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ARTICLE

Probability of live birth in women with extremely low anti-Müllerian hormone concentrations

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Abstract The aim of the present study was to investigate the clinical pregnancy and live birth rates in women with extremely low (≤0.4 ng/ml) anti-Müllerian hormone (AMH) concentrations. The study included 101 women (188 cycles) with extremely low AMH concentrations undergoing IVF cycles and compared the number of live births in women with low AMH. Moreover, the study compared the number of live births in women with or without endometriosis stage III/IV. Fourteen clinical pregnancies and 14 live births (including one pair of twins) were recorded; one woman miscarried. Significantly higher clinical pregnancy ($P = 0.046$) and live birth rates ($P = 0.018$) were found in women aged <35 years compared with older women. AMH concentration did not differ significantly between women with or without endometriosis and there were six live births in women with endometriosis. This was not significantly different from the rate in healthy women. It is concluded that live births are possible in women with extremely low AMH concentrations. The presence of endometriosis stage III/IV did not affect live birth rates in women with extremely low AMH concentrations although an important limitation of the study is the small number of women included who were affected by that disease.

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KEYWORDS: AMH, endometriosis, inhibin B, ovarian reserve, DHEA, IVF

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Introduction

Anti-Müllerian hormone (AMH) is an established marker of ovarian reserve (La Marca et al., 2010; Nelson et al., 2009) and predicts both high and low responses in ovarian stimulation cycles (Eldar-Geva et al., 2005; Nardo et al., 2009; Nelson et al., 2007). Presently, AMH helps clinicians counsel patients prior to IVF treatment (La Marca et al., 2011), despite the fact that it fails to predict who will become pregnant (Lamazou et al., 2011; Riggs et al., 2011). It has been demonstrated that poor responders can achieve both pregnancy and live birth (Weghofer et al., 2011). There are few studies regarding extremely low AMH concentrations and live births (Fraisse et al., 2008; Tocci et al., 2009; Weghofer et al., 2011) and they present either a small number of patients or limited data describing the groups of investigated patients.

Another factor affecting pregnancy rates is endometriosis, a chronic gynaecological disease characterized by the presence of functional endometrial tissue outside the uterine cavity (Koninckx et al., 1991). Many studies have reported that pregnancy rates are lower in women with endometriosis than in controls (Gupta et al., 2008; Koninckx et al., 1991 Pellicer et al., 2000). Lower AMH serum concentrations are associated with endometriosis severity (Shebl et al., 2006).

The primary objective of the present study was to assess live birth rates in women with extremely low AMH concentrations with respect to age. Additionally, another objective was to determine live birth rates in women with both extremely low AMH concentrations and endometriosis stage III/IV.

Materials and methods

Selection of subjects

This study retrospectively analysed a computer database of women with extremely low AMH concentrations treated with intracytoplasmic sperm injection (ICSI) in the (IVF) unit (invicta private fertility Centre) between May 2007 and January 2011. Serum AMH assays were included as a standard measure in the IVF program. A cut-off AMH value of $\leq$0.4 ng/ml was chosen according to Gnoth et al. (2008) and Weghofer et al. (2011). AMH concentrations were measured prior to the start of each cycle.

Women were divided into three age categories — $<$35, 35—39 and $>$39 years — according to data presented by Mosher and Pratt (1991).

Endometriosis staging was performed according to the revised classification of the American Society for Reproductive Medicine, ranging from moderate to severe (American Society For Reproductive Medicine, 1997). None of the patients had medical treatment for endometriosis within 3 months prior to laparoscopy. The interval between laparoscopy and IVF was 3.36 ± 2.8 years. Moreover, none of the participants had been taking any hormonal treatment for at least 3 months before entering the study. This study received expedited Institutional Review Board approval (reference no. 4/2011, approved 6 October 2011). Written informed consent was obtained from each included couple to perform ICSI on at least some of the retrieved oocytes. All data were de-identified and analysed anonymously. Moreover, informed consent to present our data in any publication was obtained as long as confidentiality was maintained.

