7ο ΕΚΠΑΙΔΕΥΤΙΚΟ ΣΥΜΠΟΣΙΟ
Α' ΚΛΙΝΙΚΗΣ ΕΝΤΑΤΙΚΗΣ ΘΕΡΑΠΕΙΑΣ
ΙΑΤΡΙΚΗΣ ΣΧΟΛΗΣ ΕΚΠΑ

Ο ΑΝΑΠΝΕΥΣΤΙΚΟΣ ΑΣΘΕΝΗΣ ΣΤΟ ΤΕΠ

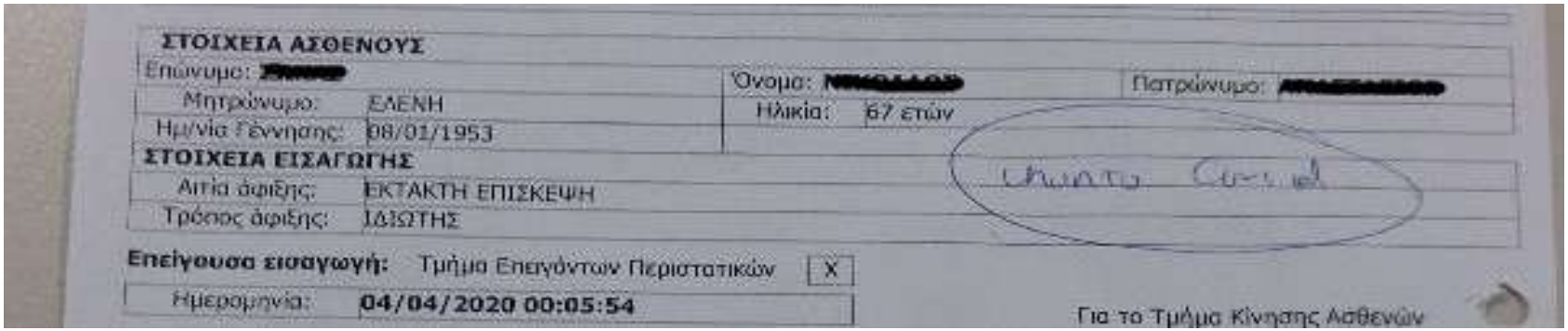
Η συμβολή του καρδιολόγου στα αναπνευστικά επείγοντα

Ιωάννης Α. Λακουμέντας

Διεθυντής Ε.Σ.Υ.

Β' Καρδιολογική Κλινική – Γ.Ν.Α. <Ο Ευαγγελισμός>

Ασθενής 67 ετών διεκομίσθη για αναπνευστική δυσχέρεια, βήχα από ωρών. Η αρχική διαλογή για Covid-19 θεώρησε το περιστατικό ως ύποπτο κρούσμα της πανδημίας και ο ασθενής οδηγήθηκε στο εξωτερικό πνευμονολογικό ιατρείο για εκτίμηση-αντιμετώπιση.



ΣΤΟΙΧΕΙΑ ΑΣΘΕΝΟΥΣ

Επώνυμο: [redacted]

Όνομα: [redacted]

Πατρώνυμο: [redacted]

Μητρώνυμο: ΕΛΕΝΗ

Ημ/νία Γέννησης: 08/01/1953

Ηλικία: 67 ετών

ΣΤΟΙΧΕΙΑ ΕΙΣΑΓΩΓΗΣ

Αιτία άφιξης: ΕΚΤΑΚΤΗ ΕΠΙΣΚΕΨΗ

Τρόπος άφιξης: ΙΔΙΩΤΗΣ

Υπόπτο Covid

Επείγουσα εισαγωγή: Τμήμα Επαγόντων Περιστατικών [X]

Ημερομηνία: **04/04/2020 00:05:54**

Για το Τμήμα Κίνησης Ασθενών

- Η πνευμονολογική εκτίμηση ανέφερε ότι επρόκειτο για βαρύ καπνιστή με πιθανή ΧΑΠ, που εργαζόταν στα καράβια και δεν είχε υποβληθεί ποτέ σε σπειρομέτρηση.

Αντικειμενική εξέταση

- Ασθενής με δύσπνοια, ψυχρά άκρα, κάθιδρος, συγχυτικός

Αναπνοές 26/min,

Ομότιμη έκπτυξη ημιθωρακίων,

Μειωμένο αναπνευστικό ψιθύρισμα άμφω,

SpO2 88%

Σφύξεις 141 bpm,

Αρτηριακή πίεση 85/50 mmHg,

Βύθιοι, ταχείς, ρυθμικοί καρδιακοί τόνοι,

Χωρίς σημεία DVT στα κάτω άκρα

Διαφορική διάγνωση οξείας δύσπνοιας για τον καρδιολόγο

- Οξεία καρδιακή ανεπάρκεια (πρωτοεμφανιζόμενη ή οξεία απορρύθμιση χρόνιας καρδιακής ανεπάρκειας)
- Άλλη αιτία (π.χ. πνευμονική λοίμωξη, σοβαρή αναιμία, οξεία νεφρική ανεπάρκεια)

- **Ποιες είναι οι 4 συχνότερες αιτίες δύσπνοιας σε ασθενείς στο ΤΕΠ;**

1. Οξεία καρδιακή ανεπάρκεια, οξύ στεφανιαίο σύνδρομο, αρρυθμία, περικαρδιακός επιπωματισμός
2. Πνευμονία, τραύμα, αλλεργία, πνιγμός
3. Χρόνια αποφρακτική πνευμονοπάθεια, οξεία καρδιακή ανεπάρκεια, οξεία επίταση άσθματος, πνευμονία

Causes of Shortness of Breath in the Acute Patient: A National Study

Zachariah E. Hale, Astha Singhal, MPH, PhD, and Renee Y. Hsia, MD, MSc

ABSTRACT

Background: Dyspnea is a common and ambiguous complaint that results in 3.4 million emergency department (ED) visits annually. While learners may encounter lists of differential diagnoses to consider while in training, often these lists are not empirically based. We sought to establish an evidence-based differential diagnosis for dyspnea and to determine whether normal vital signs can rule out a life-threatening diagnosis.

Methods: We analyzed data from the National Hospital Ambulatory Medical Care Survey from 2005 to 2014 for ED visits with a chief complaint of dyspnea and tallied the principal discharge diagnosis. We included 10,170 sampled ED visits by adults with a chief complaint of dyspnea, representing nearly 42 million visits nationally. We then calculated the most common principal discharge diagnoses by age group and the frequency of abnormal respiratory vital signs in cases with life-threatening diagnoses.

Results: The most represented age group was 45 to 64 years (31.6%). Most visits were discharged directly from the ED (57.5%), while 8.1% required admission to an intensive care unit (ICU). The most common diagnosis in patients aged 18 to 44 was acute asthma exacerbation (14.8%). Obstructive chronic bronchitis was the most common specified diagnosis in both patients ages 45 to 64 (11.1%) and patients ages 65 to 79 (12.4%), while congestive heart failure was the most common for patients ages 80 and over (15.9%). Respiratory vital signs were frequently normal in the 44.6% of visits that resulted in a potentially life-threatening diagnosis but corresponded to increased ICU utilization when abnormal.

Conclusions: For patients with dyspnea, the high utilization of ICUs highlights the importance of an accurate differential. The frequencies established here can be used as pretest probabilities in Bayesian analysis to improve the accuracy of differential diagnosis lists.

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Supervising Editor: D. Mark Courtney, MD.
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ACADEMIC EMERGENCY MEDICINE 2018;25:1227–1234.

Frequency of Life-threatening Diagnoses Most Often Associated With Dyspnea

Diagnosis	No.	Weighted %	95% CI
Asthma	1,402	12.9	11.9–13.8
COPD	1,160	11.9	10.9–12.9
Acute heart failure	848	8.0	7.3–8.7
Pneumonia or other infection	705	7.1	6.3–7.9
Injury (trauma or poisoning)	227	1.9	1.6–2.2
Arrhythmia	165	1.4	1.1–1.7
Acute coronary syndrome	96	0.9	0.7–1.2
Pulmonary embolism	59	0.5	0.3–0.6
Angioedema and anaphylaxis	10	–*	–*
Pericardial tamponade	3	–*	–*
Total of the above	4,675	44.6	

COPD = chronic obstructive pulmonary disease.
*Percentages cannot accurately be calculated for categories with less than 30 records.

- **Αντιστοίχιση ηλικιών ομάδας 1 με συχνότερα αίτια δύσπνοιας στο ΤΕΠ ομάδας 2.**

Ομάδα 1

1. Ηλικία 18-44 ετών
2. Ηλικία 45-64 ετών
3. Ηλικία ≥65 ετών

Ομάδα 2

1. Οξεία καρδιακή ανεπάρκεια
2. Οξεία επίταση άσθματος
3. Αποφρακτική χρόνια βρογχίτις

Top 10 diagnoses by age group

Age, years	Diagnosis Category*	No.	Weighted %	95% CI
18–44	Total	2,688	100	
	Other asthma with acute exacerbation	427	14.8	12.9–16.7
	Other and unspecified lower respiratory disease	359	14.3	12.5–16.1
	Other asthma without status asthmaticus or exacerbation	358	12.7	11.0–14.4
	Nonspecific chest pain	152	5.6	4.6–6.7
	Other chronic pulmonary disease	127	5.0	3.5–6.6
	Pneumonia; organism unspecified	104	4.3	2.6–5.9
	Acute bronchitis	104	4.2	2.9–5.5
	Anxiety disorders	94	3.6	2.6–4.5
	Acute URI of multiple or unspecified sites	71	2.5	1.8–3.2
	Painful respiration	54	2.9	1.8–4.1
45–64	Total	3,143	100	
	Other and unspecified lower respiratory disease	542	17.6	15.8–19.4
	Obstructive chronic bronchitis	323	11.1	9.3–12.8
	Other asthma with acute exacerbation	237	7.2	5.9–8.5
	CHF	210	6.6	5.5–7.7
	Pneumonia; organism unspecified	195	6.2	5.2–7.2
	Other asthma without status asthmaticus or exacerbation	163	5.1	4.1–6.1
	Nonspecific chest pain	150	5.1	4.0–6.2
	Chronic airway obstruction; NOS	129	4.9	3.8–6.1
	Other chronic pulmonary disease	93	2.3	1.8–2.9
	Acute bronchitis	83	2.4	1.7–3.0
65–79	Total	2,434	100	
	Other and unspecified lower respiratory disease	483	22.4	19.7–25.1
	Obstructive chronic bronchitis	318	12.4	10.6–14.3
	CHF	276	10.8	9.2–12.3
	Pneumonia; organism unspecified	193	8.1	6.7–9.6
	Chronic airway obstruction; NOS	161	6.7	4.9–8.5
	Nonspecific chest pain	78	2.8	2.0–3.7
	Other asthma without status asthmaticus or exacerbation	49	1.5	1.0–2.0
	Other asthma with acute exacerbation	49	2.1	1.4–2.8
	Other chronic pulmonary disease	46	1.7	1.0–2.3
	Pleurisy; pleural effusion	41	1.9	1.2–2.6
80+	Total	1,675	100	
	Other and unspecified lower respiratory disease	330	20.9	18.3–23.5
	CHF	284	15.9	13.6–18.2
	Pneumonia; organism unspecified	178	10.2	8.4–12.1
	Obstructive chronic bronchitis	100	6.0	4.5–7.4
	Chronic airway obstruction; NOS	81	5.2	3.5–6.8
	Atrial fibrillation	42	2.2	1.3–3.0
	Nonspecific chest pain	42	3.0	1.6–4.3
	Urinary tract infection; site not specified	31	2.3	1.3–3.4
	Other chronic pulmonary disease	25	–†	–†
	Respiratory failure	25	–†	–†

CHF = congestive heart failure; NOS = not otherwise specified; URI = upper respiratory infection.
*These categories are mutually exclusive, and their frequencies may be directly summed to create more general categories as needed.
†Percentages cannot accurately be calculated for categories with fewer than 30 records.

Στην κλινική αξιολόγηση – διαφορική διάγνωση θα βοηθήσει περισσότερο

1. Ιστορικό - Συμπτώματα - Φυσική εξέταση
2. Ηλεκτροκαρδιογράφημα
3. Ακτινογραφία θώρακος
4. Αιματολογικός – Βιοχημικός έλεγχος – Νατριουρητικά πεπτίδια
5. Υπερηχοκαρδιογράφημα
6. US πνευμόνων
7. Όλα τα παραπάνω

ΚΛΙΝΙΚΗ ΑΠΟΦΑΣΗ ΑΠΟ ΣΥΝΑΞΙΟΛΟΓΗΣΗ ΟΛΩΝ ΤΩΝ ΕΥΡΗΜΑΤΩΝ

(Μικρή ευαισθησία και ειδικότητα κάθε μεμονωμένου συμπτώματος ή σημείου οξείας καρδιακής ανεπάρκειας)

Breidthardt T, Moreno-Weidmann Z, Uthoff H, Sabti Z, Appeli S, Puelacher C, Stallone F, Twerenbold R, Wildi K, Kozhuharov N, Wussler D, Flores D, Shrestha S, Badertscher P, Boeddinghaus J, Nestelberger T, Gimenez MR, Staub D, Aschwanden M, Lohrmann J, Pfister O, Osswald S, Mueller C. How accurate is clinical assessment of neck veins in the estimation of central venous pressure in acute heart failure? Insights from a prospective study. *Eur J Heart Fail* 2018;**20**:1160–1162.

Μη επεμβατικός έλεγχος ζωτικών σημείων πρέπει να γίνεται στην αρχική εκτίμηση

In accordance with the ESC guidelines, non-invasive monitoring should be performed at the time of initial presentation (class I, level of evidence C).

Vital signs

Standard non-invasive monitoring of heart rate, rhythm, respiratory rate, oxygen saturation and blood pressure is recommended (class I, level of evidence C).

Mebazaa A, Yilmaz MB, Levy P, Ponikowski P, Peacock WF, Laribi S, Ristic AD, Lambrinou E, Masip J, Riley JP, McDonagh T, Mueller C, deFilippi C, Harjola VP, Thiele H, Piepoli MF, Metra M, Maggioni A, McMurray J, Dickstein K, Damman K, Seferovic PM, Ruschitzka F, Leite-Moreira AF, Bellou A, Anker SD, Filippatos G. Recommendations on pre-hospital & early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine. *Eur J Heart Fail* 2015;**17**:544–558.

- **Οι ασθενείς με AHF στο ΤΕΠ εμφανίζονται συνηθέστερα:**

1. Συμφόρηση, χωρίς υποάρδευση οργάνων
2. Συμφόρηση και υποάρδευση οργάνων
3. Υποάρδευση οργάνων, χωρίς συμφόρηση

Acute heart failure (AHF)

70%: Συμφόρηση, χωρίς υποάρδευση οργάνων

13,6%: Συμφόρηση και υποάρδευση οργάνων

0,9%: Υποάρδευση οργάνων, χωρίς συμφόρηση

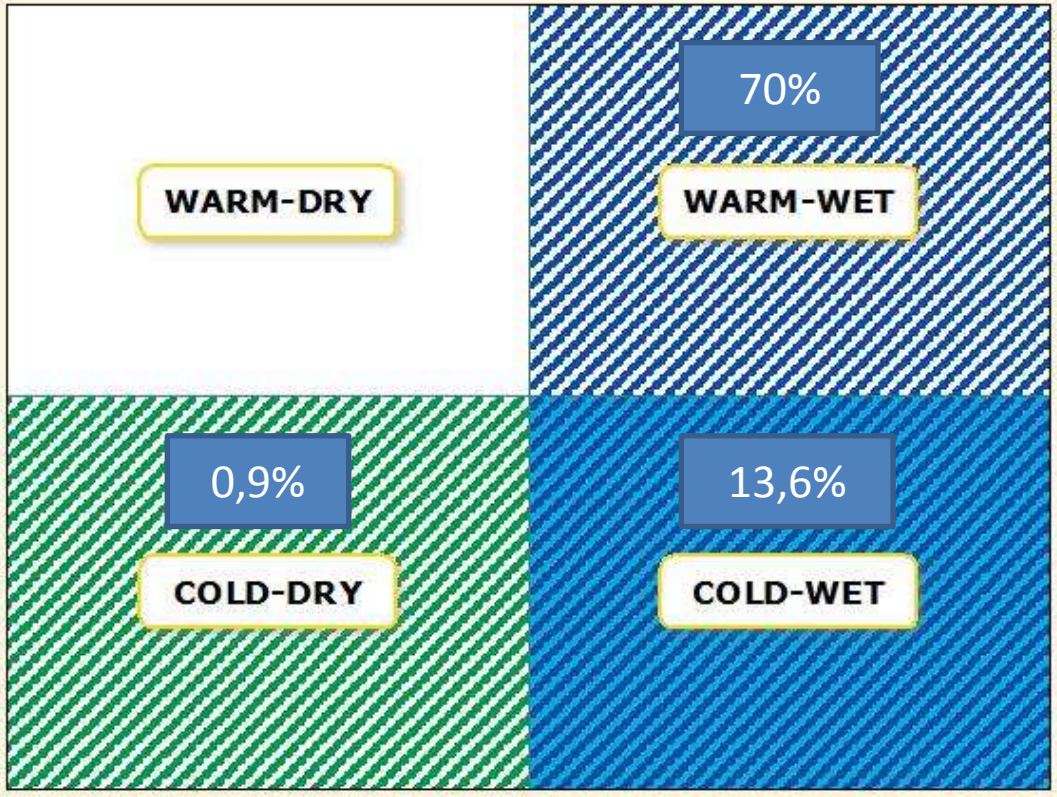
Chioncel O, Mebazaa A, Harjola VP, Coats AJ, Piepoli MF, Crespo-Leiro MG, Laroche C, Seferovic PM, Anker SD, Ferrari R, Ruschitzka F, Lopez-Fernandez S, Miani D, Filippatos G, Maggioni AP; ESC Heart Failure Long-Term Registry Investigators. Clinical phenotypes and outcome of patients hospitalized for acute heart failure: the ESC Heart Failure Long-Term Registry. *Eur J Heart Fail* 2017;**19**:1242–1254.

