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Original Paper

Histological features of uterine fibroids in relation to women's age in Ramadi city

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ABSTRACT

Uterine fibroids are the most common benign tumors in women of childbearing age. Fibroids are made of muscle cells and other tissues that grow in and around the wall of the uterus (womb). This study aimed to examine the relationship between uterine fibroids and women's age. This study included 30 patients of different ages (25–50) who were suspected to be diagnosed with uterine fibroids. Patients were recruited from Al-Rrazy private hospital and private laboratories. Patient information, including name and age, was recorded. Pathological fibroid samples were collected postoperatively and fixed in 10% formalin for histopathological analysis. The present work revealed a significant difference in uterine fibroids with increasing age; 80% of phase 1 showed a substantial increase in women aged 40–50. Histopathological findings in phase 1 showed cellular leiomyoma with mitotic figures, sheets of smooth muscle cells arranged in parallel, and no stromal collagen. In conclusion, the incidence and prevalence rates of uterine fibroids in the normal population of Al-Ramadi city are comparable to those reported in other parts of the globe. Rates of uterine fibrosis ranged between age groups and reached 35% of women. The most affected cases were between 40 and 50 years of age in phase 1.

Keywords: *Uterus, Uterine fibroid, Uterine phase, Women's age*

1 INTRODUCTION

Leiomyoma, or uterine fibroids, are benign tumors that occur in more than 75% of women [1]. They are classified histologically by the presence of smooth muscle cells, fibroblasts, and abundant stroma. Most fibroids are histologically benign, although malignant ones are reported in about 2% of cases [2]. In 10%-20% of cases, intracytoplasmic lipid accumulation occurs in the tissue; spindle cells are usually organized in fascicles, and an atypical arrangement can be identified even with the use of progestogenic substances [3]. These changes, attributed to progestogens, may mimic malignancy, including infarct-type necrosis, increased cellularity, and mitotic activity [4, 5].

Uterine fibroids remain a major health concern for women of reproductive age and present with diverse clinical manifestations and histological characteristics. The most common symptoms include menstrual irregularities, followed by abdominal pain and infertility. Histologically, the majority of fibroids are intramural; within the endometrium, the most frequent pattern is proliferative. Secondary changes, including hyaline, cystic, and myxoid degeneration, are also common [6]. Fibroids greatly affect women's reproductive health and well-being [7]. Fibroids and fertility are closely related, as their location affects implantation and the ability to carry a pregnancy [8]. Fibroids may lead to heavy menstrual bleeding and pelvic pain and may hinder conception.

Interactions with the endometrium include alterations in extracellular matrix synthesis, mechanotransduction, and abnormal microRNA secretion, which negatively affect uterine receptivity and implantation [9].

The cause remains poorly understood; however, several factors may predispose women to this condition, including age, race, family history of fibroids, obesity, lifestyle, and, most importantly, estrogen [10]. Estrogen is known to have a proliferative effect on fibroid tissue, and its level increases during pregnancy and estrogen therapy [11]. Symptoms may include dysmenorrhea (abnormally painful menstrual periods), abnormal vaginal bleeding, and pelvic pressure, although many cases are asymptomatic and may go unnoticed. Physical examination, along with imaging studies, is the most common method of diagnosis [10]. Medical, minimally invasive, and surgical treatments are available, depending on symptoms, fibroid size and nature, and whether the patient wishes to conceive in the future [11].

The documented rise in fibroids could be explained by the growth of previously existing fibroids, increased symptom severity, and a higher tendency for women in later reproductive years to undergo gynecologic procedures. If fibroids develop and grow more in later childbearing years, hormonal changes associated with the perimenopausal transition may play a significant role. Alternatively, the increase in later reproductive years might reflect cumulative exposure to estrogen and progesterone over 20–30 years of reproductive life [12]. Uterine fibroids present management challenges for individuals seeking pregnancy, those with limited access to health care, or those with non-modifiable risk factors. Hormonal and anti-inflammatory treatment approaches may play a role in slowing fibroid progression; however, emphasis is placed on optimizing outcomes with conservative surgical and treatment methods that are more likely to preserve fertility [13]. Understanding the mechanisms by which fibroids influence fertility is crucial for developing targeted treatments compatible with patients' desires for pregnancy [8]. Fibroid pathology is also vital for appropriate diagnosis and treatment, as fibroids are a major cause of hysterectomy [1].

