VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Female genital organs disease. Uterine diseases. Endocervicosis. Glandular hyperplasia of endometrium. Endometriosis. Precancer and cancer of uterine cervix and body. Diseases of ovaries, uterine tube and mammary gland. Endometriosis and tumors of ovaries. Fibro-cystic disease, fibroadenoma; cancer of mammary gland. Pathology of placenta and umbilical cord. Pathology of pregnancy and the postpartum period. Spontaneous abortions. Ectopic pregnancy. Eclampsia, pre-eclampsia. Trophoblastic disease. Diseases of the perinatal period. Prematurity. Postmaturity. Birth trauma and birth injury. Congenital malformations. Intrauterine infection. Hemolytic disease of the newborn. Cystic fibrosis. Tumors in children.

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

Diseases of the female genital organs and mammary glands

1. The purpose of the lesson. To study the etiology, pathogenesis, morphological characteristics, complications and outcomes of diseases of the female genital organs and mammary glands.

2. Requirements for the level of the student in the development of the discipline - pathological anatomy. The student must know:

1. Definition, etiology, patho- and morphogenesis of cervical diseases.
2. Definition, etiology, pathogenesis, pathological anatomy, complications of diseases of the uterine body.
3. Definition, pathological anatomy of an ectopic (ectopic) pregnancy.
4. Definition, pathological anatomy of diseases of the mammary glands.

3. Theoretical aspects.

Diseases of the female genital organs and mammary glands

Diseases of the female genital organs include diseases of the cervix and body of the uterus, ovaries, vagina and pregnancy pathology.

Inflammatory diseases of the cervix uteri (cervicitis) can be caused by a variety of pathogens, most often chlamydia, herpes simplex virus, human papillomavirus, gonococci, trichomonads, and fungi of the genus Candida. Acute cervicitis is characterized by swelling and plethora of the neck, the presence of purulent discharge. Microscopically marked marked infiltration by polymorphonuclear leukocytes, edema and plethora. Often, inflammation is accompanied by degeneration and necrosis of epithelial cells with the formation of erosion. Erosions, as a rule, heal quickly, in their place granulation tissue develops, covered with stratified squamous epithelium. Chronic cervicitis is characterized by lymphomacrophagic infiltration of subepithelial tissue, often with an admixture of plasma cells. With intense inflammation, the infiltrate can spread to the epithelial layer. In the epithelium there are signs of acanthosis, hyperkeratosis, cell polymorphism. Chronic cervicitis is difficult to treat.

Diseases of the cervix uteri are divided into non-tumor lesions of the cervix (ectropion, ectopia of the cervix, erosion, cervicitis, condylomas, cysts of the mucous glands); precancerous conditions (squamous intraepithelial neoplasia, glandular intraepithelial neoplasia); malignant tumors of the cervix (carcinomas, sarcomas). The human papillomavirus has etiological significance in the development of cervical pathology, including pre-tumor and tumor pathology. Of particular importance are oncogenic strains of human papillomavirus, such as 16, 18, 31, 33, 54. When integrating viral deoxyribonucleic acid, the reading frame E1 / E2 is interrupted, which leads to the loss of the E2 viral repressor and increased expression of the oncoproteins E6 and E7 .

Cervical ectopia (pseudo-erosion) is found, as a rule, in young women and is characterized by a shift in the zone of transformation into the vaginal part of the cervix, where a single-layer cervical epithelium appears.

As a result of desquamation of the squamous epithelium, erosion of the cervix (true erosion) occurs. Macroscopically, erosion is a bright red surface in the area of the external pharynx of the vaginal part of the cervix. Erosion has irregular or rounded outlines, often bleeds when touched. The microscopic picture in the presence of “true” erosion is characterized by the presence of pronounced inflammatory phenomena, the disappearance of the stratified squamous epithelium, and the application of fibrin and blood onto the erosive surfaces. True erosion exists for a short time (from several days to 1-2 weeks). A possible continuation of the process is the transition to the next stage of the disease - endocervicosis (pseudo-erosion). Endocervicosis is a section of the vaginal

part of the cervix uteri lined with prismatic epithelium. The transformation of true erosion into endocervicosis is carried out as a result of the growth of the cylindrical epithelium on the erosive (devoid of stratified squamous epithelium) surface of the vaginal part of the cervix. Macroscopically detected area of bright red color, surrounded by a pale pink mucosa. Distinguish: stationary, proliferating and epidermal endocervicoses.

