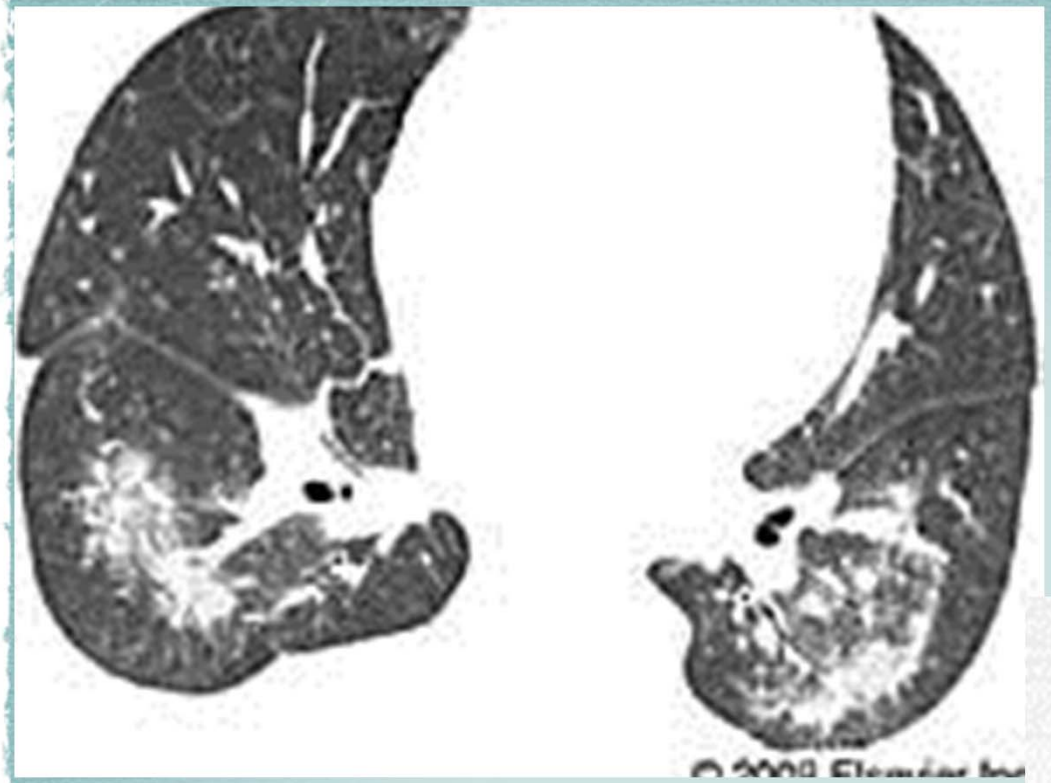
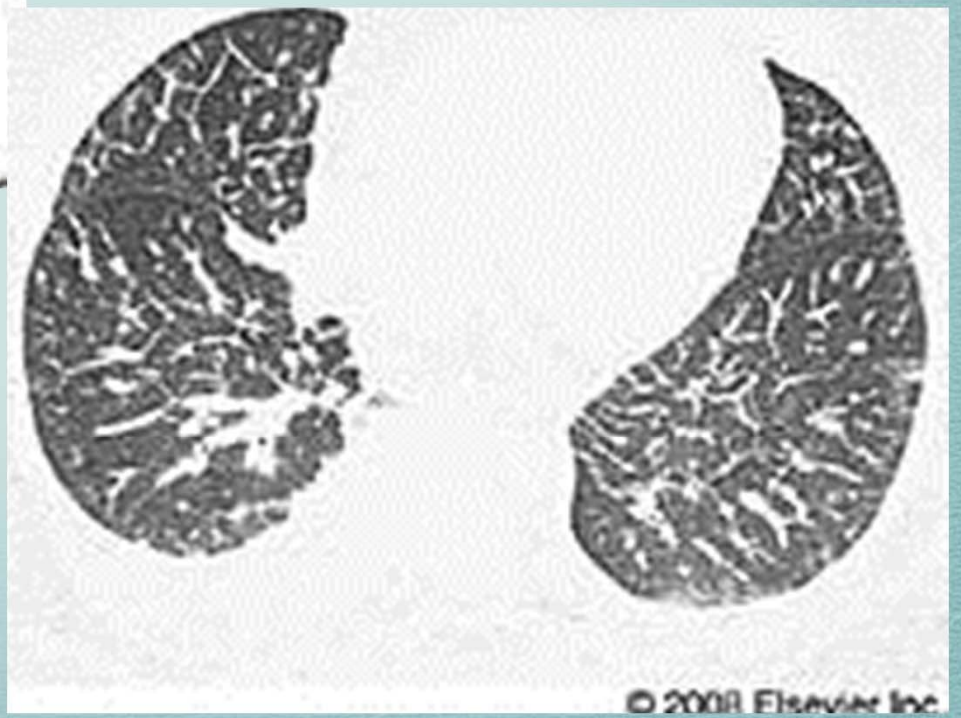
One Man One Disease

Dr Cheung Wing Sze Emily
Chairman: Dr Yan Wing Wa
PYNEH

22nd Nov 2011

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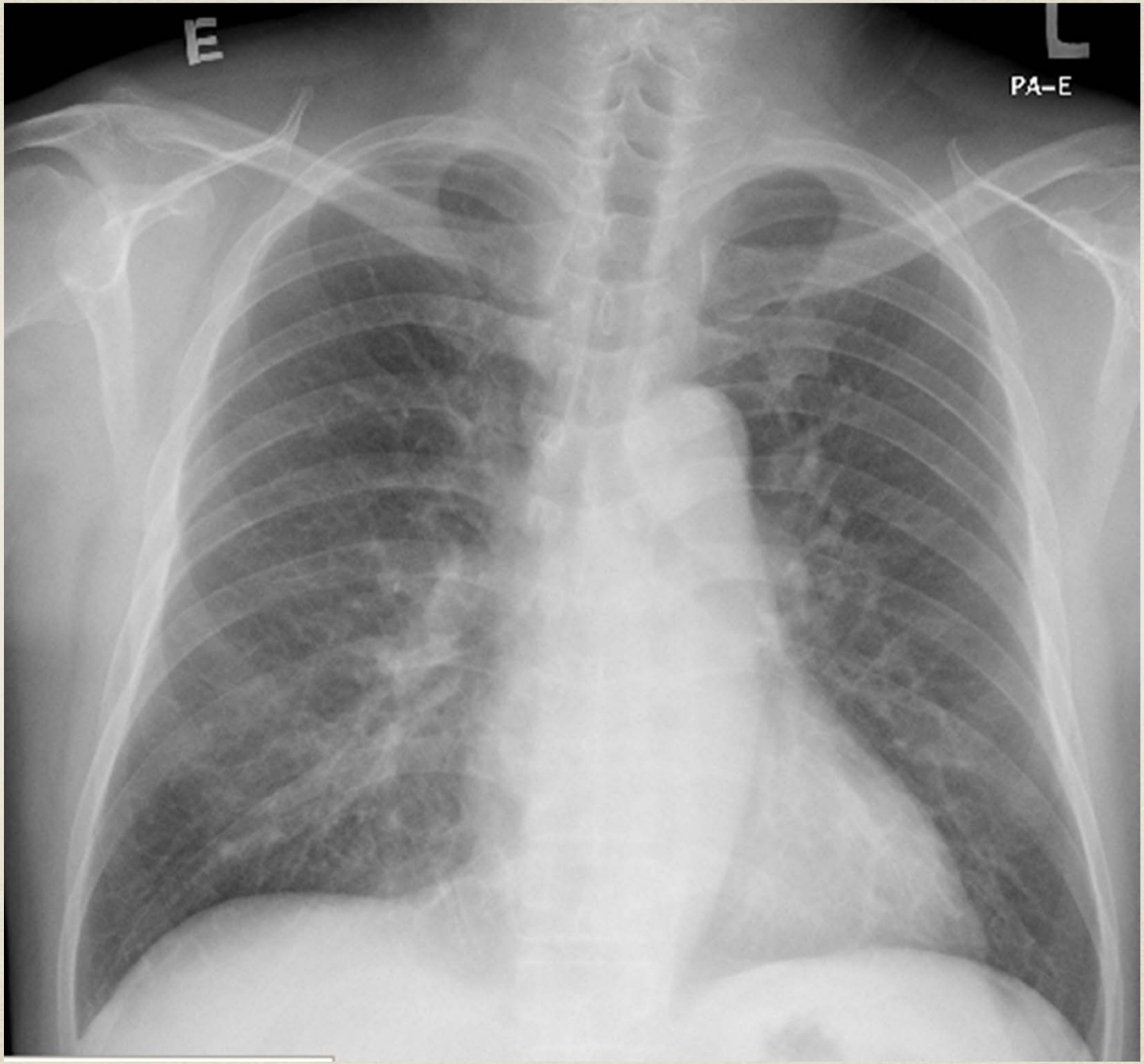
This story begins

- Mid July 2011
- 50 year-old gentleman
- Chronic smoker and social drinker
- Claimed works in logistic industry as clerical work
- Deny drug abuse

- PMH:
  - Poorly controlled hypertension
  - Chronic renal impairment with proteinuria (baseline Cr ~ 170)
  - Severe OSA once on CPAP
  - Beta thalassemia trait with baseline Hb around 10
  - Gout

- Complains shortness of breath and haemoptysis since 2 days before admission
- Admitted medical ward in Mid July
- All along afebrile and no other constitutional symptoms
- No nasal symptoms/sinusitis
- Travelled to “Yunnan province” recently with wife and friends
- Mild URTI symptoms but subsided for about a week before admission