There is a close correlation between the occurrence of uterine fibroids and women's age, through which this health condition has different effects. Young women with fibroids are more likely to have hypertension, obesity, and diabetes compared with older women with fibroids or controls and therefore have a worse cardiovascular risk profile in relatively young individuals [14]. Furthermore, research indicates that women aged 26–45 years are more likely to have uterine fibroids than any other group. Factors that may increase susceptibility in this age bracket include marrying late and being educated [15]. In addition, the size and clinical symptoms of uterine fibroids have been reported to be associated with serum vitamin D level. Moreover, vitamin D deficiency is strongly associated with an increased risk of fibroids among reproductive-age women [16]. Recognizing the effects of age is essential when designing strategies to reduce the adverse impact of uterine fibroids in women across age groups. Fibroids are associated with cardiovascular disease and cardiometabolic risk factors, with hypertension being predominant among women [17]. Body mass index is the most significant predictor of fibroid formation, while fruit and vegetable consumption, as well as vitamin D intake, are protective factors [11]. Fibroids are also related to preterm birth, and the risk of preterm birth has been found to rise with decreasing gestational age [4].

2 MATERIALS AND METHODS

In this study, 30 female patients of various ages (25–50) who were clinically suspected of having uterine fibroids participated. Patients were selected from Al-Rrazy Private Hospital in Ramadi city and some of its affiliated privately owned laboratories. To ensure a comprehensive medical record and facilitate correlation of histopathological results, personal information, including name and age, was recorded for each patient. Pathological specimens were collected after surgery directly from the removed uterine fibroids. To preserve tissue architecture and cellular integrity for subsequent microscopic analysis, these samples were promptly stored in 10% neutral buffered formalin. For the control group, normal myometrial tissue was obtained from patients who underwent hysterectomy for reasons unrelated to fibroids. The tissue sample was obtained from a non-pathological site of the uterus, fixed in 10% neutral buffered formalin, and then paraffin-embedded. All control samples were negative for any uterine pathology, as confirmed by a certified pathologist. Fixation was necessary to prevent tissue degeneration and ensure accuracy and precision in histological evaluation.

Later, standard histology procedures were used to process the preserved specimens. To highlight cellular and structural features under a microscope, these procedures included tissue dehydration, clearing, paraffin embedding,

sectioning into thin slices, and staining, typically with hematoxylin and eosin (H&E). This methodical approach enabled thorough examination of the histological features of uterine fibroids, helping to clarify the nature of the lesions in each case and validate the clinical diagnosis.

2.1 Statistical analysis

The Statistical Analysis System (SAS) was used for data analysis and to determine the significance (p-value = 0.05) of the incidence of uterine fibroids in Al-Ramadi city, using a completely randomized design (card). Then, significant microscopic changes between the studied categories were compared.

3 RESULTS

3.1 Uterine fibroid related to age

The present work revealed a significant association between uterine fibroids and increasing age (Table 1, Figure 1). The control case showed normal endometrial architecture adjacent to normal myometrium (Figure 2). Histopathological results showed phase 1 to be a cellular leiomyoma with mitotic figures, sheets of smooth muscle cells arranged in parallel, and no stromal collagen (Figure 3). It should be noted here that collagen can be observed in uterine fibroid tissues using hematoxylin and eosin (H&E) staining, where it appears pink to red. Phase 2 showed leiomyoma with randomly arranged sheets of smooth muscle cells with minimal stromal collagen (Figure 4). Phase 3 showed leiomyoma with sheets of smooth muscle cells and interlacing, haphazardly arranged eosinophilic collagen bundles (Figure 5). Phase 4 showed leiomyoma with compressed sheets of smooth muscle cells, abundant thick stromal collagen, and hyalinization (Figure 6).

