
**“MATERNAL AND NEONATAL FACTORS
INFLUENCING 17-HYDROXY PROGESTERONE
LEVELS IN NEWBORNS DELIVERED AT A
TERTIARY CARE HOSPITAL”**

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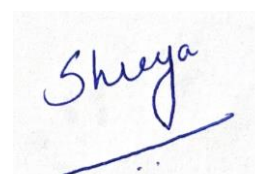
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LIST OF ABBREVIATIONS

17-OHP	-	17 hydroxyprogesterone
21-OHD	-	21-hydroxylase deficiency
ACTH	-	Adrenocorticotropic hormone
ART	-	Assisted Reproductive Techniques
CAH	-	Congenital adrenal hyperplasia
DHEA	-	Dehydroepiandrosterone
ELISA	-	Enzyme linked immunosorbent assay
EOS	-	Early onset sepsis
GDM	-	Gestational Diabetes Mellitus
HPA	-	Hypothalamic pituitary adrenal
HPLC	-	MS - high performance liquid chromatography
ICSI	-	Intracytoplasmic Sperm Injection
IUGR	-	Intrauterine Growth Restriction
IVF	-	In vitro fertilization
MAS	-	Meconium Aspiration Syndrome mass spectrometry
PCOS	-	Polycystic ovarian syndrome
PCR	-	Polymerase chain reaction
RIA	-	Radioimmunoassay
SV	-	Simple virilizing
UTI	-	Urinary Tract Infections

ABSTRACT

Maternal and Neonatal Factors Influencing 17-Hydroxyprogesterone levels in Newborn Delivered at a Tertiary Care Hospital

Introduction: Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder of adrenal steroidogenesis due to deficiency of enzymes involved in normal steroid synthesis. 21 hydroxylase being the most common enzyme deficiency accounting for 90% of the cases, of which 75% develop salt wasting crisis in the first 2 weeks of life. If undetected, CAH can lead to electrolyte abnormalities, adrenal crisis, and neonatal mortality. Screening neonates for CAH in various countries has been found to effectively reduce the neonatal morbidity and mortality. However, the levels of 17-hydroxyprogesterone (17-OHP) can be influenced by various perinatal factors including pregnancy induced hypertension, antenatal exposure to betamethasone/dexamethasone, and birth asphyxia making it difficult to determine the optimum cutoff values. Perinatal factors are known to elevate newborn 17-OHP values to levels reaching close to diagnostic cut offs for CAH which can be misleading. Higher 17-OHP levels in preterm is attributed to immaturity of the enzyme's activity, immature hepatic function leading to decreased metabolite clearance of 17-OHP, immature pituitary adrenal stress response, and cross reactions with conjugated steroids in the premature neonate's serum. Hence, necessitating the study of perinatal factors influencing 17-OHP levels in newborns.

Objectives:

1. To study the maternal and neonatal factors influencing the serum 17-hydroxyprogesterone levels in newborns

2. To estimate the recall rate using the current cut off levels of serum 17-hydroxyprogesterone levels in newborns.

METHODS

A hospital based observational study was conducted for a period of 1 year at KLE'S Dr. Prabhakar Kore Hospital. After obtaining ethical clearance from our institution and informed written consent from the parents of all the newborns who fulfilled the inclusion criteria were enrolled in the study. A questionnaire was administered by the study investigator. Information regarding mother's sociodemographic details, medical and obstetric history, and neonatal factors such as birth asphyxia, early-onset sepsis, NICU

Admission and APGAR score were recorded. Additionally, a newborn venous blood sample of 2ml was collected between 72 hours of birth and 10 days of life for evaluation of serum 17-OHP levels using Enzyme Linked Immunosorbent Assay (ELISA).

RESULTS AND ANALYSIS

The study concluded that maternal and neonatal factors significantly influence 17-hydroxyprogesterone (17-OHP) levels in newborns delivered at a tertiary care hospital. Maternal factors such as pregnancy-induced hypertension (PIH), premature rupture of membranes (PROM), and antenatal steroid administration were found to be associated with elevated 17-OHP levels, while gestational age, birth weight, and neonatal complications also played a role. Preterm newborns (≤ 32 weeks) exhibited higher 17-OHP levels compared to term newborns (≥ 37 weeks), with a borderline significant decreasing trend as gestational age increased. Although no significant

difference was observed in 17-OHP levels based on the mode of conception (spontaneous vs. assisted reproduction), newborns requiring NICU care, particularly those with neonatal respiratory distress, meconium aspiration syndrome, and early-onset sepsis, had higher 17-OHP levels. The study highlights the need for gestational age-specific reference values for 17-OHP in newborn screening programs to improve the accuracy of diagnosing congenital adrenal hyperplasia (CAH). Additionally, further large-scale, multicentric studies are recommended to validate these findings and explore the long-term clinical implications of altered 17-OHP levels in newborns.

CONCLUSION

The present study highlighted that, the maternal and neonatal factors influencing 17-OHP levels in newborns delivered at a tertiary care hospital. The findings demonstrate that, maternal age, gravida status, conception method, and perinatal factors play a role in determining neonatal 17-OHP levels. The present study revealed, that a majority of mothers were aged between 26-30 years, and primigravida cases were more common. Spontaneous conception was the predominant method, with only a small proportion of pregnancies resulting from assisted reproductive techniques. While statistical analysis did not show a significant difference in serum 17-OHP levels between spontaneous and ART-conceived pregnancies, it emphasizes, the complex interaction of maternal and neonatal factors in adrenal steroidogenesis.

KEYWORDS: Congenital adrenal hyperplasia, adrenal steroidogenesis, serum 17-hydroxyprogesterone

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INTRODUCTION

Congenital adrenal hyperplasia (CAH) is an group of hereditary disorders⁽⁴⁹⁾ disrupting the adrenal glands' normal steroid synthesis as a result of enzyme abnormalities. Deficiency of the enzyme 21-hydroxylase accounts for vast majority of cases (about 90%)⁽⁴³⁾. In the first two weeks after birth, around 75% of these affected people have salt-wasting crises, which may cause high mortality. Newborn 17-hydroxyprogesterone (17-OHP) levels maybe measured to help in the early diagnosis of congenital adrenal hyperplasia (CAH)⁽⁴⁹⁾, which in turn can reduce the likelihood of salt-wasting crises in males with normal genital organs and inaccurate gender assignment in female newborns with genital ambiguity.

CAH is primarily caused by - mutations in the **CYP21A2** gene,(encoding for the enzyme **21-hydroxylase 21-OH**), a crucial component of the **cytochrome P450** family⁽²⁷⁾. Other less common forms of CAH result from mutations in genes that code for enzymes such as:

- 11-beta-hydroxylase
- 17-alpha-hydroxylase
- 3-beta-hydroxysteroid dehydrogenase type 2 (3 β HSD2)
- Steroidogenic acute regulatory protein (StAR)
- P450 cholesterol side chain cleavage enzyme
- P450 oxidoreductase

Symptoms of external genitalia virilization may manifest in many ways in affected females. Additionally, 25% of cases have the less severe simple virilizing (SV) form of sickness. Symptoms such as acne, hirsutism, alopecia, and infertility, which are caused by elevated androgen levels, usually appear later in life with non-classical CAH.

Babies with typical CAH may develop adrenal crisis, a potentially fatal condition, if left untreated. Medical treatment with replacement hormone therapy, however, successfully restores hormonal balance. This treatment includes replacement

with hydrocortisone for cortisol deficiency and fludrocortisone for aldosterone deficiency. However, infants and children with typical CAH need treatment throughout their lives to prevent the return of symptoms and complications.

Main objective of screening CAH in newborns is for early detection as early initiation of treatment will reduce mortality and complications.

There has been a substantial drop in the cases of disease and mortality among infants afflicted with CAH since many nations instituted newborn screening programs. But it is difficult to determine an ideal threshold because 17-OHP levels can be impacted by many perinatal factors, like immediate postnatal stress, birth stress, the baby's gestational age, birth weight, delivery method (C-section vs. vaginal birth, for example), and maternal conditions (such as gestational diabetes). Additionally, it has been shown that prenatal variables may elevate newborn 17-OHP levels to levels that might be mistaken for CAH diagnostic criteria.

The approach used in the laboratory has an effect on the 17-OHP level measurement. Antibody assays such as "radioimmunoassay (RIA)" and "enzyme-linked immunosorbent assay (ELISA)" might lead to elevated 17-OHP results due to the presence of other hormones in the plasma. In contrast, "polymerase chain reaction (PCR)" and "high-performance liquid chromatography-mass spectrometry (HPLC-MS/MS)" are more precise and sensitive.

Treatment focusses on restoring hormone deficiency, reducing adrenal androgen production, and managing any problems that may arise. Potential side effects include a lack of height, rapid bone growth, premature or early puberty, infertility, and abnormal metabolic processes.

Few studies⁽³⁸⁾ examined the role of several factors on the 17-OHP level in preterm and full-term children in India, including sex, mode of delivery, gestational age, birth weight, and factors affecting both the mother and the baby. Additionally, there is no nationwide program in place to test newborns for CAH. Examining these impacts on the level of 17-OHP in neonate is the purpose of this research.

Genetics

Many people are compound heterozygotes for CAH, which is an inherited disorder with an autosomal recessive pattern. The "major histocompatibility complex" "(HLA)" class III region on chromosome 6p21.3, where "CYP21A2" gene is located. Situated next to CYP21A1P, this pseudogene is quite similar to "CYP21A2". A 30-kb deletion of the functioning CYP21A2 gene is one example of a genetic variation that arises from uneven crossing between these areas.^[5-7]

The number of known pathogenic variants of the CYP21A2 gene exceeds 300. Clinical manifestations of 21-hydroxylase enzyme activity range from salt-wasting CAH and non-classical CAH to simple virilizing CAH as a consequence of these mutations. The less severe of the two hereditary variations is usually the one that is matched with the reported clinical features. Particularly for variations linked to salt-wasting, non-classical CAH, has a very good association between genotype & phenotype, allowing for rather accurate estimates of illness severity.

As a result of combining CAH symptoms with those of "Ehlers-Danlos syndrome," a small subset of CAH individuals have been identified with CAH-X syndrome. The CAH-X mutation results from the loss of the "TENASCIN X" protein coded by the neighbouring "TNXB" gene and the "CYP21A2" gene, both of which are located in the extracellular matrix. Joint hypermobility, arthralgias, dislocations,

midline abnormalities, and hernias are common symptoms experienced by individuals with CAH-X.

Adrenal Physiology

The process of adrenal steroidogenesis involves the synthesis of adrenal hormones from scratch. All adrenal steroids originate from cholesterol, which is converted into glucocorticoids, mineralocorticoids, and androgens by a series of enzyme reactions. The steroidogenic acute regulatory protein (StAR) helps move cholesterol from outer mitochondrial membrane to inner mitochondrial membrane, where the sidechain cleavage enzyme “CYP11A1” converts it to pregnenolone⁽⁴⁹⁾.

Enzymes like “3 β -hydroxysteroid dehydrogenase (3 β -HSD), 21-hydroxylase, 11 β -hydroxylase”, and aldosterone synthase enhance the manufacture of mineralocorticoids in the zona glomerulosa⁽⁴⁵⁾. The zona fasciculata synthesis cortisol by means of 17 α -hydroxylase, which changes pregnenolone into 17 α -hydroxy-pregnenolone⁽⁴⁵⁾. Adrenocorticotrophic hormone (ACTH) operates via the “hypothalamic-pituitary-adrenal (HPA) axis” to control cortisol production⁽⁴⁶⁾. Finally, the enzyme 17,20-lyase converts 17 α -hydroxy-pregnenolone into dehydroepiandrosterone (DHEA) in the zona reticularis, where the production of adrenal androgens takes place.⁽⁴⁷⁾

Cortisol & Aldosterone Production - Cortisol deficiency → Increased ACTH (loss of negative feedback), Aldosterone deficiency → Salt-wasting and dehydration.

Accumulation of Precursors (due to enzyme block) leads to Increased 17-Hydroxyprogesterone (17-OHP)

Shunted Pathway → Excess Androgen Production 17-OHP → Androstenedione → Testosterone → Resulting in virilization (ambiguous genitalia in females)

Alternative Conversion (via 11 β -Hydroxylase)

17-OHP → 21-Deoxycortisol

21-Deoxycortisol = More Specific Biomarker for CAH Diagnosis

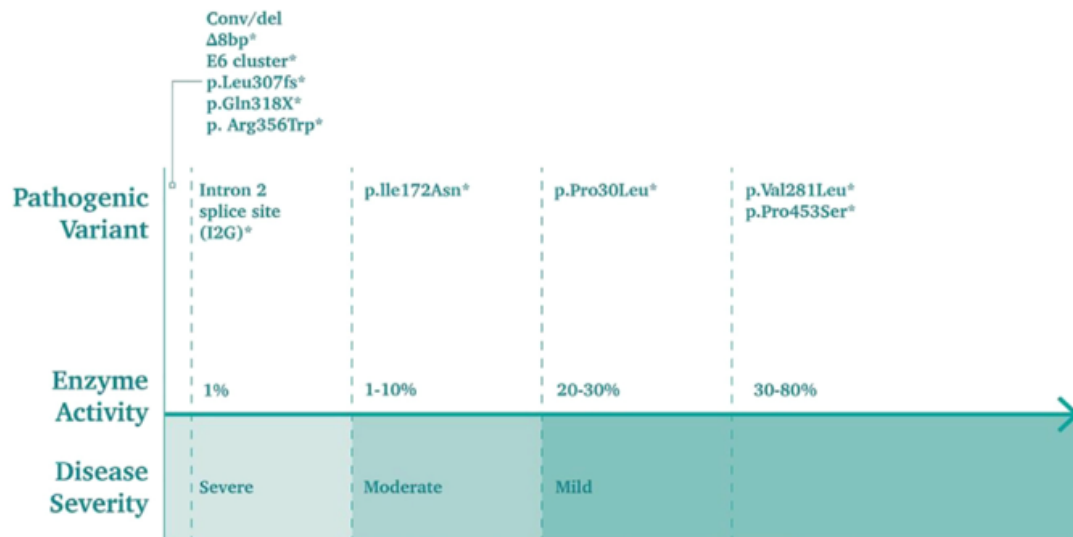


Figure 1. Correlation of Pathogenic Variant with Disease Severity. Congenital adrenal Hyper-plasia has a spectrum of clinical presentations that can be predicted fairly well from the path-ogenic variant for the severe and mild forms. Combination of variants causing severe and moderate CAH usually present as moderate CAH, while the combination of variants causing mild and moderate CAH usually present as mild CAH.

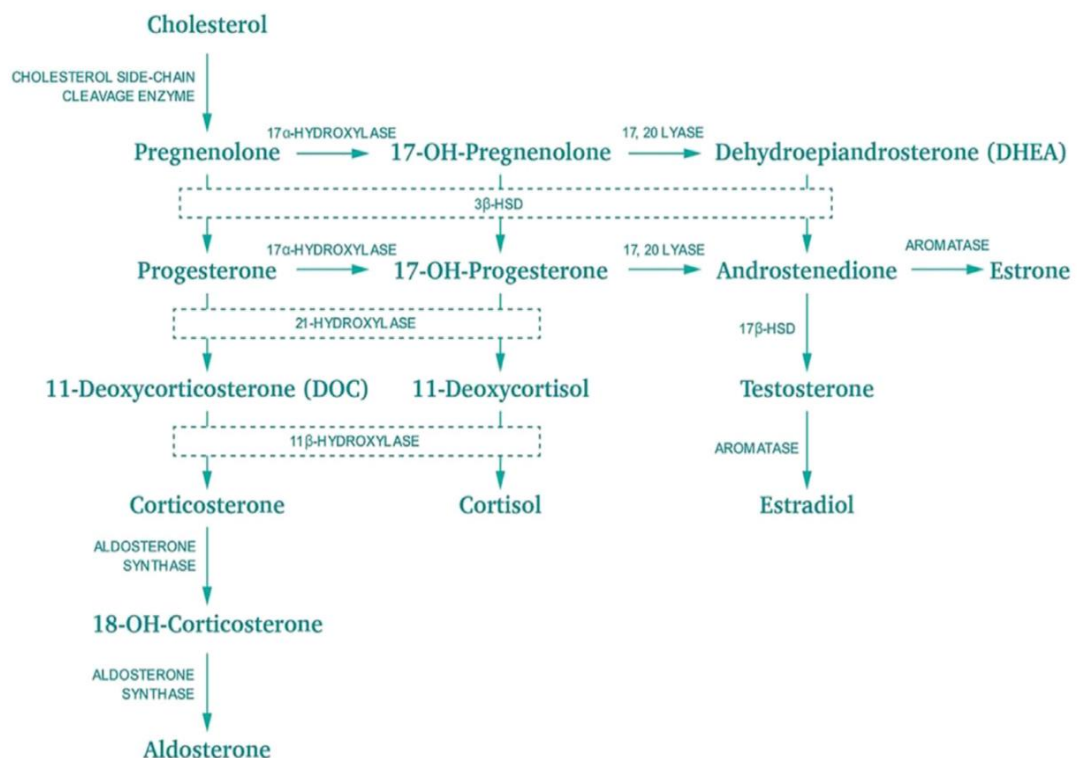


Figure 2. Steroidogenesis

Clinical Presentation

Classical (severe) and non-classical variants are the two primary categories of congenital adrenal hyperplasia (CAH) ⁽³³⁾. Additionally, 21-hydroxylase activity is almost non-existent in the classical type, which is further classified as salt-wasting CAH. A minor degree of enzyme function (around 1–10%) is retained in simple virilising CAH ⁽³³⁾. However, CAH manifests on a continuum, and instances may not necessarily fit neatly into these categories. While mineralocorticoid insufficiency is possible in simple virilising CAH, it is often milder than in the salt-wasting variety.

Salt Wasting CAH

In infants, a salt-wasting crisis may occur because to the significant shortages in aldosterone and cortisol caused by this illness, which should be detected as soon as possible ⁽²⁵⁾. Because of a blockage in the route that normally produces aldosterone and cortisol, the body of an individual with 21-hydroxylase deficiency produces an excess of androgens ⁽³⁰⁾. Atypical genitalia, as defined by the Prader Scale, develop in affected females ⁽³¹⁾. The degree of "virilization" affected by amount of androgen exposure that occurs during fetal development. The internal reproductive tissues of a female, including uterus, fallopian tubes, and ovaries, are present in individuals with this disorder ⁽⁴⁹⁾. Nevertheless, there are varying degrees of masculinization that may be seen in their external genitalia, including enlarged clitoris, partial labia fusion, and the existence of a urogenital sinus ⁽³²⁾. Despite the seemingly normal appearance of male genitalia, early detection is essential and requires strong suspicion (such as in cases when both parents are carriers) and newborn screening. ⁽³³⁾.

Simple Virilizing CAH

There is still some 21-hydroxylase activity in this milder kind of CAH ⁽²⁹⁾, which is distinct from the salt wasting subtype. Atypical genitalia presenting at birth may lead to a diagnosis in female child, although hyperandrogenism signs like "premature puberty, accelerated growth velocity, advanced bone age emerge" might delay a diagnosis until later in life. ⁽³¹⁾ These individuals usually keep up mineralocorticoid activity and have a cortisol deficit to varying degrees ⁽³⁰⁾. The newborn screen sometimes finds simple-virilizing CAH patients in addition to the more severe salt-wasting variant. ⁽²⁵⁾

Non-Classical CAH

Mildest form of CAH ⁽³²⁾. Hyperandrogenism, similar to the symptoms seen in simple virilising CAH, may occur in children, but typically their cortisol and aldosterone levels are normal ⁽²⁵⁾. It is possible for symptoms to emerge in maturity, although they may first present during adolescence ⁽³¹⁾. Underdiagnosis occurs often since many individuals do not exhibit any symptoms ⁽³²⁾. Symptoms such as acne, hirsutism, and irregular or nonexistent menstrual periods may be experienced by female patients. These symptoms might be similar to those of polycystic ovarian syndrome (PCOS) ⁽³¹⁾. Diagnosis in adult females may occur in some instances due to infertility or repeated miscarriages. ⁽³²⁾

AIMS AND OBJECTIVES

Objective of the study

PRIMARY OBJECTIVE:

- To study the maternal and neonatal factors influencing the serum 17-hydroxyprogesterone levels in newborns

SECONDARY OBJECTIVE:

- To estimate serum 17-OHP levels
- Recall rate for confirmatory test

REVIEW OF LITERATURE

“Congenital Adrenal Hyperplasia (CAH)”⁽⁴⁷⁾ encompasses a group of inherited disorders characterized by defects in adrenal steroidogenesis, leading to cortisol deficiency ⁽⁴⁹⁾and, in some cases, aldosterone deficiency. The most prevalent form, accounting for over ninety percent of CAH cases, is caused by 21-hydroxylase deficiency (21OHD)⁽⁴⁹⁾.

Different Types of CAH and Their Presentations ⁽²¹⁾

1. 21-Hydroxylase Deficiency:

- Classic Form:

- *Salt-Wasting (SW) CAH*: This severe form results in both cortisol and aldosterone deficiencies. Infants may show symptoms such as failure to thrive, dehydration, hyponatremia, and hyperkalaemia. Female infants often exhibit ambiguous genitalia due to prenatal androgen exposure, while male infants typically have normal genitalia but may develop symptoms shortly after birth.
- *Simple-Virilizing (SV) CAH*: Characterized by sufficient aldosterone production to prevent salt wasting but more androgen production leading to virilization. Females may have ambiguous genitalia at birth, whereas males usually appear normal but may show signs of early virilization.

