Twisted fate: Successful management of hypovolemic shock due to abruptio placenta secondary to uterine torsion complicated by unilateral absence of adnexa*

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ABSTRACT

Uterine Torsion is defined as rotation of the uterus of more than 45 degrees on its long axis. It is an unusual complication of pregnancy and for most obstetricians, it probably represents "once-in-a-lifetime" diagnosis. A 32-year old multipara at 30 week gestation with abdominal pain is presented. Laparotomy was performed for the diagnosis of hypovolemic shock secondary to suspected abruptio placenta. Intraoperatively, uterine torsion was observed with unilateral absence of the right adnexa. Prompt decision making with aggressive immediate management resulted to favorable maternal outcome.

Keywords: Adnexal absence, absent fallopian tube, absent ovary, uterine torsion, abruptio placenta

INTRODUCTION

Uterine torsion is the twist of the uterus between the cervix and uterine body. A minor degree of rotation of the pregnant uterus is fairly common during the 3rd trimester of pregnancy but is deemed rather negligible.[1] However, rotation of the gravid uterus more than 45 degrees is rare and its occurrence is defined as uterine torsion.

The range of associated symptoms in uterine torsion is both wide and non-specific which mimics other common conditions.

Uterine torsion is frequently associated with conditions that alter the shape or position of the uterus or adnexa. Mistaking uterine torsion as non-surgical or a medical obstetric complication which might delay correct diagnosis is potentially disastrous and catastrophic. Complications associated with uterine torsion permit little time for extensive evaluation. Since occurrence of severe uterine torsion may cause ischemic injury and serious thrombotic events, immediate diagnosis and management is crucial.

Unilateral uterine adnexal absence with a normal uterus is extremely rare but meaningful clinically. To date, this rare malformation is still not well understood.[2]

CASE

C.M. a 32-year old, Gravida 3 Para 2 (2002) with 30 weeks intrauterine pregnancy, was admitted for the first time due to dizziness. History revealed that she had only one antenatal clinic visit at 20 weeks age of gestation where physical examination was then unremarkable. She was requested to do pelvic ultrasound, however, she was lost to follow-up. The current pregnancy had been uncomplicated until the date of presentation.

Three hours prior to admission, C.M. claimed to have sudden, dull, steady, generalized non-radiating abdominal pain graded 4/10 in severity associated with persistent uterine contraction. She denied any perception of fetal movement during that time.

One hour prior to admission, still with persistence of above signs and symptoms now with increased severity of pain to 5/10, associated with dizziness and difficulty of breathing. She was then rushed to this institution and was subsequently admitted.

C.M.'s prior obstetrical history included two uncomplicated term vaginal deliveries. She claimed no history of subfertility, previous sexually transmitted infection, gynecologic surgery or menstrual irregularity.

Rest of her medical, surgical, family and personal history was not significant. Review of systems were unremarkable.

On general examination, C.M. was pale, weak-looking, drowsy and disoriented with cold and clammy skin. Blood pressure was initially not appreciated with weak pulses. She was then hooked to cardiac monitor and was noted to be tachycardic at 130 beats per minute. Temperature and respiratory rate were normal with oxygen saturation at 97 to 98%. Abdomen was globular with a fundic height of 28 cm. Uterus was noted to be tonically contracted and fetal heart tones was not appreciated. Exact fetal lie and presentation could not be fully assessed due to increased uterine tone. Admitting impression was Gravida 3 Para 2 (2002) Pregnancy Uterine 30 weeks, Hypovolemic shock probably secondary to concealed placental abruption. She

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was immediately hooked to oxygen via nasal cannula at 4-5 liters per minute. Two large bore intravenous catheters were inserted on both arms. An initial bolus of 200 ml. of plain NSS and 500 ml. hydroxyethyl starch in sodium chloride were given. An indwelling foley catheter was inserted. Blood transfusion was also started with one unit whole blood. During resuscitative efforts, blood pressure was then noted 110/60 mmHg and heart rate at 108 beats per minute. Internal examination was done which showed a firm, posterior and closed cervix drawn up to the pelvis, there was no bulging on the fornices nor blood on examining finger. Laboratory exams were requested. Complete blood count revealed anemia with hemoglobin of 99 g/L, hematocrit of 0.30, mild leukocytosis and thrombocytosis (Table 1a). Capillary blood glucose was elevated at 176 mg/dl. while other laboratory work-ups such as prothrombin time, activated partial thromboplastin time, serum sodium and potassium were all normal (Table 1b and 2).

