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FCA REFRESHER COURSE

Endovascular interventions in massive obstetric haemorrhage control

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Introduction

Obstetric haemorrhage remains one of the most common causes of maternal death and accounts for around 25% of maternal deaths worldwide.[1,2] It is also associated with significant maternal morbidity and often results in adverse fetal outcomes. Medical management of obstetric haemorrhage is well known. This review will focus on endovascular interventions in obstetric haemorrhage.

Postpartum haemorrhage

Uterine atony is the most common reason for postpartum haemorrhage (PPH), followed by abnormal placentation. Disseminated intravascular coagulation (DIC) and amniotic fluid embolism (AFE) are medical causes of bleeding.[3] Risk factors include a history of PPH (very strong predictor), previous uterine surgery, fibromas, anticoagulant use, fertility treatment, anaemia, severe preeclampsia, HELLP syndrome, and multiple pregnancy.

Anticipated major obstetric haemorrhage

Abdominal pregnancy and the conditions associated with abnormal placentation, such as placenta praevia, vasa praevia, and the placenta accreta spectrum (PAS), have the potential for massive bleeding/major obstetric haemorrhage (MOH). The bleeding may be difficult to control due to the uterus's rich blood supply, particularly to the placenta. Blood loss in placenta percreta can be more than 3 000 ml in 90% of cases.[4]

Placenta accreta spectrum

Patients with PAS should deliver at a specialist centre. Furthermore, a multidisciplinary approach to the management of PAS is critically important. Members of this team include experienced obstetricians, neonatologists, interventional radiologists, trauma surgeons, urologists, surgeons with expertise in complex pelvic surgery, anaesthetists, and intensivists.[5] There must be 24-hour access to a blood bank with the capacity to activate a massive blood transfusion protocol. A colorectal and vascular surgeon may be needed if placenta percreta is present.

The urologist inserts double-J stents into the ureters to make them easily identifiable during the surgery. The team must decide on whether and what endovascular modality will be used to control torrential bleeding should it occur. Cell salvage with a leucodepleted filter is useful to minimise the number of autologous blood products needed.

The role of the anaesthetist is crucial in managing the patient with potential haemorrhagic shock. They need to understand the considerations of obstetric haemorrhage and the technical aspects of endovascular procedures to manage the physiological changes appropriately.

Almost half of the patients with PAS deliver before 34-week gestation. Multidisciplinary discussion and planning need to occur early, and the team must be available on short notice in case an emergency delivery is needed.[5]

Risk factors for PAS:[5-7]

- Asherman syndrome
- Endometrial ablation
- In vitro fertilisation
- Maternal age > 35
- Multiparity
- Placenta praevia
- Previous caesarean delivery
- Previous dilatation and cerclage
- Previous uterine surgery

The role of the anaesthetist in the management of placenta accreta spectrum

Early evaluation by an anaesthetist is valuable to identify any clinical conditions that can be optimised before surgery. Referral to the relevant specialists should take place if significant comorbidities are present. Coagulation studies and a full blood count must be done, and any abnormalities addressed. Haemoglobin levels should be optimised. The blood bank should be informed that a massive blood transfusion protocol with the inclusion of cryoprecipitate may be activated. Cross-matched blood should be available immediately. The recommendation is

to have four units of red pack cells and four units of fresh frozen plasma available in theatre.[7]

Intraoperative management should consider the risk of massive bleeding. Two large bore (14 or 16 G) intravenous lines and intra-arterial blood pressure monitoring are standard requirements. A central venous catheter is not routinely sited and would depend on the clinical picture, the expected blood loss, and the possibility that inotropic support may be needed. Cell salvage should be available.[5,7] Tranexamic acid is given at the onset of bleeding and repeated if blood loss exceeds 1 500 ml.

Both neuraxial and general anaesthesia has been used in cases with PAS.[6,8] The benefits of general anaesthesia are that the haemodynamics are easier to manipulate in the setting of uncontrolled bleeding, and airway management is done electively and not when the patient is haemodynamically unstable. Surgery is often prolonged, and a neuraxial anaesthetic may need to be converted if the spinal block wears off or the patient becomes uncomfortable. A combined spinal-epidural provides immediate anaesthesia with the benefit of maintaining anaesthesia via the epidural catheter and extending analgesia into the postoperative period.

