A PROSPECTIVE STUDY ON EFFECT OF PREPREGNANCY BMI AND GESTATIONAL WEIGHT GAIN ON FETOMATERNAL OUTCOME

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In partial fulfillment of the regulations for the award of the degree of

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THANJAVUR MEDICAL COLLEGE

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CHENNAI, TAMILNADU

May 2021

CERTIFICATE FROM INSTITUTION

This is to certify that the dissertation titled **"A PROSPECTIVE STUDY ON EFFECT OF PREPREGNANCY BMI AND GESTATIONAL WEIGHT GAIN ON FETOMATERNAL OUTCOME"** is a bonafide work done by **Dr.C.SUGANYA,** Post graduate student, Department of obstetrics and Gynaecology, Thanjavur Medical college,Thanjavur – 04, during the period **JANUARY 2019 TO DECEMBER 2019** in partial fulfillment of rules and regulations of the Tamilnadu Dr. M.G.R Medical University,for the award of MD Degree Branch II (Obstetrics and Gynaecology) examination to be held in **May 2021 .**

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This is to certify that The Research Proposal / Project titled

EFFECT OF PREGESTATION BMI AND GESTATIONAL WEIGHT GAIN ON FETOMATERNAL OUTCOMES

submitted by Dr. C. SUGANYA **of**

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was approved by the Ethical Committee.

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DECLARATION

I solemnly declare that this dissertation titled **"A PROSPECTIVE STUDY ON EFFECT OF PREPREGNANCY BMI AND GESTATIONAL WEIGHT GAIN ON FETOMATERNAL OUTCOME"** was done by me at Department of Obstetrics and Gynaecology, Thanjavur Medical College,Thanjavur, during year 2018-2021 under the guidance and supervision of **Prof.Dr.R.RAJARAJESWARI, MD.,DGO.,DNB.** This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University,Chennai** in partial fulfillment of the University regulations for the award of **M.D. BRANCH II (Obstetrics and Gynaecology).**

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CONTENTS

INTRODUCTION

Pregnancy BMI and weight gain during pregnancy are important predictors of adverse pregnancy outcomes. The problems during pregnancy were more related to low BMI previously, but with changing lifestyle, obesity is increasing rapidly especially in urban setups and may become a major health problem in the future. "A pregnancy is defined as high risk, when the probability of an adverse outcome for the mother or child is increased over the base line risk of that outcome among the general population by the presence of one or more ascertainable risk factors"[1].

"One such pre-existing maternal morbidity that makes a pregnancy high risk is obesity". The magnitude of the obesity prevalence has been increasing in developed and developing nations, though in varying degrees. Studies have found that Gestational diabetes, Gestational Hypertension, emergency caesarean section, postpartum hemorrhage, wound infections, preterm delivery, large for gestational age(LGA), and fetal death in utero were more common in overweight and obese mothers[2.] On the other hand underweight women were at a higher risk of developing Anemia, along with adverse neonatal outcomes like Fetal Growth retardation(FGR) and prematurity.

Maternal nutritional status plays a vital role for the health and quality of life of a pregnant mother and her baby. Utmost importance needs to be given to BMI and the patterns of weight gain during pregnancy, as they are modifiable risk factors

of adverse pregnancy outcomes.[3] One should have basic knowledge and awareness regarding the symptoms and signs of adverse pregnancy outcomes. A better understanding of the complex interrelations between the mother and fetus has led to a vast improvement on antenatal recommendations. The guidelines established by the Institute of Medicine (IOM) regarding weight gain during pregnancy based on pre pregnancy BMI has aimed at obtaining good pregnancy outcomes.

AIMS AND OBJECTIVES

1. To study the association between pre pregnancy BMI and gestational weight gain on maternal complications.

2. To determine the association between pre pregnancy BMI and gestational weight gain on labour outcome.

3. To analyze the influence of pre pregnancy BMI and gestational weight gain on neonatal outcome.

4. To evaluate the risk of developing adverse maternal and fetal outcomes in women with extremes of BMI and extremes of gestational weight gain.

Study centre : Department of Obstetrics and Gynecology,
Government Raja Mirasudhar Hospital (RMH),
Thanjavur Medical College,
Thanjavur.

Duration of study : January 2019 to December 2019

Time Period : 12 months

Study design : Prospective Cohort study

REVIEW OF LITERATURE

Early pregnancy BMI and gestational weight gain are crucial predictors of adverse pregnancy outcomes. The Institute of Medicine (IOM) has brought out recommendations for weight gain during pregnancy since 1990[4].The association between BMI and adverse maternal and fetal outcomes has well been established with a huge body of literature to support it.

Increased weight gain is associated with increased incidence of GDM, GHT, cesarean section, post partum haemorrhage, macrosomia and shoulder dystocia during deliveries[5, 6, 7]. Low weight gain has been known to cause maternal anemia, small for gestational age babies and prematurity[8, 9, 10]. Studies have shown that women with high BMI may benefit from low weight gain during pregnancy[11].

The prevalence of obesity is on the rise in a rapid way probably due to the changing lifestyles especially in our urban setup, with pregnancy contributing further to it. A recent study done in UK between 2002 and 2004 showed that 1 in 5 antenatal women were obese [12].

Body composition and Energy stores: [13]

These include the products of conception (fetus, placenta, and amniotic fluid), uterine and breast tissue, extracellular fluid, and maternal fat.

A 70 kg individual has energy stores comprising of 15 kg as fat, 0.4 kg as glycogen and 6 kg as protein. 15-25 % of the total energy is stored as fat.

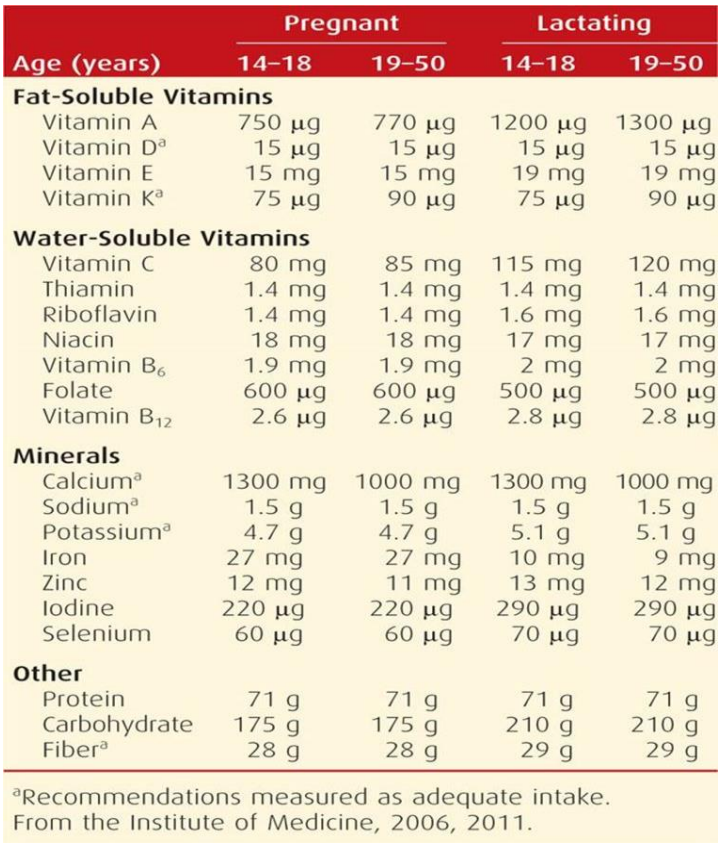
The four major components of a nutrition assessment are medical, nutrition, psychosocial, and obstetric histories. A previous low-birth-weight infant or a large-for-gestational age baby can indicate a nutrition problem requiring nutrition intervention. A psychosocial assessment should include information on the patient's feelings about the pregnancy, her support system, emotional status, and body image. The components that need to be taken into consideration regarding an individual's nutritional status assessment are dietary history, clinical examination and anthropometry.[14]

Dietary History

The dietary history should begin with a measured height and weight, pre gravid weight, BMI determination and weight gain during pregnancy. The diet history can be done by a 24-hour recall, where a person relates what they ate during the previous 24 hours

Recommended daily dietary allowances for pregnant and lactating women as per Williams obstretics edition 25[17].

Figure 1:



	Pregnant		Lactating	
Age (years)	14–18	19–50	14–18	19–50
Fat-Soluble Vitamins				
Vitamin A	750 µg	770 µg	1200 µg	1300 µg
Vitamin D[a]	15 µg	15 µg	15 µg	15 µg
Vitamin E	15 mg	15 mg	19 mg	19 mg
Vitamin K[a]	75 µg	90 µg	75 µg	90 µg
Water-Soluble Vitamins				
Vitamin C	80 mg	85 mg	115 mg	120 mg
Thiamin	1.4 mg	1.4 mg	1.4 mg	1.4 mg
Riboflavin	1.4 mg	1.4 mg	1.6 mg	1.6 mg
Niacin	18 mg	18 mg	17 mg	17 mg
Vitamin B_6	1.9 mg	1.9 mg	2 mg	2 mg
Folate	600 µg	600 µg	500 µg	500 µg
Vitamin B_{12}	2.6 µg	2.6 µg	2.8 µg	2.8 µg
Minerals				
Calcium[a]	1300 mg	1000 mg	1300 mg	1000 mg
Sodium[a]	1.5 g	1.5 g	1.5 g	1.5 g
Potassium[a]	4.7 g	4.7 g	5.1 g	5.1 g
Iron	27 mg	27 mg	10 mg	9 mg
Zinc	12 mg	11 mg	13 mg	12 mg
Iodine	220 µg	220 µg	290 µg	290 µg
Selenium	60 µg	60 µg	70 µg	70 µg
Other				
Protein	71 g	71 g	71 g	71 g
Carbohydrate	175 g	175 g	210 g	210 g
Fiber[a]	28 g	28 g	29 g	29 g

[a]Recommendations measured as adequate intake.
From the Institute of Medicine, 2006, 2011.

Table 1: Recommended dietary allowances as for non pregnant, pregnant and lactating mother[15].

Nutrient	Non-Pregnant	Pregnant	Lactation
Vitamin A	700	770	1300

Nutrient	Non-Pregnant	Pregnant	Lactation
(µg/d)			
Vitamin D (µg/d)	5	15	15
Vitamin E (mg/d)	15	15	19
Vitamin K (µg/d)	90	90	90
Folate (µg/d)	400	600	500
Niacin (mg/d)	14	18	17
Riboflavin (mg/d)	1.1	1.4	1.6
Thiamin (mg/d)	1.1	1.4	1.4
Vitamin B_6 (mg/d)	1.3	1.9	2
Vitamin B_{12} (µg/d)	2.4	2.6	2.8
Vitamin C (mg/d)	75	85	120
Calcium (mg/d)	1,000	1,000	1,000
Iron (mg/d)	18	27	9
Phosphorus (mg/d)	700	700	700
Selenium (µg/d)	55	60	70
Zinc (mg/d)	8	11	12

*Applies to women >18 years old

Clinical Examination

Measurements such as weight and height are recorded and Body Mass Index (BMI) is calculated by using QUETELET INDEX. The Quetelet Index is used far more commonly than body fat percentage to define obesity.

BMI = Weight (kg)/ Height (m^2)

Table 2: Classification of BMI by WHO (Weight / Height ^{2})

Underweight	< 18.5
Normal	18.5 – 24.9
Overweight	25.0 – 29.9
Obese	30.0 – 39.9
Morbidly obese	>40

For example, if a women weighs 50kgs with height of 150cms, her BMI is calculated as $50kg/(1.5m)^{2=}$ $22.2kg/m^2$. So she belongs to normal BMI category.

Anthropometry

The measurements of the size and proportion of the human body is an important predictor of nutritional status of the individual. This is done by calculating the proportion of fat and muscle using the formula as given under,

Mid upper arm muscle circumference (MUAC) = arm circumference– triceps skin fold

MUAC identifies underweight/thinness by measuring the circumference of the mid-upper arm and comparing it to a pre-determined cutoff. In pregnant women, low MUAC has been associated with Fetal Growth Restriction, low birth weight, and neonatal morbidity (WHO 1995a). Because MUAC is a relatively simple measurement that requires minimal equipment, is not affected by pregnancy status and can be measured at any time during pregnancy.

Table 3 : Anthropometric Mesurements[1]

	MEN	**WOMEN**	**INTEPRETATION**
Anthropometric measurements MUAC	25.5	23.0	Adequate
	20.0	18.5	Borderline
	15.0	14.0	Depletion
	10.0	9.0	Severe depletion

Energy Requirements and intake of calories[16]

The largest component of energy expenditure is attributed to the lean body mass and is known as the Basal Metabolic Rate(BMR).Caloric intake should increase by approximately 300 kcal/day during pregnancy. This value is derived from an estimate of 80,000 kcal needed to support a full-term pregnancy and accounts not only for increased maternal and fetal metabolism but for fetal and placental growth. Dividing the gross energy cost by the mean pregnancy duration (250 days after the first month) yields the 300 kcal/day estimate for the entire

pregnancy. However, energy requirements are generally the same as non-pregnant women in the first trimester and then increase in the second trimester, estimated at 340 kcal and 452 kcal per day in the second and third trimesters respectively.

Table 4: Daily energy requirements according to Physical Activity [13] as per Harrison's Principles of Internal Medicine

Circumstances	Healthy adult females	Healthy adult males
At rest	1600 kcal	2000 kcal
Light work	2000 kcal	2700 kcal
Heavy work	2250 kcal	3500 kcal

Metabolic changes in pregnancy[17]

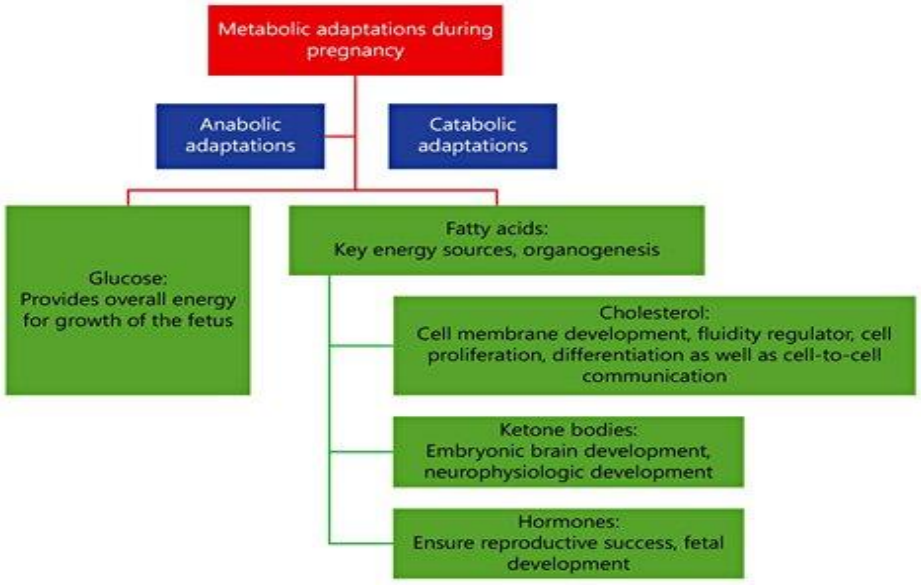
Maternal metabolism changes substantially during pregnancy. Early gestation can be viewed as an anabolic state in the mother with an increase in maternal fat stores and small increases in insulin sensitivity. Hence, nutrients are stored in early pregnancy to meet the feto-placental and maternal demands of late gestation and lactation. In contrast, late pregnancy is better characterized as a catabolic state with decreased insulin sensitivity (increased insulin resistance). An increase in insulin resistance results in increases in maternal glucose and free

fatty acid concentrations, allowing for greater substrate availability for fetal growth.

Fat metabolism[18]

Human pregnancy is characterized by alterations in maternal lipid metabolism, which could be divided into 2 phases: an anabolic phase and a catabolic phase

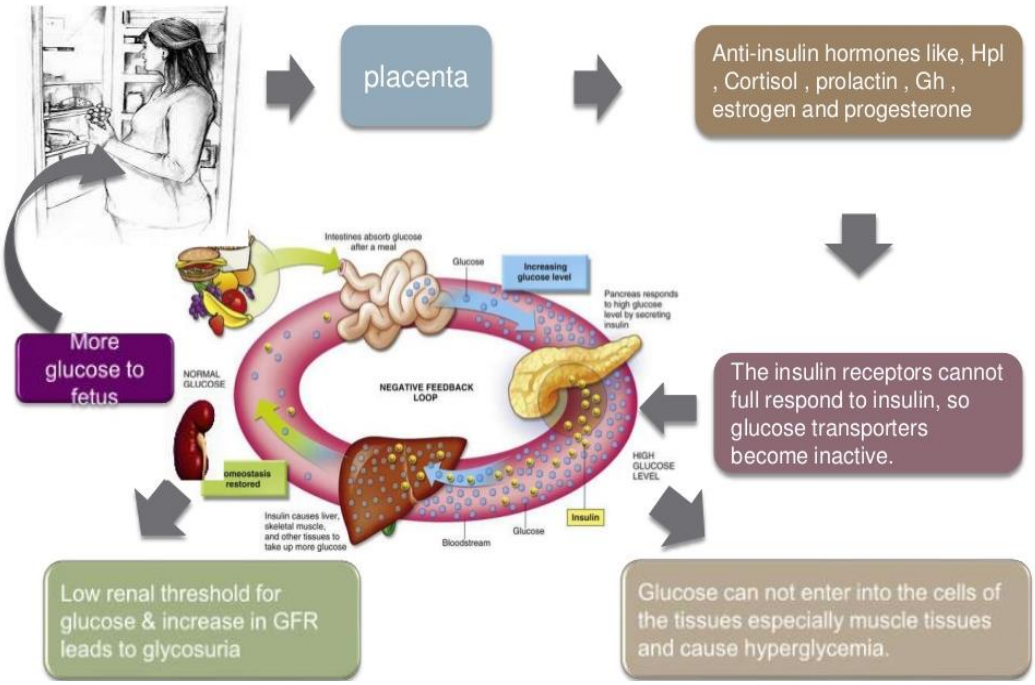
Figure 2: Metabolism of fat



There is an increase in total serum cholesterol and triglyceride levels in pregnancy. The increase in triglyceride levels is mainly as a result of increased synthesis by the liver and increased lipolytic and decreased lipoprotein lipase activity, resulting in decreased catabolism of adipose tissue. Low-density lipoprotein (LDL) cholesterol levels also increase and reach 50% at term. High-density lipoprotein levels increase in the first half of pregnancy and fall in the

third trimester but concentrations are 15% higher than non-pregnant levels. Action of progesterone and estradiol on the hepatic system also play a pivotal role.Changes in lipid metabolism accommodate the needs of the developing fetus. Increased triglyceride levels provide for the mother's energy needs while glucose is spared for the fetus. The increase in LDL cholesterol is important for placental steroidogenesis.

Figure 3: Carbohydrate metabolism [17]



Pregnancy is a diabetogenic state and adaptations in glucose metabolism allow shunting of glucose to the fetus to promote development, while maintaining adequate maternal nutrition. Insulin-secreting pancreatic beta-cells undergo

hyperplasia, resulting in increased insulin secretion and increased insulin sensitivity in early pregnancy, followed by progressive insulin resistance. This is the result of increasing secretion of diabetogenic hormones such as human placental lactogen, growth hormone, progesterone, cortisol and prolactin. These hormones cause a decrease in insulin sensitivity in the peripheral tissues such as adipocytes and skeletal muscle by interfering with insulin receptor signaling. The effect of the placental hormones on insulin sensitivity is made evident postpartum when there is a sudden decrease in insulin resistance. Insulin levels are increased in both the fasting and postprandial states in pregnancy.

Insulin resistance and relative hypoglycaemia results in lipolysis, allowing the pregnant mother to preferentially use fat for fuel called as accelerated starvation resulting in ketonemia, preserving the available glucose and amino acids for the fetus and minimising protein catabolism.. If a woman's endocrine pancreatic function is impaired, and she is unable to overcome the insulin resistance associated with pregnancy then gestational diabetes develops**.**

Water metabolism:

Two aspects of water metabolism are considered. The first centres on the fact that in early pregnancy plasma osmolality falls, a fact which is largely due to the effect of overbreathing - the mother avoids a state of diabetes insipidus by 'resetting' her osmo-receptors. The second is the phenomenon of water storage in pregnancy - an average of 2 1/2 litres in excess of that usually stored in known

sites of tissue growth which, in some individuals, can rise to as much as 8 litres. The minimum amount of extra water retention at term is approximately 6.5 L.[17] Clearly demonstrable pitting edema of the ankles and legs is seen in most pregnant women, especially at the end of the day. This fluid accumulation ,which may amount to a litre or so, results from greater venous pressure below the level of the uterus as a consequence of partial vena cava occlusion.[15] Physiological edema is beneficial to the mother, as it compensates for the blood loss occurring at the third stage of labour.

Protein metabolism[17]

During pregnancy, nitrogen, derived from the metabolism of ingested protein, is needed for growth of the fetus, the placenta, the uterus, and the mother's breasts and other tissues. A considerable amount of nitrogen also is required for the increase in the mother's red cell volume and blood plasma. The fetus's demand for nitrogen is slight at first, but during the last month of pregnancy it acquires almost half of its total protein. In the process of accumulating this store and of building a reserve for the period after delivery, the woman who is on an adequate diet retains between two and three grams of nitrogen daily during her pregnancy; by term she and the fetus will have acquired approximately 500 grams (about 1.1 pounds) of nitrogen. Thus during pregnancy about 1000 g of extra proteins is deposited with half going to fetus and placenta and the other 500 g is added to the uterus as contractile protein, to the breast glandular tissue, plasma proteins and haemoglobin.[17]

Weight gain during pregnancy

The Institute of Medicine (1990) has defined weight gain in 3 ways [4]

1. Total weight gain is defined as weight just prior to delivery minus weight prior to conception.

2. Net weight gain is the total weight gain minus the infant's birth weight.

3. Rate per week is the weight gained over a specific period divided by the duration of that period in weeks.

In our country due to lack of awareness, especially in rural areas, it's very difficult to get the pre- pregnancy body weight. Thus keeping this in view, Gestational weight gain is the total weight gain during pregnancy, taking early pregnancy weight (< 12 wk GA) into account.

During the first trimester of pregnancy, the rate of weight gain is slowest, constant during second and beginning of third trimester and slows down during the end of third trimester.

Due to changes in maternal characteristics including higher prevalence of overweight pre conception BMI and increased reports of GWG being associated with adverse maternal and infant outcomes, the IOM reconvened a committee to reexamine the impact of weight gain in pregnancy and as a result published revised guidelines in 2009.

