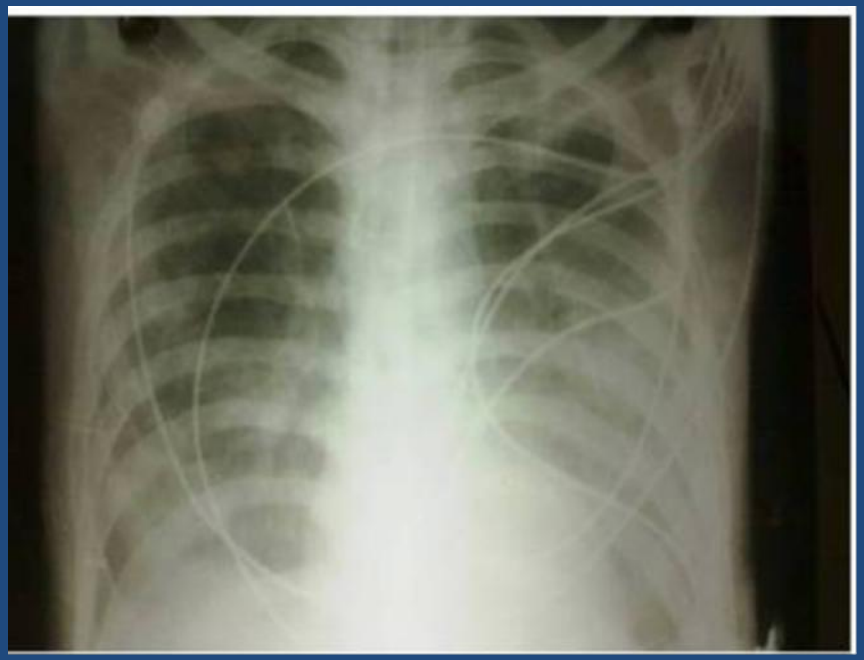
Non-cardiogenic pulmonary edema: when to think and how to treat?

Muhammad Abdur Rahim
MBBS, FCPS (Medicine)
Assistant Professor
BIRDEM Academy and Ibrahim Medical College

Case 1

- Male, 38 years
- Fever 4 days, abdominal pain and vomiting 2 days, breathlessness 1 day
- Temp 101.2°F, RR 40/min, Pulse 100/min, BP 100/70 mm Hg
- Rash, low oxygen saturation, 77% at room air
- Chest examination – breath sound normal, bilateral diffuse crackles
- Abdomen – distended, tender, no organomegaly, no ascites

