

EVERY DAY CASE

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History

55 female patient ,obese with a history of hypertension 10 years ago and type II diabetes 5 years ago, presented to ER with exertional dyspnea for 2 weeks progressed to SOB at rest with orthopnea , PND with no chest pain .

Examination

❖ Vital data →

- Temperature: 36.5 °C
- Blood pressure 180/90
- pulse 95 regular equal in both sides
- Respiratory rate: 22/min
- Oxygen saturation while breathing in ambient air 90 %

Physical Examination

- General : alert, awake, and oriented but slightly agitated
- Neck : slight jugular venous distention, no lymphadenopathy, and no carotid murmur
- Cardiovascular : S1,S2 , S4 no murmurs

Physical Examination (Cont.)

- Lungs : breath sounds diffusely decreased, in particular at the lung bases, and rales up to medium shots bilaterally.
- Abdomen : plain, no pulsatile masses, normal bowel sounds in all four quadrants, no High pitched or tinkling sounds, resonant to percussion , soft, non distended , non tender, no rebound or guarding, no hepatomegaly costal margin, no splenomegaly.
- Neurological : negative cerebellar test, cranial nerve intact, no focal deficit.

Routine Laboratory Tests

Complete blood count :

leukocytosis with neutrophilia (WBC 10.760/mm³, 91.20 % neutrophils), hemoglobin 13.5 g/dl, and platelets 248,000/ mm³

Inflammatory markers : ESR 29 mm/h and

CRP 0.6 mg/dl

Hepatic function :

SGOT normal, SGPT with slight increase (61 U/l), γ -GT 121 U/l, and

ALP, total bilirubin (direct and indirect), and coagulation : normal

ABG : performed in ambient air showed pH = 7.41, pO₂ = 58 mmHg , pCO₂ = 40 mmHg

Normal renal function, creatinine 0.82 mg/dl, BUN 38 mg/dl, eGFR 92.8 ml/min/1.73 m²)

Electrolytes(Na⁺ , K⁺ , Ca⁺⁺ , Mg⁺⁺ , Cl⁻) : normal

Fasting blood glucose: 179 mg/dl

Myocardial necrosis markers : normal CK-MB and Hs-Tnl 0.059 ng/ml (n.v. 0–0.055)

BNP : 744 pg/ml

Thyroid function: normal TSH , (ft₄ and ft₃) 2.10 pg/ml

(n.v. 2.2–4.2 pg/ml)

D-dimer : negative

Chest X-Ray :

X-ray showed cardiomegaly with signs compatible with lung congestion.

