Yüksek Riskli Pulmoner Emboli Yönetimi: Olgu Örnekleri

Dr. Nuri Tutar

Erciyes Üniversitesi Tıp Fakültesi Göğüs Hastalıkları Anabilim Dalı

Pulmoner Emboli Sınıflaması

- **ATS 1999, ESC 2000, BTS 2003, ACEP 2003, ACCP 2004, ACCP 2008**

1- MASİF (Kardiak arrest, şok, hipotansiyon)

2- SUB-MASİF (normotansif PE + sağ kalp hastalığı)

3-NON-MASİF (normotansif PE - sağ kalp hastalığı YOK)

Prognozu değerlendirmeye göre yapılmış sınıflama 2014

Table 9 Classification of patients with acute PE based on early mortality risk

Early mortality risk		Risk parameters and scores			
		Shock or hypotension	PESI class II–V or sPESI ≥ I[a]	Signs of RV dysfunction on an imaging test[b]	Cardiac laboratory biomarkers[c]
High		+	(+)[d]	+	(+)[d]
Intermediate	Intermediate–high	–	+	Both positive	
	Intermediate–low	–	+	Either one (or none) positive[e]	
Low		–	–	Assessment optional; if assessed, both negative[e]	

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Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/or comorbidity: PESI class III–V or sPESI ≥I	RV dysfunction on TTE or CTPA[b]	Elevated cardiac troponin levels[c]
High		+	(+)[d]	+	(+)
Intermediate	Intermediate–high	-	+[e]	+	+
	Intermediate–low	-	+[e]	One (or none) positive	
Low		-	-	-	Assesment optional; if assessed, negative

Prognostic Model for Pulmonary Embolism: PESI Score

Class 1	Very low risk	≤ 65
Class 2	Low risk	66-85
Class 3	Intermediate risk	86-105
Class 4	High risk	106-125
Class 5	Very high risk	> 125

Predictor	Points	Predictor	Points
Age	Age in yrs	Clinical findings:	
Male	+10	Pulse ≥ 110/min	+20
Comorbid conditions:		SBP < 100 mm Hg	+30
Cancer	+30	Respiratory rate ≥ 30/min	+20
Heart failure	+10	Temperature < 36° C	+20
Chronic lung disease	+10	Altered mental status	+60
		O_2 saturation < 90%	+20

Aujesky D, et al. *Am J Respir Crit Care Med*. 2005;172:1041-1046.

sPESI

- 1- 80 Yaş üstü
- 2- 90 saturasyon altı
- 3- 100 sistolik kan basıncı altı
- 4- 110 nabız üstü
- 5- Kronik kardiyopulmoner hastalık
- 6- Malignite
- HERBİRİ 1 PUAN, HERHANGİ BİRİNİN OLMASI YETERLİ

PESI Class III to V indicates moderate to very high 30-day mortality risk; sPESI ≥1 point(s) indicate high 30-day mortality risk.

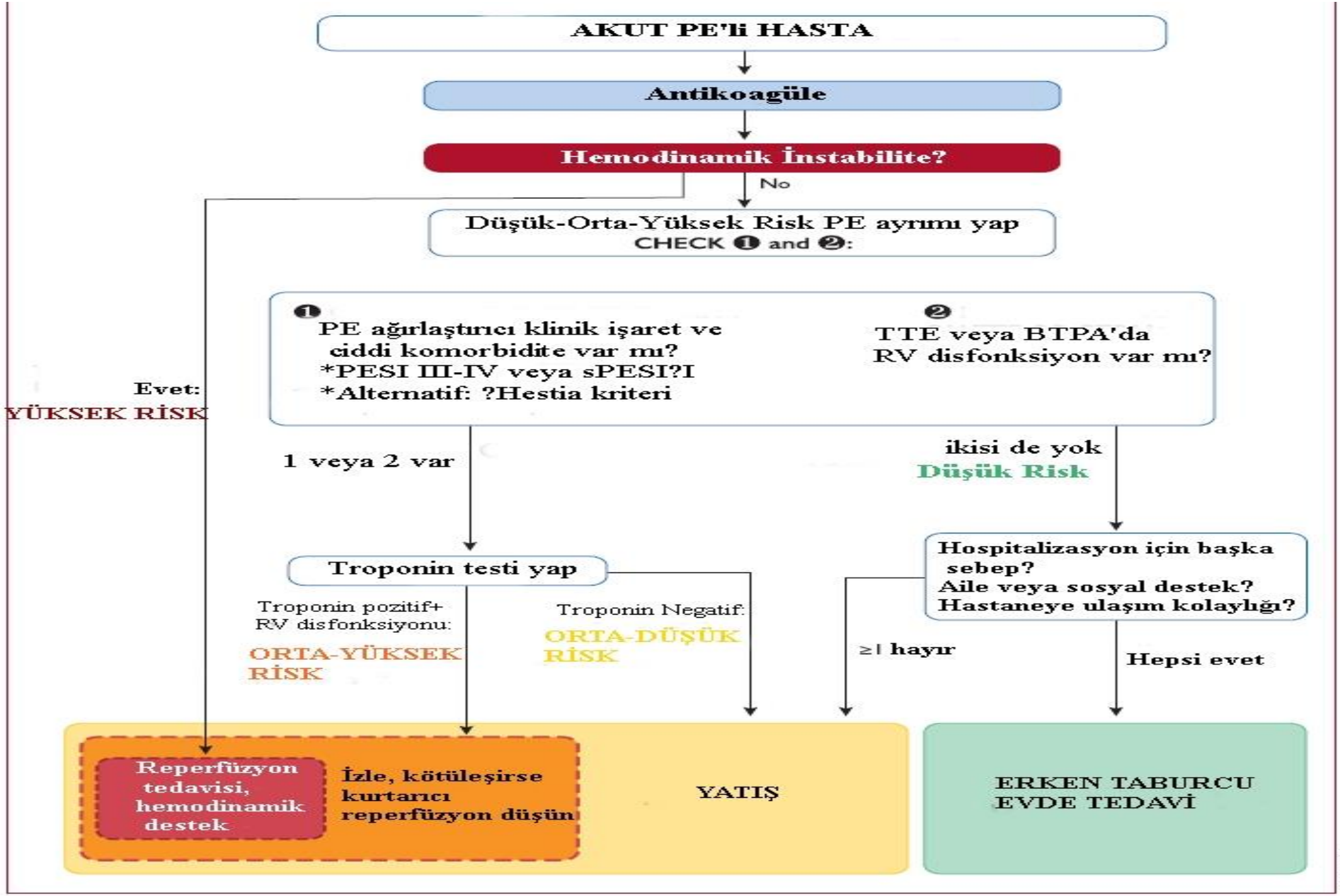
Jimenez D, et al. Arch Intern Med 2010;170:1383-9.

Table 3. Thirty-Day Mortality Within Risk Strata Derived From the Original and the Simplified PESI in the Derivation and Validation Cohorts

PESI Risk Categories	Original PESI Derivation Cohort, % (95% CI)		Simplified PESI Derivation Study Cohort, % (95% CI)	
	Patients (n=10 354)	Deaths[a] (n=953)	Patients (n=995)	Deaths (n=78)
Original				
I	19.4 (18.7-20.2)	1.1 (0.7-1.7)	14.3 (12.1-16.4)[b]	2.1 (0.2-4.5)
II	21.5 (20.7-22.3)	3.1 (2.5-4.0)	22.0 (19.4-24.6)	2.7 (0.6-4.9)
III	21.7 (20.9-22.5)	6.5 (5.5-7.6)	27.7 (25.0-30.5)[b]	5.4 (2.8-8.1)
IV	16.4 (15.7-17.1)	10.4 (9.0-11.9)	21.5 (18.9-24.1)[b]	10.3 (6.2-14.3)
V	21.0 (20.3-21.8)	24.5 (22.7-26.9)	14.5 (12.3-16.7)[b]	22.2 (15.4-29.0)
Low[d]	40.9 (40.0-41.8)	2.1 (1.7-2.6)	36.3 (33.3-39.3)[c]	2.5 (0.9-4.1)
High[d]	59.1 (58.1-60.0)	14.0 (13.1-14.9)	63.7 (60.7-66.7)	10.9 (8.5-13.3)
Simplified				
Low			30.7 (27.8-33.5)	1.0 (0.0-2.1)
High			69.3 (66.5-72.2)	10.9 (8.5-13.2)

- Sağ ventrikül disfonksiyonu için EKO da RV dilatasyonu ve /veya diastol sonu RV/LV oranının 0.9 veya 1.0 ın üstünde olması; RV serbest duvar hipokinezisi; veya BT de diastol sonu RV/LV oranının 0.9 veya 1.0 ın üstünde olması olarak tanımlanır.
- Myokard hasarı için **troponin I** veya **T** nin artışı veya sağ kalp yetmezliği sonucu olarak **natriüretik peptid** konsantrasyonunun artması olarak tanımlanır
- **Hipotansiyon veya şok** varsa ne PESI ne de laboratuar testlerine bakmak gerekli değildir.

