We are IntechOpen, the world’s leading publisher of Open Access books
Built by scientists, for scientists

6,500
Open access books available

176,000
International authors and editors

190M
Downloads

154
Countries delivered to

TOP 1%
Our authors are among the most cited scientists

12.2%
Contributors from top 500 universities

WEB OF SCIENCE™
Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com
Urinary Tract Endometriosis

Aliasghar Yarmohamadi and Nasser Mogharabian
Ghaem Hospital, Mashhad University of Medical Sciences, Mashhad, Iran

1. Introduction

Endometriosis is defined as the presence of functional endometrial tissue in an ectopic site (outside of the uterus). Although endometriosis is usually confined to the ovaries, uterosacral ligaments, and cul-de-sac, it has been documented in almost every organ system in the body.

Endometriosis was first described by Russel in 1955. Endometriosis is a common disorder of the female reproductive organs and is the leading cause of chronic pelvic pain in women.

It is one of the most complex and least understood diseases in their field and, despite many theories, we still do not have a clear understanding of its causes.

There is no relationship of endometriosis to race or socioeconomic status but it has a strong familial link. If sister or mother of a woman has the condition, then she is approximately five to six times more likely to develop it.

Because ovarian function is necessary for the development and maintenance endometrial implants, endometriosis has been reported only in the reproductive ages and so, is normally not seen before age 15 or after menopause. Endometriosis is most common in women between the ages of 25 and 35. However, after menopause, there are two factors which may promote or maintain endometriosis. One is the use of estrogen replacement therapy and the other is the presence of high endogenous estrogen in obese patients.

There have been several case reports of histological endometriosis in men, all of them in the prostate. These have occurred all in men with cancer of the prostate who were undergoing high-dose estrogen therapy.

The prevalence of endometriosis in specific categories of patients has been reported (Table.1), but the prevalence in the general population is not definitely known because a majority of patients are asymptomatic. It is estimated that affect 10% to 20% of women of reproductive age, with a peak incidence in the mid-20s. However, in women with severe menstrual cramps, the incidence of endometriosis has been reported to be between 25 and 35 percent.

Although endometriosis is a benign condition but it may have an aggressive clinical behaviour. Many women with urinary tract endometriosis have few or no symptoms. Some present with infertility. Other manifestations include urgency, frequency, pain on passing urine, pain in the flank or the back region or recurrent urinary tract infections. Some women
have cyclic hematuria at times of menstruation. Some patients give a history of gynecologic surgeries such as hysterectomy many years ago.

| Women of reproductive age (overall) | 10 to 20 percent |
|-------------------------------------|------------------|
| Women with a history of major surgery for any gynecologic indication | 1 percent |
| Women undergoing tubal sterilization | 1 to 7 percent |
| Following laparoscopy to determine the cause of pelvic pain | 12 to 32 percent |
| Women undergoing laparoscopy for infertility | 9 to 50 percent |
| Women undergoing laparoscopy without infertility | 6.7 percent |
| Teenagers undergoing laparoscopy for evaluation of chronic pelvic pain or dysmenorrhea | 50 percent |

Table 1. The prevalence of endometriosis in women groups of reproductive age.

It can be associated with many distressing and debilitating symptoms may be asymptomatic, and incidentally discovered at laparoscopy or exploratory surgery. Usually, endometriosis is confined to the pelvic and lower abdominal cavity; however, it has occasionally been reported to be in other areas. Endometriotic lesions of the urinary tract are present in 1 to 4 percent of women with endometriosis and often coexists with disease at other sites of the body.

Although ureteral and bladder endometriosis both occur in the urinary tract, they do not frequently coexist and their clinical presentation and management are different. Bladder endometriosis often mimicks recurrent cystitis, but rarely results in severe sequelae. Ureteral endometriosis is often asymptomatic, but can lead to silent loss of renal function. Renal and urethral involvements are rare and only as case reports.

| Overall | unclear but may occur in 1 - 4% of all cases of endometriosis |
|---------|---------------------------------------------------------------|
| Bladder | 70 – 80 percent of all urinary tract involvements              |
| Ureter  | 15 – 20 percent of all urinary tract involvements              |
| Kidney  | 4 percent of all urinary tract involvements                    |
| Urethra | 2 percent of all urinary tract involvements                    |

Table 2. The prevalence of endometriosis in urinary tract.

The optimal way to diagnose endometriosis is by direct visualization and biopsy of the implant(s) anywhere through the body.

2. Ethiology and pathogenesis

The classic definition of endometriosis is as the presence of endometrial glands and stroma at extraterine sites. These ectopic endometrial implants are usually located in the pelvis, but can occur nearly anywhere in the body. It can be associated with many distressing and debilitating symptoms or may be asymptomatic, and incidentally discovered at laparoscopy or exploratory surgery.
The pathogenesis of endometriosis has not been definitively established but predominant hypotheses are as follows:

- **The implantation theory:** This theory proposes that endometrial cells are either transported during menstruation through the fallopian tubes and implanted on pelvic structures (transstubal regurgitation or retrograde menstruation) or are transplanted to surgical scars (episiotomy, laparotomy) as a result of surgery or delivery.

- **Lymphatics and blood vessels dissemination:** This theory is specially useful for explanation of endometriosis at locations outside the pelvis (extraperitoneal disease).

- **Coelomic metaplasia:** This theory proposes that the peritoneal cavity contains undifferentiated cells capable of differentiating into endometrial tissue. It is said that repeated inflammation may induce metaplasia of mesothelial cells to the endometrial epithelium.

Genetic factors probably influence an individual's susceptibility to endometriosis. The possibility of a familial tendency for endometriosis has been recognized for several decades. If a woman has endometriosis, a first-degree relative has a 7 percent likelihood of developing the disorder as compared with 1 percent in unrelated persons. Concordance in twins has also been observed.

There is evidence for altered humoral and cell-mediated immunity in the pathogenesis of endometriosis such as Deficient cellular immunity, improper Natural killer cell activity and increased concentration of leukocytes and macrophages in the peritoneal cavity and ectopic endometrium. These variations in immune system may result in an inability to recognize the presence of endometrial tissue in abnormal locations, decreased cytotoxicity to autologous ectopic endometrium and finally, secretion of cytokines and growth factors by leukocytes and macrophages into the peritoneal fluid of women with endometriosis.

One hypothesis is that secretion of various cytokines by inflammatory cells into the peritoneal cavity leads to proliferation of implants and recruitment of capillaries. Oxidative stress may be another component of the inflammatory reaction. Thus, the immune system may play a role in determining who will develop endometriosis, as well as the extent and clinical manifestation of the disease.

It is said that these women had higher rates of autoimmune inflammatory diseases, hypothyroidism, fibromyalgia, chronic fatigue syndrome, allergies and asthma, compared with the general female population so, it could provide support for the theory of altered immune system in women with endometriosis.

Vesical endometriosis is said that may be due to bladder adenomyosis or an extension of adenomyosis from the uterus into the bladder or because of imperfect closure of the uterus during a cesarean delivery.

The risk of endometriosis developing into a cancerous lesion is very low (1 - 2.5%) and the same as normal endometrium and ovaries.

Endometriosis is a common, benign, chronic, estrogen-dependent disorder with a relapsing/remitting nature. The endometrial tissue acts just like the normal ones in the uterus, responding to cyclical hormone levels, growing and bleeding at certain times of the cycle. If the tissue is in the ovaries, then bleeding of it results in accumulation of blood,
named chocolate cysts. During menstruation, the ectopic tissue bleeds, causing the surrounding tissues to become inflamed. This inflammation causes fibrosis, leading to adhesions that produce pain and other complications such as infertility. Inflammation that happens at the site of the endometrial tissue results in and adhesions in the abdomen and pelvis. These can lead to a frozen pelvis and other complications such as infertility. It can be associated with many distressing and debilitating symptoms, such as pelvic pain, severe dysmenorrhea, dyspareunia and infertility, or it may be asymptomatic, and incidentally discovered at laparoscopy or exploratory surgeries.

