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STUDY ON ASSOCIATION OF ANAEMIA AND PREECLAMPSIA IN PREGNANCY AND ITS FETOMATERNAL OUTCOME

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LIST OF ABBREVIATIONS

PPH	-	Post partum Hemorrhage
IDA	-	Iron Deficiency Anemia
HCT	-	Hematocrit
SF	-	Serum Ferritin
CRP	-	C-Reactive Protein
STfR	-	Serum Transferrin Receptor
BMI	-	Body mass index
ARF	-	Acute Renal Failure
CVA	-	Cerebro vascular Accident
DIC	-	Disseminated Intravascular coagulation
IUGR	-	Intra uterine growth retardation
LBW	-	Low Birth Weight

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Introduction:-

Anaemia is a major public health problem especially among low socioeconomic class of the population in developing countries. Prevalence of anaemia among pregnant women in developing countries is 51% whereas it is only14 % in the developed countries. Prevalence of anaemia in India is 65-75% in pregnant women.[1]

Anaemia is a major public health problem throughout the world specially in developing countries like India and it is the most common nutritional deficiency disorder in the world.[2] High prevalence of anaemia among pregnant women persists in India despite the availability of effective and low-cost interventions for prevention and treatment.[3] Iron deficiency anaemia is an important public health problem for pregnant women, living in developing countries, affecting 2/3rd of pregnant women and contributes to maternal morbidity and mortality and to low birth weight.[4,5]

Anaemia has hazardous influence on maternal and fetal outcome and increases risk of postpartum haemorrhage (PPH), infection, sepsis and risk for pre-term birth, low birth weight and small for gestational age babies thereby contributing to maternal and perinatal morbidity and sometimes mortality.[6] Maternal and foetal complications affect mainly in the women with unfavourable health conditions and lower socioeconomic status. Maternal mortality rates are higher in women with moderate and severe anaemia. Premature births are more common in women with moderate anaemia. Infection, maternal deaths due to ante partum and post-partum

haemorrhage, pregnancy induced hypertension and sepsis occur in women with moderate anaemia. Severe Anaemia leads to cardiac decompensation when Hemoglobin falls below 5.0 g/dl.[7]

Hypertensive disorders of pregnancy and their complications rank as one of the major cause of maternal mortality and morbidity in the world. Amongst them, preeclampsia is emerging as one of the most common complication of pregnancy. Pre-eclampsia is a multi-system disorder of unknown aetiology, unique to pregnancy, with onset after 20 weeks of gestation. Although, the exact aetiology of preeclampsia is not yet known, many factors such as low education, primi parity, family history of hypertension, obesity, younger and advanced maternal age and malnourishment are proven as its risk factors and evidence suggests that various other factors like severe anemia could also be a risk factor for development of preeclampsia and that cannot be ignored.[8]

Several primary research studies have been conducted to identify the potential effects of maternal anaemia on adverse maternal obstetric and birth outcomes. However, findings across the studies are not consistent which makes it challenging to form evidence-based policies to reduce these adverse consequences. For instance, maternal anaemia is reported as a risk factor of pre-term birth (PTB) in a few studies but other studies reported no significant associations.[9,10]

Preeclampsia is not a preventable disease, but the reversible causes which play a role in its aetiology and which are simple to get treated, the incidence of preeclampsia

can be reduced. Thus diagnosing of reversible factors such as anemia in pregnancy and its management is important. In view of the above-mentioned adverse effects of anaemia in pregnancy this study was planned in our hospital to evaluate the fetomaternal outcome due to anaemia in pregnancy.

Aims and Objectives:-

Aim:-

- To find out the relationship between anemia and pre eclampsia in pregnancy and to look for maternal and fetal outcome.

Objectives:-

- To analyse the association of anemia with pre eclampsia in pregnancy.
- To diagnose and treat anemia earlier preventing maternal and fetal complications.

Review of Literature:-

Definition of Anemia:

As defined WHO, anemia in pregnancy develops when hemoglobin (Hb) concentration reduces to <11g/dL with haematocrit of <0.33/L.2 Anaemia in pregnancy present similarly like anaemia in other categories with signs and symptoms which includes easy development of fatigue, general weakness, reduced cognition, and attention/concentration span and when not managed the affected mothers experience preterm birth with low birth weight babies. According to WHO, during pregnancy, anemia is identified by hemoglobin levels less than 11.0g/dL and may be divided into three levels of severity: mild anemia (Hb levels 9 to 10.9g/dL), moderate anemia (Hb levels 7 to 8.9g/dL), and severe anemia (Hb levels less than 7g/dL).

Epidemiology of Anemia in Pregnancy:

Anaemia is a global public health problem affecting both developing and developed countries with major consequences for human health as well as social and economic development which results in a loss of billions of dollars annually.[11-13] According to the 2008 World Health Organization (WHO) report, anaemia affected 1.62 billion (24.8%) people globally. It had an estimated global prevalence of 42% in pregnant women and is a major cause of maternal mortality.[14] In Africa, 57.1%of the pregnant women were anemic.

Moreover, anemia in pregnant women is a severe public health problem in Ethiopia; 62.7% of pregnant women were anemic.Although the prevalence varies widely in different settings and accurate data are often lacking, in resource-limited areas terribly significant proportions of women of childbearing age particularly pregnant are anaemic. Geographically, those living in Asia and Africa are at the greatest risk.

Anemia in pregnancy is a worldwide health challenge affecting low-, middle-, and high-income countries with several impacts on health and socio-economic progress. It was approximated that nearly 40.1% of pregnant mothers develop anaemia globally.[16]

Global data now show that anaemia in pregnancy in low- and middle-income countries can be as high as 56%[17] with continental variations; sub-Saharan Africa shows 57% prevalence, South-East Asia 48%, and South America at 24.1%.

In Uganda, there has been poor utilization of (ITN) in rural areas and poor adherence to iron supplementation which thwarted the efforts to reduce anaemia in pregnancy.[19] According to Uganda Demographic and Health Surveys report, the prevalence of anemia among pregnant women was 38% in 2016[20] but with regional variations from 32.9% in Gulu, northern Uganda, 12.1% in Hoima,[21] and 32.5% in Mpigi.[22] In Itojo district hospital, very few data exist about the prevalence of anemia among pregnant women accessing antenatal clinic.

Anaemia is a major public health problem especially among low socioeconomic class of the population in developing countries. Prevalence of anaemia among pregnant women in developing countries is 51% whereas it is only14 % in the developed countries.[23] Prevalence of anaemia in India is 65-75% in pregnant women. As per WHO, anaemia during pregnancy is defined as haemoglobin concentration of less than 11 gm% (7.45 mmol/L) and haematocrit less than 33%.

India has the highest prevalence of anaemia. Women of child bearing age are at the maximum risk for development of anaemia. Anaemia is graded according to Hb level in three degrees mild (10-10.99 gm%), moderate (7.0-9.9 gm%) and severe degree (<7.0 gm%) according to WHO.[24] 87% of women have nutritional anaemia in the pregnancy due to iron deficiency. Iron deficiency during pregnancy is thought to be caused by a combination of factors such as previously decreased iron supply, the iron requirements of growing foetus and expansion of maternal plasma volume.[25]

Physiologic significance of anemia:

Mild anemia is routinely defined as a hemoglobin value within 10 g/L of the anemia cutoff value. The World Health Organization recommends that severe anemia be defined as a hemoglobin concentration < 70 g/L.[26] Hemoglobin concentrations below that of the mild anemia concentration and > 70 g/L can be regarded as indicating moderate anemia. Some investigators have defined very severe anemia as a hemoglobin concentration <50 g/L.

Hemoglobin concentrations < 50 g/L significantly increase the risk of maternal and fetal mortality because of the effects of hypoxia and anemia on the cardiovascular system, which is known as high-output heart failure.[27] Medical evidence shows that very severe anemia is a direct cause of maternal and child mortality.

Classifying very severe anemia as a major cause of maternal mortality along with eclampsia, obstructed birth, hemorrhage, and sepsis is appropriate. Ross and Thomas[28] estimated that 20% of maternal mortality can be attributed to severe anemia. In the less severe range, however, the evidence that anemia is a direct cause of poor reproductive outcomes is not clear. Epidemiologic studies that showed an association between maternal anemia and increased risk of poor birth outcomes did not establish a causal relation (evidence of risk only).[29,30] It is possible that a common factor can cause both anemia and poor birth outcomes.

Although oxygen carrying capacity is proportional to the circulating hemoglobin concentration, an individual with chronic anemia develops a compensatory mechanism to improve oxygen unloading to tissue from hemoglobin during the resting state. This compensatory mechanism can maintain adequate tissue oxygen delivery down to a hemoglobin concentration of 70–80 g/L.[31,32] In an exercise state, however, any loss of hemoglobin or red blood cell mass can be detected as loss in work capacity, even within a hemoglobin range of 120–130 g/L.[33] From a physiologic point of view, the evidence is clear that moderate anemia is undesirable, whatever the cause.

If the cause of significant anemia is iron deficiency (evidence of deficiency), prevention and correction of iron deficiency anemia are indicated. From the perspective of reproductive health outcomes, however, the evidence is not clear that anemia or iron deficiency are direct risk factors. Perhaps a concern more important than the health consequence of anemia is the cause of the anemia. Some major causes of anemia have many other damaging effects or health consequences beyond anemia. For example, malaria is well known to cause severe anemia in many tropical areas, particularly among primi gravidae, and it also contributes to the low birth weight of infants.

Iron deficiency anemia in Pregnancy:-

In 2011, 29% (496 million) of non-pregnant women and 38% (32.4 million) of pregnant women aged 15–49 years were anemic, of which about 20 million had severe anemia.[35] Although IDA is most frequent in low-income countries, recent data show that 40–50% of European non-pregnant women have low iron body stores.[36] Women are known to have a much higher iron deficiency prevalence compared to men of the same age; the prevalence rate is about 10-times higher than males. This difference is mostly due to regular blood loss during menstruation, which is often associated with low iron intake.[37] Adolescent girls are particularly vulnerable to this condition because of the elevated iron request for rapid growth, and menstrual blood loss.[38]

Furthermore, several conditions can play a determinant role in favoring insufficiency of iron in women, such as chronic gynecologic bleeding due to uterine fibroids,[39] endometriosis,[40] adenomyosis, or endometrial hyperplasia. Moreover, intestinal malabsorption problems, frequent blood donation, and benign and malignant gastrointestinal lesions are other causes of IDA in women.[41]

IDA is a frequent condition during pregnancy. The global prevalence of anemia in pregnancy is estimated to be approximately 41.8%;[42] nevertheless, the percentage of iron deficiency without anemia is unknown. The overall iron requirement during pregnancy is significantly higher than in the nonpregnant state, despite the temporary respite from iron losses incurred during menstruation. This is due to an exponential increase of iron needs to expand the plasma volume, produce a greater quantity of red blood cells, support the growth of fetal-placental unit, and compensate for iron loss at delivery.[43]

The physiological iron demand in pregnant women corresponds roughly to 1000–1200 mg for an average weight of 55 kg. This quantity includes almost 350 mg associated with fetal and placental growth, about 500 mg associated with expansion in red cell mass, and around 250 mg associated with blood loss at delivery. In the course of gestation, iron need presents a variation with a growing trend; in fact, there is a lower iron necessity in the first trimester (0.8 mg/day) and a much higher need in the third trimester (3.0–7.5 mg/day). At the beginning of pregnancy, approximately 40% of women show low or absent iron stores, and up to

90% of women have iron reserves of < 500 mg, which represent an insufficient amount to support the increased iron needs.[44,45] An overt IDA frequently develops in pregnancy even in developed countries, indicating that the physiologic adaptations are often insufficient to meet the increased requirements, and iron intake is often below nutritional needs. IDA in pregnancy, if not diagnosed and treated, can have a significant impact on maternal and fetal health.[46]

Diagnosis of anemia during pregnancy:

The definition of anemia recommended by the Centers for Disease Control and Prevention is "a Hb or hematocrit (Hct) value less than the fifth percentile of the distribution of Hb or Hct in a healthy reference population based on the stage of pregnancy".[47] Current classification lists the following levels as anemic: Hb (g/dL) and Hct (percentage) levels below 11 g/dL and 33%, respectively, in the first trimester; 10.5 g/dL and 32%, respectively, in the second trimester; and 11 g/dL and 33%, respectively, in the third trimester.58 Because of the numerous adverse consequences on maternal and fetal health that IDA causes during pregnancy, early diagnosis is essential.

Laboratory evaluation is fundamental for a definitive diagnosis of iron deficiency and IDA. As the etiology of anemia includes various causes, the diagnosis cannot be based only on Hb values. For diagnostic clarification, it is necessary to evaluate red blood count and serum ferritin (SF) levels. The most reliable

parameter to revel iron deficiency is SF, and screening of SF concentration at the beginning of pregnancy is recommended.[48] If SF is < 30 g/L, there is a high probability that iron stores are depleted, even in the absence of anemia. A SF value < 30 g/L is associated with an Hb concentration < 11 g/dL during the first trimester, < 10.5 g/dL during the second trimester, and < 11 g/dL during the third trimester are diagnostic for IDA in pregnant women.[49] Iron therapy should be considered in such cases. However, in the presence of inflammatory processes or chronic diseases, ferritin levels can be falsely normal or elevated, despite the presence of anemia. This is because ferritin reacts as an acute-phase protein.

The evaluation of C-reactive protein (CRP) levels may assist in obtaining the correct diagnosis, excluding infections or inflammation. If the CRP value is elevated, reevaluation of the SF level is recommended after the normalization of CRP concentration. Repeating SF levels measurement afterward during pregnancy is not necessary if the patient does not show symptoms of anemia. Conversely, Hb concentration should be measured in each trimester. When ferritin levels are ≥ 30 g/L, apart from measuring CRP levels, it is necessary to carry out other diagnostic investigations such as the determination of transferrin saturation and serum iron.[50]

If the level of ferritin is normal, a serum transferrin value < 15% proves a latent iron deficiency because more iron is released from blood circulation by transferrin to ensure erythropoiesis. Serum iron levels are susceptible to fluctuation

diurnal, intra- and inter-individual, so, usually, the assessment of serum iron and transferrin levels helps in diagnosis, though the SF represents the right tool.[50]

Another parameter that could be useful to detect iron deficiency during pregnancy, in the case of normal ferritin values and elevated CRPrp, is transferrin receptor (sTfR). It shows an increase in cases of iron deficiency or greater iron cellular demand. During pregnancy, the increase of sTfR values is related to increased stimulation of erythropoiesis and a major iron requirement due to iron-dependent cell proliferation. Low concentrations of sTfR in the first period of pregnancy seem to be associated with an inhibited erythropoiesis in the first trimester, as some studies have shown. Moreover, sTfR concentration is not influenced by infections or inflammatory reactions.[51]

For the differential diagnosis with other causes of anemia, such as hemoglobinopathies, infections, or chronic kidney disease, further investigations are needed. In particular, Hb electrophoresis or chromatography is indicated to exclude genetic diseases such as β-thalassemia. In cases of megaloblastic anemia, vitamin B12 should be measured since vitamin B12 deficiency is a common condition. Folic acid deficiency anemia, instead, is less frequent.[52]

Pre eclampsia:-

Eclampsia is defined as preeclampsia complicated by generalized tonic–clonic convulsion. Although eclampsia is uncommon in developed countries, it is still a major cause of maternal morbidity and mortality worldwide.'

Hypertensive disorders of pregnancy, including preeclampsia, consist of a broad spectrum of conditions that are associated with substantial maternal and fetal/ neonatal morbidity and mortality. The incidence of hypertensive disorders in pregnancy is estimated to range between 3% and 10% among all pregnancies.[53]

Worldwide, preeclampsia and related conditions are among the leading causes of maternal mortality.[54] While maternal death due to preeclampsia is less common in developed countries, preeclampsia-related maternal morbidity is high and remains a major contributor to intensive care unit admissions during pregnancy.[55]

Approximately 12–25% of growth-restricted fetuses and small-forgestational- age infants as well as 15–20% of all preterm births are attributable to preeclampsia; the associated complications of prematurity are substantial and include neonatal deaths and serious long-term neonatal morbidity. Despite major medical advances, the only known cure for preeclampsia remains delivery of the fetus and placenta.[54, 56]

Classification of Pre eclampsia:-

Preeclampsia is a pregnancy-specific syndrome that affects many organ systems and is recognized by new onset of hypertension and proteinuria after 20 weeks of gestation. It is estimated to complicate 2–8% of all pregnancies. 2 Although the precise cause is unknown, the pathophysiologic processes underlying this disorder are described as occurring in two stages.[57] The first stage is characterized by reduced placental perfusion, possibly related to abnormal placentation, with impaired trophoblast invasion and inadequate remodeling of the uterine spiral arteries. The second stage refers to the maternal systemic manifestations characterized by inflammatory, metabolic, and thrombotic responses that converge to alter vascular function, which can result in multiorgan damage.[58]

Precise classification of the various hypertensive disorders of pregnancy has remained challenging due to changing nomenclature as well as to the geographic variation in accepted diagnostic criteria. For example, terms such as “toxemia” and “pregnancy-induced hypertension” are now considered outdated. Furthermore, varying diagnostic criteria are used in different regions of the world, with disagreement about the degree of hypertension, the presence/absence of proteinuria, and the categorization of disease severity.[59] These inconsistencies have led to challenges in comparing and generalizing epidemiologic and other research findings.

