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Original Research Article

Predictors of optimal ovarian response in GnRH antagonist ovarian stimulation protocol

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ABSTRACT

Background: The outcome of assisted reproductive technology (ART) is significantly influenced by the ovarian response to controlled ovarian stimulation (COS). Identifying robust and reliable predictors of ovarian response is essential for tailoring individualized treatment strategies and optimizing reproductive success. This study aims to investigate clinical, hormonal, and stimulation-related variables that influence ovarian response among women undergoing ART and to identify significant predictors for both hyper-response and hypo-response patterns.
Methods: A retrospective cohort analysis was conducted involving 278 women who underwent COS under a gonadotropin-releasing hormone (GnRH) antagonist protocol. Based on ovarian responsiveness, participants were categorized into three groups: high responders (n=56), normal responders (n=151), and low responders (n=71). Demographic data, hormonal markers (including Anti-Müllerian hormone [AMH], antral follicle count [AFC], follicle-stimulating hormone [FSH], and luteinizing hormone [LH]), stimulation characteristics, and infertility etiologies were systematically analyzed. Statistical comparisons utilized t-tests and chi-square tests, while logistic regression identified independent predictive parameters. A p-value of less than 0.05 was considered statistically significant.
Results: Higher AFC and AMH levels, younger age, and lower baseline FSH levels were significantly correlated with high ovarian response. In contrast, women with low AMH, high FSH, reduced AFC, and prolonged stimulation duration tended to demonstrate poor ovarian responsiveness. Notably, polycystic ovary syndrome (PCOS) was more frequent in high responders, while diminished ovarian reserve (DOR) predominated in low responders. Multivariate logistic regression identified AMH and AFC as the most significant independent predictors of ovarian response.
Conclusions: Age, AMH, AFC, and baseline FSH are critical determinants of ovarian response in ART cycles. Incorporating these biomarkers into pre-treatment evaluation facilitates the customization of stimulation protocols, thereby enhancing oocyte yield and improving overall clinical outcomes. Personalized treatment planning grounded in these predictors holds promise for advancing ART success.

Keywords: Antral follicle count, Assisted reproductive technology, Controlled ovarian stimulation, Follicle-stimulating hormone, Gonadotropin-releasing hormone antagonist protocol, Ovarian response, Predictive biomarkers

INTRODUCTION

Controlled ovarian hyperstimulation (COH) represents a critical phase within the framework of assisted reproductive technology. The acquisition of an optimal quantity of high-quality oocytes post-fertilization, requisite for the development of superior embryos suitable for implantation into the uterine cavity, is paramount for achieving successful pregnancy.[1] Ovarian reactivity denotes the responsiveness of the ovary to exogenous gonadotropins (Gn) throughout the COH process. The degree of ovarian reactivity is instrumental in determining the capacity to recruit an adequate number of oocytes, which constitutes one of the pivotal determinants of COH success[2], and has a direct impact on the entirety of the ovulation induction process as well as the outcomes of assisted reproductive techniques. Ovarian reactivity can be categorized into three distinct classifications: low ovarian

response; normal ovarian response; and high ovarian response. A low ovarian response manifests as a suboptimal reaction to Gn stimulation, resulting in a minimal yield of harvested oocytes. Conversely, a high ovarian response occurs when the ovary exhibits heightened sensitivity to Gn stimulation, leading to an excessive number of oocytes being produced, a condition referred to as ovarian hyper-response, which represents a significant factor potentially precipitating ovarian hyperstimulation syndrome.

The adoption of gonadotropin-releasing hormone (GnRH) antagonists has gained prominence in clinical settings owing to their advantageous characteristics, including ease of administration, flexibility, and reduced side effects, thereby establishing them as a standard clinical protocol.[3,4] This approach effectively mitigates the down-regulatory influence associated with prolonged recovery periods and substantially diminishes the incidence of ovarian hyperstimulation syndrome, thereby enhancing the safety profile of in vitro fertilization and embryo transfer (IVF-ET) interventions. Concurrently, the treatment duration with GnRH antagonists is abbreviated, the required dosage of Gn is decreased, ovarian function exhibits rapid recovery, and patient satisfaction surpasses that observed with GnRH agonists.[5,6] Nonetheless, due to the relatively shallow inhibition of the pituitary gland, there exists a risk of an early luteinizing hormone (LH) peak, which may precipitate premature ovulation.[7] Consequently, it is imperative to prescribe an appropriate initial and cumulative dosage of Gn to elicit favorable ovarian responses while preempting ovarian overstimulation during COH. An insufficient starting dose may inadvertently induce a low ovarian response, while a dosed increase carries the potential for a high response.[8] Additionally, it is crucial to modulate the Gn dosage in accordance with the ovarian response observed during the ovulation induction phase and to incorporate antagonists when deemed necessary.

