"A COMPARATIVE STUDY OF HEMATOLOGICAL AND INFLAMMATORY MARKERS IN PRETERM LABOR AND TERM LABOR"

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LIST OF ABBREVIATIONS USED

PTB	-	Preterm Birth
CBC	-	Complete Blood Count
CRP	-	C- reactive Protein
NLR	-	Neutrophil to Lymphocyte Ratio
PLR	-	Platelet to Lymphocyte Ratio
LMR	-	Lymphocyte to Monocyte Ratio
PPROM	-	Preterm Prelabor (premature) Rupture of Membranes
BV	-	Bacterial Vaginosis
sPTB	-	Spontaneous Preterm Birth
GWAS	-	Genome-Wide Association Studies
FSHR	-	Follicle-stimulating hormone receptor
EEFSEC	-	Eukaryotic elongation factor, selenocysteine tRNA-specific
PROM	-	Prelabor (premature) Rupture of Membranes
GBS	-	Group B Streptococcus
ROM	-	Rupture of Membranes
TLRs	-	Toll-like Receptors
IL-8	-	Interleukin 8
IL-1β	-	Interleukin 1 beta
TNF-α	-	Tumor Necrosis Factor alpha
Hb	-	Hemoglobin
HCT	-	Hematocrit
WHO	-	World Health Organization
MCV	-	Mean Corpuscular Volume
RDW	-	Red Cell Distribution Width
WBC	-	White Blood Cells
MPV	-	Mean Platelet Volume
PDW	-	Platelet Distribution Width
APPs	-	Acute phase proteins
SAA	-	Serum Amyloid A
ESR	-	Erythrocyte Sedimentation Rate
ELISA	-	Enzyme-Linked Immunosorbent Assay
hs-CRP	-	High-sensitivity CRP

PCT	-	Procalcitonin
AAT	-	Alpha-1 Antitrypsin
MCH	-	Mean Corpuscular Hemoglobin
MCHC	-	Mean Corpuscular Hemoglobin Concentration
ANC	-	Absolute Neutrophil Count
ALC	-	Absolute Lymphocyte Count
AMC	-	Absolute Monocyte Count
AEC	-	Absolute Eosinophil Count
PLT	-	Platelet
ANC	-	Antenatal care
ECLIA	-	Electrochemiluminescence immunoassay
ROC	-	Receiver operator characteristics
IQR	-	Interquartile Range
AUC	-	Area Under Curve

ABSTRACT

BACKGROUND

Preterm labor, defined as birth before 37 weeks of gestation, affects 5% to 18% of all deliveries worldwide. Combining routine clinical tests like CBC and inflammatory markers can help predict preterm labor, aiding in the better management of associated maternal and fetal complications.

OBJECTIVES

This study aims to detect the variations in haematological and inflammatory markers in preterm labor and term labor and to determine diagnostic significance of NLR, LMR & PLR.

METHODOLOGY:

In this study, all singleton pregnancies at KLE's Dr. Prabhakar Kore Hospital & MRC delivering before 37 weeks were classified as Preterm Labor, and those between 37-42 weeks as Term Labor. Clinical history and lab investigations, including CBC, CRP, and procalcitonin, were collected. Venous blood (3 mL) was drawn for these tests. NLR, LMR, and PLR were calculated manually, with 100 samples taken for each group.

RESULTS:

It was found that older age group, higher parity, history of preterm birth, premature rupture of membranes, cervical insufficiency and history of preeclampsia emerged as significant risk factors that can lead to preterm labor. Investigations like WBC count, MPV, CRP, PCT, NLR and LMR shows significant variations in preterm labor. NLR was found to have highest diagnostic significance in predicting preterm labor followed by LMR.

CONCLUSION:

This study identified significant risk factors for preterm labor, including older age, higher parity, history of preterm birth, PROM, cervical insufficiency, and preeclampsia. WBC count, MPV, CRP, PCT, NLR and LMR, showed strong diagnostic potential, with NLR being the most predictive. Combining these clinical and hematological markers can help in early detection and intervention for preterm labor.

Keyword- complete blood count, CRP, neutrophil-to-lymphocyte ratio, lymphocyte-to-monocyte ratio, platelet-to-lymphocyte ratio, preterm birth, preterm delivery, preterm labor,

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INTRODUCTION

Birth before 37 complete weeks of gestation is termed as Preterm Birth (PTB). It is a very serious obstetric problem worldwide. It affects 5% - 18% of all deliveries in the world and has become a burden on health care.[1]

Prematurity is one of the leading global causes of death in children under the age of 5 years.[2]

Preterm neonates are at increased risks of short-term complications due to the underdevelopment of several organ systems, along with potential long-term neurodevelopmental complications such as cerebral palsy, intellectual disabilities, and impairments in vision or hearing. [3] [4]

The rate of PTB is increasing day by day, in spite of the development in early diagnosis and rapid treatment in recent years.[5]

Various factors have led to a general increase in preterm birth rates. These include a higher utilization of assisted reproduction methods, a rise in the occurrence of multiple births, and an increase in obstetric interventions. [6]

Risk factors for premature birth include previous premature birth, advanced maternal age, low socioeconomic status, smoking, ethnicity of the mother, infection in pregnancy, hypertension and more.[2]

The multi factorial etiology of PTB has made its accurate prediction difficult. Complete blood count (CBC) is one of the most simple and routine clinical tests done during pregnancy.[7] There are various studies that shows changes in CBC parameters in preterm birth. Similarly, many inflammatory markers have also been studied which shows significant changes in preterm labor.

Various studies have shown that delivery pain triggers an inflammatory reaction within the body, leading to changes in absolute and relative counts of neutrophils and lymphocytes.

Alongside traditional markers like C-reactive protein (CRP), alternative indices such as neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) have been proposed as potential indicators of inflammation. [8]

Currently, there is a gap in literature where CBC and inflammatory markers are studied together to predict preterm birth. Also, no such studies have been done in Karnataka.

The simple tests like CBC and inflammatory markers can be used for early detection of preterm birth and can help in better management of the same. Early detection of preterm labor can prevent maternal and fetal complications associated with it.

Therefore, this study aims to find out the variations in haematological and inflammatory markers in preterm birth and to determine their diagnostic significance so they can be used for its early detection.

AIMS & OBJECTIVES

PRIMARY OBJECTIVE: To compare hematological and inflammatory markers in preterm labor and term labor.

SECONDARY OBJECTIVE: To find out diagnostic significance of NLR, PLR & LMR in preterm labor.

REVIEW OF LITERATURE

DEFINITION OF TERM LABOR

Onset of labor any time after completed 37 weeks of gestation and before 42 weeks of gestation is considered as term labor.[9],[10]

DEFINITION OF PRETERM LABOR

Onset of labor any time before 37 weeks of gestation is considered as preterm labor.[9],[10]

According to WHO, preterm labor, on the basis of gestational age of the patient, is subcategorised further into three categories:

- Extreme preterm (less than 28 weeks)
- Very preterm (28 to 32 weeks)
- Late preterm (32 to 37 weeks)

EPIDEMIOLOGY

It has been estimated that 5 to 18% of all pregnancies end up in preterm delivery which poses an extensive healthcare burden mainly due to neonatal morbidity and mortality. Consequently, approximately 15 million neonates are born prematurely each year worldwide and about one million of them do not survive. [11]

According to Foundation for Premature Babies (2013), India contributes 23.6% of the global preterm births, of which 13% are live preterm births, 28% are with low birth weight [12],[13], and babies born per year <28 weeks are 165,800 in India. Studies have reported that in India 33,41,000 babies were born preterm per year, the ratio of boys to girls born preterm is 1.23, impaired preterm survivors per year are 80,700, and direct preterm child death per year are 3,61,600. [14]

According to study conducted in April 2020, incidence of preterm births in state of Karnataka is 28.25%.[15],[16]

PRETERM NEWBORN MORBIDITY AND MORTALITY

Preterm birth affects newborn and mother in various ways. There can be long term or short-term complications which can affect the newborn.[9]

Preterm neonates who survive are at risk for a range of short term (within one week if birth) and long- term (adulthood) morbidities, largely due to organ system immaturity.

The various morbidities are listed in the following table:

System	SHORT TERM (NEONATAL)	LONG TERM (ADULTHOOD)
Respiratory	Respiratory distress syndrome Apnea episodes, bronchopulmonary dysplasia	Asthma, Bronchopulmonary dysplasia
GIT	Jaundice, Feeding problems, growth retardation, necrotising enterocolitis	Short bowel syndrome, growth retardation, cholestasis
Immunological	Immune weakness, Early infections, Infections	Respiratory syncytial viral infection
CNS	Hydrocephalus, intraventricular hemorrhage	Cerebral palsy, neurodevelopmental delay, hearing loss
CVS	Patent ductus arteriosus, Pulmonary hypertension, hypotension	Systemic hypertension
Kidney	Electrolyte imbalance	HTN
Others	Retinopathy of prematurity, cortisol deficiency	-

Table 1. Short term and long term complications of preterm birth. Table taken from Cunningham FG, Leveno KJ, Dashe JS, Hoffman BL, Spong CY, Casey BM, editors. Williams obstetrics. 26th edition. New York: McGraw Hill Medical; 2022. [9]

CAUSES OF PRETERM LABOR

Preterm labor has multi-factorial etiology.[9,10,14] It is can be either spontaneous or iatrogenic.[17–20]

Spontaneous labor leading to premature delivery and Preterm prelabor rupture of membranes (PPROM) due to any underlying cause is referred to as spontaneous preterm labor.[9,10,18]

When labor is induced medically due to either maternal or fetal indications, then it is called as iatrogenic preterm labor.

Approximately 30-35% of premature births are indicated medically, while 40-45% occur due to spontaneous early labor, and 25-30% are a result of PPROM. Those resulting from spontaneous labor and PPROM are collectively termed spontaneous premature births.[14]

Spontaneous preterm labor is linked to many factors such as maternal demographics, previous pregnancy history, risk of current pregnancy etc.

Maternal demographics can affect the length of gestation in a woman. [14]

Maternal age below 18 years or above 40 years, black race, low socioeconomic status, low educational status, poor nutrition or poor hygiene, underweight or obese, can cause preterm delivery. [9]

When there is gap of less than 6 months subsequent pregnancy, it increases the risk of preterm labor by more than two times. In one meta-analysis, intervals <18 months and >59 months were associated with greater risks for both preterm birth and small-for-gestational-age newborns. [9,14]

Most important factor related to spontaneous preterm labor is previous history of preterm delivery. Women having history of preterm delivery are said to have 1.5 times more chances of delivering preterm in current pregnancy.[9,14, 22]

History of previous still birth are also increases the risk of preterm delivery. [9,10,14,18,21]

Mercer and colleagues found that women with previous preterm deliveries have a 2.5 times higher risk of experiencing preterm labor in their subsequent pregnancy.[14,22]
Pregnancy achieved by artificial reproductive technology often leads to preterm labor.

Multifetal pregnancy causes over distension of uterus leading to early labor. It is seen in some research that almost 60% of twin pregnancies are delivered preterm.[9,10]

Another important risk factor for premature delivery is polyhydramnios and also oligohydramnios as they can also lead to PPROM.

Uterine anomalies, such as unicornuate uterus, rudimentary horn, and uterine duplication anomalies leads to preterm delivery. Congenital structural anomalies of uterus are known as mullerian defects. It comprises of various uterine and cervical anomalies.[18]

Depending on the exact deformity and obstetric history, women with uterine abnormalities have a 25% to 50% chance of PTB.[9]

Similarly, cervical length also affects the length of gestation period. Cervical length is measured by transvaginal ultrasound. A cervical length < 3cm, at 24 weeks of gestation, indicates high chances of preterm birth. [1,10,18]

Another study shows that cervical length <2–1.5 cm at 23–24 weeks' gestation is associated with increased risk of preterm birth.[18,23,24]

A history of previous medical or mechanical termination of pregnancy, or repeated terminations, increases the risk of subsequent preterm labor.

Additionally, having undergone a cervical cone biopsy or loop electrosurgical excision procedure of the cervical transformation zone raises the likelihood of preterm delivery.[14,25]

Fetal anomalies, including abdominal wall malformations, are known to lead to preterm birth. These congenital abnormalities in premature infants are associated with high rates of newborn morbidity.

Unexplained vaginal bleeding during the first or second trimester increases the risk of preterm birth, with the risk escalating with the frequency of bleeding episodes.[21]

Maternal conditions such as acute or chronic hypertension, anemia, history of seizures, thromboembolism, and connective tissue disorders can cause preterm delivery.[18]

Mothers experiencing stress and depression are more likely to have preterm births.

Domestic violence is also linked to causing preterm labor.

Smoking, alcohol, and substance abuse are associated with preterm delivery and fetal anomalies, with tobacco use doubling the chances of preterm birth.

Excessive physical activity is associated with an increased risk of preterm delivery, with prolonged walking or standing during the second trimester posing a moderate risk of preterm labor.[14]

Intrauterine infections are a major and frequent cause of preterm birth. Women experiencing spontaneous preterm labor with intact membranes often have microorganisms from the lower genital tract in their amniotic fluid, placenta, and membranes. These microorganisms include Ureaplasma urealyticum, Mycoplasma

hominis, Fusobacterium species, Gardnerella vaginalis, peptostreptococci, and Bacteroides species. [9,10,14,18]

The likelihood of clinical and histological intraamniotic inflammation and infection rises as gestational age decreases, especially before 30 to 32 weeks. Bacteria in the amniotic fluid, detected in 20% to 60% of preterm labor cases before 34 weeks, are more prevalent with decreasing gestational age—20% to 30% after 30 weeks and up to 60% at 23 to 24 weeks. Infection evidence is less common after 34 weeks.

Intrauterine infections can present in various ways, from affecting only the decidua (the lining of the uterus) to spreading between the amnion and chorion (the fetal membranes) and potentially reaching the amniotic cavity and the fetus itself.[14]

The amniotic cavity can become infected through several routes: ascending from the vagina and cervix which is the most common route, dissemination via the bloodstream through the placenta, accidental introduction during invasive procedures, and retrograde spread through the fallopian tubes.

While the ascent is generally believed to occur during the second trimester, the exact timing is uncertain. Some women may have asymptomatic endometrial colonization before pregnancy. Symptomatic infection and early preterm birth typically occur around 20 weeks when the fetal membranes adhere to the decidua, forming an abscess.[14,26]

Bacterial vaginosis (BV) is a condition characterized by an alteration in the vaginal microbial balance, with gram-negative anaerobic bacteria (such as Gardnerella vaginalis, Bacteroides, Prevotella, Mobiluncus, and Mycoplasma species) displacing the usual dominant lactobacilli.[10,14]

BV is associated with a twofold increased risk of spontaneous preterm birth (sPTB), and this association is particularly pronounced when BV is detected early in pregnancy. However, the use of antibiotics to treat BV does not consistently reduce the risk of sPTB.

Infections occurring outside the genital tract, such as urinary tract and intraabdominal infections (e.g., pyelonephritis and appendicitis), have also been linked to a heightened risk of sPTB. The presumed underlying mechanism is the inflammation of nearby reproductive organs, but chronic infections at distant sites can also elevate the risk of sPTB.

Peridontal diseases, many oral bacteria are found in amniotic fluid like Fusobacterium nucleatum.

Genome-Wide Association Studies (GWAS) have identified significant associations between preterm birth and four genes: follicle-stimulating hormone receptor (FSHR), insulin-like growth factor 1 receptor, protein col-52, and serpin peptidase inhibitor, clade B, member 2. Additionally, Zhang et al. found that the gene EEFSEC (eukaryotic elongation factor, selenocysteine tRNA-specific) is significantly linked to both gestational length and PTB risk. These discoveries highlight the genetic complexity of PTB and the utility of GWAS in uncovering crucial genetic contributors.[9]

Premature rupture of membranes (PROM) is defined as rupture of membranes before the onset of labor. When membrane rupture occurs before labor and before 37 weeks of gestation, it is referred to as preterm PROM (PPROM).[27]

The incidence of PROM ranges from about five to ten percent of all deliveries. Preterm PROM occurs in approximately 3% of all pregnancies and causes about one third of preterm births. The prevalence of PPROM ranges from 2.2% - 4% in India.[28]

Iatrogenic preterm birth occurs when labor is induced or when a caesarean section is performed before the onset of labor or the rupture of membranes. [29]

According to reports, iatrogenic preterm delivery constitutes approximately 30%–35% of all preterm deliveries.[30]

The reasons for iatrogenic preterm delivery can differ by geographic region but can generally be categorized into four main groups[30]:

- Obstetric complications, which include conditions like hypertensive disorders of pregnancy, placental issues, preeclampsia, and antepartum bleeding.
- Fetal factors, such as fetal distress, limited fetal growth, and structural abnormalities.
- Maternal medical conditions, including heart disease, kidney problems, cancer, and sepsis.
- Iatrogenic preterm delivery that is not medically necessary.

With serious complications in pregnancy, such as preeclampsia, placenta previa and reduced fetal growth, preterm delivery may have to be induced, being examples of iatrogenic preterm delivery. It is reported that about 20%of preterm births are iatrogenic. [18]

PATHOPHYSIOLOGY OF PRETERM LABOR

Term and preterm parturition both involve common anatomical, physiological, and biochemical features within the parturition pathway. This pathway encompasses cervical changes, membrane and decidual activation, and increased uterine contractility.

However, a critical distinction exists between the two. Spontaneous labor at term occurs due to the physiologic activation of this common pathway. In contrast, preterm labor arises from a pathologic activation of the same pathway.

The underlying insult responsible for this activation may lead to asynchronous recruitment of different components within the pathway.

Asynchrony can manifest as

(1) preterm uterine contractions when impacting the myometrium,

(2) cervical insufficiency when primarily affecting the cervix, or

(3) PPROM when acting on the chorioamniotic membranes. [10]

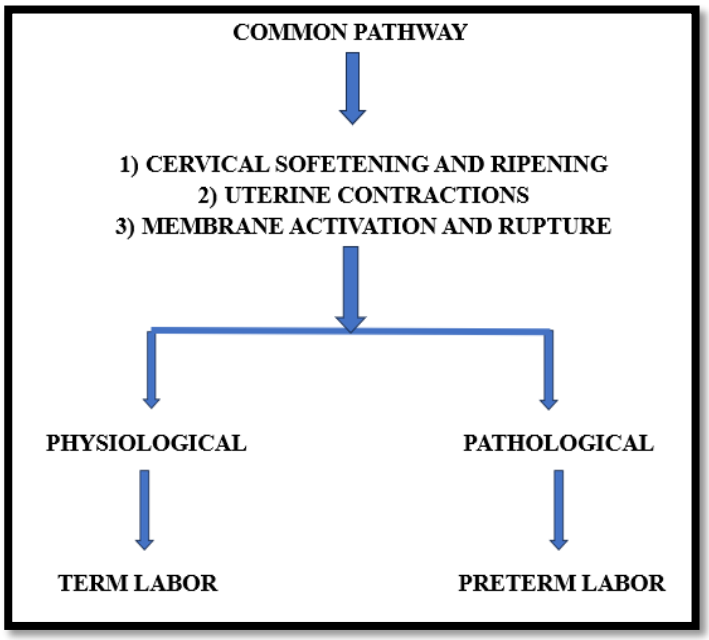

Figure 1. Common Pathway of Parturition

There are 3 main underlying causes which might lead to preterm labor.

1) Spontaneous
2) PPROM
3) Iatrogenic

Various causes discussed above will lead to spontaneous preterm labor. Pregnancies with spontaneous preterm labor yet intact fetal membranes must be distinguished from those complicated by ruptured membranes.[9]

Spontaneous preterm labor can be attributed to the disruption of the normal mechanisms that maintain uterine quiescence. This disruption may stem from the early formation of gap junctions, along with an elevation in oxytocin receptor levels. Three primary mechanisms are responsible for early uterine contractions:

1. Uterine stretching (phase 1), which can be triggered by factors such as multiple fetuses or polyhydramnios.

2. Activation of the hypothalamic–pituitary–adrenal axis in the fetus, prompted by compromised uteroplacental blood flow and hypoxia.

3. Inflammatory responses at the choriodecidual interface, induced by factors like hemorrhage, trauma, or infection. These responses lead to the production of cytokines and increased levels of prostaglandins.

Overall, these mechanisms contribute to the onset of preterm labor by prompting premature uterine contractions.[9,18]

The cervix is crucial in pregnancy and parturition. Maintaining its structure and function as a barrier during pregnancy is important. However, biochemical and biomechanical changes leading to cervical ripening are essential for delivery. The molecular processes involved in cervical ripening differ between physiological and pathological parturition and may vary among the causes of pathological parturition.[10]

Cervical insufficiency can lead to various complications such as second-trimester abortions or premature delivery.[31]

Reasons for cervical insufficiency includes congenital issues like cervical hypoplasia, exposure to a substance called diethylstilbesterol while pregnancy, past cervical surgeries like conization, multiple dilatations during abortions or Infections. [31]

Cervical epithelial dysfunction or issues with the stromal extracellular matrix can underlie preterm birth. For example, the absence of hyaluronan or the presence of group B streptococcus (GBS) in cervical tissue increases the risk. GBS produces hyaluronidase, which helps it ascend through the cervix.

Furthermore, genetic mutations affecting collagen, elastic fibers, or their related proteins can weaken the cervix, leading to cervical insufficiency and preterm birth.[9]

In general, the shorter the cervix, the greater the risk of preterm delivery. [18]

A cervical length of less than 2–1.5 cm at 23–24 weeks gestation is associated with an increased risk of preterm birth.[24] Cervical length measurement along with detection of funnelling can predict preterm delivery.[32] Routine transvaginal sonography of the cervix performed between 18 and 22 weeks can help identify patients at risk of preterm delivery. [10,18]

Prelabor rupture of membranes (PROM) is defined as rupture of membranes before the onset of labor. When membrane rupture occurs before labor and before 37 weeks of gestation, it is referred to as preterm PROM (PPROM).[27]

The incidence of PROM ranges from about five to ten percent of all deliveries. Preterm PROM occurs in approximately 3% of all pregnancies and causes about one third of preterm births. The prevalence of PPROM ranges from 2.2% - 4% in India.[28]

Spontaneous preterm birth and PPROM exhibit several overlapping features:

1) They share common etiological factors;

2) Both conditions are associated with infections

3) Infection-induced inflammation and specific biochemical markers contribute to their underlying pathophysiology;

4) Clinical and histological evidence often reveals chorioamnionitis in cases of both sPTB and PPROM.

However, while approximately 40% of women experience membrane rupture prior to the initiation of labor, the majority do not undergo rupture of membranes (ROM).

The compromised immune and mechanical properties of fetal membranes allow microbial invasion from the genital tract, triggering an inflammatory response that leads to mechanical disruption and weakening of the membranes, predisposing them to PPROM.[33,34]

PPROM is characterized by evident fetal membrane dysfunction, distinguishing it from spontaneous preterm birth without membrane rupture. Approximately 70% of PPROM cases are associated with intraamniotic infection, confirmed by positive amniotic fluid cultures or clinical signs of infection.[25]

Recent findings suggest that PPROM may also involve sterile inflammation in fetal membranes, mimicking infection but without microbial presence. Risk factors like smoking and bleeding may induce this inflammation, leading to an immunocompromised state that facilitates microbial invasion. Infection in PPROM is likely a consequence rather than a cause.[35,36]

Degradation of the collagen-rich extracellular matrix connecting the amnion and chorion layers is a crucial event in ROM. [35,37] Matrix metalloproteinases play a key role in this process, regulated by tissue-specific inhibitors of metalloproteinases. In PPROM, proteases associated with infection or inflammation can activate matrix metalloproteinases, leading to collagen turnover and membrane weakening.[35,38,39]

Bacteria can enter intrauterine tissues by

- transferring a maternal systemic infection through the placenta,

-spreading the infection through the fallopian tubes, or

- ascending through cervix and vaginal.

Current research indicates that infection-mediated preterm birth can be caused solely by microbial invasion of the reproductive system. Women who are affected may experience membrane rupture and develop symptomatic chorioamnionitis. [9]

Responses to bacterial toxins, especially lipopolysaccharide, which is recognised by receptors such as toll-like receptors (TLRs), are the reason for infection-induced preterm labor. Numerous cells, including as trophoblasts, decidual cells, cervical epithelia, and mononuclear phagocytes, have these receptors. Identification of inflammatory stimuli by maternal immune cells expressing TLRs is important in inflammation-mediated premature delivery.

Activation of Toll-like receptors (TLRs) trigger a series of events culminating in the generation of chemokines such as interleukin 8 (IL-8) and cytokines like interleukin 1 beta (IL-1β). This sequence recruits immune cells to the reproductive tract. The presence of lipopolysaccharide induces IL-1β, prompting various responses including heightened production of IL-6, IL-8, and tumor necrosis factor alpha (TNF-α), activation and movement of leukocytes, alterations in extracellular matrix proteins, and fever, among other effects. IL-1β also promotes prostaglandin formation in various tissues, inducing cervical ripening and loss of myometrial quiescence.

