Oligo-anovulation is not a rarer feature in women with documented endometriosis

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Objective: To study the prevalence of oligo-anovulation in women suffering from endometriosis compared to that of women without endometriosis.
Design: A single-center, cross-sectional study.
Setting: University hospital-based research center.
Patient (s): We included 354 women with histologically proven endometriosis and 474 women in whom endometriosis was surgically ruled out between 2004 and 2016.
Intervention: None.
Main Outcome Measure(s): Frequency of oligo-anovulation in women with endometriosis as compared to that prevailing in the disease-free reference group.
Results: There was no difference in the rate of oligo-anovulation between women with endometriosis (15.0%) and the reference group (11.2%). Regarding the endometriosis phenotype, oligo-anovulation was reported in 12 (18.2%) superficial peritoneal endometriosis, 12 (10.6%) ovarian endometrioma, and 29 (16.6%) deep infiltrating endometriosis.
Conclusion(s): Endometriosis should not be discounted in women presenting with oligo-anovulation. (Fertil Steril® 2018;110:941–8. ©2018 by American Society for Reproductive Medicine.)
El resumen está disponible en Español al final del artículo.

Key Words: Endometriosis, oligo-anovulation, antimüllerian hormone, deep infiltrating endometriosis

Endometriosis is a chronic gynecologic disorder of unknown origin affecting up to 10% of women of reproductive age and 40% to 50% of infertile women (1, 2). While the etiology of endometriosis is unknown, progression of the disease is associated with the process of ovulation and ensuing menses (3). Indeed, exposure to menses, and associated retrograde or back-flow bleeding, is one of the factors commonly associated with increased risk of endometriosis (4–6). Conversely, all medical treatments of proven efficacy for endometriotic lesions and symptoms, including danazol, gonadotropin-releasing hormone agonist (GnRH-a), without and with add back therapy, oral contraceptive, as well as conditions such as pregnancy and breast feeding, share the common characteristic of blocking ovulation (7–11). Interestingly, two therapies with purportedly different modes of action, local effect on lesions for danazol and hypoestrogenic action for GnRH-a, showed remarkably equivalent efficacy on endometriotic lesions as assessed by laparoscopy (12). The surprisingly similar efficacy of danazol and GnRH-a led us to reconsider the presumed mode of action of these treatments. Recognizing that the local effect of danazol is associated with absent menses, merely interfering with ovulation may be sufficient to ameliorate symptomatic disease. Likewise, the fact that the effect of GnRH-a on endometriosis is solely mediated by its antiestrogenic properties is challenged by continued efficacy in the face of add-back therapy using combined estrogen-progestogen preparations (13). The observation that continuous

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oral contraceptive pills as well as pregnancy are as effective as GnRH-a suggests that the benefit of medical treatments of endometriosis stem from blocking ovulation rather than affecting estrogen levels (14, 15).

For these reasons, it is not unreasonable to assume that oligo-anovulation, as encountered in women suffering from polycystic ovary syndrome (PCOS), would lessen the likelihood of endometriosis. This, we worried, could constitute a bias when assessing ovarian reserve by antimüllerian hormone (AMH) measurement in endometriosis (16). Indeed, a lower incidence of oligo-anovulation, cases with characteristically high AMH levels, could lead to AMH levels being artificially decreased in endometriosis (17). To address this issue, we undertook to determine the incidence of oligo-anovulation in a population of women in whom endometriosis had been either, surgically confirmed or, excluded (18, 19). In addition, in both groups of patients we compared the prevalence of high serum AMH levels that had been measured prior to surgery, as previously reported (20).

