

Management of the Critically ill Obstetric Patient

Dr. Saren Azer, MD, PhD,

**Fellow of Royal College of Physicians and Surgeons
of Canada,**

Clinical Immunologist, Internist, Intensivist,

My Professional Background

❖ Medical Background

- MD, University of Calgary, Canada,
- Internal Medicine Residency, University of British Columbia, Canada,
- Critical Care Subspecialty Training, University of Calgary, Canada,

❖ Research Background

- MSc and PhD training in Clinical Immunology, University of Alberta, Canada,
- Post-doctoral Fellowship in Respiratory Disease, University of Alberta, Canada,
- Parker B Francis Fellowship Training, Harvard University, United State,

❖ International Humanitarian Background,

- President of ISPHR from 1999 to 2015,
- MSF, In Various Capacities,
- International Red Cross and Red Crescent,



United Nations
Educational, Scientific and
Cultural Organization

**Tell me and I forget,
Teach me and I may remember,
Involve me and I learn**



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We Learn...

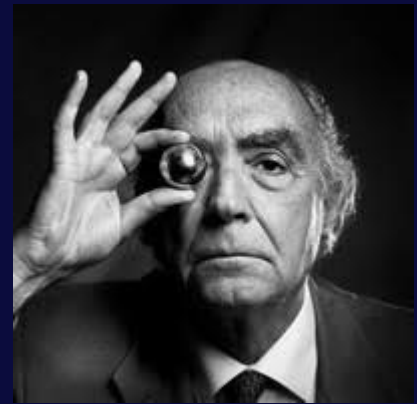
- **10% of what we READ**
- **20% of what we HEAR**
- **30% of what we SEE**
- **50% of what we SEE and HEAR**
- **70% of what is DISCUSSED with OTHERS**
- **80% of what is EXPERIENCED PERSONALLY**

José Saramago



- It is only when you are liberated from the burden of your eyes that you start to see, and hear and feel. Somehow it seems that our eyes are standing bearer between us and the world which lives vibrantly around us...

José Saramago

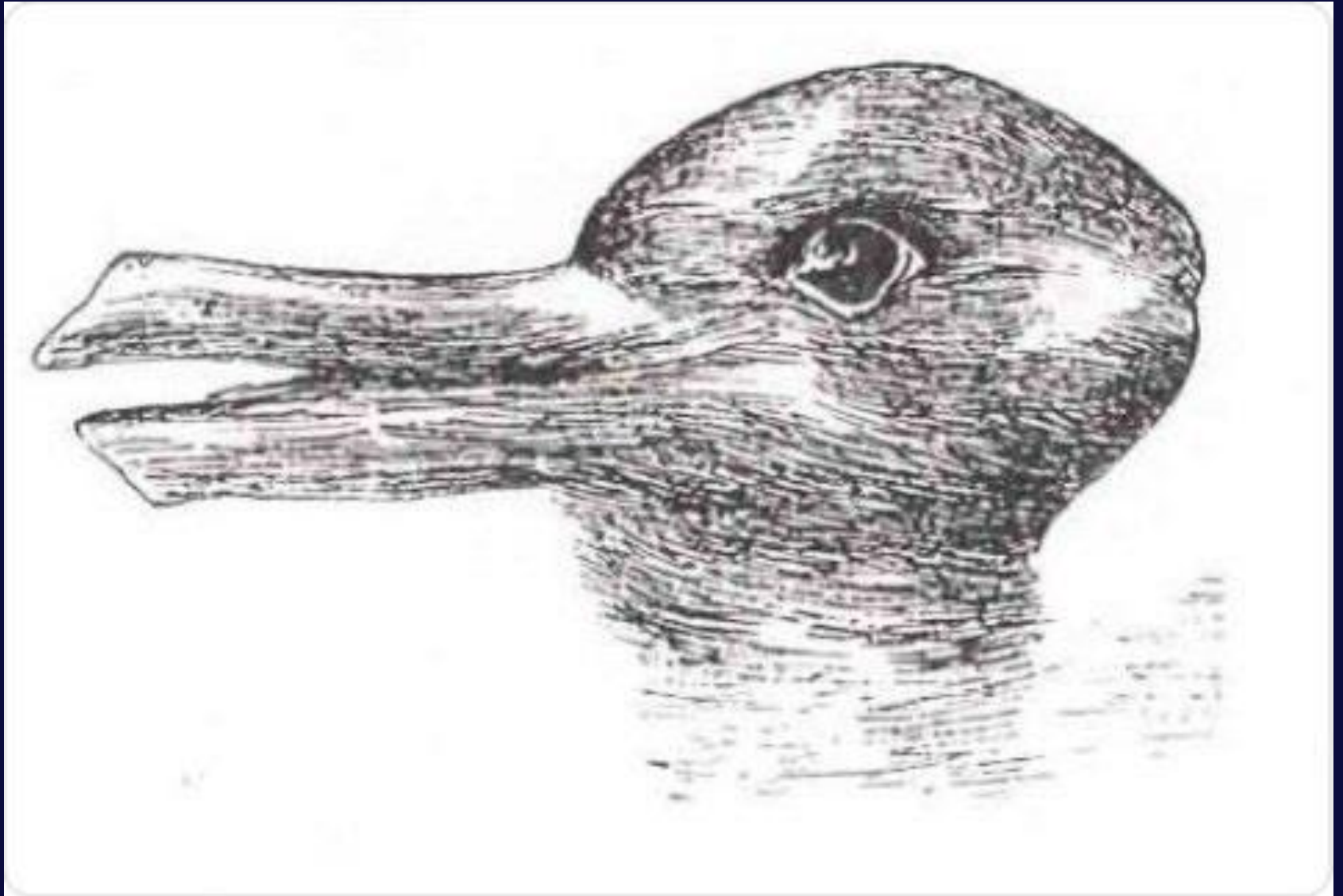


- The world suffers since we the human often look yet do not see, hear yet do not listen, and we touch yet do not feel
- For those who are set to make a difference in the madness of this world, they need to look harder, to listen deeper and touch softer...

Observation

- In an appropriate clinical encounter, always take a minute or so to look at the patient in their entirety, making your observations, if possible, from an out-of-the way perch.
- Look and see, listen and hear, touch and feel...use your every sense to assess your patient...
- Does the patient seem anxious, in pain, upset? What about their dress, hygiene, appearance? Remember, the exam begins as soon as you lay eyes on the patient. Does it look like that the patient might collapse...stop breathing...

What do you see?



What do you see?





There is a hidden
face in this
scenery, can you
find it?

pemahaman komprehensif
terhadap suatu kondisi

What do you see?



What do you see?



**“After Careful Observation, The
Heart of Every Clinical
Encounter is Vital Signs”**

Objectives of the Clinical Case is to implement,

- Medical Reasoning
- Evidence Based Approach
- Team Based Approach
- Focused and Efficient Approach
- Non-hierarchical Approach
- Patient Centered Approach
- (Your approach is entirely based on minimal history and complete physical exam)

Case # 1

- 28 years old female 32 weeks gestation presented with a single episode of loss of consciousness lasting about 20 seconds presenting to ER

Vitals Case # 1

- BP 90/54
- HR 138
- RR 28
- O2Sat 88%
- Temp. 38 C
- GCS 15
- BMI 32

Case # 1

- Your management

Case # 2

- 36 year old female 19 weeks gestation presenting with altered level of consciousness

Vitals Case # 2

- BP 76/54
- HR 144
- RR 34
- O2Sat 83%
- Temp. 41 C
- GCS 13
- BMI 34

Case #2

- Your Management

Case #3

- 23 years old female pregnant at term during advance stages of labor presenting with tonic colonic seizures.

Vitals Case # 3

- BP 210/110
- HR 144
- RR 30
- O2Sat 86%
- Temp. 40 C
- GCS 3
- BMI 28

Case #3

- Your Management

Case #4

- 24 years old female 5 days postpartum presenting with SOB and decreased exertional capacity ...

Vitals Case # 4

- BP 96/62
- HR 120
- RR 26
- O2Sat 89%
- Temp. 37 C
- GCS 15
- BMI 30

Case #4

- Your Management

Case #5

- 23 years old female day 2 postpartum with diffuse abdominal pain and sudden cardiac arrest ...

Case #5

- No pulse on palpation...how would you run this code...

Case #6

- 29 years old female, 6 hours postpartum found un-conscious in the bathroom with evidence of massive bleed.

Case #6

- No pulse on palpation...how would you run this code...

Case #7

- 39 years old female immediately after labor presenting with sudden onset of altered level of consciousness.

Vitals Case # 7

- BP 78/48
- HR 130
- RR 30
- O2Sat 81%
- Temp. 38.5 C
- GCS 6
- BMI 26

Case # 7

- Your Management

Case #8

- 32 years old female, 30 weeks gestation, presenting with several days history of productive cough, fever, chills and SOB.

Vitals Case # 8

- BP 90/52
- HR 140
- RR 36
- O2Sat 79%
- Temp. 39.2 C
- GCS 9
- BMI 28

Case # 8

- Your Management

Case #9

- 41 years old female, 28 weeks gestation, presenting with several days history of RUQ pain and tenderness, N/V and easy bruising.

Vitals Case # 9

- BP 164/102
- HR 134
- RR 26
- O2Sat 96%
- Temp. 38.4 C
- GCS 15
- BMI 31

Case # 9

- Your Management

Case #10

- 29 years old female, 21 weeks gestation, presenting with bilateral lower extremity pain, tenderness and swelling for several days.

Vitals Case # 10

- BP 114/72
- HR 84
- RR 16
- O2Sat 98%
- Temp. 38.0 C
- GCS 15
- BMI 34

Case # 10

- Your Management

Case #11

- 17 years old female, 29 weeks gestation, was involved in a motor vehicles accident, brought to the ER.

Vitals Case # 11

- BP 64/?
- HR 144
- RR 32
- O2Sat 74%
- Temp. 35.4 C
- GCS 3
- BMI ?

Case # 11

- Your Management

Case #12

- 19 years old female, 16 weeks gestation, was found unconscious at home, brought to the ER.

Vitals Case # 12

- BP 94/72
- HR 54
- RR 8
- O2Sat 84%
- Temp. 36 C
- GCS 3
- BMI ?

Case # 12

- Your Management

Case #13

- 21 years old female, 19 weeks gestation, presenting to ER with weeks history of progressive SOB, cough, orthopnea, and PND.

Vitals Case # 13

- BP 82/44
- HR 160
- RR 32
- O2Sat 87%
- Temp. 36 C
- GCS 15
- BMI 19

Case # 13

- Your Management

Case #14

- 44 years old female, 33 weeks gestation, with witnessed collapse, brought to ER.

Vitals Case # 14

- On arrival has no pulse...

Case # 14

- Your Management

Case #15

- 22 years old female, 15 weeks gestation, was found floating on cold river water, brought to the ER.

Vitals Case # 15

- On arrival has no pulse

Case # 15

- Your Management

Case #16

- 34 years old female, day 2 post NVD , was brought to the ER with sever SOB.

Vitals Case # 16

- BP 130/84
- HR 144
- RR 32
- O2Sat 85%
- Temp. 37.8 C
- GCS 13
- BMI 26

Case # 16

- Your Management

Case #17

- 41 years old female, day 8 post C-Section , with chest pain, cough, SOB, and palpitation brought to the ER.

Vitals Case # 17

- BP 84/58
- HR 138
- RR 28
- O2Sat 88%
- Temp. 38.2 C
- GCS 15
- BMI 32

Case # 17

- Your Management

Case #18

- 44 years old female, 32 weeks gestation, with a new onset prolonged seizure brought to ED.

Vitals Case # 18

- BP 154/105
- HR 128
- RR 24
- O2Sat 84%
- Temp. 39.0 C
- GCS 3
- BMI 30

Case # 18

- Your Management

Case #19

- 31 years old female, minutes after C-section under spinal anesthesia, presenting with sudden onset decreased level of consciousness.

Vitals Case # 19

- BP 92/46
- HR 142
- RR 30
- O2Sat 87%
- Temp. 38.0 C
- GCS 3
- BMI 28

Case # 19

- Your Management

Case #20

- 31 years old female, minutes after C-section under spinal anesthesia, presenting with sudden onset decreased level of consciousness.

Vitals Case # 20

- BP 92/46
- HR 142
- RR 30
- O2Sat 87%
- Temp. 38.0 C
- GCS 3
- BMI 28

Case # 20

- Your Management

Case #21

- 25 years old female, during C-section under spinal anesthesia, presenting with massive inter-operative bleed.

Vitals Case # 21

- BP 64/..
- HR 142
- RR 37
- O2Sat 82%
- Temp. 36.0 C
- GCS 3
- BMI 23

Case # 21

- Your Management

Case #22

- 27 years old female, 23 weeks gestation, post MVA, unconcise brought to ER.

Vitals Case # 22

- BP 172/104
- HR 58
- RR 28
- O2Sat 90%
- Temp. 38.2 C
- GCS 3
- BMI ?

Case # 22

- Your Management

Ultimate Global Objective

- Maternal Death is somewhat different from all other death in Intensive Care Unit and needs not to exist.
- Pregnancy is a physiological stress test and someone who is able to get pregnant and maintains a pregnancy should be able to overcome most of its potential complication
- WHO hope and aims that Maternal Mortality all but be eliminated

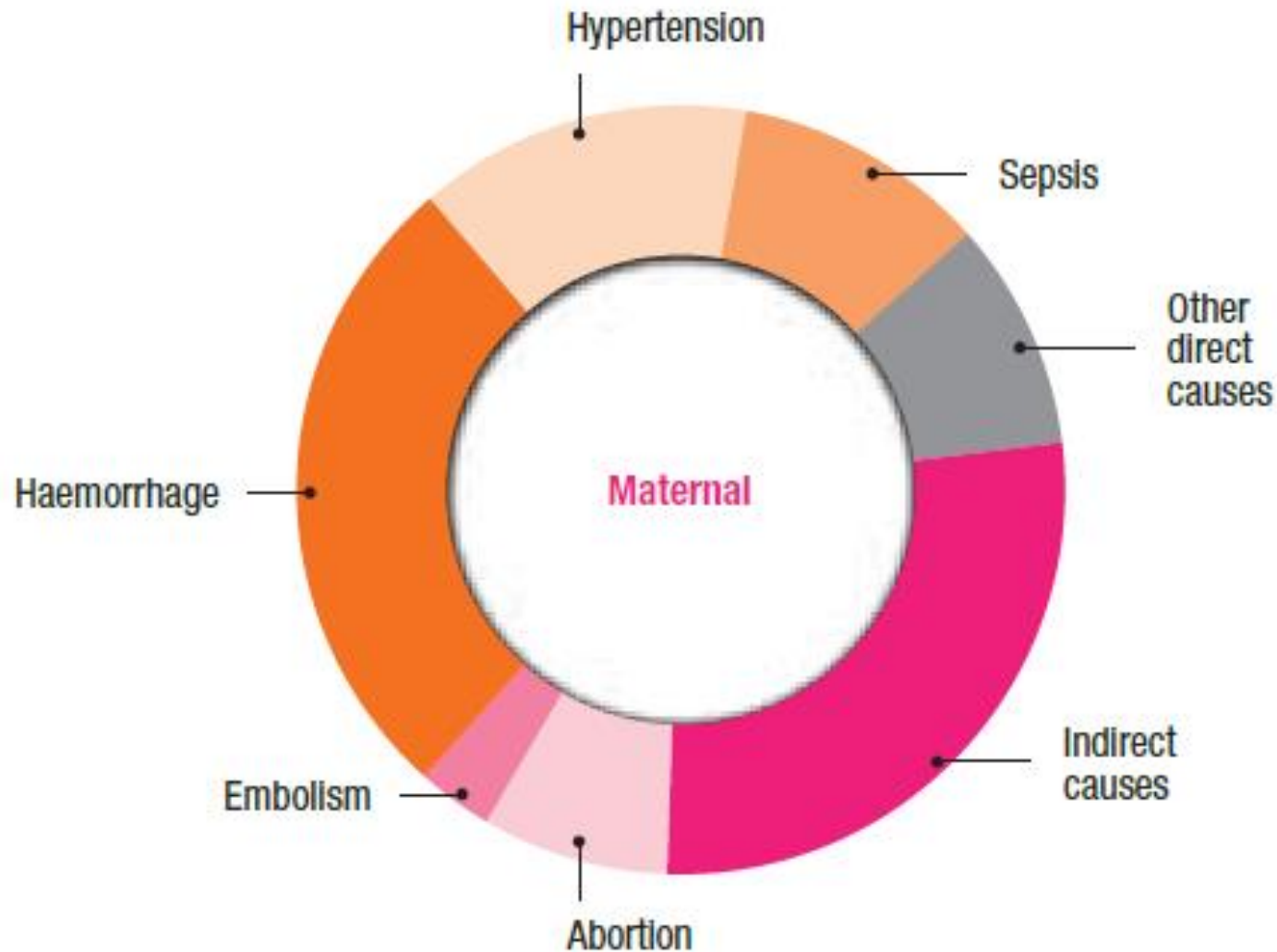
Workshop Objectives

- To facilitate an understanding of the current status of Maternal Mortality in Islamic Republic of Iran.
- To Identify areas in the current state of maternal mortality management where focused practical changes could be introduced with the anticipation that introduced changes could lead to decreased rate of Maternal Mortality.
- To facilitate a better understanding of Maternal Critical care and management of unstable maternal patients.
- To set the stage for the future collaborations with Obstetric/Gynecology and intensivist colleagues,

Causes of maternal mortality

- Maternal deaths are subdivided into direct and indirect obstetric deaths.
- Direct obstetric deaths result from obstetric complications of pregnancy, labor, or the postpartum period. They are usually due to one of five major causes hemorrhage, sepsis, eclampsia, obstructed labor, and complications of unsafe abortion.
- Indirect obstetric deaths result from previously existing diseases or from diseases arising during pregnancy (i.e. Rheumatic Heart Disease, SLE, Pulmonary Fibrosis, CKD...).

