International Surgery Journal
Wang P et al. Int Surg J. 2015 Nov;2(4):756-760
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20151123

Case Report

Abdominal compartment syndrome resulting from postpartum hematoma of wall: a rare occurrence associated with a high risk of mortality

Ping Wang[1], Zhong Jia[1]*, Zhihua Wang[2], Changwen Liu[1], Jie Liu[2], Yanhui Zhu[3]

[1]Department of Hepatobiliary Surgery, Hangzhou First People's Hospital, Zhejiang, Hangzhou, China
[2]Department of Gynaecology and Obstetrics, Hangzhou First People's Hospital, Zhejiang, Hangzhou, China
[3]Department of the First School of Clinical Medicine, Nanjing Medical University, Jiangsu, Nanjing, China

Received: 17 July 2015
Accepted: 19 August 2015

***Correspondence:**
Dr. Zhong Jia,
E-mail: jiazhong20058@hotmail.com

ABSTRACT

Objective of current study was to analyze and summarize the clinical features of postpartum abdominal hemorrhage with Abdominal Compartment Syndrome (ACS) to improve its diagnosis and treatment. A retrospective analysis of the clinical data of four cases of postpartum abdominal hemorrhage with ACS treated in our hospital from 2009 to 2014. Postpartum ACS is characterized by excessive blood loss. Symptoms mainly included oliguria, abdominal distention, shortness of breath, progressive decrease in hemoglobin and hematocrit, coagulation disorders, large volumes of ascites on computed tomography and B-type ultrasonography, and significant increases in intra-abdominal pressure. All four patients underwent surgical decompression and hemostasis, including one case of intestinal necrosis caused by ACS. Two patients fully recovered, while two patients died: one due to acute respiratory distress syndrome and one from lupus encephalopathy. Postpartum hemorrhage can lead to abdominal ACS, which is life-threatening. Physicians should attach great importance to timely diagnosis of this phenomena and early surgical decompression is key to successful treatment.

Keywords: Postnatal, Abdominal compartment syndrome, Abdominal hemorrhage

INTRODUCTION

Abdominal Compartment Syndrome (ACS) refers to induced intra-abdominal hypertension, which is due of various causes that lead to cardiovascular, pulmonary, renal, celiac viscera, abdominal wall, and brain dysfunction or organ failure. The clinical manifestations of intra-abdominal hypertension (i.e., severe abdominal distension, oliguria, respiratory distress, etc.) are prognostic of serious illness.[1]

As awareness has increased, the rate of ACS-associated mortality has decreased from almost 100% to 20%-50%.[2]

Although not common, prenatal ACS is a typical manifestation of pregnancy-induced hypertension syndrome resulting from polyhydramnios and elevated intrauterine pressure during amnioinfusion.[3]

However, postpartum ACS due to postpartum uterine pressure relief is extremely rare and thus is often overlooked. Here, we retrospectively analyzed the clinical data of four patients diagnosed with ACS who were admitted to our hospital between September 2009 and November 2014 and discuss the diagnosis and treatment of these cases.

CASE REPORT

Case 1

A 22-year-old woman (gravida 1, para 0) at gestational week 32+ with fetal cephalic presentation in the left occipito-anterior presentation was admitted on December 14, 2009 because of acute fatty liver, liver failure, Disseminated Intravascular Coagulation (DIC), hypoalbuminemia, metabolic acidosis, and fetal intrauterine distress. Emergency cesarean section was performed and the patient was admitted to our Intensive Care Unit (ICU) for observation. Postoperatively, urine production and hemoglobin (HB) concentration gradually decreased. On postoperative day 6, the patient experienced sudden onset of periumbilical pain with obvious umbilical distention due to a large solid 20 x 18 x 15 cm mass with a clear boundary, smooth surface, medium texture, and significant tension. Her abdomen resembled a state of pregnancy with signs and symptoms of abdominal distension, with a decrease in HB to 52 g/L and no urine production. B-type ultrasonography revealed a large celiac hematoma, 19 x 18 x 14 cm in size with possible origin in the abdominal wall with bladder restriction, a small amount of dark liquid in the pelvic area, the umbilical plexus located in the uterus, and a small amount of liquid in the uterine cavity with an uneven echo. On postoperative day 7, abdominal Computed Tomography (CT) with contrast revealed no significant expansion of the rectum or sigmoid colon. The small intestine was pushed to one side by the abdominal wall mass and there was a clear gap between the uterus and mass (Figure 1).

