Imaging cases

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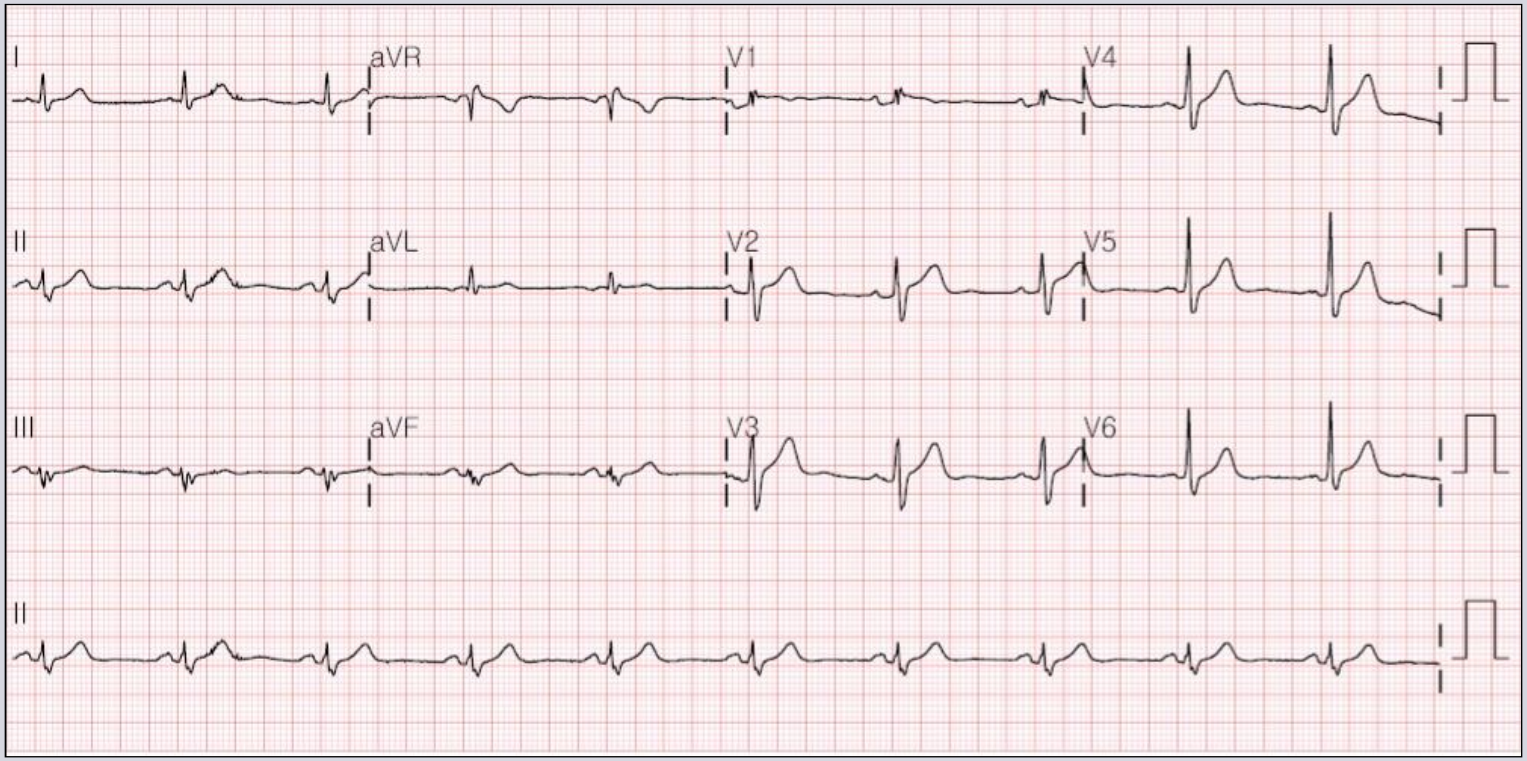
M/62

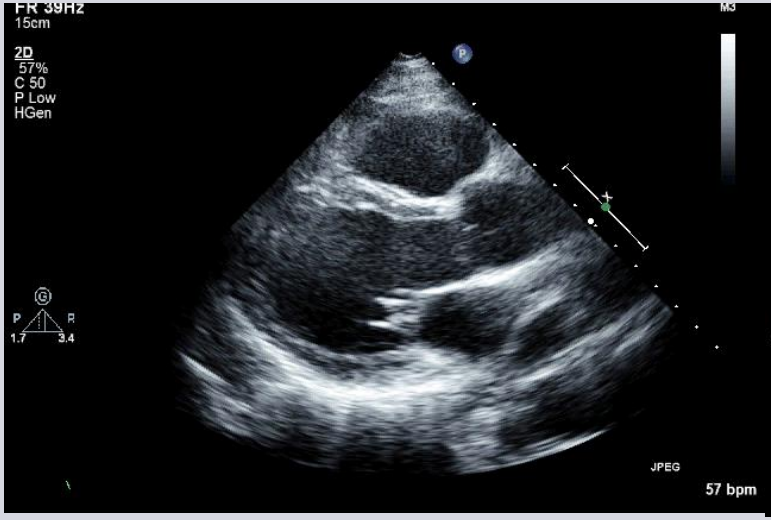
C.C.: Acute onset chest pain

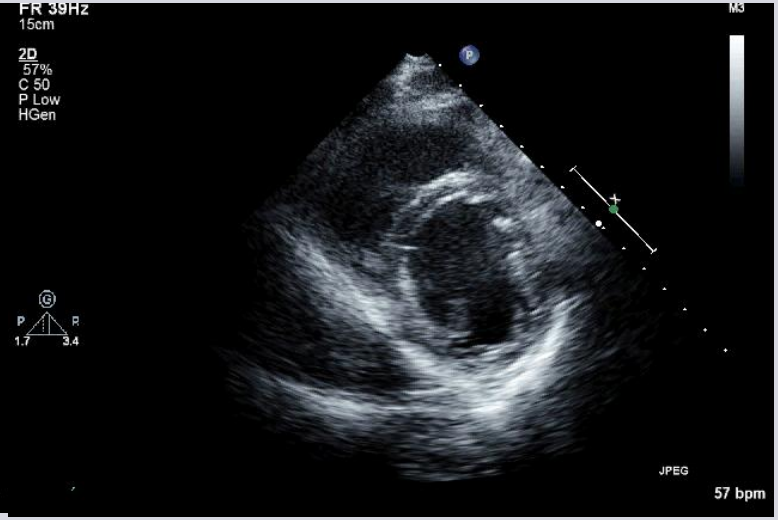
(Glottic cancer (CTx & RTx))

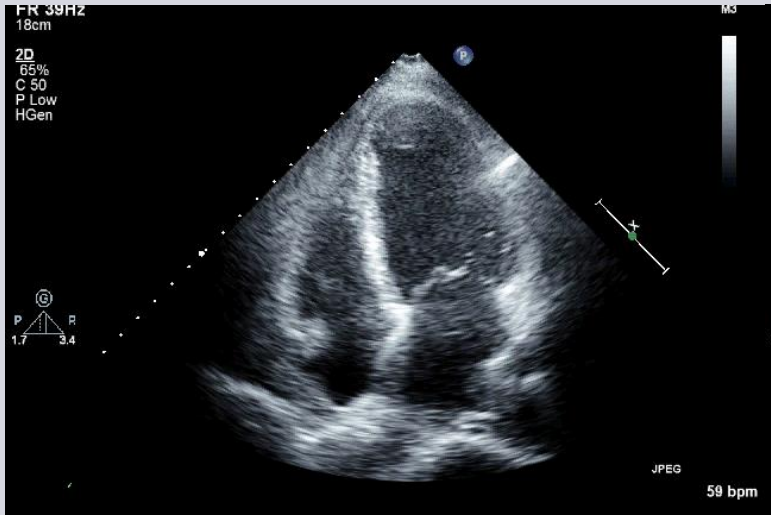
- ECG:

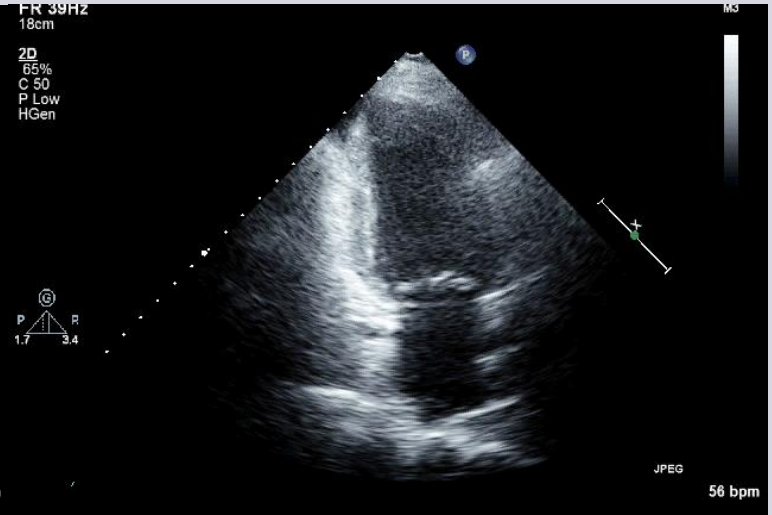


- 2D echo:

