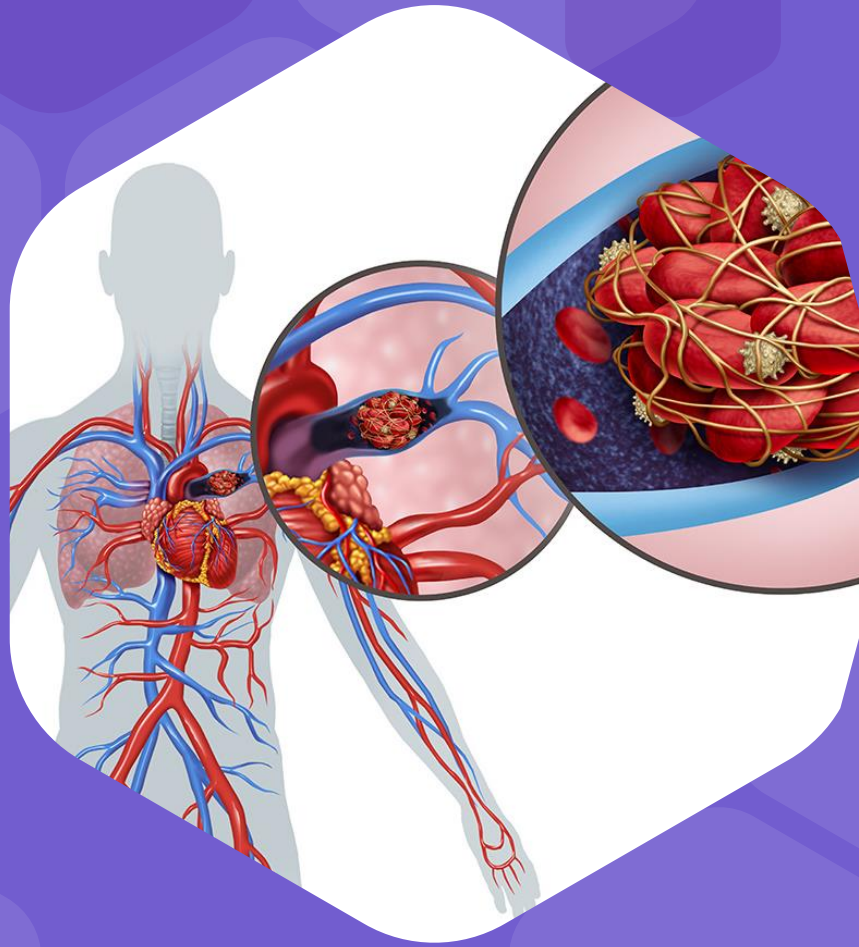
Acute pulmonary embolism (PE)

Dr. Abdulrahman Dakak



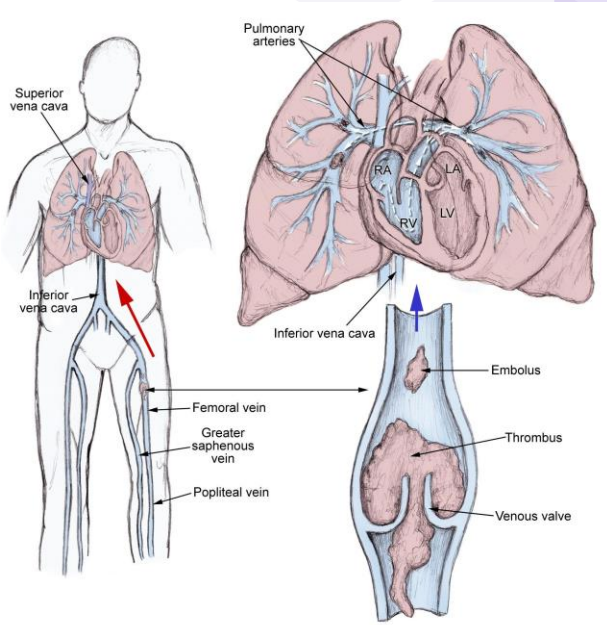
INTRODUCTION:

Acute pulmonary embolism (PE) is a **common** and sometimes **fatal disease**.

The approach to the **evaluation** should be **efficient** while simultaneously avoiding the risks of unnecessary testing so that therapy can be promptly initiated and potential **morbidity and mortality avoided.**

Definition:

Pulmonary embolism (PE) define as a blood clot (**thrombus**) becomes **lodged** in an artery in the lung and **blocks blood flow** to the lung.
usually **arises** from a thrombus that originates in the **deep venous system** of the **lower** extremities
it **rarely** also originates in the **pelvic**, **renal**, **upper extremity veins**, or **the right heart chambers.**



1

RISK FACTOR:

The PIOPED II study listed the following indicators for pulmonary embolism:

- Travel of 4 hours or more in the past month
- Surgery within the last 3 months
- Malignancy, especially lung cancer
- Current or past history of thrombophlebitis
- Trauma to the lower extremities and pelvis during the past 3 months
- Smoking
- Central venous instrumentation within the past 3 months
- Stroke, paresis, or paralysis
- Prior pulmonary embolism
- Heart failure
- Chronic obstructive pulmonary disease

2 CLINICAL PRESENTATION:

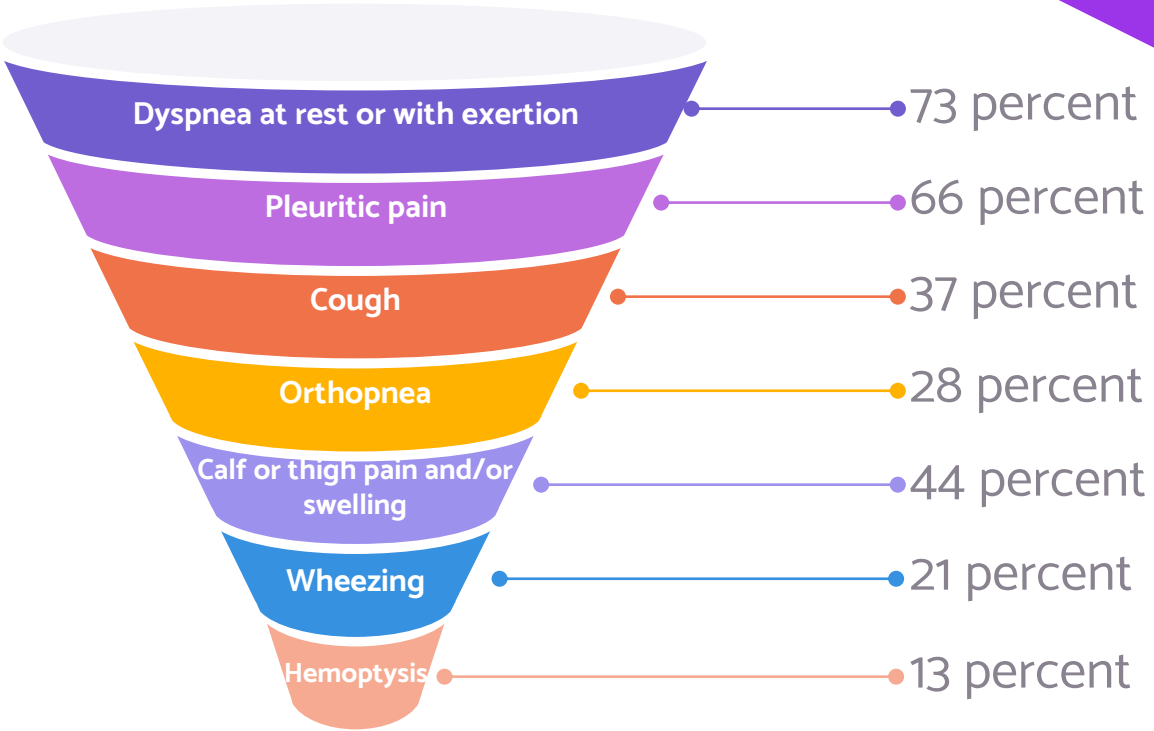
PE has a wide **variety** of presenting features, ranging from no symptoms to shock or sudden death.

The most common presenting symptom is:

- **dyspnea** usually with rapid onset, when main or lobar vessels affected.
- chest pain (classically pleuritic but often dull) due to smaller, more peripheral emboli.
- cough

History and examination

The most common symptoms in patients



Less common presentations include:

Less than 10 percent

- transient or persistent **arrhythmias** (eg, atrial fibrillation)
- **Presyncope**
- **Syncope**
- **hemodynamic collapse**
- **Hoarseness** from a dilated pulmonary artery is a rare presentation (Ortner syndrome)

Laboratory tests

CBC and serum chemistries:

Leukocytosis

ESR + serum lactate + LDH + AST ⇧

Cr and eGFR for the safety of angiography.

(ABG) and pulse oximetry:

Hypoxemia (74 percent).
Widened alveolar-arterial gradient for oxygen (62 to 86 percent)
Respiratory alkalosis and hypocapnia (41 percent).
Hypercapnia, respiratory, and/or lactic acidosis are uncommon but can be seen in patients with massive PE associated with obstructive shock and respiratory arrest.

