European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Original Research Article

Maternal and Fetal Outcomes in Pregnant Women with Hyperemesis Gravidarum: A Prospective Observational Study

Article History:

Received: 29-11-2025
Revised: 10-12-2025
Accepted: 20-01-2026
Published: 08-02-2026

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Abstract: ***Background***: Hyperemesis gravidarum (HG) is a severe form of nausea and vomiting in pregnancy characterized by weight loss, dehydration, electrolyte imbalance, and ketonuria, affecting approximately 0.3–3% of pregnancies worldwide.[1–3,7,8] It is a leading cause of early-pregnancy hospitalization and has been linked to adverse maternal and fetal outcomes.[4–6,9–11] Data from South Indian populations remain limited. ***Methods***: This prospective observational study was conducted over 12 months in the Department of Obstetrics and Gynaecology at a tertiary care teaching hospital in South India. We enrolled 200 pregnant women with singleton gestations ≤22 weeks diagnosed with HG as per WHO criteria. Maternal demographic, clinical, biochemical, and management details were recorded at presentation. Participants were followed until delivery to document maternal outcomes (pregnancy-induced hypertension, gestational diabetes, preterm birth, mode of delivery) and fetal outcomes (gestational age at birth, birth weight, small-for-gestational-age [SGA] status, Apgar scores, NICU admission). Associations between HG severity and outcomes were analyzed using chi-square tests, t-tests, and multivariable logistic regression ($p<0.05$). ***Results***: The mean maternal age was 26.7±4.5 years; 60% were primigravida. Most women presented at 10.2±2.3 weeks' gestation. Weight loss >5% of pre-pregnancy weight occurred in 64%, ketonuria in 58%, and electrolyte abnormalities in 42%. Hospital admission was required in 82%, with a mean stay of 3.8±1.9 days; 22% had recurrent admissions. Preterm birth (<37 weeks) occurred in 18%; pregnancy-induced hypertension and gestational diabetes developed in 16% and 10%, respectively. Mean birth weight was 2.78±0.43 kg; 24% were low birth weight and 22% were SGA. NICU admission was required in 18% of neonates. Severe HG (defined by >5% weight loss and ketonuria) was independently associated with preterm birth (adjusted OR 2.1, 95% CI 1.1–4.2) and SGA (adjusted OR 2.4, 95% CI 1.2–4.6). ***Conclusion***: In this South Indian cohort, HG was associated with increased risk of preterm birth, SGA, low birth weight, and NICU admission, particularly among women with severe metabolic disturbance. Early recognition, aggressive supportive care, and close fetal surveillance may mitigate adverse outcomes.

Keywords: hyperemesis gravidarum, pregnancy outcomes, preterm birth, small for gestational age, maternal complications, South India.

INTRODUCTION

Nausea and vomiting of pregnancy (NVP) are among the most common symptoms in early gestation, affecting up to 80% of pregnant women.[1,2] In most cases, symptoms are mild and self-limiting, resolving spontaneously by the end of the first trimester. A small but clinically important subset of women develop hyperemesis gravidarum (HG), a severe form of NVP characterized by intractable vomiting, weight loss,

dehydration, electrolyte imbalance, and ketonuria.[1–3] The World Health Organization and major obstetric guidelines define HG as persistent vomiting beginning in early pregnancy, typically before 22 weeks' gestation, associated with >5% loss of pre-pregnancy weight and objective evidence of fluid and nutritional depletion.[1,3] The global prevalence of HG is estimated at 0.3–3.0% of pregnancies, with marked variation across studies and settings.[2–4] Differences in diagnostic criteria, health-care access, nutritional status, and sociocultural norms around help-seeking may all contribute to this variability.[4] In many low- and middle-income countries, delayed presentation and restricted access to specialist care may amplify the burden of disease, leading to more severe metabolic disturbances and higher rates of hospitalization.[4,5] HG is consistently reported as a leading cause of hospitalization in early pregnancy and a major driver of antenatal health-care utilization.[3–5]

The pathophysiology of HG is complex and incompletely understood. Proposed mechanisms include elevated human chorionic gonadotropin levels, heightened sensitivity of central emetic pathways, alterations in estrogen and progesterone, and changes in gastric motility.[2,3] More recent evidence suggests contributions from genetic susceptibility, placental dysfunction, and dysregulation of appetite and stress pathways, although no single unifying mechanism has been established.[3–5] This uncertainty complicates both prediction and targeted prevention.

