A Review of Nonpharmacologic Prevention and Management of Postpartum Haemorrhage in Low-Resource Settings

Postpartum haemorrhage (PPH) constitutes blood loss from the genital tract in excess of 500 ml following vaginal delivery or 1000 ml after caesarean section (CS), or any amount of blood loss accompanied by signs of haemodynamic compromise irrespective of the delivery route.[1,2] PPH occurring within 24 h following delivery is primary, while secondary PPH occurs after 24 h and up to six weeks postpartum. PPH accounts for one-quarter of all maternal mortality globally. In low- and middle-income countries (LMICs), it is the leading cause of maternal death, accounting for up to 25%–43% of all maternal mortality in these countries, with 44,000–86,000 annual-related deaths.[3,4] Preventing PPH is an important intervention towards achieving Sustainable Development Goal 3 (SDG 3), with a target to reduce global maternal mortality ratio to below 70 per 100,000 live births by 2030.

Uterine blood flow at term approximates 600 ml (500–700 ml) per minute, which is ten times the rate in the nonpregnant state.[5] This high blood flow predisposes the gravid uterus to significant bleeding postpartum. The control of postpartum blood loss is primarily dependent on uterine contractions; uterine atony is implicated in up to 70%–80% of cases of PPH.[2] The muscle fibers of the uterine myometrium are arranged in a criss-cross pattern and the uterine blood vessels pass through this interlacing network of muscle fibers to supply the placental bed. When the uterus contracts, the muscle fibers effectively compress the blood vessels to arrest bleeding. This myometrial architecture constitutes the "living ligatures" or "physiological sutures" of the uterus.[6] Uterotonics, which are pharmacological agents that increase uterine muscle tone and contraction, therefore play a central role in the prevention and treatment of PPH. Uterotonics are recommended by the World Health Organisation (WHO) as a part of active management of third-stage labour to prevent PPH.[3]

Owing to poor storage conditions and adulteration, uterotonic doses in many resource-poor settings may not be optimally effective. For instance, oxytocin, the first choice uterotonic for the prevention and treatment of PPH, is degraded at high temperatures and should be stored away from light at a temperature of 2°C–8°C for optimum efficacy.[3,7] Misoprostol, an effective alternative to oxytocin, is degraded by humidity and should be stored in double aluminum blister packs.[8] These storage conditions are extremely difficult to meet in many LMICs, with humid tropical climates, and epileptic electricity supply. More so, many adulterated brands of these uterotonics are marketed in LMICs, owing to widespread poverty. A review revealed that 57.5% of oxytocin supplies in Africa were substandard; another study in Nigeria showed that 74.2% of oxytocin doses were poor quality.[7,8]

Sparse availability and high-cost limit the use of other uterotonics such as carbetocin and carboprost (15-methyl prostaglandin F2α) in low-resource settings.[3,6] Ergometrine use is excluded in women with hypertensive disorders, which are twice as prevalent in LMICs compared to high-income countries.[9] Tranexamic acid (TXA), an antifibrinolytic agent, which is effective for all PPH cases regardless of etiology, is not routinely used in many resource-poor settings, owing in part to concerns about cost-effectiveness. More so, its use is recommended within three hours of delivery for maximum efficacy.[10] Many women in LMICs, who labour and deliver outside the hospital, only present to the hospital long after complications have set in, at which time the three-hour window of optimal effectiveness of TXA would have lapsed. This is against the backdrop of the fact that unskilled health personnel are not recommended to administer TXA.[3]

Given that effective uterotonics may not be readily available in many LMICs due to poor storage, adulteration, and cost, it is important for practitioners in these settings to be very well acquainted with the nonpharmacologic measures of preventing and treating PPH. The preventive measures should start during the preconception period. Teenage and unintended pregnancies should be prevented, and chronic medical conditions, which adversely affect a woman's ability to tolerate acute haemorrhage postpartum, should be optimally controlled before pregnancy. Women should adopt contraception and adequate pregnancy spacing, because frequent pregnancies within short intervals are associated with anaemia in pregnancy, which predisposes to PPH.

