

# A Case Of Unusual Multiple Embolic Acute Myocardial Infarct

Presenter: *Young Jin, Youn, MD.*

1<sup>st</sup> PCI Operator: *Seung Hwan, Lee*

2<sup>nd</sup> PCI Operator: *Junghan, Yoon*

Yonsei University, Wonju College of Medicine  
Wonju Christian Hospital



# Case - HKS 56/M

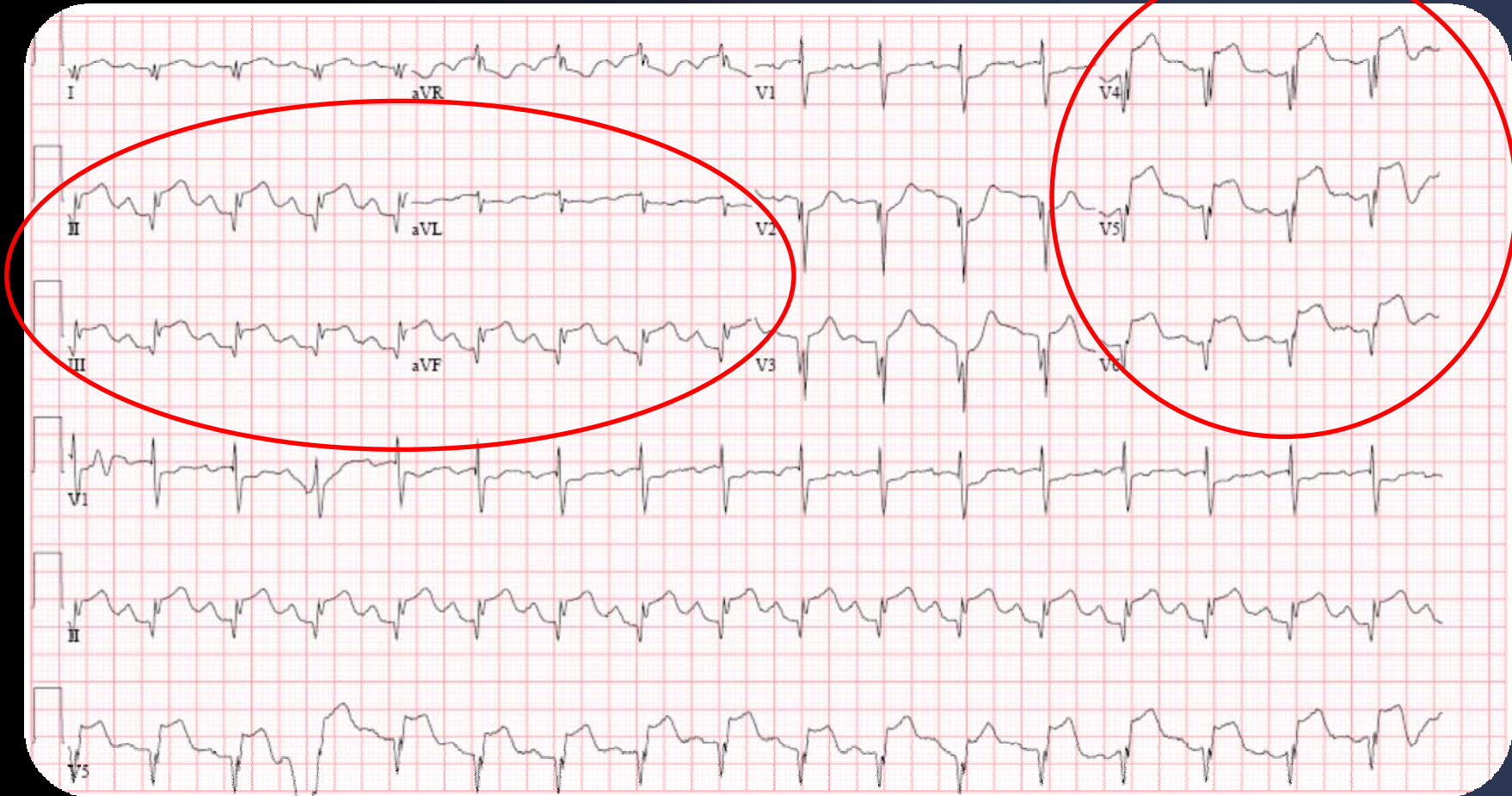
- C.C:
  - Ongoing chest pains for 1 hour
  - during walking down the mountain
- P/Hx:
  - No known history of DM, HTN and dyslipidemia
  - 20 years ago **DVT Dx.** → warfarin
  - 15 years ago **PTE Dx.** → warfarin 5mg qd (Intermittent medication)
  - Smoking: 30 pack-years (1 pack/day x 30 yrs)
- Risk factors: Smoking
- Clinical Dx: **STEMI, inferolateral, Killip class III**

*Arrival at ER: 09.9.27 15:50*



# Initial ECG

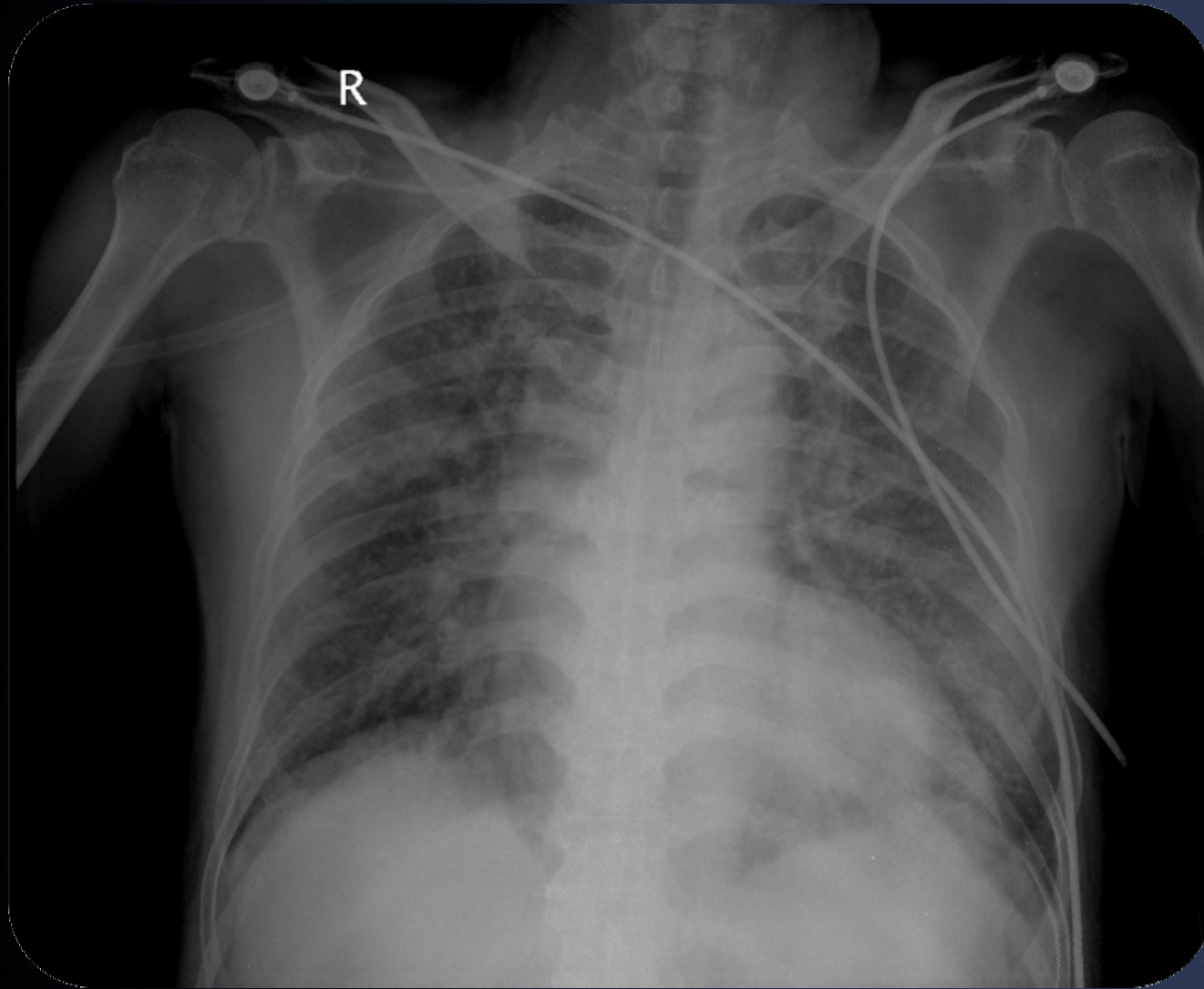
Initial V/S: BP 123/74mmHg, HR 97/min



09.9.27 15:51



# Initial Chest X-ray

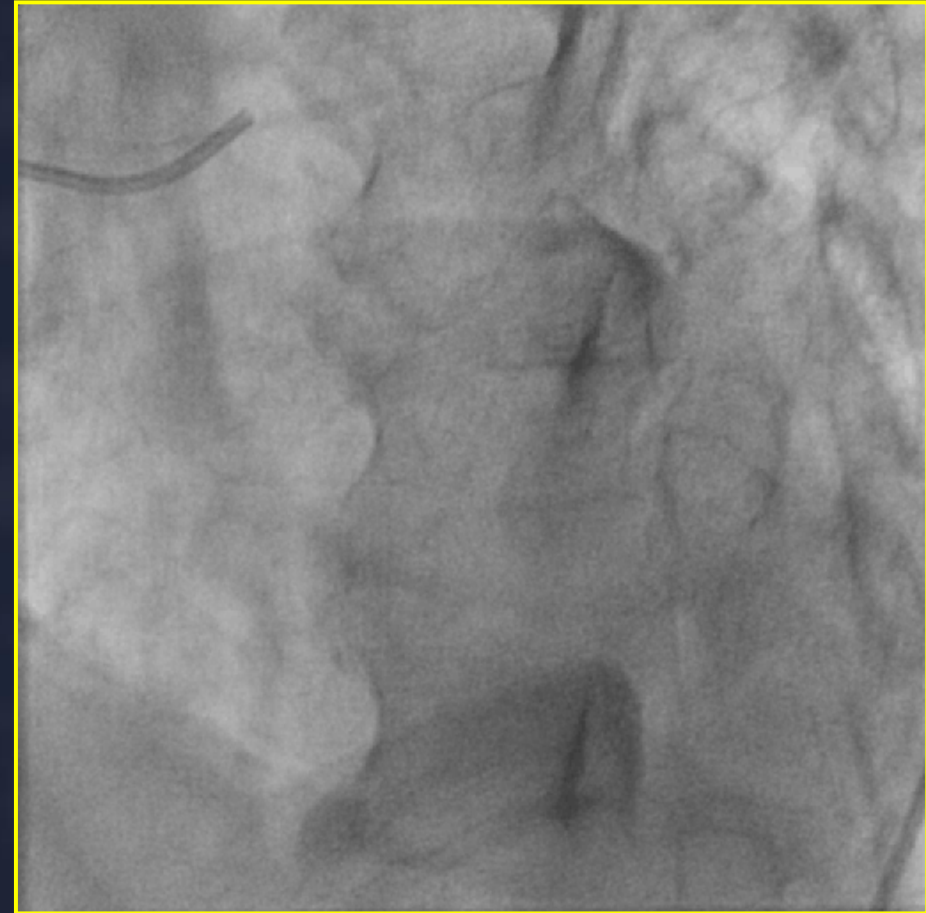
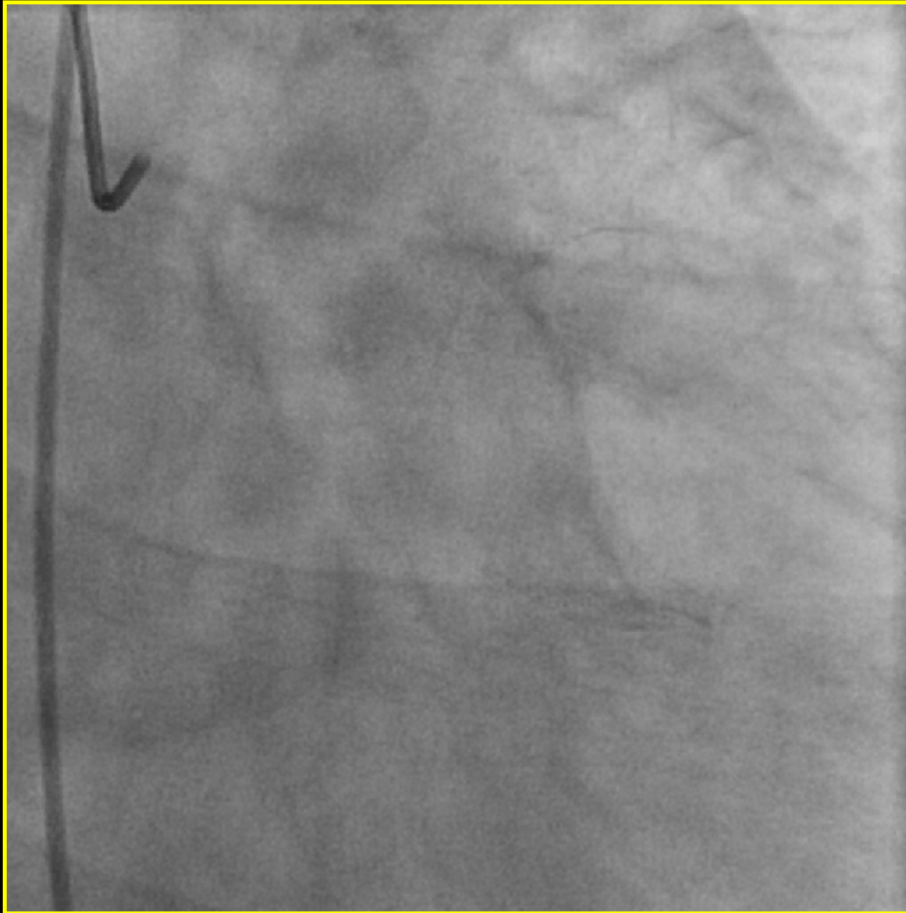


