UMBILICAL CORD THICKNESS, FETAL FAT LAYER, AND SHOULDER PAD THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL AND PREGESTATIONAL DIABETES MELLITUS

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CERTIFICATE

This is to certify that the dissertation entitled **"PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FETAL FAT LAYER, AND SHOULDER PAD THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL AND PREGESTATIONAL DIABETES MELLITUS"** is a bonafide work done by **DR.K.GOKULAPRIYA** in the Institute of Obstetrics and Gynaecology (Madras Medical College) Egmore, Chennai in partial fulfillment of the university rules and regulations for award of MD degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2010-2013.

Prof. DR.V.KANAGASABAI M.D

DEAN
Madras Medical College &
Rajiv Gandhi Govt. general hospital
Chennai – 3

Prof. DR. P.MEENALOCHANI MD,DGO
Director and Superintendent
Institute of Obstetrics & Gynaecology
Madras medical college, Chennai-3

Prof.DR.K.RUKMANI M.D., DGO.
Guide,
Institute of Obstetrics and Gynaecology
Madras medical college, Chennai-3

DECLARATION

I solemnly declare that this dissertation entitled **"PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FETAL FAT LAYER, AND SHOULDER PAD THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL AND PREGESTATIONAL DIABETES MELLITUS"** was done by me at Institute of Obsterics & Gynaecology,Madras Medical College during 2010-2013 under the guidance and supervision of, **Prof.Dr.K.RUKMANI MD.,DGO.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in Obstetrics and Gynaecology (Branch-II).

Place: Chennai Signature of Candidate

Date:

Dr.K.GOKULAPRIYA M.B.B.S
MD Post Graduate Student
Institute of Obstetrics & Gynaecology
Egmore, Chennai-8

Prof. DR.K.RUKMANI M.D., DGO.
Guide,
Institute of Obstetrics and Gynaecology
Madras medical college, Chennai-3.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. K. Gokulapriya
PG in MD Obstetrics & Gynaecology
Institute of Obstetrics & Gynaecology
Egmore, Chennai -8

Dear Dr. K. Gokulapriya

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Prospective study on sonographic measurement of umbilical cord thickness, fetal fat layer, and shoulder pad thickness as predictors of macrosomia in fetus of women with gestational and pregestational diabetes mellitus" No.41082012.

The following members of Ethics Committee were present in the meeting held on 10/08/2012 conducted at Madras Medical College, Chennai -3.

1. Dr. S.K. Rajan. M.D.,FRCP.,DSc	-- Chairperson
2. Prof. Pregna B. Dolia MD Vice Principal, Madras Medical College, Chennai -3 Director , Institute of Biochemistry, MMC, Ch-3	-- Member Secretary
3. Prof. B. Vasanthi MD Prof of Pharmacology ,MMC, Ch-3	-- Member
4. Prof. C. Rajendiran, MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
5. Prof. S. Deivanayagam MS Prof of Surgery, MMC, Ch-3	-- Member
6. Thiru. S. Govindsamy. BABL	-- Lawyer
7. Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information /' informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

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INTRODUCTION

Diabetes mellitus is one of the commonest problem encountered in pregnancy. In Asia the incidence is around 5-8 %. It affects both the mother and the fetus in many ways. Infant of diabetic mother are more prone to be macrosomic which is a major cause of fetal and maternal morbidity.

Macrosomia means large sized fetus or neonate.[1] Fetal macrosomia is defined as birth weight of greater than 4000g or greater than 90th percentile for gestational age after correcting for neonatal sex and ethnicity.[2,3] Fetal macrosomia complicates more than 10 % of all pregnancies .

Macrosomia is associated with significant fetal, neonatal and maternal risks. Macrosomia increases the risk of shoulder dystocia, brachial plexus injury, skeletal injuries in the fetus[4].Maternal complications are increased incidence of operative delivery, postpartum hemorrhage, third or fourth-degree perineal lacerations [5]. In order to avoid the complications associated with delivery of macrosomic fetuses, it is prudent to antenatally predict macrosomia such that effort can be taken to provide a modified intrapartum care. The obstetrician's intention in anticipating macrosomia is for selecting cases wherein caesarean section is warranted to avoid maternal and fetal risk due to traumatic delivery and for a vigilant intra partum management.

The antenatal detection of macrosomia has many pitfalls. The traditional technique is calculating estimated fetal weight (EFW) using fetal biometric variables like biparietal diameter, femur length, abdominal circumference. But estimated fetal weight is not a reliable indicator of macrosomia or its associated intrapartum complications [6]. To overcome this difficulty clinicians proposed the use of soft tissue measurement- including shoulder pad thickness, cheek-to-cheek diameter, umbilical cord thickness, fetal fat layer, inter-ventricular septum thickness, etc.

The purpose of this prospective study was to evaluate the diagnostic value of ultrasonographic measurement of fetal umbilical cord thickness, fetal fat layer, and shoulder pad thickness for prediction of fetal macrosomia during intrapartum period.

REVIEW OF LITERATURE

DIABETES IN PREGNANCY

GESTATIONAL DIABETES MELLITUS:

Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy[8].In majority of cases of GDM, glucose regulation will return to normal after delivery. Some women with gestational diabetes have previously undiagnosed overt diabetes.

RISK ASSESSMENT:

Fifth International Workshop- Conference On Gestational Diabetes. Recommended Screening Strategy Based On Risk Assessment For Detecting Gestational Diabetes Mellitus [9].

LOW RISK :

Blood glucose testing is not routinely performed if the following criteria are fulfilled-

- belonging to low prevalence ethnic group

- age <25 years

- no history of diabetes in first degree relative

- normal pre pregnancy weight

- no history of glucose intolerance

- no history of poor obstetric outcome

AVERAGE RISK:

Perform blood glucose testing at 24-28 weeks using either one step or two step procedure .

-high prevalence ethnic group

-age ≥ 25 years

-overweight before pregnancy

HIGH RISK:

Perform blood glucose testing as soon as possible, if one or more of the following is present. If test is negative , repeat it at 24-28 weeks.

-severe obesity

-strong family history of type II diabetes

-previous history of glucose intolerance, GDM, glycosuria

SCREENING TEST:

ORAL GLUCOSE CHALLENGE TEST:

In this test, pregnant women are given 50 g of oral glucose challenge irrespective of the previous meal status. A plasma glucose level ≥ 140 mg/dl is considered screening test positive, and warrants further confirmation with Oral Glucose Tolerance Test (OGTT).

CONFIRMATORY TEST – OGTT (Oral Glucose Tolerance Test) :

Prerequisites for OGTT:

- 3 days prior to test- normal unrestricted diet containing a minimum of 150 g of carbohydrate

- overnight fasting of 8-14 hours

Technique:

100 g of glucose given with 300 ml of water, juice of lemon can be added to avoid vomiting.

Fasting , 1 hour, 2 hour, 3 hour plasma glucose is checked.

CRITERIA FOR DIAGNOSIS:

TIME	PLASMA GLUCOSE (mg/dl)	
	CARPENTER& COUSTAN	NDDG
FASTING	95	105
1 HOUR	180	190
2 HOUR	155	165
3HOUR	140	145

GDM is diagnosed when any two values are met or exceeded. If only one value is abnormal, it is called gestational impaired glucose tolerance.

PRE GESTATIONAL DIABETES MELLITUS:

Presence of diabetes either type I or type II even before pregnancy. The likelihood of successful outcomes with overt diabetes is related to the degree of glycemic control and degree of underlying cardiovascular and renal disease. Type I DM: It is characterized by onset at young age and an absolute insulinopenia. Type II DM: It is characterized by onset at late age , obesity, and peripheral insulin resistance.

DIAGNOSIS:

Fasting glucose ≥126 mg/dl or Random blood sugar > 200 mg/dl is diagnostic of overt diabetes.

CONSEQUENCES OF DIABETES ON THE PLACENTAL FUNCION:

Placenta is situated in between the maternal and fetal circulation. In diabetes maternal and fetal hyperglycemia affects placental metabolism and growth. The surface area is increased especially in the peripheral villous structure. In diabetes, villous stroma is edematous with overexpression of hofbauer cells which causes release of placental cytokines- leptin, TNF-α, interleukin. These causes placentomegaly which is correlated with fetal macrosomia.[10]

The fetoplacental expression of insulin, IGF-1, IGF-2, and their receptors is regulated in a tissue specific manner and is affected by nutritional and endocrine factors. In the first trimester, the insulin receptors are present predominantly in the syntiotrophoblast facing the maternal circulation; whereas in third trimester it is predominantly present towards fetal circulation. The fetal IGF-1 and IGF-2 are accessible to the placental IGF-1 receptor. Fetal IGF-2 overexpression is associated with fetal overgrowth.

The IGF binding proteins are key modulators of the ligand receptor interaction. IGF-BP3 mRNA is increased in mothers with type-1 diabetes. In cord , increased IGF BP3 levels correlate with IGF-1 levels and incidence of macrosomia.

EFFECTS OF DIABETES ON PREGNANCY:

A) MATERNAL COMPLICATIONS:

1) Antepartum complications:

-Spontaneous miscarriages:

Early abortion is associated with poor glycemic control. HbA1c >12 or a persistent Fasting glucose >120 mg/dl is associated with increased risk of abortions.[11]

-Preterm labour:

About 20 % of deliveries are preterm because of infection and polyhydramnios.

-Preeclampsia:

Affects 10- 20 % of pregnant diabetics. It is associated with poor glycemic control and end organ damage.

-Infection:

There is high incidence of chorioamnionitis which in turn can lead to preterm premature rupture of membranes or preterm labour.

-Polyhydramnios:

This is usually associated with diabetic pregnancy. The possible causes are-fetal hyperglycemia causing polyuria, increased amniotic fluid glucose as an irritant to amnion resulting in excess production, and increased osmosis.[7]

-Diabetic retinopathy:

The first sign is presence of micro aneurysms. The women can have retinopathy ranging from non proliferative, pre proliferative to proliferative stage. Those with proliferative retinopathy need laser photocoagulation and strict glycemic control to prevent further deleterious effects on pregnancy.[11]

-**Diabetic nephropathy:**

This is characterized by proteinuria and hypertension. Patients with serum creatinine > 1.5 mg/dl and a proteinuria >3 g/ 24 hour can progress to end stage renal disease. [11]

-**Diabetic neuropathy:**

Most common form of neuropathy seen during pregnancy is gastroparesis.[11]

2) Intrapartum complications:

-**Prolonged labour:**

There is possibility of prolongation of first and second stage of labour due to large size of fetus.

-**Shoulder dystocia:**

This is common in pregnancies complicated by diabetes due to abnormal deposition of fat under the influence of insulin. There is increase in shoulder diameters causing difficulty in delivery of shoulder.

-**Increased operative interference:**

Cephalopelvic disproportion due to large sized fetus and fetal distress due to prolongation of labour are major causes of increased operative interference.

-**Perineal injuries:**

Due to large sized fetus and increased need for instrumental delivery there is increased risk of third and fourth degree perineal tears.

-**Postpartum hemorrhage:**

Overdistended uterus due to macrosomia & polyhydramnios, prolonged labour, trauma due to instrumental delivery are possible causes of postpartum hemorrhage.

3)Puerperium:

-Puerperal sepsis

-Lactation failure

B) FETAL COMPLICATIONS:

-**Fetal macrosomia :**

Macrosomia is fetal weight > 4000g. It results from:

a**) Maternal hyperglycemia** - PEDERSON'S HYPOTHESIS: according to this

theory, maternal hyperglycemia leads to fetal hyperglycemia which in turn causes hypertrophy of fetal β cells leading to fetal hyperinsulinaemia. This fetal hyperinsulinaemia is responsible for abnormal fat deposition, macrosomia, organomegaly.[7]

b) **Maternal hypertriglyceridemia -** Elevated maternal free fatty acids in diabetes leads to increased transfer to the fetus. This increases the triglyceride synthesis, leading to adiposity.

-Congenital malformations:

Major congenital malformations are the leading case of perinatal mortality in infants of diabetic mother. There is three to ten fold increase in the incidence of congenital anomalies, with incidence of 9.5 to 16.5%.[12,13].Poor glycemic control during the critical period of organogenesis (between 5- 8 weeks of gestation)is the main factor.

-Growth restriction:

It is associated with maternal vasculopathy.

-Sudden intrauterine death:

There is increased risk of sudden inutero death in patients with inadequate glycemic control and macrosomic fetus. The possible causes are:

-placental insufficiency due to over grown fetus

-chronic fetal hypoxia due to metabolic acidosis

-intravascular thrombosis due to elevated hematocrit

C**) NEONATAL COMPLICATIONS:**

-**Hypoglycemia:**

Serum glucose level of less than 35- 40 mg/dl

This is due to the β cell hyperplasia induced by chronic maternal hyperglycemia.

-**Hyperbilirubinemia:**

This is due to prematurity, immature hepatic bilirubin conjugating system, increased breakdown of red cells.

-**Hypocalcemia:**

This is due to prematurity, asphyxia, hypoparathyroidism. It is also attributed to maternal hypomagnesaemia.[7]

-**Polycythemia:**

Fetal hyperglycemia due to maternal hyperglycemia causes tissue hypoxia. This hypoxia stimulates fetal erythropoietin resulting in polycythemia.

-**Respiratory distress syndrome:**

Poor glycemic control delays fetal lung maturity. Hence every effort must be made to prolong pregnancy till 38 completed weeks in diabetics.

D)LONG TERM COMPLICATIONS:

-minor cognitive deficiencies

-childhood obesity

-type II diabetes

MANAGEMENT OF DIABATES COMPLICATING PREGNANCY:

PRE PREGNANACY COUNSELLING:

- Awareness about good glycemic control and risk of congenital anomalies.
- Switch over from oral hypoglycemic agents to insulin
- Folic acid supplementation
- Baseline fundus examination, renal function test
- Diet and exercise

ANTANATAL MANAGEMENT:

GLYCEMIC CONTROL:

<u>Target values</u>:

- Fasting :<95 mg/dl
- Postprandial : 1 hr : <140 mg/dl

2 hr : <120mg/dl

Therapeutic options to control hyperglycemia include:

- Diet
- Exercise
- Insulin therapy
- OHA

MEDICAL NUTRITION THERAPY:

Calorie allotment :[7]

BMI 19 to 27 : 30 kcal/kg per day

BMI 27 to 29 : 25 kcal/kg per day

BMI > 30 : 12 to 15 kcal/kg per day

BMI < 19 : 35 kcal/kg per day

Diet:

- Carbohydrates : 50-60%
- Protein : 20%
- Fat : 25-30%

This combination is given as 3main meals and 3 snacks. It is important to avoid ketosois. Diet given for 2 weeks and blood sugar values assessed.

EXERCISE:

- Regular exercise
- Brisk walking for 20- 30 min
- Upper arm ergometrics
- Stationary bicycling

- Swimming – for morbid obesity

Exercise helps in decreasing insulin resistance. Women without medical or obstetrical contraindications should been encouraged to start or continue a program of moderate exercise to lower glucose concentrations

INSULIN THERAPY:

15% requires insulin when diet fails to maintain sugars at the following levels:

-Fasting plasma glucose < 95 mg/dl

-2 hr postprandial plasma glucose <120 mg/dl

- Pre mixed insulin is not appropriate
- If FPG is high, an intermediate acting insulin is given at bedtime.
- If the postprandial blood glucose high, short acting insulin is given before the meals

Intermediate acting:

-40% of total daily dose

-Peak action- 4- 8 hours

-Duration of action-12- 18 hours

Regular Insulin:

-60% of total daily dose

-peak action-2-3 hrs

-duration of action-8-10 hrs

Short acting insulin analogues:

-Insulin lispro and aspart, currently used in pregnancy

-Acceptable safety profiles

-Minimal transfer across the placenta

-No evidence of teratogenesis

-Improve postprandial glucose excursions

-Lower risk of delayed postprandial hyperglycemia.

Long acting insulin analogues:

(Insulin Gargline and Detemir) not recommended for use in pregnancy at present.

DAWN PHENOMENON:

-High fasting blood sugars in the absence of nocturnal hypoglycemia

-Requires an increase in intermediate acting insulin at bed time

SOMOGYI'S PHENOMENON:

-High fasting blood glucose associated with nocturnal hypoglycemia

-Requires decrease in intermediate acting insulin at bed time

ORAL HYPOGLYCEMIC AGENTS:

Concerns:

Transplacental passage can lead to fetal teratogenesis and prolonged neonatal hypoglycemia .Most retrospective studies have not demonstrated an increased risk of malformed infants.

METFORMIN:

- Decreases hepatic glucose output
- Improving peripheral glucose uptake, thus reducing insulin resistance
- May be a more logical alternative to insulin for women with GDM who are unable to cope with the increasing insulin resistance of pregnancy

OBSTETRIC MANAGEMENT:

- AN check up: Patient should be seen fortnightly throughout pregnancy, and weekly in the third trimester.
- First trimester USG for nuchal translucency (screening for aneuploidy) and for accurate dating, which is so important in these patients as most of them would require induction of labour.
- Anomaly scan at 18- 20 wks to rule out congenital malformations. Fetal ECHO may be done if there is any doubt in the four chamber view of the heart.
- A triple screen test at 16- 18 wks is usually done as a screening test for Downs syndrome and neural tube defects. However it should be noted that the maternal serum AFP level is lower in diabetics and should be divided by a factor of 0.8 to get the corrected value.

- Further ultrasound to assess fetal growth may be performed at 28-30 wks and repeated at 34-36 wks.

FETAL SURVEILLANCE:

- All women should be made aware of the significance of keeping a watch over the fetal movements in the third trimester.(Fetal kick count)
- Twice weekly NST, biophysical profile
- Doppler study of umbilical artery may be useful in monitoring pregnancies in women with vasculopathy or if IUGR sets in.[7]

TIMING OF DELIVERY:

- GDM on meal plan : 40 weeks
- GDM on insulin :38-39 weeks
- Pre-GDM :38 weeks
- Earlier termination needed in case of associated PIH, fetal compromise.
- Antenatal corticosteroids given in case of preterm delivery. [7]

LABOUR MANAGEMENT:

VAGINAL DELIVERY:

- Assessment of cervix(Bishops scoring)
- If cervix is favourable-

  -Night dose of NPH need not be omitted

-Induction is started in the morning

-Omit morning insulin and meal

-Start iv infusion

- If cervix is un favourable cervix-

  -local prostaglandins to prime cervix

  -allowed to take insulin and food till labour sets in

ELECTIVE CAESAREAN:

- Planned as first case in the morning
- Night dose of regular and NPH given
- Omit morning dose of insulin

DURING LABOUR:

- Achieve mean blood glucose level – around 100 mg/ dl

- Start iv infusion with 5% dextrose at 100- 125 ml/hr

- Start insulin infusion at 1unit/ hr

- Check blood sugar every 1-2 hrs

- Blood sugar > 250mg/dl- stop 5% D and start NS

-Close FH monitoring

-Continuous electronic fetal monitoring

-Intermittent auscultation one on one basis

-Epidural analgesia

-Anticipate shoulder dystocia and PPH

POST PARTUM MANAGEMENT:

- Sharp fall in insulin requirement immediately after delivery.
- GDM on meal plan- can revert to normal diet postpartum.
- GDM on insulin- do not require insulin postpartum.
- IDDM- half the pregnancy dose/ pre pregnancy dose.
- All gestational diabetics advised:

  -GTT at 6 wks

  -Diet

  -Exercise

  -Weight reduction
- Breast feeding encouraged

CONTRACEPTION:

- BARRIER METHOD: suits all , but high failure rate
- ORAL PILLS: low dose pills can be used safely in all diabetics except those with vasculopathy / DM >20 yrs
- INTRA UTERINE DEVICE: Cu-T can be inserted taking strict aseptic precautions

- PERMANENT METHOD: tubal ligation when future pregnancy not contemplated

MACROSOMIA

Fetal macrosomia is nothing but excessive fetal growth. [14] Several definitions of fetal macrosomia exist, the most common include:

- An absolute birth weight greater than 4000 g
- An absolute birth weight greater than 4500 g
- A birth weight greater than 90% centile for gestational age

These definitions are based on measurements of the infant after delivery. The American College of Obstetrics and Gynaecologists (ACOG) defines macrosomia as an infant with an absolute birth weight greater than 4500 g irrespective of gestational age or demographic variables.

A grading system of macrosomia has been used in the research setting to determine maternal and neonatal outcomes associated with fetal macrosomia. It was first described by **Boulet et al.**. Macrosomic infants are subclassified into three groups:

Grade 1 : 4000 – 4499 g

Grade 2 : 4500 – 4999 g

Grade 3 : ≥ 5000 g

It has been shown that maternal and neonatal outcome progressively worsens from grade 1 to grade 3[15,16] Fetal macrosomia occurs with an incidence of approximately 8- 10 % in developed countries.[17]

RISK FACTORS OF FETAL MACROSOMIA:

- **Diabetes Mellitus:**

  Pregnant women with diabetes – gestational or pre gestational, have increased risk of giving birth to macrosomic infant. Maternal hyperglycemia leads to fetal hyperglycemia which in turn leads to fetal hyperinsulinism .Fetal hyperinsulinism triggers fetal macrosomia. Hence poorer the glycemic control, higher the risk of macrosomia.

