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Review

# Postpartum hemorrhage: Clinical and radiologic aspects

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ABSTRACT

Postpartum hemorrhage (PPH) is a potentially life threatening condition, and it remains the leading cause of maternal morbidity. Uterine atony, lower genital tract lacerations, uterine rupture or inversion, retained products of conception and underlying coagulopathy are some of the common causes of PPH. Most conditions can be diagnosed based on clinical and laboratory evaluation supplemented by ultrasound information. Computed tomography (CT) or magnetic resonance (MR) imaging can provide information for the detection, localization and characterization of PPH in some difficult cases. CT can accurately demonstrate the anatomic location of significant arterial hemorrhage as sites of intravenous contrast material extravasation, which can be as a guide for angiographic intervention. The presence of focal or diffuse intravenous contrast extravasation or a hematoma within the enlarged postpartum uterine cavity on CT can help the diagnosis of uterine atony when the clinical diagnosis of uterine atony is unclear. CT can also provide the information of other alternative conditions such as a puerperal genital hematoma, uterine rupture and concealed hematoma in other sites. MR imaging may be considered as a valuable complement to ultrasound where the ultrasound findings are inconclusive in the diagnosis and differential diagnosis of retained products of conception. Knowledge of the various radiologic appearances of PPH and the correlation with clinical information can ensure correct diagnosis and appropriate and prompt treatment planning in the patients with PPH.



## 1. Introduction

Postpartum hemorrhage (PPH) is an obstetrical emergency that can follow vaginal or cesarean delivery. Ultrasound (US) is considered as the primary imaging modality for the assessment of patients with PPH as the modality is safe and inexpensive to perform and it can be routinely performed at the bedside. However, US may be limited by bowel gas and pain, and US also has difficulty to identify active hemorrhage [1–3].

Computed tomography (CT) or magnetic resonance (MR) imaging is not an appropriate first-line diagnostic procedure for the evaluation of PPH. CT and MR imaging can demonstrate the anatomic location and the extent of a concealed hematoma or a puerperal genital hematoma [1,4]. Unlike other modalities such as US and MR, contrast-enhanced CT can accurately demonstrate the anatomic location of a significant arterial hemorrhage as sites of intravenous contrast material extravasation. Demonstration of active arterial bleeding on contrast-enhanced CT may help interventional radiologists to perform a precise angiographic investigation and embolization of specific arteries [5–7].

In this article, we review the basic concepts of PPH including the etiology, hemodynamic changes in pregnancy, an overview of management, and the relevant pelvic vascular anatomy and CT anatomy. We also discuss the pathological conditions that cause PPH, particularly for primary, potentially life-threatening PPH. Briefly, we also illustrate the retained products of conception (RPOC), which is the most common cause of secondary or late PPH.

## 2. Basic concepts

### 2.1. Etiology

PPH is a major cause of maternal morbidity, with sequelae such as shock, renal failure, coagulopathy and adult respiratory distress syndrome. There is no single, satisfactory definition of PPH. An estimated blood loss in excess of 500 mL following a vaginal birth or a

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blood loss of greater than 1000 mL following cesarean birth often has been used for the diagnosis of PPH, but the average volume of blood lost at a routine delivery can approach these amounts [3,8].

PPH can also be defined as primary or secondary. Primary PPH occurs within 24 h after delivery (also called early PPH) and secondary PPH occurs 24 h to 12 weeks after delivery (also called late PPH). Primary PPH, which occurs in 4–6% of pregnancies, is caused by uterine atony in 80% or more of the cases. Other causes of primary PPH include lower genital tract lacerations, RPOC, uterine rupture or inversion and underlying coagulopathy. In contrast to primary PPH, secondary PPH is most commonly caused by RPOC. Other causes of secondary PPH include infection, placental site subinvolution and underlying coagulopathy [3,8].

### 2.2. Hemodynamic changes in pregnancy

The maternal blood volume increases markedly during pregnancy to fulfill the perfusion demands of the low resistant uteroplacental unit. Uterine atony or trauma to the genital region in pregnancy results in significantly more bleeding than would occur in the non-pregnant state due to an increased blood supply to these tissues.

It is well recognized that excessive obstetric hemorrhage may lead to disseminated intravascular coagulopathy (DIC) [9,10]. DIC is characterized by acute widespread activation of coagulation, the consumption of procoagulants and platelets, and intravascular deposition of fibrin, resulting in thromboembolic complications. Concomitant diffuse hemorrhage occurs due to an overwhelming consumption of platelets and coagulation factors [9,10].

Rarely, acute massive PPH in the peritoneal cavity or extraperitoneal cavity may lead to the development of abdominal compartment syndrome [11]. Abdominal compartment syndrome is defined as an acute rise in intraabdominal pressure with multiorgan dysfunction, which includes the cardiovascular, pulmonary, renal, gastrointestinal and hepatic systems. Reported CT findings of abdominal compartment syndrome include increased abdominal girth secondary to tense fluid accumulation in the retroperitoneum or peritoneum, a collapsed inferior vena cava (IVC), shock bowel, and a raised hemidiaphragm (Fig. 1) [12–14]. The most widely accepted method of measurement of intraabdominal pressure is measurement of the intravesical pressure. In patients with abdominal compartment syndrome, the intravesical pressure usually rises above 20 mmHg [12–14].