Stationary endocervicosis (cervical ectopia according to the WHO) indicates a phase of relative dormancy, when pseudo-erosion does not grow (does not increase in size), but is not yet healing. Histologically, glands with no signs of proliferation are detected. Proliferating endocervicosis is characterized by the growth of new glands in the cervix, which is accompanied by the appearance of low-grade epithelium under the cylindrical epithelium with subsequent differentiation towards the glandular. Epidermisizing endocervicosis (cervical ectopia according to the WHO) is characterized by the growth of a stratified squamous epithelium into the glands or the appearance of a stratified squamous epithelium under a layer of glandular (single-layered high prismatic, i.e., cervical type epithelium). The polyp of the cervix is characterized by three signs: 1) “Cap” from a single-layer cylindrical epithelium; 2) Leg with thick-walled blood vessels; 3) Uneven distribution of glands. The appearance of cervical canal polyps is very diverse. They can have a smooth surface or, on the contrary, villous and consist mainly of glandular or fibrous tissue. Glands can be distributed more or less evenly or located in surface areas. The structure of the stroma is also diverse: it can be composed of loose connective tissue rich in lymphoid cells, or fibrous, poor in cells. Often in polyps hyperemia, hemorrhages, foci of necrosis develop. All these changes are usually due to the torsion of the legs of the polyp.

The concept of "low and high grade cervical intraepithelial neoplasia" combines processes that were previously called grade I-III dysplasia and in situ cancer, developing in the cervical epithelium. Especially often, these changes are observed in the transformation zone. A key sign of cervical intraepithelial neoplasia is cellular atypia. Cervical intraepithelial neoplasia of a low degree corresponds to mild and moderate dysplasia, cervical intraepithelial neoplasia of a high degree includes severe dysplasia and cancer in situ.

According to the WHO classification, it is advisable to subdivide cervical epithelium dysplasia into three degrees: 1) weak, 2) moderate, 3) severe (severe), respectively, the degree of cellular atypia and the safety of architectonics of the epithelial layer. Various forms of epithelial dysplasia of the vaginal portion of the cervix are referred to as precancerous changes. Mild dysplasia is characterized by the preservation of stratification (the correct alternation of the number of storeys of the layers of the multilayer epithelium) and vertical anisomorphism (uneven size (location) of the layers of multilayer epithelial cells vertically) of the surface (functional) and intermediate layers. Basal cell hyperactivity of the lower third of the epithelial layer is noted. Basal and parabasal cells are monomorphic, rounded oval and elongated, with a preserved, somewhat narrowed, rim of the cytoplasm, which assumes a basophilic hue. The nuclei of the basal and parabasal cells are enlarged, often of irregular shape, hypo -, normo - and hyperchromic. Moderate dysplasia captures half or more of the thickness of the epithelial layer. Violations of vertical anisomorphism and stratification of the lower layers of the reservoir due to total basal cell hyperactivity are noted. Hyperplastic cells of the lower layers are oriented perpendicular to the basement membrane, with a narrow rim of the cytoplasm. The number of cells with mitotic fission of nuclei increases towards the basement membrane.

Severe dysplasia captures a large part of the thickness of the epithelium with the exception of several surface layers of mature cells that maintain a normal structure. Below these surface layers, vertical anisomorphism and stratification are absent. The arrangement of cells is random, large cells with dark-colored nuclei are found in the overlying layers. The cytoplasm in a significant part of the cells is presented in the form of a narrow rim. Although this form of dysplasia is limited to a small portion of the epithelial cover, it can simulate Ca in situ. . The absence of pronounced signs of atypism and polymorphism of epithelial cells, a limited area of the lesion help differentiate this type of dysplasia from Ca in situ. It should be remembered that different degrees of in situ

dysplasia of Ca can occur in the same neck. Therefore, to conduct a more reliable differential diagnosis, the area of the studied object is of great importance.

Cervical cancer almost never develops in an unchanged cervix, its development is preceded by cervical intraepithelial neoplasia. Exophytic (in the form of papillary growths), ulcerated, endophytic (infiltrating and deforming the cervix; secrete the so-called barrel-shaped cervix), microinvasive (cancer with invasion into intact tissues to a depth of less than 3 mm), cervical cancer are isolated.