- **Genetic Factors:**

  Maternal obesity could lead to genetic preponderance for a large fetus [1]. Some of the inheritable syndromes associated with fetal macrosomia are listed below.

SYNDROME	CLINICAL FEATURES	INHERITANCE
Perlman's syndrome	Macrosomia, visceromegaly, hydramnios,Wilm's tumour, ileal atresia, corpus callosal agenesis	Autosomal recessive
Macrosomia adipose congenita	Macrosomia,voracious appetite,precocious skeletal development	Autosomal recessive
MOMO syndrome	Macrocephaly, retinal coloboma,nystagmus, mental retardation	Autosomal dominant
ABCD syndrome	Macrosomia, fetal intestinal dysfunction	Autosomal recessive
Beckwith Weidemann syndrome	Macrosomia,macroglossia,cardiomegaly, omphalocele,wilm's tumour	Autosomal dominant

- **Parity :**

  It has been shown that multiparity is associated with increased risk of fetal macrosomia. A large UK study demonstrated that a parity of greater than four was associated with a 2 fold increased risk of delivering a macrosomic infant.[18]

- **Gestational age :**

  Gestational age significantly affects the absolute birth weight.With each additional week beyond the expected date of delivery, there is more chance of babies weighing more than 4000g. Stotland et al found in their study that more than one third of infants born beyond 41 weeks had birth weight more than 4000 g.[16]

- **Fetal gender :**

  Fetal macrosomia has been reported more frequently in the male fetus occurring in a 2 : 1 male to female ratio. [19]

- **Previous macrosomia :**

  Previous macrosomia is a strong predictor of developing fetal macrosomia in subsequent pregnancies. A Canadian case control study found a 9 fold increased risk of macrosomia in women with previous birth weight more than 4000g.[20]

- **Race and ethnicity:**

  Majority of studies place White and Hispanic women at increased risk of fetal macrosomia[21,22]. An American study found that the rate of fetal macrosomia in White women was 16% compared to 11% in the non-white population.[2] In another study, it was found that obesity

exerts a profound effect on Hispanic women and these women are at markedly increased risk of delivering a macrosomic fetus.[18,22]

- **Maternal weight:**

  Maternal weight has significant impact on fetal weight. A UK based study demonstrated that increased body mass index(BMI) was a strong predictor of fetal macrosomia. If BMI >30 kg/m^2 , there was 2 fold increased risk of fetal macrosomia[18]. An excessive weight gain during pregnancy is also associated with increased risk of macrosomia[20]. It has also been demonstrated that maternal birth weight is a predictor of fetal birth weight.[23]

PATHOPHYSIOLOGY:

The normal growth of a fetus is a balance of 3 major factors:[24]

- Genetic drive for growth
- Environmental factors in utero
- Supply of growth substrates to the fetus

Alterations in this delicate balance may result in growth restriction or accelerated growth. Two growth pathways have been described –

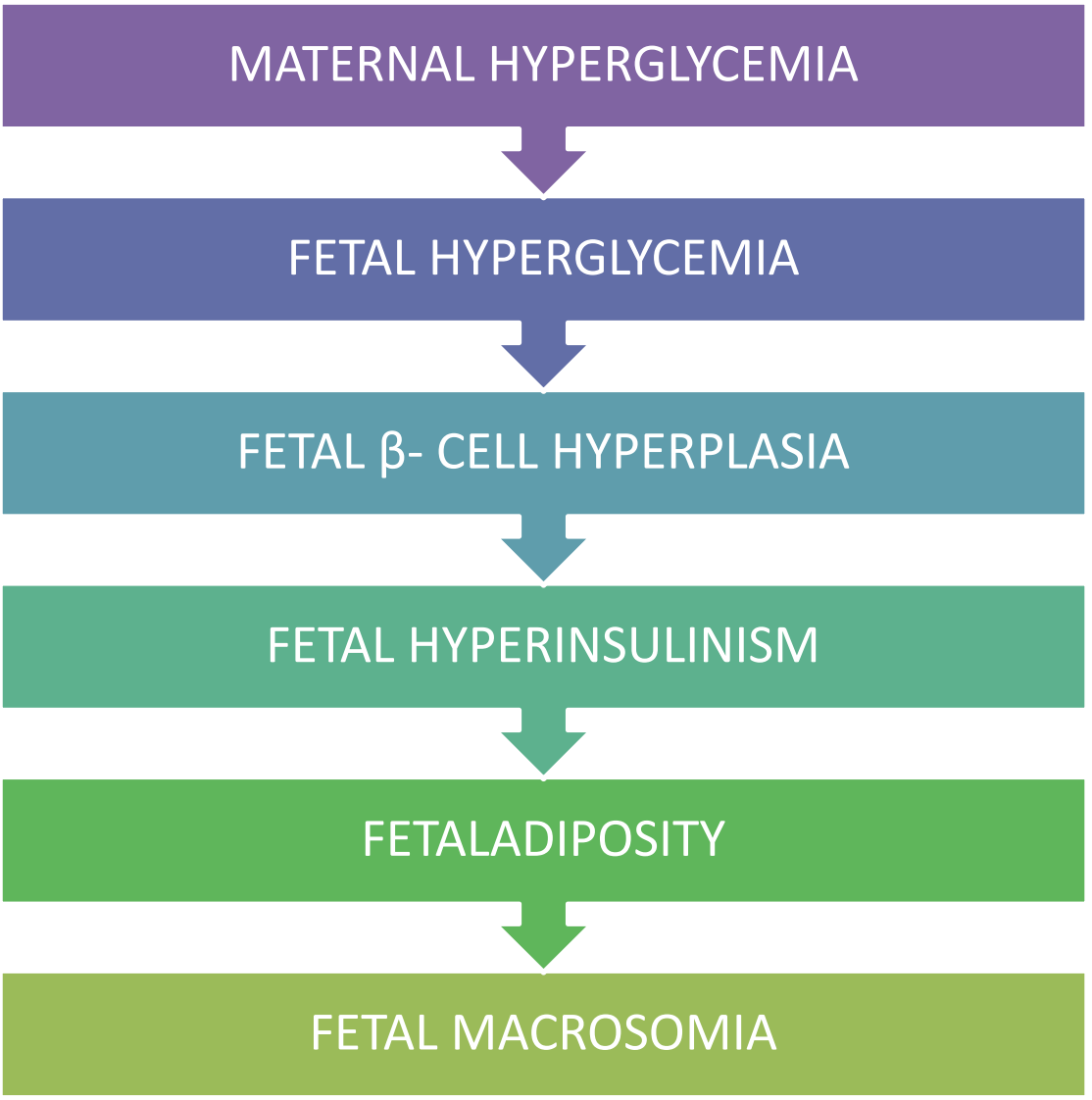
-fetal hormone dependent growth

-substrate limited growth.

The supply of substrate to the fetus is regulated by materno-placental factors. Insulin and insulin like growth factors are important regulators of fetal growth. Glucose is transferred from the maternal compartment to the fetal compartment via the placenta. The fetal glucose concentration is approximately 80% of the maternal level. Hence, as maternal blood glucose increases so will fetal blood glucose. Normal pregnant woman have reduced insulin sensitivity leading to a state of hyperglycaemia, to provide substrates to the fetus. [25] It is well documented that obesity, increasing age, diabetes mellitus reduces insulin sensitivity and increases insulin resistance. These factors may compound the already suppressed insulin sensitivity of pregnancy leading to a state of increased carbohydrate intolerance, increasing the amount of glucose available

for maternofetal transport, thus driving fetal hyperinsulinaemia and accelerated intra-uterine growth.

PEDERSON'S THEORY:



Insulin resistance is associated with higher fasting triglyceride concentrations. Triglycerides are energy – rich and placental lipases can cleave them and transfer free fatty acids into the fetal circulation. This increases energy and substrate delivery to the fetus and may further increase insulin level.[18]Certain

amino acids, such as leucine, stimulate the secretion of insulin. Insulin resistance is associated with a greater leucine turnover. It can be envisaged that this may help to promote fetal growth by increasing fetal insulin levels.

The fetal growth pattern and the type of tissue that is overgrown reflects the underlying etiology. Insulin sensitive tissues like subcutaneous fat, shoulder girdle, liver, spleen, thymus, heart, adrenal gland, show differential fat and glycogen deposition when insulin levels are high. Consequently, total body fat, shoulder circumference, upper limb skin fold thickness are disproportionately higher in macrosomic infants of diabetic mothers. The function of liver as the primary glycogen storage organ makes the preferential growth of abdominal circumference as a significant predictor of macrosomia.[14]

COMPLICATIONS OF FETAL MACROSOMIA:

FETAL COMPLICATIONS:

- **Shoulder dystocia:**

  Shoulder dystocia is difficulty in the delivery of the shoulders after delivery of head.The risk of shoulder dystocia rises sharply from 3% for birth weights <4000g to 10.3% for birth weight between 4000-4500g. [26] In diabetic women ,when birth weights are >3500 g, the incidence of

shoulder dystocia generally doubles compared to non diabetic patients for similar birth weights.

- **Asphyxia :**

  Due to prolonged second stage of labour , as with shoulder dystocia, the fetus becomes asphyxiated.

- **Brachial plexus injury ,fractures of clavicle, humerus-**

  Infants with shoulder dystocia have increased risk of these complications.

- **Meconium aspiration** –

  This is more common due to the protracted second stage of labour, which leads to fetal anoxia, which leads to passage of meconium.

- **Respiratory distress syndrome-**

  Iatrogenic prematurity following premature induction in view of avoiding shoulder dystocia

- **Neonatal hypoglycemia, hypocalcemia**
- **Macrosomic cardiomyopathy**

MATERNAL COMPLICATIONS:

- Increased incidence of operative vaginal delivery and cephalopelvic disproportion.

- Increased incidence of cesarean section.
- The risk of third or fourth degree lacerations is especially if delivery is complicated by shoulder dystocia.
- Increased incidence of postpartum hemorrhage.
- Thromboembolic events and anaesthetic complications are increased in great part because of the increased need for operative intervention.

PREDICTION OF MACROSOMIA:

The three major strategies used to predict macrosomia are:

- Assessment of clinical risk factors
- Clinical estimation of fetal weight
- Ultrasonography

CLINICAL RISK FACTORS:

The presence of risk factors can be identified by taking a detailed history. The presence of one or two risk factors must prompt an obstetrician to further investigate to rule out the possibility of macrosomia. Some of the risk factors that

are strongly associated with macrosomia are listed below:

- Maternal diabetes -gestational or pre-gestational
- Maternal obesity : pre-pregnancy weight and weight gain during pregnancy
- Previous macrosomic baby

- Maternal age
- Race and ethnicity

CLINICAL ESTIMATION:

- Estimation of fetal weight by measuring the symphysiofundal height is done using Johnson's formula. This formula can be used in vertex presentation. If vertex is above or at the level of ischial spine, estimated fetal weight is calculated by using the following formula:

  Fetal weight (grams) = (SFH-12) x 155

If vertex is below the level of ischial spine, the formula used is:

Fetal weight (grams) = (SFH-11) x 155

- Estimation of fetal weight by clinical examination of symphysiofundal height is usually inaccurate , especially at the extremes of fetal sizes.
- The amniotic fluid volume and maternal obesity complicate estimation of the size of the fetus by palpation through the abdominal wall.
- Prospective studies of fundal height measurement combined with Leopolds maneuvers report sensitivity of 10%- 43%, specificity of 99.0%-99.8%, and positive predictive values of 28%- 53% for the detection of macrosomia.[14]

ULTRASOUND:

- Ultrasonography provides a more accurate means of obtaining an estimated fetal weight. Measurement of several fetal parameters have been described for the estimation of fetal weight. Formulae incorporating measures of head circumference and abdominal circumference are the most predictive.[27]
- Hadlock's formula utilizing the abdominal circumference(AC) and femur length(FL) provides the best estimation of birth weight of macrosomic fetuses[28].
- Log10 (BW) =1.304 + 0.05281 (AC) +0.1938 (FL) - 0.004 (ACxFL)

- Nadia et al ,studied the accuracy of the sonographic fetal weight estimation in prediction of fetal macrosomia and found it to have a sensitivity and specificity of 74.5% and 85.7% respectively, a positive predictive value of 89% and a negative predictive value of 69%.[29]
- In diabetic pregnancies, abdominal circumference, especially measured serially during the third trimester of pregnancy, is the best single sonographic measurement for the detection of macrosomic fetus.[30]
- The relationship between fetal chest circumference and head circumference and abdominal circumference may be potentially useful in predicting shoulder dystocia in pregnancies at risk for this condition.

- Bethune and Bell[39] studied the usefulness of fetal fat layer and cardiac interventricular septum thickness as predictors of macrosomia. The fetal cardiac IVS thickness is greater in diabetic than in non- diabetic pregnancies. This could probably be the cause of increased risk of diabetic cardiomyopathy although the cardiac function is not altered significantly[14].

  A fetal fat layer of ≥5 mm is a useful predictor of macrosomia at term as assessed using the likelihood ratio. An AC ≥90th percentile, however, had a better sensitivity. The usefulness of routine FFL measurement in the early third trimester in the management of diabetic pregnancies is worthy of further evaluation.

- Nadia et al[29] studied the usefulness of humeral soft tissue thickness as the predictor of fetal macrosomia. It had the highest sensitivity (87.2%) and negative predictive value (78.7%), while the specificity was 74.2% and the positive predictive value 84.2%

- Landon et al [31], sonographic measurement of fetal humeral soft tissue thickness was performed for 93 women with gestational diabetes mellitus during the third trimester. He proved that-“ this measurement was the most accurate predictor of excessive birth weight compared with other standard ultrasound parameters (i.e. abdominal circumference, femoral length and others). It had a sensitivity and specificity of 82 and 95% respectively and a positive predictive value of 90%.”

- Cromi et al [32] studied the usefulness of umbilical cord thickness as a predictor of fetal macrosomia. The proportion of cases with a large umbilical cord was significantly higher in the population of macrosomic fetuses than in that of non-macrosomic ones (29/53 (54.7%) *vs*. 85/973 (8.7%), $P < 0.0001$).

MANAGEMENT OF MACROSOMIA:

- **GLYCEMIC CONTROL:**

  Accelerated fetal growth is an indicator of poor glycemic control. In such situation, increased surveillance and strict glycemic control can significantly reduce the chance of macrosomia.

- **FETAL SURVEILLANCE:**
  - Pregnant woman must carefully watch for fetal movements in the third trimester. They must maintain fetal kick chart.
  - Twice weekly NST from 32 weeks of gestation for high risk mothers
  - Doppler study useful in patients with vasculopathy
- **TIMING AND TYPE OF DELIVERY:**

  Expectant management:

  Expectant management is a valid option in managing macrosomic fetus in low risk patients. In high risk groups, like pregnancy complicated by

diabetes, expectant management might increase maternal and fetal morbidity due to difficult vaginal delivery.[33]

Elective caesarean:

While the risk of birth injury reduces with elective caesarean delivery in macrosomic fetus, it does not eliminate the risk completely. In non diabetic mothers, by adopting the policy of elective caesarean section, there is an increase in caesarean rates without much improvement in perinatal outcome.

In pregnancies complicated by diabetes, elective caesarean section for suspected macrosomia, appears to be medically and economically acceptable policy.

Early induction of labour:

Current evidence does not support the concept of early induction of labour because of:

-Increased caesarean section rate due to unfavourable cervix

-Increased neonatal morbidity associated with preterm-respiratory distress syndrome.[34]

-No significant reduction in the rate of shoulder dystocia.

- **LABOUR MANAGEMENT:**

During labour, careful attention must be given to the progress of labour and the uterine contraction pattern. Caesarean delivery must be offered when:

-fetal weight > 4500 g in diabetic women

-duration of second stage >2 hours with documented adequate contractions

(>200 Montevideo units)

-arrest of descent

-fetal distress

SHOULDER DYSTOCIA:

Shoulder dystocia is said to have occurred when additional obstetric maneuvers are

required to release the shoulders after gentle downward traction on the head has failed to affect shoulder delivery.

- Normal head to body delivery time: 24 sec
- In those with shoulder dystocia: 79 sec

Objective definition: Head to body delivery time exceeding 60 sec. Sponge et al 1995(0.15% - 1.4%)

Shoulder dystocia occurs due to impaction of anterior shoulder against the symphysis pubis(most common) or due to impaction of posterior shoulder against the sacral promontary (rare).

RISK FACTORS OF SHOULDER DYSTOCIA:

- Obesity
- Multiparity
- Maternal diabetes
- Post term pregnancy
- Prior shoulder dystocia : 2 – 16 fold increase in risk of recurrence
- Macrosomia
- Midpelvic instrumental delivery- 4.6% shoulder dystocia with operative mid pelvic delivery as against 0.2% with spontaneous delivery

PREDICTORS OF SHOULDER DYSTOCIA:

-A protracted or arrested active phase of first stage of labour.

-A protracted or arrested second stage of delivery.

-Assisted mid pelvic delivery.

-Previous history of shoulder dystocia

DIAGNOSIS:

- First sign of shoulder dystocia is recoil of the fetal chin into the perineum immediately following delivery of the head – Turtle Sign
- Anterior shoulder does not appear beneath symphysis pubis with gentle traction.

FETAL COMPLICATION:

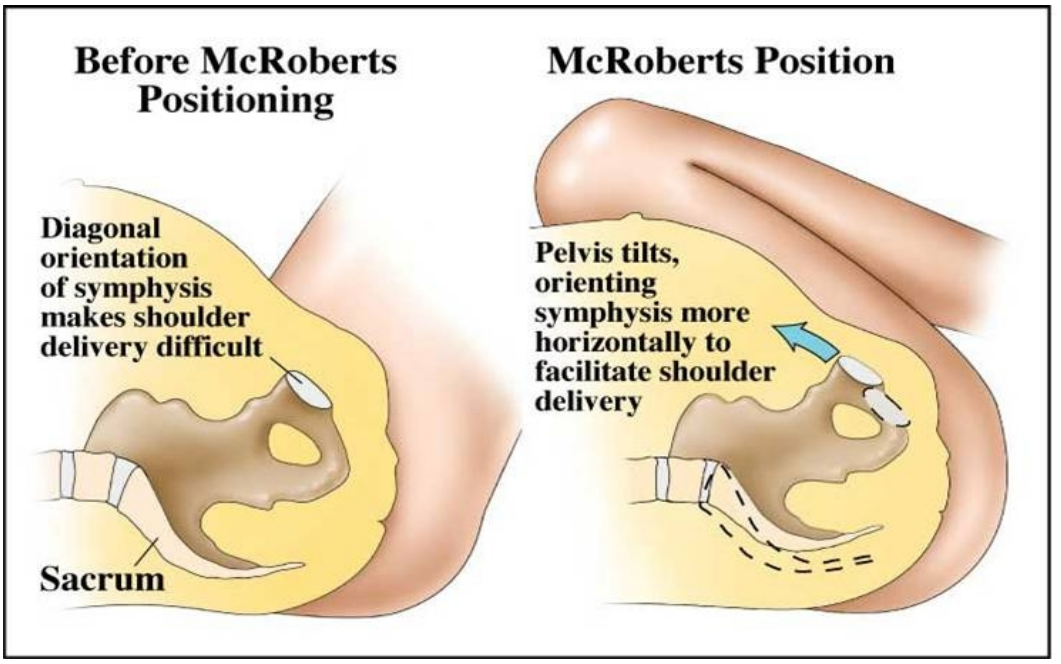
- Fetal asphyxia
- Cord occlusion
- Premature placental separation
- Spinal cord injury
- Brachial plexus injury - Incidence: 5 – 15%. Both upper & lower brachial plexus damage seen
- Fracture of clavicle (9.5%) & humerus (4.2%)

MATERNAL COMPLICATIONS:

- Genital tract injuries
- Atonic PPH
- Infection

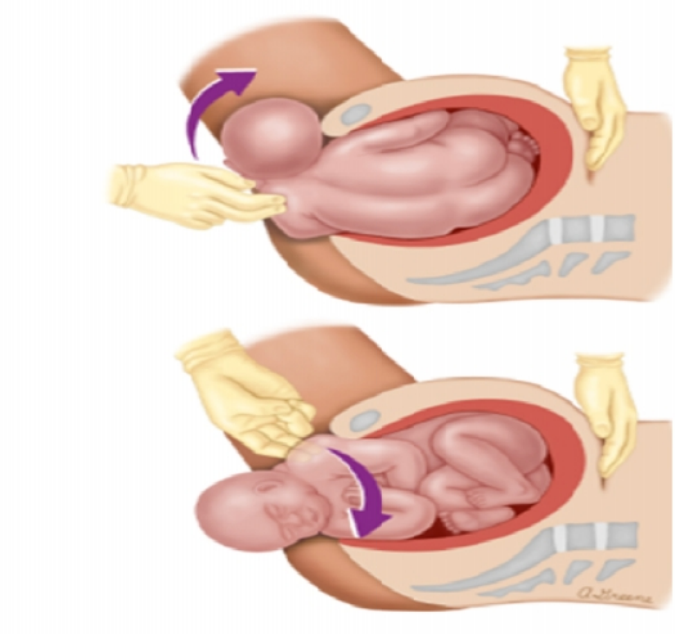
MCROBERT'S MANEUVER:

In this maneuver, the thighs are abducted and sharply flexed over abdomen. This causes straightening of the sacrum and decrease in the angle of pelvic inclination from 25 degree to 10 degree.The cephalad rotation of pubic symphysis frees the impacted anterior shoulder



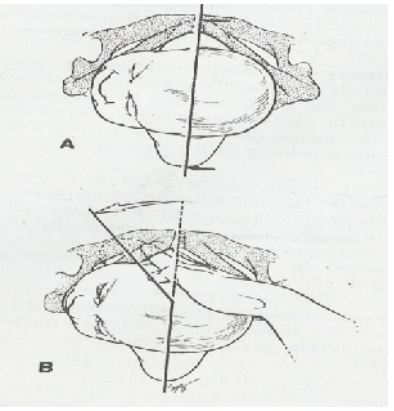
RUBINS MANEUVER:

Fetal shoulders are pushed to anterior aspect of chest, resulting adduction produces smaller diameters and disimpacts the anterior shoulder



WOODS MANEUVER:

Fingers placed anterior to the posterior shoulder and progressively rotate the posterior shoulder 180 degree in corkscrew fashion which releases the impacted anterior shoulder followed by downward traction affecting delivery of shoulders.



