REVIEW ARTICLE

CONTENTS

Uterine Leiomyoma in women of reproductive age: A systematic review

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ABSTRACT

Aim: The objective of this literature review was to determine the current aspects of the clinic, diagnosis and treatment of uterine leiomyoma in women of reproductive age.

Materials and Methods: Pubmed, Google Scholar, Web of Science, and Scopus databases were used to search for materials on current aspects of the clinic, diagnosis, and treatment of uterine leiomyoma in women of reproductive age.

Conclusions: Women who have a pregnancy in the background of leiomyomas of the uterus, constitute a high-risk group for the occurrence of obstetric and perinatal complications, therefore, in the most dangerous periods of pregnancy, hospitalization in a specialized obstetrical hospital is recommended. It is advisable to exclude the tactics of passive surveillance of women of childbearing age with leiomyoma of the uterus. Women of childbearing age with leiomyoma of the uterus are recommended to carry out organ-preserving operations in the volume of leiomyomectomy in order to preserve the reproductive function of the woman.

KEY WORDS: leiomyocyte, cytokines, growth factors, hormone-dependent canals

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INTRODUCTION

Leiomyoma of the uterus is a hormone-dependent benign tumor that develops from the smooth muscle tissue of myometrium and connective tissue, which is one of the most common tumors of female genital organs.

In the structure of gynecological morbidity, leiomyoma ranks second afte inflammatory processes of the uterus and its appendages [1]. The literature provides data on which every 5th woman in the world is ill with leiomyoma of the uterus [2]. It has been established that 20-50% of women of reproductive age suffer from this pathology, although the true morbidity is much higher, since only half of the patients have symptoms associated with leiomyoma [3]. It was believed that leiomyoma was a disease of premenopausal age, because the maximum percentage of the incidence was due to this period of life of a woman.

However, more recently, the facts of "rejuvenation" of uterine leiomyomas are becoming more common, more frequent cases of the disease occur in 18-22-year-old women [4].

Moreover, in most cases, not only one, but several tumor nodes of different sizes appear in the wall of the uterus. If one node appears, then wait for both the second and third ones.

Therefore, doctors call leiomyom a multiple tumor. So, as the main cause of the disease – a hormonal shift, leiomyomas almost never bother young girls and women in menopause [5]. It is suggested that 80% of all women between the ages of 30 and 40 have a high risk of leiomyoma in the uterus . Unfortunately, in 20-30% of cases, the latter is an etiological factor in infertility and another 15-30% causes miscarriage of pregnancy [6]. It is also worth noting the fact that 70-80% of all gynecological interventions occur in this particular disease [7]. After the onset of menopause, leiomyoma of the uterus is regressed [8].

AIM

The objective of this literature review was to determine the current aspects of the clinic, diagnosis and treatment of uterine leiomyoma in women of reproductive age.

MATERIAS AND METHODS

Pubmed, Google Scholar, Web of Science, and Scopus databases were used to search for materials on current aspects of the clinic, diagnosis, and treatment of uterine leiomyoma in women of reproductive age.

REVIEW AND DISCUSSION

This disease was included in the International Classification of Illnesses X, called leiomyoma, and not the "myoma" of the uterus, although the latter term is more common in clinical medicine as the generalized name of benign tumors of myometrium, and the term "leiomyoma" was considered to be a histological term for a long time (for tumors , where the stromal fibroblastic component is completely absent, unlike the "myoma" where it is present, but to a lesser extent, and "fibromyom", where the latter is substantially expressed). The achievements of the world's medical genetics over the last two decades have proven that uterine leiomyoma is a benign tumor of the uterus that develops from myometrium smooth muscle, regardless of the presence or absence of the fibroblastic component in the tumor site and the level of hormone dependence.

Mashal et al. [14] proved that each node of the uterine leiomyomy is a monoclonal tumor originating from one mutant smooth muscle cell of myometrium (mutant leiomyocyte): one mutant - one node, many mutant myocytes - diffuse leiomyoma of the uterus, multiple nodes. The role of genetic factors on chromosomal and gene levels in the etiology of uterine leiomyomas is beyond doubt, although not fully understood. The significance of chromosomal aberrations, namely, translocation ofregions of the 12th and 14th chromosomes, has been determined reliably: these sites change places [t (9)], deletions in the 7th chromosome [del (7) (q22q32), and also mutations of certain genes (NMGIC and NMGIY) responsible for encoding proteins that regulate DNA transcription.

SOME MECHANISMS OF MORPHOGENESIS OF UTERINE LEIOMYOMAS AT THE MOLECULAR CELLULAR LEVEL

The main mechanisms of pathological cell proliferation, hyperplasia and neoplasia in the organs of the female reproductive system are schematically depicted in Fig.1.