- P/E
  - Chest bilateral creps, no wheezing
  - HS dual, PSM over apex
- Urine stix
  - +ve RBC and protein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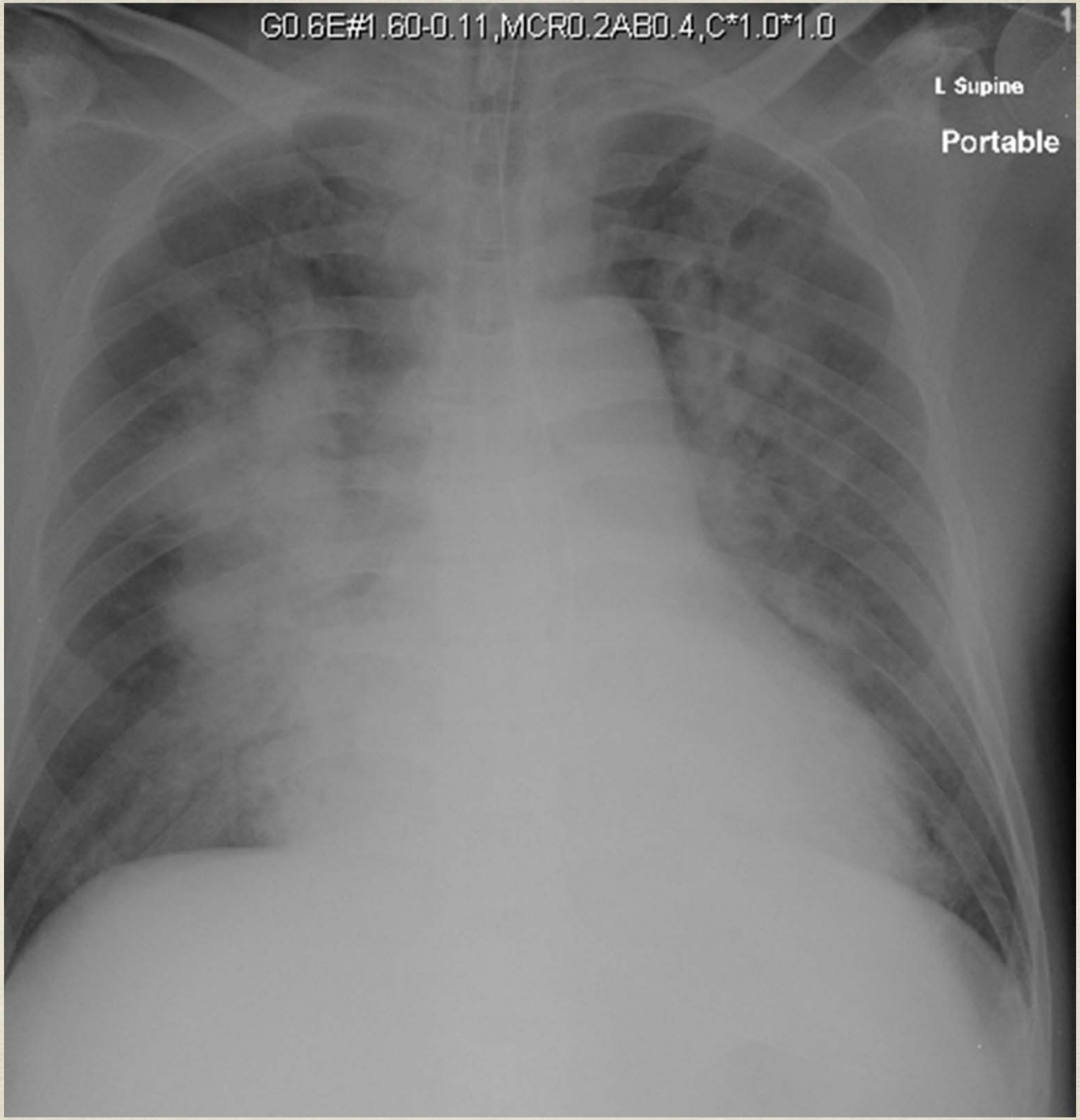
- Condition rapidly deteriorated on on Day 2
- Intubated and transferred to our unit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- High ventilatory support
  - Desaturation with SpO_2 down to 60% despite FiO_2 1.0
  - Lung recruitment done with airway pressure up to 40 cm H_2O
- Shock required high dose vasopressor support
- Acute kidney injury
  - Cr up to 320 with oliguria

What’s in your mind ?

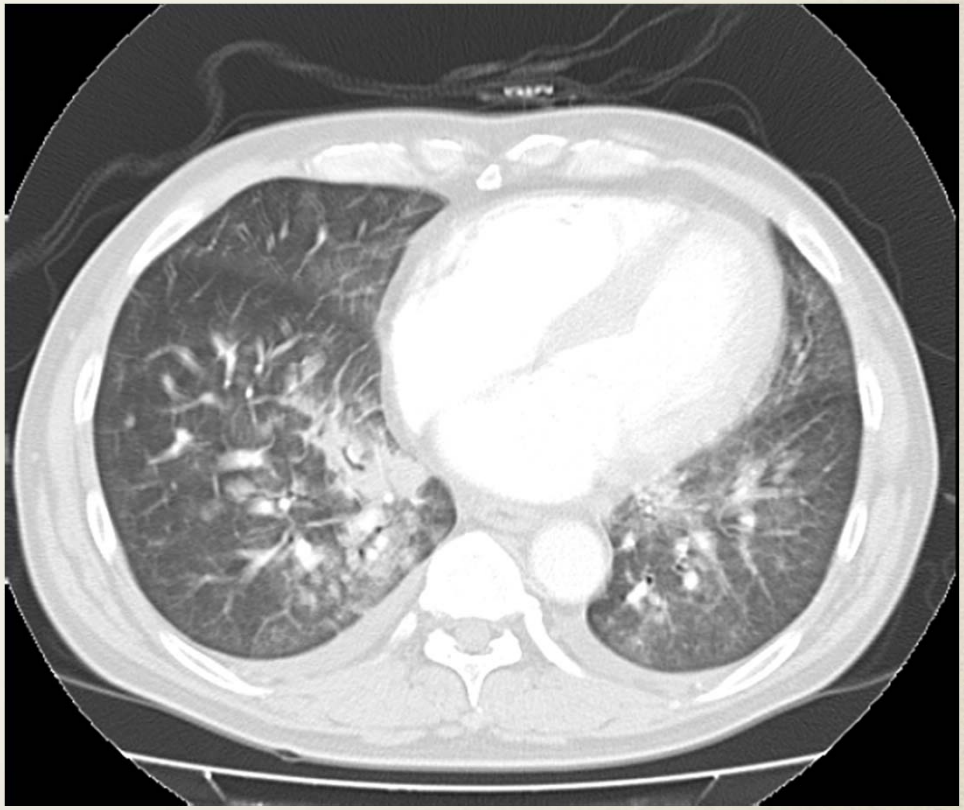
- Acute respiratory failure + Haemoptysis + Shock + AKI
- Differential Diagnosis ?!

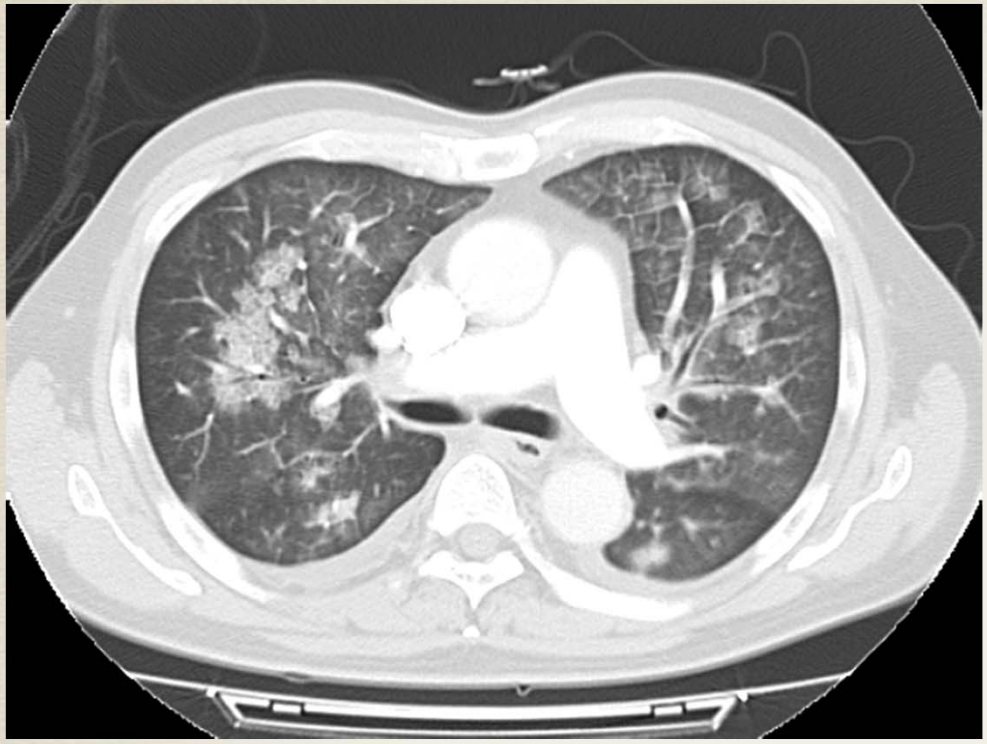
Differential Diagnosis

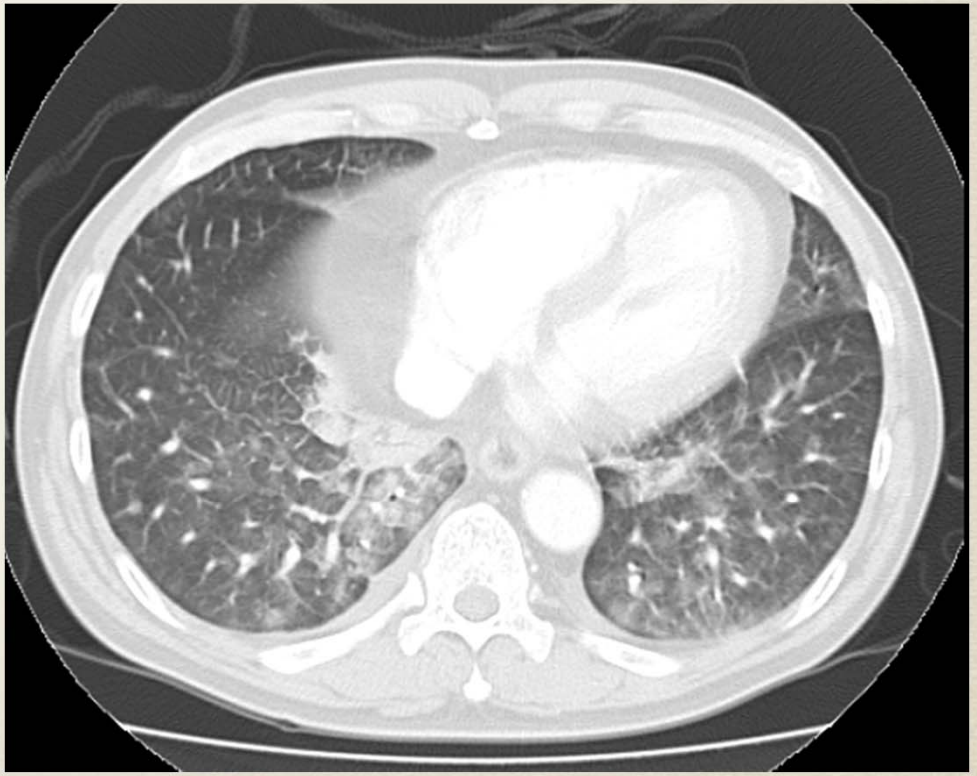
- Community acquired pneumonia with ARF
  - Includes atypical pneumonia, viral etc
- Pulmonary-renal syndrome
- Malaria in a patient with MODS and travel Hx

CT thorax

- No evidence of pulmonary embolism
- Diffuse patchy peribronchovascular and centrilobular ground-glass opacities/consolidation over all lobes of both lungs
- Cardiomegaly







Management

- VV-ECMO started on Day 3
- Given Tazocin/Doxycycline/Tamiflu
- CVVH
- FOB
  - No endobronchial lesion, bilateral airway not erythematous
  - Copious yellowish sputum with frank blood noted

