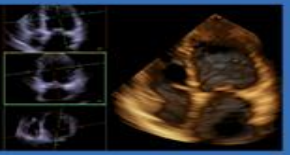
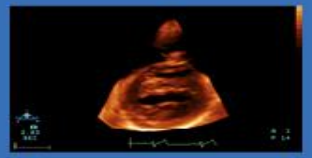


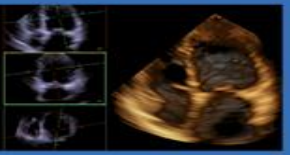
EuroValve
April 26-27, 2018



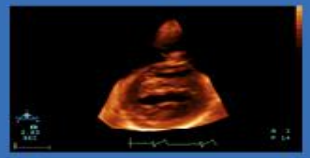
What is Your Diagnosis?

A STORM

R Dulgheru, Liege, Belgium



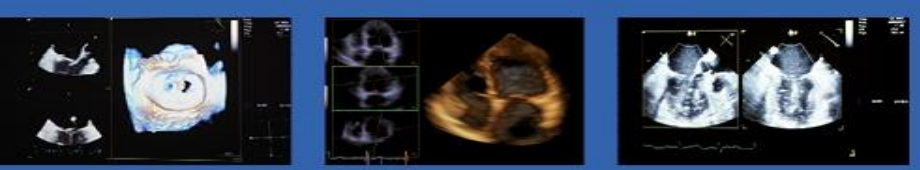
EuroValve
April 26-27, 2018



Faculty disclosure

Raluca Dulgheru

I have **no financial relationships** to disclose.



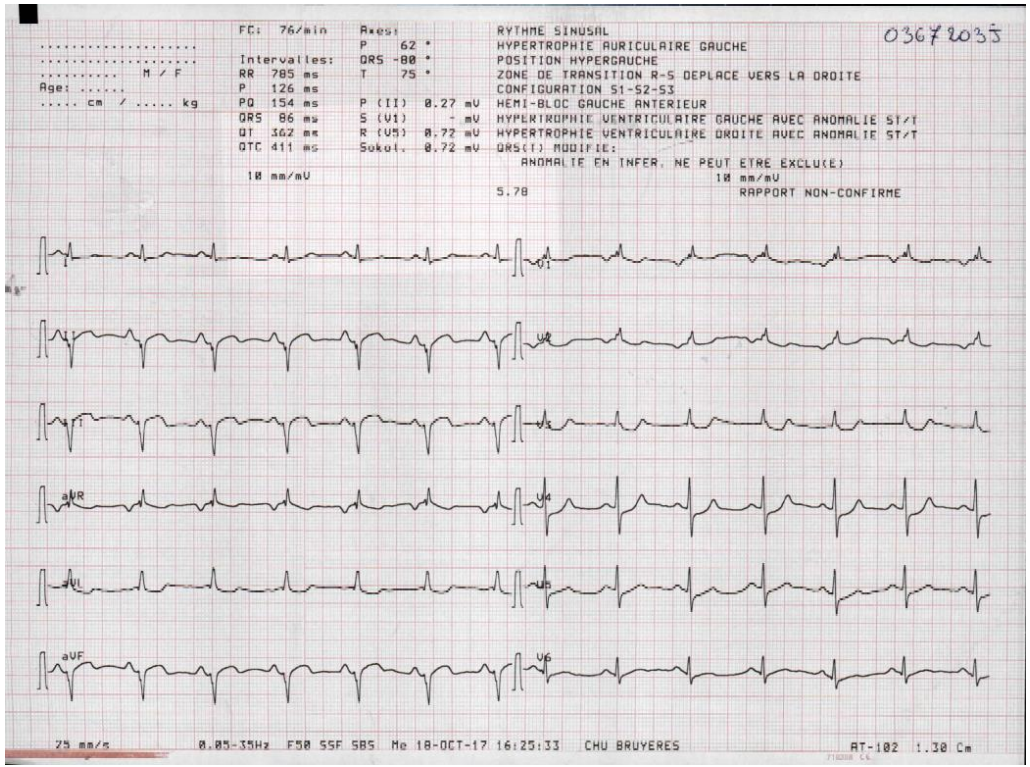
Case presentation

- 52 years old male
- ***Ongoing oppressive chest pain*** and ***severe dyspnoea*** (onset 6h before admission), but several episodes of chest pain in the past two weeks
- No known medical history
- Tabaco use (1 pac/day)
- Family history of premature heart disease (father with AMI at 45 years of age)
- Treatment: Omeprazole 20 mg/day

Clinical examination

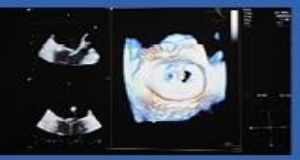
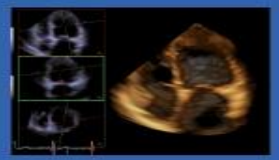
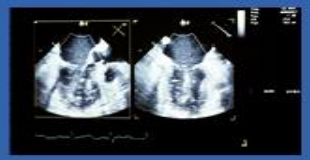
- Distress, clammy skin
- Dyspnoea
- SaO2 = 92% on ambient air
- BP = 70/50 mmHg
- HR = 98 bpm
- Bilateral crackles up to the apices of both lungs
- No audible heart murmurs

ECG at admission



Arterial blood gas test at admission

- PH = 7.27
- PCo2 = 56 mmHg
- Base excess = -2.6 mmol/L
- Standard bicarbonates 22.3 mmol/l
- Lactate 130 mg/L (normal range 50-125)