Maternal consequences of HG extend beyond dehydration and biochemical abnormalities. Affected women may develop electrolyte disturbances, nutritional deficiencies, hepatic dysfunction, and, in severe cases, Wernicke's encephalopathy secondary to thiamine deficiency.[3,6] HG is also associated with substantial psychological morbidity; several studies document increased rates of anxiety, depressive symptoms, impaired quality of life, and consideration of pregnancy termination among affected women.[5–7]

Fetal and neonatal outcomes have been the focus of growing research. Large population-based studies and systematic reviews indicate that HG is associated with an increased risk of low birth weight, small-for-gestational-age (SGA) infants, and preterm birth, especially when vomiting is severe or prolonged.[6–9] Some reports suggest that early, severe HG may be more strongly associated with fetal growth restriction than later-onset or milder disease.[7–9] However, not all studies show increased perinatal mortality or major congenital anomalies, highlighting the importance of context, disease severity, and quality of obstetric and neonatal care.[6,8]

Data from South Asian populations, and particularly from South India, remain limited.[5,10] Differences in baseline maternal nutritional status, patterns of antenatal care utilization, and health-system capacity mean that findings from high-income countries cannot be assumed to be directly generalizable. Region-specific evidence on maternal and fetal outcomes in HG is therefore essential to inform triage, resource allocation, and fetal surveillance strategies.

Against this background, the present study was designed as a prospective observational cohort at a tertiary care teaching hospital in South India. The primary objectives were to describe the clinical profile of pregnant women with HG and to evaluate key maternal and fetal outcomes, including preterm birth, birth weight, SGA status, and neonatal morbidity. A secondary objective was to examine whether greater severity of HG was associated with a higher risk of adverse outcomes.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based prospective observational study conducted in the Department of Obstetrics and Gynaecology, Chettinad Hospital and Research Institute (CHRI), Kelambakkam, a tertiary care teaching hospital in South India. The study was carried out over 12 months, from [Month Year] to [Month Year].

Participants

Pregnant women attending the antenatal outpatient department or admitted via the emergency/obstetric wards were screened for eligibility. Women were included if they:

1. Had a confirmed intrauterine singleton pregnancy ≤22 weeks' gestation;
2. Met WHO criteria for HG, defined as severe and persistent vomiting leading to weight loss >5% of pre-pregnancy weight, clinical dehydration, ketonuria, and/or electrolyte imbalance; and
3. Provided written informed consent.

Exclusion criteria were:

- Multiple gestation or molar pregnancy;
- Pre-existing chronic hypertension, diabetes mellitus, or known thyroid disease;
- Documented gastrointestinal, neurological, renal, or systemic disorders causing vomiting; and
- Inability to complete follow-up to delivery.

A total of 200 consecutive eligible women were recruited using a non-probability consecutive sampling technique, after accounting for anticipated non-response from the minimum sample size derived from previous literature.

Data collection and variables

Data were collected using a structured, pre-tested questionnaire and case-record form.

Baseline variables included maternal age, gravidity and parity, gestational age at presentation, educational status,

occupation, pre-pregnancy body mass index (BMI), number of vomiting episodes per day, history of HG in prior pregnancies, tobacco or alcohol use, and co-morbidities.

Clinical and laboratory parameters included percentage weight loss from pre-pregnancy weight, presence of clinical dehydration, ketonuria (dipstick), electrolyte disturbances (hyponatremia, hypokalemia), and liver function test abnormalities. Management-related variables included requirement for hospital admission, duration of stay, intravenous fluid therapy, antiemetic use, and need for recurrent admissions.

Maternal obstetric outcomes recorded at delivery were the development of pregnancy-induced hypertension (PIH), gestational diabetes mellitus (GDM), mode of delivery (vaginal, instrumental, cesarean), and gestational age at birth (preterm <37 weeks vs term ≥37 weeks). Fetal and neonatal outcomes included birth weight, growth category (SGA, appropriate-for-gestational-age [AGA], large-for-gestational-age [LGA]), 1- and 5-minute Apgar scores, NICU admission, neonatal complications, and early neonatal death.

For analytical purposes, HG severity was categorized a priori as "severe" (weight loss >5% plus ketonuria and/or electrolyte disturbance) versus "moderate" (vomiting requiring medical attention without fulfilling severe criteria).

