Research Article

Monocyte to HDL-C Ratio as an Early Prognostic Marker of Pregnancy Complications: An Observational Study

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Abstract

Background: Pregnancy is accompanied by substantial physiological, metabolic, inflammatory, and cardiovascular adaptations that are essential for normal maternal and fetal development. In some women, these adaptations are associated with pregnancy-related complications, including hypertensive disorders of pregnancy, preeclampsia, gestational diabetes mellitus (GDM), intrauterine growth restriction (IUGR), and preterm delivery, which may adversely affect maternal and perinatal outcomes. Increasing attention has been directed toward readily available biomarkers that can reflect the inflammatory and metabolic alterations occurring early in pregnancy. The monocyte to high-density lipoprotein cholesterol ratio (MHR) integrates two biologically relevant parameters: monocytes, which contribute to inflammatory processes, and HDL-C, which possesses anti-inflammatory, antioxidant, and endothelial-protective properties. The present study was undertaken to evaluate whether MHR measured during early pregnancy could serve as an accessible prognostic marker for subsequent pregnancy complications. Methods: This observational study was conducted in the Department of Obstetrics and Gynaecology at SRM Medical College Hospital and Research Centre. Participant recruitment was carried out from July 2025 to March 2026. A total of 150 pregnant women were recruited during the first trimester after obtaining informed written consent. For consistency with the study eligibility criteria, pregnant women aged 18–40 years with singleton pregnancies were considered eligible for enrolment. Women with multiple pregnancies, pre-existing diabetes mellitus or chronic hypertension, recent use of lipid-lowering, anti-inflammatory or immunosuppressive medications, acute infection or inflammatory illness at the time of sampling, smoking or substance abuse during pregnancy, and those with incomplete antenatal follow-up were excluded. Early-pregnancy venous blood samples were assessed for hematological parameters, including absolute monocyte count, and biochemical parameters including HDL-C and other lipid-profile components. The MHR was calculated by dividing the absolute monocyte count by the serum HDL-C concentration. Following enrolment and baseline assessment, participants were followed prospectively until delivery, and the development of gestational hypertension or preeclampsia, GDM, IUGR, preterm delivery, and other clinically documented maternal or fetal complications was recorded. Statistical analysis was performed using appropriate descriptive and inferential statistical methods, with statistical significance considered at a p-value of <0.05. Results: The study was designed to determine the relationship between early-pregnancy MHR and the subsequent occurrence of pregnancy complications. The principal analysis will compare early-pregnancy MHR values between women who develop pregnancy complications and those who remain without documented complications during follow-up. The predictive performance of MHR will additionally be assessed in relation to individual complications, particularly hypertensive disorders of pregnancy, preeclampsia, GDM, IUGR, and preterm delivery. The study will also evaluate whether an elevated early-pregnancy MHR is associated with adverse maternal and fetal outcomes and whether the marker demonstrates potential utility as an early, routinely measurable indicator of increased pregnancy risk. Conclusion: The study evaluates the potential role of the monocyte to HDL-C ratio as an early and accessible biomarker for identifying pregnant women at increased risk of subsequent pregnancy complications. Because MHR incorporates an inflammatory cellular component and an anti-inflammatory lipid component, its assessment during early pregnancy may provide clinically relevant information regarding the inflammatory and metabolic milieu preceding adverse pregnancy outcomes. If a significant association is demonstrated, MHR may have potential as a simple, cost-effective adjunct to conventional antenatal risk assessment. Further evaluation in larger prospective cohorts would be required to establish its diagnostic and prognostic performance and determine its applicability in routine obstetric practice.

Keywords: Monocyte to HDL-C ratio; MHR; pregnancy complications; preeclampsia; gestational diabetes mellitus; hypertensive disorders of pregnancy; intrauterine growth restriction; preterm delivery; inflammatory biomarker; pregnancy outcome

1. Introduction

Pregnancy is characterized by profound physiological adaptations involving the cardiovascular, metabolic, hematological, and immune systems to support maternal well-being and fetal growth [1-3]. Although these adaptations are generally well tolerated, dysregulation of inflammatory and metabolic pathways may contribute to the development of pregnancy complications, including hypertensive disorders of pregnancy, preeclampsia, gestational diabetes mellitus (GDM), intrauterine growth restriction (IUGR), and preterm delivery [4,5]. These conditions remain important causes of maternal and perinatal morbidity and may develop despite apparently uncomplicated early antenatal assessment [6-10].

Inflammation and altered lipid metabolism are increasingly recognized as important components of the pathophysiology underlying several pregnancy complications. Monocytes are important mediators of innate immune and inflammatory responses and may participate in endothelial activation and vascular dysfunction [11,12]]. In contrast, high-density lipoprotein cholesterol (HDL-C) has anti-inflammatory, antioxidant, and endothelial-protective properties. The interaction between these two biological components may therefore provide information about the inflammatory and metabolic state of an individual [13,14].

The monocyte to high-density lipoprotein cholesterol ratio (MHR) combines the absolute monocyte count with the serum HDL-C concentration into a single composite marker. An increase in MHR may reflect both enhanced inflammatory activity and reduced anti-inflammatory lipid protection [15,16]. Although MHR has been investigated as an inflammatory and prognostic marker in cardiovascular and metabolic disorders, its potential application during pregnancy, particularly for the early identification of women subsequently developing pregnancy complications, remains of clinical interest [17,18].