Clinical profiles of patients with acute heart failure based on the presence/absence of congestion and/or hypoperfusion

CONGESTION (−)

CONGESTION (+)
Pulmonary congestion
Orthopnoea/paroxysmal nocturnal dyspnoea
Peripheral (bilateral) oedema
Jugular venous dilatation
Congested hepatomegaly
Gut congestion, ascites
Hepatojugular reflux

HYPOPERFUSION (−)



HYPOPERFUSION (+)
Cold sweated extremities
Oliguria
Mental confusion
Dizziness
Narrow pulse pressure

Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension

Definitions of the terms used in Section 12 on acute heart failure (1)

Term	Definition
Symptoms/signs of congestion (left-sided)	Orthopnoea, paroxysmal nocturnal dyspnoea, pulmonary rales (bilateral), peripheral oedema (bilateral).
Symptoms/signs of congestion (right-sided)	Jugular venous dilatation, peripheral oedema (bilateral), congested hepatomegaly, hepatojugular reflux, ascites symptoms of gut congestion.
Symptoms/signs of hypoperfusion	Clinical: cold sweated extremities, oliguria, mental confusion, dizziness, narrow pulse pressure. Laboratory measures: metabolic acidosis, elevated serum lactate, elevated serum creatinine. Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension.
Hypotension	Systolic BP <90 mmHg
Bradycardia	Heart rate <40 bpm
Tachycardia	Heart rate >120 bpm
Abnormal respiratory effort	Respiratory rate >25 breaths/min with use of accessory muscles for breathing, or respiratory rate <8 breaths/min despite dyspnoea.

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Examination findings							
S3[7,8,14,15,20,53–55,57–60,64,65]	14	5,900	45.2 (44.0–46.5)	12.7 (11.5–14.0)	97.7 (97.2–98.2)	4.0 (2.7–5.9)	0.91 (0.88–0.95)
JVD[7,8,12,14–16,18,19,21,25,36,48,51,53–55,57–61,64,65]	23	8,012	47.8 (46.7–48.9)	37.2 (35.7–38.7)	87.0 (85.9–88.0)	2.8 (1.7–4.5)	0.76 (0.69–0.84)
Hepatojugular reflex[56,59,61,65]	4	1,209	60.4 (57.6–63.1)	14.1 (11.9–16.6)	93.4 (91.2–95.2)	2.2 (1.3–3.7)	0.91 (0.88–0.94)
Leg edema[7,8,10,12,14,15,16,18,19–21,23,25,48,49,51,53–55,57–62,65]	26	9,626	47.2 (46.2–48.2)	51.9 (50.5- 53.4)	75.2 (74.0–76.4)	1.9 (1.6–2.3)	0.68 (0.61–0.75)
Murmur[7,12,51,54,55,58,62,65]	8	4,004	45.3 (43.8–46.8)	27.8 (25.8–29.9)	83.2 (81.6–84.8)	1.9 (0.9–3.9)	0.93 (0.79–1.08)
Rales[7,8,10,12,15,18–21,23,25,36,48,51,53–55,58–61,65]	22	8,775	48.2 (47.1–49.2)	62.3 (60.8–63.7)	68.1 (66.7–69.4)	1.8 (1.5–2.1)	0.60 (0.51–0.69)
Wheezing[7,8,12,15,20,23,36,48,53,55,58,59,65]	13	6,970	44.2 (43.0–45.3)	22.3 (20.9–23.8)	64.0 (62.5–65.4)	0.6 (0.5–0.8)	1.19 (1.10–1.30)
Absent fever[7,23,36,49,59,62,63]	7	3,197	43.6 (41.9–45.3)	92.4 (90.9–93.8)	20.6 (18.8–22.5)	1.14 (1.02–1.27)	0.4 (0.3–0.6)

AFIB = atrial fibrillation; CAD = coronary artery disease; CHF = congestive heart failure; COPD = chronic obstructive pulmonary disease; CRI = chronic renal insufficiency; DM = diabetes mellitus; HTN = hypertension; JVD = jugular venous distension; LR+ = positive likelihood ratio; LR- = negative likelihood ratio; MI = myocardial infarction; PND = paroxysmal nocturnal dyspnea.

- **Ο ασθενής παρουσίαζε:**

- Συμπτώματα - σημεία: σύγχυση, ψυχρά άκρα, ταχύπνοια (> 25 αναπνοές/λεπτό), χαμηλό κορεσμό αρτηριακού αίματος σε οξυγόνο (SaO2 <90%), μειωμένο αναπνευστικό ψιθύρισμα, ταχυκαρδία (>120 bpm), υπόταση (SBP <90 mmHg).

- Συμβατά ευρήματα με το 2^{ο} κλινικό πρότυπο AHF (συμφόρηση και υποάρδευση οργάνων).

Αέρια αρτηριακού αίματος

- Routine arterial blood gas is not needed.

Αέρια αρτηριακού αίματος

Measurement of blood pH and carbon dioxide tension (possibly including lactate) should be considered, especially in patients with acute pulmonary oedema or previous history of COPD using venous blood. In patients with cardiogenic shock arterial blood is preferable.	IIa	C

Αέρια αρτηριακού αίματος

- Πληροφορίες αερισμού, οξυγόνωσης, μεταβολικής ισορροπίας, ιστικής υποξίας.
- Ενδείκνυται μέτρηση σε οξύ πνευμονικό οίδημα, σοβαρή AHF (στα πρόθυρα διασωλήνωσης), καρδιογενές shock και ιστορικό ΧΑΠ.
- Συνηθέστερη οξεοβασική διαταραχή σε AHF είναι η αναπνευστική αλκάλωση. Ακολουθεί η μεταβολική οξέωση, που αποτελεί δείκτη πτωχής περιφερικής αιμάτωσης.
- Αρτηριοφλεβική διαφορά pCO_2 >6 mmHg αποτελεί δείκτη κακής ιστικής αιμάτωσης.

Γαλακτικό οξύ

- Αυξημένα επίπεδα είναι αποτέλεσμα είτε υπέρμετρης παραγωγής, είτε μειωμένης κάθαρσης (π.χ. ηπατικής δυσλειτουργίας), ή και των δύο.
- Στα αρχικά στάδια είναι αποτέλεσμα χαμηλής παροχής, ιστικής υποξίας, αναερόβιου μεταβολισμού.
- Επίπεδα >2 mmol/L συσχετίζονται με αυξημένη θνητότητα, όπως και αμετάβλητες τιμές μετά από κατάλληλη θεραπεία (μετρήσεις ανά 1-2 ώρες σε αιμοδυναμικά ασταθή ή υποξαιμικό ασθενή με AHF).

Definitions of the terms used in Section 12 on acute heart failure (2)

Term	Definition
Low O_2 saturation	O_2 saturation (SaO_2) <90% in pulse oximetry Normal SaO_2 neither excludes hypoxaemia (low PaO_2) nor tissue hypoxia.
Hypoxaemia	O_2 partial pressure (PaO_2) in arterial blood <80 mmHg (<10,67 kPa) (blood gas analysis).
Hypoxaemic respiratory failure (type I)	PaO_2 <60 mmHg (<8 kPa)
Hypercapnia	CO_2 partial pressure ($PaCO_2$) in arterial blood >45 mmHg (>6 kPa) (blood gas analysis).
Hypercapnic respiratory failure (type II)	$PaCO_2$ >50 mmHg (>6,65 kPa).
Acidosis	pH <7.35
Elevated blood lactate	>2 mmol/L
Oliguria	Urine output <0.5 mL/kg/h

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Αέρια ασθενή

SaO2: 90,5%
pH: 7,043
PaO2: 84,7 mmHg
PaCO2: 67,8 mmHg
HCO2: 13,1 mmol/L
Lac: 5,4 mmol/L

Υπό ρινική κάνουλα 3 lt/min

Patient Last Name	BIPAP
Patient First Name	
Sample type	Not specified
T	37.0 °C

Blood Gas Values					
pH	7.043			-	]
pCO_2	67.8	mmHg		-	]
pO_2	84.7	mmHg		-	
Oximetry Values					]
*c*tHb	17.7	g/dL		-	]
sO_2	90.5	%		-	]
FO_2Hb	88.4	%		-	]
*F*COHb	0.8	%		-	]
*F*HHb	9.3	%		-	]
*F*MetHb	1.5	%			
Electrolyte Values					
cK^+	4.6	mmol/L		-	]
cNa^+	135	mmol/L		-	]
cCa^{2+}	1.05	mmol/L		-	]
cCl^-	103	mmol/L		-	]
Metabolite Values					
*c*Glu	308	mg/dL		-	]
*c*Lac	5.4	mmol/L		-	]
Temperature Corrected Values					
pH(T)	7.043				
$pCO_2(T)$	67.8	mmHg			
$pO_2(T)$	84.7	mmHg			
Oxygen Status					
ctO_2c	22.0	Vol%			
$p50_c$	39.78	mmHg			
Acid Base Status					
$cBase(Ecf)_c$	-11.4	mmol/L			
$cHCO_3^-(P,st)_c$	13.1	mmol/L			

Notes

c Calculated value(s)

Patient First Name				
Sample type	Not specified			
T	37.0 °C			
Blood Gas Values				
pH	7.202		[-]	
pCO_2	67.7	mmHg	[-]	
pO_2	87.3	mmHg	[-]	
Oximetry Values				
ctHb	17.5	g/dL	[-]	
sO_2	93.6	%	[-]	
FO_2Hb	91.7	%	[-]	
FCOHb	0.9	%	[-]	
FHHb	6.3	%	[-]	
FMetHb	1.1	%	[-]	
Electrolyte Values				
cK^+	4.3	mmol/L	[-]	
cNa^+	138	mmol/L	[-]	
cCa^{2+}	0.96	mmol/L	[-]	
cCl^-	102	mmol/L	[-]	
Metabolite Values				
cGlu	275	mg/dL	[-]	
cLac	4.4	mmol/L	[-]	
Temperature Corrected Values				
pH(T)	7.202			
$pCO_2(T)$	67.7	mmHg		
$pO_2(T)$	87.3	mmHg		
Oxygen Status				
ctO_2c	22.5	Vol%		
$p50_c$	34.92	mmHg		
Acid Base Status				
$cBase(Ecf)_c$	-1.5	mmol/L		
$cHCO_3^-(P,st)_c$	20.7	mmol/L		

Notes

c Calculated value(s)

Ηλεκτροκαρδιογράφημα

Electrocardiogram	12-lead ECG is recommended at admission in all patients presenting with suspected AHF (class I, level of evidence C).

Ηλεκτροκαρδιογράφημα

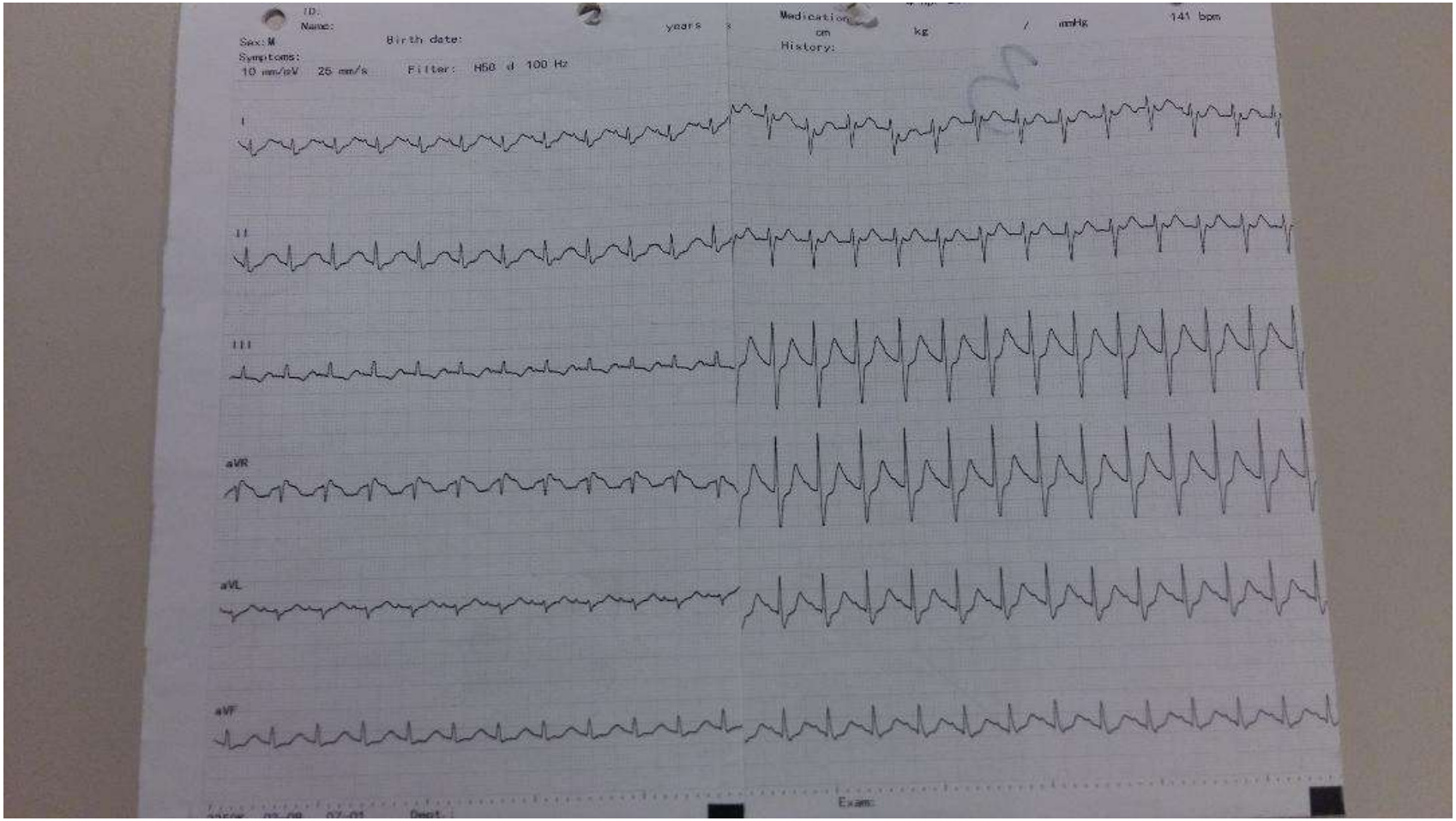
- Σπάνια φυσιολογικό σε AHF **(Υψηλή αρνητική προγνωστική αξία)**

- Συνήθως **διακρίνει υποκείμενο αίτιο** AHF:

Οξεία μυοκαρδιακή ισχαιμία (διαταραχές ST-T επαρμάτων)

Αρρυθμίες (π.χ. ταχεία κολπική μαρμαρυγή)

Το ΗΚΓ του ασθενούς παρουσίαζε φλεβοκομβική ταχυκαρδία και μη ειδικού τύπου διαταραχές αναπόλωσης


ID:
Name:
years
Medication:
Sex: M
Birth date:
cm
kg
/
mmHg
141 bpm
History:
Symptoms:
10 mm/mV
25 mm/s
Filter: H50 d 100 Hz
I
II
III
aVR
aVL
aVF
Exam:

Ακτινογραφία θώρακος

Chest X-ray	Chest X-ray is recommended at admission in all patients presenting with suspected AHF to assess signs of pulmonary congestion and detect other cardiac or non-cardiac diseases that may cause or contribute to the patient's symptoms (class I, level of evidence C).