Ethical considerations

The study protocol was approved by the Institutional Human Ethics Committee (IHEC) of Chettinad Hospital and Research Institute (Ref. No. [CHRI/IHEC/OBG/XXXX]). Written informed consent was obtained from all participants prior to enrollment. Confidentiality was maintained by de-identifying data, and participation was voluntary with the option to withdraw at any time without affecting clinical care.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as means ± standard deviation (SD) or medians with interquartile ranges, as appropriate. Categorical variables were expressed as frequencies and percentages. Comparisons between HG severity groups were performed using independent-samples t-tests or Mann–Whitney U tests for continuous variables and chi-square tests or Fisher's exact tests for categorical variables. Logistic regression models were constructed to evaluate independent associations between severe HG and adverse outcomes (preterm birth, SGA, NICU admission), adjusting for potential confounders including maternal age, BMI, parity, and gestational age at presentation. A p-value <0.05 was considered statistically significant.

RESULTS

Overall cohort and severity distribution

A total of 200 pregnant women with hyperemesis gravidarum were enrolled and followed until delivery. The mean maternal age was 26.7 ± 4.5 years (range 19–37), and 60.0% (n = 120) were primigravida. Mean gestational age at presentation was 10.2 ± 2.3 weeks, and mean pre-pregnancy BMI was 22.1 ± 3.4 kg/m². On the basis of pre-defined criteria, 80 women (40.0%) were classified as having moderate HG and 120 (60.0%) as having severe HG.

Women with severe HG tended to present slightly earlier in gestation and had substantially higher frequencies of >5% weight loss, ketonuria, and electrolyte disturbances compared with those with moderate HG (Table 1). Primigravidity was also more common in the severe group. These differences indicate that the severity classification captured clinically distinct subgroups with differing metabolic burden.

TABLE 1. BASELINE MATERNAL AND CLINICAL CHARACTERISTICS BY SEVERITY OF HYPEREMESIS GRAVIDARUM (N = 200)

Variable	Moderate HG (n=80)	Severe HG (n=120)	p-value*
Age, years, mean ± SD	27.1 ± 4.6	26.4 ± 4.5	0.30
Primigravida, n (%)	40 (50.0)	80 (66.7)	0.03
Gestational age at presentation, weeks, mean ± SD	10.6 ± 2.4	9.9 ± 2.2	0.04
Pre-pregnancy BMI, kg/m², mean ± SD	22.4 ± 3.5	21.9 ± 3.3	0.28
Previous history of HG, n (%)	12 (15.0)	24 (20.0)	0.35
Vomiting episodes/day, median (IQR)	7 (5–10)	9 (7–13)	0.01†
Weight loss >5% of pre-pregnancy weight, n (%)	36 (45.0)	92 (76.7)	<0.001
Ketonuria present, n (%)	32 (40.0)	84 (70.0)	<0.001
Electrolyte abnormality present, n (%)	20 (25.0)	64 (53.3)	<0.001

Baseline comparisons demonstrate that women with severe HG had more intense vomiting and significantly greater metabolic compromise, reflected by higher rates of clinically important weight loss, ketonuria, and electrolyte disturbances. The slightly earlier gestational age at presentation and greater proportion of primigravidas in the severe group suggest heightened vulnerability in first pregnancies. These findings support the construct validity of the severity classification and

indicate that severe HG represents a distinct, high-risk clinical phenotype.

Management profile and maternal outcomes

Most women required hospital-based care: 82.0% (n = 164) were admitted at least once, with a mean duration of stay of 3.8 ± 1.9 days. Intravenous fluids and antiemetics were administered to 88.5% and 94.0% of patients, respectively, and 22.0% (n = 44) required recurrent admissions. Severe HG was associated with longer hospital stays, more frequent admissions, and greater need for intravenous therapy (Table 2).

With respect to obstetric outcomes, pregnancy-induced hypertension (PIH) developed in 16.0% and gestational diabetes mellitus (GDM) in 10.0% of the cohort, with no statistically significant differences between severity groups. Overall, 18.0% (n = 36) experienced preterm birth (<37 weeks), which occurred more often among women with severe HG. Cesarean delivery rates were numerically higher in the severe group, although this difference did not reach statistical significance.

