

Original Research Article

Biochemical predictors of ovarian hyperstimulation syndrome in women undergoing in vitro fertilization: a retrospective cohort study from a tertiary fertility center in Pakistan

Anum Ehsan¹, Fazia Raza¹, Nosheen Shams¹, F. N. U. Sawaira²,
Abdul B. Wardak^{3*}, Ghulam M. Solangi⁴

¹Department of Obstetrics and Gynaecology, Peshawar General Hospital, Khyber Pakhtunkhwa, Pakistan

²Department of Obstetrics and Gynaecology, Khyber Girls Medical College, Khyber Pakhtunkhwa, Pakistan

³Department of Obstetrics and Gynaecology, Razia Bahlol Wardak Hospital, Kabul, Afghanistan

⁴Centre for Reproductive Medicine, Peshawar General Hospital, Khyber Pakhtunkhwa, Pakistan

Received: 30 June 2026

Revised: 12 August 2026

Accepted: 13 August 2026

*Correspondence:

Dr. Abdul B. Wardak,

E-mail: a.baseer21@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Ovarian hyperstimulation syndrome (OHSS) is a rare but serious complication of controlled ovarian stimulation during assisted reproductive technology. This study identified biochemical predictors and described outcomes of OHSS among women undergoing *in vitro* fertilization (IVF) at a tertiary fertility center in Khyber-Pakhtunkhwa, Pakistan.

Methods: Records of 316 infertile women treated at the Center for Reproductive Medicine, Peshawar General Hospital, from December 2023 to December 2024 were reviewed. Patient characteristics, infertility diagnosis, treatment parameters, anti-Müllerian hormone (AMH), peak serum estradiol (E2), antral follicular count (AFC), and total oocytes retrieved were assessed. All patients received HCG Alpha 250 µg for final oocyte maturation. OHSS was classified using Golan and Weissman (2009) criteria. Analysis was performed using IBM SPSS Statistics v26.

Results: The mean age was 32.21±4.85 years and BMI 27.42±1.71 kg/m²; 69.6% had PCOS. Sixteen patients (5.06%) developed OHSS, all with PCOS as a contributing risk factor. 25.0% had moderate, 62.5% severe, and 12.5% had critical OHSS. Compared with non-OHSS patients, mean AFC (24 versus 13), AMH (6.5 versus 3.4 ng/ml), peak E2 (35 versus 18 ng/ml), and oocytes retrieved (15 versus 9) were higher. Nine (56.25%) achieved live birth, all after frozen embryo transfer.

Conclusions: AFC, AMH, peak serum estradiol, and total oocytes retrieved are the principal biochemical markers associated with OHSS in this Pakistani IVF cohort. The universal use of standard hCG triggering in a PCOS-predominant population represents the most modifiable contributor to the high severity distribution observed from low - and -middle income country centers.

Keywords: Infertility, *In vitro* fertilization, Ovarian hyperstimulation syndrome, Biochemical predictors, PCOS, Pakistan, Low-middle-income country

INTRODUCTION

Ovarian hyperstimulation syndrome (OHSS) is a rare but serious complication of controlled ovarian stimulation (COS) during assisted reproductive technology (ART)

treatments.¹ COS drives the simultaneous development of multiple follicles, substantially increasing oocyte yield but also the risk of an exaggerated systemic response.² Severity is classified into four grades, namely mild, moderate, severe, and critical, based on clinical, sonographic, and laboratory parameters.³⁻⁵