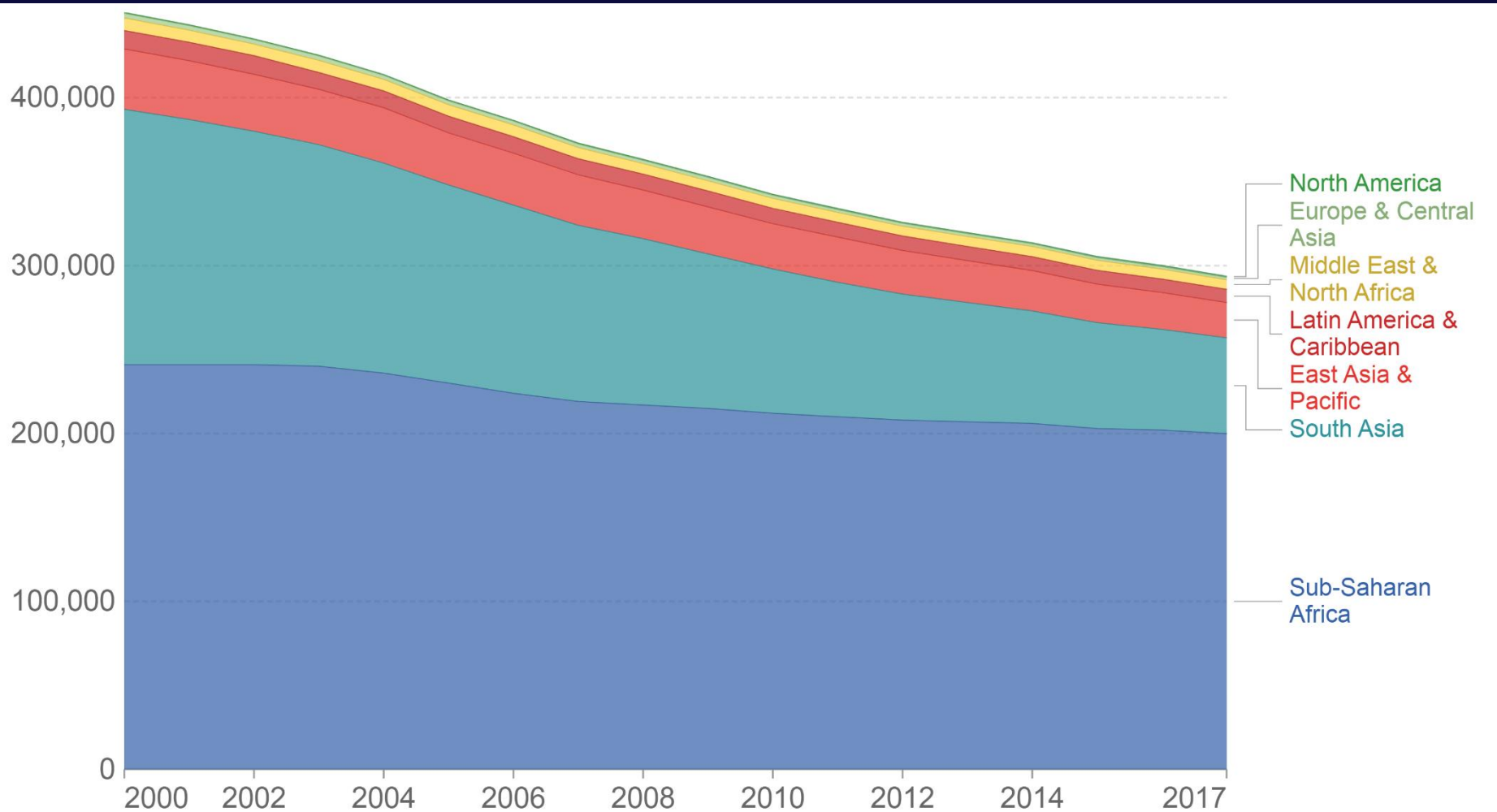
Global Major Causes of Maternal Mortality



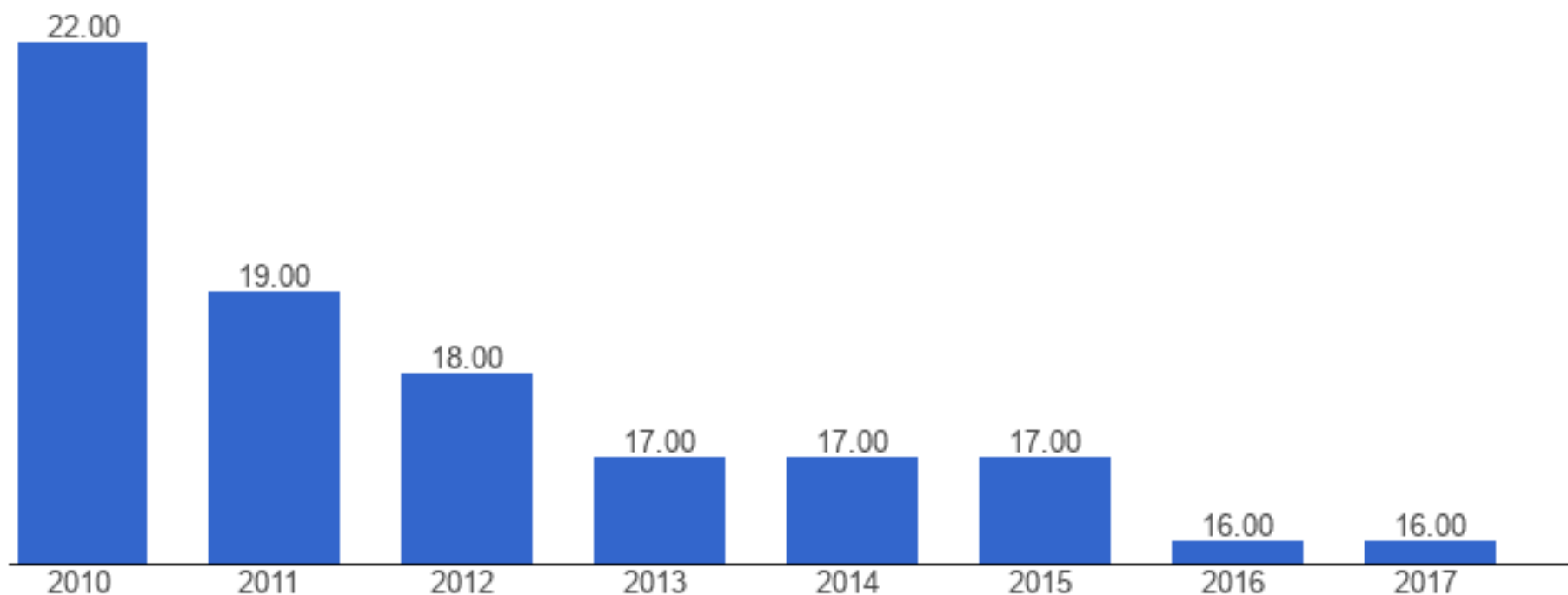
Aetiologies of Maternal Mortality

- Pregnancies with abortive outcome 3%
- Hypertensive disorders in pregnancy, 12%
- Obstetric haemorrhage 22%
- Pregnancy-related infection 7%
- Other obstetric complication 15%
- Unanticipated complications of management 2%
- Non-obstetric complications 24%
- Unknown/undetermined 9%
- Coincidental causes 2%
- Suicide 5%
- COVID-19 ?

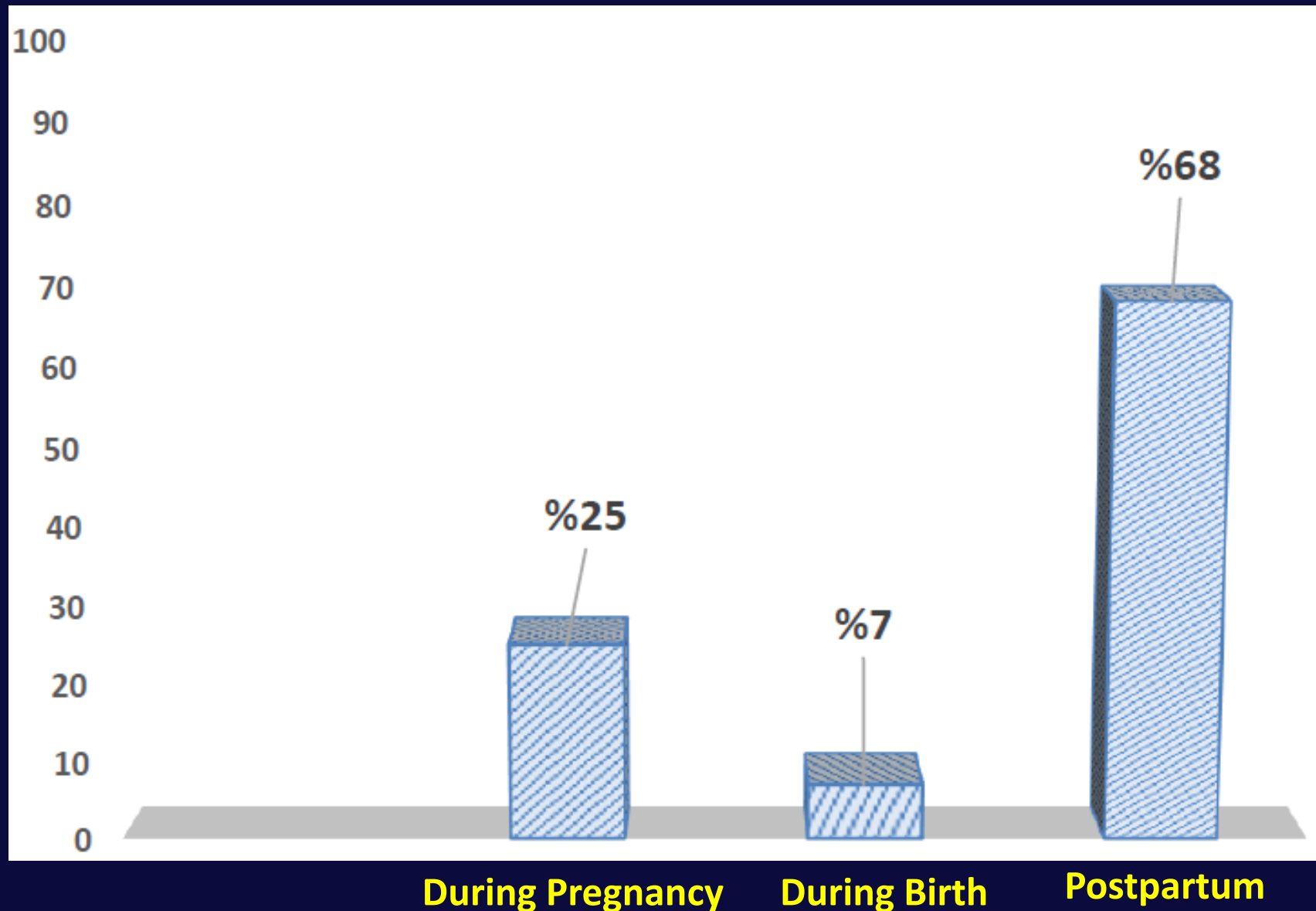
Current Trends in Global Maternal Morality



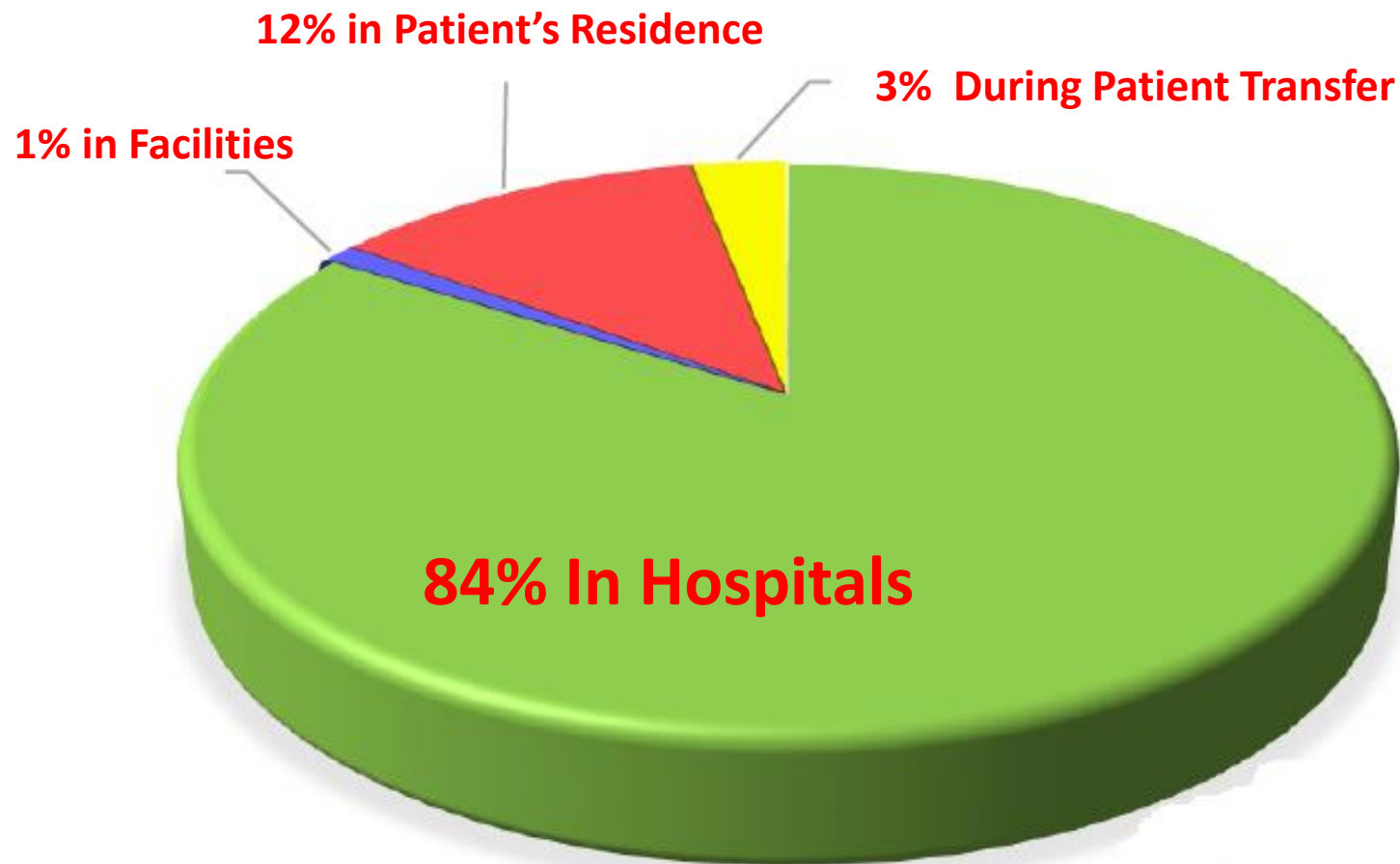
Current Maternal Mortality Statistics in Iran



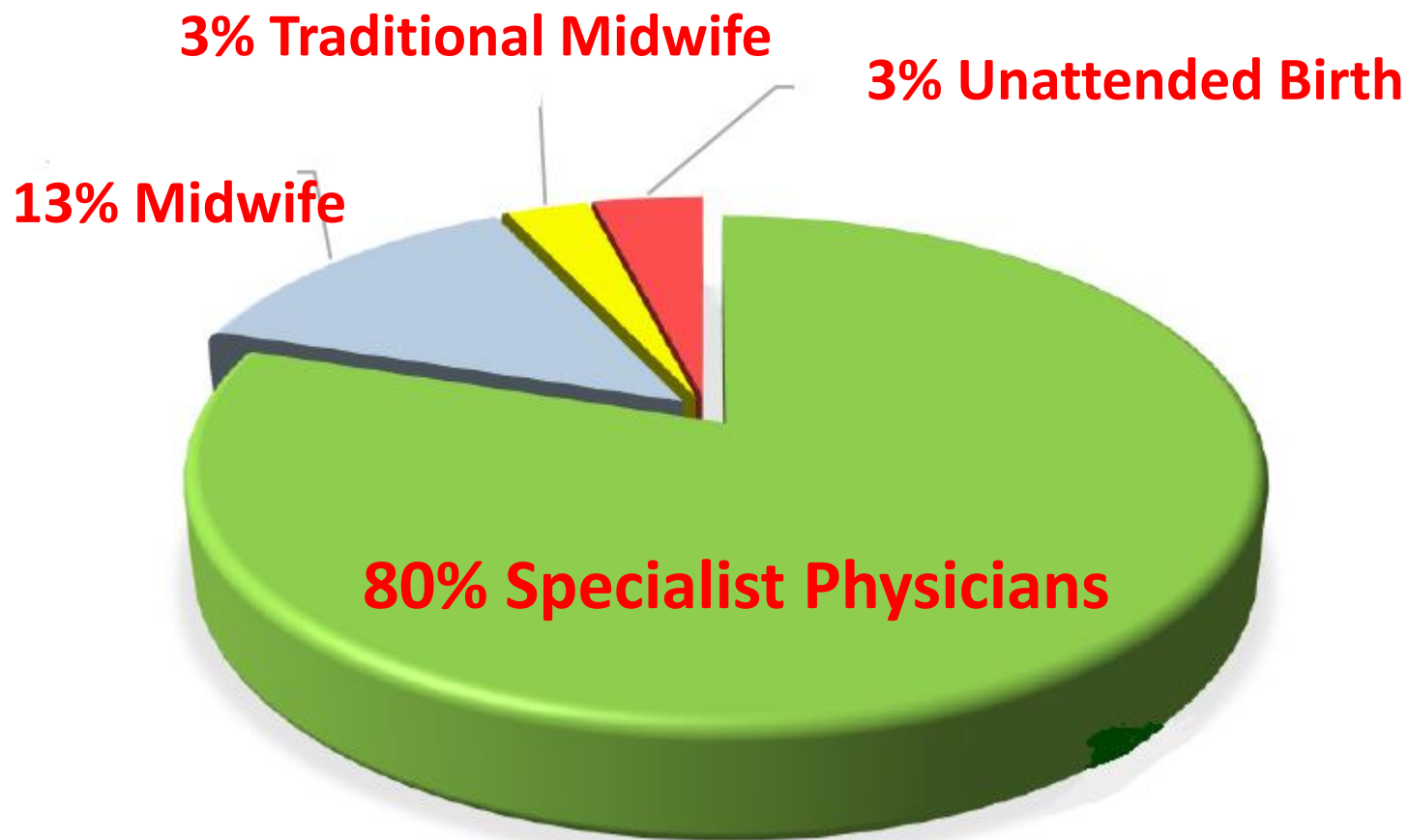
Timing of Maternal Mortality in Relation to Pregnancy



