



ESIM 3rd Winter School
Saas – Fee, Switzerland

January 20.-26., 2013.

Case Presentation

And a Mini Lecture

~ Old thing in the new suit ~

Are we ready for the new fashion change?

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Intensive Care Unit – Emergency Centre
Clinic for Gastroenterology
Clinical Centre of Serbia
Belgrade, Republic of Serbia



Presenting Patient :

□ Patient:

Male, 46 yrs., government employee, married, two small children;

- Fever (high body temperature → 37.6C) with cough;
- Without chills and rigors;
- Shortness of breath **when physically active**;
- Swollen legs;

Conversely better when lying down (recumbent)

□ 7 years ago he was diagnosed with '**Cryptogenic liver cirrhosis**';

- All the necessary workup has been done!
- No definitive 'causal' factor has been determined;
- Hepatic biopsy – 'Liver cirrhosis of unknown etiology';

□ In 1999, he had a car accident – referred to E.R. department with further diagnostics excluding 'serious injuries and trauma';

Presenting complaints started nine days ago with worsening;

□ No other medications except those prescribed by his hepatologist; Rx : Hepatoprotective agents (EMA approved), Vitamins, Regular follow ups;

Examination :

- ✓ Revealed bilateral leg edema;
- ✓ Positive hepatojugular reflex;

Auscultation (Coughing):

- ✓ Bronchitis /Wheezing/Bronchial breathing/;

Ultrasound :

- Hepatomegaly (Hepatic veins congested);
- Splenomegaly 140x80mm with laminar fluid;

Chest X-ray: Suspected small patchy areas of consolidation of left lobe middle area. Right heart enlargement.
Dg. Bronchitis Vs Bronchopneumonia

Sputum culture – after cultivating yielded no pathological findings;

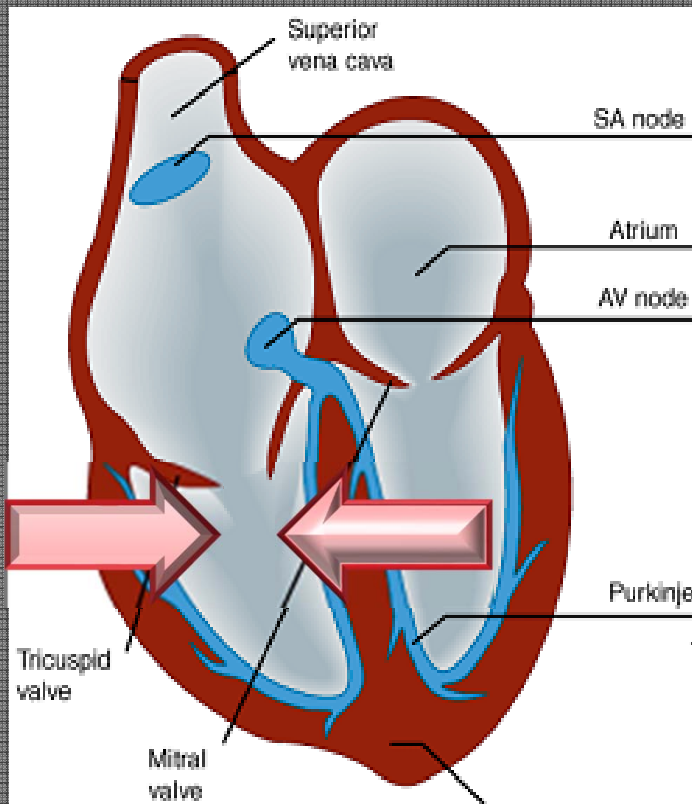
Lab. work-up :
Leucocytosis 16×10^9 (Ly \uparrow); **Plt 90**;
ESR 43; Fibrinogen 3.2;
CRP 21 (<8); **BNP taken!**
AST 56 u/l ALT 44 u/l
Albumin 30g/l PT 70% INR 1.3
Blood cultures 3x negative;
Urine & culture 2x negative;

BNP/pro=600

Notice: Pts children have the same symptoms of fever and coughing;
Started aproximally the same time;

First pts children got fever but after five days pt started developing new symptoms :
Exertional dyspnea, **Platypnea**, Leg edema, RUQ pain;

Look's like Heart Failure but Why the sign of Platypnea?



