

Laparoscopic Management of Endometriosis

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■ INTRODUCTION

Endometriosis is a progressive and often debilitating disease, affecting 10–15% of women during their reproductive years. Among gynecologic disorders, endometriosis is surpassed in frequency only by leiomyomas. Endometriosis is defined as endometrial glands and stroma that occur outside the uterine cavity. The endometriotic lesions are typically located in the pelvis but can occur at multiple sites, including the bowel, diaphragm, and pleural cavity. While endometriosis is a common and nonmalignant process, ectopic endometrial tissue and resultant inflammation can cause dysmenorrhea, dyspareunia, chronic pain, and infertility. Symptoms can range from minimal to severely debilitating. Endometriosis is an estrogen-dependent, benign, inflammatory disease that affects women during their premenarcheal, reproductive, and postmenopausal hormonal stages. Laparoscopy and the surgical laser have allowed definitive treatment following diagnosis. The debate continues as to whether laparotomy or operative laparoscopy is more effective for the treatment of endometriosis.

In women with bowel symptoms such as dyschezia, tenesmus, or cyclic rectal bleeding without any other pathology, a sigmoidoscopic examination should be done at the time of menstruation to rule out bowel involvement by an endometrial implant. However, many women do not demonstrate rectal lesions, but at the time of laparoscopy, significant bowel involvement is seen. It should be remembered that a negative sigmoidoscopy does not rule out bowel involvement in patients of endometriosis. In patients who have significant rectovaginal nodularity on physical examination, preoperative bowel preparation is necessary and antibiotics are administered on the day of surgery. Gynecologists should also consult with a general surgeon experienced in laparoscopic bowel resections. A preoperative ultrasound can assess the ovaries for endometriomas. Preoperative hormonal suppressive therapy can be useful in decreasing the inflammation, bleeding, and possible postoperative adhesion formation.

The goals of surgery are to remove all implants, resect adhesions, relieve pain, reduce the risk of recurrence, and to prevent postoperative adhesion formation. It should also

restore the involved organ's reasonable anatomical and physiological condition. In the case of infertility, restoration of tubo-ovarian relationship is essential to restore fertility.

Hysterectomy with bilateral salpingo-oophorectomy is a definitive treatment of endometriosis. In advanced disease, the ovary may have adhered to the pelvic sidewall. Ovarian dissection may increase the risk of injury to the ureter and vessels in the triangle of doom. The retroperitoneal approach is helpful in these cases and ensures complete removal of ovarian tissue. It also avoids the ovarian remnant syndrome.

Bilateral oophorectomy must be performed to eliminate the estrogen that sustains and stimulate the ectopic endometrium. Following hysterectomy and bilateral oophorectomy, the patient often requires hormone replacement therapy (HRT). Administering the minimal effective dose of estrogen is sometimes associated with a small risk of recurrence. HRT should begin postoperatively. Patients with residual disease may benefit from receiving progesterone from 2 to 6 months, followed by combined progesterone and estrogen for an additional 9 months. Conservative surgery is indicated for women who desire pregnancy and whose disease is responsible for the symptoms of pain and infertility. Surgery improves the likelihood of pregnancy and offers pain relief. Twenty-five percent of patients undergoing conservative operation may require a subsequent operation. The rate of resurgery directly depends on the extent of disease. Those who achieve pregnancy after surgery, only 10% require another operation. Conservative operations are cytoreductive, and recurrence of symptom most likely is caused by the progression of existing pathology that was missed during laparoscopy.

Complete removal of endometriosis is difficult to achieve because there is variability in appearance and visibility. Powder-burn lesions represent foci of inactive disease containing stroma and gland embedded in hemosiderin deposit. These presentations are more common in older lesions and sometimes without any symptoms. When endometriosis involve uterosacral ligament, they are palpable as tender nodule and may cause dysmenorrhea and dyspareunia. Superficial endometriosis is treated optimally by electrosurgical fulguration or ablation. If large

areas of peritoneum are involved with endometriosis or if a woman has recurrent endometriosis in an area previously ablated by electrosurgery or laser, it may be better to excise that entire area of the peritoneum to prevent a recurrence. Especially, areas with scarring or fibrosis should be excised carefully because there may be endometriosis beneath them. One concern in laser ablation or excising large areas of the peritoneum is the chance of adhesion formation. Animal studies indicate that these areas are reperitonealized in 24–48 hours by the migration of surrounding peritoneum and that adhesion formation is low after laparoscopy. However, the surgeon should be cautious, particularly when excising areas of the peritoneum, which are opposed by pelvic organs.

Sometimes atypical lesions are seen as a clear vesicle, pink vascular pattern, white scarred lesion, red lesion, yellow-brown patches, and peritoneal windows, which represent active endometriosis (**Fig. 1A**). These lesions secrete prostaglandin into the peritoneal fluid. The depth of endometrial implants may be related to the level of disease activity and symptoms. The peritoneum must be examined from all the angles and from different degrees of illumination to see all types of lesions. The peritoneal folds must be stretched and search for small and atypical lesions. Normal appearing ovaries sometime may contain endometriosis under the apparently normal cortex. By inserting the needle in the ovary, small endometriosis can be identified by observing the color of aspirated content.

