# "FOETAL MACROSOMIA - RISK FACTORS, MATERNAL AND PERINATAL OUTCOME -A ONE YEAR CROSS SECTIONAL STUDY"

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## Endorsement by the HOD, Principal/Head of the Institution

This is to certify that the dissertation entitled "**FOETAL MACROSOMIA-RISK FACTORS, MATERNAL AND PERINATAL OUTCOME -A ONE YEAR CROSS SECTIONAL STUDY**" is a bonafide research work done by **REG. NO BJ0119005.**

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# PLAGIARISM ACCEPTED LETTER

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# ABBREVIATIONS

| | | |
|---|---|---|
| PPH | - | Postpartum hemorrhage |
| LSCS | - | Lower Segment Caesarean Section |
| KLE's | - | Karnataka Lingayat Educational Society |
| BMI | - | Body Mass Index |
| RCT | - | Randomized Controlled Trial |
| DOME | - | Department of Medical Education |
| IUFD | - | Intra Uterine Fetal Demise |
| KAHER | - | KLE Academy of Higher Education and Research center |
| JNMC | - | Jawaharlal Nehru Medical College |
| Kg | - | Kilograms |
| gm | - | gram |
| m | - | meter |
| ml | - | milliliters |
| HELLP | - | Hemolysis, elevated liver enzyme, low platelet |
| i.v. | - | intravenous |
| i.m. | - | intramuscular |
| PCV | - | Packed cell volume |
| Hb | - | Hemoglobin |
| DIPSI | - | Diabetes in pregnancy study group of india |
| GDM | - | Gestational diabetes mellitus |

| | | |
|---|---|---|
| ODM | - | Overt diabetes mellitus |
| µg | - | microgram |
| WHO | – | World Health Organisation |
| g/dl | – | gram/ decilitre |

## TABLE OF CONTENTS

## LIST OF FIGURES

## LIST OF TABLES

# 1. INTRODUCTION

Fetal macrosomia is defined as excessive growth in foetus irrespective of the gestational age. Macrosomia is currently a cause of concern in most developing countries, as it leads to morbidity, mortality, and disability on a global scale. It has also been established that being born macrosomic has been associated with health risks later in life[1]. Growth above a certain limit is termed macrosomia. Fetal macrosomia commonly refers to infants with a weight above 4000 g but in countries with higher income, the upper limit is set at 4500 g. Hence for better clarity, a grading system is designed with grade 1 referring to infants weighing 4000 to 4499 g, grade 2 for 4500 to 4999 g, and grade 3 for over 5000 g. But this system refers to macrosomia in new born and cannot be applied to any other gestational age. Statistically macrosomia at any gestational age is defined as growth greater than $90^{th}$ percentile for that gestational age[2].

Assessment of fetal and new born weight should involve use of the latest and country specific percentile tables especially in countries with low and middle income, as this table varies from country to country because of ethnic and racial influence on birth. Moreover, birth weights of new born have increased over the past years rendering the older tables useless[3].

Occurrence of fetal macrosomia has been estimated to affect between 6% and 10% of all new-borns and a rise in the number of fetal macrosomia cases and babies with birth weights above the 90th percentile for gestational age were reported by researchers in various parts of the world[4]. The incidence varies with ethnicity as average birth weights varies for different races, for example Asians have lower average birth weights, hence incidence has been proposed to be between 3 to 15 % by

some some studies[5]. In Canada, the number of babies with higher-than-average weight increased from 8% in 1978 to 11.5 percent in 1995. Similarly, between 1992 and 2001, the percentage of new born babies weighing over 4500 g in Sweden rose from 3.7 to 4.6 percent. From 1994 to 2000, the incidence of macrosomia became higher in China[6].

Birth weight above the normal range is responsible for various complications in both mother and child and the risk for maternal and perinatal morbidities increases drastically, with birth weight above 4500 g[7]. The risk of complications rises as the degree of macrosomia rises. Boulet et al. found that delivery complications increase in frequency beginning at a birth weight of 4000 g[8], but neonatal morbidity increases dramatically only at a birth weight of 4500 g. At a weight of 5000 g, neonatal mortality increased[9].

It is clear from existing research that fetal macrosomia is linked to poor pregnancy outcomes for both the mother and the baby. Furthermore, there is a known connection between fetal macrosomia and long-term effects on the newborn, such as obesity, diabetes, and heart disease. Various scientific studies have reported complications in the mother due to macrosomia, which encompass emergency Caesarean section (CS) due to fetal distress or a lack of progress in the delivery, postpartum haemorrhage and injury to the anal sphincter. There's a good chance for the occurrence of uterine atony. Heavy bleeding and postpartum haemorrhage may result due to failure of contraction of uterus muscle. In macrosomic deliveries, the incidence of postpartum bleeding and genital tract injury was 3-5 times higher, as well as a greater risk of uterus tear in case of previous caesarean sections in the mother[10].

Moreover, effects of Macrosomia on new born is also substantial. These include shoulder dystocia and associated problems like brachial plexus injury,

fractured clavicle or humerus and birth asphyxia[11]. Furthermore, the babies were more likely to experience birth trauma, need resuscitation, and have an Apgar score of less than seven at five minutes of life[12]. Macrosomic babies also have a higher chance of developing type 2 diabetes, hypertension, and obesity as adults[13]. Macrosomia also considerably enhances the possibility of admission to the neonatal intensive care unit. There is a chance of preterm delivery if labor is induced before 39 weeks of pregnancy and/or the membranes are ruptured prematurely. Despite taking all appropriate precautions prior to inducing early labour, new borns are still at risk of complications associated with prematurity, such as breathing and feeding problems, infection, jaundice, and neonatal intensive care[10]. There is considerable variation in published research about the following parameters like the population studied, macrosomia distribution, design of study, and complications caused, as a result of which, although there is widespread recognition of the connection between macrosomia and these complications, there is no specific guidance about reliable evidence-based estimates of risks for the mother and the neonates[11].

Complications associated with macrosomia sometimes vary based on the maternal complication which causes this particular condition, for example gestational diabetes. Macrosomic fetuses with diabetic mothers exhibit a particular kind of overgrowth, which includes deposition of subcutaneous fat in the abdominal and interscapular areas, which results in their possessing larger shoulder and extremity circumferences, lower head-to-shoulder ratio, considerably more body fat and thick upper-extremity skinfolds. Rb's palsy, shoulder dystocia, and brachial plexus trauma are more common because fetal head size is not increased, but shoulder and abdominal girth may be significantly increased. Skeletal development is mostly unaffected. Macrosomic neonates of mother with Diabetes showed 5 times higher

rates of severe hypoglycemia and increase in neonatal jaundice as compared to new born from non-diabetic mothers. Macrosomic infants from diabetic pregnancies were also hyper insulinemic[10].

The various risk factors associated with macrosomia include previous birth of a macrosomal child, gestational diabetes, particularly with insufficiently controlled blood glucose values, maternal obesity, significant weight gain during pregnancy, male sex of the foetus, more than 41 + 0 weeks of pregnancy, as and multiparity. The presence of gestational diabetes as well as possessing a history of macrosomic birth in the reproductive period and presence of preeclampsia in the pregnancy period were found to be one of the major predictors of macrosomia[9]. Factors associated with macrosomia may also include maternal age, pre-pregnancy BMI. Its more widespread in developed counties as well as in women belonging to an affluent stratum of society. It is important to identify the local risk factors for macrosomia in order to take effective steps during the antenatal period to minimize its prevalence thereby reducing the complications associated with it[1]. Nowadays more women enter the pregnancy period with excess weight. The 2015 National Survey of Nutrition Situation in Colombia recorded a total of 39.9% of pregnant women of all age groups. Similarly studies of pregnant women in places like Peru, Brazil, and Uruguay observed 63.8%, 47.5% and 32.6%, of overweight women at the beginning of pregnancy and as mentioned above obesity in mothers is linked to macrosomia. In a survey of 23 nations, macrosomia was found to be present in 4.5 percent to 5.4 percent of the population in Latin America. It ranged from 5% to 20% in developed countries, with a 15-25% rise recorded in the last three years. Macrosomia reached 4.5% in full-term new-borns in Colombia[14]. Moreover, studies have shown a direct link of increase in BMI with occurrence of foetal macrosomia.

The most important step to counter this problem which has been rapidly posing as a complication in obstetric is the detection of macrosomia. Prevalent methods can be either clinical or ultrasound estimations, and in the majority of case clinical estimation has been proved to be as accurate as those by ultrasonography. Clinical estimation involves volume of amniotic fluid, the size and configuration of the uterus and maternal body habitus and shows high error. Ultrasonography on the other hand has been proposed as a more accurate method of estimation of fetal weight. Unfortunately, the typical mean error ranges from 300 to 550 g [15]. However, although ultrasonographic estimations are accurate for predicting birth weights up to 3500g, problems occur when the foetus is getting larger. Increased maternal BMI results in a reduction of the accuracy in estimating foetal weight by ultrasound[16]. Although diagnosis of macrosomia in infants mostly rely on ultrasonography these are not highly predictable for detection of macrosomia and the possibility of accurate diagnosis of macrosomia by ultrasound is approximately 22-37%[4]. Moreover, sensitivity of ultrasonographic measurement of the foetal weight shows a wide variation and weighing the newborn is considered the most effective way of diagnosing foetal macrosomia till date. Clinical examination using Leopold 's manoeuvres and calculating the height of the uterine fundus above the maternal symphysis pubis and ultrasonographic evaluation remain imprecise[17].

Management strategies for fetal macrosomia encompass clinical intervention, caesarean section and early induction of labor. Though clinical interventions for treating suspected macrosomia in women without Diabetes have not been reported, one clinical trial showed the efficacy of insulin administration in reducing fetal macrosomia in pregnancies complicated by diabetes. One limited clinical trial looked at the effects of dietary activity with or without the use of insulin in diabetic

pregnancies. The findings suggest that adding insulin to the treatment of early macrosomia could be beneficial (between 29 and 33 weeks of gestation)[18].

Although elective caesarean section is considered as a management strategy for macrosomia not only to prevent complications in birth as well as reduce trauma associated with birth. Unfortunately, due to the the difficulties associated with it´s diagnosis a large number of unnecessary caesarean sections would be required to avoid a single bad outcome in a pregnancy complicated by suspected fetal macrosomia. Thus, elective caesarean section for suspected macrosomia would be difficult to carry out. The other option is an early induction of labour. since the fetus gains about 230 g per week after the 37th week, induction of labor before or near term could prevent macrosomia and its complications. However, studies indicate an increase the cesarean section rate with induction[15].

# 2. OBJECTIVE

Fetal macrosomia is rapidly growing in stature as a major cause of complications in pregnancy and is responsible for causing numerous maternal and peri-natal complications. Management of macrosomia has not been streamlined till date. Moreover, diagnostic methods as well as predictors for this condition has not been sufficiently studied.

Based on the lacuna in literature, the objectives of the study are as follows:

**Primary objective**

To determine maternal and perinatal outcome associated with foetal macrosomia

**Secondary objective**

To assess the risk factors and assess the incidence of fetal macrosomia

# 3. REVIEW OF LITERATURE

Fetal macrosomia is defined as extensive intrauterine growth resulting in an increase in the weight at birth. The precise definition is ambiguous. In most studies, the limit is set at a birth weight of 4000 g, however in certain circumstances, a weight of 4500 g is also used. Excessive intrauterine growth is sometimes described as growth that exceeds the 90th percentile termed large for gestational age. IQTIG – Institute for Quality Assurance and Transparency in Health, found out that 8.9% of all newborns weighed between 4000−4499 g and 1.2% weighed more than ≥ 4500 g in 2016 in Germany. According to the observation of the German perinatal survey data 2007−2011, a rise in birth weight was not observed in Germany compared to the years 1995−1997[9] .

Macrosomic infants are classified as those with a birth weight above 4000-4500 g and researchers show a specific interest in infants whose birth weight exceeds 5000 g. Based on the alterations in cut off limits, some researchers have, subdivided macrosomia into three classes, Class I (birth weight 4000–4499 g), Class II (4500–4999 g), and Class III (≥5000 g)[12]. However, because these limits are not based on population data, where normal weight is commonly defined as between the 10th and 90th percentile for gestational age, they are ineffective in diagnosing premature macrosomic fetuses (assuming a normal population distribution). Any fetus/infant weighing more than the 90th percentile for gestational age is called big for gestational age. Some researchers also use the 95$^{th}$ percentile as the threshold for macrosomia as it corresponds to 1.90 standard deviations (SD) above the mean and defines 90 percent of the population as normal weight. Others use the 97.75$^{th}$ percentile, which corresponds to 1.96 SD above the mean and defines 95 percent of the population as

normal weight. If the population is constitutionally more uniform yet differs from published data, local tables should be considered wherever possible. When assessing estimated fetal and infant weight, it's best to utilize current country-specific percentile figures, especially in low- and middle-income nations. Because newborn weights have risen in recent decades, rendering older tables useless. Since differences in race and ethnicity exert an effect on birth weight, it should be taken into account for interpretation of estimated fetal and newborn weight[2].

The American College of Obstetricians and Gynecologists (ACOG) classified macrosomia as a birth weight of more than 4,000 grams, regardless of gestational age, or more than the 90th percentile for gestational age after accounting for newborn sex and ethnicity. These pregnancies account for 3–15 percent of all pregnancies and are responsible for the occurrence of a large number of maternal and neonatal complications[13]. Since a diagnosis of fetal macrosomia is possible only after delivery; the presence of this condition can be confirmed only after delivery of the neonate[19].

In a study done by yvonne kwun-yue cheng et al risk factors identified are maternal BMI , weight gain and multiparity , in maternal outcome prolonged labour 15% and higher incidence of operative deliveries are seen perineal tears have increased 1.5 times more ,shoulder dystocia 0.5%

In a study done by Grzegorz Piasek et al, 652 newborns were analysed shoulder dystocia 0.8%, clavicular fracture, convulsions in the newborn are significantly associated with foetal macrosomia operative vaginal delivery 3.49%

In a study done by florent Fuchs et al 15% had elective cesarean section ,10% had BMI >30, Mean weight gain was 14 kg, induction rate was 33% , perineal tears was 26% ,20% had PPH .mean birth weight of babies was 4.2 Kg

In a study done by Sahruh Turkmen et al labour induction was seen in 26%, rate of caesarean delivery was 60%, shoulder dystocia 2.5%, mean birth weight of babies was 4.6 kg, prematurity seen in 11 %. Respiratory distress in 26%.

In a study done by W N Spellacy et al macrosomic infants occured in 1,7% of deliveries 405 of high parity, most of them 45% are diabetics , higher rate of cesarean deliveries 78% most of the infants had shoulder dystocia 0.5% and low APGAR scores about 55% of babies required NICU admissions

In a study done by Mariam Mathew et al rate of macrosomic births were 3.7%. Mean birth weight of 4.2Kg, caesarean rate is 25.8% incidence of shoulder dystocia was 7.6% PPH was seen in 28% of cases

In a study done by C I Kamanu et al. 92.55 were multiparous , maternal median weight gain was 11 kg, mean maternal BMI was 30,2, GDM was seen in 2.5% , caesarean rate was 15 % ,3.8% delivered by ventouse, 4 % had shoulder dystocia ,perinatal mortality was 8%

In a study done by H U Ezegwul et al , incidence was 8.1% , mothers with macrosomic babies were older with mean age of 30, high parity of 4 , 39.5 % had previous H/O macrosomia, 27.3% of caesarean rates, 3.6% of operative vaginal deliveries , 2.3% of shoulder dystocia , still birth rate was 3,2%

In a study done by B R Rasmussen et al. incidence was 8.1%, 29.5% had previous H/O macrosomia, GDM rate was 40 %, 29.3% of caesarean rates, 3% of operative vaginal deliveries, 2.3% of shoulder dystocia, still birth rate was 1, 2%

In a study done by P Panel et al incidence is 4.5%, previous H/o macrosomia 12.6%, increase in the weight gain during pregnancy of more than 15 kg is seen in 46, 8%, 9.09% of caesarean deliveries, 23.8% was instrumental deliveries, 9.5% shoulder

dystocia, 15 % of neonatal morbidity, 14 % of maternal morbidity with 9 cases had PPH, 9 cases had perineal trauma, 6 infections

In a study done by V Porozhanova et al incidence was 2.7% , prolonged labour is seen in 9.7%, shoulder dystocia and perineal tears are seen in 2,5% , foetal distress is seen in 7.14% , GDM seen in 19% .

In a study done by H D Modanlou et al incidence is 1.4%, factors that occured frequently were maternal obesity 385 , diabetes mellitus 48% , previous h/o macrosomia 38%, rate of caesarean section was 70% , 30 % of babies required NICU admissions

In a study done by Rehab Husain Basher et al maternal age (51.7%), multiparity (61%), maternal diabetics (29.59%), significantly associated with macrosomia Total caesarean rate was 26.4% and. 3% of birth trauma

In a study done by Aisha salim said et al prevalence is 2.3%, mean birth weight of 4.2 kg, maternal age ranging between 30-39 years. Delivery weight >80 kg maternal complications like PPH (20%). prolonged labour (10%) second degree perineal tears (10%)

## 3.1 PREVALENCE OF FETAL MACROSOMIA

Any population's mean birth weight is a useful health indicator. Low mean birth weight meant inadequate development. However, if the newborns were big for gestational age (LGA), it could pose as a source of problem for the mother and the infant. Many studies from the West in recent years have depicted increase in the average birth weight, as well as an increase in the proportion of LGA babies. In Canada, the proportion of LGA infants born grew from 8% in 1978 to 11.5 percent in 1995. Similarly, between 1992 and 2001, the percentage of newborn newborns

weighing above 4500 g in Sweden grew from 3.7 to 4.6 percent. From 1994 to 2000, the incidence of macrosomia, defined as a birth weight of more than 4000 or 4500 grams, rose in China][6]. Incidence of macrosomia showed a steady increase over the past years in a number of countries, for example in Queensland, Australia it increased from 12.2% in 1988 to 12.8% in 2005 and the proportion of macrosomia increased in Beijing, China from 6.6% in 1996 to 9.5%, with an increase in gestational weight gain beyond the recommended limit. In Brazil, however, macrosomia prevalence decreased from 2001 to 2010 and stayed largely steady from 2012 to 2014. In the United States an overall decrease in macrosomia incidence from 9.9 to 8.8% was observed, even though maternal obesity increased[5] .