Neuraxial anaesthesia has the benefits of allowing the mother to participate in the birth of her baby, decreased airway and aspiration risks, decreased fetal exposure to anaesthetics and minimal uterine atony. If the mother wishes to be awake for the delivery, there is the option to start the case under neuraxial and convert to general anaesthesia once the baby is delivered.[6] A neuraxial technique, in addition to general anaesthesia, is associated with a decreased length of hospital stay.[9]The neuraxial procedure must be done before inserting any endovascular devices, as these devices preclude hip flexion.

Patients with MOH need monitoring in the postoperative period for ongoing bleeding, anaemia, DIC, organ dysfunction and fluid overload. Postoperative pain requires a multimodal approach. Intrathecal morphine is highly recommended. Truncal blocks done at the end of surgery is an alternative. Simple paracetamol and nonsteroidal anti-inflammatory drugs should be routine.[7] Patients who have endovascular interventions done should be monitored for intervention-specific complications.

Uterine blood supply[10,11]

The uterus has a dual blood supply and receives blood primarily from the left and right uterine arteries, with contributions from the left and right ovarian arteries. Ovarian arteries can contribute to uterine bleeding in around 12% of cases with PPH.[12]The uterine arteries are branches of the anterior division of the internal iliac arteries. The ovarian arteries most commonly arise directly from the descending aorta between the renal and inferior mesenteric arteries, while the left ovarian artery also commonly arises from the left renal artery. The round ligament arteries come from the inferior epigastric artery and form an intricate network of anastomoses with the ovarian artery.

The uterus has collateral supply from the ovarian, round ligament, internal pudendal, and inferior mesenteric arteries.

The uterine arteries enter the uterus around the isthmus and branch into an ascending and descending part. The ascending branch of the uterine artery becomes torturous as it travels cephalad on the side of the uterus, where it also anastomoses with the ovarian artery. It moves into the myometrium and divides into the arcuate, radial, spiral, and basal arteries.

During pregnancy, the uterine arteries almost double in size[13] and become a low-resistance system. The uterine blood supply towards the end of pregnancy increases to around 700–800 ml/min.[13] Uncontrolled bleeding from the uterus rapidly becomes life-threatening.

Endovascular interventional modalities

Endovascular haemorrhage control can be achieved through balloon occlusion of arteries or by injecting embolising material into an artery to encourage the formation of a blood clot within the artery. Sheaths are inserted under fluoroscopy using regional anaesthesia. Arterial access is via the femoral arteries. It is best achieved using ultrasound, especially in a shocked patient whose femoral pulse may be faint.[14] For arteries below the aortic bifurcation, both femoral arteries are cannulated to facilitate bilateral arterial occlusion. Balloon occlusion of the abdominal aorta requires only one side to be cannulated.

Prophylactic endovascular interventional management of PAS is associated with a decrease in blood loss, a decrease in hysterectomy rate, and a decrease in surgical time.[15-17] Endovascular balloon occlusion of the abdominal aorta is associated with the lowest hysterectomy rate, the least amount of blood loss, the least number of complications and the lowest maternal and fetal radiation doses. However, it requires a bigger insertion port with the resultant need for a vascular closure device.

Uterine artery embolisation[2]

An aortogram is done to determine the position of the uterine and ovarian arteries and excludes extravasation of contrast. A catheter is directed into the targeted arteries, and gelatine sponge particles are injected. The sponge particles are absorbed over three to six weeks, and the artery will subsequently undergo recanalisation, providing a chance for future fertility.

Bilateral uterine arteries are embolised first. The anterior divisions of the internal iliac arteries are embolised if the uterine arteries cannot be cannulated or if bilateral Uterine artery embolisation (UAE) is unsuccessful. Another aortogram is done to identify any other arteries that feed into the uterus. Other arteries that may contribute to uterine bleeding include the ovarian, vaginal, round ligament, obturator, deep circumflex iliac, pudendal, middle rectal, median sacral, iliolumbar arteries and superior rectal arteries that arise from the inferior mesenteric arteries.[1] These arteries must also be embolised if they contribute to the bleeding.