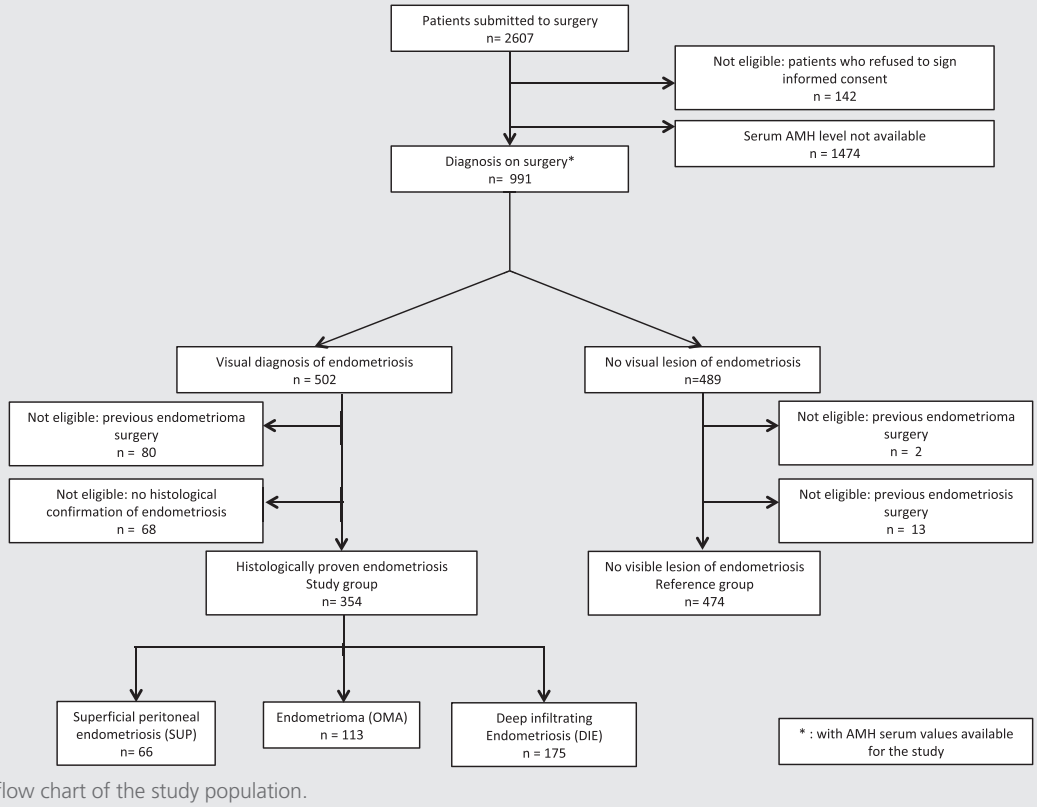
MATERIAL AND METHODS

Study Design and Population

We conducted an observational, cross sectional study using a prospectively constituted database described in details elsewhere (21). Clinical and biological information were collected in all non-pregnant patients <42 years of age who were surgically explored by operative laparoscopy or laparotomy for benign gynecological indications at our institution between January 2004 and January 2016. Women with cancer and/or who did not consent were excluded from this study. In case of multiple surgical interventions at our institution during the observation period, patients were included only once during. The inclusion flow chart is illustrated in Figure 1. Indications for surgery (possibly more than one per patient) included: pelvic pain defined as the presence, for at least six months, of dysmenorrhea, intermenstrual pelvic pain, and/or dyspareunia of moderate-to-severe intensity (22); infertility defined as at least 12 months of unprotected intercourse which did not result in pregnancy (23); pelvic mass (benign ovarian cysts and uterine myomas, etc.); and others (uterine bleeding, request for tubal ligation, and tubal diseases).

Patients were considered as presenting with endometriosis only when suspected lesions were histologically confirmed. Patients who were visually diagnosed with endometriosis but did not have histological confirmation or who had previous surgery for endometrioma were excluded from the study (21, 24). Conversely, patients who had surgery for endometriosis forms other than endometrioma were retained in the study. Patients with previous endometriosis surgery were excluded from the reference group group (25).

FIGURE 1



Longitudinal flow chart of the study population.

