REVIEW

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Myoma Uteri

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ABSTRACT

Uterine fibroids, also known as leiomyomas or myoma uteri, are benign smooth muscle tumors that arise primarily in women of reproductive age. These tumors are hormonally responsive, with their growth strongly influenced by estrogen and progesterone. Myomas can be asymptomatic or cause a wide range of clinical manifestations including menorrhagia, anemia, pelvic pressure, urinary disturbances, and infertility. The etiology involves genetic mutations, notably in the mediator complex subunit 12 and high mobility group AT-hook 2 genes, and environmental factors such as exposure to endocrine-disrupting chemicals. Epidemiological studies reveal higher prevalence in African descent and familial aggregation. Diagnosis is often achieved through pelvic examination and ultrasonography, whereas magnetic resonance ımaging remains the gold standard for complex cases. Fibroids are classified using the Federation of Gynecology and Obstetrics system based on their location relative to the endometrial and serosal surfaces. Medical management includes non-steroidal anti-inflammatory drugs, antifibrinolytics, and hormonal therapies, though they primarily target symptoms rather than tumor size. Surgical options such as hysterectomy, myomectomy, and minimally invasive techniques such as magnetic resonance-guided focused ultrasound are indicated for refractory cases. Future directions emphasize early diagnosis, individualized treatment, and the use of fibroids as a model to explore novel therapeutic strategies, particularly those aiming to interrupt fibroid pathogenesis at the molecular level. Given their accessibility and relatively benign nature, fibroids also provide a valuable platform for testing emerging technologies in gynecologic care. As research advances, the paradigm is shifting from radical surgical intervention toward personalized, fertility-preserving treatments.

Keywords: Estrogen, Federation of Gynecology and Obstetrics classification, Leiomyoma, Myoma uteri, Uterine myomas

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INTRODUCTION

Uterine myomas, also referred to as leiomyomas or fibroids, are benign tumors originating from smooth muscle cells and fibroblasts, characterized by an abundance of extracellular matrix. These growths typically arise during a woman's reproductive years, between menarche and menopause, with their development and gene expression influenced by cyclical fluctuations of gonadal steroids, particularly estrogen and progesterone. Fibroids represent a significant health concern for women of childbearing age, often leading to substantial morbidity. Common clinical manifestations include heavy or prolonged menstrual bleeding, which may result in iron deficiency anemia, as well as social discomfort. In addition, fibroids can cause uterine enlargement, leading to urinary issues such

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as frequency, nocturia, or retention, and gastrointestinal disturbances such as diarrhea or constipation. Abdominal bloating or pain may also occur, although some women remain asymptomatic even when fibroids reach considerable size.[1]

EPIDEMIOLOGY AND ETIOLOGY, INCIDENCE, AND PREVALENCE OF MYOMA UTERINE

Myoma uteri is a benign smooth muscle tumor and usually develops from the uterine corpus but can also occur in the cervix, uterine ligaments, and rarely in the ovaries and tubules. Its racial and familial characteristics indicate the importance of genetic risk factors in pathogenesis.[2] Although the etiology is not clear, the role of steroid hormones released from the ovary is important. While myoma uteri is not observed before puberty, it is observed during the reproductive years when the level of ovarian hormones increases. Unopposed estrogen increases the incidence of myoma uteri. At the same time, any factor that decreases endogenous estrogen levels and increases progesterone levels (such as pregnancy and oral contraceptive use) decreases the incidence of uterine fibroids.[3] The development and growth of fibroids are related to estrogen, progesterone, and their associated growth factors and proteins.[2]

Studies have found that fibroid tissue is significantly more sensitive to estrogen than normal myometrial cells from the same patient.[4,5] Semi-quantitative immunohistochemical studies with estrogen and progesterone show the effect of these two hormones on tumor development.[6] While myoma uteri may grow with estrogen use, most fibroids shrink after the use of gonadotropin-releasing hormone (GnRH) agonists.[7] Progestins, hormone replacement therapy, clomiphene citrate use, and pregnancy can cause rapid growth and sometimes hemorrhagic degeneration.[1] Studies based on X chromosome inactivation demonstrated by glucose-6-phosphate dehydrogenase isoform expression and other techniques show that myoma uterine is the proliferation of a single smooth muscle clone.[8]

