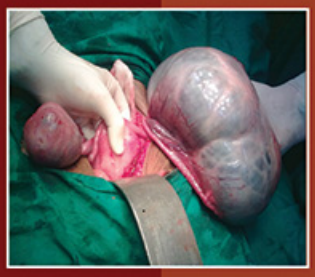
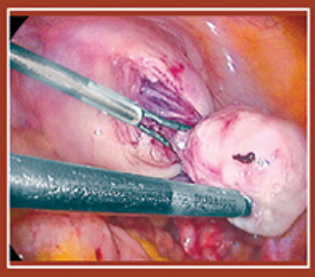
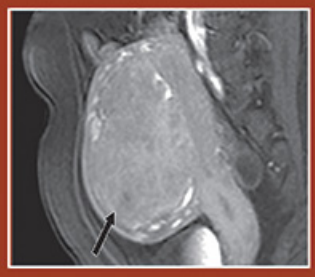
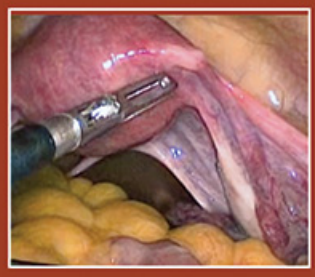

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■ Instruments 590; • Spatula, Broom (Plastic) and Cytobrush 590; • Sims' Double Bladed Posterior Vaginal Speculum 591; • Cusco's Bivalve Self-retaining Vaginal Speculum 592; • Auvard's Self-retaining Posterior Vaginal Speculum 592; • Female Rubber Catheter 593; • Female Metal Catheter 593; • Foley's Catheter 594; • Cervical Dilators 594; • Multiple Toothed Vulsellum 595; • Single Toothed Vulsellum 595; • Anterior Vaginal Wall Retractor 595; • Olive Pointed Malleable Graduated Metallic Uterine Sound 596; • Uterine Curette 596; • Uterine Dressing Forceps 597; • Sponge Holding Forceps 597;

• Ovum Forceps 598; • Allis Tissue Forceps 598; • Lanes Tissue Forceps 599; • Uterus Holding Forceps 599; • Cervical Occlusion Clamp 599; • Myoma Screw 599; • Bonney's Myomectomy Clamp 600; • Hysterosalpingography Cannula (Leech Wilkinson Variety) 600; • Kocher's Artery Forceps 601; • Landon's Bladder Retractor 601; • Insufflation Cannula 601; • Abdominal Retractors 602; • Doyen's Retractor 602; • Balfour Self-retaining Retractor 602; • Deaver's Retractor 602; • Long Straight Hemostatic Forceps (Spencer Well's) 603; • Babcock's Forceps 603; • Needle Holder 603; • Punch Biopsy Forceps 604; • Dissecting Forceps 604; • Scalpel 604; • Needles 605; • Scissors 605; • Towel Clips 606; • Loop Hook 606; • Electrocautery 606; • Cryoprobe 607; • Laparoscopic Instruments 607; • Trocar and Cannula 607; • Hysteroscopic Instruments 608; • Hodge-Smith Pessary 608; • Ring Pessary 608; • Laparoscopic Ovarian Drill Needle 609; • Processing of Instruments 609; • Sterilization of Instruments 609; ■ Suture Materials 609; • Classification 609; ■ Specimens 611; ◆ Imaging Studies in Gynecology 622; ■ Plates: Skiagraphs, Ultrasonographs, Computed Tomographs, and Magnetic Resonance Imagings 622; • Hysterosalpingogram 622; • Ultrasonographs 625; • Radiographs and CT Scans 628; • MRI 631; • Nomogram for Calculating Body Surface Area of Adults 633; • Nomogram for Calculating Body Mass Index 634

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22 CHAPTER

Endometriosis and Adenomyosis

CHAPTER OUTLINE

- **Endometriosis**
  - **Prevalence and Sites**
  - **Pathogenesis and Pathology**
  - **Diagnosis**
  - **Differential Diagnosis**
  - **Complications of Endometriosis**
  - **Staging of Endometriosis**
  - **Treatment of Endometriosis**
  - **Surgical Management of Endometriosis**
  - **Endometriosis at Special Sites**
- **Adenomyosis**
  - **Causes**
  - **Pathogenesis**
  - **Pathology**
  - **Types of Adenomyosis**
  - **Clinical Features**
  - **Treatment**

ENDOMETRIOSIS

DEFINITION

Presence of functioning endometrium (glands and stroma) in sites other than uterine mucosa is called endometriosis. It is not a neoplastic condition, although malignant transformation is possible.

These ectopic endometrial tissues may be found in the myometrium when it is called endometriosis interna or **adenomyosis**. Most commonly, however, these tissues are found at sites other than uterus and are called endometriosis externa or generally referred to as endometriosis.

Endometriosis is a disease of contrast. Although it is a benign but it is locally invasive and disseminates widely. Cyclic hormones stimulate growth but continuous hormones suppress it.

Women with extensive disease may remain asymptomatic whereas a patient with minimal disease may have incapacitating chronic pelvic pain and other symptoms. **Endometriosis is an aggressive, progressive (31%) and invasive disease**.

PREVALENCE

During the last couple of decades, the prevalence of endometriosis has been increasing both in terms of real and apparent. **The real one** is due to delayed marriage, postponement of first conception and adoption of small family norm. **The apparent one** is due to increased use of diagnostic laparoscopy as well as heightened awareness of this disease complex amongst the gynecologists.

The prevalence is about 10–15%. However, prevalence is high amongst the subfertile women (30–45%) as based on diagnostic laparoscopy and laparotomy.

SITES (TABLE 22.1)

- **Abdominal**
- **Extra-abdominal**
- **Remote**

Abdominal

It can occur at any site but is usually confined to the abdominal structures below the level of umbilicus.

Extra-abdominal

The common sites are abdominal scar of hysterotomy, cesarean section, tubectomy and myomectomy, umbilicus, episiotomy scar, vagina and cervix.

TABLE 22.1: Sites of endometriosis (Fig. 22.1).

