**DOI 10.26724/2079-8334-2026-2-96-22-25**
**UDC 618.11–006.36–085.2:618.177**

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# REPRODUCTIVE HEALTH IN THE CONTEXT OF UTERINE FIBROIDS TREATMENT OPTIONS

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The purpose of this study was to compare the hormonal, endometrial, ultrasonographic, and Doppler changes after six-month postoperative adjuvant treatment (ulipristal acetate and a levonorgestrel-releasing intrauterine system) in women of reproductive age after myomectomy for uterine fibroids. Sixty women with uterine fibroids who underwent myomectomy (study group) and 30 women without uterine pathology as controls have been examined. After surgery, 30 patients received ulipristal acetate at a dose of 5 mg daily for two 3-month courses (subgroup A), while 30 patients were treated with a levonorgestrel-releasing intrauterine system (Subgroup B). Before and after six months of treatment, endometrial thickness, uterine dimensions, uterine volume, resistance index, and serum concentrations of follicle-stimulating hormone, luteinizing hormone, prolactin, estradiol, and progesterone were assessed. ELISA method using the Access 2 Beckman Coulter automated biochemical analyzer was used for hormone levels identification, while ER7-4D ultrasound system – to assess endometrial parameters. In the ulipristal acetate group, endometrial thickness increased from 6.0±0.23 to 9.9±0.21 mm ($p<0.001$), while estradiol, prolactin, and progesterone decreased significantly. Uterine volume decreased by 15.9 %, and the resistance index decreased from 0.93 to 0.72 ($p<0.05$). In the levonorgestrel-releasing intrauterine system group, endometrial thickness decreased from 6.3±0.45 to 5.0±0.19 mm, estradiol and progesterone decreased, and prolactin increased slightly. Uterine volume decreased by 13.4 %, and the resistance index decreased from 0.92 to 0.70 ($p<0.05$). Changes in linear uterine dimensions were not statistically significant in either group. Ulipristal acetate produced a more favorable six-month endometrial and hormonal profile, whereas the levonorgestrel-releasing intrauterine system provided comparable reductions in uterine volume and vascular resistance but was associated with endometrial thinning. Treatment selection should therefore be individualized according to reproductive plans, the need for contraception, and the primary therapeutic goal.

**Key words:** uterine fibroids, reproductive age, ulipristal acetate, levonorgestrel–releasing intrauterine system, abnormal uterine bleeding, adjuvant therapy, endometrial thickness, fertility planning.

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# РЕПРОДУКТИВНЕ ЗДОРОВ'Я У КОНТЕКСТІ ВАРІАНТІВ ЛІКУВАННЯ МІОМИ МАТКИ

Метою дослідження було порівняння гормональних, ендометріальних, ультразвукових та доплерометричних змін у жінок репродуктивного віку після шестимісячного післяопераційного застосування уліпристалу ацетату та внутрішньоматкової системи, що вивільняє левоноргестрел. До дослідження було залучено 60 пацієнток, які перенесли міомектомію, з міомою матки (досліджувана група) та 30 жінок без патології матки, які склали контрольну групу. Після операції 30 пацієнток отримували уліпристалу ацетат у дозі 5 мг на добу двома курсами тривалістю по 3 місяці (підгрупа А), а 30 пацієнткам було встановлено внутрішньоматкову систему, що вивільняє левоноргестрел (підгрупа В). До початку лікування та через 6 місяців оцінювали товщину ендометрія, розміри й об'єм матки, індекс резистентності, а також сироваткові рівні фолікулостимулюючого гормону, лютеїнізуючого гормону, пролактину, естрадіолу та прогестерону. У групі уліпристалу ацетату товщина ендометрія збільшилася з 6,0±0,23 до 9,9±0,21 мм ($p<0,001$), при цьому рівні естрадіолу, пролактину та прогестерону статистично значуще знизилися. Об'єм матки зменшився на 15,9 %, а індекс резистентності знизився з 0,93 до 0,72 ($p<0,05$). У групі внутрішньоматкової системи, що вивільняє левоноргестрел, товщина ендометрія зменшилася з 6,3±0,45 до 5,0±0,19 мм, рівні естрадіолу та прогестерону знизилися, а рівень пролактину незначно підвищився. Об'єм матки зменшився на 13,4 %, а індекс резистентності знизився з 0,92 до 0,70 ($p < 0,05$). Зміни лінійних розмірів матки в обох групах не досягли статистичної значущості. Таким чином, ацетат уліпристалу забезпечував більш сприятливий шестимісячний профіль ендометріальних та гормональних змін, тоді як внутрішньоматкова система, що вивільняє левоноргестрел, мала порівнянний вплив на об'єм матки та судинний опір, але супроводжувалася витонченням ендометрія. Вибір терапії повинен бути індивідуальним з урахуванням репродуктивних планів, необхідності контрацепції та основної мети лікування.