Patient underwent emergency laparotomy due to the presumptive diagnosis of hypovolemic shock secondary to concealed placental abruption. Intraoperatively, the lower uterine segment was not formed, the serosal surface of the gravid uterus was noted to be bluish in color (Figure 1). No hemiperitoneum was evident. A low transverse cesarean section incision was immediately performed. A stillborn baby boy in frank breech presentation was delivered weighing 1,600 grams. The placenta was completely separated. It was removed with all membrane and cotyledons with approximately 500 cc. of retroplacental blood clots. After three-layer closure of the uterus, the corpus was unexpectedly observed to be completely twisted 180 degrees to the right around its axis above the level of the elongated cervix (Figure 1). The uterine incision was then realized to be at the lower segment of the posterior corpus. The grossly normal-looking left adnexa was discovered anteriorly on the maternal right side. The right ovary and right fallopian tube, on the other hand, were surprisingly absent (Figure 2). Detorsion of the uterus was then performed (Figure 3). Active management of third stage of labor was done by administration of 10 units oxytocin incorporated in 1 liter of plain NSS and slow

Table 1b

Activated Partial Thromboplastin Time		Normal Values
	April 3, 2014	
APTT	27.8	28.0-44.0 seconds
Control	38.5	31.6-40.0 seconds
Prothrombin		
Time	11.6 seconds	
Protime	94.2 %	
% Activity	0.98	
INR	11.8 seconds	
Control		

Table 2. Blood Chemistry

	April 3, 2014	
Potassium	3.80	3.80-5.50 meq/L
Sodium	136	135-145 mg/dl
Capillary Blood Glucose	176	90-120 mg/dl

Table 1: Hematology

Table 1a

Complete Blood Count	Results			Normal values
	April 3, 2014	**April 4, 2014**	**April 6, 2014**	
Hemoglobin	99	95	96	120-140g/L
Hematotocrit	0.30	0.29	0.28	0.37-0.47
RBC count	3.38	3.31	3.21	4.0-5.4 x 10 12/L
WBC count	19.9	16.6	11.0	5.0-10.0 x 10 9/L
Platelet	increased	217	279	150-400 x 10 12/L
Segmenters	0.70	0.80	0.75	0.55-0.70
Lymphocytes	0.23	0.14	0.18	0.25-0.40
Monocytes		0.06	0.01	0.02-0.08
Eosinophils	0.06		0.01-0.06	0.01-0.06
Stabs	0.01			

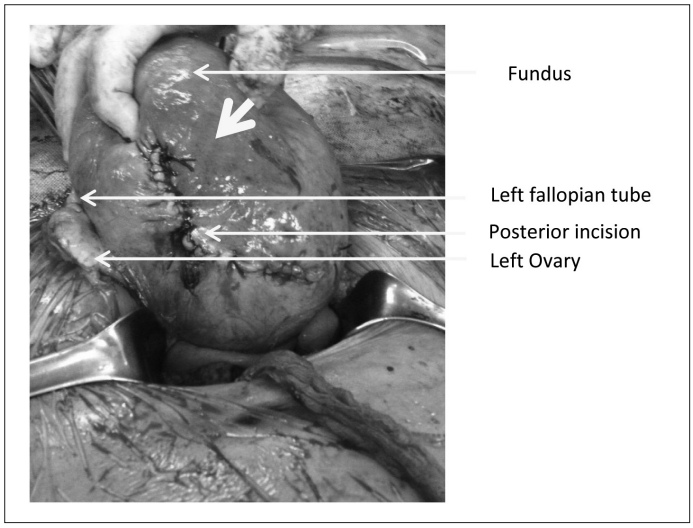

Figure 1. In this picture, the corpus was pulled upward and forward to show the incision made at the posterior surface of the lower uterine corpus after detorsion. Note the bluish discoloration of the serosal surface of the corpus (byarrow)

