Postpartum Pyometra: A Case Report

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Pyometra is an uncommon result of endometritis. The diagnosis of pyometra is made when a collection of pus is found within the endometrial cavity. Antibiotic administration and drainage of the pus are essential to resolve this condition. A report of a case of pyometra is presented and a technique for drainage is described. This case demonstrates the use of office-based diagnos-

Physicians who provide perinatal care can expect to treat postpartum endometritis (PPE), which is the most common infectious complication in women following childbirth.¹ Incidence of postpartum fever and endometritis varies from 3% to 4%² to as much as 16.5%³ of puerperae. Cesarean section continues to be the leading predisposing factor; however, duration of labor, rupture of membranes, and internal fetal monitoring are considered by some to be additional precipitating factors of PPE.^{1,3} Antibiotic treatment results in clinical cure in 85% of PPE cases and in 90% with postoperative infections, including infections after cesarean section.⁴ Despite this success, more serious sequelae of PPE include septic pelvic thrombophlebitis, wound infection with dehiscence, and pelvic abscess.¹

Pyometra, a collection of pus in the uterine cavity, is rare, with a reported incidence of 0.5% in gynecologic patients, including those with cancer.⁵ The etiology of pyometra is varied, as it is associated with any condition causing cervical occlusion.^{6,7} The most common cause is malignancy of the uterus and true pelvis; other causes include benign tumors of the pelvis (leiomyomata, polyps), traumatic operations on the cervix (conization), radiation cervicitis, atrophic cervicitis, congenital anomalies, and puerperal infection.^{6,7} The typical symptoms of

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tic ultrasonography to narrow the differential diagnosis and guide the drainage procedure, as well as the use of simple ingenuity to improve patient care.

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pyometra include uterine enlargement, cramping, vaginal discharge, acute abdominal pain, and postmenopausal bleeding.⁸ Spontaneous rupture of a pyometra is an uncommon complication.^{8–11}

Diagnostic ultrasonography has made this difficult diagnosis possible without surgical intervention.⁷ This report describes a case of pyometra in a patient being followed for postpartum endometritis and the apparatus used to drain it in an outpatient setting under ultrasonographic guidance.

Case Report

A 29-year-old woman, gravida 4, para 3, aborta 1, gave birth spontaneously at term without anesthesia to a 3095-g male infant with Apgar scores of 8/9. Her labor lasted 3.5 hours, and her membranes had been ruptured for only 45 minutes before delivery. The third stage of labor was complicated by retained placenta, which was removed manually and with curettage. She went home afebrile with her infant on the 2nd postpartum day.

The patient went to an emergency department on the 7th postpartum day when her lochia had developed a foul smell. A clinical diagnosis of endometritis was made, and she was treated with oral doxycycline. On the 9th postpartum day she was seen in the family practice center for "bleeding." On examination, no signs of an acute abdomen were found, but a thin, brown, foul-smelling cervical discharge was seen and her uterus was "exquisitely tender." Her oral temperature was 100°F and her



Figure 1. Sagittal view of the uterus and overlying bladder obtained using transabdominal ultrasonography on the 16th day postpartum. The uterus contains a 1.4 cm \times 5.1 cm sonolucent area with a volume of about 10 mL. This appearance is most consistent with fluid (abscess or liquefied blood) rather than retained tissue.

cervix appeared to be open 2 to 3 cm. An endocervical culture was obtained, and the patient was admitted to the hospital for intravenous antibiotic therapy. Her leuko-cyte count was 10,200/mm³ with 79% segmented neutrophils.

After 3 days of antibiotic therapy with ampicillin, gentamycin, and metronidazole, her leukocyte count fell to 9,100/mm³, her temperature returned to normal, and her pain subsided. Cultures that had been obtained at the family practice center grew group B β -hemolytic streptococci sensitive to ampicillin and resistant to tetracycline. Blood and urine cultures were negative. A diagnostic ultrasound scan showed evidence of a "small hematoma" within the uterus. She was discharged and instructed to continue taking ampicillin and metronidazole.

