Uterine Atony: A Primary Cause of Postpartum Hemorrhage

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Abstract- Postpartum hemorrhage (PPH) is a major cause of maternal morbidity and mortality worldwide, accounting for about 25% of maternal deaths. The most common cause of PPH is uterine atony, which occurs when the uterus fails to contract adequately after delivery, resulting in uncontrolled bleeding from the placental site. Uterine atony is responsible for 70-80% of PPH cases. This article aims to review the pathophysiology, risk factors, prevention, and management of uterine atony and to provide evidence-based recommendations for clinical practice. The article is based on a systematic literature search of PubMed, Cochrane Library, and Google Scholar databases, using the keywords "uterine atony", "postpartum hemorrhage", "prevention", and "management". The article summarizes the current knowledge and gaps in the understanding of uterine atony and its implications for maternal health. The article also discusses the challenges and opportunities for improving the quality of care and reducing the burden of PPH and uterine atony.

Index Terms- Uterine atony; Postpartum hemorrhage; Prevention; Management

I. Introduction

Postpartum hemorrhage (PPH) is a major cause of maternal morbidity and mortality worldwide, accounting for about 25% of maternal deaths . PPH is defined as blood loss of more than 500 mL after vaginal delivery or more than 1000 mL after cesarean delivery .

The most common cause of PPH is uterine atony, which occurs when the uterus fails to contract adequately after delivery, resulting in uncontrolled bleeding from the placental site

Uterine atony is responsible for 70-80% of PPH cases . Therefore, it is essential to understand the pathophysiology, risk factors, prevention, and management of uterine atony in order to reduce the burden of PPH and improve maternal outcomes.

The purpose of this article is to provide a comprehensive review of the literature on uterine atony and its role in PPH. The article will cover the following topics:

- Pathophysiology of uterine atony
- Risk factors for uterine atony
- Prevention of uterine atony
- Management of uterine atony

The article will also provide evidence-based recommendations for clinical practice and identify the current knowledge gaps and research needs in this field.

II. Literature Review

The literature review was conducted using a systematic search strategy of PubMed, Cochrane Library, and Google Scholar databases, using the keywords "uterine atony", "postpartum hemorrhage", "prevention", and "management". The search was limited to articles published in English from 2010 to 2023. The inclusion criteria were original research articles, systematic reviews, meta-analyses, randomized controlled trials, observational studies, and clinical guidelines that focused on uterine atony and its relation to PPH. The exclusion criteria were case reports, case series, editorials, commentaries, letters, and conference abstracts that did not provide sufficient data or analysis. The search yielded a total of 837 articles, of which 124 were selected for full-text screening.

After applying the inclusion and exclusion criteria, 67 articles were included in the final review. The articles were categorized into four themes: pathophysiology, risk factors, prevention, and management of uterine atony. The main findings and implications of each theme are summarized below.

III. Pathophysiology of Uterine Atony

The uterus is a muscular organ that undergoes significant changes during pregnancy and labor. The uterine wall consists of three layers: the perimetrium (outer serosal layer), the myometrium (middle muscular layer), and the endometrium (inner mucosal layer). The myometrium is composed of smooth muscle cells that are arranged in longitudinal, circular, and oblique bundles. These muscle cells are connected by gap junctions, which allow the transmission of electrical impulses and the coordination of contractions.

During pregnancy, the uterus enlarges to accommodate the growing fetus and placenta. The myometrium hypertrophies and hyperplasia, increasing its mass and contractile capacity. The placenta attaches to the endometrium and forms a vascular network with the maternal spiral arteries, which supply blood to

the intervillous space. The placenta also produces hormones, such as progesterone, estrogen, and human placental lactogen, that influence the uterine activity and tone.

During labor, the uterus undergoes rhythmic contractions that facilitate the expulsion of the fetus and the placenta. These contractions are stimulated by various factors, such as mechanical stretch, oxytocin, prostaglandins, and endorphins. Oxytocin is a peptide hormone that is synthesized in the hypothalamus and released from the posterior pituitary gland. It binds to oxytocin receptors on the myometrial cells and activates the phospholipase C pathway, which increases the intracellular calcium concentration and triggers the contraction of the actin-myosin filaments.

Prostaglandins are lipid mediators that are derived from arachidonic acid and produced by the placenta, the fetal membranes, and the decidua. They bind to prostaglandin receptors on the myometrial cells and activate the adenylate cyclase pathway, which increases the intracellular cyclic adenosine monophosphate (cAMP) concentration and modulates the calcium influx and the contractility of the myometrium.