Laboratory tests

Brain natriuretic peptide (BNP):

Elevated (NT)-proB NP may be useful prognostically for risk stratification of patients diagnosed with acute PE.

Troponin:

useful **prognostically** but not diagnostically

elevations usually resolve within 40 hours following PE, in contrast to the more prolonged elevation after acute myocardial injury

D-dimer:

An **elevated** D-dimer alone is **insufficient** to diagnosis PE, but a **normal** D-dimer can be us **rule out** PE in patients with a **low or intermediate probability** of PE

Electrocardiography

nonspecific

The most common findings are **tachycardia** and nonspecific

ST-segment and **T-wave** changes (70 percent)

S1Q3T3 pattern, **right ventricular strain**, new incomplete

RBBB are **uncommon** less than 10 percent

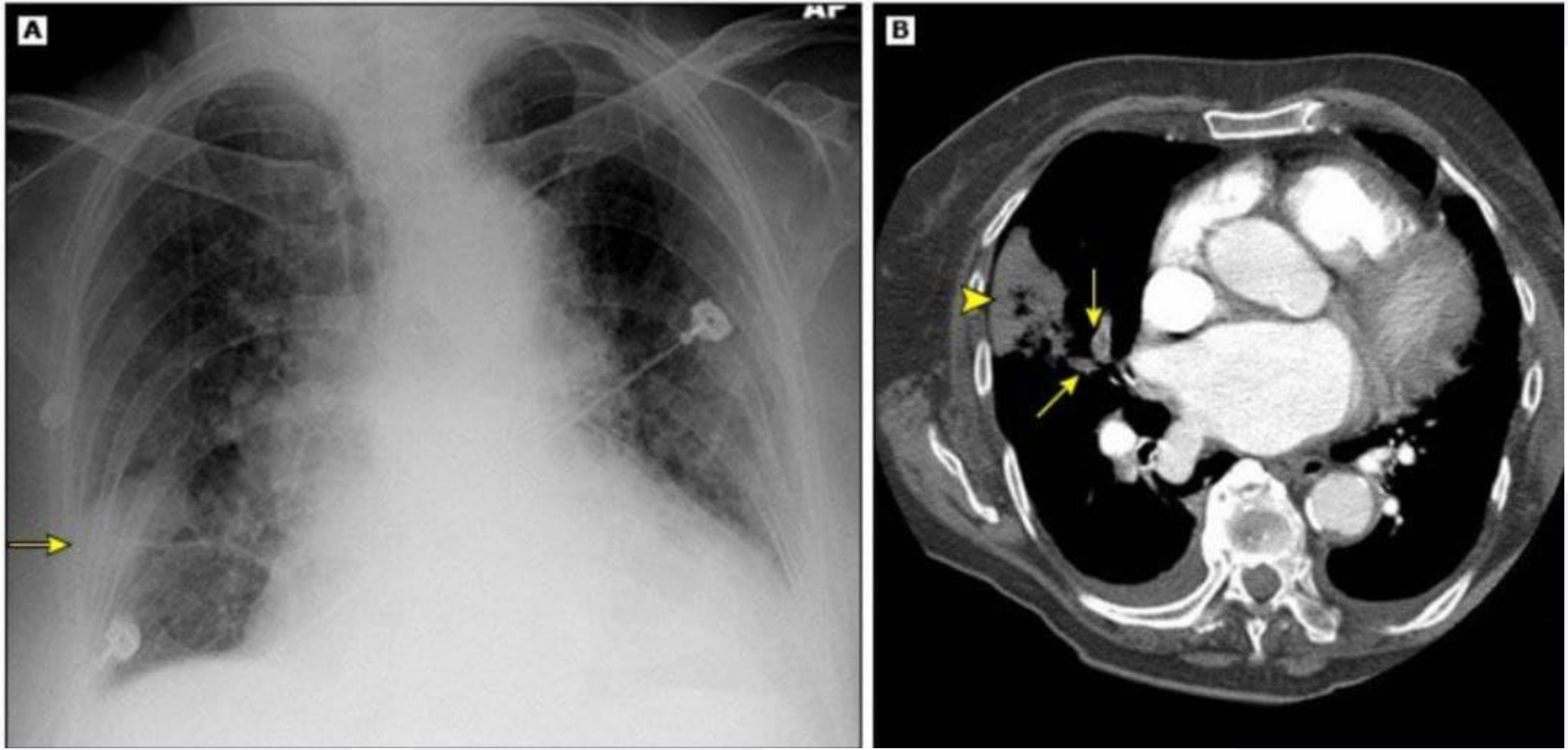
CHEST RADIOGRAPH:

Nonspecific abnormalities on chest radiography

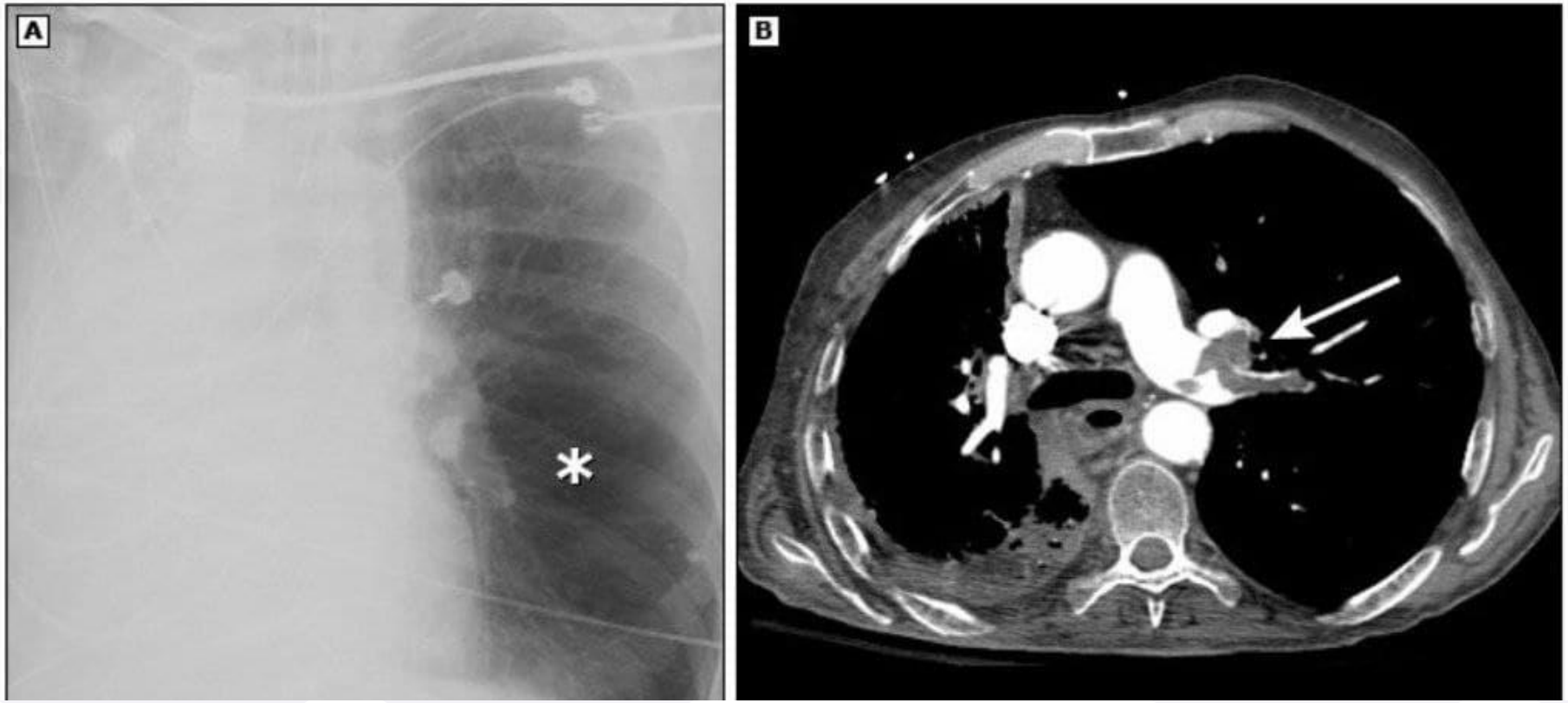
Normal in 12 to 22 percent of patients

performed to look for an **alternative cause** of the patient's symptoms.

