Intrapartum Spontaneous Uterine Rupture Following Uncomplicated Resectoscopic Treatment of Asherman's Syndrome

Chii-Shinn Shiau, MD; Ching-Chang Hsieh, MD; Chi-Hsin Chiang, MD; T'sang-T'ang Hsieh, MD; Ming-Yang Chang, MD

Since Asherman first published his series of intrauterine synechiae in 1948, only a few physicians have described the obstetric complications of patients who conceived following surgical treatment of intrauterine synechiae. We present a woman with a history of resectoscopic resection of intrauterine adhesions with a term pregnancy and spontaneous uterine rupture that occurred during the intrapartum period. At emergent cesarean section, hemoperitoneum of approximately 1500 mL was noted and a 10-cm defect was present in the lateral uterine wall; the edges of the defect were bleeding actively. Because of the potential for a disastrous outcome in the rupture of the pregnant uterus, patients treated for Asherman's syndrome should be identified early and appropriate precautions should be taken in their obstetric management. *(Chang Gung Med J 2005;28:123-7)*

Key words: uterine rupture, Asherman's syndrome.

In 1948, Asherman brought attention to intrauterine synechiae with a report of 29 patients with "menorrhea traumatica" following postpartum or postabortion curettage.[1] As reported previously, hysteroscopy is the method of choice to diagnose, treat, and follow patients with Asherman's syndrome.[2-4] Such intrauterine adhesions may vary in shape, extent, and location; extensive adhesions impair fertility, probably through interfering with the transport of the ovum or nidation.

The obstetric sequelae of patients who develop Asherman's syndrome and for those treated for this disorder are notoriously poor. Intrapartum spontaneous uterine rupture remains a serious and potentially catastrophic event for obstetricians. It may not have predisposing factors and may occur without any specific signs or symptoms.

We present a 31-year-old woman with a history of intrauterine adhesions treated successfully with resectoscopic resection of intrauterine adhesions. She experienced intrapartum spontaneous uterine rupture at term pregnancy with uneventful maternal and fetal outcomes following emergent cesarean section.

CASE REPORT

A 31-year-old woman, gravida 2, para 1, presented at 39 weeks and 4 days' gestation and was admitted to the labor ward with complaints of regular uterine contractions for the preceding 8 hours and spontaneous rupture of membrane 1 hour before admission followed by the onset of contractions occurring every 5 minutes. Significant medical and

From the Department of Obstetrics and Gynecology, Chang Gung Memorial Hospital, Taipei.
Received: Aug. 20, 2003; Accepted: May 5, 2004
Address for reprints: Dr. Ming-Yang Chang, Department of Obstetric and Gynecology, Chang Gung Memorial Hospital, No.199, Duenhua N. Rd., Sungshan Chiu, Taipei, Taiwan 105, R.O.C. Tel: 886-2-27135211 ext. 3345; Fax: 886-2-25147643; E-mail: mychang@cgmh.org.tw

