ICU GRAND ROUND

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FHKCA FHKCA(IC) FJFICM FANZCA FHKAM

- Peer Review of Interesting Case
- Scientific Discussion

Not too long ago, in a Labour Room not too far away in this Galaxy…..

CASE PRESENTATION

- F/33 G3P1
- Gestation: 39 weeks
- Previous delivery: NSD
- Good past health
- Admitted to Labour room for show and regular contraction in an early morning
- Assessed by Obstetrician at around 10 am: for syntocinon augmentation

CASE PRESENTATION

- Epidural analgesia was performed:
- 18 G Catheter at L23, 5 cm catheter in-situ
- 1st dose: 9 ml 0.18% Marcain with fentanyl 13 ug
- Top-up: 7-15 ml 0.18% Marcain with fentanyl 1.5ug/ml Q1H prn
- Started at around 10 am

CASE PRESENTATION

- Patient developed BP drop, tachycardia, cyanosis and convulsion with teeth clenching for 30 seconds at around 1220
- FHR dropped to 60/min

ANAESTHETIST'S PROBLEMS ?

- Last EA top up dose: 9 ml at 1120

CASE PRESENTATION

- Clinical assessment:
- Fever spike of 39.5 C
- Peripheral shutdown, cold peripheries, SBP 120 mmHg (at 1223), P 98, HS Normal No murmurs
- SpO2 98% ,RR~25, equal breath sounds,
- Regain consciousness and barely obeyed command, lethargic
- Gelufusine 500 ml was given

CASE PRESENTATION

- Baby was delivered at 1228 by Vacuum extraction as cervix fully dilated, uneventful
- Blood loss: 300 ml, uterus 20 weeks contracted

WHAT'S UP ?

- ? Ideas ? Comments ? Questions

WHAT'S UP ?

- Anaesthetic Vs Obstetric Vs Medical issues

WHAT'S NEXT ?

BEDSIDE ECHO

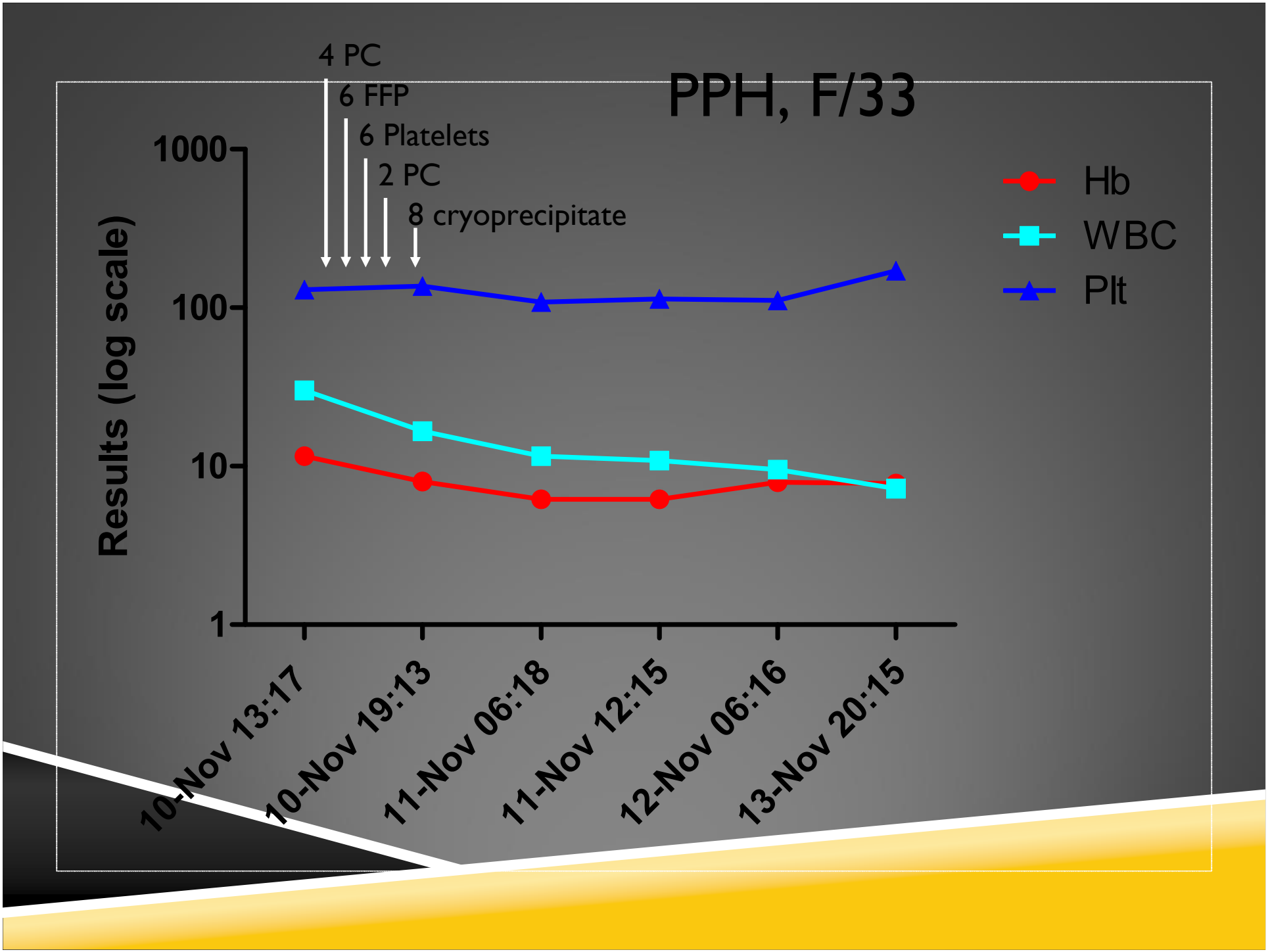
- EF 50% by eyeballing
- R heart not dilated
- No RVOT obstruction, no PR
- Normal chamber sizes and valves
- Mild decrease in LV systolic function
- No pericardial effusion
- No intra-cardiac shunt