ZAVANELLI MANEUVER:

Pushing the fetus back into the uterus and delivering by caesarean section.

Key factors in successfully managing shoulder dystocia include constant preparedness, frequent drills, a team approach and appropriate documentation. Shoulder dystocia must be anticipated, and the obstetrician must be ready to handle it. Senior obstetrician, anaesthetist, neonatologist must be avaible at the time of delivery.

AIM

To evaluate the sonographic measurement of umbilical cord thickness, fetal fat layer, and shoulder pad thickness as predictors of macrosomia in fetus of women with gestational and pre gestational diabetes mellitus

STUDY DETAILS

SETTING:

Institute of Obstetrics and Gynaecology, Egmore, Chennai – 600 008.

COLLABORATING DEPARTMENT :

Department of Radiology , IOG

STUDY DESIGN:

Prospective Analytical Study

STUDY PERIOD:

One year (2011- 2012)

MATERIALS AND METHODOLOGY

This study was conducted for a period of one year from 2011 to 2012, at Institute of Obstetrics and Gynaecology, Egmore. Antenatal women with GDM or pre GDM, diagnosed by OGTT, dates confirmed by early scans, in their late third trimester(35-36weeks), who attended Antenatal OPD at IOG, were recruited prospectively into the study. Those who met the inclusion criteria were invited to participate in the study. Ethical committee clearance was obtained for the study and informed written consent was obtained from all the participants.

INCLUSION CRITERIA:

- Pregnant women with GDM / DM in late third trimester (35-36 weeks)

- Reliable dates confirmed by dating scan

- Three vessel umbilical cord

EXCLUSION CRITERIA:

- Multiple pregnancy

- Known anomalous baby

Proforma were filled after obtaining consent from each patient. Participants underwent a third trimester scan .Three extra measurements – Umbilical cord

thickness, Fetal fat layer and Shoulder pad thickness, were measured in addition to the normal examination. These measurements took not much of an extra time. The abdominal circumference and estimated fetal weight were part of routine scan. The measurements were performed by sonologist at the department of radiology, Institute of Obstetrics and Gynaecology, Egmore with 3.5 MHz abdominal probe.

Fetal anthropometric parameters, biparietal diameter (BPD), abdominal circumference (AC) and femur length (FL), were measured in all fetuses. EFW was obtained using the formula proposed by Hadlock *et al*.

UMBILICAL CORD THICKNESS:

The sonographic cross sectional areas of the umbilical cord were measured in a free loop of umbilical cord.

UMBILICAL CORD - FREE LOOP, LATERAL SECTION AND CROSS SECTION

UMBILICAL CORD CROSS SECTION – MEASUREMENT OF CORD DIAMETER

Using the umbilical cord diameter the umbilical cord area was calculated using computer software.

FETAL FAT LAYER:

The AC plane was magnified such that AC was completely focused in the screen area. The measurement of vertical distance between the inner and the outer border of the echogenic subcutaneous fat of abdomen is measured. Measurements were avoided in the quadrant which included the fetal back.

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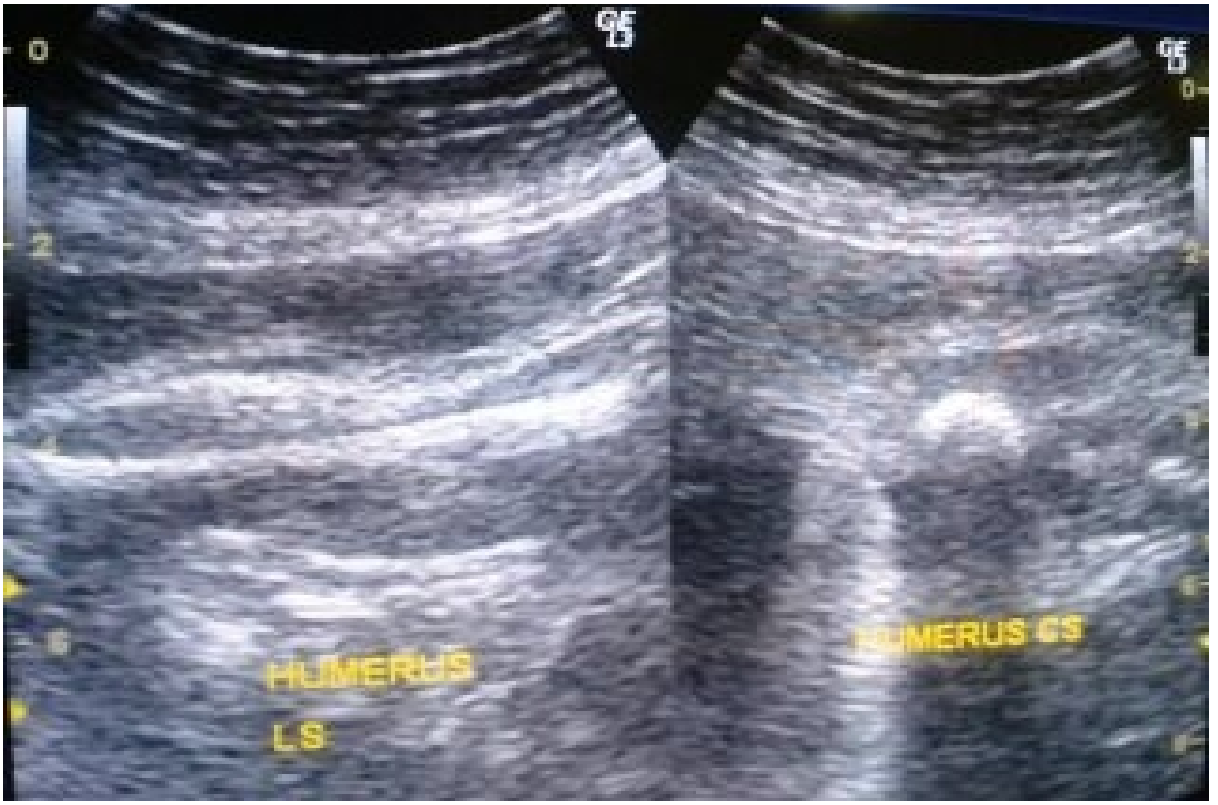
MEASUREMENT OF FETAL FAT LAYER

SHOULDER PAD THICKNESS:

The fetal humerus was visualized in a longitudinal view, and the transducer was rotated 90° then moved cephalad until the head of the humerus was found. The measurement was taken immediately below the humeral head from the outer edge of the bone to the skin surface.



HUMERUS- LONGITUDINAL SECTION AND CROSS SECTION

MEASUREMENT OF SHOULDER PAD THICKNESS

All the above three measurements was performed three times at the same occasion, and the average of the three values was compared with the estimated fetal weight for its ability to predict macrosomia. After delivery, the newborn's body weight was assessed.

Further, the patients were followed up at delivery. The delivery details and baby details were filled up in the proforma after the delivery. Then statistical analysis was done using SPSS software 16 version. Chi- square test was used to analyse significance.P- value of less than 0.005 is considered significant.

RESULTS AND ANALYSIS

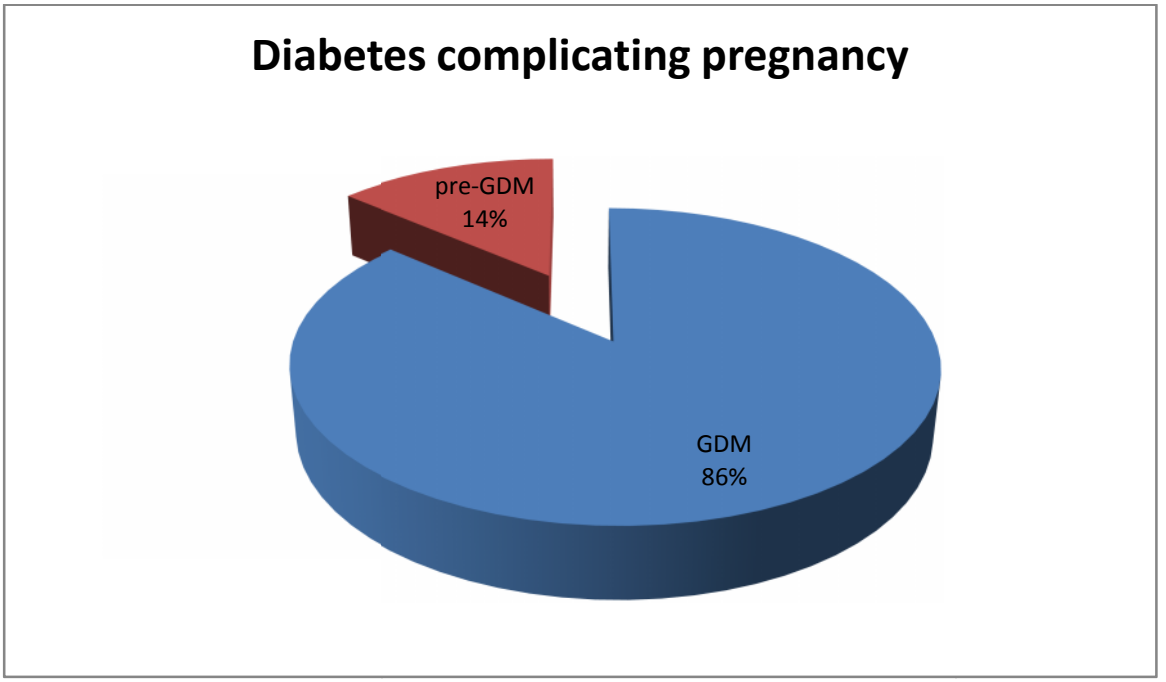
Total no. of GDM / Pre-GDM patients in late 3rd trimester : 512

Total no. of GDM / Pre GDM patients satisfying the study criteria : 437

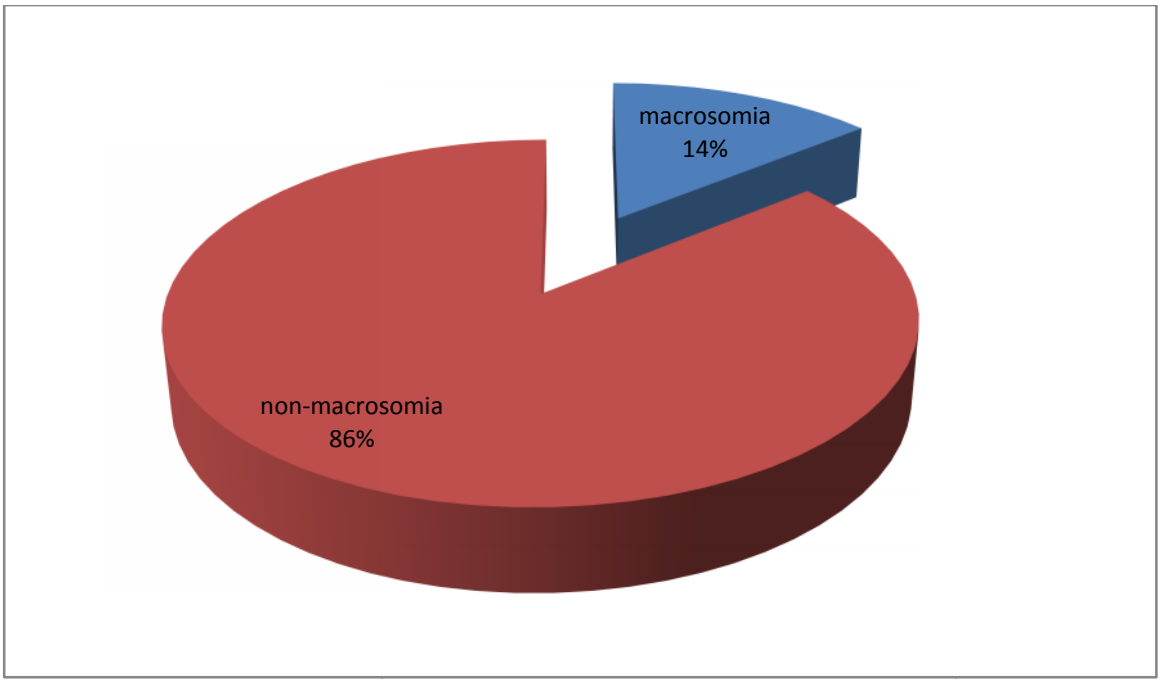
Total no. of drop outs : 032

Total no. of patients in our study : 405

Of the 405 patients under study, 350 patients had GDM, 55 patients had pre-GDM.



Of the 405 patients, 57 patients delivered macrosomic babies.



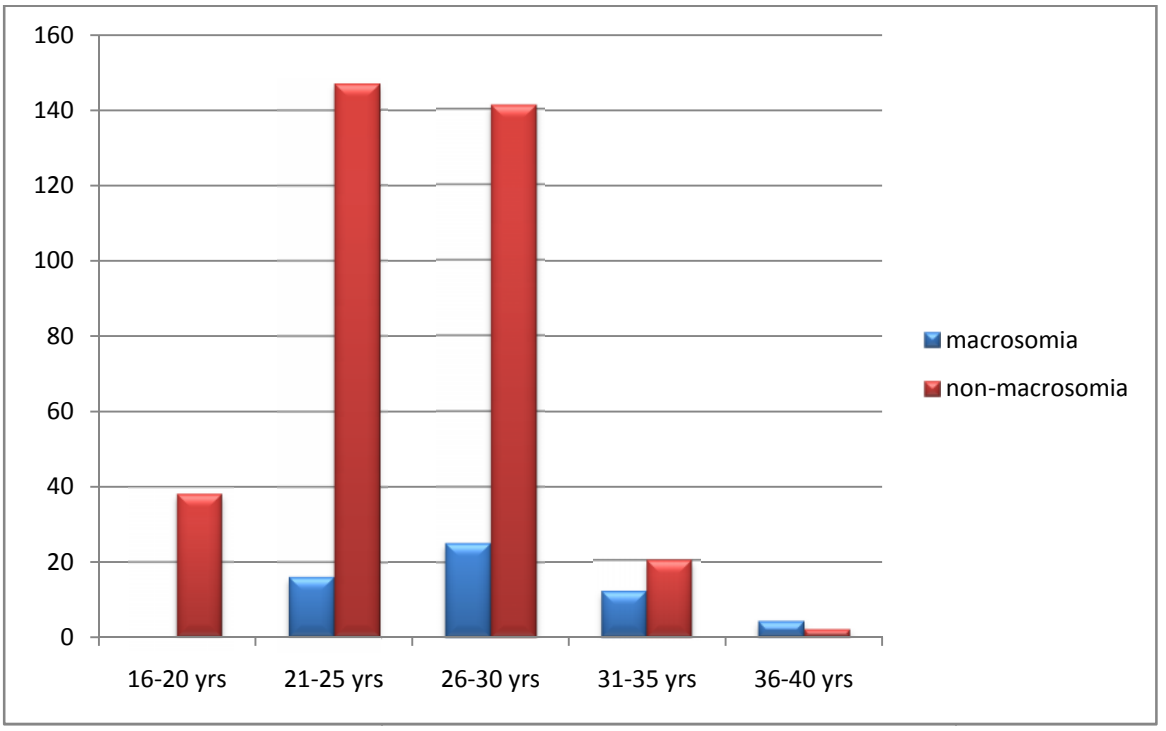
INFANT OF DIABETIC MOTHER-BIRTH WEIGHT: 4.25 KG

AGE:

AGE GROUP	TOTAL NO.OF PATIENTS	MACROSOMIA				p-VALUE
		YES		NO		
		NUMBER	%	NUMBER	%	
16-20	38	0	0	38	100	<0.001**
21-25	163	16	9.8	147	90.2	
26-30	166	25	15.1	141	84.9	
31-35	32	12	37.5	20	62.5	
36-40	6	4	66.7	2	33.3	

** - significant at 1%level

Among 405 patients under study, majority of patients belonged to the age group 26- 30 years(41%)



In the age group of 16- 20 years, there were 38 patients(9.4%) and none of them delivered macrosomic baby.

In the age group of 21-25 years, there were 163 patients(40.2%).Among this age group, 16 patients delivered macrosomic fetus(9.8%) and 147 patients delivered non-macrosomic fetus(90.2%).

In the age group of 26-30 years, there were 166 patients (41%). Among this age group, 25 patients delivered macrosomic fetus(15.1%) and 141 patients delivered non-macrosomic fetus(84.9%).

In the age group of 31-35 years, there were 32 patients (7.9%). Among this age group, 12 patients delivered macrosomic fetus (37.5%) and 20 patients delivered non-macrosomic fetus (62.5%).

In the age group of 36-40 years, there were 6 patients (1.5%). Among this age group, 4 patients delivered macrosomic fetus (66.7%) and 2 patients delivered non macrosomic fetus (33.3%).

Thus , from our study, it is proved that as the age advances the chance of a diabetic mother giving birth to a macrosomic fetus increases.

AGE GROUP	NO. PATIENTS WHO DELIVERED MACROSOMIC FETUS	PERCENTAGE WITHIN MACROSOMIA
16-20 years	0	0 .0%
21-25 years	16	28.1%
26-30 years	25	43.9%
31-35 years	12	21.1%
36-40 years	4	7.0%
TOTAL	57	100%

Among the 57 macrosomic fetus, majority(43.9%) were delivered by mothers in the age group of 26-30 years.

