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PATIENT INFORMATION: ENDOMETRIOSIS

Definition: Functional endometrial tissue that is found outside the uterus. In the ovary it is referred to as an endometrioma, and in the uterine myometrium it is called an adenomyosis.

Most common sites (in decreasing order): Ovaries, cul de sac (behind the uterus), uterosacral ligaments, posterior broad ligament, uterus, fallopian tubes, appendix, sigmoid colon, and round ligaments of the uterus. Less common sites include vagina, cervix, bladder, rectovaginal septum, cecum, ileum, ureters, and abdominal or perineal scars.

Epidemiology: Prevalence in the general population is unknown due to the fact that many women can be asymptomatic with the disease. Estimates indicate that 1% of all women undergoing surgery for any gynecologic conditions have endometriosis. In those women undergoing surgery for pelvic pain, 12-32% have lesions, and in women having surgery for infertility, 21-48% have endometriosis.[1]

Theories of pathogenesis:[2]

- Retrograde menstruation: Endometrial tissue fragments are transported backwards through the fallopian tubes into the peritoneum. Often this is due to an anatomic alteration in the pelvis or uterus.
- Direct implantation: This theory explains why endometriosis develops in tissue directly involved with a surgery or wound, i.e. c/s scars and episiotomies.
- Coelomic metaplasia: Theory suggests that there are undifferentiated cells in the peritoneum that are capable of becoming endometrial tissue. This is based on the embryology of all pelvic organ cells (including the endometrium) are derived from the cells lining the coelomic cavity.
- Vascular/lymphatic transport: The theory can explain why there are very rare cases of endometriosis in the thoracic cavity, bone, breast, pancreas, and other distant sites.

Clinical presentation: First, many women with endometriosis are completely asymptomatic and the finding may be incidental. Some of the more common symptoms involve pelvic pain (which is more severe during menses because of the pressure of the lesions on surrounding tissue). This characteristic triad of symptoms includes dysmenorrhea, dyspareunia (painful intercourse), and dyschezia (painful bowel movements). Other symptoms include infertility and perimenstrual spotting.

Some of the typical signs include a tender, immobile, retroflexed uterus, a tender and/or fixed adnexal mass, a sharp “barb-like” pain felt in the uterosacral ligament, or a palpable, tender mass in the cul de sac or uterosacral ligament while performing a bimanual exam.

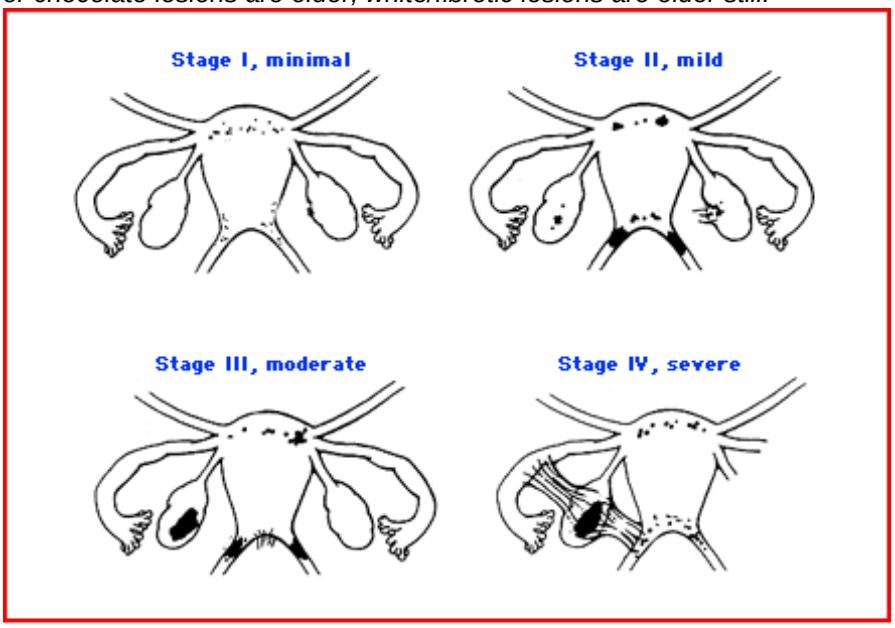
Diagnosis: The definitive diagnosis of endometriosis can only be made by visualizing the pelvic cavity through a laparoscopy or laparotomy. Until that is done the differential diagnosis includes endometriosis, acute salpingitis, pelvic inflammatory disease, hemorrhagic corpus luteum, ectopic pregnancy, and a benign/malignant ovarian neoplasm.

Staging: This is done on the basis of the total size of the lesions, the tissue they are implanted on, and whether or not there are any adhesions present.

Effect of endometriosis on fertility: Anatomical abnormalities in peritoneal, tubal, uterine, and ovarian position are a potential mechanism linking infertility and endometriosis. Adhesions can develop in tissues that can essentially put them out of position to be conducive to conception and implantation. Studies have shown that women with endometriosis have an increased volume of peritoneal fluid, increased peritoneal fluid concentration of activated macrophages (white blood cells), and increased peritoneal fluid concentration of prostaglandin, interleukin-1, tumor necrosis factor, and proteases. These abnormalities in the peritoneal environment may impair gamete, embryo, and fallopian tube function. For example, peritoneal fluid from women with endometriosis has been reported to inhibit sperm function and ciliary function, thereby possibly reducing fertility.[4]

Lesion appearance: “Blueberry” lesions are relatively young; brown

or chocolate lesions are older; white/fibrotic lesions are older still.



Examples of the classification of endometriosis Modified from the American Society for Reproductive Medicine.

Staging attempts to quantify endometriosis and correlate it with fertility potential.[1]

Treatment: There are only two generally accepted choices for the treatment of endometriosis: surgical or medical therapy.

Surgical resection is usually reserved for very large endometriomas (over 3 cm in size). If the patient is of advanced age and does not desire fertility, a TAH-BSO is done to remove all endometrial tissue and the hormone-producing ovaries which stimulate the growth of endometriosis. On the other hand, if the patient is younger and/or fertility is desired then a minimally invasive laparoscopy can be performed. In this case, endometriomas can be resected (leaving the remaining ovary intact usually if the endometrioma cyst is under 5 cm in size), and endometriosis lesions can be ablated with laser or resected.

Medical treatment involves using combination OCP's or Depo Provera to cause the endometriosis to stop growing.[2] Progesterone alone (i.e. Provera) can also be used to simulate a "pseudo" pregnancy state and temporarily calm the endometriosis tissue. A final medical approach is with a GnRH agonist like leuprolide or nafarelin. With the use of these drugs, stimulation of pituitary luteinizing hormone (LH) and follicle stimulating hormone (FSH) secretion occurs during the first 10 days of treatment. Paradoxically, chronic treatment results in a decrease in pituitary secretion of LH and FSH due to GnRH receptor down regulation and pituitary desensitization. This decrease in pituitary LH and FSH secretion suppresses ovarian follicular growth and ovulation, resulting in low levels of circulating estradiol and progesterone. Women chronically treated with clinically effective doses of parenteral GnRH agonists have circulating estradiol concentrations in the range of menopausal women. The main side effects of GnRH agonist analogues are those observed in hypoestrogenic women: vasomotor symptoms, insomnia due to hot flashes, urogenital atrophy, and accelerated bone loss.[5]

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