Fetal Death in a Patient With Intrahepatic Cholestasis of Pregnancy

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BACKGROUND: Treatment with ursodeoxycholic acid in intrahepatic cholestasis of pregnancy reduces concentration of transaminases and bile acids in maternal serum, and is thought to reduce fetal death. We report a case of fetal death in a patient with intrahepatic cholestasis of pregnancy who had responded well to ursodeoxycholic acid, demonstrated by a low bile level.

CASE: A young nulliparous woman presented with intrahepatic cholestasis of pregnancy at 28 weeks of gestation. Transaminases and bile acids decreased after ursodeoxycholic acid administration. The patient was discharged from the hospital until delivery and received biochemical markers and conventional fetal monitoring twice weekly. Due to low bile acid values (< 13 UI/L) and unfavorable cervix, the patient was followed up expectantly. Fetal death occurred at 39 weeks and 3 days, although cardiotocograph testing results were normal the day before.

CONCLUSION: When lung maturity is achieved for patients with intrahepatic cholestasis of pregnancy, delivery should be considered.

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Intrahepatic cholestasis of pregnancy (ICP) is a reversible form of cholestasis that appears during the second half of pregnancy and persists until delivery. Maternal prognosis is excellent and the symptoms, ie, intractable pruritus and serum abnormalities, resolve rapidly postpartum. However, ICP may be responsible for preterm deliveries (19–60 %) and for high rates of abnormal intrapartum fetal heart rate patterns, meconium staining of amniotic fluid (27%), and intrauterine fetal death (1% to 7%).[1] The pathophysiology of these fetal complications is poorly understood but may be related to an increased level of serum bile acids.[1]

It is now well established that ursodeoxycholic acid mitigates pruritus, reduces concentration of transaminases and bile acids in serum toward normal values, and decreases the delivery of bile acids to the fetus.[2–5] Ursodeoxycholic acid is therefore often prescribed in patients with ICP to reduce maternal pruritus and improve fetal prognosis. To date, it has not been clearly demonstrated whether ursodeoxycholic acid has a significant effect on perinatal morbidity and mortality. We report the first case of intrauterine fetal death in an ICP patient who had previously responded well to ursodeoxycholic acid treatment demonstrated by low bile acid levels.

CASE

A young nulliparous white woman at 28 weeks of gestation was referred to our obstetric department, having complained of pruritus for 2 days. The patient had no previous medical history. Pruritus was persistent day and night. On admission, laboratory results showed a cholestatic pattern, with a high level of transaminases (aspartate transaminase 470 UI/L, alanine transaminase 991 UI/L), and bile acids (58 μM). The serum biochemical analysis obtained during the course of the disease is shown in Figure 1.

Obstructive gallstone disease was excluded after ultrasonography of the liver and biliary tract. Serologic tests performed for viral hepatitis A, B, and C, cytomegalovirus, Epstein-Barr virus, and human immunodeficiency virus infections were negative. There were no signs of preeclampsia and no medications were administered for at least 15 weeks before baseline laboratory results.

Intrahepatic cholestasis of pregnancy was initially treated with cholestyramine (12 g per day in 3 oral doses)

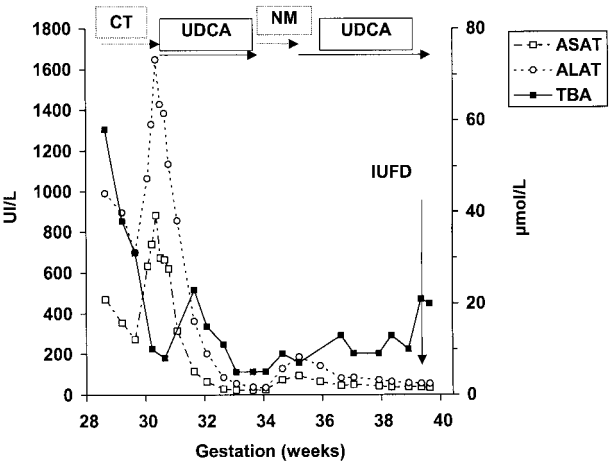

Fig. 1. Serum biochemical analysis during the course of the disease. CT, cholestyramine; NM, no medication; UDCA, ursodeoxycholic acid; AST, aspartate aminotransferase (UI/L); ALT, alanine aminotransferase (UI/L); TBA, total bile acids (μM); IUFD, Intrauterine fetal death.

Sentilhes. Bile Acid Level and Intrahepatic Cholestasis. Obstet Gynecol 2006.

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and betamethasone (12 mg intramuscularly, 2 doses separated by 24 hours) administered to achieve fetal lung maturation. Liver function tests, including serum bile acids, were performed twice weekly. Although a mitigation of pruritus was achieved, cholestyramine was replaced by ursodeoxycholic acid at 14 mg/kg body weight dosage (in 2 oral doses) due to a recurrence in the increase of cholestasis biochemical markers (Fig. 1). Biochemical markers decreased dramatically 9 days after ursodeoxycholic acid was instituted (Fig. 1). The patient was discharged from the hospital at 32 weeks and 5 days of gestation, and ursodeoxycholic acid was stopped after 25 days of treatment. It was reintroduced 1 week later and maintained until delivery due to a recurrence in the increase of transaminases and bile acids. Once again, biochemical markers decreased dramatically to reach normal values (Fig. 1).

The patient was discharged from the hospital until delivery. Obstetric, cardiotocography, and biochemical markers of cholestasis were monitored twice weekly, and biophysical profile and fetal growth were assessed weekly by ultrasonography. The patient was monitored every 2 days from 37 weeks of gestation. Due to low bile acids values (< 13 UI/L), absence of pruritus, and Bishop score less than 3, we opted for expectant management until induction of labor was possible. Intrauterine fetal death occurred at 39 weeks and 3 days of gestation, whereas cardiotocograph registrations were normal the day before, with aspartate transaminase, alanine transaminase, and bile acids measured at 38 UI/L, 55 UI/L, and 21 μM, respectively, also the day before. The patient delivered a female stillbirth weighing 3,400 g. Maternal serum aminotransferases and bile acid levels returned to normal postpartum with no further need for medication.

To identify other possible explanations for the stillbirth, all the following evaluations were performed: autopsy and pathologic examination of the placenta; bacteriologic cultures from the cervix, amnion, placenta and umbilical cord; screening for viral and toxoplasma infections by maternal serology and cultures from the amnion and placenta with polymerase chain reaction for Parvovirus B19, cytomegalovirus, and enterovirus; Kleihauer Betke test; screening for maternal diabetes mellitus or coagulation disorders (thrombophilia); placental cultures for karyotype determination; and skeletal x-rays. The autopsy results revealed the presence of meconium in the bronchioles and alveoli, with no other abnormalities. All other test results were negative. With the exception of ICP, no other possible cause of fetal death was identified.

COMMENT

We report, to our knowledge, the first case of intrauterine fetal death related to ICP despite the patient having responded well to ursodeoxycholic acid treatment, demonstrated by a reduction in bile acid levels toward normal values. It is also the first report of twice-weekly screening for transaminases and bile acids preceding intrauterine fetal death. We carried out a computerized literature search in the general bibliographic databases MEDLINE (1966 to August 2004) and Embase (1988 to August 2004), using MeSH terms for "cholestasis," "ursodeoxycholic acid," "intrauterine fetal death," and "pregnancy." The search was not limited by language or publication type (full articles or abstracts). No cases of intrauterine fetal death were reported in ICP pregnancies treated by ursodeoxycholic acid.

Our stillbirth screening was performed according to the test protocol of Petersson et al.[6] In 188 cases of intrauterine fetal death, these authors identified a possible causative factor of intrauterine fetal death in 171 (91%) cases.[6] From the results of a complete stillbirth screening, in accordance with recent literature,[6] combined with those of the autopsy, which revealed meconium in bronchioles and alveoli with no other abnormalities, we conclude that ICP was the most likely cause of intrauterine fetal death. However, it remains possible that intrauterine fetal death and ICP were unrelated.