PARITY:

PARITY	NO. OF PATIENTS	MACROSOMIA				p-VALUE
		YES		NO		
		NUMBER	%	NUMBER	%	
0	178	19	10.7%	159	89.3%	

1	172	16	9.3%	156	90.7%	<0.001**
2	36	10	27.8%	26	72.2%	
3	17	10	58.8%	7	41.2%	
4	2	2	100.0%	0	0.0%	

**- significant at 1% level

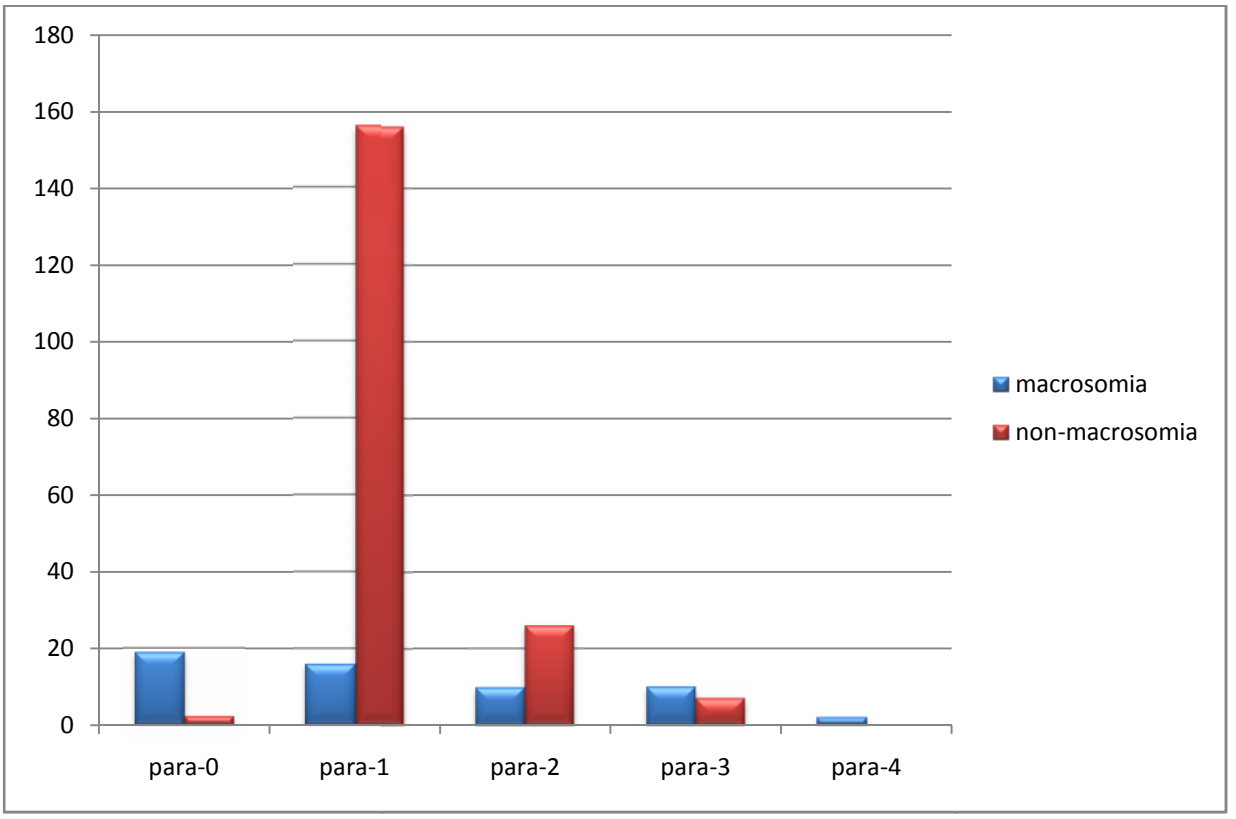
178 patients belonged to nulliparous(para-0) group, among whom 19 patients delivered macrosomic fetus(10.7%) and 159 patients delivered non-macrosomic fetus(89.3%).

172 patients belonged to primiparous(para-1) group, among whom, 16 patients delivered macrosomic fetus(9.3%) and 156 patients delivered non-macrosomic fetus(90.7%).

36 patients belonged to para-2 group, among whom,10 patients delivered macrosomic fetus(27.8%) and 26 patients delivered non-macrosomic fetus(72.2%).

17 patients belonged to para-3 group, among whom 10 delivered macrosomic fetus(58.8%) and 7 patients delivered non-macrosomic fetus(41.2%).

2 patients belonged to para-4 group, both of them delivered macrosomic fetus(100%).



Thus, from our study it is proven that as the parity increases the chance of giving birth to macrosomic baby also increases.

PARITY	NO. OF PATIENTS WHO DELIVERED MACROSOMIC FETUS	PERCENTAGE WITHIN MACROSOMIA
0	19	33.3
1	16	28.1
2	10	17.5
3	10	17.5
4	2	3.5
TOTAL	57	100

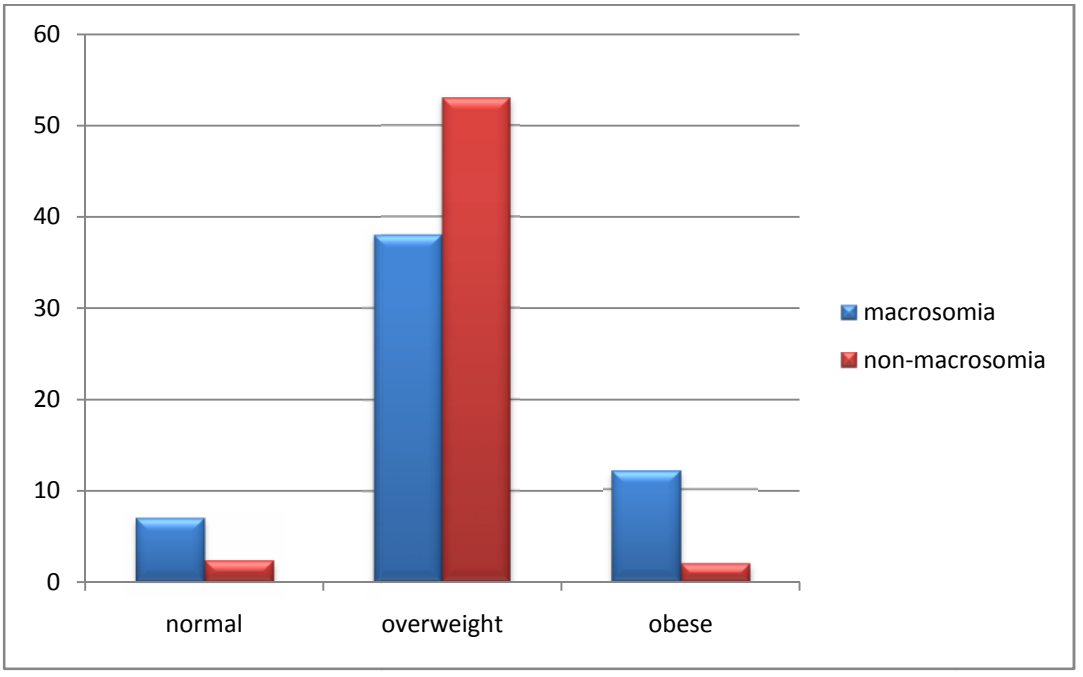
In our study, among the 405 diabetic mothers, majority(44%) were nulliparous.

BODY MASS INDEX(BMI):

BMI	NO. OF PATIENTS	MACROSOMIA				p-VALUE
		YES		NO		
		NO. OF PATIENTS	%	NO. OF PATIENTS	%	
19- 24.9	300	7	2.3	293	97.7	<0.001**
25-29.9	91	38	41.8	53	58.2	
≥30	14	12	85.7	2	14.3	

**- significant at 1% level.

Among the 57 patients who delivered macrosomic fetus, 38 (66.7%) i.e. majority were overweight.



300 patients belonged to normal BMI (19- 24.9) group. Among them, 7 patients delivered macrosomic fetus (2.3%) and 293 patients delivered non-macrosomic patients (97.7%).

91 patients belonged to over-weight (BMI 25- 29.9) group. Among them, 38 patients delivered macrosomic fetus (41.8%) and 53 patients delivered non-macrosomic fetus (58.2%).

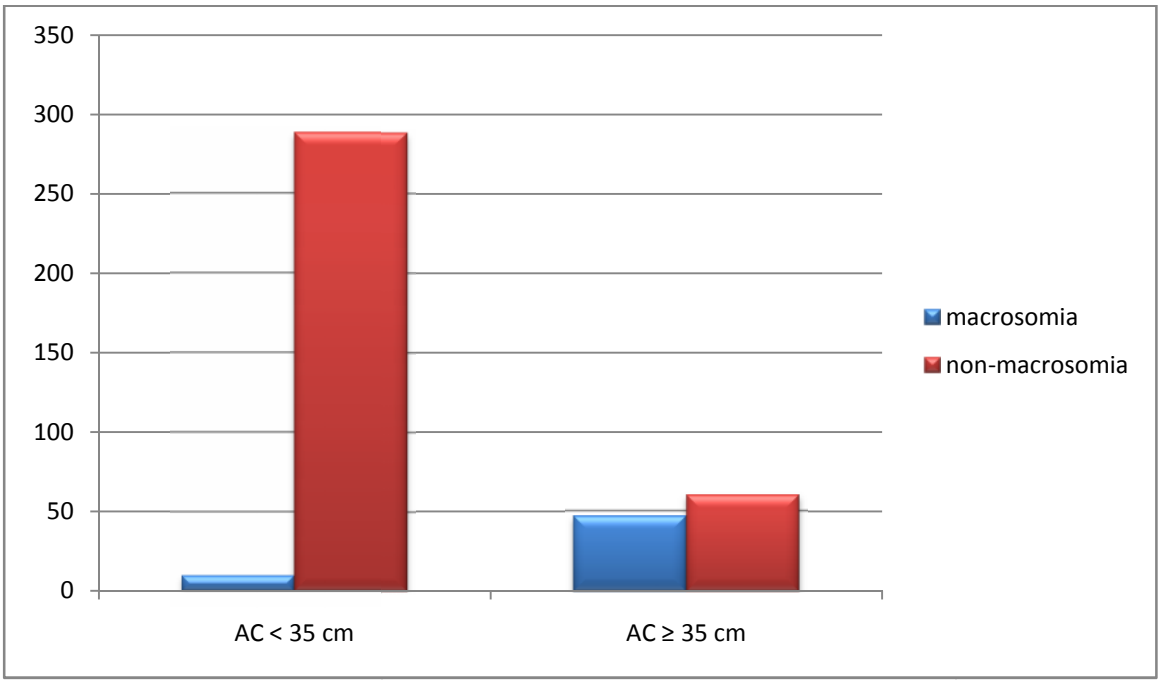
14 patients belonged to obese (BMI ≥ 30)group. Among them, 12 patients delivered macrosomic fetus (85.7%) and 2 patients delivered non-macrosomic fetus(14.3%)

Thus, as the BMI increases , the chance that a patient will deliver a macrosomic fetus increases.

ABDOMINAL CIRCUMFERENCE:

ABDOMINAL CIRCUMFERENCE	NO. OF PATIENTS	NO. OF MACROSOMIA	PERCENTAGE WITHIN MACROSOMIA	p-VALUE
AC< 35 cm	298	10	17.5	<0.001**
AC≥ 35 cm	107	47	82.5	
TOTAL	405	57	100	

**- significant at 1% level



Among 107 patients with AC ≥ 35 cm, 47 patients delivered macrosomic fetus (PPV- 43.9%) , 60 patients delivered non-macrosomic fetus.

Among 298 patients with AC <35 cm, 10 patients delivered macrosomic fetus and 288 patients delivered non-macrosomic fetus (NPV-96.6%)

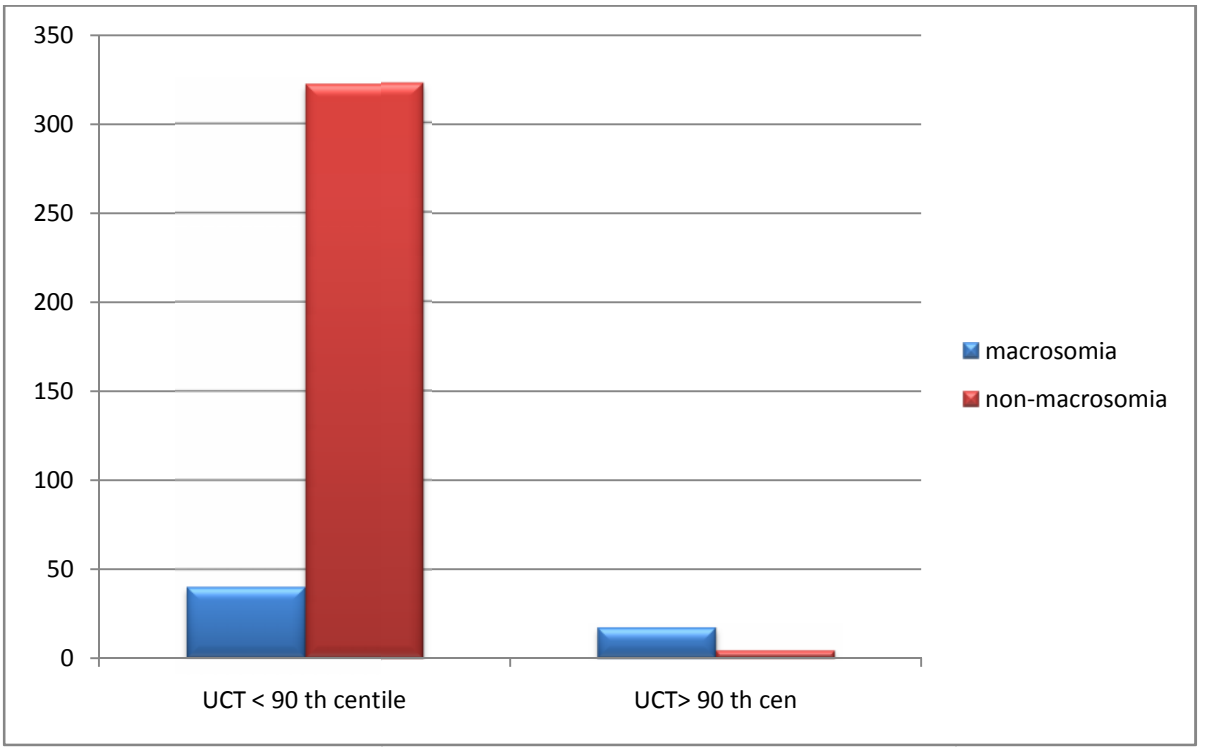
Among 57 patients who delivered macrosomic fetus, 47 patients had antenatal AC≥ 35cm(sensitivity-82.5%).

Among 348 patients who delivered non-macrosomic fetus, 288 patients had antenatal AC < 35cm(specificity-82.8%)

UMBILICAL CORD THICKNESS:

UMBILICAL CORD THICKNESS	NO. OF PATIENTS	NO. OF MACROSOMIA	PERCENTAGE	p-VALUE
BELOW 90^{TH} PERCENTILE	362	40	11.0	<0.001**
ABOVE 90^{TH} PERCENTILE	43	17	39.5	

**- significant at 1% level



Among 43 patients with umbilical cord thickness above 90^{th} percentile, 17 patients delivered macrosomic fetus. (PPV-39.5%)

Among 362 patients with umbilical cord thickness below 90th percentile, 322 patients delivered non-macrosomic fetus. (NPV-88.9%)

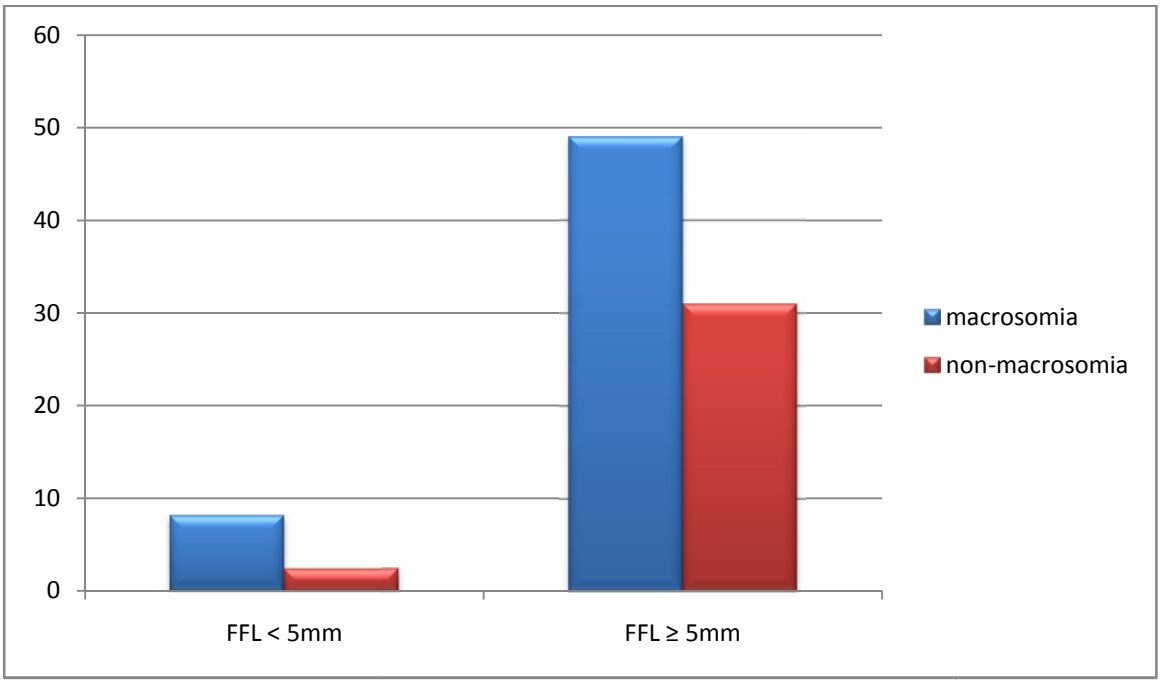
Among 57 patients who delivered macrosomic fetus, 17 patients had umbilical cord thickness above 90th percentile. (sensitivity-29.8%)

Among 348 patients who delivered non-macrosomic fetus, 322 patients had umbilical cord thickness below 90th percentile. (specificity-92.5%)

FETAL FAT LAYER:

FETAL FAT LAYER	NO.OF PATIENTS	NO. OF MACROSOMIA	PERCENTAGE WITHIN MACROSOMIA	p-VALUE
< 5 mm	325	8	14	<0.001**
≥ 5 mm	80	49	86	
TOTAL	405	57	100	

**- significant at 1% level



Among 80 patients with fetal fat layer ≥ 5 mm ,49 patients delivered macrosomic fetus.(PPV-61.3%)

Among 325 patients with fetal fat layer < 5mm, 317 patients delivered non-macrosomic fetus.(NPV97.5%)

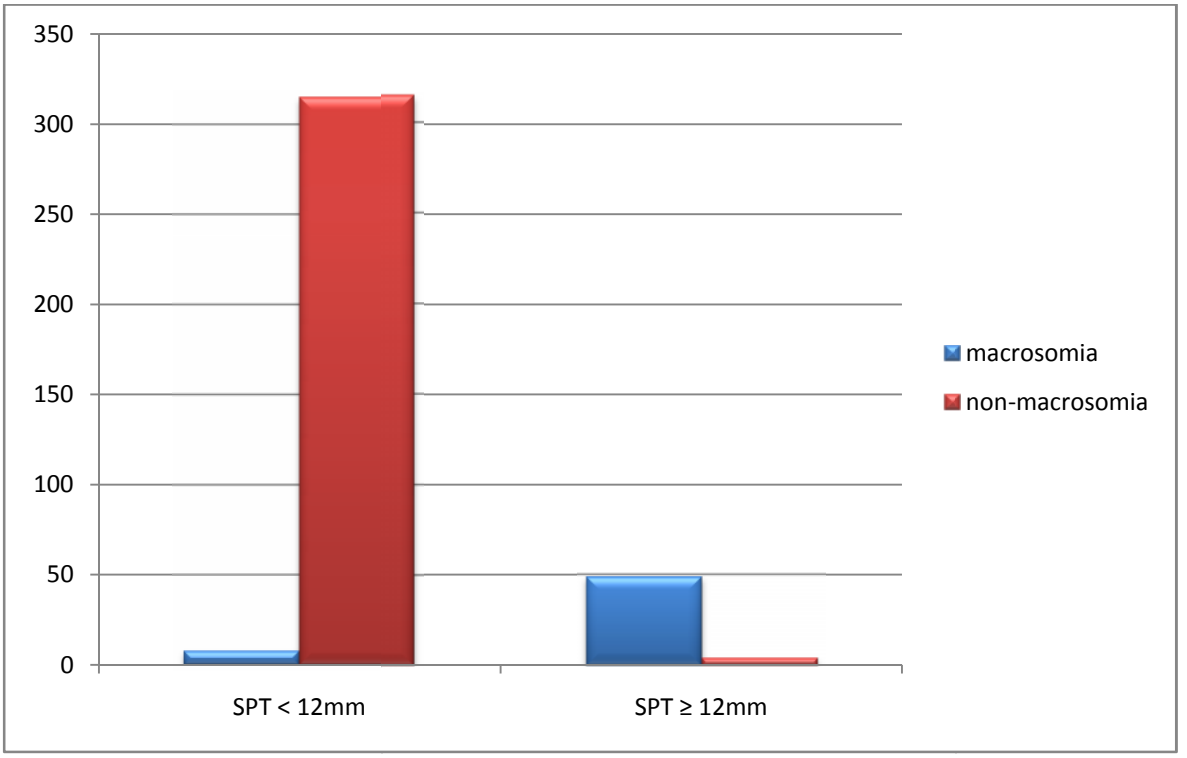
Among 57 patients who delivered macrosomic fetus, 49 patients had fetal fat layer ≥ 5mm.(sensitivity86%)

Among 348 patients who delivered non-macrosomic fetus, 317 patients had fetal fat layer < 5mm.(specificity-91.1%)

SHOULDER PAD THICKNESS:

SHOULDER PAD THICKNESS	MACROSOMIA		TOTAL	p-VALUE
	YES	NO		
< 12 mm	8	315	323	<0.001**
≥ 12 mm	49	33	82	
TOTAL	57	348	405	

**- significant at 1% level



Among 82 patients with shoulder pad thickness greater than 12 mm, 49 patients delivered macrosomic fetus. (PPV-59.8%)

Among 323 patients with shoulder pad thickness less than 12 mm, 315 patients delivered non-macrosomic fetus. (NPV-97.5%)

