

Case Report

Intravesical Hemorrhage as A Source of Postpartum Hemorrhage

Alicia N. Scribner and Alison L. Batig*

Departments of Obstetrics and Gynecology, Madigan Army Medical Center, USA

*Corresponding authors

Alison L. Batig, Department of Obstetrics and Gynecology, Madigan Army Medical Center, ATTN: MCHJ-CLG, Tacoma, Washington 98431, USA, Tel: (253) 968-1349; Fax: (253) 968-5508; E-mail: alison.l.batig.mil@mail.mil

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Abstract

Introduction: The genital tract is the most common source of acute blood loss in the initial postpartum period. Less commonly, non-genital sources of blood loss may contribute to postpartum blood loss and morbidity.

Case history: A reproductive-aged female at term underwent labor induction for severe pre-eclampsia. The active phase of labor was prolonged and the patient underwent cesarean delivery for labor dystocia. Following delivery, she experienced intravesical hemorrhage associated with anemia requiring blood transfusion and urinary retention that required continuous bladder irrigation and drainage for resolution.

Conclusion: Intravesical hemorrhage related to obstructed labor and surgical delivery can be a source of significant postpartum morbidity. When there is no evidence of a discrete urologic injury, underlying bleeding disorder, or ongoing hemorrhage, patients may be managed conservatively.

INTRODUCTION

Acute blood loss in the postpartum period is the primary cause of maternal death worldwide [1] and postpartum hemorrhage complicates 4-6% of births [2]. In most cases of postpartum hemorrhage, bleeding from the genital tract is the source of the blood loss. Uterine atony accounts for 80% of postpartum hemorrhage [2] and genital tract injury, uterine inversion or rupture, genital tract or retroperitoneal hematoma, bleeding disorders, abnormal placentation or incomplete removal of the placenta also contribute to postpartum hemorrhage [3]. When delivery is completed surgically, the surgical sites may contribute to blood loss. Assessing blood loss and acute anemia following childbirth generally involves focusing on the most common sites and etiologies of postpartum or post-operative hemorrhage.

Acute urinary retention is another potential complication in the postpartum period and may occur in 1.7-17.9% of deliveries [4]. Following cesarean delivery, 3.4-7.4% of women may demonstrate urinary retention [5,6]. Some risks factors for urinary retention include protracted labor [5,7], use of regional anesthesia [8], low body mass index [6], narcotic use [9], and instrumental delivery [10]. While the pathophysiology of postpartum urinary retention is not delineated, some propose that local changes such as periurethral and vulvar edema [11] and injury to the pelvic floor musculature and denervation resulting from the labor process and vaginal birth [12] may alter

bladder function. Untreated or inadequately treated urinary retention can cause bladder and detrusor overdistention and result in long-term micturition problems [13], so recognition of this complication in the early postpartum period is critical.

Although bleeding from the genital tract is the most common etiology of hemorrhage in the postpartum period, bleeding from non-genital sources such as the urologic tract may be a less-commonly recognized source of significant blood loss. Additionally, bleeding from the urologic tract may contribute to retention or inadequate bladder drainage with standard bladder catheterization.

CASE PRESENTATION

A 30-year-old G1 at 38 weeks estimated gestational age presented for a routine prenatal visit with new-onset severe-range blood pressures. The patient's reported medical history included generalized anxiety disorder but her pregnancy was otherwise uncomplicated. At the time of presentation, she denied neurologic or abdominal symptoms to suggest pre-eclampsia and was referred for immediate assessment and blood pressure monitoring on the Labor and Delivery ward.

Additional assessment demonstrated a reactive fetal heart rate tracing with a normal-range baseline, persistently elevated severe-range blood pressures and a spot urine creatinine ratio of 0.536. The patient's liver transaminases, creatinine, hemoglobin,

hematocrit, and platelets were within normal parameters for pregnancy (aspartate aminotransferase 25 U/L, alanine aminotransferase 19 U/L, creatinine 0.68mg/dL, hemoglobin 12.7 g/dL, hematocrit 38.1%, platelets $173 \times 10^3/\text{mL}$). Pre-eclampsia with severe features was diagnosed based on the patient's persistently elevated severe-range blood pressure. Her blood pressure was treated with intravenous labetalol and admission for labor induction advised.