CXR



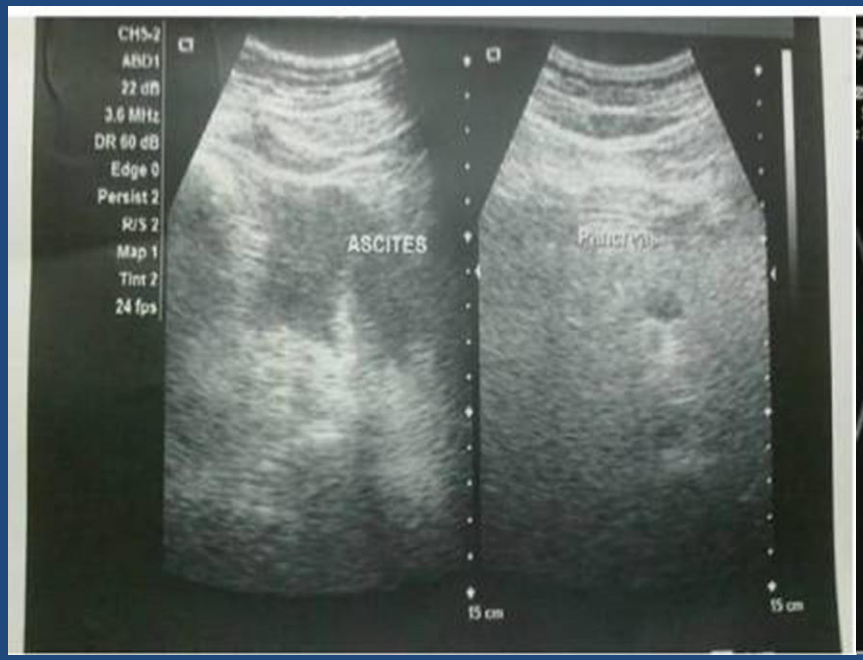
ABG

- PO_2 (80–100 mm Hg) 82 *
- PCO_2 (35–45 mm Hg) 20.8
- HCO_3 (20–24 mmol/l) 10.9
- SO_2 (%) 94.6

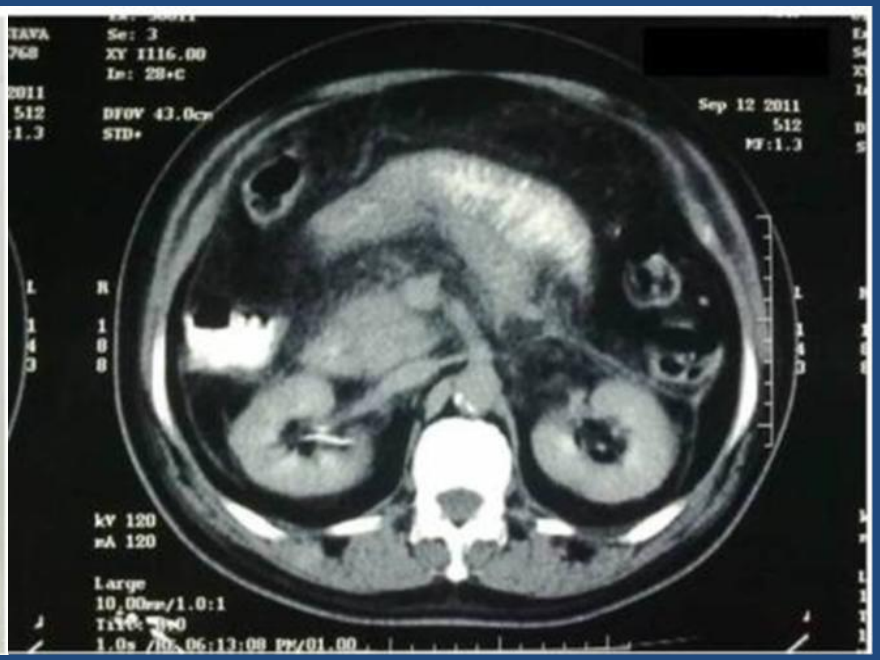
*with 2 liter/min oxygen

- Hb 9.8 gm/dL, HCT 29%
- TC of WBC 7600/cmm of blood
- Platelet 15,000/cmm of blood
- S. amylase 750 U/L (20-85 U/L)
- S. lipase 500 U/L (10-60 U/L)
- Dengue NS 1 positive

USG


CH5-2
ABD1
22 dB
3.6 MHz
DR 60 dB
Edge 0
Persist 2
R/S 2
Map 1
Tint 2
24 fps
ASCITES
15 cm
15 cm

CT scan


Se: 3
XY 1116.00
DFOV 43.0cm
STD+
Sep 12 2011
512
kV 120
mA 120
Large

- ECHO
  - No regional wall motion abnormality
  - LVEF 64%

- Management
  - ICU transfer
  - Mechanical ventilation
  - Other conservative managements

