



Global Year Against Pain in Women

real women, real pain

Endometriosis and its Association with Other Painful Conditions

Endometriosis occurs in up to 10% of women of childbearing age (1).

Endometriosis is defined by the abnormal presence of endometrial tissue (tissue with characteristics of the inner lining of the uterus) outside the uterus, usually in the abdominal/pelvic cavity (1). These abnormal tissues are referred to as “ectopic growths,” or sometimes “implants” or “cysts.”

Symptoms associated with this condition include subfertility and pelvic pains (1). The most common of the pains is dysmenorrhea, which is severe pain associated with menstruation (1, 2). Other pains include dyspareunia (pain with coitus or insertion of tampons; i.e., hypersensitivity of the vagina), dyschesia (bowel pain), and chronic pelvic pain (pain generally internally or in muscles of the abdomen, pelvic area and lower back) (1, 2).

For about 20% of women with this disorder, it co-occurs with other chronic pain conditions, such as irritable bowel syndrome, interstitial cystitis/painful bladder syndrome, vulvodynia, temporomandibular joint disorder, migraine, fibromyalgia, and/or with autoimmune disorders such as systemic lupus erythematosus, rheumatoid arthritis, chronic fatigue syndrome, Sjogren’s syndrome (1,6).

Endometriosis is a puzzling disorder due to the uncertain relationship between the defining signs of the condition (ectopic growths) and the varied symptoms (2). Some women have no or minor symptoms, yet when their internal pelvic or abdominal cavity is examined for other reasons, extensive signs are obvious. Other women have very few signs but extremely distressing and painful conditions.

It is poorly understood how the signs (growths) and symptoms (subfertility, pains, co-occurrence with other disorders) develop and become related to each other. What is generally agreed by most clinicians and scientists is that the ectopic growths develop in susceptible women due to “retrograde menstrual flow,” which is when menstrual tissue is pushed backwards through the fallopian tubes into the abdominal/pelvic cavity and then implants and grows there (1). These ectopic growths can then behave in a manner similar to the uterus, shedding their tissues and inflammatory molecules into the abdominal/pelvic cavity. It is also agreed that endometriosis is dependent on estrogens because its signs and symptoms disappear with menopause or removal of the ovaries (1). Recent studies have also shown that the active ectopic growths develop their own blood supply and nerve supply (2). It is possible that the variable relationship between the ectopic growths and symptoms may be due in part to variations in this nerve supply (2).

Treatments for endometriosis are of three main types (1): (a) over-the counter analgesics, (b) hormonal treatments that stop ovulation in order to reduce levels of estrogens, and (c) surgery (removal of ectopic growths, the uterus, or sometimes cutting certain nerves that supply the pelvic cavity). In some women, complementary/alternative treatments have been useful additions (6). One new possibility, now under study in animal models, are drugs that reduce the blood supply of the ectopic growths (4).

None of these treatments is completely satisfactory or effective (3, 5, 7). Hormonal treatments and removal of the uterus can have unpleasant side-effects and, obviously, prevent conception. Surgery is at best successful 50% of the time, can have unpleasant side-effects, and sometimes symptoms can return (3).

Nevertheless, one can remain optimistic, because considerable basic and clinical research is underway to develop a better understanding of the signs and symptoms and to develop more effective approaches for their treatment (see the following websites: <http://www.endometriosisassn.org/>; <http://www.nlm.nih.gov/medlineplus/endometriosis.html>; <http://www.endometriosis.org/>).

References:

1. Giudice LC, Kao LC. Endometriosis. *Lancet*. 2004;364:1789-1799.
2. Berkley KJ, Rapkin AJ, Papka RE. The pains of endometriosis. *Science*. 2005 10;308:1587-1589.
3. Chopin N, Vieira M, Borghese B, Foulot H, Dousset B, Coste J, Mignon A, Fauconnier A, Chapron C. Operative management of deeply infiltrating endometriosis: results on pelvic pain symptoms according to a surgical classification. *J Minim Invasive Gynecol* 2005;12:106-112.
4. Ferrero S, Ragni N, Remorgida V. Antiangiogenic therapies in endometriosis. *Br J Pharmacol* 2006;149:133-135.
5. Sinaii N, Cleary SD, Ballweg ML, Nieman LK, Stratton P. High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome and atopic diseases among women with endometriosis: a survey analysis. *Hum Reprod* 2002;17:2715-2724.
6. Sinaii N, Cleary SD, Younes N, Ballweg ML, Stratton P. Treatment utilization for endometriosis symptoms: a cross-sectional survey study of lifetime experience. *Fertil Steril* 2007 Feb 9; [Epub ahead of print].
7. Vigano P, Somigliana E, Parazzini F, Vercellini P. Bias versus causality: interpreting recent evidence of association between endometriosis and ovarian cancer. *Fertil Steril*. 2007 Feb 21; [Epub ahead of print].

Copyright International Association for the Study of Pain, September 2007.