STEMI AND INTRACRANIAL HEMORRHAGE; DOUBLE TROUBLE
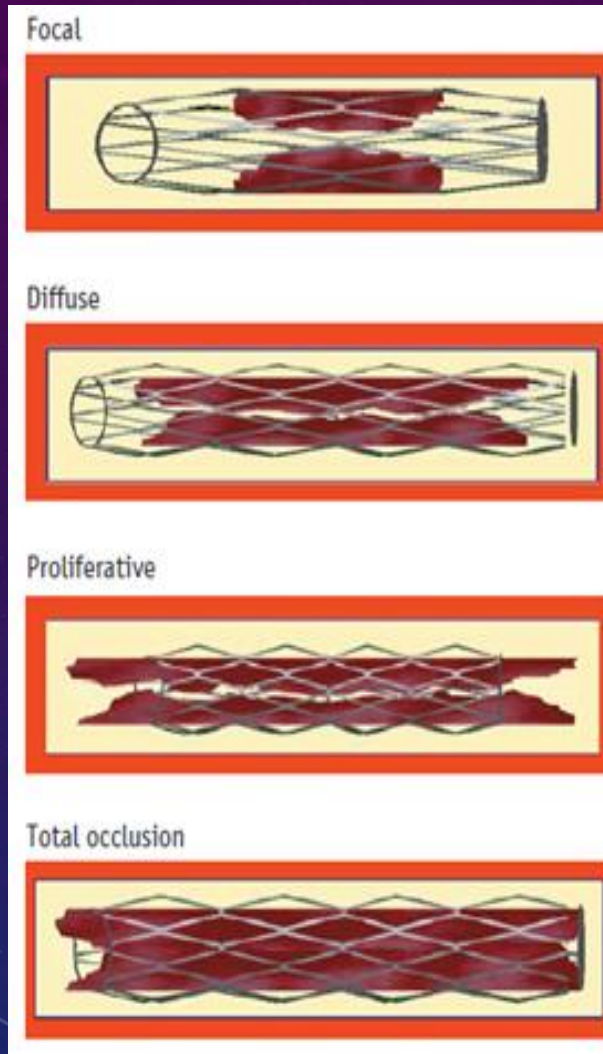
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POTENTIAL CONFLICTS OF INTEREST

Speaker's name : Amir Aziz Alkatiri

- ☑ I do not have any potential conflict of interest

STENT THROMBOSIS

Focal

Diffuse

Proliferative

Total occlusion

Rare complication

Incidence of 2-2.3% after a follow up of 22-30 months

Major clinical impact results in life-endangering conditions

Mortality rate after stent thrombosis ranging from 11% to 45%.

Numerous risk factors → Premature cessation DAPT → strongest single risk factor

1. Oirbans T. et al. interv.cardiol.2011;3:581–8.
2. Claessen BE, et al. JACC Cardiovasc Interv. 2014;7(10):1081–92.
3. Garg S, et al. (J Am Coll Cardiol 2010;56:S1–42)

INTRACRANIAL HEMORRHAGE

- DAPT is cornerstone treatment of PCI, especially ACS
- Intracranial hemorrhage (ICH) is the most serious bleeding complication related to antiplatelet therapy
- DAPT is associated with a 40% increased risk of ICH compared to aspirin alone, with fatality rate up to 60%
- Patients with intracerebral hemorrhage who were treated with DAPT had increased mortality compared with those treated with SAPT.

ICH complication → MACE odds ratio 10 (6.39–15.66, P <0.001) and 30 days mortality OR 21.50 (13.81–33.46, p <0.001)

Post PCI patients experiencing ICH: therapy → problematic

- Antiplatelet cessation increase thrombotic risk, while
- Continue therapy increase bleeding risk

Objective

Managing **Stent Thrombosis and Intracranial Haemorrhage**

Case report

A 59 -year-old male

- Hypertensive
- Severe angina since 4 hours prior to admission
- Nausea
- Diaphoresis

Day 1

- NSTEMI
- PCI 1 DES at mid PDA, 1 DES at prox LAD (2.5/32mm)

- Therapy
  - Acetylsalicylic acid 80 mg OD
  - Clopidogrel 75 mg OD
  - Enoxaparin 0.6 cc BD

Day 3

- Sudden onset headache, nausea and vomitus
- Brain CT scan showed bleeding at cerebellum and also brain infarction at basal ganglia
- Withheld DAPT and anticoagulant
- Referred to National Brain Center Hospital

Day 9

- Severe angina with nausea and diaphoresis
- BP 60/30 mmHg
- Prominent ST elevation in the anterior region
- Diagnosis
  - Cardiogenic shock due to acute STEMI anterior
  - H