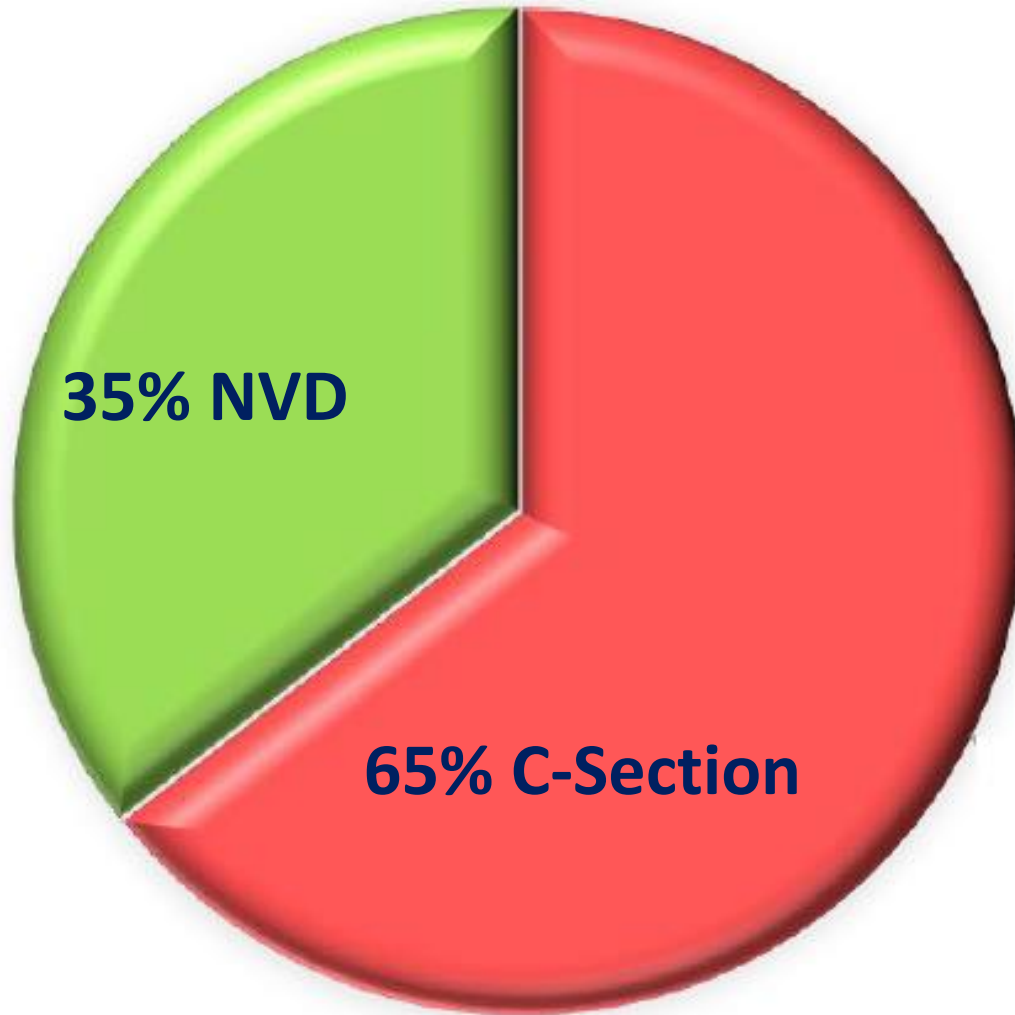
Location of Maternal Mortality



Birth Attendance for Maternal Mortality



Maternal Mortality According to Mode of Delivery



The Central Questions are;

- Often unstable maternal patients are recognised too late and by the time they are recognised and cared for, a golden opportunity for their effective management is lost...
- Are there ways to recognize the unstable maternal patients in a timely manner and make their management more effective in line with evidence based medicine and internationally recognised critical care guidelines?

Safety Net Concept in Maternal Mortality, (Pathway of MM)

1-Pregnancy Pool



2-Pool of patients at risk of pregnancy complication (~10%)



3-Complicated Pregnancy Pool



4-Unstable Maternal Patient

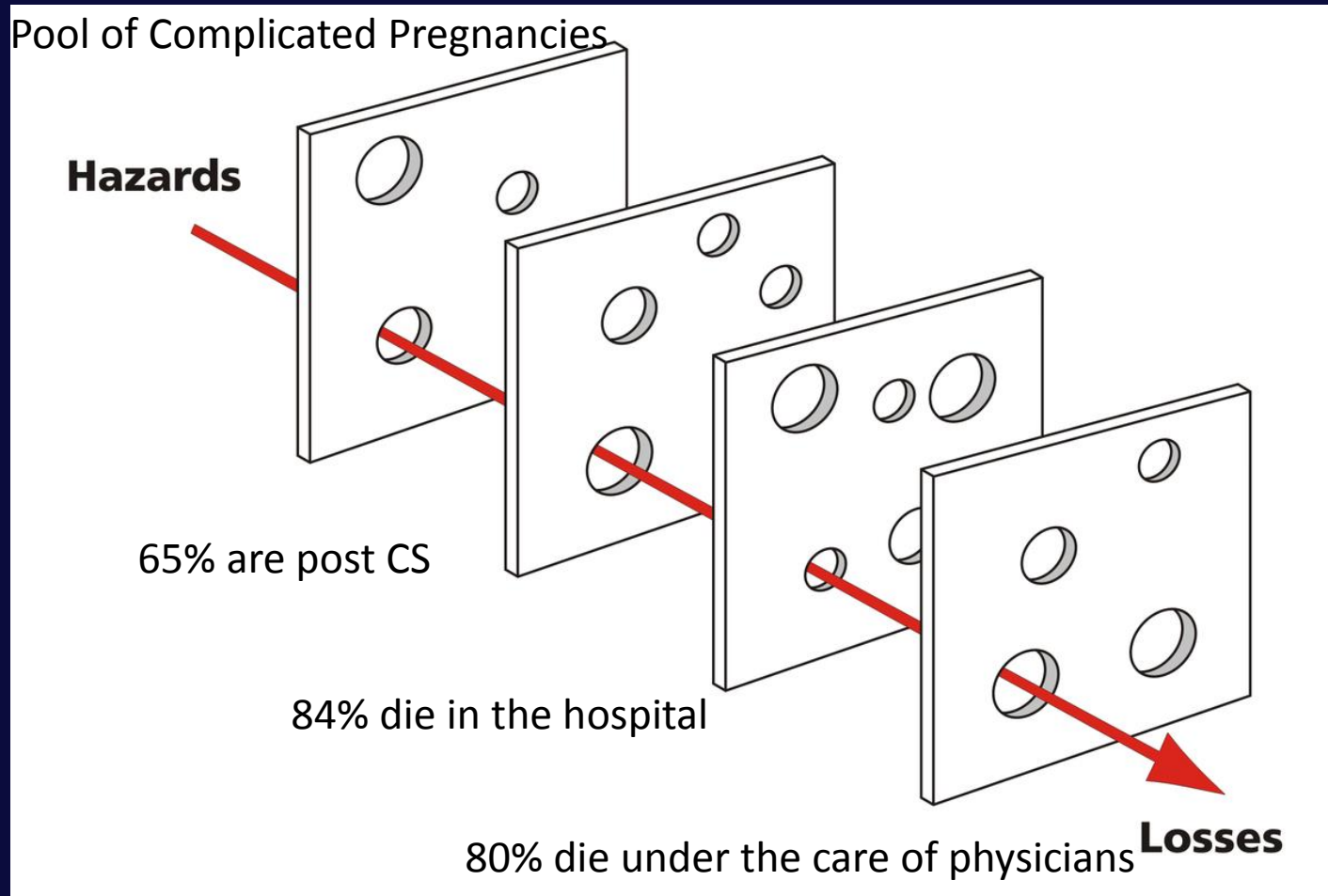


5-Coding Maternal Patient



6-Maternal Mortality

The Swiss Cheese Model of Maternal Mortality



The Highways of Death and the Sideways of Death



Respiratory Gate

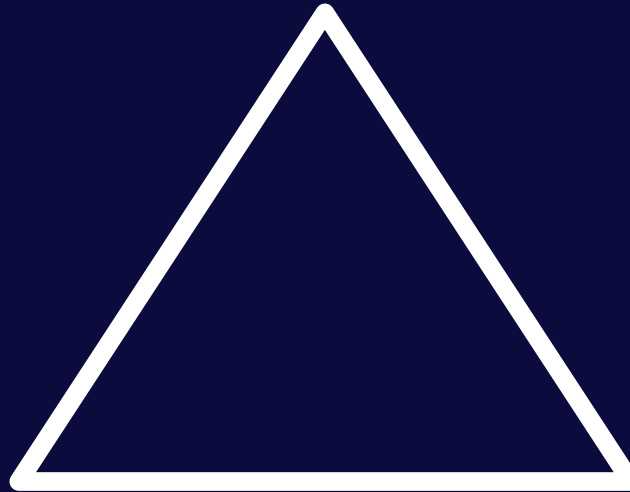
Death

CV Gate

CNS Gate

The Triangle of Maternal Mortality Highways

CNS death;
-For most intracranial bleed and raised ICP
-Brain Stem Injury
-CNS infection
Decreased LOC



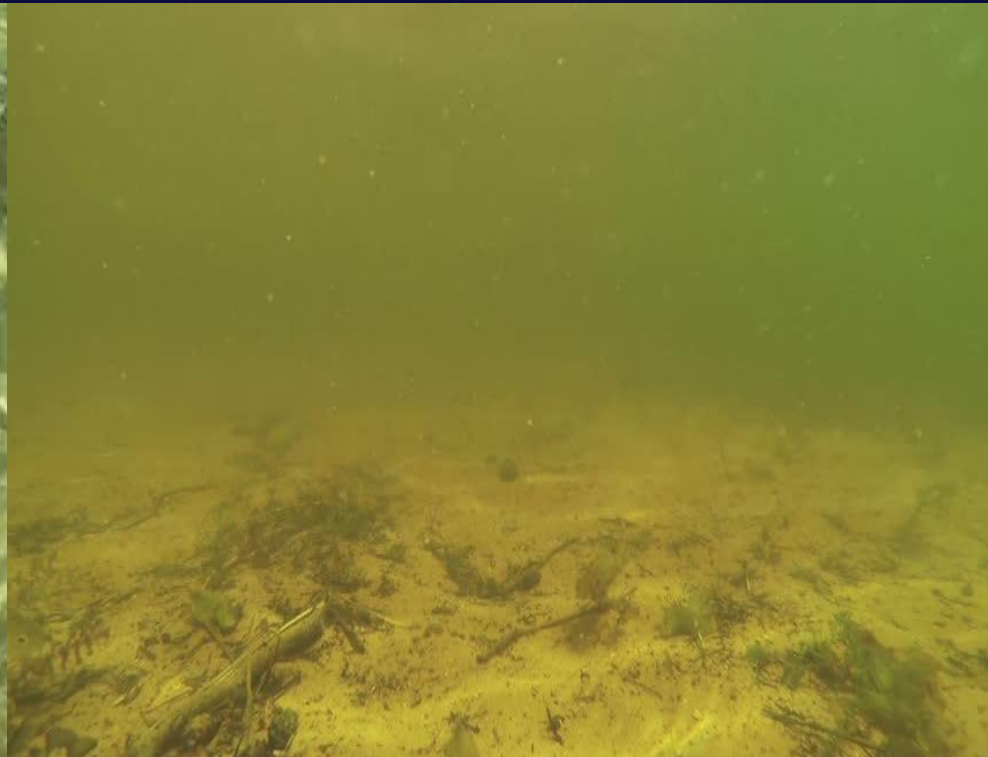
Cardiovascular death;
-For most haemorrhagic shock
-Distributive Shock (Septic Shock)
-Cardiac Dysrhythmias
-Heart Failure
-Pericardial Tamponed

Respiratory death;
-Pulmonary Infection including COVID-19
-Pulmonary Embolism
-Pulmonary Edema secondary to preeclampsia

Your first clinical impression has to be your best effort



A- Unstable maternal patient, when first arrives in a health-care facility



B-Unstable maternal patient, three days and four consultations and 5 lab work, later

Management of a critically ill obstetric patient

- Is very challenging due to the unique maternal physiological adaptations and care of another indwelling life.
- Although obstetric patients are young and healthy, maternal mortality for those admitted to an intensive care unit (ICU) ranges 5–20% in developed nations and 15–30% in developing nations.
- There is a paucity of well-defined guidelines for the management of critically ill obstetric patients.
- Maternal well-being is the priority when faced with the clinical situation of a deteriorating parturient.

Physiological Changes in Pregnancy

Complicating Management of Critical Illness

Organ system	Physiological changes in pregnancy influencing sepsis diagnosis and management	Implication in pregnancy
CVS	The uteroplacental circulation does not have auto regulation	Maternal infection can easily affect the fetus
Respiratory system	Increased minute ventilation results in decreased PaCO ₂ (compensated respiratory alkalosis)	Decreased ability to compensate a metabolic acidosis
Hematological	Leukocyte count increase and platelet count decrease Increased clotting factors and decreased fibrinolysis Hypoproteinemia results in decreased colloid osmotic pressure	Interpretation of trends in infection is complicated Favor the formation of intravascular fibrin during advanced sepsis Increased free fraction of albumin bound drugs Increased susceptibility to pulmonary edema
Renal	Increased renal blood flow, glomerular filtration and lower creatinine levels	Even normal levels can signify mild renal compromise
Gastrointestinal system	Reduced tone of lower esophageal sphincter Hypo perfusion of gastric mucosa leading to mucosal atrophy	Increased risk of aspiration Risk of trans mucosal bacterial translocation and exacerbation of the disease.
Immunological	Pregnancy is an immunomodulatory state characterized by first strong inflammatory response, second anti-inflammatory response and the last renewed inflammation phase High TLC A shift from Th1 to Th2 immunity during pregnancy	Competent immune response is crucial to protect the mother and the fetus High white blood cell count is not a reliable indicator of sepsis Viral infections are much more severe

Indications for admission of critically ill obstetric patients

- There are no commonly agreed upon criteria that determine admission of an obstetric patient to an ICU. It seems mostly based on local criteria and local practices.
- Majority are admitted to the ICU with: hemorrhage, sepsis, hypertensive disorders of pregnancy, amniotic fluid embolism, complex cardiac diseases, acute fatty liver, aspiration syndromes, infections...
- In general, if two organ systems are failing with a need for mechanical ventilation, obstetric patients should be admitted to ICU, I have personally always used the physical exam and patient's vitals to make this decision.

In Summary

- Unstable patients are admitted to ICU
- How do you make a decision if the patient is unstable: Vitals, Vitals, Vitals...

Key Point

- Vital Signs are Under-Utilized in Practice of Medicine in IRI,

- Patient is unstable and is admitted to ICU.
- What would you do next?

Case # 1

- 37 years old female, one day post C-Section, found unconscious on bathroom floor, in a pool of blood...
- You are the physician on call what would you do?
- Being an outstanding physician you ask for the patient's vitals,
- BP: 82/48, HR: 140, RR: ?, O2 Sat: 86%, T: 35, GCS: 3
-

Stepwise Approach

- A- Keep the airways patent, Head Tilt, Chin Lift, Jaw Thrust,
- B- Breathing, is patient breathing? If not start resuscitation....
- C- Does patient has a pulse? If not start CPR...IV access (Central Line...)
- D- Disability, Neurological status....

What are the relevant questions in this Case?

- What is an acceptable definition for PPH?
- How relevant PPH is to maternal mortality in the world and IRI?
- What are the risk factors for PPH?
- What are the potential etiologies of PPH?
- What are the complications of PPH?
- Are there globally recognized guidelines for the management of PPH?

What are the relevant questions in this Case?

- 1-How can I find out how severe is the PPH?
- 2- What are the ratio of PRBC, FFP, Plt for transfusion?
- 3- Is there any role for fibrinogen concentrates, tranexamic acid, Activated factor VII?
- 4- What if I have to ventilate the patient?
- 5- What complications should I expect?
- 6-What about sedation and nutrition of Patient in ICU?