Specimen collection and preparation and hormone analysis

Fasting venous blood samples (7 ml) were collected aseptically without any additives from 08:00 to 12:00 h between days 1 and 3 of the menstrual cycle prior to the beginning of stimulation. Blood was allowed to clot at room temperature and serum was separated by centrifugation (10 min at 1500 *g*). The samples were stored at −20°C until analysis. Serum FSH concentrations were measured using a standard chemiluminescence immunoassay (Immulite; DPC, Los Angeles, CA, USA) according to the manufacturer's instructions. The lower detection concentration was 0.1 mIU/ml. Serum AMH and inhibin B concentrations were measured by ELISA (Diagnostic Systems Laboratories, Webster, TX, USA). The assay limit of detection was 0.06 ng/ml for AMH and 7 pg/ml for inhibin B. The intra- and interassay coefficients of variation were $<$10% for all parameters.

Stimulation protocol

All women underwent a long protocol of pituitary suppression with the gonadotrophin-releasing hormone agonist Diphereline at a dose of 0.1 mg/day (Pharmacia Upjohn, Kalamazoo, MI, USA), beginning on day 14 of the oral contraception cycle. Fourteen days later (i.e. 7 days after the end of oral contraceptive administration and after menses), the administration of urinary gonadotrophins (Gonal-F; Serono, Feltham, UK; or Fostimon; Genevrier, Sophia-Antipolis, France) for ovarian stimulation was initiated (300 IU/day). Gonadotrophin treatment was initiated if no follicles were larger than 10 mm in diameter and oestradiol concentrations were $<$50 pg/ml. The FSH dose was based on the woman's age and AMH concentration. In this unit, 300 IU/day is routinely given to patients with extremely low AMH concentrations.

Follicular growth was monitored using a day-8 ultrasonographic scan and a serum oestradiol assay. Ovulation was induced by administration of 5000 IU human chorionic gonadotrophin (Pregnyl, Organon, Oss, Netherlands) when at least one leading follicle had reached a diameter of 17 mm and oocyte retrieval was performed 36 h later.

Embryo transfer was performed on cleavage-stage day 5 in all cases using a soft catheter. The number of embryos transferred was determined by the available number and quality of embryos and by the guidelines of the institution and ASRM (Practice Committee. Society for Assisted Reproductive Medicine and the American Society for Reproductive Medicine, 2004).

All patients were given supplementation with natural micronized progesterone (Luteina; Adamed, Czosnów, Poland), given vaginally in three divided doses of 200 mg/day, beginning on the day of oocyte retrieval. The

women received supplementation with oral 6-mg doses of micronized 17 β-oestradiol (Estrofem; Novo Nordisk, Denmark) daily during the entire luteal phase. A serum β-human chorionic gonadotrophin pregnancy test was performed 14 days after oocyte retrieval.

Clinical pregnancy was defined as the presence of an intrauterine gestational sac as visualized by transvaginal ultrasonography. Live birth was defined as the birth of at least one live child and was considered to be a successful endpoint in this investigation.

Statistical analysis

Data were evaluated with Statistica software for Windows version 10.0. All continuous variables were evaluated for normal distribution using the Kolmogorov–Smirnov test. Results were expressed as mean ± SD or median. One-way analysis of variance (ANOVA) or the Kruskal–Wallis test was used to compare the means or medians of the three age groups. The Bonferroni test for post-hoc analysis was also performed to show differences between women in different age categories. Pregnancy outcomes are shown as the mean ± SD with 95% confidence intervals (CI). Categorical variables were compared using the Pearson chi-squared and Fisher's exact tests when necessary. All tests were two-tailed and the significance level was defined as $P < 0.05$. Logistic regression of pregnancy to AMH concentration as a continuous variable was used to evaluate associations between different AMH concentrations and pregnancy potential.

Results

During the study period, 188 cycles of 101 women were investigated. Six cycles (five women) were excluded from the study as they included prenatal genetic screening.