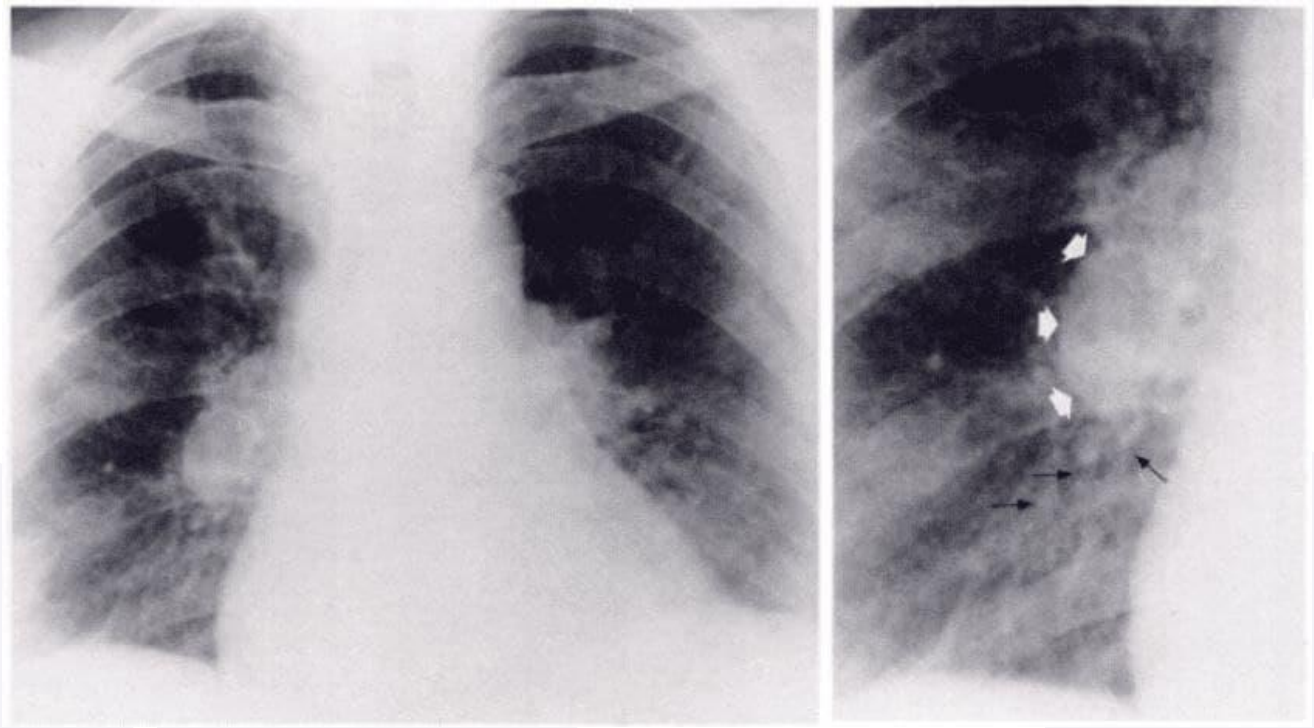
A **Hampton hump**, **Westermark** sign, and **Palla** sign are **rare** but, when present, should raise the suspicion for PE



Hamptons hump in a patient with suspected pulmonary embolus. An anterior-posterior chest radiograph (A) shows a wedge-shaped opacity in the lateral segment of the middle lobe (arrow). CT image through the mid-chest shows the corresponding wedge-shaped opacity (arrowhead) and thrombus in the pulmonary arteries (arrows).



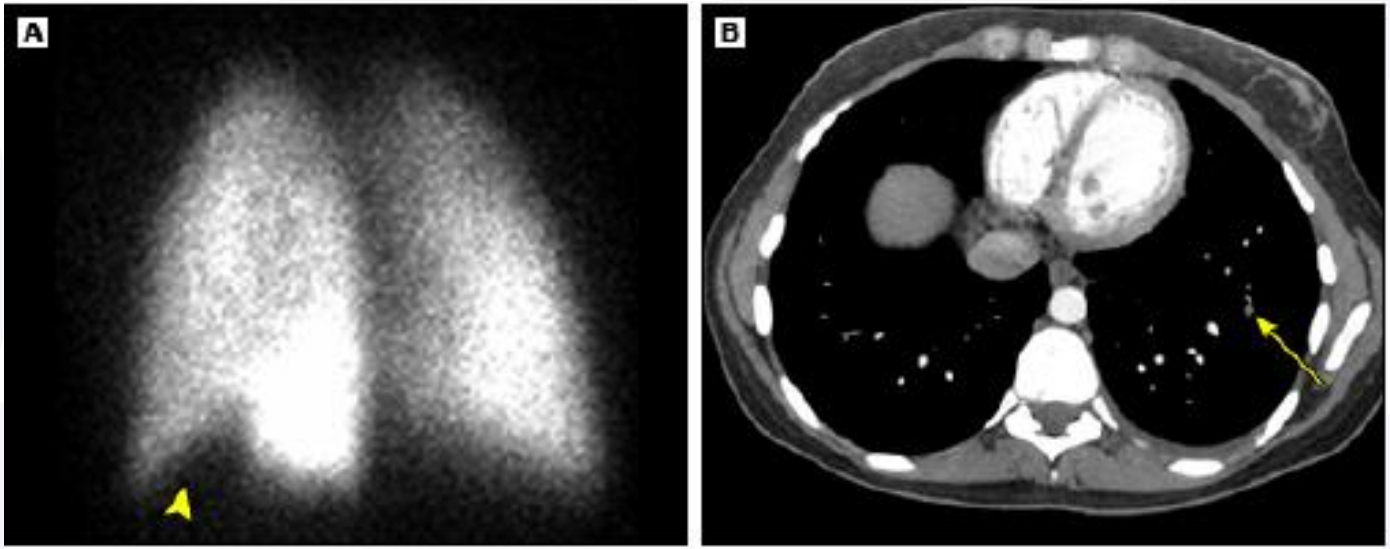
Westermark sign in a patient with occlusive pulmonary embolism.
(A) Chest radiograph magnified A-P view shows a region of oligemia in the left lower lung (asterisk).
(B) Chest CT shows a large thrombus in the left main pulmonary artery (arrow)



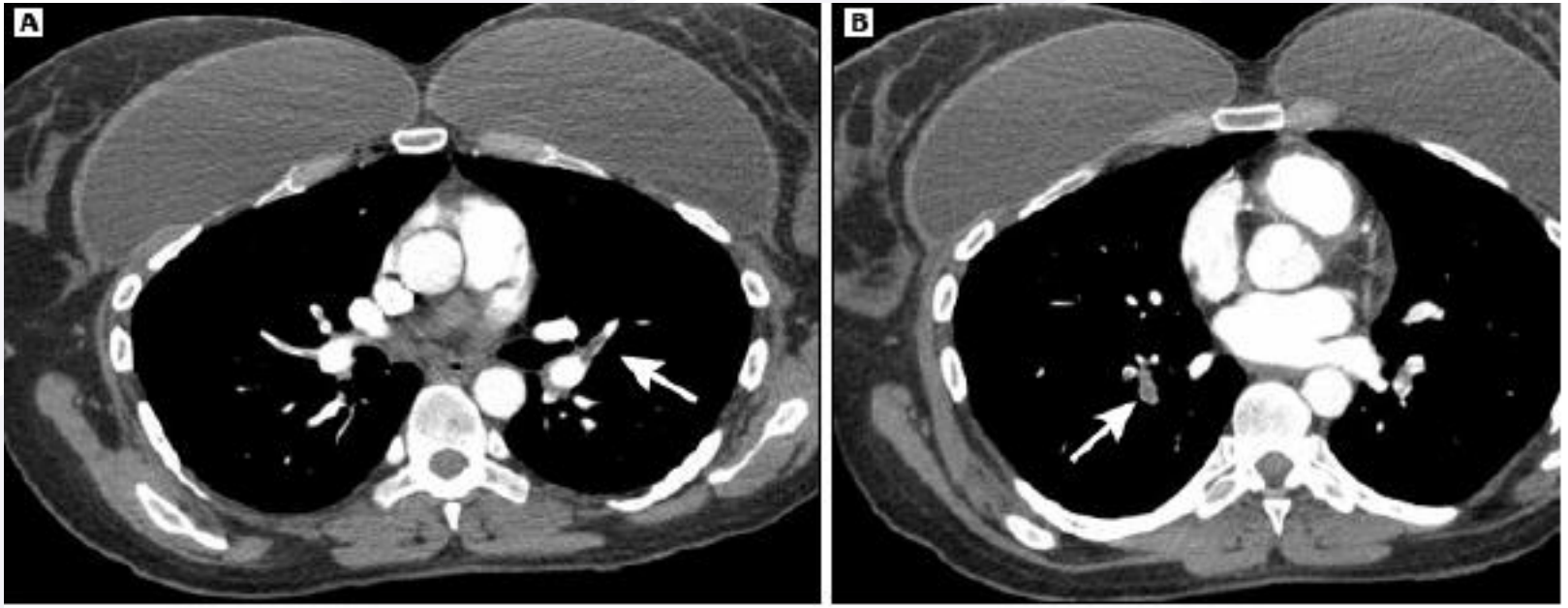
PALLA SIGN

CT pulmonary angiography (CTPA)