The definition of PPH

- PPH is defined as blood loss of more than 500 mL following vaginal delivery or more than 1000 mL following cesarean delivery. A loss of these amounts within 24 hours of delivery is termed early or primary PPH, whereas such losses are termed late or secondary PPH if they occur 24 hours after delivery.
- Estimates of blood loss at delivery are subjective and generally inaccurate. Studies have suggested that caregivers consistently underestimate actual blood loss. Another proposal suggests using a 10% fall in hematocrit value to define PPH, but this change is dependent on the timing of the test and the amount of fluid resuscitation given.

The definition of PPH

- Another consideration is the differing capacities of individual patients to cope with blood loss. A healthy woman has a 30-50% increase in blood volume in a normal singleton pregnancy and is much more tolerant of blood loss than a woman who has preexisting anemia, an underlying cardiac condition, or a volume-contracted condition secondary to dehydration or preeclampsia. For these reasons, various authors have suggested that PPH should be diagnosed with any amount of blood loss that threatens the hemodynamic stability of the woman.

The definition of PPH

- The diagnosis of PPH is usually reserved for pregnancies that have progressed beyond 20 weeks' gestation. Deliveries at less than 20 weeks' gestational age are spontaneous abortions. Bleeding related to spontaneous abortion may have etiologies and management in common with those for PPH.

Hemorrhage

- Obstetric hemorrhage is responsible for more than 30% of all maternal deaths in low income countries and over 10% of maternal deaths in high-income countries.
- Can be due to uterine atony, placentation abnormalities, uterine rupture, surgical and genital tract trauma
- The use of uterotonic agents, uterine massage, controlled cord traction, intrauterine balloon tamponade, uterine artery embolization or ligation and even hysterectomy may not be always effective.

Etiology of PPH

- PPH has many potential causes, but the most common, by a wide margin, is uterine atony, ie, failure of the uterus to contract and retract following delivery of the baby.
- PPH in a previous pregnancy is a major risk factor and every effort should be made to determine its severity and cause.
- In a recent randomized trial in the United States, birthweight, labor induction and augmentation, chorioamnionitis, magnesium sulfate use, and previous PPH were all positively associated with increased risk of PPH.

PPH Risk Factors

In a large, population-based study, significant risk factors, identified using multivariable analysis, were as follows:

- Retained placenta
- Failure to progress during the second stage of labor
- Placenta accreta
- Lacerations
- Instrumental delivery
- Large-for-gestational-age (LGA) newborn
- Hypertensive disorders
- Induction of labor
- Augmentation of labor with oxytocin

Obesity and PPH

- PPH is also associated with obesity. In a study by Blomberg, the risk of atonic uterine hemorrhage rapidly increased with increasing BMI; in women with a BMI over 40, the risk was 5.2% with normal delivery and 13.6% with instrumental delivery.
- A study by Hanley et al reported that serotonin-norepinephrine reuptake inhibitor exposure in late pregnancy was associated with a 1.6- to 1.9-fold increased risk of postpartum hemorrhage

- As a way of remembering the causes of PPH, several sources have suggested using the “4 *T*’s” as a mnemonic: tone, tissue, trauma, and thrombosis.

Prevention of PPH

- High-quality evidence suggests that active management of the third stage of labor reduces the incidence and severity of PPH.
- Active management is the combination of:
 - (1) uterotonic administration (preferably oxytocin) immediately upon delivery of the baby,
 - (2) early cord clamping and cutting, and
 - (3) gentle cord traction with uterine counter-traction when the uterus is well contracted (ie, Brandt-Andrews maneuver).

Prevention of PPH

- The value of active management in the prevention of PPH cannot be overstated. The use of active versus expectant management in the third stage was the subject of 5 randomized controlled trials (RCTs) and a Cochrane meta-analysis. These trials included more than 6000 women.

Active Vs. Passive Management of Third Stage of Labor

- The available evidence show a conclusive benefit for active management,
- An approximate 60% reduction in the, occurrence of PPH greater than or equal to 500 mL and 1000 mL, hemoglobin concentration of less than 9 g/dL at 24-48 hours after delivery, and the need for blood transfusion.
- An 80% reduction in the need for therapeutic uterotonic agents was noted.
- The results indicate that for every 12 patients receiving active rather than physiological management, one PPH would be prevented.
- For every 67 patients so treated, one patient would avoid transfusion with blood products.

Presentation of PPH

- The usual presentation of PPH is one of heavy vaginal bleeding that can quickly lead to signs and symptoms of hypovolemic shock.
- This rapid blood loss reflects the combination of high uterine blood flow and the most common cause of PPH, ie, uterine atony.
- Blood loss is usually visible at the introitus, and this is especially true if the placenta has delivered.
- If the placenta remains in situ, then a significant amount of blood can be retained in the uterus behind a partially separated placenta, the membranes, or both.

Clinical Findings in Obstetric Hemorrhage

Blood Volume Loss	Blood Pressure (systolic)	Symptoms and Signs	Degree of Shock
500-1000 mL (10-15%)	Normal	Palpitations, tachycardia, dizziness	Compensated
1000-1500 mL (15-25%)	Slight fall (80-100 mm Hg)	Weakness, tachycardia, sweating	Mild
1500-2000 mL (25-35%)	Moderate fall (70-80 mm Hg)	Restlessness, pallor, oliguria	Moderate
2000-3000 mL (35-50%)	Marked fall (50-70 mm Hg)	Collapse, air hunger, anuria	Severe

Common and Important Pitfalls in PPH

- Caregivers consistently underestimate visible blood loss by as much as 50%.
- The volume of any clotted blood represents half of the blood volume required to form the clots.
- Most women giving birth are healthy and compensate for blood loss very well.
- This, combined with the fact that the most common birthing position is some variant of semirecumbent with the legs elevated, means that symptoms of hypovolemia may not develop until a large volume of blood has been lost

Severity of Hemorrhage, Shock index (SI)

- The ratio of pulse to systolic blood pressure (using vital sign alert device) for detection of hypovolemic shock secondary to obstetric hemorrhage.
- Normal SI (no Shock) ranges 0.7–0.9 for obstetric populations. A value of 0.9 is considered to be the threshold for referral to a tertiary hospital, 1.4 as the threshold for intensive care treatment, and 1.7 as an indicator of high risk for adverse events.
- While, SI is beginning to be used, it needs stronger clinical evidence to be incorporated into the routine practice.

Severity of Hemorrhage, Fibrinogen Level

- Low fibrinogen level is an important predictor for the progression from moderate to severe PPH. A plasma level below 2 g/L in the early phase of PPH is associated with increased risk of severe hemorrhage. For each 1 g/L decrease in fibrinogen, there was a 2.6 fold increased odds of severe PPH.
- Viscoelastic Hemostatic Assays in PPH, Provide a real-time assessment of coagulation and fibrinolysis, especially when standard laboratory tests are too slow to be of clinical utility.
- If low level of fibrinogen is detected by thromboelastography, administering fibrinogen concentrates is an important early intervention.

Management of PPH

Transfusion, and Ratio of RBC, FFP and Platelet

- There is no ideal ratio of red blood cells (RBC): fresh frozen plasma (FFP) or platelets for the management of PPH,
- Yet, a commonly followed massive transfusion protocol is (RBC: FFP: platelets as 4:4:1 ratio) and (1:1:1 ratio).
- Compared to formula-driven strategies (such as fixed ratios of blood products), the use of goal-directed resuscitation using point-of-care coagulation tests has been proposed but is still being investigated.
- Always monitor patient for complications of massive transfusion...

Some of the complications of massive transfusion include

- Volume Overload,
- Hypothermia
- Dilutional Coagulopathy
- Hypocalcaemia, Hypomagnesaemia, Citrate Toxicity
- Metabolic Acidosis
- Hyperkalemia and Hypokalemia
- Immune Hemolysis,
- Air Embolism,
- TRALI

Decreasing the blood loss, early use of fibrinogen concentrates, tranexamic acid, Activated factor VII

- Fibrinogen concentrates, and Tranexamic Acid seem to have a promising role in managing PPH, however, evidence supporting it remains limited.
- Activated factor VII has gained popularity but is not US-FDA approved and is very expensive. It has been used for treating life-threatening PPH and to prevent hysterectomy, but its routine use needs stronger evidence and cost effectiveness.

Ventilator strategies for managing critically ill Obstetric Patient

Positive pressure ventilation;

- Non-invasive (NIPPV), (C-PAP, BIPAP or Optiflow) , this mode of ventilation is most suitable for individuals with normal mental status, good respiratory drive, stable hemodynamics and with no excessive pulmonary secretions
- Invasive positive pressure ventilation, (IPPV) using an endotracheal tube (ETT), A low threshold for ETT intubation should be kept in view of high aspiration risk. Airway edema in obstetric patients warrants the use of smaller size ETT which otherwise increases airway resistance and interferes with successful weaning during prolonged ventilation.
- Standard protocols for initiation of ventilation in peripartum patients should be in accordance to the physiological changes in the respiratory parameters during pregnancy.

Other Ventilator strategies in Obstetric Patient

- When conventional methods of ventilation fail, newer modes such as Airway Pressure Release Ventilation (APRV) and high-frequency oscillatory ventilation (HFOV) have been tried and may be useful particularly in patients with severe acute respiratory failure (SARF) and acute respiratory distress syndrome (ARDS)
- Although pregnancy has been considered to be a contraindication to prone position ventilation, there are individual reports of its successful use in parturient.
- Subject to availability Extracorporeal membrane oxygenation (ECMO) can be used early in acute respiratory failure with refractory hypoxemia. Major disadvantage of ECMO is that it exposes the indwelling fetus to extracorporeal circulation and systemic heparinization.

Sedation in Obstetric Patients

- Use of sedatives and analgesics is necessary in obstetric patients who are mechanically ventilated.
- Most of the commonly used ICU sedatives cross the placenta to varying degrees based upon the agent, its dose, duration of use and any organ dysfunction altering its metabolism and excretion. Risks and benefits must be assessed prior to their use.

Sedation in Obstetric Patients

Drugs (Category)	Adverse Effects	Potential Use	Safety
Opioids (e.g., fentanyl [C], morphine [C], and hydromorphone [C])	Respiratory depression Withdrawal syndrome in the newborn		Pediatrician made aware of maternal opioids
Benzodiazepines (e.g., midazolam [D] and lorazepam [D])	Respiratory depression, floppy infant syndrome, withdrawal syndrome in the newborn	Treatment of eclamptic seizures	Considered contraindicated during pregnancy
Propofol (B)	Hypotension	Higher doses (2.8 mg/kg) can result in low Apgar scores, muscle hypotony, and depressed neuromuscular activity.	Safe based on animal studies
Dexmedetomidine (C)	Increased myometrial contraction in rats (13)		Limited data regarding its safety

Nutrition in Critically ill Obstetric Patient

- The basal caloric requirement of a critically ill patient is 25 kcal/day/kg (ideal body weight) that turns out to be 2200 to 2800 kcal/day for an average sized female.
- As for the fetus nutritional requirements, the first trimester usually does not warrant need of extra calories, yet, a parturient needs additional 350 kcal/day and 450 kcal/day in the second and third trimesters, respectively.
- The protein requirement is twice that of a non-obstetric patient, and this supplementation must be carried on till lactation.
- Generally, carbohydrates meet up to 70%, and fats meet 30% of caloric requirements. Proteins in diet compensate for negative nitrogen balance and are not included in the caloric needs.

Nutrition in Critically ill Obstetric Patient

- The nutritional goals must be adjusted according to the associated illness.
- Each degree rise in temperature increases the caloric needs by 10% and sepsis steps up the need by 20%.
- Inotropes use may increase caloric requirement by 2.5 times.
- Neither weight gain nor albumin values have a role in the nutritional assessment in parturient because of the effects of pregnancy.
- Indicators such as pre-albumin and serum transferrin are used for assessing the response to nutritional support.
- An optimal nutritional solution needs to have additional amounts of zinc, folate and vitamin B12 in the first trimester.

Case # 2,

- 43 Y/O female 34 weeks pregnant, presenting to ED with altered level of consciousness...
- You check patient's vitals and here is what you find;
- BP: 84/68, HR: 134, RR:26, O2Sat:78%, T:39.2 *C, GCS: E3V4M6,

What Would You Do?

- Airway
- Breathing
- Circulation
- Disability
- Exposure

Sepsis

- Maternal sepsis causes 15% of maternal deaths worldwide.
- Diagnosing sepsis during pregnancy is a challenge because of complex pathophysiological changes that can mask its initial presentation,
- Clinical features of sepsis in a pregnant patient might be absent or be even less distinctive
- There is a lack of valid sepsis guidelines for peripartum patients.
- Genital and urinary tract infections are the most important causes of sepsis in developed nations whereas HIV, malaria and community-acquired pneumonia are important causes in developing countries

Septic Shock

- Septic shock are most commonly seen in patients with pyelonephritis, chorioamnionitis and endometritis.
- Hepatitis E virus, herpes simplex virus and malaria are much more severe in pregnancy.
- In addition to the systemic symptoms, a few local symptoms such as lower abdominal pain or perineal pain, frequent micturition and diarrhea can also be present.
- The outcome of septic shock in a peripartum patient is better than non-pregnant critically sick patients due to younger age, fewer comorbidities and usually localized source of infection.

Why is maternal sepsis on the rise?

- More women over 40 becoming pregnant
- Availability of “assisted reproductive technologies” results in more invasive interventions due to incidence of multifetal gestation
- Disorders of pregnancy such as preeclampsia, placental abruption, amniotic fluid embolism, and PPH can contribute to the rise in maternal sepsis.
- Increasing rates of Obesity, diabetes, and C/S delivery seems to contribute to the rise in maternal sepsis.
- C/S delivery: 3 times more likely to develop sepsis

Risk Factors for Sepsis

- C/S delivery
- Emergency C/S
- Prolonged Rupture of the Membranes
- Retained products of Conception
- Preterm Labor
- Multiple Vaginal Exams
- Obesity
- Diabetes
- Anemia
- Low socioeconomic status
- Winter months
- Failure to recognize severity of infection

CAUSES OF SEPSIS & SEPTIC SHOCK IN PREGNANCY & PUERPERIUM

- ✓ Pyelonephritis
- ✓ Retained Products of Conception
- ✓ Neglected Chorioamnionitis or endometritis
- ✓ Pneumonia (1. Bacterial 2. Viral (H1N1, COVID-19))
- ✓ Unrecognized or inadequately treated necrotizing fasciitis (1. Abdominal incision 2. Episiotomy/Perineal Laceration)
- ✓ Intrapерitoneal Etiology (1. Ruptured Appy 2. Acute Cholecystitis 3. Bowel Infarction)
- ✓ Urinary Tract Infections
- ✓ Mastitis

Maternal Sepsis Mortality and Morbidity During Hospitalization for Delivery

- Bauer et al Anesth Analg 2013
 - 1680 Women with severe sepsis had a known organism
- | | |
|-----------------------------|------|
| • E. coli septicemia | 27% |
| • Staphylococcal septicemia | 22% |
| • Streptococcal septicemia | 20% |
| • Gram negative septicemia | 19% |
| • Pneumococcal septicemia | 4% |
| • Pseudomonal septicemia | 2.4% |
| • Anaerobic septicemia | 2% |

Sepsis Screening Criteria for Non-OB adults vs. OB

Screening Tool - adjusted for the physiological effects of pregnancy

➤ Adult Screening Criteria

- Temp $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
- *HR > 90*
- *Resp Rate > 20*
- WBC $> 12,000$, $< 4,000$ or $> 10\%$ *immature neutrophils*
- *New mental status change*
- Blood glucose > 140 mg/dl in the absence of diabetes

➤ Perinatal Screening Criteria Adjustments

- Temp $> 38^{\circ}\text{C}$, or $< 36^{\circ}\text{C}$
- *HR > 110*
- *Resp Rate > 24*
- *WBC $> 15,000$ or $< 4,000$ or $> 10\%$ immature neutrophils*
- *Altered Mental Status present*
- Blood glucose > 140 mg/dl in the absence of diabetes