Table 1 Significant differences in the uterine fibroids with increasing age

Age range	Phases	Causes
	Percentage and phase type	Results variations
25-30 (10 cases)	70% with phase1 20% with phase2 10% with phase3	This variability of the results depending on the age increase, and other risk factors that may involve in increase and decrease the region of uterine fibroid, where Fibroid area increase age until is reaching age of menopause then fibroids region start to shrinkage as well as decrease intensity of symptoms also there are obesity,red -meat diet which can increase the fibroid area
30-40 (10 cases)	30% with phase1 50% with phase2 10% with phase3 10% with phase4	
40-50 (10 cases)	80% with phase1 20% with phase 2	

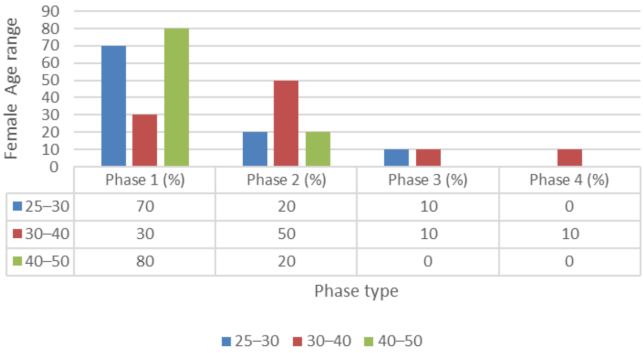

Fig. 1 Percentage of uterine fibroids with increasing age

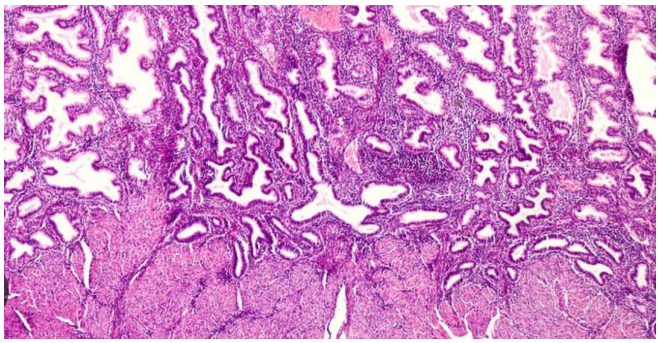

Fig. 2 Control case shows Endometrium Adjacent normal myometrium (The section taken from the part of endometrium and myometrium that is not involved by leiomyoma) (H&E) X40

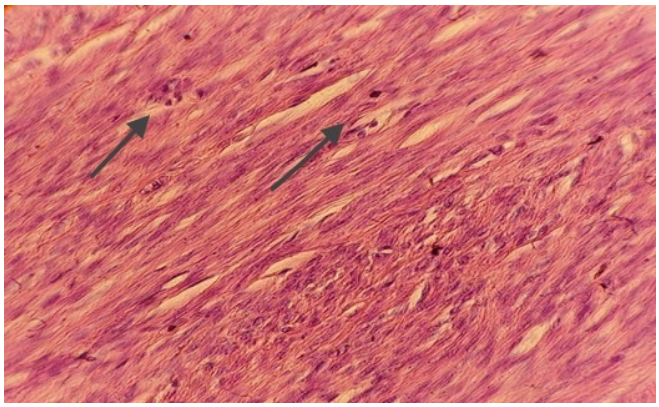

Fig. 3 Phase 1, Cellular leiomyoma with mitosis sheets of smooth muscle cells arranged in a parallel way with no stromal collagen (lack arrow) (H&E) X40