**Ключові слова:** міома матки, репродуктивний вік, уліпристала ацетат, внутрішньоматкова система з левоноргестрелом, аномальна маткова кровотеча, ад'ювантна терапія, товщина ендометрія, планування фертильності.

A strategically regulated modulation of reproductive activity can help prevent undesirable population surges while ensuring that fertility is preserved under favorable developmental conditions. Within this context, uterine fibroids (leiomyomas/myomas) represent a pressing concern, as they constitute one of the most common gynecological pathologies among women of reproductive age [12]. Although most fibroids remain asymptomatic, 25–30 % of women develop pronounced clinical manifestations, including abnormal uterine bleeding, pelvic pain, a sensation of heaviness, anemia, and dysfunction of the bladder or bowel. Leiomyomas are often detected incidentally during imaging performed for unrelated reasons, which may result in excessive diagnostic procedures or unnecessary interventions [2]. Although benign in nature, fibroids often cause symptoms such as abnormal uterine bleeding, pelvic pain, and infertility. In severe cases, they may necessitate hysterectomy [9], which remains the most common definitive treatment. However, this radical approach is not ideal for women of reproductive age, as it irreversibly eliminates fertility potential and significantly impacts quality of life. Moreover, while fibroids are non–malignant, evidence suggests that untreated or poorly managed cases may occasionally progress toward malignancy, thereby reinforcing the need for timely and effective intervention.

In recent years, contemporary medical paradigms have shifted toward organ–preserving approaches [10], emphasizing early diagnosis,

conservative management, and minimally invasive therapies [1]. Pharmacological strategies have gained increasing importance in this regard, particularly those that target the hormonal regulation of fibroid growth. Among these, selective progesterone receptor modulators [15], such as Ulipristal acetate have demonstrated significant therapeutic benefits. Ulipristal acetate exerts a proapoptotic effect on fibroid tissue, thereby reducing size and symptoms, while also modulating the hormonal environment to create favorable conditions for reproductive planning [5]. Another widely adopted modality is the levonorgestrel–releasing intrauterine system, which has been shown to suppress endometrial proliferation, reduce menorrhagia, and alleviate fibroid–related symptoms [11, 13]. Despite its effectiveness, however, concerns remain regarding its long–term impact on fertility due to endometrial thinning and its association with increased prolactin levels, which may complicate reproductive outcomes [13]. Given these considerations, there is a growing need for comparative studies evaluating conservative, fertility–preserving treatment options for uterine fibroids. Such analyses are particularly relevant for women of reproductive age who seek not only symptom relief but also preservation of their future fertility [6, 17].

**The purpose** of the study was to determine the safest and most effective therapeutic and contraceptive option for reproductive–age women with uterine fibroids myomectomy, in order to prevent recurrence, regulate hormonal balance, and preserve fertility potential after treatment.