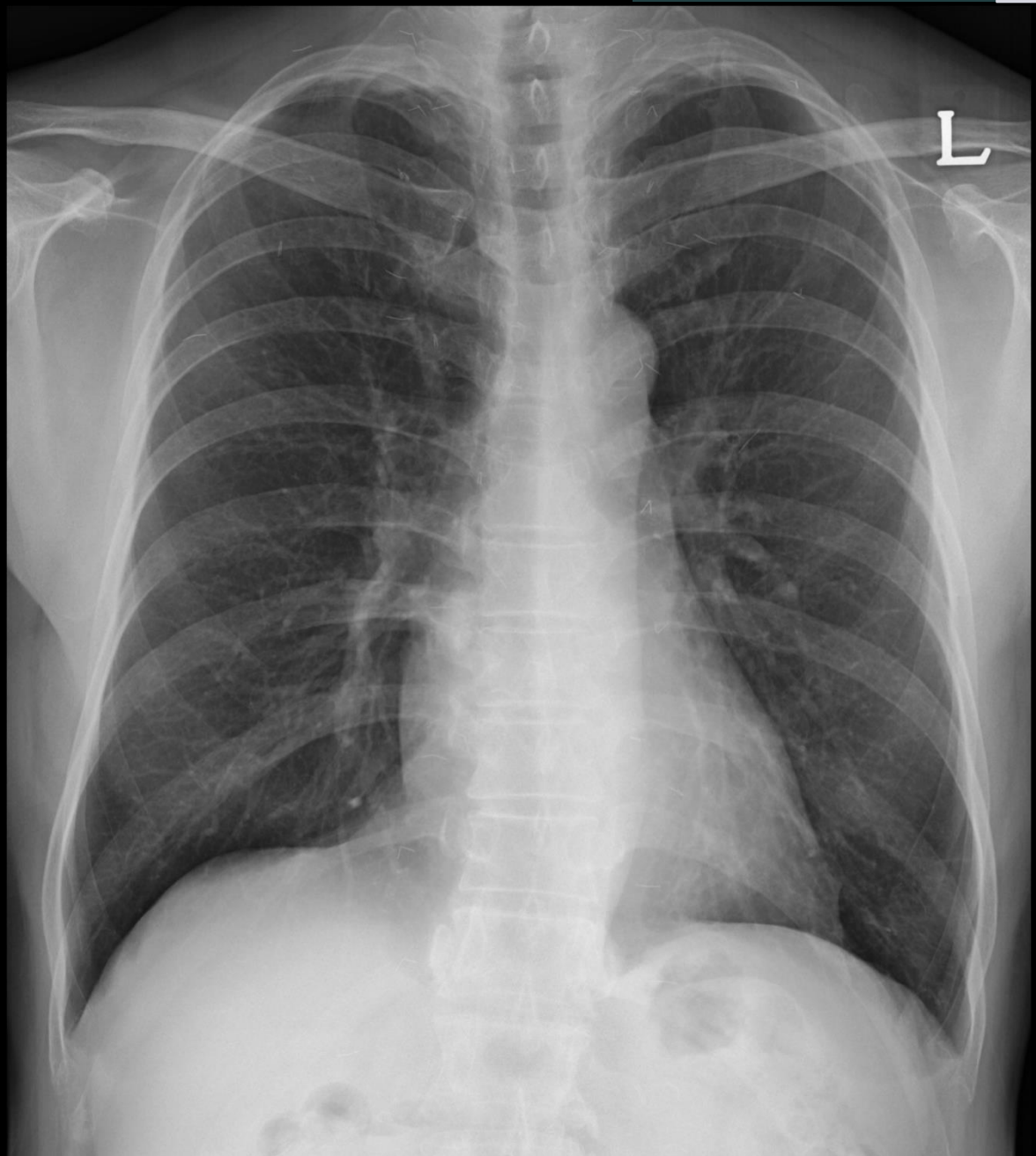

FR 39Hz
15cm
2D
57%
C 50
P Low
HGen
M3
P
R
1.7
3.4
JPEG
57 bpm


FR 39Hz
15cm
2D
57%
C 50
P Low
HGen
M3
P
R
1.7
3.4
JPEG
57 bpm

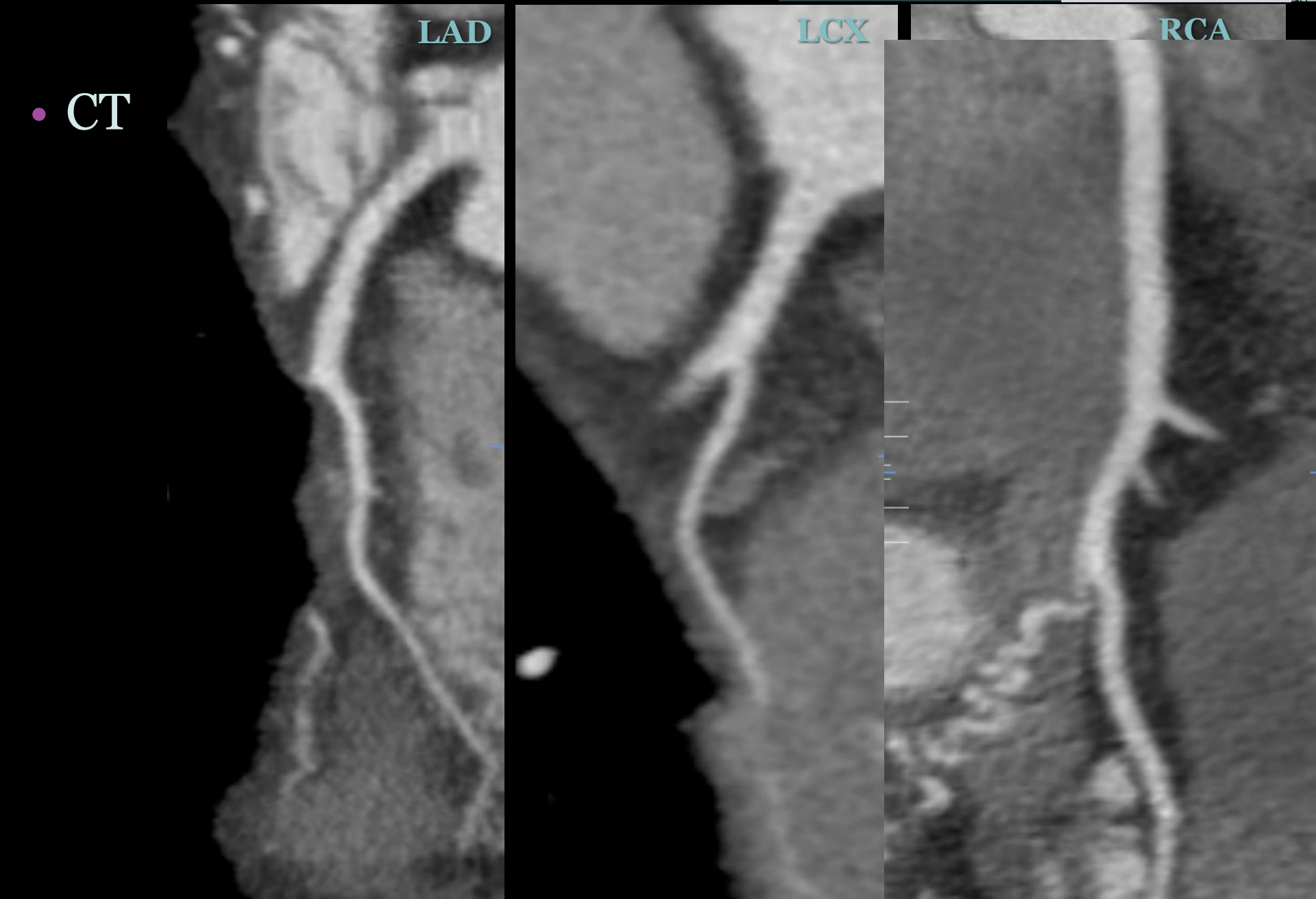

FR 39Hz
18cm
2D
65%
C 50
P Low
HGen
M3
P
R
1.7
3.4
JPEG
59 bpm


FR 39Hz
18cm
2D
65%
C 50
P Low
HGen
M3
P
R
1.7
3.4
JPEG
56 bpm

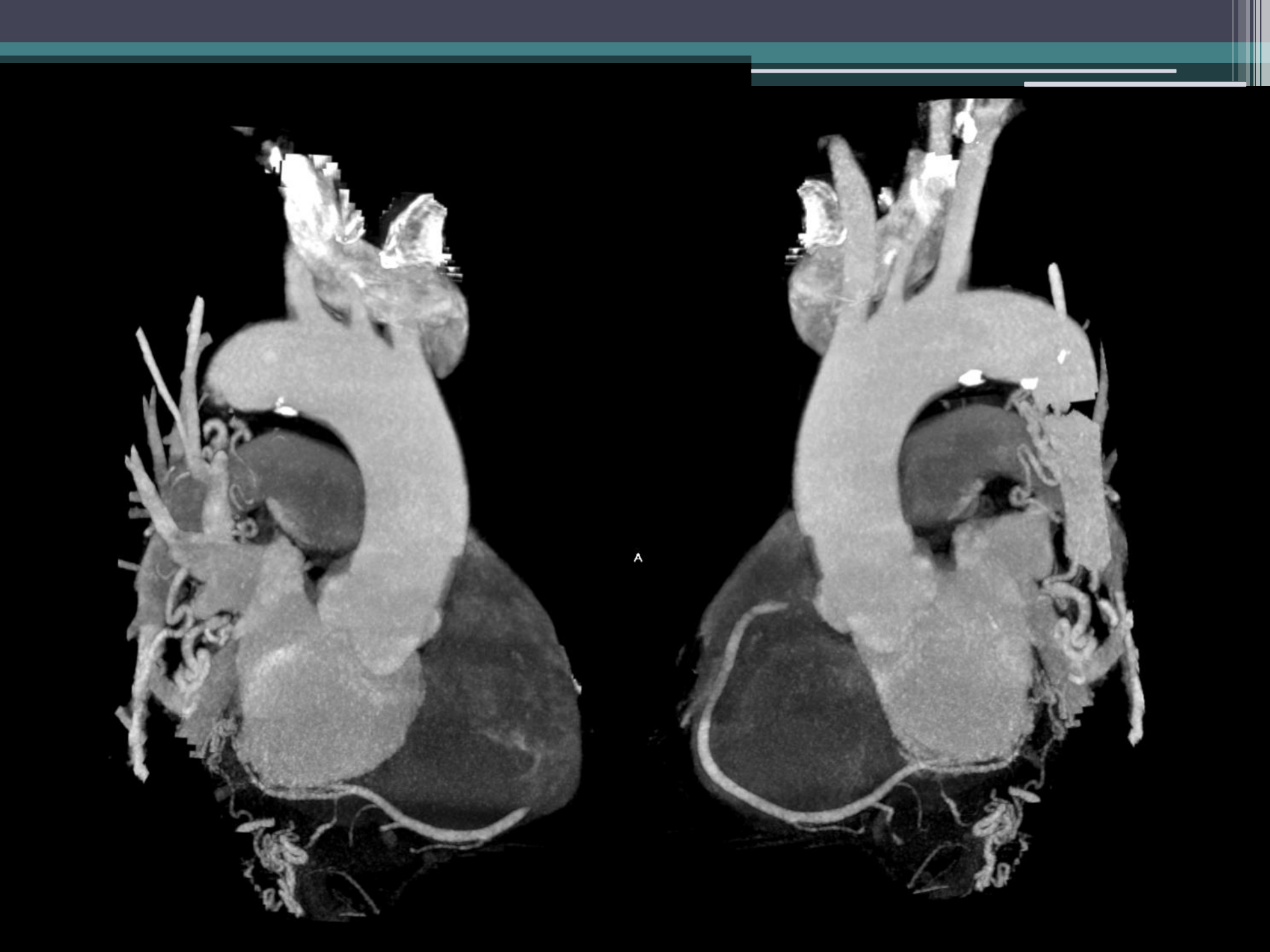
- Chest PA



- CT

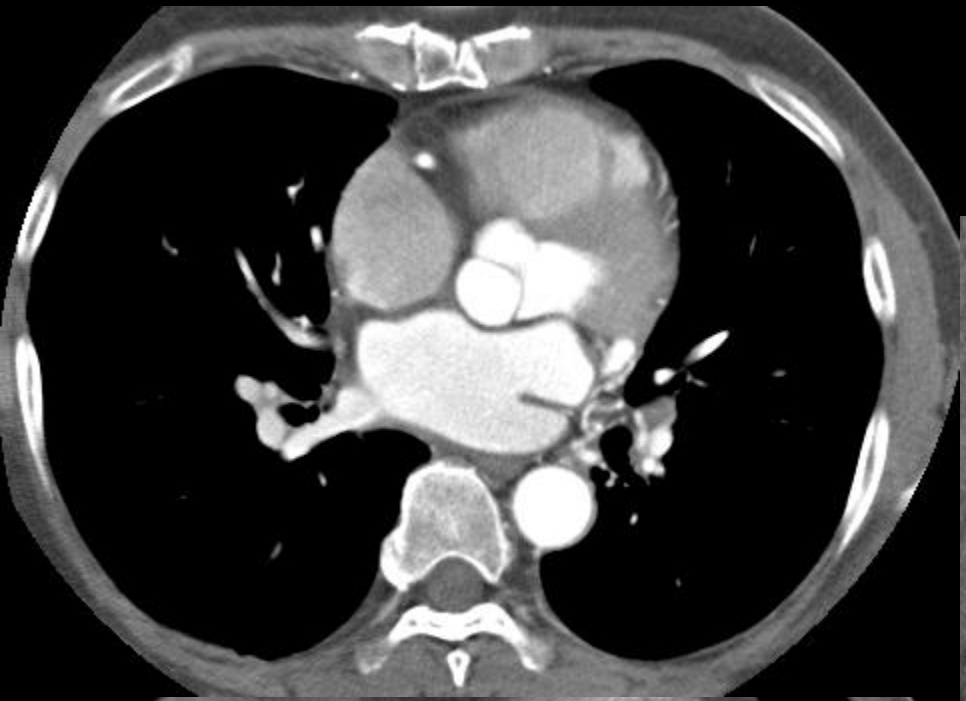


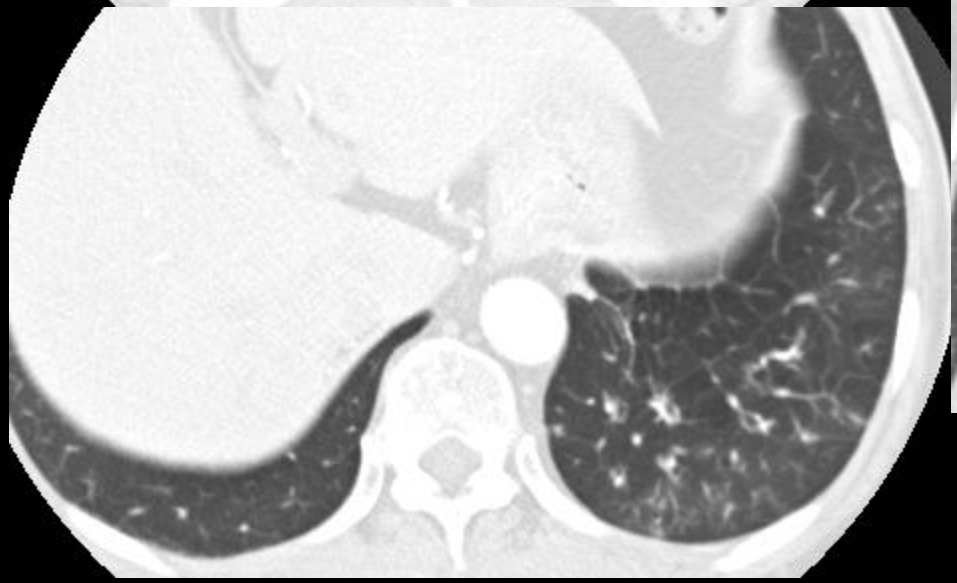

R
S
A
L
I
P

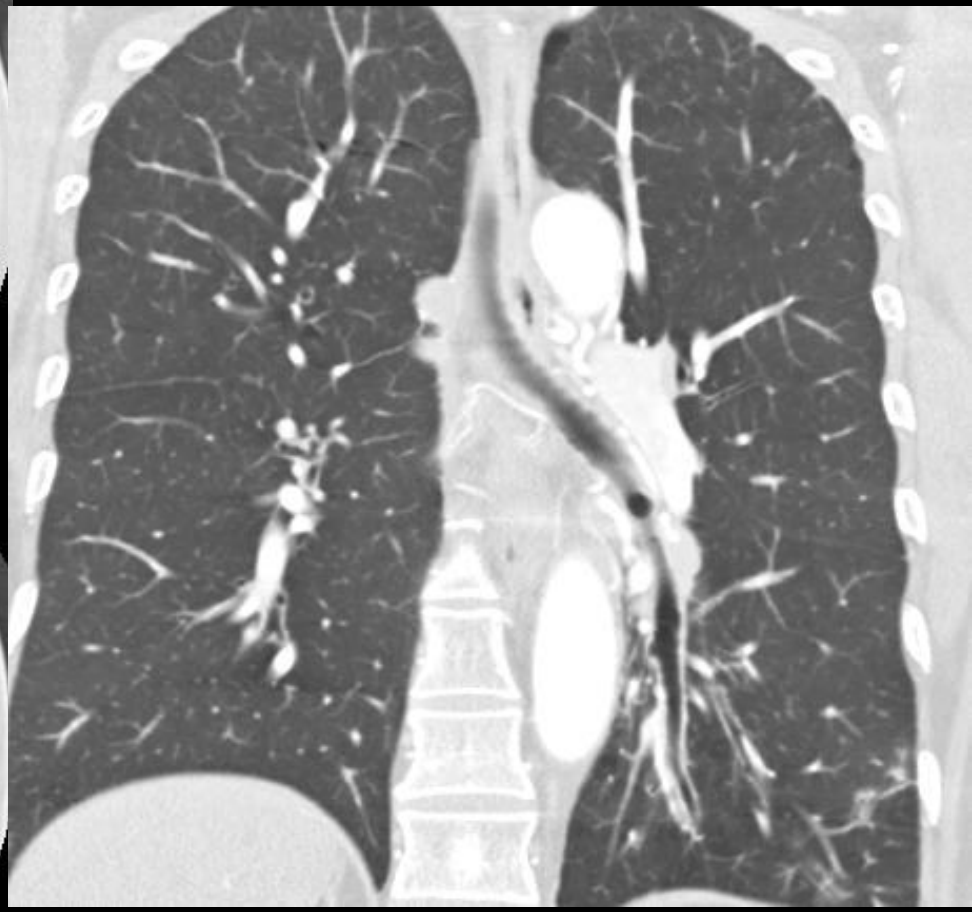

A


SPL
IAR

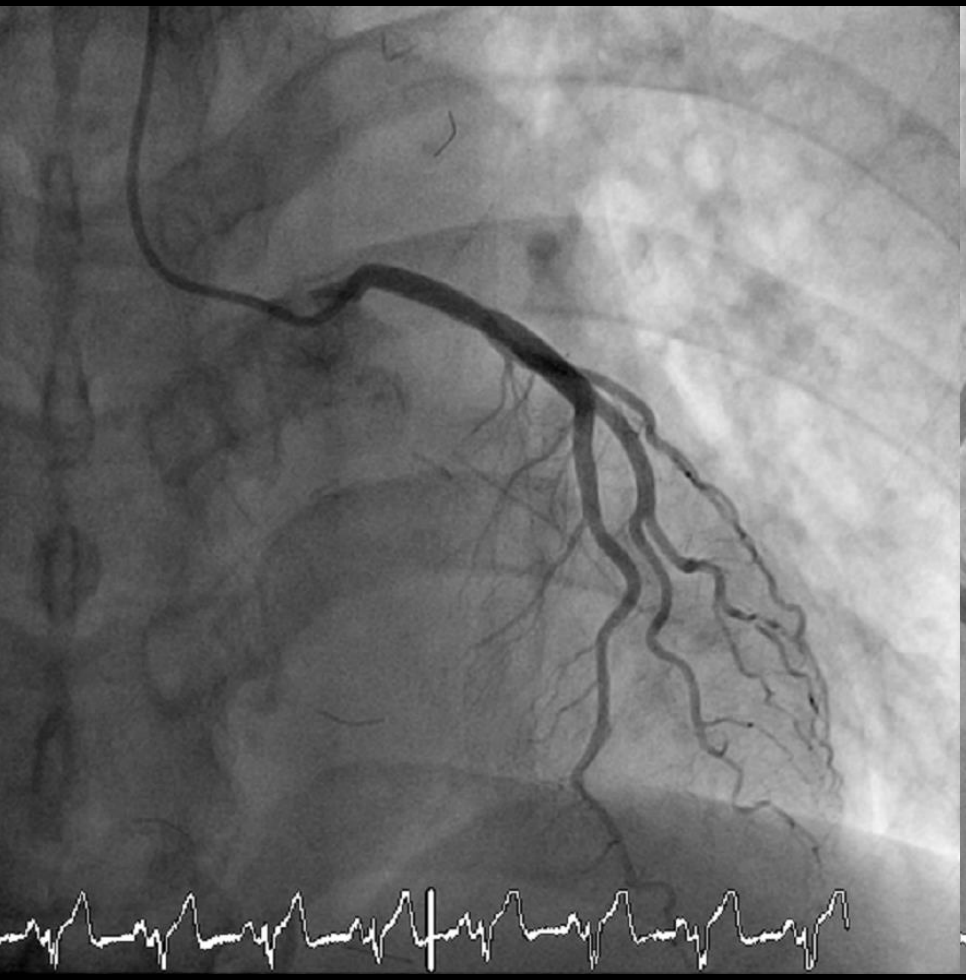

SPR







- CAG:



Coronary-to-Bronchial Artery Fistula (CBF)

- Anastomosis between coronary arteries and various systemic vessels: included..
  - pericardiophrenic, pericardial, bronchial, anterior mediastinal, intercostal, superior and inferior phrenic arteries, and esophageal branches of the aorta, etc..

- CBF are present in all patients **from birth and remain closed**: because of the **similarity in filling pressure in coronary and bronchial circulation**.

➔ Sizable CBF may result from **considerable and persistent disturbance of pressure equilibrium.**

Acta Med Scand Suppl. 1968;485:5-26.

- Usually originate from **LCX**
  - 13 of 16 cases in the series of Matsunaga et al.
  - 6 of 8 cases in the series of Lee et al.

- Cross the pericardium by means of pericardial reflections in the retrocardiac spaces.

- Associated with various cardiovascular diseases and chronic pulmonary diseases: including ,,
  - pulmonary artery hypoplasia, tetralogy of Fallot, supravalvular aortic stenosis, Takayasu arteritis, pulmonary thromboembolism, **bronchiectasis**, pulmonary tuberculosis.

Radiology. 1993;186:877-882.

