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CASE REPORT ON ANAESTHETIC MANAGEMENT OF TRAUMATIC POST PARTUM HAEMORRHAGE WITH SHOCK AFTER VBAC

Anaesthesiology

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ABSTRACT

Obstetric haemorrhage accounts for 25% of maternal deaths in developing countries and post-partum haemorrhage (PPH) is the most common type.The causes of PPH are 4T's-tone accounts for 70% of PPH cases (post-delivery poor uterine contraction),tissue(blood clots and/or retained products of conception),trauma(genital tract),thrombin(coagulation abnormalities).Clinicians should be aware of appropriate surgical measures and the timing of interventions.Trauma accounts for 15%–20% of cases and is mostly attributed to perineal,vaginal or cervical lacerations,perineal hematomas,episiotomies or uterine rupture.Failure to assess the clinical picture, underestimating blood loss,delayed treatment,lack of multidisciplinary team work and failure to seek timely senior help will lead to maternal mortality.Effective team work,pooling of resources,the presence of a 'rapid PPH response team' with timely and aggressively managed cases can improve outcome and stand with minimum mortality and morbidity.

KEYWORDS

VBAC,4T'S,Fourth degree Perineal tear, Hypovolemic shock

CASE REPORT

A 30Year old Gravid-5 female patient of 38.2 weeks POG, with previous Lscs weighing about 79kgs admitted for safe confinement by VBAC(vaginal birth after ceaserean section).Patient doesn't have any comorbidities,with Hb:11.2g/dl,TLC-8780,Plateletcount-1.7L/cumm,PT/INR13/0.96,B+ve blood group,delivered a live term male baby at 3.30pm about 3.3kgs normally through outlet forceps delivery.Immediately after delivery Inj.oxytocin 10units IV and IM were given,placenta and membranes were expelled out intoto, profuse bleeding was seen through vagina, unable to find out as patient was irritable and not supportive for per vaginal examination. Call attended from labour room for PPH resuscitation, reached in a minute, assessed patients general condition(patient was conscious, irritable,c/o pain and pallor++seen)haemodynamics(BP-90/40mmhg, HR:140BPM, SPO2:90-94% at room air) and blood loss which was around 2 litres (fully soaked blood mops were around 25 in number),Immediately secured another 18G cannula,started 6litres of oxygen though face mask,Inj Midazolam 2mg Iv,Inj Tranexamic acid 1gm Iv,Ringer's Lactate and colloid were started,requested to issue 2pint PRBC, reserve 2pint PRBC and 4pint FFP, made patient comfortable and allowed obstetrician for per vaginal examination, then found out vaginal,cervical and grade 4 perineal tear.Obstetrician sutured cervical tear, but bleeding was continued,Considering the emergency and critical condition of patient, she was taken to operation theatre for exploration after vaginal packing is done to reduce further bleeding while shifting to OT and was accepted under ASA GradeV Emergency with High risk consent after explaining risks and complications by audiovideo counselling. Blood samples(CBP & cross matching) were sent to lab.Pallor +++,peripheral pulses were feeble,carotids were feeble,HR:150/min,BP-80/36mmhg,Air entry was bilaterally equal, no added sounds and S1S2- were heard.Patient was monitored with pulse rate, NIBP, ECG,SPO2 and urine output, ventilated with 100% O2, direct laryngoscopy was done, patient was induced by RSI with Inj Midazolam 1mg iv, Inj fentanyl 100mcg Iv and Inj Atracurium 40mg iv, intubated with 7.0cuffed OETT,confirmed bilateral air entry equal and secured ET at No 20,exploration started simultaneously, maintained with sevoflurane:o2:air-0.5%:2:2,Inj Atracurium maintanence dose of 10mg in 30min interval,PR-150/min, BP-70/30mmHg on NIBP, peripheral pulses, carotids were feeble.Her investigations:Hb- 6.1gm%, PCV-18.5, TLC- 18840, PLT- 1.00, PT-20.81, INR- 1.80. As blood products were not arrived, managed with crystalloids and colloids.Cervicovaginal-perineal Exploration was done and repair was going on, during the procedure blood loss was around 1500ml to 2 litres. Urine output was nill,carotid and radial artery pulses were not palpable, pallor-papery white and BP was not recordable, ECG shows normal sinus rhythm,immediately started Inj.Noradrenaline 4mg in 50ml DNS at the rate of 0.05 mcg/kg/min infusion and 1 pint PRBC.Sent ABG,CBP,RFT, coagulation profile,serum electrolytes. Her vitals were improved with ionotrope support and blood products intraoperatively with HR 130- 140/min and BP was 90-95/60 mm of Hg.

During Intra Operative period, patient received -2pint PRBC,2 pint FFP,4pint RL,1pint NS,1pint Haemaccel,Inj.Noradrenaline infusion @ 20 mcg/min was continued in drip.Antibiotics & Inj Calcium Gluconate infusion were given. After about an hour HR settled to 120/min,BP:-110/70(on ionotropic support).Urine output was 50ml.Inj Furosemide 10mg was given to prevent fluid overload. Exploration and repair was done in 3:30hrs and patient was extubated smoothly with reversal Inj Neostigmine 2.5mg+glycopyrrolate 0.5mg.Patient was conscious, respiration was regular and adequate. Haemodynamically stable and Total urine output by the end of surgery was 750ml.Patient was shifted to SICU for further treatment and management as ionotropic support was still onflow. Her ABG report was as follows-pH:-7.38,pCO2:-33,HCO3:-19.5,Hct:30,BE:5.2, PO2:54.SerumCalcium:8.06,Na/k/cl132/4.1/96.Ionotropic support was gradually reduced and then stopped.Antibiotics were escalated.During her stay in hospital she received total 4pint PCV, 4pint FFP,1gm FCM(Ferric Carboxy Maltose).She never had fever and her wound was healthy.Her subsequent recovery was uneventful.She was shifted to ward on 4th postoperative day and was discharged home on 6th postoperative day.

