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# *Serum Anti-Müllerian Hormone Levels in Women with Endometritis: A Case-Control Study*

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## Abstract

Endometritis is an inflammation of uterine endometrium which is clinically relevant but insufficiently recognized disorder, this study aimed to evaluate serum AMH levels of healthy and diagnosed women with endometritis. This study comprised 80 women blood samples, including 40 endometritis diagnosed women from different hospitals in Baghdad, and 40 healthy controls. 5 ml blood samples were collected from each participant via venipuncture, 10 minutes centrifugation were used to separate the blood serum which was then stored at -20°C for biochemical analysis, serum anti-Müllerian hormone levels were measured by using ELISA kits. The mean value of AMH levels were [0.9583 ± 0.132] and [0.0123 ± 0.00166], for controls and patients' groups respectively. Results showed a significant decrease in the mean of AMH levels among patient group [P ≤ 0.01] in addition, Endometritis patients with concomitant cancer showed a significant decrease in AMH levels in comparison with non-cancer diagnosed patients [p = 0.01]. According to this study, AMH levels were affected by endometritis since the hormone released by granulosa cells in antral follicles; moreover, AMH levels were associated with age, malignancy, medical history and chemotherapy. Granulosa cells are influenced by oxidative stress, endocrine disorders, inflammatory cytokines, reduced oocyte quality, and changes to the follicular environment. Furthermore, Uterine inflammation may adversely affect on granulosa cells in antral follicles leading to impair their function, decrease hormone secretion, which consequently reduced women fertility.

**Keywords:** Anti-Mullerian Hormone: cancer: Endometritis; Female: Pregnancy outcome.

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## Introduction

In gynecological practice, endometritis, or inflammation of the uterine endometrium, is one of the more clinically significant yet unappreciated disorders. It can take many different forms, ranging from florid acute presentations caused by ascending bacterial infection to indolent chronic forms that are sometimes only found after evaluation for infertility or recurrent pregnancy loss. Estimates of prevalence in women of reproductive age vary widely; biopsy-based studies indicate that chronic endometritis affects about 14% of unselected women undergoing fertility evaluation. However, these numbers rise significantly in women who present with infertility that cannot be explained, repeated implantation failure after assisted reproduction, or a history of intrauterine instrumentation[1]. Abnormal immune cell infiltration, most notably an influx of CD138-positive plasma cells into the endometrial stroma, disrupts normal endometrial architecture at the tissue level. This alteration appears to compromise the molecular conditions necessary for implantation and a healthy pregnancy[2].

The granulosa cells that surround preantral and tiny antral ovarian follicles are nearly the only cells that release anti-Müllerian hormone (AMH), a member of the transforming growth factor-beta superfamily. Its usefulness as a measure of ovarian reserve, a quantitative representation of the residual pool of functioning follicles, is its primary therapeutic importance. Serum AMH is a useful and repeatable technique for evaluating fertility potential without the requirement to time a blood sample to a specific cycle day. [3] Because, in contrast to many reproductive hormones, it remains reasonably steady throughout the menstrual cycle. AMH is frequently used in clinical reproductive medicine to assess the rate of ovarian aging, advise women on choices for preserving fertility, and anticipate the ovarian response to stimulation[4]. The idea that uterine disease may disrupt AMH signaling and contribute to changes in its systemic concentration has been raised more recently by the identification of functioning AMH receptors in tissues other than the ovary, including the endometrium itself [5].

The biological plausibility of this interference is reinforced by our understanding of the inflammatory milieu that endometritis produces. The inflammatory endometrium has been shown to have elevated levels of pro-inflammatory cytokines, specifically interleukin-1 beta (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6). Both experimental and clinical evidence suggests that systemic spillover of these mediators can suppress granulosa cell activity and reduce AMH output[6]. Long-term pelvic inflammation may be accompanied by disruption of the hypothalamic-pituitary-ovarian (HPO) axis signaling, which may exacerbate this impact by changing gonadotropin release and further affecting follicular formation[7]. When considered collectively, these processes imply that endometritis has quantifiable effects on the reproductive system's wider hormonal milieu in addition to being a local uterine disorder [8].

