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ANAESTHESIA MANAGEMENT OF ACUTE PUERPERAL UTERINE INVERSION

Anesthesiology

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ABSTRACT

Although rare, uterine inversion is a potentially life threatening complication of third stage of labour. Torrential blood loss and shock disproportional to actual hemorrhage are the two basic points of diagnosis. Death may occur in 15% of the affected mothers due to pain, blood loss and shock. Timely and aggressively managed cases stand with minimum morbidity and mortality. Here we are presenting a case of acute puerperal uterine inversion with hypovolemic shock treated successfully.

KEYWORDS

Uterine inversion, hypovolemic shock, Johnson maneuver, general anesthesia.

INTRODUCTION

Uterine inversion, although rare, can be a life threatening emergency because of associated blood loss and cardiovascular instability[1]. It is associated with significant blood loss and shock which may be out of proportion to the hemorrhage.

It is well established fact that mismanagement of third stage of labor (premature traction on umbilical cord and fundal pressure) before separation of placenta is the commonest cause of acute uterine inversion[2]. There is little published information on the pathophysiology or anaesthetic management of this clinical condition. Here, we are presenting a recent case of acute puerperal uterine inversion and its successful management.

CASE REPORT:

A 30 years old gravida 5 female patient, of 38.4 weeks pregnancy, weighing 54kgs, delivered normally in the morning at the private hospital of periphery. She was referred to the tertiary care centre in view of adherent placenta and bleeding . On arrival, her general condition was poor. She was unconscious, groaning to painful stimuli. Pallor ++++, peripheral pulses were not palpable, carotids were feeble and femorals were thready. HR:150/min, unrecordable arterial pressure , S1S2- Muffled ,Air entry was bilaterally equal and no adventitious sounds were heard. Pupils were dilated bilaterally and were sluggishly reacting to light.

Her investigations were done: Hb- 3.4 gm%, PCV-9.8, TLC- 11400, DLC- 69/30/3/2, PLT- 1.05, PT- 27.81, INR- 2.32. Considering the emergency and critical condition of patient, she was taken to operation theatre for exploration and removal of placenta, and was accepted under ASA Grade V Emergency.

On table two large bore IV cannulae of 18G were taken on right and left forearm and IV Ringer's Lactate and colloid were started . On per vaginal examination it was found that the placenta was already removed but it was total inversion of placenta with profuse bleeding. She was monitored on multipara monitor with pulse rate, NIBP, ECG and SPO_2. Under all aseptic precautions, Subclavian central line was inserted. CVP was 2cm of H_2O. Inj. Nor adrenaline 8mg in 500 ml DNS was started at the rate of 0.05 mcg/kg/min. Patient was ventilated with 100% O2. On table patient was in gasping state with SPO_2 of 70%. Hence direct laryngoscopy was done, 10% LOX spray was sprayed over the vocal cords and patient was intubated with ETT no. 7.5 cuffed. Patient was maintained on 100% O_2. SPO_2 was achieved at 98%. Patient received:- 2 pint PCV, 3 pint FFP ,4 pint RL, 1 pint HES , Inj. Nor adrenaline 8mg in 500 ml DNS @ 20 mcg/min. PR-150/min, BP-80/50mmHg on NIBP, peripheral pulses were still not palpable, carotids were feeble and femorals were thready. Cervicovaginal Exploration was done by Obstetrician and Bakri balloon was inserted and monitored.

After 10 min. decision of Obstetric Hysterectomy was taken as a lifesaving procedure as the Bakri balloon came out and there was continuous bleeding. Procedure completed uneventfully within 1 hour. During Intra Operative period, patient received - Inj. Hydrocortisone 200 mg; Inj. Tranexamic acid 1gm. Inj. Nor adrenaline @ 20 mcg/min was continued in drip .Inj. Dobutamie 250 mg in 500 ml NS @ 10ml/hr was started. Her vitals were stable intraoperatively with HR 130-140/min and BP was 90-95/60 mm of Hg. Patient was further monitored in OT for another 1.5 hour, where she received, 1 pint PCV, 1 pint RDP and 2 pint FFP. Inj. Calcium Gluconate was given. Antibiotic infusion was instituted.

After about an hour HR settled to 120/min. BP:- 110/70 (on ionotropic support).Urine output was 20ml. Patient was shifted to ICU for ventilatory support with ionotropic support still on. Her ABG report was revealed on the same day in ICU as follows- pH:-7.34, pCO2:-20, HCO3:-13.4, Hct:- 30, Hb:- 10.7, TLC:-18700, PLT:- 74000

After 2 days, Patient was extubated uneventfully and was shifted to ward the next day. Her ionotropic supports were gradually reduced and then stopped.