Progress

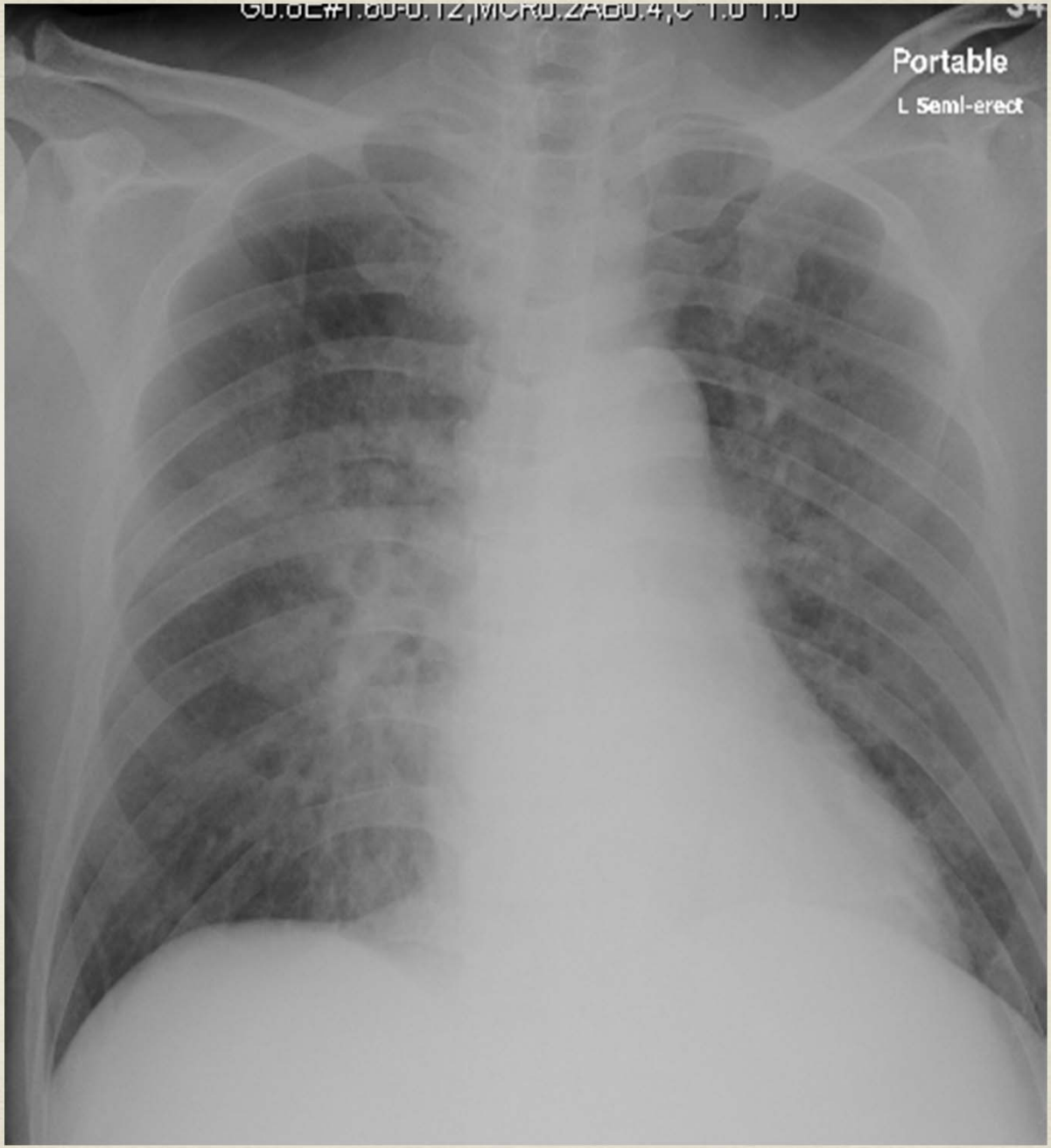
- AF required amiodarone infusion, betaloc
- Condition complicated by fluid overload
  - CXR shown congested lung fields and cardiomegaly
- Bedside Echo
  - LVEF 77%. No regional wall motion defect. Mild MR and LVOT pressure gradient not increased (29 mmHg)
- Anaemia required repeated blood transfusion
  - Plasma free Hb 393
  - Grossly elevated during ECMO

What will you do for the Anaemia

- Anaemia with grossly elevated plasma free Hb
  - Check ECMO circuit
  - Flow problem
- Rechecked Plasma Hb shown improving trend

- Microbiology results
  - ETA (Day 4): Pseudomonas/Acinetobacter ->pan-sensitive
  - NPA IF/HSI/Resp Virus negative
  - Blood/ BAL/AFB smear/fungal culture all negative
  - Widal/CMV/HSV/Atypical pneumonia PCR negative
  - 1st serum leptospirosis/rickettsia specific Ab/human metapneumovirus negative
  - Malaria blood smear negative
- Autoantibodies
  - ANCA/AntiGBM x 2/ANA/Anti-dsDNA/Anti-ENA negative

- Wean off VV-ECMO on Day 10
- Extubated on Day 12 and tolerate O_2 via nasal cannula
- Tapered down stress dose steroid gradually and stopped on Day 12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Progress

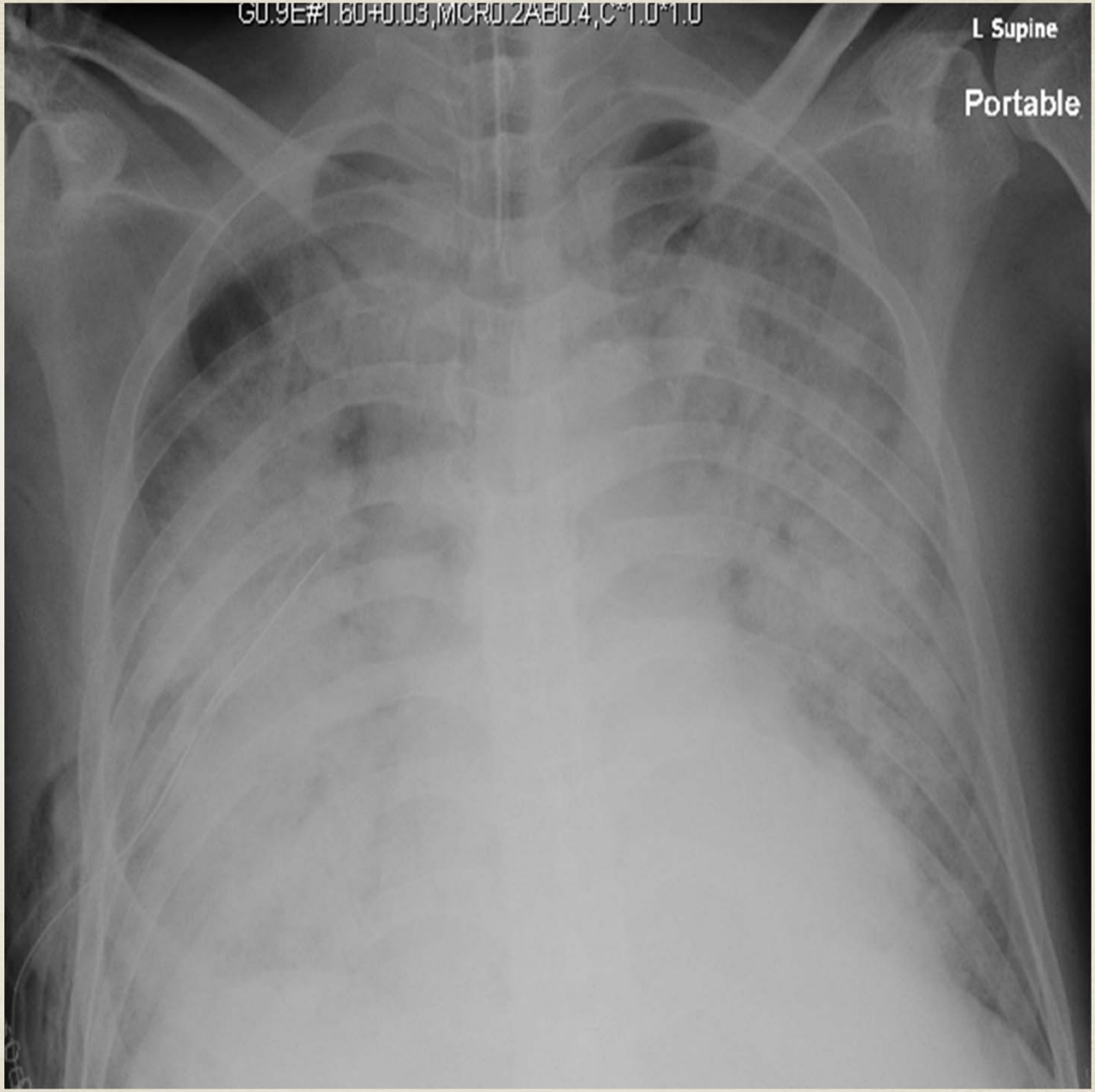
- Progressive worsening of SOB required nasal high flow oxygen up to 60%
- Repeated CXR showed increased bilateral infiltrations
- Swinging fever and elevated WCC

Differential Diagnosis

- ? HAP
  - Septic workup repeated
  - Upgrade Tazocin to Meropenem empirically
- ? Unresolved underlying lung disease - ? BOOP/Pulmonary-renal syndrome
  - Surgery consulted x open lung biopsy (done on 29 July)
  - Changed to methylprednisolone

- Lung biopsy
  - Focal intra- alveolar haemorrhage with no evidence of primary vasculitis
  - No granuloma nor hyaline membrane formation
  - Staining for mycobacterium/fungus were negative
  - Features not suggestive of BOOP
  - Eosinophils/hemosiderin-laden macrophages/malignant cells were negative
  - IF staining was not performed as specimen sent in formalin

- Developed frank haemoptysis with desaturation on late July required re-intubation
- FOB shown blood clots over RLL and RML
- CT thorax
  - Progression of diffuse peribronchovascular and centrilobular consolidation and patchy ground glass opacity
- BAE
  - No extravasation nor vascular abnormality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Differential Diagnosis

- Pulmonary disease
  - Infection (HAP) -> Culture results so far negative
  - BOOP -> Lung biopsy not suggestive of BOOP

Differential Diagnosis

- Pulmonary-Renal syndrome
  - Microscopic polyangiitis and Wegener granulomatosis ->ANCA -ve and biopsy suggestive of alveolar haemorrhage but not vasculitis
  - Churg-Strauss syndrome -> Eosinophils count not elevated and no nasal symptoms/asthma, ANCA -ve
  - Goodpasture's disease -> IF not done, antiGBM could be negative

Pulmonary-renal syndrome

Anti-GBM antibodies	Goodpasture's syndrome
ANCA-positive systemic vasculitis	Wegener's granulomatosos, Microscopic polyangiitis, Churg-Strauss Syndrome
ANCA negative systemic vasculitis	Henoch-Schonlein Purpura, IgA nephropathy, Behcet's disease
Drug related ANCA positive vasculitis	Prophlthiourail, Hydralazine, Allopurinol, Sulfasalazine
Autoimmune rheumatic disease	SLE, Scleroderma, Polymyositis, Rheumatoid arthritis
Thrombotic microangiopathy	Antiphospholipid syndrome, TTP, Infections, Neoplasmas

Anti-GBM negative Goodpasture's disease

- Goodpasture's syndrome
  - Rapidly progressive crescentic glomerulonephritis associated with pulmonary haemorrhage in more than half of patients
  - Rare cause of renal failure with an incidence of 0.5 to 1 case per million

- Pathology
  - Linear deposition of immunoglobulin along the GBM
  - Generally of IgG class
  - Antibodies bind to the noncollagenous domain of type IV collagen
- Immunoassays by ELISA/Western Blotting
  - ~ 3% may have negative serology
  - Early renal biopsy may allow correct diagnosis

- Renal team consulted for renal biopsy (Early Aug)
  - Declined as renal biopsy may not provide additional useful information
  - As RFT improving trend and less suggestive of AntiGBM disease
- Uncertain cause despite extensive investigations

Progress

- Continued on methylprednisolone and IMV
- Condition improved
- Gradual clearing of pulmonary infiltration
- CRRT with negative balance, dependent on SLED
- Able to wean off O_2
- Discharged to General Ward in Mid Aug

Story continues ..

