

---

## Laparoscopic Excision of Endometriosis

# 5

Rebecca Rossener, Luiz Fernando Pina Carvalho,  
Juan Luis Salgado, and Mauricio S. Abrao

Endometriosis is a gynecologic disease that affects more than 150 million women around the world. The disease is usually found in the pelvic cavity, affecting the ovaries, the fallopian tubes, the peritoneum, and the rectovaginal septum; it is less commonly found in the abdominal cavity affecting the small bowel, the large bowel, and other abdominal organs. It has been established that the best examination to evaluate endometriosis is transvaginal ultrasonography with bowel preparation. The laparoscopic ablation or excision of endometriotic lesions has as its objectives pain relief or fertility improvement and increasing the patient's quality of life. It is essential to remove all visible lesions and to biopsy the doubtful ones in order to reduce the

chances of recurrences and repeat surgery. Therefore, it is important to keep in mind that the laparoscopic treatment should always be a diagnostic procedure as well, regardless of the previous imaging evaluation. The appearance of peritoneal endometriotic lesions in laparoscopy is widely variable. They can have the typical black appearance or atypical red, white, or yellow lesions, or they can form adhesions and anatomic distortions. Ovarian endometriosis can present as hemorrhagic superficial lesions or hemorrhagic cysts. The surgery for endometriomas must minimize the ovarian tissue trauma in all possible situations and concurrently remove all unhealthy tissue. The deep infiltrative lesions, on the other hand, are focused on a clinical presentation centered on pain. The operation comprises liberation of the adhesions or, less commonly, bowel resection. Endometriotic lesions in the urinary tract usually have cyclical urinary alterations as symptoms. The surgery is done aiming at minimal damage to the tissues. Laparoscopic surgery has a fundamental role in the management of endometriosis. In conclusion, surgical planning must be aligned with each patient's pathologic condition. It is still the gold standard for diagnosis, and excision of lesions can reduce pain symptoms and increase the fertility rate, contributing to improving patient quality of life.

---

R. Rossener • L.F.P. Carvalho • M.S. Abrao, MD (✉)  
Department of Obstetrics and Gynecology,  
University of São Paulo, São Paulo, SP, Brazil  
e-mail: [rebecca.rossener@gmail.com](mailto:rebecca.rossener@gmail.com);  
[luizcarvalho.dr@me.com](mailto:luizcarvalho.dr@me.com); [msabrao@mac.com](mailto:msabrao@mac.com)

J.L. Salgado, MD  
Department of Obstetrics and Gynecology,  
Universidad Central Caribe School of Medicine,  
Bayamon, PR 00956, USA  
e-mail: [salgadomd@migsec.com](mailto:salgadomd@migsec.com)

## 5.1 Introduction

Endometriosis is a gynecologic disease that affects more than 150 million women around the world. It does not discriminate based on ethnicity or social background. Several studies have shown that the prevalence of the disease affects approximately 10 % of women in the reproductive age group, meaning that one 1 of every 10 women will have endometriosis [1].

Endometriosis is defined as the occurrence of endometrial stroma and glands outside the endometrial cavity. The ectopic implants can induce a chronic inflammatory reaction that leads to adhesions and distortion of the tissues and pelvic anatomy [2].

The disease is usually found in the pelvic cavity affecting the ovaries, fallopian tubes, peritoneum, rectovaginal septum; less commonly it is found in the abdominal cavity, the small bowel, the large bowel, and other abdominal organs [2]. In addition, there are reports of endometriosis affecting distal organs, such as the lungs, brains, and eyes [3].

Endometriosis is very commonly diagnosed several years after initial symptoms appear [4, 5] owing to lack of patients' awareness about the significance of their symptoms, assuming that some of them, like dysmenorrhea and dyspareunia, are normal. A lack of knowledge about the disease may also delay physician diagnosis [4].

Consequently, many patients are found with an advanced stage of endometriosis at the time of initial diagnosis. Several organizations worldwide are working to create awareness so that diagnosis is made earlier. Early diagnosis and treatment can prevent complications. Even though endometriosis is not cancer, it behaves similarly by invading and not respecting the boundaries of the organs.