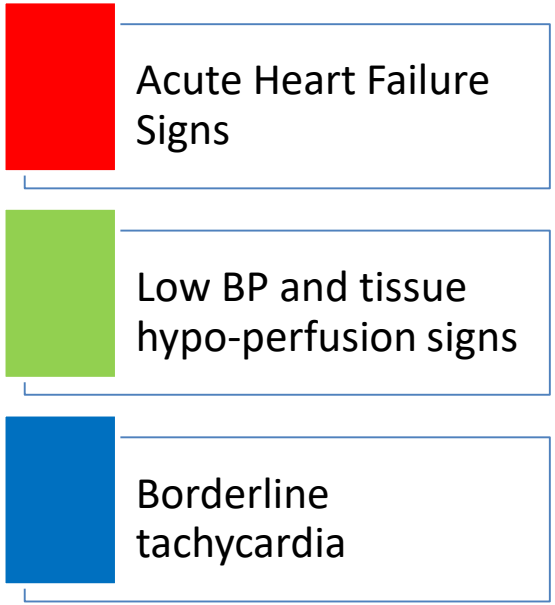




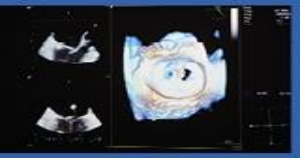
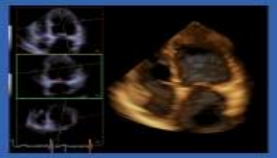
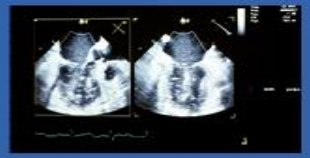

What is your diagnosis?

1. Acute pulmonary oedema
2. Acute pulmonary embolism
3. Cardiogenic shock
4. Respiratory acidosis of unknown aetiology

Cardiogenic shock

- Distress, clammy skin
- Dyspnoea
- SaO2 = 92% on ambient air
- BP = 70/50 mmHg
- HR = 98 bpm
- Bilateral crackles up to the apices of both lungs
- No audible heart murmurs

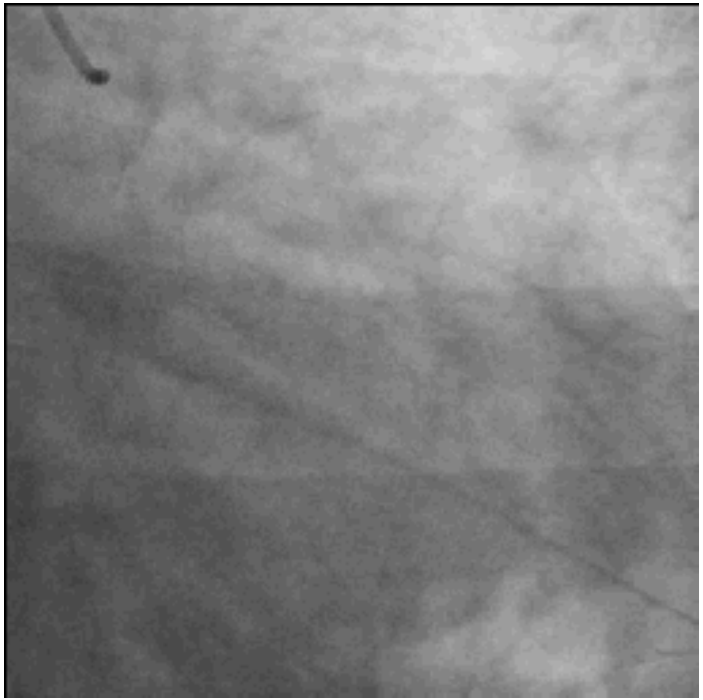


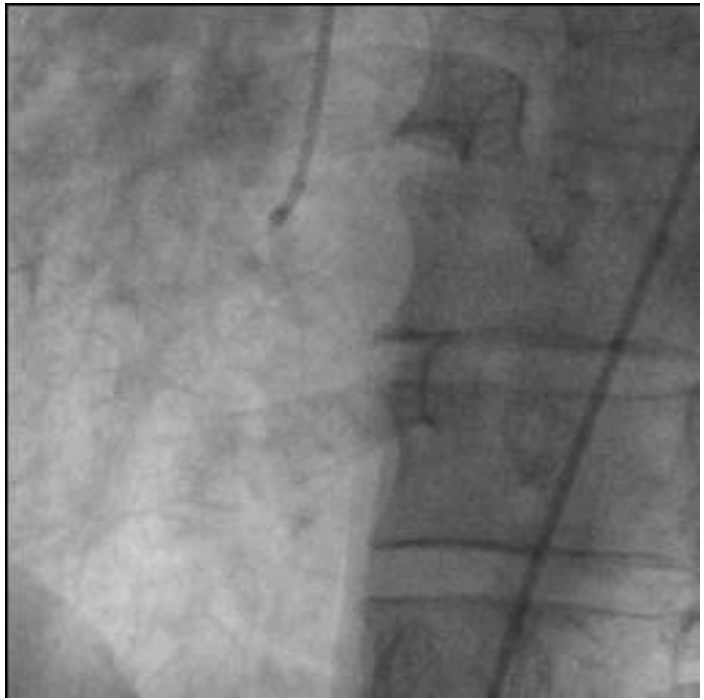





What do you do next?

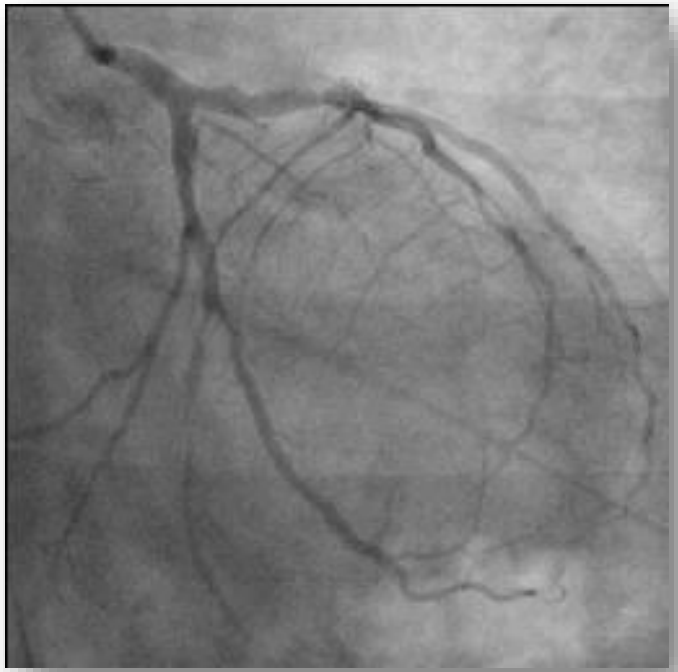
1. Send the patient to the cath lab for emergency coronary artery angiography
2. Angio CT of the thorax to rule out PE
3. Bed-side TTE
4. Bed-side chest X-ray