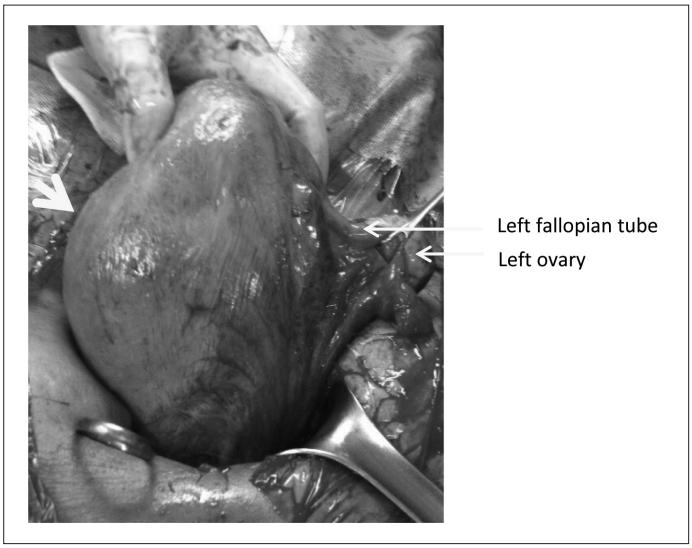

Figure 2. On laparotomy, the right ovary and right fallopian tube was noted to be absent (by arrow)

intravenous injection of 0.2 mg/ml methylergonovine maleate. The uterus was well-contracted. Abdomen was closed in layers after confirming hemostasis. Estimated blood loss was 3,500 ml. C.M.'s hemodynamic condition continually improved.

Post-operative diagnosis was Gravida 3 Para 3 (2-1-0-2) Pregnancy Uterine 30 weeks, frank breech, stillborn male weighing 1,600 grams, delivered via primary low segment cesarean section due to hypovolemic shock secondary to abruptio placenta secondary to Uterine Torsion, s/p Detorsion of the Uterus, Agenesis of the right adnexa.

Post-operatively, additional 2 units of packed RBC were transfused. A repeat complete blood count still showed anemia with hemoglobin of 95g/L and hematocrit of 0.29 on the first post-operative day and hemoglobin of 96 g/L and hematocrit of 0.28 on the third post-operative day (Table 1). C.M. was discharged improved on the fourth post-operative day.

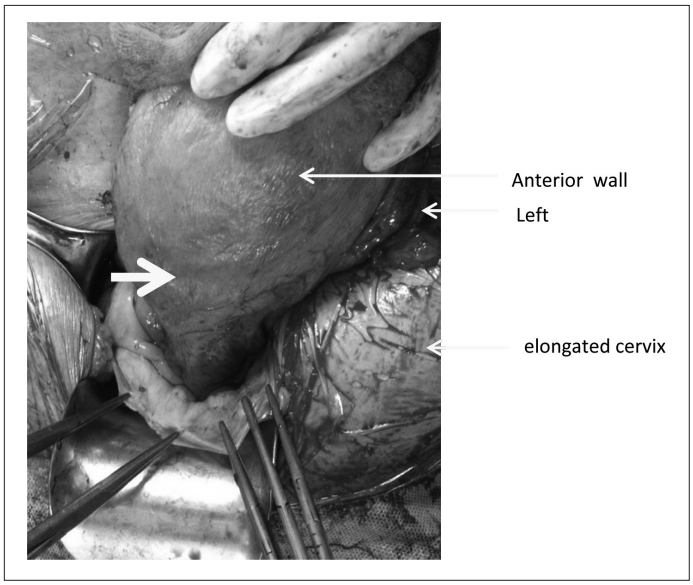

Figure 3. Laparotomy, showing the anterior surface of the uterine corpus after detorsion, still with bluish discoloration of the anterior surface of the corpus. Note that there was no uterine incision on the anterior surface of the lower uterine segment (big arrow).

DISCUSSION

Uterine torsion is defined as a rotation of more than 45 degrees of the uterus on its long axis that occurs at the junction between the cervix and the corpus.[3] Torsion of the uterus is a rare obstetric complication.[4]

A pathologic rotation of the uterusinvolves rotation beyond 45 degrees. Torsion of the entire uterus is rarely seen in obstetrical practice.[5,6,7] The limited number of cases reported and the lack of accuracy of some clinical records make the exact figure of incidence difficult to estimate with precision.

The extent of uterine torsion is usually 180 degrees. However, twists of 60 to 720 degrees[8,9] have also been reported. Dextrorotation is the most common occurrence in two-thirds of cases which is due to the orientation of the myometrial fibers. In the index patient, the uterus was rotated 180 degrees to the right.

Uterine torsion's clinical presentation is non-specific. Abdominal pain is the most common symptom, however, diagnosis is difficult since the pain may range from mild abdominal discomfort to severe pain presenting with acute abdomen and shock.[10] Torsion is also rarely asymptomatic.[11] C.M. initially presented with sudden,

dull, steady, generalized abdominal pain graded 4/10 in severity associated with persistent uterine contractions and eventually presented with signs and symptoms of hypovolemic shock.