Among 57 patients who delivered macrosomic fetus, 49 had antenatal fetal shoulder pad thickness greater than 12 mm. (sensitivity-86%)

Among 348 patients who delivered non-macrosomic fetus, 315 had antenatal fetal shoulder pad thickness less than 12 mm. (specificity-90.5%)

MODE OF DELIVERY:

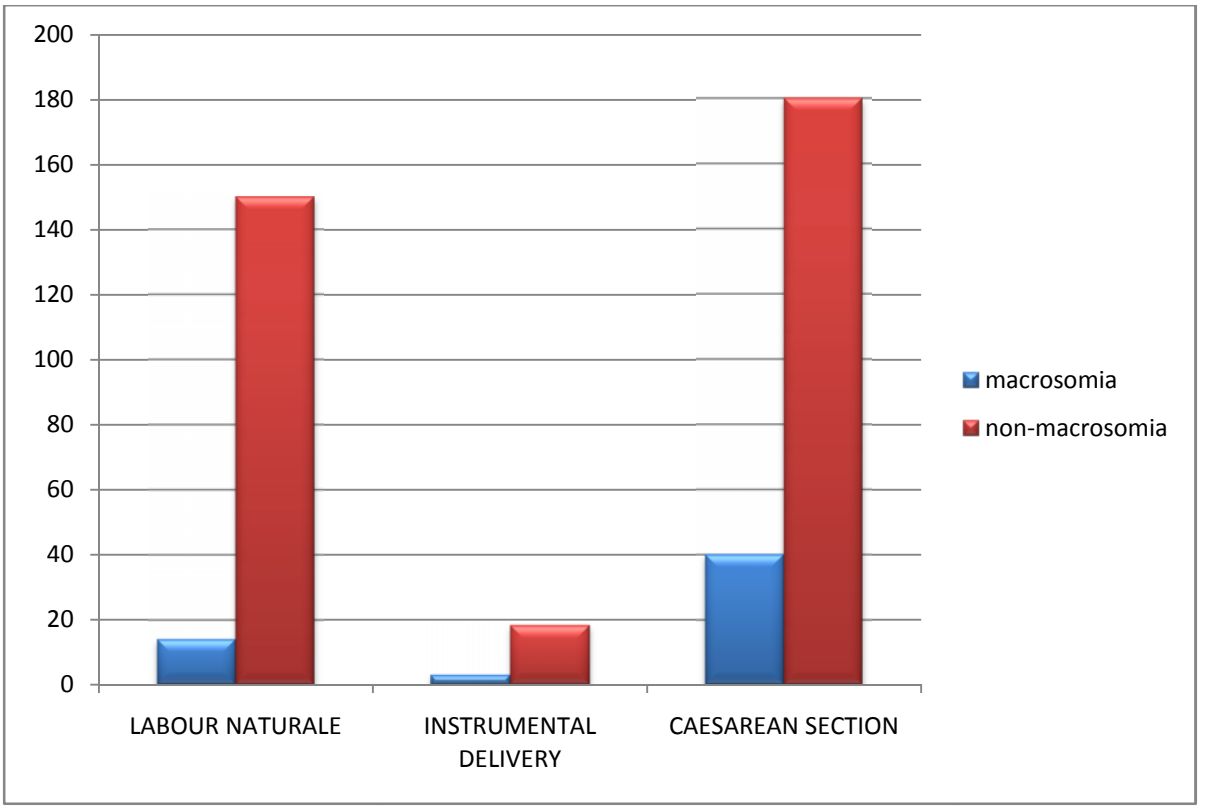
MODE OF DELIVERY	MACROSOMIA		TOTAL	p-VALUE
	YES	NO		
LABOUR NATURALE	14	150	164	
INSTRUMENTAL DELIVERY	3	18	21	0.006**
CAESAREAN DELIVERY	40	180	220	
TOTAL	57	348	405	

**- significant at 1% level

Among 57 patients who delivered macrosomic fetus, 40 patients needed caesarean section(70.2%),3 patients needed instrumental delivery(5.3%) and 14 patients delivered by labour naturale(24.6%)

Among 348 patients who delivered non-macrosomic fetus,180 patients needed caesarean section(51.7%), 18 patients needed instrumental delivery(5.2%) and 150 patients delivered by labour natural(43.1%)

Thus , from our study it is noted that, patients with macrosomic fetus were often being delivered by caesarean section than patients with non-macrosomic fetus.



BABY SEX:

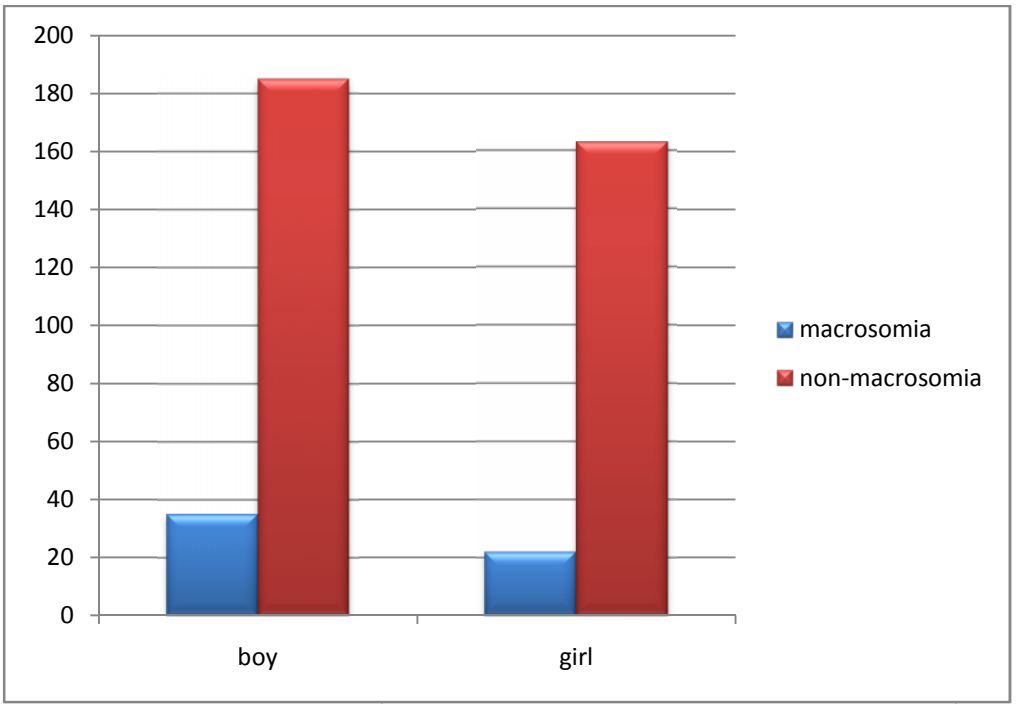
SEX	MACROSOMIA		TOTAL	p-VALUE
	YES	NO		
Boy	35	185	220	

GIRL	22	163	185	0.2(NS)
TOTAL	57	348	405	

NS- statistically not significant

Among 57 macrosomic babies, 35 were boys(61.4%) and 22 were girls(38.6%). This difference is not statistically significant.

Among 220 boys, 35 were macrosomic(15.9%) and among 185 girls, 22 were macrosomic(11.9%).



SHOULDER DYSTOCIA:

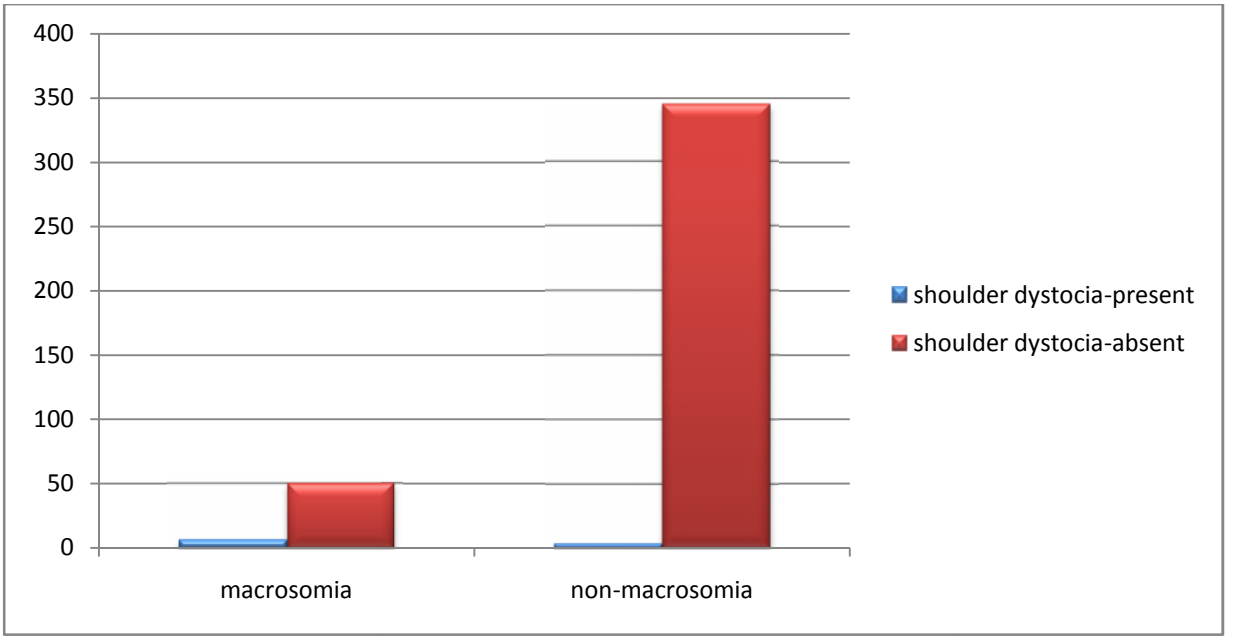
	SHOULDER DYSTOCIA		TOTAL	p-VALUE
	PRESENT	ABSENT		
MACROSOMIA	7	50	57	<0.001**
NON-MACROSOMIA	4	344	348	

	11	394	405	

**- significant at 1 % level

Among 57 deliveries of macrosomic babies, 7 were complicated by shoulder dystocia(12.3%)

Among 348 deliveries of non-macrosomic babies, 4 were complicated by shoulder dystocia(1.1%)



ESTIMATED FETAL WEIGHT AND MACROSOMIA:

Logistic regression analysis has been used to obtain EFW in predicting macrosomia.

Using the likelihood ratio Chi-Square, We obtain the p-value as <0.0001* which is significant. Therefore we conclude that the model is significant. R-

square is 42% indicates that there is a moderate relationship between the variables.

The logistic regression model is

Ln (Py/ (1-Py)) = (-18.285) + (4.545) * EFW

Parameter Estimates

Term	**P**	**Odds Ratio**	**OR (LI)**	**OR (UI)**
Constant	0.0000			
EFW in Kgs	0.0000	94.116	30.899	286.67

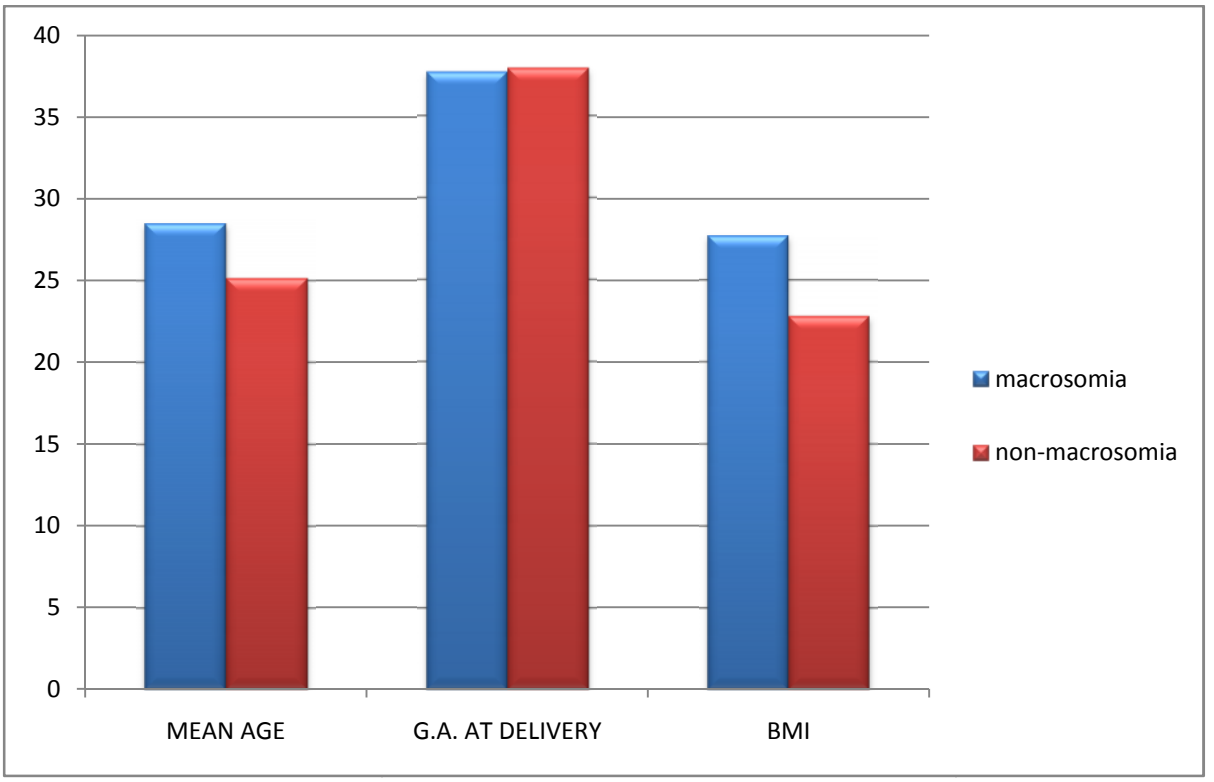
From the above table, EFW is significant in predicting Macrosomia.

Using Pearson, deviance and Hosmer- Lemeshow goodness of tests, we obtain the p-value as >0.05, hence we conclude that the logistic regression model fits the data well.

COMPARISON OF MATERNAL CHARACTERISTICS:

	< 4 kg MEAN ± SD	>4 kg MEAN ± SD	P- value	S / NS
MATERNAL AGE	25.15 ± 3.53	28.5 ± 4.2	<0.001	S
GESTATIONAL AGE AT DELIVERY	37.9 ± 0.84	37.67 ± 0.87	0.4726	NS

BMI	22.8 ± 1.88	27.72 ± 2.27	<0.001	S



The mean age of patients who delivered non-macrosomic fetus was 25.15 and the mean age of patients who delivered macrosomic fetus was 28.5. This difference was statistically significant with p-value of < 0.001.

The mean gestational age at delivery in macrosomia group and non-macrosomia group were 37.9 and 37.67 respectively. There was no significant statistical difference.

The mean BMI of patients who delivered non-macrosomic fetus was 22.8 and the mean BMI of patients who delivered macrosmic fetus was 27.72. This difference was statistically significant with a p-value of < 0.001.

DISCUSSION

In pregnancies complicated by diabetes mellitus, fetal macrosomia is common. Macrosomia can lead to adverse maternal and perinatal consequences. Antenatal prediction of macrosomia helps in identification of high risk patients such that appropriate intrapartum precautions can be taken. There are several studies on prediction of fetal macrosomia. In our study we evaluated the usefulness of sonographic measurement of umbilical cord thickness, fetal fat layer, shoulder pad thickness as predictors of macrosomia in pregnancies complicated by diabetes.

405 antenatal mothers with GDM or pre GDM were included in our study. Routine history taking, clinical examination and ultrasound examination were done. Special measurements namely umbilical cord thickness, fetal fat layer and shoulder pad thickness were taken using ultrasound. The patients were then followed up at the time of delivery.

MATERNAL CHARACTERISTICS:

AGE:

In our study among 57 macrosomic fetus a majority (43.9%) were delivered by mothers in age group of 26-30 years because a majority of the study population belong to this age group. The chance of macrosomia among the particular age group was highest in 36-40 years. In the age group of 36-40 years, 66.7%

delivered macrosomic babies as compared to 15.1% in age group of 26-30 years. Thus maternal age greater than 35 years was significantly associated with increased risk of macrosomia.

Karim et al analysed prevalence and outcome of macrosomia in Pakistan. He reported that maternal age over 35 years was associated with fetal macrosomia.[35]

Weissmann et al reported that maternal age was significantly higher among macrosomic neonates.[36]

Meshari et al reported similar results in their study in Saudi Arabia.

PARITY:

In our study population majority of the patients (44%) were nulliparous women. Hence majority (33.3%) of macrosomia occurred in this group. The risk of delivering a macrosomic baby within a group was highest among para- 4 group. Thus as parity increases the chance of delivering macrosomic fetus also increases.

Karim et al reported that grand multiparity was associated with macrosomia.[35]

Mahin Najafian et al reported that multiparity was significantly associated with macrosomia.[37]

GESTATIONAL AGE AT DELIVERY:

Our study included the patients with diabetes and they were followed up from 36 weeks of gestation until delivery. Hence post maturity was not allowed. The mean gestational age at delivery in macrosomia and non macrosomia group were 37.9 ± 0.84 and 37.67 ± 0.87 respectively. The difference was not statistically significant.

Karim et al reported that post maturity was associated with macrosomia.[35]

BMI:

In our study among 57 patients who delivered macrosomic babies, 91.2 % had BMI ≥ 26. In group with BMI ≥ 30 about 85.7% delivered macrosomic babies. Thus maternal BMI has significant association with macrosomia.

Karim et al reported that maternal obesity was associated with macrosomia.[35]

Mahin Najafian et al reported similar results.[37]

ULTRASOUND MEASUREMENTS:

ABDOMINAL CIRCUMFERENCE:

In our study a cut of abdominal circumference ≥ 35 cms in predicting macrosomia had a sensitivity of 82.5%, specificity of 82.8%, positive predictive value of 43.9 %, negative predictive value of 96.6%. Thus abdominal circumference ≥ 35 cms has got good sensitivity, specificity and negative

predictive value. Though the positive predictive value is low when abdominal circumference ≥ 35 cms, obstetrician must confirm the diagnosis by doing further evaluations.

Ratchianikon et al evaluated the diagnostic value of sonographic measurement of fetal abdominal circumference in predicting macrosomia. [38] They concluded that abdominal circumference ≥ 35 cms as predicter of macrosomia was useful with sensitivity of 87.5 %, specificity of 84.7%, positive predictive value of 41.67 %, negative predictive value of 98.19%.

UMBILICAL CORD THICKNESS:

In our study umbilical cord thickness had low sensitivity, but good specificity and negative predictive value. Hence, if umbilical cord thickness is less than 90^{th} centile the chance of macrosomia is less.

Cromi et al reported that the proportion of cases with large umbilical cord was significantly higher in the group of macrosomic compared to non macrosomic infants (54.7% vs 8.7%). [32]

FETAL FAT LAYER:

In our study the cut off of fetal fat layer ≥ 5 mm as predicter of macrosomia had sensitivity of 86% and specificity of 91.1%. Thus fetal fat layer ≥ 5 mm is a reliable predictor of macrosomia.

M. Bethune et al reported that fetal fat layer ≥ 5 mm was the most useful predictor of macrosomia at term. He reported that the risk of macrosomia increases ten fold when fetal fat layer ≥ 5 mm. [39]

SHOULDER PAD THICKNESS:

In our study a cut off of shoulder pad thickness ≥ 12 mm as predictor of macrosomia had sensitivity of 86 %, specificity of 90.5%, positive predictive value of 59.8 %. Thus shoulder pad thickness ≥ 12 mm is a reliable predictor of macrosomia.

Nadia et al reported the sonographic measurement of fetal shoulder pad thickness positively correlated with new born weight. In their study shoulder pad thickness ≥ 12 mm had sensitivity of 87.2 %, specificity of 74.2%, positive predictive value of 84.2 %. [29]

Landon et al reported similar results. [31]

ESTIMATED FETAL WEIGHT:

In our study multiple regression model was used to analyse estimated fetal weight as predictor of fetal macrosomia. It was statistically significant.

Nadia et al reported that sonographic fetal weight estimation as predictor of macrosomia was useful with sensitivity of 74.5%, specificity of 85.7%.[29]

SHOULDER DYSTOCIA:

In our study 12.3 % of deliveries of macrosomic fetus were complicated by shoulder dystocia as against 1.1% among non macrosomia group. The shoulder dystocia is much more common in macrosomic fetus than non macrosomic fetus. In pregnancies complicated by diabetes, shoulder pad thickness is higher predisposing them to shoulder dystocia.

Mahin Najafian et al reported shoulder dystocia in 11% of macrosomia group as against 0.5% in non macrosomia group.[37]

Mohammed Alkahaten et al reported that shoulder dystocia occurred in 9.6 % of macrosomia.[40]

FETAL GENDER:

In our study among 57 macrosomic babies 61.4 % were boys and 38.6 % were girls. But this difference was not statistically significant.

