The Unsuspected Massive Postpartum Haemorrhage requiring Peripartum Hysterectomy: A Regional Approach

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Background

Massive postpartum haemorrhage (PPH) requiring peripartum hysterectomy is an uncommon obstetric complication in high income countries and is more commonly associated with cases of placental adhesive disorder[1]. This case aims to examine the chain of events leading to peripartum hysterectomy and management options in a regional setting.

The Case

CK, a 31-year-old G3 now P3 woman underwent an elective repeat caesarean at 38+1 weeks in the setting of mild obstetric cholestasis and 2 previous caesareans. There was no history of PPH and no suspicion of placental adhesive disorder on both ultrasound antenatally and placental histopathology.

At elective caesarean she was noted to have a poorly developed and vascular lower segment. She initially sustained a 1010ml PPH due to trauma and was managed with suturing but became symptomatic of anaemia in recovery and received 1 unit of blood.

1 hour later, she was reviewed for a further 362ml loss secondary to tone. She was given ergometrine and carboprost with temporary resolution. 50 minutes later, she was reviewed for a further 294ml loss so she was transferred to theatre. Further uterotonics were given and an examination under anaesthesia revealed an atonic uterus with ~2L of blood and large clots (3.7L to date). No retained products were felt and the hysterotomy sutures were intact.

A Bakri balloon was inserted with further ~200ml of blood immediately removed and clinical coagulopathy was first noted. The massive transfusion protocol was activated and the decision for peripartum hysterectomy was made with a 6.5L PPH at completion (1.6 times her blood volume by Nadler's formula). In total she received 10 units of red blood cells (PRBC), 2 units of platelets, 20 units of cryoprecipitate and 3 units 20% albumin.

Her post-operative course was complicated by asymptomatic sinus bradycardia and Ogilvie's syndrome. She was debriefed 4 weeks postpartum and developed features of perinatal anxiety with flashbacks but declined ongoing psychological care. She ceased breastfeeding 3 weeks postpartum.

Discussion

This case highlights the challenges in management of massive PPH and its complications in a regional setting.

Whilst PRBCs and albumin are relatively easy to source, reduced demand means cryoprecipitate and plasma are stored frozen and a minimal supply of platelets are kept. The unpredictable nature of massive PPH and the potential need for large quantities of blood products therefore present a barrier to timely transfusion in a regional setting.

CK's post-operative course was complicated by new, transient bradycardia (nadir = 36bpm) and hypotension (nadir 90/50). Whilst a troponin and lactate were never done and CK was asymptomatic, these signs may be a prodrome for cardiogenic shock. It is hypothesized that the cause for these signs was a cardioprotective response to exsanguination, as her heart rate normalized with exercise day 5 postpartum.

While there is a paucity of high-quality evidence on breastfeeding post massive PPH, one study has suggested that breastfeeding initiation rates are more influenced by the delay to first breastfeed after birth, with no improvement in breastfeeding initiation or continuation with blood transfusion[2].

Conclusion

Whilst an uncommon obstetric complication, this case highlights how patients with minimal risk factors can go on to develop massive PPH requiring hysterectomy. While there are challenges in a regional setting to facilitating appropriate care, overall, this case was excellently managed.

References

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