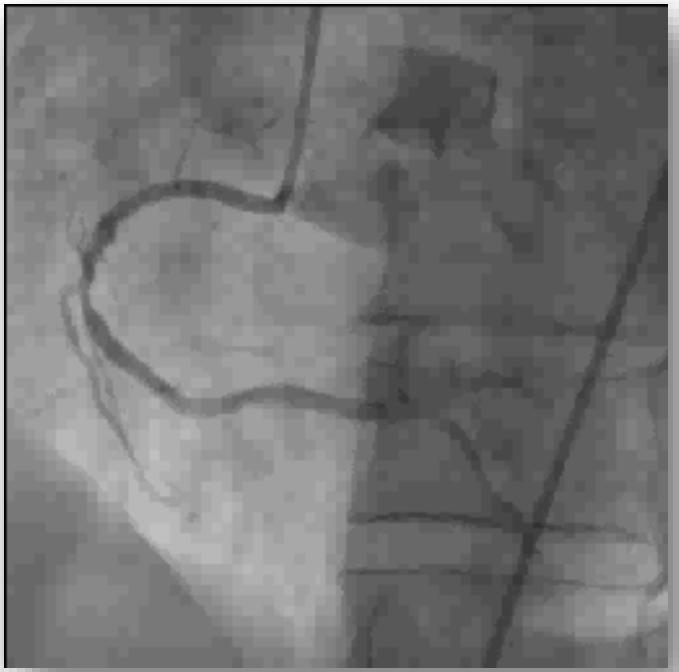
Coronary angiogram

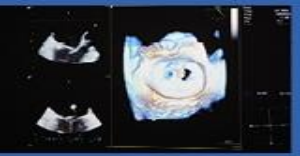
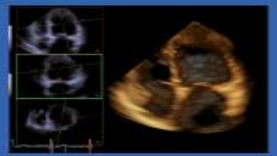
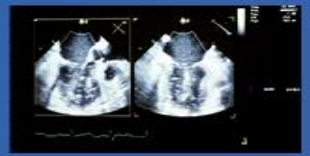




Coronary angiogram





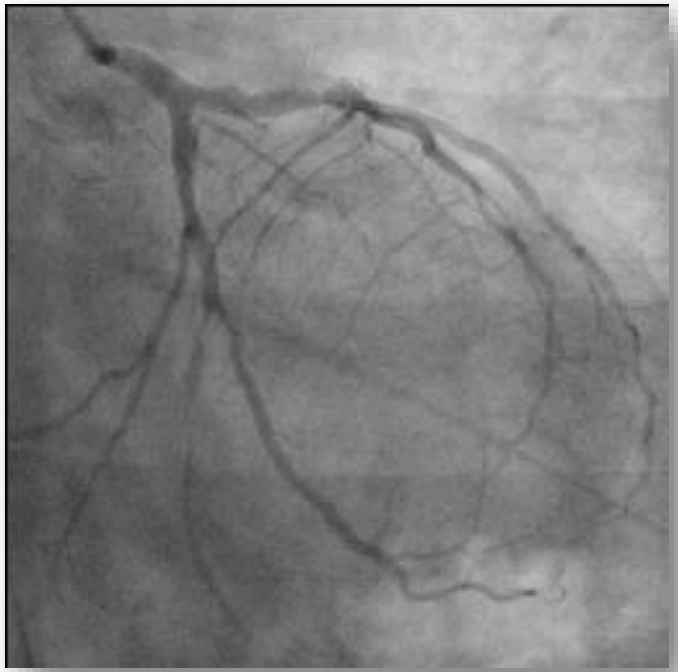

What is your diagnosis?

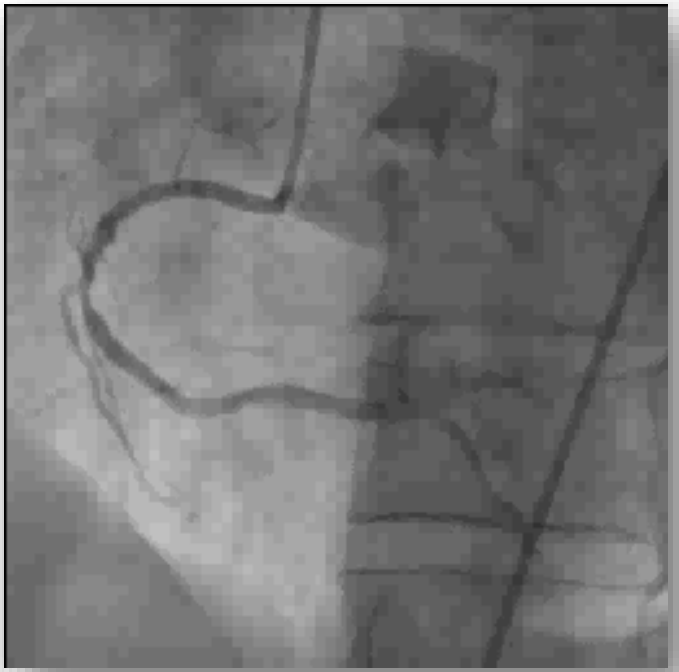
1. NSTEMI, severe three vessels disease complicated by cardiogenic shock

