What Causes Endometriosis?

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Diagnosis of Endometriosis
Current Treatment of Endometriosis

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**Sampson’s Theory**
This theory was proposed by Dr. John Sampson of Boston in the 1920’s. His theory that menstrual blood refluxed through the fallopian tubes and was deposited and grew on the pelvic peritoneum and pelvic organs remains popular, but the initial attachment of single or multiple endometrial cells on the peritoneal surface has not been demonstrated. Additionally, the time-related geographic spread of endometriosis throughout the pelvis that would be predicted to occur with repeated seeding of the peritoneum by refluxed endometrium has not been demonstrated.

The fact that 90% of women have retrograde flow but only 15% of women develop endometriosis further repudiate the validity of the theory. This theory of origin also implies that older age groups of patients with endometriosis have more disease than younger age group and a high and progressively increasing rate of recurrence after complete surgical resection. The literature, however, as well as our own experience has shown that the actual rate of persistent or recurrent disease is surprisingly low after aggressive conservative surgical excision at laparoscopy or laparotomy, and the rate of recurrent or persistent disease does not appear to increase with the passage of time following excision. Because of these deficits, Sampson’s theory is losing favor to more modern concepts.
Theory of Embryonically Patterned Metaplasia or Rests
Metaplasia means a transformation from one type of tissue to another type of tissue. During the embryonic stage, the primitive cells migrate and undergo differentiation to form the pelvic organs. This embryogenesis is controlled and directed by a sophisticated, but still incompletely understood, fetal system. This fetal developmental control system may be the fetal analog of the adult immune system. If a developmental or heritable defect in this controlling system exists, then differentiation and migration of cells may be aberrant or incomplete. Cellular morphology and functionality might be expressed overabundantly or inadequately. Such cells or tracts of cells are laid down in the migratory pathway of fetal organogenesis (forming the organs) across the posterior pelvic floor, although location anywhere might be possible, depending on the degree of aberrant differentiation or migration. These cells may be endometrium-like initially, or may possess the ability to undergo metaplasia into endometriosis after puberty. Arrest of migration across the posterior pelvis would conveniently explain the observation that endometriosis is most commonly and predictably found in the cul-de-sac, uterosacral ligaments, and medial broad ligaments.

Abnormalities of the fetal development control system may be preserved into adult life, giving rise to detectable abnormalities of the adult immune system. The degree of residual abnormality of the adult immune system may control the aggressiveness of the endometriosis that develops, with the result that some patients may develop invasive disease or adhesions, while most do not. The competence of the adult immune system might be impaired by exposure to environmental toxins (e.g. Dioxin), with endometriosis emerging as a possible result, as occurs in experimental laboratory primates, and endometriosis in male bladder or prostate in later life during estrogen treatment of advanced prostate cancer.

Hematogenous metastasis of endometriosis
This theory has been proposed to explain remote occurrence of the disease. According to this theory, exfoliated endometrial cells are swept into the venous drainage of the uterus, with subsequent deposition possible anywhere in the body. Venous blood draining from the uterus must pass through the capillary bed to the lungs. Therefore, hematogenous spread should also result in a high rate of secondary pulmonary endometriosis unless there exists a high rate of occurrence of atrial or ventricular septal defects exists, among patients with extrapelvic endometriosis. Since neither pulmonary endometriosis nor cardiac atrial or ventricular septal defects have been reported to be
more frequent in patients with endometriosis, hematogenous spread remains speculative.

**Theory of lymphatic spread**
This theory has been supported by the finding of endometriosis in lymph nodes, although the number of reported cases is quite small compared with the number of women with endometriosis. Endometriosis in lymph nodes, however, could plausibly be explained equally by a developmental process that resulted in embryonic deposition of endometriosis in lymph nodal tissue.

**Genetic Predisposition**
Multicenter studies conducted by Stephen Kennedy, M.D. Professor at Oxford University show that first degree relatives of women with endometriosis are more likely to develop endometriosis. And when there is a hereditary link, the disease tends to be worse in the next generation. Chattanooga Women’s Laser Center is one of the participating study centers for the genetic predisposition factors in endometriosis.

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