Avoidable anaesthetic deaths in parturients:

An appraisal of maternal mortality case sheets

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Tamil Nadu Government in an effort to reduce the maternal mortality rate, conducts a review of all the maternal mortality case sheets every month. An obstetrician and an anesthetist from Chennai analyse all the case sheets and conduct a video conferencing on the last Thursday of every month from the National Rural Health Mission Head Quarters. The obstetricians and Anesthetists in the districts report at the video conferencing hall in their respective collectorate offices. A face to face discussion of the events that lead to the death of the parturient

is carried out and the clinician is made to understand any omissions and commissions on his / her part.

Every month around 40 parturients die in various govt hospitals in Tamil Nadu. In the first 6 months of my evaluation, I could make out 10-12 parturient deaths to be anesthesia related.

Constant interaction with the Anaesthetist has resulted in the reduction in the no. of deaths to mere two (or) three cases in a month.

Commonest causes of anaesthetic mishaps in parturients.

1. Non Recognition of an ascending spinal anesthesia.
2. Lack of awareness about Normovolaemic pulmonary odema in severe preeclampsia.
3. Non recognition hypertensive failure in a dyspnoeic parturient.

1.Non Recognition of a ascending spinal anesthesia.

When subarachnoid block is given, an Anesthetist aims for a sensory block upto T5 or T6 level even for a Pfannenstiel incision. The dermatome to be anesthetised for pfannenstiel incision in T11, which is

a subumbilical incision. The root value for greater splanchnic nerve is T6 to T10 and for lesser splanchnic it is T11 T12.

These nerves conduct the afferent pathways from the small and large intestine. Since an obstetrician is likely to handle the bowel during the course of a caesarean section, an Anesthetist aims for a sensory block upto T6 to prevent discomfort to the patient during bowel handling.

The volume of 0.5% Bupivacaine (heavy) for LSCS is between 1.75ml to 2.25 ml depending on the stature of the patient. Anything above 2.5ml is fraught with the danger of a high subarachnoid block.

In comparison, for an acute appendicectomy surgery (with a Lanz or Mcburney's incision) even if the anesthetist gives 4ml of 0.5% Bupi heavy in a young patient, the level of block is not likely to rise beyond T6 or T5. Whereas a similar volume given to a parturient can go upto medulla oblongata because of reasons very well known to every anaesthetist.As for as the subarachnoid block in caesarean section is concerned it revolves round the question of reduced local anesthetic volume.

Even in a properly conducted SA block in LSCS, sometimes unexplained high spinal block can occur. If the anesthetist is not monitoring the chest wall movement, the patient is likely to land up in a catastrophic situation.

In women the respiratory pattern in thoraco abdominal.

Sluggish or absent chest wall expansion under the surgical drapes should alert the anesthetist regarding ascending level of block. No anesthesia text book gives an idea about how to segmentise the

ascending SA block. If there is an unexplained fall in Spo2 and if the chest wall movement is absent, the anesthetist should ask the parturient to give a firm grip of the anesthetist's fingers. If the parturient's hand grip is firm, then the level of block is still below T1. This is because the root value of upper limb innervation is C5 to T1. The anesthetist should push in the pillow from the head underneath the shoulder and give another pillow to the head. Gentle assisted ventilation will bring up the saturation and increasing the height of the torso will prevent further ascending of the block. Patient may require two or three 6mg boluses of Inj. Ephedrine to stabilise the blood pressure.

If the hand grip is sluggish, the anesthetist should look at the abdomen. If the abdomen is nicely expanding, it indicates the diaphragm is still acting and that the phrenic nerve is not involved. It indicates the block is still below C5 (Phrenic nerve root value is C3, C4, C5).

If the abdominal movement is also jerky that means the phrenic nerve is also involved and the patient will definitely require assisted ventilation to improve the saturation. The whole body chemical sympathectomy may even require addition of Dopamine or Adrenaline to the IV fluids and the fluid may have to be run in to get a palpable peripheral pulse. Once the radial pulse is felt the Vasopressor infusion can be gradually slowed down and another IV line may have to be started for volume replacement.

If the laryngoscopy evokes a gag reflex it is left to the anesthetist`s professional competence to decide about the ways and means of maintaining ventilation.

The clinical scenario in most of the on the table cardiac arrest is as follows. Spinal anesthesia given; patient turned supine; wedge kept; Even before the uterine incision, a sudden fall in saturation; IPPV given via face masks; saturation refractory to ventilation; Cardiac arrest; Patient could not be revived.