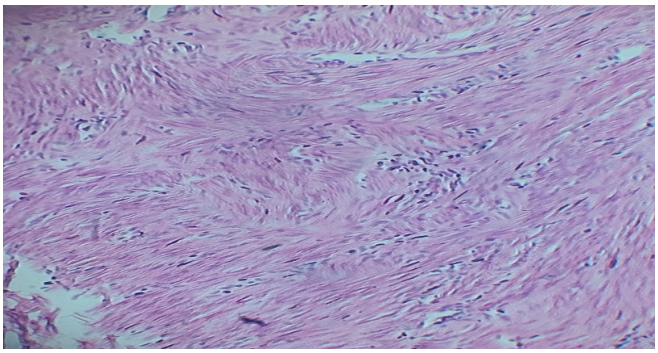

Fig. 4 Phase 2, leiomyoma with randomly arranged sheets of smooth muscle cells with minimal stromal collagen (black arrow) (H&E) X40

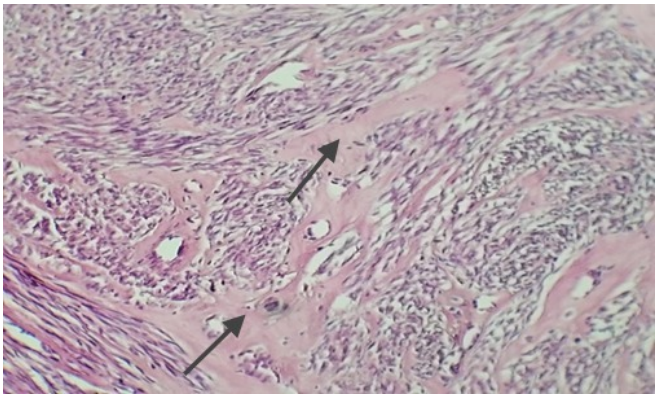

Fig. 5 Phase 3 shows leiomyoma with sheets of smooth muscle cells with interlacing haphazard eosinophilic collagen bundles. (black arrow) (H&E) X 40

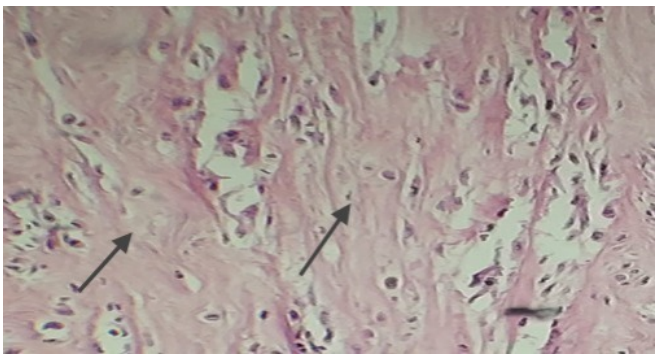

Fig. 6 Phase 4 shows a leiomyoma with compressed sheets of smooth muscle cells and abundant thick stromal collagen, with hyalinized myometrial collagen. (black arrow) (H&E) X 40

4 DISCUSSION

Several factors have been linked to the development of fibroids and the different patterns in which they become clinically evident. Some factors are modifiable and may help promote preventive health care. Numerous risk factors for fibroid development have been described, several of which may be influenced by dietary or lifestyle changes [18].