- **<u>Hipotansiyon açıklama:</u>**
- **Sistolik** kan basıncının 90 mmHg nin altında olması veya hipovolemi-sepsis-yeni başlayan aritmi olmadan 15 dk dan fazla süre **40 mmHg** den daha fazla düşüş yaşaması


AKUT PE'li HASTA
Antikoagüle
Hemodinamik İnstabilite?
No
Düşük-Orta-Yüksek Risk PE ayrımı yap
CHECK ❶ and ❷:
❶
PE ağırlaştırıcı klinik işaret ve ciddi komorbidite var mı?
*PESI III-IV veya sPESI?I
*Alternatif: ?Hestia kriteri
❷
TTE veya BTPA'da RV disfonksiyon var mı?
Evet:
YÜKSEK RİSK
1 veya 2 var
ikisi de yok
Düşük Risk
Troponin testi yap
Troponin pozitif+ RV disfonksiyonu:
ORTA-YÜKSEK RİSK
Troponin Negatif:
ORTA-DÜŞÜK RİSK
Hospitalizasyon için başka sebep?
Aile veya sosyal destek?
Hastaneye ulaşım kolaylığı?
≥I hayır
Hepsi evet
Reperfüzyon tedavisi, hemodinamik destek
İzle, kötüleşirse kurtarıcı reperfüzyon düşün
YATIŞ
ERKEN TABURCU
EVDE TEDAVİ
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Olgu 1: Hipotansiyon?
75 yaş, erkek hasta

Tanı Kodu	Türü	Tanı Adı	*18.08.2023 21:49:00*
I10	Ana Tanı	ESANSİYEL (PRİMER) HİPERTANSİYON	
R07.4	Ana Tanı	GÖĞÜS AĞRISI, TANIMLANMAMIŞ	

Şikayet / Hikaye

HİPOTANSİYON , GÖĞÜS AĞRISI
TA:68/47

HİKAYE

KY TANILI ZİHİNSEL ENGELİNDEN DOLAYI İLETİŞİMİ KISITLI HASTA 2 AY KADAR SÜREDİR GÖĞÜS VE KARIN AĞRISI OLUYORMUŞ. BURAYA GETİRDİKLERİNDE KALP DOKTROU CİĞERLERİNDE SIVI OLMA İHTİMALİNDEN VE BUNUN KY SONUCU OLABİLECEĞİ SÖYLNEMİŞ. BUGÜN ÖĞLEN GİBİ DÜZENLİ ALDIĞI
DİOVAN PULCET ECOPİRİN ALDACTONE ARLECSATRİL ÜRİKOLİZ İLAÇLARININ ALINMASI SONRASI HASTA HİPOTANSİFLESİP SARARMIŞ BAYILMA NOKTASINA GELMİŞ.
ÖĞ: KY, ZİHİNSEL ENGELLİ (DOĞUŞTAN)
OP: KALP ANJİO (BU PAZARTESİ OLMUŞ.)

Karar

ÖNERİLERLE TABURCU EDİLDİ.

YAPILAN İŞLEMLER

KAN
EKG

FİZİKİ BULGULAR

HASTA KOOPERE DEĞİL. VERBAL İLETİŞİM YOK DERECE KISITLI.
GENEL DURUM ORTA

Rapor Numarası : .1171.23122572.2023

Merkez Biyokimya Laboratuvarı Barkod No : 85764279 Numune Türü : SERUM

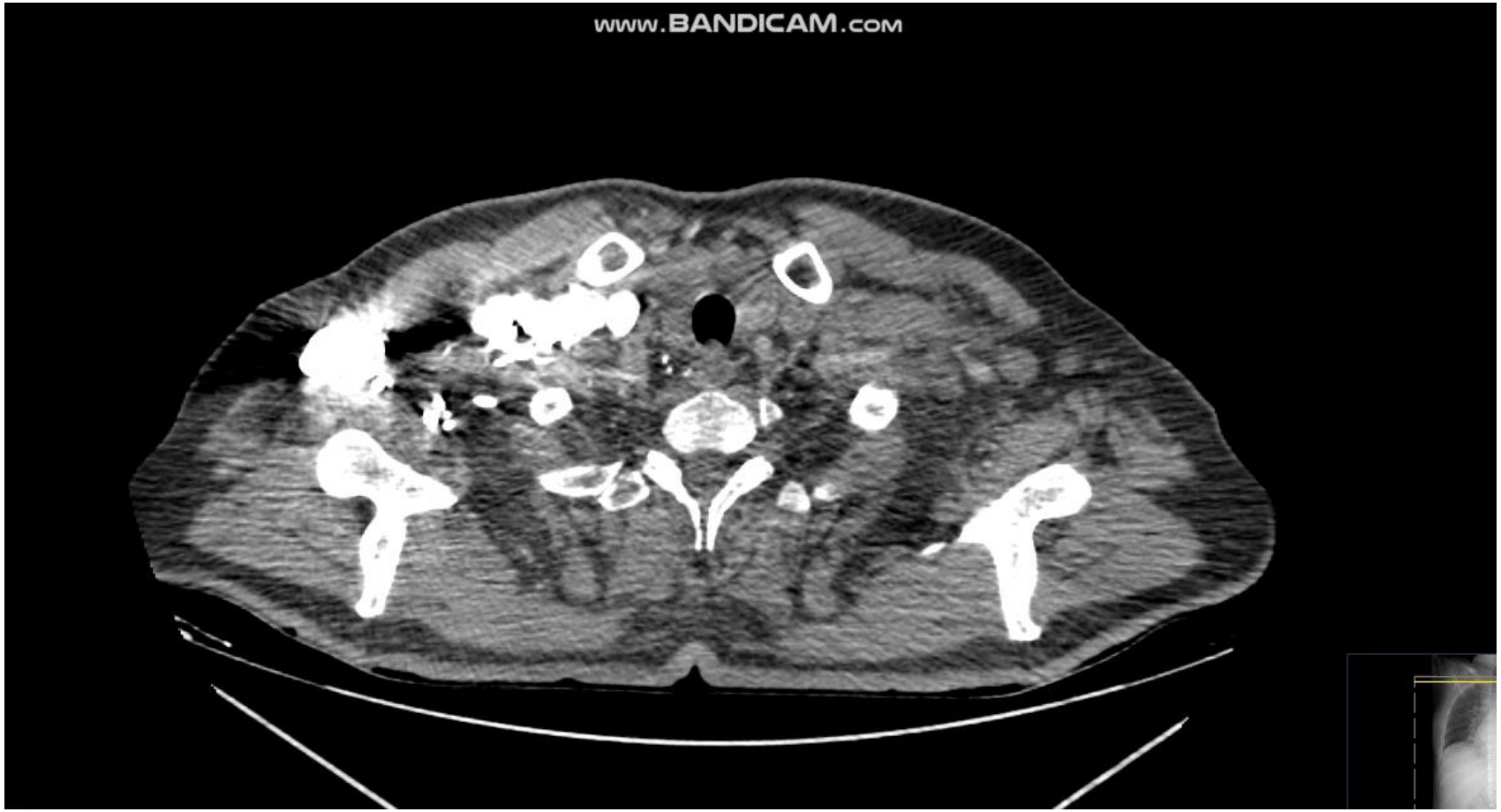
Tetkiki Isteyen :
İNSU YILMAZ
GÖĞÜS HASTALIKLARI SERVİSİ