Common sites	*Rare and remote sites*
■ Ovaries (80%), bilateral (50%)	■ Umbilicus
■ Pelvic peritoneum	■ Abdominal scar
■ Pouch of Douglas	■ Episiotomy scar
■ Uterosacral ligaments	■ Lungs
■ Rectovaginal septum	■ Pleura
■ Sigmoid colon	■ Ureter
■ Appendix	■ Kidney
■ Pelvic lymph nodes	■ Arms
■ Fallopian tubes	■ Legs
	■ Nasal mucosa

Risk Factors for Endometriosis

- Low parity
- Delayed child bearing
- Family history of endometriosis
- Genital (outflow) tract obstruction
- Environmental toxins (dioxins)
- Peritoneal fluid abnormalities **(Table 22.2)**

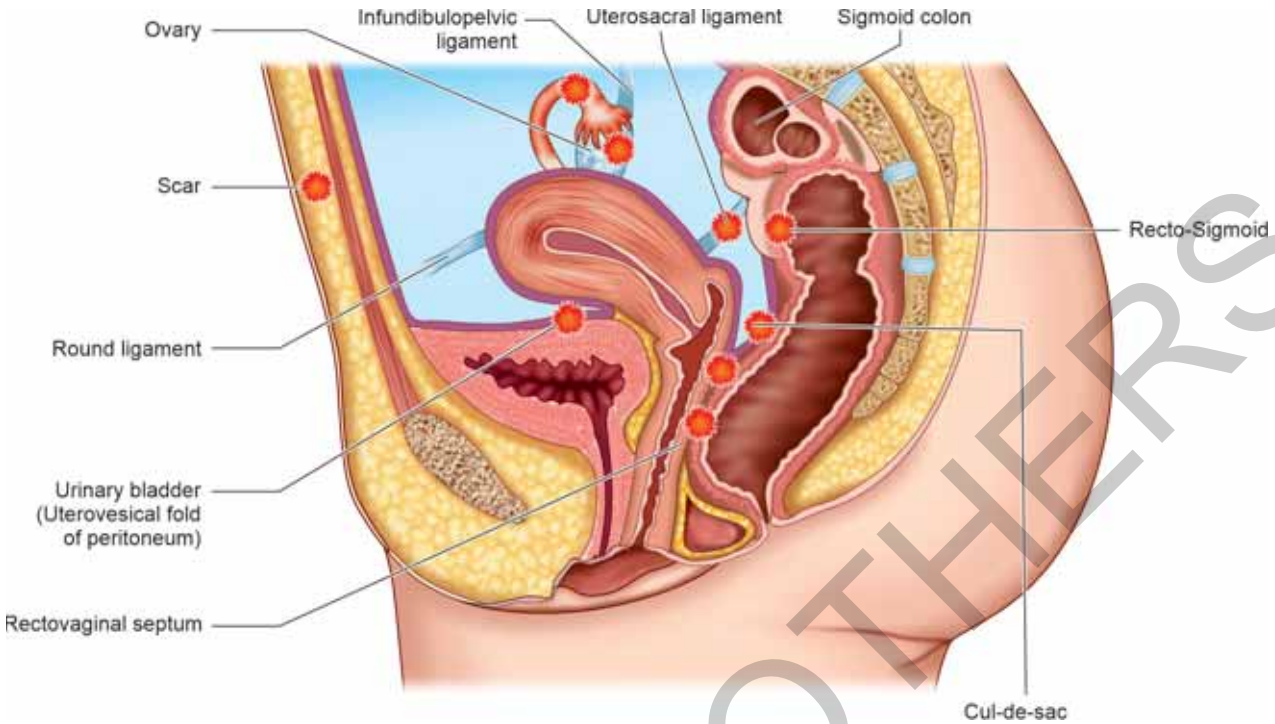


Fig. 22.1: Common sites of endometriosis.

TABLE 22.2 Peritoneal fluid: Cytokines and growth factors

Concentration increased	*Concentration unchanged*	*Concentration decreased*
Complement, glycodelin	EGF	Interleukin (IL-13)
Interleukins: IL-1, IL-6, IL-8	Basic FGF	TGFβ, VEGF
PDGF, RANTES, TGFβ, VEGF	Interleukins: IL-2, IL-4, IL-12	FGF

(TGFβ: transforming growth factor β; VEGF: vascular endothelial growth factor; FGF: fibroblast growth factor; PDGF: platelet derived growth factor)

TABLE 22.3: Theories to explain endometriosis at different sites.

Sites	*Theory*
■ Pelvic endometriosis	■ Retrograde menstruation
■ Pelvic peritoneum	■ Coelomic metaplasia
■ Abdominal viscera ■ Rectovaginal septum ■ Umbilicus	■ Coelomic metaplasia
■ Abdominal scar ■ Episiotomy scar ■ Vagina, cervix	■ Direct implantation
■ Lymph nodes	■ Lymphatic spread
■ Distant sites (lungs, pleura, skin, lymph nodes, nerves)	■ Vascular spread ■ Genetic ■ Immunologic

PATHOGENESIS

Several theories have been posited to explain the pathogenesis. The complex interplay between an individual woman's immunologic response and the amount of retrograde menstruation has been considered the important determinant. **(Table 22.3). The principal ones are:**

- **Retrograde menstruation (Sampson's theory, 1927):** There is retrograde flow of menstrual blood through the uterine tubes during menstruation. The endometrial fragments get implanted in the peritoneal surface of the pelvic organs (dependent sites, e.g., ovaries, uterosacral ligaments). **Outflow tract obstruction** in the genital tract is frequently found in women with endometriosis. This is due to the expression of adhesion molecules on the peritoneal surfaces. Subsequently, cyclic growth and shedding of the endometrium at the ectopic sites occur under the influence of the endogenous ovarian hormones. The shedded endometrial-based adult stem cells and mesenchymal cells attach to the pelvic peritoneum and grow under the hormonal influence as homologous graft. Commonly, endometriosis is observed in areas close to the tubal ostia or in the dependent area of the pelvis. **(Fig. 22.1)**. While this theory can explain pelvic endometriosis, it fails to explain the endometriosis at distant sites.
- **Coelomic metaplasia (Meyer and Ivanoff):** Chronic irritation of the pelvic peritoneum by the menstrual blood may

cause **coelomic metaplasia which results in endometriosis**. Surface epithelium of the ovary can differentiate into different histological cell types. Coelomic epithelium retains the ability for multipotential development.

This theory can explain endometriosis of the abdominal viscera, rectovaginal septum and umbilicus.