As we observed, in the overwhelming majority of cases reported in the literature, pruritus, transaminases, and bile acid levels were dramatically reduced with ursodeoxycholic acid, with no adverse effects to the fetus.[2–5] Ursodeoxycholic acid is effective in reducing maternal serum bile acids[2,4–5] and in restoring the ability of the placenta to carry out vectorial acid bile transfer.[2,4–5] Consequently, to date, the majority of authors recommend ursodeoxycholic acid treatment as first-line therapy for ICP patients.[2,4–5] However, the 3 randomized placebo-controlled trials of ursodeoxycholic acid in the treatment of ICP failed to demonstrate a reduction in intrauterine fetal death after ursodeoxycholic acid treatment, possibly due to a lack of statistical power.[2,4–5]

Recently Glantz et al[7] introduced the concept of bile acid value threshold for fetal risks. They prospectively identified 2 distinct ICP populations based solely on bile acid levels[7] and found that no increase in fetal risks was detected in ICP patients with bile acid level less than 40 μM. They therefore proposed that these women be managed expectantly to reduce medical care costs.[7] In our case, this concept does not seem sufficient, because intrauterine fetal death occurred with a bile acid level measuring 21 μM. However, the delay in obtaining results varied from 2 to 6 days due to constraints on clinical chemistry laboratories, thus emphasizing the importance of using a reference laboratory where evaluations are performed either daily or 3 times per week. In our case, obstetricians received maternal serum bile acid

level results showing a 2-fold increase in 48 hours, the day after intrauterine fetal death.

To explain intrauterine fetal death with low bile acid level, we hypothesized the possibility of a cumulative toxic effect of bile acids on the fetus and that sensitivity to bile acids varies from one fetus to another. Moreover, although the bile acid level decreased dramatically after ursodeoxycholic acid treatment, the values were frequently more than normal values ($< 10\ \mu M$), particularly at the end of pregnancy. Therefore, we could speculate that spontaneous preterm delivery in ICP plays a protective role by removing the fetus from toxic bile acids. However, because ursodeoxycholic acid treatment reduces spontaneous and iatrogenic preterm deliveries related to ICP,[2,4] it could at the same time hinder this protective effect. Thus, because ursodeoxycholic acid has become the first line treatment for ICP, this will most likely lead to a future increase in the number of ICP pregnancies reaching 37 weeks with fetuses subjected to a potential cumulative toxic effect of bile acids and with unfavorable cervix for induction of labor.

To date, there is no consensus regarding prenatal surveillance of ICP patients. Nevertheless, the only method that is associated with a reduction of intrauterine fetal death remains active management, because conventional monitoring of fetal well-being does not, in most cases, predict fetal death, which may occur within 24 hours of a normal cardiotocograph.[8] Moreover, active management may explain the low incidence of adverse perinatal outcomes and intrauterine fetal death (0.4%) in the study by Glantz et al.[7] Finally, the usefulness of active management has been reinforced by the findings of Williamson et al.[1] These authors assessed the clinical outcome of 227 white ICP patients. Among the singleton pregnancies, 20 intrauterine fetal deaths were found. The median gestation at which intrauterine fetal death occurred was 38 weeks, and only 2 intrauterine fetal deaths occurred before 37 weeks. When compared with the risk of required ventilation for respiratory distress syndrome at an earlier gestation (ie, 1:557 (0.2%) at 37 weeks and 1:1,692 at 38 weeks),[1] the arguments weigh in favor of active management.

In conclusion, the aim of ICP treatment is initially to reduce the bile acid level and preterm deliveries with ursodeoxycholic acid and, second, to remove the fetus from cumulative toxic bile acids with active management when lung maturity is achieved. Considering our case and in the absence of a randomized study, it would not be reasonable, to date, to propose expectant management solely based on bile acid levels when lung maturity is achieved for ICP patients.

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Necrotizing Fasciitis of the Scalp in a Newborn

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BACKGROUND: Fetal scalp electrode monitoring is usually without complications, but on rare occasions it can serve as a portal of entry for organisms colonizing the maternal genital tract.

CASE: We present a case of neonatal necrotizing fasciitis of the scalp that was associated with intrapartum fetal scalp electrode monitoring. Skin cultures grew Group A *Streptococcus* M11 T nontypeable serotype, an unusual cause of neonatal necrotizing fasciitis. The neonate's mother had a concurrent perineal infection and the same Group A streptococcal serotype was cultured from maternal blood and vaginal swabs.

CONCLUSION: This case highlights the emergence of life-threatening Group A *Streptococcus* causing invasive disease in both infants and mothers and the need for careful monitoring of neonates who have had intrapartum electrode monitoring.

(Obstet Gynecol 2006;107:461–3)

Necrotizing fasciitis also referred to as "flesh-eating bacteria disease" is an acute, rapidly progressive, potentially fatal infection of the superficial and deep fascia and subcutaneous tissue.[1–3] Necrotizing fasciitis, although rare in children (0.018 per 100,000 children per year), is even rarer in neonates, occurring mostly in term infants with an equal gender distribution and a mortality rate as high as 60%.[1,2] The paucity of cutaneous findings early in the course makes a high index of suspicion necessary for a prompt diagnosis.[2] Marked tissue edema, rapid progression of inflammation, and signs of septic shock are the clinical diagnostic clues.[2] Frozen section analysis, polymerase chain reaction assay, ultrasonography, computed tomography, and magnetic resonance imaging are useful diagnostic tools, but the definitive diagnosis is usually made at surgery.[2] Complications include septic shock, disseminated intravascular coagulation, multiorgan failure, and death.[2]

Neonatal necrotizing fasciitis is frequently polymicrobial, *Staphylococcus aureus*, *Escherichia coli*, *Enterococcus*, *Clostridium* spp, and *Bacteroides* spp being the predominant organisms isolated.[2] However, Group A *Streptococcus* (*S pyogenes*) has been associated in necrotizing fasciitis secondary to omphalitis, circumcision, and abdominal surgery.[4]

CASE

A term female infant weighing 3,560 g was born by vacuum-assisted vaginal delivery for poor maternal effort to a 34-year-old primigravida after 4 hours of ruptured membranes, with intrapartum fetal scalp monitoring. The latter was placed with no difficulty, because of fetal tachycardia. Her mother sustained second-degree perineal lacerations. She also complained of a sore throat and had a fever up to 38.5°C first noted 7 hours after delivery that lasted for 24 hours. She received 3 doses of intravenous cefazolin.

At 32 hours, the infant had a fever of 38°C for 4 hours; a small erythematous scalp lesion with dried yellow exudate and a right-sided cephalhematoma (thought secondary to the vacuum extraction) were noted. After normal examinations on day 3, both mother and newborn were discharged on no medications.

On the 4th day, a dark, rapidly progressive lesion was noted over the cephalhematoma. The neonate was irritable and fed poorly. In the emergency department, a diagnosis of sepsis with disseminated intravascular coagulation was made. The infant was intubated for severe respiratory distress. After resuscitation with fluids, transfusions of blood products and commencement of intravenous ampicillin, cefuroxime, and gentamicin, she was transferred to the tertiary regional hospital. Examination revealed a febrile, critically ill neonate, with a necrotic, sloughing, actively bleeding scalp hematoma extending from the crown to the posterior auricular area and neck (Fig. 1). She was generally pale, with anasarca, petechiae on the chest and arms, and extensive bruising on her thighs, pelvis, abdomen, buttocks, and mastoid area.

Abdominal distension absent bowel sounds and a cardiac murmur were found. Platelet count was 6×10^9/L, hemoglobin 84 mg/dL, glucose less than 1 mmol/L, pH 6.75 with a base deficit of –22, lactate 16.45 mmol/L, and creatinine 230 μmol/L. Leukocytes showed a 35% left shift and toxic granulations. Aspiration of the rapidly expanding scalp lesion showed bacteria. A frozen section of skin confirmed necrotizing fasciitis, and skin cultures grew Group A *Streptococcus*, type M11 T nontypeable (National Centre for *Streptococcus*, Edmonton, Alberta, Canada). Blood and urine cultures were sterile. Imaging studies showed multiorgan hypoperfusion and a subdural hematoma.

Her scalp was débrided 3 times in the first 72 hours of

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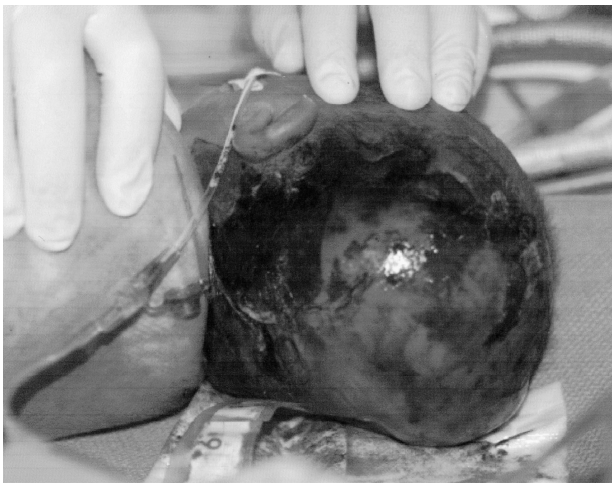

Fig. 1. Necrosis, sloughing, and bleeding of almost the entire scalp seen on admission, distinguishing necrotizing fasciitis from a cellulitis.