BETTER PICTURE ?

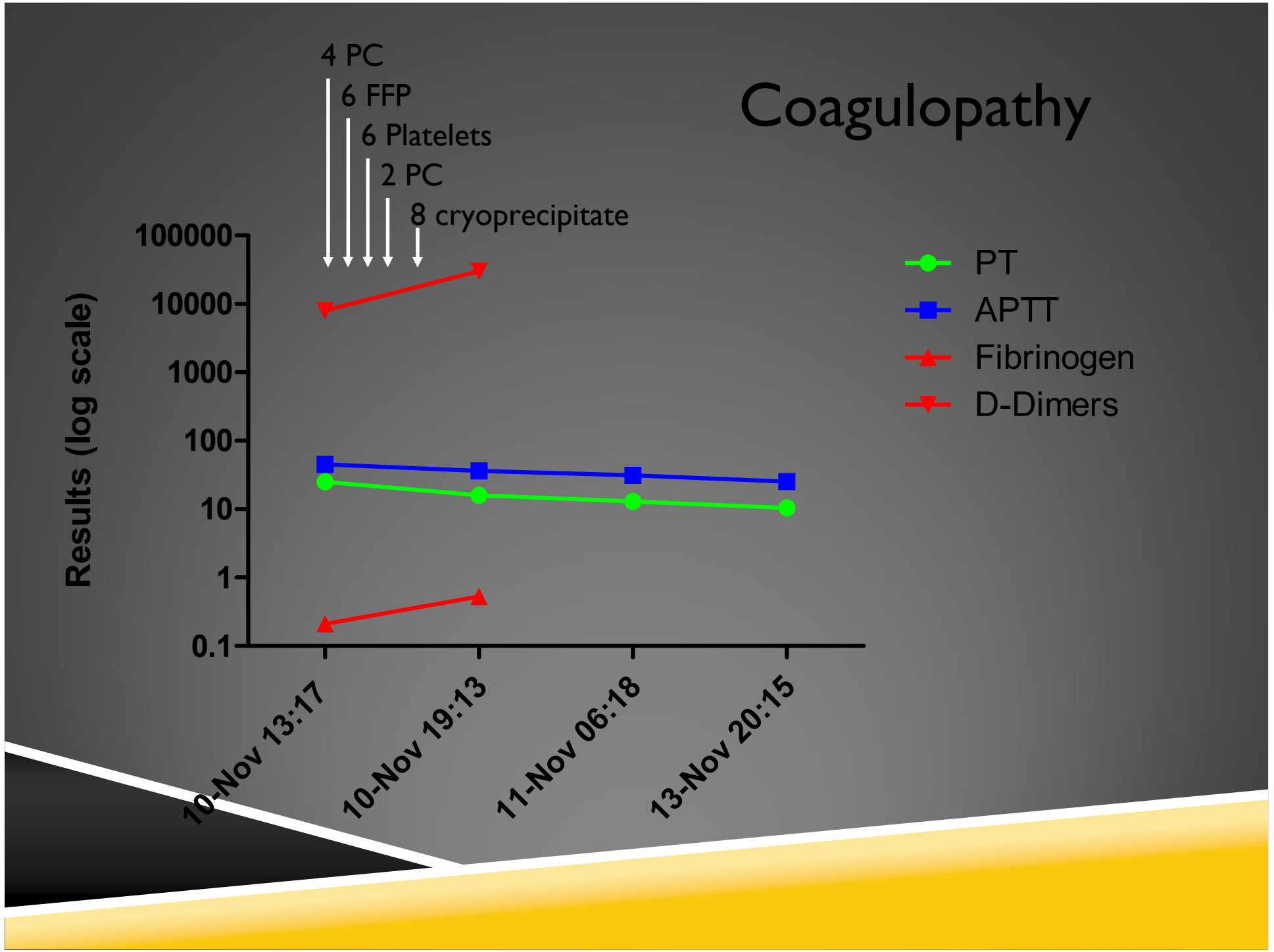
- Coagulation: profound DIC, hypofibrinogenemia
- Low platelet
- Biochemistry: compensated metabolic acidosis