As shown in Fig.1, at present, at least three main mechanisms of activation of signaling pathways stimulating leiomyocyte to pathological growth and division [8] have been clarified.

One of them involves cytokines that regulate proliferation, cell growth and apoptosis (interferon alpha, some interleukins, tumor necrosis factor, etc.) [9, 13]. The nuclear transcription activation factor (NF-kB) coming from the cytokines is a terminal cytoplasmic signal conductor. Penetrated into the nucleus of the cell, it includes the genes needed for active cell division (see Fig. 1).

The second way of stimulation of proliferation is closely related to growth factors that are involved in the processes of cell division of oncogenes and the factors that stimulate the formation of vessels necessary for tumor growth. The most potent cell division stimulants are: epidermal growth factor, insulin-like growth factor (type 1), epithelial and fibroblast growth factors. The most potent stimulant for neoangiogenesis is endothelial growth factor. The third way of stimulating myocytes to pathological growth and division is through the hormone-dependent channels. Sex steroid hormones, namely: excessive estrogenic effects combined with insufficient progesterone - play one of the key positions. There are 3 main fractions of estrogen: estron (E1), estradiol (E2) and estriol (E3). In the first two - pronounced proliferative activity, in the third - is completely absent. It should be noted that E2 is evaluated as the main regulator of the cell cycle. Progesterone (P) is not related to the activation of cell division stimulation pathways, which is associated with proliferative activity (in this case, it acts as an antagonist of estradiol and estrone), but it has a direct and indirect effect on the hyperplasia of target cells of steroid hormones, including leiomyocytes. Its effects are realized after binding to the receptors of two types: the antiproliferative effect is realized through the alpha receptors (P - antipode E2 and E1), and through receptors beta -hyperplastic effect (see Fig. 1).

Consequently, estradiol and estrone can be converted into 2 forms of metabolites: 2-OH-estrone and 16-alpha-ON-estrone as a result of catalysis by various forms of the cytochrome P-450 enzyme and cause ambiguous effects on the proliferative activity of cells:

2-OH-estron normal cell division regulator and does not stimulate excessive (proliferative) activity, while 16-alpha-ON-estrone, on the contrary, is an inducer of over active cell growth and cell division and an estrogen agonist.

All three levels of activation of signaling pathways to the cell, which subsequently lead to pathological proliferation and tumor transformation, are interrelated. This means that the biological effects of hormones, growth factors and cytokines are interrelated. Thus, interferons are antagonists for the action of E2 and E1 on cells. Therefore, in the case of a local (in a pathological center) reduction of the factors regulating the antiproliferative activity of tissues, the influence of factors that stimulate