Anaemia, which is prevalent in LMICs, is a significant risk factor for PPH. Nutritional iron supplementation is essential in preventing iron deficiency, which is the most common cause of anaemia in LMICs. The WHO cutoff of Hb <11 g/dl (rather than the <10 g/dl currently being used) for anaemia should be adopted in LMICs.[11] Women who start pregnancy with Hb of ≥11 g/dl are less at risk of PPH than those with 10 g/dl. Women should be enlightened on the benefits of skilled antenatal care and birth attendance, to facilitate early recognition of risk factors for PPH and reduce its risk. Efforts should be targeted at reducing rates of CS, which carries a higher PPH risk than vaginal delivery.

Active management of third-stage labour, which includes the nonpharmacological interventions of placental delivery by controlled cord traction and postpartum assessment for uterine tonicity through quarter-hourly abdominal palpation for the first 2 h, should be offered to all women, to reduce the risk of PPH.[3] Episiotomies and genital tract lacerations

following delivery should be promptly repaired. Early initiation of breastfeeding within one hour of delivery should be encouraged. Breastfeeding stimulates oxytocin release, which causes uterine contractions, thereby preventing PPH. At the community level, men should be incorporated as champions of PPH prevention. Male involvement has proven to positively impact access to maternity services, especially in the patriarchal LMICs.[12]

When PPH occurs, beside the administration of oxytocics, the uterus should be massaged, and if PPH persists, having excluded retained placental tissue, genital tract lacerations and coagulopathy, bimanual compression of the uterus should be conducted. This is done by inserting a hand into the vagina and forming the vaginal hand into a fist, which is used to apply pressure to the lower uterus. The other hand is used to apply external pressure to the uterine fundus so that the uterus is compressed between the abdominal and vaginal hands. The principle underlying bimanual uterine compression is that compression of haemorrhaging vessels slows or stops blood flow. For this to be effectively done, uterine compression should be maintained for at least 5–10 min and up to 30–60 min.[13] Even though bimanual uterine compression is designed to be performed by a single clinician, a single person may be unable to consistently sustain adequate uterine compression for the recommended time owing to fatigue. Bimanual uterine compression may therefore be more effective if performed by two providers: one applies external pressure to the uterine fundus, while the other applies intravaginal pressure to the lower uterus.[13]

Intrauterine balloon tamponade is also an effective mechanical method for controlling intractable atonic PPH that is unamenable to pharmacotherapy. It involves instilling up to 500 ml of fluid into an inflatable intrauterine balloon. The pressure exerted by the inflated balloon provides a tamponade effect between the catheter balloon and the uterine wall, occluding vessels on the uterine wall. The commonly used devices for balloon tamponade include the Rusch catheter, the Sengstaken–Blakemore tube, and the Bakri catheter.[14] The Bakri catheter has the advantage of a draining port, which prevents concealed haemorrhage and allows for monitoring of ongoing blood loss. In resource-poor settings, where the Rusch, Sengstaken–Blakemore and Bakri catheters are expensive and not readily available, multiple large Foley catheters can be inserted into the uterus using a sponge holding forceps and the balloon of each catheter inflated with up to 60 ml of fluid. The catheters are then anchored to the thighs of the patient in traction.[2,15] A condom can also be used for tamponade in resource-poor settings. The tubing of an intravenous fluid-giving set is inserted into the condom, and a suture material or string is tied around the condom and the tubing. Using a sponge holding forceps, the condom with the tubing is introduced transcervically into the uterus. The intravenous fluid is then opened to inflate the intrauterine condom with 300–500 ml of fluid.[16,17] As the condom expands within the uterus, it makes a seal at the level of the internal cervical os, becoming trapped within the uterus. The inflated intrauterine condom tamponades against the uterine wall to stop the bleeding. The condom can also be attached to a Foley catheter and the drip-giving set connected to the catheter to inflate the intrauterine condom. The catheter or condom is removed up to 24 h after insertion.[5]

Uterine packing with gauze for controlling PPH is not recommended. This is because the gauze packing can absorb large quantities of blood, thereby concealing ongoing haemorrhage. Furthermore, the uterus is distensible and a large amount of bleeding can occur unnoticed behind the gauze packing. There is also the risk of intrauterine infection.[18]