During labor, uterine torsion may present itself by failure of cervical dilatation or failure to progress in labor despite strong uterine contractions. Patients may also manifest with abnormal fetal heart rate or fetal distress due to reduction or worse, obstruction in uterine blood flow. Other manifestations of uterine torsion may include vaginal bleeding, uterine tenderness, twisted vaginal canal and ureteral displacement. In the case presented, C.M. had a firm, close, posterior cervix despite presence of uterine contractions. Fetal heart beat was likewise not appreciated upon admission.

Establishing the diagnosis of uterine torsion is difficult due to the variety of clinical signs and symptoms. Hence, diagnosis is usually made after laparotomy. Most of the cases reported in literature were discovered to have uterine torsion after laparotomy or cesarean section.[12]

Imaging to establish the diagnosis of uterine torsion prior to laparotomy may be of use. Although insufficient, an ultrasound scan may show a change in placental location or a change in the position of a uterine myoma. Recently, both MRI and CT scanning have been used to help make the diagnosis prior to exploratory surgery.[13,14,15] Magnetic Resonance Imaging (MRI) may be of value if there is a high index of suspicion of uterine torsion prior to laparotomy. MRI may show an x-shaped configuration of upper vagina. There was no imaging modality requested in this case since laparotomy was immediately performed due to hypovolemic shock secondary to probable concealed abruption. The diagnosis of uterine torsion was only made intraoperatively.

In rare instances, the uterine torsion is of sufficient degree to interfere with uterine circulation. Disruption of uterine circulation may lead to acute maternal symptoms or threatens the survival of the fetus by directly restricting blood flow or by inducing abruption.[16] Theoretically, direct compression of the uterine veins and probably the ovarian veins, because of the uterine torsion, increases vascular pressure within the placental cotyledons. This effect leads to placental separation. In the case of C.M., abruptio was caused by the uterine torsion which was confirmed by the presence of complete detachment of the placenta (Figure 4) which resulted in intrauterine fetal asphyxia and ultimately fetal demise (Figure 5).

The exact mechanism and etiology of torsion is not known. The mechanism of torsion can be explained by an elongated cervix with structural weakness and angulation in the isthmic region leading to torsion. The structural weakness may be developmental or acquired.[17] It has been noted to occur in the presence of intra-abdominal

Figure 4. Maternal surface of the placenta showing 100% Abruptio

Figure 5. Stillborn baby boy with a birthweight of 1.6 kg.

adhesions, ovarian tumors and fetal malpresentations. In most cases however, torsion is associated with uterine distortion and asymmetry caused by uterine myomas or uterine developmental anomalies.[18,19] Uterine torsion resulting from abdominal trauma has also been reported.[20] In the case presented, C.M.'s uterine torsion was most likely caused by the inherent absence of the right fallopian tube and right ovary. The breech fetal presentation may also be a contributory factor of the uterine torsion.[21] Other factors such as presence of intra-abdominal adhesions, ovarian tumors, or uterine myomas were not evident in C.M. She also denied any previous abdominal trauma.

Unilateral absence of the fallopian tube and ovary is quite rare.[22,23,24] The incidence of unilateral absence of the ovary and fallopian tube has been suggested to be 0.0089%.[25] The etiology of adnexal anomaly remained unclear, although torsion or congenital defects were the most likely explanation.[26] The origin of adnexal absence remains obscure, due to its rare observation. We can identify three possible etiologies: 1) adnexal torsion; 2) tubal and ovarian maldevelopment secondary to ischemia, and 3) a defect in the development of the Müllerian and mesonephric system.[27,28]

Torsion of the ovarian pedicle can occur asymptomatically in adult life or childhood, or even before birth. Infarction is subsequently accompanied by tissue necrosis and resorption. Sometimes, ovoid structures can be observed adherent on the peritoneal surface or as free-floating masses. Authors considered them as ovarian residues following torsion.[29] The agenesis may also originate from a defect of the caudal end of the Müllerian duct and the genital ridge. Moreover, it is possible to consider a global mesonephric and paramesonephric anomaly on one side. Vascular accidents or failed canalization of the upper part of one fallopian tube could also result in an agenesis of the associated gonad, with the influence of unknown autocrine and paracrine signaling.[30,31] Any diagnosis of a urogenital abnormality necessitates investigation of co-morbid renal or genital abnormality.[32] Due to close embryological development, unilateral renal agenesis may be associated with other mesonephric and paramesonephric ductal anomalies.[33] The absence of a unilateral adnexa in our case remains a mystery, if it was due to ischemia and atresia secondary to adnexal torsion or a congenital developmental anomaly. There were likewise no adhesions or masses noted on the right lateral pelvic wall that may suggest adnexal remnants or residues. Prior to discharge, C.M. was adviced to undergo a kidney-ureter-bladder (KUB) ultrasound on an outpatient basis. C.M., however, did not return for follow-up with her KUB ultrasound result.