Tetkik Istem Zamanı : 21.08.2023 14:32
Numune Alma Zamanı : 21.08.2023 14:33
Numune Kabul Zamanı : 21.08.2023 14:57
Uzman Onay Zamanı : 21.08.2023 16:19

Tetkik Adı	Sonuç	Durum	Birim	ReferansAralığı / Karar Sınırı	Onceki Sonuçlar
ACIL KARDIYOLOJI PANELI TETKİKLERİ				Numune Türü : SERUM	
Troponin Ths	**32,8**	**Y**	**ng/L**	**0 - 14**	19.08.2023 00:18.08.2023 23: 26,8 32
IDRAR KIMYASAL TETKIKLERI				Numune Türü : IDRAR	
UROBİLİNOJEN	1		mg/dL	0,2 - 1	
BİLURİBİN	**1**	**Y**	**mg/dL**	**NEGATİF - 0**	
KET (İDRAR)	Negatif		mg/dL	NEGATİF - 0	
KAN	**250**	**Y**	**RBC/µL**	**0 - 10**	
PRO (İDRAR)	**25**	**Y**	**mg/dL**	**0 - 10**	
NIT (İDRAR)	Pozitif		mg/dL	NEGATİF - 0	
LEU (İDRAR)	**100**	**Y**	**Leu/µl**	**0 - 10**	
GLU (İDRAR)	Normal		mg/dL	0 - 50	
PH (İDRAR)	5			5 - 6	
SG (İDRAR)	1.015			1,005 - 1,020	
TURB (İDRAR)	Bulanık				
IDRAR MIKROSKOBISI TETKIKLERI				Numune Türü : İDRAR	
Eritrosit	>409.09		HPF	0 - 3	
BAKTERİ (BACT)	Negatif		HPF	0 - 2	
MAYA (BYST)	Negatif		HPF	0 - 1	
EPIC (İDRAR)	Negatif		HPF	0 - 2	
HYAL (İDRAR)	Negatif		HPF	0 - 2	
MUKUS (MUCS)	Negatif		HPF	0 - 10	
KRİSTAL (UNCX)	Negatif		HPF	NEGATİF - 0	
WBC (LÖKOSİT)	**46**	**Y**	**HPF**	**0 - 4**	
KAN GAZI TETKİKLERİ				Numune Türü : KAN	
PH (KAN)	**7,5**	**Y**		**7,35 - 7,45**	
PCO2 (KAN)	**25,6**	**D**		**35 - 45**	
PO2 (KAN)	**62,3**	**D**		**80 - 100**	
SO2 (KAN)	92,9			75 - 99	
CTO2 (KAN)	**13,5**	**Y**		**0 - 1**	
CHCO3ST (KAN)	**22,3**	**Y**		**0 - 1**	
CHCO3 (KAN)	**19,6**	**Y**		**0 - 1**	
BE (KAN)	**-2,5**	**D**			
BEECF (KAN)	**-3,5**	**D**			
LAC (KAN)	1,25			0,4 - 2,2	
THB (KAN)	**10,4**	**D**		**11,5 - 17,4**	
HCT (KAN)	**31**	**D**		**35 - 50**	

NUMUNE BILGILERI	HIZLI BAKTERİ TANIMLANMASI (MALDI-TOF)(İ	**Barkod No : 85764281**	Numune Türü : IDRAR
MIKROSKOBIK İNCELEME	ESCHERICHIA COLI ÜREDİ.		
MİKROORGANİZMA		**Koloni Sayısı :**	
ANTIBIYOGRAM	**Antibiyotik Adı**	**1** **2** **3**	

NUMUNE BILGILERI	KATALAZ TESTİ	**Barkod No : 85764281**	Numune Türü : IDRAR
MİKROSKOBİK İNCELEME	KATALAZ POZİTİF		
MİKROORGANİZMA		**Koloni Sayısı :**	
ANTIBIYOGRAM	**Antibiyotik Adı**	**1** **2** **3**	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- Tedavi planı: DMAH

Olgu: 2
Pulmoner emboli trombolitik tedavi ne zaman hangi dozda?

- 32 y,
- E
- 5-6 yıldır DM, 2 yıldır hipertansiyon
- BFT bozukluğu saptanınca nefrolojiye gönderiliyor
- BUN:23, Kreat:1.5, proteinüri 1,5 gr
- Sağ böbrek küçülmüş ve parankim kalınlığında artış var, biyopsi amacı ile nefroloji servisine yatırılıyor
- 2 adet bx alınıyor, hemogram düşüşü yok, taburcu ediliyor.
- 3 gün hastane yatışı var.

Yaş / Cinsiyeti	32 / E	Tetkik İsteyen Doktor	AYDIN ÜNAL
DosyaNo	4146235478		
Numune Türü	Tetkik İstem Zamanı 12.11.2015 14:01	Numune Kabul Zamanı	12.11.2015 14:01
	Numune Alma Zamanı 12.11.2015 14:01	Uzman Onay Zamanı	24.11.2015 15:01
Eski Biyopsi No			

KLİNİK BİLGİ:
Proteinüri (+) BilinenDM + HT hastalıkları mevcut

MAKROSKOPİ :
Kayıtsız tüpte gönderilen 3 adet büyüğü 1,2x0,1x0,1 cm, en küçüğü 0,1x0,1x0,1 cm ölçülerinde bej renkli dokular, 0,3 cm lik kısmı immünoflorasan için ayrıldı. Kalan 2 parça 1 kasette takibe alındı.

