

**“PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL  
CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL  
THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH  
GESTATIONAL DIABETES MELLITUS”**

**DR. DIRISALA ANUDEEP**

**Dissertation submitted to**

**B.L.D.E. UNIVERSITY, VIJAYAPURA, KARNATAKA**



**IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE  
DEGREE OF**

**DOCTOR OF MEDICINE**

**IN**

**RADIO-DIAGNOSIS**

**UNDER THE GUIDANCE OF**

**DR. SHIVANAND V. PATIL**

**PROFESSOR,**

**DEPARTMENT OF RADIODIAGNOSIS**

**DR.SIDDROODHA SAJJAN**

**ASSOCIATE PROFESSOR,**

**DEPARTMENT OF RADIODIAGNOSIS**

**B.L.D.E (DEEMED TO BE UNIVERSITY), SHRI B M PATIL MEDICAL  
COLLEGE HOSPITAL & RESEARCH CENTRE,  
VIJAYAPURA, KARNATAKA,2025**

**B.L.D.E. UNIVERSITY'S**  
**SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &**  
**RESEARCH CENTRE, VIJAYAPURA**

**DECLARATION BY THE CANDIDATE**

I, **DR. DIRISALA ANUDEEP**, hereby declare that this dissertation entitled “**PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES MELLITUS**” is a bonafide and genuine research work carried out by me under the guidance of **DR. SHIVANAND V. PATIL**, Professor, Department of Radiodiagnosis and **DR. SIDDAROODHA SAJJAN**, Associate Professor, Department of Radiodiagnosis at B.L.D.E.U’ s Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapura.

Date:

Place: Vijayapura

**DR.DIRISALA ANUDEEP**  
Post Graduate Student,  
Department of Radiodiagnosis,  
B.L.D.E.U’ s Shri B. M. Patil Medical  
College, Hospital & Research Centre,  
Vijayapura.

**B.L.D.E. UNIVERSITY'S**

**SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &  
RESEARCH CENTRE, VIJAYAPURA**

**CERTIFICATE BY THE GUIDE**

This to certify that the dissertation entitled “**PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES MELLITUS**” is a bonafide research work done by **DR. DIRISALA ANUDEEP**, under my overall supervision and guidance, in partial fulfilment of the requirements for the degree of M.D. in Radiodiagnosis.

Date:

Place: Vijayapura

**DR. SHIVANAND V. PATIL**  
Professor,  
Department of Radiodiagnosis,  
B.L.D.E.U' s Shri B. M. Patil Medical College, Hospital  
& Research Centre, Vijayapura.

**DR. SIDDAROODHA SAJJAN**  
Associate Professor,  
Department of Radiodiagnosis,  
B.L.D.E.U' s Shri B. M. Patil Medical College, Hospital  
& Research Centre, Vijayapura.

**B.L.D.E. UNIVERSITY'S**

**SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &  
RESEARCH CENTRE, VIJAYAPURA**

**ENDORSEMENT BY THE HEAD OF DEPARTMENT**

This to certify that the dissertation entitled “**PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES MELLITUS**” is a bonafide research work done by **DR. DIRISALA ANUDEEP** under the guidance of **DR. SHIVANAND V. PATIL**, Professor, Department of Radiodiagnosis and **DR. SIDDAROODHA SAJJAN**, Associate Professor, Department of Radiodiagnosis at B.L.D.E.U' s Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura.

Date:

Place: Vijayapura

**DR. RAJASHEKHAR MUCHCHANDI**  
Professor and HOD  
Department of Radiodiagnosis,  
B.L.D.E.U' s Shri B. M. Patil Medical College, Hospital  
& Research Centre, Vijayapura.

**B.L.D.E. UNIVERSITY'S**

**SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &  
RESEARCH CENTRE, VIJAYAPURA**

**ENDORSEMENT BY THE PRINCIPAL**

This to certify that the dissertation entitled “**PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES MELLITUS**” is a bonafide research work done by **DR. DIRISALA ANUDEEP** under the guidance of **DR. SHIVANAND V. PATIL**, Professor, Department of Radiodiagnosis and **DR. SIDDAROODHA SAJJAN**, Associate Professor, Department of Radiodiagnosis at B.L.D.E.U' s Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapura.

Date:

Place: Vijayapura

**DR. ARAVIND PATIL**

Principal,  
B.L.D.E.U' s

Shri B. M. Patil Medical College, Hospital  
& Research Centre, Vijayapura.

**B.L.D.E. UNIVERSITY'S**  
**SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL &**  
**RESEARCH CENTRE, VIJAYAPURA**

**COPYRIGHT**

**DECLARATION BY THE CANDIDATE**

I hereby declare that the B.L.D.E. UNIVERSITY, VIJAYAPURA, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purposes.

Date:

Place: Vijayapura

DR.DIRISALA ANUDEEP  
Post Graduate Student,  
Department of Radiodiagnosis,  
B.L.D.E.U' s Shri B. M. Patil Medical  
College, Hospital & Research Centre,  
Vijayapura.

© BLDE UNIVERSITY, VIJAYAPURA, KARNATAKA

## **ACKNOWLEDGEMENT**

*This piece of work has been accomplished with the grace of almighty God. It brings me great joy to share my heartfelt gratitude to all. This page is devoted to all those who have assisted me in my quest to learn as much as possible.*

*I want to convey my heartfelt appreciation and sincere thanks to my guide, **Dr. Shivanand V. Patil MD.**, Professor, Department of Radiology, B.L.D.E.U' s Shri B. M. Patil Medical College, Vijayapura, for his constant and unfailing support, professional insight, valuable suggestions, motivation and exemplary guidance to carry out and complete this dissertation. I am deeply grateful to him for providing me necessary facilities and excellent supervision to complete this work.*

*I extend my sincere gratitude to **Dr. Siddaroodha Sajjan DNB.**, Associate Professor, Department of Radiodiagnosis, B.L.D.E.U' s Shri B. M. Patil Medical College, Vijayapura for her constant oversight, guidance and assistance in granting me access to all the necessary avenues making this task possible.*

*I want to express my gratitude to **Dr. R. S. Mudhol MS.** (Vice*

Chancellor), **Dr. Aravind Patil MS.** (Principal) and **Dr. Rajesh M. Honnutagi MD.** (Medical Superintendent), B.L.D.E.U' s Shri B. M. Patil Medical College, Vijayapura, for their support and inspiration.

My deep thanks to **Dr. Rajashekhar Muchchandi MD.** (Professor and HOD), **Dr. Satish D. Patil MD.** (Associate Professor), **Dr. Ravi Kumar DNB.** (Associate Professor), **Dr. Siddaroodha Sajjan DNB.** (Associate Professor), **Dr. Vishal S. Nimbal DNB.** (Assistant Professor), **Dr. Suresh Kanamadi MD.** (Assistant professor), **Dr. Pundalik Lamani MD** (Assistant Professor), **Dr. Pavan Kolekar MD** (Assistant professor), **Dr. Divyashree Koppal MD, EDiR** (Senior resident), **Dr. M. M Patil DMRD,** Department of Radio-diagnosis, B.L.D.E.U' s Shri B. M. Patil Medical College Vijayapura, for their valuable suggestions and encouragement which have definitely helped me improve my research work.