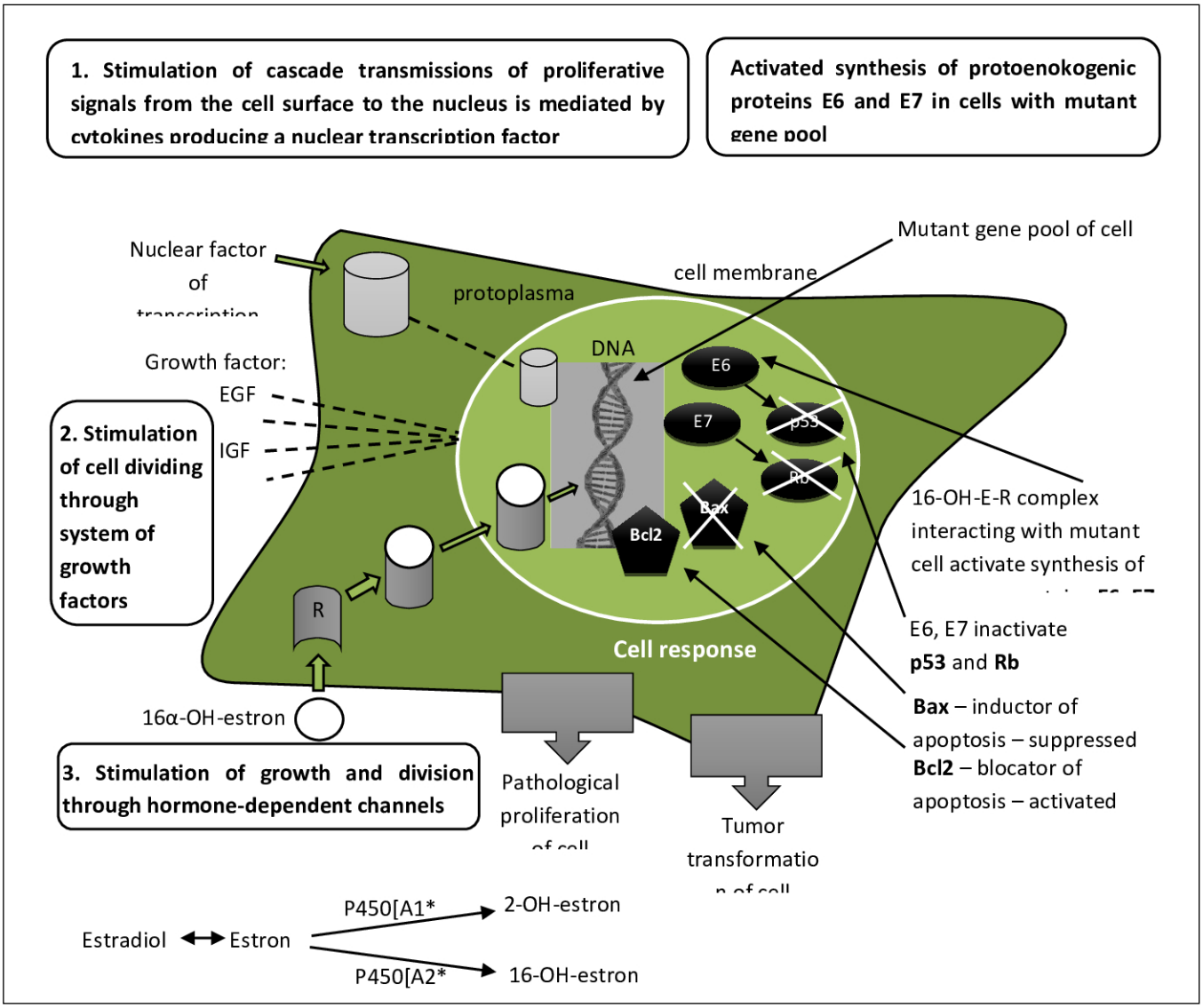

Fig. 1. The main mechanisms of pathological cell proliferation, hyperplasia and neoplasia in the organs of the female reproductive system.

proliferative activity - E2, E1, interleukin-8, epidermal growth factor, will necessarily be increased, which indicates the local growth of the proliferative potential. It is in the focus of the localization of the myoma that changes that determine the pathways of the disease - either in the direction of its progression, or in the direction of stabilization and regression. In this regard, the dynamics and intensity of the production of various cytokines, especially in the area of the myomatous nodes, as well as the microenvironment cells, may be of great importance [10].

MORPHOGENESIS AND MORPHOSTRUCTURE OF LEIOMYOMAS AT THE TISSUE LEVEL

Morphogenesis and further growth of the myomatous node undergo three stages of development:

1. Formation of active growth zone in myometrium near the microvessels in the form of accumulation of immature myocytes of different levels of differentiation, still unorganized in the beam. In contrast to the normal growth zone, in the active - much more intense metabolism and vascular-tissue perfusion.

2. Growth of a tumor without signs of differentiation (the node is identified only by microscopy).

3. Growth of the tumor with its differentiation and ripening (the node can already be determined macroscopically).

According to cytogenetic studies, all leiomyoma cells of the uterus are the descendants of a single maternal myogenic precursor cell. Recently, the possibility of formation of myoma nodules at the embryonic stage is confirmed by induction of mutagenic effects in the lessdifferentiated cells located in the zones of growth in myometrium (the "precursors of uterine leiomyo-

mas"). These focal points do not prolong themselves and activate only after menarche under the influence of sexual tropical and steroid hormones, growth factors and various altering factors of the endo- and exogenous environments of the body [11]. The factors that trigger this process are not fully understood, and they are numerous [12].

The original transformed mutant cell passes its properties only to its descendants. That is why multiple leiomyomas in one uterus are not clonally related [10].

According to the classical works of Uelzeko-Stroganova KP on morphology of the female reproductive system in the 30th years of the last millennium, the formation of precursors of myomatous nodes occurs at the embryonic stage. These data are confirmed in moder scientific research. The most recent confirmation is the theory of Fujii S., according to which smooth muscle cells of mesodermal origin are formed at the embryonic stage: from the 14th to the 30th week of fetal pregnancy [15]. That is why these undifferentiated cells can be exposed to many factors from the mother's body (trophic hormones, sex steroids, growth factors, etc.) and the environment (xenobiotics), epigenetic factors. These precursor cells (already with apparent mutations) are stored in myometrium and begin to grow under the influence of trigger factors, and predominantly after 28 menarche. The growth of these cells lasts for many years against the background of the marked activity of the ovaries under the influence of estrogens, progesterone and other hormones [11]. The development of uterine leiomyomas occurs in areas of growth, located mainly around the thin-walled vessel (active growth zones) [12].