Patient in question is usually in her second (or) third decade with an absolutely normal cardiovascular and respiratory system without any preeclampsia features. Anesthetist forgets the fact that in high spinal, there is whole body Chemical sympathectomy, diminished venous return and grossly diminished lung perfusion to match the ventilation. Heart is nearly empty for any worthwhile cardiac output. A provisional diagnosis of amniotic fluid embolism is entered in the case sheet after the death of the parturient. Moral of the story is, once spinal anesthesia is given, closely monitor the chest expansion under the surgical drapes.

Lack of awareness about Normovolemic Pulmonary Oedema in severe preeclampsia

Anesthesia for a parturient with severe Preeclampsia for emergency LSCS: Here anesthetist either performs general anesthesia or subarachnoid block for a patient with established Preeclampsia as per the hospital protocol. Before the end of surgery there is a fall in Oxygen saturation and at the sametime anesthetist notices coarse crepitations all over the chest. He makes a diagnosis of pulmonary

edema. Inspite of diuretics, intubation and ventilation, patient runs into a cardiac arrest situation.

Genesis of pulmonary edema in preeclampsia:

At pulmonary capillary level, the Mean Arterial Pressure is 15-20 mm of Hg. Since Oncotic pressure due to plasma proteins is 24 mm of Hg, fluid does not escape into pulmonary interstitium under normal pulmonary conditions.

In a normal person when left atrial pressure is greater than 15 mm of Hg, pulmonary oedema develops. The is because this 15 mm of Hg combined with an outward drag of 8 to 9 mm of Hg subatmospheric Pressure, drives the fluid from capillaries to pulmonary interstitium.

In severe preeclampsia, where there is a Leaky capillary state and low oncotic pressure [less than 24 mm of Hg] secondary to protienuria, pulmonary oedema can develop with normal circulating blood volume. Because of this fact, if a Central Venous Pressure Catheter is in position, the CVP level must be maintained within 3-4 mm of Hg or 5 cm of water. Other conditions where normovolemic pulmonary oedema is seen are

a) High altitude pulmonary oedema
b) Neurogenic pulmonary oedema due to sudden increase in intracranial tension following head injury.
c) Extubatory laryngeal spasm in paediatric age group.

In a preeclampsia patient, rushing in intravenous fluid during surgery can very easily precipitate pulmonary edema due to the above

mentioned factors. In a preeclampsia patient, once the patient is anesthetised, the anesthetist should keep a watch on the external jugular vein size. If the vein becomes more prominent and if the saturation starts falling, it should be taken as an early warning regarding the onset of pulmonary edema. If the anesthetist fails to make this early observation he / she is likely to precipitate a florid pulmonary edema situation and reviving the patient from this juncture becomes very difficult due to the Leaky capillary syndrome and hypoproteinemia

Non recognition of hypertensive failure in a dyspnoeic parturient

DEFINITION: Dyspnoea is a subjective feeling for increase in respiratory effort.

CARDIAC CAUSES OF DYSPNOEA

1.Anemia with failure

2.Congenital / acquired heart disease

3.**Hypertensive failure of pregnancy induced hypertension. (Severe PIH/PET)**

4.Peripartum cardiomyopathy

5.Pulmonary embolism

PULMONARY CAUSES OF DYSPNOEA

1.Bronchial asthma

2.Community acquired pneumonia

3.Pleural effusion

4.Pneumothorax

If a parturient presents with dyspnoea in the casualty, keep the patient in propped up position. Get the baseline pulse oximeter reading. If saturation is < 90% give 6-8 litres of humidified oxygen to the mother. If the saturation reaches beyond 95% in 60 secs, then the dyspnoea is most probably pulmonary in origin. If saturation struggles to go beyond 90% the dyspnoea is most probably cardiac in origin.

Pleural effusion and pneumothorax can mimic cardiac pathology (SPO_2<90-92% even with 0_2).

Identifying a hypertensive failure among the dyspnoeic parturients requires a high degree of suspicion on the part of the anesthetist.

What the mind does not know the eye will not see.

A severe Preeclampsia patient is likely to run into one of the following six clinical scenarios.Identifying a patient in Hypertensive Failure among the six different scenarios can help an Anesthetist to avoid a Near Miss Situation.

Common clinical scenarios in Severe Preeclampsia:

Once a patient is received in casualty with clinical signs of pre eclampsia, she is likely to be administered tablet labetalol 100mg

and 20 mg of Nifedipine. This patient can run into SIX different clinical scenarios after the administration of antihypertensives.