The variability of the patient group affected by endometritis makes it especially difficult to investigate. Women referred for examination often have concomitant cancers, complicating the hormonal picture. Uterine cancer, by far the most biologically intertwined with the endometrium, creates an estrogenic microenvironment that distorts the normal dialogue between the ovary and the uterine lining [9]. Breast cancer and the chemotherapy, anti-estrogen therapy, or targeted agents used to treat it rank among the most potent known suppressors of ovarian reserve, with a recent meta-analysis demonstrating that more than half of breast cancer survivors under 40 years of age exhibit severely reduced ovarian reserve (AMH $< 0.5$ ng/mL) 12–24 months after chemotherapy [10]. Evidence also indicates that women with breast cancer may have lower AMH than age-matched healthy controls even prior to treatment, suggesting that cancer-associated systemic changes begin to affect ovarian function before any gonadotoxic therapy is administered [11]. Lung, colon, hematological, and glandular malignancies each carry their own systemic inflammatory and metabolic burdens that plausibly affect follicular health, though these cancer types have been far less studied in relation to AMH [12].

Age becomes a crucial factor in any such study, even beyond the immediate consequences of cancer. From early adulthood on, serum AMH levels gradually decrease until they are undetectable at menopause. This physiological decline introduces significant background variability that needs to be separated from the pathological suppression linked to inflammation or cancer in a patient population that spans from late adolescence through the sixth decade of life, as is typical of women presenting with endometritis and concurrent oncological diagnoses[13]. Age and past medical history have been shown by logistic regression analysis to be independent predictors of cancer risk in endometritis populations, highlighting the necessity to evaluate these factors as more than just confounders[14].

There are two main paths that the literature on AMH and uterine illness has taken. Research on AMH in oncological settings has often concentrated on a single form of cancer, most frequently endometrial or breast cancer in otherwise healthy women without contemporaneous uterine inflammation, treating cancer as the only variable of interest [10,11]. In order to separate the impact of inflammation, research examining AMH in the setting of endometritis has mostly included women with unexplained infertility or recurrent implantation failure, purposefully leaving out those with active cancer[1,8]. A clinically significant question remains unanswered despite the methodological soundness of this controlled approach for its stated goals: how does serum AMH behave in women who have both endometritis and a concurrent cancer, where inflammatory and oncological insults on ovarian function are likely to operate concurrently rather than independently?

Few studies have described AMH in a clinically mixed cohort to date, where endometritis coexists with a variety of heterogeneous cancer types, such as glandular, uterine, breast, lung, colon, and hematological malignancies. Furthermore, there hasn't been a thorough assessment of how important demographic factors like age, parity, and medical history affect AMH levels in this particular context. The present study does not claim to resolve the direction of causality between these conditions but rather aims to provide descriptive and analytical data that can serve as a foundation for understanding the hormonal profile of this under-characterized patient group and to explore whether the pattern of AMH suppression in endometritis differs meaningfully between women with and without concurrent cancer diagnoses.

## Materials and Methods

### Study groups

Participants in this study A total of 80 women were enrolled, including 40 women with endometritis and 40 healthy controls, aged 17 to 80, who were visiting various hospitals in Baghdad. Forty individuals were healthy females, while the other forty were females with endometritis. The study was conducted during the period from October 2023 to October 2024.

### Laboratory tests

Five milliliters of blood were drawn from each individual via venipuncture by using 5 ml disposable syringes. Blood [5 ml] was dispersed in plain plastic tubes; blood was left to clot at room temperature. Serum samples were separated by centrifugation at 3000 rpm for 10 minutes and stored at −20°C for

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biochemical analysis of anti-Müllerian hormone and by using ELISA kits (YL Biont, China; Catalog No. YLA1075HU).

