

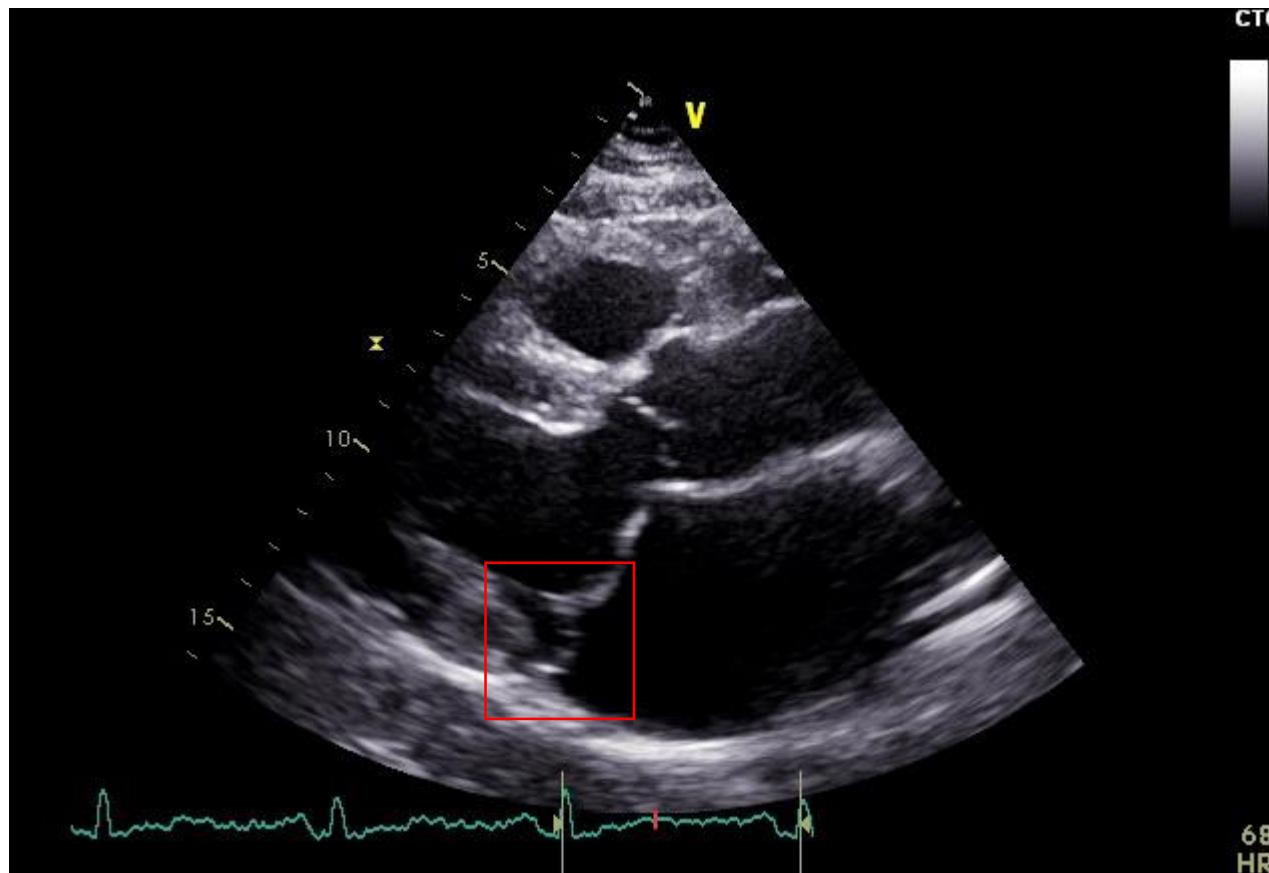


**Cardiogenic shock post mitral valve surgery: when IVUS becomes the best friend.**

Caucasian 52 years old man

- **Cardiovascular risk factors:** Mild Obesity (BMI 31 Kg/m<sup>2</sup>)
- **History:** No cardiovascular history
- **Hospital Admission:** progressively onset of exertional dyspnea (Class NYHA II) with palpitations.
- **No pharmacological therapy**
- **Initial work up:**
  - a) **Electrocardiogram:** Sinus rhythm with left axial deviation.
  - b) **Transthoracic echocardiography:** bi-leaflet mitral valve prolapse with severe valve regurgitation (regurgitant fraction >50%) associated with left atrium enlargement (LAVI = 103 ml/m<sup>2</sup>). Preserved left and right ventricle systolic function (left ventricle ejection fraction = 68%; S wave tissue doppler = 14 cm/sec) with normal left ventricle volume (LVEDVI = 68 ml/m<sup>2</sup>).
  - c) **EKG Holter:** showed moderate monomorphic ectopic ventricular beats (EVB).
  - d) **Treadmill stress test:** show an increase of number of EVB during effort without sustained ventricular tachycardia (SVT).

*Bi-leaflet mitral valve prolapse*



Indication to:

- Mini invasive mitral valve repair surgery (Heart Port) with extracorporeal circulation

***But after surgery .....***

- Impossible weaning from extracorporeal circulation for hemodynamic instability with onset of severe bi-ventricular dysfunction.
- Continuous EKG monitoring showed diffuse ST segment elevation (more evident in the infero-lateral leads)
- Intraoperative TEE excluded pericardial effusion or iatrogenic aortic dissection and confirmed severe left and right ventricular dysfunction (EF 25%) with regional hypokinesia of infero-lateral wall compatible with ischemic ST on EKG.
- Arterial and venous gas analysis show lactic acidosis due to peripheral hypoperfusion and low cardiac output (PH 7.12, HCO<sub>3</sub>- 18 mmol/l, lactate 9 mmol/L) with mixed venous oxygen saturation below 50% (SVO<sub>2</sub>= 45%)



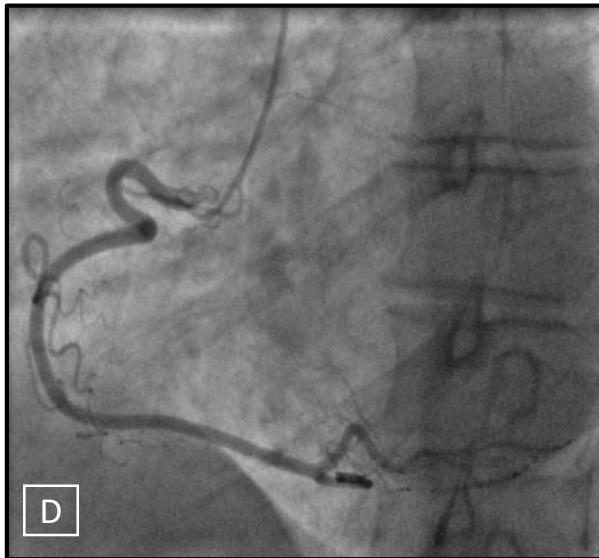
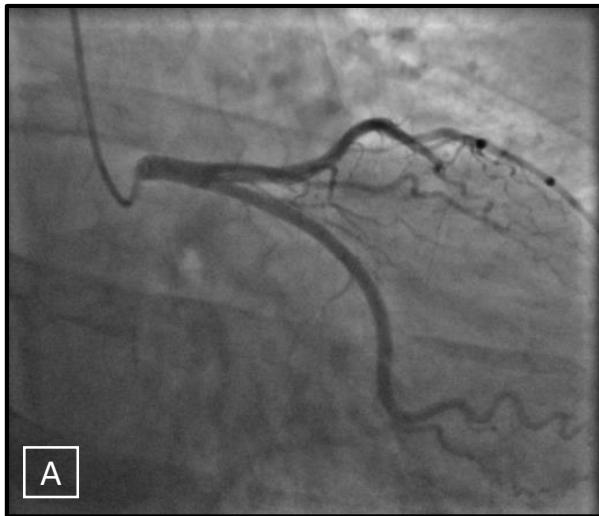
**This initial presentation satisfied the criteria for acute coronary syndrome (ACS) with ST segment elevation quickly precipitating into cardiogenic shock (CS) with low mean arterial pressure (MAP 50 mmHg) and urinary output (<1 ml/Kg/h).**