Dx – DHF complicated with acute pancreatitis, ARDS

Case 2

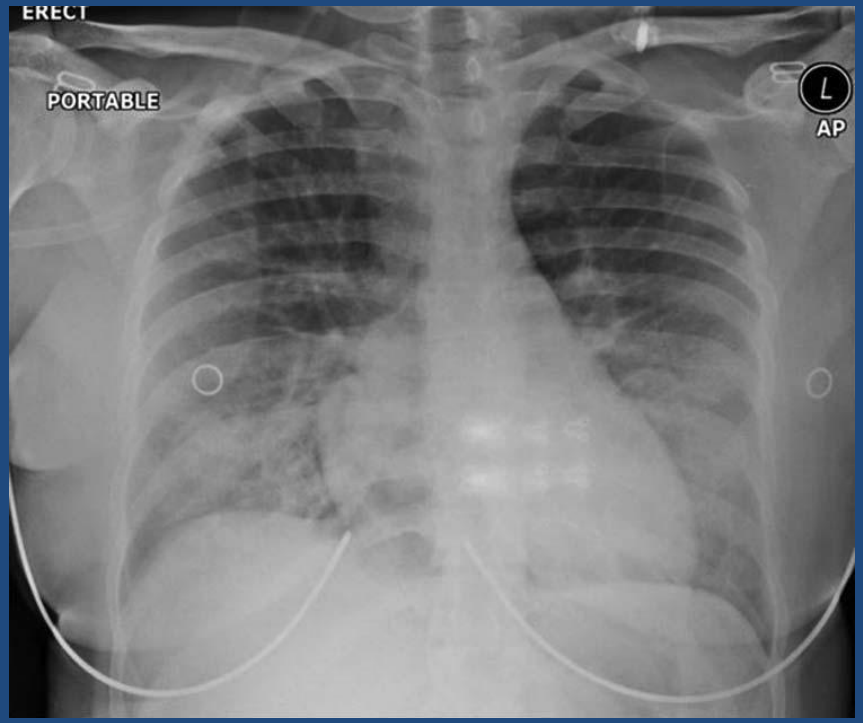
- A 35-year-old woman, h/o miscarriage
- Past history – treatment for symptomatic anaemia

- Admitted at 24 weeks' gestation with threatened pre-term labour

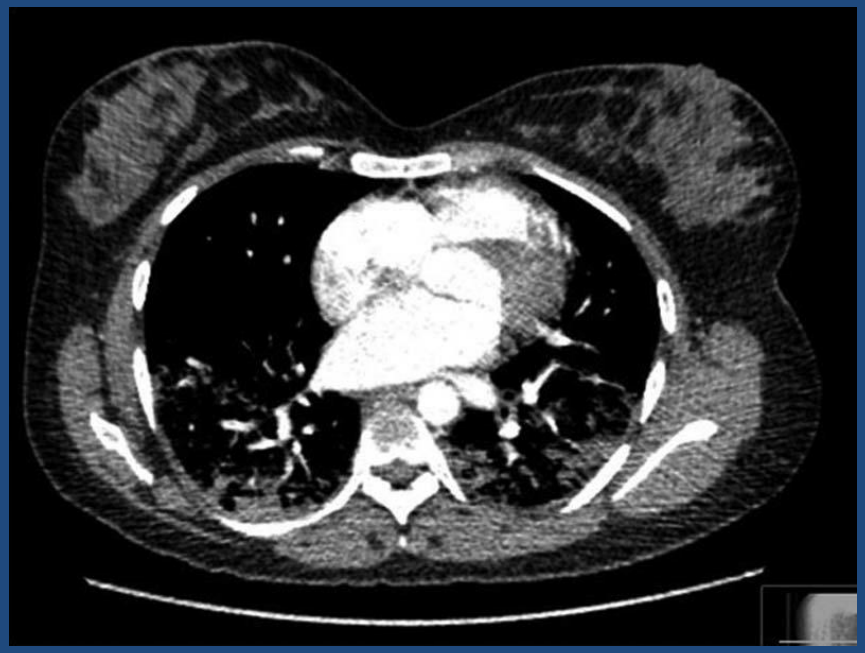
- She was given antenatal corticosteroids for fetal lung maturity and magnesium sulfate
- The contractions settled with magnesium sulfate and 28 h after magnesium therapy, her membranes ruptured spontaneously draining clear liquor and 18 h later she delivered a live infant vaginally
- She required transfusion of 1 unit of whole blood