- Therapy
  - Aspirin loading dosage 160 mg plus clopidogrel 300 mg
  - Dobutamine and norepinephrine support
  - Referred to our hospital
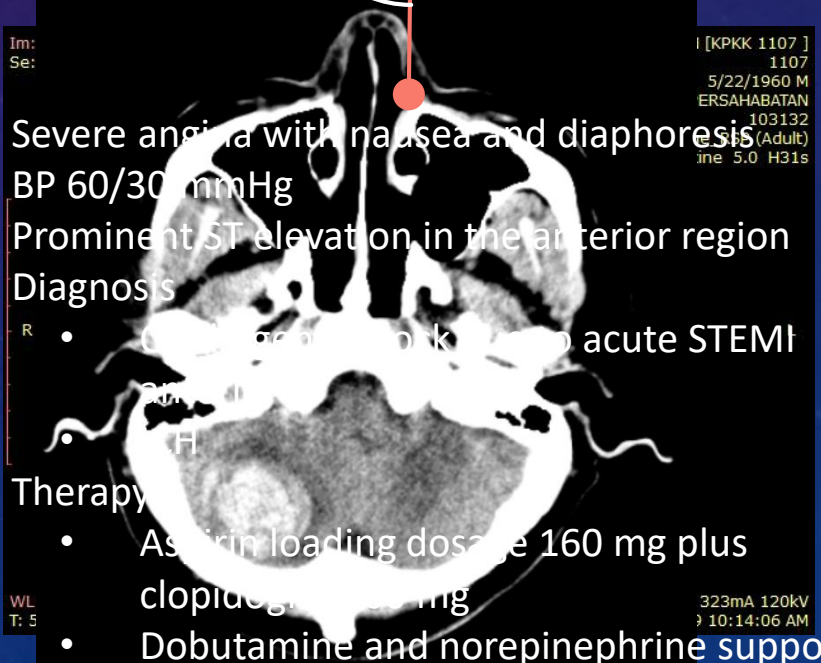
# Patient condition when arriving in our Emergency Room

- Severe chest pain with VAS 10/10.
- Patient was alert
- Blood pressure 106/70 mmHg (on dobutamine and norepinephrine support)
- Heart rate 119 bpm
- Respiratory rate 16 x/min
- SpO2 98% on Oxygen nasal 2 L/m
- Neurologic examination → slight hemiparesis on his left side without any sensory deficits.
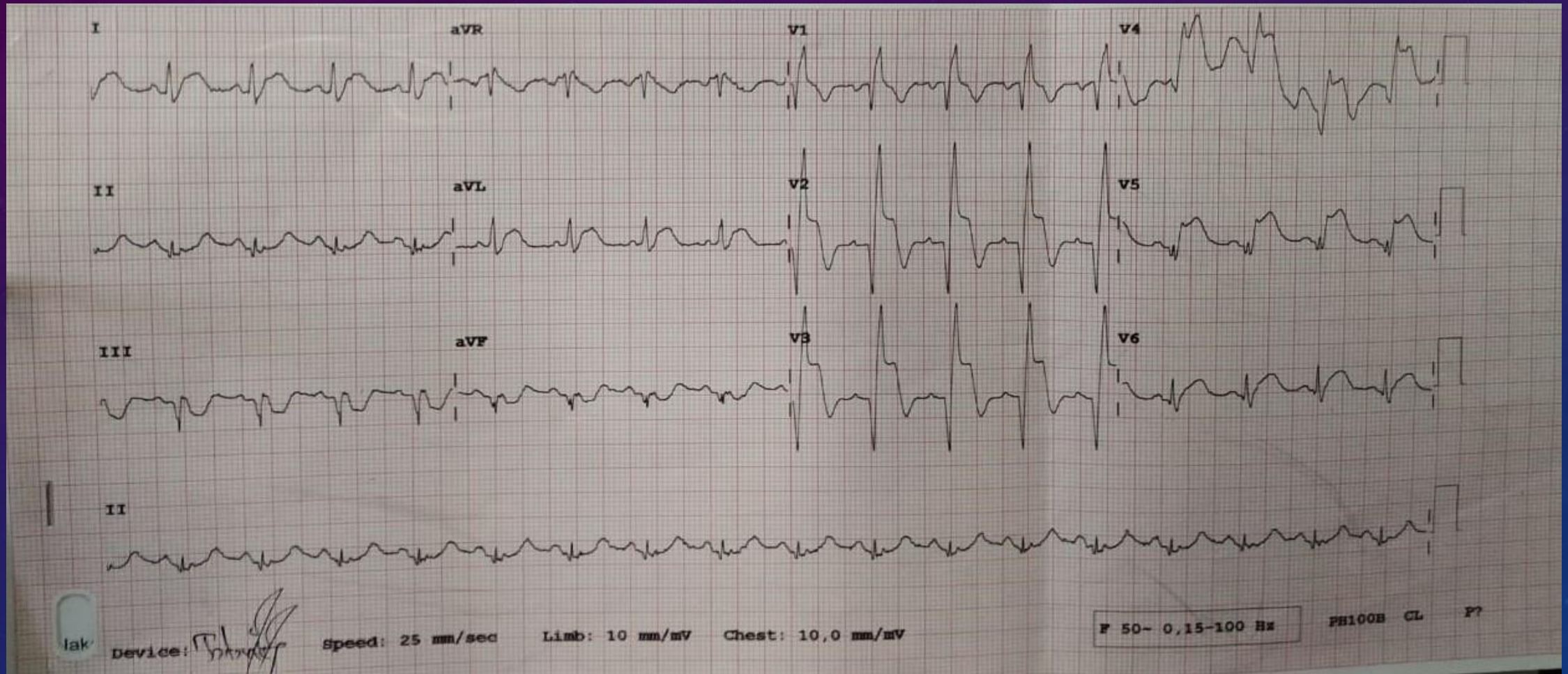
CHEST X RAY AND ECHOCARDIOGRAPHY

