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EDITORIAL

Endometriosis – still an enigmatic disease. What are the causes, how to diagnose it and how to treat successfully?

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Although the first precise description of endometriosis was made 80 years ago, the disease still belongs to the group of pathological conditions which are quite frequently called 'enigmatic disorders'. The term 'endometriosis' was introduced by John Sampson in 1927, who established retrograde flow of endometrial elements into the abdominal cavity as the most probable cause of the disease. The disease is characterized by the growth of endometrial tissue outside the uterine cavity. Multiple theories have been put forward to explain the pathogenesis of endometriosis. Today, to the retrograde menstruation theory of Sampson, we must add the proposal that the specific inflammatory response creates an environment that may promote implantation and proliferation as the result of defective 'immunosurveillance'. Recently, evidence for dysfunction of the immune system has accumulated and it has been found that changes in cell-mediated immunity and humoral immunity contribute to the development of the disease. Nevertheless, the question is still open as to whether changes in the immune system which lead to the disease, are coincidental to it, or result from it. A number of studies have been devoted to looking for some genetic background to endometriosis and to elucidating the role of genetic polymorphism as a possible factor contributing to its development. However, in the literature we meet a strikingly large amount of conflicting results. Some authors have provided evidence that the disturbed regulation of a number of differentially expressed mRNAs in eutopic/ectopic endometrium by ovarian steroids may influence the expression of specific target genes and take part in the pathogenesis of endometriosis. Our own data on the activity of matrix metalloproteinases and their inhibitors support the hypothesis that this pathway plays an important role in the pathogenesis of endometriosis. Considering different etiologic theories regarding endometriosis one should come to the conclusion that all the described mechanisms can contribute to the clinical situation of the individual woman and that the degree of contribution varies from patient to patient.

The exact prevalence of the disorder is unknown and varying figures have been published in the literature: the rough estimate is that endometriosis occurs in 3–10% of women of reproductive age and 25–35% of women with infertility problems. Endometriosis should always be suspected if any woman is complaining of infertility, dysmenorrhea and dyspareunia, although it must be remembered that the disease can be also asymptomatic. Nevertheless, endometriosis should be considered in all cases of infertility or in women with pain in the lower part of the abdomen, with the uterus in fixed retroversion, with nodularity of the uterosacral ligaments and cul-de-sac, and the precise diagnosis should always be confirmed by laparoscopy.

Treatment of the disease before initiated must be based on the extent and severity of endometriosis. The most common classification system employed in clinical practice is the one developed by the American Society for Reproductive Medicine (ASRM) based on findings at laparoscopy or laparotomy. The most important goal in the classification and staging of endometriosis is to propose an effective plan for management. But we must be aware that the staging system does not correlate well with pain relief and with a woman's chance of conception following therapy.

There are many biological mechanisms that may link endometriosis and infertility. These mechanisms involve distorted pelvic anatomy with major pelvic adhesions; altered peritoneal function, which may have an adverse effect on the oocyte, sperm, embryo

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and fallopian tube; altered hormonal and cell-mediated immunological system; abnormalities of endocrine and ovulatory function; and impaired implantation.

The therapeutic strategy depends on the goal to be achieved. Medical therapy is effective for relieving the pain associated with endometriosis. The suggested options involve treatment with danazol, gonadotropin-releasing hormone analogs (both agonists and antagonists), progestins, gestrinone and combined hormonal contraceptives. Pharmaceutical therapy is not advised in cases of infertility because several randomized controlled trials demonstrated any positive effect when infertility was associated with minimal or mild endometriosis. The recommendations of ASRM and the European Society for Human Reproduction & Embryology are similar. When formulating the management plan the patient's age, duration of infertility and stage of endometriosis should be taken into account. The diagnostic laparoscopy must be combined with surgical procedures which allow the safe ablating and excising or coagulation of the visible lesions of endometriosis. The patient's age plays the crucial role in the decision-making process. In cases of minimal or mild endometriosis in women below 35 years of the age, intrauterine insemination combined with ovarian stimulation could be the first choice for infertility treatment. For older women and for women with stage III/IV endometriosis who fail to conceive following conservative surgical therapy with laparoscopy or laparotomy, the most effective alternative is *in vitro* fertilization–embryo transfer (IVF-ET). We should remember that the impact of endometriosis on the outcome of IVF/intracytoplasmic sperm injection is the subject of controversy but these options offer the highest chance for pregnancy and taking home a baby.