- Non-Classic Form (NCCAH): A milder variant that may not manifest until later in childhood or adulthood. Symptoms can include signs of hyperandrogenism such as hirsutism, acne, irregular menstrual cycles in females, and early balding or infertility in males. Some individuals remain asymptomatic.
- 2. 11 β -Hydroxylase Deficiency: This form leads to excess deoxycorticosterone, causing hypertension and hypokalaemia. Increased androgen levels result in virilization similar to 21-hydroxylase deficiency.
- 3. “3 β -Hydroxysteroid Dehydrogenase” (3 β -HSD) Deficiency: Rare form causing impaired synthesis of adrenal steroids, leading to ambiguous genitalia in both sexes, salt wasting, and cortisol deficiency⁽⁴⁹⁾.
- 4. “17 α -Hydroxylase” Deficiency: Very rare form characterized by hypertension due to excess mineralocorticoids and sexual infantilism or ambiguous genitalia resulting from deficient sex steroid production.
- 5. “Congenital Lipoid Adrenal Hyperplasia”: Most severe form, resulting from defects in cholesterol transport into mitochondria. It leads to a near-complete lack of steroid production, causing severe salt wasting, cortisol deficiency, and ambiguous genitalia or under virilization in genetic males.

“Congenital Adrenal Hyperplasia (CAH)” in India, primarily caused by “21-hydroxylase” deficiency, has varying reported incidences depending on geographic, genetic, and socio-economic factors. The incidence is reported to be around 1 in 15,000 to 20,000 live births on national level⁽⁵¹⁾. However, this figure can vary significantly across different regions of India due to various influencing factors such as consanguinity, genetic mutations, and environmental conditions.

Global Prevalence of CAH

Global Incidence: The worldwide prevalence of CAH is estimated to be between 1 in 10,000 to 1 in 15,000 live births⁽⁴⁹⁾ for all forms of CAH, with the most common form being “21-hydroxylase deficiency” (21-OHD)⁽⁴³⁾.

A multicentric study conducted by the Indian Council of Medical Research (ICMR)⁽⁴⁸⁾ investigated the incidence of congenital adrenal hyperplasia (CAH) through newborn screening across five centres in India, including the All India Institute of Medical Sciences (AIIMS)⁽⁴⁸⁾. The study screened approximately 100,000 newborns and found an overall incidence of CAH to be 1 in 5,762 live births⁽⁴⁸⁾. Notably, there were significant regional variations, with Chennai reporting the highest incidence at 1 in 2,036, and Mumbai the lowest at 1 in 9,983⁽⁴⁸⁾. The incidence of the salt-wasting form of CAH was higher, at 1 in 6,934, compared to the simple virilizing type, which was 1 in 20,801⁽²²⁾.

In some regions with higher rates of consanguinity, particularly in rural and tribal populations, the incidence may be higher due to autosomal recessive inheritance patterns associated with CAH. In states like Tamil Nadu, where newborn screening programs are more common, there have been reports showing an incidence of about 1 in 2,575 live births for CAH (Krishna et al., 2016). As of now, India has not fully implemented nationwide neonatal screening for CAH, although there have been significant pilot programs in states like Tamil Nadu, Kerala, Maharashtra, and Delhi. Data on neonatal mortality rates specific to CAH in India are limited. However, a multicentric study⁽²²⁾ involving 104,066 neonates reported a CAH incidence of 1 in 5,762 live births.⁽²²⁾

Newborn screening for **CAH** is crucial for early diagnosis and treatment, preventing life-threatening **adrenal crises** and reducing morbidity. Most screening programs measured “**17-hydroxyprogesterone**” (**17-OHP**) levels in dried blood spot collected within the first 48–72 hours of life⁽²³⁾

1. Primary Screening Method:

- Immunoassay-based measurement of **17-OHP** in dried blood spot.
- Elevated 17-OHP suggests **21-hydroxylase deficiency (common CAH type)**.
- False positives are common in **preterm infants**, requiring adjusted cutoff values or second-tier testing⁽²⁵⁾

2. Second-Tier Testing (Confirmatory Tests):

- “**Liquid Chromatography-Tandem Mass Spectrometry (LC-MS/MS)**”: More specific for CAH diagnosis.
- **Genetic Testing**: Identifies mutations in the CYP21A2 gene for definitive diagnosis.
- **Electrolyte Monitoring**: Identifies salt-wasting crises in affected infants.

3. Global Implementation:

- NBS for CAH is **mandatory** in most of the developed countries (e.g., US, Europe, Japan).
- In **India**, pilot newborn screening programs exist, but **nationwide screening is not yet universally implemented**.

4. Challenges in India & Low-Resource Settings:

- High **false-positive rates** caused due to prematurity and stress.
- Need for cost-effective **second tier testing** increase specificity.
- **Limited awareness & infrastructure** for widespread NBS implementation.

The absence of universal screening has meant that the full burden of CAH remains undiagnosed in many parts of the country, leading to delayed diagnoses and interventions.

Understanding and studying the factors affecting 17-OHP levels in specific regions of India is crucial for optimizing CAH screening and improving neonatal health outcomes. With India's genetic diversity, varied environmental exposures, and socio-economic challenges, regional studies allow for better-targeted interventions, more accurate diagnoses, and improved public health policies. By improving neonatal screening, addressing genetic variations, and understanding local environmental and socio-economic factors, India can enhance the early detection and treatment of CAH, leading to better outcomes for affected infants.

Maternal and Neonatal Factors Affecting 17-Hydroxyprogesterone (17-OHP) Levels in Newborns

17-Hydroxyprogesterone levels in newborns are affected by a range of maternal and neonatal factors that affect foetal adrenal function and steroidogenesis. These factors must be considered when interpreting results in newborn screening programs, especially for “congenital adrenal hyperplasia” (CAH). Additionally, specific socio-economic, genetic, and healthcare challenges in countries like India further complicate these factors.

1. Maternal Factors

Maternal Health Conditions such as Gestational Diabetes Mellitus (GDM) can lead to elevated 17-OHP levels in neonates due to maternal hyperglycaemia. Hyperglycaemia alters placental function, increasing hormone transport and foetal

stress responses, stimulating adrenal steroidogenesis. Studies show neonates from poorly controlled GDM pregnancies exhibit higher 17-OHP levels compared to normoglycemic pregnancies (Bittencourt et al., 2012⁽³⁾; Wadhwa et al., 2018)⁽¹⁹⁾ In Hypertension and Preeclampsia these conditions compromise placental blood flow, leading to foetal stress and increased steroid production as hypoxia and oxidative stress enhance foetal adrenal activity.

Maternal infections or chronic inflammation activate the foetal “hypothalamic-pituitary-adrenal” (HPA) axis, leading to increased 17-OHP production. Elevated maternal cytokines cross the placenta, triggering foetal adrenal hyperactivity. Studies have shown that infections, such as urinary tract infections (UTIs) and maternal febrile illnesses, elevate foetal adrenal hormone levels (Wolfe et al., 2013)⁽²⁰⁾.

Maternal medications, lifestyle factors, stress, placental function, and labor conditions all influence fetal adrenal activity, particularly the production of 17-hydroxyprogesterone (17-OHP). Corticosteroids, commonly administered to mothers during preterm labor, suppress fetal adrenal activity by exerting negative feedback on the fetal hypothalamic-pituitary-adrenal (HPA)⁽⁵⁰⁾axis, leading to decreased endogenous steroid production, including 17-OHP. A study by Ferguson et al. (2014)⁽⁶⁾ demonstrated that corticosteroid administration resulted in lower neonatal 17-OHP levels. Similarly, anti-seizure medications such as phenytoin and valproic acid may interfere with enzymatic pathways critical for steroidogenesis, potentially increasing fetal 17-OHP levels. Barry et al. (2017)⁽²⁾ found higher 17-OHP levels in neonates of mothers taking phenytoin during pregnancy.

A study by **Wendell-Smith et al. (1998)**⁽²⁶⁾ examined the impact of antenatal corticosteroid exposure on neonatal 17-OHP levels. The findings indicated that low-

birth-weight newborns exposed to corticosteroids had a mean “17-Hydroxyprogesterone level” of 52 ng/mL, in comparison to 35 ng/mL in unexposed infants ($P < .001$). This suggests that antenatal corticosteroid administration may elevate neonatal 17-OHP levels. ⁽²⁶⁾

Maternal stress and psychological factors also play a role in fetal adrenal activity. Increased maternal cortisol, crossing the placenta, stimulates fetal adrenal steroid production, leading to elevated 17-OHP levels. Entringer et al. (2010) ⁽⁵⁾ observed that maternal stress was associated with higher neonatal 17-OHP levels, particularly in pregnancies with high perceived stress. Additionally, lifestyle factors such as smoking can impact fetal adrenal function. Nicotine exposure activates the fetal HPA axis, disrupting normal steroidogenesis, and Hackshaw et al. (2011) ⁽⁸⁾ reported elevated 17-OHP levels in neonates exposed to maternal smoking. Nutrition also plays a crucial role, as maternal malnutrition, particularly deficiencies in cholesterol and essential vitamins, affects fetal adrenal function. Cholesterol serves as a precursor for steroid hormones, and deficiencies may lead to compensatory increases in adrenal activity. Research by Ghosh et al. (2013) ⁽⁷⁾ in South Asia highlighted a direct correlation between maternal nutrition and neonatal steroid hormone production.

The maternal-fetal interaction via the placenta is another key determinant of fetal adrenal function. Placental insufficiency, often linked to conditions like preeclampsia or intrauterine growth restriction (IUGR), reduces oxygen and nutrient delivery, stimulating fetal stress responses and increasing 17-OHP levels. Parker et al. (2014) ⁽¹²⁾ demonstrated that placental insufficiency leads to abnormal fetal steroidogenesis. Additionally, placental enzyme activity, particularly 11 β -hydroxysteroid dehydrogenase, plays a crucial role in regulating corticosteroid

conversion and maternal-fetal cortisol transfer. Research by Fowden et al. (2016)⁽³⁵⁾ suggested that dysregulation of these enzymes can alter fetal adrenal hormone levels.

Finally, the timing of delivery and labor conditions influence neonatal 17-OHP levels. Neonates delivered via elective cesarean section without labor often exhibit higher 17-OHP levels due to the absence of stress-induced hormonal adaptations. Spector et al. (2015) reported elevated 17-OHP levels in neonates born via cesarean section compared to those delivered vaginally. Additionally, prolonged or difficult labor can significantly increase fetal adrenal steroid production. Research by Newnham et al. (2011) confirmed that stressful labor conditions elevate maternal and fetal cortisol, leading to increased 17-OHP levels in neonates.

Neonatal factors such as gestational age, birth weight, stress, enzymatic activity, timing of sample collection, sex differences, and genetic variations all contribute to fluctuations in 17-hydroxyprogesterone (17-OHP) levels. Preterm neonates generally have higher 17-OHP levels compared to term neonates due to immature adrenal glands and incomplete enzymatic pathway development.

Studies by Berenbaum et al. (2017)⁽¹⁾ found significantly elevated 17-OHP levels in neonates born before 32 weeks of gestation. Low birth weight (LBW) neonates may also exhibit increased 17-OHP levels due to stress or adrenal immaturity. Patole et al. (2018)⁽¹³⁾ reported higher 17-OHP levels in LBW neonates in India, likely due to fetal hypoxia. Neonatal stress and illness, such as sepsis, respiratory distress syndrome (RDS), or hypoglycemia, further elevate 17-OHP levels through activation of the neonatal hypothalamic-pituitary-adrenal (HPA) axis, which stimulates adrenal steroidogenesis.

Hagg et al. (2012)⁽⁹⁾ found that critically ill neonates had significantly higher 17-OHP levels than healthy controls. Additionally, variability in enzymatic activity, particularly 21-hydroxylase function, can impact 17-OHP levels. White et al. (2010)⁽¹⁸⁾ demonstrated that genetic mutations affecting this enzyme result in elevated 17-OHP levels, as seen in congenital adrenal hyperplasia (CAH). Timing of sample collection is another critical factor, as 17-OHP levels peak shortly after birth and decrease over the initial days of life.

Neonatal screening programs recommend sample collection between 24- and 72-hours post-birth to minimize physiological fluctuations (Parker et al., 2014)⁽¹²⁾. Furthermore, sex differences play a role, with studies by Korytkowski et al. (2016)⁽¹⁰⁾ showing slightly higher 17-OHP levels in male neonates compared to females in the early neonatal period. Genetic variations, including polymorphisms in enzymes involved in steroidogenesis, also influence 17-OHP levels. Variants in CYP21A2, the gene encoding 21-hydroxylase, can lead to variable enzymatic activity and altered hormone levels (White et al., 2010)⁽¹⁸⁾.

In India, several challenges and considerations impact newborn screening for adrenal disorders like CAH. Currently, newborn screening programs are limited to certain states and urban centers, such as Tamil Nadu, Kerala, Maharashtra, and Delhi. A pilot study in Tamil Nadu screened 20,000 newborns for CAH, reporting a prevalence of 1 in 2,575 live births (Sundararajan et al., 2017)⁽¹⁶⁾. Genetic variations in Indian populations, particularly CYP21A2 mutations, influence steroidogenesis and 17-OHP levels. Venkatesh et al. (2013)⁽¹⁷⁾ identified prevalent mutations such as I2G and Q318X among Indian CAH patients. Neonatal health and environmental factors, including high rates of low birth weight and preterm births, further contribute to elevated 17-OHP levels. Studies in Maharashtra have shown significantly higher 17-

OHP levels in LBW neonates compared to normal-weight neonates (Patole et al., 2018)⁽¹³⁾. Socioeconomic and healthcare disparities also affect neonatal screening and diagnosis. Limited access to healthcare in rural and underserved areas delays the identification of adrenal disorders, with studies from Bihar and Uttar Pradesh indicating that less than 5% of neonates undergo metabolic screening, leading to underreporting of CAH cases (Singh et al., 2015)⁽¹⁴⁾.

Maternal malnutrition, particularly micronutrient deficiencies, can impact fetal adrenal function. Choudhury et al. (2012)⁽⁴⁾ found that neonates born to malnourished mothers in West Bengal had elevated 17-OHP levels, likely due to compensatory adrenal activation. Additionally, traditional practices and cultural beliefs pose challenges, as a significant proportion of births in rural India occur at home without medical supervision, increasing neonatal morbidity and delaying the diagnosis of endocrine abnormalities.

Need for study:

Both maternal and neonatal factors play crucial roles in determining 17-OHP levels in newborns. These factors need to be carefully considered in neonate screening, particularly for CAH.

In this study the aim is to determine the effects of gender, mode of delivery, gestational age, birth weight, maternal factors such as maternal gestational diabetes mellites, pregnancy induced hypertension, PROM, use of antenatal steroids and neonatal factors such as birth asphyxia, respiratory distress, sepsis affecting 17-OHP levels. Also to estimate serum 17-OHP levels & Recall rate for confirmatory test.

MATERIALS AND METHODS

Source of Data: All neonates born alive in KLE Hospital during study period.

Method of collection of data

Study Design: Hospital - based observational study.

Study Duration : One year (September 2023 – August 2024)

Study Place : Study to be done in KLE Hospital affiliated to JNMC Belagavi.

Inclusion Criteria:

All neonates born alive during the study period of one year in KLE Hospital.

Belagavi

Exclusion Criteria:

Lethal congenital anomalies

Sample size:

$$N = \left(\frac{Z_{\alpha/2} \times SD}{E} \right)^2$$

- Confidence Level: 99%
- $Z_{\alpha/2} = 2.58$
- Standard Deviation $SD = 3.96$
- Margin of Error $E = 12.47\%$ of SD

Calculation:

$$E = 0.1247 \times 3.96 = 0.4938$$

$$N = \left(\frac{2.58 \times 3.96}{0.4938} \right)^2$$

$$N = \left(\frac{10.2168}{0.4938} \right)^2$$

$$N = (20.69)^2$$

$$N = 428 \text{ (rounded)}$$

Sample technique: Simple Random Sampling

METHODOLOGY:

PROTOCOL :

Prospective observational study data collected from a tertiary care hospital for period over 1 year (2023-2024) .Institutional ethical committee approval was taken. All neonates in the centre were tested for 17-OHP.The laboratory values and clinical history of all neonates were recorded in a performas. A total of 428 neonates were included in this study. The blood samples taken between day 3 to day 10 of life and 17-OHP levels were measured by competitive “Enzyme Linked immuno assay” ,kit used was Accubind stored at 2-8⁰ C . Serum Blood sample of 25 microlitre was taken and stored at minus twenty degree (-20⁰), cold centrifugation done at 3500 rpm over 10 minutes. The following factors were analysed maternal factors such as pregnancy induced hypertension, PROM, Gestational diabetes mellitus, hypothyroidism, anaemia, polyhydramnios, oligohydramnios,hypertension,epilepsy, neonatal factors such as neonatal respiratory distress, neonatal pneumonia, meconium aspiration syndrome(MAS), early onset sepsis(EOS), necrotizing enterocolitis, birth asphyxia, neonatal seizures, neonatal hyperbilirubinemia.

MEASUREMENT OF OUTCOMES :



Figure 3: AccuBind ELISA microwells

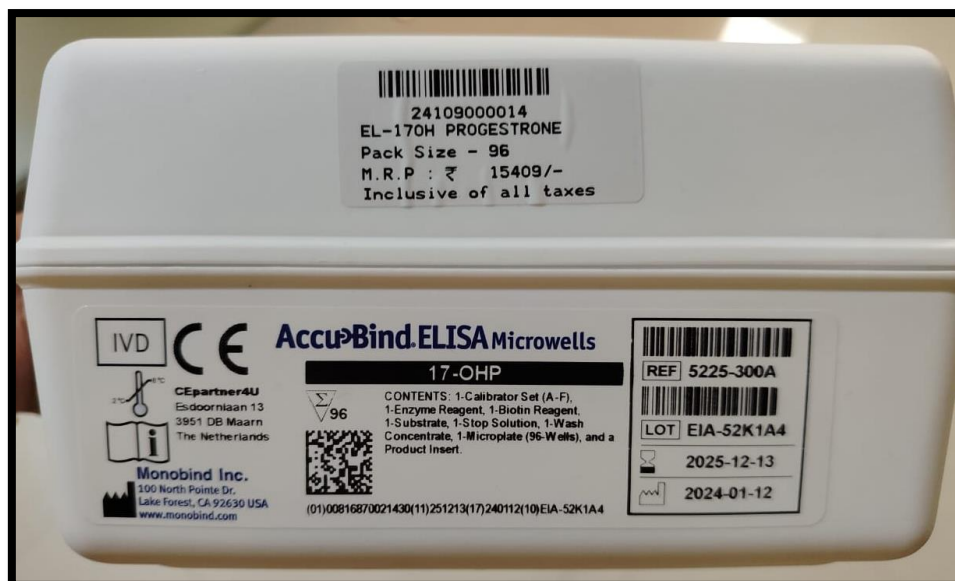


Figure 4 : AccuBind ELISA Microwells



Figure 5 : Centrifuge Machine used for the study

RESULTS

Statistical methods:

Descriptive type of analysis was done by mean and standard deviation for quantitative variables, frequency, proportion for categorical variables. Data was represented using appropriate diagrams.

Comparison of maternal and neonatal characteristics with serum 17-hydroxyprogesterone levels was performed using independent t-tests and one-way ANOVA, depending on the number of groups being compared. An independent t-test was used to compare mean serum 17-hydroxyprogesterone newborn levels between two groups, such as gravida status, maternal complications, and neonatal outcomes. A one-way ANOVA test was applied for comparisons across multiple categories, such as gestational age and weight at birth.

All analyses were conducted using standard statistical software SPSS 10.

Table 1: Age wise distribution of mothers

Mother's Age	No of mothers	% of mothers
<=20yrs	16	3.79
21-25yrs	142	33.65
26-30yrs	198	46.92
>=31yrs	66	15.64
Total	422	100.00
Mean	26.78	
SD	3.75	

The maternal age distribution in the study sample (N = 422) was as follows: ≤ 20 years 16 (3.79%), 21–25 years 142 (33.65%), 26–30 years 198 (46.92%), and ≥ 31 years 66 (15.64%). The mean maternal age was 26.78 years (SD = 3.75), indicating moderate variability. These findings suggest that the majority of mothers were in their mid-to-late twenties, with fewer in the youngest (≤ 20 years) and oldest (≥ 31 years) age groups.