I ph.

- Cirrhosis
- Inflow via VCI and VCS to the right heart

II ph.

- Primary decrease in right ventricular compliance
- Hypertrophy as compensation mechanism

III ph.

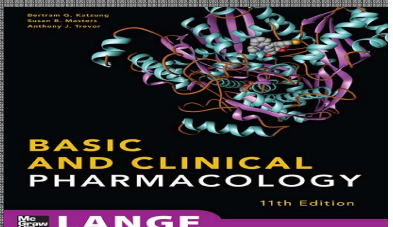
- Impaired ventricular filling during diastole
 - Hypertrophy/Restrictive cardiomyopathy
- Border-line thus called 'cirrhotic'

Jesse B. Hall

Handbook
of Critical Care

Third Edition

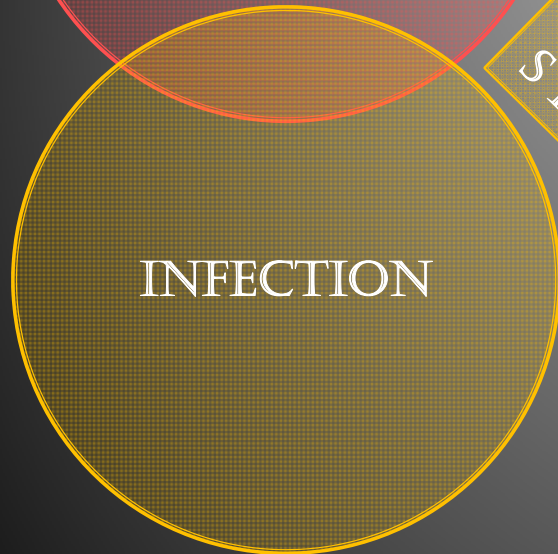
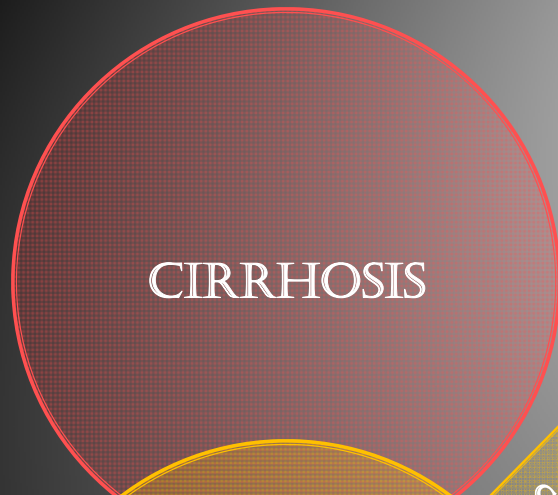
The patient is better when lying down
because the ventricular filling is being elevated
due to the gravitational forces



Think, think, and always think...



Heart Failure
Heart symptoms

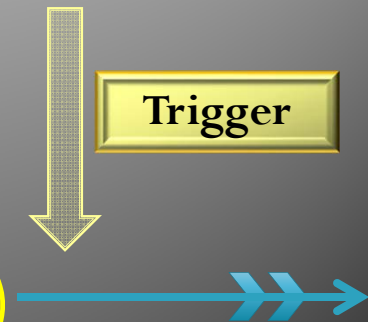
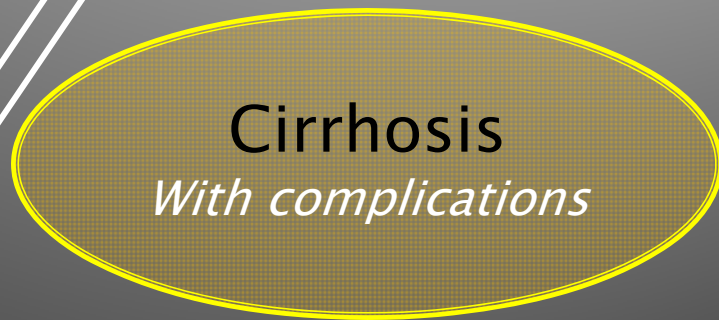