- Left ventricle ejection fraction 54% (Simpsons), TAPSE 1,8 cm
- Hypokinetic wall motion at basal-mid anteroseptal, anterior, apical region
- Normal valves
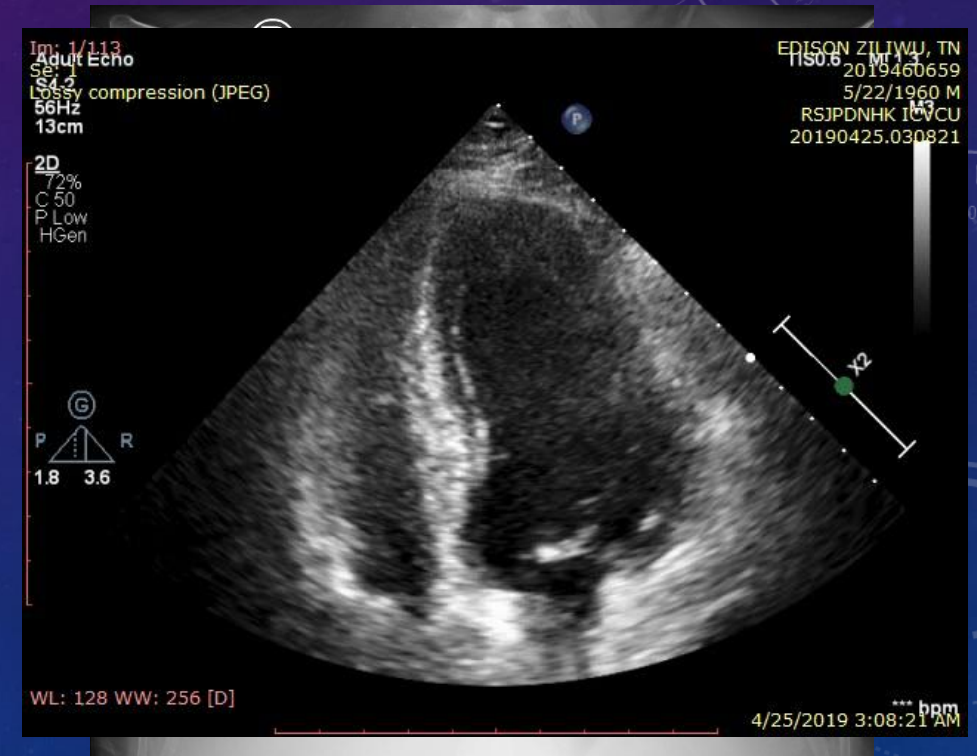