- Within 30 min of delivery, she ‘felt wheezy’ and was found to be profoundly hypoxic
- Pulse 120/min, BP 120/70 mm Hg, RR 24/min, normal JVP, normal heart sounds but-
- Chest examination revealed bilateral crepitations
- ABG was suggestive of hypoxia (saturations of 88% on room air with a partial pressure of oxygen of 6.8 kPa)

- A chest X-ray showed increased pulmonary vasculature



- CT pulmonary angiogram was normal but dependent air space opacities were consistent with acute non-cardiogenic pulmonary oedema



- Echocardiogram – normal valve morphology and good biventricular function
- The placental histology subsequently showed chorioamnionitis

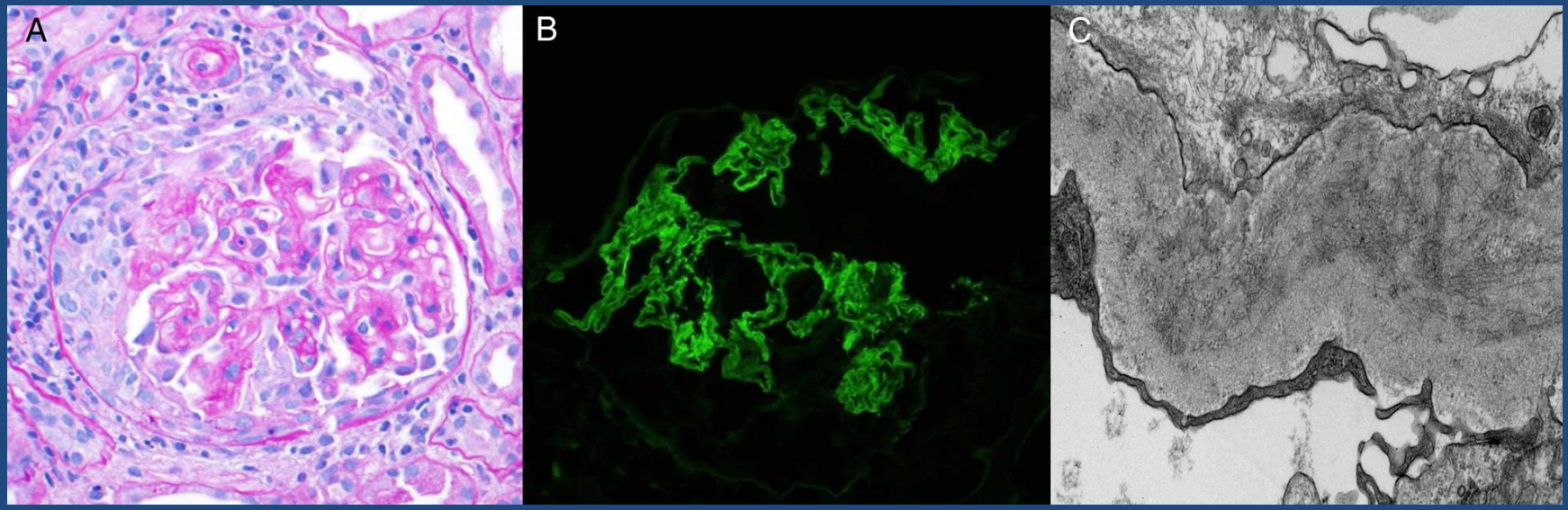
Treatment

- High dependency area
- 60% humidified high-flow oxygen
- Intravenous antibiotics and other supportive managements

Case 3

- A 68-year-old woman , DM, RA
- Progressive SOB and altered mental status
- 2 weeks prior - upper respiratory tract infection
- 1+ pitting edema
- Serum creatinine (Cr) was increased at 4.0 mg/dl
- Urine 3+ proteinuria, 10–15 RBC/HPF, eosinophils (>5%)
- ANA, ANCAs – negative, anti-GBM IgG positive at 272 units/ml (reference range 0–19 units/ml)
- CXR – bilateral infiltrates

Kidney biopsy findings showing FGN with anti-GBM disease. (A) Glomerulus with cellular crescent and mild mesangial matrix expansion. Associated interstitial inflammation is noted (periodic acid–Schiff, 40×). (B) Immunofluorescence staining with IgG demonstrates linear GBM staining as well as segmental smudgy mesangial staining. (C) Electron microscopy shows randomly oriented nonbranching fibrils distributed within mesangium as well as within the peripheral capillary loops.



