

# A STUDY OF ASSOCIATION OF MATERNAL PRE-PREGNANCY LOW BMI WITH ADVERSE PREGNANCY OUTCOMES

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## ABSTRACT

**Background:** Maternal nutritional condition prior to conception plays a critical role in determining pregnancy outcomes. A low body mass index (BMI) before pregnancy has been associated with unfavorable maternal and neonatal events; however, evidence evaluating the extent of risk across varying degrees of underweight remains limited in South Indian settings.

**Materials and Methods:** This prospective observational study was conducted over 12 months at Bhaarith Medical College and Hospital. A total of 120 antenatal women were enrolled, including 60 women with low BMI (<18.5 kg/m<sup>2</sup>) and 60 with normal BMI (18.5–24.9 kg/m<sup>2</sup>). Women with low BMI were further stratified into BMI <16, 16.1–16.9, and 17–18.4 kg/m<sup>2</sup> categories. Participants were followed from booking until delivery. Maternal characteristics along with antenatal, intrapartum, and neonatal outcome variables were systematically documented. Statistical evaluation was performed using the Chi-square test and Fisher's exact test, trend analysis, and Pearson correlation as appropriate.

**Results:** Low BMI was associated with higher prevalence of anaemia, inadequate gestational weight gain, abnormal amniotic fluid indices, and non-reactive non-stress tests. Oligohydramnios was significantly more frequent in lower BMI categories, showing an inverse trend with increasing BMI. Neonates born to women with low BMI had lower mean birth weight and higher rates of low birth weight, intrauterine growth restriction, and neonatal hypoglycaemia. Maternal body mass index demonstrated a statistically significant positive association with neonatal birth weight, with the highest frequency of adverse outcomes observed among women in the lowest BMI category.

**Conclusion:** Reduced maternal pre-pregnancy BMI is linked to unfavorable antenatal and neonatal outcomes, with the magnitude of risk progressively rising as BMI declines. Stratifying underweight women may facilitate early identification and targeted nutritional interventions.

**KEYWORDS:** Low Body Mass Index; Maternal Undernutrition; Pregnancy Outcomes; Fetal Growth Restriction; Neonatal Morbidity

## 1. INTRODUCTION

Maternal nutritional status during pregnancy is a pivotal determinant of both maternal well-being and fetal development [1]. Body mass index (BMI) is an anthropometric indicator derived by dividing body weight in kilograms by the square of height in meters (kg/m<sup>2</sup>) and is widely applied to evaluate nutritional status. The World Health Organization (WHO) defines underweight as a BMI below 18.5 kg/m<sup>2</sup> [2,3]. Although considerable attention in maternal health has been directed toward the consequences of overweight and obesity, maternal undernutrition remains a significant public health challenge, particularly in low- and middle-income countries (LMICs), including India [4].

In the Indian context, undernutrition among women of reproductive age arises from interrelated socio-economic constraints, cultural practices, and inadequate dietary intake. Although maternal health indicators have improved over time, findings from the National Family Health Survey (NFHS-5) [5] indicate that a considerable proportion of women commence pregnancy with compromised nutritional status, including a low pre-pregnancy BMI. This is especially prevalent in rural and underserved areas, including parts of Tamil Nadu, where dietary insufficiency and anaemia continue to burden maternal health outcomes [5]. In such settings, women often begin pregnancy with inadequate nutritional reserves, which can be further depleted during gestation, increasing vulnerability to a range of complications [6].

Multiple investigations have demonstrated that maternal underweight status adversely affects both maternal and fetal well-being [7]. Women with a low BMI face a greater likelihood of developing anaemia during pregnancy, experiencing insufficient gestational weight gain, oligohydramnios, and an increased occurrence of preterm labour [2,8]. Furthermore, these women often experience prolonged labour, increased need for medical interventions, and postpartum complications, which may arise due to reduced maternal energy stores and impaired placental function [9]. Additionally, undernourished mothers are more susceptible to infections and are less likely to tolerate obstetric stress, further complicating labour and delivery [10].

From the fetal standpoint, maternal undernutrition adversely affects placental development, which can restrict nutrient and oxygen delivery to the foetus [11]. Such maternal factors frequently contribute to fetal growth restriction, resulting in small-for-gestational-age (SGA) infants and low birth weight (LBW) neonates. These newborn conditions are associated with increased neonatal morbidity and mortality, adverse neurodevelopmental outcomes, and a greater predisposition to chronic diseases in adulthood [12,13]. Moreover, studies have found a dose-dependent relationship, where the severity of underweight status further amplifies the risk of adverse perinatal outcomes. For instance, Lee et al. (2015) [8] demonstrated that markedly low maternal BMI was a significant predictor of fetal growth restriction and preterm birth, particularly in resource-limited settings. In a large population-based study from Japan, Enomoto et al. (2016) [14] evaluated 97,157 pregnancies and reported that women with BMI <18.5 kg/m<sup>2</sup> had a significantly higher incidence of SGA infants compared with those within the normal BMI range. Specifically, the study reported that 15.0% of underweight women who gained weight below the Institute of Medicine (IOM) guidelines delivered SGA infants, compared to 10.4% in the normal BMI group under similar weight gain conditions. These findings underscore the increased risk of SGA associated with low maternal BMI [15].

Despite the well-documented risks, there remains a paucity of regional data that explore the spectrum of low BMI and its nuanced effects on pregnancy outcomes, particularly in South Indian populations [4]. Moreover, most existing studies categorize all underweight women into a single group (BMI <18.5 kg/m<sup>2</sup>), which may mask important variations within this group [15]. Given the high prevalence of underweight status in certain regions of Tamil Nadu, a stratified approach that examines outcomes across subgroups of low BMI could provide valuable insights. Such information is vital for healthcare providers and public health planners to design targeted nutritional and antenatal interventions that can mitigate these risks early in pregnancy [16].

The primary objective of this study is to assess the association between low maternal body mass index (BMI) and adverse maternal and fetal outcomes by comparing outcomes with those of women in the normal BMI range, and to further examine potential dose-response patterns within the underweight subgroup. Maternal outcomes including anaemia, gestational weight gain, amniotic fluid index, and intrapartum parameters were assessed alongside fetal and neonatal indicators such as non-stress test reactivity, fetal heart rate decelerations, low birth weight, small for gestational age status, and preterm birth. By incorporating both between-group comparisons and stratified analyses among underweight women, this study seeks to delineate the spectrum and severity of risks associated with progressively lower BMI. The findings are intended to strengthen evidence-based maternal healthcare strategies tailored to the demographic and nutritional context of the Tamil Nadu population.

