Review

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Serum Anti-Müllerian Hormone Levels and Endometriosis Surgery: Unraveling the Evidence

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Abstract: Assessment of ovarian reserve is important in patients with fertility intent. Anti-Müllerian Hormone (AMH) serum levels are a useful ovarian reserve marker. Endometriosis is a benign disease with three phenotypes: superficial peritoneal endometriosis (SUP), ovarian endometrioma (OMA) and Deep Endometriosis (DE). Endometriosis is linked with infertility, however, the exact impact of endometriosis per se and endometriosis surgery on AMH levels is less clear. Evidence suggests that OMA per se is linked to reduced AMH values and higher AMH decline rate over time. OMA cystectomy causes further reduction in AMH which, however, tends to recover post-operatively. Non-excisional surgery for OMA spares the ovarian parenchyma, however, an, at least temporary, decline in AMH has been observed. The effect is likely smaller than cystectomy. Non-thermal methods of hemostasis following cystectomy are likely superior in terms of AMH. AMH levels before OMA surgery appear positively correlated with the post-operative probability of pregnancy, particularly spontaneous conception, but not livebirth rates. Pre-operative AMH levels are, also, predictive of the risk of diminished ovarian reserve (DOR). Similarly, post-operative AMH levels and the rate of AMH decline at 1 year after OMA surgery appear predictive of fertility outcomes. SUP likely has little (if any) impact on AMH levels. DE per se reduces AMH levels and a further reduction following surgery is anticipated. However, a reduction in AMH values should not be interpreted as a decline in the patient's reproductive potential. Further research should focus on extra-ovarian locations of endometriosis and their impact on AMH values.

Keywords: endometriosis; endometrioma; deep endometriosis; superficial endometriosis; ovarian reserve; anti-müllerian hormone; AMH; infertility; reproductive surgery

1. Introduction

Endometriosis is a common, benign gynaecological disease of unknown aetiology, affecting 1 in 10 women of reproductive age [1]. It is characterised by the presence of ectopic, endometrium-like tissue leading to an estrogen-dependent, chronic inflammatory process and is commonly linked with pelvic pain and/or infertility [2]. Fecundity rates in couples of reproductive age with no documented infertility are estimated between 15% to 20%, whereas in those with untreated endometriosis, rates

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vary from 2% to 10% [3]. Three phenotypes of endometriosis are commonly recognised: ovarian endometrioma (OMA), superficial peritoneal endometriosis (SUP) and deep endometriosis (DE) [1].

Anti-mullerian hormone (AMH) is produced by the granulosa cells of the ovary, playing an important, regulatory role in ovarian folliculogenesis, while preventing a premature exhaustion of the ovarian reserve [4]. AMH levels in the blood serum are believed to accurately reflect the ovarian reserve [5], with no significant variations during the menstrual cycle [6], making it a useful marker in daily clinical practice. AMH levels are useful in predicting the response to controlled ovarian stimulation (COS) protocols as part of Artificial Reproductive Technology (ART) techniques [7], as the number of retrieved oocytes may depend on the AMH value [8]. Furthermore, AMH levels progressively decrease with advancing patient age [9], and age-specific AMH values may be useful in predicting age at natural menopause [10]. However, its usefulness in predicting future fecundity and chances of natural conception is limited, based on existing evidence [11–13]. However, these studies excluded women with endometriosis [11–13].

While there is a well-known link between endometriosis and infertility, there is conflicting evidence regarding the potential impact of various endometriosis phenotypes on serum AMH levels [14–18]. Regarding the effect of endometriosis surgery on AMH values, the majority of published literature focuses on patients with OMA, raising concerns regarding a potentially harmful effect of surgery on the ovarian reserve [19–21]. Furthermore, identifying the precise clinical significance of serum AMH levels, pre- and post-surgery, in terms of predicting the probability of pregnancy would be most useful in endometriosis patients with pregnancy intention.

In this narrative review, we critically present the available evidence regarding the impact of different endometriosis phenotypes per se, as well as the effect of endometriosis surgery, on AMH levels. We, also, investigate if AMH levels pre- and post- endometriosis surgery may be useful in predicting the reproductive outcomes of endometriosis patients with pregnancy intention.