Graph 1: Age wise distribution of mothers

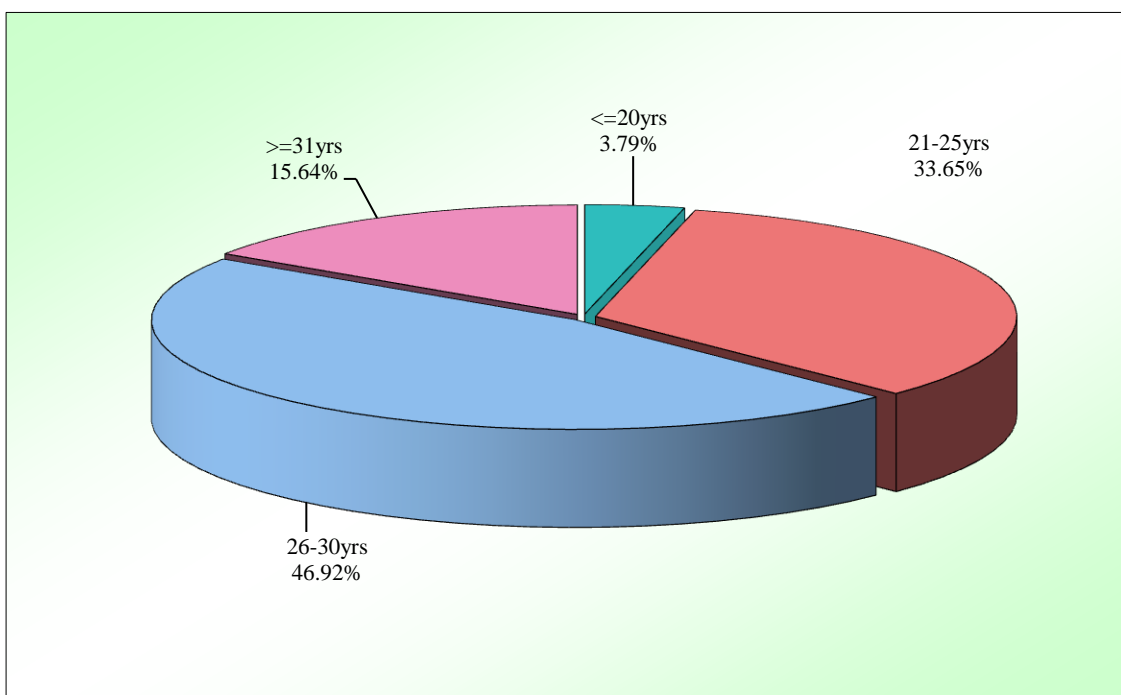


Table 2: Gravida wise distribution of mothers

Gravida	No of mothers	% of mothers
Primigravida	223	52.84
Multigravida	199	47.16
Total	422	100.00

The gravida-wise distribution of mothers in the study sample (N = 422) showed that primigravida mothers accounted for 223 (52.84%), while multigravida mothers comprised 199 (47.16%). This indicates a nearly even distribution, with a slight predominance of first-time mothers in the study population.

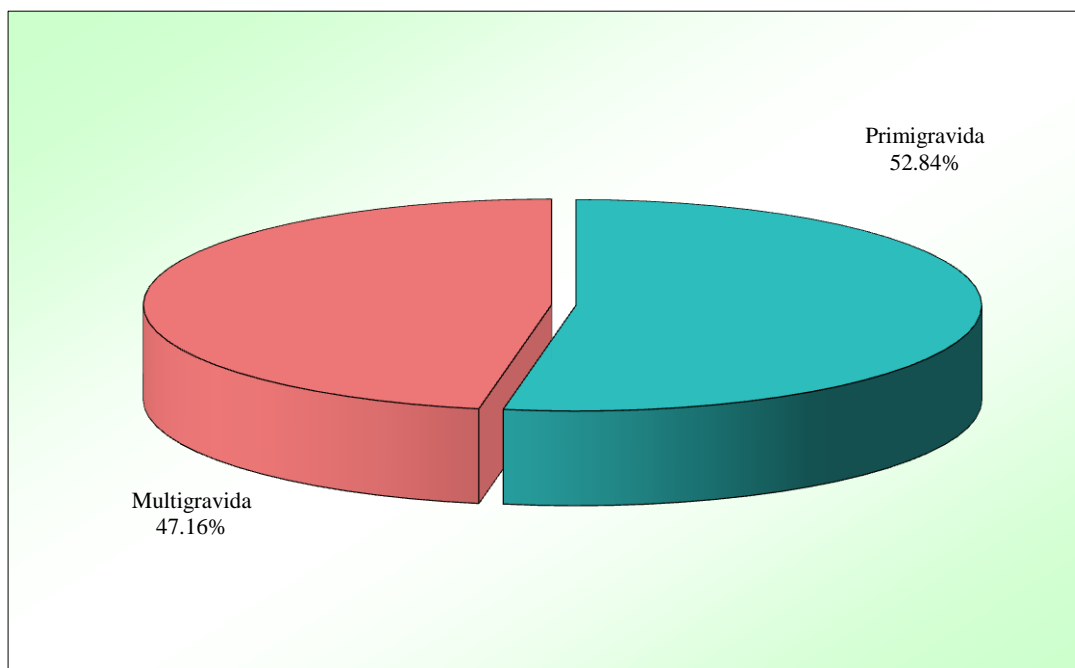
Graph 2: Gravida wise distribution of mothers

Table 3: Comparison of Gravida with Serum 17-hydroxyprogesterone levels (ng/ml) by t test

Gravida	Min	Max	Mean	SD	t-test	p-value
Primigravida	1.50	25.00	8.87	3.25	0.249	0.803
Multigravida	4.10	19.20	8.80	2.83		
Total	1.50	25.00	8.84	3.06		

The mean 17-OHP levels for the total study population (N = 422) was 8.84 (SD = 3.06), with values ranging from 1.50 to 25.00. Newborns born to primigravida mothers had a mean 17- OHP level of 8.87 (SD = 3.25), while newborns born to multigravida mothers had a mean of 8.80 (SD = 2.83).

A t-test comparing the two groups yielded a test statistic of 0.249 with p-value of 0.803, indicating no statistically significant difference in mean 17 OHP levels of newborns between primigravida and multigravida mothers.

Graph 3: Comparison of Gravida with Serum 17-hydroxyprogesterone levels (ng/ml)

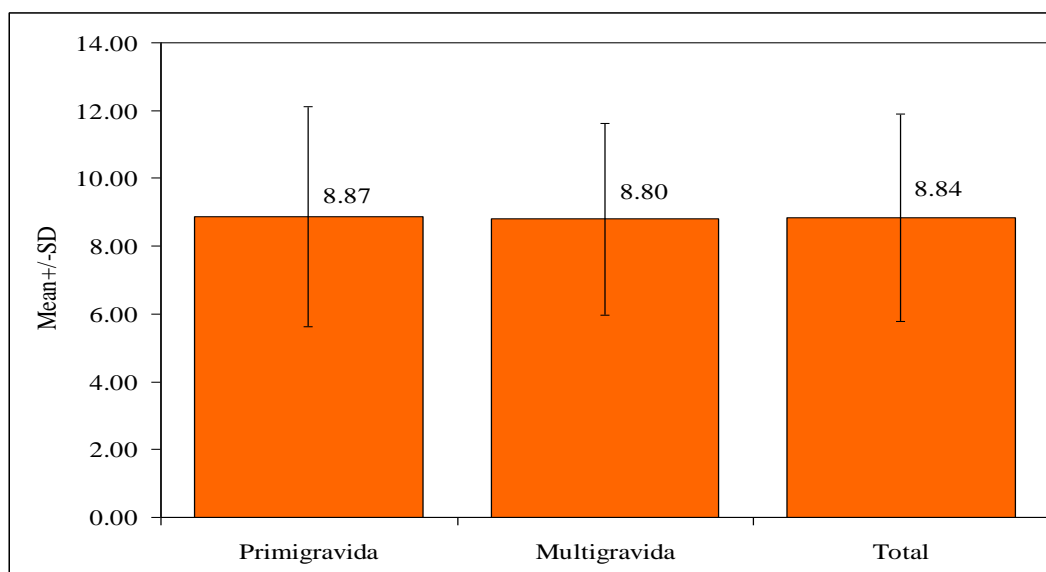


Table 4: Parity wise distribution of mothers

Parity	No of mothers	% of mothers
Primigravida	134	31.75
Multi	48	11.37

The parity-wise distribution of mothers in the study sample (N = 422) showed that primigravida mothers accounted for 134 (31.75%), while multigravida mothers comprised 48 (11.37%). This indicates that a higher proportion of mothers were experiencing their first childbirth compared to those with multiple childbirths.

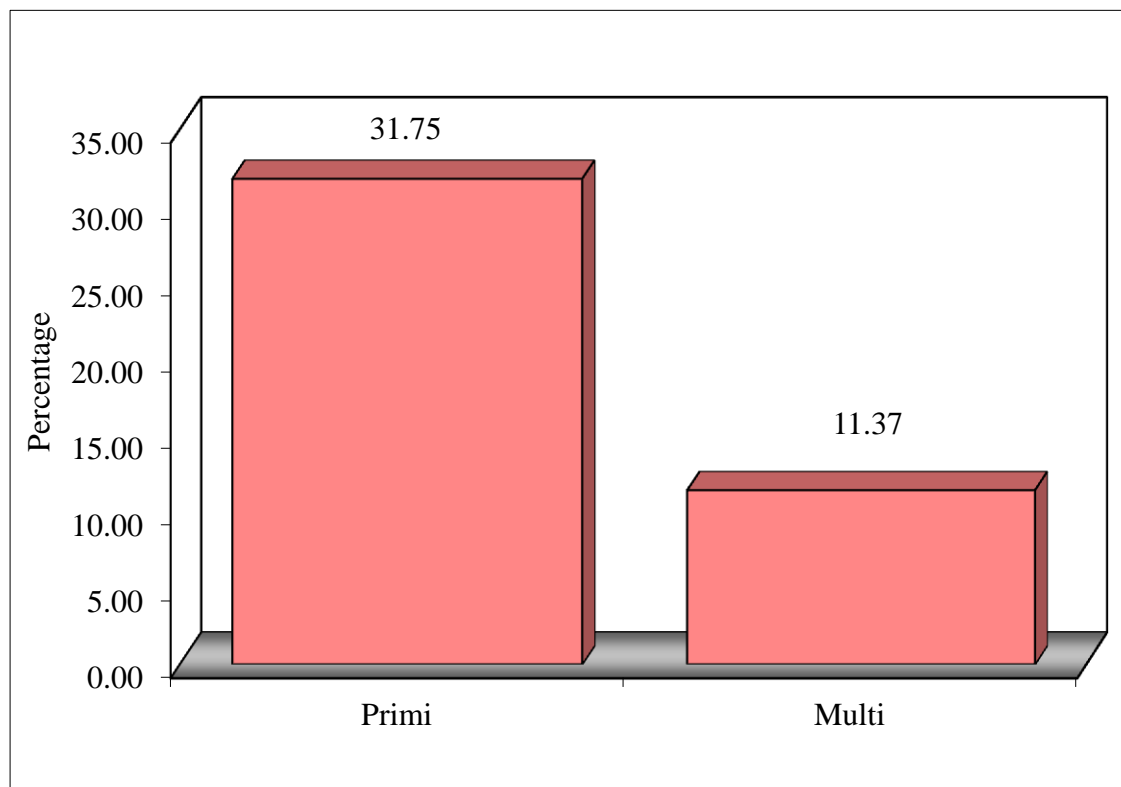
Graph 4: Parity wise distribution of mothers

Table 5: Abortions wise distribution of mothers

Abortions	No of mothers	% of mothers
No	362	85.78
One	56	13.27
Two	4	0.95
Total	422	100.00

The distribution of mothers based on abortion history in the study sample (N = 422) showed that 362 (85.78%) had no history of abortion, while 56 (13.27%) had one abortion, and 4 (0.95%) had two abortions.

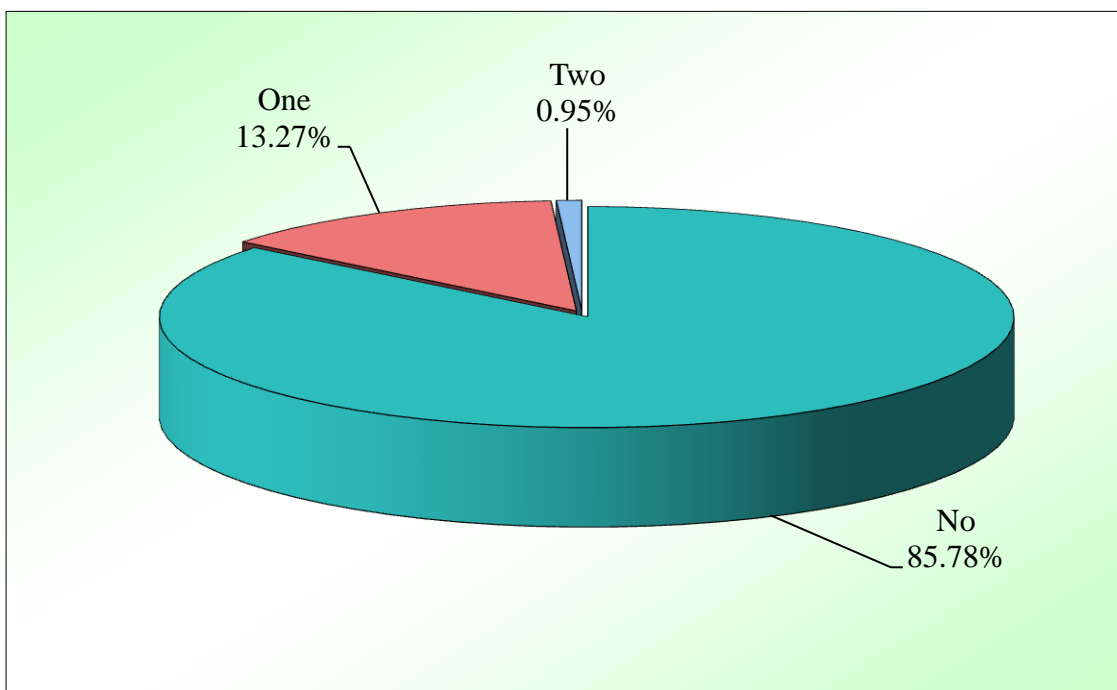
Graph 5: Abortions wise distribution of mothers

Table 6: Living children wise distribution of mothers

Living children	No of mothers	% of mothers
1	130	30.81
2	27	6.40
3	1	0.24

The distribution of mothers based on the number of living children in the study sample (N = 422) showed that 130 (30.81%) had one living child, 27 (6.40%) had two living children, and only 1 (0.24%) had three living children. This indicates that most mothers had either one or no living children, with very few having two or more living children.

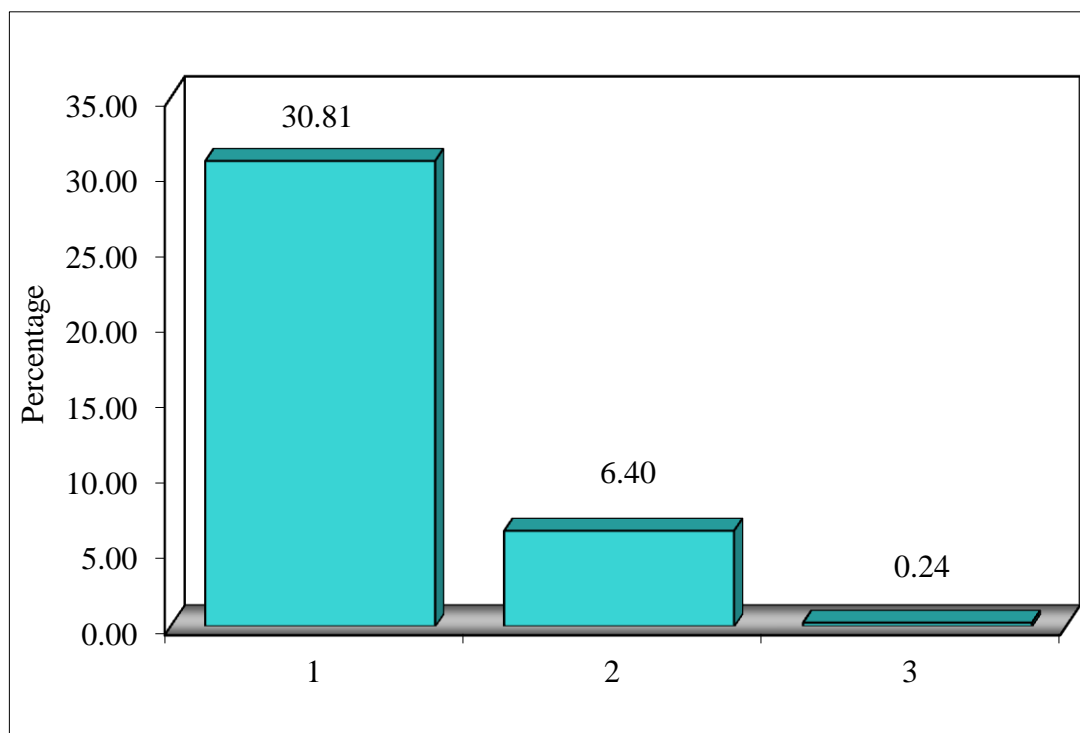
Graph 6: Living children wise distribution of mothers

Table 7: Current pregnancy wise distribution of mothers

Current pregnancy	No of mothers	% of mothers
Assisted Reproduction Technique	18	4.27
Spontaneous	404	95.73
Total	422	100.00

The distribution of mothers based on the type of conception in the study sample (N = 422) showed that 404 (95.73%) had a spontaneous pregnancy, while 18 (4.27%) conceived through Assisted Reproduction Techniques. This indicates that most pregnancies occurred naturally, with a small proportion requiring medical assistance for conception in this study.

Graph 7: Current pregnancy wise distribution of mothers

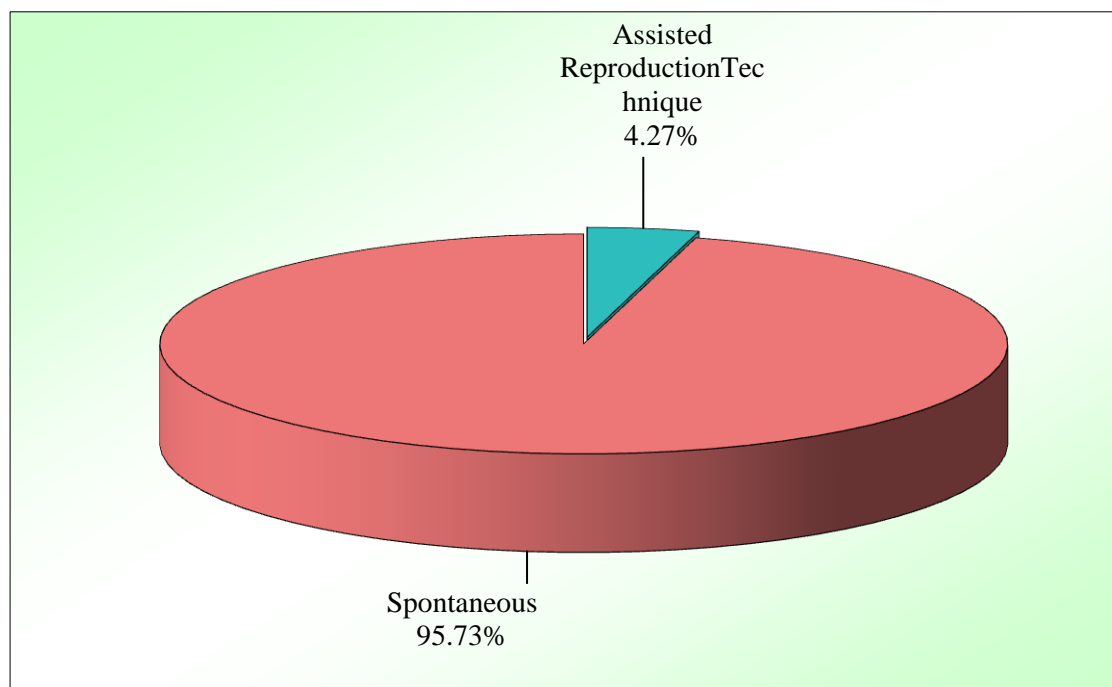


Table 8: Comparison of Conception method with Serum 17-hydroxyprogesterone levels (ng/ml) by t test

Current pregnancy	Min	Max	Mean	SD	t-test	p-value
Assisted Reproduction Technique	5.80	18.30	9.52	3.02	0.973	0.331
Spontaneous	1.50	25.00	8.81	3.06		
Total	1.50	25.00	8.84	3.06		

The comparison of serum 17-hydroxyprogesterone levels (ng/ml) between different conception methods in the study sample (N = 422) showed that the mean level was 9.52 (SD = 3.02) in mothers who conceived through Assisted Reproduction Techniques and 8.81 (SD = 3.06) in those with spontaneous conception.

A t-test yielded a test statistic of 0.973 with p-value of 0.331, indicating no statistically significant difference in serum 17-hydroxyprogesterone levels in newborns conceived by spontaneous or assisted reproductive techniques.

Graph 8: Comparison of different conception methods with Serum 17-hydroxyprogesterone levels (ng/ml)

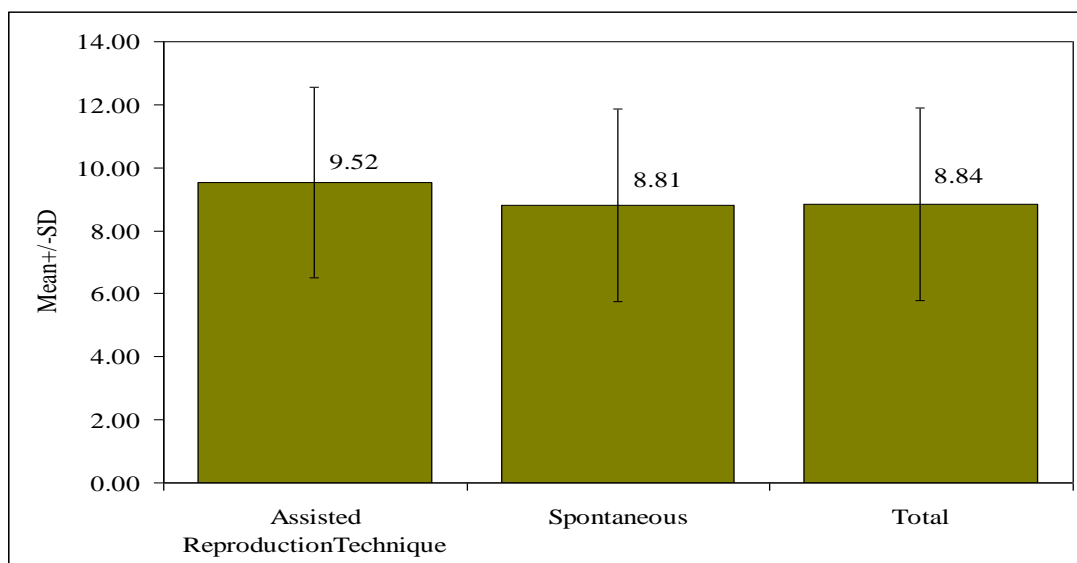


Table 9: Birth history- Period of Gestational Age

Period of Gestational Age	No of children	% of children
≤ 32 weeks	10	2.37
33-36weeks	76	18.01
≥ 37 weeks	336	79.62
Mean	37.65	
SD	1.98	
Total	422	100.00

The distribution of study population based on the gestational age in study sample (N = 422) showed that 336 (79.62%) of the children were born at ≥ 37 weeks, while 76 (18.01%) were born between 33–36 weeks, and 10 (2.37%) were born at ≤ 32 weeks. The mean gestational age at birth was 37.65 weeks (SD = 1.98), indicating that the majority of births occurred at term, with a smaller proportion of preterm deliveries in this study.