## 2. MATERIALS AND METHODS

This prospective observational study was carried out over a 12-month period in the Department of Obstetrics and Gynaecology at Bharath Medical College and Hospital, following approval from the Institutional Ethics Committee (IEC) (Proposal number: BIEC-117-24). The study population comprised a total of 120 antenatal women, divided into two groups. The study cohort comprised 60 women with a pre-pregnancy BMI <18.5 kg/m<sup>2</sup>, categorized as underweight based on WHO criteria, whereas the control cohort included 60 women with a normal pre-pregnancy BMI ranging from 18.5 to 24.9 kg/m<sup>2</sup>.

Participants were recruited from the obstetrics outpatient and inpatient departments after obtaining written informed consent. Women with multiple gestations, diabetes mellitus, systemic hypertension, congenital heart disease, or other significant systemic illnesses were excluded from the study. For both groups, detailed demographic and clinical information was collected using a structured proforma, including age, obstetric history, socioeconomic status, menstrual history, medical history, and nutritional status.

All participants underwent comprehensive general physical, systemic, and obstetric examinations. Obstetric assessment included documentation of fundal height, fetal presentation, fetal heart sounds, estimated fetal weight, and clinical signs suggestive of oligohydramnios. Baseline laboratory investigations such as haemoglobin estimation, urine analysis, blood grouping and typing, random blood sugar, bleeding time, and clotting time were performed for all enrolled women. Obstetric ultrasonography was carried out as per routine antenatal care, with particular attention to the assessment of the AFI.

Participants were followed prospectively from the booking visit until delivery. Third-trimester fetal assessment included non-stress tests (NSTs). In cases with reactive NST findings, the decision for induction of labour was made according to the Bishop's score and prevailing institutional guidelines. Intrapartum monitoring during the first and second stages of labour was performed using continuous electronic fetal monitoring. Emergency lower segment caesarean section (LSCS) was performed when indicated, including for fetal distress or non-progression of labour. When artificial rupture of membranes was undertaken, the characteristics of the amniotic fluid were documented.

Each neonate was evaluated immediately after delivery by a paediatrician. The maternal and neonatal variables recorded included onset of labour (spontaneous or induced), gestational age at birth, status of the amniotic fluid, fetal heart rate patterns, mode of delivery, Apgar scores at 1 and 5 minutes, birth weight, occurrence of preterm delivery, low birth weight (LBW <2.5 kg), and requirement for NICU admission.

Data were entered into Microsoft Excel and analysed using SPSS software (version 23.0). Quantitative data were summarized as mean ± standard deviation, and qualitative data were reported as frequencies and percentages. Differences between the low BMI and normal BMI groups were analysed using the independent Student's t-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as applicable. Trend assessment across subcategories within the low BMI group was performed using the Cochran-Armitage test. The association between maternal BMI and neonatal birth weight was examined using the Pearson correlation coefficient. A p-value <0.05 was considered indicative of statistical significance.

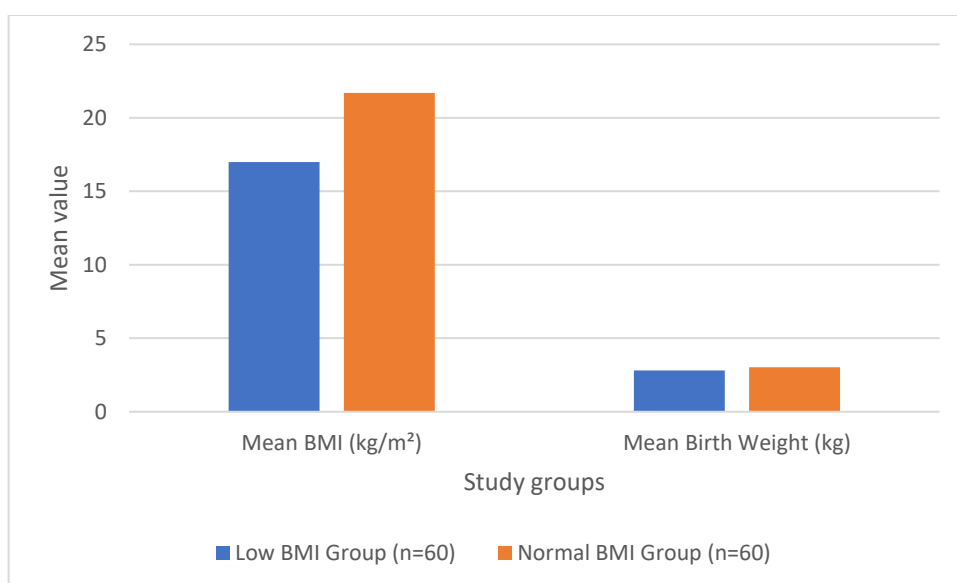
### 3. RESULTS

A total of 120 pregnant women were analysed, including 60 women with a low pre-pregnancy body mass index (BMI <18.5 kg/m<sup>2</sup>) and 60 women with a normal BMI (18.5–24.9 kg/m<sup>2</sup>), who constituted the control group. Inclusion of women with normal BMI facilitated direct comparison of maternal, antenatal, and neonatal outcomes across different nutritional categories. The two groups were largely comparable with respect to baseline demographic and obstetric characteristics, thereby reducing the likelihood of confounding. Antenatal findings, intrapartum parameters, and fetal and neonatal outcomes were evaluated and are summarized in Table 1. Group comparisons were performed using statistical tests appropriate to the type and distribution of data, with statistical significance defined as  $p < 0.05$ . The results highlight distinct variations in pregnancy outcomes associated with low maternal BMI when compared with women of normal BMI.

#### 3.1 Comparison of Maternal, Antenatal, and Neonatal Outcomes Between Low BMI and Normal BMI Groups

##### 3.1.1 Baseline Characteristics

The average maternal age did not differ significantly between the groups, measuring  $25.33 \pm 4.23$  years in the low BMI group and  $26.1 \pm 3.9$  years in the control group ( $p = 0.28$ ). Distribution across age categories (<20, 21–25, 26–30, and >30 years) was comparable between groups, with most participants falling within the 21–25-year category.



**Figure 1.** Comparison of Mean Maternal BMI and Neonatal Birth Weight Between Groups. Values represent group means. BMI: Body Mass Index. Birth weight is expressed in kilograms.