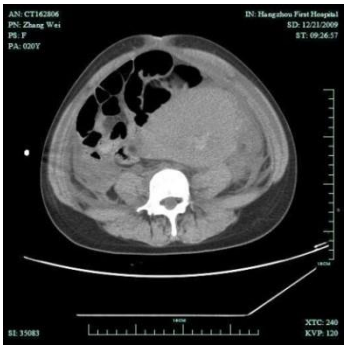

Figure 1: The small intestine was pushed to one side and a clear gap between the uterine wall and mass was observed.

The bladder was encroached by the mass, which was located outside the parietal peritoneum and continuous (Figure 2), thus we arrived at a preliminary diagnosis of a hematoma of the abdominal wall. After close observation and active treatment, the patient's HB level continued to progressively decrease to 44 g/L, erythrocyte press 0.125, and serum creatinine increased to 183 µM. On emergency laparotomy, a hematoma was observed between the parietal peritoneum and fascia, superior to the umbilical plexus, inferior to the pubic symphysis, with a clear condensing blood clot of about 2300 mL. No bleeding was observed on the uterine surface, bladder surface, or peritoneum, while the muscle layer widely exuded blood, although there was no visible vascular hemorrhage. The bleeding eventually stopped completely, postoperative liver/kidney function gradually improved, and the patient was finally discharged on January 12, 2010.

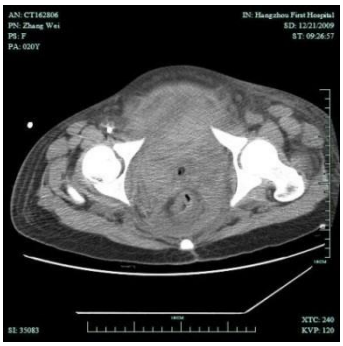

Figure 2: The bladder was compressed by a mass.

Case 2

A 35-year-old woman (gravida 2, para 2) at gestational week 41 was admitted to our hospital because of a 10 h history of postpartum vaginal hemorrhage on April 2, 2011. The night before admission, our patient gave birth at a local hospital and experienced vaginal hemorrhage for 10 min, thus total resection of the uterus was performed. Postoperatively, vaginal bleeding continued, thus a total hysterectomy and left salpingectomy were performed. Afterward, anuria, whole body swelling, and postpartum vaginal bleeding of about 8000 mL were noted. Hence, the patient returned to our hospital for quasi pregnancy, a 2 week history of eutocia, postpartum hemorrhage, uncontrolled hemorrhagic shock, and acute renal failure, with decompensated metabolic acidosis and amniotic fluid embolism.

On admission, the patient was in a shallow coma, with a Glasgow coma scale score of 6 points, and was placed on a respirator in intermittent positive-pressure ventilation mode. Findings of an initial examination were as follows: Heart Rate (HR), XX beats/min; blood pressure, 92/42 mmHg (maintenance intravenous dopamine at 5 µg/kg/h); HB, 84 g/L; Prothrombin Time (PT), 35.7 s; and serum albumin (ALB), 25.0 g/L. The patient produced no urine and had increased levels of serum creatinine, myocardial enzymes, liver enzymes, blood bilirubin, and amylase due to hematuria, with obvious multiple organ dysfunction syndrome. On the night of April 3 (postadmission day 1), the patient's HR increased to 170–200 beats/min and her blood pressure decreased, which was maintained at 90/60 mmHg by intravenous administration of dopamine at 10 µg/min/kg. Abdominal distention was obvious. Her laboratory results were as follows: HB, 55 g/L; PLT, 31 x 109/L; PT, 46.9 s; and activated partial thromboplastin time, unmeasurable. Abdominal B-type ultrasonography indicated decreased effusion (abdominal blood clot not