2. Cardiogenic shock, severe coronary artery disease but I need more to explain the clinical picture

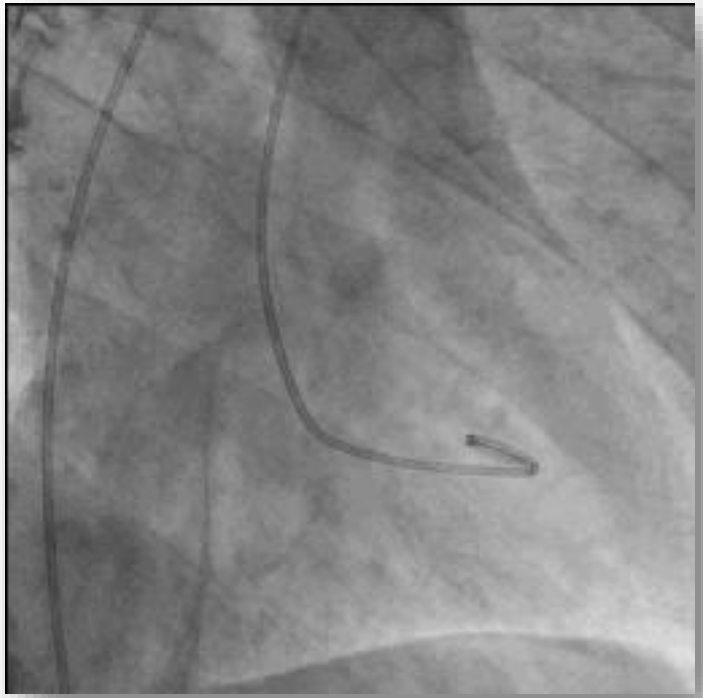
3. Acute STEMI with IVS rupture

Cardiogenic shock, severe coronary artery disease but aetiology of shock is unknown yet





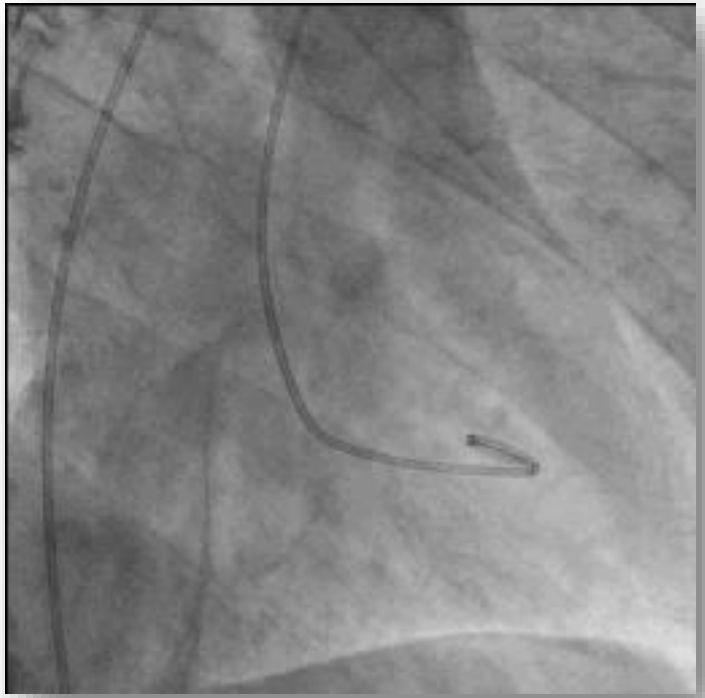
Ventriculography



What do you do next?

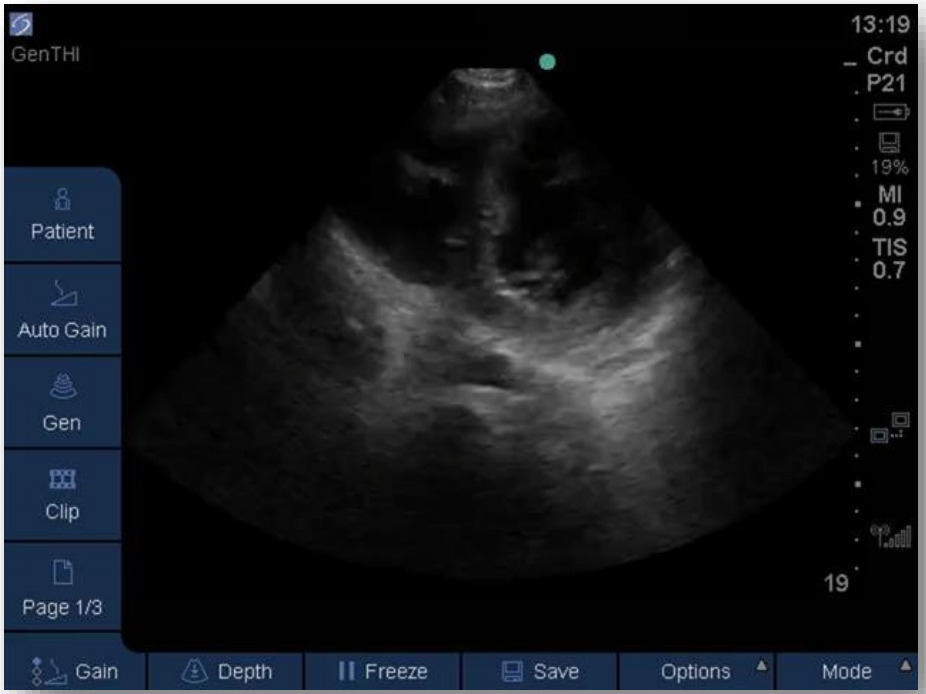
1. Call a friend to do an echo
2. Stent the right coronary artery
3. Call the lab to see the cardiac enzymes
4. Do a angio CT of the thorax

Ventriculography



- LV hyperdynamic (in discordance with the shock state)
- LA opacification in systole (Severe MR suspected)
- LA seems enlarged (chronic MR)

TTE


GenTHI
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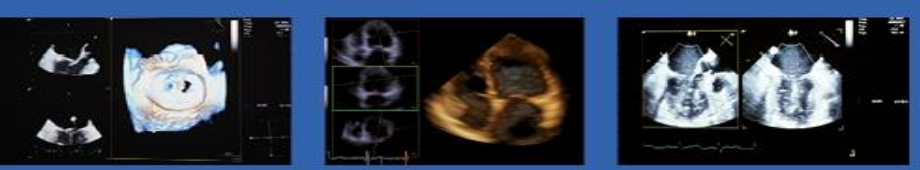
Gain
Depth
Freeze
Save
Options
Mode

Blood samples at admission

- Hb 18 g/dl, Ht 52%
- Glucose 122 mg/dl
- Creatinine 1,14 mg/dl
- LDH 393 (<250) U/l
- CK 209 (<190) U/l
- CKMB 5.4 (<6.2) microg/l
- Troponin T_{hs} 1291 ng/L (<14)



What is your diagnosis?