I acknowledge my gratitude to my seniors **Dr. Ayesha Mahaldar MD., Dr. Saad Mustafa MD., Dr. Jayanth Ganesh, Dr. Nihar Doggalli, Dr. Gautam Gutta,** my colleagues **Dr. Vaishnavi, Dr. Deep, Dr. Siddharth, Dr. Prama** and **Dr. Sumaiya,** my juniors **Dr. Saketh,**



**Dr.Prithvi Raj, Dr.Nikhil, Dr.Sahana, Dr.Yashodhan and Dr.Bharath**, Department of Radiology, B.L.D.E. U's Shri B. M. Patil Medical College, Vijaypur, for their support, advice and help in data collection. I thank **Ms. Vijaya**, Statistician for her masterly guidance and statistical analysis. I sincerely acknowledge the support and kindness shown towards me by all the staff of Central Library, Shri B. M. Patil Medical College, Vijayapura, at all times.

My heartily thanks to my beloved parents **Mr. Dirisala Yogananda Reddy and Mrs. Dirisala Mamatha** for their encouragement, support and sacrifices.

Last but not the least, my sincere thanks to all the patients of this study for their cooperation without which this study would not have been possible.

Date:

Place: Vijayapura.

**Dr. Dirisala Anudeep**

## ABSTRACT

**Introduction:** Fetal macrosomia, defined as birth weight exceeding 4000 grams, is associated with increased perinatal morbidity and mortality, particularly in pregnancies complicated by gestational diabetes mellitus (GDM). Conventional methods of estimating fetal weight often lack accuracy in predicting macrosomia in diabetic pregnancies, where abnormal fat distribution patterns may confound standard biometric measurements. This prospective study aimed to evaluate the efficacy of three sonographic parameters—umbilical cord thickness, fetal fat layer thickness, and interventricular septal thickness—as predictors of fetal macrosomia in women with GDM.

**Methods:** A total of 123 pregnant women with GDM between 34-40 weeks of gestation were enrolled in this prospective study. Comprehensive maternal data including age, BMI, and glycemic parameters were recorded. Sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness were performed, and their association with actual birth weight and delivery outcomes was analyzed.

**Results:** Out of 123 pregnancies, 77 (62.6%) resulted in macrosomic babies. Significant associations were found between macrosomia and maternal BMI ( $p < 0.001$ ), HbA1c levels ( $p < 0.001$ ), umbilical cord thickness  $\geq 25$  mm ( $p < 0.001$ ), fetal fat layer  $\geq 4.5$  mm ( $p < 0.001$ ), and interventricular septal thickness  $\geq 3.9$  mm ( $p < 0.001$ ). Umbilical cord thickness demonstrated the strongest correlation with birth weight ( $r = 0.792$ ,  $p < 0.001$ ) and showed excellent diagnostic accuracy with sensitivity of 93.3% and specificity of 85.4%. Fetal fat layer thickness exhibited high specificity (93.3%) and positive predictive value (97.3%), while interventricular septal thickness showed good specificity (85%) but lower sensitivity (71.8%).

**Conclusion:** Sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness are valuable predictors of fetal macrosomia in GDM pregnancies. Integration of these parameters with maternal factors may enhance the accuracy

of macrosomia prediction, potentially improving clinical decision-making and optimizing maternal and neonatal outcomes.

**Keywords:** Gestational diabetes mellitus, Fetal macrosomia, Umbilical cord thickness, Fetal fat layer, Interventricular septal thickness, Sonography, Pregnancy outcome.

## CONTENTS

<b>NO.</b>	<b>TITLE</b>	<b>PAGE NO</b>
1	<b>INTRODUCTION</b>	14
2	<b>AIMS AND OBJECTIVES</b>	16
3	<b>REVIEW OF LITERATURE</b>	17
4	<b>MATERIALS AND METHODS</b>	36
5	<b>RESULTS AND OBSERVATIONS</b>	40
6	<b>DISCUSSION</b>	56
7	<b>CONCLUSION</b>	65
8	<b>SUMMARY</b>	67
9	<b>BIBLIOGRAPHY</b>	70
10	<b>ANNEXURES</b>	
	<b>I. PROFORMA</b>	82
	<b>II. CONSENT FORM</b>	83
11	<b>ETHICAL CLEARANCE</b>	85

# INTRODUCTION

“Gestational diabetes mellitus (GDM) affects approximately 7-25% of pregnancies worldwide, with significant variations across populations and diagnostic criteria.”<sup>1</sup> This metabolic disorder, characterized by glucose intolerance first recognized during pregnancy, poses substantial risks for both maternal and fetal complications, particularly macrosomia.<sup>2</sup> “Fetal macrosomia, defined as birth weight exceeding 4000g or above the 90th percentile for gestational age, occurs in 15-45% of diabetic pregnancies compared to 10% in the general population.”<sup>3</sup>

The accurate prenatal prediction of macrosomia remains a critical challenge in obstetric care, particularly in GDM-affected pregnancies. Traditional methods, including clinical examination and conventional sonographic fetal biometry, have shown limited predictive accuracy, with sensitivity ranging from 50-75%.<sup>4,5</sup> This inadequacy has prompted the search for more reliable ultrasonographic markers that could better reflect the altered fetal growth patterns characteristic of diabetic pregnancies.<sup>6</sup>

Recent research has identified several promising sonographic parameters that may enhance the prediction of macrosomia.<sup>3,7</sup> These include umbilical cord thickness (UCT), which reflects the altered composition of Wharton's jelly in diabetic pregnancies; fetal fat layer measurements, which directly quantify the increased adipose tissue deposition characteristic of infants of diabetic mothers<sup>4</sup>; and interventricular septal thickness (IVS), which may indicate the cardiac adaptations to the hyperglycemic environment.<sup>7,8</sup>

While these parameters have been studied individually, their combined predictive value in GDM pregnancies remains poorly understood.<sup>9</sup> Furthermore, the temporal evolution of these measurements throughout pregnancy and their correlation with glycemic control have not been systematically evaluated.<sup>10</sup> This prospective study aims to

assess the predictive accuracy of UCT, fetal fat layer, and IVS measurements, both individually and in combination, for identifying macrosomia in GDM pregnancies.

The findings of this study could potentially revolutionize the prenatal surveillance of GDM pregnancies by providing more accurate risk stratification for macrosomia. This would enable more targeted interventions, optimized timing of delivery, and improved maternal and neonatal outcomes.

## **AIM & OBJECTIVES**

### **Objective:**

1. “To assess how well the sonographic parameters of the prenatal fat layer, umbilical cord thickness, and interventricular septal thickness predict fetal macrosomia.”