I. Responders :

Patient's blood pressure may reach normal levels in 1 or 2 hours. If she is in early stages of pregnancy, it can be allowed to continue with anti hypertensive drugs.

II. Non-Responders:

Even after 4-5 hours, if BP continues to be high, the patient may require intravenous anti hypertensives like i.v.nitroglycerin, or i.v labetolol .or i.v.hydralzine to gain control over BP.

A non responder can be given general or regional anaesthesia depending upon the clinical protocol of the institution.

If the hospital protocol is General Anesthesia,the Anesthetist should be aware of the hypertensive crisis that can occur with intubation & extubation. It can be managed with inj.Esmolol,inj Labetalol, inj.xylocard 1.5mg/kg or inj.NTG infusion.

If Regional anaesthesia is the hospital protocol, Start 2 i.v. infusions in different sites. RingerLactate in one arm & gelatin or starch solution with 30mg of ephedrine or Phenylephrine in the other IV infusion. In case of hypotension , titrate the vasopressor infusion till the time BP becomes stable. Always look for saturation & u/o since pt can develop pulmonary edema even with normal i.v fluids. If pt develops pulmonary edema, stop all i.v fluids, give inj.morphine 4mg i.v with antiemetics, inj.lasix 40-60mg i.v till u/o

comes around 750-1000ml. Incase of established pulmonary edema, inj.dopamine has to be given in syringe infusion pump. If u/o comes around 1000ml, 30ml/hr of IVF can be started & it can be increased to 50ml/hr after 1500ml of u/o & 100ml/hr after 2000ml of u/o.

III. Three Hypotensive Scenarios:

A) 1st hypotensive situation or Over-Vasodilatation:

The patient after transfer to ward may show low Blood Pressure like 90/60 mm of Hg. Patient is comfortable, not orthopnoeic, not tachypnoeic, not dyspnoeic. Oxygen saturation will be greater than 95%. This patient is in a state of over-vasodilatation. Infusion of 1 litre of Normal Saline or Ringer's Lactate over 2-3 hours can bring back BP to Normal levels and pregnancy may be allowed to continue.Even during this slow infusion of crystalloids, watch for falling saturation,increase in the respiratory rate which may be the earliest sign of pulmonary edema.

B) 2nd Hypotensive situation or Hypertensive Failure:

If patient reports to casualty with tachycardia, tachypnoea, orthopnoea, dyspnoea, and saturation < 90% and if urine shows proteinuria and even if BP is within normal range, consider the patient to be in hypertensive failure. What was 240/120 or 200/120 mm of Hg, due to Left Ventricular failure has reached 120/80 or 130/70 mm of Hg. This is not normal BP.If the urine analysis shows 2+/3+proteinuria & even if the BP is 140/90 or higher end of normal range,consider the patient to be in hypertensive failure . We should rule out organic heart pathology, anaemia with congestive cardiac failure or pulmonary

conditions like bronchial asthma, community acquired pneumonia, pleural effusion or pneumothorax before making the diagnosis of hypertensive failure in a dyspnoeic parturient .

Treatment involves propped up position, Oxygenation, Frusemide 40-100 mg i.v, inj.dopamine or dobutamine 5-10mcg/kg/mt. Once diuresis starts (u/o of 1000ml) and failure comes under control, BP is likely to go back to original levels of 240/120 or 200/120. Oxygen Saturation may climb back to 98-100%. At this stage patient may require concomitant administration of Nitroglycerine infusion to gain control over BP. If the patient can lie flat andif there is no tachycardia or tachypnoea and breathing room air SPO_2 is > 95%, she is said to be in a clinically stable condition. If saturation does not improve NIV with CPAP mask has to be initiated. If still saturation remains low the pt has to be intubated and ventilated. These type of patients show marginal improvement with inj. Hydrocortisone or Deriphylline administrated by obstetrician in the casualty / labour ward. If the obstetrician insists on an emergency LSCS in these patient(Normal BP but dyspnoeic /urine protein2+/3+)always think of hypertensive cardiac failure.

C) 3rd Hypotensive situation or Abruptio Placentae:

Patient who comes to casualty with mild pallor, minimal hypotension, and abdominal tenderness on palpation is most likely to be in a state of an ongoing placental abruption. One should anticipate coagulation problems in this patient. If coagulation profile is normal, regional anaesthesia can be given. If it is abnormal, General

Anaesthesia with Ketamine as Induction agent is the anaesthetic technique of choice.