excluded). We immediately started blood transfusion, which improved HB to 75 g/L and PLT to 26 x 109/L. On April 4, from 08:00 h to 16:30 h, a drainage tube placed in the pelvic cavity collected 2350 mL of fresh blood and drained the pelvic fluid which contained venous blood components. Abdominal B-type ultrasonography revealed dark liquid in the abdominal cavity of a depth of about 8.3 cm with celiac scattered flocculent or dot echo of about 14.0 x 10.0 cm, which indicated active bleeding. On the night of April 4, we observed a perirenal abscess with a hematoma of the abdominal wall, and thus performed surgery to remove the hematoma and maintain intraperitoneal hemostasis, small intestinal resection, and exteriorization. Intraoperatively, about 100 mL of free blood from the abdominal wall and a blood clot of about 200 mL were collected. The peritoneal cavity was opened and about 1200 mL of light hemorrhagic liquid and a blood clot of 100 mL were collected. From the back of the blind compartment, about 40-80 cm of the visible segments of the intestine were necrotic, thus about 40 cm were resected (Figure 3). We arrived at a postoperative diagnosis of postpartum hemorrhage, hemorrhagic shock, multiple organ failure, large abdominal hematoma formation, a small necrotic intestinal segment, amniotic fluid embolism, and anemia. Postoperatively, the patient remained in a coma, developed marked edema of the extremities and decreased oxygen saturation, fluctuating from 71% to 90%. Chest radiography revealed reduction in right pleural effusion. Taking into account the extended period of ischemia, hypoxia-induced pulmonary shock, and an oxygenation index of only 35, indicated changes consistent with Acute Respiratory Distress Syndrome (ARDS). In vitro membrane lung oxygenation therapy was initiated. On April 5 (postadmission day 3), her HR decreased to 62 beats/min, oxygen saturation decreased to 60%, and blood pressure fell to 79/24 mmHg, while mydriasis, light reflex (-), and deep coma developed. We immediately performed rescue measures, administered drugs, performed chest compression, and open chest heart massage to prevent irreversible brain damage. Nonetheless, the patient's family demanded immediate discharge.

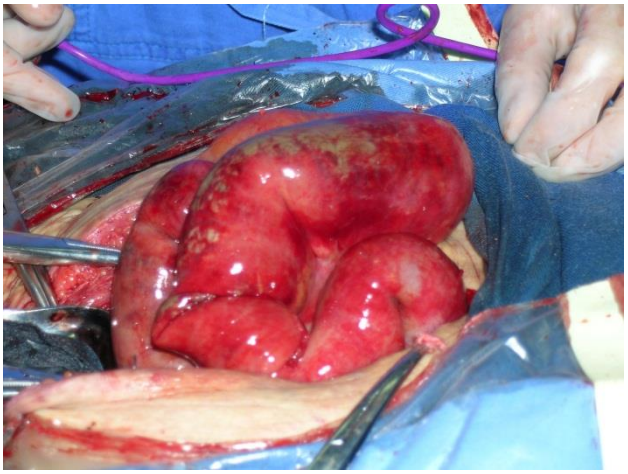

Figure 3: Segmental intestinal hyperemia necrosis.

Case 3

A 39-year-old female (gravida 3, para 2) at gestational week 38+ was admitted to our emergency department because of a sudden drop in blood pressure more than 9 h after a second cesarean section on September 2, 2011. On admission, laboratory results were as follows: BP, 92/47 mmHg; HR, 101 beats/min; HB, 81 g/L; PLT, 63 x 109/L; potassium, 2.88 mM; ALB, 28 g/L; PT, 13.8 s; and Fasting Blood Glucose (FBG), 2.28 g/L. Lung CT revealed bronchitis with infection. We arrived at the following diagnoses: dystocia with a live infant, possible amniotic fluid embolism, possible DIC, scarring of the uterus, hypoalbuminemia, hypokalemia, bronchial pneumonia, or possible intrauterine infection.