Davey. Necrotizing Fasciitis in a Newborn. Obstet Gynecol 2006.

admission. On admission, antibiotics were changed to meropenem and clindamycin, and penicillin G was added the following day after consultation with the Infectious Diseases service. She was treated with 5 days of intravenous immunoglobulin, and intravenous antibiotics were continued for 3 weeks. Her course was complicated by acute renal failure, respiratory distress syndrome, perforated necrotizing enterocolitis (requiring resection of necrotic bowel, a proximal jejunostomy, and distal ileal mucous fistula), recurrent infections, failure to thrive, and cholestatic jaundice. There was gradual resolution of multiorgan dysfunction. She later had 2 successful allografts and 1 autograft, and the bowel was reanastomosed. Interestingly, hair regrew over most of the scalp.

On the day after the neonate's admission, her mother was admitted to a nearby hospital complaining of sore throat, perineal pain, discharge, and fever with chills and rigors. There was complete breakdown of the perineal repair, with extreme induration and copious discharge, requiring 2 débridements under general anesthesia. Admission cultures of maternal vaginal swabs grew Group A *Streptococcus*, also type M11 T nontypeable, but urine, blood, and throat swab samples were negative. She was treated with intravenous penicillin G and clindamycin.

Subsequent vaginal and blood cultures were negative. After 10 days, she was discharged home with no further complications to complete a further 10 days of antibiotics with oral amoxicillin-clavulanate.

COMMENT

In the 1930s and 1940s Lancefield group A streptococci were the major cause of neonatal and maternal mortality, but after the introduction of antimicrobials, *E coli* and Group B streptococci became the predominant causes of neonatal sepsis.[5] However, during the past 20 years, the incidence of severe disease caused by Group A *Streptococcus* has been increasing in both children and adults.[5,6] Twenty-six cases of invasive neonatal Group A streptococcal infections have been reported in the last decade, 17 of these in the last 5 years, possibly reflecting an actual rise in the incidence of colonization of the vaginal epithelium of reproductive women.[5]

Seventy-five percent of early onset Group A streptococcal infections occur as a result of vertical transmission and are characterized by respiratory distress, rapid deterioration, and a high mortality rate.[5] The mothers of these neonates often have concurrent puerperal sepsis or toxic shock–like syndrome.[5] It may be that early onset neonatal Group A streptococcal infections are the consequence of hematogenous dissemination or a toxin-mediated syndrome beginning in utero.[5] This is suggested by the high fetal mortality rate among cases of maternal invasive Group A streptococcal disease with prenatal onset that has been documented in obstetric literature.[5]

The increase in invasive Group A streptococcal infections is likely due to host factors, but the acquisition of a large number of virulence factors, such as M proteins (which can trigger severe and potentially fatal infections in the susceptible host), seems to be an important factor.[6] The M protein enables the bacteria to resist phagocytosis.[6] The function of the T protein (a cell wall protein) is unclear, but it is used for epidemiologic studies and to serotype strains of Group A *Streptococcus*.[6] Of the more than 100 M proteins identified, M1T1 and M3 are the predominant serotypes associated with invasive Group A streptococcal disease in neonates.[6] The M11 serotype, 1 of the 10 most frequently identified M types in Canada (4.8% of isolates) has also uncommonly been associated with invasive Group A streptococcal infections.[7]

The incidence of complications of fetal scalp electrode monitoring, usually considered safe, varies from 0.4% to 5%.[8] Necrotizing fasciitis of the scalp (accounting for only 3% of all cases of neonatal necrotizing fasciitis), is a rare complication of the procedure.[2] Vacuum extraction, prolonged duration of monitoring, and maternal puerperal infection are factors that increase the likelihood of neonatal scalp infections.[8] Although it has been suggested that maternal Group A streptococcal disease may be preventable, the issue of prenatal Group A *Streptococcus* screening remains controversial because of the low rate of rectovaginal carriage (.03% to 0.11%) in late pregnancy.[5]

Because the incubation period for infection varies from 2 to 14 days, any infant who has had the presenting part punctured for the purpose of intrapartum monitoring should have frequent examinations of the scalp or buttocks over a period of weeks by caregivers and medical staff.[8] It may be prudent to treat neonates born to mothers with suspected invasive *S. pyogenes* infection empirically with antibiotics without delay, even in the absence of signs of infection.[8] We believe that maternal genital colonization with Group A *Streptococcus* was the source of infection in this neonate.

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Diagnosis of Placental Abscess in Association With Recurrent Maternal Bacteremia in a Twin Pregnancy

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BACKGROUND: Placental abscess formation is rarely recognized prenatally. We present a case detected ultrasonographically that developed from a central line infection and caused recurrent maternal bacteremia.

CASE: A young woman with a 21-week twin gestation presented with recurrent fevers. She had received treatment for bacteremia due to *Serratia marcescens*. The initial source of the infection was a peripherally inserted central catheter line placed in the first trimester for hyperemesis gravidarum. Fevers continued throughout the second course of antibiotics. An abscess seen sonographically in twin A's placenta was aspirated using a spinal needle, revealing *Serratia* bacteria. Aspiration was performed at 22 weeks of gestation. Amniotic fluid samples obtained from both sacs were negative for infection. Over 4 weeks, the abscess enlarged and she was delivered. Twin A died of sepsis and twin B had a relatively favorable neonatal course.

CONCLUSION: Prenatal diagnosis of placental abscess presents a difficult management dilemma. Traditional amniotic fluid studies did not predict the poor outcome of the affected fetus.

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Bacterial invasion of the fetal compartment frequently produces a fulminant infection resulting in chorioamnionitis, fetal sepsis, and spontaneous delivery. Most cases involve ascending infection from the vagina, but infection may also occur due to transfer of maternal bloodborne bacteria across the placenta. We present a case of a placental abscess detected prenatally that originated from a maternal peripherally inserted central catheter line infection.

CASE

A 28-year-old gravida 3 para 2 with a diamniotic dichorionic twin gestation at 21 weeks of gestation presented with 3 weeks of fevers accompanied by chills, malaise, and headache. She had severe hyperemesis gravidarum in her first trimester requiring home intravenous therapy through a peripherally inserted central catheter line. Three months before admission she was treated at another hospital for peripherally inserted central catheter line sepsis due to *Serratia marcescens*. She received 3 days of intravenous antibiotics before leaving the hospital against medical advice on oral antibiotics.

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On admission she had a temperature of 39.8°C with a nonfocal physical examination. Her uterus was nontender and cervix was closed. No contractions were detected on tocodynamometry, and fetal heart tones ranged between 140 and 150 beats per minute. Obstetric ultrasonography on labor and delivery showed a diamniotic dichorionic twin gestation consistent with 21 weeks of gestation. The placentas were fused along the anterior uterine wall and the amniotic fluid volume was normal in both sacs. Her white blood count was 13.0 × 109/L. Two sets of blood cultures were obtained, and intravenous ceftriaxone was initiated. Admission blood cultures subsequently grew *Serratia marcescens*.

She continued to have fevers to 39.4°C after 4 days of intravenous antibiotics despite documented susceptibility of the bacteria to ceftriaxone. She remained hemodynamically stable and otherwise asymptomatic. All subsequent blood cultures were negative and her white blood cell count was essentially unchanged. On hospital day 5 imaging studies were ordered to locate a source for persistent infection. Chest x-ray, echocardiogram, and renal and abdominal ultrasound examinations were negative. Transvaginal ultrasonography showed cervical shortening with funneling of the membranes. On transabdominal ultrasonography, a 3-cm, well-circumscribed mass was visualized within twin A's placenta. It had a thick hypoechoic wall and a sonolucent center (Fig. 1). Under ultrasound guidance, 1 mL of pink turbid fluid was aspirated from the placental mass and 15 mL of clear fluid was obtained from each amniotic sac. Amniotic fluid glucose levels were 26 mg/dL in both gestational sacs, and gram stain and cultures of amniotic fluid were negative. Culture of the fluid from the placental mass grew *Serratia marcescens*.