All the pelvic organs are inspected thoroughly. In 15% of cases, the appendix is involved, and so it should be examined. The endometriosis, which has penetrated retroperitoneally several centimeters, is called an iceberg lesion. It can be detected laparoscopically by palpating areas of the pelvis and bowel with the suction irrigation probe. With the forceps or probe, the endometriotic implant is examined, and their size, depth, and proximity to the normal pelvic structure is evaluated.

The diagnostic laparoscopy can be converted into operative one (if required) if the surgeon has preoperative consent. The operative procedure begins by removing adhesions if present between the bowel and pelvic organs to adequately expose the pelvic cavity. The ovaries and tube may be adhered with cul-de-sac or pelvic sidewall. These organs are freed from adhesions and chromotubated. Endometrial implants and endometriomas are resected or vaporized, and if the patient has significant central pelvic pain, uterosacral nerve ablation or presacral nerve resection is performed.

■ LYSIS OF BOWEL ADHESIONS

Tube-ovarian mass with bowel adhesion is a common finding in extensive endometriosis. Bowel adhesions vary in thickness, vascularity, and cohesiveness. Some adhesions are stretched without tearing the tissue and should be excised

with electrosurgery at the points of attachment to the pelvic organs. Dense adhesions are excised either with scissors or by the ultrasonic dissector. The adhered structures requiring separation are pulled apart with forciers, and a cleavage plane is formed. Hydrodissection is useful to identify and develop the dissection plane, which is ablated or excised, using dissecting scissors.

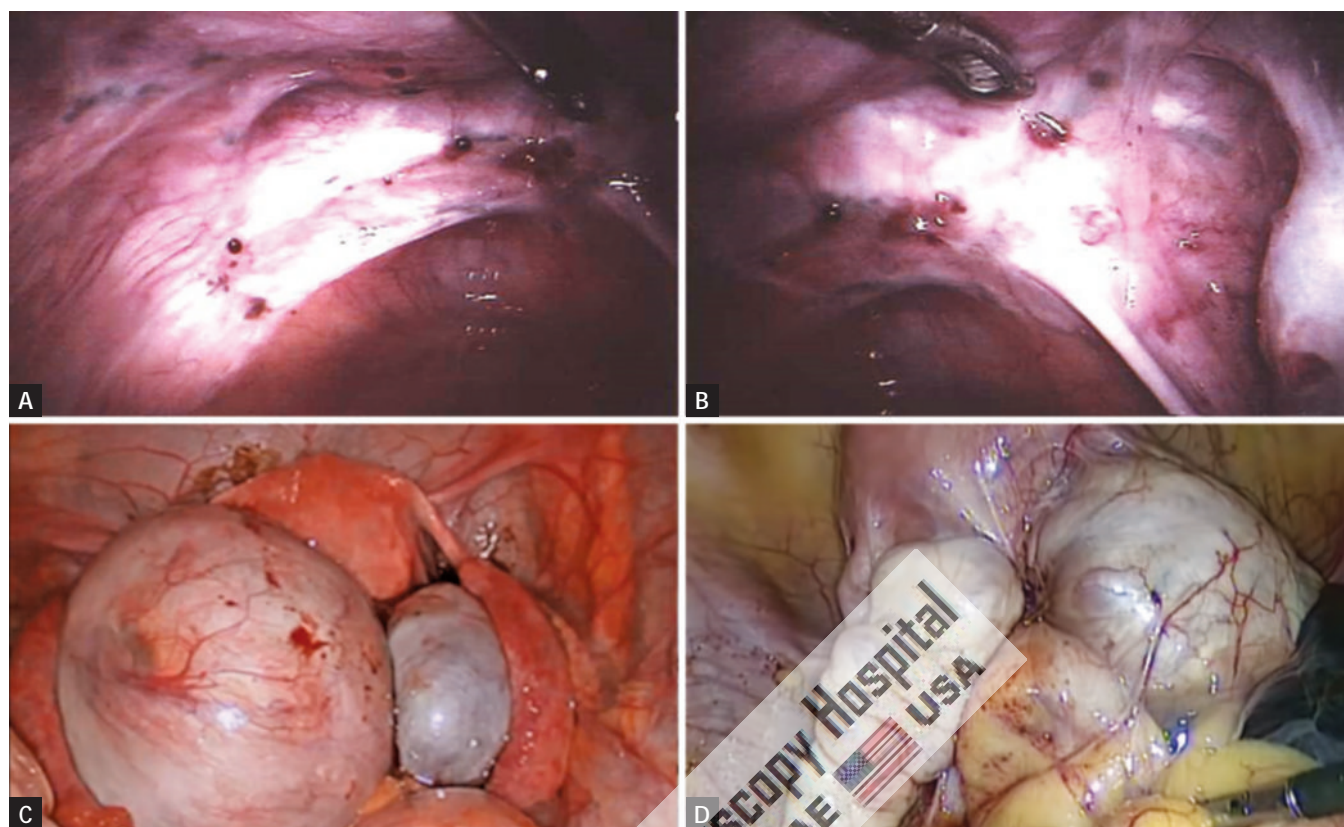
■ PERITONEAL IMPLANTS

At the time of treating peritoneal endometriosis, the implants should be destroyed in the most effective and least traumatic manner to minimize postoperative adhesions and injury to retroperitoneal vessels and nerves. Although different modalities have been used, but hydrodissection and high-power fulguration or CO₂ laser are the best choices for endometriosis treatment (**Fig. 1B**).