# Initial laboratory findings

- CBC: 17760-14.2/42.0-298000
- Na/K/Cl/CO<sub>2</sub>, mmol/L: 142/4.3/109/18.0
- BUN/Cr, mg/dl: 28/1.2
- Glucose, mg/dl: 254 (HbA1c: 5.9%)
- **D-dimer, ng/dl: 448 (ref.: <280)**
- PT, INR: **1.08**
- BNP, pg/ml: 17.9
- **CK-MB, ng/ml: 62.11 (ref.: < 5.0)**
- **Troponin I, ng/ml: 28.226 (ref.: < 0.78)**



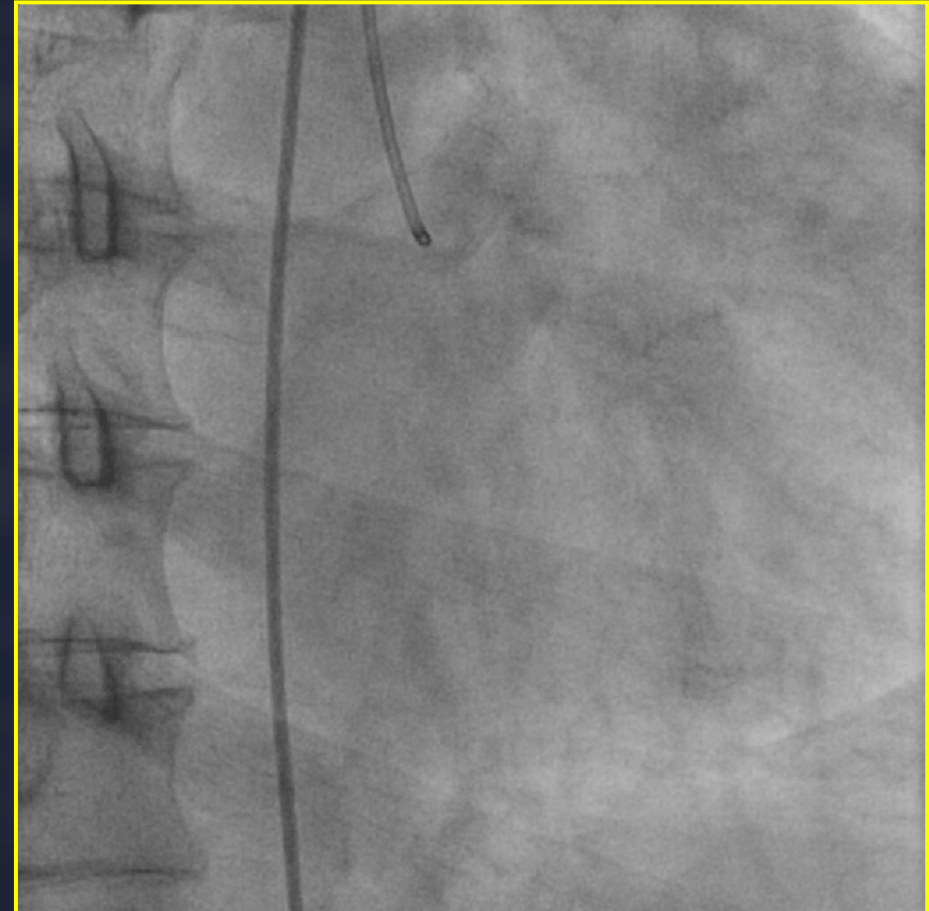
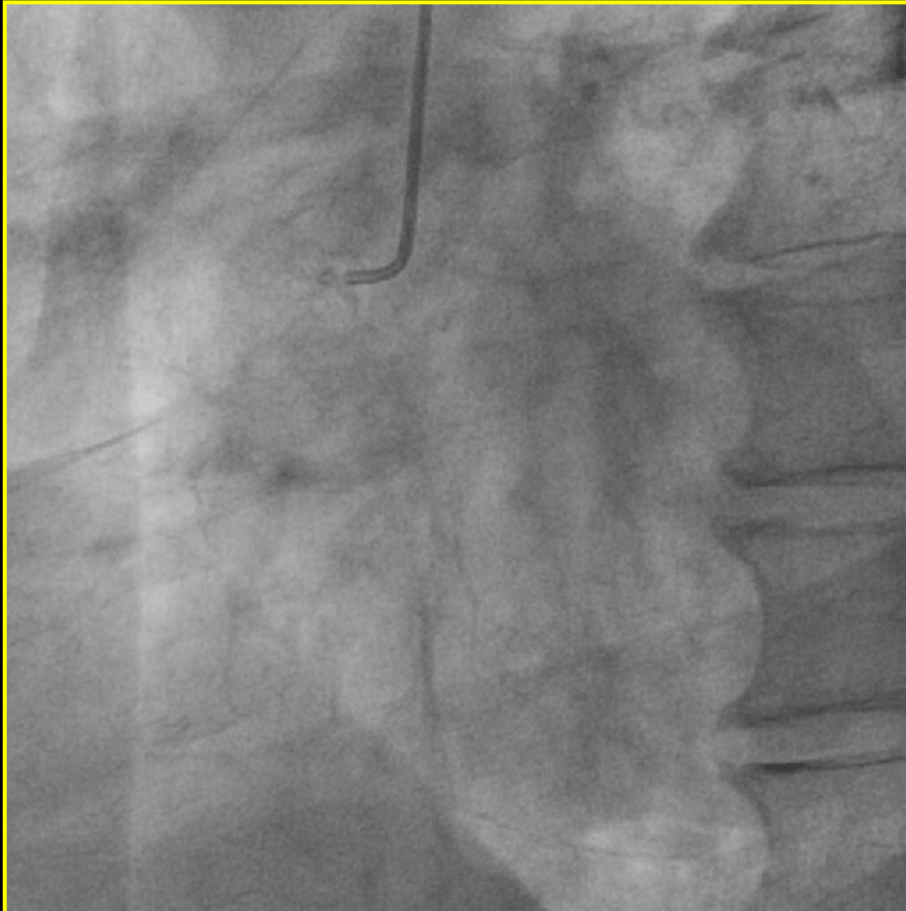
# LCA angiography



- First, right radial approach was tried but puncture was failed, so right femoral was punctured and then 7.5Fr 12cm sheath was inserted.
- By using 5Fr. JL and JR 4.0cm angio-catheter, coronary angiography was done.
- There were total occlusion at **distal LCX artery** and **distal intermediate branch**. *Cath Lab arrival time: 09.9.27 16:15*



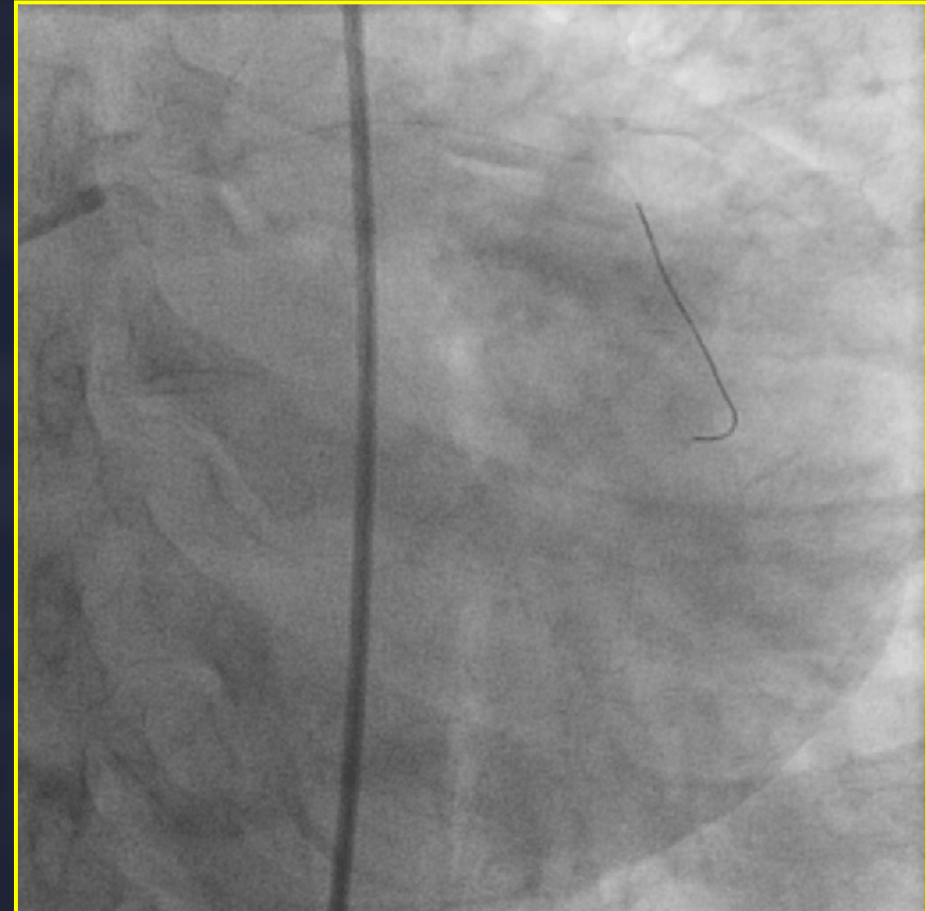
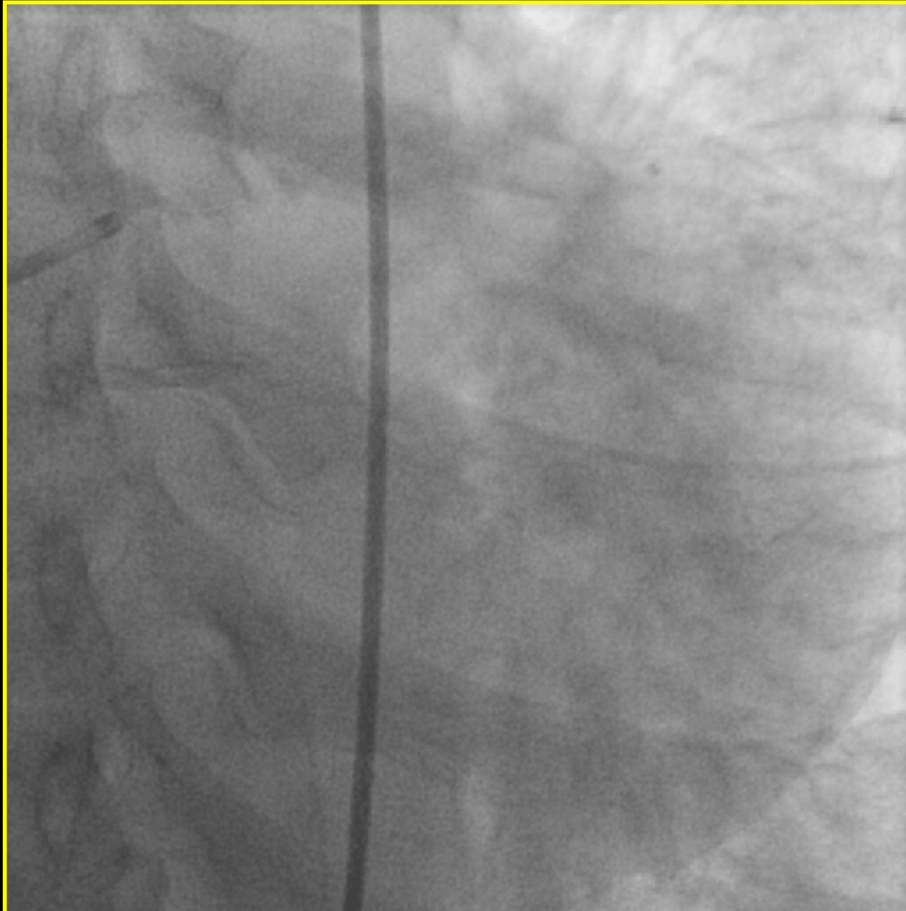
# RCA angiography



- RCA showed no significant stenosis and no collateral flow to left coronary artery.



# PCI of Ramus

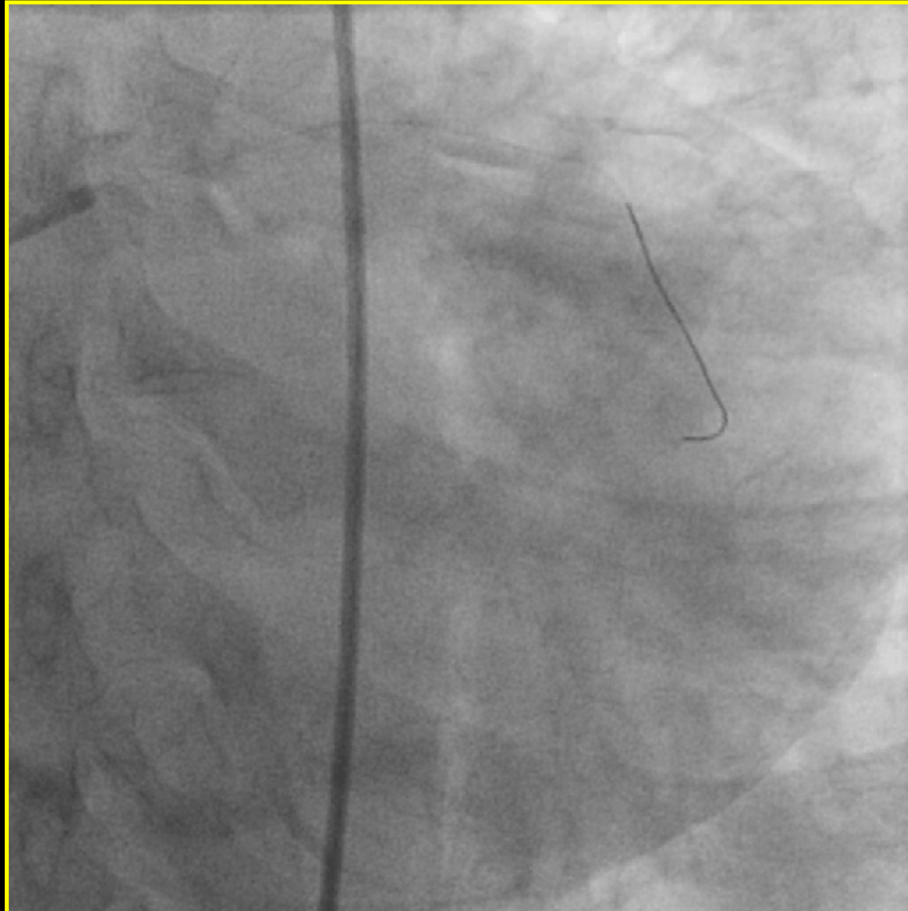
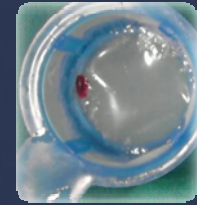


- After changing to 6Fr. XB 3.5cm guiding catheter, guidewire FIELDER was passed through intermediate branch.