On September 3 (postoperative day 2), the patient developed abdominal distension and rapid breathing. Her laboratory findings were as follows: HR, 85 beats/min; BP, 120/75 mmHg; hematuria; HB, 63 g/L; PLT, 86 x 109/L; C-reactive protein, >160 mg/L; PT, 13.0 s; and FBG, 5.06 g/L. B-type ultrasonography revealed heterogenic echoes of a pelvic mass of about 9.3 x 8.4 cm. Hepatic and renal clearance was decreased and there was a small amount of dark liquid around a visible mass of about 4.7 x 2.7 cm. The area between the lower front of the muscular layer of the uterus and bladder was not homogeneous, with an echo of the visible range that revealed several small masses of about 13.2 x 8.2 x 10.1 cm collectively, surrounding a visible flocculent echo and a small amount of liquid. The pelvic uterus was visible behind a small amount of liquid in dark areas with a small amount of liquid in the left thorax, also depicted by dark areas. We made a preliminary diagnosis of an abdominal hematoma and performed an abdominal CT, which revealed bladder endometriosis, a dimpled area, formation of a large hematoma, increasing abdominal bowel pressure, expansion of the ascending and transverse colon, gas accumulation, a gas‑liquid interface, and an altered bladder position. Intra-Abdominal Pressure (IAP) increased from 16 to 26 cmH_2O and central venous pressure was 17 mmHg. HB significantly decreased consistent with blood loss. We prioritized the large abdominal hematoma and the abdominal pressure of >20 cmH_2O. CT showed obvious caval flatulence and a left-shift of the bladder. Surgical hemostasis of the hematoma was considered incomplete causing partial tissue damage.

On September 5 (postoperative day 5) pelvic probing under general anesthesia revealed obvious bladder edema. An incision to the uterine wall was made between the bladder, vagina, and urethra, which contained a large hematoma. Both sides of the peritoneum extended to the pelvis and pelvic congestion was clear with bilateral purulent exudate. A blood clot of about 700 mL was removed between the bladder and uterus. Postoperatively, the patient gradually recovered and was finally discharged on September 19.

Case 4

A 33-year-old female was admitted to our ICU on September 5, 2014 because of a 2 day history of slurred speech, a 10 year history of systemic lupus erythematosus that was treated with prednisone, and complications associated with cesarean section. On September 10 (postoperative day 5), the patient suddenly developed chest tightness and abdominal distension. Laboratory test rests showed decreased serum HB levels. B-type ultrasonography and CT revealed pelvic hematoma, thus we were concerned with bleeding. On September 11 (postoperative day 6), emergency laparotomy revealed a large hematoma between the fascia and peritoneum, which was resected and free blood was collected. Intraoperatively, the blood clots were clear and 1000 mL of blood was collected. Postoperative CT showed that the size of the retroperitoneal hematoma decreased. On September 12 (postoperative day 7), the patient developed mydriasis, light reflex (-), and deep coma, thus we implemented immediate rescue measures. A head CT revealed intracranial hemorrhage and herniation. However, the patient's family demanded immediate discharge.

DISCUSSION

In 1984, Kron coined the term ACS. Although critical cases of ACS are relatively rare, subsequent research has shown that the incidence of severe trauma patients with ACS is 2%-15%, while the incidence of ACS among patients in the ICU is reportedly about 5%.[4] In 2007, the World Society of the Abdominal Compartment Syndrome (WSACS) held their third international meeting, during which Intra-Abdominal Hypertension (IAH) and ACS were redefined, and classification schemes were formulated. The WSACS standardized an evidence-based method to accurately monitor IAP for use in clinical diagnosis and treatment guidelines as follows: Abdominal Perfusion Pressure (APP) = mean arterial pressure MAP - IAP, with ASC defined as an APP of <60 mmHg accompanied by incomplete failure of organ function.[4]

ACS is not uncommon in obstetrics, as it often occurs due to pregnancy-induced hypertension syndrome, acute polyhydramnios, multiple pregnancies, and twin-to-twin transfusion syndrome,[3] which all appear during the prenatal period. In this article, we described three cases of ACS that occurred postpartum, which is an extremely rare event.