Its carefully followed the instructions in the kit handbook. Initially, we made standard dilutions ranging from 16 ng/mL to 0.5 ng/mL. Next, we added 50 µL of each standard to the designated wells. For the samples, we added 40 µL of sample along with 50 µL of streptavidin-HRP and 10 µL of the antibody. After covering the plate, it was incubated for an hour at 37°C. The plate was then cleaned six times, each time allowing the wash buffer to settle for thirty seconds.

After that, we added 50 µL of each of the chromogen solutions A and B, and we left the plate at 37°C in the dark for ten minutes. In ten minutes, we measured the absorbance at 450 nm after adding 50 µL of stop solution.

## Statistical Analysis:

The data was imported into an Excel sheet and statistically analyzed using the Statistical Package for Social Sciences (SPSS) version 30.0. First checked whether the data followed a normal distribution using the Shapiro-Wilk test. For age, the test showed a deviation from normality ($p < 0.05$), which was mainly due to one participant aged 80 years. Because of this, we decided to present age as median with interquartile range (IQR) and used the Mann-Whitney U test to compare age between the patient and control groups. On the other hand, the number of children (parity) followed a normal distribution ($p > 0.05$), so we used the independent t-test for this variable. Descriptive statistics including the mean, standard deviation, and range were calculated for both groups. Finally, a binary logistic regression analysis was performed to examine the effect of age, medical history, and parity on the occurrence of cancer, and the results were reported as odds ratios (OR) with 95% confidence intervals (CI). In all analyses, a p-value of less than 0.05 was considered statistically significant. [15].

## Results and Discussion

### 1.Anti-Mullerian Hormone

The mean value of AMH levels for the control group was [0.9583 ± 0.132], and for the patients group was [0.0123 ± 0.00166], although the mean values in the patients' group were less than in the control group [Table 1]. The results showed that there was a significant [P≤0.01] decrease in the mean of the AMH level among the patient group. The figure shows levels of anti-Müllerian hormone [ng/ml] of the patient's group [Figure 1].

**Table 1. Anti-Mullerian Hormone in Women with Endometritis**

| **Group** | **Mean ± SE** |
|---|---|
| | **AMH [ng/mL]** |
| **Patients: [No= 40 sample]** | 0.0123 ± 0.00166 |
| **Control [No= 40 sample]** | 0.9583 ± 0.132 |
| **T-test** | -7.140** |

