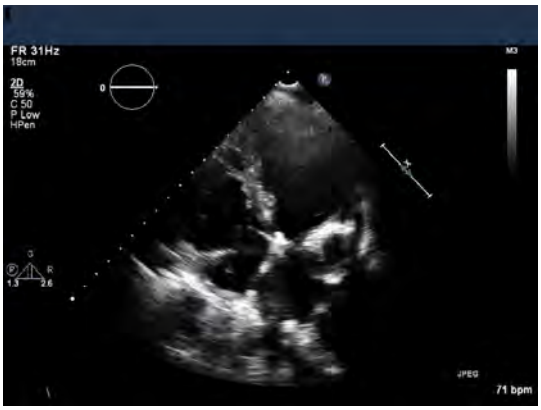

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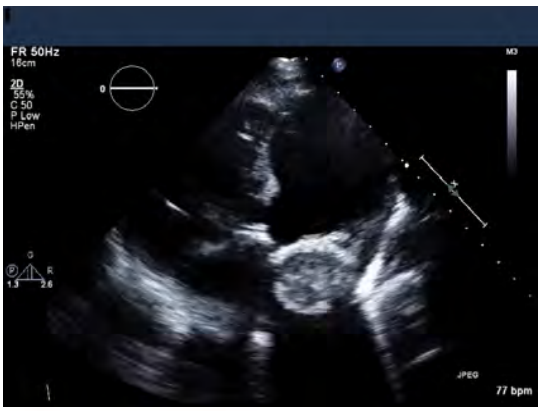
Baseline



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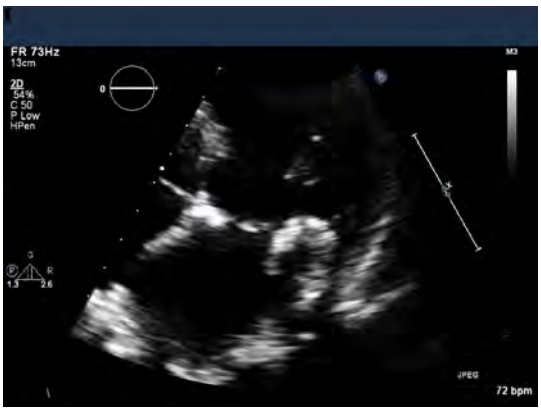

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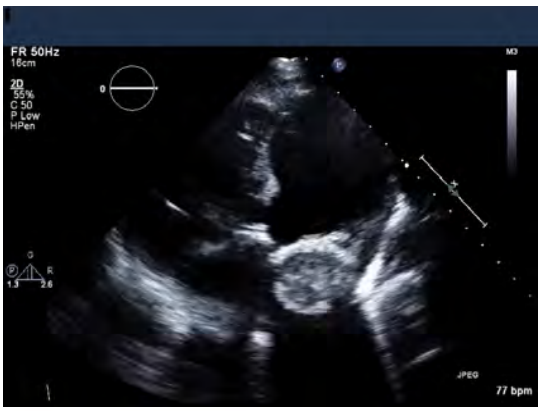
Baseline



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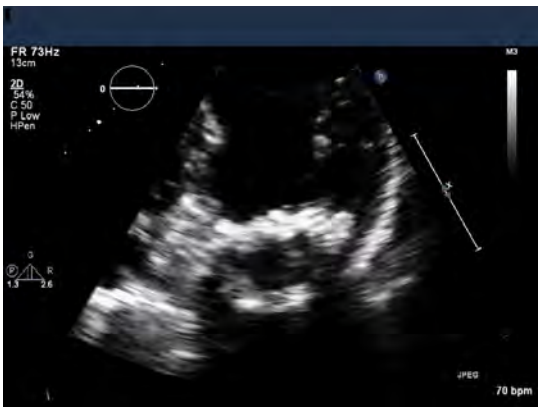

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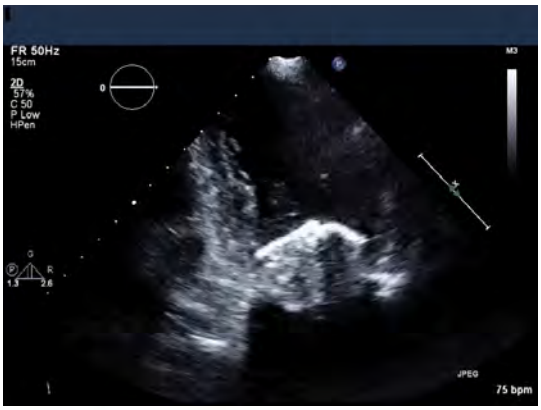
Baseline



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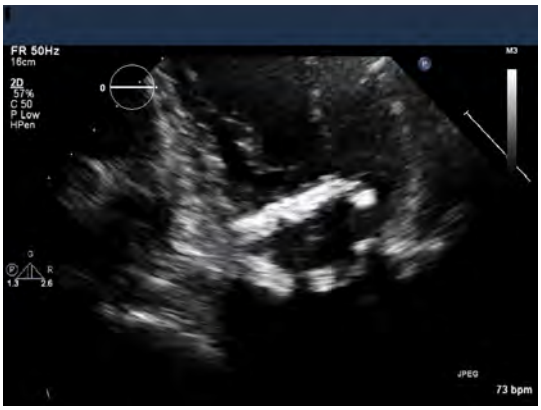

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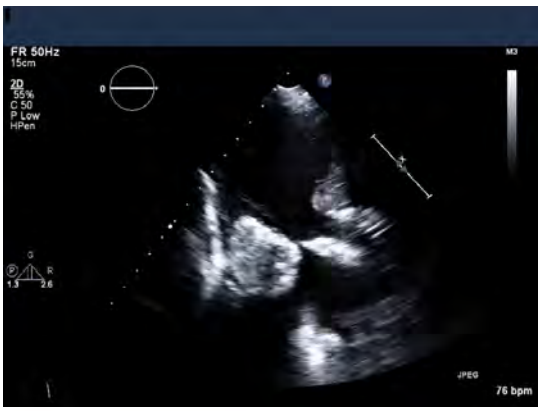
Baseline



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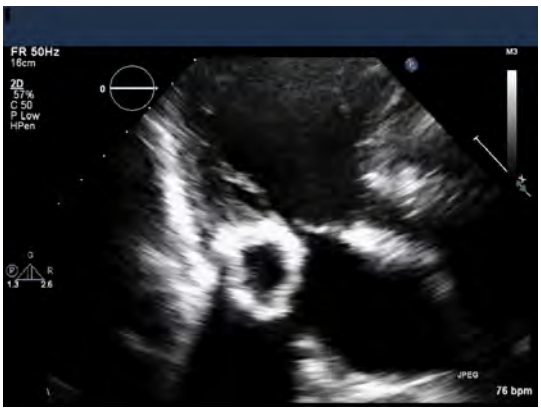

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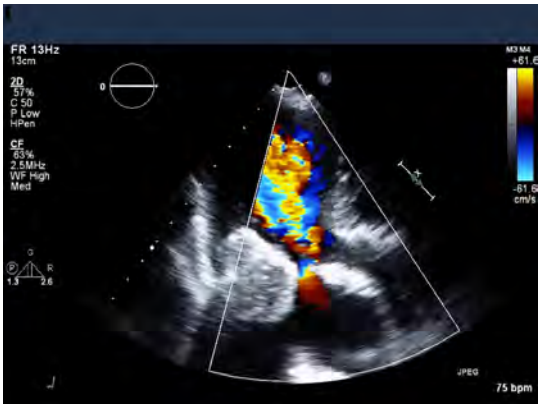
Baseline



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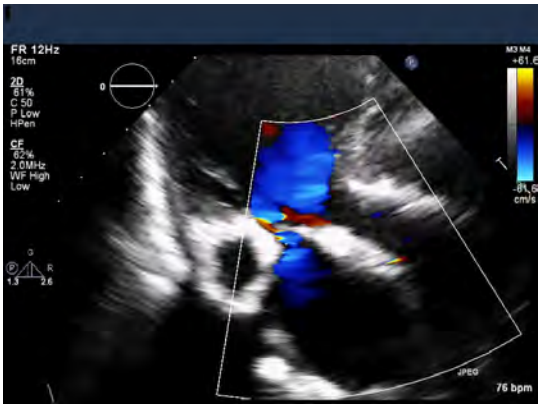

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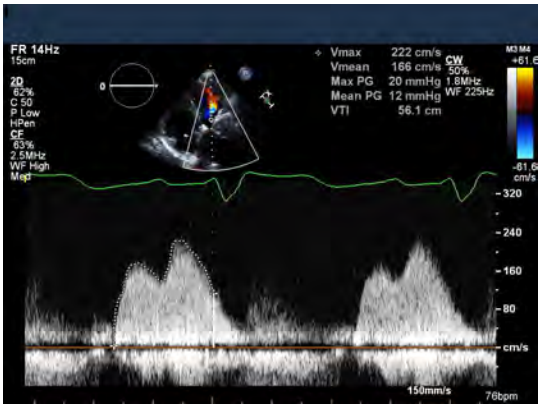
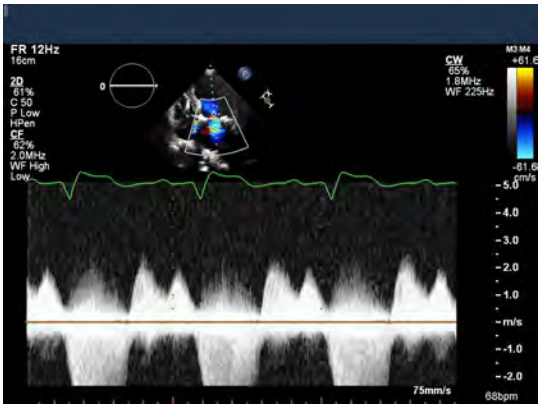
Baseline



Peak gradient = 20 mmHg
Mean gradient = 12 mmHg

Follow-up



Peak gradient = 20 mmHg
Mean gradient = 6.5 mmHg

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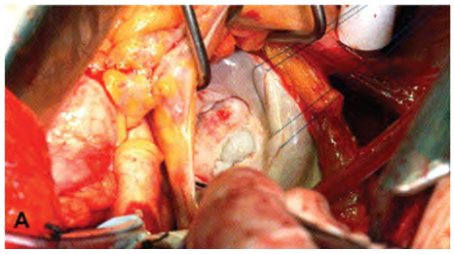
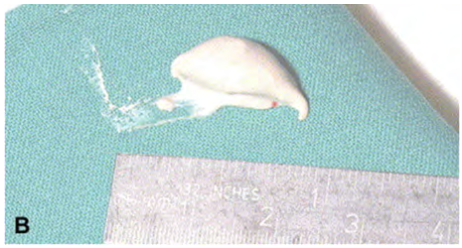
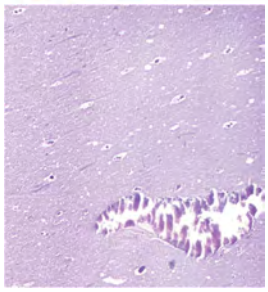
Caseous MAC







