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UTERINE FIBROIDS AND MODERN CONCEPTIONS: A REVIEW

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Abstract: Uterine fibroids (leiomyomas) are the most common benign smooth-muscle tumours in women of reproductive age and represent a significant source of morbidity—ranging from heavy menstrual bleeding and pelvic pain to infertility and adverse pregnancy outcomes. Contemporary conceptions of fibroids emphasise their heterogeneity, molecular underpinnings, hormonal and environmental drivers, and evolving therapeutic options that aim to preserve fertility and minimise invasiveness. This review synthesises current literature on fibroid aetiology, pathophysiology, clinical manifestations, diagnostics, and modern and emerging management strategies with a view toward “modern conceptions” of the disease. The review draws on ten or more recent scientific studies and reviews, identifies gaps in our knowledge, and highlights future directions, including integration with artificial-intelligence methods for diagnosis and treatment planning.

Introduction

Benign tumours of the uterus, commonly called uterine fibroids or leiomyomas, affect a large proportion of women of reproductive age. Estimates suggest lifetime prevalence among pre-menopausal women ranges from 40 % to 89 %, depending on detection method and population. While many women remain asymptomatic, fibroids are a leading cause of hysterectomy and contribute substantial economic and quality-of-life burden. Historically, fibroids were treated by radical surgery with little emphasis on underlying biology; modern conceptions now emphasise

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molecular mechanisms, patient-centred care, fertility preservation, and minimally invasive approaches. This review will: (1) summarise the aetiology and pathophysiology of fibroids; (2) discuss clinical presentation and diagnostics; (3) review modern treatment paradigms; (4) highlight emerging and future directions; and (5) integrate how AI and data-driven technologies may influence fibroid care.

Etiology and Pathophysiology

Genetic and molecular mechanisms :Recent molecular and genetic investigations reveal that each fibroid arises from a single mutated myometrial smooth-muscle cell (i.e., they are monoclonal). Frequent driver mutations include those in the MED12 gene, HMGA2 rearrangements, and fumarate hydratase (FH) mutations in rare cases. Once initiated, the mutated cell drives abnormal proliferation, expansion of the extracellular matrix (ECM), and growth factor signalling. The ECM itself is markedly increased in fibroids compared with normal myometrium, providing a scaffold for growth and fibrotic stiffening.

Hormonal and growth-factor influences: Fibroid growth is highly responsive to ovarian steroids (estrogen and progesterone). These hormones up-regulate growth factors (for example, transforming growth factor-β, insulin-like growth factor) and promote cellular proliferation and ECM deposition. Moreover, local tissue aromatase expression within fibroid tissue may create an intra-tumoural estrogenic microenvironment. Environmental and lifestyle factors also modulate risk: obesity, vitamin D deficiency, diet, and exposure to endocrine-disrupting chemicals have been implicated.

Functional and structural uterine alterations

Beyond mere growth of a mass, fibroids exert effects on the uterine environment. For example, they alter endometrial gene expression, impair decidualization, and reduce levels of implantation-relevant cytokines (such as leukocyte inhibitory factor). A recent meta-analysis showed that women with fibroids have alterations in uterine vascularisation, increased stiffness measured by elastography, and altered contractility compared to controls. These changes help explain symptoms (bleeding, infertility) beyond simple mechanical distortion.

Risk factors

Well-documented risk factors include African ancestry (and more severe disease in Black women), age (especially perimenopausal), hypertension, and early menarche. Emerging associations include thromboembolic risk and fibroids: a recent 2025 review found associations between fibroids and acute/chronic venous thromboembolic disease.

Clinical Manifestations

Symptoms depend on fibroid size, number, and location (submucosal, intramural, subserosal). Common clinical features include: Heavy or prolonged menstrual bleeding (abnormal uterine bleeding – AUB) leading to anemia. Pelvic pressure, urinary frequency/urgency, constipation due to mass effect. Pelvic pain or dysmenorrhea: Reproductive symptoms: infertility, miscarriage, preterm labor, placental abruption, and other obstetric complications, especially when submucosal or intramural fibroids distort the uterine cavity. Many fibroids are asymptomatic and

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found incidentally; the decision to treat frequently depends on symptom burden, fertility desires, and risks.

Diagnostic Strategies

Transvaginal ultrasound remains the first-line diagnostic modality due to accessibility and cost-effectiveness; three-dimensional ultrasound can improve mapping of fibroid number, size, and location. Magnetic resonance imaging (MRI) is increasingly used for complex cases (e.g., fertility preservation, surgical planning) due to its superior soft-tissue contrast and ability to map fibroid anatomy.

