World Journal of Medical and Health
www.worldmedicaljournals.com
Online ISSN:
Received: 25-01-2026, Accepted: 27-02-2026, Published: 12-03-2026
Volume 2, Issue 1, Jan-Mar, 2026, Page No. 24-29

Evaluation of ovarian reserve following uterine fibroid embolization compared to myomectomy in premenopausal women

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Abstract
Background: Uterine fibroids are highly prevalent benign tumors in premenopausal women. While hysterectomy is definitive, uterine-sparing treatments such as uterine fibroid embolization (UFE) and myomectomy are preferred for women desiring future fertility. A primary concern with UFE is the potential for non-target embolization of the ovarian vasculature, which may compromise ovarian reserve.
Objective: This study aimed to compare the impact of UFE versus laparoscopic myomectomy on ovarian reserve, as measured by serum Anti-Mullerian Hormone (AMH) levels, over a 12-month period.
Method: A simulated retrospective cohort study was conducted using synthesized academic training data of 240 premenopausal women (aged 25-45 years) with symptomatic fibroids. Patients were stratified into a UFE group (n=120) and a laparoscopic myomectomy group (n=120). Serum AMH levels were assessed at baseline, 6 months, and 12 months post-procedure.
Results: Both groups experienced a decline in AMH levels at 6 months, but the decline was significantly more pronounced in the UFE group (mean decrease of 22.4% versus 8.1%, $p<0.001$). At 12 months, the myomectomy group showed recovery to near baseline levels, whereas the UFE group exhibited a sustained, though partially recovered, reduction (mean decrease of 15.2% from baseline, $p=0.002$).
Conclusion: In this simulated dataset, UFE was associated with a greater short-term and sustained reduction in ovarian reserve compared to laparoscopic myomectomy. These findings suggest that myomectomy may be the preferred uterine-sparing option for premenopausal women who prioritize the preservation of maximal ovarian function.

Keywords: Uterine fibroid embolization, myomectomy, ovarian reserve, anti-mullerian hormone, leiomyomas

Introduction

Uterine leiomyomas, commonly known as fibroids, represent the most prevalent benign solid pelvic tumors in women of reproductive age, affecting up to 70% of women by the age of 50. While many fibroids are asymptomatic, a significant proportion of affected women experience debilitating symptoms, including heavy menstrual bleeding, chronic pelvic pain, bulk symptoms, and reproductive dysfunction such as infertility and recurrent pregnancy loss. The management of symptomatic uterine fibroids has evolved significantly over the past few decades, shifting away from the historical default of hysterectomy toward a variety of uterine-sparing treatment modalities designed to preserve future fertility and avoid the psychological and physical impacts of organ removal.

Among the fertility-sparing options, surgical myomectomy whether performed via hysteroscopy, laparoscopy, or laparotomy—has long been considered the gold standard. Myomectomy involves the direct excision of fibroids while meticulously reconstructing the uterine wall, thereby restoring normal uterine anatomy and theoretically optimizing the endometrial environment for embryo implantation. However, myomectomy is an invasive surgical procedure associated with inherent risks, including significant blood loss, adhesion formation, and the potential need for subsequent cesarean delivery due to the risk of uterine rupture in subsequent pregnancies.

In recent years, uterine fibroid embolization (UFE) has emerged as a highly effective, minimally invasive alternative to surgical management. During UFE, an interventional radiologist selectively catheterizes the uterine arteries and injects embolic particles, which occlude the blood supply to the fibroids, leading to their ischemic necrosis and subsequent shrinkage. UFE offers numerous advantages, including shorter hospital stays, faster recovery, reduced blood loss, and a lower risk of post-procedural adhesions. Multiple randomized controlled trials have demonstrated that UFE provides symptom relief equivalent to surgery in the short to medium term.

Despite these advantages, the adoption of UFE in women desiring future fertility remains controversial. The primary concern stems from the unique dual blood supply of the ovaries. The ovaries receive blood from both the ovarian arteries and the anastomotic branches of the uterine arteries. Because UFE relies on the occlusion of the uterine arteries, there is a theoretical risk of non-target embolization of the ovarian arterial branches, leading to decreased perfusion of the ovarian cortex and subsequent ischemic damage to the primordial follicle pool.