Tumor-specific chromosomal abnormalities are detected in approximately 40–50% of fibroids. Among these abnormalities, t(12-14), (q15;q23-24), del(7) (q22q32), trisomy 12 and 3q deletion are the most common. According to many studies, estrogen hormone supports the relationship between the growth of myoma uterine and tumor formation.[9] Estrogen exerts its physiological effects on the target cell by binding to specific nuclear receptors. These receptors are ERα and ERβ.[10]

Compared to normal myometrium, the expression of many genes (such as connexin 43 gap junction protein, type I and type III collagen, insulin-like growth factor-1 [IGF-1], parathyroid hormone-like, peptide, and progesterone receptor genes) is increased in myoma uteri tissue. Aromatase P450, an estrogen synthetase involved in the synthesis of estrogen from androgen, is involved in fibroid growth. It is suggested that GnRH agonist treatment inhibits aromatase P450 enzyme and causes regression of fibroids.[11,12] Estrogen exerts a mitogenic effect on fibroid cells through phosphorylation of intracellular proteins (growth-related protein, phosphatidylinositol 3-kinase, phospholipase C, platelet-derived growth factor [PDGF], etc.) by protein kinase. Estrogen mediates the release of growth factors such as endothelial growth factor (EGF), IGF, and PDGF. Progesterone also plays an important role in the pathogenesis of myoma uteri.

Progesterone stimulates mitotic activity and proliferation in myoma.[13,14] The most common angiogenic factors in myoma uteri are vascular EGF and adrenomedullin. These factors are found in higher concentrations in myoma tissue than in normal myometrium. Heparin-binding growth factor (HBGF) is associated with fibroid formation and is mitogenic in fibroblasts and smooth muscle cells. HBGF is more potent than EGF and shows more affinity for EGF receptors.[15] Fibroid tissue contains an abundant extracellular matrix, which is why these tumors are also called fibroids. Although myoma uteri has a low mitotic index, it can grow rapidly. This suggests a mechanism other than mitosis for growth, namely alteration and remodeling of the extracellular matrix content.[16] The factors involved in the etiopathogenesis of myoma uteri are schematized in Figure 1.

Fibroids have been linked to events that occur during the fetal period in the formation of the uteri. The development of smooth muscle cells of mesoderm origin (up to 30 weeks of gestation) is slower than those of endoderm origin (up to 12 weeks of gestation). Therefore, these undifferentiated cells of mesodermal origin have a longer labile period during the fetal period. It has been suggested that fibroid progenitor cells are formed during this period as a result of the influence of some unknown factors and grow in the post-menarcheal period when both estrogen and progesterone are dominant.[17,18]

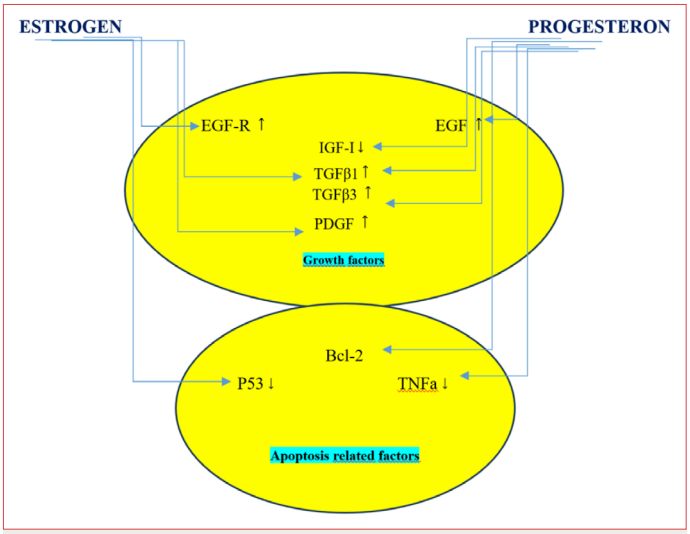


Figure 1. Schematic representation of the factors involved in the etiopathogenesis of myoma uteri.[17]