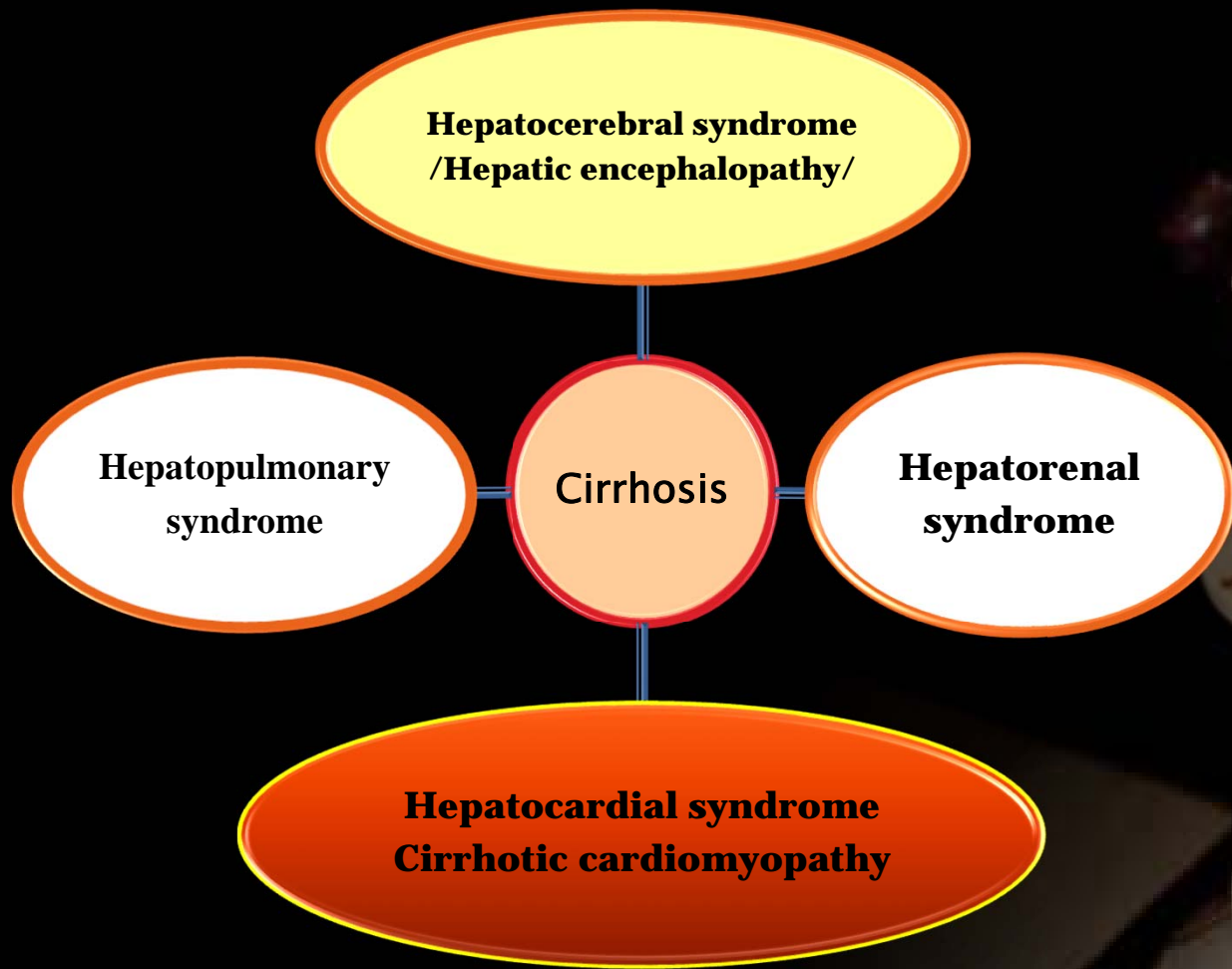
SIRS/SEPSIS

~ SBP ~
Spontaneous
Bacterial
Peritonitis



Trigger





- △ Acute on chronic right heart failure
- △ Usually about 5–10 years to develop
- △ Selective cardiomyopathy
- △ Long term subclinical until triggered
- △ Many times misdiagnosed
- △ Or underestimated



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~ Cirrhotic cardiomyopathy ~

Pathophysiological precursor of Right Heart Failure

Why do cirrhotic patients develop Congestive Heart Failure?