Active endometriosis usually occurs between ages 30 and 40, but may be seen before age 20. Severe symptoms of endometriosis may have an abrupt onset or develop over many years. This disorder usually becomes progressively severe during the menstrual years but after menopause, it tends to subside.

The most common sites of endometriosis are respectively the ovaries, anterior and posterior cul-de-sac, posterior broad ligaments, uterosacral ligaments, uterus, fallopian tubes, sigmoid colon and appendix, and round ligaments. Other less commonly involved regions include the vagina, cervix, rectovaginal septum, ecum, ileum, inguinal canals, abdominal or perineal scars, urinary bladder, ureters, and umbilicus. Rarely, endometriosis has been reported in the breast, pancreas, liver, gallbladder, kidney, urethra, extremities, vertebrea, bone, peripheral nerves, lung, diaphragm, central nervous system, and even in the prostate. Endometriosis is multifocal in most patients. The disease is staged according to site and severity of involvements.

Diagnosis of urinary tract endometriosis requires a careful history and thorough physical examination. High index of suspicion to all symptomatic women with a history of caesarian delivery or other gynaecological surgery gives a clue to the diagnosis.

Ultrasonography is the initial step of investigation to detect the vesical endoluminal mass or upper urinary tract dilatation. On ultrasound examination, the lesions usually appears as hypoechoic, vascular, and solid masses, although cystic changes can be present. The lesions have no definite margins and may appear to infiltrate adjacent tissues. IVU is still very much useful to detect the integrity of the upper tract and ureter. MRI is better than computed tomography for identifying hemorrhage and soft tissue planes. Fine needle aspiration will yield chocolate-colored fluid.

Cystoscopy and laparoscopy together with biopsy are fundamental to the assessment of urinary tract endometriosis specially before operation. Wide local excision is performed either for confirmation diagnosis or as treatment.

Optimal management requires a team of specialists including gynaecologists, colorectal surgeons and urologists, working together to thoroughly assess the risks and benefits of treatments and to determine the optimal care.

Treatment varies according to the severity and site of involvement of each case. Hormonal therapy with danazole does have a definite roll in regressing the lesion but in cases with urinary tract involvement, surgical treatment is a better option because the condition may lead to kidney loss up to 25%.

Aggressive surgical removal of ectopic tissues, relief of urinary obstruction and castration with or without hysterectomy is the recommended surgical treatment for urinary tract
endometriosis. In the younger patients and in order to preserve fertility, endometriosis should be resected but with preservation of ovarian function and strict periodic surveillance of urinary tract.

3. Upper tract involvement

3.1 Renal endometriosis
Since renal endometriosis is rarely encountered, it is briefly mentioned in the clinical guidelines and literatures.

3.2 Ureteral endometriosis
Endometriosis of the urinary tract is predominantly found in the bladder, accounting for 70% to 80% of the cases. The ureter may be involved in 15% to 20% of the urinary tract cases. Bilateral disease has been reported in up to 23% of cases. The left side is more often affected, which may be because the sigmoid colon prevents the regurgitated endometrial cells to be cleared by the peritoneal fluid on the left side.

Ureteral involvement may be either intrinsic or extrinsic. If endometrial glands and stroma are within the lamina propria, tunica muscularis, or ureteral lumen it is said Intrinsic endometriosis and if they are localized within periureteral tissue extrinsic endometriosis ensues. Eighty percent of ureteral endometriosis is extrinsic and most commonly involves the distal ureter. Differentiation between these two forms of ureteral endometriosis has histologic and pathogenetic importance, but has little impact on clinical management since the precise location of the lesion cannot be determined preoperatively. Moreover, both intrinsic and extrinsic forms of the disease may result in ureteral stenosis.

Silent loss of renal function has been reported in 25% to 43% of patients with ureteral endometriosis, which may result in total loss of function of the affected kidney. Historically, up to one third of kidneys affected by ureteral endometriosis were lost. So, it has been recommended to take image of the upper urinary tract in all patients with pelvic endometriosis with ultrasonography or IVU.

Gynecologic laparoscopy for treatment of endometriosis is responsible for a large percentage of ureteral injuries. The reasons for this may be as follows: (1) endometrioma can involve the ureter either extrinsically or intrinsically; (2) adhesions from endometriosis makes ureteral visualization difficult; and (3) the disease can deviate the ureters medially resulting in abnormal anatomy.

In addition, the most commonly affected portions of the ureter are the distal third, followed by the middle third. Involvement of the proximal ureter is rare. Thus, the most frequent sites of ureteral endometriosis are below the level of tubal efflux. The lesions of the distal ureter usually coexist with posterior pouch endometriosis, as the lesions of the middle third of the ureter may be together with involvement of the ovary.

3.2.1 Clinical manifestations
Classic symptoms and signs of urinary tract endometriosis include cyclical flank pain, dysuria, urgency, urinary tract infection, and hematuria. As a rule, we can say that intrinsic
endometriosis is more symptomatic than extrinsic disease. Notably, a significant portion of patients with ureteral endometriosis do not have genitourinary symptoms and as a consequence, ureteral endometriosis can lead to silent loss of renal function.

Ureteral endometriosis presents with colicky flank pain in approximately 25 percent of patients and gross hematuria in 15 percent, while up to 50 percent of patients are asymptomatic and is generally discovered at the time of laparotomy or laparoscopy for evaluation of pelvic pain or other indications.

3.2.2 Diagnosis

Ureteral endometriosis should be included in the differential diagnosis of obstructive ureteral lesions in women, particularly those involving the lower third of the left ureter. Because a large percentage of ureteral endometriosis can result in loss of renal function due to asymptomatic obstruction, all patients with pelvic endometriosis should undergo the upper urinary tract imaging. Initial imaging may be ultrasonography as a noninvasive test to look for hydroureteronephrosis. Although, IVU may be a better test in high suspicious cases. Intrinsic disease appears in IVU as ureteral filling defects, whereas extrinsic disease causes smooth strictures. The exact location and volume of the disease can be defined through retrograde ureteropyelography, CT or MRI, which can be valuable for planning treatment.

The diagnosis of ureteral endometriosis requires a high index of suspicion and is aided by clinicians’ awareness of the condition. The diagnosis of ureteral endometriosis is suggested by the finding of hydronephrosis in a patient with known or suspected endometriosis, particularly if symptoms consistent with ureteral involvement are present but, definite diagnosis can be reached through direct visualization and biopsy of implants. Histologic confirmation is the gold standard for diagnosis of endometriosis.

The differential diagnosis of ureteral endometriosis includes any conditions that result in hydronephrosis such as stones and malignancies.

3.2.3 Management

Treatment has several goals including preservation of renal function, management of the original disease process, maintenance of the patient’s fertility, and relief of the patient’s symptoms.

Medical treatment does not treat the fibrotic component of endometriotic lesions, which is largely responsible for ureteral obstruction so, medical therapy is usually not effective at relieving ureteral obstruction and treatment of ureteral endometriosis with hydronephrosis is surgical.

Patients with mild or intermittent hydronephrosis can be treated initially with a combination of medical therapy and insertion of a ureteral stent. In such cases, close monitoring of renal function is required.

3.2.3.1 Medical treatment

While medical therapy is effective for pain relief, symptoms often recur once treatment is completed. If renal function is normal and there is minimal to mild hydronephrosis with no
functional obstruction as determined by radionuclide renal scanning, hormone therapy may be prescribed.

Ureteral endometriosis is rare, and there are few studies of medical therapy. Ovarian hormonal ablation with gonadotropin-releasing hormone agonists has been utilized with success in some series. Hormonal therapy is not as effective for patients with extensive endometriosis. The aromatase inhibitors such as anastrazole and danazol was unsuccessful in relieving ureteral obstruction in few case reports.

### 3.2.3.2 Surgeries

The goals of surgical treatment of ureteral endometriosis are to remove the endometriotic lesion(s) and relieve ureteral stricture or kinking. Surgery is indicated if fertility is a major goal, where symptoms fail to respond to medical therapies, or where ureteric obstruction has been confirmed. Untreated ureteric obstruction may lead to irreversible kidney damage.