# Heart team discussion/therapy

After Heart Team discussion

1. Maximal pharmacological support with mid-high dose inotropic drugs (epinephrine 0.09  $\mu$ , norepinephrine 0.1  $\mu$ ) and iv diuretic (furosemide 250 mg/day) were started
  
2. Mechanical circulatory support (MCS) were placed
  - a) Peripheral VA-ECMO with arterial cannula (14 Fr) in the right femoral artery up to ascending aorta and venous cannula (22 Fr) in the right femoral vein up to distal part of inferior vena cava near the right atrium with basal flow 4-4.7 L/min; 75000 rpm (systemic perfusion ---> bridge to decision)  
+
  - b) Intra-aortic balloon pump (IABP) sheathless from right femoral artery with rate 1:1 (for left ventricular unloading)
  
3. Emergent coronary angiography

# PRE Surgery Coronary Angiography



Setting:

- 6 Fr radial access;
- JR and JL 5 Fr diagnostic catheter;

Figure A, B and C ---> IVA and LCx  
Figure D and E ---> CDx

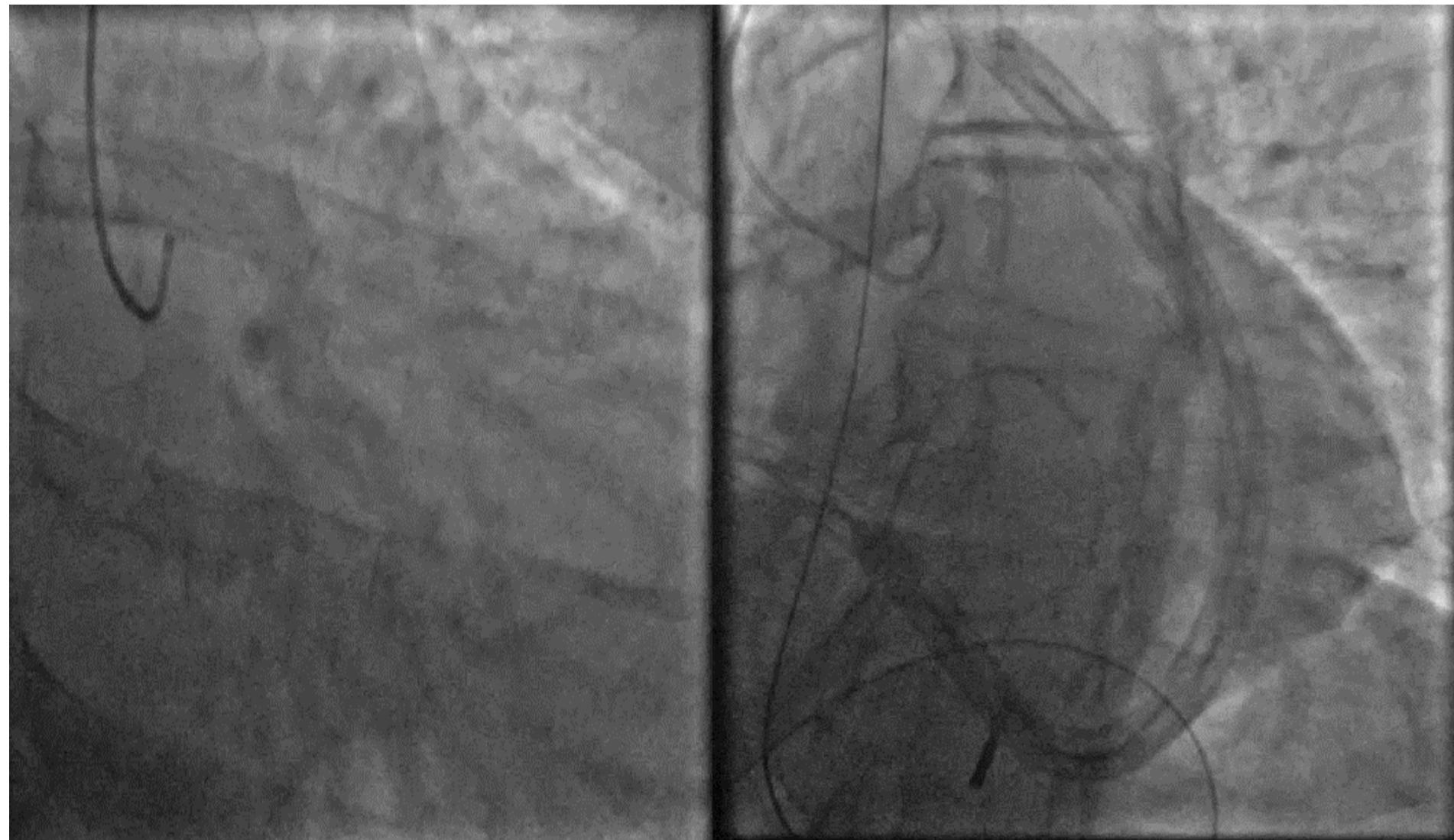


No epicardial stenosis

# POST Surgery Coronary Angiography

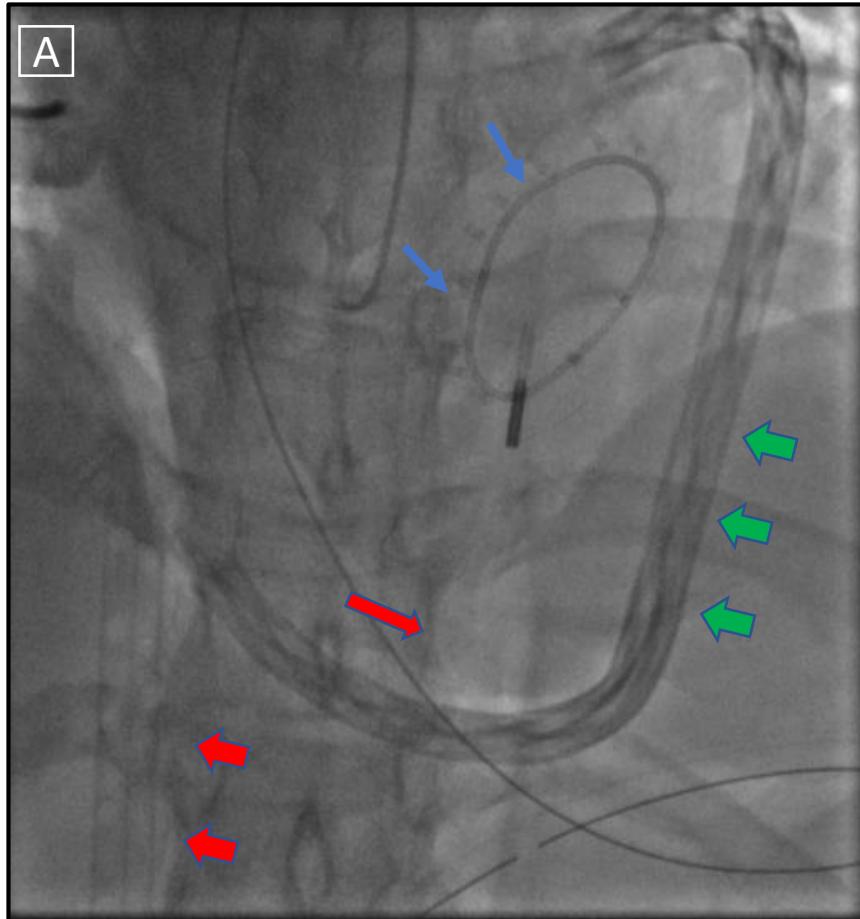
Before Surgery

After Surgery



# Hemodynamic support setting

A



- Figure A. Angiographic view during coronary angiography.
  - Blue arrow ---> Surgery mitral ring
  - Red arrow (wide) ---> Venous VA-ECMO cannula
  - Red arrow (narrow) ---> Intraortic balloon pump
  - Green arrow --> Pericardial surgical drainage