LEFT VENTRICULAR FAILURE:

- ❖ **Dyspnoea** (rest or after exercise) and **Orthopnea**;
- ❖ Associated with **paroxysmal nocturnal dyspnoea**;
- ❖ Episodes of **acute pulmonary edema** ;
- ❖ **Cheyne - Stokes respiration** may also be observed;
- ❖ Marked **tachycardia**;
- ❖ **Third heart sound** during diastole (gallop rhythm);
- ❖ **Moist crepitations over lung** basal areas;

Hmm ... RHF in cirrhotic patient without previous heart disease – Maybe it's cirrhotic cardiomyopathy ?

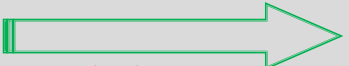

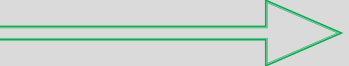

RIGHT HEART FAILURE (RHF)

- Right upper quadrant pain;
- Engorgement of the **venous tree**;
- Distension of the **jugular veins** ;
- Distension of the **liver**;
- Retention of fluid, **ascites**;
- Producing dependent **oedema of the legs**;
- **Hepatojugular reflex**;





Etiologies of Congestive Heart Failure

- Ischemic 
- Hypertensive 
- Valvular 
- Familial 
- Metabolic/Diabetes CHECK 

Don't forget :
Cirrhosis plus suspected bronchitis
with signs of Right Heart Failure
~ Correlation ~



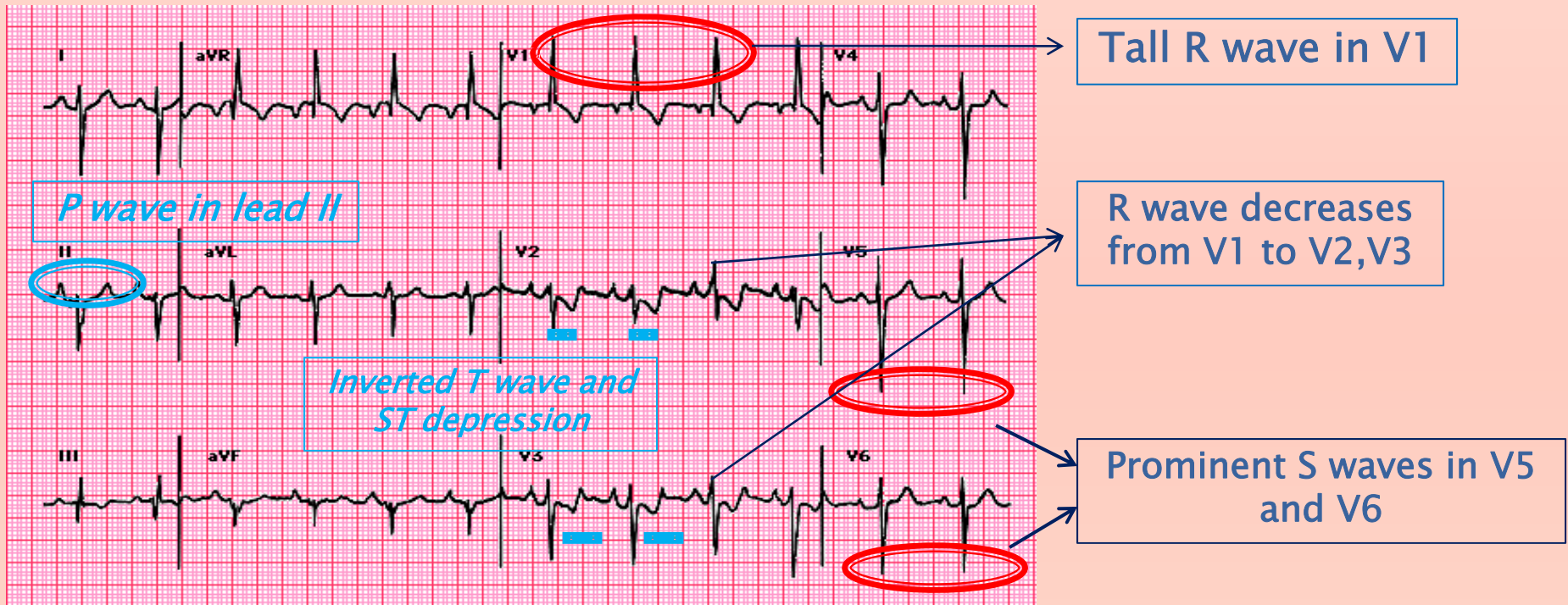
- Toxic
 - Alcoholic
 - Radiation
- No exposure**