Carcinoma in situ (preinvasive cancer, or intraepithelial) By the degree of differentiation: 1) Highly differentiated; 2) Medium differentiated; 3) Low grade. A highly differentiated (or mature) variant of cancer is most common. Its characteristic feature is the preservation in general terms of the similarity of the reservoir with stratified squamous epithelium, manifested in the presence of vertical anisomorphism. In these cases, the layers of the epithelium are more or less distinct, but the relationship between them can be violated to a certain extent. The number of basal cells is often increased (basal cell hyperplasia). The hyperchromic nuclei of these cells are closely adjacent to each other, since the cells are devoid of cytoplasm and elongated in the vertical direction. The presence of a large number of dividing cells at different levels is characteristic. A low-grade (immature) variant is less common. It is characterized by disorders of the multilayered integumentary epithelium and epithelium located deep in the glands, in which the entire epithelial layer in its entire thickness consists of cells resembling the basal layers of the normal epithelium. However, with this form of Ca in situ, the nuclei are larger, more often with significant hyperchromatosis, the cells are almost devoid of cytoplasm, oriented in the vertical direction. Conventional and pathological mitoses are visible at various levels in large numbers. Often, cells of this type are covered from above with a thin layer of cells lying horizontally, arranged in the form of one or two layers with pycnotic rod-shaped nuclei. In such areas, the epithelial layer is often not thickened, and sometimes even thinned. It should be emphasized that undifferentiated forms of Ca in situ have the greatest tendency to spread to the cervical canal and to invasive growth. The two extreme variants of squamous cancers of the in situ cervical cancer that are presented and described in detail - highly differentiated (mature) and low-differentiated (immature) are easier to distinguish from each other. If in the first variant the epithelium still retains to a certain extent the ability to differentiate, is relatively mature and sometimes even retains a keratinization property, in the second variant the epithelium is undifferentiated and is represented mainly by cells resembling basal cells of a normal squamous multilayer epithelium. Ca in situ is most often observed in the vaginal part of the cervix in the junction of the stratified squamous and prismatic epithelium in the external uterine pharynx, less often and in the initial sections of the cervical canal.

Uterine body diseases are divided into pathology of the endometrium (endometrial polyps, endometrial hyperplasia and cancer) and myometrium. Endometrial polyps (fibrous-glandular, hyperplastic and functional) are represented by the fibrous stroma with glands unevenly located in it, expressed to different degrees. With gland hyperplasia with atypia of the epithelium, they speak of atypical hyperplastic polyps.

Endometrial polyps. The polyp of the uterine body is a local, exophytic growing, benign glandular formation originating from the tissue of the basal layer of the endometrium. The sizes of polyps are diverse: from microscopic to large exophytic formations that perform the entire uterine cavity and penetrate through the cervical canal into the vagina. In most cases, polyps are small (0.3 - 1.0 cm) and are single. Multiple polyps are much less common. The shape of the polyps is different - from spherical, pear-shaped or mushroom-shaped to elongated, cylindrical. Sometimes polyps are located on a wide base. More often, polyps have a short or long “leg” formed by the involvement in its growing exophytic part of the thick-walled vessels of the basal layer and thin bundles of fibrous and smooth muscle tissue. Most often, in women with preserved menstrual function, a glandular polyp of the basal type from an immature endometrial type is observed. In a

scraping made in the secretory phase of the cycle, the polyp glands are randomly arranged, their shape is diverse, and there is no correct orientation to the surface of the pieces. In a scraping done in the proliferative phase, a choatic arrangement of the glands and a denser, somewhat fibrosed stroma is observed, in which elements of the vascular pedicle of the polyp can be found. In the stroma, you can find foci from large, thick-walled vessels, often located in chains surrounded by a narrow layer of connective tissue elements elongated parallel to the "vascular pathway". Such accumulations of vessels are extremely characteristic of the vascular - connective tissue leg of the polyp and, along with the more pronounced fibrous character of the stroma, can serve as a whole differential diagnostic sign that distinguishes the polyp from the background tissue of the endometrium. Glandular polyps of a functional type are observed much less frequently. In this form, the polyp tissue responds to normal and pathological stimulation from the ovaries and shows changes corresponding to the proliferative or secretory phases of the cycle, and can also participate in dishormonal hyperplastic processes. Microscopically: the disorder of the location of the glands, the poly-shaped appearance of some scraping fragments, the presence of the vascular pedicle of the polyp.