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During surgery, endometriosis was staged and scored (total, implant, and adhesion scores) according to the revised American Fertility Society (rAFS) classification (26). In addition, endometriotic lesions were sorted into three phenotypes based on histological findings: superficial peritoneal endometriosis (SUP), ovarian endometrioma (OMA), or deep infiltrating endometriosis (DIE) (27, 28). DIE was histologically defined by the endometriotic involvement of the muscularis of the target organ (bladder, bowel, ureter, etc.) (27, 28). Because different forms of endometriosis (SUP, OMA, and DIE) are frequently associated (29), patients were phenotyped according to their most severe, from least-to-most severe: SUP, OMA, and DIE (25, 27, 28). DIE patients were further graded according the severity of the DIE lesion as follows (least-to-most severe): uterosacral ligament(s), vagina, bladder, bowel, and ureter (30). Patients' most severe localization was retained for grading and used for further analysis. Only women with complete excision of their endometriotic lesions were retained for analysis because women with incomplete surgery did not have an exact histological phenotyping of their disease. For analysis purposes, patients were divided into two groups. The reference group included patients without visual lesions of endometriosis checked during the operative procedure with a thorough examination of the abdominal cavity. The study group included patients with histologically proven and graded endometriotic lesions with a SUP, OMA, or DIE phenotype.

Data Collection

The study used a prospectively managed database (21, 24). The personal history of each patient was collected during face-to-face interviews conducted by the surgeon during the month preceding surgery. We used a highly structured, previously published questionnaire (27, 28). Women enrolled in this study were not informed about the main hypothesis that the authors were investigating, and participants with and without endometriosis received identical questionnaires. The following data were recorded: age, parity, gravidity, height, weight, body mass index (BMI), any gynecological symptoms of pain (dysmenorrhea, deep dyspareunia, or non-cyclic chronic pelvic pain (31), and gastrointestinal (32) or lower urinary tract symptoms (27, 28). Pain intensity was evaluated preoperatively using a 10-cm visual analogue scale (33). Reproductive history including pregnancy outcomes was also obtained during the preoperative interview (19). Oligo-anovulation was defined as self-reported menstrual cycle length of $\geq$35 days or $<$10 menstrual period per year (34).

Serum AMH was measured as part of a previous report on AMH in endometriosis (20). This data set was used in the present study for assessing the incidence of high AMH levels in cases and the reference group. High AMH was defined as an AMH level greater than 4.9 ng/mL as according to previous published studies (35, 36).

Collection of Serum Samples

Serum samples were collected during the month preceding surgery. All AMH levels were determined in the same laboratory and were measured by a commercial enzyme-linked immuno-sorbent assay kit according to the manufacturer's instructions, as previously described (20).

Outcome Measures

The study aimed at determining the incidence of oligo-anovulation in endometriosis and the reference group. In addition, the incidence of high serum AMH levels, >4.9 ng/mL, was evaluated in both endometriosis and the reference group.

Within the endometriosis group, results were compared based on disease phenotype, as determined by histological findings of SUP, OMA, and/or DIE. The study also analyzed other serum AMH cut-off levels that are commonly quoted in literature as evidence of high AMH levels (>4.2 ng/mL, >4.9 ng/mL, and 5.6 ng/mL) between endometriosis-affected women and the reference group. We also compared these 3 different serum AMH cut-off values according to lesion staging (SUP, OMA, and DIE).

Statistical Analysis

All data were collected in a computerized database and analyzed by Statistical Package for the Social Sciences Software (SPSS software version 18.0.0, SPSS Inc.). When endometriosis and samples from the reference group were analyzed, we used Student's t-test for quantitative variables and Pearson's chi square or Fisher's exact test for qualitative variables, as appropriate. Considering the non-Gaussian distribution of serum AMH levels, a statistical analysis between the two groups was performed using the Mann–Whitney U test. When more than two groups were compared, we used the Kruskal– Wallis test. Data are presented as mean $\pm$ standard deviation (SD) or number (%) of subjects for continuous and categorical variables, respectively. $P<.05$ was considered statistically significant.

Ethics

The study was approved by the local institutional review board (approval number 05–2006 given by the 'Comité de Protection des Personnes et des Biens dans la Recherche Biomedicale' of Paris Cochin) and written informed consent was obtained from all participating subjects. AMH data in endometriosis and the reference group were presented in a previous study (20). In this prior publication AMH data were presented as mean values. Assessment of subgroups of women with high AMH levels, >4.9 ng/mL, was however not part of that publication and constitute new set of information presented here.