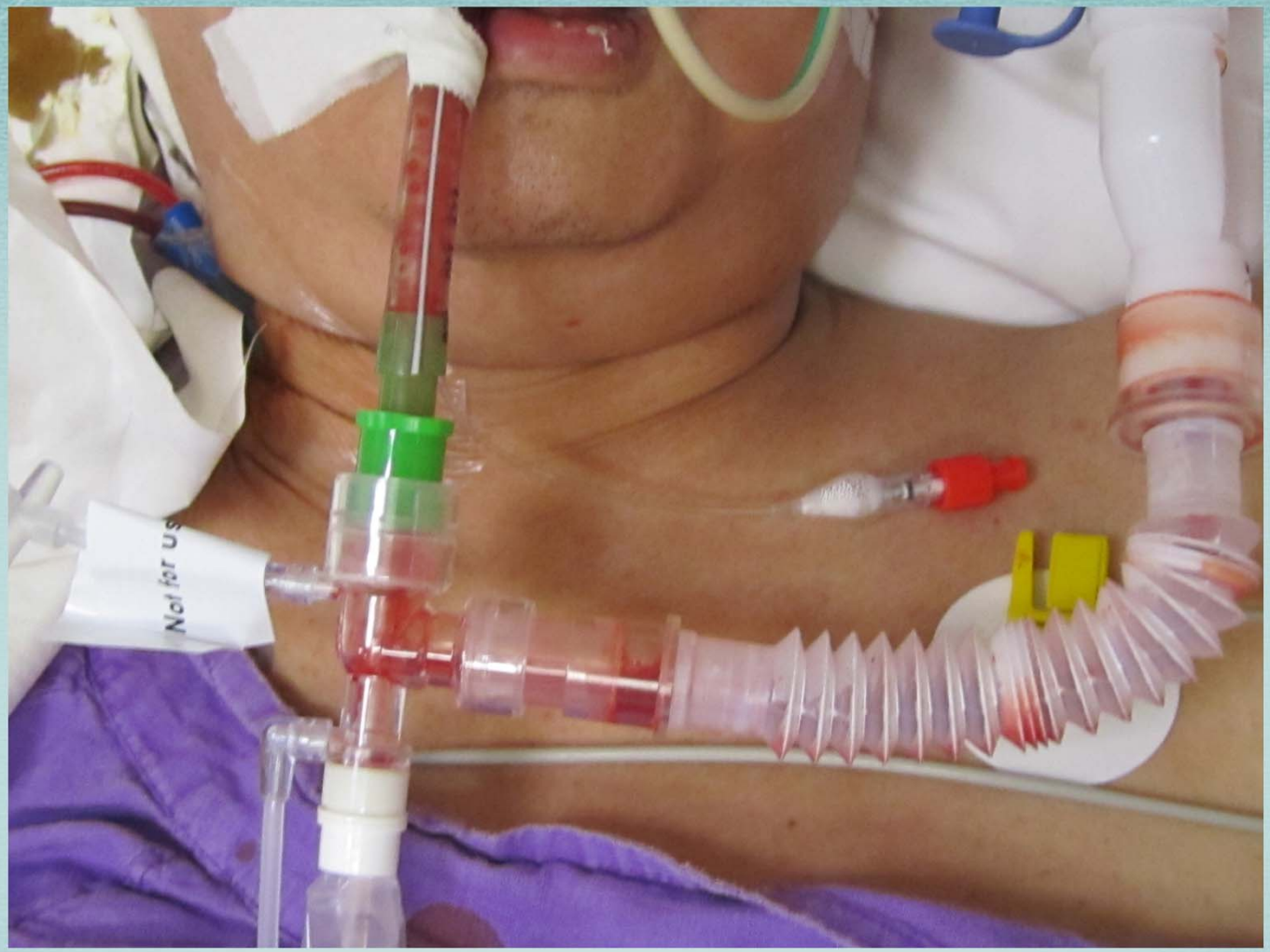
- Echo done by Cardiologist (Mid Aug)
  - Mild concentric LVH with preserved LV systolic function
  - Normal RV size and systolic function
  - Anterior MV leaflet prolapse with moderate eccentric jet of MR
  - Trivial AR, trivial TR

Story continues ..

- Complicated by CMV pneumonitis
  - Evidence by elevated CMV pp65 up to 31 (2x10e5)
  - Treated with Ganciclovir
- Increased O_2 requirement with CXR shown increasing ground glass opacity
- Re-admitted ICU in late Aug

2nd ICU admission

- Patient volunteered acute deterioration after blood transfusion
  - Received packed cells transfusion within 6 hours of deterioration
- Developed frank haemoptysis and re-emergence of pulmonary infiltrates
- Re-intubated on Day 2 (2^{nd} ICU admission)



On the day of deterioration

Question

- What is your differential diagnosis now ?

Differential Diagnoses

- Pulmonary haemorrhage ? Pulmonary-renal syndrome
- ANCA negative vasculitis/NE
  ANCA vasculitis (Possibility of false negative lung biopsy)
- CMV Pmeumonitis in immunocompromised host
- TRALI

- Infection
  - Treated with ganciclovir for CMV
  - BAL sent to rule out concomitant infection
- Pulmonary-renal syndrome
  - Re-consulted renal team for renal biopsy, withheld due to risk > benefit (As patient got thrombocytopenia with Plt count down to 10)
  - Plan for plasmapheresis if deteriorates before biopsy confirmation
- TRALI
  - No eosinophilia + acute transient drop in blood neutrophil count
  - Leukocyte-depleted RBCs with leukocyte filter for all blood transfusions but patient symptoms recurred despite filter usage
  - Screening not possible from Red Cross

- Plasmapheresis and pulse steriod followed by renal replacement therapy started -> Massive haemoptysis and desaturation with 100% O2
  - to cover for possible Goodpasture's disease
- Sputum: acinetobacter/stenotrophomonas
  - started Meropenem -> levofloxacin and Sulperazon
- Condition stabilized with plasmapheresis/CVVH
- Extubated on Day 8 (2nd ICU admission)

Condition complicated by haemolysis

- Hb continues to drop despite resolution of haemoptysis
  - Raised bilirubin ~ 36.5 with increased unconjugated bilirubin 14.2
  - Haptoglobin < 0.06
- Thrombocytopenia (platelet down to ~ 10)
  - ? Heparin induced thrombocytopenia
    - Possible sources :ECMO, SLED, Heparin lock, LMWH for DVT prophylaxis
  - PNH/Intravascular lymphoma

Heparin–Induced Thrombocytopenia

Type 2 heparin-induced thrombocytopenia

- More serious form
- Immune mediated disorder with Antibodies against the heparin-platelet factor 4 complex
- Usually onset after 5 to 10 days
- Associated with a fall of >50 percent
- Heparin-dependent antibodies usually develop between day 5 to 8 after exposure to heparin
- Spontaneous bleeding is unusual

Type 1 heparin-induced thrombocytopenia

- No clinical consequence
- Lesser fall in platelet count
- Usually first 2 days after heparin initiation and returns to normal with continuous administration
- Non immune
- Appears to be due to a direct effect of heparin on platelet activation

- Clinical manifestations
  - Thrombosis (both venous and arterial)
  - Unknown mechanism
- Diagnostic test
  - 14C-serotonin release assay
  - Heparin – induced platelet aggregation
  - ELISA immunoassay

- **Haematologists opinion**
  - Peripheral blood smear: no schistocytes and Not suggestive of intravascular haemolysis
  - Coombs tests and cold agglutinin tests negative
  - PNH screening negative
  - ? Intravascular lymphoma
    - Bone marrow -> Active erythropoiesis with no definite abnormality seen
- **Avoid heparin usage**
  - Anti-heparin-PF4 -> negative

One disease with following Manifestations ?!

- Recurrent Acute Respiratory Failure with Pulmonary Haemorrhage
- AKI
- Intravacular Haemolysis
- Thrombocytopenia

Progress

- Fluctuating clinical course
- Developed frank haemoptysis each time and required repeated intubations (total 3 times during this ICU admission)
- Improved each time with negative fluid balance from CRRT

- On further questioning ..
  - Patient works in bar instead of logistics industry
  - ? Possible cocaine usage
  - His Answer “ When I am conscious, I am sure I didn’t abuse cocaine”

Differential Diagnosis

- CMV Pneumonitis
  - CMV pp65 decreased on treatment but haemoptysis recurred
- TRALI
  - On leucocyte filter during transfusion already
  - Cannot possibly occur in every bag of transfusion
- Pulmonary-renal syndrome
  - Goodpasture's syndrome (However AntiGBM negative x 3 times)
  - Cocaine related Pulmonary Renal syndrome
  - Wegener granulomatosis and Microscopic polyangiitis
- ? APO due to heart failure

Case Report

Pulmonary hemorrhage and antiglomerular basement membrane antibody-mediated glomerulonephritis after exposure to smoked cocaine (crack): A case report and review of the literature

Ginesa Mª García-Rostán y Pérez, Federico García Bragado and Ana Mª Puras Gil

Department of Pathology, Hospital Virgen del Camino, Osasunbidea-Servicio Navarro de Salud, Pamplona, Navarra, Spain

Pathology International; 1997

Antiglomerular Basement Membrane Antibody-Mediated Glomerulonephritis after Intranasal Cocaine Use

Ramón Peces Rafael A. Navascués José Baltar Miguel Seco
Jaime Alvarez

Services of Nephrology and Pathology, Hospital Central de Asturias, Oviedo, Spain

Nephron 1999; 81

Cocaine related Pulmonary renal syndrome

- Direct effect of cocaine and residual solvent contaminants in the smoked material
- Cocaine tissue damage may probably expose pulmonary basement membrane antigens -> Antibodies formation

Differential Diagnosis - Pulmonary renal syndrome

- Renal biopsy finally performed on 28 Sept
  - Speciman sent fresh for IF
  - Procedure was complicated by perinephric haematoma

- UNFORTUNATELY
  - No glomerulus seen in renal biopsy (Pathologist present)
  - Further biopsy was not taken at that time as procedure was complicated by haematoma
  - Overall features suggest some chronic tubulointerstitial nephritis

What will you do ?