The patient was afebrile on hospital days 6 through 10, but at 22 weeks and 4 days of gestation she began spiking daily fevers up to 38.6°C. Repeat blood cultures were negative and she was asymptomatic. Ultrasonographically, the abscess cavity doubled in size and became a multiloculated, cystic collection with low-level echoes. Evacuation of the uterus was recommended, but the patient refused induction of labor. Her antibiotics were changed from ceftriaxone to ertapenem and she was afebrile for 13 days. On hospital day 24 she had a temperature of 38.2°C, and repeat amniocentesis was negative for intra-amniotic infection. Four days later, at 25 weeks of gestation, she had 2 temperature elevations to 39.1°C. On transabdominal ultrasonography, the abscess seemed to have spread to twin B's placenta (Fig. 2). Both fetuses had reassuring heart rate tracings and normal biophysical profiles. Delivery was again recommended for worsening maternal condition and progression of the placental abscess.

Twin male infants weighing 710 g (22nd percentile) and 775 g (27th percentile) were delivered by low transverse cesarean. Apgar scores were 2 at 1 and 5 minutes and 5 at 10 minutes for twin A and 2 at 1 and 5 minutes and 6 at 10 minutes for twin B. Arterial cord gases on both infants were within the normal range. Twin A was treated for hypotension and metabolic acidosis with antibiotics, vasopressors, and a high-frequency oscillating ventilator. He died of suspected sepsis 2 days after birth despite negative blood cultures. Twin B initially required vasopressors and ventilator support after birth, but was extubated within 1 week. He was initially treated for jaundice, patent ductus arteriosus, and retinopathy of prematurity. Serial cranial ultrasound findings were normal. At 3 months of age, his only active problem was chronic lung disease, requiring supplemental low-flow oxygen. The mother defervesced after delivery and was discharged home on 14 days of intravenous imipenem.

Gross examination of the placenta revealed a 4-cm abscess cavity in twin A's placenta. Necrotic placental

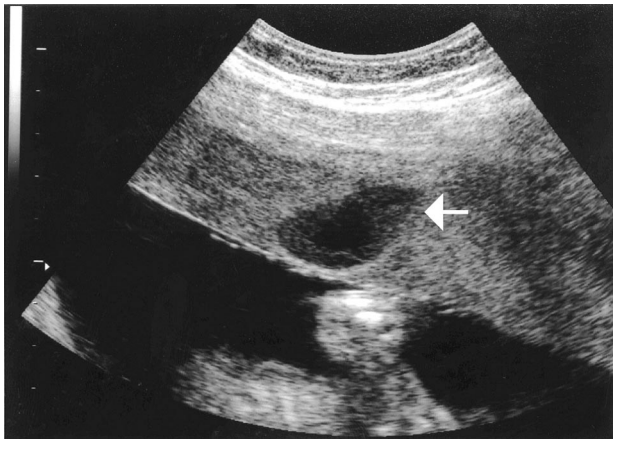

Fig. 1. Transabdominal ultrasonogram at 21 weeks of gestation showing a complex mass *(arrow)* in the placenta of twin A.

Meirowitz. Placental Abscess With Recurrent Bacteremia. Obstet Gynecol 2006.

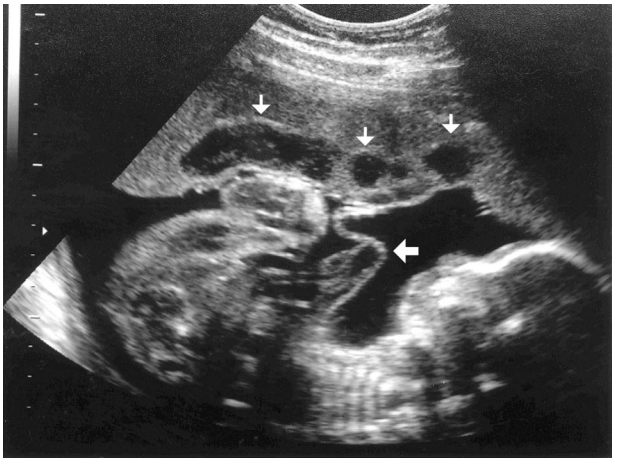

Fig. 2. Transabdominal ultrasonogram at 25 weeks and 1 day of gestation suggesting extension of the original placental abscess *(thin arrows)* into twin B's placenta. The thick arrow indicates the intertwin membrane.

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tissue extended from the margin of the abscess into twin B's placenta. Microscopic examination showed extensive necrotizing deciduitis and intervillositis of both placentas, with greater involvement of twin A's placenta.

COMMENT

Several bacteria are known to cause infection of the fetal compartment by hematogenous spread across the placenta. Maternal bloodborne bacteria are transmitted either by entering the villous space and crossing into the placental villi or by direct spread from infected maternal decidua. *Listeria monocytogenes* is the bacterium most known to cause hematogenous transplacental infection. This organism typically produces a fulminant picture characterized by placental abscess formation, chorioamnionitis, and disseminated fetal infection. Although there are case reports of antibiotic treatment of *Listeria* infection with continuation of the pregnancy,[1] there are no reports of prenatal treatment of *Serratia* infection of the fetal compartment. A MEDLINE search of the English Literature (1966 to February 2005) using a combination of search terms "*Serratia*" and "pregnancy" and "*Serratia*" and "chorioamnionitis" identified 2 reports of *Serratia* chorioamnionitis.[2,3] Both cases resulted in spontaneous abortion.

S marcescens is a gram-negative facultative anaerobe responsible for nosocomial infections of the bloodstream, lower respiratory and urinary tract infections, and infection of surgical wounds of hospitalized patients. It can occasionally be found on the skin of healthy patients, which was the origin of infection in our patient. In one report, *Serratia* bacteremia was believed to be secondary to a chorionic villus sampling 6 weeks before the onset of symptoms.[2] Chorioamnionitis and spontaneous abortion occurred at 21 weeks of gestation after 3 weeks of intravenous antibiotics. In the second report, *Serratia* infection thought to have initially involved the urinary tract led to recurrent bacteremia despite appropriate antibiotic therapy.[3] Because the patient became afebrile immediately after spontaneous abortion, the authors concluded that the chorioamnion served as a site for persistent infection.

Our patient had a peripherally inserted central catheter line infection as the clear source of her septicemia. The recurrence of fevers many weeks after the initial antibiotic therapy was likely due to inadequate treatment. The patient received only 3 days of intravenous antibiotics rather than the recommended 14-day course. We hypothesize that bacteria seeded the placenta early on in the course of her infection, well before her symptoms recurred. This is consistent with hematogenously acquired bacterial chorioamnionitis, which typically has an indolent course. Once a nidus is established in the fetal compartment, intravenous antibiotics have difficulty penetrating the placenta to eradicate the infection. We believe that repeated febrile episodes during her hospitalization despite intensive antibiotics were caused by episodic reseeding of her bloodstream from the placental abscess (a timeline of key clinical events is presented in Fig. 3). Much knowledge has been gained regarding the adverse effects caused by the fetal inflammatory response.[4,5] Twin A died soon after birth with a clinical condition indistinguishable from bacterial sepsis. Negative blood cultures in this neonate could be explained by partial treatment with antibiotics before delivery. Alternatively, twin A's condition could have been caused by the direct effects of fetal cytokines generated in response to maternal infection. Individuals may differ in the intensity of their inflammatory response to foreign antigens. Both fetuses were exposed equally to *Serratia* bacteria, because it was disseminated hematogenously, but one appeared to be relatively unaffected. Long-term follow-up of this infant will be necessary to detect delayed effects of intrauterine inflammation, such as brain injury and cerebral palsy.

