

Postlaparoscopic Small Bowel Obstruction Secondary to Unrecognized Nodular Endometriosis of the Terminal Ileum

W. Paul Dmowski, M.D., Ph.D., Nasir Rana, M.D., M.P.H., and Nercy Jafari, M.D.

Abstract

Small bowel involvement by endometriosis occurs in about 0.5% of patients, but nodular endometriosis involving the entire wall of the terminal ileum is extremely rare. Endometriotic nodules protruding into the intestinal lumen may lead to chronic, partial, or acute complete small bowel obstruction and associated clinical changes. If obstruction is partial, preoperative diagnosis is difficult and seldom suspected, and no reliable diagnostic tests are available. At laparoscopic surgery, performed typically for associated pelvic endometriosis, bowel lesions may easily be overlooked, especially in women with abdominal adhesions from earlier surgery. Surgical injury, tension tears, or postoperative edema may contribute in such cases to the development of acute, complete small bowel obstruction, which may be difficult to differentiate from postoperative ileus. The patient may deteriorate rapidly and develop abdominal sepsis and multiple organ failure with high risk of mortality. Because of increased production of tumor necrosis factor- α by autologous monocytes, endometriosis may predispose to development of severe sepsis and septic shock.

(*J Am Assoc Gynecol Laparosc* 8(1):161-166, 2001)

Acute postoperative small bowel obstruction (SBO) is an infrequent event that is a serious diagnostic and therapeutic problem. It is difficult to differentiate from paralytic ileus on clinical grounds alone. Furthermore, both conditions may represent different points along a single disease continuum, and elements of both may frequently occur in the same patient. Yet, treatment of these entities is quite different, and immediate surgical intervention may be life saving when obstruction with strangulation is present.

The most common causes of SBO are adhesions, neoplasms, hernias, both external and internal, inflammatory bowel disease, intraabdominal abscess or hematoma, and volvulus.^{1,2} A multicenter study reported 24 cases of intestinal obstruction after laparoscopic abdominal surgery in 10,327 patients, with an overall SBO prevalence of 0.21%.³ This was only

slightly lower than 0.69% prevalence of bowel obstruction after open abdominal procedures in 8098 patients.³ The most common causes of obstruction in these series were adhesions or fibrotic bands and intestinal incarceration. Most obstructions occurred at umbilical cannula sites even when fascia was closed. Individual cases of SBO also occurred after pelvic diagnostic and operative laparoscopies.⁴⁻⁸ These occurred 2 days to several months after surgery, and were caused by external or internal hernias and incarceration of small bowel in laparoscopic cannula sites or in intraabdominal adhesions.

Acute SBO by extensive endometriosis is exceedingly rare, and only individual reports are on record. According to reviews, the first report appeared in 1912 in Dutch medical literature. Subsequently, only 3 more cases were reported in Dutch, 5 in French, 1 in

From the Institute for the Study and Treatment of Endometriosis, Oak Brook, Illinois (all authors).

Address reprint requests to W. Paul Dmowski, M.D., Institute for the Study and Treatment of Endometriosis, 2425 West 22nd Street, Oak Brook, IL 60523; fax 630 954 0064.

Accepted for publication September 13, 2000.

Spanish, and 31 in English literature.^{9,10} Our MEDLINE search identified 11 other cases of acute SBO caused by endometriosis.¹⁰⁻¹⁷ In one study, endometriosis was identified as the cause of obstruction in only 1 of 314 operations for SBO during 3 years.¹ In most patients, obstruction was the result of external constriction by adhesions, with annular strictures being common, or was caused by extrinsic pressure deformity secondary to external endometriomas. Endometriotic implants in such cases involved only bowel serosa. Occasionally, intestinal intussusception or volvulus was caused by endometriotic tissue. Very infrequently, nodular endometriotic lesions involving the entire thickness of the intestinal wall and protruding into the lumen caused SBO.