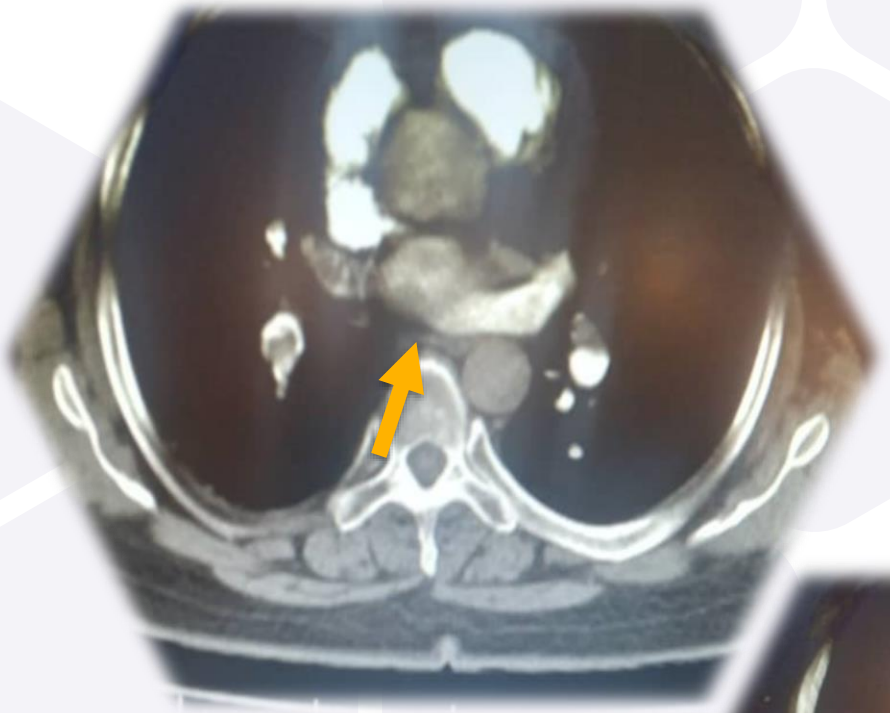
For most patients with suspected PE, CTPA is the first-choice diagnostic imaging modality because it is sensitive and specific for the diagnosis of PE.
A filling defect in any branch of the pulmonary artery (main, lobar, segmental, subsegmental) that becomes evident after contrast enhancement is diagnostic of PE

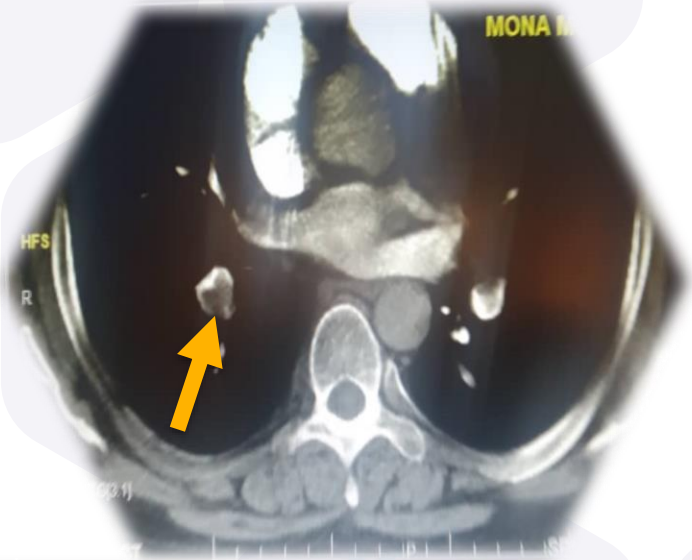


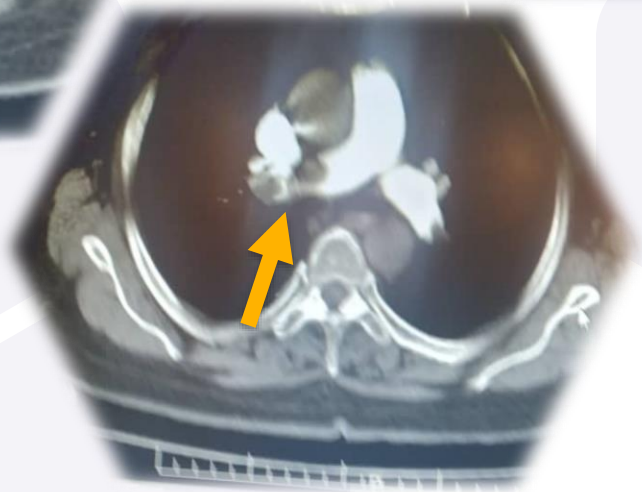
Small pulmonary emboli. VQ scan left anterior oblique view perfusion image (A) shows a subsegmental defect (arrowhead) reported as intermediate probability of pulmonary embolism. Chest CT pulmonary angiogram (B) shows a thrombus in one of the left lower lobe pulmonary artery branches (arrow).

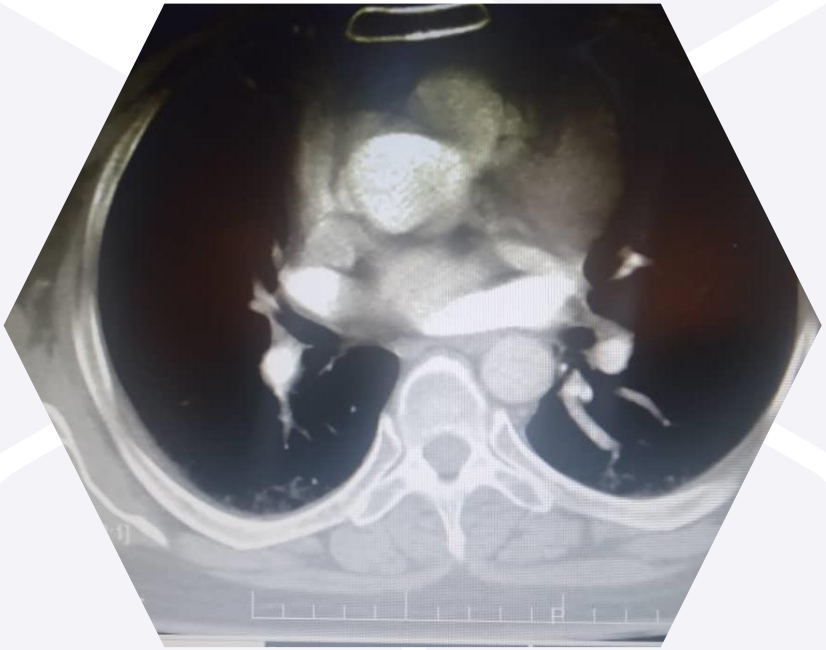


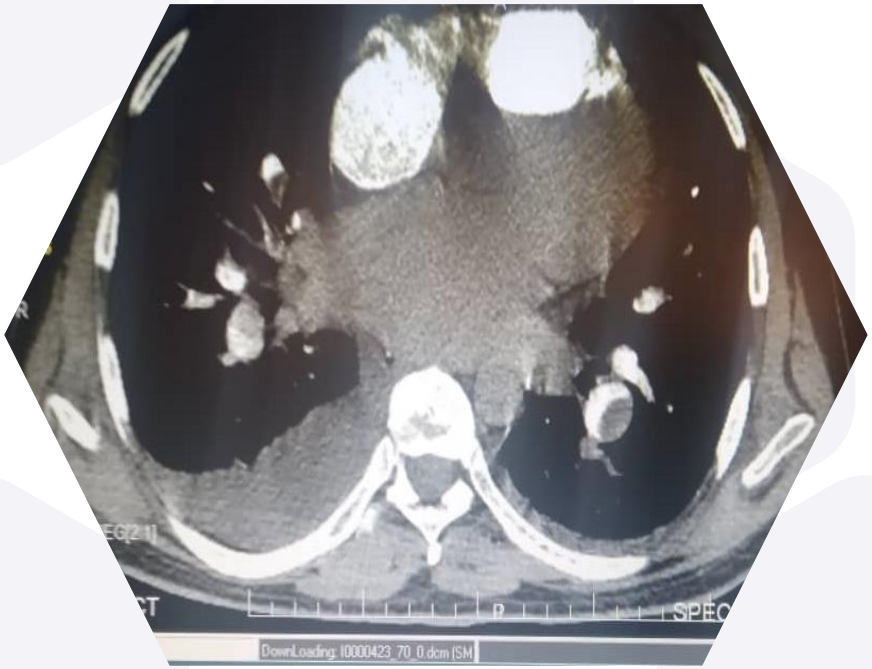
Multifocal pulmonary emboli. Chest CT angiogram images show filling defects in the pulmonary arteries of tthe lingula (A, arrow) and right lower lobe (B, arrow).