Anti-Mullerian hormone (AMH), inhibin B, chronological age, antral follicle count (AFC), and baseline sex hormones are frequently employed in clinical practice to forecast ovarian responsiveness.[9,10] These parameters, however, exhibit limitations in their predictive capacity concerning ovarian responsiveness, with cut-off values lacking standardization, and it is infeasible to assess ovarian responsiveness comprehensively through individual indicators in a singular patient.[11] Our objective was to identify independent risk factors influencing ovarian responsiveness in the context of GnRH antagonists through stepwise regression analysis, and to formulate a nomogram model aimed at predicting ovarian responsiveness predicated upon the regression coefficients of these variables. Each female patient undergoing in vitro fertilization/intracytoplasmic single sperm microinjection-embryo transfer (IVF/ICSI-ET) was provided with a tailored ovulation management plan, designed to secure the requisite number of oocytes and thereby enhance pregnancy outcomes.

METHODS

Study design and setting

This retrospective cohort study was conducted at the Department of Reproductive Medicine, Madras Medical mission and Hospital, Chennai. It included women who underwent in vitro fertilization (IVF) or intracytoplasmic sperm injection (ICSI) cycles using a gonadotropin-releasing hormone (GnRH) antagonist protocol between 2018 to 2024.

Ethical Consideration

Prior to initiation, ethical clearance was obtained from the Institutional Ethics Committee (IEC) of Madras Medical Mission. The retrospective design ensured that all patient identifiers were anonymized, and confidentiality was preserved in accordance with ethical guidelines.

Study population

A total of 278 women undergoing controlled ovarian stimulation for ART were included. The inclusion criteria were: Age between 21 and 40 years, body mass index (BMI) between 18 and 30 kg/m², regular menstrual cycles (24–35 days), undergoing the first or second ART cycle, and availability of complete clinical and hormonal data.

Women were excluded if they had: Chromosomal or genetic abnormalities, congenital uterine anomalies, history of ovarian surgery, recurrent pregnancy loss, use of donor oocytes or surrogacy, and systemic illness or incomplete documentation.

Ovarian stimulation protocol

Controlled ovarian stimulation was initiated with recombinant FSH (rFSH) or human menopausal gonadotropin (HMG) from day 2 or 3 of the menstrual cycle. The initial dose (150–300 IU/day) was individualized based on age, BMI, ovarian reserve markers including anti-Müllerian hormone (AMH) and antral follicle count (AFC), and previous response to stimulation, if any. GnRH antagonist (cetrorelix acetate 0.25 mg/day) was introduced when at least one follicle reached ≥14 mm in diameter, generally between days 5-6 of stimulation, and was continued until the day of ovulation trigger.

Final oocyte maturation was achieved using either recombinant human chorionic gonadotropin (hCG) or a GnRH agonist, depending on individual risk assessment for ovarian hyperstimulation syndrome (OHSS). Oocyte retrieval was carried out 35-36 hours post-trigger under transvaginal ultrasound guidance.

Classification of ovarian response

Based on the number of oocytes retrieved, participants were stratified into three groups: Low responders: ≤4

oocytes, Normal responders: 5-14 oocytes, and High responders: ≥15 oocytes.

This classification is consistent with established clinical criteria for ovarian response evaluation.

Data collection

Demographic and clinical parameters recorded included age, BMI, duration of infertility, and infertility etiology. Hormonal data such as basal FSH, LH, estradiol (E2), and AMH levels (on days 2-3 of the menstrual cycle) were obtained. AFC was assessed via transvaginal ultrasonography by experienced sonographers. Stimulation-related characteristics including duration of gonadotropin use, total gonadotropin dose, number of mature follicles on trigger day, and estradiol levels were documented.

Outcome measures

The primary outcomes were the number of oocytes retrieved, total gonadotropin dose, estradiol concentration on the trigger day, and ovarian response categorization. Secondary outcomes included distribution of infertility etiologies such as diminished ovarian reserve (DOR), endometriosis, male factor infertility, and polycystic ovary syndrome (PCOS).