Thus, leiomyoma of the uterus is a tumor of monoclonal origin. Primitive cells of myoma are transformed into myoblasts and fibroblasts. Active areas of growth are characterized by high levels of exchange and high vascular-tissue permeability. Leiomatous node repeats in its development parenchymal-stromal features of the layer of myometrium, from which it develops. Clinically, this is manifested by the fact that numerous leiomatous nodes in one uterus develop independently of each other. Their different growth rates are due to cell division, which is not clonally linked [10].

Most scientists [11,12,14] believe that leiomyoma of the uterus meets the criteria for a true tumor. The latter is confirmed by its monoclonal developmental nature, large size, autonomous growth of the tumor under the influence of growth factors and cytokines, activation of the process of angiogenesis, genetic instability. As a result of mutations, the accuracy of the reproduction of the genetic apparatus decreases, the mechanism of DNA repair is violated, the regulation of the cell cycle in the deformed cells changes, which leads to tumor progression.

There are two theories of the occurrence of precursors of uterine leiomyomas:

the first one is ontogenetic second - secondary somatic mutation in the normal cell myometrium under the influence of various factors [12].

The literature data of recent years (Khadartsev A. A. et al., 2013) reveal the value of stem cells (SC) in the onset and growth of uterine leiomyomas.

Researchers believe that the cause of adenomyosis, uterine leiomyomas is the tissue of the endometrium, which was formed even in the embryonic development period. Stem cells of the bone marrow can penetrate myometrium, resulting in diffuse changes and pathology development. The reason is the tissue cells of the endometrium, which remained in the myometrium during the period of intrauterine development. Such cells may be stem cells (SC).

Many years ago it was established that SC is located just in the basal layer of the endometrium. It is proved that there is an imbalance between the index of proliferation in different layers of the endometrium [9]. SC is a cell of mesenchymal origin that produces a growth factor.

According to most scientists, uterine leiomyoma occurs due to numerous somatic mutations in normal cells of myometrium, which leads to a gradual decrease in regulation and their growth. The tumor grows from the precursor cell in which the initial mutation occurred, and is a consequence of a violation of tissue homeostasis, supported by the balance between cell proliferation and the process of apoptosis.

The ontogenetic development of smooth muscle cells of ectodermal origin (digestive system, excretory system) runs faster than smooth muscle cells of mesodermal origin, to which the sexual system belongs. Undifferentiated cells, which are further differentiated into uterine smooth muscle cells, have a longer unstable period, during which they are exposed to various maternal factors (sex hormones, growth factors, viruses, toxins, etc.), resulting in their structure defects.

Such defective cells are referred to as precursor cells for the leiomatous node. The activity of cells begins with the presence of trigger factors. Regardless of when the precursor cell is formed, mutations in it promote the stimulation of proliferative processes.

Yakovleva IA, Kukute BG. (1979) have shown that in most myomatous nodes (about 75% of all cases) stroma predominates over parenchyma, and the mitotic activity of myocytes is practically equal to 0 (no mitoses are present), while in other nodes (about 25%) myogenic elements are more numerous. The second group of leiomyome clinically manifests itself in rapid growth, multiple nodes [10].

Hyperplasia of the connective tissue (fibroplastic) component of myometrium is secondary and can be expressed in the nodes of the uterine leiomyomas to a greater or lesser extent, depending on the nature and intensity of the harmful effects of the factors of the exogenous and endogenous environments of the organism on the genetic fund of its cells.

CLASSIFICATION OF UTERINE LEIOMYOMAS ACCORDING TO CLINICAL AND MORPHOLOGICAL TYPE AND ACTIVITY OF PROLIFERATIVE PROCESSES

1. Simple myoma, develops as benign muscular hyperplasia - slow growth, proliferative processes are not expressive.
2. Proliferating myoma is a vertebral tumor with high mitotic activity.
3. Pre-sarcoma - characterized by the presence of multiple atypical elements, heterogeneity of cell nuclei with large hyperchromic nuclei**.**

The tumor node repeats the morphological structure of one of the three layers of myometrium from which it originally developed. That is why, according to the histological evaluation of the tissues of the site, the composition of the parenchyma and stroma is different [11].

The morphostructure of the uterine fibroids is not constant. Depending on the number of muscle cells, the degree of their proliferation, differentiation and the presence of signs of atypia, there are three forms of uterine leiomyomas: simple, proliferative and pre-sarcomatous. According to clinical and morphological studies, leiomyoma of the uterus of a simple type is benign, inactive, slowly growing and contains mainly connective tissue elements [12]. This form of the tumor is characterized by a phenotypic transformation of myocytes, a decrease in blood flow in myometrium and myomatous nodes. The proliferative form of uterine leiomyomas is benign, active, multiple, rapidly growing by a tumor with a high proliferative potential and is often accompanied by proliferative processes in the endometrium, cervix, benign and malignant ovarian, mammary glands [9].

