

**Snippets from the Quarterly CRMD meeting held on 13/10/2024 at SAT Hospital, Tiruvananthapuram.**

23 cases of maternal deaths were discussed maintaining absolute anonymity. The causes are given in the following table.

It is disheartening to see that maternal deaths due to Placenta accreta spectrum are still taking toll. Timely resuscitation, immediate delivery with correction of DIC in AFE, Relaparotomy if required in Post Caesarean PPH etc at the same centre can save life. Valuable time is lost in referral to another centre. All Obstetricians and Nurses should be trained in resuscitation. Sepsis should be prevented by infection control measures. A handful of cases had a long hospital stay where better work up could have been done for a better outcome.

<b>Cause</b>	<b>Number</b>
<b>PPH (Placenta accreta =2)</b>	<b>5</b>
Sepsis	4
AFE	3
Hypertensive disease (Eclampsia +ICH – 1, Eclampsia – 1)	2
Hypokalemic periodic paralysis	1

Meningitis	1
Ischemic Hepatitis	1
Wernicke's encephalopathy	1
Acute pulmonary edema	1
Subarachnoid hemorrhage	1
Anaesthesia complications	1
Rheumatic heart disease- MS, MR	1
Septic abortion	1

We analysed each of these deaths and subcategorised into the following :

<b>TYPE OF DELAY</b>	<b>NUMBER</b>
1.Delay from the part of patient and family	2
2.Delay in reaching the institution	0
3.Delay after reaching the institution	12
4.No delay identified	5
5.Cannot comment	4

<b>AVOIDABLE OR NOT</b>	<b>NUMBER</b>
1.Unavoidable	4
2.Avoidable in an average medical setting	9
3.Avoidable only in the best settings	10
4.Cannot comment	0

### **Observations 1 and 2 :**

1) A 29 year old Gravida 3 Para 2 Live 2 Previous 2 CS, LSCS done for Placenta Previa in a private hospital. On the table, profuse bleeding after placental removal. Diagnosed placenta accreta. Patient developed hypovolemic shock. Referred to a higher centre. On arrival there PR 106/mt. BP 90/60 on Noradrenaline.

Immediately shifted to OT. TAH with bladder injury repair done. Adherent bits of Placenta in the lower segment. Post operatively on ventilatory support and

inotropes; shifted to ICU. ABG showed metabolic acidosis. Massive transfusion initiated. 12 units PRBC. 26 Units Platelets, 18units FFP and 4 units

Cryoprecipitate given. Patient developed AKI. Hemodialysis done. Extubated the next day. Patient became hemodynamically stable. Oral fluids started. But

LFT & RFT values showed worsening. On POD 3, she desaturated, developed ARDS and cardiac arrest while undergoing hemodialysis. CPR initiated. Revived, but cardiac arrest again on POD 6 and expired.

2) A 39 year old G2P1L1 at 33 weeks Previous CS. diagnosed with Placenta Previa. Admitted with bleeding at 33 weeks in a private hospital, She developed pain and Emergency CS done. While removing Placenta, Placenta adherent to lower segment and bladder. Continuous ooze from suture site. Managed with blood

products, vasopressors and uterotonics. Patient had vaginal bleeding post op. Vaginal bleed ligated and packed. Patient went into hemorrhagic shock.

Relaparotomy done. Hysterectomy with bladder tear sutured. Hemostasis attained. Had massive transfusion. LFT deranged severe Lactic acidosis. Patient

regained consciousness, developed AKI Ischemic hepatitis and later on went on to sepsis. She was referred to higher centre on POD 4 at 2.53 AM. She

developed GTCS AT 5.00 AM. CT diffuse cerebral oedema with impending herniation. Condition worsened and had cardiac arrest at 2.20 PM, Death declared

at 2.30 PM

**Cause of death in both the cases :** PPH Placenta Accreta, Hemorrhagic shock, MODS.

**Recommendations :** Even the second centre did not use Aorta clamp which is undoubtedly the first aid measure in such cases. TVUAC can safely be used on the actively bleeding veins of the bladder base to control bleeding. This patient had actually improved and then worsened and expired. 6 units of platelets and FFP were transfused on POD 3 which might have led to TRALI and sudden deterioration.

Suspect Placenta accreta spectrum in all cases of anterior placenta previa and try to rule out accreta. An MRI can support the diagnosis if suspected by US. But it is not always needed. All Previous Caesarean patients with anterior and central Placenta previa have to be referred to higher centre even if no accreta in USS because bleeding after placental delivery can be torrential and unmanageable. Even in posterior placenta previa troublesome bleeding from the placental bed should be expected. *If Placenta accreta spectrum is diagnosed on the table by a vascular lower segment or percreta Do not hesitate to close the abdomen and Refer* . Only a centre with experienced Obstetricians , Anaesthesiologists , Urologists and Blood Bank should take up such cases .