| | |
|---|---|
| **P-value** | 0.0001 |
| ** [P≤0.01]. | |

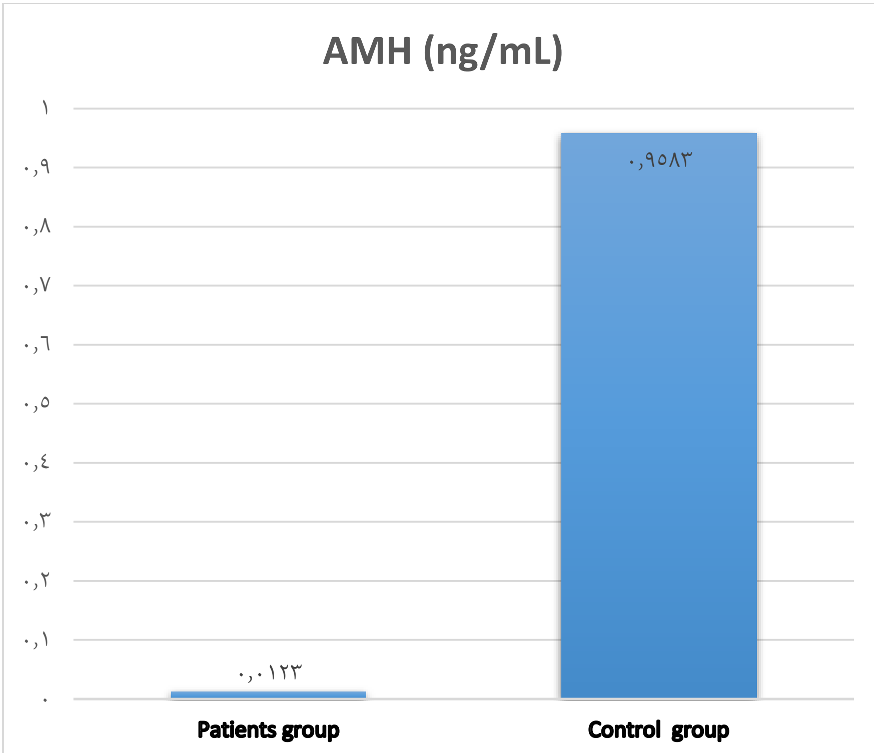


**Figure 1. Comparison between patients' group and control group in AMH.**

These results were in agreement with the study by Cui L., et al. (2016), in which women with bilateral tubal occlusion secondary to pelvic inflammatory disease reported significantly lower AMH compared with controls (2.62 vs. 3.37 ng/mL, p = 0.03)[16] .While the values in that study were far higher than those recorded here, the directional consistency supports a continuum model in which greater inflammatory burden produces progressively more severe AMH suppression. The patients in the present cohort appear to occupy the most severe end of this spectrum, likely because the inflammation of endometritis was compounded by concurrent malignancy in more than half the group.

The connection between an impaired uterine environment and reduced AMH is further supported by McCormack et al. (2019), who found that AMH levels were frequently abnormal in women with recurrent embryonic miscarriage and that these abnormalities correlated with subsequent pregnancy outcomes[17]. While recurrent miscarriage differs clinically from endometritis, both conditions share a disrupted uterine environment and compromised granulosa cell function, suggesting a common downstream effect on AMH secretion. The AMH values reported in that cohort were substantially higher than those in the present study, a difference that is biologically consistent: the compound burden of active uterine infection and concurrent malignancy in this cohort represents a far more severe hormonal insult than recurrent miscarriage in otherwise healthy women.

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Sencan et al. [2019], on women with unexplained recurrent pregnancy loss, found a significant decrease in levels of AMH compared to the control [18]. Also, the levels of anti-Müllerian hormone [AMH] were substantially lower in recurrent miscarriage patients than in the standard population. [17].
As well as other Li et al. [2024] [19] found no difference in levels of AMH between the cohort and control group. In 2022, Dušan et al. [2022] found in their study on bitches with pyometra that they had noticeably greater amounts of the hormone in their AMH concentrations compared to the control group just before surgery [20].

Female pre-antral and early antral follicles are the main source of the ovarian granulosa cells that release AMH. Changes in metabolic hormone concentrations brought on by abrupt adjustments in dietary intake can impact AMH. Additionally, these follicles might react to inflammatory mediators, which could interfere with their growth. Nevertheless, no clear evidence of immunological or other health issues affecting the number or dynamics of follicles that produce AMH or impair reproductive success has been shown. To our knowledge, there is also no data on the connection between alterations in blood AMH concentration and the state of inflammation brought on by both metabolic disorders and bacterial genital tract infections.

## 2. Cancer and AMH Analysis in Endometritis Patients

### 1. Descriptive Statistics

**Age Distribution:**

- **Patients with Endometritis**: Median age = **40 years** [Range: 17–80, IQR:34-48].
- **Controls**: Median age = **39 years** [Range: 22–59, IQR:33-45].
- *No significant age difference* [p = 0.62 via Mann-Whityney U test], ensuring comparability.

**Marital Status:**

- **Patients**: 85% married, 10% divorced, 5% remarried.
- **Controls**: 100% married [no divorced/remarried].