The precursor to proliferative distinguishes the presence of multicentric centers of proliferation with signs of atypia (heterogeneity of nuclei and cells, rarely occurring multicore cells with enlarged hyperchromic neclei of round or oval form). Cells with figures of mitosis often occur in it, including and atypical ones - this is an obvious path to the malignancy of the

uterine leiomyomas. In a simple leiomyoma of the uterus, there is virtually no mitosis, in

proliferative - mitotic activity is elevated. The latter form and, moreover, pre-sarcomatous are diagnosed much more often in women with a rapid growth of tumors, whereas for simple leiomyoma of the uterus more characteristic of slow and moderate growth rates.

A simple uterine leiomyoma is macroscopically represented by nodes of dense fibrous white fibers with clear boundaries. The sizes of knots can be different: from 1 to 4 cm in diameter. Placed nodes are mostly sub-serous and intramuscular. A simple leiomyoma is composed of smooth muscle bundles that are chaotic. The macroscopic node of the uterine leiomyomas has small dimensions, dense consistency, clear boundaries, pseudocapsule. With a pseudocapsule cut, the knot can easily be removed to its base, where the vessels are located.

By correlation of parenchyma and stroma in simple leiomyoma of the uterus, connective tissue elements predominate. Myocytes in these zones of myometrium show increased synthetic activity due to extracellular matrix production. Leiomyoma of a simple type is characterized by active processes of ripening, differentiation of tumor myocytes with their subsequent aging. Synthetic functions prevail, namely the production of elements of the extracellular matrix over proliferative activity. The stroma is presented of connective tissue with a large amount of collagen fibers. Vessels are sinusoid type [8].

Leiomyoma proliferative type is characterized by large sizes (up to 9 10 cm in diameter) of numerous nodes. Localization of nodules is predominantly submucosal, intramural.

Myocytes of these tumors are characterized by rapid proliferation (hyperplasia, hypertrophy). The consistency of the nodes of the fibroids is more softer than the simple ones. Cells of proliferation of tumor myocytes are localized in the perivascular spaces around the vessels. Proliferative myocytes are represented by large complex muscle cells with "juicy" hyperchromic nuclei. Stroma in proliferative zones contains predominantly connective tissue.

The tissues of myometrium outside the zones of nodes are represented by hypertrophied muscle fibers with a large number of cells of "active growth zones" [9].

THE MAIN MECHANISMS OF PATHOGENESIS OF LEIOMYOMAS

Of great importance in the pathogenesis of uterine leiomyomas is given to the central mechanism of regulation of menstrual function. It is believed that the basis of the development of leiomyomas of the uterus is the syndrome of psychoemotional stress, which leads to the disruption of adaptive-compensatory responses at different levels of the circulatory system of the hypothalamus-pituitary-ovary-uterus due to the violation of macro and microcirculation and tissue hypoxia. Emotional shocks or craniocerebral traumas often (in

73% of patients) are found in premorbid background in patients with leiomyoma of the uterus [10].

Violations of the functional state of the hypothalamic-pituitary system lead to changes in the cyclic secretion of the gonadotropin nucleus gonadotropin releasing-hormone (GnRH), as a result - the rates of peak exacerbations of luteinizing hormone (LH) and follicle stimulating hormone (FSH) increase substantially beyond the ovulatory, which can be observed in different phases of the menstrual cycle. Data on the content of blood in patients with LH and FSH in the presence of uterine leiomyomas are ambiguous, indicating several pathogenesis of uterine leiomyomas and corresponds to the assumption about possible (as an option of one of the ways) damage to the limbicoreticular structures of the brain that precede the development of the disease.

The largest number of studies is devoted to the study of estrogen-progesterone relationships in patients with leiomyoma of the uterus. There are also ambiguous data: possible variants of absolute, relative or combined hypertrophy; biphasic (2/3 cases) or single-phase (1/3 cases) menstrual cycles; with signs of progesterone deficiency (more often) or at elevated baseline gestagenic stimulation (less often). Significant prevalence of cases of systemic and local hyperestrogenism, rhythm changes and displacement of estrogen secretion peaks, estrogen conjugation disorders, qualitative changes in the concentration of various metabolites of these hormones, with the advantage of the formation of active substances in a proliferative relationship with metabolic fractions such as, for example, 16-α-Hydroxyestron and others.