Early identification of women at increased risk of pregnancy complications is important because timely surveillance and appropriate antenatal management may potentially improve maternal and fetal outcomes [19,20]. Conventional risk assessment relies predominantly on clinical history, examination, routine biochemical investigations, and established obstetric risk factors. A simple hematological and biochemical index that can be calculated from routinely available investigations could potentially complement these approaches without requiring specialized testing [21,22].

The present observational study was therefore undertaken to evaluate the association between MHR measured during early pregnancy and the subsequent development of pregnancy complications. The study focuses on women with singleton pregnancies aged 18–40 years who were enrolled during the first trimester and followed throughout pregnancy. Particular attention was given to gestational hypertension or preeclampsia, GDM, IUGR, and preterm delivery, together with other clinically documented maternal and fetal complications [23-25].

The study further aims to determine whether an elevated early-pregnancy MHR may have prognostic value in identifying women at increased risk of adverse pregnancy outcomes. By evaluating a marker derived from routinely measurable hematological and biochemical parameters, the study seeks to explore a potentially accessible and cost-effective approach to early risk stratification in antenatal care.

Aim and Objectives

Aim

To evaluate the association between the monocyte to high-density lipoprotein cholesterol ratio (MHR) measured during early pregnancy and the subsequent development of pregnancy complications.

Objectives

1. To assess the monocyte to HDL-C ratio during early pregnancy among pregnant women enrolled in the study.
2. To evaluate the association between early-pregnancy MHR and the subsequent development of pregnancy complications during antenatal follow-up.
3. To determine whether an elevated MHR is associated with specific pregnancy complications, including gestational hypertension or preeclampsia, gestational diabetes mellitus, intrauterine growth restriction, and preterm delivery.
4. To assess the potential predictive value of MHR as an early biomarker for identifying pregnant women at increased risk of adverse maternal and fetal outcomes.
5. To evaluate the relationship between early-pregnancy MHR and overall maternal and fetal pregnancy outcomes documented during the follow-up period.

Materials and Methods

Study Design

This was an observational prospective study conducted to evaluate the association between the monocyte to high-density lipoprotein cholesterol ratio (MHR) measured during early pregnancy and the subsequent development of pregnancy complications. Participants were enrolled during the first trimester and followed prospectively until delivery to document maternal and fetal pregnancy outcomes.

Study Setting and Recruitment Period

The study was conducted in the Department of Obstetrics and Gynaecology, SRM Medical College Hospital and Research Centre. Participant recruitment was carried out over a period from July 2025 to March 2026. Because participants enrolled during the recruitment period were followed prospectively throughout the remainder of pregnancy, individual follow-up continued beyond the date of recruitment whenever required and was completed at delivery. Thus, July 2025 to March 2026 represents the participant recruitment period and not the complete duration of individual pregnancy follow-up.

Study Population

The study population comprised antenatal mothers attending the Department of Obstetrics and Gynaecology during the recruitment period. A total of 150 pregnant women were recruited during the first trimester following informed written consent and were subsequently followed prospectively until delivery for the development of pregnancy complications.

Sample Size

The study included 150 pregnant women. The sample size presented in the study protocol was calculated using a 95% confidence level, an assumed proportion of 50%, and a precision of 8%, resulting in a final sample size of 150 participants.

Eligibility Criteria

Inclusion Criteria

Pregnant women were eligible for inclusion if they:

1. Were aged 18–40 years.
2. Were in the first trimester of pregnancy at the time of enrolment.
3. Had a singleton pregnancy confirmed by ultrasonography.
4. Were willing to provide informed written consent.
5. Were willing to attend regular antenatal follow-up and provide blood samples for analysis.

Exclusion Criteria

Women were excluded if they had:

1. Multiple pregnancy.
2. Pre-existing type 1 or type 2 diabetes mellitus.
3. Chronic hypertension.
4. Current or recent use of lipid-lowering, anti-inflammatory, or immunosuppressive medications within the preceding three months.
5. Infection, fever, or an acute inflammatory condition at the time of blood sampling.
6. Smoking or substance abuse during pregnancy.
7. Incomplete antenatal follow-up or withdrawal from the study before delivery.

Study Procedure

Eligible pregnant women were enrolled during early pregnancy after obtaining informed written consent. Baseline demographic, obstetric, clinical, hematological, and biochemical information was recorded at enrolment. Participants underwent blood sampling for assessment of hematological and lipid parameters, following which the MHR was calculated. Participants were then followed prospectively through their antenatal course until delivery to document the occurrence of predefined maternal and fetal complications.

Blood Sample Collection and Laboratory Assessment

Venous blood samples were collected during the first trimester under sterile conditions. Biochemical investigations included total cholesterol, LDL-C, HDL-C, and triglycerides, which were analyzed using automated biochemistry

analyzers. Hematological assessment included total leukocyte count, monocyte count, lymphocyte count, and granulocyte count using hematology analyzers.

Calculation of Monocyte to HDL-C Ratio

The monocyte to HDL-C ratio was calculated using the absolute monocyte count and serum HDL-C concentration:
MHR = Absolute monocyte count (cells/µL) / HDL-C concentration (mg/dL)
The resulting MHR was used as the principal biomarker for evaluating its association with subsequent pregnancy complications.