In resource-poor settings, where many women deliver outside the hospital and in midwifery-led units, the nonpneumatic antishock garment (NASG) can stabilise women with PPH and prevent mortality while they are being transferred to a hospital for definitive treatment. The NASG is a compression suit that has five neoprene segments secured tightly with Velcro, with a detachable foam ball for additional pressure in the abdominal section. The segments are applied tightly around the legs, pelvis, and abdomen, to redistribute/shunt blood from the lower extremities and abdomen to the vital organs of the heart, lungs, and brain.[19]

If PPH persists despite the administration of uterotonics and application of bimanual uterine massage and balloon tamponade, surgical treatment should be considered. While the patient is being moved to the theatre for surgical management, external aortic compression can help reduce blood loss. Aortic compression decreases distal aortic and hence uterine blood flow, thus controlling life-threatening haemorrhage.[20] To perform anterior abdominal aortic compression, the closed fist is placed just above the umbilicus and slightly to the left. Downward pressure is then directly applied and sustained through the anterior abdomen to compress the abdominal aorta against the lumbar vertebra. The effectiveness of the aortic compression is checked by palpating for the femoral pulse.[21]

Conservative surgical treatment of PPH includes uterine compression sutures, so-called "brace sutures," first described in 1997 by Christopher Balogun-Lynch (B-Lynch).[22] The B-Lynch suture is successful in more than 90% of cases for the management of PPH. It can be applied at CS or following a laparotomy and hysterotomy, if delivery is vaginal. A hysterotomy in this instance allows for exploration of the uterine cavity and evacuation of any placental fragments or clots, as well as for correct placement of the suture to avoid obliteration of the uterine cavity.[13] A large suture (B-Lynch originally used no. 2 catgut) is used to prevent breaking, and the suture should be rapidly absorbable to avoid the risk of bowel herniating through a persistent loop of suture after the uterus has involuted.[2]

To apply the B-Lynch suture, the uterus is exteriorised, and suture bites are taken approximately 3 cm below and 3 cm above the right edge of the uterine incision. The suture is

then looped over the uterine fundus and down to the posterior wall of the uterus opposite the uterine incision. The suture is passed through the posterior uterine wall into the uterine cavity. Another bite is taken to exit the uterine cavity on the left side, approximately 4 cm from the left lateral margin of the uterus. The suture is looped over the posterior uterine wall down to the anterior wall, and bites are taken 3 cm above and then 3 cm below the left edge of the uterine incision. The two ends of the suture are then pulled taut while an assistant applies anteroposterior compression of the uterus with both hands. The ends of the sutures are then tied in 3–4 knots to secure tension, and the uterine incision is closed as usual.[22] This compresses the arcuate vessels running through the anterior and posterior uterine walls. The suture running across at the level of the isthmus compresses the vessels running upward from the level of the cervix.[20] Modifications of the B-Lynch suture include the Cho and Hayman sutures.[2]

Where compression sutures fail to arrest PPH, bilateral uterine artery ligation is an effective next step, as the uterine artery accounts for up to 90% of the uterine blood supply.[22] As described by Waters in 1952 and O'Leary in 1964, the uterine artery is ligated by passing a no. 1 chromic catgut suture anteroposteriorly through the muscle of the lower uterine segment, 2 cm medial to the lateral margin of the uterus, and 2 cm below the line of a transverse lower segment uterine incision. The suture is then brought forward through the avascular area at the base of the broad ligament and tied.[23,24] If bilateral uterine artery ligation fails to control PPH, the ovarian and internal iliac arteries can be ligated sequentially. Internal iliac artery ligation is however time-consuming, technically challenging, less successful (50%–60% success rate), and carries the risk of injury to contiguous structures; it is, therefore, less frequently done.[2,18] Early recourse to total or subtotal hysterectomy in cases of intractable PPH refractory to medical and conservative surgical treatment measures is often lifesaving.

In conclusion, nonpharmacologic measures may just be the hitherto neglected silver bullet needed for reducing maternal mortality and morbidity from PPH in resource-poor settings, where available doses of uterotonics may be suboptimally effective due to poor storage and adulteration, with some, unavailable, owing to high cost. It becomes very necessary to reemphasise this as the WHO is currently tasking all countries to identify and prevent all avoidable causes of maternal mortality and morbidity, with just seven years to the SDGs target year of 2030.

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