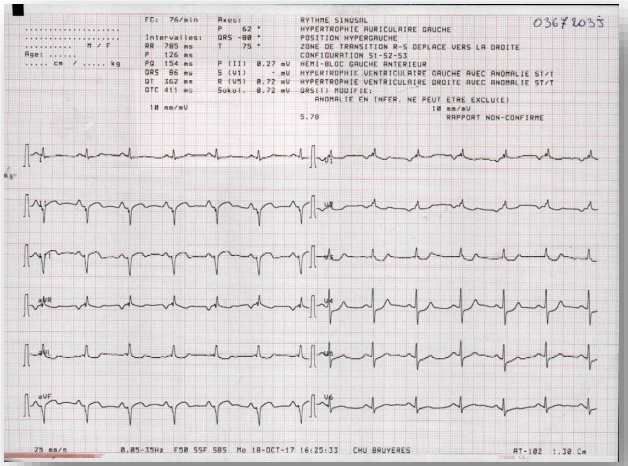
1. Cardiogenic shock, three vessels disease and IVS septal rupture

2. Cardiogenic shock and infero-posterior STEMI

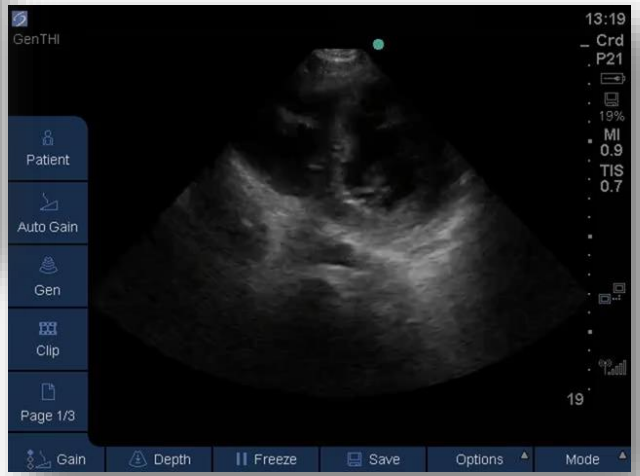
3. Cardiogenic shock, NSTEMI, three vessels disease and severe chronic secondary MR

4. Cardiogenic shock, probably subacute MI, PM rupture, severe acute MR and three vessels disease

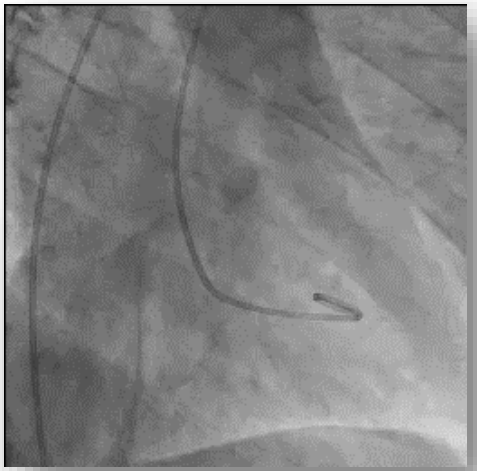
Q waves infero-posterior



Inferior and infero-lateral wall akinesis



Acute severe MR

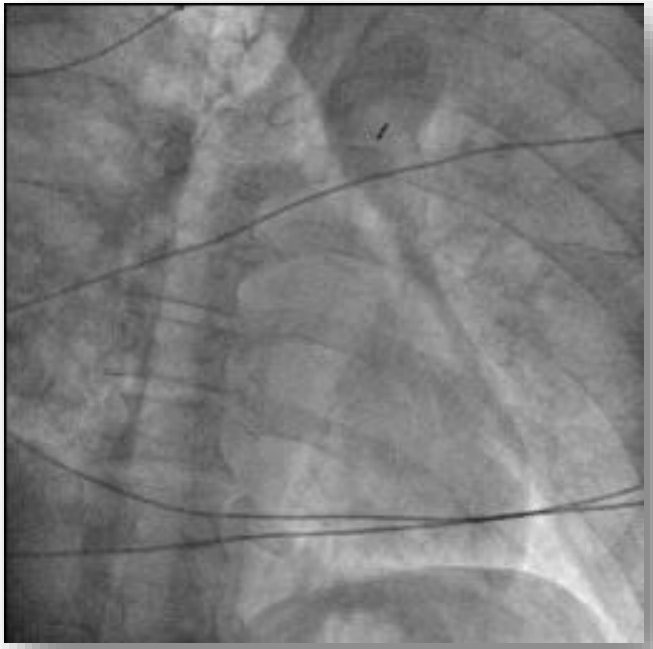


Cardiac enzyme tail

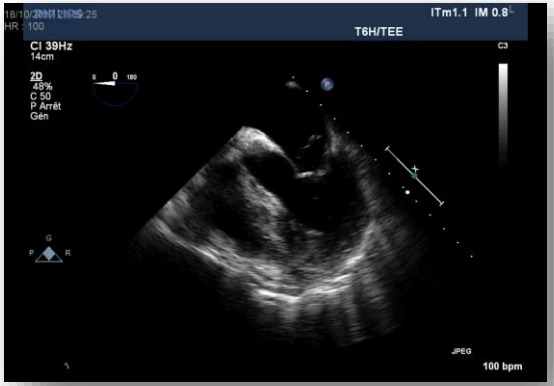
- LDH 393 (<250) U/l
- CK 209 (<190) U/l
- CKMB 5.4 (<6.2) microg/l
- Troponin T_{hs} 1291 ng/L (<14)

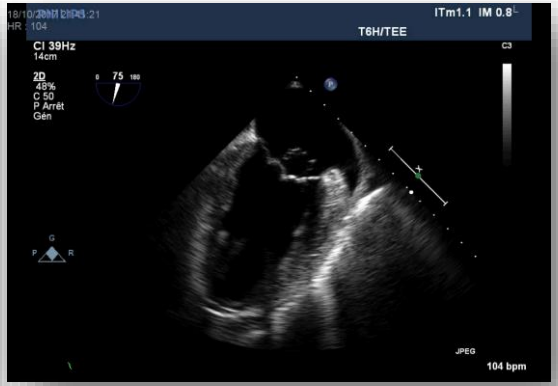
Floppy postero-medial PM suggestive of PM rupture