The results of this study show that most uterine fibroids in women aged 40–50 years are in the first histological phase (phase 1) (Table 1, Figure 1), and the histological changes showed cellular leiomyoma with mitosis, with sheets of smooth muscle cells arranged in a parallel pattern and no stromal collagen (Figure 3). This prevalence in the 40–50 age group aligns with broader epidemiological data indicating that uterine fibroids are highly prevalent in women approaching menopause, with studies showing that approximately 70%–90% of women may develop fibroids by age 50 [19]. This can be attributed to the gradual decline in hormonal activity as women approach menopause, during which the secretion of estrogen and progesterone, the primary hormones responsible for stimulating fibroid growth, decreases. This hormonal decline leads to a slower rate of cellular proliferation in fibroid tissues and a reduction in their growth rate, resulting in most fibroids remaining in early histological stages. This mechanism is consistent with established knowledge of fibroid biology, where the withdrawal of ovarian steroid hormones following menopause typically leads to fibroid regression and a reduction in proliferative activity [20]. Additionally, reduced blood supply to larger fibroids in this age group may contribute to slower progression; poor vascularization leads to gradual shrinkage of fibroids and to the transformation of previously active fibers into stable fibrous masses, consistent with the characteristics of phase 1 [21]. This agrees with other studies noting that most fibroids are benign and have a self-limited growth phase in the first phase, which takes 3 days; the increase of myocytes without much collagen matrix is reminiscent of the response to experimentally inflicted injury in vascular smooth muscle [22]. This could be interpreted as proliferation of uterine myocytes in response to hypoxia arising from vasoconstriction during menstruation, especially in women with myometrial hypercontractility associated with primary dysmenorrhea [23]. The proliferation of smooth muscle cells is consistently observed in this tissue mass under prolonged exposure to estrogen and progesterone

and can develop into a biclonal or monoclonal population, as some cell types have growth advantages.

Figure 4 clearly shows phase 2: leiomyoma with randomly arranged sheets of smooth muscle cells with minimal stromal collagen. In phase 2, myocytes begin to express collagen (<10%) and switch to a proliferative/synthetic phenotype, which may occur at the onset of some tumors [24]. Furthermore, this reflects peak hormonal activity during the reproductive years, when estrogen and progesterone levels are at their highest. Elevated hormonal stimulation promotes active growth and progression of fibroids from early to more advanced histological phases. The predominance of phase 2 (50%) indicates that most fibroids in this age range are in a state of active proliferation and development [25].

In phase 3, leiomyoma showed sheets of smooth muscle cells with interlacing, haphazardly arranged eosinophilic collagen bundles (Figure 5), and 10% of women were aged 25–30 and 30–40 (Table 1, Figure 1). These findings specify the proliferation–secrete–fibroblast life cycle: proliferation is evident, followed by a transition to collagen synthesis (10%–50% collagen), with continual maturation and buildup, and possibly a preponderance, of the extracellular space. Given that excess matrix places myocytes farther from nearby capillaries, angiogenesis seems to contribute to tumor growth in size [26]. The relative deficiency of fibrogenic growth factors relative to angiogenic growth factors, such as FGF and TGF-FG, compared with VEGF, may explain a later stepwise increase in microvessel density as the tumor forms and matures. This is because all cells must be situated reasonably close to capillaries to obtain adequate oxygen and nutrients, leading to interstitial ischemia. The histological consequence of such interstitial ischemia is cellular atrophy, which has been described in phase 4 tumors and is a common feature of late phase 3 tumors [27].

In the terminal phase (phase 4) of a fibroid's lifecycle (Figure 6), this is considered the involution phase, characterized by large areas of hyaline matrix with only occasional islands of atrophic myocytes, and about 10% of women were within the ages of 30–40 (Table 1, Figure 1). The presence of phase 4 fibroids in women aged 30–40 years can be explained by the high hormonal activity characteristic of this reproductive period. This aligns with other studies, which clarify that during these ages, estrogen and progesterone levels peak, providing strong stimulation for fibroid growth. This enhanced hormonal environment can lead to rapid fibroid enlargement, which, in some cases, results in degenerative and fibrotic changes. Necrosis following infarction is often associated with phase 4, and excessive epithelial and mesenchymal components are overproduced [28]. Schizophrenic apoptotic cells are also occasionally seen [28]. However, there is a third form of cell death, namely diagnosis, which is an atrophic change attributed to inadequate cellular nutrition; this is another feature unique to the final stage [29, 30].

The growth pattern and location of fibroids are significant factors that dictate symptomatology and the occurrence of complications [31]. Therefore, it is possible to have a small lesion that is symptomatic if it is situated in the uterine cavity compared with a large lesion found in the extra-metrium. Different locations are classified as follows:

1. Intramural fibroids affect the muscular layer of the uterus and are the most common type of neoplasm [32]. If they are not bulky, they may be asymptomatic. Intramural fibroids arise from smooth muscle proliferation within the muscular wall of the uterus. Intramural fibroids also grow inward; hence, they may distort and elongate the corpora of the uterus over time.