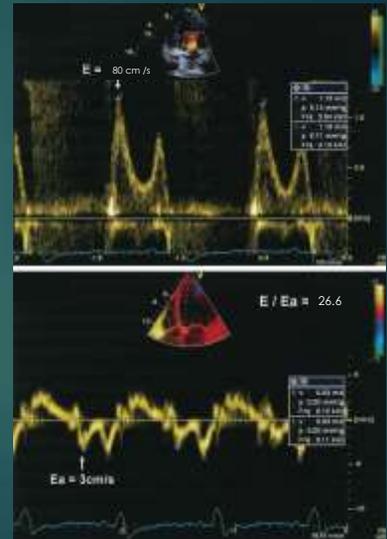
EKG : NSR with insignificant changes

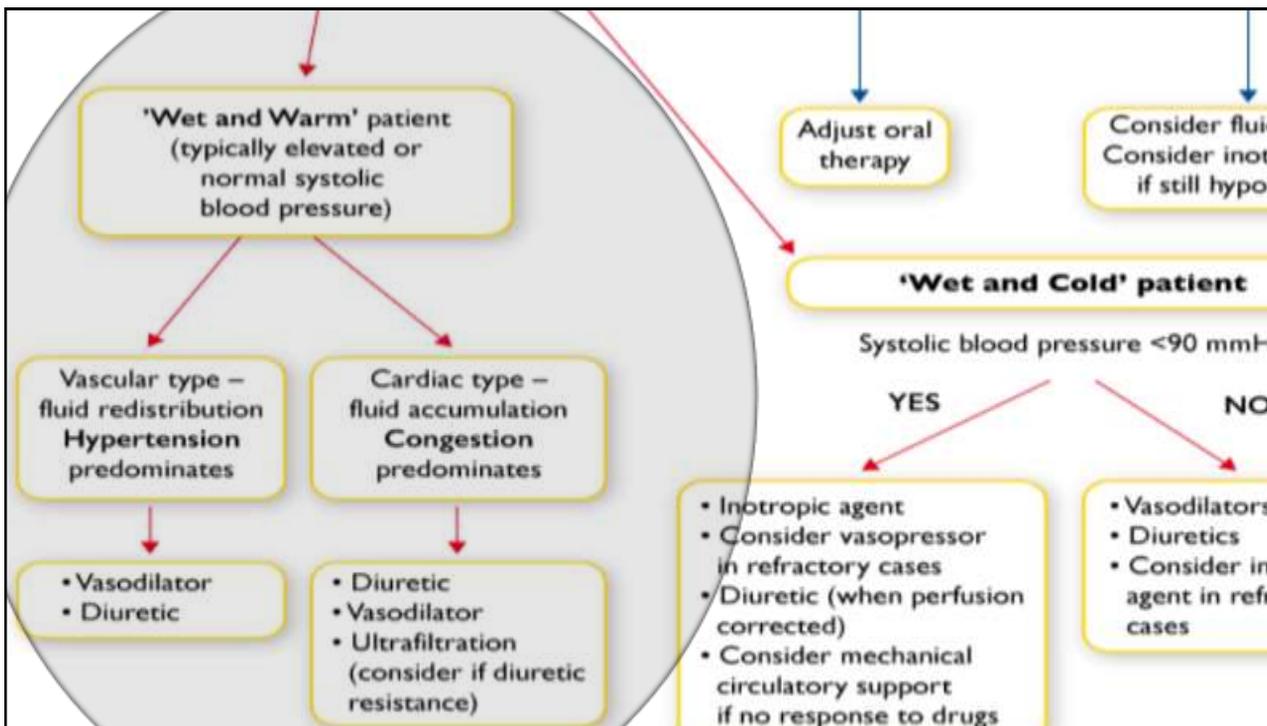
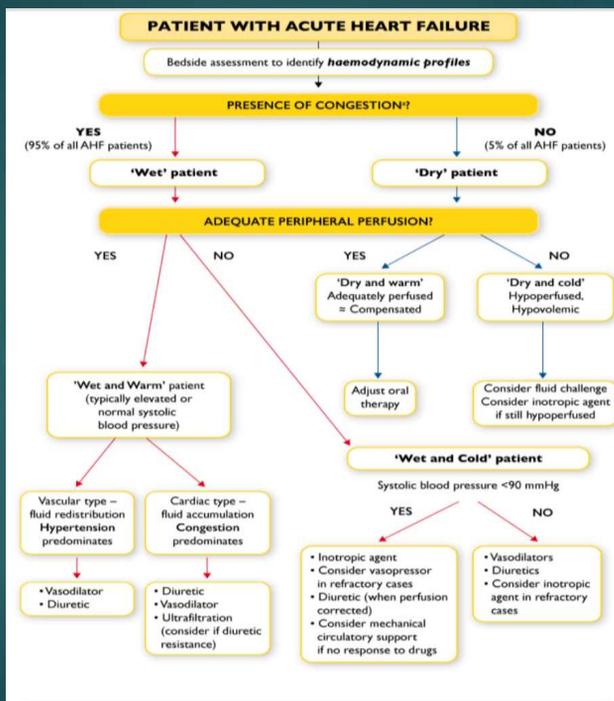
Possible Causes of Worsening Dyspnea