## **REVIEW OF LITERATURE**

### **GESTATIONAL DIABETES MELLITUS:**

With incidence rates ranging from 1.7% to 13.2% in rural areas and 4.6% to 14% in urban areas, diabetes is a serious public health issue in India.<sup>11</sup> Type 2 diabetes mellitus (DM) affects an estimated 62 million persons in India; by 2025, that figure is predicted to rise to 79.4 million.<sup>11</sup> Effective methods are desperately needed to manage this epidemic since managing diabetes and its complications places a significant financial load on society. “It should come as no surprise that the prevalence of gestational diabetes mellitus (GDM), or diabetes diagnosed during pregnancy, appears to be rising in tandem with the prevalence of diabetes. The prevalence of gestational diabetes has been reported to range from 3.8% in Kashmir,<sup>12</sup> to 6.2% in Mysore,<sup>13</sup> 9.5% in Western India<sup>14</sup> and 17.9% in Tamil Nadu.<sup>15</sup> In more recent studies, using different criteria, prevalence rates as high as 35% from Punjab<sup>16</sup> and 41% from Lucknow have been reported.”<sup>17</sup> Pregnant women in these areas vary in age and/or socioeconomic position, which has been linked to the regional variations in prevalence. In India, it is believed that 4 million women suffer from GDM at any given moment.<sup>18</sup>

### **Impact of gestational diabetes mellitus:**

“GDM raises the risk of Type 2 diabetes in the future for both the mother and the child. It also affects immediate maternal outcomes (preeclampsia, stillbirths, macrosomia, and requirement for cesarean section) and neonatal outcomes (hypoglycemia, respiratory distress).” According to a recent meta-analysis, women who had gestational diabetes are much more likely to develop Type 2 diabetes (relative risk 7.43, 95% CI 4.79–11.51).<sup>19</sup> “According to conventional guidelines, women with GDM were given an oral glucose tolerance test (OGTT) six weeks after giving birth in a recent study conducted in North India.” After birth, a startlingly high percentage of women with GDM had some kind of ongoing glucose problem. 6.4% had overt Type 2 diabetes, 8% had both impaired glucose tolerance (IGT) and impaired fasting glucose



(IFG), and 14.5% had both.<sup>20</sup> These numbers serve as a reminder that GDM needs to be given top attention in our public health system. “Compared to siblings born to the same parents in a non-GDM pregnancy, children of moms with uncontrolled diabetes—whether preexisting or developing during pregnancy—have a four to eight times higher chance of developing diabetes in later life, according to global data.”<sup>21</sup>

#### **Definition of GDM by different criteria:**

Criteria	Method	Fasting (mg/dl)	1 h (mg/dl)	2 h (mg/dl)
WHO 1999	Fasting OGTT With 75 g glucose	-	-	≥140
IADPSG	Fasting OGTT With 75 g glucose	≥92	≥180	≥153
DIPSI	Nonfasting OGTT With 75 g glucose	-	-	≥140

GDM: Gestational diabetes mellitus, WHO: World Health Organization, IADPSG: International Association of Diabetes and Pregnancy Study Groups, DIPSI: Diabetes in Pregnancy Study Group in India, OGTT: Oral glucose tolerance test

It is evident that, by international standards, GDM is quite common in India, regardless of the criteria applied. There is also a significant conversion rate to frank type 2 diabetes. There are not enough resources for healthcare. The public's awareness is insufficient. As a result, many people are reluctant to seek treatment for conditions like GDM that have less "obvious" consequences.<sup>22</sup>

Pregnancy-related and postpartum interventions offer significant chances to enhance the lives of mothers and children now and lower the prevalence of diabetes in subsequent generations. Pregnancy-related diabetes screening and management offer a special chance to avoid Type 2 diabetes in two generations. “One of the reasons GDM is given little attention in India's public health delivery system is a lack of understanding in the general public.”<sup>22</sup>

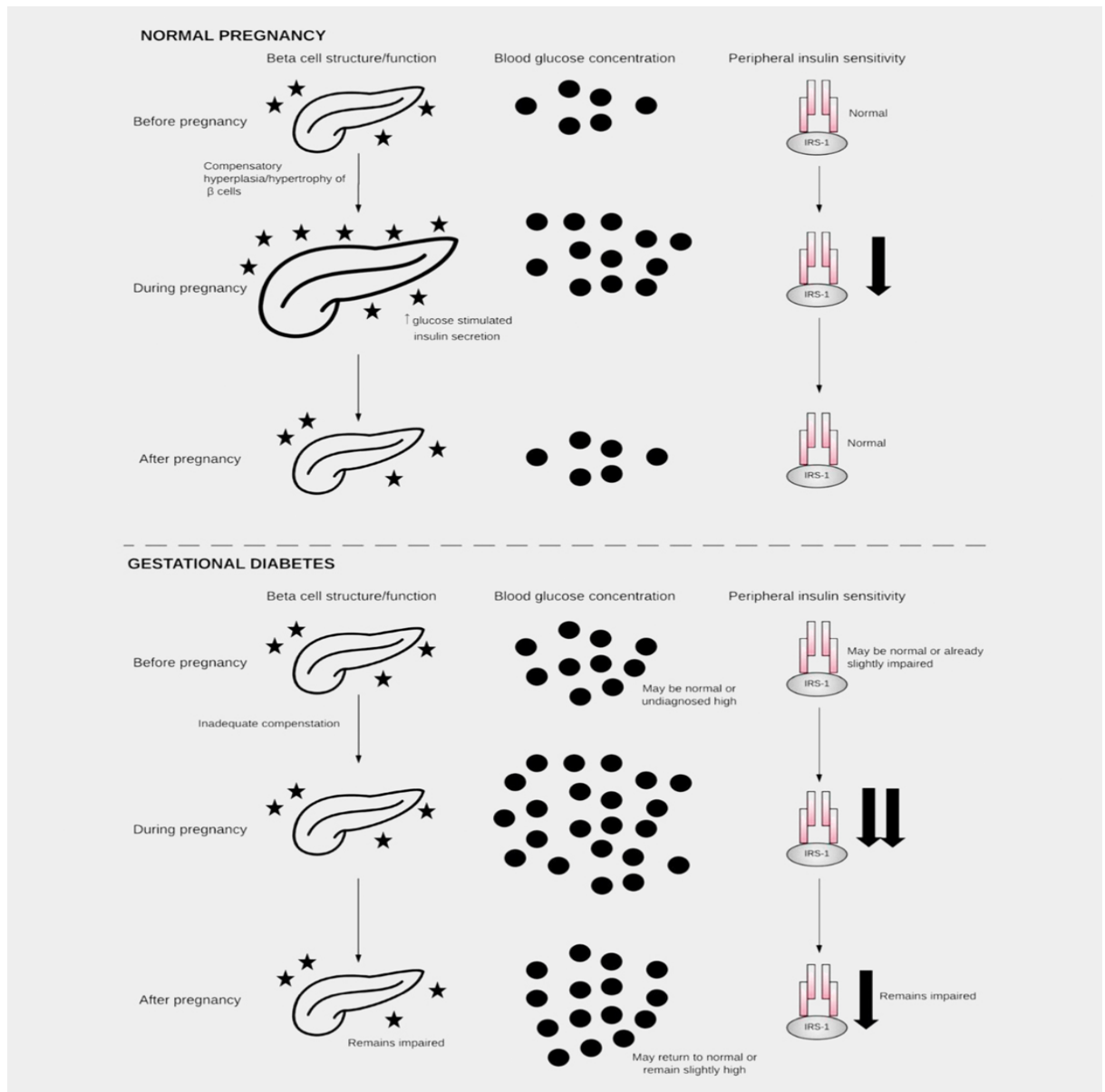
#### **Pathophysiology:**

“GDM is a pregnancy-specific condition characterized by glucose intolerance

diagnosed during pregnancy.” It occurs when a pregnant woman's body cannot effectively utilize glucose, leading to elevated blood glucose levels that affect both maternal and fetal health. The pathogenesis involves insulin resistance and  $\beta$ -cell dysfunction, typically present before conception and progressing over time. Multiple organs including the placenta, heart, brain, liver, kidney, and adipose tissues contribute to GDM development.<sup>23</sup>