The most commonly used classification system in the United States is based on the Working Group Report on High Blood Pressure in Pregnancy, in which four major categories are defined: gestational hypertension, preeclampsia-eclampsia, chronic hypertension, and preeclampsia superimposed on chronic hypertension (see Table 1 for criteria).[60]

Preeclampsia is defined as the new onset of sustained elevated blood pressure (≥140 mmHg systolic or ≥90 mmHg diastolic on at least two occasions 6 h apart) and proteinuria (at least 1+ on dipstick or ≥300 mg in a 24-h urine collection), first occurring after 20 weeks of gestation.

Although the symptoms and signs of preeclampsia occur along a continuum, the syndrome is often categorized as mild or severe to communicate the severity of disease and the management approach. Preeclampsia is considered severe when at least one of the following is present in addition to the defining blood pressure and proteinuria criteria8: 1) systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg; 2) urinary protein excretion of >5 g in a 24-h collection; 3) neurologic disturbances (visual changes, headache, seizures, coma); 4) pulmonary edema; 5) hepatic dysfunction (elevated liver transaminases or epigastric pain); 6) renal compromise (oliguria or elevated serum creatinine concentration; creatinine ≥1.2 mg/dL is considered abnormal in women without a history of renal disease); 7) thrombocytopenia; 8) placental abruption, fetal growth restriction, or oligohydramnios.

Eclampsia refers to seizures in a preeclamptic woman that cannot be attributed to other causes. The hypertensive disorder referred to as HELLP syndrome is defined by the presence of hemolysis (H), elevated liver transaminases (EL), and low platelet counts (LP). This may or may not occur in the presence of hypertension or proteinuria, but it is considered to be along the spectrum of preeclampsia.

The diagnosis of preeclampsia can be particularly challenging in women with preexisting hypertension and/or renal disease, since both blood pressure and urinary protein excretion increase toward the end of pregnancy. Thus, the diagnosis is based on a sudden increase in blood pressure or proteinuria and/or evidence of end-organ damage (Table 1).[60]

A major criticism of the various classification systems is that none have been independently evaluated for the ability to identify the subgroup of women who are at increased risk of adverse pregnancy outcomes. Recent studies have sought to develop clinically relevant definitions guided by evidence and based on predictors of adverse outcomes.[61]

Table 1 **Classification of hypertensive disorders of pregnancy.**

Hypertensive disorder	Characteristics
Mild preeclampsia	New onset of sustained elevated blood pressure after 20 weeks of gestation in a previously normotensive woman (≥140 mmHg systolic or ≥90 mmHg diastolic on at least two occasions 6 h apart) Proteinuria of at least 1+ on a urine dipstick or ≥300 mg in a 24-h urine collection after 20 weeks of gestation
Severe preeclampsia (criteria for mild preeclampsia plus any of the conditions listed at right)	Blood pressure ≥160 mmHg systolic or ≥110 mmHg diastolic Urinary protein excretion of at least 5 g in a 24-h collection Neurologic disturbances (visual changes, headache, seizures, coma) Pulmonary edema Hepatic dysfunction (elevated liver transaminases or epigastric pain) Renal compromise (oliguria or elevated serum creatinine concentration; ≥1.2 mg/dL is considered abnormal in women with no history of renal disease) Thrombocytopenia Placental abruption, fetal growth restriction, or oligohydramnios
Eclampsia	Seizures in a preeclamptic woman that cannot be attributed to other causes
Superimposed preeclampsia	Sudden and sustained increase in blood pressure with or without a substantial increase in proteinuria New-onset proteinuria (≥300 mg in a 24-h urine collection) in a woman with chronic hypertension and no proteinuria prior to 20 weeks of gestation Sudden increase in proteinuria or a sudden increase in blood pressure in a woman with previously well-controlled hypertension or in a woman with elevated blood pressure and proteinuria prior to 20 weeks of gestation Thrombocytopenia, abnormal liver enzymes, or a rapid worsening of renal function Precise diagnosis is often challenging. High clinical suspicion is warranted, given the increased maternal and fetal/neonatal risks associated with superimposed preeclampsia
HELLP syndrome	Presence of hemolysis (H), elevated liver enzymes (EL), and low platelet counts (LP). HELLP syndrome may or may not occur in the presence of hypertension and is often considered a variant of preeclampsia
Gestational hypertension	New onset of sustained elevated blood pressure after 20 weeks of gestation in a previously normotensive woman (≥140 mmHg systolic or ≥90 mmHg diastolic on at least two occasions 6 h apart) No proteinuria

Epidemiology:-

A systematic review by the World Health Organization indicates that hypertensive disorders account for 16% of all maternal deaths in developed countries, 9% of maternal deaths in Africa and Asia, and as many as 26% of maternal deaths in Latin America and the Caribbean.10 Where maternal mortality is high, most of the deaths are attributable to eclampsia rather than preeclampsia.

Based on data from the United States National Hospital Discharge Survey, the prevalence of preeclampsia during admission for labor and delivery increased by 25% from 1987 to 2004; during the same period, the rate of eclampsia decreased by 22%, but this was not statistically significant. 1 Severe morbidity associated with preeclampsia and eclampsia includes renal failure, stroke, cardiac dysfunction or arrest, respiratory compromise, coagulopathy, and liver failure. In a study of hospitals managed by Health Care America Corporation, preeclampsia was the second leading cause of pregnancy-related intensive care unit admissions, after obstetric hemorrhage.

Fetal and neonatal effects- Fetal and neonatal outcomes related to preeclampsia vary around the world. Approximately 12–25% of fetal growth restriction and small-for-gestational-age infants as well as 15–20% of all preterm births are attributable to preeclampsia. The associated complications of prematurity are substantial and include neonatal deaths and serious long-term neonatal morbidity

One-quarter of stillbirths and neonatal deaths in developing countries are associated with preeclampsia/eclampsia. Infant mortality associated with preeclampsia is three times higher in low resource settings than in high-income countries, largely due to the lack of neonatal intensive care facilities.

Recurrence in subsequent pregnancies -Studies have reported a 7–20% chance of preeclampsia recurrence in a subsequent pregnancy.[62-64] This risk is further increased if a woman has had two prior preeclamptic pregnancies and is also influenced by the gestational age of onset.[65] Estimates of the recurrence of preeclampsia vary widely, depending on the quality of the diagnostic criteria used. In a study performed in Iceland using strict diagnostic criteria for preeclampsia and other hypertensive disorders, the estimated recurrence of preeclampsia or superimposed preeclampsia in a second pregnancy was 13%.[66]

Preclampsia and later-life cardiovascular disease Dr. Leon Chesley, a pioneer in the field of preeclampsia, and his coworkers demonstrated that, compared with controls, women who had eclampsia in any pregnancy after their first one had a mortality risk that was two- to fivefold higher over the next 35 years.[67] Following this early report, others demonstrated an association between preeclampsia and later-life cardiovascular disease and related mortality.

The risk of cardiovascular disease was increased eightfold in a Scandinavian population of healthy nulliparous women who developed preeclampsia severe enough to necessitate a preterm delivery.[68] In a cohort of women delivering in Jerusalem, there was a twofold higher risk of mortality at 24- to 36-year follow-up in women with prior preeclampsia than in women with no history of this diagnosis.18 The deaths were largely related to cardiovascular causes.

These findings have also been confirmed in other populations.Hypertension, dyslipidemia, insulin resistance, endothelial dysfunction, and vascular impairment have all been observed months to years after preeclampsia, further supporting the link between preeclampsia and subsequent cardiovascular disease.[69] It remains unresolved as to whether these common risk factors lead to the development of preeclampsia and later-life cardiovascular disease or whether preeclampsia itself may contribute to this future risk. On the basis of these data, preeclampsia should be considered a cardiovascular risk factor, and women with a history of preeclampsia should have ongoing, close surveillance to prevent and/or detect cardiovascular disease.

Risk factors for preeclampsia:

The etiology of preeclampsia is unknown: numerous models have attempted to explain its roles in the pathogenesis of immunology, cytokines, and growth factors, including tumor necrosis factor, endothelial damage, platelet dysfunction, and genetics has been implicated in the pathogenesis of preeclampsia.

The epidemiology of preeclampsia reflects a wide range of risk factors as well as the complexity and heterogeneity of the disease. Risk factors can be classified into pregnancy-specific characteristics and maternal preexisting features (Table 2). The incidence of preeclampsia is increasing in the United States and may be related to the higher prevalence of predisposing disorders such as hypertension, diabetes, obesity, and delay in childbearing, as well as to the use of artificial reproductive technologies, which results in a higher rate of multifetal gestation.[70,71]

Pregnancy-specific features:

Parity:

Nulliparity is a strong risk factor that almost triples the risk of preeclampsia (odds ratio 2.91, 95% confidence interval [95%CI] 1.28–6.61), according to a systematic review of controlled studies.[72] It is estimated that two-thirds of all cases occur in first pregnancies that progress beyond the first trimester.[73] New paternity also increases the risk of preeclampsia in a subsequent pregnancy. The association between primiparity and preeclampsia suggests an immunological mechanism, such that later pregnancies are protected against those paternal antigens.[74]

Supporting this concept, previous pregnancy loss, increased duration of sexual activity prior to pregnancy, or prolonged prepregnancy cohabitation confer a lower risk of preeclampsia.[75] Conversely, the risk of preeclampsia is increased with the use of barrier contraceptives, with new paternity, and with donor sperm insemination.[76]

Placental factors:

Excess placental volume, as occurs with hydatidiform moles and multifetal gestation, is also associated with the development of preeclampsia.[77,78] The disease process may occur earlier in the pregnancy and have more severe manifestations in such cases. The risk increases progressively with each additional fetus.

Maternal characteristics-

Age:

Extremes of childbearing age have been associated with preeclampsia.1However, once adjustments for parity are made in the younger age group (since most first pregnancies occur at a younger age), the association between younger age and preeclampsia is lost. Multiple studies demonstrate a higher incidence of preeclampsia among older women, independent of parity; however, many of these

do not control for preexisting medical conditions. After controlling for baseline differences, women who were 40 years of age or older had almost twice the risk of developing preeclampsia (risk ratios of 1.68 [95%CI 1.23–2.29] among primiparas and 1.96 [95%CI 1.34–2.87] among multiparas).[79]

Race:

The association between African-American race and preeclampsia has been confounded by the higher prevalence of chronic hypertension, often undiagnosed, in this group. While some studies demonstrate a higher risk of preeclampsia among African-American women, larger prospective studies that rigorously defined preeclampsia and controlled for other risk factors did not find a significant association between preeclampsia and African-American race.[80] More severe forms of preeclampsia may be associated with maternal non white race.[81]

Preexisting conditions:

Many of the maternal risk factors for preeclampsia are similar to those for cardiovascular disease. Preexisting hypertension, diabetes, obesity, and vascular disorders (renal disease, autoimmune conditions) are all associated with preeclampsia.[82] Risk is correlated with the severity of the underlying disorder. Women with underlying chronic hypertension have a 10–25% risk of developing preeclampsia compared with the general population of pregnant women.[83]

This risk is increased to 31% in women with a longer duration of hypertension (at least 4 years) or more severe hypertension at baseline.With pregestational diabetes, the overall risk of developing preeclampsia is approximately 21%.[84]

However, the risk is 11–12% with diabetes of less than 10 years' duration, which increases to 36–54% among women with longer-standing diabetes associated with microvascular disease.[85] The risk of preeclampsia is estimated at 20–25% in pregnant women with mild renal disease (serum creatinine of <1.5 mg/dL) but increases to greater than 50% in pregnant women with severe renal disease.[86] Preeclampsia also occurs more frequently among pregnant women with autoimmune conditions such as systemic lupus erythematosus and antiphospholipid antibody syndrome.

Obesity:

Elevated body mass index (BMI) is also associated with preeclampsia. Given the obesity epidemic in the United States and around the world, this is one of the largest attributable and potentially modifiable risk factors for preeclampsia. This will be discussed in further detail below.

Family history of preeclampsia:

A family history of preeclampsia nearly triples the risk of preeclampsia.

Smoking:

Paradoxically, cigarette smoking during pregnancy is associated with a reduced risk of preeclampsia,[87] possibly due to modulation of angiogenic factors.[88]

Table 2 **Common risk factors for preeclampsia.**

Category	Risk factors
Pregnancy-specific factors	Nulliparity
	Partner-related factors (new paternity, limited sperm exposure [e.g., barrier contraception])
	Multifetal gestation
	Hydatidiform mole
Preexisting maternal conditions	Older age
	African-American race
	Higher body mass index
	Pregestational diabetes
	Chronic hypertension
	Renal disease
	Antiphospholipid antibody syndrome
	Connective tissue disorder (e.g., systemic lupus erythematosus)
	Family or personal history of preeclampsia
	Lack of smoking

Box 4: Factors associated with an increased risk of pre-eclampsia (adapted from Duckitt et al[6])

- First pregnancy
- Pre-eclampsia in a previous pregnancy
- ≥ 10 years since previous pregnancy
- ≥ 40 years of age
- Body mass index ≥ 35 at booking in
- Family history of pre-eclampsia (especially mother or sister)
- Diastolic blood pressure ≥ 80 mm Hg at booking in
- Proteinuria at booking in
- Multiple pregnancy
- Underlying medical condition:
  Chronic hypertension
  Renal disease
  Diabetes
  Presence of antiphospholipid antibodies

Association between Anemia and Pre eclampsia:-

Hypertensive disorders of pregnancy and their complications rank as one of the major cause of maternal mortality and morbidity in the world. Amongst them, preeclampsia is emerging as one of the most common complication of pregnancy. Pre-eclampsia is a multi-system disorder of unknown aetiology, unique to pregnancy, with onset after 20 weeks of gestation.[89]

Although, the exact aetiology of preeclampsia is not yet known, many factors such as low education, primi parity, family history of hypertension, obesity,

younger and advanced maternal age and malnourishment are proven as its risk factors and evidence suggests that various other factors like severe anemia could also be a risk factor for development of preeclampsia and that cannot be ignored.

The uncertainty of cause of a disease of such common occurrence worldwide, make it to be more studied. Several studies have shown the association between severe anemia and preeclampsia and thus considers anemia as one of the main and treatable risk factor for preeclampsia.[90]

Anemia during pregnancy is a major public health problem especially in developing countries which itself increases the maternal mortality, in addition it further adds to the maternal and perinatal morbidity associated with preeclampsia, being a risk factor. It affects 41.8% women globally. In India overall prevalence of anemia is 65-70% and it contributes to 40 % maternal deaths.[91]

Gupta G et al did a case control study to evaluate correlation of anemia with severe preeclampsia. They found that Higher incidence of maternal complications abruption (8.88% v/s 0.5%), ARF (2.2% v/s 0.5%), PPH (2.8%), pulmonary edema (5%), CCF (3.3%), HELLP (1.6%), CVA (1.1%), pulmonary embolism and DIC in 0.5% and maternal mortality seen in cases. Perinatal complications like pregnancy wastage (22.8% v/s 7.8%), IUGR (55.8% v/s 32%), early neonatal death (7.5% v/s 2.4%), NICU admission (31.3% v/s 20.7%) were more in cases. Perinatal and maternal complications are significantly associated with

severity of anemia in preeclampsia women. Anemia being a easily detectable and modifiable risk factor, detection of anemia in early gestation can be a key to prevent or decrease the severity of preeclampsia.[92]

Anaemia during pregnancy is a major public health problem, especially in developing countries. It affects 41.8% of pregnant women globally, with the highest prevalence in Africa.[93] There is however significant variation in the prevalence of anaemia both within and between countries, necessitating a need for local data to help to improve preventive programmes. Anaemia during pregnancy, especially severe anaemia, is associated with increased maternal morbidity and mortality and contributes to 20% of the maternal mortality in Africa.[94]

Anaemia during pregnancy is associated with a negative impact on both the woman and neonate. Fetal anaemia, low birth weight (LBW), preterm birth and stillbirth have been associated with anaemia.[95] There is conflicting literature regarding the association between anaemia and perinatal outcomes. Some recent studies[96] have demonstrated a strong association between anaemia and adverse perinatal outcomes such as preterm delivery and LBW, while other previous studies found no association.[97]

A meta-analysis showed that anaemia during early pregnancy, but not during late pregnancy, is associated with slightly increased risk of preterm delivery

and LBW. Many studies have used different definitions and were undertaken in areas with a low prevalence of anaemia. There is therefore insufficient information to conclusively assess the effect of maternal anaemia on maternal and perinatal outcomes. Furthermore, most studies were not able to study anaemia according to its severity.[98]

Ali et al did a study on Severe anaemia is associated with a higher risk for preeclampsia and poor perinatal outcomes in Kassala hospital, eastern Sudan. They found the association between anaemia and preeclampsia and eclampsia. The prevalence of preeclampsia and eclampsia was significantly higher in women with severe anaemia (8.2% and 3.3%, respectively), table 2. The corrected risk for preeclampsia (OR = 3.6, 95% CI: 1.4-9.1, P = 0.007) increased only in severe anaemia, table 3.