Inflammatory cytokines prompt the activation of proteases such as matrix metalloproteinases, which break down components of the extracellular matrix. This process disrupts the structural stability of fetal membranes or the cervix.[9]

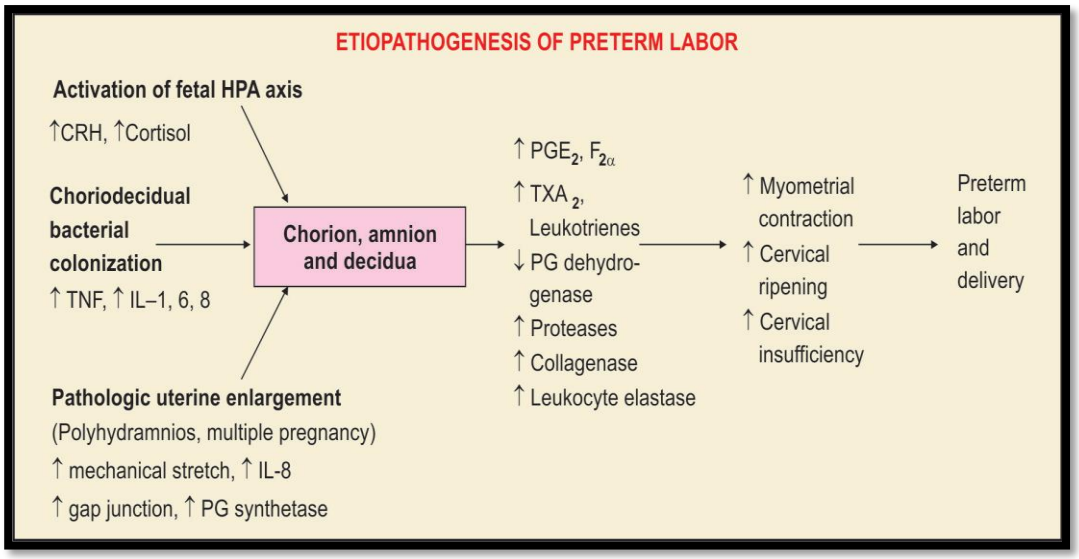

Figure 2. Preterm Labor Pathogenesis. Image taken from Dutta DC, Konar H. DC Dutta's textbook of obstetrics: including perinatology and contraception. Eighth edition. New Delhi: Jaypee, The Health Sciences Publisher; 2015, pg 366 [40]

HEMATOLOGICAL AND INFLAMMATORY MARKERS

PLASMA VOLUME & RED CELL MASS

During pregnancy, the total blood volume typically increases by approximately 1.5 liters, primarily to meet the demands of the developing vascular system and to compensate for potential blood loss during delivery.[35,41]

It also shields both the mother and fetus from the adverse effects of diminished venous return in both supine and upright positions. [9]

The process of maternal blood volume expansion initiates around the sixth week of pregnancy and continues steadily until it stabilizes between the 30th and 34th weeks. On average, this expansion ranges from 40% to 50%.[10,42]

This expansion of blood volume is more significant in cases of multiple pregnancies and in individuals with iron deficiency. [43]

By approximately the 10th week of pregnancy, the red cell mass also increases due to increased erythropoietin, coupled with moderate expansion in erythroid cells within the bone marrow and a slight increase in the reticulocyte count. [40]

But this increase is not as much as the rise in plasma volume due to which, the concentration of Hemoglobin (Hb) and Hematocrit (HCT) decreases, causing dilutional anemia. [9,10,40]

The drop in hemoglobin is typically by 1–2 g/dL by the late second trimester and stabilizes thereafter in the third trimester, when there is a reduction in maternal plasma volume (owing to an increase in levels of atrial natriuretic peptide). [43]

Iron supplementation causes red blood cell mass to rise upto 400 to 450 mL, equivalent to a 30% increase, leading to increased hemoglobin levels. [9]

Parameters (unit)	I Trimester (N=87)	II Trimester (N=99)	III Trimester (N=112)	ANOVA (p value)
Blood Sugar	92.43±6.81	92.4±10.2	94.07±9.17	0.1726 (NS)
Hb (gm/dl)	10.48±0.89	10.06±1.04	10.02±1.26	0.1721 (NS)
RBC(million/cumm)	4.003±0.42	4.067±0.24	4.157±1.83	0.1837 (NS)
PCV (%)	37.51±2.6	32.88±2.96	33.7±3.27	0.0080 (S)*
RDW	14.07±1.01	15.07±2.43	18.9±3.85	0.0083 (S)*
Platelet count	3.33±0.63	3.12±3.99	2.54±0.43	0.0073(S)*
BLOOD INDICES				
MCV (cumm)	81.86±7.43	82.89±8.35	85.69±13.9	0.6562 (NS)
MCH (pg)	26.15±2.86	26.96±3.31	27.31±2.54	0.2217 (NS)
MCHC (%)	31.49±2.87	32.99±1.97	30.47±6.65	0.8049(NS)
ABSOLUTE AND DIFFRENTIAL LUCOCYTE COUNT				
TLC (/cumm)	7846.88±1414.9	9700±2427.8	10166±2114.34	0.001 (HS)**
Neutrophils (%)	63.44±9.09	73.77±7.22	70.66±9.05	0.001 (HS)**
Lymphocytes (%)	28.5±8.47	19.65±5.91	19.89±6.81	0.001 (HS)**
Eosinophils (%)	3.75±0.44	3.58±1.13	3.60±1.37	0.0493(NS)
Monocytes (%)	4.31±0.59	3.38±0.81	3.40±1.10	0.001(HS)**

Figure 3. Hematological parameters through all three trimesters during pregnancy. Image taken from Purohit G, Shah T, Harsoda DJM. Hematological profile of normal pregnant women in Western India. Scholars Journal of Applied Medical Sciences, 2015; 3(6A):2195-2199 [44]

According to the World Health Organization (WHO), pregnant women are classified as anemic if their hemoglobin levels are below 11.0 g/dl in the first and third trimesters, and below 10.5 g/dl in the second trimester.[45]

During pregnancy, red blood cell indices typically stay stable with minor changes. Mean corpuscular volume (MCV) increases slightly, averaging around 4 femtoliters in women

with adequate iron levels, peaking at 30–35 weeks of gestation. This rise is due to increased red blood cell production and a higher proportion of larger, young red blood cells, not vitamin B12 or folate deficiencies. Significant changes in MCV are uncommon. However, if hemoglobin levels drop below 9.5 g/dL and MCV is less than 84 fl, it may indicate iron deficiency or other health issues. [43,46]

In women experiencing preterm labor, both hemoglobin and hematocrit levels are observed to rise. One possible explanation for the elevated hb levels in the second trimester and the increased risk of preterm birth is insufficient plasma volume expansion. This may lead to increased blood viscosity, reduced placental blood flow, and impaired fetal development. [1]

Red cell distribution width (RDW) is higher in late preterm pregnancies compared to full-term pregnancies. This suggests that high RDW values, particularly in preterm pregnancies, indicate unstable erythropoiesis and/or stress conditions..[5]

IRON

During pregnancy, iron demand increases to around 1000 mg, with 500 mg for maternal red blood cell mass, 300 mg for the fetus, and 200 mg for daily losses. This requires an average absorption of 3.5 mg of iron per day. Iron needs escalate from 0.8 mg/day in the first trimester to 6-7 mg/day in the third trimester. Maternal iron deficiency anemia is linked to adverse outcomes like low birthweight and preterm birth.[9]

WBC COUNT

During labor, neutrophilic leukocytosis can reach levels as high as 13,000/mm3 due to elevated estrogen and cortisol. Pregnancy shifts immune responses towards humoral and innate immunity. Administering betamethasone increases neutrophil count by 35% and decreases lymphocyte count by 45%, with a mean total white blood cell count of 13 ×

10^9/L, not exceeding 20 × 10^9/L. Neutrophil rise peaks 24 hours post-administration and can last up to 5 days. Immature white blood cells like myelocytes and metamyelocytes are present during pregnancy. Lymphocyte count decreases in the first two trimesters, then rises in the third, while monocyte levels initially rise, then decline. Eosinophil and basophil count remain stable. These changes persist for about 6 to 8 weeks post-delivery. [40]

Increases in white blood cells in pregnant women can occur in a number of conditions, such as sepsis, thrombotic thrombocytopenic purpura, hemolytic-uremic syndrome, low platelet count (HELLP syndrome), severe preeclampsia, hemolysis, elevated liver enzymes, and acute fatty liver of pregnancy.

PLATELETS

Platelet count declines during gestation, possibly due to increased destruction or haemodilution, dropping notably in the third trimester. Approximately 8% of pregnant women develop gestational thrombocytopenia, with platelet counts between 70,000 and 150,000/mm3 in the third trimester, not associated with increased pregnancy complications. Platelet counts return to normal within 1 to 2 weeks post-childbirth. [9,10,47]

Despite the decrease in platelet count, there are alterations in platelet morphology and interactions with erythrocytes, leading to increased platelet aggregability. [10]

In general, dilutional thrombocytopenia exists with an compensatory increase in MPV and platelet distribution width (PDW) levels during the pregnancy. (3, 49–52)

Gasparyan et al. proposed that MPV, serving as a prothrombotic or proinflammatory agent, is gaining prominence across various diseases. Platelet indices have been found to be altered in obstetric conditions like recurrent pregnancy loss, preeclampsia, gestational diabetes, and preterm labor. (5, 53)

Ahmed et al. suggested that pregnant women with high MPV in the second, and third trimester are at risk of development of preeclampsia.[53] In a study performed by Myatt et al., reported that MPV was significantly higher in the first trimester of women who developed preeclampsia.[54]

COAGULATION PROFILE

Pregnancy elevates thromboembolic disease risk five to six times due to factors such as venous stasis, vessel wall injury, and alterations in the coagulation cascade, leading to hypercoagulability. Venous stasis stems from pressure on pelvic veins and the inferior vena cava by the expanding uterus. Hypercoagulability arises from increased procoagulants, decreased natural coagulation inhibitors, and reduced fibrinolytic activity. These changes are crucial for controlling bleeding during childbirth.[10]

During pregnancy, coagulation factors I, VII, VIII, IX, and X increase notably, while II, V, and XII remain relatively stable. Factors XI and XIII decrease. Fibrinogen levels rise progressively, peaking at 50% higher than pre-pregnancy levels in the third trimester, accompanied by an increase in the erythrocyte sedimentation rate. The fibrinolytic system is diminished, leading to reduced circulating plasminogen activator levels, a two- to threefold increase in plasminogen activator inhibitor 1, and a 25-fold increase in plasminogen activator inhibitor -2, primarily sourced from the placenta. Protein S levels decline steadily, particularly free protein S, while protein C and antithrombin III levels remain stable during pregnancy. Most coagulation tests are unaffected by pregnancy. Prothrombin time, activated partial thromboplastin time, and thrombin time, Bleeding time and whole blood clotting times remain unchanged. Coagulation factor levels return to normal around two weeks after childbirth.

Previous research suggested a link between hypercoagulation and preterm labor. However, a recent study revealed that women with preterm labor and placental umbilical cord abnormalities had significantly shorter prothrombin time and activated partial thromboplastin time compared to those with normal term deliveries. This indicates activation of both the intrinsic and extrinsic pathways of coagulation during preterm labor..[55]

ACUTE PHASE REACTANTS

Acute phase proteins are plasma proteins produced by the liver that experience alterations in concentration in reaction to infection, tissue damage, and inflammation. [56]

Following infection, the liver rapidly adjusts its protein synthesis. There is notable rise in certain blood proteins known as positive acute phase proteins (APPs), like C- reactive protein (CRP), fibrinogen, haptoglobin etc. This increase in hepatic mRNA expression of these positive APPs coincides with a decrease in the production of regular blood proteins, such as transthyretin, retinol binding protein , cortisol binding globulin, transferrin, and albumin, which are categorized as negative APPs. [57]

Positive acute phase proteins play crucial roles in bolstering the body's defence mechanisms against microorganisms and their byproducts. They aid in opsonization and trapping of pathogens, activate the complement system, bind cellular debris like nuclear components, neutralize enzymes, scavenge free hemoglobin and radicals, and modulate the host's immune response. [57,58]

C- REACTIVE PROTEIN	α_1 ACID GLYCOPROTEIN
FIBRINOGEN	SERUM AMYLOID A
HAPTOGLOBIN	FERRITIN
PROCALCITONIN	α_1 - ANTITRYPSIN

Table 2. Positive Acute Phase Reactants

Changes in acute phase protein levels mainly result from alterations in hepatocyte production. The extent of increase varies, with CRP and serum amyloid A (SAA) experiencing around a 50% rise. Hepatocytes synthesize and release APPs under the influence of interleukins (e.g., IL-1, IL-2) and tumor necrosis factor-alpha (TNF-α).[58]

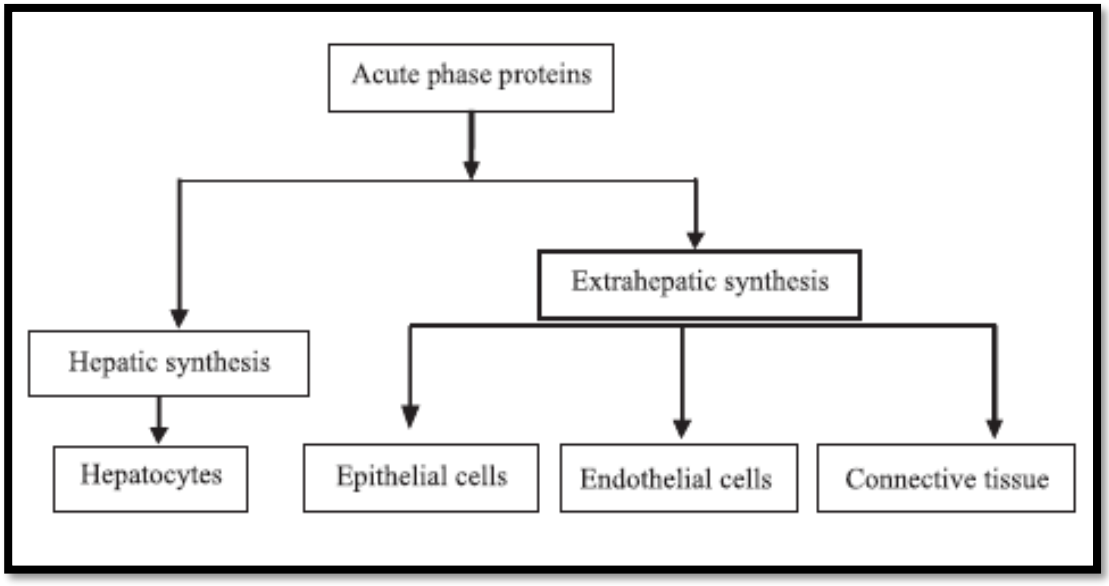

Figure 4. Acute phase reactants production. Image taken from Jain S, Gautam V, Naseem S. Acute-phase proteins: As diagnostic tool. J Pharm Bioallied Sci 2011;3(1):118. [58]

C REACTIVE PROTEIN

C-reactive protein ,CRP , is a ring consisting of five 23 000 Da units (pentraxin), which is the first described acute phase protein.[58,59]

It was discovered in 1930, found to bind to C-polysaccharide in the cell wall of Streptococcus pneumonia; hence the name C-reactive protein. [56]

It binds directly to several microorganisms, degenerating cells and cell remnants, and activates complement by the classical C1q pathway, and acts as opsonin.[58]

CRP is primarily synthesized in the liver in response to interleukin-6 from macrophages, serving as a sensitive inflammation marker. Its levels rise quickly and stay elevated

during inflammation. Measuring CRP is a straightforward, non-invasive, and safe method for assessing and predicting risk and morbidity for both mother and fetus. Research shows that elevated CRP levels are associated with a 1.45 to 2.55 times higher risk of preterm delivery. [60–62]

CRP tests are available for serum estimation and are preferred among acute phase reactants. CRP levels rise earliest and return to normal quickly after successful therapy, unlike other acute phase proteins. Hormones or drugs do not affect CRP levels, and no deficiency state exists. CRP levels rise more significantly than other reactants and correlate well with ESR in acute disease. Compared to ESR, CRP is earlier to rise and return to normal, more sensitive, and more specific, unaffected by anemia, polycythemia, red cell shape alterations, and paraproteinemia.[56]

Normal levels of CRP is expected to be < 5.0 mg/L.[63]

CRP levels typically increase rapidly, often surpassing 10 mg/l along with a simultaneous elevation in erythrocyte sedimentation rates (ESR).[64,65]

Recent advancements have introduced advanced techniques for accurately measuring CRP levels with high sensitivity and rapid results. These methods include immunonephelometry, immunoturbidimetry, high-sensitivity enzyme-linked immunosorbent assay (ELISA), and resonant acoustic profiling, capable of detecting CRP within a sensitivity range of 0.01 to 10 mg/l. [64–66]

These highly sensitive assays are particularly useful for detecting subtle levels of systemic inflammation, even when clear signs of systemic inflammatory or immunologic disorders are absent. High-sensitivity CRP (hs-CRP) has been extensively researched as a biomarker to better predict overall cardiovascular disease risk.[64]

Study done by Watts et al. revealed CRP values for women who were not in labor ranged from 7- 9 mg/L, with 95% under 15 mg/L. For women in labor at term, the median CRP was 13 mg/L, with 32% over 15 mg/L. CRP values in normal pregnancies are higher than in nonpregnant individuals and increase further during labor. Understanding how CRP levels change during normal pregnancy and labor can help determining its use in complicated pregnancies. [67]

Studies showed that a marked correlation of elevated CRP in women with preterm delivery was observed when compared to women without preterm delivery. [62,68]

Farzaneh et al also established that hs-CRP inflammatory parameters were significantly higher in mothers with preterm labor than those with full-term deliveries.[8]

PROCALCITONIN

Procalcitonin (PCT), a 14.5 kDa peptide, is stimulated by cytokines such as IL-6, IL-1, and TNF-alpha. It is mainly produced by thyroid parafollicular C cells and converted to calcitonin. During inflammation, PCT can also be synthesized in various tissues, entering the bloodstream directly. This makes PCT a sensitive marker for detecting infection progression.[69]During normal pregnancy, extra villous trophoblast and decidual stromal cells also produce PCT.[70] Around delivery, physiological and immune changes in pregnant women induce inflammatory markers like PCT.

Different studies done by Paccolat et al., and Joyce et al. in clinically well pregnant women, determined that the PCT decision threshold of 0.25 ng/ml could be used during the third trimester, at delivery and the immediate postpartum to rule out infection.[71,72]

For healthy term pregnancy, PCT of 0.04 ng/ml is considered normal.[71]

PCT concentrations of 0.5– 2.0 ng/ml are indicative of possible systemic infection, with values between 2.0 and 10 ng/ml highly indicative of sepsis.[73]

FIBRINOGEN

Fibrinogen, a blood plasma protein, is essential for blood clot formation and inflammation. In nonpregnant women, fibrinogen levels average 300 mg/dL, ranging from 200-400 mg/dL. During pregnancy, these levels rise by about 50%, averaging 450 mg/dL in late pregnancy (range: 300-600 mg/dL). This increase in fibrinogen, along with elevated plasma globulin, significantly elevates the erythrocyte sedimentation rate (ESR) in normal pregnancy. [9]

They found that low fibrinogen levels increase the risk of postpartum hemorrhage, overt DIC, and the need for transfusions. Additionally, low fibrinogen levels can lead to adverse neonatal outcomes. Predelivery fibrinogen levels can predict placental abruption severity and potential risks for mother and baby.[74]

ERYTHROCYTE SEDIMENTATION RATE (ESR)

The ESR measures how quickly erythrocytes settle in anticoagulated blood and is elevated in many organic diseases. While not specific or diagnostic for any particular disease, ESR helps differentiate functional from organic disease, indicating the presence of an underlying condition requiring evaluation. Most inflammatory and neoplastic diseases show increased ESR, which correlates with disease activity. Thus, ESR is useful for monitoring and assessing therapy response in conditions like acute rheumatic fever, bacterial endocarditis, tuberculosis, rheumatoid arthritis, temporal arteritis, polymyalgia rheumatica, and Hodgkin's disease.[56]

During pregnancy, the ESR is elevated due to increased circulating fibrinogen and plasma expansion, as well as decreased hemoglobin concentration from anemia, which promotes rouleaux formation. [75]

In preterm labor, especially with underlying etiology as preterm premature rupture of the membranes (PPROM), an increase in erythrocyte aggregation is found in the peripheral blood.[76]

SERUM AMYLOID A (SAA), Apolipoprotein SAA is linked with high-density lipoprotein and is classified as an acute phase protein. It plays multiple roles, including transporting cholesterol to the liver for bile secretion, recruiting immune cells to inflammatory sites, and inducing enzymes that degrade amyloidosis, atherosclerosis, and rheumatoid arthritis.[69]

Studies have shown that SAA levels remains unaffected in preterm labor.[77]

FERRITIN serves the critical function of binding iron to hinder microbial iron acquisition. During malignancy or infection, ferritin levels increase to limit the availability of free iron for tumor cells or pathogens, respectively. Its expression is heightened by proinflammatory cytokines. [69]

As pregnancy is said to be an inevitable state of iron deficiency anemia, serum ferritin levels are expected to be lower during pregnancy.[40] However, increased serum ferritin levels in the second trimester can predict early spontaneous preterm delivery. This could be due to an acute-phase response to subclinical infections, which are closely linked to premature delivery.[78]

ALPHA-1 ANTITRYPSIN (AAT), is a type of serine protease inhibitor, classified as a serpin, responsible for inhibiting the action of neutrophil elastase. Its primary function is to safeguard cells from the deleterious effects of neutrophil elastase activity. Deficiency in AAT can result in conditions such as hepatitis, liver cirrhosis, and panacinar emphysema.[69]

NEUTROPHIL TO LYMPHOCYTE RATIO (NLR)

A new parameter of immune inflammatory reaction and neuro-endocrine stress was established twenty years ago, which is now known as a neutrophil-to-lymphocyte ratio (NLR).

Neutrophils are central to the innate immune response, engaging in phagocytosis and releasing various cytokines and mediators. Decrease in lymphocyte count signifies stress, while inflammation results from demargination, redistribution, and accelerated apoptosis.

The NLR reflects the balance between innate and adaptive immunity, serving as a strong indicator of both inflammation and stress. [79]

Recently, easily calculable ratios have emerged as dependable predictive and prognostic markers across various conditions.[80]

It has been studied in diseases like acute appendicitis, acute pancreatitis, sepsis, systemic inflammatory response syndrome, coronary diseases, various cancers like gastric carcinoma or colorectal carcinoma, various gynaecological diseases etc.[79]

NLR is a highly sensitive yet less specific hematologic parameter that mirrors the degree of systemic infection, inflammation, stress, and the severity of diseases originating from diverse causes, including COVID-19 infection. The patients with COVID-19 infection had significantly higher values of NLR than non-COVID patients. [79]

Studies have demonstrated that a higher NLR is associated with a higher mortality rate in those with coronary artery disease.[81]

The NLR has demonstrated prognostic and predictive significance in numerous studies related to both pregnancy and non-pregnancy conditions. Elevated NLR levels have been documented in conditions such as intrahepatic cholestasis [82], hyperemesis gravidarum[83], and preeclampsia[84]. Moreover, studies indicate higher NLR values in

instances of spontaneous preterm labor and PPROM, showcasing its utility in predicting preterm labor.[80,85]

The neutrophil-to-lymphocyte ratio was calculated on the basis of absolute peripheral granulocyte (as a representative for the neutrophil count) and lymphocyte blood counts, using the formula: NLR = N/L. [86]

A study done on Indian population found normal mean value of Neutrophil Lymphocyte Ratio to be 1.9±0.6, (i.e. 1.3-2.5) [87]

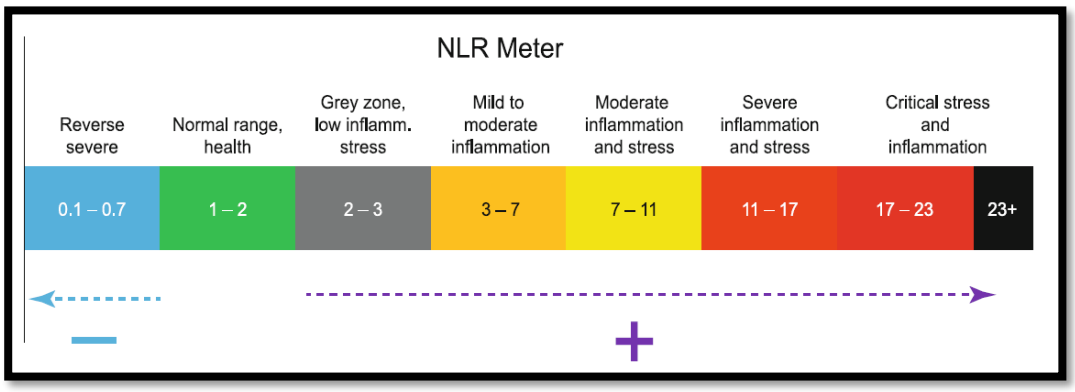

Figure 5. Image shows cut off values for NLR with varying degree of inflammation. The NLR-meter assesses immune-inflammatory responses and stress levels using the NLR. An NLR of 2.3–3.0 falls within a "grey zone" suggesting mild inflammation. NLR values of 3–7 indicate mild-to-moderate inflammation, while 7–11 suggest moderate to severe inflammation. NLRs of 11–17 point to severe conditions such as sepsis. An NLR of 17–23 reflects critical immune-inflammatory reactions, and NLRs of 23 or higher signify severe systemic inflammation, which can occur in situations like major surgery or terminal cancer. Image taken from Zahorec R. Neutrophil-to-lymphocyte ratio, past, present and future perspectives. Bratisl Med J 2021;122(07):474–88. [79]

PLATELET TO LYMPHOCYTE RATIO (PLR)

PLR is a ratio between the total platelet count and the absolute lymphocyte count.[88]

Chronic inflammation often leads to increased production of megakaryocytes and decreased levels of lymphocytes due to significant cell death. Consequently, common markers derived from complete blood counts, such as the platelet-to-lymphocyte ratio, can be influenced in severe chronic inflammatory conditions.

PLR, being easily accessible and efficient, has been suggested as a valuable predictive and prognostic parameter for various diseases including cardiovascular conditions and cancers. Additionally, research has established connections between PLR and gestational diabetes mellitus, acute appendicitis, preeclampsia, recurrent pregnancy loss, and preterm labor in expectant mothers. [85]

The platelet-to-lymphocyte ratio was calculated on the basis of peripheral platelet and lymphocyte blood counts, using the formula: PLR = P/L.[86]

PLR normal 36.63-149.13 for males, and between 43.36-172.68 for females. [89]

The mean PLR in Indian population is taken to be 91.77±26.95, (i.e. 64.82-118.72). [87]

LYMPHOCYTE TO MONOCYTE RATIO (LMR)

LMR has been suggested as an alternative marker for inflammation and is considered significant for prognosis. Additionally, abnormal counts of monocytes or lymphocytes have been associated with negative outcomes in various diseases. [90]

Elevated LMR has been recognized as a negative prognostic factor in conditions such as gastric cancer [91] and early-stage Hodgkin lymphoma. [92,93]

In obstetrics, numerous studies have investigated the relationship between CBC parameters and obstetric complications, including the predictive capacity of CBC for preterm birth. [90]

Nulliparous women who experienced miscarriage were found to have higher levels of autophagy in peripheral blood mononuclear cells compared to those with normal pregnancies, as observed by Osmanağaoğlu, M.A . [96- 97]

The lymphocyte-to-monocyte ratio was calculated on the basis of Lymphocyte and Monocyte blood counts, using the formula: LMR = L/M

Lymphocyte-to-Monocyte Ratio. normal 3.46 to 26.67 [89]

METHODOLOGY

Source of Data: KLE's Dr. Prabhakar Kore hospital & MRC, Belagavi.

The clinical data of the pregnant females visiting the hospital is collected. The investigations are performed in the laboratory. These investigations are Complete hemogram, CRP and procalcitonin.

Study Design: A CROSS-SECTIONAL STUDY

Study Period: January 1st, 2023 to December 31st,2023

Sample Size: 100 preterm pregnant female samples and 100 term pregnant female samples were collected before delivery.

Inclusion Criteria:

All singleton pregnancies, registered at antenatal care of KLE's Dr. Prabhakar Kore Hospital & MRC, and are delivering between 28-37 weeks of gestation are taken as PRETERM LABOR.

All singleton pregnancies, registered at antenatal care of KLE's Dr. Prabhakar Kore Hospital & MRC, who are delivering after completion of 37 weeks of gestation (but before 42 weeks) are taken as TERM LABOR.

Exclusion Criteria:

Females having Chronic inflammatory disorders (including Crohn's disease, Ulcerative colitis and Rheumatic arthritis), heart diseases, history of antibiotics and drug affecting platelets, are excluded.

Data collection procedure:

Pregnant females who are registered at obstetric and gynaecological dept of KLE' s Dr. Prabhakar Kore Hospital & MRC, Belagavi are enrolled in this study. All blood reports of pregnant females during their antenatal care visit are performed in Dr. Prabhakar Kore Hospital laboratory.

The data collected comprise of relevant clinical history and the blood investigations of pregnant females before delivery.

Phlebotomy is performed under strict aseptic precautions and 3 mL of venous blood is collected in EDTA, plain and in fluoride test tubes. Blood is tested for CBC, CRP and procalcitonin.

Automated hematology analyzer - The Sysmex XN- 1500 automated hematology analyzer uses Fluorescence Flow Cytometry and hydrodynamically focused impedance method. The hydrodynamic focusing method improves the blood count accuracy and reproducibility. As the blood cells pass through the aperture in a line, it also prevents the generation of abnormal blood cell pulses.

Tests performed in this machine are RBC (red blood cell) count, Hb (hemoglobin), HCT (hematocrit), MCV (Mean Corpuscular Volume), MCH (Mean Corpuscular Hemoglobin), MCHC (Mean Corpuscular Hemoglobin Concentration), RDW (Red Cell Distribution Width), Retic count, WBC count, ANC (Absolute Neutrophil Count), ALC (Absolute Lymphocyte Count), AMC (Absolute Monocyte Count), AEC (Absolute Eosinophil Count), Platelet count and MPV (Mean Platelet Volume).

FIGURE 6. SYSMEX SN 1500

Hs- CRP and Procalcitonin were tested using COBAS C 503 ANALYSER:

Hs-CRP is measured on the basis of Turbidimetric immunoassay where CRP in the patient's serum binds to anti-CRP antibodies on latex particles, resulting in antigen-antibody complexes which cause increased turbidity. The analyser measures the turbidity, which is proportional to CRP concentration and results are calibrated against known standards and expressed in mg/L.

Procalcitonin is measured using an electrochemiluminescence immunoassay (ECLIA), where PCT in the serum forms a complex with biotinylated and ruthenium-labelled antibodies, which then binds to streptavidin-coated magnetic microparticles. This complex is magnetically captured, and a voltage induces chemiluminescence from the

ruthenium. The emitted light is measured which is proportional to PCT concentration and results are expressed in ng/ml.

FIGURE 7. COBAS C 503 ANALYZER

INFLAMMATORY MARKER RATIOS WERE CALCULATED AS FOLLOWS:

NLR = Absolute neutrophil count / Absolute lymphocyte count Normal range= 1.3- 2.5

PLR= Platelet count / Absolute lymphocyte count Normal range= 64.82-118.72

LMR= Absolute lymphocyte count / Absolute monocyte count Normal range= 3.46 to 26.67

Data processing and analysis/statistical analysis:

The data entry for the values was done in Microsoft excel spreadsheet and the results were obtained with Independent t test/ Mann- Whitney U test, ROC curve (Receiver operator characteristic) and Chi-square test. The statistical analyses were done using STATA software (version 17).

A p value of lesser than 0.05 was considered as statistically significant.