2. Materials and Methods

We performed a systematic search for relevant articles in the following databases: Pubmed, Embase, Scopus, Web of Science and Cochrane Library. We used combinations of the following MESH terms and keywords: ''endometriosis'', ''endometrioma'', ''ovarian endometrioma'', ''ovarian endometriosis'', ''superficial endometriosis'', ''deep endometriosis'', ''deep infiltrating endometriosis'', ''bowel endometriosis'', ''colorectal endometriosis'', ''bladder endometriosis'', ''anti-mullerian hormone (AMH)'', ''ovarian reserve''. We included English-language articles only, published between years 2000 and 2025. We included both original research as well as review articles. Conference abstracts were excluded. The search was performed independently by two authors (G.G., A.P.) who reviewed the abstracts and relevant full texts. A third author (I.B.) was allocated to resolve any disagreements on article selection. No disagreements were noted.

3. Results

3.1. The Impact of Endometriosis Per Se on AMH Levels

It has been well demonstrated that all phenotypes of endometriosis have a negative impact on fertility [22], through a variety of pathophysiological mechanisms, such as altered peritoneal environment, distorted pelvic anatomy, altered endometrial receptivity, impaired implantation, poor oocyte quality, abnormal utero-tubal transport, endocrine abnormalities, cell signalling and epigenetic changes [23].

Recently published data from the Nurses' Health Study II (NHS II) identified women with laparoscopically-confirmed endometriosis (n=119) to have 29.6% lower AMH levels (95 % CI: - 45.4, - 9.2) compared to those without endometriosis (n=1842), a finding that did not vary based on parity or infertility history [24]. However, the mechanisms responsible for reduced AMH levels in patients with endometriosis, particularly those without OMA, are not entirely clear: Chronic inflammation and oxidative stress due to endometriosis may exert a negative effect on ovarian reserve [17].

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Endometriosis patients exhibit an increase in apoptosis of the cumulus cells surrounding the oocyte, in particular granulosa cells [25,26], with the apoptosis likely being proportional to disease severity [27]. Furthermore, a ''burn-out'' effect on the follicular pool, through continuous activation and depletion of follicles as seen in cases of chemotherapy-induced gonadotoxicity, may also be present in cases of endometriosis [28], possibly explaining why a diagnosis of endometriosis has been linked with an earlier age of menopause [29,30]. Carrarelli et al. suggested that AMH itself may, actually, play a role in the pathogenesis and growth of endometriosis, as they identified an increased AMH and AMHRII mRNA and protein expression in the endometrium of patients suffering from endometriosis compared to controls, as well as in endometriotic lesions, although no significant difference in serum AMH levels was noted between the two groups [31]. Furthermore, the peritoneal fluid of advanced-stage endometriosis patients exhibits reduced AMH levels compared to controls [32].

With respect to SUP (or minimal/mild endometriosis-MME), its negative effect on fertility outcomes is well established [33]. However, the potential impact of SUP on ovarian reserve is, to date, less clear: In Lessan's recent case-control study, AMH levels in patients with histologically proven SUP and no co-existent OMA or DE did not differ significantly from controls [3.0 ± 2.8 ng/mL and 2.8 ± 1.9 ng/mL, respectively (P = 0.71)] [14]. However, controls did not have SUP ruled out via laparoscopy and, as such, it is possible that some of them might, actually, have had SUP. Similarly, a prospective cohort study did not identify a significant difference in baseline AMH levels between those with SUP and endometriosis-free controls (p=0.19) [21]. A case-control study by Shebl et al. identified no significant difference in serum AMH levels between MME patients and those undergoing IVF/ICSCI due to male-factor [18]. Follicular-fluid AMH levels did not differ significantly between those with MME undergoing natural IVF and non-endometriotic controls [34]. In contrast to these finding, Lemos et al. identified that patients with infertility and laparoscopically-proven MME have lower AMH values (1.26 +/- 0.7 ng/mL) compared to infertile patients due to tubal occlusion (2.02 +/- 0.72 ng/mL, p=0.004) [35]. We need to bear in mind, though, the small sample size (n=34), the inclusion of infertile patients only and the fact that the results were not adjusted to parity or hormonal treatment. Lemos et al.'s findings are supported by a recently published study in a rat model where surgically-induced peritoneal endometriosis led to a decline in measured AMH levels [36].