The overall incidence of OHSS varies across studies. Moderate OHSS is reported in 3-6% of ART cycles, while severe OHSS occurs in 0.1-2%.⁶ Established pre-treatment risk factors include a previous history of OHSS, polycystic ovarian syndrome (PCOS), now termed polycystic metabolic ovarian syndrome (PMOS), as well as young age, low body weight, and elevated anti-Müllerian hormone (AMH) levels.^{2,7-12} During stimulation, controlled ovarian hyperstimulation (COH)-related risk factors include retrieval of more than 20 oocytes, increased follicle number and size, and high serum estradiol (E2) concentrations.^{6,8,13} PCOS is particularly relevant in this context, as the condition is associated with a large resting follicle pool, exaggerated gonadotropin sensitivity, and amplified E2 and AMH responses during stimulation, all of which potentiate OHSS risk.⁷⁻¹⁰ The pathophysiology of OHSS involves vascular endothelial growth factor (VEGF)-mediated increases in vascular permeability and hemoconcentration under the influence of exogenous gonadotropins.^{14,15} Activating mutations in the FSH receptor that permit cross-reactivity with human chorionic gonadotropin (hCG) have also been implicated in a subset of cases.¹⁶

There is a paucity of data from low- and lower-middle-income countries (LMICs) on OHSS incidence and its biochemical predictors in women undergoing IVF.¹⁷ Pakistan represents such a setting, where sociocultural, economic, and religious barriers significantly limit ART access and utilization, resulting in small patient volumes and limited institutional experience with high-risk ovarian stimulation. PCOS prevalence is substantial in South Asian populations, making this population particularly susceptible to OHSS during IVF, yet data characterizing the biochemical risk profile of OHSS in this specific population remain extremely limited.^{8,9} The primary objective of this study was to identify biochemical predictors of OHSS and describe its clinical outcomes in women undergoing IVF at a tertiary fertility center in Khyber-Pakhtunkhwa, Pakistan.

METHODS

We conducted a retrospective cohort study analyzing the medical records of 316 infertile women treated at the Center for Reproductive Medicine, Peshawar General Hospital, Peshawar, Pakistan, from December 2023 to December 2024. The study was approved by the Institutional Ethics Committee of Peshawar General Hospital, and all data were anonymized prior to analysis in accordance with the declaration of Helsinki.¹⁸ This study was reported in accordance with the STROBE guidelines for observational studies.¹⁹

Inclusion criteria were all female patients who underwent at least one complete IVF cycle at the center during the study period. Exclusion criteria were: incomplete medical records with missing primary outcome data; patients who withdrew from treatment before oocyte retrieval; and and

cycles cancelled prior to trigger due to poor ovarian response. Data were collected by convenience sampling.

Ovarian stimulation was performed using a standard ovarian stimulation protocol. Gonadotropin type and starting dose were individualized based on patient age, BMI, AFC, and AMH levels. Final oocyte maturation was triggered with hCG alpha 250 µg, administered when the leading follicle reached a mean diameter of 18 mm on transvaginal ultrasound. Oocyte retrieval was performed 34-36 hours after trigger under ultrasound guidance.

Data extracted from records included patient characteristics (age, BMI, parity, occupation, education level, area of residence), medical and obstetric history, infertility diagnosis, duration of infertility, number of prior IVF attempts, and cycle outcomes. Biochemical variables collected included serum AMH levels, peak serum E2 on the day of trigger, antral follicular count (AFC) on baseline transvaginal ultrasound, total number of oocytes retrieved, and maximum follicular diameter at time of trigger. Estradiol levels were recorded in ng/ml as per the laboratory reporting format at this center.

OHSS was classified according to the Golan and Weissman (2009) criteria: mild OHSS was defined as bilateral ovarian enlargement with multiple follicular cysts up to 8 cm, with abdominal bloating and mild pain; moderate OHSS shared these features with additional sonographic ascites; severe OHSS was defined by ovarian cysts >12 cm, clinical ascites with or without hydrothorax, hemoconcentration (hematocrit >45%), oliguria (<300 ml/day), and electrolyte disturbances; and critical OHSS required hematocrit >55%, white cell count >25,000/ml, oliguria/anuria, creatinine ≥1.6 mg/dl, or acute respiratory distress syndrome.^{3,4,5}

Statistical analysis was performed using IBM SPSS statistics version 26. Continuous variables were expressed as mean (standard deviation not available from records; see limitations) and described separately for the OHSS and non-OHSS groups; formal inferential comparison between groups was not possible given the absence of standard deviations. Categorical variables were expressed as frequencies and percentages; no formal inferential comparison of categorical variables between groups was performed. Univariate binary logistic regression was planned to quantify the independent predictive association of each biochemical marker (AFC, AMH, estradiol, oocytes retrieved) with OHSS development; however, this analysis could not be completed due to missing standard deviations and variable-level data (see limitations). Cases with missing primary outcome data were excluded from analysis.