Logistic regression analysis showed that maternal age was also a risk factor for preeclampsia, with a higher risk in women aged < 20 years (OR = 7.6, 95% CI: 2.9-19.9) and in women aged > 35 years (OR = 10.2, 95% CI: 3.2- 32.2). The risk for eclampsia was not increased in women with anaemia, table 3. There were three maternal deaths due to heart failure in the group with severe anaemia. The greater the severity of the anaemia during pregnancy, the greater the risk of preeclampsia, preterm delivery, LBW and stillbirth. Preventive measures should be undertaken to decrease the prevalence of anaemia in pregnancy.[99]

Complications of pre eclampsia:-

Box 2: Complications of pre-eclampsia

Central nervous system
- Eclampsia (seizures)
- Cerebral haemorrhage (stroke)
- Cerebral oedema
- Cortical blindness
- Retinal oedema
- Retinal blindness

Renal system
- Renal cortical necrosis
- Renal tubular necrosis

Respiratory system
- Pulmonary oedema
- Laryngeal oedema

Liver
- Jaundice
- HELLP syndrome (haemolysis, elevated liver enzymes, and lowered platelets)
- Hepatic rupture

Coagulation system
- Disseminated intravascular coagulation
- Microangiopathic haemolysis

Placenta
- Placental infarction
- Placental abruption

Baby
- Death
- Preterm birth
- Intrauterine growth restriction

The four major hypertensive disorders related to pregnancy are preeclampsia, chronic hypertension, preeclampsia superimposed upon chronic hypertension, and gestational hypertension. The development of hypertension and proteinuria in pregnancy is usually due to preeclampsia, particularly in a primigravida. These findings have typically been apparent in the latter part of the third trimester and progress until delivery, but some women develop symptoms in the latter half of the second trimester, or intrapartum, or the early postpartum period. Preeclampsia is characterized as mild or severe. Severe hypertension, coagulopathy, thrombocytopenia, liver function abnormalities, and fetal growth restriction are features of severe disease. Laboratory evaluation should assess hemoglobin/hematocrit and platelet count, and hepatic function, as well as assessment of fetal well-being and growth.[104]

Preeclampsia complications do arise in about 3 % of pregnancies, and all hypertensive disorders affect about 5–10 % of pregnancies. Hypertensive disorders are associated with higher rates of maternal, fetal, and infant mortality, and severe morbidity, especially in cases of severe preeclampsia, eclampsia, and hemolysis, elevated liver enzymes, and low platelets syndrome.[105]

Features of severe preeclampsia include severe proteinuria hypertension and symptoms of central nervous system dysfunction, hepatocellular injury thrombocytopenia, oliguria, pulmonary edema, cerebrovascular accident, and severe intrauterine growth restriction. Women with severe preeclampsia must be hospitalized to confirm the diagnosis, to assess the severity of the disease, to monitor the progression of the disease, and to try to stabilize the disease.[106]

Preeclampsia is a pregnancy-specific hypertensive syndrome associated with significant morbidity and mortality in mother and baby. With the increasing understanding of the disease process, the number of complications, as well as the maternal and perinatal deaths, have fallen over the past few decades in the developed countries. In other parts of the world, the rates of mortality and morbidity still remain high.[107]

One of the rare effects of severe preeclampsia on the eye is sudden loss of vision due to involvement of the occipital cortex or the retina [6]. Subcapsular hepatic hematoma caused mainly by the development of disseminated intravascular coagulation is one of the rare complications experienced with severe preeclampsia and eclampsia.[108]

Globally, preeclampsia and eclampsia account for 10–15 % of maternal deaths. A majority of deaths in developing countries result from eclampsia, while in developed countries, complications of preeclampsia are more often the cause. Pulmonary edema is a rare, serious problem, resulting in complications in as many as 3 % of cases of severe preeclampsia.[109]

Screening and Diagnosis:-

Assessment usually begins when a woman presents to a general practitioner or midwife requesting antenatal care. Women at high risk are then offered further visits and testing, with referral for specialist care. Screening of low risk women is based primarily on blood pressure measurement and urine analysis. The search for additional tests continues. Despite initial optimism for uterine artery Doppler ultrasonography, it has only limited accuracy in predicting preeclampsia.[110]

Women with blood pressure > 140/90 mm Hg should be referred for specialist assessment. A relative rise in blood pressure or oedema is not related to outcome, and neither is an indication for routine screening.[111] Because of cardiovascular changes, automated blood pressure monitors systematically underestimate blood pressure in pregnancy and preeclampsia. If used, they should be calibrated regularly against a mercury sphygmomanometer (box 5).[112]

If possible, proteinuria should be confirmed in a 24 hour collection. The diagnosis of pre-eclampsia is more certain if other organ systems are implicated Onset of pre-eclampsia may be rapid, and prompt diagnosis and treatment often depends on awareness among women and primary care workers of these signs and symptoms.

Box 5: How to measure blood pressure during pregnancy

- Mercury sphygmomanometers are preferable to automated blood pressure monitors
- If automated devices are used they should be calibrated, and checked regularly, against a mercury sphygmomanometer
- Use an appropriate size cuff
- Woman should be seated or lying at 45° angle, with arm at level of the heart
- Record blood pressure to the nearest 2 mm Hg
- Use phase V Korotkoff sound (sound disappearance) to measure diastolic blood pressure

Management of Pre eclampsia:-

As the cause of pre-eclampsia is unclear, treatment remains symptomatic with little evidence that any intervention alters the underlying pathophysiology. Complementary medicines, such as Chinese herbal medicines, offer alternative strategies that are attracting growing interest, but none has been evaluated in randomised trials.

Once blood pressure rises above a certain level it may lead to direct vascular damage, which leads to life threatening complications such as renal failure, stroke, and fetal distress. This risk is not specific to pregnancy. Antihypertensive drugs are mandatory for systolic blood pressure ≥ 170 mm Hg or diastolic pressure ≥ 110 mm Hg, although lower thresholds are advisable if signs or symptoms are present. The aim is to bring about a smooth reduction in blood pressure to levels that are safe for both mother and fetus, avoiding sudden drops.

Antihypertensive drugs do lower high blood pressure during pregnancy, but with insufficient evidence about effects on other outcomes to conclude that one drug is preferable to another (fig B on bmj.com). Best avoided are diazoxide at high doses, since it is associated with a greater risk of hypotension and caesarean section than labetalol, and the serotonin receptor antagonist ketanserin, which led to far more persistent hypertension than hydralazine.54

An alternative analysis suggested avoiding hydralazine as first line treatment but this review included quasi-random designs and pooled studies comparing hydralazine with various drugs, regardless of whether they were likely to be better or worse. There has been concern about the combined use of nifedipine and magnesium sulphate, but this seems unfounded.[113]

Magnesium sulphate is clearly the anticonvulsant of choice for treating eclampsia, with substantial reductions in the risk of further seizures compared with diazepam, phenytoin, and lytic cocktail. It is also better at preventing maternal death than diazepam. Compared with phenytoin, magnesium sulphate has a lower risk of pneumonia and ventilation, as well as being safer for the baby. Lytic cocktail has no place in treatment of eclampsia. Training of healthcare staff by means of practice drills and on-site simulation may also improve care.

The only definitive "cure" for pre-eclampsia is to deliver the placenta. However, the risk of hypertension or pre-eclampsia does not resolve immediately. Pre-eclampsia and eclampsia can both present for the first time after the birth. Whether antihypertensive drugs should be offered routinely after delivery to women who had antenatal hypertension is unclear, as is the choice of drug.

Materials and Methods:-

Study design:

This was a Hospital based Prospective analytical observational study

Study setting:

The present study was carried out in the Department of Obstetrics and Gynecology at Government Mohan kumaramangalam Medical College and Hospital, Salem.

Study period:

This study was conducted during the period of January 2020 to June 2021 for almost for the period of two years.

Sample size:

200 Anaemic pregnant women reporting to antenatal clinic or admitted to labour ward was included in this study.

Study population:

Patients admitted in Antenatal ward & Labour ward with hemoglobin value less than 11gms and after 20 weeks of gestation were selected for the study after the informed Oral and written consent in the local language with the following inclusion and exclusion criteria's.

Inclusion criteria –

- Antenatal women diagnosed as anemia in pregnancy
- Antenatal women diagnosed as gestational hypertension,pre eclampsia

Exclusion criteria –

- Patients who are not willing to participate in this study

Study procedure and data collection:

According to the severity of anemia and the gestational age, patients were treated with oral iron, parenteral iron,or by blood transfusion. All preeclampsia patients were classified in to mild and severe type, according to the severity gestational age, they were treated with antihypertensives, prophylactic anticonvulsant regimen,and termination of pregnancy. They were followed through the antepartum,intrapartum,and post partum period .

All the routine haematological and biochemical investigations were carried out, complete haemogram and peripheral blood smear. Urine routine examination and microscopy and culture sensitivity LFT,LDH,RFT,Urine albumin, Serum Iron/Folate/ferritin in selected patients, Ultrasound for fetal wellbeing. Patient history, socio demographic details, BP monitoring, Anthropometric measurements, details such as mode of delivery, type of delivery. Perinatal follow up of new born, APGAR, Admission to NICU, Maternal morbidity and mortality.

The data collection was done using pre-validated questionnaire and interviews which was scheduled to take place during pregnancy while admitted in ward and by means of phone/mobile contacts after discharge.

Statistical Analysis:

Data was entered in Ms excel and imported into SPSS 18 Software package. Descriptive statistics like mean, standard deviations and percentage proportions were used to describe baseline study participant parameters. Parametric tests were used to analyse the parametric data if it passed the tests of normality; if it failed then non-parametric tests were used for analysis. Chi-square test was used to analyse categorical data.

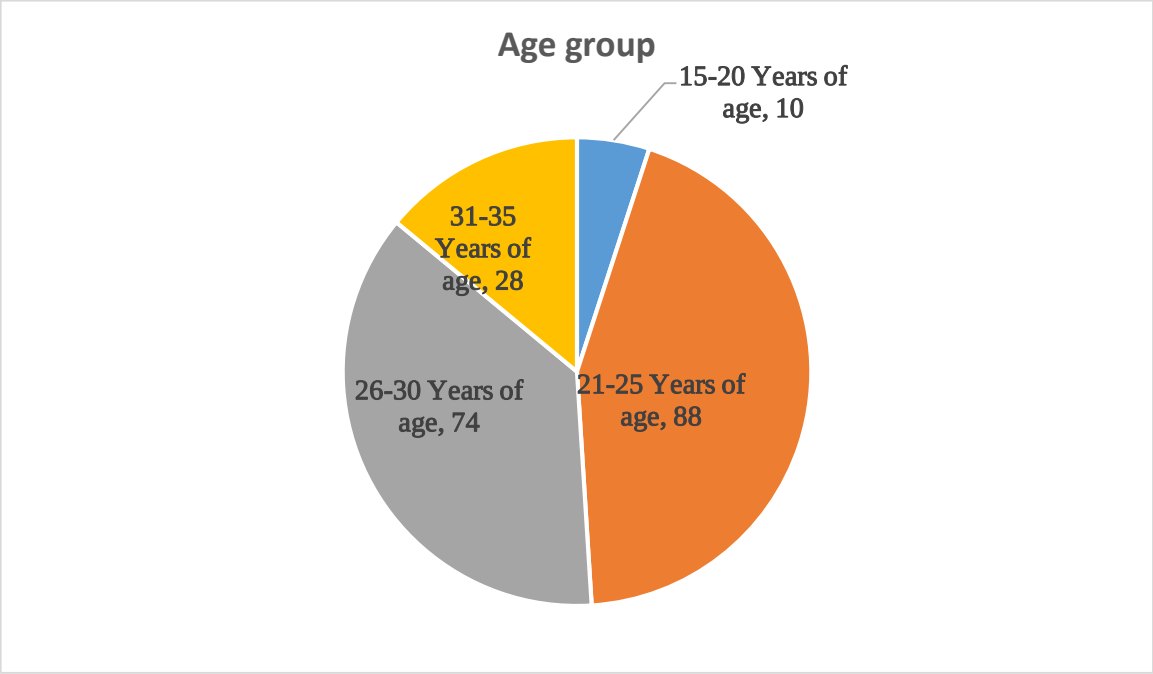
Ethics:

This study was conducted after obtaining the Intuitional Ethics and Research Committee approval.

Results:-

Table:1 Age wise distribution of participants

Age wise distribution		
Age group	Frequency	Percentages
15-20 Years of age	10	5%
21-25 Years of age	88	44%
26-30 Years of age	74	37%
31-35 Years of age	28	14%

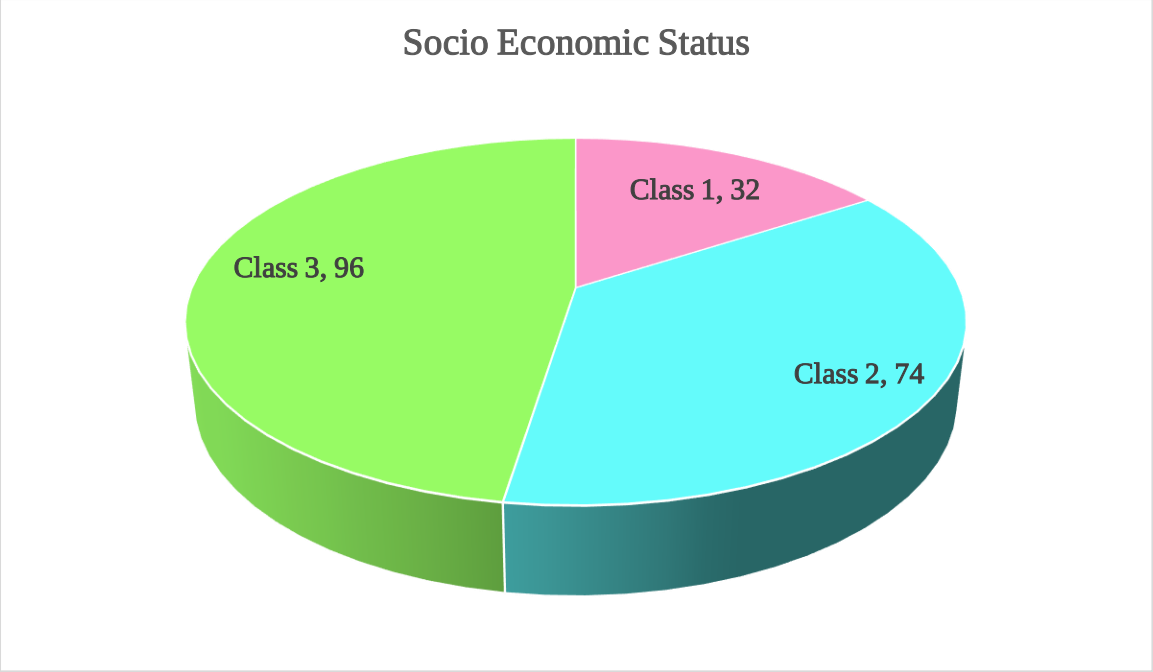


As seen in the above table, the age wise distribution was shown. Out of 200 pregnant mothers in this study, 10 were in 15-20 years of age, 88 belongs to the age group of 21-25 years of age. 74 were belongs to 26-30 years of age and 28 were belongs to the age group of 31-35 years of age.