Studies on Turkish women, revealed macrosomia rates in the range of 5.2% to 7.6% it was assumed to be around 5.9% in pregnant women who didn't have gestational diabetes. The incidence of fetal macrosomia in a study among similar patient population living in Aegean territory was found to be 8.6%, which is much higher than the prevalence reported in other parts of Turkey. This difference may be attributed to the variations in characteristics and socioeconomic strata of participants[4]. According to more recent estimates, macrosomia affects around 8% of all live births in the United States. However, there is no evaluation of current developments. Despite the fact that macrosomia is less common than other unfavorable pregnancy outcomes, it remains a significant public health concern due to the lifetime hazards it entails, including baby and maternal difficulties[5].

Fetal macrosomia is assumed to be higher in developed countries as their nutritional levels are higher. The prevalence of fetal macrosomia has been reported to be higher, recording an increase of 15-20% in developed countries which owes its existence to an increase in maternal obesity and diabetes whereas in developing

countries Nigeria, Uganda and Algeria, respectively an incidence of 7.5%, 8.4% and 14.9% has been recorded.

According to other studies, macrosomia ranges from 5% to 20% at all births in developed countries, and there has been an increase in its prevalence of 15–25% in the past 20 years. The frequency of macrosomia ranged from 0.5 percent in India to 14.9 percent in Algeria, according to research conducted in 23 developing nations throughout Asia, Africa, and Latin America. According to the conclusions of two studies, the macrosomia rate in Chad is 7.6 percent, whereas in Iran it is 11.8 percent, and the frequency of macrosomia in Ethiopia ranges from 6.7 percent to 19.1 percent[20].

## 3.2 PATHOPHYSIOLOGY OF FETAL MACROSOMIA

The pathophysiology of macrosomia is based on the underlying maternal condition associated with it. The major factors related to macrosomia include diabetes, obesity of mother, and uncontrolled weight gain in the mother, all of which are characterised by intermittent periods of hyperglycemia in common. this results in an increase in human placental lactogen, free and total cortisol, and prolactin in the 2nd half of pregnancy, which results in insulin resistance, that is in turn confronted by post-prandial hyperinsulinemia. Gestational diabetes results in individuals, who are unable to exert a hyperinsulinemic response. Fetal hyperglycemia occurs when glucose crosses the placenta by facilitated diffusion, thereby causing fetal hyperinsulinemia and transfer of glucose into fetal cells which leads to fetal macrosomia. Macrosomia is caused by fetal hyperinsulinemia, which results either directly by its anabolic influence on nutrition intake and utilization, or indirectly by related peptides such insulin like growth factors. Insulin, insulin-like growth factors,

growth hormone, and other growth hormones are stimulated in the fetus as a result of hyperglycemia., subsequently resulting in fetal growth and deposition of fat and glycogen. Insulin, growth hormone (GH), and growth factors (insulin-like growth factors and their binding proteins (IGFBPs affect embryonic development and the production and release of hormones leptin and ghrelin Insulin-like growth factor I (IGF-I) affects fetal development later in pregnancy. Its is affected by placental glucose transfer, which regulates production of fetal insulin. In addition, insulin has a adipogenic impact on the fetus, and fetal growth hormone (GH) may have additional fetal growth modes of action. Both maternal and fetal IGF-I influences placental metabolism[21].

According to recent studies white adipose tissue is an active endocrine organ releasing hormones, which play a significant role in regulation of metabolism, energy homeostasis, and growth. Leptin and ghrelin, are two important members of this system, which send signals regarding nutritional status and energy storage levels to the hypothalamic feeding centres. Leptin plays an important role in increased fetal growth; which is supposed to be due to its interaction with the IGF system. Wiznitzer et showed a significant association between umbilical cord leptin and insulin-like growth factor-I levels and birth weight. Moreover umbilical cord leptin concentration was also proved to pose as an independent risk factor for occurrence of fetal macrosomia[22]. It was proved that both maternal and fetal ghrelin increase with the length of gestation at delivery. Studies proved production of higher ghrelin during late gestation, which later goes to fetus, moreover they showed presence of, acyl and des–acyl ghrelin t in the maternal and fetal circulations in the 2$^{nd}$ pregnancy phase and also indicate a role of maternal and fetal ghrelin in the fetal development. These studies

established the role of hormones and growth factors in development of fetal macrosomia[23] .

### 3.3 DIAGNOSIS OF FETAL MACROSOMIA

Diagnosis of macrosomia during pregnancyis difficult to detect, the only signs being large fundal height and excessive amniotic fluid. During prenatal visits, the fundal height is measured , which is the distance between uterus top to pubic bone, and an unusually high value indicates presence of fetal macrosomia. Similarly, presence of excessive amniotic fluid (polyhydramnios), may indicate presence of a macrosomic baby[13]. According to ACOG, the two basic procedures for clinical fetal weight estimates are Leopold's procedures and measuring the height of the uterine fundus above the maternal symphysis pubis. Since only one of these measures cannot predict fetal macrosomia, they must be used together to obtain a more accurate assessment.

Prenatal ultrasonography has essentially supplanted clinical diagnosis for macrosomia in developed nations. Fetal macrosomia is diagnosed with a sensitivity of 10-43 percent and a positive predictive value of 28-53 percent using fundal height measurement alone or in conjunction with symphysis-fundal height assessment. The poor diagnosis rate of fetal macrosomia by clinical approaches might be owing to aberrant fetal position, oligo- or polyhydramnios, maternal obesity, a scarcity of skilled obstetricians, or a vaguely defined fundus fixed point. Recent studies have revealed the efficacy of ultrasound measurement of fetal abdominal circumference (AC) in detecting variable fetal weights better than clinical examination. Ultrasound measures bone length and fetal body circumference more accurately thereby making

prenatal sonography a more effective option for carrying out weight estimates before birth.

Several studies have proved the efficacy of the AC method for detecting fetal macrosomia. Moreover, advances in sonographic imaging in the late 1990s have made three-dimensional views of fetal structures easy. The best formula for diagnosing fetal macrosomia combines of two- and three-dimensional biometrics and is given below:

Fetal estimated weight = -1,478.557 + 7.242 × thigh volume + 13.309 × upper-arm volume + 852.998 × $\log_{10}$ abdominal volume + 0.526 × $BPD^3$

Though it reduces the mean error by 6-7%, still the time-consumingnature and the scarcity of this method renders it ineffective. Moreover, small sample size of macrosomic fetuses and the heterogeneous study population pose as hampering factors.

Various factors affect the accuracy of ultrasound method for macrosomia like suboptimal views with oligohydramnios, obesity in mother, multiple pregnancies and fetal defects like gastroschisis, omphalocoele, and hydrocephalus which alter in biometric calculations[24].

Ultrasonographic measuring of the fetus can rule out fetal macrosomia, which may help to reduce maternal morbidity, however it´s accuracy in macrosomia detection is not deemed any better than Leopold's procedure[18] (Chatfield et al., 2001). Moreover, the ultrasound techniques do not possess high accuracy in detection and prediction of macrosomia and the probability of correct diagnosis by ultrasound is only 37-22%[13]. Hence measuring the weight of the newborn after delivery may be the only way for accurate diagnosis of macrosomia.

### 3.4 RISK FACTORS FOR FETAL MACROSOMIA

The various factors which may result in development of macrosomia in newborns include maternal obesity, gestational age at delivery, maternal pre-pregnancy body mass index (BMI weight gain during pregnancy, hypertension and cigarette smoking. Another study showed that gestational diabetes was the most important risk factor behind macrosomia births, other factors being history of macrosomic births and development of preeclampsia during pregnancy. Moreover, it also showed the significant effect of diabetes, as well as age of the mother and BMI on macrosomic birth[13]. Existence of depression in mother, 1 poor maternal education, low socioeconomic situation, and lack of parental connection or stimulation with the child are some other risk factors. It has also been proposed that a poor uterine environment can have both immediate and long-term consequences for a fetus, particularly during critical developmental stages[25] .

On the contrary a considerable decrease in the chances of developing fetal macrosomia was observed among mothers who had pregnancy-related hypertension, chronic hypertension and eclampsia[5]. ACOG committee has named the following as predictors of fetal macrosomia history of macrosomia, maternal weight prior pregnancy, weight gain during pregnancy, multiparity, male fetus, gestational age more than 40 weeks, ethnicity, maternal birth weight, maternal height, maternal age less than 17 years, ethnicity[18] . Analysis of effect of various risk factors on fetal macrosomia was studied and it was observed that age, parity, BMI, GWG and fetal sex were significantly associated with fetal macrosomia rate. Moreover ROC analysis revealed fetal macrosomia was considerably higher in women with age ≥ 30, parity ≥ 1 and gestational weight gain ≥ 12 in the study population. The probability of giving birth to macrosomic fetus increased in mothers ≥ 30 years of age, >1 of parity, a pre-

pregnancy BMI of ≥ 25, ≥ 12 of GWG and male fetal sex. Moreover, pre-pergnancy BMI and GWG were shown to have a strong relation with fetal macrosomia[4].

**Maternal obesity:** The risk of macrosomia in newborns of obese women was double the risk for those of normal weight women. Additionally, there was a 15 times high risk probability of obese moms developing macrosomia was fifteen times higher than normal weight moms. Recent research has revealed that pre-pregnancy BMI is a key predictor of macrosomia[10] Maternal obesity has been reported to pose twice the risk of macrosomia as compared to women with normal BMI. Some studies showed 1.5-2.3 increase in chances of large for gestational age newborns being born in obese women. According to a study by Usta et al., it was observed that compared to pregnant women with normal BMI, overweight and obese pregnant women showed an increase in the odds of delivering macrosomic infants. They also observed that pregnant women with more than 25 kg/m$^2$ pre-pregnancy BMI show increased risk of delivering macrosomic babies[4]. Another study reported that women with BMI ≥25 had slightly increased risk were at an increased risk for macrosomica in their babies[26]. This was similar to observations by Bergmann et al. who reported higher chance of macrosomic births in women with pre-gestational BMI ≥26 kg/m$^2$

BMI ≥30 in the father exerts an influence in the occurrence of macrosomia which might show the involvement of father's genetic factors development of macrosomia[26].

**Diabetes:** Studies on effect of diabetes on infant growth reveal that fetal birth weight is associated with second- and third-trimester postprandial blood sugar levels but not with fasting or mean glucose levels. Macrosomia is observed in 20% of infants, whose mothers had postprandial glucose levels of average 120 mg/dl or less, and

when values are as high as 160 mg/dl, macrosomia rate becomes 35%. In diabetic pregnancies, macromic fetuses acquire a distinct pattern of overgrowth, comprising central deposition of subcutaneous fat in the abdominal and interscapular region. They have thicker upper-extremity skinfolds, bigger shoulder and extremity circumferences, a lower head-to-shoulder ratio, and much more body fat. Moreover, the risk of Erb's palsy, shoulder dystocia and brachial plexus trauma is more due to increase in shoulder and abdominal breadth, whereas head size is not increased. Skeletal growth remains same[27]. Effect of severity of maternal fasting hyperglycemia and the risk of shoulder dystocia, was reported by a Australian Carbohydrate Intolerance Study in Pregnant Women, which reported a 1-mmol increase in fasting glucose increasing the risk for shoulder dystocia to2.09[10].

Fetal macrosomia can be caused by irregularities in maternal postprandial blood glucose levels and increased insulin release during pregnancy, especially in the second and third trimesters. The Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study discovered a consistent link between maternal glucose and newborn weight gain. Treatment of gestational diabetes mellitus (GDM) was successful in lowering the incidence of macrosomia, preeclampsia, and shoulder dystocia, according to Falavigne et al. 2012[28]. For pregnant women with pre-gestational diabetes mellitus or GDM, the risk of fetal macrosomia should be recognized throughout prenatal treatment. One study showeda higher risk of delivering macrosomic babies in women who had untreated borderline gestational diabetes, in case gestational diabetes is not diagnosed and subsequently not treated, risk of begetting macrosomic babies increases to 20 percent[18].

**Weight gain during pregnancy**: Studies have shown the strong corelation between weight gained during pregnancy and macrosomic babies and chances of giving birth

to a macrosomic baby in mothers with ≥16 kg weight gain during pregnancy were 11 times higher than normal weight gain. Similar results were reported in another study which showed that chances of macrosomia with mothers who gained more than ≥16 kg was10.2 times greater than mothers who gained < 16 kg[29]. Pregnant women who gained too much weight had a nearly 2.5-fold increased risk of hypertensive disorders of pregnancy and a 2.7-fold increased risk of preeclampsia, according to research. Usta et al., showed that gestational weight gain was considerably more in macrosomia group as compared to control and weight gain of 12 kg during the pregnancy, increased the risk of fetal macrosomia by 5.5 fold[4]. Maternal weight gain above 16 kg during pregnancy was found to be a risk factor by Nkwabong et al which could also indicate that increase in nutritional intake during pregnancy may pose as a risk factor for macrosomia[26].

**History of macrosomic births**: Previous history of macrosomic births exerts a strong influence on the current pregnancy. According to a research, the chances of giving birth to a macrosomic infant were 7 times higher in moms who had previously given birth to a macrosomic baby than in women who had never given birth to a macrosomic baby[1]. Women who had previously had macrosomic babies had a higher chance of having another macrosomic baby which shows that previous history of macrosomic baby birth poses as a significant risk for macrosomic babies in subsequent pregnancies[26].

**Maternal age:** The association of macrosomia with maternal age was also evident in this study; the odds of delivering macrosomic babies by mothers aged ≥30 kg during pregnancy were 2.6 times greater than those from mothers aged < 30 years. It was supported by various researchers. Said and Manji reported the significance of

maternal age ranging from 30 to 39 years in prediction of macrosomia[30]. A report from United Kingdom observed a 40% increase in chances of having macrosomic babies in women between 35 and 39 years as compared to 35 years old mothers as well as 20% increase in risk for over 40 years old mothers. Studies in Turkey also reported similar observations, that maternal age above 35 years increases chances of fetal macrosomia by 3 times. These results prove that Maternal age is an important risk factor for fetal macrosomia. It might be attributed to the fact that changes in metabolism result with an increase in age, and hormonal and endocrine factors, may result in higher fetal growth rates leading to increase in macrosomic births in older women[4]. A 2–3-fold elevated risk of macrosomia in women with higher age was reported by a study conducted[5].

**Parity:** Parity is also considered as predictor of fetal macrosomia and and researchers have shown the corelation between parity and macrosomial births. Approximately 70% multiparity rate was observed in macrosomic group[31]. A multiparity rate of 64% was observed in mothers with macrosomic newborns andsignificant high parity was observed in macrosomia group as compared to controls[4]. Multiparity has an effect on the occurrence of macrosomia, according to our findings. In fact, women with parity 3 were more likely than those with parity 3 to have macrosomic babies. This might be because birth weight increases with parity in the same mother[26].

**Sex of the newborn**: Macrosomia was found to be more common among male sex than females[26].

**Gestational age:** Gestational age at delivery exerts an effect on development of macrosomia, Post-term delivery has been termed as a risk factor [for development of fetal macrosomia[26].

A study by Malik et al reported that mothers older than 35 years hadmore macrosomic babies than those mothers who were under 35 years of age. They also showed the relation between educational level of the mother with occurrence of macrosomia. Moreover, mothers belonging to higher strata of society were reported to have higher chances of giving birth to macrosomic babies than mothers belonging to lower socioeconomic group. However, this correlation of macrosomia with mother‘s age, education and occupation was found to show little statistical significance. It showed an increase in occurrence of of macrosomia (10.3%) in diabetic mothers as compared to nondiabetic mothers (0.7%). Non anemic mothers showed increase in occurrence of macrosomic babies than anemic mothers[32].

### 3.5 MACROSOMIA RELATED COMPLICATIONS IN MOTHERS

Infection, postpartum hemorrhage, protracted labor, high degree perineal rips, cesarean birth, anesthetic mishaps, and thromboembolic events are normally conditions associated with fetal macrosomia[4]. Mohhemmadbeigi et al., also reported association of macrosomia with complications like prolonged labor, labor augmentation with oxytocin, cesarean delivery, postpartum hemorrhage, infection, 3rd- and 4th-degree perineal tears, thromboembolic events, and anesthetic accidents. Several complications occur during the delivery of a macrosomic baby, for example complications in vaginal birth occur when the baby is abnormally large[13]. Along with chances of prolonged labor , where there is a possibility of fetus getting stuck resulting in the necessity for instrument to pull the baby out or emergency cesarean section. The danger of laceration and tear of the vaginal tissue during delivery is higher than when the infant is of normal size, and the muscle between the vagina and the anus may tear (perineal tear). There's also a possibility of uterine atony. Heavy

bleeding and postpartum hemorrhage can occur if the uterine muscle fails to contract correctly.