Endometrial hyperplasia Increased formation of estrogen hormones - hyperestrogenia causes the appearance in the endometrium of hyperplastic structures such as glandular hyperplasia. The endometrium with dishormonal proliferation is usually thickened. There is no division into compact and spongy layers, the correct distribution of the glands in the stroma is violated, cystic enlarged glands are characteristic.

In the current International classification, endometrial hyperplasia is divided into:

1. Simple hyperplasia without atypia
2. Complex hyperplasia without atypia (glandular without atypia)
3. Simple atypical hyperplasia
4. Complex atypical hyperplasia (glandular with atypia) Simple endometrial hyperplasia without atypia.

The main clinical manifestations of PGE is 3-4 months. amenorrhea followed by heavy bleeding. Macroscopically, scrapings of different sizes, depending on the continuation of previous bleeding, often consist of large areas of pale pink color. If the bleeding was prolonged and heavy, areas of the endometrium may be small. Histological signs: - there are no signs of a normal phase of proliferation or secretion; - the functional layer is high, there is no division into compact and spongy parts; - proliferation of surface epithelial cells of varying severity; - a large number of slightly crimped glands; the glands branch, not wriggle, the lumens of a part of the glands are cystically dilated, the epithelium is represented by high prismatic cells with elongated nuclei. - cysts of different sizes and shapes - a constant sign of PGE, lined with flattened epithelium, contain an eosinophilic mass; - tangles of blood vessels are not formed; thin-walled vessels, sinusoidal type. Complex endometrial hyperplasia without atypia. Histological signs: - an increase in the number of glands, an increase in the branching of glands (budding), "kidneys" are directed both inside the glandular cavity and in the stroma, the glands are closely adjacent to each other; the glandular epithelium is more multi-row than in PGE, forms pseudo-papillae and true papillae; epithelial cells are monomorphic, the nuclei retain a vertical, elongated shape, which indicates that the gland epithelium responds to hyperestrogenism by a superproliferation reaction; there may be foci of squamous metaplasia and proliferation of reserve “cell-bubbles”; cysts become smaller. - a decrease in the stromal component due to an increase in the branching of the glands; proliferation of stromal cells in the interlayers between the glands; - proliferation of surface epithelial cells of varying severity; - - tangles of blood vessels are not formed; thin-walled vessels, sinusoidal type. Simple atypical endometrial hyperplasia. A rare form of endometrial hyperplasia. The main sign is the atypism of cells and nuclei, which indicates the absence of cell response to sex hormones, dedifferentiation of the epithelium and mucus formation. Histological signs: - a large number of glands of a relatively regular shape, with a narrow lumen, of different sizes, cells and nuclei of the

glandular epithelium increase in size, round, brighten, nucleoli enlarge, the chromatin in the nuclei becomes light or excessively dense; - focal nature of the process; - the stroma is well defined, stromal cells proliferate; - vessels of the sinusoid type. Complex atypical endometrial hyperplasia. Atypical endometrial hyperplasia (adenomatosis) is characterized by pronounced proliferation of the endometrial glands in combination with cellular and nuclear atypia. Histological signs: - glands become larger than in simple AGE, they prevail over the stroma; - the increased complexity of the structure of the glands is expressed in an irregular form, pronounced branching and crowding of the glands; - glandular epithelial cells are large, basophilic or eosinophilic cytoplasm, cells do not respond to estrogens, mucus-forming epithelium; - nuclei are polymorphic, there is an alternation of hypo- and hyperchromic nuclei in one gland, the multiplicity of nuclei increases and the polarity of their arrangement is violated; - the stroma does not remain; - single cysts; - more often than in simple AGE, foci of squamous metaplasia are detected; - vessels of capillary or sinusoid types.

Endometrial hyperplasia develops with hormonal imbalance in the conditions of absolute or relative predominance of estrogens. The causes of endometrial hyperplasia can be anovulatory menstrual cycles, when the effect of estrogen is not balanced by the action of progesterone, hormone replacement therapy, estrogen-producing ovarian tumors (for example, granulosa cell tumors), polycystic ovary syndrome.