Statistical analysis

All data were analyzed using IBM SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent sample t-tests or one-way ANOVA as applicable. Categorical variables were presented as frequencies and percentages and analyzed using the Chi-square test. To identify independent predictors of low and high ovarian response compared to the normal response group, multivariate logistic regression analysis was performed. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. A p-value <0.05 was considered statistically significant.

RESULTS

The study cohort was divided into three groups based on ovarian response: high (n=56), normal (n=151), and low (n=71). Upon analyzing demographic variables, it was observed that the mean age significantly differed among the groups. Women in the high response group had a younger mean age (29.05±3.24 years) compared to those in the normal (32.78±4.61 years) and low response groups (34.90±4.72 years), with p-values indicating statistical significance when comparing low versus normal (p=0.002) and high versus normal (p=0.001).

Table 1: Comparison of clinical parameters across different patient groups (high, normal, and low).

	High=56		Normal=151		Low=71		Low vs normal P value	High vs normal P value
Outcome	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**	**Mean**	**Std. Deviation**		
DOM	4.88	1.73	6.09	2.38	6.42	3.22	0.383	0.001
Duration of infertility	4.73	1.69	5.99	2.35	5.37	2.93	0.092	0.000
Total dose	1639.38	1127.36	2134.40	1294.06	2261.73	1067.39	0.471	0.012
E2 level on trigger day	5537.14	2629.31	3562.94	1658.59	1299.39	999.17	0.000	0.000
Age	29.05	3.24	32.78	4.61	34.90	4.72	0.002	0.000
BMI	28.04	5.67	27.32	3.96	26.52	4.66	0.188	0.305
Duration of GN	10.38	0.89	10.36	0.90	10.72	1.49	0.029	0.939
FSH	6.00	1.29	6.09	1.49	7.37	2.56	0.000	0.659
AFCRL	20.41	5.22	10.56	3.66	6.76	2.91	0.000	0.000
Serum LH	5.19	2.42	4.21	2.15	4.30	3.44	0.799	0.005
AMH	6.05	2.83	3.11	1.81	1.55	1.41	0.000	0.000

Body mass index (BMI) did not show a statistically significant difference between the groups. Although the mean BMI was slightly higher in the high response group (28.04±5.67 kg/m²) compared to the normal (27.32±3.96 kg/m²) and low groups (26.52±4.66 kg/m²), the differences were not statistically meaningful (p=0.188 and p=0.305, respectively).

When examining the duration of infertility, the high responders demonstrated a shorter mean duration (4.73±1.69 years) compared to the normal responders (5.99±2.35 years) and low responders (5.37±2.93 years). The comparison between low versus normal (p=0.092) approached significance, whereas the difference between high versus normal was statistically significant (p=0.001).

Table 2: Comparative analysis of clinical and hormonal parameters among high, normal, and low groups.

	High (n=56)	Normal (n=151)	Low (n=71)	P value	P value
	Mean (SD)	Mean (SD)	Mean (SD)	Low vs normal	High vs normal
Age	29.05 (3.24)	32.78 (4.61)	34.9 (4.72)	0.002	0.001
BMI	28.04 (5.67)	27.32 (3.96)	26.52 (4.66)	0.188	0.305
Duration of infertility	4.73 (1.69)	5.99 (2.35)	5.37 (2.93)	0.092	0.001
DOM	4.88 (1.73)	6.09 (2.38)	6.42 (3.22)		
Infertility reason n (%)					
Others (reference)	2 (2.8)	32 (21.2)	5 (8.9)	0.001	0.001
DOR	23 (32.4)	0 (0.0)			
Endometriosis	42 (59.2)	26 (17.2)	3 (5.4)		
Male factor	3 (4.2)	88 (58.3)	10 (17.9)		
PCOS	1 (1.4)	5 (3.3)	38 (67.9)		
AMH	6.05 (2.83)	3.11(1.81)	1.55 (1.41)	0.001	0.001
AFCRL	20.41 (5.22)	10.56 (3.66)	6.76 (2.91)	0.001	0.001
FSH7	6 (1.29)	6.09 (1.49)	7.37 (2.56)	0.001	0.659
Serum LH	5.19 (2.42)	4.21 (2.15)	4.3 (3.44)	0.799	0.005
Total dose	1639.38 (1127.36)	2134.4 (1294.06)	2261.73 (1067.39)	0.471	0.012
Duration of GN	10.38 (0.89)	10.36 (0.9)	10.72 (1.49)	0.029	0.939
E2 level on trigger day	5537.14 (2629.31)	3562.94 (1658.59)	1299.39 (999.17)	0.001	0.001

Table 3: Comparative analysis of clinical and hormonal parameters among high and normal group.