Treatment

- IV methyl prednisolone followed by oral immunosuppressives
- Haemodialysis

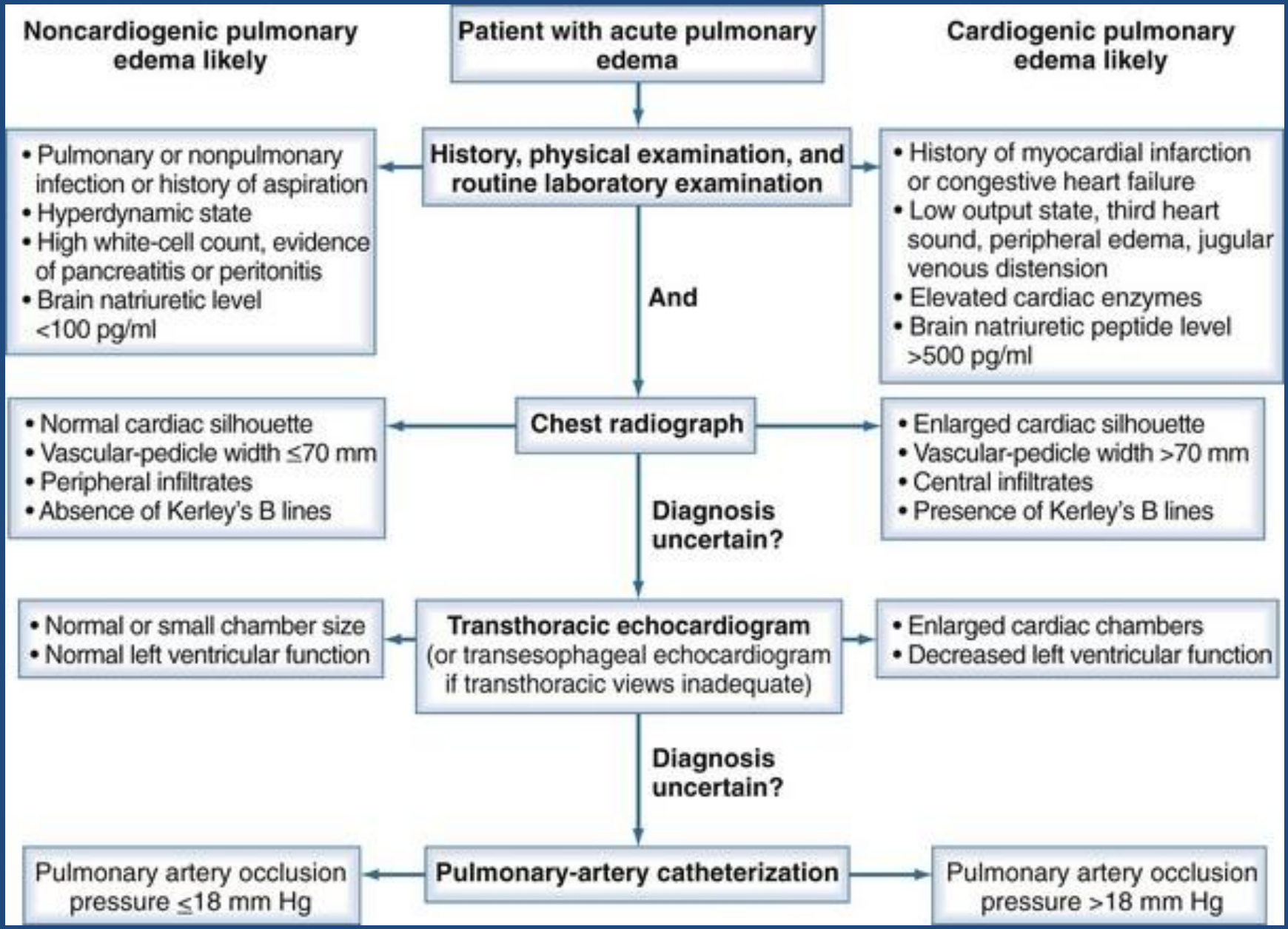
Case 4

- 63-year-old male, smoker, DM, HTN
- 1-h h/o chest pain, sweating, SOB
- Pulse 92/min, BP 165/92 mm Hg, RR 23/min
- Precordium – unremarkable, Chest – bi-basal creps
- ECG – ST-elevation V1-V4
- CXR – pulmonary oedema
- ECHO – antero-septal hypokynesia, EF 47%

Treatment

- Transfer to Cath Lab
- Primary PCI

Difference between cases 1-3 *vs* case 4


Noncardiogenic pulmonary edema likely
Patient with acute pulmonary edema
Cardiogenic pulmonary edema likely
• Pulmonary or nonpulmonary infection or history of aspiration
• Hyperdynamic state
• High white-cell count, evidence of pancreatitis or peritonitis
• Brain natriuretic level <100 pg/ml
History, physical examination, and routine laboratory examination
• History of myocardial infarction or congestive heart failure
• Low output state, third heart sound, peripheral edema, jugular venous distension
• Elevated cardiac enzymes
• Brain natriuretic peptide level >500 pg/ml
And
• Normal cardiac silhouette
• Vascular-pedicle width ≤70 mm
• Peripheral infiltrates
• Absence of Kerley's B lines
Chest radiograph
• Enlarged cardiac silhouette
• Vascular-pedicle width >70 mm
• Central infiltrates
• Presence of Kerley's B lines
Diagnosis uncertain?
• Normal or small chamber size
• Normal left ventricular function
Transthoracic echocardiogram (or transesophageal echocardiogram if transthoracic views inadequate)