Baseline patient characteristics are shown in Table 1. With the exception of patient age and AMH concentration, clinical characteristics of all groups were similar. In women aged >39 years, AMH concentrations were significantly lower than in women aged <35 years and in those aged 35–39 years ($P < 0.05$). The median age of all patients included in the study population was 38 years.

There was a total of 188 cycles. One embryo was transferred in 42 cycles, two were transferred in 27 cycles and three were transferred in five cycles. In 111 cycles, there was no embryo transfer (no oocytes were aspirated in 23 cycles, no metaphase-II oocytes in 26 cycles, fertilization failure in 41 cycles, embryonic arrest in 16 cycles and frozen embryos in five cycles). Three cycles were cancelled due to a lack of response to gonadotrophin.

Table 2 presents the clinical pregnancy and live birth rates per patient and per cycle of 101 women (188 cycles). Fourteen clinical pregnancies were recorded (7.4% per cycle started and 13.9% cumulative) and 14 live births in 13 women (one pair of twins). Four live births occurred after the first cycle, seven live births occurred after the second cycle, two live births occurred after the third cycle and one live birth occurred after the fourth cycle. One woman miscarried.

When evaluated according to age, there were significantly higher clinical pregnancy and live birth rates in women aged <35 years (both 25.0%) compared with women aged 35–39 years (both 10.3%) and >39 years (5.6% and 2.8%, respectively) ($P = 0.046$ and $P = 0.018$, respectively; Table 2). Univariate regression analysis did not reveal a significant association between AMH concentration (<0.1 to 0.4 ng/ml) and either pregnancy potential or live birth (OR 1.36, 95% CI 0.74–2.47).

In this series, 23 women (49 cycles) had endometriosis stage III/IV. The mean AMH concentration was 0.27 ± 0.12 ng/ml and was distributed as follows: 0.27 ± 0.12 ng/ml in women aged <35 years, 0.26 ± 0.14 ng/ml in women aged 35–39 years and 0.18 ± 0.11 ng/ml in women aged >39 years; there were no statistically significant differences between the age groups. There were also no statistically significant differences when AMH concentrations were compared in both women with endometriosis (0.27 ± 0.12,

Table 1 Baseline patient characteristics and IVF cycle characteristics in women with extremely low AMH.

Characteristic	*All women* (n = *101*)	*<35 years* (n = *36*)	*35–39 years* (n = *29*)	*>39 years* (n = *36*)
Cycles	188	67	57	64
Age (years)	37.7 ± 5.1 (38)	32.4 ± 2.3 (33)	37.5 ± 1.0 (37)	43.0 ± 2.5 (43)[a]
FSH (mIU/ml)	15.6 ± 17.6 (11)	13.0 ± 9.4 (11)	15.1 ± 20.9 (10.3)	19.7 ± 21.1 (12.3)
AMH (ng/ml)	0.3 ± 0.1 (0.3)	0.3 ± 0.1 (0.3)	0.3 ± 0.1 (0.3)	0.2 ± 0.1 (0.2)[a]
Inhibin B (pg/ml)	25.5 ± 27.3 (13.7)	22.7 ± 26.6 (7.8)	21.8 ± 20.9 (7)	21.1 ± 24.0 (16.2)
AFC	3.0 ± 4.7 (2)	3.5 ± 5.3 (2)	2.8 ± 2.7 (2)	2.8 ± 5.3 (2)
Total gonadotrophin dosage	2409.8 ± 1287.3 (2475)	2420.3 ± 876.8 (2550)	2553 ± 1251 (2475)	2267.3 ± 1728.8 (2175)
Oocytes >17 mm	3.3 ± 3.1 (3)	3.9 ± 4.3 (3)	3.5 ± 2.4 (3)	2.5 ± 1.7 (2)
Oocytes retrieved	2.5 ± 3.4 (2)	3.3 ± 4.7 (2)	2.7 ± 2.7 (2)	1.7 ± 1.6 (1)
MII oocytes	1.7 ± 2.3 (1)	2.5 ± 3.4 (1)	1.6 ± 1.6 (1)	1.1 ± 1.2 (1)
Cycles per patient	2.0 ± 1.7 (1)	1.7 ± 1.0 (1)	2.6 ± 2.4 (2)	1.9 ± 1.3 (1)

Values are mean ± SD (median).
AFC = antral follicle count; AMH = anti-Müllerian hormone; MII = metaphase-II.
[a]Significantly lower compared with both women aged <35 years and women aged 35–39 years ($P < 0.05$).