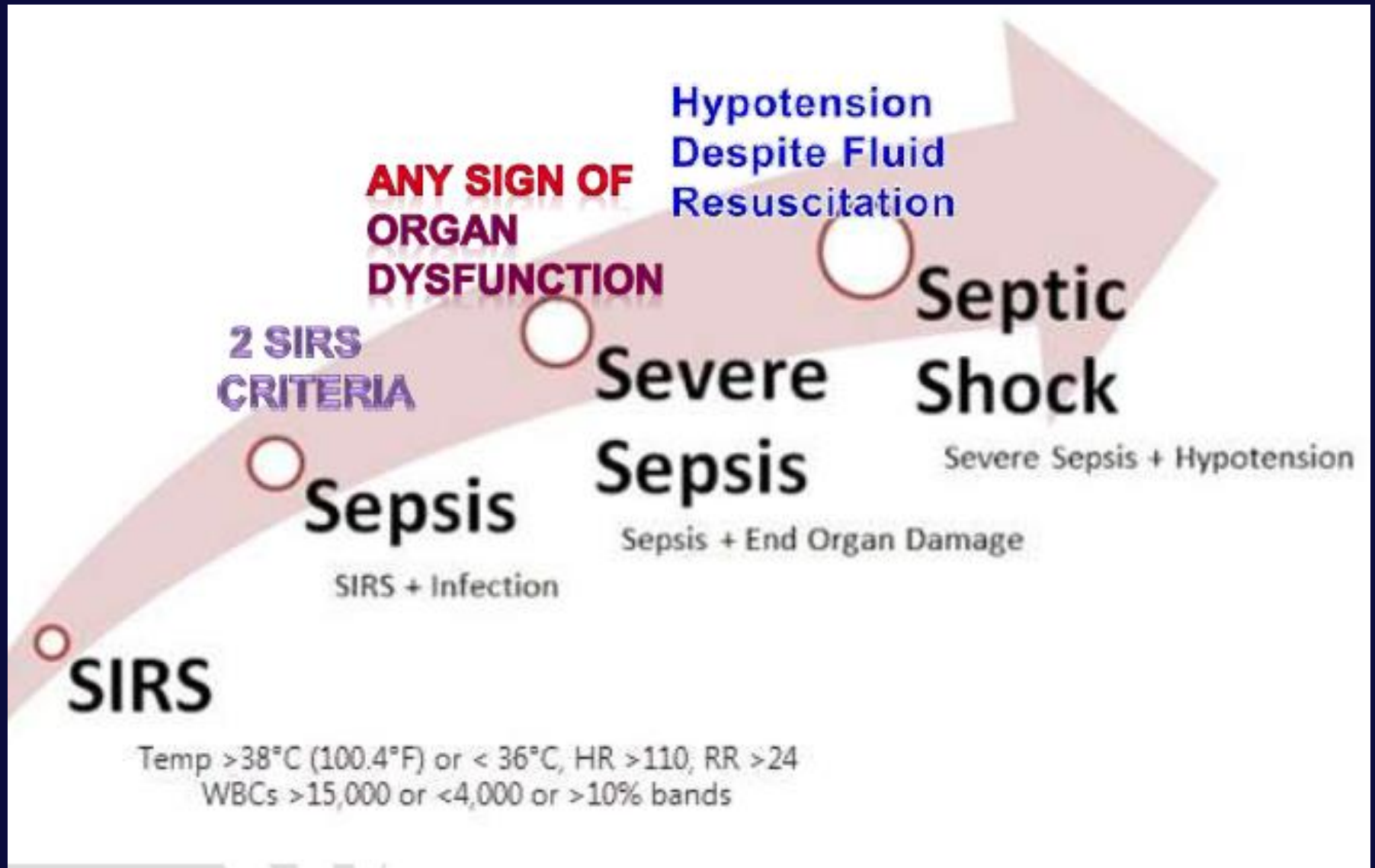
Perinatal Parameters and Sepsis

- ✓ Because of the physiology of pregnancy and labor, we adjusted the screening criteria for Perinatal patients
- ✓ Increase in blood volume increases maternal heart rate by 10-20 bpm
- ✓ Minute volume ($RR \times \text{Tidal Volume}$) increases 50% due to an increase in Tidal Volume
- ✓ Due to diaphragm position, lung volumes change causing increased respiratory rate
- ✓ Increase in WBC in labor and immediate postpartum
- ✓ Increase in blood flow to the kidneys causes a decrease in the creatinine level

When should the sepsis screening be performed?

- Upon arrival to the unit (triage or direct admit)
- EVERY SHIFT and/or assuming care of patient
- PRN for suspicion/indication of new infection
- Perhaps the “Sepsis Screening Form” can be incorporated into admission files.

Sepsis Syndrome



Definition of SIRS

- Systemic Inflammatory Response Syndrome (SIRS)
 - Inflammatory process that can be generated by infection or by non-infectious causes (burns, trauma)
 - **Non-pregnant:** 2 or more of the following
 - Temperature $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$
 - HR > 90 beats/min
 - RR >20 breaths/min or $\text{PaCO}_2 <32$ mmHg
 - WBC $> 12,000/\text{mm}^3$, $< 4,000/\text{mm}^3$ or $>10\%$ bands

**It is likely that if you climb 3-4 floors of stairs
you may be SIRS positive**

Sequence of Events in Maternal Patient with Sepsis

- ✓ An Insult leading to activation of Inflammatory system and release of pro-inflammatory mediators, (histamines, serotonin, cytokines...).
- ✓ Pro-inflammatory mediators cause increase vascular permeability and vasodilation
- ✓ Increased Vascular Permeability leads to Migration of leukocytes to site of injury
- ✓ Pro-inflammatory mediators also lead to decreased vascular tone combined with vascular permeability, results in pooling of blood, causing a relative decrease in intravascular volume; plasma & molecules leak into extravascular space
- ✓ Small molecules such as Na, H₂O and larger molecules such as ALBUMIN will escape through leaky vessels (loss of osmotic pressure)
- ✓ The overall Loss of fluid from intravascular space leads to intravascular volume depletion (hypotension and tachycardia...poor end organ perfusion...)

These Cell and Molecular Events Results in Cardinal Sepsis Symptoms

- Hypotension
- Tachycardia
- Decreased Oxygen to the Organs
- Organ Dysfunction

Consequences of Accumulation of Extravascular Fluid

- Peripheral Edema
- Pulmonary Edema
- Renal Impairment
- Liver Impairment

Consequences of Poor Oxygen Delivery in Septic Maternal Patient

- Both Pregnancy and Sepsis lead to Increase Tissue Oxygen Demand
- Given that in Septic Maternal Patient the Oxygen Demand of the Tissues is Not Met by Oxygen Delivery, Anaerobic Respiration Occurs Leading to the Production of Lactic Acid (serum lactate) and Metabolic Acidosis,
- Metabolic Acidosis is Known to Increased Respiratory Rate and Cause Cardiac depression (Hypotension) and Confusion (Decreased Level of Conciseness)

Disseminated Intravascular Clotting

Sepsis
causes
widespread
clotting

This causes
consumption of
platelets, clotting
factors and
fibrinogen,

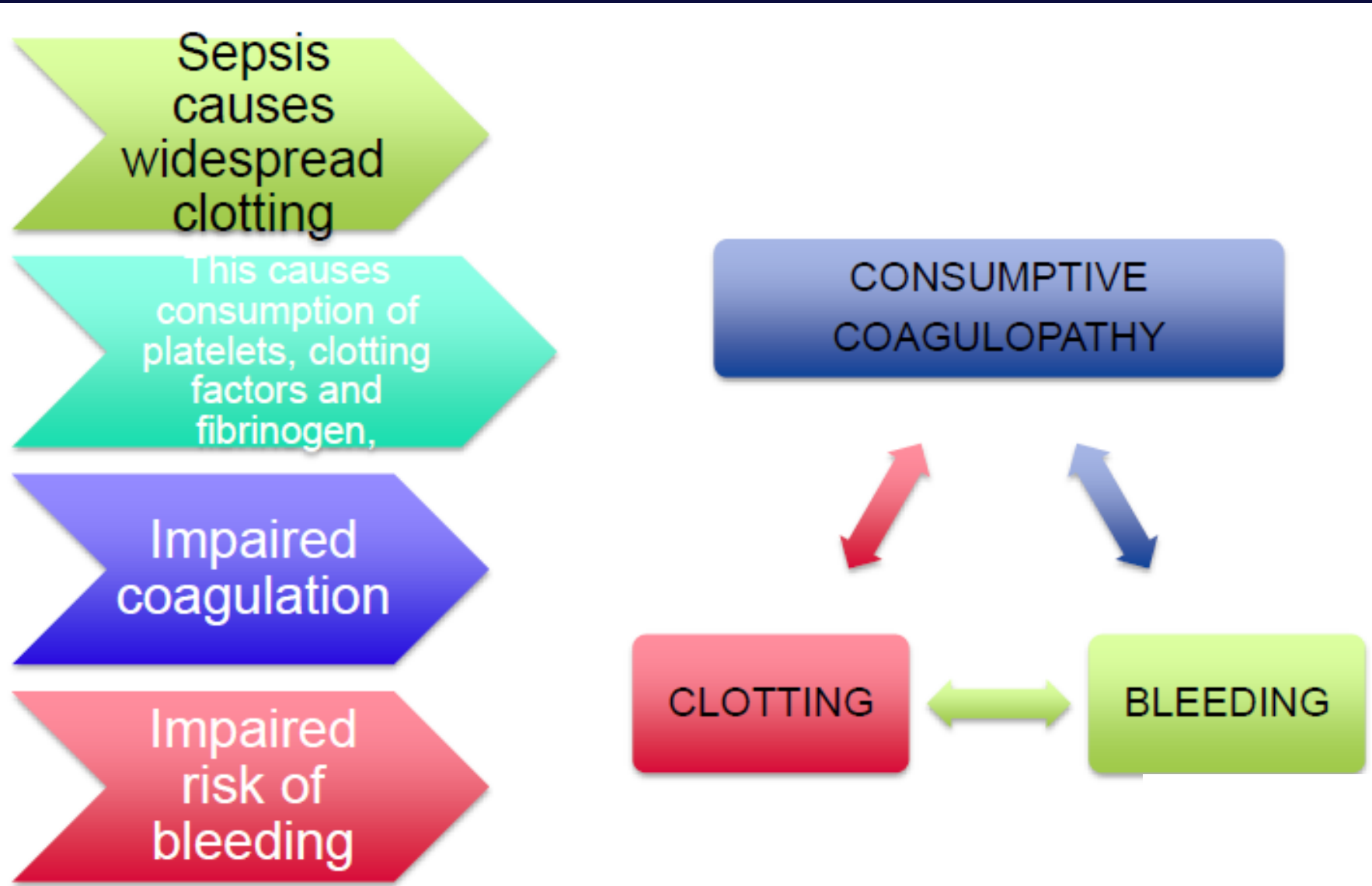
Impaired
coagulation

Impaired
risk of
bleeding

CONSUMPTIVE
COAGULOPATHY

CLOTTING

BLEEDING



Clinical Manifestations of Sepsis

- Early stages
 - RECOGNITION KEY TO SUCCESSFUL TREATMENT
 - Shaking chills, fever (most common in pregnancy), tachycardia, flushing
 - Warm extremities, nausea, vomiting, diarrhea
 - Subtle changes in mental status
 - May be difficult to diagnose early in pregnant women, particularly in labor

Clinical Manifestations of Sepsis

- Laboratory findings
 - mild leukopenia or leukocytosis, hyperglycemia
 - early DIC : thrombocytopenia, decreased fibrinogen, increased PTT and PT
 - transient respiratory alkalosis with increasing metabolic acidosis
 - Increased serum lactate
 - Low arterial pH
 - Increased base deficit

Clinical Manifestations

- Later stages
 - Generalized vasoconstriction - cold extremities
 - oliguria, peripheral cyanosis
 - tachycardia, severe hypotension
 - Depressed cardiac output, low SVR
 - Laboratory findings
 - profound metabolic acidosis
 - electrolyte imbalance
 - generalized DIC
 - Multiple end-organ failure

Multiple Organ Effects with Sepsis and Shock

- CNS Effects : confusion, coma, somnolence, fever
- Cardiovascular: decreased CO, hypotension, myocardial depression, tachyarrhythmia
- Pulmonary: hypoxemia, diffuse infiltrates, pleural effusion,
- Renal: hypoperfusion, acute tubular necrosis, ARF
- Hematologic: thrombocytopenia, leukocytosis, leukopenia, consumptive coagulopathy

Why do Maternal Patients die from septic shock? Organs in question...

- Cardiac; myocardial depression, cardiac output usually maintained due to tachycardia and cardiac dilatation....
- Lungs; ARDS, death from hypoxemia or hypercarbia
- Renal failure : dialysis will prevent death
- Liver dysfunction : hepatic encephalopathy rare?

Laboratory Evaluation

- Complete blood count
 - differential and platelets
- Coagulation profile
 - PT, PTT, FDP, Fibrinogen
- Electrolytes, glucose
- Creatinine and blood urea nitrogen
- Urinalysis and culture
- Blood culture and gram stain
- Cultures of infected sites
- Chest X-ray
- CT, ultrasound, MRI to localize infectious etiology

Laboratory Evaluation

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Surviving Sepsis Campaign

- Bundless (Order Sets)

Elements when used together, improve outcomes more than when used separately!
(Evidence based)



Why Do We Need Bundles For Early Recognition?

- Delay in diagnosis and treatment of sepsis has been shown to increase mortality
- Pregnant patients look deceptively well before rapidly deteriorating
- Early recognition and treatment of maternal sepsis will improve survival, decrease length of stay, and length of stay in the ICU

Barton & Sibai, 2012

International Guidelines for Management of Severe Sepsis and Septic Shock,

- It involves taking care of the well being of both the mother and child
- It is done in accordance with the Surviving Sepsis (SS) Campaign guidelines
- Initial resuscitation: 'resuscitation bundle' goals within the first 6 h of diagnosis of patients with sepsis-induced tissue hypoperfusion (defined as hypotension persisting after initial fluid challenge or blood lactate concentration ≥ 4 mmol/L)

Severe Sepsis Bundle: TO BE COMPLETED WITHIN 3 HOURS

- Time zero = time of confirmed positive sepsis screen
- Measure lactate level
- Obtain blood cultures **prior** to administration of antibiotics
- Administer broad spectrum antibiotic(s)
- Administer 30 mL/Kg crystalloid for hypotension or lactate > 3.9 mmol/L

Implementation of Sepsis Bundle for Early Recognition

- Randomly assigned 263 patient who presented to ED with severe sepsis/septic shock
- Received either 6 hours of EGDT or conventional care before ICU
- Mortality was 30.5% in patients receiving EGDT
- Mortality was 46.5% in patients receiving conventional care

Early Goal Directed Therapy (first 6 h of resuscitation)

- (a) Central venous pressure 8–12 mmHg
- (b) Mean arterial pressure (MAP) \geq 65 mmHg
- (c) Urine output \geq 0.5 mL kg⁻¹ h
- (d) Central venous (superior vena cava) or mixed venous oxygen saturation 70 or 65 %,
- (e) Critically ill obstetric patients need judicious fluid administration due to vulnerability to fluid overloading. Fluid resuscitation should be crystalloid based,
- (f) If vasopressor is needed, norepinephrine is the first-choice vasopressor. If patient has low cardiac output in the presence of adequate volume resuscitation, dobutamine is the treatment of choice.
- (g) Nutritional support and Thromboprophylaxis should be given to all septic obstetric patients.

Management of Septic Shock

- Overall goals
 - Treat the mother!
 - Resuscitating the mother will resuscitate the fetus
 - Delivery attempts increase maternal and fetal mortality assuming the source is not intrauterine
 - Improve functional intravascular volume
 - Establish and maintain an adequate airway
 - Determine the septic foci
 - Empiric antibiotic therapy : know the most common pathogens

Management of Septic Shock

- Volume resuscitation
 - Aggressive therapy will optimize afterload, preload and cardiac contractility
 - Normalize mixed venous oxygen saturation, lactate concentrations, base deficit and pH
 - Blood products, colloid, crystalloid
 - Central venous access recommended
 - Pulmonary artery catheter may cause more harm

The Importance of Blood Cultures in Sepsis

- Recommended to draw prior to antibiotic administration, but should NOT delay antibiotics
- If antibiotics have been administered, still have cultures drawn
- When patient not responding to antibiotic regime, blood culture results are used to narrow antibiotic treatment to most appropriate antibiotic choice
- The culture results are also used to narrow down the antibiotic choice

The Importance of Measured Lactate Level

- Prognostic value of raised lactate levels are well established in septic shock patients
- Elevated levels in sepsis support aggressive resuscitation
- Mortality is high (46.1 %) in septic patients with both hypotension and lactate > 3.9 mmol/L
- Mortality in severely septic patients with Lactate >3.9 mmol/L alone is 30%

Fluid Resuscitation in Septic Patient

- Administer 30ml/kg Crystalloid for Hypotension or Lactate > 3.9 mmol/L, NS
- Patients with severe sepsis/septic shock experience ineffective circulation due to the vasodilation associated with infection or impaired cardiac output
- Poorly perfused tissue beds result in global tissue hypoxia, which result in serum lactate level

Fluid Resuscitation in Septic Patient

- A serum lactate is correlated with severity of illness and poorer outcomes even if hypotension is not present.
- Patients with hypotension or lactate > 3.9 mmol/L require intravenous fluids to expand circulating volume and restore perfusion pressure

Antibiotics/Antivirals/Antifungals

- US Food and Drug administration (FDA) categorizes drugs from A to D (in order of increasing fetal risks) and X (contraindicated) according to the risk to fetus.
- Commonly used antimicrobials such as penicillin, cephalosporin, macrolides and acyclovir fall into category A whereas aminoglycosides, quinolones, vancomycin and amphotericin fall in category C.
- Parturient have high incidence of vaginal candidiasis because of increased secretion of sex hormones. Topical anti-fungals should be the first line in the first trimester. There is a raised concern regarding oral fluconazole (category D) being associated with an increased risk of spontaneous abortions and stillbirths in addition to its teratogenicity in high dosage.