A warning sign that UAE alone may not be sufficient to arrest the bleeding is when the ovarian arteries are direct branches of the aorta. Additional ovarian artery embolisation (OAE) may be warranted. OAE is technically challenging and may not be achievable in a patient who is bleeding heavily. There should be no delay in proceeding to a hysterectomy in such cases. OAE can result in ovarian ischaemia that compromises fertility in patients where a hysterectomy is averted. Thus, the patient should be fully informed regarding the risks and benefits of arterial embolisation.

The success rate of UAE may be as high as 90%[1] but may fail under certain conditions. In addition to blood supply from other arteries, another reason for failure is the presence of DIC.[18] The gelatine sponges used in the embolisation process form a physical barrier to blood flow and provide a scaffold for the formation of a blood clot.[19] Sugai et al.[18] postulate that the deficiency of clotting factors in DIC contributes to the failure to form the blood clot. A different material known as N-butyl cyanoacrylate (NBCA) can be used to overcome this problem. NBCA results in permanent occlusion in the vessel, whereas gelatine sponges result in recanalisation of the artery in about two to six weeks.

Complications of arterial embolisation:

- Lower extremity deep vein thrombosis
- Dissection of internal iliac artery
  - Haemoperitoneum
- Endometriosis
- Uterine necrosis
- Damage to a persistent sciatic artery (rare anomaly)
  - Irreversible ischaemic damage to lower limb
- Altered menstruation
- Uterine artery dissection
- Post-embolisation syndrome
  - Fever
  - Abdominal pain
  - Mild leucocytosis
- Puncture site haematoma

The role of the anaesthetist in UAE is to provide analgesia and sedation for the endovascular procedure that takes place in the angiography suite, followed by the anaesthetic management of the caesarean delivery and hysterectomy. An epidural catheter is usually inserted before the UAE procedure, and a low dose of local anaesthetic is administered. The anaesthetic considerations for the surgical procedure include the need for invasive haemodynamic monitoring, aspiration prophylaxis, airway management, blood transfusion management and complications associated with massive blood loss.

Intra-arterial balloon occlusion

The success of intra-arterial balloon occlusion depends on the vessel in which the balloon is inflated. The arteries that can be occluded include bilateral internal iliac arteries, bilateral uterine arteries, bilateral common iliac arteries, and the abdominal aorta.[17] Occlusion at the level of the internal iliac vessels often fails due to the presence of collateral blood supply to the uterus. Balloon occlusion in the common iliac arteries has been used successfully.[20] Another option is balloon occlusion of the abdominal aorta (AABO). AABO risks occluding the inferior mesenteric artery with resultant bowel ischaemia.

Resuscitative endovascular balloon occlusion of the aorta

The earliest use of resuscitative endovascular balloon occlusion of the aorta (REBOA) dates back to the 1950s when it was used in the Korean War on two moribund patients.[21] A decade later, it was also used in a ruptured abdominal aorta aneurysm.[22] There has been renewed interest in the concept in the last few years, especially in locations where trauma is common and blood resources scarce.

REBOA is a bridging intervention used in uncontrolled bleeding until bleeding can be controlled with surgical or endovascular measures. It maintains cerebral and coronary perfusion in an otherwise haemodynamically unstable patient.[23] It has been used successfully in trauma cases and ruptured abdominal aorta aneurysms (rAAA) and is now employed in other non-compressible bleeding cases. These other indications include obstetric haemorrhage, pelvic bleeding, and massive gastrointestinal bleeding.

There are three types of REBOA occlusion. The first is complete REBOA where there is no flow past the balloon. Partial REBOA (P-REBOA) allows for some continuous flow past the balloon to allow some perfusion to organs distal to the occlusion, and intermittent REBOA where there is repeated inflation and deflation of the balloon.[24]

Blood loss is significantly decreased in PAS cases where REBOA are used.[25-27] REBOA also causes a significant increase in systolic blood pressure (SBP) in haemodynamically unstable patients. Two recent systematic reviews and meta-analyses[28,29] show an increase between 50–80 mmHg depending on the indication for the REBOA. Twenty-four-hour and in-hospital mortality is decreased.[28-30]

Aortic zones for the deployment of the resuscitative endovascular balloon occlusion of the aorta