Table 2 Pregnancy outcomes in women with extremely low AMH concentrations Chi -squared and Fisher's exact tests.

Outcome	*All women* (n = *101*)	*<35 years* (n = *36*)	*35–39 years* (n = *29*)	*>39 years* (n = *36*)	P-*value*[a]
Cycles	188	67	57	64	
Clinical pregnancy	14	9	3	2	
Per cycle	7.4 (4.1–12.3)	13.4 (6.3–23.9)	5.3 (1.1–14.6)	3.1 (0.4–10.8)	>0.05
Per patient	13.9 (7.7–22.1)	25.0 (12.1–42.2)	10.3 (2.2–27.3)	5.6 (0.6–18.6)	0.046
Live birth	14[b]	10[b]	3	1	
Per cycle	6.9 (3.7–11.5)	13.4 (6.3–23.9)	5.3 (61.1–14.6)	1.6 (0.0–8.4)	0.024
Per patient	12.9 (7–21)	25.0 (12.1–42.2)	10.3 (2.2–27.3)	2.8 (0.1–14.5)	0.018

Values are *n* or% (95% CI).
[a]Compared with women aged 35–39 years and >39 years.
[b]Including one pair of twins.

Table 3 Pregnancy outcome in women with extremely low AMH concentrations with and without endometriosis stage III/IV.

Outcome	*<35 years*		*35–39 years*		*>39 years*	
	Endometriosis (n = *12*)	*Without endometriosis* (n = *24*)	*Endometriosis* (n = *4*)	*Without endometriosis* (n = *25*)	*Endometriosis* (n = *7*)	*Without endometriosis* (n = *29*)
Clinical pregnancy	5	4	1	2	0	2
Live birth	5	20	1	2	0	1

Values are *n*.
No statistically significant differences were found.

median 0.3 ng/ml) and those without endometriosis (0.26 ± 0.11, median 0.3 ng/ml).

Of the six live births achieved in women with endometriosis, five were in women aged <35 years (live birth rate of 41.7%) and one was in a woman aged 35–39 years (li birth rate of 25.0%). No pregnancies were recorded in women aged >39 years. There were no statistically significant differences between women with and without endometriosis in any of age groups (**Table 3**).

Discussion

This study investigated the probability of live birth following assisted reproduction treatment in women with extremely low AMH concentrations. Moreover, it compared live birth rates in women with extremely low AMH concentrations with or without endometriosis stage III/IV. In the past, many studies have concluded that AMH concentrations could predict pregnancy success (Broer et al., 2009; Knauff et al., 2009; Singer et al., 2009) However, only a few large studies have shown the relationship between AMH concentrations and live births (Gleicher et al., 2010; La Marca et al., 2011; Lee et al., 2009; Nelson et al., 2007). A current diagnostic issue for clinicians is the treatment of women with extremely low AMH concentrations. In that group of patients can be expected poor ovarian response, which can lead to cycle termination, thus lowering the probability of pregnancy (La Marca et al., 2010). It seems clear that clinicians should communicate the probability of live birth when the woman has extremely low AMH concentrations to allow both the couples and the doctors to either begin treatment (when a low probability of live birth is accepted) or present other possibilities to achieve pregnancy (e.g. oocyte donation).