The timing of delivery was the most difficult aspect of this case. There is little knowledge on which to support continuation of pregnancy with a placental

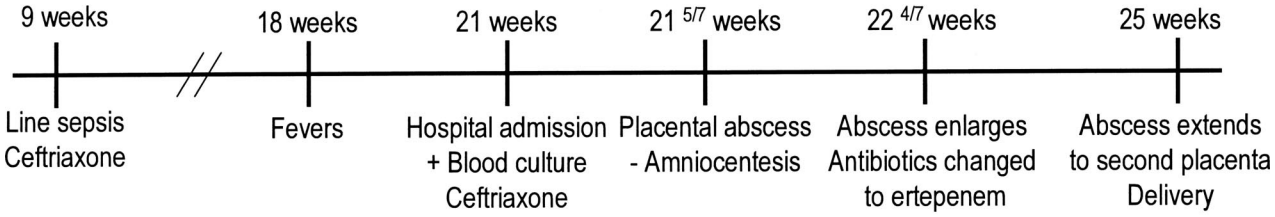

Fig. 3. Timeline depiction of the key clinical events from the initial infection to delivery.
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abscess. The abscess was believed to be the cause of treatment failure and a threat to the mother's health at a time before the fetuses were viable. Therefore, termination of pregnancy was presented to her as the safest option. The mother elected to take the risk of continuing and was encouraged by the negative amniotic fluid studies that she could reach viability. She finally agreed to be delivered at 25 weeks gestation when the abscess seemed to extend into the second twin's placenta.

Intrauterine infection presents a difficult management dilemma when the fetus is very premature. Because inflammation itself is known to cause fetal injury, negative amniotic fluid cultures are not entirely reassuring. Perhaps assessment of amniotic fluid cytokines would assist in the management of these cases.

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Torsion of the Appendix Mimicking Ovarian Torsion

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BACKGROUND: Appendiceal torsion is rare and generally seen more frequently in children than adults. Untreated it can lead to necrosis, ulceration, and subsequent peritonitis.

CASE: A middle-aged female presented with a 5-day history of cramping abdominal pain and nausea and vomiting. Abdominal wall guarding and rebound tenderness was noted on examination. Computerized tomography showed an 8 × 4 cm mass anterior to the uterus, suggestive of degenerating fibroid versus ovarian dermoid cyst. Laparoscopy was performed for presumed ovarian torsion. Torsion of the appendix was discovered and treated by laparoscopic appendectomy.

CONCLUSION: Patients with presumed ovarian torsion should undergo urgent laparoscopy for diagnosis and attempted ovarian salvage. The possibility of conditions that may require different surgical interventions, such as appendiceal torsion, should be considered.

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With only 20 previous cases reported, appendiceal torsion remains a rare and unexplained phenomenon.[1,2] First documented by Payne[3] over 85 years ago, torsion of the vermiform appendix generally mimics acute appendicitis, but in the following unique case, the presentation was most consistent with ovarian torsion.

CASE

A nulliparous middle-aged woman presented to an area hospital with a 3-day history of generalized cramping abdominal pain. Computerized tomography of the abdomen with oral contrast showed a ''4 by 8 cm complex adnexal mass, consistent with a dermoid cyst or a degenerating pedunculated fibroid.'' No ascites was noted. The patient was discharged with narcotic analgesics and told to follow up with her personal gynecologist. The patient subsequently presented to our facility 2 days later with intractable nausea and vomiting. Her pain had become more severe and was now colicky in nature and had localized to the right lower quadrant. She was requiring hydrocodone every 4 hours to control her pain.

Her medical and surgical histories were noncontributory, as were her family and social history. Her obstetric history was significant for 2 elective abortions; her gynecologic history was unremarkable.

Physical examination revealed tenderness to deep palpation in both lower quadrants (right greater than left). She had involuntary abdominal wall guarding and rebound tenderness. Bimanual examination was somewhat limited by pain, but revealed a 10-week size midline, anteverted uterus with abdominal fullness in the lower right quadrant. Evaluation by the General Surgery service was thought to be inconsistent with a nongynecologic etiology. Laboratory results revealed the following data: hematocrit of 34.3%, white blood count of 8.7 with 75% granulocytes, 13.6% lymphocytes, 10.7% monocytes, and 0% bands. Her met-

abolic profile was normal. Repeat computerized tomography again showed an 8 × 4 cm mass anterior to the uterus. Given the report and her nausea and vomiting accompanied by colicky severe abdominal pain not relieved by narcotics, a preliminary diagnosis of adnexal mass with ovarian torsion was made. She was immediately scheduled for diagnostic laparoscopy.

Intraoperative findings revealed normal fallopian tubes and ovaries bilaterally and a 10-week sized midline uterus with multiple leiomyomas. The appendix was identified as an inflamed elongated mass with an area of rupture and extrusion of mucous from the distal tip. The appendix was twisted multiple times around its base in a clock-wise manner (Fig. 1). After the base was ligated with endoloops, the appendix was resected laparoscopically without difficulty and removed from the abdomen in a laparoscopic pouch. The patient's subsequent recovery was uncomplicated.

The pathology report showed a vermicular appendix measuring 12.0 × 5.0 × 4.5 cm, demonstrating both acute appendicitis with recent transmural hemorrhage and evidence of appendiceal rupture. A mucocele with underlying cystadenoma was also noted.

COMMENT

Torsion of the appendix is rare, with only 20 cases previously reported, 12 of these in children.[2] Several theories have been proposed to explain torsion of the vermiform appendix. Anatomic variations, such as long mesoappendix, appendiceal tumors, and vigorous physical exercise have been suggested to predispose to appendiceal torsion.[2] It has long been known that mucinous cystadenoma of the vermiform appendix can predispose to not only appendiceal torsion, but also appendiceal granuloma formation and appendiceal intussusception.

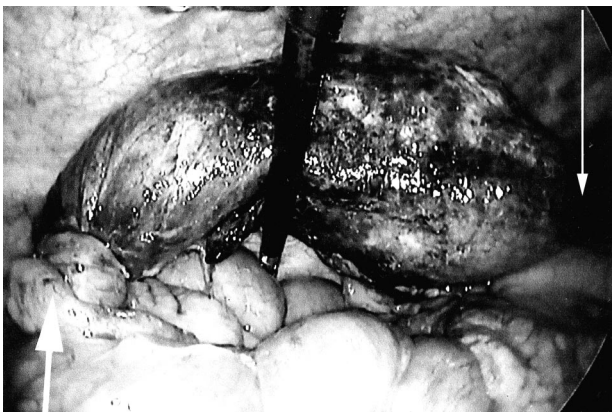

Fig. 1. Large twisted appendix. The *wide arrow* is at the point of clockwise torsion. The *narrow arrow* is at the tip of the enlarged 8-cm long appendix.

Bowling. Torsion of the Appendix. Obstet Gynecol 2006.

Mucinous cystadenoma is the most common mucinous neoplasm of the appendix.[4] It is a rare, yet fortunately, benign tumor. Accumulation of mucous within the lumen of the appendix results from the tumor and, in approximately 20% of the cases, can lead to perforation of the appendix. The growth of the appendiceal tumor can predispose to appendiceal torsion in much the same way as an adnexal mass can predispose to ovarian torsion.[5]

Radiographically, it frequently presents as a globular, broad-based mass invaginating into the cecum and nonfilling of the appendix with oral contrast. On computed tomography the mass shows homogenous content of near water or soft tissue density. On ultrasound examination it appears as a purely cystic mass, as a cystic mass with fine echoes, or as a complex cystic mass with high-level echoes. Peripheral rimlike calcifications may be noted on both computed tomography and ultrasonography.[6]

Ovarian torsion should be considered in any patient with intermittent, acute, lower abdominal pain associated with nausea and vomiting, especially in the face of radiologic studies suggesting an adnexal mass. Patients with a presumptive diagnosis of ovarian torsion should undergo urgent diagnostic laparoscopy for definitive diagnosis and to attempt ovarian salvage. Physicians should be alert to the possibility of other diagnoses, such as appendicial torsion, which may be discovered and which might require surgical interventions other than that planned.

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Acute Abdominal Pain as the Presenting Symptom of Isolated Iliac Vein Thrombosis in Pregnancy

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BACKGROUND: The incidence of iliac vein thrombosis in pregnancy is quite low; however when it does occur, delay in diagnosis can have significant sequelae. We report a pregnant woman who presented with severe acute abdominal pain secondary to isolated iliac vein thrombosis.

CASE: A primigravida presented at 33 weeks of gestation with acute onset of severe left-sided lower abdominal pain. A magnetic resonance imaging study revealed isolated left iliac vein thrombosis. After treatment with heparin her pain completely resolved.