High vs normal	B	P value	Odds ratio	95% C.I. for EXP (B)	
				Lower	Upper
Age	-0.203	0.140	0.816	0.623	1.069
BMI	0.056	0.579	1.058	0.868	1.289
Duration of GN	0.265	0.640	1.303	0.430	3.949
FSH	-0.125	0.781	0.882	0.364	2.139
AFCRL	0.919	0.001	2.507	1.539	4.084
Serum LH	-0.179	0.400	0.836	0.551	1.268
AMH	0.312	0.193	1.366	0.854	2.184
Infertility reason (others)		0.003			
Infertility reason EDO	1.390	0.433	4.013	0.124	129.702
Infertility reason male	-2.132	0.141	0.119	0.007	2.020
Infertility reason PCOS	3.151	0.044	23.363	1.093	499.343
Constant	-13.651	0.106	0.000		

In terms of ovarian stimulation outcomes, the number of oocytes retrieved (Degree of Ovarian Response, DOM) was significantly higher in the high response group (mean 4.88±1.73) compared to the normal (6.09±2.38) and low response groups (6.42±3.22), with statistically significant differences noted.

Evaluation of hormonal profiles revealed notable differences. Baseline FSH levels were significantly lower in high responders (6.00±1.29 mIU/ml) compared to normal (6.09±1.49 mIU/ml) and low responders (7.37±2.56 mIU/ml), with p-values indicating significance for comparisons between low versus normal (p=0.001) and high versus normal (p=0.659). AMH levels were markedly higher in high responders (6.05±2.83 ng/mL) relative to normal (3.11±1.81 ng/ml) and low responders (1.55±1.41 ng/mL), and the differences were highly significant (p=0.001 for both comparisons).

Similarly, antral follicle count (AFC) was substantially greater in the high response group (20.41±5.22) compared to the normal (10.56±3.66) and low (6.76±2.91) response groups, with statistically significant differences observed (p=0.001 for both comparisons).

Estradiol (E2) levels on the day of trigger were also significantly elevated among high responders (5537.14±2629.31 pg/ml) compared to normal (3562.94±1658.59 pg/ml) and low responders (1299.39±999.17 pg/ml), with strong statistical significance in both comparisons (p=0.001).

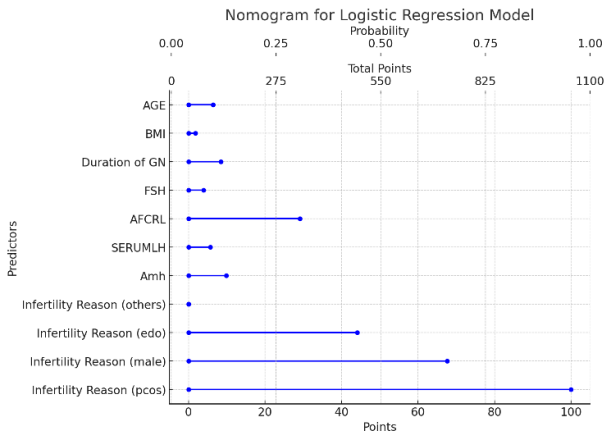

Figure 1: Nomogram for logistic regression model.

Assessment of infertility etiologies showed that diminished ovarian reserve (DOR) was predominantly seen in the high response group, while male factor infertility was more prevalent in the normal group, and polycystic ovarian syndrome (PCOS) was overwhelmingly associated with the low response group.

Multivariate logistic regression analysis identified several independent predictors of ovarian response. In the high versus normal comparison, an increased antral follicle count (AFC) was significantly associated with a higher likelihood of being in the high response group (odds ratio [OR]: 2.507; 95% confidence interval [CI]: 1.539-4.084; p=0.000). Additionally, PCOS as the cause of infertility was strongly associated with high response (OR: 23.363; 95% CI: 1.093-499.343; p=0.044).

Table 4: Comparative analysis of clinical and hormonal parameters among low and normal group.