## 5.2 Clinical Aspects

There are many ways to classify endometriosis and the distribution of lesions found at each stage, but none have been fully accepted. The most commonly used classification is from the American Society for Reproductive Medicine (ASRM) [6], in which Stage I disease is minimal, Stage II is mild, Stage III is moderate, and Stage IV is severe as seen in Fig. 5.1). Therefore, the classification of disease can only be determined after surgical pelvic evaluation [6].

Owing to the difficulties found in the use of the existing methods of classification, the American Association of Gynecological Laparoscopists (AAGL) is formulating a new classification for the disease based on pain, infertility, and surgical difficulty, which are criteria that are not considered by the ASRM classification [7]. The main symptoms of endometriosis are dysmenorrhea, chronic pelvic pain, dyspareunia, cyclical intestinal alterations, cyclical urinary alterations, and infertility, with the latter occurring in 40 % of women with endometriosis [2, 8]. Pain is usually assessed by a visual scale analysis. The clinical examination, including the gynecologic examination, can be normal or unspecific. Some of the alterations that can be found are visible lesions on external genital organs, pain during the mobilization of the cervix, nodules, and thickenings on palpable ligaments.

Many scientists have attempted explanations Physiopathology for the development of endometriosis, such as endometrial tissue and cell reflux during menstruation as described by Sampson [9], the differentiation of other cells from endometrial cells (metaplasia), and the hematogenous and lymphatic spread of endometrial cells [9, 10]. However, the disease has been evidenced in patients with the absence of a uterus or in men who have taken high doses of estrogen for prostatic cancer, showing that no theory fully explains the physiopathology of endometriosis [9, 10].



**AMERICAN SOCIETY FOR REPRODUCTIVE MEDICINE  
REVISED CLASSIFICATION OF ENDOMETRIOSIS**

Patient's Name \_\_\_\_\_ Date \_\_\_\_\_  
 Stage I (Minimal) - 1-5  
 Stage II (Mild) - 6-15  
 Stage III (Moderate) - 16-40  
 Stage IV (Severe) - >40  
 Total \_\_\_\_\_

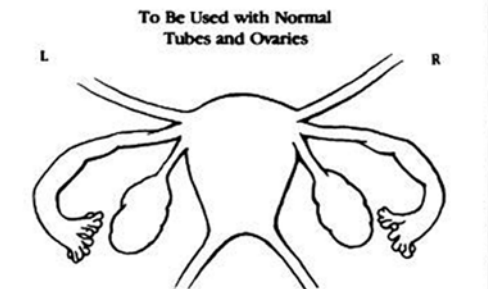
Laparoscopy \_\_\_\_\_ Laparotomy \_\_\_\_\_ Photography \_\_\_\_\_  
 Recommended Treatment \_\_\_\_\_  
 Prognosis \_\_\_\_\_

PERITONEUM	<b>ENDOMETRIOSIS</b>	<1cm	1-3cm	>3cm
	Superficial	1	2	4
	Deep	2	4	6
OVARY	R Superficial	1	2	4
	Deep	4	16	20
	L Superficial	1	2	4
	Deep	4	16	20
<b>POSTERIOR CULDESAC OBLITERATION</b>		Partial	Complete	
		4	40	
OVARY	<b>ADHESIONS</b>	<1/3 Enclosure	1/3-2/3 Enclosure	>2/3 Enclosure
	R Filmy	1	2	4
	Dense	4	8	16
	L Filmy	1	2	4
	Dense	4	8	16
TUBE	R Filmy	1	2	4
	Dense	4*	8*	16
	L Filmy	1	2	4
	Dense	4*	8*	16

\*If the fimbriated end of the fallopian tube is completely enclosed, change the point assignment to 16.  
 Denote appearance of superficial implant types as red [(R), red, red-pink, flamelike, vesicular blobs, clear vesicles], white [(W), opacifications, peritoneal defects, yellow-brown], or black [(B) black, hemosiderin deposits, blue]. Denote percent of total described as R \_\_%, W \_\_% and B \_\_%. Total should equal 100%.