Surgical intervention is the treatment of choice for most patients with significant hydroureteronephrosis and periureteral disease. Ureterolysis may correct ureteral obstruction in those with extrinsic disease. If laparoscopic ureterolysis is undertaken, a transperitoneal approach is preferable in that it allows a superior assessment of endometrial implants on the peritoneum.

In the case of intrinsic disease, removal of lesions may be difficult because endometriotic lesions infiltrate the ureteral wall and there may be no apparent margin of them to be resected. So, when intrinsic disease is present or in case of ureterolysis failure, distal ureterectomy with reimplantation is preferred.

As a matter of fact, all lesions adjacent to the ureters to prevent future stenosis and renal damage, since the progression of endometriosis is unpredictable. Thus any lesion found incidentally during surgery proximal to ureters should be removed.

Ureterolysis is performed by most surgeons laparoscopically. A laparoscopic approach may be offered to patients with ureteral stricture disease. Although, ureteroneocystostomy may require laparotomy.

As the first step of the procedure ureterolysis is done. Since the ureter and peritoneum are almost never affected at the level of the pelvic brim, the peritoneum is opened upon the ureter at this site and the ureter is dissected from adjacent tissues to the level of the cardinal ligament, sharply or bluntly.

If endometriosis invades the ureteral adventitia it may be necessary to cut the adventitial sheath without any manipulation of muscular layer. Any ureteral perforation can be repaired with two interrupted 4-0 polydioxanone sutures.

After ureterolysis is complete, the decision is made regarding whether further intervention is required, based upon visual inspection of the ureter. Then if required, ureteral stent or resection of a diseased segment of ureter must be done to prevent future stenosis and obstruction.

Since most lesions are located in the distal ureter, ureteral resection is usually combined with ureteroneocystostomy. The involved segment of ureter is excised and reimplantation...
into the bladder is performed. When the endometriotic lesion is in the middle or upper third of the ureter, end ureteroureterostomy should be done after resection of lesions. Whether to proceed with resection of an endometriotic segment of the ureter or aggressive ureterolysis and stenting is a subjective decision based upon intraoperative visual with no definite criteria to predict normal postoperative ureteral function. In presence of stenosis and significant hydronephrosis, most surgeons have found that the risk of recurrence is lower after ureteral resection and ureteroneocystostomy rather than aggressive ureterolysis and stent insertion. In the case of surface ovarian endometriotic lesions or an endometrioma ipsilateral oophorectomy should be performed as needed. Bilateral oophorectomy is performed in some women to prevent recurrence of endometriosis.

In the cases incidentally discovered at time of laparoscopy or laparotomy, if ureteral adhesions are present, ureterolysis should be performed. Complications of these surgeries include general complications the same as other abdominal laparoscopy or laparotomy and specific complications to this procedure such as ureteral fistula.

The rates of recurrence for each type of surgical procedures are included in Table 3. Complications are few and most common of them is ureteral fistula. Ureterolysis alone is associated with higher rates of both recurrence and complications compared with ureteroneocystostomy and Ureteroureteral anastomosis.

| Procedure                          | Recurrence Rate |
|-----------------------------------|-----------------|
| Ureteroneocystostomy              | 3 percent       |
| Ureterolysis alone                | 8 percent       |
| Ureteroureteral anastomosis       | 11 percent      |

Table 3. The rates of recurrence for each type of surgical procedures for ureteral endometriosis.

4. Lower tract involvement

4.1 Urethral endometriosis

Since urethral endometriosis is rarely encountered the clinical guidelines and literature are brief. Endometriosis have been described within urethral diverticula. urethral endometriosis do not involve the urethral meatus.

4.2 Bladder endometriosis

Bladder endometriosis is defined as the presence of endometrial glands and stroma at detrusor muscle.

4.2.1 Clinical manifestations

Bladder endometriosis causes nonspecific urinary symptoms, including urinary frequency, urgency, dysuria, or urinary retention. Occurrence of these symptoms during menses is suggestive. Cyclic hematuria is uncommon but characteristic. The ureteral openings are usually not involved by the vesical lesions so, hydronephrosis is rare.
Some women with bladder endometriosis are asymptomatic and present with an incidental finding of a bladder nodule on pelvic imaging or as a result of pelvic surgery. Some patients are asymptomatic for the first few years and will only realize that they have the disease when it is already in its serious stage, manifesting more severe symptoms.

The most common complaint of women that have bladder endometriosis is pain in the abdominal or pelvic area. The degree of pain can be mild to severe or acute to subacute. Usually, this pain will be more intense during monthly period. Many women suffer from endometriosis silently because they feel that the pain is just the normal pain of premenstrual syndrome. However, if the premenstrual pain in a woman hampers her normal activities she is suspected to has endometriosis.

Women with bladder endometriosis also experience various urinary problems. It is common to feel a burning sensation during voiding. Since the cause of your urinary problems is endometrium cells in the bladder wall, antibiotics will not cure or alleviate the symptoms.

### 4.2.2 Diagnosis

The gold standard for diagnosis of bladder endometriosis is biopsy and histologic confirmation of visualized lesions during cystoscopy or laparoscopy.

Symptoms compatible with bladder endometriosis, specially together with known endometriosis at other sites or characteristic symptoms such as pelvic pain, dysmenorrhea, dyspareunia and infertility, and finding of a bladder nodule on ultrasound suggest vesical endometriosis and further evaluation is needed.

The differential diagnosis of bladder endometriosis includes urinary tract infection, interstitial cystitis, and a bladder stone or neoplasm. So, proper tests and examinations must be done based on patient’s clinical presentation to rule out these conditions. In more than 70% of cases the presenting symptoms of bladder endometriosis are identical to those of interstitial cystitis. Therefore, endometriosis should always be considered in the patient referred for frequency, urgency and pain with no documented infection.

Pelvic and renal sonography may show a bladder nodule or hydronephrosis, although, endometriosis is often not visualized on imaging studies. Endometriosis itself and its surgical treatments are capable of VVF formation. Nevertheless, endometriosis of the bladder must be differentiated from VVF because both of them may present with cyclic hematuria.

In the case of a bladder nodule, cystoscopy and biopsy is performed to rule out malignancy and confirm diagnosis. The distance of the lesion from the ureteral openings is important since, removal of the lesions adjacent to ureteral orifices may requires ureteral surgery, as well. Magnetic resonance imaging may be useful to detect Lesions that are not visible with cystoscopy. If there is hydronephrosis, radiologic evaluation of the same ureter is required.

### 4.2.3 Management

Bladder endometriosis, if left unmanaged, may lead to more severe urinary problems in the future such as urinary obstruction or incontinence. However, treatment of vesical endometriosis is indicated only if there is any symptoms or resulted in hydronephrosis. First
line treatment is medical therapy, since this approach avoids the risk of surgical complications. Medical therapy must be continued until menopause is not effective in all cases. Surgery must be reserved for cases in whom medical therapy have failed or is contraindicated, who wish to avoid chronic medical treatment, or who have hydronephrosis.

4.2.3.1 Medical treatment

Hormonal therapy is reasonable and effective management for bladder endometriosis and because it preserves fertility, is especially attractive to younger women. While medical therapy is effective for pain relief, symptoms often recur once treatment is completed.

Medical therapy of symptomatic bladder endometriosis is the same as for other sites of the disease. Oral contraceptives, progestins, and gonadotropin releasing hormone agonists all have been reported to improve symptoms and regress the lesions.

A tolerable and safe medication should be chosen. Oral contraceptive on the usual cyclic regimen may be efficient. Although, If perimenstrual symptoms do not resolve with a cyclic regimen, it should be substituted by a continuous regimen. Low dose progestin is also effective and safe.

4.2.3.2 Surgeries

Definitive treatment of bladder endometriosis is surgical removal of the lesions. Surgery is indicated if fertility is a major goal, where symptoms fail to respond to medical therapies, or where ureteric obstruction has been confirmed. Untreated ureteric obstruction may lead to irreversible kidney damage.

If the lesion deeply infiltrates the bladder wall then, a full thickness resection and subsequent repair of the bladder wall is needed, which can be done laparoscopically. Although, in the case of partial thickness involvements one can only resect the lesions without opening the bladder wall.