PROGRESS

- Further blood loss from uterus
- IV Nalardo and syntocinon infusion was started


PPH, F/33
4 PC
6 FFP
6 Platelets
2 PC
8 cryoprecipitate
Hb
WBC
Plt
Results (log scale)
1000
100
10
1
10-Nov 13:17
10-Nov 19:13
11-Nov 06:18
11-Nov 12:15
12-Nov 06:16
13-Nov 20:15

Coagulopathy



PROGRESS

- Stabilized with no further BP drop episode
- No ICU bed available
- Vitals stable and observed in Labour ward for 1 day
- Uneventful discharge at Day 4 post delivery

- Clinical Diagnosis: Amniotic Fluid Embolism

AMNIOTIC FLUID EMBOLISM

- 1926- Meyer reported passage of amniotic fluid into the maternal circulation
- 1941- Steiner and Lushbaugh reported an autopsy series showing fetal mucin and squamous cells in the pulmonary vasculature of eight women who died of sudden shock during labor

DEFINING THE SYNDROME

- UK and US Registries(Clark and Tuffnell et al):
  - acute hypotension or cardiac arrest
  - acute hypoxia
  - coagulopathy
  - onset during labor, cesarean delivery or dilation and evacuation or within 30 min of evacuation of the uterus

HOW SIGNIFICANT ?

- between 1 in 8000 and 1 in 80,000 deliveries
- reported mortality rates as high as 60% even with aggressive and immediate treatment (Clark SL et al. Am J Obstet Gynecol 1995)
- 27% in a population-based study performed in 1999 and 37% in the UK registry from 2005
- Better ICU care ? Denominator effect ?

HOW SIGNIFICANT ?

- only 15% of survivors may be neurologically intact
- Neonatal mortality rate : 20%–25%, only 50% may be neurologically intact (Clark and Tuffnell)

CLINICAL PRESENTATION

- classically occurs acutely during labor and delivery or in the immediate postpartum period
- as late as 48 hrs postpartum or following cesarean delivery, amniocentesis, or removal of the placenta or with first and second trimester abortions

CLINICAL PRESENTATION

- Risk factors:
- turbulent labor, trauma, multi- parity, use of oxytocin, increased maternal age, increased gestational age, male fetus, and cesarean section
- Inconsistent support for the association of these various factors to amniotic fluid embolus

Table 1. Cardinal signs/symptoms of amniotic fluid embolism syndrome

Hypoxemia
Shock (typically obstructive, cardiogenic, or distributive)
Coagulopathy/disseminated intravascular coagulation
Altered mental status/hypoxic encephalopathy

Clark SL, Hankins G, Dudley DA, et al: Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol* 1995

- Incidence: between 80% and 100%
- Rapid patient deterioration and death often result from the abrupt onset and fulminant course of these symptoms
- Mortality has been reported as high as 50% in the first hour after presentation due to cardiopulmonary manifestations

Table 2. Other common presenting signs/ symptoms of amniotic fluid embolism

Seizure activity
Confusion
Agitation
Constitutional (fever, chills, headache, nausea, vomiting)
Evidence of fetal distress (late decelerations, bradycardia)

Table 2. Differential Diagnosis of Amniotic Fluid Embolism

Obstetric causes
Acute hemorrhage
Placental abruption
Uterine rupture
Uterine atony
Eclampsia
Peripartum cardiomyopathy
Anesthetic causes
High spinal anesthesia
Aspiration
Local anesthetic toxicity
Nonobstetric causes
Pulmonary embolism
Air embolism
Anaphylaxis
Sepsis/septic shock