HFS
R





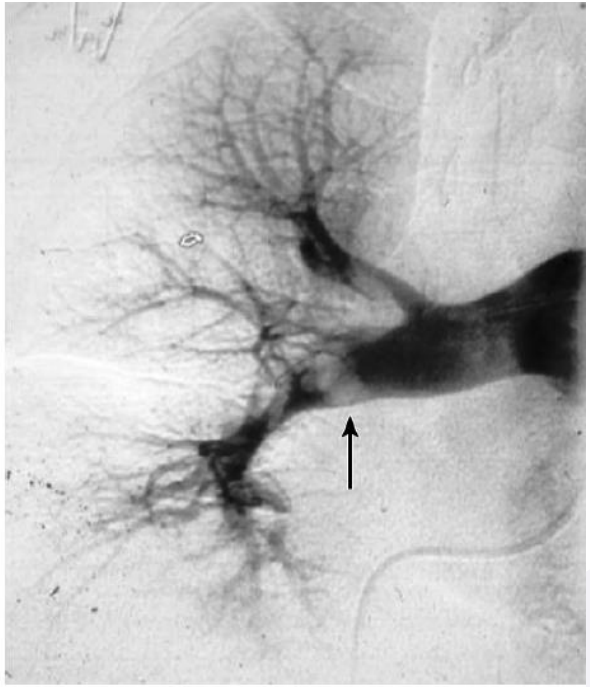

DownLoading: I0000423_70_0.dcm [SM

Ventilation perfusion (V/Q) scanning

A segmental or subsegmental perfusion defect with normal ventilation are diagnostic of PE.
Images are interpreted as high, intermediate, or low probability of PE or normal.
A high-probability V/Q scan and high probability of PE confirms PE. A high-probability V/Q scan and high probability of PE confirms PE.

Catheter-based pulmonary angiography

The demonstration of a filling defect or abrupt cutoff of a vessel is diagnostic of an embolus.



Lower-extremity ultrasound with Doppler

new diagnosis of DVT in the setting of symptoms consistent with PE is highly suggestive, although not definitively diagnostic, of PE.

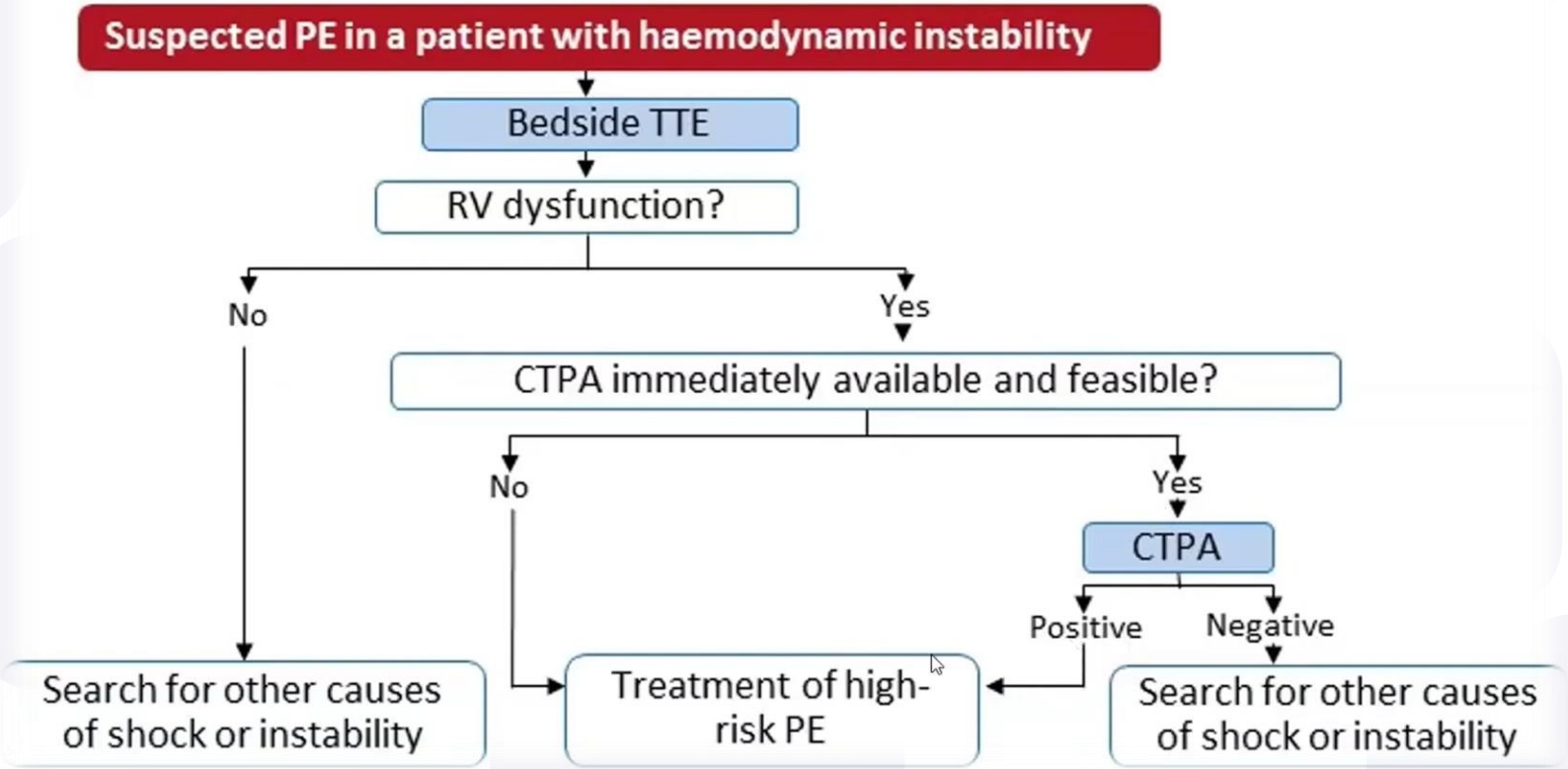
useful for patients suspected of having a PE but definitive imaging is contraindicated, or delayed.

3 DIAGNOSIS

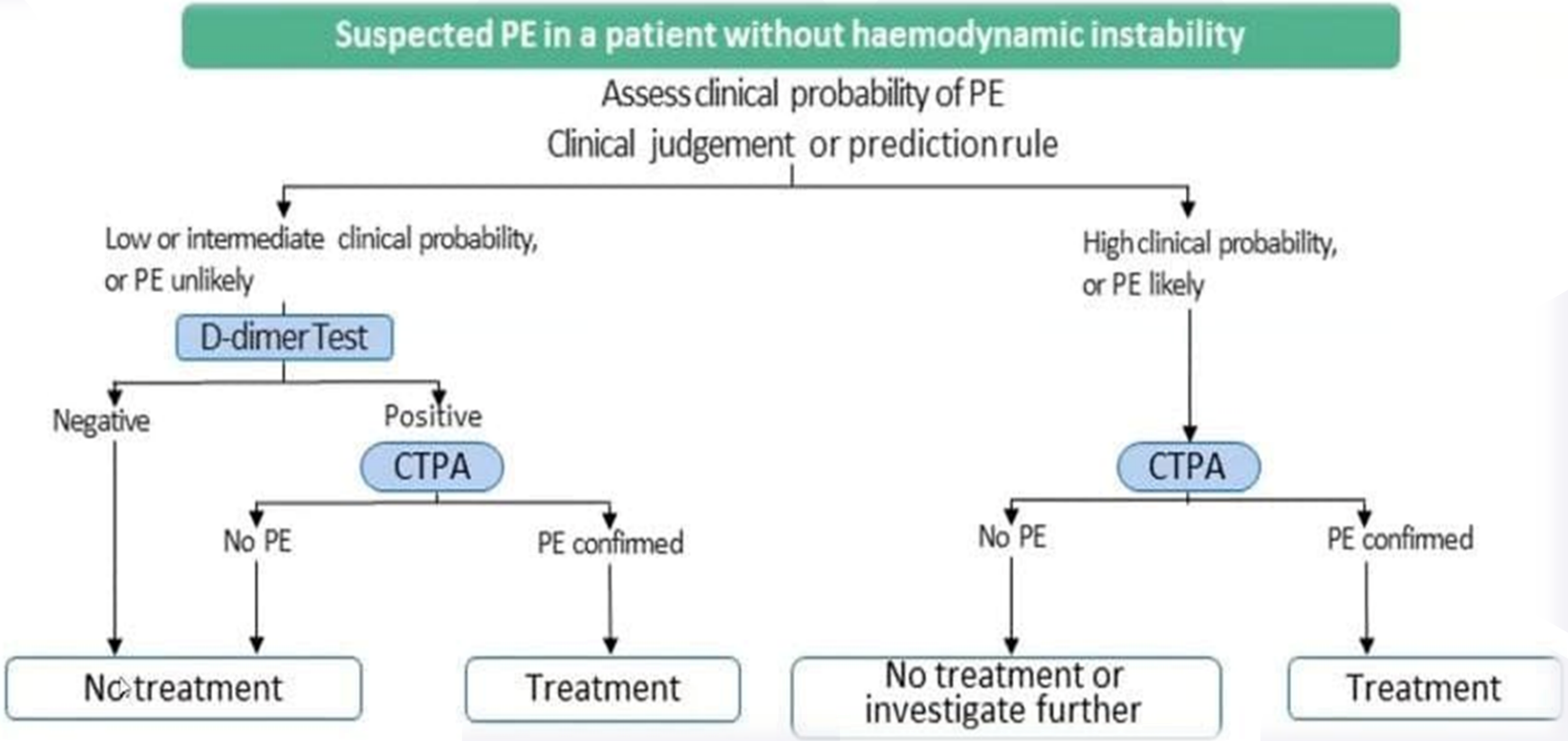
Definition of hemodynamic instability

(1) Cardiac arrest	(2) Obstructive shock	(3) Persistent hypotension
Need for cardiopulmonary resuscitation	Systolic BP <90 mmHg, or vasopressors required to achieve a BP ≥90 mmHg despite adequate filling status ***And*** End-organ hypoperfusion (altered mental status; cold, clammy skin; oliguria/anuria; increased serum lactate)	Systolic BP <90 mmHg, or systolic BP drop ≥40 mmHg, either lasting longer than 15 minutes and not caused by new-onset arrhythmia, hypovolaemia, or sepsis