Additional Endometriosis: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

Associated Pathology: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_



**Fig. 5.1** Revised classification of endometriosis by American Society for Reproductive Medicine. The localization and size of the endometriotic lesions are the main markers used to provide classification of the disease

(According to the American Society for Reproductive Medicine, classification can only be determined after surgery)

**Table 5.1** Main symptoms of endometriosis

Symptom	Peritoneal	Ovarian	Deep	P
Severe dysmenorrhea	22 (51.8 %)	126 (48.5 %)	229 (62.9 %)	0.005
Chronic pain	96 (50.3 %)	143 (54.8 %)	233 (63.5 %)	0.006
Infertility	56 (28.7 %)	66 (25.2 %)	124 (34.1 %)	0.03
Cyclic dyschezia	21 (11.4 %)	33 (13 %)	120 (33.5 %)	<0.001
Cyclic dysuria	27 (14.1 %)	34 (13 %)	56 (15.3 %)	0.71
Dyspareunia	97 (51.6 %)	138 (52.9 %)	227 (63.4 %)	0.007

A study of 819 patients helped define main symptoms and their variance of the disease [8]

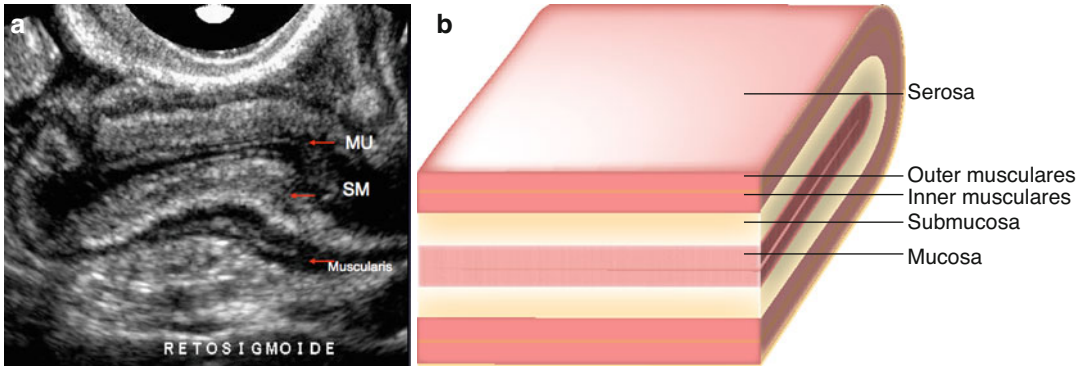
What is known is that genetics plays an important role in its development. Patients who have first-degree relatives who have or have had the disease have a six times higher possibility of developing endometriosis than those without such a relative. The prevalence is 4–9 % in first-degree relatives [11]. Diagnosis histology is still the gold standard to diagnose endometriosis. Biopsy is done by laparoscopy. However, surgical diagnosis is considered an invasive procedure, and efforts are being made to develop less invasive techniques with high sensibility and specificity, such as transvaginal ultrasonography and the CA 125 test.

The CA 125 blood levels measurement is a laboratory test commonly used as an ovarian cancer marker of epithelial origin and also to evaluate patient response to chemotherapy. But it can also be used as a predictor of endometriosis [12]. However, many studies demonstrate that it is not as good a predictor for early stages such as I and

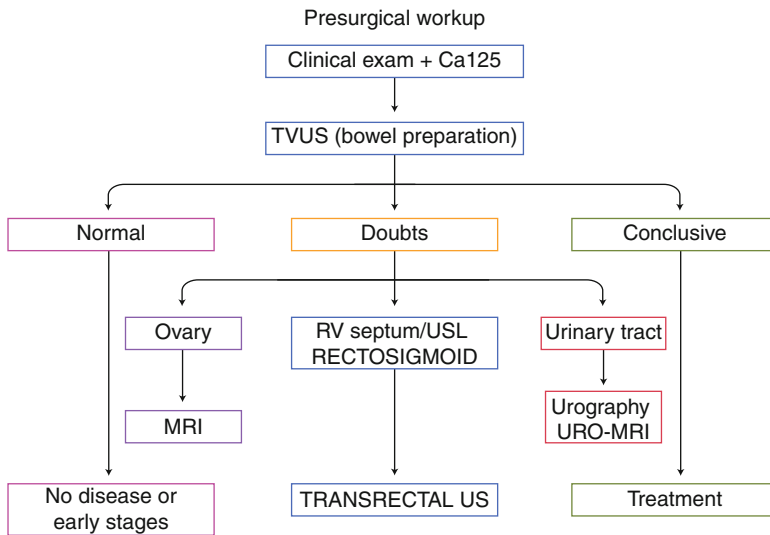
II (mild and moderate endometriosis) as it is for severe endometriosis [13].