Parameter	Result
Hb	16
Hematocrit	43.6
Leukocyte	14,040
Trombocyte	243,000
Ureum	48.6
Creatinin	1.23
eGFR	60
Sodium	134
Patassium	3.3
Chloride	93
Calcium	2.29
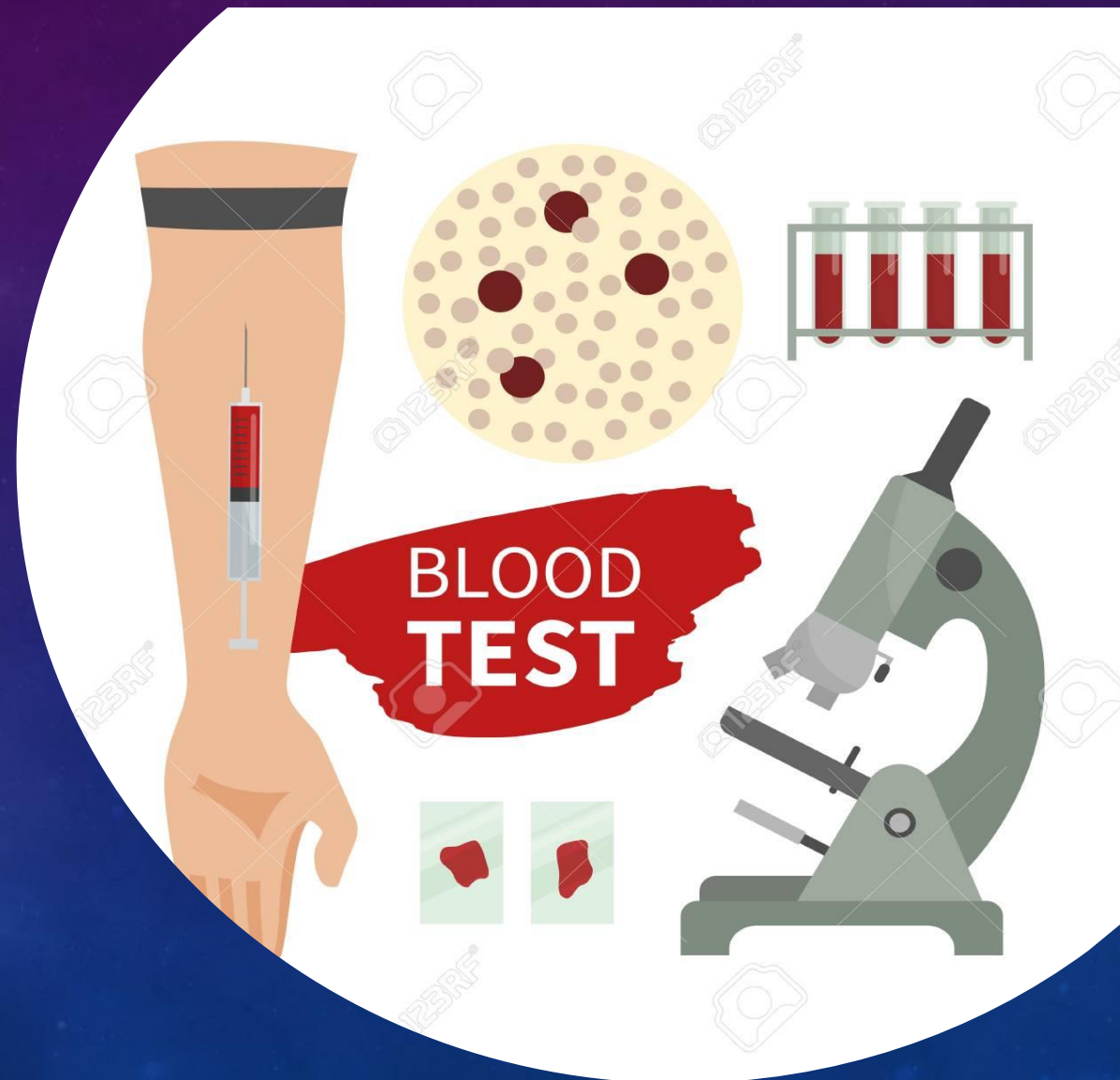
Diagnosis