AMH concentration and age are independently associated with live birth (La Marca et al., 2010, 2011). Both AMH concentrations and full-term pregnancy decrease with increasing age (La Marca et al., 2010). Both La Marca et al. (2010) and Nelson et al. (2007) independently showed that cut-off AMH values between 0.7 and 0.75 ng/ml predict poor ovarian response. Nelson et al. (2007) estimated the probability of live birth in women with AMH concentrations in this range to be 15%. La Marca et al. (2010) found that, for women with very low AMH concentrations, either cycle termination or poor response could be anticipated. Muttukrishna et al. (2004) found that women with very low AMH concentrations (0.1–0.35 ng/ml) are at very high risk for cycle termination, and La Marca et al. (2010) proposed that these patients should be refused treatment. However, Tocci et al. (2009) described a case of a 34-year-old woman who had a successful delivery with AMH concentrations <0.5 ng/ml.

An excellent study was performed by Weghofer et al. (2011) which showed that, of 128 women with extremely low AMH concentrations (≤0.4 ng/ml), 70 women aged <42 years presented with 16 clinical pregnancies, resulting

in 10 deliveries, but only four clinical pregnancies with two deliveries. All patients received dehydroepiandrosterone (DHEA) supplementation. According to the literature, DHEA may improve ovarian reserve (Gleicher et al., 2010; Wiser et al., 2010). The current study reported 14 clinical pregnancies in 13 women with extremely low AMH concentrations and, additionally, a clinical pregnancy rate (13.9%) similar to that 15.6%) in the study of Weghofer et al., 2011), but only one woman miscarried in the current study compared with eight (40%) in the other study. These differences are probably due to the younger ages of the patients in this study (median 38 years versus 41.6 years in the study of Weghofer et al., 2011). In contrast to Weghofer et al. (2011), this study centre did not supplement women with DHEA during the time period under investigation (since the latter half of 2011, DHEA supplementation or acupuncture has been used for to women with extremely low AMH concentrations). Thus, these results confirm that, even without supplementation, the likelihood of live birth is still a reasonable reason to begin treatment.

Another aspect of this study was to compare live birth rates between women with low AMH concentrations with or without endometriosis stage III/IV. As far as is known, this is the largest study in this field. Researchers have shown that endometriosis can affect reproductive outcome, resulting in low-quality embryos with reduced implantation rates (Garrido et al., 2000). Endometriosis may also lead to diminished ovarian reserve and worse IVF outcome (Barnhart et al., 2002). AMH concentrations are also considered a useful clinical predictor of poor ovarian response in patients with endometriosis (Shebl et al., 2006). The current study in women with low AMH concentrations found no statistically significant differences between women with or without endometriosis with respect to the number of live births, although more pregnancies were recorded in women without endometriosis. The limitation of this study is the small number of women and pregnancies in the group with endometriosis.

Counselling women with extremely low AMH concentrations can be difficult, because, as La Marca et al. (2010) stated, the predictive value for AMH concentrations is not absolute; its false-positive rate may have previously prohibited women from entering an IVF programme. The strength of that study is the large number of patients with extremely low AMH concentrations who did not receive DHEA supplementation. Moreover, as far as is known, this is the first study to evaluate women with extremely low AMH concentrations and endometriosis stage III/IV. Although this work presents pilot information in this field, it can also be stated that women with endometriosis stage III/IV can achieve pregnancy and live births.

This study is not without limitations. First, it did not analyse women with endometriosis stage I/II, and the overall number of women and pregnancies with endometriosis is not large. The results may reflect that, in women with extremely low AMH concentrations, pregnancy chances are reduced but do not further decline in the presence of endometriosis. Because the number of women with endometriosis and pregnancies was small, a power analysis was performed. The statistical power was low (0.49), and to demonstrate differences between groups in clinical pregnancies, at least 108 women would have been required in each arm, accepting a type I error as 0.05 with the power of 80%. Thus, that the lack of difference observed may be a result of the small study group. Secondly, as stated previously, analysis of the effect of endometriosis was not the main point of this study; thus, this study does not present the characteristics of the women according to presence or absence of endometriosis. Finally, this study is limited by its retrospective nature.