Το θώρακος

- Κυρίως διάκριση εξωκαρδιακών αιτίων δύσπνοιας (π.χ. πνευμονία – ARDS)

Το θώρακος

ΕΥΡΗΜΑΤΑ ΣΕ ΑΗF

1. Πνευμονική φλεβική συμφόρηση-Γραμμές Kerley-B
2. Διάμεσο-κυψελιδικό πνευμονικό οίδημα
3. Υπεζωκοτικές συλλογές
4. Μεγαλοκαρδία

Το θώρακος

ΕΥΡΗΜΑΤΑ ΣΕ ARDS

Όταν

- Λείπουν 1-3-4
- Υπάρχει περιφερική διαύγαση

- **Η ακτινογραφία θώρακος στο ΤΕΠ μπορεί να αποκλείσει ΑΗF;**

1. ΝΑΙ
2. ΟΧΙ

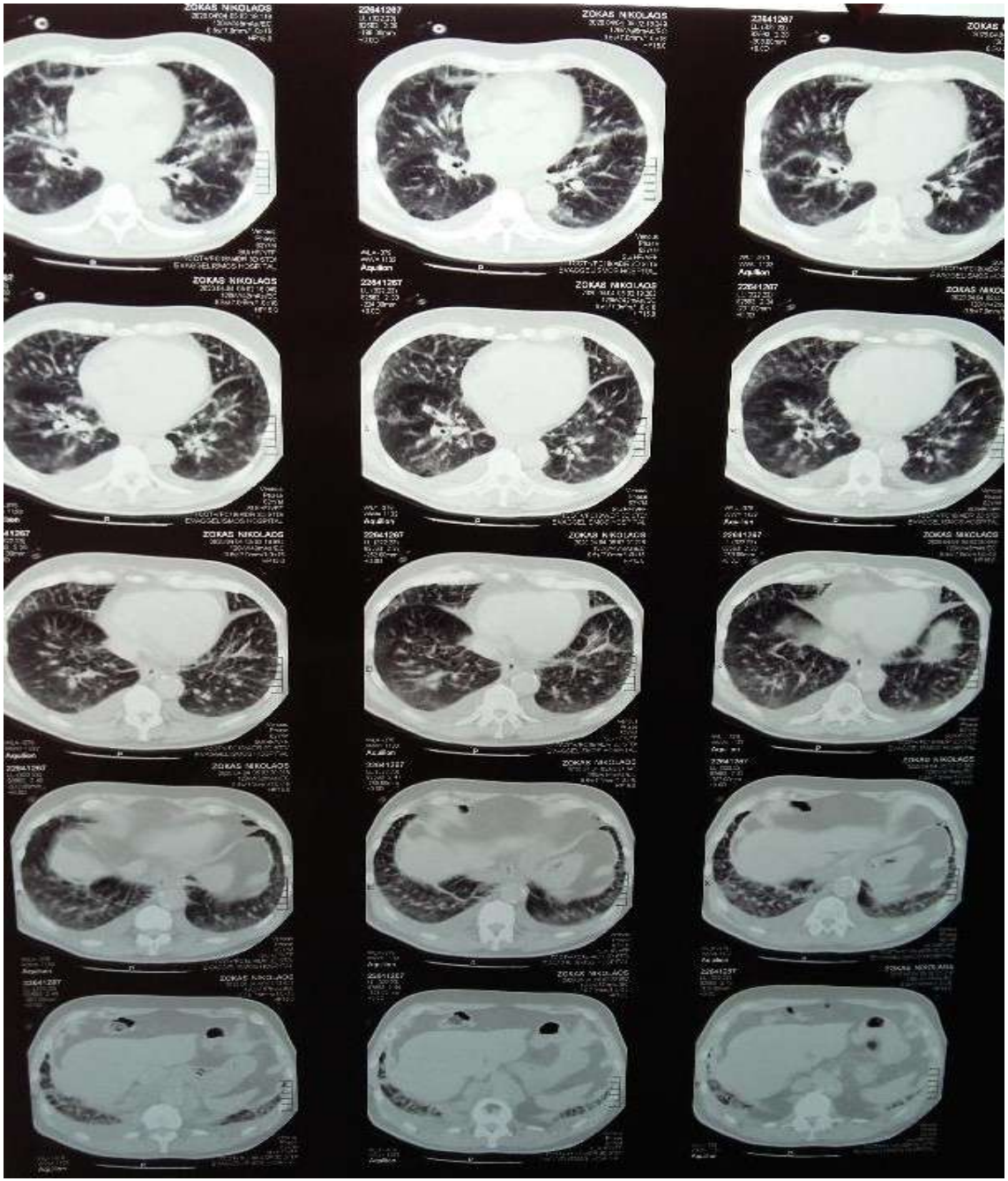
Όχι , γιατί σε περίπου 20% των ασθενών με AHF, η ακτινογραφία θώρακος στο ΤΕΠ παρουσιάζει μικρή ή καθόλου συμφόρηση.

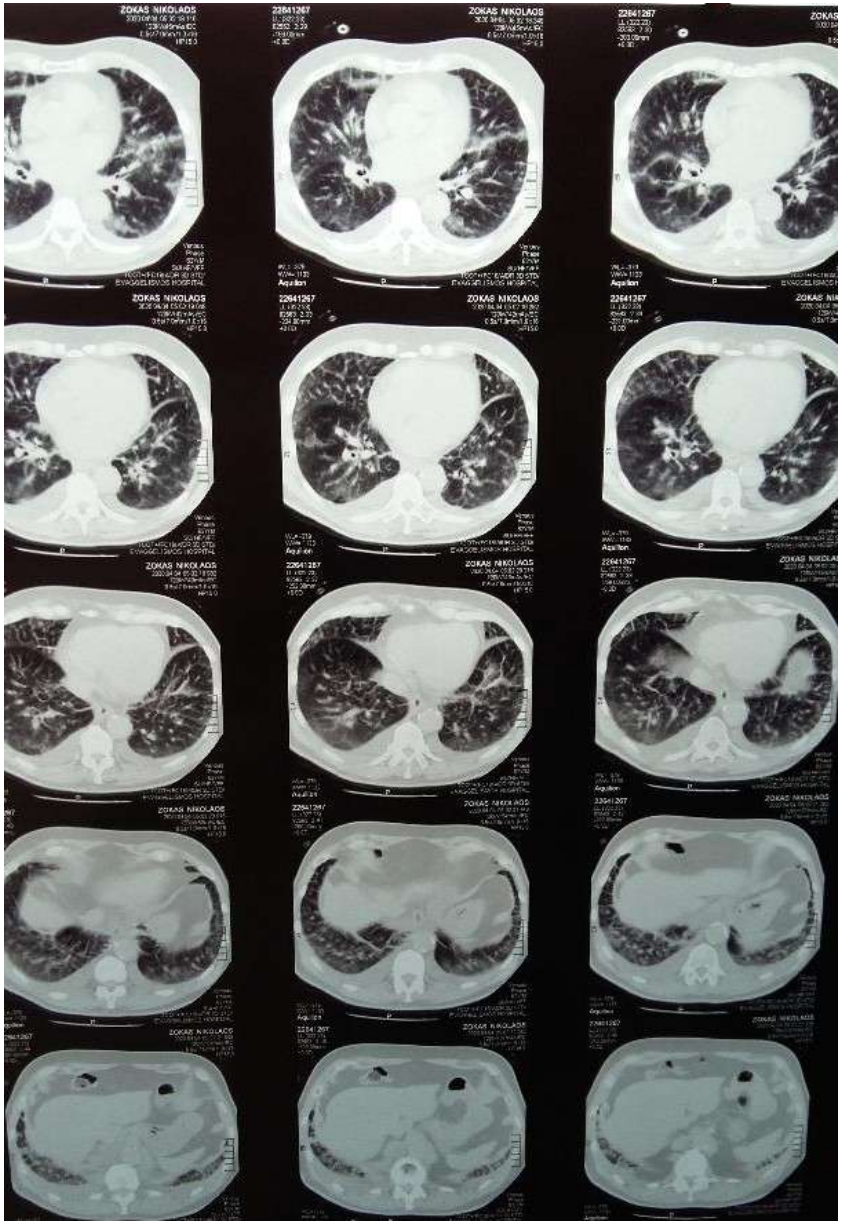
Collins SP, Lindsell CJ, Storrow AB, Abraham WT. Prevalence of negative chest radiography results in the emergency department patient with decompensated heart failure. *Ann Emerg Med* 2006;**47**:13–18.

- Η ακτινογραφία του ασθενή:
  σκιάσεις βάσεων άμφω – δικτυωτό πρότυπο;


R

Αξονική τομογραφία θώρακος


ZOKAS NIKOLAOS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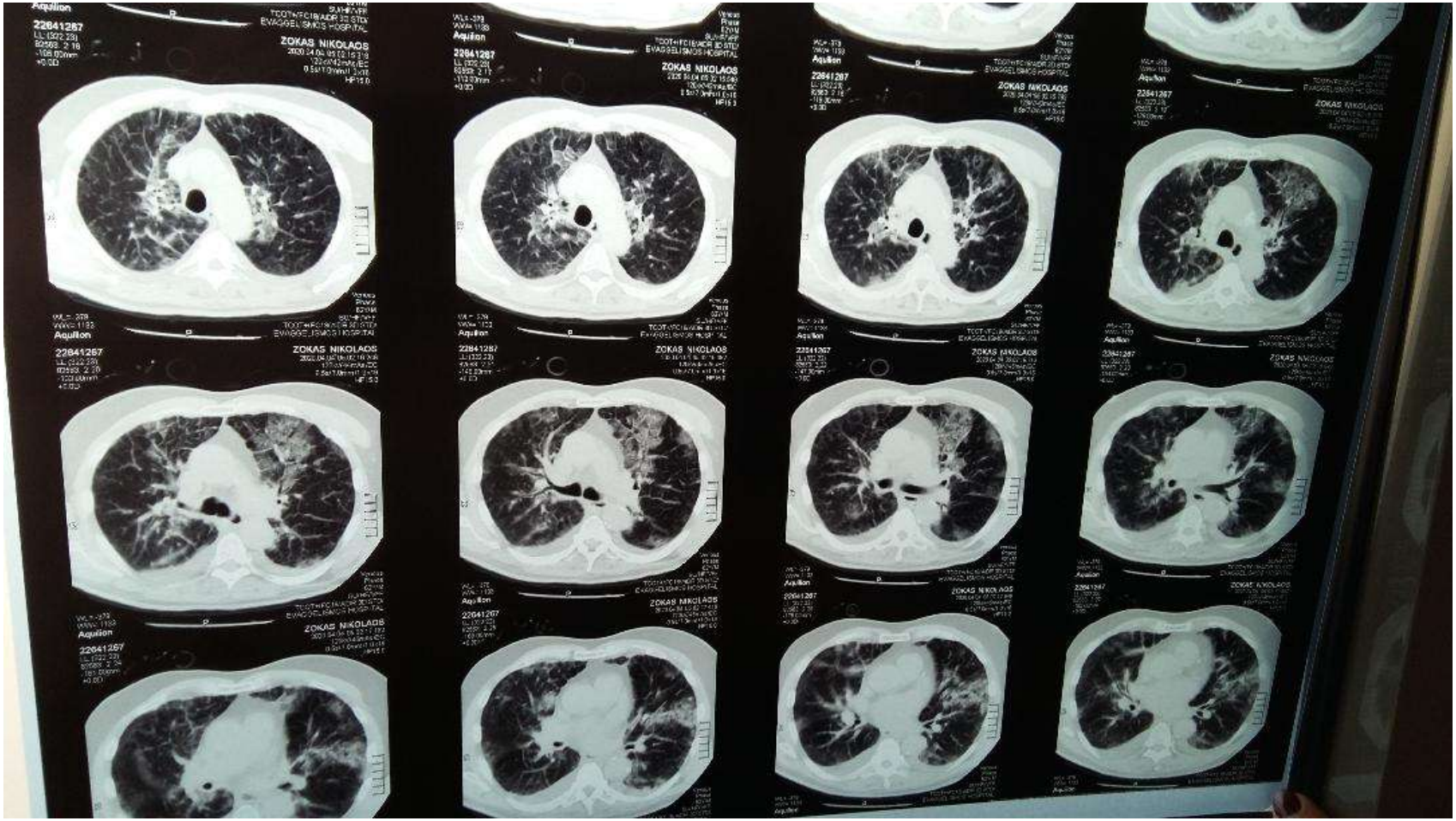
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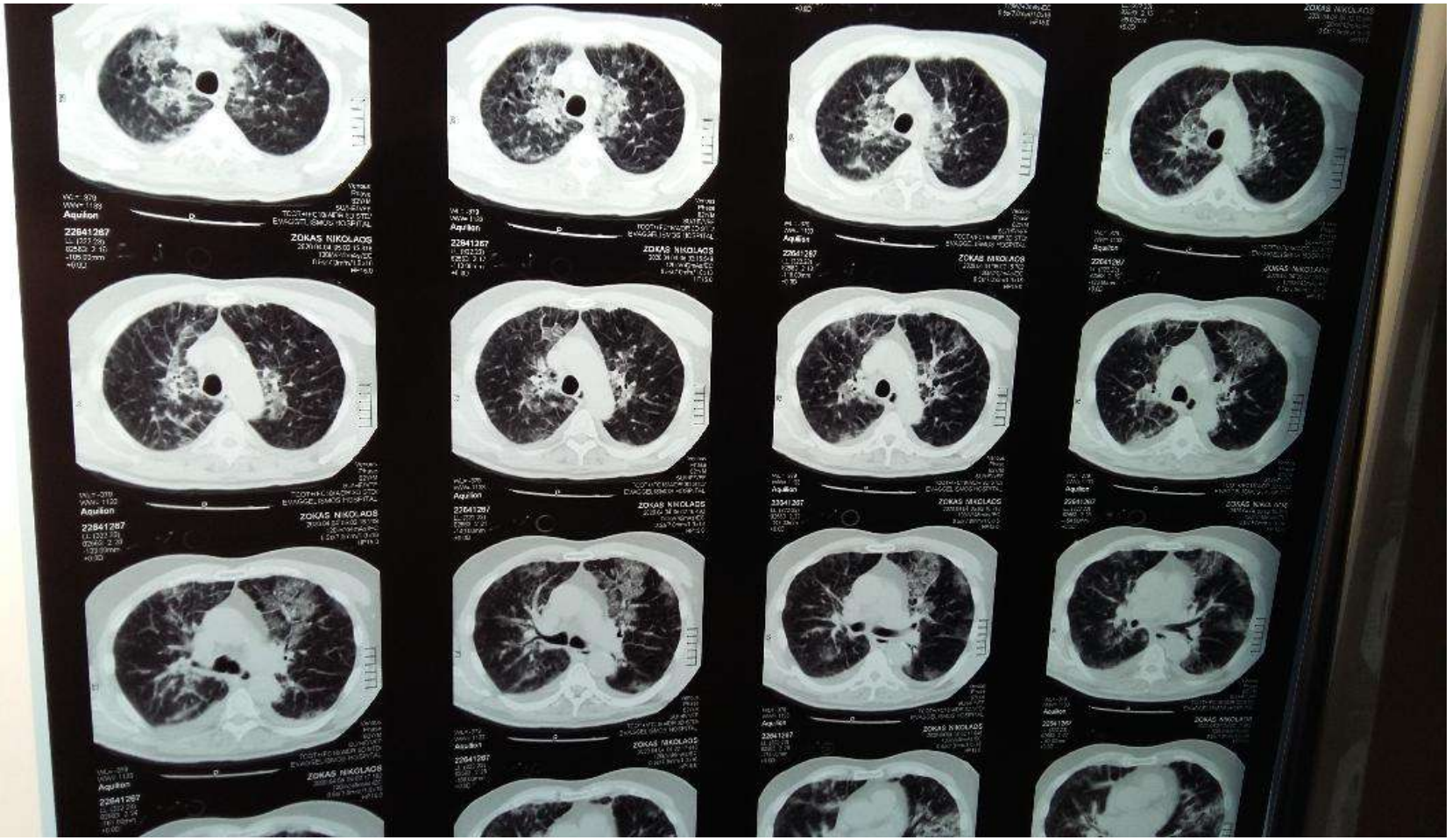
ZOKAS NIKOLAOS
EVAGGELISMOS HOSPITAL


Aquilion
22841287
ZOKAS NIKOLAOS

CT ΑΓΓΕΙΟΓΡΑΦΙΑ

Έγινε έλεγχος της πνευμονικής αρτηρίας με αγγειογραφικό πρωτόκολλο, καθώς και της θωρακικής και της κοιλιακής αορτής πριν και μετά την ενδοφλέβια χορήγηση σκιαγραφικού.

Δεν απεικονίσθηκαν ελλείμματα σκιαγράφησης σε στελεχιαίους, λοβαίους και τμηματικούς κλάδους της πνευμονικής αρτηρίας.
Το εύρος της πνευμονικής αρτηρίας και των στελεχιαίων κλάδων είναι εντός του φυσιολογικού.
Παλινδρόμηση σκιαγραφικού στις ενδαηπατικές φλέβες ενδεικτικό καρδιακής ανεπάρκειας.
Υπεζωκοτικές συλλογές άμφω με επίταση των μεσολοβιδίων διαφραγματίων σε όλο το πνευμονικό παρέγχυμα με αμφοτερόπλευρη ασυμμετρη κατανομή διηθημάτων δίκην πλακόστρωτου. Κλινικοεργαστηριακή συνεκτίμηση στην κατεύθυνση καρδιακής συμφόρησης, ενώ δεν μπορεί να αποκλειστεί και το ενδεχόμενο συνύπαρξης λοίμωξης.
Εμφυσηματικές αλλοιώσεις.

Δεν απεικονίσθηκαν παθολογικά διογκωμένοι λεμφαδένες (βραχείας διαμέτρου >1εκ) στα ανατομικά διαμερίσματα του μεσοθωρακίου και στις μασχαλιαίες χώρες.
Δεν απεικονίσθηκε περικαρδιακή συλλογή.

Αιματολογικός – Βιοχημικός έλεγχος

Νατριουρητικά πεπτίδια (BNP/NT-proBNP)

Biomarkers	Upon presentation, a measurement of plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP) is recommended in all patients with acute dyspnoea and suspected AHF to help in the differentiation of AHF from non-cardiac causes of acute dyspnoea (class I, level of evidence A).