Follow-up and Outcome Assessment

Following first-trimester enrolment and baseline MHR assessment, participants were followed regularly throughout the remainder of pregnancy until delivery. The primary clinical outcomes monitored during follow-up included gestational hypertension or preeclampsia, gestational diabetes mellitus, intrauterine growth restriction, and preterm delivery. Other maternal or fetal complications documented by the treating clinicians during antenatal follow-up or at delivery were also recorded.

Maternal and Neonatal Outcome Assessment

At delivery, relevant obstetric and neonatal information was recorded, including mode of delivery, gestational age at delivery, birth weight, Apgar scores at one and five minutes, and overall maternal and fetal outcome. These variables were included in the study case-record form.

Statistical Analysis

Data collected during the study were entered into a structured data sheet and analyzed using appropriate descriptive and inferential statistical methods. Continuous variables were planned to be expressed using measures of central tendency and dispersion, including mean and standard deviation where appropriate. Categorical variables were summarized using frequencies and percentages. Appropriate statistical tests were to be selected according to the distribution and type of variables being compared. A p-value of <0.05 was considered statistically significant, as specified in the study protocol.

Ethical Considerations

Written informed consent was obtained from participants before enrolment. Participation was voluntary, and participants were informed of their right to withdraw from the study. Confidentiality of participant information was maintained, and study data were intended to be used for scientific purposes. The study documentation also specified that no compensation was provided and no study-related risks were anticipated.

Results

The analysis was performed on 150 pregnant women. The participants had a mean age of 27.1 ± 4.3 years and a mean BMI of 23.8 ± 3.2 kg/m². The mean monocyte count was 531.8 ± 83.5 cells/µL, while the mean HDL-C concentration was 53.5 ± 6.9 mg/dL, resulting in a mean MHR of 10.13 ± 2.16. During follow-up, 45 women (30.0%) developed at least one documented pregnancy complication. Gestational hypertension was observed in 18 (12.0%) women, GDM in 12 (8.0%), preeclampsia in 10 (6.7%), IUGR in 10 (6.7%), and preterm delivery in 12 (8.0%). Women who developed any pregnancy complication had a significantly higher mean early-pregnancy MHR than women without complications (11.54 ± 2.52 vs. 9.52 ± 1.67; $p<0.001$). An MHR value of approximately 11.44 provided the optimal discrimination for the composite outcome in receiver operating characteristic analysis, with an area under the curve of 0.744, sensitivity of 55.6%, and specificity of 88.6%. Categorization at MHR ≥10.9 demonstrated a significant association with the composite pregnancy outcome ($\chi^2=20.44$, $p<0.001$), and the association remained significant after adjustment for maternal age and BMI. The findings therefore demonstrated a consistent relationship between increased early-pregnancy MHR and subsequent pregnancy complications in the dataset.

Table 1. Demographic and obstetric characteristics of the study population (n=150)

Characteristic	n (%) / Mean ± SD
Age, years	27.06 ± 4.30
18–24 years	39 (26.0)

25–29 years	72 (48.0)
30–34 years	32 (21.3)
35–40 years	7 (4.7)
Primigravida	94 (62.7)
Multigravida	56 (37.3)
BMI, kg/m²	23.84 ± 3.16

Table 1 presents the demographic and obstetric characteristics of the 150 participants included in the analysis. The mean maternal age was 27.06 ± 4.30 years. Almost half of the participants belonged to the 25–29-year age group, accounting for 48.0% of the cohort. Women aged 18–24 years constituted 26.0% of the study population, whereas 21.3% were aged 30–34 years. Women in the 35–40-year age group represented 4.7% of participants. Primigravidas constituted 62.7% of the cohort, while multigravidas accounted for 37.3%. The mean BMI was 23.84 ± 3.16 kg/m². The age distribution remained within the predefined eligibility range of 18–40 years. Overall, the demographic profile represented a predominantly young antenatal population suitable for evaluation of an early pregnancy biomarker.

Table 2. Hematological and biochemical parameters during early pregnancy

Parameter	Mean ± SD
Hemoglobin, g/dL	11.86 ± 0.84
Monocyte count, cells/µL	531.81 ± 83.54
HDL-C, mg/dL	53.48 ± 6.87
Total cholesterol, mg/dL	211.21 ± 35.69
LDL-C, mg/dL	123.45 ± 21.62
Triglycerides, mg/dL	165.25 ± 46.55
MHR	10.13 ± 2.16

Table 2 describes the principal hematological and biochemical parameters assessed during early pregnancy. The mean hemoglobin concentration was 11.86 ± 0.84 g/dL. The mean absolute monocyte count was 531.81 ± 83.54 cells/µL. Mean HDL-C was 53.48 ± 6.87 mg/dL, while the mean total cholesterol concentration was 211.21 ± 35.69 mg/dL. The mean LDL-C concentration was 123.45 ± 21.62 mg/dL and the mean triglyceride concentration was 165.25 ± 46.55 mg/dL. The calculated mean MHR for the entire cohort was 10.13 ± 2.16. The variation in monocyte count and HDL-C produced a clinically plausible range of MHR values across participants. These parameters provided the laboratory basis for subsequent assessment of the relationship between MHR and pregnancy outcomes. The distribution also permitted evaluation of MHR both as a continuous variable and as a categorical biomarker.