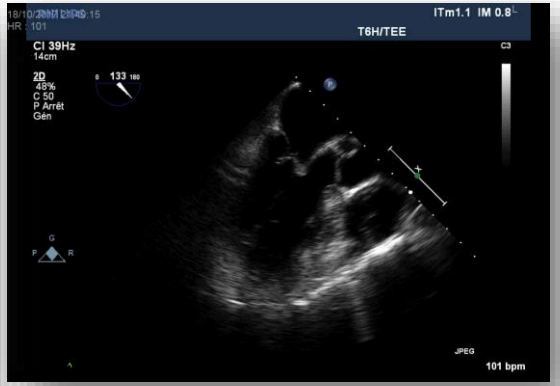
IABP and call a friend

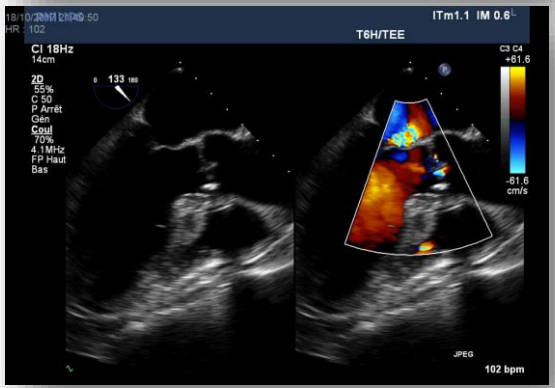


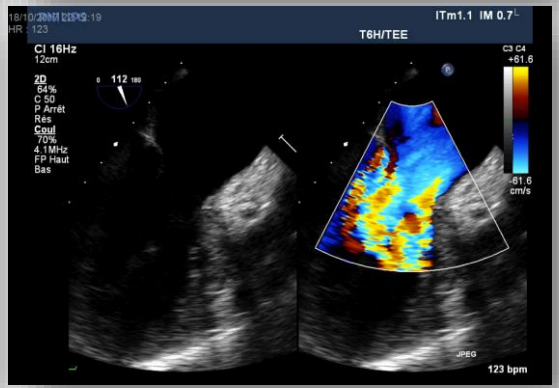
TOE in the OR

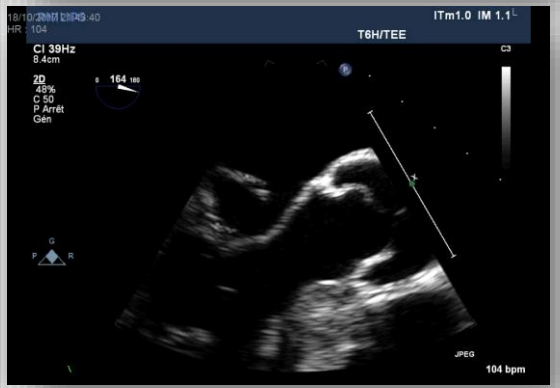

T6H/TEE
100 bpm

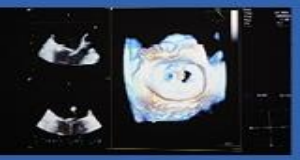
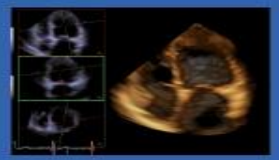
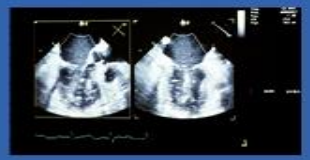

T6H/TEE
104 bpm


T6H/TEE
101 bpm


T6H/TEE
102 bpm


T6H/TEE
123 bpm

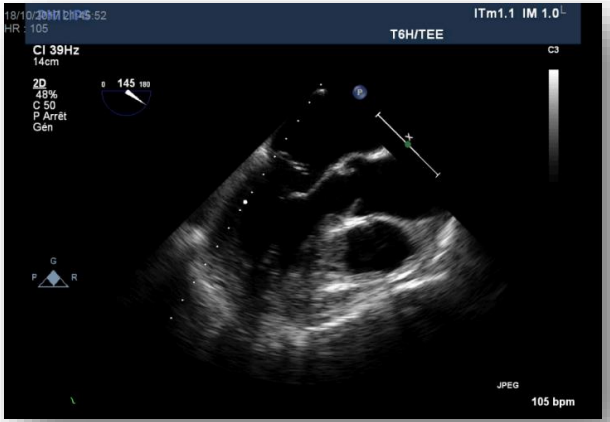

T6H/TEE
104 bpm

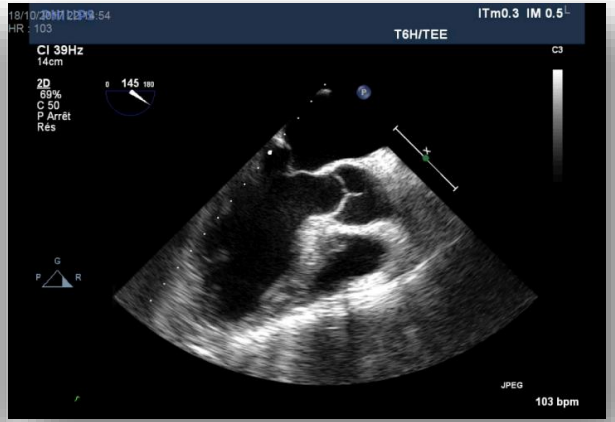




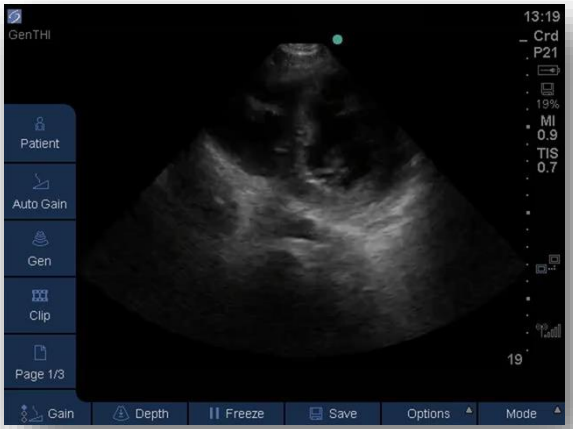

What does the echo show?