Table 5. Distribution of maternal weight in grams

Cumulative increase in weight (g) Modified from Hytten[19]

Tissues & fluids	10weeks	20weeks	30weeks	40weeks
Fetus	5	300	1500	3400
Placenta	20	170	430	650
Amniotic fluid	30	350	750	800
Uterus	140	320	750	800
Breasts	45	180	360	405
Blood	100	600	1300	1450
Extra vascular fluid	0	30	80	1480
Maternal store(fat)	310	2050	3480	3345
Total	650	4000	8500	12500

a, the total weight gain throughout pregnancy in healthy primigravidas is approximately 12.5 kg [46]. The rate of weight gain varies according to the stage of pregnancy. In the first 3months, there may be no substantial gain, especially if there has been vomiting and anorexia. The rate between 20weeks and delivery is about 0.5kg/week or 2kg/month. Pregnant women gaining more than 0.5 kg/week is considered abnormal and the risk of developing preeclampsia and GDM is more in such individuals.

Factors affecting Gestational Weight Gain

Diet

Abnormal gestational weight gain (GWG) is currently a serious obstetric problem. Abnormal GWG can have significant importance for both short-term pregnancy outcomes and for the long-term health of the offspring and the

mother. Health risks related to inadequate weight gain during pregnancy involve, first and foremost, a greater risk of premature birth and a low birth weight baby and/or fetal growth restriction and consequently, an increased risk of mortality and morbidity. Excessive weight gain is indicated as a risk factor for giving birth to a LGA (large for gestational age) baby compared with its gestational age giving birth to a baby with macrosomia, gestational diabetes, gestational hypertension , caesarean delivery, longer infant hospital stays and the persistence of a higher postpartum weight for the mother after childbirth, which predisposes one to obesity later in life .

Maternal pre-pregnancy body mass index (BMI), diet, physical activity, smoking status and socio-demographic factors are listed as the main determinants of GWG.

Physical activity

Studies during the years have shown that the amount of physical activity during pregnancy is inversely proportional to the amount of GWG. [20]

OTHER FACTORS

Factors such as pre pregnancy BMI, parity, maternal age, ethnicity,smoking, education status, caffeine and stress are associated with weight gain during pregnancy.

Pre pregnancy BMI

The most important factor seems to be that of pre pregnancy BMI which is associated with excessive weight gain in pregnancy. It was seen that obese and overweight women gained excess weight than the recommended GWG by IOM.[20,21] The average gestational weight gain among obese and overweight women is lower than women with normal BMI because the weight gain recommendations for obese women are lower and vice versa. It is said that hormones such as leptin, insulin and ghrelin which are in higher proportion in obese women could be the reason for higher gestational weight gain [22 , 23.]

Parity

One other factor linked to gestational weight gain is parity . Primipara gained more weight during pregnancy than multi para [24 , 25]. Although the risk of SGA was consistently higher in primipara than in multipara with similar gain, the steeply increasing risk of postpartum weight retention with increasing gain was independent of parity. At the same time, a considerable excess risk of LGA was only present in obese primiparae and multiparous women.

Education status

When it came to education status, studies shown that educated women had a weight gain within the recommended range in comparison with uneducated

women, probably due to awareness and knowledge regarding the adverse pregnancy outcomes associated with extremes of BMI[21].

Psychological factors

There are either not enough studies or the studies done have shown conflicting results regarding the association between weight gain and psychological factors such as stress, depression and social support [20, 26, 27].

Weight gain in pregnant adolescents

Pregnant adolescents face problems of anemia and low birth weight babies because all the energy consumed by them goes partly for their own growth besides the growth of the fetus and placenta. Compared to adult standards, adolescents may be considered underweight with limited fat stores, when actually they might be underweight or malnourished for their age and maturity[28]. Excess body fat during adolescence contributes to a greater health risk in adolescence and adulthood, particularly in childbearing years when excess weight can lead to poor pregnancy outcomes.

Weight gain in Multiple pregnancy

The literature suggests that in twin pregnancies weight gain is higher than the weight accounted for by the mass of the additional conceptus. A maternal weight

gain of 40-45pounds is associated with outcome of twins weighing around 2.5kg. A Study done by Lantz showed that weight gain should be according to the pre pregnant BMI. In women with low BMI a weight gain of 1.75 pounds/week after 20 weeks gestation is recommended, whereas in women with normal BMI, it was 1.5pounds / week during the second half of pregnancy[29.]

Recommendation for weight gain in pregnancy

Institute of Medicine (IOM) has recommended a weight gain of 1.0 to 3.5 kg during the first trimester. For underweight women, an average weight gain of 0.5 kg per week is recommended during the second and third trimester, 0.4 kg for normal weight women and 0.3 kg for overweight women. The recommended weight gain for a teenage mother would be the upper end of the recommended weight gain as she needs energy for her own growth along with the fetus and placenta. Around 16- 20 kg weight gain is recommended for twin pregnancies.

Table 6: Institute of Medicine 2009 Gestational Weight Gain Guidelines by pre pregnant BMI[30, 31]

	Total Weight Gain		**Incremental weight gain during the 2nd and 3rd Trimester**	
Preconception BMI	**Range in kg**	**Range in lbs**	**Mean (range) in kg/wk**	**Mean (range) in lbs/wk**

	Total Weight Gain		Incremental weight gain during the 2nd and 3rd Trimester	
Preconception BMI	**Range in kg**	**Range in lbs**	**Mean (range) in kg/wk**	**Mean (range) in lbs/wk**
Underweight (<18.5 kg/m^2)	12.5 – 18	28 – 40	0.51 (0.44 – 0.58)	1 (1 – 1.3)
Normal weight (18.5 – 24.9 kg/m^2)	11.5 – 16	25 – 35	0.42 (0.35 – 0.50)	1 (0.8 – 1)
Overweight (25.0 – 29.9 kg/m^2)	7 – 11.5	15 – 25	0.28 (0.23 – 0.33)	0.6 (0.5 – 0.7)
Obese (≥ 30.0 kg/m^2)	5 – 9	11 – 20	0.22 (0.17 – 0.27)	0.5 (0.4 – 0.6)

Recreated from IOM 2009 GWG Report

OBESITY AND ITS EFFECT ON PREGNANCY OUTCOME

Obesity is a significant health issue for women during pregnancy and the puerperium. It is well recognised that maternal obesity is associated with an increased risk of antenatal, peripartum and neonatal complications. In India, previously the problems during pregnancy were more related to low BMI but with changing lifestyle, the incidence and prevalence of obesity is increasing rapidly especially in urban set ups and may become a major health problem in the future , with pregnancy contributing further to it.

Definition

WHO defined overweight as a BMI of more than 25 kg/m2and obesity as a BMI of more than 30 kg/m2(waist : hip ratio >0.85). In 2002, Freedman and colleagues classified obesity further into Class 1 (BMI – 30 to 34.9 kg/m2), Class 2 (BMI – 35 to 39.9kg/m2) and Class 3 (BMI - >40 kg/m2)[17].

According to Freedman and Colleagues[32] 2002 obesity is further classified as:

CATEGORY BMI

Class I (Moderate obesity) 30-34.9 (kg/m2)

Class II (Severe obesity) 35-39.9 (kg/m2)

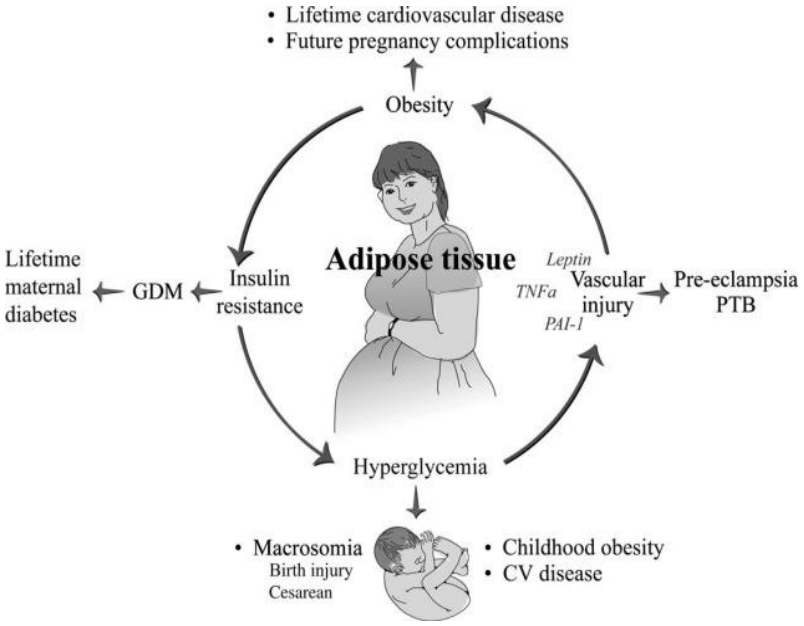
Class III (Very severe obesity) >40 (kg/m2)

PATHOPHYSIOLOGY OF OBESITY IN PREGNANCY

Obesity and excessive weight gain are associated with a greater amount of adipose (fatty) tissue that has been deposited in the body, including within the placenta. Leptin, also known as the “obesity hormone”, is a protein released by adipose tissue that regulates fat metabolism and storage in addition to controlling the mechanisms of appetite. The level of circulating leptin is directly proportional to the amount of fatty tissue that is stored in the body. Overweight and obese individuals experience higher levels of circulating leptin, leading to the desensitization and resistance from body cells to the effects of leptin. This allows for increased build-up of adipose tissue and uncontrolled increases in

appetite, further perpetuating the cycle of obesity. “Obesity represents a state of altered hormonal and inflammatory activity, associated with the function of adipose or fatty tissue. Adipose tissue has been demonstrated to be a source of production of peptides and non-peptide compounds involved in cardiovascular homeostasis. The peptide, interleukin 6 (IL6) is secreted by adipose tissue and modulates the production of C-reactive protein (CRP). Elevated CRP is a known marker of a chronic inflammation associated with an increased risk of cardiovascular disease.[33]

Figure 4: PATHOPHYSIOLOGY OF OBESITY IN PREGNANCY



EPIDEMIOLOGICAL FACTORS

The etiology of obesity is complex and is one of the multiple causations.

Age: Obesity can occur at any age but generally increase with age.

Childhood obesity: Infants with excessive weight gain have an increased incidence of

obesity in later life. One third of obese adults have been so since childhood.

Antenatal complications associated with Obesity.

Sex: Women generally have higher rate of obesity than men, although men have higher rate of overweight.

Pregnancy and Parity: It has been claimed that women's BMI increases with successive pregnancy. The evidence suggested that this increase is likely to be about 1kg / pregnancy. Hence multiparous women are obese when compared to nulliparous women.

Genetic Factors:

There is a genetic component in the etiology of obesity.

Physical Inactivity:

Sedentary life style particularly sedentary occupation and inactive recreation promote it. Physical inactivity may cause obesity which in turn restricts activity. This is a vicious cycle.

Socio Economic Status:

Inverse relationship between socio economic status and obesity exist.

Eating Habits:

Eating in between meals, preference in sweets, refined foods and fats composition of the diet, periodicity with which it is eaten and the energy derived from it are all relevant to the etiology of obesity.

Familial Tendency:

Obesity frequently runs in families.

Endocrine Factors: These factors may be involved in occasional cases.

Eg. Cushings syndrome, growth hormone deficiency, hypothyroidism.

Alcohol:

The relationship between alcohol and adiposity is positive for men and negative for women.

Education:

In affluent countries, inverse relationship between education and prevalence of obesity is seen.

Ethnicity:

Ethnic groups in many industrialized countries appear to be especially susceptible to the development of obesity and its complications. This may be due to genetic predisposition.

Drugs:

Use of certain drugs e.g. Corticosteroids, Contraceptives, Insulin, Beta blockers can promote weight gain.

HAZARDS OF OBESITY

Metabolic Syndrome:

Obesity interacts with inherited factors and leads to the onset of insulin resistance. This metabolic abnormality in turn is responsible for altered glucose metabolism and a predisposition to type 2 diabetes and cardio vascular diseases and accelerate its course. The most important are type 2 diabetes, dyslipidemia and hypertension. Prevalence is increased with age. According to NHANES III, prevalence was about 6% in those with20years of age, 14% in those with 30-39 years of age, 20% in those with 40-49 years of age, >30% for women over 50years of age and 20% in reproductive age group**.**

Figure 5 -Criteria for the diagnosis of Metabolic syndrome as in Harrisons principles of internal medicine edition 20:

NCEP:ATPIII 2001 Criteria		
Three or more of the following:		
Central obesity: Waist circumference >102 cm (M), >88 cm (F)		
Hypertriglyceridemia: Triglycerides ≥150 mg/dL or specific medication		
Low HDL cholesterol: <40 mg/dL (M) and <50 mg/dL (F) or specific medication		
Hypertension: Blood pressure ≥130 mm systolic or ≥85 mm diastolic or specific medication		
Fasting plasma glucose ≥100 mg/dL or specific medication or previously diagnosed type 2 diabetes		
IDF Criteria		
Differ from NCEP: ATPIII 2001 criteria by more stringent and ethnicity-specific waist circumference limits. Other criteria are the same.		
Waist circumference:	Europid,	≥94 cm (M), ≥80 cm (F)
	Sub-Saharan African	
	Eastern and Middle Eastern	
	South Asian, Chinese	≥90 cm (M), ≥80 cm (F)
	South and Central American	
	Japanese	≥90 cm (M), ≥80 cm (F)

Abbreviations: HDL, high-density lipoprotein; IDF, International Diabetes Foundation; NCEP:ATPIII, National Cholesterol Education Program, Adult Treatment Panel III.

EFFECT OF OBESITY ON PREGNANCY

ANTEPARTUM COMPLICATIONS

Infertility:

Overweight and obese women need longer time to conceive and undoubtedly are at higher risk of infertility. However, as early symptoms of dysfunctional oocyte maturation and hormone derangements, oligomenorrhea and alterations of menstrual cycles should primarily alert overweight and obese women on their potentially defective fertility. The impact of obesity on reproductive function, especially associated with ovulatory disorders, is mainly due to neuroendocrine mechanisms, which interfere with ovarian functions, and are able to affect the ovulation rate and the endometrial receptivity. Obese women, even with normal menstrual cycles and apparently normal fertility, have lower circulating levels of gonadotropins, estradiol and inhibin in the follicular phase, suggesting an inhibitory effect of the obesity condition per se on the production of these hormones.

Since the obesity is pathogenically associated to inflammation, all mechanisms enrolled in oocyte differentiation and maturation including hormones, proteins and soluble factors released by adipocytes are deregulated and affected in their physiology. The direct effect of adipose tissue in lowering the fertility potential in women is thus based on the derangement of major molecular mechanisms that regulate the normal biological activity of cell components of the woman

reproductive organs also controlled through the hypothalamus hypophysis ovaries axis.

Pre-Pregnancy Medical Disorders:

Due to their strong association with obesity in the general population essential hypertension and diabetes mellitus are the two most common medical complications of obese gravida.

Other obesity associated morbidities such as Coronary heart disease, stroke and cancer have a low prevalence in the reproductive age group. Obstructive sleep apnea is a rare but serious obesity related morbidity. Data on this complication during pregnancy though limited suggested that obstructive sleep apnea may be precipitated or exacerbated during pregnancy and may be associated with hypertensive disorders during pregnancy and impaired fetal growth.

Pregnancy Specific Complications[12]

-Overt and gestational diabetes

-Pregnancy induced Hypertension

-Respiratory complications such as asthma and sleep apnoea

-Thromboembolic disease

-Infections of urinary tract , wound infections and endometritis.

Gestational Diabetes Mellitus

Obesity is a risk factor for carbohydrate intolerance in both pregnant as well as non pregnant women. Out of the 17 % of obese women, approximately 1 – 3 % develop Gestational diabetes. Studies have shown that the association between

diabetes, hypertension and obesity may be a manifestation of the X syndrome. Screening for gestational diabetes needs to be performed twice in obese patients, at 24 weeks and again at 32 – 34 weeks even with the first result being normal, as women with a risk of developing diabetes has also an increased risk of developing hypertension, macrosomia in the infant and finally predisposing the woman at a risk of developing type 2 diabetes in later life. Studies have also shown that obese women have two times the risk of delivering babies with congenital malformations than those without. Diabetic women were also seen to have increased weight retention in the postpartum period.[34]

Hypertensive Disorders*:*

The association between obesity and hypertensive disorders during pregnancy has been a consistent finding in the obstetrical literature. Specifically, maternal weight and BMI have been validated as independent risk factors for pre-eclampsia. Sibai et al reported a significant difference in the incidence of pre-eclampsia for women with an early second trimester BMI <20 kg/m2 (4.3%) as compared to when the BMI was > 34 kg/m2 (12.6% , $P < 0.0001$). The mechanism by which obesity imparts an increased resistance and subclinical inflammation and endothelial dysfunction are also responsible for the increased incidence of pre-eclampsia in obese gravidas.

Pre-existing heart disease

"Morbidly obese women are almost five times more likely to develop preeclampsia, even when diabetes and hypertension are taken into account". In

other words, excessive weight gain and obesity directly affects the likelihood of developing preeclampsia in pregnancy, regardless of preexisting diabetes or high blood pressure. In addition, cardiac complications can arise throughout pregnancy secondary to excessive weight gain and obesity. "Heart disease complicates more than 1% of pregnancies and is now the leading cause of indirect maternal deaths". In particular, excessive weight gain and obesity exacerbates underlying heart conditions due to the increased cardiac demands throughout pregnancy. Hemodynamic changes in pregnancy include "expected increases in preload, cardiac output, and oxygen consumption coupled with the normal decrease in after load" which "may unmask or worsen cardiac disease in the pregnant woman"

Respiratory complications

Increased BMI has been shown to be associated with sleep apnoea and asthma. It was shown that obese women had a 4% oxygen desaturation when compared to non obese women. The probable cause could be the deposition of excess fatty tissue around the neck causing obstruction and difficulty in breathing during sleep leading to apnoea. A neck circumference of 40.5 cm in women and 43 cm in women has shown to be associated with difficulty inbreathing, recurring upto 30 times a night. Problems associated with sleep apnoea are stroke, arrhythmias, pulmonary hypertension, right heart failure and even death due today time somnolence- while driving.

Thrombo – embolic complications

Thrombo- embolism is another risk factor associated with obesity. Obesity is associated with a 12 fold increase in thromboembolism. Many deaths have occurred due to thrombo- embolism in pregnancy. Studies have shown that the ratio of deaths from thrombo- embolism in pregnant women is 1 in 70,000, when compared to 1 in a million in a non pregnant state. The effective prophylaxis in pregnancy is Low molecular weight heparin which can be given to all antenatal women during the early pregnancy period, who are at high risk of developing thrombo- embolism, obesity being one of them.[35]

Adverse labour outcomes associated with Obesity[12]

- Dysfunctional Labour
- Shoulder dystocia
- Induction of labour
- Operative intervention rates (emergency LSCS and vaginal tears)

Dysfunctional Labour

Investigations on the labour characteristics of the obese gravidas are limited and conflicting. Gross et al found no difference in the major dysfunctional labour patterns between obese and non obese parturients. Ekblad et al also found no difference in the duration of the first and second stage of labour between obese parturients or those with excessive weight gain and controls. However Johnson et al reported a higher risk for labour abnormalities with both increasing pre pregnancy BMI and gestational weight gain.

Shoulder dystocia

Shoulder dystocia has been found to be associated with obesity, probably due to the link between diabetes and obesity. Babies of overweight and obese women are 60 – 100 g heavier than normal weight women ,thus increasing the chances of shoulder dystocia .

Induction of Labour

The incidence of labour induction is 1.7 – 2.2 folds more in obese women compared to women with normal weight. There is increased incidence of postdated pregnancies, prolonged labour and failure to progress in obese women and lower chances of spontaneous onset of labour at term. The need for early induction is because of factors such as hypertension, pre- eclampsia and diabetes associated with obesity.

Operative interventions

Operative intervention rates such as emergency LSCS and vaginal tear repairs associated with shoulder dystocia and macrosomia are much higher in obese women than women with normal BMI. The primary intrapartum complication of obesity is an increased risk for cesarean delivery. Both pre-pregnancy obesity and excessive maternal weight gain contribute to an increased cesarean risk. Importantly these associations appear to be independent of obesity related antenatal complications, short maternal stature, higher infant birthweights, and gestational age at delivery. The factors that contribute to obesity related increased cesarean risks are not clear. In a large population based cohort study of

nulliparas conducted in Sweden, Cnattingius et al demonstrated that cesarean rates increased consistently with decreasing maternal height and increasing pre pregnancy BMI. Subsequently, Young et al reported that among a large cohort of nulliparous women the obesity related increase in cesarean was primarily mediated through an increase in cesarean for cephalopelvic disproportion, failure to progress, which was independent of maternal height. As previously discussed, there is a lack of consistent evidence to support a higher incidence of specific dysfunctional labour patterns among obese parturients. These preliminary data therefore suggest that obesity may lead to dystocia due to increased soft tissue deposition of the pelvis.

Intraoperative Complications

Cesarean in the obese gravida is more often performed emergently and is associated with prolonged incision to delivery interval, blood loss >1000ml, longer operative times and difficulty in delivering the baby. The incidence for LSCS for obese women was over20 % compared to 10 % for normal weight women.

Studies have shown that the need for instrumental deliveries by forceps has also doubled in obese women. It has been seen that Primary caesarean section is most commonly done for cephalopelvic disproportion.

Postnatal complications associated with Obesity

• Postpartum haemorrhage

• Postpartum wound infections

• Longer hospital stay

• Maternal mortality

• Lactation problems

Whether delivered vaginally or by cesarean the obese gravida is at higher risk of postpartum endomyometritis, laceration/episiotomy infection and wound infection. Several studies reported a lack of association between postpartum hemorrhage and maternal obesity. Lactation dysfunction may be another postpartum complication of obesity.

The cumulative effect of obesity related complications during the postpartum period is a resultant prolongation of hospitalization. Prolonged hospitalization for the obese gravida ultimately translates into increased health care costs.[36]

Postpartum haemorrhage

Obese women are more prone to postpartum haemorrhage probably due to trauma associated with difficult deliveries and inability of the uterus to contract adequately in the postpartum period.

Wound infections:

Delayed wound healing is a common postpartum complication associated with obesity, due to the increase in abdominal wall fat thickness preventing healing by primary intention, which is further delayed in the presence of diabetes. This increases the hospital stay. In such cases, an abdominal drain can be kept in situ which will drain out any collected fluid and promote faster wound healing.

Maternal mortality

Postoperative chest complications may lead to venous stasis and thrombosis. In such patients, prophylactic administration of low molecular weight heparins could be helpful and life saving until ambulation.

Lactation problems

There is failure of initiation of lactation and also decreased duration of lactation in obese women.

Adverse fetal outcomes associated with obesity[12, 17]

• Congenital malformations (neural tube defects, cleft palate)

• Macrosomia

• Miscarriage and intrauterine death.

• Fetal growth restriction (FGR)

• Increased NICU admissions

• Fetal distress and low Apgar score

Congenital malformations

Obese women are at an increased risk for neural tube defects, the risk being around 7%. Studies have shown that obese women have a two - three fold increased incidence in heart defects, omphalocele and other anomalies.