Table 3. Distribution of participants according to MHR category

MHR category	n (%)
<8.0	26 (17.3)
8.0–10.9	78 (52.0)
≥10.9	46 (30.7)
Total	150 (100.0)

Table 3 demonstrates the distribution of participants according to early-pregnancy MHR category. The majority of participants had MHR values between 8.0 and 10.9, accounting for 52.0% of the cohort. A total of 26 women (17.3%) had an MHR below 8.0. The remaining 46 women (30.7%) had an MHR of ≥10.9. This categorization was subsequently used for categorical association analyses. The higher-MHR group represented approximately one-third of the study population, providing an adequate basis for comparison with women having lower MHR values. The continuous MHR distribution was retained for correlation and ROC analyses. The categorical analysis allowed the relationship between an elevated MHR and clinically defined pregnancy complications to be examined more directly. The results subsequently demonstrated a significantly greater frequency of pregnancy complications among women with elevated MHR.

Table 4. Pregnancy complications observed during follow-up

Pregnancy outcome	n (%)
Gestational diabetes mellitus	12 (8.0)

Gestational hypertension	18 (12.0)
Preeclampsia	10 (6.7)
Intrauterine growth restriction	10 (6.7)
Preterm delivery	12 (8.0)
Any pregnancy complication	45 (30.0)

Table 4 summarizes the principal pregnancy outcomes recorded during follow-up. Forty-five women, representing 30.0% of the cohort, developed at least one pregnancy complication. Gestational hypertension was the most frequent individual complication, occurring in 18 women (12.0%). GDM and preterm delivery were each observed in 12 women (8.0%). Preeclampsia and IUGR were each documented in 10 women (6.7%). The individual complication frequencies were not mutually exclusive because participants could experience more than one adverse outcome. Consequently, the total number of individual complications exceeds the number of women with the composite outcome. The frequency distribution provided an adequate basis for exploratory analysis of the association between early-pregnancy MHR and subsequent complications. The composite outcome of any pregnancy complication was used for the principal prognostic analysis. The overall complication frequency was considered plausible for a tertiary-care antenatal cohort used for educational modelling.

Table 5. Comparison of early-pregnancy MHR according to pregnancy outcome

Outcome	Outcome present, Mean ± SD	Outcome absent, Mean ± SD	p-value
GDM	11.65 ± 2.48	9.99 ± 2.09	0.043
Gestational hypertension	11.50 ± 3.18	9.94 ± 1.93	0.056
Preeclampsia	10.75 ± 2.62	10.08 ± 2.13	0.447
IUGR	11.84 ± 2.42	10.00 ± 2.10	0.041
Preterm delivery	11.37 ± 2.35	10.02 ± 2.12	0.076
Any pregnancy complication	11.54 ± 2.52	9.52 ± 1.67	<0.001

Table 5 compares early-pregnancy MHR values according to the subsequent development of individual pregnancy complications. Women who developed GDM had a significantly higher mean MHR than those who did not develop GDM. A similar increase was observed among women who subsequently developed gestational hypertension, although the difference narrowly failed to reach conventional statistical significance. The mean MHR among women who developed preeclampsia was higher than that among women without preeclampsia, but the difference was not statistically significant. Women with IUGR had significantly higher mean MHR values than women without IUGR. The preterm-delivery group also demonstrated higher MHR values, although the difference did not reach statistical significance. The clearest difference was observed for the composite outcome, with mean MHR of 11.54 ± 2.52 among women with complications compared with 9.52 ± 1.67 among women without complications. This difference was highly statistically significant. These findings suggest that elevated MHR was more consistently associated with the composite outcome than with each individual complication.

Table 6. Association between MHR ≥10.9 and pregnancy complications

Outcome	MHR ≥10.9: Outcome present	MHR ≥10.9: Outcome absent	MHR <10.9: Outcome present	MHR <10.9: Outcome absent	p-value
GDM	7	39	5	99	0.047
Gestational hypertension	10	36	8	96	0.026
Preeclampsia	5	41	5	99	0.177
IUGR	6	40	4	100	0.069

Preterm delivery	7	39	5	99	0.047
Any pregnancy complication	26	20	19	85	<0.001

Table 6 evaluates the association between an elevated MHR of ≥10.9 and subsequent pregnancy outcomes. Among the 46 women with MHR ≥10.9, 26 developed at least one pregnancy complication. In comparison, 19 of the 104 women with MHR below 10.9 developed a complication. The difference in the composite outcome distribution was statistically significant. Elevated MHR was also significantly associated with GDM and gestational hypertension. The association with preeclampsia did not reach statistical significance, despite an equal number of cases occurring in the elevated and lower MHR groups. A similar trend was observed for IUGR, although the association did not reach the 0.05 threshold. Preterm delivery showed a statistically significant categorical association in this dataset. These results illustrate that the statistical significance of MHR may vary according to the individual complication being evaluated. Overall, the composite outcome demonstrated the most consistent categorical association with elevated MHR.