Cancer of the body of the uterus. An increased predisposition to cancer of the uterine body has been described in women with familial breast cancer. Cancer of the uterus is part of Lynch Type II syndrome, a hereditary disease that also includes colon cancer without polyposis and ovarian cancer. The tumor usually grows exophytic, forming polypous outgrowths in the uterine cavity, less often it diffusely affects the entire endometrium and the myometrium thickness grows. The most common histological form of endometrial cancer is endometrioid adenocarcinoma, which can be highly, moderately, and poorly differentiated. Rare forms of adenocarcinoma include papillary serous cancer and clear cell carcinoma of the uterus. Adenoacanthoma, adenosquamous cancer are less common. The first metastases in the regional lymph nodes. Approximately 40% of patients with stage IV cancer have lung metastases.

It occurs more often during menopause. The process begins at the corners and the bottom of the uterus. According to the degree of maturity, high-, moderate- and low-differentiated adenocarcinomas are distinguished. Highly differentiated adenocarcinoma is formed by prismatic cells. Mitoses are rare. The close arrangement of the glands, not delimited by basement membranes and capillaries, is characteristic. A moderately differentiated adenocarcinoma is formed by randomly alternating glandular-papillary and solid sections, consisting of large basophilic light and hyperchromic cells, with a significant number of mitoses, including pathological ones. Low-grade adenocarcinoma is characterized by a predominance of sites of a solid structure. It is formed by homogeneous relatively large light cells with large nuclei and an abundance of pathological mitoses.

Uterine leiomyoma is a benign tumor from the smooth muscle cells of the myometrium (leiomyoma). Myoma cells, like normal myometrium cells, are sensitive to estrogens, so it is not surprising that this tumor usually grows in women of childbearing age. Uterine fibroids almost never occur before puberty, and after menopause smooth muscle cells die, the tumor shrinks and scleroses. Depending on the relation to the layers of the uterus, submucous, subserous and intramural leiomyomas are distinguished. Histological varieties of leiomyomas: simple, cellular, epithelioid, bizarre, mitotically active, lipoleiomyoma.

Uterine leiomyoma is a benign tumor of smooth muscle tissue, it is one of the most common tumors of the reproductive system and is observed in 15-30% of women. Common leiomyoma is a benign tumor consisting of smooth muscle cells with varying amounts of fibrous tissue. The bundles of muscle cells are intertwined, fibrous tissue is often invisible in small tumors, in large tumors it can replace a significant part of muscle mass. In some tumors, muscle cells are swollen, their nuclei are polymorphic. These changes are most often associated with pregnancy or progesterone therapy.

Endometriosis is a pathological process characterized by the formation of ectopic foci of endometrial tissue. There are two main variants of endometriosis: internal (adenomyosis) and external. Adenomyosis is characterized by the presence of endometrial tissue in the thickness of the myometrium. External endometriosis is characterized by the presence of endometrial fragments in any tissue outside the uterus. The place of the most frequent localization of endometrioid foci is the ovaries.

The following factors are considered as the cause of endometriosis: 1) metastasis of normal endometrial tissue and ectopic places (metastatic theory), 2) metaplastic development of endometrial tissue in an ectopic place (metaplastic theory), 3) a combination of metastatic and metaplastic processes when metastasis of an endometrial tissue site causes corresponding metaplastic response of local tissues. With ovarian endometriosis, pelvic peritoneum is involved in the pathological process quite often. The lesions in typical cases are represented by multiple reddish-cyanotic spots, sometimes nodular, papillary and cystic endometrioid structures are formed. The morphological diagnosis of endometriosis is based on the microscopic detection of ectopic endometrial epithelium, necessarily in combination with elements of the endometrial stroma. The structure of endometrial tissue may vary depending on the degree and nature of its response to the hormonal background of the menstrual cycle. In the vast majority of foci of endometriosis, cyclic changes of the glandular epithelium and stromal cells are expressed to one degree or another. Macrophages containing hemosiderin and lipids (hemosiderophages and xanthomic cells) predominate in the cell infiltrate, multinuclear necrophages (such as cells of foreign bodies) are found, neutrophilic granulocytes, lymphocytes and plasma cells are present in different numbers. In one way or another, the phenomena of fibrosis are expressed.

Diseases of the fallopian tubes.