Regarding the presence of OMA per se, there is overwhelming evidence suggesting that it is associated with a reduction in AMH levels compared to controls [15,19–21]: Muzii et al. 's systematic review and meta-analysis of 17 studies found that patients with OMA had lower AMH compared to those with other types of benign ovarian cysts (mean difference= -0.85, 95% CI: -1.37 to -0.32), as well as those with healthy ovaries (mean difference= -0.61, 95% CI: -0.99 to -0.24) [15]. An earlier prospective study had found patients with OMA to have lower AMH compared with controls at baseline (4.2+2.3 versus 2.8+2.2 ng/ml, respectively, P= 0.02) [20]. Another prospective study confirmed the above finding (baseline AMH values in the OMA group= 1.8ng/mL, 95% CI: 1.2 – 2.4ng/mL vs. negative laparoscopy group= 3.2ng/mL, 95% CI: 2.0 – 4.4ng/mL; p < 0.02) [21]. Furthermore, patients with OMA tend to have a more rapid decline in AMH values over time, compared to controls [37]. Bilaterality of OMA has, also, been demonstrated to exhibit a negative effect on AMH values, compared to unilateral OMA [38,39].

Regarding DE, the existing evidence suggests a detrimental effect on AMH levels [16–18]. Pacchiarotti et al. demonstrated that patients with stage III/IV endometriosis had significantly lower AMH levels (0.97 +/- 0.59 ng/ml), compared to presumed endometriosis-free, age-matched controls (1.72 +/- 0.63 ng/ml, p=0.001) [17]: however, the authors did not clarify what percentage of DE patients had co-existent OMA, while controls did not have a diagnosis of endometriosis ruled-out by laparoscopy. A case-control study reported that patients with stage III/IV endometriosis had significantly lower AMH levels (2.38 +/- 1.83 ng/ml) compared to controls undergoing IVF/ICSI due to male factor (3.58 +/- 2.46 ng/ml, p<0.0001), whereas, there was no statistically significant difference between those with stage I/II disease and controls [18]. The same study identified a significant

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difference when comparing AMH levels between patients with stage I/II and stage III/IV endometriosis (stage I/II: 3.28 +/- 1.93 ng/ml vs. stage III/IV: 2.38 +/- 1.83 ng/ml, p< 0.01) [18]. While controls were matched for age, frequency of infertility and Body Mass Index (BMI), the authors did not clarify the percentage of stage III/IV patients with OMA. These findings may be viewed as anticipated, since superior post-operative fertility has been demonstrated in milder (stage I-II) cases compared to more advanced (stage III-IV) endometriosis [40]. In Kim et al.'s retrospective, case-control study comparing AMH levels between patients with OMA and age-matched controls without ovarian cyst, multiples of the median for AMH (AMH-MoM) levels were negatively correlated with the estimated endometriosis score (r^2=0.13, p<0.01) [41]: in particular, patients with rASRM stage IV endometriosis and OMA had significantly lower AMH and AMH-MoM compared to controls (2.1±0.3 vs. 3.1±0.4 ng/mL, p=0.02 and 1.1±0.1 vs. 1.7±0.2, p=0.03, respectively), but there was no statistically significant difference in terms of AMH and AMH-MoM when patients with stage III endometriosis and OMA were compared to controls (3.7±0.5 vs. 3.4±0.5 ng/mL and 1.6±0.2 vs. 1.5±0.2, respectively) [41]. Streuli' s case-control study did not identify a significant difference in AMH levels between cases (various endometriosis phenotypes and disease stages) and controls (other, benign gynaecological pathologies) [cases: 3.6 +/-3.1 ng/ml, controls: 4.1+/-3.4 ng/ml, mean difference: 0.45 (95% CI -0.02 to 0.96) ng/ml, p=0.06], with the only exception being patients with previously operated OMA [42].