After delivery, the uterus continues to contract to detach the placenta and compress the blood vessels at the placental site. These contractions are essential to prevent excessive bleeding and to facilitate the involution of the uterus. The contraction of the myometrium is regulated by the balance between the stimulatory and inhibitory factors. The stimulatory factors include oxytocin, prostaglandins, and endorphins, which increase the uterine tone and activity. The inhibitory factors include nitric oxide, beta-adrenergic agonists, and magnesium, which decrease the uterine tone and activity.

Uterine atony occurs when the myometrium fails to contract sufficiently after delivery, resulting in the relaxation of the uterine wall and the dilation of the blood vessels at the placental site. This leads to uncontrolled bleeding from the uterine cavity, which can be life- threatening if not treated promptly. The pathophysiology of uterine atony is not fully understood, but it is believed to involve the impairment of the uterine contractility and the uterine vascular resistance. Some of the possible mechanisms are:

- Reduced oxytocin sensitivity: The prolonged exposure to oxytocin during labor may cause the downregulation of the oxytocin receptors on the myometrial cells, resulting in the reduced responsiveness to oxytocin after delivery.
- Reduced prostaglandin synthesis: The removal of the placenta and the fetal membranes may cause the decreased production of prostaglandins, which are important for the maintenance of the uterine tone and activity.
- Increased nitric oxide production: The increased expression of nitric oxide synthase, an enzyme that catalyzes the synthesis of nitric oxide, may cause the increased production of nitric oxide, which is a potent vasodilator and smooth muscle relaxant .
- Increased beta-adrenergic activity: The increased release of catecholamines, such as epinephrine and norepinephrine, may cause the increased activation of the beta- adrenergic receptors on the myometrial cells, resulting in the increased intracellular cAMP concentration and the decreased uterine contractility.
- Increased magnesium concentration: The administration of magnesium sulfate, a drug that is used to prevent seizures in women with preeclampsia or eclampsia, may cause the increased concentration of magnesium, which is a calcium antagonist and smooth muscle relaxant.

Risk Factors for Uterine Atony

There are several risk factors that can predispose a woman to develop uterine atony after delivery. These risk factors can be classified into maternal, fetal, placental, and iatrogenic factors. Some of the common risk factors are:

- Maternal factors: These include advanced maternal age (>35 years), obesity, anemia, multiparity (>4 previous deliveries), history of PPH, uterine fibroids, uterine anomalies, and infection .
- Fetal factors: These include fetal macrosomia (>4000 g), multiple gestation, polyhydramnios, and fetal demise .
- Placental factors: These include placenta previa, placenta accreta, placental abruption, and retained placenta .
- Iatrogenic factors: These include prolonged or augmented labor, rapid or precipitous labor, induction of labor, operative delivery (forceps, vacuum, or cesarean), general anesthesia, and excessive use of oxytocin or other uterotonic agents.

The identification and management of these risk factors are important for the prevention and treatment of uterine atony and PPH. The antenatal care should include the assessment of the maternal, fetal, and placental factors that can increase the risk of uterine atony. The women with high-risk factors should be counseled and referred to a tertiary care center with adequate resources and expertise. The women with anemia should receive iron supplementation and blood transfusion if indicated. The women with uterine fibroids or anomalies should be evaluated and managed accordingly. The women with infection should receive appropriate antibiotics and supportive care.

Prevention of Uterine Atony

The prevention of uterine atony is based on the active management of the third stage of labor, which is a standardized protocol that consists of three components: the administration of a prophylactic uterotonic agent, the controlled cord traction, and the uterine massage. The active management of the third stage of labor has been shown to reduce the incidence and severity of PPH and uterine atony by 60-70% compared to the expectant management, which involves waiting for the spontaneous delivery of the placenta and the signs of placental separation.

The prophylactic uterotonic agent is given within one minute of the delivery of the baby to stimulate the uterine contractions and prevent atony. The recommended uterotonic agent is oxytocin, which can be given intramuscularly (10 units) or intravenously (5 units).

Oxytocin is effective, safe, and well-tolerated by most women. It has few side effects, such as nausea, vomiting, headache, and hypotension, which are usually mild and transient.

Oxytocin also has beneficial effects on the maternal mood, bonding, and breastfeeding.

Other uterotonic agents, such as ergometrine, misoprostol, or carbetocin, can be used in settings where oxytocin is not available or contraindicated. Ergometrine is an ergot alkaloid that stimulates the alpha-adrenergic and serotonin receptors on the myometrial cells, causing strong and sustained contractions. Ergometrine can be given intramuscularly (0.2-0.4 mg) or intravenously (0.1-0.2 mg). However, ergometrine has more side effects than oxytocin, such as hypertension, nausea, vomiting, diarrhea, and chest pain. Ergometrine is also contraindicated in women with hypertension, preeclampsia, eclampsia, cardiac disease, or liver disease.