Upon admission, her cervix was 2cm dilated, 50% effaced, and the fetal head was at -3 station. Labor induction started with oxytocin and magnesium sulfate was administered for maternal neuroprophylaxis. She requested and received epidural anesthesia while still in latent labor. She entered active labor approximately 18 hours later following continued oxytocin administration and amniorrhexis. Her blood pressures remained elevated with sporadic severe-range blood pressures, but required no further antihypertensive therapy. As the patient progressed through latent labor, her creatinine increased, first to 0.71 mg/dL, then to 1.32 mg/dL. Concurrently, the patient's urine output remained $>0.5\text{cc/kg/hr}$ and was clear in appearance. Her liver transaminases, platelets, hemoglobin, and hematocrit remained stable. After progressing to 7-8cm cervical dilation, the patient's active phase became protracted, her urine output decreased below 0.5cc/kg/hr , and her creatinine rose to 1.57 mg/dL. An intrauterine pressure catheter was placed, the oxytocin gradually increased to obtain adequate contractions and the patient was monitored for labor progress. Over the course of the next 8-9 hours, adequate contractions were achieved but these failed to result in labor progression. Simultaneously, fluid resuscitation with albumin and crystalloid fluid failed to improve her urine output. The patient underwent primary cesarean delivery for arrest of dilation at 8cm cervical dilation.

Surgical delivery of the infant was remarkable only for poor tone of the uterus immediately after delivery, which resolved with oxytocin administration, and a small, non-evolving hematoma in the left broad ligament. The patient's epidural was used for anesthesia after re-dosing with fentanyl and lidocaine prior to the procedure. A long-acting narcotic was also used in the epidural to assist with post-operative pain relief. The patient's preoperative hematocrit was 39%. The estimated intraoperative blood loss was 800cc based on the amount of blood collected in the operative suction canisters and the amount of blood visualized in the surgical field. During the procedure, the patient's urine output was 200cc and no frank blood was appreciated in the collected urine. The operative time was 47 minutes. The patient's magnesium sulfate was discontinued during the procedure but resumed upon its completion.

Shortly after leaving the operating room, the patient developed frank hematuria and urine output averaged 0.48cc/kg/hr in the first 3 hours after surgery despite a mild improvement in her creatinine to 1.27 mg/dL. Approximately 4 hours after surgery, her hematocrit was 30.4%. Over the course of the next 4-5 hours, the output in the Foley catheter increased to just under 400cc/hr (approximately 4.8cc/kg/hr) but frank hematuria persisted, prompting concern for an unrecognized urologic injury. The output appeared frankly bloody and we were unclear how much of the output represented urine versus blood

loss. Attempts to assess for a urologic injury using cystoscopy were unsuccessful due to the significant amount of obscuring blood, despite multiple attempts to flush out the blood and clot through the cystoscope. Therefore, a computerized tomography (CT) cystogram was performed to assess urologic integrity. The CT demonstrated no evidence of intra-abdominal bleeding or urologic injury, but there were findings consistent with a large intravesical thrombus (~8cm). The uterus and pelvis had an unremarkable postpartum/postsurgical appearance with no suggestion of significant intra-abdominal bleeding or hematoma formation outside of the bladder space (Figure 1). The output from the patient's bladder remained frankly bloody and the Foley catheter repetitively clogged with thrombi, which prevented adequate bladder drainage despite multiple attempts to flush out the clots and several replacements of the catheter. The patient's fibrinogen and coagulation studies remained within normal limits. Her blood pressures in the postpartum period remained normotensive or mildly elevated. About 18 hours after surgery and with ongoing inability to keep the bladder drained via catheterization secondary to recurrent thrombi in the tubing, the Foley catheter was removed. The patient was able to void soon after the catheter was removed, so ongoing management included serial assessment of her hematocrit and voiding function. She was able to void 100-200cc hourly initially. Her urine remained grossly bloody and we were unable to quantify how much of her voided output was urine or frank blood. Twenty-four hours after delivery, her magnesium sulfate was discontinued and a repeat hematocrit performed which demonstrated a hematocrit decrease to 21%. Her creatinine continued to normalize (0.99 mg/dL). The quantity of lochia observed was assessed as normal and there was no clinical evidence to suggest excessive blood loss from the genital tract. Approximately 6 hours after the Foley catheter was removed, the patient reported voiding difficulty and suprapubic pain. An ultrasound assessment of her bladder suggested urinary retention and the Foley catheter was replaced with 1300cc of frankly bloody urine output. At this point, her hematocrit was reassessed and had fallen to 19.9%. Her Foley became clogged with thrombus again and could not be flushed and cleared to allow drainage despite repeated attempts. Due to ongoing concern for intravesical hemorrhage and inability to maintain adequate bladder drainage by either spontaneous voiding or catheter drainage, we proceeded with a transfusion of packed red blood cells and consulted Urology for further evaluation and assistance with management.

