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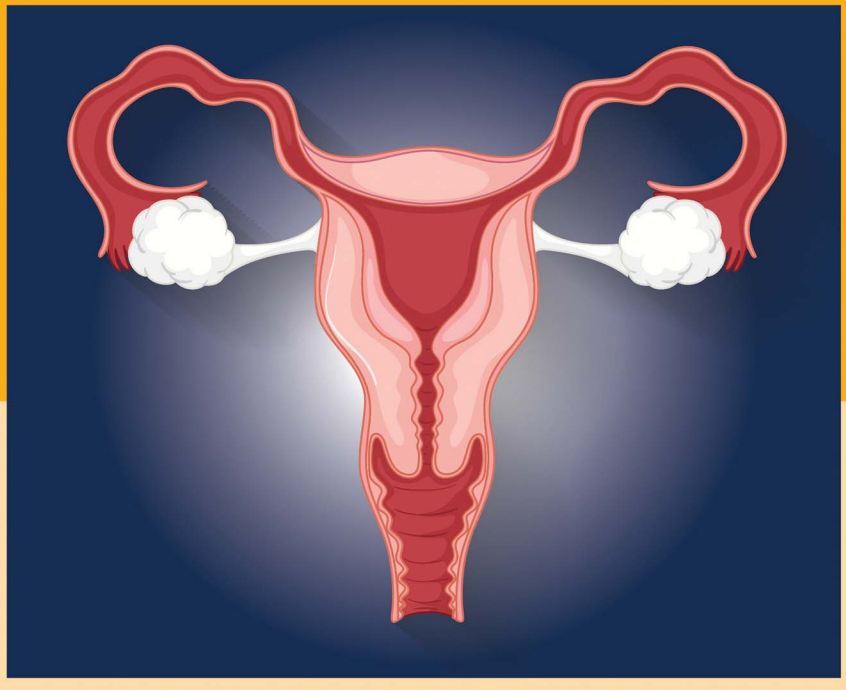



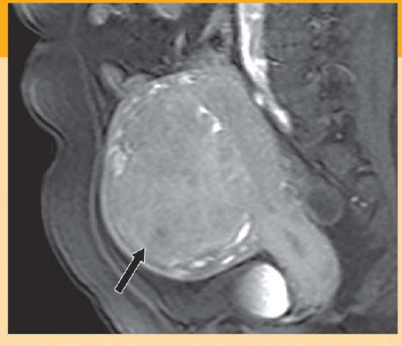




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# Contents


**1 Anatomy of the Female Pelvic Organs** 1

External Genital Organs (Syn: Vulva, Pudendum) 1; Mons Veneris (Mons Pubis) 1; Labia Majora 1; Labia Minora 1; Clitoris 1; Vestibule 1; Bartholin's Gland 2; Perineum 2; Blood Supply of the Vulva 2; Nerve Supply of the Vulva 2; Internal Genital Organs 2; Vagina 3; Uterus 6; Fallopian Tube (Syn: Uterine Tube) 8; Ovary 9; Female Urethra 10; Other Internal Organs 12; Urinary Bladder 12; Pelvic Ureter 12; Rectum 13; Anal Canal 13; Pelvic Muscles 14; Pelvic Floor (Syn: Pelvic Diaphragm) 14; Perineum 16; Anatomical Perineum 16; Obstetrical Perineum (Syn: Perineal Body, Central Point of Perineum) 17; Pelvic Peritoneum 17; Broad Ligament 17; Pelvic Fascia and Cellular Tissue 18; Pelvic Fascia 18; Pelvic Cellular Tissue 18; Mackenrodt's Ligaments (Syn: Cardinal Ligament, Transverse Cervical) 18; Uterosacral Ligaments 18; Pubocervical Fascia (Bladder Pillar) 18; Round Ligaments 19; Ovarian Ligaments 20; Extraperitoneal Surgical Spaces 20

**2 Blood Vessels, Lymphatic Drainage and Innervation of Pelvic Organs** 22

Pelvic Blood Vessels 22; Arterial Supply 22; Pelvic Veins 24; Pelvic Lymphatics 24; Inguinal Nodes 24; Parametrial Node 24; External Iliac Nodes 25; Sacral Group 25; Subaortic and Aortic Nodes 25; Lymphatics of the Body of the Uterus 25; Lymphatics of the Cervix 25; Lymphatics from the Fallopian Tube and Ovary 25; Lymphatics of the Vagina 26; Lymphatics of the Vulva 26; Lymphatics of Bladder and Urethra 26; Pelvic Nerves 26; Somatic 26; Autonomic 27; Ovarian Plexus 27

**3 Development of Genital Organs and Gonads** 29

Development of External Genital Organs 29; Development of Internal Genital Organs 30; The Genital Duct System 30; Fallopian Tubes 31; Uterus 31; Broad Ligaments 32; Vagina 32; Development of the Ovary 33; Site 33; Sources 33; Bipotential or Primitive Gonad 33; Definitive Gonad 33; Descent of the Ovary 35; Developmental Anomalies of Female Reproductive Organs 35;

**4 Congenital Malformation of Female Genital Organs** 36

Developmental Anomalies of the External Genitalia 36; Perineal or Vestibular Anus 36; Ectopic Ureter 36; Vaginal Abnormalities 36; Narrow Introitus 36; Hymenal Abnormalities 36; Vaginal Maldevelopments 38; Partial Agenesis of Upper Vagina 39; Associated Abnormalities 40; ASRM Mullerian Anomalies Classification 2021 (ASRM MAC-2021) 40; Abnormalities of the Fallopian Tubes 44; Anomalies of the Ovaries 44; Wolffian Remnant Abnormalities 44; Parovarian Cyst 44; Other Abnormalities 44

**5 Puberty—Normal and Abnormal** 46

Puberty 46; Definition 46; Morphological Changes 46; Endocrinology in Puberty 46; Common Disorders of Puberty 47; Precocious Puberty 48; Delayed Puberty 50; Heavy Menstrual Bleeding in Puberty (Menorrhagia) 51

**6 Menopause and Care of Mature Women** 54

Definition 54; Age of Menopause 54; Clinical Importance 54; Endocrinology of Menopausal Transition and Menopause 54; Hypothalamo-Pituitary Gonadal Axis 54; Menopause Transition 55; Supportive Criteria for the Stages 55; Estrogens 55; Androgens 55; Progesterone 55; Gonadotropins 55; Organ Changes and Symptoms of Menopause 55; Bone Metabolism 56; Cardiovascular System 56; Menstruation Pattern Prior to Menopause 56; Menopausal Symptoms 56; Brain Functions and the Role of Estrogen 57; Diagnosis of Menopause 59; Management 59; Prevention 59; Treatment 59; Abnormal Menopause 62