For the first patient, we performed emergency cesarean section 6 days after the appearance of abdominal pain, significant abdominal distention, and a large protrusion on the navel, accompanied by a progressive decrease in serum HB level, no urine production, kidney function exhaustion, and IAP of >35.0 mmHg. In this case, B-type ultrasonography and CT confirmed a hematoma originating from the abdominal wall that restricted the intestinal canal and bladder. ACS caused by peritoneal external hematoma is very rare, as the only other case was noted in a hospital in western Pennsylvania, USA.[5] In this case, the patient developed ACS, which may have been associated with the following factors: (i) the specific position of the hematoma, as the mass in this patient was located in the parietal peritoneum and muscle layer, resulting from the muscular layer relative to the stronger peritoneum during pregnancy, thus the abdominal mass decreased the abdominal cavity resulting in increased celiac pressure; (ii) the size of the hematoma relative to the abdominal wall, as the large hematoma occupied almost the entire abdomen, thereby restricting abdominal compliance and capacity, causing the abdominal cavity pressure to further increase;[5] (iii) tension of the abdominal wall hematoma because the muscle layer forces blood flow, thereby increasing hematoma tension unceasingly, which can also increase celiac pressure; and (iv) poor liver function, which is the root of blood coagulation disorders and, along with synthesis of clotting factors and insufficient thrombocytopenia in combination with other related factors, forces the muscle layer, peritoneum, and viscera surface to exude blood, resulting in anemia, inadequate intestinal microcirculation perfusion, bowel dysfunction, gut cavity pneumatosis, merging of the hematoma with the mesenteric vein, and restriction of the portal vein due to intestinal edema, which further elevates IAP. These events form a vicious circle in a situation similar to ACS caused by retroperitoneal hematoma.

Oliguria progression to anuria and no reaction to the expansion of prerenal azotemia are characteristic of renal insufficiency caused by ACS. When IAP is 15~20 mmHg (1 mmHg = 0.133 kPa), oliguria can occur. When IAP is increased to 30 mmHg or higher, anuria occurs. Thus, such expansion negates the effects of dopamine and loop diuretics.[1] Large hematomas can inhibit urine production, thus resection of the hematoma may lead to recovery of renal function.

Our second patient developed a massive hemorrhage after eutocia and after surgery, no urine was produced, kidney function was exhausted, and alterations in consciousness appeared. B-type ultrasonography revealed intraperitoneal hemorrhage, which resulted in the development of ARDS.

Acute increases in internal pressure may lead to hypoxia and hypercapnia, which are characteristic of respiratory failure. The diaphragm may lead to static and dynamic decreases in lung compliance. IAP can also necessitate total lung ventilation, resulting in decreased functional residual capacity and residual volume, imbalances in air flow ratio, and insufficient ventilation, which is caused by hypoxia and hypercapnia. Decreased alveolar oxygen tension and increased chest internal pressure can increase pulmonary vascular resistance.[1] Recent studies have shown that abdominal bleeding and rehydration can increase pressure in the abdominal cavity caused by high arterial and coronary pressure, leading to deterioration in

lung function. In such cases, ARDS may be a consequence of ACS,[6] which, in turn, increases intracranial pressure and reduces cerebral perfusion pressure, resulting in disrupted consciousness.