**$\beta$ -Cell Dysfunction:** “ $\beta$ -Cell dysfunction occurs when pancreatic  $\beta$ -cells fail to adequately sense blood glucose levels and produce sufficient insulin in response.” The dysfunction can arise from defects in multiple processes including pro-insulin production, post-translational modifications, insulin storage, glucose sensing, and granule exocytosis. “Key genes like KQT-like 1 (Kcnq1) and glucokinase (Gck) are linked to  $\beta$ -cell function in GDM.” Persistent hyperglycemia leads to glucotoxicity, creating a destructive cycle of  $\beta$ -cell damage and worsening insulin resistance.<sup>24-26</sup>

**Chronic Insulin Resistance:** Insulin resistance in GDM manifests as cellular unresponsiveness to insulin release. The condition involves impaired glucose transporter 4 (GLUT4) translocation to plasma membranes, resulting in a 54% reduction in insulin-mediated glucose uptake compared to normal pregnancy. While insulin receptor numbers remain unchanged, altered phosphorylation patterns and changes in downstream regulators like IRS-1, PI3K, and GLUT4 contribute to persistent insulin resistance, potentially leading to Type 2 diabetes.<sup>25,27-29</sup>



**Adiponectin:** Adiponectin, primarily produced by adipocytes, regulates glucose levels and fatty acid breakdown. Its plasma concentration inversely correlates with adipose tissue mass, and reduced levels are associated with GDM. It enhances insulin sensitivity through PPAR $\alpha$  activation in the liver and AMPK within insulin-sensitive cells. Placental adiponectin affects insulin signaling and amino acid transport, with its gene methylation linked to maternal glucose intolerance and fetal macrosomia.<sup>30-32</sup>

**Placental Movement:** The placenta contributes to insulin resistance through hormone and cytokine production. In GDM, placental transport of glucose, amino acids, and lipids is affected by hyperglycemia. System A and L activity increases for amino acid transport, influenced by pro-inflammatory cytokines. Notably, lipid pathways account for 67% of changes in placental gene expression in GDM, compared to 9% for glucose pathways. Recent studies have also identified global DNA hypermethylation in GDM placentas.<sup>33-36</sup>

### **MACROSOMIA:**

An obstetric disorder called macrosomia is linked to additional potentially fatal issues for the mother and the fetus.<sup>37</sup> The Greek terms "macro," which means huge, and "somia," which means the body, are the roots of the phrase macrosomia, or "big body." "The earliest use of the term was from the work of Robley Dunglison (1798-1859), an English physician and a medical writer. However, "large for gestational age" (LGA) and "macrosomia" are the two terminology used in modern medicine to describe excessive fetal growth, according to the American College of Obstetrics and Gynecology (ACOG). The term "large for gestational age" typically refers to a birth weight that is at least 90 percentile for the gestational age in question. No matter the gestational age, the word "macrosomia" denotes growth above an absolute birth weight, traditionally 4,000 g or 4,500 g, while it is difficult to define macrosomia in a way that is widely agreed upon."<sup>38</sup>

### **Epidemiology of Macrosomia:**

"As per the review by Harvey L et al<sup>39</sup> prevalence of macrosomia ranged from 0.5% (India) to 13.9% (China) while prevalence of LGA ranged from 4.3% (Korea) to 22.1% (China), indicating substantial variation in prevalence within and between Asian countries."

## **Etiology:**

“There are two main classes into which the etiology of fetal macrosomia falls”:

### **Maternal Causes**

1. “Maternal diabetes: diabetes during pregnancy may be drug-induced/chemical, insulin-dependent, or gestational. In 1920, Jordan Pederson postulated that maternal hyperglycemia is linked to both fetal hyperglycemia and fetal hyperinsulinemia, which ultimately causes the fetus to overuse glucose and, consequently, exhibit aberrant growth.”<sup>40</sup>
2. Obesity: Obesity is currently on the rise worldwide. In all demographic groups, obesity is a substantial risk factor for diabetes mellitus. Specifically, there is a 4–12 times increase in the risk of fetal macrosomia associated with maternal obesity. It is generally accepted that elevated insulin resistance and hyperinsulinemia are the metabolic foundation of macrosomia.
3. Multiparity: Multiparity is not a significant risk factor for macrosomia in comparison to other maternal risk factors. However, it can exacerbate maternal obesity and diabetes mellitus, which are more significant reasons. Macrosomic kids are more likely to be born to women whose parity is larger than three. 41 Each pregnancy can be connected with a weight gain of 100 to 150 grams, which raises the patients' long-term risk of macrosomia.
4. Previous LGA (big for gestational age) babies: mothers who have previously given birth to a macrocosmic child are five to ten times more likely to do so again.
5. Postdate pregnancy: Because the developing fetus receives a constant supply of nutrients and oxygen-rich blood, a gestational period longer than 42 weeks is more likely to be associated with an elevated risk of macrosomia.

### **Fetal Causes**

1. Gender of the fetus: Males are more likely than females to have macrosomia. This is partially explained by the fact that male fetuses typically weigh around 150 grams more than female fetuses.
2. “Genetic and congenital disorders: Few of congenital syndromes are associated with macrosomia and LGA fetuses are:
  - Fragile X

- Sotos
- Beckwith – Weiderman
- Weaver syndrome”

### **Pathophysiology:**

During pregnancy, a variety of physiological and endocrine changes take place with the goal of providing the growing fetus with enough nourishment. Maternal and fetal risk factors could be considered the main underlying pathophysiology of macrosomia. Nonetheless, the most important contributing component to the pathophysiology of macrosomia seems to be maternal hyperglycemia. During the second trimester of pregnancy, small levels of maternal insulin resistance are caused by elevated levels of stress hormones like cortisol, human placenta lactogen (HPL), and prolactin. However, physiologic postprandial hyperinsulinemia counteracts this. “Hyperglycemia may occur in patients with metabolic syndrome or other preexisting risk factors because they are unable to establish a sufficient hyperinsulinemic response.” Fetal hyperglycemia is caused by the enhanced diffusion of glucose across the placenta. The fetal pancreatic beta islet cells then become hyperplastic as a result, which causes the fetus to use glucose excessively and hence exhibit abnormally high growth.