JAYPEE BROTHERS

**7 Neuroendocrinology in Relation to Reproduction** 64

■ Hypothalamus 64; • Connections 64; • Secretions 65; • Control of GNRH Secretion 66; ■ Pituitary Gland 67; • Anterior Pituitary 68; • Posterior Pituitary 69; ■ Ovary 70; • Ovarian Steroidogenesis 70; • Anti-Müllerian Hormone 73; ■ Thyroid Glands 74; ■ Adrenal Glands 74; • Control of Cortical Secretions 74; ■ Hypothalamo-Pituitary-Ovarian Axis 74; • Feedback Loop 75; • Fetal Life 75; • Infancy and Childhood 75; • Prepuberty and Puberty 75; ■ Ovarian and Menstrual Cycle 75; • Following Delivery 75; • Menopausal Transition Period 76

**8 Menstruation** 78

• Definition 78; • Anatomical Aspect 78; ■ Follicular Growth, Maturation and Atresia 78; • GERM Cells 78; • Primordial Follicle 79; • Menstrual Cycle 80; • Follicular Atresia 84; ■ Endometrial Cycle 84; • Basal Zone 84; • Functional Zone 85; • Mechanism of Menstrual Bleeding 87; • Role of Prostaglandins 87; • Luteal–Follicular Shift 88; ■ Menstrual Symptoms 89; • Menstrual Hygiene 89; • Anovular Menstruation 89; • Artificial Postponement of Menstruation 91

**9 Examination of a Gynecological Patient and the Diagnostic Procedures** 93

• History 93; ■ Clinical Examination 94; • General and Systemic Examination 94; • Gynecological Examination 94; ■ Diagnostic Procedures 100; • Blood Values 100; • Urine 100; ■ Cervical and Vaginal Smear for Exfoliative Cytology 102; • Examination of Cervical Mucus 105; • Colposcopy 106; • Endometrial Sampling 107; • Culdocentesis 108

**10 Imaging Techniques, Other Diagnostic Procedures and Laser in Gynecology** 110

■ Imaging Techniques in Gynecology 110; • X-ray 110; • Ultrasonography 111; • Uterine Artery Embolization 113; • Computed Tomography (CT Scan) 114; • Magnetic Resonance Imaging 115; • Positron Emission Tomography 116; • Interventional Procedures Under Radiologic Guidance 116; ■ Other Diagnostic Procedures in Gynecology 116; • Endoscopy in Gynecology 116; • Laparoscopy 116; • Hysteroscopy 117; • Salpingoscopy 117; • Examination Under Anesthesia 117; • Urodynamic Study 117; ■ Laser in Gynecology 118; • Physics of Laser 118; • Uses of Laser in Gynecology 118; • Advantages of Laser 118; • Limitations of Laser 118; • Hazards of Laser Systems 118

**11 Pelvic Infection** 120

• Defense of the Genital Tract 120; ■ Acute Pelvic Infections 121; • Pelvic Inflammatory Disease 121; • Pelvic Infection Following Delivery and Abortion 126; • Pelvic Infection Following Gynecological Procedures 128; • IUCD and Pelvic Infection 128; • Chronic Pelvic Infection 128; ■ Genital Tuberculosis 129; • Incidence 129; • Pathogenesis 129; • Mode of Spread 129; • Pathology of Pelvic Organs 129; • Clinical Features 131; • Investigations 131; • Diagnosis 133; • Treatment 133; • Results of Treatment 134; ■ Vulvar Tuberculosis 135; ■ Cervical Tuberculosis 135; ■ Actinomyces Infection 135

**12 Sexually Transmitted Infections** 137

• Gonorrhea 137; • Syphilis 139; • Chlamydial Infections 140; • Chancroid (Soft Sore) 141; • Lymphogranuloma Venereum 141; • Granuloma Inguinale (Donovanosis) 142; • Bacterial Vaginosis 142; • Herpes Genitalis 143; • Human Immunodeficiency Virus Infection and Acquired Immunodeficiency Syndrome 144; • Genital Warts (Condyloma Acuminata) 147; • Molluscum Contagiosum 147; • Pediculosis Pubis 147; • Scabies 148

**13 Infections of the Individual Pelvic Organ** 149

■ Vulvar Infection 149; • Vulvitis due to Specific Infection 149; ■ Infections of Bartholin's Gland 149; • Bartholin's Abscess 150; • Bartholin's Cyst 150; ■ Vaginal Infection (Vaginitis) 151; • Vulvovaginitis in Childhood 151; • Trichomonas Vaginal Infection 151; • Candida Vaginitis (Moniliasis) 153; • Atrophic


JAYPEE BROTHERS

Vaginitis (Senile Vaginitis) 154; ■ Toxic Shock Syndrome 154; ■ Cervicitis 155; • Acute Cervicitis 155; • Chronic Cervicitis 155; ■ Endometritis 155; • Acute Endometritis 156; • Chronic Endometritis 156; • Atrophic Endometritis (Senile Endometritis) 156; ■ Pyometra 156; ■ Salpingitis 157; • Acute Salpingitis 157; • Chronic Salpingitis 158; ■ Oophoritis 160; ■ Parametritis 160; • Clinical Features 160; • Treatment 160; ■ Pelvic Abscess 161; • Etiology 161; • Clinical Features 161; • Investigations 161; • Treatment 161; ■ Syndromic Management of Sexually Transmitted Infections (WHO – 1991) 161; • Principle 161; • Method 162; • Advantages of Syndromic Management 162; • Limitations 162; ■ Other Gynecologic Infections 162; • Postoperative Infections 162

**14 Dysmenorrhea and Other Disorders of Menstrual Cycles** 165

■ Dysmenorrhea 165; • Primary Dysmenorrhea (Spasmodic) 165; • Secondary Dysmenorrhea (Congestive) 168; • Pelvic Congestion Syndrome 168; ■ Other Disorders to Cause Menstrual Pain 169; • Mittelschmerz's Syndrome (Ovulation Pain) 169; • Premenstrual Syndrome and Premenstrual Dysphoric Disorder (Syn: Premenstrual Tension) 169; • Menstrual Migraine 171