Assessing the true impact of UFE on ovarian function requires an accurate and reliable biomarker of ovarian reserve. Anti-Mullerian Hormone (AMH) has emerged as the most optimal marker for this purpose. Produced directly by the granulosa cells of small pre-antral and antral follicles, AMH is independent of the menstrual cycle, demonstrates minimal intra-cycle variability, and correlates strongly with the remaining ovarian follicular pool (10). While some studies have reported transient or permanent declines in AMH following UFE, the data remains heterogeneous, and few studies have directly compared the AMH trajectory

following UFE to that following laparoscopic myomectomy in strictly premenopausal women with comparable baseline characteristics. Therefore, this study aimed to systematically evaluate and compare the impact of UFE and laparoscopic myomectomy on serum AMH levels over a 12-month follow-up period using a simulated, well-matched patient cohort.

Literature Review

The pathophysiological basis for the concern regarding ovarian reserve following UFE is deeply rooted in pelvic vascular anatomy. The ovarian blood supply is complex and highly variable. Typically, the ovarian artery arises from the abdominal aorta and anastomoses with the ovarian branch of the uterine artery within the mesosalpinx. This collateral circulation means that the uterine arteries contribute substantially to ovarian perfusion. When embolic particles are injected into the uterine arteries during UFE, there is a documented risk that these particles will migrate antegrade into the ovarian arterial tree. This non-target embolization can cause microvascular occlusion within the ovarian cortex, leading to focal ischemia, follicular atresia, and a measurable decline in ovarian reserve.

Conversely, laparoscopic myomectomy approaches the fibroids from the serosal surface of the uterus. While it does not involve the intentional occlusion of major arterial vessels, it is not entirely devoid of risk to ovarian function. The surgical trauma of myomectomy can trigger a localized inflammatory response, and the meticulous dissection required to separate fibroids from the myometrium can potentially disrupt the adjacent fallopian tubes and the utero-ovarian ligaments. Furthermore, the use of electrosurgical energy (such as bipolar cautery) during myomectomy to achieve hemostasis can generate thermal spread, which, if applied too close to the ovarian hilum or the mesosalpinx, could theoretically cause collateral thermal damage to the ovarian cortex and its follicular reserve.

The clinical utility of Anti-Mullerian Hormone as a metric for ovarian reserve cannot be overstated in this context. Historically, follicle-stimulating hormone (FSH) measured on day 3 of the menstrual cycle was the standard marker. However, FSH has a high degree of inter-cycle variability and only rises after significant follicular depletion has already occurred. AMH, in contrast, is produced by the actively growing cohort of small follicles and provides a direct, quantitative snapshot of the resting primordial follicle pool. A decline in AMH following a gynecological intervention is now widely accepted as the most sensitive early indicator of iatrogenic ovarian damage.

Despite the availability of this sensitive biomarker, the existing literature comparing the ovarian impact of UFE versus myomectomy is characterized by significant methodological limitations and conflicting results. Several early studies on UFE reported no significant change in AMH levels post-procedure, suggesting that the ovarian collateral circulation is sufficient to protect the ovaries from ischemia in most women. However, these studies often included perimenopausal women, who already have diminished baseline ovarian reserve, potentially masking a subtle treatment effect.

More recent prospective cohort studies focusing strictly on younger, premenopausal women have yielded concerning findings. A study by Hehenkamp *et al*. demonstrated a significant 21% reduction in AMH at 3 months following UFE, with only partial recovery at 12 months. In comparison, data regarding myomectomy generally show a mild, transient drop in AMH immediately post-surgery, attributed to the acute inflammatory response and anesthesia, followed by a complete return to baseline levels by 6 to 12 months.

A critical research gap persists because very few studies have performed a direct, head-to-head comparison of these two modalities within the same methodological framework. Many existing studies suffer from small sample sizes, heterogeneous fibroid sizes and locations, and inconsistent AMH sampling timelines. Without direct comparative data, it is exceedingly difficult for clinicians to provide accurate, evidence-based counseling to premenopausal women facing the choice between a minimally invasive embolization procedure and a traditional surgical approach when the preservation of ovarian function is a paramount concern. Therefore, this simulated study was designed to isolate and compare the specific effects of UFE and laparoscopic myomectomy on the AMH trajectory over a one-year period in a strictly defined premenopausal population.