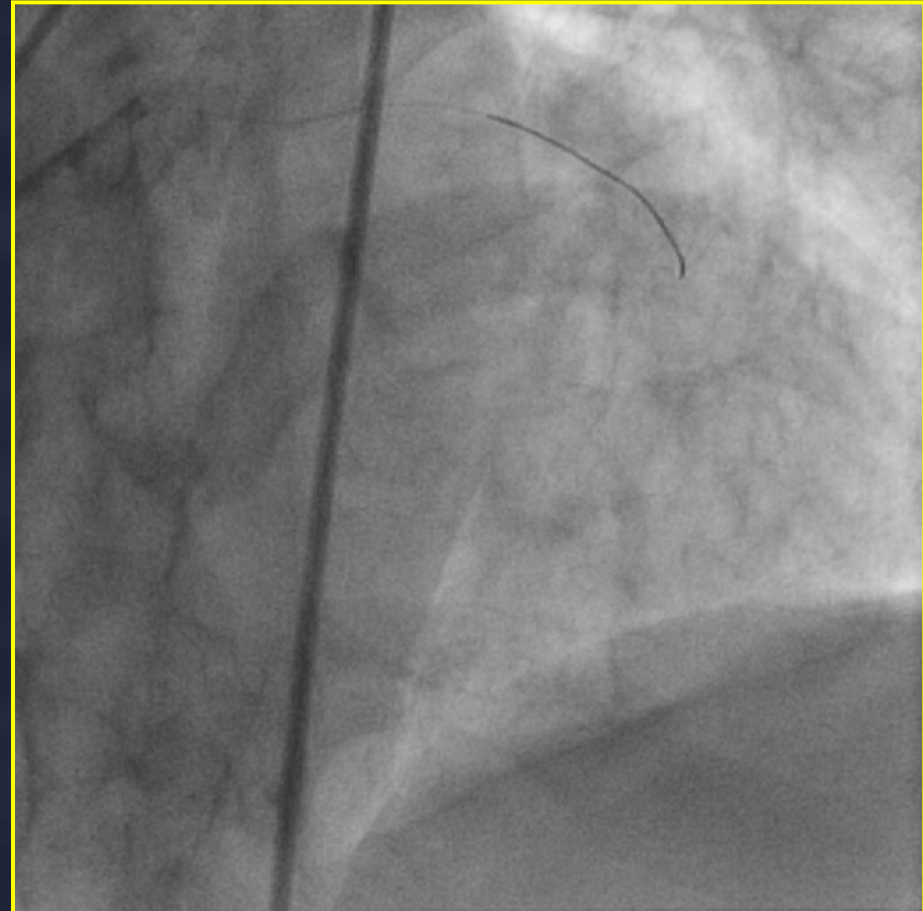


# Aspiration at Ramus



Before thrombus aspiration

- Thrombus was manually aspirated by THROMBUSTER II. Red thrombi was observed.
- Because the territory of distal part was small, PCI for intermediated branch was stopped.

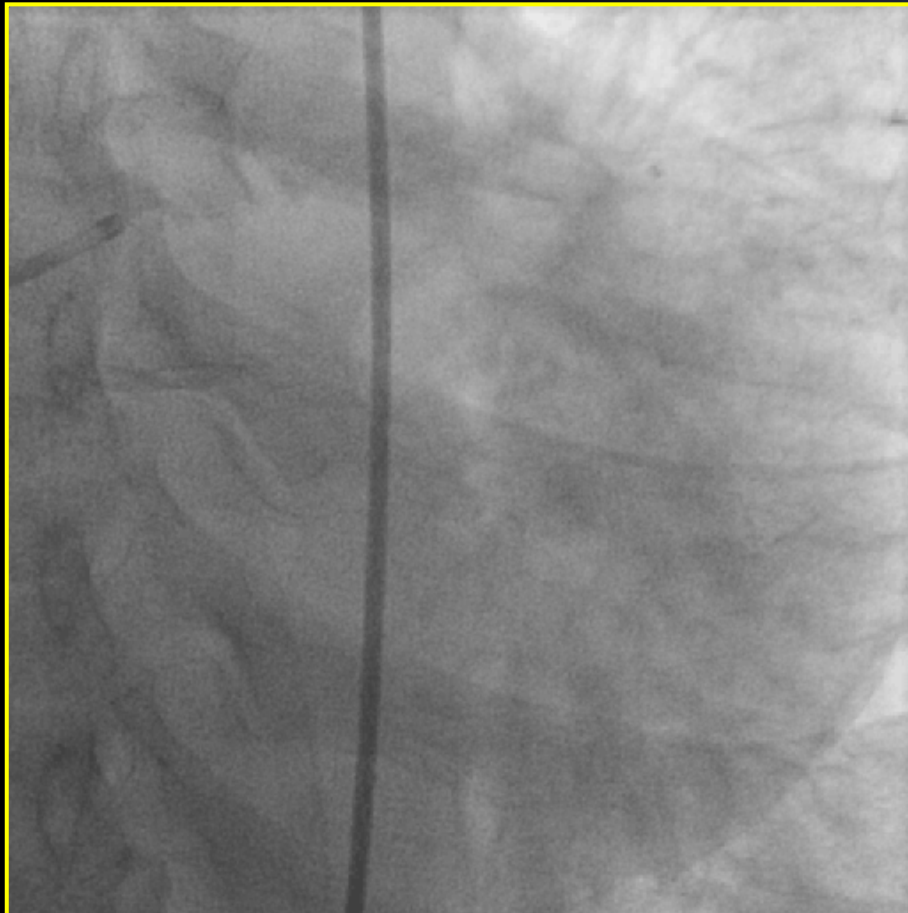
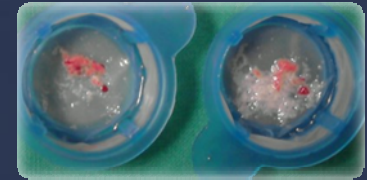


After thrombus aspiration

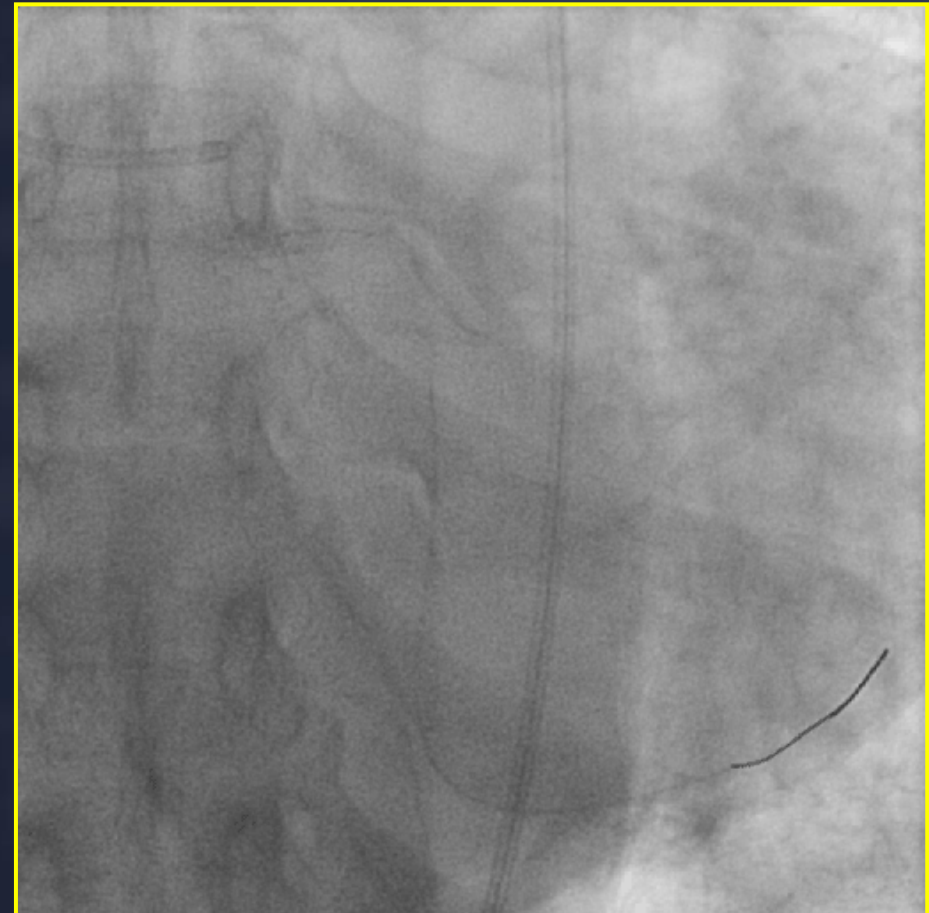
*First balloon time: 09.9.27 16:42 (DTB: 52 min)*



# Aspiration at LCX



Before thrombus aspiration



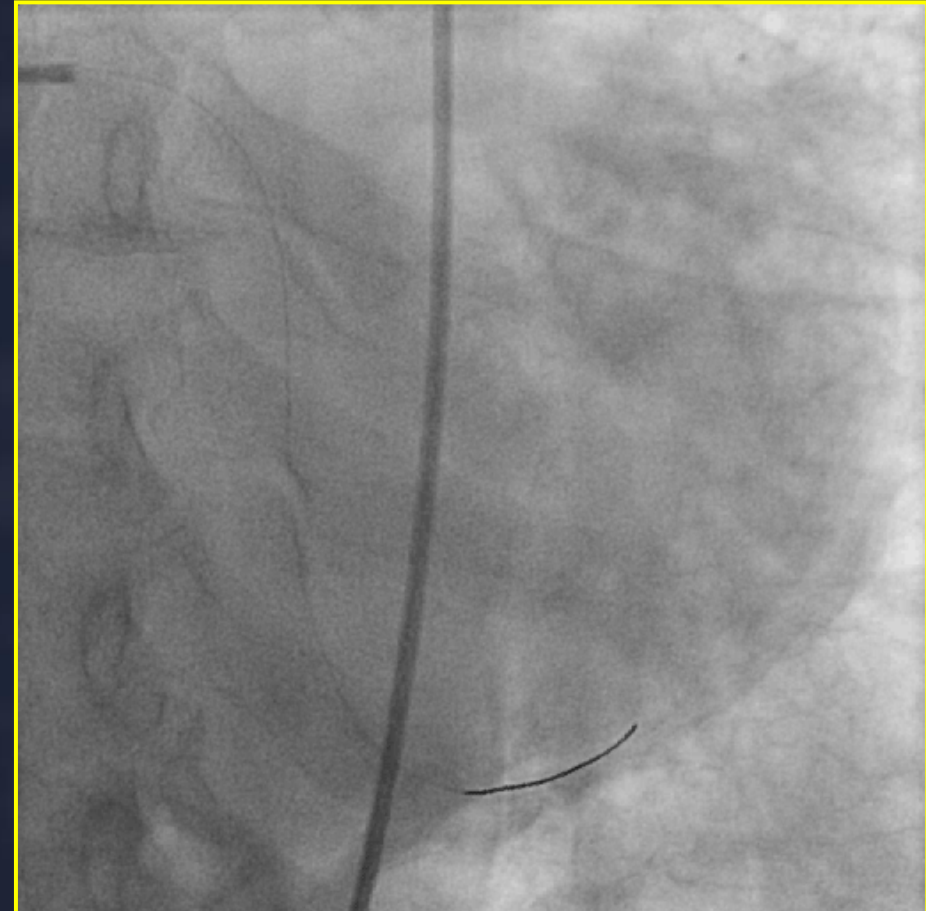
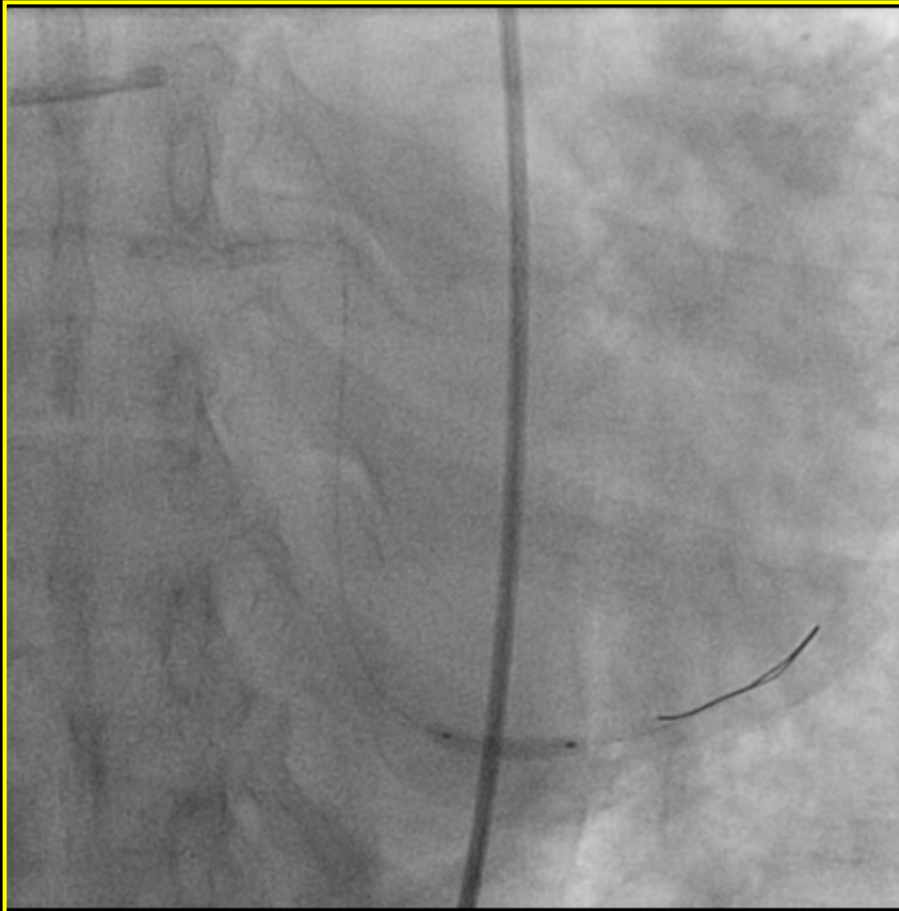
After thrombus aspiration

- 0.035" guidewire, FILEDER was passed through LCX and then thrombus was aspirated three times by using THROMBUSTER II (picture at next page).