**Observation 3:** A 33 year old G3 P1L1 , No comorbidities. Previous CS 12 years back. Elective LSCS from private hospital at 38 weeks as she got admitted with neck

pain and numbness of left upper Limb. Male baby delivered at 9.01 am grade 2 MSAF. At 2 pm on the same day desaturated. Shifted to ICU. Resuscitated,

intubated, put on inotropes and referred to MCH. Patient reached the ED in cardiac arrest at 4.15 PM. Rhythm was pulseless electrical activity. Resuscitated

as per ACLS Protocol. After 4 mts ROSC attained. Patient was in severe metabolic acidosis and lactic acidosis. MTP activated. Patient had repeated cardiac

arrests and expired at 6.57 PM

**Cause of death :** Post LSCS ,PPH, DIC

**Recommendations:** Though the reporting doctor put the cause as AFE, this was a clear cut case of PPH as her Hb was 2.8 g%, INR > 180 and APTT > 180 . She was referred as a case of PPCM as Echo had shown global hypokinesia which might have been only stress cardiomyopathy. Relaparotomy in the first centre would have saved precious time and blood loss and might have saved the patient. In such cases cardiac global hypokinesia is not the cause but the result of the insult.

**Observation 4:** A 26 year old Primi IVF ICSI Conception. DCDA twins reduced to singleton at 12 weeks in view of omphalocele. Amniocentesis at 15 weeks. Admitted with fever of acute onset with chills and rigor at 24 weeks gestation. Temperature 105 degree. USG showed IUD. Started on IV antibiotics, leaking at 8.20 pm shifted to

Labor room. PR 130/mt , BP 70/40. 2units Normal saline rushed. CRP 128. Antibiotics stepped up along with supportive measures. Next day at 12.50 am

patient c/o abdominal pain, sudden collapse at 4.30 am. In spite of aggressive resuscitative efforts, Patient expired at 5.08 am

**Cause of death :** Sepsis

**Recommendations:** Here surviving sepsis campaign guidelines were not adhered to: aggressive fluid management , Inotropes, Cultures etc. She should have been managed in ICU as she was hemodynamically unstable.

**Observation 5 :** A 28 year old G2 P1L1 Prev. LSCS GDM on Insulin , Emergency CS with sterilization at 7.06 am for previous CS in labour, 4 kg baby. On 4th day had fever and oozing from wound. Culture showed Staph aureus and was started on Piptaz . On the 9th day patient was referred to MCH with c/o abdominal distension,

fever and breathing difficulty. On arrival vitals stable. Abdomen distended, Abdominal wall edema present. Wound clean, Lochia Healthy, USG showed mild to

moderate ascites with internal echoes 3.6 cm in thickness with septations anterior to uterus. Bilateral pleural effusion. Patient had fever and loose stools. CECT

on 11<sup>th</sup> day showed loculated ascites. Consulted surgeon. Emergency Laparotomy done next day. Drainage of Pus, Peritoneal lavage and Lt Salpingectomy done.

Patient continued to have fever spikes. Tracheostomy done. Meropenam and Vancomycin added. Condition deteriorated and expired on POD 17.

**Cause of death :** Post CS Sepsis, Multi organ Failure

**Recommendations :** SSI ( surgical site infections) is caused by flaws in intra operative factors like atmospheric temperature, number of air circulations in the theatre, skin preparation with Povidone iodine and Chlorhexidine , Prophylactic Antibiotic within one hour ,vaginal cleansing with Povidone iodine , meticulous surgical techniques etc. Peri operative glycemic control, proper inspection and palpation of the wound ,probing the wound in the presence of induration and letting out collection , early laparotomy etc might have made a change in the final outcome .Sepsis can lead to Multiorgan failure. If liver enzymes  $>10000$  it is due to ischaemic hepatitis.

**Observation 6:** A 40 year old Gravida 2 Para 1 Live 1 at 32+5 weeks Prev LSCS for Hypertension 10 years at 36 weeks. Patient was on Aspirin. Was on Labetalol for 2 months. Dose increased with BP 160/100. Patient c/o severe tiredness, waited till evening to attend a function at home. On the way to hospital, she had convulsions. She was declared dead on arrival .