• Enlarged cardiac chambers
• Decreased left ventricular function
Diagnosis uncertain?
Pulmonary artery occlusion pressure ≤18 mm Hg
Pulmonary-artery catheterization
Pulmonary artery occlusion pressure >18 mm Hg

Case 5

Int J Obs Anaes 2009 Jul;18(3):268-71. doi: 10.1016/j.ijoa.2009.02.003. Epub 2009 May 22.

Prone positioning for ARDS following blunt chest trauma in late pregnancy.

Kenn S, Weber-Carstens S, Weizsaecker K, Bercker S

Abstract

After a road traffic accident a pregnant patient at 34 weeks of gestation developed ARDS following blunt chest trauma, for which she required mechanical ventilation. Twenty-four hours after the accident, ongoing severe hypoxaemia with atelectasis mainly in the dorsal parts of the lung led to the decision to manage the patient in the prone position. Prone positioning over 8h resulted in a persistent improvement of oxygenation, which allowed extubation the following day. At term, however, our patient was admitted with dyspnoea, chest pain, haemodynamic instability and fetal bradycardia, for which she required emergency caesarean section followed by thoracotomy for haemothorax, from which she eventually made a full recovery. We have demonstrated that prone positioning can be used safely and effectively in a pregnant patient. It might be superior to other therapeutic options for improvement of oxygenation in pregnant patients. Careful positioning avoiding any external abdominal pressure and continuous fetal monitoring are mandatory.

