ECRONICON OPEN ACCESS

EC GYNAECOLOGY
Guest Editorial

To the Question of the Etiopathogenesis of Uterine Fibroids

Linde Viktor Anatolievich[1]*, Tatarova NA[1], Reznik MV[2], Tarasenkova VA[2], Sadykhova EE[2] and Sobakina DA[2]

[1]Department of Obstetrics, Gynecology and Reproductology, FSBEI HE PSPbGMU Named After I.I. I.P. Pavlova, Russia
[2]Gynecological Service of St. Petersburg GBUZ "Alexander Hospital", Russia

***Corresponding Author:** Linde Viktor Anatolievich, Professor of the Department of Obstetrics, Gynecology and Reproductive, Medicine, Pavlov First St. Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, St. Petersburg, Russia.

Received: July 07, 2023; **Published:** May 31, 2024

Introduction

Uterine fibroids (MM) is one of the most common gynecological diseases of non-inflammatory origin [15,22]. At the same time, the significance of the inflammatory process in the formation and growth of myomatous nodes (MU) is currently being actively studied [21]. However, it is obviously secondary.

The frequency of occurrence of MM according to different authors varies in a very wide range. L.V. Adamyan., *et al*. [1] find that it is diagnosed in 30 to 35% of women of reproductive age. A.E. Karmon., *et al*. [17] believe that MM occurs in 70% of women. Considering that at the end of the last - beginning of this century, MM was found in 10 - 20% of women aged 30 years and older [3,4] and the continuing increase in the incidence of this pathology in young women, it seems that 35 - 40% more or less corresponds to the real situation.

Recently, it is considered proven that MM has a monoclonal character, i.e. grows from one mutated cell or one clone of cells, which is typical for tumors [12]. And, although not all authors agree with this point of view, even a term has appeared to refer to this process clonal expansion [9,12]. This, however, does not simplify our understanding of the pathogenesis of the disease. Recent studies have shown that myoma cells can arise as a result of re-peated mutations of various types, i.e. the concept of MM is not homogeneous. And its various types (kinds) can develop in different ways [20].

The cytological characteristics of the myoma cells themselves are also very diverse, and sometimes even contradictory. The main structural elements of the myomatous node (MU) are mature smooth muscle cells without signs of atypia [9]. Some MM cells, in contrast to mature myometrial cells, exhibit stem cell characteristics [25]. One type of MM with bizarre nuclei is a rare tumor with histological features that can mimic atypical mitoses, often leading to con-fusion with leiomyosarcoma [14]. Some authors observe an increase in the expression of both estrogen and progesterone receptors in myomatous nodes with a decrease in the expression of vitamin D receptors [11]. Other researchers find, on the contrary, their decline [5].

Of the more or less generally accepted risk factors for the development of MM, the most reliable are the Negroid race, early menarche, hereditary pre-disposition and no history of childbirth [1,10,20,24].

The relationship between MM and adenomyosis (AM) is interesting. Some authors believe that AM and MM arise independently of each other [18]. Others find that MM is much more common in AM patients than in the general population [24]. The possibility of such a scenario is indirectly indicated by the fact that the appearance and growth of endometrial glands and stroma in the myometrial tissue is accompanied by hypertrophy and hyperplasia of myocytes around heterotopic foci [16]. Others, including us, on the contrary, tend to consider the presence of MM as a risk factor for the development of AM [7,19,23].

It seems that there is no unequivocal answer to the question: what is uterine fibroids? - does not currently exist. Yes, according to N. Chabbert., *et al*. [13] MM can be a consequence of genetic disorders, and hormonal origin, and arise as a result of any intrauterine disorders. In other words, it is likely that under the term uterine fibroids we combine pathological processes similar in substrate and clinical manifestations, but different in origin and, at least, in the initial stages of pathogenesis [6].

Regarding the assessment of the significance of hyperestrogenism in the pathogenesis of MM, everything is not so simple. On the one hand, it seems obvious. And I would like to associate rejuvenation and an increase in the frequency of MM in modern women with it. Since the change in reproductive behavior with the delay of the first birth leads to a sharp increase in menstrual cycles (MC) in the lives of modern women. According to V.E. Radzinsky and A.M. Fuchs [8] the average number of MCs during the reproductive period of a woman's life has increased from 60 at the beginning of the 20th century to 400 at the beginning of the 21st. This inevitably leads to an increase in periods of relative hyperestrogenism. On the other hand, it is considered proven that in approximately 2/3 of patients the development of the tumor takes place against the background of hormonal ratios corresponding to the normal menstrual cycle [4].