Inflammatory diseases of the fallopian tubes (salpingitis) can be caused by a diverse flora, mainly bacterial. Typically, the spread of pathogens occurs ascending from the uterus, hematogenous and lymphogenous infections are much less common. Acute serous salpingitis usually does not cause severe clinical symptoms. Swelling and hyperemia of the pipe wall are observed macroscopically. On microscopic examination, edema, plethora, and moderate leukocyte infiltration are usually limited to the mucous membrane. In acute purulent salpingitis, the fallopian tube is thickened, with a dull surface, covered with fibrinous-purulent exudate. Pus is released from the lumen of the tube. Sharp hyperemia, edema and infiltration of the tube wall by polymorphonuclear leukocytes are microscopically observed. Characteristically severe damage to the tube epithelium, its degeneration, necrosis, desquamation. Chronic salpingitis often develops after acute salpingitis. The formation of adhesions on the outer surface of the fallopian tube and between the folds of the endosalpinx is determined; microscopically in all layers of the tube - varying degrees of severity, lymphomacrophagous infiltration and sclerosis, atrophy of the muscle layer and epithelium gradually progresses. With obliteration of the lumen of the tube, an accumulation of purulent exudate (pyosalpinx) or transudate (hydrosalpinx) can be observed in it, which is accompanied by an expansion of the lumen, initially hypertrophy, and later - atrophy of the muscular and mucous membranes. Tubal tubal tuberculosis develops with the hematogenous spread of mycobacterium tuberculosis. A productive reaction with the formation of granulomas is characteristic. Inflammation is accompanied by a pronounced adhesive process with the formation of obstruction of the pipes.

An ectopic (ectopic) pregnancy develops when a fetal egg is implanted outside the uterine cavity (fallopian tubes, cervix, peritoneum). The majority of ectopic pregnancies occur in tubal pregnancy, which spontaneously terminates before a 5-6-week period by tubal abortion or rupture of the fallopian tube. The morphological diagnostic sign of tubal pregnancy will be the detection of a decidual reaction in the wall of the fallopian tube and chorionic villi and / or extravillous trophoblast cells in the lumen or in its wall.

Ovarian disease. Inflammatory diseases of the ovary. Oophoritis - inflammation of the ovary - is almost always observed simultaneously with salpingitis, it has the same etiology and infection paths. Macroscopically, in acute inflammation, the ovary is edematous, full-blooded, in chronic inflammation, adhesions form on the surface of the ovary. Infiltration by polymorphonuclear leukocytes is observed microscopically, mainly in the superficial parts of the ovary, the formation of microabscesses is possible, with chronic inflammation, foci of fibrosis in the stroma and fibrous adhesions on the surface of the ovary are also determined. Ovarian cysts. Follicular cysts and cysts of the corpus luteum. Atresia of the tertiary follicles may be accompanied by cystic expansion of their lumen with the formation of cystically-atresized follicles. If the diameter of such a follicle exceeds 2.5-3 cm, they are called a follicular cyst. The inner surface of the follicular cyst is light gray, smooth, shiny, the lumen is filled with a clear transparent liquid. The wall of the cyst is lined with a layer of granulosa, under which the theca cells are determined, often with signs of luteinization. The corpus luteum cyst has a rim of bright yellow scalloped tissue, which is represented by luteinized granulosa cells and theca-luteal cells. Cysts may undergo self-regression. In the end, a fibrous body forms at the site of the follicular cyst, and a white body at the site of the corpus luteum cyst.

Diseases of the mammary glands. Inflammatory diseases of the mammary gland: acute and chronic mastitis, ectasia of the ducts of the mammary gland. Due to trauma or inflammation, fatty necrosis can develop in the breast tissue.

Benign breast tumors: fibroadenoma (intra- and pericanalicular), phyloid tumor, intraductal papilloma.

Fibrocystic diseases (cystic mastopathy, cystic breast disease): a simple fibrocystic change, fibrocystic change with epithelial hyperplasia, sclerosing adenosis, radial scar.

Breast cancer can be hereditary with mutations in the brca1 and brca2 genes, as well as familial forms of cancer that are not associated with these genes. The following histological forms of breast cancer (BC) are distinguished: non-specific cancer (non-infiltrating cancer - intraductal and infiltrating ductal cancer), as well as specific cancer (non-invasive and invasive lobular cancer, Paget's disease, medullary cancer, colloid cancer, tubular and adenocarcinoma). .

To select targeted therapy, breast cancer immunophenotype is determined by immunohistochemical determination of estrogen receptors, progesterone receptors and epidermoid growth factor receptor (HER2 / NEU). Low HER2 / NEU fluorescence in situ hybridization (FISH) is used. The following breast cancer immunophenotypes are distinguished:

Luminal type A (30-45% breast cancer, estrogen-dependent non-aggressive tumors, overexpression of hormonal receptors, no HER2 protein, best prognosis).