The descending aorta is divided into three zones. Zone 1 starts at the left subclavian until the coeliac trunk. Zone 2 stretches from the coeliac trunk to the lower of the two renal arteries, and zone 3 from the renal arteries to the aortic bifurcation. The level at which the REBOA is deployed depends on the area of bleeding. Bleeding from the uterus can usually be controlled by inflating the REBOA in the distal part of zone 3 below the inferior mesenteric artery and the aortic bifurcation. The deployment distal to the inferior mesenteric artery allows blood flow to the ovaries and bowel. Balloon occlusion in zone 2 is contraindicated due to the high risk of bowel ischaemia.[24]

Resuscitative endovascular balloon occlusion of the aorta catheter designs[31]

The ideal REBOA catheter would be safe to use in injured blood vessels with a low risk of aorta injury or rupture, resistant to migration, easy and quick to insert, and amenable to use in the pre-hospital environment.[32] There are a few different types of REBOA catheters currently on the market. Each new product on the market aims to improve on previous designs to improve the device's safety and make it easier to use.

Currently, some of the market leaders in REBOA catheter design are Tokai Medical Products™, Prytime Medical™ (ER-REBOA™) and Frontline Medical Technologies™ with its newest addition, the COBRA-OS™. A prominent feature is a soft, flexible tip that allows for easy insertion without the need for a guidewire or imaging to confirm placement. The ER-REBOA™ has a trademarked 'P-tip' that has an extra curl compared to the 'J-tip' found in the COBRA-OS™. The function of the 'P-tip' is to mitigate the risk of catheter migration.

The Prytime™ device has a built-in arterial line port that can be connected to a transducer to monitor the pressure in the proximal aorta. Without such a port, the radial or brachial arterial line is used for proximal blood pressure monitoring. Blood pressure monitoring in the distal aorta (below the balloon) can be measured by attaching another transducer to the fluid line on the access port.

The catheters have radio-opaque lines adjacent to the balloon to indicate its position on fluoroscopy or X-ray. They also have length markers to assist in positioning the balloon in the correct area of the aorta. The landing area for zone 3 placement is typically around 28 cm from the insertion site, while those in zone 1 catheters are around 48 cm in the average patient.[31]

Physiological effects of resuscitative endovascular balloon occlusion of the aorta

The physiological effects of employing the REBOA depend on the level of occlusion of the aorta and whether it is completely or partially occluded.[33] Zone 1 occlusions are associated with more severe physiological effects and a more significant risk for complications. Mean arterial pressure proximal to the occlusion in zone 1 is associated with an increase of 172% over baseline in a swine model, while the change in zone 3 is a mere 10%.[33]

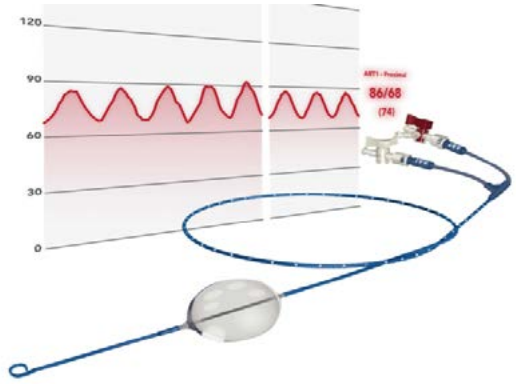

Figure 1: Prytime Medical™ ER-REBOA™
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Balloon occlusion increases the afterload and results in supra-physiological perfusion pressures to organs proximal to the occlusion, while organs distal to the occlusion are deprived of blood flow and become ischaemic.[34] Microcirculatory compromise is most pronounced in the colon and small bowel after zone 1 occlusion, while microcirculation in the intraabdominal organs and extremities is unchanged during zone 3 occlusion.[33]

A study in swine[34] shows an increase in end-diastolic pressure (ESP) while the end-systolic volume stays the same. REBOA also causes resolution of the reverse coronary artery blood flow that is characteristic of the shock state. However, the coronary blood flow shows a massive supraphysiological increase above baseline values, which persists even on partial REBOA. There is a clinically significant increase in lactate from baseline to post-REBOA and a significant decrease in pH. Troponin levels did not change from baseline to post-REBOA.