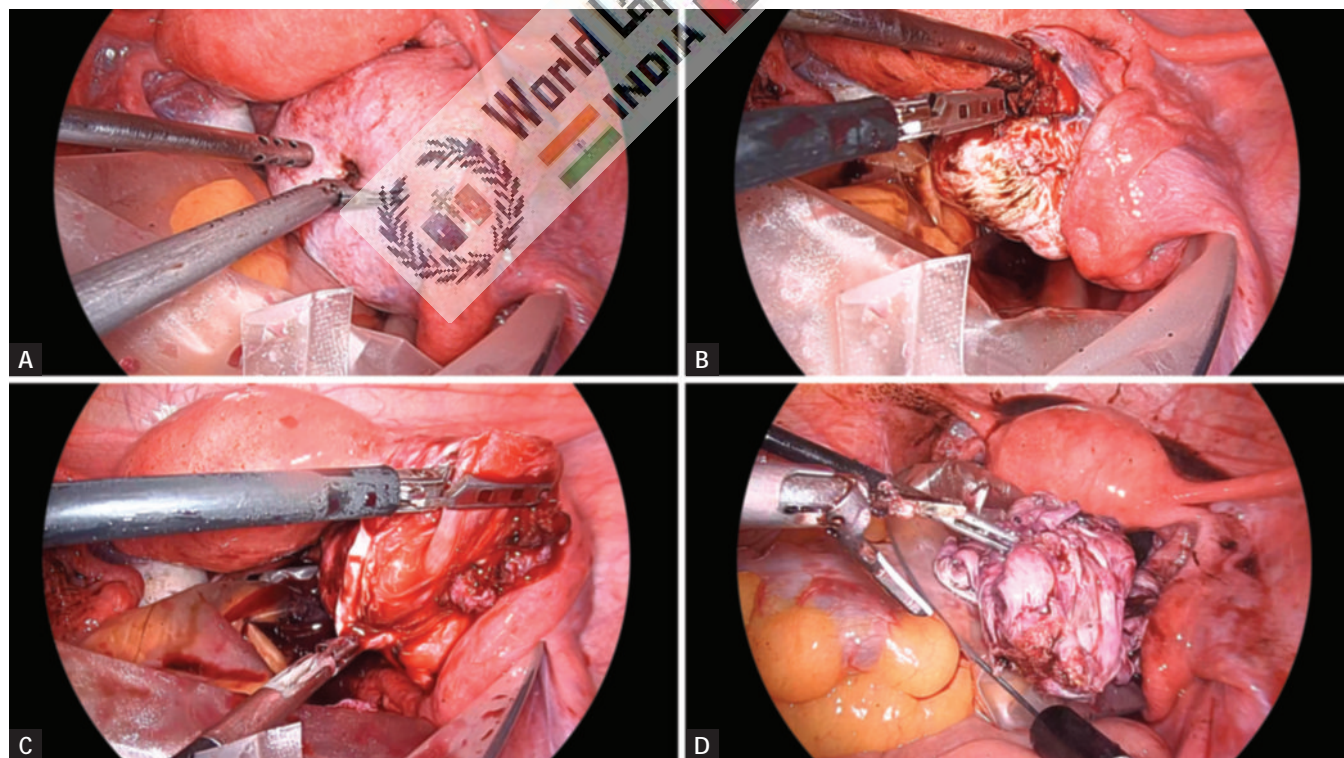
Superficial peritoneal endometriosis may be vaporized with monopolar or bipolar current or excised. Implants less than 2 mm are coagulated, vaporized, or excised. As lesions exceed 3 mm, vaporization or excision is needed. For lesions greater than 5 mm, deep vaporization or excisional techniques are required. If vaporization is chosen, it is important to copiously irrigate and remove the charred areas to confirm complete removal of the lesion and to avoid confusing endometriosis with a carbon deposit.

■ RESECTION OF OVARIAN ENDOMETRIOSIS

The ovaries are a common site for endometriosis. Endometrioma is formed when ectopic endometrial tissue within the ovary bleeds and results in a hematoma surrounded by duplicated ovarian parenchyma (**Figs. 1C and D**). Both ovaries are involved in one-third of patients. Endometrial implants or endometriomas less than 2 cm in diameter are coagulated, laser-ablated, or excised using scissors, biopsy forceps, or electrodes. For successful eradication, all visible lesions and scars must be removed from the ovarian surface. Entrapment of oocytes within the luteinized ovarian follicle, as reported in experimental animal models, must be avoided. Endometriomas more than 2 cm diameter must be resected thoroughly to prevent recurrence (**Figs. 2A to D**). Draining the endometriomas or partial resection of its wall is inadequate because the endometrial tissue lining the cyst is likely to remain functional and can cause the symptoms to recur (**Figs. 3A and B**). Many gynecologists like to perform ovarian cytology and biopsy of the cyst wall before ablating the cyst. By using a double optic laparoscope, which involves the passage of a smaller operative endoscope through the channel of the main laparoscope, the ovarian cyst may be punctured, drained, the fluid sent for cytology, and the lining of the inner cystic wall is visually inspected. Once it is confirmed that the cyst is not malignant, its wall is ablated to a depth of 3–4 mm.