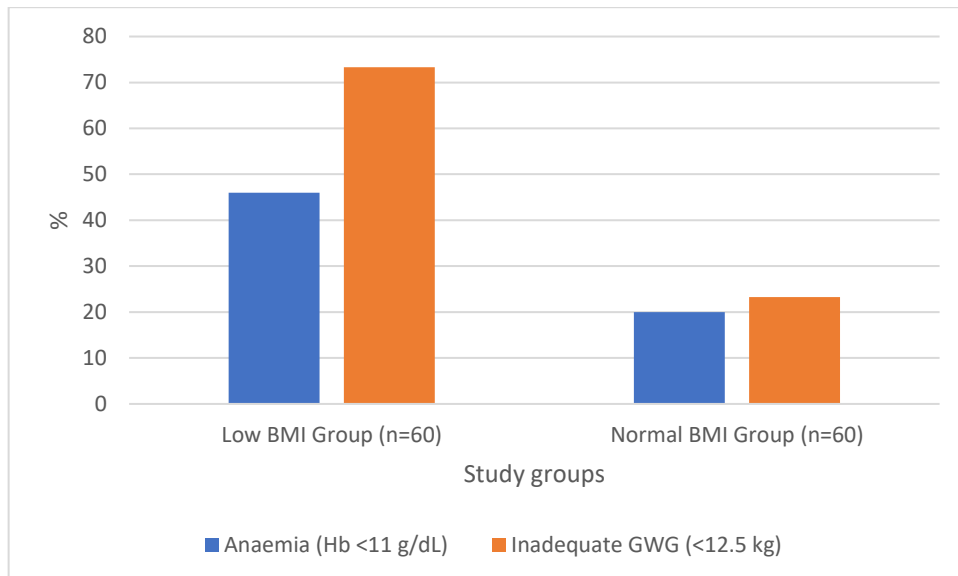
As expected, and as seen in Figure 1, the mean BMI was significantly lower in the low BMI group ( $16.98 \pm 0.39$  kg/m<sup>2</sup>) compared with the control group ( $21.7 \pm 1.9$  kg/m<sup>2</sup>;  $p < 0.001$ ). Within the low BMI group, most women had a BMI between 17 and 18.4 kg/m<sup>2</sup> (63%), whereas all women in the control group fell within the normal BMI range, predominantly between 18.5 and 22.9 kg/m<sup>2</sup> (63.3%). Assisted reproductive technology was used in only a small proportion of participants, with no statistically significant difference observed between the groups ( $p = 0.56$ ).

Baseline obstetric parameters, including gravidity and parity, were similar in both cohorts. Primigravidae accounted for 46.7% of women in the low BMI group and 50% in the control group ( $p = 0.89$ ). Likewise, nulliparous women comprised 50% of the low BMI group and 53.3% of the control group ( $p = 0.83$ ). The mean baseline haemoglobin concentration was significantly lower among women with low BMI ( $10.6 \pm 1.2$  g/dL) compared to controls ( $p < 0.001$ ).

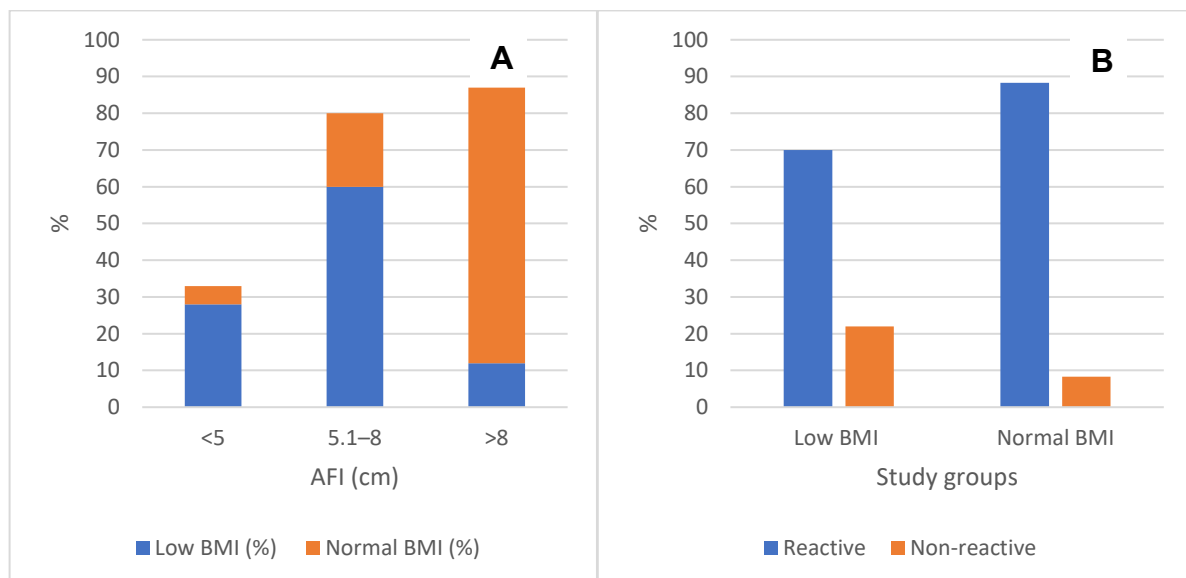
##### 3.1.2 Antenatal Parameters

Inadequate gestational weight gain (<12.5 kg) was observed significantly more frequently in the low BMI group (73.3%) compared with the control group (23.3%;  $p < 0.001$ ). Marked differences were also noted in amniotic fluid index (AFI) distribution. An AFI <5 cm was observed in 28% of women in the low BMI group, compared with 5% in the control group. In contrast, AFI values >8 cm was predominantly noted among controls (75%) versus 12% in the low BMI group. The differences across all AFI categories were statistically significant ( $p < 0.001$ ). The distribution of AFI categories and NST reactivity between the groups is depicted in Figure 3.

At 36 weeks of gestation, the mean fundal height was significantly reduced in the low BMI group ( $31.5 \pm 1.8$  cm) compared with the control group ( $33.4 \pm 1.5$  cm), demonstrating statistical significance ( $p < 0.001$ ). Non-stress test (NST) findings also differed between groups. Reactive NSTs were less frequent among women with low BMI (70%) compared with controls (88.3%), while non-reactive NSTs were more common in the low BMI group (22% vs. 8.3%), with these differences reaching statistical significance. Figure 2 illustrates the comparative distribution of anaemia and insufficient gestational weight gain between the two study groups.



**Figure 2.** Distribution of anaemia and inadequate gestational weight gain in the low BMI and normal BMI groups. Data are presented as percentages. BMI: Body Mass Index. Detailed statistical analysis is provided in Table 1.