**Materials and methods.** This study included a primary cohort of 60 women, aged 18 to 52 years admitted to the II Department of Obstetrics and Gynecology of Azerbaijan Medical University (Baku) between 2022 and 2024 (Group I). Inclusion criteria: women with a 1–7 year history of uterine fibroids requiring surgical intervention. Exclusion criteria: presence of intrauterine malignancy/metastases or any comorbid conditions associated with uterine myoma that could impact treatment outcomes. The control group (Group II) consisted of 30 reproductive–age women, with a mean age of 37.2±1.21 years, who exhibited no uterine pathology. Within Group I, patients undergoing augmentation therapy were further stratified into two subgroups. Subgroup A (n=30) received Ulipristal acetate (Esmya/Esmia, Gedeon Richter, Budapest, Hungary), administered in the form of 5 mg tablets. Patients ingested one tablet once daily for 6 months, starting on postoperative day 16. Subgroup B (n=30) underwent treatment with the levonorgestrel–releasing intrauterine system (Mirena, Bayer, Germany) administered 52 mg levonorgestrel with release rate approximately 20 mcg/day. Results were obtained after 6 months of IUD administration. Endometrial thickness was assessed using an ER7–4D ultrasound system, equipped with a multi–frequency transabdominal convex transducer (3.5 MHz frequency). Measurements were taken perpendicularly to the longitudinal uterine axis. Following myomectomy, postoperative therapy was administered as follows: Ulipristal acetate (ulipristal acetate, 5 mg) was administered orally, once a day, 2 courses of 3 months each, and LRSLevonorgestrel (20 mcg levonorgestrel–releasing intrauterine device) was inserted postoperatively, a month after enucleation. The ultrasound examination (USM) was performed both pre– and post–treatment. Hormonal profiling – encompassing follicle–stimulating hormone (FSH), luteinizing hormone (LH), progesterone, prolactin, and estradiol levels – was conducted via the Bioscreen MS 500 Macro ELISA method using the Access 2 Beckman Coulter automated biochemical analyzer. For statistical analysis, non–parametric integrity criteria were employed, including the Wilcoxon–Mann–Whitney test and Pearson's $\chi^2$ test, utilizing the EXCEL 7.0 and STATGRAPH 5.1 software packages (Microsoft, USA).

**Results of the study.** At baseline, the two treatment subgroups were broadly comparable for the measured hormonal and ultrasonographic parameters. Compared with the control group, estradiol was markedly elevated in subgroup A (593.0±32.83 vs 51.6±5.70 pg/mL) and subgroup B (511.6±31.46 vs 51.6±5.70 pg/mL). Baseline endometrial thickness was 6.0±0.23 mm in subgroup A and 6.3±0.45 mm in subgroup B, compared with 7.8±2.3 mm in controls. Baseline uterine dimensions and volume were also greater in both fibroid subgroups than in the control group (Table 1).

After six months of postoperative ulipristal acetate therapy (subgroup A), endometrial thickness increased from 6.0±0.23 to 9.9±0.21 mm ($p<0.001$), an increase of 65.0 %. Estradiol decreased from 593.0±32.83 to 164.0±15.92 pg/mL ($p<0.001$; 72.3 % reduction), prolactin decreased from 16.9±0.89 to 13.9±0.46 ng/mL ($p<0.01$; 17.8 % reduction), and progesterone decreased from 1.62±0.12 to 0.83±0.20 ng/mL ($p<0.001$; 48.8 % reduction). FSH increased from 6.3±0.41 to 9.7±0.29 mIU/mL, whereas LH decreased from 7.5±0.49 to 6.5±0.28 mIU/mL.

Ultrasonographic measurements in subgroup A showed reductions in uterine length from 62.3±1.41 to 59.7±1.32 mm (4.2 %), uterine width from 57.9±1.72 to 53.9±1.49 mm (6.9 %), and anteroposterior size from 52.8±1.64 to 49.8±1.55 mm (5.7 %); these dimensional changes were not statistically significant (all $p>0.05$). Uterine volume decreased from 87.0 to 73.2 mm³, corresponding to a 15.9 % reduction. The resistance index in the secretory phase decreased from 0.93 (0.87–0.96) to 0.72 (0.68–0.75) ($p<0.05$).