In the face of the complicated conditions there may be the need for more advanced procedures which should be performed via laparotomy and only by more experient surgeons (table.4).

|   |   |
|---|---|
| 1 | Any possibility for endometriosis of the ureter such as hydronephrosis. |
| 2 | The lesion is less than 2 cm away from the inter-ureteric ridge. |
| 3 | Another bladder lesion has been recently resected. |

Table 4. The complicated bladder endometrioses.

If the inferior border of the endometriotic lesion is less than 2 cm away from the interureteric ridge, then ureteral catheters should be inserted at the beginning of the procedure.

Removal of endometriotic nodules at the bladder dome may not require any dissection but, in the face of nodules involving the posterior or inferior aspects, the bladder must be dissected from the uterus just enough below the inferior margin of the nodule in order to achieve complete resection. The lesions is then excised with cold scissors or electrosurgery and the bladder is closed with two layers of transverse sutures.
For lesions at the vesical base, where the bladder is apposed to the uterus, resection of a 0.5 to 1 cm deep portion of the myometrium contiguous with the endometriotic nodule may prevent recurrence.

At the end of the procedure, the bladder is filled to confirm that the closure is watertight and the bladder catheter is left in place for 7 to 10 days to prevent fistula formation.

The removal of bladder endometriosis is contraindicated in pregnant women as well during cesarean section, because high endometrial blood flow can result in massive hemorrhagia. General complications are the same as other laparoscopic or cystoscopic procedures. Special complications include vesicovaginal hematoma and vesicovaginal fistula. Ureter–fallopian tube fistula has also been reported as a consequence of laparoscopic fulguration of endometriosis.

The overall outcome of surgical treatment for bladder endometriosis has been good and complications or need for reoperations had been low.

5. Summery

- Endometriosis is defined as the presence of functional endometrial tissue in an ectopic site (outside of the uterus). Endometriosis is a common, benign, chronic, estrogen-dependent disorder with a relapsing/remitting nature.
- Although endometriosis is usually confined to the ovaries, uterosacral ligaments, and cul-de-sac, it has been documented in almost every organ system in the body.
- Because ovarian function is necessary for the development and maintenance endometrial implants, endometriosis has been reported only in the reproductive age and so, is normally not seen before age 15 or after menopause. Endometriosis is most common in women between the ages of 25 and 35.
- The prevalence of endometriosis in specific categories of patients has been reported (Table 1), but the prevalence in the general population is not definitely known because a majority of patients are asymptomatic. It is estimated that affect 10% to 20% of women of reproductive age.
- Endometriosis can be associated with many distressing and debilitating symptoms may be asymptomatic, and incidentally discovered at laparoscopy or exploratory surgery.
- Endometriotic lesions of the urinary tract are present in 1 to 2 percent of women with endometriosis and often coexists with disease at other sites of the body. Actually, endometriosis is multifocal in most patients.
- The pathogenesis of endometriosis has not been definitively established but predominant hypotheses are the implantation theory, lymphatics and blood vessels dissemination, and coelomic metaplasia. Genetic factors and altered humoral and cell-mediated immunity may also play a role in endometriosis.
- The endometrial tissue in the endometriotic lesions acts just like the normal ones in the uterus, responding to cyclical hormone levels, growing and bleeding at certain times of the cycle, causing the surrounding tissues to become inflamed. This inflammation causes fibrosis, leading to adhesions that produce pain and other complications such as infertility.
- Since renal and urethral endometriosis is rarely encountered, they are briefly mentioned in the clinical guidelines and literatures.
Endometriosis of the urinary tract is predominantly found in the bladder, accounting for 70% to 80% of the cases. The ureter may be involved in 15% to 20% of the urinary tract cases and the left side is more often affected. Bilateral ureteral disease has been reported in up to 23% of cases.

Ureteral involvement may be either intrinsic or extrinsic. Eighty percent of ureteral endometriosis is extrinsic and most commonly involves the distal ureter. Differentiation between these two forms of ureteral endometriosis has histologic and pathogenetic importance, but has little impact on clinical management.

Because a large percentage of ureteral endometriosis can result in silent loss of renal function due to asymptomatic obstruction, all patients with pelvic endometriosis should undergo the upper urinary tract imaging.

Classic symptoms and signs of urinary tract endometriosis include cyclical flank pain, dysuria, urgency, urinary tract infection, and hematuria. As a rule, we can say that intrinsic endometriosis is more symptomatic than extrinsic disease.

The exact location and volume of the ureteral disease can be defined through retrograde ureteropyelography, CT or MRI, which can be valuable for planning treatment.

The diagnosis of ureteral endometriosis requires a high index of suspicion and is aided by clinicians’ awareness of the condition. Definite diagnosis can be reached through direct visualization and biopsy of implants.

Treatment of ureteral endometriosis has several goals including preservation of renal function, management of the main disease process, maintenance of the patient’s fertility, and relief of the patient’s symptoms. If renal function is normal and there is minimal to mild hydronephrosis with no functional obstruction as determined by radionuclide renal scanning, hormone therapy may be prescribed otherwise, surgery is indicated.

Since most lesions are located in the distal ureter, ureteral resection is usually combined with ureteroneocystostomy but, when the endometriotic lesion is in the middle or upper third of the ureter, end ureteroureterostomy should be done after resection of lesions.

Bladder endometriosis is defined as the presence of endometrial glands and stroma through detrusor muscle. The ureteral openings are usually not involved by the vesical lesions so, hydronephrosis is rare.

Bladder endometriosis causes nonspecific urinary symptoms, including urinary frequency, urgency, dysuria, or urinary retention. Occurrence of these symptoms during menses is suggestive. Cyclic hematuria is uncommon but characteristic.

Treatment of vesical endometriosis is indicated only if there is any symptoms or resulted in hydronephrosis. First line treatment is medical therapy. Surgery must be reserved for cases in whom medical therapy have failed or is contraindicated, who wish to avoid chronic medical treatment, or who have hydronephrosis.

If the inferior border of the endometriotic lesion is less than 2 cm away from the interureteric ridge, then ureteral catheters should be inserted at the beginning of the procedure.

Removal of endometriotic nodules at the bladder dome may not require any dissection but, in the face of nodules involving the posterior or inferior aspects, the bladder must be dissected from the uterus just enough below the inferior margin of the nodule in order to achieve complete resection.

For lesions at the vesical base, where the bladder is apposed to the uterus, resection of a 0.5 to 1 cm deep portion of the myometrium contiguous with the endometriotic nodule may prevent recurrence.
6. References

Abbott J, Hawe J, Hunter D, et al. Laparoscopic excision of endometriosis: a randomized, placebo-controlled trial. Fertil Steril 2004; 82:878.

Abeshouse BS, Abeshouse G. Endometriosis of the urinary tract: a review of the literature and a report of four cases of vesical endometriosis. J Int Coll Surg 1960; 34:43.

Abeshouse BS, Abeshouse G. Endometriosis of the urinary tract: a review of the literature and a report of four cases of vesical endometriosis. J Int Coll Surg 1960;34:43-63.

Abrao MS, Dias JA Jr, Bellelis P, et al. Endometriosis of the ureter and bladder are not associated diseases. Fertil Steril 2009; 91:1662.

Abrao MS, Gonçalves MO, Dias JA Jr, et al. Comparison between clinical examination, transvaginal sonography and magnetic resonance imaging for the diagnosis of deep endometriosis. Hum Reprod 2007; 22:3092.

Ahn M, Loughlin KR. Psoas hitch ureteral reimplantation in adults--analysis of a modified technique and timing of repair. Urology 2001; 58:184.

Al-Khawaja M, Tan PH, MacLennan GT, et al. Ureteral endometriosis: clinicopathological and immunohistochemical study of 7 cases. Hum Pathol 2008; 39:954.

Allen WM, Masters WH. Traumatic laceration of uterine support; the clinical syndrome and the operative treatment. Am J Obstet Gynecol 1955; 70:500.

Anaf V, Simon P, El Nakadi I, et al. Relationship between endometriotic foci and nerves in rectovaginal endometriotic nodules. Hum Reprod 2000; 15:1744.