“The results of the Hyperglycemia and Adverse Pregnancy Outcomes study indicate a clear linear correlation between fetal obesity, fetal hyperinsulinemia, and maternal glucose concentration and large for gestational age (LGA) fetuses.”<sup>42</sup> A later meta-analysis of the association between maternal glucose levels and macrosomia (weight greater than 4,000 g) in women without diabetes shows that macrosomia is linked to either an abnormal value on oral glucose tolerance tests or a fasting blood glucose level. In a cohort of nearly 13,000 women, LGA newborns occurred in 29 percent of women with GDM type A1, 30 percent

of women with GDM type A2, and 38 percent of women with preexisting diabetes.<sup>38</sup>

### **ROLE OF ULTRASONOGRAPHY IN MONITORING FETAL GROWTH:**

ACOG recommends beginning fetal surveillance at 32 weeks for women requiring pharmacologic intervention or with uncontrolled GDM.<sup>43,44</sup> Fetal macrosomia ( $\geq 4000\text{g}$  or  $>90\text{th}$  percentile) is monitored due to risks of shoulder dystocia and trauma.<sup>45-47</sup> “Standard parameters include head circumference, biparietal diameter, abdominal circumference, and femur length.”<sup>48</sup> Studies show ultrasound tends to overestimate LGA in GDM pregnancies, with only 22.6% of predicted LGA fetuses actually being LGA at birth.<sup>49</sup> Liu et al. demonstrated that fetal hemodynamic indices (UA, MCA, renal artery) could improve birth weight prediction in GDM pregnancies.<sup>50</sup> Additionally, fetal liver length at 23 weeks correlates with OGTT results at 24 weeks.<sup>51</sup> Ultrasound findings alone shouldn't determine delivery mode, requiring comprehensive clinical assessment.<sup>52</sup>

### **ROLE OF ULTRASONOGRAPHY IN DETECTING CONGENITAL ABNORMALITIES:**

While PGDM shows higher risk (RR: 2.44), GDM has a slightly increased risk (RR: 1.11) of major congenital malformations.<sup>53</sup> Early detection is possible at 11-14 weeks, with confirmation at 20-22 weeks.<sup>54</sup> Cardiac anomalies account for 50% of perinatal mortality, with detection rates varying between 35-86%.<sup>55</sup> Fetal echocardiography shows higher accuracy (92%) compared to four-chamber view (33%) for cardiac malformations.<sup>56</sup> Neural tube defects occur more frequently in diabetic pregnancies (20/1000 vs 2/1000 in general population).<sup>54</sup>

## **ROLE OF ULTRASONOGRAPHY IN MONITORING PLACENTAL CHANGES:**

GDM affects placental development, with studies showing varied results regarding placental volume changes.<sup>57,58</sup> Recent research using three-dimensional Doppler ultrasonography demonstrated that first-trimester changes in vascularization index (VI) and vascularization flow index (VFI) could predict GDM development.<sup>59</sup> These changes occur before clinical diagnosis, attributed to increased thromboxane and tumor necrosis factor alpha levels causing vasoconstriction.<sup>60,61</sup>

## **ROLE OF ULTRASONOGRAPHY IN PREDICTING PREGNANCY OUTCOMES:**

Fetal hyperinsulinism from maternal hyperglycemia contributes to macrosomia, though its incidence is relatively low (1.1%) in GDM pregnancies.<sup>62,63</sup> Ultrasound-estimated fetal weight correlates with increased cesarean delivery rates, with LGA diagnosis via ultrasound tripling cesarean delivery risk.<sup>49</sup> Studies show that ultrasound can overestimate fetal weight by up to 10%.<sup>64</sup> While Conway et al. demonstrated reduced shoulder dystocia risk using ultrasound-based delivery timing,<sup>65</sup> the complication can still occur in neonates <4000g, highlighting ultrasound's limitations in risk prediction.<sup>49</sup> Further research is needed to identify low-risk GDM pregnancies where routine ultrasound monitoring may be unnecessary.

## **UMBILICAL CORD THICKNESS:**

**Normal Values and Variations:** The umbilical cord diameter shows progressive growth throughout gestation, with normative values established through cross-sectional studies.<sup>66</sup> At mid-pregnancy (20-24 weeks), mean diameter ranges from 7-11mm, increasing to 12-20mm at term.<sup>67</sup> Factors affecting normal variation include gestational age, fetal gender, and maternal BMI. Multiple studies have established nomograms for different gestational ages, with the 95th percentile commonly used as the upper limit of normal.<sup>68,69</sup>



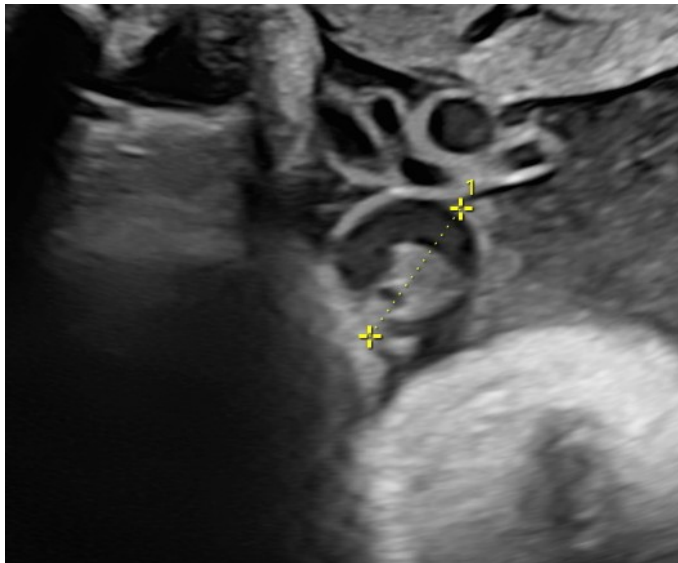
Correlation with Fetal Growth Strong positive correlations exist between umbilical cord thickness (UCT) and fetal growth parameters. Raio et al. demonstrated that increased UCT correlates significantly with increased abdominal circumference and estimated fetal weight ( $r=0.68$ ,  $p<0.001$ ). Abnormal cord thickness ( $>95$ th percentile) has been associated with a 4.2-fold increased risk of fetal macrosomia.<sup>70</sup>

Studies on UCT in GDM In pregnancies complicated by GDM, UCT tends to be significantly larger compared to non-diabetic controls. Cromi et al<sup>68</sup> discovered that, when comparing the group of macrosomic infants to the group of non-macrosomic infants, the percentage of instances with a big umbilical chord was much greater (54.7% vs. 8.7%). “In the prediction of birth weight  $> 4000$  g and  $> 4500$  g (odds ratio (95% CI), 20.6 (9.2-45.9) and 4.2 (1.2-17.7), respectively, multiple regression models showed an independent effect of the big cord.” “A sonographic big umbilical chord had sensitivity, specificity, and positive and negative predictive values of 54.7%, 91.3%, 25.4%, and 97.4%, respectively.” 100% of newborns with macrosomia were expected to have an abdomen circumference more than 95(th) centile and a big cord. “When comparing macrosomic fetuses of diabetic mothers to those of non-diabetic mothers, the percentage of umbilical cords with a Wharton's jelly area  $> 95$ (th) centile for gestation was considerably greater in the former group.” Measurement Techniques and Standardization Standard measurement involves:

- Cross-sectional view of the free loop of cord
- Measurement at mid-portion between placental and fetal insertion
- Outer-to-outer diameter measurement perpendicular to cord axis

**Quality criteria include:**

- Clear visualization of all three vessels
- Circular cross-section
- Measurement in a loop free of coiling



## **FETAL FAT LAYER<sup>71-75</sup>**

Anatomical Considerations Fetal subcutaneous fat develops primarily in the third trimester, with distinct anatomical patterns. Key measurement sites include:

- Anterior abdominal wall
- Cheek area
- Shoulder region
- Thigh
- The anterior abdominal wall demonstrates the most consistent correlation with overall fetal adiposity.