JCAT 2008;32:444-447

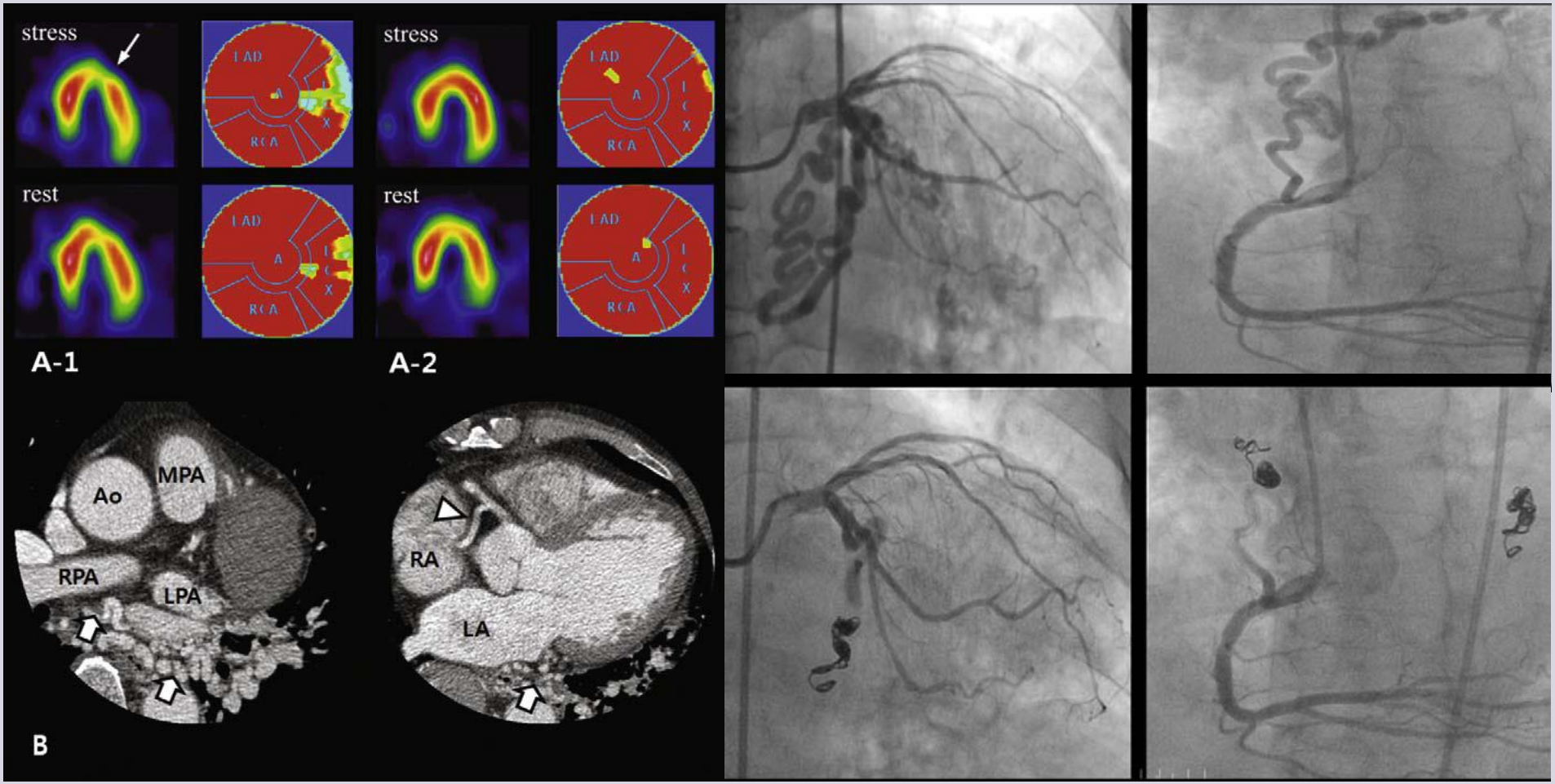
- Symptoms
  - Asymptomatic (m/c)
  - Cardiovascular symptoms (rare)
    - continuous machinery murmur, angina (d/t coronary steal phenomenon), congestive heart failure, infective endocarditis, rupture of an aneurysmal fistula.
  - Can be source of hemoptysis

- Treatment:
  - Coronary steal phenomenon; Stent graft or coil embolization
  - Severe coronary artery disease; CABG with surgical ligation

Cardiovasc Intervent Radiol. 1999;22:251Y254.

Catheter Cardiovasc Interv. 1999;46:214Y217.

M/35, operation for bronchiectasis 3 years ago, exertional chest pain.



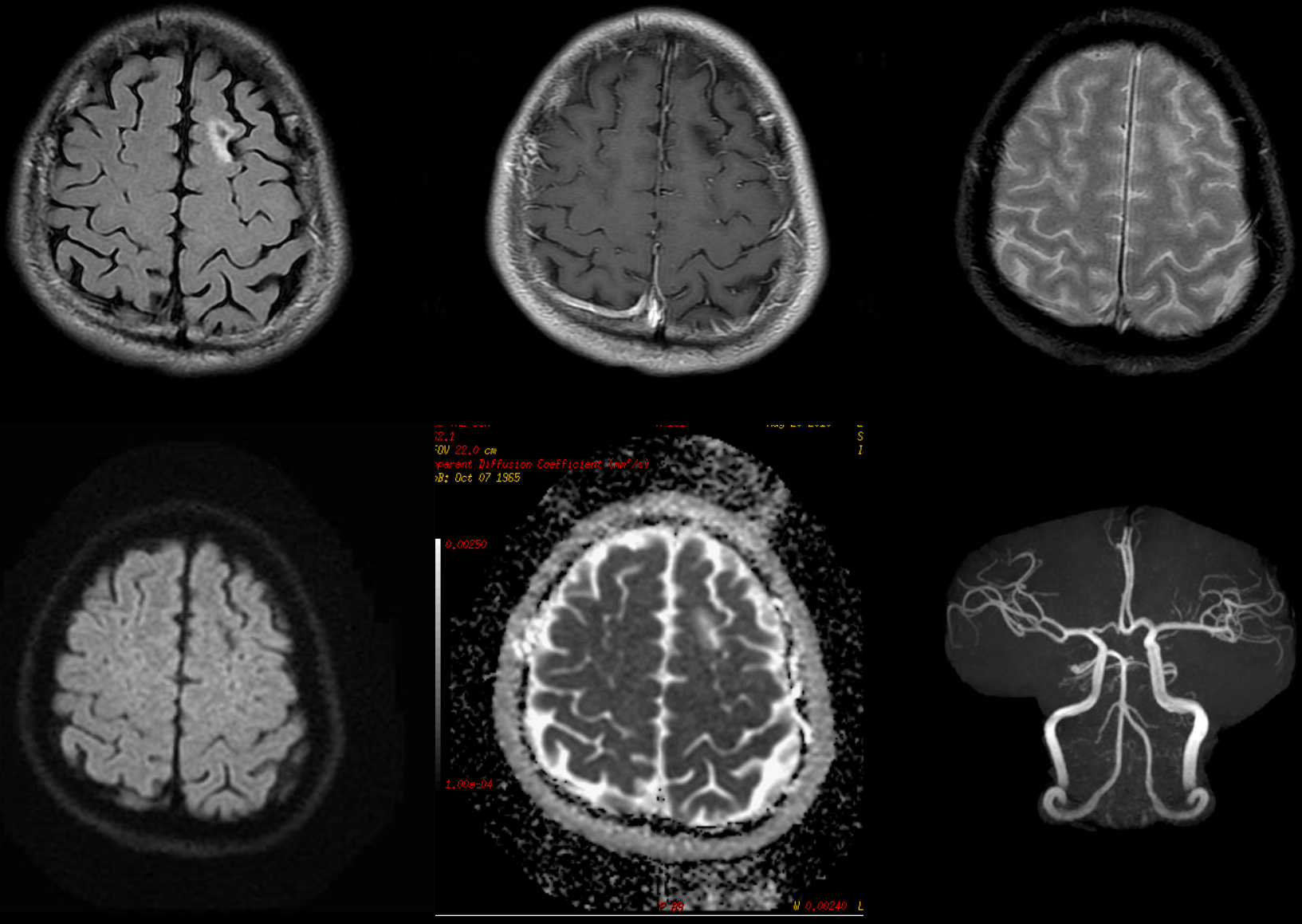
European Journal of Cardio-thoracic Surgery 2011;39: 278

M/45

C.C.: Transient Right leg weakness

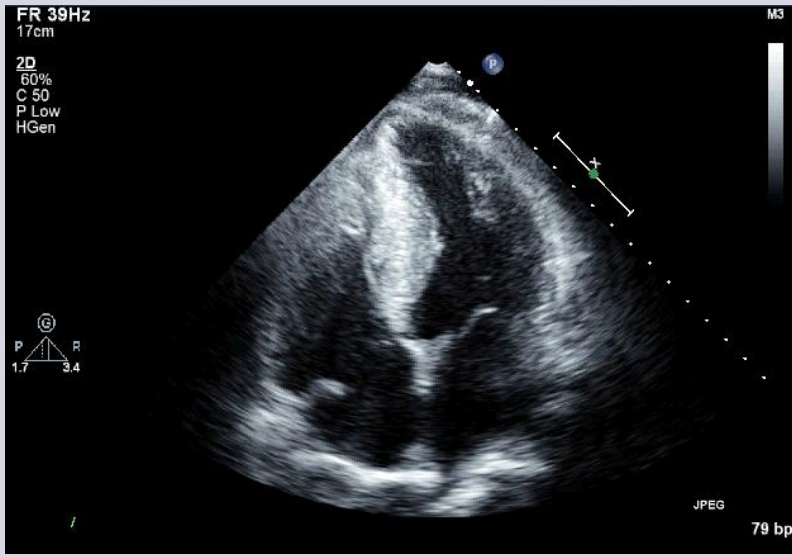
- 20 years ago; Diagnosis with HCMP
- 5 years ago; Right side weakness
  → Left ACA infarction Dx

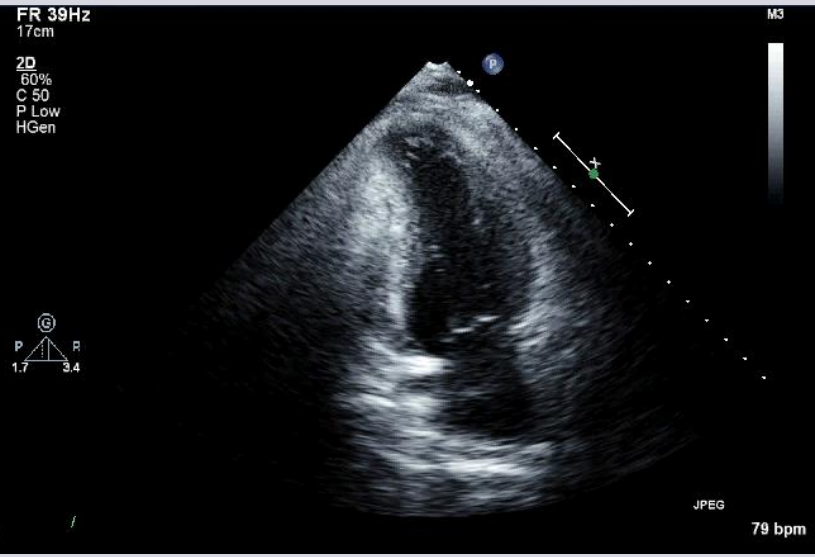
- Brain MRI:


FOV 22.0 cm
Apparent Diffusion Coefficient (mm²/s)
DOB: Oct 07 1965
0.00250
1.00e-04
W 0.00240

- 2D echo:

2008/5/9 (2 years ago)