- Anterior STEMI onset of 4 hours onset Killip IV TIMI 10/14
- Intracranial hemorrhage
- Slight hypokalemia
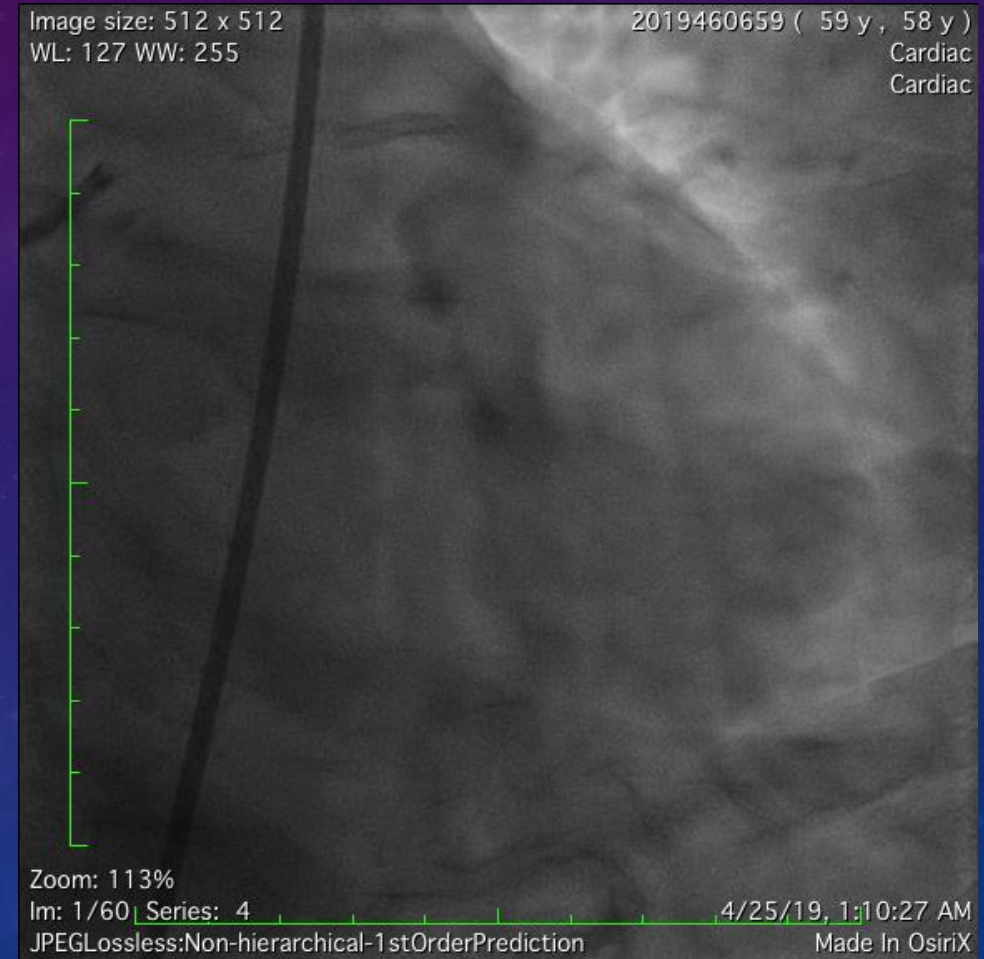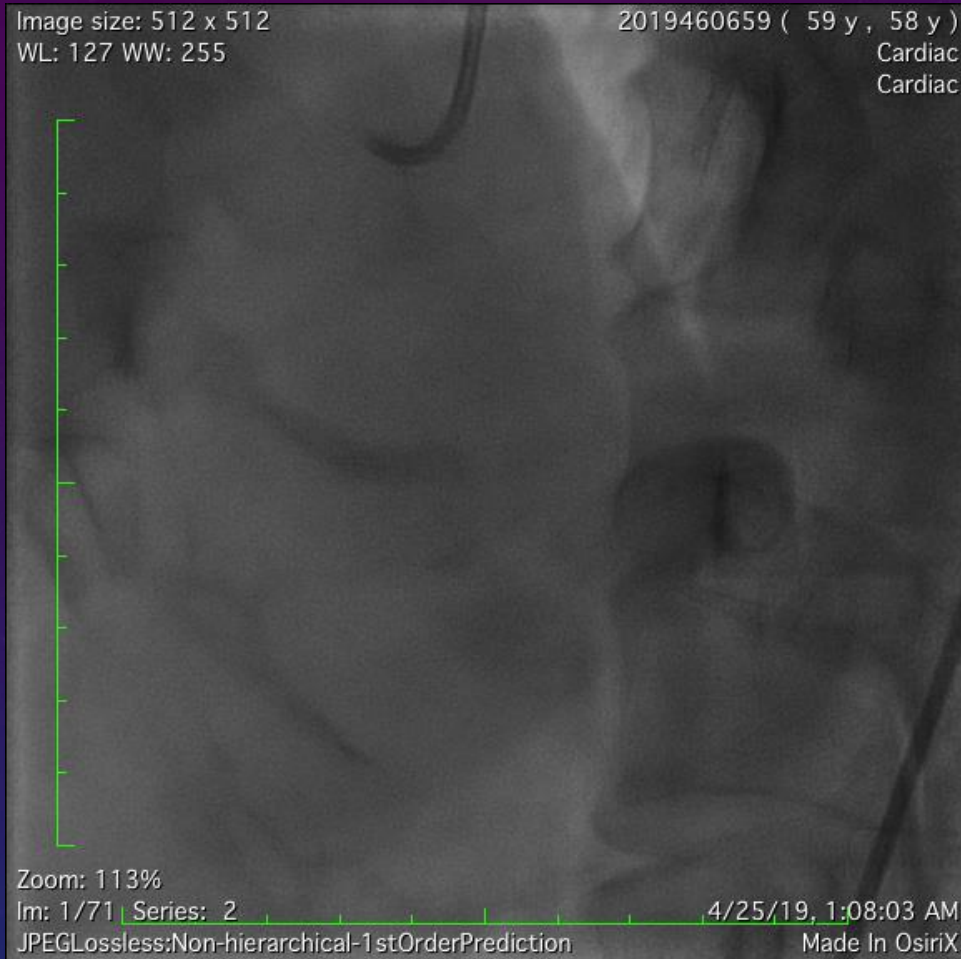
Angiography revealed stent thrombosis at LAD

