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Case Report

Intestinal obstruction by deep enteric endometriosis: case report and literature review

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ABSTRACT

Deep endometriosis (DE) is an uncommon cause of bowel obstruction; preoperative diagnosis is a challenging task due to its rarity and pathological confirmation. Surgery is the appropriate treatment and complications are common. A 26-year-old Latin female was admitted to emergency department with 72 hours history of abdominal pain associated with inability to pass stool or gas, vomiting and nausea. Abdominal distention and pain without acute abdomen signs. Laboratory tests reported normal. Abdominal contrast-enhanced computed tomography showed distal small bowel obstruction. Patient underwent exploratory laparotomy with segmental resection bearing ileal strictures and Brook´s ileostomy was performed. Postoperative course of patient was uneventful and after pathology report treatment with dienogest was established. DE remains challenging entity to treat, medical treatment can reduce symptoms, but surgical resection is required. Bowel resection is reserved for mayor stenosis lesions. Anastomotic leakage is frequent. Surgery represents the definitive treatment for bowel obstruction by DE. Resection improves pain and intestinal symptoms. Recurrence, stenosis, and anastomotic leakage rates vary across the studies. Surgical and medical treatment should be considered.

Keywords: Bowel endometriosis, Bowel obstruction, Segmental resection

INTRODUCTION

Intestinal obstruction is a frequent cause of emergency surgery, representing around 50% of urgent laparotomies worldwide, with significant morbidity and hospital costs.[1] The main cause of intestinal obstruction is intestinal adhesions in 75% of cases second to abdominal surgical procedures, however, in those patients in whom there are no previous abdominal surgical events, they are referred to as small bowel obstruction in a virgin abdomen (SBO-VA).[2] The most common causes of SBO-VA are inflammatory bowel disease, neoplasms, bezoars, and, on rare occasions, deep endometriosis.[3,4]

Endometriosis is a chronic inflammatory disease in which the endometrial tissue is found outside the uterine cavity, mainly in the pelvic cavity including the peritoneum, ovaries, ligaments, bowel, and bladder. Endometriosis has a prevalence in women of childbearing age of 10-15%.[5] It comes in three main variants: superficial endometriosis (SE), endometriotic nodules (EN) and deep endometriosis (DE); occasionally accompanied by scarring fibrosis and adhesions.[6] The most frequent presentation of deep intestinal endometriosis is found in the sigmoid colon and rectum, sometimes leading to intestinal obstruction requiring resection and stoma creation surgery.[7]

CASE REPORT

A 26-year-old Latin female patient admitted to the emergency department due to abdominal pain of 72 hours of colic-like evolution associated with inability to channel gas and evacuate, accompanied by nausea and vomiting of stomach content. Managed by primary care with antispasmodic, prokinetic and proton pump inhibitor without improvement. Normal family history. She denies allergies, chronic drug use and transfusions.

She refers to a history of laparoscopic appendectomy two years prior. Menarche at 10 years of age, dysmenorrhea, nulliparous, sexually active with two years of use of intrauterine device with prolonged release of ethinylestradiol. Physical examination revealed abdominal distension, painful on deep generalized palpation with no evidence of peritoneal irritation.

The paraclinical tests reported normal blood counts, renal function, and electrolytes (Table 1). The patient was initially managed with intravenous fluids, broad-spectrum antibiotics, and bowel rest. The abdominal-pelvic tomography with intravenous contrast showed intestinal obstruction in the distal ileum (Figure 1). After authorization, the patient and his relatives signed the informed consent form and the abdominal surgical pathology understanding form, and an urgent exploratory laparotomy was performed under total intravenous anesthesia (TIVA). During surgery, two circumferential stenosing lesions were identified at 30 and 70 millimeters from the ileocecal valve that caused retrograde dilatation of the small intestine up to 45 millimeters and multiple mesenteric adenopathies of 15 to 20 millimeters (Figure 2).

Table 1: Admission laboratories.