Case Report

A 28-year-old nulligravid woman was first seen because of symptomatic pelvic endometriosis. She had a history of progressive chronic pelvic pain since the late teens. At age 17 years, because of severe dysmenorrhea, she was prescribed oral contraceptives, which she continued until age 24, when she stopped in order to conceive. Dysmenorrhea, both lower quadrant abdominal pain, deep dyspareunia, occasional dysuria, dyschezia, and hematochezia worsened progressively. She was seen on several occasions in the emergency room and was told she had ovarian cysts. Because of pressure on the bowel, constipation, flatus, and dyschezia, a lower gastrointestinal workup, including colonoscopy, was performed but was apparently negative.

At age 26, because of bilateral ovarian enlargement by echogenic cysts (right 70 × 65 × 76 mm, left 80 × 58 × 80 mm), she underwent laparoscopy and laparotomy at which endometriosis was diagnosed. According to the surgical report, extensive pelvic adhesions and bilateral ovarian chocolate cysts were identified and resected. There were multiple endometriotic implants in the pelvis and endometriotic plaques involving pelvic sidewalls as well as serosa of the rectosigmoid. Some of these were electrocoagulated. According to the pathology report, endometrial glands and stroma were identified microscopically in the resected portion of each ovary. The woman experienced some symptomatic improvement and was prescribed ovarian stimulation with clomiphene and then human menopausal gonadotropin-human chorionic gonadotropin during three consecutive

cycles. She did not conceive and her symptoms worsened.

Pelvic examination in our office showed bilateral adnexal enlargement and fixation, and tender cul-de-sac nodules. On pelvic ultrasound the right and left ovaries measured 28.5 × 51.4 and 28.3 × 33.1 mm, respectively, and contained several echogenic, most likely endometriotic cysts ranging between 1 and 3 cm in diameter. The patient was prescribed danazol 200 mg 4 times/day, which she continued for 6 months. During that time she had almost complete relief of symptoms and no side effects except for 6-pound weight gain. On pelvic ultrasound both ovaries decreased in size, the right to 16.9 × 34 mm, and left to 19.8 × 21.5 mm. Both contained several small echogenic cysts.

First Laparoscopy

After 6 months of danazol therapy the woman underwent open laparoscopic resection of ovarian endometriosis and pelvic adhesiolysis. Extensive postoperative omental adhesions to the earlier laparotomy scar were identified and divided. The posterior cul-de-sac was completely obliterated by loops of large and small bowel, densely adherent to the ovaries, fallopian tubes, and uterine fundus. There were no endometriotic lesions, but multiple hemosiderin deposits were present on peritoneal surfaces. Adhesions of large and small bowel were divided with laparoscopic scissors, constant traction and countertraction, and careful, delicate technique. Both ovaries and fallopian tubes were freed from the attached bowel. Numerous small endometriomas containing thick chocolate-like material were present in each ovary and were resected. Endometriotic implants were ablated with neodymium:yttrium-aluminum-garnet laser. The appendix and loops of small bowel adherent to the right ovary were freed using the same technique and were retracted out of the pelvis. No endometriosis was detected on either large or small bowel. No attempt was made to free small bowel adhesions located outside of the pelvis, since that may carry additional morbidity. At the end of the procedure, which lasted 2.5 hours, the pelvic anatomy was restored to normal. There were no pelvic adhesions or residual endometriosis, hemostasis was complete, and both fallopian tubes were patent.

Microscopic examination of ovarian and peritoneal specimens showed multiple endometrial glands and stroma.

Postoperative Course

After surgery, the patient ambulated and resumed a regular diet. Seventy-two hours later she complained of generalized abdominal pain, nausea, vomiting, and frequent small loose stools. She had abdominal distention, pelvic and abdominal tenderness, and decreased bowel sounds, but no rebound. Plain radiographs of the abdomen showed distention of the stomach and loops of small bowel with multiple air-fluid levels. The patient had a low-grade fever and pulse rate of 120/minute, but otherwise vital signs were normal. She had no leukocytosis and hemoglobin was normal.