**15 Abnormal Uterine Bleeding** 173

■ Common Causes of Abnormal Uterine Bleeding 173; • PALM–COEIN (FIGO – 2011) 173; ■ Patterns of Abnormal Uterine Bleeding 173; • Frequent Bleeding (Polymenorrhea) 174; • Intermenstrual Bleeding (Metrorrhagia) 174; • Infrequent Menstruation (Oligomenorrhea) 174; • Light Menstruation (Hypomenorrhea) 175; • AUB-O (Dysfunctional Uterine Bleeding) 175; • Differential Diagnosis of Abnormal Uterine Bleeding 175; • Pathophysiology of AUB-E 176; • Diagnostic Approach for Women with AUB 177; • Management 179; • Management Options for a Case with AUB 184

**16 Pelvic Organ Prolapse and Other Displacements of the Uterus** 186

■ Pelvic Organ Prolapse 186; • Supports of Uterus 186; • Supports of Vagina 187; • Etiology and High Risk Factors for Pelvic Organ Prolapse 188; • Clinical Types of Pelvic Organ Prolapse 189; • Morbid Changes 192; • Symptoms 192; • Pelvic Organ Prolapse Quantification 192; • Clinical Examination and Diagnosis of POP 193; • Differential Diagnosis 195; • Management of Prolapse 196; • Surgical Management of Prolapse 196; • Vaginal Hysterectomy with Pelvic Floor Repair 202; • Vault Prolapse 204; • Cervicopexy or Sling Operation (Purandare's Operation) 205; • Shirodker's Operation 205; • Khanna's Sling Operation 206; • Management of POP Using Mesh 206; • Complications of Vaginal Repair Operations 206; • Complications of Vaginal Hysterectomy with PFR 207; • Chronic Inversion of the Uterus 207; • Retroversion of the Uterus 208

**17 Infertility** 212

• Definition 212; • Incidence 212; • Physiological Consideration 212; • Causes of Infertility 212; • Causes of Female Infertility (FIGO) 214; • Investigations of Infertility 215; • Treatment of Infertility 223; ■ Artificial Insemination 231; • Intrauterine Insemination 231; • Artificial (Therapeutic) Insemination Donor 231; • Fallopian Tube Sperm Perfusion 232; ■ Assisted Reproductive Technologies 232; • In Vitro Fertilization and Embryo Transfer 232; • Gamete Intrafallopian Transfer 235; ■ Micromanipulation 235; • Intracytoplasmic Sperm Injection 235; • Embryo or Oocyte Donation 236; • Gestational Carrier Surrogacy 236; • Prognosis of Infertility 237

**18 Benign Lesions of the Vulva and Vagina** 240

■ Vulvar Non-neoplastic Epithelial Disorders 240; • Terminology, Definition and Types 240; • Etiology 240; • Diagnosis of Vulvar Epithelial Disorders 240; • Lichen Sclerosus 241; • Lichen Planus 241; • Extramammary Paget's Disease 242; • Vulvar Crohn's Disease 242; • Psoriasis 242; • Relation with Malignancy 242; ■ Vulvar Ulcers 243; • Vulvar Manifestations of Systemic Diseases 244; ■ Miscellaneous Swellings 244; • Vulvar Cysts 244; ■ Benign Tumors of the Vulva 244; • Vulvar Endometriosis 245; ■ Vaginal Wall Cysts 245; • Gartner's Cyst 245; • Inclusion Cyst 245; ■ Vulvar Pain Syndrome 245; • Vulvodynia 245

JAYPEE BROTHERS

19 **Ben**ign **Lesi**ons **o**f **t**he **Cer**vix 248

• Cervical Ectopy (Erosion) 248; • Eversion (Ectropion) 249; • Cervical Tear 250; • Cervical Cysts 250; • Elongation of the Cervix 251

20 **Ben**ign **Lesi**ons **o**f **t**he **Ute**rus 253

■ Leiomyomas 253; • Incidence 253; • Histogenesis 253; • Types 254; ■ Body or Corporeal Fibroids 255; • Pathology 255; ■ Secondary Changes in Fibroids 256; • Degenerations 256; • Clinical Features 257; • Complications 258; • Investigations 258; • Differential Diagnosis 259; ■ Management of Fibroid Uterus 259; • Asymptomatic Leiomyomas (75%) 259; • Symptomatic Leiomyomas 260; ■ Cervical Fibroid 264; • Symptoms 264; • Treatment 265; ■ Uterine Polyps 265; • Endometrial Polyps 265; • Management 265; • Fibroid Polyp 266

21 **Ben**ign **Neopl**asms **o**f **t**he **Ova**ry 269

■ Normal Ovary 269; ■ Ovarian Enlargement 269; • Non-neoplastic 269; • Benign Ovarian Neoplasms 271; ■ Borderline Epithelial Tumors of the Ovary 281; ■ Parovarian Cyst 282

22 **Endomet**riosis **a**nd **Adenom**yosis 284

■ Endometriosis 284; • Definition 284; • Prevalence 284; • Sites 284; • Pathogenesis 285; • Pathology 286; • Clinical Features of Pelvic Endometriosis 287; • Diagnosis 289; • Differential Diagnosis 289; • Complications of Endometriosis 290; • Staging 290; • Treatment of Endometriosis 292; • Treatment Options for Pelvic Endometriosis 292; • Surgical Management of Endometriosis 295; • Endometriosis at Special Sites 296; ■ Adenomyosis 297; • Definition 297; • Causes 297; • Pathogenesis 297; • Pathology 298; • Clinical Features 298; • Treatment 299

23 **Premal**ignant **Lesi**ons 302

■ Premalignant Vulvar Lesions 302; • Vulvar Intraepithelial Neoplasia 302; • Paget's Disease 303; ■ Vaginal Intraepithelial Neoplasia 303; ■ Cervical Intraepithelial Neoplasia 304; • Nomenclature 304; • Pathogenesis 305; • Epidemiology 305; • Diagnosis of CIN 307; • Treatment of CIN 309; ■ Premalignant Endometrial Lesion 312

