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ETIOPATHOGENESIS, CLINICAL COURSE AND MANAGEMENT TACTICS OF ATONIC BLEEDING IN WOMEN AFTER CHILDBIRTH

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ABSTRACT

Postpartum hemorrhage is considered a medical emergency and is one of the five leading causes of maternal death. Uterine atony and the risk factors leading to it are the main causes of bleeding after childbirth. In recent years, the rate of atonic bleeding has been increasing in many developed countries. Uterine atony is caused by an inadequate response of the smooth muscle fibers of the uterine body in response to endogenous oxytocin, which provides the contractile properties of the uterine muscle fibers. Normally, the contraction of the uterus after childbirth leads to compression of its spiral arteries, resulting in hemostasis, but as a result of changing in the muscle fibers of the spiral arteries and factors that prevent uterine contraction, uterine contraction does not occur or is insufficient after the third stage of labor, due to hypotonia, bleeding occurs from the spiral arteries, which leads to atonic type of bleeding after childbirth. This article briefly describes the factors that lead to atonic bleeding after childbirth, diagnostic criteria, management strategies and preventive measures.

Key words: pregnancy, bleeding, uterine atony, hypotonic bleeding, diagnosis of atonic bleeding, hemostasis.

INTRODUCTION

Postpartum hemorrhage (PPH) is one of the leading causes of maternal morbidity and mortality worldwide. Uterine atony, where the uterus fails to contract after delivery, is a significant contributor to PPH.

Global Impact: PPH is responsible for around 25% of maternal deaths globally, especially in low-resource settings [10]. The incidence of uterine atony is rising due to factors such as increasing rates of cesarean sections and multiple pregnancies[20].

Objectives: This article explores the pathophysiology, risk factors, clinical management, and emerging research on uterine atony as a major contributor to PPH.

Pathophysiology of Postpartum hemorrhage (PPH) and Uterine Atony

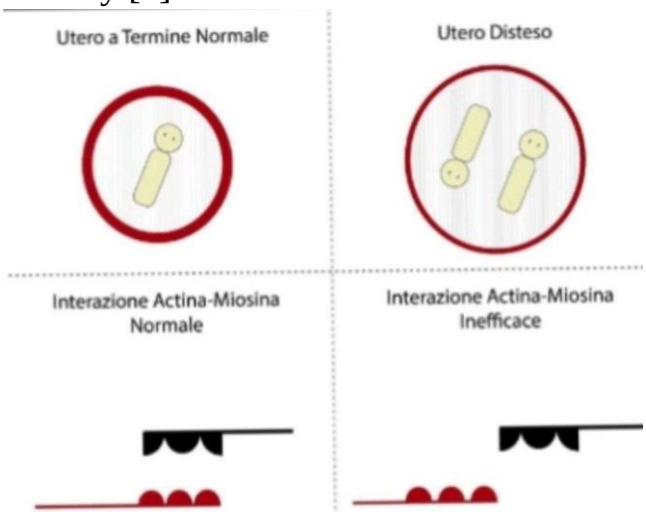
Postpartum hemorrhage (PPH) is a critical condition marked by excessive bleeding after childbirth. It is often caused by uterine atony, which occurs when the uterus fails to contract effectively after delivery. This condition disrupts the body's ability to control bleeding, particularly at the site where the placenta was attached. Understanding the pathophysiology of uterine atony and its role in PPH requires exploring the mechanisms of uterine muscle function, placental detachment, and coagulation.

Uterine Atony and its Role in PPH

Uterine atony occurs when the smooth muscle of the uterus fails to contract adequately after delivery, leading to uncontrolled bleeding. Normally, after the placenta is expelled, the uterus should contract to clamp down on the blood vessels at the placental site. This contraction is essential for hemostasis (the stopping of bleeding). However, when uterine atony occurs, the muscle fibers are unable to generate sufficient force, resulting in persistent bleeding from the placental site and other uterine blood vessels [7].

The failure of uterine contraction can be attributed to several factors:

- Overdistension: When the uterus is overstretched due to factors like multiple pregnancies (twins or triplets), polyhydramnios (excessive amniotic fluid), or a large fetus, the uterine muscles become fatigued and less capable of contracting effectively [2].