After consultation with a general surgeon, the patient was admitted to the hospital for diagnostic evaluation and nasogastric suction. Computerized tomographic (CT) scan with oral and intravenous contrast was suggestive of SBO, but no specific lesions or transitional zone could be identified. About 14 hours later, CT scan with additional oral contrast showed increased distention of proximal and midsmall bowel, with poor contrast progression through bowel and no distention of the colon. A small amount of extraluminal fluid in the right flank suggested intestinal perforation in addition to SBO. Four days after laparoscopy, because of increasing symptoms and progressive deterioration, the patient underwent open laparoscopy with the general surgeon in attendance.

Second Laparoscopy

Massively distended loops of small bowel were seen through the laparoscope, but the site of SBO could not be identified. At laparotomy through a midline incision, a distended small bowel and obstruction at the terminal ileum were identified. A loop of terminal ileum about 20 cm long was tightly coiled, thickened, extensively nodular, and densely adherent to the right side of the abdominal wall outside of the pelvis. Distally, the remaining ileum and colon were collapsed. Just proximal to the site of obstruction in the distended and adherent wall of bowel, a small area of perforation was identified. The distorted loop of bowel and the perforation were freed from adhesions and resected, and ileo-ileal anastomosis was performed. Appendectomy was also performed.

On gross pathologic examination the resected loop of bowel was covered with dense fibrous adhesions and shaggy fibrinous exudate. Many fibrotic nodules extended from the mesenteric surface and protruded into the lumen. At one point the nodules were almost

confluent, totally obstructing the lumen (Figure 1). The bowel mucosa was distorted by the nodules and had focal areas of ulceration. The bowel wall was thickened up to 1.5 cm in diameter. Microscopically, endometrial glands and stroma were noted within the nodules (Figure 2), as well as considerable hypertrophied muscle, together with chronic and superimposed acute peritonitis. No histologic evidence of thermal injury was present at the site of perforation. There was also endometriosis of the appendix.

Postoperatively, the patient developed intraperitoneal sepsis, hypotension, adult respiratory distress syndrome, anemia, cardiac arrhythmia, coagulopathy,



FIGURE 1. Cross section through the diseased portion of small bowel. Note endometriotic nodule (heavy arrow) extending from mesenteric surface to mucosa, with mucosal ulceration and complete obstruction of bowel lumen (thin arrow). (Hematoxylin & eosin; magnification 2.5 \times .)

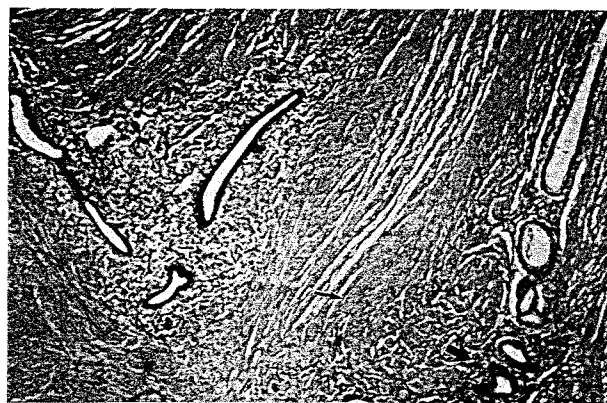


FIGURE 2. Photomicrograph of the endometriotic nodule. Note nests of endometrial glands and stroma (heavy arrow) within hypertrophied smooth muscle (thin arrow). (Hematoxylin & eosin; magnification 25 \times .)

pleural effusion, and pulmonary embolism, all of which gradually responded to intensive therapy. After a stormy postoperative course, she was discharged 24 days after admission. Six months later she was doing well, with no symptoms of bowel obstruction and no evidence of recurrent endometriosis.