*Second balloon time: 09.9.27 16:48 (DTB: 58 min)*



# PCI at LCX



- Because we could observe haziness at that lesion, balloon, NERO 2.0mm x 15mm was inflated with 18 atm. After ballooning, there was also embolization to other branch. So **GIIb/IIIa inhibitor**, Clotinab was injected.
- Although result was suboptimal, PCI was stopped and patient was stable and moved to ward.

*End of PCI: 09.9.27. 17:03*



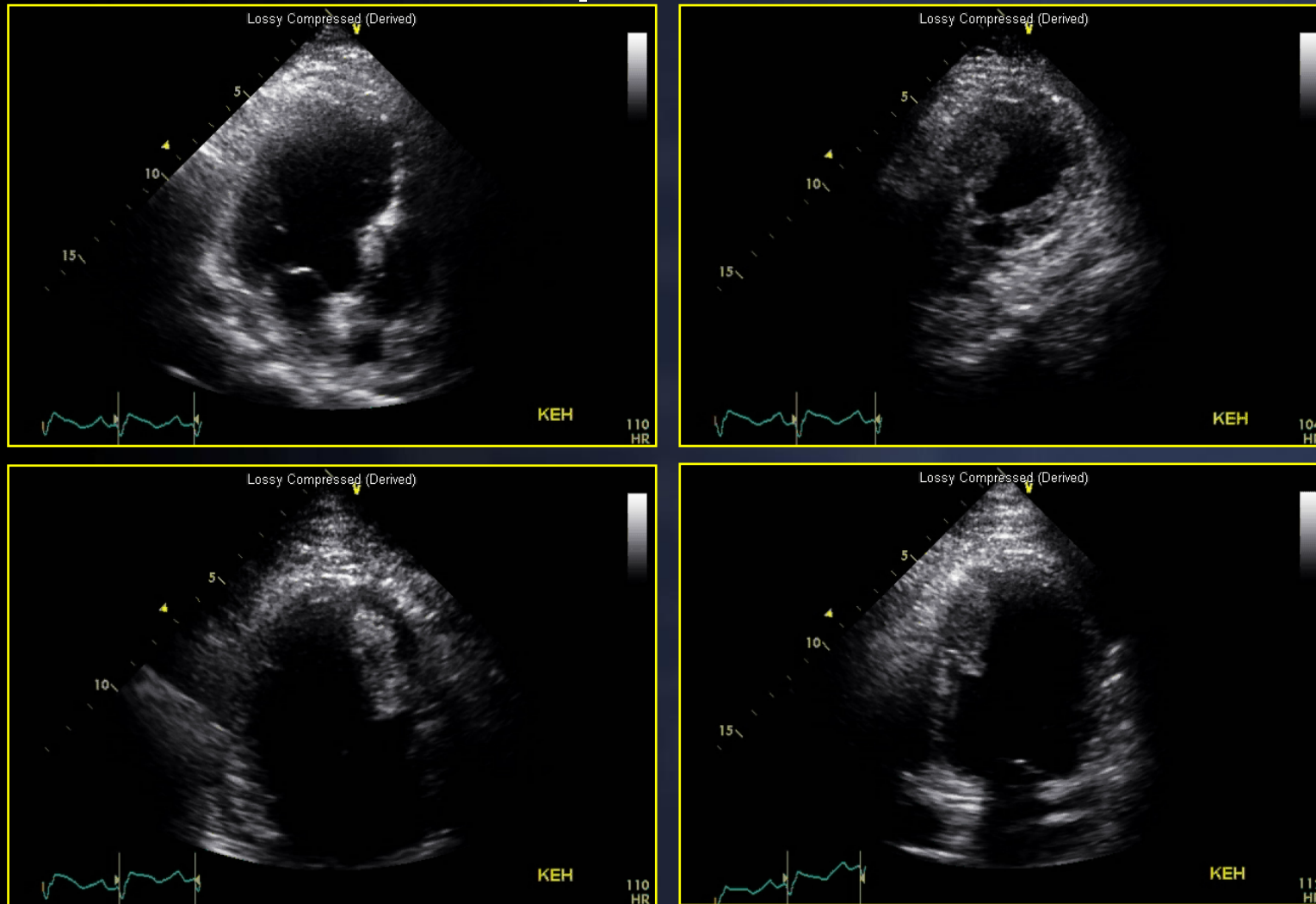
# Question.

**What could be the cause of multiple embolic infarct?**



# Echocardiography

at **25 hrs after** onset of chest pain



- Echocardiography showed **aneurysmal change** on all segments from mid to apex. EF was about 14%.
- Interestingly, there was a **3.94 x 1.35 cm** sized huge thrombi at IVS of LV.
- **But, RWMA was not correlated with territory of MI. So, DDx between stress-CMP and old MI was needed.**

## What could be the cause of multiple embolic infarct?

- From LV thrombus?
- From Paradoxical emboli from DVT? (Hx of young age onset DVT and PTE)

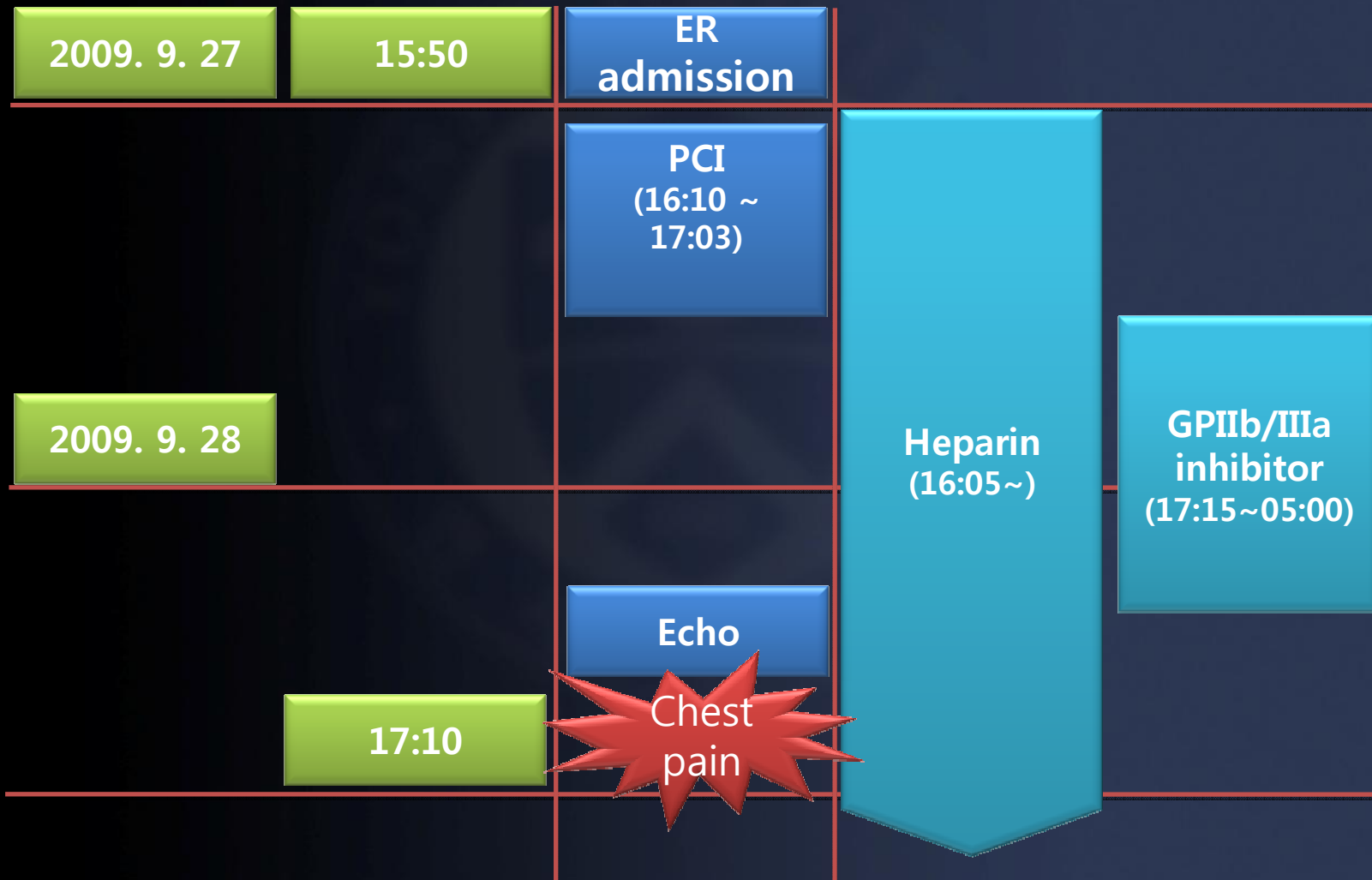
## If the cause was the LV thrombus, what could be the cause of LV thrombus?

- Aneurysmal change due to MI?
- Stress CMP?
- He had thrombophilia? (Hx of young age onset DVT and PTE)

Let's think about it.

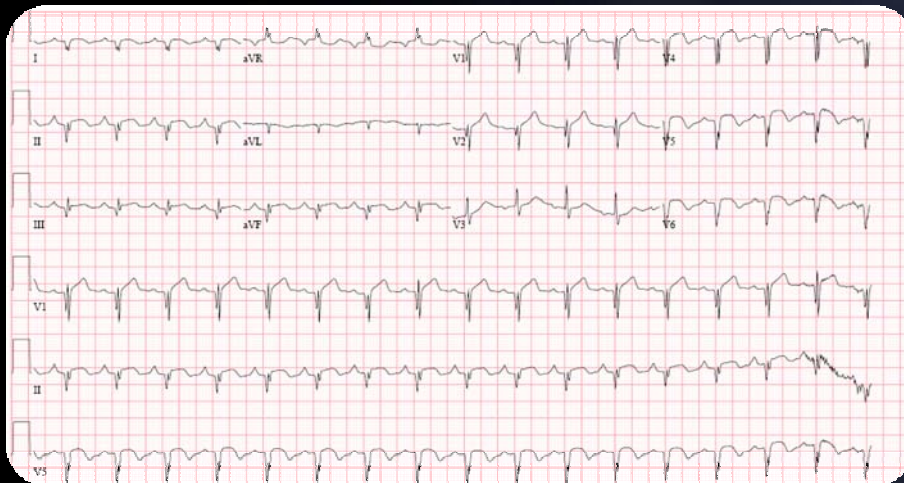


# Progress



# Collapse

- He was stable until 5 pm, next day of PCI.
- He complained chest pain again. He was **suddenly collapsed at 5:45 pm.** Ventricular fibrillation was observed. So DC cardioversion was applied. but PEA was persisted after that.
- Blood pressure was recovered during CPR. So, LM or proximal coronary artery disease were suspected.
- We planned salvage PCI to this patient.



2008.9.28 14:17 (Before collapse)