Complications of resuscitative endovascular balloon occlusion of the aorta

Iatrogenic injuries range from 2.6% in rAAA to 5.3% in non-traumatic bleeding, which includes obstetric bleeding.[28] The best outcomes are associated with using REBOA in specialised high-volume centres[35] with clear protocols for the indications and management of the patient in whom REBOA is used. These protocols are developed in consultation with vascular surgeons. Risk factors associated with an increased risk of complications are practitioners who are insufficiently trained in its management, practitioners with limited experience, limited resources, and the necessity of using REBOA in high-stakes emergency situations.

It is suggested that REBOA should only be used in PPH if medical management and uterine balloon occlusion have failed.[36]

Complications can be divided into perfusion-related injuries and procedure-related problems.[24]

Perfusion-related complications:[24,37,38]

- Organ dysfunction
- Reperfusion injury
- Tissue ischaemia, including spinal cord injuries with balloon placement in zone 1

Procedure-related complications:[24,37,38]

- Access site complications
  - Arterial disruption
  - Arterial rupture
  - Bleeding
  - Dissection
- Embolic complications
- Femoral artery thrombosis
- Iatrogenic aorta or iliac artery injuries

- Intimal tears
- Loop formation (catheter kinking on itself)
- Migration of the catheter
- Pseudo-aneurysm
- Retroperitoneal bleeding
- Ruptured balloon
- Vascular access failure

Placement of resuscitative endovascular balloon occlusion of the aorta

The REBOA device should be inserted by practitioners trained and qualified to manage the device. Ideally, the surgeon or interventional radiologist responsible for controlling the bleeding should do the insertion.[37] A practitioner that can stop the bleeding should be available immediately to decrease the ischaemic time.

The femoral artery is cannulated, and an access port is inserted. The size of the access points depends on the type of REBOA catheter used and ranges between 4 French (COBRA-OS™) and 12 French (COBRA™). The catheter is inserted and positioned at the desired level. The balloon is slowly inflated according to the manufacturer's recommendations, starting with an initial lower volume and titrating to the desired volume. The systolic blood pressure response is observed, and tiny volumes are added to the balloon until there is an improvement in the proximal haemodynamics. The catheter then needs to be secured to avoid migration.[39,40]

Weaning from resuscitative endovascular balloon occlusion of the aorta to partial resuscitative endovascular balloon occlusion of the aorta[23]

The maximum safe occlusion time is not clearly defined. A general guide is that ischaemic time with REBOA in zone 1 should not exceed 30 min or 60 min if deployed in zone 3. P-REBOA is a technique where the balloon is slowly deflated and titrated to allow a small amount of blood to pass the balloon. It still allows perfusion pressure to the brain and heart while creating a permissive hypotensive state below the balloon. It creates a balance between limiting blood loss and maintaining some distal perfusion. P-REBOA lessens the magnitude of the reperfusion injury and the supraphysiological haemodynamic values in the proximal aorta. It has a cardioprotective effect.[34] It is recommended that full REBOA be transitioned to P-REBOA as soon as is feasible, provided that the proximal mean arterial pressure is decent and that the necessary resuscitation and haemostatic control are ongoing. Enough time should be allowed for clot stabilisation before partial flow is restored to the distal aorta.[23]

The proximal and distal aortic blood pressure measurements are needed to guide transitioning to P-REBOA. The ER-REBOA™ has an integrated port to measure arterial pressure in the proximal aorta. For other catheters, it is acceptable to use the arterial blood pressure from the radial or brachial arterial line. For distal invasive blood pressure monitoring, a second transducer is attached to the flush port of the access device. Alternatively, the contralateral femoral artery is cannulated. A monitor that can show both arterial blood pressure traces simultaneously is needed. The scale on the trace that monitors blood pressure below the balloon is decreased to 0–30 mmHg so that very low pressures can be displayed. The distal tracing still shows a low pressure even during complete occlusion, and this is due to collateral flow to the distal aorta. There can be a small pulsatile waveform, but it is usually not the case in a severely volume-depleted patient.