- **NPs** have high sensitivity, and normal levels in patients with suspected AHF makes the diagnosis unlikely

  (thresholds: BNP 100 pg/mL, NT-proBNP 300 pg/mL, MR-proANP 120 pg/mL)

Causes of elevated concentrations of natriuretic peptides

Cardiac	Heart failure
	Acute coronary syndromes
	Pulmonary embolism
	Myocarditis
	Left ventricular hypertrophy
	Hypertrophic or restrictive cardiomyopathy
	Valvular heart disease
	Congenital heart disease
	Atrial and ventricular tachyarrhythmias
	Heart contusion
	Cardioversion, ICD shock
	Surgical procedures involving the heart
	Pulmonary hypertension

Non-cardiac	Advanced age
	Ischaemic stroke
	Subarachnoid haemorrhage
	Renal dysfunction
	Liver dysfunction (mainly liver cirrhosis with ascites)
	Paraneoplastic syndrome
	Chronic obstructive pulmonary disease
	Severe infections (including pneumonia and sepsis)
	Severe burns
	Anaemia
	Severe metabolic and hormone abnormalities (e.g. thyro-toxicosis, diabetic ketosis)

European Heart Journal (2016) **37**, 2129-2200 - doi:10.1093/eurheartj/ehw 128

Νατριουρητικά πεπτίδια (BNP/NT-proBNP)

Table 3
Pooled Test Performance Characteristics for Natriuretic Peptides

Assay	Cutoff (pg/mL)	*N*	*n*	% AHF (95% CI)	Sensitivity % (95%CI)	Specificity % (95%CI)	LR+ (95% CI)	LR– (95% CI)
BNP								
Triage, Biosite	100[14,19,35,50,51,55,58,60,66,67,73–78,82,84,85]	19	9,143	44.7 (43.7–45.8)	93.5 (92.6–94.2)	52.9 (51.6–54.2)	2.2 (1.8–2.7)	0.11 (0.07–0.16)
	200[11,14,19,51,66,73,75–78,84]	11	3,279	50.4 (48.7–52.1)	85.9 (84.2–87.6)	72.2 (69.9–74.4)	3.1 (2.3–4.0)	0.18 (0.12–0.27)
	500[14,51,58,74–78]	8	3,915	46.7 (45.1–48.3)	67.7 (65.5–69.9)	89.8 (88.5–91.1)	9.1 (4.1–20.2)	0.34 (0.26–0.45)
AxSym, Abbott	100[18,21,36,46]	4	684	52.3 (48.6–56.1)	93.3 (90.2–95.7)	53.1 (47.5–58.6)	1.9 (1.5–2.4)	0.15 (0.08–0.29)
iSTAT, Abbott	100[85,86]	2	585	42.6 (38.6–46.6)	94.4 (90.7–96.9)	64.6 (59.2–69.7)	3.0 (1.2–7.4)	0.05 (0.02–1.23)
NT-proBNP								
Elecsys, Roche diagnostic	300[7,20,46,64,73,75,77,81,83,87]	10	3,498	45.0 (43.4–46.7)	90.4 (88.9–91.8)	38.2 (36.0–40.4)	1.8 (1.4–2.2)	0.09 (0.03–0.34)
	1,000[7,46,62,73,75,77,81,83]	8	2,988	44.8 (43.0–46.6)	84.8 (82.8–86.7)	65.5 (63.2–67.8)	2.7 (1.9–3.9)	0.20 (0.12–0.33)
	1,550[10,46,61,75,77,79–81,83]	9	3,043	37.3 (35.6–39.0)	75.5 (73.4–77.9)	72.9 (70.6–75.0)	3.1 (2.3–4.3)	0.32 (0.20–0.51)
Dimension, Dade Behring	300[70]	1	401	30.4 (26.0–35.2)	95.9 (90.7–98.6)	48.0 (42.0–54.1)	1.9 (1.6–2.1)	0.09 (0.04–0.20)

AHF = acute heart failure; BNP = B-type natriuretic peptide; LR+ = positive likelihood ratio; LR– = negative likelihood ratio; *N* = number of studies; *n* = number of patients; NT-proBNP = N-terminal proB-type natriuretic peptide.

ΒΙΟΧΗΜΙΚΟΣ ΕΛΕΓΧΟΣ		ΤΙΜΕΣ ΑΝΑΦΟΡΑΣ
Γλυκόζη ορού	**153**	70 - 110 mg/dL
Ουρία ορού	**55**	10 - 50 mg/dL
Κρεατινίνη ορού	**1.5**	0.6 - 1.4 mg/dL
Νάτριο ορού	136	135 - 147 mmol/L
Κάλιο ορού	4.6	3.5 - 5.1 mmol/L
AST(SGOT) ορού	25	5 - 37 IU/L
ALT(SGPT) ορού	17	5 - 40 IU/L
Αλκαλική Φωσφατάση ορού	59	40 - 129 IU/L
γ-GT ορού	14	8 - 49 IU/L
Ολική Χολερυθρίνη ορού	0.55	< 1 mg/dL
Άμεση Χολερυθρίνη ορού	0.16	< 0.3 mg/dL
Χολερυθρίνη έμμεση ορού	0.39	< 0.75 mg/dL
LDH ορού	**249**	< 225 IU/L
Κρεατινική Κινάση(CK) ορού	**245**	10 - 173 IU/L 37°C
Κρεατινική Κινάση(CKMB) ορού	**50**	1 - 18 IU/L
Ασβέστιο Ολικό ορού	9.8	8.5 - 10.5 mg/dL
Φώσφορος ορού	4.5	2.5 - 5 mg/dL
Μαγνήσιο ορού	1.93	1.58 - 2.55 mg/dL
Αμυλάση ορού	**119**	10 - 100 IU/L 37°C
CRP ορού	0.4	< 0.5 mg/dL
Troponin-T high sensitive ορού	237	< 12 pg/mL Φυσιολογικό 12 - 52 pg/mL Επανάληψη μετά 3ωρ > 52 pg/mL Παθολογικό
NT-ProBNP ορού	244	< 450 pg/mL 1 - 49 Ετών < 900 pg/mL 50 - 75 Ετών < 1800 pg/mL > 76 Ετών

Άλλες εργαστηριακές μετρήσεις

Laboratory assessments	Cardiac troponins, blood urea nitrogen, creatinine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests, and thyroid stimulating hormone are recommended at admission in all patients presenting with suspected AHF (class I, level of evidence C).

Καρδιακές τροπονίνες

- Διάγνωση οξέος στεφανιαίου συνδρόμου σε δυναμικές αλλαγές (προέχων αίτιο επεισοδίου δύσπνοιας)
- Διαστρωμάτωση κινδύνου-επιλογή θεραπείας σε πνευμονική εμβολή με θετική τροπονίνη

Markers of liver function

Liver function tests should be performed on admission in accordance with current guidelines (class I, level of evidence C).[6] Liver dysfunction in AHF is most commonly the result of elevated central venous pressure and manifested by increases in alkaline phosphatase and gamma-glutamyl transferase[58] or total bilirubin;[59] these abnormalities often improve with decongestion. Elevated transaminases occur with hypoxic liver injury in the setting of hypoperfusion, which is less common, but associated with worse outcome.[13]

13. Harjola VP, Mullens W, Banaszewski M, Bauersachs J, Brunner-La Rocca HP, Chioncel O, Collins SP, Doehner W, Filippatos GS, Flammer AJ, Fuhrmann V, Lainscak M, Lassus J, Legrand M, Masip J, Mueller C, Papp Z, Parissis J, Platz E, Rudiger A, Ruschitzka F, Schafer A, Seferovic PM, Skouri H, Yilmaz MB, Mebazaa A. Organ dysfunction, injury and failure in acute heart failure: from pathophysiology to diagnosis and management. A review on behalf of the Acute Heart Failure Committee of the Heart Failure Association (HFA) of the European Society of Cardiology (ESC). *Eur J Heart Fail* 2017;**19**:821–836.

- Assessment of **procalcitonin** levels may be considered in patients with AHF with suspected coexisting infection, particularly for the differential diagnosis of pneumonia and to guide antibiotic therapy, if considered.

Maisel A, Neath SX, Landsberg J, Mueller C, Nowak RM, Peacock WF, Ponikowski P, Mo¨ ckel M, Hogan C, Wu AHB, Richards M, Clopton P, Filippatos GS, Di Somma S, Anand I, Ng LL, Daniels LB, Christenson RH, Potocki M, McCord J, Terracciano G, Hartmann O, Bergmann A, Morgenthaler NG, Anker SD. **Use of procalcitonin for the diagnosis of pneumonia in patients presenting with a chief complaint of dyspnoea: results from the BACH (Biomarkers in Acute Heart Failure) trial**. Eur J Heart Fail 2012;14:278–286.

ΓΕΝΙΚΗ ΕΞΕΤΑΣΗ ΑΙΜΑΤΟΣ

Λευκά Αιμοσφαίρια και Τύπος		
Λευκά αιμοσφαίρια WBC	**13.14**	4 - 10.5
Ουδετερόφιλα NEU%	**71.2**	40 - 70
Λεμφοκύτταρα LYM%	**23.4**	25 - 45
Μονοκύτταρα MONO%	4.6	2 - 10
Βασεόφιλα BASO%	**0.2**	0.3 - 1
Ηωσινόφιλα EOS%	**0.6**	1 - 6
NEU#	**9.35**	2 - 7.7
LYM#	3.08	1.5 - 4
MONO#	0.61	0.02 - 1
BASO#	0.02	0.01 - 0.1
EOS#	0.06	0.04 - 0.4
Ερυθρά και ερυθροκυτταρική σειρά		
Ερυθρά αιμοσφαίρια (RBC)	4.99	4.5 - 5.9
Αιματοκρίτης (HCT)	45.2	41 - 51
Αιμοσφαιρίνη (HGB)	16.3	13.5 - 17.5
MCV	90.6	80 - 98
MCH	32.7	27 - 33
MCHC	**36.1**	32 - 36
RDW-SD	**46.2**	38 - 43
RDW-CV	14.1	11 - 16
Αιμοπετάλια		
Αιμοπετάλιο (PLT)	167	140 - 450
PDW	13.3	9 - 17
MPV	**11.1**	6 - 11
P-LCR	33.5	13 - 43
PCT	0.19	0.17 - 0.35
Δικτυοερυθροκύτταρα		
ΔΕΚ%	1.21	0.5 - 2
Λοιπά στοιχεία		
Εμπύρηνα ερυθρά (NRBC%)	0.0	/100WBC

ΕΛΕΓΧΟΣ ΑΙΜΟΣΤΑΣΗΣ

Χρόνος Quick PT	11.7	sec
Χρόνος Quick (PT%)	**130**	80 - 110 %

ΠΡΟΕΛΕΥΣΗ ΠΝΕΥΜΟΛΟΓΙΚΟ ΤΕΠ ORDERID 011209520
ΗΜΕΡ.ΕΞΕΤΑΣΗΣ 4/4/2020 01:06 πμ ΑΡΙΘΜ.ΜΗΤΡΩΟΥ 010909520

ΕΛΕΓΧΟΣ ΑΙΜΟΣΤΑΣΗΣ		ΤΙΜΕΣ ΑΝΑΦΟΡΑΣ	
INR	0.92		
APTT	29.9	26 - 38	sec
Ινωδογόνο	**422**	200 - 400	mg/dL
D-dimer	**2.04**	< 0.5	μg/mL
ΑΙΜΑΤΟΛΟΓΙΚΟΣ ΕΛΕΓΧΟΣ			
ΛΟΙΠΕΣ ΕΞΕΤΑΣΕΙΣ			
ΤΚΣ	24	< 30	mm/h

Ο/Η ΙΑΤΡΟΣ

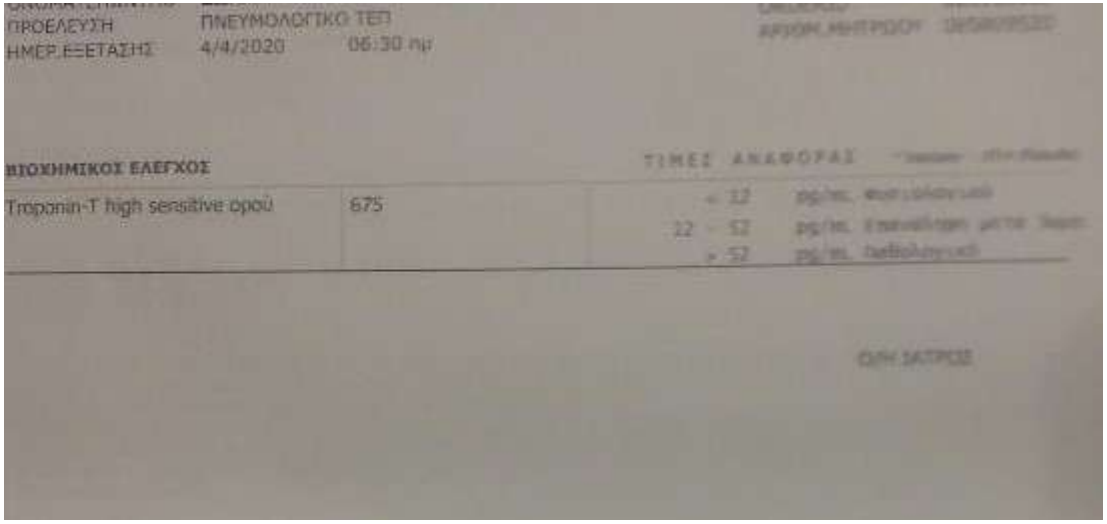
ΠΡΟΕΛΕΥΣΗ	ΕΞΩΤΕΡΙΚΟΣ		ORDERID	120309520
ΗΜΕΡ.ΕΞΕΤΑΣΗΣ	4/4/2020	09:58 πμ	ΑΡΙΘΜ.ΜΗΤΡΩΟΥ	0000300028044

ΕΡΓΑΣΤΗΡΙΟ ΜΟΡΙΑΚΗΣ ΔΙΑΓΝΩΣΤΙΚΗΣ ΤΙΜΕΣ ΑΝΑΦΟΡΑΣ *Validator OKUSER

COVID-19 TEST	
COVID-19 TEST	ΑΡΝΗΤΙΚΟ (SARS-CoV-2 RNA μη ανιχνεύσιμο)

Παρατηρήσεις : Η εξέταση πραγματοποιήθηκε με Reverse Transcriptase Real Time PCR η οποία διαθέτει άδεια διαγνωστικής χρήσης (CE/IVD) για τη διάγνωση του SARS-CoV-2 σε δείγματα του αναπνευστικού. Στόχοι: RdRp gene, E gene, N gene Έλεγχος απομόνωσης νουκλεϊκών οξέων και παρουσίας ανασταλτικών ουσιών (inhibitors): human RNAse P Αναλυτική ευαισθησία (analytical sensitivity): 10 copies /reaction για όλα τα γονίδια στόχους (RdRp, E gene, N gene). Η εξέταση επιβεβαιώθηκε με δεύτερη μέθοδο Reverse Transcriptase Real Time PCR η οποία επίσης διαθέτει άδεια διαγνωστικής χρήσης (CE/IVD) για τη διάγνωση του SARS-CoV-2 σε δείγματα του αναπνευστικού. Στόχοι: ORF1ab, N gene Αναλυτική ευαισθησία (analytical sensitivity):>=10RNA copies/reaction για τα γονίδια στόχους ORF1ab, N gene

Ο/Η ΙΑΤΡΟΣ



ΠΡΟΕΛΕΥΣΗ ΠΝΕΥΜΟΛΟΓΙΚΟ ΤΕΠ
ΗΜΕΡ.ΕΞΕΤΑΣΗΣ 4/4/2020 06:30 πμ

ΒΙΟΧΗΜΙΚΟΣ ΕΛΕΓΧΟΣ

		ΤΙΜΕΣ ΑΝΑΦΟΡΑΣ
Troponin-T high sensitive opou	675	< 12 pg/ml, φυσιολογικά 12 - 52 pg/ml, ενδιαμέσων μετρα θερα > 52 pg/ml, παθολογικά

ΟΔΗΓΟΣ

Υπερηχοκαρδιογράφημα

- Για τη διαφοροδιάγνωση *AHF* και *ARDS* θα βοηθήσει ιδιαίτερα το ηχοκαρδιογράφημα

Echo καρδιάς επειγόντως στο ΤΕΠ

- Σε αιμοδυναμική αστάθεια (ιδιαίτερα καρδιογενές shock)
- Σε υποψία απειλητικής για τη ζωή δομικής ή λειτουργικής καρδιακής διαταραχής (μηχανικής επιπλοκής, οξείας βαλβιδικής ανεπάρκειας, αορτικού διαχωρισμού)

