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REPRODUCTIVE PHYSIOLOGY AND DISEASE

The effect of endometriosis on the antimüllerian hormone level in the infertile population

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Abstract

Purpose To determine whether the presence of endometriosis in infertile women without prior ovarian surgery influences markers of ovarian reserve, AMH and FSH.

Methods A retrospective cohort study included three groups of women who presented for IVF treatment at our tertiary care center from 04/27/2015 to 05/31/2017: women with endometriosis and prior ovarian surgery (EnSx), women with endometriosis without prior ovarian surgery (En), and women with a primary diagnosis of male factor infertility (MF; reference group).

Results There were 671 patients that met inclusion criteria (78 EnSx, 60 En, and 533 MF). Compared to the MF group (3.6 ± 3.0), a lower mean AMH level (ng/mL) was observed in the EnSx group (2.5 ± 2.5; aβ − 1.21; 95% CI [− 1.79, −0.62]) and in the En group (2.5 ± 2.2; aβ − 1.11; 95% CI [− 1.68, − 0.54]). Both endometriosis groups had a statistically significantly higher proportion of patients with an AMH < 1 (EnSx, 24.4%; OR, 2.39 [95% CI, 1.31, 4.36]; En, 28.3%; OR, 2.67 [95% CI, 1.41, 5.08]) compared to the MF group (13.9%). The mean baseline FSH level (lU/L) was statistically significantly higher in both endometriosis groups (EnSx, 8.6 ± 4.3; β, 1.37 [95% CI, 0.39, 2.34]; En, 8.4 ± 3.7; β, 0.96 [95% CI, 0.04, 1.87]) compared to the MF group (7.3 ± 2.2).

Conclusions Among infertility patients with endometriosis, with and without a history of ovarian surgery, ovarian reserve markers were worse (lower AMH and higher FSH) and a higher proportion had decreased ovarian reserve as measured by AMH compared to women with MF.

Keywords Endometriosis · Antimüllerian hormone · AMH · Ovarian reserve · Infertility

Introduction

A woman's reproductive lifespan is guided by ovarian reserve, the pool of primordial follicles available for follicular recruitment and maturation [1]. Several markers are commonly used to measure ovarian reserve, including antimüllerian hormone (AMH), follicle-stimulating hormone (FSH), estradiol, and antral follicle count. AMH levels have been identified as a reliable method to determine a woman's remaining oocyte supply and to help guide prognostic discussions related to infertility [2, 3]. In addition, AMH levels are relatively stable throughout the menstrual cycle and can therefore be drawn at any time making this an easier value to obtain for both patients and clinicians [4, 5]. For these reasons, this hormone has become a commonly used proxy for monitoring natural and iatrogenic declines in ovarian function over time in the infertile population [6, 7].

AMH levels in women with endometriosis may be lower than the general population. In women with endometriomas that are surgically managed, this may be caused by direct oocyte injury which can occur at the time of laparoscopic stripping of endometriomas [8, 9]. Yet, research has shown that even women who have an endometrioma and do not have surgery have lower AMH levels and a faster rate of AMH decline compared to healthy controls [10]. Accelerated rates of decline have been associated with a shorter time to menopause and a shorter reproductive window [11]. An earlier age at menopause not only affects a woman's fertility potential but has a significant impact

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on her long-term health and mortality [12, 13]. Thus, it is important to understand which disease processes may pathologically decrease a woman's reproductive lifespan.

Currently, it is not well understood what effect endometriosis has on a woman's ovarian reserve or to what extent this disease influences the rate of AMH decline in these patients. The objective of this study was to determine whether the presence of endometriosis in women without a history of ovarian surgery influences markers of ovarian reserve.

Methods

This was a retrospective cohort study of women with surgically diagnosed endometriosis who initiated an evaluation for IVF treatment at our infertility clinic between 06/01/2012 and 05/31/2017. A comparison group of women with a primary infertility diagnosis of male factor infertility and no history of endometriosis who presented to our clinic during the study period were also included. Data were collected from our prospectively maintained departmental database and the hospital electronic medical record system. Exclusion criteria included patient age ≥ 41.0 years, diagnosis of anovulation, or a history of oophorectomy, hysterectomy, or gonadotoxic chemotherapy.