RESULTS

A total of 316 women underwent IVF during the study period and met inclusion criteria. The mean age of the cohort was 32.21±4.85 years and mean BMI was

27.42±1.71 kg/m². The mean duration of infertility post-marriage was 6.27±2.63 years, and the mean number of prior IVF attempts was 1.03±0.175, indicating the vast majority were undergoing their first IVF cycle. Most patients resided in urban settings (town or city: 91.4%; village: 8.5%). Regarding educational attainment, 51.2% held a bachelor's degree, 21.8% had completed higher secondary education, 14.2% held a master's degree, 12.6% had secondary education, and none had only primary-level education. Most patients were unemployed (65.8%). A history of PCOS was present in 220 patients (69.6% of the total cohort). No patient had a prior history of OHSS.

Table 1: Social demographics and medical history of total patients.

Variables	Mean±SD/percentage	
Age (years)	32.21±4.85)	
BMI (kg/m ²)	27.42±1.71	
Duration of infertility post marriage (years)	6.266±2.63	
No of IVF attempts	1.03±0.175	
Occupation	Unemployed	65.8
	Employed	34.2
Education level	Primary	0.0
	Secondary	12.6
	Higher secondary	21.8
	Bachelors	51.2
	Masters	14.2
Residency	Village	8.54
	Town/city	91.4
History of PCOS	N=220	69.62
History of OHSS	N=0	0.0

Of the 316 patients, 16 (5.06%) developed OHSS. PCOS was identified as a contributing risk factor in all 16 OHSS patients. Notably, despite a 69.6% PCOS prevalence in the overall cohort, OHSS occurred in only 5.06% of all patients, underscoring the role of additional biochemical markers in discriminating which PCOS patients are at highest risk. Among the 16 OHSS cases, no patient had mild OHSS; 4 (25.0%) had moderate, 10 (62.5%) had severe, and 2 (12.5%) had critical OHSS. Recovery from OHSS occurred within 7-10 days. The predominance of severe and critical cases likely reflects the use of standard hCG triggering in all patients and the absence of routine prophylactic OHSS protocols at this center.

Comparison of laboratory characteristics between OHSS (n=16) and non-OHSS (n=300) patients is summarized in Tables 2 and 3. On descriptive comparison, mean AFC was 24 in OHSS patients versus 13 in non-OHSS patients. Mean AMH was 6.5 ng/ml in the OHSS group versus 3.4 ng/ml in non-OHSS patients. Mean peak serum estradiol was 35 ng/ml in the OHSS group versus 18 ng/ml in non-OHSS patients. The mean number of oocytes retrieved was 15 in the OHSS group versus 9 in non-OHSS patients. The mean diameter of the leading follicle at trigger was 18 mm in both groups, and hCG alpha 250 µg was used as the

trigger in all patients. Standard deviations and inferential p values were not available from records; these differences are reported descriptively only.

Table 2: Severity and laboratory characteristics of OHSS patients (n=16).

Variables	N/mean
Mild OHSS	N=0
Moderate OHSS	N=4
Severe OHSS	N=10
Critical OHSS	N=2
Antral follicular count	Mean=24
Anti-müllerian hormone levels	Mean=6.5 ng/ml
Estradiol levels	Mean=35 ng/ml
Average size of mature follicle at time of trigger	18 mm
Trigger for final maturation was induced by hCG alpha	250 µg
Average time of recovery from OHSS	7-10 days
Number of oocytes retrieved >	Mean=15

Table 3: Laboratory characteristics of non-OHSS patients post IVF (n=300).