The balloon port is attached to a 20 ml syringe to inflate the balloon. A 3-way tap is inserted between the port and the 20 ml, and an empty 3 ml or 5 ml syringe is attached to the 3-way tap. The transitioning is started by withdrawing 0.5 ml incrementally from the balloon until a pulsatile pressure wave is observed. Time is allowed to assess haemodynamics proximal to the balloon. If the blood pressure in the proximal aorta remains acceptable, more fluid is withdrawn from the balloon in tiny increments. After each withdrawal, at least 30 seconds are given to allow observation of the proximal blood pressure. The target systolic blood pressure in the distal aorta is between 20–50 mmHg.[40] The surgery is then completed under P-REBOA. If the patient starts bleeding again, the balloon can be inflated at any time.

Resuscitative endovascular balloon occlusion of the aorta removal

When the REBOA is no longer required, the balloon is deflated slowly to allow for gradual reperfusion and attenuation of the reperfusion injury. The balloon is deflated until the SBP distal to the balloon has increased by 50%. A five-minute period is given to allow for equilibration before the balloon is deflated again. The aim again is a 50% increase from the previous value. After complete deflation, extra negative pressure is applied to the balloon port to ensure complete deflation and the 3-way tap is closed to avoid reinflation of the balloon while it is withdrawn from the artery.[40]

Neurovascular monitoring is done for 24 hours to timeously detect lower limb and vascular access site complications.[40]

In some cases, arterial embolisation is performed after REBOA.[14] The same access port can be used with contralateral access also needed for bilateral embolisations.

Reperfusion

Deflation of the balloon is associated with reperfusion to ischaemic organs distal to the occlusion. Reperfusion injury is worse when REBOA is deployed in zone 1 but can still occur in zone 3 occlusion. The effects of reperfusion include a drop in pH, an increase in lactate levels, hyperkalaemia, hypocalcaemia, hypotension, and persistent hypoglycaemia.[41] A significant reperfusion injury and lactic acidosis depress cardiac performance, which can contribute to hypothermia. The triad

of hypothermia, coagulopathy and acidosis can be lethal in a patient with haemorrhagic shock.[41]