3.2. The Impact of Endometriosis Surgery on AMH Levels

The majority of published evidence focuses on patients operated for OMA. The optimal method of surgical management of OMA is still debated, with different approaches including ovarian cystectomy, ablative techniques (laser, plasma energy or bipolar diathermy), ethanol sclerotherapy and combined approaches [19]. Cystectomy has been linked to reduced risk of OMA recurrence and endometriosis-associated pain [43], as well as increased chances of spontaneous conception [44]. However, it may reduce ovarian volume [45], while it presents an estimated risk of ovarian failure of 2.4% [46]. These concerns have led to the development of the aforementioned, non-excisional alternatives.

A detrimental effect of OMA cystectomy on AMH levels has been demonstrated [47,48], with various studies suggesting that the decline is greater when compared to cystectomy for other benign cysts [49–51]. The effect is more profound in case of bilateral OMAs [52,53]: A systematic review and meta-analysis reported an AMH drop by 39.5% and 57.0%, for unilateral and bilateral OMA cystectomy respectively, compared to baseline [52]. The decline may be viewed as a result of loss of healthy follicles, although a direct association between the number of follicles inadvertently removed and the extent of AMH reduction has not been proven [54]. Surgery for bilateral OMAs has been linked with an earlier age of menopause and an increased risk of premature ovarian failure (POF) [55].

Other factors that affect post-cystectomy AMH reduction include pre-operative AMH value [20,56–59], advancing patient age [57], duration of surgery [54], and cyst size [56,60], although a recent meta-analysis of seven prospective studies did not identify significant differences on AMH values after surgery, depending on cyst size [61]. With regards to endometriosis severity, a prospective study found that, although there was no significant correlation between pre-operative AMH levels and rASRM score (r=-0.219, p=0.187), a positive correlation between rASRM score and rate of AMH decline post-OMA cystectomy was noted (r= 0.473, P= 0.00273), with patients with more advanced disease experiencing a higher loss of ovarian reserve [62].

Various systematic reviews and meta-analyses have reported that the use of diathermy for haemostasis following cystectomy has a deleterious effect on AMH values [63–65]: Ata's meta-analysis of 4 studies (213 patients) found sutures or haemostatic sealants to cause a 6.95% less reduction in AMH at 3 months after cystectomy, compared with bipolar diathermy [63]. Another meta-analysis of 3 studies, despite significant heterogeneity, identified bipolar electrocoagulation to cause a significantly higher AMH decline at 3 months after surgery, compared to non-thermal haemostasis methods (mean difference: −0.79 ng/mL, 95% confidence interval: −1.19 to −0.39) [64].

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The detrimental effect of bipolar electrocoagulation was maintained at 12-months after surgery, according to a meta-analysis by Ding et al. (weighted mean difference: −1.01 ng/ml; 95% confidence interval: −1.85 to −0.17) [65]. This effect may be unique to OMA cystectomy and not apply to cystectomy for other benign cysts [66]. In contrast to the above, a recent randomized controlled trial (RCT) found no significant differences in AMH values at 6 months after cystectomy for unilateral OMA between bipolar coagulation, haemostatic sealants and sutures [67]. Similarly, an earlier RCT had found no significant difference in AMH values, at 3-months after OMA cystectomy, between bipolar coagulation and use of haemostatic sealant, although the latter led to higher antral follicular count (AFC) value post-operatively [68].

Cystectomy for recurrent OMA is a technically challenging procedure. A case-control study of 36 patients linked cystectomy in this scenario with a significant reduction in AMH (AMH before second surgery: 2.7 +/-1.9 ng/ml, versus AMH after second surgery: 1.2 +/- 1.2 ng/ml, p<.001), although AMH values did not differ significantly between unoperated cases (AMH: 2.7 +/-1.9 ng/ml) and controls (AMH: 3.1 +/-1.9 ng/ml, p=0.59) [69]. Therefore, the authors concluded that recurrent OMA per se does not cause a reduction in AMH values unless operated [69]. In this setting, ethanol sclerotherapy may be of use, as a retrospective study showed no difference in AMH decline rates between primary and recurrent OMA managed by this technique [70].