Variables	N/mean
Antral follicular count	Mean=13
Anti-müllerian hormone levels	Mean=3.4 ng/ml
Estradiol levels	Mean=18 ng/ml
Average size of mature follicle at time of trigger	18 mm
Trigger for final maturation was induced by hCG alpha	250 µg
Number of oocytes retrieved >	Mean=9

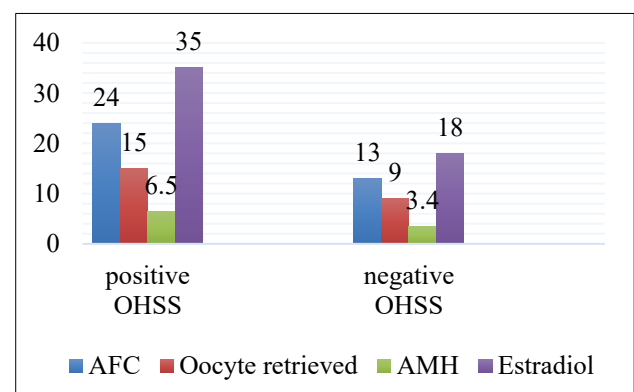


Figure 1: Comparison of biochemical markers in OHSS positive versus negative patients.

The four markers, namely AFC, AMH, peak serum estradiol, and total oocytes retrieved, demonstrated the largest descriptive differences between groups and represent the strongest candidates for formal regression-based predictor analysis in future prospective studies with complete data.

Regarding embryo transfer strategy and reproductive outcomes, frozen embryo transfer (FET) was performed in 13 of 16 OHSS patients (81.25%). A total of 9 of 16 OHSS patients (56.25%) achieved a live birth (take-home baby), all following FET. Of the 9 live births, 7 occurred in patients with severe OHSS and 2 in patients with moderate OHSS. No live births were recorded in the 2 patients with critical OHSS, though the small number precludes firm conclusions. Remaining cryopreserved embryos were retained for future ART use. Outcomes of the 7 OHSS patients who did not achieve a live birth were not available from records. No thromboembolic events or maternal deaths were recorded during the study period.

DISCUSSION

This retrospective cohort study from Khyber-Pakhtunkhwa, Pakistan, contributes data on OHSS in a PCOS-predominant IVF cohort from an LMIC setting, where such data remain scarce. The biochemical markers most strongly associated with OHSS in our cohort, specifically AFC, AMH, peak serum estradiol, and total oocyte yield, are consistent with those identified in studies from higher-income settings, suggesting broadly similar risk factor profiles across contexts, though formal cross-setting comparisons were not within the scope of this study.²⁰

The overall OHSS incidence of 5.06% in our cohort represents all-severity cases and falls within the 1-10% range reported for total OHSS incidence globally.^{6,21,22} This rate should not be compared directly with severity-specific benchmarks: globally, moderate OHSS occurs in 3-6% and severe OHSS in 0.1-2% of ART cycles.⁶ The high proportion of severe (62.5%) and critical (12.5%) cases within our OHSS cohort is clinically notable. A key contributing factor was the universal use of hCG alpha 250 µg as the trigger agent in all patients. In a cohort where 69.6% had PCOS, a condition strongly associated with exaggerated ovarian response, the continued use of standard hCG triggering rather than a GnRH agonist trigger is among the most modifiable risk factors in this dataset. GnRH agonist triggering in GnRH antagonist cycles substantially reduces the risk of severe OHSS by producing a physiological LH surge of limited duration, avoiding the prolonged luteotropic stimulus of exogenous hCG; this approach is most applicable in GnRH antagonist IVF protocols.^{23,24} The absence of this strategy in a high-risk PCOS cohort likely accounts substantially for the severe and critical OHSS distribution observed. Furthermore, the high PCOS burden in the overall cohort (69.6%) yet OHSS rate of only 5.06% underscores that PCOS alone is insufficient to predict OHSS: it is the combination of elevated AFC, AMH, estradiol, and oocyte yield that identifies the highest-risk patients within a PCOS-predominant population.⁷⁻¹⁰