Mahin Najafian et al reported that fetal sex influences macrosomia tendency. Male infants weigh more than female infants at any gestational age.[37]

MODE OF DELIVERY:

In our study 70.2 % of macrosomic fetus needed caesarean section. Thus, macrosomia increases the rate of caesarean section.

Batuhan et al reported caesarean section rate of 58.7 % in birth weight of 4 - 4.249 kg and 63.6 % in birth weight of 4.250 - 4.5 kg. [41]

Mahin Najafian et al reported that the incidence of caesarean section in macrosomic group was 89 % compared to 11 % in non macrosomia group. [37]

SUMMARY

405 antenatal mothers with GDM or pre GDM were included in our study. This study was conducted to analyse the usefulness of soft tissue measurements in predicting macrosomia in pregnancy complicated by gestational and pre gestational diabetes. In addition, the association between maternal characteristics and macrosomia were studied.

- Among the maternal characteristics increased maternal age, parity and BMI were significantly associated with macrosomia.
- Among the ultrasound parameters, a cut of abdominal circumference ≥ 35 cms, fetal fat layer ≥ 5mm, shoulder pad thickness ≥ 12 mm had good sensitivity, specificity and negative predictive value as predictors of macrosomia. Umbilical cord thickness greater than 90th percentile had good negative predictive value.
- 70.2 % of macrosomic fetus needed caesarean section. Thus, macrosomia increases the rate of caesarean section.
- 12.3 % of deliveries of macrosomic fetus were complicated by shoulder dystocia as against 1.1% among non macrosomia group. The shoulder dystocia is much more common in macrosomic fetus than non macrosomic fetus.

CONCLUSION

Macrosomia is every obstetrician’s nightmare and hence clinicians are in constant search for better methods of prediction. Fetal fat layer and shoulder pad thickness are good predictors of fetal macrosomia. In the assessment of risk of macrosomia in addition to the ultrasonographic measurements the clinical risk factors must be considered. Further studies are needed to evaluate the clinical value of incorporating these soft tissue measurements in formulas for estimation of fetal weight.

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PROFORMA

NAME AGE IP NO

ADDRESS

CONTACT NO.

OBSTETRIC SCORE

LMP EDD GA

GA BY DATING SCAN

MENSTRUAL HISTORY

MARITAL HISTORY

OBSTETRIC HISTORY

MEDICAL HISTORY

GENERAL EXAMINATION

PRE PREGNANCY WEIGHT PRESENT WEIGHT

HEIGHT WEIGHT GAIN

BMI

VITALS

ABDOMINAL EXAMINATION

INVESTIGATIONS:

URINE SUGAR

HB

OGCT OGTT

FBS PPBS

USG :

BPD FL AC

AFI FH

SPECIAL MEASUREMENTS:

UMBLICAL COIL THICKNESS

SHOULDER PAD THICKNESS

FETAL FAT LAYER

DELIVERY DETAILS:

MODE OF DELIVERY

LABOUR NATURALE

INSTRUMENTAL DELIVERY

CAESAREAN

BABY DETAILS:

TERM/PRETERM SEX

BIRTH WEIGHT APGAR

COMPLICATIONS

CONSENT FORM

STUDY TITLE :

PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FETAL FAT LAYER, AND SHOULDER PAD THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL AND PREGESTATIONAL DIABETES MELLITUS

STUDY CENTRE :

INSTITUTE OF OBSTETRICS AND GYNAECOLOGY, EGMORE, CHENNAI

PARTICIPANT NAME : **AGE:** **SEX :** **LD. NO.**

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

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

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

I hereby consent to participate in this study titled **"PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FETEL FAT LAYER, AND SHOULDER PAD THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL AND PREGESTATIONAL DIABETES MELLITUS"**

Signature of Investigator Place

Study Investigators Name Date

Signature/thumb impression of patient Institute

S.No	NAME	AGE	OBS. SCORE	parity	GA AT DEL(wks)	BMI	EFW	AC	UC THICKNESS	FFL	SP THICKNESS	mode of delivery	SEX	BIRTH WT	MACROSOMIA	APGAR		SD (+/-)
1.															YES/NO	1MIN	5MIN	
2.	Amulu	26	primi	0	38	31	4	36	3	5	12	EM LSCS	boy	4	yes	8	9	-
3.	Aparna	26	G2P1L1	1	37	24	3.2	32	2.2	3	11	LN	boy	3	no	8	9	-
4.	Deepika	23	G2A1	0	37	23	3.5	36	2.5	3	12	EM LSCS	girl	4	no	7	8	-
5.	Bakiyam	28	G3P2L2	2	38	22	3	30	2.4	4	11	EM LSCS	girl	3	no	7	8	-
6.	Hemalatha	21	primi	0	38	23	3	32	2.5	4	11	EM LSCS	boy	3	no	8	9	-
7.	Abitha	20	primi	0	38	24	3	33	2.6	4	11	EM LSCS	boy	3	no	8	9	-
8.	Adhilakshmi	24	G2P1L1	1	38	25	2.5	33	2.6	4	10	LN	boy	3	no	8	9	-
9.	Chandrakala	25	G3A2	0	38	26	3.3	32	2.5	3	9	EM LSCS	girl	3	no	7	8	-
10.	Sivagami	28	G2P1L1	1	39	30	4.1	35	3.1	5	12	EL LSCS	boy	4	yes	8	9	-
11.	Gandhimathi	26	G3P2L2	2	38	24	3	31	2.4	3	10	LN	boy	3	no	7	8	-
12.	Ambika	25	primi	0	37	22	3	30	2.4	3	9	EM LSCS	girl	3	no	7	8	-
13.	Bhavani	24	primi	0	37	20	3.5	30	2.2	4	9	LN	boy	3	no	8	9	-
14.	Indra	29	G2P1L1	1	37	23	4	31	2.7	5	13	EM LSCS	girl	4	no	7	8	-
15.	Kayilvani	26	G2P1L1	1	37	23	3	31	2.7	4	10	EL LSCS	boy	3	no	8	9	-
16.	Elavarasi	27	G3P1L1A1	1	38	22	2.8	35	2.8	3	10	LN	boy	3	no	8	9	-
17.	Kalaivani	30	G3P2L1	2	37	29	4	35	2.9	5	13	EM LSCS	boy	4	yes	7	8	-
18.	Julie	20	primi	0	38	21	3.3	36	3	3	11	FORCEPS	girl	4	no	6	7	-
19.	Ammakannu	27	G3P2L1	2	38	20	3.3	32	2.9	5	11	LN	boy	3	no	8	9	-
20.	Vanitha	30	G3P2L2	2	37	27	4.4	33	3.2	6	13	EM LSCS	girl	4	yes	7	8	-
21.	Kalaimagal	28	G2P1L1	1	37	19	2.8	32	2.9	4	10	LN	girl	3	no	7	8	-
22.	Narayani	19	primi	0	37	24	3	31	2.8	5	9	EM LSCS	boy	3	no	7	8	-
23.	Banumathy	30	G3P2L2	2	37	25	3.5	35	2.7	3	10	LN	boy	4	no	7	8	-
24.	Chitra	31	G4P3L3	3	39	26	3	33	2.6	4	9	LN	boy	3	no	8	9	-
25.	Floret	29	G3P1L1A1	1	39	24	4	33	2.8	5	12	EM LSCS	boy	4	no	8	9	-

26.	Ammu	22	primi	0	38	25	3	30	2.5	3	10	FORCEPS	girl	4	no	6	7	-
27.	Loganayaki	25	G2P1L1	1	38	26	3	30	2.4	4	10	LN	girl	3	no	7	8	-
28.	Manimala	26	G2P1L1	1	38	24	3.2	31	2.3	3	11	EL LSCS	girl	3	no	8	9	-
29.	Oorvasi	27	G3P1L1A1	1	39	23	3.5	32	2.5	4	10	FORCEPS	girl	4	no	6	8	+
30.	Lidiya	23	primi	0	37	22	3.5	33	2.2	3	9	EM LSCS	boy	4	no	8	9	-
31.	Gowri	29	G2P1L1	1	38	31	4	36	3.3	6	14	EL LSCS	girl	4	yes	7	8	-
32.	Anandha	24	G3A2	0	37	21	3.5	34	2.1	3	10	LN	girl	3	no	8	9	-
33.	Balambigai	25	G2P1L1	1	38	20	3	37	3.3	3	9	EL LSCS	boy	3	no	7	8	-
34.	Muthuselvi	26	G2P1L1	1	38	20	3.2	34	2.4	3	11	EM LSCS	boy	3	no	7	8	-
35.	Nisha	22	primi	0	38	21	4	34	2.6	4	11	EM LSCS	girl	4	no	7	8	-
36.	Kalaiselvi	22	G2P1L1	1	38	22	3	34	2.4	4	10	LN	boy	3	no	8	9	-
37.	Anitha	21	primi	0	39	22	3.8	32	2.4	3	11	EM LSCS	boy	4	no	8	9	-
38.	Margaret	20	primi	0	38	23	3.5	30	2.6	4	11	EM LSCS	boy	4	no	7	8	-
39.	Chitra	25	G2P1L1	1	37	24	2.5	32	2.8	3	11	LN	girl	3	no	7	8	-
40.	Poongodi	27	G2A1	0	37	25	2.5	33	2.8	3	10	LN	boy	3	no	8	9	-
41.	Savitha	28	G3P2L2	2	37	25	2.5	34	2.5	3	10	LN	boy	3	no	7	8	-
42.	Gunasundari	24	primi	0	38	30	4	34	3	5	12	EM LSCS	boy	4	yes	8	9	-
43.	Vasantha	27	G3A2	0	38	26	4	36	3.2	4	12	EL LSCS	boy	4	no	7	8	-
44.	Kasthuri	25	primi	0	37	26	2.5	34	2.2	3	9	LN	Girl	3	no	8	9	-
45.	Ambika	26	G2P1L1	1	38	23	3	34	2.4	3	9	EM LSCS	boy	3	no	7	8	-
46.	Pachiammal	28	G3P1L1A1	1	38	23	2.5	33	2.6	4	10	LN	boy	3	no	7	8	-
47.	Vijaya	30	G2P1L1	1	39	22	2.6	33	2.7	3	10	LN	girl	3	no	7	8	-
48.	Kala	24	G3P1L1A1	1	38	21	3.5	32	2.7	4	11	FORCEPS	boy	4	no	7	8	-
49.	Stella	26	G2P1L1	1	38	20	2.8	31	2.8	3	10	LN	girl	3	no	8	9	-
50.	Vasanthi	22	primi	0	37	22	3	36	3.2	5	9	LN	boy	3	no	7	8	-
51.	Muniyammal	22	primi	0	37	22	3.5	36	3	3	10	EM LSCS	girl	3	no	8	9	-
52.	Kamala	25	primi	0	38	28	4.1	35	3.1	5	11	EM LSCS	boy	4	yes	7	8	-

53.	Nirmala	24	G2P1L1	1	38	24	3.2	34	2.5	3	11	EM LSCS	boy	3	no	7	8	-
54.	Anu	25	G2A1	0	38	24	3.2	33	2.4	4	11	LN	boy	4	no	8	9	-
55.	Padma	26	G2P1L1	1	39	23	3	32	2.2	4	10	EL LSCS	boy	3	no	7	8	-
56.	Priya	28	G4P2L2A1	2	39	26	2.8	32	2	3	10	LN	boy	3	no	7	8	-
57.	Chandrakala	21	primi	0	38	22	3.5	34	2.2	5	9	EM LSCS	girl	3	no	7	8	-
58.	Noornisha	25	G2P1L1	1	37	24	3	34	2.2	3	9	EM LSCS	boy	3	no	8	9	-
59.	Shweta	26	G2P1L1	1	37	19	3	35	2.4	3	10	EM LSCS	boy	3	no	7	8	-
60.	Suganya	28	G2A1	0	37	20	4	34	2.3	4	11	EL LSCS	girl	4	no	7	8	-
61.	Nitya	31	G3P1L1A1	1	37	21	3.6	34	2.4	4	11	EM LSCS	girl	3	no	7	8	-
62.	Gayathri	32	G4P3L3	3	38	22	2.8	36	3	3	10	LN	boy	3	no	8	9	-
63.	Girija	29	G2P1L0	1	38	23	4.2	35	3.3	5	13	EL LSCS	boy	4	no	7	8	-
64.	Susila	34	G4P3L3	3	39	31	4.2	37	3.4	5	14	EL LSCS	boy	4	yes	8	9	-
65.	Kalavathi	21	primi	0	38	24	2.8	34	2.6	3	9	LN	boy	3	no	7	8	-
66.	Logammbal	19	primi	0	37	26	2.5	34	2.7	3	9	LN	girl	3	no	8	9	-
67.	Varalakshmi	27	G2P1L1	1	37	25	3.5	33	2.7	4	10	EM LSCS	girl	3	no	7	8	-
68.	Mithra	23	primi	0	37	27	3.4	32	2.5	4	11	LN	girl	3	no	8	9	-
69.	Kanaga	19	primi	0	39	25	4	35	3.2	4	13	EM LSCS	boy	4	no	7	8	-
70.	Godavari	20	primi	0	39	24	3.6	36	2.9	4	11	EM LSCS	boy	4	no	7	8	-
71.	Devi	24	G2P1L1	1	39	23	3.6	32	2.8	4	11	EL LSCS	boy	3	no	8	9	-
72.	Vasumathi	25	G2P1L1	1	38	22	3.5	33	2.4	3	10	EL LSCS	girl	3	no	8	9	-
73.	Parvathi	31	G2P1L1	1	39	30	4.1	36	3.3	5	14	EL LSCS	girl	4	yes	8	9	-
74.	Kaliammal	24	primi	0	39	22	3.5	33	2.2	3	11	EM LSCS	boy	4	no	8	9	-
75.	Uma	23	primi	0	38	20	3	31	2.1	3	10	LN	girl	3	no	7	8	-
76.	Brindha	24	primi	0	37	21	3.2	30	2.4	5	9	EM LSCS	boy	3	no	7	8	-
77.	Karunya	20	primi	0	38	21	2.8	30	2.6	4	9	LN	boy	3	no	7	8	-
78.	Sarika	30	G3P2L2	2	40	29	4	36	3.3	4	12	EM LSCS	girl	4	no	8	9	-
79.	Gandhimathi	24	primi	0	37	24	3.1	32	2.5	4	10	LN	boy	3	no	8	9	-
80.	Kuyili	26	G2P1L1	1	38	24	3.3	33	2.7	3	10	LN	boy	3	no	8	9	-
81.	Malliga	22	primi	0	37	29	3.9	37	3.2	5	12	EM LSCS	girl	4	yes	8	9	-

82.	Sheeba	28	G2A1	0	38	23	3	34	2.4	4	11	EM LSCS	girl	3	no	7	8	-
83.	Vishalakshi	31	G3P1L1A1	1	39	22	3.2	34	2.5	3	11	FORCEPS	boy	3	no	6	8	-
84.	Venda	26	G3P1L1A1	1	38	21	2.8	33	2	3	11	EM LSCS	girl	3	no	7	8	-
85.	Malar	22	G2P1L1	1	38	21	2.7	32	2.2	4	10	LN	boy	3	no	7	8	-
86.	Ashwini	20	primi	0	37	20	4	31	3	5	13	EL LSCS	boy	4	no	7	8	-
87.	Balambigai	21	G3A2	0	37	19	3	30	2.5	3	11	EM LSCS	boy	3	no	8	9	-
88.	Caroline	28	G2P1L1	1	38	20	2.8	31	2.5	3	11	EM LSCS	boy	3	no	7	8	-
89.	Bakiya	27	G2P1L0	1	37	28	4.1	37	3.2	5	14	EL LSCS	boy	4	yes	7	8	-
90.	Sheela	26	G2P1L1	1	38	21	2.5	31	2.6	3	9	LN	girl	3	no	8	9	-
91.	Devaki	28	G3P1L1A1	1	40	22	2.5	32	2.6	4	11	EM LSCS	girl	3	no	7	8	-
92.	Chandra	21	primi	0	38	23	2.6	33	2.4	4	9	LN	boy	3	no	8	9	-
93.	Kumari	24	G2A1	0	38	23	3.1	34	2.3	4	11	LN	girl	3	no	8	9	-
94.	Radha	28	primi	0	38	24	3.2	34	2.2	3	11	EM LSCS	boy	3	no	8	9	-
95.	Ishwariya	26	G2P1L1	1	37	25	2.4	36	3.2	5	10	LN	girl	3	no	7	8	-
96.	Viji	29	G2P1L1	1	37	24	2.2	33	2.5	3	9	LN	boy	3	no	7	8	-
97.	Piraimathy	40	Primi	0	37	28	4.3	36	3.4	4	13	EL LSCS	boy	5	yes	7	8	-
98.	Asha	22	primi	0	38	24	3.9	36	2.6	3	12	EM LSCS	girl	4	no	7	8	-
99.	Nandhini	24	primi	0	38	23	2.2	32	2.4	3	10	LN	boy	3	no	8	9	-
100.	Malarkodi	27	G2P1L1	1	39	22	2.4	32	2.4	3	11	EM LSCS	girl	3	no	8	9	-
101.	Bama	32	G3P2L2	2	38	21	3.5	31	2.2	4	10	EM LSCS	girl	3	no	8	9	-
102.	Saras	30	G3P1L1A1	1	38	20	2.5	30	2.2	4	9	LN	boy	3	no	7	8	-
103.	Selvi	20	primi	0	38	24	3.9	32	2.4	3	11	EM LSCS	boy	4	no	8	9	-
104.	Tamilselvi	24	G2P1L1	1	37	26	4.1	36	3.3	4	13	EM LSCS	boy	4	no	6	8	-
105.	Suganti	32	G3P2L2	2	37	26	3.9	36	3	5	13	LN	girl	4	yes	7	8	+
106.	Karthika	22	primi	0	38	22	3.2	33	2.6	4	11	EM LSCS	boy	4	no	7	8	-
107.	Vidya	23	primi	0	37	23	3.5	34	2.5	3	11	LN	boy	3	no	7	8	-
108.	Gayathri	26	G3P1L1A1	1	37	23	3	34	2.5	3	9	EM LSCS	boy	3	no	8	9	-
109.	Swarna	25	G2P1L1	1	38	22	3.2	34	2.4	4	10	EM LSCS	girl	3	no	7	8	-
110.	Pandiammal	24	G2A1	0	39	20	3.5	33	2.4	3	11	EM LSCS	girl	3	no	7	8	-

111.	Priya	28	primi	0	39	21	2.2	30	3.2	3	9	LN	boy	3	no	6	8	-
112.	Sakuntala	35	G5P4L4	4	37	32	4.3	37	3	5	12	EM LSCS	boy	5	yes	7	8	-
113.	Baby	19	primi	0	39	25	3	36	2.2	4	10	LN	boy	3	no	8	9	-
114.	Girija	21	primi	0	39	26	2.9	36	2.1	3	11	EM LSCS	girl	3	no	8	9	-
115.	Sasi	25	G2P1L1	1	38	22	3	32	2.4	4	11	EM LSCS	boy	3	no	7	8	-
116.	Renuka	26	primi	0	38	22	2.6	33	2.5	3	9	LN	girl	3	no	8	9	-
117.	Vijaya	28	G3P1L1A1	1	38	23	4	34	2.8	4	12	EM LSCS	girl	4	no	8	9	-
118.	Lakshmi	29	G3P2L2	2	37	24	3.4	33	2.6	5	11	LN	boy	4	no	6	8	-
119.	Lurdh	34	G4P2L2A1	2	39	23	3	35	2.4	4	10	LN	boy	3	no	7	8	-
120.	Mary	29	G3P2L2	2	37	22	2.9	32	2.6	3	9	LN	girl	3	no	7	8	-
121.	Ragini	28	G2P1L1	1	38	22	2.9	32	2.5	4	9	EM LSCS	girl	3	no	7	8	-
122.	Fathima	26	G2P1L0	1	38	24	4	34	2.6	5	13	EM LSCS	girl	4	no	7	8	-
123.	Philomeena	20	primi	0	37	21	2.7	30	2.2	3	10	LN	boy	3	no	7	8	-
124.	Vinodhini	19	primi	0	37	20	2.8	31	2	5	10	LN	boy	3	no	7	8	-
125.	Gunavathy	23	primi	0	39	26	4.1	37	3.1	6	14	EL LSCS	boy	4	yes	7	8	-
126.	Deepa	24	G2P1L1	1	40	20	3.3	33	2.1	4	11	EL LSCS	Girl	3	no	8	9	-
127.	Narmada	22	primi	0	39	21	3.12	34	2.2	3	9	LN	boy	3	no	8	9	-
128.	Sharmili	27	G2P1L1	1	39	23	3	32	2.2	4	9	LN	Girl	3	no	8	9	-
129.	Kalaimagal	23	primi	0	38	23	3.2	32	2.5	4	10	FORCEPS	boy	3	no	8	9	-
130.	Leela	30	G3P1L1A1	1	38	24	3.6	33	2.4	3	11	LN	boy	4	no	7	8	-
131.	Nadhiya	29	G3A2	0	37	24	3.5	36	3.2	4	11	EL LSCS	Girl	4	no	7	8	-
132.	Latha	25	primi	0	37	24	3.8	34	3.1	5	11	LN	Girl	4	no	5	8	+
133.	Neela	35	G3P2L2	2	40	24	3	34	3	3	9	LN	boy	3	no	8	9	-
134.	Soniya	30	G2P1L1	1	37	22	3	31	2.8	4	10	EL LSCS	boy	3	no	8	9	-
135.	Sameera	26	G2P1L1	1	38	27	3.9	36	2.9	6	12	EM LSCS	girl	4	yes	7	8	-
136.	Kumari	26	G2P1L1	1	38	26	3.6	35	3	3	11	EL LSCS	boy	4	no	8	9	-
137.	Vaijainthi	27	G2P1L1	1	38	23	3.5	32	2.4	3	11	EL LSCS	Girl	4	no	7	8	-
138.	Radhika	29	G3P2L1	2	37	22	3.2	32	2.4	4	11	LN	boy	3	no	7	8	-
139.	Divya	23	G2A1	0	37	22	3.2	31	2.2	3	10	EM LSCS	boy	3	no	8	9	-