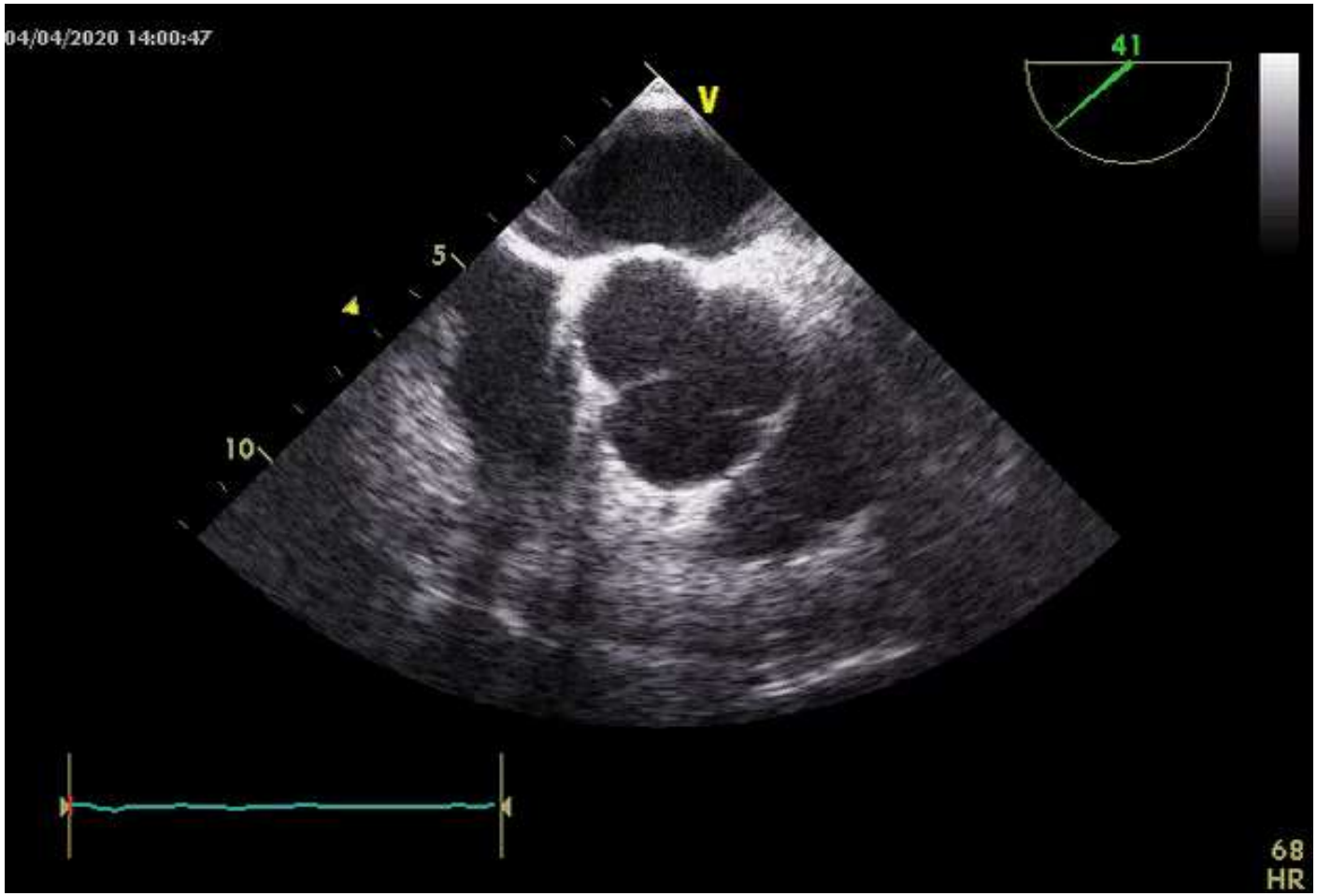
Ηχοκαρδιογράφημα

- Συστολικοί δείκτες: Μειωμένο κλάσμα εξώθησης αριστερής κοιλίας - Αυξημένη τελοδιαστολική διάμετρος αρ. Κοιλίας – Μειωμένη καρδιακή παροχή
- Διαστολικοί δείκτες: Περιοριστικό σήμα διαμιτροειδικής ροής - E/e’>14 (αυξημένες πιέσεις πλήρωσης αρ. κοιλίας)
- Διάμετρος και αναπνευστική διακύμανση κάτω κοίλης φλέβας

Table 4 Echocardiography parameters to estimate cardiac pressures

Parameter and findings	Interpretation
For estimation of LAP	
E/A ratio <0.8	Normal or low
Peak E velocity ≤50 cm/s[99]	
E/A ratio ≥2	Elevated LAP
Deceleration time <160 ms (in patients with reduced LVEF)[99]	
Average E/e' <8[99]	Normal or low LAP
Average E/e' >14[99]	Elevated LAP
Increased systolic and diastolic PAP estimated by CW Doppler TR systolic jet velocity	Elevated in approximately 70% of HF cases[157] (match of left and right); can also be encountered in normal or low LAP
E/A ratio >0.8 and <2[99]	Recommended assessment of peak velocity of TR jet by CW Doppler, E/e' ratio, and LA volume index for accurate evaluation
For estimation of RAP	
IVC <2.1 cm that collapses >50% with a sudden inspiratory manoeuvre (i.e. sniff)[158]	Suggests normal RAP of 3 mmHg (range 0–5 mmHg)[158]
IVC diameter >2.1 cm that collapses <50% with a sniff[158]	Suggests high RAP of 15 mmHg (range 10–20 mmHg)[158]
Scenarios in which IVC diameter and collapse do not fit the above paradigms[158]	An intermediate value of 8 mmHg (range 5–10 mmHg) may be used or, preferably, other indices of RAP should be integrated to downgrade or upgrade to the normal or high values of RAP[158]
IVC ≤12 mm (in patients with positive pressure ventilation degree of IVC collapse cannot be used)[159]	RAP <10 mmHg[159]
Hepatic vein flow pattern Vs >Vd (systolic predominance in hepatic vein flow)[158]	Low or normal RAP[160]
Hepatic vein systolic filling fraction (VTIs/VTIs + VTId) <55%[160] or Vs/Vd <1[158] (lost systolic predominance)	Elevated RAP[160]

CVP, central venous pressure; CW, continuous wave; HF, heart failure; IVC, inferior vena cava; LAP, left atrial pressure; LV, left ventricular; LVEF, left ventricular ejection fraction; PAP, pulmonary artery pressure; RAP, right atrial pressure; TR, tricuspid regurgitation; Vd, diastolic wave; Vs, systolic wave; VTI, velocity–time integral.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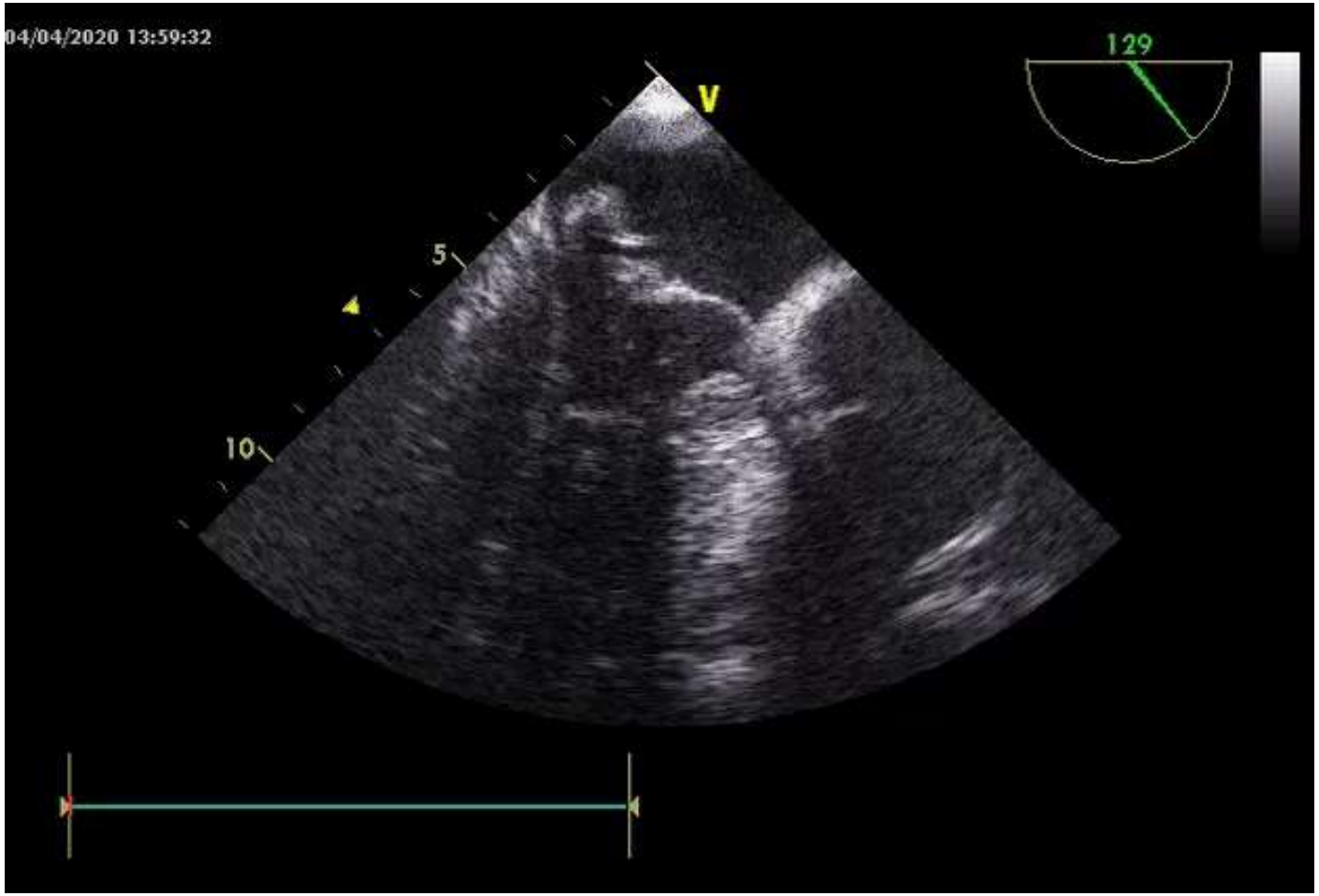
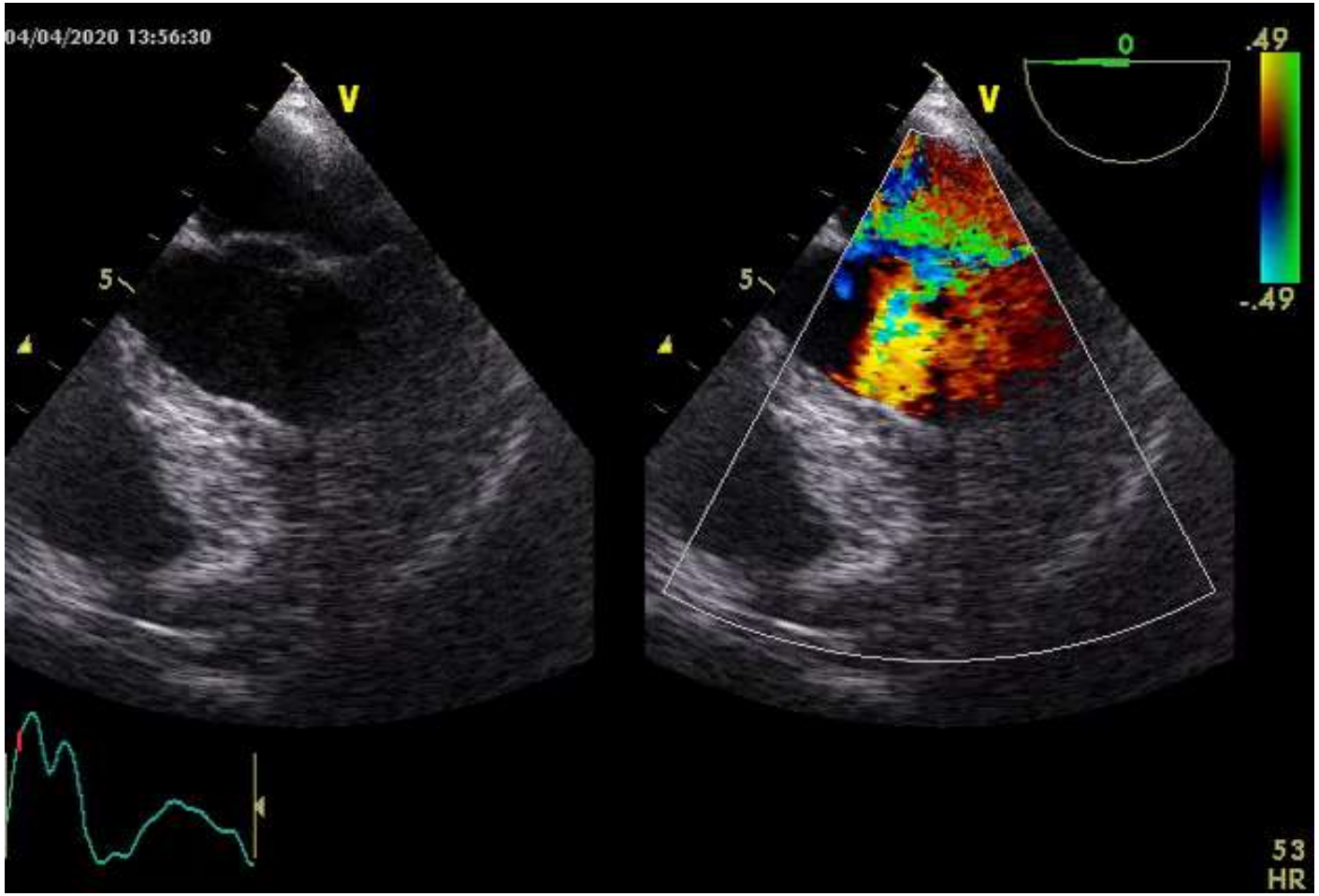
41
V
5
10
68
HR


04/04/2020 13:59:32
129
V
5
10


04/04/2020 13:56:30
V
5
V
5
0
.49
-.49
53
HR

Regarding applied diagnostic measurements

Recommendations	Class	Level
Upon presentation a measurement of plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP) is recommended in all patients with acute dyspnoea and suspected AHF to help in the differentiation of AHF from non-cardiac causes of acute dyspnoea.	I	A
At admission in all patients presenting with suspected AHF, the following diagnostic tests are recommended:		
a. 12-lead ECG;	I	C
b. chest X-ray to assess signs of pulmonary congestion and detect other cardiac or non-cardiac diseases that may cause or contribute to the patient's symptoms;	I	C
c. the following laboratory assessments in the blood: cardiac troponins, BUN (or urea), creatinine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests and TSH.	I	C
Echocardiography is recommended immediately in haemodynamically unstable AHF patients and within 48 hours when cardiac structure and function are either not known or may have changed since previous studies.	I	C

European Heart Journal (2016) **37**, 2129-2200 - doi:10.1093/eurheartj/ehw 128

US πνευμόνων (Lund ultrasound)

- Απεικόνιση B-lines (δείκτες πνευμονικής συμφόρησης) και υπεζωκοτικών συλλογών
- Ανεύρεση τουλάχιστον 3-B lines σε δύο πνευμονικές ζώνες άμφω (σάρωση 8 ζωνών – 4 πρόσθια και 4 πλάγια). Μειώνονται το λιγότερο σε 3 ώρες με θεραπεία καρδιακής ανεπάρκειας-απώλειας υγρών.

Καλύτερη μέθοδος αποκλεισμού διάμεσου-κυψελιδικού πνευμονικού οιδήματος:

1. Chest X-ray
2. Lung ultrasound

Volpicelli G, ElBarbary M, Blaivas M, Lichtenstein DA, Mathis G, Kirkpatrick AW, Melniker L, Gargani L, Noble VE, Via G, Dean A, Tsung JW, Soldati G, Copetti R, Bouhemad B, Reissig A, Agricola E, Rouby JJ, Arbelot C, Liteplo A, Sargsyan A, Silva F, Hoppmann R, Breitkreutz R, Seibel A, Neri L, Storti E, Petrovic T. International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med* 2012;38:577–591.

Al Deeb M, Barbic S, Featherstone R, Dankoff J, Barbic D. Point-of-care ultrasonography for the diagnosis of acute cardiogenic pulmonary edema in patients presenting with acute dyspnea: a systematic review and meta-analysis. *Acad Emerg Med* 2014;21:843–852.

Διαφοροδιάγνωση καρδιακής-εξωκαρδιακής αιτιολογίας οξείας δύσπνοιας στο ΤΕΠ

Pivetta E, Goffi A, Lupia E, Tizzani M, Porrino G, Ferreri E, Volpicelli G, Balzaretti P, Banderali A, Iacobucci A, Locatelli S, Casoli G, Stone MB, Maule MM, Baldi I, Merletti F, Cibinel GA, Baron P, Battista S, Buonafede G, Busso V, Conterno A, Del Rizzo P, Ferrera P, Pecetto PF, Moiraghi C, Morello F, Steri F, Ciccone G, Calasso C, Caserta MA, Civita M, Condo C, D'Alessandro V, Del Colle S, Ferrero S, Griot G, Laurita E, Lazzero A, Lo Curto F, Michelazzo M, Nicosia V, Palmari N, Ricchiardi A, Rolfo A, Rostagno R, Bar F, Boero E, Frascisco M, Micossi I, Mussa A, Stefanone V, Agricola R, Cordero G, Corradi F, Runzo C, Soragna A, Sciullo D, Vercillo D, Allione A, Artana N, Corsini F, Dutto L, Lauria G, Morgillo T, Tartaglino B, Bergandi D, Cassetta I, Masera C, Garrone M, Ghiselli G, Ausiello L, Barutta L, Bernardi E, Bono A, Forno D, Lamorte A, Lison D, Lorenzati B, Maggio E, Masi I, Maggiorotto M, Novelli G, Panero F, Perotto M, Ravazzoli M, Saglio E, Soardo F, Tizzani A, Tizzani P, Tullio M, Ulla M, Romagnoli E; SIMEU Group for Lung Ultrasound in the Emergency Department in Piedmont. Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED: a SIMEU multicenter study. *Chest* 2015;**148**:202–210.

Χρήσιμος ο συνδυασμός lung ultrasound και NPs στην άμεση εκτίμηση ασθενών με δύσπνοια στο ΤΕΠ

Liteplo AS, Marill KA, Villen T, Miller RM, Murray AF, Croft PE, Capp R, Noble VE. Emergency thoracic ultrasound in the differentiation of the etiology of shortness of breath (ETUDES): sonographic B-lines and N-terminal pro-brain-type natriuretic peptide in diagnosing congestive heart failure. *Acad Emerg Med* 2009;**16**:201–210.