The predominance of severe and critical OHSS in this cohort highlights the need for proactive risk stratification and targeted prevention in LMIC fertility centers. In terms

of management, the use of frozen embryo transfer (FET) in 81.25% of OHSS patients in this cohort is commendable and consistent with current evidence supporting elective freeze-all strategies to decouple ovarian stimulation from embryo implantation, thereby reducing the amplifying effect of endogenous HCG from an early pregnancy on an already hyper-stimulated ovary.^{23,25} This practice likely contributed to the 56.25% live birth rate achieved despite the high severity distribution. However, FET manages OHSS once it has occurred; it does not prevent it. The primary prevention gap in this cohort was the use of standard hCG triggering in all patients. Prophylactic strategies that should be incorporated into local protocols for high-risk patients (defined by AFC >15, AMH >3.5 ng/ml, or retrieval of >15 oocytes, per ASRM guideline thresholds include: GnRH agonist triggers where a GnRH antagonist protocol is employed; prophylactic cabergoline 0.5 mg daily for 8 days post-trigger, which has demonstrated efficacy in reducing moderate-to-severe OHSS in Cochrane-level evidence; and individualized gonadotropin dosing guided by AFC and AMH from the outset of stimulation.^{23,24,26-29} Comprehensive prevention protocols for high-risk patients are detailed in current clinical practice guidelines.³⁰ Intravenous albumin has a role in secondary prophylaxis at the time of oocyte retrieval in very high-risk cases and in management of established severe OHSS but is not a primary preventive strategy.³¹

The descriptive differences between OHSS and non-OHSS patients are numerically substantial across all four biochemical markers. Mean AFC was nearly double in OHSS patients (24 versus 13), consistent with published thresholds of AFC ≥14-16 as the upper-risk boundary for excessive response.²⁰ Mean AMH was 6.5 ng/ml in OHSS patients versus 3.4 ng/ml in non-OHSS patients, a near-twofold difference corroborating the meta-analytic data of Broer et al, who identified AMH as the strongest individual predictor of excessive ovarian response, consistent with prior evidence.³²⁻³⁴ Mean peak estradiol was 35 ng/ml in OHSS patients versus 18 ng/ml in non-OHSS patients. Mean oocytes retrieved was 15 in the OHSS group versus 9 in non-OHSS patients, consistent with Asch et al who defined retrieval of >20 oocytes as a high-risk threshold.¹³ These findings corroborate Enskog et al, who prospectively demonstrated that elevated AFC and estradiol during COH were the strongest markers for OHSS development.⁷ Formal regression analysis in future studies would quantify these associations with precision and adjust for confounders.

The 56.25% take-home baby rate in our OHSS cohort, all achieved via frozen embryo transfer, is a reassuring reproductive outcome given the severity distribution. Ganer Herman et al demonstrated that OHSS does not preclude satisfactory reproductive outcomes; our findings are consistent with this reassurance and support elective cryopreservation with deferred FET as an effective management strategy in LMIC settings.¹ Courbiere et al similarly reported acceptable pregnancy rates in OHSS

patients managed with FET, supporting the strategy employed at this center.³⁵ The absence of thromboembolic events or maternal deaths in our cohort, despite 12.5% critical OHSS, reflects adequate clinical management of the most severe cases, which include thromboembolic disease and respiratory compromise as the most feared complications.³⁶

Our cohort profile sheds light on the sociocultural landscape of IVF utilization in Khyber-Pakhtunkhwa. Despite a relatively educated sample (51.2% held a bachelor's degree and 14.2% a master's degree), 65.8% of patients were unemployed, reflecting the predominantly dependent economic status of women in this region and the financial inaccessibility of ART for many infertile couples. The small dataset (316 patients over 12 months at a single center) itself reflects the limited uptake of ART in this population. These findings are consistent with qualitative evidence from Pakistan on barriers to ART utilization, including financial constraints, social stigma, and limited awareness of fertility services.³⁷ While IVF is broadly permissible under Islamic jurisprudence when conducted within marriage, misinformation and cultural barriers persist in conservative communities. Targeted public health education and subsidized ART programs may be necessary to improve access in this population.