Macrosomia

Fetal growth is strongly associated with increased maternal pre-pregnancy weight and decreased pre-pregnancy insulin sensitivity. Macrosomia is defined as birth weight > 4,000 grams. Studies have shown that with every 5 kg increase

in weight during pregnancy, there was a 30 % increase in the risk of macrosomia. The incidence of macrosomias

- 8.3% in normal weight
- 13.3% in obese
- 14.6% in morbidly obese

In early pregnancy, increased maternal insulin resistance alters placental function and also increases feto placental availability of glucose, free fatty acids and amino acids.

Miscarriage and Intra uterine death

Studies have shown that, obese parous women had a significantly increased risk of late fetal death relative to women with normal BMI .There was a 3 fold increased risk of ante partum still birth in morbidly obese women.

- 1.6 folds increase when BMI was 25 to 29.9 kg/m2
- 2.6 fold increase when BMI was >30 kg/m2
- 3 fold late still birth rate when BMI >40 kg/m2

Rapid fetal growth due to fetal hyperglycemia increases the risk of still birth. In such situations, placenta cannot transfer sufficient oxygen for metabolic requirements, causing hypoxia and death.

Small for gestational age and FGR

With increasing BMI, the risk of delivery a SGA baby decreases. The same is associated with increasing BMI and FGR. But studies have shown that prematurity and low birth weight is associated with obesity.

Relation between NICU admissions, Low Apgar and Obesity

Obese women are at a higher risk of having difficult deliveries using instrumentation. This could be because of the large size of the fetuses that can be delivered only with instrumentation. The traumatic delivery associated with it and the increased risk of fetal distress with low Apgar Scores in such women has also lead to higher rates of NICU admissions.

Morbidity in children born to obese women

• Childhood obesity

• Reduced breast feeding and increased over feeding

• Increased exposure to a high calorie diet after weaning

• Increased risk of cardio- metabolic complications in adult life

Preconception counseling[37]

The goal of preconception counseling is to improve pregnancy related outcomes by identifying and modifying any health risks of the potential mother before conception and educating her on methods to reduce these risks. These include:

- Diet, Exercise, and Weight Loss Counseling at Preconception Visits

- Strategies for Weight Control, Diet, and Exercise Counseling before conception

ACOG purports that "preconception assessment and counseling are strongly encouraged and should include the provision of specific information concerning the maternal and fetal risks of obesity in pregnancy. At the initial prenatal visit, height and weight should be recorded for all women to allow calculation of BMI, and recommendations for appropriate weight gain, guided by IOM recommendations, should be reviewed both at the initial visit and periodically throughout pregnancy. Nutrition consultation should be offered to all overweight or obese women, and they should be encouraged to follow an exercise program. Nutrition and exercise counseling should continue postpartum and before attempting another pregnancy"

In pregnancy

Women should be counseled to limit weight gain according to their BMI. IOM has proposed recommendations regarding adequate weight gain in pregnancy according to the respective BMI.

Obese women should ideally not lose weight during pregnancy due to increased risk of ketosis. Early signs of diabetes or hypertension should be carefully watched for. Standard screening tests for fetal anomalies is to be done. Accurate assessment of fetal growth using serial sonography is to be done.[17]

Post partum

Obese women require longer period of hospitalization. Graduated compression stocking, hydration and early mobilization is recommended. Low molecular weight heparin can be given prophylactically in postoperative patients until they start mobilizing. Breast feeding should be encouraged. Obese women have a tendency for increased postpartum weight retention. Exercise and diet control is crucial at this stage.[12]

Contraception:

Oral contraceptive pill failure is more likely in overweight women. According to Holt and Colleagues 2002 women in the highest weight quartile had sixteen- fold increased risk of pregnancy. Women who used very low dose OCP had 4-5 fold increase in pregnancy rate**.**

Combined OC pills are contraindicated in morbid obesity. There is also increased risk of venous and arterial thrombo-embolism and increased failure rate in obese women. Intrauterine devices have shown to be effective.[12]

Underweight and Pregnancy[39]

According to WHO, Underweight is defined as BMI<18.5 kg /m2.

Nutrients are classified as Macronutrients (Proteins, carbohydrates and fats), Micronutrients (Vitamins and minerals), and water. As a result, we can suffer from serious deficiency diseases that can affect the quality of our lives.

What Causes Malnutrition?

1. Ignorance: Not knowing about the importance of nutrients can lead to malnutrition as the individual will not have a healthy, balanced diet.

2. Illness and Infections: Diarrhoea and vomiting can prevent a person from getting adequate nutrition. Illnesses, infections, and mental illnesses like depression can also affect a person's ability to consume and digest nutritious food. They can cause a loss of appetite and affect the digestive system.

3. Socio-Economic Conditions

Families in low-income groups may lack financial resources to buy healthy food. This can lead to malnutrition in such individuals.

4. Medication

Use of some kinds of medicines can disrupt nutrient absorption in the body, thus causing malnutrition.

5. Morning Sickness

Severe morning sickness during pregnancy can hamper a woman's ability to consume healthy food and may lead to malnutrition.

6. Insufficient Intake

During pregnancy, a woman requires around 300 extra calories a day. If the woman does not consume adequate quantities of healthy food, it can lead to malnutrition.

Health Risks of Malnutrition In Pregnancy

Malnutrition during pregnancy can cause several health problems in both the mother-to-be and her developing baby. Here are the health risks of malnutrition during pregnancy:

1. Risks for the Mother

- **Maternal Mortality –** Women who are under-nourished before and during pregnancy have a higher risk of dying during pregnancy or childbirth.
- **Risk of Miscarriage –** Under-nourished women are at a higher risk of miscarrying.
- **Dental Problems –** Moms-to-be who are malnourished can suffer from tooth decay and other dental problems.
- **Osteomalacia –** This is a condition where the bones of a malnourished woman become too soft and brittle due to lack of calcium.
- **Anaemia –** Iron deficiency can cause anaemia. This means that they have fewer red blood cells than normal, so the body's cells do not receive enough oxygen.
- **Preeclampsia** - a condition where the blood pressure and the protein level in the blood of a pregnant woman are dangerously high. This can endanger the life of both the mother and the baby.

2. Risks for the Baby

Malnutrition during pregnancy effects on the baby inside the womb, too.

- **Stillbirth –** Babies that are malnourished do not grow and develop properly and could die in the womb.

- **Premature Birth –** Babies born prematurely are underdeveloped and could suffer from various problems such as poor vision, weak muscles, brain damage, poor growth rate, etc. They can also get necrotising enterocolitis, where bacteria invade and destroy their intestines.
- **Perinatal mortality –** Babies of women who were undernourished during pregnancy have a higher risk of dying in the 1st week of birth.
- **Birth Defects –** Deficiency of micronutrients during pregnancy can cause serious birth defects in the baby. For example, deficiency of folic acid can cause Spina bifida in babies, where the baby is born with a deformed spinal cord. This affects their ability to walk, and control bowel and bladder movements.
- **Underdeveloped Organs –** Malnourished babies can be born with underdeveloped organs, which can seriously affect the quality of their lives.

3. Long-Term Health Risk for the Child

- **Diabetes Mellitus –** Malnourished babies are at a much higher risk of developing type-2 diabetes later in their lives.
- **Cardiovascular Diseases –** These babies also develop high blood pressure and heart disease in adulthood.
- **Osteoporosis –** Under-nourished babies suffer from osteoporosis, a condition where the bones are weak and brittle and prone to fractures.
- **Low IQ and Cognitive Impairment –** Under-nourishment also causes babies to grow up with lower IQ than normal and suffer from cognitive impairment, where

a person has problems learning new things, remembering, and making decisions in daily life.

How Can Malnutrition Be Prevented?

Management options[17]

Pre pregnancy

Women with anorexia or bulimia who wish to conceive are advised to wait until the remission period. Women who wish to conceive with problems of anovulatory infertility, should be advised to gain weight rather than being started on ovulation drugs.

Pre Natal

In view of the increased risk of low birth weight, early detection of FGR should be made. Careful dating of gestation is important. Patient is asked to gain weight adequately and weight monitoring has to be done including daily calorie intake chart. Anemia may be corrected with oral or parental preparations.

Labor and Delivery

If fetal growth restriction is suspected, the patient should be admitted to a higher center where continuous electronic fetal heart rate monitoring is advised, in order for timely detection of fetal distress. Emergency neonatal services should be readily available for resuscitation. Anemia, if present may be corrected with blood products and iv fluids may be given as needed.

Post Natal

The diet of a lactating women should be fairly liberal and of a varied nature. Plenty of proteins, meat, fish, fresh fruits and green vegetables. The total diet should have an additional 400-700kcal to cover the energy requirements of lactation (Mudhaliar and Menon's Obstetrics edition 12).

Anemia may be corrected with oral or parental preparations and iron calorie rich diet is advised to be taken. Malnutrition can be prevented by having a balanced diet which includes plenty of fruits, vegetables, water, dietary fibre, proteins, fats, and carbohydrates. Signs and symptoms of malnutrition in pregnancy include fatigue, anaemia, low pregnancy weight, dizziness, high blood pressure, hair loss, dry skin, dental problems, and low immunity.

To prevent malnutrition, women who plan to conceive should take prenatal vitamins, eat healthy food and exercise regularly. This ensures that both mother and the newborn are hale and healthy.

METHODOLOGY

Prospective Cohort Study:

In this prospective cohort study, data will be collected from pregnant women in their first trimester, while attending RMH OPD. They are then followed up.

INCLUSION CRITERIA :

- Singleton pregnancy.
- Women belonging to the age group between 20 to 34 years.
- Pregnant women on regular follow up.

EXCLUSION CRITERIA :

- Multiple pregnancy
- Women with overt diabetes and chronic hypertension.
- Those with severe systemic or surgical diseases before pregnancy.
- Women with irregular follow up and insufficient information about their gestational weight gain.
- Women with anomalous baby
- Women <20years and >35years.

RESULTS AND ANALYSIS

The Study conducted at Thanjavur Medical College, involves 300 pregnant women aged between 20 to 34 years, who qualified the inclusion and exclusion criteria and attended our AN OPD for the first time preferably in the first trimester. Of the women selected, self reported pre pregnancy weight obtained and they were categorized according to Asian BMI Classification as

- Underweight
- Normal weight
- Overweight
- Obese

The selected women were then followed up and their Gestational weight gain (GWG) was calculated as the difference between the women's pre gestational weight and her weight at delivery ,and further were categorised based on IOM (Institute of Medicine) recommended GWG and their association with fetomaternal outcomes were studied.

Institute of Medicine recommended Gestational weight gain classifies women into:

- Excessive weight gain
- Adequate weight gain

- Inadequate weight gain

The IOM-2009 are the most widely accepted recommendations for GWG, but it is not clear if these guidelines are also applicable to developing countries. In addition, there is also no such recommendation for GWG cut-off points available for all Asians. The present thesis focuses on pregnancy outcome and other pregnancy-related complications for singleton pregnancies among Indian with respect to IOM-2009 weight gain recommendations. It assesses the utility of IOM guidelines-2009 among the pregnant women of India in terms of maternal and fetal outcomes.

The results were tabulated and presented in chart for easy interpretation. Data are expressed as percentage (%). Chi Square test was applied to find statistically significant association between groups based on fetomaternal outcomes. $p<0.05$ was considered to be statistically significant.

Statistical analysis:

Data were entered in the excel spread sheet and variables were coded accordingly. The statistical analyses were performed using Graph pad Prism version 5 software. Data were presented as mean with Standard deviation for normal distribution/scale data. Data were presented as frequency with proportion n(%) for categorical data. Fisher's exact test was used to compare the frequencies between the groups.$p<0.05$ were considered statistically significant.

Table 7 Frequency distribution of type of BMI class observed in the study.

S.No	Type of BMI	N	%
1	Underweight	90	30
2	Normal	120	40
3	Overweight	60	20
4	Obese	30	10

Data are expressed as n with %. The total N=300

- Majority of pregnant women in the study had normal BMI .Out of 300 pregnant women included 120 women had normal BMI, contributing to 40% .
- 30% of pregnant women were under weight.
- The least distribution was seen among obese class.

Figure 6:

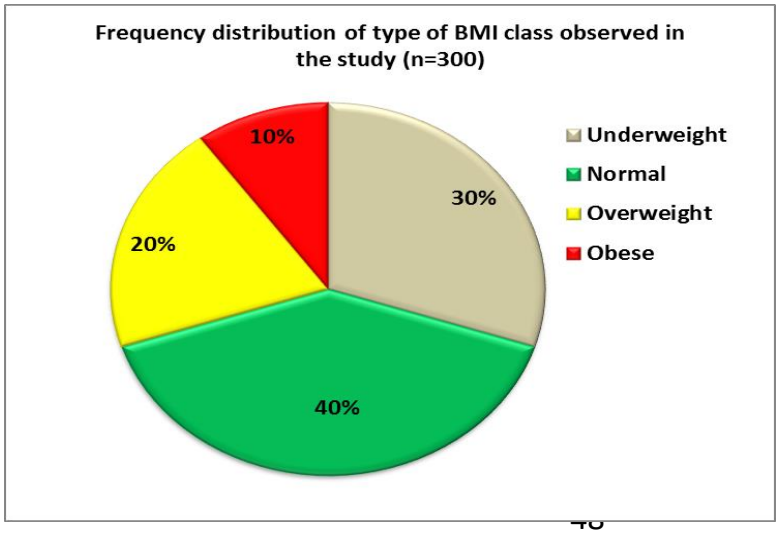


Table 8. Frequency distribution of type of Gestational weight gain based on IOM criteria observed in the study.

S.No	Gestational weight gain based on IOM	N	%
1	Appropriate	146	48.7
2	Inadequate	56	18.7
3	Excess	98	32.6

Data are expressed as n with %. The total N=300

- Majority of pregnant women included in the study had appropriate weight gain.

Figure 7:

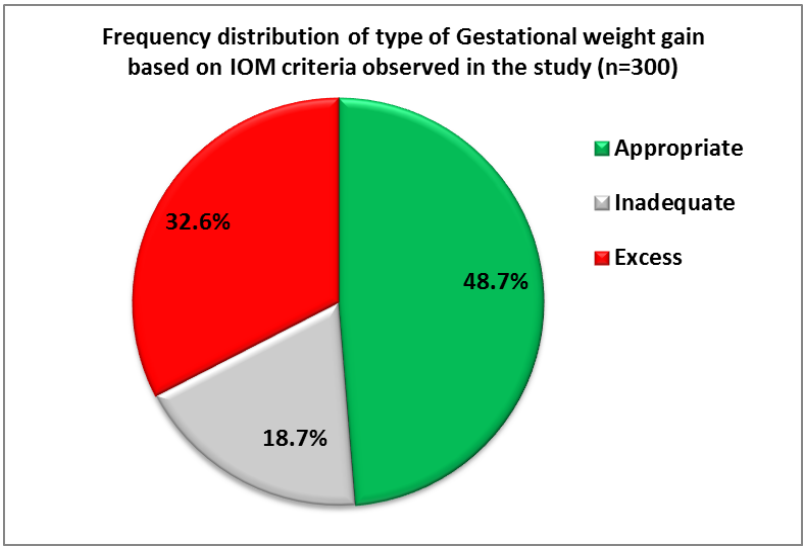


Table 9. Frequency distribution of age category of patients observed in the study.

S.No	Age category	N	%
1	20 – 24 years	148	49.3
2	25 – 29 years	115	38.3
3	≥30 years	37	12.3

Data are expressed as n with %. The total N=300.

- This table includes gestational age at which patient first fulfilled the inclusion criteria of the study.

- We can see from the table that majority of pregnant women attending RMH Thanjavur belonged to the age group between 20 to 24 years.

Table 10. Comparison of age category with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		N	%	N	%	N	%	n	%		
1	20 – 24 years	63	70	79	65.8	6	10	0	0	140.6	<0.001*
2	25 – 29 years	25	27.8	36	30	42	70	12	40		
3	≥30 years	2	2.2	5	4.2	12	20	18	60		

Data are expressed as n with %. Fisher's exact test was used to compare the frequency between the groups.

*indicates p<0.05 and considered statistically significant.

- Women with obese pre gestational BMI category were mostly elderly ,more than 30yrs of age ,contributing

to 60% of the total category.

- Women who were underweight mostly belonged to 20 to 24 yrs age group

about 70 % of the underweight BMI category

- Women who were overweight belonged mostly to 25-29years age group.

Table 11.Comparison of age category with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Intervention	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	N	%		
1	20 – 24 years	51	34.9	12	21.4	85	86.7	179.8	<0.001*
2	25 – 29 years	90	61.6	14	25	11	11.2		
3	≥30 years	5	3.4	30	53.6	2	2		

Data are expressed as n with %. Fisher's exact test was used to compare the frequency between the groups.

*indicates p<0.05 and considered statistically significant.

- It is clear from the table that,women who gained inadequate weight were mostly aged between 20-24 years contributing

to 86.7% of the total women belonging to the category.

- Women who gained weight excessively during pregnancy belonged to age group >30years contributing to 53.6% of total women belonging to the category.

- Women with appropriate weight gain were more among 25-29 years of age.

Table 12.Frequency distribution of type of socio economic status observed in the study.

S.No	Type of socioeconomic status	N	%
1	Class I	15	5
2	Class II	92	30.7
3	Class III	126	42
4	Class IV	59	19.7
5	Class V	8	2.6

Data are expressed as n with %. The total N=50

- Most of the pregnant women in the study belonged to socioeconomic class III followed by class II

- The least distribution was among class V followed by class I.

Figure 8:

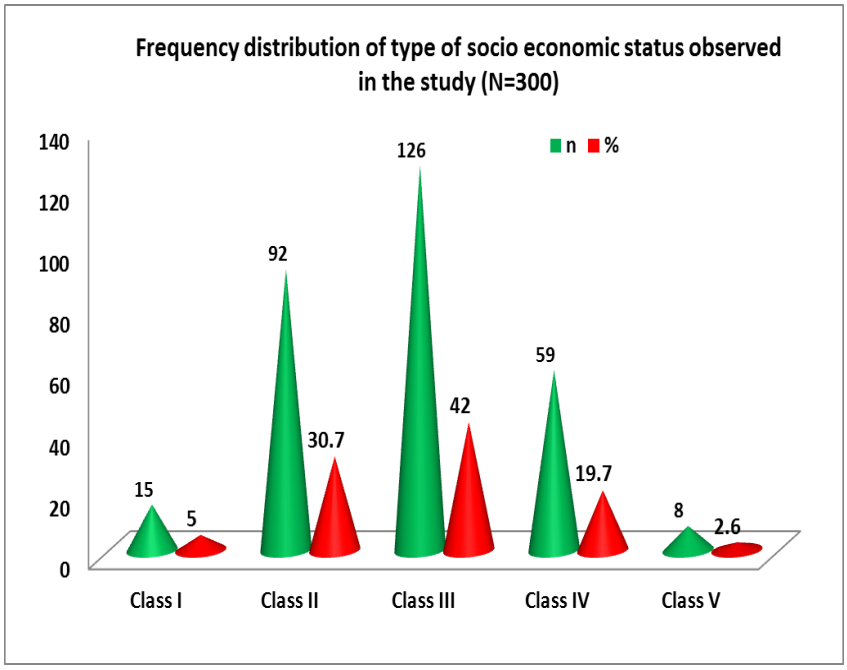


Table 13.Comparison of socio economic class with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	n	%	N	%	N	%		
1	Class I	1	1.1	0	0	12	20	2	6.7	178.9	<0.0001*
2	Class II	14	15.6	20	16.7	40	66.7	18	60		
3	Class III	28	31.1	81	67.5	8	13.3	9	30		
4	Class IV	39	43.3	19	15.8	0	0	1	3.3		
5	Class V	8	8.9	0	0	0	0	0	0		

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups.*indicates $p<0.05$ and considered statistically significant.

- Women who were underweight before conception ,significantly belonged to SEC IV (43.3%) and III (31.1%)
- Women who were overweight and obese significantly belonged to SEC II
- Majority of women with normal prepregnancy BMI in the study belonged to SEC III.

Figure 9:

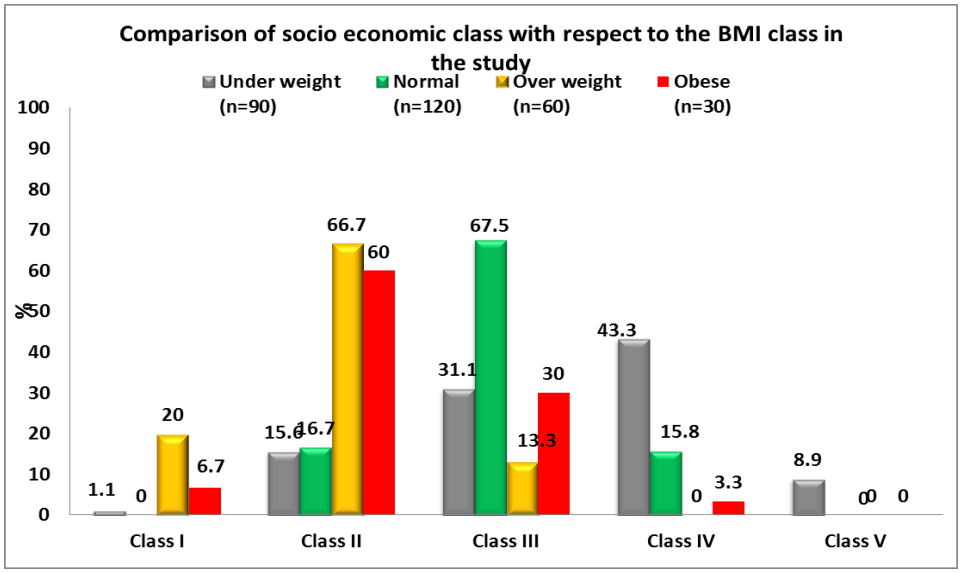


Table 14.Comparison of socio economic class with respect to the IOM class of weight gain in pregnancy in the study.

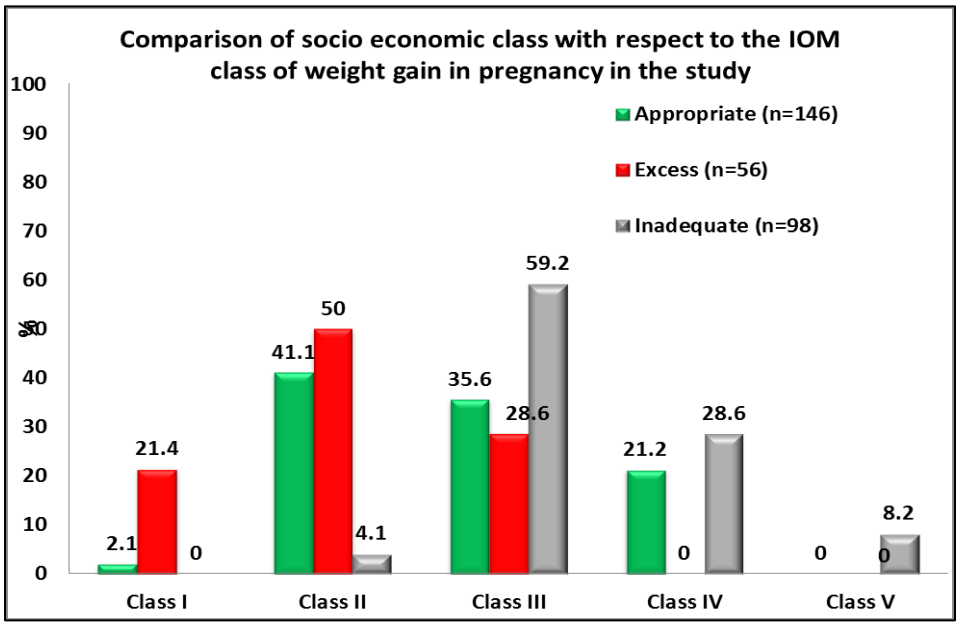
S.No	Intervention	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	n	%		
1	Class I	3	2.1	12	21.4	0	0	114.6	<0.001*

2	Class II	60	41.1	28	50	4	4.1		
3	Class III	52	35.6	16	28.6	58	59.2		
4	Class IV	31	21.2	0	0	28	28.6		
5	Class V	0	0	0	0	8	8.2		

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups.