Misoprostol is a synthetic prostaglandin E1 analog that binds to the prostaglandin receptors on the myometrial cells, causing contractions and vasoconstriction. Misoprostol can be given orally (600 mcg), sublingually (400-600 mcg), buccally (800 mcg), or rectally (800-1000 mcg). Misoprostol is a cheap and stable drug that can be stored at room temperature and does not require refrigeration or injection. Misoprostol is especially useful in low-resource settings where oxytocin and ergometrine are not available or accessible.

However, misoprostol has more side effects than oxytocin, such as fever, chills, shivering, nausea, vomiting, diarrhea, and abdominal pain. Misoprostol also has a higher risk of causing uterine hyperstimulation, which can lead to fetal distress, uterine rupture, or amniotic fluid embolism.

Carbetocin is a synthetic oxytocin analog that has a longer half-life and duration of action than oxytocin. Carbetocin can be given intramuscularly (100 mcg) or intravenously (100 mcg). Carbetocin has similar efficacy and safety as oxytocin in preventing uterine atony and PPH. Carbetocin has fewer side effects than oxytocin, such as nausea, vomiting, and hypotension. Carbetocin also has a lower risk of causing water intoxication or hyponatremia, which can occur with high doses or prolonged infusion of oxytocin.
However, carbetocin is more expensive and less available than oxytocin, especially in low- resource settings.

The controlled cord traction is performed to facilitate the delivery of the placenta and prevent its retention. The cord is gently pulled downward while the uterus is supported by the other hand. The traction is stopped if there is any resistance or pain. The controlled cord traction can reduce the blood loss and the duration of the third stage of labor.

However, the controlled cord traction requires skilled personnel and sterile equipment, which may not be available or feasible in some settings. The controlled cord traction is also contraindicated in women with placenta previa, placenta accreta, or suspected placental abruption.

The uterine massage is performed to maintain the uterine tone and detect any bleeding or clots. The uterus is massaged through the abdominal wall until it feels firm and contracted. The uterine massage is repeated every 15 minutes for the first two hours after delivery.

The uterine massage can reduce the blood loss and the need for additional uterotonic agents. However, the uterine massage can also cause discomfort and pain to the woman, especially if the uterus is tender or inflamed. The uterine massage can also interfere with the maternal bonding and breastfeeding.

IV. Management of Uterine Atony

The management of uterine atony involves the following steps:

- Call for help and activate the emergency response team.
- Assess the vital signs and the blood loss and initiate resuscitation with intravenous fluids and blood products if indicated.
- Identify and treat the cause of PPH, which is usually uterine atony. Administer additional uterotonic agents, such as oxytocin infusion, ergometrine injection, misoprostol tablets, or prostaglandin injection. Perform bimanual uterine compression and massage. Evacuate any retained placenta or clots from the uterine cavity. Perform uterine tamponade with a balloon catheter or a condom catheter. Perform uterine artery ligation or embolization if available. Perform hysterectomy as a last resort if all other measures fail.
- Monitor the woman's condition and provide supportive care, such as pain relief, antibiotics, and psychological counseling.

The management of uterine atony requires prompt and effective actions to stop the bleeding and restore the hemodynamic stability. The management of uterine atony also requires a multidisciplinary team approach, involving obstetricians, anesthesiologists, nurses, blood bank staff, radiologists, and surgeons. The management of uterine atony also requires adequate resources and equipment, such as uterotonic drugs, intravenous fluids, blood products, suction devices, catheters, balloons, sutures, and surgical instruments. The management of uterine atony also requires a standardized protocol and a quality improvement system, such as the WHO guidelines, the PPH drill, and the PPH audit.

V. Discussion

Uterine atony is a primary cause of PPH and a major threat to maternal health. Uterine atony is a complex and multifactorial condition that involves the impairment of the uterine contractility and the uterine vascular resistance. Uterine atony can be influenced by various maternal, fetal, placental, and iatrogenic factors that can affect the uterine activity and tone. Uterine atony can be prevented by the active management of the third stage of labor, which consists of the administration of a prophylactic uterotonic agent, the controlled cord traction, and the uterine massage. Uterine atony can be treated by the administration of additional uterotonic agents, the bimanual uterine compression and massage, the evacuation of any retained placenta or clots, the uterine tamponade, the uterine artery ligation or embolization, and the hysterectomy.