What is the strategy?

POBA LAD with NC 3.5/15mm
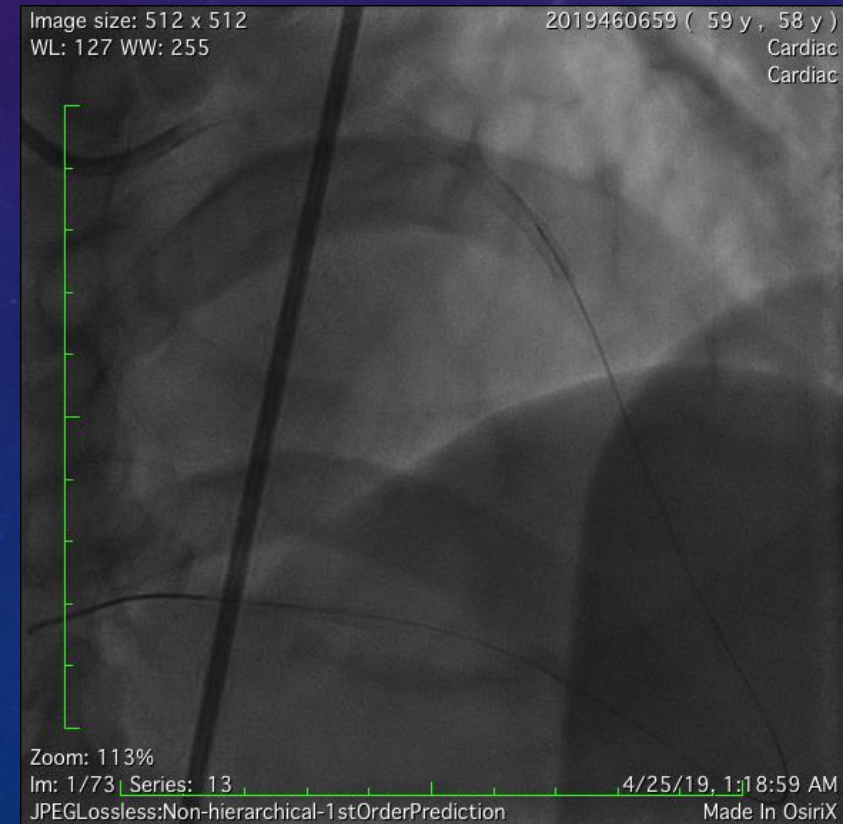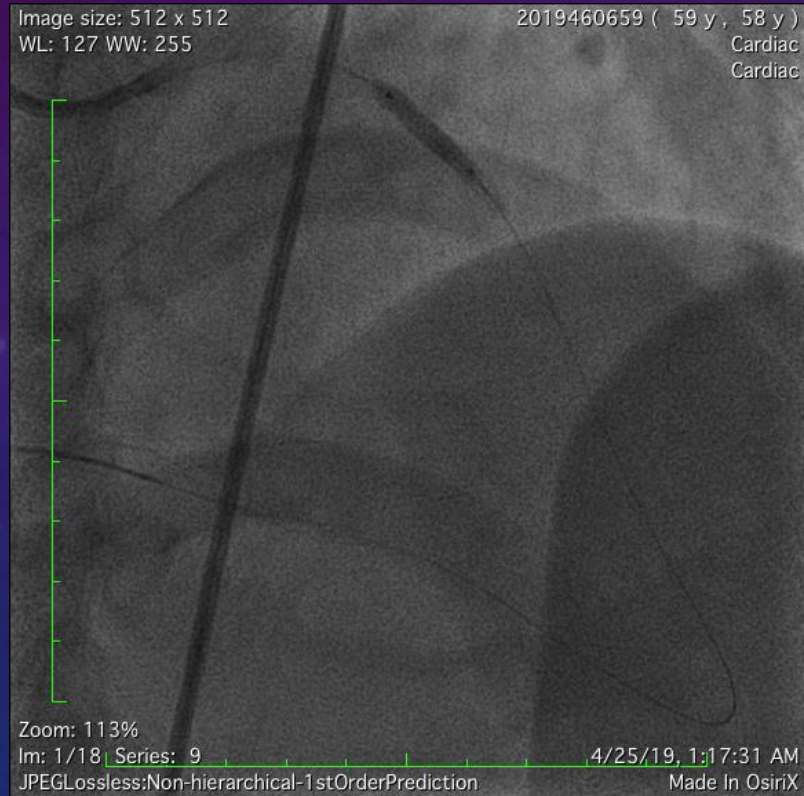
Post POBA LAD