- Continue immunosuppressant/plasmapheresis
- Repeat renal biopsy
- Other causes ?!

Differential diagnosis - ? Cardiac causes

- Previous Echo revealed moderate MR
- Haemoptysis and SOB improved after betaloc added for heart rate control
- CRRT to achieve negative balance
- Echo repeated by cardiac team

Is this diagnosis possible ?

CASE REPORT

Hemolytic anemia in a patient with hypertrophic obstructive cardiomyopathy

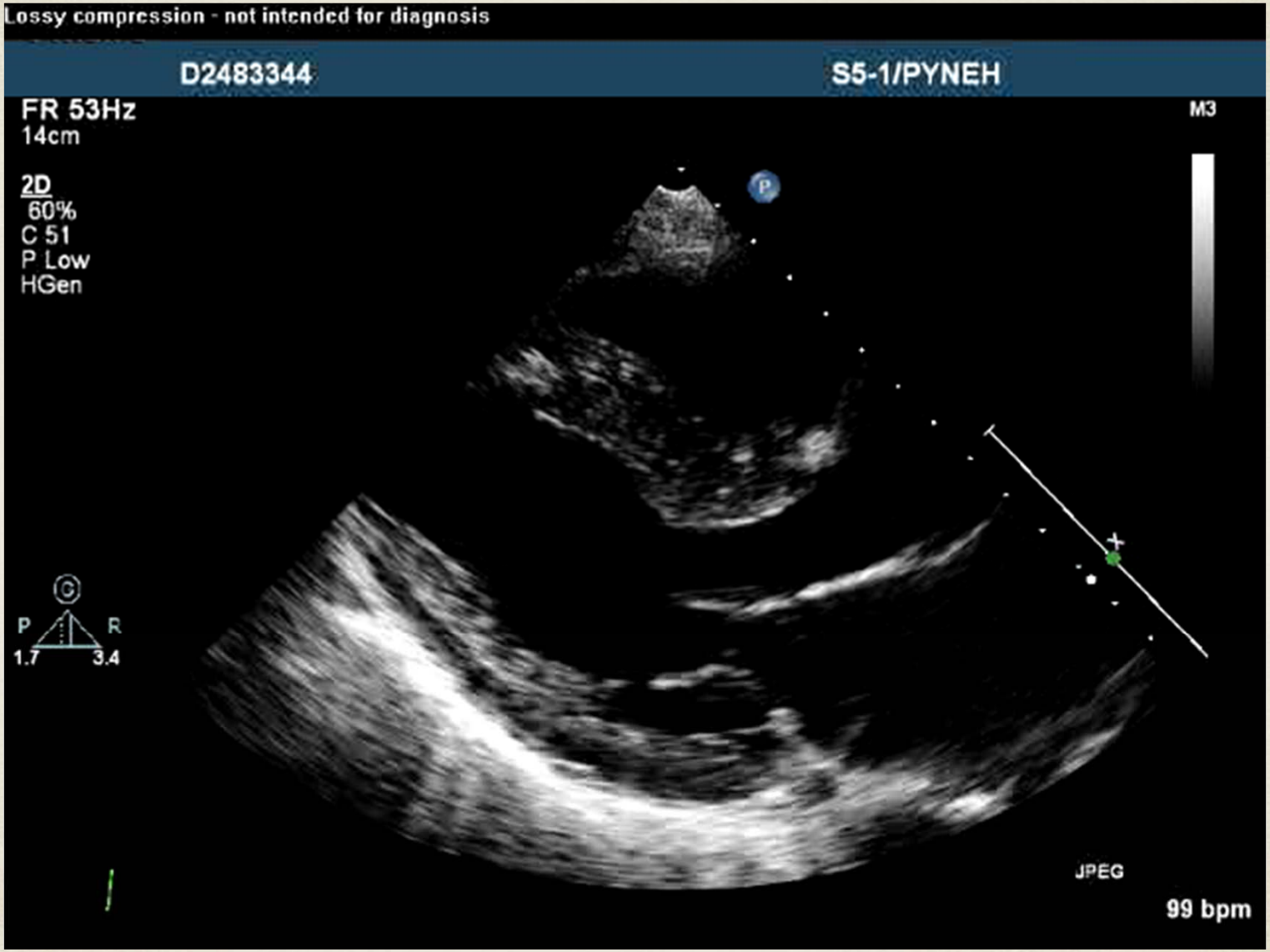
Toru Kubo (MD), Hiroaki Kitaoka (MD, FJCC), Yasunobu Terauchi (MD), Shinjiro Tamura (MD), Makoto Okawa (MD), Naohito Yamasaki (MD), Toshikazu Yabe (MD), Yoshinori L. Doi (MD, FJCC)*

Department of Medicine and Geriatrics, Kochi Medical School, Kochi, Japan

Journal of Cardiology; 2010

- Hypertrophic obstructive cardiomyopathy causing haemolytic anaemia
  - Very high LVOT gradient causing mechanical intravascular haemolysis
  - Increases ventricular contractility and causes severe outflow tract obstruction
  - Vicious obstruction-anaemic-tachycardia circle