Discussion

It is not unusual for endometriotic lesions to involve extrapelvic locations and be present in a variety of tissues and organs. Bowel involvement, because of its proximity to the reproductive system, is quite frequent, as reflected by a recent statement that "ovarian endometriosis appears to be a marker for more extensive pelvic and intestinal disease."¹⁸ In a review of 7177 women with endometriosis, 12.3% had intestinal involvement,¹⁹ and the prevalence of intestinal endometriosis was 25% in 1785 patients with the disease.¹⁸ Typically, intestinal endometriosis is identified in the cecum, appendix, rectosigmoid colon, and small bowel, with the rectosigmoid most frequently affected. Involvement is usually limited to bowel serosa. Only occasionally endometriotic lesions narrow the intestinal lumen, causing symptoms of partial or complete bowel obstruction. Small bowel endometriosis, involving usually the terminal ileum, is much less frequent than endometriosis of the large bowel. In a series of 7200 women with endometriosis, 36 (0.5%) had small bowel involvement and 11 (0.15%) had symptoms of SBO.²⁰ In another report, small bowel involvement was present in 4.1% of patients.¹⁸

Nodular endometriotic lesions extending from bowel serosa to mucosa with mucosal ulceration, and partial or complete SBO as seen in our patient, are even less frequent. We identified only six reported cases with similar gross and microscopic description of lesions as in our patient.^{10,14-17,21} Five of these had acute, complete, and one occult partial SBO.²¹ It is interesting that similar endometriotic lesions in the ileocecal region causing partial or complete intestinal obstruction were reported in 4 of 80 rhesus monkeys in whom effects of oral polychlorinated biphenyls (PCBs) were studied.²² The PCBs and other organochlorides such as dioxin are known immunotoxicants and were implicated as a potential pathogenetic factor in development of endometriosis.²³

It is unclear to what extent our patient's preoperative symptoms were actually a reflection of chronic partial intestinal obstruction. Endometriotic implants

on large bowel serosa were described in the operative report from her earlier laparotomy, but no mention was made of small bowel involvement. Colonoscopy was normal, and no small bowel studies were performed. Furthermore, substantial symptomatic improvement during treatment with danazol did not raise a suspicion of obstructive small bowel lesion, and we had no reason to study the small bowel before surgery. Moreover, such studies are notoriously unreliable. At laparoscopy after danazol therapy, the rectosigmoid was empty of fecal content, and no nodules in the wall of large intestine were suggestive of endometriosis. The extent of intraabdominal adhesive disease precluded thorough examination of small bowel. However, it should be emphasized that nodular endometriosis of the bowel can be missed easily at laparoscopy because tissues cannot be palpated satisfactorily. Only bowel adhesions that were in the pelvis and were considered to interfere with reproductive function were resected, and we found no evidence of bowel endometriosis in those areas.

There is no question that bowel adhesiolysis and manipulation, probably through postoperative edema, contributed to the development of SBO in this woman. At 72 hours after laparoscopic surgery the clinical picture was suggestive but not diagnostic of SBO, which had to be differentiated from postoperative ileus and bowel injury. Abdominal radiographs and CT scans further suggested SBO, but no transitional zone or cause of obstruction was identifiable. Although subject to interobserver variability, CT scans with oral and intravenous contrast media have high sensitivity (up to 94%) and specificity (up to 96%) in differentiating between SBO and postoperative ileus.²⁴ We were not sure what was the cause of postoperative SBO in our patient, and CT scans were not helpful in this regard. Postlaparoscopic ileus is rather uncommon, and bowel injury, intraabdominal hematoma, and incarcerated ventral hernia were included in our differential diagnosis.

Open laparoscopy with a blunt Hasson cannula is safe and accepted in selected cases of SBO.²⁵ It was performed in our patient with the expectation that a cause of SBO could be corrected laparoscopically. A variety of lesions causing SBO such as intraabdominal adhesions, fibrotic bands, intestinal incarceration, and ventral hernias can be corrected laparoscopically. However, in our patient, laparoscopy was ineffective in identifying and correcting the obstruction because of massive small bowel dilatation and extensive

adhesions. At laparotomy, massively distorted terminal ileum adhered to the abdominal sidewall was identified as the site of obstruction. Just proximal to the obstruction, a distended loop of ileum coiled and adherent to obstructed bowel contained a small area of perforation. This was suggested by the second CT scan. The entire loop of bowel was outside of the pelvis and most likely represented spontaneous perforation proximal to the obstruction. It is also possible that the perforation occurred in the area weakened during laparoscopic adhesiolysis, or in a tension tear that, because of obstruction, became overdistended by bowel content, or that a "microperforation" became expanded by distention due to obstruction. These possibilities, however, seem unlikely.