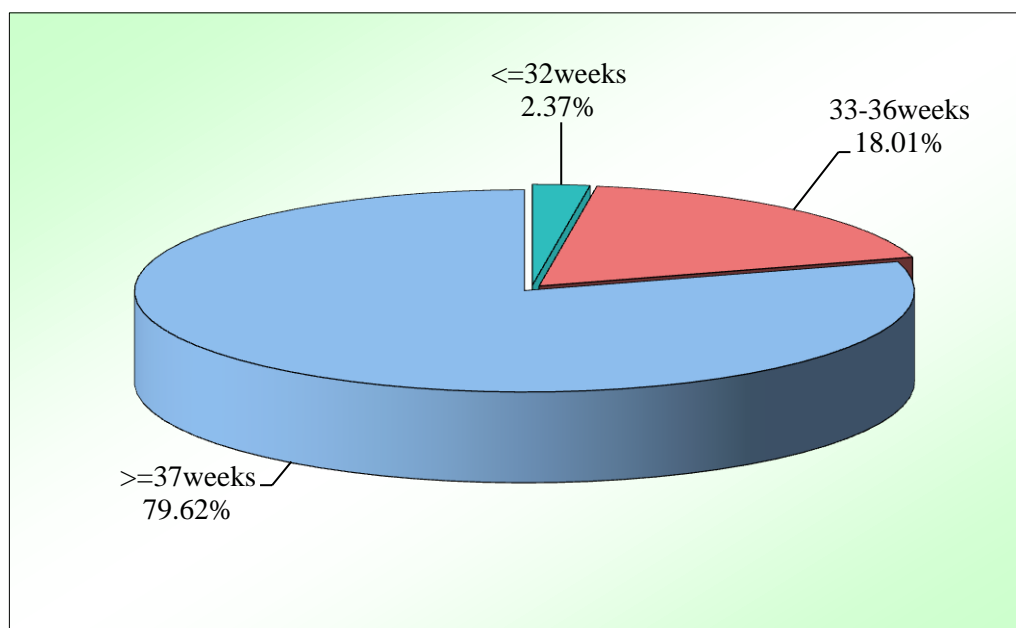
Graph 9: Birth history- Period of Gestational Age

Table10: Comparison of Period of Gestational Age with Serum 17-hydroxyprogesterone levels (ng/ml) in newborns by one way ANOVA test

Period of Gestational Age	Min	Max	Mean	SD	F-test	p-value
≤32weeks	4.90	19.50	9.92	4.03	2.9393	0.0540
33-36weeks	4.00	22.00	9.48	3.96		
≥37weeks	1.50	25.00	8.66	2.76		
Total	1.50	25.00	8.84	3.06		

Mean serum 17-hydroxyprogesterone levels varied across different gestational age groups. Newborns born at ≤32 weeks had the highest mean levels (Mean = 9.92, SD = 4.03), followed by those born between 33–36 weeks (Mean = 9.48, SD = 3.96). The lowest mean levels were observed in newborns born at ≥37 weeks (Mean = 8.66, SD = 2.76).

The overall difference in serum 17-hydroxyprogesterone levels across the gestational age groups approached statistical significance (F = 2.939, p = 0.054).

Graph 10: Comparison of Period of Gestational Age with Serum 17-hydroxyprogesterone levels (ng/ml)

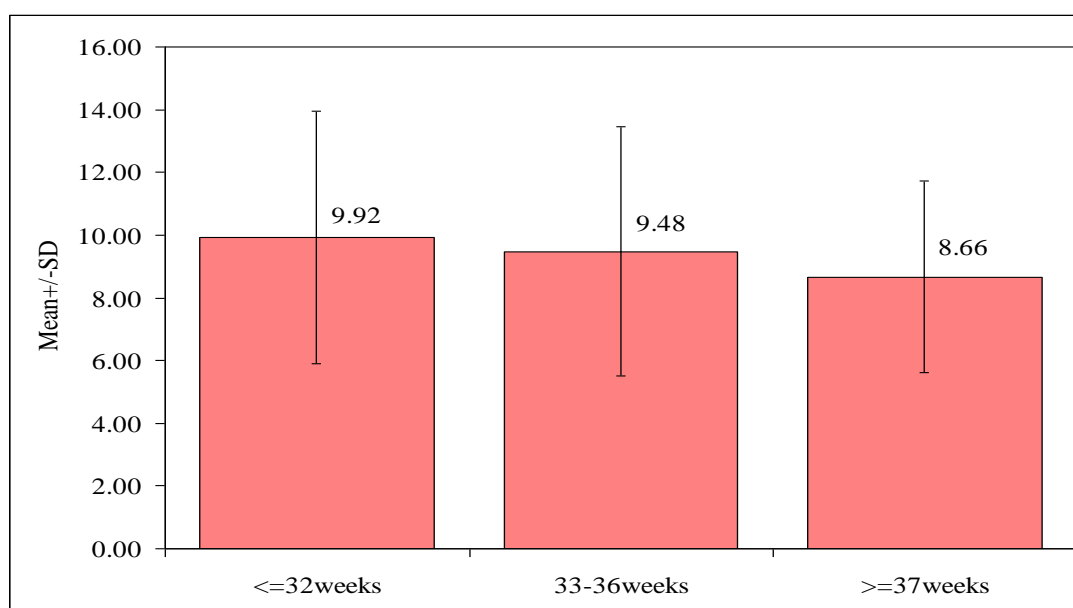


Table 11: Birth history- Gender of baby

Gender of baby	No of children	% of children
Male	248	58.77
Female	174	41.23
Total	422	100.00

The distribution of newborns by gender in the study sample (N = 422) showed that 248 (58.77%) were male, while 174 (41.23%) were female. This shows a higher proportion of male births in comparison to female births in the study population.

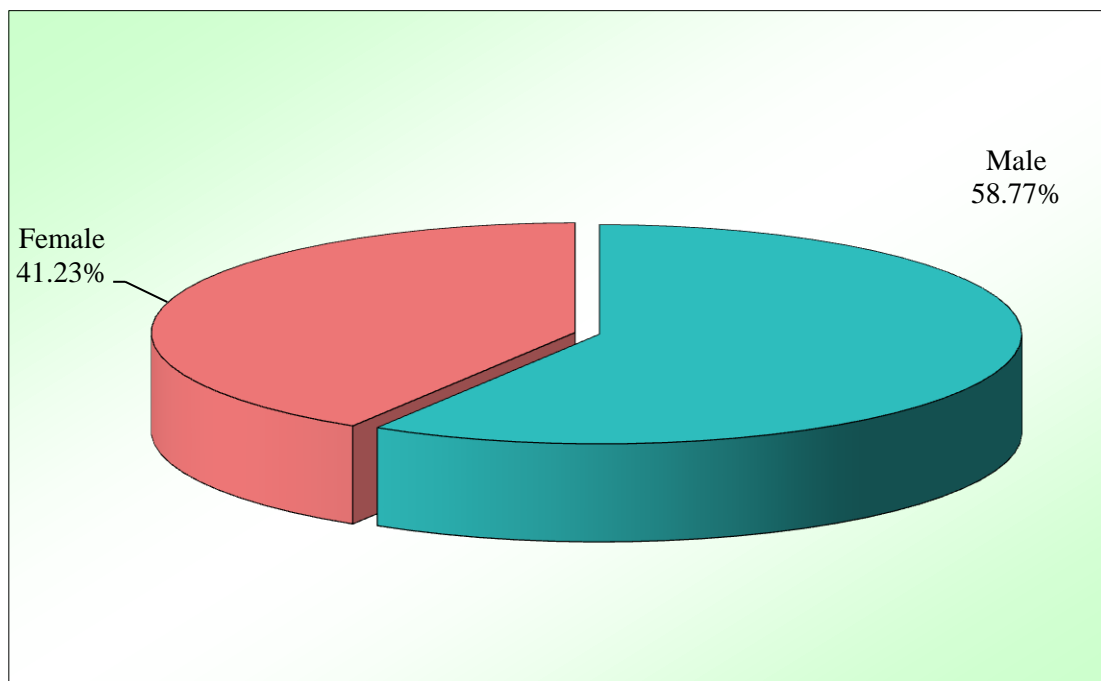
Graph 11: Birth history- Gender of baby

Table 12: Birth history- Birth weight of baby

Birth weight of baby	No of children	% of children
<1kg	5	1.18
1-2.4kg	134	31.75
≥ 2.5 kg	283	67.06
Mean	2.57	
SD	0.49	
Total	422	100.00

The distribution of birth weight among newborns in the study sample (N = 422) showed that 283 (67.06%) had a birth weight of ≥ 2.5 kg, while 134 (31.75%) weighed between 1–2.4 kg, and 5 (1.18%) weighed <1 kg. The mean birth weight was 2.57 kg (SD = 0.49), indicating majority of newborns had a normal birth weight, with a smaller proportion falling into the low or very low birth weight categories in the study.

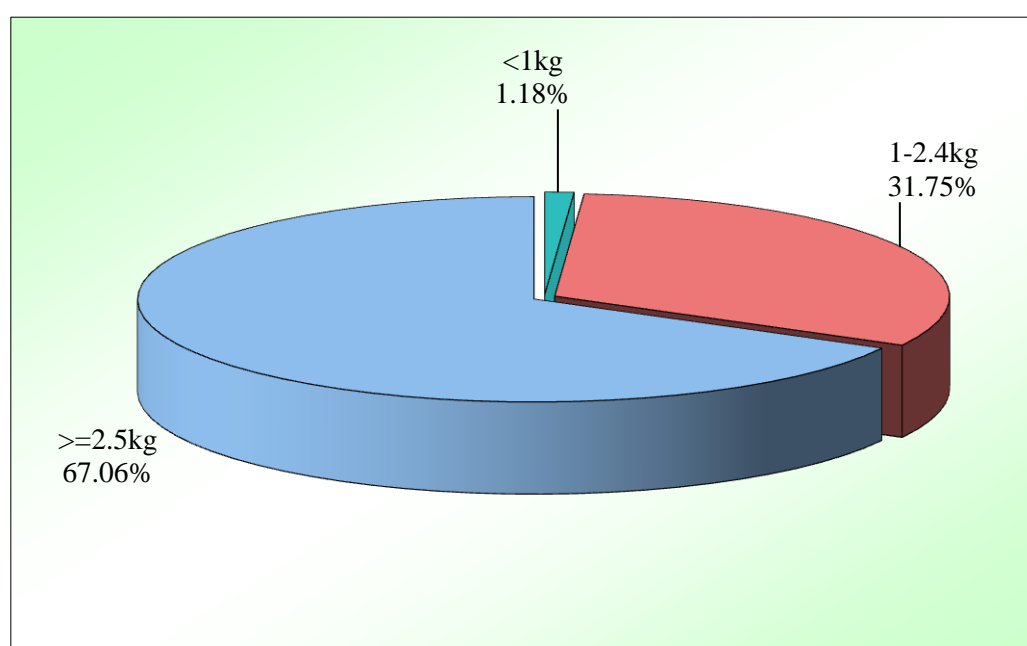
Graph 12: Birth history- Birth weight of baby

Table 13: Comparison of Birth weight of baby with Serum 17-hydroxyprogesterone levels (ng/ml) in newborns by one way ANOVA test

Birth weight of baby	Min	Max	Mean	SD	F-test	p-value
<1kg	7.00	12.00	8.66	2.04	1.4635	0.2326
1-2.4kg	4.00	25.00	9.21	3.87		
≥ 2.5 kg	1.50	18.00	8.66	2.59		
Total	1.50	25.00	8.84	3.06		

Mean serum 17-hydroxyprogesterone levels showed slight variation across different birth weight categories. Newborns weighing 1–2.4 kg had the highest mean levels (Mean = 9.21, SD = 3.87), followed by those weighing <1 kg (Mean = 8.66, SD = 2.04) and those weighing ≥ 2.5 kg (Mean = 8.66, SD = 2.59).

The difference in serum 17-hydroxyprogesterone levels in newborns across birth weight categories not statistically significant (F = 1.464, p=0.233).

Graph 13: Comparison of Birth weight of baby with Serum 17-hydroxyprogesterone levels (ng/ml)

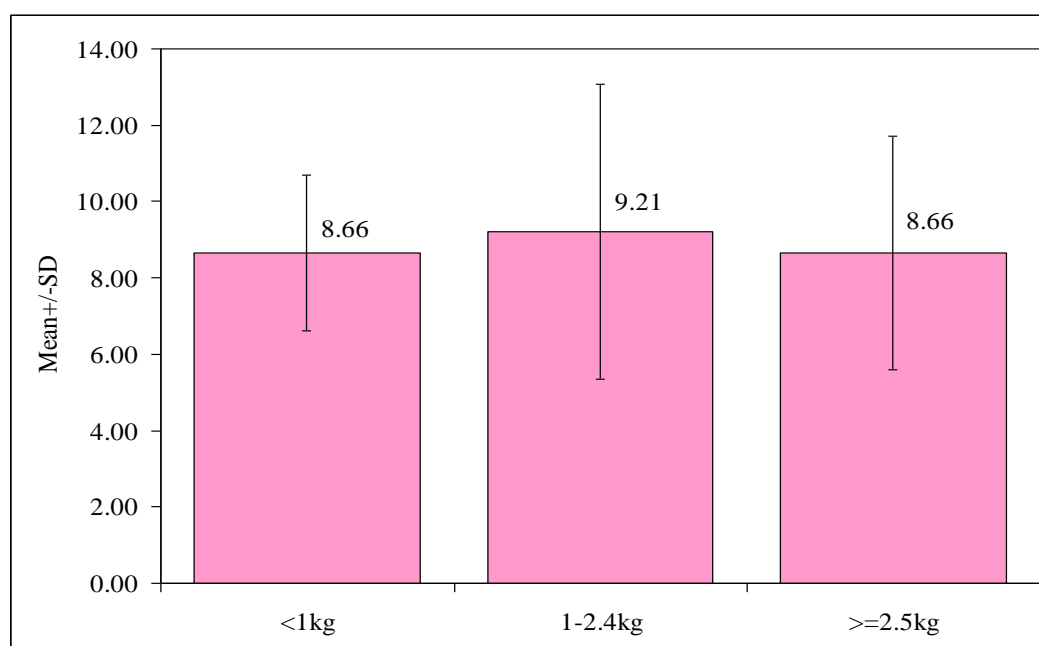


Table 14: Comparison of APGAR scores at 1 minute, 5 minute, and 10 minutes by dependent t test

APGAR at	Mean	Std.Dv.	Mean Diff.	Std.Dv. Diff.	t-value	p-value
1 minute	7.08	1.03				
5 minutes	8.24	0.96	-1.16	0.78	-30.5483	0.0001*
1minute	7.08	1.03				
10minutes	9.24	0.81	-2.16	0.85	-52.2503	0.0001*
5minutes	8.24	0.96				
10minutes	9.24	0.81	-1.00	0.54	-37.6268	0.0001*

*p<0.05

Graph 14: Comparison of APGAR scores at 1 minute, 5 minute, and 10 minutes

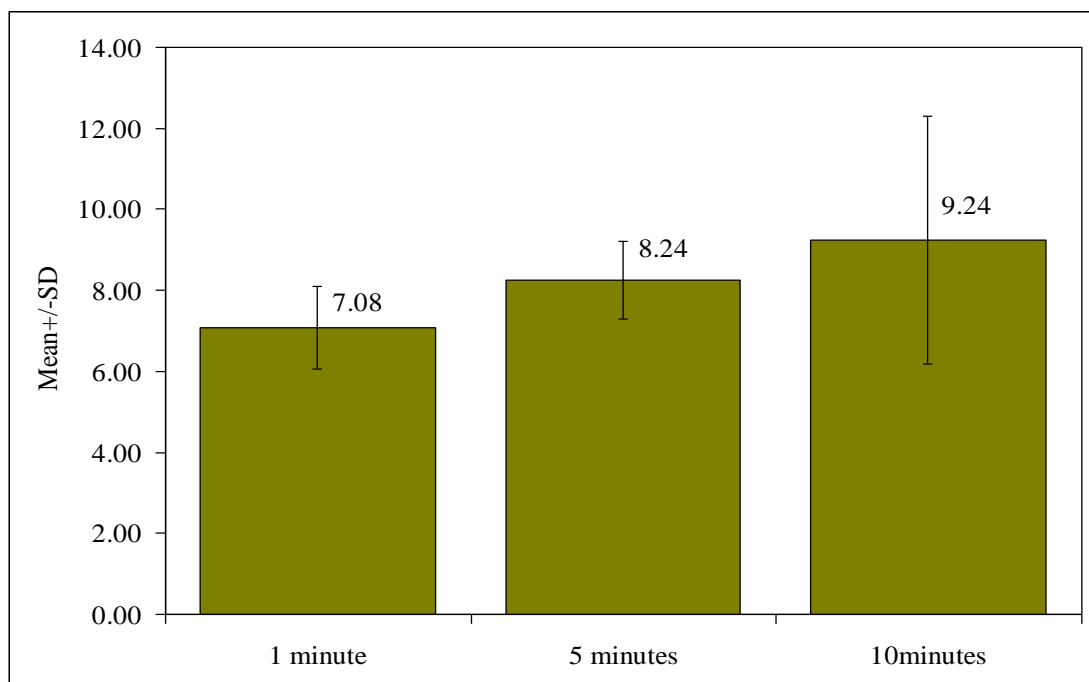


Table 15: Maternal complications

Maternal factors	No of mothers	% of mothers
Anaemia	9	2.13
Diabetes Mellitus	3	0.71
Epilepsy	3	0.71
Gestational diabetes mellitus	16	3.79
Gestational thrombocytopenia	1	0.24
Hypertension	5	1.18
Hypothyroidism	87	20.62
Oligohydramnios	2	0.47
Polyhydramnios/Oligohydramnios	9	2.13
Pregnancy Induced Hypertension	2	0.47
Pregnancy Induced hypertension/Eclampsia	14	3.32
PROM	33	7.82

The distribution of maternal complications in the study sample (N = 422) showed that the most common condition was hypothyroidism, affecting 87 (20.62%) mothers. Other notable complications included premature rupture of membranes (PROM) in 33 (7.82%) mothers and gestational diabetes mellitus in 16 (3.79%). Pregnancy-induced hypertension(PIH), either alone or with eclampsia, was observed in 16 (3.79%) mothers. Anaemia and polyhydramnios/oligohydramnios were each present in 9 (2.13%) mothers, while hypertension was reported in 5 (1.18%). Less

frequently observed conditions included oligohydramnios, gestational thrombocytopenia, and PIH, each affecting fewer than 1% of the study population.

These findings highlight hypothyroidism as the most prevalent maternal complication, with a relatively low occurrence of other conditions.