Postpartum hemorrhage and genital tract damage may also occur. Moreover, in case of mothers with a previous history of cesarean section, risk of uterus tear along the scar line of the previous surgery is higher[10]. In a study by Said et al., 56.3 % of mothers in the macrosomic group were associated with minimum one complication. Whereas only 38.8 % of mothers in the control group showed any ($p < 0.05$). Most general complications reported included prolonged labor (27.2 %), 2nd degree perineal tears (22.3 %) and post-partum hemorrhage (PPH) (17.5 %). PPH was five times more frequent in mothers who had macrosomic babies than in controls. PPH was caused by uterine atony, perineal tears, and uterine rupture. In the macrosomia group, three women experienced shoulder dystocia, and two moms experienced uterine rupture (1.9 percent). In the macrosomia group, there was one maternal fatality; the cause of death was uterine atony. It was observed in a study by Nazafian et al., that maternal problems like uterine atony (11%), cervix/vaginal laceration (4.9%), and uterine rapture (0.4%) had a considerable correlation with macrosomia[19].

**Prolonged labor:** Women bearing macrosomic infants had longer labors, and the chance of miscarriage increases as the baby's birth weight rises. The first and second stages of labor are both lengthier in macrosomic pregnancies than in non macrosomic pregnancies, and descent of baby may stop in the second stage as a result of macrosomia. When delivering a macrosomic infant weighing more than 4,500 g, primigravidae had a greater risk of protracted labor than multiparous women. Maternal problems such as surgical delivery and postpartum haemorrhage might be exacerbated by prolonged labor caused by macrosomia[33].

**Risk of emergency Caesarean section**: With growing macrosomia, the manner of delivery changes dramatically. Macrosomic babies have a greater rate of vaginal operative birth and cesarean section (CS). The total percentage of CS in newborns weighing less than 4,000 grams varies significantly between studies, ranging from 14 percent to 44 percent. The chances of CS increase with a rise in birth weight and the odds of vaginal delivery aided by instrument reduces with an increase in birth weight. This increase in the risks of CS has been reported from various countries and in ethnic groups, with the odds being considerably high for primiparous mother[34]. Thirteen studies evaluated the risk of an emergency CS in macrosomia-affected pregnancies to those without it, with ten of them comparing data from 8 581 904 non-macrosomic pregnancies to 1 265 929 pregnancies with BW > 4000 g. In pregnancies with BW > 4000 g, the pooled summary OR for emergency CS was 1.98 (95 percent CI, 1.80–2.18). Meta-analysis from eight studies which encompassed 226 911 macrosomic neonates with BW > 4500 g, compared to 8 142 794 without macrosomia, showed increase in the chances of emergency CS by 2.5-fold[11]. For babies weighing 4.5 kg or more, the emergency CS rate is 45% and the instrumental delivery rate 19%[35].

**Post-partum haemorrhage**: After the delivery of macrosomic infants, postpartum haemorrhage (PPH) is more common, and the risk increases with increasing birth weight. This can either be attributed to large baby or other factors such as protracted labor, labor induction, surgical vaginal delivery, uterine atony, and perineal tears[36]. Higher chances of PPH in pregnancies with macrosomia have been reported in 11 studies. Nine among these carried out the analysis the differences between 7 960 844 non-macrosomic pregnancies and 1 042 965 pregnancies with BW > 4000 g. In pregnancies with a BW > 4000 g, OR for PPH was 2.05. Similarly, a meta-analysis of data from eight trials that included 182 276 macrosomic newborns with a BW > 4500

g compared to 7 508 373 without macrosomia found that the risk of PPH (postpartum haemorrhage) was raised 3-fold[11].

**Perineal trauma:** In situations of macrosomia, the incidence of perineal tears increases 1.5- to 2-fold. Some researchers claim that the risk of large perineal tears increases with birth weight, however this has been refuted. Asian, Filipino, and Indian women appear to be at a greater risk than Caucasian women, which might be related to variations in body shape and differences in structure of perineum. Major perineal damage, such as a third- or fourth-degree rupture, can result in substantial long-term anal incontinence, lowering a woman's quality of life[36]. There were eight research that looked at the link between macrosomia and obstetric anal sphincter damage (OASIS). The pooled OR was 1.91 in seven trials, with 683 121 pregnancies without macrosomia compared to 68 837 with BW > 4000 g. When the prevalence of OASIS was examined between 221 850 non-macrosomic pregnancies and 5183 with severe macrosomia in the same study, there was a 2.5-fold higher risk. Due to a higher head circumference (HC), extended labor, and difficult delivery, birth weights more than 4 kg increase the risk of perineal damage, particularly third- and fourth-degree rips[35].

**Anal incontinence**: Anal incontinence poses as a cause of trauma and social problem, though vaginal delivery has been identified to be the principal cause, macrosomia has been identified as a risk factor for anal sphincter problem. Moreover, shoulder dystocia associated with macrosomic infants results in perineal and anal sphincter damage. Anal sphincter injury has a significant role in maternal fecal incontinence[35].

**Vaginal prolapse**: Macrosomic babies may also damage the fascial supports of the pelvic floor resulting in damage of pelvic and pudendal nerves, leading to vaginal prolapse[35].

### 3.6 MACROSOMIA RELATED COMPLICATIONS IN INFANTS

Macrosomic fetuses had higher chances of developing neonatal hypoxia, meconium aspiration, clavicular fracture, brachial plexus damage, and shoulder dystocia, according to the American College of Obstetricians and Gynecologists (ACOG) practice bulletin. Additionally, prior studies have found that macrosomic babies are more likely to acquire hypertension, obesity, and type 2 diabetes later in life[18]. Similar observations were reported by other researchers. Macrosomia results in complications in infants like shoulder dystocia, brachial plexus injury, skeletal injuries, meconium aspiration, prenatal asphyxia, hypoglycemia, and fetal death as well as have higher chances of deveoping of type 2 diabetes mellitus, hypertension, and obesity at older age[13]. Polycythemia and hypocalcemia are observed in macrosomic babies born to diabetic mothers[30] (Said et al., 2016). A policy of cesarean section for fetuses over 4500 g has been advocated by various researchers. But increase in cesarean section rate may not result in a decrease in trauma and asphyxia of macrosomic babies. Though induction of labor before term has been advocated, it is beset with its own share of comorbidities[37]. In a study by Said et al., it was observed that complications in infants were more frequent in the macrosomic group (44.3 %) compared to the controls ($p < 0.05$). Hypoglycemia (22.7%), respiratory distress (16.5%), birth asphyxia (14.4%), and delivery trauma were the most frequent newborn problems in the macrosomic group (14.4%). The macrosomia group had a substantially greater mortality rate than the controls (p0.05). Stillbirth (6 cases and 1 control) and birth asphyxia were the most common causes of death among the cases. Three of the twelve macrosomic newborns that died had diabetes moms. Cephalhematoma and fractures (5.7 percent) were the most frequent injuries, followed by nerve palsy (3.1 percent ). Hypoglycemia was observed to be more predominant in

the macrosomic group compared to the controls (22.7 vs. 6.9 %) and occurred within 6 h of deliveryin 96 % cases. It was also more frequent in infants born to mothers without diabetes mellitus (23.5 %) compared to those born to diabetic mothers (16.6 %); however, the difference was not significant (*p*-value 0.7)[30].

**Preterm Birth**. There is a risk of preterm birth due to early induction of labor before 39 weeks and premature rupture of membranes. Despite taking all required measures prior to inducing early labor, babies are still at risk of problems associated with preterm, such as breathing and feeding problems, infection, jaundice, and neonatal intensive care unit hospitalization[10].

**Shoulder Dystocia and Erb's Palsy**. Shoulder dystocia, which is related with birth trauma, is one of the most severe consequences of vaginal delivery in macrosomic infants. The risk of delivery trauma is 6 times higher in newborns weighing 4,500 g or more, and the risk of brachial plexus damage is roughly 20 times greater when the birth weight is above 4,500 g[27] . Risk of shoulder dystocia in macrosomic pregnancies were reported by 10 studies, as compared to normal births. Eight of these analysed data between 349 400 non-macrosomic pregnancies and 39 481 pregnancies with BW > 4000 gand reported an OR for shoulder dystocia in pregnancies with BW > 4000 g to be 9.54 Additionally, meta-analysis of data obtained from six reports which analysed 5757 macrosomic neonates with BW > 4500 g, compared to 240 589 without macrosomia, showed an increase of 15-fold, in the chances of occurrence of shoulder dystocia[11].

Shoulder dystocia is responsible for long term damage in the infants. Average risk of shoulder dystocia is 1.4 percent generally in infants, but when the birth weight is more than 4500 g, the risk ranges from 9 to 24 percent. It was found to be 14.3 percent for non-diabetic newborns weighing 4500–4750 g and 21.1 percent for newborns

weighing 4750–5000 g. Shoulder dystocia was found in 23% of babies with birth weights between 4500 and 4750 g and 29% of those with birth weights between 4750 and 5000 g in vacuum- or forceps-assisted deliveries[35].

In Caucasians prevalence o of shoulder dystocia ranges lies between 0.58% and 0.70%. It also appears to vary with ethnicity, with an incidence of only 0.3% in the Chinese population.4 It has been reported consistently in the literature that the risk of shoulder dystocia escalates with increasing birth weight.4,6,23,24 However, the incidence of shoulder dystocia in different birth weight groups varies widely between studies. In a recent study in Norway,24 the incidence was approximately 1%, 2%, 4%, and 6% for birth weights of 4,000–4,199 g, 4,200–4,399 g, 4,400–4,599 g, and $4,600 g, respectively, whereas another study reported an incidence of over 20% when the birth weight was above 4,500 g. Nevertheless, despite such an association, half or even more of the births complicated by shoulder dystocia occur in babies with a birth weight less than 4,000 g[38].

**Hypoglycemia at Birth**. It is one of the most frequently occurring complications arising in infants due to macrosomia at birth. It arises as a result of the fetus's hyperinsulinemia in reaction to maternal hyperglycemia in gestation. Hypoglycemia can cause more significant consequences, such as severe central nervous system and cardiac problems. Neurologic damage resulting in mental retardation, recurrent seizure activity, developmental delay, and personality problems are among the most serious long-term consequences. Meshari et al., reported a rise in hyperbilrubinemia and hypoglycemia were observed in macrosomic babies of GDM mothers. The risk of newborn hypoglycemia is greater in heavy babies, and the risk rises as the birth weight rises. When compared to those of proper gestational age, neonates with a birth weight of 4,500 g had a seven-fold greater risk of neonatal hypoglycemia and it

increases in mothers with gestational diabetes[37]. Infants from nondiabetic mothers had a 2.4% chance of developing neonatal hypoglycemia, whereas those with mothers who had gestational diabetes reported an incidence of 5.3%[39].

**Neonatal Jaundice**. Prematurity, decreased hepatic conjugation of bilirubin, and increased enterohepatic circulation of bilirubin as a result of inadequate nutrition are all possible causes of jaundice. Neonatals with macrosomia have a high oxygen demand, which leads to increased erythropoiesis and, eventually, polycythemia. As a result, when these cells die, bilirubin (a by product of red blood cells) rises, leading in jaundice.

**Childhood Obesity and Metabolic Syndrome**. It was observed that macrosomic infants from mothers with gestational diabetes (GDM) were heavier as compared to offsprings from non GDM mothers after approximately 5 years of age[40].

**Fetal distress**

Intrapartum fetal discomfort and meconium in liquor, as well as the danger of meconium aspiration, are more common in macrosomic infants. Post maturity is associated with fetal discomfort and liquor staining from meconium, which is considerably enhanced in macrosomics. In the macrosomic babies, however, there was no increase in the frequency of hypoxia in newborns[37].

**Neonatal mortality**: Neonatal mortality is directly associated with increase in birth weight and increases rapidly in babies > 4.5 kg. this might be attributed to the association of macrosomia with longer gestationtonal age and diabetes in the mother as well as increase in the number of emergency CS and delivery assisted by instruments followed by occurrence of birth trauma in women with macrosomic fetus[41]. Numerous epidemiologic studies have shown a distinct relationship between

birth weight and neonatal and infant mortality, and have consistently demonstrated a reverse J pattern of weight-specific mortality in all populations, where the mortality rates increase at the extremes of birth weight. Compared with a normosomic group of infants with a birth weight of 3,000–3,999 g, babies with a birth weight .5,000 g had a 2–3-fold increase in risk of neonatal death, and a 1.6–2.0-fold increased risk of postneonatal and infant mortality, respectively. Such an association was not identified in babies with a birth weight of 4,000–4,999 g[6].

However, a recent study by Zhang et al,[7] which included close to 6 million births from the USA, showed that neonates with a birth weight of 4,500 g also had a higher early neonatal death rate (OR 1.8), but there was no increase in late or postneonatal death. Early, late, and postneonatal deaths were all significantly increased in those weighing $5,000 g, with ORs of 6.4, 5.2, and 2.3, respectively. The leading cause of early neonatal death in macrosomic babies was asphyxia. Sudden infant death syndrome is another concern for macrosomic babies, but the current data are conflicting. The majority of postneonatal deaths reported by Zhang et al[7] were due to sudden infant death syndrome. Infants with a birth weight $5,000 g have a more than 2-fold increase in risk. However, such a detrimental effect was not identified in other studies, and excessive intrauterine growth (birth weight .90th percentile) has even been shown to have a protective role in sudden infant death syndrome.

**Obstetric brachial palsy injury (OBPI):** A strong corelation between macrosomia and OBPI has been observed. The typical rate of OBPI in the obstetric population is 0.5–1.9/1000, with an 18–21-fold higher risk for babies born weighing more than 4.5 kg. 2. Macrosomic children born following shoulder dystocia had a greater risk of OBPI than non-macrosomic newborns. Overall, chances of developing OBPI in

macrosomic infants delivered vaginally is 4–8%[35]. Chances of permanent injury attributed to OBPI is about 10%.

According to a study, only 16.7% of OBPI cases spontaneously resolved, while 27% were permanent and severe (arm unusable for the rest of one's life) and 56% were permanent and moderate (abduction and rotation confined to less than 30 degrees)[42]. Moreover persistent OBPI has been reported to be six times more common in case of ≥ 4.0 kg birth weight as compared to < 4.0 kg birth weight.

**Birth trauma**: This includes brachial plexus and skeletal injuries, and has been reported to rise in case of overweight births. Brachial plexus injury (BPI) describes flaccid paresis of an upper part of body due to traumatic stretching. The incidence is different in different countries and is around 1.5 cases per 1,000 live births. Although damage is temporary, there have been cases of permanent damage (5% cases). High birth weight is the 2$^{nd}$ most significant risk factor for BPI and results in a 14-fold increase in risk[43]. Prevalence of BPI showed an increase with increase in infant weight, 3% of neonates had BPI in the 4,500–5,000 g group and 6.7% in the 5,000 g group. Chances increase in the presence of both macrosomia and gestational diabetes. Intensity of BPI among infants weighing >4,000 g was observed to be more than in the nonmacrosomic. It is difficult to detect and prevent congenital BPI because the two primary risk factors, shoulder dystocia and macrosomia, are not easily anticipated. Injuries to the bones, skeletal injuries, like BPI, are prevalent in the context of shoulder dystocia and are linked to big babies. In macrosomic newborns, clavicle fracture is five times more likely[36].

**Chorioamnionitis**: Chorioamnionitis risk shows a slow and steady rise with increase in birth weight birth with OR of 1.94, 2.17, and 2.42 for birth weight groups of 4,000–4,499 g, 4,500–4,999 g, and $5,000 g, respectively[44].

**Aspiration of meconium**: Risk of occurrence of this problem is related to macrosomia, as reported by several studies. The danger rises as the baby's birth weight rises. For newborns weighing 4,000–4,499 g, 4,500–4,999 g, and.5,000 g, the ORs are 1.28, 1.65, and 2.61, respectively. Other researchers, on the other hand, concluded that this association was not statistically significant[45].

**Perinatal asphyxia**: As compared to normosomic babies, the risk of perinatal asphyxia in macrosomic newborns is 2–4 times higher. Perinatal asphyxia increases dramatically with increasing birth weightthe OR was 2.3 at birth weights of 4,500–4,999 g and 10.5 for birth weights of.5,000 g[7] .

**Low Apgar scores**: The presence of macrosomia has been linked to lower Apgar scores. Low Apgar scores are more likely the higher the birth weight. Furthermore, when the delivery is exacerbated by shoulder dystocia, the chance of a poor Apgar score is eight times higher in macrosomic infants[46].

**Intrauterine fetal death:** Macrosomia has been consistently shown to be associated with a 2–3-fold increase in intrauterine fetal death. A significantly increased risk of stillbirth was observed with increase in birth weight, the risk rose dramatically with a birth weight of 5,000 Fetal death rate rose in macrosomic fetuses in both diabetic and nondiabetic pregnancies, a

though the birth weight limit was different, which was4,250 g in nondiabetic women and 4,000 g in diabetic mothers[36].

**Long-term complications:** Since events during fetal development have a significant influence on the risk for occurrence of diseases in adults higher birth weight is associated with development of diseases later in life like hypertension, obesity, and insulin resistance. Alternative reasons, primarily genetic variables, for the link between prenatal growth and subsequent illnesses have also been offered. Increased birth weight has been linked to obesity, insulin resistance, and metabolic syndrome later in life. When macrosomia and maternal gestation coincide, the risk of developing metabolic syndrome in infancy is greatest[47] (Boney et al., 2005). Interestingly, high birth weight has been linked to breast cancer in a number of studies. The most apparent increase in risk (OR 3.10, 95 percent CI 1.18–7.97) was seen in those with a relatively high birth weight (4,500 g). This link is thought to be mediated in part by hormonal processes that impact prenatal growth and mammary gland development favorably[36].

## 3.7 MANAGEMENT OF FETAL MACROSOMIA

### Management of labor and vaginal delivery

In the case of suspected fetal macrosomia, the most essential factor for labor and delivery is midpelvic surgical vaginal delivery. A cesarean birth should be undertaken for midpelvic arrest of a fetus with probable macrosomia, with the exception of grave situations. If a cesarean birth is necessary due to probable macrosomia, the incision should be large enough[18] (Chatfield et al., 2001). There are three options for labor management in cases with risk of macrosomia, namely induction of labour, expectant management or elective caesarean delivery.