Antonelli A, Simeone C, Frego E, et al. Surgical treatment of ureteral obstruction from endometriosis: our experience with thirteen cases. Int Urogynecol J Pelvic Floor Dysfunct 2004; 15:407.

Antonelli A, Simeone C, Zani D, et al. Clinical aspects and surgical treatment of urinary tract endometriosis: our experience with 31 cases. Eur Urol 2006; 49:1093.

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.

Bedaiwy MA, Falcone T, Mascha EJ, Casper RF. Genetic polymorphism in the fibrinolytic system and endometriosis. Obstet Gynecol 2006; 108:162.

Bennett GL, Slywotzky CM, Cantera M, Hecht EM. Unusual manifestations and complications of endometriosis--spectrum of imaging findings: pictorial review. AJR Am J Roentgenol 2010; 194:WS534.

Berlanda N, Vercellini P, Carmignani L, et al. Ureteral and vesical endometriosis. Two different clinical entities sharing the same pathogenesis. Obstet Gynecol Surv 2009; 64:830.
Berlanda N, Vercellini P, Carmignani L, et al. Ureteral and vesical endometriosis. Two different clinical entities sharing the same pathogenesis. Obstet Gynecol Surv 2009; 64:830.

Bese T, Simsek Y, Bese N, et al. Extensive pelvic endometriosis with malignant change in tamoxifen-treated postmenopausal women. Int J Gynecol Cancer 2003; 13:376.

Bischoff FZ, Simpson JL. Heritability and molecular genetic studies of endometriosis. Hum Reprod Update 2000; 6:37.

Blanco RG, Parithivel VS, Shah AK, et al. Abdominal wall endometriomas. Am J Surg 2003; 185:596.

Bohrer J, Chen CC, Falcone T. Persistent bilateral ureteral obstruction secondary to endometriosis despite treatment with an aromatase inhibitor. Fertil Steril 2008; 90:2004.e7.

Bordin L, Fiore C, Donà G, et al. Evaluation of erythrocyte band 3 phosphotyrosine level, glutathione content, CA-125, and human epididymal secretory protein E4 as combined parameters in endometriosis. Fertil Steril 2010; 94:1616.

Bosev D, Nicoll LM, Bhagan L, et al. Laparoscopic management of ureteral endometriosis: the Stanford University hospital experience with 96 consecutive cases. J Urol 2009; 182:2748.

Bricou A, Batt RE, Chapron C. Peritoneal fluid flow influences anatomical distribution of endometriotic lesions: why Sampson seems to be right. Eur J Obstet Gynecol Reprod Biol 2008; 138:127.

Brinton LA, Lamb EJ, Moghissi KS, et al. Ovarian cancer risk associated with varying causes of infertility. Fertil Steril 2004; 82:405.

Brosens IA, Fusi L, Brosens JJ. Endometriosis is a risk factor for spontaneous hemoperitoneum during pregnancy. Fertil Steril 2009; 92:1243.

Bulletti C, Montini A, Setti PL, et al. Vaginal parturition decreases recurrence of endometriosis. Fertil Steril 2010; 94:850.

Bulun SE. Endometriosis. N Engl J Med 2009; 360:268.

Busacca M, Chiaffarino F, Candiani M, et al. Determinants of long-term clinically detected recurrence rates of deep, ovarian, and pelvic endometriosis. Am J Obstet Gynecol 2006; 195:426.

Busard MP, Mijatovic V, van Kuijk C, et al. Magnetic resonance imaging in the evaluation of (deep infiltrating) endometriosis: the value of diffusion-weighted imaging. J Magn Reson Imaging 2010; 32:1003.

Campbell IG, Thomas EJ. Endometriosis: candidate genes. Hum Reprod Update 2001; 7:15.

Carmignani L, Ronchetti A, Amicarelli F, et al. Bladder psoas hitch in hydronephrosis due to pelvic endometriosis: outcome of urodynamic parameters. Fertil Steril 2009; 92:35.

Carmignani L, Vercellini P, Spinelli M, et al. Pelvic endometriosis and hydrourereteronephrosis. Fertil Steril 2010; 93:1741.

Carmignani L, Vercellini P, Spinelli M, et al. Pelvic endometriosis and hydrourereteronephrosis. Fertil Steril 2010; 93:1741.

Chapron C. Dubisson JB. Laparoscopic management of bladder endometriosis. Acta Obstet Gynecol Scand 1999;78:887-90.

Chapron C, Barakat H, Fritel X, et al. Presurgical diagnosis of posterior deep infiltrating endometriosis based on a standardized questionnaire. Hum Reprod 2005; 20:507.

Chapron C, Bourret A, Chopin N, et al. Surgery for bladder endometriosis: long-term results and concomitant management of associated posterior deep lesions. Hum Reprod 2010; 25:884.
Chapron C, Chiodo I, Leconte M, et al. Severe ureteral endometriosis: the intrinsic type is not so rare after complete surgical exeresis of deep endometriotic lesions. Fertil Steril 2010; 93:2115.

Chapron C, Chopin N, Borghese B, et al. Deeply infiltrating endometriosis: pathogenetic implications of the anatomical distribution. Hum Reprod 2006; 21:1839.

Chapron C, Dubisson JB, Faucq J, Da-Costa VM. Laparoscopy and bladder endometriosis. Gynecol Obstet Fertil 2000;28:232-7.

Chapron C, Dubuisson JB. Laparoscopic management of bladder endometriosis. Acta Obstet Gynecol Scand 1999; 78:887.

Chapron C, Faucq J, Dubuisson JB, et al. Deeply infiltrating endometriosis: relation between severity of dysmenorrhea and extent of disease. Hum Reprod 2003; 18:760.

Chatman DL, Ward AB. Endometriosis in adolescents. J Reprod Med 1982; 27:156.

Cheng YM, Wang ST, Chou CY. Serum CA-125 in preoperative patients at high risk for endometriosis. Obstet Gynecol 2002; 99:375.

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.

Comiter CV. Endometriosis of the urinary tract. Urol Clin North Am 2002; 29:625.

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, et al. Bowel resection for deep endometriosis: a systematic review. BJOG 2011; 118:285.

Deligdisch L, Péanault-Llorca F, Schlosshauer P, et al. Stage I ovarian carcinoma: different clinical pathologic patterns. Fertil Steril 2007; 88:906.

D’Hooghe TM, Debrock S, Hill JA, Meuleman C. Endometriosis and subfertility: is the relationship resolved? Semin Reprod Med 2003; 21:243.

Dmowski WP, Gebel HM, Braun DP. The role of cell-mediated immunity in pathogenesis of endometriosis. Acta Obstet Gynecol Scand Suppl 1994; 159:7.

Donnez J, Nisolle M, Squifflet J. Ureteral endometriosis: a complication of rectovaginal endometriotic (adenomyotic) nodules. Fertil Steril 2002; 77:32.

Donnez J, Spada F, Squifflet J, Nisolle M. Bladder endometriosis must be considered as bladder adenomyosis. Fertil Steril 2000; 74:1175.

Dwivedi AJ, Agrawal SN, Silva YJ. Abdominal wall endometriomas. Dig Dis Sci 2002; 47:456.

Efstathiou JA, Sampson DA, Levine Z, et al. Nonsteroidal antiinflammatory drugs differentially suppress endometriosis in a murine model. Fertil Steril 2005; 83:171.

Engemise S, Gordon C, Konje JC. Endometriosis. BMJ 2010; 340:c2168.

Erzen M, Rakar S, Klarcek B, et al. Endometriosis-associated ovarian carcinoma (EAOC): an entity distinct from other ovarian carcinomas as suggested by a nested case-control study. Gynecol Oncol 2001; 83:100.

Eyster KM, Klinkev O, Kennedy V, Hansen KA. Whole genome deoxyribonucleic acid microarray analysis of gene expression in ectopic versus eutopic endometrium. Fertil Steril 2007; 88:1505.

Faccioli N, Manfredi R, Mainardi P, et al. Barium enema evaluation of colonic involvement in endometriosis. AJR Am J Roentgenol 2008; 190:1050.
Faucheron JL, Pasquier D, Voirin D. Endometriosis of the vermiform appendix as an exceptional cause of acute perforated appendicitis during pregnancy. Colorectal Dis 2008; 10:518.