Diagnostic algorithm for suspected PE haemodynamic instability



Diagnostic algorithm for suspected PE without haemodynamic instability



Assessment of pulmonary embolism severity and the risk of early death

Hemodynamic instability

Clinical parameter of pe severity (sPSI)

Cardiac troponin levels

RV dysfunction on imaging Echo or CT

Original and simplified pulmonary embolism severity index:

predictors	points
Demographic characteristics	
age	1 pt y
Male sex	+10
Comorbid illnesses	
Cancer	+30
Heart failure	+10
Chronic lung disease	+10
Clinical findings	
Pulse >110/min	+20
SBP< 100mmhg	+30
RR> 30/min	+20
Temp< 36c	+20
AMS	+60
Arterial O2 sat	+20

class	score	30 day mortality
I	< 65	1.1%
II	66-85	3.1%
III	86-105	6.5%
IV	106-125	10.4%
V	>125	24.5%

Smplified pulmonary embolism severity index (sPESI)

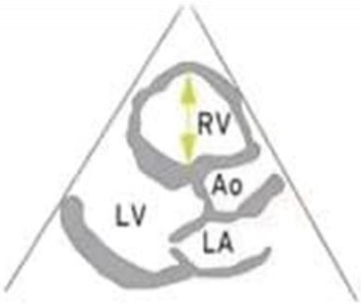
Parameters	*Points*
Age >80 years	+1
History of cancer	+1
History of cardiopulmonary disease	+1
Systolic BP <90 mm Hg	+1
Heart rate >110 beats/minute	+1
O_2 saturation <90%	+1

0 points: = 30 day mortality risk 1.0%
1 point(s) = 30 day mortality risk 10.9%

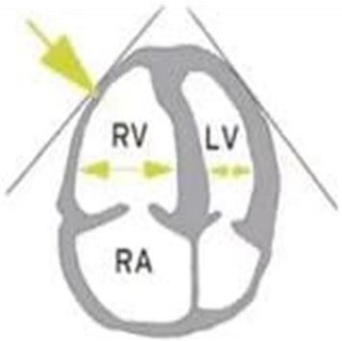
Wells criteria and modified Wells criteria: Clinical assessment for pulmonary embolism

■ Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
■ Other diagnosis less likely than pulmonary embolism	3.0
■ Heart rate >100	1.5
■ Immobilization (≥3 days) or surgery in the previous four weeks	1.5
■ Previous DVT/PE	1.5
■ Hemoptysis	1.0
■ Malignancy	1.0
Probability	**Score**
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

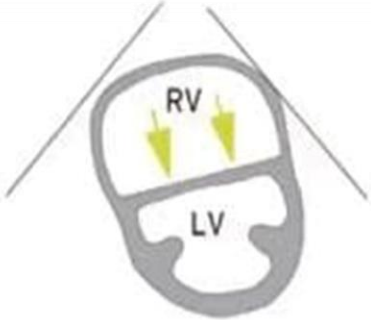
Echocardiography



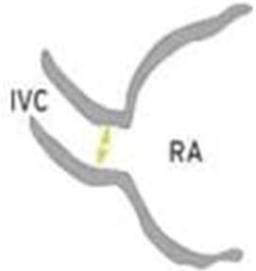
A. Enlarged right ventricle, parasternal long axis view



B. Dilated RV with basal RV/LV ratio >1.0, and McConnell sign (arrow), four chamber view

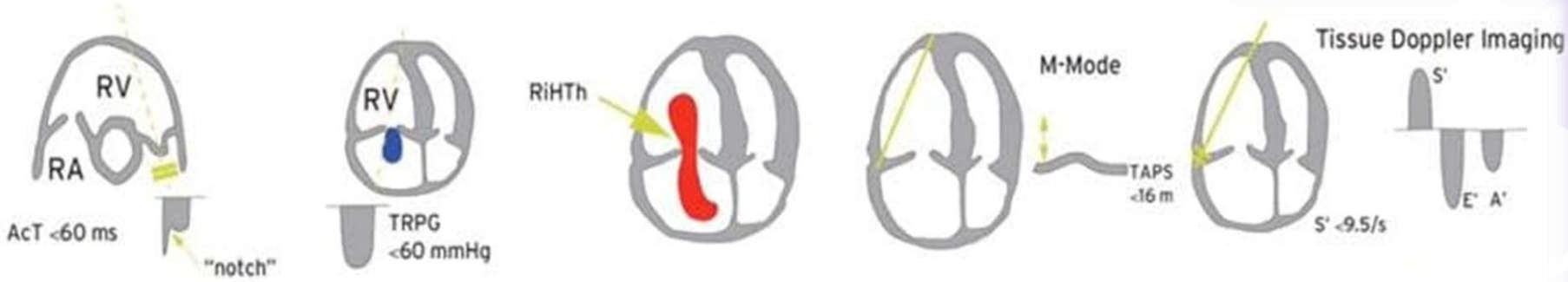


C. Flattened interventricular septum (arrows) parasternal short axis view



D. Distended inferior vena cava with diminished inspiratory collapsibility, subcostal view

Echocardiography



E. 60/60 sign: coexistence of acceleration time of pulmonary ejection <60 ms and midsystolic "notch" with mildy elevated (<60 mmHg) peak systolic gradient at the tricuspic valve

F. Right heart mobile thrombus detected in right heart cavities (arrow)

G. Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<16 mm)

H. Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s)

Acute Pulmonary Embolism Classification:

Early mortality risk		Indicators of risk			
		Haemo-dynamic instability	Clinical parameters of PE severity/ comorbidity: PESI III–V or sPESI ≥1	RV dysfunction on TTE or CTPA	Elevated cardiac troponin levels
High		+	(+)	+	(+)
Interme-diate	Intermediate–high	-	+	+	+
	Intermediate–low	-	+	One (or none) positive	
Low		-	-	-	Assessment optional; if assessed, negative

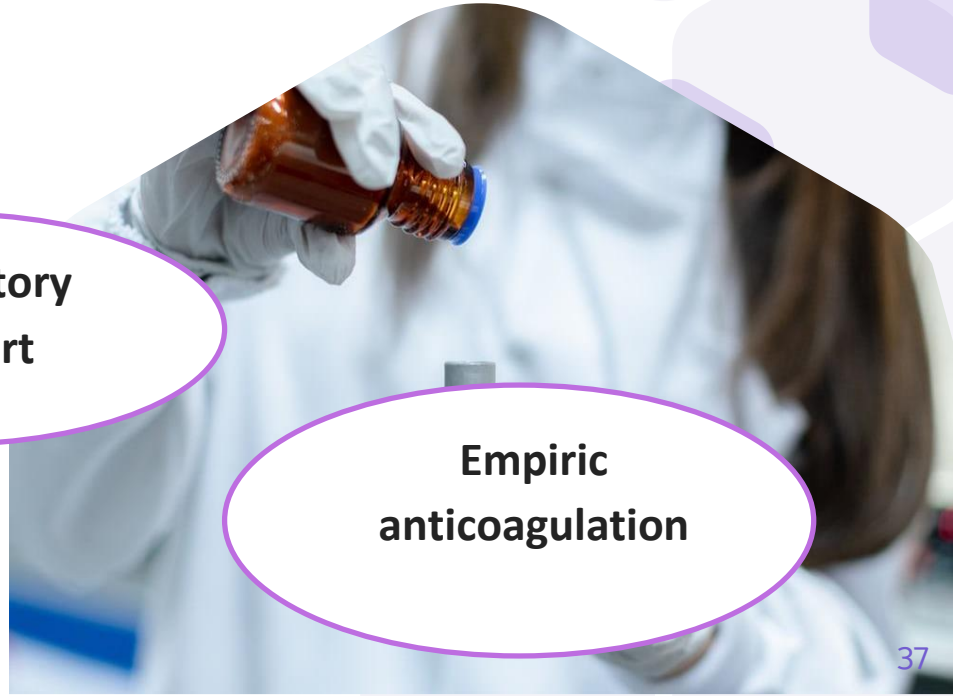
4 TREATMENT

INITIAL THERAPIES

Hemodynamic support

Respiratory support

Empiric anticoagulation



1 Hemodynamic support:

Intravenous fluid

first-line therapy for patients with hypotension

In general, we prefer small volumes usually 250 to 500.