24 **Geni**tal **Malig**nancy 316

• General Considerations 316; ■ Vulvar Carcinoma 316; • Incidence 316; • Etiology 316; • Pathology 316; • Spread 317; • Clinical Features 318; • Differential Diagnosis 318; • Staging 318; • Causes of Death 318; • Management 318; • Prognosis 319; • Melanoma 319; • Bartholin's Gland Carcinoma 320; ■ Vaginal Carcinoma 320; • Primary Vaginal Carcinoma 320; • Secondary Carcinoma of the Vagina 321; ■ Carcinoma Cervix 322; • Magnitude of the Problem 322; • Epidemiology 322; • Gross Pathology 322; • Mode of Spread 323; • Staging 323; • Prognosis 324; • Surgical Staging of Cancer Cervix 326; • Diagnosis 326; • Patient Profile 326; • Differential Diagnosis 327; • Complications 327; • Causes of Death 327; • Management of Carcinoma Cervix 327; • Treatment Modalities of Carcinoma Cervix 328; • Planning of Treatment Modalities 332; • Comprehensive Palliative Care 332; • Carcinoma of Cervix and Pregnancy 333; • Results of Therapy for Carcinoma Cervix 333; ■ Endometrial Carcinoma 334; • Incidence 334; • Etiology 334; • Pathology 335; • Molecular Classification of Endometrial Carcinoma and the Prognosis 335; • Spread 336; • Clinical Features 337; • Diagnosis of Endometrial Carcinoma 337; • Surgical Staging 338; • Prediction of Prognosis for Endometrial Cancer 339; • Management of Endometrial Carcinoma 339; • Treatment Modalities of Carcinoma Endometrium 340; • Recurrent Disease 341; ■ Gestational Trophoblastic Disease 343; • Risk Factors for Development of GTN 343; • Gestational Trophoblastic Neoplasia 343; • Placental Site Trophoblastic Tumor 343; • Invasive Moles (Chorioadenoma Destruens) 344; • Choriocarcinoma 344; • Spread of GTN 345; • Clinical Features of GTN 345; • Special Investigations 345; • Diagnosis of Metastases 346; • Staging 346; • Management of GTN 347; • Surveillance During and After Therapy

JAYPEE BROTHERS

of GTN 348; ■ Malignant Tumors of the Ovary 350; • Incidence 350; • Epidemiology and Etiological Factors 350; • Genetics and Ovarian Malignancy 350; • Familial Cancer 351; • Categories of Ovarian Malignancy 351; • General Considerations 351; • Primary Epithelial 351; • FIGO Staging of Carcinoma Ovary 352; • Spread 353; • Clinicopathologic Explanation 354; • Clinical Features 355; • Special Investigations 356; • Diagnosis 356; • Management of Epithelial Ovarian Cancer 357; • Treatment of Malignant Ovarian Tumor 358; • Adjuvant Chemotherapy 359; • Prognostic Factors in Ovarian Malignancy 361; • Prevention of Ovarian Cancer 362; • Primary Peritoneal Carcinoma 362; ■ Germ Cell Tumors of the Ovary 362; • Teratoma 363; • Dysgerminoma 363; • Endodermal Sinus Tumor (Yolk Sac Tumor) 364; • Nongestational Ovarian Choriocarcinoma 364; • Mixed Germ Cell Tumors 364; • Sex Cord Stromal Tumors 365; • Granulosa Cell Tumors 365; • Thecoma-Fibroma Group 366; • Sertoli-Leydig Cell Tumor (Androblastoma, Arrhenoblastoma) 366; • Gynandroblastoma 367; ■ Metastatic Tumors of the Ovary 367; ■ Fallopian Tube Carcinoma and Peritoneal Cancer 369; • Primary Fallopian Tube Carcinoma 369; • Secondary Fallopian Tube Carcinoma 370; ■ Sarcoma Uterus 370; • Classification of Uterine Sarcomas 370; • Microscopic Appearance 371; • Spread 371; • Diagnosis 372; • Treatment 372; • Prognosis 372; • Sarcoma Botryoides (Embryonal Rhabdomyosarcoma) 372

**25 Urinary Problems in Gynecology** 374

■ Anatomy of Vesicourethral Unit 374; • Bladder 374; • Urethra 375; • Support to Bladder Neck and Urethra 375; • Nerve Supply of the Vesicourethral Unit 375; ■ Physiology of Micturition 376; • Storage Phase 376; • Voiding Phase 377; • Urinary Continence 377; • Urinary Incontinence 378; • Urge Incontinence 385; • Bladder Pain Syndrome 388; • Overflow Incontinence 388; • Voiding Disorder 388; • Urinary Tract Infections 389; • Urethral Diverticulum 391; • Urethral Prolapse 391; ■ Urethral Caruncle 391; • Definition 391; • Pathogenesis 392; • Clinical Features 392; • Treatment 392

**26 Genitourinary Fistula** 394

■ Genitourinary Fistula 394; • Definition 394; ■ Vesicovaginal Fistula 394; • Definition 394; • Causes 394; • Types 395; • Clinical Features 396; • Treatment of VVF 397; ■ Urethrovaginal Fistula 400; • Causes 400; • Diagnosis 400; • Treatment 400; ■ Injury to the Ureter During Gynecological Surgery 400; • Causes 400; • Diagnosis 401; • Management of Ureteric Injury 401; • Principles of Ureteric Repair 402; • Ureteric Transection 402; ■ Rectovaginal Fistula 402; • Definition 402; • Diagnosis 403; • Investigations 403; • Treatment 403

**27 Genital Tract Injuries and Anorectal Dysfunctions** 405

■ Complete Perineal Tear 405; • Definition 405; • Etiology 405; • Clinical Features 406; • Differential Diagnosis 406; • Treatment 407; ■ Coital Injuries 409; • Management 409; ■ Sexual Assault and Rape Victims 409; ■ Rape Victims 409; • Forensic Considerations 409; • Management 410; ■ Direct Trauma and Injury to Genital Organs 410; • Management 410; ■ Foreign Bodies 411; • Effects 411; • Management 411; ■ Functional Anorectal Dysfunctions 411; • Fecal Incontinence 411

**28 Disorders of Sexual Development** 414

• Definition 414; • Embryological Considerations 414; • Determination of Sex 414; • Nomenclature 414; ■ 46, XX Disorders of Sexual Development 414; • Female Pseudohermaphroditism 414; ■ 45, XO Sex Chromosome DSD 416; • Gonadal Dysgenesis (45, XO DSD) 416; • Turner's Syndrome 416; • Klinefelter's Syndrome (47, XXY DSD) 417; ■ 46, XY Disorder of Sexual Development 418; • Androgen Insensitivity Syndrome (Testicular Feminization) 418; ■ 46, XY DSD (Swyer Syndrome) 419; • Complete Gonadal Dysgenesis 419; ■ True Hermaphroditism 419; • Ovotesticular Disorders (46, XY DSD) 419; ■ Diagnosis of DSD 419; • Diagnosis of Ambiguous Genitalia 420; ■ Management of DSD 420; • Basic Guidelines 420