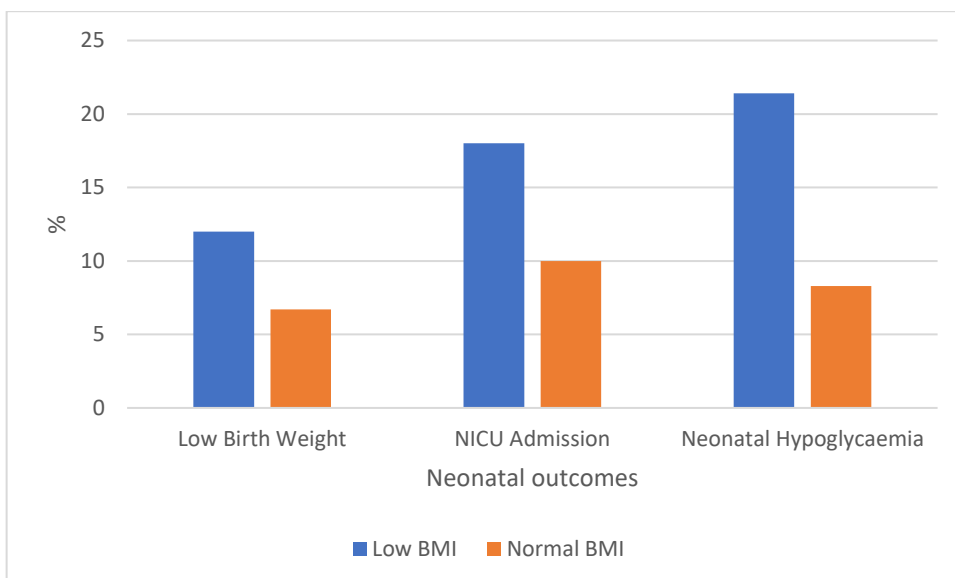
**Figure 3.** (A) Proportion of participants across amniotic fluid index (AFI) categories and (B) distribution of Non-Stress Test (NST) reactivity in the low BMI and normal BMI groups. Data are shown as percentages. AFI: Amniotic Fluid Index; NST: Non-Stress Test. Statistical comparisons are presented in Table 1.

### 3.1.3 Fetal and Neonatal Outcomes

The average neonatal birth weight was significantly lower among women in the low BMI group ( $2.81 \pm 0.29$  kg) than in the control group ( $3.02 \pm 0.34$  kg), with the difference reaching statistical significance ( $p < 0.001$ ). A greater proportion of infants born to mothers with low BMI weighed  $<2.5$  kg (11.7% vs. 6.7%); however, this variation was not statistically significant. In addition, the frequencies of low birth weight (12% vs. 6.7%), intrauterine growth restriction (10.5% vs. 5%), small for gestational age (14% vs. 8.3%), and preterm delivery (8.2% vs. 6.7%) were comparable between the two groups, with no statistically significant differences observed.

Lower APGAR scores were more frequent among neonates born to women with low BMI, with APGAR scores  $<7$  at 1 minute observed in 13% compared with 5% in the control group, and at 5 minutes in 6.5% versus 1.7%, although these differences did not reach statistical significance. NICU admissions were more frequent among infants born to women in the low BMI group (18%) compared with the control group (10%); however, the difference did not reach statistical significance.

The incidence of neonatal hypoglycaemia was significantly higher in the low BMI group (21.4%) than in the control group (8.3%), with statistical significance demonstrated ( $p = 0.04$ ). Among the affected neonates, moderate hypoglycaemia accounted for the majority of cases in both cohorts. Neonatal sepsis occurred in 11.2% of infants in the low BMI group and 5% of those in the control group, without a statistically significant difference. The proportions of early-onset and late-onset sepsis were similar between the two groups. A comparative overview of selected neonatal outcomes is presented in Figure 4.



**Figure 4.** Distribution of selected neonatal outcomes in the low BMI and normal BMI groups. Data are expressed as percentages. Corresponding statistical analyses are detailed in Table 1.

### 3.1.4 Maternal Outcomes

The frequency of labour induction was comparable between the low BMI and control groups (35% vs. 30%;  $p = 0.55$ ). There was no statistically significant difference in the mode of delivery, with vaginal birth being the predominant mode in both the low BMI (49%) and control (60%) groups. The proportions of lower segment caesarean section and instrumental delivery were likewise similar across the two cohorts.

No significant intergroup differences were observed in the rates of premature rupture of membranes, preeclampsia/eclampsia, or overall postpartum complications. Among women who experienced postpartum morbidity, postpartum haemorrhage was the most common complication in both groups, followed by wound infection and urinary tract infection.

**Table I. Comprehensive Profile of Baseline Characteristics and Maternal-Neonatal Outcomes**

Variable	n (%)		p-value
	Test Group (60)	Control group (60)	
<b>Baseline characteristics</b>			
<b>Age (years)</b>	25.33±4.23*	26.1 ± 3.9	0.28 <sup>a</sup>
<20	6 (10)	5 (8.3)	
21-25	28 (46.67)	29 (48.3)	
26-30	18 (30)	18 (30)	
>30	8 (13.33)	8 (13.4)	
<b>BMI (kg/m<sup>2</sup>)</b>	16.98 ± 0.39*	21.7 ± 1.9	<0.001 <sup>a</sup>
<16	8 (13.33)	-	
16-16.9	14 (23.33)	-	
17-18.4	38 (63)	-	
18.5- 22.9	-	38 (63.3)	
23- 24.9	-	22 (36.7)	
<b>Assisted reproduction</b>	1 (1.67)	2 (3.3)	0.56 <sup>c</sup>
<b>Gravida</b>			
G1	28 (46.70)	30 (50.0)	0.89 <sup>b</sup>
G2	20 (33.30)	19 (31.7)	
G3	12 (20)	11 (18.3)	
<b>Parity</b>			
P0	30 (50)	32 (53.3)	0.83 <sup>b</sup>
P1	20 (33.30)	18 (30.0)	
P2	10 (16.70)	10 (16.7)	
<b>Baseline Hb (g/dL)</b>	10.6 ± 1.2	10.6 ± 1.2	<0.001 <sup>a</sup>
<b>Anemia (Hb &lt;11g/dL)</b>	28 (46)	12 (20)	0.003 <sup>b</sup>
<b>Antenatal Parameters</b>			
<b>Gestational weight gain &lt; recommended (12.5kg)</b>	44 (73.30)	14 (23.3)	<0.001 <sup>b</sup>
<b>AFI (cm)</b>			
<5	17 (28)	3 (5)	<0.001 <sup>b</sup>