Overdistension and normal actin-miozin interaction (picture number 1) [15]

- Hormonal Imbalances: Oxytocin, a hormone crucial for uterine contractions, plays a significant role in the immediate postpartum period. Insufficient levels of oxytocin or the failure of the uterus to respond to it can impair uterine contraction and increase the risk of bleeding [9].
- Prolonged or Rapid Labor: Both prolonged labor (lasting many hours) and rapid labor (short and intense) can lead to uterine exhaustion. In both cases, the muscle fibers of the uterus fail to contract effectively, increasing the risk of atony [5]

Placental Detachment and Vascular Resistance

Following placental delivery, the uterus is left with exposed blood vessels that previously supplied the placenta. The hemostatic response involves the contraction of these blood vessels to limit bleeding. However, uterine atony impairs this ability to constrict blood vessels, resulting in uncontrollable hemorrhage. Under normal circumstances, as the uterus contracts, these vessels are compressed, reducing blood flow. Without proper contraction, bleeding from these vessels is uncontrolled, leading to excessive blood loss [22].

Coagulation and Blood Clotting in Uterine Atony

The body's response to bleeding also involves the coagulation cascade, a series of complex steps that form blood clots to stop further blood loss. However, in cases of uterine atony, the clotting mechanism is often overwhelmed. Prolonged bleeding within the uterus prevents the effective formation of clots, contributing to the severity of PPH [13]. This is particularly true when the uterine muscle is unable to compress the vessels and stop the flow of blood.

Inflammatory and Immune Response in Postpartum Hemorrhage

An often-overlooked aspect of the pathophysiology of PPH is the involvement of inflammatory and immune responses. Cytokines and inflammatory mediators play a role in the development of uterine atony and may promote the breakdown of uterine muscle fibers [11].For example, elevated levels of prostaglandins, which mediate inflammation and uterine smooth muscle contraction, can also contribute to an imbalance in uterine contractility during the postpartum period.

Risk Factors for Postpartum Hemorrhage and Uterine Atony

Maternal Risk Factors:

- Age: Advanced maternal age, particularly women over 35, have a higher risk for PPH [8].
- Multiple Gestation: Women carrying multiples are more likely to experience uterine overdistention, increasing the likelihood of uterine atony [19]

- History of PPH: A previous instance of postpartum hemorrhage can significantly increase the risk of recurrence in subsequent deliveries [21].
- Obesity: Higher body mass index (BMI) has been identified as a significant risk factor due to the impact on uterine tone [11].

Obstetric Risk Factors:

- Prolonged Labor: Prolonged labor, especially when coupled with the use of forceps or vacuum, can increase the risk of uterine atony [12].
- Cesarean Section: The increasing rate of cesarean sections has been associated with higher incidences of uterine atony due to scar tissue and abnormal uterine contractility [3].
- Placental Abnormalities: Conditions like placenta previa and accreta can increase the likelihood of PPH [17].

Independent factors favouring uterine atony (table number 1)

Intra-partum risk factors	Anamnestic risk factors
Prolonged or precipitous labour ; Induced labour; Uterine overdistension (for multiple pregnan-cy, polyhydramnios, fetal macrosomia); Presence of uterine fibroids; Chorionamniosite; Uterine relaxants such as deep anaesthesia (especially halogenat-ed anaesthetic agents) and magnesium sulphate; Inappropriate use of oxytocin; Manual removal of the placenta post-partum ;	Previous haemorrhage; Antepartum haemorrhage ; (placenta previa or placental abruption); Obesity (with Body Mass Index > 40); PreeclampsiaCongenital or acquired uterine abnormalities; White and Hispanic ethnicity;

Clinical Manifestations and Diagnosis of Postpartum Hemorrhage

Signs and Symptoms: Common manifestations include excessive vaginal bleeding (more than 500mL after vaginal birth or 1000mL after cesarean section), a soft and boggy uterus, and rapid maternal pulse [6]. Diagnosis is clinical, supported by the volume of blood loss and physical examination findings. Blood loss exceeding the defined thresholds should prompt immediate intervention. In some cases, uterine rupture or retained placental tissue may need to be ruled out through imaging or ultrasound [1].

Advanced Management Strategies for Postpartum Hemorrhage

Initial Management:

- Active Management of the Third Stage of Labor: Administration of oxytocin immediately after delivery of the baby reduces the risk of uterine atony by stimulating uterine contractions [14].
- Bimanual Compression and Massage: In cases where the uterus is soft and atonic, bimanual compression or uterine massage can help stimulate contractions and control bleeding [18].