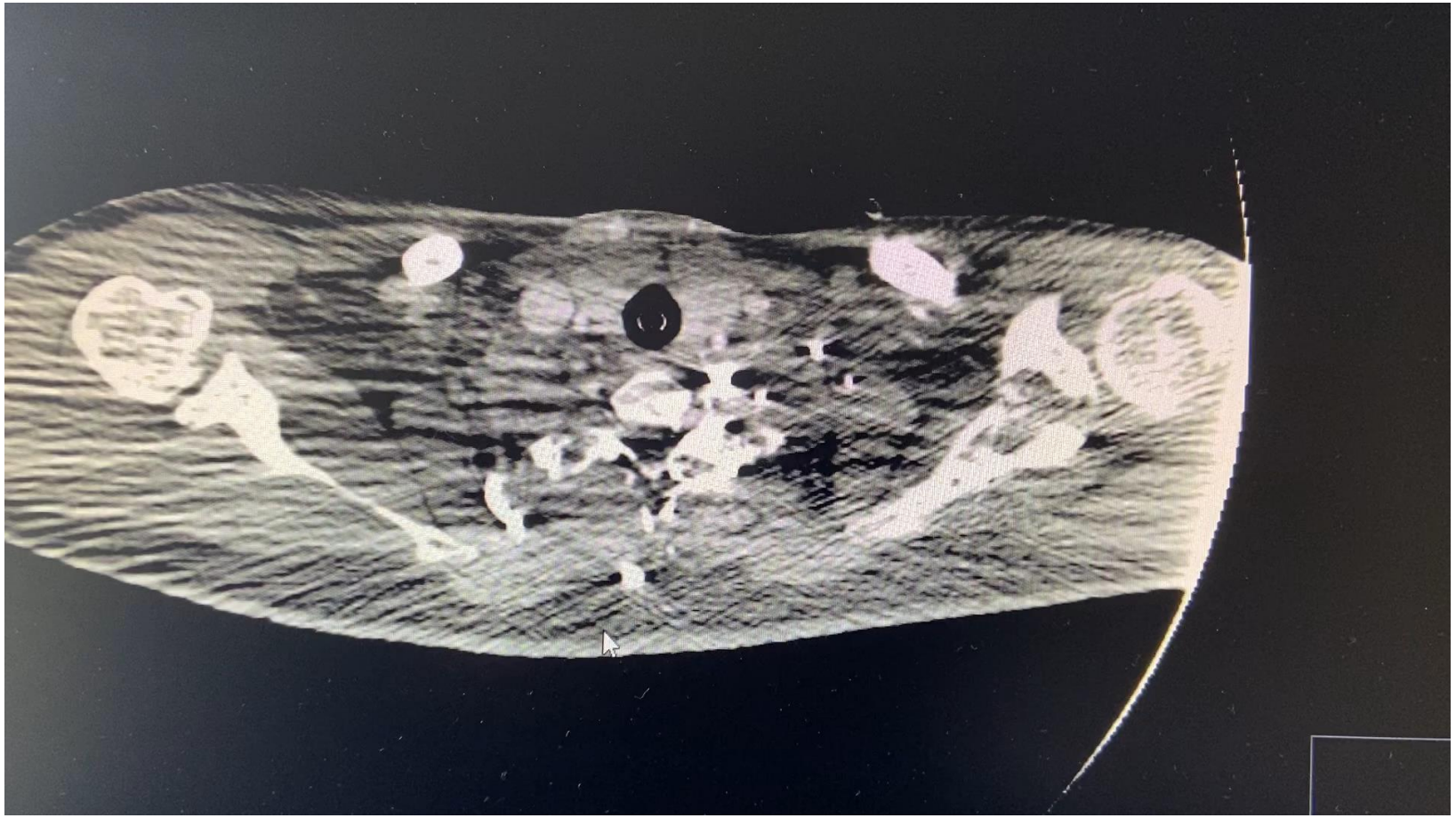
MİKROSKOPİ :
Kesitlerde, böbreğe ait doku parçaları izlendi. Dokunun %80`i korteksten oluşmuştur. Seri kesitlerde en fazla 8 adet glomerül görülmektedir. Glomerüllerden 2 si tamamen sklerotiktir. Diğer 8 glomerülde değişen oranalrada mezengial matriks artışı vardır.
Az sayıda atrofik tüp vardır.
İnterstisyumda hafif şiddette iltihabi mononükleer hücre infiltrasyonu vardır.İki glomerül çevresinde periglomerüler fibrozis görülmektedir.
Damar duvarlarında fibröz intimal kalınlaşma vardır.
Histokimyasal olarak yapılan Kongoı Red boyası ile negatif sonuç elde edildi.
Histokimyasal olarak yapılan PAS ve MT boyaları yukardaki bulguları destekler niteliktedir.
Yapılan immünoflorasan çalışmalarda;
C3 ile glomerül görülmedi. Boyanma izlenmedi.
Ig G ile glomerül görülmedi. Boyanma izlenmedi.
Ig A ile glomerül görülmedi. Boyanma izlenmedi.
Ig M ile glomerül görülmedi. Boyanma izlenmedi.
C4 ile glomerül görülmedi. Boyanma izlenmedi.
C1q ile glomerül görülmedi. Boyanma izlenmedi.
Kappa ile glomerül görülmedi. Boyanma izlenmedi.
Lambda ile glomerül görülmedi. Boyanma izlenmedi.

TANI :
DİFFÜZ GLOMERÜLOSKLEROZ, BÖBREK İĞNE BİYOPSİSİ

ARŞ.GÖR.ŞERİFE SEÇİL BARATALI
Dipl Tescil No 155202

PROF.DR.HÜLYA AKGÜN
Dipl Tescil No 76108

- Hasta hastaneden çıkıp otobüs durağına yürüyerek gidiyor,
- Orada nefes darlığı ve göğüs ağrısı gelişiyor,
- Tekrar hastaneye dönerek acile başvuruyor,
- Acile başvuruda hastanın Genel durumu orta, takipnesi var,
- Birkaç dakika sonra solunumu yüzeyelleşiyor ve hasta entübe ediliyor.
- Sonra kardiyak arrest, CPR ile geri dönüyor
- Kontrastlı BT çekiliyor
- Bu esnada D Dimer sonucu çıkıyor = **190**



ELSEVIER

Thrombosis Research

Volume 120, Issue 2, 2007, Pages 195-200

REGULAR ARTICLE

Diagnostic value of D-dimer in patients with suspected pulmonary embolism: Results from a multicentre outcome study ☆

Florence Parent [a], Sophie Maître [a], Guy Meyer [c], Chantal Raherison [d], Hervé Mal [e], Rémi Lancar [f], Francis Couturaud [g], Dominique Mottier [g], Philippe Girard [b], Gérald Simonneau [a], Christophe Leroyer [g] ✉

Results

Three hundred and fifty two patients were included in 4 centres. Prevalence of PE was 38.6%. PCP was low in 82 (23.3%), intermediate in 176 (50%) and high in 94 (26.7%) patients. Sensitivity of D-dimer was 96.3% (95% CI: 93–99) and negative predictive value reached 94.4% (95% CI: 90–99). Five patients with a confirmed PE had a D-dimer level below 500 ng/ml (two patients with a high PCP). Among 258 patients with low or intermediate PCP, 80 (31%) had a negative D-dimer test result; three of them had a false negative result and the number needed to test was 3.3. Among 94 patients with a high PCP, 9 had a negative D-dimer test result; two of them had a false negative result and the number needed to test was 13.5.

Conclusion

These results confirm that rapid assays used in this study can safely exclude PE in first-line testing only in non-high CP patients.

Risk factors of venous thromboembolism in focal segmental glomerulosclerosis with nephrotic syndrome

Shi-jun Li [1], Yuan-Mao Tu [2], Chang-sheng Zhou [3], Li-Hua Zhang [2], Zhi-hong Liu [2]

Affiliations + expand

PMID: 26220221 DOI: 10.1007/s10157-015-1149-4

Abstract

Background: Venous thromboembolism (VTE) is an important and potentially life-threatening complication in focal segmental glomerulosclerosis (FSGS). The aim of this study was to investigate the prevalence and predisposing risk factors of venous thromboembolism in patients with FSGS with nephrotic syndrome.

Methods: A total of 120 FSGS patients with nephrotic syndrome were enrolled in this study. Venous thromboembolism was confirmed by contrast-enhanced dual-source computed tomography angiography or magnetic resonance venography. Potential clinical and laboratory risk factors for VTE were screened.

Results: Venous thrombosis was demonstrated in 12 (10 %) patients. Venous thrombosis occurred during the first episode of nephrotic syndrome in 3 patients and during a relapse in 9 patients. Eight patients had a pulmonary embolism, four had a renal vein thrombosis, three had a lower limb deep vein thrombosis, one had a cerebral sinovenous thrombosis, and one had a portal vein thrombosis. The positive predictive value for the D-dimer level was 22.4 % in the patients with FSGS, and the negative predictive value for the D-dimer level was 100 %. Of the screened risk factors, higher hematocrit and relapse of nephrotic syndrome were risk factors for VTE. Other risk factors, such as proteinuria, hypoalbuminemia, platelet count, fibrinogen level, and antithrombin III level, were not risk factors for VTE in patients with FSGS.

Reperfüzyon tedavisi

- PE de trombolitik tedavi ile pulmoner obstrüksiyon, pulmoner arter basıncı ve pulmoner vasküler rezisdansta UFH ne göre çok daha hızlı düzelme sağlanır.
- İlk 48 saatte en yüksek etkiyi gösterir ama 6-14 güne kadar etkilidir.
- Metaanalizde **%9.9** ciddi kanama yaptığı ve **%1.7** intrakranial kanama yaptığı gösterilmiştir.
- Yüksek riskli PE hastalarında Kanıt 1B olarak önerilmektedir.
- 100 mgr tPA 2 saatte öneriliyor (alteplaz)

Molecule	Regimen	Contraindications to fibrinolysis
rtPA	100 mg over 2 h	**Absolute**
	0.6 mg/kg over 15 min (maximum dose 50 mg)[a]	History of haemorrhagic stroke or stroke of unknown origin
Streptokinase	250 000 IU as a loading dose over 30 min, followed by 100 000 IU/h over 12–24 h	Ischaemic stroke in previous 6 months
	Accelerated regimen: 1.5 million IU over 2 h	Central nervous system neoplasm
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h over 12–24 h	Major trauma, surgery, or head injury in previous 3 weeks
	Accelerated regimen: 3 million IU over 2 h	Bleeding diathesis
		Active bleeding
		Relative
		Transient ischaemic attack in previous 6 months
		Oral anticoagulation
		Pregnancy or first post-partum week
		Non-compressible puncture sites
		Traumatic resuscitation
		Refractory hypertension (systolic BP >180 mmHg)
		Advanced liver disease
		Infective endocarditis
		Active peptic ulcer

BP = blood pressure; IU = international units; rtPA, recombinant tissue-type plasminogen activator.