### 2.3. Overview of management

The treatment of PPH is centered on resuscitation of the patient and arrest of the bleeding. Initially, the administration of fluid and blood products is required in patients with PPH. Subsequently, more directed therapy including intravenous administration of uterotonics and uterine massage to promote uterine contraction in uterine atony, vaginal packing, removal of RPOC and surgical repair of lacerations should be performed [3,8,9]. If bleeding cannot be successfully controlled with conservative treatment, there are a number of other treatment options. In order of increasing invasiveness, the options include selective arterial embolization, ligation of the uterine or internal iliac arteries, and, as a last resort, a hysterectomy [15–19]. Surgical ligation of the uterine or internal iliac artery may not be effective to control PPH in 50% of patients due to an abundant collateral blood supply of the uterus [15]. Hysterectomy should be considered only when all conservative measures fail to achieve hemostasis following a life-threatening PPH. This procedure is associated with high morbidity and loss of subsequent fertility [16]. Transcatheter arterial embolization has emerged as a highly effective technique for controlling PPH. The rate of success is as high as 90%, with the added advantage of preserving fertility, few complications and avoidance of surgical exploration [17–19].

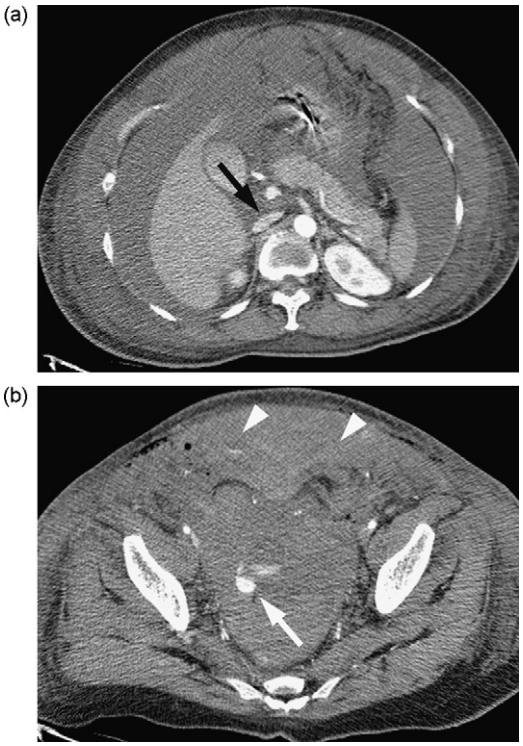


**Fig. 1.** Abdominal compartment syndrome in a 35-year-old woman as a complication of postpartum hemorrhage. (a) Contrast-enhanced CT image after a subtotal hysterectomy for the treatment of intractable postpartum hemorrhage at the level of the root of the superior mesenteric artery shows increased abdominal girth secondary to massive hemoperitoneum, compression of the inferior vena cava (arrow) and subcutaneous edema. (b) Contrast-enhanced CT image at the level of the pelvis shows intravenous contrast extravasation (arrow) in the pelvic peritoneal cavity, gas bubbles at the recent incision site, bilateral rectus sheath hematomas (arrowheads) and hemoperitoneum.

### 2.4. Vascular anatomy of the pelvis

The vascular supply of the uterus is primarily derived from the uterine artery, with a potential collateral supply from the ovarian artery. The uterine artery originates from the anterior division of the internal iliac artery. The uterine artery crosses over the ureter as it passes medially in the parametrium. In the parametrium, the uterine artery divides into a smaller descending vaginal branch (cervicovaginal artery) and a larger ascending branch, which tortuously ascends along the uterine margin at the medial edge of the broad ligament (Fig. 2). The ascending branch is divided into two terminal branches. The ovarian branch of the uterine artery forms anastomosis with the terminal branch of the ovarian artery, and the tubal branch makes its way through the mesosalpinx and supplies part of the uterine tube (Fig. 2) [20–23]. In the uterine wall, uterine arteries divide and run circumferentially as a group of anterior and posterior arcuate arteries between the outer and middle thirds of the myometrium. The radial arteries arise from the arcuate arteries and the radial arteries are directed toward the uterine cavity to become the spiral arteries in the endometrium [24,25]. The vaginal artery may arise either from the anterior trunk of the internal iliac artery or from the uterine artery. There are extensive anastomoses with the vaginal branches of the uterine artery [20–25].

The ovarian arteries originate from the aorta, just below the origin of the renal arteries. The ovarian arteries cross over the external or common iliac vessels as they approach the pelvic brim. The ovarian artery descends to the suspensory ligament of the ovary, and