**Induction of labor**

Since growth of fetus continues even after 37 weeks of pregnancy at 230 g/week induction of labor near term could control macrosomia and its complications. But induction of labour should be done keeping these two factors in mind, fetal lung maturation and a ripe cervix in the mother with a Bishop score of ≥6. The lung maturity of fetuses born to diabetes mothers has been demonstrated to be delayed. Normally, pulmonary maturation occurs between 34 and 35 weeks of pregnancy. 99 percent of fetuses are developed by 37 weeks. The lung in a diabetic mother's fetus, on the other hand, may not develop until 38.5 weeks. Absence of ripe cervix may result in failure of induction, finally resulting in cesarean section. Current data does not support induction of labor in term patients with suspected fetal macrosomia. Recent studies show that inducing labor more than doubles the chance of cesarean birth without lowering the risk of shoulder dystocia or neonatal morbidity, however the findings are skewed by the small sample size and retrospective nature of the studies[18].There are mixed outcomes when it comes to the benefits of labor induction. According to a Cochrane study and comprehensive review, inducing labor for suspected fetal macrosomia increases the caesarean delivery rate without enhancing neonatal outcomes[24]. One study examined the results of individuals who were suspected of having macrosomia before birth versus those who were not. Even after adjusting for birth weight and other confounding factors, the authors discovered that the likelihood of cesarean delivery was significantly in pregnancies where macrosomia was suspected and this is attributed to failure of induction methods[48].

**Elective Caesarean Section**

Many studies recommend that patients who are suspected of having a macrosomic baby, particularly those with GDM, insulin-dependent diabetes, and a previous high-birth-weight baby, have a caesarean section to avoid maternal and fetal delivery stress. Unfortunately, methods for calculating the fetus's weight are imprecise. Moreover, the role of caesarean delivery in managing fetal macrosomia remains fraught with uncertainties. While the risk of birth trauma increases with greater birth weight when delivered vaginally, caesarean delivery decreases, but does not eliminate, this risk. Furthermore, when any precise predicted fetal weight is uncertain, randomized clinical trial findings have not proven the therapeutic efficacy of preventive caesarean birth. Large cohort and case-control studies show that allowing a labor trial for an estimated fetal weight of greater over 4,000 g is safe. Nonetheless, the findings of these studies, as well as published cost-effectiveness statistics, do not justify prophylactic caesarean delivery for suspected fetal macrosomia with estimated weights of less than 5,000 g, while other experts believe that it may be necessary in rare cases[18].

To avoid birth trauma and labor dystocia, an elective caesarean delivery has been recommended in cases of suspected fetal macrosomia. When having a trial of labor, the majority of babies and mothers have a positive result, but 3,700 women with an estimated fetal weight of 4,500 g would need to have an elective caesarean birth to avoid one incidence of lifelong brachial plexus damage. During counselling about mode of delivery, patient's obstetric history, labour progress, and any proof of fetopelvic disproportion needs to be considered[24]. Because it is difficult to anticipate macrosomia and most women who have a trial of labor have a positive outcome, a huge number of needless caesarean sections would be required to avoid a single poor

outcome in a pregnancy complicated by suspected fetal macrosomia. It was estimated that to prevent one permanent brachial plexus injury, 3,700 women with an estimated fetal weight of 4,500 g would have to undergo caesarean. Hence elective caesarean section for suspected macrosomia alone is difficult to support[15].

**Management of the Neonate**

Congenital malformations (congenital heart defects, tracheoesophageal fistula, and central nervous system abnormalities) and delivery trauma are of greater concern in neonates with a diabetic mother. They should be closely monitored and assessed for hypoglycemia, polycythemia, hyperbilirubinemia, and electrolyte abnormalities. Level of glucose in blood, needs to be checked within 1 h of life, hourly for the next 6-8 h. Oral feeding, ideally breast feeding, is advised, and when that is not enough, an intravenous infusion needs to be administered.

**Clinical intervention**

There are no reports on clinical interventions for macrosomia treatment although in case of mothers with GDM, one study observed effect of insulin and results showed that the addition of insulin may be instrumental in treating early macrosomia (between 29 and 33 weeks of gestation). The research participants who received insulin in addition to dietary management had a lower chance of having a baby with a birth weight greater than the 90th percentile, dropping from 45 percent to 13 percent.

### 3.8 NEED OF STUDY

Fetal macrosomia is a rising concern in most developing nations, and it adds to illness, death, and disability on a global scale, either directly or indirectly. The causes of macrosomia are complicated and poorly understood. The study's main goal is to determine the incidence, causes, maternal and perinatal outcome in foetal macrosomia. Hence in our current work all the women delivering macrosomia babies at labor room at KAHER's Dr Prabhakar Kore Charitable Hospital, Belagavi were studied for risk factors associated with macrosomal births as well as maternal and perinatal outcome of this condition.

# 4.1 MATERIALS AND METHODS

## 4.1 STUDY DESIGN

The present study was a hospital based observational study to evaluate maternal and perinatal outcome associated with macrosomia and the risk factors associated with macrosomia. This study was conducted at KAHER's Dr. Prabhakar Kore Hospital, Belagavi for a period of 12 months. Data was collected from women who delivered macrosomia babies and had been informed about the study's purpose. Patients who expressed an interest in taking part in the trial were enrolled after signing a written informed consent form.

## 4.2 STUDY SETTING

The study was conducted at the Department of Obstetrics and Gynaecology of KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi, Karnataka. The hospital is a clinical training facility that provides free health care to the underprivileged in basic specialties. KAHER's Dr. Prabhakar Kore Charitable Hospital is recognized by the Medical Council of India, and Government of Goa along with industries in Maharashtra and Karnataka.

## 4.3 STUDY PERIOD

The study was conducted for a period of 1year and 6 months (from January, 2020 to june, 2021). The study period included enrolment of participants and data collection followed by analysis and reporting.

## 4.4 STUDY POPULATION

The study population consisted of women delivering macrosomia babies at labour labour room at the Department of Obstetrics and Gynecology, KAHER'S Dr.

Prabhakar Kore Hospital, Belagavi during the study period fulfilling the inclusion criteria and consenting to participate in the study.

**4.5 SAMPLE SIZE**

Sample size was obtained by the formula:

$$n = Z^2pq/d^2$$

Where:

*n* = sample size

Z = 1.96 corresponding to 95% confidence interval

p = proportion of participants (19% for this study)

q= 100-p

d = margin of error set at 5%

The minimum sample size was calculated to be 108.

During the study period of one year, it was not possible to achieve this number from cases presented to the department. As an alternative, all cases presented to the labor room and meeting the inclusion and exclusion criteria were considered.

**4.6 SAMPLING METHODS**

Universal sampling method was adopted for this study.

**4.7 SELECTION CRITERIA**

**4.7.1 Inclusion criteria:**

- Gestational age has been confirmed by 1st trimester ultrasound
- Fetal macrosomia diagnosed by ultrasound (abdominal circumference and estimated fetal weight [AC > $90^{th}$ percentile or EFW >90%]

### 4.7.2 Exclusion criteria:

- Multiple pregnancies
- Foetal malformations

## 4.8 DATA COLLECTION AND SAMPLING TECHNIQUES

Data was collected conveniently among the eligible women participating in the study. Eligibility was sought by checking their files for the diagnoses. The participants were interviewed and a pre-designed structured questionnaire was used to collect information after taking informed consent from the women delivering macrosomia babies. Data on newborn children was collected from clinical records. Relevant biochemical investigations were carried out employing routine methods.

Details of data collected are as follows:

### 4.8.1 Socio-demographic characteristics and history

Data on socio-demographic like maternal age, occupation, marital status, educational status of the mother, family size, physical exercise in a day, pregnancy intervals in years, pre-pregnant body mass index (BMI), total weight gain during pregnancy were obtained via interview. Obstetric history including gravida, parity, living, abortion (spontaneous/induced), still birth and gestational age were recorded on the pre-designed proforma.

Information of risk factors was also obtained from history of the patients, which included:

- Age
- Parity
- Weight gain during pregnancy
- Pre-pregnancy BMI

- Previous history of macrosomia
- Gestational age at delivery
- Anemia during pregnancy
- Diabetes during pregnancy
- Hypertension during pregnancy

**4.8.2 Clinical investigations**

Data on patient's VCTC, Hepatitis B surface antigen (HBsAg), blood group/Rh typing, haemoglobin levels, routine and microscopic examination of urine, peripheral smear, pregnancy induced hypertension (PIH) profile, DIPSI and thyroid profiles were collected and recorded.

Findings of obstetric ultrasound for crown rump length, gestational age in weeks, expected date of delivery (EDD) and corrected EDD were also recorded.

**4.8.3 Delivery related details**

Details on mode of delivery either normal vaginal delivery (spontaneous vaginal delivery, assisted breech delivery, vacuum extraction, forceps delivery) or Caesarean delivery (elective or emergency) were noted in the questionnaire.

**4.8.4 Maternal complications during delivery**

Information on presence of absence of complications during delivery including postpartum hemorrhage, prolonged labour, uterine rupture, $2^{nd}$ degree tear, shoulder dystocia, NICU admissions and maternal death were recorded.

**4.8.5 Neonatal characteristics**

Data on neonatal characteristics such as birth weight, height, head circumference, gender of neonate (male or female) were recorded.

### 4.8.6 Neonatal complications by mode of delivery

Complications seen in the new-born either by vaginal delivery or by Caesarean delivery were obtained via questionnaire. Parameters such as death, hypoglycaemia, polycythaemia, birth trauma, birth asphyxia and respiratory distress were included under neonatal complications arising due to macrosomia.

## 4.9 FOLLOW UP

Participants of the study were followed up after delivery to evaluate possible development of complications.

## 4.10 STATISTICAL ANALYSIS

Analysis of collected data was done using descriptive statistics since the study was an observational study. The data obtained was coded and entered into Microsoft Excel Worksheet. Statistical Package for the Social Sciences (SPSS) for Windows version 20.0 was employed for statistical analysis and interpretation of collected data.

Continuous quantitative variables were represented by mean ± SD (minimum, maximum). Data was divided into two groups with respect to certain qualitative characteristic for comparison. Krushal-Wallis one-way analysis of variance was used to compare distribution between the groups and pre and post treatment measures. Discrete variables were represented by median ± SD using non-parametric tests. Categorical data were expressed in terms of frequencies and percentages. Chi-square test was used to measure the strength of associations between the categorical data including outcomes, clinical and demographic characteristics. Suitable graphs were used to depict the comparisons. Probability values ('p' values) of $<0.05$ at 95% confidence interval were considered to be statistically significant.

## 4.11 ETHICAL ISSUE AND ETHICAL CLEARANCE

An informed consent was given by each participant based on the participant's full understanding of the methods, including its characteristics, actions, and possible risks and benefits. The participants' consent was sought and obtained after adequate information about all aspects covered by the study. During the process of obtaining consent, the rights to decline participation or to withdraw participation at any time of the study should they wish to do so, were emphasized. Information regarding privacy and confidentiality of the patient was provided. It was also ensured that the participants were educated about warning signs and the need for follow- ups.

Ethical clearance for this study was obtained from the Institutional Ethics and Research Committee, KAHER's Dr. Prabhakar Kore Hospital, Belagavi, Karnataka in prescribed format.

# 5. RESULTS

## 5.1 Recruitment of study participants

The total number of mothers screened were 118, 7 were excluded as 5 had twins with macrosomia and 2 had anamolous babies. The total number of participants enrolled for this study was 111. The age of the participants ranged from 19 years to 39 years with mean maternal age 27.03 ± 3.97 years.

## 5.2 Distribution of study participants based on age and obstetrics parameters

The distribution of study participants based on age and obstetric parameters has been given in Table 1.

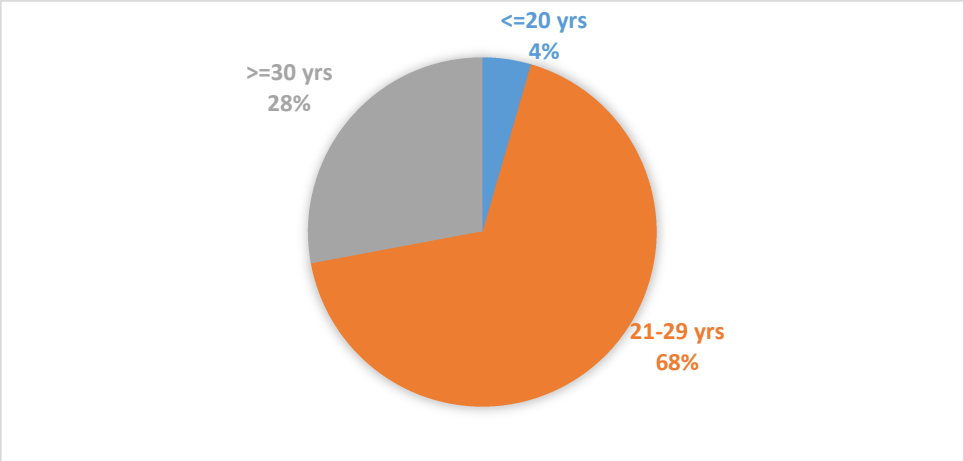
**Table 1:** Distribution of study participants based on age and obstetric parameters

| Sl.No | Parameter | Subgroup | No. of study participants | % of study population |
|---|---|---|---|---|
| 1 | Age | 19-20 years | 05 | 4.5% |
| | | 21-29 years | 75 | 67.6% |
| | | 30-39 years | 31 | 27.9% |

### (1) Age

Maximum number of women who delivered macrosomia babies were in the age group of 21-29 years (67.6%; n=57), followed by the age group of 30-39 years (28%; n=31). The least number of women were in the age group of 19-20 years (4.5%; n=5) (Figure 1).

**Figure 1:** Distribution of women with macrosomia based on age of study participants

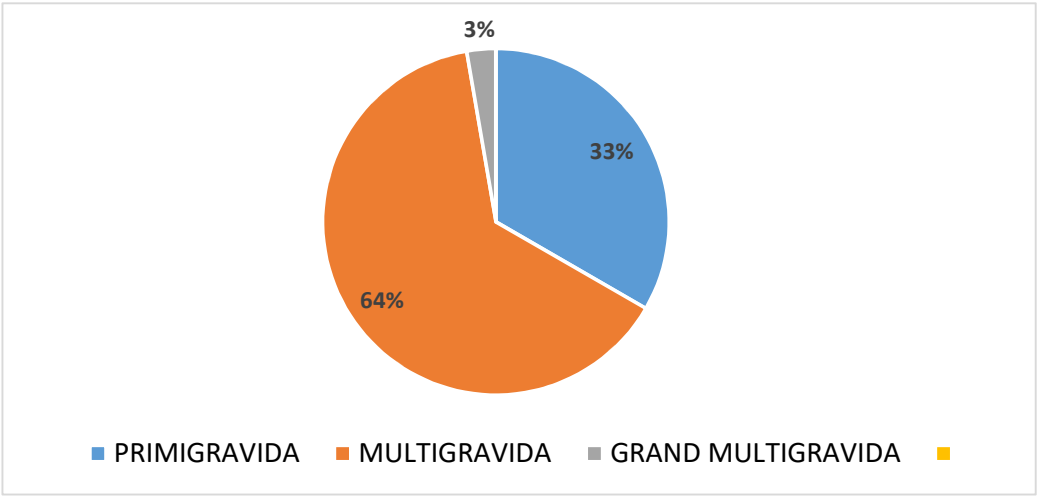



**(2) Gravida**

| | | | | |
|---|---|---|---|---|
| 2 | Gravida | Primigravida | 37 | 33.3% |
| | | Multigravida | 71 | 64% |
| | | Grand multigravida | 03 | 2.7% |

Maximum number of study participants were multigravida (64%; n= 71). 33% of the participants were primigravida (n=37) and only three women were grand multigravida (2.7%) (Figure 2).

**Figure 2:** Distribution of women with macrosomia based on gravida



**(3) Gestational age**

| | | | | |
|---|---|---|---|---|
| 3 | Gestational age | Preterm (35 to 36 wks 6 days) | 29 | 26.1% |
| | | Term (37 to 39 wks 6 days) | 79 | 71.2% |
| | | Post datism (40 to 41 wks 6 days) | 03 | 2.7% |
| | | Post term (> 42 wks) | 0 | 0% |

Maximum proportion of study participants (71.7%) carried the pregnancy to full-term (37 weeks-39 weeks 6 days). 26% of the women with macrosomia babies had preterm deliveries (35 weeks to 36 weeks 6 days) and 2.7% of the study population has deliveries post-due date (40 weeks to 41 weeks 6 days). None of the study participants

had post-term deliveries (Figure 3).