[a]This is the accelerated regimen for rtPA in pulmonary embolism; it is not officially approved, but it is sometimes used in extreme haemodynamic instability such as cardiac arrest.

KONTRENDİKASYONLAR

Web Table 4 Contraindications to thrombolytic therapy (adapted from ref. 312)

Absolute contraindications:[a]

- Haemorrhagic stroke or stroke of unknown origin at any time
- Ischaemic stroke in the preceding 6 months
- Central nervous system damage or neoplasms
- Recent major trauma/surgery/head injury in the preceding 3 weeks
- Gastrointestinal bleeding within the last month
- Known bleeding risk

Relative contraindications

- Transient ischaemic attack in the preceding 6 months
- Oral anticoagulant therapy
- Pregnancy, or within one week postpartum
- Non-compressible puncture site
- Traumatic resuscitation
- Refractory hypertension (systolic blood pressure >180 mm Hg)
- Advanced liver disease
- Infective endocarditis
- Active peptic ulcer

ESC 2014, European Heart Journal

Absolute contraindications:[a]

[a]Absolute contraindications to thrombolysis might become relative in a patient with immediately life-threatening high-risk PE.

- Hastaya hangi dozda trombolitik verilir?
- A- 100 mgr TPA 2 saatte
- B- 50 mgr TPA 2 saatte
- C- 50 mgr TPA 15 dk da
- D- 100 mgr TPA 30 dk da

- Hastaya hangi dozda trombolitik verilir?
- A- 100 mgr TPA 2 saatte
- B- 50 mgr TPA 2 saatte
- C- 50 mgr TPA 15 dk da
- D- 100 mgr TPA 30 dk da

- 100 mgr TPA 2 saatte veriliyor
- Hb düşüşü oluyor
- 2 ünite ES veriliyor
- Tekrar arrest ve exitus oluyor

Hangi trombolitik dozu?

Efficacy and Safety of Low Dose Recombinant Tissue-Type Plasminogen Activator for the Treatment of Acute Pulmonary Thromboembolism

A Randomized, Multicenter, Controlled Trial

Chen Wang, MD, PhD, FCCP; Zhenguo Zhai, MD, PhD; Yuanhua Yang, MD; Qi Wu, MD,

Background: Optimal dosing of the recombinant tissue-type plasminogen activator (rt-PA) is important in treating pulmonary thromboembolism (PTE). The aim of this study was to compare the efficacy and safety of a 50 mg/2 h rt-PA regimen with a 100 mg/2 h rt-PA regimen in patients with acute PTE.

Methods: A prospective, randomized, multicenter trial was conducted in which 118 patients with acute PTE and either hemodynamic instability or massive pulmonary artery obstruction were randomly assigned to receive a treatment regiment of either rt-PA at 50 mg/2 h (n = 65) or 100 mg/2 h (n = 53). The efficacy was determined by observing the improvements of right ventricular dysfunctions (RVDs) on echocardiograms, lung perfusion defects on ventilation perfusion lung scans, and pulmonary artery obstructions on CT angiograms. The adverse events, including death, bleeding, and PTE recurrence, were also evaluated.

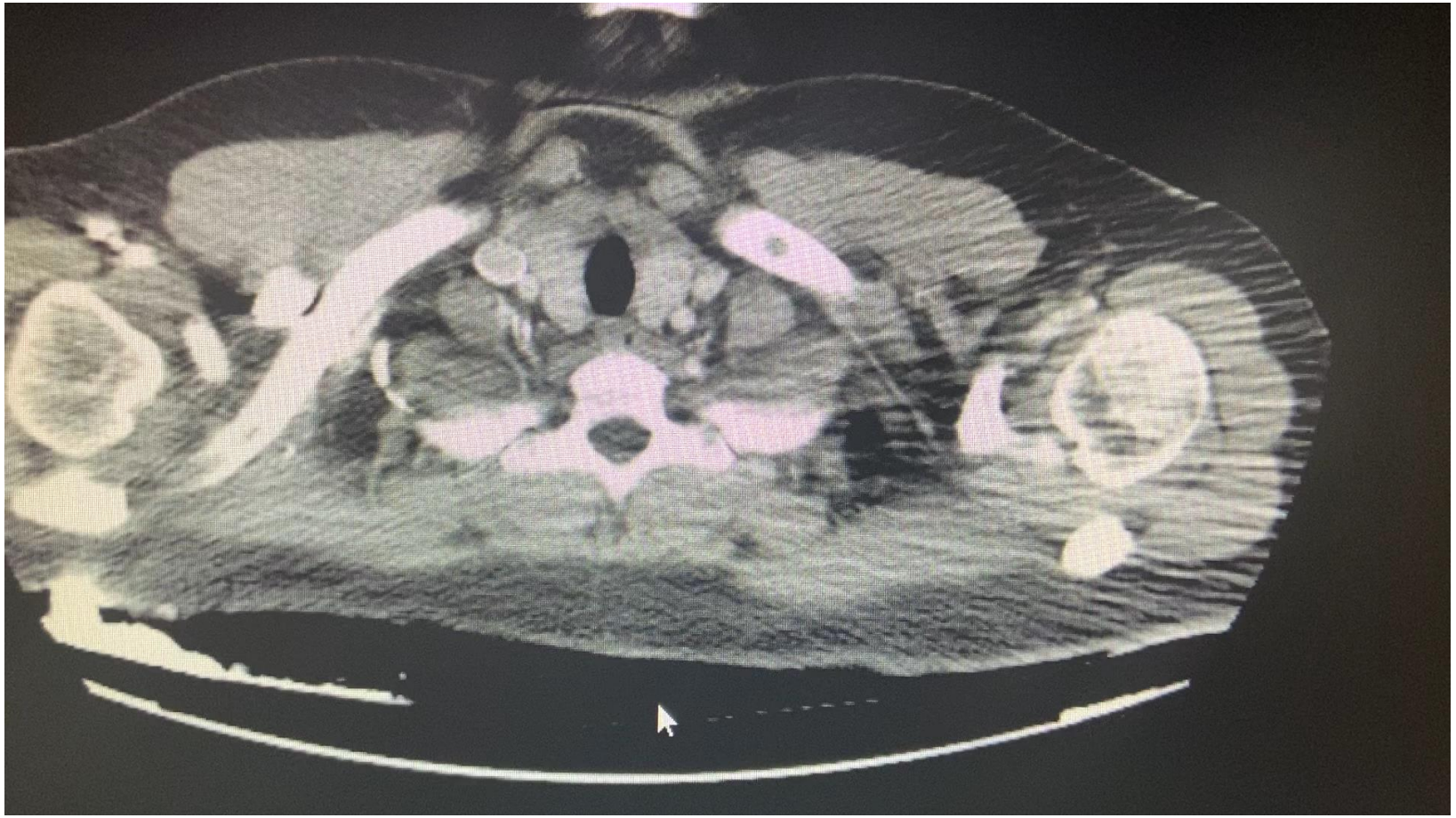
Results: Progressive improvements in RVDs, lung perfusion defects, and pulmonary artery obstructions were found to be similarly significant in both treatment groups. This is true for patients with either hemodynamic instability or massive pulmonary artery obstruction. Three (6%) patients in the rt-PA 100 mg/2 h group and one (2%) in the rt-PA 50 mg/2 h group died as the result of either PTE or bleeding. Importantly, the 50 mg/2 h rt-PA regimen resulted in less bleeding tendency than the 100 mg/2 h regimen (3% vs 10%), especially in patients with a body weight < 65 kg (14.8% vs 41.2%, $P = .049$). No fatal recurrent PTE was found in either group.