Several limitations of this study merit consideration. First, the retrospective single-center design is susceptible to data incompleteness, referral bias, and selection bias; results may not be generalizable to the broader Pakistani IVF population. Second, the small number of OHSS events (n=16) substantially limits statistical power and precludes multivariate logistic regression, meaning the influence of individual markers cannot be adjusted for confounders; all estimates should be interpreted cautiously. Third, standard deviations for the biochemical variables were not available from records, precluding formal inferential statistical comparison between groups; the reported differences are descriptive only. Fourth, the specific ovarian stimulation protocol (GnRH agonist long versus antagonist) and individualized gonadotropin doses were not systematically captured from records, representing important unstandardized confounders. Fifth, long-term obstetric and neonatal outcomes beyond the index live birth were not recorded. Sixth, outcomes of the 7 OHSS patients who did not achieve a live birth were not available from records, representing a data gap in the reproductive outcome analysis.

Despite these limitations, this study provides a foundational real-world dataset on OHSS characteristics and outcomes from Khyber-Pakhtunkhwa, Pakistan, and reports locally observed mean values for AFC, AMH, estradiol, and oocyte yield that provide a baseline for risk characterization in similar LMIC settings. Prospective multicenter studies with standardized stimulation protocols, complete laboratory data including standard deviations, precise OHSS onset timing, and long-term

follow-up are needed to build a robust evidence base for OHSS prevention and management in Pakistan.³⁸

CONCLUSION

This study demonstrates that AFC, AMH, peak serum estradiol, and total oocyte yield are the principal biochemical markers associated with OHSS in women undergoing IVF at a tertiary center in Khyber-Pakhtunkhwa, Pakistan. PCOS was present in all OHSS cases, reinforcing its central role as a predisposing condition. The predominance of severe and critical OHSS despite the use of FET in 81.25% of affected patients reflects the absence of upstream preventive strategies, specifically GnRH agonist triggering where antagonist protocols are employed and prophylactic cabergoline, and highlights the need for their adoption in LMIC fertility centers. Future prospective multicenter studies with standardized stimulation protocols and complete laboratory data are essential to build a robust evidence base for OHSS prevention and management in Pakistan and similar settings.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Ganer Herman H, Mizrahi Y, Horowitz E, Weissman A, Sabban B, Gluck O, et al. Obstetric outcomes following ovarian hyperstimulation syndrome in IVF: a comparison with uncomplicated fresh and frozen transfer cycles. *BMC Pregnancy Childbirth.* 2022;22(1):573.
2. Delvigne A, Dubois M, Battheu B, Bassil S, Meuleman C, De Sutter P, et al. The ovarian hyperstimulation syndrome in in-vitro fertilization: a Belgian multicentric study. II. Multiple discriminant analysis for risk prediction. *Hum Reprod.* 1993;8:1361-6.
3. Aboulghar MA, Mansour RT. Ovarian hyperstimulation syndrome: classifications and critical analysis of preventive measures. *Hum Reprod Update.* 2003;9(3):275-89.
4. Namavar Jahromi B, Parsanezhad ME, Shomali Z, Bakhshai P, Alborzi M, Vaziri NM, et al. Ovarian hyperstimulation syndrome: a narrative review of its pathophysiology, risk factors, prevention, classification, and management. *Iran J Med Sci.* 2018;43(3):248-60.
5. Golan A, Weissman A. A modern classification of OHSS. *Reprod Biomed Online.* 2009;19(1):28-32.
6. Delvigne A, Rozenberg S. Epidemiology and prevention of ovarian hyperstimulation syndrome (OHSS): a review. *Hum Reprod Update.* 2002;8(6):559-77.
7. Enskog A, Henriksson M, Unander M, Nilsson L, Brännström M. Prospective study of the clinical and