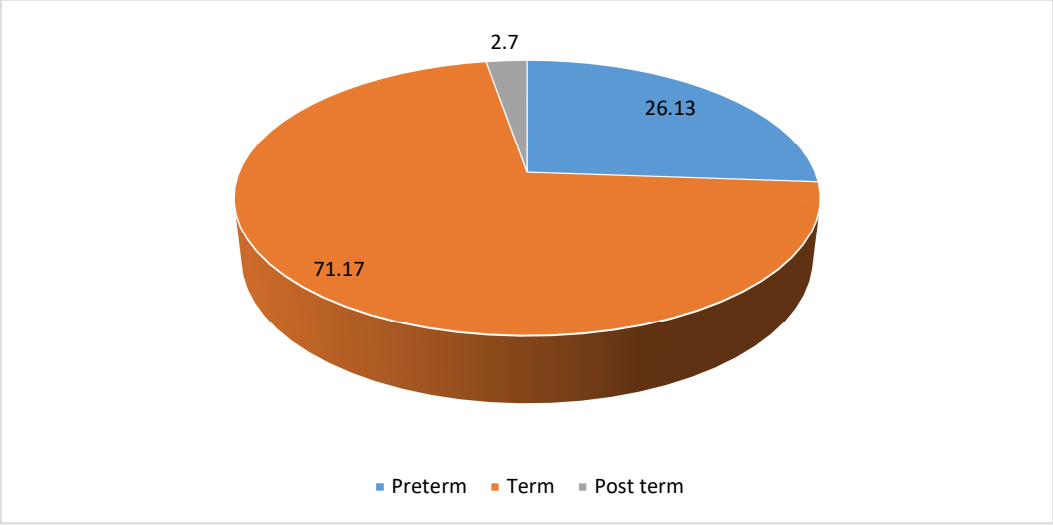



**Figure 3:** Distribution of women with macrosomia based on gestational age

### 5.3 Physical characteristics of the participants

his data was collected from the records of the patients during first trimester, 40.5% (n=45) were observed to be overweight (BMI: 17.6-22.9). Pre-pregnant weight (BMI) of 45% (n=50) of the study participants was in the normal range (< 17.5). Ten study participants were in the obese range (9%). This indicated that proportion of overweight and obese women with macrosomia babies was higher (Table 2 and Figure 4).

**Table 2:** Distribution of study participants based on weight

| Sl.No | Parameter | Subgroup | No. of study participants | % of study population |
|---|---|---|---|---|
| 1 | Pre-pregnant BMI | Normal weight (BMI < 17.5) | 50 | 45 % |
| | | Overweight (BMI between 17.6 to 22.99) | 45 | 40. 54% |
| | | Obese (BMI between 23 to 27.99) | 10 | 9.1% |
| 2 | Weight at delivery | 50 kg-80 kg | 75 | 67.6% |
| | | 81 kg-89 kg | 32 | 28.8% |
| | | 90 kg-99 kg | 4 | 3.6% |
| 3 | Weight gain during pregnancy | 5 kg-10 kg | 45 | 40.5% |
| | | 10 kg-15 kg | 56 | 50.4% |
| | | 16 kg-20 kg | 10 | 9.1% |

67.6% of the women weighed between 50-80 kg during delivery and 28, 2% registered weights between 81-89 kgs at delivery. The proportion of women with weights between 90-99 kgs was 3.6% (Table 2).

Weight gain during pregnancy for 50.4% of the study participants was between 1-15 kg. 40% of the study participants gained 5-10 kgs of weight during pregnancy. Around 9% of the women gained >16 kg during pregnancy (Table 2 and Figure 5).

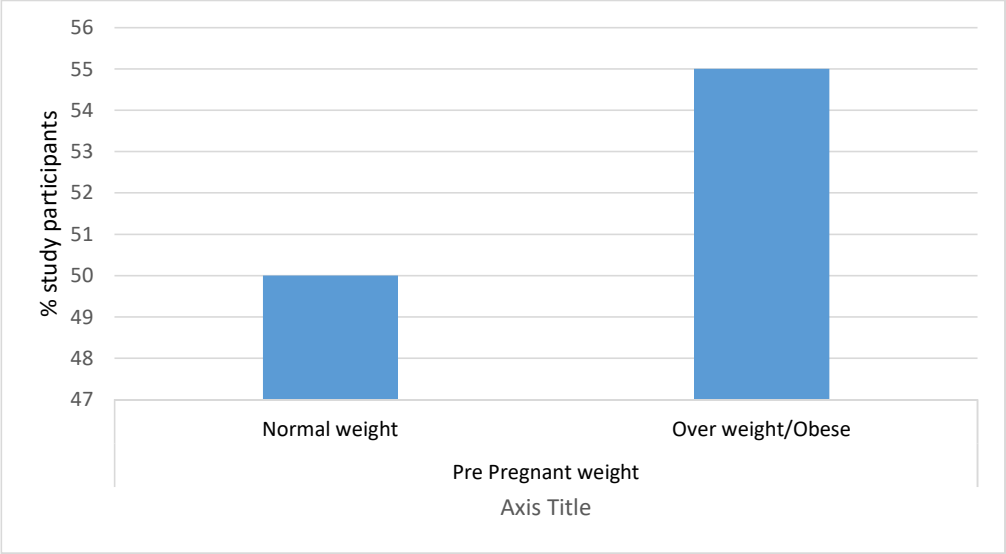



**Figure 4:** Distribution of women based on pre-pregnant weight

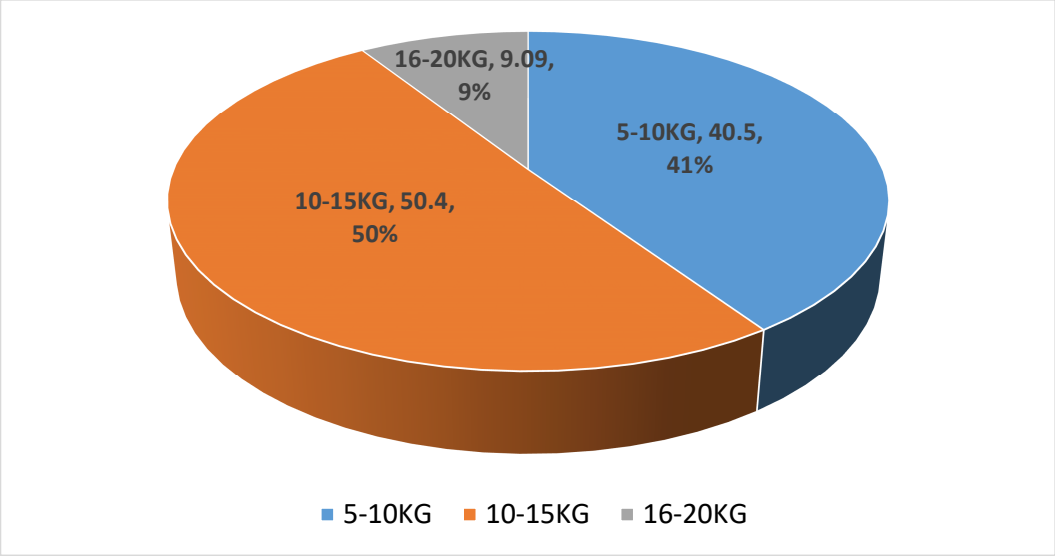



**Figure 5:** Weight gain during pregnancy of women with macrosomia babies

### 5.4 Medical history of study participants

Majority of the study participants (40.5%) reported diabetes mellitus in the history, followed by hypertension (11%). 28% of the study participants reported a previous history of macrosomia. Anemia (8%), hypothyroidism (13.5%) and hyperthyroidism (1%) were some of the other reported medical conditions in the patients' past history (Table 3 and Figure 6).

Among the 45 study participants with diabetes as medical history, 62.2% (n=28) had gestational diabetes, 11 (24%) reported overt diabetes mellitus and 6 women (13%) had impaired glucose tolerance (Table 3). 46.7% of the patients were screened for diabetes by the Diabetes in Pregnancy Study Group of India (DIPSI) method, 42.2% were screened by the oral glucose tolerance test (OGTT) and 8.9% were detected as diabetics by HbA1c levels.

Of these diabetics group 15 (33.3%) of the participants required insulin and rest of them were on medical nutritional therapy, 3 participants had uncontrolled sugars inspite of using insulin

Among the 15 participants who reported hypertension in past medical history, 13 (87%) had gestational hypertension, with 1 woman (6.7%) each reporting mild and severe pre-eclampsia (Table 3).

**Table 3:** Medical history of study participants

| Sl.No | History | Subcategory | No. of study participants | % of study population |
|---|---|---|---|---|
| 1 | Diabetes mellitus | | 45 | 40.54% |
| | | Impaired glucose tolerance | 6 | 5.41% |
| | | Gestational diabetics mellitus | 28 | 25.22% |
| | | Overt diabetics mellitus | 11 | 9.915 |
| 2 | Hypertension | Gestational hypertension | 13 | 12.61% |
| | | Mild PE | 1 | 0.9% |
| | | Severe PE | 1 | 0.9% |
| 3 | Previous history of macrosomia | | 31 | 27.9% |
| 4 | Anemia | | 9 | 8.1% |
| 5 | Hypothyroidism | | 15 | 13.5% |
| 6 | Hyperthyroidism | | 1 | 0.9% |

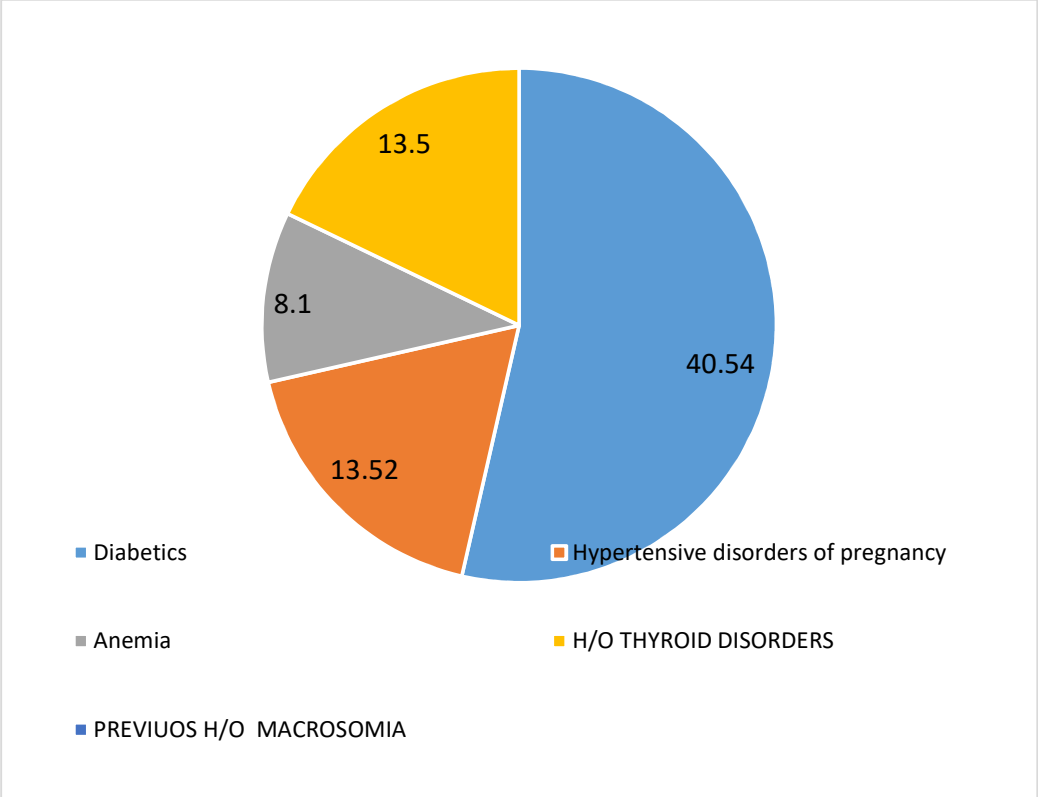
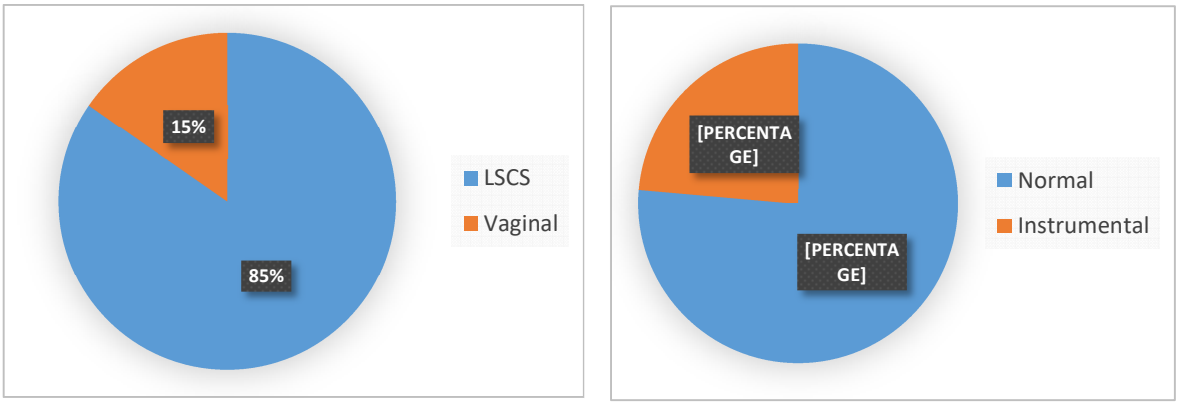



**Figure 6:** Distribution of women with macrosomia babies based on history

### 5.5 Mode of delivery of study participants

Lower segment Cesarean section (*LSCS*) was the most common mode of delivery among study participants, with 94 out of the 111 (85%) study participants delivered by LSCS and 17 women (15%) with normal vaginal deliveries (Figure 7a). For vaginal deliveries, instrumental vaginal deliveries were carried out for 4 patients (24%) (Figure 7b)




(a) (b)

**Figure 7:** Distribution of women with macrosomia babies based on (a) mode of delivery and (b) type of vaginal delivery

### 5.6 Indication for LSCS for study participants

A total of 94 women delivered via LSCS. Previous LSCS With macrosomia was the most common indication of LSCS among the study participants, with 30 women (27.92%) reporting previous LSCS (Table 4). This was followed by fetal distress , Cephalopelvic disproportion (CPD) , failed induction seen in 25.2%, 15.31% , and 13.51% of the women who delivered by LSCS respectively. The other indications were , previous two LSCS (6.3%), breech presentation (4.2%). The less common indications for LSCS were delivery on request [CDMR] (2.1%), precious

pregnancy (2.1%) uncontrolled diabetics (2.1%), anamnios (1.06%) and rheumatic heart disease (1.06%).

| Variables | No of subjects (n=111) |
|---|---|
| Previous LSCS with macrosomia | 30(27.92%) |
| Fetal distress | 28(25.22%) |
| Failed induction | 15(13.51%) |
| CPD | 17(15.31%) |
| Previous 2 LSCS | 6(6.3%) |
| Breech | 5(5.3%) |
| CDMR | 3 (3.1%) |
| Precious pregnancy | 3(3.1%) |
| Uncontrolled diabetics | 2 (2.1%) |
| Anamnios | 1 (1.06%) |
| RHD | 1 (1.06%) |

**5.7 Macrosomia**

Distribution of macrosomia based on percentile values has been given in Figure 9. Maximum number of women delivered were in between 95-97percentile groups

**Association of percentile with delivery outcome**

Highest number of babies in all percentile ranges were born to women with LSCS, followed by induced labor (Table 4). Most of the babies were in the 95-97 percentile groups for all modes of delivery.

**Association of percentile with birth weight**

Highest proportion of women had children in weighing between 3.1-3.6 kg with more children in the 90-94 percentile group for this weight range. Babies with birth weight > 4.0 kg were in the > 95 percentile group (Table 4). For 2.7-3.0 kg weight category, there were no babies in the > 98 percentile group.

**Table 4:** Association of percentile with various parameters

| **Parameter** | **Percentile range for macrosomia** | | | | | |
|---|---|---|---|---|---|---|
| | **90 to 94 percentile range** | | **95 to 97 percentile range** | | **>98 percentile range** | |
| **Delivery outcome** | **N** | **%** | **N** | **%** | **N** | **%** |
| Induced | 14 | 28% | 30 | 60% | 6 | 28% |
| LSCS | 37 | 39.4% | 44 | 46.8% | 13 | 13.8% |
| Vaginal delivery | 4 | 23.5% | 10 | 58.8% | 3 | 17.6% |
| **Birth weight range** | **N** | **%** | **N** | **%** | **N** | **%** |
| 2.7 to 3 kg | 8 | 80% | 2 | 20 | 0 | 0% |
| 3.1 to 3.6 kg | 27 | 62.8% | 15 | 34.9% | 1 | 2.3% |
| 3.7 to 3.9 kg | 9 | 18.7% | 30 | 62.5% | 9 | 18.75% |
| 4.0 to 4.3 kg | 0 | 0% | 4 | 40% | 6 | 60% |

### 5.8 Maternal complications

A total of 47 women had maternal complications due to macrosomia. Postpartum hemorrhage was the most prevalent maternal complication seen in study participants. Atonic postpartum hemorrhage (PPH) was observed in 27 women (24.32% of total study population) followed by traumatic PPH, seen in 10 women (9%), prolonged labour and shoulder dystocia were observed in 18 (16.2%) and 4 (3.6%) of the women respectively. (Figure 8).