Graph 15: Maternal complications

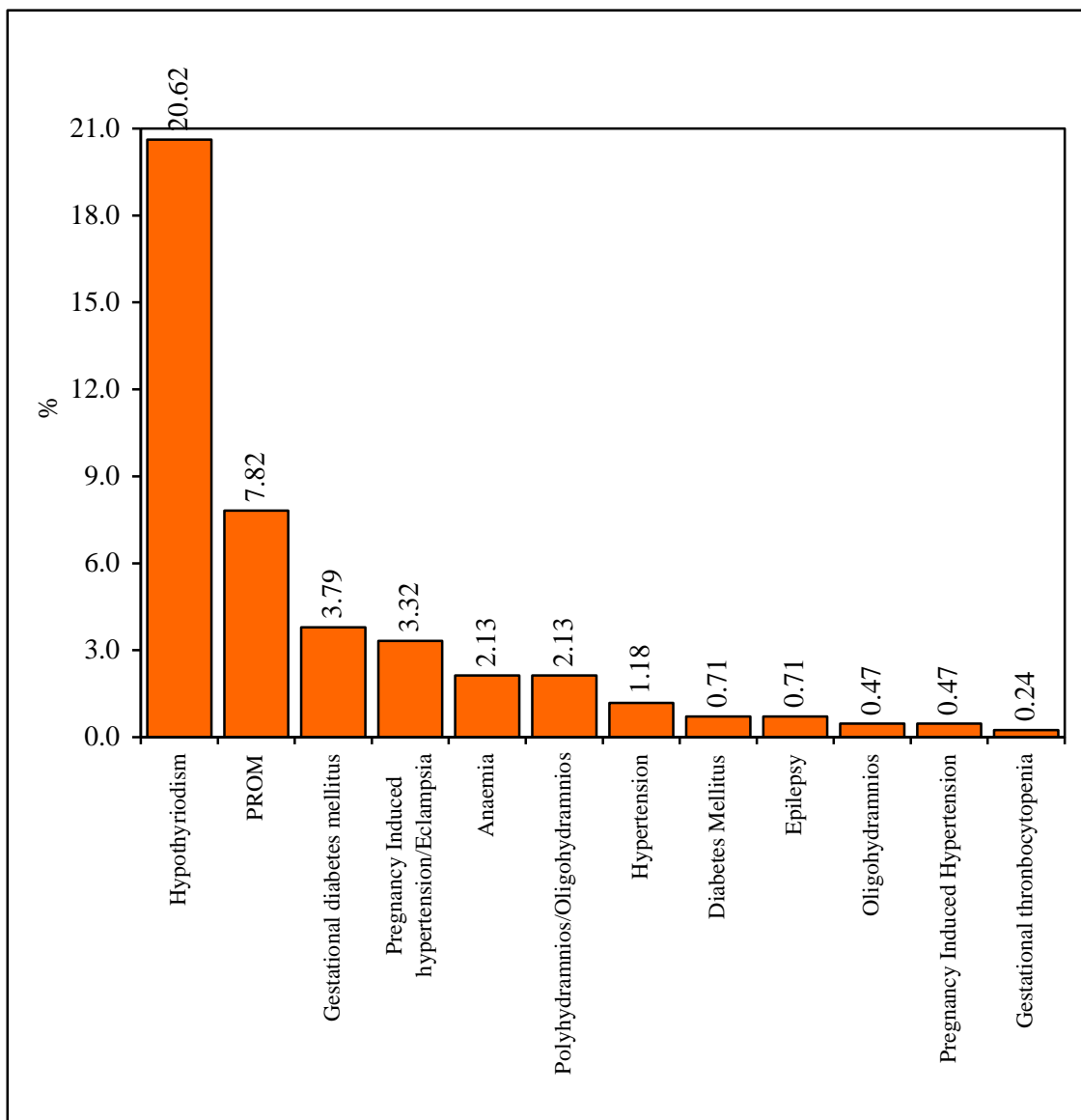


Table 16: Summary of Serum 17-hydroxyprogesterone levels (ng/ml) according to maternal factors

Maternal factors	n	Min	Max	Mean	SD
Anaemia	9	4.60	11.20	8.52	2.01
Diabetes Mellitus	3	5.80	10.10	8.47	2.33
Epilepsy	3	8.00	14.00	10.00	3.46
Gestational diabetes mellitus	16	4.90	16.00	9.45	3.33
Gestational thrombocytopenia	1	7.60	7.60	7.60	0.00
Hypertension	5	7.60	10.20	8.56	1.18
Hypothyroidism	87	4.10	14.00	9.21	2.24
Oligohydramnios	2	10.00	12.00	11.00	1.41
Polyhydramnios/Oligohydramnios	9	4.80	13.00	7.46	2.95
Pregnancy Induced Hypertension	2	10.50	18.00	14.25	5.30
Pregnancy Induced hypertension/Eclampsia	14	6.40	18.30	9.85	3.16
PROM	33	4.30	19.50	10.06	4.10

Table 17: Status of presence of perinatal factors

Perinatal factors	Yes	%	No	%
Pregnancy induced hypertension	16	3.79	406	96.21
Prom	33	7.82	389	92.18
Gestational diabetes mellitus	16	3.79	406	96.21
Hypothyroidism	88	20.85	334	79.15
Anaemia	11	2.61	411	97.39
Polyhydramnios/ oligohydramnios	12	2.84	410	97.16
Diabetes mellitus	3	0.71	419	99.29
Hypertension	5	1.18	417	98.82
Epilepsy	3	0.71	419	99.29
Antenatal medication (Steroids)	5	1.18	417	98.82

The distribution of perinatal factors in the study sample (N = 422) showed that hypothyroidism was the most prevalent condition, affecting 88 (20.85%) mothers, while 334 (79.15%) did not have this condition. Premature rupture of membranes (PROM) was reported in 33 (7.82%) mothers, with 389 (92.18%) unaffected. PIH, gestational diabetes mellitus were each observed in 16 (3.79%) mothers.

Less frequently reported factors included anaemia in 11 (2.61%) mothers, polyhydramnios/ oligohydramnios in 12 (2.84%), hypertension in 5 (1.18%), and antenatal steroid use in 5 (1.18%). Diabetes mellitus and epilepsy were the least common, each affecting 3 (0.71%) mothers.

These findings indicate that hypothyroidism was the most common perinatal factor, while other conditions were relatively rare in the study population.

Graph 16: Status of presence of perinatal factors

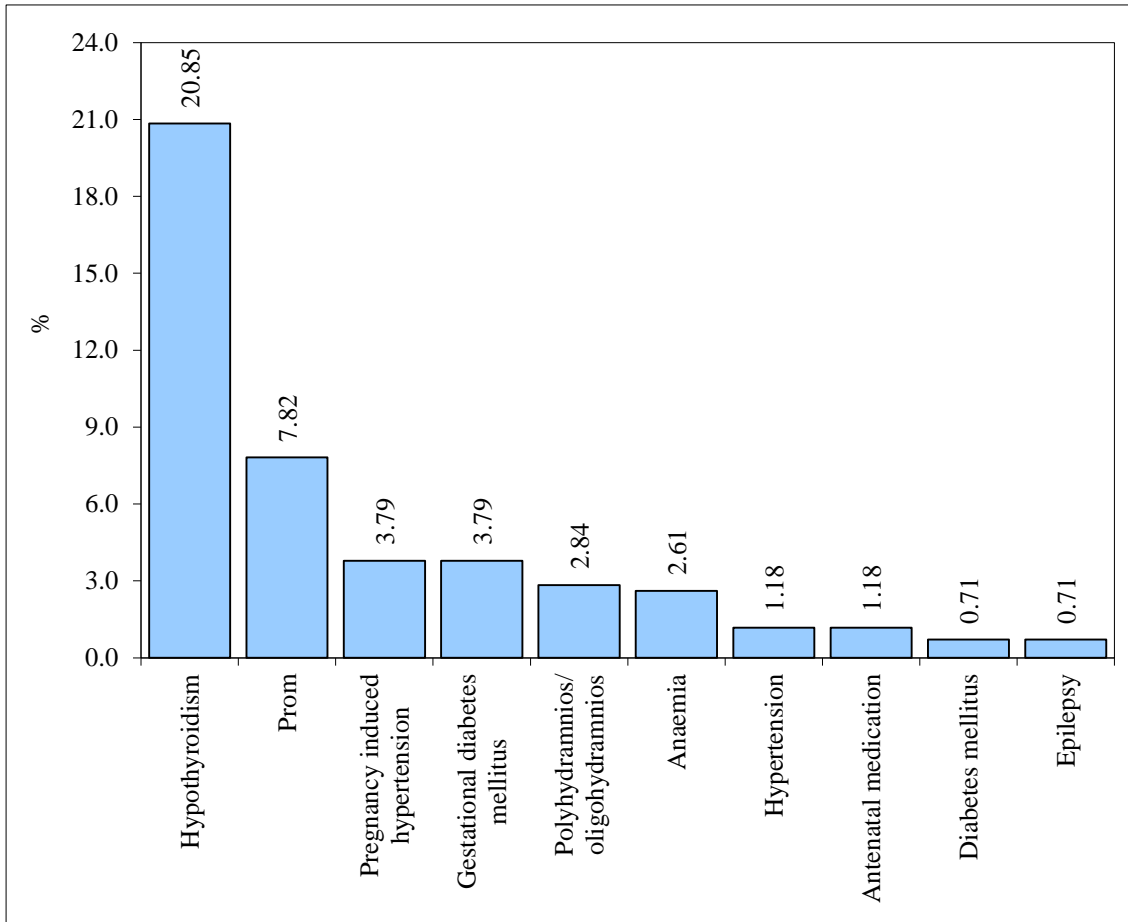


Table 18: Comparison of frequent maternal factors with Serum 17-hydroxyprogesterone levels (ng/ml) by t test

Maternal factors	Min	Max	Mean	SD	t-test	p-value
Pregnancy Induced Hypertension						
No	1.50	25.00	8.77	3.02	-2.095	0.037*
Yes	6.40	18.30	10.40	3.58		
PROM						
No	1.50	25.00	8.74	2.94	-2.260	0.024*
Yes	4.30	19.50	9.98	4.06		
Gestational Diabetes Mellitus						
No	1.50	25.00	8.81	3.05	-0.819	0.413
Yes	4.90	16.00	9.45	3.33		
Hypothyroidism						
No	1.50	25.00	8.75	3.24	-1.189	0.235
Yes	4.10	14.00	9.18	2.23		
Anaemia						
No	1.50	25.00	8.84	3.08	0.200	0.842
Yes	4.60	11.20	8.65	1.90		
Polyhydramnios/ Oligohydramnios						
No	1.50	25.00	8.86	3.06	0.894	0.372
Yes	4.80	13.00	8.06	2.90		
Gestational Hypertension						
No	1.50	25.00	8.84	3.07	0.203	0.839
Yes	7.60	10.20	8.56	1.18		
Antenatal medication (Steroids)						
No	1.50	25.00	8.79	3.01	-3.111	0.002*
Yes	7.10	18.00	13.02	4.03		
Total	1.50	25.00	8.84	3.06		

*p<0.05

Pregnancy-Induced Hypertension

The mean serum 17-hydroxyprogesterone level was significantly higher in newborns born to mothers with pregnancy-induced hypertension (Mean = 10.40, SD = 3.58) compared to those without the condition (Mean = 8.77, SD = 3.02). A *t*-test value of -2.095 ($p = 0.037$) indicated a statistically significant difference, suggesting a potential association between elevated serum 17-hydroxyprogesterone newborn levels and pregnancy-induced hypertension in mothers.

PROM

Mothers who experienced premature rupture of membranes (PROM) had a significantly higher mean serum 17-hydroxyprogesterone level (Mean = 9.98, SD = 4.06) compared to those without PROM (Mean = 8.74, SD = 2.94). The *t*-test value of -2.260 ($p = 0.024$) suggested a significant relationship between PROM in mothers and increased serum 17-hydroxyprogesterone levels in newborns.

Gestational Diabetes Mellitus

The mean serum 17-hydroxyprogesterone level was slightly higher in mothers with gestational diabetes mellitus (Mean = 9.45, SD = 3.33) compared to those without the condition (Mean = 8.81, SD = 3.05). However, difference was not statistically significant ($t = -0.819$, $p = 0.413$), indicating no strong association between gestational diabetes in mother and serum 17-hydroxyprogesterone levels in newborns.

Hypothyroidism

Mothers with hypothyroidism had a mean serum 17-hydroxyprogesterone level of 9.18 (SD = 2.23), while those without the condition had a mean of 8.75 (SD = 3.24). The *t*-test value of -1.189 ($p = 0.235$) showed no statistically significant difference, suggesting that hypothyroidism did not have a affect serum 17-hydroxyprogesterone levels in newborns.

Anaemia

The mean serum 17-hydroxyprogesterone level was similar between the newborns of mothers with anaemia (Mean = 8.65, SD = 1.90) and newborns of mothers without anaemia. (Mean = 8.84, SD = 3.08). A *t*-test value of 0.200 ($p = 0.842$) indicated no significant association between maternal anaemia and serum 17-hydroxyprogesterone levels in newborns.

Polyhydramnios/Oligohydramnios

Newborns born to mothers with polyhydramnios or oligohydramnios had a mean serum 17-hydroxyprogesterone level of 8.06 (SD = 2.90), compared to 8.86 (SD = 3.06) in those newborns born to mothers without these conditions. The *t*-test value of 0.894 ($p = 0.372$) showed no significant difference, suggesting that variations in amniotic fluid levels did not strongly correlate with serum 17-hydroxyprogesterone levels in newborns.

Gestational Hypertension

The mean serum 17-hydroxyprogesterone levels in newborn was 8.56 (SD = 1.18) born to mothers with hypertension and 8.84 (SD = 3.07) born to mothers without hypertension .A *t*-test value of 0.203 ($p = 0.839$) indicated no statistically

significant association between maternal hypertension and serum 17-hydroxyprogesterone levels in newborn.

Antenatal Medication (Steroids)

Newborns born to mothers who received antenatal steroid medication had a significantly higher mean serum 17-hydroxyprogesterone level (Mean = 13.02, SD = 4.03) compared to those newborns born to mothers who didn't receive antenatal steroids (Mean = 8.79, SD = 3.01). The *t*-test value of -3.111 ($p = 0.002$) indicated a highly significant difference, suggesting a strong association between maternal antenatal steroid use and elevated serum 17-hydroxyprogesterone levels in newborns.

Graph 17: Comparison of frequent Maternal factors with Serum 17-hydroxyprogesterone levels (ng/ml)

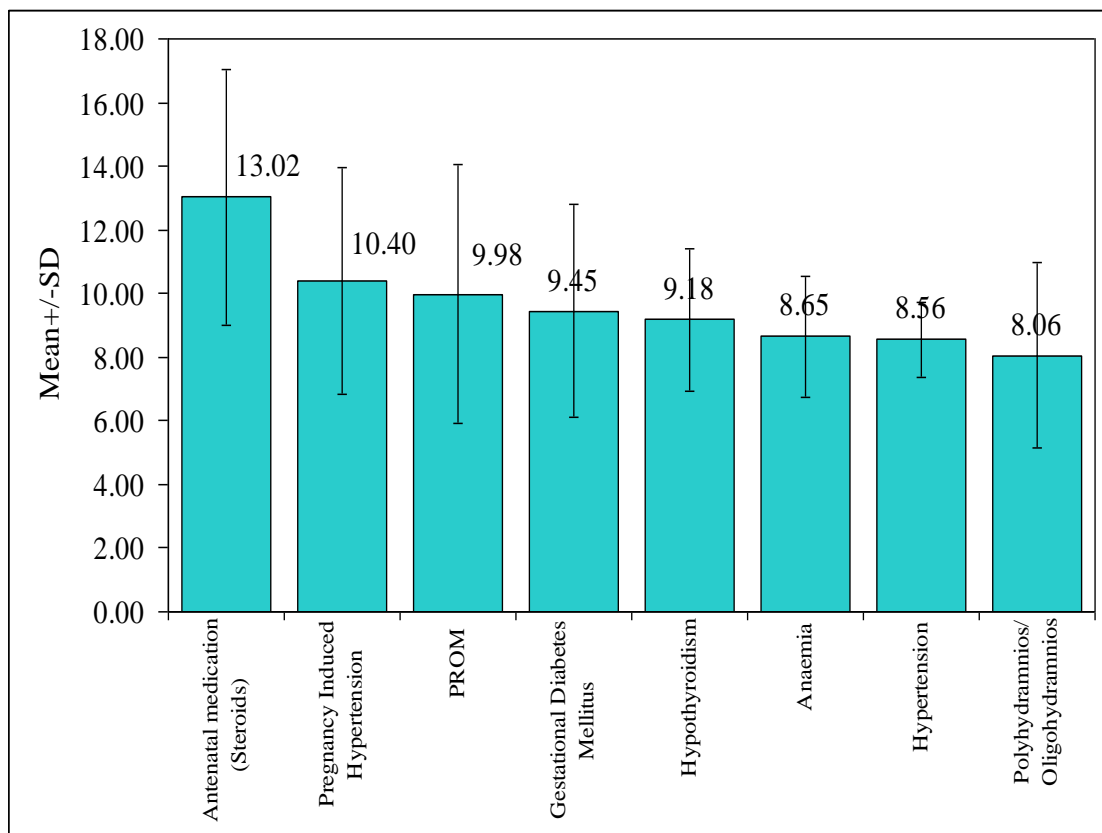


Table 19: NICU admissions

NICU admissions	No	%
No	193	45.73
Yes	229	54.27
Total	422	100.00

NICU admission was required for 229 (54.27%) newborns, while 193 (45.73%) did not require NICU care. The proportion of NICU admissions was higher, indicating a significant need for neonatal medical support in the study population.

Graph 18: NICU admissions

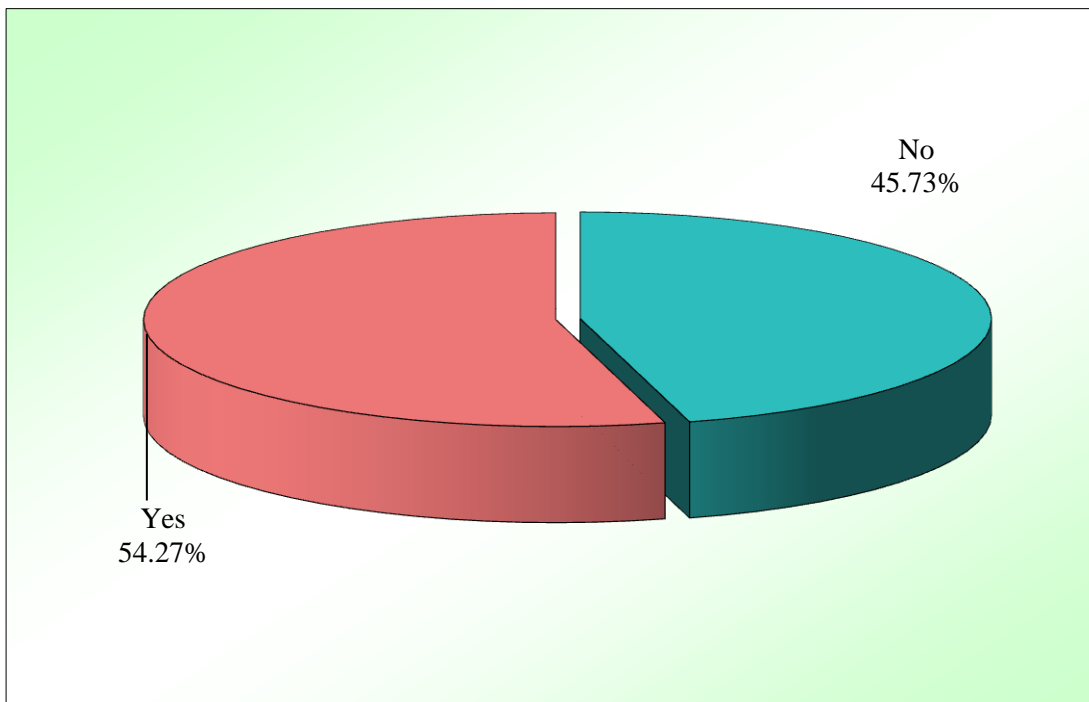


Table 20: Reasons /perinatal factors for NICU admissions

Reasons /perinatal factors	No	%
Neonatal Respiratory Distress	12	2.84
Neonatal Pneumonia	2	0.47
Meconium aspiration syndrome	4	0.95
Early onset sepsis	4	0.95
Necrotizing enterocolitis	1	0.24
Birth asphyxia	4	0.95
Neonatal seizures	0	0.00
Neonatal Hyperbilirubinemia	215	50.95
Others	3	0.71

Neonatal hyperbilirubinemia was the most common reason for NICU admission, affecting 215 (50.95%) newborns. Neonatal respiratory distress was observed in 12 (2.84%) cases, while meconium aspiration syndrome, early-onset sepsis, and birth asphyxia each accounted for 4 (0.95%) admissions. Neonatal pneumonia was reported in 2 (0.47%) cases, and necrotizing enterocolitis in 1 (0.24%) case. Other reasons contributed to 3 (0.71%) admissions, while no cases of neonatal seizures were reported in the study population.

Graph 19: Reasons for NICU admission

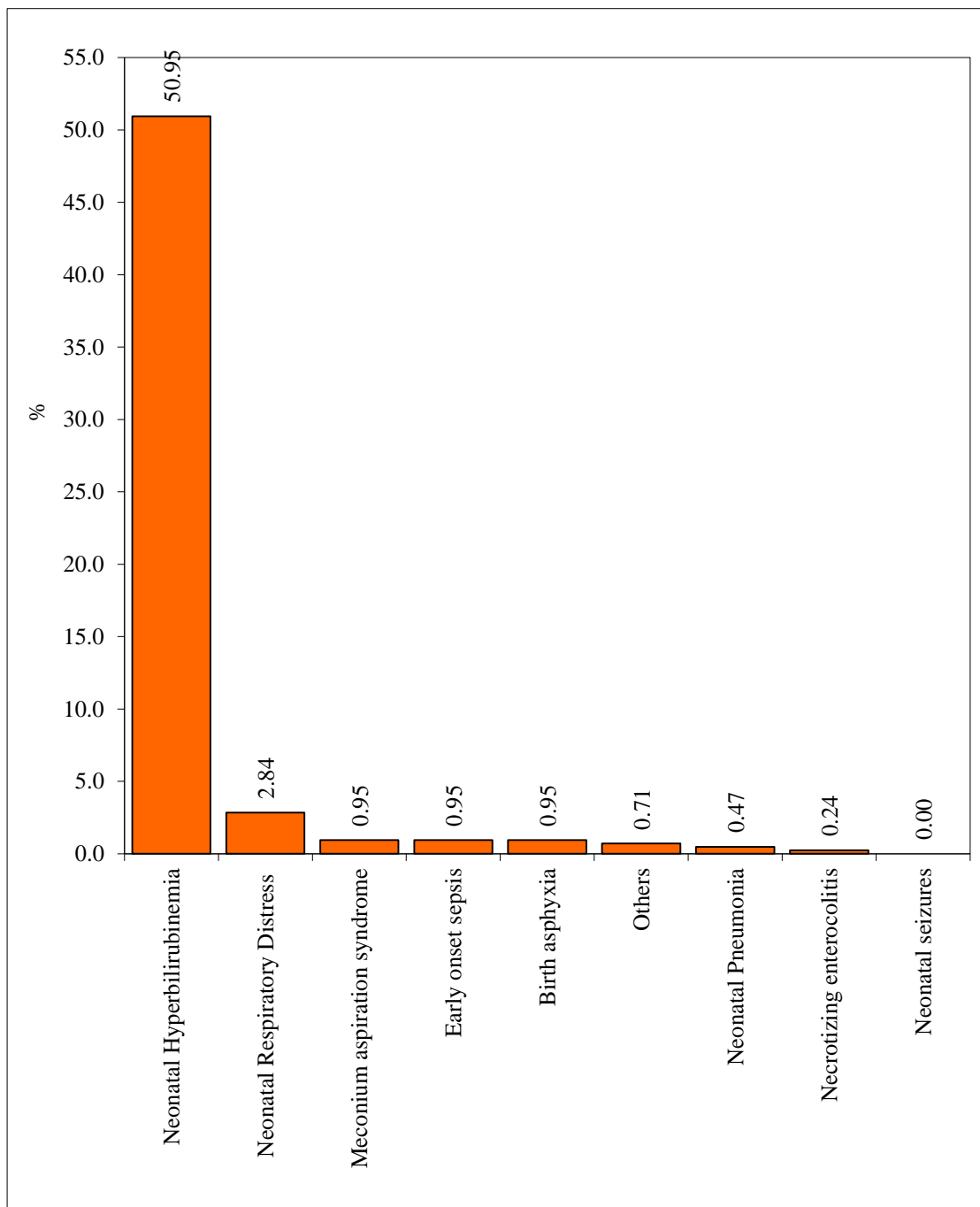


Table 21: Comparison of frequent maternal factors with Serum 17-hydroxyprogesterone levels (ng/ml) by t test

Maternal factors	Min	Max	Mean	SD	t-test	p-value
Neonatal Respiratory Distress						
No	1.50	25.00	8.69	2.90	-6.012	<0.001*
Yes	7.10	22.00	13.86	4.06		
Meconium aspiration syndrome						
No	1.50	25.00	8.78	3.01	-3.921	<0.001*
Yes	12.80	18.00	14.70	2.27		
Early onset sepsis						
No	1.50	25.00	8.79	3.01	-3.217	0.001*
Yes	8.50	19.20	13.67	4.54		
Birth asphyxia						
No	1.50	25.00	8.82	3.04	-1.127	0.260
Yes	6.40	15.10	10.55	4.49		
Neonatal Hyperbilirubinemia						
No	3.60	25.00	8.86	3.13	0.1335	0.8938
Yes	1.50	19.50	8.82	2.99		

*p<0.05

Neonatal Respiratory Distress:

Mean serum 17-hydroxyprogesterone levels are significantly higher in newborns with neonatal “respiratory distress” (Mean = 13.86, SD = 4.06) compared to those without the condition (Mean = 8.69, SD = 2.90). This difference was statistically significant ($t = -6.012$, $p < 0.001$).