- Caseous mitral annular calcification (CMAC) is a variant of MAC with a central liquefaction necrosis with a prevalence of only 0.6%
  - The lesion is typically composed of an admixture of calcium, fatty acids, and cholesterol and has “toothpaste like” texture.
  - This pasty material is composed of amorphous acellular basophilic substance with scattered calcifications and sparse histiocytes

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MAC and Stroke

Patients with MAC have a two-fold increased risk of stroke, and patients with caseating MAC have an even higher risk of stroke than patients with MAC.[1,2]

References

1. **Benjamin EJ, Plehn JF, D'Agostino RB, et al. Mitral annular calcification and the risk of stroke in an elderly cohort. *The New England journal of medicine.* 1992;327(6):374-379.**
2. **Dietl CA, Hawthorn CM, Raizada V. Risk of Cerebral Embolization with Caseous Calcification of the Mitral Annulus: Review Article. *Open Cardiovasc Med J.* 2016;10:221-232.**

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Caseous Calcification Mitral Annulus: Transthoracic Echo

- CCMA appears as a large, round, echo-dense mass with smooth borders situated in the periannular region, with no acoustic shadowing artifact and contains central areas of echolucencies resembling liquefaction.
- It may appear as a semilunar mass in the parasternal short-axis view.
- The mass is usually located in the posterior region, at the junction between the left atrium and the left ventricle, bulging into the left atrial cavity or into the adjacent left ventricular posterior wall.
- The mass rarely extends along the whole mitral annulus
  - On the contrary, MAC appears as a J-, C-, or U-shaped dense echo band, which creates an acoustic shadowing and lacks the echolucent center
  - On color Doppler, the central zone of CCMA lacks any flow.

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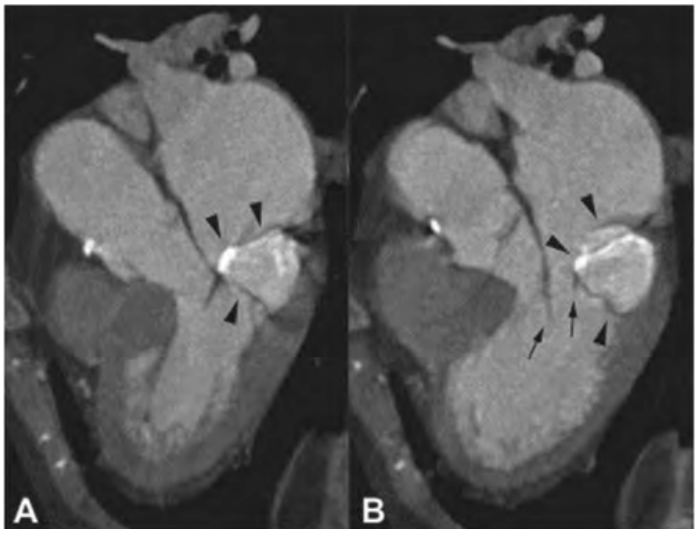
Caseous MAC

- On CT scan, CCMA appears as a well-defined oval or crescent-shaped hyperdense mass with peripheral calcification, usually along the posterior mitral annulus, has very high Hounsfield units, and lacks contrast enhancement.
- The central hyperdensity is thought to be secondary to the liquefactive material that fills the center of the mass



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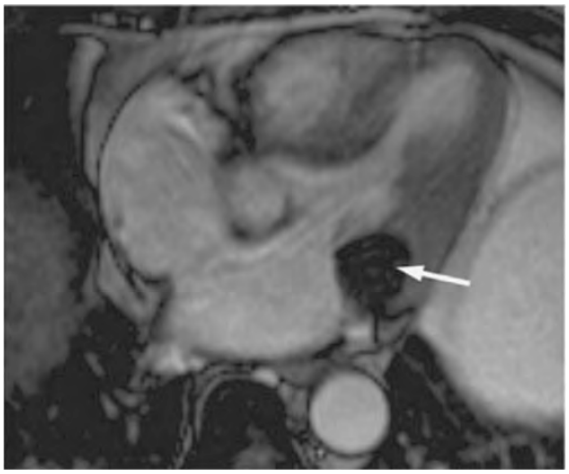
Caseous MAC

- Cardiac MRI is considered to be the technique of choice in doubtful cases.
- Findings of CCMA on cardiac MRI include a well-defined mass with hyperintense center and hypointense rim, discrete from the adjacent myocardium and posterior mitral valve on T1-weighted fast spin-echo imaging.
- A similar centrally hyperintense mass with a peripheral rim of hypointensity is also visualized with T1-weighted spoiled gradient-echo imaging techniques
- On T2-weighted MRI sequences, CCMA appears as a mass devoid of a central signal but with a ring of high intensity compared with the surrounding myocardium



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Differential Diagnosis of Caseous Calcification Mitral Annulus

- **Tumors:**
  - Myxoma is usually mobile, pedunculated, and attached to the myocardial wall along the interatrial septum in the region of fossa ovalis via a thin stalk. It lacks the calcifications in the borders that are seen in CCMA. Additionally, it is highly vascular compared with CCMA, in which no blood flow should be visualized by color Doppler
  - Uncommon tumors include hemangioma of the mitral valve and malignant lesions like leiomyosarcoma. In hemangiomas, the echolucencies (representing the tumor vascularity) extend all the way to the periphery of the mass and no calcification is found.

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Differential Diagnosis of Caseous Calcification Mitral Annulus

- **Myocardial Abscess**
  - A myocardial abscess appears as a mass within the myocardium or annular region, with homogenous echogenic appearance on echocardiography. It lacks calcifications and may show systolic blood flow by color Doppler. It can closely resemble CCMA. Not surprisingly, the first reported cases of CCMA were described as “sterile myocardial abscess.”

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Differential Diagnosis of Caseous Calcification Mitral Annulus

- **Infective Endocarditis**
  - Infective endocarditis (IEC) is diagnosed with the use of Duke criteria. One of the major criteria is a positive echocardiographic finding. IEC appears as an oscillating mass on a support structure, an abscess (undefined structure), a partial dehiscence of a prosthetic valve, or new valve regurgitation

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Indications for Surgery for CCMA

- **The current indications for surgical intervention include**
  - Mitral valvular dysfunction (either stenosis or regurgitation)
  - Embolic manifestations, or when it is impossible to rule out the possibility of a tumor.
    - Mitral valve replacement is preferred to mitral valve repair
    - It should be noted that aggressive debridement may increase the risk of perforation of the left ventricle, and simple unroofing of the cavity would continue to expose necrotic debris to the systemic blood flow.
    - Therefore, a technique of non-everting annular sutures is recommended
    - Anticoagulation should be considered in patients with CCMA who present with embolic manifestations

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Case Presentation

8/29/19

MARK LEBEHN, MD

1

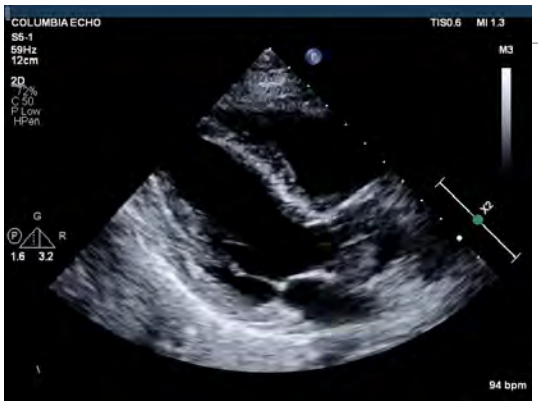
Case 1

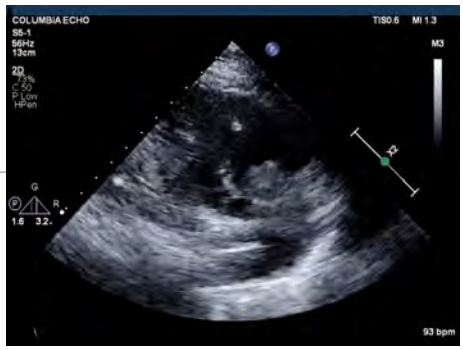
Ms AE is a 55 yo woman with a history of breast CA S/P lumpectomy and adjuvant radiation therapy (2010), metastatic pancreatic adenocarcinoma ongoing chemotherapy, and DVTs (bilateral), who presented with symptoms of 1 week of fatigue, dyspnea on exertion, and intermittent heart burn.

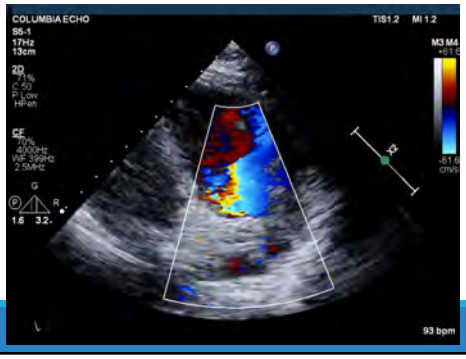
The day of admission she underwent a routine staging CT chest/abd/pelvis with contrast and Transthoracic Echocardiogram leading to referral to ED.