- ▶ Acute myocardial infarction
- ▶ Hypertensive emergency
- ▶ Arrhythmias
- ▶ Acute myopericarditis
- ▶ Lung diseases (bacterial pneumonia)
- ▶ ARDS
- ▶ Pulmonary embolism
- ▶ Valvular disease (acute mitral regurgitation)

Echocardiography

- Normal lv internal dimensions with normal LV systolic function (**EF 65 %**)
- No SWMA
- Concentric LVH**
- Dilated left atrium**
- Abnormal LV relaxation pattern (grade 3)**
- Normal mitral valve with mild MR
- Normal RT side
- No pericardial effusion





Goals of treatment in Acute heart failure

Immediate (ED/ICU/CCU)

Improve haemodynamics and organ perfusion.

Restore oxygenation.

Alleviate symptoms.

Limit cardiac and renal damage.

Prevent thrombo-embolism.

Minimize [ICU](#) length of stay.

Intermediate (in hospital)

Identify aetiology and relevant co-morbidities.

Titrate therapy to control symptoms and congestion and optimize blood pressure.

Initiate and up-titrate disease-modifying pharmacological therapy.

Consider device therapy in appropriate patients.

Initial Management

▶ The patient was then treated with [furosemide bolus and infusion of nitroglycerin](#) to reduce high blood pressure initially encountered.

▶ [A CPAP](#) (continuous positive airway pressure) was positioned and was set FiO₂ of 50 % and PEEP (positive end-expiratory pressure) of 10 cmH₂O.

The patient showed : -

marked improvement of dyspnea, and blood gas analysis showed a significant increase in pO₂ (pO₂ = 58 mmHg → 139 mmHg).

[This favorable response to treatment could make us exclude a non-cardiogenic acute pulmonary edema \(ARDS\).](#)

which is characterized by severe hypoxemia refractory to increased FiO₂ and reduced lung compliance.

Coronary Angiography

- ❑ An invasive coronary angiography documented the absence of hemodynamically significant stenosis .

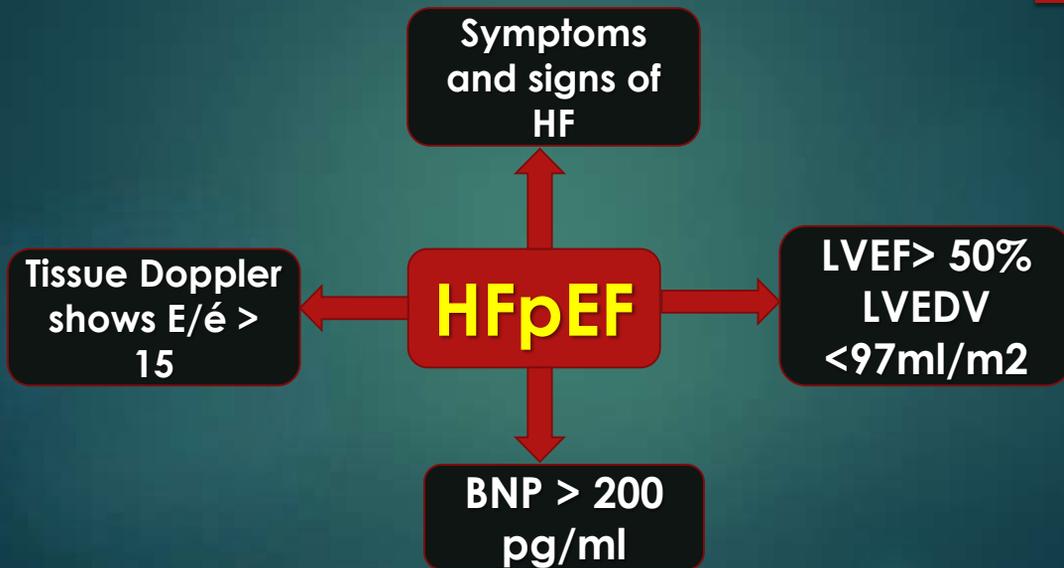
According to these data : -

- ▶ Acute myocardial infarction
- ▶ Hypertensive emergency
- ▶ Arrhythmias
- ▶ Acute myopericarditis
- ▶ Lung diseases (bacterial pneumonia)
- ▶ ARDS
- ▶ Pulmonary embolism
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Associated
with