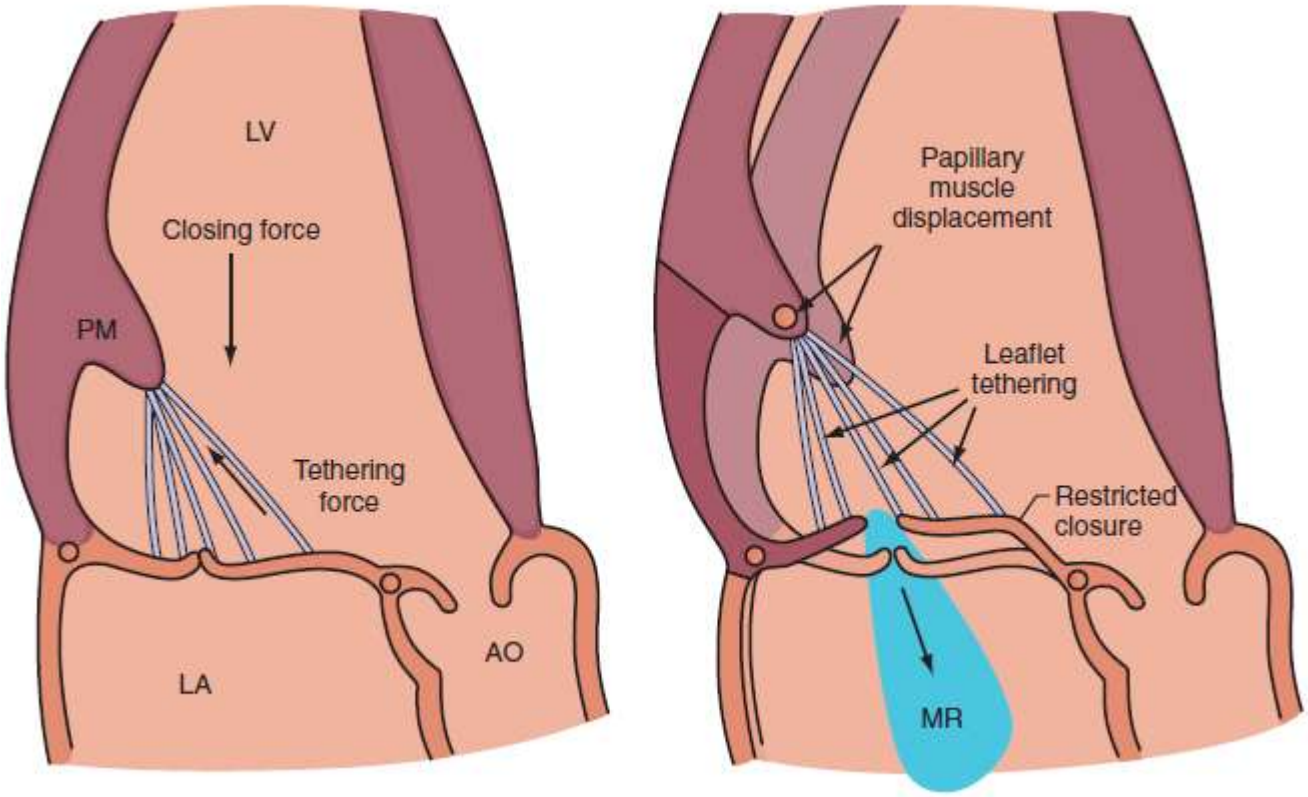
Acute mitral regurgitation

Ischemic acute MR

Nonischemic acute MR

Ischemic MR

Papillary muscle rupture due to acute myocardial infarction or
papillary muscle displacement due to myocardial infarction or ischemia


LV
Closing force
PM
Tethering force
LA
AO
Papillary muscle displacement
Leaflet tethering
Restricted closure
MR

Nonischemic MR

Ruptured mitral chordae tendineae (flail leaflet) due to:

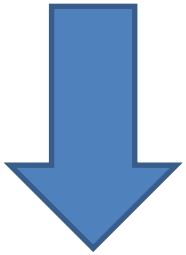
myxomatous disease (mitral valve prolapse),
infective endocarditis,
trauma,
rheumatic heart disease (acute rheumatic fever or chronic rheumatic mitral valve disease), or
spontaneous rupture.

Left atrium is not compliant

increase in left atrial volume

elevation in pressure within the left atrium

elevation in pressure into the pulmonary circulation



pulmonary edema

effective forward flow is limited

forward cardiac output falls

hypotension - cardiogenic shock

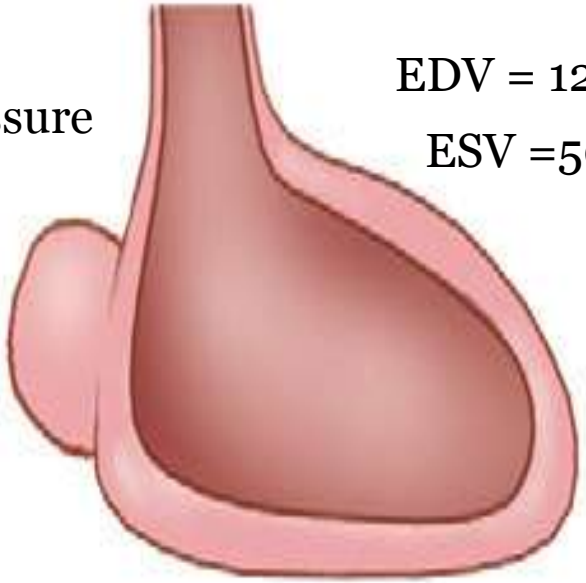
neurohormonal activation increases SVR  worsens the severity of mitral regurgitation

Normal

LA pressure 10

EDV = 120

ESV =50

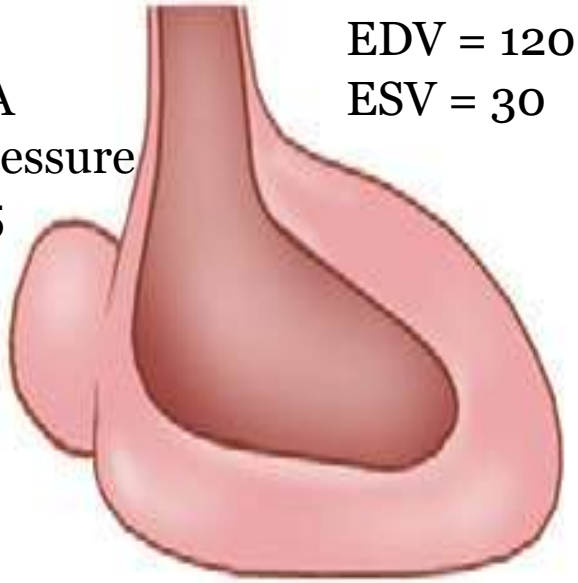


TSV = 70
FSV = 70
RSV = 0
EF = 0.58

Acute MR

EDV = 120
ESV = 30

LA pressure 25



TSV = 90
FSV = 45
RSV = 45
EF = 0.75

In acute MR, an increase in preload and a decrease in afterload cause an increase in end diastolic volume (*EDV) and a decrease in end-systolic volume (ESV), producing an* increase in total stroke volume (*TSV). However, forward stroke volume* (*FSV) is diminished because 50% of the TSV regurgitates as the regurgitant* stroke volume (*RSV), resulting in an increase in LA pressure.*

CLINICAL MANIFESTATIONS

- sudden onset and rapid progression
- **pulmonary edema**
- **hypotension,** and signs and symptoms of **cardiogenic shock**
- **acute right-sided heart failure**
- history mimics an acute pulmonary process (such as infection or acute respiratory distress syndrome)

Physical examination

- pulmonary edema
- evidence of poor tissue perfusion with peripheral vasoconstriction, pallor, and diaphoresis
- arterial pulse is often rapid and of low amplitude
- the neck veins become distended in right-sided pressure with a marked "v" wave
- cardiac apical impulse is hyperdynamic but is usually normal in location

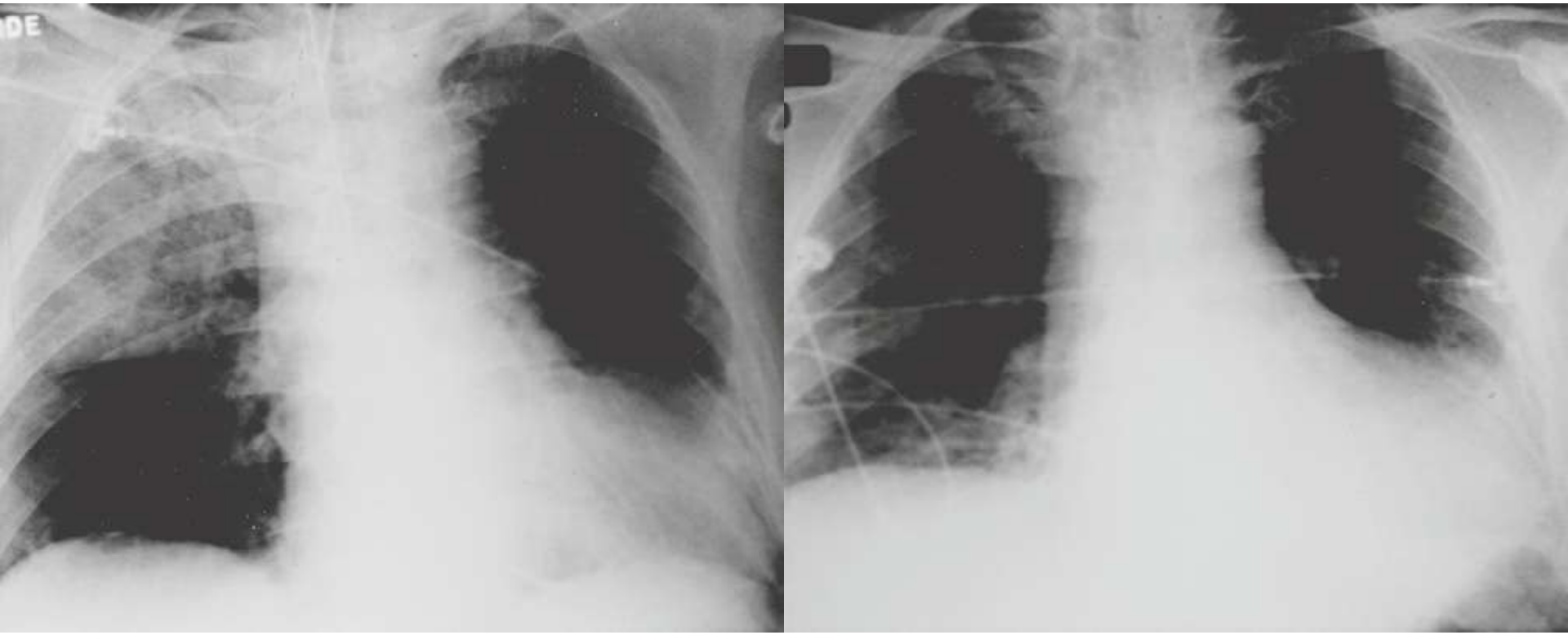
- **Cardiac auscultation**

The murmur of acute MR may be early systolic, midsystolic, or holosystolic.

Early termination of the murmur in acute mitral regurgitation results from rapid equalization of LA and LV pressures and suggests a greater degree of regurgitation

ECG

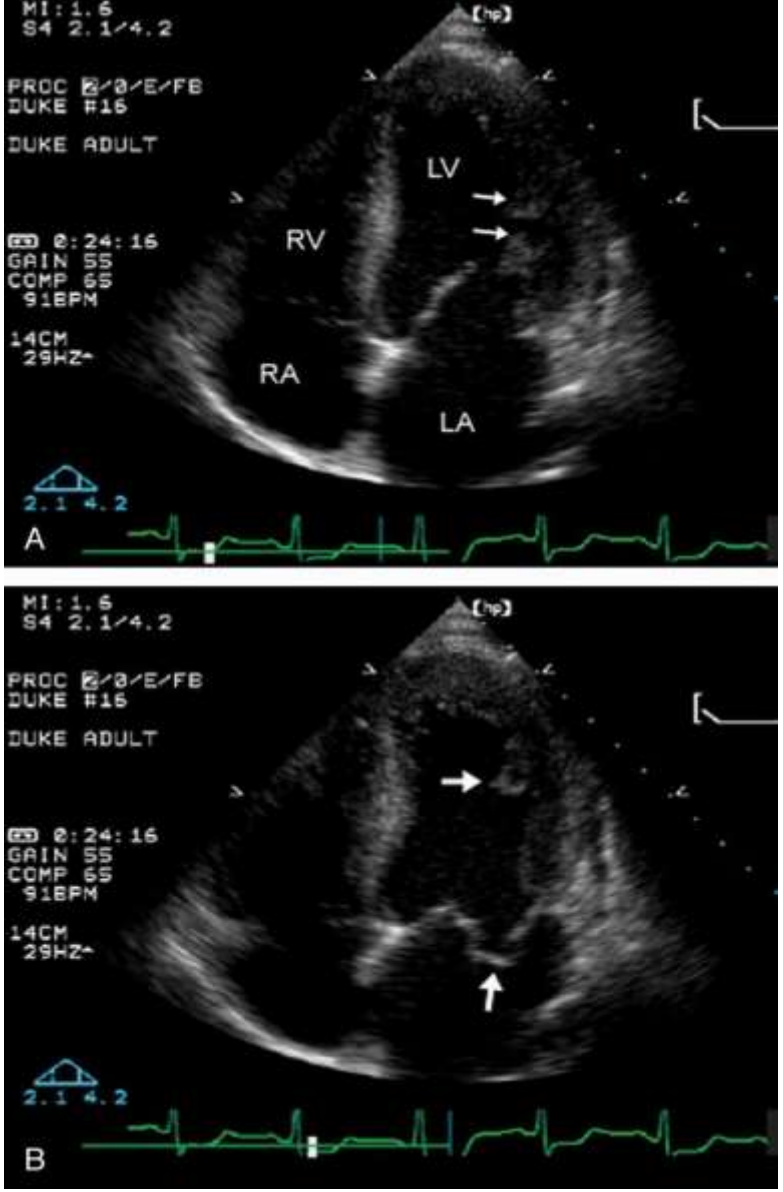
- sinus tachycardia
- negative deflection of the P wave in lead V1
- Nonspecific ST segment and T wave abnormalities
- Ischemia or infarction



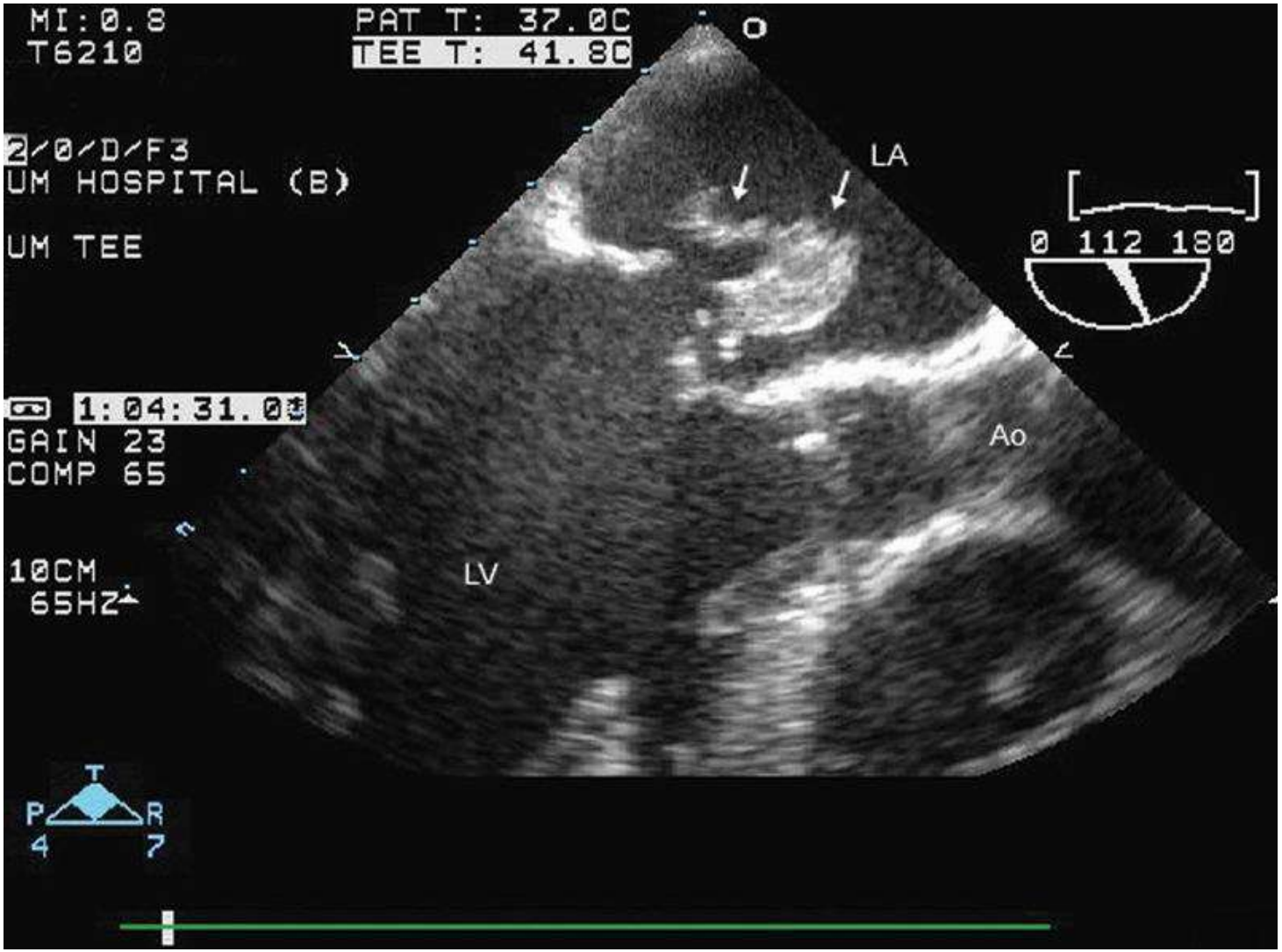
Unusual radiographic appearance of acute mitral regurgitation mimicking lobar pneumonia.
A, Prominent right upper lobe alveolar infiltrate.
B, Rapid resolution occurred in 48 hours with diuretic therapy.