Echo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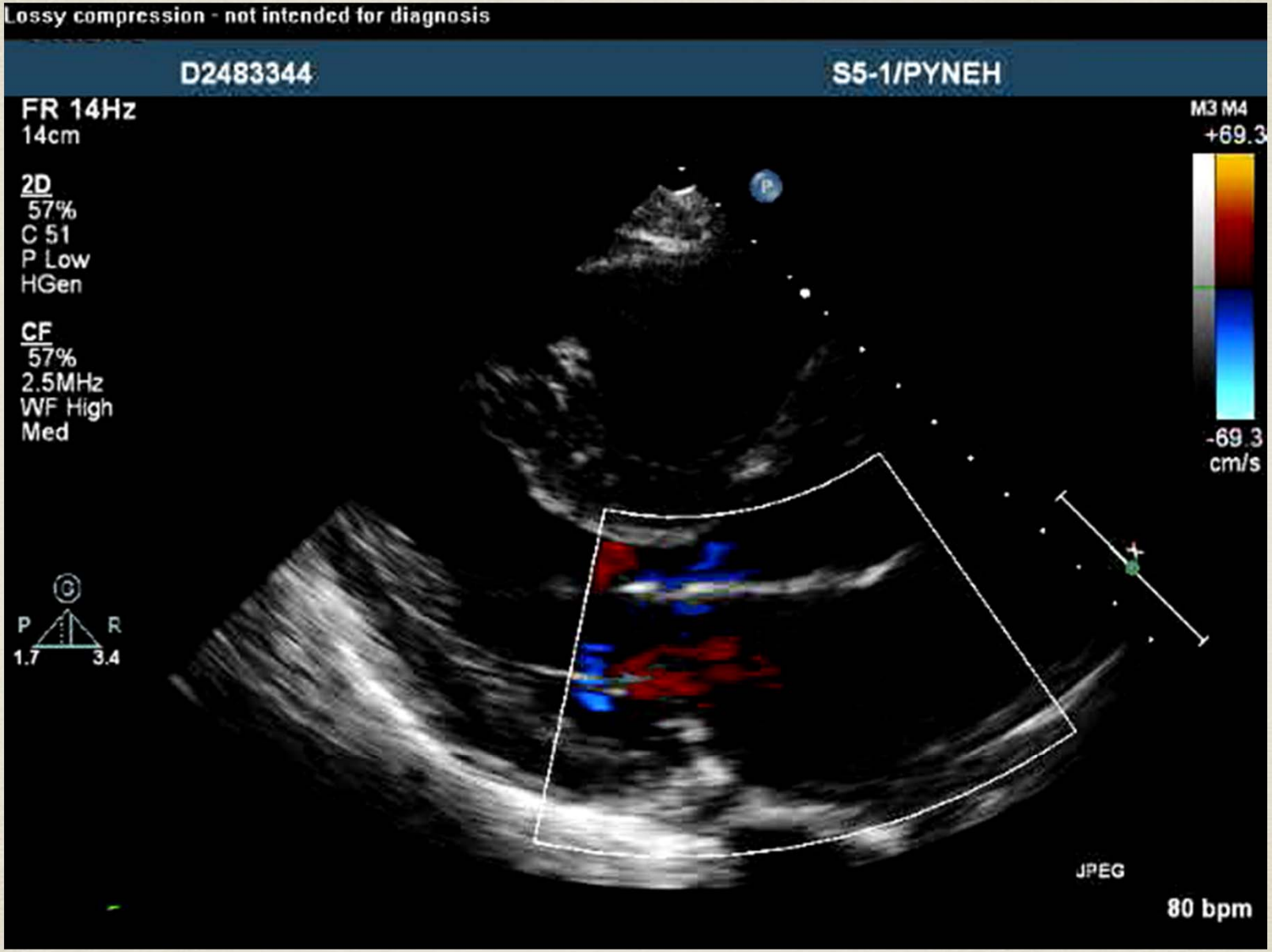
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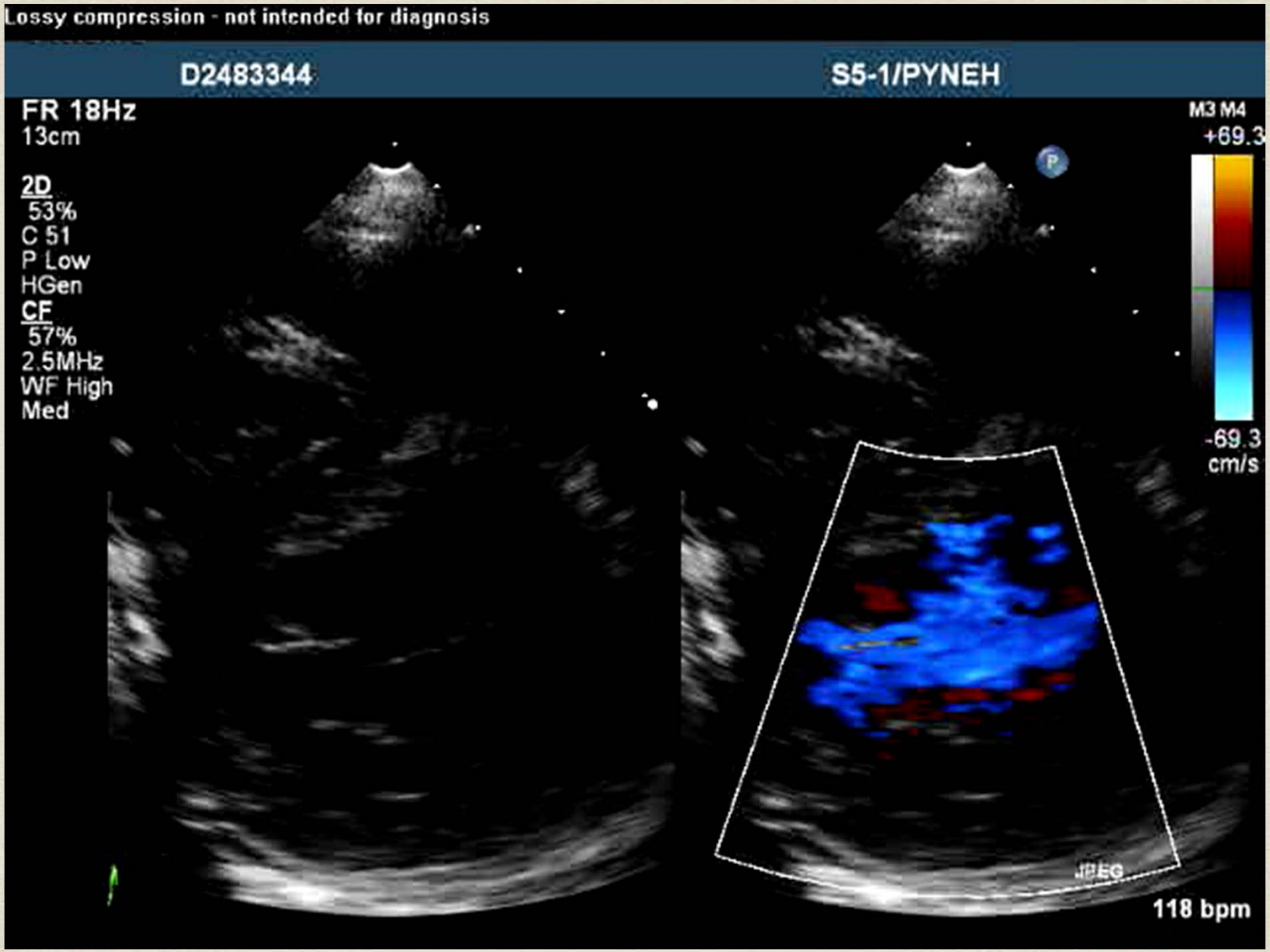
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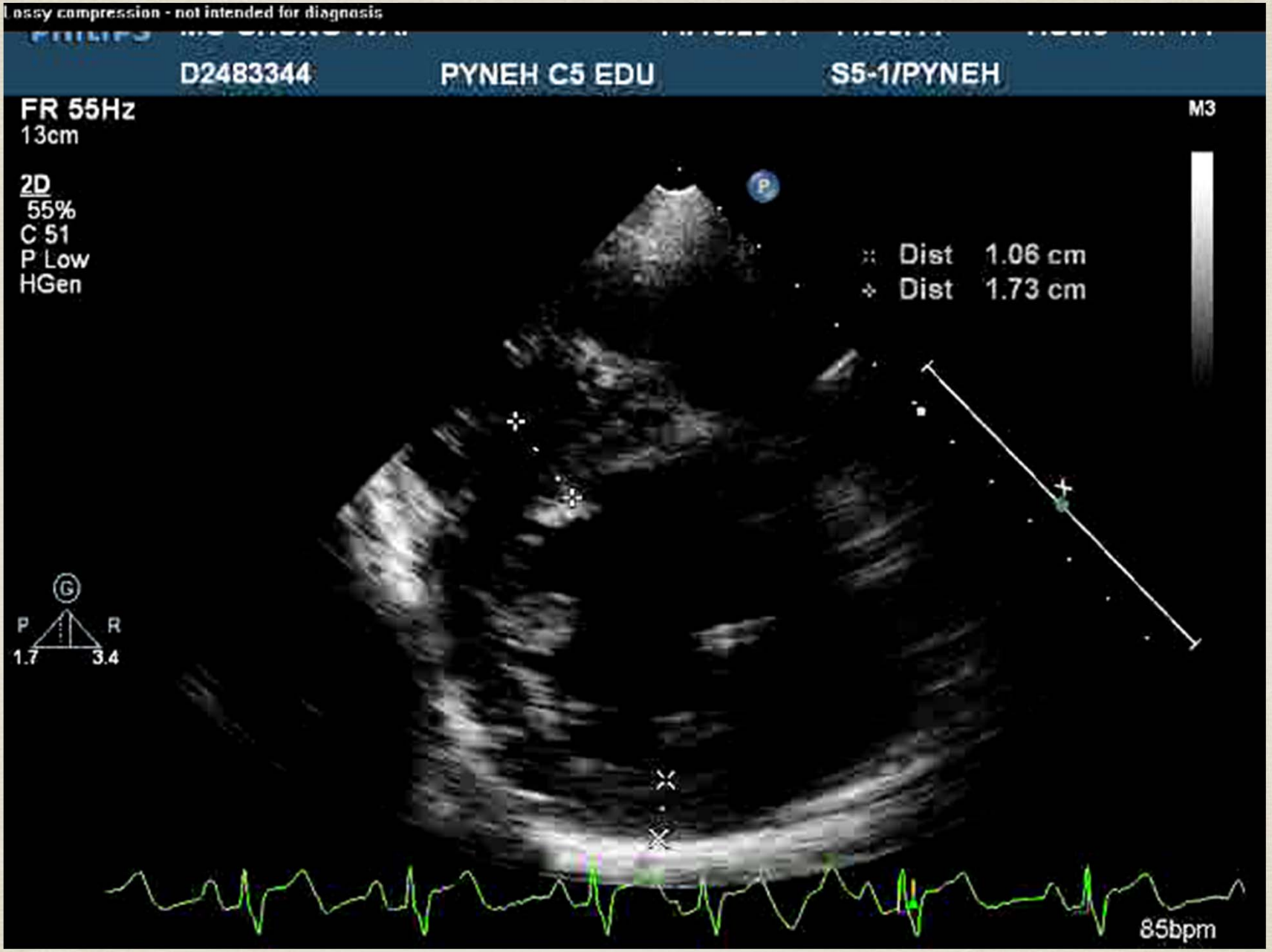
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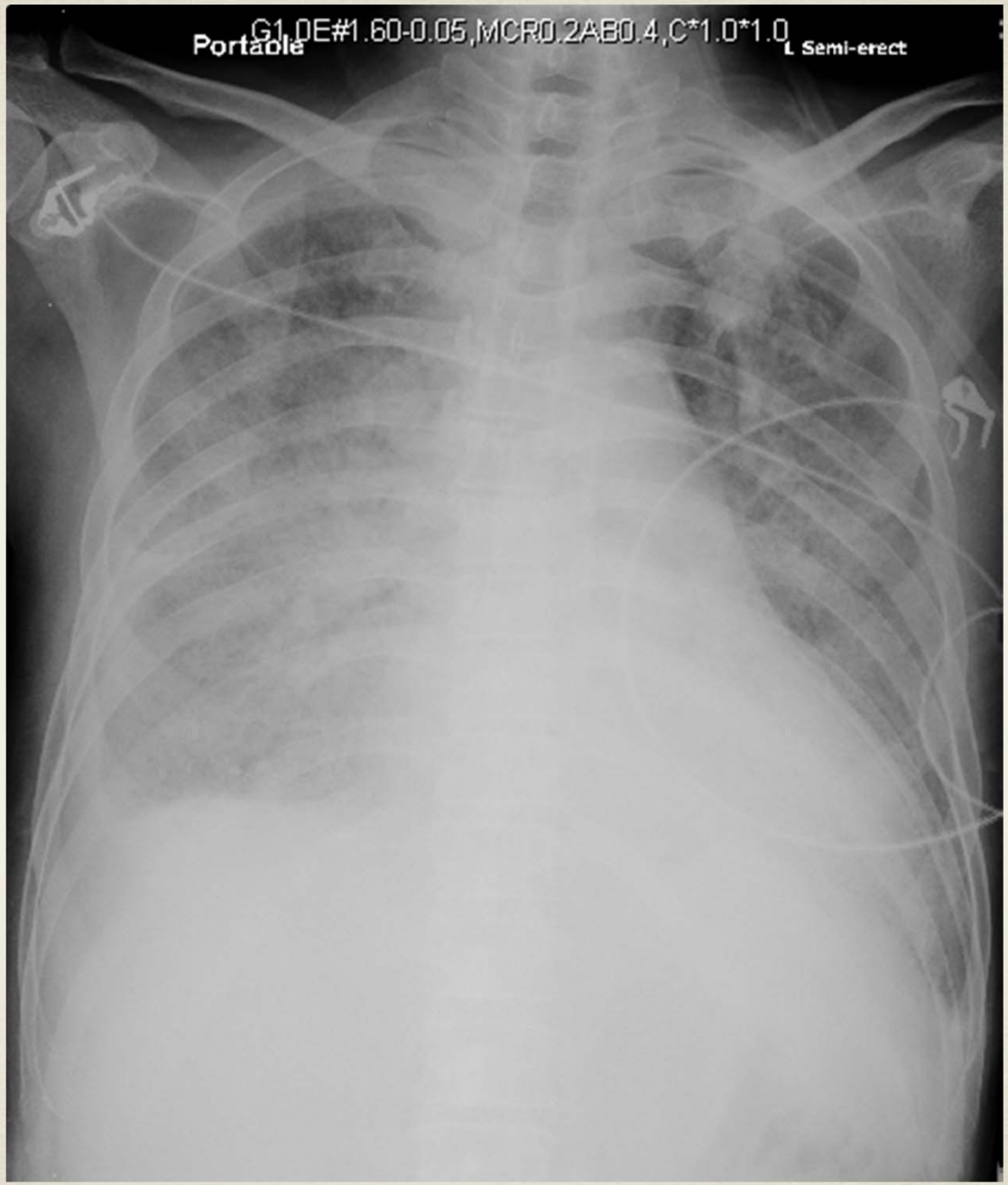
3.4
85bpm

- Echo report from cardiologist
  - Asymmetrical septal hypertrophy of moderate severity
  - No LVOT/intra-cavitary systolic gradient
  - No ASM of anterior mitral valve
  - Severe MR due to flail posterior MV leaflet
  - Mild pulmonary hypertension (RVSP ~ 40 mmHg)

- Condition improved after heart rate control with decreased rate of haemolysis
- Discharged from ICU in Mid Oct
- Still uncertain about his diagnosis …

Story .. repeated ..