Maximum number of the patients in the study population were in the age group of 21 to 25 years of age. The participant's age ranges from minimum 19 years of age to 32 years of age. The mean age of the pregnant mothers in our study was 25.73 $\pm$ 3.8 years of age.

Table:2 Frequencies of socio economic status

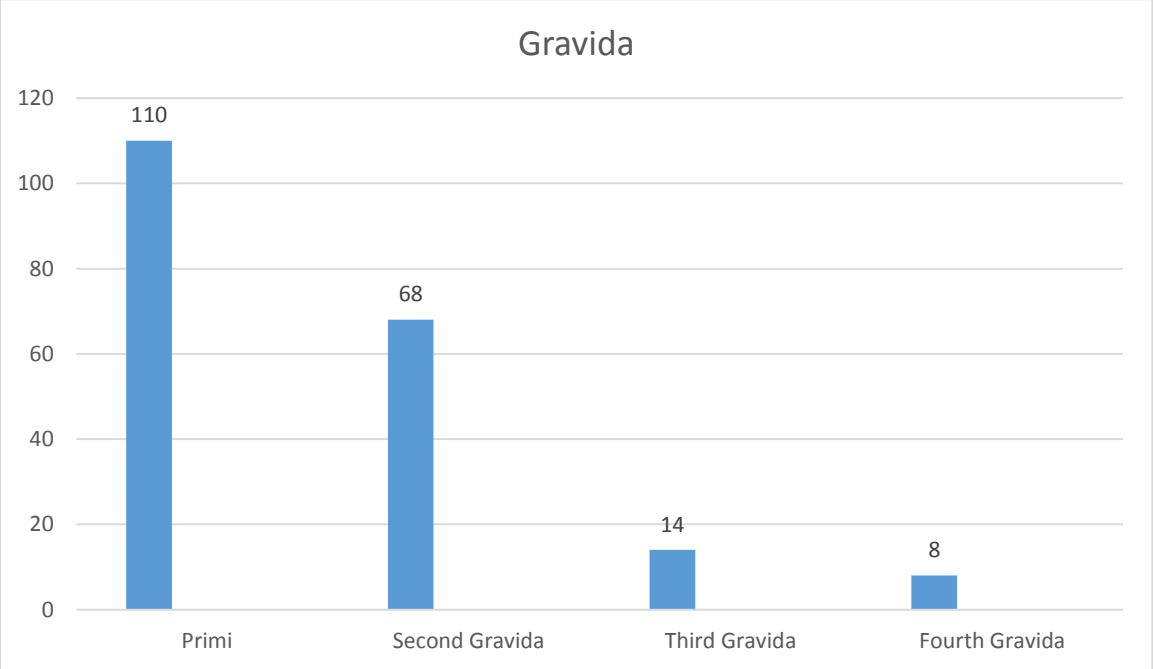
Frequencies of Socio Economic Status		
Status	Frequency	Percentages
Class 1	32	16%
Class 2	74	37%
Class 3	94	47%



Regarding the Socio economic status, Out of 200 Anaemic Pregnant mothers in this study, and 32 pregnant women belonged to Class 1, 74 belonged to Class 2 and majority of them belonged to class 3 with 94 cases. The Socio Economic status was classified based on the Modified B G Prasad's Classification.

Table:3 Distribution of gravida

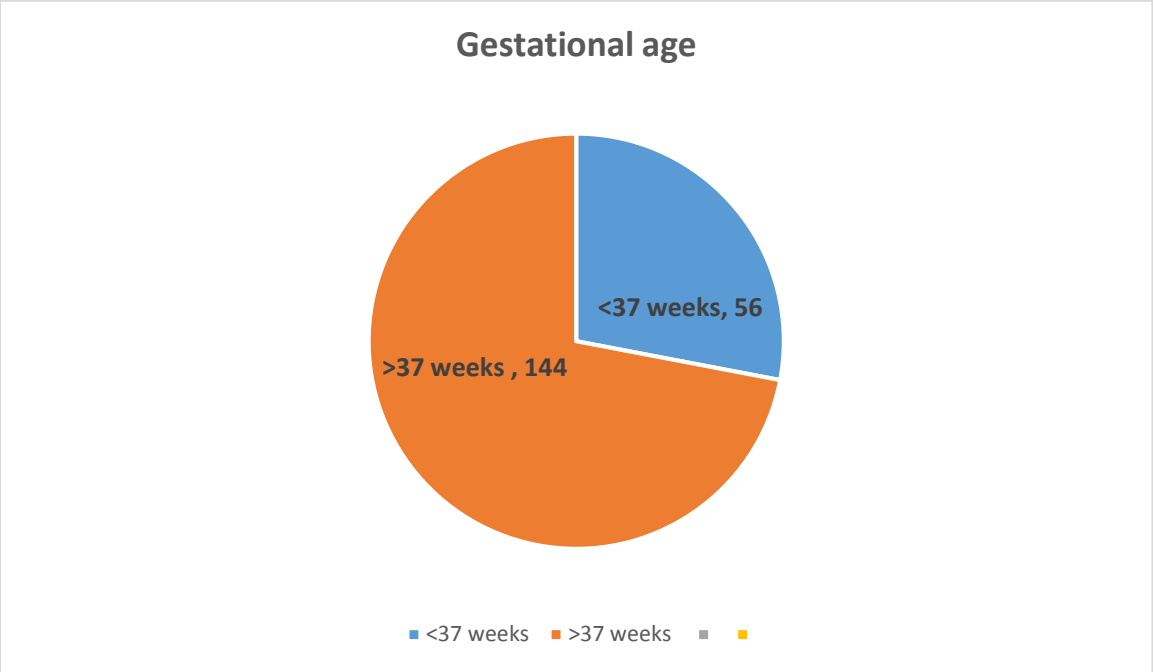
Distribution of Gravida		
Parity	**Frequency**	**Percentages**
Primi	110	55%
Second Gravida	68	34%
Third Gravida	14	7%
Fourth Gravida	8	4%



As seen above the distribution of gravida, 110 were Primi, 68 were second gravida, 14 were third gravida and 8 were in fourth gravida. More than half of the participants were in Primi.

Table:4 Distribution of gestational age

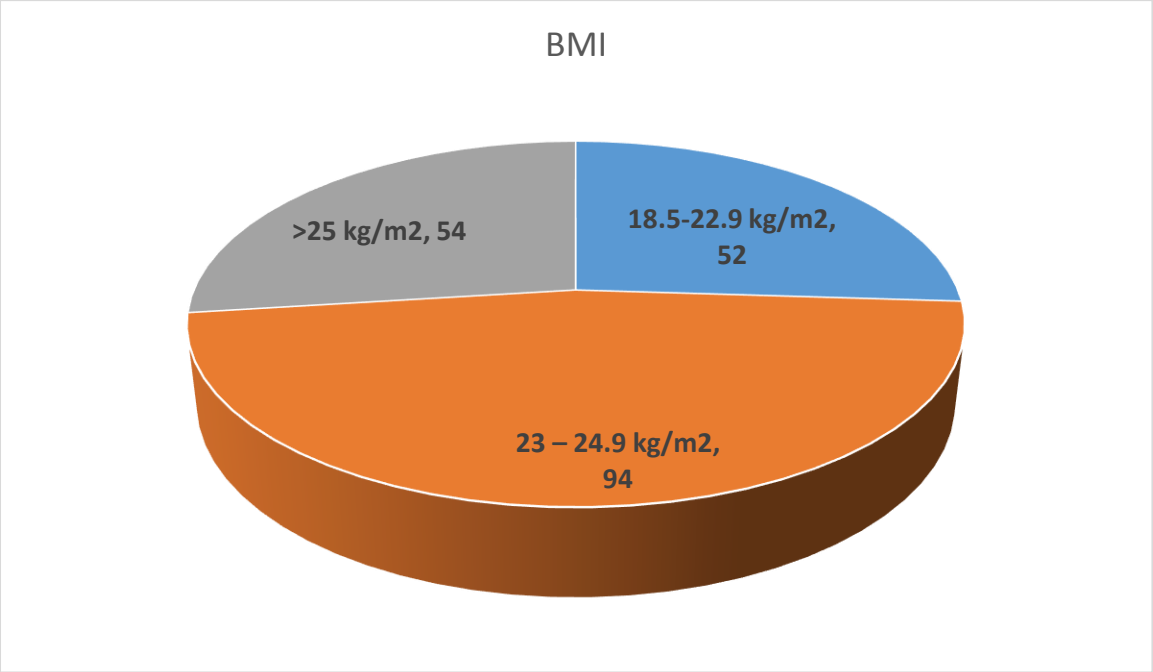
Gestational age		
	Frequency	Percentages
<37 weeks	56	28%
>37 weeks	144	72%



As seen above the gestational ages of the study participants ,56 were in less than 37 weeks and 144 were present above the 37 weeks of gestational age.

Table :5 Body mass index of all participants

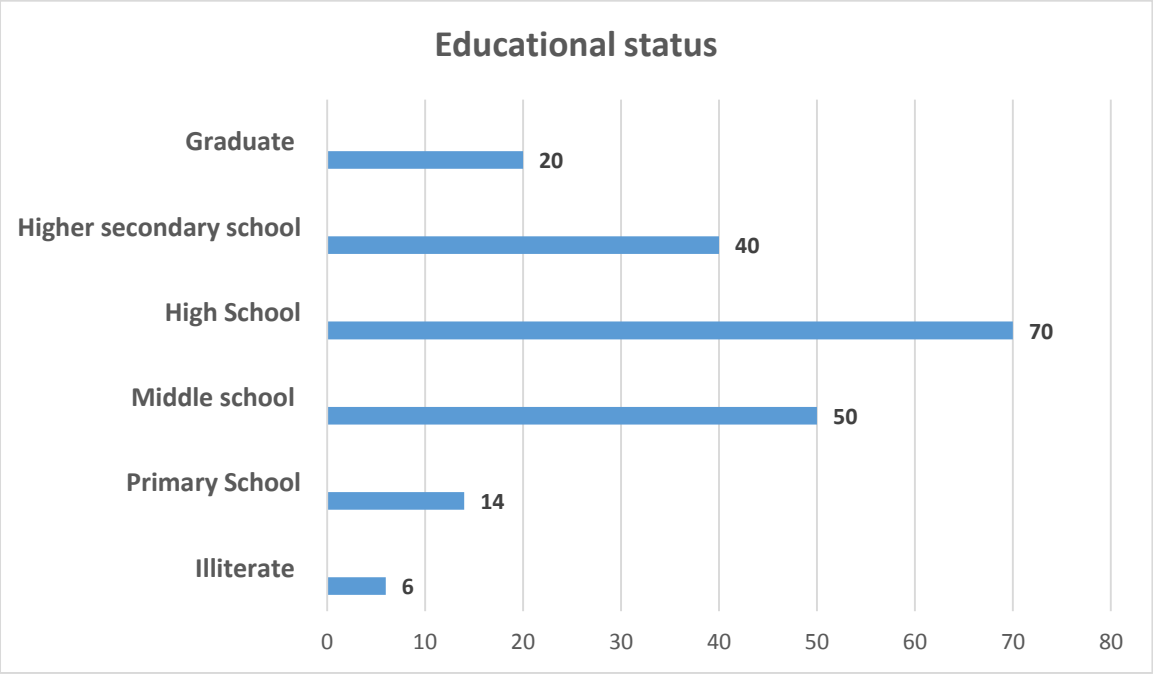
Body Mass Index		
BMI	Frequency	Percentages
18.5-22.9 kg/m^2	52	26%
23 – 24.9 kg/m^2	94	47%
>25 kg/m^2	54	27%



Regarding the Body Mass index of the study participants, about 52 were in Normal BMI, 94 were in Over Weight and 54 were in obese.

Table :6 Educational status of study population

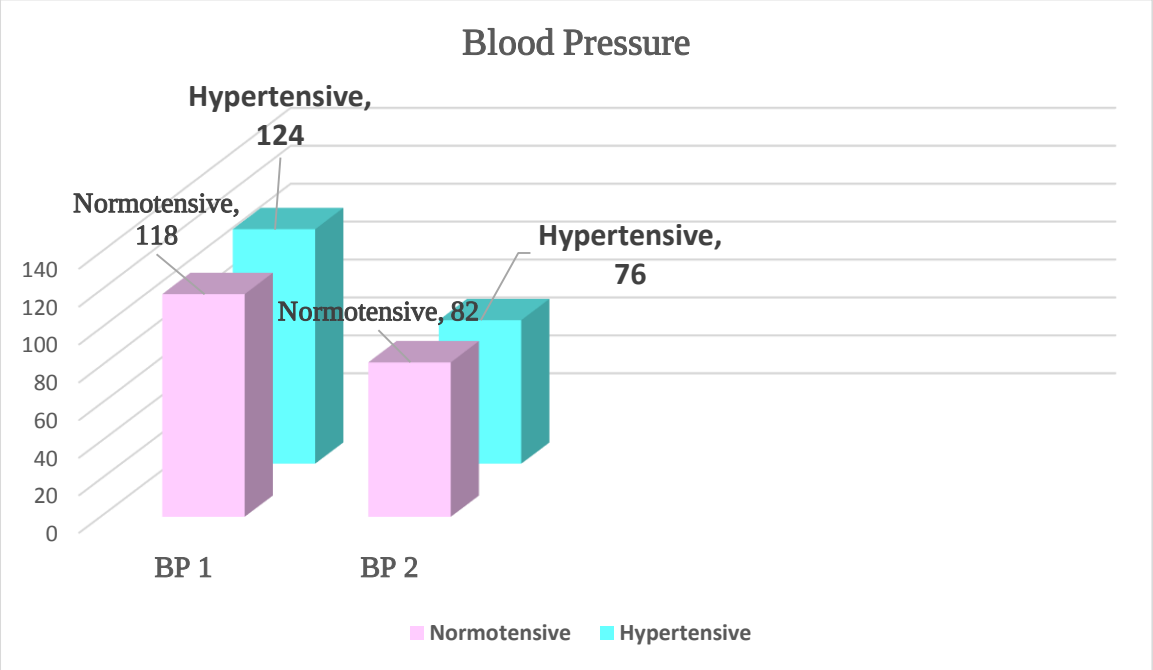
Educational status		
Status	Frequency	Percentages
Illiterate	6	3%
Primary School	14	7%
Middle school	50	25%
High School	70	35%
Higher secondary school	40	20%
Graduate	20	10%



Regarding the educational status of the study participants, about 6 were illiterate, 14 went to primary school, 50 were studied up to middle school, 70 reported to complete high school, 40 of them have studied upto higher secondary education and 20 were graduated.

Table: 7 Distribution of blood pressure

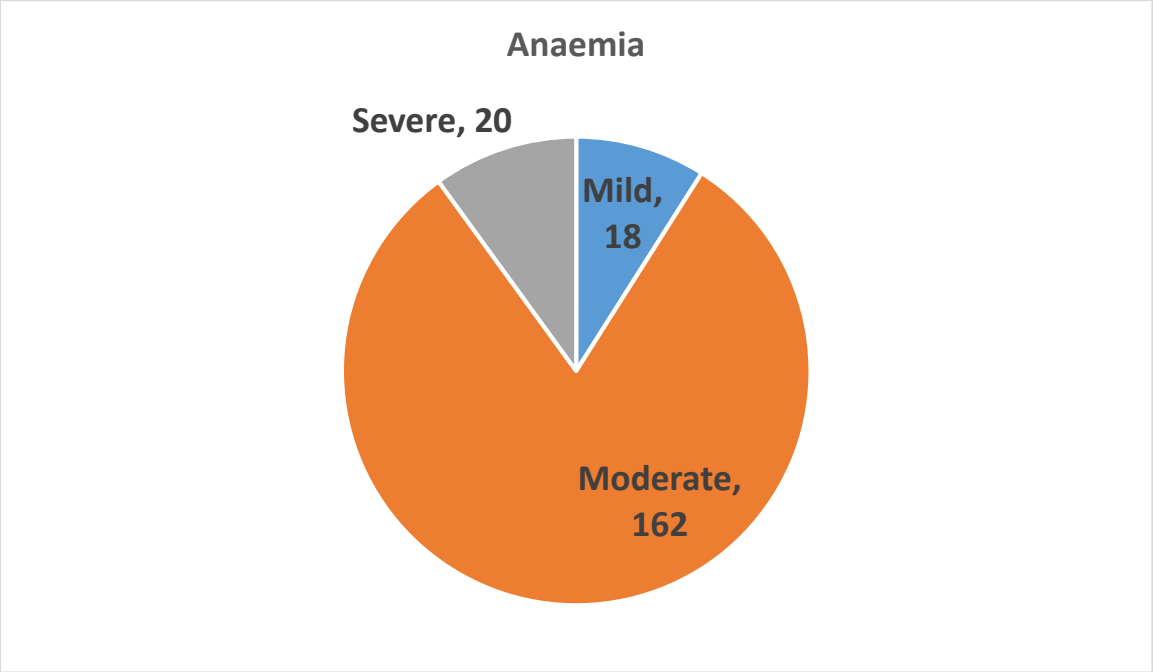
Distribution of Blood Pressure			
	BP 1	BP 2	P Value
Normotensive	118	124	
Hypertensive	82	76	0.5413



As seen above the distribution of Blood Pressure, for the first BP measurement about 118 were normotensive and 82 were hypertensive and for the second BP measurement 124 were Normotensive and 76 hypertensive. The association was statistically insignificant (0. 5413).