Low VS normal	B	P value	Odds ratio	95% C.I. for EXP(B)	
				Lower	Upper
Age	-0.163	0.05	0.85	0.72	1.00
BMI	0.062	0.37	1.06	0.93	1.22
Duration of GN	0.856	0.01	2.35	1.23	4.50
FSH	0.545	0.01	1.72	1.17	2.54
AFCRL	-0.393	0.00	0.67	0.55	0.83
Serum LH	-0.201	0.14	0.82	0.63	1.07
AMH	-0.775	0.01	0.46	0.26	0.82
Infertility reason (others)		0.01			
Infertility reason DOR	24.651	1.00	50798028144.66	0.00	
Infertility reason ENDO	3.187	0.01	24.21	3.03	193.63
Infertility reason male	-0.594	0.60	0.55	0.06	4.98
Infertility reason PCOS	4.318	0.02	75.01	1.85	3039.66
Constant	-5.847	0.15	0.00		

In the low versus normal comparison, a longer duration of gonadotropin stimulation (OR: 2.35; 95% CI: 1.23-4.50; p=0.01) and higher FSH levels (OR: 1.72; 95% CI: 1.17-2.54; p=0.01) were significant predictors of low ovarian response. Conversely, lower AFC (OR: 0.67; 95% CI: 0.55-0.83; p=0.00) and lower AMH levels (OR: 0.46; 95% CI: 0.26-0.82; p=0.01) were associated with an increased likelihood of being in the low response group. Furthermore, PCOS as an infertility factor was also significantly associated with low response (OR: 75.01; 95% CI: 1.85-3039.66; p=0.02).

Overall, the study findings highlight that younger age, higher AMH levels, higher antral follicle count, and PCOS are associated with a high ovarian response, whereas higher FSH levels, prolonged stimulation duration, lower AFC, and lower AMH levels are predictive of a low ovarian response. These parameters can aid clinicians in tailoring individualized ovarian stimulation protocols to optimize ART outcomes.

DISCUSSION

Earlier study identifies key factors influencing ovarian response, including age, anti-Mullerian hormone, antral follicle count, and others. A nomogram prediction model was developed, achieving a 76.4% consistency in predicting ovarian response during assisted reproductive technology.[12] Various study had identified age, body mass index (BMI), follicle-stimulating hormone (FSH), antral follicle count (AFC), and anti-mullerian hormone (AMH) as independent risk factors for high ovarian response, leading to a prediction model with an AUC of 0.884.[13] The study constructed a nomogram model using FSH/LH ratios at basal,E2 level on the trigger day to predict ovarian response and reproductive potential, highlighting the importance of these ratios for optimizing the gonadotropin-releasing hormone antagonist protocol outcomes.[14] Another study identifies basal LH level, LH on triggering day, and bLH/hLH ratio as independent predictors of clinical pregnancy and live birth rates, recommending specific LH suppression thresholds for

normal, high, and poor ovarian responses during the GnRH antagonist protocol.[15]

Arms et al (2023) had found that serum AMH levels significantly predict ovarian response, with 47% of variation attributed to AMH alone. Including age, body weight, and total gonadotropin dose increased this to 50.9%, enhancing the prediction model's accuracy for optimal response.[16] Xu et al (2025) had identified optimal luteinising hormone levels on trigger day as a key factor influencing ovarian response and pregnancy outcomes in GnRH antagonist protocols, emphasizing the importance of individualized stimulation protocols to balance oocyte yield and reproductive success.[17]

AMH, AFC, and serum FSH were identified as significant predictive factors for ovarian response in a GnRH antagonist protocol. The study established models for predicting low and high ovarian responses based on these factors, highlighting inter-cycle variability.[18]

Oehninger et al (2011) had reported that anti-Mullerian hormone (AMH), antral follicle count (AFC), age, and follicle-stimulating hormone (FSH) as predictors for high ovarian response, while older age, higher AMH, higher AFC, and longer menstrual cycle length predicted low ovarian response in the GnRH antagonist protocol.[19] The study evaluates antral follicle count (AFC), anti-Müllerian hormone (AMH), and basal follicle-stimulating hormone (FSH) as predictors of ovarian response in women undergoing controlled ovarian stimulation.[20] The study identifies female age, AFC, and basal serum FSH and LH as key prognostic factors for ovarian response. It presents models predicting high and low responses, achieving AUCs of 0.82 and 0.80, respectively, for effective treatment personalization.[21]

CONCLUSION

The present study comprehensively evaluated the clinical, hormonal, and stimulation-related factors influencing ovarian response among women undergoing assisted reproductive technology (ART) using a gonadotropin-releasing hormone (GnRH) antagonist protocol. It was observed that younger age, higher anti-Müllerian hormone (AMH) levels, greater antral follicle count (AFC), and lower follicle-stimulating hormone (FSH) concentrations were significantly associated with a high ovarian response. Conversely, elevated baseline FSH, lower AMH and AFC, longer duration of gonadotropin stimulation, and diminished ovarian reserve were predictive of a low ovarian response.

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