

CASE PRESENTATION – RUPTURE, OR NOT?

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CASE PRESENTATION 1

- 40 yo male
- Smoker
- No former medical Hx
- Transient angina in the past few weeks
- At the morning of his admission day felt pericardial CP
- BP 120/70, P 72, Sat 100% RA, no fever
- Troponin 56

ECG





TTE ADMISSION

- Severe segmental LV systolic dysfunction EF=30%
- Septo-Apical Akinesis
- Large apical thrombus
- Preserved RV function
- Small circumferential Pericardial Effusion, no Echo signs of tamponade

QUESTIONS CASE 1

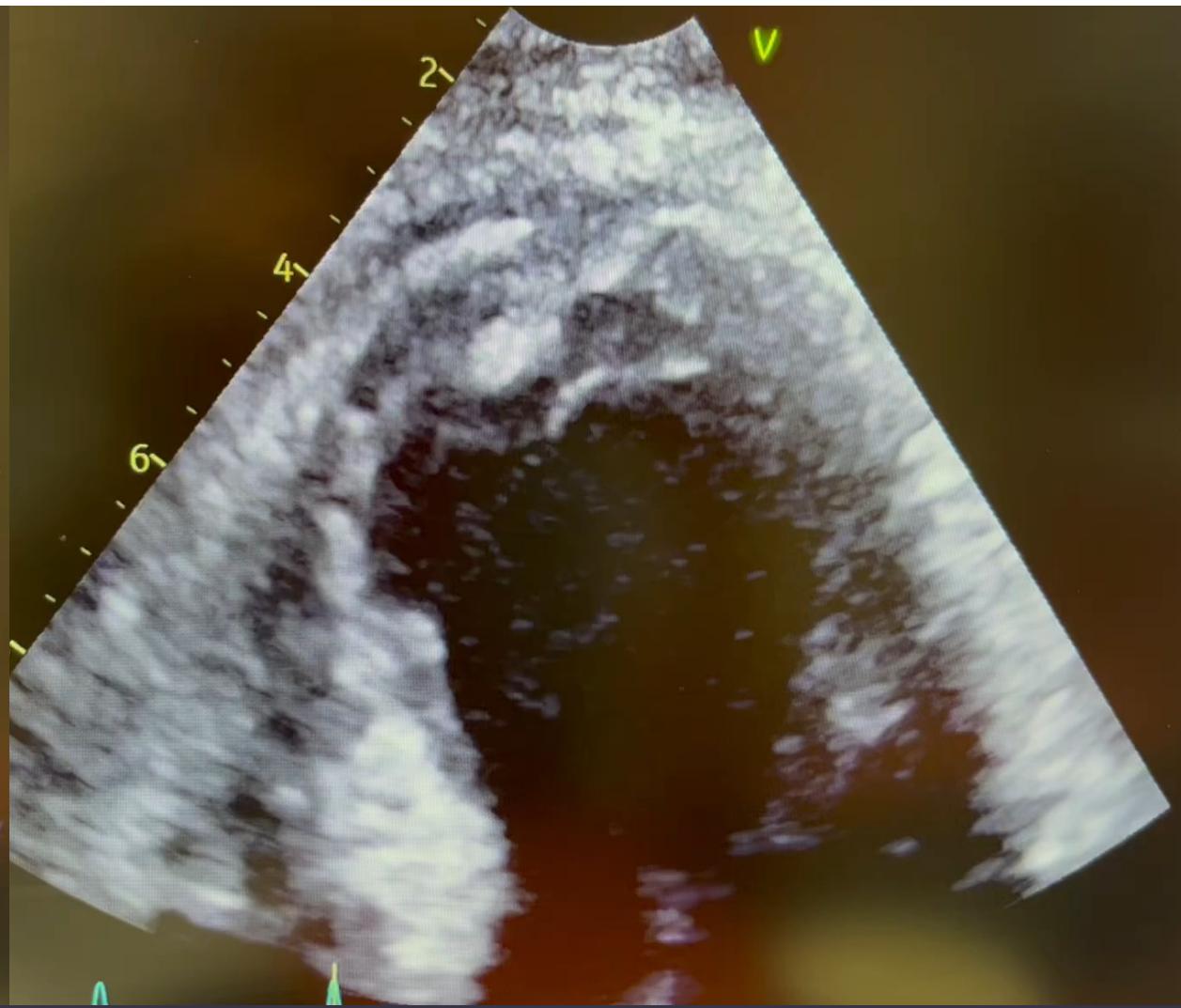
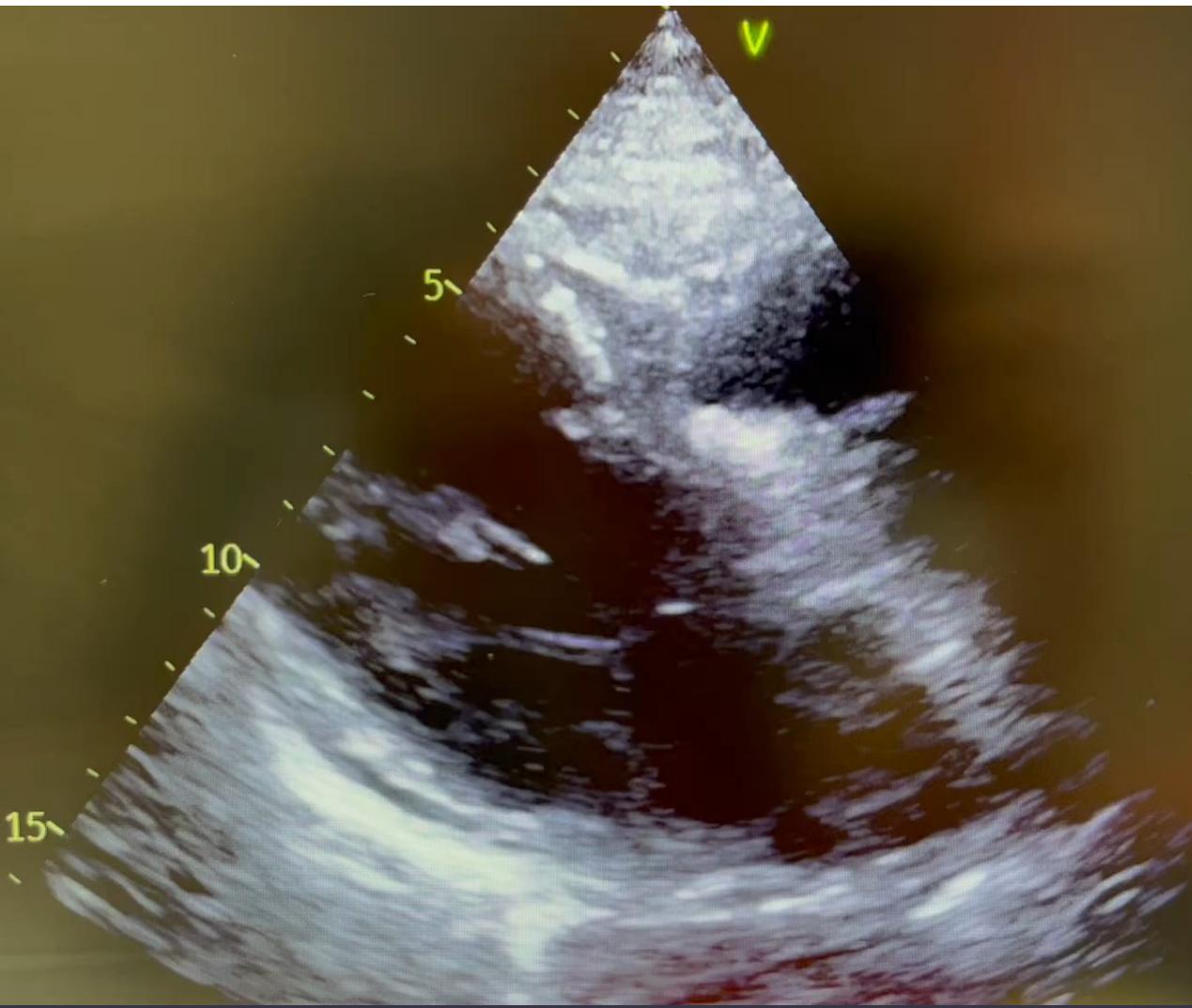
- What is the diagnosis?
- What is the etiology for the presentation?
- What is the next step?