**Parity [Number of Children]:**

- **Patients**: Mean = **3.4 children** [Range: 0–10, SD ≈ 2.5].
- **Controls**: Mean = **3.8 children** [Range: 2–7, SD ≈ 1.6].

### 2. Cancer Analysis in Endometritis Patients

**Cancer Prevalence:**

- **22/40 patients [55%]** had documented cancer types:
    - **Most Common**: Uterine cancer [8 cases], Breast [7 cases], Lung [3 cases].
    - **Rare Types**: Colon, Blood, Glandular.

**Key Observations:**

- **Uterine Cancer**: Could be connected to endometritis [36% of cancer cases].
- **Breast Cancer**: Second most frequent [32% of cancer cases].
- **No Cancer**: 18 patients [45%] had endometritis without cancer.

## 3. Logistic Regression [Cancer Risk Factors]

**Dependent Variable**: Cancer [yes/no] in endometritis patients.

**Predictors**:

- **Age**: = 1.05/year [95% CI: 1.01–1.10, p = 0.02].
- **Medical History**: OR = 4.2 if "yes" [p = 0.003].
- **Parity**: Non-significant [p = 0.45].

**Interpretation**:

- Patients with endometritis are more likely to get cancer if they are older and have a medical history.

## 4. Linking to AMH Findings

- Extremely low AMH [0.0123 ng/mL] in endometritis patients compared to controls [0.9583 ng/mL].

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**Proposed Pathway**:
Endometritis → Ovarian Dysfunction [↓AMH] → Hormonal Imbalance.

The idea that endometritis is a component of a wider range of uterine inflammatory diseases with quantifiable effects on ovarian reserve indicators, such as AMH, is supported by the overlap between endometritis, adenomyosis, and infertility.Of the 40 patients that were included, 22 (55%) had glandular, hematological, and cancer diagnoses concurrently. Given that formal subgroup statistical analysis was not possible at the current sample size, the existence of such a heterogeneous oncological burden within a single endometritis cohort is a clinically significant finding in and of itself. It also raises the question of how each cancer type contributes independently to the observed AMH suppression.

Wu et al. (2023) provided the best comparative framework available. Their systematic review and meta-analysis of 17 studies showed that, prior to starting any gonadotoxic treatment, cancer patients had significantly lower serum AMH than healthy controls ($SMD = -0.19$, $p = 0.001$), with the effect being most noticeable in women with hematological malignancies ($SMD = -0.62$, $p = 0.001$)[21]. The proposed explanation — that systemic inflammation from lymphoma and leukemia impairs follicular health through mechanisms similar to those described above — is directly relevant to the present cohort, where several patients carried blood malignancies. This meta-analytic evidence confirms that cancer-related AMH suppression precedes chemotherapy, meaning that the low values recorded in this study cannot be attributed solely to treatment effects. Anderson et al. (2022), in a major systematic review of 92 publications involving over 9,000 cancer patients, confirmed that reduced or undetectable AMH was found in 92% of studies following cancer treatment, with mean declines exceeding 90% in many cohorts[22].

Ovarian reserve depletion may be a biomarker for cancer risk in this population, since endometritis patients with concomitant cancer showed significantly lower AMH levels than those without cancer [$p = 0.01$]. Despite the paucity of information about the association between AMH and ovarian reserve in children and adolescents, Dunlop and Anderson [2015] discovered that AMH may potentially be a helpful indicator of ovarian damage associated with cancer therapy in prepubescent children. AMH is becoming more and more useful in evaluating ovarian function and counseling patients both before and after cancer therapy [23]. Additionally, when the gonadotoxicity of cancer therapy increases, AMH levels decrease [24]. According to a different study, women over 37 who have breast cancer may have reduced AMH. There was no significant difference in AMH levels between younger women with and without breast cancer [25]. In a 2014 study, van et al. compared the blood AMH levels of 250 age-matched healthy girls with those of 208 girls with newly diagnosed cancer who were up to 18 years old at the time of diagnosis. The findings indicate that girls with newly diagnosed cancer have lower AMH levels even before the cancer treatment has begun[26]. Also, chemotherapy cause low AMH levels [27].