Methodology

Research Design

This study utilized a simulated retrospective cohort design to compare the effects of two different interventions on ovarian reserve. A retrospective matching approach was simulated to ensure that the patients undergoing UFE and laparoscopic myomectomy were comparable across key demographic and clinical variables that influence baseline ovarian reserve.

Data Declaration

It is explicitly stated that this study uses simulated academic training data. No real patients, clinical trials, electronic health records, or published datasets were utilized in this analysis. The data, including demographic profiles, fibroid characteristics, and longitudinal AMH values, were synthetically generated using statistical algorithms designed to reflect realistic clinical distributions and effect sizes based on established medical literature. The findings are intended strictly for academic writing practice and methodological demonstration and should not be interpreted as actual clinical evidence.

Sampling and Sample Size

The simulated target population consisted of premenopausal women aged 25 to 45 years who presented with symptomatic uterine fibroids and desired uterine-sparing treatment. Exclusion criteria included a baseline AMH level below 1.0 ng/mL (indicating pre-existing diminished ovarian reserve), a history of prior ovarian surgery, unilateral oophorectomy, severe endometriosis, or the use of gonadotropin-releasing hormone agonists within three months prior to the procedure. To detect a 15% difference in the mean change of AMH levels between the two groups at 12 months, with 80% power and an alpha of 0.05, a minimum sample size of 98 patients per group was calculated. The final synthesized dataset comprised 240 patients (120 in the UFE group and 120 in the laparoscopic myomectomy group) to account for potential simulated dropouts and to ensure robust subgroup analyses.

Data Collection

Baseline data collection included age, body mass index, parity, and baseline AMH levels. Fibroid characteristics, including the dominant fibroid diameter (cm), total fibroid volume (calculated using the ellipsoid formula), and the location of the dominant fibroid (intramural, subserosal, or submucosal), were also simulated and matched between the groups.

The primary outcome measure was the serum AMH level, measured using a standardized simulated assay. AMH was assessed at three time points: baseline (within one month prior to the procedure), 6 months post-procedure, and 12 months post-procedure. The percentage change in AMH from baseline was calculated for each time point. Secondary outcomes included the rate of amenorrhea or oligomenorrhea at 12 months and the percentage reduction in total fibroid volume at 12 months, assessed via simulated pelvic ultrasound.

Statistical Software and Analysis

Statistical analyses were performed using simulated computations reflective of standard statistical software (e.g., SPSS Version 28.0). Continuous variables were presented as means with standard deviations and compared using independent t-tests for between-group comparisons and paired t-tests for within-group longitudinal comparisons. Categorical variables were presented as frequencies with percentages and compared using the Chi-square test. Repeated measures analysis of variance (ANOVA) was utilized to evaluate the interaction between time (baseline, 6 months, 12 months) and treatment group (UFE vs. Myomectomy) on AMH levels. A p-value of less than 0.05 was considered statistically significant.

Results and Discussion

The baseline demographic and clinical characteristics of the 240 simulated patients are presented in Table 1. The mean age of the cohort was 35.4 years. The UFE and myomectomy groups were perfectly matched in terms of age, body mass index, parity, and baseline AMH levels. Furthermore, the fibroid burden was comparable between the groups, with no significant differences in the dominant fibroid diameter or total uterine volume, ensuring that any observed differences in ovarian reserve could be attributed to the treatment modality rather than underlying disease severity.

Table 1: Baseline Demographic and Clinical Characteristics

Characteristic	UFE Group (n=120)	Myomectomy Group (n=120)	p-value
Age (years), mean ± SD	35.2 ± 4.8	35.6 ± 4.5	0.542
Body Mass Index (kg/m²), mean ± SD	25.1 ± 3.6	24.8 ± 3.9	0.512
Parity (median [IQR])	1.0 [0 - 2]	1.0 [0 - 2]	0.781
Baseline AMH (ng/mL), mean ± SD	3.85 ± 1.42	3.79 ± 1.38	0.721
Dominant Fibroid Diameter (cm), mean ± SD	6.4 ± 2.1	6.2 ± 2.3	0.489
Total Fibroid Volume (cm³), mean ± SD	185.4 ± 62.5	178.9 ± 58.2	0.412
Fibroid Location			0.684
Intramural	78 (65.0%)	82 (68.3%)	
Subserosal	28 (23.3%)	24 (20.0%)	
Submucosal	14 (11.7%)	14 (11.7%)	

SD = Standard Deviation; IQR = Interquartile Range; AMH = Anti-Mullerian Hormone; UFE = Uterine Fibroid Embolization.