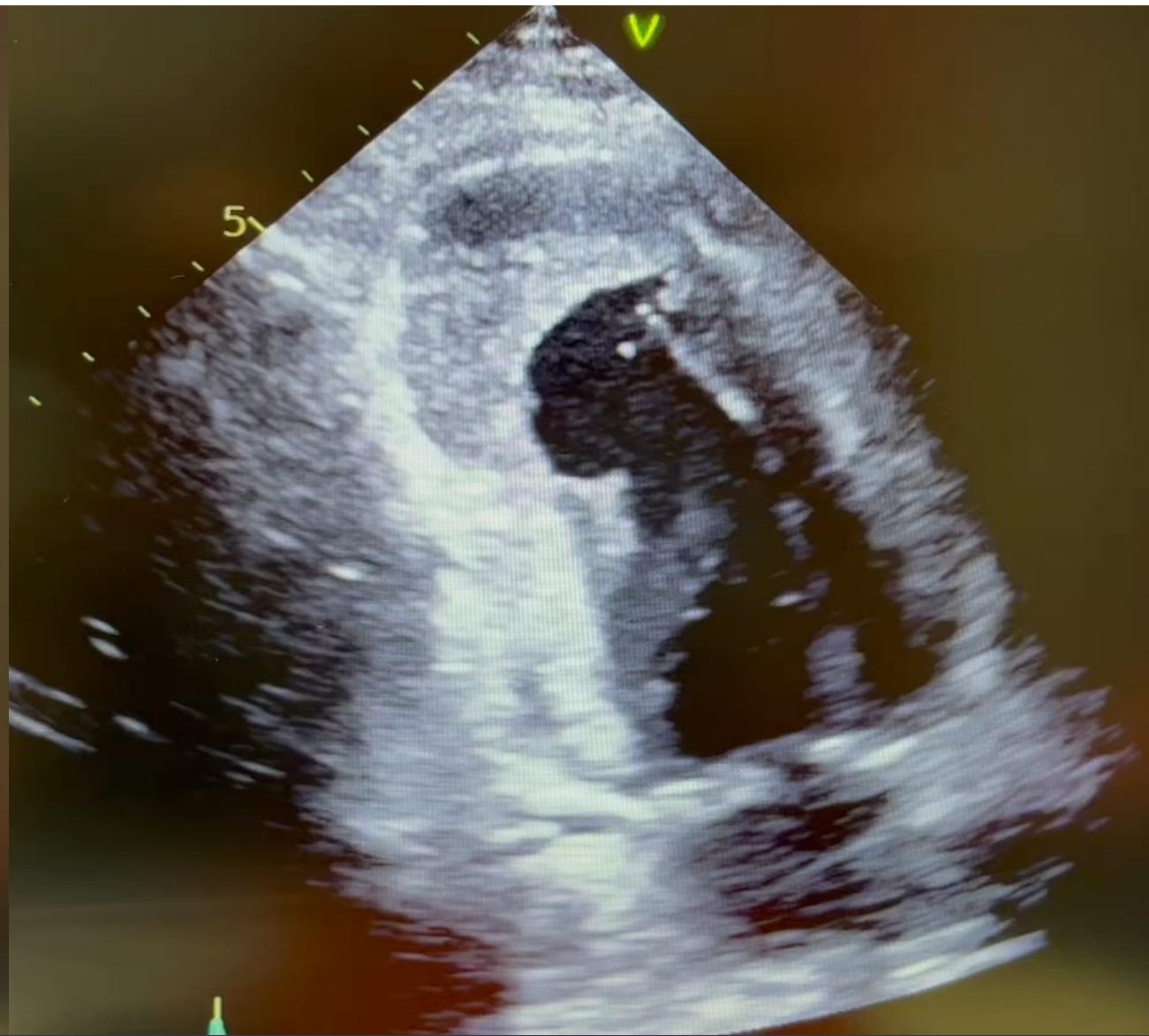
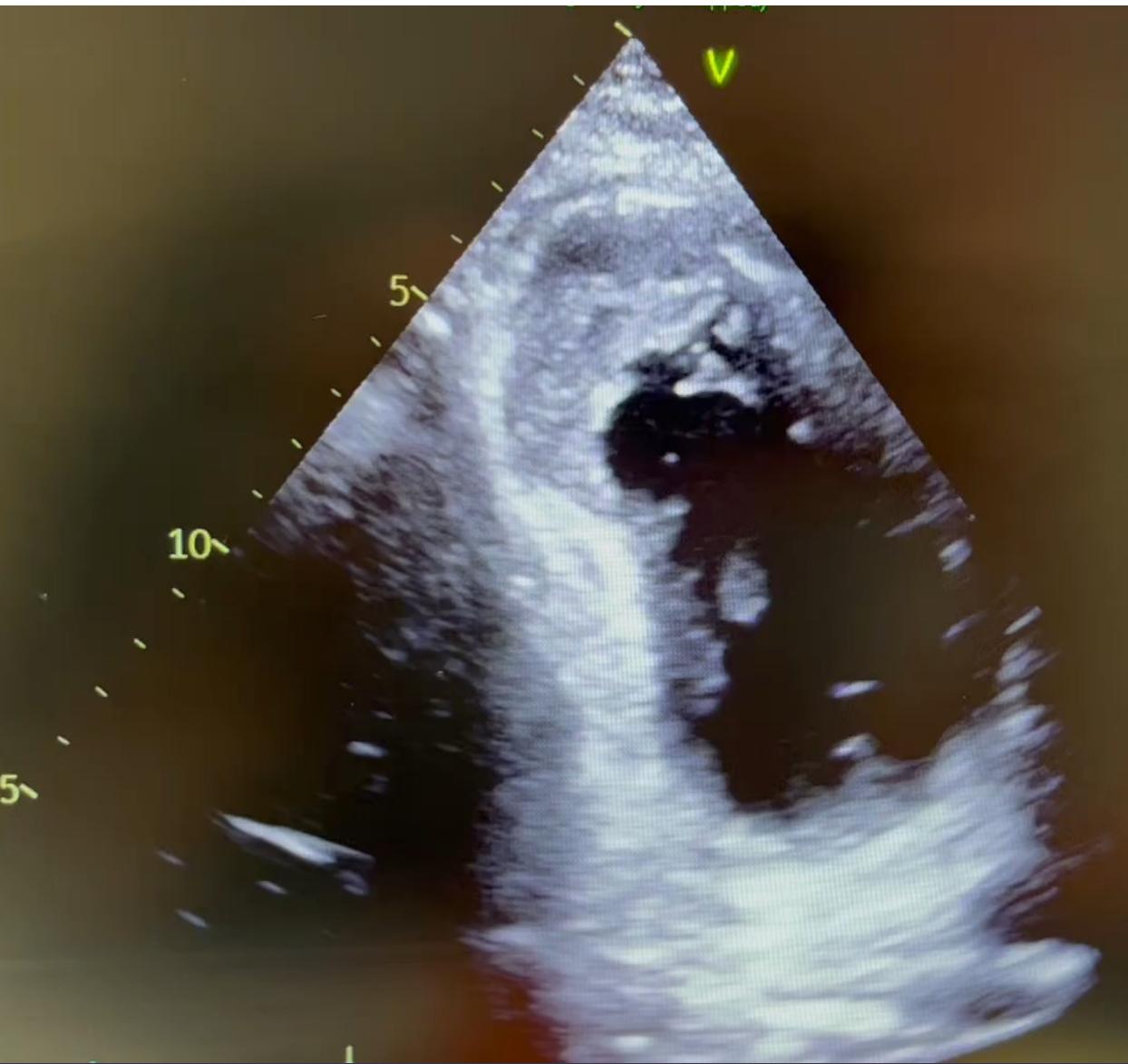
CASE PRESENTATION

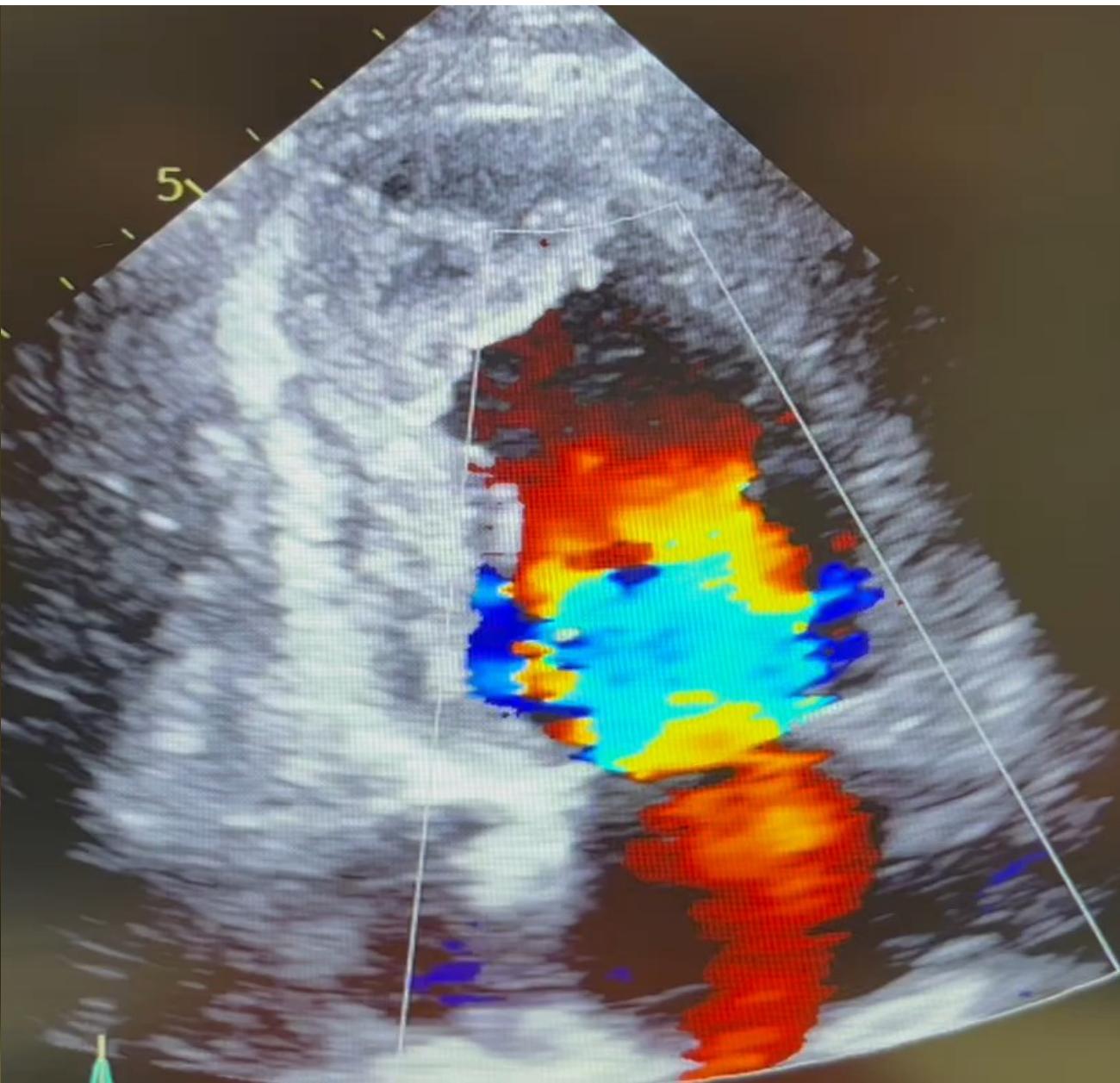
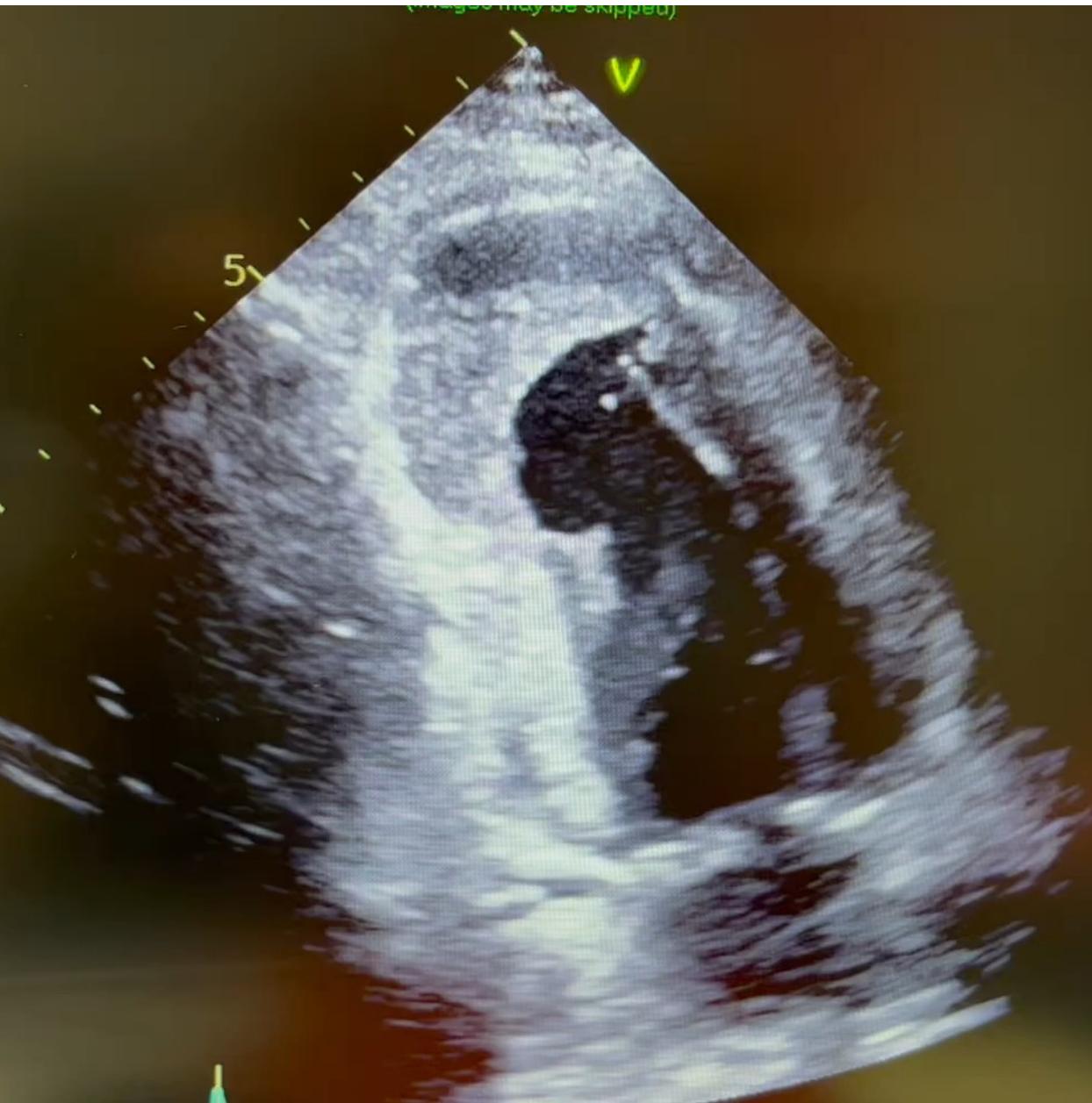
- Late Arrival of Ant. STEMI
- CP d/t Dressler synd.
- Large apical thrombus that mandates anticoagulation
- AL to monitor pericardial effusion under Heparin IV
- Treated in ICCU with beta blockers, Ace-I, DAPT