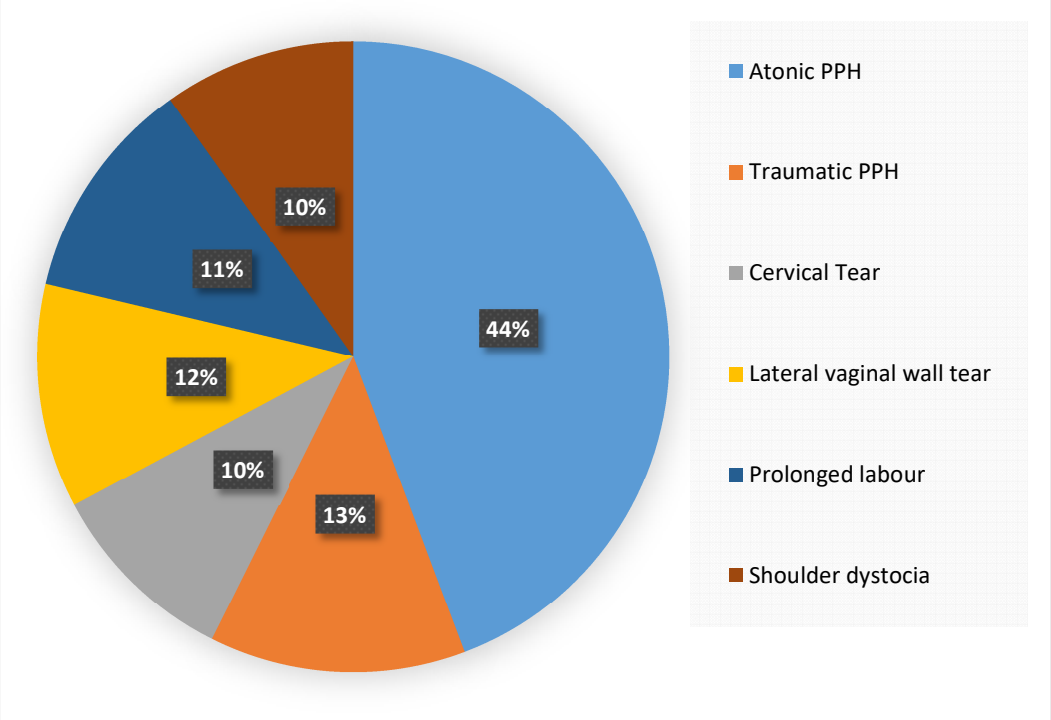



**Figure 8:** Distribution of women with macrosomia babies based on maternal complications

**5.9 Perinatal outcomes**

Of the 111 deliveries of babies with macrosomia, a total of 71 babies (64%) required admission to neonatal ICU. Still birth was observed in two cases (1.8%), both of which were macerated still births (MSB) (Figure 9). These still birth mothers are associated with complications like diabetics with uncontrolled sugar levels.

One mother had HBA1C of 9 with uncontrolled sugar and associated hypertension with BP of 140/90 started on insulin and antihypertensives and patient was induced and delivered vaginally and baby had shoulder dystocia which was managed by McRoberts maneuver and suprapubic pressure

One mother had HBA1C of 9 with uncontrolled sugars came in labour and was found to be MSB and insulin has been titrated according to sugars and delivered vaginally

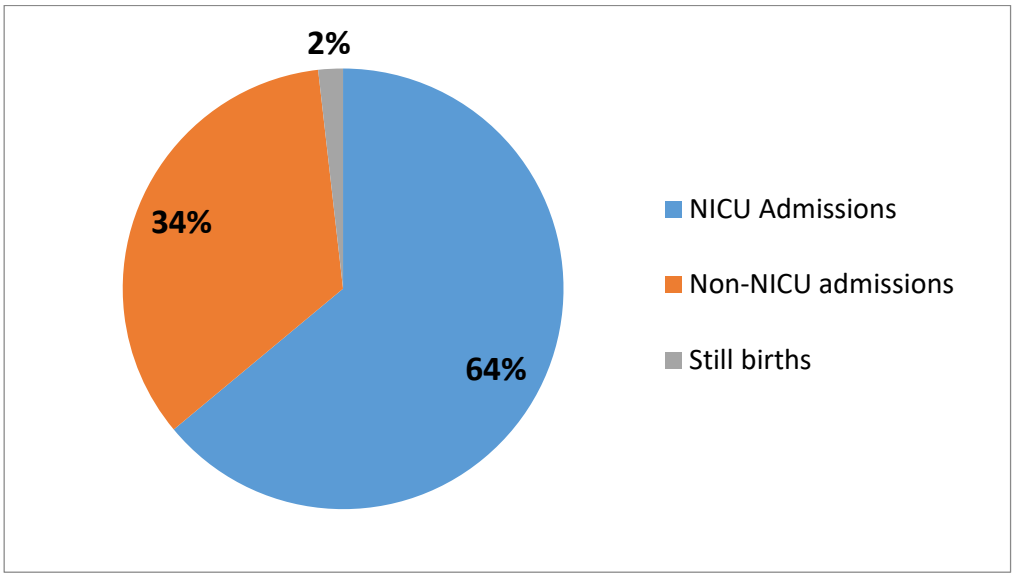



**Figure 9:** Distribution of cases based on perinatal outcomes

Respiratory distress was the most prevalent perinatal complication observed in this study, with babies of 28 women (25.2%) presenting respiratory distress at birth. This was followed by hypoglycemia, seen in 26 cases (23.8%) and hypocalcemia seen

in 16 cases (14.7%). There was only a single case of birth asphyxia (3.4%) and birth trauma was not observed in any of the macrosomia babies (Figure 10).

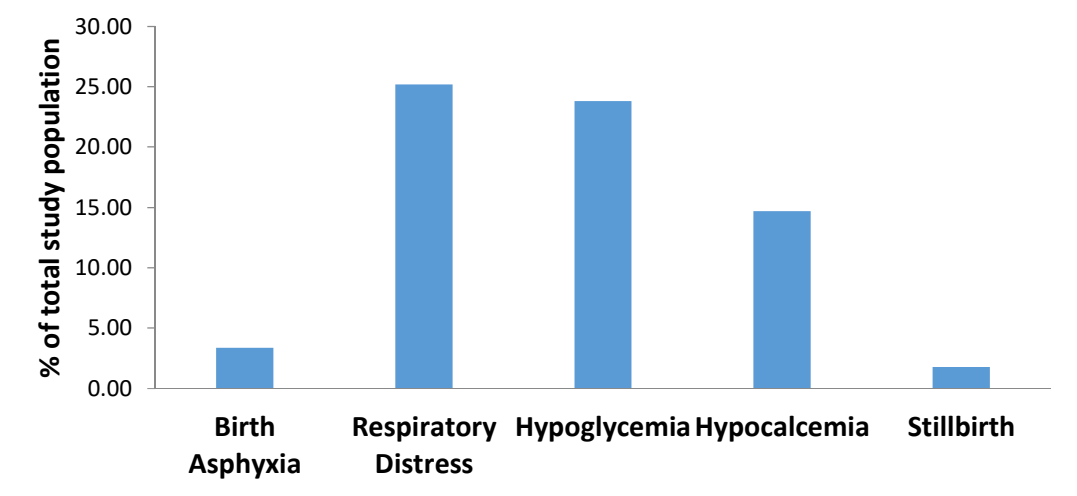



**Figure 10:** Distribution of macrosomia babies based on perinatal complications

**5.10 Indications for NICU admissions (n=71)**

Of the 26 preterm macrosomia babies admitted to NICU, 10 cases (14.08% ) were preterm and 10 cases (14.08% )were preterm with respiratory distress and 6 cases (8.4%) of preterm birth with hypoglycemia was observed. Of the term deliveries admitted to NICU, 21 babies (29.5%) had respiratory distress, followed by hypoglycemia with hypocalcaemia 16 (22.53%), hypoglycemia 7 (9.85%) and one birth asphyxia (1.4%) (Figure 11).

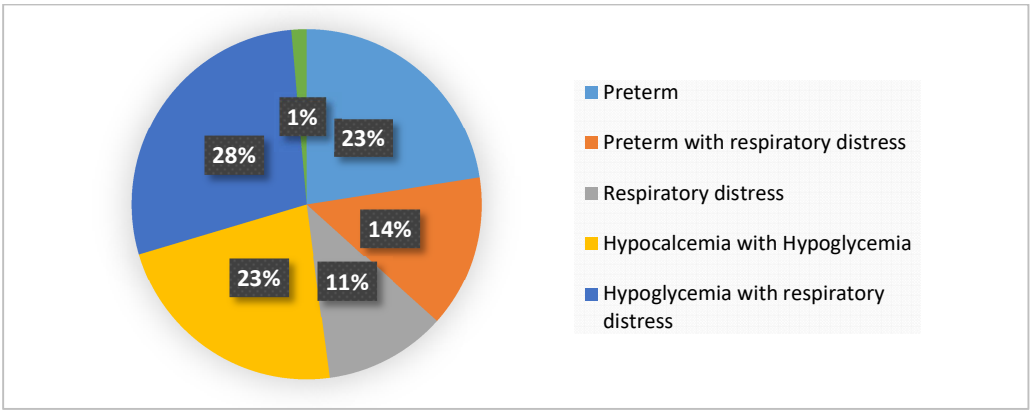



**Figure 11:** Distribution of macrosomia babies based on perinatal complications

### 5.11 Distribution of babies based on birth weight

Maximum babies (48) were observed to be in the weight range of 3.6 kg-3.9 kg (43.2%). 43 babies were in the weight range of 3.1 kg-3.5 kg (38.7%). A total of 10 babies were in the weight range of 4 kg-4.5 kg and 10 babies were in the weight range of 2.7 kg-3 kg (9% each) (Figure 12).

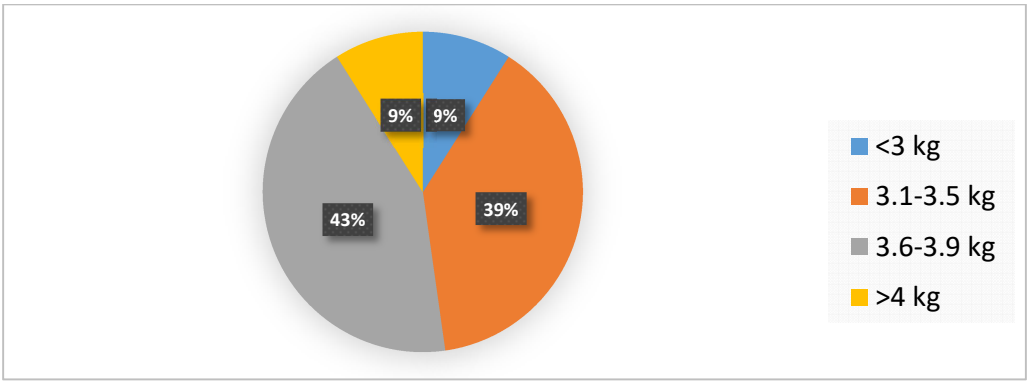



**Figure 12:** Distribution of macrosomia babies based on perinatal complications

There was correlation between weight estimated during scanning and actual birth weight. During scanning 12.6% of the babies were estimated to be over 4 kg and on basis of actual birth weight 9% of the babies were above > 4 kg. Similar correlations were observed for other weight ranges as given in Table 5.

**Table 5:** Correlation between weights obtained via scanning and actual birth weights of babies

| Birth weight | No. of women scanned (n=111) | Actual birth weight estimations in women (n=111) |
|---|---|---|
| 2.7-3KG | 6 (5.4%) | 10 (9.0%) |
| 3.1-3.4 KG | 39 (35.1%) | 43 (38.7%) |
| 3.5- 3.9 KG | 52 (46.8%) | 48 (43.2%) |
| 4KG-4.3kg | 14 (12.6%) | 10 (9.0%) |

# 6. DISCUSSION

Macrosomia, defined as newborns with a birth weight ≥ 4000 g, has grown more common as a result of increased maternal obesity and diabetes[49]. Large-baby pregnancies are linked to a higher risk of difficulties for both the babies and mothers. Overweight babies can have both acute and long-term implications, the incidence of these negative outcomes and associated risk factors, such as an increased risk of neonatal mortality, birth injury, obesity, cardiovascular disease, and malignancies later in life have been extensively investigated in previous studies[1,50]. The present study was taken up with the objective to determine macrosomia associated maternal and perinatal outcome, and to assess the risk factors and incidence of fetal macrosomia in the study population.

The present study was a hospital based observational study conducted at KAHER's Dr. Prabhakar Kore Hospital, Belagavi for a period of 12 months. Data on demographic and clinical parameters was collected from 111 women who delivered macrosomia babies in form of detailed proforma after obtaining written consent from the participants of the study. Patients who expressed an interest in taking part in the trial were enrolled after signing a written informed consent form. Statistical tools were employed to understand the correlation between various clinical and sociodemographic factors with macrosomia and to elucidate the risk factors for macrosomia in Indian women.

The age of the participants ranged from 19 years to 39 years with mean maternal age 27.03 ± 3.97 years. Most of the study participants were multigravida (64%). Maximum macrosomia babies were born to women aged between 21-29 years. In the 30-39 years age group, 37% of women had macrosomic neonates. Maternal age has been indicated as an independent risk factor for macrosomia with average age of

29 years[51]. in previous studies, which correlates with findings of this study. Metabolic changes occurring in the woman's body with increase in age have been reported to be important factors for babies born with higher birth weights[52,53]. A recently published meta-analysis also found that the odds of having macrosomic babies increases with advanced maternal age[54], and this has been established from findings of this study.

| AUTHORS | YEAR | MATERNAL AGE |
|---|---|---|
| Yi Li et al | 2015 | 29.22 (61%) |
| Aisha salim said et al | 2016 | 30.11(62%) |
| Shanshan Wang et al | 2020 | 29 (60%) |
| Gabkika Bray Madoue et al | 2017 | 32 (58%) |
| Present study | 2020 | 29 (67%) |

Gestational age for highest proportion of macrosomia babies was between 37 weeks to 39 weeks, with > 70% of the woman in that gestational age group. In studies by Terada et al., the gestational age was found to be the most important factor affecting weights of singleton babies[51]. Prolonged pregnancy beyond the intended delivery date has been linked to a higher risk of macrosomia, as fetuses gain 150–200 gm each week near term[34,52,55,56]. Macrosomia accounted for 3–10% of post-term deliveries, with advanced gestational age resulting in a higher birth weight[52]. However, in this study, there were no post term deliveries (> 44 weeks), but 2.7% of the women delivered post due date (> 40 weeks), which is similar to the previous study. Macrosomia was relatively lower in pre-term birth in this study.

| AUTHORS | YEAR | GESTATIONAL AGE |
|---|---|---|
| Ai koyanagi et al | 2013 | 40.1+- 2.5 |
| Hong Ju et al | 2009 | 40 +- 2.5 |
| Gabkika Bray Madoue et al | 2017 | 41,7+_2,5 |
| Shanshan Wang et al | 2020 | 39+-2.5 |
| Present study | 2020 | 39+-2.5 |

Compared to nulliparous women, primiparous and multiparous women had higher rates of macrosomia in this study. Since birth weight increases with parity, multiparity has been linked to macrosomia, with rates of macrosomia 2–3 times greater than women without this risk factor[34,52]. Similar findings have been obtained in this study, which established multiparity as one of the factors for macrosomia.

| AUTHORS | YEAR | PARITY |
|---|---|---|
| Hong Ju et al | 2009 | >3 |
| Gabkika Bray Madoue et al | 2017 | >3 |
| Lili Zhang et al | 2020 | >2 |
| Present study | 2020 | >1 |

There is an important correlation between maternal weight and overall weight gain during pregnancy with macrosomia. Maternal obesity has been identified as an independent risk factor for infant macrosomia in several studies[50,57–59]. Pre-gestational BMI has been shown to influence foetal growth[54,60,61]. Obese mothers were more likely to give birth to macrosomic

Koyanagi et al. found that BMI raised the incidence of macrosomia considerably[34].

| AUTHORS | YEAR | PRE PREGNANT BMI |
|---|---|---|
| Salvatore Alberico et al | 2014 | OVERWEIGHT(10.4%)<br>OBESE(15,7%) |
| Yi Li et al | 2015 | OVERWEIGHT (8.55%)<br>OBESE (8.55%) |
| Nianhong Yang et al | 2021 | OVERWEIGHT (11%)<br>OBESE(5%) |
| Jimena pereda et al | 2020 | OVERWEIGHT (20.9%)<br>OBESE (10.7%) |
| Present study | 2020 | OVERWEIGHT (40.5%)<br>OBESE (9.1%) |

Increased insulin resistance, which leads to increased hepatic glucose production and high foetal glucose and insulin concentrations, may contribute to macrosomia in pregnant women[62]. In 43,705 Norwegian women, Fleten et al. discovered a direct link between pre-pregnancy BMI and birthweight, with a 20.3-g increase in birthweight for every one-unit rise in BMI[63] (Fleten). In addition, a systematic review and meta-analysis of 31 studies including 1,443,499 women found that maternal obesity is linked to foetal overgrowth, with a greater risk of delivering a newborn weighing less than 4000 grams[12]. In this study, women in the over-weight and obese category of pre-pregnant BMI constituted the larger proportion of mothers with macrosomia babies, making pre-pregnant weight an important factor for predisposition towards macrosomia.

Maternal weight gain, regardless of pre-pregnancy BMI, is a well-known risk factor for macrosomia[61,64]. In a systematic review and meta-analysis of over 1 million pregnant women, 47% percent had gestational weight gain that exceeded Institute of Medicine criteria. The report suggested that gestational weight gain greater than recommended weight guidelines was linked to a higher risk of unfavourable mother and baby outcomes[65]. In another cohort study on women with gestational diabetes, it was established that weight gain during pregnancy and maternal pre-pregnancy weight appear to be major and independent risk factors for macrosomia in women with gestational diabetes[66].

| AUTHORS | YEAR | MATERNALWEIGHT GAIN |
|---|---|---|
| Salvatore Alberico et al | 2014 | .14 KG +_4 |
| YI LI et al | 2015 | 19 KG +-4 |
| Chaoqing Tian et al | 2015 | 14 KG +-4 |
| Present study | 2020 | 13KG +-2 |

The association of gestational weight gain with increased risk for macrosomia has been also found in a meta-analysis of 15 studies conducted by Tian et al.[64] In this study, it was observed that the proposition of women macrosomia babies increased with increase in gestational age from 5 kg to 15 kg, with 50% of the macrosomia cases noted for women who gained 10-15 kgs additional weight during pregnancy. In this study, out of 111 macrosomia cases, 10 women had gained > 16 kg during pregnancy. Along with pre-pregnancy weight, gestational weight again appeared to be a factor for macrosomia in this cohort study.

Multiparity has an effect on the occurrence of macrosomia, according to results obtained in this study. In fact, women with parity ≥3 were four times more likely than those with parity < 3 to have macrosomic kids. This could be owing to the fact that birth weight increases with parity in the same woman. In their series, Abena et al. found that having more than five children was a risk factor[67].

