Double emergency during elective lower uterine segment Caesarean section

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We present a case of respiratory compromise after induction of anaesthesia for lower segment Caesarean Section (LSCS) and a concurrent major haemorrhage. Clinical features of pulmonary oedema and a high urine protein creatinine ratio (PCR) suggest a diagnosis of atypical late-onset pre-eclampsia (PET) as the primary aetiology.

30 year old P1 for code 4 LSCS at 39/40

High Body Mass Index (43)
Recent 'cold'
Previous forceps delivery in theatre and shoulder dystocia – ***requests LSCS under general anaesthetic (GA) due to traumatic experience***
Airway; Mallampati 3, reasonable mouth opening, single tooth top denture, good neck range of motion

Surgical Events

Unanticipated breech presentation
Rapid blood loss on uterine incision followed by atony
BP stable but HR 110 →140
Call to Intensive Care Unit (ICU) for senior help, call to theatre coordinator for additional Operating Department Practitioner (ODP) support. Obs & Gynae consultant called

Massive Obstetric Haemorrhage (MOH) management

Proceeded as per OBS Cymru[1] (Obstetric Bleeding Strategy care bundle)
MOH alert - 2 units packed red cells (RBC) urgently requested
Initial ROTEM: FibTem A5 22, Extem CT 44, repeat also within normal limits
Venous Blood Gas: pH 7.29, lac 1.7
Arterial line; arterial gas: pH 7.26, hb 108, PaO_2 9.58, SpO_2 91%.
Fluids: Hartmans 1000ml, Gelspan 1500ml, RBC 4 units, Cell Salvage 874ml
Syntometrine x 2, TXA x 2, Haemobate x 4, misoprostol PR
Maternal blood loss 4.1 L

Planning for challenges

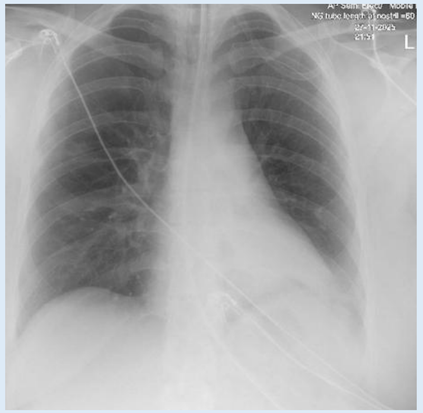
- ✓ Awake catheter
- ✓ GA; Ramped position, Nasal high-flow oxygen ($NHFO_2$), Rapid Sequence Induction (RSI), Videolaryngoscopy (C-MAC)
- ✓ Tranexamic Acid (TXA) 1g on incision
- ✓ Syntometrine and oxytocin infusion
- ✓ Cell salvage
- ✓ Paediatric doctor present

Meanwhile at the top end…ongoing hypoxia SpO_2 80%

STOP AND THINK…what are the differentials?
?Bronchospasm
Pulmonary oedema; cardiogenic ?cardiomyopathy (pregnancy, post-viral) vs non-cardiogenic (?PET)
? Drug reaction or anaphylaxis
?? Pulmonary embolism
??? Amniotic fluid embolus
Furosemide 50mg IV + 50mg IV, magnesium 5g IV
Slow improvement to SpO_2 95-97% although high FiO_2 requirement
→ transferred intubated and ventilated to ICU

ICU Management

Respiratory; Persistent O_2 requirement FiO2 0.7-1.0, bilateral crackles on auscultation
Chest Xray; nil focal, generalised fluffiness
Further magnesium IV
Circulatory; on metaraminol. Tachycardic 110-120,
Fluid restriction implemented
Further furosemide IV - good diuresis
Mast Cell Tryptase not elevated
ProBNP 48
Transthoracic Echocardiogram normal
Urine PCR 129



Anaesthetic

$NHFO_2$ and preoxygenation with mask to FiO_2 0.76
Drugs: Thiopentone 500mg, Suxamethonium 200mg
C-MAC - poor view, secretions ++, cricoid removed, POGO 25%, bougie and ETT passed, 22cm at lips…starting to desaturate
Bagged FiO_2 1.0/sevoflurane - slow $EtCO_2$. Desaturation continued, high airway pressures. ***$Sp0_2$ 50% - MAC adequate, surgeons told to expedite delivery due to maternal hypoxia***
AmbuScope; ETT position ok but frothy pink sputum in airways

Outcome and Discussion

Extubated uneventfully the next day
Uses Ventolin inhaler for 'asthma', although not recorded in records

- Atypical PET +/- negative pressure pulmonary oedema
- Bronchospasm from heightened airway reactivity (vapes, recent infection) +/- asthma, +/- drug reaction
- Unfortunate concurrent MOH but ?Exacerbated by GA ?hurried surgery and surprise breech with large baby + impact of PET
- No documented hypertension – acutely masked by GA, hypovolaemia and ongoing sedation? Brief hypertensive response to laryngoscopy - ?significance

Would spinal anaesthesia have been preferable? It would have avoided hypertensive response to laryngoscopy, and bronchospasm, and potentially less bleeding (maybe even avoid MOH?) **BUT** very high chance of intra-op conversion to GA which would have carried additional risk of airway difficulty.

Late onset PET can present unexpectedly with severe cases carrying the risk of pulmonary oedema secondary to endothelial dysfunction[2] - although it is rarely the presenting feature. PET is a common cause of non-cardiogenic pulmonary oedema in the perinatal period[3] so maintaining high vigilance and index of suspicion to ensure prompt and appropriate management is vital.

1. www. phw.nhs.wales/services-and-teams/improvement-cymru/our-work1/maternity-cymru/obs-cymru/
2. Masini G, Foo FL, et al. Preeclampsia has two phenotypes which require different treatment strategies. Am J Obstet Gynecol 2022; S1006-S1017.
3. Glass DM, Zehrer T, Al-Khafaji A. Respiratory Diseases of Pregnancy. Evidence-Based Critical Care. 2019 Jul 24:743–747.