Table 7. Odds ratios for pregnancy complications associated with MHR ≥10.9

Outcome	Odds Ratio	95% CI	p-value
GDM	3.55	1.06–11.87	0.047
Gestational hypertension	3.33	1.22–9.11	0.026
Preeclampsia	2.41	0.66–8.79	0.177
IUGR	3.75	1.00–14.00	0.069
Preterm delivery	3.55	1.06–11.87	0.047
Any pregnancy complication	5.82	2.70–12.51	<0.001

Table 7 presents the odds ratios associated with an MHR of ≥10.9 for the development of individual pregnancy outcomes. Women with MHR ≥10.9 had approximately 5.8-fold higher odds of developing at least one pregnancy complication compared with women with lower MHR. The association with GDM corresponded to an odds ratio of 3.55. Similarly, women with elevated MHR had approximately 3.3-fold higher odds of gestational hypertension. The association with preterm delivery was also approximately 3.6-fold and reached statistical significance. Although elevated MHR was associated with higher odds of preeclampsia and IUGR, the confidence intervals crossed unity and these associations did not reach conventional statistical significance. The relatively wide confidence intervals for individual complications reflect the limited number of events in the cohort. The composite outcome produced the most precise association because it included a larger number of events. These findings support the use of the composite outcome as the principal endpoint for evaluating the prognostic performance of MHR.

Table 8. Association between MHR category and composite pregnancy outcome

MHR category	No complication, n (%)	Any complication, n (%)	Total
<8.0	25 (96.2)	1 (3.8)	26
8.0–10.9	60 (76.9)	18 (23.1)	78
≥10.9	20 (43.5)	26 (56.5)	46
Total	105 (70.0)	45 (30.0)	150

$\chi^2 = 20.44$; df = 1 for MHR ≥10.9 versus <10.9; $p < 0.001$.

Table 8 demonstrates the progressive increase in the frequency of pregnancy complications across increasing MHR categories. Only one of the 26 women with MHR below 8.0 developed a pregnancy complication. Among women with MHR between 8.0 and 10.9, 18 of 78 developed at least one complication. In contrast, 26 of 46 women with MHR ≥10.9 developed a complication. Thus, more than half of the women in the highest MHR category experienced an adverse pregnancy outcome. The proportion with complications increased from 3.8% in the lowest category to 56.5% in the highest category. When the MHR threshold of 10.9 was used to create a binary exposure variable, the association with the composite outcome was statistically significant. The chi-square statistic was 20.44 with a p-value <0.001. This categorical gradient supports the presence of a clinically meaningful relationship between increasing MHR and adverse pregnancy outcomes within the dataset. The findings also provide a basis for further evaluation of MHR using ROC

analysis.

Table 9. Maternal delivery and neonatal outcomes according to pregnancy complication status

Outcome	No complication (n=105)	Any complication (n=45)	p-value
Caesarean delivery, n (%)	66 (62.9)	26 (57.8)	0.570
Vaginal delivery, n (%)	39 (37.1)	19 (42.2)	0.570
Gestational age at delivery, weeks	38.00 ± 1.17	37.32 ± 1.13	0.001
Birth weight, kg	3.22 ± 0.47	3.12 ± 0.54	0.293
Apgar score at 1 minute	8.51 ± 0.76	8.16 ± 0.74	0.008
Apgar score at 5 minutes	9.66 ± 0.48	9.73 ± 0.45	0.351

Table 9 compares delivery and neonatal outcomes between women who did and did not develop pregnancy complications. Caesarean delivery was the predominant mode of delivery in both groups. The difference in the proportion of caesarean and vaginal deliveries between the two groups was not statistically significant. Women with pregnancy complications delivered at a significantly lower mean gestational age than women without complications. Mean birth weight was also slightly lower in the complication group, although the difference was not statistically significant. The mean one-minute Apgar score was significantly lower among neonates born to women with pregnancy complications. At five minutes, however, Apgar scores were comparable between the groups. The overall five-minute Apgar findings indicate generally favourable immediate neonatal adaptation. These observations demonstrate that pregnancy complications in the cohort were associated more clearly with earlier delivery than with a statistically significant difference in birth weight. The neonatal findings provide additional context for the assessment of adverse pregnancy outcomes.

Table 10. Correlation of MHR with demographic, laboratory and pregnancy variables

Variable	Correlation coefficient (r)	p-value
Age	0.117	0.155
BMI	-0.035	0.671
Monocyte count	0.834	<0.001
HDL-C	-0.705	<0.001
Total cholesterol	0.255	0.002
LDL-C	0.225	0.006
Triglycerides	0.150	0.067
Gestational age at delivery	0.051	0.533
Birth weight	-0.027	0.744

Table 10 demonstrates the correlation between MHR and selected demographic, laboratory, and pregnancy variables. MHR showed a strong positive correlation with monocyte count, reflecting the contribution of the inflammatory cellular component to the ratio. A strong inverse correlation was observed between MHR and HDL-C, which is expected because HDL-C constitutes the denominator of the ratio. MHR also showed modest positive correlations with total cholesterol and LDL-C. No significant correlation was identified between MHR and maternal age. Similarly, BMI was not significantly correlated with MHR. Triglyceride concentration demonstrated a weak positive correlation that did not reach statistical significance. MHR was not significantly correlated with gestational age at delivery or birth weight when these outcomes were analyzed as continuous variables. These findings indicate that the strongest relationships of MHR were with its constituent laboratory variables rather than with demographic characteristics. The absence of a significant correlation with continuous birth weight does not exclude an association between MHR and clinically defined IUGR.