There were three patient groups included in this study. The first group included women with surgically diagnosed endometriosis and any history of prior ovarian surgery (EnSx) [$n = 78$], which typically included a cystectomy or ovarian fulguration. A second group included women with surgically diagnosed endometriosis without prior ovarian surgery (En) [$n = 60$]. A third group included women with a primary infertility diagnosis of male factor infertility [$n = 533$, referent group]. The groups were further subdivided into their corresponding Society for Assisted Reproductive Technology (SART) age group. Surgical staging of endometriosis utilized the revised American Society for Reproductive Medicine scoring system [14]. Patients were either staged at the time of surgery or retrospectively staged by one of the authors based on the findings dictated in the operative note (PR). Ovarian surgery was performed with various methods including cold scissor excision, electrocautery, and CO_2 laser depending on the surgeon preference, the pathology involved, and the indication for surgery.

Baseline AMH, FSH, and estradiol were collected between days 2 and 4 of the patient's menstrual cycle as part of the routine workup for infertility. Serum testing of FSH and estradiol was performed at Brigham and Women's Reproductive Endocrinology Laboratory. The intra-assay coefficients of variation (CV) for both the FSH and estradiol assay were less than 5%. The inter-assay correlation coefficients for both the FSH and estradiol assay were greater than 0.999.

AMH samples were shipped to the Mayo Medical Laboratories in Rochester, Minnesota for analysis. At the beginning of the study period, this laboratory utilized the Beckman Coulter Generation II enzyme-linked immunosorbent assay (ELISA) to measure AMH level in a sample. The laboratory then transitioned to the Ansh assay for AMH level measurement on 4/27/2015. For the Beckman AMH assay, the inter-assay CV and the intra-assay CV was less than 10%. For the Ansh AMH assay, the inter-assay CV and intra-assay CV was less than 10%. The AMH assay method changed during the study period as described above. A comparative evaluation between the Beckman assay and the Ansh assay reported a good correlation among these two assays on parallel samples (Spearman correlation coefficient = 0.917) [15]. In order to account for any differences in calibration between the two assays, the Passing-Bablok regression equation Ansh = 1.767 × Beckman − 0.167 (ng/mL), reported by Li et al., was used [15].

This study was approved by the Partners Institutional Review Board at the Brigham and Women's Hospital. Linear regression using "robust" standard errors were used to estimate the beta coefficients (β) with a 95% confidence interval (CI) and logistic regression models were used to estimate odds ratios (OR) with a 95% CI. The analysis was adjusted a priori for patient age, body mass index (BMI), race (white vs. non-white), and smoking history due to the known effect between these demographic characteristics and markers of ovarian reserve [16–19]. Each group was further subdivided into SART age groups for analysis of baseline AMH. A test for linear trend was performed for the mean baseline AMH by stage of endometriosis analysis. Statistical analyses were performed using Statistical Analysis Software version 9.3 (SAS Institute, Inc.).

Results

This study consisted of 671 patients. Among them, 78 had endometriosis and any history of prior ovarian surgery, 60 had endometriosis without prior ovarian surgery, and 533 had male factor infertility. Among all patients with surgically staged endometriosis, 20 (14.9%) were stage I, 16 (11.9%) were stage II, 29 (21.6%) were stage III, and 69 were stage IV (51.5%). In four patients, the surgical stage was not available. Demographic characteristics for patients are shown in Table 1. Age, BMI, and race are known confounders of ovarian reserve markers and were comparable between groups. Patients with endometriosis and prior ovarian surgery had a mean age of 33.6 ± 3.9 years and BMI of 24.6 ± 5.0 kg/m^2. Patients with endometriosis without prior ovarian surgery had a mean age of 34.2 ± 3.8 years and BMI of 24.4 ± 4.7 kg/m^2. Patients with male factor infertility had a mean age of 34.1 ± 3.6 years and BMI of 26.1 ± 6.6 kg/m^2. The proportion of patients classified as white race in each group was 59.0% in the endometriosis and prior ovarian surgery group, 73.3% in the endometriosis without prior ovarian surgery group, and 69.2% in the male factor infertility group. The proportion of