- **Direct Implantation (iatrogenic) theory:** According to the theory, the endometrial or decidual tissues start to grow in susceptible individual when implanted in the new sites. Iatrogenic dissemination explains the development of endometriotic implants at the scar tissues. Such sites are abdominal scar following hysterotomy, cesarean section, tubectomy, and myomectomy. Endometriosis at the episiotomy scar, vaginal or cervical site can also be explained with this theory.

  This theory, however, fails to clarify endometriosis at sites other than mentioned.
- **Lymphatic and vascular theory (Halban, 1925):** It may be possible for the normal endometrium to metastasize the pelvic lymph nodes (30%) through the draining lymphatic channels of the uterus.
- **Vascular theory:** Hematogenous spread of endometrium can explain endometriosis at distant sites such as lungs, arms or thighs.
- **Genetic and immunological theory:** Women developing endometriosis have peritoneal macrophages that are larger and hyperactive. These cells secrete multiple growth factors and cytokines to stimulate the development of endometriosis. This explain that not all women develop endometriosis though most have retrograde menstruation. The peritoneal fluid NK cells of women without endometriosis have more cytotoxic effect compared to women with endometriosis.
- **Autoimmune theory of endometriosis:**
  - **Neoangiogenesis**
  - **Upregulation of aromatase, cyclooxygenase-2 (COX-2) activity** with increased local estradiol and PGE2 concentration.
  - **Down regulation** of 17-β HSD2 in the stromal cells in endometriosis.
  - **Down regulation of progesterone receptor-β isoform and development of progesterone resistance**
  - **Increased mitotic activity** of endometriotic cells with the presence of interleukins and growth factors.
  - **Proliferation** of glands and stromal cells
- **Genetic basis and familial predisposition:** Familial occurrence of endometriosis (mothers and daughters) is known (seven-fold increase). It is with polygenic inheritance. Epigenetic factors enhanced by environmental factors are significant. Ethnic variance is noted, as Asian women has nine-fold increased aberrantly expressed genes or gene products in the endometrium. Asian women have increased risk (nine-fold) of endometriosis.

Aberrant Expression of Gene and Gene Products in Endometrium of Women with Endometriosis

- Aromatase
- Hepatocyte growth factor
- Matrix metalloproteinases (MMP) (3,7,11)
- 17β hydroxysteroid dehydrogenase
- Complements (C3)
- Progesterone receptor isoforms
- VEGF
- Glycodelin

- **Environment theory:** suggests somatic mutations of cells due to environmental factors (pollutants, dioxins). Ovarian and deep infiltrating endometriotic lesions are explained with this theory.

Thus, it is certain that, not all cases of endometriosis at different sites can be explained by a single theory.

Summary of etiopathogenesis of endometriosis

- *Genetic mutations (familial clustering)*
- *Immunological*
- *Molecular defects*
- *Mechanical (outflow tract obstruction)*
- *Environmental toxins (dioxins)*
- *Others: Neoangiogenesis, Upregulation of aromatase, down-regulation of 17-β HSD2 and decreased expression of PR-β (progesterone receptor-β) and ultimate development of progesterone resistance.*

PATHOLOGY

General Considerations

- **The endometrium (glands and stroma) in the ectopic sites** has got the potentiality to undergo changes under the action of ovarian hormones.
- **Proliferative changes** are constantly evidenced, the secretory changes are conspicuously absent. It may be due to deficiency of steroid receptors in the ectopic endometrium.
- **Cyclic growth and shedding** continue till menopause. The periodically shed blood may remain encysted or else, the cyst becomes tense and ruptures.
- **Blood is irritant and it causes** dense tissue reaction surrounding the lesion with ultimate fibrosis. If it happens to occur on the pelvic peritoneum, it produces adhesions and puckering of the peritoneum.
- **Deep lesions** with penetration >5 mm are more progressive (DIE).
- **When encysted, the cyst enlarges with cyclic bleeding.** The serum gets absorbed in between the periods and the content inside becomes chocolate colored. Hence, the cyst is called **chocolate cyst** which is commonly located in the ovary. **Chocolate cyst may also** be due to hemorrhagic follicular or corpus luteum cyst or bleeding into a cystadenoma. For this reason, the term **endometrial cyst or endometrioma (Fig. 22.2) is preferred** to chocolate cyst.

takes time, even after cessation of period, to get relief of pain (co-menstrual dysmenorrhea). Pain usually begins after few years pain-free menses. The site of pain is usually deep seated and on the back or rectum.

Increased secretion of PGF 2α, thromboxane β_2 from endometriotic tissue is the cause of pain.

Dyspareunia (20–40%): The dyspareunia is usually deep. It may be due to stretching of the structures of the pouch of Douglas or direct contact tenderness. As such, it is mostly found in endometriosis of the rectovaginal septum or pouch of Douglas and with fixed retroverted uterus.

Chronic pelvic pain: The pain varies from pelvic discomfort to lower abdominal pain or backache. The causes of pain is multifactorial.

Causes of pain in endometriosis

- *Peritoneal inflammation (PGF, cytokines)*
- *Tissue necrosis*
- *Adhesion formation*
- *Nerve irritation due to deep penetration*
- *Release of local inflammatory mediators*
- *Endometrioma formation*

The pain aggravates during the period.

Abdominal pain: There may be variable degrees of abdominal pain around the periods. Sometimes, the pain may be acute due to rupture of chocolate cyst.

Abnormal uterine bleeding (AUB) (15–20%): Menorrhagia is the predominant abnormality. If the ovaries are also involved, polymenorrhea or epimenorrhagia may be pronounced. There may be premenstrual spotting.

Infertility (40–60%): Endometriosis is found in 30–45% of infertile women, whereas in about 40–50% patients with endometriosis suffer from infertility. The multiple factors involved in producing infertility have been depicted in page 215 **(Table 17.2)**. Miscarriage may be due to implantation failure.

Other Symptoms

The symptoms are related to the organ involved.

- **Urinary**—frequency, dysuria, hydronephrosis **(Fig. 22.4)** back pain or even hematuria.

Case History (Fig. 22.4)

Mrs CR, 43 years, seen with the complaints of persistent pelvic pain, worsening during menstruation. Severity of pain gradually deteriorated even with the use of medications. She felt pain radianting to the back and the loin. She developed features of recurrent pyelonephritis. Investigations (USG, MRI) revealed deep infiltrating endometriosis, involving both the ovaries. Ureteric obstruction was observed at the level of the pelvic floor.