Vasopressors

administered when adequate perfusion is not restored with IVF.

norepinephrine is generally preferred.

2 Respiratory support:

The O2 saturation target ≥90 percent.

Severe hypoxemia and hemodynamic collapse, should

prompt consideration of mechanical ventilation.

THROMPOYTIC THERAPY:

Recombinant tissue type plasminogen activator

Absolut Indication ;

Massive PE

Potential indications:

Patients with severe right ventricular dysfunction due to PE.

Presence of severe hypoxemia.

Patients with acute PE who appear to be decompensating but are not yet hypotensive.

Extensive clot burden.

CONTRAINDICATIONS FOR THROMPOYTIC THERAPY :

Absolute Contraindications	Relative Contraindications
Major trauma, surgery, head trauma within 3 weeks	Cancer
Prior hemorrhagic stroke	Age > 75-80
Ischemic stroke within prior 6 months	Transient ischemic attack within 6 months
Central nervous system neoplasm	Oral anticoagulant therapy
Gastrointestinal bleeding within one month	Noncompressible punctures
Active bleeding	Traumatic resuscitation
	Refractory hypertension
	Advanced liver disease
	Infective endocarditis
	Active peptic ulcer
	Pregnancy or within one week postpartum

For most patients **who do not have hemodynamic compromise** due to acute PE, However, **thrombolysis may be administered on a case-by-case basis** in those assessed to be at the **highest risk of death** from PE,
In whom the benefits are considered by the clinician to outweigh the risk of hemorrhage

THROMPOIYTIC AGENTS:

alteplase:

100mg /2 hours IV
20mg bolus IV over 15 min then 80 mg/ 2 hrs.
Less bleeding

Streptokinase:

250000 units IV over 30 min then 100000 units / hr for 24 hrs.

Urokinase:

4400 units /kg IV over 10 min then 4400 units /kg /hr for 12 hrs.
UK & SK are not longer available for this indication in US.

Following thrombolysis patients should be anticoagulated with IV UFH without bolus.

We avoid LMWH & oral agents.

Empiric anticoagulation:

The administration depends upon the **risk of bleeding**, **clinical suspicion** for PE and the **expected timing of diagnostic** tests.
initial parenteral therapy is recommended with n **(LMWH) above** (**UH**) **infusion** due to the rapid rise of therapeutic drug levels and decreased risk of heparin-induced thrombocytopenia.
Heparin infusions should be considered if there is concern for impending **hemodynamic compromise** and consideration for imminent **endovascular intervention**.

Classified based on risk for bleeding

Low risk for bleeding

We start the treatment with anticoagulation if:

1. Wells score >6
2. Wells score 2 to 6 and the diagnostic evaluation take longer than 4h
3. Wells score <2 and the evaluation take longer than 24 hours

high risk for bleeding

recent surgery, hemorrhagic stroke, active bleeding aortic dissection, intracranial or spinal cord tumors

alternate therapies:

1. inferior vena cava filter,

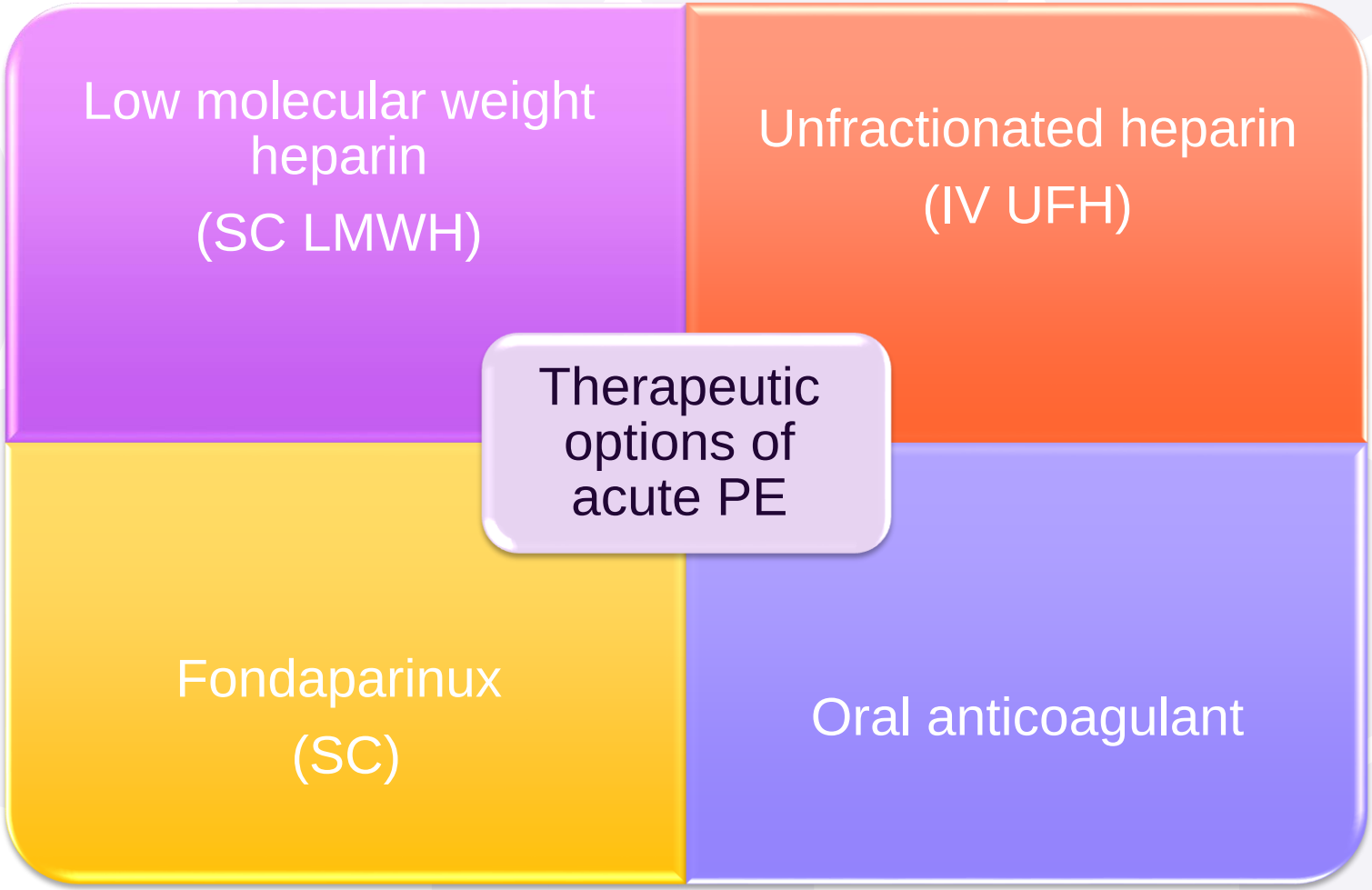
2. embolectomy

can be initiated if PE is confirmed.

Moderate risk for bleeding

anticoagulant therapy administered on a case-by case basis according to the assessed risk-benefit ratio

Typically, **menstruation**, **epistaxis**, and the presence of **minor hemoptysis** are not **contraindications** to anticoagulation but should be **monitored** during anticoagulant therapy.


Low molecular weight heparin
(SC LMWH)
Unfractionated heparin
(IV UFH)
Therapeutic options of acute PE
Fondaparinux
(SC)
Oral anticoagulant

Acute phase treatment of intermediate – low risk PE

Recommendations
Initiation of anticoagulation
Initiation of anticoagulation is recommended without delay in patients with high or intermediate clinical probability of PE, while diagnostic work-up is in progress.
If anticoagulation is initiated parenterally, LMWH or fondaparinux is recommended (over UFH) for most patients.
Oral anticoagulants
When oral anticoagulation is started in a patient with PE who is eligible for a NOAC (apixaban, dabigatran, edoxaban, or rivaroxaban), a NOAC is recommended in preference to a VKA.

Acute phase treatment of intermediate – low risk PE

Recommendations
Reperfusion treatment
Rescue thrombolytic therapy is recommended for patients with haemodynamic deterioration on anticoagulation treatment.
As an alternative to rescue thrombolytic therapy, surgical embolectomy or percutaneous catheter- directed treatment should be considered for patients with haemodynamic deterioration on anticoagulation treatment.
Routine use of primary systemic thrombolysis is not recommended in patients with intermediate- or low-risk PE.

Definitive therapy for confirmed PE

Hemodynamically stable low-risk/nonmassive PE

1.**Low bleeding risk** we start with anticoagulant therapy.

2.**high bleeding risk** inferior vena cava (IVC) filter be placed

Hemodynamically stable intermediate-risk/submassive PE

anticoagulation should be administered and patients monitored closely for deterioration. Thrombolysis and/or catheter-based therapies may be considered.

Definitive therapy for confirmed PE

Hemodynamically unstable PE

No contraindications to thrombolysis

systemic thrombolytic therapy followed by anticoagulation rather than anticoagulation alone.

Contraindications to thrombolysis

catheter or surgical embolectomy rather than observation.