It has been established that the best examination to evaluate endometriosis is transvaginal ultrasonography (TV-USG) with bowel preparation. It can detect lesions that are larger than 2 cm. For deep infiltrating endometriosis (DIE), studies have demonstrated that the TV-USG has 98 % sensibility and 100 % specificity, which are better than the results of magnetic resonance imaging (MRI) [14]. Therefore, it is important and feasible to diagnose the disease early in life in order to prevent long-term complications.

Likewise, ovarian endometriosis has been well documented with TV-USG as well as with MRI. Both show the same results, but the former is less expensive. On the other hand, lesions involving ureters are better evaluated by MRI than by TV-USG for a more accurate identification of the site of the lesion.



**Fig. 5.2** Transvaginal ultrasound showing intestinal layers. (a) A rectosigmoid section showed by ultrasound with all layers delimited. (b) With careful analysis of each layer, endometriotic lesions can be diagnosed by ultrasound. *MU* musculares, *SM* submucosas



**Fig. 5.3** Flow chart for diagnosis of endometriosis. The investigation starts with the clinical history and the physical examination; although the measure of CA 125 levels can be used as well, it has low sensibility and specificity. The next step is to perform a transvaginal ultrasound (TVUS) with bowel preparation by professionals specializing in finding lesions of endometriosis in ultrasound images. If

these steps are conclusive for endometriosis, then treatment should be planned. With a normal image examination, there is probably no disease or it is in very early stages. If the initial investigation is inconclusive, other imaging examinations can be indicated according to the probable location of the lesions. *MRI* magnetic resonance imaging, *URO-MRI* urologic magnetic resonance imaging

### 5.3 Treatment

Treatment for endometriosis should be individualized for each patient. Management of the disease should only be initiated after careful analysis of symptoms and image examinations, after thorough discussion with the patient regarding the consequences of each procedure and reason for treatment (pain, pregnancy). Furthermore, endometriosis is frequently a multisystem disease, and this is the reason why treatment requires a multidisciplinary team to provide the best care for patients [15]. This often includes other areas of medicine, such as proctology and urology, as well as other health care areas, such as psychology and physiotherapy.

Treatment of endometriosis consists mainly of three procedures: pharmacologic ovarian suppression, painkillers, and video-assisted laparoscopic surgery. The pharmacologic treatment involves any agent that blocks ovarian hormone production and theoretically reduces pain, as endometriotic lesions are responsive to hormone levels [16].

### 5.4 Laparoscopic Surgery

The benefits of laparoscopic surgery versus laparotomy for endometriosis have been established in the literature [17]. Laparoscopic surgery provides better visualization of the pelvic cavity, less formation of adhesions, faster discharge of the patient, lower costs, and less postoperative pain, the last being essential for a patient who already suffers pain owing to the disease.

The objectives of laparoscopic ablation or excision of endometriotic lesions include pain relief, improved fertility, and improved patient quality of life [18]. It is imperative to remove all visible lesions and to biopsy any doubtful lesions to reduce the chance of recurrence and reoperation [19]. However, even with complete resection, the recurrence rate is high [19]. Fibrotic tissue surrounding the lesions should be removed as well because the tissue is reactive to hormones and can lead to recurrence of disease [19].

The main indications for laparoscopic surgery are pain, pelvic pain refractory to pharmacologic treatment, severe disease with anatomic distortion, large endometriomas, bowel involvement, urinary obstructions, contraindication for hormone therapy, and potential malignant disease. There is also scientific evidence supporting the benefits of laparoscopic surgery in infertile endometriosis patients who have previously failed to conceive spontaneously or by in vitro fertilization (IVF). Laparoscopic surgery has been shown to increase pregnancy outcome [20].