*indicates $p<0.05$ and considered statistically significant.

Figure 10:



- Women with appropriate BMI mostly belonged to SEC II (41.1%) and III (35.6%)

- Women with inappropriate BMI significantly belonged to class III (59.2%) and class IV (28.6%)

- Those who had excessive weight gain during pregnancy were of high SEC II (50%)

Table 15. Frequency distribution of type of parity observed in the study:

S.No	Parity	N	%
1	Primigravida	189	63
2	Multigravida	111	37

Data are expressed as n with %. The total N=300

- Most of the pregnant women in the study were Primigravida.

Figure11:

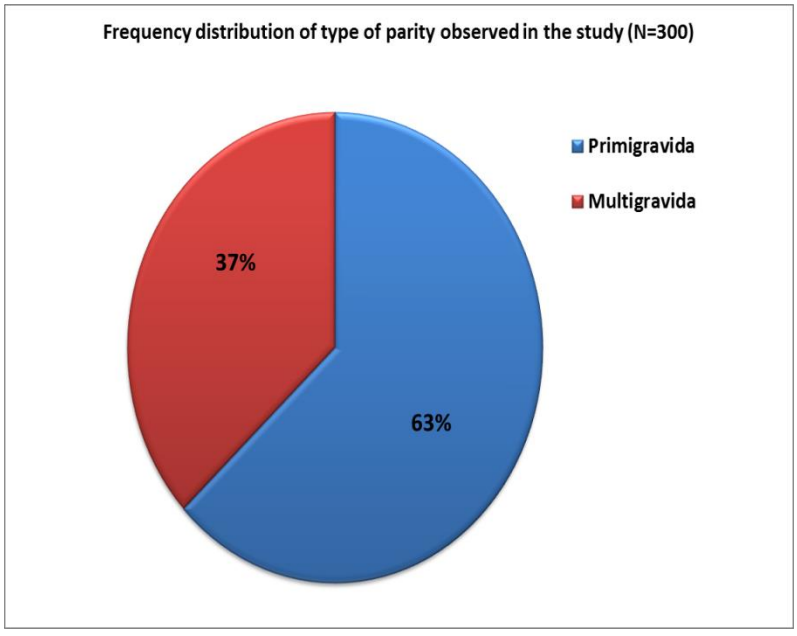
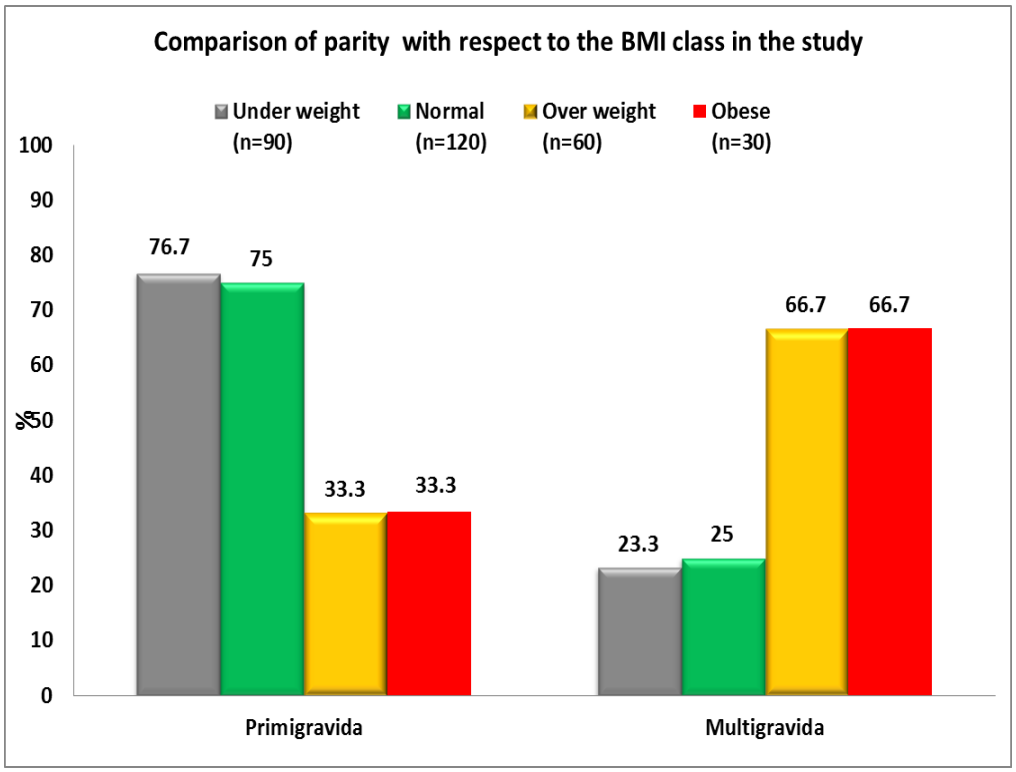


Table 16.Comparison of parity with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		N	%	n	%	n	%	n	%		
1	Primigravida	69	76.7	90	75	20	33.3	10	33.3	48.6	<0.0001*
2	Multigravida	21	23.3	30	25	40	66.7	20	66.7		

Figure 12:



Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups.*indicates p<0.05 and considered statistically significant.

- Multigravida women were mostly belonging to Obese (66.7%) and Overweight BMI (66.7%).
- Underweight women were mostly primigravidas with 76.7%.

Table17. Comparison of parity with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Intervention	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		N	%	N	%	N	%		
1	Primigravida	79	54.1	30	53.6	80	81.6	21.6	<0.001*
2	Multigravida	67	45.9	26	46.4	18	18.4		

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups.*indicates p<0.05 and considered statistically significant.

- Inadequate gestational weight gain were more among primigravida with 81.6% overall.

- In the study multigravida women had 46.4% of excess gestational weight gain and 45.9% appropriate weight gain**.**

Figure 13:

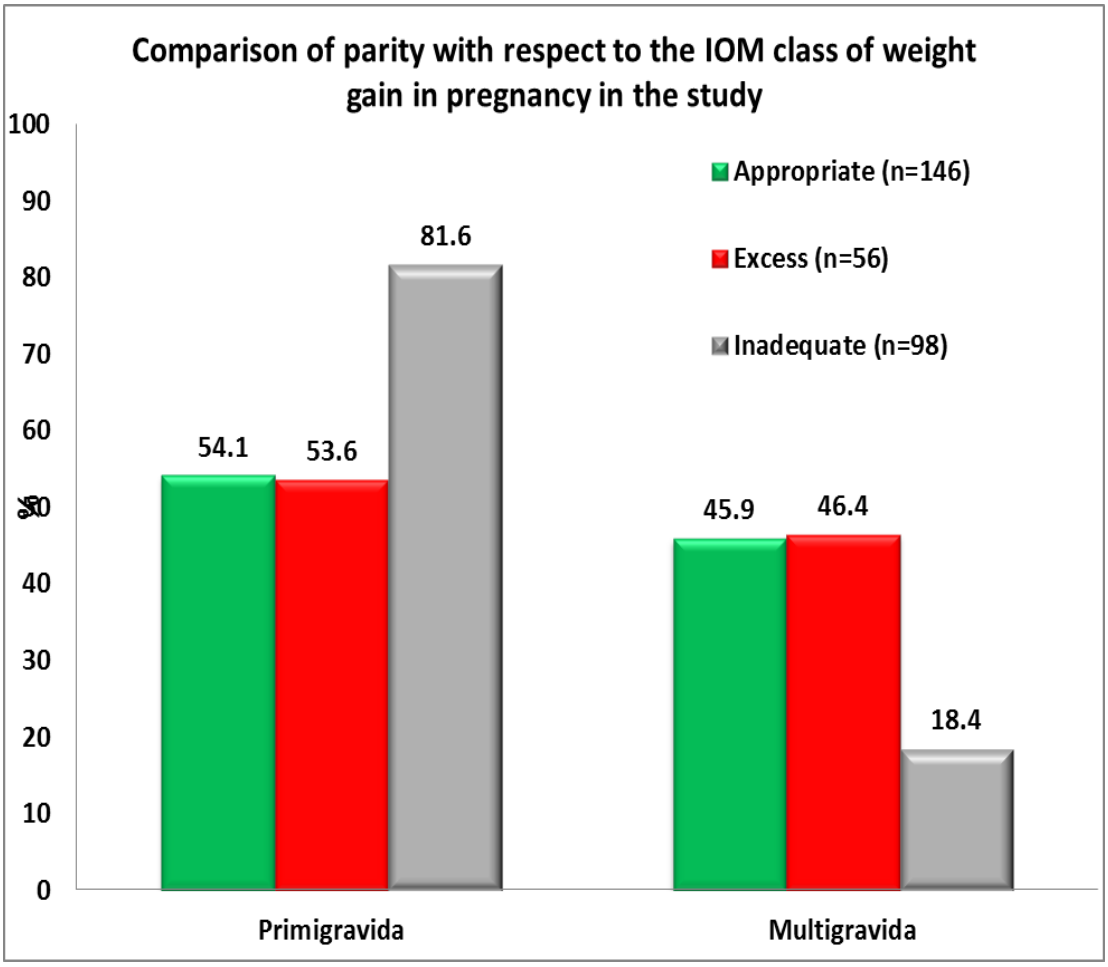


Table 18.Frequency distribution of gestational week at delivery observed in the study.

S.No	Gestational week at delivery	N	%
1	Term	133	44.3

2	Preterm	102	34
3	Post term	65	21.7

Data are expressed as n with %. The total N=300

- This table shows that majority of the women delivered at term contributing to 44%
- Only 21% post term deliveries were observed .

Figure 14:

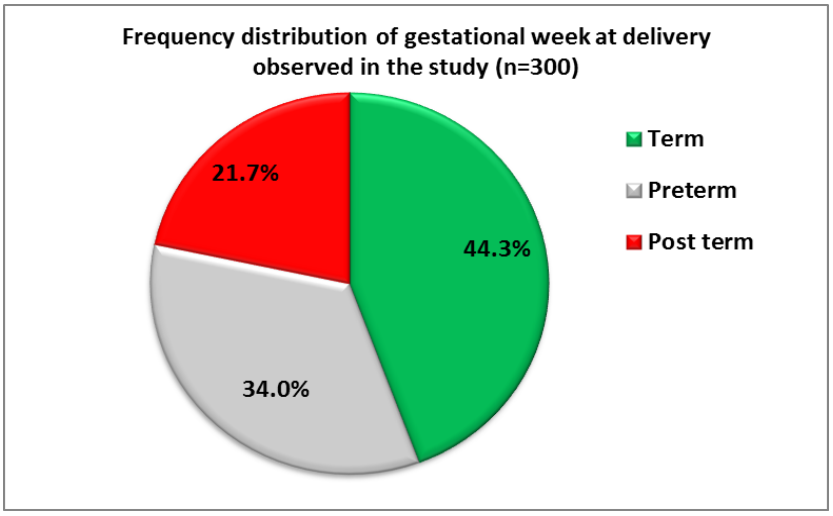


Table 19.Comparison of gestational week of delivery with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	n	%	n	%	N	%		
1	Post term	3	3.3	8	6.7	36	60	18	60	120.3	<0.0001*
2	Preterm	35	38.9	59	49.2	6	10	2	6.7		
3	Term	52	57.8	53	44.2	18	30	10	33.3		

Data are expressed as n with %. Fisher's exact test was used to compare the frequency between the groups.

*indicates $p<0.05$ and considered statistically significant.

- Post term deliveries were significantly more among women who were obese and overweight.

- 60% of deliveries in obese group were posterm deliveries . 60% of deliveries in overweight group were

significantly posterm deliveries.

- Compared to overweight and obese category ,preterm deliveries were on the higher side for underweight women with 38.9%, but not of much significance with normal BMI . This was because, underweight women ,who gained adequate weight gain delivered at term.

Figure 15:

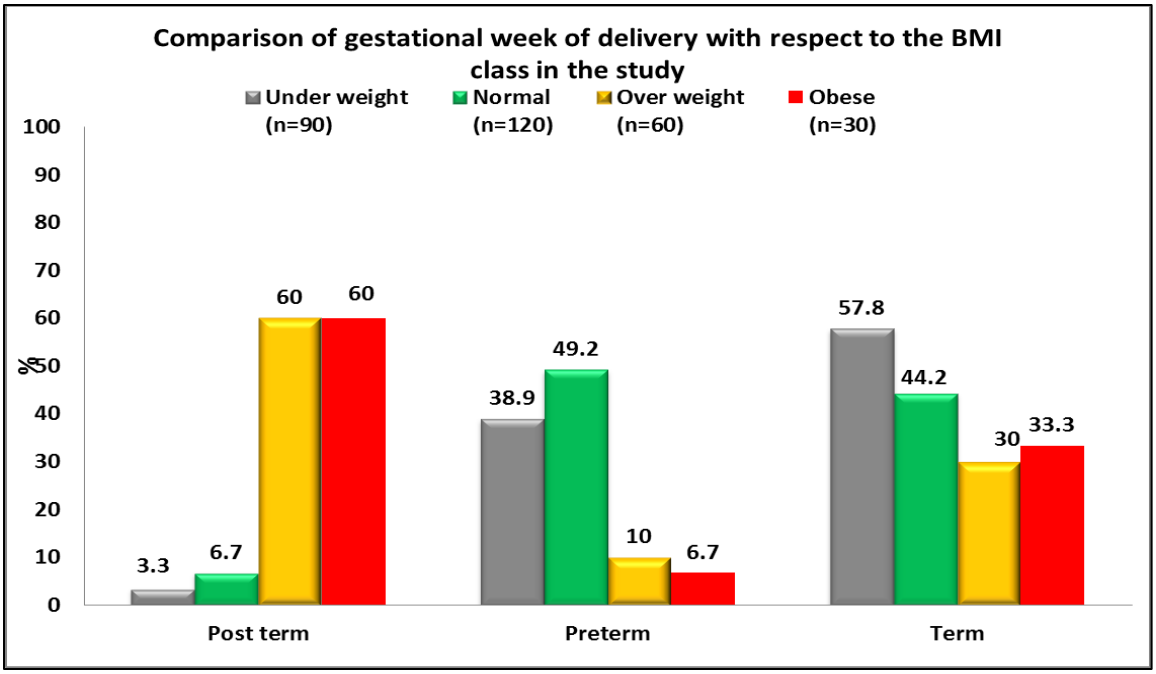


Table 20. Comparison of gestational week of delivery with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Intervention	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	n	%		
1	Post term	29	19.9	36	64.3	0	0	157.2	<0.001*
2	Preterm	26	17.8	4	7.1	72	73.5		
3	Term	91	62.3	16	28.6	26	26.5		

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups.

*indicates $p<0.05$ and considered statistically significant.

- Preterm deliveries were significantly more among woman with inadequate weight gain
- 73.5 % of preterm deliveries among inadequate GWG pregnant mothers.
- Post term deliveries were more in number among women who had excessive GWG, contributing to 64.3%.
- Majority of women included in the study with appropriate weight gain had delivery at term (62.3%).

Figure 16:

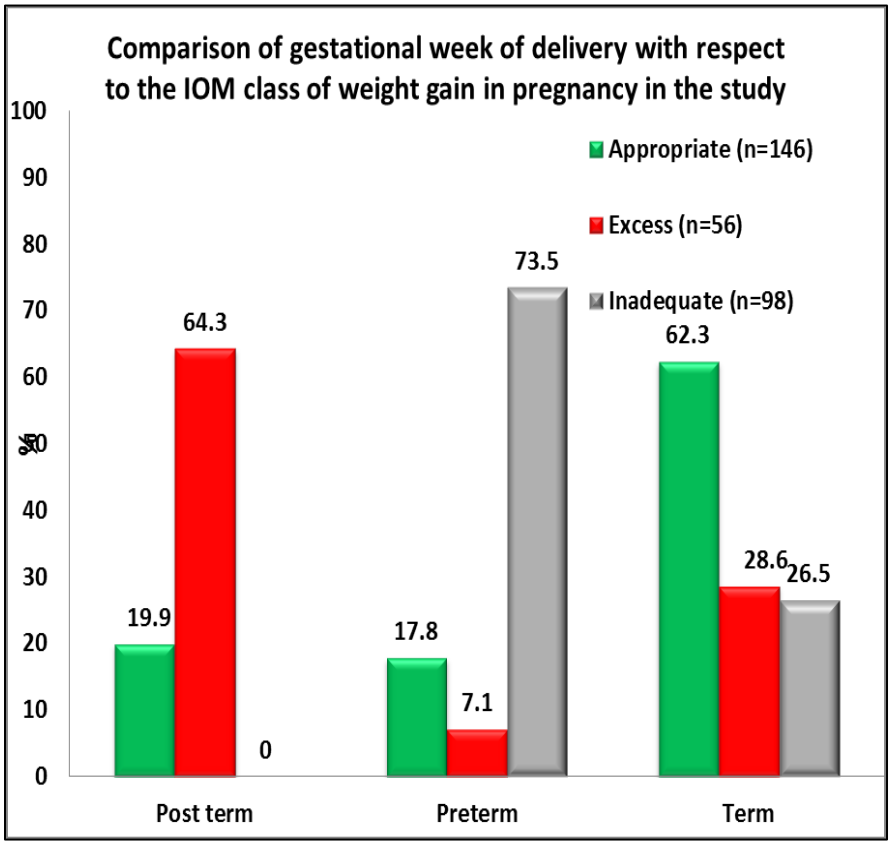


Table 21. Frequency distribution of mode of delivery observed in the study.

S.No	Mode of delivery	n	%
1	Labour natural	169	56.3
2	LSCS	102	34
3	Instrumental	29	9.7

Data are expressed as n with %. The total N=300

- Out of 300 deliveries included in the study, 56% were by labour natural ,being the highest.
- Followed by which interventional deliveries like LSCS were 34%.

Figure 17:

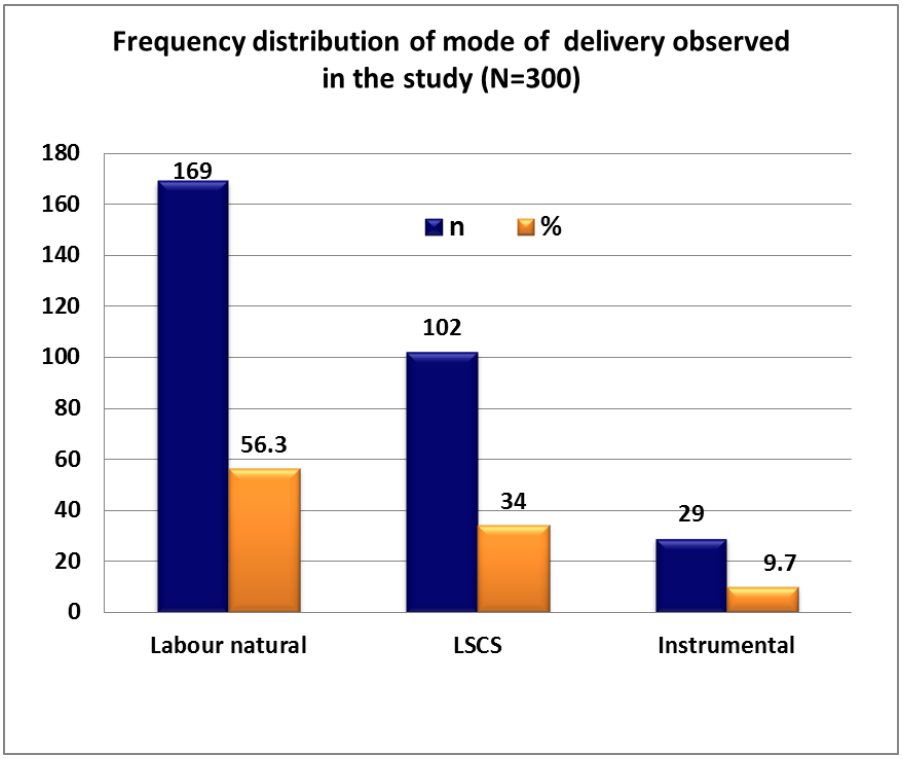


Table 22.Frequency distribution of requirement of induction of labour observed in the study

S.No	Induction of labour	N	%
1	Performed	114	38
2	Not done	186	62

Data are expressed as n with %. The total N=300

- In total, 38% of pregnant women included in the study were induced.

Figure 18:

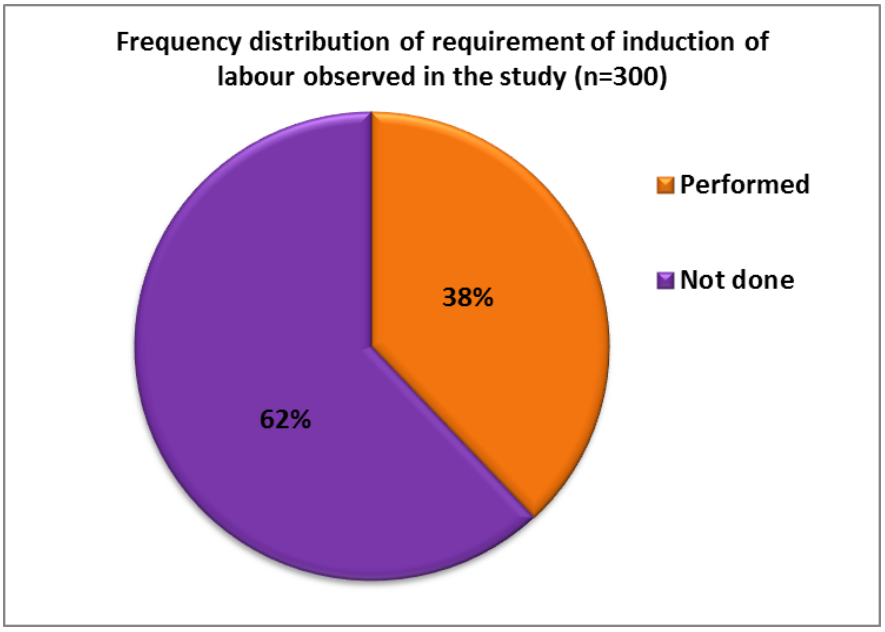


Table 23.Frequency distribution of reason for induction of labour observed in the study.