Myoma uteri is the most common benign tumor of the female pelvis and uterus and ranks first among all soft-tissue tumors. It has been detected in 50% of women in postmortem examinations.[9] It is seen in 20–30% of women of reproductive age. It is the most common indication for hysterectomy in the United States.[19] Myoma uteri is most commonly observed in women aged 50 years. Although it is very rare under the age of 30, cases occurring in adolescence have also been reported.[20] The actual incidence of myoma uterine is very difficult to determine. In pathologic examination of hysterectomy materials, the incidence of myoma uteri is as high as 77%.[21] In a Scandinavian study, asymptomatic women aged 25–40 years were evaluated by ultrasonography, and it was found that the prevalence of myoma uteri in these women was 5.4%, and the prevalence increased with age.[22] Clinical symptoms occur in 20–25% of women of reproductive age.[23] These tumors can be quite large in size and cause no symptoms or very small in size and cause symptoms. It is observed 3 times more frequently in the black race than in the white race. Myoma is frequently found in the family history of patients with myoma uteri.[24] It is 2.2 times more likely to be observed in 1st-° female relatives. Myoma in the pre-clinical stage can be detected in 24.7% of first-degree relatives. The incidence is approximately 12.8/1000.[25,26] Epidemiologic studies have shown a positive association between myoma uteri and a history of infertility and obesity and a negative association with parity, older age at delivery, and smoking.[27]

Myoma uteri are grouped according to their location in the uterus. In the uterus, fibroids are most commonly located in the corpus (91.2%), followed by the isthmus (7.2%) and cervix (2.6%).[28] Those located in the uterine corpus may be intramural (the most common location), subserous (under the visceral layer of the peritoneum), and submucous.

PATHOLOGY

Myoma uteri is usually a well-circumscribed, firm, round, gray-white tumor. It may appear raised and interlocked on the cross-sectional surface. It is a tumor made up of spindle cells. The cells fuse to form long intertwined ribbons. The structure of the cells is similar to normal myometrium tissue. Atypia and giant cells can be seen without increased mitosis. The cells around the tumor are concentrically flattened, appearing encapsulated (pseudocapsule), although not encapsulated due to the surrounding fibrous tissue. Blood supply comes from the periphery of the tumor, and the center of the tumor is relatively avascular, so necrosis and degeneration can be seen in the center of the tumor. Softening and yellow-brown discoloration can be seen in some areas, which are red regions of degeneration.[29]

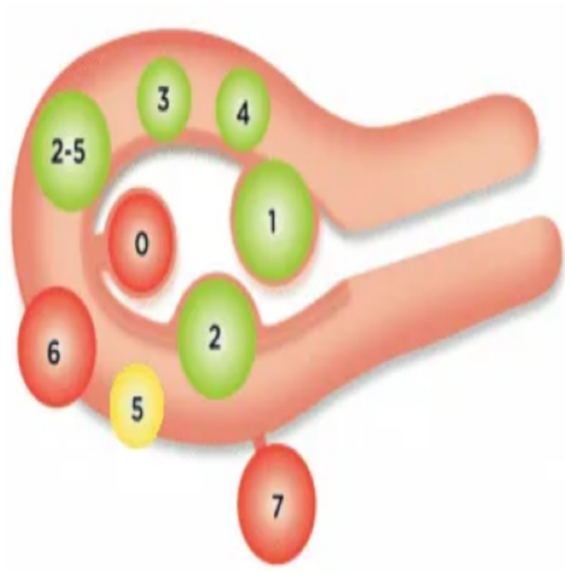
PATHOGENESIS

Fibroids, also known as leiomyomas, are thought to originate from a single mutated leiomyoma stem cell. This transformation is believed to occur following a genetic alteration, specifically, a point mutation affecting either the mediator complex subunit 12 gene or the high mobility group AT-hook 2 gene, the latter located on chromosome 12's long arm. Leiomyomas consist of three distinct cell types: fully differentiated cells, cells with intermediate differentiation, and fibroid stem cells. The rate of tumor growth is influenced by the proportion of these cell populations, with a higher concentration of stem cells associated with more rapid expansion. In addition, exposure to endocrine-disrupting chemicals potentially influenced by environmental conditions, race, or ethnicity may play a role in triggering these genetic mutations within myometrial stem cells.[30]