140.	Punitha	21	primi	0	38	21	3	31	2.5	3	10	EM LSCS	Girl	3	no	7	8	-
141.	Prema	20	primi	0	38	20	3.2	30	2.6	3	9	LN	boy	3	no	8	9	-
142.	Ashwini	23	G2P1L1	1	39	21	3	33	2.2	4	9	LN	Girl	3	no	7	8	-
143.	Mariyammal	25	primi	0	37	27	4	36	3.2	4	11	EM LSCS	Girl	4	yes	7	8	-
144.	Kannathal	25	primi	0	39	26	2.6	36	3.2	3	10	LN	boy	3	no	8	9	-
145.	Jannet	26	G2P1L1	1	38	22	4	35	2.6	4	13	EM LSCS	Girl	4	no	8	9	-
146.	Fathima	28	G3P2L2	2	38	23	3.2	35	2.4	3	11	LN	Girl	3	no	7	8	-
147.	Akila	21	primi	0	37	22	3.5	33	2.5	3	10	EM LSCS	boy	3	no	8	9	-
148.	Amaravathi	20	primi	0	37	21	2.7	34	2.5	4	9	LN	girl	3	no	8	9	-
149.	Sowmya	19	primi	0	38	21	3	34	2.4	4	11	LN	boy	3	no	8	9	-
150.	Kaveri	25	G2P1L1	1	38	20	3	33	2.4	3	11	FORCEPS	girl	3	no	8	9	-
151.	Bindhu	22	primi	0	39	19	3.5	32	2.2	4	10	LN	girl	3	no	7	8	-
152.	Florence	29	G3P2L1	2	38	32	4.5	38	3.3	6	14	EL LSCS	boy	5	yes	8	9	-
153.	Gujalambal	26	G3P2L2	2	40	20	2.5	32	2	3	9	EM LSCS	boy	3	no	7	8	-
154.	Vinodhini	22	primi	0	39	27	2.5	36	2.7	3	9	LN	girl	3	no	8	9	-
155.	Rasathi	28	G2P1L1	1	39	22	2.5	33	2.5	4	9	LN	girl	3	no	8	9	-
156.	Tamilselvi	24	G3P1L1A1	1	38	23	3.5	34	2.4	4	10	EM LSCS	boy	3	no	8	9	-
157.	Vani	23	G2A1	0	38	24	3	34	2.2	3	11	EM LSCS	boy	3	no	7	8	-
158.	Vahita	26	G3P1L1A1	1	37	24	3	32	2.2	3	11	EM LSCS	girl	3	no	7	8	-
159.	Amirtham	27	G2P1L1	1	37	22	4.2	32	2.4	5	13	LN	boy	4	no	8	9	-
160.	Aalaya	28	G2P1L1	1	39	25	3.5	36	3.2	4	11	EM LSCS	girl	3	no	8	9	-
161.	Bharathi	24	primi	0	40	22	3.3	31	2.4	3	11	EM LSCS	boy	3	no	7	8	-
162.	Anu	23	primi	0	38	21	3	30	2.6	4	9	LN	girl	3	no	8	9	-
163.	Janaki	33	G4P3L3	3	37	25	3.3	35	2.9	4	11	LN	girl	4	yes	7	8	-
164.	Bindhu	30	G2P1L1	1	38	21	3.3	32	2.6	3	9	EM LSCS	boy	3	no	8	9	-
165.	Ragini	31	G2P1L1	1	39	20	4	33	2.4	5	12	LN	boy	4	no	8	9	-
166.	Mumtaz	23	primi	0	38	22	3.5	32	2.4	3	11	EL LSCS	girl	3	no	7	8	-
167.	Murugeshwari	25	G2P1L0	1	37	22	3.2	30	2.2	3	11	EM LSCS	girl	3	no	7	8	-
168.	Karpagham	29	G2P1L1	1	38	23	3.5	31	2.3	4	9	EM LSCS	girl	3	no	7	8	-

169.	Bhanu	24	primi	0	37	31	4.2	36	3.4	6	13	EM LSCS	boy	4	yes	8	9	-
170.	Roja	19	primi	0	38	24	2.7	32	2.4	3	10	LN	boy	3	no	8	9	-
171.	Rupa	20	primi	0	38	23	3	33	2.6	3	11	FORCEPS	boy	3	no	8	9	-
172.	Divya	24	G2P1L1	1	38	24	2.9	33	2.4	4	9	LN	girl	3	no	7	8	-
173.	Diana	26	G3P1L1A1	1	37	22	3	34	2.5	4	10	EM LSCS	boy	3	no	7	8	-
174.	Aarthi	25	G2P1L1	1	37	20	2.5	30	2.5	3	10	LN	boy	3	no	7	8	-
175.	Bhuvana	23	primi	0	38	21	2.5	32	2.6	4	9	LN	boy	3	no	8	9	-
176.	Kalpana	28	G3P2L2	2	39	26	4.1	36	3.2	3	13	EM LSCS	boy	4	no	8	9	-
177.	Punitha	24	primi	0	38	21	3.5	32	2.2	4	10	EM LSCS	boy	3	no	7	8	-
178.	Anjana	23	primi	0	38	23	3.5	34	2.1	3	11	LN	girl	3	no	7	8	-
179.	Karthiga	27	G2P1L1	1	38	29	4	35	3.2	4	13	EL LSCS	boy	4	yes	8	9	-
180.	Rama	26	G2P1L1	1	37	23	3	35	2.6	4	11	EM LSCS	girl	3	no	7	8	-
181.	Puspha	27	G2P1L1	1	40	22	3.3	34	2	3	11	LN	girl	4	no	7	8	-
182.	Rupa	25	primi	0	38	22	2.7	33	2.2	3	9	EM LSCS	girl	3	no	8	9	-
183.	Rubini	22	primi	0	39	23	3	32	2.2	3	10	EM LSCS	boy	3	no	8	9	-
184.	Narmada	26	G3P2L1	2	38	27	3.6	37	2.8	4	11	EM LSCS	girl	4	no	8	9	-
185.	Rukmani	27	G2P1L1	1	37	24	3.5	34	2.4	4	9	EM LSCS	boy	3	no	8	9	-
186.	Lakshmi	20	primi	0	37	25	3.8	34	2.5	4	9	EM LSCS	girl	3	no	7	8	-
187.	Margaret	26	G2P1L1	1	37	29	4	35	2.6	5	12	EM LSCS	boy	4	yes	8	9	-
188.	Krishnaveni	21	G2A1	0	37	25	3.5	33	2.5	4	10	LN	boy	3	no	7	8	-
189.	Meena	27	G3P2L2	2	37	24	3	33	2.2	3	10	EM LSCS	boy	3	no	7	8	-
190.	Usha	24	primi	0	38	23	3.7	33	2.8	5	12	EM LSCS	girl	4	no	7	8	-
191.	Gayathri	28	G2P1L1	1	37	27	4.1	36	3.2	5	12	EL LSCS	Girl	4	yes	8	9	-
192.	Vandana	30	G4P2L2A1	2	38	23	3.5	34	2.7	4	11	LN	girl	4	no	7	8	-
193.	Shrisha	26	primi	0	37	26	3.8	36	3.3	3	10	EM LSCS	boy	4	no	8	9	-
194.	Myil	29	G2P1L1	1	37	23	3.5	31	2.8	5	11	EL LSCS	boy	4	no	8	9	-
195.	Amala	30	G2P1L1	1	38	22	3.5	31	2.2	3	9	EL LSCS	girl	3	no	8	9	-
196.	Kasthuri	31	G2P1L0	1	37	19	2.5	30	1.8	3	11	LN	girl	2	no	7	9	-
197.	Venda	26	G2P1L1	1	38	22	3	32	2.5	3	10	LN	girl	3	no	7	8	-

198.	Parvathy	37	G4P3L3	3	39	23	3.5	33	2.6	4	11	LN	boy	3	no	7	8	-
199.	Radha	19	primi	0	37	24	3	31	2.7	4	9	LN	girl	3	no	8	9	-
200.	Ambika	27	G2P1L1	1	40	24	3.2	31	2.6	3	11	LN	girl	3	no	8	9	-
201.	Prashati	28	primi	0	39	25	4	36	3.1	5	12	EL LSCS	boy	4	no	8	9	-
202.	Fathima	26	G3P1L1A1	1	37	23	3.5	33	2.5	4	11	FORCEPS	girl	3	no	6	7	-
203.	Reshmi	19	primi	0	38	26	3.75	36	2.8	4	11	EM LSCS	boy	4	no	8	9	-
204.	Jhansi	21	G2A1	0	37	25	3.3	34	2.4	3	11	EM LSCS	boy	4	no	8	9	-
205.	Ayesha	24	G2P1L1	1	37	22	2.8	33	2.2	4	9	LN	girl	3	no	7	8	-
206.	Ilavarasi	26	G2P1L1	1	37	22	3.5	36	2.7	3	10	EM LSCS	boy	4	no	7	8	-
207.	Rani	27	G3P1L1A1	1	38	21	3	33	2.3	3	10	LN	girl	3	no	7	8	-
208.	Anitha	37	G5P3L3A1	3	37	27	3.7	34	3	5	12	FORCEPS	boy	4	yes	5	7	+
209.	Anu	23	primi	0	39	21	2.8	34	2.3	3	10	LN	boy	3	no	8	9	-
210.	Yamini	22	primi	0	38	20	2.7	32	2.4	4	9	LN	girl	3	no	8	9	-
211.	Rajammal	21	primi	0	38	21	2.6	32	2.8	3	9	LN	boy	3	no	8	9	-
212.	Sowbakia	26	G2P1L1	1	40	22	3	31	2	3	11	EM LSCS	girl	3	no	7	8	-
213.	Roja	25	G3A2	0	38	26	4	37	3.1	6	13	EL LSCS	boy	4	yes	8	9	-
214.	Kamala	27	G2P1L1	1	39	23	3.5	31	2.1	4	11	EL LSCS	Girl	3	no	7	8	-
215.	Kavitha	28	G3P1L1A1	1	38	23	3.5	30	2	3	11	EL LSCS	boy	3	no	7	8	-
216.	Anupama	21	primi	0	39	21	3	32	2.4	3	10	FORCEPS	boy	3	no	8	9	-
217.	Vengammal	22	G3A2	0	38	21	2.8	32	2.6	3	9	LN	Girl	3	no	7	8	-
218.	Kannathal	25	G4P1L1A2	1	38	21	2.8	33	2.7	3	9	EM LSCS	Girl	3	no	7	8	-
219.	Priya	24	G2P1L1	1	37	22	3.6	34	2.9	3	10	EM LSCS	boy	4	no	8	9	-
220.	Sangeetha	23	G2P1L0	1	38	27	4.2	37	3.3	5	12	EL LSCS	boy	4	no	8	9	-
221.	Nivedhitha	25	primi	0	38	24	3.2	34	2.5	4	10	LN	Girl	3	no	8	9	-
222.	Vani	24	primi	0	39	24	3.5	34	2.5	4	11	EM LSCS	boy	4	no	7	8	-
223.	Banu	28	G2P1L2	1	38	23	3	32	2.4	4	9	LN	Girl	3	no	8	9	-
224.	Viji	20	primi	0	37	22	3	31	2.3	3	9	FORCEPS	boy	3	no	7	8	-
225.	Aruna	21	G2A1	0	37	21	3.2	31	2.1	4	10	EM LSCS	Girl	3	no	7	8	-
226.	Kuppammal	29	primi	0	37	26	4	32	3.2	4	12	EM LSCS	girl	4	yes	8	9	-

227.	Ambika	24	G2P1L1	1	38	21	2.5	30	2.1	3	10	LN	boy	3	no	7	9	-
228.	Padma	26	G3P1L1A1	1	39	23	3.2	31	2.4	4	11	EM LSCS	Girl	3	no	6	8	-
229.	Rajeshwari	29	G2P1L1	1	40	23	2.2	32	2.4	4	9	LN	boy	3	no	7	8	-
230.	Kamatchi	32	G4P3L3	3	39	24	2.6	32	2.2	3	10	LN	Girl	3	no	7	8	-
231.	Gajalakshmi	28	G2P1L1	1	38	24	2.5	33	2.2	3	10	EM LSCS	boy	3	no	6	8	-
232.	Sripriya	30	primi	0	37	28	4.4	36	3.2	5	12	EL LSCS	boy	4	no	7	8	-
233.	Vijayalakshmi	23	primi	0	37	23	3.5	34	2.4	4	11	EM LSCS	Girl	3	no	8	9	-
234.	Fathima	34	G4P3L3	3	37	27	3.8	36	3.2	6	12	LN	boy	4	yes	6	8	+
235.	Parimala	32	G3P2L2	2	37	23	4	36	2.8	4	13	EM LSCS	boy	4	no	7	8	-
236.	Roopa	21	primi	0	37	22	3.75	32	2.5	4	11	EM LSCS	Girl	4	no	7	8	-
237.	Krithika	23	G2A1	0	38	23	3.5	33	2.4	3	11	EM LSCS	boy	3	no	7	8	-
238.	Alamelu	24	G2P1L1	1	38	22	3.5	33	2.4	4	11	EM LSCS	Girl	3	no	7	8	-
239.	Andal	25	G2P1L1	1	39	25	2.5	36	2.8	3	9	LN	boy	3	no	8	9	-
240.	Datchayani	30	G3P2L2	2	38	29	4.2	36	2.9	6	13	EM LSCS	boy	4	yes	7	8	-
241.	Jothi	28	G3P2L2	2	37	24	2.5	34	2.6	3	10	LN	Girl	3	no	8	9	-
242.	Indra	27	G3P1L1A1	1	37	23	3	34	2.5	4	10	EM LSCS	boy	3	no	8	9	-
243.	Hemavathy	24	G2P1L1	1	37	23	3	33	2.4	3	10	LN	Girl	3	no	8	9	-
244.	Vasanthi	23	primi	0	38	26	4.1	36	3.3	4	12	EL LSCS	boy	4	no	8	9	-
245.	Kumari	20	primi	0	38	24	2.5	33	2.4	3	9	LN	boy	3	no	7	8	-
246.	Baby	19	primi	0	38	23	2.8	32	2.3	4	9	LN	Girl	3	no	7	8	-
247.	Josephine	21	primi	0	37	22	3	31	2.3	3	11	FORCEPS	Girl	3	no	7	8	-
248.	Kanmani	23	primi	0	38	21	3	31	2.2	4	11	EM LSCS	boy	3	no	6	8	-
249.	Noornisha	24	primi	0	38	26	4	36	2.7	5	12	EM LSCS	Girl	4	yes	7	8	-
250.	Amala	24	G2A1	0	38	20	3.2	30	2	3	11	LN	boy	3	no	7	8	-
251.	Gangammal	25	G2P1L1	1	39	20	2.2	34	2.2	4	9	LN	Girl	2	no	7	8	-
252.	Petchiammal	24	G2P1L1	1	39	19	3	30	2	3	9	EM LSCS	Girl	2	no	6	8	-
253.	Poongodi	26	G2P1L1	1	38	20	3.3	34	2.4	4	10	LN	Girl	3	no	8	9	-
254.	Nirmala	26	primi	0	38	31	4.3	37	3.4	6	14	EL LSCS	boy	5	yes	7	8	-
255.	Dhanalakshmi	27	G3P1L1A1	1	38	22	3.2	33	2.6	3	10	LN	boy	3	no	8	9	-

256.	Annie suchithra	29	G4P1L1A2	1	38	22	2.8	32	2.6	4	10	EM LSCS	girl	3	no	8	9	-
257.	Nabisha	30	G3A2	0	37	21	3.9	32	2.7	3	11	EM LSCS	boy	3	no	8	9	-
258.	Parameshwari	31	G2P1L1	1	38	21	3	33	2.6	3	9	LN	boy	3	no	8	9	-
259.	Kiliyammal	28	G2P1L1	1	38	23	4	34	2.5	5	12	FORCEPS	boy	4	no	5	8	+
260.	Sasikala	24	primi	0	38	24	2.7	32	2.5	4	9	LN	girl	3	no	7	8	-
261.	Vimala	25	G2P1L1	1	37	25	3.4	35	3.1	6	13	LN	boy	4	yes	7	8	-
262.	Rupavathy	20	primi	0	39	24	2.8	31	2.4	3	11	LN	girl	3	no	7	8	-
263.	Sumathy	30	G2P1L0	1	39	27	4.5	37	3.2	5	13	EL LSCS	boy	4	no	7	8	-
264.	Vaidegi	21	primi	0	39	24	3.4	31	2.4	4	11	LN	girl	3	no	8	9	-
265.	Vannamayil	28	G3P1L1A1	1	38	30	4.1	35	3.2	5	13	EM LSCS	boy	4	yes	7	8	-
266.	Selvi	22	primi	0	38	23	3.8	34	2.3	3	11	EM LSCS	boy	4	no	7	8	-
267.	Narayani	28	G3P1L1A1	1	40	26	3.8	36	2.9	3	9	EL LSCS	girl	3	no	8	9	-
268.	Sathya	25	G2P1L1	1	38	23	3.5	31	2.4	4	9	EL LSCS	boy	3	no	8	9	-
269.	Malarkodi	32	G4P3L2	3	37	24	3.2	35	2.2	3	10	FORCEPS	boy	4	no	6	8	+
270.	Ponnarayi	21	primi	0	37	24	3.2	32	2.2	4	11	EM LSCS	girl	3	no	7	8	-
271.	Anjalidevi	22	primi	0	37	24	3.5	33	2.3	4	11	EM LSCS	girl	3	no	8	9	-
272.	Menaka	33	G3P2L2	2	37	24	3.9	31	2.8	5	13	LN	girl	4	yes	7	8	-
273.	Kalpana	20	primi	0	38	24	3	33	2.8	3	9	EM LSCS	boy	3	no	7	8	-
274.	bharathi	21	primi	0	38	22	3.8	36	3.3	3	9	EM LSCS	boy	4	no	6	8	-
275.	Kayalmani	24	G2P1L1	1	37	22	3	32	2.4	4	11	LN	boy	3	no	7	8	-
276.	Sangeetha	37	G5P4L4	4	37	26	3.8	36	3	5	13	LN	girl	4	yes	5	7	+
277.	Navaneetham	25	G2P1L1	1	37	23	2.8	32	2.2	4	11	LN	boy	3	no	8	9	-
278.	Gomathi	23	G2A1	0	37	24	3	30	2.3	3	10	EM LSCS	girl	3	no	6	8	-
279.	Jayalalitha	21	primi	0	38	24	3.3	30	2	3	10	EM LSCS	girl	3	no	7	8	-
280.	Gangammal	27	G4P1L1A2	1	39	23	3	31	2.2	3	9	LN	boy	3	no	7	8	-
281.	Rekha	26	G3P1L1A1	1	40	26	4.1	36	2.6	4	12	EM LSCS	girl	4	no	8	9	-
282.	Gnaneshwari	27	G2P1L1	1	37	23	3.6	35	2.5	4	11	LN	boy	4	yes	7	8	-
283.	Vimala	24	G2P1L1	1	39	22	2.9	31	2	4	11	LN	girl	3	no	8	9	-
284.	Margaret	20	primi	0	39	22	3.1	31	2.2	4	11	FORCEPS	boy	3	no	8	9	-