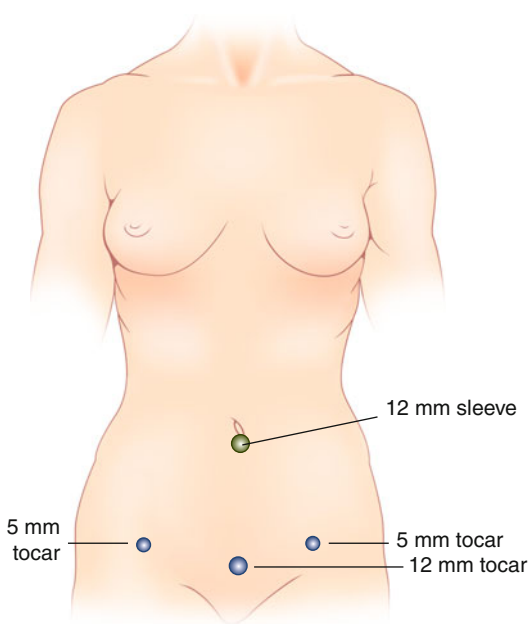
The first step of laparoscopic treatment should always be a diagnostic procedure, despite previous image evaluation. Careful analysis of pelvic anatomy during surgery can help with more precise excision of lesions and in classification of disease stage. Many implants are called “iceberg lesions,” when the deep level of the disease is not consistent with its superficial laparoscopic appearance, another reason to insist on a complete examination of pelvic lesions.

There are two possible surgical approaches: conservative and complete. Conservative surgery consists of maintaining the uterus and as much of the ovarian tissue as possible in order to preserve fertility. On the other hand, complete surgery is

characterized by total removal of the uterus with or without removal of the ovaries. According to the ASRM, the indications for a complete surgery are recurrent conservative surgeries, disabling pain without reproductive desire, and associated uterine diseases that must be treated with hysterectomy [21].

A low-residue diet is recommended before all surgeries, and bowel preparation is necessary when lesions are in the rectovaginal septum or when there is need for a hysterectomy. The anesthesia is general and can be associated with peridural blockage. The patient is put in a Trendelenburg position between a 10-degree and 45-degree incline in order to keep the intestinal loops away from field of vision [22].

Laparoscopy is initiated with a vertical incision in the inferior portion of the umbilical scar and insufflation of carbon dioxide with a Veress needle to deliberately create a pneumoperitoneum. A 10-mm trocar is inserted into the umbilical incision, and two other auxiliary punctures

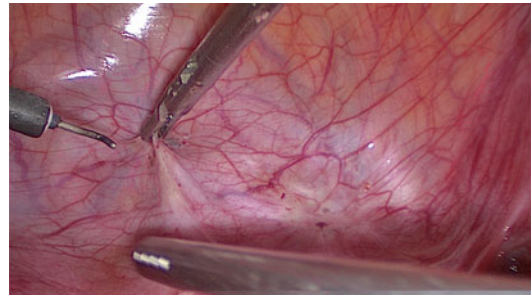


**Fig. 5.4** Position of trocars in a laparoscopic procedure. Generally, a 12-mm trocar is inserted into the umbilical incision, and two 5-mm trocars are inserted in the supra-pubic area. Depending on the surgeon, another 5-mm trocar can be inserted in the supra-pubic area

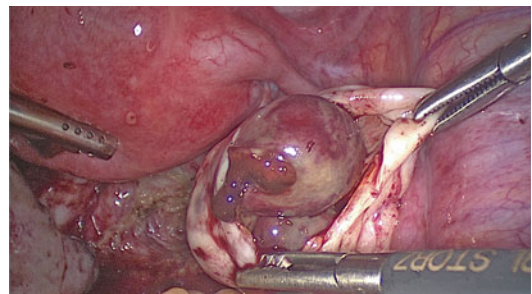
are made in the supra-pubic area to insert 5-mm trocars. In some cases, another 5-mm port can be placed for better triangulation [22]. If the content to be removed is larger, one of the auxiliary trocars can be substituted for a 12-mm port.

The appearance of peritoneal endometriotic lesions in laparoscopy is widely variable. They can have a typical black or atypical red, white, or yellow appearance, or form adhesions and anatomic distortions [6]. Removal of lesions is usually done by excision or cauterization. Excision includes carbon dioxide laser or monopolar current for bigger lesions. Cauterization is used to treat more punctiform lesions with a bipolar current laser until normality is restored.