In addition to reduced arterial blood flow, increased IAP also directly restricts the mesenteric and portal veins, resulting in increased venous pressure and bowel edema. Visceral edema further elevates IAP, thus leading to a vicious circle, so that gastrointestinal blood perfusion decreases, further promoting tissue ischemia. In cases in which IAP continues to increase may lead to necrosis of the intestines, especially the ileum and right colon.[1] Necrosis often appears at about 40-80 cm from the ileocecal sphincter.

In our third case, 2 h after the second cesarean section, the patient developed shortness of breath, chest tightness, hypotension, and coagulation dysregulation. Conservative treatment had no effect, while urine production was compromised and hematuria occurred. B-type ultrasonography and CT confirmed a large hematoma of the bladder uterine lacunar, which restricted the bladder and intestinal canal. IAP increased to 26 cmH_2O and central venous pressure increased to 17 mmHg. The intraperitoneal hematoma was believed to be caused by incomplete intraoperative hemostasis or local tissue injury, which progressed to ACS.

Increases in internal pressure can decrease cardiac output because of direct compression of the inferior vena cava and portal vein, resulting in decreased blood flow with simultaneous increased pressure on the chest and further reduction in blood flow through the inferior vena cava. An increase in pleural pressure increases cardiac compression and decreases end-diastolic ventricular volume.[1] IAH can obviously increase after cardiac load. Thus, IAP may increase up to 26 cmH_2O with a simultaneous increase in central venous pressure to 17 mmHg, which can lead to reduced cardiac output and increased vicarious HR.

To sum up, the compliance of the abdominal wall during pregnancy is usually good. However, although postpartum ACS is relatively rare, it is easily overlooked, which can lead to illness if treatment is delayed, and even threaten the fetus. Therefore, physicians should be aware of postpartum ACS, especially under the following circumstances: (i) uterine surgery; (ii) primary symptoms of oliguria, abdominal distension, and wheezing; (iii) excessive postoperative blood loss; (iv) detection of a large hematoma of large accumulation of fluid in the abdomen by B-type ultrasonography ultrasound or CT; and (v) continued increase in IAP. When conservative treatment is deemed poor or insufficient, and abdominal cavity pressure reaches the standard for diagnosis of ACS, prompt surgery is the only treatment choice. For such cases, surgery should include: (i) clear hematoma or effusion to reduce abdominal pressure; (ii) thorough evaluation of hemostasis and removal of the pressure source; and (iii) probe and address secondary damage caused by ACS, such as intestinal necrosis.

In conclusion, postpartum abdominal bleeding can cause ACS and compromise survival; therefore, physicians should pay great attention to and improve their understanding of this phenomena, as timely diagnosis and positive early surgical decompression are key to successful treatment outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Malbrain ML, Cheatham ML. Definitions and pathophysiological implications of intra-abdominal hypertension and abdominal compartment syndrome. Am Surg. 2011;77(Suppl 1):S6-11.
2. Cheatham ML, Safcsak K, Sugrue M. Long-term implications of intra-abdominal hypertension and abdominal compartment syndrome: physical, mental, and financial. Am Surg. 2011;77(Suppl 1):S78-82.
3. Sanda RB, Aziz R, Bhutto A, Seliem SI. Abdominal compartment syndrome complicating massive hemorrhage from an unusual presentation of ruptured ectopic pregnancy. Ann Afr Med. 2011;10(3):252-5.
4. Malbrain ML, Cheatham ML, Kirkpatrick A, Parr M, De Waele J, Balogh Z, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. Intensive Care Med. 2006;32(11):1722-32.
5. O'Mara MS, Semins H, Hathaway D, Caushaj PF. Abdominal compartment syndrome as a consequence of rectus sheath hematoma. Am Surg. 2003;69(11):975-7.
6. Cheatham ML, Malbrain ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. II. Recommendations. Intensive Care Med. 2007;33(6):951-62.

Cite this article as: Wang P, Jia Z, Wang Z, Liu C, Liu J, Zhu Y. Abdominal compartment syndrome resulting from postpartum hematoma of wall: a rare occurrence associated with a high risk of mortality. Int Surg J 2015;2:756-60.