Functional imaging and emerging techniques

Modern advanced imaging studies evaluate fibroid stiffness (via elastography) and vascular perfusion metrics, which correlate with symptom severity and may predict treatment response. In the future, AI-based segmentation of fibroids on MRI is being explored to assist quantitative assessment and treatment planning.

Laboratory and ancillary assessment

While there is no biomarker specifically for fibroids, evaluation generally includes hemoglobin/hematocrit (for bleeding), iron studies, and assessment of fertility status if indicated. Pre-treatment planning may assess renal/bladder compression, and preoperative imaging for surgical risk stratification.

Modern Therapeutic Approaches

Medical (pharmacological) management

The paradigm has shifted away from hysterectomy as the default. Options now include: Gonadotropin-releasing hormone (GnRH) agonists/antagonists to induce temporary hypo-estrogenism (reduce volume, relieve bleeding) — limited by menopause-like side-effects and rebound growth. Selective progesterone receptor modulators (SPRMs) such as ulipristal acetate show promising reductions in bleeding and volume but have regulatory constraints in some jurisdictions. Emerging options include oral GnRH antagonists, aromatase inhibitors, and tranexamic acid for bleeding control. Medical therapy is increasingly considered as adjunctive (e.g., pre-surgery volume reduction) or bridge to minimally invasive surgery rather than sole long-term therapy.

Minimally invasive and interventional therapies

Uterine artery embolisation (UAE) reduces fibroid size by ischemia, effective in symptom relief, but fertility outcomes may be variable. MRI-guided focused ultrasound surgery (MRgFUS) offers non-invasive ablation under imaging guidance — suitable for selected cases.Radiofrequency ablation and laparoscopic ultrasound-guided techniques are also available.These approaches reflect the shift to uterus-preserving, less invasive therapy.

Surgical management

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Myomectomy (hysteroscopic, laparoscopic, open) remains the gold standard for women who desire fertility, especially for submucosal or intramural fibroids that distort the cavity. Hysterectomy remains definitive for women who have completed childbearing and whose symptoms are refractory—but modern minimally invasive laparoscopic/robotic hysterectomy have reduced morbidity.Surgical planning increasingly uses preoperative imaging to tailor approach, minimise blood loss, and preserve fertility.

Integrative decision-making

Patient-centred care emphasises counselling on symptom burden, fertility desires, risk/benefit of treatments, recurrence risk, and timing (e.g., before assisted reproduction). Shared decision-making is key.

Emerging and Future Directions

Molecular-targeted therapies & anti-fibrotic approaches

As our understanding of fibroid biology deepens, therapies targeting growth-factor signalling, ECM deposition, and fibrosis are in early development.

Data science and artificial intelligence (AI) integration

AI methods — including automated segmentation of fibroids on imaging, prediction of treatment outcomes (e.g., high-intensity focused ultrasound success) and personalized risk stratification — are emerging. Integration of AI into fibroid care may improve efficiency, optimise therapeutic selection, and enhance fertility outcomes.

Fertility and reproductive outcomes

More research is required on long-term fertility, obstetric outcomes, and timing of intervention. For example, submucosal fibroids clearly impair implantation; emerging evidence suggests even intramural fibroids may affect uterine functional parameters (vascularity, contractility) and thus fertility.

Prevention and lifestyle/modifiable risk factors

Emerging evidence explores modifiable risk factors (vitamin D deficiency, diet, obesity, hypertension) as potential targets for prevention.

Health-equity and global burden

Fibroids disproportionately affect women of African descent, and socio-economic and racial disparities in diagnosis/treatment exist. Research into global burden, access to minimally invasive care, and cost-effectiveness is warranted.

Conclusion

Uterine fibroids represent a heterogeneous, common condition with significant impact on women's health, fertility, and quality of life. Modern conceptions emphasise molecular heterogeneity, functional uterine alterations beyond mere bulk, individualized diagnostics, and a

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shift toward minimally invasive, fertility-preserving therapies. Emerging technologies, including AI-based imaging and outcome prediction, hold promise for refining care. Nonetheless, key gaps remain—particularly in prevention, long-term fertility outcomes, and equitable access to care. A multidisciplinary, patient-centred approach integrating the latest diagnostic, therapeutic, and digital-health tools is essential to advance fibroid management in the twenty-first century.

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