

Endometriosis with Massive Bloody Ascites

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Endometriosis is commonly found in 10 to 15 percent of women aged between 25 and 44 years,¹ and it has been estimated that 25 to 40 percent of infertile women have endometriosis.¹ In this report, we describe a patient with endometriosis whose signs were massive bloody ascites. The purpose of this report is to increase awareness among physicians of the varied clinical manifestations of intestinal endometriosis.

Case Report

A 44-year-old black woman (gravida 1, para 0, abortus 1) began to notice a gradual increase in her abdominal girth 1 year before her admission. She also was aware of fatigue, nausea, and dizziness. She denied melena, hematemesis, or change in bowel habits. Her medical history was notable for endometriosis for which she was taking medroxyprogesterone acetate. The diagnosis of endometriosis was made by laparoscopy 10 years earlier, when the patient was examined for dysmenorrhea and menorrhagia that had continued since menarche. She had no history of any blood coagulation disorders or platelet dysfunction.

On admission she weighed 145 pounds; her blood pressure was 128/88 mmHg, pulse 114 beats per minute, respirations 22/min, and temperature 98.2°F. Positive findings included a distended, tense abdomen without fluid waves. The fundus of the uterus was not palpable, nor were the adnexa. No masses were felt on a rectal examination, and her stool was guaiac negative.

A vaginal sonogram showed an ill-defined, right adnexal complex mass (65 × 66 mm) with deviation of the uterus to the left and a large amount of ascites. A paracentesis yielded bloody ascites. Her erythrocyte count was 112,500/ μ L, leukocytes 300/ μ L (62 percent polymorpho-

nuclear cells, 7 percent lymphocytes, 3 percent eosinophils, 7 percent monocytes, and 22 percent histocytes), amylase 139 U/L, glucose 39 mg/dL, and lactate dehydrogenase 1,029 U/L. Cultures of the ascites were negative, and there were normal findings on cytologic examination.

Serum tumor markers and other laboratory tests were all within normal limits, including CA-125 antigen 7 U/mL (normal, 0 to 34 U/mL), carcinoembryonic antigen 2.9 ng/mL (normal, less than 3 ng/mL), alpha-fetoprotein less than 2 ng/mL (normal, less than 2 ng/mL), hematocrit 39 percent, peripheral leukocyte count 7,300/ μ L, peripheral platelet count 384,000/ μ L, serum sodium 142 mEq/L, potassium 4.7 mEq/L, chloride 101 mEq/L, bicarbonate 26 mEq/L, blood urea nitrogen 11 mg/dL, creatinine 0.6 mg/dL, serum albumin 3.5 g/dL, total bilirubin 0.3 mg/dL, alkaline phosphatase 86 U/L, aspartate aminotransferase (AST) 20 U/L, and alanine aminotransferase (ALT) 17 U/L.

Findings on an electrocardiogram and chest radiographs were also unremarkable. Computed tomography of the abdomen and pelvis confirmed massive ascites and a right adnexal mass, which suggested an ovarian neoplasm as the source of the bloody ascites. A gynecologist and surgeon were consulted, who recommended exploratory laparotomy.

The implications of these findings were discussed with the patient. Her presumptive diagnosis was ovarian tumor. She consented to undergo the surgical procedure, resection of the ovarian tumor (if possible), and operative drainage of the ascites.

Severe intra-abdominal and pelvic fibrosis and adhesions were seen during the laparotomy. The uterus, fallopian tubes, and ovaries were grossly normal. The abdominal organs were encased in dense fibrous tissue, and individual organs were difficult to discern. The fibrotic process involved the liver, spleen, and entire gastrointestinal tract; no attempt was made to lyse the adhesions. Multiple biopsies of the thickened, fibrotic peritoneum were obtained and sent for frozen section

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preparation. Microscopic examination of these frozen sections done intraoperatively showed chronic fibrosis without any evidence of malignant processes.

In view of the patient's history of endometriosis, the surgeon's impression was that the dense adhesions and bloody ascites were complications of long-standing endometriosis and that a total abdominal hysterectomy and bilateral salpingo-oophorectomy were indicated for failure of medical management. The operation also included drainage of approximately 10 L of bloody ascitic fluid. Final histopathologic examination of the biopsies of the thickened peritoneum confirmed fibrosis, endometriosis, adenomyosis, and pseudoxanthomatous nodules, whereas normal tissues were found on histologic sections of the uterus, fallopian tubes, and ovaries. The patient's postoperative recovery was uneventful. She had follow-up examinations monthly during the first 3 months postoperatively and then every 3 months thereafter. She was well, asymptomatic, and free of ascites at a 15-month follow-up visit.