Table 1 Demographic characteristics among women with endometriosis and male factor infertility

Characteristics at baseline AMH	Endometriosis and prior ovarian surgery ($n = 78$)	Endometriosis without prior ovarian surgery* ($n = 60$)	Male factor infertility ($n = 533$)
Age (years)	33.6 ± 3.9	34.2 ± 3.8	34.1 ± 3.6
< 35	47 (60.3)	31 (51.7)	315 (59.1)
35–37	19 (24.4)	20 (33.3)	130 (24.4)
38–40	12 (15.4)	9 (15.0)	88 (16.5)
BMI (kg/m^2)	24.6 ± 5.0	24.4 ± 4.7	26.1 ± 6.6
< 25	49 (62.8)	39 (65.0)	311 (58.4)
25–25.9	17 (21.8)	13 (21.7)	118 (22.1)
≥ 30	12 (15.4)	8 (13.3)	104 (19.5)
Race			
White	46 (59.0)	44 (73.3)	369 (69.2)
Black	2 (2.6)	8 (13.3)	37 (6.9)
Asian	18 (23.1)	7 (11.7)	71 (13.3)
Hispanic	5 (6.4)	0 (0.0)	29 (5.4)
Other/declined	7 (9.0)	1 (1.7)	27 (5.1)
Never smoker	69 (88.5)	53 (88.3)	463 (86.9)
Former smoker	9 (11.5)	4 (6.7)	60 (11.3)
Current smoker	0 (0)	3 (5.0)	10 (1.9)
Gravidity†	0.5 ± 0.8 0 (0–1)	0.6 ± 1.1 0 (0–1)	0.6 ± 1.0 0 (0–1)
Parity†	0.2 (0.5) 0 (0–0)	0.2 (0.4) 0 (0–0)	0.2 (0.5) 0 (0–0)
Assay type			
Beckman Coulter Ansh ultrasensitive	49 (62.8) 29 (37.2)	31 (51.7) 29 (48.3)	293 (55.0) 240 (45.0)
Stage of endometriosis	$n = 76$	$n = 58$	
I	1 (1.3)	19 (32.8)	–
II	5 (6.6)	11 (19.0)	–
III	20 (26.3)	9 (15.5)	–
IV	50 (65.8)	19 (32.8)	–
Clinical pregnancy after first embryo transfer	($n = 74$) 31 (41.9)	($n = 50$) 19 (38.0)	($n = 497$) 206 (38.7)

AMH, antimüllerian hormone; BMI, body mass index

Data are mean ± standard deviation or *n* (%) unless otherwise noted

*Ovarian surgery was performed with various methods including cold scissor excision, electrocautery, and CO_2 laser depending on the surgeon preference, the pathology involved, and the indication for surgery

† Data are mean ± standard deviation and median (interquartile range)

current or former smokers in each group was 11.5% in the endometriosis and prior ovarian surgery group, 11.7% in the endometriosis without prior ovarian surgery group, and 13.1% in the male factor infertility group.

Baseline ovarian reserve measurements for each group are shown in Table 2. The overall mean baseline AMH level (ng/mL) was statistically significantly lower in both endometriosis groups (EnSx, 2.5 ± 2.5; β, − 1.21 [95% CI, − 1.79, − 0.62]; En, 2.5 ± 2.2; β, − 1.11 [95% CI, − 1.68, − 0.54]) compared to the male factor infertility group (3.6 ± 3.0). This pattern remained consistent among patients less than 35 years old in both endometriosis groups (EnSx, 3.0 ± 2.6; β, − 1.22 [95% CI, − 2.01, − 0.42]; En, 2.9 ± 2.5; β, − 1.27 [95% CI, − 2.13, − 0.42]) compared to the male factor infertility group (4.1 ± 2.9). Among women age 35–37, both endometriosis groups suggested lower mean AMH levels compared to the male factor infertility group (3.5 ± 3.2); however, only patients with endometriosis without prior ovarian surgery reached the threshold of statistical significance (EnSx, 2.3 ± 2.6; β, − 1.12 [95% CI, − 2.49, 0.25]; En, 2.3 ± 1.8; β − 1.10 [95% CI, − 2.00, − 0.20]). Conversely, among women 38–40, only patients with endometriosis and prior