Conclusions: Compared with the 100 mg/2 h regimen, the 50 mg/2 h rt-PA regimen exhibits similar efficacy and perhaps better safety in patients with acute PTE. These findings support the notion that optimizing rt-PA dosing is worthwhile when treating patients with PTE.

Trial registration: clinicaltrials.gov; Identifier: NCT00781378

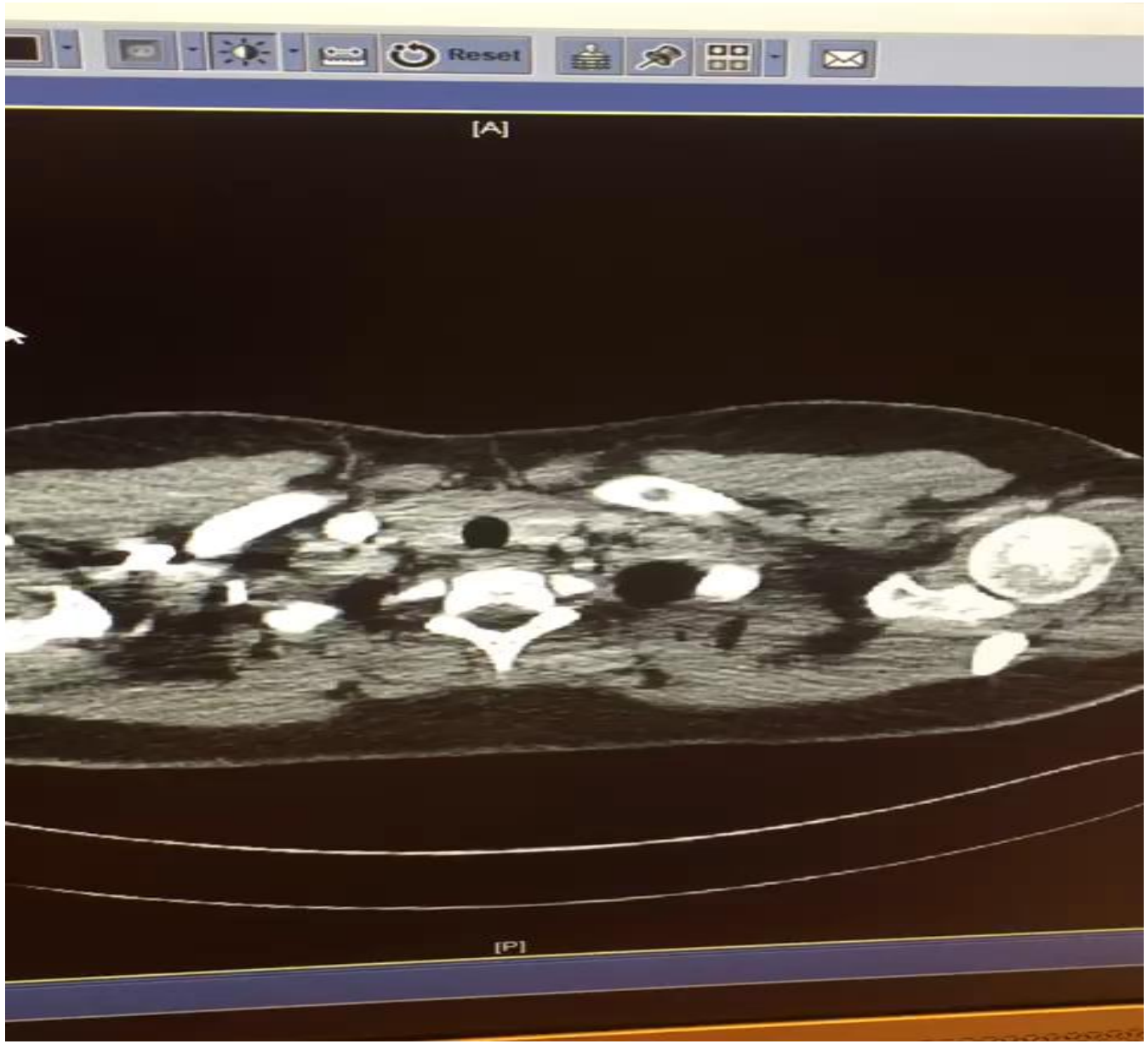
Olgu 2

- 24 y
- Erkek hasta
- Hemoptizi, göğüs ağrısı ve ateş şikayeti ile geliyor,
- D Dimer 3860
- Troponin negatif



- Antibiyotik veriliyor,
- Poliklinik kontrol öneriliyor

- Sabah tekrar polikliniğe geliyor,
- Solunum sayısı: 40
- Nabız:122
- KB:140/70
- SPO2:85
- Tekrar BT


Reset
[A]
[P]

- YBÜ yatış,
- Solunum sayısı=48
- Nazal kanülden 4 lt/dk dan oksijen alırken spo2:90-91, NIMV ye alınıyor= 40 soluyor
- Troponin +
- Yatak başı EKO yapılamıyor ancak BT de RV/LV oranı 1.

- 1- Hasta hangi grupta?

sPESI

- 1- 80 Yaş üstü
- 2- 90 saturasyon altı
- 3- 100 sistolik kan basıncı altı
- 4- 110 nabız üstü
- 5- Kronik kardiyopulmoner hastalık
- 6- Malignite
- HERBİRİ 1 PUAN, HERHANGİ BİRİNİN OLMASI YETERLİ

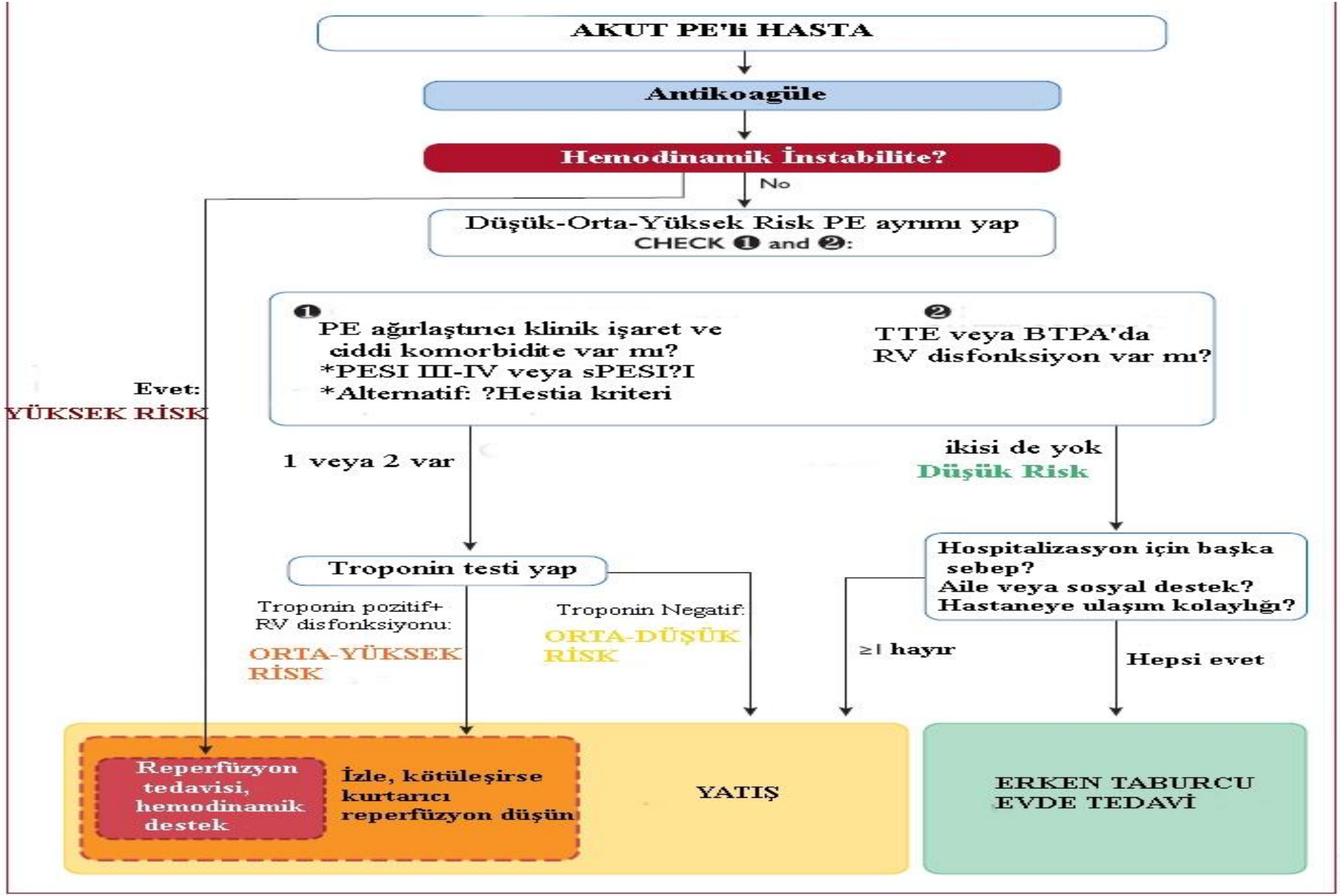
PESI Class III to V indicates moderate to very high 30-day mortality risk; sPESI ≥1 point(s) indicate high 30-day mortality risk.