2

Transthoracic Echo







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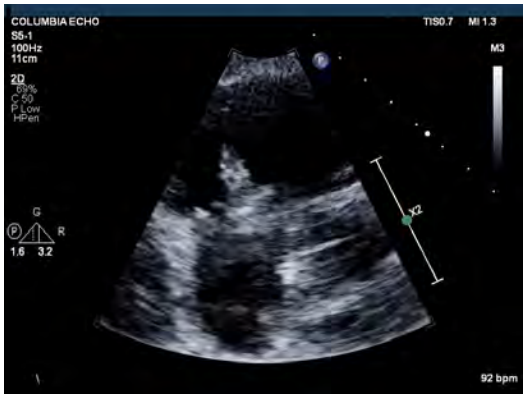
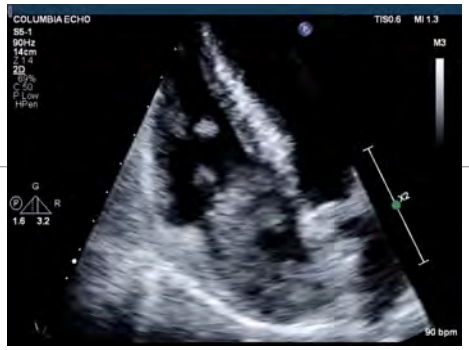
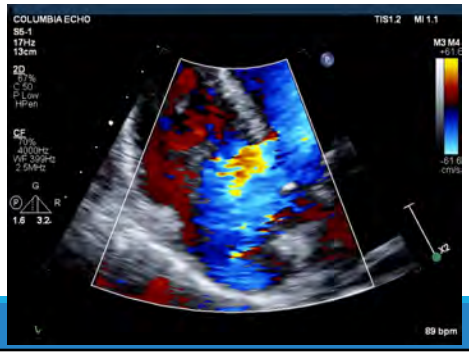
Transthoracic Echo







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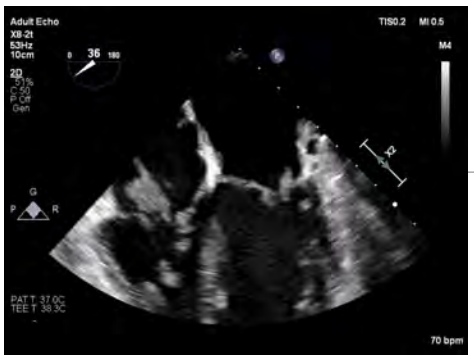
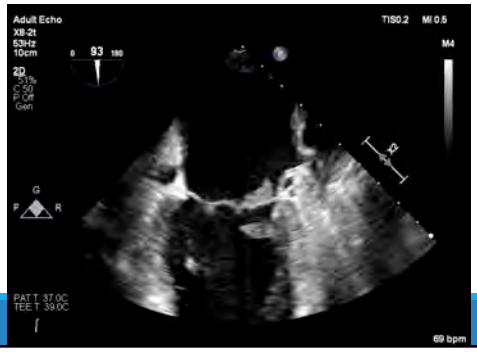
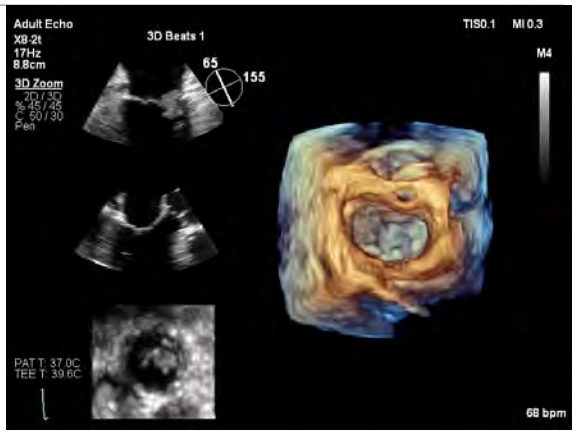
Transesophageal Echo







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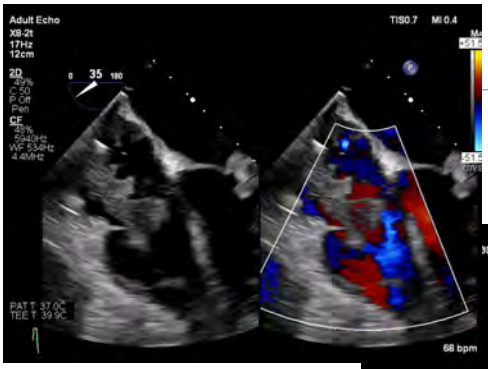
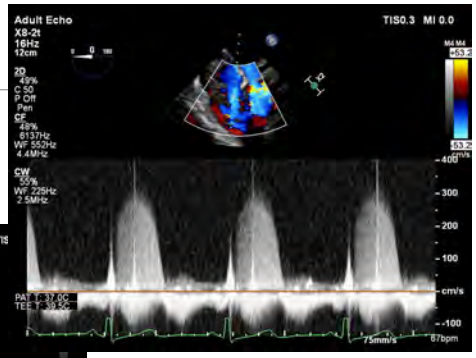
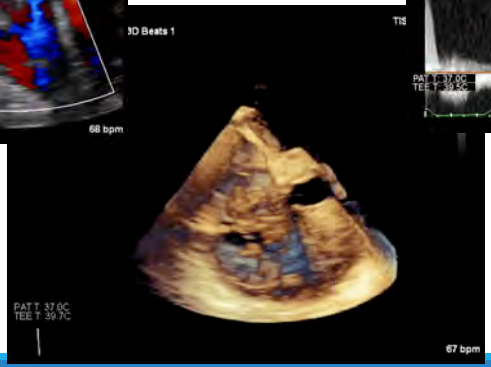
Transesophageal Echocardiogram







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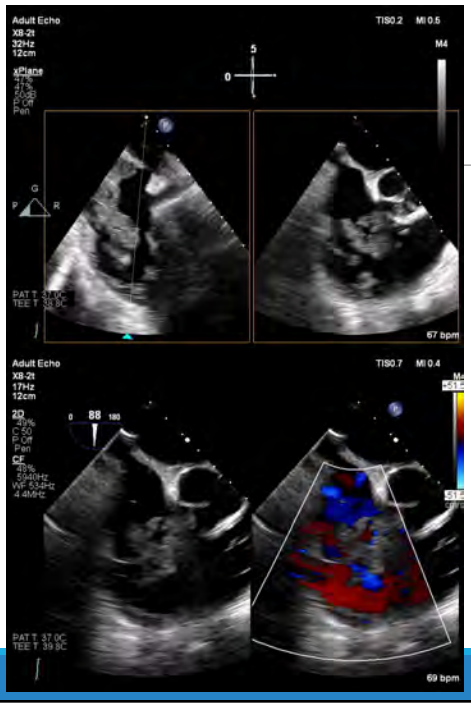
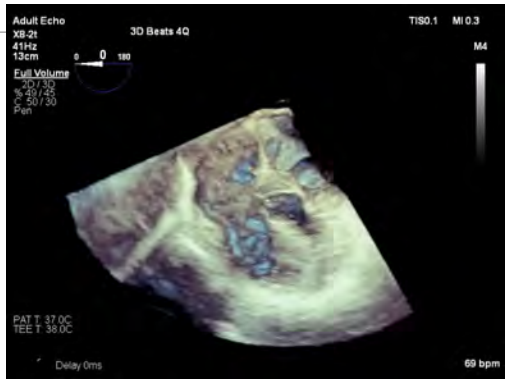
Transesophageal Echo





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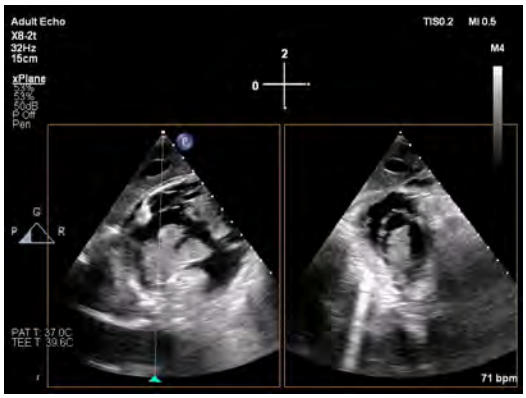
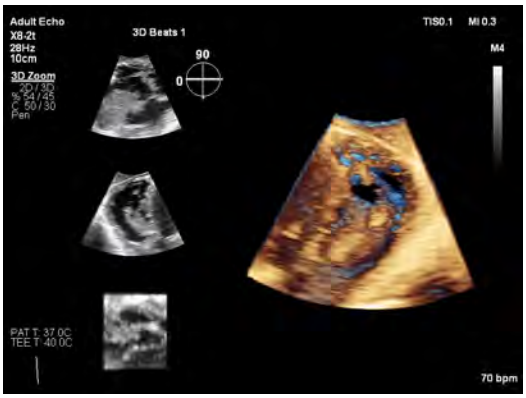
Transesophageal Echo

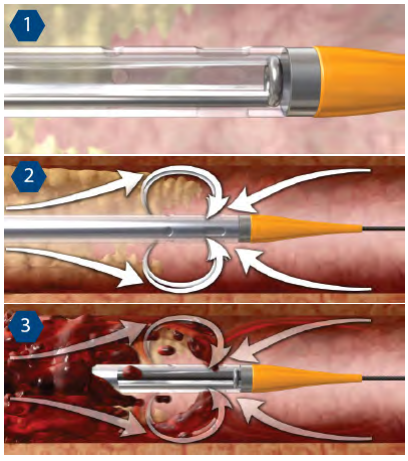




8

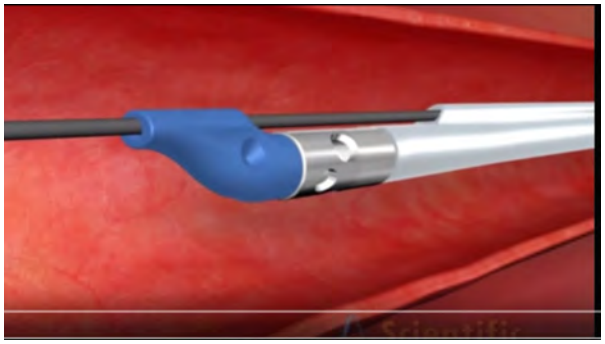
- Rheolytic thrombectomy procedures have already proved their efficacy and safety in coronary as well as in extra-coronary domains, such as peripheral arterial and venous diseases.

9



Mechanism of Action

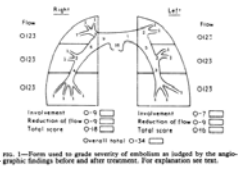
ANGIOJET uses high pressure saline to create a vacuum at the tip of the catheter to break up and remove thrombus.



1. Saline jets travel backwards at high speed to create a negative pressure zone (less than -600 mmHg) causing a powerful vacuum effect.
2. Cross-Stream™ windows optimize the fluid flow for more effective thrombus removal.
3. Thrombus is drawn into the catheter where it is fragmented by the jets and evacuated from the body.

10

Rheolytic thrombectomy (RT)



1. Zeni et al in 2003 applied the AngioJet® RT in 17 patients presenting with PE. In this series, the AngioJet® RT procedure was generally associated with the administration of an intrapulmonary thrombolysis, mortality rate: 11.8%.
2. Chechi et al suggested the safety and efficacy of this specific RT procedure in 51 high-risk (defined as a Miller index ≥17 at angiography) PE patients; mortality rate: 15.7%.
3. Nassiri et al have reported the AngioJet® RT ± power-pulse spray technique in 15 patients presenting mainly with non-high-risk PE (93% of the included sample); no mortality.
4. Bonvini et al piloted the AngioJet® RT in a series of patients presenting cardiogenic shock with technical success in all cases; 30d mortality was 70%.