MRI revealed gross hydronephrotic and hydroureteric changes in the right side **(Fig. 22.4)**. Laparotomy was done. Total hysterectomy with bilateral salpingo-oophorectomy with removal of bilateral endometriomas were done. Adhesiolysis and ureterolysis were done. Resection of the cicatrized ureteric segment (4 cm) had to be done **(Fig. 22.4)**. Ureter was implanted in the bladder following mobilization. She had uneventful recovery.

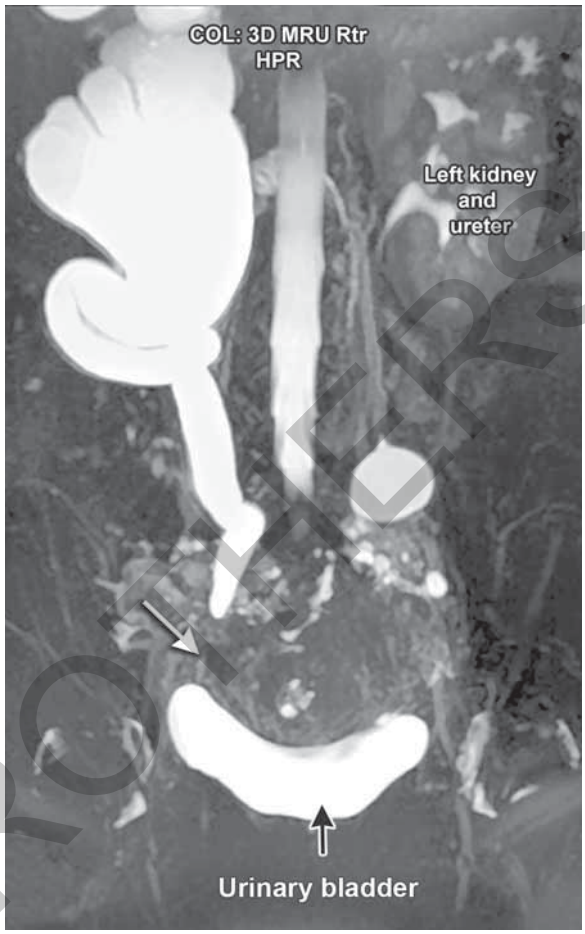


Fig. 22.4: Right ureter showing gross hydroureteric and hydronephrotic changes in a case with advanced (stage IV) deep infiltrating endometriosis (See case history).

- **Sigmoid colon and rectum**—painful defecation (dyschezia), diarrhea, constipation, rectal bleeding or even melena. Patient may also present with symptom s/o IBS.
- **Chronic fatigue**, perimenstrual symptoms (bowel, bladder).
- **Hemoptysis** (rarely), catamenial chest pain, hemothorax.
- **Surgical scars**—cyclical pain and bleeding bloody pleural fluid (described later).

Abdominal examination: Abdominal palpation may not reveal any abnormality. A mass may be felt in the lower abdomen arising from the pelvis—enlarged chocolate cyst or tubo-ovarian mass due to endometriotic adhesions. The mass is tender with restricted mobility.

Pelvic examination: Bimanual examination may not reveal any pathology. **The expected positive findings are**—pelvic tenderness, nodules in the pouch of Douglas, nodular feel of the uterosacral ligaments, fixed retroverted uterus or unilateral or bilateral adnexal mass of varying sizes.

Speculum examination may reveal bluish spots in the posterior fornix or the cervix.