Laparoscopy was performed by open technique, and neither Veress needle nor sharp cannula was used. Small bowel adhesiolysis was performed with laparoscopic scissors using careful, delicate technique. The site of obstruction and perforation were well outside of the pelvis, buried in adhesions to the lateral abdominal wall, where laparoscopic surgery was not carried out. Occasionally, electrical sparking can cause thermal injury of bowel, which is followed by perforation. This was not the case in our patient. We did not use unipolar coagulation, and histologic examination showed no evidence of thermal injury.

The woman's postoperative course was complicated by abdominal sepsis, which carries a mortality rate in excess of 30%.²⁶ Although the pathophysiology of sepsis-induced multiple organ failure is complex and not entirely clear, tumor necrosis factor (TNF)- α has emerged as a central cytokine of septic shock.²⁶ Main sources of TNF- α are monocytes and macrophages, and production of this cytokine is characterized by high individual variability. It was reported that TNF- α levels were increased due to polymorphism in the TNF- α gene in patients susceptible to sepsis who had fatal outcome.²⁷ The levels were not measured in our patient, but our previous studies showed that in endometriosis, TNF- α production by peripheral monocytes and peritoneal macrophages is significantly increased.^{28,29}

Conclusion

This report should remind laparoscopic surgeons that small bowel endometriosis, although rare, may accompany ovarian endometriosis and, especially, extensive pelvic disease. Nodular endometriotic

lesions of bowel may be only partially obstructive and without symptoms, and are therefore difficult to diagnose preoperatively. If bowel involvement is suspected, laparotomy and bowel resection should be considered, and the patient and staff appropriately prepared. At the time of laparoscopic surgery, all efforts should be made to examine the abdominal cavity carefully and systematically, and to explore and resect potentially obstructive bowel lesions and adhesions. This may require conversion to laparotomy and involvement of a general surgeon. Unresected, partially obstructive small bowel lesions may lead postoperatively to complete SBO, which can be difficult to differentiate from postoperative ileus. Further delay of surgical resection may lead to intestinal perforation, abdominal sepsis, septic shock and multiple organ failure. Prompt diagnosis and resection of diseased bowel, combined with aggressive therapy and constant vigilance, are necessary to prevent a catastrophic outcome.

References

1. Mucha P: Small intestinal obstruction. *Surg Clin North Am* 67:597-620, 1987
2. Wilson MS, Ellis H, Menzies D, et al: A review of the management of small bowel obstruction. *Ann R Coll Surg Engl* 81:320-328, 1999
3. Duron JJ, Hay JM, Msika S, et al: Prevalence and mechanisms of small intestinal obstruction following laparoscopic abdominal surgery. *Arch Surg* 135:208-212, 2000
4. Sauer M, Jarret JC: Small bowel obstruction following diagnostic laparoscopy. *Fertil Steril* 42:653-654, 1984
5. Bourke JB: Small intestinal obstruction from a Richter's hernia at the site of insertion of a laparoscope. *Br Med J* 2:1393-1394, 1977
6. Burney TL, Jacobs SC, Naslund MJ: Small bowel obstruction following laparoscopic lymphadenectomy. *J Urol* 150:1515-1517, 1993
7. Kurtz BR, Daniell JF, Spaw AT: Incarcerated incisional hernia after laparoscopy: A case report. *J Reprod Med* 38:643-644, 1993
8. Saidi MH, Sarosdy MF, Hollimon PW, et al: Intestinal obstruction and bilateral ureteral injuries after laparoscopic oophorectomy in a patient with severe endometriosis. *J Am Assoc Gynecol Laparosc* 2:355-358, 1995