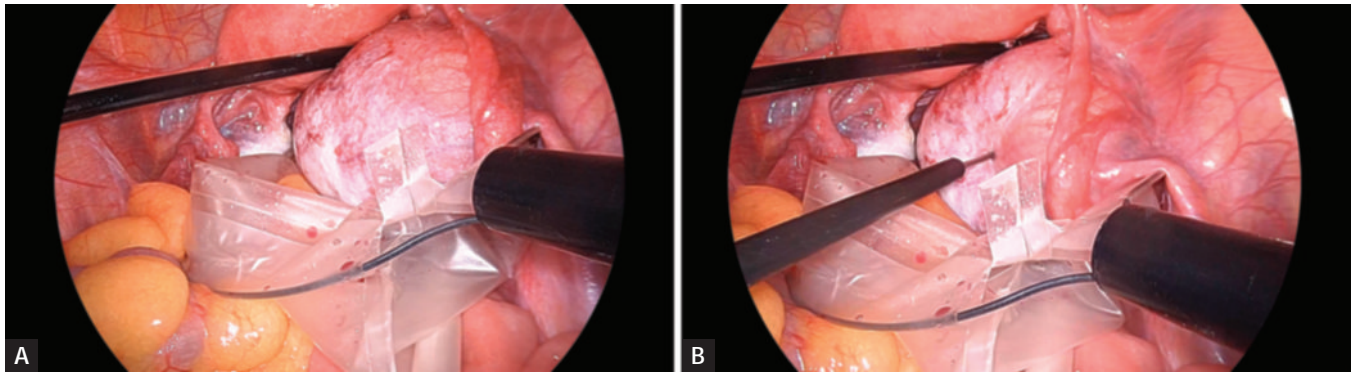
Figs. 1A to D: Endometriosis and endometrioma.



Figs. 2A to D: Endometrioma dissection.

For endometriomas over 2 cm in diameter, the cyst is punctured with aspiration needle and aspirated with the suction-irrigator probe. Deroofing of the cyst wall is

performed. The cyst wall should be removed by grasping its base with laparoscopic forceps and peeling it from the ovarian stroma. If the peeling of the remaining wall is not possible, it



Figs. 3A and B: Chocolate cyst kept inside endobag and aspirated.

should be ablated using electrosurgical fulguration. When the entire cyst wall is ablated, representative biopsies are taken for histological diagnosis.

Cyst wall closure is not necessary, according to animal experiments for large defects that result from resecting endometriomas larger than 5 cm; the edges of the ovarian cortex are approximated with a single suture placed within the ovarian stroma. Fibrin sealant has been described to atraumatically approximate the edges of large ovarian defects, without adhesion formation. Although rare, some patients present with localized symptoms and severe involvement of only one ovary with disease and adhesions while the opposite ovary is normal. These patients are benefited by unilateral salpingo-oophorectomy. By removing the diseased ovary, the risk of disease recurrence is minimized, and the fertility potential is improved by limiting ovulation to the healthy side.

■ GENITOURINARY ENDOMETRIOSIS

Ureteral involvement has been reported in 1–11% of women diagnosed with endometriosis. Endometriosis of the urinary tract generally tends to be superficial but can be invasive and cause complete ureteral obstruction. The symptom constellation of dysuria, urinary frequency and urgency, bladder infection, and hematuria is suggestive of bladder endometriosis in both women with known endometriosis and those who have not been diagnosed with endometriosis (Fig. 4).

Decreased bladder capacity and stability, which is unresponsive to conventional therapy, may result from endometriosis. When bladder symptoms are present, a course of danazol may be tried to see the improvement in bladder instability. Clinicians should consider endometriosis in cases of refractory and unexplained urinary complaints.

If urinary tract endometriosis is suspected, a complete preoperative evaluation is performed, including an intravenous pyelogram, ultrasound of the kidneys, and routine blood and urine workup. In selected cases of recurrent hematuria, cystoscopy is indicated. Superficial implants over the ureter can be treated by a variation of hydrodissection. Approximately 20–30 mL of Ringer's