Table 2 Baseline measurements of ovarian reserve

Baseline values	Endometriosis and prior ovarian surgery ($n = 78$)	Endometriosis without prior ovarian surgery ($n = 60$)	Male factor infertility ($n = 533$)
Baseline AMH (ng/mL)	2.5 ± 2.5 − 1.21 (− 1.79, − 0.62)	2.5 ± 2.2 − 1.11 (− 1.68, − 0.54)	3.6 ± 3.0 0.00 (Ref)
Baseline AMH by SART age group			
< 35 ($n = 393$)	($n = 47$) 3.0 ± 2.6 − 1.22 (− 2.01, − 0.42)	($n = 31$) 2.9 ± 2.5 − 1.27 (− 2.13, − 0.42)	($n = 315$) 4.1 ± 2.9 0.00 (Ref)
35–37 ($n = 169$)	($n = 19$) 2.3 ± 2.6 − 1.12 (− 2.49, 0.25)	($n = 20$) 2.3 ± 1.8 − 1.10 (− 2.00, − 0.20)	($n = 130$) 3.5 ± 3.2 0.00 (Ref)
38–40 ($n = 109$)	($n = 12$) 1.1 ± 1.0 − 1.06 (− 1.82, − 0.31)	($n = 9$) 1.6 ± 1.5 − 0.48 (− 1.48, 0.53)	($n = 88$) 2.1 ± 2.1 0.00 (Ref)
AMH < 1*	19 (24.4%) 2.39 (1.31, 4.36)	17 (28.3%) 2.67 (1.41, 5.08)	74 (13.9%) 1.00 (Ref)
AMH undetectable*	4 (5.1%) 2.39 (0.75, 7.64)	6 (10.0%) 4.64 (1.67, 12.95)	13 (2.4%) 1.00 (Ref)
Day 3 FSH (lU/L)	8.6 ± 4.3 1.37 (0.39, 2.34)	8.4 ± 3.7 0.96 (0.04, 1.87)	7.3 ± 2.2 0.00 (Ref)
Day 3 estradiol (pg/mL)	45 ± 32 6.73 (− 0.45, 13.91)	48 ± 56 8.58 (− 5.76, 22.92)	38 ± 22 0.00 (Ref)

AMH, antimüllerian hormone; SART, Society for Assisted Reproductive Technology; FSH, follicle-stimulating hormone

▲ Data are mean ± standard deviation with beta coefficient (95% confidence interval). Linear regression models using robust standard errors adjusted a priori for patient age, body mass index, race, and smoking status unless otherwise noted

*Data are *n* (%) with odds ratio (95% confidence interval). Logistic regression models adjusted a priori for patient age, body mass index, race, and smoking status

ovarian surgery had a statistically significantly lower mean AMH level (EnSx, 1.1 ± 1.0; β − 1.06 [95% CI, − 1.82, − 0.31]; En, 1.6 ± 1.5; β, − 0.48 [95% CI, − 1.48, 0.53]) compared to the male factor infertility group (2.1 ± 2.1). Compared to the male factor infertility group (13.9%), both endometriosis groups had a statistically significantly higher proportion of patients with an AMH < 1 (EnSx, 24.4%; OR, 2.39 [95% CI, 1.31, 4.36]; En, 28.3%; OR, 2.67 [95% CI, 1.41, 5.08]). There was no statistically significant difference for the ovarian reserve markers between the two endometriosis groups.

The mean AMH level by patient age for all women with endometriosis and for women with male factor infertility is depicted in Fig. 1. There was no statistically significant difference between the two regression equations. However, women with endometriosis had a statistically significant lower *X*-axis intercept (p = <0.001) and therefore, regardless of age, these women were observed to have a lower AMH level.

The mean baseline FSH level (lU/L) was statistically significantly higher in both endometriosis groups (EnSx, 8.6 ± 4.3; β, 1.37 [95% CI, 0.39, 2.34]; En, 8.4 ± 3.7; β, 0.96 [95% CI, 0.04, 1.87]) compared to the male factor infertility group (7.3 ± 2.2). Estradiol level (pg/mL) was not significantly different in either endometriosis group when compared to the male factor infertility group.