Despite concerns with surgery-induced decrease in ovarian reserve, various studies have suggested a progressive recovery in AMH values post-operatively [71–75]: In a prospective study of 39 patients, 20 of them had higher AMH levels at 1 year compared to 1 month after OMA cystectomy [71]. Of note, the follicular loss during surgery was higher (p = 0.035) for this group, suggesting that mechanisms other than follicular loss may be involved in cases of sustained reduction in AMH post-surgery [71]. Another prospective study with 171 patients showed that, at 12 months after cystectomy, AMH levels did not differ significantly from the preoperative values in OMAs </= 7 centimetres , unilateral cysts, and stage III endometriosis [72]. Two further prospective studies have reported non-significant differences in AMH values at 12 months post-cystectomy, compared to baseline [74,75]. This recovery appears less likely in case of bilateral OMAs [76]. Recovery is likely a result of inflammation-mediated injury triggered by the surgical procedure which is, then, followed by the recruitment and growth of primordial follicles and granulosa cells activation, resulting in a re-organisation of the follicular cohort, including follicles transitioning from ''silent'' to ''active'' state, thus increasing the production of AMH [72]. The estimated 180-day duration of folliculogenesis may explain the delay in AMH recovery observed after surgery [76].

Non-excisional surgical techniques for OMA spare the ovarian parenchyma, therefore, one would anticipate a minimal effect on AMH values. However, published evidence suggests a temporary, yet significant, detrimental impact on AMH values: a recent prospective study evaluating Dual Wavelength Laser System (DWLS) diode laser use for endometrioma ablation reported a significant reduction in AMH levels at 3 months compared to baseline (p=.034). However, at 6 and 12-months, values were not significantly lower to baseline [77]. Similarly, an earlier prospective study reported a significant decrease in AMH levels at 3 months after plasma energy ablation of OMA, followed by an increase at >6 months. No significant difference was noted between pre-operative and post-operative levels at the end of follow-up [78]. Laparoscopic 95% ethanol sclerotherapy of OMA also led to a significant decrease in AMH values [79].

Various comparative studies have found cystectomy to cause a greater depression in AMH values compared to non-excisional approaches to OMA [80–82]. Saito et al. reported that bilateral cystectomy for endometriomas led to a significantly higher drop in AMH up to 12 months after surgery, compared to bipolar coagulation (p=0.02) [83]. However, no significant difference between the two approaches was seen in case of unilateral OMA, although AMH values were significantly reduced in both approaches, when compared with pre-operative levels [83]. In case of unilateral OMA <5 centimetres in diameter, AMH values at 3 months were comparable between cystectomy and bipolar coagulation, however, for OMA > 5 centimetres, AMH decline was significantly higher in the cystectomy group [84]. A multi-centre RCT found cystectomy to cause a significant decrease in

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AMH levels at 3 months (from 2.6 +/- 1.4 to 1.8 +/- 0.8 ng/mL; 95% CI: -1.3 to -0.2; p = 0.012), whereas, the decline caused by CO2 laser, compared to baseline, was not significant (from 2.3 +/- 1.1 to 1.9 +/- 0.9 ng/mL; 95% CI: -1 to -0.2; p = 0.09) [81]. A small RCT compared OMA cystectomy (Group 1, n=10) with the ''three-step procedure'' (Group 2, n=10) (laparoscopic drainage of the OMA followed by 3 months of gonadotrophin-realising hormone agonists and, then, ablation of the cyst wall) [82]: the authors observed a significant decline in Group 1 AMH (from 3.9 +/- 0.4 to 2.9 +/- 0.2 ng/ml, p=0.026), whereas, no significant change was noted in Group 2 (from 4.5 +/- 0.4 to 3.99 +/- 0.6 ng/mL, p > 0.05) [82]. A recent meta-analysis, however, found no significant difference between cystectomy and ablative OMA approaches in terms of AMH decline [85].