Case 6

BMJ Case Reports

Novel diagnostic procedure

Utility of lung ultrasound in near-drowning victims

Christian B Laursen, Jesper Rømhild Davidsen, Poul Henning Madsen

Department of Respiratory Medicine, Odense University Hospital,

Correspondence to Dr Christian B Laursen, Christian.b.laursen@ouh.regionsyddanmark.dk

Summary

Drowning and near-drowning are common causes of accidental death worldwide and respiratory complications such as non-cardiogenic pulmonary oedema, acute respiratory distress syndrome and pneumonia are often seen. In other settings lung ultrasound can accurately diagnose these conditions; hence lung ultrasound may have a potential role in the evaluation of drowning or near-drowning victims. In this case report the authors describe a 71-year-old man who was brought to hospital with acute respiratory failure after a near-drowning accident. Lung ultrasound showed multiple B-lines on the anterior and lateral surfaces of both lungs, consistent with pulmonary oedema. Focus assessed transthoracic echocardiography showed no pericardial effusion and a normal global left ventricular function. Based on these findings the patient was diagnosed as having non-cardiogenic pulmonary oedema. Subsequent chest x-ray showed bilateral infiltrates consistent with pulmonary oedema. The case report emphasises the clinical value of lung ultrasound in the evaluation of a near-drowning victim.

Case 7

Edema pulmonar no cardiogénico recurrente tras la administración oral de hidroclorotiazida

Recurrent non-cardiogenic lung edema secondary to the oral administration of hydrochlorothiazide

La hidroclorotiazida (HCT) es un diurético de la familia de las tiazidas muy empleado para la hipertensión arterial sistémica (HTA) y estados edematosos[1]. Son bien conocidos sus efectos secundarios más comunes como: alteraciones electrolíticas, debilidad, espasmos musculares, trastornos gastrointestinales e hiperuricemia. Pero hay descritas reacciones idiosincrásicas menos frecuentes y de mayor gravedad como hipotensión y edema agudo pulmonar (EAP).

Presentamos el caso de una paciente que desarrolló un cuadro de hipersensibilidad a HCT consistente en EAP no cardiogénico.

Se trata de una mujer de 83 años sin alergias medicamentosas conocidas, HTA de larga evolución en tratamiento con bloqueantes de receptores AT_1 de la angiotensina II, diabetes mellitus tipo 2, dislipidemia, hemorragia digestiva secundaria a úlcera duodenal hace años y artrosis generali-

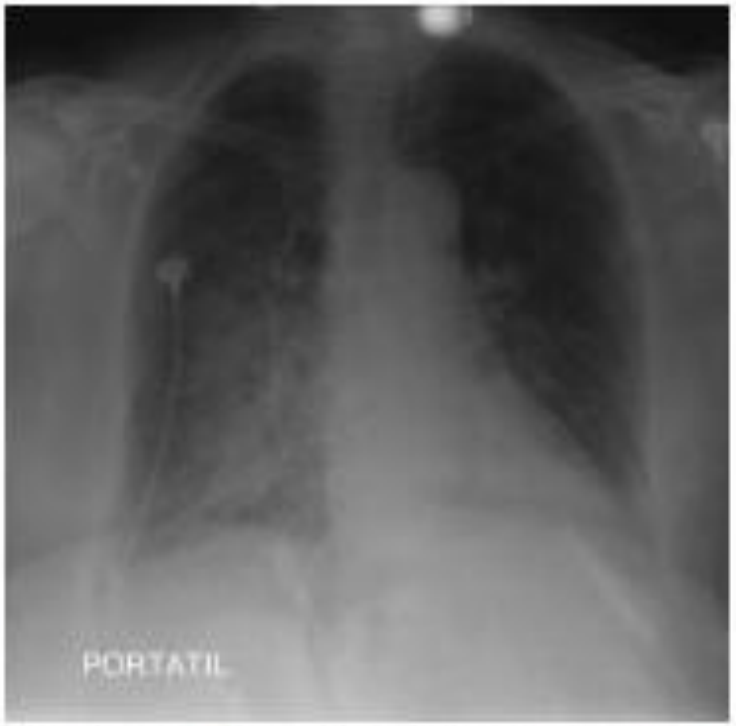


Figura 1 Infiltrados alveolo-intersticiales bilaterales compatibles con EAP.