There is strong evidence that estradiol, both systemically and locally, is involved in the increase in MU. Data on participation in the accumulation of progesterone in the nodes of cell mass are more controversial. Depending on the experimental conditions, progestins can either stimulate or inhibit cell proliferation *in vitro*. However, there is increasing evidence that MU cell proliferation is controlled jointly by estradiol and progesterone [13]. Indirect confirmation of the significance of the participation of progesterone in the pathogenesis of MM can be considered the effectiveness of the drug of the group of selective modulators of progesterone receptors ulipristal acetate in the treatment of patients with this pathology [2].

Bibliography

1. Adamyan LV., *et al*. "Uterine fibroids: diagnosis, treatment and rehabilitation". M (2015): 100.
2. Bezhenar VF., *et al*. "Innovative approaches to restoring reproductive function in patients with uterine myoma". *Obstetrics and Gynecology* 1 (2016): 80-87.
3. Beck U., *et al*. "Obstetrics and gynecology: Per. from English". M: GEOTAR MEDICINE (1997): 719.
4. Vikhlyaeva EM. "Guidelines for the diagnosis and treatment of uterine leiomyoma". M: MED press-inform (2004): 400.
5. Kustarov VN., *et al*. "Myoma uterus". SPb: SPbMAPO (2001): 32.
6. Linde VA., *et al*. "Uterine myoma and myomectomy". M: "Sweet Group" (2010): 94.
7. Linde VA and Tatarova NA. "Endometriosis". M: GEOTAR-Medicine (2010): 192.
8. Radzinsky VE and Fuchs AM. "Gynecology". GEOTAR-Media (2016): 988.
9. Savitsky GA and Savitsky AG. "What is uterine fibroids?". St. Petersburg: ELBI-SPb (2016): 216.

10. Tikhomirov AL., *et al*. "Etiology and pathogenesis of uterine leiomyoma - facts, hypotheses, reflections". AG-info 3 (2006): 3-8.

11. Al Hendi SP., *et al*. *The Journal of Clinical Endocrinology and Metabolism* 100.4 (2015): E572-E582.

12. Bulun SE., *et al*. "Uterine leiomyoma. Stem cells: Progestonic growth stimulation". *Reproductive Medicine* 33.5 (2015): 357-365.

13. Chabbert N., *et al*. "Myoma growth and medical options for treatment". *Fertility and Sterility* 102.3 (2014): 630-639.

14. Croce S., *et al*. *The American Journal of Surgical Pathology* 38.10 (2014): 1330-1339.

15. Donnez J., *et al*. "Ulipristal acetate versus placebo for fibroid treatment before surgery". *The New England Journal of Medicine* 366.5 (2012): 409-420.

16. Jiang JF., *et al*. "Aberrant expressed long non-coding RNA in the eutopic endometrium of patients with myoma adenomyosis". *European Journal of Obstetrics and Gynecology and Reproductive Biology* 199 (2016): 32-37.

17. Karmon AE., *et al*. "MicroRNAs in the development and pathobiology of uterine leiomyoma: is this evidence of support for future strategies for clinical intervention?" *Human Reproduction Update* 20.5 (2014): 670-687.

18. Khan KN., *et al*. "Participation factor-induced epithelial-mesenchymal transition of human hepatocyte growth in adenomyosis". *Biology of Reproduction* 92.2 (2015): 35.

19. Leyendecker G., *et al*. "Adenomyosis and endometriosis. Re-examination of their association to obtain a more complete understanding of the mechanisms of automatic trauma". *Archives of Gynecology and Obstetrics* 291.4 (2015): 917-932.

20. Mehine M., *et al*. *Proceedings of the National Academy of Sciences of the United States of America* 113.5 (2016): 1315-1320.

21. Owen C and Armstrong AY. "Clinical Management of Leiomyoma". *Obstetrics and Gynecological Clinics* 42.1 (2015): 67-85.

22. Soliman AM., *et al*. "Direct and indirect costs for uterine fibroids: systematic review of literature between 2000 and 2013". *American Journal of Obstetrics and Gynecology* 213.2 (2015): 141-160.

23. Shutders I., *et al*. "Activation of MAPK / ERK Cell by a signal cascade in the uterine cells of smooth muscles of women with Adenomyosis". *Reproductive Sciences* 22.12 (2015): 1549-1560.

24. Struble J., *et al*. "Adenomyosis: Clinical Overview". *Journal of Minimally invasive Gynecology* 23.2 (2016): 164-185.

25. Yn R., *et al*. "American association of clinical endocrinologists and American college of endocrinology - clinical practice guidelines for developing a diabetes mellitus comprehensive care plan – 2015". *The Journal of Clinical Endocrinology and Metabolism* 100.4 (2015): 601-606.

Volume 13 Issue 6 June 2024