After six months of treatment with the levonorgestrel-releasing intrauterine system (subgroup B), endometrial thickness decreased from 6.3±0.45 to 5.0±0.19 mm (20.6 % reduction). Estradiol decreased from 511.6±31.46 to 404.5±23.64 pg/mL ($p<0.001$; 20.9 % reduction), while prolactin increased from 16.1±0.90 to 17.0±0.55 ng/mL ($p<0.01$; 5.6 % increase). Progesterone decreased from 1.8±0.32 to 1.0±0.25 ng/mL ($p<0.05$; 44.4 % reduction). FSH increased from 5.3±0.37 to 13.0±0.33 mIU/mL, and LH changed minimally from 7.1±0.39 to 7.0±0.35

mIU/mL. In subgroup B, uterine length decreased from 63.4±1.77 to 60.7±1.75 mm (4.3 %), uterine width from 57.7±1.81 to 54.8±1.83 mm (5.0 %), and anteroposterior size from 50.9±2.37 to 48.5±2.17 mm (4.7 %); these changes were not statistically significant (all p>0.05). Uterine volume decreased from 85.1 to 73.7 mm³ (13.4 %). The resistance index decreased from 0.92 (0.90–0.94) to 0.70 (0.66–0.74) (p<0.05).

Table 1

**Variations in clinical and hormonal parameters across study groups following adjuvant therapy (M±m)**

| Indicators | | group I (uterine myoma), N=60 | | group II (control), N=30 |
|---|---|---|---|---|
| | | subgroup A (Ulipristal acetate ), N=30 | subgroup B (levonorgestrel-releasing intrauterine system), N=30 | |
| Endometrial thickness, mm | Pre–treatment | 6.0±0.23 | 6.3±0.45 | 7.8±2.3 |
| | Post–treatment | 9.9±0.21*** | 5.0±0.19 | |
| FSH, mIU/mL | Pre-treatment | 6.3±0.41 | 5.3±0.37 | 6.6±0.47 |
| | Post-treatment | 9.7±0.29 | 13.0±0.33*** | |
| LH, mIU/mL | Pre-treatment | 7.5±0.49 | 7.1±0.39 | 9.4±0.61 |
| | Post–treatment | 6.5±0.28 | 7.0±0.35 | |
| Prolaktin, ng/ml | Pre-treatment | 16.9±0.89 | 16.1±0.90 | 16.4±1.58 |
| | Post-treatment | 13.9±0.46** | 17.0±0.55** | |
| Estradiol, pg/ml | Pre-treatment | 593.0±32.83 | 511.6±31.46 | 51.6±5.70 |
| | Post-treatment | 164.0±15.92*** | 404.5±23.64*** | |
| Progesterone, ng/mL | Pre-treatment | 1.62±0.12 | 1.8±0.32 | 1.5±0.14 |
| | Post–treatment | 0.83±0.20*** | 1.0±0.25* | |
| Uterine length, mm | Pre-treatment | 62.3±1.41 | 63.4±1.77 | |
| | Post-treatment | 59.7±1.32, p>0.05 | 60.7±1.75, p>0.05 | 54.1±1.12 |
| Uterine width, mm | Pre–treatment | 57.9±1.72 | 57.7±1.81 | 43.8±1.14 |
| | Post-treatment | 53.9±1.49, p>0.05 | 54.8±1.83, p>0.05 | |
| Anteroposterior size, mm | Pre-treatment | 52.8±1.64 | 50.9±2.37 | 36.4±1.18 |
| | Post-treatment | 49.8±1.55, p>0.05 | 48.5±2.17, p>0.05 | |
| Uterine volume, mm³ | Pre-treatment | 87.0 | 85.1 | 39.4 |
| | Post-treatment | 73.2 | 73.7 | |
| Resistance index (secretory phase) | Pre-treatment | 0.93 (0.87–0.96) | 0.92 (0.90–0.94) | 0.57–0.61 |
| | Post-treatment | 0.72 (0.68–0.75), p<0.05 | 0.70 (0.66–0.74), p<0.05 | |

Note: Statistical significance of post-treatment change relative to baseline: * – p<0.05; ** – p<0.01; *** – p<0.001.