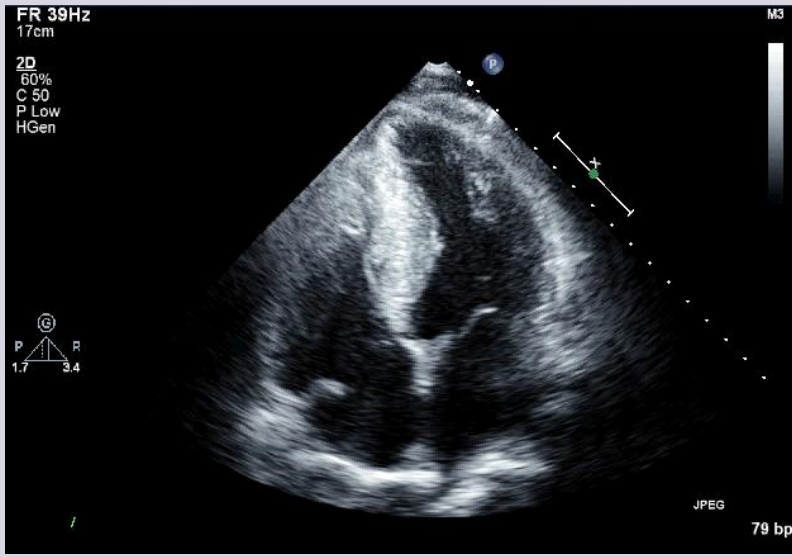


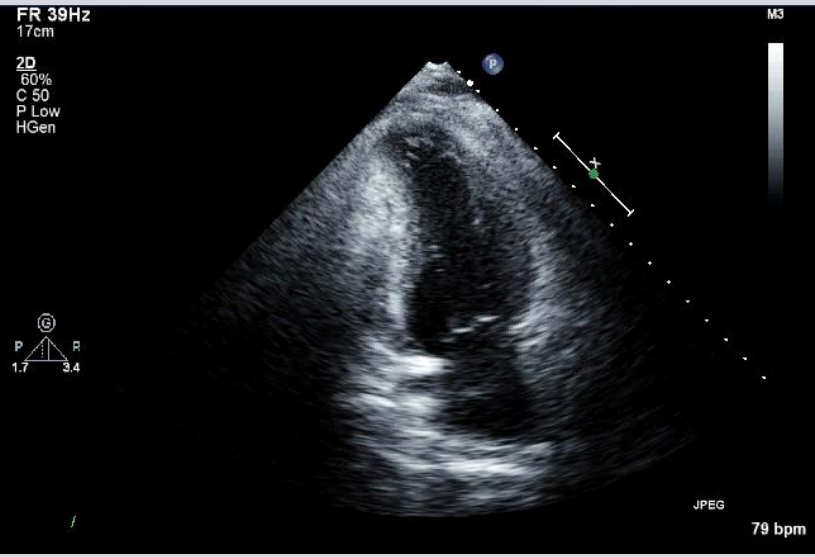


2010/5/24

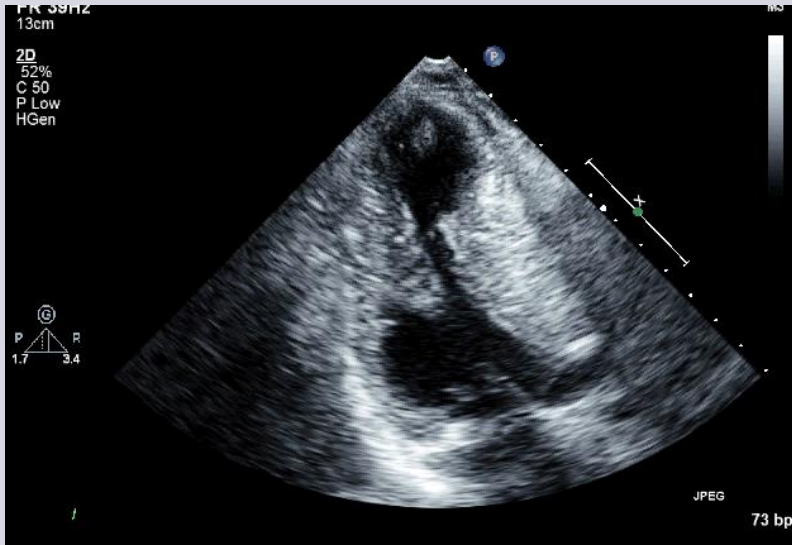
- 2D echo:

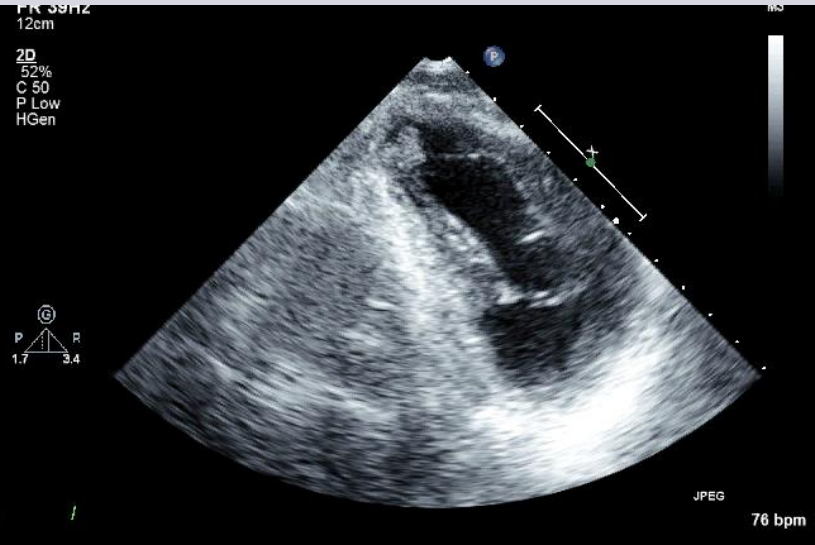
2008/5/9 (2 years ago)