Comparing the concentration of steroid hormones in the systemic and local (uterine vessels) streams in patients with leiomyoma of the uterus and endometrial hyperplasia, the researchers concluded that the myomatous nodes and the hyperplastic processes of the endometrium can serve as stimulators of relative local hyperteriogenicity and contribute to the formation of a false circle by type of stimulation of consumption, that is, the greater the mass of myometrium and endometrium, the greater the estrogen-consuming substrate, which is an active regulatory factor in the tumor system, is the tumor carrier [11].

The activity of the specific receptive state and the density of the receptor distribution of various sex hormones in the tumor (uterus) and, especially in the tumor itself, determines the final result of the hormonal action. In myomatous uterus, the higher activity of estrogen receptors compared with progesterone: the amount of bound estradiol reaches 60-65%, whereas in the normal uterus - only 37% [12]. Consequently, the content of estradiol and progesterone in the myometric uterus is higher than in normal myometrium, but lower than in the endometrium. Dependence of the content of the receptors of estradiol and progesterone on the magnitude and rate of growth of myomatous nodes was revealed: the highest content of estradiol receptors and the smallest - progesterone receptors were detected in nodes of large sizes with pronounced signs of proliferation; in the case of prolonged existence of myomatous nodes without a tendency to increase, the increase in the concentration of progesterone receptors in both the tissues of the host and in myometrium, while the content of estrogen receptors is relatively low, and in nodes it is lower than in the cells of myometrium surrounding them [10].

Under the influence of treatment with progestogens, increased receptor activity of all tissues [9]. However, it should be noted that in the case of rapid growth of uterine leiomyomas, as well as with the size of a leiomyomatous uterus in the size of 12 and more weeks of pregnancy, degenerative degenerative changes in the receptor apparatus occur, that the proliferative process (local or diffuse) loses sensitivity to the effects of hormones medication correction, which makes the latter inappropriate. Low-dose oral contraceptives, although not increasing the risk of developing uterine leiomyomas, do not prevent the growth of already existing nodes of the uterine leiomyomas.

Three pathogenetic variants of the development of leiomyomas of the uterus according to the level of primary lesions and perimondal background are commonly known:

The 1st variant is caused by violations of the function of the hypothalamic-pituitary system (with an increase or decrease in the production of gonadotropins);

2nd – develops against the background of impaired ovarian function due to inflammatory, atrophic and other changes;

3rd – occurs on the background of violations of the structure and function of the receptor apparatus of the uterus, which was usually the result of abortions, manual and instrumental studies of the uterus, the long-term use of the intrauterine devices (IUD), chronic metroendometrites.

However, it should be kept in mind that primary lesions may occur at any level, at any age and under the influence of numerous damaging factors on a different premorbid background, but sooner or later will necessarily induce the development of the disease all three levels, indicating that that the leiomyoma of the uterus is a systemic disease, not a local one. In his presence, psycho-vegetative, vegetative-vascular and metabolic-endocrine disorders are often observed.

The commonality of certain disorders in the regulation of the functional state of various organs of the

reproductive system leads to a frequent combination of uterine leiomyomas with hyperplastic processes, including endometrial cancer, endometriosis, ovarian polycystic ovary, fibro-cystic mastopathy, and other variants of hormone-dependent breast pathology, as well as cervical diseases (including dysplasia and neoplasia), with pathology of the thyroid gland, adrenals and other endocrinopathies with metabolic disorders. This circumstance emphasizes the necessity of oncological alertness and conducting of a comprehensive examination for the active detection of pre-tumor, benign and malignant neoplasms of various localizations in organs of the reproductive system, as well as related somatic diseases, especially of the liver, intestines, cardiovascular and urinary systems, in patients under dispensary supervision over leiomyomas of the uterus.

MUTUAL INFLUENCE OF UTERINE LEIOMYOMAS AND PREGNANCY

It is known that during pregnancy the growth of nodes of uterine leiomyomas is accelerated; Pregnancy contributes to a violation of hemodynamics in the tumor and, as a result, hemorrhages in the node, swelling of the tumor, necrobiosis and necrosis of the nodes, rupture of the capsule, secondary local and generalized suppurative complications. On the other hand, pregnancy with leiomyoma of the uterus is aggravated by various complications in the 1-st, 2-nd, 3-rd trimesters and in childbirth [8]. Thus, in the early stages of pregnancy, the likelihood of not being worn (due to the violation of the migration processes and the implantation of the pond egg in the localization of nodes near the isthmic parts of the fallopian tubes) increases; In the 1st and 2nd trimesters (due to increased tonus and excitability of myometric myometric biopsy, as well as hormonal dysfunctions), the risk of involuntary miscarriage is increased. Later leiomyoma of the uterus can cause premature birth, placental insufficiency, abnormalities in placental placement, formation of false posture and fetal pregnancy. Occasionally placement of nodes, especially in the cervical orthopedic region of the uterus, may become a barrier to birth per vias naturalis. Often, there is a weakness of labor, untimely departure of around-vegetative waters, hypoxia of the fetus in childbirth, and in the third period of childbirth, the delay of detachment of the placenta and its parts and hypotonic bleeding. In the postpartum period, women with leiomyoma of the uterus also significantly increase the risk of hypotonic bleeding, uterine subinvolution, endometritis, and damage to the trophic nodes of the uterine leiomyomas.