CLASSIFICATION OF MYOMA

Fibroids are heterogeneous in size and location. The International Federation of Gynecology and Obstetrics (FIGO) has developed a staging system that shows the location of fibroids according to mucosal and serosal surfaces. The International FIGO has developed a classification system for the causes of abnormal uterine bleeding in women of reproductive age based on imaging data. The system uses an 8-point numerical system to describe the location of fibroids relative to the endometrium (submucosal surface) and serosal surface, as given in Figure 2, with low numbers indicating a central location.[1,31]

- Type 0: Stalked fibroid localized in the submucosa and extending into the uterine cavity
- Type 1: More than 50% in the endometrial cavity, less intramural
- Type 2: <50% in the endometrial cavity, more intramural
- Type 3: Intramural fibroid adjacent to the endometrium. It does not show intracavitary extension
- Type 4: Myoma in the center of the myometrium, not associated with the endometrium or serosa
- Type 5: <50% subserous fibroids, more in the myometrium
- Type 6: More than 50% subserous fibroids and less intramural fibroids
- Type 7: Subserous fibroid with a stalk
- Type 8: Cervical fibroids and parasitic fibroids are included in this group.[1]



Category	Type	Description
Submucosal	Type 0	Completely intracavitary, pedunculated
	Type 1	Intramural portion less than 50%
	Type 2	Intramural portion more than 50%
Others	Type 3	In contact with endometrium, 100% intramural
	Type 4	Intramural
	Type 5	Subserosal, more than 50% intramural
	Type 6	Subserosal, less than 50% intramural
	Type 7	Pedunculated subserosal
	Type 8	Others (cervical, parasitic, etc.)

Figure 2. Federation of Gynecology and Obstetrics classification of myoma uteri.[1,31,32]

THE DIAGNOSIS

Diagnosing uterine fibroids presents several challenges due to the wide variability in their size, number, and anatomical location across patients. Moreover, the clinical presentation of fibroids is highly diverse, with symptoms that often overlap with other gynecological conditions such as ovulatory dysfunction, endometriosis, or endometrial polyps. Since many of the associated symptoms are common and non-specific, women may not immediately associate them with fibroids, leading to delayed diagnosis. In addition, asymptomatic fibroids can go unnoticed, allowing them to enlarge significantly over time, as illustrated in Figure 3.

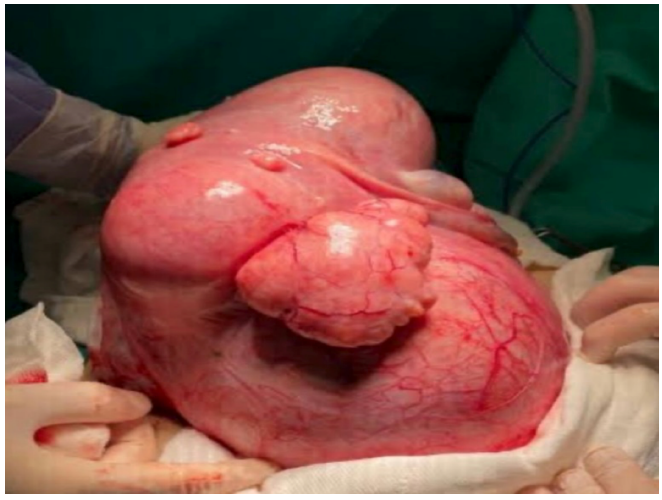


Figure 3. Intraoperative appearance of multiple fibroids reaching large sizes.

Fibroid-related symptoms may involve gynecological, urinary, or gastrointestinal systems. The most frequently reported issue is heavy menstrual bleeding, though patients may also experience prolonged menstruation, pelvic discomfort or pressure, intermenstrual bleeding, and, in some cases, anemia due to excessive blood loss. Urinary complaints often include increased frequency and, less commonly, incontinence. In rare instances, fibroids may compress the ureter, potentially leading to hydronephrosis requiring intervention. Gastrointestinal manifestations, such as constipation or tenesmus (a persistent urge to defecate), can also occur. Furthermore, some individuals report back or leg pain linked to fibroid growth.