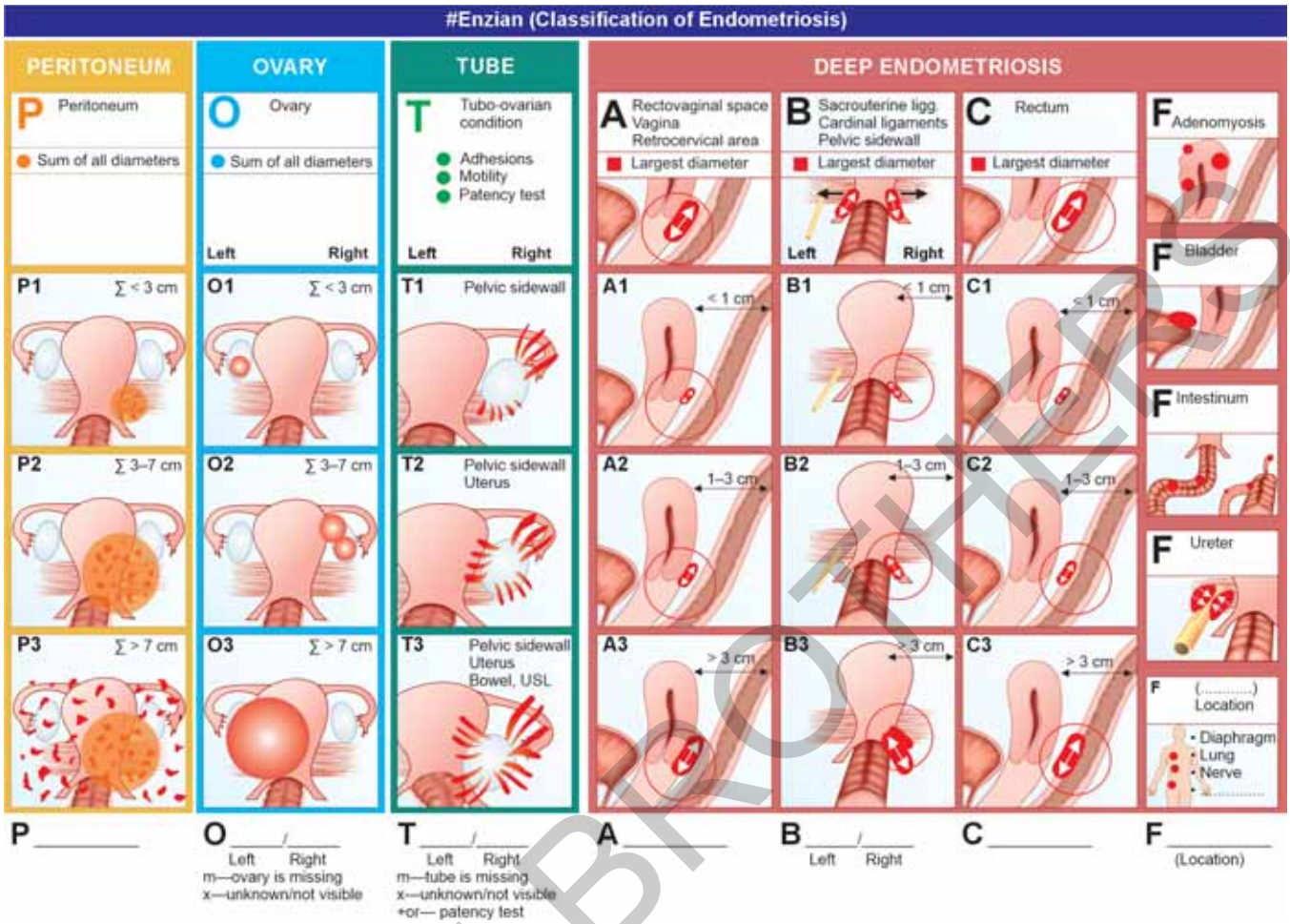


Fig. 22.7: #Enzian classification of endometriosis.

#Enzian classification summary and coding (example): The diameter of a virtual circle is calculated, in which all endometrial foci can be included.

- Superficial endometriosis on the peritoneum <3 cm (P) = P 1; • Ovarian endometriosis Rt. 4 cm (O) = O 0/2; • No adhesion on the tubo-ovarian unit (T) = T 0/0; • DE left USL <1 cm, Rt. USL 1–3 cm = B 1/2; • DE in the rectum 2 cm (C) = C 2; • Hydroureter right (FU) = FU (r); For better reliability only affected compartments or organ should be listed; #Enzian P 1, O 0/2, B 1/2, C 2, FU (r)

The above coding is independent of the imaging modality (TVS, MRI) and types of surgery. In order to mention the modality of evaluation of the pathology in the #Enzian: • #Enzian (u)—assessment by ultrasound; • #Enzian (m)—assessment by MRI; • #Enzian (s)—assessment by surgery

All the lesions with subperitoneal infiltration >5 mm are classified as DE. #Enzian classification includes DE which affects the extraperitoneal space (RVS, bowels, ovaries, bladder or uterus). Endometriomas <3 cm are usually not treated. Patency of fallopian tube is documented with HSG, chromopertubation during surgery. This classification system is thought to be anatomically logical and allows a consistent and uniform assessment till date.

form is filled up. Predictive value for pregnancy with treatment after surgery using the **EFI form** is strongly correlated. For details see author's book ***Clinics in Gynecology***.

The #Enzian classification (Fig. 22.7) is for DE using **three compartments A: vagina, rectovaginal space (RVS); B: uterosacral ligaments (USL)/cardinal ligaments/pelvic sidewall** and **C: rectum** as well as so-called **F (i.e. far locations)** such as the **urinary bladder (FB)**, the **ureters (FU)**, and other **extragenital lesions (FO)**. It additionally covers the involvement of the **peritoneum (P), ovary (O), other intestinal locations** (sigmoid colon, small bowel; **F**), as well as **adhesions,** involving the tubo-ovarian unit (T), and, optionally, tubal patency.

- Individual compartments or organ involvement are identified with capital letters (P, O, T, A, B, C, F) and arranged in this order.
- The extent of endometriosis is represented by the numbers 1, 2 and 3 in compartments P, O, T, A, B, and C.
- Paired organs (ovary, tube, USL, parametrium, ureter): the severity is arranged separately after the letter (left/right).

TABLE 22.5: American Society of Reproductive Medicine (ASRM) scoring system of endometriosis (Revised classification).

	Endometriosis	<1 cm	1–3 cm	>3 cm
Peritoneum	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
	Posterior cul-de-sac obliteration	Partial	Complete	
		4	40	
	Adhesions	<1/3 Enclosure	1/3–2/3 Enclosure	>2/3 Enclosure
Ovary	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.

- ***Stage I*** (minimal) = 1–5
- ***Stage II*** (mild) = 6–15
- ***Stage III*** (moderate) = 16–40
- ***Stage IV*** (severe) = >40

The findings are depicted in a pictorial chart

- Missing/invisible ovary or tube are described with suffix (m, missing; x, unknown).

The individual anatomical locations and their annotation are described below and based on the findings during imaging techniques and/or surgery. In case of ureteral involvement, the side is annotated in a bracket, ie (r) or (l).

No single classification system adequately classifies endometriosis although ASRM classification is most widely used. The Enzian classification describes DE involving the retroperitoneal structures **(Fig. 22.7)**.

Endometriosis Fertility Index (EFI) Surgery Form: Least function score at the end of surgery is obtained as below:

- **Score ranges from 0-4:** absent or nonfunction (=0), severe dysfunction (=1), moderate dysfunction (=2), mild dysfunction (=3), normal (=4).
- **To calculate LF (least function) score:** add together least score for the left side with that of right side. The organs examined are – fallopian tube, fimbria and ovary. If an ovary is absent on one side, the LF score is obtained doubling the least score on the side with ovary.
- **Endometriosis Fertility Index (EFI)** is derived by addition of **(a)** total historical factors from history + **(b)** total surgical factors. **EFI = historical + surgical.**

Factors from the history include point scoring (0–2) – **(a)** Age: ≤35 years =2; 36–39 years =1; ≥40 years =0. **(b)** Years of infertility: ≤3 years =2; >3 years = 0. **(c)** Prior pregnancy: h/o pregnancy=1; no h/o pregnancy=0.

TREATMENT OF ENDOMETRIOSIS

Endometriosis needs to be treated as it is a progressive, aggressive and invasive disease (30–60%).
Goals of treatment are: (a) Pain relief; **(b)** Improvement of fertility and; **(c)** To arrest the progression of the disease.

- **Preventive**
- **Curative**

Preventive

The following guidelines may be prescribed to prevent or minimize endometriosis:

- To avoid tubal patency test immediately after curettage or around the time of menstruation.
- Forcible pelvic examination should not be done during or shortly after menstruation.
- Married women with family history of endometriosis are encouraged not to delay the first conception but to complete the family.

Curative

The objectives are:

- To abolish or minimize the symptoms—pelvic pain and dyspareunia
- To improve the fertility
- To arrest the progress of the disease.
- To prevent recurrence.

The results of treatment are difficult to evaluate because of lack of uniform staging or grading. **The following facts are to be borne in mind.**

- Asymptomatic in good number of cases.
- Subjective symptoms are not proportionate to objective signs.
- Frequent association with infertility.
- Remission during pregnancy and menopause.