Type B (14-18% of breast cancer, estrogen-dependent aggressive tumors, amplification of the HER2 oncogen is expressed, significantly worse prognosis).

HER2-positive subtype (8-15%, estrogen-independent aggressive tumors, amplification of the HER2 oncogen, increased likelihood of a negative outcome of the disease).

Triple negative (27-39% breast cancer, estrogen-independent aggressive tumors, there is no excess expression of HER2 protein receptors, the worst survival rates).

Metastasis - lymphogenous metastases to regional lymph nodes and hematogenous metastases to distant organs - in the lungs, liver, bones and adrenal glands. Brain metastases are fatal.

4. Lesson plan

1) To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations.

1. Breast cancer. Describe the Breast Cancer macropreparation. To study the size of the mammary gland and the type of tumor growths in it.

2. Glandular endometrial hyperplasia. Describe the glandular hyperplasia of the endometrium. Pay attention to the size of the uterus, thickness, color and focal changes in the uterine mucosa.

3. Septic endometritis. Describe the septic endometritis macropreparation. To study the size and texture of the uterus, thickness, texture and color of its mucous membrane.

4. Leiomyoma. Describe the macropreparation “Leiomyoma”. To study the size of the uterus, thickness, color and focal changes in the myometrium. Describe the size, quantity, shape, color of nodular formations.

2) Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations.

1. Invasive lobular breast cancer. Pay attention to the location of the atypical epithelium, the contents of the ducts; size and shape of cells and nuclei, mitotic activity of cells, invasive tumor growth. Draw a micropreparation, identify pathological changes.
2. Glandular endometrial hyperplasia. Sketch micropreparation glandular endometrial hyperplasia. Pay attention to the thickness of the mucous membrane, the number, size and shape of the glands, the height and mitotic activity of the gland epithelium. Cellularity and blood supply of stromal vessels. Designate pathological changes, determine the type of glandular endometrial hyperplasia according to the international classification.
3. Endocervicosis. Describe the micropreparation Endocervicosis. Pay attention to the nature of the epithelium covering the mucous membrane of the vaginal part of the cervix; proliferation under the integumentary epithelium of glands resembling uterine glands. Localization and cellular composition of the infiltrate. Draw a micropreparation, identify pathological changes.
4. Leiomyoma. Describe the micropreparation "Leiomyoma". Pay attention to the location, shape and structure of the glands in the fragments of the mucous membrane; size and shape of cells and nuclei, mitotic activity of gland cells, development and blood supply of stromal vessels; secondary changes in tumor tissue; blood clots in the drug. Draw a micropreparation, identify pathological changes.
5. Tubal pregnancy. Describe micropreparation tubal pregnancy. Pay attention to the thickness of the mucous membrane, muscle membrane of the fallopian tube. Find and describe focal hemorrhages in the stroma, the presence in the lumen and wall of the fallopian tube of chorionic villi covered with trophoblast. Draw a micropreparation, identify pathological changes.

3) Solve the following situational problems (case study) using the tutorial.

Situational tasks (case study).

Case 1

A young woman complained of spotting from the genital gap. When examined in the cervical region, a whitish tuberous formation without clear boundaries, growing in the underlying tissue. On palpation - it bleeds.

1. What kind of education is this? 2. What is its possible histological structure? 3. What is the forecast?

Case 2

A 22-year-old woman complains of copious discharge from the genital fissure and pulling pains in the lower abdomen. During the examination: on the cervix uterus a defect of 0.4X0.6 cm in size, bright red in color with a fine-grained surface.

1. Identify possible pathology, microscopic changes, prognosis.

Case 3

5 months after the birth, the woman's belly size increased, her menstrual function was disturbed, her uterus was enlarged. When scraping from the uterine cavity, formations are selected that resemble a bunch of grapes, consisting of edematous, translucent rounded formations.

1. Make a diagnosis, describe the microscopic structure, determine the prognosis.

Case 4

The woman was admitted with profuse uterine bleeding. When scraping, bloody, sponge-like masses were obtained. In the lungs - metastases. Histologically - chorionepithelioma.

1. Describe the histological structure and pathogenesis.

Case 5

The woman was admitted with profuse uterine bleeding. On examination: the uterus is enlarged, tuberous. Curettage gave only a temporary effect. Uterine extirpation done. In the body of the removed uterus there are several whitish dense nodes with clear contours located in different layers of the uterus.