The phase of the menstrual cycle at which OMA cystectomy takes place influences the degree of AMH decline inflicted by surgery, according to a recent RCT [86]: Serum AMH at postoperative six months in group ''late luteal phase'' (''LLP'') was significantly higher than that in group ''early follicular phase'' (''EFP'') (3.35 ± 1.67 vs. 2.61 ± 1.15, $p = 0.018$) [86]. Furhtermore, AMH decrease values at postoperative six months were significantly higher in group ''EFP'' than that in group ''LLP'' (1.54 ± 0.93 vs. 1.91 ± 1.06; $P < 0.001$) [86]. However, the above findings were not confirmed by a prospective cohort study [87]: AMH decline rate at 6 months post-cystectomy did not differ based on the phase of the menstrual cycle that cystectomy was performed (follicular phase: 24.5% versus luteal phase: 19.5%, $p > 0.05$) [87].

Robotic surgery (or robot-assisted laparoscopy) has, recently, been gaining popularity in the surgical management of endometriosis [88]. However, comparative data focusing on whether it conveys any benefit over conventional laparoscopy, in terms of AMH values, are scarce: A recently-published, retrospective study found no significant differences in AMH values between robotic and laparoscopic OMA cystectomy [89]. Another retrospective study comparing single-port, robot-assisted laparoscopy versus single-port laparoscopy found the former approach linked to higher AMH values post-procedure, however, this was only the case for cases of stage I/II endometriosis [90].

Evidence on the impact of surgery for extra-ovarian localisations of endometriosis on the AMH values is limited: Regarding SUP, AMH levels among endometriosis-free controls and those that had superficial disease excised laparoscopically did not differ at 1-month (p=0.16) or 6-month (p=0.59) follow-up after excision, suggesting that surgery for this endometriosis phenotype may not have a deleterious effect on ovarian reserve [21]. The higher AMH decline following OMA cystectomy, when this co-exists with DE [53], may be attributed to a reduction in ovarian blood flow as a result of extensive adhesiolysis required at the time of surgery. A recent cross-sectional study reported that, when OMA co-exists with DE, surgery yields a higher drop in AMH compared to OMA alone or DE alone [91].

3.3. AMH Levels as Predictor of Fertility Outcomes After Endometriosis Surgery

The latest European Society of Human Reproduction and Embryology (ESHRE) guideline on endometriosis suggests that the patient ‘s ovarian reserve be taken into account when considering surgery to enhance chances of natural conception [43]. The precise clinical significance of AMH levels, in terms of predicting the probability of pregnancy after endometriosis surgery, is an interesting topic that, however, needs to be further investigated. Stochino-Loi et al. use an AMH cut-off value of 2 ng/mL, with patients having levels >/= 2 ng/ml considered as ''normal AMH'' levels and those with values < 2 ng/ml as ''low AMH'' levels [92]: they further classify those with levels < 1 ng/ml in the ''very low AMH'' group. Similarly, in a prospective cohort study of patients planned to undergo ovarian cystectomy for OMA, patients were grouped in a ''high AMH'' group (AMH> 2 ng/mL) and a ''low AMH'' group (AMH </= 2 ng/mL) [93]. The AMH threshold value of 2 ng/ml had previously been suggested by Reichman et al. [94], however, this was based on cancellation rates of IVF cycles and was not restricted to endometriosis patients.

Concrete evidence on an optimal pre-operative AMH value to predict post-operative fecundity is currently lacking. Zhou et al.’ s prospective study included 103 patients with OMA that underwent

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laparoscopic cystectomy [93]: They observed that the cumulative pregnancy rate (CPR) during the 2-year follow-up after surgery was significantly higher in the ''high AMH'' group (AMH> 2 ng/mL, n=61) compared to the ''low AMH'' group (AMH </=2 ng/mL, n=42) (p<0.001), suggesting that high pre-operative AMH is a strong predictor of spontaneous conception after OMA cystectomy. Specifically, the likelihood of conception during the 12-24 months postoperatively was 50,82% and 69,44% in the ''high AMH'' group, while the CPR was 28,57% and 33,61% in the ''low AMH'' group. They identified the optimal pre-operative AMH value to be 3.545 ng/mL. AMH values dropped significantly in both groups after surgery but the decline was significantly higher in the low AMH group (p<0.001). Another observational study proposed a very similar optimal AMH value of 3.68 ng/mL before surgery to be linked with increased spontaneous conception rates after OMA cystectomy [95].