Case 8

Non-cardiogenic Pulmonary Edema and Life-Threatening Shock Due to Calcium Channel Blocker Overdose: A Case Report and Clinical Review

Tauseef Afaq Siddiqi MD, Jennifer Hill MD, Yvonne Huckleberry PharmD BCPS, and Sairam Parthasarathy MD

Calcium channel blockers (CCBs) overdose can be life-threatening when manifest as catastrophic shock and non-cardiogenic pulmonary edema. We describe a case of massive overdose of multiple medications, including sustained-release verapamil, which was resistant to conventional support. Initial treatment for CCB overdose is primarily supportive, and includes fluid resuscitation. The mechanism of non-cardiogenic pulmonary edema is not well known, and reported cases have been successfully treated with mechanical ventilation. Circulatory shock may fail to respond to atropine, glucagon, and calcium in severely poisoned patients, and vasopressors are usually required. Attempting to overcome calcium-channel antagonism with the supra-therapeutic doses of calcium salts is clinically indicated to reverse hypotension and bradycardia. There is evidence that hyperinsulinemia-euglycemia therapy is superior to other therapies for CCB poisoning, and the mechanism is thought to be the insulin-mediated active transport of glucose into the cells, which counters the CCB-induced intra-cellular carbohydrate-deficient state. Conventional decontamination measures are ineffective in accelerating clearance of CCB. Experience with intravenous lipid emulsion for lipophilic drug overdose, such as verapamil, is limited, but has been proposed as a rescue therapy and might improve cardiac inotropy through intravascular sequestration of the lipophilic CCB. *Key words: calcium channel blockers; overdose; shock; toxicology; verapamil; hyperinsulinemia; euglycemia therapy; intravenous lipid emulsion; ARDS; pulmonary edema.* [Respir Care 2014;59(2):e15–e21.
© 2014 Daedalus Enterprises]

Case 9

Clinical Medicine & Research
Volume 10, Number 3: 131-136
©2012 Marshfield Clinic
clinmedres.org

Case Report

Non-Cardiogenic Pulmonary Edema Complicating Electroconvulsive Therapy: Short Review of the Pathophysiology and Diagnostic Approach

Janaki R. Manne, MD; Yusuf Kasirye, MD; Narendranath Epperla, MD; and Romel J. Garcia-Montilla, MD, PhD, MSc

Acute pulmonary edema complicating electroconvulsive therapy is an extremely uncommon event that has rarely been described in the literature. Different theories, including one suggesting a cardiogenic component, have been proposed to explain its genesis. The present report describes a classic presentation of this condition with review of its potential mechanisms and diagnostic approach. After successful completion of a session of electroconvulsive therapy, a 42-year-old woman with major depressive disorder developed acute systemic high blood pressure, shortness of breath, and hemoptysis. A chest radiograph demonstrated diffuse bilateral pulmonary infiltrates. Initially cardiogenic pulmonary edema was presumed, but an extensive diagnostic work-up demonstrated normal systolic and diastolic left ventricular function, and with only supportive measures, a complete clinical and radiographic recovery was achieved within 48 hours. The present case does not support any cardiogenic mechanism in the genesis of this condition.

Keywords: Airway obstruction; Electroconvulsive therapy; Pulmonary edema

Principle of treatment of non-cardiogenic pulmonary oedema

- Admission in HDU/ICU
- Oxygenation including use of mechanical ventilators
- Reduce lung inflammation
- Treatment of underlying cause

Summary

- Non-cardiogenic pulmonary oedema – not an uncommon entity
- May occur in wide-spread clinical contexts – common and uncommon
- High index of suspicion in needed
- Sometimes invasive diagnostics are needed
- Treatment is aimed to increase oxygenation by using ± ventilators and addressing underlying cause

Thank you all