Gestational diabetes mellitus (GDM), defined as glucose intolerance that begins or manifests during pregnancy, is one of the most frequent pregnancy problems, affecting 2–10% of all pregnancies in the United States[68].

GDM prevalence in urban China has risen from 2.3% in 1999 to 8.1 percent in 2010[7,69]. According to several research, offspring of mothers with GDM had a higher risk of neonatal adiposity and childhood obesity[70] and women with GDM have a higher risk of hypertensive problems during pregnancy[71].

In a meta-analysis, gestational diabetes was found to be an independent risk factor for macrosomia[72]. Because fasting plasma glucose levels rise from early to late pregnancy, pre-gestational diabetes carries a larger risk of macrosomia than gestational diabetes, according to our findings[73]. Hyperglycemia in the mother causes hyperglycemia in the fetus, as well as pancreatic beta cell hyperplasia and hyperinsulinism, resulting in fat accumulation and rapid foetal growth[44]. There was a link between increased maternal hyperglycemia and excessive newborn obesity in the HAPO research[74].

Obesity is a known risk factor for both PGD and GD. Macrosomia, as well as overweight/obesity at a young age and type 2 diabetes in the kids, are known risk factors for diabetes and an elevated maternal BMI[75,76]. This could be a vicious cycle, as obese offspring may pass on an aberrant metabolic environment to their children while they are still in the womb[59,75]. According to Hillier et al., an overfed state

caused by extra glucose or overall calories may imprint the child's metabolism for an overfed state[76]. In the absence of ongoing environmental stressors, developmental programming may have an impact on succeeding generations, resulting in a generational transfer of obesity[77]. As a result, it has the potential to prolong a cycle of metabolic problems and obesity.

The prevalence of gestational diabetes in this study was 62.2%, greater than the expected 17% using the updated diagnostic IADPSG criteria adopted in 2010. Diabetes was found in 46.7% percent of the patients using the Diabetes in Pregnancy Study Group of India (DIPSI) technique, 42.2 percent using the oral glucose tolerance test (OGTT), and 8.9 percent using HbA1c values. Between 2005 and 2015, a review of the global prevalence of GD revealed that the Middle East and North Africa had the highest prevalence, with a median estimate of 12.9%, followed by Southeast Asia (11.7%), Western Pacific (11.7%), South and Central America (11.2%), Africa (8.9%), and North America and the Caribbean (8.9%). With a median of 5.8%, Europe had the lowest prevalence[78]. The Middle East and North Africa had a median prevalence of 15.2%, 15.0% in Southeast Asia, 10.3% in the Western Pacific, 11.2% in South and Central America, 7.0% in North America and the Caribbean, and 6.1% in Europe, according to an update of the previous review that included studies from 2015 to 2018[79]. Previous research has found that considering some GDM management criteria, such as detecting, diagnosing, and treating pregnant women with GDM using unified methodology and standard protocol for diabetes screening, can minimize the chance of developing macrosomia. Pregnancy appropriate medications and non-pharmaceutical therapies can also be recommended to the patients.

| AUTHORS | YEAR | DIABETIC |
|---|---|---|
| Kamana K C et al | 2015 | 10.1% |
| Ulla kampmann et al | 2015 | 11.2% |
| Thomas A Buchanan et al | 2015 | 32.1% |
| Kaiwei Lee et al | 2018 | 11.5% |
| Robyn l Lawrence et al | 2019 | 16,2% |
| H.David mclntyre et al | 2020 | 30.5 % |
| Present study | 2020 | 40.5 % |

In this study, 11% of the women had hypertension as medical history. Association of hypertensive disorders during pregnancy with macrosomia has been established in previous studies[7]. In Tianjin, China, a cross-sectional study conducted on a large cohort of mothers with hypertensive disorders and neonates showed that maternal hypertension was linked to a two-fold higher risk of delivery of larger neonates[7]. This risk is higher for applicable to mothers who have hypertension along with gestational diabetes and pre-delivery obesity.

Previous history of macrosomia was found to be another factor for macrosomia in this study with 28% women reporting a previous macrosomic baby. In a cross sectional analytical study, it was found that the risk of macrosomia was 15 times higher with previous history of macrosomia[26].This was also confirmed by the findings of Kamanu et al. who reported that a previous history of macrosomic baby delivery was a major risk factor for macrosomia in subsequent pregnancies[10].

| AUTHOR | YEAR | LSCS RATE |
|---|---|---|
| Yi Li et al | 2015 | 82.91% |
| Jiun How Lim et al | 2012 | 54% |
| Sahruh Turkmen et al | 2018 | 28% |
| Mahin Najafian et al | 2012 | 86% |
| Present study | 2020 | 85 5 |

Maternal complications such as postpartum hemorrhage (PPH), perineal trauma, and neonatal complications such as shoulder dystocia, obstetric brachial plexus injury (OBPI), birth fracture of the humerus or clavicle, and birth asphyxia have been linked to fetal macrosomia in previous studies[11,44,53,69]. In this study, majority of the women underwent LSCS for delivery. Macrosomia is linked to a greater rate of Caesarean section deliveries (twice as often as the control group) and birth canal lacerations during vaginal delivery[16,49] (Gabkika, Voldner) This was validated in this study, with an 84% Caesarean section rate against 16% in the vaginal group. With rising birth weight, the chance of Caesarean section increases, whereas the proportion of vaginal instrumental deliveries drops[8,36]. The higher Caesarean section rate has been observed in a variety of countries and ethnic groups, with the odds being especially high for primiparous. Similar finding has been observed in this study also.

Along with increased incidence of LSCS, several other complications such as atonic postpartum hemorrhage (PPH) in 27 women (24.32% of total study population) followed by traumatic PPH, seen in 10 women (9%), cervical tear, lateral vaginal wall tear, prolonged labour and shoulder dystocia were observed in the study population.

Since macrosomia is most typically identified during atypical labour, maternal problems related to macrosomia may arise during emergency CS. As a result, the foetal head is already fully engaged at the time of LSCS. This gives rise to PPH. In this study, PPH was the most prevalent maternal complication arising due to macrosomia. Macrosomia is further complicated by PPH due uterine atony and genital lacerations. Out of 27 women who had atonic PPH, 6 delivered vaginally 21 women delivered by LSCS out of which 20 participants required additional uterotonics like methergin and carboprost and 6 patients are managed with haymans sutures and 3 were managed with Blynch.

In traumatic PPH group out of 10 women 4 had cervical tears and 4 had lateral vaginal wall tear 2 had both cervical and lateral vaginal wall tears.

Out of 37 participants who had PPH around 15 patients required blood transfusion.

However, PPH can be adequately controlled by meticulously following the WHO guidelines

Shoulder dystocia, which is linked to birth trauma, is one of the most significant consequences of vaginal delivery in macrosomic newborns. According to current research, having a larger baby increases the chance of shoulder dystocia and irreversible brachial plexus injury. Shoulder dystocia occurred in 3.6% of the cases in this study, which is lower to the 10.5% described by Esakoff et al.[39] or 9.6% described by Alsammani et al. 2012[80]. According to Langer et al., 76% of occurrences of shoulder dystocia can be avoided if the rate of CS is increased by 2.6%[81]. However, because most macrosomic newborns are diagnosed retrospectively, this finding is difficult to put into effect. Incidence of shoulder dystocia due to macrosomia be reduced by accurate clinically or biometrical estimation of foetal

weight prior to delivery. Out of 4 participants who had shoulder dystocia their birth weights are ranging between 3.8 kg -4.2kg and they were managed by McRoberts maneuver and suprapubic pressure. out of which one was IUD and 1 baby has been shifted to NICU i/v/o grunting and other 2 babies are with mother none of the babies had injuries

| AUTHOR | YEAR | SHOULDER DYSTOCIA |
|---|---|---|
| Katherine A volpe et al | 2012 | 2% |
| Brett c young et al | 2012 | 3.4% |
| Meghan G Hill et al | 2016 | 2% |
| Karin heinonen et al | 2020 | 0.18% |
| Present study | 2020 | 3.6% |

Prolonged labour was observed in 16.2% of the study participants as one of the maternal complications due macrosomia. . Compared to delivery of non-macrosomic babies, the first and second stages of labour are both lengthier in case of macrosomic pregnancies. In some cases, arrest of descent in the second stage might has been reported to occur as a result of macrosomia[33]. The likelihood of shoulder dystocia is 1.2 times higher in macrosomic infants weighing more than 4.5 kg when the second stage lasts longer than 2 hours, as seen in one study[46]. The incidence of prolonged labour with macrosomic babies weighing > 4.5 kg is higher in case of primigravidae as compared to multiparous women. Other maternal complications like chance of LSCS and PPH are often associated with prolonged labour Out of 6 participants who had prolonged labour, 4 had cephalo pelvic disproportion and 1

patent had cervical dystocia who underwent LSCS with birth weights are ranging between 3.5-3.8kg and 1 participant had inadequate uterine contractions for which labour was augmented with oxytocin and patient delivered vaginal by ventouse.

| AUTHOR | YEAR | PROLONGED LANOUR | PERINEAL TEARS | PPH |
|---|---|---|---|---|
| J Beta et al | 2019 | 16.2% | 20% | 22.25 |
| Cheng YK et al | 2013 | 12,1% | 21.2% | 25.3% |
| Aisha salim said | 2016 | 19.2% | 19.6% | 23.2% |
| Mehan G hill et al | 2016 | 13.6% | 25.2% | 24.2% |
| Present study | 2020 | 16.2% | 22% | 24.3% |

Cervical and lateral vaginal tears were noted on 12% of this study participants. Studies have shown that the incidence of perineal tears increases 1.5- to 2-fold in case of macrosomia as compared to non-macrosomic deliveries[82,83]. The risk of serious perineal tears increasing with increased birth weight has been suggested by some researchers[84] (King 20), but has not been proven[85]. Ethnicity plays an important role in appearance of vaginal tears and Asian, Filipino, and Indian women appear to be at a higher risk compared to Caucasian women for this maternal complication of macrosomia[86]. Differences in body type and perineal architecture possibly accounts for this ethnic disparities. Major perineal damage, such as a 3$^{rd}$ or 4$^{th}$ -degree vaginal

rip, can result in significant long-term anal incontinence, lowering a woman's quality of life. However, none of the participants reported 3rd degree tear in this study.

Birth asphyxia, birth trauma, and hypoglycemia are some of the complications seen in macrosomic neonates[7,10,30,53,59]. Infants born to diabetes mothers are more likely to have polycythemia and hypocalcemia. These infants may also be at a higher risk of obesity and diabetes later in life[47,69]. A larger proportion of macrosomic babies (64%) required NICU admission right after birth owing to complications. Respiratory distress was the most prevalent fetal complication seen in this study, which was seen in 25% of the neonates admitted to NICU. Obstructed labor and fetal distress are possibly the most common cause for respiratory distress in macrosomic children. Since majority of the babies were delivered by C-section, it can be suggested that respiratory distress was higher among neonates born via C-section. In the macrosomia group, Das et al. discovered a comparable high rate of respiratory distress, which they explain to the effect of greater caesarean births and maternal diabetes on lung maturity[87]. As a result, the decision to deliver macrosomic babies through LSCS may need to be balanced against the risks of respiratory distress and higher neonatal unit hospitalizations.

Hypoglycemia was observed in 23% of the macrosomic was higher than what Oral et al.[45]. In contrast to previous studies findings, there was no link between hypoglycemia and maternal diabetes or a birth weight > 4.5 kg. The fact that infants delivered to non-diabetic mothers also presented with hypoglycemia, even if it is not substantial. This could be attributed to modest changes in maternal glucose metabolism, implying that macrosomic newborns, independent of maternal diabetes, require strict monitoring for hypoglycemia. The majority of hypoglycemia episodes occurred in infants born via LSCS, which is as expected. Neonatal mortality observed

in this study was 1.8% and was attributed to macerated still births. These macerated still births are associated with complications like diabetics with uncontrolled sugars.

| Author | Year | Birth asphyxia | Respiratory distress | hypoglycemia | hypocalcemia | Birth trauma | death |
|---|---|---|---|---|---|---|---|
| Aisha salim said et al | 2016 | 14.4% | 16.5% | 22.7% | 1% | 14.4% | 9.7% |
| Alina Weissmann et al | 2012 | 3% | 22,1% | 24.6% | 20% | 2% | 1% |
| Mahin Najafian et al | 2012 | 4% | 23.1% | 25,2% | 22,2% | 2.45 | 0 |
| Subash chandra shah et al | 2021 | 1.2% | 19.4% | 1.2% | 2.4% | 0 | 0 |
| Present study | 2020 | 3.4% | 25.2% | 23.8% | 14.7% | 0 | 0 |

# CONCLUSION

In obstetrics, foetal macrosomia is prevalent, causing complications for both the mother and the infant. It has been linked to an increased risk of morbidity and mortality. The global trend in foetal macrosomia has been observed to be growing throughout the years. Till date, there are no accurate perinatal diagnostic methods for macrosomia. Hence, assessment of risk factors is important for early detection and management of unfavourable maternal and fetal outcomes due to macrosomia.

The key conclusions of this study are as follows:

(i) Pre-pregnant weight, gestational diabetes and previous history of macrosomia are important factors for fetal macrosomia.

(ii) Incidence of spontaneous deliveries are lower in case of macrosomia, however, rates of LSCS and labour induction are higher.

(iii) Post-partum hemorrhage and prolonged labour are the most prevalent maternal complications seen in macrosomia.

(iv) Macrosomia is associated with NICU admissions in neonates.

(V) Respiratory distress and prematurity are the major negative fetal outcomes seen in macrosomic neonates

## SUMMARY

The present study was a cross-sectional study conducted to determine macrosomia-associated maternal and fetal outcomes and to assess the risk factors of fetal macrosomia. The study was carried out at the Department of Obstetrics and Gynaecology of KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi, Karnataka for a period of one year and six months. Patient population included women who delivered macrosomia babies in labour room from January 2020 to July 2021. A total of 111 women were selected for enrollment based on inclusion criteria. Data regarding sociodemographic and clinical characteristics was collected in form of structured questionnaires and analyzed statistically.

Key findings of this study have been summarized as follows:

- The age of the participants ranged from 19 years to 39 years with mean maternal age 27.03 ± 3.97 years. Maximum number of women who delivered macrosomia babies were in the age group of 21-29 years (67.6%; n=57).
- More multigravidae (64%) had macrosomic deliveries compared to primigravidae (37%) in this study
- Maximum proportion of study participants (71.7%) carried the pregnancy to full-term (37 weeks-39 weeks 6 days) and only 2.7% of the study population has deliveries post-due date (40 weeks to 41 weeks 6 days). None of the study participants had post-term deliveries.
- Proportion of women with higher pre-pregnant weight (overweight and obese) was higher compared to women with lower BMI. Pre-pregnant weight is an important determinant of macrosomia.

- Weight gain during pregnancy for 50.4% of the study participants was between 1-15 kg. 40% of the study participants gained 5-10 kgs of weight during pregnancy. Around 9% of the women gained >16 kg during pregnancy.
- Presence of gestational diabetes, history of hypertension and previous macrosomic births are important factors for predisposition to macrosomia. In this study, among the 45 study participants with diabetes as medical history, 62.2% (n=28) had gestational diabetes, 11 (24%) reported overt diabetes mellitus and 6 women (13%) had impaired glucose tolerance.
- Most of the deliveries of macrosomic neonates was by LSCS with less number of normal vaginal deliveries. Previous LSCS was the most common indication of LSCS among the study participants, with 30 women (32%) reporting previous LSCS
- Maximum number of women delivered macrosomia babies were in the 95-97 percentile range (46%; n=51). 40% of the babies were in the range of 90-94 and 14.4% of the woman delivered babies in the >98 percentile range.
- Postpartum hemorrhage was the most prevalent maternal complication seen in study participants. .
- A total of 71 babies (64%) required admission to neonatal ICU. Still birth was observed in two cases (1.8%), both of which were macerated still births (MSB).
- Respiratory distress was the most prevalent perinatal complication observed in this study, followed by hypoglycemia and hypocalcemia. There was only a single case of birth asphyxia (3.4%) and birth trauma was not observed in any of the macrosomia babies. Preterm births were around 28%, with >80% of neonates needing NICU admission.

**LIMITATIONS OF THE STUDY**

Following are limitations of this study:

- This study was conducted in a tertiary center therefore the findings may not adequately reflect the entire Belagavi region.
- Conclusions and deductions from this study cannot be assertive because of the small sample size.
- The study did not consider the socioeconomic status of participants, such as income, living standards, nutritional status among others, which could be key determinants of fetal macrosomia.

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# ANNEXURE - I - ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
**JAWAHARLAL NEHRU MEDICAL COLLEGE,**
**NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)**
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

**Ref: MDC/DOME/** 189 **Date: 24/12/2019**

To,

**REG. NO. BJ0119005**
PG student in Obstetrics and Gynecology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled " **FETAL MACROSOMIA –RISK FACTORS, MATERNAL AND PERINATAL OUTCOME – A ONE YEAR CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

**(Dr. Anita Dalal)**
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

**(Dr. Roopa M Bellad)**
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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# ANEXXURE II- CONSENT FORM

## CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Mrs. _____ we are requesting you to enroll yourself in study title is conducted by **REG. NO. BJ0119005**, Post Graduate in M.S. Obstetrics and Gynaecology under the guidance of **DR._____**, Department of Obstetrics and Gynaecology, J.N. Medical College, Belgaum under KLE university, Belgaum.