1. Zeni PT Jr, Blank BG, Peeler DW. J Vasc Interv Radiol. 2003;14:1511-5.
2. Chechi T, Vecchio S, Spaziani G, Giuliani G, Giannotti F, Arcangeli C, Rubboli A, Margheri M. Catheter Cardiovasc Interv. 2009;73:506-13.
3. Nassiri N, Jain A, McPhee D, Mina B, Rosen RJ, Giangola G, Carroccio A, Green RM. Ann Vasc Surg. 2012;26:18-24.
4. Bonvini RF, Roffi M, Bounameaux H et al. Eurintervention 2013 Apr 22;8(12):1419-27. doi: 10.4244/EIJV8I12A215.

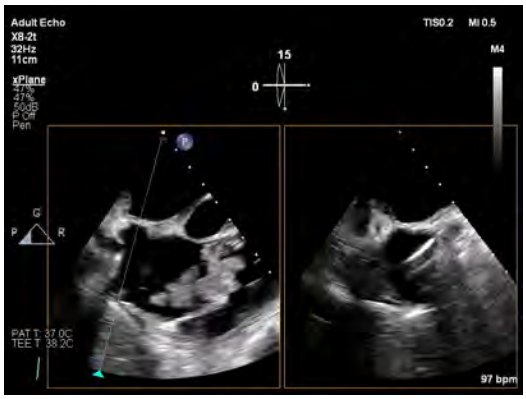
11

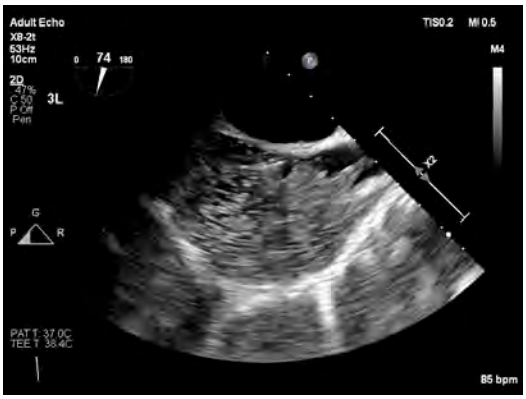
POTENTIAL ANGIOJET®-RELATED COMPLICATIONS

1. Fragmentation of the clot induces significant hemolysis which may be associated with a massive release of neurohormonal substances such as adenosine and bradykinin at the pulmonary vasculature level.
   - This phenomenon, associated with the concomitant activation of stretch receptors in the pulmonary arteries, is considered to be the leading cause of procedure-related bradyarrhythmias and
2. Hemolysis related severe hyperkalemia and haemoglobinuria.
   - Hyperkalaemia may contribute to worsening the electrical instability, finally leading to severe ventricular arrhythmias, while haemoglobinuria causes further deterioration of renal function, which is often already impaired by the concomitant severe low cardiac output which occurs during high-risk PE.

12

Transesophageal Echo





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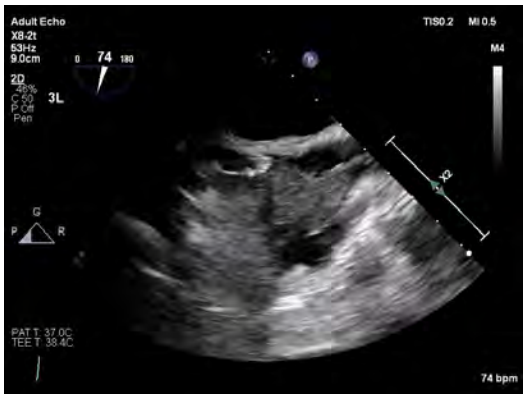
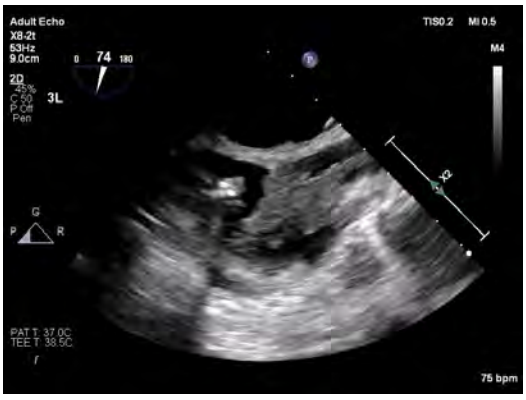
Transesophageal Echo





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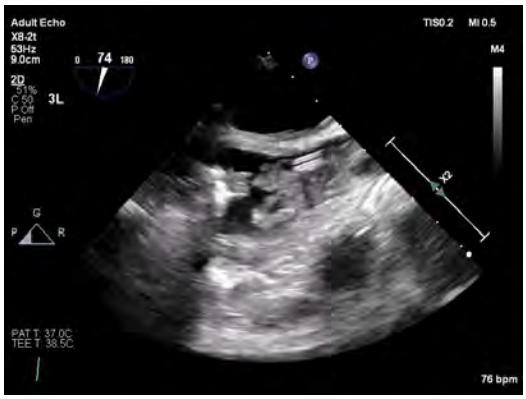
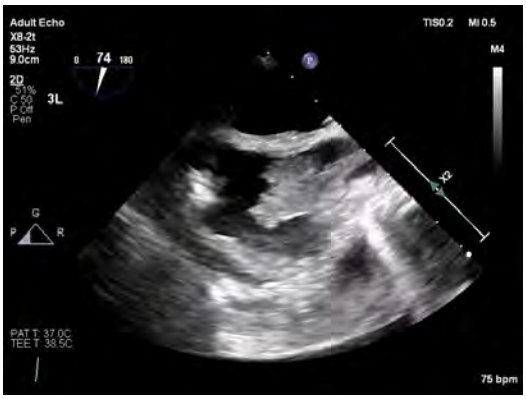
Transesophageal Echo





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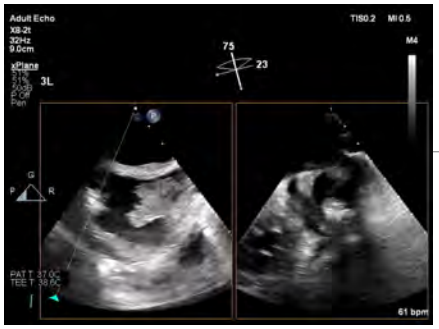
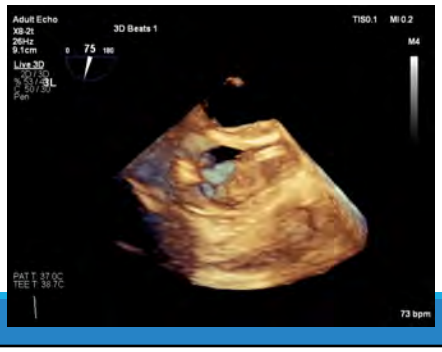
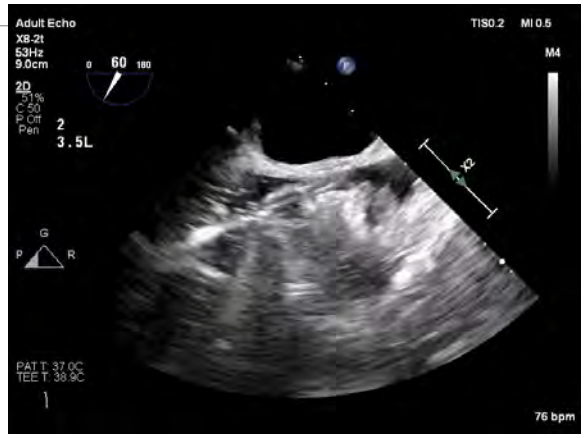