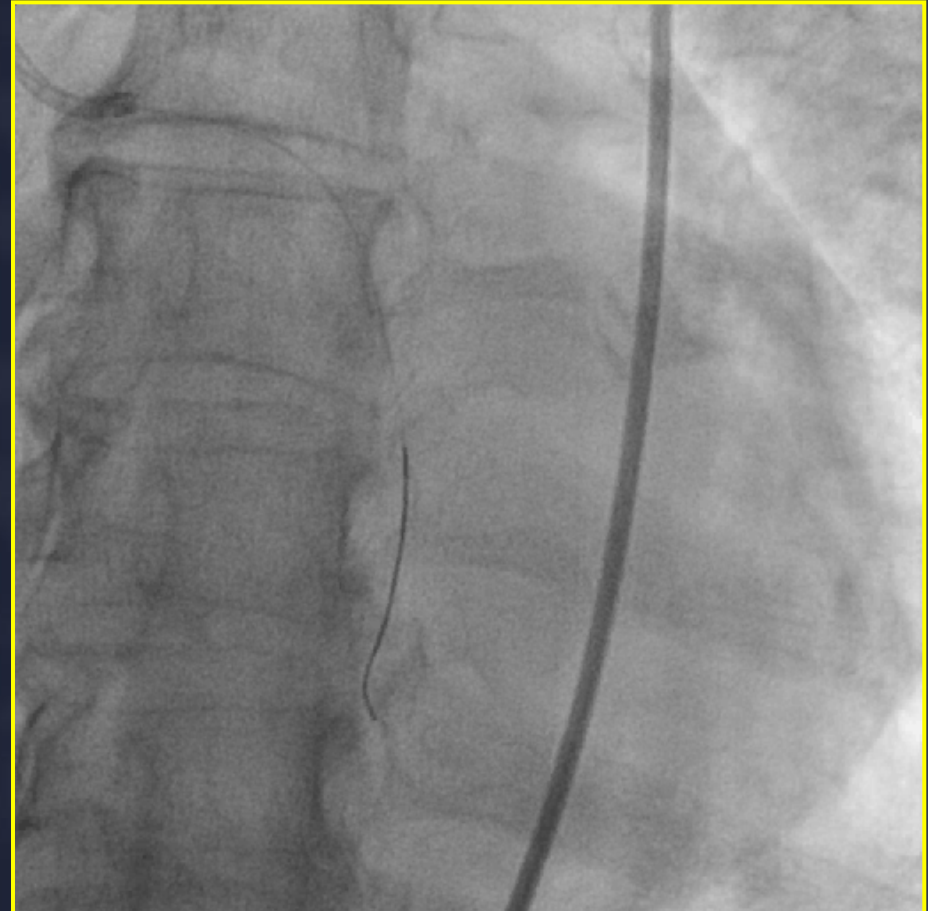
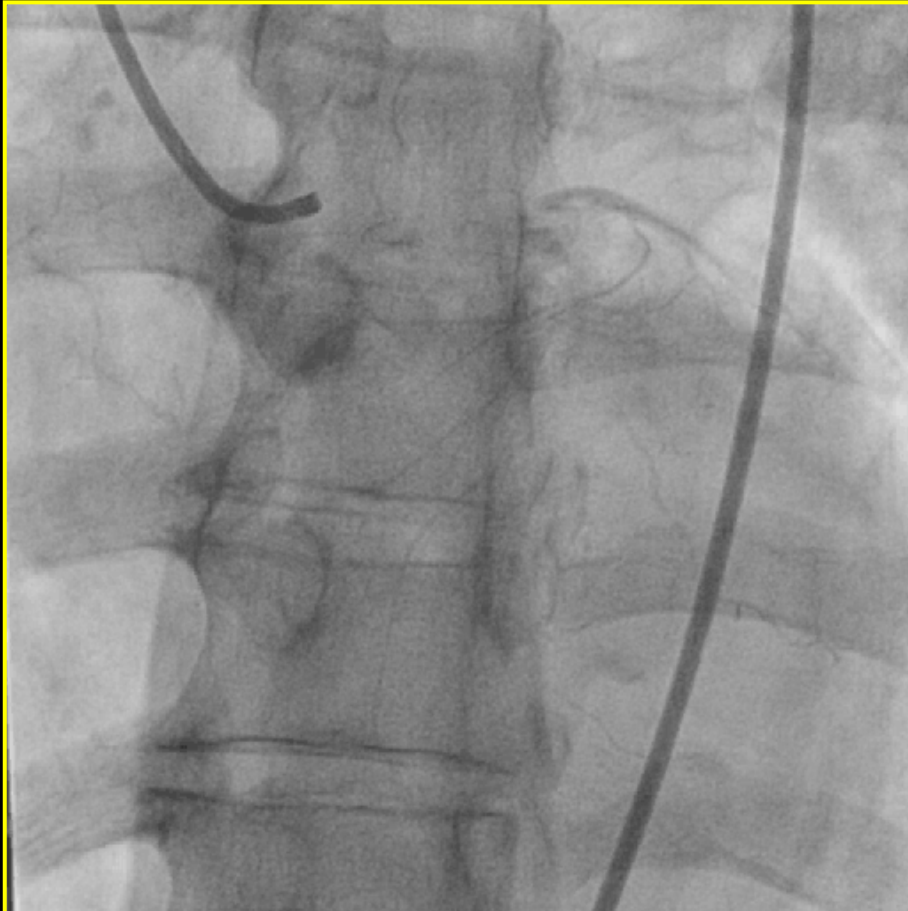


2008.9.28 18:00 (after collapse)





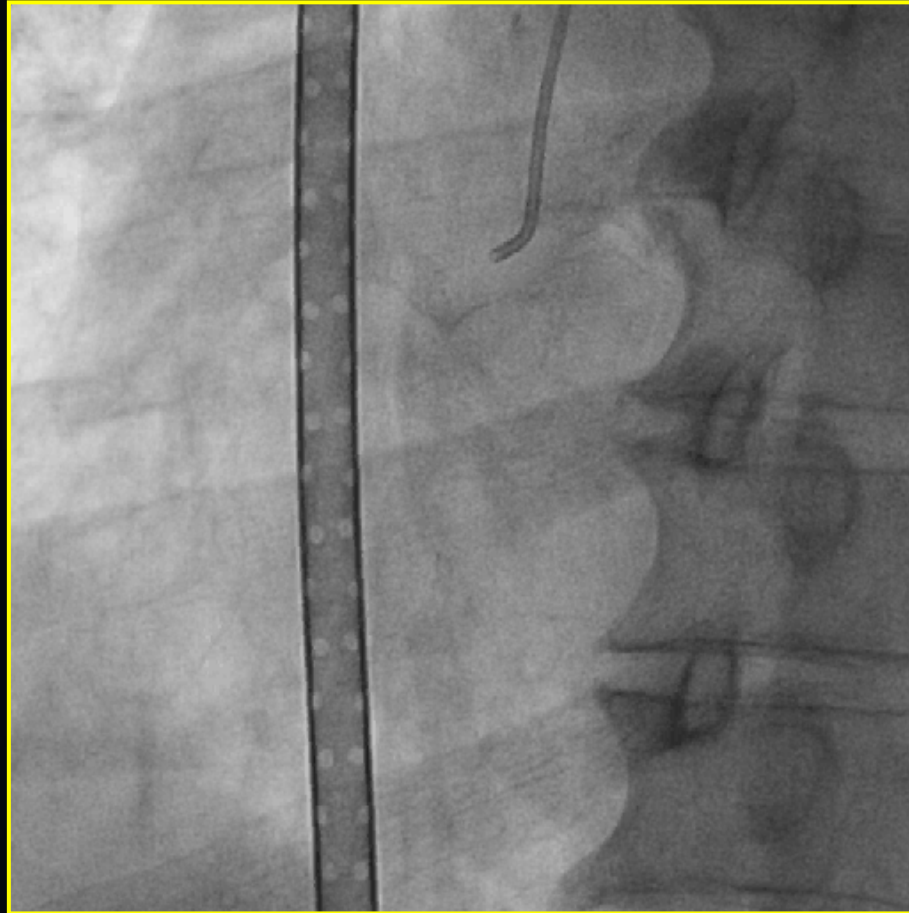
# 2<sup>nd</sup> PCI – LCA angiography



- Patient was hemodynamically unstable, so **emergency bypass system (EBS)** was applied
- Because guiding shot showed significant narrowing at distal LCX, guide-wire, RUNTHROUGH NS was passed through LCX. But following angiography showed no stenosis at suspected lesion but total occlusion at previously embolized distal LCX.
- Intermediate branch also showed **no difference compared with first PCI film.**



# 2<sup>nd</sup> PCI – RCA angiography

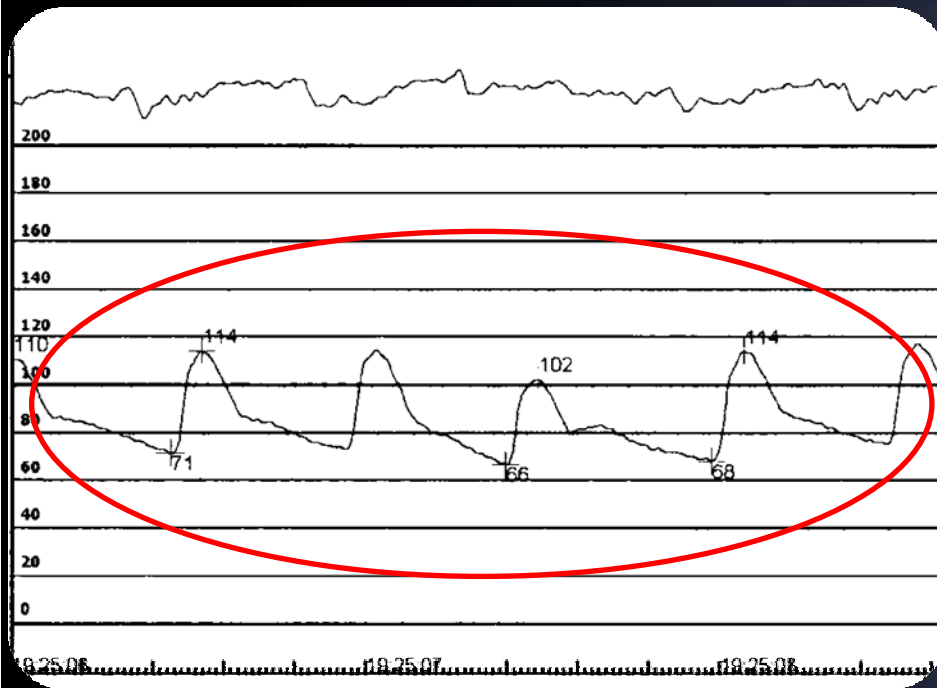


- Because there was no difference at left coronary artery, right coronary angiography was done after engaging 5 Fr. JR 4.0cm catheter.
- But there was **no significant lesion at RCA**, either.

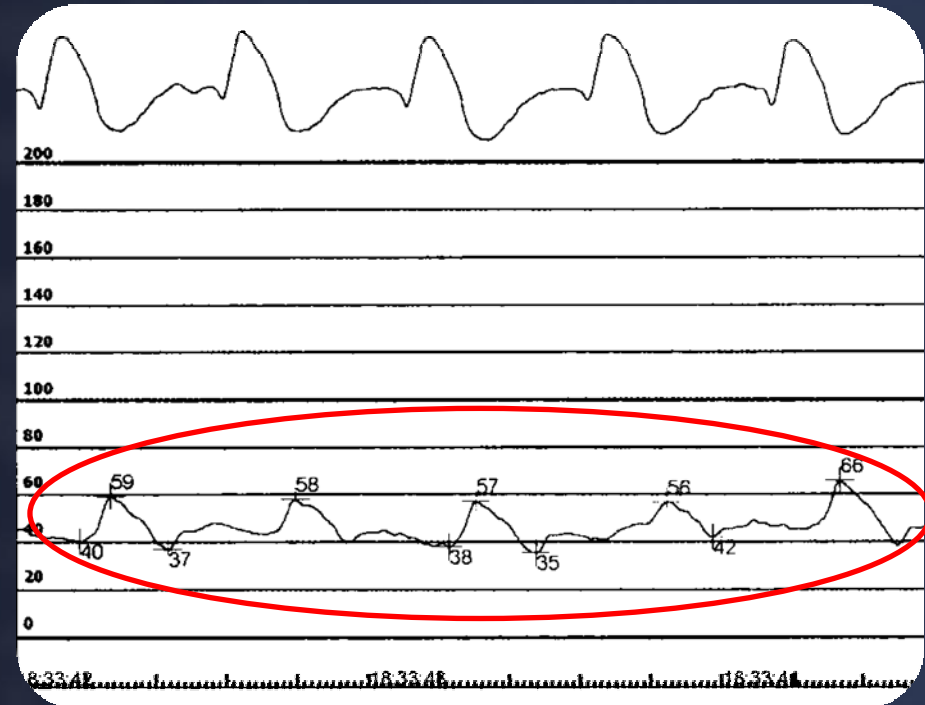


# Blood pressure tracing during PCI

After shot for angiography



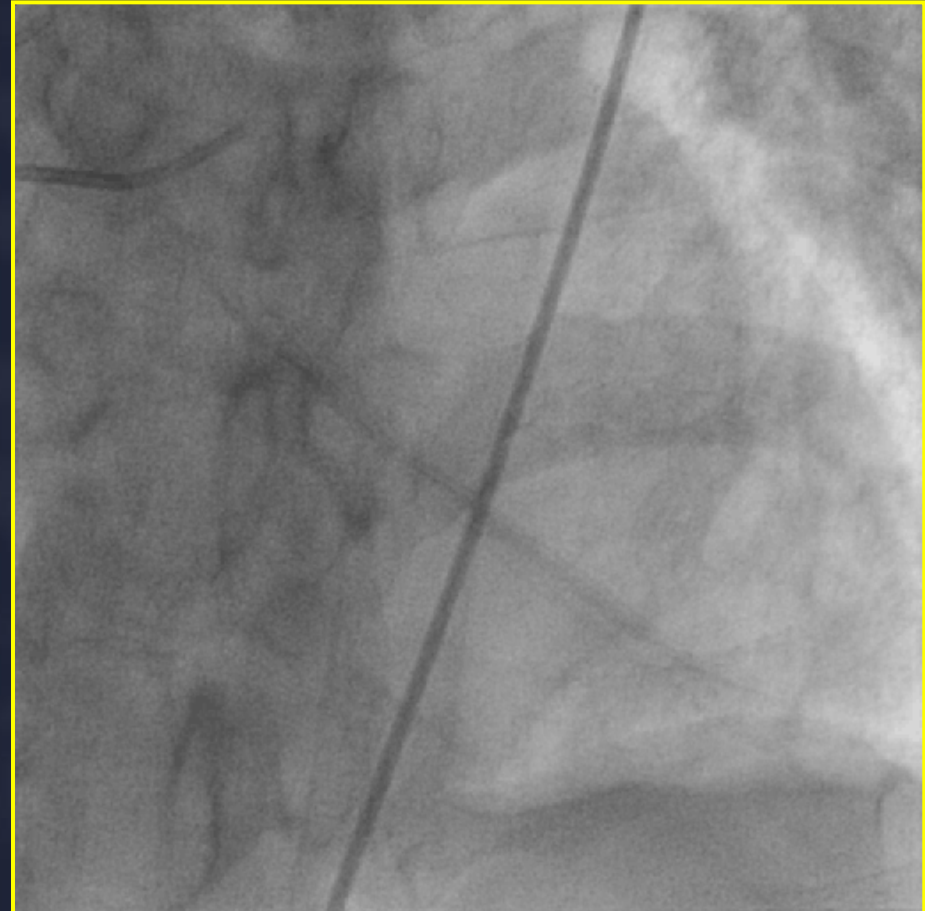
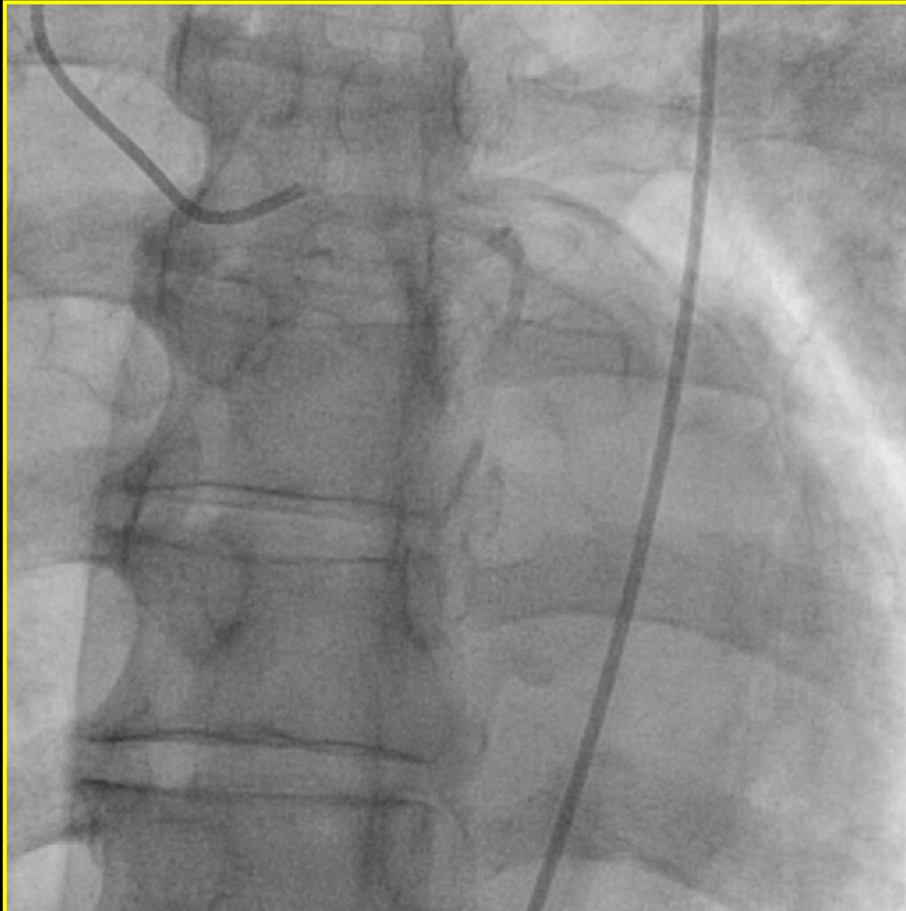
After catheter removal



