this screening can be carry out in the postoperative stage.

The second stage is the surgical deleting of all visible E foci. It allows to reduce the whole time of treatment, volume and duration of the subsequent inhibitory hormonal therapy, to decrease negative effects of the hormone large doses on different female extragenital organs and systems. The surgery stage of treatment can be limited or even eliminated (at adenomyosis) if woman reproductive plans are not realized.

The third obligatory stage of E treatment should be the complete turn off hypothalamo-hypophisial system resulting in ovary block and developing of a temporary amenorrhea from endometrium atrophy. Only this allows to achieve the liquidation of the hidden E foci. The drug (gestagens, testosteron derivates, gonadoliberin analogs) is choosing individually with relation to the degree of E, individual portability and possible negative effects. In this position buserilin has one lack – the loss of calcium which may compensated easily.

Criteria of the drug dose sufficiency is not only complete losing of menstruation, but also absence of the hidden cycles showing a complete ovary inhibition. The temporary appearance of hot flash is the reliable marker of ovary turning off. The time of ovary turning off should not be less than 6-8 months.

The last stage of E treatment is the prophylaxis of its recurrences. For patients, interested in the subsequent pregnancy, the prophylaxis is achieved by restoring of ideal menstrual cycles. Monophasic COC provides as the prophylaxis of hyperplastic processes, as the contraceptions for majority other women. Hyperprolactinemy is the contraindication for COC. The restoring of menstrual cycles by selective doses of dopamin agonists in these patients is the prophylaxis of the E recurrences, but does not provide the contraceptive effect. The contraception must be non hormonal for these women. For the patients with a thyroid or adrenal genesis of E is necessary the corresponding hormonal therapy starting from the diagnosis installation.

The prophylaxis of any hyperplastic processes must last until the end of reproductive period of life. The age turn off of the ovarian function eliminates a hormonal basis of these diseases. In some transition age women (after 45 years) medicamental pseudomenopause can accelerates the beginning of the natural menopause.

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PERITONEAL ENDOMETRIOSIS AND INFERTILITY – INTERCONNECTION OF PATHOGENETHICS PROCESSES

Objective. To estimate the role of hormone-dependent changes of the microrelief of endometrial epitheliocytes in pathogenesis of infertility associated with peritoneal endometriosis.

Methods. Laparoscopy, biopsy of endometrial and peritoneal samples with/without endometriosis, aspirat of peritoneal fluid with/without endometriosis, organotypic culture of endometrium and peritoneal samples into diffusion chambers with millipore filters implanted subcutaneously to ovarioectomized rats and substitution therapy with sex steroids, light and scanning electron microscopy.

Results. In peritoneal endometriosis and deficiency of the ovarian function in secretory phase of the cycle endometrium has pathological condition in both structural level (defective modifications of glands, stroma and vessels) and ultrastructural level (deficiency of secretory transformation of the microrelief of endometrial epitheliocytes – persistence of microvilli and cilia). At total damage of the endometrium epitheliocytes the uterine infertility arises. Mosaic damage of a microrelief leads to formation of heterogenous structure of preovulatory endometrium at which implantation of blastocyst in the given cycle is possible, but in the further there is a high risk of pregnancy loss. At the beginning of menstruation viable cells with microvilli and cilia are kept in endometrium. These cells have increased adhesive potential, high proliferative activity and ability to survive geterotopically for a long time. Deficiency of the ovarian function is the reason of retrograde menstruation. In case of retrograde reflux in peritoneum cavity of the endometrium' aggressive cells with the raised ability to intercellular interactions, invasion and ectopic proliferation the peritoneal endometriosis is formed. Active spots of endometriosis maintain ovarian deficiency and establish conditions for uterine infertility.

Conclusions. Peritoneal endometriosis and associated infertility are pathogenetically interconnected. Chronic deficiency of the ovarian function forms the basis of these pathogenic processes. The persistence of microvillous relief of endometrial epitheliocytes in late secretory phase of the cycle in peritoneal endometriosis indicates the deficiency of endometrial secretory transformation, deficiency of the ovarian function and results in a disorder of the ovicell implantation, infertility or pregnancy loss.

ISSN 1684-0461