- laboratory parameters of patients in whom ovarian hyperstimulation syndrome developed during controlled ovarian hyperstimulation for in vitro fertilization. *Fertil Steril.* 1999;71:808-14.
8. Tummon I, Gavrilova-Jordan L, Allemand MC, Session D. Polycystic ovaries and ovarian hyperstimulation syndrome: a systematic review. *Acta Obstet Gynecol Scand.* 2005;84:611-6.
 9. Teede HJ, Khomami MB, Morman R, Laven JSE, Joham AE, Costello MF, et al. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. *Lancet.* 2026;407(10545):2329-39.
 10. Delvigne A, Demoulin A, Smits J, Donnez J, Koninckx P, Dhont M, et al. The ovarian hyperstimulation syndrome in in-vitro fertilization: a Belgian multicentric study. I. Clinical and biological features. *Hum Reprod.* 1993;8:1353-60.
 11. Navot D, Relou A, Birkenfeld A, Rabinowitz R, Brzezinski A, Margalioth EJ. Risk factors and prognostic variables in the ovarian hyperstimulation syndrome. *Am J Obstet Gynecol.* 1988;159:210-5.
 12. Nakhuda GS, Chu MC, Wang JG, Sauer MV, Lobo RA. Elevated serum müllerian-inhibiting substance may be a marker for ovarian hyperstimulation syndrome in normal women undergoing in vitro fertilization. *Fertil Steril.* 2006;85:1541-3.
 13. Asch RH, Li HP, Balmaceda JP, Weckstein LN, Stone SC. Severe ovarian hyperstimulation syndrome in assisted reproductive technology: definition of high risk groups. *Hum Reprod.* 1991;6:1395-9.
 14. Le Gouez A, Naudin B, Grynberg M, Mercier FJ. Ovarian hyperstimulation syndrome. *Ann Fr Anesth Reanim.* 2011;30(4):353-62.
 15. Schirmer DA, Kulkarni AD, Zhang Y, Kawwass JF, Boulet SL, Kissin DM. Ovarian hyperstimulation syndrome after assisted reproductive technologies: trends, predictors, and pregnancy outcomes. *Fertil Steril.* 2020;114(3):567-78.
 16. Vasseur C, Rodien P, Beau I, Desroches A, Gérard C, de Poncheville L, et al. A chorionic gonadotropin-sensitive mutation in the follicle-stimulating hormone receptor as a cause of familial gestational spontaneous ovarian hyperstimulation syndrome. *N Engl J Med.* 2003;349(8):753-9.
 17. Zareen N. Management and obstetric outcome of ovarian hyperstimulation syndrome. *J Coll Physicians Surg Pak.* 2008;18(2):86-90.
 18. World Medical Association. Declaration of Helsinki: ethical principles for medical research involving human subjects. *JAMA.* 2013;310(20):2191-4.
 19. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Ann Intern Med.* 2007;147(8):573-7.
 20. Humaidan P, Nelson SM, Devroey P, Coddington CC, Schwartz LB, Gordon K, et al. Ovarian hyperstimulation syndrome: review and new classification criteria for reporting in clinical trials. *Hum Reprod.* 2016;31(9):1997-2004.
 21. Nastri CO, Teixeira DM, Moroni RM, Louzada VF, Martins WP. Ovarian hyperstimulation syndrome: pathophysiology, staging, prediction and prevention. *Ultrasound Obstet Gynecol.* 2015;45(4):377-93.
 22. Budev MM, Arroliga AC, Falcone T. Ovarian hyperstimulation syndrome. *Crit Care Med.* 2005;33(10):S301-6.
 23. Devroey P, Polyzos NP, Blockeel C. An OHSS-free clinic by segmentation of IVF treatment. *Hum Reprod.* 2011;26(10):2593-7.
 24. Papanikolaou EG, Pozzobon C, Kolibianakis EM, Camus M, Tournaye H, Fatemi HM, et al. Incidence and prediction of ovarian hyperstimulation syndrome in women undergoing gonadotropin-releasing hormone antagonist in vitro fertilization cycles. *Fertil Steril.* 2006;85(1):112-20.
 25. Ferraretti AP, Gianaroli L, Magli MC, Fortini D, Selman HA, Felicini R. Elective cryopreservation of all pronucleate embryos in women at risk of ovarian hyperstimulation syndrome: efficiency and safety. *Hum Reprod.* 1999;14(6):1457-60.
 26. Practice Committee of the American Society for Reproductive Medicine. Prevention and treatment of moderate and severe ovarian hyperstimulation syndrome: a guideline. *Fertil Steril.* 2016;106(7):1634-47.
 27. Tang H, Hunter T, Hu Y, Zhai SD, Sheng X, Hart RJ. Cabergoline for preventing ovarian hyperstimulation syndrome. *Cochrane Database Syst Rev.* 2012;2:CD008605.
 28. Fiedler K, Ezcurra D. Predicting and preventing ovarian hyperstimulation syndrome (OHSS): the need for individualized not standardized treatment. *Reprod Biol Endocrinol.* 2012;10:32.
 29. Lensen SF, Wilkinson J, Leijdekkers JA, La Marca A, Mol BWJ, Marjoribanks J, et al. Individualised gonadotropin dose selection using markers of ovarian reserve for women undergoing in vitro fertilisation plus intracytoplasmic sperm injection. *Cochrane Database Syst Rev.* 2018;2(2):CD012693.
 30. Humaidan P, Quartarolo J, Papanikolaou EG. Preventing ovarian hyperstimulation syndrome: guidance for the clinician. *Fertil Steril.* 2010;94(2):389-400.
 31. Youssef MA, Al-Inany HG, Evers JL, Aboulghar M. Intra-venous fluids for the prevention of severe ovarian hyperstimulation syndrome. *Cochrane Database Syst Rev.* 2011;2:CD001302.
 32. Broer SL, Dölleman M, Opmeer BC, Fauser BC, Mol BW, Broekmans FJ. AMH and AFC as predictors of excessive response in controlled ovarian hyperstimulation: a meta-analysis. *Hum Reprod Update.* 2011;17(1):46-54.
 33. La Marca A, Sighinolfi G, Radi D, Argento C, Baraldi E, Artenisio AC, et al. Anti-Müllerian