Ischemic acute MR

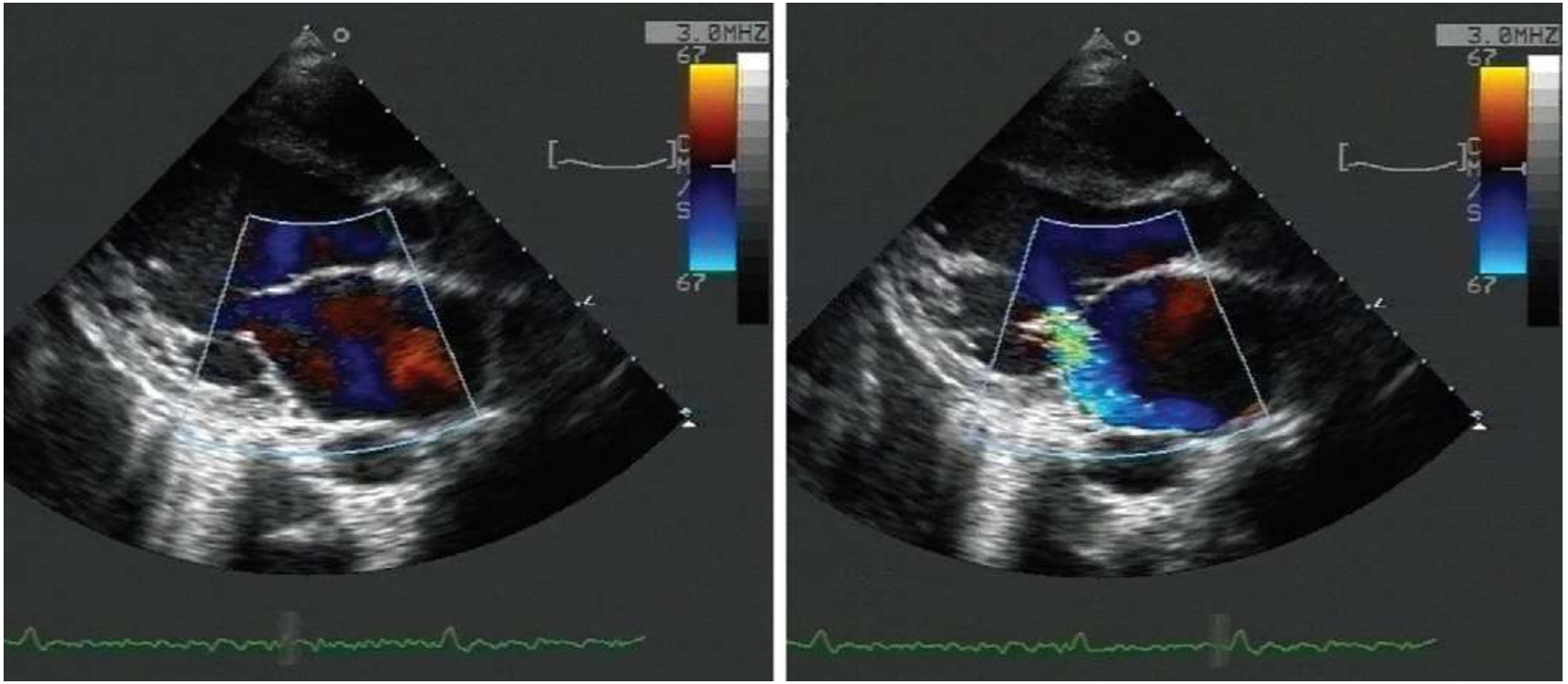
(Valve Morphology-2D)



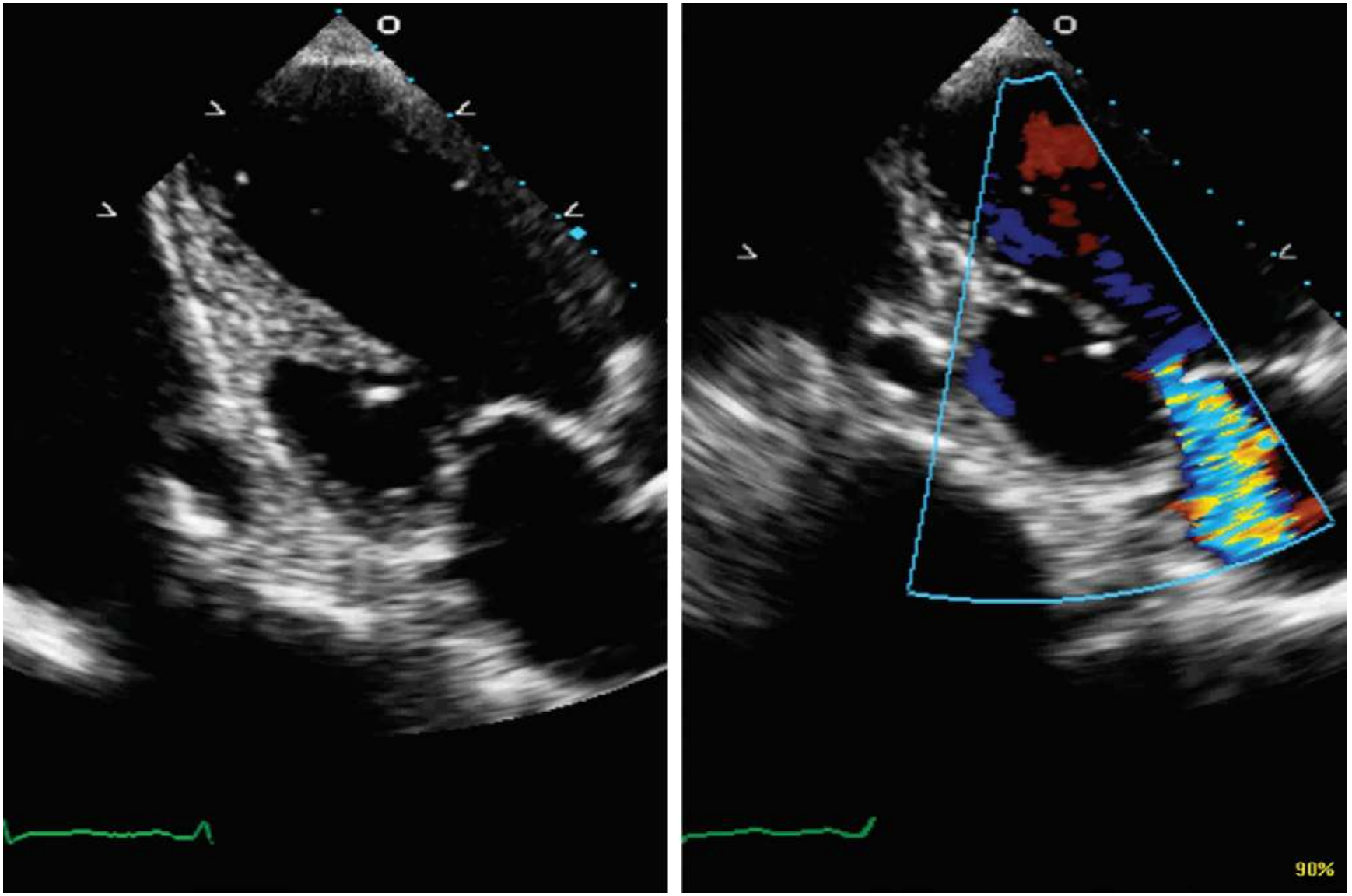
Apical four-chamber view A: Note the break in the continuity of the posterolateral papillary muscle with two portions of the papillary muscle head (arrows). B: Recorded in systole. Note the marked buckling of the mitral valve leaflet into the left atrium (LA) (upward-pointing arrow) and the ruptured papillary muscle base in the cavity of the left ventricle (horizontal arrow). LV, ventricle; RA, right atrium; RV, right ventricle.



Transesophageal echocardiogram recorded in a longitudinal plane in a patient with inferior myocardial infarction and acute severe mitral regurgitation. This frame was recorded in systole, and a large portion of the papillary muscle head can be seen prolapsing into the left atrium (LA) (arrows). Ao, aorta; LV, left ventricle.



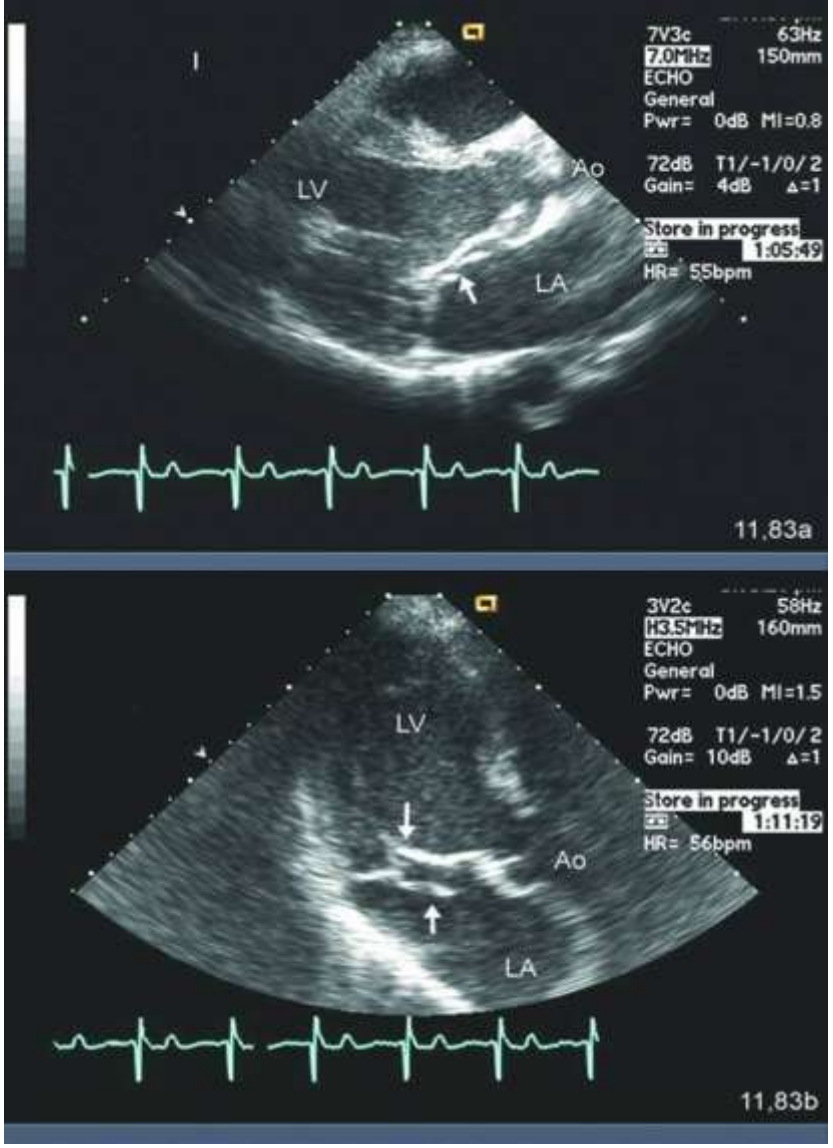
Ischemic mitral regurgitation in patient with inferolateral myocardial infarction. Note normal thickening in systole of anterior interventricular septum compared to lack of thickening of akinetic inferolateral wall (systolic frame at *right compared with end-diastolic frame at left), with resultant posteriorly directed jet of mitral regurgitation.*



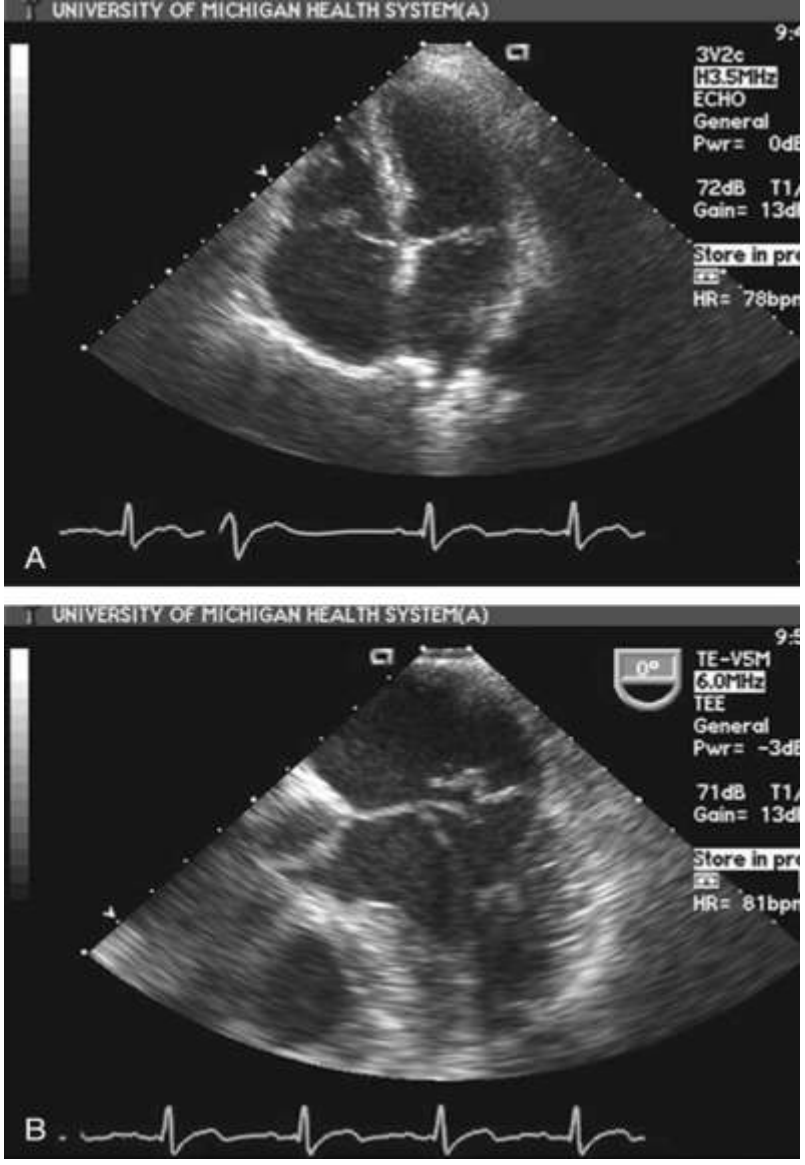
Left panel shows aneurysm of inferior wall from infarction, which results in distortion of the LV wall leading to mitral leaflet tethering and restricted closure with development of mitral regurgitation (*right panel)* .

Nonischemic acute MR

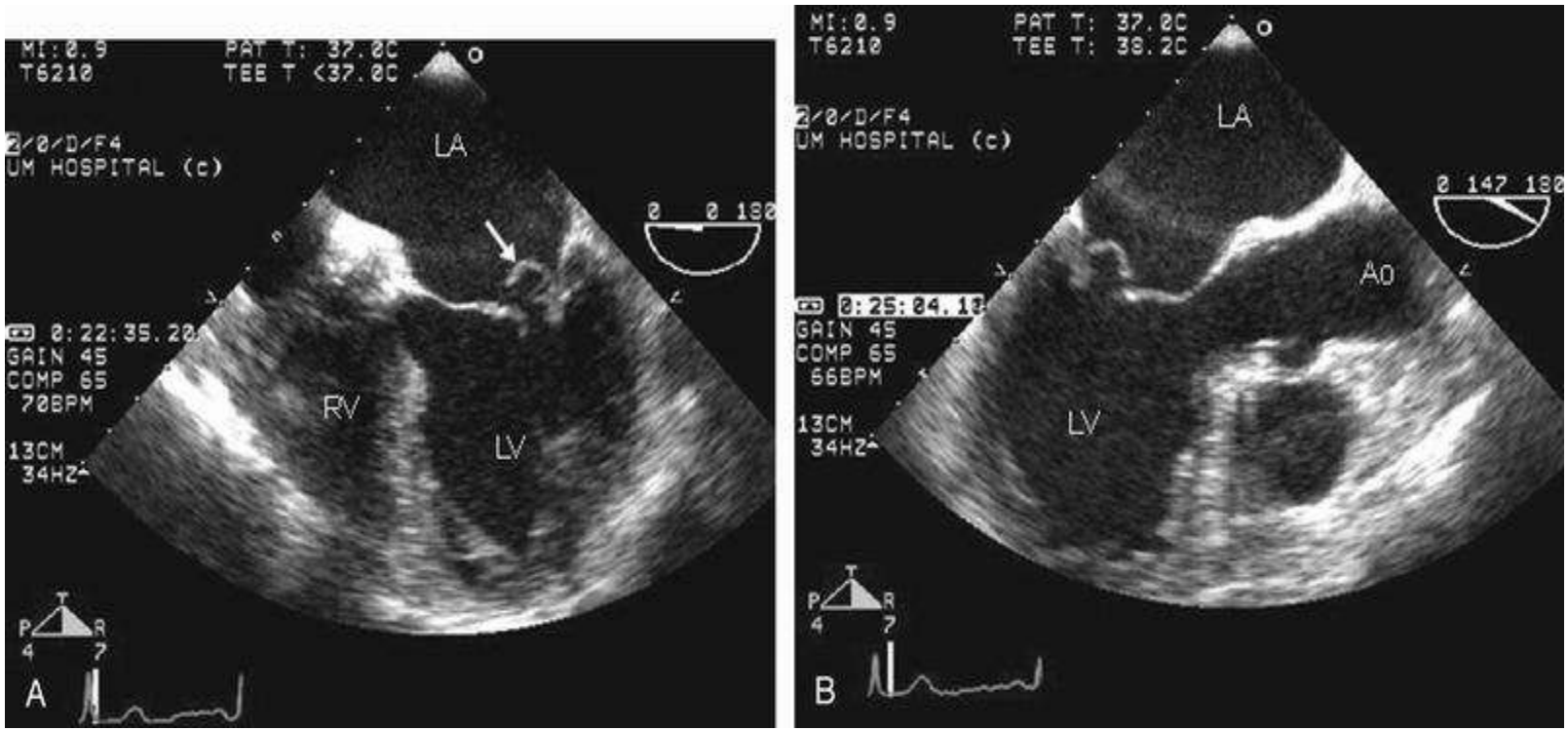
(Valve Morphology-2D)