CONCLUSION: Iliac vein thrombosis may cause acute abdominal pain in pregnant women and should be considered in the differential diagnosis when other common causes of pain have been excluded.
(Obstet Gynecol 2006;107:468–70)

Abdominal pain in pregnancy is fairly common and has been associated with gastrointestinal, urological, inflammatory, infectious, gynecologic, and obstetric processes. Although these entities account for the great bulk of cases, other rare causes of pain can be of great consequence if their diagnosis is missed. Because pregnancy is a hypercoagulable state, one such entity is pelvic vein thrombosis. While deep vein thrombosis (DVT) of the lower extremities has long been a concern of physicians, thrombosis of the venous system can occur anywhere and has been reported to occur in the pelvic and ovarian veins.[1,2] The incidence of iliac vein thrombosis in pregnancy is not clearly known, in part because the diagnostic tools needed for a confirmed diagnosis are relatively new additions to clinicians' armamentarium. The potential sequelae of iliac vein thrombosis can be clinically significant and may include thrombosis of major vessels like the inferior vena cava and renal veins, pulmonary embolus, and even death.

CASE

The patient was a 20-year-old primigravida at 33 weeks of gestation who presented with a sharp, constant, and nonradiating severe left lower quadrant abdominal pain (scale 10/10) of sudden onset. The pain was associated with nausea and multiple episodes of nonbloody, nonbilious vomitus. She denied any change in bowel movements or hematochezia. Her pregnancy was otherwise uneventful. She had not undergone any recent surgical procedures, nor had she experienced any recent trauma or period of immobilization. She felt the baby moving, and denied any vaginal bleeding or leaking of fluid. She was not known to have any medical problems. Her family history was only significant for a mother with nephrolithiasis and a father with hypertension.

On arrival her vital signs were stable and she was afebrile. Her examination was positive only for left costovertebral angle tenderness. Abdominal examination was otherwise normal and her height measurement was compatible with her dates. Both lower extremities were normal, with no erythema, swelling, or edema. The pelvic examination was unremarkable, with no cervical changes. The fetal heart tracing was reactive and there were no contractions. Blood tests including complete blood count, chemistry, and liver function tests were normal. Urinalysis revealed few red blood cells and few white blood cells, and urine culture results were negative. The patient was admitted for presumed nephrolithiasis and treated with intravenous fluids and morphine.

An abdominal ultrasonogram showed no ovarian masses and a normal gallbladder and appendix. The renal ultrasonogram was normal, with no hydronephrosis and no ureteric or kidney stones. She required frequent doses of morphine for pain (almost every 3 hours), from which she obtained only mild relief. After consultation with a Maternal–Fetal Medicine specialist and a gastroenterologist, a magnetic resonance imaging study (MRI) was performed to assess the possibility of bowel or vascular pathology (for example, bowel ischemia or mesenteric thrombosis) because the abdominal pain seemed to be out of proportion to the physical findings. The scan revealed a left iliac vein thrombosis; the bowel itself was unremarkable with no evidence of obstruction or ischemia. To delineate the extent of the iliac vein thrombosis, Doppler studies were performed that demonstrated no evidence of DVT in the lower extremities or in the visualized left iliac vein. However, the most superior aspect of the left iliac vein was not visible and therefore DVT in that region could not be excluded. Intravenous heparin was started and vascular surgery was consulted. Two days after starting heparin, the patient's symptoms improved dramatically and then disappeared on day 3. The vascular surgery team inserted an inferior vena cava filter to prevent the occurrence of a pulmonary embolism. The patient was discharged on low molecular

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weight heparin on hospital day 8. Thrombophilia workup, including factor V and prothrombin mutation, antithrombin III and protein C activity, anticardiolipin antibody, and homocysteine assay, was normal, with protein S activity found to be 51% (60–140% is the normal nonpregnant range, with normal pregnancy values reported to be lower). The patient returned with spontaneous rupture of membranes at 34 weeks. She was delivered by cesarean section for nonreassuring fetal electrocardiograph and the neonate did very well. There was no evidence of placental abruption or intrapartum hemorrhage. The patient has been followed up postpartum by vascular surgery and she is currently on oral anticoagulation.

COMMENT

Abdominal pain is one of the most common complaints in pregnancy. The differential diagnosis of acute abdominal pain in that setting includes appendicitis, cholecystitis, gastroenteritis, bowel obstruction, renal calculi, pancreatitis, pyelonephritis, and ulcer disease. Less commonly, adnexal torsion, hemorrhagic cyst, red degeneration of fibroid, placental abruption, and hemolysis, elevated liver enzymes, low platelets syndrome can cause abdominal pain in pregnancy.[3] Given that pregnancy is a hypercoagulable state, other diagnoses need to be considered, especially when none of the more common entities explain the patient's discomfort.

Isolated pelvic DVT is uncommon, with the reported incidence varying from less than 2% to 4%.[4,5] Although DVTs of the lower extremities and subsequent pulmonary embolism in pregnancy have always been a concern for obstetricians, they may be less aware that isolated iliac vein thromboses can also occur. We performed a MEDLINE and OVID search between the years 1966 and 2005 with the key words "iliac vein thrombosis and pregnancy" and "abdominal pain and pregnancy." Although approximately 40 cases of isolated iliac vein thromboses have been reported in pregnancy, to our knowledge this is the first pregnant patient reported with acute abdominal pain resulting from isolated iliac vein thrombosis with no lower extremity symptoms, ie, swelling, pain, or skin discoloration. Fogarty et al[6] and Cegelski et al[7] reviewed 14 antepartum patients with iliofemoral venous thrombosis, but all had lower extremity symptoms and signs that led to the diagnosis.

In our case, no predisposing factor was identified apart from pregnancy, and there were no clinical findings that suggested the presence of DVT. This led us to evaluate the retroperitoneal space and to look for less common causes of abdominal pain using MRI. A duplex Doppler ultrasound study of the lower extremities performed afterward failed to identify any thrombosis in either lower limb or pelvis. Duplex Doppler ultrasound, due to its relatively low cost and ease of use, has been established as the method of choice for screening patients with acute DVT; however, it is known to be unreliable in demonstrating thrombosis of the pelvic veins.[5] Therefore, it is not surprising that it failed to reveal the thrombosis in our patient. The use of MRI to identify pelvic vessel thrombosis has been described. Dupas et al[8] reported on 25 patients who underwent MRI venography, duplex and color Doppler ultrasonography, and ascending venography of the pelvic and common femoral veins and found that MRI venography was 100% sensitive and 98.3% specific, whereas ultrasonography was only 90.5% sensitive and 96.7% specific. In that study, color duplex ultrasonography was unable to depict any internal iliac veins, including each of the 8 that contained clots. In another study, Spritzer et al[9] reviewed 34 cases of isolated pelvic vein thrombosis detected with MRI venography. In 7 of these cases, duplex Doppler ultrasonography had been performed within 24 hours before the MRI; all 7 were negative for acute DVT by ultrasonography.

The treatment of choice for DVT of the iliac veins is still controversial. It has been suggested that it should include heparin; however, other treatment modalities such as thrombolysis have been used in pregnancy and found to be safe.[10] Whether heparin is sufficient to prevent pulmonary embolism in pregnancies complicated by isolated iliac vein thrombosis remains to be determined. This report reinforces the need to consider pelvic vein thrombosis in the differential diagnosis of abdominal pain in pregnancy when the more common causes have been excluded.

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Inadvertent Vesicular Placement of a Vaginal Contraceptive Ring Presenting as Persistent Cystitis

Stephanie B. Teal, MD, MPH, and Winfield M. Craven, MD

BACKGROUND: The NuvaRing is a soft, flexible, ring-shaped vaginal contraceptive device that is placed by the user herself. Incorrect placement has not been described, because any intravaginal position allows appropriate hormonal delivery through the vaginal mucosa.

CASE: A 22-year-old otherwise healthy woman presented with 2 months of urinary urgency, frequency, and pelvic pain, which were unresponsive to antibiotic therapy. Her symptoms began immediately after placement of a NuvaRing vaginal contraceptive device, which she was subsequently unable to locate. Thorough evaluation revealed the ring in the urinary bladder.