9. Wickramasekera D, Hay DJ, Fayz M: Acute small bowel obstruction due to ileal endometriosis: A case report and literature review. *J R Coll Surg Edinb* 44:59-60, 1999
10. Martin-Duce A, Diez M, Muguerza JM, et al: Acute small bowel obstruction caused by endometriosis. *Eur J Surg* 162:747-749, 1996
11. Perry EP, Peel ALG: The treatment of obstructing intestinal endometriosis. *J R Soc Med* 81:172-173, 1988
12. Hall L-LH, Malone JM, Ginsburg KA: Flare-up of endometriosis induced by gonadotropin-releasing hormone agonist leading to bowel obstruction. *Fertil Steril* 64:1204-1206, 1995
13. Urbach DR, Reedijk M, Richard CS, et al: Bowel resection for intestinal endometriosis. *Dis Colon Rectum* 41:1158-1164, 1998
14. Payne SP, Pace RF, Benjamin IS: Isolated endometriosis of the small bowel presenting as acute small bowel obstruction. *Br J Clin Pract* 45:57-58, 1991
15. Hunt TM, Kelly MJ: Endometriosis—The problems of intestinal obstruction. *Br J Clin Pract* 47:159-160, 1993
16. Gomez-Rubio M, Fernandez R, de Cuenca B, et al: Intestinal endometriosis as a cause of chronic abdominal pain leading to intestinal obstruction. *Am J Gastroenterol* 92:525-526, 1997
17. Agha FP, Elta G, Abrams GD: Ileal endometriosis causing acute small-bowel obstruction. *Mt Sinai J Med* 53:497-500, 1986
18. Redwine DB: Ovarian endometriosis: A marker for more extensive pelvic and intestinal disease. *Fertil Steril* 72:310-315, 1999
19. McAfee CH, Greer HL: Intestinal endometriosis: A report of 29 cases and a review of the literature. *J Obstet Gynaecol (Br)* 67:539-555, 1960
20. Martinbeau PW, Pratt JH, Gaffey TA: Small bowel obstruction secondary to endometriosis. *Mayo Clin Proc* 50:239-243, 1975
21. Gindoff PR, Jewelewicz R: Ileal resection in the operative treatment of endometriosis. *Obstet Gynecol* 69:511-513, 1987
22. Campbell JS, Wong J, Tryphonas L, et al: Is simian endometriosis an effect of immunotoxicity? [abstr]. Presented at the 48th annual meeting of the Ontario Association of Pathologists, London, Ontario, Canada, October 3-5, 1985
23. Rier SE, Martin DC, Bowman RE, et al: Endometriosis in rhesus monkeys (*Macaca mulatta*) following chronic exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Fundam Appl Toxicol* 21:433-441, 1993
24. Ha HK, Shin BS, Lee SI, et al: Usefulness of CT in patients with intestinal obstruction who have undergone abdominal surgery for malignancy. *AJR* 171:1587-1593, 1998
25. Strickland P, Lourie DJ, Suddleson EA, et al: Is laparoscopy safe and effective for treatment of acute small-bowel obstruction? *Surg Endosc* 13:695-698, 1999
26. Wheeler AP, Bernard GR: Treating patients with severe sepsis. *N Engl J Med* 340:207-214, 1999
27. Mira J-P, Cariou A, Grall F, et al: Association of TNF2, a TNF- α promoter polymorphism, with septic shock susceptibility and mortality. A multicenter study. *JAMA* 282:561-569, 1999
28. Braun DP, Gebel H, House R, et al: Spontaneous and induced synthesis of cytokines by peripheral blood monocytes in patients with endometriosis. *Fertil Steril* 65:1125-1129, 1996
29. Rana N, Braun DP, House R, et al: Basal and stimulated secretion of cytokines by peritoneal macrophages in women with endometriosis. *Fertil Steril* 65:923-930, 1996