5.1-8	36 (60)	12 (20)	<0.001 <sup>b</sup>
>8	7 (12)	45 (75)	<0.001 <sup>b</sup>
<b>Fundal height at 36 weeks (cm)</b>	31.5±1.8*	33.4 ± 1.5*	<b>&lt;0.001<sup>a</sup></b>
<b>NST</b>			
Reactive	42 (70)	53 (88.3)	0.02 <sup>a</sup>
Non-reactive	13 (22)	5 (8.3)	0.03 <sup>a</sup>
<b>Fetal and neonatal outcomes</b>			
<b>Birth weight (kg)</b>	2.81 ± 0.29*	3.02 ± 0.34	<0.001 <sup>a</sup>
<b>&lt;2.5</b>	7 (11.7)	4 (6.7)	0.34 <sup>b</sup>
<b>2.5-3.0</b>	37 (61.7)	24 (40)	-
<b>&gt;3</b>	16 (26.6)	32 (53.3)	-
<b>LBW</b>	7 (12)	4 (6.7)	0.53 <sup>c</sup>
<b>IUGR</b>	6 (10.5)	3 (5)	0.30 <sup>b</sup>
<b>SGA</b>	8 (14)	5 (8.3)	0.37 <sup>b</sup>
<b>Preterm birth</b>	5 (8.2)	4 (6.7)	0.73 <sup>b</sup>
<b>APGAR &lt;7</b>			
at 1 minute	8 (13)	3 (5)	0.12 <sup>b</sup>
at 5 minutes	4 (6.5)	1 (1.7)	0.17 <sup>c</sup>
<b>NICU Admission</b>	11 (18)	6 (10)	0.21 <sup>b</sup>
<b>Neonatal Hypoglycaemia</b>	13 (21.4)	5 (8.3)	0.04 <sup>b</sup>
Mild	3 (23.8)	3 (60)	
Moderate	7 (53.4)	2 (40)	
Severe	3 (23.8)	0 (0)	
<b>Neonatal Sepsis</b>	7 (11.2)	3 (5)	0.32 <sup>c</sup>
Early onset	4 (63)	2 (66.67)	
Late onset	3 (37)	1 (33.33)	
<b>Maternal outcomes</b>			
<b>Labor Induction</b>	21 (35)	18 (30.0)	0.55 <sup>b</sup>
<b>Mode of Delivery</b>			
- Vaginal delivery	29 (49)	36 (60)	0.22 <sup>b</sup>
- LSCS	26 (43)	20 (33.3)	0.27 <sup>b</sup>
- Instrumental delivery	5 (8)	4 (6.7)	0.73 <sup>c</sup>
<b>PROM</b>	3 (4.7)	4 (6.7)	0.70 <sup>c</sup>
<b>Eclampsia/Preeclampsia</b>	1 (2.4)	2 (3.3)	0.56 <sup>c</sup>
<b>Postpartum complications</b>	6 (9.5)	5 (8.3)	0.75 <sup>b</sup>
PPH	4 (66.67)	3 (60)	
Wound infection	1 (16.67)	1 (20)	
UTI	1 (16.67)	1 (20)	

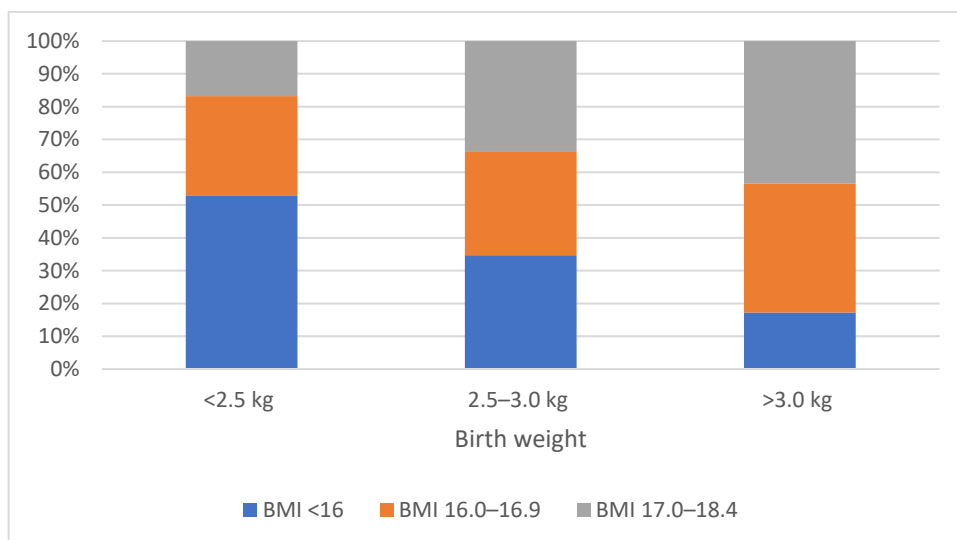
Data are presented as mean ± standard deviation or as number (%), as appropriate. Group comparisons were conducted using <sup>a</sup>Independent Student's t-test for continuous variables, <sup>b</sup>Chi-square test for categorical variables with sufficient expected cell counts, and <sup>c</sup>Fisher's exact test when expected cell counts were <5. P-values shown in bold denote statistical significance ( $p < 0.05$ ). Abbreviations: BMI, Body Mass Index; Hb, Haemoglobin; AFI, Amniotic Fluid Index; NST, Non-Stress Test; LBW, Low Birth Weight; IUGR, Intrauterine Growth Restriction; SGA, Small for Gestational Age; LSCS, Lower Segment Caesarean Section; PROM, Premature Rupture of Membranes; PPH, Postpartum Haemorrhage; UTI, Urinary Tract Infection.