Karetsky M, Ramirez M. Acute respiratory failure in pregnancy: an analysis of 19 cases. Medicine (Baltimore) 1998;77:41–9

HYPOXIA

- 93% of patients according to the national registry
- often seen concomitantly with respiratory arrest and cyanosis during the initial presentation

HYPOXIA

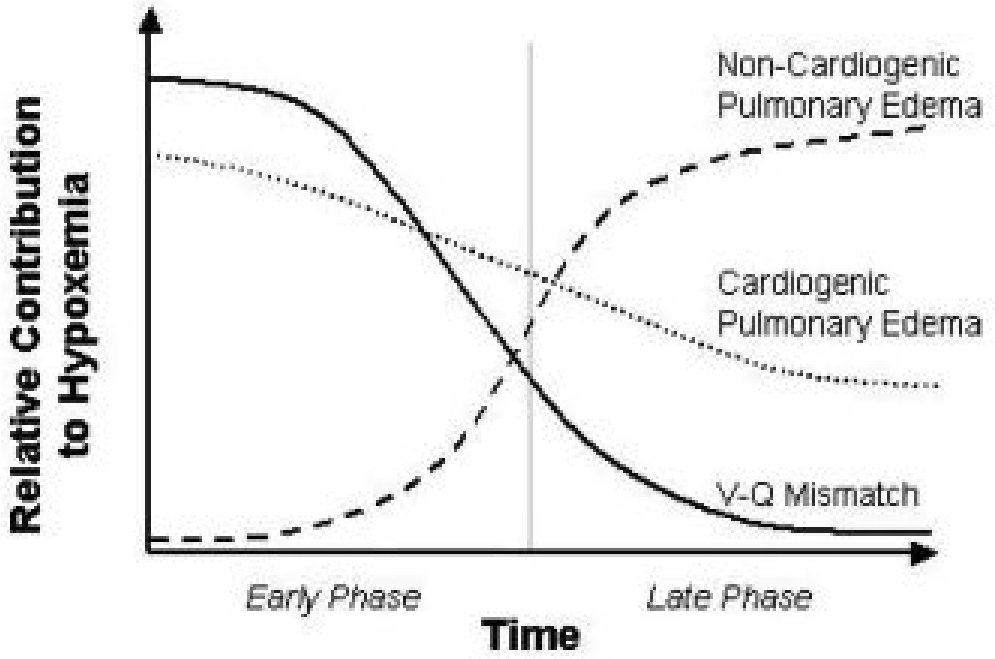


Figure 1. Postulated relative contributions to hypoxemia in patients with amniotic fluid embolism.

- widespread neurologic deficiencies and brain death

SHOCK

- uniformly present for patients with severe disease
- early phase: pulmonary hypertension is thought to be due to pulmonary vasospasm and the onset of left ventricular dysfunction
- Cardiac arrhythmias including bradycardia, asystole, ventricular fibrillation, and pulseless electrical activity may also be present