RESULTS

Table 3. Comparison of laboratory profile of study participants among preterm and term group (N=200)

Variables	Mean (SD)/ Median (IQR)		P value*
	Preterm	Term	
Age (years)	26.0 ±4.1	25.1 ±3.9	0.11
POG (weeks)	32.3 ±2.2	38.3 ±1.1	**<0.001**
hs- CRP (mg/L)	25.9 (12.8-54.1)	4.6 (2.5-7.6)	**<0.001**
PCT (ng/ml)	0.05 (0.03-0.2)	0.03 (0.02-0.05)	**<0.001**
HB (g/dL)	10.9 (10.1-11.8)	11.0 (9.9-11.9)	0.89
RBC COUNT x 10^6/µl	3.9 ±0.5	3.8 ±0.5	0.08
HCT (%)	33.9 (31.1-36.2)	34.5 (31.8-37.2)	0.46
MCV (fL)	87.5 ±9.5	85.9 ±9.9	0.23
MCH (pg)	28.2 ±3.3	27.1 ±4.0	**0.04**
MCHC (g/dl)	31.8 ±1.5	31.4 ±1.6	0.10
RDW %	14.8 (13.3-16.5)	15 (13.9-16.7)	0.18
RETIC %	1.3 (0.5-1.9)	0.9 (0.5-1.7)	0.23
WBC COUNT x 10^3/µl	14.6 ±9.9	10.5 ±3.9	**<0.001**
PLATELET x10^3/µl	233.8 ±67.5	243.7 ±83.6	0.35
ANC x 10^3/µl	10.7 ±3.9	7.5 ±3.7	**<0.001**
ALC x 10^3/µl	2.0 ±0.8	2.1 ±0.7	0.33
AMC x 10^3/µl	0.7 ±0.3	0.5 ±0.2	**<0.001**
AEC x 10^3/µl	0.1 (0.1-0.3)	0.2 (0.1-0.3)	**0.04**
MPV	9.5 (8.6-10.4)	9.2 (8.4-10.1)	**0.04**

*Independent t test/ Mann-Whitney U test

Table 3 describes comparison of laboratory profile of study participants among preterm and term group. The mean (SD, Standard Deviation) age among preterm group was 26.0 ±4.1 years and in term group was 25.1 ±3.9 years, with no statistical difference (p=0.11).

The mean (SD) period of gestation among preterm group was 32.3±2.2 weeks and in term group was 38.3 ±1.1 weeks, with statistically significant difference (p<0.001).

The median (IQR, interquartile range) hs-CRP level among preterm group was 25.9 mg/L (IQR= 12.8-54.1mg/L) and in term group was 4.6 mg/L (IQR= 2.5-7.6 mg/l), with statistically significant difference (p<0.001). Figure 8 shows a box plot demonstrating these findings in preterm and term group with hs-CRP values being significantly higher in preterm group.

The median (IQR) PCT level among preterm group was 0.05 ng/ml (IQR= 0.03-0.2 ng/ml) and in term group was 0.03 ng/ml (IQR= 0.02-0.05 ng/ml), with statistically significant difference (p<0.001). Figure 9 shows a box plot demonstrating these findings in preterm and term group with PCT values being significantly higher in preterm group.

The mean (SD) WBC count among preterm group was 14.6 ± 9.9 x 10^3/ µl and in term group was 10.5 ±3.9 x 10^3/ µl, with statistically significant difference (p<0.001). Figure 10 shows a box plot which demonstrate that though WBC counts are on higher side of normal range in both groups, preterm group has significantly higher WBC count than term group.

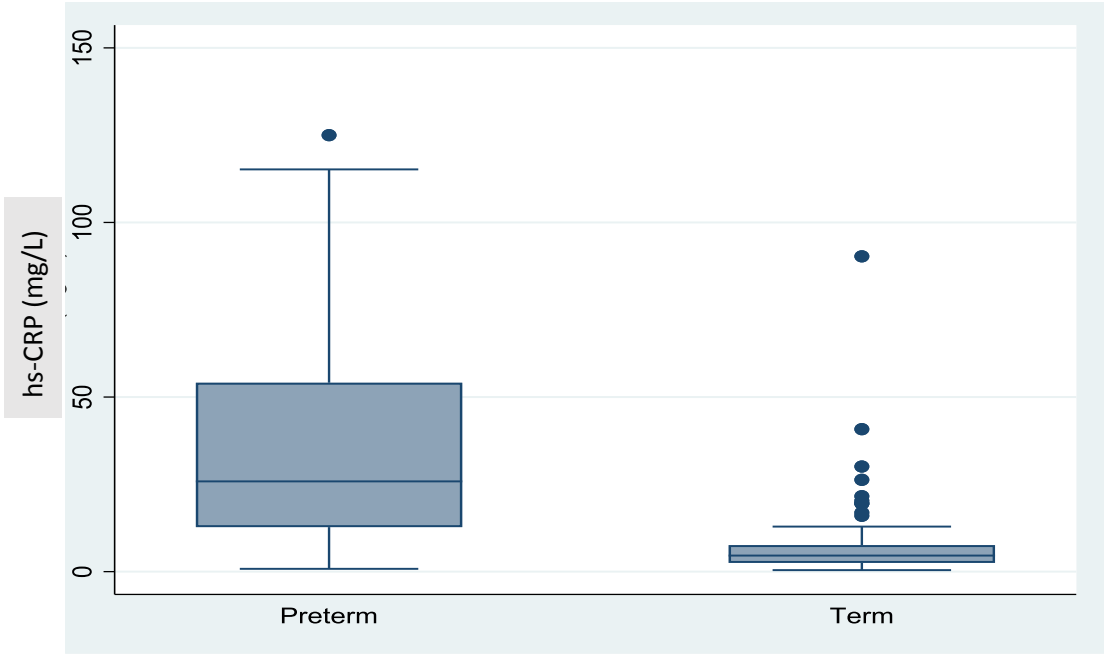

Figure 8. Box Plot showing comparison of hs-CRP of study participants among preterm and term group

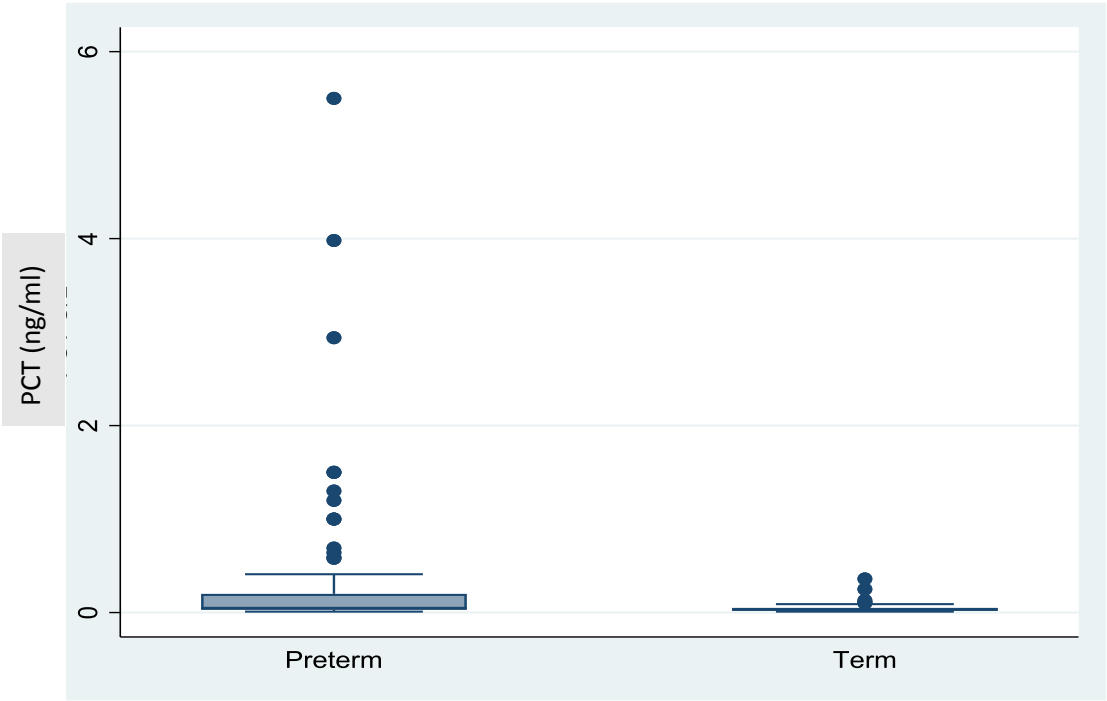

Figure 9. Box Plot showing comparison of PCT of study participants among preterm and term group

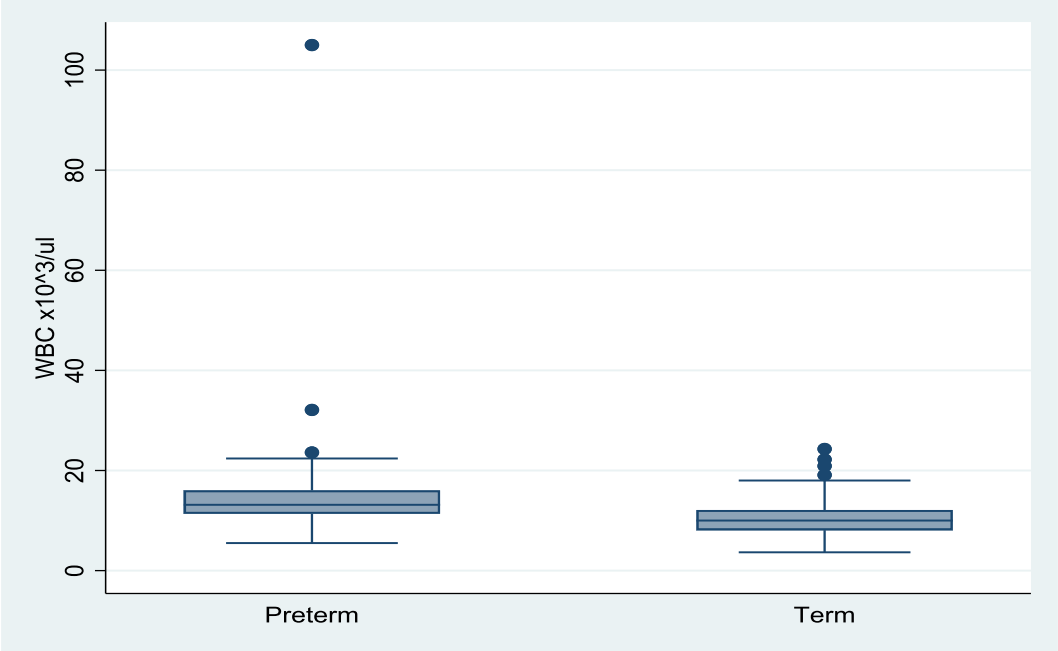

Figure 10. Box Plot showing comparison of WBC of study participants among preterm and term group

Table 4. Comparison of significant laboratory tests of study participants among preterm and term group (N=200)

Variables	Mean (SD)/ Median (IQR)		P value*
	Preterm	Term	
MCH (pg)	28.2 ±3.3	27.1 ±4.0	**0.04**
ANC x 10^3/ µl	10.7 ±3.9	7.5 ±3.7	**<0.001**
AMCx 10^3/ µl	0.7 ±0.3	0.5 ±0.2	**<0.001**
AECx 10^3/ µl	0.1 (0.1-0.3)	0.2 (0.1-0.3)	**0.04**
MPV (fL)	9.5 (8.6-10.4)	9.2 (8.4-10.1)	**0.04**

Table 4 describes comparison of significant laboratory tests of study participants among preterm and term group. The mean (SD) MCH among preterm group was 28.2 ±3.3 pg and in term group was 27.1 ±4.0 pg, with significant statistical difference (p=0.04).

The mean (SD) ANC among preterm group was 10.7 ±3.9 X 10^3/ mm^3 and in term group was 7.5 ±3.7 X 10^3/ mm^3, with significant statistical difference (p<0.001).

The mean (SD) AMC among preterm group was 0.7 ±0.3 X 10^3 / mm^3 and in term group was 0.5 ±0.2 X 10^3/ mm^3, with significant statistical difference (p<0.001).

The median (IQR) AEC level among preterm group was 0.1 X 10^3/ mm^3 (IQR=0.1-0.3 X 10^3/ mm^3) and in term group was 0.2 X 10^3/ mm^3 (IQR= 0.1-0.3 X 10^3/ mm^3), with significant statistical difference (p= 0.04).

The median (IQR) MPV level among preterm group was 9.5 fL (IQR= 8.6-10.4 fL) and in term group was 9.2 fL (IQR= 8.4-10.1fl), with significant statistical difference (p= 0.04).

Table 5. Comparison of inflammatory markers of study participants among preterm and term group (N=200)

Variables	**Mean (SD)/ Median (IQR)**		**P value***
	Preterm	**Term**	
NLR	5.1 (3.6-7.5)	3.2 (2.3-4.6)	**<0.001**
LMR	2.8 (2-4.4)	4.0 (2.7-6)	**<0.001**
PLR	135.9 ±79.7	127.6 ±73.6	0.43

*Independent t test/ Mann-Whitney U test

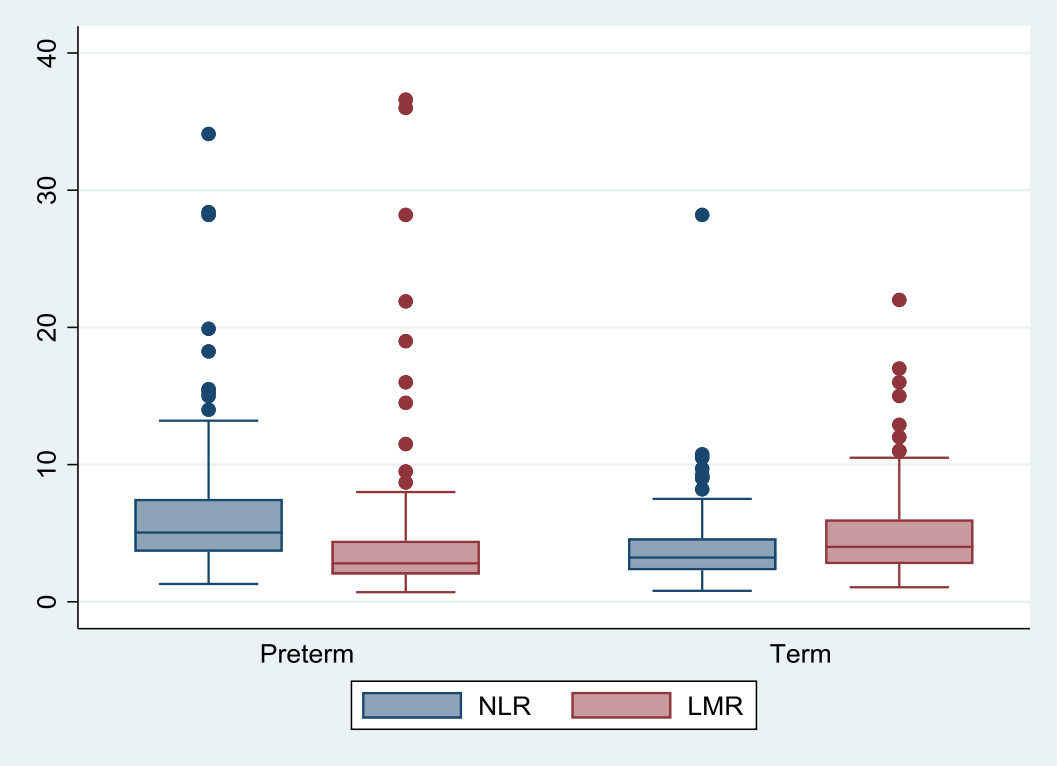

Figure 11. Box Plot showing comparison of NLR and LMR of study participants among preterm and term group

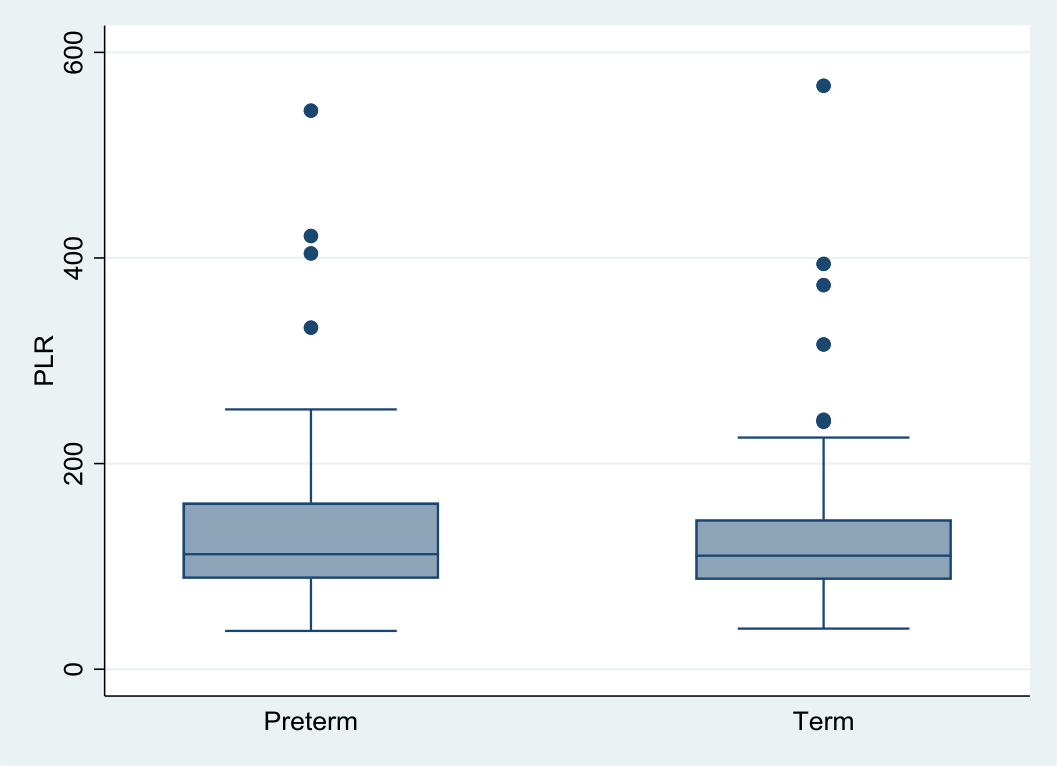

Figure 12. Box Plot showing comparison of PLR of study participants among preterm and term group

Comparison of inflammatory markers of study participants among preterm and term group is depicted in Table 5. The median (IQR) levels of NLR among preterm group was 5.1 (3.6-7.5) and among term group was 3.2 (2.3-4.6) and this difference was statistically significant (p<0.001) (figure 11). The median (IQR) levels of LMR among preterm group was 2.8 (2-4.4) and among term group was 4.0 (2.7-6) and this difference was statistically significant (p<0.001) (figure 11). The mean (SD) level of PLR among preterm group was 135.9 ±79.7 and among term group was 127.6 ±73.6 and this difference was statistically non-significant (p=0.43) (figure12).

Table 6. Comparison of CRP levels among preterm and term group (N=200)

CRP	n (%)		P value*
	Preterm	Term	
≤ 5	11 (11)	60 (60)	<0.001
>5	89 (89)	40 (40)	
Total	**100 (100)**	**100 (100)**	

Comparison of CRP levels of study participants among preterm and term group is depicted in Table 6 and figure 13. Among preterm group 89% had CRP levels >5 mg/L, while 40% in term had CRP levels >5 mg/L. The difference was statistically significant (p<0.001).

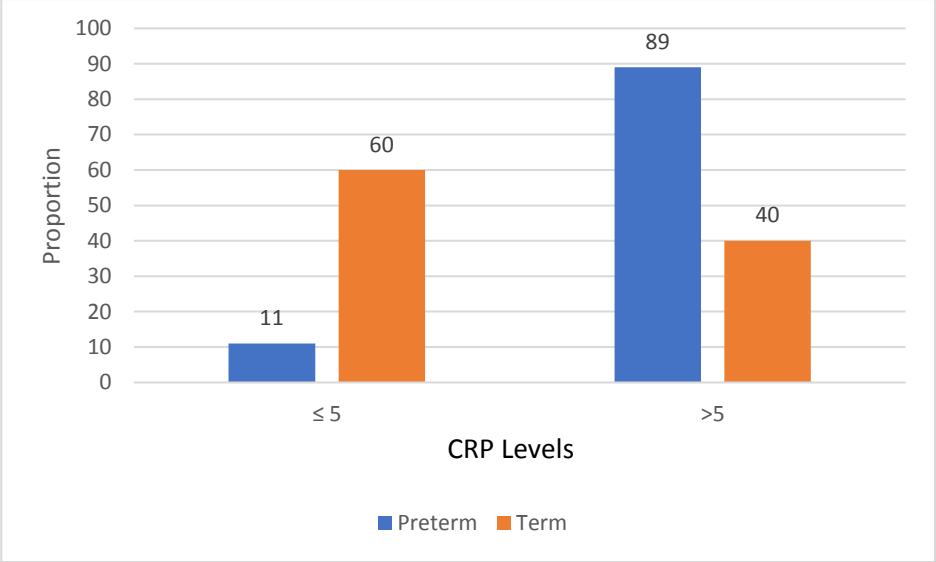

Figure 13. Comparision of hs-CRP in preterm and term labor with cut-off of 5mg/L

Table 7. Comparison of WBC count among preterm and term group (N=200)

WBC COUNT x 10^3/uL	n (%)		P value*
	Preterm	Term	
≤13	48 (48)	80 (80)	<0.001
>13	52 (52)	20 (20)	
Total	**100 (100)**	**100 (100)**	

Comparison of WBC counts of study participants among preterm and term group is depicted in Table 7 and figure 14. Among preterm group 52% had WBC count between more than 13 x 10^3/uL, while 20% in term had WBC count above 13 x 10^3/uL. Among preterm group 48% had WBC count less than 13 x 10^3/uL, while 80% in term had WBC count below 13 x 10^3/uL. The difference was statistically significant (p<0.001).

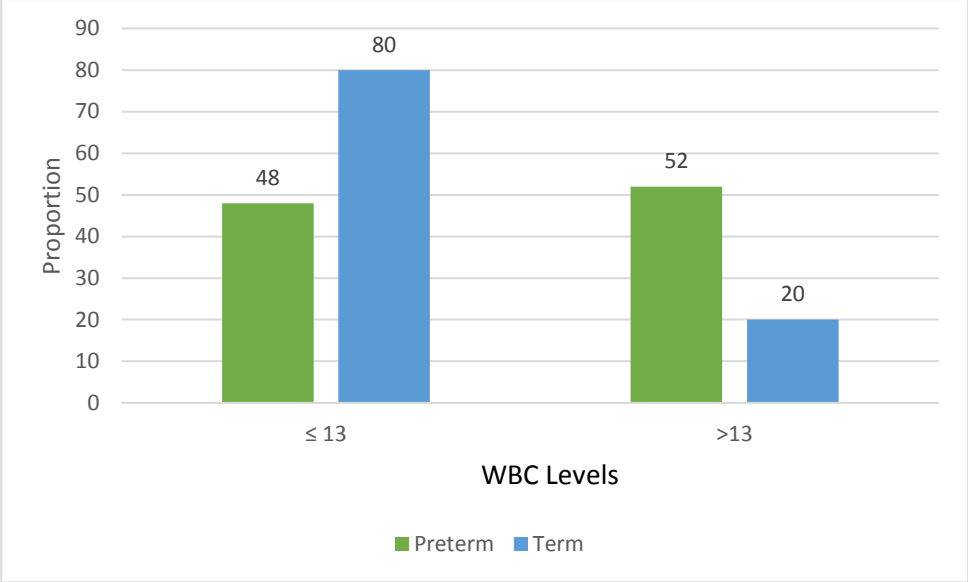

Figure 14. Comparision of WBC count in preterm and term labor with cut-off of 13 x 10^3/ul

Table 8. Comparison of ANC count among preterm and term group (N=200)

ANC COUNT x 10^3/uL	n (%)		P value*
	Preterm	Term	
≤10	49 (49)	80 (80)	**<0.001**
>10	51 (51)	20 (20)	
Total	**100 (100)**	**100 (100)**	

Comparison of ANC counts of study participants among preterm and term group is depicted in Table 8 and figure 15. Among preterm group 51% had ANC count more than 10 x10^3/uL, while 20% in term had ANC count more than 10 x 10^3/uL. Among preterm group 49% had ANC count less than 10 x 10^3/uL, while 80% in term had ANC count less than 10 x 10^3/uL. The difference was statistically significant (p<0.001).

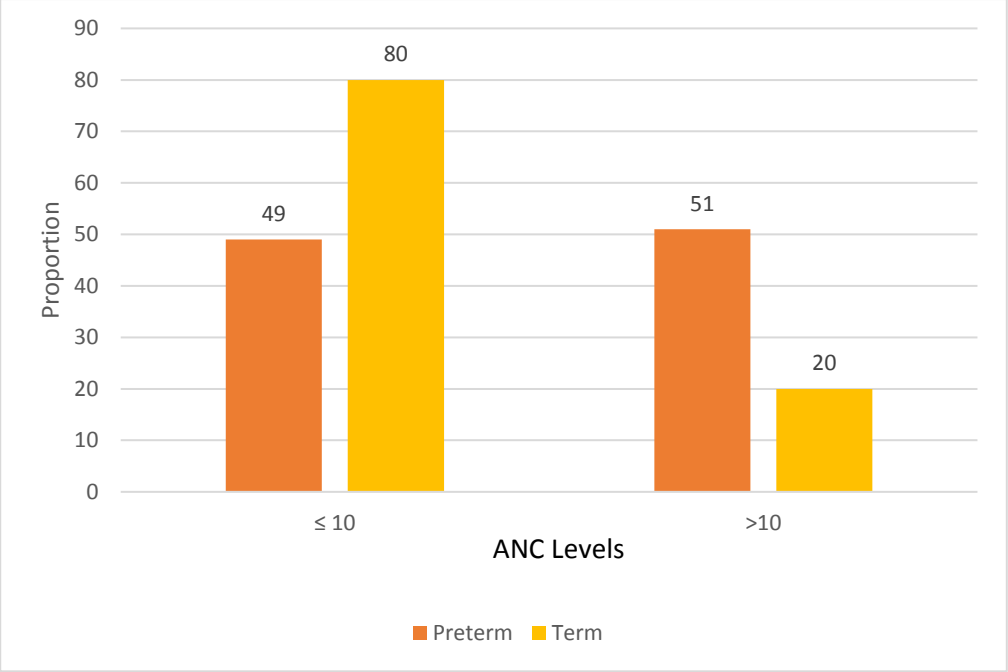

Figure 15. Comparison of ANC in preterm and term labor with cut-off of 10 x 10^3/ul

Table 9. Comparison of NLR, LMR and PLR for predicting preterm labor with optimal cut-off points (N=200)

	AUC	95% CI	Cut-off point	Sensitivity (%)	Specificity (%)
Preterm birth					
NLR	0.73	0.66-0.80	>3.55	80.0	56.0
LMR	0.64	0.56-0.72	<2.66	78.0	48.0
PLR	0.52	0.44-0.60	>111.2	51.0	51.0

ROC (Receiver operating characteristic) curve results are illustrated in figures 16-18. Comparison of NLR, LMR and PLR for predicting preterm labor with optimal cut-off points is depicted in Table 9. The c-index based on area under the ROC curve (AUC) values for the NLR levels in predicting preterm labor was calculated as 0.73 (95%CI: 0.66-0.80). The optimal cut-off point predicting preterm labor in the study group according to NLR levels was 3.55 with 0.80 sensitivity and 0.56 specificity.

The c-index based on area under the ROC curve (AUC) values for the LMR levels in predicting preterm labor was calculated as 0.64 (95%CI: 0.56-0.72). The optimal cut-off points for predicting preterm labor in the study group according to LMR levels was 2.66 with 0.78 sensitivity and 0.48 specificity.

The c-index based on area under the ROC curve (AUC) values for the PLR levels in predicting preterm labor was calculated as 0.52 (95%CI: 0.44-0.60). The optimal cut-off point predicting preterm labor in the study group according to PLR levels was 111.2 with 0.51 sensitivity and 0.51 specificity.