- Developed respiratory distress again 2 days after discharge from our unit
- Required high nasal flow oxygen/NIV
- CMV pp65 Ag rising again -> another course of Ganciclovir
- Fast AF required amiodarone/betaloc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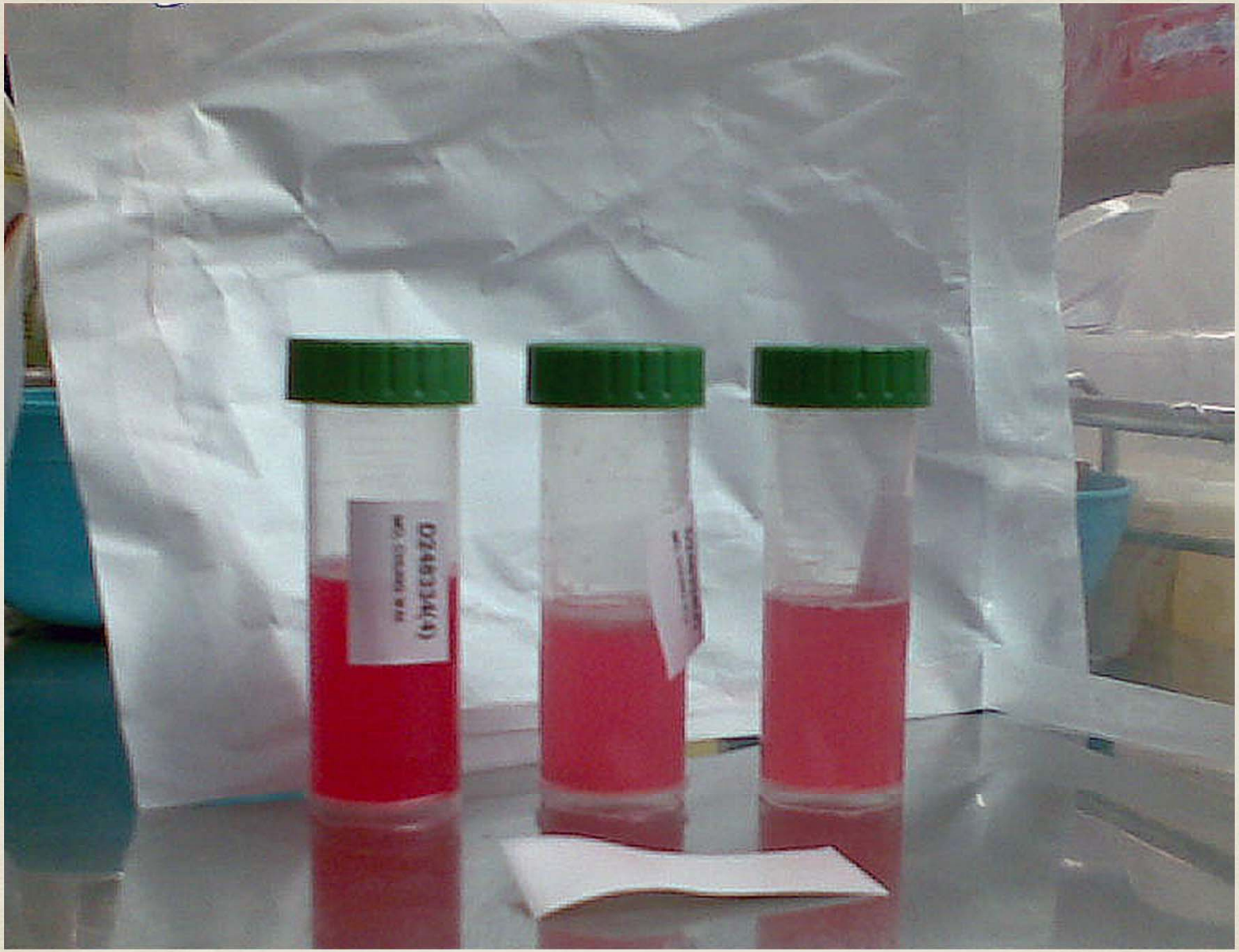
- Complicated by 2 episodes of hypoxemic cardiac arrest
  - Short period of CPR -> 3 mins and 2 mins respectively
  - Full neurological recovery

Workup ..

- FOB
  - Normal airway with no secretion inside
  - Progressive increasing bloody return after wedging
  - BAL shown increasing haemorrhage return + abundant hemosiderin laden macrophages -> Diffuse alveolar haemorrhage



Diffuse alveolar haemorrhage

- Haemoptysis, anaemia, diffuse radiographic pulmonary infiltrations and hypoxemic respiratory failure
- Other symptoms are non specific
- Non pulmonary signs and symptoms ->related to underlying systemic disease
- Presence of intraalveolar RBCs and fibrin -> accumulation of hemosiderin-laden marcophages

Table 1—*Etiology and Histology of Diffuse Alveolar Hemorrhage*

Pulmonary capillaritis
ANCA-associated granulomatous vasculitis
Microscopic polyangiitis
Isolated pulmonary capillaritis (ANCA positive and negative)
SLE
Rheumatoid arthritis
Mixed connective tissue disorder
Scleroderma
Polymyositis
Primary antiphospholipid antibody syndrome
Henoch-Schönlein purpura
Behçet Syndrome
IgA nephropathy
Goodpasture syndrome
Idiopathic glomerulonephritis (pauciimmune or immune complex-related)
Acute lung transplant rejection
Idiopathic pulmonary fibrosis
Diphenylhydantoin
Retinoic acid toxicity
Autologous bone marrow transplantation
Myasthenia gravis
Cryoglobulinemia
Ulcerative colitis
Propylthiouracil

- Bland pulmonary hemorrhage
  - IPH
  - Goodpasture syndrome
  - SLE
  - Coagulation disorders
  - Trimellitic anhydride
  - Isocyanate exposure
  - Penicillamine
  - Amiodarone
  - Nitrofurantoin
  - Mitral stenosis
  - Subacute bacterial endocarditis
  - Polyglandular autoimmune syndrome
  - Multiple myeloma
- Diffuse alveolar damage
  - Bone marrow transplantation
  - Crack cocaine inhalation
  - Cytotoxic drug therapy
  - SLE
  - Radiation therapy
  - ARDS

Workup .. for Pulmonary Renal syndrome

- Renal biopsy repeated on Mid Oct
  - Features consistent with hypertensive changes
  - No evidence for AntiGBM disease
  - Immunosuppression gradually tailed down and stopped

Workup .. Cardiac causes

- Cardiac consulted with serial Echo reviewed
  - Prolapsed posterior MV leaflet with mod-severe eccentric MR, suspected rupture chordae
  - No obvious LVOT obstruction
  - No vegetations