SHOCK

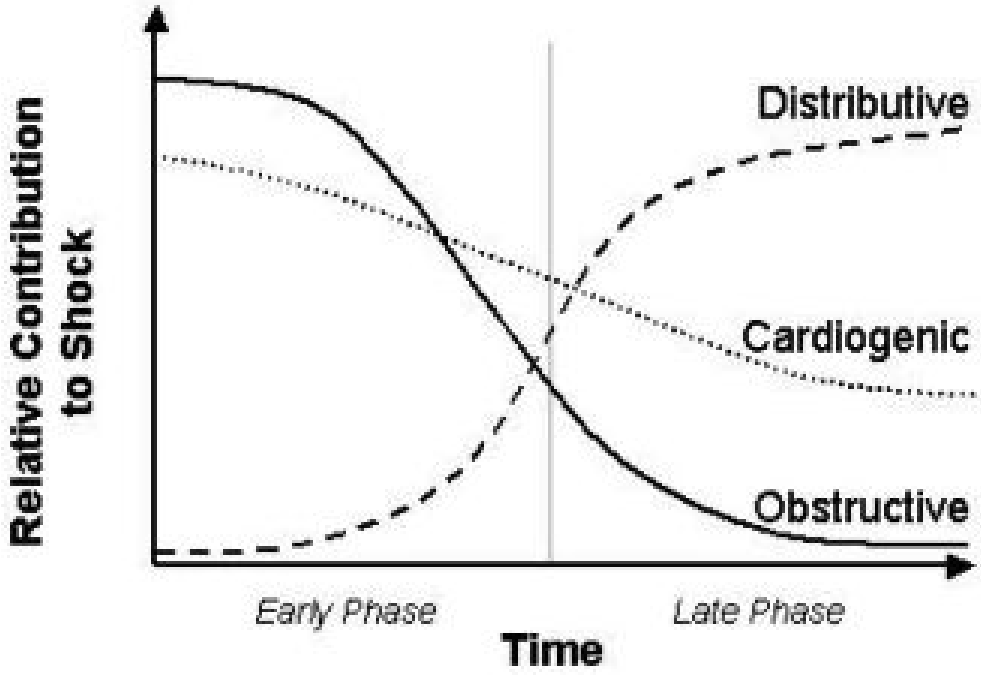


Figure 2. Postulated relative contributions to shock in patients with amniotic fluid embolism.

SHOCK

- Case report : transesophageal echocardiography during the early phase of disease: right ventricular failure, suprasystemic right-sided pressures, bulging of the interatrial and interven- tricular septum from right to left, and severe tricuspid regurgitation

Farrar SC, Gherman RB: Serum tryptase in a woman with amniotic fluid embolism: A case report. J Reprod Med 2001; 46:926 –928