CONCLUSION: Bladder or urethral foreign body should be considered in the evaluation of patients with chronic cystitis, especially if the patient uses vaginal medical devices. Intravesicular placement of a device may occur even without psychiatric or physical comorbidities.
(Obstet Gynecol 2006;107:470–2)

The vaginal mucosa provides an excellent route for steroid absorption.[1] Several intravaginal ring delivery systems have been developed for both hormone replacement therapy and contraceptive indications. This type of delivery system allows convenient, long-acting hormonal therapy under the patient's own control. The rings are soft, flexible devices made of various polymers. The NuvaRing (Organon, Roseland, NJ) combined hormonal contraceptive device specifically is composed of ethylene vinyl acetate, and has an outer ring diameter of 54 mm with a cross-sectional diameter of 4 mm. It is placed by the patient herself each month, and is removed by the patient after 21 days of use. We report a case of a contraceptive vaginal ring device inadvertently placed by a patient into the bladder through the urethra.

CASE

A 22-year-old woman, gravida 2 para 2, presented to her family health center complaining of 2 months of urinary frequency, dysuria, and suprapubic pain. She described the dysuria as feeling like glass was coming out of her urethra at the end of each void. The patient stated all these symptoms began following the insertion of a NuvaRing contraceptive device. Her medical history was significant only for 2 prior cesarean deliveries, and one-half pack per day tobacco use. She used no medications other than the NuvaRing. She had used the NuvaRing for 3 prior cycles without complication, although she noted some discomfort with placement each month. Two months before presentation, she placed the device and felt a ''pinching'' sensation with insertion. She attempted to check the position, and was not able to find the device in her vagina. She looked all over the bathroom floor for it, and worried that it had entered her cervix and was intrauterine. Approximately 10 minutes later, she developed urinary urgency, and then a pressure-like pain in the lower abdomen. She presented to the emergency department at her local hospital and underwent pelvic examination and urinalysis, both of which showed negative results. She was reassured that the device must have fallen out of her vagina without her notice, and she received no treatment.

Two days later, the patient was seen at the local health department clinic, where she was diagnosed with an uncomplicated cystitis, and treated with trimethoprim and sulfamethoxazole and phenazopyridine. She received a replacement NuvaRing which she placed without incident. She remained convinced the device was ''somewhere inside'' her. Her symptoms did not improve, and she received subsequent treatment for urinary tract infection from the same clinic. She presented to her family health center 6 weeks later with persistent symptoms and was found to have a urinalysis ''positive for urinary tract infection'' and significant suprapubic tenderness to palpation. She was treated with 10 days of ciprofloxacin and 2 days of phenazopyridine. Her urine culture was negative. She returned to clinic 2 weeks later for follow-up with persistent

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symptoms and was given a pelvic examination that confirmed the suprapubic and bladder tenderness without other significant findings. A transvaginal ultrasonogram was obtained with the bladder empty, which was read as normal. Because of the persistence of the symptoms, computed tomography imaging studies, without and with contrast, were performed; both showed a ring-like lucency within the urinary bladder (Fig. 1). The patient was referred to a urologist, who performed office cystoscopy under local anesthesia. He noted a normal-appearing urethral meatus, normal urethra, a NuvaRing device in the bladder, and no bladder mucosal defects, ulcerations, or inflammation. The device was easily extracted with a single pull. The patient's symptoms subsequently resolved. She changed her contraceptive method to combined oral contraceptive pills.

COMMENT

Almost all reported bladder foreign bodies result from either intentional transurethral placement of various objects or erosion of a medical device originally placed in the vagina, uterus, or abdominal cavity. The literature is replete with reports of intraurethral placement of foreign bodies for sexual stimulation, in cases of coexisting psychiatric disease, or due to childhood curiosity.[2] Particularly, perforation of the uterus at the time of intrauterine device placement with subsequent bladder migration of the intrauterine device has been extensively reported.[3] Erosion into the urethra or bladder of medical devices such as tension-free vaginal tape, suture, bolsters used at bladder neck suspension procedures, and Filshie sterilization clips has also been described.[4–6] This is the first report of inadvertent placement of a steroidal vaginal ring device directly into the urinary bladder. This patient had normal pelvic anatomy, no history of urologic or perineal surgery, no history of psychiatric conditions, and had used the ring successfully for several cycles before this incident. The patient immediately noted the absence of the device from the vagina and developed lower urinary tract symptoms soon thereafter, but the possibility of intravesicular placement was not considered.

Although uncomplicated cystitis may be treated empirically, persistent symptoms after treatment failure require a urine culture be done. Culture-negative urgency and frequency symptoms may result from bladder malignancy, bladder calculi, distal ureteral calculi, neurogenic bladder, overactive bladder, radiation cystitis, interstitial cystitis, urinary retention, or an externally impinging pelvic mass. Clinicians should be aware that foreign body also must be considered in the differential diagnosis of persistent lower urinary tract symptoms, especially in patients using intravaginal medical devices. This diagnosis must be entertained even for otherwise mentally and physically healthy individuals.

Review of the literature revealed 2 other cases of accidental urethral insertion of a medical device intended for the vagina: a slim-fit tampon placed into the bladder by a 14-year-old girl with a partially imperforate hymen[7] and urethral insertion of a contraceptive spermicidal suppository.[8] No reports of inadvertent placement of contraceptive diaphragms, sponges, the female condom, pessaries, or other vaginal rings were found. Like the slim-fit tampon, the NuvaRing can be compressed to just less than 1 cm total cross-sectional diameter, and this may have contributed to this patient's ability to place it into the bladder with minimal initial discomfort. The vaginal mucosa is an excellent site for steroid absorption, and more vaginal rings are being developed as delivery systems, increasing the likelihood that mishaps of this type may occur.

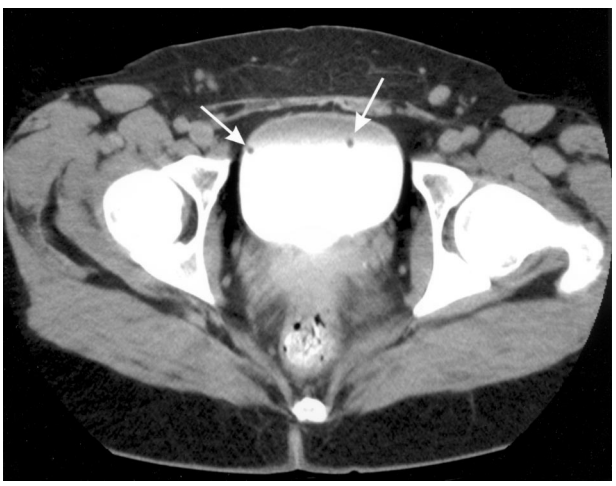

Fig. 1. A computed tomography image of the patient's pelvis after intravenous contrast, showing the intravesicular contraceptive ring in cross-section *(arrows).*

Teal. Intravesicular Contraceptive Ring. Obstet Gynecol 2006.

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Five Cases of Tape Erosion After Transobturator Surgery for Urinary Incontinence

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BACKGROUND: Before introducing the transobturator tape into our practice we undertook 52 transobturator tape procedures. The transobturator tape procedures were undertaken with an "outside-in" approach, using nonwoven polypropylene mesh with average pore size of 50 μm.

CASES: Five cases of vaginal erosions have been identified, 1 complicated by a groin abscess. All cases required further procedures to trim (n = 3), resect (n = 1) or remove (n = 1) the tape. One woman had a tension-free vaginal tape procedure. To date, 3 women remain incontinent.

CONCLUSION: Possible reasons for the complications include 1) surgical inexperience (unlikely, given that we have undertaken more than 2000 tension-free vaginal tape procedures without similar complication rates); 2) inherent susceptibility of the "hammock" position of the transobturator tape; or 3) the nonwoven polypropylene tape with mesh size of 50 μm itself may predispose to erosion or abscess.

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The transobturator tape procedure was developed by Delorme[1] in 2001 as a new minimally invasive procedure for urinary stress incontinence. Its proposed advantage over the tension-free vaginal tape (TVT) was that by avoiding the transpelvic introduction there would be a decrease in significant complications, such as bladder, bowel, and vascular injury.[1] Because it is also a tension-free approach, theoretically, transobturator tape would have the same continence outcomes as the established tape procedures such as TVT.

Before introducing transobturator tape into general use in our practice, the members of the Division of Pelvic Floor Disorders and Reconstructive Surgery decided to evaluate the product. After training in a cadaver laboratory and appropriate preceptoring, 4 surgeons undertook 52 transobturator tape procedures as part of the surgical quality assurance program. All 52 women had stress incontinence diagnosed by examination and subtracted urodynamic studies.