2. Subserosal fibroids are found beneath the uterus's serosa. They can also protrude into the surrounding tissue and be connected to the body's surface by a narrow stalk; these are known as pedunculated fibroids [33].

3. Submucosal fibroids are situated in the muscular layer beneath the endometrium of the uterus, and they alter the configuration of the cavity; even small tumors at this site can cause bleeding and infertility [34].

4. Cervical fibroids are situated in the body of the cervix, that is, in the neck of the uterus. Occasionally, lesions involve the supporting structures. Fibroids may be solitary or multiple. Uterine fibroids originate in the smooth muscle tissue of the uterus's body [35, 36].

Finally, uterine fibroid is a widespread problem in women of reproductive age, leading to multiple bleeding and painful symptoms that affect various aspects of women's lives [37].

5 CONCLUSION

In conclusion, the prevalence and incidence of uterine fibroids in Al-Ramadi city appear similar to those in other regions worldwide. We recommend conducting well-designed surveys with sufficient geographic representation to examine the epidemiologic characteristics of uterine fibroids in Al-Ramadi city and provide a clearer picture of age, sex, trends, and geographic distribution of these cases. In this research, we found rates of uterine fibrosis that varied across age groups, reaching 35% in women. Our results showed that most affected cases were between 40 and 50 years old, with phase 1, which was lower than the rates observed in developed countries. Although health care indices in Al-Ramadi city have improved in recent years, further studies to reevaluate these rates are needed, and policymakers' attention is essential to improve the diagnostic rate of uterine fibroids.

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Data availability

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DECLARATIONS

Conflict of interest

The authors confirm that they have no known conflicts of interest associated with this publication and no significant financial source has been received that could have influenced the study's outcomes.

Consent to publish

N/A

Ethical approval

All subjects gave their informed consent for inclusion before they participated in the study. The study was conducted in accordance with the Declaration of Helsinki

REFERENCES

[1] Baird, DD, Dunson, DB, Hill, MC, Cousins, D Schectman, JM. High cumulative incidence of uterine leiomyoma in black and white women: ultrasound evidence. American journal of obstetrics and gynecology. 2003; 188(1), 100-107

[2] Segars JH. Uterine Fibroid Research: A Work in Progress. Reproductive Sciences. 2014;21(9):1065–1066. 10.1177/1933719114546644

[3] Boyd C, McCluggage WG. Unusual morphological features of uterine leiomyomas treated with progestogens. Journal of Clinical Pathology. 2011;64(6):485–489. 10.1136/jcp.2011.089664

[4] Kaushal DA, Gupta DDPD, Kumar DA. Clinicopathological profile of uterine fibroid at a medical college hospital. Tropical Journal of Pathology and Microbiology. 2020;6(2):155–160. 10.17511/jopm.2020.i02.07

[5] Alkhrait S, Malasevskaia I, Madueke-Laveaux OS. Fibroids and Fertility. Obstetrics and Gynecology Clinics of North America. 2023;50(4):663–675. 10.1016/j.ogc.2023.08.006

[6] Don EE, Mijatovic V, Huirne JAF. Infertility in patients with uterine fibroids: a debate about the hypothetical mechanisms. Human Reproduction. 2023;38(11):2045–2054. 10.1093/humrep/dead194

[7] Navarro A, Bariani MV, Yang Q, Al-Hendy A. Understanding the Impact of Uterine Fibroids on Human Endometrium Function. Frontiers in Cell and Developmental Biology. 2021;9. 10.3389/fcell.2021.633180