Table: 8 Severity of anemia in study population

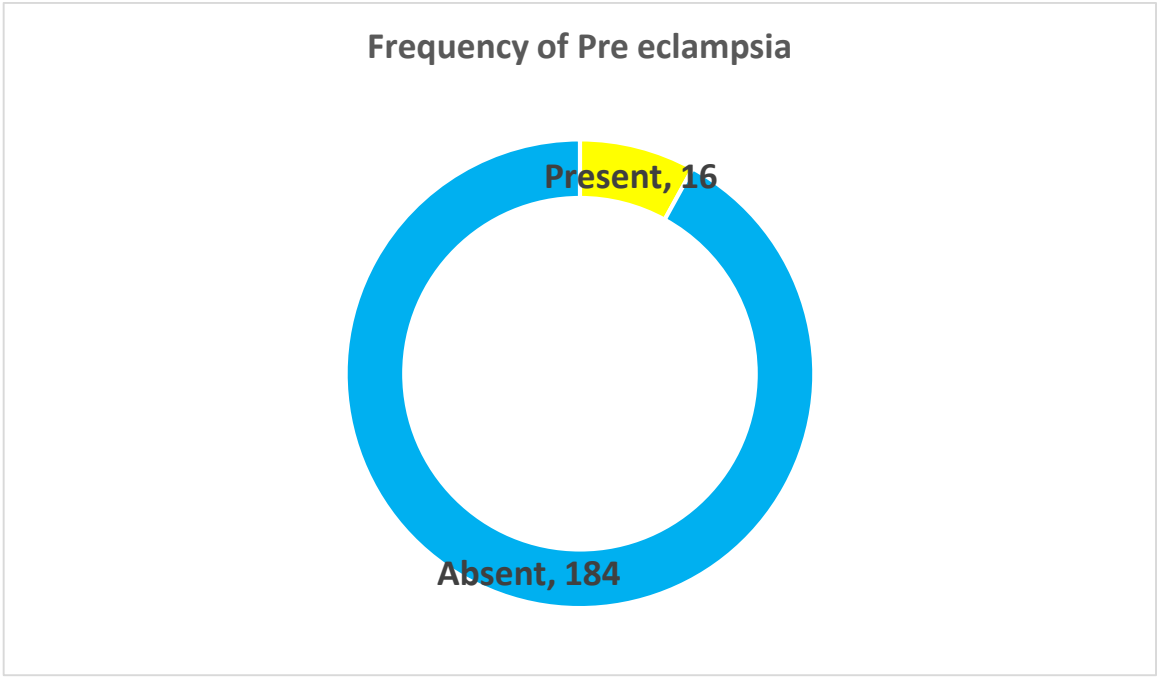
Severity of Anaemia		
Haemoglobin	**Frequency**	**Percentages**
10-11	18	9%
7-10	162	81%
<7	20	10%



Regarding the severity of the anaemia, About 18 were mild anaemic, majority of the participants were moderately anaemic 162 and 20 were severely anaemic.

Table: 9 Frequencies of preeclampsia

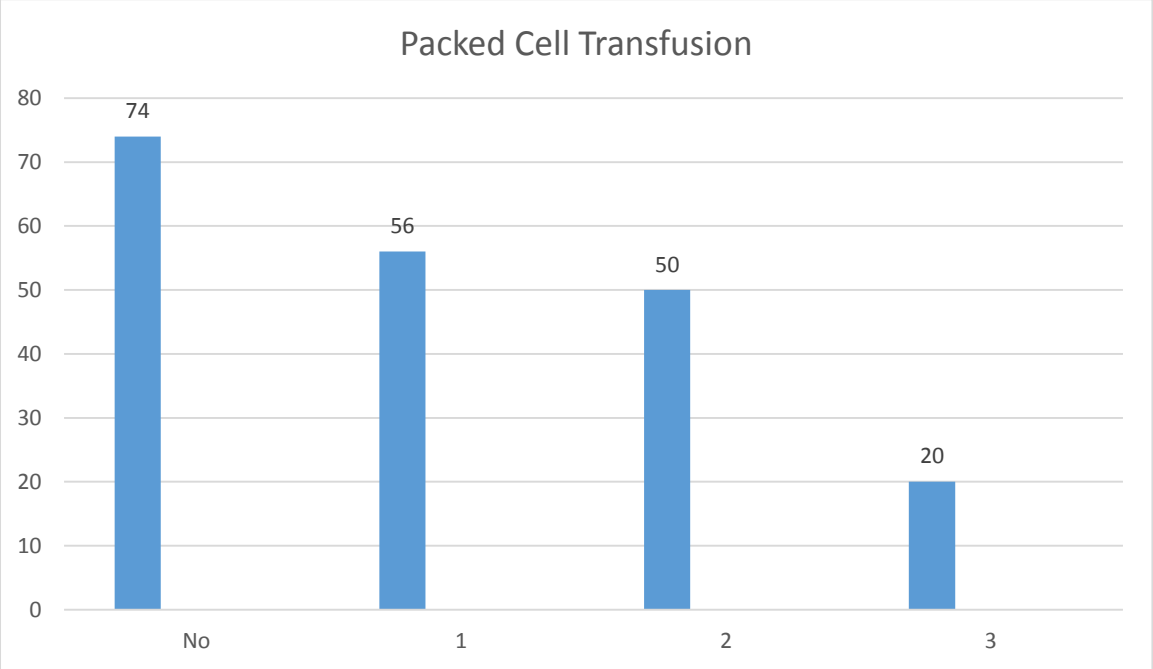
Frequency of Pre eclampsia	
Present	16
Absent	184



The frequency of pre eclampsia among 200 anaemic pregnant were found to 16 had pre eclampsia and remaining 184 didn't develop pre eclampsia.

Table: 10 Packed cell transfusion in study population

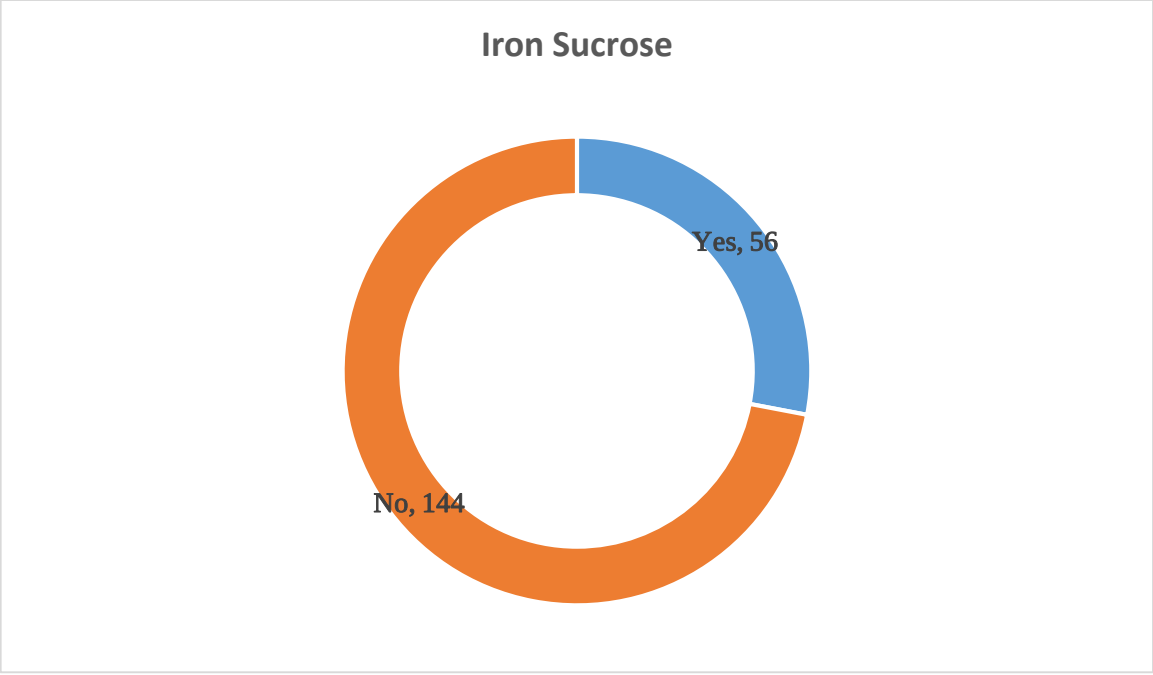
Packed Cell Transfusion		
N	Frequency	Percentages
No	74	37%
1	56	28%
2	50	25%
3	20	10%



Regarding the packed cell transfusion, about 74 didn't required Packed cell transfusion, 56 had one packed cell transfusion, 50 had 2 packed cell transfusion and 20 had 3 packed cell transfusion.

Table: 11 Distribution of Iron Sucrose

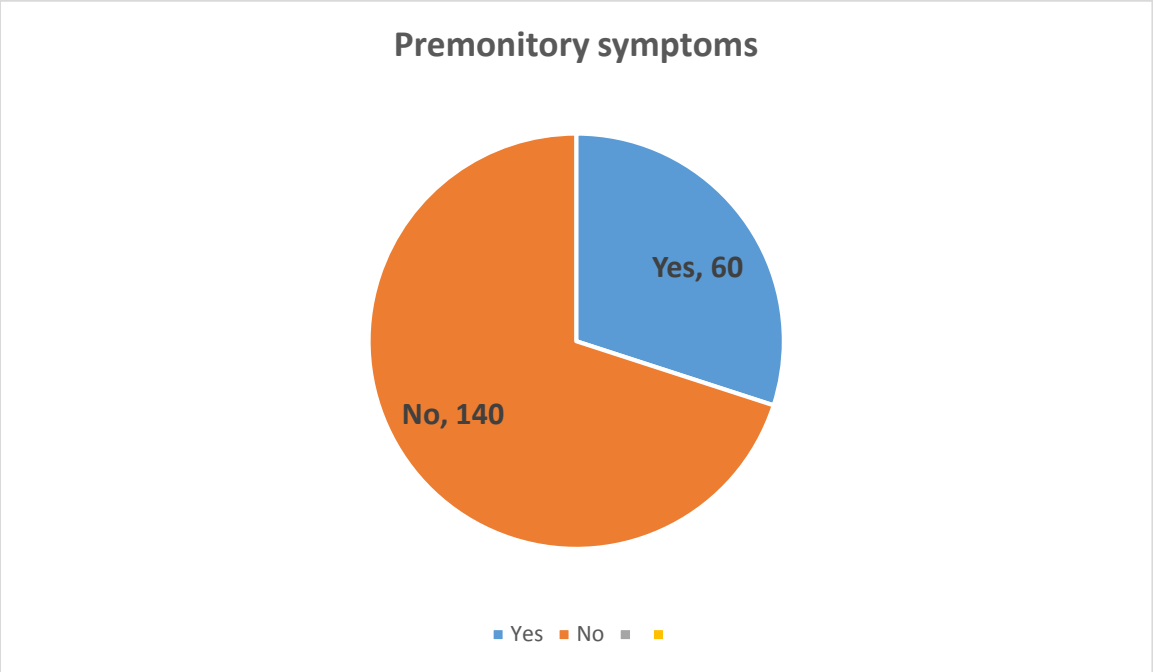
Distribution of Iron Sucrose		
	Frequency	Percentages
Yes	56	28%
No	144	72%



Distribution of Iron sucrose among the study participants were, about 56 had iron sucrose transfusion and 144 didn't had the iron sucrose.

Table: 12 Distribution of premonitory symptoms

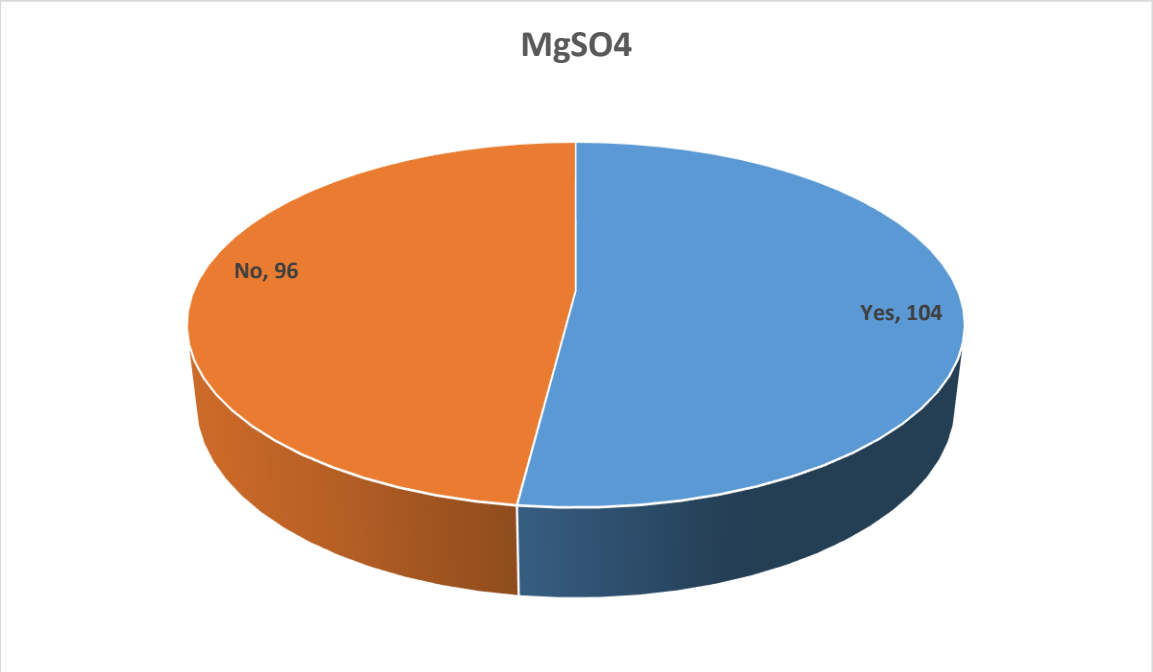
Premonitory Symptoms		
Symptoms	**Frequency**	**Percentages**
Yes	60	30%
No	140	70%



Distribution of Premonitory symptoms among the study participants were, about 60 had Premonitory symptoms and 140 didn't had premonitory symptoms.

Table :13 Distribution of MgSO4 requirement

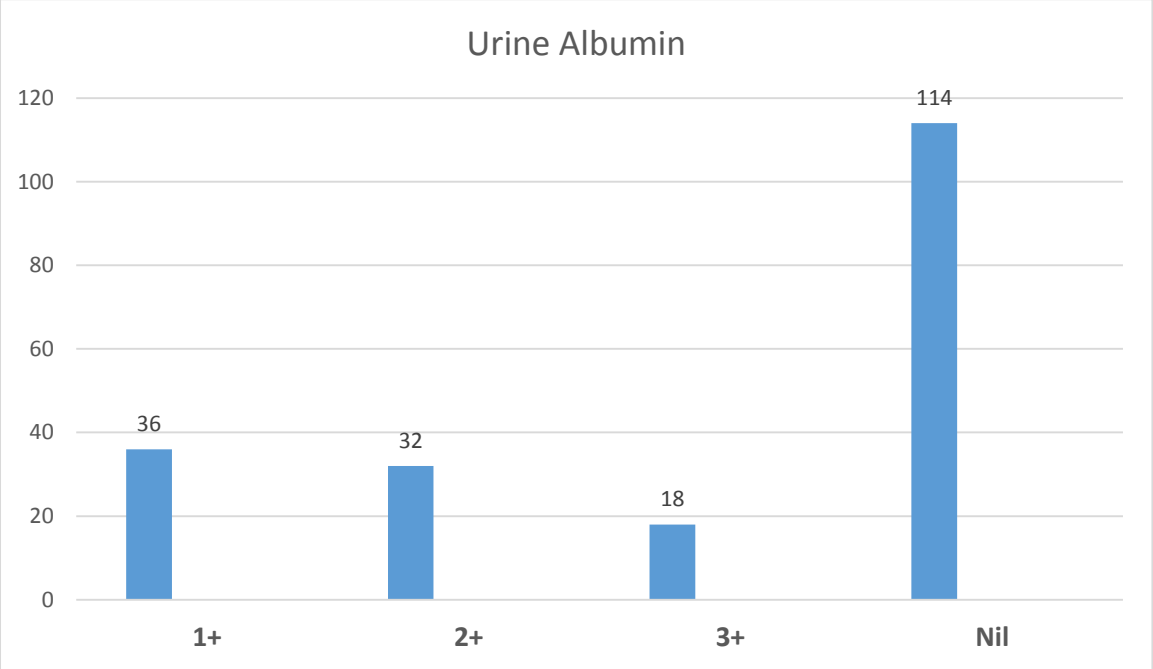
Distribution of MgSO4 requirement		
	Frequency	Percentages
Yes	104	52%
No	96	48%



Frequency of MgSO4 among the study participants were, about 104 had MgSO4 and 96 didn't had.