Parameters	Range	Parameters	Range
Leukocytes (10^3/mcrl)	6.24	**Glucose (mg/dl)**	78
Neutrophils (10^3/mcrl)	5.08	**BUN (mg/dl)**	8.6
Lynphocytes (10^3/mcrl)	0.71	**Urea (mg/dl)**	18.5
Monocytes (10^3/mcrl)	0.41	**Creatinine (mg/dl)**	0.61
Eosinophils (10^3/mcrl)	0.01	**Sodium (mmol/l)**	136
Basophils (10^3/mcrl)	0.03	**Potassium (mmol/l)**	3.6
Hemoglobin (gr/dl)	15.9	**Chlorine (mmol/l)**	101
Hematocrit (%)	47.3	**INR**	1.09
Platelets (10^3/mcrl)	226	**Trombin activity (%)**	88

INR: International normalized ratio

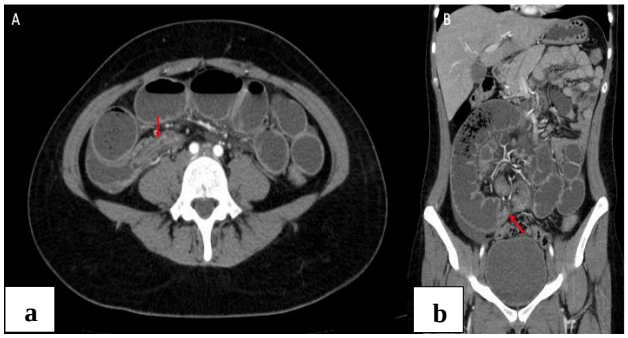

Figure 1: Preoperative abdominal contrast-enhanced computed tomography showing small bowel obstruction (red arrow) with retrograde intestinal dilatation (a) axial reconstruction, and (b) coronal reconstruction.

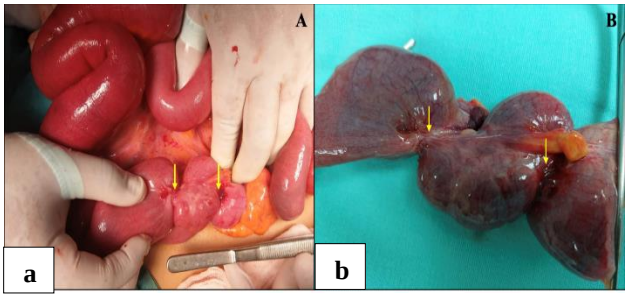

Figure 2: Terminal ileum strictures (yellow arrow) (a) operative findings, and (b) surgical specimen.

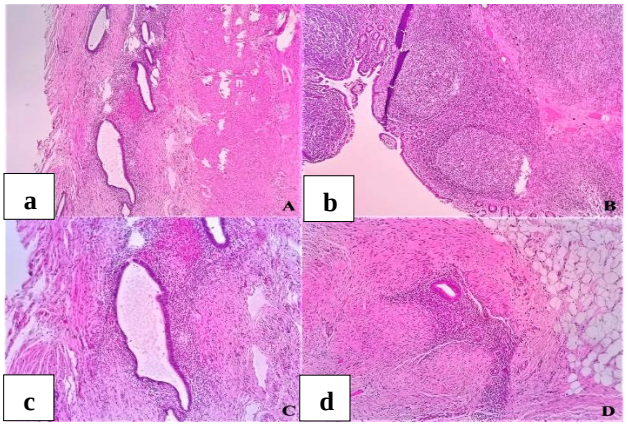

Figure 3: (a) Islands of endometrial glands are observed within the thickness of the muscle layer (HE, 4x); (b) intestinal mucosa with hyperplasia of the lymphoid tissue, with the presence of lymphoid follicles with prominent germinal centers (HE, 4x); (c) endometriosis that affects the muscularis propria of the intestine, showing the characteristic stroma associated with the glandular epithelium (HE, 10x); and (d) endometriosis that affects the muscularis propria of the intestine, showing the characteristic stroma associated with the glandular epithelium, as well as spiral arterioles (HE, 10x).

HE: Hematoxylin-eosin stain

After an exhaustive review, no evidence of intestinal adhesions, areas of ischemia, necrosis or perforation was found. An 8-centimeter segmental resection was performed with macroscopic lesion-free margins with a wide mesenteric segment and an end ileostomy was performed with the Brooke technique. The patient started oral nutrition at 48 hours, was treated with levofloxacin 500 milligrams intravenously every 24 hours for 5 days and enoxaparin sodium 4,000 international units subcutaneously per day for 9 days. She had an uneventful post-surgical course, the Blake-type abdominal drain was removed on the fifth day with post-surgical paraclinical tests without alterations. The patient was discharged on the ninth stable postoperative day for follow-up by outpatient general surgery.