Jimenez D, et al. Arch Intern Med 2010;170:1383-9.

2019

Early mortality risk		Indicators of risk			
		Haemodynamic instability[a]	Clinical parameters of PE severity and/or comorbidity: PESI class III–V or sPESI ≥I	RV dysfunction on TTE or CTPA[b]	Elevated cardiac troponin levels[c]
High		+	(+)[d]	+	(+)
Intermediate	Intermediate–high	-	+[e]	+	+
	Intermediate–low	-	+[e]	One (or none) positive	
Low		-	-	-	Assesment optional; if assessed, negative

- 1- Hasta hangi grupta?
- intermediate high
- 2- Hastaya tedavi planı ne olmalı?


AKUT PE'li HASTA
Antikoagüle
Hemodinamik İnstabilite?
No
Düşük-Orta-Yüksek Risk PE ayrımı yap
CHECK ❶ and ❷:
❶
PE ağırlaştırıcı klinik işaret ve ciddi komorbidite var mı?
*PESI III-IV veya sPESI?I
*Alternatif: ?Hestia kriteri
❷
TTE veya BTPA'da RV disfonksiyon var mı?
Evet:
YÜKSEK RİSK
1 veya 2 var
ikisi de yok
Düşük Risk
Troponin testi yap
Troponin pozitif+ RV disfonksiyonu:
ORTA-YÜKSEK RİSK
Troponin Negatif:
ORTA-DÜŞÜK RİSK
Hospitalizasyon için başka sebep?
Aile veya sosyal destek?
Hastaneye ulaşım kolaylığı?
≥I hayır
Hepsi evet
Reperfüzyon tedavisi, hemodinamik destek
İzle, kötüleşirse kurtarıcı reperfüzyon düşün
YATIŞ
ERKEN TABURCU
EVDE TEDAVİ
©ESC 2019

- 2- Hastaya tedavi planı ne olmalı?
- Rehbere göre antikoagülan

- Takipnesi kırılmaması nedeniyle trombolitik veriliyor,
- 100 mgr 2 saatte,
- Genel durumu 1 gün sonra düzeliyor,
- Solunum sayısı O2 le 20 lere düşüyor,
- Takip eden günlerde servis devri ve tab ediliyor

Intermediate high risk hastalarda trombolitik verelim mi?

- Evet
- Hayır

Moderate Pulmonary Embolism Treated With Thrombolysis (from the "MOPETT" Trial)

Mohsen Sharifi, MD[a,b,*], Curt Bay, PhD[b], Laura Skrocki, DO[a], Farnoosh Rahimi, MD[a], and Mahshid Mehdipour, DMD[a,b], "MOPETT" Investigators

The role of low-dose thrombolysis in the reduction of pulmonary artery pressure in moderate pulmonary embolism (PE) has not been investigated. Because the lungs are very sensitive to thrombolysis, we postulated that effective and safe thrombolysis might be achieved by a lower dose of tissue plasminogen activator. The purpose of the present study was to evaluate the role of this "safe dose" thrombolysis in the reduction of pulmonary artery pressure in moderate PE. During a 22-month period, 121 patients with moderate PE were randomized to receive a "safe dose" of tissue plasminogen activator plus anticoagulation (thrombolysis group [TG], n = 61 patients) or anticoagulation alone (control group [CG], n = 60). The primary end points consisted of pulmonary hypertension and the composite end point of pulmonary hypertension and recurrent PE at 28 months. Pulmonary hypertension and the composite end point developed in 9 of 58 patients (16%) in the TG and 32 of 56 patients (57%) in the CG ($p < 0.001$) and 9 of 58 patients (16%) in the TG and 35 of 56 patients (63%) in the CG ($p < 0.001$), respectively. The secondary end points were total mortality, the duration of hospital stay, bleeding at the index hospitalization, recurrent PE, and the combination of mortality and recurrent PE. The duration of hospitalization was 2.2 ± 0.5 days in the TG and 4.9 ± 0.8 days in the CG ($p < 0.001$). The combination of death plus recurrent PE was 1 (1.6%) in TG and 6 (10%) in the CG ($p = 0.0489$). No bleeding occurred in any group, and despite a positive trend in favor of a "safe dose" thrombolysis, no significant difference was noted in the rate of individual outcomes of death and recurrent PE when assessed independently. In conclusion, the results from the present prospective randomized trial suggests that "safe dose" thrombolysis is safe and effective in the treatment of moderate PE, with a significant immediate reduction in the pulmonary artery pressure that was maintained at 28 months. (Am J Cardiol 2013;111:273—277)

INTERMEDIATE RİSKTE (SUBMASİF) TROMBOLİTİK

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Fibrinolysis for Patients with Intermediate-Risk Pulmonary Embolism

Guy Meyer, M.D., Eric Vicaut, M.D., Thierry Danays, M.D., Giancarlo Agnelli, M.D.,

N Engl J Med 2014;370:1402-11.

- Randomize, çift kör, hipotansiyonu olmayan intermediate yüksek risk hastalarda **tenekteplaz+ heparin** ile **plasebo + heparin** tedavisi karşılaştırılıyor.
- 1. haftada ölüm, hemodinamik bozukluk ve kanama oranları karşılaştırılıyor.
- 1005 hasta

Table 3. Efficacy Outcomes.*

Outcome	Tenecteplase (N = 506)	Placebo (N = 499)	Odds Ratio (95% CI)	P Value
Primary outcome — no. (%)	13 (2.6)	28 (5.6)	0.44 (0.23–0.87)	0.02
Death from any cause	6 (1.2)	9 (1.8)	0.65 (0.23–1.85)	0.42
Hemodynamic decompensation	8 (1.6)	25 (5.0)	0.30 (0.14–0.68)	0.002
Time between randomization and primary efficacy outcome — days	1.54±1.71	1.79±1.60		
Recurrent pulmonary embolism between randomization and day 7 — no. (%)	1 (0.2)	5 (1.0)	0.20 (0.02–1.68)	0.12
Fatal	0	3 (0.6)		
Nonfatal	1 (0.2)	2 (0.4)		

Table 4. Safety Outcomes in the Intention-to-Treat Population.*

Outcome	Tenecteplase (N=506)	Placebo (N=499)	Odds Ratio (95% CI)	P Value
	no. (%)			
Bleeding between randomization and day 7				
Major extracranial bleeding	32 (6.3)	6 (1.2)	5.55 (2.3–13.39)	<0.001
Minor bleeding	165 (32.6)	43 (8.6)		
Major bleeding†	58 (11.5)	12 (2.4)		
Stroke between randomization and day 7	12 (2.4)	1 (0.2)	12.10 (1.57–93.39)	0.003
Ischemic stroke	2 (0.4)	0		
Hemorrhagic stroke‡	10 (2.0)	1 (0.2)		