JAYPEE BROTHERS

**29 Amenorrhea** 423

• Definition 423; • Clinical Types 423; ■ Primary Amenorrhea 424; • Definition 424; • Investigations 425; • Management 428; ■ Secondary Amenorrhea 430; • Definition 430; • Etiology 430; • Common Causes 430; ◆ Clinical Features and Diagnosis of Secondary Amenorrhea 432; ■ Uterine Factors 432; • Tubercular Endometritis 432; • Uterine Synechiae (Syn: Asherman's Syndrome) 432; ■ Ovarian Factors 432; • Polycystic Ovary Syndrome 432; • Premature Ovarian Insufficiency 438; • Masculinizing Ovarian Tumor 439; ■ Hypothalamic Amenorrhea 439; • Weight Related Amenorrhea 439; • Anorexia Nervosa 439; • Obesity 439; • Kallmann's Syndrome 439; ■ Pituitary Factors 440; • Adenoma 440; • Sheehan's Syndrome 441; ■ Adrenal Factors 441; • Adrenal Tumor or Hyperplasia 441; • Cushing's Syndrome 442; ■ Thyroid Dysfunction 442; ■ Postpill Amenorrhea 442; ■ General Disease 444; ■ Diagnostic Evaluations of Secondary Amenorrhea 444; • Detailed History 444; • General Examination 444; ■ Management of Secondary Amenorrhea 445; • No Abnormality Detected 445; • Cases with Detectable Cause 445; • Hyperprolactinemia, Inappropriate Galactorrhea 445; • Premature Ovarian Insufficiency 445; • Adrenal Disorders 445;

**30 Population Dynamics and Control of Conception** 448

■ Control of Conception 448; • Family Planning 448; • Contraception 449; ■ Methods of Contraceptions 449; • Temporary Methods 450; • Intrauterine Contraceptive Devices 450; ■ Steroidal Contraceptions 457; • Combined Hormonal Contraceptives 457; • Centchroman (Saheli) 462; • Progestogen-Only Contraceptions 462; • Injectable Progestins 462; • Implant 463; • Emergency Contraception (Syn: Postcoital Contraception) 464; • Summary of Oral Contraceptives 465; ■ Drug Interaction with Hormonal Contraception 466; ■ Sterilization 466; • Presterilization Counseling 466; • Male Sterilization 467; • Female Sterilization 468; ■ Barrier Methods 472; ■ Types of Barrier Methods 472; • Condom (Male) 472; • Female Condom (Femidom) 472; • Diaphragm 472; • Vaginal Contraceptives 473; • Fertility Awareness Method 473; ■ Contraceptive Counseling and Prescription 474; • Prescription 474; ■ Ongoing Trials and Selective Availability 475; • Transcervical Sterilization 476

**31 Basic Principles of Radiation Therapy, Chemotherapy, Immunotherapy and Gene Therapy in Gynecology** 477

■ Radiation Therapy 477; • Radiobiology of Normal Tissues 477; • Techniques of Radiation Therapy 479; • Measurement of Radiation 480; • Radiopotentiators and Hypoxic Cell Sensitizers 481; • New Technology for Radiation Therapy 482; ■ Chemotherapy 483; • General Considerations 483; • Principles of Chemotherapy and Guidelines 483; • Classification of Cytotoxic Drugs 484; ■ Immunotherapy 490; • Cytokines and Cancer Therapy 490; • Principles of Immunotherapy 490; • Modulation of Immune System (Biological Response Modifiers) 490; ■ Genetics and Gynecologic Malignancy 490; • Tumor Markers 491

**32 Hormones in Gynecological Practice** 494

■ Nomenclatures 494; • Cellular Function 494; ■ Hypothalamic Hormones 494; • GnRH 494; • Gonadotropins 496; • Antigonadotropins 499; ■ Gonadal Hormones 500; • Mechanism of Action 500; • Estrogens 500; • Antiestrogen 502; • Progesterone 503; • Combined Preparations (Estrogen and Progestogen) 505; • Antiprogesterone 505; • Androgens 506; ■ Adrenocortical Hormones 507; • Diagnostic 507; • Therapeutic 507; ■ Thyroid Hormone 507

**33 Gynecological Problems from Birth to Adolescence** 510

• Gynecological Examination in a Child 510; • Neonates, Toddlers and Infants 511; • Premenarcheal 512; • Postmenarcheal to Adolescence 514

**34 Special Topics** 519

■ Abnormal Vaginal Discharge 519; • Introduction 519; • Leukorrhea 519; • Pruritus Vulvae 521; ■ Pelvic Pain 522; • Neurophysiology of Pain 522; • Acute Pelvic Pain 522; • Chronic Pelvic Pain 523; • Retention (Trapped) Ovarian Syndrome 526; ■ Ovarian Remnant Syndrome 526; ■ Postmenopausal


JAYPEE BROTHERS

Bleeding 526; • Causes 526; • Investigations 526; ■ Low Backache 527; • Causes of Low Backache 528; ■ Breast Diseases in Gynecology 528; • Anatomy of the Adult Breast 528; • Anatomical Defects 529; • Benign Breast Disorders 530; • Evaluation of a Breast Lump 531; • Nipple Discharge 533; ■ Emotional, Psychosexual Issues and Female Sexual Dysfunction 534; • Sexual Function Disorders 534; • Intimate Partner Violence 535; • Abdominopelvic Lump 536; • Adnexal Mass 536; ■ Androgen Excess in Women 538; • Nomenclature 538; • Androgen Sources in Female 539; • Androgens and Pilosebaceous Unit 540; • Mechanism of Excessive Hair Growth 540; • Pathophysiology of Androgen Excess 540; • Causes of Androgen Excess 541; • Investigations 542; • Management Principles of Androgen Excess 543; ■ Galactorrhea 545; • Treatment 545