HFpEF

According to guidelines



HFpEF results from abnormalities of active & passive ventricular relaxation leading to decrease SV & COP.

High ventricular filling pressure stimulates release of ANP, BNP to inhibit RAS system with diuresis effects

BNP has protective effects on myocardium [prevent remodeling & fibrosis]

elevated levels of BNP correlated with more increased LV filling P&L V dysfunction

The prognosis is comparable with HFrEF & worsened by high level of BNP, old age, MI, DM

AHA Guidelines 2017 update

AHA Guidelines 2017 focused mainly on

- 1 Biomarkers [BNP]
- 2 Uses of aldosterone antagonists
- 3 anemia
- 4 Hypertension management
- 5 Biomarkers of myocardial injury

Biomarkers NP

Prevention Utilizing NP as screening for those at risk for developing HF to optimizing medical therapy to prevent LV dysfunction

[class II a –level of evidence B]

Diagnosis Measurement of NP for patients presented by dyspnea to support the diagnosis of HF or exclusion

[class I level of evidence A]

Prognosis Measurement of NP for establishing the prognosis ,severity of HF

[class I level of evidence A]

Pre discharge Measurement of NP during hospitalization for post discharge prognosis

[class IIa level of evidence B]

Measurement biomarkers of myocardial injury in patients with chronic HF for additive risk stratification

[class IIa level of evidence B]

Aldosterone Antagonists

Uses of aldosterone antagonists in selected patients with HFpEF [EF >45% ,elevated NP ,GFR>30,Cr <2.5,serum K>5meq/L]for decrease hospitalization

[class IIa level of evidence B]

Routine uses of nitrates or phosphodiesterase inhibitors for increase QoL

[class III level of evidence B] [No benefit]

Routine uses of nutritional supplements

[class III level of evidence B][No benefit]

Anemia

I V iron in patients with NYHA CLASS II-III with serum ferritin <100ng/ml or 100-300ng/ml with transferrin<20% to improve QoL

[class IIb level of evidence B]

Erythropoietin stimulating agents should not be used to improve morbidity and mortality

[class III level of evidence B]

Hypertension

Optimal treatment of hypertension targeting B/P >130/80 mmhg in those patients with increased risk of AHF

[class I level of evidence B]

Titration of GDMT to attain SBP>130mmhg

[class I level of evidence C]

Titration of GDMT to attain SBP>130mmhg in patients HFpEF after treating volume overload

[class I level of evidence C]

CHRONIC MANAGEMENT

Recommendations for treatment of patients with heart failure with preserved ejection fraction and heart failure with mid-range ejection fraction

Recommendations	Class ^a	Level ^b
It is recommended to screen patients with HFpEF or HFmrEF for both cardiovascular and non-cardiovascular co-morbidities, which, if present, should be treated provided safe and effective interventions exist to improve symptoms, well-being and/or prognosis.	I	C
Diuretics are recommended in congested patients with HFpEF or HFmrEF in order to alleviate symptoms and signs.	I	B

Take



Home

Diastolic heart failure is common , especially in elderly ,hypertensive , females

Symptoms are similar to systolic heart failure.

Impaired LV relaxation results in stiffness of the left ventricle.

BNP , ECG ,chest X ray and transthoracic ECHO are simple initial investigations .

Treatment consists of diuretics and spironolactone as well as maintaining sinus rhythm and good blood pressure control .

Thank You