Meconium Aspiration Syndrome:

Newborns with meconium aspiration syndrome had significantly higher serum 17-hydroxyprogesterone levels (Mean = 14.70, SD = 2.27) than those without the condition (Mean = 8.78, SD = 3.01). This difference was statistically significant ($t = -3.921$, $p < 0.001$).

Early-Onset Sepsis:

Serum 17-hydroxyprogesterone levels were significantly higher in newborns diagnosed with early-onset sepsis (Mean = 13.67, SD = 4.54) compared to those without the condition (Mean = 8.79, SD = 3.01). This difference was statistically significant ($t = -3.217$, $p = 0.001$).

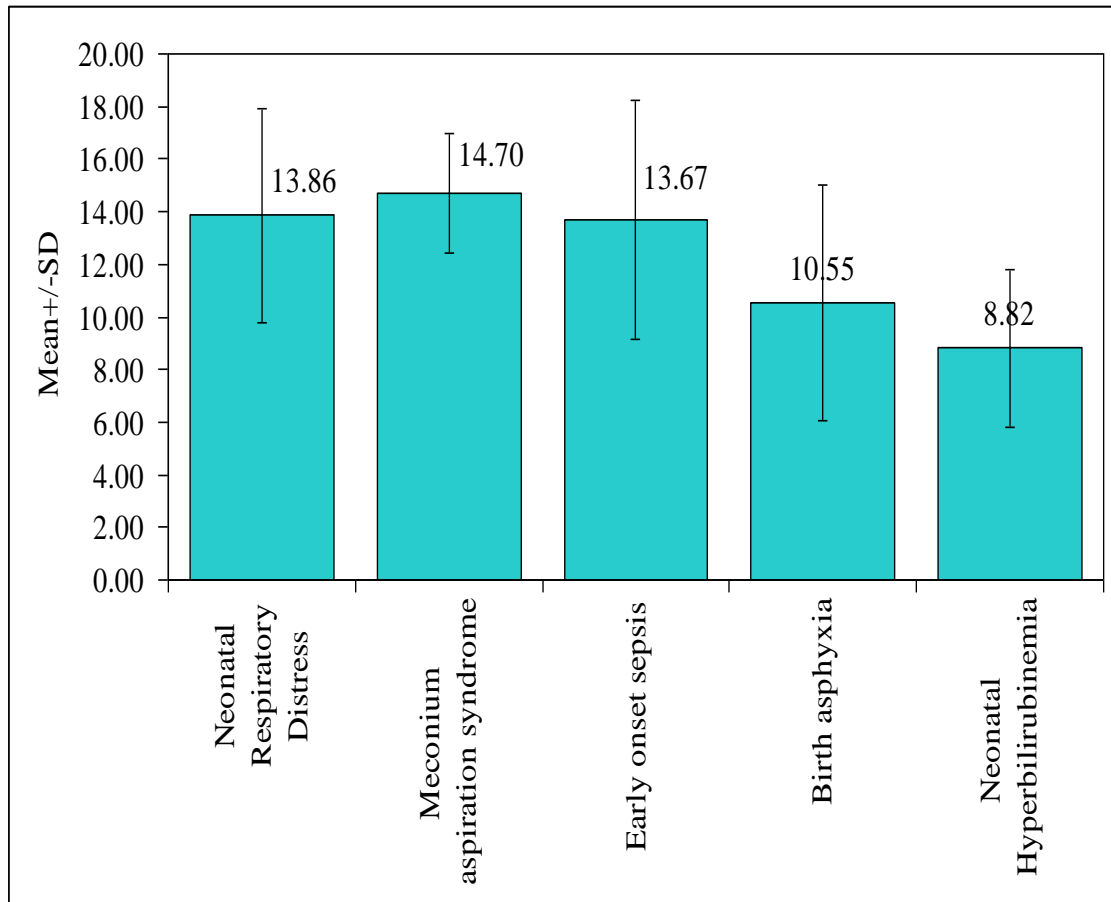
Birth Asphyxia:

Newborns with birth asphyxia had higher serum 17-hydroxyprogesterone levels (Mean = 10.55, SD = 4.49) than those without the condition (Mean = 8.82, SD = 3.04), though the difference was not statistically significant ($t = -1.127$, $p = 0.260$).

Neonatal Hyperbilirubinemia:

There is no significant difference in serum 17-OHP levels between newborns with neonatal hyperbilirubinemia (Mean = 8.82, SD = 2.99) and those without the condition (Mean = 8.86, SD = 3.13). The statistical test showed no significant association ($t = 0.134$, $p = 0.894$).

Graph 20: Comparison of neonatal factors with Serum 17-hydroxyprogesterone levels (ng/ml)



DISCUSSION

17-Hydroxyprogesterone (17-OHP) is a steroid hormone that serves as a key intermediate in the biosynthesis of glucocorticoids and androgens. It is primarily synthesized in the adrenal glands and, to a lesser extent, in the gonads and placenta. In neonates, 17-OHP levels are commonly measured as part of newborn screening programs to detect congenital adrenal hyperplasia (CAH), an autosomal recessive disorder characterized by enzyme deficiencies, most commonly 21-hydroxylase deficiency, leading to cortisol deficiency and androgen excess.

Several maternal and neonatal factors can influence 17-OHP levels in newborns. Maternal factors such as gestational age at delivery, maternal stress, pregnancy complications (such as preeclampsia and gestational diabetes), and antenatal steroid administration can affect foetal adrenal steroidogenesis and consequently alter neonatal 17-OHP levels. Neonatal factors, including prematurity, birth weight, mode of delivery, and perinatal stress, also play a crucial role in determining 17-OHP levels. Premature infants often exhibit elevated 17-OHP levels due to immaturity of the hypothalamic-pituitary-adrenal (HPA) axis and decreased enzymatic activity.

- Thereby, the significance of 17-OHP in neonatal health, this study aims to analyse how maternal and neonatal factors influence its levels in newborns delivered at a tertiary care hospital.
- Recall rate for confirmatory test was found to be nil, as none of the 17-OHP levels were found to be above the cut off values for the same.

❖ **Age wise distribution of mothers:**

In the present study, the majority of mothers in the study 46.92% were aged 26-30 years, followed by 33.65% in the 21-25 years group. Mothers aged ≥ 31 years constituted 15.64%, while the youngest group ≤ 20 years accounted for only 3.79%. The mean maternal age was 26.78 years, with a standard deviation of 3.75, demonstrating a relatively young population with moderate variability in age distribution.

❖ **Gravida wise distribution of mothers:**

In the current study, primigravida mothers constituted the majority, accounting for 52.84% (n=223), while multigravida mothers comprised 47.16% (n=199). Additionally, a history of abortion was observed in 56 cases (13.27%). In our study, P value was 0.803 and is not statistically significant .

Table 22 : Gravida wise distribution of mothers

Sr. No.	Studies	Gravida	No. of cases
1	The present study	Primigravida	223 (52.84%)
		Multigravida	199 (47.16%)
		More than three times	13 (26)
		Abortion	7 (14)

Table 23: Comparison of Gravida with Serum 17-hydroxyprogesterone levels (ng/ml) by t test

Gravida	Mean	SD	p-value
Primigravida	8.87	3.25	0.8034
Multigravida	8.80	2.83	
Total	8.84	3.06	

❖ **Living children wise distribution of mothers:**

Our study results showed that, most mothers (30.81%) had only one living child, followed by 6.40% who had two living children, and only 0.24% who had three living children.

❖ **Current pregnancy wise distribution of mothers:**

We conducted a statistical analysis, and on a cohort of 422 pregnancies, it was observed that the majority, 404 cases, were conceived spontaneously, whereas 18 pregnancies resulted from assisted reproductive techniques.

❖ **Comparison of Conception method with Serum 17-hydroxyprogesterone levels (ng/ml) by t test:**

We did the comparison of serum 17-OHP levels (ng/ml) between women who conceived through assisted reproductive techniques (ART) and those who conceived spontaneously. The mean 17-hydroxyprogesterone levels in the ART group was 9.52 ± 3.02 ng/ml, ranging from 5.80 to 18.30 ng/ml. In contrast, the spontaneous conception group had a mean 17-OHP level of 8.81 ± 3.06 ng/ml, with values ranging from 1.50 to 25.00 ng/ml. The overall mean 17-OHP level for all participants was 8.84 ± 3.06 ng/ml. Statistical analysis using the t-test yielded a t-value of 0.973 and a

p-value of 0.331, demonstrating no statistically significant difference in serum 17-OHP levels between the two conception methods. Thus, the mode of conception does not appear to have a significant impact on serum 17-OHP levels in the studied population. According to some studies such as White PC ⁽³⁴⁾ “Assisted reproductive technologies” (ART), such as “in vitro fertilization” (IVF) and “intracytoplasmic sperm injection” (ICSI), have been associated with certain perinatal outcomes, including an increased incidence of preterm birth and low birth weight. These factors can influence neonatal 17-hydroxyprogesterone (17-OHP) levels indirectly.

Table 24: Method of conceptions with mean 17 OHP levels

Current pregnancy	Mean	SD	t-test	p-value
Assisted Reproduction Technique	9.52	3.02	0.9733	0.3310
Spontaneous	8.81	3.06		
Total	8.84	3.06		

❖ **Birth history- Period of Gestational Age:**

In this study, we noted the gestational age of the study participants was 37.65 ±1.98 weeks.

Table 25: Comparision of newborn gestational age in our study and Anandi VS et al study

Sr. No.	Studies	Gestational age (Mean ± SD)	No. of cases
1.	The present study	37.65 ±1.98	422
2.	Anandi VS et al., (2017)	36.58 ± 1.88	3080

In the present study, the mean gestational age of the participants was 37.65 ± 1.98 weeks among 422 cases. In comparison, Anandi VS et al., (2017)⁽³⁸⁾ reported, a mean gestational age of 36.58 ± 1.88 weeks. These findings revealed that, the variability in gestational age across different studies, likely influenced by differences in study populations, inclusion criteria and obstetric factors.

❖ Comparison of Period of Gestational Age with Serum 17-hydroxyprogesterone levels (ng/ml) in newborns by one way ANOVA test:

Our study demonstrates a mean value across the three gestational age groups, with the highest mean observed in newborns ≤ 32 weeks (9.92 ± 4.03). This value progressively declines as gestational age advances, measuring 9.48 ± 3.96 in the 33–36 weeks group and further decreasing to 8.66 ± 2.76 in newborns ≥ 37 weeks. Notably, the highest variability was recorded in the ≤ 32 weeks group. Although the p-value (0.0540) is marginally above the conventional threshold for statistical significance ($p < 0.05$), it suggests a potential difference that may reach significance with a larger sample size or further analytical refinement.

These findings align with previous studies by **Chennuri VS, et al., (2013)⁽³⁹⁾** and **al-Nuaim AR, et al., (1995)⁽⁴⁰⁾**, both of which reported significantly elevated 17-hydroxyprogesterone (17-OHP) levels in neonates with lower birth weight and lower gestational age ($p < 0.001$) compared to those with normal birth weight and term gestation. Similar observations were documented in a multicentric study by **Zhang Q, et al., (2014)⁽⁴¹⁾**, which emphasized the necessity of adjusting neonatal 17-OHP cutoff values based on both gestational age and birth weight to enhance diagnostic accuracy.

Furthermore, **Ryckman KK, et al., (2012)** ⁽⁴²⁾ explored the impact of multiple factors on neonatal 17-OHP concentrations in preterm infants, demonstrating that both gestational age and birth weight significantly influenced 17-OHP variability. These collective findings emphasize the complex relationship between gestational maturity, neonatal weight, and hormonal levels, highlighting the need for gestational age-specific reference ranges in clinical practice.

Table 26: Gestational age and mean 17 OHP levels of newborn with p- value

Period of Gestational Age	Mean	p-value
<=32weeks	9.92	0.0540
33-36weeks	9.48	
>=37weeks	8.66	
Total	8.84	

❖ **Birth history- Gender of baby:**

When we analysed the gender of newborns in the present study, we found (n=248) 58.77% were male newborns and 174 (41.23%) were female newborns. This indicates that there is no significant difference in 17-OHP values between females and males.

Table 27: Comparison of mean of serum 17-hydroxyprogesterone levels (ng/ml) between gender (N=422)

Parameter	Gender (Mean± SD)		P value
	Male (N=248)	Female (N=174)	
Serum 17-hydroxyprogesterone levels (ng/ml)	8.86 ± 3.15	8.81 ± 2.93	0.879

The present study aligns with the finding of Anandi VS et al., (2017)⁽³⁸⁾ who reported - no significant differences in 17-OHP values between females and males.

❖ Comparison of Birth weight of baby with Serum 17-hydroxyprogesterone levels (ng/ml) in newborns by one way ANOVA test:

The comparison of serum 17-OHP levels (ng/mL) across different birth weight categories among neonates was conducted using a one-way ANOVA test. The highest mean 17-OHP levels were observed in neonates weighing between 1–2.4 kg (9.21 ± 3.87 ng/mL), while those in the <1 kg and ≥ 2.5 kg birth weight groups exhibited lower mean levels (8.66 ± 2.04 ng/mL and 8.66 ± 2.59 ng/mL, respectively). Although, variations were noted among the groups, the ANOVA test didn't demonstrate statistical significance ($F = 1.4635$, $p = 0.2326$), demonstrating no significant association between serum 17-OHP levels and birth weight in this study population. Therefore, we recommended that, further investigations with a larger sample size to validate the present study findings.

The present study aligns with the findings of Atarod Z, et al. (2020)⁽³⁰⁾, who reported no significant differences between case and control groups in terms of

preterm labor (PTL) risk at less than 35 and 37 weeks of gestation when analysed separately by gestational age and birth weight.

In contrast, **Ryckman KK, et al., (2012)⁽⁴²⁾** highlighted that, gestational age and birth weight were the most influential factors contributing to variations in 17-OHP concentrations. This contrasts with the present study, where no significant association between 17-OHP levels and birth weight was observed.

The inverse relationship between birth weight and serum 17-OHP levels suggests that lower birth weight neonates are more likely to have elevated 17-OHP concentrations. This elevation can lead to a higher rate of false-positive results in CAH newborn screening. Therefore, adjusting 17-OHP cutoff values based on birth weight and gestational age is recommended. The study by **Anandi and Bhattacharyya (2017)⁽³⁸⁾** evaluated the effect of various maternal and neonatal factors on newborn **17-hydroxyprogesterone (17-OHP) levels**. Their findings indicate a significant **inverse relationship between birth weight and 17-OHP levels**. Neonates with **lower birth weight (<1.5 kg)** exhibited the highest **mean 17-OHP levels (8.97 ± 7.43 ng/mL)**, whereas those in the **1.5–2.5 kg category** had a mean level of **5.65 ± 3.87 ng/mL**. In contrast, neonates with **normal birth weight (2.5–4 kg)** demonstrated significantly lower **mean 17-OHP levels (4.86 ± 2.47 ng/mL)**. The study found no significant difference in 17-OHP levels between the **2.5–4 kg and >4 kg birth weight groups**. This suggests that **preterm and low birth weight infants are at a higher risk of elevated 17-OHP**, potentially leading to false-positive results in **newborn screening for congenital adrenal hyperplasia (CAH)**.

Table 28: Comparison of Birth weight of baby with Serum 17-hydroxyprogesterone levels in our study.

Birth weight of baby	Mean
<1kg	8.66
1-2.4kg	9.21
>=2.5kg	8.66
Total	8.84

Table 29: Comparison of Birth weight of baby with Serum 17-hydroxyprogesterone levels in study by Anandi and Bhattacharyya (2017) ⁽³⁸⁾.

Birth Weight Category (kg)	Mean 17-OHP Level (ng/mL)
<1.5 kg	8.97
1.5-2.5	5.65
2.5-4	4.86

❖ Comparison of frequent maternal factors with Serum 17-hydroxyprogesterone levels (ng/ml) by t test:

The comparison of maternal factors with serum 17-OHP levels using the t-test revealed significant associations with PIH, Premature rupture of membranes(PROM) and antenatal steroid administration. Newborns born to women with PIH exhibited significantly higher mean serum 17-OHP levels (10.40 ± 3.58 ng/ml) compared to

those newborn born to mothers without PIH (8.77 ± 3.02 ng/ml) ($p = 0.037$). Similarly, newborns born to mothers with PROM was associated with elevated 17-OHP levels (9.98 ± 4.06 ng/ml) versus newborns born to mothers with no PROM cases (8.74 ± 2.94 ng/ml) ($p = 0.024$). The more pronounced difference observed in newborns born to women receiving antenatal steroids, where the mean 17-OHP levels (13.02 ± 4.03 ng/ml) were significantly higher than those newborn born to mother who had not received steroids (8.79 ± 3.01 ng/ml) ($p = 0.002$). In contrast, GDM, hypothyroidism, anaemia, polyhydramnios/oligohydramnios, and gestational hypertension did not show statistically significant differences in 17-OHP levels ($p > 0.05$). These findings suggest that PIH, PROM, and antenatal steroid use are associated with increased serum 17-OHP levels, potentially indicating their influence on hormonal regulation in newborns. This was supported by **Anandi SV, et al.**, in **2017** ⁽³⁸⁾ who reported that, among term babies, stress factors like PIH ,EOS significantly increase the 17-OHP levels.

To the best of our knowledge, this study represents the investigation in India to examine the association between serum 17-OHP levels and maternal conditions, including PIH, PROM, and antenatal steroid administration. Although, 17-OHP plays a crucial role in foetal adrenal steroidogenesis and pregnancy maintenance, its relationship with these maternal factors remains not documented well in the literature. Some previous research work, has predominantly focused on 17-OHP in the setting of congenital adrenal hyperplasia and preterm birth risk, with limited evidence addressing its potential role .

The present findings demonstrate that, a significant association between elevated 17-OHP levels and PIH, PROM and antenatal steroid exposure, present novel understandings into hormonal alterations in these conditions. As the first study

to report these associations in an Indian cohort, these results emphasize, the need for further largescale, multi-centre studies in order to confirm these findings and elucidate there, clinical implications in interpretation of 17 -OHP levels in newborn screening.

❖ **NICU admissions:**

This study included 422 neonates, of which 229 (54.27%) had NICU admissions, while 193 (45.73%) were without NICU care. This was similar to the findings of **Pauwels G, et al., (2012)**⁽⁴⁴⁾, who conducted a case-control study among 91 newborns. They reported that, these 91 newborns represented 7.1% of all children admitted to the NICU during the two-year study period.

❖ **Reasons /perinatal factors for NICU admissions:**

Neonatal hyperbilirubinemia was the most prevalent perinatal condition, affecting 50.95% of cases, followed by neonatal respiratory distress (2.84%) and birth asphyxia (0.95%). Other conditions, including meconium aspiration syndrome, early-onset sepsis, and necrotizing enterocolitis, were observed at lower frequencies. Notably, neonatal seizures were absent. The high prevalence of neonatal hyperbilirubinemia and respiratory distress suggests potential maternal influences, including hormonal imbalances. fetal stress responses and impaired neonatal adaptation, may contribute to Elevated neonatal serum 17-hydroxyprogesterone levels necessitating further investigation.