285.	Valarmathy	21	primi	0	39	23	3.5	33	2.2	3	10	LN	girl	3	no	7	8	-
286.	Jayasathya	29	G3P2L2	2	38	24	3	33	2.4	5	11	LN	girl	3	no	7	9	-
287.	Kantha	28	G2P1L1	1	37	24	3.7	35	2.6	3	9	EM LSCS	girl	4	no	8	9	-
288.	Juliet	23	primi	0	37	27	4	34	2.3	5	12	EM LSCS	boy	4	no	7	9	-
289.	Deepa	22	primi	0	38	23	3.2	32	2.3	3	9	EM LSCS	girl	3	no	7	8	-
290.	Sameena	23	primi	0	38	25	3.6	35	3	5	13	FORCEPS	boy	4	yes	5	8	+
291.	Guruvammal	24	primi	0	38	22	2.5	31	2.2	4	10	LN	girl	3	no	8	9	-
292.	Bhuvaneshwari	25	G2P1L0	1	38	22	2.5	31	2	3	11	LN	girl	3	no	7	8	-
293.	Vijayakumari	26	G2P1L1	1	37	23	2.8	33	2	3	11	EM LSCS	girl	3	no	7	8	-
294.	Jesima	27	G3P1L1A1	1	38	23	3.7	34	2.3	4	11	EL LSCS	boy	3	no	7	8	-
295.	Gowri	25	primi	0	36	24	3.6	35	2.8	5	12	LN	girl	4	yes	5	7	+
296.	Selvi	30	G4P3L3	3	39	27	3.8	37	3.2	5	13	LN	boy	4	no	7	8	-
297.	Samundeswari	31	G2P1L1	1	40	23	2.5	34	2.4	4	9	LN	boy	3	no	7	8	-
298.	Kasiammal	26	G2A1	0	38	24	2.5	34	2.5	3	9	LN	girl	3	no	7	8	-
299.	Areeta	23	primi	0	37	24	2.8	32	2.5	3	9	EM LSCS	girl	3	no	8	9	-
300.	Panchalai	31	G4P2L2A1	2	38	27	4.3	36	2.6	6	12	EM LSCS	girl	5	yes	8	9	-
301.	Logeswari	22	primi	0	38	22	3	31	2.2	3	10	EM LSCS	boy	3	no	8	9	-
302.	Ashraff	26	G3A2	0	39	26	3	36	2.8	4	11	EM LSCS	girl	3	no	8	9	-
303.	Saraswathy	30	G3P2L2	2	36	30	4	35	2.5	5	12	LN	Girl	4	yes	8	9	-
304.	Kulamagal	19	primi	0	38	21	3	30	2.1	3	11	EM LSCS	Girl	3	no	7	8	-
305.	Kalavathy	28	G2P1L1	1	37	20	3	31	2.3	3	10	LN	boy	3	no	8	9	-
306.	Selvi	29	G2P1L1	1	37	20	3.2	31	2.2	3	10	EL LSCS	Girl	3	no	8	9	-
307.	Sudha	31	G3P1L1A1	1	38	21	3	30	2.3	3	11	LN	boy	3	no	7	8	-
308.	Ramya	27	G2P1L1	1	37	23	2.9	31	2.2	3	10	EM LSCS	boy	3	no	7	8	-
309.	Renuka	19	primi	0	38	24	3.2	32	2.1	4	11	EL LSCS	boy	3	no	8	9	-
310.	Esther	24	G2P1L1	1	37	26	4	35	3	6	12	EM LSCS	Girl	4	yes	7	8	-
311.	Tamilselvi	19	primi	0	38	20	2.8	33	3	3	11	LN	boy	3	no	8	9	-
312.	Sathiya	26	G2P1L1	1	37	26	3.5	36	3.3	4	10	EM LSCS	boy	4	no	7	8	-
313.	Kanimozhi	23	primi	0	38	22	3	32	2.4	4	10	LN	Girl	3	no	7	8	-

314.	Dhanalakshmi	33	G3P2L2	2	37	22	2.5	33	2.3	3	9	LN	boy	3	no	8	9	-
315.	Nithyakalyani	28	G2P1L1	1	37	27	4.1	36	2.3	5	12	EM LSCS	boy	4	yes	8	9	-
316.	Dhanakodi	28	G2P1L1	1	37	23	3	32	2.3	5	11	EL LSCS	boy	3	no	7	8	-
317.	Meera	21	primi	0	38	24	2.4	33	3.2	3	9	LN	boy	3	no	7	8	-
318.	Amutha	23	G2A1	0	39	27	4	37	2.9	5	12	EM LSCS	Girl	4	no	8	9	-
319.	Subashini	28	primi	0	37	23	4.5	35	3.1	5	12	EM LSCS	boy	4	yes	8	9	-
320.	Lalitha	24	G2P1L1	1	38	20	2.8	31	2.1	3	9	EL LSCS	boy	3	no	8	9	-
321.	Geetha	21	primi	0	37	21	3	31	2.1	4	10	EM LSCS	boy	3	no	8	9	-
322.	Dhanalakshmi	22	primi	0	37	20	2.9	32	2.3	3	10	EM LSCS	Girl	3	no	8	9	-
323.	Janaki	26	G2P1L1	1	38	21	2.8	33	2.4	3	9	LN	boy	3	no	7	8	-
324.	Mallishwari	24	G2A1	0	37	26	4	36	3	5	12	EM LSCS	boy	4	yes	8	9	-
325.	Mohana	27	G2P1L1	1	39	22	2.9	34	2.4	3	9	EM LSCS	Girl	3	no	7	8	-
326.	Ishrath	25	primi	0	37	26	4	36	3.2	4	12	EM LSCS	boy	4	no	7	8	-
327.	Akila	28	G3P1L1A1	1	40	22	3	34	2.5	4	10	FORCEPS	boy	3	no	8	9	-
328.	Syed Ali Fathima	21	primi	0	38	24	3	33	2.5	3	10	LN	Girl	3	no	7	8	-
329.	Anandhi	32	G4P3L3	3	38	25	3.7	36	2.9	6	12	LN	boy	4	yes	6	7	+
330.	Sabin	26	G2P1L0	1	39	24	3.3	32	2.4	4	10	EM LSCS	boy	3	no	7	8	-
331.	Vanitha	27	G2P1L1	1	38	24	2.9	33	2.3	3	9	LN	Girl	3	no	8	9	-
332.	Mary	21	primi	0	37	23	3	34	2.2	3	9	FORCEPS	boy	3	no	8	9	-
333.	Vimaladevi	37	G5P3L3A1	3	38	31	3.8	35	2.8	6	12	EM LSCS	girl	4	yes	7	8	-
334.	Uma	20	primi	0	38	22	3.5	32	2.1	3	10	EM LSCS	Girl	3	no	7	8	-
335.	Manimegalai	19	primi	0	39	22	3.5	32	2.1	4	11	EM LSCS	boy	3	no	8	9	-
336.	Ravichandrika	28	G3P1L1A1	1	38	23	4.1	36	2.1	4	11	EM LSCS	boy	4	no	7	8	-
337.	Kala	29	G2P1L1	1	38	23	3.5	34	2.2	3	11	LN	Girl	3	no	8	9	-
338.	Thangam	25	primi	0	38	20	3.5	30	2	3	11	EM LSCS	boy	3	no	8	9	-
339.	Sophia	28	G2P1L1	1	39	19	2.3	29	1.9	3	11	EM LSCS	girl	2	no	6	8	-
340.	Amirthakala	22	G2A1	0	38	22	2.8	31	2.1	4	11	LN	boy	3	no	7	8	-
341.	Vasuki	27	G3P2L2	2	39	24	3.5	34	2.5	6	11	FORCEPS	boy	4	yes	7	8	-
342.	Jayanthi	20	primi	0	38	25	4.1	35	2.7	5	12	EL LSCS	boy	4	no	8	9	-

343.	Saranya	36	G2P1L1	1	37	22	2.8	32	2.4	3	9	LN	girl	3	no	7	8	-
344.	Anitha	24	G2P1L1	1	38	20	2.5	30	2.3	3	9	LN	girl	3	no	7	8	-
345.	Kanchana	25	G2P1L1	1	37	22	3	31	2.5	4	10	EM LSCS	girl	3	no	8	9	-
346.	Chinni	30	G3P1L1A1	1	37	23	2.7	33	2.5	4	11	LN	girl	3	no	8	9	-
347.	Priya	19	primi	0	38	22	3.2	30	2.4	3	11	EM LSCS	boy	3	no	7	8	-
348.	Shanthi	30	G4P3L3	3	39	29	4.4	35	2.9	5	12	EM LSCS	boy	4	yes	7	8	-
349.	Grace	24	G2P1L1	1	38	22	3	33	2.5	4	11	EM LSCS	boy	3	no	8	9	-
350.	Ponniammal	26	G2P1L1	1	37	23	3	33	2.5	3	10	EL LSCS	girl	3	no	7	8	-
351.	Sumathy	20	primi	0	37	21	2.7	32	2.4	4	9	LN	boy	3	no	8	9	-
352.	Gowthami	22	primi	0	38	21	2.8	34	2.2	4	9	LN	boy	3	no	8	9	-
353.	Elakiya	28	G2P1L0	1	37	26	4.2	37	3.3	4	13	EL LSCS	boy	4	no	8	9	-
354.	Samundeswari	26	G2P1L1	1	37	28	3.9	36	3	4	12	LN	boy	4	yes	7	8	-
355.	Sathya	27	G2P1L1	1	37	20	3	34	2.2	3	10	EM LSCS	girl	3	no	7	8	-
356.	Pushpavalli	29	G3P1L1A1	1	39	23	3.1	33	2.4	3	10	EM LSCS	girl	3	no	7	8	-
357.	Kalaiarasi	23	primi	0	39	24	2.8	33	2.4	3	9	LN	girl	3	no	8	9	-
358.	Sampoornam	22	primi	0	38	23	3	32	2.6	4	11	EM LSCS	boy	3	no	8	9	-
359.	Meenatchi	26	G2P1L1	1	40	22	3	32	2.6	3	11	EL LSCS	boy	3	no	7	8	-
360.	Roseline	25	G2A1	0	38	22	3.4	30	2.5	4	11	LN	girl	3	no	7	8	-
361.	Poongavanam	25	primi	0	40	28	3.7	37	3	5	13	EM LSCS	boy	4	yes	8	9	-
362.	Meharunisha	21	primi	0	38	21	3	31	2.5	4	11	EM LSCS	boy	3	no	8	9	-
363.	Mohana	27	G2P1L1	1	37	20	2.5	31	2.8	3	9	LN	girl	3	no	8	9	-
364.	Usha	29	G2P1L1	1	37	20	3.2	34	2	4	10	LN	boy	3	no	8	9	-
365.	Sarasu	25	primi	0	38	19	2.7	32	2.3	3	10	EM LSCS	girl	3	no	8	9	-
366.	Umarani	20	primi	0	39	20	3	32	2.3	5	11	LN	girl	3	no	7	9	-
367.	Kokila	29	G3P1L1A1	1	37	20	4	34	2.4	3	9	EM LSCS	boy	4	no	8	9	-
368.	Prabha	30	G2P1L1	1	38	27	4	36	3.2	5	12	EM LSCS	Girl	4	no	8	9	-
369.	Naseera	27	G3P1L1A1	1	39	28	4	35	3	6	12	EL LSCS	Girl	4	yes	8	9	-
370.	Jeeva	28	G2P1L0	1	39	21	2.5	33	2.4	3	9	LN	boy	3	no	7	8	-
371.	Elamathy	32	G4P3L3	3	39	24	3.2	36	2.8	4	10	LN	Girl	3	no	6	8	-

372.	Thavamani	30	primi	0	37	27	3.5	35	2.9	4	12	EM LSCS	boy	4	no	8	9	-
373.	Anjalidevi	33	G3P2L2	2	40	23	2.8	32	2	3	9	LN	Girl	3	no	7	8	-
374.	Rahmathnisha	19	primi	0	39	23	2.4	33	2.2	4	11	LN	Girl	3	no	8	9	-
375.	Haripriya	24	primi	0	37	27	4.1	36	2.9	6	12	EM LSCS	Girl	4	yes	8	9	-
376.	Saroja	24	G3A2	0	38	26	3.5	34	2.4	3	10	EM LSCS	boy	4	no	6	8	-
377.	Malini	26	G2P1L1	1	38	24	2.8	30	2.3	4	10	LN	boy	3	no	7	8	-
378.	Asha	26	G2P1L1	1	39	28	4.6	37	3	5	12	EM LSCS	boy	5	yes	7	8	-
379.	Sujitha	28	G2P1L1	1	38	24	3.3	30	2.4	3	11	EM LSCS	boy	4	no	7	8	-
380.	Porkilai	25	G2P1L1	1	37	22	3	30	2.5	4	11	LN	Girl	3	no	8	9	-
381.	Beula	24	primi	0	37	24	2.9	34	2.6	3	9	EM LSCS	Girl	3	no	8	9	-
382.	Jaya	23	primi	0	37	26	2.5	36	3.2	4	10	LN	Girl	3	no	7	8	-
383.	Pramila	33	G4P3L3	3	39	29	3.9	32	2.8	6	11	LN	boy	4	yes	8	9	-
384.	Madhavi	24	primi	0	38	24	3.5	34	2.4	4	10	EM LSCS	Girl	4	no	7	8	-
385.	Suganya	25	primi	0	38	23	3.2	33	2.5	4	10	EM LSCS	boy	3	no	7	8	-
386.	Thangammal	26	G2P1L1	1	37	22	3	31	2.2	3	9	LN	Girl	3	no	8	9	-
387.	Arputham	29	G3P1L1A1	1	39	23	2.6	30	2	3	9	EM LSCS	Girl	3	no	8	9	-
388.	Annal	31	G2P1L1	1	40	23	2.9	30	2	3	9	LN	boy	3	no	8	9	-
389.	Prasanna	34	G4P3L3	3	37	27	3.6	33	3	4	10	LN	Girl	4	yes	8	9	-
390.	Mahalakshmi	32	G3P2L2	2	37	24	3.4	33	2.6	4	9	LN	boy	3	no	7	8	-
391.	Shoba	30	G2P1L1	1	38	25	3.3	32	2.5	3	9	LN	Girl	3	no	7	8	-
392.	Ruksana	28	G2A1	0	39	24	3.7	35	2.9	4	11	EM LSCS	Girl	4	no	8	9	-
393.	Amudha	28	G3P2L1	2	37	27	3.5	36	2.7	5	12	EM LSCS	Girl	4	yes	7	8	-
394.	Bhavani	26	G3P1L1A1	1	37	24	3.2	32	2.2	4	10	LN	boy	3	no	7	8	-
395.	Anthonypriya	25	primi	0	38	23	3	30	2	3	9	LN	boy	3	no	8	9	-
396.	Najima bee	26	G2P1L1	1	38	22	2.9	30	1.9	3	9	LN	Girl	3	no	7	8	-
397.	Suguna	23	primi	0	39	22	3.8	35	2.6	4	11	EM LSCS	Girl	3	no	8	9	-
398.	Radhika	22	primi	0	38	30	4.2	34	3.2	5	13	EL LSCS	boy	5	yes	8	9	-
399.	Rita	20	primi	0	38	23	2.7	31	2.5	4	10	EM LSCS	Girl	3	no	7	8	-
400.	Sivagami	21	G2A1	0	37	24	3	34	2.4	4	11	EM LSCS	Girl	3	no	8	9	-

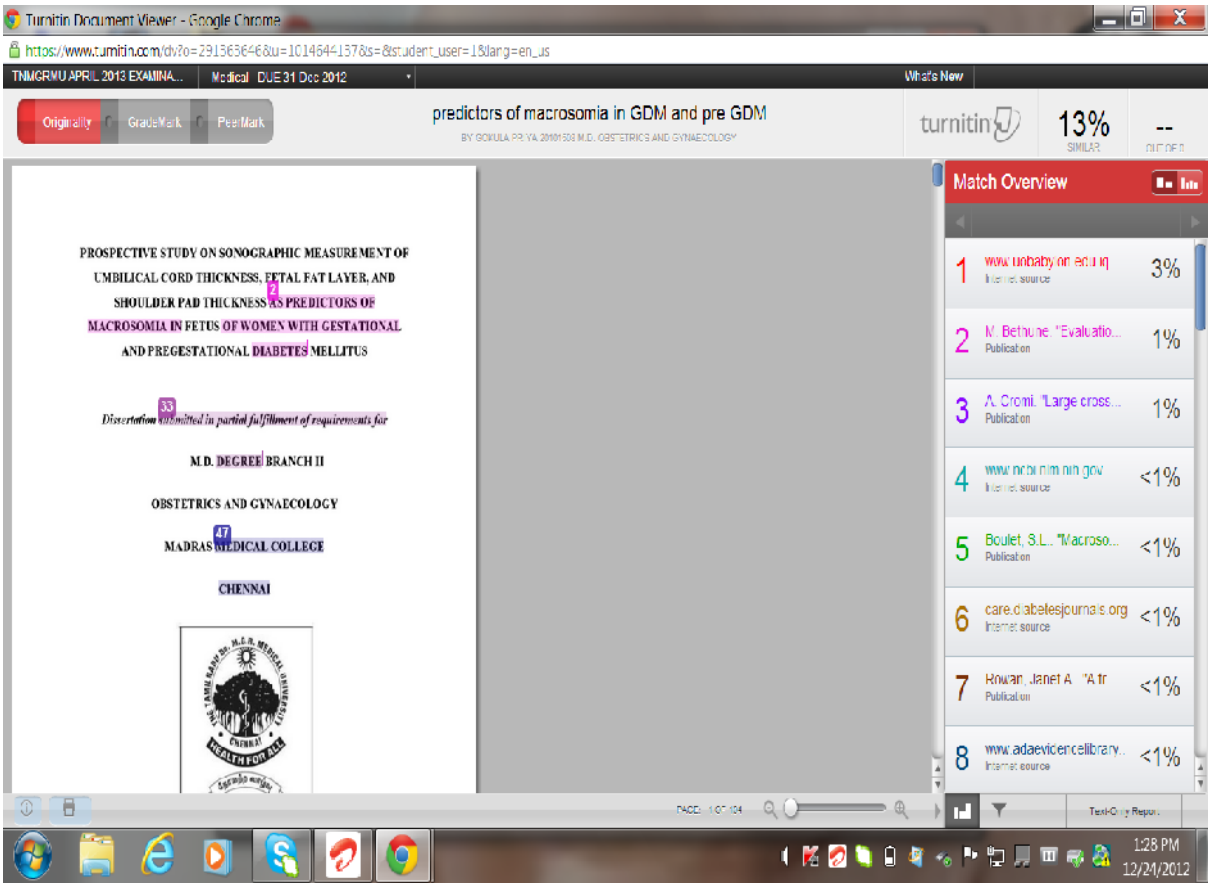
401.	Caroline mary	27	G3P2L2	2	40	25	3.7	35	2.9	4	11	FORCEPS	boy	4	no	8	9	-
402.	Lakshmi	29	G2P1L1	1	38	22	2.9	33	2.6	3	9	LN	boy	3	no	8	9	-
403.	Anupama	25	G2P1L0	1	37	21	3.2	32	2.2	3	9	EM LSCS	Girl	3	no	8	9	-
404.	Kaliammal	31	G4P3L3	3	39	28	3.8	32	3	6	11	LN	boy	4	yes	7	8	-
405.	Veeralakshmi	24	primi	0	38	21	3	30	2.3	3	9	LN	Girl	3	no	7	8	-
406.	Kaveri	27	G2P1L1	1	39	23	3	30	2.4	4	10	EM LSCS	boy	3	no	7	8	-

KEY TO MASTER CHART

OBS. SCORE	- OBSTETRIC SCORE
GA	- GESTATIONAL AGE
BMI	- BODY MASS INDEX
AC	- ABDOMINAL CIRCUMFERENCE
FFL	- FETAL FAT LAYER
UCT	- UMBILICAL CORD THICKNESS
SPT	- SHOULDER PAD THICKNESS
LN	- LABOUR NATURALE
EM.LSCS	- EMEGENCY LOWER SEGMENT CAESAREAN SECTION
EL.LSCS	- ELECTIVE LOWER SEGMENT CAESAREAN SECTION

ABBREVIATIONS

GDM	- Gestational Diabetes Mellitus
Pre-GDM	- Pre-Gestational Diabetes Mellitus
GA	- Gestational Age
AN	- Antenatal
BMI	- Body Mass Index
FBS	- Fasting Blood Sugar
PPBS	- Postprandial blood sugar
OGCT	- Oral Glucose Challenge Test
OGTT	- Oral Glucose Tolerance test
IGF	- Insulin like growth factor
AFP	- Alpha fetoprotein
NST	- Non Stress Test
PPH	- Post partum hemorrhage
BW	- Birth weight
HB	- Hemoglobin
AC	- Abdominal Circumference
FL	- Femur length
BPD	- Bi parietal diameter
AFI	- Amniotic Fluid Index
FH	- Fetal heart
FFL	- Fetal Fat Layer
SPT	- Shoulder Pad Thickness
UCT	- Umbilical Cord Thickness
NPV	- Negative predictive value
PPV	- Positive predictive value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