Direct comparison showed that the reduction in uterine volume was slightly greater with ulipristal acetate than with the levonorgestrel-releasing intrauterine system (15.9 % vs 13.4 %). Both treatments reduced the resistance index significantly and produced small, nonsignificant reductions in uterine dimensions. The clearest between-treatment differences concerned the endometrium and hormonal profile: ulipristal acetate was associated with increased endometrial thickness and larger reductions in estradiol and prolactin, whereas the levonorgestrel-releasing intrauterine system was associated with endometrial thinning and a small increase in prolactin.

**Discussion**. The present findings demonstrate that both postoperative regimens were associated with reductions in uterine volume and vascular resistance, but their endometrial and endocrine effects differed. The modest reductions in uterine length, width, and anteroposterior diameter did not reach statistical significance, whereas the resistance index decreased significantly in both groups. Thus, the ultrasonographic data suggest broadly similar effects on uterine size and blood-flow resistance over six months, with a slightly larger reduction in uterine volume in the ulipristal acetate group. The greater decrease in estradiol observed with ulipristal acetate is consistent with the pharmacological activity of selective progesterone receptor modulators and with reports of reduced leiomyoma proliferation and increased apoptosis after ulipristal acetate exposure [5, 15]. In the current study, ulipristal acetate also reduced prolactin and increased endometrial thickness. These findings may be clinically relevant when treatment is selected for women who wish to preserve reproductive potential; however, pregnancy and live-birth outcomes were not measured, so a direct fertility benefit cannot be concluded from the present data.

The levonorgestrel-releasing IUD produced a smaller estradiol reduction and was associated with endometrial thinning, which is compatible with its local antiproliferative action on the endometrium and its established role in controlling heavy menstrual bleeding [11, 13]. The small increase in prolactin observed in this subgroup should be interpreted cautiously because post-treatment values remained close to the baseline range and the study did not assess ovulation or clinical reproductive outcomes. Its therapeutic value may therefore be greater when bleeding control and contraception are the principal goals. The significant reduction in resistance index in both subgroups indicates a change in uterine vascular resistance following treatment. Simultaneously, the absence of statistically significant changes in the three linear uterine dimensions suggests that a six-month interval may be insufficient to demonstrate major anatomical remodeling, or that volume and Doppler indices are more responsive than individual diameters. These additional findings extend the

comparison beyond hormonal and endometrial effects and support the use of combined biochemical, structural, and Doppler assessment in follow-up [17]

Treatment selection should remain individualized [14]. Current approaches to uterine fibroid management emphasize symptom severity, fibroid characteristics, safety, and reproductive plans [1, 3, 9]. In this cohort, ulipristal acetate showed the more favorable six-month endometrial and hormonal pattern, whereas the levonorgestrel-releasing intrauterine system provided comparable reductions in uterine volume and resistance index but caused endometrial thinning [7]. Because ulipristal acetate has recognized safety concerns requiring appropriate patient selection and monitoring, these results should not be interpreted as supporting unrestricted first-line use [4]. The study is limited by the modest sample size, six-month follow-up, absence of randomization details, lack of bleeding-volume and quality-of-life measures, and absence of pregnancy, recurrence, and long-term safety outcomes. Consequently, the findings support a comparative short-term physiological effect rather than definitive superiority for fertility preservation. Larger prospective studies with standardized symptom scores, liver-safety monitoring, and reproductive outcomes are needed [4, 8].

## Conclusion

After six months of postoperative treatment, ulipristal acetate produced a greater reduction in estradiol and prolactin, increased endometrial thickness, and reduced uterine volume. The levonorgestrel-releasing intrauterine system also reduced uterine volume and estradiol but was associated with endometrial thinning and a slight increase in prolactin. These findings suggest that ulipristal acetate may be more appropriate for women who wish to preserve reproductive potential, whereas the levonorgestrel-releasing intrauterine system may be preferable when contraception and control of uterine bleeding are the main treatment goals.

*Prospects for further research. Future prospective studies should evaluate the long-term safety, fibroid recurrence, menstrual outcomes, subsequent pregnancy and live-birth rates after ulipristal acetate and levonorgestrel-releasing intrauterine system treatment.*

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**Conflict of interest.** The authors have no conflicts of interest to declare.

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Article received: 8.04.2025