During her stay in hospital she received total 9 pint PCV, 12pint FFP, 8pint RDP. She never had fever and her wound was healthy. Her subsequent recovery was uneventful. She was shifted to ward on 5th postoperative day and was discharged home on 11th postoperative day.

DISCUSSION:-

Puerperal uterine inversion is due to, displacement of the fundus of the uterus, usually occurring during the third stage of labor. It is classified as 1) First degree (incomplete) where the inverted fundus extends not beyond cervical ring; 2) Second degree (incomplete) where it extends the cervical ring but remains in vagina; 3) Third degree (complete) where the inverted fundus extends down to the introitus; 4) Fourth degree (complete) where the vagina is also inverted.

Pathophysiology-

It can be explained on the following 3 points as 1) A portion of uterine wall prolapses through the dilated cervix or indents forward. 2) Relaxation of part of the uterine wall. 3) Simultaneous downward traction on the fundus leading to the uterine inversion.

It is associated with placenta previa or fundal implantation, antepartum use of Mgso4, and umbilical cord traction with vigorous, fundal pressure[3].The displaced uterus while delivering the placenta, usually is in association with PPH and clinical shock (Hypotention and inadequate tissue perfusion), out of proportion to blood loss. This shock is thought to be due to parasympathetic effect of traction on the ligament supporting the uterus which may be associated with bradycardia due to neurogenic shock followed by severe hypovolumia due to blood loss causing tachycardia.

The immediate treatment of the hemorrhagic shock and reposition of the uterus play key role in the management. Resuscitation should be started immediately while attempts are made to repose the uterus manually. The chance of immediate reduction is between 22-43%[4]. If

unsuccessful, further attempts should wait till the patient is haemodynamically stable[5]. If the uterus remains inverted, contracted cervix may require relaxation by General Anaesthesia or Tocolytic therapy. Severe and resistant cases may require obstetric hysterectomy[5].

Once the diagnosis is made, uterine reposition is best done manually by Johnson maneuver, as delay can render it more difficult and increase the risk of hemorrhage. Tocolysis has a role in relaxing the uterus for manual reposition and use of the hydrostatic method. Many tocolytics have been used in acute inversion. These include: Magnesium sulphate (4-6g intravenously over 20 minutes), Nitroglycerine (100micrograms intravenously slowly, achieving uterine relaxation in 90 seconds when given sublingually) and Terbutaline (0.25mg intravenously slowly). Terbutaline and magnesium sulphate take 2 and 10 minutes, respectively, to be effective[2]. Abouleish et al.[1]recommended terbutaline as the first line treatment because of its rapid onset of action, short half-life and ease of use[2].

There are reported cases of using the Bakri balloon catheter[6] and Rusch Balloon catheter[7] to create hydrostatic pressure. These have been used in instances where the placenta is already separated. An additional advantage of this method is, after repositioning the uterus, the balloon will help to prevent re-inversion and reduce PPH.

When manual reduction in labour room fails or seems difficult, it is necessary to transfer the patient to the OT. Indeed, moving the patient to the OT at the appropriate time forms an important part of modern management of PPH. Administration of GA is recommended for management of uterine inversion[1]especially for pain relief and possible relaxation of uterine muscle, aiding reposition failing which, obstetric hysterectomy becomes the only viable option.

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[8].

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 first choice vasopressor to reverse hypotension in vasodilatory shock[9].

Dobutamine is the synthetic catecholamine with predominantly beta adrenergic effect. As a result of beta 1 receptor-mediated positive ionotropic, and beta 2 receptor-mediated vasodilatory action, dobutamine increases cardiac output and decreases systemic and vascular resistance.

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[10].

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 grater metabolic requirements[11]. Severe haemorrhage impairs the delivery of O_2 and nutrients to the tissue and produces shock. Lowering regional vascular resistance by adenosine, prostaglandins and nitric oxide induces hypoxic redistribution of blood flow[12]. Inspite of this organ specific microvasculature response, all organ with the possible exception of the heart, experiences decrease in blood during severe hypovolemia[13].

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[14].

The early administration of red blood cells (RBCs) and fresh frozen plasma (FFP) is a priority to maintain arterial O_2 delivery and restore an effective coagulation. The administration of RBCs is considered indispensible when the Hb level is < 7 gm/dl[15]. This recommendation is based mainly on the results of the Transfusion Requirement in Critical Care (TRICC) study[16]. The administration of FFP should be associated as soon as possible with RBC transfusion to compensate for the deficit in coagulation factors. The initial recommended dose of FFP is 10-15 ml/kg[15]. It is recommended when PT or aPTT is 1.5 times the normal value. Platelet transfusion is recommended when PC < 50000 /L.

CONCLUSION:

Puerperal uterine inversion is an obstetric emergency requiring immediate management. Identification and treatment of Hemorrhage by aggressive resuscitation 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 acute puerperal inversion.

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