Very interestingly, patient's blood pressure was recovered after shot. but blood pressure fell after removing catheter. This phenomena was repeated.



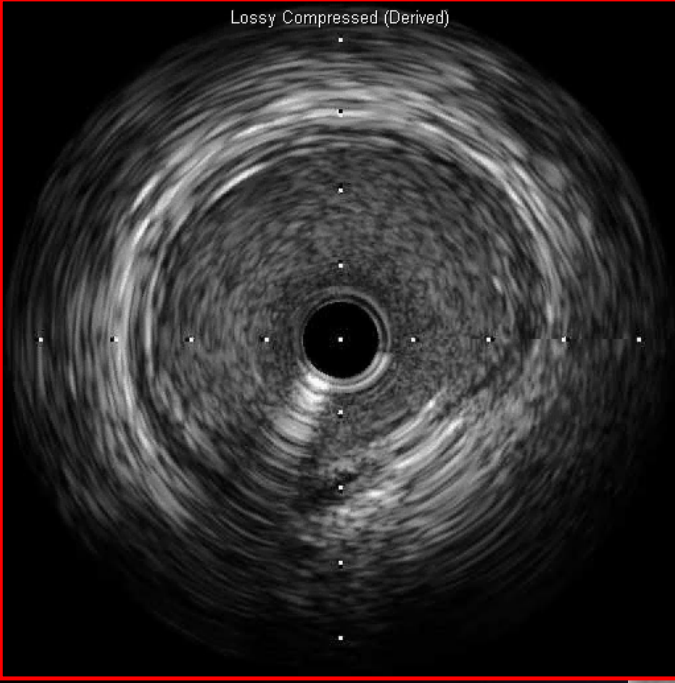
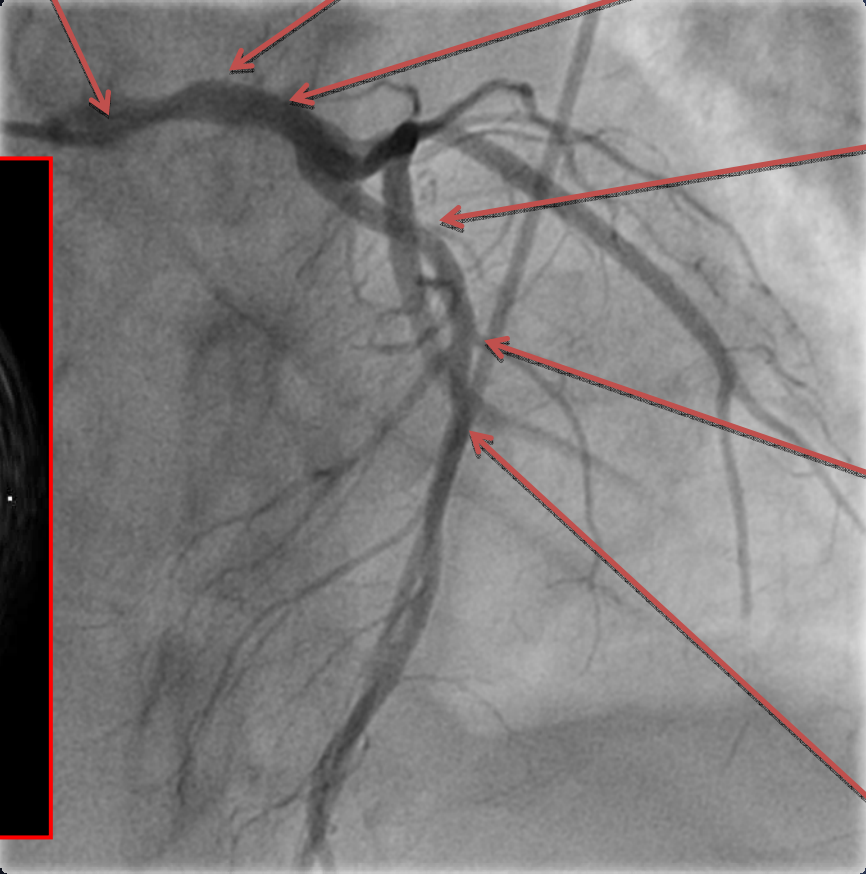
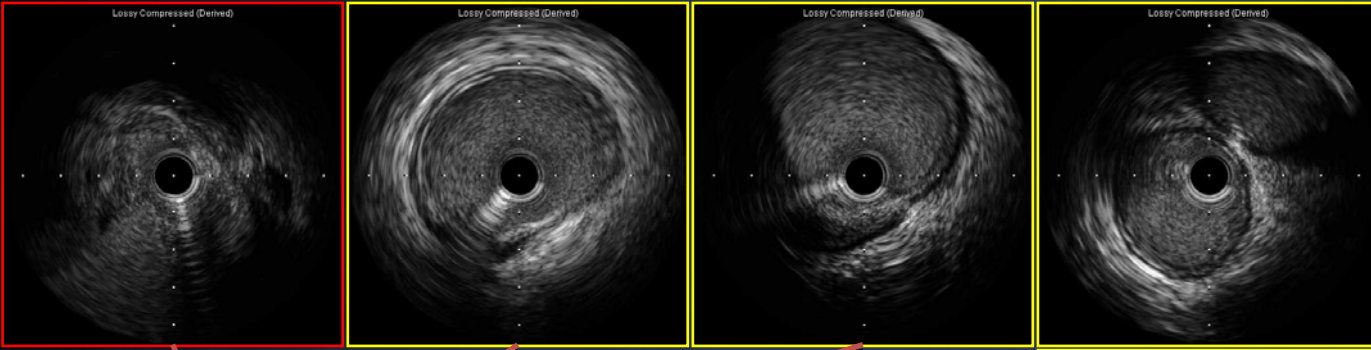
# 2<sup>nd</sup> PCI – LCA angiography



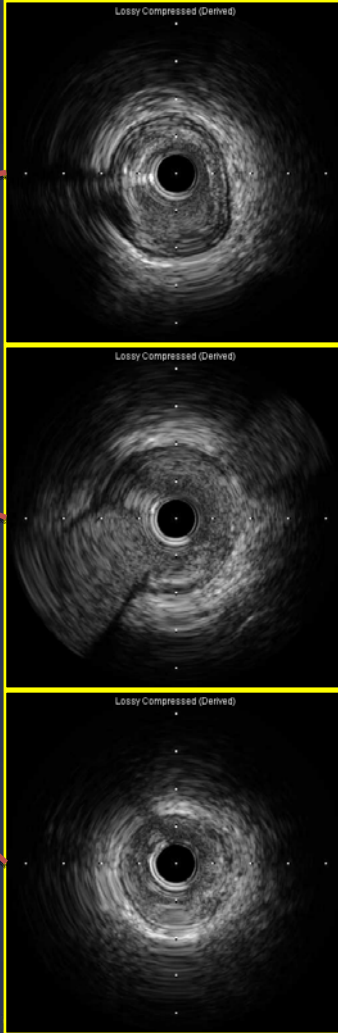
- Because we could not find the reason for collapse.
- Coronary angiography was repeated at left coronary artery.
- There was suspicion of **haziness at proximal LAD**, we decided to do **IVUS**.



# 1st IVUS

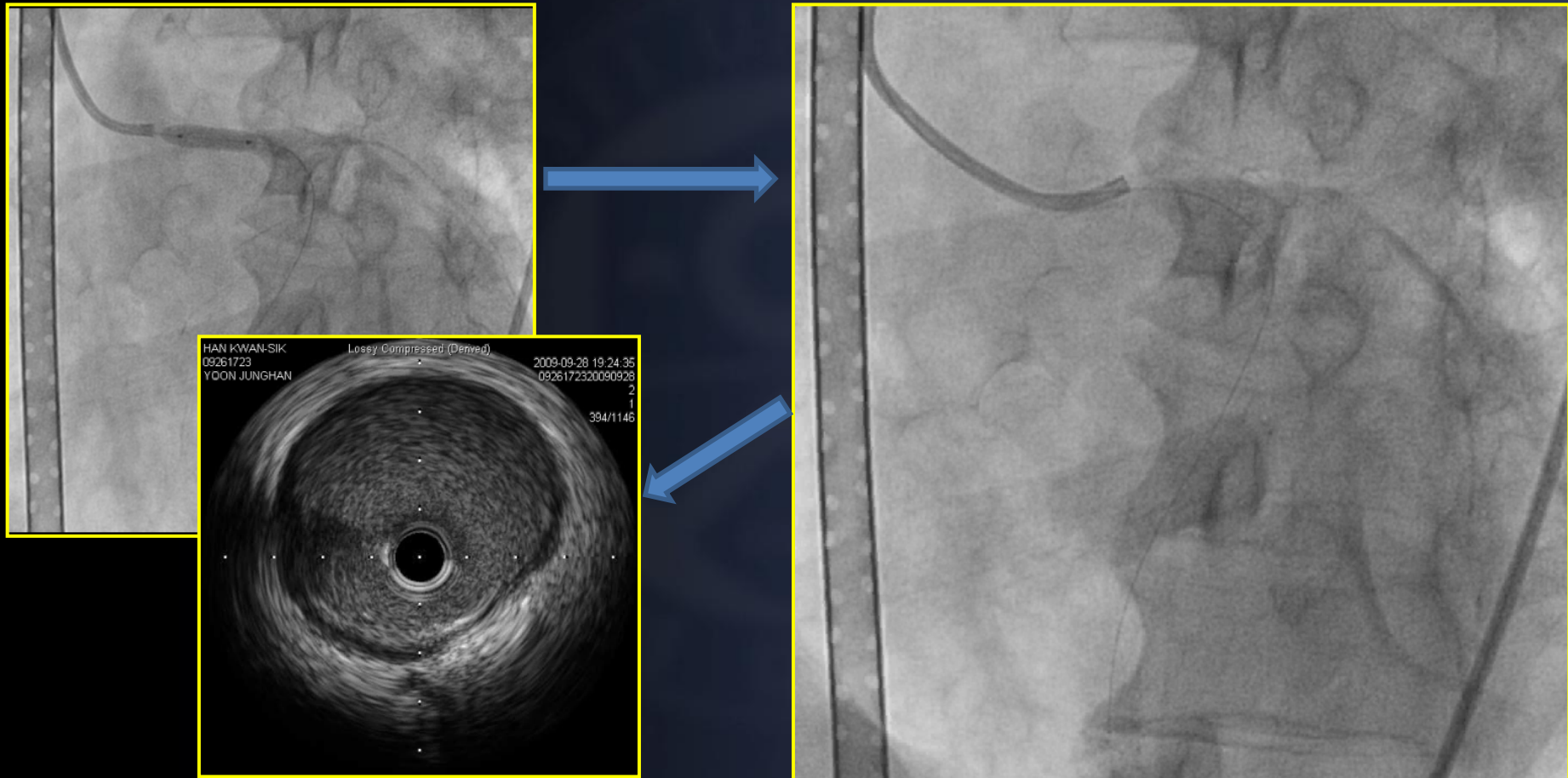


IVUS from mLM to pLM



- Surprisingly, **huge thrombus and ruptured plaque at LM** was suspected by IVUS