Prerequisites prior to therapy are accurate diagnosis with the help of laparoscopy along with staging and pictorial documentation.

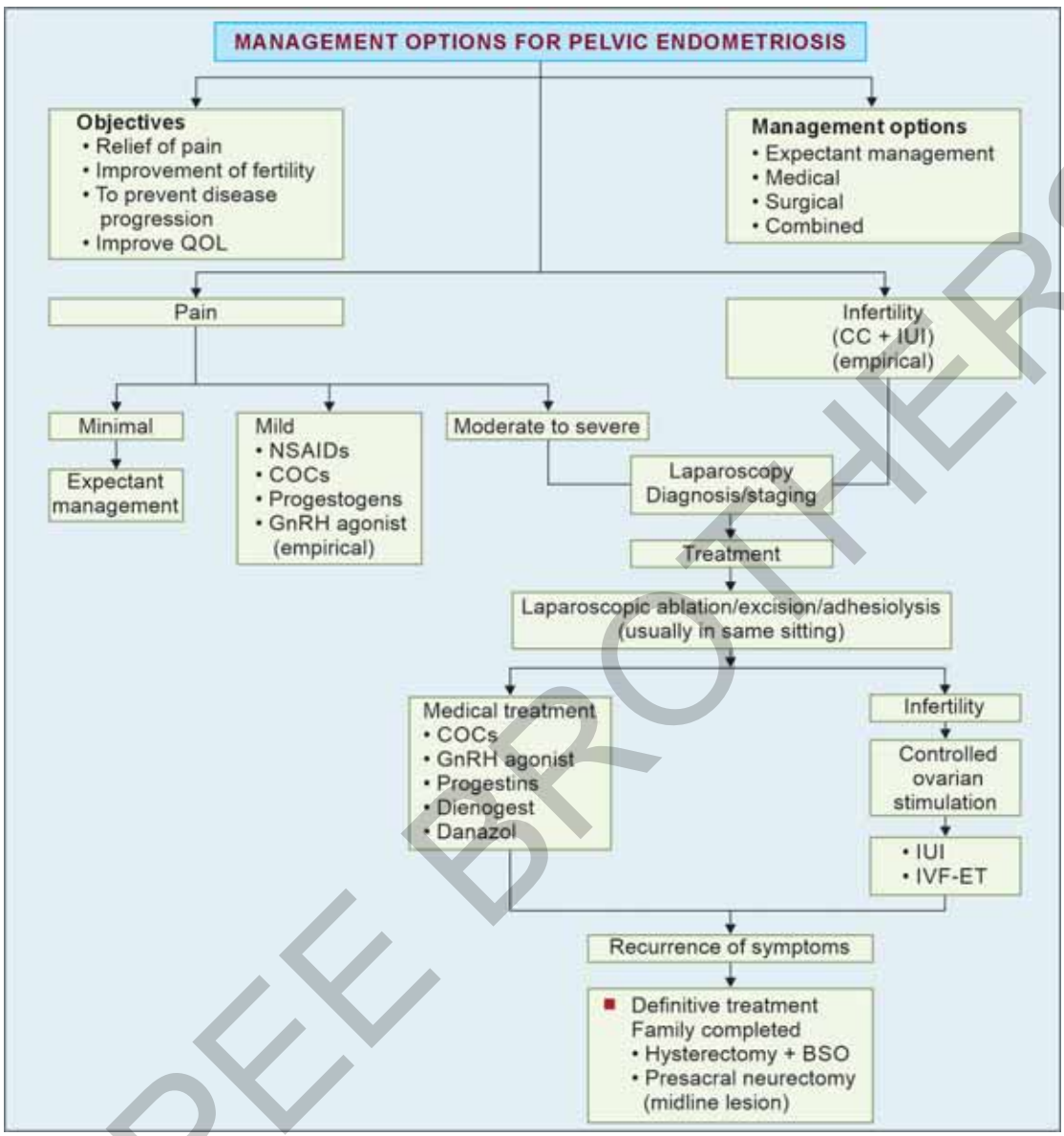
TREATMENT OPTIONS FOR PELVIC ENDOMETRIOSIS (FLOWCHART 22.1)

- **Expectant management** (observation only)
- **Medical therapy:** • Hormones • Others
- **Surgery:** • Conservative • Definitive
- **Combined therapy:** • Medical • Surgical

Determinants of Treatment Options

- Age of the patient
- Location of disease
- Size and extent of lesions
- Desire for fertility
- Severity of symptoms
- Results of previous therapy

Flowchart 22.1: Therapeutic approach to a patient with pelvic endometriosis.



(CC: clomiphene citrate; QOL: quality of life IUI: intrauterine insemination; NSAIDs: nonsteroidal anti-inflammatory drugs; COCs: combined oral contraceptives; GnRH: gonadotropin-releasing hormone; IVF-ET: in vitro fertilization and embryo transfer; BSO; bilateral salpingo-oophorectomy)

Expectant Treatment

Endometriosis is a progressive disease in about 30–60% of women. It is not possible to predict in which woman it will progress. Some form of treatment is reasonable for 3 months to arrest the progress of the disease. However, in women with minimal to mild endometriosis role of any treatment is controversial. *Cumulative pregnancy rate is similar when expectant treatment is compared with conservative surgery. Case selection is important* **(Table 22.6)**.

TABLE 22.6: Case selection for expectant treatment.

- Minimal endometriosis with no other abnormal pelvic finding
- Unmarried
- Young married who are ready to start family
- Approaching menopause

Protocols for Expectant Management

Observation with administration of nonsteroidal anti-inflammatory drugs (NSAIDs) or prostaglandin synthetase inhibiting (PSI) drugs are used to relieve pain. COX-2 inhibitors are effective as the lesions of endometriosis are found to express high levels of COX-2. Ibuprofen 800–1200 mg or mefenamic acid 150–600 mg a day is quite effective.

The married women are encouraged to have conception. Pregnancy usually improve the condition. This is due to absence of shedding and decidual changes in the ectopic endometrium causing its necrosis and absorption.