[8] Bano A, Wei CR, Memon AAQ, Osama M, Shaikh S, Shah Q, et al. A COMPREHENSIVE REVIEW OF UTERINE FIBROIDS: PATHOGENESIS, DIAGNOSIS, TREATMENT, AND FUTURE PERSPECTIVES. Journal of Population Therapeutics and Clinical Pharmacology. 2023:1961–1974. 10.53555/jptcp.v30i18.3385

[9] Datir SG, Bhake A. Management of Uterine Fibroids and Its Complications During Pregnancy: A Review of Literature. Cureus. 2022. 10.7759/cureus.31080

[10] Wong JYY, Gold EB, Johnson WO, Lee JS. Circulating Sex Hormones and Risk of Uterine Fibroids: Study of Women's Health Across the Nation (SWAN). The Journal of Clinical Endocrinology& Metabolism. 2016;101(1):123–130. 10.1210/jc.2015-2935

[11] Donnez J, Dolmans MM. Uterine fibroid management: from the present to the future. Human Reproduction Update. 2016;22(6):665–686. 10.1093/humupd/dmw023

[12] Behairy MS, Goldsmith D, Schultz C, Morrison JJ, Jahangiri Y. Uterine fibroids: a narrative review of epidemiology and management, with a focus on uterine artery embolization. Gynecology and Pelvic Medicine. 2024;7:23. 10.21037/gpm-23-57

[13] Okesola MA, Ogunlana OO, Adegboye BE, Afolabi IS, Bello FA, Lasisi AJ. The Multi-risk Factors Promoting Uterine Fibroids in Women. In: 2023 International Conference on Science, Engineering and Business for Sustainable Development Goals (SEB-SDG). IEEE; 2023. p. 1–6. 10.1109/seb-sdg57117.2023.10124571

[14] Hammad AA, Elbohoty SB, Hazzaa SM, Hewedy MS, El-Gharib MN. Relation between Vitamin D and Uterine Fibroid. Journal of Advances in Medicine and Medical Research. 2022:135–146. 10.9734/jammr/2022/v34i1931447

[15] Brewster LM, Haan Y, van Montfrans GA. Cardiometabolic Risk and Cardiovascular Disease in Young Women With Uterine Fibroids. Cureus. 2022. 10.7759/cureus.30740

[16] Keizer AL, Semmler A, Kok HS, van Kesteren PJM, Huirne JAF, Hehenkamp WJK. Modifiable prognostic factors in uterine fibroid development: a systematic review of literature. Journal of Obstetrics and Gynaecology. 2023;44(1). 10.1080/01443615.2023.2288225

[17] Landman AJEMC, Don EE, Vissers G, Ket HCJ, Oudijk MA, de Groot CJM, et al. The risk of preterm birth in women with uterine fibroids: A systematic review and meta-analysis. PLOS ONE. 2022;17(6):e0269478. 10.1371/journal.pone.0269478

[18] Yang Q, Ciebiera M, Bariani MV, Ali M, Elkafas H, Boyer TG, et al. Comprehensive Review of Uterine Fibroids: Developmental Origin, Pathogenesis, and Treatment. Endocrine Reviews. 2021;43(4):678–719. 10.1210/endrev/bnab039

[19] Ulin M, Ali M, Chaudhry ZT, Al-Hendy A, Yang Q. Uterine fibroids in menopause and perimenopause. Menopause. 2019;27(2):238–242. 10.1097/gme.0000000000001438

[20] Grube M, Neis F, Brucker SY, et al. Uterine Fibroids – Current Trends and Strategies. Surgical Technology International. 2019;34:257-63

[21] Walker CL, Stewart EA. Uterine Fibroids: The Elephant in the Room. Science. 2005;308(5728):1589–1592. 10.1126/science.1112063

[22] Machado-Lopez A, Simón C, Mas A. Molecular and Cellular Insights into the Development of Uterine Fibroids. International Journal of Molecular Sciences. 2021;22(16):8483. 10.3390/ijms22168483

[23] Stewart EA, Nowak RA. Uterine Fibroids: Hiding in Plain Sight. Physiology. 2022;37(1):16–27. 10.1152/physiol.00013.2021