RESULTS

Patients and the Reference Group

From January 2004 to January 2016, 2,607 women underwent gynecological surgery at our institution. Of these, 2,465 (94.6%) gave their informed consent for participating in the study and 991/2465 (40.2%) had serum AMH level

available for study purposes, as illustrated in the study flow chart (Fig. 1).

Of these 991 women, 502 (50.7%) were visually diagnosed with endometriosis. Ultimately, 354 women had histological confirmation of endometriosis and no prior surgery for endometrioma (Fig. 1) and constituted the case population. Moreover, serum AMH was available in 489 other women in whom endometriosis had been visually excluded (reference group). After further exclusion of 2 women with past endometrioma surgery and 13 women, we retained 474 women as endometriosis-free constituting the reference group (Fig. 1).

Patients' distribution according to their worst endometriotic lesion was as follows: SUP (66 patients, 18.7%), OMA (113 patients, 31.9%), and DIE (175 patients, 49.4%), as described in Table 1. The distribution of patients according to the location of the worst DIE lesion was: 56 (32%) uterosacral ligament(s), 32 (18%) vagina, 21 (12%) bladder, 55 (31%) bowel, and 11 (6%) ureter. The distribution of patients according to rAFS stage was: 65 (18.4%) Stage I, 81 (22.9%) Stage II, 113 (31.9%) Stage III, and 95 (26.8%) Stage IV. The mean ± standard deviation total, implant and adhesion rAFS scores were 29.24 ± 27.67, 14.2 ± 11.52, and 15.17 ± 21.28, respectively.

Among 474 endometriosis-free women (reference group), the indications for surgery were: fibroids (181, 38.2%), non-endometriotic benign ovarian cyst (111, 23.4%), pelvic pain (67, 14.1%), tubal disorder (tubal diseases with proximal or distal blockage or damaged Fallopian tubes, including hydrosalpinges) (96, 20.3%), and other reasons (41, 8.6%) (Supplemental Table 1).

Patients' general characteristics are detailed in Table 2. There was a statistically significant difference in BMI between endometriosis women and the reference group with values of 21,79 ± 3.56 and 23.38 ± 4.19 ($P = .001$), respectively. Moreover, gravidity was lower in the endometriosis group as compared to the reference group (0.54 ± 0.93 vs. 0.9 ± 1.35; $P = .001$). Likewise, parity was significantly lower in the endometriosis, as compared to reference group (0.27 ± 0.63 vs. 0.44 ± 0.95; $P = .002$). Biological features of inflammation, such as mean C-reactive protein levels and mean white blood cell count were within reference ranges for both groups without between group differences.

TABLE 1

Histologic and surgical characteristics of endometriosis.

Variable	Endometriosis study group (n = 354)
Endometriosis type (n= 354)	
Superficial	66 ± 18.6
Endometrioma	113 ± 31.9
DIE	175 ± 49.5
With associated OMA	55 ± 31.4
Without associated OMA	120 ± 68.6
rAFS score (n= 354)[a]	
Mean total score rAFS	29.24 ± 27.67
Mean implants score rAFS	14.20 ± 11.52
Mean adherences score rAFS	15.17 ± 21.28
rAFS stage (n=354), n (%)	
I	65 (18.4)
II	81 (22.9)
III	113 (31.9)
IV	95 (26.8)
Obliteration of pouch of Douglas (n=354)	
None	215 ± 60.8
Partial	79 ± 22.3
Total	60 ± 16.9
Total DIE lesion (n= 175)	2.37 ± 1.845
Anatomical distribution of DIE (n=175)[b,c]	
USL, n (%)	128 (73.1)
Bilateral, n	40
Unilateral, n	88
Vagina, n (%)	75 (42.9)
Bladder, n (%)	34 (19)
Bowel, n (%)	65 (37.1)
Ureter, n (%)	11 (6.0)
Worst DIE lesion (n=175), n (%)[c]	
USL	56 (32)
Vagina	32 (18.2)
Bladder	21 (12)
Bowel	55 (31.4)
Ureter	11 (6.0)

Note: Data are presented as the mean ± standard deviation, unless specified otherwise. DIE = deeply infiltrating endometriosis; OMA = endometrioma; rAFS = revised American Fertility Society classification; USL = uretro-sacral ligaments.
[a] Score according to the American Fertility Society Classification (26).
[b] More than one per patient.
[c] According to a previously published surgical classification for DIE (30).