surgical history of this patient included dilatation and curettage for delayed postpartum hemorrhage, which had been performed 3 years prior to this admission for a preceding pregnancy. She developed hypomenorrhea and dysmenorrhea after the therapeutic dilatation and curettage. She underwent diagnostic hysteroscopy, confirming some intrauterine adhesion bands (Fig. 1). She was then arranged for hysteroscopic treatment of intrauterine synechiae using the resectoscopic procedure. The adhesions were removed using the loop electrode resectoscope with an Aspen Laboratory electrocautery unit (Aspen Labs, Littleton, Co) at 30 W/second cutting current. The operating time was 15 minutes. Placement of a Cu-375 intrauterine device was used to keep the uterine walls apart after the operation and to prevent recurrent adhesions. The woman was treated for the first 2 months postoperatively with 2.5 mg/day of conjugated equine estrogen on days 1-25 and 10 mg/day medroxyprogesterone acetate on days 16-25. Her menstrual cycle was restored after the treatments and she conceived spontaneously 6 months later after the removal of the intrauterine device. The course of pregnancy was normal, with clinical and ultrasonographic evidence of a normally developing fetus. The results of all investigations appeared to be normal. The pregnancy progressed unremarkably until 39 weeks' gestation when she presented with regular uterine contractions and came to our delivery room. Vaginal examination on admission revealed a favorable cervix and the fetal head was engaged. A nonstress test showed a reactive fetal heart rate (FHR) with a baseline of 140 beat per minute (bpm) and regular uterine contractions every 5 minutes. Results of laboratory studies were within normal limits, and her blood pressure measurement was 120/75 mmHg. Ultrasound examination revealed a 3300 g fetus in the cephalic presentation with adequate amniotic fluid (AF). The placenta was lateral-posterior with no apparent signs of abruptio placentae. Four hours after admission, contractions remained regular every 3 to 4 minutes, and cervical dilatation progressed to 8 cm. The FHR was 130 bpm, with good beat-to-beat variability and accelerations. Five hours after admission, the FHR showed persistent decelerations and a slow return to baseline. After the administration of oxygen, hydration, and change of maternal position, cardiotocographic monitoring still revealed repeated variable deceleration with regular uterine contraction every 3 to 4 minutes. Pelvic examination of the patient at that time demonstrated the absence of a presenting part and a strange lump in her abdomen. The patient was immediately transported to the operating room. There, the fetal heart rate could not be detected by Doppler, and ultrasound examination demonstrated a rate of 30 bpm. A viable 3223 g female infant was delivered by emergent cesarean section 10 minutes after the onset of fetal bradycardia and there was approximately 1500 mL of fresh blood and AF in the maternal abdominal cavity. The infant was resuscitated vigorously and her Apgar scores were 1, 8, and 9 at 1, 5, and 10 minutes, respectively. The placenta was located in the fundal area of the uterus and was manually removed easily. After the delivery of the placenta, uterine inspection revealed a 10 cm linear rupture of the left posterior uterine wall (Fig. 2), which was bleeding profusely. The uterine defect extended from the level of the internal os to the left cornu and included the left utero-ovarian and broad ligaments. Four units of packed red blood cells were transfused and the estimated total blood loss was 1500 mL. After closure of the uterine incision, the lateral wall defect was closed in two layers with 1-0 vicryl suture. The first layer was a running stitch to reapproximate the deep myometrium; two separate running stitches, each including both the superficial myometrium and the uterine serosa, completed the closure. The infant was doing well without complications and was dis-

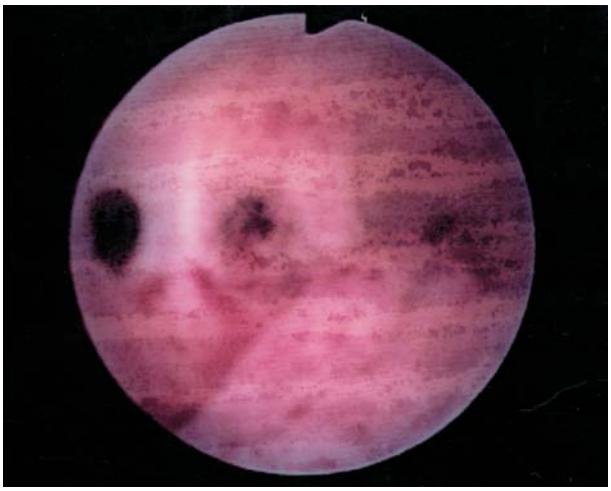

Fig. 1 Diagnostic hysteroscopy revealed some intrauterine adhesion bands

charged with the mother 5 days after the cesarean section.

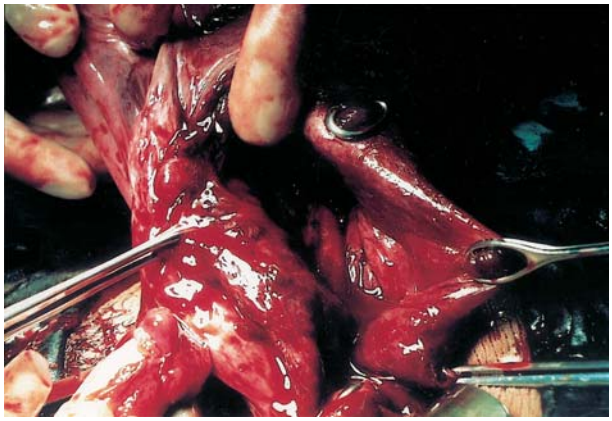

Fig. 2 Uterine inspection revealed a 10-cm linear rupture of the left posterior uterine wall with active bleeding

DISCUSSION

A simple etiologic classification of parturient uterine ruptures would include rupture of a previous cesarean section scar, traumatic rupture of the intact uterus, or spontaneous rupture of the intact uterus, as initially proposed by Garnet.[5] Spontaneous rupture of an intact uterus is especially prone to occur in women of great parity, particularly when associated with disproportion, low-dose oxytocin augmentation of labor and/or uterine hyperstimulation monitor patterns.