- Drug-related (anthracyclines)
 - Heavy metals (cobalt, lead, arsenic)
- No exposure**

- Systemic diseases
 - Hypothyroidism
 - Connective tissue disease
 - Diabetes
 - Sarcoidosis
- In previous hospitalization already examined; But we double checked!**

- Amyloidosis
 - Hemochromatosis
- Markers and histopathological evaluation already done**

Cardiological aspect - The specificities



Combo diagnostics with TEUS (TransEsophageal US)

- Signs of right ventricular hypertrophy
- Signs of Right Heart Failure / Diastolic HF /

Cirrhosis induced adrenal stimulation gives catecholamin overproduction for maintaining:

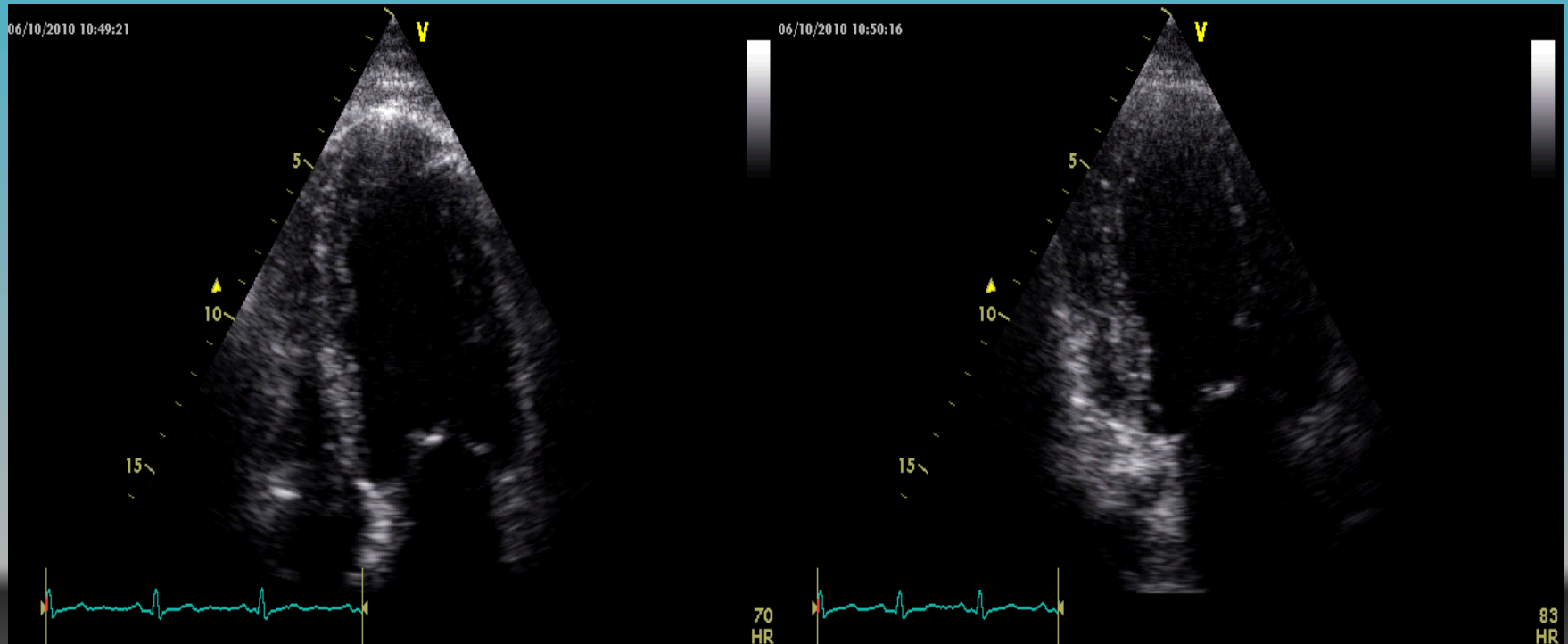
- *Adequate CO (Cardiac Output)*
- *Normal BP (Blood Pressure)*



