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Pathophysiology and Pathogenesis of Endometriosis
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Introduction
- Endometriosis is a chronic gynecologic condition characterized by the presence of endometrial tissue outside of the uterus. This tissue forms lesions that expand and contract, causing pain and discomfort.
- Many women experience various symptoms associated with endometriosis.

Presentation/Diagnoses
- For some women, receiving a proper diagnosis of endometriosis can prove to be a long, frustrating process.
- Burden and Giudice (2012) found that it takes approximately 6.7 years on average to achieve a definitive diagnosis. Pain associated with endometriosis often, but not always, coincides with the timing of the menstrual cycle.

Theories of Pathophysiology
- Dependent on the areas involved, there are various theories of origin of endometriosis. It was first proposed that the TVLs and those that present that endometrial debris back flows through the fallopian tubes and into the peritoneal cavity. (Vialle et al., 2014).
- Although this is the long-standing theory, it is controversial. It is possible that women who experience retrograde menstruation (Burden & Giudice, 2012), however, a small percentage of women develop endometriosis. This theory does not explain the presence of endometriosis in other, non-menstruating populations, such as pre-pubertal girls, newborns, and males (Sourial et al., 2014).

Coelomic Metaplasia
- Coelomic metaplasia theory proposes that metaplastic tissue forms in the peritoneum to convert to endometrial tissue (Young, Brown, Saunders, & Horne, 2015). It is suggested that endometriosis disrupts the developmental mechanisms that contribute to this transformation, but this is not well understood.
- Unlike other metaplastic diseases, endometriosis does not appear to worsen with age (Young et al., 2015). The source of endometriosis primarily in the abdominal cavity also contradicts the nature of other metaplastic diseases.
- Implantation theory is clearly related to endometriotic metaplasia, but stop further to propose that conversion to endometrial disease is not readily facilitated disease, but that cells must “complete a more restrictive metaplasia, proliferation” (Young et al., 2015, p.559).

Immune Dysfunction
- Barlow and Giudice (2012) observe strong support for the theory that the immune system of women with endometriosis has a higher incidence of other autoimmune diseases related, such as a raised number of progestogen receptors (Burden & Giudice, 2012). According to Sourial et al. (2014), the “Pathogenesis commonly occurs as a proliferation, activation, and overexpression of estrogen-stimulated epithelicious tissues” (p. 3). Endocrine disruptors in the environment have also been implicated in the pathogenesis of endometriosis.
- They act by mimicking estrogen in the body and can thereby cause a hyperstimulation of the system with the body’s estrogen receptors and increasing circulating levels (Sourial et al., 2014).

Oxidative Stress and Inflammation
- Changes in the immune system, and women with the disease have a “higher concentration of activated macrophages, neutrophils, and basophils, increased immunity, and a suppressed natural killer (NK) cell function” (Sourial et al., 2014, p. 35). The theory of estrogen stimulation is currently a dominating factor as it is associated with endometriosis.
- This cycle leads to prolonged inflammation and increased angiogenesis, fostering the proliferation of endometrial implants.

Bacterial Contamination
- The bacterial contamination hypothesis is relatively new, and proposes that the naturally barrier-rich environment in the vagina inadequately contributes to the facilitation of endometriosis, mainly through Escherichia coli (E. coli) contaminated retrograde menstruation and subsequent inflammation and immune response. Lynch et al. (2014) reported that Gram-negative bacteria activate cells of the innate immune system, leading to the formation of a unique vascular endothelial cell growth factor (VEGF), which stimulates angiogenesis.
- A parallel theory looks to the microbes of the gut for an explanation of endometriosis development. This theory relates to the central role of the cycling of estrogen, and disruptions in normal functioning can be perceived as disruptions in the system (Laschinger & Menger, 2009). Therefore, both of these pathways in the peritoneum is believed to be impacted by gut inflammation and peritoneal immunity.

Mullerianiasis
- The theory of Mullerianiasis, also known as mullerianiasis theory that deals with Mullerian development of disease, was proposed by Nathan, which states that the ovaries are involved in the development of endometriosis when polypoid tissue and proliferative epithelia are observed (Barlow & Giudice, 2012). This is similar to the metaplastic theory, and involves the Mullerian development of disease in the mullerian ducts of female fetuses. (Klemm & Stensballe, 2018)

Signs & Symptoms
- Pelvic pain
- Irregular uterine bleeding
- Dysmenorrhea
- Dyspareunia
- Metroschmerz
- Dysfunctional uterine bleeding
- Fertility impairment

References

Additional Sources