The hope of pregnancy is one of the most important things that a couple can expect from their doctor. This study concludes that live births are possible in women with extremely low AMH concentrations and hopes that its finding will have a positive impact on treatment strategies and will be important for decision making among couples seeking assistance becoming pregnant.

References

American Society For Reproductive Medicine, 1997. Revised American society for reproductive medicine classification of endometriosis: 1996. Fertil. Steril. 67, 817—821.

Barnhart, K., Dunsmoor-Su, R., Coutifaris, C., 2002. Effect of endometriosis on in vitro fertilization. Fertil. Steril. 77, 1148—1155.

Broer, S.L., Mol, B.W.J., Hendriks, D., Broekman, F.J., 2009. The role of antimullerian hormone in predicting outcome after IVF: comparison with antral follicle count. Fertil. Steril. 91, 705—714.

Eldar-Geva, T., Ben-Chetrit, A., Spitz, I.M., Rabinowitz, R., Markowitz, E., Mimoni, T., Gal, M., Zylber-Haran, E., Margalioth, E.J., 2005. Dynamic assays of inhibin B, anti-Mullerian hormone and estradiol following FSH stimulation and ovarian ultrasonography as predictors of IVF outcome. Hum. Reprod. 20, 3178—3183.

Fraisse, T., Ibecheole, V., Streuli, I., Bischof, P., de Ziegler, D., 2008. Undetectable serum anti Mullerian hormone levels and occurrence of ongoing pregnancy. Fertil. Steril. 89, 723e729—711.

Garrido, N., Navarro, J., Remoh, J., Simón, C., Pellicer, A., 2000. Follicular hormonal environment and embryo quality in women with endometriosis. Hum. Reprod. Update 6, 67—74.

Gleicher, N., Weghofer, A., Barad, H., 2010. Anti-Mullerian hormone (AMH) defines, independent of age, low versus good live-birth chances in women with severely diminished ovarian reserve. Fertil. Steril. 9, 2824—2827.

Gnoth, C., Schuring, A.N., Friol, K., Tigges, J., Mallmann, P., Godehardt, E., 2008. Relevance of anti-Mullerian hormone measurement in a routine IVF program. Hum. Reprod. 23, 1359—1365.

Gupta, S., Goldberg, J.M., Aziz, N., Goldberg, E., Krajcir, N., 2008. Pathogenic mechanisms in endometriosis-associated infertility. Fertil. Steril. 90, 247—257.

Koninckx, P.R., Meuleman, C., Demeyere, S., Lesaffre, E., Cornillie, F.J., 1991. Suggestive evidence that pelvic endometriosis is a progressive disease, whereas deeply infiltrating endometriosis is associated with pelvic pain. Fertil. Steril. 55, 759—765.

Knauff, E.A., Eijkemans, M.J., Lambalk, C.B., ten Kate-Booij, M.J., Hoek, A., Beerendonk, C.C., Laven, J.S., Goverde, A.J., Broekmans, F.J., Themmen, A.P., de Jong, F.H., Fauser, B.C., 2009. Anti-Mullerian hormone, inhibin B, and antral follicle count in young women with ovarian failure. J. Clin. Endocrinol. Metab. 94, 786—792.

Lamazou, F., Genro, V., Fuchs, F., Grynberg, M., Gallot, V., Achour-Frydman, N., Fanchin, R., Frydman, R., 2011. L'AMH serique n'a pas de valeur predictiveen fecondation in-vitro en cycle naturel modifie: analyse de 342 cycles. J de Gynécologie Obstétrique et Biologie de la Reprod 40, 205—210.

La Marca, A., Sighinolfi, G., Radi, D., Argento, C., Baraldi, E., Artenisio, A.C., Stabile, G., Volpe, A., 2010. Anti-Mullerian hormone-as a predictive marker in assisted reproductive technology (ART). Hum. Reprod. Update 16, 113—130.