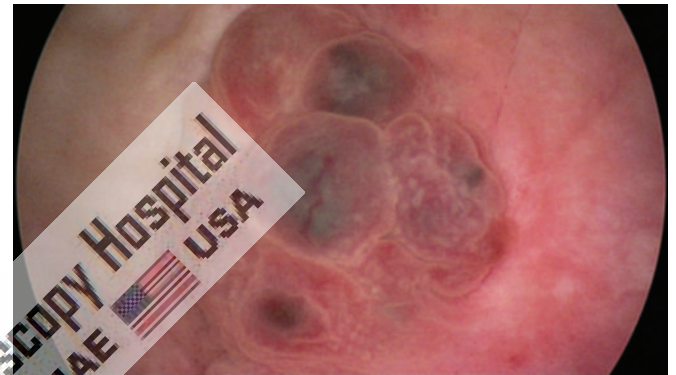
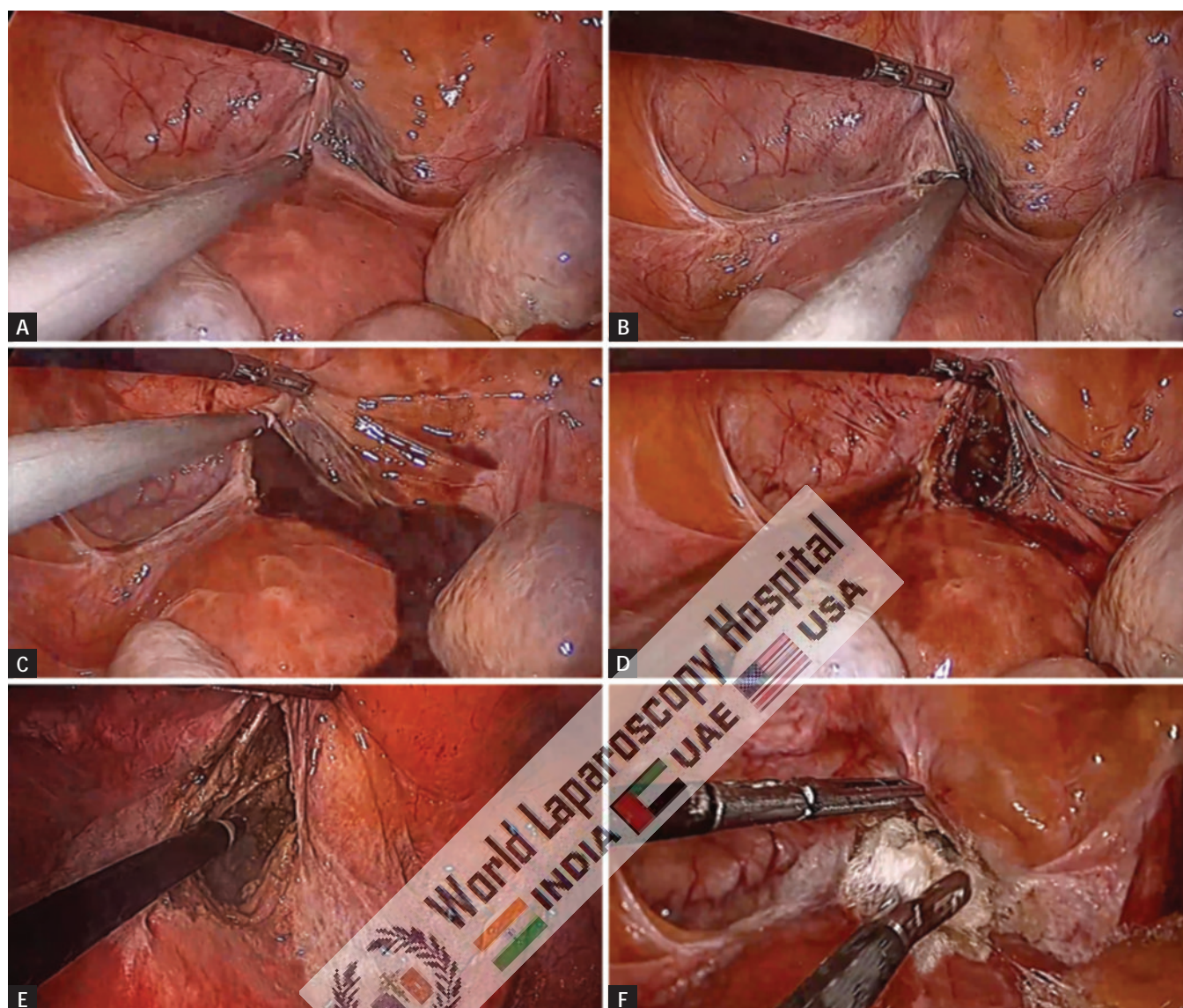


Fig. 4: Bladder endometriosis hysteroscopic view.

lactate is injected subperitoneally on the lateral pelvic wall; this elevates the peritoneum and backs it with a bed of fluid to prevent injury at the time of fulguration. The peritoneum is held with an atraumatic grasping forceps and peeled away with the help of a suction irrigation probe. Following hydrodissection of the broad ligaments and the pelvic sidewall, many patients develop swelling of the external genitalia, most likely from the penetration of water through the inguinal canal to the labia majora. This swelling resolves in most cases within 1–2 hours without sequelae.

The incidence of ureteral obstruction by endometriosis is low, and conventional therapy previously consisted of laparotomy followed by resection of the obstructed segment of the ureter. Laparoscopic ureteroureterostomy can be performed under direct laparoscopic observation.

The bladder wall is one of the sites least frequently involved with endometriosis. If the lesions are superficial, hydrodissection and vaporization are adequate for removal. Using hydrodissection, the areolar tissue between the serosa and muscularis beneath the implants is dissected. The lesion is circumscribed with the laser or monopolar and fluid is injected into the resulting defect. The lesion is grasped with forceps and dissected with the help of either sharp or electrosurgical dissection. Traction allows the small blood vessels supplying the surrounding tissue to be coagulated as the lesion is resected. Frequent irrigation is necessary



Figs. 5A to F: Dissection of bladder endometriosis, fulguration and packing with surgical snow.

to remove char, ascertain the depth of vaporization, and ensure that the lesion does not involve the muscularis and the mucosa. After proper fulguration of endometriotic tissue bladder integrity should be checked and surgical snow can be packed in the defect (**Figs. 5A to F**).