Broad Spectrum Antibiotics within 3 hours of T-0– (Administer as soon as possible)

- Administration of APPROPRIATE antibiotics reduces mortality in patients with Gram-positive and Gram negative bacteremia
- Although restricting antibiotics is important for limiting super-infection and decreasing development of antibiotic resistance, patients with severe sepsis and septic shock warrant broad spectrum antibiotic therapy until antibiotic susceptibilities are defined.
- Combination therapy is more effective than monotherapy until causative organism is found

Gold Standard Antibiotics for Common Infections In Obstetrical Patients

➤ *Chorioamnionitis*

- ✓ Ampicillin 2 g IV Q6hr over 60 minutes
- ✓ Gentamicin 1.5mg/kg/dose IV Q8H over 60 min
- ✓ Add Clindamycin 900mg IV Q8H over 30 min (for anaerobe coverage if patient has C/S)

➤ *Endometritis*

- ✓ Ampicillin 2 g IV Q6H for 60 min
- ✓ Gentamicin 5mg/kg/dose, IV Q24H for 60 min
- ✓ Clindamycin 900mg IV Q8H for 30 min

Gold Standard Antibiotics for Common Infections In Obstetrical Patients

➤ *Pyelonephritis*

- ✓ Ceftriaxone 1g in 50ml NS IV Q24H over 30 min
- ✓ For Ceftriaxone allergy, order Ampicillin 1 g IV Q6h over 60 min and Gentamicin 1.5 mg/kg/dose, IV Q8h over 60 min

➤ *Community Acquired Pneumonia*

- ✓ Ceftriaxone 1g IV Q24H over 30 min
- ✓ Azithromycin 500mg IV Q24H over 60 min
- ✓ IF MRSA suspected, Add Vancomycin 1mg IV Q12H

Choice of Antibiotics in Severe Sepsis & Septic Shock

- Discontinue all current antibiotics,
- Give Piperacillin-Tazobactam 3.375 G IV now and continue pharmacy dosing
- Add Vancomycin Per pharmacy dosing schedule
- If penicillin allergy: Cefepime 2 gm IV now and continue pharmacy dosing

Management of Septic Shock

- Inotropic agents
 - Dopamine hydrochloride (α -adrenergic and β -adrenergic effects)
 - Dobutamine
 - Norepinephrine – now considered first line therapy
 - Increases mean arterial pressure
 - Can reduce uterine artery blood flow
 - Isoproterenol

Concept of Septic Shock in 2015

- Early in sepsis there is an increase in inflammatory mediators - then SHIFTS
- Mid- to late sepsis consistent with immunosuppression
 - loss of delayed hypersensitivity
 - inability to clear infection
 - predisposition to nosocomial infections

Thromboprophylaxis

- Obstetric critically ill patients have four times higher risk of developing deep vein thrombosis compared to other critically ill patients. This is because all three components of Virchow's triad are potentiated during pregnancy:
- Stasis: Gravid uterus compresses IVC blood flow
- Hypercoagulability: Increased secretion of clotting factors due to high estrogen levels
- Endothelial injury: Utero-placental vascular injury at the time of delivery

Thromboprophylaxis

ACOG	ACCP	RCOG	Swedish guidelines
Perioperative mechanical thromboprophylaxis recommended for all patients undergoing caesarean delivery	Perioperative mechanical thromboprophylaxis recommended for all patients undergoing caesarean delivery	Risk factors (LMWH recommended for any of the following risk factors)	Heparin administered if risk score is 2 points or higher ≥ 2 points
Pharmacologic prophylaxis (LMWH or UFH) recommended for High-risk thrombophilias	Pharmacologic prophylaxis (LMWH or UFH) recommended for High-risk thrombophilias	Previous VTE	Prior VTE
Any prior VTE event	Any prior VTE event	Antenatal anticoagulation	Antenatal anticoagulation
A family history of VTE thrombophilia	A family history of VTE and a thrombophilia	Caesarean in labour	Immobilization
	Medical conditions	Asymptomatic thrombophilia	Mechanical heart prosthesis
	Systemic Lupus erythematosus	Prolonged admission	1 point
	Heart disease	Major medical co-morbidities (e.g. heart or lung disease, systemic Lupus erythematosus, cancer, inflammatory conditions, nephrotic syndrome, sickle cell disease, intravenous drug user)	Low risk thrombophilia
	Sickle cell disease		BMI >28 kg/m
	Blood transfusion		Family history of VTE
	Postpartum infection	Age >35	Age >40
	Minor risk factors (two needed for prophylaxis)	BMI >30 kg/m	Pre-eclampsia
	BMI >30 kg/m	2	Hyperhomocysteinaemia
	2	Parity ≥ 3	Placental abruption
	Multiple pregnancy	Smoker	Inflammatory bowel disease
	Emergency caesarean	Any surgical procedure	Other major risk factors
	Smoking >10 cigarettes/day	Gross varicose vein	
	Fetal growth restriction	Current systemic infection	
	Thrombophilia	Immobility	
	Protein C deficiency	Pre-eclampsia	
	Protein S deficiency	Mid-cavity rotational operative delivery	
	Pre-eclampsia	Labour >24 hours	
		PPH >1 litre or transfusion	

ACOG=The American Congress of Obstetricians and Gynecologists, ACCP=American College of Chest Physicians, RCOG=Royal College of Obstetricians and Gynecologists

Case Study # 3

- 34 y/o female, obese, DM2, 14 weeks gestation, Mother of 4, with 6 total pregnancy, 2 previous miscarriage, seen in outpatient clinic,
- Which pool does she belong to?
- What would you do?

Case #3 Continued

- Patient returns at 38 weeks gestation, with preeclampsia which was diagnosed at 24 weeks of gestation,
- Admitted for previously arranged C-Section.
- Which pool does she belong to?

Case #3 Continues

- 3 weeks post-C-Section returns with abdominal pain and secretions from surgical site?
- Surgical site is debrided and an abscess was drained
- Which pool does she belong to?
- What would you do?

Case # 3 Continues

- 6 PM, phone call from nursing station reports a brief episode of “loss of consciousness”
- What is on your differential diagnosis?
- What would you do?

Case # 3, History and Physical!

- No details of the event is documented!
- No Physical Exam is Documented with the exception of a set of vitals:
- HR: 110, BP: 85/60, RR 24, O2 Sat 89%
- What should have we done?

Case # 3 Continues

- At 6 AM patient was found unconscious, (no pulse), in her hospital bed?
- In terms of resuscitation what are you thinking about?
- What would you do?
- Do we know ACLS in peripartum?

Case #3 Continued

- Why would this patient have a cardiac arrest?
- What if this patient was still pregnant?
- Given you don't know the time of the arrest would you still run a code on the patient?

Outstanding Questions

- What are the risk factors of DVT and PE in pregnancy?
- Is pregnancy in itself a risk factor for DVT and PE?
- Does the stage of pregnancy matter?
- What are the cardinal symptoms of DVT and PE in pregnancy?
- What are the cardinal sings of DVT and PE in pregnancy?

Outstanding Questions

- What percentage of pregnant patients with DVT would also have PE?
- Does the location of clot differ in pregnancy?
- Does other complication of pregnancy such as preeclampsia/eclampsia relate to DVT and PE
- Does C-section relate to higher risk of DVT/PE?
- How about treatment of DVT and PE in pregnancy?
- What is the role of DVT/PE in maternal mortality in IRI?

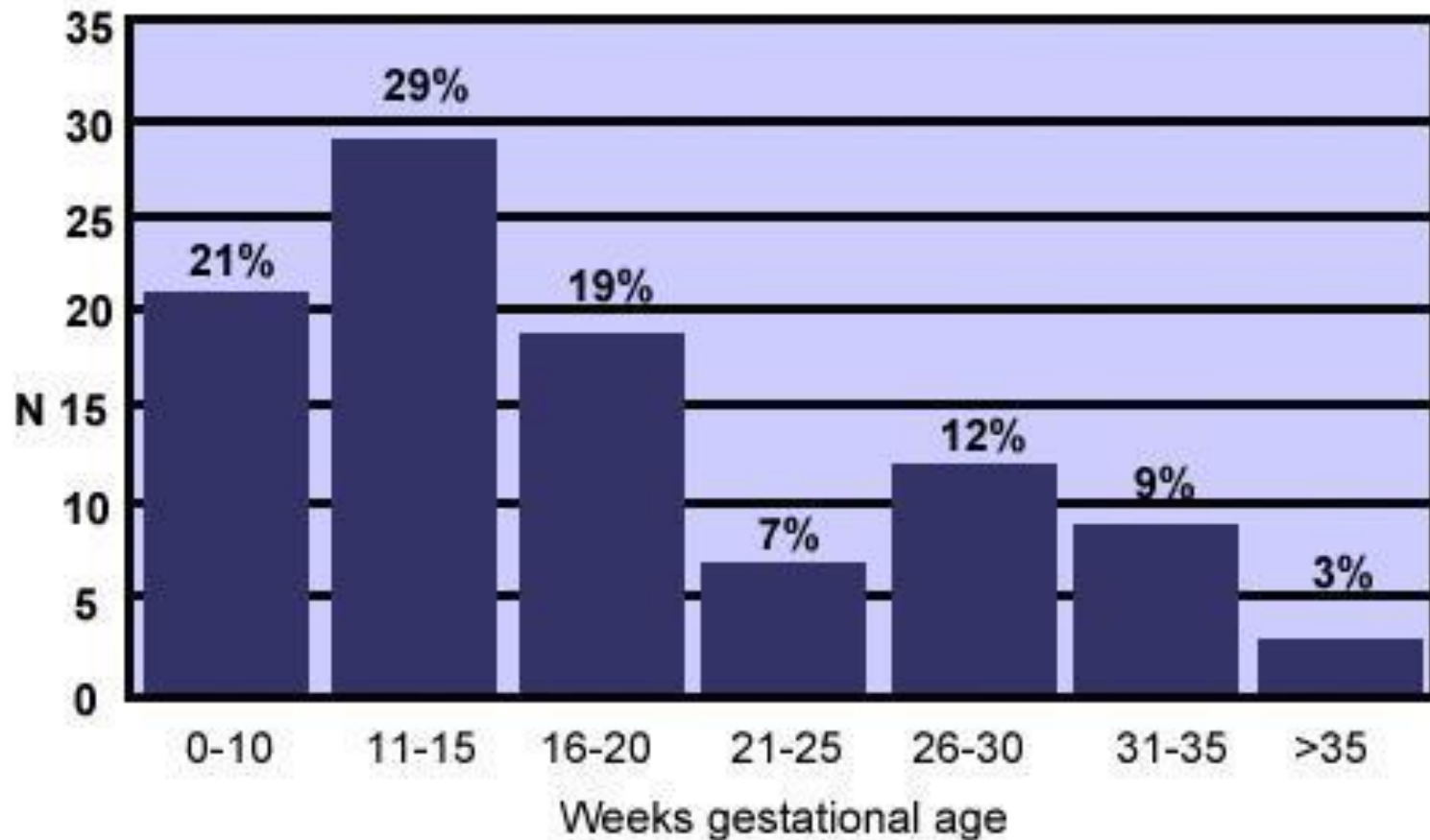
Back to Our Patient

- Let us take a systematic and evidence based approach to this patient so that we can identify the gaps.
- How would you start?

Background

- Pregnancy increases the risk of venous thromboembolism (VTE) 4- to 5-fold over that in the non-pregnant state.
- Most evidence suggests that VTE is more common in the postpartum period. In a 30-year population-based study, Heit et al documented that the risk of VTE and pulmonary embolism (PE) was 5-fold and 15-fold, respectively, in the postpartum period compared to during pregnancy.
- The two manifestations of VTE are deep venous thrombosis (DVT) and pulmonary embolus (PE).
- Although most reports suggest that VTE can occur at any trimester in pregnancy, studies suggest that VTE is more common during the first half of pregnancy.
- Sequelae of DVT and PE include complications such as pulmonary hypertension, post-thrombotic syndrome, and venous insufficiency.

VTE Trimester in Pregnancy



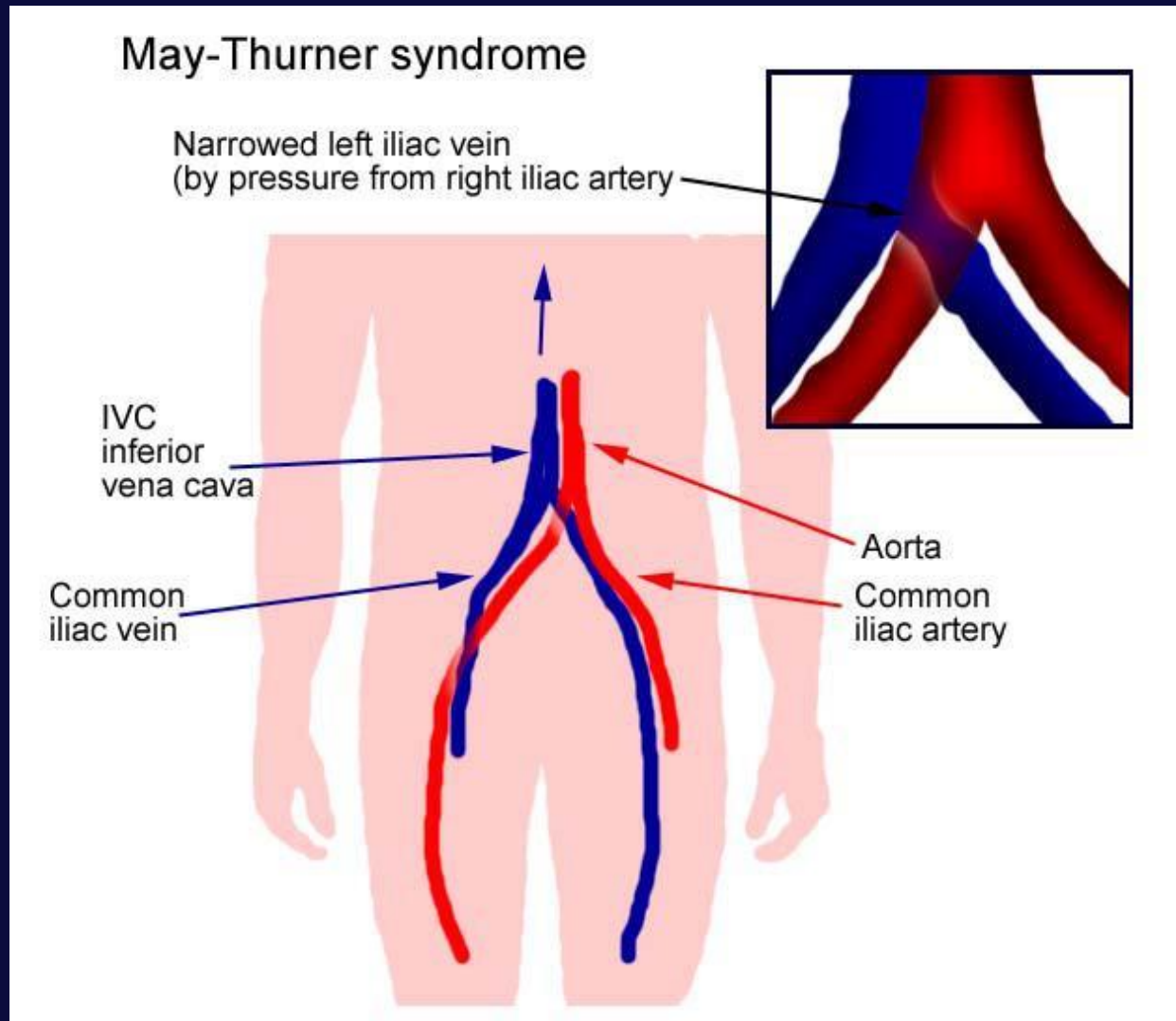
Estimated gestational age at time of diagnosis of antepartum deep venous thrombosis (n=94).

Special considerations in pregnancy

(Location of DVT)

- In pregnancy, deep venous thrombosis is much more likely to occur in the left leg compared with the right leg. One study examined 60 cases of DVT in pregnancy: 58 occurred in the left leg, 2 were bilateral, and none occurred in the right leg. The predilection for left lower extremity DVT is postulated to be the consequence of May-Thurner syndrome, in which the left iliac vein is compressed by the right iliac artery.

The May-Thurner Syndrome



Special considerations in pregnancy (Location of DVT)

- Another special consideration relevant to accurate diagnosis in pregnancy is that 12% of DVTs in pregnancy are in pelvic veins, whereas only 1% of DVTs in the general population are in pelvic veins. This predilection for pelvic site DVTs in pregnancy warrants special consideration when Doppler ultrasound studies of the lower extremities do not demonstrate non-compressible regions of lower extremity veins, yet suspicion for VTE is high or Doppler flow analysis of venous flow is abnormal.

Pathophysiology of VTE

- Pregnancy is a state characterized by Virchow's triad (1: hypercoagulability, 2: venous stasis and turbulence, 3: endothelial injury and dysfunction).
- Pregnancy is a state of hypercoagulability due to alterations of coagulation proteins. Factors I, II, VII, VIII, IX, and X increase in pregnancy.
- Pregnancy increases resistance to the anti-thrombotic factors such as Protein C, and Protein S.
- Thrombophilias can exacerbate these changes in coagulation proteins, further increasing the patient's risk for VTE

Thrombosis in Pregnancy

Factors affecting thrombosis: Pregnancy

Activation-Procoagulant

↑ Fibrinogen
Factors II, V, VII,
VIII, X

Coagulation

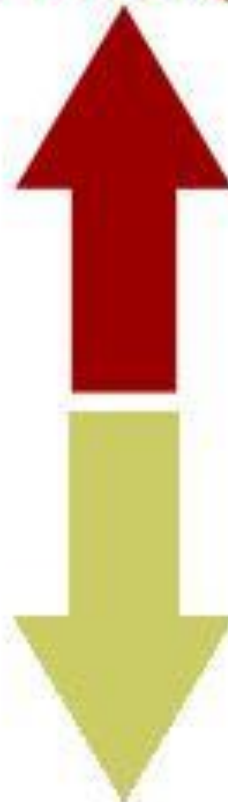
↓ ATIII
↓ Protein C
↓ Protein S
↓ TFPI
↑ Protein C
↑ resistance

Plasminogen

Fibrinolysis

↓ PAI-I
↓ Fibrinolysis

Inhibition-Anticoagulant



Venous stasis in Pregnancy

- Venous stasis also increases as dilation of lower extremity veins occurs followed by venous compression by the gravid uterus and enlarging iliac arteries.
- Situations of decreased mobility (eg, surgery, cesarean delivery, bed rest, prolonged travel or air travel) may exacerbate these factors.
- Endothelial injury may transpire at time of delivery. These factors work together to increase risk of VTE in the pregnant and postpartum patient.