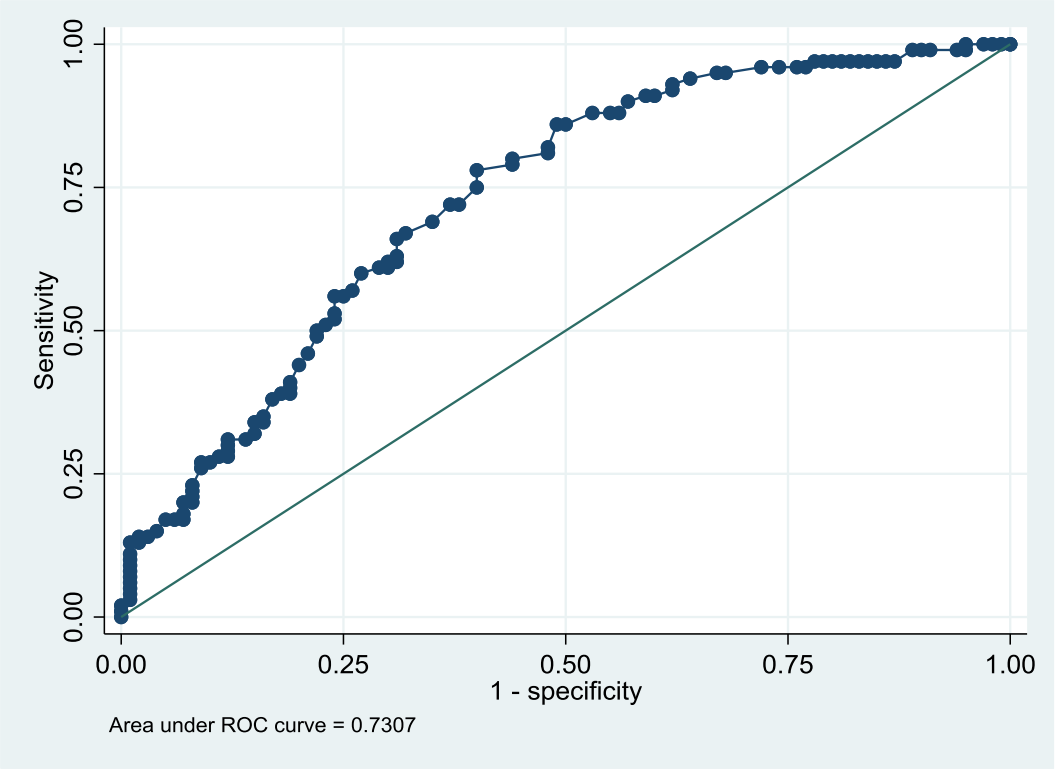

Figure 16. Receiver operating characteristic (ROC) curve representing the cut-off point of NLR in prediction of preterm labor

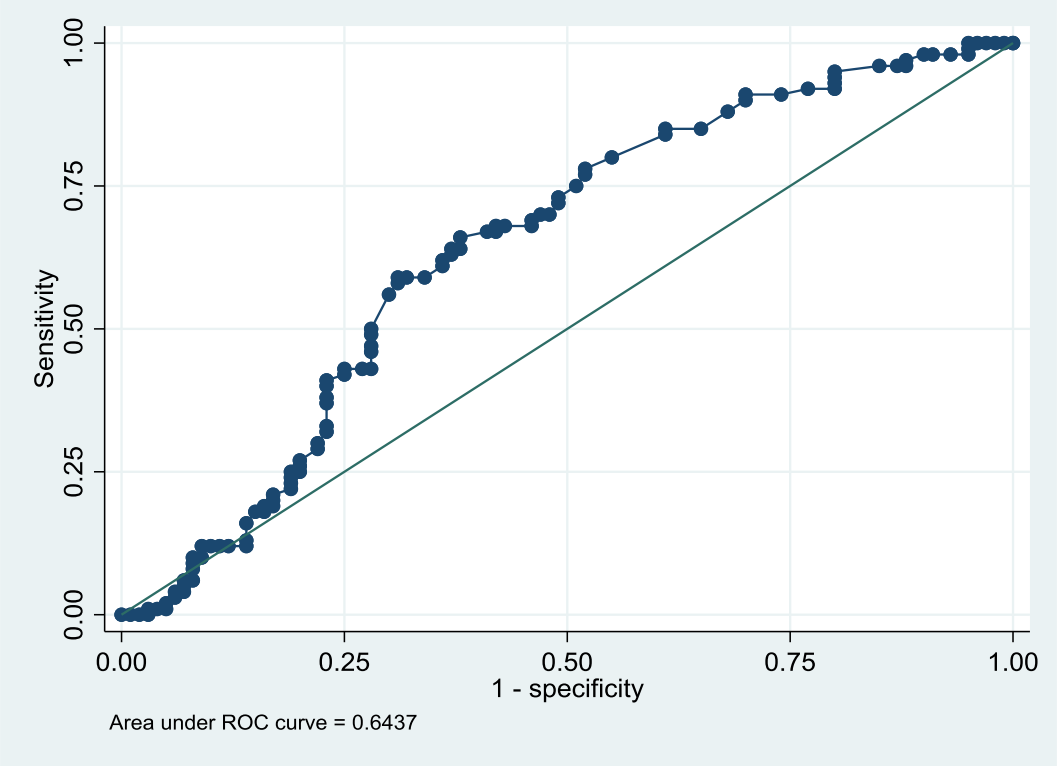

Figure 17. Receiver operating characteristic (ROC) curve representing the cut-off point of LMR in prediction of preterm labor

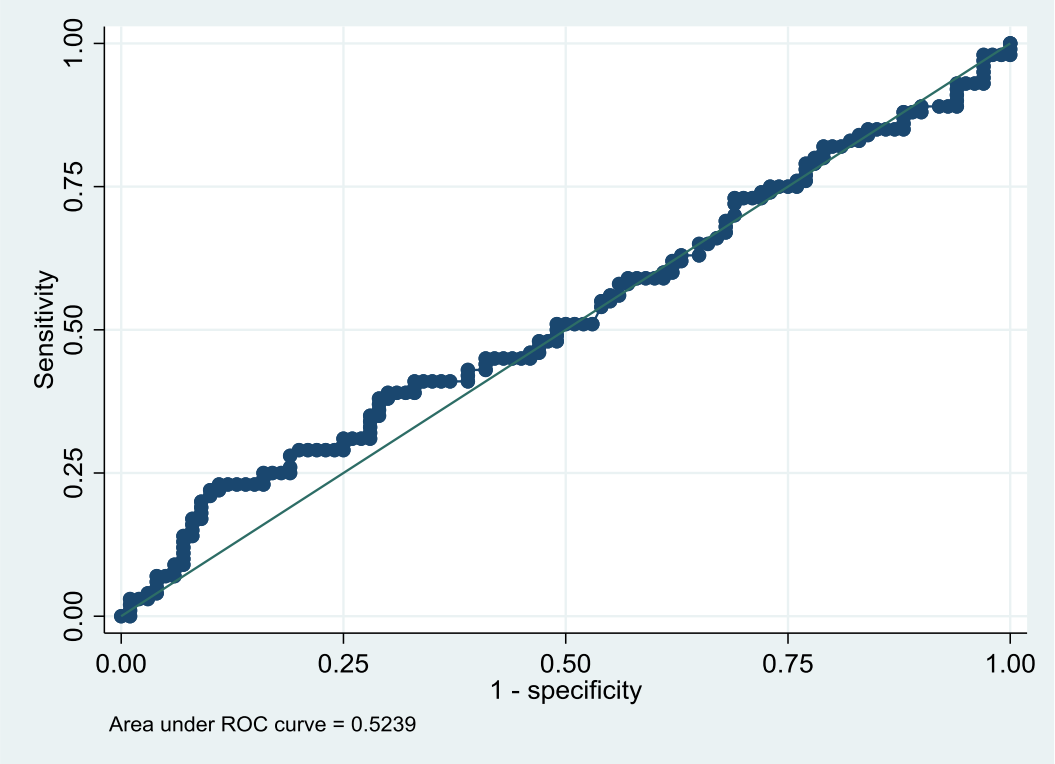

Figure 18. Receiver operating characteristic (ROC) curve representing the cut-off point of PLR in prediction of preterm labor

Table 10. Comparison of parity of study participants among preterm and term group (N=200)

Parity	n (%)		P value*
	Preterm	Term	
1	82 (82.0)	82 (82.0)	0.24
2	15 (15.0)	17 (17.0)	
3	3 (3.0)	0 (0.0)	
4	0 (0.0)	1 (0.0)	
Total	**100 (100)**	**100 (100)**	

*Chi-square test

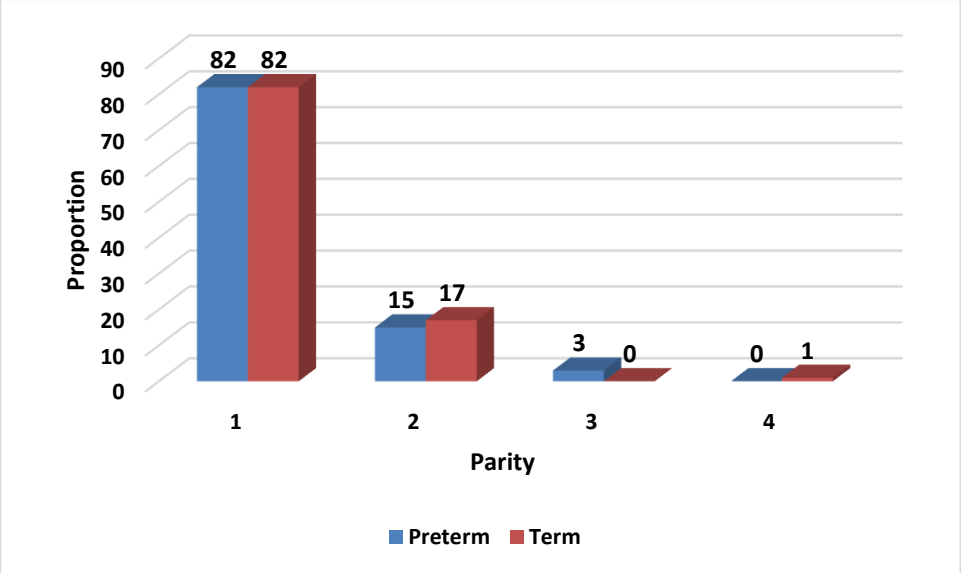

Figure 19. Comparison of parity of study participants among preterm and term group

Comparison of parity of study participants among preterm and term group is depicted in Table 10 and figure 19. Among preterm and term group 82% had parity as one, while 17% in term had parity as two and 15% in preterm group had parity two. The difference was statistically non-significant (p=0.24)

Table 11. Comparison of clinical history among preterm and term group (N=200)

Variables	Preterm n (%)	Term n (%)	P value*
History of Preterm birth	18 (18)	3 (3)	**<0.001**
History of Still birth	3 (3)	3 (3)	1.00
History of bacterial vaginosis	8 (8)	4 (4)	0.23
History of BPV in FT	4 (4)	3 (3)	0.70
History of UTI	6 (6)	4 (4)	0.51
Hypertension	2 (2)	1 (1)	0.56
Hypothyroidism	2 (2)	0 (0)	0.16
Cervical Insufficiency	4 (4)	0 (0)	**0.04**
PROM	40 (40)	3 (3)	**<0.001**
Preeclampsia	18 (18)	6 (6)	**0.009**
Oligohydramnios	11 (11)	8 (8)	0.47

*Chi-square test

Table 11 describes comparison of clinical histories among preterm and term group. Among preterm group 18% (n=18) had history of previous preterm birth while in term group 3% (n=3) had history of preterm birth. This difference was statistically significant ($p<0.001$).

Among both preterm group and term group, 3% (n=3) each had history of still birth. Hence, there was no statistical difference (p=1.00).

Among preterm group 8% (n=8) had history of bacterial vaginosis while in term group 4% (n=4) had history of bacterial vaginosis. This difference was statistically non-significant (p=0.23).

Among preterm group 4% (n=4) had history of bleeding per vagina in first trimester while in term group 3% (n=3) had history of bleeding per vagina in first trimester. This difference was statistically non-significant (p=0.70).

Among preterm group 6% (n=6) had history of urinary tract infection while in term group 4% (n=4) had history of urinary tract infection. This difference was statistically non-significant (p=0.51)

Among preterm group 2% (n=2) had hypertension while in term group 1% (n=1) had hypertension. This difference was statistically non-significant (p=56).

Among preterm group 2% (n=2) had hypothyroidism while in term group none had hypothyroidism. This difference was statistically non-significant (p=16).

Among preterm group 40% (n=40) had premature rupture of membrane while in term group 3% (n=3) had premature rupture of membrane. This difference was statistically significant (p<0.001).

Among preterm group 4% (n=4) had cervical insufficiency while in term group none had cervical insufficiency. This difference was statistically significant (p=0.04).

Among preterm group 4% (n=4) had cervical insufficiency while in term group none had cervical insufficiency. This difference was statistically significant (p=0.04).

Among preterm group 11% (n=11) had oligohydramnios while in term group 8% (n=8) had oligohydramnios. This difference was statistically non-significant (p=47).

Table 12. Comparison of history of previous preterm birth among preterm and term group (N=200)

History of Preterm birth	Preterm n (%)	Term n (%)	P value*
Yes	18 (18)	3 (3)	**<0.001**
No	82 (82)	97 (97)	
Total	**100 (100)**	**100 (100)**	

*Chi-squared test

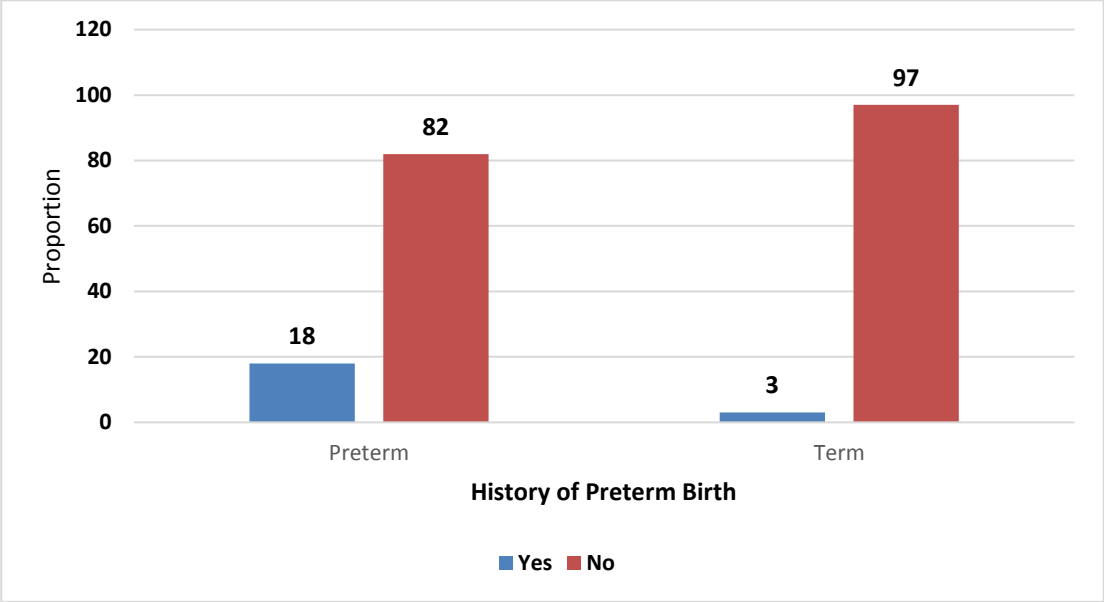

Figure 20. Comparison of previous history of preterm birth among preterm and term group

Table 12 and figure 20 describes comparison of history of preterm birth among preterm and term group. Among preterm group 18% (n=18) had history of preterm birth while in term group 3% (n=3) had history of preterm birth. This difference was statistically significant (p<0.001).

Table 13. Comparison of Cervical Insufficiency among preterm and term group (N=200)

Cervical Insufficiency	Preterm n (%)	Term n (%)	P value*
Yes	4 (4)	0 (0)	**0.04**
No	96 (96)	100 (100)	
Total	**100 (100)**	**100 (100)**	

*Chi-square test

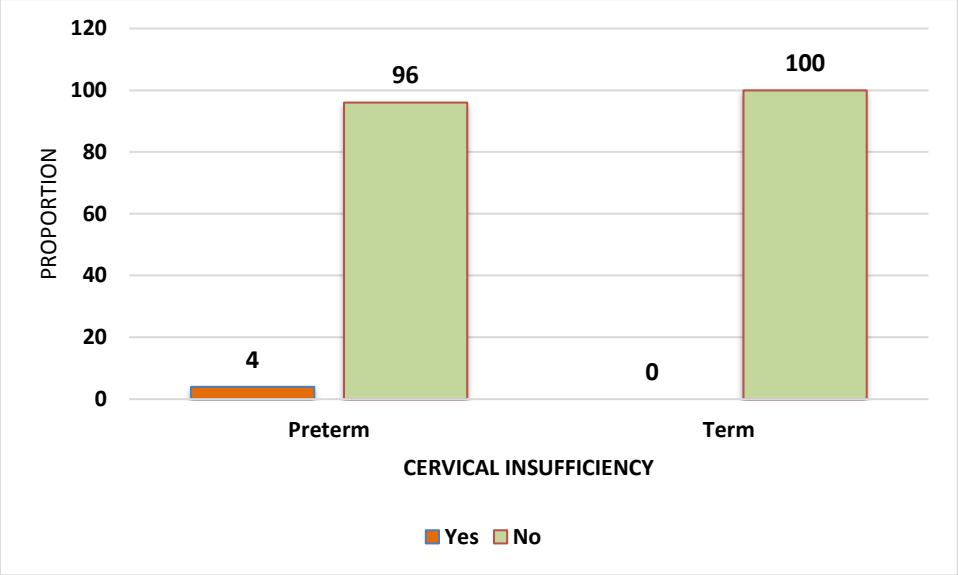

Figure 21. Comparison of Cervical Insufficiency among preterm and term group

Table 13 and figure 21 depicts comparison of cervical insufficiency among preterm and term group. Among preterm group 4% (n=4) had cervical insufficiency while in term group none had cervical insufficiency. This difference was statistically significant (p=0.04).

Table 14. Comparison of PROM among preterm and term group (N=200)

PROM	Preterm n (%)	Term n (%)	P value*
Yes	40 (40)	3 (3)	**<0.001**
No	60 (60)	97 (97)	
Total	**100 (100)**	**100 (100)**	

*Chi-square test

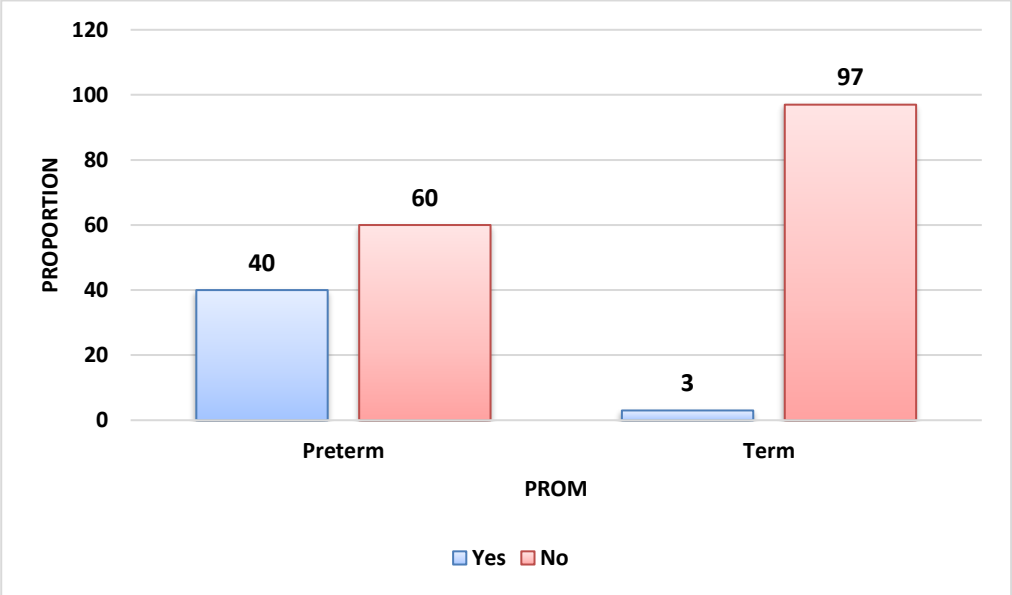

Figure 22. Comparison of PROM (premature rupture of membrane) among preterm and term group

Table 14 and figure 22 shows comparison of premature rupture of membrane among preterm and term group. Among preterm group 40% (n=40) had premature rupture of membrane while in term group 3% (n=3) had premature rupture of membrane. This difference was statistically significant ($p<0.001$).

Table 15. Comparison of preeclampsia among preterm and term group (N=200)

Preeclampsia	Preterm n (%)	Term n (%)	P value*
Yes	18 (18)	6 (6)	**0.009**
No	82 (82)	94 (94)	
Total	**100 (100)**	**100 (100)**	

*Chi-squared test

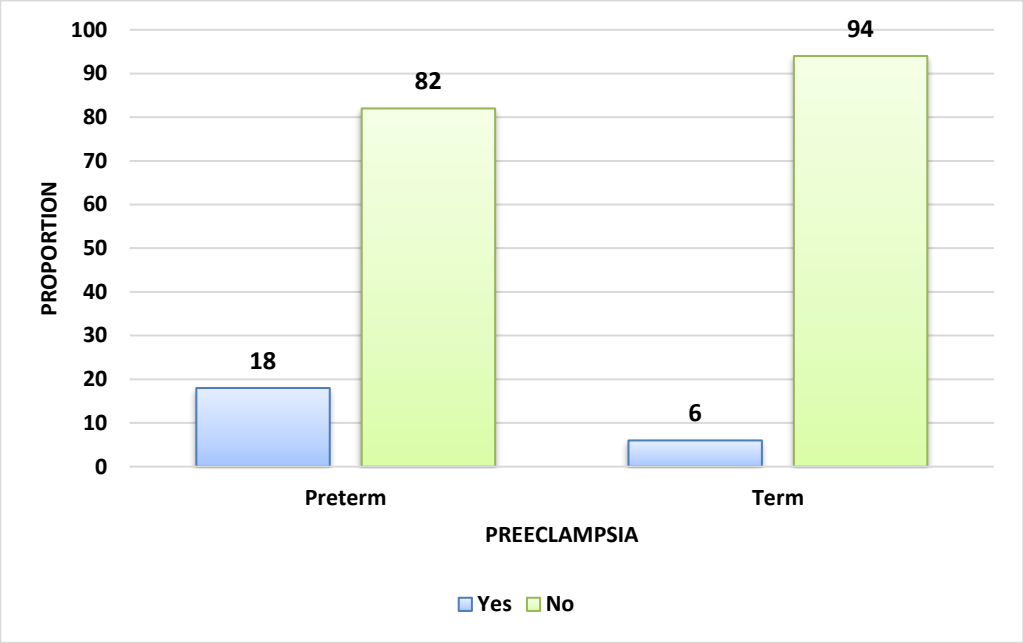

Figure 23. Comparison of preeclampsia among preterm and term group

Table 15 and figure 23 shows comparison of preeclampsia among preterm and term group. Among preterm group 18% (n=18) had preeclampsia while in term group 6% (n=6) had preeclampsia. This difference was statistically significant (p=0.009).

DISCUSSION

The early diagnosis of preterm labor remains a significant challenge in obstetric care. Prompt diagnosis of preterm labor allows for interventions that can potentially delay delivery and improve neonatal outcomes. Prediction strategies like cervical length screening and fetal fibronectin levels have been widely used to diagnose PTB. But these are particularly limited for asymptomatic populations and Some studies have demonstrated that these two tests have low predictive accuracy. Currently, there's no single test that can definitively diagnose preterm labor with high accuracy.

Infection and inflammation are well-established significant risk factors for preterm birth. Complete blood count is one of the routine tests done during antenatal care. Recently, CBC-derived parameters, such as the NLR, LMR and PLR, have been recognized as inflammatory markers for preterm labor because of their ability to predict systemic inflammation.

This study explores the utility of hematological parameters and inflammatory markers in predicting preterm labor. The primary objective of this study is to assess and compare hematological and inflammatory markers between preterm labor and term labor.

Table 16. Age difference between preterm and term labor

Study	Mean (SD)/ Median (IQR)	
	Preterm	**Term**
Khatoon et al.[95]	26.38 ± 5.32	26.68 ±4.12
Mittal et al.[96]	25.43	25.4
Sureshbabu et al.[97]	29.3 ± 5.1	28.1 ± 4.4
Rao et al.[98]	27.5 ± 3.97	27.1 ± 3.62
Daglar et al.[99]	26.7± 8.4	26.4 ± 8.2
Farzaneh et al.[8]	27.61±5.14	25.53±3.86
Mei ma et al.[1]	29.93 ± 4.039	30.07 ± 4.201
Hrubaru et al.[100]	29.6 ± 4.9	29.9 ± 5.0
Isik et al.[101]	28.7±5.1	28.1±3.5
Melissa et al.[102]	28.1 ± 5.6	27.3 ± 6.1
Djusad et al[103]	31(15-41)	30(17-43)
Our study	26.0 ±4.1	25.1 ±3.9

The mean age of our study is comparable to other similar studies in the literature. Sureshbabu et al., Farzaneh et al., Melissa et al., Djusad et al. and our study found slightly older ages in preterm groups compared to term. Literature has reported that older maternal age is associated with an increased risk of pregnancy complications, including preterm birth. Advanced maternal age can bring higher rates of underlying health issues, such as hypertension and diabetes, which are known risk factors for preterm labor [104,105]. These factors together could contribute to the observed age difference between the preterm and term labor groups in our study.

Table 17. Parity of study participants among preterm and term group across various studies

Study	Parity	%		P value*
		Preterm	Term	
Sureshbabu et al.[97]	1	40.3	44.5	0.415
	>1	59.7	55.5	
Reddy et al.[106]	1	39.2	42.7	0.51
	>1	60.7	57.2	
Mavalankar et al.[107]	1	37.1	36.7	<0.05
	2	24.1	26.0	
	3	18.9	21.4	
Koullali et al.[108]	1	4.1	-	-
	2	4.2	-	
	3	4.9	-	
	4	5.9	-	
Konyala et al.[109]	1	45.45	-	-
	2	40.91	-	
	3	11.36	-	
	4	0.76	-	
Yadav et al.[110]	1	53.8%	-	-
	2	28.6	-	
	3	13.2	-	
	4	4.4	-	
Our study	1	82.0	82	0.24
	2	15.0	17	
	3	3	0	
	4	0	0	

In most of the studies, mothers with parity 1 have a higher prevalence of preterm birth. First-time mothers may be more prone to pregnancy complications due to physiological factors. Additionally, first pregnancies can be more stressful and associated with higher levels of anxiety and uncertainty, which may contribute to adverse outcomes. However,

the comparison to term birth was not statistically significant. This lack of statistical significance could be due to the relatively small differences in parity distribution between preterm and term groups within each study.

Table 18. Clinical history of previous preterm birth across various studies

Study	**Preterm %**	**Term %**	**P value***
Sureshbabu et al.[97]	8.9	2	0.003
Mahajan A et al.[111]	26	9	<0.01
Reddy et al.[106]	7.41	1.61	0.008
Mavalankar et al.[107]	10.6	1.6	<0.05
Renzo et al.[112]	7.2	1.8	0.009
Sehgal et al.[113]	57	-	-
Roy et al.[114]	18.4	-	-
Bangal et al.[115]	11.3	-	-
Our study	18	3	**<0.001**

The comparison of history of preterm birth between preterm and term groups across various studies shows a consistent trend: history of preterm birth in previous pregnancy is significantly more common among those who experienced preterm labor. Our study showed 18% in the preterm group compared to 3% in the term group ($p<0.001$). These findings indicate that a history of preterm birth is a strong predictor of subsequent preterm labor. A history of preterm birth is a strong predictor of subsequent preterm labor due to several factors, including persistent underlying health conditions, genetic predispositions, structural abnormalities in the cervix or uterus, and recurring infections or inflammation. Behavioural and socioeconomic factors, such as stress and inadequate prenatal care, also contribute. Additionally, the body's consistent physiological response to pregnancy increases the likelihood of repeated preterm labor. These combined factors make a history of preterm birth a significant indicator of future preterm labor risks.[116,117]

Table 19. Premature rupture of membranes (PROM) across various studies

Study	Preterm %	Term %	P value*
Mahajan A et al.[111]	19	2	< 0.01
Rao et al.[98]	17.5	6.3	<0.001
Sureshbabu et al.[97]	25.2	5.5	<0.001
Soundarajan t al.[22]	28.7	16.6	<0.001
Reddy et al.[106]	21	5.2	<0.001
Mavalankar et al.[107]	12	10.2	<0.05
Mei ma et al.[1]	64.8	29.2	<0.001
Mohapatra et al.[118]	29.96	-	
Konyala et al.[109]	26.52	-	
Yadav et al.[110]	30.8	-	
Sudhir et al.[119]	7.47	-	
Our study	40	3	<0.001

The comparison of the prevalence of premature rupture of membranes (PROM) between preterm and term groups across various studies shows that PROM is significantly more common in preterm births. Mahajan A et al., Rao et al., Sureshbabu et al., Soundarajan et al., Reddy et al., Mavalankar et al., and Mei Ma et al. all reported significantly higher PROM rates in preterm groups compared to term groups, with p-values indicating strong statistical significance (<0.05 to <0.001). Our study also showed a marked difference, with 40% in the preterm group versus 3% in the term group (p<0.001).

The increased prevalence of PROM in preterm births can be attributed to several factors. Weakness in the fetal membranes, infections, and inflammatory responses can lead to premature rupture. Additionally, conditions like cervical insufficiency and certain maternal health issues contribute to the higher incidence of PROM in preterm pregnancies.[120]

CLINICAL HISTORY OF CERVICAL INSUFFICIENCY

Sayres et al., states that a shortened cervix (i.e., generally less than 3.0 cm) or a funneling configuration at the internal os observed in second trimester transvaginal ultrasonography increases the likelihood of preterm delivery.[17,24,121]

4% of females in preterm group were found to have cervical insufficiency in our study.