Endometriosis extending to the muscularis but without mucosal involvement can be treated laparoscopically and any residual or deeper lesions may be treated successfully with postoperative hormonal therapy. When endometriosis involves full bladder wall thickness, the lesion is excised and the bladder may be reconstructed laparoscopically.

■ GASTROINTESTINAL ENDOMETRIOSIS

Gastrointestinal endometriosis is believed to occur in 3–37% of women suffering from endometriosis. Endometriosis can involve rectovaginal septum, rectosigmoid colon, between the small intestines and anal canal. The symptoms are lower

abdominal pain, backache, dysmenorrhea, dyspareunia, diarrhea, constipation, and tenesmus. Occasionally rectal bleeding is also noticed. Typically these symptoms occur cyclically at or about the time of menstruation. Surgical intervention is necessary to dissect and resect the infiltrating bowel endometriosis. Intestinal endometriosis involves the rectum and sigmoid colon in 76% of cases, the appendix in 18%, and the cecum in 5%. Appendiceal lesion requires an appendectomy. In cases of severe disease of the bowel wall, resection and anastomosis is done laparoscopically. In cases of cul-de-sac endometriosis, because ureter is lateral to the uterosacral ligament, surgeons should try to dissect in between them. If the dissection is extended lateral to the uterosacral ligament, the ipsilateral ureter should be identified by opening the overlying peritoneum and kept safe and away from the area of the lesion. The ureter, uterine artery, and vein should be identified, and bipolar forceps or titanium clips must be used if bleeding starts.

■ DIAPHRAGMATIC ENDOMETRIOSIS

Endometriosis sometimes affects diaphragm also. In these cases, pleuritic shoulder or upper abdominal pain is present at the time of menses. Laparoscopy is an excellent modality to diagnose and treat diaphragmatic endometriosis. Follow-up medical treatment is necessary because extensive surgery can damage or even rupture the diaphragm. Bilateral oophorectomy is promising, and further intervention may not be necessary. Three cannulas are required in the upper quadrant according to the site of the lesion on the diaphragm. A liver retractor is used by one port, and lesions are removed using hydrodissection and vaporization or excision. If the injury to diaphragm happens, it should be repaired with a 4-0 PDS. Cardiopulmonary resuscitation may be necessary after surgery.

■ PROGNOSIS

Complete surgical removal of endometriosis is associated with long-term control of symptoms. In the largest case series of 275 women surgically treated for deeply infiltrating endometriosis, 10% required repeat surgery, all women reported an improvement in pain symptoms, and 77% reported the pain improvement as “excellent” during a mean follow-up of 5 years. The incidence of endometriosis in women with dysmenorrhea is up to 40–60%, whereas in women with subfertility is up to 20–30%. The recurrence of endometriosis varies greatly among different studies. The overall recurrence rates range between 6 and 67% according to the criteria that are taken into consideration. Which of the various reasons is more predictive for recurrence is still unclear and controversial. The main aim of postoperative medical treatment is suppressing ovarian activity leading to atrophy of endometriotic lesions. The success of treatment depends on the resorption of all visible residual lesions and the eradication of microscopic implants. The recurrent lesions might originate from residual lesions or from de novo cells. Determining risk factors for recurrence may allow the identification of subgroups at risk for disease control. Potential biomarkers for recurrence could also maintain targeted therapy. It is difficult to eliminate the risk of endometriosis recurrence. Various risk factors have been suggested for recurrence in the literature, and these are both patient- and surgeon-dependent. According to the risk factor and the follow-up period, the recurrence rates may also alter. Finding an ideal biomarker may clarify the recurrence process so that individualized treatment can be maintained.