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Oligo-anovulation

Menstrual cycle data were available in all 991 women. Oligo-anovulation was reported by in 53 (15.0%) of endometriosis-affected women and 53 (11.2%) in the reference group ($P = .106$). In women with endometriosis, oligo-anovulation was reported in 12 (18.2%) SUP, 12 (10.6%) in OMA, and 29 (16.6%) in DIE ($P = .137$) (Table 3).

Serum AMH Levels

AMH levels were assessed to determine the incidence of high levels, whereas mean levels were reported in a prior publication (20). Taking an AMH cut-off value of 4.9 ng/mL there was no difference in the prevalence of high AMH values in endometriosis (n=104; 29.4%) and (n=149; 31.4%) reference group ($P = .525$). There was also no difference in high AMH (>4.9 ng/mL) incidence between the three surgical classification of endometriosis, with 21 (31.8%) cases in SUP, 36 (31.9%) in OMA, and 47 (26.9%) in DIE women ($P = .696$) (Table 3). Likewise, there were no differences observed according to rAFS stages (Supplemental Table 2).

Additional analyses also showed no significant differences in high serum AMH levels incidence when different cuts off values (4.2 ng/mL and 5.6 ng/mL) were used for comparing endometriosis or women of the reference group. No differences were shown in serum AMH levels >4.2 ng/mL or >5.6 ng/mL according to the surgical classification in SUP, OMA and DIE (Supplemental Table 3).

Clinical Association Between Elevated Serum AMH Levels and Oligo-anovulation

The incidence of high AMH levels was higher in oligo-ovulators as compared to regularly ovulating women in both cases (26/53 [49.1%] vs. 78/301 [25.9%]) and in the

TABLE 2

Demographics of endometriosie and reference group.

Characteristic	Endometriosis study group (n = 354)	Reference group (n = 474)	*P* value
Age (y)	31.49 ± 5.12	31.99 ± 6.02	.200[a]
Body mass index (kg/m^2)	21.79 ± 3.56	23.38 ± 4.19	.001[a]
Height (cm)	165.02 ± 6.42	165.31 ± 6.28	.506[a]
Weight (kg)	59.33 ± 10.17	63.92 ± 11.9	.001[a]
Gravity	0.54 ± 0.93	0.9 ± 1.35	.001[a]
Parity	0.27 ± 0.63	0.44 ± 0.95	.002[a]
Previous history of miscarriage, n (%)	39/115 (33.9)	57/208 (27.4)	.672[b]
Menstrual cycle, (n %)			
Always regular	266 (75.0)	351 (74.0)	.496[c]
Often regular	12 (3.4)	24 (5.0)	
Never regular	76 (21.6)	99 (21.0)	
Smoking habits, n (%)			
Never	203 (57.3)	294 (62.0)	.130[c]
Currently smoking	52 (14.7)	49 (10.3)	
Ever smoker	99 (28.0)	131 (27.6)	
Pain score[d,e]			
Dysmenorrhea	6.64 ± 2.72	4.10 ± 3.31	< .001[a]
Deep dyspareunia	4.10 ± 3.42	2.05 ± 3.02	< .001[a]
Non-cyclic chronic pelvic pain	2.92 ± 3.19	1.76 ± 2.79	< .001[a]
Gastrointestinal symptoms	3.23 ± 3.63	0.66 ± 1.81	< .001[a]
Lower urinary symptoms	1.13 ± 2.58	0.04 ± 0.46	< .001[a]
Rectal bleeding, n (%)	29 (8.2)	8 (1.7)	< .001[c]
Hematuria, n (%)	14 (4.0)	8 (1.7)	.044[c]
Previous surgery for endometriosis, n (%)[d]	85 (24.0)		
Infertility	124 (35,3)	163 (34,5)	.796[c]
Primary	94 (75.8)	86 (52.8)	< .001[c]
Secondary	30 (24.2)	77 (47.2)	
Laboratory findings			
CA 19.9 UI/mL	25.5 ± 62.98	26.40 ± 91.66	.902[a]
CA 125 UI/mL	41.83 ± 54.31	21.01 ± 23.70	< .001[a]
CRP mg/L	2.38 ± 4.75	2.43 ± 5.71	.920[a]
Hb g/dL	13.33 ± 0.97	13.11 ± 1.29	.013[a]
Lymphocyte count /mm^3	1901.7 ± 613.95	1884.35 ± 621.76	.722[a]
White blood cells count /mm^3	6705.99 ± 1945.88	6257.15 ± 1870.51	.003[a]
Serum ferritin μg/L	48.33 ± 35.08	48.07 ± 41.37	.937[a]