- hormone (AMH) as a predictive marker in ART. *Hum Reprod Update.* 2010;16(2):113-30.
34. Lekamge DN, Barry M, Kolo M, Lane M, Gilchrist RB, Tremellen KP. Anti-Müllerian hormone as a predictor of IVF outcome. *Reprod Biomed Online.* 2007;14(5):602-10.
35. Courbiere B, Oborski V, Braunstein D, Desparoir A, Noizet A, Gamberre M. Obstetric outcome of women with in vitro fertilization pregnancies hospitalized for ovarian hyperstimulation syndrome: a case-control study. *Fertil Steril.* 2011;95(5):1629-32.
36. Vloeberghs V, Peeraer K, Pexsters A, D'Hooghe T. Ovarian hyperstimulation syndrome and complications of ART. *Best Pract Res Clin Obstet Gynaecol.* 2009;23(5):691-709.
37. Inhorn MC, Patrizio P. Infertility around the globe: new thinking on gender, reproductive technologies and global movements in the 21st century. *Hum Reprod Update.* 2015;21(4):411-26.
38. Mathur RS, Akande AV, Keay SD, Hunt LP, Jenkins JM. Distinction between early and late ovarian hyperstimulation syndrome. *Fertil Steril.* 2000;73(5):901-7.

Cite this article as: Ehsan A, Raza F, Shams N, Sawaira FNU, Wardak AB, Solangi GM. Biochemical predictors of ovarian hyperstimulation syndrome in women undergoing in vitro fertilization: a retrospective cohort study from a tertiary fertility center in Pakistan. *Int J Sci Rep* 2026;12(10):329-35.