1. Complete transection of the postero-medial PM
2. Detachment of one of the antero-lateral PM heads
3. Complete transection of the antero-lateral PM
4. Detachment of one of the postero-medial PM heads

Detachment of one of the postero-medial PM heads







Clinical highlights

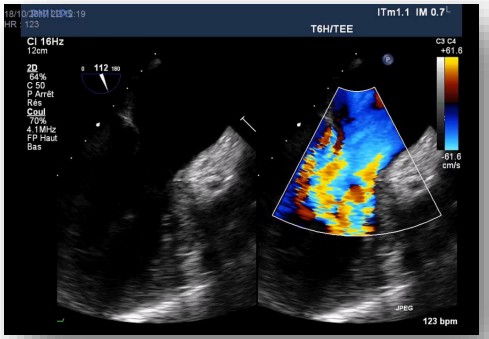
1. Angina recurrence in the context of subacute MI accompanied by sudden dyspnoea and followed by haemodynamic deterioration is suggestive of a mechanical complication of MI (PM rupture, IVS rupture, free-wall rupture)

2. Absence of cardiac murmur does not rule out acute MR

3. Cardiogenic shock in a patient with ACS needs prompt assessment of LV systolic function

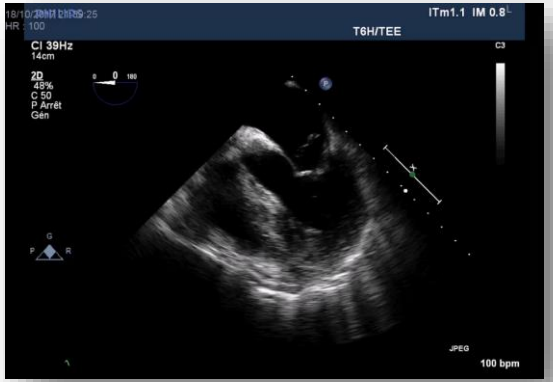
4. Good/excellent LV systolic function suggests acute MR

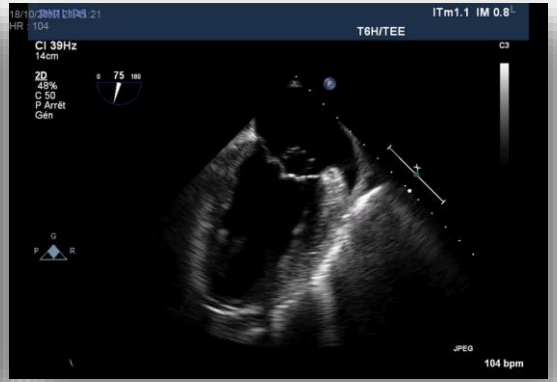
5. Echocardiography is key exam to detect the MR aetiology



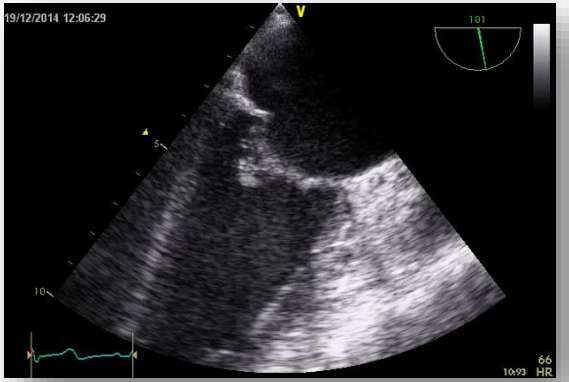
Echo highlights

- ✓ ***Complete transection*** of the papillary muscle is relatively ***rare***, whereas rupture of the tip is more common





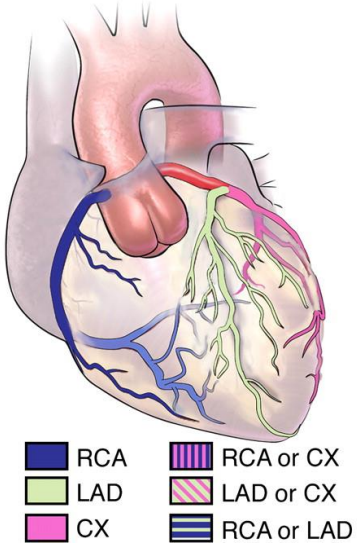
Rupture of the tip of the PM
Anterior mitral leaflet non involved, normal motion