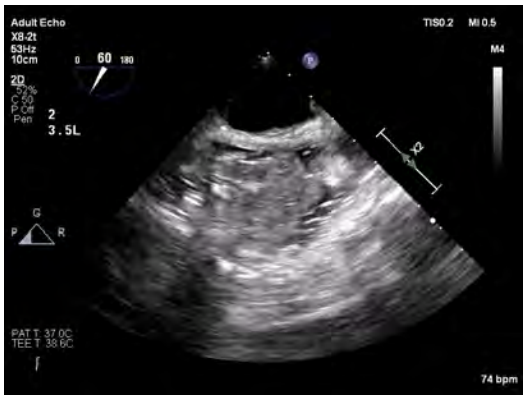


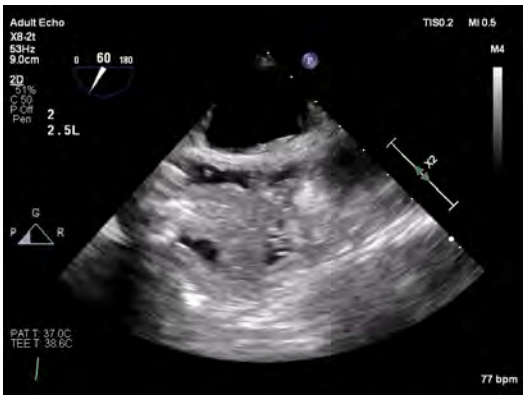
Transesophageal Echo



16

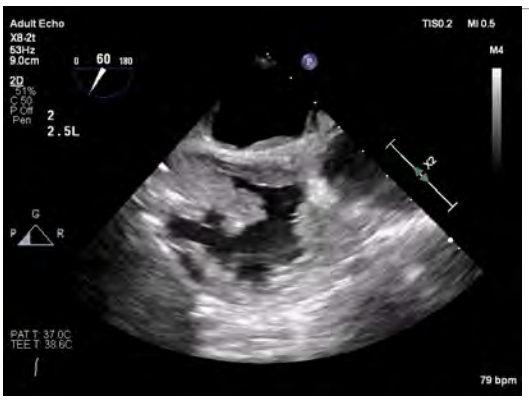
Transesophageal Echo

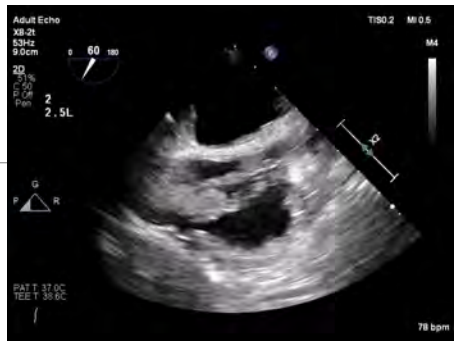


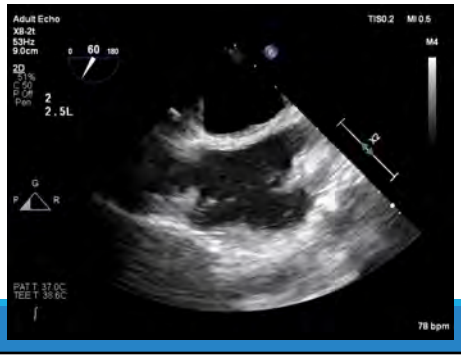


17

Transesophageal Echo

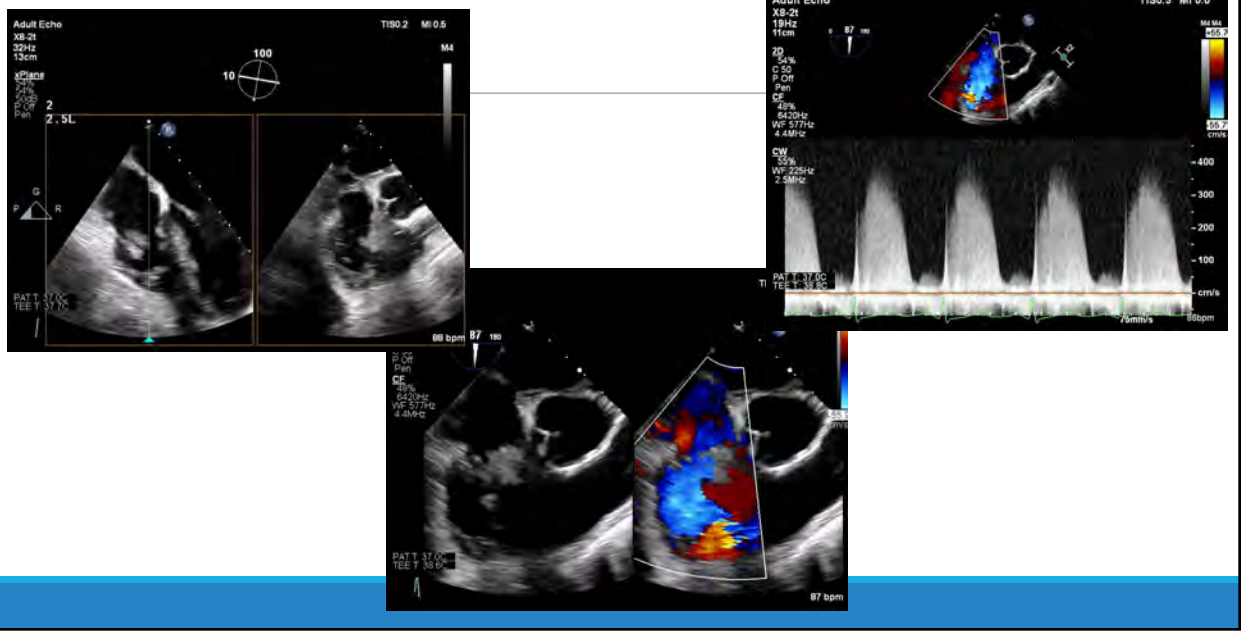






18

Transesophageal Echo



19

Pathology

- Friable bown-white irregular tissue measuring (2.0 x 2.0 x 1.4 cm)
- Fragments of thrombus and no malignant neoplasm seen

20

Hospital course:

- Admit 8/6, PERC team consultation
- Angiovac thrombectomy 8/7
- Extubated 8/8
- Uneventful hospital course and discharged 8/11

21

Non-Infectious Endocarditis (NIE)

NIE was first described by Zeigler in 1888 as fibrin deposition on cardiac valves

Autopsy study 409 cases of IE and 405 cases of NIE showed:

- The prevalence of NIE in this large cohort was 3.7%
- NIE was diagnosed three times more often than IE; moreover, although the majority of IE cases had been diagnosed clinically, none of the 405 NIE cases had been diagnosed pre-mortem.
- The location and characteristics of the vegetations in NIE and IE largely overlap, which suggests that rheological factors or prior valve injury predispose to the formation of lesions in specific areas.
- Other Terms: thromboendocarditis, terminal or marantic endocarditis, non-bacterial thrombotic endocarditis, and degenerative verrucous endocarditis.

BUSSANI R ET AL. IN VIVO 2019 SEP-OCT; 33(5): 1565–1572.

22

Nonbacterial thrombotic endocarditis (NBTE)

- Prothrombotic condition (eg **malignancy**, systemic lupus erythematosus, antiphospholipid antibody syndrome) leading to deposition of thrombi in the absence of bloodstream infection
- Prevalence is unknown, by autopsy study NBTE prevalence was 1.6% in an adult population

IBRAHIM AM, SIDDIQUE MS. LIBMAN SACKS ENDOCARDITIS.

23

NBTE

- NBTE is a rare and may occur in 4% of all patients with disseminated cancer
- Highest rate in patients with underlying malignancy compared to the general population on autopsy (1.25% versus 0.2%; highest incidence with pancreatic adenocarcinoma)
  - Lung, pancreas, gastric, ovarian and adenocarcinoma of unknown origin
  - Adenocarcinoma - most frequent histological type

LOBO FERREIRA, T., ALVES, R., JUDAS, T., & DELERUE, M. F. (2017). MARANTIC ENDOCARDITIS AND PARANEOPLASTIC PULMONARY EMBOLISM. *BMJ CASE REPORTS, 2017*

24

NBTE

- Pathogenesis: macrophage interaction with malignant cells →cytokine release →endothelial damage → platelet deposition and thrombus formation→ propagated by hypercoagulable state
- Most commonly involves the aortic and mitral valves
- 40%-50% of patients with NBTE present due to embolic phenomena
- 15% of patients with cancer suffer a thromboembolic event

LOBO FERREIRA, T., ALVES, R., JUDAS, T., & DELERUE, M. F. (2017). MARANTIC ENDOCARDITIS AND PARANEOPLASTIC PULMONARY EMBOLISM. *BMJ CASE REPORTS, 2017*

25

NBTE

- H&P, modified Duke's criteria w/ blood cultures, hypercoagulable workup, TTE, brain MRI, though no laboratory tests available to confirm the diagnosis
- Rx- treat the underlying disease process, anticoagulation, follow up echo Q3-6 months, palliative care
- DDx- thrombus, infective endocarditis, fibroelastoma, and Lambl's excrescences

26

Pathogenesis

- The pathogenesis involves macrophage interaction with malignant cells releasing cytokines, which damage endothelium, promoting platelet deposition, and thrombus formation.
- Overactivated coagulation cascade enhances the hypercoagulable state, promoting thrombus growth.
- In this condition, vegetations are sterile, composed of small platelet-fibrin aggregates.
- There is no evidence of inflammatory reaction at the site of vegetation attachment, which makes them easier to detach and embolize

LOBO FERREIRA, T., ALVES, R., JUDAS, T., & DELERUE, M. F. (2017). MARANTIC ENDOCARDITIS AND PARANEOPLASTIC PULMONARY EMBOLISM. *BMJ CASE REPORTS, 2017*

27

Case Presentation

Rebecca T. Hahn

Mark Lebehn

1

History

- Ms XY is a 33 yo woman with a history of PVCs, PACs, mitral valve leaflet prolapse, and mild mitral regurgitation.
  - Treadmill stress ECG terminated in less than 1 minute due to frequent PVCs and NSVT (5 beats).

  Medications: metoprolol ER 100 mg daily

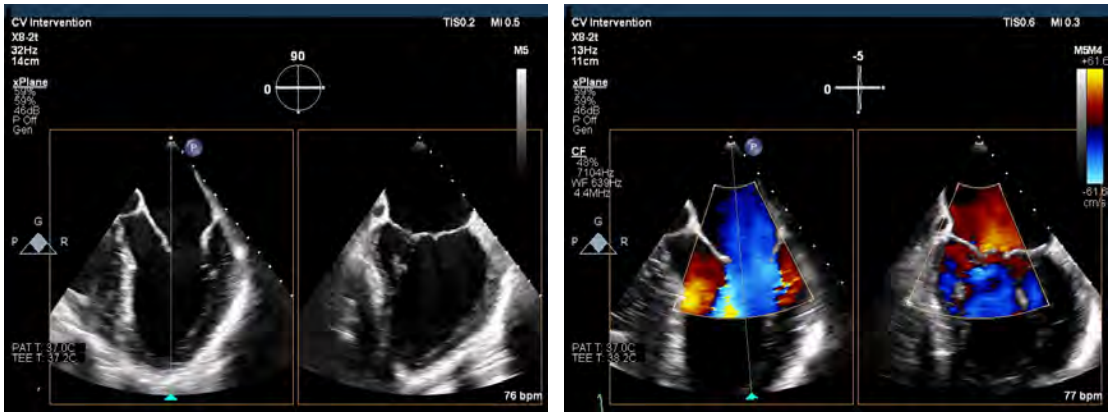
  PSH: None

  FH: Father- CAD; Mother- HLD

  SH: Independent for ADLs. Non smoker. Rare social drinker. Runs a Hebrew School and works in the home.

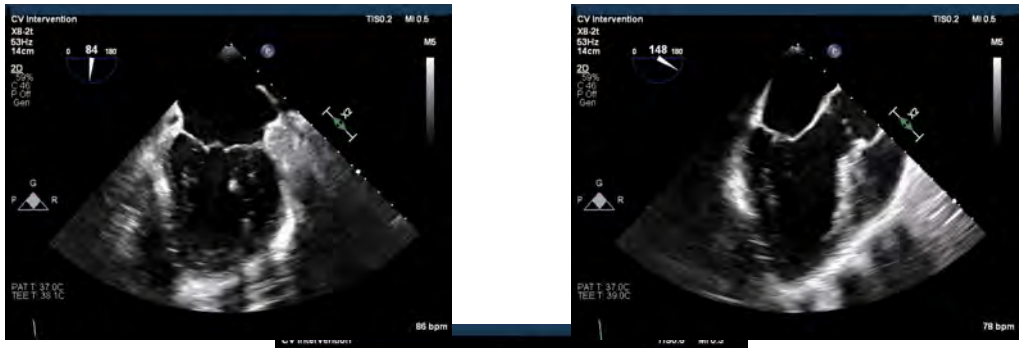
2

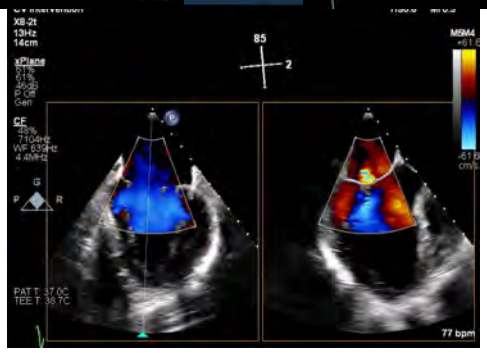
TEE to Assess Severity of MR



What is the etiology of the arrhythmia?