Table: 14 Distribution of urine albumin

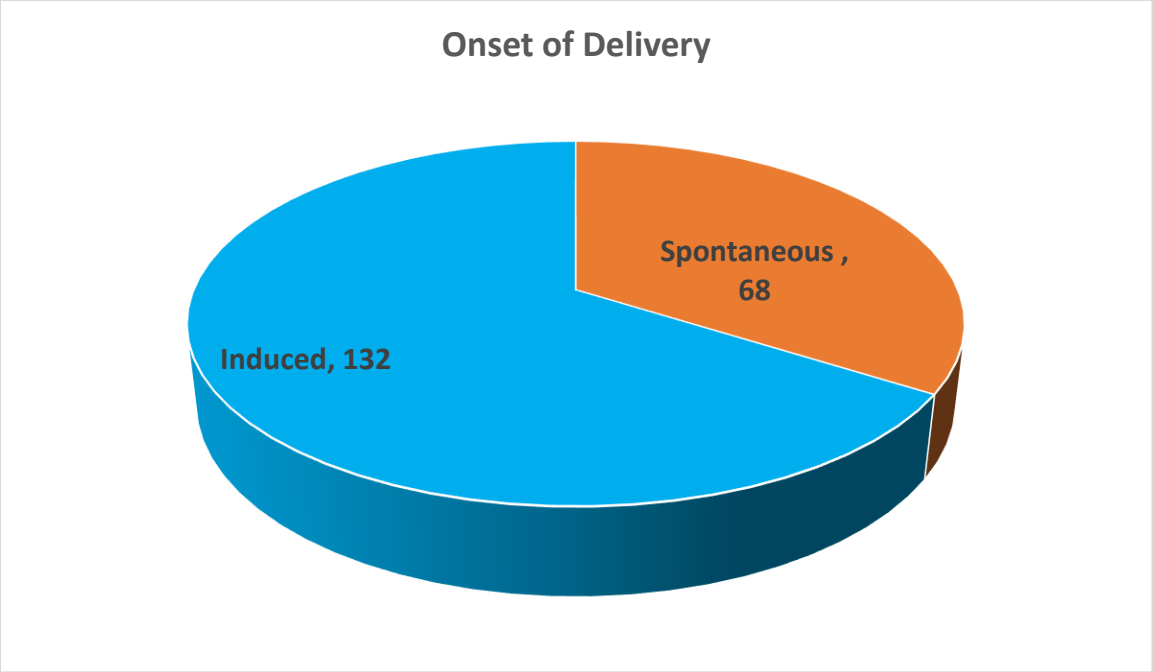
Distribution of Urine Albmin		
	Frequency	**Percentages**
1+	36	18%
2+	32	16%
3+	18	9%
Nil	114	57%



Regarding the urine albumin profile, 36 had 1+, 32 had 2+ , 18 had 3+ and 114 had NIL.

Table 15: Mode of onset of delivery

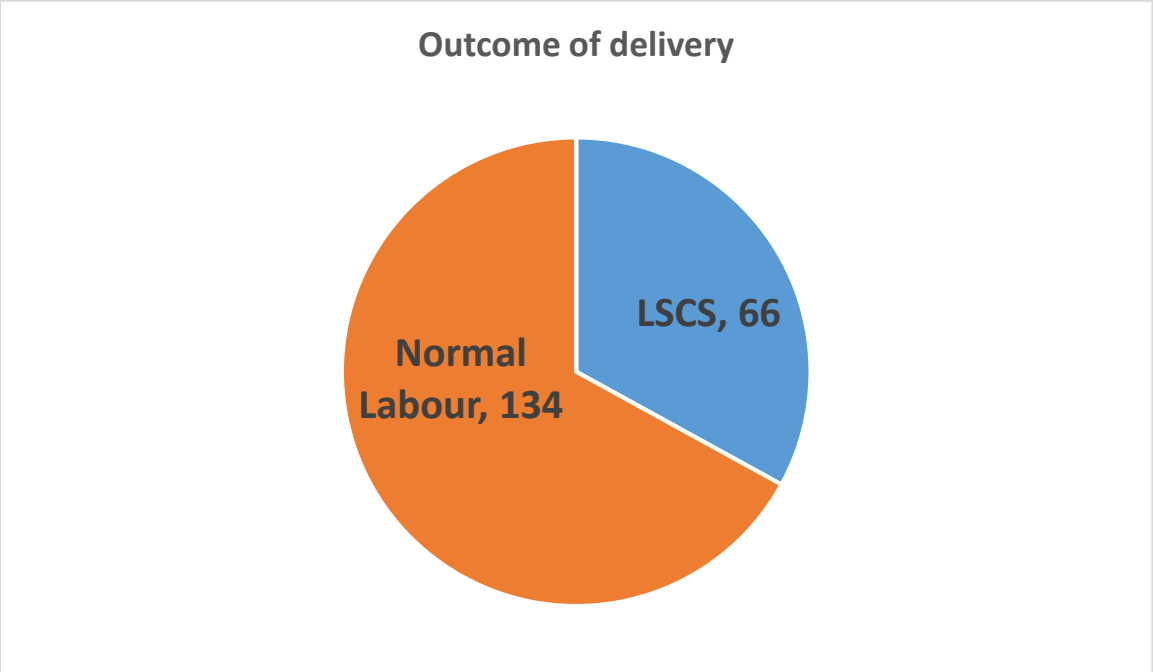
Onset of Delivery		
	Frequency	Percentages
Spontaneous	68	34%
Induced	132	66%



Out of 200 anaemic pregnant women in this study, 68 had the spontaneous delivery and 132 had induced labour.

Table: 16 Outcome of delivery

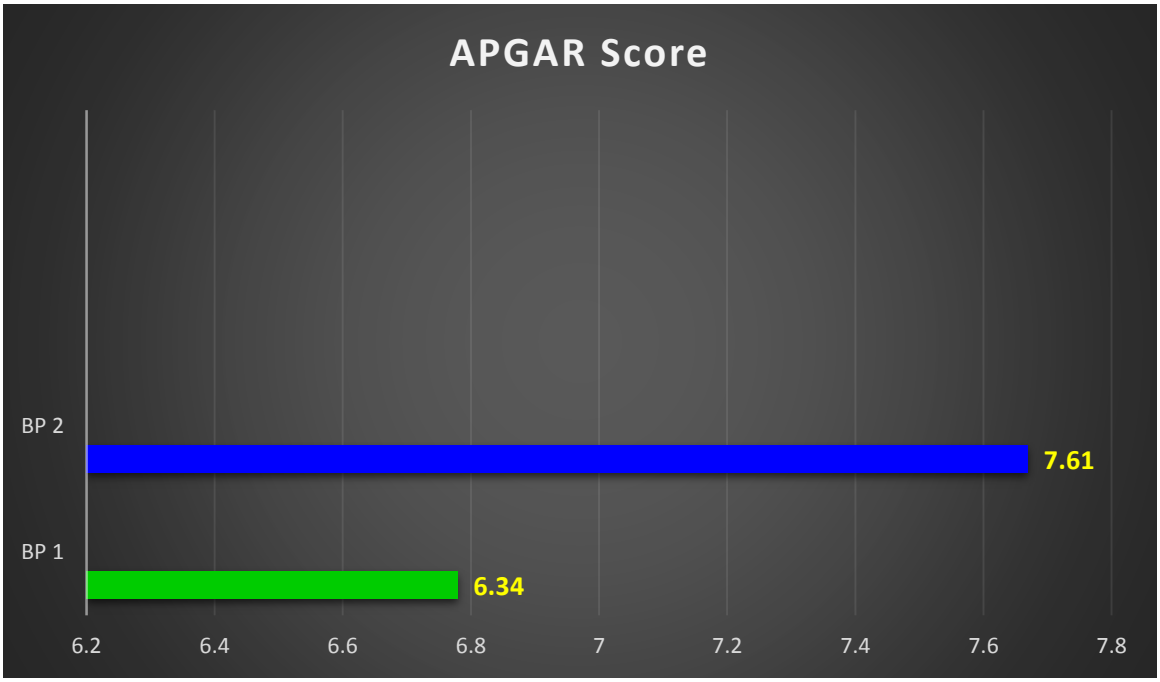
Outcome of delivery		
	Frequency	Percentages
LSCS	66	33%
Normal Labour	134	67%



The frequencies of outcome of delivery was, out of 200 anaemic pregnant women in this study, for 66 pregnant women LSCS was done and 134 had Normal labour.

Table: 17 APGAR score

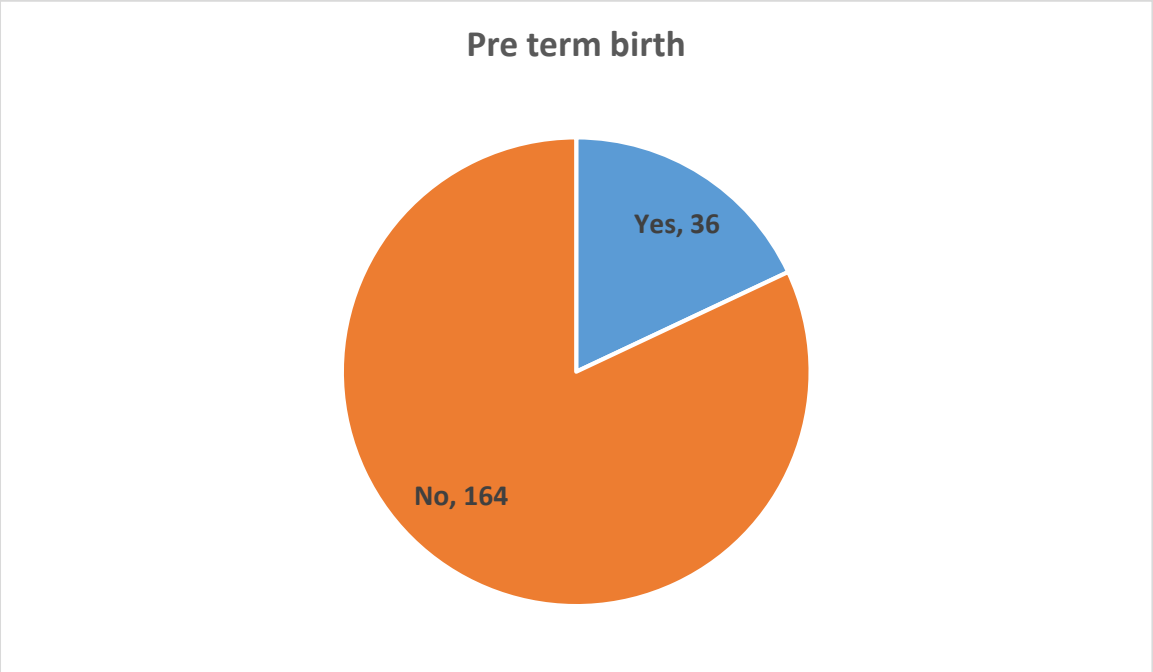
APGAR Score			
	1 Min APGAR	5 Min APGAR	P value
Mean APGAR score	6.34± 0.4	7.61 ±0.9	<0.001



Regarding the APGAR Score of the new-borns, the mean APGAR score at the 1 Min was 6.34± 0.4 and Mean APGAR score at 5th Min was 7.61± 0.9. The difference was statistically significant since the p value is <0.001

Table: 18 Preterm birth

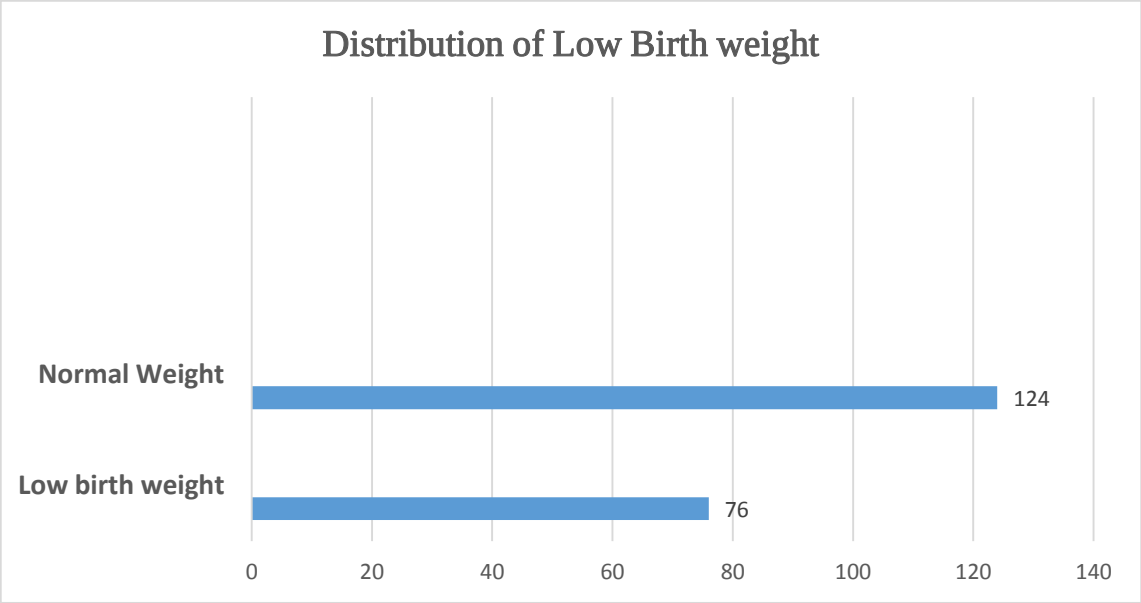
Pre term birth		
	Frequency	Percentages
Yes	36	18%
No	164	82%



The frequencies of Pre term delivery was, out of 200 anaemic pregnant women in this study, for 36 pregnant women had Pre term delivery and 164 had term baby.

Table: 19 Distribution of low birth weight

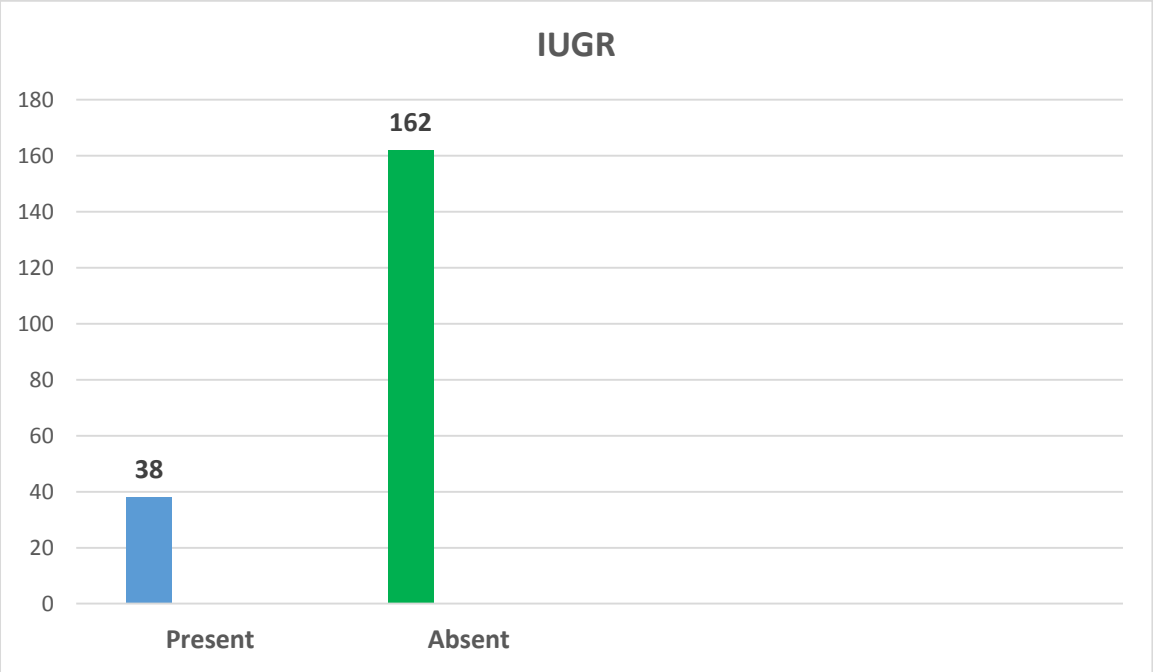
Distribution of Low Birth weight		
	Frequency	Percentages
Low birth weight	76	38%
Normal Weight	124	62%



Regarding the distribution of Low birth weight among the 200 anaemic pregnant women, 76 had low birth weight babies less than 2.5kgs and remaining 124 of the mothers had normal weighed babies.