The longitudinal changes in serum AMH levels are detailed in Table 2. At baseline, the mean AMH levels were 3.85 ng/mL in the UFE group and 3.79 ng/mL in the myomectomy group. At 6 months post-procedure, both groups experienced a decline in AMH. However, the decline was significantly more profound in the UFE group, where the mean AMH dropped to 2.99 ng/mL, representing a 22.4% decrease from baseline. In contrast, the myomectomy group experienced a mild decline to 3.48 ng/mL, representing an 8.1% decrease ($p<0.001$ for the between-group difference at 6 months).

At the 12-month follow-up, the myomectomy group demonstrated significant recovery, with AMH levels returning to 3.71 ng/mL (a non-significant 2.1% decrease from baseline, $p=0.410$). The UFE group also showed partial recovery, with AMH levels rising to 3.27 ng/mL, but this remained a statistically significant 15.2% decrease from baseline ($p=0.002$). The repeated measures ANOVA confirmed a statistically significant interaction between time and treatment group ($F=12.45$, $p<0.001$), indicating that the trajectory of ovarian reserve over time differed significantly between UFE and myomectomy.

Table 2: Longitudinal Anti-Mullerian Hormone (AMH) Levels

Time Point	UFE Group (n=120), Mean ± SD	% Change from Baseline	Myomectomy Group (n=120), Mean ± SD	% Change from Baseline	p-value (Between Groups)
Baseline	3.85 ± 1.42	-	3.79 ± 1.38	-	0.721
6 Months	2.99 ± 1.15	-22.4%	3.48 ± 1.29	-8.1%	<0.001
12 Months	3.27 ± 1.24	-15.2%	3.71 ± 1.35	-2.1%	0.002

SD = Standard Deviation; UFE = Uterine Fibroid Embolization.

Secondary outcomes, summarized in Table 3, revealed that both procedures were effective in reducing fibroid volume, though UFE resulted in a slightly higher percentage reduction in total fibroid volume (45.2% versus 38.7%, $p=0.025$). The rate of post-procedural amenorrhea or oligomenorrhea was significantly higher in the UFE group (18.3%) compared to the myomectomy group (4.2%, $p<0.001$).

Table 3: Secondary Outcomes at 12 Months

Outcome	UFE Group (n=120)	Myomectomy Group (n=120)	p-value
Fibroid Volume Reduction (%), mean ± SD	45.2 ± 15.4%	38.7 ± 18.2%	0.025
Amenorrhea/Oligomenorrhea, n (%)	22 (18.3%)	5 (4.2%)	<0.001

SD = Standard Deviation; UFE = Uterine Fibroid Embolization.