Danazol (p. 499)

Danazol therapy is to be started from the day 5 of the menstrual cycle. Danazol induces atrophic changes in the endometrium and the endometrial implants. The dose (100–200 mg daily) is variable and depends upon the extent of the lesions but should be adequate enough to produce amenorrhea. **The patient should use barrier methods of contraception** to avoid virilization of a female fetus in accidental pregnancy. **Danazol produces hypoestrogenic and hyperandrogenic state. Duration of treatment is usually 6–9 months. Most (90%) have objective improvement as evidenced on second laparoscopy. Fertility improvement is about 40%. Recurrence rate is 15%–30%.** The side effects are at times intense and intolerable to the extent of discontinuation of the therapy.

Gestrinone

It has got the same mechanism of action like that of danazol. The side effects are less than danazol. Administration is simple, twice a week **(Table 22.7)**.

GnRH Agonists (p. 495)

Commonly Used

When used continuously act as medical oophorectomy, a state of hypoestrinism and amenorrhea. It causes down regulation and desensitization of the pituitary. This results in low levels of circulating estrogen with ultimately amenorrhea (medical oophorectomy). The endometrium becomes atrophic. Commonly used GnRH agonists, the preparations, dose, and route are mentioned (P. 495, Ch. 32). The goal is to maintain a reduced level of serum estrogen (30–45 pg/mL) so that growth of endometriosis is suppressed. **The side effects** are more tolerable than danazol. The drugs are expensive. **Empiric use** of GnRH agonist may be done in women >18 years if pain persists after NSAIDs and combined oral contraceptives (COCs) (ACOG). Long-term therapy (more than 6 months) should be avoided (add-back therapy).

Add-back therapy may be needed to reduce the vasomotor symptoms, vaginal atrophy or the demineralization of bones.

The common side effective are: hot flushes, vaginal dryness and insomnia. With prolonged use (≥ 6 months) bone demineralization has been demonstrated (especially in the lumbar and trabecular bones).

GnRH agonist improves the symptoms in 75 to 90% women. Ovarian function usually returns back to normal by another 6 weeks or more, after use of GnRH agonist for 6 months.

GnRH antagonist can also be used. **It has no "flare" effect. Oral drug (elagolix)** at 150–250 mg is found safe and as effective as that of leuprolide acetate. Bone loss and other side effects are less.

Several newer drugs are currently being used with good response **(Table 22.8)**.

Aromatase inhibitors (Table 22.8) reduces the levels of estrogen in blood as well as in the endometriotic tissues. In premenopausal women it stimulates gonadotropins. It induces ovulation.

Results

The efficacy of the hormone therapy is judged by relief of symptoms, reduction of the volume of the lesions as revealed by second look laparoscopy, improvement of fertility and prevention of recurrence. For quick relief of symptoms and reduction of the volume of the lesion, GnRH analogs are the best. Progestogens take some time to achieve these objectives. Danazol is placed midway between the two.

Taking every aspect together (pain relief, pregnancy rates, recurrence rates, costs, and side effects), no single medical treatment is superior to others.

Following medical suppression or other conservative surgery, residual endometriotic lesions may regenerate once the ovarian function is re-established. **Overall recurrence rate is about 40% after 5 years**.

SURGICAL MANAGEMENT OF ENDOMETRIOSIS

Indications

- Endometriosis with severe symptoms unresponsive to hormone therapy.
- Severe and deeply infiltrating endometriosis (DIE) to correct the distortion of pelvic anatomy.
- Severe endometriosis with pelvic adhesive disease.
- Endometriomas of more than 1 cm.

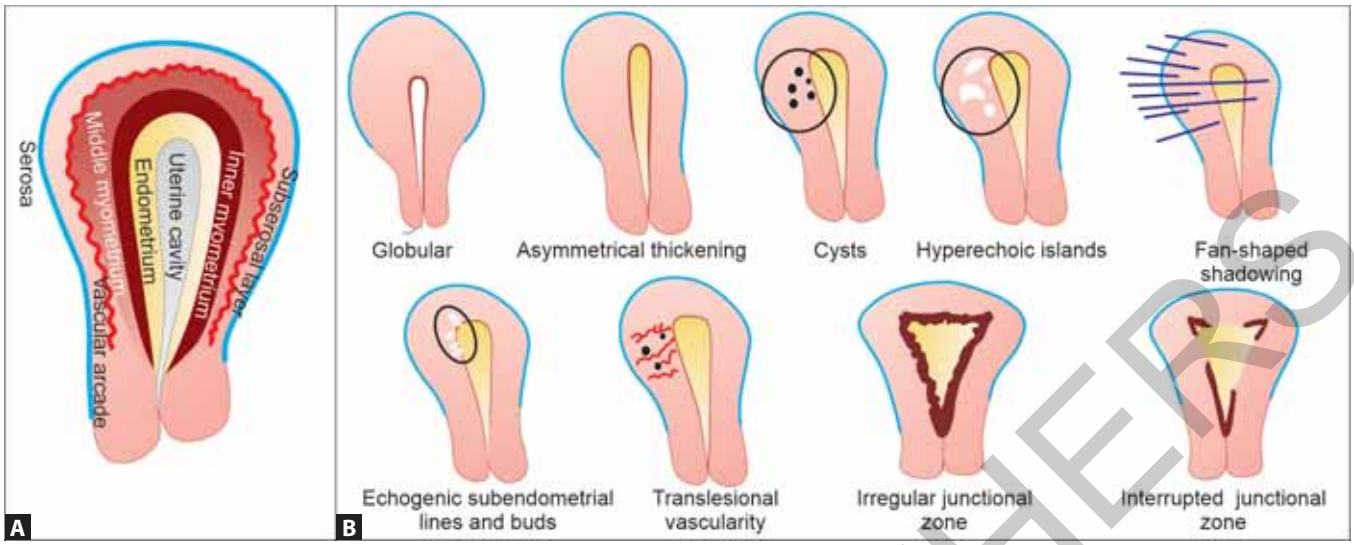
Surgery may be conservative or definitive.

Conservative Surgery

Conservative surgery is aimed to preserve the reproductive function and to restore the normal pelvic anatomy.

TABLE 22.8: Other medications used for the management of endometriosis.

- **Progesterone antagonists** (mifepristone): 50 mg/day PO; may cause endometrial hyperplasia.
- **Selective progesterone-receptor modulators (SPRMs):** Asoprisnil induces endometrial atrophy and amenorrhea.
- **GnRH antagonists:** Oral (elagolix): 150–200 mg/day **as effective as leuprolide acetate, less demineralization of bones, no hypoestrogenic symptoms.**
- **Aromatase inhibitors:** Anastrozole (1 mg) or letrozole (2.5 mg) reduces pain symptoms.
- **Pentoxifylline** an anti-inflammatory immunomodulators, is found to have some promise in treatment.