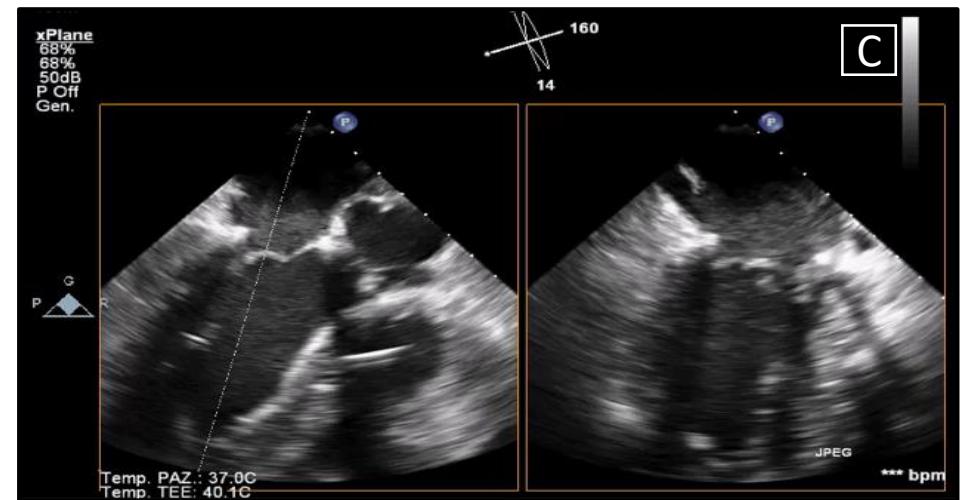
B



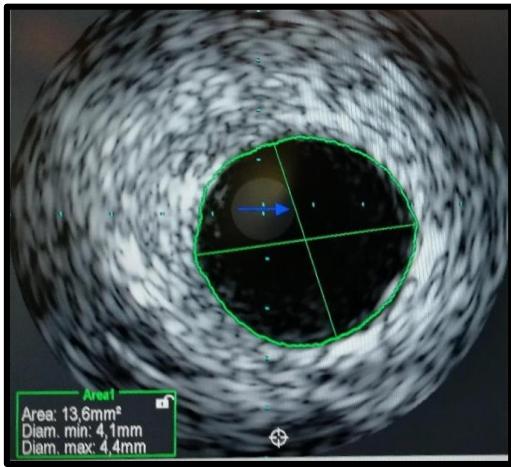
## Transesophageal echocardiography

- Figure B ---> Swan Ganz catheter on TEE (yellow star)
- Figure C ---> Smoke effect with inconstant opening of aortic valve due to VA-ECMO LV underloading