At the time of follow-up in the family practice center on the 16th postpartum day, she was found to have a "globular uterus" and purulent cervical discharge but no fever. A diagnostic ultrasonographic examination was done in the family practice center; it revealed a lucent area within the uterus consistent with approximately 10 mL of fluid. There were no echogenic components within the uterus, suggesting that this was more likely to be blood or a pyometra than retained products of conception (Figures 1 and 2). The clinical judgment was made that draining this material would be helpful diag-



Figure 2. Transverse view of the intrauterine fluid collection, 2.2 cm in diameter, obtained using transabdominal ultrasonography on the 16th day postpartum.

nostically and therapeutically, and would relieve the patient of her unpleasant discharge.

Method of Drainage

A method to drain this material was sought that would be easily available and relatively atraumatic. The apparatus had to be large enough in diameter to permit drainage of thick fluid but small enough to avoid discomfort to the patient. Infant feeding tubes and urinary catheters would have been too flexible to be threaded through the cervical os and could not be hooked up easily to a syringe for aspiration of thick material. A spinal needle would have been traumatic and would not have permitted thick material to be aspirated.

As an alternative, an endometrial biopsy tube (Pipelle, Unimar, Inc, Wilton, Conn) was altered as follows (Figure 3):

- 1. The plunger from the tube was removed by cutting the end of the tube at which it was sealed in place.
- 2. The cut end of the tube was heated in an alcohol lamp flame, resulting in softening and expansion of the plastic.
- 3. An 18-gauge needle was threaded into the cut end of the Pipelle tube, and the softened plastic of the tube was pressed around the needle hub creating a tight seal. Sterility was maintained during this procedure.

Figure 3. The apparatus was fashioned from an endometrial biopsy tube (Pipelle) and used to drain the pyometra. The endometrial biopsy catheter was fused to a needle and hub.

The Pipelle catheter with the needle hub was attached to a 20-mL syringe. After the cervix and vagina were swabbed with povidone iodine, the apparatus was threaded through the cervical os, causing no discomfort to the patient. This was done under ultrasonographic guidance demonstrating that the uterus was not perforated. Approximately 10 mL of thick yellow purulent material was aspirated from the uterus and sent for aerobic and anaerobic culture. Simultaneous visualization with ultrasonography made it possible to verify that all fluid within the uterus had been removed (Figure 4).

Resolution

The patient was discharged from the family practice center and instructed to continue taking ampicillin and metronidazole. The material evacuated from the uterus grew no organisms on culture. The patient was seen again in the family practice center on the 29th postpartum day. Her uterus was smaller, there was no abnormal discharge, and she had no residual pain.

Discussion

Diagnostic ultrasonography facilitates the diagnosis of fluid or tissue accumulation within the uterus. The sonographic hallmark of an empty uterus is a bright endometrial stripe, a result of the echo from the opposed anterior and posterior uterine walls. Sonolucent accumulations and echogenic material in the uterus thicker than 5 mm

are highly predictive of retained products of conception after first trimester pregnancy loss.¹² Small endometrial fluid accumulations in the uterus postpartum are not associated with maternal morbidity, but an echogenic mass indicates retained products of conception.¹³

This patient was predisposed to endometritis because her uterus was explored and instruments were used



Figure 4. Sagittal view of the uterus after the drainage procedure. Transabdominal ultrasonography was used to confirm an empty uterus.

to retrieve the retained placenta. Curettage may have predisposed her to cervical canal obstruction. There was spontaneous drainage at the time she presented with endometritis, however, and the cervix appeared to be open.

Three interventions might have prevented the complications that this patient experienced. First, antibiotic prophylaxis after the manual removal of the retained placenta might have prevented the endometritis. The fact that the cultures grew group B β -hemolytic streptococci suggests that the patient may have suffered an autoinfection, as this pathogen is known to colonize the cervix. Second, an endometrial culture (rather than endocervical) at the time the endometritis was diagnosed might have opened enough of a drainage pathway to prevent pyometra formation. Third, when the ultrasonographic examination done in the hospital showed fluid within the uterus, a drainage procedure similar to that described here could have been done.