### 3.2 Association of different categories of maternal Low BMI with adverse outcomes

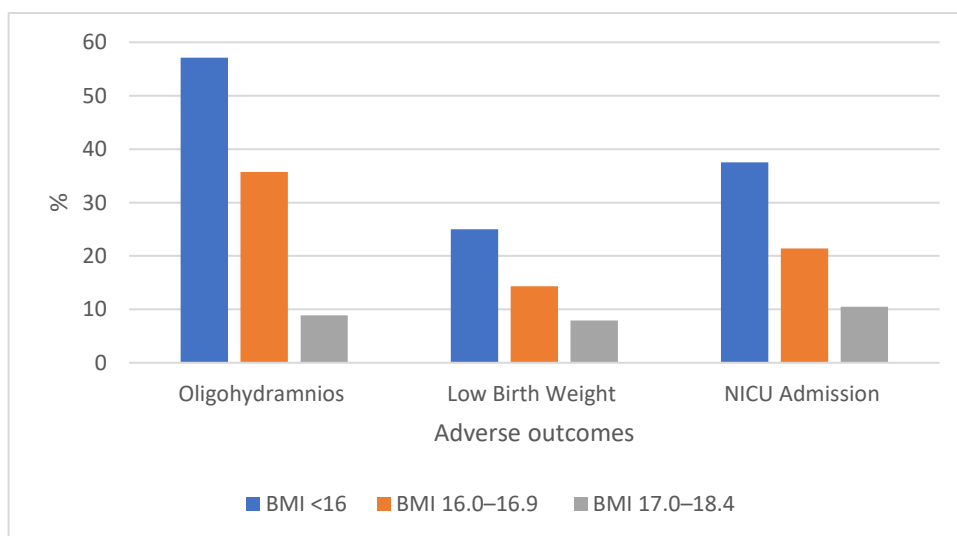
Within the low maternal BMI cohort, adverse maternal and neonatal outcomes were additionally evaluated across BMI subcategories to explore potential dose–response trends within the underweight spectrum, as presented in Table II. In this analysis, multiple maternal and neonatal parameters were examined in relation to the specified maternal BMI categories detailed in Table II. Anaemia was significantly associated with lower BMI ( $\chi^2 = 13.60$ ,  $p = 0.0011$ ), with a clear inverse trend ( $z = -3.60$ ,  $p = 0.0003$ ). AFI <5 cm was also significantly more common in lower BMI groups ( $\chi^2 = 14.75$ ,  $p = 0.0006$ ), with a decreasing trend as BMI increased ( $\chi^2$  for trend = 10.54,  $p = 0.0012$ ). Borderline AFI (5.1–8 cm) showed a borderline association ( $\chi^2 = 5.82$ ,  $p = 0.054$ ) without a significant trend ( $\chi^2$  for trend = 0.77,  $p = 0.38$ ), while AFI >8 cm showed no association ( $\chi^2 = 0.98$ ,  $p = 0.61$ ) or trend ( $\chi^2$  for trend = 0.52,  $p = 0.47$ ). NST reactivity did not reach statistical significance ( $\chi^2 = 3.96$ ,  $p = 0.138$ ), though a borderline increasing trend was observed ( $\chi^2$  for trend = 3.59,  $p = 0.058$ ). In contrast, non-reactive NSTs were significantly more prevalent among lower BMI groups ( $\chi^2 = 19.36$ ,  $p < 0.0001$ ), with a

strong decreasing trend as BMI increased ( $\chi^2$  for trend = 14.69,  $p = 0.0001$ ). Fetal heart rate decelerations were also more common in lower BMI categories ( $\chi^2 = 8.43$ ,  $p = 0.015$ ), with a significant inverse trend ( $\chi^2$  for trend = 7.86,  $p = 0.0051$ ). Low birth weight was significantly associated with lower BMI ( $\chi^2 = 9.48$ ,  $p = 0.009$ ), with a corresponding trend ( $\chi^2$  for trend = 8.23,  $p = 0.0041$ ). IUGR also showed a significant association (Fisher's  $p = 0.0046$ ) and trend ( $\chi^2$  for trend = 9.34,  $p = 0.0022$ ). SGA was significantly linked to lower BMI (Fisher's  $p = 0.0079$ ), with a strong decreasing trend ( $p$  for trend = 0.0002). As illustrated in Figure 5, decreasing maternal BMI was linked to an increased proportion of neonates with birth weights below 2.5 kg, whereas comparatively higher BMI subgroups demonstrated a larger proportion of infants weighing more than 3 kg. This distribution reinforces the observed positive relationship between maternal BMI and fetal growth, indicating that even within the underweight range, incremental increases in BMI are associated with improved birth weight outcomes. Preterm delivery was not significantly associated with BMI categories on group-wise comparison ( $p = 0.1276$ ); however, a statistically significant linear trend was observed across categories ( $p$  for trend = 0.0229). An Apgar score  $<7$  at 5 minutes approached statistical significance in intergroup analysis ( $p = 0.0823$ ), while the trend analysis demonstrated significance ( $p$  for trend = 0.0308). NICU admissions were significantly more frequent in the lowest BMI subgroup (Fisher's  $p = 0.0035$ ), with a pronounced decreasing trend across increasing BMI categories ( $p$  for trend = 0.0005). Mild neonatal hypoglycaemia did not show a significant association ( $p = 0.3542$ ) or trend ( $p$  for trend = 0.2164). In contrast, moderate ( $p = 0.0435$ ;  $p$  for trend = 0.0021) and severe hypoglycaemia ( $p = 0.0442$ ;  $p$  for trend = 0.0039) were significantly more common in the lowest BMI subgroup.

As illustrated in Figure 6, a clear gradient was observed between decreasing maternal BMI and the frequency of adverse outcomes. The lowest BMI category ( $<16$  kg/m<sup>2</sup>) demonstrated the highest proportion of oligohydramnios, low birth weight, and NICU admissions, with a progressive decline in these outcomes across higher BMI subgroups. This trend highlights a dose-response relationship, suggesting that the severity of maternal undernutrition is directly associated with increasing maternal and neonatal risk.



**Figure 5.** Neonatal birth weight distribution across categories of low maternal BMI. Birth weight categories are expressed as percentages within each maternal BMI subgroup.



**Figure 6.** Distribution pattern of adverse maternal and neonatal outcomes across graded categories of low maternal BMI. Data are presented as percentages within each BMI subgroup. BMI: Body Mass Index; NICU: Neonatal Intensive Care Unit.

Early-onset neonatal sepsis did not demonstrate a significant association across BMI categories ( $p = 0.0823$ ); however, trend analysis indicated statistical significance ( $p$  for trend = 0.0308). In contrast, late-onset sepsis was significantly associated with BMI ( $p = 0.0442$ ) and exhibited a significant decreasing trend with increasing BMI ( $p$  for trend = 0.0039). Labour induction showed no significant association with BMI categories ( $p = 0.1165$ ), although a significant trend was identified ( $p$  for trend = 0.0384). Vaginal delivery rates increased across higher BMI subgroups but were not significantly associated ( $p = 0.2566$ ), and no significant trend was observed ( $p$  for trend = 0.1058). Instrumental deliveries were more frequent in lower BMI categories ( $p = 0.1276$ ), with a significant inverse trend across increasing BMI ( $p$  for trend = 0.0229). Rates of lower segment caesarean section (LSCS) remained uniform across groups ( $p = 0.9188$ ) without a significant trend ( $p$  for trend = 0.7170).