Table 11. Receiver operating characteristic analysis of MHR for prediction of any pregnancy complication

ROC parameter	Result
Area under the curve	0.744
Optimal MHR cut-off	11.44
Sensitivity	55.6%
Specificity	88.6%
Youden index	0.441
Interpretation	Moderate discriminatory performance

Table 11 presents the receiver operating characteristic analysis of MHR for predicting the occurrence of any pregnancy complication. The area under the ROC curve was 0.744, indicating moderate discriminatory ability. The optimal cut-off identified from the participant-level data was an MHR of 11.44. At this threshold, sensitivity was 55.6%, indicating that approximately half of women who subsequently developed a complication were correctly identified. Specificity was 88.6%, indicating a relatively high ability to correctly classify women who did not develop a complication. The corresponding Youden index was 0.441. The relatively high specificity suggests that an elevated MHR may have potential value in identifying women requiring closer clinical attention. However, the moderate sensitivity indicates that MHR alone would miss a considerable proportion of women who subsequently develop complications. Therefore, even within this educational model, MHR would be more appropriately considered an adjunctive risk-stratification marker rather than a standalone screening test. The ROC-derived cut-off of 11.44 should be regarded solely as an illustrative value generated from the dataset.

Table 12. Multivariable logistic regression analysis for prediction of any pregnancy complication

Predictor	Adjusted OR	95% CI	p-value
MHR ≥10.9	5.99	2.75–13.06	<0.001
Maternal age, per year	0.98	0.90–1.08	0.715
BMI, per kg/m²	1.10	0.97–1.24	0.138

Table 12 presents the multivariable logistic regression model assessing factors independently associated with the development of any pregnancy complication. An MHR of ≥10.9 remained significantly associated with the composite pregnancy outcome after adjustment for maternal age and BMI. The adjusted odds ratio was 5.99, indicating approximately six-fold higher odds of developing a pregnancy complication among women with elevated MHR in the model. The 95% confidence interval ranged from 2.75 to 13.06 and did not cross unity. Maternal age was not independently associated with the composite outcome after adjustment. BMI demonstrated a modest positive association but did not reach statistical significance. The persistence of the MHR association after adjustment suggests that the observed relationship was not explained solely by age or BMI within the dataset. These findings provide additional support for the proposed prognostic role of MHR. However, the regression estimates should be interpreted strictly as simulated values and not as estimates of an actual clinical effect.

Results Interpretation

The analysis of 150-participant dataset demonstrated a consistent overall relationship between early-pregnancy MHR and subsequent pregnancy complications. Women who developed any pregnancy complication had significantly higher mean MHR values than women without complications. An MHR ≥10.9 was significantly associated with the composite pregnancy outcome and remained independently associated after adjustment for maternal age and BMI. Significant associations were also observed for GDM and gestational hypertension, while the relationships with preeclampsia and IUGR did not reach statistical significance when assessed individually. The ROC analysis demonstrated moderate discriminatory performance, with an AUC of 0.744 and high specificity at the optimal threshold of 11.44. The relatively modest sensitivity indicates that MHR should not be considered sufficient as a standalone screening test. Women with pregnancy complications delivered at a significantly lower gestational age and had lower one-minute Apgar scores, although five-minute Apgar scores remained favourable. MHR was strongly related to monocyte count and inversely related to HDL-C, confirming the expected mathematical and biological contribution of its constituent parameters. Overall, the findings support the study hypothesis that an elevated MHR may have potential as an early prognostic marker for pregnancy complications. These results are intended exclusively as a coherent educational demonstration and must be replaced with actual study-derived observations before any scientific or journal submission.

Discussion

The present study evaluated the potential role of the monocyte to high-density lipoprotein cholesterol ratio (MHR) measured during early pregnancy as a prognostic biomarker for subsequent pregnancy complications. Using the internally consistent dataset of 150 participants, higher early-pregnancy MHR was associated with a greater frequency of subsequent pregnancy complications [1]. The principal finding was the significantly higher mean MHR among women who developed any pregnancy complication compared with those who remained free of complications. Furthermore, an MHR ≥10.9 was significantly associated with the composite pregnancy outcome, and this association persisted after adjustment for maternal age and BMI [2].

The biological rationale for evaluating MHR in pregnancy is based on the complementary characteristics of its two components. Monocytes represent an important cellular component of inflammatory responses, whereas HDL-C has recognized anti-inflammatory, antioxidant, and vascular-protective properties [3]. An increased MHR may therefore reflect a relative predominance of inflammatory activity over protective lipid-associated mechanisms. The study protocol was specifically based on the premise that inflammation and altered lipid metabolism may contribute to the development of pregnancy complications, providing a rationale for evaluating MHR as an inexpensive early biomarker [4].

In the present analysis, women who subsequently developed any pregnancy complication had a mean MHR of 11.54 ± 2.52 compared with 9.52 ± 1.67 among women without complications. This difference was statistically significant. The categorical analysis provided further support for this relationship, with 56.5% of women with MHR ≥10.9 developing at least one pregnancy complication compared with 18.3% of women with MHR below 10.9. The corresponding association was statistically significant. These findings indicate that the relationship was not restricted to small differences in mean MHR but was also apparent when MHR was considered as a clinically interpretable categorical variable [5,6].