Surgical management of OMA is not routinely recommended before IVF, as no clear benefit has been demonstrated and surgery may reduce the ovarian reserve [43]. However, if infertility treatments are eventually required after laparoscopic OMA cystectomy, AMH levels at 1 year after surgery are higher in pregnant patients, compared to their non-pregnant counterparts, despite no significant differences in values before, or at 1-month after surgery, between the two groups [96]. Furthermore, not only the AMH value but, also, the rate of AMH decline after surgery is of importance, as those that conceived spontaneously had a lower rate of AMH decline at 1 year postoperatively, compared to those that required infertility treatments [51]. A progressively declining AMH value after cystectomy may, therefore, be viewed as an indication for earlier referral to IVF, in light of these findings.

Pre-operative AMH levels may be used to predict the risk of diminished ovarian reserve (DOR) (AMH< 1.1 ng/ml) after laparoscopic OMA cystectomy, according to the Bologna criteria by the European Society of Human Reproduction and Embryology [ESHRE] [97]: For unilateral cystectomy, a pre-operative AMH cut-off value of 2.1 ng/ml was predictive of DOR, whereas for bilateral cystectomy, the cut-off value was 3.0 ng/ml [98]. Bilaterality of OMA was predictive of post-operative DOR. This finding is of clinical importance in the ART setting, as patients with post-operative DOR may behave as poor ovarian responders and be linked to lower clinical and livebirth rates following IVF, compared to idiopathic poor responders [99]. Furthermore, the authors observed that the cumulative spontaneous pregnancy rate of the DOR group was significantly lower than that of the non-DOR group (14.3 % vs. 59.2 % respectively, p = 0.04) [98].

Surgical management of DE has been demonstrated to enhance fecundity in appropriately selected cases [100,101]. In Arfi et al.'s retrospective study of 118 patients operated for DE without bowel involvement and with pregnancy intention, an AMH level > 2ng/ml was a predictive factor of obtaining spontaneous conception post-operatively, compared to pregnancy through ART (p=0.0006), although it was not predictive of a livebirth [102]. It is worth noting that 36 patients (30.6%) required a cystectomy for concurrent OMA. Another retrospective study that focused on laparoscopic management of bladder DE, identified that the only patients with pregnancy intention that failed to conceive after surgery were those with documented DOR pre-operatively [103]: however, the actual number of patients (9 patients with pregnancy intention, 5 spontaneous conceptions and 1 through IVF) was too small to allow for meaningful conclusions [103]. Conversely, another retrospective study did not identify pre-operative AMH values to be predictive of conception (natural or through IVF) after surgery for stage III and IV endometriosis patients, including cases operated for colorectal endometriosis, with a mean age of 30 years [92]: conception rates, livebirth rates and probability of pregnancy at 12 and 24 months did not differ significantly between those with normal, low or very low AMH, with the majority of conceptions being spontaneous. Based on those findings, surgery may be viewed as a valid option for young patients with severe endometriosis and low AMH, in whom the results of IVF are anticipated to be suboptimal. However, the authors recognise that the small number of patients with low or very low AMH (n=46), in comparison to those with normal AMH (n=134), may have impacted interpretation of the results, as increasing the sample size in the former group may have led to a reduction in the livebirth rate compared to the normal

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AMH group. First-line surgery has recently been linked to improved pregnancy and livebirth rates compared to first-line IVF in infertile women with DE without bowel involvement and low AMH (< 2 ng/ml) [104].

A recent retrospective study confirmed that, for patients with endometriosis-related infertility, a low pre-operative AMH value was negatively associated with chances of conception after laparoscopic surgery, however, it did not affect livebirth rates [40]: according to the authors, this finding is a reflection of the significance of AMH as an ovarian reserve marker but not a marker of the quality of the follicle.