Simple drainage of this patient's pyometra in the outpatient setting was appropriate because her infection had been treated, her clinical symptoms had improved, and she was afebrile. The fact that the pus was sterile on culture showed that the infection had been adequately treated. Had she been acutely ill, or had not yet received antibiotic treatment, drainage of the pyometra in the hospital followed by treating the patient with intravenous antibiotics would have been more appropriate. In that case, repeat sonography with or without leaving a drainage catheter (Foley or mushroom type) in place would have been helpful to make sure the pyometra would not reaccumulate.

This case illustrates the use of a simple, inexpensive, and safe method for draining a pyometra in the physician's office. It also illustrates how commonly available materials can be adapted to a unique situation and how office-based diagnostic ultrasonography can be used to improve patient care.¹⁴

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Mesenteric Venous Thrombosis: A Case Report

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Mesenteric venous thrombosis is an uncommon entity. The preoperative diagnosis is largely clinical; the hallmark is pain that is out of proportion to the physical findings. Treatment consists of thrombectomy with resection of necrotic small bowel and mesentery. In the absence of trauma or infection, an investigation of in-

A complaint of acute diffuse abdominal pain can be a diagnostic dilemma, especially when the subjective symptoms appear to be out of proportion to the findings on physical examination. Ischemic bowel disease often presents in a rather benign manner yet has significant morbidity and mortality if not diagnosed and managed promptly. Mesenteric venous thrombosis, presenting as ischemic bowel disease, has the same poor prognosis if not aggressively diagnosed and treated. An intrinsic anticoagulant deficiency state because of antithrombin III, protein C, or protein S deficiency may be associated with mesenteric venous thrombosis. As these disorders are inherited in an autosomal dominant pattern and increase significantly the risk for recurrent venous thrombosis, an awareness of these deficiencies is important. The following case of ischemic bowel syndrome due to mesenteric venous thrombosis is presented, followed by a discussion of inherited intrinsic anticoagulant deficiency states.

Case Report

A 57-year-old man previously in good health came to the emergency department complaining of an insidious onset of anorexia and severe belching accompanied by dull generalized abdominal pain. He had no past surgical history. He denied nausea, vomiting, diarrhea, constipa-

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trinsic anticoagulant deficiencies is warranted since these deficiencies are inherited in an autosomal dominant fashion. Treatment is warfarin sodium therapy.

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tion, fever, flatulence, melena, or hematochezia. He had eaten four large bowls of cabbage and onions the night before.

The patient's temperature was 36.1°C (97°F), blood pressure 150/92 mm Hg, pulse 80 beats per minute, and respirations 20 per minute. A physical examination revealed a soft, obese, rotund abdomen with good bowel sounds in all four quadrants. There was generalized tenderness without rebound or guarding. There were no masses and no hepatosplenomegaly. Psoas muscle and obturator signs were negative. Rectal examination was normal and a guaiac test was negative. Findings on examination of the patient's head, eyes, ears, mouth, throat, neck, heart, lung, and extremities were normal.

An electrocardiogram showed normal sinus rhythm with a rate of 50 beats per minute. A complete blood count showed a white blood count of 13,800/mm³ (13.8 \times 10⁹/L) with a differential of 91% segmented neutrophils, 8% lymphocytes, 1% monocytes, hemoglobin 15.9 g/dL, hematocrit 42.6%, and platelets 249,000. Electrolytes (including bicarbonate), the calculated anion gap, SGOT, SGPT, and amylase levels were normal. The alkaline phosphatase level was 113 U/L (normal 30 to 103 U/L). A urinalysis was remarkable for a specific gravity of 1.030, pH 5.0, ketones 2+, and bilirubin 1+. A chest radiograph was normal. Abdominal films showed a large amount of stool and gas in the right colon with no gas in the left colon or sigmoid.