For all patients with endometriosis, the mean baseline AMH level by the stage of endometriosis is shown in Table 3. There was no correlation observed between AMH and increasing stage of endometriosis (test for linear trend, p = 0.58).

Discussion

The objective of this study was to identify whether ovarian reserve markers are worse among women with endometriosis without prior ovarian surgery. In the cohort of patients who presented to our IVF unit, it was observed that women with endometriosis without prior ovarian surgery had lower mean AMH levels, a higher proportion with AMH levels less than 1.0 ng/mL, and higher mean FSH levels when compared to women with male factor infertility. There was no difference in baseline estradiol level. These findings were also observed in the group of women with endometriosis and prior ovarian surgery when compared to women with male factor infertility. There was no identified association between stage of endometriosis and AMH level or between the two endometriosis groups.

It has previously been established that women with endometriosis who undergo ovarian surgery are at risk for iatrogenic damage to ovarian reserve, related to thermal injury or devascularization [8, 9]. Our findings provide evidence that infertile women with endometriosis may have a lower ovarian reserve, compared to a control group, regardless of whether they had prior ovarian surgery. This finding is consistent with prior studies which have observed that women with mild endometriosis [20] and women with a non-surgical endometrioma [10] had lower baseline levels of AMH compared to women without these conditions.

The hypothesized mechanism behind lower ovarian reserve in patients with endometriosis is through the known chronic inflammation and altered immune modulation that is related to the disease [21, 22]. Inflammation has previously been shown to cause follicular damage and dysfunction in other diseases such as autoimmune primary ovarian insufficiency [23]. Women with ovarian autoimmunity can develop steroidogenic autoantibodies and an inflammatory immune response which can lead to premature follicular damage and early onset menopause [24]. In patients with endometriosis, the inflammatory response caused by the disease could similarly lead to follicular dysfunction and damage the quality or quantity of the ovarian follicular pool. This could explain the finding in this

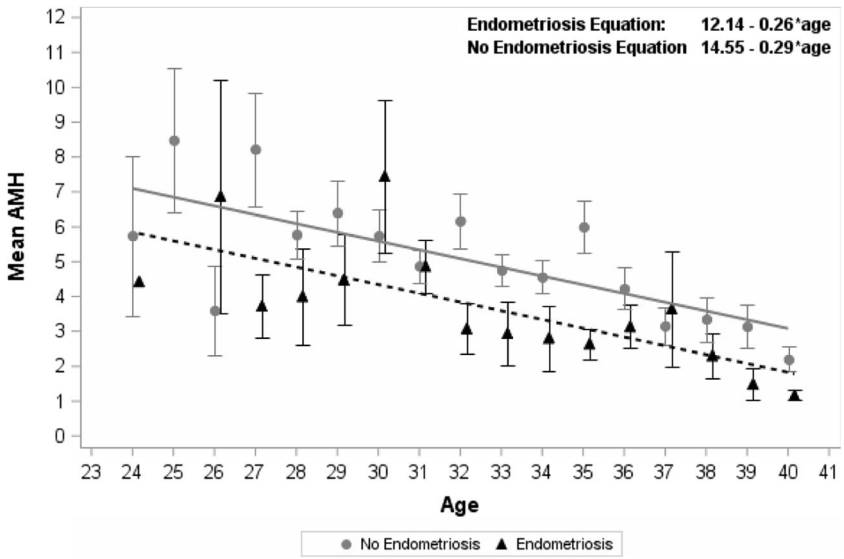

Fig. 1 Mean baseline antimüllerian hormone values by patient age. Mean baseline AMH values by year of patient age in all women with endometriosis (n = 138) and male factor infertility (n = 533). The dot represents the mean AMH level, whereas the error bars represent the standard deviation. The line represents the line of best fit

Table 3 Baseline antimüllerian hormone by stage of endometriosis

	Stage I ($n = 20$)	Stage II ($n = 16$)	Stage III ($n = 29$)	Stage IV ($n = 69$)	Male factor ($n = 533$)	Test for linear trend[†]
Mean baseline AMH* (ng/mL)	4.0 ± 4.0	3.1 ± 2.0	3.9 ± 3.2	3.2 ± 3.5	4.9 ± 4.3	0.58

AMH, antimüllerian hormone

All patients with a diagnosis of endometriosis were combined in this analysis

Data are mean ± standard deviation

*Baseline AMH values were collected between days 2 and 4 of the patient's menstrual cycle

[†] p value for linear trend includes endometriosis groups only

study that both groups of women with endometriosis had lower mean AMH levels regardless of age, compared to women with male factor infertility, as shown in Fig. 1.