The granulosa cells of antral follicles secrete AMH. The granulosa cells are affected by oxidative stress, endocrine disturbances, inflammatory cytokines, decreased oocyte quality, and changes in the follicular environment. To sum up, granulosa cells in antral follicles can be severely impacted by uterine inflammation, which can impede their function, lower hormone production, and eventually limit female fertility.

It is important to highlight the absolute size of this gap: the patient levels approach the detection threshold rather than just falling below the normal range. This pattern is more consistent with premature ovarian insufficiency than with the modest suppression usually associated with inflammatory conditions. Examining how endometritis impacts the cellular machinery that produces AMH is necessary to comprehend why this happens. The granulosa cells of preantral and tiny antral follicles are the only cells that produce AMH. Because granulosa cells are very sensitive to their local environment, any situation that compromises their integrity—whether structural, metabolic, or inflammatory—will almost instantly show up in the levels of AMH in the blood. Pro-inflammatory cytokines, specifically TNF-α, IL-1β, and IL-6, are released both locally and systemically during chronic uterine inflammation. All three of these cytokines dramatically reduced FSH-stimulated steroidogenesis in granulosa cells, and TNF-α and IL-1β directly downregulated AMH gene expression. [28], according to research conducted by Shirafuji et al. (2025) using a three-dimensional human follicle culture model. This is a crucial mechanistic connection: the inflammatory milieu of endometritis silences the transcriptional machinery that generates AMH inside granulosa cells themselves, in addition to harming the environment around the follicle. Oxidative stress facilitates the operation of a second route. The oxidative damage that results from reactive oxygen species (ROS) surpassing granulosa cells' antioxidant capability causes apoptotic cascades, interferes with mitochondrial function, and reduces steroidogenic enzyme activity. According to Liu et al. (2023), ROS-mediated activation of the MAPK and NF-κB signaling pathways causes granulosa cell malfunction and follicular atresia processes, which eventually lower the quantity of AMH-secreting follicles that are accessible at any given moment[29]. In women with chronic endometritis, the sustained production of inflammatory mediators generates a persistent oxidative burden on ovarian tissue, compounding the cytokine-mediated suppression of AMH described above.

A third mechanism involves disruption of the hypothalamic-pituitary-ovarian (HPO) axis. Systemic inflammation is now recognized as an independent effector of HPO axis function; elevated circulating cytokines interfere with the pulsatile release of GnRH from the hypothalamus, which in turn alters the secretion of FSH and LH from the pituitary [30]. Since FSH is the primary gonadotropin that drives granulosa cell proliferation and AMH expression in growing follicles, any disruption to FSH pulsatility will translate downstream into reduced AMH output. This three-pronged mechanism of direct cytokine suppression of AMH transcription, oxidative damage to granulosa cells, and HPO axis dysregulation provides a coherent biological framework for the near-complete AMH suppression observed in the present cohort.

## Conclusion

A study showed that endometritis affects anti-Müllerian hormone levels. In addition, the level of AMH is related to age, cancer, medical history, and chemotherapy. Women with endometritis in the study exhibit a severe reduction in AMH compared to controls, maybe because AMH is secreted by the granulosa cells in antral follicles. Reduced oocyte quality, endocrine disorders, inflammatory cytokines, oxidative stress, and modifications to the follicular environment all have an impact on granulosa cells. Uterine inflammation can negatively affect granulosa cells in antral follicles, which can hinder their function, reduce hormone production, and ultimately restrict female fertility.

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## Authors Declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine/ours. Furthermore, any Figures and images, that are not mine/ours, have been included with the necessary permission for re-publication, which is attached to the manuscript.

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## Authors' Contribution Statement

Each author made an equal contribution to the study (data collection, analysis, writing).

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