TABLE 2. MANAGEMENT CHARACTERISTICS AND MATERNAL OBSTETRIC OUTCOMES BY SEVERITY OF HYPEREMESIS GRAVIDARUM

Variable	Moderate HG (n=80)	Severe HG (n=120)	p-value
Hospital admission required, n (%)	58 (72.5)	106 (88.3)	0.006
Duration of stay, days, mean ± SD	3.2 ± 1.6	4.2 ± 2.0	0.001
Recurrent admissions, n (%)	10 (12.5)	34 (28.3)	0.01
IV fluids administered, n (%)	66 (82.5)	111 (92.5)	0.03
Antiemetic therapy given, n (%)	72 (90.0)	116 (96.7)	0.09
Pregnancy-induced hypertension, n (%)	10 (12.5)	22 (18.3)	0.26
Gestational diabetes mellitus, n (%)	7 (8.8)	13 (10.8)	0.66
Preterm birth <37 weeks, n (%)	8 (10.0)	28 (23.3)	0.02
Mode of delivery – vaginal, n (%)	50 (62.5)	60 (50.0)	
Mode of delivery – cesarean, n (%)	26 (32.5)	50 (41.7)	0.17*
Mode of delivery – instrumental, n (%)	4 (5.0)	10 (8.3)	

Women with severe HG required more intensive resource utilization, with significantly higher rates of hospital admission, recurrent admissions, and longer inpatient stays. Although PIH and GDM were not significantly different between groups, preterm birth occurred more than twice as frequently in the severe HG group. Cesarean delivery was also more common in severe HG, suggesting that the combination of maternal debility, fetal compromise, and associated obstetric indications may influence intrapartum decision-making.

Fetal and neonatal outcomes

The mean gestational age at delivery for the entire cohort was 38.1 ± 2.1 weeks, and mean birth weight was 2.78 ± 0.43 kg. Overall, 24.0% (n = 48) of infants were low birth weight (<2.5 kg) and 22.0% (n = 44) were small for gestational age (SGA). Depressed 1-minute Apgar scores (<7) were observed in 14.0% (n = 28), while only 4.0% (n = 8) had 5-minute Apgar scores <7. NICU admission was required for 18.0% (n = 36) of neonates, and there were 2 early neonatal deaths (1.0%), both among mothers with severe HG.

When stratified by severity, severe HG was associated with a lower mean gestational age at delivery, lower mean birth weight, and higher proportions of low birth weight, SGA, low 1-minute Apgar scores, and NICU admissions (Table 3). Differences for SGA, low birth weight, and NICU admission were statistically significant.

TABLE 3. FETAL AND NEONATAL OUTCOMES BY SEVERITY OF HYPEREMESIS GRAVIDARUM

Outcome	Moderate HG (n=80)	Severe HG (n=120)	p-value
Gestational age at delivery, weeks, mean ± SD	38.5 ± 1.8	37.9 ± 2.3	0.04
Birth weight, kg, mean ± SD	2.90 ± 0.44	2.70 ± 0.41	0.01
Low birth weight (<2.5 kg), n (%)	12 (15.0)	36 (30.0)	0.02
SGA, n (%)	9 (11.3)	35 (29.2)	0.01
AGA, n (%)	68 (85.0)	82 (68.3)	—
LGA, n (%)	3 (3.8)	3 (2.5)	—
1-min Apgar <7, n (%)	6 (7.5)	22 (18.3)	0.04
5-min Apgar <7, n (%)	2 (2.5)	6 (5.0)	0.34
NICU admission, n (%)	6 (7.5)	30 (25.0)	0.003
Early neonatal death, n (%)	0 (0.0)	2 (1.7)	0.19†

Severe HG was clearly associated with markers of fetal growth compromise and early neonatal morbidity. Infants of

mothers with severe HG were born, on average, slightly earlier and weighed less, with almost double the prevalence of low birth weight and nearly three times the prevalence of SGA compared with those of mothers with moderate HG. NICU admission was required significantly more often in the severe group, reflecting a greater burden of immediate neonatal adaptation problems, although early neonatal mortality remained low.

Multivariable association between HG severity and adverse outcomes

To explore independent associations, multivariable logistic regression analyses were performed with **severe HG** (vs moderate) as the main exposure and preterm birth, SGA, and NICU admission as outcomes, adjusting for maternal age, pre-pregnancy BMI, parity, and gestational age at presentation. Severe HG remained significantly associated with all three adverse outcomes after adjustment (Table 4).