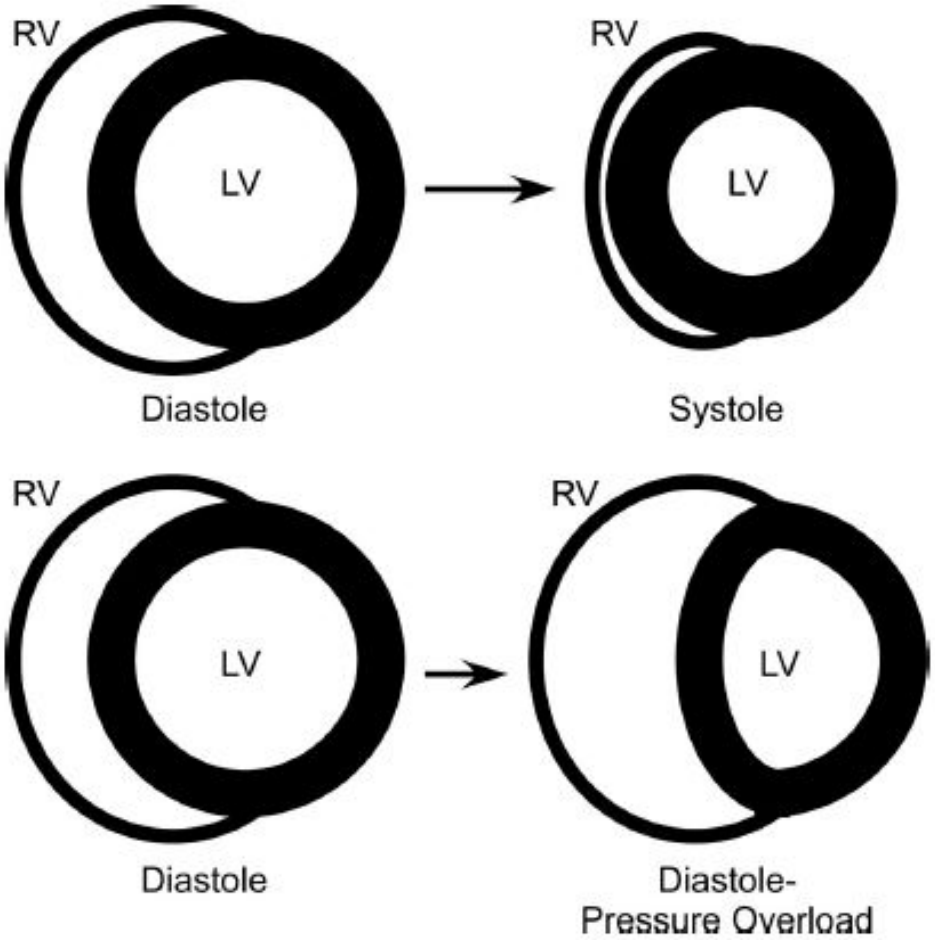
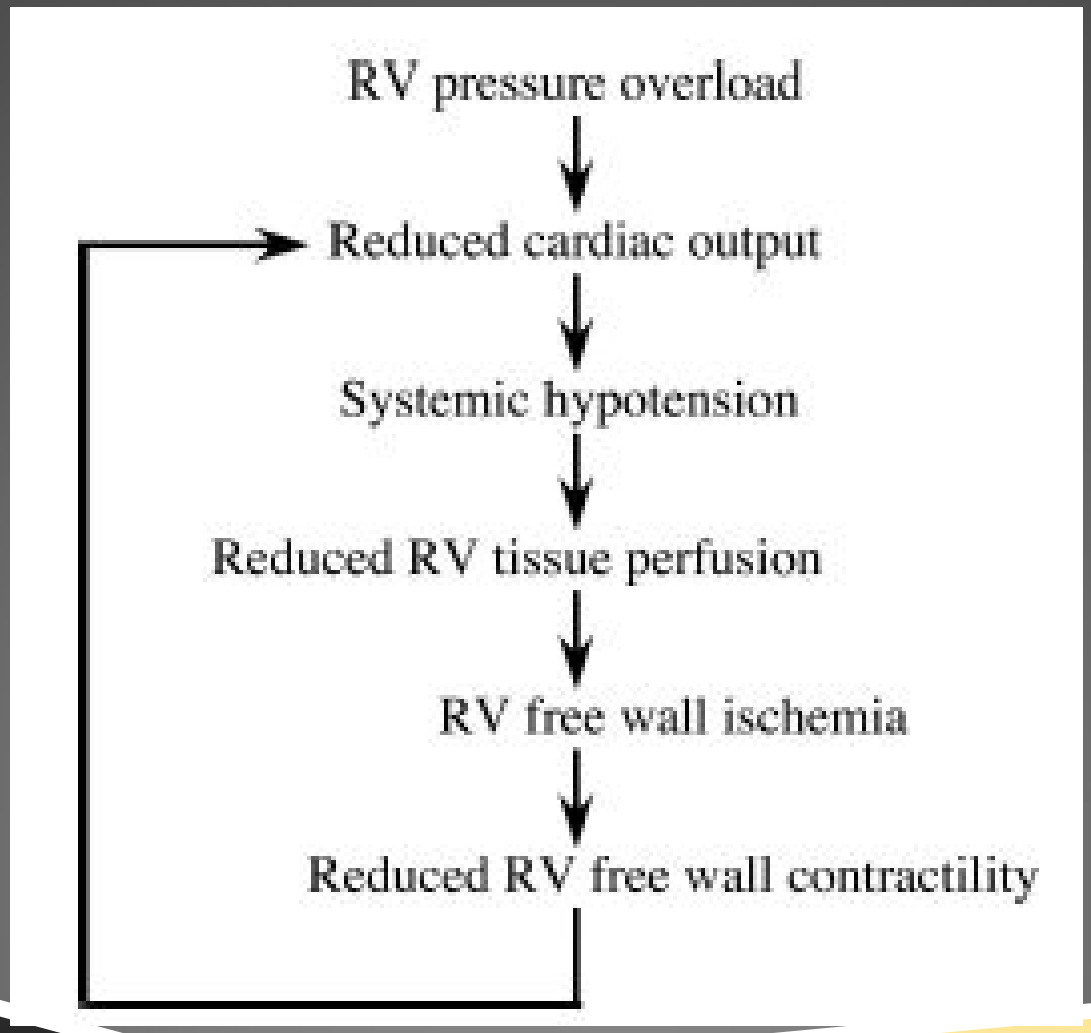


Figure 1. Illustration of how the geometry of the right ventricle (*RV*) changes with contraction and is affected by pressure overload. *Top*, the crescentic RV flattening in systole, leading to a large-volume change with minimal change in RV free wall area. *Bottom*, how a shift of the interventricular septum during acute pressure overload permits RV end-diastolic volume to increase with no change in end-diastolic RV free wall area, a decrease in interventricular septal surface area, and a corresponding decrease in left ventricle (*LV*) end-diastolic volume. Because the RV free wall does not stretch, there is no recruitment of RV function via the Frank-Starling mechanism, while at the same time there is a loss of function via the Frank-Starling mechanism in the LV.