[24] Williams ARW. Uterine fibroids – what's new? F1000Research. 2017;6:2109. 10.12688/f1000research.12172.1

[25] Vannuccini S, Petraglia F, Carmona F, Calaf J, Chapron C. The modern management of uterine fibroids-related abnormal uterine bleeding. Fertility and Sterility. 2024;122(1):20–30. 10.1016/j.fertnstert.2024.04.041

[26] Owens GK. Regulation of differentiation of vascular smooth muscle cells. Physiological Reviews. 1995;75(3):487–517. 10.1152/physrev.1995.75.3.487

[27] Jonker SS, Louey S, Roselli CE. Cardiac myocyte proliferation and maturation near term is inhibited by early gestation maternal testosterone exposure. American Journal of Physiology-Heart and Circulatory Physiology. 2018;315(5):H1393–H1401. 10.1152/ajpheart.00314.2018

[28] Machtinger R, Fennessy FM, Stewart EA, Missmer SA, Correia KF, Tempany CM. MR-guided focused ultrasound (MRgFUS) is effective for the distinct pattern of uterine fibroids seen in African-American women: data from phase III/IV, non-randomized, multicenter clinical trials. Journal of Therapeutic Ultrasound. 2013;1(1). 10.1186/2050-5736-1-23

[29] Simon SM, Nogueira AA, Almeida ECSd, Poli Neto OB, Silva JCRe, Reis FCd. Leiomiomas uterinos e gravidez. Revista Brasileira de Ginecologia e Obstetrícia. 2005;27(2). 10.1590/s0100-72032005000200007

[30] Kempson RL, Hendrickson MR. Smooth Muscle, Endometrial Stromal, and Mixed Müllerian Tumors of the Uterus. Modern Pathology. 2000;13(3):328–342. 10.1038/modpathol.3880055

[31] Faerstein E, Szklo M, Rosenshein N. Risk Factors for Uterine Leiomyoma: A Practice-based Case-Control Study. I. African-American Heritage, Reproductive History, Body Size, and Smoking. American Journal of Epidemiology. 2001;153(1):1–10. 10.1093/aje/153.1.1

[32] Ciavattini A, Di Giuseppe J, Stortoni P, Montik N, Giannubilo SR, Litta P, et al. Uterine Fibroids: Pathogenesis and Interactions with Endometrium and Endomyometrial Junction. Obstetrics and Gynecology International. 2013;2013:1–11. 10.1155/2013/173184

[33] Clement PB. The Pathology of Uterine Smooth Muscle Tumors and Mixed Endometrial Stromal-Smooth Muscle Tumors: A Selective Review with Emphasis on Recent Advances. International Journal of Gynecological Pathology. 2000;19(1):39–55. 10.1097/00004347-200001000-00006

[34] Puri K, Famuyide AO, Erwin PJ, Stewart EA, Laughlin-Tommaso SK. Submucosal fibroids and the relation to heavy menstrual bleeding and anemia. American Journal of Obstetrics and Gynecology. 2014;210(1):38.e1-38.e7. 10.1016/j.ajog.2013.09.038

[35] Laughlin SK, Baird DD, Savitz DA, Herring AH, Hartmann KE. Prevalence of Uterine Leiomyomas in the First Trimester of Pregnancy: An Ultrasound-Screening Study. Obstetrics & Gynecology. 2009;113(3):630–635. 10.1097/aog.0b013e318197bbaf

[36] Marshall L. Variation in the Incidence of Uterine Leiomyoma Among Premenopausal Women by Age and Race. Obstetrics & Gynecology. 1997;90(6):967–973. 10.1016/s0029-7844(97)00534-6

[37] Zimmermann A, Bernuit D, Gerlinger C, Schaefers M, Geppert K. Prevalence, symptoms and management of uterine fibroids: an international internet-based survey of 21,746 women. BMC Women's Health. 2012;12(1). 10.1186/1472-6874-12-6

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