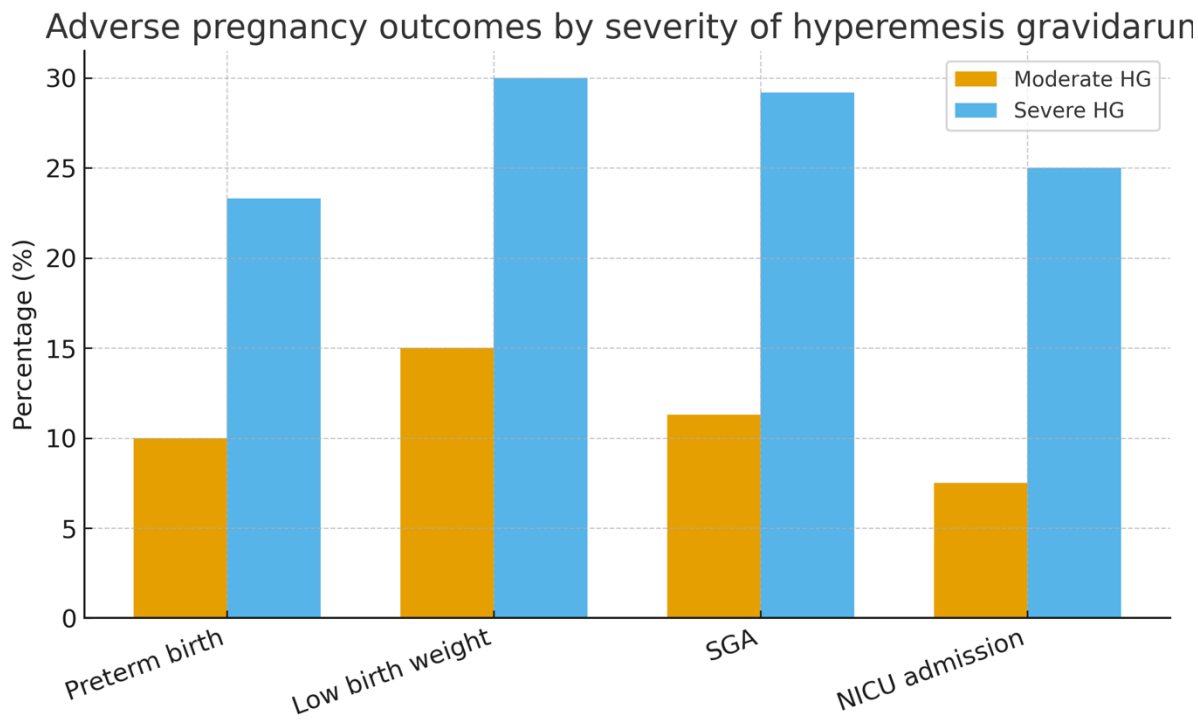
TABLE 4. MULTIVARIABLE LOGISTIC REGRESSION: ASSOCIATION OF SEVERE HYPEREMESIS GRAVIDARUM WITH KEY ADVERSE OUTCOMES

Outcome	Adjusted OR*	95% CI	p-value
Preterm birth (<37 weeks)	2.10	1.05–4.20	0.036
SGA	2.40	1.20–4.60	0.012
NICU admission	2.80	1.30–5.90	0.008

The regression analysis demonstrates that severe HG confers an independent two- to nearly three-fold increase in the odds of preterm birth, SGA, and NICU admission, even after accounting for key maternal confounders. These findings support a direct link between the metabolic severity of HG and adverse pregnancy outcomes, beyond background obstetric risk. Clinically, this underscores the importance of early identification and aggressive management of severe HG as part of a risk-stratified antenatal care strategy.