The majority of patients with Asherman's syndrome are of reproductive age. Therefore, achievement of pregnancy, not merely restoration of the cycle, is the ultimate therapeutic goal. Pregnancy following the treatments reveals a high incidence of complications, such as recurrent abortion, premature labor, abnormal fetal presentation, and abnormalities of labor and delivery, as well as anomalous insertion of the placenta. However, reports of pregnancy outcomes after such an occurrence are rare. Here, we present a patient of spontaneous uterine rupture at term pregnancy following an earlier uneventful operative hysteroscopic procedure and review the literature regarding patients with Asherman's syndrome who had received prior hysteroscopic surgery (Table 1) resulting in spontaneous uterine rupture.[6]

Complications after intrauterine adhesiolysis of Asherman's syndrome using the resectoscope were detailed by Friedman et al.[7] Thin myometrium was believed to contribute to sacculation formation and predispose to abnormal placentation, uterine rupture, and postpartum hemorrhage. Regarding our patient, we believe that the rupture was caused by weakening of the uterine wall secondary to the destruction of the myometrium by previous surgical procedures. It is possible that the electrocautery used to lyse the intrauterine adhesions could have resulted in thermal injury and weakening of the uterine wall. Perhaps the relative avascularity of the myometrium adjacent to the adhesion bands after electrical loop procedures predisposed the site to suboptimal healing and weakened scar formation, which subsequently contributed to the uterine rupture during the intrapartum period. It would seem prudent at the current time, for patients who undergo resectoscopic adhesiolysis of intrauterine adhesions and desire future pregnancy to choose sharp scissor dissection over electrosurgical dissection to prevent potential thermal injury to the normal myometrial tissue.

Table 1. Salient Features of Uterine Rupture Following Hysteroscopic Management of Asherman's Syndrome

Case	Method of	Perforation adhesiolysis	Presentation	Finding
Deaton et al[6]	Cold scissor	Yes	1. Severe epigastric pain and moderate vaginal bleeding 2. Acute bradycardia of fetus at 31 weeks	Hemoperitoneum 1000 cc with two bleeding rupture sites in the uterine fundus
Present patient	Electrical loop	No	1. Intrapartum repeated fetal heart beat variable deceleration 2. Absence of a presenting part and a strange lump in abdomen	Hemoperitoneum 1500 cc with a 10-cm linear rupture of the left posterior uterine wall

The need for early diagnosis and prompt surgical intervention in such a potentially catastrophic obstetric emergency has been previously mentioned.[8] The diagnosis of uterine rupture depends on a high degree of recognition of clinical signs and symptoms. In addition, fetal heart rate abnormalities were found in most cases.[9-11] Classically the pain of uterine rupture is severe, which can be shearing and continuous between contractions. Commonly reported signs and symptoms related to uterine rupture are abdominal pain and tenderness, shock, vaginal bleeding, undetectable fetal heart beat, palpable fetal body parts, cessation of contractions, and signs of intraperitoneal bleeding. In our patient, fetal heart beat abnormalities were present during the intrapartum period and subsequently a palpable strange lump was noted in the patient's abdomen, indicating the possibility of uterine rupture. When the diagnosis of uterine rupture is highly suggested, prompt surgical intervention by an obstetrician should be obtained due to its association with a high perinatal mortality rate and significant maternal morbidity. Early surgical intervention usually is the key to successful treatment of uterine rupture and better perinatal outcome.