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Impact of Thrombolytic Therapy on the Long-Term Outcome of Intermediate-Risk Pulmonary Embolism

Stavros V. Konstantinides, MD, PhD,[a,b] Eric Vicaut, MD, PhD,[c] Thierry Danays, MD,[d] Cecilia Becattini, MD,[e]

- 709 hasta
- 1 ay ve 24 aylık sonuçlar karşılaştırılıyor

TABLE 3 Overall and Cause-Specific 30-Day and Long-Term Mortality

	Tenecteplase (N = 359)	Placebo (N = 350)	p Value
Death from any cause between randomization and day 30	8 (2.2)	10 (2.9)	0.595
Hemodynamic collapse	1 (0.3)	1 (0.3)	
Stroke (ischemic or hemorrhagic)	4 (1.1)	0 (0.0)	
Recurrent pulmonary embolism	0 (0.0)	2 (0.6)	
Respiratory failure	0 (0.0)	2 (0.6)	
Extracranial bleeding	1 (0.3)	0 (0.0)	
Sudden unexplained death	0 (0.0)	2 (0.6)	
Other	2 (0.6)	3 (0.9)	
Death from any cause between day 30 and long-term follow-up	65 (18.1)	53 (15.1)	
Stroke	1 (0.3)	2 (0.6)	
Acute myocardial infarction	0 (0.0)	1 (0.3)	
Respiratory failure	2 (0.6)	1 (0.3)	
Sudden unexplained death	2 (0.6)	0 (0.0)	
Cancer	8 (2.2)	9 (2.6)	
Bleeding	0 (0.0)	1 (0.3)	
Chronic heart failure	1 (0.3)	0 (0.0)	
Other	19 (5.3)	4 (1.1)	
Unknown cause	32 (8.9)	35 (10.0)	
Death from any cause between randomization and long-term follow-up	73 (20.3)	63 (18.0)	0.430

TABLE 4 Findings in Patients With Echocardiographic Long-Term Follow-Up

	Tenecteplase (N = 144)	Placebo (N = 146)	p Value
Right ventricular end-diastolic diameter >30 mm	34 (23.6)	22 (15.1)	0.058
Missing data	12 (8.3)	11 (7.5)	
Right/left ventricular end-diastolic diameter >0.9	34 (23.6)	22 (15.1)	0.834
Missing data	12 (8.3)	11 (7.5)	
Hypokinesia of the right ventricular free wall (any view)	6 (4.2)	5 (3.4)	0.740
Missing data	4 (2.8)	4 (2.7)	
Tricuspid annulus plane systolic excursion reduced	14 (9.7)	7 (4.8)	0.107
Mean, mm Hg	23.6 ± 4.8	23.9 ± 3.6	
Median, mm Hg	24.0 (20.0–27.0)	24.0 (21.0–26.0)	0.551
Missing data,	19 (13.2)	18 (12.3)	
Tricuspid systolic velocity >2.6 m/s	22 (15.3)	27 (18.5)	0.412
Missing data	11 (7.6)	14 (9.6)	
Systolic pulmonary artery pressure, mm Hg			
Mean	31.6 ± 12.3	30.7 ± 10.2	0.527
Median	30.0 (24.0–35.0)	30.0 (25.0–35.0)	
Missing data	33 (22.9)	39 (26.7)	

CONCLUSIONS

In a large, prospective randomized controlled trial of patients with intermediate- to high-risk PE, thrombolytic treatment with tenecteplase did not affect long-term mortality rates, and it did not appear to reduce residual dyspnea, functional limitation, or persisting RV dysfunction, which were mostly mild in both treatment arms. These results suggest that

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PMCID: PMC10156731
PMID: 37137999

Mortality and bleeding associated with the management of sub-massive pulmonary embolism: a systematic review and Bayesian network meta-analysis

Don Mathew,[✉1,5] Jay Kim,[1] Bhanu Prasad Kosuru,[1] Deepthi Devagudi,[2] Akil Sherif,[3] Utsav Shrestha,[4] and Prabhjot Bedi[1]

- 14 RKÇ, 2132 hasta
- tPA grubu antikoagülan grubuna göre mortalitede üstün
- Major kanamada artış yok, minör kanamada artış var
- Rekürren emboli riski daha düşük
- Ancak daha çok klinik çalışmaya ihtiyaç var

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PMCID: PMC9197594

PMID: 34560806

Reduced-Dose Intravenous Thrombolysis for Acute Intermediate–High-risk Pulmonary Embolism: Rationale and Design of the Pulmonary Embolism International THrOmbolysis (PEITHO)-3 trial

Olivier Sanchez,[1,2,3,4] Anaïs Charles-Nelson,[5,6] Walter Ageno,[7] Stefano Barco,[8,9] Harald Binder,[10] Gilles Chatellier,[3,5,6] Daniel Duerschmied,[11] Klaus Empen,[12] Melanie Ferreira,[13] Philippe Girard,[4,14] Menno V. Huisman,[15] David Jiménez,[16] Sandrine Katsahian,[3,5,6,17] Matija Kozak,[18] Mareike Lankeit,[8,19,20] Nicolas Meneveau,[4,21,22] Piotr Pruszczyk,[23]

Intermediate–high-risk pulmonary embolism (PE) is characterized by right ventricular (RV) dysfunction and elevated circulating cardiac troponin levels despite apparent hemodynamic stability at presentation. In these patients, full-dose systemic thrombolysis reduced the risk of hemodynamic decompensation or death but increased the risk of life-threatening bleeding. Reduced-dose thrombolysis may be capable of improving safety while maintaining reperfusion efficacy. The Pulmonary Embolism International THrOmbolysis (PEITHO)-3 study (ClinicalTrials.gov Identifier: NCT04430569) is a randomized, placebo-controlled, double-blind, multicenter, multinational trial with long-term follow-up. We will compare the efficacy and safety of a reduced-dose alteplase regimen with standard heparin anticoagulation. Patients with intermediate–high-risk PE will also fulfill at least one clinical criterion of severity: systolic blood pressure ≤110 mm Hg, respiratory rate >20 breaths/min, or history of heart failure. The primary efficacy outcome is the composite of all-cause death, hemodynamic decompensation, or PE recurrence within 30 days of randomization. Key secondary outcomes, to be included in hierarchical analysis, are fatal or GUSTO severe or life-threatening bleeding; net clinical benefit (primary efficacy outcome plus severe or life-threatening bleeding); and all-cause death, all within 30 days. All outcomes will be adjudicated by an independent committee. Further outcomes include PE-related death, hemodynamic decompensation, or stroke within 30 days; dyspnea, functional limitation, or RV dysfunction at 6 months and 2 years; and utilization of health care resources within 30 days and 2 years. The study is planned to enroll 650 patients. The results are expected to have a major impact on risk-adjusted treatment of acute PE and inform guideline recommendations.

0.6 mg/kg Alteplaz: 15 dk da

Review > World J Radiol. 2022 Aug 28;14(8):286-292. doi: 10.4329/wjr.v14.i8.286.

Progress in interventional radiology treatment of pulmonary embolism: A brief review

Alessandro Posa [1], Pierluigi Barbieri [1], Giulia Mazza [1], Alessandro Tanzilli [2], Roberto Iezzi [1], Riccardo Manfredi [1], Cesare Colosimo [1]

Affiliations + expand

PMID: 36160834 PMCID: PMC9453319 DOI: 10.4329/wjr.v14.i8.286

Free PMC article

Abstract

Pulmonary embolism represents a common life-threatening condition. Prompt identification and treatment of this pathological condition are mandatory. In cases of massive pulmonary embolism and hemodynamic instability or right heart failure, interventional radiology treatment for pulmonary embolism is emerging as an alternative to medical treatment (systemic thrombolysis) and surgical treatment. Interventional radiology techniques include percutaneous endovascular catheter directed therapies as selective thrombolysis and thrombus aspiration, which can prove useful in cases of failure or infeasibility of medical and surgical approaches.


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