Sequelae of VTE

- Sequelae of DVT and PE include complications such as pulmonary hypertension, post-thrombotic syndrome, and venous insufficiency.
- Post-thrombotic syndrome is not well defined; however, most definitions consist of a constellation of symptoms (pain, cramps, heaviness, pruritus, and paresthesia) and signs (edema, skin induration, hyperpigmentation, redness, ... and in more severe forms, venous stasis ulcer).
- Mild post-thrombotic syndrome occurs in 20-40% of patients after VTE, and severe post-thrombotic syndrome occurs in 5% of patients after VTE.

Epidemiology of Thromboembolism in Pregnancy

- According to the Center for Disease Control's National Pregnancy Registry Surveillance System, between 1991 and 1999, pulmonary embolism (PE) was the leading cause of maternal mortality. Of the 4,200 pregnancy related deaths reported in the United States during those years, 20% of maternal death was attributed to PE, surpassing pregnancy-related hypertensive disorders, postpartum hemorrhage, and infection. The CDC Pregnancy Mortality Surveillance System data from 2011-2012 also reported that 9.0% of maternal death within a year of pregnancy termination that was determined to be pregnancy related was attributable to thromboembolism.

Epidemiology of Thromboembolism in Pregnancy

- In IRI PE remains among the top 4 etiologies of maternal mortality
- It is likely that PE as one of the major etiologies of maternal mortality is underestimated
- Perhaps what is categorized as maternal death of unknown etiology, may in part be caused by PE.

Clinical Presentation, History and Physical Examination (DVT)

- Signs and symptoms of VTE are nonspecific and common in pregnancy. Most pregnant women experience mild tachycardia, tachypnea, dyspnea, and lower extremity edema. This makes the diagnosis of VTE by physical examination somewhat challenging.
- The 2 most common symptoms of DVT are pain and swelling of the lower extremity and 80% of pregnant women with DVT experience these symptoms.
- In a cohort of 53 women diagnosed with a DVT either antepartum (n=34) or postpartum (n=19), the 2 most common symptoms were edema (80-88%) and discomfort in the extremity (80-95%).

History and Physical Examination (DVT)

- A cross-sectional study of 194 pregnant women with no prior history of VTE evaluated the reliability of 3 variables in assessing risk of DVT by physical examination. Chan et al used the mnemonic LEFt:
 - L- Symptoms in the left lower extremity
 - E-Edema: Mid-calf circumference difference of $\geq 2\text{cm}$
 - Ft- First trimester presentation
- DVT was not diagnosed in women in the absence of any of these factors. Of the women with one finding, 16% had DVTs; 58% of the women with 2 or 3 findings were diagnosed with DVT.

History and Physical Examination (PE)

- Clinical signs and symptoms of PE are nonspecific. The classic symptoms of PE are dyspnea (82%), abrupt onset chest pain (49%), and cough (20%).
- The most common presenting signs are tachypnea, crackles, and tachycardia. No commonly used scoring system for the prediction of PE has been studied systematically in pregnancy.
- All of these signs and symptoms of PE are only rarely encountered together. These symptoms and signs are also commonly found in the pregnant patient, confounding the clinician's ability to make the diagnosis of this life-threatening process.

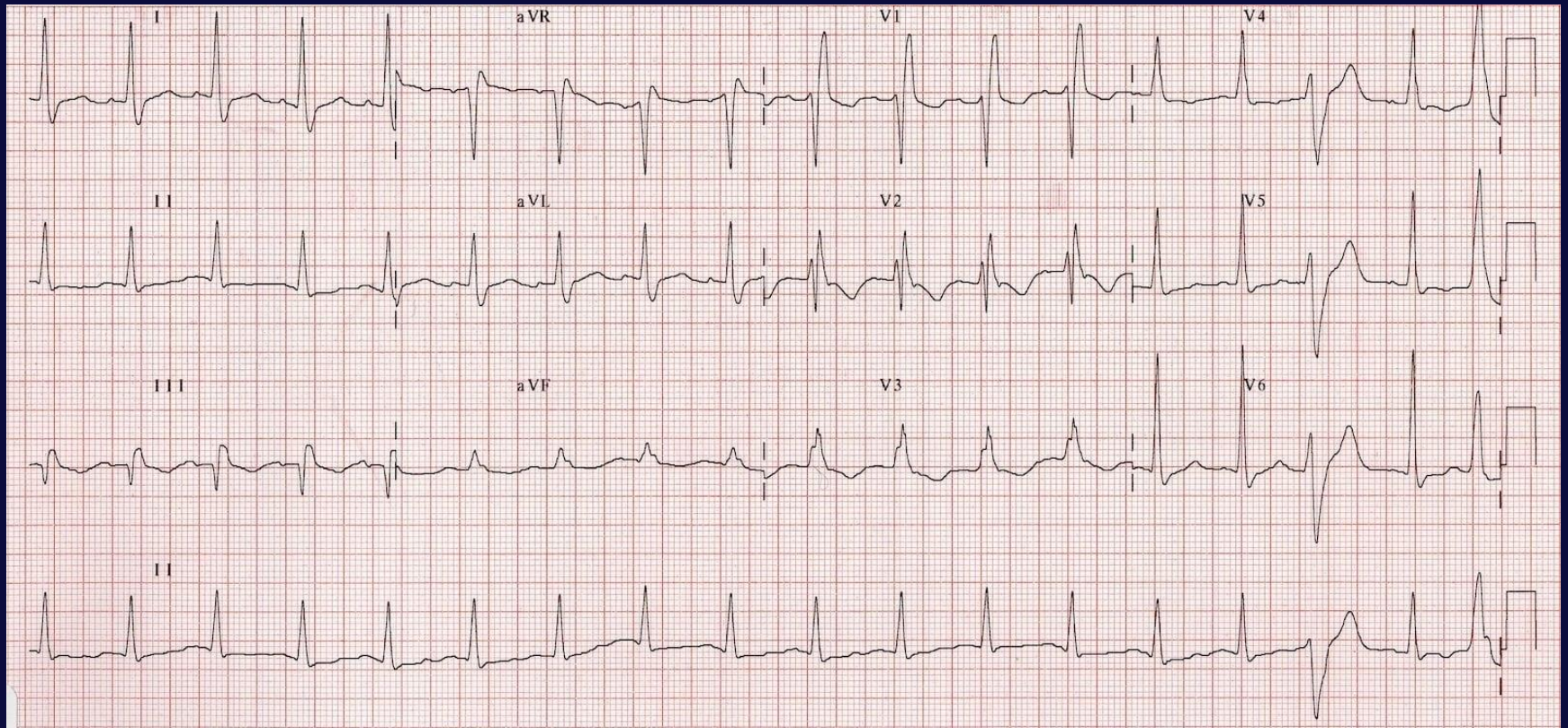
History and Physical Examination (PE)

- If the clinician suspects PE, anticoagulation therapy and appropriate immediate diagnostic testing should be performed until the diagnosis is made or eliminated as a possibility.
- Patients with massive PE may present with syncope, hypotension, pulseless cardiac electrical activity, or death.

Electrocardiogram of PE Patient

- An electrocardiogram from a maternal patient with PE may exhibit findings, such as right ventricular strain and the S1Q3T3 pattern suggestive of pulmonary embolism, but these findings are infrequent and generally nonspecific. Seventy percent of patients with PE have nonspecific EKG abnormalities, findings such as tachycardia, nonspecific ST segment, and T-wave abnormalities.

Electrocardiogram of PE Patient



Thrombophilia

- Thrombophilia is a common risk factor for VTE in pregnancy and can be found in 20-50% of pregnant women presenting with VTE.
- Screening for thrombophilias should be done if the results are likely to alter management.
- Screening is unnecessary when treatment is indicated for other reasons. The results of screening may be affected if the patient is currently pregnant, currently has an acute VTE, or is currently receiving anticoagulation therapy.

Laboratory Evaluation for DVT

- D-dimer is often used in non-pregnant patients due to its high negative predictive value.
- D-dimer increases progressively throughout gestation, adding to the difficulty in selecting an appropriate cut off value for reasonable specificity in pregnancy.
- The high negative predictive value of d-dimer for DVT in pregnancy has also been demonstrated prospectively; Note, however, data to support its use in the setting of suspected PE is sparse, and D-dimer may have lower sensitivity in pregnancy.

Laboratory Evaluation for DVT

- A study by Chan et al suggests that DVT may be safely excluded if the d-dimer is negative and the compression duplex ultrasonography (CUS) is normal. 149 consecutive pregnant women were evaluated for possible DVT. Twelve were diagnosed with DVT and 1 with PE. D-dimer testing was positive for all 13 patients (100% sensitivity), and the specificity was 60%.
- This study demonstrated a 100% negative predictive value in pregnancy. Use of a sensitive d-dimer test coupled with compression duplex ultrasonography may be a useful algorithm for evaluation of DVT in pregnancy, although more research is needed in this area.

- Early diagnosis and intervention is key to successful management of the maternal critical care patients....stop the train before the cliff...



Case # 4

- 18 years old female 5 months pregnant, presented to ED at 4 PM with SOB and cough
- Several Weeks History of Progressive Cough and SOB, Significant weight loss and constitutional Symptoms
- So far has seen 4 physicians in last 5 weeks and has taken 4 doses of antibiotics and used several inhalers, with no improvement but only worsening symptoms

- As a receiving physician what would you do?
- What would you start with?

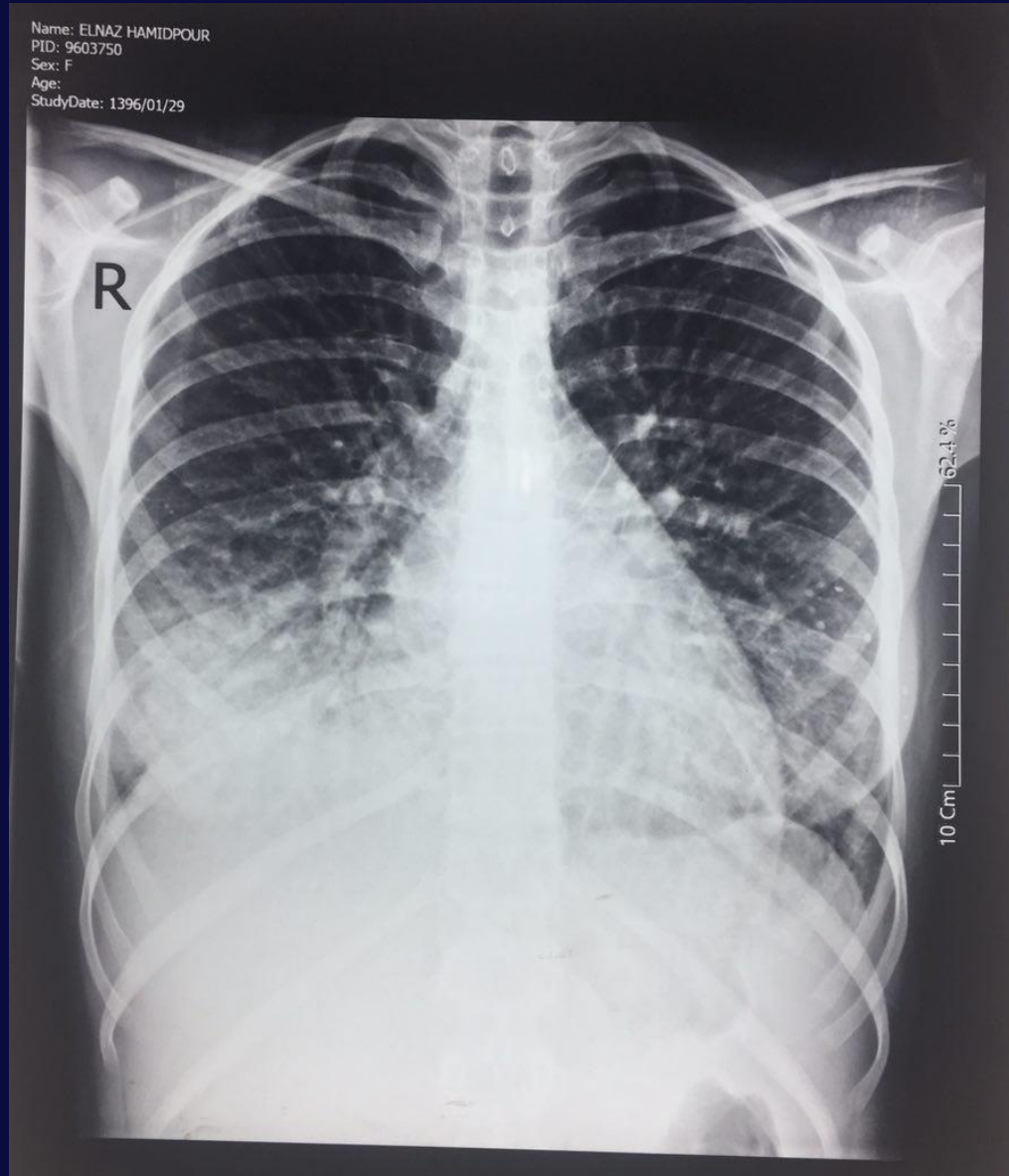
Physical Exam

- Vital Signs: BP 85/58, HR 130, RR 40, O2Sat 85%, Temp: 38.8, GCS 15,
- Underweight, clearly in respiratory distress, normal mental status but unable to speak in full sentences,
- On Physical Exam; Soft Heart Sounds, Bilateral Inspiratory Crackles, Elevated JVP, cold extremities,....
- How would you proceed with stabilization?

Sequence of Stabilization

- Maximum O2 therapy
- Central IV access
- Before attempting intubation, need to stabilize hemodynamic parameters and preoxygenation;
- Would you give fluid to this patient?
- What inotrope will you give to this patient?

What do you think about this CXR?



Case Unfolding!

- Day after admission to the infectious disease ward, patient was in severe respiratory fatigue, hypoxic, hypotensive, with altered mental status,
- Patient was given 2 units PRBC for her low hemoglobin, following which became profoundly hypoxic,
- Had a cardiac arrest during intubation and resuscitation was unsuccessful,

احتراماً بیکر خانم ۱۸ ساله که بدن و تنه بی رمی قبلی سرور را از چند ماه قبل از
بی کس که طی روزهای گذشته افزایش یافته و همراه با تشنجه و ریسش به برده است
بی دردی حین خواب و نشسته تحت درمان آرم (طبی) یعنی قفسه عین داخل (برده
است طی مجبوری نداشته؛ بی دردی بکشی ۴۰ دگر است گنگاهی داشته و ریسش تشنجه
واقع زرد (علی رقم ۵۵۲ کفایت با ۵۰۰ نازل) و شامی کی روی ۱۴۰-۱۳۰
نیز زرد؛ خوابگاه است بی دردی زرد ۲۱۵ آهلی به ریسش محمود و زرد

مشاهدات و نظریات پزشک معالج مشاور (خلاصه نظریات، تشخیص و توصیه ها) : (دکتر رضا حسینی)

ادب سے بہترین کیلئے روحِ انت و شکر کو بہ ہر

سلام و اقلیم بسیار زیاده. خانم ۱۸ ساله که به همراه گدازه سنگی نرسیده است و طبق شرح بالا
با تستر اسم در حال درمان بوده و از پروژا Consolidation در قاعده ریه راست با تستر بزرگ
و در این شروع شده است در معاینه هوای ریه راست ریه بزرگ دارد. معاینات و تست
تستر استفاده می کنند. است. که SO_2 به کرده است. که به دور CO_2 85-80 پایین می آید
فاکس و با استیک ۹۰٪ است. در معاینه علام حیاتی $HR=125-130$ $RR=40$ $BP=80$
شک ششها و اعصاب:

نام پزشک مشاور و امضاء: نام بیمار:	درماتیت ریه‌ها صدهای ریه و ریویژور و قریه است. رال و خراش صدای ریه و ریویژور و قریه است. رال و خراش صدای ریه و ریویژور و قریه است. رال و خراش صدای ریه و ریویژور و قریه است.
BP: 84 / RR: 40 / HR: 125-130 تاریخ:	Consultant Physician Name & Signature:

Complex cardiac diseases

- Cardiac disorders affect 1–3% pregnant women and account for 10–15% of maternal mortality.
- Peripartum cardiac diseases in pregnancy range from congenital anomalies to valvular heart diseases, myocardial infarction, dilated cardiomyopathy, pulmonary hypertension, etc., which are further worsened by high cardiac output physiology of Pregnancy
- Cardiogenic shock can be managed with timely correction of pump failure that includes early revascularization, inotropic support for impaired myocardial contractibility and even mechanical circulatory support for refractory cases.
- In cardiogenic shock patients in whom medical management fails and valvular repair is not feasible, use of intra-aortic balloon pump (IABP), ECMO and continuous flow left ventricular assist device (LVAD) have been tried, but data on their use is limited.