Hence, transvaginal ultrasonography is recommended in late second and early third trimesters.

CLINICAL HISTORY OF PREECLAMPSIA

A study done by Mohapatra et. Al., found that 13% of females who delivered prematurely had history of preeclampsia. Out of which, 60 % females had to undergo preterm labor iatrogenically. They found that Preeclampsia and IUGR were more significantly associated with the iatrogenic preterm delivery. (122) In our study we found that 18% females in preterm group had history of preeclampsia. Therefore, Preeclampsia is an important contributor to preterm delivery and potentially useful condition to target in order to reduce preterm rates.

HEMATOLOGICAL PARAMETERS

Table 20. WBC Count values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Khatoon et al.[95]	$10.16 \pm 4.04 \times 10^3$	$8.84 \pm 2.16 \times 10^3$	Not significant (0.140)
Mittal et al.[96]	$11.6 \pm 1.95 \times 10^3$	$8.04 \pm 1.11 \times 10^3$	Significant (<0.001)
Rani et al[122]	$10.13 \pm 2.08 \times 10^3$	$8.54 \pm 1.71 \times 10^3$	Significant (<0.001)
Farzaneh et al.[8]	$9.30 \pm 1.46 \times 10^3$	$8.05 \pm 1.56 \times 10^3$	Significant
Mei ma et al.[1]	$10.18 \pm 2.54 \times 10^3$	$9.90 \pm 1.95 \times 10^3$	Not significant
Hrubaru et al.[100]	$9.22 \pm 5.70 \times 10^3$	$8.94 \pm 5.21 \times 10^3$	Significant
Isik et al.[101]	$11.7 \pm 3.7 \times 10^3$	$10.6 \pm 2.6 \times 10^3$	Significant (0.026)
Djusad et al[103]	13.38 (6.14 – 40.00) $\times 10^3$	11.70 (4.36 – 29.79) $\times 10^3$	Not significant (0.057)
Our study	$14.6 \pm 9.9 \times 10^3$	$10.5 \pm 3.9 \times 10^3$	Significant

Our study shows a significant difference in mean WBC counts between preterm and full-term labor groups. It was observed that both the WBC count and neutrophil levels are elevated in both groups but these values are significantly higher in cases of preterm labor compared to term pregnancies. This finding aligns with the majority of studies mentioned above. The higher WBC counts in preterm labor can be attributed to underlying infections or inflammatory responses, which are common triggers for preterm labor. Elevated WBC counts, ANC & AMC indicate the body's response to infection or inflammation, suggesting that these conditions are more prevalent in preterm labor cases.[26]The significant associations found in many studies support the idea that infection and inflammation are key factors in the pathogenesis of preterm labor.

Table 21. Hemoglobin values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Khatoon et al.[95]	10.45 ± 1.53	10.60 ± 1.15	Non-significant (0.41)
Malathi et al.[123]	10.33± 1.98	10.88 ± 1.52	Non-significant
Isik et al.[101]	11.3±1.7	11.8±1.4	Significant (0.008)
Farzaneh et al.[8]	11.88±0.97	11.56±1.24	Significant (0.037)
Hrubaru et al.[100]	11.72 ± 1.54	12.99 ± 1.60	Significant (<0.001)
Daglar et al.[99]	12.4 ± 0.6	12.1 ± 0.2	Non-significant
Our study	10.9 (10.1-11.8)	11.0 (9.9-11.9)	Non-significant (0.89)

In our study and in most of the studies mentioned, hemoglobin levels tend to be lower in the preterm group compared to the term group, although the differences are not always statistically significant. Our study also observed lower HB levels in the preterm group, but the difference was not statistically significant. Low hemoglobin levels, or anemia, in mothers can significantly increase the risk of preterm birth through various mechanisms.

Anemia reduces the oxygen-carrying capacity of blood, compromising fetal oxygenation and growth, potentially triggering preterm labor. Additionally, anemic mothers may experience physiological stress and immune system impairment, leading to increased susceptibility to infections and inflammatory responses that can initiate preterm labor. Furthermore, anemia can result in poor placental development and function, exacerbating uteroplacental insufficiency and hormonal imbalances that induce preterm contractions.[124,125]

Table 22. Mean Platelet Volume (MPV) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Alva et al.[126]	8.40±0.59	8.13±0.36	Significant (0.008)
Mittal et al.[96]	10.4 ± 0.92	8.8 ± 0.62	Significant (<0.001)
Rani et al[122]	10.01±1.55	9.66±1.43	Significant (0.003)
Mei ma et al [1]	10.33±1.00	10.10 ± 0.90	Significant (.037)
Isik et al.[101]	8.7± 1.2	8.4± 1.1	Not Significant (0.064)
Kannar et al.[127]	8.29±0.80	-	-
Wasiluk et al.[128]	8.02±0.92	-	-
Our study	9.5 (8.6-10.4)	9.2 (8.4-10.1)	Significant (0.04)

In our study, we observed a significant difference in MPV between preterm and term groups, with the preterm group having a higher MPV (9.5, IQR: 8.6-10.4) compared to term (9.2, IQR: 8.4-10.1; p=0.04). This is in line with other studies mentioned above. The variations in MPV between preterm and term groups could be attributed to differences in platelet activity and function in response to inflammatory or pathological conditions associated with preterm labor. These findings suggest that MPV may serve as a potential biomarker for identifying individuals at risk of preterm labor.

INFLAMMATORY MARKERS

Table 23. C- reactive protein (hs- CRP) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Rani et al[122]	2.66±0.85	1.06±0.37	Significant (0.002)
Khatoon et al.[95]	8.56 ± 4.86	7.40 ± 1.88	Not Significant (0.159)
Kumawat et al.[129]	31.62 ± 49.30	6.60 ± 0.97	Significant (0.001)
Shahshahan et al.[62]	5.28 ± 8.2	2.14 ±5.3	Significant (0.007)
Daglar et al.[99]	1.6 ± 1.1	0.59 ± 1.7	Significant (0.01)
Farzaneh et al.[8]	10.75±5.09	8.11±5.25	Significant (0.00)
Our study	25.9 (12.8-54.1)	4.6 (2.5-7.6)	Significant (<0.001)

Our study shows that there is significant difference in CRP levels between pre-term and term labor groups. Hs-CRP levels consistently appear elevated in mothers who experienced preterm labor compared to those who had full-term labor across all the studies cited. This pattern suggests an association between preterm labor and systemic inflammation. The elevated hs-CRP levels likely reflect the inflammatory processes inherent in preterm labor, including infections, placental dysfunction, and fetal distress. Additionally, underlying health conditions commonly associated with preterm labor, such as preeclampsia or PROM, further contribute to elevated hs-CRP levels. The timing of CRP measurement may also play a role, capturing peak levels of inflammation closer to the onset of preterm labor.[130]

Table 24. Procalcitonin (PCT) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Isik et al.[101]	0.239± 0.53	0.172± 0.31	Significant (0.001)
Rani et al[122]	0.22±0.06	0.20±0.04	Significant (0.001)
Ducarme et al.[131]	0.043 (0.02–0.07)	0.042 (0.02–0.13)	Not Significant (0.56)
Mei ma et al [1]	0.22 ± 0.05	0.22 ± 0.04	Not Significant (0.971)
Our study	0.05 (0.03-0.2)	0.03 (0.02-0.05)	Significant (<0.001)

Procalcitonin is a biomarker commonly used to diagnose bacterial infections and sepsis. For healthy term pregnancies, a PCT level around 0.04 µg/L is considered normal, whereas levels between 0.5–2.0 µg/L indicate possible systemic infection, and values between 2.0 and 10 µg/L are highly indicative of sepsis.[71] In our study the values are not indicative of infection, however most of the studies mentioned above have PCT level above the normal. The variability in findings highlights the need for further research to standardize PCT measurement and interpretation during pregnancy.

Table 25. Neutrophil to lymphocyte ratio (NLR) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Khatoon et al.[95]	4.30 ± 1.68	4.29 ± 6.04	Not significant (0.984)
Mittal et al.[96]	7.2 ± 3.07	2.7 ± 0.64	Significant (<0.001)
Rani et al[122]	4.75±2.1	3.57±1.20	Significant (<0.001)
Farzaneh et al.[8]	4.18±1.39	3.23±0.77	Significant (<0.001)
Mei ma et al [1]	4.42 ± 1.41	3.86 ± 1.09	Significant (<0.001)
Hrubaru et al.[100]	13.75 ± 9.13	9.06 ± 7.17	Significant (<0.001)
Isik et al.[101]	5.1 ± 4.1	4.0 ± 1.9	Not significant (0.273)
Melissa et al.[102]	5.9 ± 5.1	4.7 ± 3.2	Not significant (0.007)
Djusad et al[103]	5.46 (1.03-35.17)	5.50(1.55-36.21)	Not significant (0.795)
Our study	5.1 (3.6-7.5)	3.2 (2.3-4.6)	Significant (<0.001)

Our study revealed neutrophil-to-lymphocyte ratio to be significantly higher in preterm patients which is consistent with the majority of the studies stated above. NLR has been shown to significantly correlate with systemic inflammatory response and has proven effective as a prognostic marker in many diseases.

Placental lesions indicative of maternal vascular under perfusion and infective etiology of pretem labor are linked to elevated NLR to preterm delivery.[100]

Table 26. Lymphocyte to monocyte ratio (LMR) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Khatoon et al.[95]	8.98 ± 8.81	13.71 ± 13.15	Not significant (0.239)
Farzaneh et al.[8]	11.37±3.67	8.5±2.46	Significant (<0.001)
Hrubaru et al.[100]	0.86 ± 0.33	0.79 ± 0.21	Significant (0.005)
Daglar et al.[99]	3.67 ± 2.9	2.62 ± 2.4	Significant (0.02)
Horansali et al[132]	3.40±1.38	3.53±1.13	Not significant (0.46)
Mei ma et al [1]	2.94 ± 0.97	3.28 ± 0.98	Significant (0.003)
Our study	2.8 (2-4.4)	4.0 (2.7-6)	Significant (<0.001)

Our study shows that LMR was lower in preterm group than term group. This aligned with other two studies mentioned above, however other studies reported varying findings. LMR as diagnostic marker for preterm birth has not been studies much. The lymphocyte-to-monocyte ratio is an inflammatory marker that reflects the balance between the lymphocytic and monocytic immune response. In general, a lower LMR can indicate a heightened inflammatory state, which is often associated with adverse pregnancy outcomes such as preterm birth. Additionally, abnormal counts of monocytes or lymphocytes have been associated with negative outcomes in various diseases.[133]

Table 27. Platelet to lymphocyte ratio (PLR) values across various studies

Study	Mean (SD)/ Median (IQR)		Association
	Preterm	Term	
Rani et al[122]	12.95±6.3137	11.31±4.89	Significant (0.019)
Khatoon et al.[95]	132.32 ± 75.09	154.14 ± 216.20	Non-significant (0.746)
Mittal et al.[96]	155.5 ± 38.28	104.9 ± 18.91	Significant (<0.001)
Daglar et al.[99]	137 ± 40	136 ± 88	Non-significant
Hrubaru et al.[100]	286.2 ± 195.4	237.0 ± 203.8	Significant (0.007)
Horansali et al[132]	130.07±57.05	126.16 ± 39.92	Non-significant (0.076)
Djusad et al.[103]	146.44(17.08-81.01)	146.05(34.47-131.03)	Non-significant (0.475)
Mei ma et al [1]	128.75 ± 39.58	117.96 ± 31.32	Significant (0.016)
Our study	135.9 ±79.7	127.6 ±73.6	Non-significant (0.43)

Our study shows that PLR is higher in preterm group. This aligns with majority of the studies given below. It has been shown that the widely-used marker platelet-to-lymphocyte ratio (PLR) may predict thrombotic events, inflammatory illnesses, and cancers as chronic inflammation often leads to increased production of megakaryocytes and decreased levels of lymphocytes due to significant cell death. Many previous studies reported a significant association between increased PLR and major adverse outcomes in cardiovascular diseases, and reduced survival in malignancies such as pancreatic, colorectal cancer, and endometrial cancer. PLR has also been studied in various pregnancy-related conditions, including pre-eclampsia, gestational diabetes and preterm premature rupture of membranes (PPROM). PLR was found to have a positive correlation with PROM.[122] [85]

Table 28. Comparison of NLR, LMR and PLR for predicting preterm labor with optimal cut-off points

		AUC	95% CI	Cut-off point	Sensitivity (%)	Specificity (%)
Mittal et al.[96]	NLR	0.98	0.96-1.00	3.25	97.8	84.8
	PLR	0.92	0.86-0.98	111.5	93.5	69.6
Farzaneh et al.[8]	NLR	0.76	0.70- 0.82	>3.21	94.50	62.73
	PLR	0.77	0.71-0.82	>8.53	89.91	53.64
Hrubaru et al.[100]	NLR	0.694	0.56-0.84	9	71	66
	PLR	0.682	0.55-0.81	250	70	69
Yuce et al.[80]	NLR	0.92	0.84–0.97	>5	90	92.11
	PLR	0.98	0.91-0.99	>139	97.5	100
Daglar et al.[99]	LMR	0.728	0.72-0.91	4.25	75.2	78.7
Mei ma et al [1]	NLR	0.625	0.57-0.68	>3.56	78.1	46.2
	LMR	0.615	0.55-0.66	≤2.31	31.4	87.1
	PLR	0.57	0.52-0.63	>108.17	74.3	41.3
Ozer et al.[134]	NLR	0.86	0.82–0.90	3.92		
	PLR	0.56	0.50-0.61	141.83		
Balciunience et al[135]	NLR	-	-	5.97	77	95
Our study	NLR	0.73	0.66-0.80	>3.55	80.0	56.0
	LMR	0.64	0.56-0.72	<2.66	78.0	48.0
	PLR	0.52	0.44-0.60	>111.2	51.0	51.0

Most of the studies have evaluated the predictive value of the NLR, and PLR for preterm labor. LMR has not been studied much. The NLR generally shows higher prognostic value for predicting preterm labor compared to LMR and PLR, with higher AUC values and better sensitivity and specificity in several studies. Mittal et al. and Yuce et al. reported particularly high AUC values for NLR, indicating strong predictive power. PLR, while also investigated, shows more variable prognostic value, with lower AUC values in some studies. LMR has been studied less extensively but shows moderate prognostic value in predicting preterm labor.

Our study found NLR as a strong marker for prediction of preterm labor followed by LMR. PLR, however has not shown much values

STRENGTHS AND LIMITATIONS

Strengths:

1. This study provides a detailed comparison of various hematological parameters and inflammatory markers between preterm and term labor, offering a broad perspective on potential predictive markers.
2. By examining multiple markers, the study increases the likelihood of identifying reliable predictors of preterm labor, enhancing its clinical relevance.
3. The study establishes specific cut-off points and assesses the area under the curve (AUC) for each marker, providing valuable information on their diagnostic accuracy and utility.
4. The findings could have significant implications for early diagnosis and intervention in preterm labor, potentially improving maternal and neonatal outcomes.
5. This study has got further scope of correlating preterm labor with ferritin, fibrinogen, fetal fibronectin etc.

Limitations:

1. The sample size is small, it may limit the generalizability of the findings
2. Variability in Measurements: Differences in timing of blood sample collection could affect the accuracy and comparability of the hematological parameters.
3. The study might not capture changes in hematological parameters and inflammatory markers over time, limiting the understanding of their dynamic nature during pregnancy.

SUMMARY

This cross-sectional study aimed to compare hematological and inflammatory markers in preterm and term labor and to assess the utility of NLR, LMR and PLR as markers for predicting preterm labor. It was conducted between January 2023 and December 2023 in KLE's Dr. Prabhakar Kore Hospital & MRD, Belagavi. 200 antenatal patients (100 in each group) were assessed for clinical history lab investigations.

Higher age and primigravidae are associated with preterm labor. Most common risk factor for preterm labor was found to be Premature Rupture of Membranes (40%), history of previous preterm birth (18%), preeclampsia (18%) and cervical insufficiency (4%).

The hematological parameters were found to be higher in preterm group than term group with most significant being WBC count and MPV. Analysis of inflammatory markers revealed higher levels of hs- CRP, PCT and NLR and lower LMR in preterm group demonstrating its association with the outcome.

NLR was proved to be the most sensitive marker (sensitivity 80%) to predict preterm followed by LMR (sensitivity 78%).

Further research is required to validate these findings with larger sample size and exploring links between preterm and other inflammatory markers.

CONCLUSION

This study highlights the complexity of diagnosing preterm labor and the importance of identifying reliable predictive markers. In this study, age, parity, history of preterm birth, and premature rupture of membranes (PROM) emerged as significant risk factors associated with preterm labor. Hematological parameters such as white blood cell (WBC) count, mean platelet volume (MPV), and inflammatory markers like C-reactive protein (CRP) and procalcitonin (PCT) showed consistent associations with preterm labor. Elevated levels of these markers suggest underlying infection or inflammation, supporting their potential as diagnostic indicators for preterm labor. Neutrophil-to-lymphocyte ratio (NLR) emerged as a promising prognostic marker, demonstrating high diagnostic accuracy in predicting preterm labor followed by lymphocyte-to-monocyte ratio (LMR). A comprehensive assessment of these markers, along with clinical risk factors, can enhance early detection and intervention strategies, ultimately improving maternal and neonatal outcomes. Further research with large sample sizes and assessing markers over time can help in validating the diagnostic significance of these markers and to develop standardized predictive models for preterm labor assessment in clinical practice.

BIBLIOGRAPHY

1. Ma M, Zhu M, Zhuo B, Li L, Chen H, Xu L, et al. Use of complete blood count for predicting preterm birth in asymptomatic pregnant women: A propensity score-matched analysis. J Clin Lab Anal 2020;34(8):e23313.
2. Gat R, Kachko E, Kloog I, Erez O, Yitshak-Sade M, Novack V, et al. Differences in environmental factors contributing to preterm labor and PPROM – Population based study. Environ Res 2021;196:110894.
3. Romero R, Dey SK, Fisher SJ. Preterm labor: One syndrome, many causes. Science 2014;345(6198):760–5.
4. Mwaniki MK, Atieno M, Lawn JE, Newton CRJC. Long-term neurodevelopmental outcomes after intrauterine and neonatal insults: a systematic review. Lancet Lond Engl 2012;379(9814):445–52.
5. Kurban Y, Alan Y, Alan M, Kurt M, Gurlek B, Cegilli B, et al. A new marker in preterm labor: RDW and MPV. Obstet Gynecol Int J [Internet] 2020 [cited 2024 Mar 29];11(6). Available from: https://medcraveonline.com/OGIJ/a-new-marker-in-preterm-labor-rdw-and-mpv.html
6. Hudić I, Stray-Pedersen B, Tomić V. Preterm Birth: Pathophysiology, Prevention, Diagnosis, and Treatment. BioMed Res Int 2015;2015:1–1.
7. Ozel A, Alici Davutoglu E, Yurtkal A, Madazli R. How do platelet-to-lymphocyte ratio and neutrophil-to-lymphocyte ratio change in women with preterm premature rupture of membranes, and threaten preterm labor? J Obstet Gynaecol 2020;40(2):195–9.
8. Farzaneh F, Ghobadi S, Absalan A. The accuracy of selected hematology and inflammatory indices for predicting preterm labor; a cross-sectional study. Immunopathol Persa 2019;5(1):e05–e05.

9. Cunningham FG, Leveno KJ, Dashe JS, Hoffman BL, Spong CY, Casey BM, editors. Williams obstetrics. 26th edition. New York: McGraw Hill Medical; 2022.
10. Landon MB, Galan HL, Jauniaux E, Driscoll DA, Berghella V, Grobman WA, et al., editors. Gabbe's obstetrics: normal and problem pregnancies. 8th edition. Philadelphia, PA: Elsevier; 2021.
11. Vakili S, Torabinavid P, Tabrizi R, Shojazadeh A, Asadi N, Hessami K. The Association of Inflammatory Biomarker of Neutrophil-to-Lymphocyte Ratio with Spontaneous Preterm Delivery: A Systematic Review and Meta-analysis. Mediators Inflamm 2021;2021:1–7.
12. Tucker JM, Goldenberg RL, Davis RO, Copper RL, Winkler CL, Hauth JC. Etiologies of preterm birth in an indigent population: is prevention a logical expectation? Obstet Gynecol 1991;77(3):343–7.
13. Ananth CV, Vintzileos AM. Epidemiology of preterm birth and its clinical subtypes. J Matern-Fetal Neonatal Med Off J Eur Assoc Perinat Med Fed Asia Ocean Perinat Soc Int Soc Perinat Obstet 2006;19(12):773–82.
14. Goldenberg RL, Culhane JF, Iams JD, Romero R. Preterm Birth 1 Epidemiology and causes of preterm birth. 2008;371.
15. Kuppusamy N, Vidhyadevi A. Prevalence of Preterm Admissions and the Risk Factors of Preterm Labor in Rural Medical College Hospital. 2016;4(9).
16. Thangjam Chitralekha Devi, Singh HS. Prevalence and associated risk factors of preterm birth in India: A review. J Public Hlth Dev [Internet]. 2021 May 25 [cited 2024 Jun. 29];19(2):209-26. Available from: https://he01.tci-thaijo.org/index.php/AIHD-MU/article/view/246332
17. Sayres WG. Preterm labor. Am Fam Physician 2010;81(4):477–84.

18. Haram K, Mortensen JHS, Wollen A. Preterm delivery: an overview. Acta Obstet Gynecol Scand 2003;82(8):687–704.
19. Georgiou HM, Di Quinzio MKW, Permezel M, Brennecke SP. Predicting Preterm Labor: Current Status and Future Prospects. Dis Markers 2015;2015:1–9.
20. Iams JD. Prediction and Early Detection of Preterm Labor. 2003;101(2).
21. Steer P, Flint C. ABC of labor care: Preterm labor and premature rupture of membranes. BMJ 1999;318(7190):1059–62.
22. Mercer BM, Goldenberg RL, Moawad AH, Meis PJ, Iams JD, Das AF, et al. The Preterm Prediction Study: Effect of gestational age and cause of preterm birth on subsequent obstetric outcome. Am J Obstet Gynecol 1999;181(5):1216–21.
23. Heath VC, Southall TR, Souka AP, Elisseou A, Nicolaides KH. Cervical length at 23 weeks of gestation: prediction of spontaneous preterm delivery. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 1998;12(5):312–7.
24. Iams JD. Cervical ultrasonography. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 1997;10(3):156–60.
25. King G. PREGNANCY | Characteristics [Internet]. In: Encyclopedia of Dairy Sciences. Elsevier; 2002 [cited 2024 Apr 3]. page 2283–90.Available from: https://linkinghub.elsevier.com/retrieve/pii/B0122272358004399
26. Romero R, Espinoza J, Gonçalves L, Kusanovic J, Friel L, Hassan S. The Role of Inflammation and Infection in Preterm Birth. Semin Reprod Med 2007;25(1):021–39.
27. Dayal S, Hong PL. Premature Rupture of Membranes [Internet]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 Apr 7]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK532888/

28. Pisoh DW, Mbia CH, Takang WA, Djonsala OGB, Munje MC, Mforteh AA, et al. Prevalence, Risk Factors and Outcome of Preterm Premature Rupture of Membranes at the Bamenda Regional Hospital. Open J Obstet Gynecol 2021;11(03):233–51.
29. Fritz T, Källén K, Maršál K, Jacobsson B. Outcome of extremely preterm infants after iatrogenic or spontaneous birth. Acta Obstet Gynecol Scand 2018;97(11):1388–95.
30. Valencia CM, Mol BW, Jacobsson B, the FIGO Working Group for Preterm Birth. FIGO good practice recommendations on modifiable causes of iatrogenic preterm birth. Int J Gynecol Obstet 2021;155(1):8–12.
31. Koucký M, Germanová A, Hájek Z, Parízek A, Kalousová M, Kopecký P. Pathophysiology of preterm labor. Prague Med Rep 2009;110(1):13–24.
32. Guzman ER, Walters C, Ananth CV, O'Reilly-Green C, Benito CW, Palermo A, et al. A comparison of sonographic cervical parameters in predicting spontaneous preterm birth in high-risk singleton gestations. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2001;18(3):204–10.
33. DiGiulio DB, Romero R, Kusanovic JP, Gómez R, Kim CJ, Seok KS, et al. Prevalence and diversity of microbes in the amniotic fluid, the fetal inflammatory response, and pregnancy outcome in women with preterm pre-labor rupture of membranes. Am J Reprod Immunol N Y N 1989 2010;64(1):38–57.
34. Kumar D, Moore RM, Mercer BM, Mansour JM, Redline RW, Moore JJ. The physiology of fetal membrane weakening and rupture: Insights gained from the determination of physical properties revisited. Placenta 2016;42:59–73.
35. Menon R, Richardson LS. Preterm prelabor rupture of the membranes: A disease of the fetal membranes. Semin Perinatol 2017;41(7):409–19.

36. Rosen T, Kuczynski E, O'Neill LM, Funai EF, Lockwood CJ. Plasma levels of thrombin-antithrombin complexes predict preterm premature rupture of the fetal membranes. J Matern Fetal Med 2001;10(5):297–300.
37. Bryant-Greenwood GD, Yamamoto SY. Control of peripartal collagenolysis in the human chorion-decidua. Am J Obstet Gynecol 1995;172(1 Pt 1):63–70.
38. Tsuruda T, Costello-Boerrigter LC, Burnett JC. Matrix metalloproteinases: pathways of induction by bioactive molecules. Heart Fail Rev 2004;9(1):53–61.
39. Vincenti MP, White LA, Schroen DJ, Benbow U, Brinckerhoff CE. Regulating expression of the gene for matrix metalloproteinase-1 (collagenase): mechanisms that control enzyme activity, transcription, and mRNA stability. Crit Rev Eukaryot Gene Expr 1996;6(4):391–411.
40. Dutta DC, Konar H. DC Dutta's textbook of obstetrics: including perinatology and contraception. Eighth edition. New Delhi: Jaypee, The Health Sciences Publisher; 2015.
41. Yang ST, Wang PH. Preterm premature rupture of membranes (PPROM) and secondary intra-amniotic infection/inflammation. Taiwan J Obstet Gynecol 2023;62(1):187–8.
42. Hana MM, Ramzun MR, Nabela Z, Zahirah NAN, Nik Abdul Razak NNA, Azhar AR, et al. Estimation of the Hematological Change of Red Blood Cell and Platelet Count of Healthy Pregnant Women in Saudi Arabia. J Phys Conf Ser 2018;995:012024.
43. Chandra S, Tripathi AK, Mishra S, Amzarul M, Vaish AK. Physiological Changes in Hematological Parameters During Pregnancy. Indian J Hematol Blood Transfus 2012;28(3):144–6.

44. Purohit G, Shah T, Harsoda DJM. Hematological profile of normal pregnant women in Western India. Sch. J. App. Med. Sci., 2015; 3(6A):2195-2199

45. Moghaddam Tabrizi F, Barjasteh S. Maternal Hemoglobin Levels during Pregnancy and their Association with Birth Weight of Neonates. Iran J Pediatr Hematol Oncol 2015;5(4):211–7.

46. Taylor DJ, Lind T. RED CELL MASS DURING AND AFTER NORMAL PREGNANCY. BJOG Int J Obstet Gynaecol 1979;86(5):364–70.

47. Reese JA, Peck JD, Deschamps DR, McIntosh JJ, Knudtson EJ, Terrell DR, et al. Platelet Counts during Pregnancy. N Engl J Med 2018;379(1):32–43.

48. Quinn JA, Munoz FM, Gonik B, Frau L, Cutland C, Mallett-Moore T, et al. Preterm birth: Case definition & guidelines for data collection, analysis, and presentation of immunisation safety data. Vaccine 2016;34(49):6047–56.

49. Artunc Ulkumen B, Pala HG, Calik E, Oruc Koltan S. Platelet distribution width (PDW): a putative marker for threatened preterm labor. Pak J Med Sci [Internet] 2014 [cited 2024 Apr 2];30(4). Available from: http://pjms.com.pk/index.php/pjms/article/view/4991

50. Dundar O, Yoruk P, Tutuncu L, Akyol Erikci A, Muhcu M, Ergur AR, et al. Longitudinal study of platelet size changes in gestation and predictive power of elevated MPV in development of pre-eclampsia. Prenat Diagn 2008;28(11):1052–6.

51. Beyan C, Kaptan K, Ifran A. Platelet count, mean platelet volume, platelet distribution width, and plateletcrit do not correlate with optical platelet aggegation responses in healthy volunteers. J Thromb Thrombolysis 2006;22(3):161–4.