Medical Interventions:

- Pharmacologic Agents: Oxytocin, misoprostol, and ergometrine are commonly used to enhance uterine contractions. Prostaglandins like carboprost and dinoprostone may also be used in resistant cases [4].

Pharmacologic agents used in uterine atony management include:

- *Oxytocin:* The International Federation of Gynecology and Obstetrics recommends giving oxytocin 10 international units (IU) IV or intramuscularly (IM) first-line for uterine atony if not administered prophylactically. The oxytocin hormone is naturally produced by the posterior pituitary and works rapidly, with an onset of action within 1 to 6 minutes, to cause uterine contraction following IV administration. Oxytocin has minimal adverse events and may be given during bimanual massage in response to hemorrhage.

- *Methylergonovine*: Ergot alkaloids (eg, ergometrine, ergonovine, and methylergonovine) are serotonergic receptor agonists and partial α-adrenergic receptor agonists that cause sustained uterine contractions. The onset of action is approximately 1 to 3 minutes. Methylergonovine 200 µg IM or IV is typically recommended but is relatively contraindicated in patients with hypertension.
- *Carboprost*: As a 15-methyl prostaglandin F2-α analog, carboprost acts on prostaglandin receptors to stimulate uterine contractions. The recommended dosage is 250 µg IM or intramyometrially every 15 to 90 minutes for a maximum of 8 doses, with peak serum concentrations reached in approximately 15 minutes. Carboprost is contraindicated in severe hepatic, renal, and cardiovascular disease and may cause bronchospasm in patients with asthma.

- *Misoprostol*: Misoprostol is a prostaglandin E1 analog with a more prolonged onset of action than other uterotonics, depending on the administration route, which includes oral, sublingual, rectal, or buccal routes. The analog should be avoided in patients with anticoagulant therapy or cardiovascular disease; adverse effects may include nausea, diarrhea, and fever.

- Tranexamic Acid: Antifibrinolytics like tranexamic acid have been shown to reduce blood loss by inhibiting fibrinolysis [23]. Tranexamic acid (TXA) is not uterotonic but inhibits fibrinolysis and is frequently used with uterotonic medications. The recommended dosage is 1 g of tranexamic acid IV over 10 minutes within 3 hours of delivery after a PPH diagnosis. TXA's onset of action is typically 5 minutes and is contraindicated in patients with a history of hypercoagulopathy

Surgical Interventions:

Should the medications fail with persisting excess bleeding, then surgical management is engaged.

Tamponade Techniques

1. Uterine packing with gauze (with vaginal packing to ensure its retention, thus a uterovaginal packing) with Foley catheter insertion to allow bladder drainage. The uterine packing should be tight and uniform, and it is a quickly and efficiently achieved with rolled gauze ribbons.
2. Bakri balloon (with vaginal packing to ensure its retention) with Foley catheter insertion to facilitate bladder drainage.

Surgical Management Techniques

1. Uterine curettage for retained products
2. Uterine artery ligation (O' Leary), with an option to for extending arterial ligation to tubo-ovarian vessels.
3. Compression sutures such as the B-Lynch are typically reserved for clinical scenarios where bimanual compression of the uterus leads to arrest in bleeding.
4. Hypogastric artery ligation.
5. Hysterectomy: In extreme cases where all conservative measures fail, a hysterectomy may be necessary to save the mother's life [15].

Conclusion

Atonic postpartum hemorrhage (PPH) remains one of the leading causes of maternal morbidity and mortality worldwide. Its pathophysiology, characterized by uterine atony and inadequate uterine contraction following childbirth, highlights the critical need for early recognition and intervention. Risk factors, including uterine overdistension, prolonged labor, and previous PPH, necessitate proactive

surveillance and management in high-risk patients. Effective management involves a stepwise approach, starting with uterine massage and pharmacological agents like oxytocin, and extending to surgical interventions in severe cases.

The advancement of management strategies, including the use of uterine tamponade devices and embolization, has significantly improved maternal outcomes. However, the variability in practice across different settings underlines the importance of institutional protocols, training, and team coordination. Additionally, emerging research continues to explore the molecular mechanisms underpinning uterine contractility and potential future treatments.

As clinical and technological advancements evolve, the role of healthcare providers in managing atonic PPH must be continually adapted. Multidisciplinary collaboration, research, and tailored treatment protocols are essential to improving maternal outcomes and reducing the global burden of postpartum hemorrhage.

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