The transobturator tape procedure was undertaken with an "outside-in" approach using a nonwoven polypropylene mesh with average pore size of 50 μm (Obtape, Mentor-Porges, Le Plessis Robinson, France). No intraoperative or immediate postoperative complications were reported. All procedures were performed under local anesthesia with sedation. All patients received antibiotics intraoperatively and had normal cystoscopic examination. Follow-up was to be undertaken at 6 weeks, and 6 and 12 months. Additional treatments (eg, antibiotics) were used at the discretion of the treating physician.

We report 5 cases of vaginal erosions, 1 complicated by a groin abscess, that occurred in this cohort of women. Written consent was obtained from all women with complications.

CASE 1

A 37-year-old with a previous abdominal hysterectomy and failed Marshall-Marchetti-Krantz procedure with porphyria cutanea tarda had an uneventful transobturator tape procedure for her mixed incontinence. She failed to attend clinic for her postoperative visit but reported continued incontinence. The patient presented to her local community hospital 10 months after surgery, with a 1–2 month history of thigh pain and continuing incontinence. During the previous week the area had become swollen and erythematous. She also reported intermittent fevers and sweating and symptoms suggestive of a urinary tract infection (UTI), which had been treated

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with antibiotics. A large 6 cm × 16 cm abscess was seen on computed tomography scan, extending through the obturator foramina into the obturator internus muscle and into the adductor muscles. Under general anesthetic the thigh abscess was incised and drained of a large amount of purulent discharge and irrigated with normal saline. Two Penrose drains were placed, and the patient was treated with clindamycin every 6 hours. Thirteen months after the original surgery, the patient presented again, this time with a tape erosion from the midline to the left obturator space. She was taken to the operating room, and under general anesthesia the tape was removed through the vaginal wound. The tape was easily pulled free from the left side and was pulled from the right side with more difficulty. The patient was given cefazolin and metronidazole intraoperatively and then ciprofloxacin and metronidazole for 7 days. Culture of the tape found heavy mixed gram-positive and gram-negative organisms and heavy mixed anaerobes. At 15 months after the original procedure she returned to the clinic with ongoing incontinence suggestive of a mixed type. The vagina was well healed with no evidence of scarring. The thigh abscess was also well healed. A diagnostic cystoscopy was undertaken. The patient had a postvoid residual of 900 mL and severe stress leakage with coughing. There were no significant urge symptoms. The patient has an atonic bladder with overflow incontinence and we planned video urodynamic assessment.

CASE 2

A 49-year-old woman with previous hysterectomy and not on any hormone replacement therapy underwent an uneventful initial procedure for stress incontinence. At 6 weeks postoperatively, the patient was continent and the examination was normal. At 14 weeks postoperatively the patient returned with complaints of postcoital spotting. Examination revealed a small midline erosion. The tape seemed loose and nonscarred. The patient underwent a tape revision and repair of the vaginal erosion in the operating room with local anesthesia and intravenous sedation. The tape was trimmed under traction through the vaginal wound. Examination at 21 weeks after the original transobturator tape procedure was normal. At 26 weeks, the patient had mild recurrent stress incontinence.

CASE 3

A 45-year-old woman with a previous hysterectomy and taking daily oral estradiol underwent an uneventful procedure for stress incontinence. Two passes through the left obturator foramina were required. On postoperative day 2, the patient sneezed, felt a sharp pulling pain in the right groin, and began having mild stress incontinence. At the examination 7 weeks postoperatively, the tape could be felt by palpation, but there was no erosion or pain at the site. The patient reported ongoing pain deep in the pelvis and a pulling sensation in the right groin. A cystoscopy and examination were undertaken at 10 weeks postoperatively, due to persistent microscopic hematuria. The cystoscopy showed no abnormality apart from recurrent stress incontinence. The patient visited her family doctor with vaginal bleeding at 21 weeks postoperatively. He called the surgeon to report a loop of tape present in the vagina. The patient returned to the surgeon's office with the right half of the tape (approximately 4 cm) extruding through the mid vagina. She also reported having been treated for UTIs 3 times since her last visit. The tape was trimmed under traction in the office with no antibiotic use. Approximately 5 cm of tape was removed. The patient experienced no further UTIs after the tape was removed, and underwent a successful TVT procedure 22 weeks after the initial surgery. She attended clinic at 40 weeks after the initial transobturator tape procedure and was continent at that time. She experienced no further UTIs.

CASE 4

A 47-year-old woman on oral contraceptive pills for dysfunctional uterine bleeding with mixed incontinence and a previous failed Burch urethropexy underwent an uneventful procedure. At 6 weeks postoperatively she was continent and experiencing no problems. Routine examination revealed a 3 mm × 5 mm erosion to the right of midline that was treated with conjugated equine estrogen cream. At 11 weeks postoperatively she was taken to the operating room, where 2 cm of unincorporated mesh was trimmed in the midline under local anesthetic with sedation. The vaginal epithelium was resutured in the midline. Antibiotics were administered in the operating room. A return appointment at 20 weeks showed approximately 4 cm of mesh extruding from the left side of the midline. It was trimmed under traction in the office with no antibiotic coverage. The patient reported a return of mild stress urinary incontinence symptoms at this visit but declined further intervention.

CASE 5

A 54-year-old woman with previous hysterectomy and taking daily oral estradiol underwent an uneventful transobturator tape procedure for stress incontinence. At 8 weeks postoperatively she complained of a small amount of foul vaginal discharge and spotting. Examination revealed an extrusion of the tape in the mid part underlying the vaginal wound. She was treated with 2 weeks of oral antibiotics and local conjugated equine estrogen cream. On reexamination the erosion was unchanged, and she was booked for a surgical revision. Throughout this time she remained continent. At 12 weeks postoperatively she was taken to the operating room, and under local anesthetic with sedation the vagina was undermined, and the vaginal wall was reapproximated. At 20 weeks postoperatively from the initial surgery she again went back to the operating room for an excision of 1 cm of tape that had eroded through the vagina. The vaginal wound was reclosed. She received antibiotics each time. She remained continent 6 weeks after her last surgery.

COMMENT

To date, despite the gaining popularity of transobturator tape, there have been no North American reports of vaginal erosion or abscess. Our literature search used MEDLINE, employing the terms "complication," "obturator," and "incontinence," from January 1966 to May 2005. Five articles reporting erosions and inguinal abscesses after transobturator tape procedures have been published by European authors.[2–6] Four of the complications (in 1 report) occurred with the same tape as we used (Obtape),[2] and 9 (in 4 reports) with a similar nonwoven polypropylene tape with a suburethral silicone modification (Uratape, Mentor-Porges).[2–5] The fifth report involved 2 cases where a knitted polypropylene mesh was used (Monarc, American Medical Systems, Minnetonka, Minnesota).[6]

In our small study, we have therefore reported a high rate of vaginal tape erosion (10%) and abscess formation (2%), similar to the rates reported by surgeons in Spain.[2] The explanation for our unexpected finding could be surgical inexperience. This explanation is unlikely because we have undertaken over 2,000 TVT procedures during the past 4 years without similar complications, and our complications were not clustered at the start of the transobturator tape series. The low rate of reporting of transobturator tape complications by others is likely due to underrecognition or underreporting.

We suggest that our complications may be the result of 2 explanations. First, there may be inherent susceptibility of the transobturator approach to erosion due to the "hammock" positioning allowing more tape to be in close apposition to the vaginal wall. It is possible that this positioning may be more susceptible to disruption during sexual activity, and this explanation would tie in with the report by But[6] of an abrasion of a husband's penis caused by tape that had become exposed.

Second, the nonwoven polypropylene tape itself may predispose it to erosion or abscess. This could be due to the nonknitted, nonwoven mesh with small pore size (< 50 μm.), which theoretically may restrict the passage of macrophages and fibroblasts.[7] The characteristics of the tape are implicated in the cases reported by Domingo et al.[2] In our cases, restriction caused by small pore size would account for the observation that when tape erosions were diagnosed, there was no evidence of tissue ingrowth into the mesh.

The importance of sling erosion as a potentially devastating complication after sling procedures for stress urinary incontinence is well recognized, although uncommon, with an incidence between 0.3% and 4.4%.[8] Our observed complication rate after transobturator tape is a source of concern, and prolonged follow-up of our cohort of 52 patients will identify the incidence of late erosions and other complications. In addition, we are currently planning a randomized trial comparing outcome of transobturator tape with TVT. We believe that careful evaluation of new technologies is essential before adopting them into clinical practice.

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