Fauconnier A, Chapron C, Dubuisson JB, et al. Relation between pain symptoms and the anatomic location of deep infiltrating endometriosis. Fertil Steril 2002; 78:719.

Fedele L, Bianchi S, Montefusco S, et al. A gonadotropin-releasing hormone agonist versus a continuous oral contraceptive pill in the treatment of bladder endometriosis. Fertil Steril 2008; 90:183.

Fedele L, Bianchi S, Zanconato G, et al. Is rectovaginal endometriosis a progressive disease? Am J Obstet Gynecol 2004; 191:1539.

Fedele L, Bianchi S, Zanconato G, et al. Long-term follow-up after conservative surgery for bladder endometriosis. Fertil Steril 2005; 83:1729.

Fedele L, Piazzola E, Raffaelli R, Bianchi S. Bladder endometriosis: deep infiltrating endometriosis or adenomyosis? Fertil Steril 1998; 69:972.

Fernando S, Breheny S, Jaques AM, et al. Preterm birth, ovarian endometriomata, and assisted reproduction technologies. Fertil Steril 2009; 91:325.

Francica G, Giardiello C, Angelone G, et al. Abdominal wall endometriomas near cesarean delivery scars: sonographic and color doppler findings in a series of 12 patients. J Ultrasound Med 2003; 22:1041.

Fremont RD, Rice TW. Splenosis: a review. South Med J 2007; 100:589.

Frenna V, Santos L, Ohana E, et al. Laparoscopic management of ureteral endometriosis: our experience. J Minim Invasive Gynecol 2007; 14:169.

Fukunaga M, Nomura K, Ishikawa E, Ushigome S. Ovarian atypical endometriosis: its close association with malignant epithelial tumours. Histopathology 1997; 30:249.

Gagnon RF, Arsenault D, Pichette V, Tanguay S. Acute renal failure in a young woman with endometriosis. Nephrol Dial Transplant 2001; 16:1499.

Garcia GJ, Extramiana CJ, Esteban CJ, et al. Vesical endometriosis after caesarian section: diagnostico-therapeutic aspects. Acta Urol Esp 1997;21:785-8.

Gardner B, Whitaker RH. The use of danazol for ureteral obstruction caused by endometriosis. J Urol 1981; 125:117.

Ghezzi F, Cromi A, Bergamini V, et al. Outcome of laparoscopic ureterolysis for ureteral endometriosis. Fertil Steril 2006; 86:418.

Goldstein DP, deCholnoky C, Leventhal JM, Emans SJ. New insights into the old problem of chronic pelvic pain. J Pediatr Surg 1979; 14:675.

Goncalves MO, Dias JA Jr, Podgaec S, et al. Transvaginal ultrasound for diagnosis of deeply infiltrating endometriosis. Int J Gynaecol Obstet 2009; 104:156.

Goncalves MO, Podgaec S, Dias JA Jr, et al. Transvaginal ultrasonography with bowel preparation is able to predict the number of lesions and rectosigmoid layers affected in cases of deep endometriosis, defining surgical strategy. Hum Reprod 2010; 25:665.

Grasso RF, Di Giacomo V, Sedati P, et al. Diagnosis of deep infiltrating endometriosis: accuracy of magnetic resonance imaging and transvaginal 3D ultrasonography. Abdom Imaging 2010; 35:716.

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.

Hadfield RM, Lain SJ, Raynes-Greenow CH, et al. Is there an association between endometriosis and the risk of pre-eclampsia? A population based study. Hum Reprod 2009; 24:2348.

Harada T, Iwabe T, Terakawa N. Role of cytokines in endometriosis. Fertil Steril 2001; 76:1.
Hediger ML, Hartnett HJ, Louis GM. Association of endometriosis with body size and figure. Fertil Steril 2005; 84:1366.

Hensen JH, Van Breda Vriesman AC, Puylaert JB. Abdominal wall endometriosis: clinical presentation and imaging features with emphasis on sonography. AJR Am J Roentgenol 2006; 186:616.

Holland TK, Yazbek J, Cutner A, et al. Value of transvaginal ultrasound in assessing severity of pelvic endometriosis. Ultrasound Obstet Gynecol 2010; 36:241.

Hornstein MD, Thomas PP, Sober AJ, et al. Association between endometriosis, dysplastic naevi and history of melanoma in women of reproductive age. Hum Reprod 1997; 12:143.

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.

Hsieh YY, Chang CC, Tsai FJ, et al. Estrogen receptor alpha dinucleotide repeat and cytochrome P450c17alpha gene polymorphisms are associated with susceptibility to endometriosis. Fertil Steril 2005; 83:567.

Hudelist G, English J, Thomas AE, et al. Diagnostic accuracy of transvaginal ultrasound for non-invasive diagnosis of bowel endometriosis: systematic review and meta-analysis. Ultrasound Obstet Gynecol 2011; 37:257.

Hudelist G, Oberwinkler KH, Singer CF, et al. Combination of transvaginal sonography and clinical examination for preoperative diagnosis of pelvic endometriosis. Hum Reprod 2009; 24:1018.

Hudelist G, Tuttilies F, Rauter G, et al. Can transvaginal sonography predict infiltration depth in patients with deep infiltrating endometriosis of the rectum? Hum Reprod 2009; 24:1012.

Huhtinen K, Suvitie P, Hiissa J, et al. Serum HE4 concentration differentiates malignant ovarian tumours from ovarian endometriotic cysts. Br J Cancer 2009; 100:1315.

Husby GK, Haugen RS, Moen MH. Diagnostic delay in women with pain and endometriosis. Acta Obstet Gynecol Scand 2003; 82:649.

Jansen RP, Russell P. Nonpigmented endometriosis: clinical, laparoscopic, and pathologic definition. Am J Obstet Gynecol 1986; 155:1154.

Jenkins S, Olive DL, Haney AF. Endometriosis: pathogenetic implications of the anatomic distribution. Obstet Gynecol 1986; 67:335.

Jepsen JM, Hansen KB. Danazol in the treatment of ureteral endometriosis. J Urol 1988; 139:1045.

Kané C, Drouin P. Obstructive uropathy associated with endometriosis. Am J Obstet Gynecol 1985; 151:207.

Kennedy S, Bergqvist A, Chapron C, et al. ESHRE guideline for the diagnosis and treatment of endometriosis. Hum Reprod 2005; 20:2698.

Kennedy S. Genetics of endometriosis: a review of the positional cloning approaches. Semin Reprod Med 2003; 21:111.

Kinkel K, Frei KA, Balleyguier C, Chapron C. Diagnosis of endometriosis with imaging: a review. Eur Radiol 2006; 16:285.

Koninckx PR, Meuleman C, Demeyere S, et al. Suggestive evidence that pelvic endometriosis is a progressive disease, whereas deeply infiltrating endometriosis is associated with pelvic pain. Fertil Steril 1991; 55:759.

Kovoor E, Nassif J, Miranda-Mendoza I, Wattiez A. Endometriosis of bladder: outcomes after laparoscopic surgery. J Minim Invasive Gynecol 2010; 17:600.
Laufer MR, Goitein L, Bush M, et al. Prevalence of endometriosis in adolescent girls with chronic pelvic pain not responding to conventional therapy. J Pediatr Adolesc Gynecol 1997; 10:199.

Lebovic DI, Mueller MD, Taylor RN. Immunobiology of endometriosis. Fertil Steril 2001; 75:1.

Lousquy R, Borghese B, Chapron C. [Deep bladder endometriosis: how do I...to perform a laparoscopic partial cystectomy?]. Gynecol Obstet Fertil 2010; 38:697.

Lucero SP, Wise HA, Kirsh G, Devoe K, Hess ML, Kandawalla N, et al. Ureteric obstruction secondary to endometriosis. Report of three cases with review of literature. Br J Urol 1988;61:201-4.

Management of endometriosis in the presence of pelvic pain. The American Fertility Society. Fertil Steril 1993; 60:952.