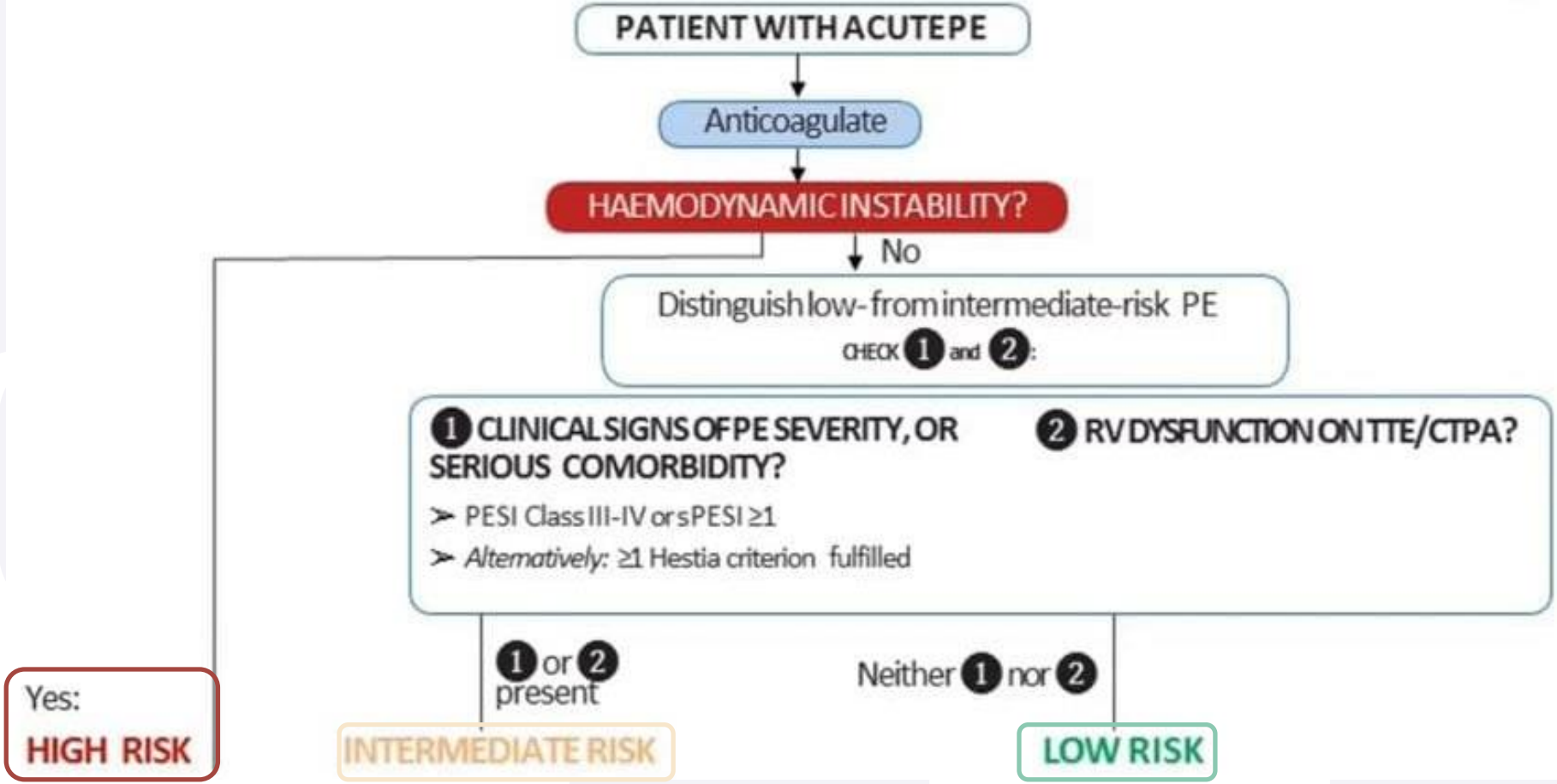
Failed thrombolysis

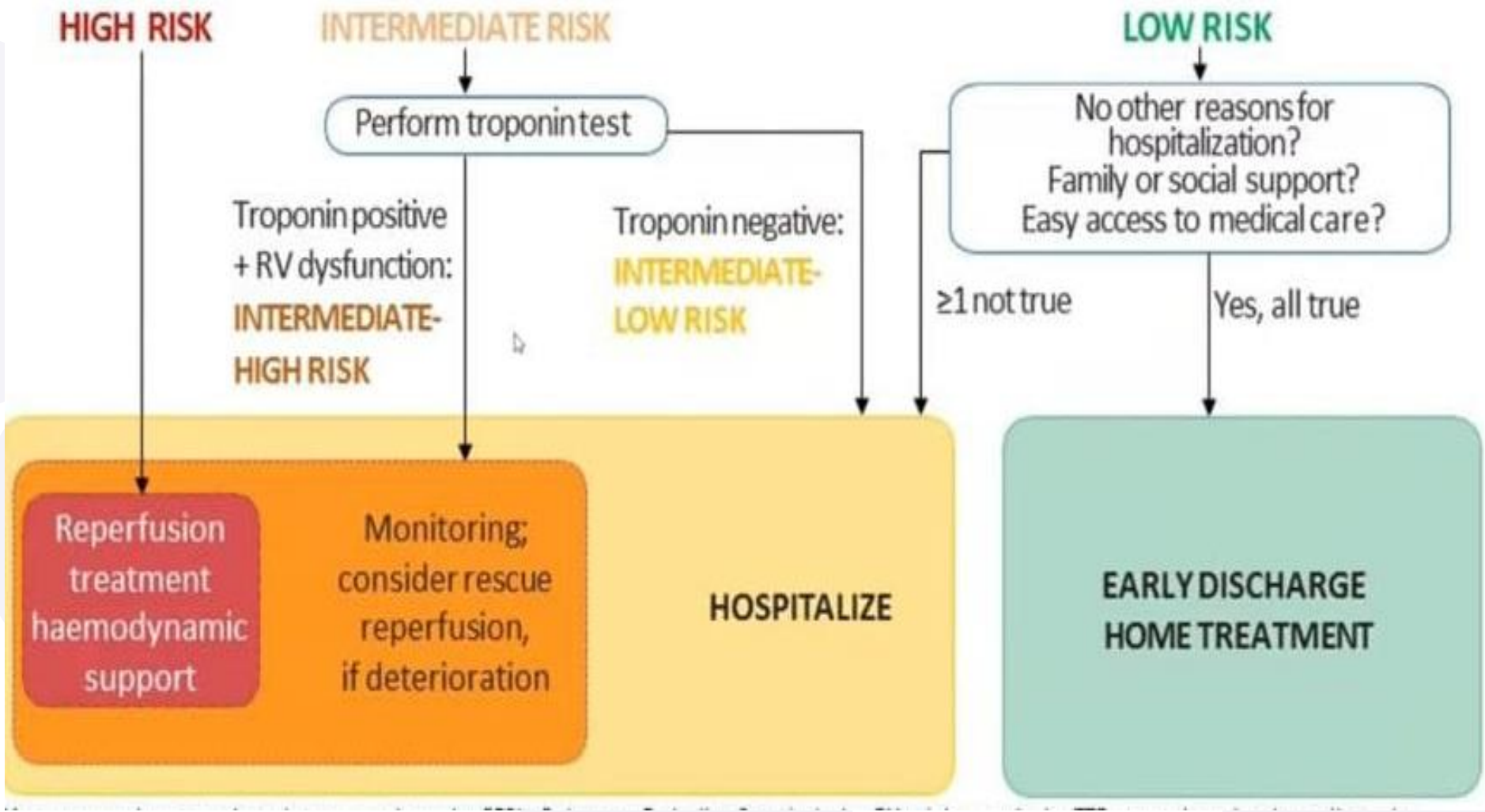
Repeat systemic thrombolysis

catheter-directed thrombolysis

catheter or surgical embolectomy

Mangement strategy for acute PE




HIGH RISK
INTERMEDIATE RISK
LOW RISK
Perform troponin test
No other reasons for hospitalization?
Family or social support?
Easy access to medical care?
Troponin positive + RV dysfunction: INTERMEDIATE-HIGH RISK
Troponin negative: INTERMEDIATE-LOW RISK
≥1 not true
Yes, all true
Reperfusion treatment haemodynamic support
Monitoring; consider rescue reperfusion, if deterioration
HOSPITALIZE
EARLY DISCHARGE
HOME TREATMENT

Lenghth of treatment for PE:

In general, the following applies

Minimum duration: For most patients minimum of three months regardless of whether or not the event was provoked

Extending anticoagulation: we suggest extending anticoagulation for a finite period until the risk factor is resolved, rather than stopping anticoagulation at three months

Monitoring: patients on factor Xa and direct thrombin inhibitors and those >75 years should be monitored clinically for recurrence and bleeding

Bleeding: Major bleeding rates on warfarin and LMW heparin during the first three months of anticoagulant therapy but they are less than 2%.

THANK YOU