PROM was not significantly associated with BMI categories ( $p = 0.3542$ ), although the trend analysis reached statistical significance ( $p$  for trend = 0.0394). Neither eclampsia/preeclampsia nor wound infection demonstrated significant association or trend ( $p = 1.0000$ ;  $p$  for trend = 0.4830 for both outcomes). UTIs were not significantly associated with BMI on categorical comparison ( $p = 0.1333$ ); however, a significant trend was observed ( $p$  for trend = 0.0353), indicating increased occurrence among women in the lowest BMI subgroup.

**Table II. Adverse Outcomes Stratified by Maternal Low BMI Categories**

Variables	BMI <16 kg/m <sup>2</sup> (n=8) (%)	BMI 16.1-16.9 kg/m <sup>2</sup> (n=14) (%)	BMI 17-18.4 kg/m <sup>2</sup> (n=38) (%)	p-value
<b>G.W.G &lt;12.5kg (n=44)</b>	6 (75)	10 (71.43)	28 (73.68)	0.980* 1.000#
<b>Anemia (n=28)</b>	7 (87.5)	10 (71.43)	11 (28.9%)	0.0011* 0.0003#
<b>AFI (cm)</b>				
<5	4 (50)	3 (21.43)	4 (10.53)	0.0006* 0.0012#
5.1-8	3 (37.5)	7 (50)	20 (52.63)	0.054* 0.38#
>8	1 (12.5)	4 (28.57)	16 (42.11)	0.61* 0.47#
<b>NST</b>				
Reactive	2 (25)	6 (42.86)	30 (78.95)	0.138* 0.058#
Non-reactive	4 (50)	5 (35.71)	5 (13.16)	0.0001* 0.0001#
Decelerations	2 (25)	3 (21.43)	3 (7.89)	0.015* 0.0051#
<b>Birth weight (kg)</b>				
<2.5	3 (37.5)	3 (21.43)	1 (2.63)	r= 0.929 <sup>S</sup> p<0.0001
2.5 – 2.9	4 (50)	8 (57.14)	25 (65.79)	
>3	1 (12.5)	3 (21.43)	12 (31.58)	
<b>LBW</b>	3 (37.5)	3 (21.43)	1 (2.63)	0.009* 0.0041#
<b>IUGR</b>	3 (37.5)	2 (14.29)	1 (2.63)	0.0046^ 0.0022#
<b>SGA</b>	4 (50)	3 (21.43)	1 (2.63)	0.0079^ 0.0002
<b>Preterm birth</b>	2 (25)	2 (14.29)	1 (2.63)	0.1276^ 0.0229#
<b>APGAR&lt;7 @5min</b>	2 (25)	1 (7.14)	1 (2.63)	0.0823^ 0.0308
<b>NICU admission</b>	5 (62.5)	3 (21.43)	3 (7.89)	0.0035^ 0.0005#
<b>Neonatal hypoglycemia</b>				
Mild	1 (12.5)	1 (7.14)	1 (2.63)	0.3542^ 0.2164#
Moderate	3 (37.5)	3 (21.43)	1 (2.63)	0.0435^ 0.0021#
Severe	2 (25)	1 (7.14)	0	0.0442^ 0.0039#
<b>Neonatal sepsis</b>				
Early onset	2 (25)	1 (7.14)	1 (2.63)	0.0823^ 0.0308#
Late onset	2 (25)	1 (7.14)	0	0.0442^

				0.0039 <sup>#</sup>
<b>Labor induction</b>	5 (62.5)	6 (42.86)	10 (26.32)	0.1165* 0.0384 <sup>#</sup>
<b>Mode of delivery</b>				
Vaginal	2 (25)	6 (42.86)	21 (55.26)	0.2566 <sup>^</sup> 0.1058 <sup>#</sup>
LSCS	4 (50)	6 (42.86)	16 (42.11)	0.9188* 0.7170 <sup>#</sup>
Instrumental	2 (25)	2 (14.29)	1 (2.63)	0.1276 <sup>^</sup> 0.0229 <sup>#</sup>
<b>PROM</b>	1 (12.5)	2 (14.29)	0	0.3542 <sup>^</sup> 0.0394 <sup>#</sup>
<b>Eclampsia/ preeclampsia</b>	0	1 (7.14)	0	1.000 <sup>^</sup> 0.4830 <sup>#</sup>
<b>PPH</b>	2 (25)	2 (14.29)	1 (2.63)	0.1276 <sup>^</sup> 0.0229 <sup>#</sup>
<b>Wound infection</b>	0	0	1 (2.63)	1.000 <sup>^</sup> 0.4830 <sup>#</sup>
<b>UTI</b>	1 (12.5)	0	0	0.1333 <sup>^</sup> 0.0353 <sup>#</sup>

\*- Chi square test, <sup>#</sup>-Trend analysis using Cochran-Armitage, <sup>^</sup>- Fisher exact test, <sup>§</sup>- Pearson correlation

#### 4. DISCUSSION

This study demonstrates that low maternal BMI is associated with a range of adverse pregnancy outcomes. Key findings demonstrated significant associations with higher rates of anaemia, inadequate gestational weight gain, abnormal amniotic fluid index values, non-reassuring fetal surveillance findings, and an increased occurrence of adverse neonatal outcomes. Stratified analysis within the underweight cohort further revealed that risks escalated with worsening degree of undernutrition.

##### 4.1 Anaemia and Nutritional Status

Maternal anaemia was significantly higher among women with low BMI, and within low BMI subcategories, the prevalence increased as BMI decreased. According to Black et al. (2013) [1], maternal undernutrition contributes to micronutrient deficiencies and anaemia, especially in low-income settings, which is consistent with our findings that women with lower BMI had more pronounced anaemia. Similarly, Tan et al. (2020) [17] reported a high prevalence of anaemia among undernourished pregnant women; our study extends this evidence by showing a clear **BMI–anaemia gradient**, indicating that lower BMI is associated with more severe haematological compromise.