Laparotomy is warranted in patients with acute symptoms or with suspected uterine torsion. If the diagnosis is made in early pregnancy, manual detorsion of the uterus should be made with correction of any precipitating factors like adhesiolysis, myomectomy or ovarian cystectomy. However, hysterectomy should be performed if there is associated uterine necrosis or thrombosis of blood vessels due to prolonged torsion.

In patients with uterine torsion diagnosed at term, the uterus should be manually untwisted followed by delivery of the fetus by a cesarean section. However, if manual correction is not possible prior to cesarean section, a deliberate posterior hysterotomy may be performed to deliver the fetus.[34,35,36,37] A vertical or transverse incision may be employed, however, the risk of rupture is theoretically less with a posterior transverse incision than a posterior vertical incision although the exact risk is unknown. Hence, a transverse incision is preferred when feasible. Identification of the anatomical landmarks mainly the anterior position of the round ligaments, should be done prior to uterine incision to prevent or minimize any accidental injury to blood vessels or other organs. Manual uterine detorsion can be performed after delivery. Any precipitating conditions like intraabdominal adhesions, ovarian cyst or uterine myoma should be addressed to prevent recurrence of uterine torsion. In the index case, the transverse uterine incision was inadvertently made on the lower segment of the posterior corpus. Manual correction of the uterine torsion was employed after hysterotomy.

Although there are no studies that have been done on the effectiveness of different uterine stabilizing techniques, plication of the round ligaments or the lax uterosacral ligaments can be performed to prevent recurrence of uterine torsion. Round ligament plication may aid to keep the uterus in anterior position, reduce posterior uterine adhesion and future dyspareunia. Uterosacral ligament plication may provide resistance to torsion as well as prevent long term recurrence of uterine torsion. The intent in this plication procedure was to stabilize the uterus to prevent the recurrence of torsion. However, the effectiveness and the necessity of this treatment is unknown. There was no ligament plication procedure performed in C.M.

Torsion of the uterus is associated with significant morbidity and mortality. The overall maternal mortality is around 13% and is directly proportional to the duration of gestation and degree of torsion. Fetal mortality rates reported with the condition are 18%.[38] The immediate diagnosis of hypovolemic shock due to concealed abruption, coupled with the rapid response of the obstetrical team, brought forth a favorable maternal outcome in this case. However, fetal demise was not overlooked from the start of admission.

For future pregnancies, since the risk of uterine

rupture remains unknown with a posterior lower uterine segment incision, cesarean section is therefore recommended. C.M., prior to discharge, was advised that a repeat cesarean section is suggested for her next future pregnancy. She was also advised to return immediately once there is any symptom of abdominal pain since the risk of retorsion is unknown.

RESULTS

Absence of unilateral adnexa is a rare situation and its possible etiology still remains obscure. Torsion or congenital defect might be the possible etiologic factors.

The clinical presentation of uterine torsion is variable and clinical examination and ultrasonographic scanning may be insufficient for diagnosis. The diagnosis of uterine torsion is usually made at the time of laparotomy. A case of a 32-year old multipara at 30 weeks age of gestation with non-specific abdominal pain was presented. An emergency laparotomy was performed with the diagnosis of hypovolemic shock secondary to concealed placental abruption. Uterine torsion was then discovered intra-operatively with an incidental finding of an absent right adnexa. The placental abruption was precipitated by the uterine torsion which caused an increased in venous pressure. Given the wide range of possibilities, establishing standard guidelines for management at the time of surgical exploration is nearly impossible. Hence, uterine torsion, because of its elusive diagnosis remains a clinical challenge. Diligent and prompt management of the obstetrical team lead to favorable maternal outcome. Uterine torsion is seldom reported and is considered a treacherous complication of pregnancy. It is a rare event that merits numerous critical management considerations.

Although the incidence of uterine torsion is rare, it should be considered as a differential diagnosis of acute abdomen in pregnancy when there is sudden onset of shock, abdominal pain or vaginal bleeding that could not be explained by common obstetric conditions.

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