La Marca, A., Nelson, S.M., Sighinolfi, G., Manno, M., Baraldi, E., Roli, L., Xella, S., Marsella, T., Tagliasacchi, D., D'Amico, R., Volpe, A., 2011. Anti-Mullerian hormone-based prediction model for a live birth in assisted reproduction. Reprod. Biomed. Online 22, 341—349.

Lee, T.H., Liu, C.H., Huang, C.C., Hsieh, K.C., Lin, P.M., Lee, M.S., 2009. Impact of female age and male infertility on ovarian reserve markers to predict outcome of assisted reproduction technology cycles. Reprod. Biol. Endocrinol. 7, 100.

Mosher, W.D., Pratt, W.F., 1991. Fecundity and infertility in the United States: incidence and trends. Fertil. Steril. 56, 192—193.

Muttukrishna, S., Suharjono, H., McGarrigle, H., Sathanandan, M., 2004. Inhibin B and anti-Mullerian hormone: markers of ovarian response in IVF/ICSI patients? BJOG 111, 1248—1253.

Nardo, L.G., Gelbaya, T.A., Wilkinson, H., Roberts, S.A., Yates, A., Pemberton, P., Laing, I., 2009. Circulating basal anti-Müllerian hormone levels as predictor of ovarian response in women undergoing ovarian stimulation for in vitro fertilization. Fertil. Steril. 92, 1586—1593.

Nelson, S.M., Yates, R.W., Fleming, R., 2007. Serum anti-Mullerian hormone and FSH: prediction of live birth and extremes of response in stimulated cycles-implications for individualization of therapy. Hum. Reprod. 22, 2414—2421.

Nelson, S.M., Yates, R.W., Lyall, H., Jamieson, M., Traynor, I., Gaudoin, M., Mitchell, P., Ambrose, P., Fleming, R., 2009. Anti-Mullerian hormone-based approach to controlled ovarian stimulation for assisted conception. Hum. Reprod. 24, 867—875.

Pellicer, A., Albert, C., Garrido, N., Navarro, J., Remohi, J., Simón, C., 2000. The pathophysiology of endometriosis-associated infertility: follicular environment and embryo quality. J. Reprod. Fertil. Suppl. 55, 109—119.

Practice Committee. Society for Assisted Reproductive Medicine and the American Society for Reproductive Medicine, 2004. Guidelines on the Number of Embryos Transferred: A Practice Committee Report. Fertil. Steril. 82, 772—774.

Riggs, R., Kimble, T., Oehninger, S., Bocca, S., Zhao, Y., Leader, B., Stadtmauer, L., 2011. Anti-Mullerian hormone serum levels predict response to controlledovarian hyperstimulation but not embryo quality or pregnancy outcome in oocyte donation. Fertil. Steril. 95, 410—412.

Shebl, O., Ebner, T., Sommergruber, M., Sir, A., Tews, G., 2006. Anti Muellerian hormone serum levels in women with endometriosis: a case-control study. Gynecol. Endocrinol. 25, 713—716.

Singer, T., Barad, D.H., Weghofer, A., Gleicher, N., 2009. Correlation of antimullerian hormone and baseline follicle-stimulating hormone levels. Fertil. Steril. 91, 2616—2619.

Tocci, A., Ferrero, S., Iacobelli, M., Greco, E., 2009. Negligible serum anti-mullerian hormone: pregnancy and birth after a 1-month course of an oral contraceptive, ovarian hyperstimulation, and intracytoplasmic sperm injection. Fertil. Steril. 92, 395, e399—395, e312.

Weghofer, A., Dietrich, W., Barad, D.H., Gleicher, N., 2011. Live birth chances in women with extremely low-serum anti-Mullerian hormone levels. Hum. Reprod. 26, 1905—1909.

Wiser, A., Gonen, O., Ghetler, Y., Shavit, T., Berkovitz, A., Shulman, A., 2010. Addition of dehydroepiandrosterone (DHEA) for poor-responder patients before and during IVF treatment improves the pregnancy rate: a randomized prospective study. Hum. Reprod. 25, 2496—2500.

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