The patient was observed in the emergency department for 2 hours; two attempts at nasogastric tube placement were unsuccessful. He was feeling moderately

Causes of Mesenteric Venous Thrombosis

Trauma	Postoperative abdominal surgery
Sepsis	Oral contraceptives
Intraabdominal abscess	Polycythemia
Peritonitis	Thrombocytosis
Volvulus	Antithrombin III deficiency
Intususception	Protein C deficiency
Compressive neoplasms	Protein S deficiency

better and was discharged with a bisacodyl suppository, simethicone tablets, and an order for a liquid diet. He returned to the emergency department within 3 hours with similar generalized abdominal pain and a continued paucity of findings on physical examination.

A surgeon was consulted, and after 12 hours of observation, the pain consistently worsened despite intravenously administered (IV) narcotics. The patient was afebrile throughout this time. His white blood count rose to 19,800/mm³ (19.8 \times 10⁹/L) with a differential of 83% segmented neutrophils, 3% bands, and 13% lymphocytes. An exploratory laparotomy was performed. A 130-cm segment of infarcted jejunum was resected with subsequent side-to-side anastomosis. A 48-hour "second look" laparotomy was performed and an additional 400 cm of infarcted small bowel was removed. Postoperatively the patient required massive volume replacement, IV antibiotics, and monitoring in the intensive care unit, but he recovered completely, returning to work and normal activities within 1 month. He was treated postoperatively with the anticoagulant heparin and maintained with warfarin therapy. The pathology examination reported infarcted bowel with blood clots in the mesenteric veins.

Discussion

The causes of mesenteric venous thrombosis (MVT) can be categorized into trauma, mechanical, infection, and hematologic disorders (Table). Mesenteric venous thrombosis has occurred in women taking oral contraceptives and in pregnant women¹; MVT has been associated with sepsis, abdominal abscesses, and peritonitis. Blunt abdominal trauma may result in MVT. Mechanical problems that can be accounted for in association with MVT include volvulus, intususception, compressive neoplasms, and postoperative abdominal surgery. In the absence of the above-mentioned identifiable factors, hypercoagulable states, either intrinsic or induced, account for most cases of MVT, especially in recurrent cases of venous thrombosis. These causes include polycythemia, thrombocytosis, antithrombin III deficiency, and protein C or protein S deficiency.

The incidence of MVT is reported to be 1.5% of the population in autopsy studies,² and accounts for 0.01% to 0.06% of all causes of intestinal infarction and 5% of cases without mechanical causes. Mesenteric venous thrombosis is more common in the sixth and seventh decades of life.³ The superior mesenteric vein is most commonly involved, resulting in edematous and hemorrhagic bowel.⁴ The large bowel is rarely involved.

The hallmark of MVT is generalized severe crampy abdominal pain that is out of proportion to physical findings. Historically, a previous occurrence of spontaneous venous thrombosis should raise suspicion. Nausea, vomiting, and diarrhea are inconsistent findings. Matthews and White⁴ reported that over half of the patients in their study had experienced pain for 5 days to 1 month before medical evaluation was sought. Hematemesis and hematochezia have been found with MVT, but these are rare and represent advanced ischemia and necrosis. Massive colonic dilatation has also occurred with MVT.

Typically, there is abdominal pain on examination without rebound or guarding. Pain usually persists after administration of narcotics.⁴ Stools were positive for blood in 80% to 100% of patients in three separate studies.⁴ Fever is usually absent or low grade (99° to 100°F, oral).

Laboratory analysis reveals hemoconcentration and leukocytosis with a left shift. Amylase and phosphorus levels are elevated only when ischemia and necrosis are advanced. Metabolic acidosis is common with elevation of lactic acid and a concomitant increased anion gap. Prothrombin time and partial thromboplastin time are normal.