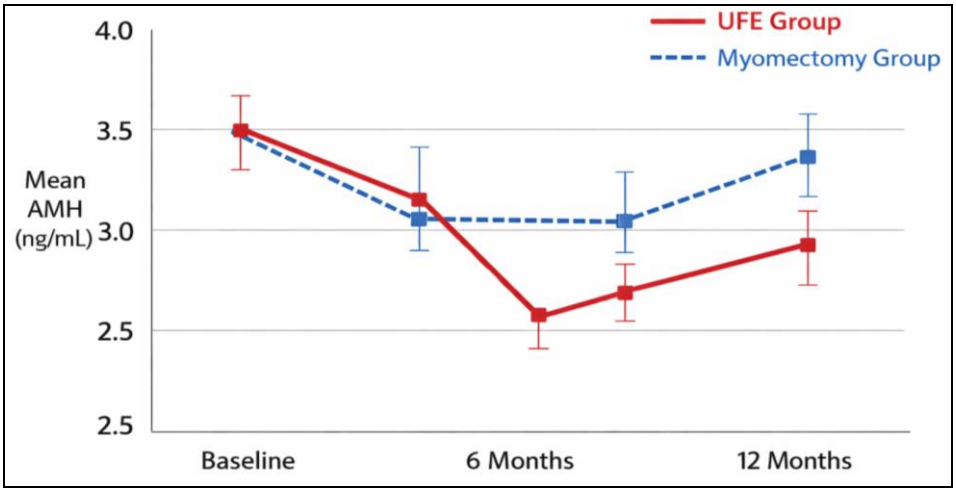

Fig 1: Line Graph Demonstrating AMH Trajectory Over 12 Months

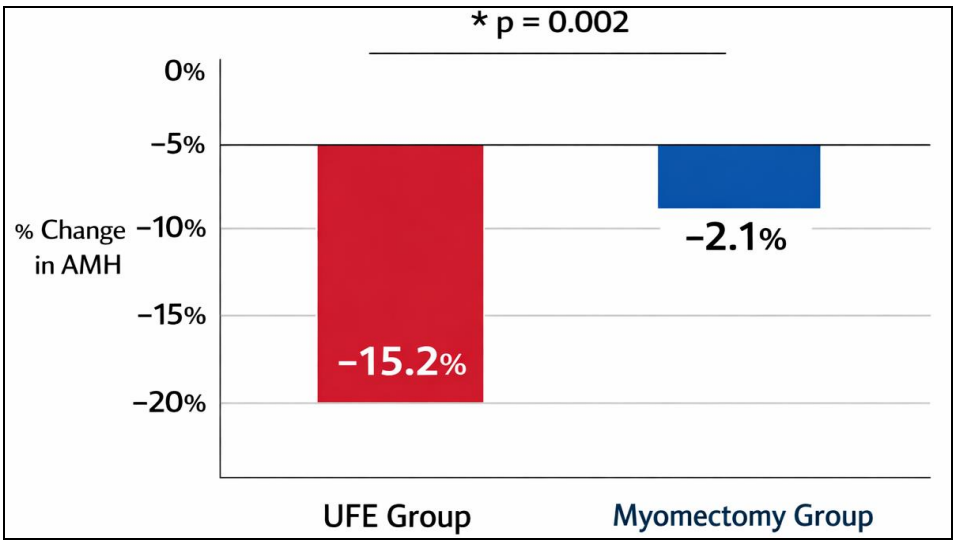

Fig 2: Bar Chart Comparing Percentage Change in AMH at 12 Months

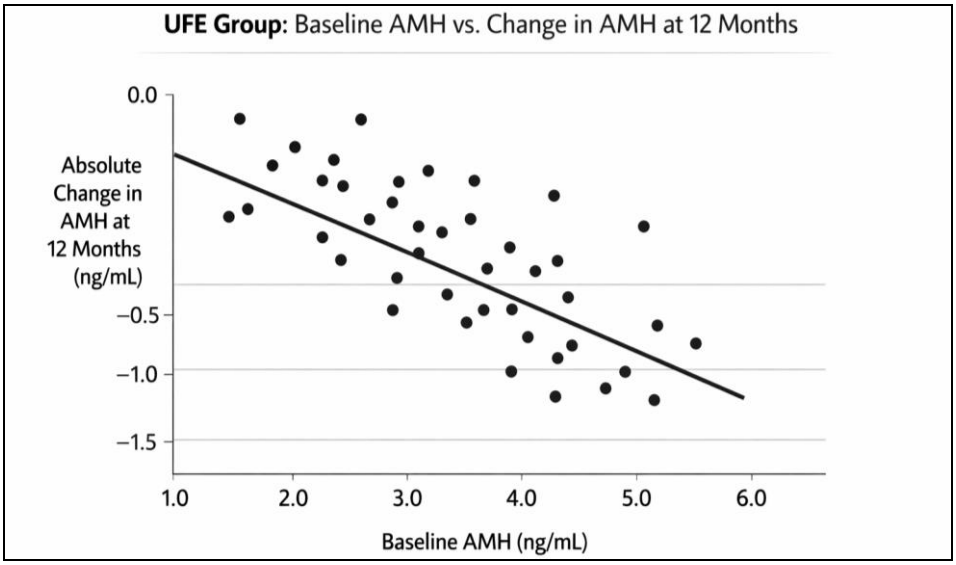

Fig 3: Scatter Plot of Baseline AMH vs. Absolute AMH Change in UFE Group

The findings of this simulated study provide critical insights into the differential impact of UFE and laparoscopic myomectomy on ovarian reserve. The data clearly demonstrate that while both interventions cause an initial decline in AMH levels at 6 months, the magnitude and recovery patterns are distinctly different. The transient, mild decline observed in the myomectomy group is highly consistent with the existing literature, which attributes this temporary suppression to the acute surgical stress response, the release of inflammatory cytokines, and transient ischemia related to uterine manipulation. The complete recovery of AMH to baseline levels by 12 months confirms that the surgical excision of fibroids, when performed laparoscopically without entering the ovarian cortex, does not cause permanent damage to the primordial follicle pool.