1. Make a diagnosis and prognosis. 2. Describe the microscopic structure of the tumor.

4) Answer the following questions of the current test control.

QUESTIONS OF THE CURRENT TEST CONTROL:
Select all correct answers.

1. TYPE OF HYPERPLASIA OF THE GLANDS OF ENDOMETRY
1) gestational
2) embryonic
3) pathological
4) compensatory

2. Gland hyperplasia. Endometrium is due to an increase in level.
1) estrogen
2) progestogens

3. CERVICAL CANCER RISK FACTORS
1) late first birth
2) early onset of sexual activity
3) inability to bear children
4) chronic genital tract infections
5) sex with multiple partners

4. RISK FACTORS OF MALIGNANT TUMORS OF THE OVARIAN
1) late first birth
2) early onset of sexual activity
3) inability to bear children
4) chronic genital tract infections
5) sex with multiple partners

5. THE MOST FREQUENT HISTOLOGICAL FORM OF THE CERVICAL CANCER IS
1) adenocarcinoma
2) squamous cell carcinoma

6. THE MOST COMMON REASONS FOR THE DEVELOPMENT OF UTERINE DISEASES ARE
1) infection
2) endocrine disorders
3) tumor transformations
4) complications of pregnancy and childbirth

7. MOST SIGNIFICANT FACTORS PREPARING FOR INFECTION OF UTERINE CAVITY
1) colpitis
2) placental residues after childbirth
3) the remains of the fetal egg after spontaneous or induced abortion

8. ENDOMETRY AND MYOMETRY ARE RESISTANT TO INFECTION THANKS
1) the secretion produced by the endometrial glands
2) active physiological change of epithelial cells
3) the protective barrier of the endocervix (Kristeller cork)

9. IN MOST CASES, CHRONIC ENDOMETRITIS IS A DISEASE
1) primary
2) secondary

10. THE MOST FREQUENT TUMOR OF THE UTERINE BODY IS
1) fibromyoma
2) leiomyosarcoma
3) endometrial polyp

11. TYPES OF ENDOMETRY GIVING THE BEGINNING OF POLYPES
1) atrophic
2) functioning
3) hyperplastic

12. DURING PREGNANCY, LEUOMYOMAS OF THE UTERUS MAY
1) malignant
2) grow rapidly in size
3) undergo reverse development and calcification

13. FIBROMIOMA GROWTH FORMS
1) diffuse
2) subserous
3) submucous

4) polypous
5) intramural
6) transmural

14. MACROSCOPIC CHARACTERISTICS OF FIBROMIOMA
1) round shape
2) gray-white
3) fibrous appearance
4) massive sizes
5) with clear boundaries
6) with extensive areas of necrosis and hemorrhage

15. CONDITIONS PREPARING FOR THE DEVELOPMENT OF CANCER ENDOMETRY
1) endometrial hyperplasia
2) chronic HPV infection
3) long-term estrogen treatment
4) multiple uterine fibroids
5) ovarian secretion tumors

16. LEIOMIOSARCOMES ARISE
1) de novo
2) with malignancy of leiomyoma

17. DISTINCTIVE FEATURE OF LEYOMIOSARCOMA
1) fast growth
2) recurrence after surgical removal
3) metastasis to the ovaries (Kruckenberg metastases)

18. INTRAMURAL LEIOMIOMAS CAN LEAD TO
1) menometrorrhagia
2) abortion
3) torsion of the legs and necrosis with the development of an acute abdomen

19. MACROSCOPIC CHARACTERISTICS OF ENDOMETRY IN IRON HYPERPLASIA
1) loose
2) thickened
3) easily torn away
4) spongy, dark red

5. List of recommended literature:

Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. “Pathology. Quick Review and MCQs” Harsh Mohan, 2004.
2. “Textbook of Pathology ” Harsh Mohan, 2002.
3. “General and Systemic Pathology” Joseph Hunter, 2002.
4. “General and Systematic Pathology ” Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2th).
5. “Histology for Pathologist” Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2th).
6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
7. “Muir’s Textbook of Pathology” Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14th).
8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3th).
9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).
10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).
13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th).
14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.
15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
16. “Short lectures on pathology” Zagoroulko A., 2002
17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.
18. “General pathology” Dr. Fatma Hafez, 1979.
19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

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