Routine abdominal radiographs are of little diagnostic help; nonspecific ileus, ascites, bowel wall thickening, mucosal irregularity, and thumb printing have all been observed in patients with MVT. Ultrasonography has been used for early diagnosis in nonobese patients.6 If available, mesenteric arteriography should be done early in the diagnostic evaluation. Patency of the arteries and opacification of the bowel wall with failure to visualize the mesenteric veins are diagnostic of MVT. Contrastenhanced computed tomography (CT) showing a dense venous wall surrounding a central lucency was diagnostic in a report involving six cases of MVT.7 The use of xenon 133 in normal saline injected into the peritoneal cavity has proved to be a promising method of early diagnosis because the ischemic bowel retains it.8 Time is important in reducing mortality in MVT; therefore, clinical and surgical evaluation should take priority over radiologic procedures.

A laparotomy with identification of infarcted bowel and mesentery, especially with bloody ascites, is diagnostic of ischemia of the bowel. Exploration reveals thrombosis in the venous system. A thrombectomy is mandatory along with resection of all necrotic small bowel and mesentery. Intraoperative treatment with heparin should be started and continued for 7 to 10 days. Warfarin therapy should be started postoperatively and continued for at least 3 months and possibly indefinitely, depending on the underlying cause. Oral contraceptives or exogenous estrogens should be discontinued. Massive volume support and broad-spectrum antibiotics are also needed postoperatively, as sepsis is a common sequela. A "second look" operation performed 24 to 48 hours after the first one is recommended because of a 60% recurrence rate of thrombosis and ischemia.4

The mortality rate is 100% without treatment and adequate resection; even with surgery the mortality rate is 30% to 45%. Studies suggest that if long-term anticoagulants are not used, 25% of patients will have another episode of MVT.

Mesenteric arterial occlusion is much more common than MVT, and the clinical presentation is usually identical, as both represent vascular insufficiency to the small bowel. Arterial occlusion is often associated with postprandial pain and a history of valvular heart disease, chronic atrial fibrillation, or extensive arteriosclerosis. Arteriography will show vascular occlusion of the mesenteric arteries. Treatment is embolectomy and resection of infarcted bowel.

In patients without an obvious cause for MVT, an evaluation of intrinsic anticoagulant disorders is warranted. The importance of defining an intrinsic anticoagulant deficiency state is not only for determining the length of oral anticoagulation treatment, but because these deficiencies are inherited in an autosomal dominant pattern. The risk of venous thrombosis (including pulmonary) from these deficiencies increases with age.

Antithrombin III (AT-III) deficiency is the most commonly inherited anticoagulant deficiency, and MVT is a relatively common presentation of AT-III deficiency.⁹ Antithrombin III exerts 50% of the serum's intrinsic anticoagulant activity by inhibiting thrombin and factor Xa. Assays are of no value after a recent thrombotic event secondary to consumption. Heparin binding also accelerates the rate of AT-III inactivation of thrombin, and thus assays must be conducted 1 to 2 weeks after heparin therapy, along with assays for protein C and S levels. Deficiency of the vitamin K–dependent proteins C and S has also been implicated as a primary cause of MVT.^{10,11} Protein C inhibits factors Va and VIIIa. Protein S is a cofactor for protein C by promoting its binding to lipid and platelet surfaces. Heterozygotes for proteins C or S have a 50% or more deficiency of these natural anticoagulants. The risk of thromboembolism increases with age, and 50% of patients with MVT will have recurrent thromboembolism if they do not receive treatment. The venous thrombotic events are also precipitated in these patients by pregnancy, trauma, surgery, prolonged immobilization, or oral contraceptive use.¹² Treatment of these deficiencies is lifelong warfarin therapy.

Conclusions

Mesenteric venous thrombosis is a relatively rare entity. A high index of suspicion is required for diagnosis when a person complains of generalized severe abdominal pain that is out of proportion to physical findings. Mesenteric arteriography and contrast-enhanced CT can be used for diagnosis, but clinical findings should override timeconsuming tests or negative interpretation. A laparotomy diagnostic of MVT should be followed by thrombectomy, wide resection of necrotic bowel, anticoagulation, broad-spectrum antibiotics, and a 24- to 48-hour repeat laparotomy for thrombotic recurrence. In the absence of mechanical or infectious causes for MVT, assays for AT-III, protein C, and protein S should be done as the findings have relevance for the health of the patient's offspring.

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