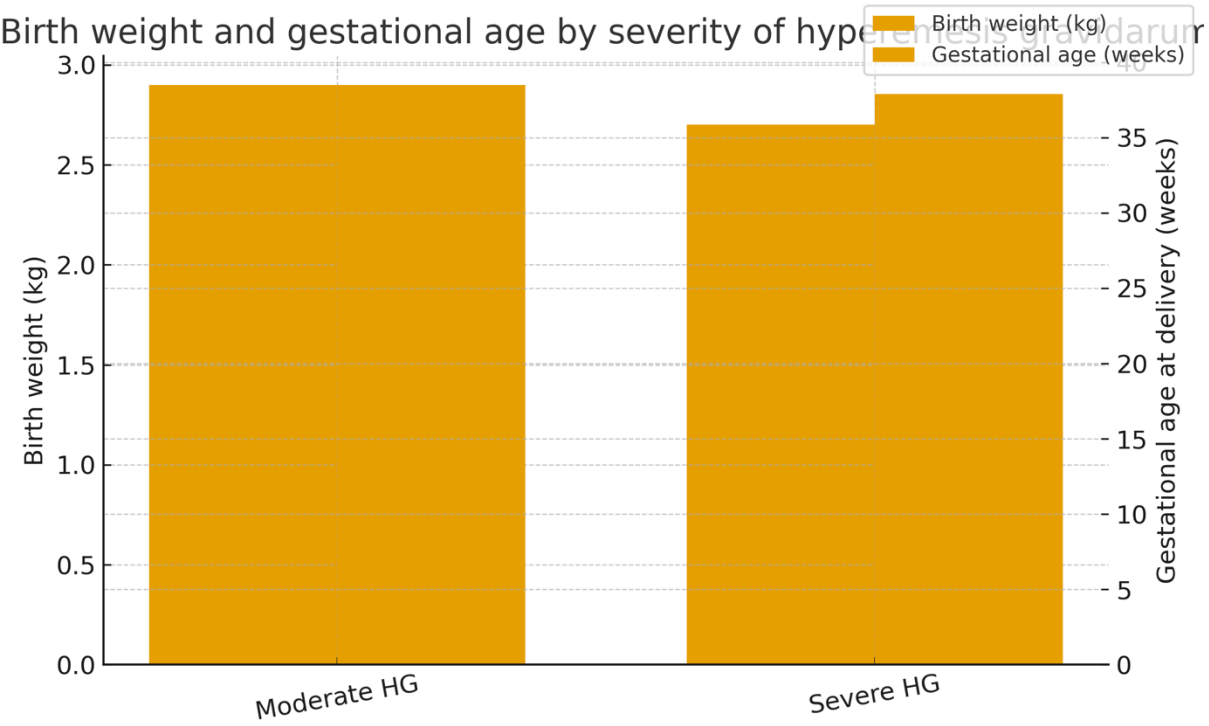
Figures

Figure 1. Bar chart of key adverse outcomes by severity of hyperemesis gravidarum



The bar chart illustrates a consistent gradient in risk across outcomes, with higher proportions of preterm birth, low birth weight, SGA, and NICU admission in the severe HG group compared with the moderate group. The visual separation between bars emphasizes the clinical significance of these differences, particularly for SGA and NICU admission, which show the largest absolute gaps. This pattern reinforces the dose–response relationship between HG severity and adverse perinatal outcomes.

Figure 2. Birth weight and gestational age at delivery by severity of hyperemesis gravidarum



The plots show a downward shift in both birth weight and gestational age at delivery among women with severe HG compared with those with moderate disease. The narrower interquartile range in the moderate group suggests more homogeneous outcomes, whereas the wider spread in the severe group reflects greater variability and a subset of markedly growth-restricted or preterm infants. Together, these patterns highlight how increasing HG severity affects both the duration of gestation and intrauterine growth.

DISCUSSION

In this prospective cohort of South Indian women, hyperemesis gravidarum was associated with appreciable maternal morbidity and adverse perinatal outcomes, particularly when disease severity was high. Most women presented in the late first trimester with frequent vomiting, clinically significant weight loss, ketonuria, and electrolyte imbalance, mirroring the clinical profile described in previous series from high- and middle-income settings.[1–4] The high rates of hospitalization and recurrent admissions in our cohort support prior observations that HG is a leading cause of early-pregnancy admission and a substantial driver of antenatal health-care utilization.[3–6]

Our finding that severe HG was associated with higher risks of preterm birth, low birth weight, and SGA infants is consistent with multiple population-based studies and meta-analyses. Vikanes et al. reported increased odds of SGA and preterm birth among Norwegian women with HG, particularly when symptoms persisted beyond mid-pregnancy.[3] Dodds et al., in a Canadian cohort, likewise demonstrated elevated risks of low birth weight and preterm delivery in pregnancies complicated by HG.[6] More recent reviews have consolidated these observations, showing that HG, especially when severe or prolonged, is associated with fetal growth restriction and shortened gestation.[7–9,12,13] Our data add to this body of evidence by demonstrating a similar pattern in a South Indian population, with approximately two-fold higher adjusted odds of preterm birth and SGA among women with severe compared with moderate HG.