Therapy
- Chest pain alleviated
- Hemodynamic was improved
  - Weaning norepinephrine and dobutamine
- Clopidogrel 1 x 75 mg po

Evaluation
- Aspirin alternate given at 3th day
  - Platelet aggregation
  - Coagulation analysis
  - Clopidogrel resistance analysis → within normal limits
  - CT scan evaluation demonstrated stable ICH

Observation in the intensive cardiovascular care unit

Stable hemodynamic

Subsided recurrent chest pain

Relieving of intracranial haemorrhage symptom

Multidisciplinary team decided uptitrated aspirin once daily after two weeks

Further coronary evaluation can be performed after optimal DAPT therapy

Follow up

- Discharge after 2 weeks
- Plan for DAPT 3-6 months
- Angina and neurological symptom improved after 2 months
- Had cardiac arrest and COVID positive few months after

Level of Certainty	Timing
Definite	**Early**
Angiographic or pathological confirmation of partial or total thrombotic occlusion within the peri-stent region	Acute (<24 h)
AND at least 1 of the following additional criteria:	Subacute (24 h to 30 days)
Acute ischemic symptoms	
Ischemic electrocardiogram changes	
Elevated cardiac biomarkers	
Probable	**Late**
Any unexplained death <30 days of stent implantation	31 days to 1 yr
Any myocardial infarction related to documented acute ischemia in the territory of the implanted stent without angiographic confirmation of stent thrombosis and in the absence of any other obvious cause	
Possible	**Very Late**
Any unexplained death beyond 30 days	>1 yr

Academic Research Consortium **Classification of stent thrombosis** based on **level of certainty** and **timing** of events
This patient:
ACS confirmed with stent thrombosis at angiography
ACS event occurred nine days after PCI

Claessen BE, et al. JACC Cardiovasc Interv. 2014;7(10):1081–92.

STENT THROMBOSIS RISK FACTOR

Precipitant of Stent Thrombosis	
Patient Factors	Percutaneous Coronary Intervention for Acute Coronary Syndrome/ST-segment myocardial infarction
	Diabetes Mellitus
	Renal Failure
	Impaired Left Ventricular Function
	Premature Cessation of dual anti-platelet therapy
	Clopidrogel non responsiveness
	Prior Brachytherapy
Lesion Characteristics	Lession/stent length
	Vessel/stent diameter
	Complex lesion (Bifurcation lesions, Chronic total occlusions)
Procedural Factors	Inadequate stent expansion
	Incomplete stent apposition
	Stent deployment in necrotic core
Device factor	Hypersensitivity to drug coating or polymer
	Incomplete endhotelialization
	Stent design

Premature DAPT cessation → **strongest risk factor** for stent thrombosis.
Stent thrombosis risk highest during the first seven days after DAPT cessation (HR 18)

1. **Garg S, et al. Am Coll Cardiol 2010; 56: S1-42**
2. Claessen BE, et al. JACC Cardiovasc Interv. 2014;7(10):1081–92.

Antiplatelet → important role to prevent thrombus formation → spontaneous ICH may occur → worse outcomes and increased mortality

Swedish Register (RIKS-HIA)

- ICH incidence after acute myocardial infarct 0.35%
- Risk factor
  - Advanced age
  - Decreased eGFR
  - Previous ischemic stroke
  - Previous haemorrhagic stroke.
  - Hypertension
  - Anticoagulant therapy

Graipe A, et al. JAHA. 2010;1–9.