Ovarian endometriosis can present as hemorrhagic superficial lesions or hemorrhagic cysts. Those lesions hardly respond to treatment and is the reason that the presence of large endometriomas is an important indication for surgery. It has already been demonstrated that ovarian endometriosis is



**Fig. 5.5** Peritoneal endometriotic lesions. In this image of a laparoscopic surgery, red lesions of peritoneal endometriosis can be seen



**Fig. 5.6** Endometrioma. A large endometrioma looking like a hemorrhagic (or chocolate, in current language) cyst that is leaking

associated with an increased risk of ovarian cancer [23]. Therefore, it is absolutely necessary to look for malignant aspects of the ovaries during surgery. When performing a conservative procedure, trauma to the ovarian tissue must be minimized, with concurrent removal of all unhealthy tissue [24].

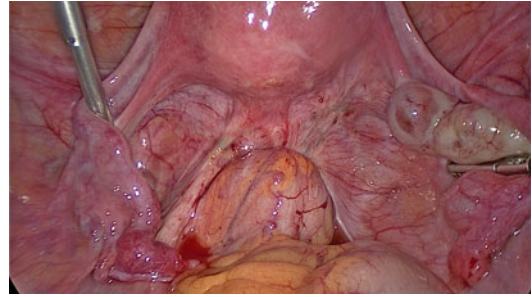
There are many different ways to remove an endometrioma. The two most common procedures are drainage of the cysts or cystectomy. There is a great deal of discussion about the advantages and detriments of each technique [25]. If the cyst is smaller than 3 cm, incision, drainage, aspiration, and capsule cauterization can be performed. If the endometrioma is larger than 3 cm, implying greater ovarian damage, a cystectomy with complete removal of the capsule is recommended [26].

Deep infiltrative lesions are responsible for clinical presentation of pain. It can affect the rectovaginal septum and the ureters and bowel, and preoperative care involves bowel preparation and antibiotics. The disease can be total or partial and is determined during the surgery. If the cul-de-sac is normal, impairment is partial. If the rectum is adhered to the rectovaginal septum or the uterus, there is total impairment. The operation comprises the liberation of the anterior wall of the rectum and the uterosacral ligaments with laser, electrosurgery, or scissors. According to Koninckx and coworkers [27], bowel resections are rare in these surgeries unless the nodule is in the bowel.

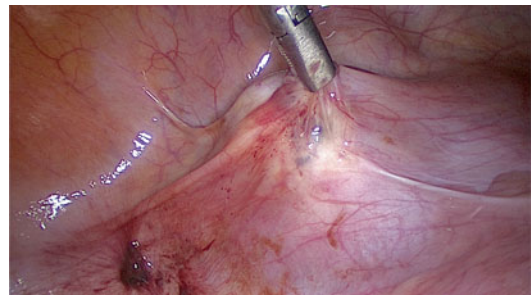
The laparoscopic segmental resection of the rectum affected by endometriosis includes the following successive steps:



**Fig. 5.7** Deep infiltrative endometriosis. Laparoscopic image showing the anatomic pelvic distortion caused by deep infiltrative disease



**Fig. 5.8** Bowel endometriosis. Laparoscopic image of small bowel endometriotic lesions



**Fig. 5.9** Endometriosis of the urinary tract. Laparoscopic image of endometriosis in the bladder

1. Placement of an umbilical incision and insufflation of CO<sub>2</sub> through the Veress needle to obtain proper pneumoperitoneum and subsequent insertion of a 10-mm trocar and optics.
2. Insertion of three auxiliary trocars, two at the iliac fossa (10–12 mm on the right and 5 mm on the left) and a 5-mm trocar in the left flank.
3. Examination of the abdominal and pelvic cavities and identification of all the sites affected by endometriosis.
4. Lysis of the any adhesions affecting the adnexal regions, uterine fundus, posterior cul-de-sac, and uterosacral ligaments and relevant bowel adhesions.
5. Release of the sigmoid from the left lateral abdominal wall and from the retroperitoneum and identification of the left ureter up to the level of the pelvic brim.
6. Opening of the mesosigmoid.
7. Mobilization of the rectum by dissecting its anterior wall from the posterior surface of the