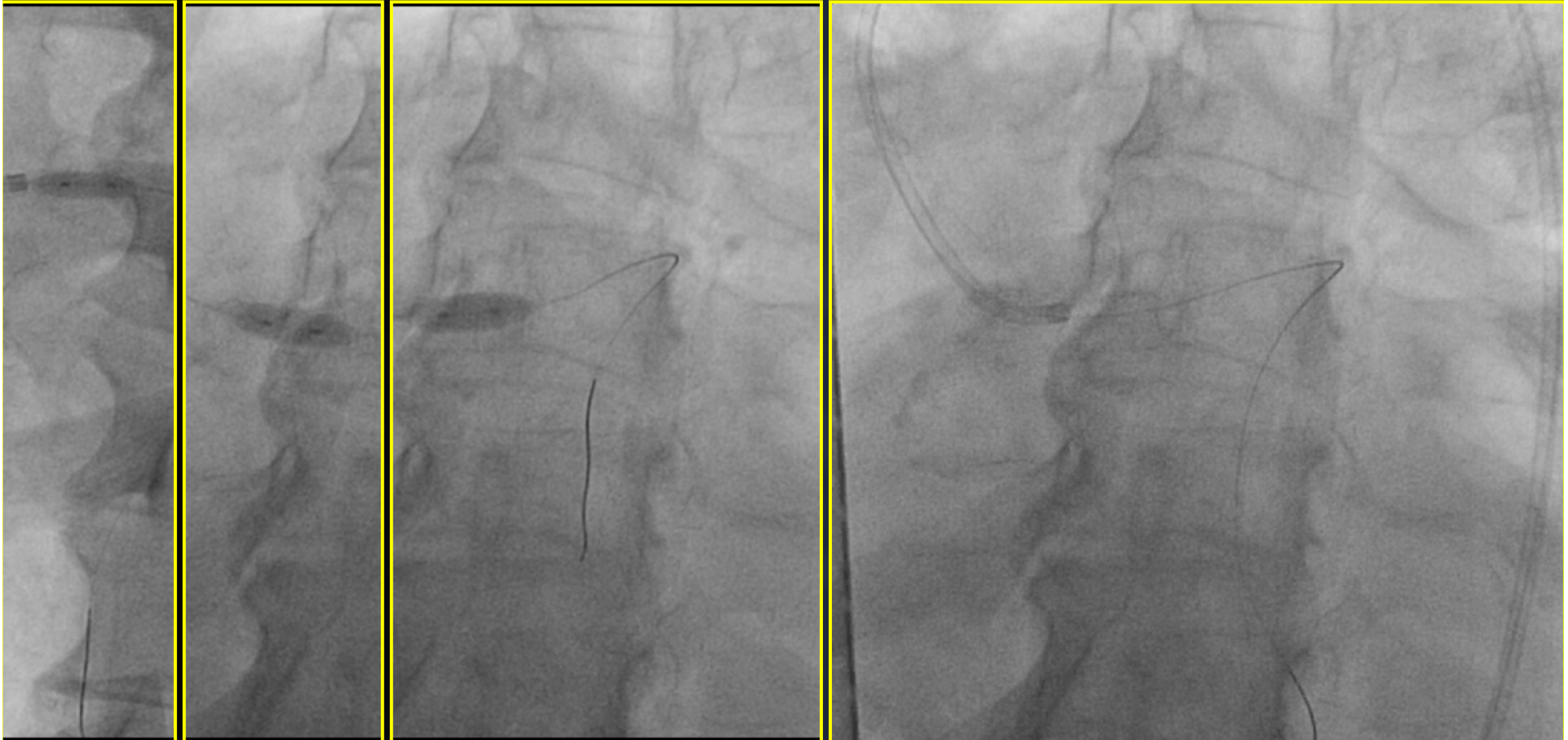
# 2<sup>nd</sup> PCI – LM stenting



- **Direct stenting** with DES, RESOLUTE 4.0mm x 12mm was done.
- Following angiography showed residual stenosis.
- So, IVUS was done. Malapposition of stent was shown at distal edge of stent.



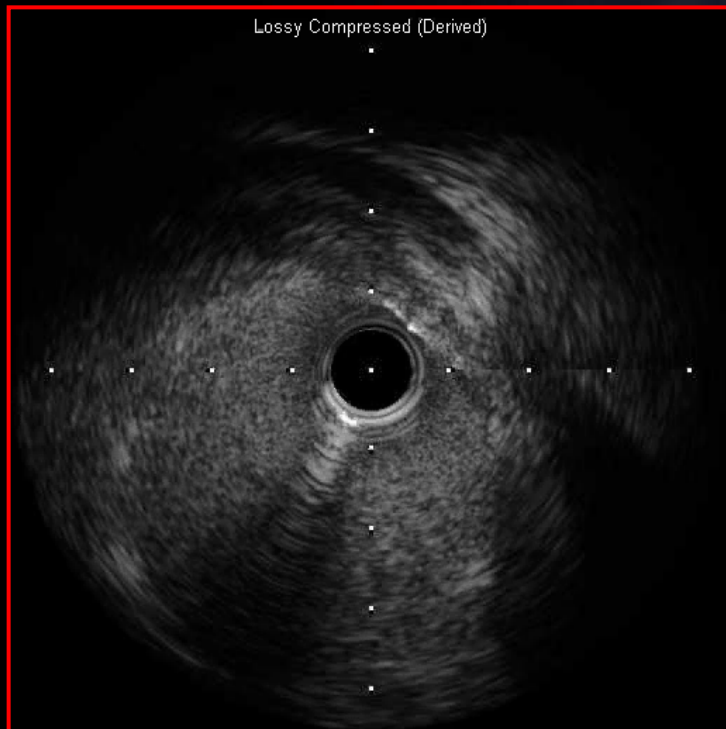
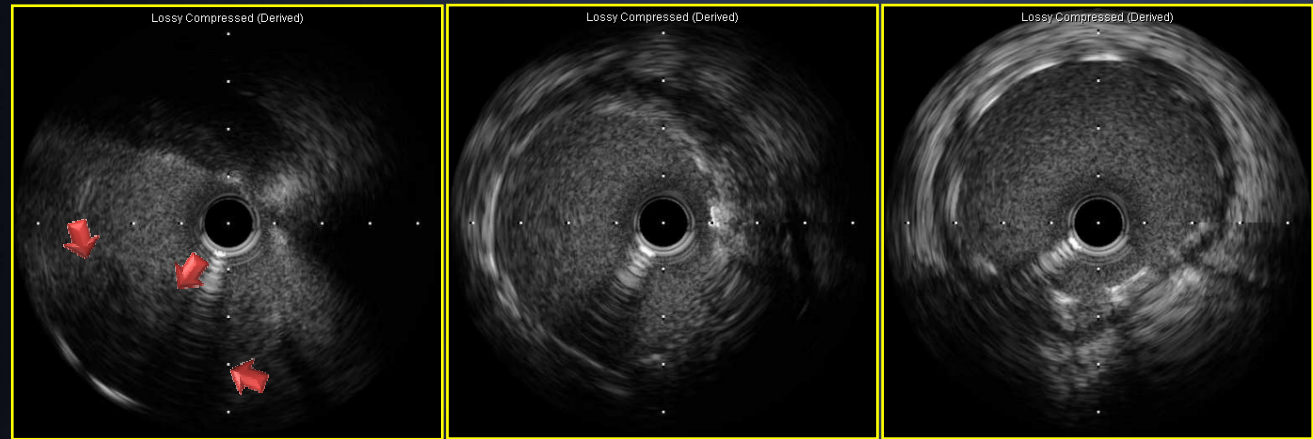
# 2<sup>nd</sup> PCI – adj. ballooning



- **Adjunctive ballooning** with QUANTUM 5.0mm x 8mm was inflated 3 times up to 20 atm. at LM. Following angiography showed no residual stenosis. IVUS was done, again.



# 3<sup>rd</sup> IVUS



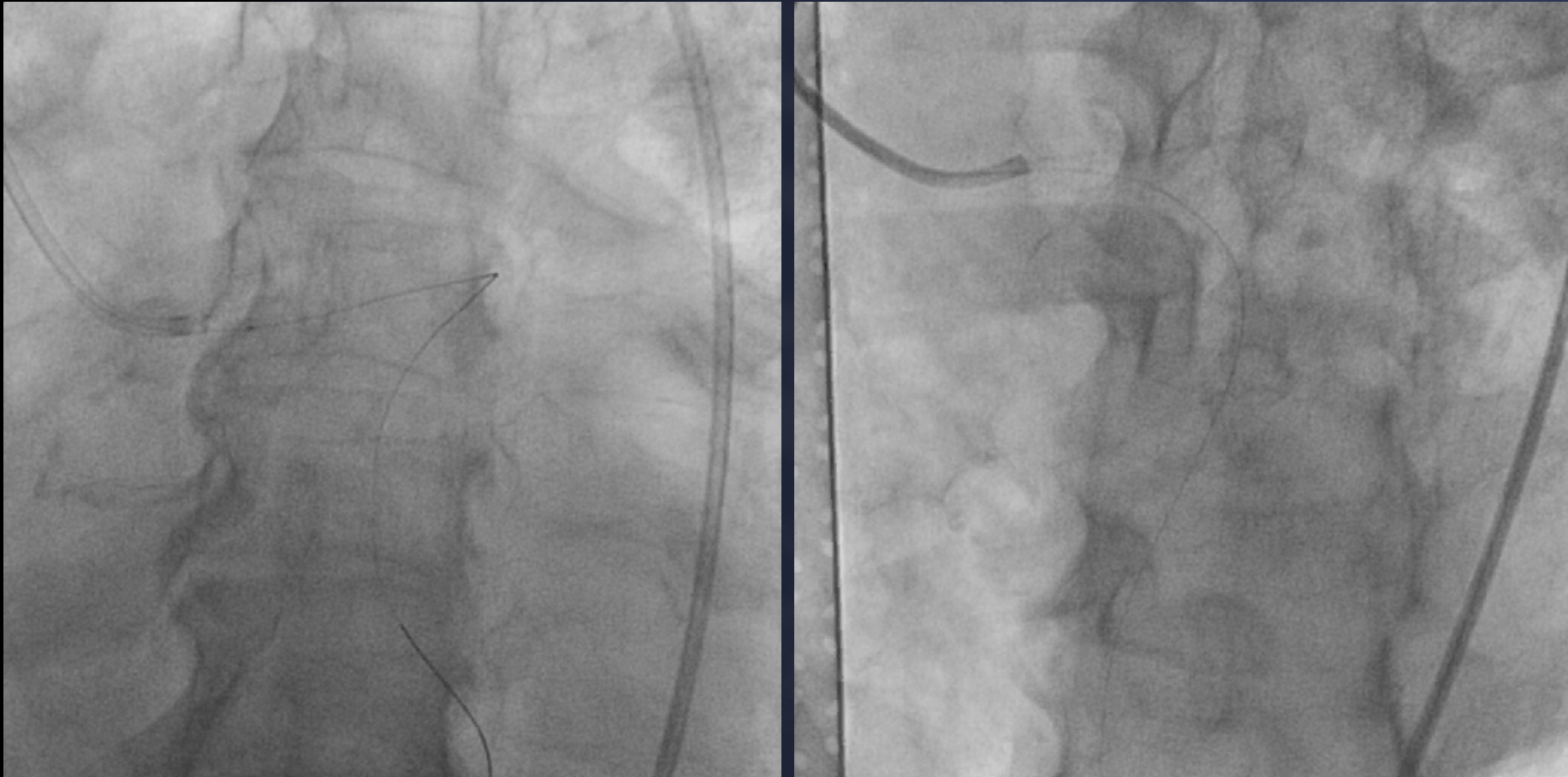
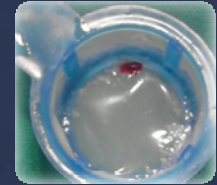
IVUS from dLM to pLM

- IVUS showed **hypoechoic inhomogeneous density** at 6 to 8 o'clock at proximal LM.





# 2<sup>nd</sup> PCI - Aspiration

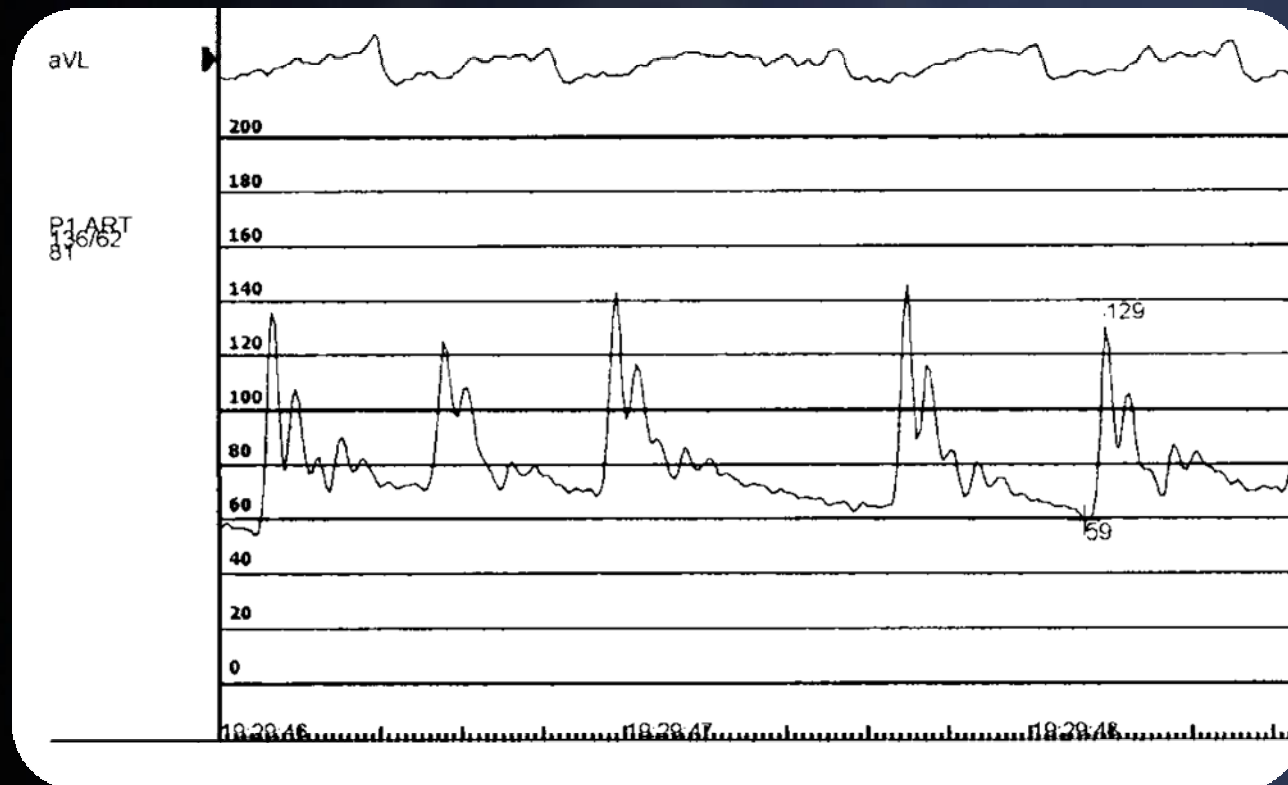


- Suspecting thrombus, aspiration was tried and **red thrombus** was observed. After confirming no residual thrombus by IVUS with manual pull-back, final angiography was done. There was no residual stenosis.