Complex cardiac diseases

- Different vasopressors such as norepinephrine (Category C) and phenylephrine (Category C) and inotropes such as dobutamine (Category B) and milrinone (Category C) and the inotrope/vasopressor epinephrine (Category C) have been used in pregnancy. The potential risk of placental vasoconstriction by vasopressors must be weighed against the risk of hypoperfusion caused by systemic hypotension.
- Obstructive valvular lesions such as mitral stenosis (MS) and aortic stenosis (AS) have worsened functional status with pregnancy due to increased cardiac output, ventricular volume, transvalvular pressure, decreased diastolic time and associated pulmonary hypertension.
- Severe MS may present with pulmonary edema and atrial fibrillation while AS may develop fatal arrhythmias and refractory heart failure. American heart association (AHA) recommend beta-blockers, nondihydropyridine calcium channel blockers (All class C to D) and digoxin for atrial fibrillation in pregnancy.

Case # 5

- 28 years old female, 28 weeks pregnant, has presented several times to emergency department of a rural hospital with escalating headache for last two weeks, now presenting with several episode of tonic clonic seizures and loss of consciousness, some lasting several minutes.
- You are the physician on call what would you do?

Case # 5 Continues,

- How would you start?

Case # 5 Continues,

Patient is conscious but confused,
does not obey command,

What do you need, to make clinical
decisions?

Case # 5 Continues,

At the heart of every clinical encounter is vitals,

Patient's Vitals:

BP 150/64, HR 54, RR 24, T 39.2,
O2 Sat 86%, GCS 8, (E2V2M4),

Principles of Stabilizing an Unstable Patient

- The aim of the initial treatment is to keep the patient alive, and achieve some clinical improvement. This will buy time for further treatment and making a diagnosis.
- Airway, Indication for intubation and securing of the airways,
- Breathing, what should one do in this regard?
- Circulation, How could we ensure circulation?
- Disability, What does this mean in terms of Resuscitation?
- Exposure, What would one look for in Exposing the patient?

Stabilization

- In the management of an unstable patient stabilization takes priority and diagnosis only becomes relevant if it helps the stabilization
- Diagnostic work up should not compromise stabilization

Case # 5 Continues,

- With that in mind what would you do?
- Should we sedate and intubate the patient?
- What would you use for sedation in a pregnant women?
- What about anti-seizure medication in a pregnant patient?
- Would you terminate the pregnancy?
- Can we analyze the history and vitals to understand the clinical case better and make relevant decisions?
- How would this patient die?

How Would You Investigate this Patient?

- Can we agree on a differential diagnosis so that we can carry the stabilization to the treatment phase?
- Can you choose the three most relevant investigations that you could ask for?

Differential Diagnosis?

DIMS as a Pneumonic for “Decreased Level of Conciseness”,

- D, Drugs, Could that be drug toxicity,
- I, Infection/Inflammation/Ischemia,
- M, Metabolic Disorder,
- S, Structural Abnormality, Stroke, Trauma, Cerebrovascular thrombosis
-

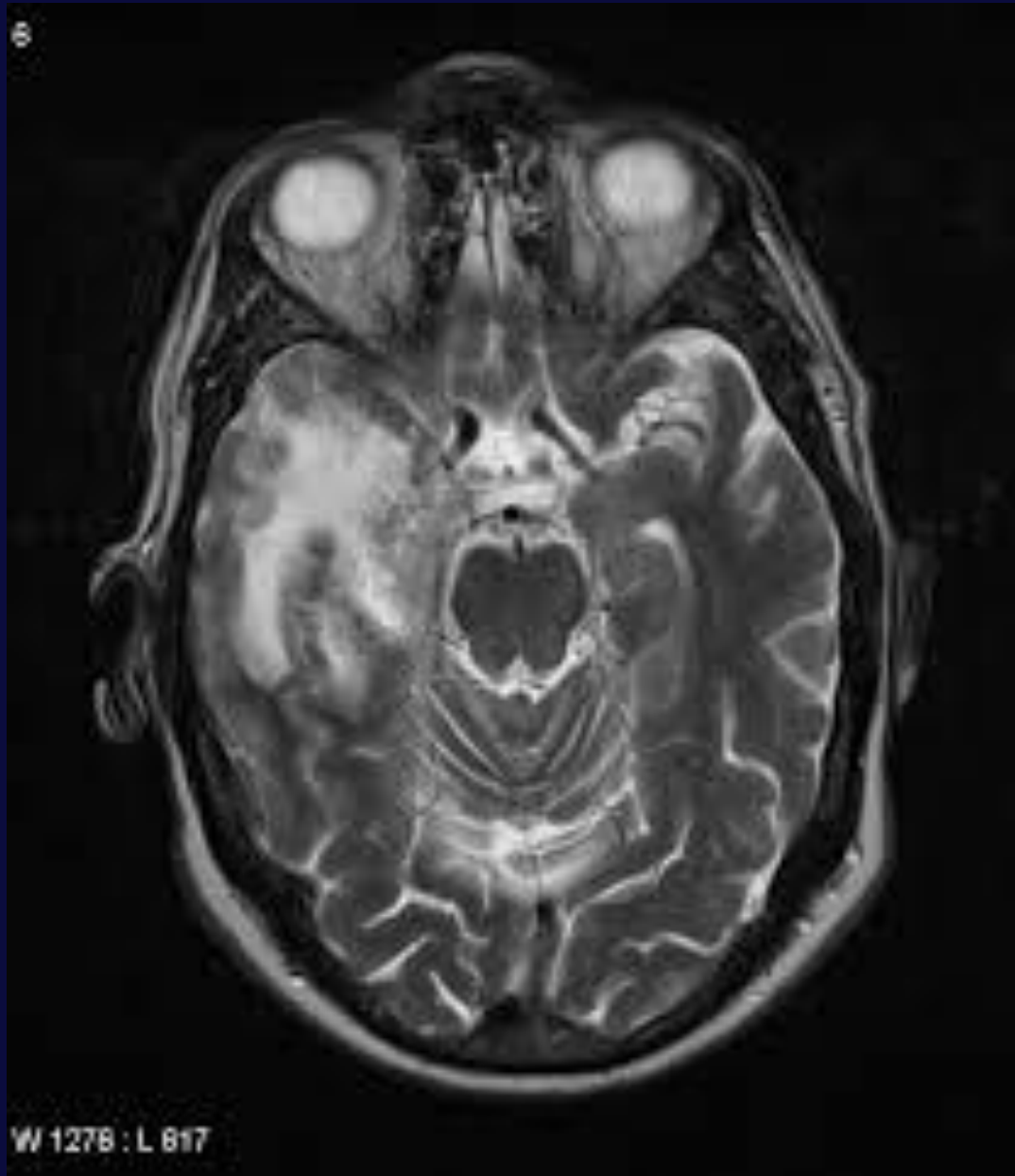
Relevant Investigations

- If you are asking for more than 3 investigations, perhaps you need to understand the case better!
- Toxicology (Urine and Blood Toxic Screen)
- CBC
- MRI of the Brain,
- Why this could not be a case of Metabolic Disorder?

Investigations Outcome

- WBC 14, Lymphocytes 60%,
- Toxicology Screen Negative
- MRI to follow,

MRI image of the Patient's head



Case # 5 Continues,

What would you find in the physical exam of this patient?

Do you remember the vitals?

What should we do to manage a raised ICP?

Case# 5 Outcome

- Patient suffered significant raised ICP
- Her raised ICP was managed medically
- Given the worsening clinical course, surgical intervention was implemented
- She survived the clinical complications and she continues to be followed...

Case Presentation # 6

- 41 years old female, with past medical history of CKD, Mitral Regurgitation, and preeclampsia, at 39 weeks gestation, presented with ruptured membrane 9 hours ago, she was in active labor, when presented with an acute episode of tachypnea, SOB, decreased level of consciousness followed by loss of consciousness...
- You are the attending physician at the scene, how would you proceed?

Case Presentation # 6

- Let us take a systematic and evidence based approach to this patient so that we can identify the gaps.
- How would you start?

Patient's Vitals

- BP 96/54
- HR 138
- RR 28
- O2Sat 80%
- Temp 38 C
- GCS 3

Outstanding Questions

- What is AFE and does it really exist?
- What are the risk factors of AFE?
- What is the prevalence of AFE?
- What is the mortality rate of AFE?
- How does the AFE present?
- What are the cardinal symptoms of AFE?
- What are the cardinal sings of AFE?
- What about the management of AFE?
- What is the role of AFE in maternal mortality in IRI?

Definition/Prevalence

- Amniotic fluid embolism (AFE) is a life threatening obstetric emergency characterized by sudden cardiorespiratory collapse and disseminated intravascular coagulation.
- AFE occurs in 2-8 per 100,000 deliveries and is responsible for between 7.5% to 10% of maternal mortality in the United States.

History

- Steiner and Luschbaugh first described AFE in 1941, after they found fetal debris in the pulmonary circulation of women who died during labor. Data from the National Amniotic Fluid Embolus Registry suggest that the process is more similar to anaphylaxis than to embolism, and the term *anaphylactoid syndrome of pregnancy* has been suggested because fetal tissue or amniotic fluid components are not universally found in women who present with signs and symptoms attributable to AFE.

Diagnosis Controversy

- The diagnosis of AFE has traditionally been made at autopsy when fetal squamous cells are found in the maternal pulmonary circulation; however, fetal squamous cells are commonly found in the circulation of laboring patients who do not develop the syndrome. The diagnosis is essentially one of exclusion based on clinical presentation. Other causes of hemodynamic instability should not be neglected.

Etiology

- Amniotic fluid embolism (AFE) is considered an unpredictable and unpreventable event with an unknown cause.
- In the national registry, 41% of patients had a history of allergies.
- Reported risk factors for development of AFE include multiparity, advanced maternal age, male fetus, and trauma.
- In a retrospective review of a 12-year period encompassing 180 cases of AFE, of which 24 were fatal, medical induction of labor increased the risk of AFE. In the same study, AFE was positively associated with multiparity, cesarean section or operative vaginal delivery, abruption, placenta previa, and cervical laceration or uterine rupture.

Epidemiology

- The true global incidence of amniotic fluid embolism is unknown because of inaccurate diagnoses and inconsistent reporting of nonfatal cases.
- In 2011, AFE was the leading cause of death during childbirth in Germany.
- In Australia, AFE is cited as the leading direct cause of maternal death. Estimates range from 1 in 8000 to 1 in 80000 deliveries.
- In the United Kingdom incidence is estimated at 1.9 per 100000 to 7.7 per 100000 deliveries.

Prognosis, Mortality/Morbidity

- Maternal mortality approaches 80%. However, it was 61% in the US national registry, which listed 46 cases.
- Amniotic fluid embolism (AFE) is the cause of 5-10% of maternal mortality in the United States.
- Of patients with AFE, 50% die within the first hour of onset of symptoms. Of survivors of the initial cardiorespiratory phase, 50% develop a coagulopathy.
- A population-based study using the California Office of Statewide Planning and Development database reviewed 1,094,248 deliveries over a 2-year period. Of 53 cases of AFE, 14 patients (26.4%) died and 35 patients (66%) developed DIC.

Prognosis, Mortality/Morbidity

- Maternal survival is uncommon, although the prognosis is improved with early recognition and prompt resuscitation.
- The United Kingdom AFE registry reported a mortality of 37%; of the women who survived AFE, 7% were neurologically impaired.
- Neonatal survival has been reported to be 79% in the US registry and 78% in the UK registry. The intact infant survival rate is 70%. Neurologic status of the infant is directly related to the time elapsed between maternal arrest and delivery.
- The risk of recurrence is unknown. Successful subsequent pregnancies have been reported. The recommendation for elective cesarean delivery during future pregnancies in an attempt to avoid labor is controversial.

Complications

- Pulmonary edema is a common occurrence in survivors (pay close attention to fluid input and output).
- Left heart failure may occur. Some sources recommend inotropic support.
- Treat DIC with blood components. Consider activated factor VIIa for severe hemorrhage. Bilateral uterine artery embolization has been successful in controlling blood loss in 2 reported cases.

Clinical Presentation/ History

- In a study of 33 cases of AFE in Australia and New Zealand, the most common initial symptoms were:
- A feeling of agitation or of impending doom (27%),
- Hypotension (21%), dyspnea (15%) and evidence of fetal compromise (15%).
- Coagulopathy was the initial symptom in only 3% of patients but 73% ultimately developed a coagulopathy, 85% required transfusion of blood products.
- Six percent of patients presented with either cardiac arrest or an arrhythmia.

Clinical Presentation/ History

- The classic history is that a woman in the late stages of labor becomes acutely dyspneic and hypotensive. There may be a preceding period of agitation or a sense of impending doom. Altered mentation may or may not be present. She may experience seizures quickly followed by cardiac arrest. If undelivered, the fetus will demonstrate loss of heart rate variability followed by decelerations and ultimately a terminal bradycardia. Massive DIC-associated hemorrhage follows and then death. Most patients die within an hour of onset.

Clinical Presentation/ History

- Data from the United Kingdom Obstetric Surveillance System (UKOSS) from 2005-2011 showed that 53% of women given a diagnosis of AFE presented at or before delivery. The remaining patients presented an average of 19 minutes after delivery.
- In the United States AFE registry 70% of patients presented during labor, 19% presented during cesarean delivery and 11% presented after delivery.
-

Clinical Presentation/ History

- There are case reports of AFE occurring during abortion, after abdominal trauma, amniocentesis and during amnioinfusion.
- A uniform diagnostic criteria for amniotic fluid embolism has been suggested in order to ensure that researchers use the same definition when reporting events. All of the following must be present for a diagnosis of AFE:

Clinical Criteria for the Diagnosis of AFE

1. Sudden onset of cardiorespiratory arrest or both hypotension (systolic BP < 90mm HG) and respiratory compromise (dyspnea, oxygen saturation < 90%)
2. Documentation of overt DIC following the events in item 1. Coagulopathy must be detected prior to the loss of enough blood to itself be the cause of a dilutional or consumptive coagulopathy.
3. Clinical onset during labor or within 30 minutes of the delivery of the placenta.
4. No fever (≥ 38.0 C) during labor

Assessment of DIC in Pregnancy

SCORE	0	1	2
Platelets	>100,000/mL	< 100,000/mL	< 50,000/mL
Prothrombin time or INR	< 25% increase	25-50% increase	>50% increase
Fibrinogen	>200 mg/L	< 200 mg/L	

International Society of Thrombosis and Hemostasis Scoring System for the diagnosis of DIC. A score ≥ 3 is consistent with overt DIC in pregnancy.

Physical Findings of AFE

- The classic triad of AFE is hypoxia, hypotension and coagulopathy.
- The following signs and symptoms are indicative of possible AFE:
- Hypotension: Blood pressure may drop significantly with loss of diastolic measurement.
- Dyspnea: Labored breathing and tachypnea may occur.
- Seizure: Tonic clonic seizures are seen in 50% of patients.
- Cough: This is usually a manifestation of dyspnea.
- Cyanosis: As hypoxia/hypoxemia progresses, circumoral and peripheral cyanosis and changes in mucous membranes may manifest.

Differential Diagnoses

- Anaphylaxis
- Aortic Dissection
- Abruptio Placentae
- Aspiration
- Cholesterol Embolism
- Myocardial Infarction
- Pulmonary Embolism
- Septic Shock

Amniotic Fluid Embolism Treatment & Management

- Admit the patient with amniotic fluid embolism (AFE) into the intensive care unit.
- Treatment is supportive and includes the following:
- Administer oxygen to maintain normal saturation. Intubate if necessary.
- Initiate cardiopulmonary resuscitation (CPR) if the patient arrests. If she does not respond to resuscitation, perform a perimortem cesarean delivery.
- Treat hypotension with crystalloid and blood products. Use pressors as necessary.

Amniotic Fluid Embolism Treatment & Management

- Avoid excessive fluid administration. During the initial phase, right ventricular function is suboptimal. Excess fluid may overdistend the Right ventricle which could increase the risk of a right sided myocardial infarction.
- Consider pulmonary artery catheterization in patients hemodynamically unstable.
- Continuously monitor the fetus. Deliver immediately following cardiac arrest if gestational age is ≥ 23 weeks.
- Early evaluation of clotting status and early initiation of massive transfusion protocols is recommended.
- Treat coagulopathy with FFP for a prolonged aPTT, cryoprecipitate for a fibrinogen level less than 100 mg/dL, and transfuse platelets for platelet counts less than 20,000/ μ L.

Treatment & Management

- Lim and colleagues reported a case of AFE in which the coagulopathy was treated with activated recombinant factor VIIa. The range of doses to treat serious bleeding is from 20-120 mcg/kg.
- Hemodialysis with plasmapheresis and extracorporeal membrane oxygenation (ECMO) with intra-aortic balloon counterpulsation have been described in case reports with successful outcomes in treating AFE patients with cardiovascular collapse. The use of anticoagulation during ECMO may worsen bleeding in patients with AFE. Use of ECMO is not routinely recommended.

Amniotic fluid embolism

- Amniotic fluid embolism (AFE) is a clinical diagnosis of exclusion which carries very high mortality (approximately 40%). The most widely accepted mechanism of its occurrence is 'anaphylactic reaction of pregnancy' associated with AFE.
- AFE is a potentially catastrophic condition with high maternal mortality and treatment is still only supportive.
- Management of AFE is mainly supportive and should be managed in an ICU setting by a multi-disciplinary team. Intra-aortic balloon counter pulsation, cardiopulmonary bypass and extracorporeal membrane oxygenation have been tried. Recombinant factor VII can be used as treatment option in DIC associated with AFE when the hemorrhage cannot be stopped by massive blood component replacement.

My Contact Information

- Please get in touch with me for lectures, workshops, research projects and clinical consults, in clinical medicine and maternal critical care, I will be in Iran for another year
- Telephone: 0914-485-0551
- Email: baran.serbest4@gmail.com