It occurs as a result of the fact that the cells of the uterus spontaneously begin to actively share. The causes of this phenomenon are not fully understood, but it is established that it is stimulated by hormones and is connected with all with increased secretion of estrogens [9] However, the normal content of estrogen and progesterone in the blood does not always clearly indicate the absence of leiomyomas. This is due to the fact that local changes in the level of estrogen in the uterus are not reflected or very slightly reflected in the content of hormones in this group in the blood [10].

However, from 25 to 50, the chances of getting it are quite large. It appears only in the reproductive period, when the ovaries produce estrogens. While ovaries are functioning, leiomyoma grows, at best - is in a stable condition. In postmenopausal leiomyomas no longer occur, and those small nodes, which by that time have already been in women, begin to decrease in size [11]. The fact that leiomyomas began to occur more often is connected with the obvious way of life - with high tempo, high loads. Especially sharply this probability grows during pregnancy, inflammatory processes of genital organs and general decrease of immunity [12].

Leiomyoma of the uterus can provoke hereditary predisposition, problems with the menstrual cycle, infertility, miscarriage, metabolic disorders (obesity, diabetes mellitus), multiple abortions, besides that, scientists have recently discovered that there is a direct relationship between leiomyoma uterus and stress [11]. The uterine leiomyoma predominantly runs asymptomatic, but sometimes women may be disturbed by excessive bleeding during menstruation, cycle disorders, abdominal pain or lower back. there are some signs that a woman may suspect in my uterus. Significant bleeding can lead to anemia, the tumorous node can compress adjacent organs, and sometimes there is pain, problems with urination, there are constipation [10].

If leiomyoma is not completely immersed in the wall of the uterus, then it is possible torsion of "legs"; in this case, a woman is at risk of necrosis of the tumor, its inflammation and rupture; from the outside it is manifested by bleeding, acute abdominal pain and a sharp increase in temperature.

Other problems of leiomyomas are problems with conception, pregnancy and delivery [9]. To determine the size of leiomyomas, an analogy with pregnancy is used. The treatment of leiomyomas depends on the age and the patient's plans. If during many years, leiomyoma does not increase, and the woman is not going to get pregnant, most doctors propose to treat the tumor conservatively. Hormonal drugs can slow down and, in some cases, stop tumor growth. Young women who want to have a baby, doctors advise not to delay the operation.