# Progress

- After stenting at LM, patient's blood pressure was recovered.
- At the end of procedure, there was no need of inotropics.
- Patient was **moved to ward with stable vital sign.**

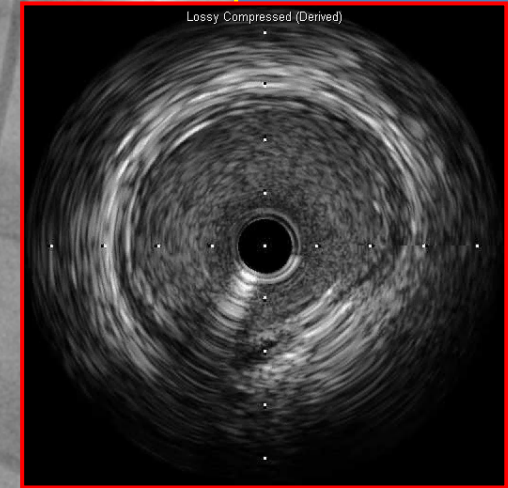
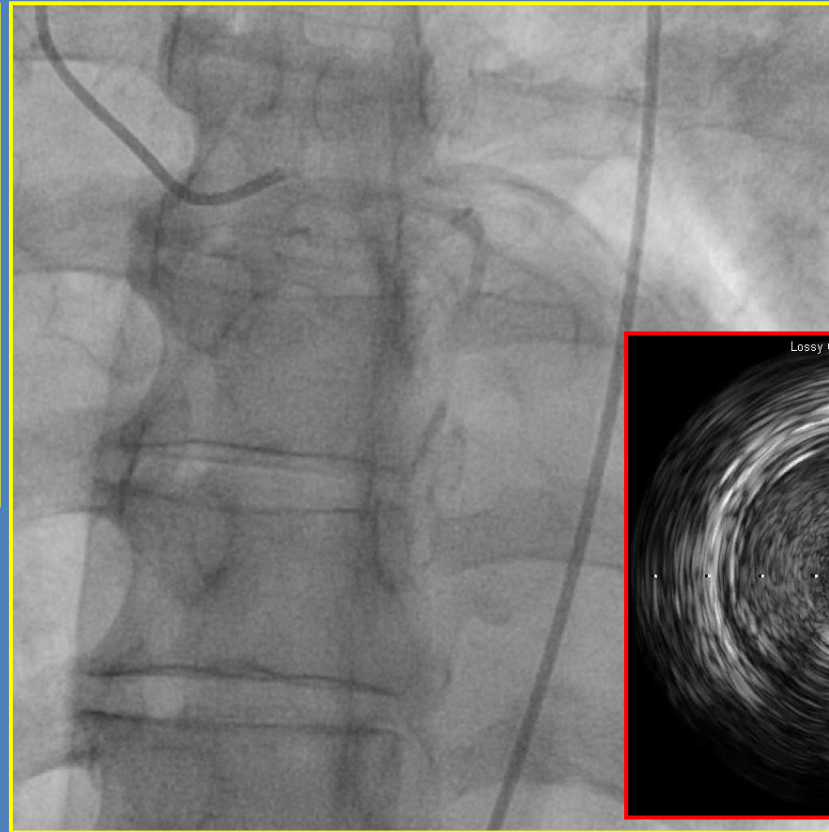
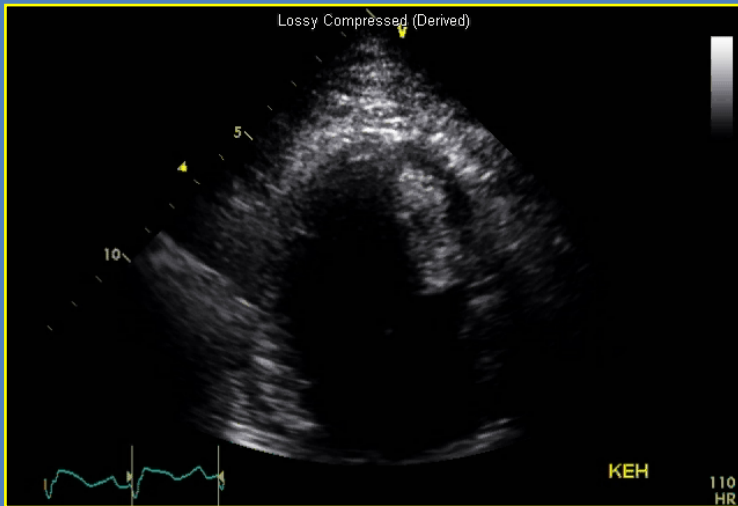


# Discussion point

- **What could be the cause of multiple embolic infarct?**
  - **From Left main?**
    - Ulcerating plaque at LM by IVUS
  - **From LV thrombus?**
    - That's what you see.
    - But there was no size difference of LV thrombus after re-MI.
  - **From DVT? (Paradoxical embolism?)**
    - Forget it.

# Conclusion

- Although, other possibilities could not be ruled out completely
- Multiple embolic MI might be caused by
  - **thrombus at LM lesion** or **LV thrombus**



- **IVUS should be considered if the cause of embolic MI is unclear.**

# Discussion point?

- **If the cause was the LV thrombus, what could be the cause of LV thrombus?**
  - Aneurysmal change due to MI?
  - Stress CMP?
  - **He had thrombophilia?** (Hx of young age onset DVT and PTE)

# LV aneurysm formation after AMI

J Cardiogr. 1985 Mar;15(1):55-66.

## [On the time of cardiac aneurysm formation following acute myocardial infarction]

[Article in Japanese]

Kikuchi H, Honda T, Hayasaki K.

It has been said that ventricular aneurysm is formed in the relatively late stage after the onset of acute myocardial infarction. We examined the time of its formation using digital subtraction angiography (DSA) performed immediately after infarction and at various intervals thereafter. We also examined correlations between aneurysm formation and the degree of rest after infarction, blood pressures, sites of infarction and coronary angiographic findings. The subjects consisted of 35 hospitalized patients with acute myocardial infarction. They were examined by DSA immediately, and one week and one month after their admissions. DSA was performed in the 30 degree right anterior oblique projection, and cardiac aneurysms were diagnosed by the presence of regional protrusion or of dyskinesis of the left ventricular wall on left ventriculography. The results were as follows: Cardiac aneurysms were noted in eight men and four women. The mean age was 69.2 +/- 8.1 years. Infarctions were located in the anteroseptal region (nine patients), in the broad anterior wall (two patients) and in the inferior wall (one patient). The average onset-to-admission interval was 5.6 hours in the aneurysm group, and eight hours in the aneurysm-free group. Cardiac aneurysms were demonstrated by DSA immediately after hospital admission in all 12 patients in the aneurysm group and the size did not increase appreciably with time. The peak CPK was significantly higher in the aneurysm group (3,163) than in the aneurysm-free group (1,655), but there was no group-related difference in risk factors, hypertension, the duration of rest after infarction, or coronary angiographic manifestations. Cardiac aneurysm has been considered as a late complication of myocardial infarction. Many investigators have reported that its formation begins one to four weeks after the onset of infarction with gradual protrusion. In the present study however, the formation of aneurysms was complete at very early stages after the onset of the myocardial infarction and often encountered in patients with relatively extensive infarction.

We examined the time of its formation using digital subtraction angiography (DSA) performed immediately after infarction and at various intervals thereafter.

They were examined by DSA immediately, and one week and one month after their admissions.

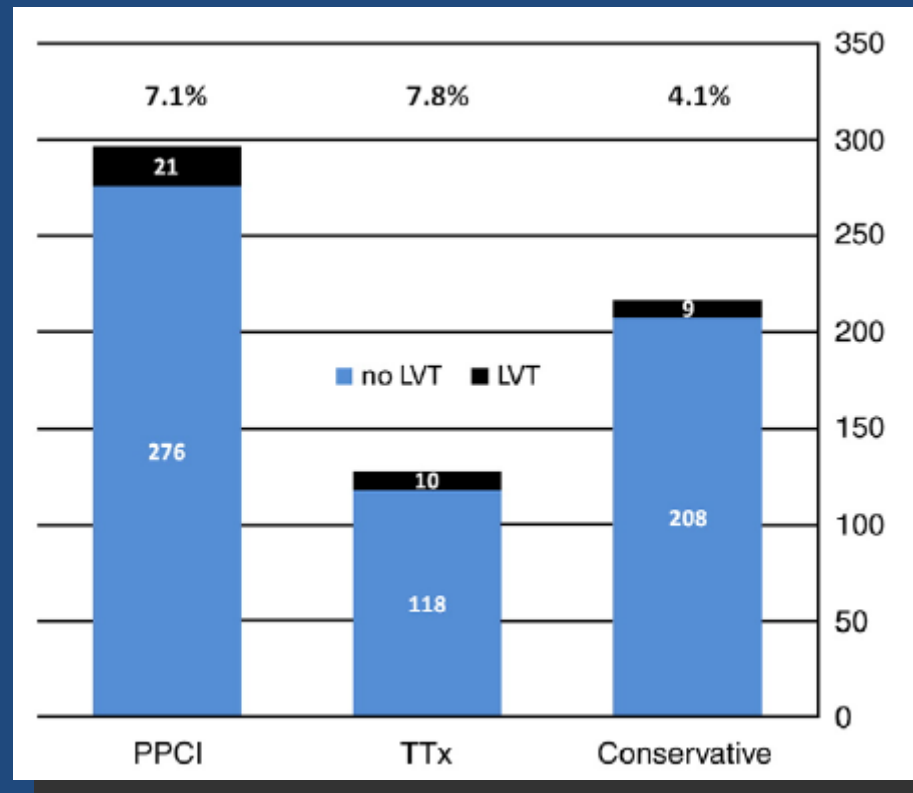
Cardiac aneurysms were demonstrated by DSA immediately after hospital admission in all 12 patients in the aneurysm group

The formation of aneurysms was complete at very early stages after the onset of the myocardial infarction

*J Cardiogr. 1985 Mar;15(1):55-66.*

# LV thrombus formation after AMI

642 consecutive pts with AMI and Echo.  
Echocardiography after a median of 2 days from admission (1-3 days).

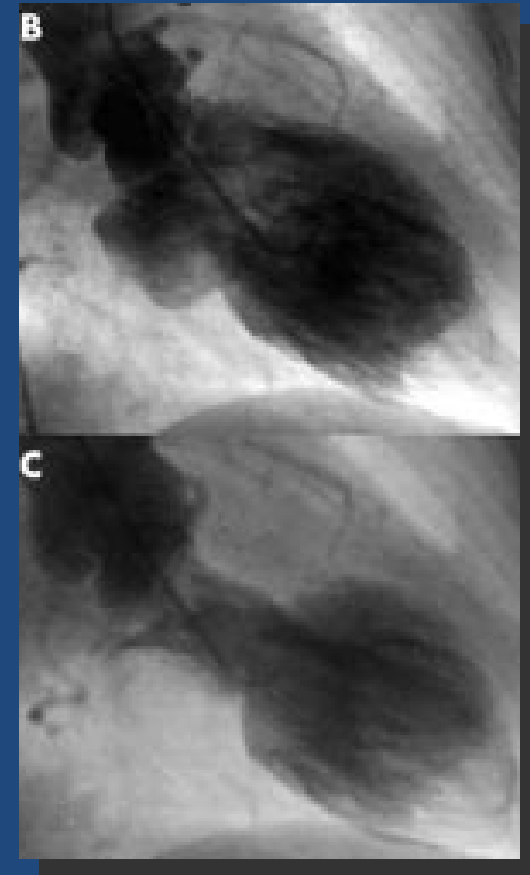


The median time to thrombus detection was 4 days (1-11 days).

# LV thrombus formation after SCM

## Apical thrombus associated with LV apical ballooning

- 64 year old woman
- C.C: continuous atypical chest pain **for 2 days**
- Coronary angiography: normal coronary arteries
- LV angiography:
  - apical asynergy with basal hyperkinesia (“apical ballooning”) with striking **filling defect at the apex**
- Prognosis
  - Discharge with anticoagulation
- 3 months later
  - Normal ECG, Normal wall motion, EF 62% with complete thrombus resolution





# Thrombophilia, maybe...

Lab	unit	Reference	27 Sep. 2009	28 Sep. 2009
FDP	µg/ml	<5		20.2 ↑
D-dimer	ng/ml	<280	448	3033 ↑
<b>AT-III</b>	%	<b>77-123</b>		<b>46 ↓</b>
Fibrinogen	mg/dl	200-400		332
Anti-CCP	U/ml			Negative
ANA				Negative
Anti_DNA				Negative
Cardiolipin IgG				Negative
Cardiolipin IgM				Negative
ANCA				Negative
<b>Protein C</b>	%	<b>70-130</b>		<b>33 ↓</b>
<b>Protein S</b>	%	<b>73.7-146.3</b>		<b>51 ↓</b>

**Warfarin produces a marked reduction in the functional activity of protein C and protein S** and a lesser decline in immunologic levels.

However, coagulation assays for protein C and protein S can give falsely low values if the factor V Leiden mutation is present; as a result, reliable application of coagulation assays must initially assess whether the factor V Leiden mutation is present.