Methods of Measurement Standardized techniques include:

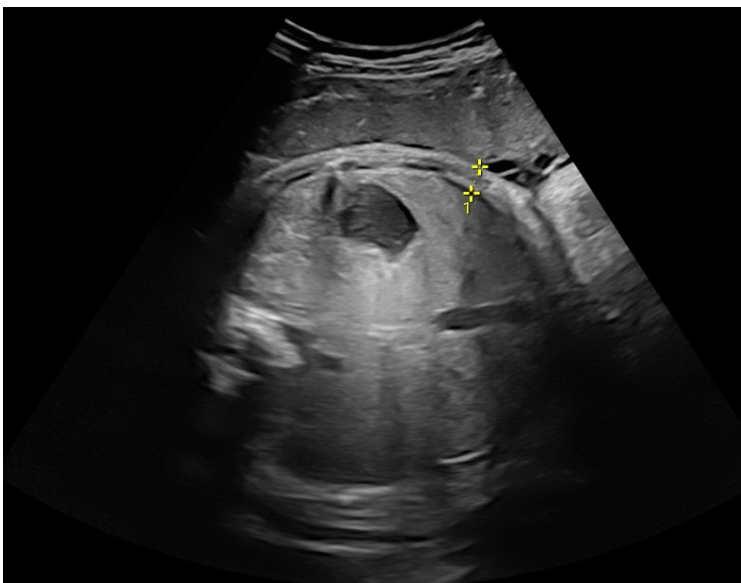
- Sagittal plane measurement at the level of the anterior abdominal wall
- Measurement between the outer skin surface and the anterior surface of liver
- Multiple measurements (usually 3) averaged for accuracy
- Current ultrasound technology allows measurement precision to 0.1mm.

Significance in Predicting Macrosomia Fetal fat layer thickness shows high predictive value for macrosomia:<sup>76-80</sup>

- Sensitivity: 81-93%
- Specificity: 75-88%
- Positive predictive value: 78% when measured at 36 weeks Cut-off values of >5mm at the anterior abdominal wall at 36 weeks demonstrate significant association with birth weight >4000g (OR 3.8, 95% CI 2.1-6.9).

Previous Studies on Fetal Adiposity in GDM Multiple studies have established:

- Earlier onset of fat accumulation in GDM pregnancies
- Different distribution patterns compared to non-diabetic pregnancies
- Stronger correlation with adverse outcomes A landmark study by Langer et al. demonstrated that fetal fat layer measurements  $>4.5\text{mm}$  at 34 weeks had 87% sensitivity for predicting macrosomia in GDM pregnancies..



### **INTERVENTRICULAR SEPTAL THICKNESS:<sup>81-92</sup>**

Cardiac Changes in Infants of Diabetic Mothers Maternal diabetes significantly affects fetal cardiac development. The primary manifestation is interventricular septal hypertrophy, predominantly affecting the anterior portion. This hypertrophy occurs due to fetal hyperinsulinemia, which promotes increased glucose uptake and glycogen storage in cardiac myocytes. The septal thickening typically develops in the third trimester and may persist for several months after birth.

Measurement Techniques Standard measurement protocols include:

- Four-chamber view of the fetal heart at end-diastole
- Measurement perpendicular to the septum at the level of AV valve leaflets
- Average of three consecutive measurements
- Modern ultrasound equipment with high-resolution capabilities allows precise measurements to 0.1mm accuracy.

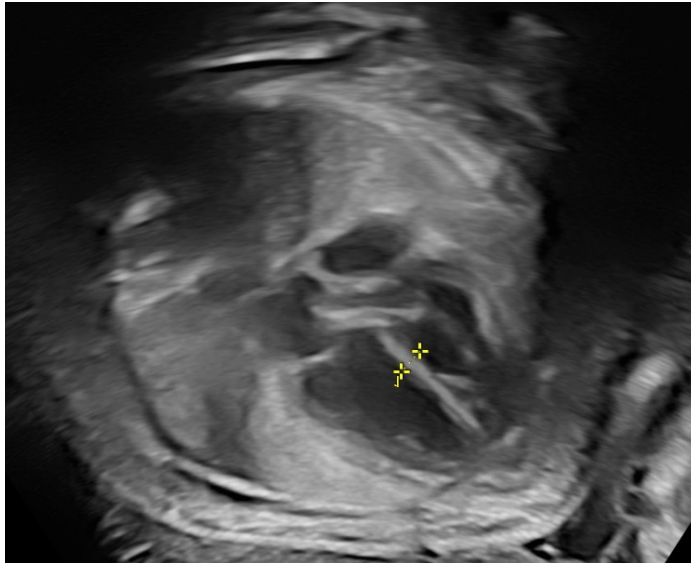
Normal Ranges and Variations IVS thickness shows gestational age-dependent variation:

- 24-28 weeks: 2.0-3.0mm
- 28-32 weeks: 2.5-3.5mm
- 32-36 weeks: 3.0-4.0mm
- 36 weeks: 3.5-4.5mm

Values exceeding the 95th percentile for gestational age are considered abnormal. In GDM pregnancies, IVS measurements typically exceed these ranges by 20-40%.

Correlation with Macrosomia: Studies demonstrate strong correlation between increased IVS thickness and fetal macrosomia:

- Sensitivity: 83-87%
- Specificity: 77-81%
- Positive predictive value: 75% for birth weight >4000g



IVS measurements  $>6\text{mm}$  at 36 weeks show significant association with:

- Macrosomia (OR 4.2, 95% CI 2.8-6.3)
- Neonatal complications
- Need for NICU admission

Clinical Implications Serial measurements of IVS thickness can:

- Help predict macrosomia risk
- Guide diabetes management
- Indicate need for closer fetal surveillance
- Aid in delivery timing decisions

## **REVIEW OF RELATED ARTICLES:**

In a study by Janani N et al<sup>93</sup> “the thickness of the umbilical cord had a negative predictive value and high sensitivity. “Therefore, the likelihood of macrosomia is lower if the umbilical cord thickness is less than the 90th percentile. The cut off of the fetal fat layer  $\geq 5\text{mm}$  as a predictor of macrosomia had a sensitivity of 84.2 percentage, specificity of 86.4

percentage, and a negative predictive value of 95.9 percentage. The cutoff of the interventricular septal thickness  $\geq 3.9$  mm had a sensitivity of 84.2 percentage, specificity of 64.2 percentage, and negative predictive value of 95.9 percentage”.