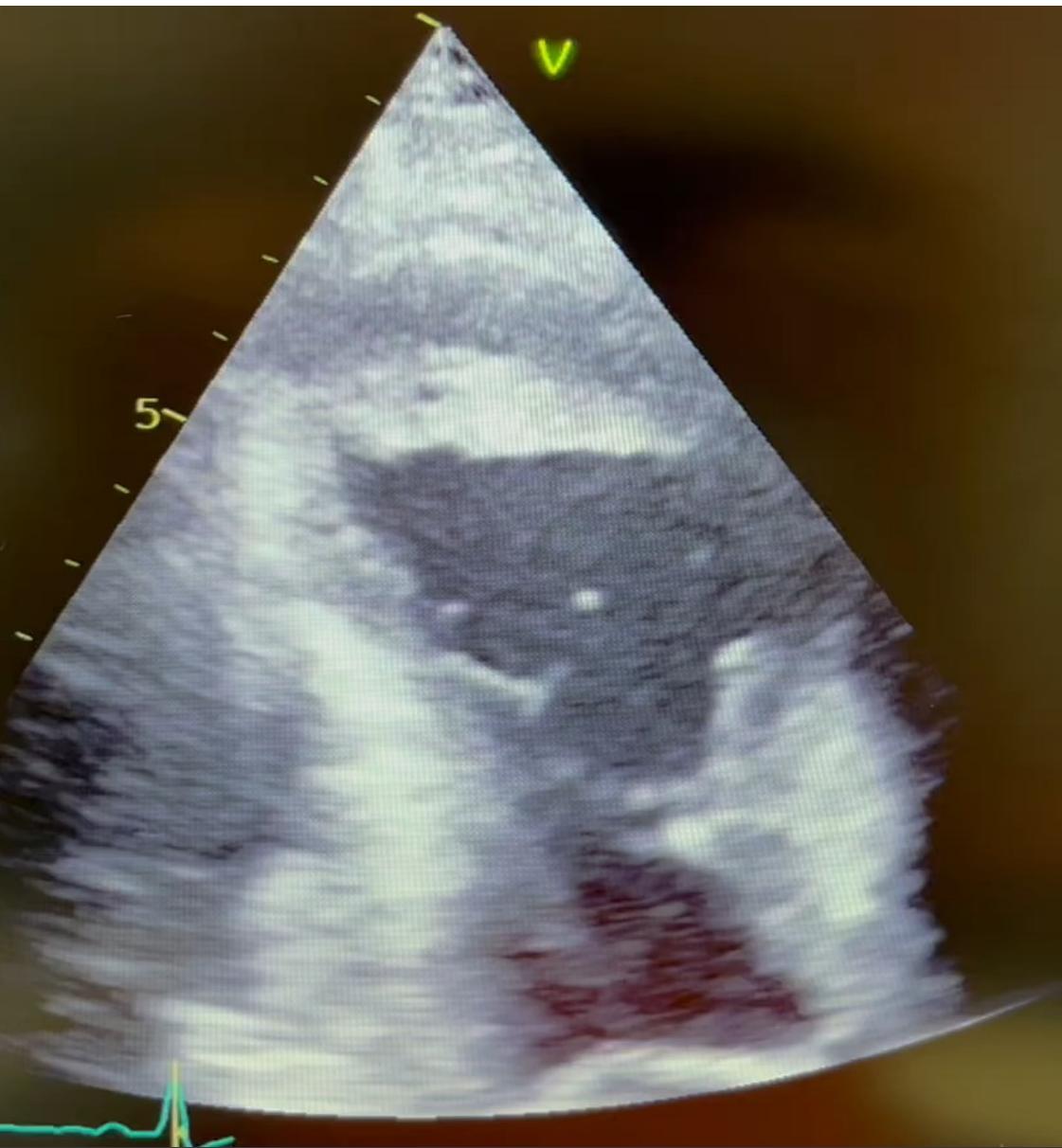
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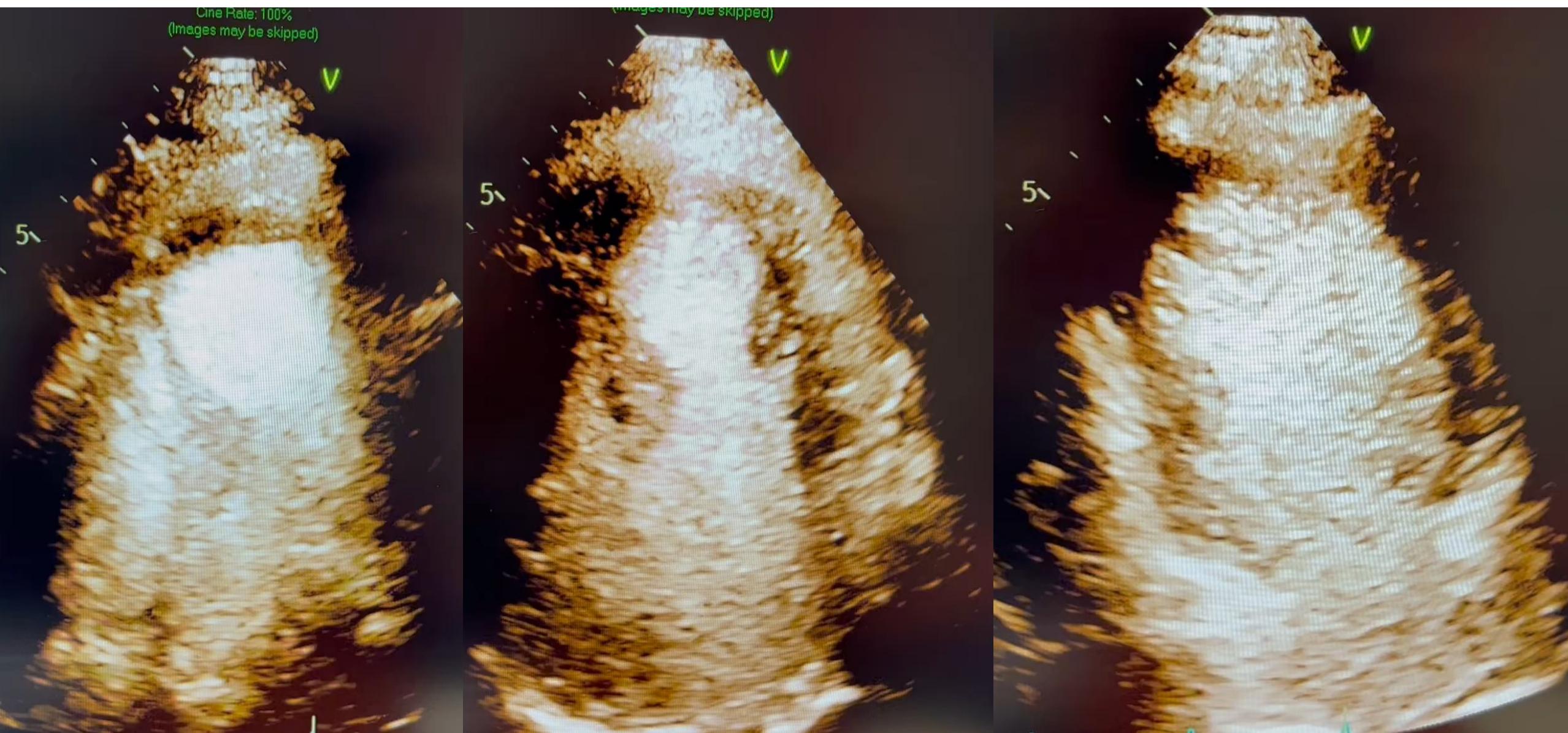
- After 48h:
 - CATH: 1VD occluded MID LAD, no PCI
 - Repeated TTE:







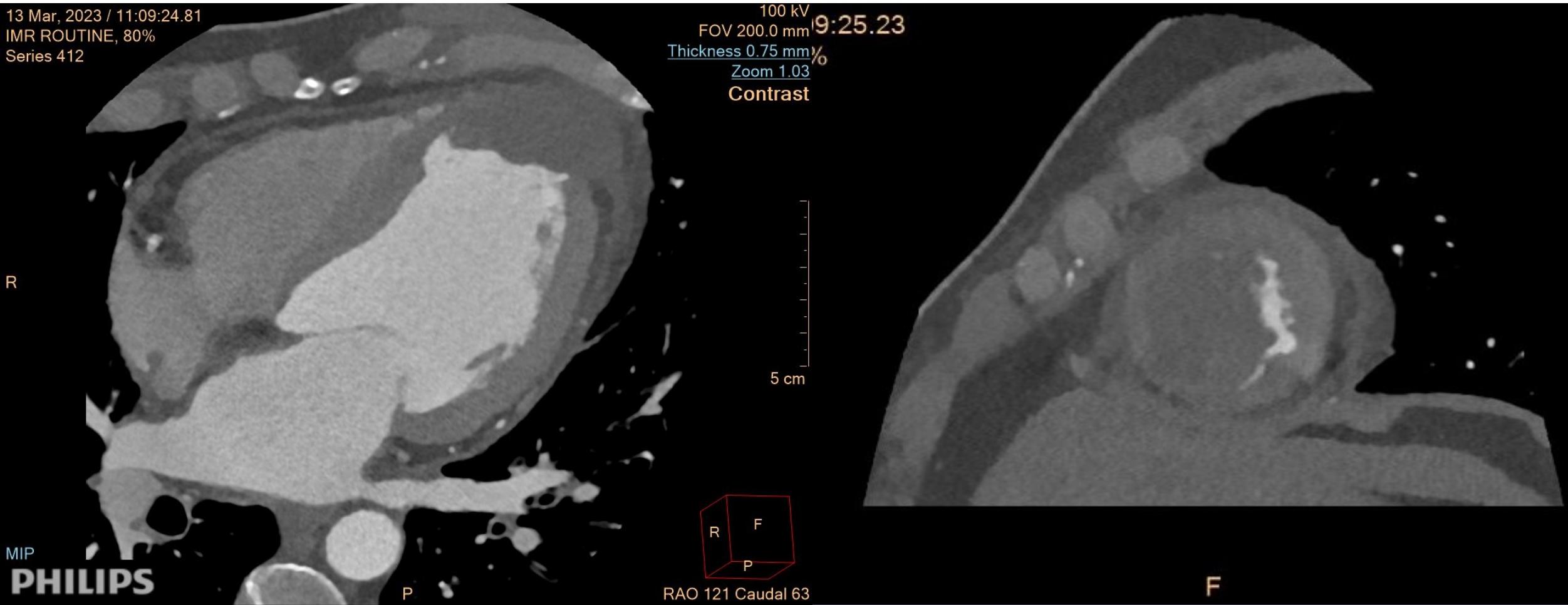




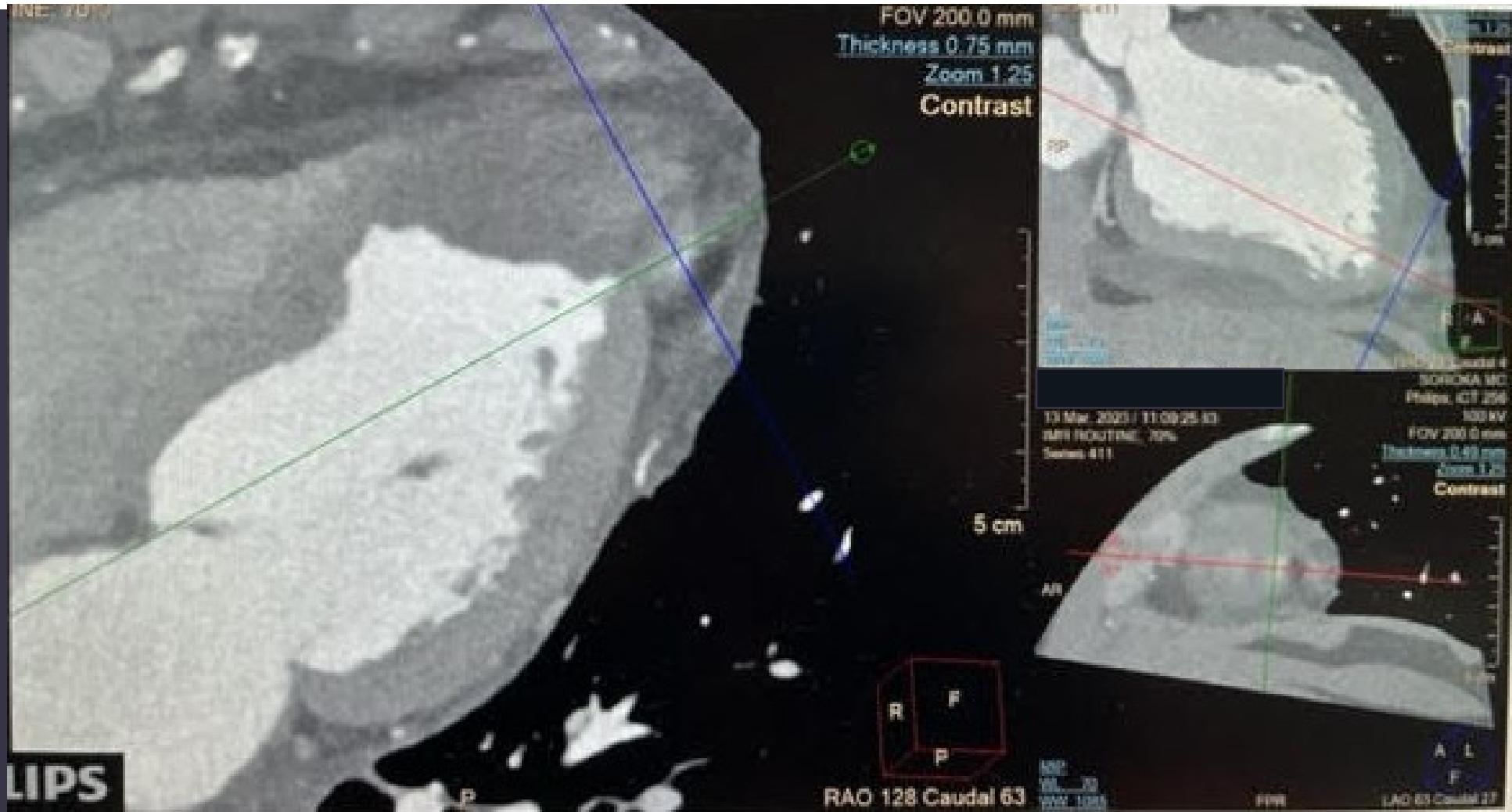
TEE DAY 2

- Contained Apical Rupture???
- Urgent CCTA:

CCTA

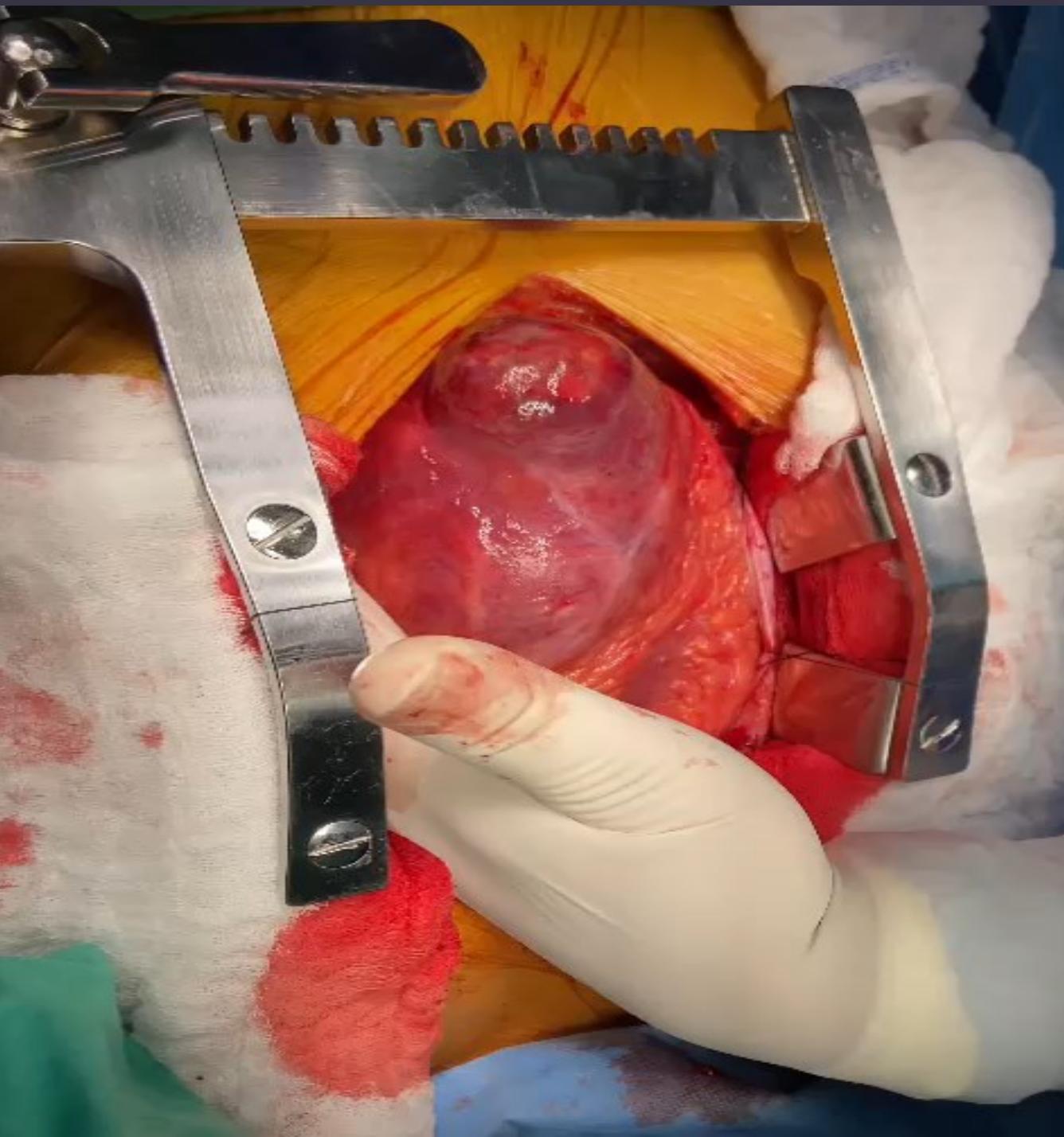


CCTA



CASE PRESENTATION 1- CCTA

- Moderate LV systolic dysfunction
- Apical thrombus 3.4X3.7X3.7 cm
- Pseudo aneurysm neck 4mm with contrast media inside the pericard → M/P Contained rupture