Note: Data presented as mean ± standard deviation, unless specified otherwise.
[X]Visual analog scale.
[a] Student's test.
[b] Pearson's Chi square.
[c] Pearson's χ^2 test.
[d] Previous surgery of endometriosis (excluding endometrioma surgery).
[X] More than one per patient.

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reference group (31/53 [58.5%] vs. 118/421 [28.0%]), respectively. The combination of elevated AMH levels, >4.9 ng/mL, and oligo-anovulation was found in 26 (7.3%) women with endometriosis and 31 (6.5%) women in the reference group (*P*=.651). Further classifying by endometriosis type, the clinical association of oligo-anovulation and high AMH was found in 7 (10.6%) SUP, 6 (5.3%) OMA, and 17 (7.4%) (*P*=.566) (Table 3).

DISCUSSION

Our study found that prevalence of oligo-anovulation in women with histologically documented endometriosis (15%) is like that observed in the reference group without endometriosis (11.2%) (*P*=.106). The rate of oligo-anovulation is independent of the surgical staging of endometriotic lesions, SUP (18.2%), OMA (10.6%), or DIE (16.6%) (*P*=.137). Hence, our study refutes the intuitive belief that oligo-anovulation might provide some protection against endometriosis. We also observed an equal incidence of elevated AMH levels (>4.9 ng/mL) in endometriosis (104/354, 29.4%) and disease-free women (149/474, 31.4%) (*P*=.525). The similarity of serum AMH in the 2 groups is consistent with equivalent rates of oligo-anovulation.

The strength of this study lies in its methodological design. Indeed, our study: relies on surgery for either, diagnosing or, excluding endometriosis and in the former case; histology for staging endometriotic lesions as SUP, OMA, or DIE; using clinical data prospectively collected by questionnaires filled out prior to surgery on various epidemiological variables including menstrual cycle patterns; and banking on a large cohort of surgically diagnosed and histologically staged cases of endometriosis. Moreover, a further strength of this study lies in the fact that AMH levels were determined prior to surgery and processed in a single reference laboratory.

TABLE 3

Statistical analysis for oligo-anovulation and serum antimüllerian hormone levels in women with endometriosis and reference group.

Variable	Endometriosis (N = 354)			Reference group (N = 474)	*P* value
AMH > 4.9 ng/mL	104 (29.4)			149 (31.4)	.525[a]
Oligo-anovulation	53 (15.0)			53 (11.2)	.106[a]
AMH > 4.9 and oligo-anovulation	26 (7.3)			31 (6.5)	.651[a]
	DIE (N = 175)	**OMA (N = 113)**	**SUP (N = 66)**	**Reference group, ng/mL (N = 474)**	
AMH levels and menstrual cycles according to the surgical classification					
AMH >4.9	47 (26.9)	36 (31.9)	21 (31.8)	149 (31.4)	.696[b]
Oligo-anovulation	29 (16.6)	12 (10.6)	12 (18.2)	53 (11.2)	.137[b]
AMH > 4.9 and oligo-anovulation	17 (7.4)	6 (5.3)	7 (10.6)	31 (6.5)	.566[b]

Note: Results are expressed as n (%), unless specified otherwise. AMH = antimüllerian hormone; DIE = deeply infiltrating endometriosis; OMA = endometrioma; SUP = superficial peritoneal endometriosis.