■ BIBLIOGRAPHY

- Adamson GD, Kennedy S, Hummelshoj L. Creating solutions in endometriosis: global collaboration through the World Endometriosis Research Foundation. *J Endometriosis*. 2010;2:3-6.
- Anglesio MS, Papadopoulos N, Ayhan A, Nazeran TM, Noë M, Horlings HM, et al. Cancer-associated mutations in endometriosis without cancer. *N Engl J Med*. 2017;376:1835.
- Audebert A, Petousis S, Margioulas-Siarkou C, Ravanos K, Prapas N, Prapas Y, et al. Anatomic distribution of endometriosis: a reappraisal based on series of 1101 patients. *Eur J Obstet Gynecol Reprod Biol*. 2018;230:36-40.
- Ballard KD, Seaman HE, de Vries CS, Wright JT. Can symptomatology help in the diagnosis of endometriosis? Findings from a national case-control study--Part 1. *BJOG*. 2008;115:1382.
- Blanco RG, Parthivel VS, Shah AK, Gumbs MA, Schein M, Gerst PH. Abdominal wall endometriomas. *Am J Surg*. 2003;185:596.
- Brosens I, Puttemans P, Benagiano G. Endometriosis: a life cycle approach? *Am J Obstet Gynecol*. 2013;209:307.
- Brosens IA, Puttemans PJ, Deprest J. The endoscopic localization of endometrial implants in the ovarian chocolate cyst. *Fertil Steril*. 1994;61:1034.
- Burney RO, Giudice LC. Pathogenesis and pathophysiology of endometriosis. *Fertil Steril*. 2012;98:511.
- Canlorbe G, Laas E, Cortez A, Darai E. Spontaneous hymeneal endometriosis: a rare cause of dyspareunia. *BMJ Case Rep*. 2014;2014.
- Chatman DL, Ward AB. Endometriosis in adolescents. *J Reprod Med*. 1982;27:156.
- Clement PB. The pathology of endometriosis: a survey of the many faces of a common disease emphasizing diagnostic pitfalls and unusual and newly appreciated aspects. *Adv Anat Pathol*. 2007;14:241.
- Czernobilsky B, Morris WJ. A histologic study of ovarian endometriosis with emphasis on hyperplastic and atypical changes. *Obstet Gynecol*. 1979;53:318.
- De Cicco C, Corona R, Schonman R, Mailova K, Ussia A, Koninckx P. Bowel resection for deep endometriosis: a systematic review. *BJOG*. 2011;118:285.
- Dovey S, Sanfilippo J. Endometriosis and the adolescent. *Clin Obstet Gynecol*. 2010;53:420.
- Du H, Taylor HS. Contribution of bone marrow-derived stem cells to endometrium and endometriosis. *Stem Cells*. 2007;25:2082.
- Dwivedi AJ, Agrawal SN, Silva YJ. Abdominal wall endometriomas. *Dig Dis Sci*. 2002;47:456.
- Eskenazi B, Warner ML. Epidemiology of endometriosis. *Obstet Gynecol Clin North Am*. 1997;24:235.
- Farland LV, Eliassen AH, Tamimi RM, Spiegelman D, Michels KB, Missmer SA. History of breast feeding and risk of incident endometriosis: prospective cohort study. *BMJ*. 2017;358:j3778.
- Fukunaga M, Nomura K, Ishikawa E, Ushigome S. Ovarian atypical endometriosis: its close association with malignant epithelial tumours. *Histopathology*. 1997;30:249.
- Giudice LC. Clinical practice. Endometriosis. *N Engl J Med*. 2010;362:2389.
- Goldstein DP, deCholnoky C, Emans SJ, Leventhal JM. Laparoscopy in the diagnosis and management of pelvic pain in adolescents. *J Reprod Med*. 1980;24:251.
- Gruenwald P. Origin of endometriosis from the mesenchyme of the celomic walls. *Am J Obstet Gynecol*. 1942;44:470.
- Gustofson RL, Kim N, Liu S, Stratton P. Endometriosis and the appendix: a case series and comprehensive review of the literature. *Fertil Steril*. 2006;86:298.
- Halme J, Hammond MG, Hulka JF, Raj SG, Talbert LM. Retrograde menstruation in healthy women and in patients with endometriosis. *Obstet Gynecol*. 1984;64:151.
- Harris HR, Wieser F, Vitonis AF, Rich-Edwards J, Boynton-Jarrett R, Bertone-Johnson ER, et al. Early life abuse and risk of endometriosis. *Hum Reprod*. 2018;33:1657.
- Hediger ML, Hartnett HJ, Louis GM. Association of endometriosis with body size and figure. *Fertil Steril*. 2005;84:1366.
- Hickey M, Ballard K, Farquhar C. Endometriosis. *BMJ*. 2014;348:g1752.