CASE 2

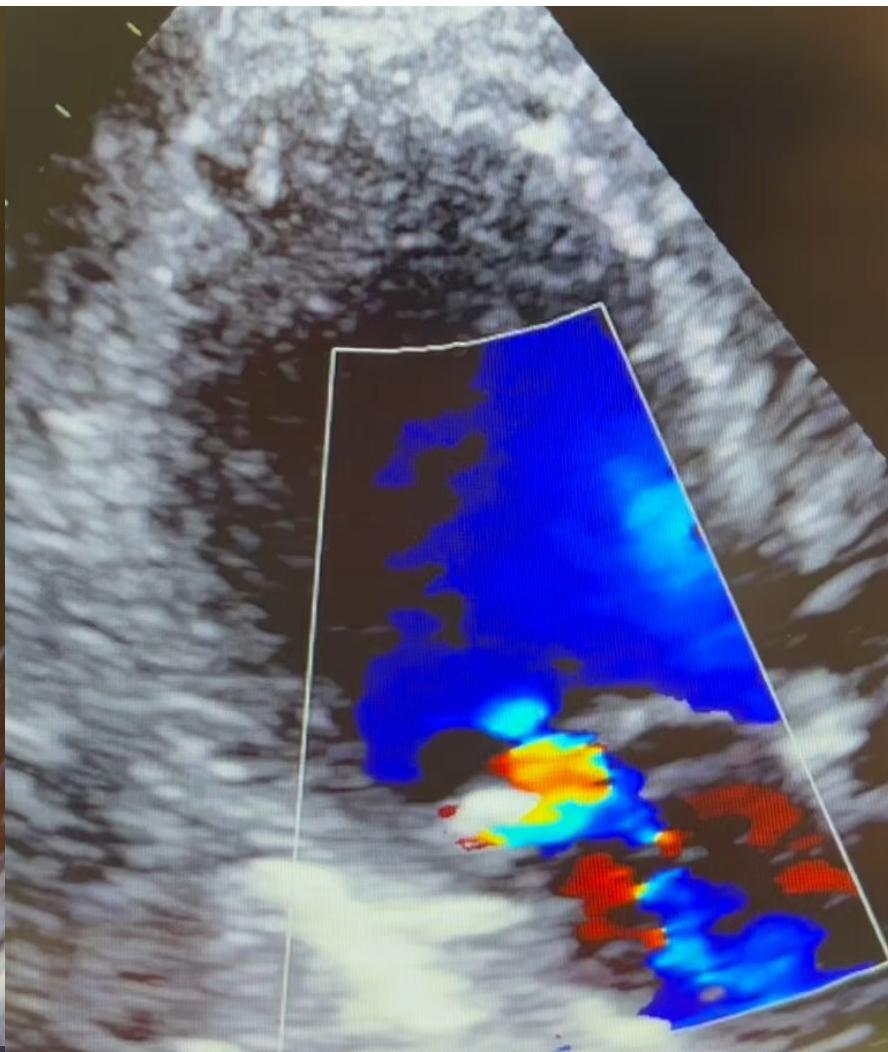
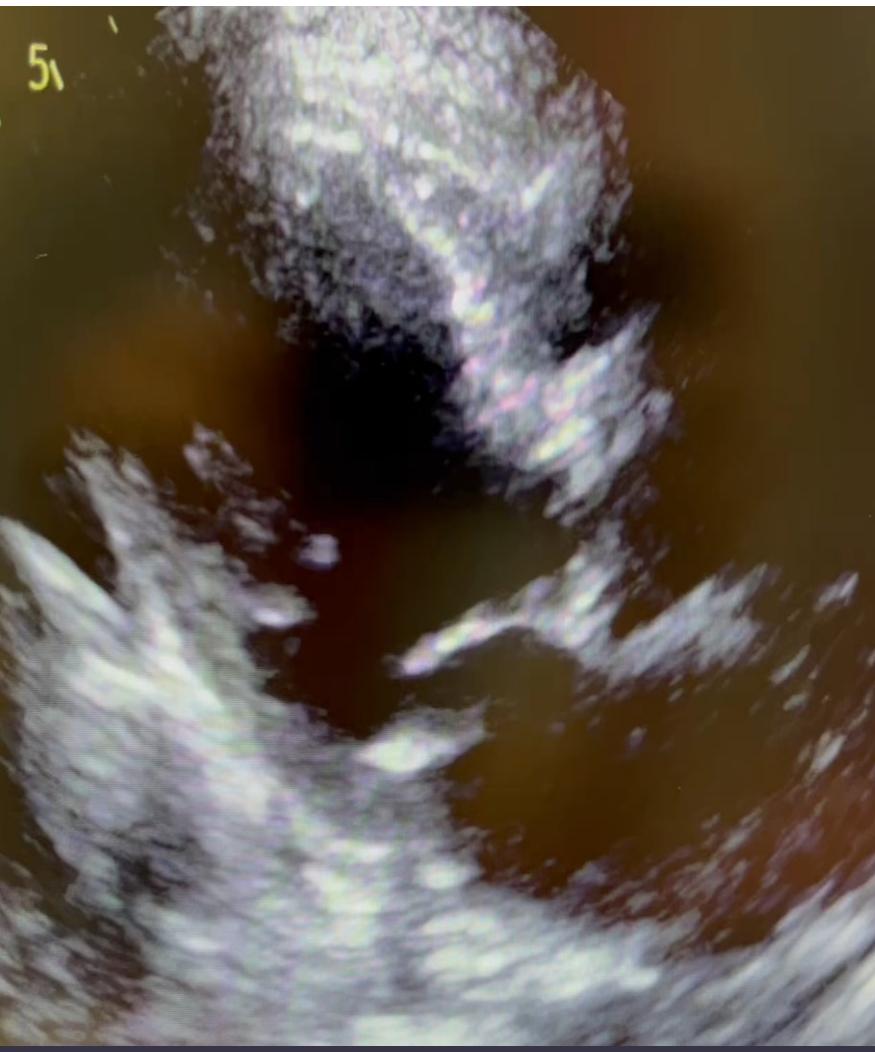
- 79 yo male
- CIHD, HTN, DM, Dyslipidemia
- S/P STEMI 2007 LAD+RCA, EF=45-50%
- Admitted d/t CP with pulmonary edema after 1m of effort dyspnea
- Troponin = 1300

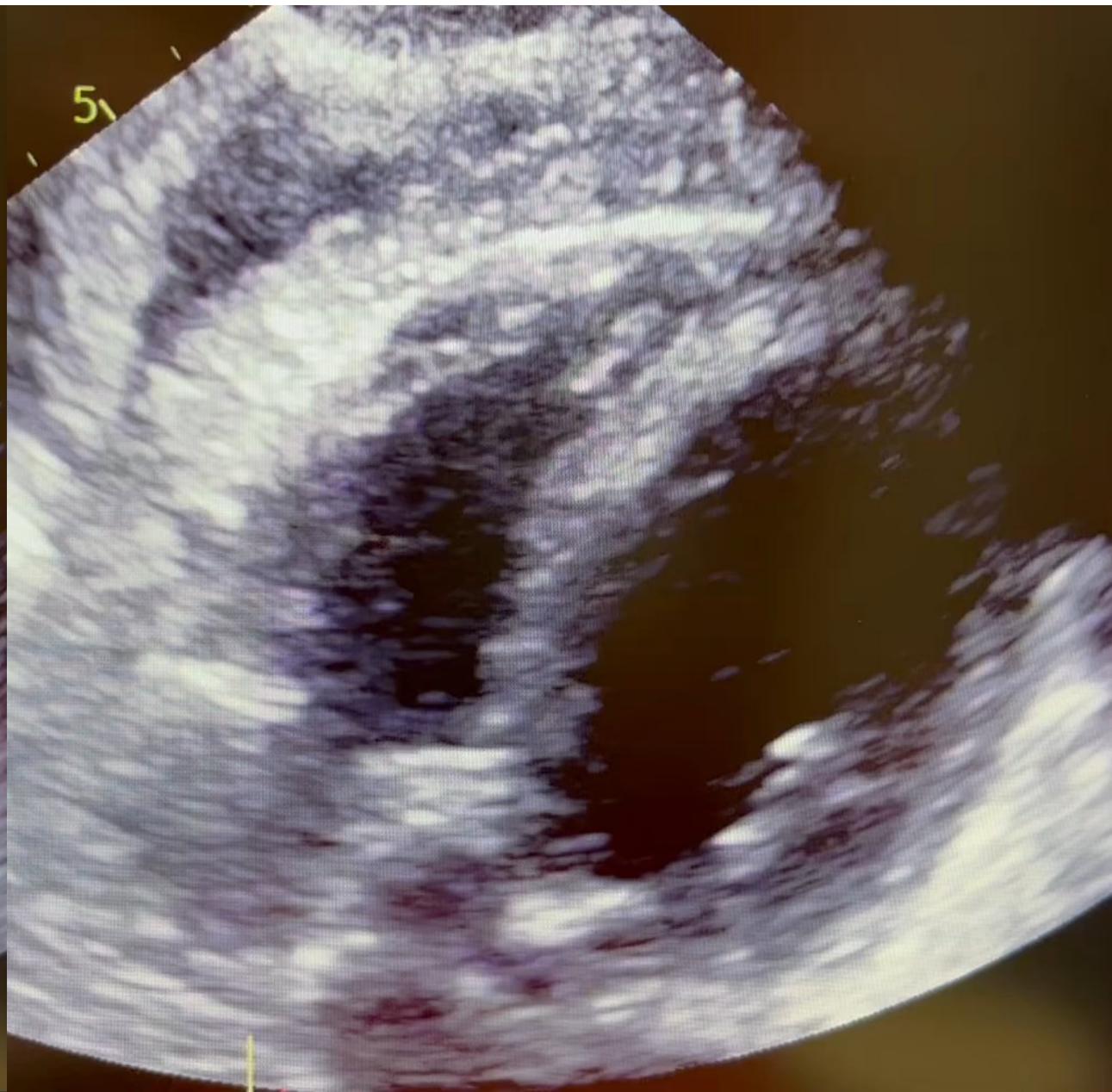
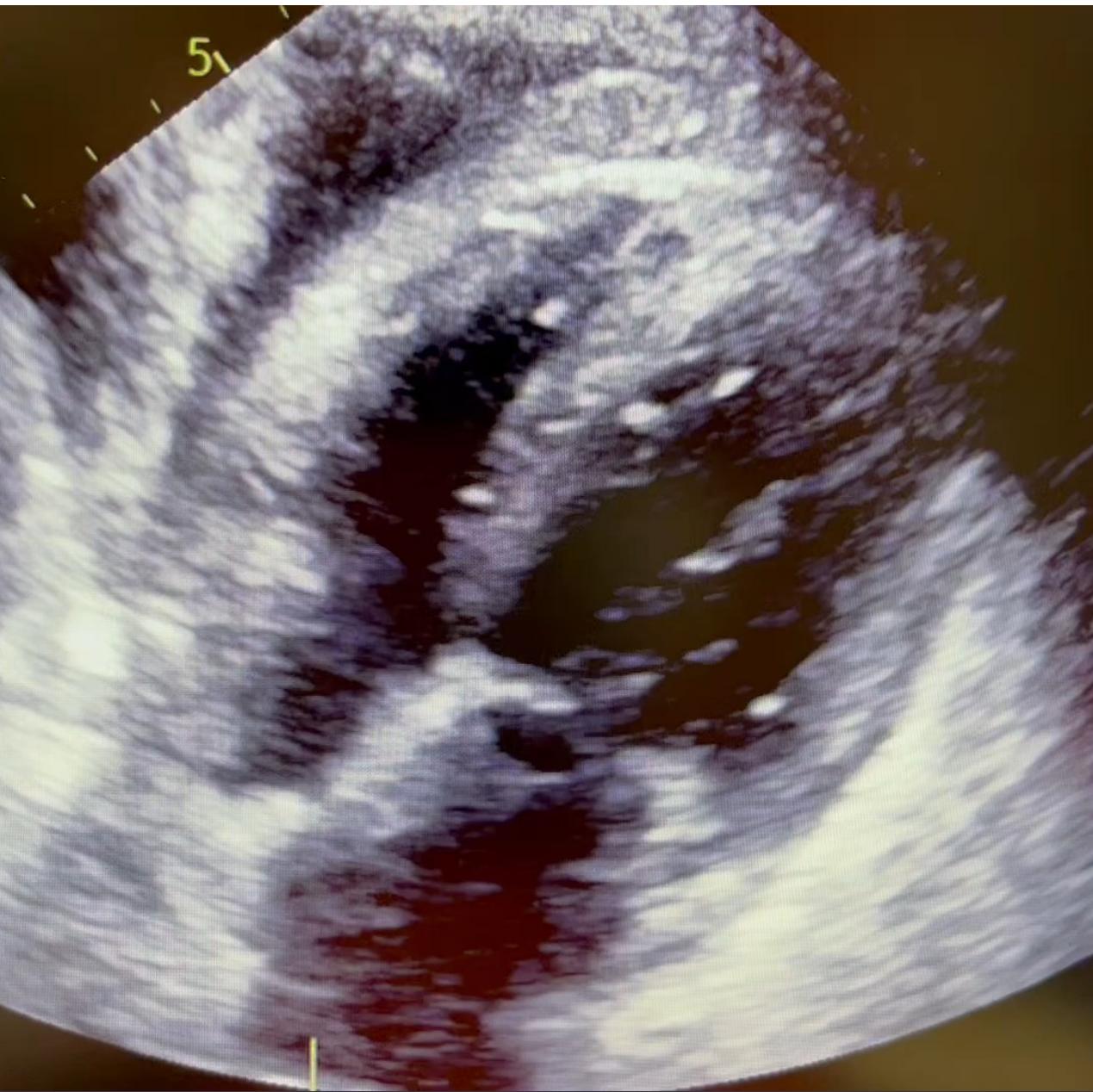
ECG 2



CASE 2

- PPCI: 3VD – RCA 100%, LAD ISS 95%, LCX + M1 90%
- Attempted PCI to RCA – failed – probably CTO
- No symptoms





TTE CASE 2

- EF = 20%
- Moderate to Severe Functional MR
- Anterior pericardial effusion with clot / fibrin on RV free wall?