S.No	Reason for induction of labour	N	%
1	GDM	36	31.5
2	Post dated	32	28.1
3	GHT	32	28.1
4	FGR	24	21
5	Oligohydraminos	26	22.8
6	Rh-VE	10	8.7
7	PROM	9	7.9
8	PPROM	12	10.5
9	Severe PE	8	7

Data are expressed as n with %. The total N=114

- Overall the most common reason for induction were due to maternal co-morbitities which included GHT(gestational hypertension) including severe preeclampsia with 35.1% followed by GDM with 31.5%

- Other reasons for induction with close proximity were postdatism 32%, oligohydramnios 26% and FGR babies 24%.

Figure 19:

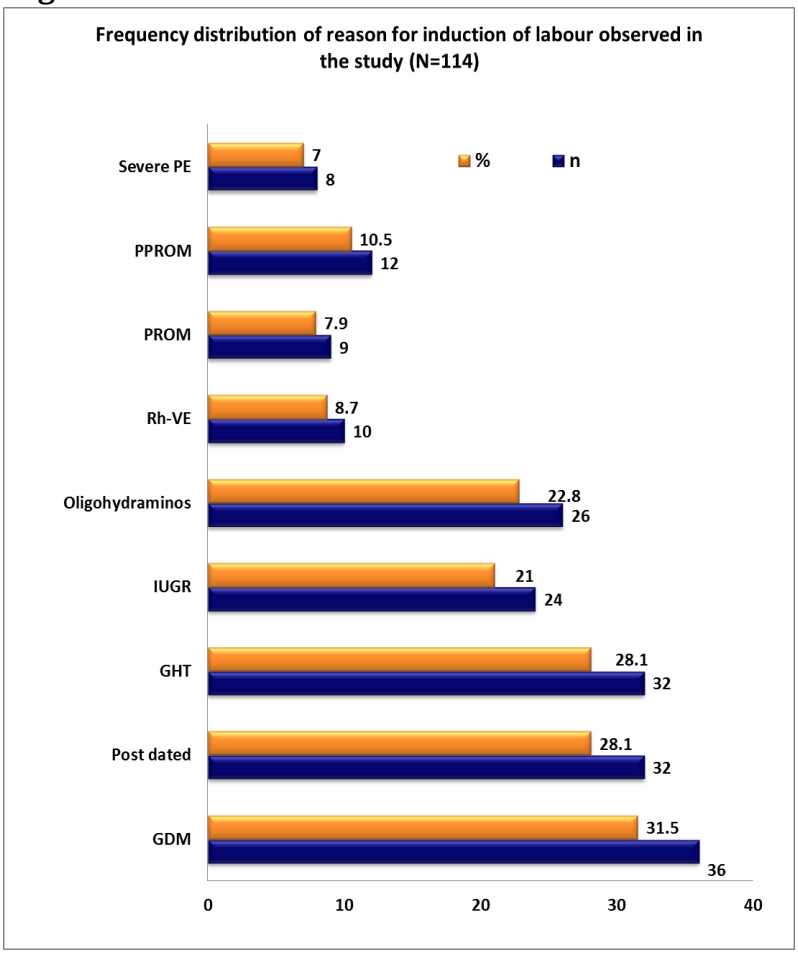


Table 24.Comparison of occurrence of labour natural with respect to the BMI class in the study.

S. No	Parameter	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		N	%	n	%	n	%	n	%		
1	Labournaturale	55	61.1	92	76.7	17	28.3	5	16.7	102.1	<0.0001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant

- Normal vaginal delivery was 76.7% among normal BMI women and 61.1% among underweight women.
- Only 16.7% of obese and 28.3% of overweight had normal vaginal deliveries.

Table 25.Comparison of occurrence of labour naturale with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Parameter	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	N	%		
1	Labournaturale	87	59.6	14	25	68	69.4	46.4	<0.0001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

Table 26.Frequency distribution of outcome for induction of labour observed in the study.

S.No	Outcome for induction of labour	N	%
1	Failure	47	41.2
2	Success	67	58.8

Data are expressed as n with %. The total N=114

- As we can see from the table that , majority of the induction for labour performed were

successful with 58.8%.

Figure 20:

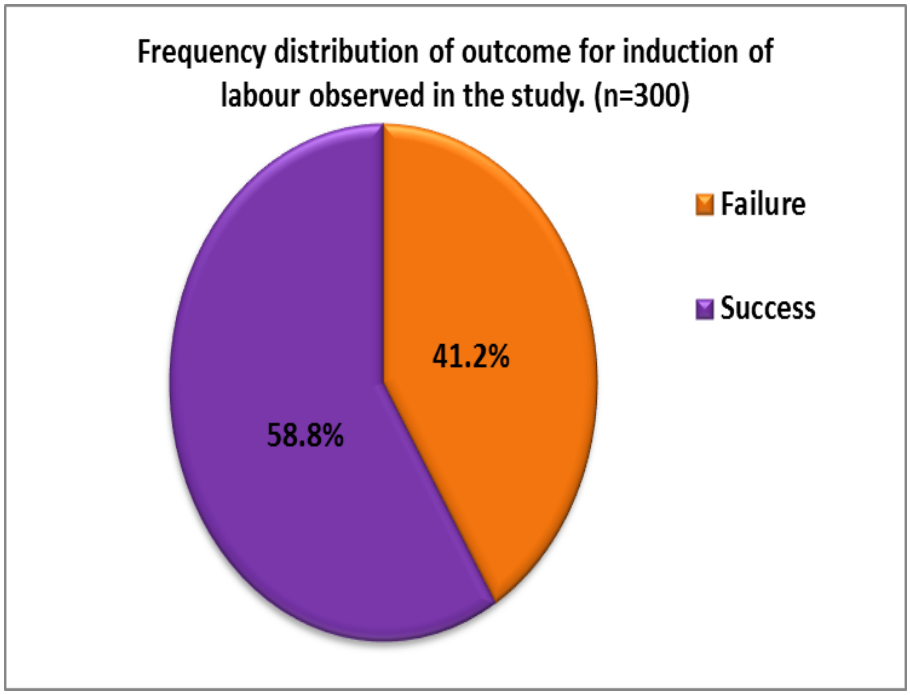


Table 27.Comparison of interventional mode of delivery with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	n	%	n	%	n	%		
1	LSCS	35	38.9	28	23.3	23	38.3	16	53.3	102.1	<0.001*
2	Induction of labour	39	43.3	22	18.3	35	58.3	18	60	37.4	<0.001*
3	Instrumental	0	0	0	0	20	33.3	9	30	102.1	<0.001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

1.While comparing the interventional mode of delivery with different BMI class, pregnant women who were obese and overweight had the most interventional deliveries 2.Induction of labour were maximum among obese women (60%) and overweight women 58.3% .

3.Women of the Underweight category also had a significant increase in interventional deliveries compared to the normal BMI women.

Figure 21:

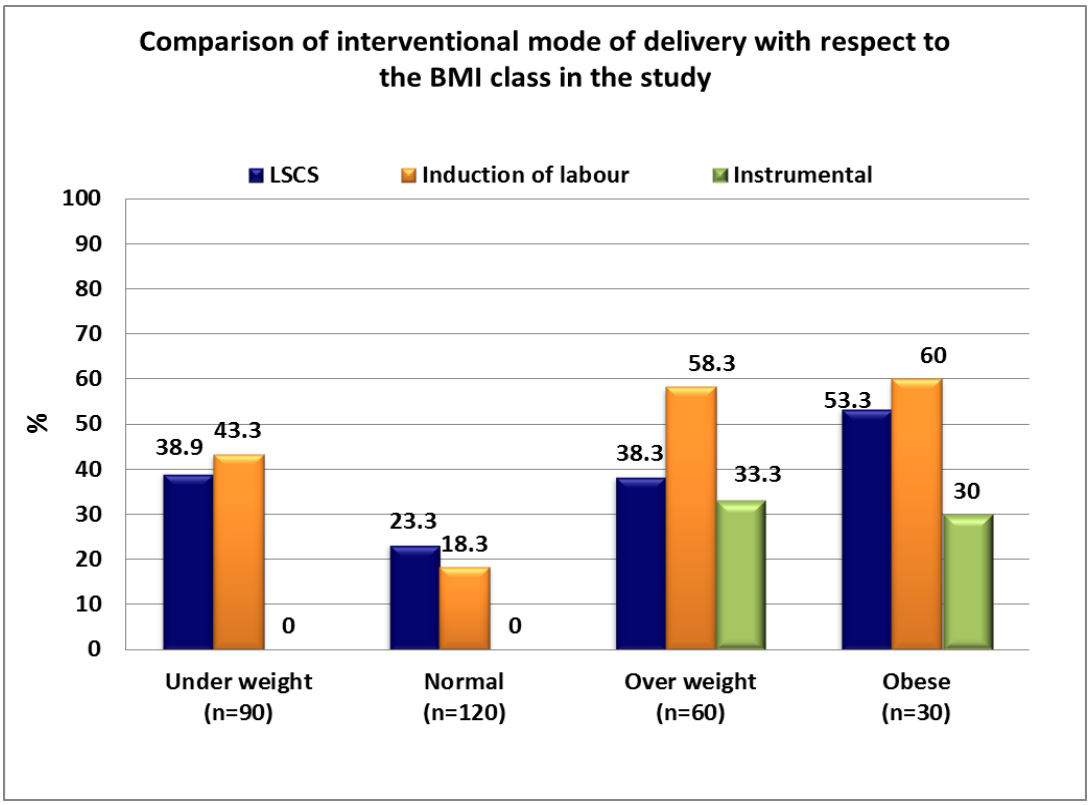
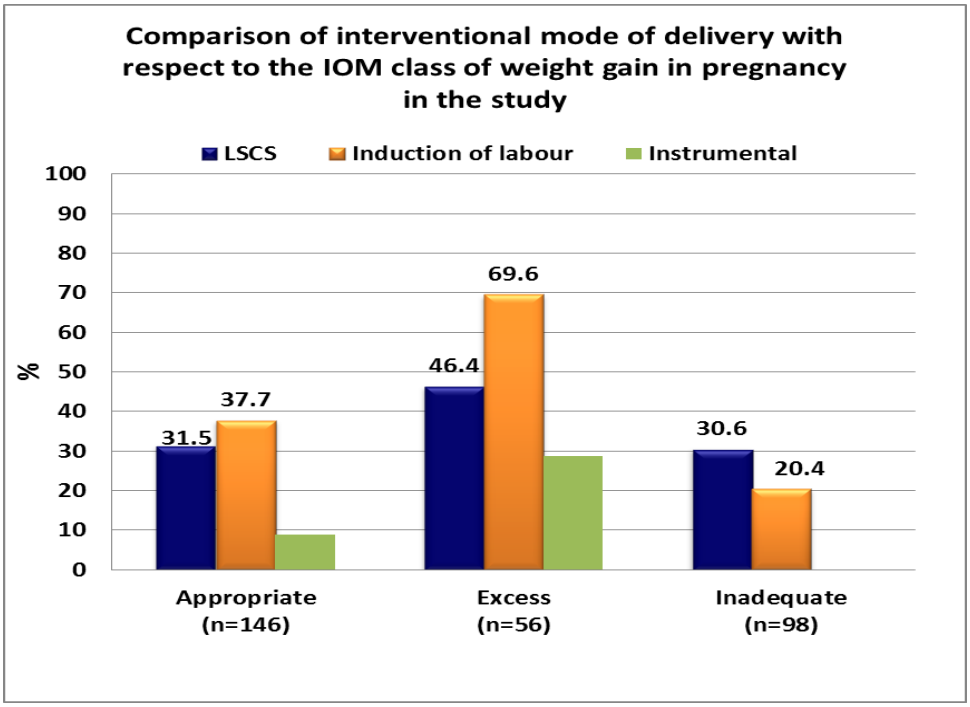


Table 28.Comparison of interventional mode of delivery with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Intervention	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	N	%		
1	LSCS	46	31.5	26	46.4	30	30.6	46.4	<0.001*
2	Induction of labour	55	37.7	39	69.6	20	20.4	36.7	<0.001*
3	Instrumental	13	8.9	16	28.6	0	0	46.4	<0.001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

Figure 22:



Interventional deliveries were highest among women who gained excessive weight gain.There was not much interventional mode of delivery among women with inadequate weight gain.

Table 29.Frequency distribution of Occurrence of comorbidity observed in the study.

S.No	Comorbidity	N	%
1	Anemia	136	45.3
2	GHT	82	27.3
3	GDM	70	23.3

4	Abruption	13	4.3
5	Oligohydraminos	77	25.7
6	Polyhydraminos	66	22
7	FGR	106	35.6
8	CVT/DVT	27	9
9	PPH	70	23.3

Data are expressed as n with %. The total N=300

- Majority of the patients included in the study were found to be anemic .
- Maternal co-morbidities like GHT ,GDM ,PPH were on increasing scales.
- Fetal co-morbiities including FGR were observed the most in the study.

Figure 23:

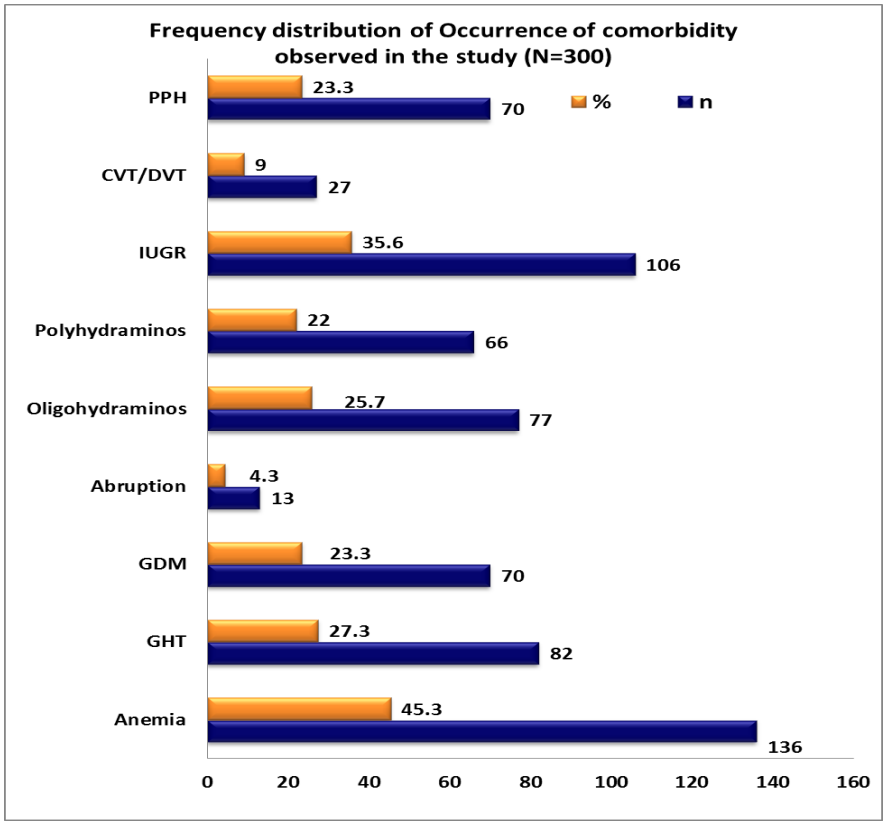


Table 30.Comparison of comorbidity with respect to the BMI class in the study.

S.No	Intervention	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	n	%	n	%	n	%		
1	Anemia	48	53.3	71	59.2	9	15	8	26.7	38.1	<0.001*
2	GHT	10	11.1	15	12.5	33	55	24	80	90.3	<0.001*
3	GDM	2	2.2	6	5	40	66.7	22	73.3	149	<0.001*
4	Abruption	4	4.4	3	2.5	4	6.7	2	6.7	2.15	0.541 (NS)
5	Oligohydraminos	35	38.9	32	26.7	4	6.7	6	20	20.1	<0.001*
6	Polyhydraminos	4	4.4	11	9.2	36	60	15	50	91.8	<0.001*
7	FGR	38	42.2	58	48.3	4	6.7	6	20	35.4	<0.001*
8	CVT/DVT	3	3.3	1	0.8	10	16.7	13	43.3	60.7	<0.001*
9	PPH	11	12.2	11	9.2	30	50	18	60	66.07	<0.001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant. NS = Not significant

- In the study, pregnant women with pre pregnancy BMI as normal(59.2%) and underweight(53.3%) were significantly anemic compared with overweight and obsese category.

- Whereas maternal co-morbities like GHT, GDM, PPH, CVT/DVT, Polyhydramnios of pregnancy were were significantly observed among pregnant women with prepregnancy BMI obese and overweight.

- Complications including Oligohydramnios and FGR babies were on the higher side for women with underweight prepregnancy BMI pregnant women.

Table 31.Comparison of comorbidity with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Comorbidity	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		N	%	N	%	N	%		
1	Anemia	45	69.2	13	23.2	78	79.6	69.8	<0.001*
2	GHT	33	22.6	39	69.6	10	10.2	66.5	<0.001*
3	GDM	31	21.2	39	69.6	0	0	97.3	<0.001*
4	Abruption	5	3.4	3	5.4	5	5.1	0.572	0.751 (NS)
5	Oligohydraminos	28	19.2	7	12.5	42	42.9	23.4	<0.001*
6	Polyhydraminos	30	20.5	36	64.3	0	0	86.1	<0.001*

7	FGR	33	22.6	6	10.7	67	68.4	72.01	<0.001*
8	CVT/DVT	4	2.7	22	39.3	1	1	77.3	<0.001*
9	PPH	31	21.2	34	60.7	5	5.1	62.3	<0.001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant. NS = Not significant

- Pregnant women with inadequate weight gain were significantly anemic upto 79.6 % and had maximum of oligohydramnios 42.9 % and FGR babies 68.4% compared to those who gained appropriate weight gain.

- Whereas maternal co-morbidities like GHT(69.6%) ,GDM(69.6%) , CVT/DVT(39.3%), PPH(60.7%) and polyhydramnios(64.3%) of pregnancy were more among those who gained excessive weight gain compared with those who gained inadequately and appropriately.

- In this study there was not much significance of occurrence of abruption with gestational weight gain.

Table 32.Comparison of delayed wound healing with respect to the BMI class in the study.

S. No	Parameter	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	n	%	n	%	n	%		
1	Delayed wound healing	9	10	7	5.8	16	26.7	19	63.3	63.4	<0.0001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

- Delayed wound healing was seen in 63.3% of obese individuals ,which is highly significant.
- Only 10% of underweight women and 5.8% of normal BMI women had delayed wound healing.

Table 33.Comparison of delayed wound healing with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Parameter	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	N	%	n	%		
1	Delayed wound healing	12	8.2	36	64.3	3	3.1	110.2	<0.0001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

- Women who gained excessive weight gain had 64.3% incidence of delayed wound healing .

Delayed Wound healing was only seen in 3.1% among inadequate weight gain and 8.2% among appropriate weight gain.

Table 34. Frequency distribution of neonatal outcome observed in the study.

S.No	Parameter	N	%
1	Outcome		
	Alive	291	97
	Still birth	9	3
2	Newborn weight		
	Appropriate for age	172	57.3
	Low birth weight	102	34
	Macrosomia	26	8.7
3	NICU admission		
	No	168	56
	Yes	132	44

Data are expressed as n with %. The total N=300

- This table shows that out of 300 deliveries 291 babies were born alive (97%) and 9 babies were still born(3%)
- Out of these 291 babies 132 seeked NICU admission(44%) .
- Most of the babies born in this study cohort were appropriate for age (57.3%)

Figure 24:

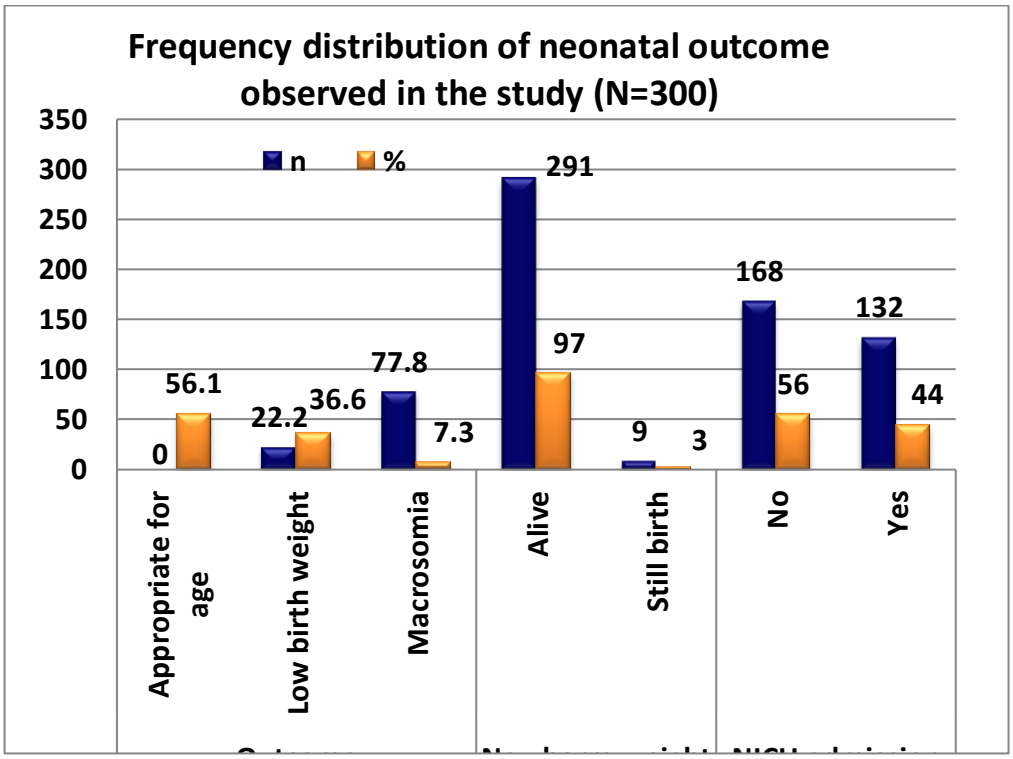


Table 35. Frequency distribution of reason for NICU admission observed in the study.

S.No	Reason for NICU admission	N	%
1	IDM	34	25.8
2	Observation	37	28.0
3	RDS	56	42.4
4	LBW	98	74.2
5	Early preterm	1	0.8
6	Late preterm	58	43.9
7	Birth asphyxia	13	9.8
8	MSAF	5	3.8

9	PROM	1	0.8
10	PPROM	3	2.3
11	FGR	2	1.5
12	LGA	31	23.5

Data are expressed as n with %. The total N=132

Out of the 132 babies who needed NICU care were mostly low birth weight infants (LBW)

- approximately 74% and late preterm infants 58% and those with respiratory distress(RDS) approximately 42%.