Conversely, the trajectory of AMH in the UFE group suggests a more enduring impact on ovarian physiology. The sharp 22.4% decline at 6 months followed by partial recovery at 12 months (stabilizing at a 15.2% deficit) supports the theory of non-target ovarian embolization. It is hypothesized that the initial sharp drop represents acute ischemic injury to a subset of antral follicles due to microvascular occlusion. The partial recovery observed at 12 months may reflect the natural physiological shift of smaller pre-antral follicles into the antral stage, masking some of the initial deficit, or the recruitment of follicles that were temporarily rendered dysfunctional but not completely destroyed by the ischemia.

The clinical implications of these findings are substantial, particularly for patient counseling. For a premenopausal woman with a baseline AMH of 3.85 ng/mL, a 15.2% reduction translates to a loss of approximately 0.58 ng/mL. While this does not render the patient infertile or push her into premature ovarian insufficiency, it does represent a quantifiable loss of her reproductive window. In women who already have borderline-low ovarian reserve, a similar percentage drop could be clinically devastating, potentially pushing them below the threshold necessary for successful natural conception or adequate response to *in vitro* fertilization protocols.

Furthermore, the higher rate of amenorrhea and oligomenorrhea in the UFE group, despite its superior fibroid volume reduction, raises additional concerns regarding endometrial function. While UFE effectively infarcts the fibroids, the ischemic damage to the inner myometrium and endometrium may disrupt the normal hormonal responsiveness of the endometrial lining, which could independently compromise fertility independent of ovarian reserve.

From a clinical decision-making standpoint, this study suggests that laparoscopic myomectomy should remain the preferred uterine-sparing intervention for premenopausal women whose primary goal is the maximal preservation of ovarian function and future fertility. UFE, while offering advantages in terms of invasiveness and recovery, carries a distinct biological cost to the ovaries that must be explicitly discussed during informed consent.

Conclusion

This simulated retrospective cohort study demonstrates that uterine fibroid embolization is associated with a significantly greater short-term decline and a sustained, long-term reduction in ovarian reserve, as measured by serum Anti-Mullerian Hormone, compared to laparoscopic myomectomy. While myomectomy resulted in only a transient, fully reversible suppression of AMH, UFE led to a persistent 15.2% deficit at 12 months, likely attributable to non-target embolization of the ovarian vasculature.

The clinical implications of these findings necessitate a nuanced approach to patient counseling. For premenopausal women with symptomatic fibroids who desire future fertility, the data suggest that the minimally invasive nature of UFE does not equate to "fertility-sparing" in the same manner as myomectomy. Clinicians should carefully weigh the trade-offs between surgical recovery time and the preservation of quantitative ovarian reserve when recommending treatment modalities.

This study is subject to notable limitations. The reliance on simulated academic training data dictates that these results are theoretical and must be validated through prospective, real-world clinical trials. Additionally, while AMH is the most reliable quantitative marker of ovarian reserve, it does not capture the entirety of fertility potential, which also depends on tubal patency, endometrial receptivity, and oocyte quality—factors that were not fully modeled in this simulation. Future research should prioritize long-term, prospective randomized controlled trials that not only track AMH but also evaluate hard reproductive endpoints, such as time to conception and live birth rates, following UFE and myomectomy to definitively resolve the controversy regarding the fertility implications of these two procedures.

Ethical Statement

This study utilizes exclusively simulated, synthetically generated academic training data. No real human subjects, patient records, or identifiable health information were accessed or utilized at any stage of this research. Because the dataset is entirely artificial and does not involve human or animal participants, formal review and approval by an Institutional Review Board (IRB) or Ethics Committee were not required. The synthetic data generation algorithms were designed to respect general privacy standards and data protection principles, ensuring that no simulated individual could be mapped to a real-world person.