OR + CTICU

- Porcelain Aorta → CABG on beating heart
- **NOTHING IN THE PERICARD OR ON RV FREE WALL!**
- ARF + Acidosis
- ECMO + HEMOFILTRATION
- Severe refractory shock

CASE 3

- 71 yo male
- HTN, CVA with no residual damage, Gout, CRF
- Admitted d/t CP + dyspnea over 48h
- Troponin 8000+

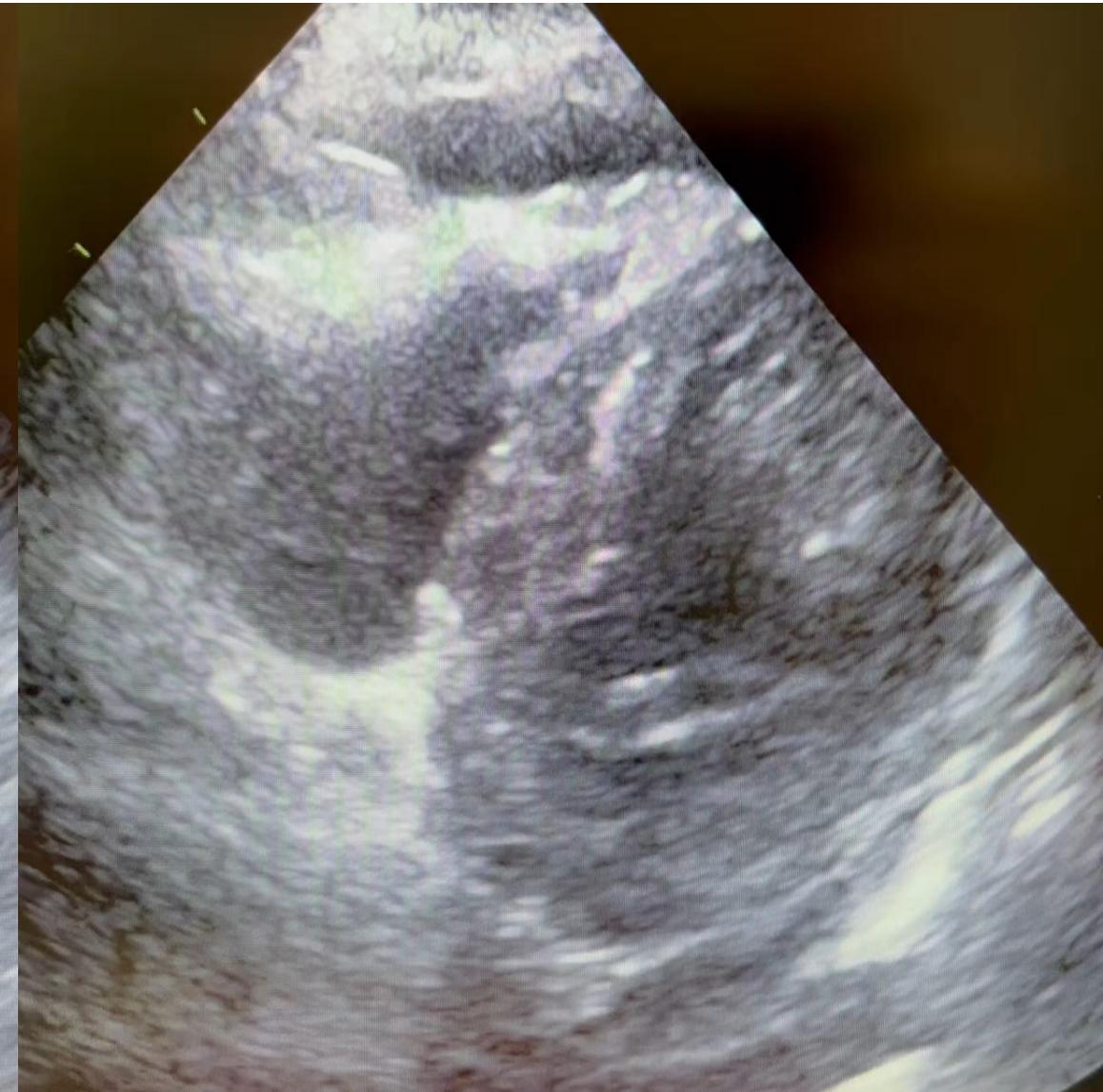
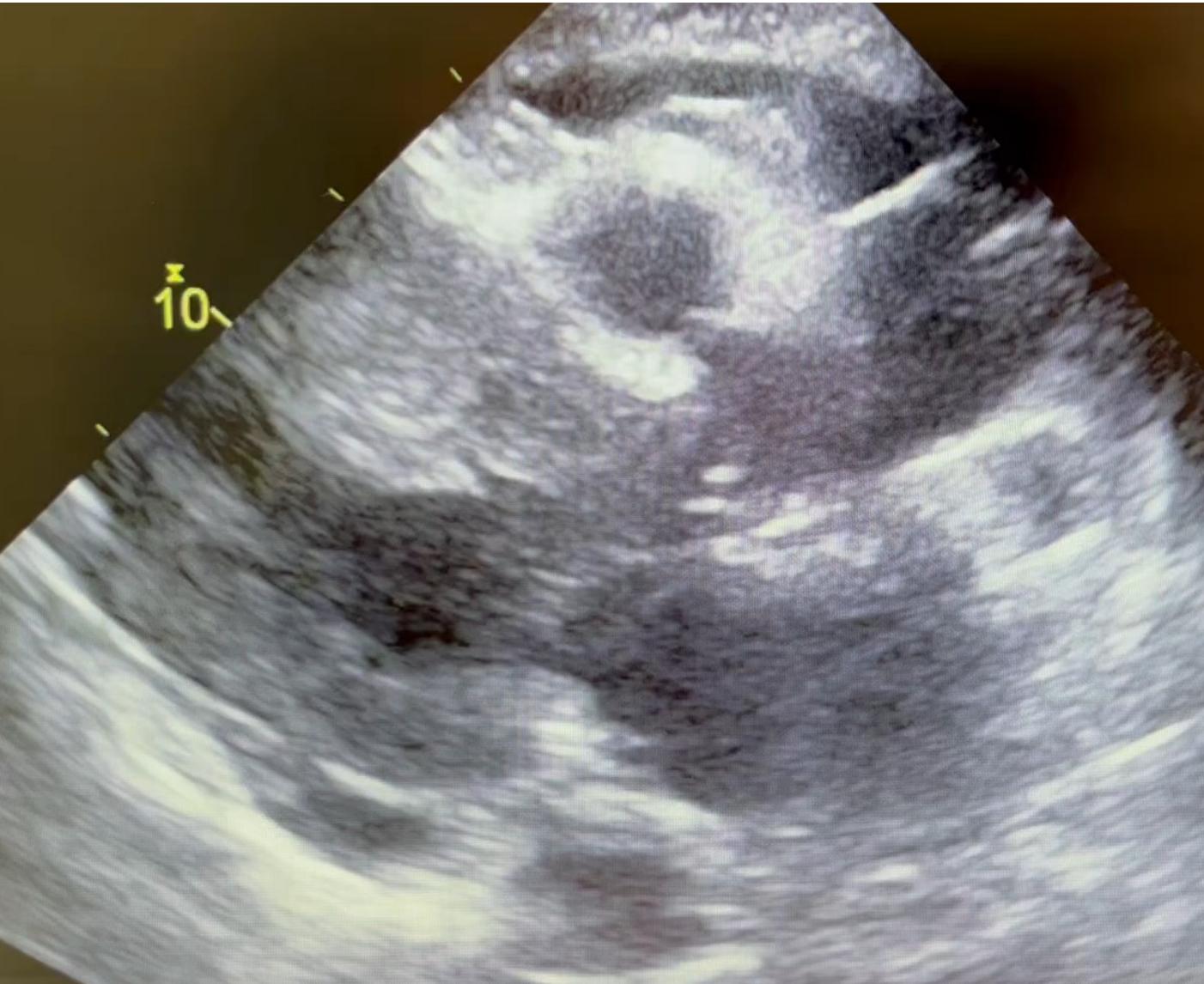
ECG