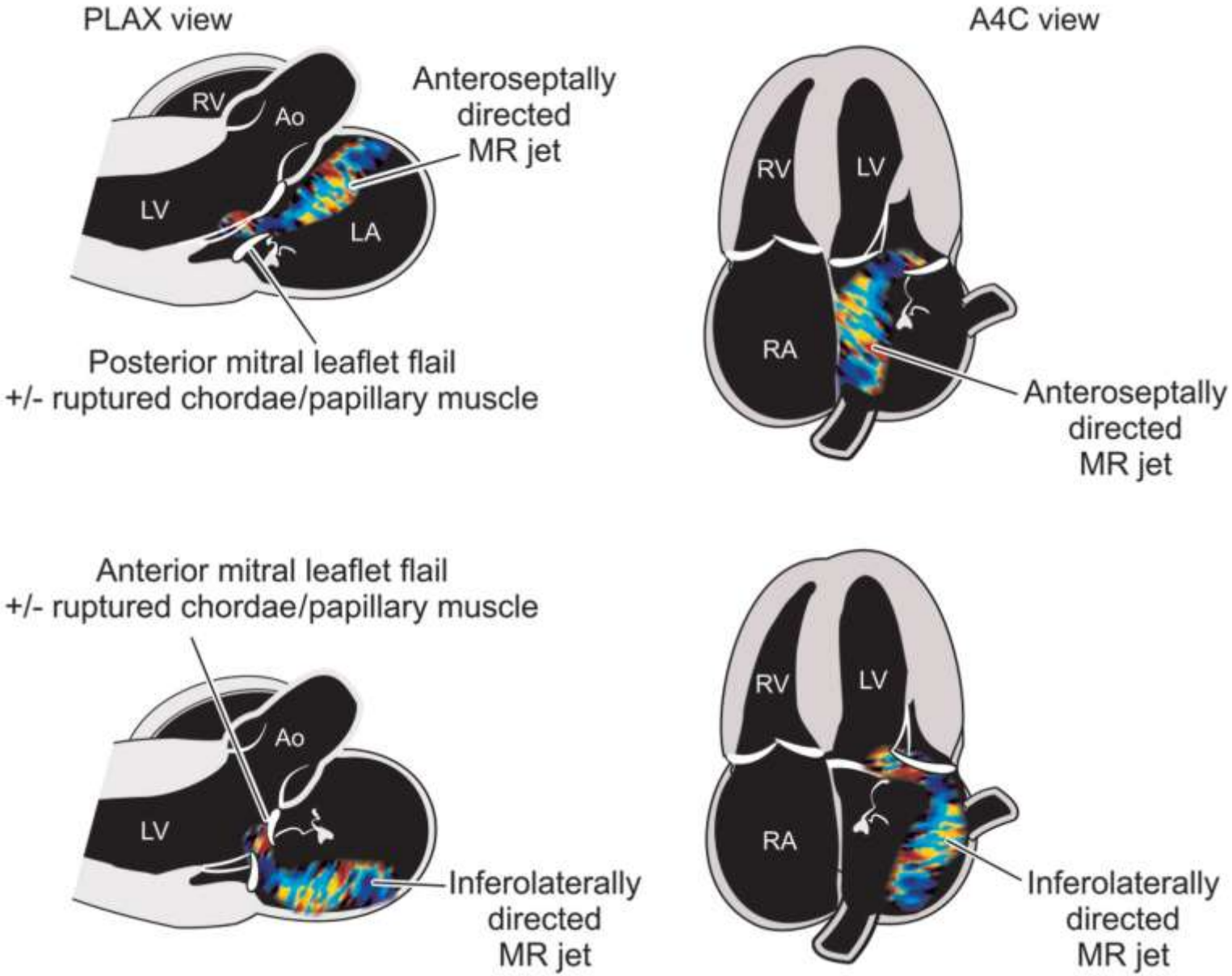
Transthoracic echocardiogram recorded in parasternal long-axis and apical views in a patient with a flail posterior leaflet A: Note that the posterior leaflet and attached chordae are positioned behind the anterior leaflet (arrow), indicative of disruption of the anatomic attachment of the posterior leaflet. B: Recorded from apical transducer position, this phenomenon is better visualized. The downward-pointing arrow denotes the normally closed anterior leaflet and the upward-pointing arrow indicates the flail posterior leaflet. Ao, aorta; LA, left atrium; LV, left ventricle.

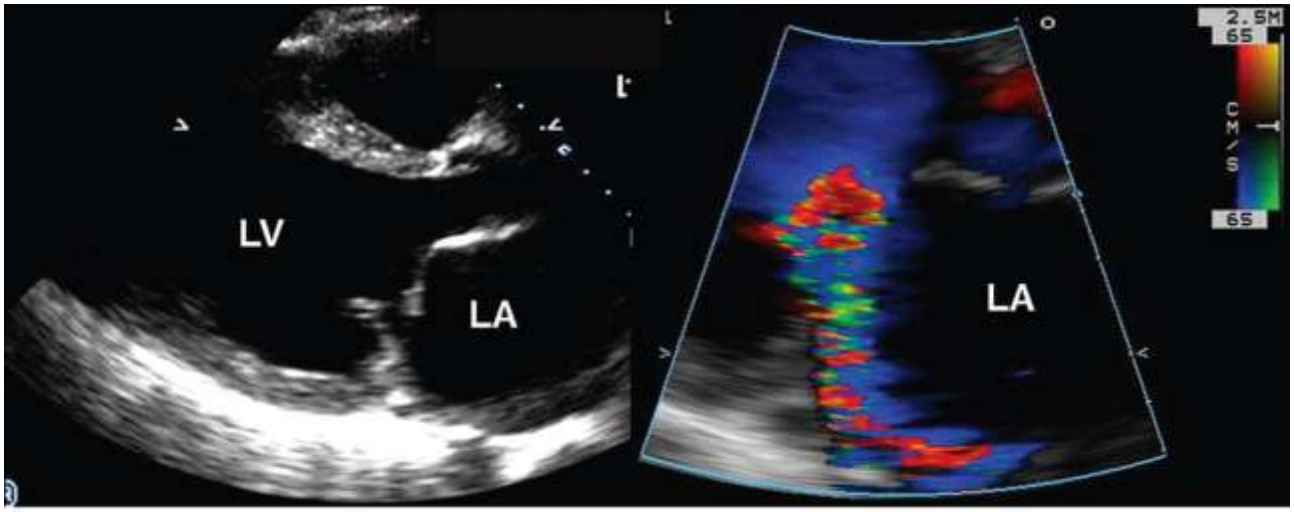


Transthoracic (A) and transesophageal (B) echocardiograms recorded in a patient with a flail posterior mitral valve leaflet. A: Note the distinct buckling of the posterior leaflet, the tip of which points away from the left ventricular apex. B: This phenomenon is more clearly demonstrated when recorded from the transesophageal approach in the same patient.



Transesophageal echocardiogram recorded in a transverse plane (A) and longitudinal plane (B) in a patient with a flail posterior scallop (P2) of the mitral valve. A: Note the portion of the posterior leaflet of the mitral valve curling behind the anterior leaflet in systole (arrow). B: A similar phenomenon is noted recorded at 147 degrees. In both of these planes, the posterior scallop (P2) is at the mid portion of the mitral valve (Fig. 11.4). RV, right ventricle; Ao, aorta; LA, left atrium; LV, left ventricle.


PLAX view
RV
Ao
LV
LA
Anteroseptally directed MR jet
Posterior mitral leaflet flail
+/- ruptured chordae/papillary muscle
A4C view
RV
LV
RA
Anteroseptally directed MR jet
Anterior mitral leaflet flail
+/- ruptured chordae/papillary muscle
Ao
LV
Inferolaterally directed MR jet
RV
LV
RA
Inferolaterally directed MR jet


LV
LA
LA
2.5M
65
CM\S
65

Treatment

- focus on

  reducing LVEDP,

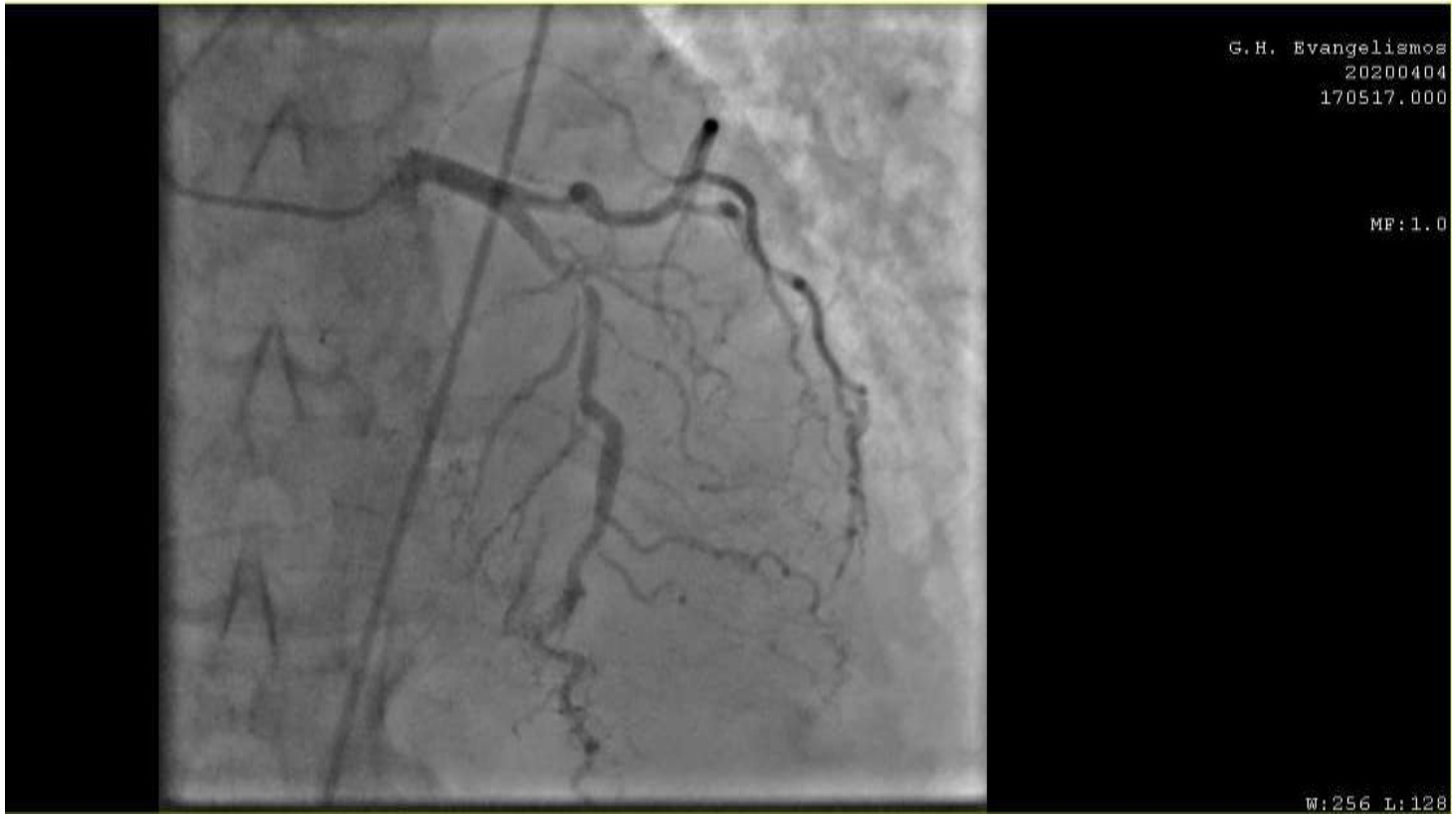
  decreasing aortic impedance to LV ejection (so that blood flow can be directed in a forward rather than retrograde direction),

  initiating specific therapy for the precipitating
  etiology

- Vasodilator therapy is the key component of medical management, and the preferred agent is intravenous **nitroprusside**
- An additional degree of afterload reduction may be provided by placing an **IABP**
- If there is significant hypotension, **dopamine**
- If LV contractility is impaired and cardiac output is significantly reduced, the addition of **dobutamine**
- **Diuretics** are useful to reduce pulmonary congestion.

- **The criteria for ICU/CCU admission include any of the following:**
  - need for intubation (or already intubated)
  - signs/symptoms of hypoperfusion
  - oxygen saturation (SpO2) <90% (despite supplemental oxygen)
  - use of accessory muscles for breathing, respiratory rate >25/min
  - heart rate <40 or >130 bpm, SBP <90 mmHg

Mebazaa A, Yilmaz MB, Levy P, Ponikowski P, Peacock WF, Laribi S, Ristic AD, Lambrinou E, Masip J, Riley JP, McDonagh T, Mueller C, DeFilippi C, Harjola V-P, Thiele H, Piepoli MF, Metra M, Maggioni A, McMurray J, Dickstein K, Damman K, Seferovic PM, Ruschitzka F, Leite-Moreira AF, Bellou A, Anker SD, Filippatos G. Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the HFA of the ESC, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine. *Eur J Heart Fail* 2015;**17**:544–558.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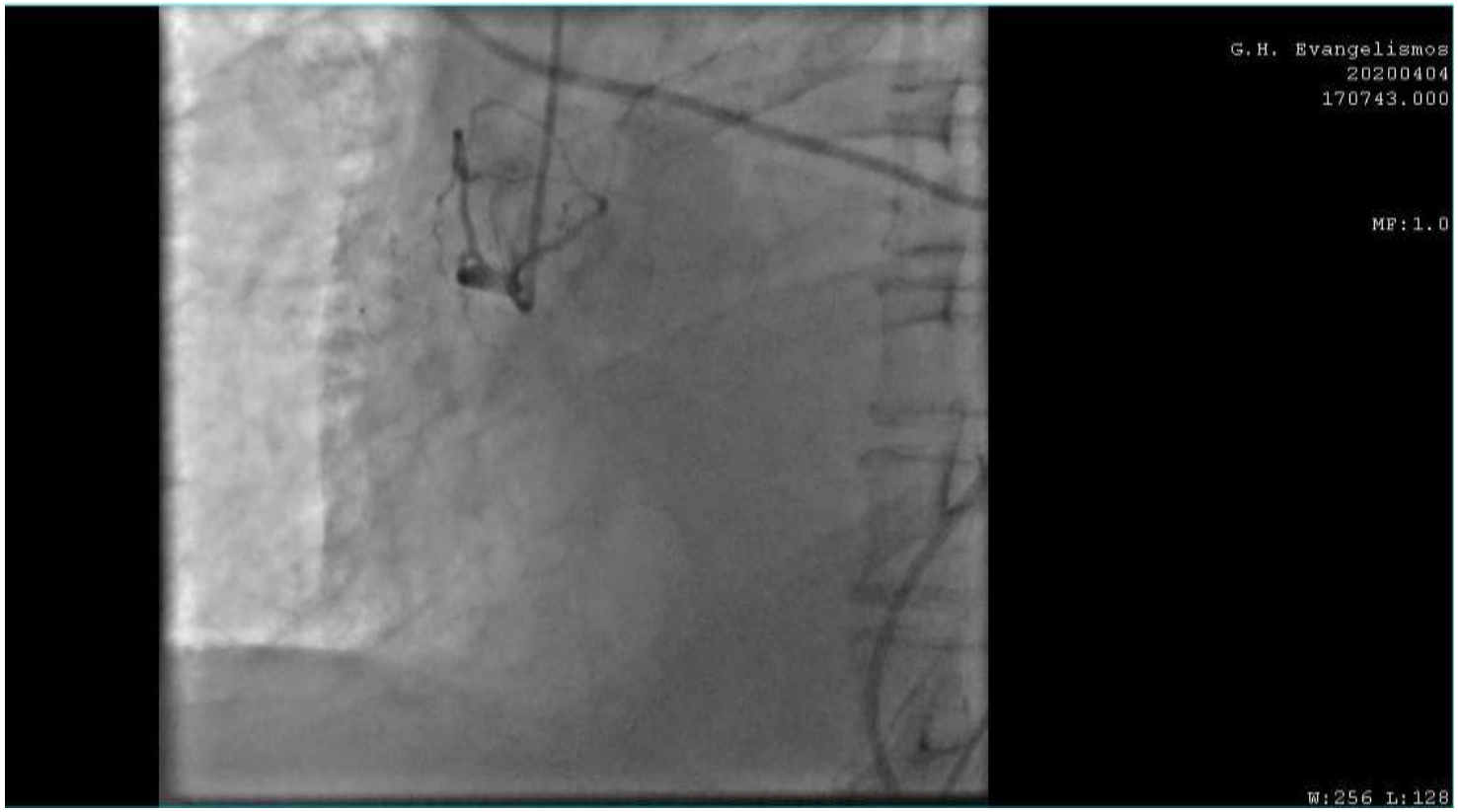
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