##### 4.2 Gestational Weight Gain and Amniotic Fluid Status

Insufficient gestational weight gain was notably more prevalent among women with low BMI, with the highest frequency observed in those within the lowest BMI category. This aligns with Ramakrishnan et al. (2012) [6], who observed that maternal undernutrition is linked with insufficient gestational weight gain [6]. We also found that abnormal AFI values, especially oligohydramnios (AFI <5 cm), were more frequent in lower BMI categories. Kramer (2003) [7] suggested that poor maternal nutrition may impair uteroplacental blood flow, impacting amniotic fluid volume, which supports our observation of declining AFI with decreasing maternal BMI.

##### 4.3 Fetal Surveillance: NST Abnormalities

Our finding of a higher proportion of non-reactive NSTs in women with lower BMI mirrors physiological understandings reported by Cibils (1976) [18], who linked adverse fetal heart rate patterns with compromised fetal condition. This suggests that reduced maternal reserves may impair fetal adaptation during stress. Godfrey et al. (1996) [11] further underscored the influence of maternal nutrition on fetal physiological integrity, a notion supported by the gradient of NST abnormalities seen across BMI subgroups in our study.

##### 4.4 Fetal Growth Outcomes

Birth weight outcomes in our cohort reflect established associations between maternal underweight and fetal growth restriction. Han et al. (2011) [2] reported that low maternal BMI significantly increases the risks of LBW and SGA infants, consistent with our finding of higher proportions of these outcomes in the lowest BMI categories. Lee et al. (2013) [8] noted global patterns wherein maternal undernutrition contributes substantially too small for gestational age births, which resonates with our data showing greater SGA prevalence among women with lower BMI. Indian-specific evidence by Ramesh et al. (2019) [9] and Mamidi et al. (2022) [15] also documented LBW and growth restriction in undernourished populations, further corroborating our results in a regional context.

##### 4.5 Gestational Duration and Preterm Birth

Although preterm birth did not show statistically significant differences overall, the trend across BMI categories indicated a possible relationship. Han et al. (2011) [2] identified maternal underweight as a modest risk factor for preterm birth,

which is reflected in our trend analysis. Short et al. (2018) [19], in rural South Asian cohorts, also observed similar trends, suggesting that subtle nutritional deficiencies may influence gestational duration even when categorical differences are not significant.

#### **4.6 Neonatal Metabolic Complications**

Neonatal hypoglycaemia, particularly in moderate and severe forms, was more common among infants born to mothers with lower BMI. Christian (2010) [10] described how intrauterine nutritional deprivation can reduce fetal glycogen stores, increasing the risk of postnatal hypoglycaemia, aligning closely with our findings. Gupta et al. (2004) [20] further noted higher metabolic stress in neonates from undernourished mothers, supporting the idea that low maternal BMI is linked to compromised neonatal metabolic adaptation.

#### **4.7 Neonatal Infection**

While neonatal sepsis did not show a statistically significant association, trends across BMI categories suggest that extremely low maternal BMI could increase susceptibility. Sebayang et al. (2012) [16] highlighted that maternal nutritional status may influence neonatal immune competence and infection risk, which may explain the directional trends observed in our study, even in the absence of significant categorical differences.

#### **4.8 Intrapartum and Delivery Outcomes**

Mode of delivery and induction rates did not differ significantly across BMI categories in this study. Chen et al. (2020) [21] reported that low maternal BMI has a more pronounced influence on neonatal and fetal outcomes than on maternal labour mechanics, a finding that is consistent with our data. Enomoto et al. (2016) [14] similarly observed limited effects of low BMI on caesarean rates and labour outcomes, emphasizing that nutritional status may exert more influence on fetal growth than on labour progression.

#### **4.9 Integration with Existing Evidence**

Taken together, the patterns observed in this study including increased anaemia, abnormal gestational indices, growth restriction, and neonatal metabolic complications closely align with established literature showing the adverse perinatal consequences of maternal underweight. Where trends are evident but not statistically significant, such as preterm birth and neonatal sepsis, these directional patterns are still consistent with broader epidemiological findings, suggesting that the magnitude of effect may be subtle or influenced by sample size.

### **5. LIMITATIONS AND FUTURE DIRECTIONS**

This study has certain limitations that should be considered when interpreting the findings. The sample size was modest and recruitment was limited to a single tertiary care centre, which may restrict the generalisability of the results to other populations or healthcare settings. Although a comparative analysis was performed, the observational nature of the study limits the ability to infer causality between low maternal BMI and adverse pregnancy outcomes.

In addition, detailed assessment of potential confounding factors such as dietary intake, micronutrient status, physical activity, and socioeconomic determinants was beyond the scope of this study and may have influenced the observed associations. Variations in antenatal nutritional supplementation and adherence to dietary advice were also not systematically quantified.

Future research should focus on larger, multicentric prospective studies to validate these findings across diverse populations. Longitudinal studies incorporating detailed nutritional assessments, biochemical markers of micronutrient status, and structured nutritional interventions would provide greater insight into the mechanisms linking low maternal BMI with adverse outcomes. These findings may provide important direction for developing focused antenatal care interventions and informing public health policies designed to enhance maternal and neonatal outcomes among undernourished populations.

### **6. CONCLUSION**

The present study indicates that low maternal pre-pregnancy BMI is linked to a greater burden of adverse antenatal and neonatal outcomes. Women in the lower BMI categories exhibited higher rates of anaemia, insufficient gestational weight gain, abnormal amniotic fluid index patterns, non-reassuring fetal surveillance findings, and adverse neonatal outcomes, including reduced birth weight and increased metabolic complications. Importantly, stratified analysis within the underweight category revealed a graded increase in risk, with the most adverse outcomes observed among women with the lowest BMI.

These findings highlight the clinical significance of maternal nutritional status at the time of conception and throughout pregnancy. Early identification of underweight women and implementation of tailored nutritional counselling and monitoring during antenatal care may play a crucial role in mitigating adverse outcomes. Strengthening nutritional screening and support within routine maternal healthcare, particularly in regions with a high prevalence of undernutrition, has the potential to improve pregnancy outcomes and neonatal health. Further large-scale studies are warranted to strengthen the evidence base and inform effective, context-specific maternal nutrition interventions.

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- **Dr. Abirama Sundari S:** Concept and study design, data collection, data analysis, manuscript drafting and final approval.

- **Dr. Chellammal K R:** Supervision of study design, interpretation of data, critical manuscript review, and final approval of the submitted version.

- **Dr Yasodha S:** Supervision of study design, critical manuscript review, and final approval of the submitted version.

All authors meet the ICMJE criteria for authorship and take full responsibility for the integrity and accuracy of the work.

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