❖ **Comparison of frequent neonatal factors with Serum 17-hydroxyprogesterone levels (ng/ml) by t test:**

The present study evaluates the association between newborn 17-OHP levels and various neonatal conditions using t-tests for mean value comparisons. The

findings present a significant association between 17-OHP levels and neonatal respiratory distress, as neonates without respiratory distress had a significantly lower mean value (8.69 ± 2.90) compared to those with respiratory distress (13.86 ± 4.06), with a highly significant p-value (<0.001). Similarly, a statistically significant difference was observed in neonates with and without meconium aspiration syndrome, with those affected exhibiting a higher mean value (14.70 ± 2.27) than their unaffected counterparts (8.78 ± 3.01) ($p < 0.001$). Early onset sepsis also showed a significant association with 17-OHP as neonates with the condition had a higher mean value (13.67 ± 4.54) than those without (8.79 ± 3.01) ($p = 0.001$). However, no statistically significant difference was noted in neonates with and without birth asphyxia ($p = 0.260$), suggesting that 17-OHP levels were not influenced by conditions like birth asphyxia. Similarly, neonatal hyperbilirubinemia did not show a significant association, as the mean values were comparable between affected and unaffected neonates ($p = 0.8938$). A study by **Pauwels et al.** ⁽⁴⁴⁾ demonstrated, a significant correlation between elevated 17-OHP levels and prolonged respiratory support duration ($p < 0.0001$).

The present study was first to provide novel understandings into the role of maternal factors and neonatal factors influencing 17-OHP levels in newborn, particularly 17-OHP levels of newborn in association with neonatal respiratory distress, meconium aspiration syndrome, and early onset sepsis. The findings emphasized that, the necessity for **adjusted 17-OHP cutoff values** based on **gestational age, birth weight, and neonatal stress factors** to **reduce false-positive results in CAH screening programs**. However, no significant correlation was found with birth asphyxia and neonatal hyperbilirubinemia. As

this is the first study to be done in Indian sub-continent, presenting these findings, further research with larger sample sizes and multi-center validation is required to confirm these associations and explore the underlying mechanisms.

CONCLUSION

The present study highlighted the maternal and neonatal factors influencing 17-OHP levels in newborns delivered at a tertiary care hospital. The findings demonstrate that, maternal gravida status, conception method, and perinatal factors play a role in determining neonatal 17-OHP levels. The present study revealed, that a majority of mothers were aged between 26-30 years, and primigravida cases were more common. Spontaneous conception was the predominant method, with only a small proportion of pregnancies resulting from assisted reproductive techniques. While statistical analysis did not show a significant difference in serum 17-OHP levels between spontaneous and ART-conceived pregnancies, it emphasizes, the complex interaction of maternal and neonatal factors in adrenal steroidogenesis.

The present study sheds light on, maternal and neonatal factors influencing 17-OHP levels in newborns delivered at a tertiary care hospital. As the first study in the literature to examine these associations comprehensively, our findings contribute to the understanding of perinatal hormonal regulation and its implications for neonatal health. Our study highlights significant maternal factors, such as gestational age, maternal health conditions like pregnancy induced hypertension, PROM, antenatal steroids, mode of conception as well as neonatal parameters, like respiratory distress, early onset sepsis, meconium aspiration syndrome, birth weight that may influence 17-OHP levels in newborns. These results underscore the importance of screening and early identification of neonates at risk for adrenal dysfunction or congenital adrenal hyperplasia. Further research with larger cohorts and multicentric studies is warranted to validate these findings and explore their long-term clinical relevance.

SUMMARY

- In present study, total of 427 newborns were included on the basis of above-mentioned predefined inclusion and exclusion criteria.
- The mean maternal age was 26.78 years, with the majority aged 26–30 years.
- Primigravida mothers slightly outnumbered multigravida mothers.
- The mean neonatal serum 17-OHP level was 8.84 ng/ml.
- No significant difference in 17-OHP levels of newborn was seen between newborns born to primigravida and multigravida mothers.
- Assisted Reproduction Technique (ART) pregnancies (4.27%) had slightly higher mean 17-OHP levels compared to spontaneous pregnancies. However, this difference was not statistically significant ($p = 0.331$).
- Majority of births occurred at ≥ 37 weeks.
- Mean 17-OHP levels were highest in preterm newborns and lowest in term newborns
- The difference in 17-OHP levels across gestational age groups was borderline significant
- The analysis of serum 17-hydroxyprogesterone levels across different gestational age groups showed a decreasing trend with increasing gestational age.
- Newborns born at ≤ 32 weeks had the highest mean serum 17-hydroxyprogesterone levels
- Newborns born between 33–36 weeks had slightly lower mean levels
- The lowest mean levels were observed in newborns born at ≥ 37 weeks
- The variation in serum 17-hydroxyprogesterone levels across these gestational age groups approached statistical significance.

- The study sample consisted of 422 newborns, with a greater proportion of males compared to females.
- Birth weight analysis showed that most newborns had birth weight of ≥ 2.5 kg. A total of 31.75% of newborns weighed between 1–2.4 kg. Only 1.18% of newborns had a birth weight of less than 1 kg.
- Mean birth weight was 2.57 kg, indicating that most newborns had a normal birth weight.
- Comparison of serum 17-hydroxyprogesterone levels across birth weight categories revealed minor variations.
- Newborns weighing 1–2.4 kg had the highest mean levels.
- Newborns in the <1 kg and ≥ 2.5 kg categories had identical mean levels of 8.66 ng/ml
- These differences in serum 17-hydroxyprogesterone levels across birth weight categories were not statistically significant.
- Hypothyroidism was the most prevalent maternal complication, affecting 20.62% of mothers.
- Premature rupture of membranes (PROM) was observed in 7.82% of cases.
- Gestational diabetes mellitus was present in 3.79% of mothers.
- Pregnancy-induced hypertension, either alone or with eclampsia, was also observed in 3.79% of cases.
- Anemia and polyhydramnios/oligohydramnios were each reported in 2.13% of cases.
- Hypertension was present in 1.18% of mothers.
- Serum 17-hydroxyprogesterone levels of newborns varied across maternal conditions.

- The highest mean levels were observed in neonates of mothers with pregnancy-induced hypertension
- Neonates of mothers with pregnancy-induced hypertension/eclampsia had a mean level of 9.85 ng/ml
- Hypothyroidism was associated with a mean level of 9.21 ng/ml
- Other maternal conditions, including anemia, diabetes mellitus, and hypertension, showed minor variations in mean serum 17-hydroxyprogesterone levels of newborn.
- No significant pattern was evident across maternal complications regarding serum 17-hydroxyprogesterone levels.
- Overall, the findings suggested a trend of decreasing serum 17-hydroxyprogesterone levels with increasing gestational age and birth weight.
- However, these trends did not reach statistical significance.
- APGAR scores showed significant improvement over time, indicating better neonatal stability.
- Hypothyroidism was the most common maternal complication observed in the study.
- The present study findings provide valuable insights into the neonatal and maternal factors associated with serum 17-hydroxyprogesterone level

STRENGTHS OF THE PRESENT STUDY:

- Specific good sample size study in the present study.
- Comprehensive evaluation of maternal and neonatal parameters, ensuring a thorough analysis of factors influencing 17-OHP levels in newborns.

LIMITATIONS OF THE PRESENT STUDY:

- Limited availability of supporting literature poses challenges to validate the present study findings.
- Laboratory assessment of 17-OHP levels remains a costly procedure, especially in low- and middle-income countries, restricting its prevalent application for newborn screening programs.
- The study was conducted in a resource-limited setting, ELISA technique was used to assess the 17-OHP levels, better sensitive tests like LC MS /MS could not be done, which may impact the generalizability of the findings.

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ANNEXURES

ANNEXURE – I - PARTICIPANT INFORMATION SHEET

STATEMENT OF CONSENT

Mother IP number:

Neonate IP number:

Name of principal investigator: _____

Name of Guide: _____

Contact number: +_____

Contact Address: PG Resident, Department of Paediatrics, KAHER's J.N Medical College, Belagavi – 590010

The content of the provided information sheet has been carefully read by me/ explained to me in my vernacular language and have understood it's content. I confirm that I have had the opportunity to ask questions and have received satisfactory responses.

The nature and purpose of study and its potential risks/ benefits and expected duration of study, and relevant details of study have been explained to me in detail. I understand that the participation of my newborn in the study is entirely voluntary and that I am free to withdraw my consent at any time without the need to provide an explanation, knowing that it will not affect my my medical care or legal rights.

Signature of the subject / left thumb impression:

Name of the participant:

Place:

Date:

Name of the parent/ guardian:

Signature of parent / guardian:

This is to certify that above consent has been obtained in my presence.

Name of principal investigator: _____

Signature of principal investigator:

Name of witness:

Signature of witness:

Mother IP number:

Neonate IP number:

Name of principal investigator: _____

Contact number: _____

Contact Address: Junior resident, Department of Paediatrics, KAHER's JNMC, Belagavi

Project title: Maternal and Neonatal factors influencing 17-hydroxyprogesterone levels in newborns delivered at a tertiary care hospital.

Participation in the study: Your participation in the study is entirely voluntary. You may refuse to take part in the study or withdraw from the study and this will not affect your treatment at KLE Hospital. You won't have to pay any money for participating in the study.

To become a part of the study, authorise the use, and disclosure of your personal health information, you or your legal representative must sign and date the consent form.

Purpose of the study: Participation of your infant in this study will help us analyse factors affecting 17-hydroxyprogesterone levels. Blood sample of 2ml venous blood will be drawn between 72 hours of birth and day 10 of life by the investigator.

Costs of participation: The cost of the study will be borne by the researcher. It involves the cost of determining the serum 17-hydroxyprogesterone levels. You will not be monetarily compensated for your contribution in this study.

Confidentiality: The identity of the participant will be kept strictly confidential, both during the study, and while publishing results of the study. All personal information will be encoded and kept in locked files.

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

ಭಾಗವಹಿಸುವವರ ಮಾಹಿತಿ ವಿವರ

ರೋಗಿಯ IP ಸಂಖ್ಯೆ	:
ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು	:
ಸಂಪರ್ಕ ಸಂಖ್ಯೆ	:
ಸಂಪರ್ಕ ವಿಳಾಸ	:ಜೂನಿಯರ್ ರೆಸಿಡೆಂಟ್, ಚಿಕ್ಕಮಕ್ಕಳ ಚಿಕಿತ್ಸಾ ವಿಭಾಗ, ಕೆಎಲ್ಇ ಸಂಸ್ಥೆಯ ಜೆಎನ್ ವೈದ್ಯಕೀಯ ಮಹಾವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ

ಯೋಜನೆಯ ಶೀರ್ಷಿಕೆ: ತೃತೀಯ ಆರೈಕೆ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನವಜಾತ ಶಿಶುಗಳ ಮೂತ್ರ ಜನಕಾಂಗದ ಹೈಪರ್ಪ್ಲಾಸಿಯಾಗೆ 17-ಹೈಡ್ರಾಕ್ಸಿಪ್ರೋಜೆಸ್ಟರಾನ್ ಪರೀಕ್ಷೆ ಮೇಲೆ ಪ್ರಭಾವ ಬೀರುವ ತಾಯಿಯ ಮತ್ತು ನವಜಾತ ಶಿಶುಗಳ ಮೇಲಿನ ವಿಶ್ಲೇಷಣೆ

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆ: ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ಕೆಎಲ್ಇ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಯಾವುದೇ ಹಣವನ್ನು ಪಾವತಿಸಬೇಕಾಗಿಲ್ಲ.

ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು, ನಿಮ್ಮ ವೈಯಕ್ತಿಕ ಆರೋಗ್ಯ ಮಾಹಿತಿಯ ಬಳಕೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ದೃಢೀಕರಿಸಿ, ನೀವು ಅಥವಾ ನಿಮ್ಮ ಕಾನೂನು ಪ್ರತಿನಿಧಿಯು ಒಪ್ಪಿಗೆ ನಮೂನೆಗೆ ಸಹಿ ಮಾಡಬೇಕು ಮತ್ತು ದಿನಾಂಕವನ್ನು ನೀಡಬೇಕು.

ಅಧ್ಯಯನದ ಉದ್ದೇಶ: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಶಿಶುವಿನ ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ 17-ಹೈಡ್ರಾಕ್ಸಿಪ್ರೋಜೆಸ್ಟರಾನ್ ಮಟ್ಟದ ಪರಿಣಾಮ ಬೀರುವ ಅಂಶಗಳನ್ನು ವಿಶ್ಲೇಷಿಸಲು ನಮಗೆ ಸಹಾಯವಾಗುತ್ತದೆ. 2 ಮಿಲಿ ರಕ್ತವನ್ನು 72 ಗಂಟೆಗಳಿಗೊಮ್ಮೆ, 10 ನೇ ದಿನದ ವರೆಗೆ ತನಿಖಾಧಿಕಾರಿ ತೆಗೆದುಕೊಳ್ಳುತ್ತಾರೆ.

ಭಾಗವಹಿಸುವಿಕೆಯ ವೆಚ್ಚಗಳು: ಇದರ ಅಧ್ಯಯನದ ವೆಚ್ಚವನ್ನು ಸಂಶೋಧಕರು ಭರಿಸುತ್ತಾರೆ. ಇದು ಸೀರಮ್ 17-ಹೈಡ್ರಾಕ್ಸಿಪ್ರೋಜೆಸ್ಟರಾನ್ ಮಟ್ಟಗಳ ವೆಚ್ಚವನ್ನು ಒಳಗೊಂಡಿರುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಕೊಡುಗೆಗಾಗಿ ನಿಮಗೆ ಯಾವುದೇ ಮೊತ್ತವನ್ನು ಪಾವತಿಸಲಾಗುವುದಿಲ್ಲ.

ಗೌಪ್ಯತೆ: ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಮತ್ತು ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸುವಾಗ ಭಾಗವಹಿಸುವವರ ಗುರುತನ್ನು ಕಟ್ಟುನಿಟ್ಟಾಗಿ ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ. ನಿಮ್ಮ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಬಗ್ಗೆ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಎನ್‌ಕೋಡ್ ಮಾಡಲಾಗುತ್ತದೆ ಹಾಗೂ ಲಾಕ್ ಮಾಡಿದ ಫೈಲ್‌ಗಳಲ್ಲಿ ಇರಿಸಲಾಗುತ್ತದೆ.

ಹಿಂತೆಗೆದುಕೊಳ್ಳುವ ಹಕ್ಕು: ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಇದರೊಂದಿಗೆ ಬೆಳಗಾವಿಯ ಜೆಎನ್‌ಎಂಸಿ ಮತ್ತು ಕೆಎಲ್‌ಇ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನೀವು ಪಡೆಯುತ್ತಿರುವ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ.

ಪ್ರಶ್ನೆಗಳು: ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಹೊಂದಿದ್ದರೆ, ನೀವು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ _____, ಪಿಜಿ ಎಂಡಿ ಪಿಡಿಯಾಟ್ರಿಕ್ಸ್ 2023 ಪ್ರವೇಶ ಬ್ಯಾಚ್, ಪಿಡಿಯಾಟ್ರಿಕ್ಸ್ ವಿಭಾಗ, ಜೆ.ಎನ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಬೆಳಗಾವಿ 590010, ದೂರವಾಣಿ ಸಂಖ್ಯೆ: _____ ಇವರನ್ನು ಸಂಪರ್ಕಿಸಬೇಕು.

ಒಪ್ಪಿಗೆಯ ಪತ್ರ

ರೋಗಿಯ ಸಂಖ್ಯೆ:

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು :

ಮಾರ್ಗದರ್ಶಕರ ಹೆಸರು :

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ :

ಸಂಪರ್ಕ ವಿಳಾಸ : ಜೂನಿಯರ್ ರೆಸಿಡೆಂಟ್, ಚಿಕ್ಕಮಕ್ಕಳ ಚಿಕಿತ್ಸಾ
ವಿಭಾಗ,

ಕೆಎಲ್ಇ ಸಂಸ್ಥೆಯ ಜೆಎನ್ ವೈದ್ಯಕೀಯ
ಮಹಾವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ

ಮೇಲೆ ಒದಗಿಸಿದ ಮಾಹಿತಿ ವಿವರವನ್ನು ನಾನು ಎಚ್ಚರಿಕೆಯಿಂದ ಓದಿದ್ದೇನೆ

/ ನಾನು ಗ್ರಹಿಸುವ ಭಾಷೆಯಲ್ಲಿ ನನಗೆ ಸಂಪೂರ್ಣವಾದ ಮಾಹಿತಿಯನ್ನು ನೀಡಿದ್ದಾರೆ. ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ.

ಅಧ್ಯಯನದ ಸ್ವರೂಪ ಮತ್ತು ಉದ್ದೇಶ ಮತ್ತು ಅದರ ಸಂಭಾವ್ಯ ಅಪಾಯಗಳು/ ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅಧ್ಯಯನದ ನಿರೀಕ್ಷಿತ ಅವಧಿ ಮತ್ತು ಅಧ್ಯಯನದ ಸಂಬಂಧಿತ ವಿವರಗಳನ್ನು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ನವಜಾತ ಶಿಶುವಿನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಮತ್ತು ನನ್ನ ವೈದ್ಯಕೀಯ ಆರೈಕೆ ಅಥವಾ ಕಾನೂನು ಹಕ್ಕುಗಳಿಗೆ ಧಕ್ಕೆಯಾಗದಂತೆ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂಪಡೆಯಲು ನಾನು ಮುಕ್ತನಾಗಿದ್ದೇನೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ ಮತ್ತು ನನ್ನ ಯಾವುದೇ ವೈದ್ಯಕೀಯ ಟಿಪ್ಪಣಿಗಳ ವಿಭಾಗದಿಂದ ನನ್ನ ಬಗ್ಗೆ ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯು ಸಂಬಂಧಿತ ನಿಯಂತ್ರಕ ಅಧಿಕಾರಿಗಳಿಂದ ಜವಾಬ್ದಾರಿಯುತ ವ್ಯಕ್ತಿಗಳಿಂದ ನೋಡಬಹುದಾಗಿದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಮೇಲೆ ಹೇಳಲಾದ ತನಿಖಾಧಿಕಾರಿಗಳಿಗೆ ದಾಖಲೆಗಳನ್ನು ನೋಡಲು ನಾನು ಅನುಮತಿ ನೀಡುತ್ತೇನೆ.

ವ್ಯಕ್ತಿಯ ಸಹಿ / ಎಡ ಹೆಚ್ಚರಳನ ಗುರುತು:

ಭಾಗವಹಿಸುವವರ ಹೆಸರು:

ಸ್ಥಳ: ದಿನಾಂಕ:

ಪಾಲಕರು/ಪೋಷಕರ ಹೆಸರು:

ಪಾಲಕ/ಪೋಷಕರ ಸಹಿ:

ನನ್ನ ಉಪಸ್ಥಿತಿಯಲ್ಲಿ ಮೇಲಿನ ಒಪ್ಪಿಗೆಯನ್ನು ಪಡೆಯಲಾಗಿದೆ ಎಂದು ಪ್ರಮಾಣೀಕರಿಸುತ್ತೇನೆ.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು -

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಸಹಿ

ಸಾಕ್ಷಿಯ ಹೆಸರು

ಸಾಕ್ಷಿ ಸಹಿ

वैद्यकीय संशोधन सहभाग माहिती फॉर्म

- १) पेशंट आयडी नंबर : _____
- २) मुख्य संशोधकाचे नाव :
- ३) मोबाईल नंबर :
- ४) पत्ता : जूनियर रेसिडेंट, बालरोग विभाग, के.एल.ई. जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगावी.

वैद्यकीय संशोधनाचा विषय :-

टरशरी केअर हॉस्पिटल मध्ये कंजेनाईटल एड्डीनल हायपरप्लासिया साठी ठेवलेल्या नवजात बालकांमध्ये असणाऱ्या 17- हायड्रोक्सीप्रोजेस्टेरॉन वर होणाऱ्या मॅटरनल आणि नीवोन्टल घटकांचा विश्लेषणात्मक अभ्यास.

संशोधनामधील सहभाग :-

या संशोधनामधील आपला सहभाग हा पूर्णपणे स्वयं अनुमती पूर्ण आणि स्वयंभावी आहे. तुम्ही या संशोधन प्रकल्पातून जर माघार घेतली किंवा संशोधनासाठी जरी नकार दर्शवला तरी त्याचा तुमच्या के.एल.ई. हॉस्पिटलमध्ये सुरू असलेल्या उपचारावर कोणताही परिणाम होणार नाही. तुम्हाला या संशोधन प्रकल्पामध्ये सहभागी होण्यासाठी कोणत्याही प्रकारचे पैसे भरावे लागणार नाहीत. या वैद्यकीय संशोधनामध्ये सहभागी होत असताना तुमची आरोग्यविषयक माहिती अभ्यासासाठी वापरता यावी यासाठी तुम्हाला किंवा तुमच्या वतीने कोणत्याही एका कायदेशीर व्यक्तीला एक अनुमती पत्र सादर करावे लागेल.