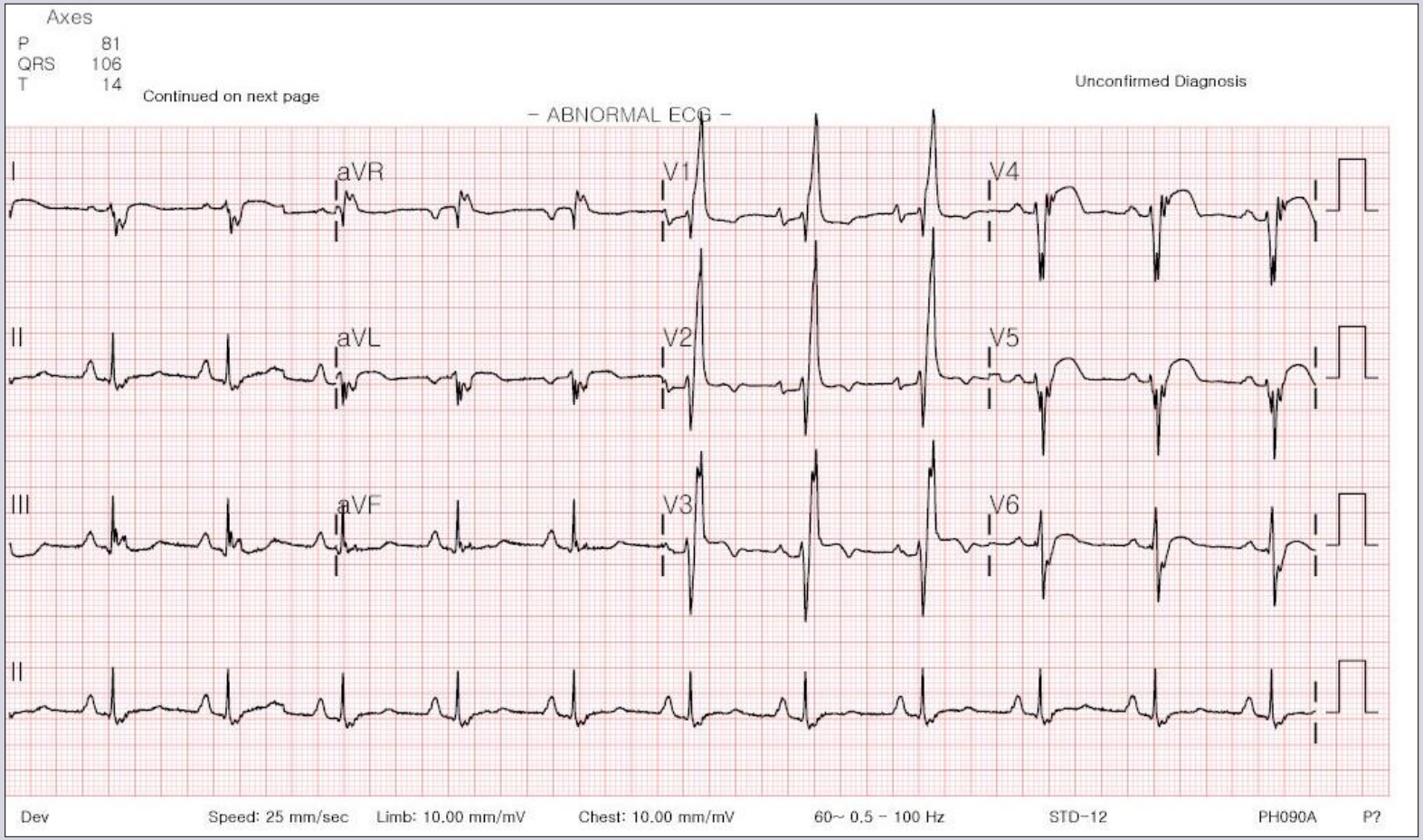


2010/5/24

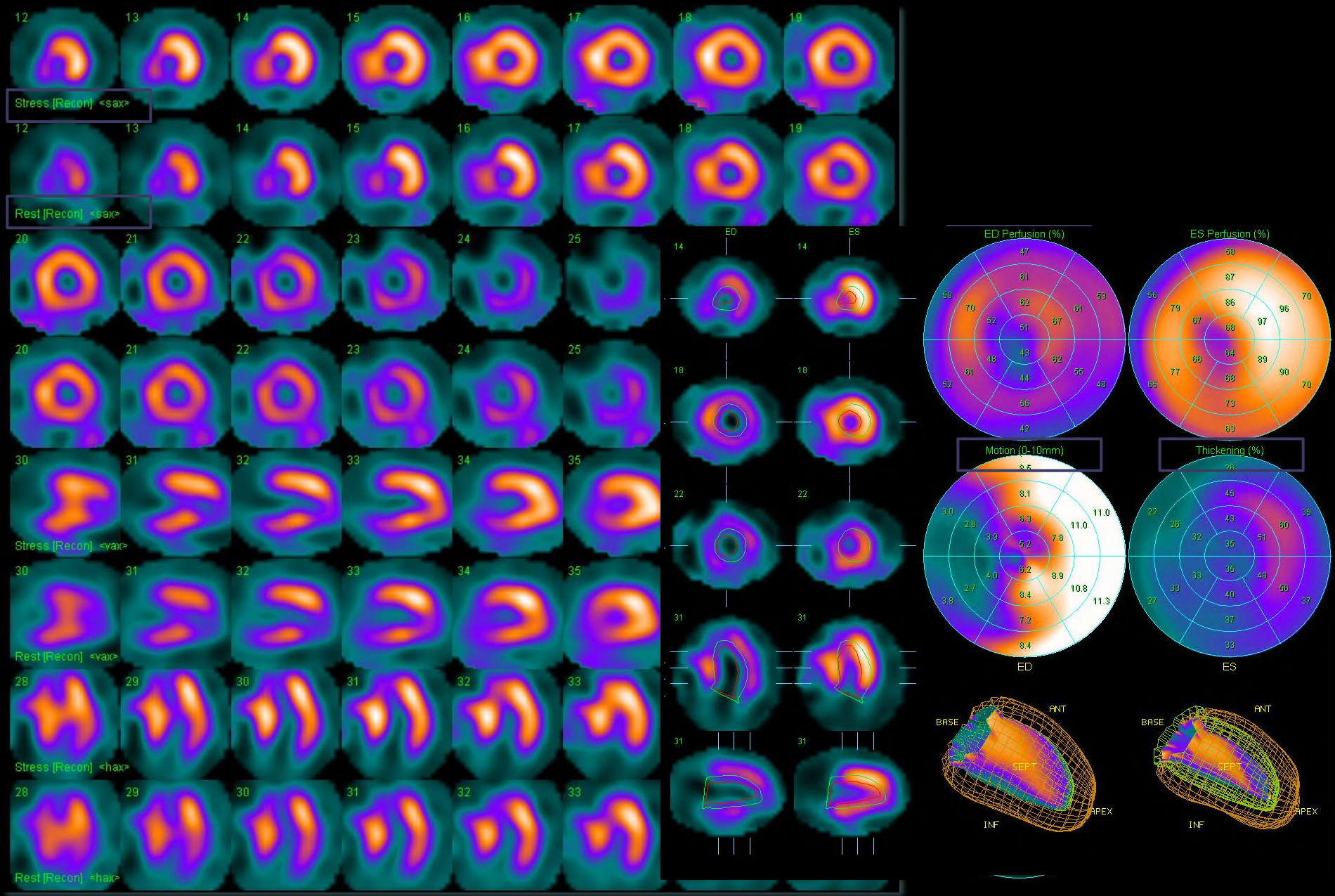




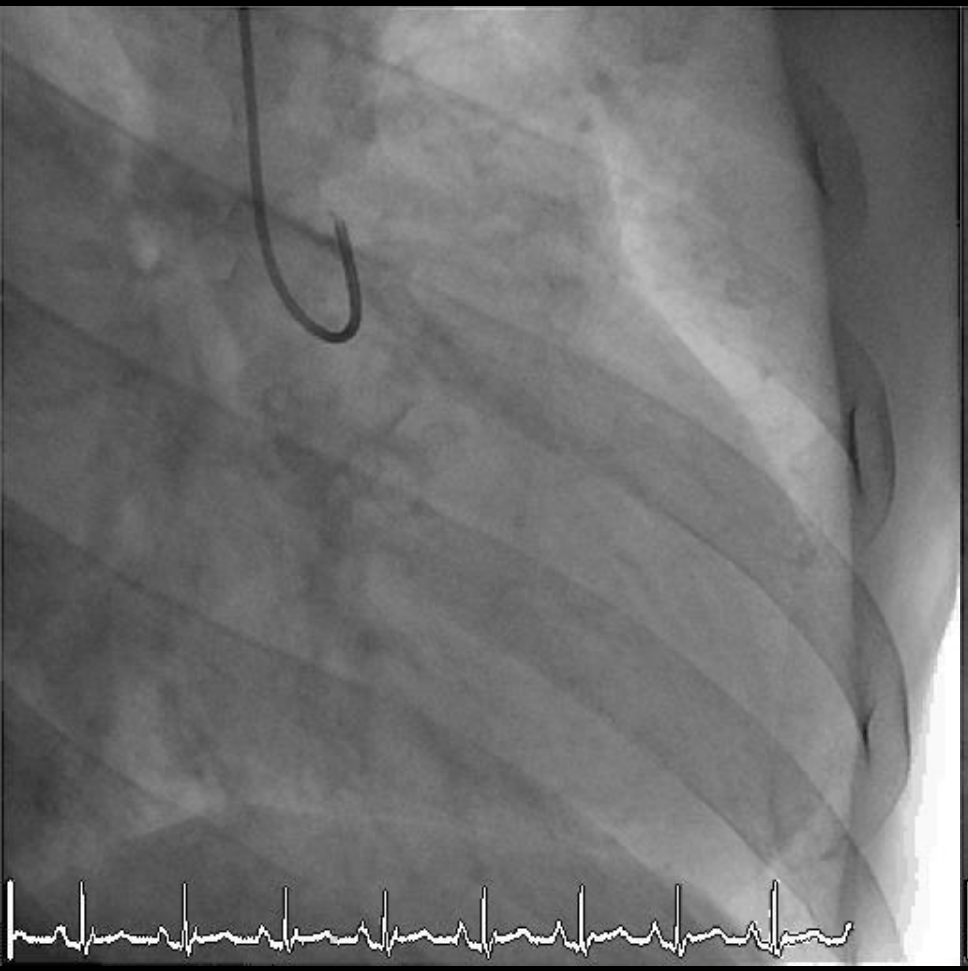
- ECG:

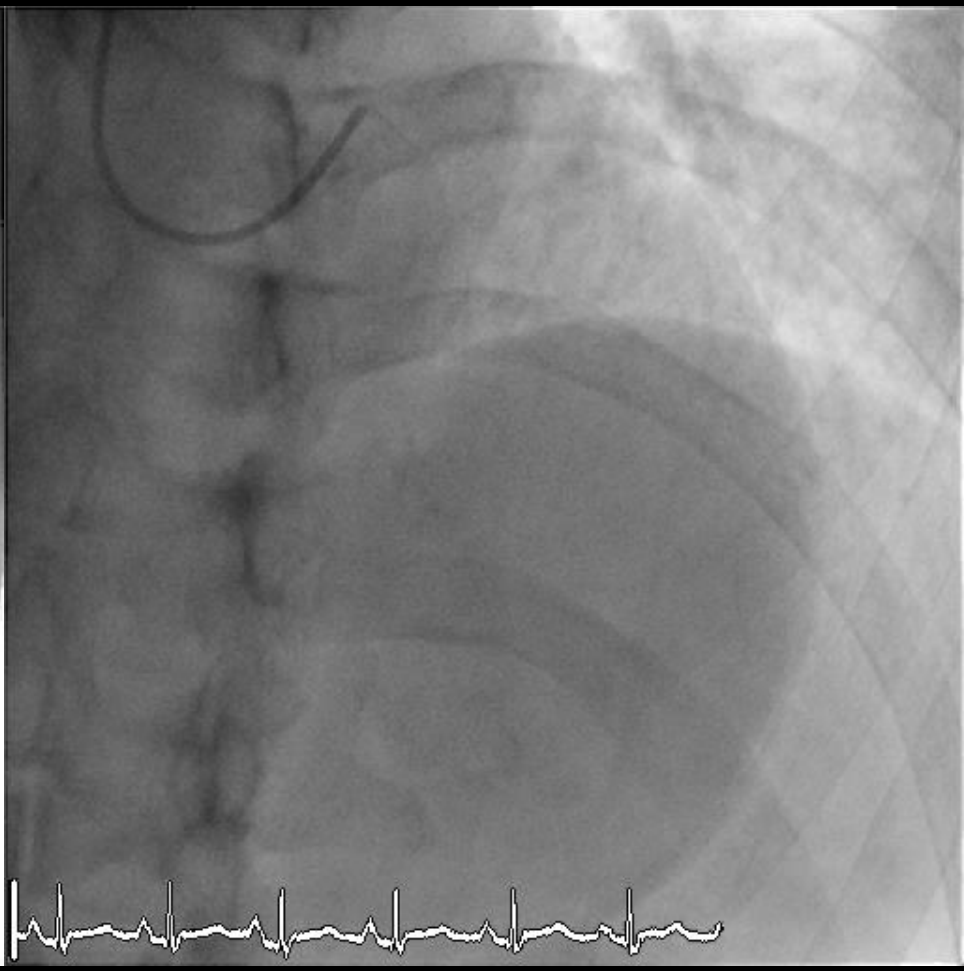

Axes
P 81
QRS 106
T 14
Continued on next page
Unconfirmed Diagnosis
- ABNORMAL ECG -
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Dev
Speed: 25 mm/sec
Limb: 10.00 mm/mV
Chest: 10.00 mm/mV
60~ 0.5 - 100 Hz
STD-12
PH090A
P?