DISCUSSION

Post-partum haemorrhage (PPH) refers to an estimated blood loss in excess of 500 ml following a vaginal birth and a loss greater than 1000 ml during a Caesarean deliveries, occurring within the first 24 h of delivery[1].Major haemorrhage is defined as an estimated blood loss of more than 2500 ml or the transfusion of 5 or more units of blood or treatment of coagulopathy[2].Patients with a low body mass index have a lower blood volume of 70 ml/kg and anaemic women have fewer reserves to withstand blood loss and hence will decompensate sooner.Thus, a useful definition takes into account any blood loss that causes a major physiological change like a fall in blood pressure, as the risk of dying from PPH depends on the amount and rate of blood loss and the woman's health[2].

Pathophysiology: The blood vessels supplying the placental bed pass through an interlacing network of muscle fibres of the myometrium. Myometrial contraction causes placental separation and causes blood vessels to constrict[3].This haemostatic mechanism is known as the 'physiological sutures' or 'living ligatures' control the bleeding from the placental bed when the placenta separates.The physiological increase in clotting factors during labour helps to control blood loss after separation of the placenta[3].Uterine atony results in a failure of these 'living ligatures' to stop the bleeding.The active management of the third stage of labour is associated with a reduction in mortality and morbidity[3].

Fourth Degree perineal tear is Tearing of the anal sphincter complex and the rectal mucosa.Severe perineal lacerations,which include third- and fourth-degree lacerations,are referred to as obstetric anal sphincter injuries (OASIS)[14]

Fourth-degree tears usually need to be repaired in an operating room rather than in the delivery room. Indeed, moving the patient to the OT at the appropriate time forms an important part of modern management of PPH. Proper repair of third- and fourth-degree lacerations is more complex; clinical expertise and correct technique are essential to improve patient outcomes[14]

Rapid sequence induction needs to be performed while induction of General Anaesthesia to prevent the risk of aspiration of gastric contents as parturients are considered to have full stomach and delayed gastric emptying[13].Administration of General Anaethesia is recommended for management of PPH especially for pain relief, and when obstetric hysterectomy becomes the only viable option[13].

If the patient is hypotensive then the first step must be to restore an adequate BP. For all patients, volume should be infused intravenously and the clinician should "bridge" the patient with vasopressor (Dopamine or Norepinephrine) while volume is being infused to maintain an adequate BP, because progressive hypotension can lead to decreased coronary perfusion, further worsening cardiac function, hypotension and Myocardial ischemia[4].

Norepinephrine is an endogenous catecholamine that has potent alpha 1 and beta 1 adrenergic effects. The primary vasoactive effect of Norepinephrine is arterial and venoconstriction.The ionotropic properties of norepinephrine are usually offset by increase in afterload.Because of its marked vasoconstrictive characteristics, Norepinephrine seems the logical drug of choice in distributive form of shock,increasing MAP,effective circulating blood volume,venous return and preload, with minimal increase in heart rate or stroke volume.Norepinephrine is more potent than Dopamine and is the choice vasopressor to reverse hypotension in vasodilatory shock[5].

Despite the fear that norepinephrine could severely constrict renal microvasculature, it has been shown to improve renal function,as measured by increased medullary blood flow ,creatinine clearance and urine flow in experimental and human septic shock[6].

Decrease in circulating blood volume during severe hemorrhage can depress cardiac output and lower organ perfusion pressure to tissues as blood flow is preferentially distributed to organs with greater metabolic requirements[7]. Severe haemorrhage impairs the delivery of O2 and nutrients to the tissue and produces shock.Lowering regional vascular resistance by adenosine, prostaglandins and nitric oxide induces hypoxic redistribution of blood flow[8].Inspite of this organ specific microvasculature response,all organ with the possible exception of the heart,experiences decrease in blood during severe hypovolemia[9].

Recent trends in damage control resuscitation attempt focus on "Haemostatic Resuscitation"which pushes for early use of blood products rather than on abundance of crystalloid in order to minimize the metabolic derangement, resuscitation-induced coagulopathy and the hemodilution that occurs with crystalloid resuscitation.A recent study has shown no significant difference in mortality at 24 hrs or 30 days between ratios of 1:1:1 and 1:1:2 of plasma to platelets to packed RBS's. However patients who receive the more balanced ratio of 1:1:1 were less likely to die as a result of exsanguination in 24 hrs and were more likely to achieve hemostasis[10].

The early administration of red blood cells (RBCs) and fresh frozen plasma (FFP) is a priority to maintain arterial O2 delivery and restore an effective coagulation.The administration of RBCs is considered indispensible when the Hb level is < 7 gm/dl[11].This recommendation is based mainly on the results of the Transfusion Requirement in Critical Care (TRICC) study[12].The administration of FFP should be associated as soon as possible with RBC transfusion to compensate for the deficiency in coagulation factors.The initial recommended dose of FFP is 10-15ml/kg[11].It is recommended when PT or aPTT is 1.5 times the normal value. Platelet transfusion is recommended when PC < 50000 /L.

CONCLUSION

PPH is a major cause of maternal morbidity and mortality.Catastrophic and life-threatening haemorrhage is often unpredictable.Identification of the cause of bleeding,rapid,aggressive and prompt resuscitation of the patient with effective restoration of the circulating blood volume, should be performed in a multidisciplinary team setting can save patient's life;failing which the outcome of patient may be guarded.The key to successful outcome is teamwork with prompt action and multidisciplinary approach in managing the case of pph.

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