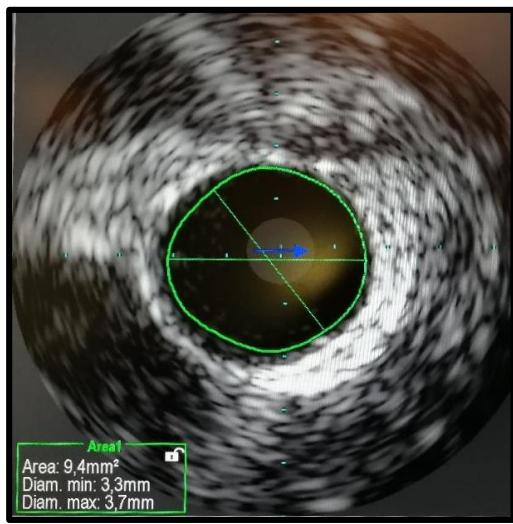
C



# IVUS evaluation during index PCI



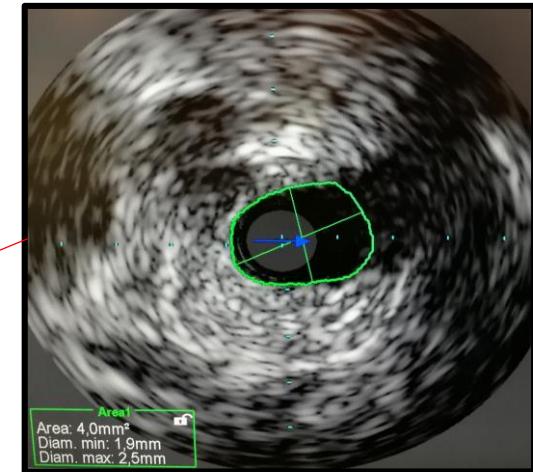
Proximal reference diameter



Distal reference diameter

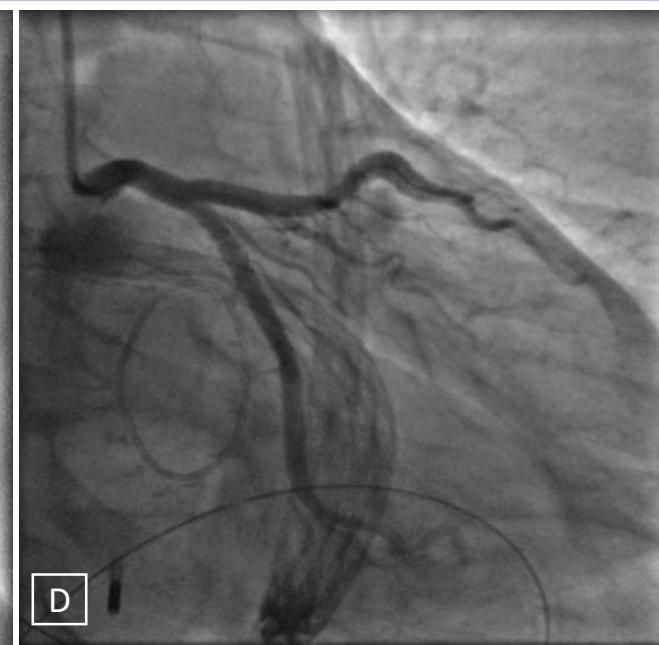


LCx: “eccentric distortion with angiographically intermediate lumen reduction with no evidence of thrombus”



MLA = 4.0 mm<sup>2</sup>

Significant mean lumen area reduction in the absence of atheromasia



*Final result with TIMI flow 3*

# Follow up and Conclusion

## Follow up

In Hospital ---> In the next few days progressive weaning from pharmacological and mechanical support was achieved and patient was transferred to low intensive care unit, hemodynamically stable with negative values cardiac enzymes.

Pre discharge --> Transthoracic echocardiography demonstrated LVEF recovery (LVEF 35-38%) associated to mild mitral regurgitation as mitral annuloplasty outcome.

At discharge --> the patient was in new york class association (NYHA) I and post ischemic therapy with ACE-I and beta blocker was recommended. Oral anticoagulant therapy (OAC) with Warfarin in association with dual antithrombotic therapy (Warfarin plus ASA) for 3 months, followed by life-long ASA alone was recommended.

## Conclusion

Cardiogenic shock (CS) is a common cause of mortality. The use of MCS as bridge to decision give the necessary time to reach a complete assessment of the patients and evaluating the balance between risk and benefit of each interventional strategy. This case show the importance of IVUS during PCI to correct understanding the pathophysiology ST elevation. Furthermore, it represented a useful tool to tailor the best treatment. Indeed, considering the non-atheromatous etiopathology of ACS and the high bleeding setting, anatomical information derived by IVUS was essential to choise the best antithrombotic strategy