Table 36. Comparison of neonatal outcome with respect to the BMI class in the study.

S.No	Parameter	Under weight (n=90)		Normal (n=120)		Over weight (n=60)		Obese (n=30)		Chi square value	P value
		n	%	N	%	n	%	n	%		
1	Outcome alive	88	97	118	98.3	60	100	25	83.3	12.13	<0.001*
2	Outcome dead	2	3	2	1.7	0	0	5	16.7		
3	AGA	56	62.2	60	50	40	66.7	16	53.3	84.7	<0.001*

4	LBW	34	37.8	59	49.2	6	10	3	10		
5	Macrosomia	0	0	1	0.8	14	23.3	11	36.7		
6	NICU admission	31	34.4	62	51.7	21	35	18	60	11.2	0.012*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant. NS = Not significant

- The incidence of stillbirth is significant in the obese group.
- Macrosomic babies were born mostly to mothers with increased prepregnancy BMI i.e. overweight and obese mothers.
- LBW infants were seen mostly with normal prepregnancy BMI (49.2%)mothers followed by underweight mothers(37.8%).
- Majority of the newborns born to obese (60%) mothers were admitted in NICU .

Figure 25:

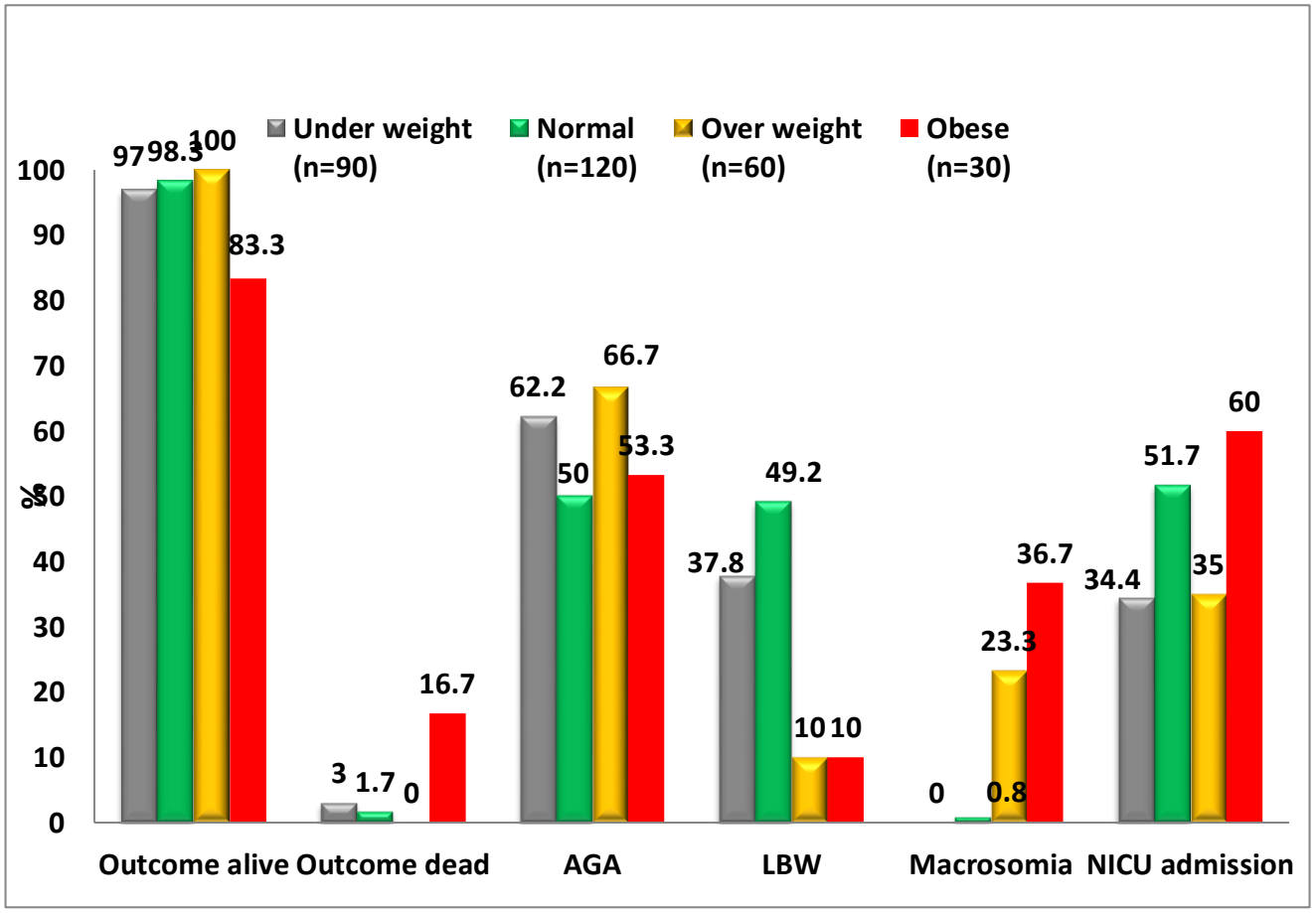


Table 37.Comparison of neonatal outcome with respect to the IOM class of weight gain in pregnancy in the study.

S.No	Parameter	Appropriate (n=146)		Excess (n=56)		Inadequate (n=98)		Chi square value	P value
		n	%	n	%	N	%		
1	Outcome alive	146	100	51	91	94	96	16.4	<0.001*
2	Outcome dead	0	0	5	9	4	4		
3	AGA	114	78.1	30	53.6	28	28.6	153.1	<0.001*

4	LBW	27	18.5	5	8.9	70	71.4		
5	Macrosomia	5	3.4	21	37.5	0	0		
6	NICU admission	35	24	29	51.8	68	69.4	50.7	<0.001*

Data are expressed as n with %.. Fisher's exact test was used to compare the frequency between the groups. *indicates $p<0.05$ and considered statistically significant.

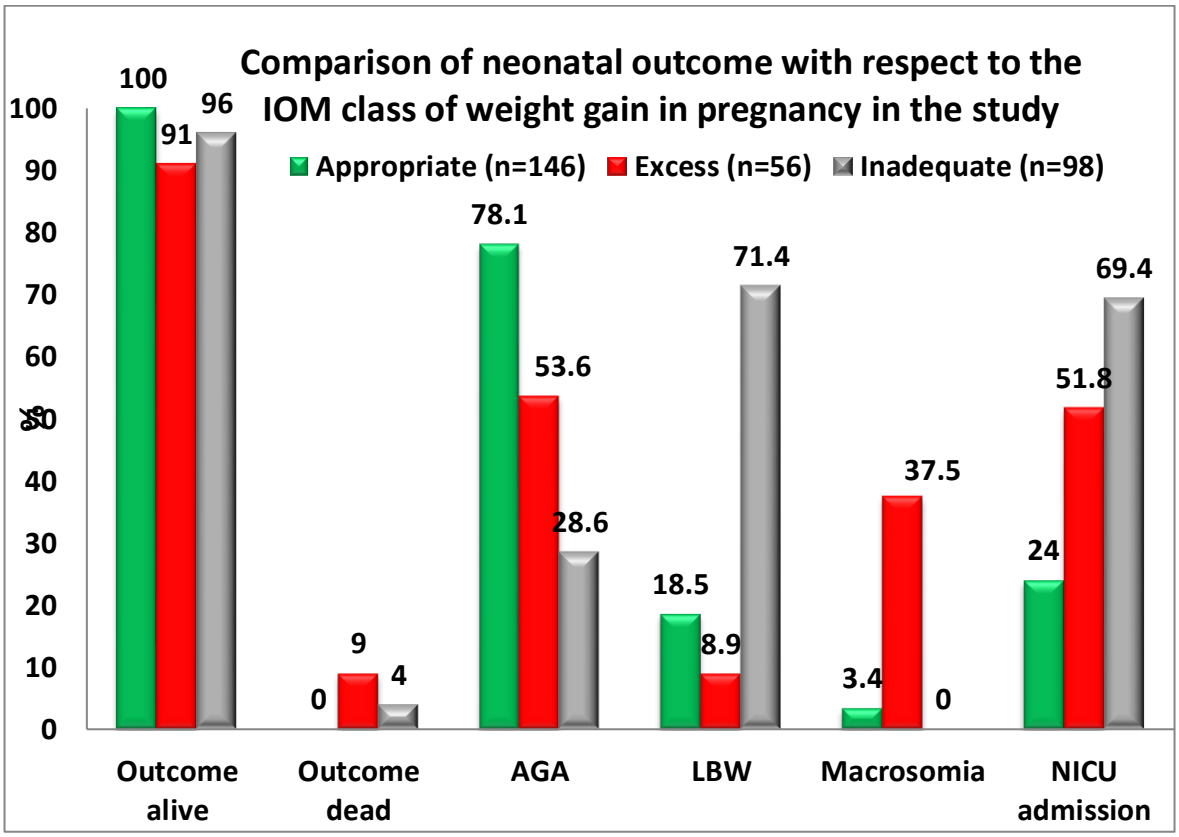
- From the table we come to an inference that stillbirth or dead born were significantly increased among pregnant women with excessive weight gain compared to those who gained appropriately.

- Macrosomia was seen more among women with excessive gestational weight gain than compared to those with appropriate weight gain.

- Babies born to mothers with inadequate gestational weight gain were mostly of LBW with 71.4% .

- On the whole ,NICU admissions were more among newborns that were born to mothers who had inadequate gestational weight gain(69.4%) and excessive weight gain(51.8%)compared to those mothers who had adequate weight gain.

Figure 26:


Comparison of neonatal outcome with respect to the IOM class of weight gain in pregnancy in the study
Appropriate (n=146)
Excess (n=56)
Inadequate (n=98)
%
100
90
80
70
60
50
40
30
20
10
0
100
91
96
0
9
4
78.1
53.6
28.6
18.5
8.9
71.4
3.4
37.5
0
24
51.8
69.4
Outcome alive
Outcome dead
AGA
LBW
Macrosomia
NICU admission

DISCUSSION

A Prospective observational study comprising 300 antenatal women with singleton uncomplicated pregnancies, booked at Government Raja Mirasudhar Hospital, Thanjavur within the first 12 weeks of gestation and delivering at term has been conducted during the period of January 2019 to December 2019 .

The age of the subjects in the present study were in the range of 20– 34 years. The mean age of the subjects was 23 years.

In the study, 63% of the study population were nullipara and 37% were multipara.

44.3 % of the subjects delivered at term (gestational age 37- 40 weeks) and 34 % of the subjects delivered preterm (gestational age less than 37 weeks) and 21.7% of the subjects delivered postdated (gestational age more than 40 weeks).

It was seen that 48.7% of the subjects showed a weight gain appropriate with respect to their BMI as per IOM guidelines. 18.7% of the subjects showed inadequate weight gain during pregnancy. 32.6 % of the women showed excessive weight gain.

The study comprised women, who were divided into four BMI groups based on their early pregnancy BMI. The BMI at presentation of <18.5 kg/m2 was seen in 30% of the population.BMI between 18.5 - 24.9 kg/m2 was seen in 40% of the women. BMI between 25.0 - 29.9 kg/m2 was seen in 20 % of the women. BMI of 30kg/m2 and above was seen in 10 % of the women.

Majority of the women (40 %) in the study population belonged to the normal BMI group and the overweight and obese group together comprised as large as 30 %. This is of significance as with changing lifestyle, obesity is increasing rapidly especially in urban set ups and may become a major health problem in the future.

GESTATIONAL DIABETES MELLITUS:

In the present study, 5 % of the patients in the Normal BMI group developed GDM. Maximum number of patients in the obese group developed GDM (73.3%) and minimum number of patients in the underweight group developed GDM (2 %).

Association between Diabetes Mellitus and BMI was performed using Chi-Square analysis and it has been shown that Gestational Diabetes Mellitus is significantly associated with increasing BMI ($p< 0.01$). A study conducted by D.A. Dohertya on 331 women found that 188 women were obese (6.6%) and that obese women were more likely to develop gestational diabetes ($p<0.001$).[40] Upon calculating the relative risk it was seen that maximum risk of acquiring Diabetes is seen in obese women (15 fold) compared with normal BMI women , overweight women were associated with a 13 fold increase and underweight women showing no increased risk of acquiring Diabetes.

With respect to Gestational weight gain (GWG), compared with appropriate GWG, women with excessive weight gain in pregnancy had maximum percentage of GDM (69.6%) and none among women with inadequate weight

gain had GDM.Association between Diabetes Mellitus and GWG in pregnancy was performed using Chi-Square analysis and it has been shown that Gestational Diabetes Mellitus is significantly associated with excessive GWG ($p< 0.01$). Upon calculating the relative risk it was seen that maximum risk of acquiring Diabetes is seen in obese women (3 fold) compared to women with appropriate GWG and inadequate GWG women showing no increased risk of acquiring Gestational Diabetes Mellitus.

	STUDY	PERCENTAGE IN OBESE
GESTATIONAL DIABETES	Our study Gross et al (1980)42 Ehren Berg et al 200226 Glady et al 200536 Glady et al 200536	73.3% 6.5% 8% 8% 14% (Asians)

GESTATIONAL HYPERTENSION:

In the current study, it was seen that 12.5 % of the subjects belonging to the Normal BMI group developed GHT. 10 % in the underweight group developed GHT. 55 % in the overweight BMI group developed GHT and 80% in the obese group were seen to develop GHT.

Maximum number of obese patients (80%) developed GHT, compared to women of normal BMI(12.5%).Analysis was done using Chi-square, which showed a strong association between increasing BMI and GHT.($p < 0.01$) A similar study

done by Meenakshi, Srivastava Reena (FOGSI) showed that obese women were associated with adverse outcomes like GHT with a $p < 0.05$.

Upon calculating the relative risk, maximum risk of acquiring GHT is seen in obese women (6 fold) compared with normal BMI. Overweight women are at a 4 fold increased risk of GHT compared with normal BMI women. In the study, not much risk was noted with respect to underweight in developing GHT.

With respect to Gestational weight gain (GWG), compared with appropriate GWG ,women with excessive weight gain in pregnancy had maximum percentage of GHT (69.6%) and only 10.2% among women with inadequate weight gain had GHT.Association between GHT and GWG in pregnancy was performed using Chi-Square analysis and it has been shown that Gestational Hypertension is significantly associated with excessive GWG ($p< 0.01$).

Upon calculating the relative risk it was seen that maximum risk of acquiring GHT is seen in excessive GWG women (3 fold) compared to women with appropriate GWG. Inadequate GWG women shows no increased risk of acquiring GHT.

	STUDY	PERCENTAGE IN OBESE
GESTATIONAL HYPERTENSION	Our study	80%
	Joshua .L. Weiss et al 200453	57%

ANAEMIA:

It was observed that, 59.2 % with Normal BMI developed anemia. Thus anemia was maximum among normal BMI also in the rural population, and this shall be attributed to inadequate weight gain in the pregnancy period, low socio economic class and low nutritional quality of food . 53.3 % of the underweight patients developed anemia.15% of overweight women and 26.7% obese women developed anemia. Apart from anemia in normal BMI (59.2%) ,maximum percentage of Underweight women (53.3%)developed Anemia, followed by overweight women (15%).

Analysis was done using Chi square, which showed a significant association between low BMI and anemia in my study ($p< 0.05$). A study done by Adam I,[41] 46 on 1136 showed that 26.5% of the underweight women developed anemia with a significant association between anemia and low BMI being $p < 0.05$. Importance has to be given, because ours is a developing country, and the incidence of Anemia is high especially in the rural areas.

With respect to Gestational weight gain (GWG), compared with appropriate GWG,

women with inadequate weight gain in pregnancy had maximum percentage of anemia (79.6%) and only 23.2% among women with excessive weight gain had anemia.Association between anemia and GWG in pregnancy was performed using Chi-Square analysis and it has been shown that anemia is significantly associated with inadequate GWG ($p< 0.01$).

Upon calculating the relative risk it was seen that maximum risk of acquiring anemia is seen in inadequate GWG women (one fold) compared to women with appropriate GWG and excess GWG womenshowing no increased risk of acquiring anemia.

POLYHYDRAMNIOS:

In the current study, 60 % of the overweight patients had Polyhydraminos, whereas 50 % of the obese patients had polyhydraminos when compared to patients belonging to the normal BMI group(9.2 %). In the underweight category, only 4.4% of the patients had polyhydraminos, but maximum number of patients in the underweight group had oligohydraminos(38.9 %) when compared with the normal BMI category (26.7 %) .It was seen that maximum number of patients with increased BMI had polyhydraminos, whereas maximum number of underweight patients had oligohydraminos.

Using Chi - square test, it was found that there was an association between BMI and liquor volume.($p < 0.05$).Upon calculating the relative risk, it was found that Obese women had an five fold increased risk of polyhydraminos compared with normal BMI while Overweight women were found to have a six fold increased risk of polyhydraminos compared with normal BMI. Not much patients in the Underweight group had polyhydraminos so the relative risk could not be calculated.

It was observed that maximum risk of oligohydraminos was seen in the Underweight group (1.5 fold) compared with normal BMI.

With respect to Gestational weight gain (GWG),compared with appropriate GWG, women with excessive weight gain in pregnancy had maximum percentage of polyhydramnios(64.3%) and none among women with inadequate weight gain had polyhydramnios.Association between polyhydramnios and GWG in pregnancy was performed using Chi-Square analysis and it has been shown that it is significantly associated with excessive GWG ($p< 0.01$). Upon calculating the relative risk it was seen that maximum risk of polyhydramnios is seen in excessive GWG women (3 fold) compared to women with appropriate GWG(20.5%) and inadequate GWG women showing no increased risk of acquiring polyhydramnios.

MODE OF DELIVERY:

In the study, it was seen that subjects in the normal(76.7%) and underweight(61.1%) groups had a higher percentage of uncomplicated vaginal deliveries when compared to the overweight(28.3%) and obese(16.7%) groups. In my study it can be seen that a much higher percentage of obese group (53.3%) subjects have had C-section either in the form of emergency or elective when compared to normal group individuals. Women of the underweight category also had an increased percentage (38.9%) of C-section when compared with the normal BMI.

Using Chi square analysis, it was found that there was a strong association between BMI and Cesarean section.($p<0.05$).) A study done on 215 women by Meenakshi, Srivastava Reena(FOGSI) showed that 79 out of 170 obese women

were associated with Cesarean sections with a $p < 0.01$.In my study all the Obese groups are at a two fold increased risk

of undergoing LSCS and both overweight and underweight groups was at 1.5 fold increased risk of undergoing LSCS when compared to women with normal BMI.

In the obese group, our results supported a number of previous studies (Joshua-L-Weiss et al 2001[43] and Marie -I - Cedergren 2004[44]) that have demonstrated an

increased risk for cesarean delivery in this group.

With respect to Gestational weight gain (GWG), there was not much significance of each group with LSCS in my study.

In my study it can be seen that a much higher percentage of overweight group (33.3%) , obese subjects(30%) had instrumental delivery when compared to normal group individuals. There was also a much high percentage of instrumental delivery among those who had excessive weight gain (28.6%) ,with a 3 fold increase in relative risk compared to appropriate GWG women(8.9%).

INDUCTION OF LABOUR:

In my study it can be seen that a much higher percentage of obese group (60%) , overweight subjects(58.3%) and underweight subjects (43.3%) have had labour induction when compared to normal group individuals(18.3%). There was a 3 fold increase in relative risk of labour induction among obese and overweight

individuals and 2 fold increase in risk among underweight BMI comapared with normal BMI.

The most common reason for induction was GHT and GDM among obese and overweight groups. Among underweight category oligohydramnios and FGR were the most common reasons for induction of labour.

With respect to Gestational weight gain (GWG), compared with appropriate GWG , women with excessive weight gain in pregnancy had higher percentage of labour induction(69.6%) Association between labour induction and GWG in pregnancy was performed using Chi-Square analysis and it has been shown that it is significantly associated with excessive GWG ($p< 0.01$).

Upon calculating the relative risk it was seen that maximum risk of labour induction is seen in excessive GWG women (2 fold) compared to women with appropriate GWG(37.7%).

POSTPARTUM HEMORRHAGE:

In my study, it was found that obese (60%) and overweight women(50%) had a higher percentage of Post partum hemorrhage(PPH) when compared to the normal(9.2%) and underweight groups(12.2%).

Using Chi square analysis, it was found that there was a strong association between BMI and PPH.($p<0.05$). A study done by Meenakshi, Srivastava Reena(FOGSI) showed that only 5 out of 170 obese and overweight women

developed PPH with a $p > 0.05$,showing no significant association between PPH and increasing BMI, while our study showed a significant association between BMI and PPH. ($P < 0.05$)

In the present study, the maximum number of obese patients have an increased risk of PPH(6.5 fold) when compared to women with normal BMI. Overweight women have a five fold increase in the risk of PPH, whereas underweight women do not have much increased risk of PPH when compared to women with normal BMI. PPH was more among women with excessive GWG (60.7%) with a 2 fold increase in relative risk compares with appropriate GWG women (31%). There was no significane of PPH with inadequate GWG women.Using Chi square analysis, it was found that there was a strong association between excessive GWG and PPH.($p<0.05$).

DELAYED WOUND HEALING:

The percentage of delayed wound healing was much higher in the obese groups (63.3%) and in those who had excessive weight gain (64.3%) women when compared with the other groups. In the current study, obese women have a 12 fold increase in risk of delayed wound healing when compared to normal BMI women, whereas overweight and underweight women do not have a risk of delayed wound healing.

Using Chi square test, it was found that there was an association between BMI and delayed wound healing. A study done on 215 women by Meenakshi,

Srivastava Reena(FOGSI) 1 showed that 25 out of 170 obese and overweight women were associated with delayed wound healing with a $p < 0.05$.

THROMBOEMBOLIC EVENTS:

Thrombo embolism as a complication occurred only in the obese BMI group (43.3%).Using Chi square analysis, it was seen that there was a strong association between BMI and thrombo embolism. ($X^2 = 13.937$, $p < 0.005$). From the p value, it can be seen that there is a significant association between BMI and thrombo embolism.

In the current study, obese (43.3%) women have a 53 fold increase in risk of developing thrombo embolism and overweight(16.7%) women had 20 fold increase, when compared to normal BMI(0.8%).

CVT/DVT was more among women with excessive GWG (39.3%) with a 14 fold increase in relative risk compares with appropriate GWG women(2.7%). There was no significane of CVT/DVT with inadequate GWG women. Using Chi square analysis, it was found that there was a strong association between excessive GWG and CVT/DVT ($p<0.05$).

BIRTH WEIGHT:

In the present study, it was seen that maximum percentage of LGA babies were born to obese women (36.7%) compared to women with normal BMI. Likewise maximum percentage of SGA babies were born to women in the normal (49.2%) and underweight group(37.8%) when compared to other groups. Results showed that there was a significant association between lower BMI and low birth weight,

obesity and LGA babies (p<0.001). Similar findings were noted in a study by Ihunnaya O Frederick et al and J.E.Brown et al with p<0.001 and p value 0.0009 respectively.