3

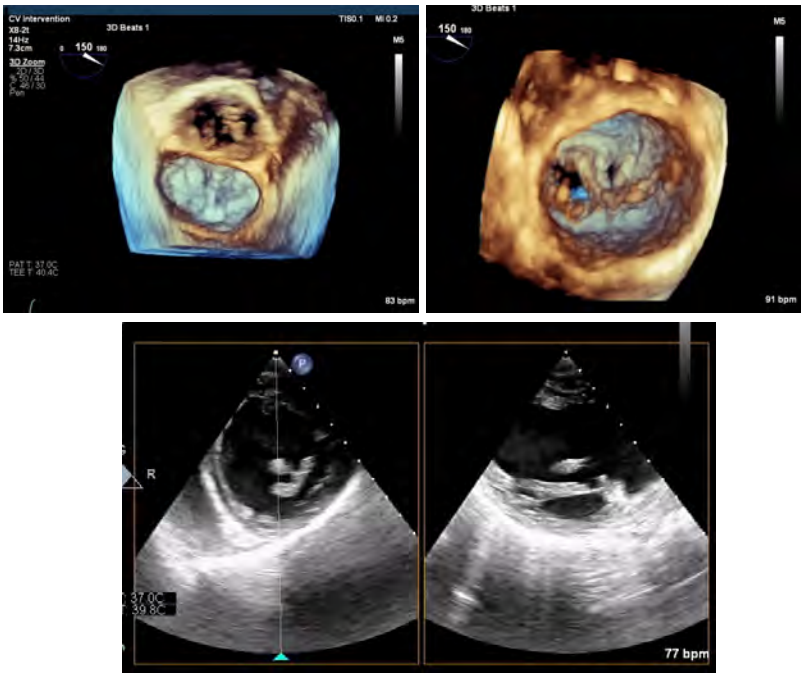




Arrhythmic Mitral Valve Prolapse Syndrome (with Mitral Annular Disjunction)

4

Arrhythmic Mitral Valve Prolapse Syndrome (with Mitral Annular Disjunction)



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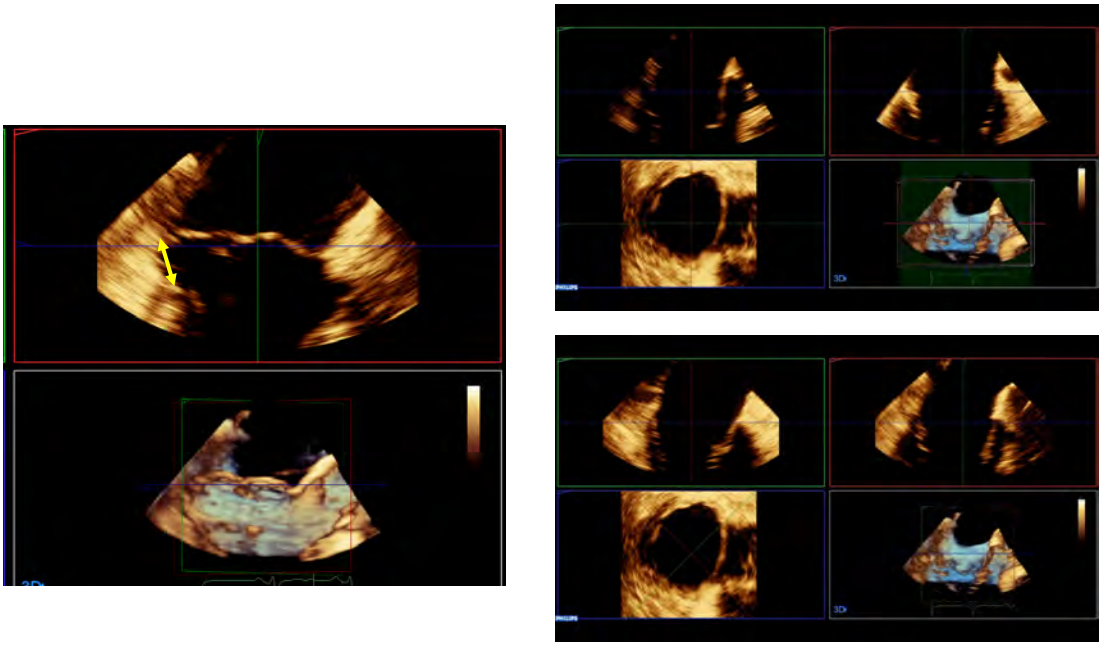

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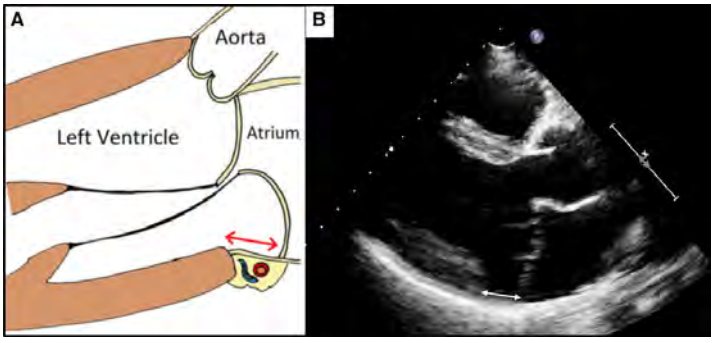
Mitral Annular Disjunction Arrhythmic Syndrome

- 979 patients with MVP with severe MR undergoing surgical repair
- 637 patients with Barlows Disease and 342 with Fibroelastic Deficiency
- MAD in 16% (BD 21.8%, FED 5.8%)
- Most frequent location P1 and P2 scallops
- MAD+ patients were younger, larger MVA dimensions, lower incidence of chordal rupture, higher prevalence of bileaflet prolapse
- MV replacement after failed initial repair in MAD+ 2.8% vs MAD- 6.5%

Mantegazza, V., Tamborini, G., Muratori, M., Gripari, P., Fusini, L., Italiano, G., … Pepi, M. (2019). Mitral Annular Disjunction in a Large Cohort of Patients With Mitral Valve Prolapse and Significant Regurgitation. *JACC: Cardiovascular Imaging*, *12*(11), 2278–2280. doi: 10.1016/j.jcmg.

7

Measurement of MAD



- MAD is measured from the left atrial wall–MV posterior leaflet junction to the top of the LV posterior wall during end-systole (double-headed gray arrow

Basso et al. Circulation. 2019;140:952–964.

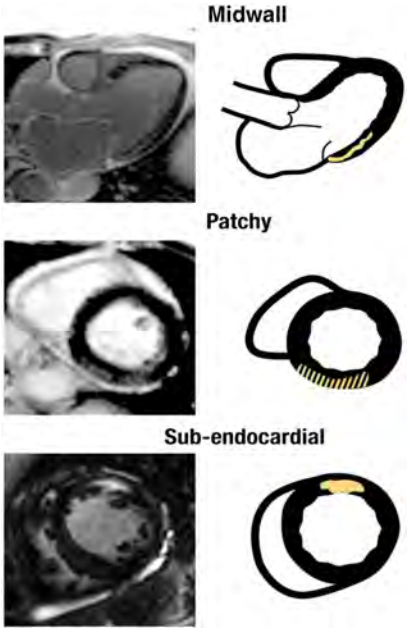
- MAD with disjunction > 8.5 mm was associated with nonsustained ventricular tachycardia (OR 10 95% CI 1.28-78.1).
- Late gadolinium enhancement in anterolateral papillary muscle was strongly associated with serious arrhythmic event (OR 7.35 95% CI 1.15-47.02).

Bennett S, et al . Mitral annular disjunction: A systematic review of the literature. Echocardiography. 2019;36:1549–1558.

8

LGE detected by MR

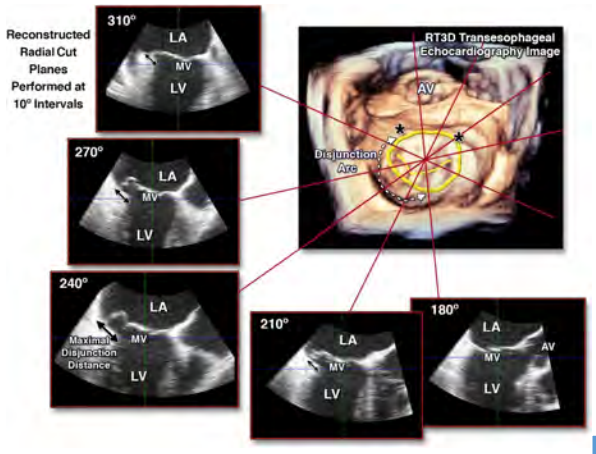
- LV fibrosis was more prevalent in the MVP group than the non-MVP group (36.7% vs. 6.7%; p < 0.001).
- During follow-up (median 1,354 days), MVP patients with LV fibrosis had the highest event rate for arrhythmic events.



Kitkungvan D. et al. J Am Coll Cardiol 2018;72:823–34.

9

RT3DE Datasets of the Annulus



- There is annular disjunction **(doubled arrows)** spanning circumferentially from 210° to 310° (i.e., disjunction arc degree = 100°).
- The maximal disjunction distance, defined as the maximal separation between the atrial wall–MV attachment and the basal LV musculature, is 10 mm.
- The disjunction index, calculated as the product of the disjunction arc degree and the maximal disjunction distance, is 100° × 10 mm = 1,000° · mm.

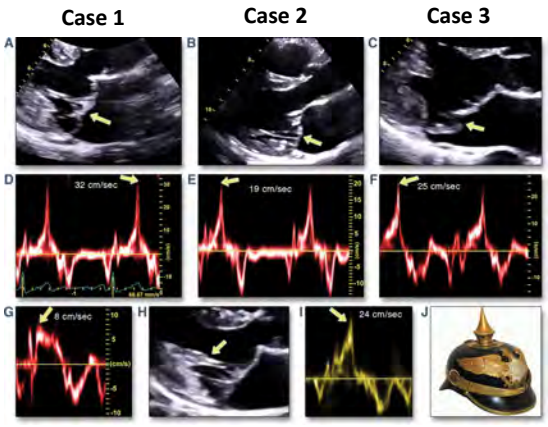
Yellow line depicts the true atrial–ventricular junction.
Asterisks indicate the fibrous trigones

Most common location is P2 and P1 (less common P3).