4. Conclusions

Regarding OMA, evidence suggests that its presence per se is linked to reduced AMH levels and higher rate of AMH decline over time compared to controls. On the other hand, its surgical management causes a more pronounced AMH decline: OMA cystectomy causes a higher reduction compared to non-excisional approaches and the effect appears larger compared to cystectomy for other benign ovarian cysts. Non-excisional approaches to OMA also cause an AMH reduction in the short-term post-operative period, however, the effect is likely smaller than cystectomy and recovers. Regarding OMA cystectomy, a recovery in AMH over time may, also, be anticipated, a phenomenon less likely to occur in bilateral OMAs. The post-operative recovery occurs as a result of follicular cohort re-organisation induced by surgery. Furthermore, it should be remembered that factors other than follicular loss during surgery may be involved in the post-operative AMH decline, such as advancing patient age. Data on long-term follow-up after OMA surgery are scarce and, therefore, should be interpreted with caution. OMA cystectomy, bilateral OMAs, increasing stage/severity of endometriosis and advanced patient age are linked to a more pronounced AMH decline after surgery. Meta-analyses had found non-thermal methods of haemostasis following OMA cystectomy to cause a greater fall in AMH compared to non-thermal methods, however, two well-designed RCTs did not identify significant differences between these approaches. Recurrent OMAs per se are not linked to reduced AMH levels, however, cystectomy for recurrent OMA causes a significant decline in AMH. It may, therefore, be prudent to avoid this procedure, if possible, particularly in case of patients with an already-low AMH value and pregnancy intention. The impact of OMA size on AMH values is, currently, not well established.

AMH levels before OMA surgery are positively correlated with the post-operative probability of pregnancy, particularly spontaneous conception, however, a link between AMH levels and livebirth rates has not been established based on the existing evidence. Pre-operative AMH levels are, also, predictive of the risk of DOR after OMA cystectomy, particularly important in the IVF setting. Similarly, post-operative AMH levels and the rate of AMH decline at 1 year after OMA surgery appear predictive of the fertility outcomes.

To date, concrete evidence suggesting a superiority of robotic surgery over conventional laparoscopy in terms of post-operative AMH values is lacking.

Available evidence on AMH levels for extra-ovarian endometriosis is limited. For the SUP phenotype, its impact on AMH levels (if any) is likely to be less pronounced compared to OMA. One study found that excision of SUP did not significantly reduce AMH values. DE appears to exert a negative impact on AMH levels through a variety of proposed mechanisms. Surgical management of DE causes a reduction in AMH values, possibly due to a reduction in ovarian blood supply secondary to extensive adhesiolysis. The post-operative AMH decline is higher when OMA co-exists with DE, compared to OMA alone or DE alone, according to one observational study. Limited available evidence for DE cases does not support that AMH values are predictive of post-operative fertility outcomes. Therefore, surgery is a valid option, particularly for young patients with DE and low AMH wishing to conceive, for whom IVF outcomes are anticipated to be suboptimal due to poor ovarian reserve.

When examining the available evidence, various limitations need to be taken into account, both relevant to study design (small sample size, lack of control groups and reduced follow-up duration

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of most studies), as well as secondary to the heterogeneity of the disease, current diagnostic limitations and different surgical modalities available.

Last but not least, it should be remembered that any AMH decline following endometriosis surgery does not reflect an absolute decline in the fertility and reproductive potential of the patient. More research is needed to clarify the exact role of AMH values, pre- and post-operatively, in predicting the fertility outcomes of patients operated for endometriosis, particularly extra-ovarian locations of the disease.

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Abbreviations

The following abbreviations are used in this manuscript:

AFC	Antral Follicular Count
AMH	Anti-Müllerian Hormone
ART	Artificial Reproductive Technology
BMI	Body Mass Index
CPR	Cumulative Pregnancy Rate
DE	Deep Endometriosis
DOR	Diminished Ovarian Reserve
DWLS	Dual Wavelength Laser System
ICSI	Intracytoplasmic Sperm Injectio
IVF	In-vitro Fertilization
OMA	Ovarian Endometrioma
POF	Premature Ovarian Failure
RCT	Randomized Controlled Trial
SUP	Superficial Peritoneal Endometriosis

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