MANAGEMENT

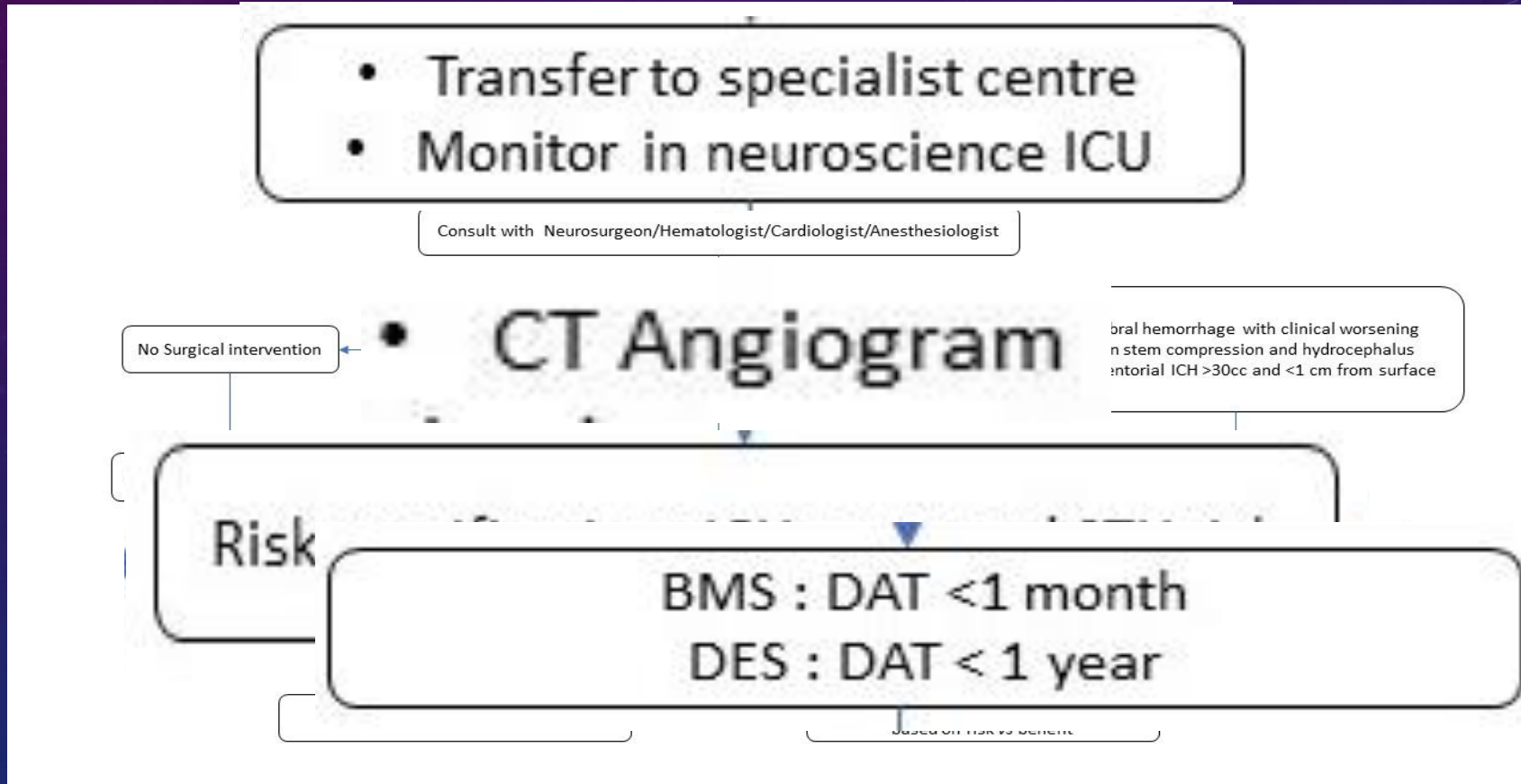There is **no well supported algorithm**

The **decision** to withhold or continue DAPT depends on ischemic vs. recurrent or prolonged bleeding risks

Management based on **expert opinions** and requires **multidisciplinary approaches**

The principle management of ICH post PCI patient:

- Close neurological monitoring
- Early intervention for ongoing intra cerebral hemorrhage expansion
- Avoiding any major cardiovascular events during temporary dual antiplatelet cessation

Treatment algorithm for ICH after PCI

- Transfer to specialist centre
- Monitor in neuroscience ICU

Consult with Neurosurgeon/Hematologist/Cardiologist/Anesthesiologist

No Surgical intervention ← • CT Angiogram

...bral hemorrhage with clinical worsening
...n stem compression and hydrocephalus
...entorial ICH >30cc and <1 cm from surface

Risk

BMS : DAT <1 month
DES : DAT < 1 year

...based on risk vs benefit

Naik BH et al. Anesthesiology 2014; 121:644-53

TAKE HOME MESSAGE

Limited data on the management of stent thrombosis and ICH

Antiplatelet regimens in patients with concomitant stent thrombosis and ICH present an important clinical dilemma

Management of an hemorrhagic stroke with DAPT currently based on expert opinions and requires multidisciplinary approaches.

The decision to withhold or continue DAPT in this setting largely depends on ischemic vs. recurrent/prolonged bleeding risk

THANK YOU