Lee AP, et al. JACC Cardiovasc Imaging. 2017 Dec;10(12):1424-1433.

10

Pickelhaube Sign

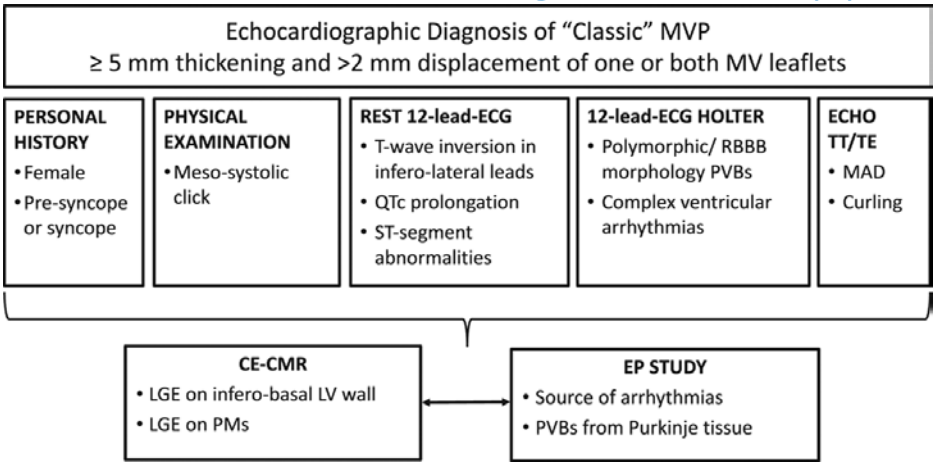


- TTE demonstrating myxomatous bileaflet MVP **(arrows)**
- High-velocity mid-systolic spikes on tissue Doppler (lateral annulus) at different times in systole
- G represents a normal medial annulus tissue Doppler

The Pickelhaube Sign: Novel Echocardiographic Risk Marker for Malignant Mitral Valve Prolapse Syndrome.
Muthukumar L, Rahman F, Jan MF, Shaikh A, Kalvin L, Dhala A, Jahangir A, Tajik AJ.
JACC Cardiovasc Imaging. 2017 Sep;10(9):1078-1080. doi: 10.1016/j.jcmg.2016.09.016. Epub 2016 Dec 21.

11

Clinical Profile and Diagnostic Tools For Risk Stratification and Targeted Therapy



Basso et al. Circulation. 2019;140:952–964.

12

January 22nd, 2020

Case Presentation: Is This Severe AS or Pseudo AS (Echo and CT Imaging)

REBECCA T. HAHN, MD, FACC
PROFESSOR OF MEDICINE
COLUMBIA UNIVERSITY COLLEGE OF MEDICINE
@hahn_rt

1

Case

Clinical History	
Age	83 years
Gender	Male
Medical history	Heart Failure, HTN, Hypercholesterolemia S/P MI, MidLAD and RCA Stent
Rx	ASA 81 mg QD Metoprolol ER 50 mg QD Furosemide 40 mg QD Spironolactone 37.5 mg QD Atorvastatin 20 mg QD

Heart Failure NYHA class III, ACC/AHA stage C

Creatinine 0.81 mg/dL

NT pro-BNP 9 952 pg/mL

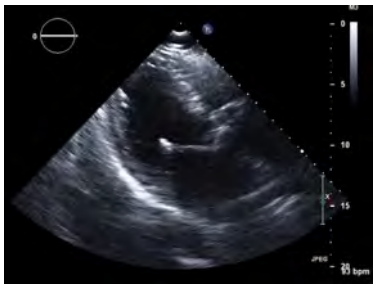
ECG: Sinus rhythm

Physical Examination
Height (cm): 177, Weight (kg): 70 BSA 1.87 m^2
BP 124/70; HR 68 bpm
No jugular venous distention Soft S2 III/VI SM RUSB Clear lungs No edema

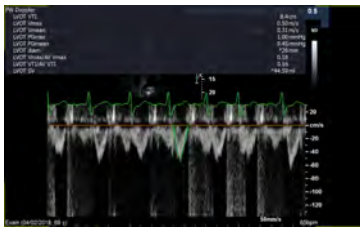
2

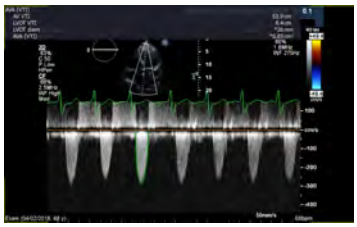
Baseline TTE

Echo Variable (TTE/TEE)	Measure
Jet Velocity (m/s)	2.98
Mean Gradient (mmHg)	21.3
Calculated AVA (cm^2)	0.83
Calculated AVA index (cm2/m2) **1.87 m2** **BMI = 22.3 kg/m^2**	0.46 (severe)
DVI	0.16
TTE LVOT TTE annulus diameter	2.55 cm 2.62 cm
Ejection Fraction (%)	35%
LV Stroke Volume (ml)	44.6 ml 24 ml/m^2
Severity of AR	1-2+
Severity of MR	1-2+
RV Pressure (mmHg)	38 mmHg



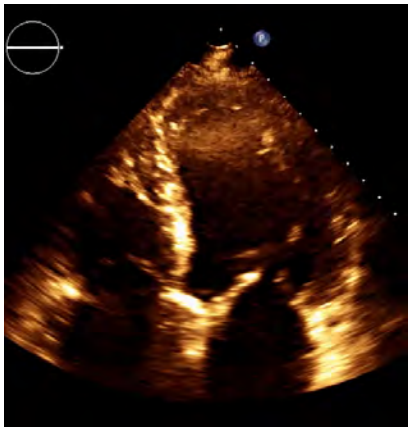
Very severe calcification of the aortic valve

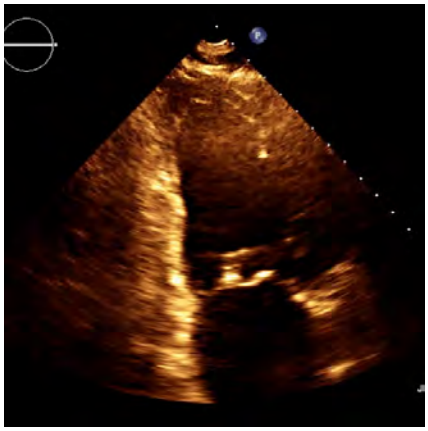




3

Baseline EF 35%





S/P RCA and LAD stents

4

Stages of Valvular AS

Stage	Definition	Valve Anatomy	Valve Hemodynamics
D1	Symptomatic severe high-gradient AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic Vmax ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically is ≤1.0 cm2 (or AVAi ≤0.6 cm2/m2) but may be larger with mixed AS/AR
D2	Symptomatic severe low-flow/low-gradient AS with reduced LV EF	Severe leaflet calcification with severely reduced leaflet opening	• AVA ≤1.0 cm2 with Aortic Vmax <4 m/s or mean ΔP <40 mm Hg • Dobutamine stress echocardiography shows AVA ≤1.0 cm2 with Vmax ≥4 m/s at any flow rate
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification with severely reduced leaflet opening	• AVA ≤1.0 cm2 with Aortic Vmax <4 m/s or mean ΔP <40 mm Hg • AVAi ≤0.6 cm2/m2 and • Stroke volume index <35 mL/m2 • Measured when patient is normotensive (systolic BP <140 mm Hg)

Nishimura, RA et al JAmCollCardiol 2014 Jun 10;63(22):2438-88

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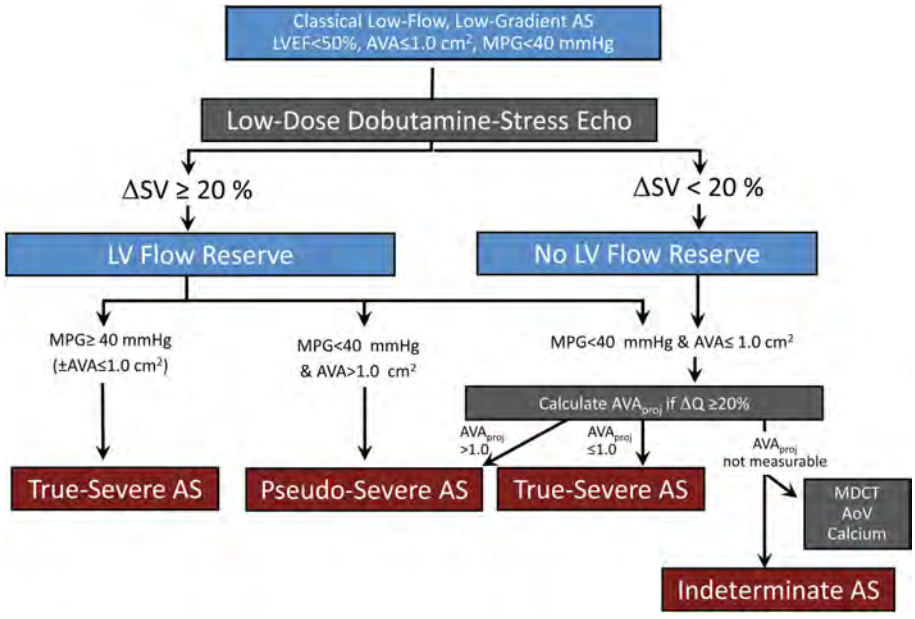

Lancellotti, P et al. J Am Soc Echocardiogr 2017;30:101-38

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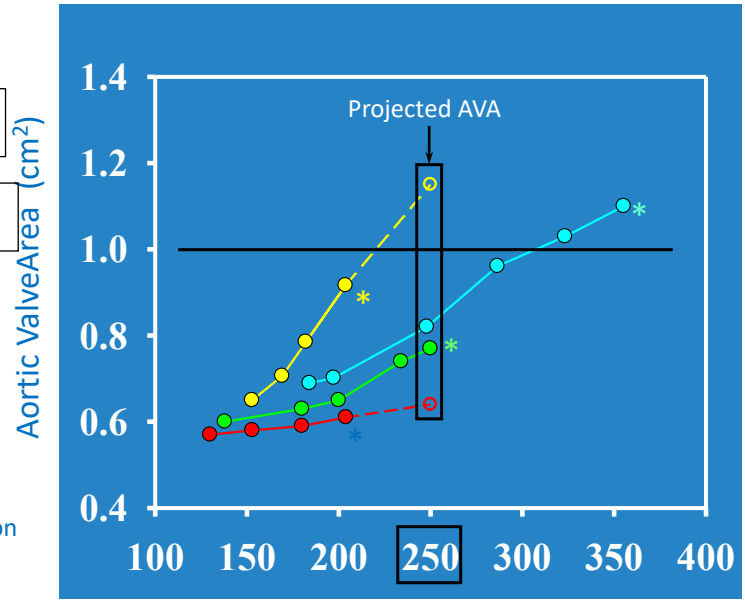
Concept of the Projected AVA (250 mL/s)