संशोधनाचा उद्देश :-

या संशोधनामध्ये असणारा तुमच्या बालकाचा सहभाग हा नवजात शिष्यंमध्ये असणाऱ्या 17- हायड्रोक्सी प्रोजेस्टेरॉन वर होणाऱ्या घटकांचा विश्लेषणात्मक अभ्यास करण्यासाठी आम्हाला मदत करेल. त्यासाठी शिशु चे 2 एम. एल. रक्त 72व्या तासापासून ते दहाव्या दिवसापर्यंत संशोधका द्वारे घेतले जाईल

वैद्यकीय संशोधनामधील सहभागाचे मूल्य :-

या संशोधनासाठी केला जाणारा सर्व खर्च हा पूर्णतः संशोधका द्वारे केला जाईल शिशुच्या रक्तातील 17- हायड्रोक्सी प्रोजेस्टेरॉनची पातळी तपासण्यासाठी केल्या जाणाऱ्या टेस्टचा खर्चही यात समाविष्ट आहे जो संशोधका द्वारे केला जाईल. आपणास या संशोधनामध्ये सहभागी झाल्याबद्दल कोणतेही मानधन दिले जाणार नाही

संशोधना मधील गुप्तता :-

या वैद्यकीय संशोधनामध्ये सहभागी झालेल्या व्यक्तीची माहिती ही अतिशय काटेकोरपणे आणि पूर्णपणे गुप्त राखली जाईल. संशोधनादरम्यान आणि संशोधनानंतर प्रकाशित करण्यात येणाऱ्या रिपोर्टमध्ये देखील सहभागी झालेल्या व्यक्तीची माहिती ही पूर्णपणे गुप्त राखली जाईल. आपली व आपल्या कुटुंबाची अथवा समाजाविषयीची वैयक्तिक माहिती ही पूर्णपणे गुप्त राखली जाईल.

संशोधनातील माघार :-

या संशोधनामधील आपला सहभाग हा पूर्णपणे स्वयं अनुमती पूर्ण आणि स्वयंभावी आहे तुम्ही या संशोधन प्रकल्पातून जर माघार घेतली किंवा संशोधनासाठी जरी नकार दर्शवला तरी त्याचा तुमच्या के.एल ई. हॉस्पिटलमध्ये सुरू असलेल्या उपचारावर कोणताही परिणाम होणार नाही. तुम्हाला या संशोधन प्रकल्पामध्ये सहभागी होण्यासाठी कोणत्याही प्रकारचे पैसे भरावे लागणार नाहीत या वैद्यकीय संशोधनामध्ये सहभागी होत असताना तुमची आरोग्यविषयक माहिती अभ्यासासाठी वापरता यावी यासाठी तुम्हाला किंवा तुमच्या वतीने कोणत्याही एका कायदेशीर व्यक्तीला एक अनुमती पत्र सादर करावे लागेल.

संशोधनाविषयीचे प्रश्न :-

सदर वैद्यकीय संशोधन संदर्भात जर आपणास काही प्रश्न विचारायचे असतील तर आपण खाली नमूद केलेल्या पत्त्यावर मुख्य संशोधकाशी संपर्क साधावा.

अनुमती पत्र

- रुग्ण क्रमांक : _____
- मुख्य संशोधकाचे नाव :
- मार्गदर्शकाचे नाव :
- भ्रमणध्वनी : ;
- संपर्कासाठी पत्ता : जूनियर रेसिडेंट, बालरोग विभाग, के. एल. ई. जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगावी.

वैद्यकीय संशोधन सहभाग माहिती फॉर्म मधील पूर्ण मजकूर / माहिती मी वाचलेली आहे / मला कळेल अशा भाषेत सांगण्यात आलेली आहे आणि ती माहिती मला पूर्णपणे समजलेली आहे. या संशोधना संदर्भात प्रश्न विचारण्याची संधी मला मिळालेली होती. या संशोधनाचे स्वरूप, उद्देश, संशोधनाशी संबंधित धोके / फायदे, संशोधनासाठी लागणारा कालावधी आणि संशोधनाशी संबंधित सर्व माहिती मला विस्तृतपणे देण्यात आलेली आहे. मला हे माहित आहे की माझ्या नवजात बालकाचा या संशोधना मधील सहभाग हा पूर्णपणे स्वयंभावी आहे आणि मला हेही माहित आहे की मी आणि माझे नवजात शिशु या संशोधनातून कोणतेही कारण न देता कधीही माघार घेऊ शकतो आणि त्याचा माझ्या कायदेशीर अधिकारांवर आणि सुरू असलेल्या वैद्यकीय उपचारांवर कोणताही परिणाम होणार नाही. मला हे पूर्णतः माहित आहे की या वैद्यकीय संशोधनातील माझ्या सहभागातून मिळवण्यात आलेली माझी / माझ्या आरोग्य विषयीची माहिती ही आवश्यकतेनुसार वैद्यकीय क्षेत्रातील संबंधित व जबाबदार व्यक्तींकडून पाहण्यात अथवा हाताळण्यात येईल. अशी माहिती वापरण्यास माझी पूर्णपणे अनुमती आहे व या संदर्भात माझा कोणताही आक्षेप नाही.

सही /डाव्या अंगठ्याचा ठसा: _____

सहभागी होणाऱ्या चे नाव: _____

स्थळ: _____

तारीख: _____

पालकाचे नाव: _____

पालकाची सही: _____

असे प्रमाणित करण्यात येते की वरील अनुमती पत्र हे माझ्या समक्ष आणि माझ्या उपस्थितीत घेण्यात आलेले आहे.

मुख्य संशोधकाचे नाव: _____

मुख्य संशोधकाची सही: _____

साक्षीदाराचे नाव: _____

साक्षीदाराची सही: _____

२

ANNEXURE – II -

PROFORMA

Date: _____ **Neonate's IP Number:** _____

Mother's Name: _____ **Mother's IP Number:** _____

Mother's Age:

Obstetric history:

Gravida: _____ **Parity:** _____ **Abortions:** _____ **Living:** _____

Current pregnancy: _____

Conception: Spontaneous/ Assisted Reproduction Technique (Specify: _____)

Last Menstrual Period: _____

Expected Date of Delivery: _____

Birth history

Date of birth: _____

Time of birth: _____

Mode of delivery: Normal vaginal delivery / Instrumental delivery/ C- section delivery

Gestational Age at birth: Term / Preterm **Period of gestation:** _____

Sex: Male / Female

Birth Weight: _____

APGAR Score:

1. 1 minute: _____
2. 5 minutes: _____
3. 10 minutes: _____

Perinatal Factors

Maternal factors:

1. Pregnancy Induced hypertension / Eclampsia
2. PROM
3. Gestational Diabetes mellitus
4. Hypothyroidism
5. Anaemia
6. Polyhydramnios / Oligohydramnios
7. Diabetes Mellitus
8. Hypertension
9. Epilepsy
10. Antenatal medications:
 - a. Antenatal Steroid – Yes / No

If yes,
 - i. Partial []
 - ii. One complete courses []
 - iii. Multiple courses []
 - b. Progesterone: Yes / No
11. Others: _____

Neonatal factors:

NICU admission: Yes / No

Reason:

1. Neonatal Respiratory Distress
 - a. Oxygen
 - b. CPAP
 - c. Ventilator
2. Neonatal Pneumonia
3. Meconium aspiration syndrome
4. Early onset sepsis
5. Necrotizing enterocolitis
6. Birth asphyxia
7. Neonatal seizures
8. Neonatal Hyperbilirubinemia
9. Others: _____

Investigations: Serum 17-hydroxyprogesterone levels: _____

ANNEXURE III – MASTER CHART

69	Ashwini	23	2	Multigravida	2	1	2	Spontaneous	Term	38.5	3	1	3.1	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7	
70	Sukanya	31	4	Multigravida	1	0	1	Spontaneous	Late preterm	35Week5	3	2	1.7	2	7	9	10	PROM	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.3	
71	Sukanya	31	4	Multigravida	1	0	1	Spontaneous	Late preterm	35.5	2	1	2.6	3	9	10	10	PROM	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.7	
72	Fatima.K.soudagar	25	2	Multigravida	2	0	2	Spontaneous	Term	37.5	3	2	2.2	2	8	10	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.2		
73	Roopali	19	1	Primigravida		0		Spontaneous	Late preterm	36.6	2	1	2.6	3	9	10	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.4			
74	Pooja	23	2	Multigravida		1		Spontaneous	extreme preterm	25.3	1	1	0.75	1	8	10	10	None	0	0	0	0	0	0	0	0	0	0	0	1	1	Respiratory distress syndrome	1	0	0	0	0	0	0	0	0	1	7.1				
75	Savita N.Patil	23	2	Multigravida	1	0	1	Spontaneous	Term	38.4	3	1	2.7	3	4	8	10	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	6.6				
76	Mahalaxmi	23	2	Multigravida	1	0	1	Spontaneous	Term	39.3	3	2	2.9	3	7	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	6.4				
77	Priyanka	24	2	Primigravida		0		Spontaneous	Late preterm	35.2	2	1	1.2	2	6	8	10	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.5				
78	Nehanali	24	2	Multigravida	2	0	2	Spontaneous	Late preterm	36.6	2	1	2.6	3	9	10	10	PROM	0	1	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.8				
79	Savita	23	2	Multigravida	1	1	1	Spontaneous	Term	38.4	3	1	3	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.5				
80	Snehal	26	3	Multigravida		1		Spontaneous	Late preterm	33.6	2	1	1.6	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.7					
81	Deepa	30	3	Primigravida		0		Spontaneous	Term	40.1	3	1	2.4	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0																5.5	
82	Ronjonsa	22	2	Primigravida		0		Spontaneous	Term	40	3	2	2.4	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0																8	
83	Chandni	24	2	Primigravida		0		Spontaneous	Term	38.2	3	2	3	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0																		8.3
84	Akshata	20	1	Primigravida		0		Spontaneous	Term	40	3	1	2.9	3	9	10	10	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	8.7				
85	Saniya.Hubli	27	3	Multigravida	1	1	1	Spontaneous	Late preterm	36	2	1	2.1	2	7	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	8.4					
86	Laxmi	30	3	Multigravida	1	0		Spontaneous	Term	38	3	1	3.3	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	6.5					
87	Sheetal	28	3	Primigravida		0		Spontaneous	Term	39.6	3	1	2.3	2	7	8	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.3					
88	Rasika	20	1	Multigravida		1		Spontaneous	Term	38	3	2	2.7	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0																	6.3	
89	Kajal	24	2	Primigravida		0		Spontaneous	Term	37.6	3	1	2.3	2	7	9	10	None	0	0	0	0	0	0	0	0	0	0	0																	5.6	
90	Sushmita	26	3	Primigravida		0		Spontaneous	Term	40	3	1	2.6	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	6					
91	Fiza	31	4	Primigravida		0		Spontaneous	Late preterm	35.6	2	2	2.1	2	7	8	9	PROM	0	1	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	12					
92	Hasina	28	3	Multigravida	1	1	1	Spontaneous	Term	38.4	3	2	2.5	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0																		6.9
93	Sita	22	2	Multigravida	1	0	1	Spontaneous	Term	37	3	1	1.9	2	9	10	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	6.8					
94	Geeta	27	3	Primigravida		0		Spontaneous	Term	38.1	3	2	2.75	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0																			5
95	Supriya	27	3	Primigravida		0		Spontaneous	Late preterm	33.2	2	1	1.86	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4					
96	Preeti	20	1	Multigravida	1	0	1	Spontaneous	Late preterm	36.5	2	1	2.2	2	7	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	9					
97	Veena	22	2	Multigravida	1	1	1	Spontaneous	Late preterm	36.2	2	2	1.9	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.9					
98	Laxmi	23	2	Primigravida		0		Spontaneous	Term	38.1	3	1	2.7	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0																			4.8
99	Apeksha	29	3	Primigravida		0		Spontaneous	Term	39.6	3	2	2.8	3	8	10	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4					
100	Rekha	23	2	Primigravida		0		Spontaneous	Term	39.2	3	2	2.1	2	7	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.6					
101	Basavva	28	3	Multigravida	1	0	1	Spontaneous	Term	38.3	3	1	3.2	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.4					
102	Poonam	28	3	Primigravida		0		Spontaneous	Term	38	3	1	3.2	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.7					
103	Iqra	20	1	Primigravida		0		Spontaneous	Late preterm	33	2	1	1.6	2	6	7	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.6					
104	Manisha	25	2	Primigravida		0		Spontaneous	Late preterm	34.4	2	1	1.6	2	5	8	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.7					

253	Priyanka	28	3	Primigravida		0	Assisted ReproductionT echnique	Late preterm	36.1	2	1	2.3	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	10.2	
254	Mahek	24	2	Multigravida	1	0	1	Spontaneous	Term	40	3	1	2.6	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	6.9	
255	Chanda	29	3	Multigravida	1	0	1	Spontaneous	Term	39.3	3	2	2.4	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4.3
256	Rohini	33	4	Primigravida		0		Spontaneous	Term	39.2	3	2	2.1	2	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4.1	
257	Aishwarya	33	4	Multigravida	1	1		Spontaneous	Late preterm	35.4	2	1	2	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.2	
258	Priyanka	38	4	Primigravida		0		Spontaneous	Term	39	3	1	3	3	6	7	8	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	4.3		
259	Mangala	34	4	Multigravida	1	0	1	Spontaneous	Term	38.2	3	2	2.6	3	8	9	10	PROM	0	1	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	5.2		
260	Bhagyashree	28	3	Multigravida	1	0	1	Spontaneous	Term	39.3	3	1	2.4	2	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	11.2			
261	Madhavi	21	2	Multigravida	1	0	1	Spontaneous	Term	37weeks3	3	1	2.5	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	11.3			
262	Nandini	24	2	Primigravida		0		Spontaneous	Late preterm	35.6	2	2	1.8	2	6	7	8	PROM	0	1	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	9.8			
263	Alen	26	3	Multigravida	1	1		Spontaneous	Term	38.1	3	2	2.1	2	8	9	10	Anaemia	0	0	0	0	1	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.3			
264	Saniya	29	3	Multigravida	2	0	2	Spontaneous	Term	39.2	3	1	3.3	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10		
265	Madhavi	28	3	Primigravida		0		Assisted ReproductionT echnique	Term	39.4	3	2	2.9	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7.3	
266	Saraswati	31	4	Multigravida	1	0	1	Spontaneous	Term	37.2	3	1	2.8	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10.7	
267	Mayuri	30	3	Multigravida	2	0	2	Spontaneous	Term	39.2	3	2	3	3	8	9	10	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8.1	
268	Mahek	27	3	Primigravida		0		Spontaneous	Term	38.5	3	1	3.3	3	8	9	10	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	6.7	
269	Saba	22	2	Primigravida		0		Spontaneous	Term	39.6	3	2	2.9	3	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8	
270	Ramona	28	3	Primigravida		0		Spontaneous	Late preterm	36.5	2	2	2	3	6	7	8	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	7.9		
271	Poonam	28	3	Primigravida		0		Spontaneous	Term	40.3	3	1	2.3	2	8	9	10	None	0	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	12.1		
272	Priyanka	31	4	Primigravida		0		Spontaneous	Late preterm	34.3	2	2	2	2	6	7	8	Pregnancy Induced hypertension/Ecla mpsia	1	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	13			
273	Asha	32	4	Primigravida		0		Spontaneous	Term	38	3	1	2.3	2	7	8	9	gestational diabetes mellitus	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8.2		
274	Dani	30	3	Multigravida	1	0	1	Assisted ReproductionT echnique	Term	39.4	3	1	2.6	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8	
275	Usha	25	2	Multigravida	2	0	1	Spontaneous	Term	37.3	3	1	3	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7.3	
276	Priyanka	26	3	Primigravida		0		Spontaneous	Term	39.3	3	1	2.8	3	6	7	8	None	0	0	0	0	0	0	0	0	0	0	1	birth asphyxia, neonatal resp distress	1	0	0	0	0	0	1	0	0	0	15.1			
277	Nisha	26	3	Multigravida	1	0	1	Spontaneous	Term	39.3	3	1	2.7	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	10			
278	Sunita	24	2	Primigravida		0		Spontaneous	Term	37.3	3	1	2.9	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	11.3			
279	Fardana	29	3	Multigravida	1	0	1	Spontaneous	Term	39.2	3	1	3	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	12.5			
280	Sonali	36	4	Multigravida	1	0	1	Assisted ReproductionT echnique	Term	37.3	3	1	2.8	3	6	7	8	PROM	0	1	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	13.4			
281	Soniya	32	4	Multigravida	2	0	2	Spontaneous	Late preterm	36.3	2	1	2.4	2	6	7	8	None	0	0	0	0	0	0	0	0	0	1	meconium aspiration syndrome	0	0	1	0	0	0	0	0	0	0	12.8				
282	Deepa	28	3	Multigravida	1	0	1	Spontaneous	Term	39.5	3	2	2.7	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	0	1	0	10.2			
283	Savita	27	3	Primigravida		0		Spontaneous	Term	39.2	3	2	2.3	2	7	8	9	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7.3			
284	Sonam	23	2	Multigravida	1	1		Spontaneous	Term	39.1	3	1	2.6	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	5.3		
285	Sushma	30	3	Multigravida	2	0	1	Spontaneous	Term	38.3	3	1	3.4	3	6	7	8	None	0	0	0	0	0	0	0	0	0	0	1	birth asphyxia, neonatal resp distress	1	0	0	0	0	1	0	0	0	0	13.7			

324	Komal	30	3	Multigravida	1	1	Assisted ReproductionT echnique	Term	37.3	3	1	2.5	3	6	7	8	Hypertension	0	0	0	0	0	0	0	0	1	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.6
325	Annapurna	21	2	Primigravida		0	Spontaneous	Term	38.5	3	2	2.8	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.3	
326	Shubhangi	28	3	Multigravida	1	0	1	Spontaneous	Term	37.3	3	1	2.6	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	8.3
327	Archana	30	3	Primigravida		0	Spontaneous	Term	39.3	3	2	2.9	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0			0	0	0	0	0	0	0	0	0	3.6	
328	Shubhangi	30	3	Multigravida	2	0	1	Spontaneous	Term	37.3	3	2	2.5	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	7.8	
329	Madhuri	26	3	Primigravida		0	Spontaneous	Term	37.6	3	2	2.8	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	9.4	
330	Pooja	23	2	Primigravida		0	Spontaneous	Term	39.3	3	1	2.5	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0			0	0	0	0	0	0	0	0	0	9.4	
331	Akshata	26	3	Primigravida		0	Spontaneous	Term	37.6	3	1	2.5	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	8.5	
332	Sonali	24	2	Multigravida	1	0	1	Spontaneous	Term	38.5	3	2	2.7	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	7.4	
333	Aishwarya	24	2	Primigravida		0	Spontaneous	Term	39.5	3	1	2.6	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.3		
334	Bharati	29	3	Primigravida		0	Spontaneous	Term	37.6	3	2	3	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	10		
335	Reshma	31	4	Primigravida		0	Spontaneous	Term	39.3	3	1	3	3	6	7	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	11.5	
336	Mithali	32	4	Multigravida	1	0	1	Spontaneous	Term	38.3	3	1	2.9	3	6	7	8	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	11.8	
337	Manisha	30	3	Primigravida		0	Spontaneous	Term	39.3	3	1	2.8	3	6	7	8	None	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	10.3		
338	Divya	29	3	Multigravida	1	1		Spontaneous	Late preterm	33.3	2	1	1.1	2	5	6	7	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	15.1	
339	seema	30	3	Multigravida	2	0	1	Spontaneous	Term	37.4	3	1	2.5	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	10.2
340	Sushma	27	3	Multigravida	1	1	Assisted ReproductionT echnique	Late preterm	34.3	2	2	1.4	2	6	7	8	Pregnancy Induced hypertension/Ecla mpsia	1	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	18.3	
341	Surekha	23	2	Primigravida		0	Spontaneous	Term	39.6	3	1	2.6	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	11.3	
342	Reshma	30	3	Multigravida	1	0	1	Spontaneous	Term	38.1	3	2	2.4	2	7	8	9	gestational diabetes mellitus	0	0	1	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	12.4	
343	pooja	23	2	Primigravida		0	Spontaneous	Term	38.6	3	2	2.75	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	8	
344	smita	26	3	Multigravida	2	0	2	Spontaneous	Term	39.3	3	1	2.7	3	7	8	9	None	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	5		
345	sridevi	24	2	Primigravida		0	Spontaneous	Term	37.6	3	1	2.7	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	4.8		
346	Bhakti	28	3	Primigravida		0	Spontaneous	Term	39.3	3	1	3	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	5.1		
347	Basavva	28	3	Primigravida		0	Spontaneous	Term	37.1	3	1	2.5	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	meconium aspiration syndrome, respiratory distress	1	0	1	0	0	0	0	0	0	18		
348	basavva twin 2	28	3	Primigravida		0	Spontaneous	Late preterm	35.1	2	1	2.1	2	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Respiratory distress syndrome	1	0	0	0	0	0	0	0	0	22		
349	Uma	24	2	Primigravida		0	Spontaneous	Term	38.3	3	1	2.6	3	7	8	9	None	0	0	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	13		
350	Gangavva	27	3	Primigravida		0	Spontaneous	Term	39.2	3	1	2.7	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	8.8	
351	Ruksha	32	4	Multigravida	1	0	1	Spontaneous	Term	39.3	3	1	2.8	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.6
352	Rajeshwari	21	2	Primigravida		0	Spontaneous	Term	37.3	3	1	2.5	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7		
353	Renuka	26	3	Primigravida		0	Spontaneous	Term	39.3	3	2	2.8	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	6.5	
354	Anita	28	3	Multigravida	1	0	1	Spontaneous	Term	39.4	3	1	2.9	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	6
355	Mahadevi	29	3	Multigravida	1	0	1	Spontaneous	Term	38.3	3	1	3	3	7	8	9	None	0	0	0	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.2	
356	Sona	31	4	Multigravida	1	0	1	Spontaneous	Term	39.6	3	1	3.1	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	7.9
357	Keertana	26	3	Primigravida		0	Spontaneous	Term	39.1	3	1	3.2	3	7	8	9	Hypothyroidism	0	0	0	1	0	0	0	0	0	0	1	Neonatal Hyperbilirubinemia	0	0	0	0	0	0	0	1	0	8.8	