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References

1. Chen C, Lee SM, Kim JW, Shin JH. Recent update of embolization of postpartum hemorrhage. Korean J Radiol. 2018;19(4):585-96. https://doi.org/10.3348/kjr.2018.19.4.585.
2. Aoki M, Tokue H, Miyazaki M, et al. Primary postpartum hemorrhage: outcome of uterine artery embolization. Br J Radiol. 2018;91(1087):20180132. https://doi.org/10.1259/bjr.20180132
3. Kamijo K, Nakajima M, Shigemi D, et al. Resuscitative endovascular balloon occlusion of the aorta for life-threatening postpartum hemorrhage: A nationwide observational study in Japan. J Trauma Acute Care Surg. 2022;93(3):418-23. https://doi.org/10.1097/TA.0000000000003650
4. Hudon L, Belfort MA, Broome DR. Diagnosis and management of placenta percreta: a review. Obstet Gynecol Surv. 1998;53(8):509-17. https://doi.org/10.1097/00006254-199808000-00024
5. Einerson BD, Weiniger CF. Placenta accreta spectrum disorder: updates on anesthetic and surgical management strategies. Int J Obstet Anesth. 2021;46:102975. https://doi.org/10.1016/j.ijoa.2021.102975
6. Fiszer E, Weiniger CF. Placenta accreta. A review of current anesthetic considerations. Best Pract Res Clin Anaesthesiol. 2022;36(1):157-64. https://doi.org/10.1016/j.bpa.2022.01.003.
7. Reale SC, Farber MK. Management of patients with suspected placenta accreta spectrum. BJA Educ. 2022;22(2):43-51. https://doi.org/10.1016/j.bjae.2021.10.002
8. Warrick CM, Markley JC, Farber MK, et al. Placenta accreta spectrum disorders: Knowledge gaps in anesthesia care. Anesth Analg. 2022;135(1):191-7. https://doi.org/10.1213/ANE.0000000000005862.
9. Munoz JL, Pfeiffer AF, Curbelo J, Ramsey PS, Ireland KE. Neuraxial to general anesthesia conversion has equitable intraoperative and improved post-operative outcomes compared to general anesthesia in cesarean hysterectomy for placenta accreta spectrum (PAS). J Matern Fetal Neonatal Med. 2021:1-5. https://doi.org/10.1080/14767058.2021.1990885.
10. Chaudhry R, Chaudhry K. Anatomy, abdominal and pelvic, uterine arteries: Treasure Island (FL): Stat Pearl Publishing; 2022 [Updated 9 Sept 2021]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482267/.
11. Osol G, Mandala M. Maternal uterine vascular remodeling during pregnancy. Physiology (Bethesda). 2009;24:58-71. https://doi.org/10.1152/physiol.00033.2008.
12. Kim J-E, So YH, Kim BJ, et al. Postpartum hemorrhage from non-uterine arteries: clinical importance of their detection and the results of selective embolization. Acta radiol. 2018;59(8):932-8. https://doi.org/10.1177/0284185117738547.
13. Palmer SK, Zamudio S, Coffin C, et al. Quantitative estimation of human uterine artery blood flow and pelvic blood flow redistribution in pregnancy. Obstet Gynecol. 1992;80(6):1000-6.
14. Stensaeth KH, Sovik E, Haig IN, Skomedal E, Jorgensen A. Fluoroscopy-free resuscitative endovascular balloon occlusion of the aorta (REBOA) for controlling life threatening postpartum hemorrhage. PLoS One. 2017;12(3):e0174520. https://doi.org/10.1371/journal.pone.0174520.
15. D'Antonio F, Iacovelli A, Liberati M, et al. Role of interventional radiology in pregnancy complicated by placenta accreta spectrum disorder: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2019;53(6):743-51. https://doi.org/10.1002/uog.20131.
16. Nankali A, Salari N, Kazeminia M, et al. The effect prophylactic internal iliac artery balloon occlusion in patients with placenta previa or placental accreta spectrum: a systematic review and meta-analysis. Reprod Biol Endocrinol. 2021;19(1):40. https://doi.org/10.1186/s12958-021-00722-3.
17. Shahin Y, Pang CL. Endovascular interventional modalities for haemorrhage control in abnormal placental implantation deliveries: a systematic review and meta-analysis. Eur Radiol. 2018;28(7):2713-26. https://doi.org/10.1007/s00330-017-5222-0.
18. Sugai S, Nonaka T, Tamegai K, et al. Successful repeated uterine artery embolization in postpartum hemorrhage with disseminated intravascular coagulation: a case report and literature review. BMC Pregnancy Childbirth. 2021;21(1):710. https://doi.org/10.1186/s12884-021-04191-9.
19. Loffroy R, Guiu B, Cercueil JP, Krausé D. Endovascular therapeutic embolisation: an overview of occluding agents and their effects on embolised tissues. Curr Vasc Pharmacol. 2009;7(2):250-63. https://doi.org/10.2174/157016109787455617.
20. Murayama Y, Seki H, Takeda S. Intra-arterial balloon occlusion to reduce operative bleeding for placenta previa accreta spectrum. Surg J (N Y). 2021;7(Suppl 1):S11-19. https://doi.org/10.1055/s-0040-1721491.
21. Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery. 1954;36(1):65-68.
22. Heimbecker RO. An aortic tampon for emergency control of ruptured abdominal aneurysm. Can Med Assoc J. 1964;91:1024-25.