35 **Opera**tive **Gynec**ology 547

■ Preoperative Preparations 547; • Investigations 547; • Preoperative Counseling and Informed Consent 548; ■ Intraoperative Care 549; • Incisions 549; • Drains 550; • Closure of Peritoneum 550; ■ Postoperative Care 550; • Aims of Postoperative Care 550; • Return from Operation Theater 550; • In the Ward 550; • Discharge 551; ■ Gynecological Operations 551; • Dilatation of Cervix 552; • Dilatation and Curettage 552; • Dilatation and Insufflation (D and I) 553; • Hysterosalpingography 553; • Cervical Biopsy 554; • Thermal Cauterization 555; • Cryosurgery 556; • Perineoplasty 556; • Amputation of Cervix 557; ■ Major Surgical Operations 557; • Abdominal Hysterectomy 557; ■ Postoperative Complications and Care 561; • Complications of Hysterectomy 561; • Complications of Vaginal Hysterectomy and Other Operations 562; ■ Operations on the Ovary 562; • Ovarian Cystectomy 562; • Ovariotomy 563; • Wedge Resection 563; • Laparoscopic Ovarian Drilling 564; • Ovarian Biopsy 564; • Salpingectomy 564; • Salpingo-oophorectomy 564; • Broad Ligament Cyst or Leiomyoma 564; ■ Operations for Inversion of the Uterus 565; • Haultain's Operation 565; • Kustner's Operation 565; ■ Ventrosuspension Operation 565; ■ Abdominal Myomectomy 566; • Measures to Control Blood Loss During Myomectomy 566; • Actual Steps 566; • Cervical Fibroid 566; • Broad Ligament Myoma 566; • Complications of Myomectomy 566; ■ Radical Hysterectomy (Syn: Class IV Radical Hysterectomy) 567; • Principal Steps of the Operation 567; ■ Operations on the Vulva 570; • Simple (Conservative) Vulvectomy 570; ■ Radical Vulvectomy with Bilateral Inguinofemoral Lymphadenectomy 570; • Principal Steps 571;

36 **Endos**copic **Surg**ery **in** **Gynec**ology 573

• History 573; • Advantages of Laparoscopic Surgery 574; ■ Basic Instruments and Electrosurgical Units for Laparoscopic Surgery 574; • Accessory Instruments 574; • Hemostasis During Laparoscopic Surgery 576; • Indications of Laparoscopic Surgery 578; • Contraindications 578; • Operative Procedures for Laparoscopy 578; ■ Surgical Techniques 579; ■ Operative Laparoscopy 580; • Procedure of Laparoscopic Hysterectomy 581; • Complications of Laparoscopy 581; ■ Robotic Surgery in Gynecology 582; • Technology of Robotic Surgery 583; ■ Hysteroscopy 583; • Anesthesia and the Procedures 584; • Indications of Diagnostic Hysteroscopy 585; • Indications of Operative Hysteroscopy 585; • Levels of Hysteroscopic Procedures 586; • Contraindications of Hysteroscopy 586; • Operative Hysteroscopy 586; • Hysteroscopic Myomectomy 586; • Complications of Hysteroscopy 586

37 **Curr**ent **Top**ics **in** **Gynec**ology 588

• Stem Cells and Therapies in Gynecology 588; • Use of Embryonic Stem Cells in Regenerative Medicine 588

38 **Pract**ical **Gynec**ology 590

■ 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;

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• 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

***Index*** ***635***

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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)**

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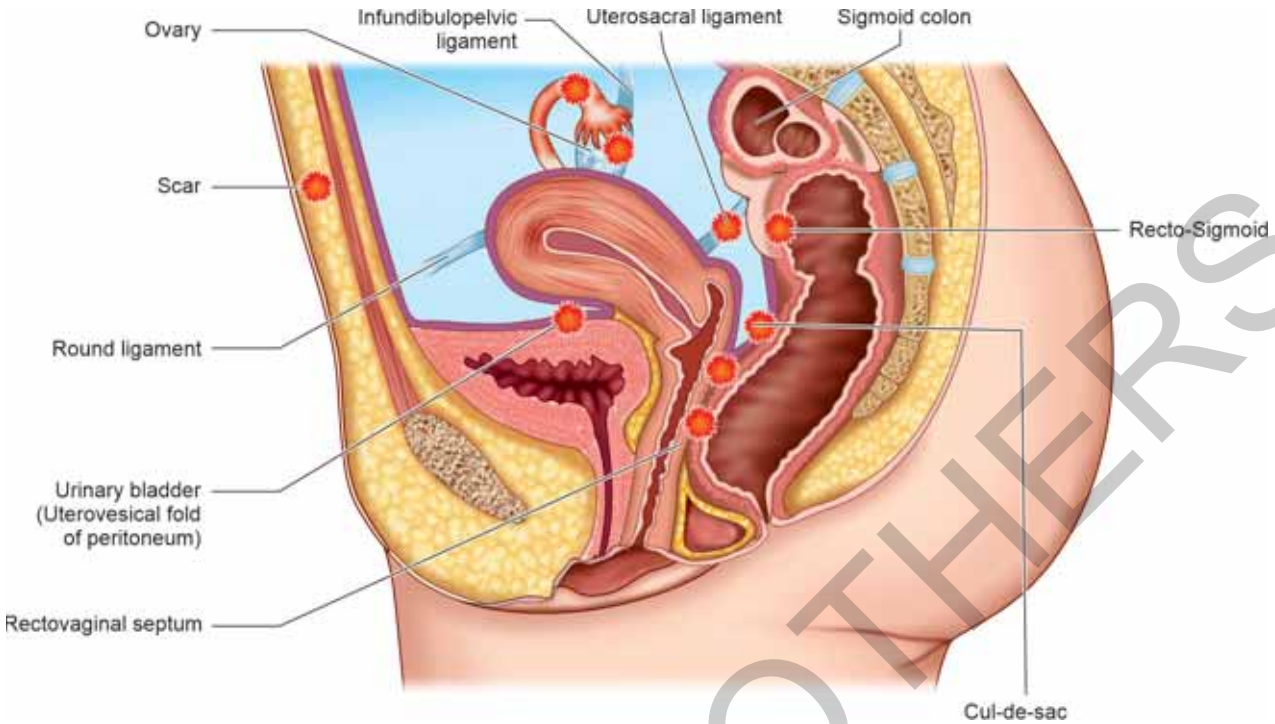


**Fig. 22.1:** Common sites of endometriosis.

**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<br>■ Rectovaginal septum<br>■ Umbilicus | ■ Coelomic metaplasia |
| ■ Abdominal scar<br>■ Episiotomy scar<br>■ Vagina, cervix | ■ Direct implantation |
| ■ Lymph nodes | ■ Lymphatic spread |
| ■ Distant sites (lungs, pleura, skin, lymph nodes, nerves) | ■ Vascular spread<br>■ Genetic<br>■ 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

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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.