Figs. 22.13A and B: (A) Schematic diagram showing uterine layers (color-coded)—endometrium is yellow, junctional zone (inner myometrium) is dark red, middle myometrium, located between vascular arcade (red) and junctional zone, is light red; outer myometrium (subserosa), located between vascular arcade and serosa, is pink and serosa is blue; (B) Morphological Uterus Sonographic Assessment (MUSA) criteria for diagnosis of adenomyosis (Adopted from Bosch-2019).

Surgical Management

(A) Conservative surgery: (a) Adenomyomectomy; **(b)** Uterine mass reduction (laparotomy or laparoscopy); **(c)** Uterine artery embolization

Or

(B) Definitive surgery: Hysterectomy (parous and aged women).

Hysterectomy is the definitive treatment provided, it is appropriate to the woman. UAE has been done to relieve symptoms.

POINTS

- **Endometriosis** is the presence of functioning endometrium (stroma and glands) in sites other than uterine mucosa. **Endometriosis** is a disease seen in the reproductive years of a woman as its growth depends on estrogen. The incidence is about 10% but incidence is high (30–40%) amongst infertile women as based on diagnostic laparoscopy and laparotomy. The most common abdominal site is ovary followed by pouch of Douglas and uterosacral ligaments (organs on the dependent part of the pelvis).
- ***Biochemical mediators and the associated pathology in endometriosis are:***
  - ***Cytokines, interleukin-I, TNFα*** → • *Growth of ectopic endometrial cells* • *Prevention of cell apoptosis* • *Adhesion formation*
  - ***VEGF—A*** → *neoangiogenesis* • ***Matrix metalloproteinase*** → *invasion* • ***Estrogens*** → *cell proliferation*
  - ***Macrophages*** → *Sperm phagocytosis* • ***Prostaglandin and cytokines*** → *Inflammation.*
  - ***Causes of pain in endometriosis is due to:*** *Peritoneal inflammation, tissue necrosis, adhesions formation, nerve irritation due to deep penetration, release of local inflammatory mediators and/or endometrioma formation.*
- **Endometriosis is a disease of contrast**. It is a benign disease but it is locally invasive, disseminates widely and proliferates in the lymph nodes. **Minimal disease** may have severe pain whereas large endometriosis may remain asymptomatic. Cyclic hormones stimulates growth whereas continuous hormones suppress it.
- ***Endometriotic tissues*** *produce estrogen and many inflammatory cytokines locally. Estrogen causes proliferation and growth of endometriotic tissues. Whereas the cytokines and prostaglandins are the causes of pain and the infertility. The endometriotic tissues are resistant to progesterone.*
- ***Endometriotic lesions*** *may appear as red, brown, black, yellow, pink or in the form of vesicles, or subovarian adhesions. Red and the blood filled lesions are the most active.*
- **Abdominal scar** is the most common site of endometriosis following hysterectomy, hysterotomy, cesarean section, tubectomy or myomectomy **(Fig. 22.6)**.
- **The disease is full of theories** and no one single theory can explain endometriosis at all sites. Genetic basis and defect of local cellular immunity may be implicated **(Table 22.3)**. In spite of dense adhesions amongst the pelvic structures, the fallopian tubes are usually patent. In pelvic endometriosis, typically there are small black dots—'powder burns' or 'gunshot' seen on uterosacral ligaments and pouch of Douglas. Other lesions are flame-shaped polypoidal, hemorrhagic and white patches.
- **Etiopathogenesis** of endometriosis is not well understood. Factors involved are genetic mutations, immunological, molecular defects, mechanical factors and environmental toxins (dioxins).
- **Symptoms:** About 25% have got no symptom. **Symptoms** are not related to the extent of lesion. Severity of endometriosis and the degree of pelvic pain are not always proportional. Unlike primary dysmenorrhea, the pain lasts for many days before and after the menstruation.
- ***Pain from the ovary****, outer 2/3rd of the fallopian tube and upper ureter are carried via thoracic sympathetics, celiac and superior mesenteric plexus to the T_9–T_{10} spinal segments.*

Contd...

DC Dutta's Textbook of Gynecology

Salient Features

- Presentation is simple, lucid and concise.
- Core knowledge (must-know area) has been highlighted.
- Enriched with numerous flowcharts, tables, boxes, sketches, drawings and imaging studies.
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- Equally informative to Nursing, Homeopathy and Ayurvedic students as well as for general practitioners and practicing gynecologists.

Reviews

Dutta's Textbook of Gynecology is indeed a formidable work. It is beautifully illustrated with comprehensive coverage in a concise manner. It is an immensely useful study resource for all.
—S Ponda, *Professor & head; AIIMS, Kalyani, India*

The text is superbly written and illustrated with diagrams, graphs, images, sketches and beautiful photographs. The presentation is lucid, and easy to read and understand. It is of immense benefit to the students both the nursing and midwifery.
—Michael P O 'Connel *MD*
Consultant, Coombe Women and Infants University Hospital, Dublin, Ireland

Dutta's Textbooks come out at a regular interval enriched with all the progress of research and knowledge. Ninth edition of Gynecology is superbly written. It is profusely illustrated, for simple understanding. This book has been the regular companion of all the medical and nursing students, residents and the practitioners.
—Himadri Bal *MD, Professor, Dr DY Patil Medical College, Pune, Maharashtra, India*

Dutta's Textbook of Gynecology needs special appreciation for the presentation with its standardized headings, subheadings, flowcharts, boxes and the key points. It should be the regular companion of all the medical students, residents and the clinicians.
—Jaya Chaturbedi*, Professor, AIIMS, Rishikesh, India*

The ninth edition of Dutta's Textbook of Gynecology with total 38 chapters covers the entire course in gynecology. All the chapters are updated, focused and concise. The changes in the present edition are significant as it is structured on the curriculum as designed by the National Medical Commission (NMC). The Chapter 38 is a special attraction due to its huge practical importance. Dutta's Textbook of Gynecology, authored by Professor Hiralal Konar, should be the companion of all the medical students, residents, nursing students and the practitioners.
—The Journal of Obstetrics and Gynaecology of India

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