23. Johnson MA, Neff LP, Williams TK, DuBose JJ. Partial resuscitative balloon occlusion of the aorta (P-REBOA): Clinical technique and rationale. J Trauma Acute Care Surg. 2016;81(5):S133-7. https://doi.org/10.1097/TA.0000000000001146.
24. Aoki R, Kobayashi Y, Nawata S, et al. Computed tomography imaging of resuscitative endovascular balloon occlusion of the aorta (REBOA): pearls and pitfalls. Jpn J Radiol. 2021;39(12):1133-40. https://doi.org/10.1007/s11604-021-01166-w.
25. Manzano-Nunez R, Orlas CP, Herrera-Escobar JP, et al. A meta-analysis of the incidence of complications associated with groin access after the use of resuscitative endovascular balloon occlusion of the aorta in trauma patients. J Trauma Acute Care Surg. 2018;85(3):626-34. https://doi.org/10.1097/TA.0000000000001978.
26. Manzano-Nunez R, Escobar-Vidarte MF, Naranjo MP, et al. Expanding the field of acute care surgery: a systematic review of the use of resuscitative endovascular balloon occlusion of the aorta (REBOA) in cases of morbidly adherent placenta. Eur J Trauma Emerg Surg. 2018;44(4):519-26. https://doi.org/10.1007/s00068-017-0840-4.
27. Ordoñez CA, Manzano-Nunez R, Parra MW, et al. Prophylactic use of resuscitative endovascular balloon occlusion of the aorta in women with abnormal placentation: A systematic review, meta-analysis, and case series. J Trauma Acute Care Surg. 2018;84(5):809-18. https://doi.org/10.1097/TA.0000000000001821.
28. Borger van der Burg BLS, Van Dongen TTCF, Morrison JJ, et al. A systematic review and meta-analysis of the use of resuscitative endovascular balloon occlusion of the aorta in the management of major exsanguination. Eur J Trauma Emerg Surg. 2018;44(4):535-50. https://doi.org/10.1007/s00068-018-0959-y.
29. Granieri S, Frassini S, Cimbanassi S, et al. Impact of resuscitative endovascular balloon occlusion of the aorta (REBOA) in traumatic abdominal and pelvic exsanguination: a systematic review and meta-analysis. Eur J Trauma Emerg Surg. 2022. https://doi.org/10.1007/s00068-022-01955-6.
30. Harfouche MN, Madurska MJ, Elansary N, et al. Resuscitative endovascular balloon occlusion of the aorta associated with improved survival in hemorrhagic shock. PLoS One. 2022;17(3):e0265778. https://doi.org/10.1371/journal.pone.0265778.
31. McGonigal M. REBOA: A comparison of hardware from two companies. 2021. Available from: https://thetraumapro.com/2021/01/22/reboa-a-comparison-of-the-hardware-from-two-companies/.
32. Smith SA, McAlister VC. A novel REBOA system: prototype and proof of concept. Can J Surg. 2018;61(6):S188-94. https://doi.org/10.1503/cjs.015718.
33. Halvachizadeh S, Mica L, Kalbas Y, et al. Zone-dependent acute circulatory changes in abdominal organs and extremities after resuscitative balloon occlusion of the aorta (REBOA): an experimental model. Eur J Med Res. 2021;26(1):10. https://doi.org/10.1186/s40001-021-00485-y.
34. Stonko DP, Edwards J, Abdou H, et al. The underlying cardiovascular mechanisms of resuscitation and injury of REBOA and partial REBOA. Front Physiol. 2022;13:871073. https://doi.org/10.3389/fphys.2022.871073.
35. Gorman E, Nowak B, Klein M, et al. High resuscitative endovascular balloon occlusion of the aorta procedural volume is associated with improved outcomes: An analysis of the AORTA registry. J Trauma Acute Care Surg. 2021;91(5):781-9. https://doi.org/10.1097/TA.0000000000003201.
36. Brede JR, Søvik E, Rehn M. Resuscitative endovascular balloon occlusion of the aorta: the postpartum haemorrhage perspective. Crit Care. 2022;26(1):57. https://doi.org/10.1186/s13054-022-03942-0.
37. Brenner M, Bulger EM, Perina DG, et al. Joint statement from the American College of Surgeons Committee on Trauma (ACS COT) and the American College of Emergency Physicians (ACEP) regarding the clinical use of Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA). Trauma Surg Acute Care Open. 2018;3(1):e000154. https://doi.org/10.1136/tsaco-2017-000154.
38. Ribeiro Junior MAF, Feng CYD, Nguyen ATM, et al. The complications associated with resuscitative endovascular balloon occlusion of the aorta (REBOA). World J Emerg Surg. 2018;13:20. https://doi.org/10.1186/s13017-018-0181-6.
39. ER-REBOA PLUS Catheter Quick Reference Guide 'No One Should Bleed To Death' Available from: https://prytimemedical.com/wp-content/uploads/2020/09/ADV-061-Rev-C_ER-REBOA-PLUS-Catheter-Quick-Reference-Guide-8.5-x-11.pdf.
40. Van Skike CE, Baer DG, Spalding MC, Radomski M. Complete and partial resuscitative endovascular balloon occlusion of the aorta for hemorrhagic shock. J Vis Exp. 2022;(183). https://doi.org/10.3791/63767.
41. Abid M, Neff LP, Russo RM, et al. Reperfusion repercussions: a review of the metabolic derangements following resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2020;89(2S):S39-44. https://doi.org/10.1097/TA.0000000000002761.