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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 $\beta_2$ 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.

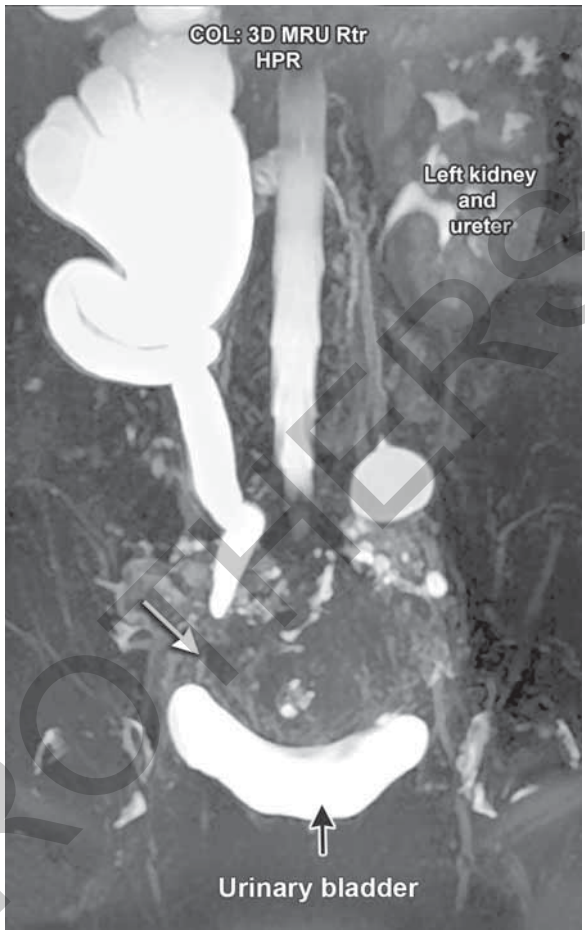


**Fig. 22.4:** Right ureter showing gross hydroureteric and hydronephrotic changes in a case with advanced (stage IV) deep infiltrating endometriosis (See case history).

- **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.

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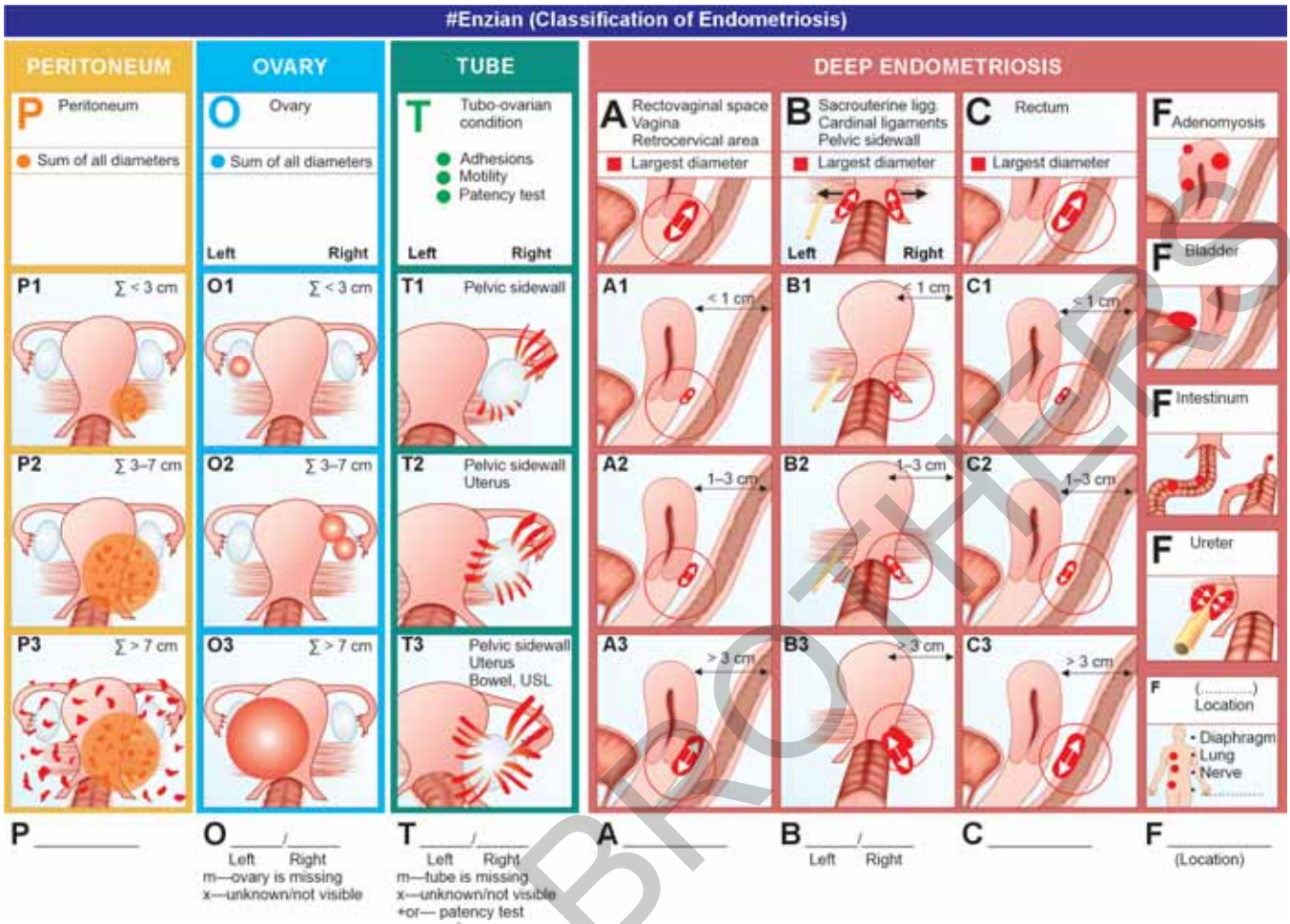


**Fig. 22.7:** #Enzian classification of endometriosis.

**#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).

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**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