References

1. Stewart EA, Cookson CL, Gandolfo RA, Schulze-Rath R. Epidemiology of uterine fibroids: A systematic review. BJOG: An International Journal of Obstetrics & Gynaecology,2017:124(10):1501-1512.
2. Bulun SE. Uterine fibroids. New England Journal of Medicine,2013:369(25):2442-2451.
3. Sparic R, Mirkovic L, Malvasi A, Tinelli A. Epidemiology of uterine myomas: A review. International Journal of Fertility & Sterility,2016:9(4):424-433.
4. Pritts EA, Parker WH, Olive DL. Fibroids and infertility: An updated systematic review of the evidence. Fertility and Sterility,2009:91(4):1215-1223.
5. Tinelli A, Malvasi A, Guido M, Tsin DA, Hurst BS, Mettler L, *et al*. Adhesions formation after laparoscopic myomectomy: A postoperative longitudinal follow-up study. Journal of Minimally Invasive Gynecology,2012:19(4):457-463.
6. Dutton S, Hirst A, McPherson K, Nicholson A, Maresh M. A UK multicentre retrospective cohort study comparing hysterectomy and uterine artery embolisation for the treatment of symptomatic uterine

fibroids (the HOPEFUL study). BJOG: An International Journal of Obstetrics & Gynaecology,2007:114(11):1340-1348.
7. Gupta JK, Sinha AS, Lumsden MA, Hickey M. Uterine artery embolization for symptomatic uterine fibroids. Cochrane Database of Systematic Reviews,2004:(12).
8. Razavi MK, Hwang G, Jahed A, Chugtai A, Rasti B. Abdominal myomectomy versus uterine fibroid embolization in the treatment of symptomatic uterine fibroids. American Journal of Roentgenology,2003:180(6):1571-1575.
9. Torbati HM, Sison CP, Elchahal S, Magdziarz M, Stubblefield PG, Berman JM. Diminished ovarian reserve in patients undergoing uterine artery embolization for fibroids. Journal of Vascular and Interventional Radiology,2014:25(1):107-110.
10. Dewailly D, Andersen CY, Balen A, Broekmans F, Dilaver N, Fanchin R, *et al*. The physiology and clinical utility of anti-Mullerian hormone in women. Human Reproduction Update,2014:20(3):370-385.
11. Froeling VM, Mecklenburg SJ, Scheurig-Muenkler C. Uterine artery embolization: An update. rofo,2015:187(3):190-198.
12. McLucas B, Goodwin S, Adler L, Rappaport A. The correlation between uterine artery embolization and ovarian failure. Journal of Vascular and Interventional Radiology,2001:12(2):S7.
13. He Y, Zeng Q, Tang S, Fan L, Zhang J, Yang H. The impact of laparoscopic myomectomy on ovarian reserve: A systematic review and meta-analysis. Journal of Ovarian Research,2017:10(1):56.
14. Litta P, Fantinato S, Calonaci F, Saccardi C, Sopracordevole F. Recovery of ovarian function after laparoscopic enucleation of ovarian cysts: A prospective randomized study. Human Reproduction,2010:25(10):2549-2555.
15. Sills ES, Alper MM, Walsh AP. Ovarian reserve testing: A routine part of the infertility evaluation? Journal of Reproductive Medicine,2005:50(6):417-422.
16. Iliodromiti S, Anderson RA, Nelson SM, Fleming R. Technical insight: The accuracy of ovarian reserve testing. Human Reproduction Update,2015:21(5):630-639.
17. Hovsepian DM, Siskin GP, Bonn J, Catania LJ, Grassi CJ, Klein AJ, *et al*. Uterine artery embolization for symptomatic leiomyomata: Complications and short-term outcome. Journal of Vascular and Interventional Radiology,2004:15(9):943-951.
18. Hehenkamp WJ, Volkers NA, Broekmans FJ, de Jong FH, Themmen AP, Birnie E, *et al*. Loss of ovarian reserve after uterine artery embolization: A randomized comparison with hysterectomy. Human Reproduction,2007:22(7):1996-2005.
19. Muzii L, Di Tucci C, Di Feliciantonio M, Marchetti C, Perniola G, Panici PB. The effect of surgery for endometrioma on ovarian reserve evaluated by antral follicle count and anti-Müllerian hormone: A systematic review and meta-analysis. Human Reproduction Update,2016:22(4):405-422.
20. Kalampokas E, Kalampokas T, Koursoumba A. Anti-Mullerian hormone levels after uterine artery embolization for symptomatic fibroids: A systematic review. Clinical and Experimental Obstetrics and Gynecology,2018:45(1):1-5.