The pathology piece consisted of a terminal ileum measuring 7×2 centimeters in which endometriosis was reported in the serosa and muscular wall with nodular hyperplasia of mucosa-associated lymphoid tissue (Figure 3). An assessment was requested by the gynecology department, who started dienogest 2 milligrams orally once daily, and was asymptomatic with no gastrointestinal side effects.

DISCUSSION

The term virgin abdomen refers to the abdominal cavity in which there is no history of surgery, radiotherapy, or peritonitis.[2] 16% of cases of intestinal obstruction correspond to SBO-VA.[8] SBO-VA occurs more frequently in males in 80% of cases, with a peak incidence between 55-65 years and a surgical management rate of 80%.[9,10] Approximately up to 10% of patients with endometriosis present deep intestinal endometritis and intestinal obstruction occurs in 0.2-3.2%.[11-14]

Deep intestinal endometritis is defined as glands and endometrial stroma infiltrating at least the intestinal muscle layer, clinically it presents as nonspecific intermittent abdominal pain, bloating, dyschezia, hematochezia, defecation urgency, rectal tenesmus, constipation, or diarrhea that are exacerbated in relation to the menstrual cycle.[15,16]

The origin and pathophysiology of endometriosis is not exactly known, the main hypotheses of its origin are retrograde menstruation, coelomic metaplasia, lymphovascular metastasis and neonatal uterine bleeding, however, despite this, other factors are necessary for the development of endometriosis disease such as proliferation, maintenance, immunological alterations, and angiogenesis.[17-19]

Among the genetic factors, mutations in chromosome 7p15.2 and 10q26 are related to familial endometriosis, mutations in the signaling pathways of the Wnt[23], MAPK[24] and STAT3[25] proteins are associated with sporadic endometriosis and alterations in ARID1A[26] and PIK3CA[27] have been related to the association with ovarian cancer.[20-27] The epigenetic alterations studied have focused on methylation of genes expressed in the endometrial secretory phase, the main ones being HOXA10, CDH1 and PGR.[28,29]

The phenomena that develop from endometrial seeding and that modify its microenvironment are responsible for the clinical manifestations of endometriosis. The main hormonal factor involved in the trophism of endometrial cells is estrogen and the regulatory pathways that are modified promote the maintenance and proliferation of endometrial seeding.[30] The increased expression of SF1 favors the local expression of aromatase with the consequent conversion of androgens to estrogens, and the low expression of HSD17B2 decreases the ability to oxidize estradiol into less potent metabolites, thus generating a hyperestrogenic microenvironment in endometrioid lesions. The hyperestrogenic microenvironment favors the upregulation of ERα and ERβ increasing mitogenesis.[31]

In endometrioid lesions, the expression of chemokine ligands for macrophages (CCL2, CCL5, and CCL11) and neutrophils (CXCL1, CXCL5, CXCL8, and CXCL12) promotes activation of B cells, T cells, and platelet dysfunction.[32]

The endometrial cycle results in cyclical changes in the functional polarization of the macrophage population with proinflammatory M1 and proangiogenic M2 immunophenotypes, and the expression of proinflammatory cytokines induces the expression of NF-κB, which induces the production of reactive oxygen species, causing local tissue injury and progressive scarring fibrosis ultimately leading to obstruction of the intestinal lumen.[33-35]

Resection is the definitive treatment for intestinal obstruction due to deep endometriosis.[36,37] On some occasions, the placement of a stent by endoscopy is used as bridging therapy before elective surgery.[38]

Medical treatment of endometriosis is based on two principles, local and systemic hormonal suppression, local inhibition being mostly supported by evidence of local resistance to progesterone in endometrioid lesions.[39]

Dienogest is a fourth-generation progestogen with high specificity for progesterone receptors, strong antiproliferative, antiangiogenic, and anti-inflammatory effects, with a good tolerability profile and a 15-month follow-up. The use of dienogest in the postoperative management of deep endometriosis has been shown in short trials to significantly decrease the rate of recurrence.[40]

CONCLUSION

Intestinal obstruction due to deep endometriosis represents a diagnostic and therapeutic challenge due to its low

incidence, nonspecific imaging and clinical findings in its presentation. Surgical resection is the definitive treatment and the association with hormonal blockade is the cornerstone to improve results and prognosis.

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