**Cause of death:** Eclampsia

**Recommendations:** Though prima facie it looks like delay on part of the patient, she was at home with Tab Labetalol 100 mg tid. A woman taking such a high dose should have been admitted in hospital rather than at home. She should have been given Steroids and delivered early.

**Observation 7:** A 44 year old G3P1L1 at 29 weeks first FTND 21 years died in RTA. 1 abortion. Conceived with IVF. DCDA twins, overt DM, hypothyroid, admitted with high BP, urine PC ratio 10. Started on Labetalol. Developed impending symptoms on day 5 of admission, Magsulph given. LSCS done at 1.20 pm. At 5.35 pm patient had 1 episode of GTCS. Magsulph infusion and Lorazepam 4mg given. Labetalol 20 mg IV given twice and referred to MCH. She had convulsions in the ambulance. BP 220/90. On admission labetalol IV given and BP controlled. But patient had desaturated by then. Intubated and managed by a multi-disciplinary team. CT could not be done. On day 2 CT showed large intraparenchymal hematoma 5.8\*3.8\*3.2cm. Left frontal lobe with subarachnoid and intraventricular extension, mass effect and midline shift. Antiepileptics and supportive measures continued. On day 5 of admission to the higher centre she had cardiac arrest and succumbed.

**Cause of death:** Eclampsia, Massive intracerebral hemorrhage

**Recommendations:** The interventions that could have changed the outcome are : she should have been on low dose aspirin starting from 12 weeks ;since hypertension was diagnosed and urinary PCR was 10 she should have been delivered at 29 weeks itself after giving neuroprotective dose of magnesium sulphate and steroids; in case of IV Labetalol the dose can be increased to 40 mg, then 80 mg then another 80 mg to control the hypertension ; a second antihypertensive should have been added by this time. It has to be remembered that intracerebral hemorrhage is the killer in most of the cases of hypertension complicating pregnancy.

**Observation 8:** A 32 year old G4P3L3 at 39 +3 weeks , 3 FTND LCB. 3 years admitted with mild pain. CX 50 % effaced Os admits 2F. Induced with Pg E1 25 ugm orally at 11.30 am and 6.00 pm. Membrane ruptured spontaneously at 4.30 am and immediately developed seizures. Resuscitated, intubated. Resuscitative hysterotomy done, baby asphyxiated. Patient referred to a higher centre. Cardiac arrest from there and expired at 6.18 pm.

**Cause of death:** AFE

**Recommendations:** This multipara was already in early labour when she was admitted . Would not it have been more prudent to leave her alone so that she would have got into spontaneous labour ? Multiple PGE1 and Rupture of membranes in a Multi would have triggered off AFE. Timely intervention and Resuscitative hysterotomy are appreciated.

## **Message to KFOG Members in the light of CRMD Meetings**

There was one maternal death due to LAST : Local anaesthetic systemic toxicity. Patient had recurrent seizures soon after spinal anaesthesia. 20% lipid emulsion is the antidote if toxicity is suspected.

Rheumatic heart disease though a rare entity in Kerala now, still exists . Such patients should be treated only in tertiary centres. Diligent history taking and clinical examination at first visit should not become a forgotten art.

Oral PGE1 is the drug of choice for IOL and not vaginal PGE1 as vaginal PGE1 increases the risk of cervical and vaginal lacerations.

Trans vaginal/abdominal uterine artery clamp and suction cannula are the **FIRST AID** measures in PPH along with resuscitative measures and drugs.

Those institutions with facility of ROTEM and TEG should utilise it in the management of severe PPH.

After Caesarean section make sure the os is open, and remove all the clots in the uterine cavity during vaginal toileting.

Rare infections like Meliodosis in our ICUs should raise the alarm of hospital acquired infections

The order is ARM followed by Oxytocin after one hour sos and not Oxytocin before ARM.

We had one death due to Wernicke's encephalopathy ; Thiamine has to be started early in Hyperemesis .IV Dextrose is harmful in hyperemesis as it utilises the available B1 vitamin in the body and pushes the patient to encephalopathy

To make our Confidential enquiry more effective, We would request each district MDNMSR team to report all maternal deaths of the district to CRMD team along with a copy of the case records. Facility based audit is regularly taking place in the hospitals involved ; a KFOG member is invariably a part of the Audit team. It will be greatly appreciated if the KFOG member participating in the audit could forward a case sheet copy to our team. Revised forms A and B are put up in the KFOG website ; they are to be filled up and sent to CRMD team in the eventuality of a maternal death along with anonymised case sheet. It is strictly a 'NO BLAME' game. We thank you for your continuing support.