On physical examination, findings such as a firm, irregularly enlarged uterus or palpable masses originating from the uterus are suggestive of fibroids. Nevertheless, ultrasonography remains the primary diagnostic tool in clinical settings, allowing for the definitive identification of fibroids and differentiation from other pathologies, including ovarian malignancies.[1]

The detection rate of uterine fibroids varies depending on the diagnostic method used. While bimanual pelvic examination identifies fibroids in approximately 17% of women, this rate increases to 25.8% when transvaginal ultrasonography is employed. Several imaging techniques are available to enhance diagnostic accuracy. For instance, hysterosalpingography (an X-ray procedure assessing the uterus and fallopian tubes) can

assist in detecting fibroids; however, its diagnostic performance is limited by low sensitivity and specificity (50% and 20%, respectively) due to the absence of continuous real-time 3D imaging.

Magnetic resonance ımaging (MRI) offers a superior alternative, boasting near-perfect sensitivity and specificity rates approaching 100%. Despite its higher cost and more labor-intensive nature compared to ultrasonography, MRI is particularly valuable in select cases, such as patients with obesity, a history of pelvic surgery, or those unable to undergo transvaginal ultrasound or tolerate contrast agents. Moreover, MRI provides detailed anatomical and vascular information, which is crucial for pre-operative planning in complex surgical interventions or minimally invasive treatments such as uterine artery embolization and magnetic resonance-guided focused ultrasound (MRgFUS) therapy. Notably, the diagnostic yield of these imaging modalities improves with advancing age, paralleling the increased prevalence of uterine fibroids in older women.[1]

DIFFERENTIAL DIAGNOSIS

Although fibroids are easy to diagnose, thanks to the variety of pelvic examination and imaging modalities, they can be confused with some pathologies. The main differential diagnoses of fibroids are gynecologic: ovarian cysts, paraovarian-paratubal cysts, ectopic pregnancy, hematometra, hydrosalpinx, endometrial polyp, adenomyosis, adenomyoma, and malignancies (uterine sarcoma, endometrial cancer, and metastatic tumors).[1]

CLINICAL OUTCOME

At present, there is no standardized screening program for uterine fibroids, even among women identified as having a higher risk for developing these tumors. However, in cases where women present solely with infertility – without other typical fibroid-associated symptoms imaging techniques serve as effective tools to detect fibroids that could interfere with conception or pregnancy maintenance.[1]

MEDICAL TREATMENT

Various pharmacological options exist to manage symptoms related to uterine fibroids, including non-steroidal anti-inflammatory drugs (NSAIDs), antifibrinolytic agents, and hormonal therapies such as contraceptive steroids or the levonorgestrel-releasing intrauterine device (IUD). Despite their widespread use, systematic reviews highlight a lack of robust, high-quality evidence supporting the efficacy of many of these treatments. Nonetheless, this does not imply ineffectiveness or harm.

NSAIDs have demonstrated modest benefits in alleviating dysmenorrhea and reducing menorrhagia associated with fibroids, though they are generally less effective than hormonal therapies. Their affordability and over-the-counter availability make them a practical choice in many settings. Since excessive menstrual bleeding linked to fibroids is partly driven by local fibrinolytic activity, antifibrinolytics such as tranexamic acid are frequently recommended as first-line therapy. Tranexamic acid has been shown to significantly reduce menstrual blood loss, is well tolerated, and carries a favorable safety profile. However, neither NSAIDs nor antifibrinolytics impact fibroid volume.[1]

