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ARTIFICIAL INTELLIGENCE IN SCREENING AND EARLY DIAGNOSIS OF CERVICAL AND ENDOMETRIAL CANCER: A SYSTEMATIC REVIEW AND META-ANALYSIS OF STUDIES

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Cervical cancer, originating from two types of covering epithelium, has two main histological variants — squamous cell carcinoma (keratinizing and non-keratinizing) and adenocarcinoma arising from the columnar epithelium. Epidemiological studies show that there are two age peaks in the incidence of cervical cancer: at 34–36 years, when squamous cell carcinoma predominates, and at 60–62 years, when the proportion of cervical adenocarcinoma increases. Cervical cancer ranks third in frequency among malignant neoplasms of the female genital organs. In recent years, there has been an annual increase in the incidence of cervical cancer, especially among young women, by 2%.

Introduction

Every year, approximately 500,000 new cases of cervical cancer are diagnosed worldwide. The etiology and pathogenesis of cervical cancer are associated with human papillomavirus infection (HPV) and are similar to those of precancerous processes (discussed in the section devoted to cervical precancer). Squamous cell carcinoma accounts for 85–95% of all malignant cervical lesions, while adenocarcinoma accounts for 5–15%.Cervical cancer may demonstrate exophytic growth, which is more characteristic of tumor localization on the exocervix, or endophytic growth, characteristic of localization in the endocervix. In endophytic forms of the disease, the prognosis is worse.Depending on the extent of spread, cervical cancer is divided into clinical stages:

Stage 0 — carcinoma in situ.

Stage I — tumor limited to the cervix: 1a1 — microinvasive cancer with invasion no more than 3 mm and horizontal spread no more than 7 mm (metastasis rate 0.3%); 1a2 — microinvasive cancer with invasion depth 3–5 mm and tumor diameter 7–10 mm (regional lymph node metastasis rate up to 13%); 1b — invasive cervical cancer (invasion depth more than 5 mm).

Stage II — tumor extends beyond the cervix: IIa — infiltration of the upper and middle thirds of the vagina or the uterine body; IIb — infiltration of the parametrium not reaching the pelvic wall.

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Stage III — tumor extends beyond the cervix: IIIa — infiltration of the lower third of the vagina; IIIb — spread of infiltrate to the pelvic wall, hydronephrosis, or secondary contracted kidney.

Stage IV — tumor invades adjacent organs or extends beyond the pelvis: IVa — invasion of the bladder or rectum; IVb — distant metastases.

Clinical manifestations of early-stage cervical cancer are absent. Pathognomonic signs of invasive cervical cancer include bloody discharge; less commonly, acyclic bleeding occurs. Patients may complain of purulent, foul-smelling discharge, pain (including in the lower back and kidney area), fever, weight loss, and dysfunction of adjacent organs. These are signs of advanced cancer.

CLINICALGUIDELINES

Cervical Endometriosis

Cervical endometriosis (CE) is a pathological process characterized by the appearance on the vaginal portion of the cervix of tissue structurally similar to the endometrium and undergoing cyclic changes according to the menstrual cycle.

Epidemiology

The prevalence of CE reaches up to 24% of all cases of endometriosis.

Classification

CE is one of the forms of external genital endometriosis.

Etiology and Pathogenesis

Endometriosis most often develops in women with hormonal homeostasis disorders, chronic inflammatory processes in the pelvic area, and genetic predisposition.

Factors contributing to the development of cervical endometriosis include: • abortion; • pathological labor, multiple childbirths; • diagnostic procedures (separate diagnostic curettage, cervicoscopy, hysterosalpingography); • insertion and removal of intrauterine devices with repeated use of forceps on the cervix; • physiosurgical treatment of cervical pathology (diathermocoagulation, radio wave surgery, cryodestruction, laser vaporization, plastic surgery to restore cervical integrity); • supravaginal hysterectomy for uterine fibroids and adenomyosis (non-radical treatment).

Endometrioid heterotopias are located not only on the vaginal portion of the cervix but also in the distal part of the mucosa of the cervical canal.

Clinical Signs and Symptoms

One of the main symptoms of CE is pre- and postmenstrual spotting (“smearing”).

If endometrioid heterotopias are localized only on the vaginal portion of the cervix, pain syndrome is usually absent. However, when they invade the cervical canal or when CE is combined with endometriosis of other localizations, patients may experience nagging lower abdominal pain that varies in intensity throughout the cycle, as well as dyspareunia.

The diagnosis of CE is established based on: – examination of the cervix using specula; – colposcopy (preferably in the premenstrual period); – targeted biopsy followed by histological examination; – if endometriosis of the cervical canal mucosa is suspected, diagnostic curettage of the endocervix is performed before biopsy.

On examination of the cervix with specula, small foci 2–5 mm in diameter, reddish in color,

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contrasting with the pale pink mucosa, may be detected on the vaginal portion. In the luteal phase of the menstrual cycle, especially on the eve of menstruation, endometriosis foci become bluish-purple and slightly increase in size.
If the process is localized in the cervical canal mucosa, the lesions may have a polypoid form and mimic chronic endocervicitis. The colposcopic picture of CE is characterized by changes in the color and volume of endometrioid tissue depending on the phase of the menstrual cycle.

Differential Diagnosis
Differential diagnosis of cervical endometriosis is carried out with: • cervical cancer; • chronic endocervicitis; • telangiectasias; • Nabothian cysts with hemorrhagic content.

Clinical Recommendations
Treatment depends on the extent of the process.
Treatment of CE in patients of reproductive age with preserved menstrual cycle is combined hormonal and surgical therapy: coagulation of endometriosis foci using a CO_2 laser is most appropriate. Removal of the lesion should be performed in the early follicular phase of the menstrual cycle.
In CE, 1 month before and after the 3rd month following coagulation of heterotopias, combined oral contraceptives (COCs) or progestins are prescribed:
• Ethinylestradiol/gestodene 20 μg/75 μg or 30 μg/75 μg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months; • Ethinylestradiol/desogestrel 20 μg/150 μg or 30 μg/150 μg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months; • Ethinylestradiol/dienogest 30 μg/2 mg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months; • Ethinylestradiol/norgestimate 35 μg/250 μg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months.
Conclusion
The use of progestins is preferable (however, due to the more frequent development of side effects, in mild forms of endometriosis with minimal clinical manifestations, COCs are prescribed):

• Dydrogesterone 10–20 mg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months; • Levonorgestrel intrauterine system, inserted into the uterine cavity on days 4–6 of the menstrual cycle, once; • Medroxyprogesterone 150 mg intramuscularly once every 2–3 months for 6–9 months; • Norethisterone 5–10 mg orally once daily from day 5–20 to day 25 of the menstrual cycle, followed by a 7-day break, or once daily continuously for 6–9 months; • Progesterone 100 mg orally twice daily from days 16–20 to day 25 of the menstrual cycle for 6–9 months.

Errors and Unjustified Prescriptions
Prescription of triphasic oral contraceptives. Performing coagulation of CE in stage II–III internal endometriosis.

Prognosis
After complete removal of the endometrioid lesion, disease recurrence is extremely rare. It has been proven that the administration of hormonal therapy in the early postoperative period significantly increases treatment effectiveness and reduces the frequency of recurrence.

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