52. Gasparyan AY, Ayvazyan L, Mikhailidis DP, Kitas GD. Mean platelet volume: a link between thrombosis and inflammation? Curr Pharm Des 2011;17(1):47–58.

53. Nooh AM, Abdeldayem HM. Changes in Platelet Indices during Pregnancy as Potential Markers for Prediction of Preeclampsia Development. Open J Obstet Gynecol 2015;05(12):703–12.
54. Myatt L, Clifton RG, Roberts JM, Spong CY, Hauth JC, Varner MW, et al. First-Trimester Prediction of Preeclampsia in Nulliparous Women at Low Risk: Obstet Gynecol 2012;119(6):1234–42.
55. Keren-Politansky A, Breizman T, Brenner B, Sarig G, Drugan A. The coagulation profile of preterm delivery. Thromb Res 2014;133(4):585–9.
56. Kawthalkar S. Essentials of Clinical Pathology. 1/e. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd;2010.
57. Gruys E, Toussaint MJM, Niewold TA, Koopmans SJ. Acute phase reaction and acute phase proteins. J Zhejiang Univ-Sci B 2005;6(11):1045–56.
58. Jain S, Gautam V, Naseem S. Acute-phase proteins: As diagnostic tool. J Pharm Bioallied Sci 2011;3(1):118.
59. Pepys MB. C-reactive protein fifty years on. Lancet Lond Engl 1981;1(8221):653–7.
60. Offenbacher S, Lieff S, Boggess KA, Murtha AP, Madianos PN, Champagne CM, et al. Maternal periodontitis and prematurity. Part I: Obstetric outcome of prematurity and growth restriction. Ann Periodontol 2001;6(1):164–74.
61. Kluft C, de Maat MPM. Sensitive markers of inflammation make it possible to study the chronic process: the rise of interest in low levels of C-reactive protein. Vascul Pharmacol 2002;39(3):99–104.
62. Shahshahan Z, Iravani H. Comparison of CRP and ALK-P serum levels in prediction of preterm delivery. Adv Biomed Res 2016;5(1):17.

63. Dati F, Schumann G, Thomas L, Aguzzi F, Baudner S, Bienvenu J, et al. Consensus of a group of professional societies and diagnostic companies on guidelines for interim reference ranges for 14 proteins in serum based on the standardization against the IFCC/BCR/CAP Reference Material (CRM 470). International Federation of Clinical Chemistry. Community Bureau of Reference of the Commission of the European Communities. College of American Pathologists. Eur J Clin Chem Clin Biochem J Forum Eur Clin Chem Soc 1996;34(6):517–20.

64. Kamath DY, Xavier D, Sigamani A, Pais P. High sensitivity C-reactive protein (hsCRP) & cardiovascular disease: An Indian perspective. Indian J Med Res 2015;142(3):261–8.

65. Black S, Kushner I, Samols D. C-reactive Protein. J Biol Chem 2004;279(47):48487–90.

66. Roberts WL. CDC/AHA Workshop on Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: Laboratory Tests Available to Assess Inflammation—Performance and Standardization: A Background Paper. Circulation [Internet] 2004 [cited 2024 May 4];110(25). Available from: https:// www.ahajournals. org/doi/10.1161/01 .CIR .0000148986.52696.07

67. Watts DH, Krohn MA, Wener MH, Eschenbach DA. C-reactive protein in normal pregnancy. Obstet Gynecol 1991;77(2):176–80.

68. Shahshahan Z, Rasouli O. The use of maternal C-reactive protein in the predicting of preterm labor and tocolytic therapy in preterm labor women. Adv Biomed Res 2014;3:154.

69. Gulhar R, Ashraf MA, Jialal I. Physiology, Acute Phase Reactants [Internet]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 [cited 2024 May 8]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK519570/

70. Mangogna A, Belmonte B, Agostinis C, Ricci G, Gulino A, Ferrara I, et al. Pathological Significance and Prognostic Value of Surfactant Protein D in Cancer. Front Immunol 2018;9:1748.

71. Joyce CM, Deasy S, Abu H, Lim YY, O'Shea PM, O'Donoghue K. Reference values for C-reactive protein and procalcitonin at term pregnancy and in the early postnatal period. Ann Clin Biochem Int J Lab Med 2021;58(5):452–60.

72. Paccolat C, Harbarth S, Courvoisier D, Irion O, de Tejada BM. Procalcitonin levels during pregnancy, delivery and postpartum. J Perinat Med 2011;39(6):679–83.

73. Tujula B, Kokki H, Räsänen J, Kokki M. Procalcitonin; a feasible biomarker for severe bacterial infections in Obstetrics and Gynecology? Acta Obstet Gynecol Scand 2018;97(5):505–6.

74. Köroğlu N, Tayyar A, Soydar A, Albayrak N, Aydın T, Çetin BA. Pre-delivery fibrinogen level is a predictor for severity of placental abruption. Eur J Obstet Gynecol Reprod Biol X 2023;20:100264.

75. Van Den Broek NR, Letsky EA. Pregnancy and the erythrocyte sedimentation rate. Br J Obstet Gynaecol 2001;108(11):1164–7.

76. Levin I, Helpman L, Maslovitz S, Pauzner D, Shapira I, Gamzu R, et al. Erythrocyte aggregation is increased in preterm premature rupture of the membranes. Eur J Obstet Gynecol Reprod Biol 2006;125(2):199–201.

77. Kristensen K, Wide-Swensson D, Lindstrom V, Schmidt C, Grubb A, Strevens H. Serum amyloid a protein and C-reactive protein in normal pregnancy and preeclampsia. Gynecol Obstet Invest 2009;67(4):275–80.

78. Tamura T. Serum Ferritin: A Predictor of Early Spontaneous Preterm Delivery. Obstet Gynecol 1996;87(3):360–5.

79. Zahorec R. Neutrophil-to-lymphocyte ratio, past, present and future perspectives. Bratisl Med J 2021;122(07):474–88.

80. Yuce E. Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR) Can Predict Spontaneous Preterm Birth? J Inflamm Res 2023;Volume 16:2423–9.

81. Durmus E, Kivrak T, Gerin F, Sunbul M, Sari I, Erdogan O. Neutrophil-to-Lymphocyte Ratio and Platelet-to-Lymphocyte Ratio are Predictors of Heart Failure. Arq Bras Cardiol [Internet] 2015 [cited 2024 May 7];Available from: https://www.scielo.br/scielo.php?script=sci_arttext&pid=S0066-782X2015002500606

82. Kirbas A, Biberoglu E, Daglar K, İskender C, Erkaya S, Dede H, et al. Neutrophil-to-lymphocyte ratio as a diagnostic marker of intrahepatic cholestasis of pregnancy. Eur J Obstet Gynecol Reprod Biol 2014;180:12–5.

83. Kurt R, Güler A, Silfeler D, Özçil M, Karateke A, Hakverdi A. Relation of inflammatory markers with both presence and severity of hyperemesis gravidarum. Pol Gynaecol [Internet] 2014 [cited 2024 May 7];85(8). Available from: http://www.journalssystem.com/gp/Relation-of-inflammatory-markers-with-both-presence-and-severity-of-hyperemesis gravidarum,1776,0,2.html

84. Kurtoglu E, Kokcu A, Celik H, Tosun M, Malatyalioglu E. May ratio of neutrophil to lymphocyte be useful in predicting the risk of developing preeclampsia? A pilot study. J Matern Fetal Neonatal Med 2015;28(1):97–9.

85. Toprak E, Bozkurt M, Dinçgez Çakmak B, Özçimen EE, Silahlı M, Ender Yumru A, et al. Platelet to Lymphocyte Ratio: A New Inflammatory Marker For Diagnosis

of Preterm Premature Rupture of Membranes. J Turk-Ger Gynecol Assoc [Internet] 2017 [cited 2024 May 7];Available from: http://cms.galenos.com.tr/Uploads/Article_1877/JTGGA-18-3.pdf

86. Fest J, Ruiter R, Ikram MA, Voortman T, Van Eijck CHJ, Stricker BH. Reference values for white blood-cell-based inflammatory markers in the Rotterdam Study: a population-based prospective cohort study. Sci Rep 2018;8(1):10566.
87. Jhamb R, Kumar R, Gogoi P, Ranga GS, Kashyap B. Reference values of neutrophil lymphocyte ratio and platelet lymphocyte ratio in healthy adults in a tertiary care center in north india. Bionature [Internet]. 2020 Dec. 31 [cited 2024 Jun. 29];40(3):44-52. Available from: https://globalpresshub.com/index.php/BN/article/view/911
88. Ravindra R, Ramamurthy P, Aslam S SM, Kulkarni A, K S, Ramamurthy PS. Platelet Indices and Platelet to Lymphocyte Ratio (PLR) as Markers for Predicting COVID-19 Infection Severity. Cureus [Internet] 2022 [cited 2024 May 11];Available from: https://www.cureus.com/articles/108383-platelet-indices-and-platelet-to-lymphocyte-ratio-plr-as-markers-for-predicting-covid-19-infection-severity
89. Maravi P, Kushwah SS, Shakya M, Rure D, Prajapati J, Rawat M. Relationship of PLR, NLR and LMR with Health Section Metabolic Syndrome in Schizophrenic Patients on Antipsychotics: A Longitudinal Case-control Study. J Clin Diagn Res [Internet] 2023 [cited 2024 May 1];Available from:https://www.jcdr.net/article_fulltext.asp?issn=0973709x&year=2023&volume=17&issue=2&page=VC06&issn=0973-709x&id=17551

90. Cha HH, Kim JM, Kim HM, Kim MJ, Chong GO, Seong WJ. Association between gestational age at delivery and lymphocyte-monocyte ratio in the routine second trimester complete blood cell count. Yeungnam Univ J Med 2021;38(1):34–8.

91. Pan YC, Jia ZF, Cao DH, Wu YH, Jiang J, Wen SM, et al. Preoperative lymphocyte-to-monocyte ratio (LMR) could independently predict overall survival of resectable gastric cancer patients. Medicine (Baltimore) 2018;97(52):e13896.

92. Romano A, Parrinello NL, Vetro C, Chiarenza A, Cerchione C, Ippolito M, et al. Prognostic meaning of neutrophil to lymphocyte ratio (NLR) and lymphocyte to monocyte ration (LMR) in newly diagnosed Hodgkin lymphoma patients treated upfront with a PET-2 based strategy. Ann Hematol 2018;97(6):1009–18.

93. Feng QT, Chen C, Yu QY, Chen SY, Huang X, Zhong YL, et al. The benefits of higher LMR for early threatened abortion: A retrospective cohort study. PloS One 2020;15(4):e0231642.

94. Jayaram A, Kanninen T, Sisti G, Inglis SR, Morgan N, Witkin SS. Pregnancy History Influences the Level of Autophagy in Peripheral Blood Mononuclear Cells From Pregnant Women. Reprod Sci Thousand Oaks Calif 2018;25(9):1376–81.

95. Khatoon F, Gupta H, Sinha P, Tiwari K. Prediction of Preterm Birth on the Basis of Complete Blood Count Parameters. J South Asian Fed Obstet Gynaecol 2021;12(5):288–90.

96. Mittal S, Vyas J. A study to evaluate the association of maternal inflammatory biomarkers with spontaneous preterm delivery. Indian j appl res 2023;13(3).

97. Sureshbabu RP, Aramthottil P, Anil N, Sumathy S, Varughese SA, Sreedevi A, et al. Risk Factors Associated with Preterm Delivery in Singleton Pregnancy in a Tertiary Care Hospital in South India: A Case Control Study. Int J Womens Health 2021;13:369–77.

98. Rao CR, de Ruiter LEE, Bhat P, Kamath V, Kamath A, Bhat V. A Case-Control Study on Risk Factors for Preterm Deliveries in a Secondary Care Hospital, Southern India. ISRN Obstet Gynecol 2014;2014:935982.

99. Daglar HK, Kirbas A, Kaya B, Kilincoglu F. The value of complete blood count parameters in predicting preterm delivery. Eur Rev Med Pharmacol Sci 2016;20(5):801–5.

100. Hrubaru I, Motoc A, Moise ML, Miutescu B, Citu IM, Pingilati RA, et al. The Predictive Role of Maternal Biological Markers and Inflammatory Scores NLR, PLR, MLR, SII, and SIRI for the Risk of Preterm Delivery. J Clin Med [Internet] 2022 [cited 2024 Jun 1];11(23). Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9738289/

101. Isık H, Aynıoglu O, Sahbaz A, Arıkan İ, Karçaaltıncaba D, Sahin H, et al. Can plateletcrit, an underestimated platelet parameter, be related with preterm labor? J Obstet Gynaecol 2015;35(7):676–80.

102. Cl M, Hg A, Hbs J, Gr A, A J. Neutrophil to Lymphocyte Ratio and Red Blood Cell Distribution Width Levels in Preterm vs. Term Births. J Mol Genet Med [Internet] 2018 [cited 2024 Mar 29];12(1). Available from: https://www.omicsonline.org/open-access/neutrophil-to-lymphocyte-ratio-and-red-blood-cell-distribution-width-levels-in-preterm-vs-term-births-1747-0862-1000317-97867.html

103. Djusad S, Surya IU, Purnadiputra A, Pribadi MI, Wicaksono MA. Neutrophil-Lymphocyte Ratio, Platelet-Lymphocyte Ratio, and Length of Cervix as Predictors of Premature Delivery during the Covid-19 Pandemic at Cipto Mangunkusumo General Hospital. Indones J Obstet Gynecol 2024;23–7.

104. Esposito G, Mauri PA, Cipriani S, Franchi M, Corrao G, Parazzini F. The role of maternal age on the risk of preterm birth among singletons and multiples: a retrospective cohort study in Lombardy, Norther Italy. BMC Pregnancy Childbirth 2022;22:234.

105. Tseng KT, Peng CC, Chang JH, Hsu CH, Lin CY, Jim WT, et al. The impact of advanced maternal age on the outcomes of very low birth weight preterm infants. Medicine (Baltimore) 2019;98(5):e14336.

106. Reddy KM, Ravula SR, Palakollu S, Betha K. Prevalence of preterm birth and perinatal outcome: A rural tertiary teaching hospital-based study. J Fam Med Prim Care 2022;11(7):3909–14.

107. MAVALANKAR DV, GRAY RH, TRIVEDI CR. Risk Factors for Preterm and Term Low Birthweight in Ahmedabad, India. Int J Epidemiol 1992;21(2):263–72.

108. Koullali B, van Zijl MD, Kazemier BM, Oudijk MA, Mol BWJ, Pajkrt E, et al. The association between parity and spontaneous preterm birth: a population based study. BMC Pregnancy Childbirth 2020;20(1):233.

109. Konyala V, M P, K.B S. A retrospective analysis of maternaland fetal outcomes of preterm deliveries in a tertiary care hospital in south india. Int J Sci Res 2021;42–4.

110. Yadav R, Kumar PS, B. MK, Kumar GM, N C. Maternal risk factors associated with preterm birth in atertiary rural care center. Int J Sci Res 2021;38–42.

111. Mahajan A, Magon S. Study of Risk Factors for Preterm Births in a Teaching Hospital: A Prospective Study. Int J Med Dent Sci 2017;6(1):1407.

112. Renzo GCD, Giardina I, Rosati A, Clerici G, Torricelli M, Petraglia F. Maternal risk factors for preterm birth: a country-based population analysis. Eur J Obstet Gynecol Reprod Biol 2011;159(2):342–6.

113. Sehgal A, Telang S, Passah SM, Jyothi MC. Maternal and Neonatal Profile and Immediate Outcome in Extremely Low Birth Weight Babies in Delhi. Trop Doct 2004;34(3):165–8.

114. Roy KK, Baruah J, Kumar S, Malhotra N, Deorari AK, Sharma JB. Maternal antenatal profile and immediate neonatal outcome in VLBW and ELBW babies. Indian J Pediatr 2006;73(8):669–73.

115. Bangal V, Shinde K, Khanvelkar G, Patil N. A study of risk factors and perinatal outcome in preterm labor at tertiary care hospital. Int J Biomed Res 2012;3.

116. Mercer BM, Goldenberg RL, Moawad AH, Meis PJ, Iams JD, Das AF, et al. The Preterm Prediction Study: Effect of gestational age and cause of preterm birth on subsequent obstetric outcome. Am J Obstet Gynecol 1999;181(5):1216–21.

117. Koire A, Chu DM, Aagaard K. Family history is a predictor of current preterm birth. Am J Obstet Gynecol MFM 2021;3(1):100277.

118. Mohapatra V, Saraogi S, Misra S. Demographic Profile, Etiology, and Perinatal Outcome Associated With Preterm Birth in a Tertiary Hospital of Eastern India: A Retrospective Study. Cureus [Internet] 2022 [cited 2024 Apr 3];Available from: https://www.cureus.com/articles/101101-demographic-profile-etiology-and-perinatal-outcome-associated-with-preterm-birth-in-a-tertiary-hospital-of-eastern-india-a-retrospective-study

119. P SS, Mishra S. A Prospective study of etiology and outcome of Preterm Labor in a Rural Medical College. Obs Gyne Rev J Obstet Gynecol 2016;2(4):44–8.

120. Addisu D, Melkie A, Biru S. Prevalence of Preterm Premature Rupture of Membrane and Its Associated Factors among Pregnant Women Admitted in Debre Tabor General Hospital, North West Ethiopia: Institutional-Based Cross-Sectional Study. Obstet Gynecol Int 2020;2020:4034680.

121. Owen J. Evaluation of the cervix by ultrasound for the prediction of preterm birth. Clin Perinatol 2003;30(4):735–55.

122. Rani A, Karya U, Nehra S. Significance of maternal hemogram parameters as new inflammatory markers for prediction of threatened preterm labor and preterm premature rupture of membranes. Int J Reprod Contracept Obstet Gynecol 2023;12(6):1701–5.

123. Malathi RM, Ramesh S, Kumar RA. Comparative study of hemoglobin and packed volume levels in term and preterm babies delivered in RMMCH.

124. Rahman MA, Khan MN, Rahman MM. Maternal anaemia and risk of adverse obstetric and neonatal outcomes in South Asian countries: A systematic review and meta-analysis. Public Health Pract 2020;1:100021.

125. Symington EA, Baumgartner J, Malan L, Wise AJ, Ricci C, Zandberg L, et al. Maternal iron-deficiency is associated with premature birth and higher birth weight despite routine antenatal iron supplementation in an urban South African setting: The NuPED prospective study. PLoS ONE 2019;14(9):e0221299.

126. Alva SR. Comparative Study of Platelet Indices between Term, Preterm and Small for Gestational Age Newborns. Scholars journal of applied medical sciences 2016;4(11):3985-89.

127. Kannar V, Mariyappa N, Deepthi A, Kumar M, Junjegowda K. Effect of gestational age, prematurity and birth asphyxia on platelet indices in neonates. J Clin Neonatol 2014;3:144.

128. Wasiluk A, Mantur M, Kemona H, Szczepański M, Jasińska E, Milewski R. Thrombopoiesis in small for gestational age newborns. Platelets 2009;20(7):520–4.

129. Kumawat P, Fiza B, Sinha M, Yogi J. Role of C-reactive Protein and Serum Magnesium in Preterm Labor. J Mahatma Gandhi Univ Med Sci Technol 2020;4:68–70.

130. Najat Nakishbandy BM, Barawi SAM. Level of C - reactive protein as an indicator for prognosis of premature uterine contractions. J Prenat Med 2014;8(1–2):25–30.

131. Ducarme G, Desroys du Roure F, Le Thuaut A, Grange J, Vital M, Dimet J. Efficacy of serum procalcitonin to predict spontaneous preterm birth in women with threatened preterm labor: a prospective observational study. BMC Pregnancy Childbirth 2018;18:65.

132. Eriç Horasanlı J, Alp EC, Bülbül R. Evaluation of Complete Blood Cell Count Parameters in the Diagnosis of Threatened Preterm Labor and Premature Rupture of Membranes. Dubai Med J 2022;5(3):157–62.

133. Yoon CI, Bae SJ, Cha CH, Park SE, Ahn SG, Jeong J. A negative prognosis of radiotherapy-induced lower lymphocyte to monocyte ratio in patients with breast cancer. Ann Oncol 2019;30:v74.

134. Özer PT, Özer M, Oğlak SC, Budak A, İnan AH, Tosun G, et al. Evaluating the Predictive Potential of Neutrophil-to-Lymphocyte and Platelet-to-Lymphocyte Ratios in the Second-Trimester Preterm Premature Rupture of Membranes (PPROM): A Retrospective Case-Control Study. Clin Exp Obstet Gynecol 2024;51(2):28.

135. Balciuniene G, Kvederaite-Budre G, Gulbiniene V, Dumalakiene I, Viliene R, Pilypiene I, et al. Neutrophil–lymphocyte ratio for the prediction of histological chorioamnionitis in cases of preterm premature rupture of membranes: a case-control study. BMC Pregnancy Childbirth 2021;21:656.

ANNEXURES

INFORMED CONSENT FORM

"A COMPARATIVE STUDY OF HEMATOLOGICAL AND INFLAMMATORY MARKERS IN PRETERM LABOR AND TERM LABOR."

Student/Principal Investigator: Reg. No. BN0121001

Guide/Co Investigators: Dr._____

Objective: The purpose of this study is to predict preterm labor with haematological and inflammatory markers.

Explanation of procedure: During this study, your blood will be withdrawn for routine investigation during pregnancy. The principal investigator of the study is Reg. No. BN0121001 under the guidance of Dr._____.

If you agree to enroll yourself in this study, your blood reports will be used for research purpose.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: The benefit would be to know a better way to predict preterm labor for better management and to prevent harmful outcomes due to the same.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: "Reg. No. BN0121001, Department of Pathology, J.N. Medical College"

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**A COMPARATIVE STUDY OF HEMATOLOGICAL AND INFLAMMATORY MARKERS IN PRETERM LABOR AND TERM LABOR.**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Investigator: Reg. No. BN0121001

Signature of the investigator:

PROFORMA

PATIENT HISTORY IP no:

- Name:
- Age:
- Period of gestation: G= P= L= A= D=
- History of present pregnancy:

  bleeding per vagina / cervical insufficiency/ bacterial vaginosis/ urinary tract infection/ preeclampsia/ PROM (or PPROM)
- History of past pregnancy: h/o previous preterm labor
  h/o previous still birth
- Menstrual history:
- Past medical/surgical history: hypertension/ hypothyroidism/ crohn,s/ SLE/ Rheumatoid arthritis etc
- Family history:
- Personal history:

General Physical Examination

- Build:
- Nutrition:
- Height:
- Weight:
- Pallor:
- Jaundice:
- Oedema of legs:
- Pulse:
- Blood pressure:

Systemic Examination

- CNS:
- CVS:
- RS:
- PA:

Obstetrical Examination

- Abdominal,
- vaginal.

Routine investigation

- Blood
- urine

USG Findings:

Hematological investigation

HB		**TC**	
RBC COUNT		**DC NEUTROPHILS**	
PCV (HCT)		**LYMPHOCYTES**	
MCV		**EOSINOPHILS**	
MCH		**MONOCYTES**	
MCHC		**ANC**	
RETIC COUNT		**ALC**	
RDW		**AEC**	
PLATELET COUNT		**AMC**	
MPV			

Inflammatory Markers

Hs- CRP	
PCT	
NLR	
LMR	
PLR	

KEY TO MASTER CHART

S no.	-	Serial Number
POG	-	Period of Gestation
PROM	-	Prelabor Rupture of Membranes
PE	-	Preeclampsia
OLIGO	-	Oligohydramnios
HTN	-	Hypertension
H/O PTB	-	History of preterm birth
H/O SB	-	History of spontaneous preterm birth
H/O BV	-	History of Bacterial Vaginosis
H/O BPV IN FT	-	History of Bleeding per vagina in first trimester
H/O UTI	-	History of Urinary Tract Infection
CX INS	-	Cervical insufficiency
CRP	-	C- reactive Protein
PCT	-	Procalcitonin
Hb	-	Hemoglobin
RBC	-	Red Blood Cell
HCT	-	Hematocrit
MCV	-	Mean Corpuscular Volume

MCH	-	Mean Corpuscular Hemoglobin
MCHC	-	Mean Corpuscular Hemoglobin Concentration
RDW	-	Red Cell Distribution Width
WBC	-	White Blood Cells
ANC	-	Absolute Neutrophil Count
ALC	-	Absolute Lymphocyte Count
AMC	-	Absolute Monocyte Count
AEC	-	Absolute Eosinophil Count
MPV	-	Mean Platelet Volume
NLR	-	Neutrophil to Lymphocyte Ratio
PLR	-	Platelet to Lymphocyte Ratio
LMR	-	Lymphocyte to Monocyte Ratio
Y	-	Yes
N	-	No
0	-	Term Group
1	-	Preterm Group