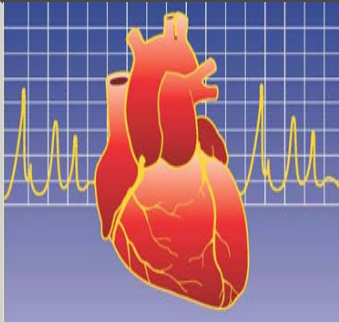
BP = 130/90mmHg HR=70-83/min

Regular rhythm, with proper sounds, no murmurs;

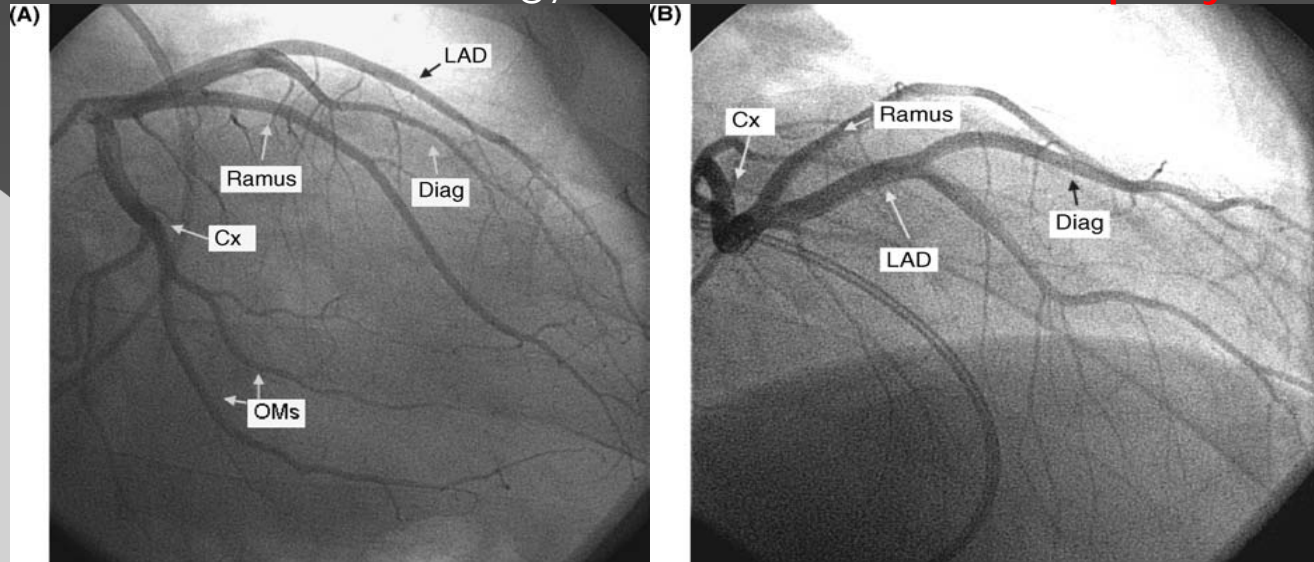
Transesophageal US: RV impaired relaxation. EF ~55% but ventricular filling is low. Intraventricular pressure high due to the prominent RV hypertrophy - Stiff heart syndrome.