28. Horton JD, Dezee KJ, Ahnfeldt EP, Wagner M. Abdominal wall endometriosis: a surgeon's perspective and review of 445 cases. *Am J Surg.* 2008;196:207.
29. Jaime TJ, Jaime TJ, Ormiga P, Leal F, Nogueira OM, Rodrigues N. Umbilical endometriosis: report of a case and its dermoscopic features. *An Bras Dermatol.* 2013;88(1):121-4.
30. Jansen RP, Russell P. Nonpigmented endometriosis: clinical, laparoscopic, and pathologic definition. *Am J Obstet Gynecol.* 1986;155:1154.
31. Javert CT. The spread of benign and malignant endometrium in the lymphatic system with a note on coexisting vascular involvement. *Am J Obstet Gynecol.* 1952;64:780.
32. Jenkins S, Olive DL, Haney AF. Endometriosis: pathogenetic implications of the anatomic distribution. *Obstet Gynecol.* 1986;67:335.
33. Kavoussi SK, Odenwald KC, As-Sanie S, Lebovic DI. Incidence of ovarian endometrioma among women with peritoneal endometriosis with and without a history of hormonal contraceptive use. *Eur J Obstet Gynecol Reprod Biol.* 2017; 215:220.
34. Kirshon B, Poindexter AN 3rd. Contraception: a risk factor for endometriosis. *Obstet Gynecol.* 1988;71:829.
35. Laufer MR, Goitein L, Bush M, Cramer DW, Emans SJ. Prevalence of endometriosis in adolescent girls with chronic pelvic pain not responding to conventional therapy. *J Pediatr Adolesc Gynecol.* 1997;10:199-202.
36. Lee HJ, Park YM, Jee BC, Kim YB, Suh CS. Various anatomic locations of surgically proven endometriosis: a single-center experience. *Obstet Gynecol Sci.* 2015;58:53.
37. Longo LD. Classic pages in obstetrics and gynecology. Aberrant portions of the müllerian duct found in an ovary: William Wood Russell Johns Hopkins Hospital Bulletin, vol. 10, pp. 8-10, 1899. *Am J Obstet Gynecol.* 1979;134:225.
38. Mahmood TA, Templeton A. Prevalence and genesis of endometriosis. *Hum Reprod.* 1991;6:544.
39. Marinis A, Vassiliou J, Kannas D, Theodosopoulos TK, Kondi-Pafiti A, Kairi E, et al. Endometriosis mimicking soft tissue tumors: diagnosis and treatment. *Eur J Gynaecol Oncol.* 2006;27:168.
40. Marsh EE, Laufer MR. Endometriosis in premenarcheal girls who do not have an associated obstructive anomaly. *Fertil Steril.* 2005;83:758.
41. Minaglia S, Mishell DR Jr, Ballard CA. Incisional endometriomas after cesarean section: a case series. *J Reprod Med.* 2007;52:630.
42. Missmer SA, Chavarro JE, Malspeis S, Bertone-Johnson ER, Hornstein MD, Spiegelman D, et al. A prospective study of dietary fat consumption and endometriosis risk. *Hum Reprod.* 2010;25:1528.
43. Missmer SA, Hankinson SE, Spiegelman D, Barbieri RL, Malspeis S, Willett WC, et al. Reproductive history and endometriosis among premenopausal women. *Obstet Gynecol.* 2004;104: 965-74.
44. Missmer SA, Hankinson SE, Spiegelman D, Barbieri RL, Marshall LM, Hunter DJ. Incidence of laparoscopically confirmed endometriosis by demographic, anthropometric, and lifestyle factors. *Am J Epidemiol.* 2004;160:784.
45. Missmer SA, Hankinson SE, Spiegelman D, Barbieri RL, Michels KB, Hunter DJ. In utero exposures and the incidence of endometriosis. *Fertil Steril.* 2004;82:1501.
46. Morales Martínez C, Tejuca Somoano S. Abdominal wall endometriosis. *Am J Obstet Gynecol.* 2017; 217:701.
47. Mowers EL, Lim CS, Skinner B, Mahnert N, Kamdar N, Morgan DM, et al. Prevalence of endometriosis during abdominal or laparoscopic hysterectomy for chronic pelvic pain. *Obstet Gynecol.* 2016;127:1045.
48. Muzii L, Bianchi A, Bellati F, Zullo MA, Angioli R, Panici PB, et al. Histologic analysis of endometriomas: what the surgeon needs to know. *Fertil Steril.* 2007;87:362.
49. Nnoaham KE, Webster P, Kumbang J, Kennedy SH, Zondervan KT. Is early age at menarche a risk factor for endometriosis? A systematic review and meta-analysis of case-control studies. *Fertil Steril.* 2012;98:702.
50. Ogawa S, Kaku T, Amada S, Kobayashi H, Hirakawa T, Ariyoshi K, et al. Ovarian endometriosis associated with ovarian carcinoma: a clinicopathological and immunohistochemical study. *Gynecol Oncol.* 2000;77:298.
51. Olive DL, Henderson DY. Endometriosis and müllerian anomalies. *Obstet Gynecol.* 1987;69:412.
52. Parazzini F, Cipriani S, Bianchi S, Gotsch F, Zanconato G, Fedele L. Risk factors for deep endometriosis: a comparison with pelvic and ovarian endometriosis. *Fertil Steril.* 2008;90:174.
53. Prefumo F, Todeschini F, Fulcheri E, Venturini PL. Epithelial abnormalities in cystic ovarian endometriosis. *Gynecol Oncol.* 2002;84:280.
54. Rahmiloglu N, Nyholt DR, Morris AP, Missmer SA, Montgo GW. Genetic variants underlying risk of endometriosis: insights from meta-analysis of eight genome-wide association and replication datasets. *Hum Reprod Update.* 2014;20:702.
55. Redwine DB. Diaphragmatic endometriosis: diagnosis, surgical management, and long-term results of treatment. *Fertil Steril.* 2002;77:288.
56. Reese KA, Reddy S, Rock JA. Endometriosis in an adolescent population: the Emory experience. *J Pediatr Adolesc Gynecol.* 1996;9:125.
57. Sampson JA. Peritoneal endometriosis due to the menstrual dissemination of endometrial tissue into the peritoneal cavity. *Am J Obstet Gynecol.* 1927;14:422.
58. Sangi-Haghpeykar H, Poindexter AN 3rd. Epidemiology of endometriosis among parous women. *Obstet Gynecol.* 1995;85:983.
59. Seidman JD. Prognostic importance of hyperplasia and atypia in endometriosis. *Int J Gynecol Pathol.* 1996;15:1.
60. Shafirir AL, Farland LV, Shah DK, Harris HR, Kvaskoff M, Zondervan K, et al. Risk for and consequences of endometriosis: a critical epidemiologic review. *Best Pract Res Clin Obstet Gynaecol.* 2018;51:1.

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