Discussion

An estimated 10 to 15 percent of women of child-bearing age have endometriosis, and in a third of patients with endometriosis, the intestine can be involved.^{1,2} Endometriosis commonly affects segments of the bowel that lie in the pelvis in proximity to the reproductive organs (such as the sigmoid colon and rectum).¹⁻³ The small intestine, cecum, and appendix are rarely affected.³ Endometriosis is generally asymptomatic and when symptoms do occur, they are usually due to intestinal obstruction,^{3,4} which usually results from marked angulation or kinking of the bowel.⁵ Less frequently, obstruction results from luminal narrowing, volvulus, intussusception, or intramural hemorrhage.^{3,5} The symptoms are often related to the onset of menses.⁶

Diagnosing endometriosis requires that the physician distinguish it from neoplastic or other inflammatory processes.³ In general, asymptomatic lesions found incidentally at laparotomy for another condition require no treatment, whereas symptomatic disease should be treated by local resection of the involved intestine coupled with concurrent medical treatment for the underlying endometriosis.³

Hemorrhagic ascites is characterized by bloody

appearance of abdominal fluid and is defined as ascitic fluid with an erythrocyte count of more than 50,000/ μ L.⁷ The most common causes of ascites include hepatoma, metastatic ovarian carcinoma, and tuberculous peritonitis.⁷ Other less frequent causes include mesothelioma, Budd-Chiari syndrome, perforated duodenal ulcer, primary splenic lymphoma, mesenteric cyst, multiple myeloma, and cirrhosis.⁷

The association between endometriosis and large-volume bloody ascites was first described in 1954 by Brews.⁸ Since then only 28 cases (including ours) have been reported in the English language literature.⁷⁻²⁸ These patients were generally young (average age 30 years). Eighteen of the 24 patients for whom parity was reported were nulliparous, and 14 of 21 patients for whom race was reported were black.⁷⁻²⁸ The most common presentation was increasing abdominal girth, often accompanied by abdominal pain, nausea, diarrhea, or dysmenorrhea.²⁸ Massive amounts of ascitic fluid, varying from 150 to 10,000 mL (average: 3500 mL), were present in all cases⁷⁻²⁸ (and in our case). The ascitic fluid was characteristically dark-brown or bloody, and malignant cells were not found on a cytologic examination. Furthermore, 7 patients had associated pleural effusion.²⁸ In all 28 reported cases⁷⁻²⁸ (and in our case), the diagnosis of bloody ascites associated with endometriosis was documented at laparotomy and by excluding other known causes of bloody ascites.

The pathogenesis of bloody ascites secondary to endometriosis has not been elucidated. Bernstein et al¹² proposed a mechanism involving rupture of endometriotic cysts, releasing into the peritoneal cavity both blood and endometrial cells that could then act as irritants on peritoneal surfaces, leading to formation of ascites and dense adhesions.¹⁷

The diagnosis of ascites secondary to endometriosis is usually unsuspected preoperatively.²⁰ Other known causes of bloody ascites, especially a malignant process, must be excluded. In addition to endometriosis involving various gastrointestinal organs, other findings such as adhesions and fibrosis are frequently encountered at laparotomy. The diagnosis must be confirmed histologically.¹⁷ Definitive management consists of surgical resection of the endometriotic tissue, coupled with surgical castration (ie, hysterectomy

and bilateral salpingo-oophorectomy) or long-term hormonal suppression therapy (eg, progestin, danazol, or gonadotropin-releasing hormone agonists).^{7,28} Long-term hormonal suppression is generally reserved for young nulliparous patients because it obviates the need for surgical castration and can preserve fertility in some cases.

In conclusion, endometriosis should be considered in the differential diagnosis when bloody ascites and a pelvic mass are encountered, particularly in young female patients, for which there is no obvious explanation. Our case highlights the protean manifestations and gastrointestinal involvement of a common gynecologic problem frequently encountered by primary care physicians, the difficulty in obtaining an accurate preoperative diagnosis, and the need for histologic diagnosis before initiating treatment. Our case report also illustrates the systematic approach to diagnostic evaluation and therapeutic options for intestinal endometriosis.

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