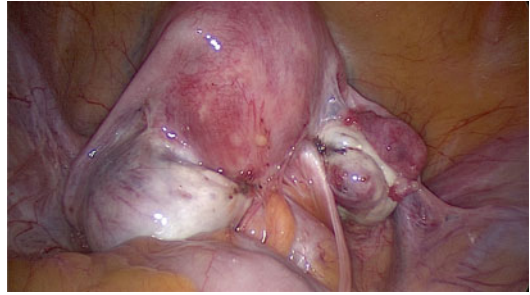


cervix, followed by a linear stapler applied distal to the area affected by the disease.

8. The 10–12-mm incision on the right iliac fossa is enlarged sufficiently to exteriorize the divided bowel enclosing the diseased portion. The proximal stump is sutured to form a pouch; the ogive of the circular stapler is placed inside the stump.
9. The bowel containing the ogive is reintroduced into the abdominal cavity; the abdominal incision is closed.
10. The circular stapler is introduced through the anus, connected to the ogive, and activated to form the end-to-end anastomosis.

The next step is to ascertain the integrity of the ureters and the anastomotic site. The latter is checked under laparoscopic control by injecting 120 cc of air into the rectum, which is submerged in irrigation fluid to ensure that there is no leakage. In addition, a dilute solution of methylene blue is introduced into the rectum to confirm the absence of leakage.

Endometriotic lesions in the urinary tract usually present cyclical urinary alterations as symptoms. The surgery is done aiming at minimal damage to the tissues. However, in cases with ureteral obstruction, a more aggressive approach is needed with extensive resection of the endometriosis, uretero-ureteral reanastomosis, and ureteral reimplantation. It is important to remember that in all cases of endometriosis in the bladder, a cystoscopic examination is necessary in order to observe the anatomy of the bladder.



**Fig. 5.10** Frozen pelvis in advanced deep infiltrating endometriosis

### Conclusion

Laparoscopic surgery plays a central role in the management of endometriosis. It is still the gold standard for diagnosis of the disease, excision of the lesions, reduction of the pain, and increasing fertility—all of which contribute to improving patient quality of life. Thus, some essential principles of laparoscopic treatment should be kept in mind, such as the principle of the “one-shot” surgery, which involves careful analysis of the pelvic cavity with excision and biopsy of all confirmed and suspected lesions. In addition, surgical planning must be aligned with each patient’s individual needs, especially concerning fertility preservation after surgery, which determines the surgeon’s choice of a complete or conservative approach.