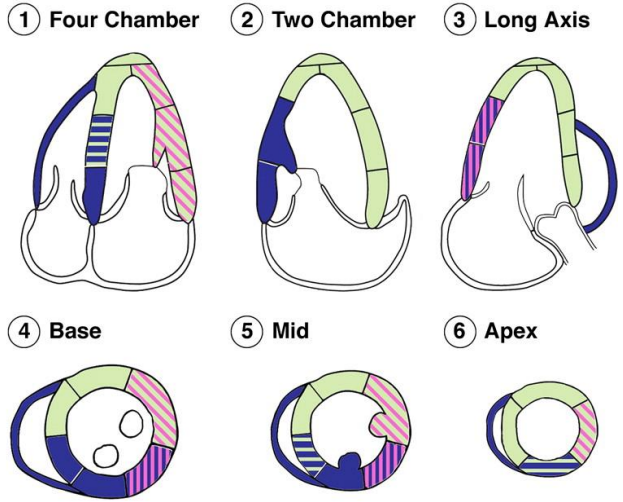


Complete transection
Both leaflets with excessive motion

Echo highlights

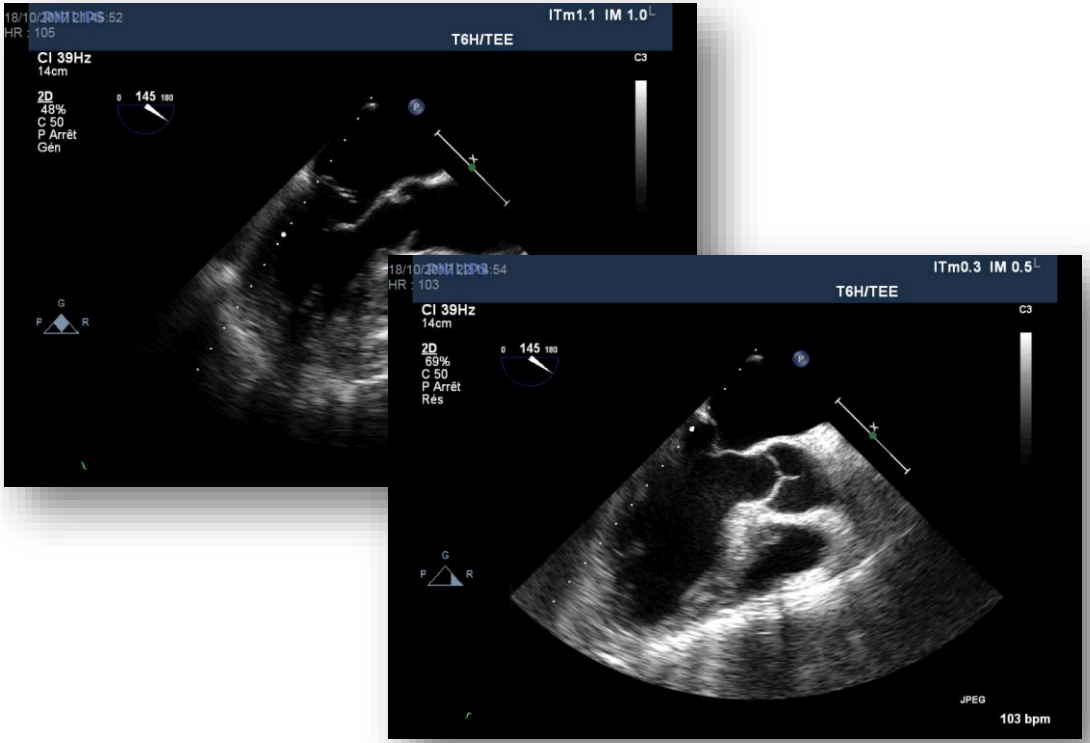
- ✓ Rupture of the ***posteromedial papillary muscle*** (supplied by a single coronary artery) occurs ***more often than*** rupture of the ***anterolateral papillary muscle*** (dual coronary supply)

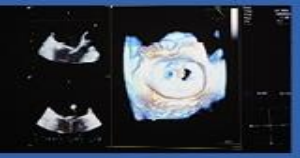
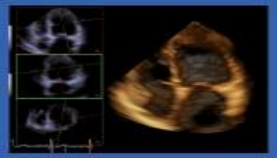
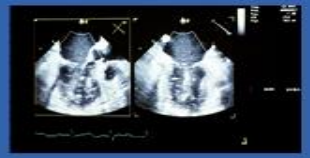




Echo highlights

- ✓ Regional wall motion abnormalities may be subtle/unrecognized (hyperdynamic LV due to sudden decrease in afterload)
- ✓ Suboptimal transthoracic imaging windows (dyspnea, acute pulmonary oedema, patient lying flat)
- ✓ TOE may be required to confirm diagnosis (after endotracheal intubation in a non fasting patient)

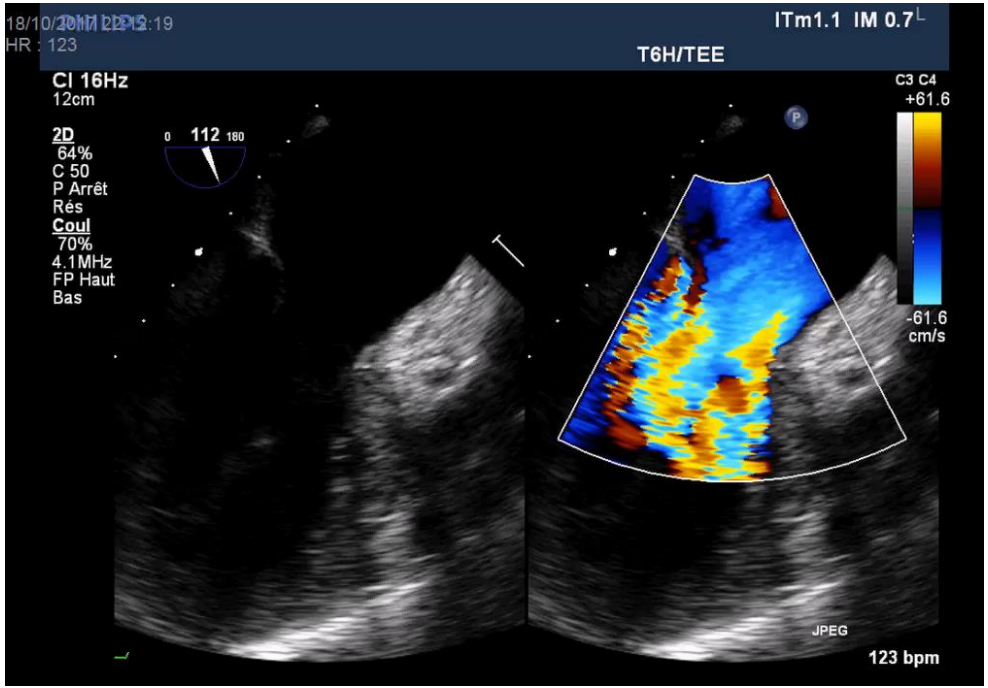


Case particularities

- No occlusion of a coronary artery has been identified by coronarography which suggests spontaneous reperfusion, “culprit” artery was not identified by angio
- Wall motion abnormality was missed by conventional ventriculography but identified with TTE and TOE, indicating also the ‘’culprit” vessel (RCA)
- Absence of severe tachycardia is not explained (chronotropic incompetence)
- Anterior leaflet was not involved because just one of the heads of the postero-medial papillary muscle was ruptured, probably the one giving chordae to the posterior leaflet
- Patient had three-vessels disease and late presentation after AMI which increases the risk of complications (such as PM rupture)

The “silent” storm



Acknowledgements to Dr. Mathieu Lempereur and Prof. Mark Radermecker and his colleagues