LGA babies were more among women with excessive GWG (37.5%) with a 11 fold increase in relative risk compared with appropriate GWG women (3.4%) in my study. There was no significance of LGA babies with inadequate GWG women(0).Using Chi square analysis, it was found that there was a strong association between excessive GWG and LGA babies(p<0.05).

Comparing our study with other study with respect to macrosomia :

Study	Underweight	Normal	Overweight	Obese
A Meher-un- nisa et nal[45] d	Not included in the study	0.96	4.5	7
Our study	0	0.8	23.3	36.7

also SGA babies were more among women with inadequate GWG (71.4%) with a 4 fold increase in relative risk compared with appropriate GWG women(18.5%).Using Chi square analysis, it was found that there was a strong association between excessive GWG and SGA babies(p<0.05)

In my study, women in the underweight group had the maximum increase in risk (2 fold) of delivering a SGA baby when compared to overweight and obese women but not with normal BMI women. The risk of delivering LGA babies among obese patients was forty five fold (45fold) , whereas overweight patients

had 29 fold increase in risk of delivering LGA babies when compared to the normal BMI.

NICU ADMISSION:

Infants born to women with inadequate weight gain had higher percentage of NICU admission rates (69.4%) and those to excess GWG had 51.8 % NICU admission rates compared to appropriate GWG (24%).Still births were on higher side for infants born to women with excessive GWG(9%) and inadequate GWG (4%) compared to women with normal GWG(0).Using Chi square analysis, it was found that there was a strong association between inadequate and excessive GWG on NICU admission and still birth.($p<0.05$)

The most common reason for NICU admission in my study was preterm (44.9%) , LBW (74.2%) and respiratory distress of newborn (42.5%).

There was not much significance with NICU admission with respect to BMI in my study.

Maternal nutritional status plays a vital role for the health and quality of life of a pregnant mother and her baby. Early pregnancy BMI and weight gain during pregnancy are important predictors of adverse pregnancy outcomes. In my study, the association between extremes of early pregnancy BMI and adverse maternal and fetal outcomes have been analyzed. It was seen that there was a strong association between BMI and adverse maternal outcomes with a p value <0.05. It was seen that overweight and obese women had a much higher risk of developing adverse maternal outcomes like gestational diabetes, pregnancy

induced hypertension, increased liquor volume, increased rate of instrumental deliveries and cesarean sections, postpartum complications like post partum hemorrhage delayed wound healing and delivering LGA babies. On the other hand, Underweight women were seen to develop anaemia, reduced liquor volume, increased rate of cesarean sections and deliver SGA babies.Weight gain during pregnancy in relation to BMI was also analyzed. It was seen that overweight and obese women gained more weight than women with normal BMI, and least weight was gained by underweight women. My study tried to find an association between early pregnancy BMI and weight gain in relation to various pregnancy outcomes, which was justified. The relative risk of various pregnancy outcomes that a patient with high and low BMI can develop has also been evaluated in my study and the results were justified.

CONCLUSION

Early pregnancy BMI and gestational weight gain have a strong effect on adverse maternal and neonatal outcomes, which is supported by a huge body of literature. In the present study, it was seen that there was a strong association between BMI and GWG on adverse maternal and fetal outcomes .Underweight women were seen to develop anemia, reduced liquor volume and increased rate of cesarean sections. Women with inadequate pregnancy GWG also had adverse outcomes like anemia, oligohydramnios, FGR babies , SGA babies,increased NICU admissions.It was seen that overweight and obese women and women who gained excessive weight during pregnancy, had a much higher risk of developing adverse maternal outcomes like gestational diabetes, pregnancy induced hypertension, increased liquor volume, increased rate of instrumental deliveries and cesarean sections, postpartum complications like post partum haemorrhage ,delayed wound healing and delivering LGA babies. Still births were increased among infants born to excessive weight gain mothers and NICU admission was increased in infants born to inappropriate GWG . It was observed that overweight and obese women gained more weight than women with normal BMI, and least weight was gained by underweight women. The relative risk of various pregnancy and fetal outcomes that a patient with high or low BMI and inappropriate or exceesive GWG women can develop was also evaluated and the results were justified.

Utmost importance needs to be given to BMI and the patterns of weight gain during pregnancy, as they are modifiable risk factors of adverse pregnancy outcomes. One should have basic knowledge and awareness regarding the symptoms and signs of adverse pregnancy outcomes. A better understanding of the complex interrelations between the mother and fetus has led to a vast improvement on antenatal recommendations.

Many studies have been done in the Western countries whereas only few studies have been done on the Asian population. In India, previously the problems during pregnancy were more related to low BMI but with changing lifestyle, obesity is increasing rapidly especially in urban set ups and may become a major health problem in the future. By performing this study it was possible to evaluate the association between BMI and GWG on adverse effect on pregnancy and fetal outcome. The relative risk of various pregnancy outcomes that a patient with extremes of BMI and GWG can develop was evaluated by doing this study and the results justified. It was also possible to analyze the association between BMI and gestational weight gain in our Indian set up, the results of all of which are significant.

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GLOSSARY

BMI: Body mass index

GWG: Gestational weight gain

IOM: Institute of Medicine

PPROM: Preterm Premature rupture of membranes

FGR: Fetal Growth Retardation

LSCS: Lower segment caesarean section

MUAC: Mean upper arm muscle circumference

BMR: Basal metabolic rate

LDL: Low density lipoprotein

GA: Gestational age

SGA: Small for gestational age

LGA: Large for gestational age

WHO: World health organization

PPH: Postpartum hemorrhage

GHT: Gestational Hypertension

GDM: GestationalDiabetes Mellitus

CVT: Cortical vein thrombosis

DVT: Deep vein thrombosis

NICU: Neonatal intensive care unit

PROFORMA

NAME:

AGE:

IPno/OPno:

HUSBAND NAME:

PHONE NO:

PARITY:

SOCIOECONOMIC CLASS:

HEIGHT:

PREGESTATIONAL WEIGHT:

BMI:

UNDERWEIGHT/NORMAL/OVERWEIGHT/OBESE:

WEIGHT AT DELIVERY:

GWG WHETHER INADEQUATE/ADEQUATE/EXCESS:

GESTATIONAL WEEKS AT DELIVERY:

MODE OF DELIVERY:LABOUR

NATURAL/INSTRUMENTAL/CESAREAN SECTION

INDUCED OR NOT:

REASON FOR INDUCTION:

INDUCTION SUCCESFUL/FAILED:

MATERNAL MORBIDITIES : GHT/GDM/ANEMIA

LIQUOR ABNORMALITIES: OLIGOHYDRAMNIOS/POLYHYDRAMNIOS

FGR:

ABRUPTION OF PLACENTA:

DELAYED WOUND HEALING:

THROMBOEMBOLIC EVENTS:CVT/DVT

ALIVE BABY/STILL BORN:

WEIGHT OF BABY:

LBW/APPROPRIATE WEIGHT/MACROSOMIA:

NICU ADMISSION:

REASON FOR ADMISSION:

S.NO	AGE	AGE GROUP (A- 20-24; B-25-30; C> 30)	SOCIO ECONOMIC CLASS	PARITY	PREPREGNANCY BMI	WHO BMI CLASS (1- under weight, 2- normal weight, 3- over weight, 4- obese)	GESTATIONAL WEIGHT GAIN	IOM CLASS OF GWG (I- Inadequate, A- adequate, E-excess)	GESTATIONAL WEEKS AT DELIVERY	MODE OF DELIERY	LABOUR INDUCTION	REASON FOR INDUCTION	INDUCTION	ANEMIA	GHT	GDM	ABRUPTION	OLIHYDRAMNIOS	POLYHYDRAMNIOS	IUGR	CVT/DVT	DELAYED WOUND HEALING AND INFECTION	PPH	NEONATL OUTCOME (A- alive, S-still birth)	NEONATAL BIRTH WEIGHT (A- normal wt, L-LBW, M-macrosomia)	NICU ADMISSION	REASON FOR NICU ADMISSION
1	21	A	IV	1	17.5	1	15	A	T	LN	Y	OLI	S	N	N	N	N	Y	N	N	N	N	N	A	A	N	-
2	23	A	III	1	18	1	16	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
3	26	B	III	2	17	1	17	A	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
4	24	A	IV	1	16.5	1	18	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	Y	A	A	N	-
5	21	A	IV	1	17	1	11	I	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/RDS
6	26	B	III	1	16	1	12	I	Pr	LSCS	Y	OLI/IUGR/PPROM	F	N	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/RDS

7	23	A	IV	1	17.5	1	11	I	Pr	LSCS	Y	OLI	F	Y	N	N	N	Y	N	N	Y	N	N	A	L	Y	LBW/RDS
8	20	A	V	1	17.1	1	10.5	I	Pr	LSCS	N		-	Y	N	N	Y	Y	N	Y	N	N	N	S	L	N	-
9	27	B	II	1	16.5	1	21	E	T	LN	Y	OLI	S	N	N	N	N	Y	N	N	N	N	N	A	A	N	-
10	23	A	II	1	17.5	1	20	E	T	LN	N	-	-	N	N	Y	N	N	Y	N	N	N	Y	A	A	N	-
11	24	A	III	2	16.4	1	21	E	T	LSCS	N	-	-	N	Y	N	N	N	N	N	N	N	N	A	A	N	-
12	23	A	II	1	17.1	1	19	E	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
13	24	A	II	2	17	1	19	E	Po	LN	Y	PD/GHT	S	Y	Y	N	N	N	N	N	N	Y	N	A	A	N	-
14	23	A	II	2	17.5	1	20	E	T	LN	Y	TERM/GDM	S	Y	N	Y	N	N	N	N	N	N	N	A	A	N	-
15	21	A	II	1	18	1	19.5	E	Po	LN	Y	PD	S	Y	N	N	N	N	N	N	N	Y	Y	A	A	N	-
16	20	A	II	2	17.6	1	20.5	E	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
17	24	A	III	1	18	1	20	E	T	LSCS	N	-	-	N	N	N	N	N	Y	N	N	Y	N	A	A	N	-
18	22	A	IV	1	17	1	12	I	Pr	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/BA

19	23	A	V	1	16	1	11	I	Pr	LSCS	Y	OLI/IUGR	F	Y	Y	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/BA
20	23	A	V	1	16.5	1	11.5	I	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PND
21	21	A	IV	1	17.5	1	11.3	I	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	
22	24	A	V	1	16.5	1	11.2	I	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/RDS
23	20	A	IV	2	18	1	11	I	Pr	LN	Y	OLI/PPROM	S	N	N	N	N	Y	N	N	N	N	N	A	L	Y	LBW/BA
24	20	A	V	1	17.3	1	12.1	I	Pr	LSCS	Y	OLI/IUGR	F	Y	Y	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/RDS
25	23	A	IV	1	17.2	1	11.4	I	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/BA
26	21	A	IV	1	17.6	1	11.8	I	Pr	LN	Y	OLI	S	N	N	N	N	Y	N	N	N	N	N	A	L	Y	LBW/OBS
27	20	A	V	1	17.4	1	11.9	I	Pr	LSCS	Y	PPROM	F	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/OBS
28	21	A	IV	2	18.1	1	11.4	I	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PND
29	20	A	V	1	18.2	1	10	I	Pr	LSC	N	-	-	Y	Y	N	Y	Y	N	Y	N	N	N	S	A	N	-

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30	24	A	IV	2	17.8	1	10.5	I	Pr	LN	Y	PPROM	S	Y	N	N	N	Y	N	Y	N	N	Y	A	L	Y	LBW/OBS
31	20	A	V	1	17.3	1	10.6	I	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	
32	23	A	IV	2	18	1	16.1	A	Pr	LSCS	Y	OLI/IUGR	F	N	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/BA
33	21	A	III	1	16.2	1	15.7	A	Pr	LN	Y	OLI/IUGR	S	N	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/BA
34	20	A	IV	1	17.1	1	14.7	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
35	23	A	IV	2	18.1	1	14.8	A	T	LN	N	-	-	N	N	N	N	N	Y	N	N	N	N	A	A	N	-
36	24	A	IV	1	16.2	1	14.7	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
37	20	A	III	1	16.4	1	14.8	A	T	LN	Y	TERM/IUGR	S	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
38	23	A	IV	1	16.9	1	14.6	A	T	LSCS	Y	TERM/RH-VE	F	N	N	N	N	N	N	N	N	N	N	A	A	N	-
39	24	A	I	1	16.5	1	14.2	A	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	Y	A	A	N	-

40	21	A	IV	1	17.2	1	14.5	A	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	-
41	23	A	IV	1	17.2	1	13.9	A	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	-
42	24	A	IV	1	15.9	1	12.7	A	Pr	LSCS	Y	OLI/IUGR	S	Y	Y	N	N	Y	N	Y	Y	N	N	A	L	Y	LBW/PND
43	21	A	IV	1	16	1	15.9	A	Po	LSCS	Y	TERM/PD	F	Y	N	N	N	N	N	N	N	Y	N	A	A	N	-
44	21	A	IV	1	16.1	1	15.4	A	Pr	LSCS	N	-	-	Y	N	N	Y	Y	N	Y	N	N	N	A	A	N	-
45	23	A	IV	1	16.9	1	16.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	Y	N	A	A	N	-
46	20	A	III	1	16.8	1	17.1	A	T	LSCS	N	-	-	N	Y	N	N	N	N	N	N	N	N	A	A	N	-
47	23	A	IV	1	16.3	1	14	A	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
48	21	A	IV	1	16.2	1	14.6	A	Pr	LSCS	Y	OLI/IUGR	F	Y	Y	N	N	Y	N	Y	N	N	N	A	L	N	-
49	20	A	IV	1	16.7	1	12.4	A	T	LSCS	N	-	-	Y	N	N	Y	N	N	N	N	N	N	A	A	N	-
50	24	A	IV	1	16.4	1	18	A	T	LSC	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-

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51	20	A	III	1	17.1	1	17.2	A	Pr	LSCS	N	PPROM	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/OBS
52	23	A	III	1	17.3	1	13.4	A	T	LN	Y	TERM/RH-VE	S	N	N	N	N	N	N	N	N	N	Y	A	A	N	-
53	21	A	IV	1	17.5	1	12.9	A	T	LN	Y	TERM/IUGR/PROM	S	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/RDS
54	24	A	III	1	17.6	1	14.9	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
55	23	A	IV	2	17.9	1	15.2	A	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
56	21	A	III	1	17.4	1	15.6	A	T	LN	Y	OLI/IUGR/GHT	S	Y	Y	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/BA
57	24	A	III	2	18	1	14.7	A	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/OBS
58	23	A	III	2	18.1	1	15.6	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
59	21	A	IV	2	18.3	1	14.3	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
60	20	A	III	1	17.6	1	15.4	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-

61	21	A	III	2	17.9	1	12.9	A	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
62	23	A	III	1	17.4	1	17.3	A	T	LSCS	Y	TERM/ IUGR	F	Y	N	N	N	N	N	Y	N	N	N	A	A	N	LBW/RDS
63	24	A	IV	1	17.4	1	15.3	A	Pr	LSCS	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	N	-
64	21	A	III	1	17.2	1	14.6	A	T	LN	N	-	-	Y	N	N	N	N	Y	N	N	Y	Y	A	A	N	-
65	24	A	III	2	17.9	1	16.5	A	T	LN	Y	TERM/IUGR	-	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/BA
66	25	B	III	1	17.4	1	14.3	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
67	26	B	IV	1	17.1	1	14.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
68	27	B	III	1	17.4	1	15.4	A	Pr	LSCS	Y	OLI/IUGR	F	Y	Y	N	N	Y	N	Y	Y	N	Y	A	L	Y	LBW/BA
69	25	B	III	2	18.1	1	15.4	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
70	24	B	III	1	17.4	1	15.6	A	T	LN	Y	PROM	S	N	N	N	N	N	N	N	N	N	Y	A	A	N	-
71	26	B	III	2	17.9	1	15	A	Pr	LSC	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/R

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72	27	B	IV	1	17.4	1	15.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
73	28	B	IV	1	16	1	15.6	A	T	LSCS	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
74	26	B	IV	1	16.4	1	15.2	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
75	28	B	II	1	16.7	1	15.1	A	T	LSCS	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
76	29	B	IV	1	16.5	1	14.5	A	Pr	LN	Y	OLI	-	Y	N	N	N	Y	N	Y	N	Y	N	A	A	N	-
77	25	B	III	1	16.9	1	14.3	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	Y	N	A	A	N	-
78	31	C	IV	2	17.4	1	14.6	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
79	32	C	IV	2	18	1	14.5	A	Pr	LN	Y	PPROM	S	N	N	N	N	N	N	N	N	N	Y	A	L	Y	LBW/PND
80	26	B	II	1	17.1	1	13.6	A	T	LN	Y	TERM/IUGR	S	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/RDS
81	28	B	IV	1	16.9	1	13.5	A	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/RDS

82	27	B	III	1	17.6	1	14.3	A	T	LN	Y	PROM	S	N	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/OBS
83	26	B	III	1	17.4	1	15.2	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
84	20	A	IV	1	17.6	1	17	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
85	26	B	II	2	18	1	16.1	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
86	27	B	II	1	18.1	1	16	A	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	Y	A	L	Y	LBW/RDS
87	28	B	II	1	16.4	1	15.6	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
88	27	B	III	1	16.9	1	15.2	A	Pr	LN	Y	PPROM	-	N	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/PND
89	28	B	II	1	16.3	1	14.5	A	T	LN	N	-	-	N	N	N	N	N	N	Y	N	Y	N	A	A	N	-
90	26	B	II	1	16.4	1	14	A	Pr	LN	Y	OLI/IUGR	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/RDS
91	31	C	II	2	32.3	4	13.6	E	Po	LSCS	Y	GHT/GDM	F	Y	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
92	32	C	II	2	31.6	4	15.9	E	Po	LSCS	Y	GHT/GDM	F	N	Y	Y	N	N	Y	N	N	Y	Y	A	M	Y	LGA/IDM

93	30	C	II	2	32.4	4	14.9	E	Po	LSCS	Y	SEVERE PE	F	Y	Y	Y	N	Y	N	Y	Y	Y	Y	A	A	N	
94	30	C	II	2	33.6	4	16.2	E	Po	LSCS	Y	SEVERE PE	F	Y	Y	Y	N	Y	N	Y	Y	Y	Y	A	M	Y	LGA/IDM
95	31	C	II	2	33.4	4	15.3	E	Po	LSCS	Y	GHT/GDM	F	Y	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
96	33	C	II	2	32.6	4	15.4	E	Po	LSCS	Y	SEVERE PE	F	Y	Y	Y	N	Y	N	Y	Y	Y	Y	A	M	Y	LGA/IDM
97	34	C	II	2	31.9	4	14.9	E	Po	LSCS	Y	GHT/GDM	F	Y	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
98	32	C	II	2	34.6	4	14.6	E	Po	I	Y	GHT/GDM	S	N	Y	Y	N	N	Y	N	N	Y	Y	A	M	Y	LGA/IDM
99	31	C	II	2	33.4	4	14.5	E	Po	I	Y	RH-VE/GHT/GDM	S	Y	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
100	32	C	II	2	30.4	4	14.6	E	T	I	N	-	-	N	Y	Y	N	N	Y	N	N	Y	Y	A	M	Y	LGA/IDM
101	32	C	II	2	30.7	4	14.9	E	T	LSCS	N	-	-	N	Y	Y	Y	N	Y	N	N	Y	N	A	A	Y	LGA/IDM
102	31	C	II	2	32.9	4	14.2	E	T	LSCS	Y	SEVERE PE	F	N	Y	Y	N	Y	N	Y	Y	Y	N	A	L	N	LGA/IDMMSAF

103	34	C	II	2	31.9	4	15.1	E	Po	LSCS	Y	GHT/GDM	F	N	Y	Y	N	N	Y	N	N	N	Y	A	A	Y	LGA/IDM/RDS
104	32	C	II	2	30.9	4	16.1	E	Po	I	Y	GHT/GDM	S	N	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
105	34	C	II	2	30.1	4	15.9	E	Po	I	Y	GHT/GDM	S	N	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM/RDS
106	32	C	II	2	34.1	4	15.7	E	Po	I	Y	GHT/GDM	S	N	Y	Y	N	N	Y	N	Y	Y	Y	A	A	Y	LGA/IDM/RDS
107	31	C	III	2	33.4	4	15.3	E	Po	LSCS	Y	GHT/GDM	F	Y	Y	Y	N	N	Y	N	Y	Y	Y	A	A	Y	LGA/IDM
108	33	C	III	2	32.9	4	15.2	E	Po	LSCS	Y	GHT/GDM	F	N	Y	Y	N	N	Y	N	N	N	Y	A	A	Y	LGA/IDM/RDS
109	24	B	III	2	34.1	4	16.1	E	Po	I	N	-	-	N	Y	Y	N	N	Y	N	N	N	N	A	A	Y	LGA/IDM
110	26	B	III	2	33.7	4	14.3	E	Pr	LN	Y	SEVERE PE	S	N	Y	Y	N	Y	N	Y	Y	Y	Y	A	L	N	-
111	27	B	III	1	32.6	4	16.9	E	Pr	LN	Y	SEVERE PE	S	N	Y	N	N	Y	N	Y	N	Y	N	A	L	N	-
112	28	B	III	1	32.7	4	8.6	A	T	LSCS	N	-	-	N	Y	N	Y	N	N	N	N	N	Y	A	A	N	-
113	26	B	III	1	33.4	4	8.	A	T	LSC	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-

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114	25	B	III	1	32.6	4	8.6	A	Po	I	N	-	-	N	N	Y	N	N	N	N	N	N	N	A	A	N	-
115	26	B	III	1	32.6	4	8.7	A	Po	I	N	-	-	N	N	Y	N	N	N	N	N	N	N	A	A	Y	RDS
116	28	B	I	1	34.5	4	8.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
117	24	B	I	1	33.7	4	8.8	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
118	28	B	IV	1	31.8	4	8.9	A	T	LSCS	N	-	-	N	Y	N	N	N	N	N	N	N	N	A	A	N	-
119	29	B	II	1	32.6	4	12.1	E	T	LSCS	N	-	-	N	Y	N	N	N	N	N	N	Y	N	A	A	N	-
120	27	B	II	1	34.5	4	8.6	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
121	26	B	II	2	26.7	3	8.9	A	Po	I	Y	GHT/GDM/PD	S	N	Y	Y	N	N	N	N	N	N	N	A	A	N	-
122	28	B	II	2	28	3	11	A	Po	I	Y	PD/RH-VE	S	N	N	N	N	N	N	N	N	N	Y	A	A	N	-
123	29	B	II	2	29	3	10.6	A	Po	LSCS	Y	GHT/PD	F	Y	Y	N	N	N	N	N	N	N	Y	A	A	N	-
124	28	B	II	2	29.6	3	10	A	Po	LN	Y	GDM/PD	S	N	N	Y	N	N	N	N	N	N	Y	A	A	N	-