Simplified method:
Slope=ΔAVA/ ΔQ

AVAproj=
AVA_{rest}+slope(250-Q_{rest})



Slide courtesy of Philippe Pibarot

* Peak AVA during DSE

Blais et al, Circulation 2006;113:711-721

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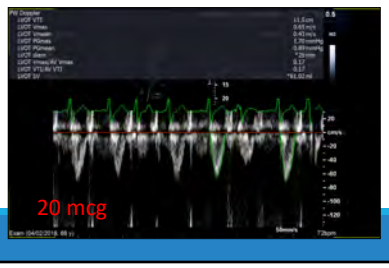
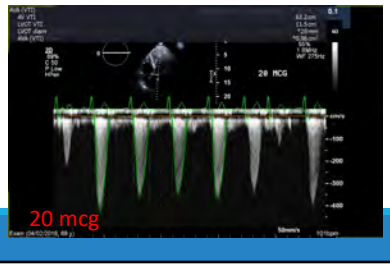
Dobutamine Stress Case 2

	LVOT VTI (cm)	SV (ml)	ET (msec)	Flow (ml/s)	AV Pk Vel (m/s)	AV Mn Grad (mmHg)	AV VTI (cm)	DVI	AVA (cm^2)
Rest	8.4	44.6	264	169	2.98	21.3	53.9	0.16	0.83
5 mcg	9.7	51.5	264	195	3.08	20.2	53	0.18	0.96
10 mcg	9.9	52.6	235	223	3.07	21.0	51	0.19	1.00
20mcg	11.04	56.4	232	**253**	3.52	23	55.4	0.20	1.02



31% increase in stroke volume



AVAi = 0.545

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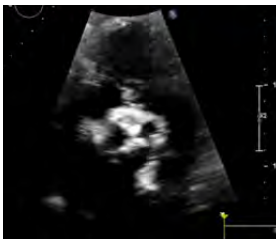
D2: Low Flow, Low EF

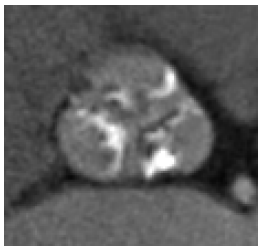
Baseline	
Peak gradient	35.5 mmHg
Mean gradient	21.3 mmHg
AVA	**0.83 cm^2 (0.44 cm^2/m^2)**
DVI	0.16
SV	44.6 ml (24 ml/m^2)
LVEF	25%

Peak dose (20 mcg/kg/min)	
Peak gradient	49 mmHg
Mean gradient	23 mmHg
AVA	**1.02 cm^2 (0.55 cm^2/m^2)**
DVI	0.20
SV	58.6 ml (31.3 ml/m^2)
LVEF	30%

31% increase in stroke volume with dobutamine
Severe aortic stenosis by indexed AVA



Subcostal SAX View



Calcium Score = 2750 AU

	Women	Men
AVC	**1,274 AU**	**2,065 AU**
AVC density	**292 AU/cm2**	**476 AU/cm2**

Clavel MA et al. J Am Coll Cardiol. 2013; *62*:2329–2338.

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Guidelines Indications for AVR in Classical Low-Flow, Low-Gradient AS

Stage D2 Definition: AVA≤1.0 cm^2, Mean gradient<40 mmHg, LVEF<50%

Guidelines	Recommendation for AVR	Class
ACC-AHA 2014/2017	AVR is reasonable in symptomatic patients with low LVEF, low-flow/low-gradient severe AS **with a DSE that shows a mean gradient ≥40 mm Hg with an AVA ≤1.0 cm^2** at any dobutamine dose	**IIa**
ESC-EACTS 2017	AVR should be considered in symptomatic patients with low LVEF, low-flow/low-gradient **severe AS (mean gradient ≥40 mmHg) with flow reserve on DSE**	**I**
ESC-EACTS 2017	AVR may be considered in symptomatic patients with low LVEF, low-flow/low-gradient **severe AS without flow reserve on DSE, particularly when CT calcium scoring confirms severe AS**	**IIa**

Vahanian et al. EHJ 2012 *Nishimura, Otto et al. JACC 2014*

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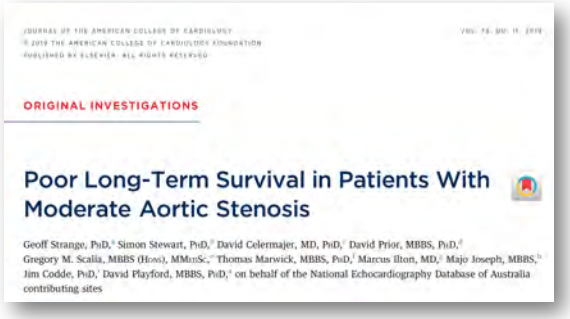
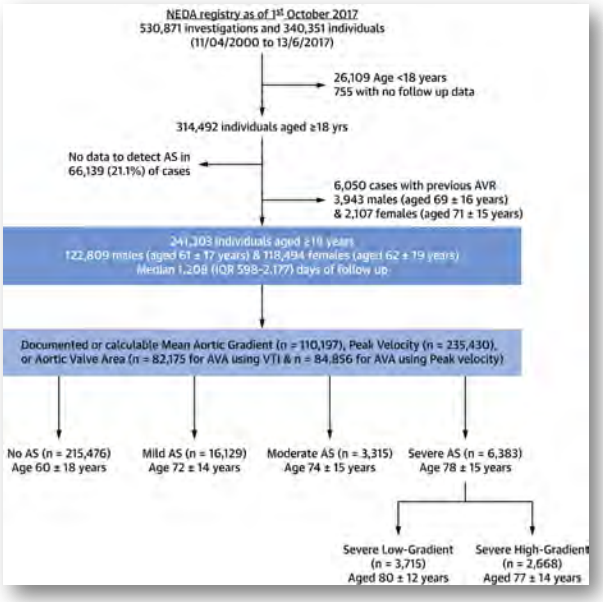


ORIGINAL INVESTIGATIONS

Poor Long-Term Survival in Patients With Moderate Aortic Stenosis

Geoff Strange, PhD,[a] Simon Stewart, PhD,[b] David Celermajer, MD, PhD,[c] David Prior, MBBS, PhD,[d] Gregory M. Scalia, MBBS (Hons), MMedSc,[e] Thomas Marwick, MBBS, PhD,[f] Marcus Ilton, MD,[g] Majo Joseph, MBBS,[h] Jim Codde, PhD,[i] David Playford, MBBS, PhD,[a] on behalf of the National Echocardiography Database of Australia contributing sites

National Echocardiographic Database of Australia (NEDA), with the capacity to individually link echocardiographic findings with long-term mortality, in a large, unselected patient group



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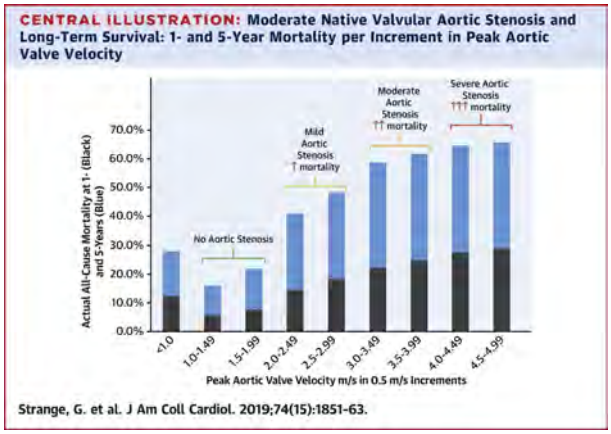
Moderate Aortic Stenosis



Strange, G. et al. J Am Coll Cardiol. 2019;74(15):1851-63.

- In this large analysis of survival across the full spectrum of native valve AS severity, we found high rates of mortality associated with both moderate and severe AS during long-term follow-up
- Individuals presenting with a mean AV gradient >20.0 mm Hg or peak AV velocity >3.0 m/s (or DVI 0.25-0.3) had a high risk of dying in the longer term that was similar to the risk in patients presenting with severe AS at baseline.

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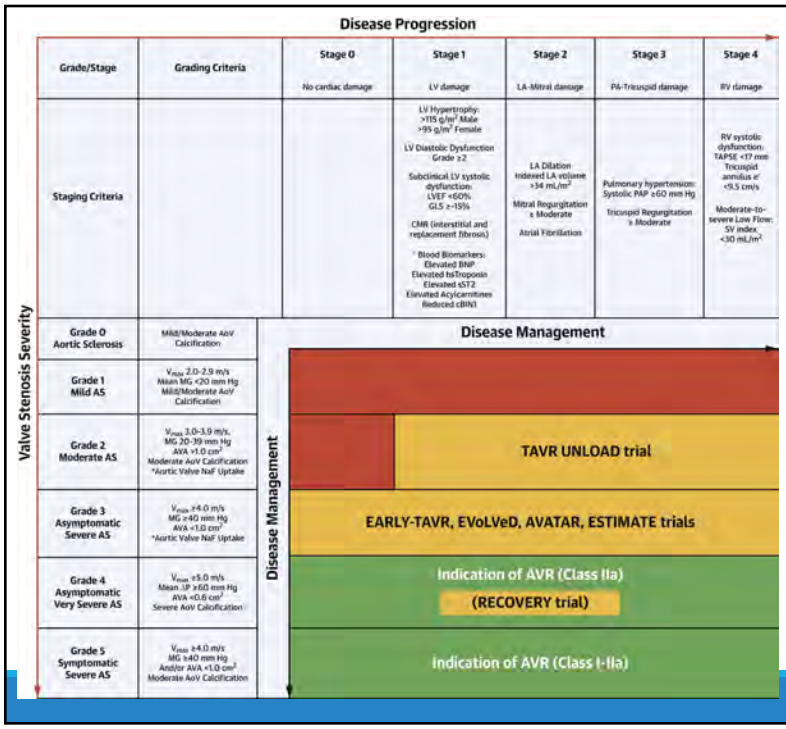

Algorithm for Treatment os AS

Red indicates conservative (follow-up) management.
Yellow indicates no evidence yet for intervention, but ongoing trials will be instructive.
Green represents current guideline-based indications for intervention in AS

Vannan M et al. J Am Coll Cardiol 2019;74(15);1864-67

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