Mohamed MF et al<sup>94</sup> in their research found that the “umbilical cord thickness (cm) 2.77 0.72 versus 2.06 0.44, the interventricular septum thickness (cm) 0.85 0.20 versus 0.53 0.08, and the placental volume (cm<sup>3</sup>) were all statistically significantly higher in the GDM and macrosomic group compared with the controls; however, the placental thickness (cm) was statistically significantly lower in the GDM and macrosomic group compared with the controls.” They concluded that most accurate measure of fetal macrosomia in pregnancies with gestational diabetes mellitus was the thickness of the interventricular septum as determined by sonography.

In a study by Geetha M et al,<sup>95</sup> “A cut of belly circumference  $>35$  cm and fetal fat layer  $>5$  mm were the ultrasonography characteristics with the best sensitivity, specificity, and negative predictive value for predicting macrosomia. The negative predictive value was good when the umbilical cord thickness was more than the 90th percentile.”

Pandey D et al<sup>96</sup> “The average age and BMI of the women in their study were  $27.9 \pm 2.84$  years and  $26.05 \pm 1.32$  kg/m<sup>2</sup>, respectively. For CT and CSA, the big cord cut-off was 2.8 cm and 3.56 cm<sup>2</sup>, respectively. Seventy percent of the study group had large cords. Macrosomia was discovered in 17.5% of the research group's cases, and sonographically detected umbilical-cord parameters were noticeably greater in macrosomic fetuses than in nonmacrosomic fetuses. To predict macrosomia, the cord parameters' sensitivity, specificity, positive predictive value, and negative predictive value were, respectively, 57.1, 96.9, 80, and 91.4% for CT and 65.7, 63.6, 46.2, and 87.5% for CSA.”

Abdelrahman RM et al<sup>97</sup> discovered that research participants with gestational diabetes had an increased umbilical cord diameter ( $3.03 \pm 1.26$ ) compared to the control group. “Fetal

macrosomia in diabetes patients has been associated with an increase in interventricular septal thickness ( $0.85 \pm 0.51$  cm.” Patients with GDM had higher observed fetal macrosomia cases, as shown by their HbA1c values ( $7.0 \pm 1.2\%$ ). In situations of pregnant diabetes mellitus, the research study demonstrated the sonographic use of evaluating the thickness of the umbilical cord, the interventricular septum, and HbA1c as a prediction tool for fetal macrosomia.

Stanirowski PJ et al<sup>98</sup> “According to their findings, patients with GDMG2/T1DM had significantly higher measurements of the fetal SSFM, AFM, MTFM, MTFM/MTLM ratio, HeC, HeA, IVS, LL, UmC, UmA, UaC, UaA, UveA, and WjA when compared to GDMG1 and/or control groups ( $p < .05$ ). Maternal height, fetal biparietal diameter, abdominal circumference (AC), AFM, and LL measures were all independent predictors of the FBW, according to the regression analysis ( $p < 0.05$ ).” Furthermore, a substantial likelihood of fetal macrosomia incidence was linked to increases in fetal AFM, AC, and femur length (FL) ( $p < .05$ ). “For the subgroup of women with T1DM, the FBW estimate equation  $[FBW(g) = -2254,942 + 17,204 * FL (mm) + 105,531 * AC (cm) + 131,347 * AFM (mm)]$  yielded a considerably lower mean absolute percent error than the usual method (5.7% vs 9.4%,  $p < 0.05$ ).” Furthermore, in the prediction of fetal macrosomia, a novel equation incorporating the AC, FL, and AFM parameters produced a sensitivity of 93.8%, specificity of 77.7%, positive predictive value of 54.5%, and negative predictive value of 97.8%.

Garg S<sup>99</sup> in his research revealed that the women in the study had a mean age of  $27.9 \pm 2.84$  years and a BMI of  $26.05 \pm 1.32$  kg/m<sup>2</sup>. For CT and CSA, the big cord cut-off was 2.8 cm and 3.56 cm<sup>2</sup>, respectively. Seventy percent of the study group had large cords. “Macrosomia was discovered in 17.5% of the research group's cases, and sonographically detected umbilical-cord parameters were noticeably greater in macrosomic fetuses than in nonmacrosomic fetuses. In order to predict macrosomia, the cord parameters' sensitivity, specificity, positive predictive value, and negative predictive value were 57.1, 96.9, 80, and



91.4% for CT and 65.7, 63.6, 46.2, and 87.5% for CSA, respectively.”

Bethune M et al<sup>100</sup> found that a “fetal fat layer of  $\geq 5$  mm was the most useful predictor of macrosomia at term as determined by the likelihood ratio. This study, which involved 90 patients, evaluated the measurement of the fetal abdominal fat layer (FFL), cardiac interventricular septum (IVS), and abdominal circumference (AC) percentile in the early third trimester as predictors of macrosomia at birth in the fetuses of women with gestational diabetes.” Nonetheless, the sensitivity was higher with an AC  $\geq 90$ th percentile. They came to the conclusion that more research should be done to determine whether routine FFL assessment in the early third trimester is helpful in managing diabetes pregnancies.

Cromi A<sup>68</sup> has out a study to ascertain whether fetal macrosomia is predicted by a large cross sectional area of the umbilical cord. In comparison to the group of infants who were not macrosomic, they discovered that the percentage of instances with a big umbilical chord was much higher in the macrosomic group (54.7% vs. 8.7%,  $P < 0.0001$ ). “In the prediction of birth weight  $> 4000$  g and  $> 4500$  g (odds ratio (95% CI), 20.6 (9.2–45.9) and 4.2 (1.2–17.7), respectively, multiple regression models showed an independent effect of the big cord.” “A sonographic big umbilical chord had sensitivity, specificity, and positive and negative predictive values of 54.7%, 91.3%, 25.4%, and 97.4%, respectively.” 100% of newborns with macrosomia were predicted to have an abdomen circumference more than the 95th centile and a big cord. Macrosomic fetuses of diabetic moms had a considerably larger percentage of umbilical cords with a Wharton's jelly area  $> 95$ th centile for gestation than fetuses of non-diabetic mothers.

A prospective observational case–control study by Ghuman GK et al<sup>101</sup> was carried out with 35 GDM patients and 35 healthy pregnancies. The f EFT and f IVST were measured ultrasonographically between 24 and 32 weeks of gestation. “The threshold value, sensitivity, specificity, and diagnostic accuracy of these two parameters for the prediction of GDM were

determined using statistical analysis and receiver operating characteristic curves. GDM could be predicted with a sensitivity of 68.6%, specificity of 91.4%, PPV of 88.9%, NPV of 74.4%, and diagnostic accuracy of 80% when a threshold value of 1.3 for the f EFT was adopted. GDM could be predicted with a sensitivity of 80%, specificity of 77.14%, PPV of 77.8%, NPV of 79.4%, and diagnostic accuracy of 78.5% when a cut-off value of 2.6 for f IVST was established.”

## MATERIAL AND METHODS

- **Study design:** Prospective study
- **Study area:** Department of Radio-Diagnosis, Shri B. M. Patil Medical College and Hospital, Vijayapura.
- **Study period:** Research study was conducted from April 2023 to April 2025. Below is the work plan.