**Objectives /purpose of study:**

Respected Madam we request you to participate in our study as you are eligible for participating and your participation in this study is important as it helps us to know the factors associated with macrosomia and its risk reduction in future pregnancies

Your participation in research is voluntary. Your decision whether to participate in the study or not will not change present or future health care services offered to you and will not affect your relationship with J.N. Medical College. If you decide to participate you are free to withdraw at any time. All pregnant women meeting the inclusion criteria will be recruited in our study.

**Procedure Involved:**

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history, then you will be clinically examined in detail and investigated which may or may not cause pain. The procedures don't cause any temporary or lasting problems to you. Your co-operation is necessary as the investigation may be repeated number of times as required.

**Risks and Benefits:**

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is your participation being valuable contribution to medical research to improvise treatment currently practiced.

**Alternative:**

There are no other options of treatment. If you decline to participate it will affect the results of our study and you will get the routine line of management. You will be informed about any new information that may affect your decision to participate in the study.

**Withdrawal from study:**

You can withdraw at any time from the study. There will be no penalty for withdrawal. You can be removed from the study if necessary.

**Privacy and Confidentiality:**

The only people who will know that you are the research subject will be the members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission except:

1. In emergency to protect your rights and welfare.
2. If required by law.

**Institutional/sponsor's policy:**

In the event of any injury related to the study, treatment will be made available through KLE's Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. If you are injured you may contact **REG. NO. BJ0119005** Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital& MRC or by Ph. No: _____.

**Financial Incentives for participation:**

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator. You will not be reimbursed for any expenses for participation in this research.

**Contact details:**

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact **REG. NO. BJ0119005** Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital and MRC, Ph. No:_____.

**Authorization to Publish Results:**

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential. Results of the study will be used to improve maternal and perinatal outcome.

**Consent statement:**

I, _____ voluntarily agree for participating in this study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read form in my own vernacular language, including the risks and the benefits and having all my questions answered.

Participant Name : _____

Signature or the Left Thumb Print of Participant : _____

Investigators Name: _____ Signature: _____

**Witness Name : _____ Signature: _____**

Date: _____

# ANNEXURE III – PROFORMA

| | |
|---|---|
| Name | |
| Age | |
| Address | |
| | |
| Phone number | |
| | |
| Patient No: | |

**History**

Present risk factors in mother

| | |
|---|---|
| Age of the mother | |
| Family size | |
| Physical exercise in a day | |
| Pregnancy interval (years) | |
| Prepregnant BMI | |
| Total pregnancy weight gain | |
| Fetal macrosomia in previous pregnancy | |
| Diabetes | |
| Hypertension | |
| Thyroid disorder | |
| Anemia | |

Obstetric history:

| | |
|---|---|
| Gravida | |
| Para | |

| | |
|---|---|
| Living | |
| Abortion | |
| Spontaneous | Induced |
| | |

**Menstural H/o**

| | |
|---|---|
| | |
| LMP | |
| EDD | |
| Period of gestation | |
| Preterm | |
| Postterm | |

**GENERAL PHYSICAL EXAMINATION**

| | |
|---|---|
| Pulse rate | |
| Blood pressure | |
| Weighr (prepregnant) | |
| Height(prepregnant) | |
| BMI (prepregnant) | |
| Total weight gain during pregnancy | |
| Pallor | |
| oedema | |

**Perabdominal examination**

| | |
|---|---|
| Symphysiofundal height | |
| Estimated fetal weight | |

**Investigations-**

| | |
|---|---|
| Date | |
| Pt. VCTC | |
| Pt. HBSAG | |
| Blood grouping & Rh typing | |
| Hemoglobin | |
| Urine routine & Microscopy | |
| DIPSI | |
| HBA1C | |
| PIH Profile | |
| Thyroid profile | |

OBSTETRIC ULTRASOUND

| | |
|---|---|
| Date of dating scan | |
| Crown rump length | |
| Gestational age (weeks) | |
| Expected date of delivery | |
| Corrected EDD | |

Growth scan characteristics

| | |
|---|---|
| Gestational age | |
| BPD | |
| HC | |
| AC (centile value) | |
| FL | |
| Estimated fetal weight | |

Neonatal characteristics

| | |
|---|---|
| Birth weight , g | |
| Gender of fetus | |
| Male | |
| Female | |

MATERNAL RISK FACTORS

| | | | | |
|---|---|---|---|---|
| Weight at delivery | <80 | 80-90 | >.90 | |
| | | | | |
| | | | | |
| Pre pregnant BMI | Underweight | Normal weight | Over weight | obese |
| | | | | |
| | | | | |
| Age | <20 | 20-30 | 30-40 | >40 |
| | | | | |
| | | | | |
| Gravida | 0-1 | 2-4 | >5 | |
| | | | | |
| | | | | |
| Weight gain during pregnancy | <,16kg | >16kg | | |
| | | | | |
| | | | | |
| Previous macrosomia | Yes | NO | | |
| | | | | |
| | | | | |

| Diabetics | Overt DM | GDM | No | |
|---|---|---|---|---|
| | | | | |

Mode of delivery

| | |
|---|---|
| Vaginal delivery | |
| Spontaneous vaginal delivery | |
| Assisted breech delivery | |
| Vacuum extraction | |
| Forceps delivery | |
| Caesarean section | |
| Elective | |
| Emergency | |
| Indication | |

Maternal Complication

| | YES | NO |
|---|---|---|
| Atonic PPH | | |
| Traumatic PPH | | |
| Cervical tear | | |
| Lateral vaginal wall tears | | |
| 3rd degree perineal tear | | |
| Uterine rupture | | |
| Prolonged labour | | |
| Shoulder dystocia | | |

Neonatal Complication

| | YES | NO |
|---|---|---|
| Birth asphyxia | | |
| Respiratory distress | | |
| Hypoglycemia | | |
| Hypocalcemia | | |
| Birth trauma | | |
| Death | | |
| NICU admissions | | |

| DIABETIC | HYPERTENSION | MODE OF DELIVERY | INDICATION | ATONIC PPH | TRAUMATIC PPH | CERVICAL TEAR | LATERAL VAGINAL WALL TEAR | 3RD DEGREE PERINEAL TEAR | UTERINE RUPTURE | PROLONGED LABOUR | SHOULDER DYSTOCIA | BIRTH ASPHYXIA | RESPIRATORY DISTRESS | HYPOGLYCEMIA | HYPOCALCEMIA | BIRTHTRAUMA | DEATH | NICU ADMISSION | BIRTH WEIGHT OF BABY | centile values of abdominal circumference | INSTRUMENTAL DELIVERY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ODM | NO | LSCS | PREVIOUS 2 LSCS | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |
| NO | NO | LSCS | FAILED INDUCTION | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | 2.7 | 92 | |
| NO | NO | LSCS | FAILED INDUCTION | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.1 | 94 | |
| GDM | GESTATIONAL HTN | LSCS | PREVIOUS LSCS WITH GDM | YES | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | YES | NO | NO | YES | 3.8 | 98 | |
| GDM | NO | LSCS | BREECH | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.1 | 92 | |
| NO | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | YES | NO | No | YES | YES | YES | NO | NO | YES | 3.8 | 98 | |
| IGT | NO | VAGINAL | | NO | YES | NO | YES | NO | NO | YES | NO | NO | YES | NO | NO | NO | NO | NO | 3.1 | 92 | VENTOUSE |
| NO | NO | VAGINAL | | NO | NO | YES | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | YES | 3.3 | 94 | |
| IGT | GESTATIONAL HTN | LSCS | FAILED INDUCTION | NO | NO | NO | NO | NO | NO | YES | YES | NO | YES | YES | YES | NO | NO | YES | 3.9 | 98 | |
| GDM | NO | LSCS | PREVIOUS LSCS WITH GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.7 | 97 | |
| NO | NO | LSCS | PREVIOUS LSCS IN LABOUR | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 94 | |
| NO | NO | LSCS | ANAMNIOS | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 3.7 | 97 | |
| NO | NO | VAGINAL | | NO | NO | YES | YES | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 4 | 99 | VENTOUSE |
| NO | NO | LSCS | PREVIOUS LSCS IN LABOUR | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 97 | |
| NO | NO | LSCS | CPD | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 94 | |
| NO | NO | LSCS | SECOND STAGE ARREST | NO | YES | NO | NO | NO | NO | YES | NO | NO | YES | NO | NO | NO | NO | YES | 3.6 | 97 | |
| NO | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 96 | |
| NO | GESTATIONAL HTN | LSCS | MACROSOMIA WITH MILD PE | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.8 | 92 | |
| NO | NO | VAGINAL | | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.1 | 93 | |
| NO | NO | LSCS | RHD | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | YES | 3.5 | 96 | |
| NO | NO | LSCS | CDMR | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 3.7 | 98 | |
| GDM | NO | LSCS | CDMR | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 96 | |
| GDM | NO | VAGINAL | | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 93 | |
| NO | NO | LSCS | MSL | NO | NO | NO | NO | NO | NO | YES | NO | NO | YES | YES | YES | NO | NO | YES | 4 | 99 | |
| IGT | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 4.1 | 99 | |
| NO | GESTATIONAL HTN | VAGINAL | | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 92 | |
| GDM | NO | VAGINAL | | NO | YES | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.6 | 97 | |
| ODM | NO | LSCS | UNCONTROLLED SUGARS | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | YES | 3.5 | 96 | |
| NO | NO | LSCS | FOETAL DISTRESS | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | 3.2 | 93 | |
| ODM | NO | LSCS | PREVIOUS LSCS WITH ODM | NO | YES | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 2.7 | 92 | |
| ODM | GESTATIONAL HTN | LSCS | PREVIOUS LSCS WITH ODM | YES | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 3.1 | 94 | |
| NO | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 94 | |
| GDM | GESTATIONAL HTN | VAGINAL | | NO | NO | YES | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | 3.5 | 94 | |
| GDM | NO | LSCS | PATHOLOGICAL TRACE | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| NO | NO | LSCS | PREVIOUS LSCS IN LABOUR | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 94 | |
| ODM | NO | LSCS | PREVIOUS LSCS WITH ODM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.9 | 98 | |
| NO | NO | LSCS | MSL | YES | NO | NO | NO | NO | NO | YES | NO | NO | YES | NO | NO | NO | YES | 3.6 | 97 | |
| GDM | GESTATIONAL HTN | LSCS | FAILED INDUCTION | YES | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | YES | NO | NO | YES | 3.9 | 99 | |
| NO | NO | LSCS | MSL | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.8 | 98 | |
| ODM | NO | VAGINAL | | NO | NO | YES | NO | NO | NO | YES | YES | IUFD | | | | | | 4.1 | 99 | |
| NO | NO | LSCS | CPD | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | 3.9 | 97 | |
| NO | NO | VAGINAL | | NO | NO | NO | YES | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 95 | |
| NO | NO | LSCS | PREVIOUS LSCS IN LABOUR | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 94 | |
| NO | NO | LSCS | FETAL DISTRESS | NO | NO | NO | NO | NO | NO | YES | NO | NO | YES | YES | YES | NO | NO | YES | 3.9 | 98 | |
| NO | NO | LSCS | FAILED INDUCTION | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 94 | |
| NO | NO | VAGINAL | | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 94 | |
| ODM | NO | LSCS | CPD | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | YES | 3.9 | 98 | |
| ODM | NO | LSCS | FAILED INDUCTION | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NN | NO | 3.1 | 92 | |
| NO | NO | VAGINAL | | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.1 | 92 | |
| NO | NO | LSCS | MACROSOMIA | NO | NO | NO | NO | NO | NN | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |
| NO | NO | LSCS | PREVIOUS 2 LSCS | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NN | NO | NO | NO | 3.5 | 94 | |
| NO | NO | LSCS | BREECH | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 94 | |
| NO | NO | LSCS | FOETAL DISTRESS | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.7 | 92 | |
| NO | NO | LSCS | FETAL DISTRESS | NO | NO | NO | No | NO | NO | YES | NO | YES | NO | NO | NO | NO | NO | NO | 3.1 | 92 | |
| NO | NO | LSCS | PREVIOUS LSCS IN LABOUR | NO | No | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 96 | |
| NO | NO | LSCS | NON PROGRESS OF LABOUR | YES | No | No | NO | NO | NO | YES | NO | NO | YES | NO | NO | NO | NO | NO | 3.1 | 92 | |
| NO | NO | LSCS | PRECIOUS PREGNANCY | NO | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.8 | 98 | |
| NO | NO | LSCS | FETAL DISTRESS | YES | No | No | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |
| GDM | NO | LSCS | PREVIOUS LSCS WITH GDM | NO | No | No | No | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | YES | 3.6 | 97 | |
| NO | NO | LSCS | MSL | NO | No | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.7 | 92 | |
| NO | NO | LSCS | PREVIOUS 2 LSCS | NO | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.3 | 93 | |
| NO | GESTATIONAL HTN | LSCS | MAROSOMIA | NO | No | No | No | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | YES | 3.4 | 94 | |
| GDM | NO | LSCS | UNCONTROLLED SUGARS | YES | No | No | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 3.4 | 94 | |
| NO | YES | LSCS | PRECIOUS PREGNANCY | YES | No | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 93 | |
| ODM | GESTATIONAL HTN | LSCS | MACROSOMIA | NO | No | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.6 | 96 | |
| IGT | NO | VAGINAL | | NO | No | YES | YES | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 3.4 | 95 | |
| NO | GE STATIONAL HTN | LSCS | PREVIOUS LSCS IN LABOUR | NO | No | No | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 4 | 99 | |
| NO | NO | VAGINAL | | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 96 | |
| GDM | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | NO | NO | NO | NO | 3.6 | 96 | |
| GDM | NO | LSCS | PREVIOUS LSCS WITH GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.6 | 96 | |
| GDM | NO | LSCS | CPD | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | NO | NO | NO | YES | 3.5 | 95 | |
| NO | NO | LSCS | CPD | YES | NO | NO | NO | NO | NO | NO | NO | NO | YES | YES | YES | NO | NO | YES | 3.8 | 98 | |
| GDM | NO | LSCS | MSL | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 94 | |
| GDM | NO | LSCS | PREVIOUS 2 LSCS | YES | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 3 | 92 | |
| GDM | NO | LSCS | NPL WITH SUSPICIOUS TRACE | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |
| ODM | NO | LSCS | PREVIOUS LSCS WITH ODM | YES | NO | NO | NO | NO | NO | NO | NO | MSB | | | | | | NO | 2.9 | 92 | |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| GDM | NO | LSCS | MACROSOMIA | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.8 | 92 | |
| NO | NO | LSCS | MACROSOMIA | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.1 | 94 | |
| GDM | NO | LSCS | PRECIOUS PREGNANCY | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.2 | 94 | |
| NO | NO | LSCS | FOETAL DISTRESS | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 95 | |
| GDM | NO | LSCS | BREECH | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.8 | 92 | |
| GDM | NO | LSCS | PREVIOUS LSCS WITH GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | 3.2 | 93 | |
| GDM | NO | LSCS | PREVIOUS 2LSCS | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 3.2 | 93 | |
| GDM | NO | LSCS | PREVIOUS LSCS WITH GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.3 | 93 | |
| GDM | No | LSCS | Previous LSCS with GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 2.8 | 95 | |
| ODM | Mild PE | LSCS | BREECH WITH MACROSOMIA | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | Yes | NO | NO | YES | 3.9 | 98 | |
| No | No | LSCS | MSL | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.8 | 98 | |
| No | No | VAGINAL | | NO | YES | YES | YES | NO | NO | NO | YES | NO | NO | Yes | Yes | NO | NO | YES | 4.3 | 99 | VENTOUSE |
| No | No | LSCS | Previous lscs | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.8 | 98 | |
| No | No | LSCS | Previous LSCS with GDM | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 4 | 99 | |
| GDM | No | LSCS | Previous 2 LSCS | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.9 | 93 | |
| No | No | LSCS | Failed induction | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 2.9 | 92 | |
| No | No | LSCS | DTA | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | NO | NO | NO | NO | YES | 3.6 | 96 | |
| No | No | LSCS | Previous LSCS with macrosomia | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | Yes | NO | NO | YES | 3.7 | 96 | |
| No | No | LSCS | CDMR | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 96 | |
| IGT | Gestational HTN | LSCS | IGT with macrosomia | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3 | 92 | |
| No | No | LSCS | MSL | NO | NO | NO | NO | NO | NO | NO | NO | NO | YES | NO | NO | NO | NO | YES | 3.2 | 92 | |
| No | Gestational HTN | VAGINAL | | NO | YES | NO | YES | NO | NO | NO | YES | NO | NO | Yes | Yes | NO | NO | YES | 4.2 | 99 | VENTOUSE |
| Yes | No | LSCS | MSL | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | NO | NO | NO | NO | YES | 3.8 | 98 | |
| No | No | LSCS | Oblique lie with macrosomia | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 96 | |
| GDM | No | LSCS | Previous LSCS with macrosomia | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 4 | 99 | |
| No | No | LSCS | BREECH | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | Yes | NO | NO | YES | 4.5 | 99 | |
| No | No | LSCS | Previous LSCS with macrosomia | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 4 | 99 | |
| No | No | LSCS | CPD | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.8 | 99 | |
| No | No | LSCS | Foetal macrosomia | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 97 | |
| IGT | Gestational HTN | VAGINAL | | NO | NO | No | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.5 | 97 | |
| No | No | LSCS | Failed induction | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.4 | 96 | |
| No | No | LSCS | MSL | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | 3.7 | 96 | |
| No | No | LSCS | Fetal distress | YES | NO | NO | NO | NO | NO | NO | NO | NO | Yes | NO | NO | NO | NO | YES | 3.7 | 96 | |
| No | No | LSCS | Fetal distress | NO | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | Yes | NO | NO | YES | 3.8 | 97 | |
| GDM | No | LSCS | Oblique lie | YES | NO | NO | NO | NO | NO | NO | NO | NO | NO | Yes | Yes | NO | NO | YES | 3.8 | 98 | |