S.No.	IP no.	Name	Group	Age	POG	Obstretic score	Prity	PROM	PE	OLIGO	HTN	H/O PTB	H/O SB	H/O BV	H/O BPV IN FT	H/O UTI	HYPOTHY ROIDISM	CX INS	CRP(mg/dl)	PCT U/L	Hb(g/dl)	RBC (x10^6/ul)	HCT	MCV	MCH	MCHC	RDW	Retic count	WBC x10^3/ul	Platelet (x10^3/ul)	ANC	ALC	AMC	AEC	MPV	NLR	LMR	PLR	Group_LMR
1	1206102	PRASANNA KUMARI	Preterm	28	33.4	P1	1	N	Y	N	N	N	N	N	N	N	N	N	8.2	2.94	13.4	4.74	44.8	100	28.30	28.3	12.7	0.5	13.9	208	10.3	2.7	0.8	0	10.3	3.8	3.3	77	0
2	1205617	RAJEHSWARI SANTOSH	Preterm	29	35.5	P1	1	N	N	N	Y	N	N	N	N	N	N	N	39.5	5.5	10	4.18	30.2	88.6	27.30	32.2	12.6	1	10.8	276	8.2	1.5	0.8	0.3	9.8	5.4	1.8	184	0
14	1199741	BISMILLA MULLA	Preterm	30	32.4	G4P2L2A1	2	Y	N	N	N	N	N	N	N	N	N	N	112.2	3.98	13.1	4.29	39.3	80.5	30.00	30.5	12.5	0.5	11.7	158	10.5	0.8	0.2	0.1	9.8	13.1	4	197.5	0
18	1130223	CHAITRA BALESH	Preterm	23	30.5	G3P2L1D1	2	N	N	N	N	N	N	N	N	Y	N	N	22.1	1.5	11.9	3.95	36.7	88.5	28.70	32.4	18	1.3	18.2	181	13.6	3.3	1.1	0.2	9.8	4.12	3	54.8	0
26	1166111	GANGAVVA IRANNA	Preterm	27	30.6	G2P1L1	1	N	Y	N	N	N	N	N	N	N	N	N	74.9	1.5	11.7	4.04	36.3	85.2	32.10	31.5	14.5	0.5	5.5	163	4.6	0.3	0.4	0.1	11.1	15.3	0.75	543.3	0
12	1200197	PRAMILA YELAJAR	Preterm	28	32.1	P1	1	Y	N	N	N	N	N	N	N	N	N	N	115.2	1.30	9.8	3.50	30.3	86.6	28.00	32.3	23.3	3.4	22.4	184	18.6	2.5	1.3	0.0	9.9	7.44	1.9	73.6	0
98	7223592	GANGAVVA	Preterm	28	28.5	G3P2L2	2	N	N	N	N	Y	N	N	N	N	N	N	20.4	1.2	10	2.93	29.2	99.7	34.10	32.4	14.9	1.6	14.28	399	11.4	1.84	0.9	0.06	9.4	6.1	2	216.8	0
91	7457315	NINGAVVA	Preterm	28	29.3	G3P2L2	2	Y	N	N	N	N	N	N	N	N	N	N	67.4	1	11.4	5.03	36.4	72.4	22.60	31.2	17	1.2	12.5	297	10.3	1.6	0.3	0.3	8.6	6.4	5.3	185.6	0
97	7235438	SEETA	Preterm	22	34	P1	1	N	Y	N	N	N	N	N	N	N	N	N	13	1	8.9	3.20	26.4	82.4	28.00	33.9	19	1.4	17.7	185	15.5	1	1	0.2	9.4	15.5	1	185	0
44	1166209	NEHA	Preterm	29	30.5	G2A1	1	N	N	N	N	N	N	Y	N	N	N	N	36.5	0.69	11	3.65	33.7	92.3	30.10	32.6	16.9	0.5	14.61	396	10.6	2.4	1.8	0.3	9.5	5.3	1.3	165	0
13	1200066	DEEPA RAMESH	Preterm	20	34.5	P1	1	N	N	N	N	N	N	Y	N	N	N	N	99.9	0.64	11.0	3.91	33	82.5	29.40	33.5	14.5	1.5	12.6	147	9.1	2.6	0.7	0.2	10.3	3.5	3.7	56.5	0
6	1203302	SUDHA SHANKAR	Preterm	25	35.5	G2A1	2	N	Y	N	N	Y	N	N	N	N	N	N	114.1	0.59	6.4	3.10	20.9	67.4	20.60	30.6	23.6	0.5	19.4	260	16.7	1.4	1.1	0.2	9.2	11.9	1.2	185.7	0
5	1202485	ANSAR WASEEM	Preterm	19	31.3	P1	1	Y	N	N	N	N	N	N	N	N	N	N	12.5	0.58	8.1	2.78	26.4	95	29.10	33.1	14	1	14.4	230	8.8	4.1	0.9	0.6	9.4	2.1	4.5	56	0
4	1203678	KIRTI PARSHURAM	Preterm	19	35.6	P1	1	N	N	N	N	N	N	N	Y	N	N	N	71.3	0.41	8.8	3.88	26.4	75.1	25.00	32.1	16.2	0.5	17.3	205	12.8	2.6	1.5	0.5	10.3	4.9	1.7	78.8	0
22	1164369	MUSKAN SALMAN ATTAR	Preterm	22	34.2	P1	1	N	N	N	N	N	N	Y	N	N	N	N	39.8	0.37	11.6	4.21	35.4	84.1	27.60	32.8	14.8	0.5	16.81	331	13.6	1.8	1.2	0.15	9.4	7.5	1.5	106.6	0
103	1156806	HUVAKKA VITHAL	Term	19	39.6	P1	1	Y	N	N	N	N	N	N	N	N	N	N	21.6	0.36	10.4	3.74	32.3	86.3	27.80	32.2	21	1.1	7.5	364	4.7	2	0.3	0.5	10	2.3	6.6	182	0
69	7313545	SWATI	Preterm	24	30.5	G3P2L1A1	2	N	N	N	Y	N	N	Y	N	N	N	N	36.5	0.34	8.1	3.22	27.1	84.2	25.20	29.9	16.8	4	14.29	343	10.7	3.15	0.85	0.16	11.2	3.3	3.7	108.8	0
55	1174683	MAYA ANMOL	Preterm	23	34.5	P1	1	Y	N	N	N	N	N	N	N	N	N	N	13.1	0.33	12.6	4.06	35	98.2	33.50	35	12	0.5	10.7	150	7.7	2.3	0.2	0.1	8.5	3.34	11.5	65.2	0
45	1167815	VARSHA GANJI	Preterm	25	32.6	G2P1L1	1	N	Y	N	N	N	N	Y	N	N	N	N	24.5	0.31	9.9	4.40	31.8	71.9	22.40	31.2	16.5	1.5	21.2	295	19.8	0.7	0.5	0.1	10.1	28.2	1.4	421.4	0
90	7167757	NASIMA	Preterm	25	33	P1	1	Y	N	N	N	N	N	N	N	N	N	N	99	0.3	9.4	3.60	31.7	88.1	26.20	29.7	17.5	0.75	19.1	271	14.8	2.5	1.3	0.5	9.5	5.9	1.9	108.4	0
35	1157465	PREETI	Preterm	27	35.2	G3P2L2	2	N	N	N	N	N	N	Y	N	Y	N	N	44.5	0.29	10.7	4.40	35.3	80.2	24.30	30.3	16.6	0.6	10	196	7.21	2.42	0.32	0.03	11.6	3	7.5	80.9	0
89	7856322	SUNITA GHATIGE	Preterm	32	33.3	P1	1	N	N	N	N	N	N	Y	N	Y	N	N	52.8	0.28	8.5	2.58	27.2	105.4	32.90	31.3	15.2	1.5	9.38	230	7.41	1.43	0.45	0.09	11.2	1.7	3.17	160.8	0
15	1201383	SHIVALEELA ANAND	Preterm	31	35.2	G2P1L1	1	N	Y	Y	N	Y	N	N	N	N	N	N	36.5	0.25	11.4	3.53	34.2	79.2	29.00	32.2	12.5	0.7	9.5	164	7	1.7	0.7	0.1	7.9	4.1	2.4	96.5	0
78	6256747	SHEETAL	Preterm	23	34.2	P1	1	N	N	N	N	N	N	Y	N	N	N	N	36.8	0.25	10.8	4.29	34.2	79.7	25.20	31.6	16	1.2	12.4	263	8.8	2.1	0.9	0.6	8.3	4.1	2.3	125.3	0
109	1172614	DHANESHWARI	Term	25	40	P1	1	N	N	N	N	N	N	Y	N	N	N	N	90.3	0.25	11.3	3.87	35.9	92.9	29.20	31.4	14.9	1.6	16.2	224	12.3	2.8	1.1	0	8.8	4.3	2.5	80	0
32	1187500	HARSHA YALLAPPA	Preterm	33	32.1	G3A2	1	N	N	Y	N	N	N	N	N	N	N	N	35.5	0.2	10.5	4.30	29.9	79	25.00	30	16	0.8	8.2	299	6.5	0.9	0.7	0.1	9.6	7.2	1.2	332.2	0
71	7266231	SANDHYA	Preterm	20	34.2	P1	1	N	Y	N	N	N	N	N	N	N	N	N	11.6	0.2	85	4.10	27.3	66.6	20.90	31.3	25	2.1	12.5	236	9.9	1.7	0.7	0.1	8.9	5.8	2.4	138.8	0
76	7167434	REKHA	Preterm	23	30.5	P1	1	Y	N	N	N	N	N	N	N	N	N	N	47.1	0.2	10.3	3.71	36.1	97.3	27.80	28.5	14.2	2	16.5	302	12.2	3.38	0.75	0.15	9.6	3.6	4.5	89.3	0
88	7577562	AFAREEN	Preterm	31	31	G3P2L1A1	2	N	N	N	N	N	Y	N	N	N	N	N	19.5	0.2	11.7	3.88	37.3	96.1	30.20	31.4	14.3	1.93	12.69	241	8.7	2.97	0.88	0.3	10.9	2.9	3.3	81.1	0
39	1160403	MUKTA MUKUND	Preterm	26	28.5	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	35.7	0.18	9.1	3.31	28	84.5	27.50	32.5	13	0.5	16.81	132	13.6	1.8	1.2	0.15	9.5	7.5	1.5	73.3	0
34	11551887	TASBAYA ASLAM	Preterm	23	34	P1	1	N	Y	Y	N	N	N	N	N	N	N	N	104.8	0.14	10.7	4.94	37.6	82.1	23.40	28.5	16.2	2.5	32.1	306	29.9	1.5	0.6	0	8.3	19.9	2.5	204	0
24	1167512	NIKKITA PATIL	Preterm	25	30.6	G2A1	1	N	Y	Y	N	N	N	N	N	N	N	N	25.4	0.13	11.5	3.59	33.6	99.5	29.50	33.4	13.7	1.2	12.3	199	8.4	3.1	0.7	0.1	8.9	2.7	4.4	64.1	0
198	7236507	POOJA TARALE	Term	28	38.2	G2P1L1A1	1	N	N	Y	N	N	N	N	N	N	N	N	11.5	0.13	11.7	4.40	35.1	79.4	26.50	33.4	14.3	1.8	8.5	192	6.5	1.3	0.6	0.1	8.8	5	2.16	147.6	0
7	1201362	MOBIN ALISAB	Preterm	23	34.4	P1	1	Y	N	N	N	N	N	N	N	Y	N	N	81.4	0.11	11.1	3.77	33.3	80.1	29.50	32.1	12	0.4	14.3	200	11.2	2.1	0.9	0.1	98	5.3	2.3	95.2	0
42	1163005	ANKITA NAGRAJ	Preterm	25	28.3	G3P2L2	2	N	N	N	N	Y	N	N	N	N	N	N	20.5	0.11	10.5	3.97	32.6	82.1	26.40	32.2	14.2	0.9	11.18	250	9.06	1.9	0.1	0.11	8.1	4.7	19	131.4	0
117	1167022	ASHA PAGAD	Term	20	39	P1	1	N	N	N	N	N	N	N	N	N	N	N	19.5	0.11	8.1	3.29	25.5	77.5	24.10	31.6	21.1	1.5	13.6	366	9.1	2.8	1.4	0.2	7.3	3.25	2	130.7	0
43	1163613	NOORJAHAN	Preterm	30	30.6	G6P4L4A1	4	Y	N	N	N	Y	Y	N	N	N	N	N	42.5	0.1	12.7	4.61	40.5	88	27.60	31.3	16.1	2.4	18.4	158	14.8	2.8	0.5	0.3	8.9	5.2	5.6	56.4	0
73	7199959	BHAGYASHREE	Preterm	28	34.2	G3P2L2	2	N	N	N	N	Y	N	N	N	N	N	N	4.1	0.1	11.2	3.56	34.9	98	31.50	32.1	13	2.5	13.33	150	10.8	1.57	0.87	0.07	11.5	6.8	1.8	95.5	0
87	7164036	POOJA PATIL	Preterm	27	30.5	G2P1L1	1	N	N	N	N	Y	N	N	N	N	N	N	11.5	0.1	10.3	3.86	33	85.5	26.70	31.2	15.1	2.04	14.25	224	10.8	2.44	0.95	0.05	10.7	4.4	2.5	91.8	0
113	1170364	ROOPA ANAND	Term	27	39.2	P1	1	N	N	Y	N	N	N	N	N	N	N	N	12.7	0.1	11.1	4.59	36.3	79.1	24.20	30.6	15.9	0.5	11.19	218	8.3	2.2	0.1	0.1	8.5	3.7	22	99	0
56	1175121	ASHWINI	Preterm	24	34	G4P1L1A1	1	N	N	N	N	N	N	N	Y	N	N	N	5.3	0.09	13.4	4.30	41.3	95.6	31.10	32.6	12.4	0.5	6.7	174	4.56	1.74	0.2	0.1	9.5	2.6	8.7	99.7	0
68	7044705	TAYAVVA	Preterm	24	30.4	G2P1L1	1	N	N	N	N	Y	N	N	N	N	N	N	19.5	0.09	9	4.84	29.1	60.1	18.60	31	16.6	4.1	13.4	209	11.2	1.3	0.8	0.1	8.2	8.6	1.6	160.7	0
104	1155690	TASBIYA ARBAZ SHAIK	Term	20	39.4	P1	1	N	N	N	N	N	N	N	N	N	N	N	12.9	0.09	9.4	4.36	29.5	67.5	21.40	31.7	14.4	0.6	16.07	248	13.9	1.9	0.2	0.1	9.6	7.3	9.5	130.5	0
110	1170731	SUDHARANI	Term	25	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	26.3	0.09	11.2	4.35	37.2	85.2	25.70	30.1	15.1	2.4	13.3	98	10.6	1.4	1.3	0	7.2	7.5	1.07	70	0
190	7145263	GEETA PATIL	Term	32	38.4	G3P2L2	2	N	N	N	N	N	N	N	N	N	N	N	2.4	0.09	10.9	3.53	32.2	91.4	30.80	33.7	13.3	1.6	7.2	169	5.9	1	0.3	0.4	6.7	5.9	3.33	169	0
99	7231638	URMILA	Preterm	23	33	P1	1	N	N	Y	N	N	N	N	N	N	N	N	31.5	0.08	9.7	2.96	30.3	102.4	32.80	32	18.6	4.1	16.94	273	14.3	1.08	1.4	0.12	9.9	13.2	0.7	252.7	0
157	1181487	MANGALA SHIVANAND	Term	31	37.6	G2P1L1	1	N	N	N	N	N	N	Y	N	Y	N	N	20.3	0.08	12.2	3.73	36.2	97.1	32.70	33.6	14.1	2.4	8.7	222	5.5	2.1	0.9	0.2	7.6	2.6	2.3	105.71	0
168	1187454	DHANESHWARI KALAKARI	Term	24	40.2	P1	1	N	N	N	N	N	N	N	Y	N	N	N	5	0.08	10.6	3.50	32.1	90.4	29.90	33.1	12.9	0.5	12.13	274	9.2	2.3	0.363	0.24	9.2	4	6.3	119.2	0
174	7018167	AHILYA	Term	24	38.6	G2P1A1	1	N	N	N	N	Y	Y	N	N	N	N	N	16	0.08	10.7	5.04	35.3	70	21.20	30.3	15.9	1.2	9.52	359	5.84	3.01	0.56	0.06	11	1.9	5.3	119.2	0
33	7006398	VIJAYSHREE	Preterm	33	27.3	P1	1	Y	N	N	N	N	N	N	N	N	N	N	100.5	0.07	11.3	3.58	37.8	105.7	31.50	29.8	12.3	0.6	8.83	190	6.05	2.19	0.1	0.1	9.4	2.75	21.9	86.7	0
158	1181547	POOJA HODDAR	Term	23	40.2	P1	1	N	N	N	N	N	N	N	N	N	N	N	0.5	0.07	12	3.94	38.4	97.4	31.90	32.8	12.7	0.5	5.9	156	4.07	0.49	0.2	0.05	8.2	8.2	2.47	315.87	0
193	7620520	SHEETAL GHODAKE	Term	24	38	P1	1	N	N	N	N	N	N	N	N	N	N	N	10.5	0.07	7.1	2.90	23.2	78.3	23.60	30.4	17.2	2.6	5.1	150	3.7	1.2	0.3	0.1	8.6	3.08	4	125	0
36	1156326	GAYTHARI SHIDLI	Preterm	25	35.3	G2P1L1	1	N	N	N	N	Y	N	N	N	N	N	N	18.6	0.06	9.6	3.40	34.6	89.6	25.60	28.6	18	3.3	16.8	272	12.9	3.2	0.4	0.2	7.4	4	6.4	85	0
53	1173834	NIKITA MANE	Preterm	22	34	P1	1	N	N	N	N	N	N	N	N	N	N	Y	0.8	0.06	11	3.96	33.5	84.6	28.30	33.4	13	0.5	13.6	265	10.2	2.31	0.3	0.2	8.5	4.4	7.7	114.5	0
64	1187771	SAVAKKA JENAKATTI	Preterm	23	33.4	P1	1	Y	N	N	N	N	N	N	N	N	N	N	6.5	0.06	10.8	3.50	34.9	89.5	28.70	32.1	15.6	2.8	13	208	10	2.1	0.6	0.3	8.6	4.7	3.5	99	0
85	7113652	SULBHA PATIL	Preterm	36	34.5	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	39.7	0.06	11.7	3.96	36.4	98.6	31.70	32.1	16.3	2.4	14.48	156	12.3	1.51	0.61	0.05	10.4	8.1	2.4	103.3	0
94	7533040	SUMAIYA	Preterm	25	31.4	G2P1L1	1	N	N	N	N	Y	N	N	N	N	N	N	102.5	0.06	10.5	3.60	33.3	90.5	28.40	31.4	14.6	2.3	14.5	261	11.3	1.9	1	0.2	8.8	5.9	1.9	137.3	0
105	1173298	ASMITA SUNIL	Term	22	40	P1	1	N	N	N	N	N	N	N	N	N	N	N	5.8	0.06	11.5	3.95	34.5	86.3	27.50	31.2	14.2	0.5	18	412	13.9	3.5	0.6	0.1	10.5	3.9	5.8	117.7	0
136	1160317	AKSHATA LAD	Term	22	39	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.5	0.06	8.8	2.79	27.3	97.8	31.50	32.2	15.8	0.9	8.57	165	5.61	2.22	0.64	0.15	11.4	2.5	3.46	74.32	0
141	1160460	ARCHANA PATIL	Term	31	40	G3P2L2	2	N	N	N	N	N	N	N	N	N	N	N	2.9	0.06	10.6	3.39	31.3	92.3	31.30	33.9	13.7	0.5	12.15	177	9.92	1.52	0.65	0.13	10.9	6.5	2.3	116.4	0
152	1172475	UJJAWALA	Term	25	36.6	G3P2L1	2	N	Y	N	N	N	N	N	N	N	N	N	5.1	0.06	12.3	4.21	38	90	29.20	32.4	14.9	1	8	289	3.8	3.6	0.3	0.3	9.5	1.05	12	80.2	0
156	1180547	SHAILA MANJUNATH	Term	26	38.1	G3P2L1	2	N	N	N	N	N	N	N	Y	N	N	N	6	0.06	10	3.84	30.7	79.9	26.00	32.5	17	2.4	16.2	260	13.6	1.5	1	0.2	7.7	9	1.5	173.3	0
163	1183353	LAXMI GODARI	Term	25	38	G2P1L1	1	N	Y	N	N	N	N	N	N	N	N	N	4.6	0.06	11.4	5.10	56.3	110	36.10	32.7	20.9	3.6	11.5	224	4.8	5.6	0.8	0.2	9.4	0.8	7	40	0
166	1186883	SUJATA	Term	21	39	P1	1	N	N	N	N	N	N	N	N	N	N	N	9.4	0.06	10.8	3.37	32.2	95.4	32.00	33.6	14.9	0.5	7.5	218	4.9	2.1	0.3	0.2	8.2	2.3	7	103.8	0
179	7093406	MAHADEVI	Term	28	38	G3P1L1A1	1	N	N	N	Y	N	N	N	N	N	N	N	5.1	0.06	8.5	3.30	27	81.8	25.80	31.5	13	1.9	10.43	230	7.1	2.3	0.6	0.03	9.1	3.08	3.8	100	0
8	1193220	PRADNYA PATIL	Preterm	21	28.6	P1	1	Y	N	N	N	N	N	N	N	N	N	N	23.3	0.05	11.5	4.76	33.1	79.5	27.90	33.4	14.2	0.6	9.2	189	6.4	1.9	0.9	0.1	8.1	3.3	2.1	99.7	0
16	1118786	RENUKA PATIL	Preterm	27	32.2	P1	1	Y	N	N	N	N	N	N	N	N	Y	N	5.9	0.05	11.8	3.65	35.4	81.5	30.10	33.5	11.5	0.6	12.5	169	9.9	1.5	0.2	0.0	10.4	6.6	7.5	112.6	0
21	1164422	SHRIDEVI S BAGEWADI	Preterm	31	31.2	P1	1	N	N	N	N	Y	N	N	N	N	N	N	3.2	0.05	13.6	5.10	40.8	83.4	28.20	31.5	15	1.5	19.1	192	16.6	1.8	0.7	0	8.6	9.2	2.5	183.8	0
25	1167067	SUMITRA PRAKASH	Preterm	30	28.5	P1	1	N	N	N	N	N	Y	N	N	N	N	N	12.3	0.05	12.8	4.42	38.4	80.5	32.50	34.1	12.5	1.5	12.1	299	8.7	2.2	0.7	0.4	10.8	3.9	3.1	135.9	0
47	1168226	AKASHATA	Preterm	25	35	G2P1L1	1	Y	N	N	N	Y	N	N	N	N	N	N	62.5	0.05	10.3	3.70	33.4	89.3	27.50	30.8	14.4	1.3	16.3	349	12	3.1	1	0.1	10.2	3.8	3.1	112.5	0
50	1170825	MEGHA PATIL	Preterm	30	33.6	P1	1	N	Y	N	N	N	N	N	N	N	N	N	6.9	0.05	11.2	3.41	33	98.6	33.00	34.1	13.9	1.9	9.9	192	7	1.8	0.7	0.4	9.6	3.8	2.5	106.6	0
54	1174518	GANGAMMA	Preterm	36	35	G3P2L1	2	Y	N	N	N	N	N	N	N	N	N	N	82.5	0.05	12	3.80	38.4	100.4	31.60	31.5	14.1	1.7	13	188	9.4	2.6	0.8	0.1	9.2	3.6	3.25	72.3	0
60	1181814	SHILPA MADAE	Preterm	20	32.1	P1	1	N	N	N	N	Y	N	N	N	N	N	N	84.2	0.05	9.8	3.38	29.2	86.6	29.00	33.5	15	2.6	12.7	283	9	2.6	0.6	0.6	7.6	3.4	4.3	108.8	0
61	1180655	NIKITA CHALLAGIDAD	Preterm	22	30.3	P1	1	N	N	Y	N	N	N	N	N	N	N	N	34.9	0.05	10.5	4.09	33.5	81.9	25.70	31.4	16.3	1.5	13.3	324	10.3	2.2	0.6	0.1	9.4	4.6	3.6	147.2	0
63	1186196	BHARTI GUNDAKALLA	Preterm	35	34.2	P1	1	Y	N	N	N	N	N	N	N	N	N	N	25	0.05	12.5	3.96	35.8	90.3	31.40	34.8	12.8	1.4	14.3	361	11.3	2	0.8	0.1	7.7	5.6	2.5	180.5	0
66	7131057	TABSSUM	Preterm	26	28.2	G2P1L1	1	N	N	N	N	Y	N	N	N	N	N	N	15.6	0.05	10.6	3.32	34.4	103	31.90	30.8	16.4	5.14	8.83	41	6.75	1.1	0.73	0.3	9.5	6.1	36.6	37.2	0
79	723495	SAVITA	Preterm	29	29	P1	1	Y	N	N	N	N	N	N	N	N	N	N	24.8	0.05	8.2	3.73	26.7	71.5	21.90	30.6	15	2.1	14.3	214	10.3	2.6	1.1	0.3	8.5	3.9	2.3	82.3	0
81	7249647	NOOR	Preterm	25	34.2	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	77.5	0.05	8.9	3.25	27.5	84.7	27.40	32.3	15	0.6	13.2	186	11.2	0.8	1	0.1	72	14	0.8	232.5	0
92	7535370	NIKITA	Preterm	25	31.4	P1	1	N	N	N	N	N	N	N	N	N	N	Y	10.5	0.05	11.5	4.27	36	84.3	26.90	33.9	13.3	1.5	13	277	9.55	2.58	0.82	0.02	11.2	3.7	3.1	107.3	0
107	1173170	SAVITHA AYADI	Term	28	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.3	0.05	13.2	4.67	39.6	90.2	29.30	31.4	14.5	0.5	22.2	229	20.1	1.9	0.2	0	9.5	10.5	9.5	120.5	0
111	1171445	ROOPA BASAVRAJ	Term	22	40	G2P1L1	1	N	Y	N	N	N	N	N	N	N	N	N	40.8	0.05	10.7	3.59	33.3	93	29.80	32	16.2	3.3	13.2	244	10.6	1.5	0.8	0.3	7.2	7	1.875	162.6	0
119	1168405	BHARTI PATIL	Term	23	39.3	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	0.6	0.05	12.5	4.00	38.1	88.2	27.30	31.2	13.4	0.5	13.09	242	10.2	1.7	0.1	0.28	10	6	17	142.3	0
130	1202987	SONALI VIJAY	Term	29	38.3	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	3.2	0.05	12	4.02	36.1	82.1	28.10	30.5	15.1	0.8	9	248	5.8	2.2	0.2	0.4	8.5	2.6	11	112.7	0
132	1186330	BHARATI SHIVANAND	Term	32	37	G3P1L1A1	1	N	N	Y	N	N	N	N	N	N	N	N	4.1	0.05	11	5.17	36.1	69.8	21.30	30.5	14.2	0.5	10.6	289	6.6	2.7	0.8	0.3	8.5	2.4	3.3	107	0
139	1160495	PREVEENA NADAF	Term	32	37.2	G3P2L2	2	N	N	N	N	N	N	N	N	N	N	N	1.5	0.05	10.7	4.45	36.4	81.8	24.00	29.4	24.8	0.7	15.1	271	11	3.12	0.82	0.15	10.1	3.5	3.8	86.8	0
154	1179162	SAKKUBAJI	Term	32	38.2	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	10.6	0.05	12	4.50	36.9	81.9	26.50	32.4	13	1.5	10.5	256	7.5	1.6	0.6	3	9.6	4.6	2.6	160	0
155	1179184	CHANDRAKALA AGASAL	Term	32	39	G2P2L1	2	N	N	N	N	N	N	N	N	N	N	N	3.2	0.05	13	4.30	38.5	95.2	33.00	32.5	12	0.5	10.4	185	7.5	2.3	0.5	0.1	8.6	3.2	4.6	80.43	0
165	1185754	MEGHA UPATIMATH	Term	22	37.2	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	2	0.05	11.4	3.49	34.5	93	30.70	33	13.7	0.5	8.8	197	6.4	1.8	0.6	0.1	8.2	3.5	3	109.4	0
187	7233575	KEERTI GUNJAN	Term	31	37.1	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	5.7	0.05	12.2	4.17	39.9	83.9	25.80	30.9	16	1.2	8.17	252	4.29	2.61	0.65	0.5	12.4	1.64	4.01	96.5	0
191	7236503	SURAIYA	Term	23	37	G2P1L1	1	N	N	Y	N	N	N	N	N	N	N	N	3.5	0.05	10.2	4.60	33.4	72	21.90	30.5	19	1.3	5.6	150	3.6	1.1	0.5	0.2	10.4	3.2	2.2	136.3	0
9	1193783	LAXMI YALLAPPA	Preterm	28	28.4	G2P1L1	1	N	N	N	N	N	N	N	N	Y	N	N	1.9	0.04	8.7	2.62	26.1	77.5	22.50	27.2	16.5	1.5	5.7	126	4.7	0.6	0.1	0.1	10.6	7.8	6	210	0