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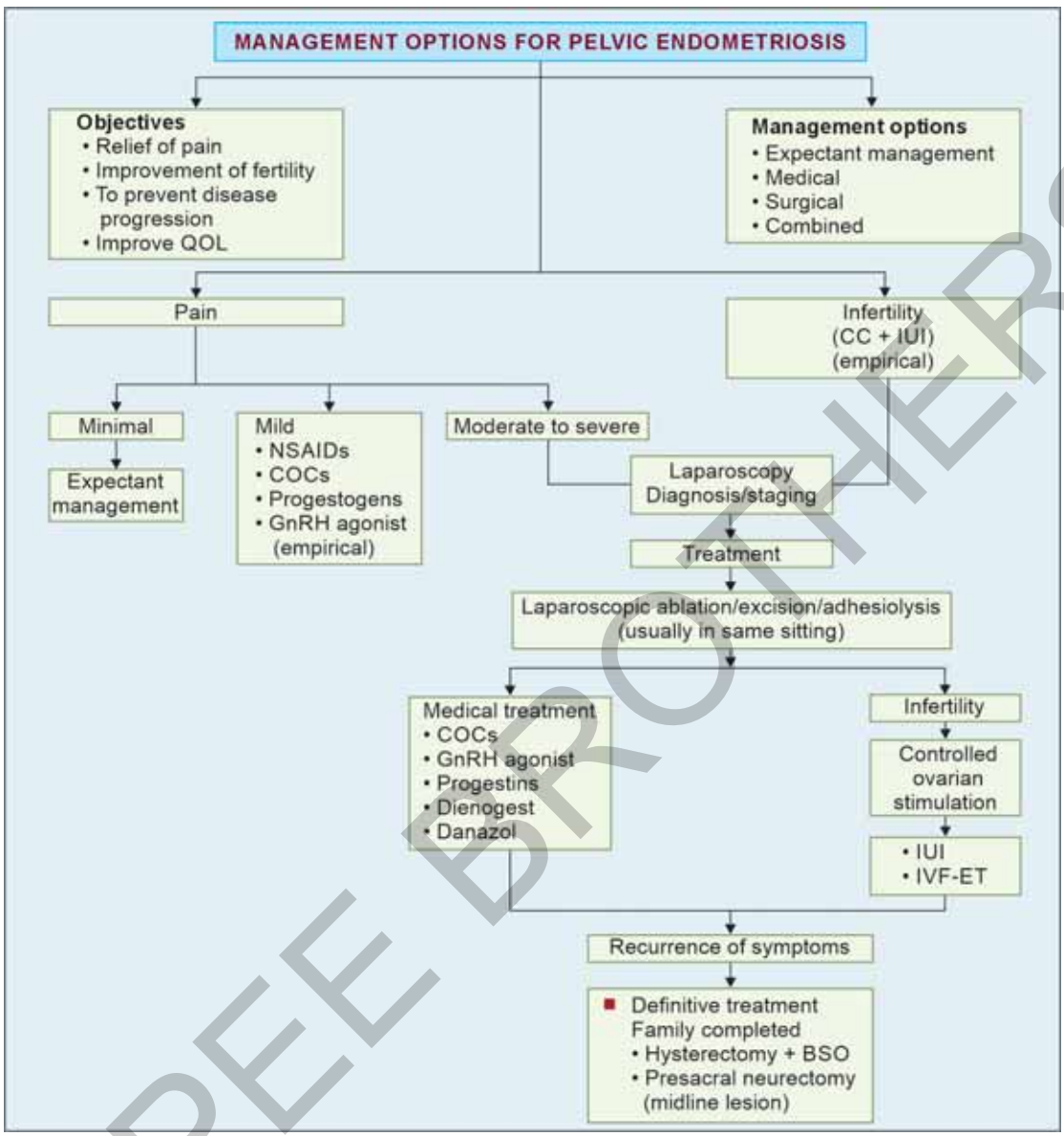
**Flowchart 22.1:** Therapeutic approach to a patient with pelvic endometriosis.




(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.

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### *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.

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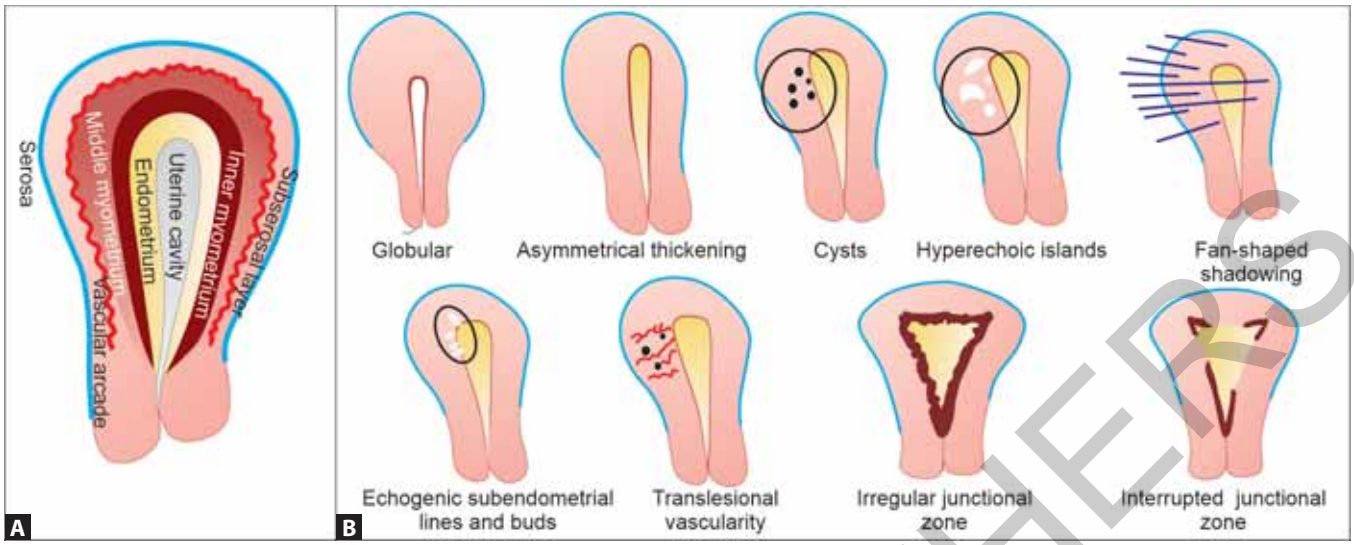


**Figs. 22.13A and B:** (A) Schematic diagram showing uterine layers (color-coded)—endometrium is yellow, junctional zone (inner myometrium) is dark red, middle myometrium, located between vascular arcade (red) and junctional zone, is light red; outer myometrium (subserosa), located between vascular 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...*

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ISBN 978-93-5696-855-4