Surgical management of a uterine rupture depends on the type and site of the suture, the extent of bleeding and the wishes of the woman with regard to future fertility. At the time of exploratory laparotomy, the patient should be evaluated for possible uterine repair or hysterectomy. Whether repair is feasible will depend on the hemodynamic status of the patient at the time of exploratory laparotomy. In early reports, uterine ruptures were managed with hysterectomy and several authors considered hysterectomy the procedure of choice.[5,12,13] Our patient was only 31 years old and just delivering her second baby. Considering her relatively young age and the possibility of desiring future fertility, we decided to preserve her uterus through primary closure of the ruptured uterine wound. Repair of the uterine rupture, however, raises the possibility of rupture recurrence during a subsequent pregnancy, which has a reported incidence of 4.3-19%.[14]

This report raises the question of who specifically is at heightened risk for obstetric uterine rupture subsequent to resectoscopic adhesiolysis of intrauterine adhesions. As experience with resectoscopic resection of the intrauterine adhesions increases, more information will become available regarding the risk of uterine rupture during labor in those women whose procedures were entirely uneventful. As for occurrence of uterine rupture per se, awareness of risk factors, recognition of clinical signs and symptoms, and prompt surgical intervention are the keys to optimal maternal and perinatal outcomes.

REFERENCES

1. Asherman JG. Amenorrhea traumatica (atretica). J Obstet Gynaec Brit Emp 1948;55:23.
2. March CM, Israel R. Intrauterine adhesion secondary to elective abortion: Hysteroscopic diagnosis and management. Obstet Gynecol 1976;48:422.
3. Levine RU, Neuwirth RS. Simultaneous laparoscopy and hysteroscopy for intrauterine adhesion. Obstet Gynecol 1973;42:441.
4. March CM, Israel R. Hysteroscopic management of intrauterine adhesions. Am J Obstet Gynecol 1978;130:653.
5. Garnet JD. Uterine rupture during pregnancy. Obstet Gynecol 1964;23:898-905.
6. Deaton JL, Maier D, Andreoli J. Spontaneous uterine rupture during pregnancy after treatment of Asherman's syndrome. Am J Obstet Gynecol 1989;60:1053-4.
7. Friedman A, DeFazio J, DeCherney A. Severe obstetric complications after aggressive treatment of Asherman's syndrome. Obstet Gynecol 1986;67:864-7.
8. Turner MJ, Robson MS, MacDonald D, Stronge JM. Successful outcome after antepartum explusion of placenta and fetus into abdominal cavity: a case report. Eur J Obstet Gynecol Reprod Biol 1989;33:187-8.
9. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: maternal and fetal consequences. Am J Obstet Gynecol 1993;169:945-50.
10. Molloy BG, Sheil O, Duignan NM. Delivery after cesarean section: review of 2176 consecutive cases. Br Med J 1987;294:1645-74.
11. Flannelly GM, Turner MJ, Rassmussen MJ, Stronge JM. Rupture of the uterus in Dublin; an update. J Obstet Gynecol 1993;13:440-3.
12. Eden RD, Parker RT, Gall SG. Rupture of the pregnant uterus: A 53-year review. Obstet Gynecol 1986;68:671-4.
13. Weingold AM, Sall S, Sherman DH, Brenner PH. Rupture of the gravid uterus. Surg Gynecol Obstet 1966;122:1233-8.
14. Aguero O, Kizer S. Obstetric prognosis of the repair of uterine rupture. Surg Gynecol Obstet 1968;127:528-30.

子宮腔粘連經子宮鏡治療後合併待產過程自發性子宮破裂

蕭啓信 謝景璋 江其鑫 謝燦堂 張明揚

1948年 Asherman 報告子宮腔粘連的病例以來，討論接受子宮腔粘連手術後病患的產科併發症之相關報告甚少。本報告提出一婦女因子宮腔粘連接受子宮鏡治療後，於妊娠足月併於待產過程中併發自發性子宮破裂之病例。在緊急剖腹生產過程中，發現母體腹腔內積血一千五百毫升，同時於子宮左側壁發現約10公分長度不規則破裂的子宮傷口。自發性子宮破裂的診斷，端賴臨床醫師的高度警覺，以減少母體與嬰兒方面的後遺症。當病患因子宮腔粘連接受治療後，在妊娠或待產中的處置，都應該視爲產科處置的一項危險因子。(長庚醫誌 2005;28:123-7)

關鍵字：子宮破裂，Asherman 症候群。

長庚紀念醫院　台北院區　婦產科系
受文日期：民國92年8月20日；接受刊載：民國93年5月5 日。
索取抽印本處：張明揚醫師，長庚紀念醫院　婦產科系。台北市105敦化北路199號。Tel: (02)27135211轉3345; Fax: (02)27197368; E-mail: mychang@cgmh.org.tw