The consulting Urologist agreed there was substantial clot burden in the bladder. The bladder was hand-irrigated with high-volume irrigant and removal of significant quantities of clot. A continuous bladder irrigation (CBI) catheter was placed. Over the next 24 hours, the bladder was continuously irrigated using the CBI catheter with rapid resolution of the gross hematuria. Her hematocrit improved to 27.2% following transfusion. The catheter was removed after 24 hours and the patient was able to void spontaneously without difficulty and with no evidence of urinary retention. No further gross hematuria was observed during the course of her admission.

Approximately 6 weeks after delivery, the patient underwent office cystoscopy, retrograde cystogram, and re-evaluation in the Urology Clinic with no abnormal findings and no recurrence of the hematuria (Figure 2).

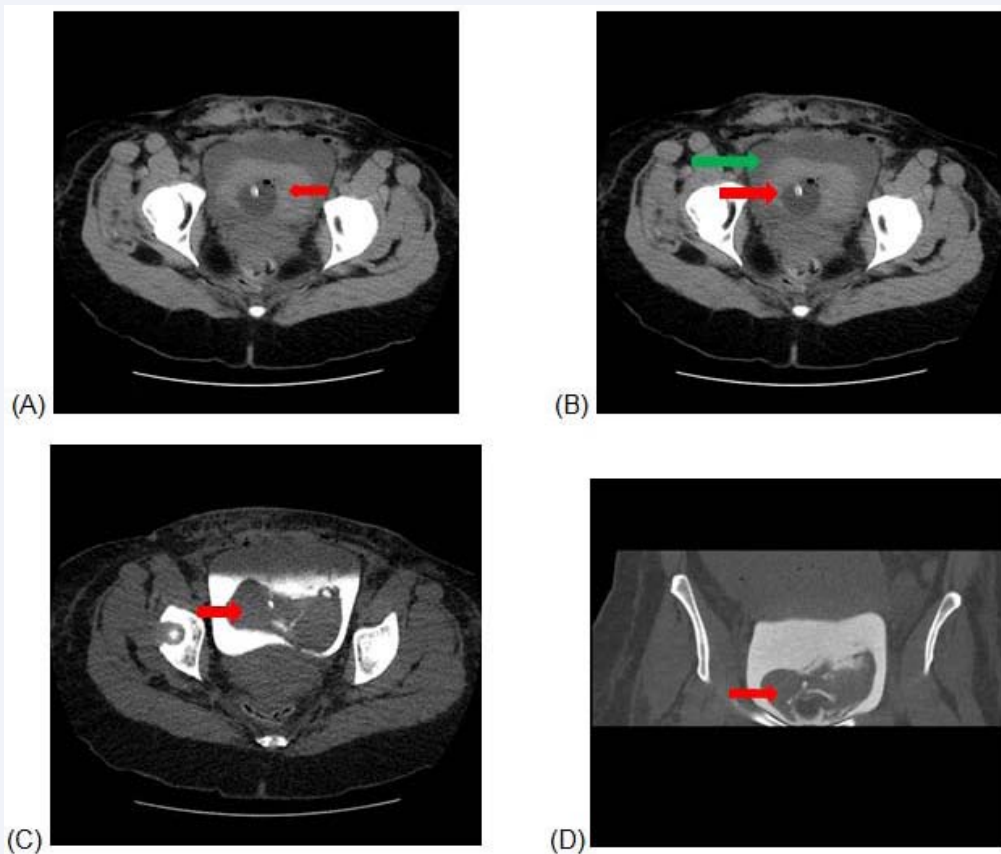


Figure 1 (A) Axial unenhanced CT image demonstrating a hyper-attenuated mass (red arrow) compatible with a thrombus within the bladder surrounding the Foley catheter. (B) Sagittal reformatted CT image without contrast demonstrating a hyperdense mass (red arrow) within the bladder consistent with a thrombus. An enlarged, postpartum uterus (green arrow) with air and fluid in the endometrial cavity is also seen. (C) Following instillation of contrast into the bladder, a filling defect (red arrow) consistent with a thrombus is demonstrated and no contrast is observed outside the bladder. (D) Coronal reformatted image following instillation of contrast into the bladder demonstrating the thrombus (red arrow) as large filling defect.