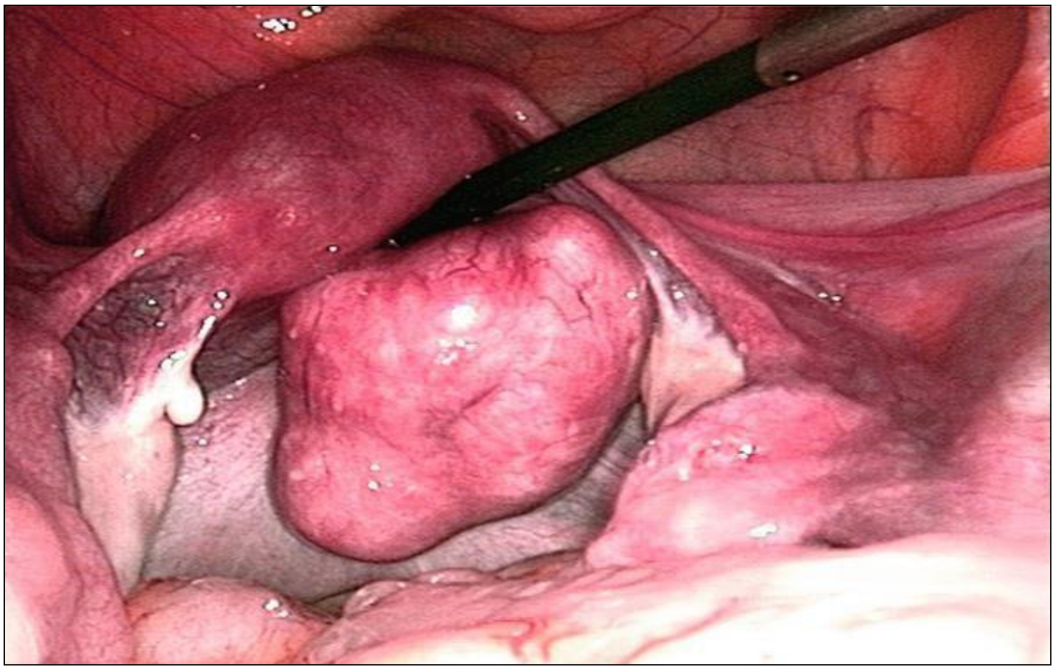

Fig. 2. The type of uterine leiomyoma during the review laparoscopy of patient O., 41.

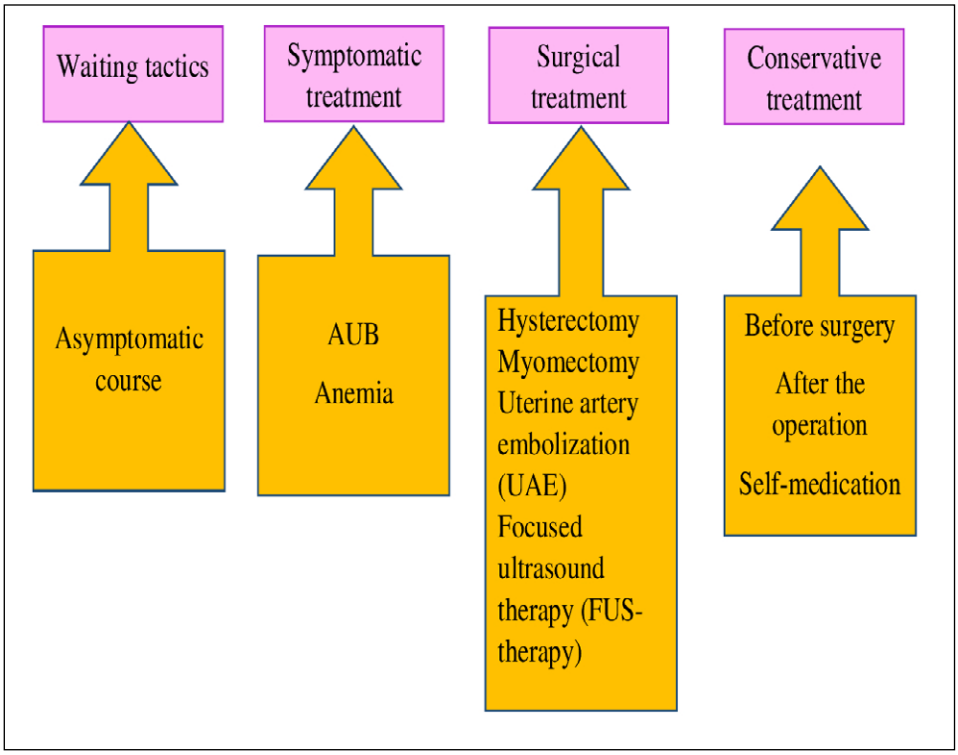

Fig. 3. Modern management of uterine fibroids

Organ-preserving surgery is a myomectomy, when only the nodes themselves are cut out: the uterus is not damaged while normal pregnancy is possible [8]. If the tumor nodes are very large, or too much, then you have to remove the entire uteru.

The impact on reproductive health is often accompanied by infertility, miscarriage, placental dysfunction (distress and growth retardation, pregnancy loss), a high probability of surgical delivery with removal of the uterus [14].

According to the recommendations of authoritative international publications [15], modern management of uterine fibroids involves both conservative and surgical treatment (Fig. 3).

It should be noted that treatment tactics should be selected individually based on the following factors:

- presence and severity of symptoms;
- the patient's desire to receive radical treatment;
- the desire to preserve childbearing function;
- the importance of preserving the uterus;
- infertility associated with deformation of the uterine cavity;
- complications during a previous pregnancy related to leiomyoma.

CONCLUSIONS

1. 80% of women between the ages of 30 and 40 have a high risk of developing uterine leiomyoma, which is the cause of infertility in 20-30% of cases.
2. In the tactics of patient management, an individual approach prevails with a combination of surgical and conservative methods of treatment, taking into account the age of the patients, their reproductive history, and the presence of concomitant genital and somatic pathology.
3. The use of modern conservative and operative methods of uterine leiomyoma treatment makes it possible to reduce the number of its complications, preserve the uterus and reproductive function of a woman, increase the effectiveness of uterine leiomyoma treatment, and the quality of life of patients.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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