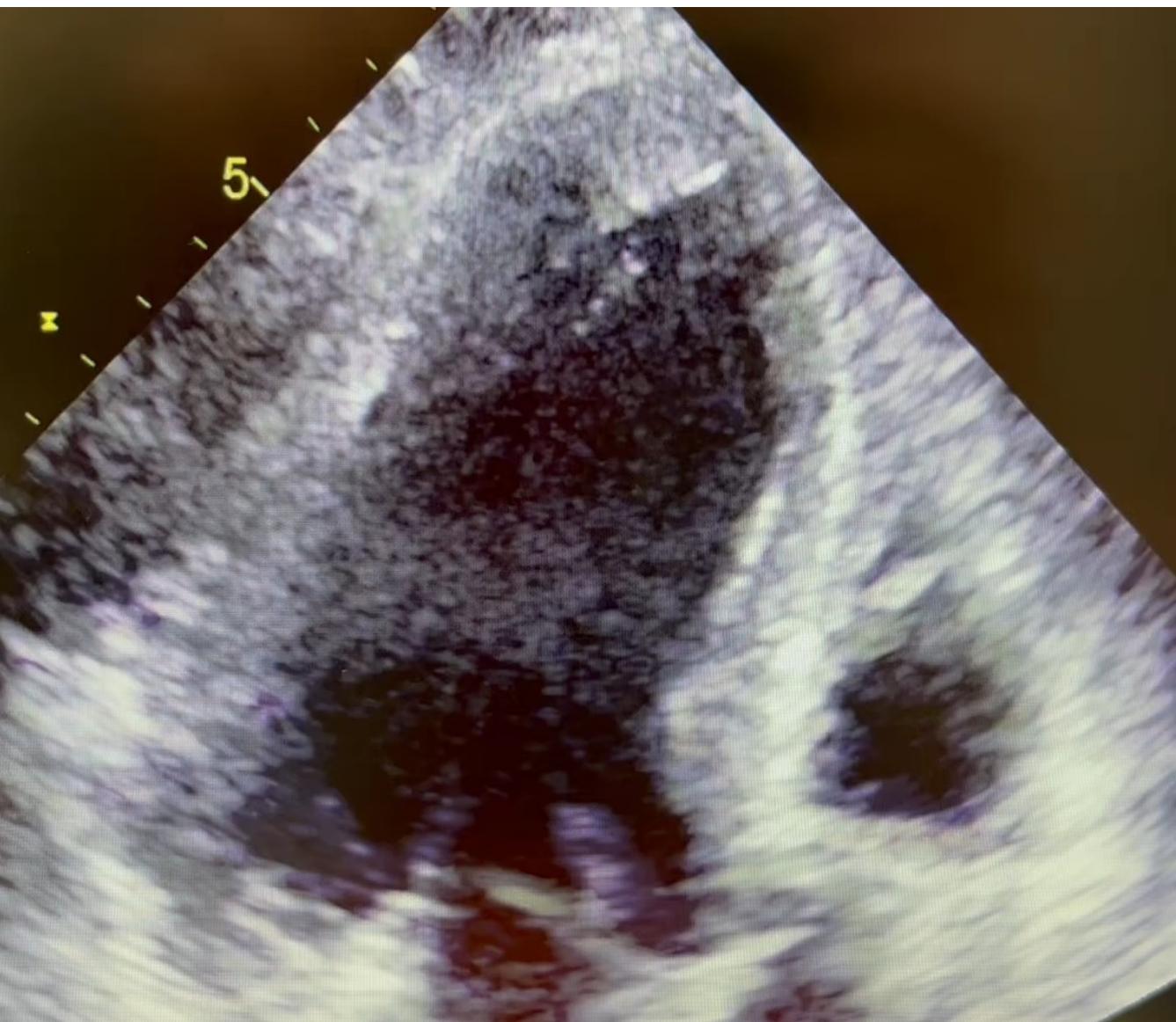
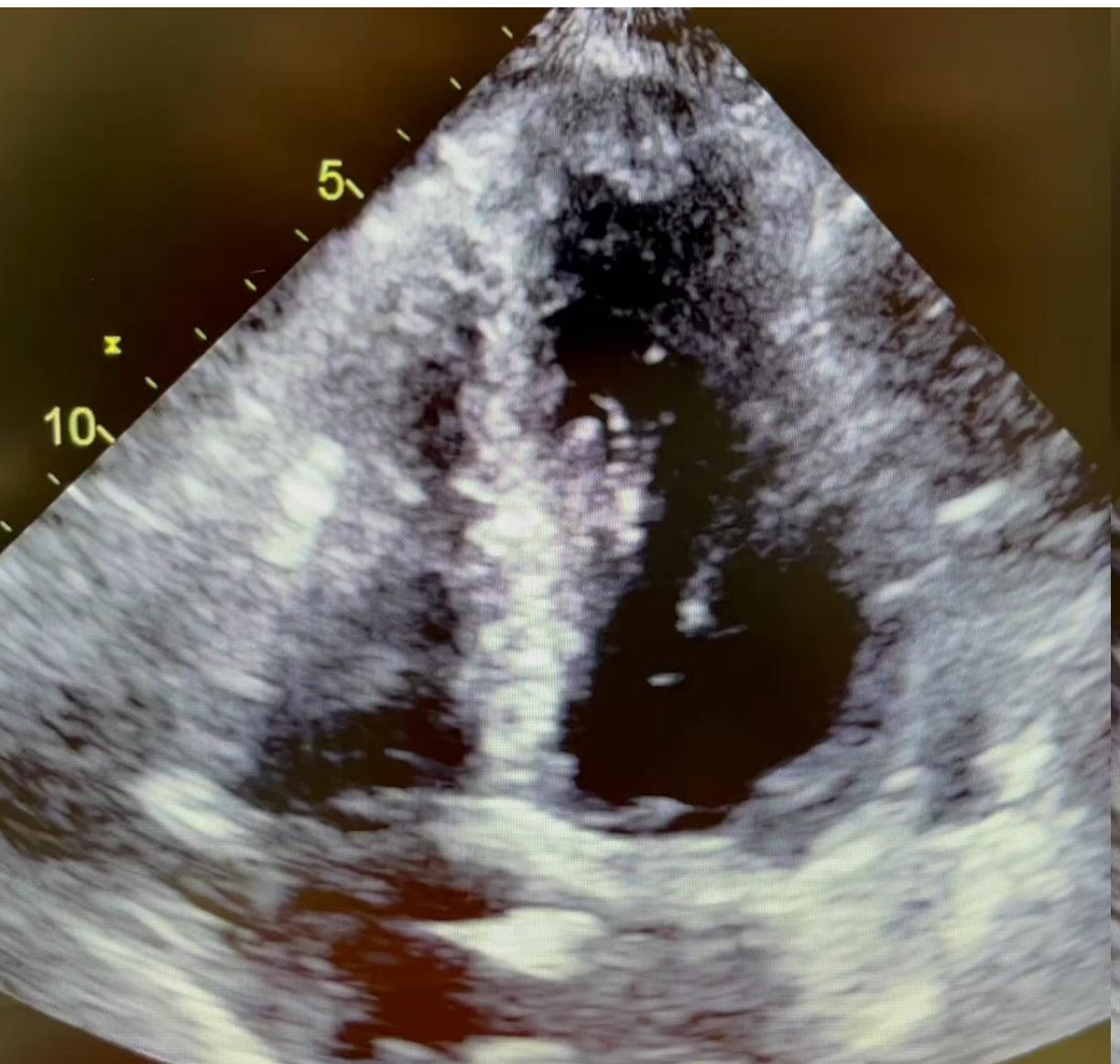


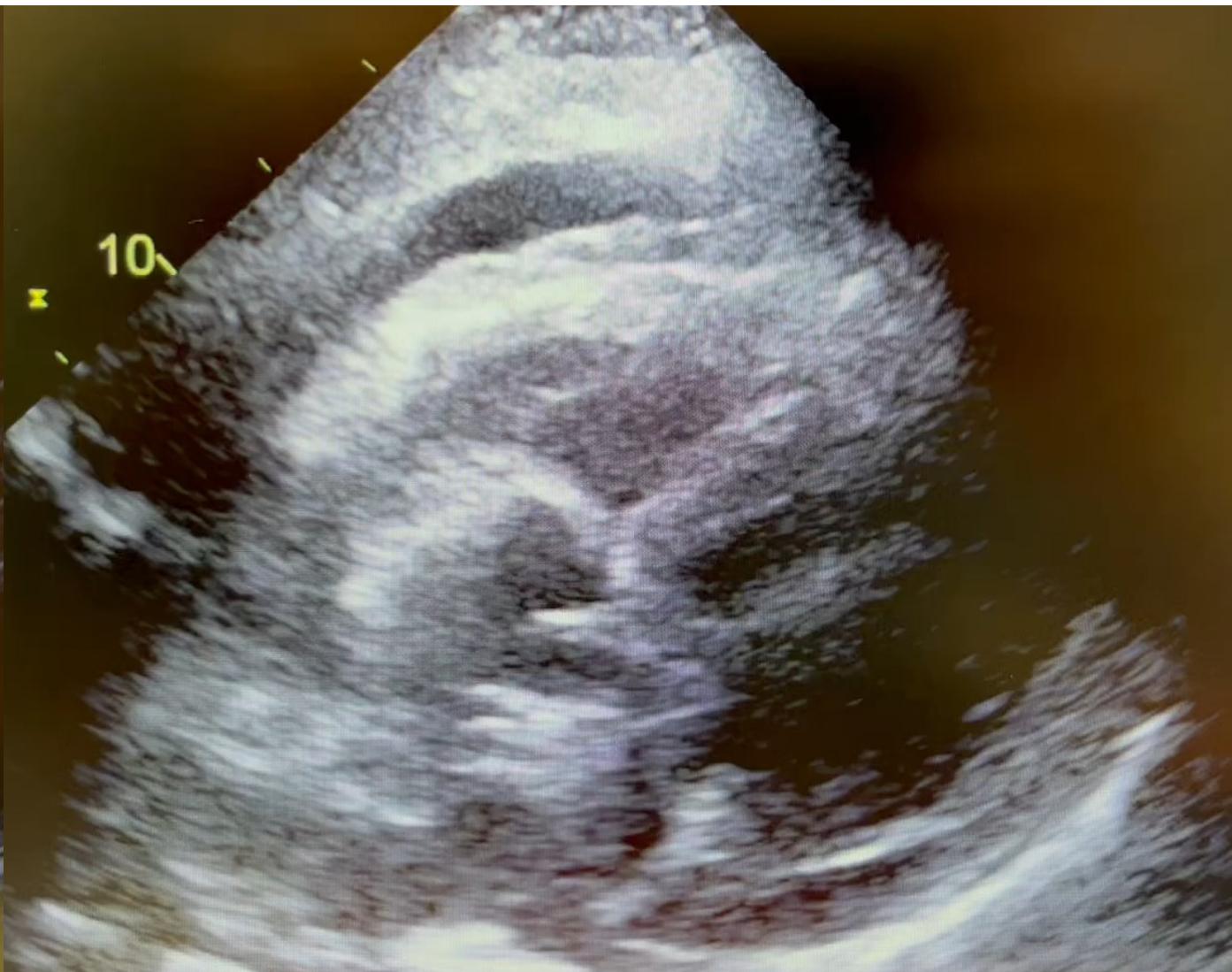
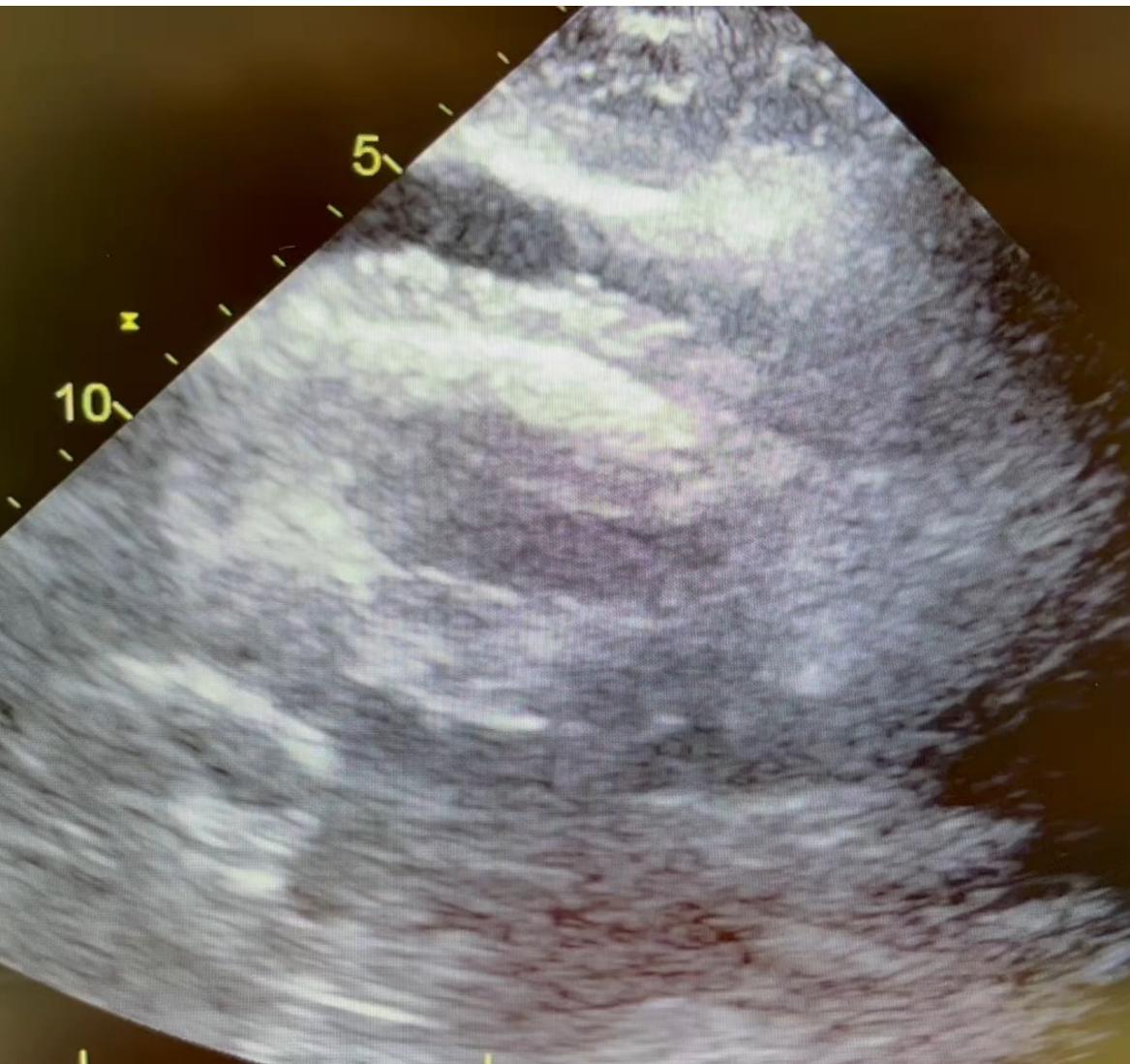
PTCA