## References

1. Burney RO, Giudice LC. Pathogenesis and pathophysiology of endometriosis. *Fertil Steril*. 2012;98:511–9.
2. Giudice LC. Clinical practice. Endometriosis. *N Engl J Med*. 2010;362:2389–98.
3. Huang H, Li C, Zarogoulidis P, Darwiche K, Machairiotis N, Yang L, et al. Endometriosis of the lung: report of a case and literature review. *Eur J Med Res*. 2013;18:3.
4. Arruda MS, Petta CA, Abrão MS, Benetti-Pinto CL. Time elapsed from onset of symptoms to diagnosis of endometriosis in a cohort study of Brazilian women. *Hum Reprod*. 2003;18:756–9.
5. Nnoaham KE, Hummelshoj L, Webster P, d'Hooghe T, de Cicco Nardone F, et al. World Endometriosis Research Foundation Global Study of Women's Health Consortium. Impact of endometriosis on quality of life and work productivity: a multicenter study across ten countries. *Fertil Steril*. 2011;96:366–73.
6. Revised American Society for Reproductive Medicine classification of endometriosis: 1996. *Fertil Steril*. 1997;67:817–21.
7. Chapron C, ABrao MS, Miller CE. Endometriosis classification need to be revised: A new one is arriving. *NewsScope*. 2012; 26:9.
8. Bellelis P, Dias Jr JA, Podgaec S, Gonzales M, Baracat EC, Abrão MS. Epidemiological and clinical aspects of pelvic endometriosis – a case series [in English and Portuguese]. *Rev Assoc Med Bras*. 2010;56:467–71.
9. Sampson JA. Metastatic or embolic endometriosis, due to the menstrual dissemination of endometrial tissue into the venous circulation. *Am J Pathol*. 1927;3:93–110.
10. Carvalho L, Podgaec S, Bellodi-Privato M, Falcone T, Abrão MS. Role of eutopic endometrium in pelvic endometriosis. *J Minim Invasive Gynecol*. 2011;18:419–27.
11. Nouri K, Ott J, Krupitz B, Huber JC, Wenzl R. Family incidence of endometriosis in first-, second-, and third-degree relatives: case–control study. *Reprod Biol Endocrinol*. 2010;8:85.
12. Abrão MS, Podgaec S, Pinotti JA, de Oliveira RM. Tumor markers in endometriosis. *Int J Gynaecol Obstet*. 1999;66:19–22.
13. Patrelli TS, Berretta R, Gizzo S, Pezzuto A, Franchi L, Lukanovic A, et al. CA 125 serum values in surgically treated endometriosis patients and its relationships with anatomic sites of endometriosis and pregnancy rate. *Fertil Steril*. 2011;95:393–6.
14. Abrão MS, Gonçalves MO, Dias Jr JA, Podgaec S, Chamie LP, Blasbalg R. Comparison between clinical examination, transvaginal sonography and magnetic resonance imaging for the diagnosis of deep endometriosis. *Hum Reprod*. 2007;22:3092–7.
15. Dell'oro M, Collinet P, Robin G, Rubod C. Multidisciplinary approach for deep endometriosis: interests and organization [article in French]. *Gynecol Obstet Fertil*. 2013;41:5864.
16. Triolo O, Laganà AS, Sturlese E. Chronic pelvic pain in endometriosis: an overview. *J Clin Med Res*. 2013;5:153–63.
17. Chapron C, Dubuisson JB, Fernandez B, Dousset B. Surgical treatment of endometriosis [article in French]. *Rev Prat*. 1999;49:276–8.
18. Bianchi PH, Pereira RM, Zanatta A, Alegretti JR, Motta EL, Serafini PC. Extensive excision of deep infiltrative endometriosis before in vitro fertilization significantly improves pregnancy rates. *J Minim Invasive Gynecol*. 2009;16:174–80. Erratum in *J Minim Invasive Gynecol*. 2009;16:663.
19. Paka C, Miller J, Nezhat C. Predictive factors and treatment of recurrence of endometriosis. *Minerva Ginecol*. 2013;65:105–11.
20. Chapron C, Fritel X, Dubuisson JB. Fertility after laparoscopic management of deep endometriosis infiltrating the uterosacral ligaments. *Hum Reprod*. 1999;14:329–32.
21. Practice Committee of American Society for Reproductive Medicine. Treatment of pelvic pain associated with endometriosis. *Fertil Steril*. 2008;90(5 Suppl):S260–9.
22. Khan J, Gill M, Clarke H. Onset of benign paroxysmal positional vertigo after total laparoscopic hysterectomy in the Trendelenburg position. *J Minim Invasive Gynecol*. 2012;19:798–800.
23. Vargas-Hernández VM. Endometriosis as a risk factor for ovarian cancer [in Spanish]. *Cir Cir*. 2013;81:163–8.
24. Donnez J, Squifflet J, Jadoul P, Lousse JC, Dolmans MM, Donnez O. Fertility preservation in women with ovarian endometriosis. *Front Biosci (Elite Ed)*. 2012;4:1654–62.
25. Somigliana E, Benaglia L, Vigano P, Candiani M, Vercellini P, Fedele L. Surgical measures for endometriosis-related infertility: a plea for research. *Placenta*. 2011;32 Suppl 3:S238–42.
26. Sugita A, Iwase A, Goto M, Nakahara T, Nakamura T, Kondo M, et al. One-year follow-up of serum antimüllerian hormone levels in patients with cystectomy: are different sequential changes due to different mechanisms causing damage to the ovarian reserve? *Fertil Steril*. 2013;100(2):516–22.e3.
27. Koninckx PR, Ussia A, Adamyan L, Wattiez A, Donnez J. Deep endometriosis: definition, diagnosis, and treatment. *Fertil Steril*. 2012;98:564–71.