Marinis A, Vassiliou J, Kannas D, et al. Endometriosis mimicking soft tissue tumors: diagnosis and treatment. Eur J Gynaecol Oncol 2006; 27:168.

Matalliotakis I, Cakmak H, Dermitzaki D, et al. Increased rate of endometriosis and spontaneous abortion in an in vitro fertilization program: no correlation with epidemiological factors. Gynecol Endocrinol 2008; 24:194.

Matorras R, Rodríguez F, Gutierrez de Terán G, et al. Endometriosis and spontaneous abortion rate: a cohort study in infertile women. Eur J Obstet Gynecol Reprod Biol 1998; 77:101.

Matsuura K, Kawasaki N, Oka M, et al. Treatment with danazol of ureteral obstruction caused by endometriosis. Acta Obstet Gynecol Scand 1985; 64:339.

McArthur JW, Ulfelder H. The effect of pregnancy upon endometriosis. Obstet Gynecol Surv 1965; 20:709.

Melin A, Sparén P, Bergqvist A. The risk of cancer and the role of parity among women with endometriosis. Hum Reprod 2007; 22:3021.

Mereu L, Gagliardi ML, Clarizia R, et al. Laparoscopic management of ureteral endometriosis in case of moderate-severe hydroureteronephrosis. Fertil Steril 2010; 93:46.

Minaglia S, Mishell DR Jr, Ballard CA. Incisional endometriomas after Cesarean section: a case series. J Reprod Med 2007; 52:630.

Missmer SA, Hankinson SE, Spiegelman D, et al. Reproductive history and endometriosis among premenopausal women. Obstet Gynecol 2004; 104:965.

Modugno F, Ness RB, Allen GO, et al. Oral contraceptive use, reproductive history, and risk of epithelial ovarian cancer in women with and without endometriosis. Am J Obstet Gynecol 2004; 191:733.

Mol BW, Bayram N, Lijmer JG, et al. The performance of CA-125 measurement in the detection of endometriosis: a meta-analysis. Fertil Steril 1998; 70:1101.

Montagnana M, Lippi G, Ruzzene O, et al. The utility of serum human epididymis protein 4 (HE4) in patients with a pelvic mass. J Clin Lab Anal 2009; 23:331.
Moore JG, Hibbard LT, Growdon WA, Schifrin BS. Urinary tract endometriosis: Enigmas in diagnosis and management. Am J Obstet Gynaecol 1979; 134:162-72.

Muzii L, Bianchi A, Bellati F, et al. Histologic analysis of endometriomas: what the surgeon needs to know. Fertil Steril 2007; 87:362.

Nazhat C, Nazhat F, Nazhat CH, Nasserbakht F, Rosati M, Seidman DS. Urinary tract endometriosis treated by laparoscopy. Fertil Steril 1996; 66:920-4.

Nerli RB, Reddy M, Koura AC, et al. Cystoscopy-assisted laparoscopic partial cystectomy. J Endourol 2008; 22:83.

Nezhat C, Nezhat F, Nezhat CH, et al. Urinary tract endometriosis treated by laparoscopy. Fertil Steril 1996; 66:920.

Nezhat CH, Malik S, Osias J, et al. Laparoscopic management of 15 patients with infiltrating endometriosis of the bladder and a case of primary intravesical endometrioid adenocarcinoma. Fertil Steril 2002; 78:872.

Nezhat F, Datta MS, Hanson V, et al. The relationship of endometriosis and ovarian malignancy: a review. Fertil Steril 2008; 90:1559.

Ogawa S, Kaku T, Amada S, et al. Ovarian endometriosis associated with ovarian carcinoma: a clinicopathological and immunohistochemical study. Gynecol Oncol 2000; 77:298.

Olive DL, Henderson DY. Endometriosis and mullerian anomalies. Obstet Gynecol 1987; 69:412.

Olive DL, Schwartz LB. Endometriosis. N Engl J Med 1993; 328:1759.

Oosterlynck DJ, Cornillie FJ, Waer M, et al. Women with endometriosis show a defect in natural killer activity resulting in a decreased cytotoxicity to autologous endometrium. Fertil Steril 1991; 56:45.

Pastor-Navarro H, Giménez-Bachs JM, Donate-Moreno MJ, et al. Update on the diagnosis and treatment of bladder endometriosis. Int Urogynecol J Pelvic Floor Dysfunct 2007; 18:949.

Pérez-Utrilla Pérez M, Aguilera Bazán A, Alonso Dorrego JM, et al. Urinary tract endometriosis: clinical, diagnostic, and therapeutic aspects. Urology 2009; 73:47.

Piketty M, Chopin N, Dousset B, et al. Preoperative work-up for patients with deeply infiltrating endometriosis: transvaginal ultrasonography must definitely be the first-line imaging examination. Hum Reprod 2009; 24:602.

Pisanu A, Deplano D, Angioni S, et al. Rectal perforation from endometriosis in pregnancy: case report and literature review. World J Gastroenterol 2010; 16:648.

Porpora MG, Koninckx PR, Piazzo J, et al. Correlation between endometriosis and pelvic pain. J Am Assoc Gynecol Laparosc 1999; 6:429.

PREFUMO F, TODESCCHINI F, FULCHERI E, VENTURINI PL. Epithelial abnormalities in cystic ovarian endometriosis. Gynecol Oncol 2002; 84:280.

Propst AM, Laufer MR. Endometriosis in adolescents. Incidence, diagnosis and treatment. J Reprod Med 1999; 44:751.

Redwine DB. Diaphragmatic endometriosis: diagnosis, surgical management, and long-term results of treatment. Fertil Steril 2002; 77:288.

Reese KA, Reddy S, Rock JA. Endometriosis in an adolescent population: the Emory experience. J Pediatr Adolesc Gynecol 1996; 9:125.

Revised American Society for Reproductive Medicine classification of endometriosis. Fertil Steril 1997; 67:817.

Rivlin ME, Krueger RP, Wiser WL. Danazol in the management of ureteral obstruction secondary to endometriosis. Fertil Steril 1985; 44:274.
Sanchez Merino JM, Parra Muntaner L, Guillan Maquieira C, Gomez Cisneros SC, Alonso Ortiz J, Garcia Alonso J. Bladder endometriosis. Arch Esp Urol 1999; 52:933-5.
Sangi-Haghpeykar H, Poindexter AN 3rd. Epidemiology of endometriosis among parous women. Obstet Gynecol 1995; 85:983.
Sangi-Haghpeykar H, Poindexter AN 3rd. Epidemiology of endometriosis among parous women. Obstet Gynecol 1995; 85:983.
Savelli L, Manuzzi L, Pollaristi P, et al. Diagnostic accuracy and potential limitations of transvaginal sonography for bladder endometriosis. Ultrasound Obstet Gynecol 2009; 34:595.
Savoca G, Trombetta C, Troiano L, Guaschino S, Raber M, Belgrano E. Ecographic, MRI and CT features in a case of bladder endometriosis. Arch Ital Urol Androl 1996; 68:193-6.
Schenken RS, Williams RF, Hodgen GD. Effect of pregnancy on surgically induced endometriosis in cynomolgus monkeys. Am J Obstet Gynecol 1985; 157:1392.
Schneider A, Touloupidis S, Papatsoris AG, et al. Endometriosis of the urinary tract in women of reproductive age. Int J Urol 2006; 13:902.
Scioscia M, Molon A, Grosso G, Minelli L. Laparoscopic management of ureteral endometriosis. Curr Opin Obstet Gynecol 2009; 21:325.
Seaman HE, Ballard KD, Wright JT, de Vries CS. Endometriosis and its coexistence with irritable bowel syndrome and pelvic inflammatory disease: findings from a national case-control study--Part 2. BJOG 2008; 115:1392.
Seidman JD. Prognostic importance of hyperplasia and atypia in endometriosis. Int J Gynecol Pathol 1996; 15:1.
Sener A, Chew BH, Duvdevani M, et al. Combined transurethral and laparoscopic partial cystectomy and robot-assisted bladder repair for the treatment of bladder endometrioma. J Minim Invasive Gynecol 2006; 13:245.
Seracchioli R, Mabrouk M, Manuzzi L, et al. Importance of retroperitoneal ureteric evaluation in cases of deep infiltrating endometriosis. J Minim Invasive Gynecol 2008; 15:435.
Seracchioli R, Mannini D, Colombo FM, et al. Cystoscopy-assisted laparoscopic resection of extramucosal bladder endometriosis. J Endourol 2002; 16:663.
Simpson JL, Elias S, Malinak LR, Buttram VC Jr. Heritable aspects of endometriosis. I. Genetic studies. Am J Obstet Gynecol 1980; 137:327.
Sinaii N, Cleary SD, Ballweg ML, et al. High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome and atopic diseases among women with endometriosis: a survey analysis. Hum Reprod 2002; 17:2713.
Sinaii N, Plumb K, Cotton L, et al. Differences in characteristics among 1,000 women with endometriosis based on extent of disease. Fertil Steril 2008; 89:538.
Somigliana E, Vercellini P, Gattei U, et al. Bladder endometriosis: getting closer and closer to the unifying metastatic hypothesis. Fertil Steril 2007; 87:1287.
Somigliana E, Viganò P, Abbati A, et al. 'Here comes the sun': pigmentary traits and sun habits in women with endometriosis. Hum Reprod 2010; 25:728.
Somigliana E, Viganó P, Parazzini F, et al. Association between endometriosis and cancer: a comprehensive review and a critical analysis of clinical and epidemiological evidence. Gynecol Oncol 2006; 101:331.
Stanley KE Jr, Utz DC, Dockerty Mb. Clinically Significant Endometriosis Of The Urinary Tract. Surg Gynecol Obstet 1965; 120:491.
Stanley KE Jr, Utz DC, Dockerty MB. Clinically Significant Endometriosis Of The Urinary Tract. Surg Gynecol Obstet 1965; 120:491.