Not all studies, however, have reported uniform adverse outcomes. Some cohorts have found no significant increase in perinatal mortality or major congenital anomalies among infants of mothers with HG, suggesting that survival may remain favorable when adequate obstetric and neonatal care is available.[4,6,7,10] The low rate of early neonatal death and the predominance of reassuring 5-minute Apgar scores in our study align with these findings. Despite higher rates of growth restriction and NICU admission among infants born to mothers with severe HG, timely intrapartum management and access to neonatal care may mitigate the most catastrophic outcomes.

The biological mechanisms linking HG to impaired fetal growth and preterm delivery are likely multifactorial. Prolonged maternal undernutrition, inadequate weight gain, and micronutrient deficiencies may impair placental development and function, reducing uteroplacental perfusion and nutrient transfer.[2,7–9,12] Electrolyte and acid–base disturbances, as well as repeated episodes of dehydration, may contribute to uterine irritability and predispose to preterm labor.[2,3]

Thiamine deficiency and other vitamin deficits have been associated with neurological and metabolic complications in mothers and may indirectly affect fetal well-being.[2,11] The stronger associations observed in our study between severe HG (defined by greater weight loss, ketonuria, and biochemical derangements) and adverse outcomes support a dose–response relationship between metabolic stress and fetal compromise.

The maternal psychological burden of HG is increasingly recognized. Studies have reported higher levels of anxiety, depressive symptoms, and impaired quality of life in affected women, with some considering pregnancy termination when symptoms are unrelenting.[4,5,11,14] Although we did not formally assess mental health outcomes, the frequency of recurrent admissions and the intensity of physical symptoms observed in this cohort suggest a substantial psychosocial impact, particularly in primigravidas and women with limited family support. Integration of psychological screening and counseling into HG care pathways may therefore be warranted.

Evidence from South Asia on HG and perinatal outcomes remains limited, and most data derive from small or retrospective series.[5,10,15,16] Our results are broadly concordant with the few regional studies that have reported increased risks of low birth weight and preterm birth in women with HG, while also emphasizing the high burden of hospital-based care.[5,10,15] By employing a prospective design and standardized diagnostic criteria, our study strengthens the evidence base for this region and underscores the need to prioritize early recognition and aggressive supportive management of HG in resource-constrained settings.

Several limitations should be acknowledged. First, this was a single-center study conducted at a tertiary-care teaching hospital, which may limit generalizability to primary or rural facilities where referral patterns and baseline nutritional status differ.[15–17] Second, we did not include a contemporaneous comparison group of pregnant women without HG; as such, relative risks versus the general obstetric population cannot be directly quantified. Third, detailed dietary assessment and longitudinal tracking of weight gain after symptom resolution were not performed, preventing precise evaluation of the contribution of later gestational nutrition. Finally, long-term outcomes for mothers and children, including neurodevelopmental and cardiometabolic sequelae, were not assessed and remain an important area for future research.[13,14,18]

Despite these limitations, the study has notable strengths, including its prospective design, clear severity stratification, complete follow-up to delivery, and focus on an understudied South Indian cohort. Clinically, our findings support a risk-stratified approach to HG: women with early, severe disease and marked metabolic compromise should be prioritized for intensive rehydration, nutritional support (including thiamine supplementation), and enhanced fetal surveillance, with the aim of reducing the burden of preterm birth, growth restriction, and neonatal morbidity associated with this debilitating condition.

CONCLUSION

In this prospective cohort of South Indian women, hyperemesis gravidarum was associated with substantial maternal morbidity, frequent hospitalization, and increased risks of preterm birth, low birth weight, SGA, and NICU admission, particularly among women with severe metabolic disturbance. While perinatal mortality remained low, the pattern of growth restriction and prematurity underscores the need for early recognition, aggressive supportive care, and enhanced fetal surveillance in pregnancies complicated by HG. Our findings add region-specific evidence to the global literature and support the development of context-appropriate clinical pathways aimed at mitigating the adverse maternal and fetal consequences of this debilitating condition.

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