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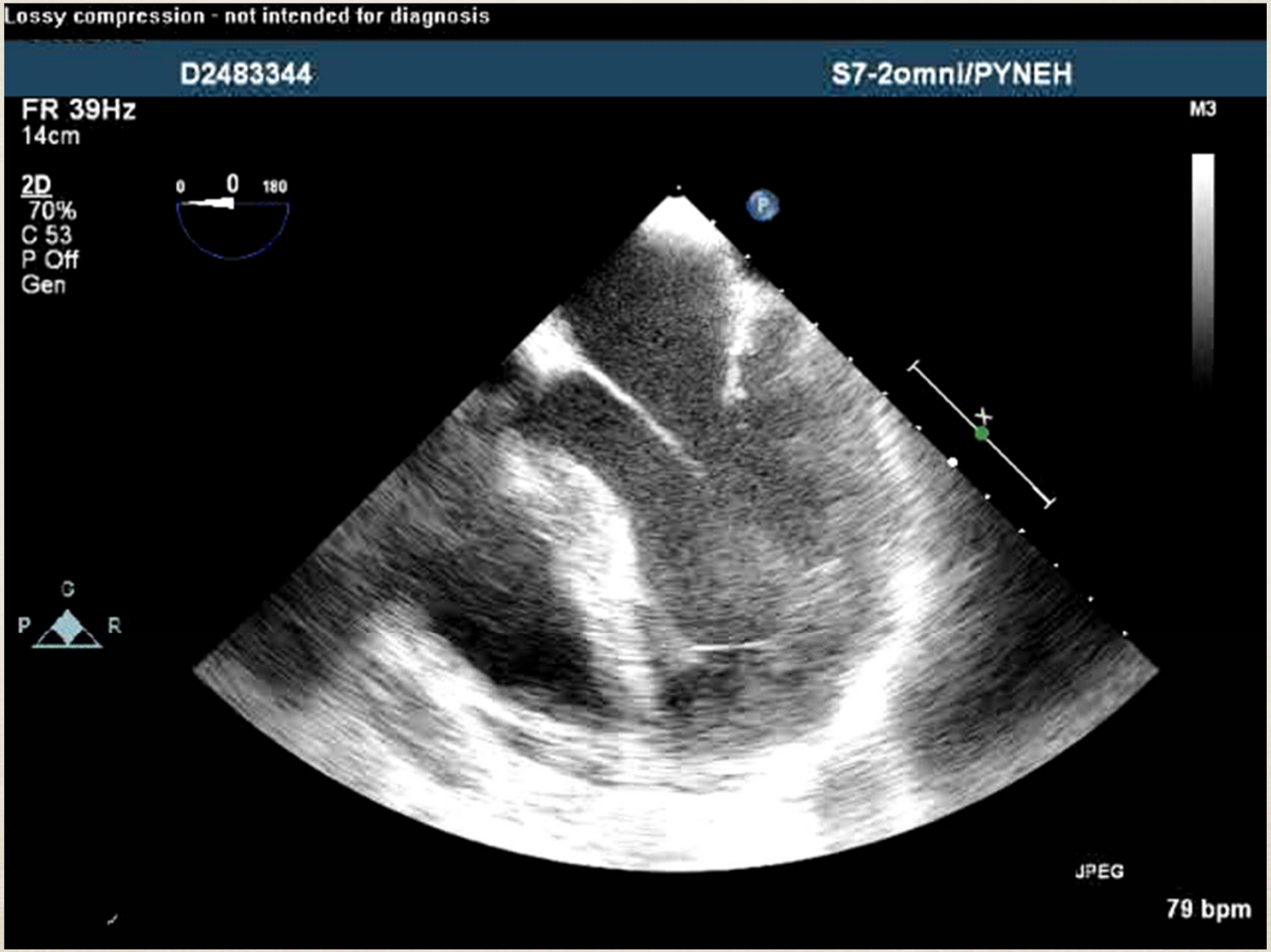
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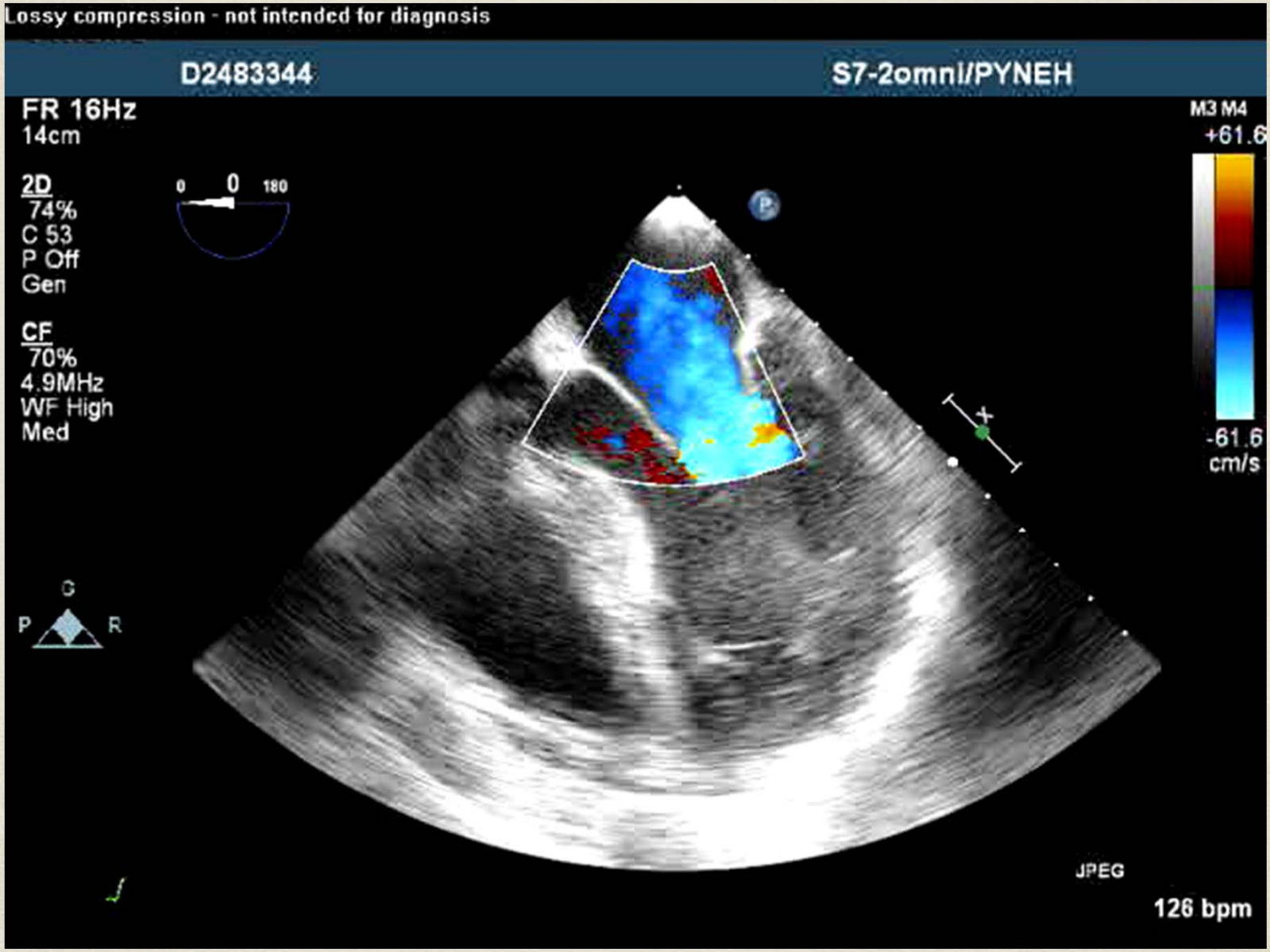
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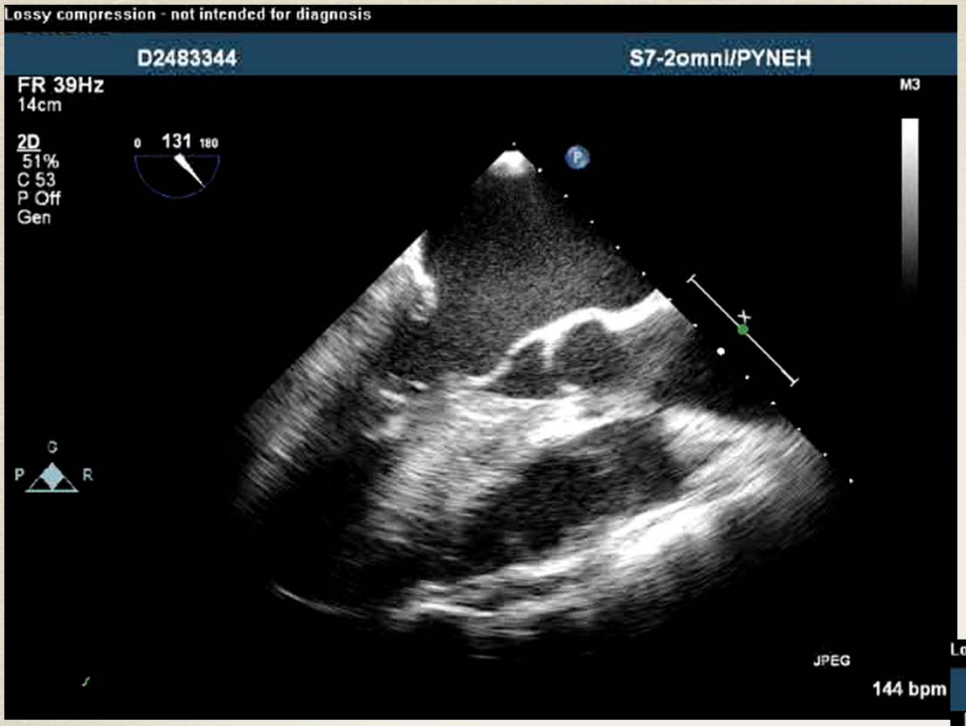
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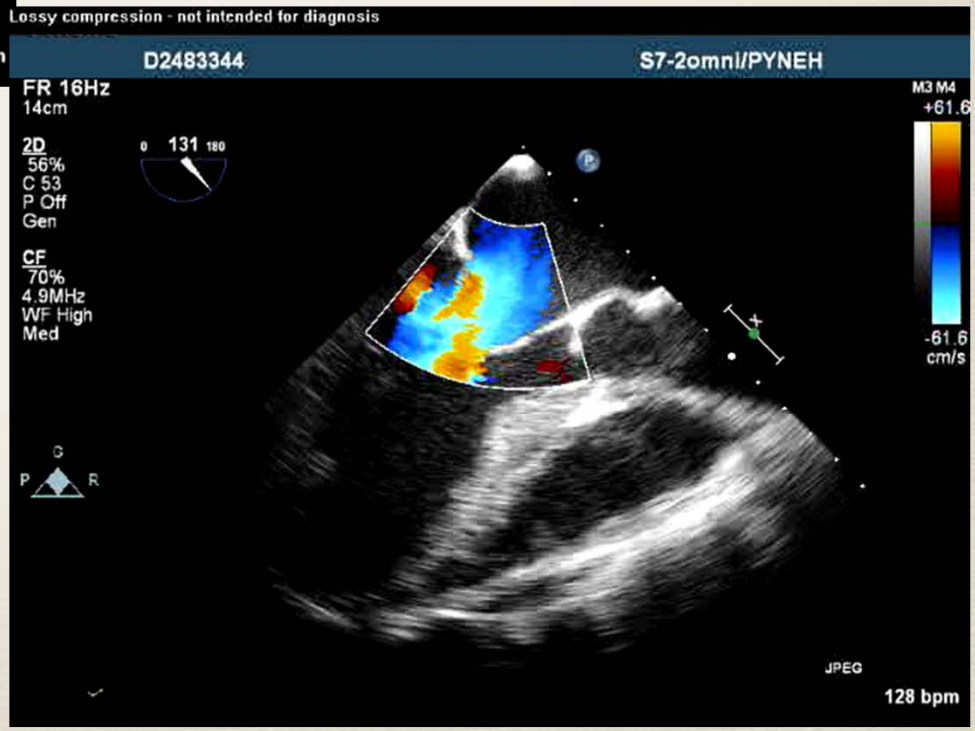
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Having excluded other DDx ..

One man One disease

- Acute MR with rupture chordae
- Presented with recurrent APO and pulmonary haemorrhage (atypical presentation)
- ? Underlying cause for rupture chordae

Progress

- QMH CTS contacted for MVR
- Perm cath inserted for intermittent HD

Diffuse Alveolar Hemorrhage Syndrome Due to 'Silent' Mitral Valve Regurgitation

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ABSTRACT: A variety of clinical diseases are associated with diffuse alveolar hemorrhage. Although mitral valve disease can cause hemoptysis, it rarely is associated with diffuse alveolar hemorrhage at presentation. A 49-year-old woman was admitted to the hospital with the abrupt onset of fever, anemia, dyspnea, azotemia, and diffuse alveolar infiltrates. Two-dimensional echocardiography done several months earlier to evaluate atypical chest pain had been unremarkable. Fiberoptic bronchoscopy 2 days after admission to the hospital revealed fresh blood throughout the tracheobronchial tree. The infiltrates resolved rapidly and completely during systemic steroid therapy only to reappear as the steroids were tapered, suggesting a beneficial therapeutic response. Results of serologic evaluation were negative. Transbronchial biopsies showed inflammation and hemosiderin-laden macrophages; no specific diagnosis was established. The patient was scheduled for open lung biopsy . The surgeon was concerned about the history of chest pain and requested placement of a pulmonary artery catheter, which revealed severe pulmonary hypertension. Transesophageal echocardiography and subsequent cardiac catheterization showed severe mitral regurgitation. Mitral valve replacement resulted in complete elimination of symptoms.

Southern Medical Journal 2000

Acute MR with Pulmonary Haemorrhage

- Acute mitral regurgitation usually has a devastating presentation
- Causing pulmonary hypertension -> Pulmonary Haemorrhage
- Causes of Pulmonary Haemorrhage
  - Disruption of the alveolar-capillary membrane or
  - Rupture of submucosal bronchial varices

Acute MR with Pulmonary Haemorrhage

- Transthoracic echocardiography may underestimates the severity of mitral regurgitation when compare with TEE
- Especially in thickened and calcified valve

Severity of mitral regurgitation (MR) in adults

	Mild	Moderate	Severe
Structural parameters			
Left atrial (LA) size	Normal, unless other causes of left atrial dilation	Normal or dilated	Dilated, except acute MR
Left ventricular size	Normal, unless other causes of left ventricular dilation	Normal or dilated	Dilated, except acute MR
Mitral leaflets or support apparatus	Normal or abnormal	Normal or abnormal	Abnormal/flail leaflet, ruptured papillary muscle
Doppler parameters			
Color Doppler jet area	Small, central jet, (<4 cm2 or <20 percent of LA area)	Greater than mild but no criteria for severe MR	Vena contracta width >0.7 cm with large central MR jet (area 40 percent of LA area or a wall impinging jet of any size, swirling in LA
Doppler vena contracta width	<0.30 cm	0.30 to 0.69 cm	≥0.70 cm
Quantitative parameters			
Regurgitant volume	<30 mL/beat	30 to 59 mL/beat	≥60 mL/beat
Regurgitant fraction	<30 percent	30 to 49 percent	≥50 percent
Regurgitant orifice area	<0.20 cm2	0.20 to 0.39 cm2	≥0.40 cm2

Adapted from Zoghbi, WA, Enriquez-Sarano, M, Foster, E, et al. American Society of Echocardiography Report. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003; 16:777; and Bonow, RO, Carabello, BA, Chatterjee, K, et al. ACC/AHA 2006 guidelines for the management of patients with valvular heart disease. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing committee to revise the 1998 guidelines for the management of patients with valvular heart disease). J Am Coll Cardiol 2006; 48:e1.

Acute mitral regurgitation

- Typically presented as cardiac emergency
- Rapid progression of pulmonary edema, hypotension and signs and symptoms of cardiogenic shock
- Often not recognized at presentation -> mimics an acute pulmonary process (e.g. infection or ARDS)

- Etiology
  - Flail leaflet due to IE or trauma
  - Chordae tendineae rupture
    - Trauma, spontaneous rupture, IE, acute rheumatic fever
  - Papillary muscle rupture or displacement
    - Acute myocardial infarction, trauma

Our Patient

- Atypical presentation for a common disease
  - Concomitant ARF and Pulmonary haemorrhage
- “Typical” presentation
  - Acute pulmonary edema with cardiogenic shock

Thank you

Questions are welcome