31	1189160	AMNBIKA HOSAMANI	Preterm	22	30.3	P1	1	N	Y	N	N	N	N	N	N	N	N	N	3.8	0.04	13	4.12	36	83	27.00	31.5	15	0.5	9.7	288	6.7	1.8	0.5	0.7	9.2	3.7	3.6	160	0
38	1160595	SUMAYYA MAKANDAU	Preterm	20	35.2	P1	1	Y	N	N	N	N	N	N	N	N	N	N	42.8	0.04	11.3	3.39	33.5	98.7	33.40	33.8	12.2	0.5	13.95	197	10.4	3.6	0.1	0.15	10.4	2.8	36	54.7	0
46	1168139	POOJA	Preterm	22	35	P1	1	N	N	Y	N	N	N	N	N	N	N	N	55.6	0.04	10.5	3.23	33	102	32.50	31.8	14.1	0.6	8	209	5.8	1.3	0.6	0.3	8.5	4.4	2.1	160.7	0
51	1173200	KAVERI	Preterm	25	35	P1	1	N	N	N	N	N	N	N	Y	N	N	N	7.9	0.04	12	4.13	38.5	93.2	29.30	31.4	17	0.5	12.27	226	9.2	2.82	0.1	0.12	10	3.2	28.2	80	0
58	1179884	AISHWARAYA	Preterm	30	29.5	P1	1	Y	N	N	N	N	N	N	N	N	N	N	43.1	0.04	12.2	4.11	36	95.5	30.50	32.1	13.2	0.5	105	322	5.9	3.4	0.6	0.6	9.6	1.7	5.6	94.7	0
59	1179652	RAJASHRI KAVERI	Preterm	28	33.3	G3P2L2	2	Y	N	N	N	N	N	N	N	N	N	N	63.7	0.04	10.5	4.03	33.6	83.4	26.00	31.3	17.3	0.09	10.81	261	8.65	1.6	0.1	0.1	7.5	5.4	16	163.1	0
72	7211789	SHAHEEN	Preterm	25	35	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	5.6	0.04	11	3.58	35	97.8	30.70	31.4	12.9	1.8	11.85	297	8.12	2.48	1.1	0.06	9.5	3.2	2.2	119.7	0
80	7249550	SHRIDEVI	Preterm	27	33.1	G2P1L1	1	N	Y	N	N	N	N	N	N	N	N	N	10.9	0.04	10.7	3.73	33.5	90	28.80	32	13	1.3	10.7	178	8.3	1.6	0.7	0.2	7.7	5.1	2.2	111.2	0
82	6963592	PAKIJA IELAAI	Preterm	27	33	P1	1	N	Y	Y	N	N	N	N	N	N	N	N	15	0.04	9.1	4.18	30	71.8	21.80	30.3	19.5	1.47	11.7	244	8.88	2.43	0.35	0.01	11.5	3.6	6.9	100.4	0
83	7145263	GEETA PATIL	Preterm	24	34.2	P1	1	Y	N	N	N	N	N	N	N	N	N	N	13.2	0.04	8.8	2.82	26.5	94	31.20	33.2	13.2	3	13.09	234	11.5	1.18	0.35	0.03	8.7	9.7	3.3	198.3	1
93	7197970	BHAVANI	Preterm	27	31	P1	1	N	N	Y	N	N	N	N	N	N	N	N	19.8	0.04	11.6	4.36	39.7	91.1	26.50	29.1	18.4	1.6	17.6	170	14.3	1.2	1.6	0.4	11.5	11.9	0.75	141.6	1
101	1160296	POOJA KAMBLE	Term	27	40.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	4.6	0.04	13.5	4.20	38.9	92.4	32.10	34.7	13.7	0.7	10.08	156	7.2	2.2	0.3	0.3	8.5	3.2	7.3	70.9	1
106	1173265	SALIKA SAJEED	Term	33	38.3	G4P2L2A1	2	N	N	N	N	Y	Y	N	N	N	N	N	30.1	0.04	9.9	3.36	29.7	77.6	21.40	26.5	15.3	1	10	214	8.1	1.5	0.4	0	8.5	5.4	3.75	142.6	1
108	1172676	PADMA KOLE	Term	30	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	7.8	0.04	14.4	4.85	43.2	85.2	27.60	32.5	14.9	0.5	11.3	180	8.4	2.2	0.6	0.1	8.2	3.8	3.6	81.8	1
123	1166827	POOJA VISHWANATH	Term	22	37.2	G3A2	3	N	N	N	N	Y	Y	N	N	N	N	N	3.7	0.04	9.9	3.56	31.5	88.6	27.80	31.3	17.2	1.7	10.8	229	6.5	3.2	0.7	0.4	9.7	2	4.5	71.5	1
127	1202986	ASHWINI GUNJANAL	Term	26	39.3	P1	1	N	N	N	N	N	N	N	N	N	N	N	4.2	0.04	13.1	4.11	43.3	100.1	31.90	30.3	12.3	0.5	10.17	274	7.9	1.6	0.4	0.2	9.5	4.9	4	171.25	1
129	1203138	SUNANDA MANGOLI	Term	24	37.6	P1	1	N	N	N	N	N	N	N	N	N	N	N	4.6	0.04	11.5	4.10	33.1	79.1	28.90	33.1	12.5	0.5	10.8	153	7	2.7	0.4	0.5	7.5	2.5	6.75	56.6	1
140	1160064	SAKUBAI NINGAPPA	Term	22	37.3	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.5	0.04	8.8	3.00	28.3	94.3	29.30	31.1	16.7	0.5	8.5	115	6.27	1.83	0.32	0.1	9.9	3.4	5.7	62.84	1
149	1168024	AKSHATA	Term	25	39.5	P1	1	N	N	N	N	N	N	N	N	N	N	N	0.9	0.04	12	3.70	37.3	100	32.80	32.7	14.2	0.5	13	206	9.5	2.4	0.9	0.1	10.5	3.9	2.66	85.83	1
151	1167238	SUSHMA MALLAPPA	Term	27	36.1	G3P1L1A1	1	N	N	Y	N	N	N	N	N	N	N	N	2.5	0.04	11.7	4.39	35.5	80.8	26.70	33	14.3	1.7	11	237	8	2.1	0.5	0.5	10.5	3.8	4.2	112.8	1
164	118767	SHEELA	Term	27	38.2	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.9	0.04	11.2	3.59	35.2	98.1	31.10	31.7	13.1	0.5	12.1	204	8.23	3.03	0.3	0.1	8.4	2.7	10.08	67.43	1
171	7136465	LIPIKA	Term	22	37.2	P1	1	N	N	N	N	N	N	N	N	N	N	N	9.9	0.04	5.4	3.84	21.5	56	14.10	25.1	23	2.3	7.14	330	4.37	2.11	0.58	0.08	9.2	2.07	3.6	156.39	1
176	70457570	MARISSA	Term	20	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	8.2	0.04	9.6	4.28	31.3	73	22.40	30.7	15.2	0.8	8.69	350	5.06	2.9	0.54	0.17	10.6	1.7	5.3	120.6	1
180	4486044	RUDRAVVA	Term	23	37	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	4.2	0.04	7.1	2.70	24.9	92.2	26.30	28.5	23	2.69	3.66	180	2.27	1.09	0.22	0.07	8.9	2.08	4.9	165.1	1
186	7904811	BHAGYASHREE KOLADUR	Term	27	39.5	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	4.1	0.04	11.3	4.18	35.9	86	27.10	31.5	16.4	1.02	6.7	242	4.3	2.1	0.2	0.1	8.4	2.04	10.5	115.2	1
188	7178943	MADHAVI KOTAGI	Term	36	38	G4P1L1A2	2	N	N	N	N	N	N	N	N	N	N	N	2.4	0.04	10.1	4.17	31.8	76.3	24.20	31.8	14.6	0.87	10.17	467	7.27	1.94	0.47	0.45	10.4	3.74	4.12	240.7	1
189	7443840	PPORNIMA	Term	22	39	P1	1	N	N	N	N	N	N	N	N	N	N	N	6.4	0.04	10	4.36	32.1	73.6	22.90	31	18	2.14	8.6	159	6	2	0.5	0.1	9.2	3	4	79.5	1
197	7030663	POOJA MALAJE	Term	23	40.1	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	7.6	0.04	10.3	3.74	32.6	87.1	27.40	31.5	12.7	1.2	7.4	203	4.3	2.6	0.4	0.1	10.7	1.65	6.5	78.07	1
3	1205531	POONAM SATISH	Preterm	28	28.6	P1	1	Y	N	N	N	N	N	N	N	N	N	N	1.5	0.03	10.8	3.49	32.4	79.2	27.20	33	13.5	0.6	12.6	134	9.4	2	1.1	0.3	11.2	4.7	1.8	67	1
10	1195293	KASTURI KADAPPA	Preterm	28	30	G2P1L1	1	Y	N	Y	N	N	N	N	N	N	N	N	29.5	0.03	11.1	3.75	33.3	90.2	27.80	32.5	14	0.4	14.7	413	11.8	2	0.7	0.1	11.4	5.9	2.8	206.5	1
11	1197235	NEETA VINAYAK	Preterm	22	32.6	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	22.6	0.03	11.2	3.68	33.5	90.1	28.10	30.8	15	0.5	10.14	239	7.0	2.8	0.7	0.1	10.1	2.5	4	85.3	1
19	1162490	PARVATI	Preterm	31	31	G3P2L1	2	N	N	N	N	Y	N	N	N	N	N	N	41.5	0.03	12.9	4.23	38.7	80.9	26.50	31.5	14.3	1.2	15.1	191	13	1.5	0.5	0	9.4	8.6	3	127.3	1
30	1168913	SAVITA	Preterm	25	34.5	P1	1	N	N	N	N	Y	N	N	N	N	N	N	7.7	0.03	12.4	3.50	35.6	101.1	35.40	35	12.2	0.1	11.9	163	9.6	1.6	0.2	0.1	10.6	6	8	101.8	1
41	1161428	GEETA HIREMATH	Preterm	29	33.3	P1	1	Y	N	N	N	N	N	N	N	N	N	N	36.4	0.03	10.7	3.47	34.6	99.7	30.80	30.9	13.8	0.5	14.38	211	11.4	2.08	0.71	0.15	10.6	5.4	2.9	101.4	1
57	1175060	SIDAVVA RAJAHATI	Preterm	21	35.1	P1	1	N	Y	N	N	N	N	N	N	N	N	N	3.5	0.03	13.3	4.35	40.1	92.1	30.50	33.1	14.2	0.5	16.17	206	12.3	2.91	0.2	0.32	8.5	4.2	14.5	70.7	1
70	7232531	GIRIJA	Preterm	22	29.1	P1	1	Y	N	N	N	N	N	N	N	N	N	N	100.7	0.03	8.9	3.73	29.7	79.6	23.90	30	21.1	3.9	12.73	265	9.85	1.94	0.72	0.12	9.8	5	2.6	136.5	1
74	7210394	RUPA	Preterm	29	29.6	G3P2L2	2	N	N	N	N	N	N	N	N	N	N	Y	17.5	0.03	10.3	3.30	31.4	95.1	31.20	32.8	14	2.9	11.4	205	8.9	1.7	0.7	0.1	8.5	5.2	2.4	120.4	1
77	7256929	KAVERI	Preterm	33	32.1	G4P1L1A1	1	N	N	N	N	Y	N	N	N	N	N	N	35.4	0.03	10.7	3.58	32.5	90.9	30.00	33	14.7	1.64	17.3	191	14.3	1.8	1	0.1	10	7.9	1.8	106.1	1
118	1168028	SUJATA	Term	20	39.5	P1	1	N	Y	N	N	N	N	N	N	N	N	N	4.8	0.03	12.3	4.18	37.9	90.8	29.50	32.5	14.6	2.1	12.4	97	9.3	2	0.9	0.2	10.9	4.65	2.2	48.5	1
122	1167455	ANITA GANPATI	Term	19	38.4	P1	1	N	N	N	N	N	N	N	N	Y	N	N	4.4	0.03	11.8	3.67	35.8	97.6	21.10	32.9	13.5	0.5	17.27	254	12.9	3.4	0.6	0.18	9.8	3.7	5.6	74.7	1
128	1203122	NAGRATNA BASAVRAJ	Term	21	37.3	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.5	0.03	10.8	3.61	31.5	86.1	27.30	32.4	14.1	0.6	8.8	165	5.7	2.2	0.4	0.4	7.5	2.5	5.5	75	1
131	1189825	NEETA PATIL	Term	23	37.3	P1	1	N	N	N	N	N	N	N	N	N	N	N	5	0.03	9.9	4.05	32.5	79	27.00	31	15	0.6	11.36	213	8.5	2	0.5	0.2	9.6	4.25	4	106.5	1
137	1160573	PREETI LOTTAR	Term	21	38.5	P1	1	N	N	N	N	N	N	N	N	N	N	N	3.1	0.03	11.2	3.73	36.1	96.8	30.00	31	15.3	0.6	15.36	349	11.7	2.64	0.94	0.15	9.2	4.4	2.8	132.19	1
160	1182548	SONA BAI	Term	19	39.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.6	0.03	12.8	4.06	38.2	94.1	31.50	33.5	16.3	2.6	10	304	5.3	3.3	0.8	0.6	10.2	1.6	4.1	92.12	1
173	7132304	GAURAVVA	Term	21	39	P1	1	N	N	N	N	N	N	N	N	Y	N	N	4.8	0.03	9.4	5.10	32.5	63.4	18.30	28.9	17.5	1.2	7.44	354	3.68	3.01	0.43	0.25	9.7	1.2	7	117.6	1
177	7081420	RUPA	Term	27	37.6	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	3.5	0.03	9.2	3.50	30.2	86.3	26.30	30.5	20.6	1.7	6.2	200	4.36	1.27	0.46	0.08	10.6	3.4	2.7	157.4	1
17	1125475	SNEHAL SHIPULKAR	Preterm	20	30.2	P1	1	Y	N	N	N	N	N	N	N	N	N	N	62.5	0.02	13.8	4.41	41.4	90.5	32.10	33.4	14.8	0.6	23.6	180	19.9	4.7	0.7	0.4	11.9	28.4	6.7	38.2	1
20	1162793	PRACHI B JOGANI	Preterm	33	32.6	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	3.7	0.02	14	4.55	40.1	82.8	27.00	31.6	16	1.1	12.8	230	9.1	2.7	0.8	0.1	8.2	3.3	3.37	85.1	1
23	1166981	SHAINAZ R	Preterm	23	31.5	G3P2L1	2	Y	N	N	N	N	N	N	N	N	N	N	39.4	0.02	10.3	3.29	30.9	100.2	32.10	31.9	13.4	2.1	11.3	300	6	4.5	0.7	0.1	9.3	1.3	6.4	66.6	1
27	1170065	NAMEETA RAO	Preterm	38	35	G3P1L1A1	1	N	N	N	N	Y	N	N	N	N	N	N	2.5	0.02	12.3	4.30	36.2	88.5	29.40	32.3	15	0.6	12.4	249	9.1	2.1	0.8	0.4	8.1	4.3	2.6	118.5	1
28	1169413	NAGAVVA PATIL	Preterm	31	33.4	G2P1L1	1	Y	N	N	N	N	N	N	N	N	N	N	36.5	0.02	10.5	4.99	31.2	79.1	25.60	30.1	14	0.5	10.2	186	8	1.4	0.7	0.1	11.4	5.7	2	132.8	1
37	1159842	BIBIAYESHA PATHAN	Preterm	22	35.1	P1	1	N	Y	N	N	N	N	N	N	N	N	N	15.6	0.02	118	3.85	36.8	95.6	30.60	32.1	11.9	0.6	10.9	312	7.6	2.3	0.8	0.2	11	3.3	2.8	135.6	1
40	1160055	RASIKA RAVI	Preterm	28	32.1	G2P1L1	1	N	Y	N	N	N	N	N	N	N	N	N	26.3	0.02	12	3.86	34	80.2	24.30	30.3	14	0.5	9.5	212	6.8	2.2	0.5	0.1	8.1	3	4.4	96.3	1
48	1168938	RENUKA	Preterm	32	32	G3P1L1A1	1	Y	N	N	N	N	N	N	N	N	N	N	65.8	0.02	14.2	4.80	44.2	91.7	29.70	32.2	13.8	1.7	20.5	172	19.3	0.8	0.3	0.1	8.2	34.1	2.6	215	1
49	1168368	RUKMANI	Preterm	25	29.1	G3P2L0	2	N	N	Y	N	N	N	N	N	N	N	N	3.1	0.02	12	3.70	36.8	58.1	33.30	33.8	12.6	0.5	9	150	6.8	1.3	0.6	0.3	10.5	5.2	2.1	115.3	1
52	1173487	SUPRIYA	Preterm	26	35	P1	1	N	N	N	N	N	N	N	N	N	Y	N	1.8	0.02	9.2	3.39	27.6	84	27.00	33	13	0.5	14.36	272	10.9	2.87	0.3	0.14	8.5	3.7	9.5	94.7	1
62	1185032	RUKMA KADAKAL	Preterm	20	33.3	P1	1	N	Y	N	N	N	N	N	N	N	N	N	16.4	0.02	10.6	3.40	35.7	93.5	31.20	33.3	12.7	1.5	21.9	246	18.1	2.6	1.2	0.1	9.4	6.9	2.1	94.6	1
67	4983292	CHANNABASSVVA	Preterm	29	27.3	G3P2L2	2	Y	N	N	N	N	N	N	N	N	N	N	56.4	0.02	10.9	3.84	34.2	89.1	28.40	31.9	19.2	1.01	5.82	295	4.16	1.17	0.17	0.31	9.8	3.5	6.8	252.1	1
100	7224783	SHRISHTI	Preterm	25	32.1	G2P1L1	1	N	N	N	N	N	N	N	Y	N	N	N	20	0.02	11.8	4.26	37.5	88	27.70	31.5	15.1	2.3	12.34	274	9.83	1.31	0.94	0.26	10.5	7.5	1.3	209.1	1
102	1156855	SANGEETHA DASHRATH	Term	30	37.1	G4P3L3	3	N	N	N	N	N	N	N	N	N	N	N	16.9	0.02	9.2	4.36	33.9	77.8	21.20	27.1	25.3	0.5	11.4	460	8.6	2.5	0.5	0.5	10.5	3.4	5	184	1
112	1170812	ASHWINI PATIL	Term	28	37	G2P1L1	1	N	N	N	N	N	N	Y	N	N	N	N	5	0.02	11.8	4.29	36.7	85	27.60	32.3	16.4	1.9	17.2	249	13.5	2.3	1	0.3	7.7	5.8	2.3	108.2	1
114	1169839	AISHWARYA MISAL	Term	23	39.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	19.5	0.02	11.5	3.93	34.5	90.3	28.50	32.4	13.5	0.5	13.4	140	10.9	1.5	0.1	0.1	7.5	7.2	15	93.3	1
116	1168650	MADHURA KUMAR	Term	20	37.5	P1	1	N	N	N	N	N	N	N	Y	N	N	N	4.8	0.02	12.3	4.39	36.9	88.1	25.70	30	14	0.5	9	95	5.9	2.4	0.5	0.1	6.5	2.4	4.8	39.5	1
121	1167320	HEENAKOUSAR HALANSAE	Term	27	38.4	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	2	0.02	6.4	3.15	21.1	67.1	20.30	30.2	17.3	2.4	19.1	196	16.6	1.8	0.7	0	9	9.2	2.5	108.8	1
124	1167844	KEERTHI SUNIL	Term	20	37.6	P1	1	N	N	N	N	N	N	N	N	N	N	N	4	0.02	10.9	3.96	31.6	88	29.00	32.5	14.2	0.5	15.5	444	10.7	3.6	0.9	0.3	9.5	2.9	4	123.3	1
126	1202329	KRANTI PATIL	Term	25	38.1	G2A1	2	Y	N	N	N	N	N	N	N	N	N	N	2.5	0.02	11.7	3.93	34.1	80.2	27.50	32.1	15	0.8	8.3	271	5.3	2.1	0.6	0.3	9.4	2.5	3.5	129	1
134	1153455	LAXMI	Term	29	40.3	G4P3L1	3	Y	N	N	N	N	N	N	N	N	N	N	2.5	0.02	8.3	3.31	26.3	79.4	25.20	31.7	16.1	2.4	9.6	150	7.1	1.7	0.6	0.2	9.2	4.16	2.83	88.23	1
135	1160038	SONALI BALU	Term	22	37.5	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.5	0.02	9.4	3.88	32.1	82.7	24.20	29.3	155	0.7	5.93	269	4.7	0.72	0.38	0.13	9.2	6.5	1.89	373.6	1
138	1160483	BHAGYASHREE PATIL	Term	23	37.1	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	2	0.02	12.3	4.10	384	93.7	30.00	32	14.2	0.5	11.09	219	8.79	1.36	0.72	0.1	10.8	6.4	1.88	161.02	1
142	1160488	SUCHITA KADDOR	Term	30	40	P1	1	N	N	N	N	N	N	N	N	N	N	N	5	0.02	10.2	3.24	32.3	99.7	31.50	31.6	14	0.5	16.4	152	13.8	1.52	0.97	0.49	10.6	9.07	1.56	100	1
144	1161026	VIDYA	Term	29	39.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	5.1	0.02	10.1	3.09	32.9	106	32.70	30.7	16	0.7	8.5	166	6.2	1.8	0.32	0.1	11.2	3.4	5.6	92.22	1
145	1161200	RESHMA SAVAPERI	Term	28	38	G3P2L2	2	N	N	Y	N	N	N	N	N	N	N	N	0.6	0.02	11.5	3.80	36.5	95.3	30.00	31.5	14.8	0.5	10	223	7.1	2.56	0.4	0.5	10.6	2.7	6.4	87.1	1
148	1180160	VANITA NAGRAJ	Term	25	38.4	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	1.7	0.02	13	4.68	40.1	85.7	28.50	33.2	14.2	2	10	159	7	2.5	0.5	0.5	9.5	2.8	5	63.6	1
150	1168430	RUDRAMMA	Term	20	38.4	P1	1	N	Y	N	N	N	N	N	N	N	N	N	1.9	0.02	12.7	3.90	38.7	98.6	32.30	32.8	16.1	0.5	8.15	241	5.71	1.6	0.5	0.41	10.5	3.5	3.2	150.6	1
153	1178010	ANUSHREE	Term	29	39	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.9	0.02	9.7	4.59	33.1	72.1	21.10	29.3	16.2	3.4	9.5	175	6.5	2.5	0.6	0.1	8.5	2.6	4.1	70	1
162	1183013	ASHWINI BASAVRAJ	Term	24	39.6	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	5.9	0.02	11.3	4.24	37.1	87.5	26.70	30.5	13.1	1	6.2	264	3	2.5	0.4	0.3	8.8	1.2	6.2	105.6	1
169	1187691	PRAGATI PATIL	Term	22	40	P1	1	N	N	N	N	N	N	N	N	N	N	N	4.9	0.02	12.5	4.07	39.9	89.5	33.60	32.5	15	0.5	8.6	233	5.59	2.58	0.2	0.1	8.1	2.1	12.9	90.27	1
170	4081939	ANNPURNA	Term	30	39.5	G2P1L1	1	N	N	Y	N	N	N	N	N	N	N	N	6.5	0.02	9.8	4.00	33	82.5	24.50	29.7	16.2	1.53	6.93	399	3.86	2.36	0.56	0.13	8.9	1.6	4.2	169.23	1
172	7136857	KAMLA	Term	18	37.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	5.6	0.02	7.7	3.59	25.7	71.6	21.40	30	20	0.7	8.8	317	4.83	2.73	0.93	0.25	11	1.7	2.9	115.97	1
175	6304799	PRANALI	Term	22	38.3	P1	1	N	N	N	N	N	N	N	N	N	N	N	8.7	0.02	8.4	4.75	30.3	63.8	17.70	27.7	19.9	1.2	5.7	441	2.58	2.31	0.48	0.3	9.9	1.1	4.8	190.9	1
185	7185632	SABIYA BADRISHA	Term	23	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.6	0.02	10.7	3.62	33.6	92.8	29.60	32.2	12.4	1.53	11.42	234	6.99	3.4	0.77	0.26	9.7	2.02	4.41	68.8	1
194	7178651	PADMA BHIMASI	Term	23	38.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	6.4	0.02	9.5	3.60	32.5	90	26.30	29.2	20	2.4	6.34	211	3.7	1.92	0.63	0.04	10.7	1.92	3.04	109.8	1
199	7236503	NAZREEN	Term	32	38.5	G3P2LID1	2	N	N	N	N	N	N	N	N	N	N	N	10.1	0.02	10.4	4.60	32.9	70.3	22.30	31.8	19	1.3	6.2	153	4.1	1.4	0.5	0.2	13.2	2.9	2.8	109.2	1
29	1169923	HEENA PATIL	Preterm	22	34.3	G2P1A1	1	Y	N	N	N	N	N	N	N	N	N	N	41.3	0.01	11.2	3.67	35.8	97.6	21.10	32.9	13.5	0.5	19.1	196	16.6	1.8	0.7	0	10.5	9.2	2.5	108.8	1
65	6738232	SUGANDHA	Preterm	25	30.5	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	Y	125	0.01	9.2	3.00	29	96.7	30.70	31	17.1	1.95	13.26	155	11.5	1.31	0.35	0.01	11	8.7	3.74	118.3	1
75	7504871	CHANDRA	Preterm	24	35	P1	1	N	N	N	N	N	N	N	N	Y	N	N	14.6	0.01	11.5	4.60	36	78.2	25.00	32	16.5	2.5	15	364	13.5	0.9	0.5	0.1	8.8	15	1.8	404.4	1
84	7241701	KASTURI SHINGA	Preterm	22	32	P1	1	Y	N	N	N	N	N	N	N	N	N	N	55.4	0.01	8.9	3.52	30.7	87.2	25.30	29	20.1	1.25	17.1	334	15.2	1.41	0.52	0.02	9.4	10.7	2.7	237.1	1
86	7118785	AKANKSHA	Preterm	23	31.6	P1	1	N	Y	N	N	N	N	N	N	N	N	N	17.9	0.01	10.7	4.04	35.1	86.9	26.50	30.5	16	1.36	13.03	311	10.3	2.13	0.55	0.07	9.9	4.8	3.8	145.5	1
95	7095352	SUMA BADIGER	Preterm	22	34	P1	1	Y	N	N	N	N	N	N	N	N	N	N	56.4	0.01	10.6	3.96	33.2	83.9	26.90	32.1	17.3	1.8	15.9	202	14.6	0.8	0.4	0.2	8.5	18.25	2	252.5	1
96	7236512	LAXMI BAGEWADI	Preterm	23	33.1	P1	1	Y	N	N	N	N	N	N	N	N	N	N	15.7	0.01	11.2	4.20	35.3	83.4	26.50	31.8	14.8	1.7	13.8	273	9.9	2.4	1.4	0.1	7.5	4.1	1.7	113.7	1
115	1169829	NETHRA PATIL	Term	27	40.4	P1	1	N	N	N	N	N	N	N	N	N	N	N	11.1	0.01	10	3.75	30.2	85.2	27.60	31.8	12.6	0.5	24.3	247	21.5	2	0.8	0	8	10.75	2.5	123.5	1
120	1164708	SUJATHA PUJERI	Term	24	39.3	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	2.5	0.01	13.1	4.56	41.4	90.8	28.70	31.6	14.6	1.2	11.5	161	9.2	1.6	0.1	0.1	10.2	5.75	16	100.6	1
125	116724	SUSHMA SIDDHANGOUDA	Term	22	37.2	P1	1	N	N	N	N	N	N	N	N	N	N	N	2.1	0.01	12.1	4.19	37.5	90	30.00	32.5	12.3	0.5	10.2	186	7.1	2.5	0.5	0.5	8.5	2.84	5	74.4	1
133	1160010	GIRIJA HANAMANT	Term	32	37.5	P1	1	N	N	N	N	N	N	Y	N	N	N	N	0.43	0.01	13.9	4.80	42.8	89.2	29.00	32.5	15	0.5	20.9	272	19.5	0.69	0.65	0.2	9.1	28.2	1.06	394.2	1

143	1160563	ALFIYA ASIF	Term	21	39	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	3.6	0.01	11.7	4.15	38.2	92	28.20	30.6	13	0.5	10	238	7	2.5	0.4	0.5	9.8	2.8	6.25	95.2	1
146	1161368	DHANASHREE VITTAL	Term	23	37.2	P1	1	N	Y	N	N	N	N	N	N	N	N	N	2.9	0.01	10.1	3.86	33	85.5	26.20	30.6	19.3	0.6	12	184	9.47	1.8	0.7	0.13	10.5	5.2	2.5	102.2	1
147	1167820	MEGHA	Term	23	40	G2A1	2	N	N	N	N	N	N	N	N	N	N	N	3.2	0.01	12.5	3.80	37.5	96.7	32.20	33.3	16.4	1.1	16	333	11.8	3	0.9	0.3	8.3	3.9	3.33	111	1
159	1181856	SAVITA IRRANNA	Term	25	38.3	G2P2L2	2	N	N	N	N	N	N	N	N	N	N	N	5.6	0.01	11.9	4.01	36.1	89.8	29.60	32.9	12.8	0.5	12.7	286	10.2	1.91	0.381	0.25	7.7	5.3	5	150.5	1
161	1181931	SHOBHA CHOUGALE	Term	28	35	G3P2L2	2	N	N	N	N	N	N	N	N	N	N	N	6.5	0.01	12.4	3.64	36.5	100	34.20	34.1	11.6	0.5	6.94	205	4.07	2.42	0.345	0.07	7.7	1.6	6.9	84.9	1
167	1187421	MALAPRABHA	Term	25	39.5	G2P1L1	1	N	N	Y	N	N	N	N	N	N	N	N	7.6	0.01	11.9	4.19	37.4	89.4	28.50	31.9	13	0.5	10	232	7.4	2.2	0.2	0.2	8.2	3.3	11	105.4	1
178	7129591	SHILPA	Term	19	37.1	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.6	0.01	8.9	3.40	30.2	88.8	26.20	29.5	16.6	3.2	8.41	500	5.44	2.44	0.35	0.15	9.4	2.2	6.9	204.9	1
181	7249312	RATNAVVA	Term	27	37.1	G2P1A1	1	N	N	N	N	N	N	N	N	N	N	N	3.7	0.01	11.8	4.53	37	81.8	26.10	31.9	21.1	0.5	6.5	209	3.7	2.1	0.4	0.2	9	1.76	5.2	99.5	1
182	7292541	RAVEENA	Term	26	37	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	1.9	0.01	11.8	4.17	35.4	84.9	28.30	33.3	26	1.9	4.3	293	2.4	1.3	0.5	0.2	9.4	1.84	2.6	225.3	1
183	7412589	HEENA	Term	25	38.5	P1	1	N	N	N	N	N	N	N	N	N	Y	N	8.2	0.01	11.2	4.13	34.5	83.6	27.10	32.4	14	1.9	7.6	307	4.8	1.9	0.7	0.2	8.8	2.52	2.7	161.5	1
184	7504412	SUREKHA	Term	28	39.6	G2P1L1	1	N	N	N	N	N	N	N	N	N	N	N	7.1	0.01	8.9	3.09	26.6	86.1	28.70	33.3	15	3	9.8	241	7	2	0.5	0.2	7.5	3.5	4	120.5	1
192	7231474	SHILPA SANTI	Term	21	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	9.6	0.01	11.6	4.30	39.1	89.8	26.70	29.7	15.9	1.7	9.3	364	6.5	1.5	0.8	0.5	7.7	4.3	1.8	242.6	1
195	7229959	RANI SAGAR	Term	25	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	3.5	0.01	10.6	4.39	35.9	81.8	24.10	29.5	14.6	2.6	8.6	233	5.96	1.97	0.56	0.08	8.5	3.02	3.5	118.2	1
196	7235490	RENUKA GUNDANI	Term	30	39	G2P2L1	2	N	N	N	N	N	N	N	N	N	N	N	1.65	0.01	7.7	4.40	26.4	59.1	17.30	29.3	18.1	1.1	5.7	302	2.9	2.1	0.5	0.2	8.2	1.38	4.2	143.8	1
200	7196014	MALAPRAVA	Term	25	37	P1	1	N	N	N	N	N	N	N	N	N	N	N	1.3	0.01	9.8	3.46	31.7	91.6	28.30	30.9	12.4	1.04	6.45	306	5.63	0.58	0.2	0.04	9.7	9.7	2.9	567.5	1