Figure 2 Retrograde cystogram completed 6 weeks after delivery demonstrating an intact bladder (red arrow) without filling defect.

DISCUSSION

Transient hematuria observed during labor and after vaginal birth or cesarean delivery, especially in cases of prolonged or obstructed labor, is usually self-limited and likely related to diffuse local trauma to the bladder. In the case we describe, we believe an intravesical hemorrhage occurred and contributed significantly to the patient's postpartum anemia and subsequent need for blood transfusion, as well as her urinary retention. The patient's protracted labor likely resulted in diffuse local trauma to the bladder and perivesical space during the course of labor and may have predisposed to acute injury of the bladder tissue at the time of her surgical birth. The visible presence of blood in the Foley catheter immediately after surgery suggests the blood loss from the bladder started in relation to the surgical delivery. We presume this injury and the bleeding that resulted was diffuse and non-localized and we have no indication to believe the bleeding came from a higher level in the urologic tract, although that is possible. Diffuse bleeding from the injured vesical tissue likely continued during the patient's initial recovery and lead to formation of a large thrombus in the bladder that was evident on CT cystogram and cystoscopy. The size of the thrombus and the amount of blood seen in the patient's catheter, along with the timing of the corresponding decrease in her hematocrit values imply the blood loss from the bladder was a significant contributor to the resulting anemia. Additionally, acute blood loss from the bladder contributed to the observation of increased urine output after surgery. In actuality, the observed increase in output may have resulted mostly from drainage of intravesical blood instead of an actual increase in urine production. Her improved serum creatinine following delivery suggests the renal function and urinary output did recover, however.

In addition to anemia, this patient's intravesical hemorrhage was associated with urinary retention that failed to resolve with simple bladder catheterization. The patient had multiple risk factors for postpartum urinary retention, including obstructed labor, surgical delivery, and narcotic use [14]. While we cannot delineate a causative association between the thrombus formation and acute urinary retention, we surmise this contributed to the presentation and may also explain why basic bladder drainage failed to resolve the problem and why urinary retention recurred after the initial Foley catheter was removed. Possible mechanisms for this include mechanical obstruction of the bladder neck with the thrombus acting as a ball-valve once the Foley catheter was removed, mechanical obstruction of the catheter and urethra by clot and local irritation of bladder urothelium from blood.

After initial evaluation failed to demonstrate a discrete urologic injury or correctable underlying bleeding disorder as a potential etiology, this case was effectively and conservatively managed with bladder irrigation and placement of a CBI catheter. It appears this intervention, along with the additional 24 hours of recovery time during which the catheter was placed, assisted with clearing the bladder space of blood and clot and allowed adequate bladder drainage. In the event the hematuria persisted despite this intervention, further assessment for urologic injury

or abnormality, such as malignancy, arteriovenous malformation, or undetected urologic injury, would have been considered.

Significant postoperative anemia after vaginal or surgical childbirth is generally associated with blood loss from the genital tract [15]. However, non-genital sources of blood loss can occur in relation to delivery and result in significant morbidity. While usually self-limited and of nominal clinical impact, intravesical bleeding can be a source of postpartum hemorrhage and requires prompt attention and management. Beyond acute anemia and its clinical implication, intravesical hemorrhage may be associated with other morbidity, such as acute urinary retention. When significant clot burden is present in the intravesical space, traditional Foley catheter placement may fail to decompress the bladder and evacuate the thrombus. In the case we describe, after evaluating for a discrete urologic injury as the etiology of the patient's presentation, conservative management in collaboration with the consulting Urologist, supportive care, and continued surveillance resulted in resolution.

DISCLAIMER

The views expressed are those of the author(s) and do not reflect the official policy of the Department of the Army, the Department of Defense or the U.S. Government.

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