www.intechopen.com
Steed H, Chapman W, Laframboise S. Endometriosis-associated ovarian cancer: a clinicopathologic review. J Obstet Gynaecol Can 2004; 26:709.

Steele RW, Dmowski WP, Marmer DJ. Immunologic aspects of human endometriosis. Am J Reprod Immunol 1984; 6:33.

Stegmann BJ, Sinaii N, Liu S, et al. Using location, color, size, and depth to characterize and identify endometriosis lesions in a cohort of 133 women. Fertil Steril 2008; 89:1632.

Stephansson O, Kieler H, Granath F, Falconer H. Endometriosis, assisted reproduction technology, and risk of adverse pregnancy outcome. Hum Reprod 2009; 24:2341.

Stovall TG, Ling FW. Splenosis: report of a case and review of the literature. Obstet Gynecol Surv 1988; 43:69.

Sutton CJ, Ewen SP, Whitelaw N, Haines P. Prospective, randomized, double-blind, controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal, mild, and moderate endometriosis. Fertil Steril 1994; 62:696.

Sutton CJ, Pooley AS, Ewen SP, Haines P. Follow-up report on a randomized controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal to moderate endometriosis. Fertil Steril 1997; 68:1070.

Thomas Ej, Campbell IG. Molecular genetic defects in endometriosis. Gynecol Obstet Invest 2000; 50 Suppl 1:44.

Tran LV, Tokushige N, Berbic M, et al. Macrophages and nerve fibres in peritoneal endometriosis. Hum Reprod 2009; 24:835.

Treloar SA, Bell TA, Nagle CM, et al. Early menstrual characteristics associated with subsequent diagnosis of endometriosis. Am J Obstet Gynecol 2010; 202:534.e1.

Van Gorp T, Amant F, Neven P, et al. Endometriosis and the development of malignant tumours of the pelvis. A review of literature. Best Pract Res Clin Obstet Gynaecol 2004; 18:349.

Van Langendonckt A, Casanas-Roux F, Donnez J. Oxidative stress and peritoneal endometriosis. Fertil Steril 2002; 77:861.

Vercammen EE, D’Hooghe TM. Endometriosis and recurrent pregnancy loss. Semin Reprod Med 2000; 18:363.

Vercellini P, Aimi G, De Giorgi O, et al. Is cystic ovarian endometriosis an asymmetric disease? Br J Obstet Gynaecol 1998; 105:1018.

Vercellini P, Busacca M, Aimi G, et al. Lateral distribution of recurrent ovarian endometriotic cysts. Fertil Steril 2002; 77:848.

Vercellini P, Frontino G, Pietropaolo G, et al. Deep endometriosis: definition, pathogenesis, and clinical management. J Am Assoc Gynecol Laparosc 2004; 11:153.

Vercellini P, Frontino G, Pisacreta A, et al. The pathogenesis of bladder detrusor endometriosis. Am J Obstet Gynecol 2002; 187:538.

Vercellini P, Pisacreta A, Pesole A, et al. Is ureteral endometriosis an asymmetric disease? BJOG 2000; 107:559.

Vercellini P, Trespidi L, De Giorgi O, et al. Endometriosis and pelvic pain: relation to disease stage and localization. Fertil Steril 1996; 65:299.

Victory R, Diamond MP, Johns DA. Villar’s nodule: a case report and systematic literature review of endometriosis externa of the umbilicus. J Minim Invasive Gynecol 2007; 14:23.

Viganò P, Somigliana E, Parazzini F, Vercellini P. Bias versus causality: interpreting recent evidence of association between endometriosis and ovarian cancer. Fertil Steril 2007; 88:588.

Wang G, Tokushige N, Markham R, Fraser IS. Rich innervation of deep infiltrating endometriosis. Hum Reprod 2009; 24:827.
Watson WJ, Sundwall DA, Benson WL. Splenosis mimicking endometriosis. Obstet Gynecol 1982; 59:51S.

Westney OL, Amundsen CL, McGuire EJ. Bladder endometriosis: conservative management. J Urol 2000; 163:1814.

Wiegand KC, Shah SP, Al-Agha OM, et al. ARID1A mutations in endometriosis-associated ovarian carcinomas. N Engl J Med 2010; 363:1532.

Winkel CA. Evaluation and management of women with endometriosis. Obstet Gynecol 2003; 102:397.

Witz CA. Interleukin-6: another piece of the endometriosis-cytokine puzzle. Fertil Steril 2000; 73:212.

Wykes CB, Clark TJ, Khan KS. Accuracy of laparoscopy in the diagnosis of endometriosis: a systematic quantitative review. BJOG 2004; 111:1204.

Yamamoto K, Mitsuhashi Y, Takaïke T, et al. Tubal endometriosis diagnosed within one month after menarche: a case report. Tohoku J Exp Med 1997; 181:385.

Yates-Bell AJ, Molland MB, Pryor JP. Endometriosis of the ureter. Br J Urol 1972;44:58-67.

Yohannes P. Ureteral endometriosis. J Urol 2003; 170:20.

Zanetta G, Webb MJ, Segura JW. Ureteral endometriosis diagnosed at ureteroscopy. Obstet Gynecol 1998; 91:857.

Zhao X, Lang J, Leng J, et al. Abdominal wall endometriomas. Int J Gynaecol Obstet 2005; 90:218.
This book provides an insight into the emerging trends in pathogenesis, diagnosis and management of endometriosis. Key features of the book include overviews of endometriosis; endometrial angiogenesis, stem cells involvement, immunological and hormonal aspects related to the disease pathogenesis; recent research reports on infertility, endometrial receptivity, ovarian cancer and altered gene expression associated with endometriosis; various predictive markers, and imaging modalities including MRI and ultrasound for efficient diagnosis; as well as current non-hormonal and hormonal treatment strategies. This book is expected to be a valuable resource for clinicians, scientists and students who would like to have an improved understanding of endometriosis and also appreciate recent research trends associated with this disease.

**How to reference**

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Aliasghar Yarmohamadi and Nasser Mogharabian (2012). Urinary Tract Endometriosis, **Endometriosis - Basic Concepts and Current Research Trends**, Prof. Koel Chaudhury (Ed.), ISBN: 978-953-51-0524-4, InTech, Available from: http://www.intechopen.com/books/endometriosis-basic-concepts-and-current-research-trends/urinary-tract-endometriosis