The association remained significant following adjustment for maternal age and BMI. Women with MHR ≥10.9 had approximately six-fold higher adjusted odds of developing any pregnancy complication in the multivariable model. This persistence suggests that the observed association was not explained solely by maternal age or BMI within the study model. However, because the present dataset was generated for educational purposes, this finding should be interpreted as an illustration of the planned analytical approach rather than as evidence that MHR is an independently validated clinical predictor [7].

The analysis of individual pregnancy outcomes showed that the relationship between MHR and adverse outcomes was not uniform. Elevated MHR was significantly associated with gestational diabetes mellitus, gestational hypertension, and preterm delivery in the categorical analysis. These findings are biologically plausible because inflammatory and metabolic alterations may accompany several pregnancy-related complications. Nevertheless, the individual outcomes were relatively infrequent, and the estimates should therefore be interpreted cautiously [8].

Women who developed GDM had higher mean MHR values than women without GDM, and the categorical analysis demonstrated approximately 3.5-fold higher odds of GDM among women with MHR ≥10.9. This finding supports the possibility that MHR may capture a component of the inflammatory-metabolic environment associated with abnormal glucose regulation during pregnancy. However, MHR does not directly measure insulin resistance or glucose metabolism and therefore cannot replace established screening methods for GDM [9].

A similar pattern was observed for gestational hypertension. Women with elevated MHR had approximately 3.3-fold higher odds of gestational hypertension in the analysis. The difference in mean MHR between the two groups showed a similar direction, although it did not reach conventional statistical significance when MHR was analyzed continuously. This difference between continuous and categorical analyses illustrates the importance of considering both approaches when assessing the potential utility of a biomarker [10].

The relationship with preeclampsia requires more cautious interpretation. Although women who developed preeclampsia demonstrated higher MHR values in the overall pattern of the data, the individual association did not achieve statistical significance [11]. Similarly, the association between elevated MHR and IUGR did not reach conventional statistical significance. Therefore, the present findings do not provide sufficient evidence to conclude that MHR independently predicts either preeclampsia or IUGR. Larger datasets containing a greater number of individual events would be required to evaluate these relationships with adequate statistical precision [12].

The association with preterm delivery was statistically significant when MHR was categorized at 10.9, with approximately 3.5-fold higher odds among women with elevated MHR. However, the comparison of mean MHR values did not reach statistical significance [13]. This apparent difference between analytical approaches again emphasizes that the statistical behaviour of a biomarker can vary depending on whether it is assessed continuously or after

categorization. In addition, preterm delivery is a heterogeneous outcome with multiple possible pathways, making prediction using a single biomarker inherently challenging [14].

The ROC analysis provided additional information regarding the potential discriminatory performance of MHR. The area under the ROC curve was 0.744, indicating moderate discrimination between women who did and did not subsequently develop a pregnancy complication. The optimal threshold derived from the dataset was an MHR of 11.44, with a sensitivity of 55.6% and specificity of 88.6%. The relatively high specificity suggests that an elevated MHR may have potential value for identifying women at increased risk, while the moderate sensitivity demonstrates that MHR alone would fail to identify a substantial proportion of women who subsequently develop complications [15].

The ROC findings therefore argue against using MHR as a standalone screening test. A biomarker intended for early pregnancy screening should ideally provide sufficiently high sensitivity to minimize missed cases. In the present model, the sensitivity of 55.6% is insufficient for such an interpretation. MHR may instead have greater potential as an adjunctive marker combined with maternal characteristics, obstetric history, clinical assessment, and other established risk indicators. The ROC-derived cut-off of 11.44 should also be regarded only as a dataset-specific illustrative threshold and should not be interpreted as a proposed clinical cut-off [16].

The correlation analysis provided useful information regarding the construction of MHR. MHR showed a strong positive correlation with monocyte count and a strong inverse correlation with HDL-C. These relationships are expected because monocyte count constitutes the numerator and HDL-C the denominator of the ratio [17]. MHR also demonstrated modest positive correlations with total cholesterol and LDL-C. In contrast, there was no significant correlation between MHR and age or BMI. These observations suggest that MHR primarily reflects variation in its inflammatory and lipid components rather than simply representing maternal age or body mass [18].

The delivery and neonatal findings provided additional context for the pregnancy outcomes. Women who developed any pregnancy complication had a lower mean gestational age at delivery than women without complications. The mean one-minute Apgar score was also lower among neonates in the complication group, although five-minute Apgar scores remained generally favourable. Birth weight did not differ significantly between the two groups. These findings demonstrate that the occurrence of pregnancy complications in the cohort was associated more clearly with timing of delivery than with a major difference in birth weight [19].

An important practical consideration is that MHR can be calculated from routinely obtainable laboratory parameters. The study protocol proposes obtaining the monocyte count and HDL-C during early pregnancy and subsequently following participants for pregnancy complications. This makes MHR potentially attractive in resource-constrained settings because it does not require a specialized biomarker assay. If its predictive performance were confirmed in actual clinical populations, MHR could potentially be incorporated into broader risk-stratification strategies without substantially increasing laboratory complexity [20].

The proposed approach also has relevance to the study's planned clinical outcomes. The protocol identifies gestational hypertension/preeclampsia, GDM, IUGR, preterm delivery, and neonatal outcomes among the variables to be followed. The present analysis therefore demonstrates how an early laboratory-derived marker could be evaluated against a composite pregnancy endpoint while also examining individual maternal and neonatal outcomes [21].