							.4																			
125	26	B	II	2	28.5	3	10.3	A	Po	LSCS	Y	GHT/PD	F	N	Y	N	N	N	N	N	N	Y	N	A	A	N	-
126	27	B	II	2	27.6	3	10.5	A	Po	LN	N	-	S	N	N	Y	N	N	Y	N	N	N	N	A	A	N	-
127	28	B	II	2	26.9	3	10.8	A	Po	LSCS	Y	GHT/PD	F	N	Y	N	N	Y	N	Y	N	N	Y	A	L	Y	LBW/IUGR/RDS
128	26	B	II	2	25.9	3	11.1	A	Po	LN	Y	GDM/PD	S	N	N	Y	N	N	Y	N	N	N	N	A	A	N	-
129	27	B	II	2	26.4	3	11.3	A	Po	LN	N	-	-	Y	Y	N	N	N	N	N	N	N	Y	A	A	N	-
130	29	B	II	2	28.3	3	10.9	A	Po	LSCS	Y	GDM/PD	F	N	N	Y	N	N	Y	N	N	N	N	A	A	N	-
131	26	B	II	2	29.1	3	9.9	A	Po	LN	N	-	-	N	Y	Y	N	N	Y	N	N	N	N	A	A	N	-
132	29	B	II	2	28	3	10	A	Po	I	Y	GDM/PD	S	N	N	Y	N	N	Y	N	N	N	Y	A	A	N	-
133	26	B	II	2	26.3	3	9.6	A	Po	I	Y	GHT/GDM/PD	S	N	N	Y	N	N	Y	N	N	N	Y	A	A	N	-
134	27	B	II	2	29.2	3	9.7	A	Po	I	Y	PD/RH-VE	S	Y	N	Y	N	N	Y	N	N	N	N	A	A	N	-

135	29	B	II	2	28.1	3	9.6	A	Po	I	N	GDM/PD	S	N	N	Y	N	N	Y	N	N	N	Y	A	M	Y	LGA/IDM/OBS
136	28	B	II	2	27.6	3	9.3	A	Po	LSCS	Y	GHT/GDM/PD	F	N	Y	Y	N	N	Y	N	N	Y	N	A	M	Y	LBW/IDM/OBS
137	25	B	II	2	29.1	3	9.4	A	Po	LSCS	Y	GHT/PD	F	N	Y	N	N	N	N	Y	N	Y	Y	A	L	Y	LBW/RDS
138	26	B	II	2	28.1	3	10.8	A	Po	LSCS	N	-	-	N	Y	N	N	N	N	N	N	Y	N	A	A	N	-
139	27	B	II	2	26.4	3	10.5	A	Po	LSCS	Y	GDM/PD	F	N	N	Y	N	N	Y	N	N	Y	N	A	M	Y	LGA/IDM/RDS
140	29	B	II	2	26.9	3	10.4	A	T	LN	N	-	-	N	Y	Y	N	N	Y	N	N	N	N	A	A	N	-
141	28	B	II	2	26.8	3	8.9	A	T	I	N	-	-	Y	Y	Y	N	N	Y	N	N	N	Y	A	A	N	-
142	29	B	II	2	27.4	3	9.9	A	T	LN	N	-	-	N	Y	N	N	N	N	N	N	N	N	A	A	N	-
143	29	B	II	2	27.6	3	9.3	A	T	LN	N	-	-	N	Y	Y	N	N	Y	N	N	N	Y	A	A	N	-
144	28	B	II	2	27.9	3	11.2	A	T	LN	N	-	-	N	N	Y	N	N	Y	N	N	N	N	A	A	N	-

145	27	B	II	2	27.1	3	11.1	A	T	LN	N	-	-	N	N	Y	N	N	Y	N	N	N	N	A	A	N	-
146	27	B	II	2	27.6	3	10.4	A	T	LSCS	N	-	-	N	Y	N	N	N	N	N	N	N	Y	A	A	N	-
147	26	B	II	2	27.4	3	10.9	A	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
148	27	B	II	2	28.6	3	11	A	T	LN	Y	TERM /GDM	S	N	N	Y	N	N	Y	N	N	N	Y	A	M	Y	LGA/IDM/BA
149	29	B	II	2	28.1	3	10.6	A	T	I	N	-	-	Y	Y	N	N	N	N	N	N	N	N	A	A	N	-
150	26	B	II	2	28.6	3	10.9	A	T	I	Y	PROM	S	N	N	N	N	Y	N	N	N	N	Y	A	A	N	-
151	27	B	II	2	28.9	3	10.3	A	T	LSCS	N	-	-	N	Y	N	Y	N	N	N	N	N	N	A	A	N	-
152	28	B	II	2	26.4	3	10.2	A	T	I	Y	RH-VE/TERM	S	N	N	N	N	N	N	N	N	N	N	A	A	N	-
153	26	B	II	2	26.9	3	10.6	A	T	LN	N	-	-	N	Y	Y	N	N	N	N	Y	N	N	A	M	Y	LGA/IDM
154	29	B	II	2	26.4	3	10.7	A	Pr	LN	Y	SEVERE PE	F	Y	Y	Y	N	Y	Y	Y	N	N	Y	A	L	Y	LATE Pr/LBW/RDS

155	26	B	II	2	26.7	3	9.9	A	Pr	LN	Y	PPROM	F	N	Y	Y	N	N	Y	N	N	N	N	A	A	Y	LATE Pr/LBW/PPROM
156	27	B	II	2	26.9	3	10.5	A	Pr	LSCS	Y	SEVERE PE	F	Y	Y	Y	N	Y	Y	Y	Y	N	N	A	L	N	-
157	29	B	II	2	26.8	3	10.2	A	Pr	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	N	A	A	Y	LATE Pr/LBW/PNDION
158	28	B	II	2	26.2	3	10.7	A	T	LSCS	N	-	-	N	N	Y	Y	N	N	N	N	N	Y	A	A	N	-
159	29	B	II	2	26.8	3	10.6	A	T	I	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
160	24	A	II	2	26.7	3	10.9	A	T	LN	N	-	-	N	N	Y	N	N	Y	N	N	N	Y	A	A	N	-
161	23	A	III	1	27.9	3	10.4	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
162	23	A	III	1	27.6	3	15.1	E	Po	LSCS	Y	GHT/GDM/PD	F	N	Y	Y	N	N	Y	N	N	Y	Y	A	A	N	-
163	22	A	III	1	27.3	3	13.6	E	Po	LSCS	Y	GDM/PD/GHT	F	N	Y	Y	N	N	Y	N	N	Y	Y	A	A	N	-

164	24	A	III	1	28.6	3	12.9	E	Po	LSCS	Y	GDM/PD/GHT	F	N	Y	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM
165	23	A	III	1	28.9	3	13	E	Po	LSCS	Y	GDM/PD/GHT	F	N	Y	Y	N	N	Y	N	N	Y	Y	A	A	N	-
166	31	C	III	1	28.6	3	13.6	E	Po	I	Y	GHT/RH-ve	S	N	Y	N	N	N	N	N	Y	Y	Y	A	A	N	-
167	30	C	III	1	29.4	3	13.9	E	Po	I	Y	GHT/PD/GDM	S	N	Y	Y	N	N	Y	N	N	Y	N	A	M	Y	LGA/IDM
168	32	C	III	1	29.3	3	13.8	E	Po	I	Y	GDM/PD	S	N	N	Y	N	N	Y	N	Y	Y	Y	A	A	N	-
169	33	C	I	1	29.1	3	14	E	Po	I	Y	GDM/PD	S	N	N	Y	N	N	Y	N	N	N	Y	A	M	Y	LGA/IDM/RDS
170	31	C	I	1	26.7	3	12.5	E	Po	I	Y	GDM/PD/GHT	S	N	Y	Y	N	N	Y	N	N	N	N	A	A	N	-
171	34	C	I	1	26.9	3	12.9	E	Po	I	Y	GDM/PD	S	N	N	Y	N	N	Y	N	N	N	Y	A	M	Y	LGA/IDM/RDS
172	32	C	I	1	26.7	3	12.6	E	Po	LSCS	Y	PD/GDM	F	Y	N	Y	N	N	Y	N	Y	Y	Y	A	M	Y	LGA/IDM/RDS
173	32	C	I	1	26.9	3	13.6	E	Po	LSCS	Y	PD	F	N	N	N	N	N	N	N	N	Y	N	A	M	Y	LGA/IDM
174	31	C	I	1	26.1	3	13	E	Po	LSC	Y	PROM	F	N	N	Y	Y	N	Y	N	Y	Y	N	A	M	Y	LGA/ID

						.9			S																M/RDS
175	30	C	I	1	25.9	3	13.8	E	Po	I	Y	RH-ve/PD/GHT	S	N	Y	N	N	N	N	N	Y	N	Y	A	A	N	-
176	31	C	I	1	25.4	3	14.2	E	Po	I	N	-	F	N	Y	Y	N	N	Y	N	N	N	N	A	M	Y	LGA/IDM
177	32	C	I	1	25.8	3	14.6	E	Po	I	N	-	F	N	Y	Y	N	N	Y	N	N	N	Y	A	M	Y	LGA/IDM
178	26	B	I	1	26.9	3	13.9	E	Po	LSCS	Y	PROM	F	N	Y	Y	N	N	Y	N	Y	Y	Y	A	A	N	-
179	27	B	I	1	28.1	3	15	E	Pr	LSCS	N	-	F	N	Y	Y	Y	N	Y	N	N	N	N	A	L	Y	LBW/RDS
180	29	B	I	1	29.6	3	14.6	E	Pr	LN	N	-	F	N	Y	Y	N	N	Y	N	Y	N	Y	A	L	Y	LBW/RDS
181	21	A	III	1	19	2	10.1	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS
182	22	A	III	1	19.5	2	10.2	I	Pr	LN	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS

183	20	A	III	1	19.6	2	10.6	I	T	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	
184	21	A	III	1	19.5	2	10.9	I	Pr	LN	Y	PPROM	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS
185	22	A	III	1	19.7	2	10.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS
186	21	A	III	1	19.2	2	10.4	I	Pr	LSCS	N	-	-	Y	Y	N	Y	N	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS
187	23	A	III	2	19.2	2	11.2	I	T	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	
188	21	A	III	1	19.6	2	0.6	I	Pr	LN	N	-	-	N	N	N	N	Y	N	Y	N	N	N	A	L	Y	LATE PRETERM/LBW/PNDION
189	20	A	III	1	18.6	2	10.8	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	EARLY PRETERM/LB

																									W/RDS
190	22	A	III	1	18.7	2	10.9	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	PRETERM/LB W/RDS
191	23	A	III	1	18.6	2	11.2	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	Y	A	L	Y	LATE PRETERM/LB W/BA
192	24	A	III	1	18.8	2	11.1	I	Pr	LN	N	-	-	Y	Y	N	N	Y	N	Y	N	N	N	A	L	Y	LATE PRETERM/LB W/RDS
193	21	A	III	1	18.9	2	11.3	I	T	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	-
194	21	A	III	1	19	2	10.9	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	PRETERM/LB W/RDS /MSAF
195	23	A	III	1	19.2	2	10.7	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	PRETERM/LB W/OBS
196	22	A	III	1	19.6	2	10.8	I	Pr	LSCS	N	-	-	Y	Y	N	Y	N	N	N	N	N	N	S	L	Y	PRETERM/LB

																									W/OBS
197	23	A	III	1	19.7	2	9	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	Y	A	L	Y	LATE PRETE RM/LB W/RDS
198	23	A	III	2	19.8	2	9.5	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETE RM/LB W/RDS
199	21	A	III	1	19.6	2	9.9	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	PRETE RM/LB W/RDS /MSAF
200	20	A	III	1	19.7	2	10.6	I	Pr	LN	Y	PPROM	S	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETE RM/LB W/RDS /PPROM
201	24	A	III	1	18.7	2	10.6	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LATE PRETE RM/LB W/RDS
202	32	C	III	1	18.9	2	10	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LATE PRETE

						.7																			RM/LB W/RDS
203	20	A	III	1	18.6	2	10 .3	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	N	N	N	N	A	L	Y	LATE PRETE RM/LB W/BA
204	23	A	III	1	18.6	2	10 .6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	PRETE RM/LB W/RDS
205	21	A	III	1	19.1	2	10 .4	I	Pr	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	L	Y	LATE PRETE RM/LB W/RDS
206	31	C	III	1	19.2	2	10 .6	I	T	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	-
207	23	A	III	1	19.3	2	10 .8	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	PRETE RM/LB W/OBS
208	21	A	III	1	19.5	2	10 .3	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	late preter m/LB W/RDS
209	27	B	III	1	19.4	2	11	I	Pr	LN	Y	PPROM	S	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/P RETER

						.1																			M/OBS
210	23	A	III	2	20	2	11.2	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PRETERM/RDS
211	21	A	III	1	20.1	2	8.5	I	Pr	LN	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
212	20	A	III	1	20.6	2	9.9	I	Pr	LSCS	N	-	-	Y	Y	N	Y	Y	N	N	N	N	N	S	L	Y	LBW/PRETERM/BIRTH ASP
213	23	A	III	1	19.6	2	9.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
214	21	A	III	1	19.6	2	9.3	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	Y	N	A	L	Y	LBW/PRETERM/PPROM
215	20	A	III	1	19.4	2	10.1	I	Pr	LN	N	-	-	N	N	N	N	N	N	N	N	Y	N	A	L	Y	LBW/LATEPRETERM/RDS
216	21	A	III	1	19.3	2	11	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PRETER

						.2																			M/OBS
217	25	B	III	1	19.5	2	10.3	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
218	26	B	III	2	20.1	2	10.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
219	23	A	III	1	20.3	2	10.4	I	Pr	LSCS	N	-	-	N	N	N	N	Y	N	N	N	N	N	A	L	Y	LBW/PRETERM/RDS
220	26	B	III	1	20.1	2	10.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
221	21	A	III	1	20.4	2	9.6	I	Pr	LN	Y	PPROM	S	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
222	20	A	III	1	19.6	2	8.9	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
223	23	A	IV	1	19.5	2	8.8	I	Pr	LN	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
224	24	A	III	1	19.4	2	8.	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/P

						7																			RETER M/OBS
225	21	A	III	1	19.2	2	9.1	I	Pr	LSCS	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
226	20	A	III	1	19.1	2	9.2	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
227	23	A	III	1	19.2	2	9	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
228	26	B	IV	1	19.1	2	8.2	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
229	21	A	III	1	19.6	2	8.8	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
230	20	A	III	1	19.3	2	9.1	I	Pr	LN	N	-	-	Y	N	N	N	Y	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS
231	23	A	III	1	19.4	2	9.3	I	Pr	LN	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETER M/OBS

232	20	A	III	1	19.6	2	9.1	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/OBS
233	21	A	III	1	19.7	2	9.9	I	Pr	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/PRETERM/OBS
234	20	A	III	1	19.4	2	9.8	I	Pr	LN	N	-	-	N	N	N	N	Y	N	N	N	N	Y	A	L	Y	LBW/PRETERM/OBS
235	23	A	III	1	19.6	2	9.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/PRETERM/RDS
236	23	A	III	1	19.8	2	9.7	I	Pr	LN	N	-	-	Y	Y	N	N	N	N	N	N	N	N	A	L	Y	LBW/PRETERM/OBS
237	21	A	III	1	20	2	9.9	I	T	LN	N	-	-	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-
238	22	A	III	1	20.1	2	9.6	I	Pr	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	L	N	LBW/PRETERM/OBS
239	22	A	II	1	20.3	2	8.6	I	T	LN	Y	PROM	S	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	-
240	21	A	II	1	20.6	2	8.	I	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-

						7																			
241	20	A	IV	1	20.4	2	8.9	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
242	27	B	II	1	20.2	2	8.4	I	T	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	-
243	26	B	IV	1	20.3	2	8.9	I	T	LSCS	N	-	-	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-
244	29	B	II	1	20.4	2	8.6	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
245	28	B	IV	1	20.7	2	8.4	I	T	LSCS	Y	OLI/IUGR	F	Y	N	N	N	Y	N	Y	N	N	N	A	A	N	-
246	27	B	III	1	21	2	8.2	I	Pr	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	L	Y	LATE PRETERM/LBW/RDS
247	24	A	IV	1	21.5	2	8.6	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	Y	N	A	A	N	-
248	21	A	IV	1	20.6	2	8.7	I	T	LN	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	-
249	20	A	IV	1	19.2	2	8.1	I	T	LSCS	N	-	-	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-

250	23	A	IV	2	19.4	2	8.9	I	T	LSCS	N	-	-	Y	N	N	N	N	N	Y	N	N	N	A	A	N	-
251	24	A	IV	2	19.4	2	8.4	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
252	21	A	IV	2	19.6	2	8.6	I	T	LN	Y	OLI/IUGR	S	Y	Y	N	N	Y	N	Y	N	N	N	A	A	N	-
253	20	A	IV	2	19.4	2	8.4	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
254	21	A	IV	2	19.7	2	8.6	I	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
255	20	A	IV	2	19.6	2	8.9	I	T	LSCS	N	-	-	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
256	23	A	IV	2	19.1	2	8.1	I	T	LSCS	N	-	-	N	N	N	N	Y	N	N	N	N	N	A	A	N	-
257	24	A	IV	2	19.6	2	8.6	I	T	LN	Y	RH-VE /TERM	S	N	N	N	N	N	N	N	N	N	N	A	A	N	-
258	23	A	IV	2	19.3	2	9.1	I	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
259	22	A	IV	2	18.7	2	10.9	I	Pr	LSCS	N	-	-	Y	Y	N	N	N	N	N	N	N	Y	A	L	Y	LBW/RDS/PRETERM

260	21	A	IV	2	18.6	2	11	I	T	LSCS	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
261	20	A	III	1	21.3	2	12.5	A	Pr	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/PRETERM/OBS
262	21	A	III	1	21.6	2	13.5	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
263	26	B	III	1	21.5	2	14.6	A	Pr	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	L	Y	LBW/Observation
264	24	A	III	1	20.9	2	15	A	T	LN	N	-	-	Y	N	N	N	N	N	N	N	N	N	N	A	A	Y	AGA/MSAF/RDS
265	27	B	II	1	20.4	2	15.5	A	Pr	LN	N	-	-	N	Y	Y	N	N	Y	N	N	N	Y	A	A	N	-
266	28	B	III	1	20.5	2	16	A	T	LN	N	-	-	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
267	26	B	III	1	20.7	2	15.6	A	T	LSCS	Y	OLI/TERM	F	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-
268	27	B	III	1	22.3	2	14.6	A	Po	LN	N	-	-	N	N	Y	N	N	Y	N	N	N	Y	A	A	N	-
269	23	A	III	1	22.5	2	15.9	A	Pr	LN	N	-	-	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-

270	22	A	III	1	22.4	2	15.7	A	Po	LSCS	N	-	-	N	Y	N	N	N	N	N	N	N	N	A	L	N	-
271	27	B	II	1	20.4	2	14.9	A	T	LN	Y	IUGR/TERM	S	N	N	N	N	N	N	Y	N	N	N	A	L	Y	LBW/IUGR/RDS
272	29	B	III	1	21.6	2	14.8	A	Po	LN	Y	PD	S	N	N	N	N	N	N	N	N	N	N	A	A	N	-
273	28	B	III	2	22.6	2	14.7	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
274	21	A	III	2	24.1	2	14.2	A	T	LN	Y	IUGR/TERM	S	N	N	N	N	N	N	Y	N	N	N	A	A	N	-
275	29	B	III	2	24.3	2	15.8	A	Po	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	Y	AGA/RDS/PROM
276	28	B	II	2	23.9	2	15.9	A	T	LN	N	-	-	N	N	N	N	Y	N	N	N	N	N	A	A	N	-
277	26	B	III	2	23.4	2	15.8	A	T	LN	Y	PROM	S	Y	N	N	N	N	N	N	N	N	N	A	A	N	-
278	25	B	III	2	23.5	2	15.4	A	T	LN	N	-	-	N	Y	N	N	N	Y	Y	N	N	N	A	A	N	-
279	24	A	III	2	22.3	2	15.6	A	Po	LN	N	-	-	N	N	Y	N	N	N	N	N	N	Y	A	A	N	-

280	23	A	III	2	22.5	2	15.8	A	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
281	26	B	III	2	22.9	2	15.4	A	T	LN	Y	GDM/TERM	S	Y	N	Y	N	Y	Y	N	N	N	N	N	A	A	N	-
282	25	B	III	1	22.1	2	12.6	A	T	LSCS	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
283	24	A	III	1	22.7	2	12.9	A	Po	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
284	26	B	II	1	22.9	2	13.6	A	T	LN	Y	GHT/TERM	S	N	Y	N	N	N	Y	N	N	N	Y	A	A	N	-
285	28	B	II	1	22.6	2	13.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
286	29	B	II	1	23.4	2	14.5	A	T	LN	Y	OLI/TERM	S	Y	N	N	N	Y	N	N	N	N	N	N	A	A	N	-
287	31	C	II	2	23.8	2	14.6	A	T	LN	Y	RH-VE/TERM	S	N	N	N	N	N	N	N	N	N	N	N	A	A	N	-
288	30	C	II	2	22.1	2	12.8	A	Po	LN	N	-	-	N	Y	N	N	N	Y	N	N	N	N	N	A	A	N	-
289	33	C	II	2	23.7	2	12.9	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	Y	N	A	A	Y	AGA/ MSAF/ RDS

290	27	B	III	2	23.8	2	13.9	A	T	LN	Y	PROM	-	N	N	N	N	N	Y	N	N	N	N	A	A	N	-
291	28	B	II	1	23.7	2	14.6	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
292	23	A	III	1	21.6	2	14.9	A	T	LN	Y	TERM/GHT	S	N	Y	N	N	N	N	N	N	N	N	A	A	N	-
293	24	A	II	1	21.9	2	14.6	A	T	LN	Y	TERM/GDM	S	N	N	Y	N	Y	Y	N	N	N	Y	A	A	Y	AGA/IDM/RDS
294	25	B	II	1	23.9	2	14.3	A	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
295	26	B	II	1	22.4	2	16.9	E	T	LN	N	-	-	N	Y	N	N	N	N	N	Y	N	Y	A	A	N	-
296	27	B	III	1	21.9	2	16.8	E	T	LN	N	-	-	N	N	N	N	N	N	N	N	N	N	A	A	N	-
297	29	B	III	1	23.9	2	17	E	T	LSCS	Y	TERM/GDM	F	N	N	Y	N	N	Y	N	N	N	N	A	A	Y	AGA/IDM/OBS
298	28	B	II	1	24.1	2	17.2	E	T	LN	N	-	-	N	N	N	N	N	Y	N	N	Y	N	A	A	N	-
299	26	B	II	2	23.9	2	17.2	E	T	LN	N	-	-	N	N	N	N	N	N	N	N	Y	N	A	A	N	-

300	27	B	II	2	23.1	2	16.7	E	Po	LSCS	Y	PD/GHT	F	N	Y	N	N	N	Y	N	N	Y	Y	A	M	N	-
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