- 201-Thallium SPECT

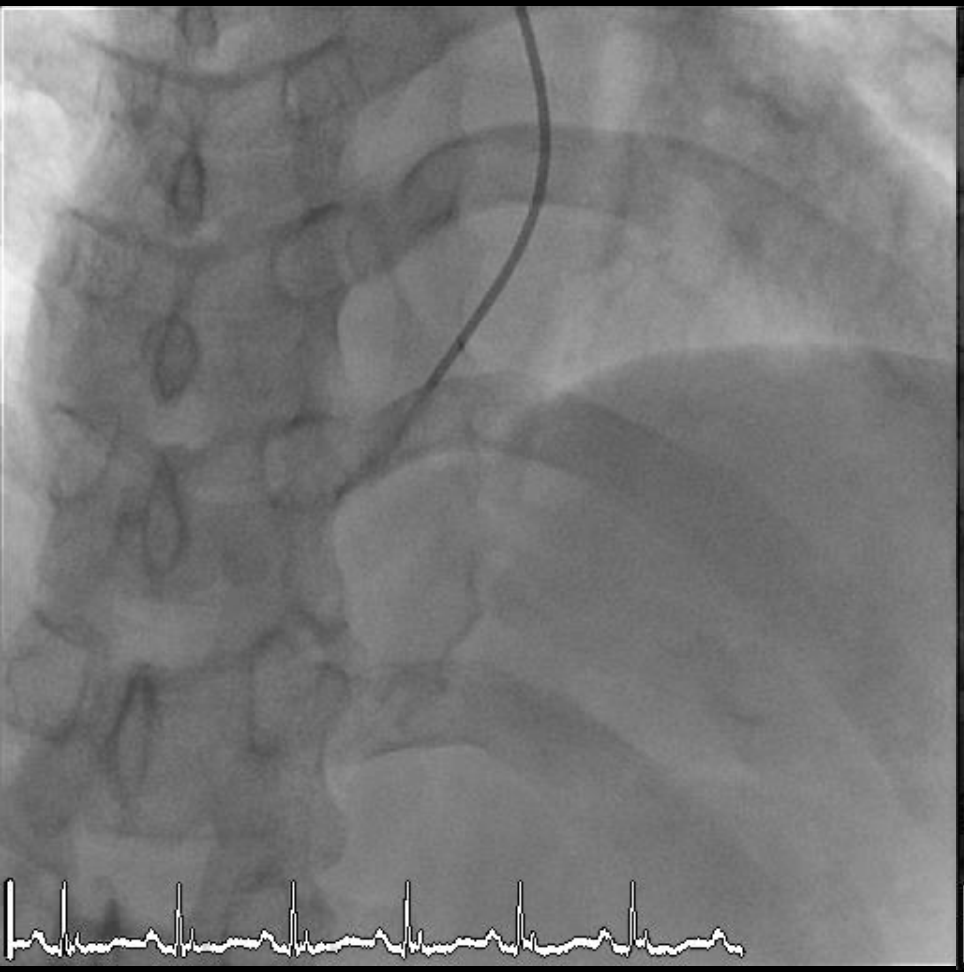


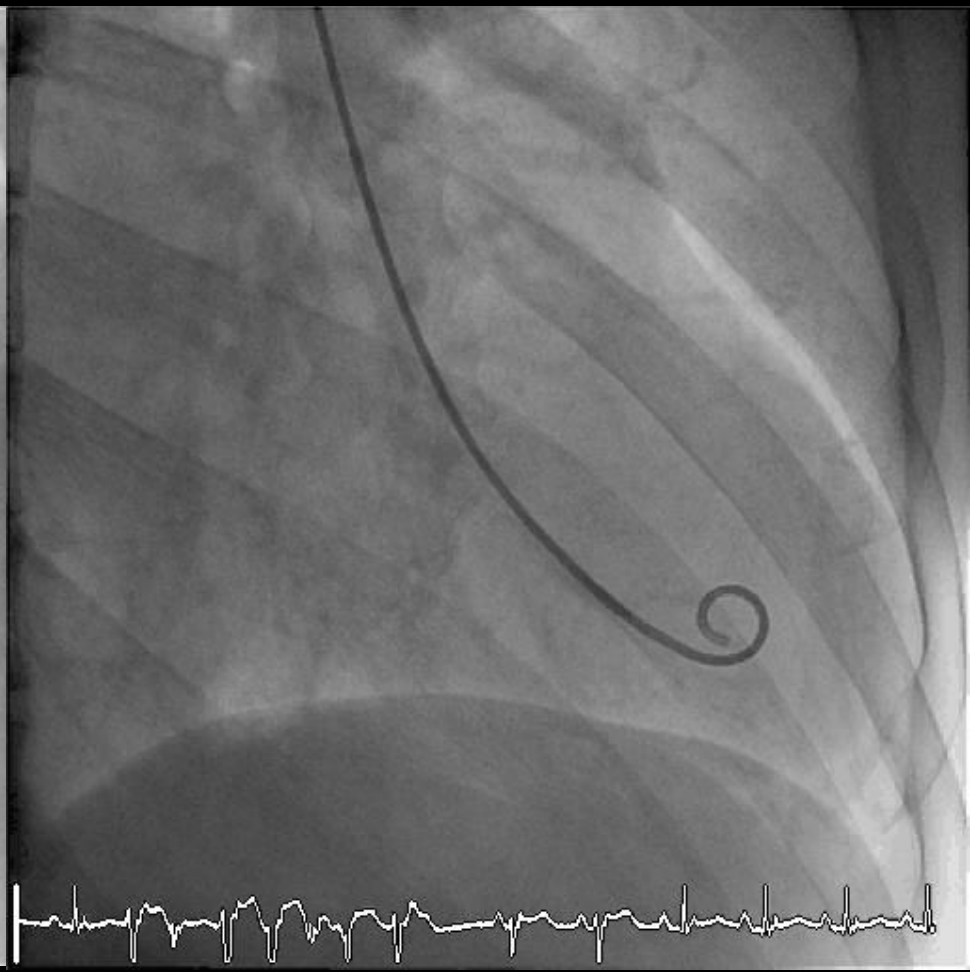
- CAG:





LVEDP 20-22cmH2O
LVOT pressure gradient <10cmH2O





- Heparization + Warfarin start

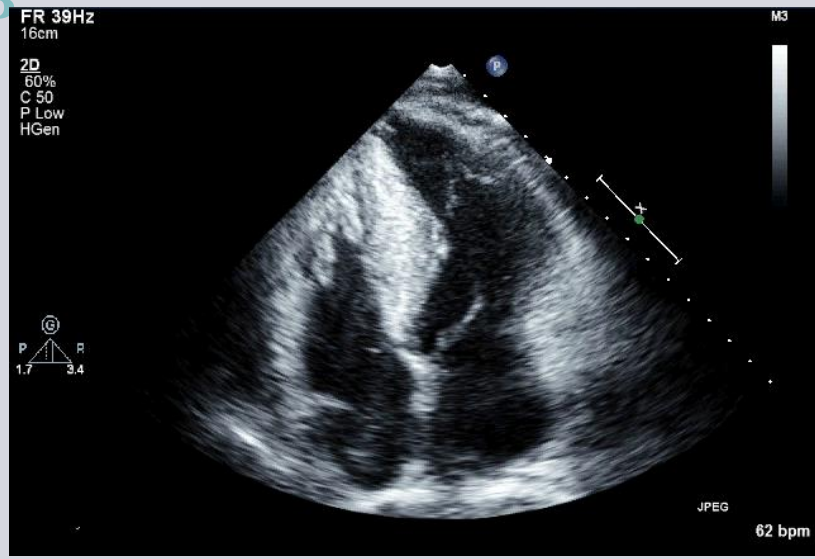
  → general condition good, discharge.

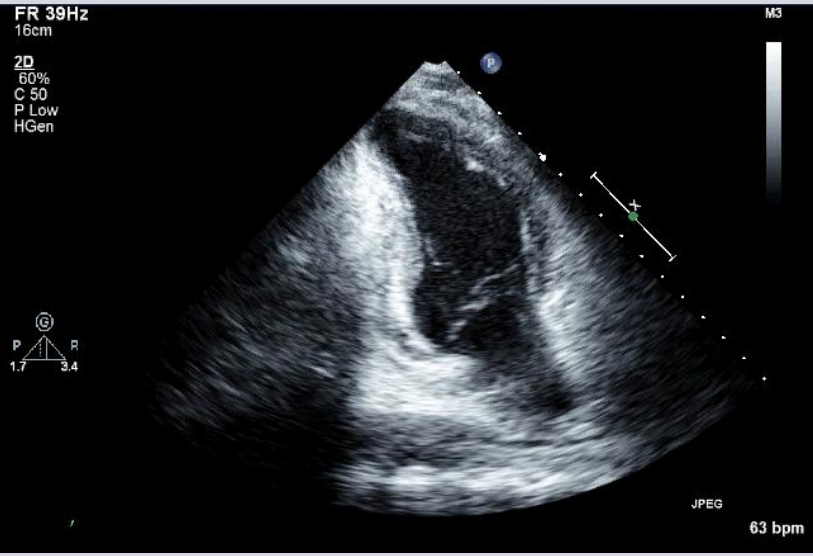
- OPD F/U with echocardiography

- 6 months later

- 2D echo:

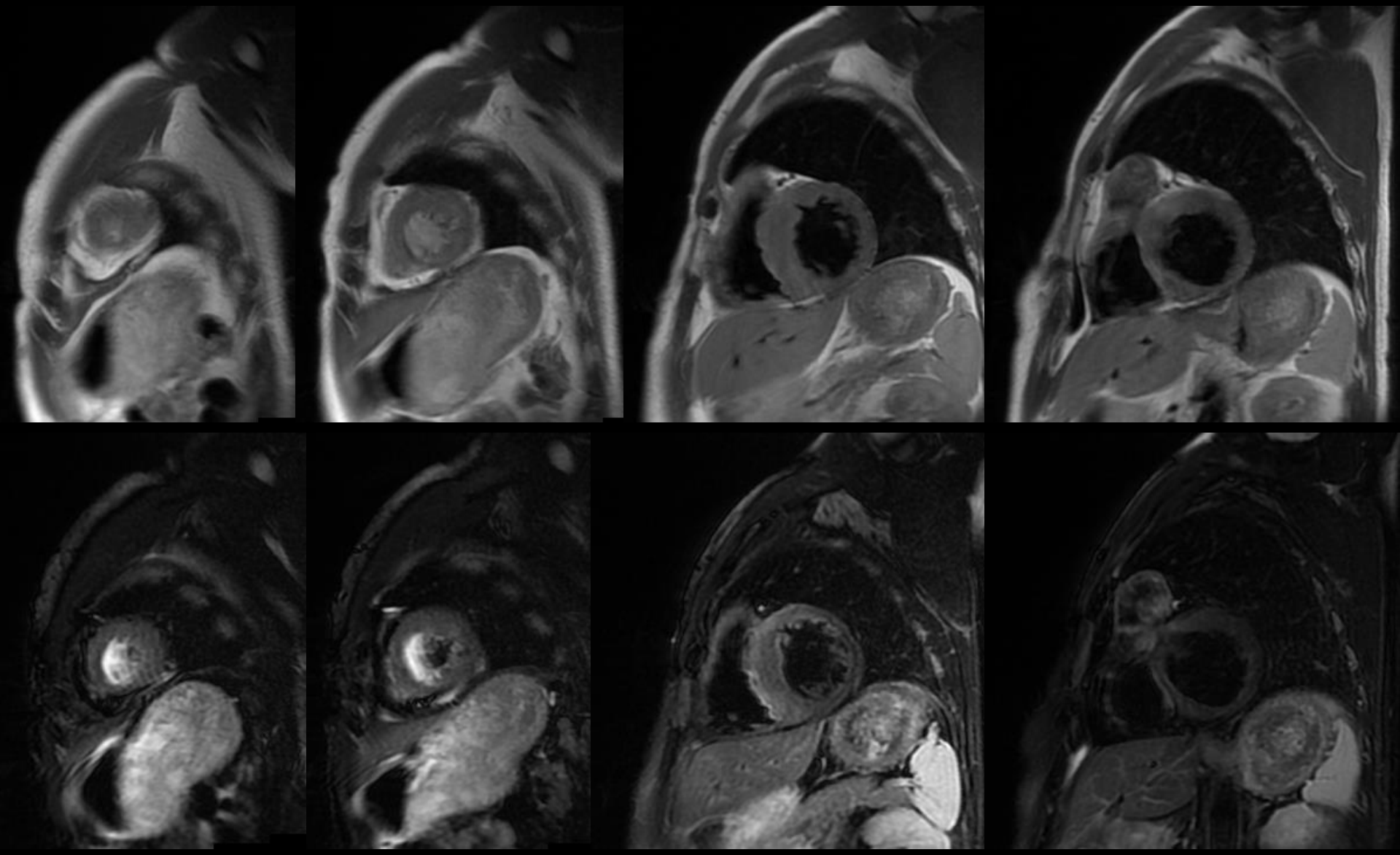
2010/11/23



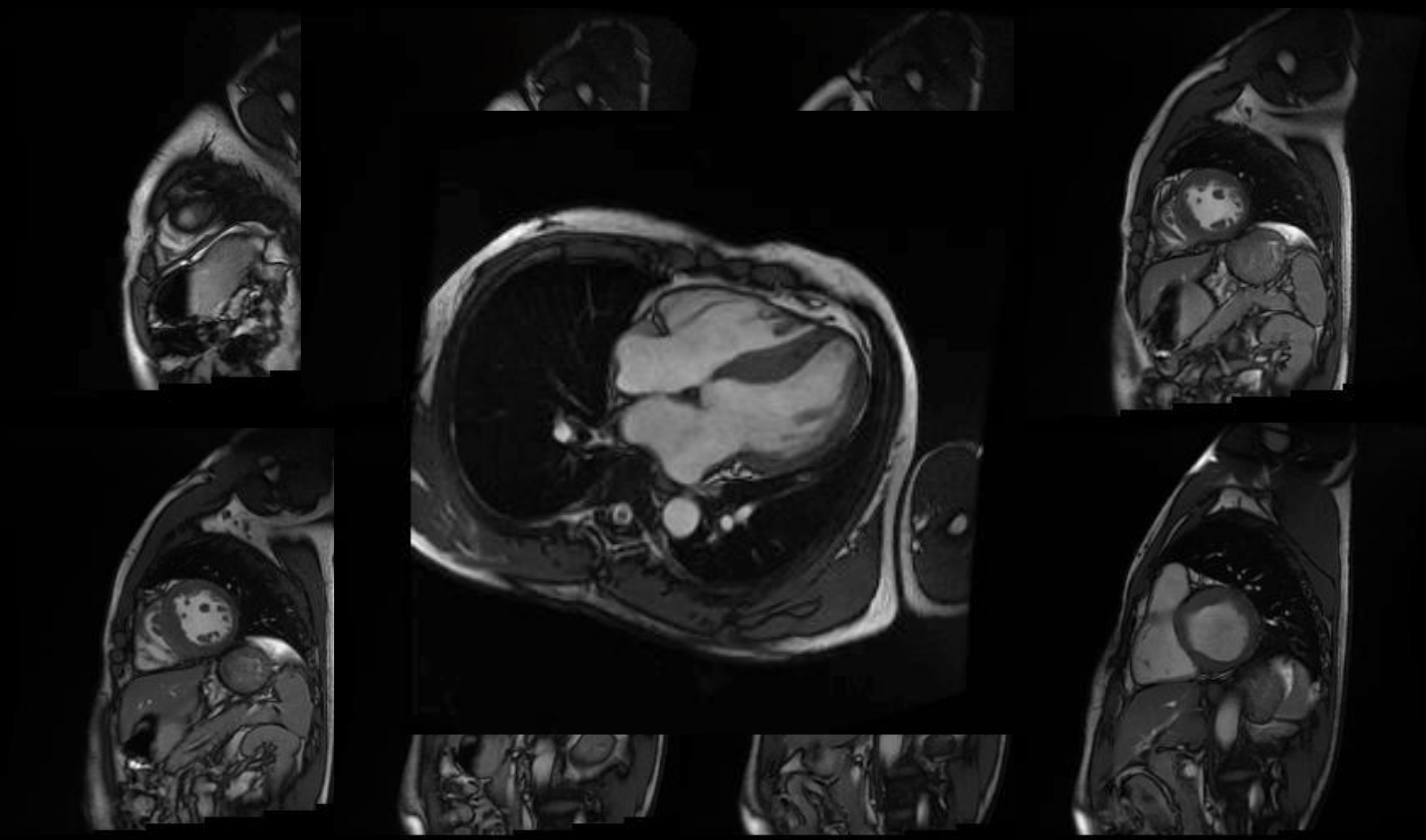


2010/5/24

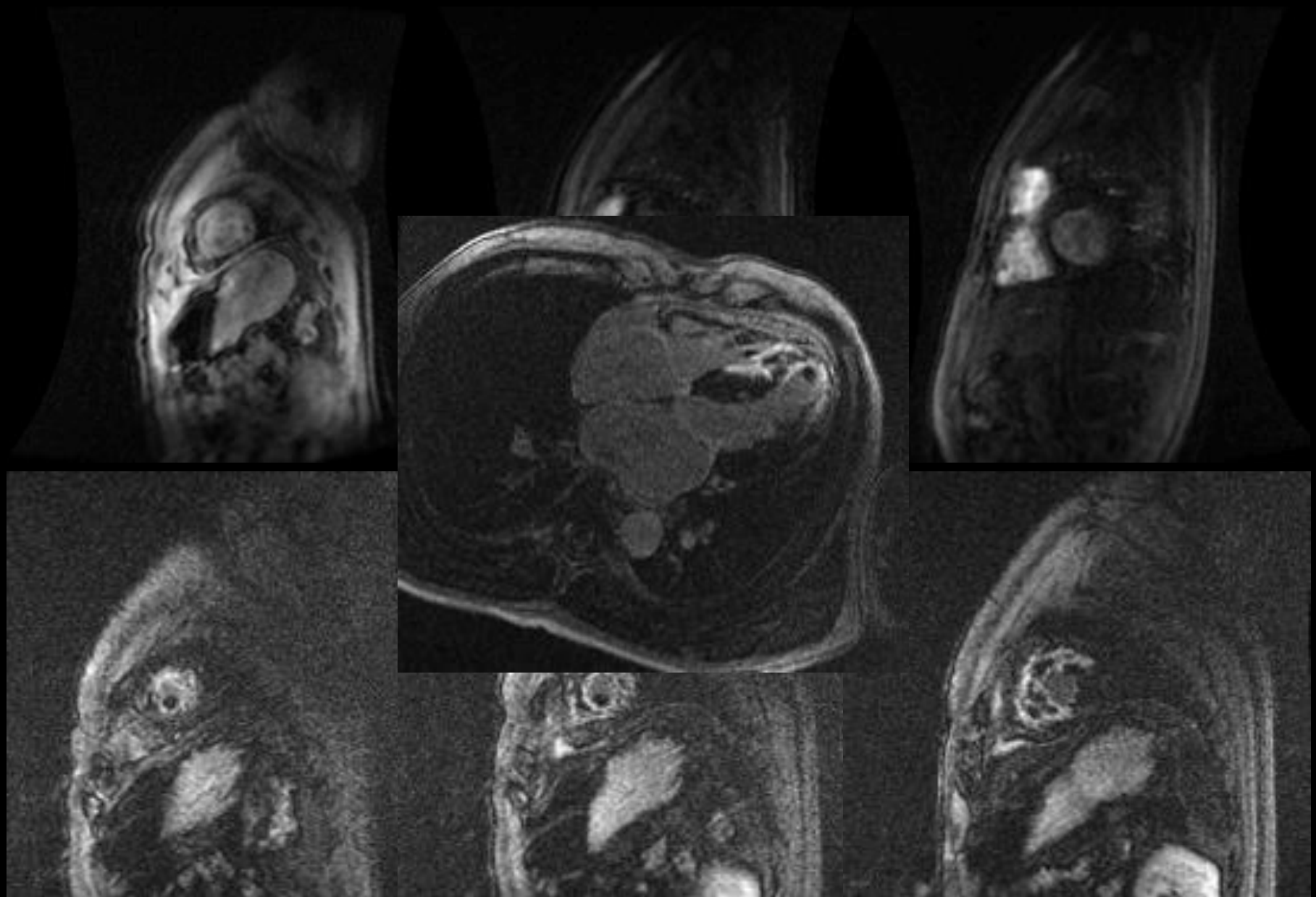
- MR; Double IR T1, Double IR T2 FAT



- MR; SA cine



- MR; Rest perfusion, DE



- Septal HCM and transmural myocardial infarction in LV apex without significant atherosclerosis of coronary arteries.

- **What is the most likely diagnosis?**
  - Apical HCM → massive myocardial fibrosis, ischemic change → apical aneurysm, LV thrombus
  - HCM induced pressure overload → **apical aneurysm**, apical thrombus formation
  - Apical infarction due to **acute coronary artery obstruction from microthrombus** of ruptured atherosclerotic plaque underlying HCM → LV thrombus

Burned-out Phase of Apical HCM

- Characterized by systolic dysfunction, luminal dilatation, wall thinning
- Thought to be due to ischemia that results from reduced capillary density, hyperplasia of the arterial media, increased perivascular fibrosis.
- Hypokinesia can occur **after an acute myocardial infarction** or it can **develop gradually without a clinical infarction**.

AJR 2007; 189:1335–1343

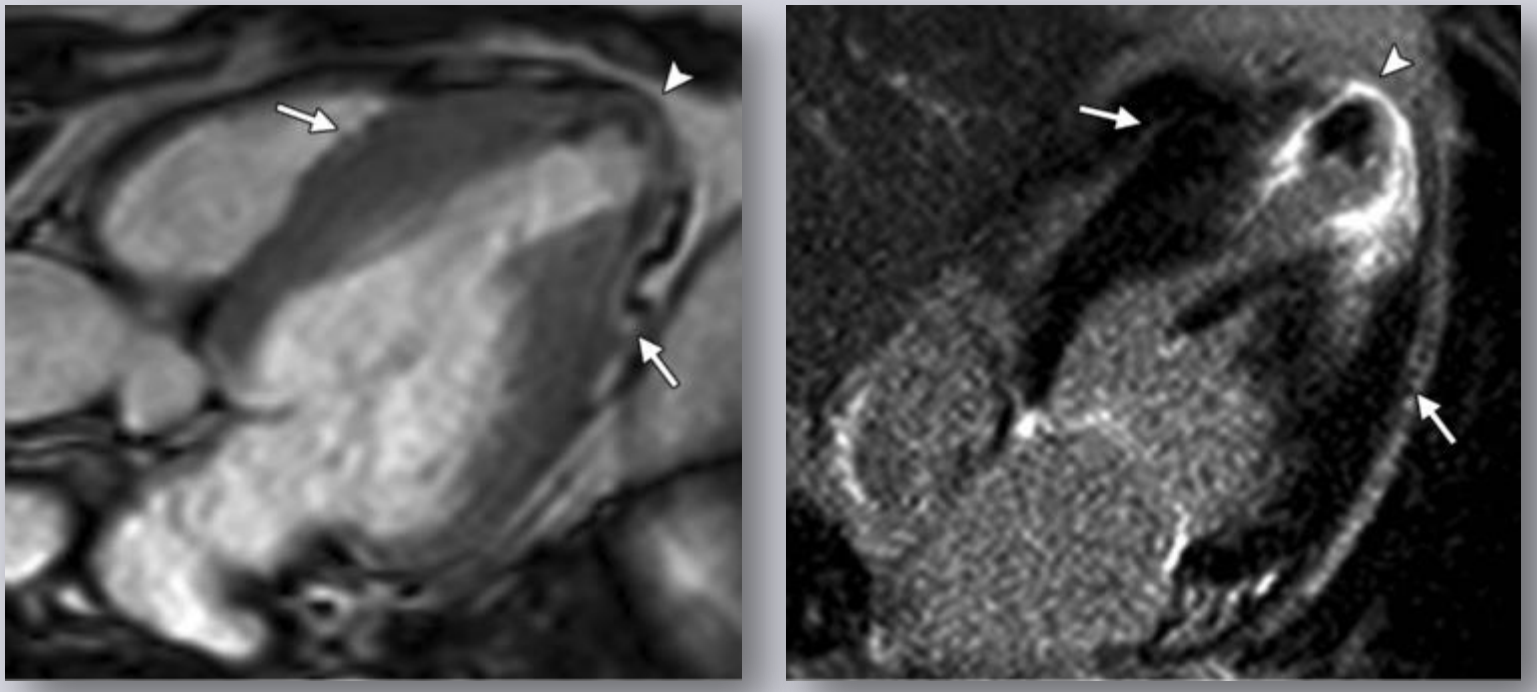
- MR: dilated-hypokinetic evolution of HCM
  - Thin-walled apical aneurysm formation
  - DE: transmural enhancement
    - that extends into substantial area of the contiguous **interventricular septum and LV free wall (not associated with coronary vascular territory)**
  - Thrombus (frequently associated) : low signal mass with lack of enhancement

- CAG: usually occurs in normal epicardial coronary arteries

RadioGraphics 2010; 30:1309–1328

AJR 2007; 189:1335–1343

F/43, Midventricular to apical HCM in the burned-out phase



- Usually progressed to a heart failure unresponsive to therapy with medications

RadioGraphics 2010; 30:1309–1328