Hormonal contraceptives containing synthetic estrogen and progestins remain the cornerstone of medical management for fibroid-related heavy menstrual bleeding, reflecting the hormone-responsive nature of fibroids in women of reproductive age. Limited clinical trial data suggest that the levonorgestrel-releasing IUD effectively reduces bleeding in women whose fibroids do not distort the endometrial cavity. While levonorgestrel promotes endometrial thinning and offers long-acting reversible contraception, it does not significantly shrink fibroids. In addition, fibroid patients face a higher risk (12–16% over 3 years) of IUD expulsion, though predictive factors remain unclear. Concerns regarding potential long-term cardiovascular risks associated with systemic levonorgestrel require further investigation. Other progestin-only contraceptives, such as depot medroxyprogesterone acetate and implants, have shown promise in reducing fibroid risk, but their role in symptom management post-fibroid development remains underexplored. Notably, oral progestins at non-contraceptive doses have not proven effective for fibroid-related menorrhagia.[1]

SURGICAL TREATMENT

Surgical intervention is generally considered a secondary approach, reserved for women with FIGO type 3 or higher fibroids and persistent heavy menstrual bleeding unresponsive to medical therapy. Endometrial ablation, a minimally invasive procedure targeting the destruction of endometrial tissue, is suitable for women who have completed childbearing. It may be performed alone or in conjunction with hysteroscopic myomectomy, particularly in cases involving submucosal fibroids. However, since ablation is irreversible and does not offer contraceptive protection, it is often viewed as less favorable compared to the levonorgestrel IUD. Figures 4 and 5 illustrate a case of submucosal fibroid managed through hysteroscopic myomectomy followed by endometrial ablation.

For women seeking definitive treatment without future fertility desires, hysterectomy with ovarian conservation remains a highly effective option for managing fibroid-induced menorrhagia. Nonetheless, this approach carries greater procedural risks and morbidity compared to less invasive alternatives such as endometrial ablation.[1]

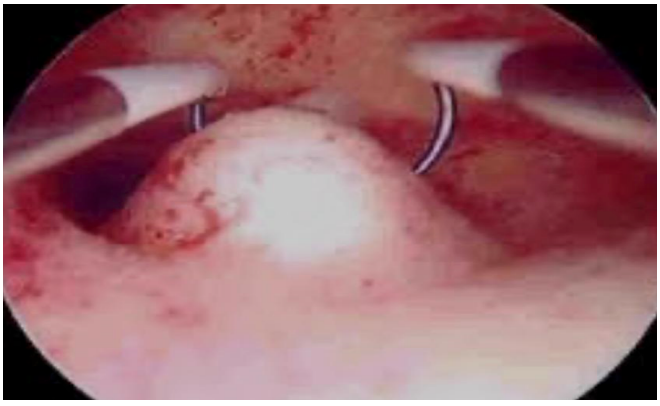


Figure 4. Hysteroscopic appearance of submucous myoma.

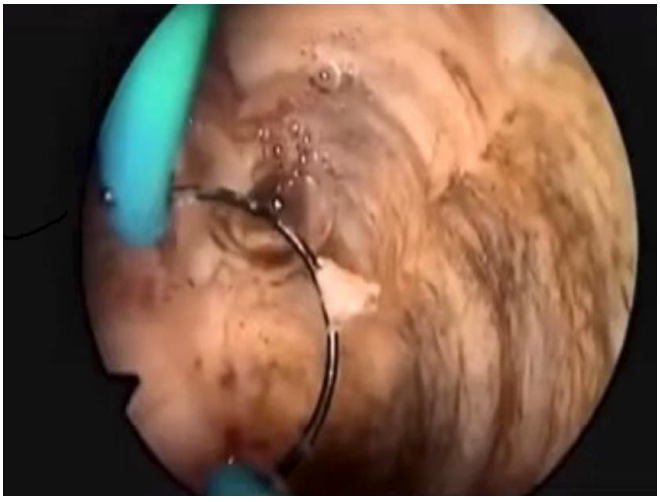


Figure 5. Hysteroscopic view of endometrial ablation.