Table: 20 Frequencies of IUGR

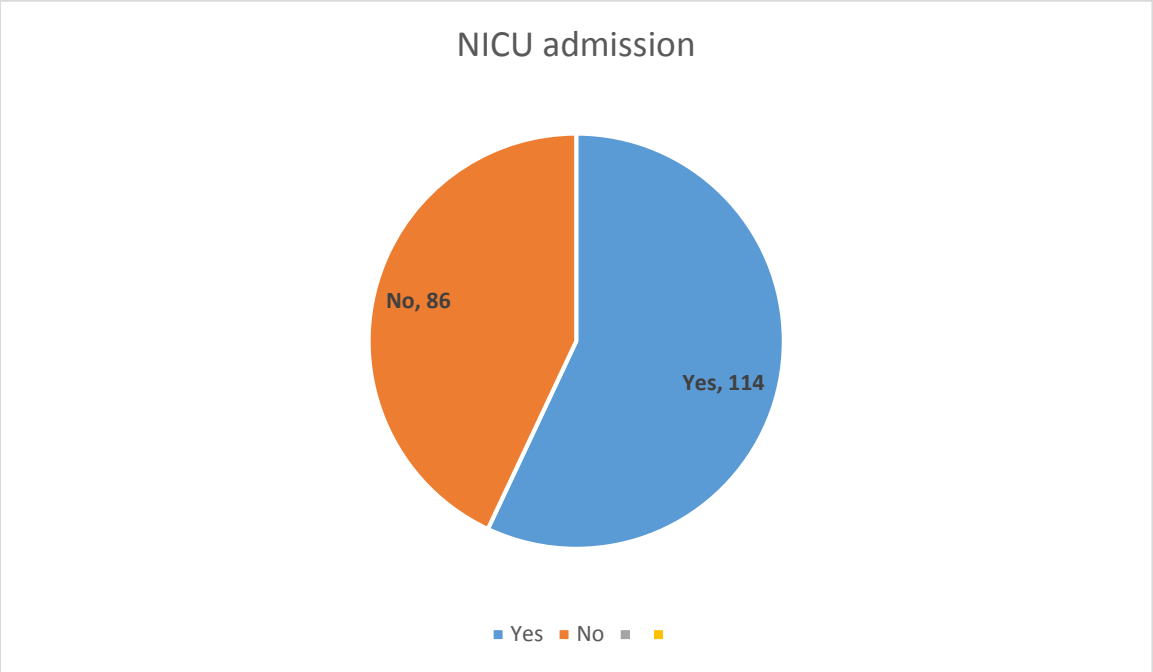
Frequencies of IUGR		
	Frequency	Percentages
Present	38	19%
Absent	162	81%



Regarding the frequencies and percentages of IUGR among the 200 anaemic pregnant women, 38 had IUGR and remaining 162 didn't have IUGR.

Table: 21 Admission to NICU

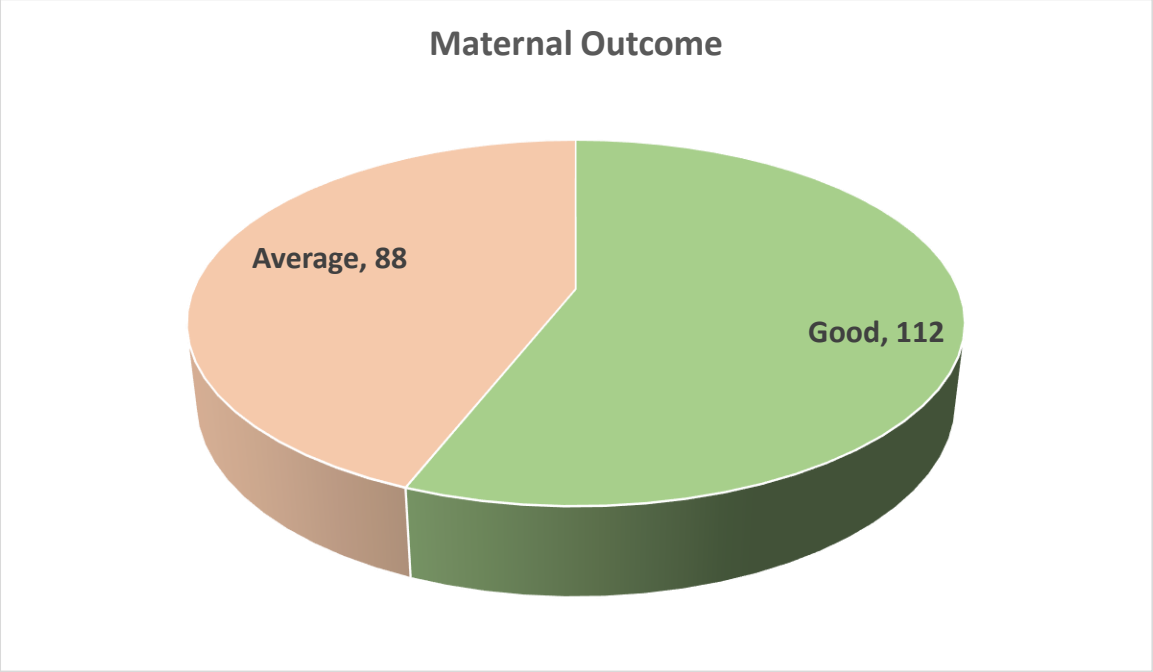
Admission to NICU		
	Frequency	Percentages
Yes	114	57%
No	86	43%



Regarding the frequencies and percentages of Requirement of NICU among the 200 anaemic pregnant women were, 114 required NICU admission and remaining 86 newborn doesn't required NICU admission.

Table 22: Maternal outcome

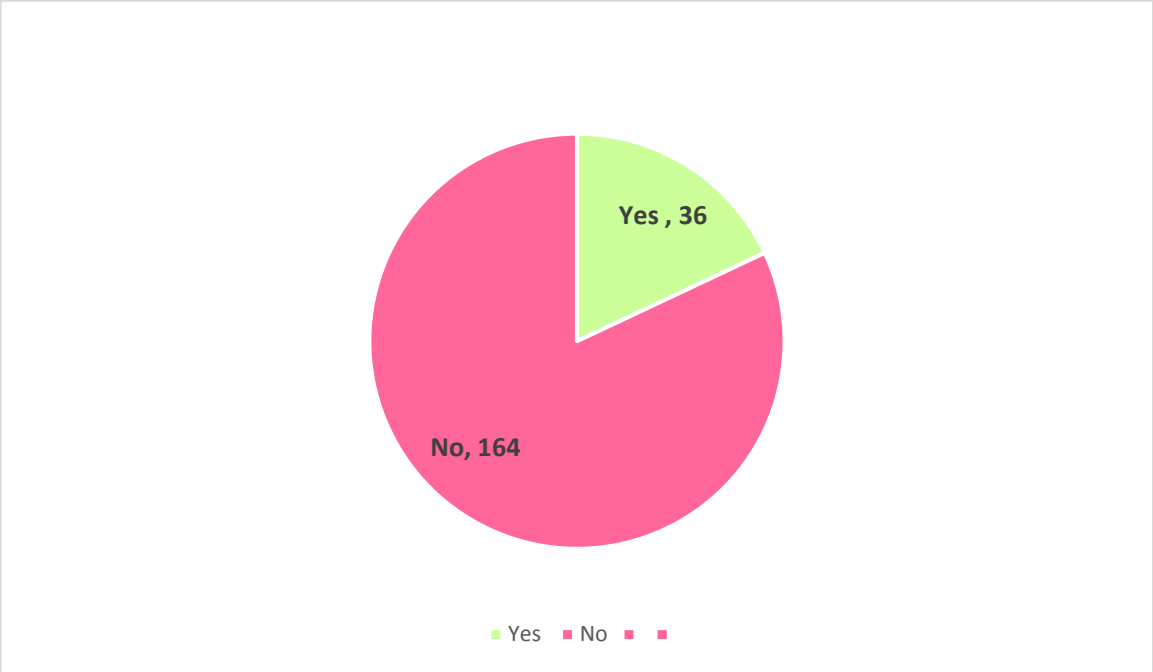
Maternal Outcome		
	Frequency	Percentages
Good	112	56 %
Average	88	44 %



Regarding the frequencies and percentages of maternal outcome among the 200 anaemic pregnant women were, 112 had good outcome and remaining 88 had average outcome.

Table: 23 Distribution of Abruption

Abruption		
	Frequency	Percentages
Yes	36	18%
No	164	82%



Regarding the frequencies and percentages of Abruption among the 200 anaemic pregnant women were, 36 yes and remaining 164 had No abruption.

Discussion:-

In our study Maximum number of the patients in the study population were in the age group of 21 to 25 years of age as 44%. The participant's age ranges from minimum 19 years of age to 32 years of age. The mean age of the pregnant mothers in our study was 25.73 $\pm$ 3.8 years of age. The average age of the patients, according to Jaiprakash et al, was 26.655 years.[115] Enaruna et al identified a link between age and preeclampsia risk at 30 years (range: 19 to 41), and Assis et al found a link between age and preeclampsia risk at 30 years (range: 19 to 41).[91,16]

In the current study, about 9% had mild anaemic, majority of the participants were moderately anaemic 81% and 10% were severely anaemic. The prevalence of anaemia was found to be 69.3 percent in a comparable study conducted in rural Tamilnadu. [121] Although the DLHS-3 study showed a prevalence of 97 percent, a subsequent district level household and facility survey found that 41 percent of pregnant women have mild anaemia, 53 percent have moderate anaemia, and 3 percent have severe anaemia. Haryana had a prevalence of only 59.6 percent in a survey conducted in 2012-13, with rural regions having a slightly higher prevalence of 60.5 percent. [120,122]

From our study to 8% had pre eclampsia and remaining 92% didn't develop pre eclampsia. In a research in Scotland, Jaiprakash et al discovered a comparable incidence rate of 5.8%. [115] In Indian studies, Mudaliar and Menon found a prevalence of preeclampsia of 7-9 percent.[117] Pre-eclampsia affects about 2–8% of all pregnancies around the world.[91]

In the current study, about 3 were illiterate, 7 went to primary school, 25 were studied up to middle school, 35 reported to complete high school, 20 of them have studied up to higher secondary education and 10 were graduated. According to their study, 42 percent of the 180 patients had no formal education, compared to 30 percent of the controls who were illiterate. In comparison to controls, women with a lower education level were more likely to develop preeclampsia and severe anaemia. Illiteracy was linked to a 2-fold increased chance of presenting hypertensive illness during pregnancy, according to Tebeu et al (OR: 1.7; 95 percent CI: 1.1-2.4). [118] Dutta et al investigated the impact of literacy status on anaemia and found it to be substantial.[123]

In our study 16% pregnant women belonged to Class 1, 37% belonged to Class 2 and majority of them belonged to class 3 with 47%. The Socio Economic status was classified based on the Modified B G Prasad's Classification. Majority of subjects belonged to class IV in anaemic (40%) as compared to 14% among non anaemic. Bedi R et al also observed the same in their study, 50.26% among socio-economic status IV and V women were significantly more anaemic as

compared to non anaemic group.[124] This association can probably be explained by numerous contributing factors like poor nutrition, logistic and financial constraints, hesitation in approaching healthcare facilities being some of them.

In our study 200 anaemic pregnant women, 19 %had IUGR and remaining 81% didn't have IUGR. Nirmala Devi et al found IUGR in 12.77% patients of severe anemia.[19] In this study, 56% had good outcome and remaining 44 % had average outcome. The majority of patients in the control group were normal, meaning they were discharged in good health.

In the present study for 18 % pregnant women had Pre term delivery and 82% had term baby. And 28% had low birth weight babies less than 2.5kgs and remaining 62% of the mothers had normal weighed babies. K. Jagdeesh Kumar discovered a 6.5 percent rise in the prevalence of low birth weight kids and an 11.5 percent increase in preterm deliveries in moms who were anaemic in their third trimester in a study.[125] The number of low birth weight babies was higher among anaemic moms in both studies. Low maternal haemoglobin levels were also linked to an increased risk of preterm delivery and LBW newborns in a study conducted by V.B. Sangeeta. [126] In this study 18% abruption and remaining 82 % had No abruption.

Conclusion:

Maximum number of the patients in the study population were in the age group of 21 to 25 years of age, which shows that younger population had the risk of developing preeclampsia. Out of the study population, more than half of the participants were primigravida.

Regarding the educational status, 35% of them attended high school which indicates educational status also aids in preventing anemia thereby preventing preeclampsia.

81% of the study population had moderate anaemia.

8% of the anemic pregnant women had pre eclampsia

28% of the patients with anemia were treated with iron sucrose transfusion

30% of the patients with gestational hypertension had Premonitory Symptoms such as headache and blurring of vision.

Regarding the onset of delivery, nearly 66% of them had labour induction and 67% had Normal Vaginal delivery

18% of them had preterm delivery and 57% of them required NICU admission.19% of them had IUGR.

Disorders in pregnancy like preeclampsia and anemia have negative impact on fetus like intrauterine growth restriction, premature delivery and low birth weight. The greater the severity of anemia in pregnancy, greater the risk of Preeclampsia, Preterm

delivery, low birth weight and still birth. Maternal wellbeing, optimal nutritional status and freedom from systemic disorder are essential in providing in utero environment for proper growth and development of fetus.

Perinatal and maternal morbidity and mortality are enhanced by anaemia and preeclampsia. The current study looked at the link between anaemia and severe preeclampsia, and found that the risk of neonatal and maternal morbidity and mortality increases with the severity of anaemia when preeclampsia is present. Thus, based on the findings of this study and past research, it can be inferred that detecting anaemia early in pregnancy can be a key to preventing preeclampsia.

Anaemia has a significant negative impact on the fetomaternal result, and modest measures performed to cure anaemia in women planning to get pregnant or who are already pregnant can have far-reaching consequences. To improve maternal and fetal outcome, primary health care has to be strengthened. Prevention, Early diagnosis and treatment of anemia in pregnancy is to be given priority.

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PROFORMA

NAME:

AGE:

IP.NO:

D.O.A:

S.E.S:

EDUCATION:

ADDRESS:

OCCUPATION:

COMPLAINTS:

Breathlessness on exertion

Fatigue

Chestpain/Palpitation

Headache/Bluring of vision/Epigastric pain/Vomiting/Reduced urine output

MENSTRUAL HISTORY:

MARITAL HISTORY

OBSTETRIC HISTORY:

Parity issues

Iron sucrose transfusion/Packed cell transfusion in antenatal period

Previous history of GHTN/Anemia

PERSONAL HISTORY:

PAST MEDICAL HISTORY:

PAST SURGICAL HISTORY:

FAMILY HISTORY:

GENERAL EXAMINATION:

Height: Pallor:

Weight: Icterus:

BMI: Edema:

VITALS:

Temperature: Pulse Rate:

Blood pressure: Respiratory rate:

SYSTEMIC EXAMINATION:

CVS-

RS-

CNS-

P/A:

P/V:

Urine Albumin:

ONSET OF DELIVERY:

MODE OF DELIVERY

BIRTH WEIGHT OF BABY:

APGAR:

NICU ADMISSION:

PATIENT CONSENT FORM

STUDY TITLE:

STUDY ON ASSOCIATION OF ANAEMIA AND PREECLAMPSIA IN PREGNANCY AND ITS FETOMATERNAL OUTCOME

DEPARTMENT OF OBSTETRICS & GYNAECOLOGY, GMKMCH SALEM.