- D-dimer in normal range;
- ECG without signs of Pulmonary Embolism;
- Transesophageal US without kinetic or segmental distortions;
- No vegetations;



Coronary angiography > coronary arteries intact
 No ischemic etiology → **No ischemic cardiomyopathy**



Rankovic et al. 'Grouping and damasking of hepatic cirrhosis global pathophysiology' Mainz University Hospital – Conference in association with dr. Falk, Deutschland 2012.

Exclude the coronary vessels
FIRST!

Exclude the lungs
SECOND!

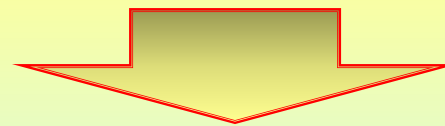
Test	Abbreviation
Peak expiratory flow	PEF
Forced expiratory flow in one second	FEV ₁
Forced vital capacity	FVC
Relaxed vital capacity	RVC
Total lung capacity	TLC
Residual volume	RV
Functional residual capacity	FRC
Maximum expiratory flow at lower lung volumes	MEF _{50%} etc
Airways resistance	Raw
Specific conductance	sGaw
Transfer factor	TL _{CO}
Transfer factor per unit lung volume (diffusion coefficient)	K _{CO}

No
 morphological or functional evidence that RHF could be a consequence of **Lung dysfunction**

Additional imaging:

■ **MDCT** → useful in identifying heart structural lesions thus excluding other RHF etiologies;

■ **24-hr holter monitor** → to screen for potential lethal arrhythmias;



✚ No structural heart defects, lesions or injuries;
✚ No arrhythmias found;

MDCT (MSCT) when performing for heart was performed **for lungs also**.

Chest X-ray
+
Clinical examination

Bronchitis – Trigger
for RHF

Symptoms
Clinical features
Epidemiology



Approach to the pt from systematic point of view

Approach is vital :

~ Open eye-sight for whole network of pathophysiologic changes ~

Cirrhosis

- Hepatic dysfunction
- ↓
- Systemic dysfunction

Cirrhotic Cardiomyopathy

- History, Symptoms
- Physical examination
- CXR, ECG,
- Biochemical panel

Integrative treatment

- Treat the liver
- Important
Treat the consequences

Therapeutics or What should we do? Just some key notes!

- ◆ Only review papers, textbooks no guidelines so far;

Opportunities to improve outcome I :

- ◆ Digoxin (decrease HR if Pt is tachycardic);
- ◆ Or option for Verapamil if EF is preserved;
- ◆ Diuretics (Not the corner stone if refractory ascites present);
- ◆ Perform paracentesis;
- ◆ Adding diuretics can sometimes lead to complications;
- ◆ ACE inhibitor (ATII antagonist's) decreasing preload/afterload(Kidneys?);
- ◆ Already on beta-blocker therapy (Caution!);

Opportunities to improve outcome II

- ◆ Dobutamin (Augmentation of Right Ventricle output);
- ◆ Milrinon (Phosphodiesterase inhibitor);
- ◆ Nesiritid/Natrecor (recombinant human BNP);
- ◆ Levosimendan (Calcium Sensitizer);
- ◆ Endothelin antagonists;

Controversial



Managing Acute Decompensated Heart Failure

Editors

Christopher M. O'Connor • Wendy Gattis Stough
Mihai Gheorghiu • Kirkwood F. Adams Jr.

Due to his primary illness (cirrhosis) patient has developed **cirrhotic cardiomyopathy** !
Cirrhotic cardiomyopathy is a **new entity** amongst other cardiomyopathies.

- It is in general subclinical and inapparent;
- It's **latency period depends on exposure to 'triggers'**;
- Triggers can be : Infection, surgical interventions, physical activity...etc.
- Always **selective** – so must be differentiated from other causes;

Our patient had a pulmonary infection which led him to Right Heart Failure due to the underlying Cirrhotic Cardiomyopathy.



In patients with hepatic dysfunction
always fear from the selective
cardiomyopathy development .
Disable precipitating "Trigger" factors.

Thank you for your attention !



And dear friends, let me invite you to the
International Gastroenterological Congress with
UEG Postgraduate course this October in
Belgrade, Serbia _october_2013.



Let us be your hosts !

www.ugs.rs

www.uis.org.rs



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