Kasapoglu et al. observed that women with endometriomas had a lower AMH value at baseline and experienced a faster AMH decline over time compared to healthy women [10]. The results of our study agreed with these findings, in that women in both endometriosis groups had lower baseline AMH values compared to patients with male factor infertility. This could suggest an increased rate of decline in ovarian reserve over time in women with endometriosis, both with and without a history of prior ovarian surgery. This could result in a shorter reproductive window as well as an earlier onset of menopause in women with endometriosis, which is associated with long-term health consequences [25, 26].

When the patients were subdivided into their SART age groups, the relationship with AMH no longer reached the threshold of statistical significance in two groups (women with endometriosis and prior ovarian surgery age 35–37 [$n = 19$] and women with endometriosis without prior ovarian surgery age 38–40 [$n = 9$]). This is most likely explained by a lack of power to detect statistical differences in the smaller subgroups. However, this is an area that should be investigated in future studies with larger groups of patients.

The lack of a clear trend in AMH level with each increasing stage of endometriosis suggests that any ovarian damage that is secondary to endometriosis may be independent of the severity or invasiveness of the disease. This is analogous to the lack of correlation between pain severity and disease stage or lesion type in patients with endometriosis [27]. In addition, the similar ovarian reserve markers observed in both endometriosis groups suggests that iatrogenic surgical injury is not the only cause of ovarian reserve damage in patients with endometriosis. These findings support the hypothesis that the altered immune response and inflammatory cascade which occurs in endometriosis [21, 22] leads to follicular damage within the ovary and a lower ovarian reserve compared to healthy women, and may act independent of disease stage or surgical treatment.

The study strengths include the ability to compare two patient groups with endometriosis: one with any history of prior ovarian surgery and one without surgery, allowing for an observation of how ovarian markers are affected by endometriosis regardless of any previous iatrogenic damage. Women were also subdivided into their corresponding (SART) age group as an additional method to account for the strong correlation between patient age and ovarian reserve. Additionally, the exposure misclassification was minimized by including only women with a surgical diagnosis of endometriosis.

This study has several limitations. The laboratory transition between two different AMH assays during the study period could have led to calibration differences between the assays, but as mentioned, the two assays were shown to have good correlation and the Beckman AMH assay values were converted with the published regression equation to help correct for any differences in calibration [15]. Additionally, the study only included women with infertility which may represent a more severe or different phenotype than women with endometriosis without infertility. The statistical power to detect differences in AMH levels may be limited, particularly between small subgroups classified by different stages of endometriosis, which may represent important disease heterogeneity. The generalizability of these results to women with endometriosis and normal fertility may be limited and should not be applied to this population, as ovarian reserve markers have not been associated with fertility in non-infertile women [28].

In summary, this study demonstrates that infertile women with endometriosis, both with and without a history of prior ovarian surgery, have worse ovarian reserve markers (lower AMH, higher FSH) when compared to women with male factor infertility. Both endometriosis groups also had a higher proportion of women with decreased ovarian reserve at the time of presentation to the infertility clinic. These findings may suggest that there is a pathologic process related to endometriosis which alters ovarian reserve, independent of any surgical injury which may occur in women who undergo ovarian surgery. Future studies could further investigate the relationship between endometriosis and rate of ovarian reserve decline over time and whether the age of menopause onset is altered in both infertility and non-infertility populations.

Compliance with ethical standards

Conflict of interest Mark D. Hornstein is an author for UpToDate.com and is on the medical advisory board for WinFertility, Aetion, and AbbVie. The remaining authors declare that they have no conflicts of interest.

Human rights For this type of study formal consent is not required.

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