RV pressure overload
Reduced cardiac output
Systemic hypotension
Reduced RV tissue perfusion
RV free wall ischemia
Reduced RV free wall contractility

SHOCK

- patients who survive the first several hours of amniotic fluid embolism often have improvement in left ventricular function
- Cardiac indexes from various reports range between 2.1 and 6.9
- “septic shock-like” process may be significant during the later phase

SHOCK

- Hemorrhagic shock
- poor prognostic factor
- Different forms of shock may be present simultaneously

DIC

- 83% of patients with amniotic fluid embolism, and half of these patients develop coagulopathy within 4 hrs of initial presentation
- profound hemorrhage
- Can present solely as main clinical manifestation
- Early: substances in amniotic fluid
- Late: SIRS

ALTERED MENTAL STATUS

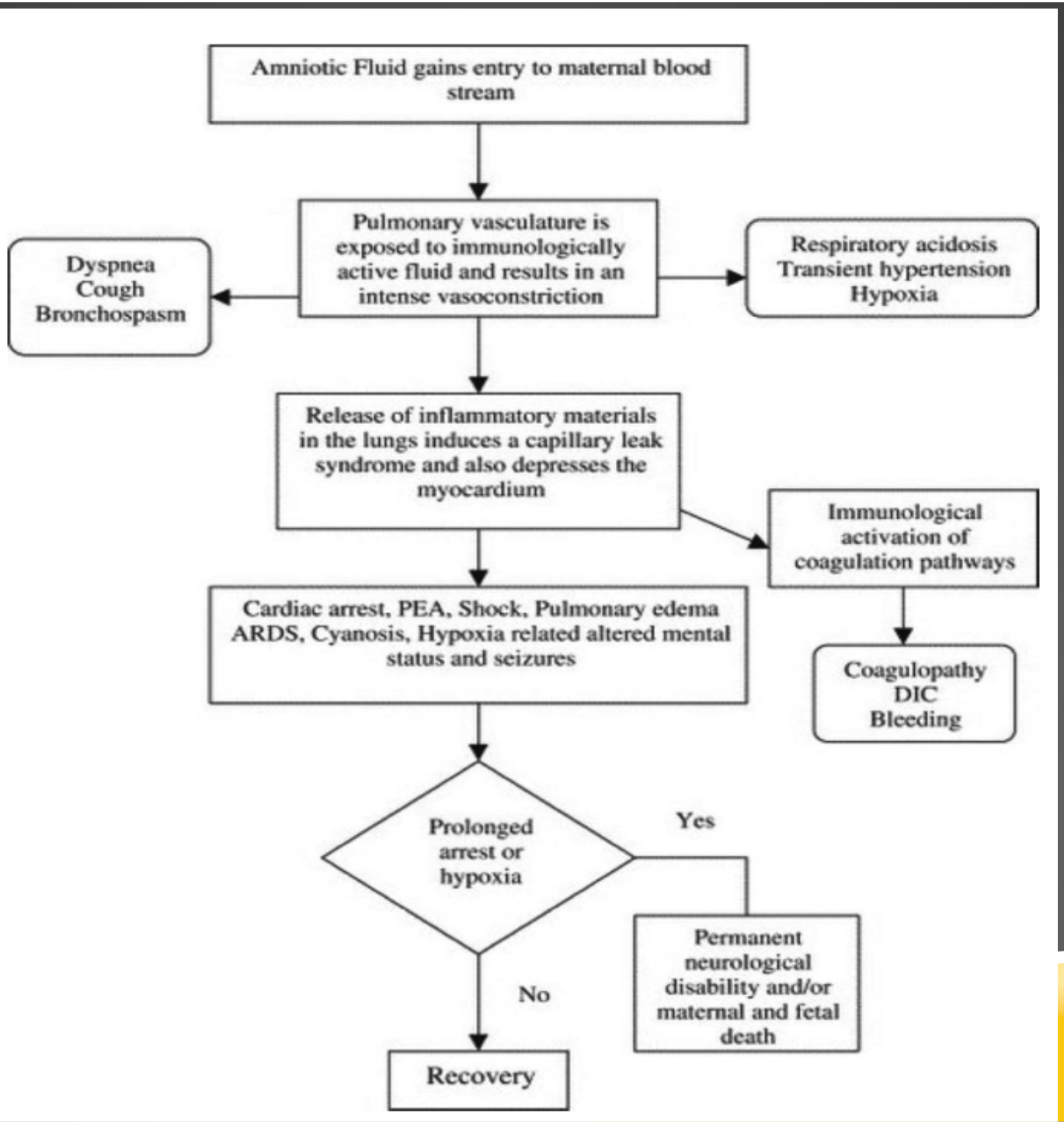
- **Hypoxia**
- direct effect on the central nervous system by either vascular obstruction or other pathways remains unproven
- 85% of patients may suffer permanent neurologic sequelae as a result of amniotic fluid embolism
- Can present solely as main clinical manifestation