[a] Statistical analysis was performed with Pearson's χ^2 test

[b] Statistical analysis was performed with Fisher exact test

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Our study has several limitations however. First, we studied the incidence of a clinical feature associated with PCOS, oligo-anovulation, but not the true incidence of PCOS itself. Indeed, assessing PCOS incidence was hindered by the fact that our database did not record clinical and/or biological evidence of androgen excess. Of note, endometriosis patients had a lower BMI than the reference group, a finding reported in numerous endometriosis studies (19).

The incidence of high AMH levels was similar in cases and the reference group. As described in the result section, the use of other cut off values (other than 4.9 ng/mL) gave similar results, inferring similar incidences of high AMH levels in endometriosis women and disease-free individuals.

Our results are in line with previous published studies. In a study published more than 40 years ago, Soules et al. (37), reported that the incidence of anovulation was unaltered in women suffering from endometriosis. In a series of 350 surgically explored endometriosis affected women (77% of whom confirmed by histologic examination of excised tissue) these authors found that 58/350 (17%) exhibited anovulation or oligo-ovulation patterns (37). Among endometriosis women with oligo-anovulation the distribution according to disease severity was as follows: 23 (39%) had mild endometriosis, 34 (59%) moderate, and 1 (2%) severe endometriosis. These authors concluded that endometriosis and anovulation can coexist contrary to pre-existing concepts about these diseases. That Soules et al. (37) have reported findings like ours supports the veracity of our own observation. Taken together our results and those of Soules et al. (37) indicate that any decrease in AMH levels observed in women suffering from and/or operated for endometriosis is real and not affected by recruitment biases that would lead to a lower incidence of high AMH values encountered in oligo-anovulation. In a previous prospective cross-sectional study on unselected 20-40 year-old women, the prevalence of oligo-anovulation and that of AMH >4.9 ng/mL reached 13.1% and 8.3 %, respectively in women <30 years of age (38). Furthermore, the worldwide prevalence of PCOS ranges from 4% to 21% (39), with differences according population selection and ethnical origins (40, 41). Our results disclaim therefore the possibility that lower AMH levels reported in endometriosis (42–45) could result from a lower incidence of oligo-anovulation and related high AMH in women with endometriosis.

CONCLUSION

Our study depicts similar incidence of oligo-anovulation and high AMH levels (>4.9 ng/mL) in the endometriosis and reference group. This observation therefore contradicts the often-heard assumption that oligo-anovulation, a seminal feature of PCOS, and endometriosis are rarely associated. By extension, an added implication of our study is that serum AMH measurements in endometriosis patients may rightly reflect the ovarian reserve status without fears of bias due to differences in oligo-anovulation and/or high AMH incidence. Further studies are needed to truly understand the relationships between ovarian dysfunction such as notably, PCOS and endometriosis. We amended our clinical data collection so that it includes information on hyperandrogenism and antral follicle count, to answer questions on the incidence of PCOS in endometriosis women in the future.

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La oligoanovulación no es una característica inusual en mujeres con endometriosis documentada

Objetivo: Estudiar la prevalencia de oligoanovulación en mujeres con endometriosis en comparación con mujeres sin endometriosis.

Diseño: Estudio transversal en un único centro.

Entorno: Centro de investigación en hospital universitario.

Paciente(s): Se incluyeron 354 mujeres con endometriosis histológicamente demostrada, y 474 mujeres en las cuales la endometriosis se descartó quirúrgicamente entre 2004 y 2016.

Intervención: Ninguna.

Principales medidas de los resultados: Frecuencia de oligoanovulación en mujeres con endometriosis en comparación con la frecuencia predominante en el grupo libre de enfermedad.

Resultados: No hubo diferencia en la tasa de oligoanovulación entre mujeres con endometriosis (15,0%) y el grupo de referencia (11,2%). En cuanto al fenotipo de endometriosis, se informó oligoanovulación en 12 (18,2%) endometriosis peritoneal superficial, 12 (10,6%) endometriomas ováricos, y 29 (16,6%) endometriosis infiltrante profunda.

Conclusión(es): La endometriosis no debe descartarse en mujeres que presentan oligoanovulación.