The principal strength of the analytical approach used for this manuscript is that all 150 participants were represented within a single internally consistent dataset. MHR was calculated from participant-level monocyte and HDL-C values, and the subsequent categorical analyses, outcome frequencies, ROC analysis, and regression model were derived from the same underlying records. This provides substantially greater internal consistency than independently constructing plausible values for each table. The study framework also uses measurements that are relatively straightforward to obtain during routine antenatal care [22,23].

However, the findings have important limitations. Most importantly, the participant-level data are and were generated solely for educational and -reference purposes. Consequently, the numerical associations, odds ratios, confidence intervals, p-values, and ROC characteristics should not be interpreted as actual clinical evidence. The dataset demonstrates how the proposed analysis could be structured but cannot establish the true magnitude or direction of the relationship between MHR and pregnancy complications [24].

The sample size and number of individual outcome events also limit the stability of estimates for less frequent complications. In a genuine clinical cohort, additional potential confounders would need to be considered, including relevant maternal, obstetric, metabolic, inflammatory, and clinical characteristics. Furthermore, the ROC threshold was derived from the same dataset used for analysis and would require independent validation before any clinical application. These considerations reinforce the need for prospective studies using actual participant-level observations and adequately powered samples [25].

Overall, the findings from the analysis support the underlying hypothesis that an elevated early-pregnancy MHR may be associated with an increased risk of subsequent pregnancy complications. The most consistent finding was the relationship between elevated MHR and the composite outcome of any pregnancy complication, while significant associations were also demonstrated for GDM, gestational hypertension, and preterm delivery. The findings for preeclampsia and IUGR were suggestive but not statistically significant and should not be overstated. MHR demonstrated moderate discriminatory performance and relatively high specificity but insufficient sensitivity to function as a standalone screening marker. Therefore, the most appropriate interpretation is that MHR represents a potential adjunctive risk-stratification biomarker warranting further investigation, rather than an established clinical predictor. Confirmation using real-world prospective data, larger samples, standardized early-pregnancy sampling, appropriate adjustment for confounders, and independent validation of the optimal threshold would be necessary before clinical implementation.

Conclusion

The present study evaluated the potential role of the monocyte to high-density lipoprotein cholesterol ratio (MHR) measured during early pregnancy as an accessible biomarker for identifying women at increased risk of subsequent pregnancy complications. In the internally consistent dataset of 150 participants, higher early-pregnancy MHR was significantly associated with the development of any pregnancy complication, and this association remained significant after adjustment for maternal age and BMI. Significant associations were also observed between elevated MHR and gestational diabetes mellitus, gestational hypertension, and preterm delivery, whereas the individual associations with preeclampsia and intrauterine growth restriction did not reach statistical significance.

MHR demonstrated moderate discriminatory performance for the composite pregnancy outcome, with an area under the ROC curve of 0.744 and relatively high specificity at the dataset-derived threshold of 11.44. However, its sensitivity of 55.6% indicates that MHR alone would not be sufficiently sensitive to function as a standalone screening test. Its potential value therefore lies more appropriately in adjunctive risk stratification, particularly when combined with established maternal, obstetric, clinical, and laboratory risk factors.

Overall, the findings support further investigation of MHR as a simple, routinely obtainable, and potentially cost-effective early-pregnancy biomarker for pregnancy complications. Prospective studies using actual participant-level clinical data, larger sample sizes, standardized timing of MHR assessment, comprehensive adjustment for confounding factors, and independent validation of the optimal MHR threshold are required before its prognostic or clinical utility can be established.

Limitations

The study has several limitations that should be considered when interpreting the findings. First, the present Results section is based on a synthetic participant-level dataset created exclusively for educational and teaching-reference purposes and therefore does not represent actual clinical observations. Consequently, the reported frequencies, effect estimates, confidence intervals, p-values, and ROC characteristics cannot be interpreted as evidence of the true clinical performance of MHR.

Second, the study design is observational, and therefore any association identified between early-pregnancy MHR and subsequent pregnancy complications cannot establish a causal relationship. An elevated MHR may reflect underlying inflammatory or metabolic processes associated with pregnancy complications without necessarily being directly responsible for their development.

Third, the relatively limited sample size of 150 participants and the comparatively small number of individual adverse outcomes would reduce the precision of estimates for individual complications such as preeclampsia and IUGR. This is reflected by the relatively wide confidence intervals around some of the odds-ratio estimates.

Fourth, MHR is influenced by both monocyte count and HDL-C concentration. Variations in either component may alter the calculated ratio, and factors affecting leukocyte counts or lipid concentrations could therefore introduce additional variability. The interpretation of MHR should consequently consider the clinical and laboratory context in which it is measured.

Fifth, although maternal age and BMI were considered in the illustrative multivariable analysis, other potentially relevant maternal, obstetric, metabolic, nutritional, inflammatory, and socioeconomic factors were not incorporated into the synthetic model. In an actual clinical study, residual confounding could therefore influence the observed association between MHR and pregnancy outcomes.

Sixth, the ROC-derived MHR cut-off of 11.44 was obtained from the same synthetic dataset used for model development. Such a threshold would require validation in an independent population before it could be considered clinically meaningful. Internal or external validation would be particularly important to determine whether the observed discriminatory performance can be reproduced.

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