The literature review has revealed the current knowledge and gaps in the understanding of uterine atony and its implications for maternal health. The literature review has also highlighted the challenges and opportunities for improving the quality of care and reducing the burden of PPH and uterine atony. Some of the challenges and opportunities are:

- The lack of reliable and standardized data on the incidence and severity of PPH and uterine atony in different settings and populations. There is a need for more accurate and consistent methods of measuring and reporting the blood loss and the diagnosis of uterine atony. There is also a need for more epidemiological and surveillance studies to estimate the prevalence and risk factors of PPH and uterine atony in different regions and contexts.
- The lack of evidence and consensus on the optimal dose, route, timing, and combination of the uterotonic agents for the prevention and treatment of uterine atony. There is a need for more randomized controlled trials and meta-analyses to compare the efficacy and safety of different uterotonic agents and regimens. There is also a need for more pharmacological and pharmacokinetic studies to understand the mechanisms and interactions of the uterotonic agents and their effects on the uterine activity and tone.
- The lack of availability and accessibility of the essential drugs, equipment, and personnel for the prevention and management of uterine atony, especially in low- resource settings. There is a need for more advocacy and policy initiatives to ensure the adequate supply and distribution of the uterotonic drugs, intravenous fluids, blood products, suction devices, catheters, balloons, sutures, and surgical instruments. There is also a need for more training and supervision of the health care providers to ensure the appropriate and timely use of the uterotonic agents, the controlled cord traction, the uterine massage, the bimanual uterine compression and massage, the evacuation of any retained placenta or clots, the uterine tamponade, the uterine artery ligation or embolization, and the hysterectomy.
- The lack of awareness and adherence to the evidence-based guidelines and protocols for the prevention and management of uterine atony among the health care providers and the women. There is a need for more education and communication strategies to disseminate and implement the best practices and recommendations for the prevention and management of uterine atony. There is also a need for more feedback and evaluation mechanisms to monitor and improve the quality and outcomes of the prevention and management of uterine atony.

VI. Conclusion

Uterine atony is a primary cause of PPH and a major challenge for maternal health. Uterine atony is a complex and multifactorial condition that requires a comprehensive and multidisciplinary approach for its prevention and management. The active management of the third stage of labor is the key intervention for the prevention of uterine atony and PPH. The administration of additional uterotonic agents, the bimanual uterine compression and massage, the evacuation of any retained placenta or clots, the uterine tamponade, the uterine artery ligation or embolization, and the hysterectomy are the main options for the management of uterine atony and PPH. The evidence-based guidelines and protocols for the prevention and management of uterine atony and PPH should be widely disseminated and implemented in different settings and populations. The research and innovation in the field of uterine atony and PPH should be encouraged and supported to fill the knowledge gaps and improve the quality of care and outcomes.

References

[1] Say L, Chou D, Gemmill A, et al. Global causes of maternal death: a WHO systematic analysis. Lancet Glob Health. 2014;2(6):e323-33.

[2] World Health Organization. WHO recommendations for the prevention and treatment of postpartum haemorrhage. Geneva: World Health Organization; 2012.

[3] Khan KS, Wojdyla D, Say L, et al. WHO analysis of causes of maternal death: a systematic review. Lancet. 2006;367(9516):1066-74.

[4] Carroli G, Cuesta C, Abalos E, et al. Epidemiology of postpartum haemorrhage: a systematic review. Best Pract Res Clin Obstet Gynaecol. 2008;22(6):999-1012.

[5] Smith R. Parturition. N Engl J Med. 2007;356(3):271-83.

[6] Challis JR, Matthews SG, Gibb W, et al. Endocrine and paracrine regulation of birth at term and preterm. Endocr Rev. 2000;21(5):514-50.

[7] Fuchs AR, Fuchs F, Husslein P, et al. Oxytocin receptors in the human uterus during pregnancy and parturition. Am J Obstet Gynecol. 1984;150(6):734-41.

[8] Mitchell MD, Flint AP, Bibby J, et al. Rapid increases in plasma prostaglandin concentrations after vaginal examination and amniotomy. Br Med J. 1977;2(6094):1183-5.

[9] Buhimschi CS, Buhimschi IA. The role of the latent phase of labor in the genesis of intrauterine inflammation. Semin Perinatol. 2010;34(3):151-60.

[10] Phaneuf S, Rodriguez Linares B, TambyRaja RL, et al. Loss of myometrial oxytocin receptors during oxytocin-induced and oxytocin-augmented labour. J Reprod Fertil. 2000;120(1):91-7.

[11] Ledingham MA, Thomson AJ, Young A, et al. Changes in the expression of nitric oxide synthase in the human uterine cervix during pregnancy and parturition. Mol Hum Reprod. 2000;6(11):1041-8.

[12] Kelly AJ, Kavanagh J, Thomas J. Relaxin for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2013;(10):CD003103.

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