ALTERED MENTAL STATUS

- **Seizure** activity is present in 50% of patients and exacerbate **brain injury**
- **Encephalopathy** remains one of the most common causes of morbidity in survivors of amniotic fluid embolism
- less severe presentation of amniotic fluid embolism with variable incidence of major symptoms and a milder clinical course

PATHOGENESIS

- Unclear
- Endocervical veins
- Amniotic fluid components have been demonstrated in pregnant women without clinical evidence of amniotic fluid embolism


Amniotic Fluid gains entry to maternal blood stream
Pulmonary vasculature is exposed to immunologically active fluid and results in an intense vasoconstriction
Dyspnea
Cough
Bronchospasm
Respiratory acidosis
Transient hypertension
Hypoxia
Release of inflammatory materials in the lungs induces a capillary leak syndrome and also depresses the myocardium
Immunological activation of coagulation pathways
Cardiac arrest, PEA, Shock, Pulmonary edema ARDS, Cyanosis, Hypoxia related altered mental status and seizures
Coagulopathy
DIC
Bleeding
Prolonged arrest or hypoxia
Yes
No
Permanent neurological disability and/or maternal and fetal death
Recovery

DIAGNOSIS

- Clinical diagnosis
- diagnosis of exclusion
- acute, abrupt, and dramatic presentation with profound shock and cardiovascular collapse associated with severe respiratory distress
- DIC may be the first sign
- restlessness and alterations in mental status

DIAGNOSIS

- no specific laboratory tests
- Experimental: monoclonal TKH-2 antibodies in maternal serum and lung tissue, zinc coproporphyrin, tissue factor, tryptase level
- definitively confirmed postmortem by the presence of fetal squamous cells and other elements of fetal debris in the pulmonary vessels

MANAGEMENT

- no prophylactic measures to prevent the development of amniotic fluid embolism
- rapid maternal cardiopulmonary stabilization
- prevent additional hypoxia and subsequent end-organ failure
- **immediate delivery** of the fetus

Table 4. General supportive measures in the treatment of amniotic fluid embolism

1. Treat hypoxia with 100% oxygen.
2. Treat hypotension by fluid resuscitation with isotonic solutions and vasopressors.
3. Treat left ventricular diminished contractility with fluids and inotropic therapy.
4. Treat DIC and coagulopathy with FFP, cryoprecipitate, fibrinogen, and factor replacement.
5. Treat hemorrhage with RBC transfusions and thrombocytopenia with platelets.

DIC, disseminated intravascular coagulation; FFP, fresh frozen plasma; RBC, red blood cells.

MANAGEMENT

- be prepared to **treat** the ***uterine atony*** with standard medical and surgical resuscitative methods, including aggressive blood product replacement, uterotonic medications, and a hysterectomy if indicated

Table 5. Newer strategies in the treatment of amniotic fluid embolism

1. Intra-aortic balloon counterpulsation (41)
2. Extracorporeal membrane oxygenation (41)
3. Cardiopulmonary bypass (17)
4. Plasma exchange transfusions (42, 43)
5. Uterine artery embolization (40, 44)
6. Continuous hemofiltration (43)
7. Cell-salvage combined with blood filtration (45)
8. Serum protease inhibitors (46)
9. Inhaled nitric oxide (46)
10. Inhaled prostacyclin (46)
11. High-dose corticosteroids (46)

Case Report

Successful Placement of a Right Ventricular Assist Device for Treatment of a Presumed Amniotic Fluid Embolism

Nimesh P. Nagarsheth, MD*

Sean Pinney, MD†

Adel Bassily-Marcus, MD‡

Anelechi Anyanwu, MD§

Lynn Friedman, MD*

Yaakov Beilin, MD*‖

Amniotic fluid embolism is a rare and often fatal complication of pregnancy. We report the successful multidisciplinary management of a woman who developed a coagulopathy from a presumed amniotic fluid embolism after forceps-assisted vaginal delivery requiring recombinant factor VIIa, and pulmonary arterial hypertension requiring a right ventricular assist device.

(Anesth Analg 2008;107:962–4)

PROGNOSIS

- improved significantly with **early recognition** of this syndrome and prompt and **early resuscitative measures**
- In some cases, death is inevitable despite early and appropriate management
- morbidity remains high with severe sequelae, particularly neurologic impairment
- Neonatal survival is reported at 70%

CONCLUSION

- a catastrophic disease
- high index of suspicion and rapid resuscitation
- Don’t “drown the right Heart”
- multidisciplinary approach
- Novel treatment maybe indicated in selected cases