IV. Last or Sixth clinical scenario:

If patient shows any one of the lateralizing signs like unequal pupils, weakness of one limb or hemipariesis, she is likely to be having an ongoing Intra Cerebral Haemorrage.

In these pts, General Anaesthesia is the anaesthetic technique of choice if the obstretician wants to take up the patient for LSCS. Sub arachnoid block can precipitate medullary coning due to increased intra cranial pressure.

Transfusion protocol for anaemic patients parturients in congestive cardiac failure:

The pathophysiology of anaemia complicating pregnancy is well known to all medical practitioners. But there is no protocol or guidelines for blood transfusion in an anaemic patients in congestive cardiac failure. Injudious transfusion in CCF has precipitated pulmonary edema and death of several parturients. So following are the transfusion guidelines in anaemia with co-existing congestive cardiac failure.

1) Propped up position.
2) Oxygen through facemask.
3) Inj.Frusemide 40 -100 mg I.V.
4) Whatever may be the degree of anaemia, blood transfusion should not be given in the presence of congestive cardiac failure.

5) If the urine output is more than 1000ml, transfuse 1 unit of packed red blood cells.
6) Transfuse second unit of PRBCs, when the urine output is more than 1500 ml.
7) If the urine output is not adequate, start Inj.Dopamine infusion at the rate of 2-3 mcg/kg in syringe infusion pump.
8) If the urine output is not adequate after these measures, intubate & ventilate the patient.

If the urine output is not satisfactory even after the correction of hypoxia, consider hemodialysis. During hemodialysis excess of volume can be taken out & packed cells transfusion can also be given.

All the above mentioned factors if well understood would prevent anesthetic mishaps in perioperative period.

Reference:

1. Moise Jr KJ, Cotton DB. The use of colloid osmotic pressurein pregnancy. Clin Perinatol 1986; 13: 827-842.
2. Sibai BM. Mabie WC. Hemodvnamics of nre-eclamusia. ClinPerinatol1991; 18: 7217747
3. Barton JR, Sibai BM. Acute life-threatening emergencies inpre-eclampsia-eclampsia. Clin Obstet Gynecol 1992; 35;402-413
4. Rout CC, Akoojee SS, Rocke DA, Gouws E. Rapidadministration of crystalloid preload does not

decrease theincidence of hypotension after spinal anaesthesia for electivecaesarean section. BJA 1992; 68: 394397.

5. Rout CC, Akoojee SS, Rocke DA, Gouws E. Rapidadministration of crystalloid preload does not decrease theincidence of hypotension after spinal anaesthesia for electivecaesarean section. BJA 1992; 68: 394397.
6. Jackson R, Reid JA, Thorburn J. Volume preloading is notessential to prevent spinal induced hypotension at caesareansection. BJA 1995; 75: 262-265
7. Birnbach DJ, Datta S. Intravenous fluid preload in theprevention of spinal block induced hypotension in parturients.Anesthesiology 1993; 79: 262-269
8. Park GE, Hauch ME, Curlin F, Datta S, Bader AM. Theeffects of varying volumes of crystalloid administration beforecesarean delivery on maternal hemodynamics and colloidoncotic pressure. Anesth Analg 1996; 83: 299-303
9. Chadwick HS, Easterling T. Anesthetic concerns in thepatient with pre-eclampsia. Semin Perinatol 1991; 15:397409.
10. MacLennan FM. Lung water in pregnancy and the earlypuerperium. MD thesis Aberdeen University, Scotland, 198

11. Brown MA, Gallery ED. Volume homeostasis in normalpregnancy and pre-eclampsia: physiology and clinical
12. Kirshon B, Cotton DB. Invasive hemodynamic monitoring inthe obstetric patient. Clin Obstet Gynecol 1987; 30: 5799590
13. Anthony J, Coetzee EJ, Dommisse J, Smith P Pre-eclampsiaand CVPs. S Afr Med J 1996; 86: 273
14. Pulmonary Artery Catheter Consensus ConferenceParticipants. Pulmonary artery catheter consensusconference: consensus statement. Crit Care Med 1997; 25:910-925
15. BendettiT,Kates R,Williams. Hemodynamic observations in severe Preeclampsia complicated by pulmonary edema. Am J Obstet Gynecol 1985;152: 330-4.
16. James Brown Anaesthesia for Caesarean section.ATOTW 85 May 2008