- Severe 3VD:
- LAD 80% + D1 80%
- LCX 100% + M1 90%
- RCA CTO

10



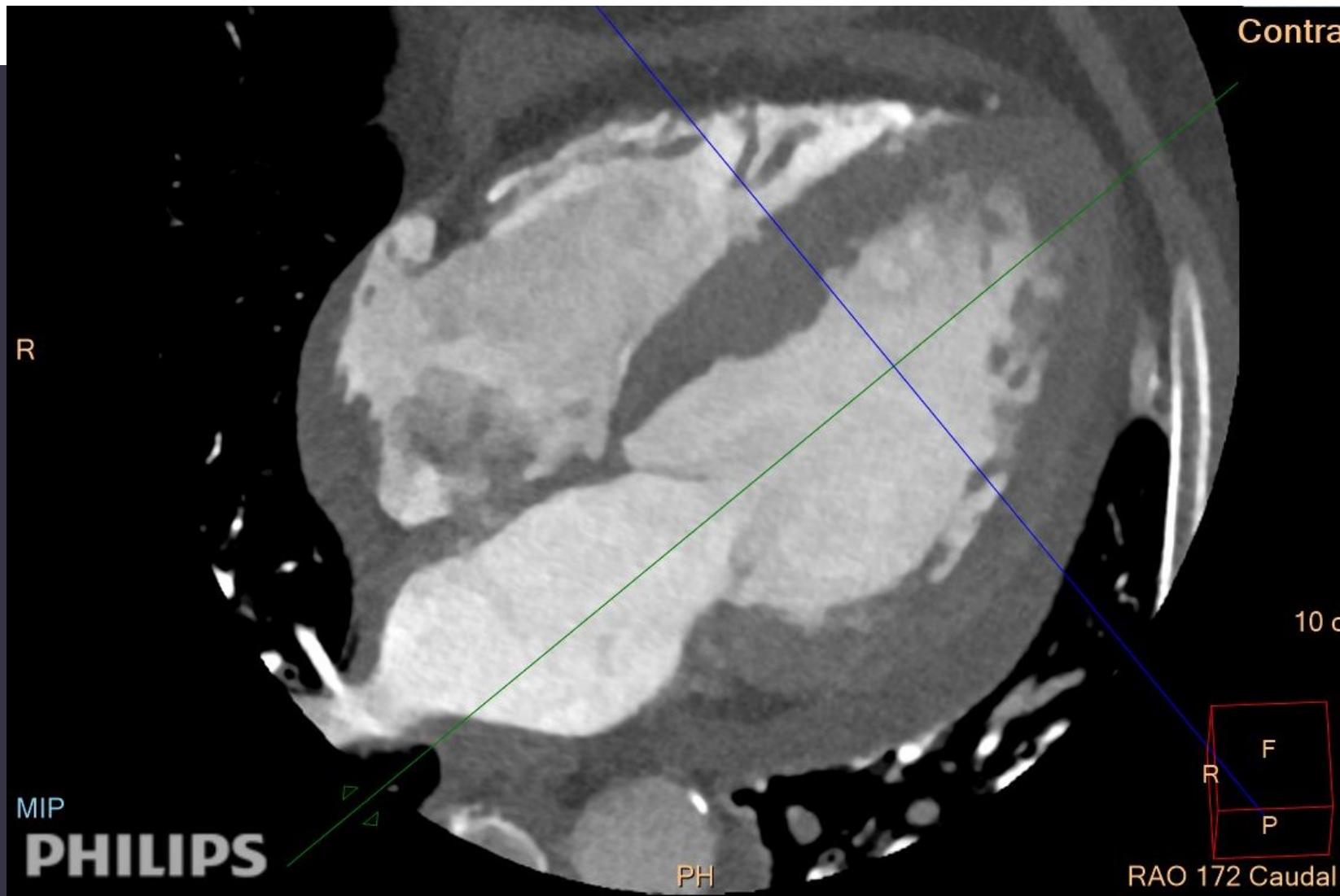




TTE CASE 3

- Severe LV Dysfunction EF=20%
- Dyskinetic Apex + apical thrombus
- Small + circumferential pericardial effusion
- Fibrin / clot on RV free wall?

CCTA



CCTA

- Dilated LV, Severe dysfunction
- Dyskinetic LV Apex
- Apical thrombus 11X13X15 mm
- Small – Moderate circumferential pericardial effusion
- No evidence of LV wall rupture

OR

- Nothing in pericardial space but minimal serotic fluid
- LIMA → LAD, SVG → M + RCA
- D1 post surgery – lt. hemiplegia
- Large Temporo-Pariatal CVA
- Major disability

DISCUSSION

- ECHO criteria for rupture
- Blood with clots VS Early Dreesler syn. With Fibrin?
- TTE vs TEE
- Contrast Echo?
- Next step CCTA?

NO GUIDELINES...

- Mortality up to 75%
- ESC GL STEMI 2017: “Diagnosis is made by echocardiography, CMRI can complement the diagnosis”
- ASE general recommendation on all 3 modalities
- TEE is advised mainly when VSR is suspected

LV Apical Rupture Complicating Acute Myocardial Infarction: The Role of CMR

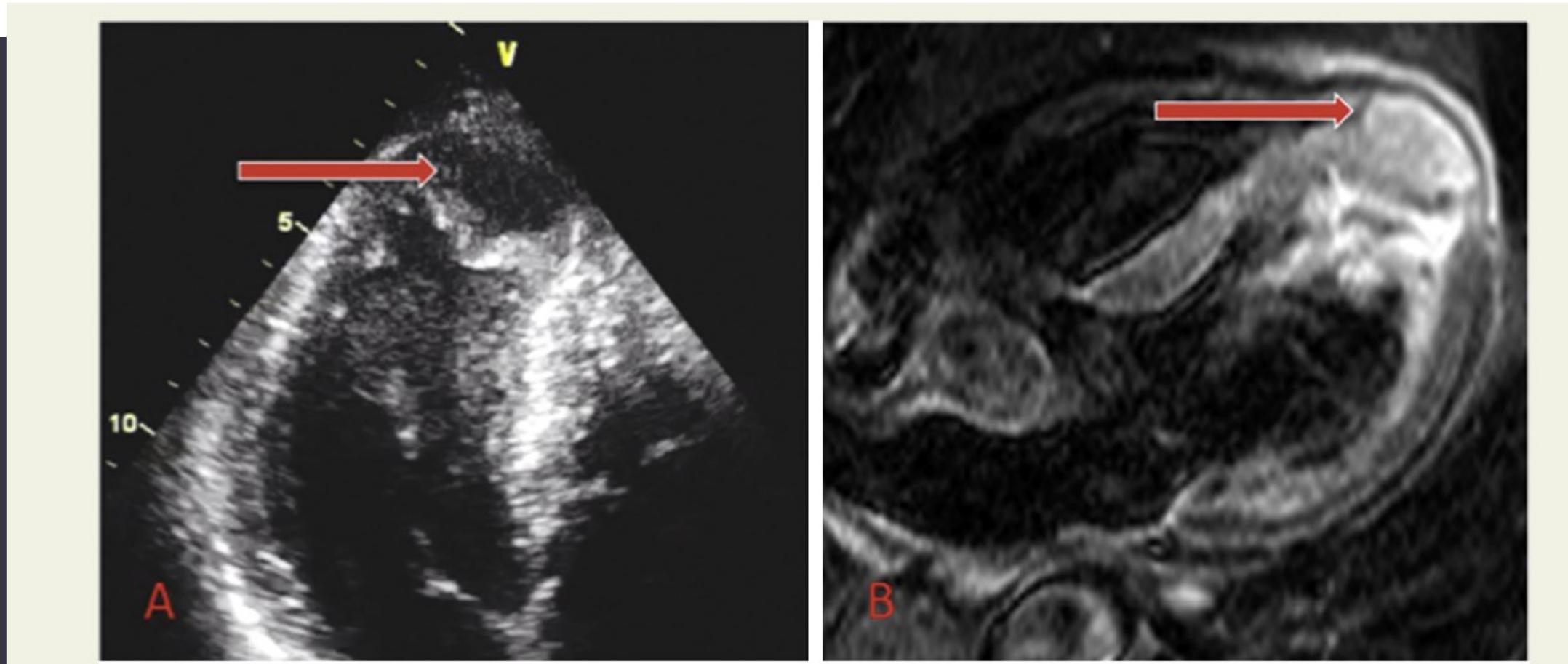


Image 2 A: Contrast Trans-thoracic Echocardiography showing a large apical thrombus, separated from LV cavity by the myocardium.

B: 4C - STIR imaging showing increased signal intensity in the thinned anterior and septal walls from mid ventricle through to apex, strongly suggestive of an acute infarct pattern also noted apical thrombus, separated from LV cavity by myocardium.

Clinical Applications of Ultrasonic Enhancing Agents in Echocardiography: 2018 American Society of Echocardiography Guidelines Update



Key Points and Recommendations for the Use of UEAs in Detecting LV Cavity Abnormalities and Intracardiac Masses

1. Ultrasound enhancement should be used in patients in whom LV thrombus cannot be ruled in or out with noncontrast echocardiography (COR I, LOE B-NR).
2. Ultrasound enhancement should be considered in patients in whom structural abnormalities of the left ventricle (noncompaction cardiomyopathy, apical hypertrophy and aneurysms) cannot be adequately assessed with noncontrast echocardiography (COR IIa, LOE B-NR).
3. Ultrasound enhancement should be used for ruling in or out an LV pseudoaneurysm (COR I, LOE B-NR). (This item is highlighted with a red border)
4. Ultrasound enhancement with VLMI imaging should be used in the differential diagnosis of cardiac masses by assessing the vascularity of the mass (COR IIa, LOE B-NR).
5. Ultrasound enhancement should be considered during TEE whenever the atrial appendage has significant spontaneous contrast or cannot be adequately visualized with unenhanced imaging (COR IIa, LOE B-NR).

ECHO CONTRAST ROLE

Intravenous use

Patients with unstable cardiopulmonary status

ECG monitoring should be performed in high-risk patients as clinically indicated and a close medical supervision is recommended.

Use extreme caution when considering the administration of SonoVue in patients with recent acute coronary syndrome or clinically unstable ischaemic cardiac disease, including: evolving or ongoing myocardial infarction, typical angina at rest within last 7 days, significant worsening of cardiac symptoms within last 7 days, recent coronary artery intervention or other factors suggesting clinical instability (for example, recent deterioration of ECG, laboratory or clinical findings), acute cardiac failure, Class III/IV cardiac failure, or severe rhythm disorders

DISCUSSION

- ECHO criteria for rupture
- Blood with clots VS Early Dreesler syn. With Fibrin?
- TTE vs TEE
- Contrast Echo?
- Next step CCTA?

THANK YOU

(תודה לפרופ. קובל, ד"ר שלו, ד"ר מצא)