PARTICIPANT NAME: AGE: SEX: I.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time: Patient name:

Date: Signature / Thumb Impression of Patient:

Place: Name and signature of the Investigator

ஆராய்ச்சி ஒப்புதல் படிவம்

பெயர்	:	தேதி	:
வயது	:	உள்நோயாளி எண்	:
பாலினம்	:	ஆய்வு சேர்க்கை எண்	:

இந்த ஆய்வின் நோக்கம் மற்றும் விவரங்கள் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது .இவ்வாய்வில் இருந்து நான் எந்த நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எனக்கு எந்த பாதிப்பும் இல்லை என்பதையும் தெளிவாக புரிந்து கொண்டேன்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

இந்த ஆய்வில் எவ்வித நிர்பந்தமும் இன்றி எனது சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகின்றேன்.

நான் சுயநினைவுடனும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் சேர்த்துக்கொள்ள சம்மதிக்கின்றேன்.

ஆராய்சியாளர் ஒப்பம்

பங்கேற்பாளர் ஒப்பம்
(அ)
இடது பெருவிரல் ரேகை

MASTER CHART

S.NO	IP no	Name	Age	Socioeconomic status	Parity	Gestational age	BMI	Education	Hemoglobin(%)	packed cell transfusion	Iron sucrose infusion	BP 1	BP 2	Premonitory symptoms	MgSO4	Urine albumin	Onset of delivery	Outcome of delivery	Preterm birth	Low birth wt	IUGR	APGAR	Admission	Maternal outcome	Eclampsia	Abruption
1	203695	Karthiga	31	class 2	primi	37 wks	25	10th	8.5	2	no	120/80	130/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
2	206935	Seetha	27	class 3	second	37 wks	22	5th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
3	215486	vanitha	27	class 3	primi	33 wks	25	10th	9.9	no	no	130/90	130/90	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
4	213698	Ishwarya	28	Class 2	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
5	213666	Priya	27	class 3	second	37 wks	22	12th	7.2	2	no	160/110	150/100	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
6	225698	Pooja	25	class 3	second	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
7	226987	Saraswati	31	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
8	225896	Sivaranjani	27	class 3	primi	33 wks	25	10th	9.9	no	no	120/80	130/90	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
9	227548	Ranjani	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
10	269852	Kokila	22	Class 1	second	38 wks	24	10th	5.7	3	no	130/90	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
11	254984	Monisha	30	class 3	third	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
12	306985	Geetha	27	class 3	second	37 wks	22	12th	7.2	2	no	120/80	150/100	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
13	302589	Priya	31	class 3	second	38 wks	24	10th	5.7	3	no	120/80	130/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
14	300265	Priyadharshini	18	Class 1	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
15	265892	Jeeva	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
16	258963	Fathima	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
17	245243	Geethalaksmi	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	130/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
18	200132	Anandhi	31	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	120/80	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
19	200145	Sivanya	21	class 2	second	37 wks	25	10th	8.5	2	no	160/110	120/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
20	305914	Rizwana Banu	30	Class 2	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
21	236985	Hema	31	class 3	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
22	306296	Sathya	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
23	233698	Latha	28	class 3	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
24	256985	Karunya	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
25	265954	Amin	31	class 3	second	38 wks	24	10th	5.7	3	no	160/110	120/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
26	210465	Jeeva	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
27	211659	Kalaivani	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
28	283178	Rani	22	Class 1	second	38 wks	24	10th	5.7	3	no	130/90	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
29	333047	Kalaiarasi	25	Class 1	primi	37 wks	22	5th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
30	301456	karthiga	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
31	366985	keerthi	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
32	344567	kalyani	22	Class 2	third	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
33	344258	Ponni	28	Class 2	second	39 wks	24	2th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
34	301475	yasmin	27	Class 2	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
35	369852	keerthana	21	class 3	second	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
36	206458	Mythili	27	Class 2	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
37	200469	Kokila	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	130/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
38	206985	Gayathri	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
39	214569	Pavithra	27	Class 2	primi	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
40	245698	Radha	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
41	247789	Seetha	27	class 3	second	37 wks	22	12th	7.2	2	no	120/80	150/100	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
42	245669	Latha	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/80	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
43	256698	Priya	22	class 3	second	38 wks	24	10th	5.7	3	no	130/90	130/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
44	246985	Panchali	30	Class 1		39 wks	30	5th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
45	259856	suganthi	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no

S.NO	IP no	Name	Age	Socioeconomic status	Parity	Gestational age	BMI	Education	Hemoglobin(%)	packed cell transfusion	Iron sucrose infusion	BP 1	BP 2	Premonitory symptoms	MgSO4	Urine albumin	Onset of delivery	Outcome of delivery	Preterm birth	Low birth wt	IUGR	APGAR	Admission	Maternal outcome	Eclampsia	Abruption
46	258963	Meena	32	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
47	254789	Meera	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
48	326598	Bakiyalakshmi	30	class 3	third	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
49	326587	Anitha	31	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
50	326599	Jothilakshmi	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
51	233652	Sanjana	31	class 2	primi	37 wks	25	10th	8.5	2	no	120/80	130/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
52	221456	Manju	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
53	211457	leela	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/80	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
54	258977	Gayathri	21	class 3	second	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
55	220569	Yasmin	23	class 3	second	38 wks	20	10th	7.5	2	no	140/90	130/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
56	221456	Pavithra	23	class 3	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
57	224896	Roja	26	Class 1	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
58	224789	Lalitha	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	120/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
59	214365	Saradha	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	130/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
60	214778	Maniyammal	31	Class 1	second	38 wks	24	10th	5.7	3	no	160/110	120/80	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
61	211369	Kiruba	23	class 3	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
62	244896	Jeevitha	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
63	244789	chandralekha	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
64	245698	Katheeja	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
65	247569	Gopika	27	class 3	second	37 wks	22	5th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
66	259863	Lotus	30	Class 1		39 wks	30	5th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
67	254896	Priyanka	18	Class 1	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
68	254899	Kasthuri	27	Class 1	third	37 wks	22	12th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
69	253698	Kavitha	30	Class 2	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
70	266666	Sumathi	23	Class 2	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
71	266589	malathi	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
72	266868	lakshmi	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
73	266896	Revathi	30	Class 1	third	39 wks	30	illiterate	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
74	266485	Sundari	23	class 3	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
75	277896	Jothikala	26	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
76	277963	Lalitha	26	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
77	306451	Akila	21	Class 1	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
78	304589	Bharathi	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	120/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
79	304522	Meenatchi	23	Class 2	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
80	304658	Jeeva	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	120/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
81	304552	kaviya	27	Class 1	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
82	306999	kumudha	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	120/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
83	298172	Shanthi	27	Class 1	primi	33 wks	25	10th	9.9	no	no	130/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
84	306988	Meena	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	130/90	130/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
85	307556	Tamil	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
86	307566	Selvi	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
87	308469	Jothi	27	Class 1	second	37 wks	22	5th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
88	308999	Priya	31	class 3	second	38 wks	24	10th	5.7	3	no	120/80	120/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
89	310036	Kokila	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
90	290986	Keerthiga	23	class 2	second	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no

S.NO	IP no	Name	Age	Socioeconomic status	Parity	Gestational age	BMI	Education	Hemoglobin(%)	packed cell transfusion	Iron sucrose infusion	BP 1	BP 2	Premonitory symptoms	MgSO4	Urine albumin	Onset of delivery	Outcome of delivery	Preterm birth	Low birth wt	IUGR	APGAR	Admission	Maternal outcome	Eclampsia	Abruption
91	301896	Konkana	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
92	280956	Mary	25	Class 2	primi	37 wks	22	4th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
93	310078	Jasmine	23	class 2	second	34 wks	24	8th	10.6	no	yes(3)	150/110	120/80	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
94	310036	Princy	23	class 3	primi	38 wks	20	illiterate	7.5	2	no	120/80	120/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
95	310458	Lalitha	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	120/80	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
96	301169	Kavitha	27	class 3	second	33 wks	25	10th	9.9	no	no	140/90	120/80	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
97	315896	Manju	22	class 3	second	38 wks	24	10th	5.7	3	no	130/90	130/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
98	314598	Hema	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
99	317589	Latha	22	class 3	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
100	266610	kavya	21	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
101	325899	Jancy	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
102	358996	Pavithra	25	class 3	second	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
103	358963	Kavilakshmi	25	class 3	primi	37 wks	22	12th	9	1	no	120/80	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
104	302589	Manjula	25	Class 2	second	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
105	314789	Gokulapriya	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
106	324789	Vanitha	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/80	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
107	325698	Jeevitha	22	Class 1	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
108	325888	Jothika	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
109	325669	Lakshmipathi	31	class 3	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
110	325703	veena	31	Class 1	second	38 wks	24	10th	5.7	3	no	160/110	120/80	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
111	325596	Meena	27	Class 1	second	37 wks	22	5th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
112	325888	lakhmi	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
113	325899	Nandhini	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
114	323045	shalini	23	class 2	second	34 wks	24	8th	10.6	no	yes(3)	150/110	120/80	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
115	332689	Sathya	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	130/90	130/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
116	345569	Snega	27	Class 1	third	37 wks	22	12th	7.2	2	no	120/80	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
117	344899	anushya	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
118	344529	Nirmala	27	Class 1	primi	33 wks	25	10th	9.9	no	no	130/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
119	344999	Thangamani	25	Class 2	primi	37 wks	22	4th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
120	345555	Meenatchi	23	class 3	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
121	301245	Arivoli	23	class 2	second	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
122	325896	Alagi	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
123	355896	Kalki	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
124	378956	ponnammal	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/80	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
125	305978	Harini	21	class 2	second	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
126	302669	Yasodha	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
127	308999	Janaki	21	Class 1	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
128	300006	Umrin	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
129	300059	Kavitha	23	Class 1	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
130	300529	Prema	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
131	300458	Mani	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
132	452624	Preethi	21	class 2	second	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
133	304521	Ravina	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
134	344465	Gayathri	31	class 2	second	37 wks	25	5th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
135	344215	Faridha	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no

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136	344756	Hema	21	class 2	primi	37 wks	25	10th	8.5	2	no	120/80	130/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	dead born	no	average	no	yes
137	12517	Amudha	27	class 3	second	37 wks	22	12th	7.2	2	no	130/90	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
138	316427	Obuli	21	class 2	second	37 wks	25	illiterate	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	no
139	299664	Priyadharshini	18	Class 1	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
140	6427	Kalaivani	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	120/80	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
141	456233	Keerthi	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
142	345895	Tamilarasi	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
143	324563	Imran	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
144	336985	Hemalatha	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
145	345669	Immaculate mary	28	class 3	primi	39 wks	24	12th	10	no	yes(3)	140/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
146	290986	Mohanapriya	23	class 3	primi	38 wks	20	illiterate	7.5	2	no	120/80	120/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
147	236455	Kanaga	27	class 3	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
148	256498	Preethi	21	class 2	second	37 wks	25	illiterate	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
149	254263	Jeeva	30	Class 1	third	39 wks	30	illiterate	8.9	1	no	120/80	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
150	254489	Barathi	25	Class 1	primi	37 wks	22	5th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
151	256698	varalakshmi	31	class 3	second	38 wks	24	10th	5.7	3	no	160/110	120/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
152	236548	Kopika	27	class 3	second	37 wks	22	12th	7.2	2	no	160/110	150/100	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
153	332654	Yesther	30	Class 2	Third	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
154	369852	Rani	21	class 2	primi	37 wks	25	10th	8.5	2	no	120/80	130/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	dead born	no	average	no	yes
155	336952	Eshwari	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
156	312549	Anitha	22	Class 1	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
157	324588	Lalitha	27	Class 1	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
158	32162	Hemalatha	31	class 3	second	38 wks	24	10th	5.7	3	no	120/80	130/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
159	264846	Rani	28	class 3	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
160	369832	Tamilarasi	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
161	364985	Reeta	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	130/90	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
162	324596	Pavithra	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	130/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
163	245795	Punitha	21	class 3	primi	39 wks	24	12th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
164	245565	Jothi	22	Class 2	third	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
165	227689	Hema	28	class 3	primi	39 wks	24	12th	10	no	yes(3)	140/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
166	342465	Priya	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	150/110	160/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
167	268762	nithya	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	130/90	130/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
168	246589	kavitha	27	Class 1	primi	33 wks	25	10th	9.9	no	no	140/90	120/80	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
169	275952	pooja	30	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
170	643259	Poovizhi	18	Class 1	primi	38 wks	23	8th	8.6	1	no	140/90	160/110	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
171	266543	Komala	23	class 3	second	38 wks	20	10th	7.5	2	no	140/90	130/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
172	288756	Mythili	27	class 3	second	37 wks	22	12th	7.2	2	no	130/90	130/90	No	yes	2+	spont	LSCS	no	no	no	7/10,8/10	no	good	no	no
173	234595	Rasika	23	class 3	primi	38 wks	20	10th	7.5	2	no	120/80	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
174	255556	Chitra	27	class 3	second	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
175	336632	Banumathi	28	Class 2	second	39 wks	24	2th	10	no	yes(3)	120/80	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
176	394656	Bala	27	class 3	primi	33 wks	25	10th	9.9	no	no	130/90	130/90	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
177	225486	Vijaya	32	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
178	334265	Manimala	22	class 3	second	38 wks	24	10th	5.7	3	no	160/110	150/100	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
179	256985	Poojitha	23	Class 1	primi	38 wks	20	10th	7.5	2	no	140/90	140/90	no	no	nil	Induced	NVD	no	1.96kg	yes	7/10,8/10	Yes	good	No	No
180	243259	Banupriya	27	Class 2	primi	33 wks	25	10th	9.9	no	no	140/90	150/100	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no

S.NO	IP no	Name	Age	Socioeconomic status	Parity	Gestational age	BMI	Education	Hemoglobin(%)	packed cell transfusion	Iron sucrose infusion	BP 1	BP 2	Premonitory symptoms	MgSO4	Urine albumin	Onset of delivery	Outcome of delivery	Preterm birth	Low birth wt	IUGR	APGAR	Admission	Maternal outcome	Eclampsia	Abruption
181	211546	fathima	31	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	120/80	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
182	215589	Anushya	21	class 2	second	37 wks	25	10th	8.5	2	no	160/110	120/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
183	254559	Vennila	18	class 3	primi	38 wks	23	8th	8.6	1	no	140/90	120/90	No	yes	nil	Induced	LSCS	No	2.4kg	no	7/10,8/10	no	Average	no	yes
184	245596	Kanaga	27	Class 1	primi	33 wks	25	10th	9.9	no	no	140/90	120/80	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
185	216459	Hemalatha	30	class 3	Fourth	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
186	245671	Priyanka	25	Class 2	second	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
187	267868	Vanaja	31	class 2	second	37 wks	25	5th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
188	267816	Rani	31	class 3	second	38 wks	24	10th	5.7	3	no	120/80	120/90	yes	yes	2+	Induced	NVD	no	1.6kg	yes	7/10,8/10	Yes	Average	no	no
189	268578	aneesh	32	class 2	primi	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
190	233345	Abirami	25	class 3	primi	37 wks	22	12th	9	1	no	120/80	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
191	233678	nirmala	27	class 3	second	33 wks	25	10th	9.9	no	no	140/90	120/80	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no
192	245646	Kumudha	25	class 3	primi	37 wks	22	12th	9	1	no	130/90	130/90	no	no	nil	induced	NVD	no	1.75kg	yes	7/10/8/10	yes	good	no	no
193	286869	Priya	30	Class 2	Third	39 wks	30	8th	8.9	1	no	130/90	130/90	no	no	nil	Induced	NVD	no	no	no	7/10,8/10	no	good	no	no
194	304747	Vanitha	28	Class 2	primi	39 wks	24	12th	10	no	yes(3)	130/90	130/90	No	no	nil	spont	NVD	no	2.35kg	no	7/10,8/10	yes	good	no	no
195	316858	Kavya	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
196	335696	Priyanka	26	class 2	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	130/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
197	336467	Jamuna	21	class 2	primi	37 wks	25	10th	8.5	2	no	160/110	140/90	yes	Yes	1+	Spont	LSCS	yes	2.0kg	no	7/10/8/10	no	average	no	yes
198	308647	Yazhini	23	class 2	primi	34 wks	24	8th	10.6	no	yes(3)	130/90	130/90	No	yes	1+	Induced	NVD	no	1.3kg	yes	6/10,7/10	yes	Average	no	no
199	342573	Jeevitha	26	Class 1	second	39 wks	23	graduate	9.6	no	yes(3)	120/80	140/90	no	no	nil	spont	NVD	no	2.3kg	no	7/10,8/10	no	good	no	no
200	300098	Lalitha	27	class 3	primi	33 wks	25	10th	9.9	no	no	120/80	130/90	yes	yes	3+	Induced	LSCS	yes	yes(1.5kg)	no	7/10,8/10	yes	Average	no	no