For women experiencing fibroid-related heavy menstrual bleeding who are also seeking to enhance their fertility prospects, intramural fibroids may be surgically removed through laparoscopic, robotic, or traditional abdominal myomectomy approaches. Figure 6 illustrates a laparoscopic myomectomy procedure. Minimally invasive techniques, such as laparoscopy or robotic-assisted surgery, are generally favored due to reduced recovery times and lower complication rates, whereas open abdominal myomectomy is typically reserved for cases involving larger fibroids exceeding 10 cm in diameter. Before proceeding with surgical intervention, it is essential to conduct a comprehensive fertility assessment. This evaluation should include an analysis of ovulatory function, ovarian reserve, tubal patency, and semen parameters of the male partner to accurately identify underlying causes of infertility. Priority should be given to addressing other contributing factors

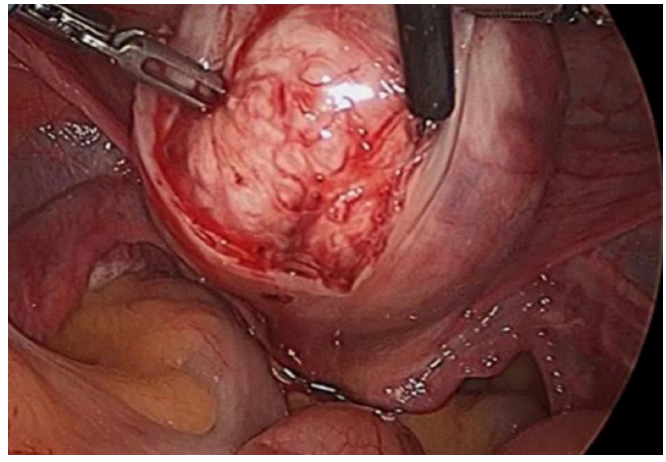


Figure 6. Intraoperative view of laparoscopic myomectomy.

to infertility prior to fibroid surgery, as operative management carries risks such as adhesion formation or inadvertent injury to pelvic structures that may further compromise reproductive potential.[1]

CONCLUSION AND RECOMMENDATIONS

The FIGO subclassification system remains the recommended framework for categorizing leiomyomas, providing a standardized approach essential for both clinical management and research. There is a critical need to expand and refine investigations into endometrial receptivity, particularly focusing on expression patterns within the tumor environment and adjacent endometrial tissue in cases involving type 1 through type 4 leiomyomas. Well-structured pre- and post-myomectomy studies are necessary to accurately classify fibroids based on type and other defining features, enabling meaningful comparisons of endometrial changes relative to baseline conditions. Furthermore, with the emergence of pharmacological agents designed for the long-term management of leiomyomas, it is imperative to assess their potential role in secondary prevention following surgical intervention, especially in younger patient populations.

The landscape of uterine fibroid treatment is poised for a significant evolution in the coming decade, driven by the urgent need to modernize outdated therapeutic paradigms. Similar to the shift from radical mastectomy to breast-conserving therapies in oncology, ongoing research is expected to facilitate a transition away from hysterectomy as the default treatment for fibroids. The future direction emphasizes the identification of prognostic markers and the development of personalized treatment strategies, promoting early intervention alongside both primary and secondary prevention measures.

Minimally invasive surgical techniques and medical therapies are anticipated to become the cornerstone of fibroid management. Innovations such as focused ultrasound therapy and the advancement of hysteroscopic or laparoscopic radiofrequency ablation offer targeted treatment of individual fibroids with reduced morbidity. In addition, the integration of image-guided interventions and molecularly targeted therapies promises to minimize the risks associated with conventional surgical approaches. Given that the transformation of myometrial stem cells into fibroid precursors appears to be a widespread biological process, future therapeutic strategies will likely focus on interrupting the progression phase of fibroid growth. As our understanding of the molecular and genetic mechanisms underlying fibroid pathogenesis deepens, novel therapies aimed at inhibiting tumor growth or inducing regression are expected to emerge.

Enhanced insights into fibroid biology may also improve the diagnostic differentiation between benign, pre-malignant, and malignant uterine conditions.

Finally, uterine fibroids present a valuable model for advancing treatment modalities applicable to oncology. Their prevalence, accessibility due to size, and the relatively low risk associated with incomplete treatment make fibroids an ideal platform for evaluating emerging therapies. This rationale underpinned their selection as the first indication for MRgFUS therapy. Consequently, fibroids can serve as an effective model system for assessing the true morbidity and efficacy of investigational treatments across a range of clinical applications.

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