**Table 1: Work plan of the study with percentage of allocation of study time and duration in months**

Work plan	% of allocation of study time	Duration in months
Understanding the problem, preparation of questionnaire.	5-10%	April 2023 to June 2023
Pilot study, Validation of questionnaire, data collection and manipulation	Upto 80%	July 2023 to September 2024
Analysis and interpretation	5-10%	October 2024 to January 2025
Dissertation write-up and submission	5-10%	February 2025 to April 2025

- **Sample size:** 123

As per the study done by Divya pandey in her thesis stated that “sonographically detected umbilical cord thickness and interventricular septal thickness are good predictors of fetal macrosomia”.

By above considerations average prevalence of macrosomia in pregnant women with gestational diabetes can be considered as 8.75% .Considering the confidence limit of these

studies to be 95% with 5% level of significance and margin of error 0.05. The sample size computed using the following formula

$$\text{Sample size (n)} = (Z^2 * p * (1-p)) / d^2$$

Where,

**z** is the z score= 1.96

**d** is the margin of error= 0.05

**n** is the population size

**p** is the population proportion = 0.0875

The estimated sample size of this study is **123**.

- **Inclusion criteria:**

- **1. Pregnant woman:**

- Identified as having gestational diabetes mellitus, either by insulin or a meal plan
- A gestational age > 27 weeks+ 6 days.
- Singleton pregnancy
- 3 vessel umbilical cords;
- Reliable dates verified by dating scans;

- **Exclusion criteria**

1. A gestational age of < 27 weeks+ 6 days.
2. More than 1 gestations.
3. A patient who is either overtly diabetic or not.

## **METHODOLOGY:**

The research was performed at Shri B. M. Patil Medical College and Hospital, Vijayapura over a period of 2 years, after obtaining approval from the Institutional Ethics Committee and obtaining informed consent from all participating patients. The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants. Data confidentiality was maintained throughout the study period.

## **Data Collection**

Demographic data including maternal age, body mass index (BMI), parity, and family history of diabetes were collected at enrollment. Glycemic control was monitored through fasting and postprandial blood glucose levels, and HbA1c values were recorded monthly.

## **Equipment**

Ultrasound examinations were performed using two machines:

1. GE Voluson S8 BT18
2. GE Versana Premier

Both machines were equipped with curvilinear transducers suitable for obstetric scanning.

## **Sonographic Measurements**

All ultrasound examinations were performed using a Voluson S8 BT18 ultrasound machine (GE Healthcare) equipped with a 4-8 MHz transabdominal probe. Three certified sonographers, who were blinded to the maternal glycemic status, conducted the measurements. Each parameter was measured three times, and the average value was recorded.

### **Umbilical Cord Thickness**

The umbilical cord thickness was measured in a cross-sectional view at the mid-portion of the cord, perpendicular to its long axis. The measurement included the diameter of the cord from outer edge to outer edge of Wharton's jelly, avoiding any coiling or vessels.

### **Fetal Fat Layer**

Subcutaneous fat thickness was measured at three standardized locations: the anterior abdominal wall at the level of liver, mid-thigh, and mid-arm. Measurements were taken perpendicular to the skin surface, excluding the skin layer.

### **Interventricular Septal Thickness**

The interventricular septum was measured in the four-chamber view of the heart during diastole, using the leading-edge-to-leading-edge method. Care was taken to avoid including the moderator band or trabeculae in the measurement.

### **Follow-up and Outcome Assessment**

Participants underwent regular antenatal check-ups every two weeks until delivery. Sonographic measurements were repeated at 4-week intervals. Birth weight was recorded immediately after delivery, and macrosomia was defined as birth weight >4000g or >90th percentile for gestational age.

### **STATISTICAL ANALYSIS**

Data was entered in excel sheet and analyzed using SPSS version 21. Results were presented in tabular and graphical forms Mean, median, standard deviation and ranges were calculated for quantitative data. Qualitative data were expressed in terms of frequency and percentages. Student t test (Two Tailed) was used to test the significance of mean and P value <0.05 was considered significant.

### **RESULTS**

The present study was conducted in the department of Radiodiagnosis to analyze the fetal fat layer, umbilical cord thickness, and interventricular septal thickness sonographic measures to predict fetal macrosomia.

Total Of 123 patients were included in the study.

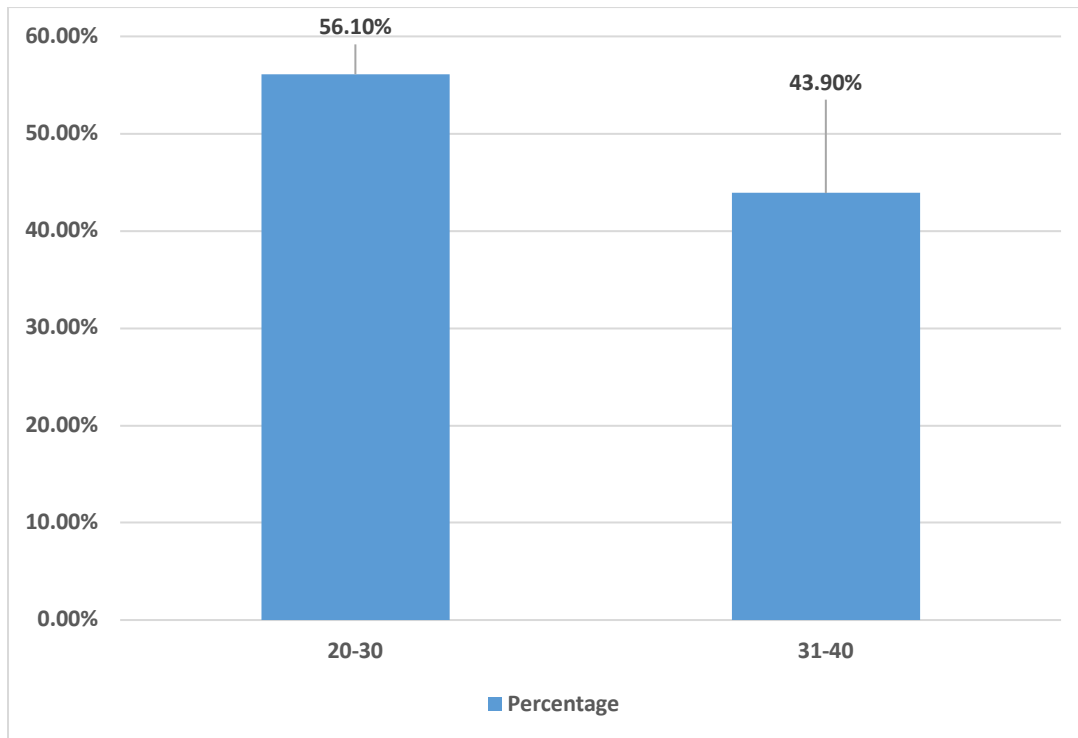
Following were the results of the study:

**Table 1: Distribution of patients according to age**

Age (in years)	Frequency	Percentage
20-30	69	56.1%
31-40	54	43.9%
Total	123	100%

Table 1 and graph 1 shows the age distribution of the study participants, with a majority (56.1%) falling in the 20-30 years age group, while 43.9% were in the 31-40 years age group, for a total of 123 participants.

**Graph 1: Distribution of patients according to age**



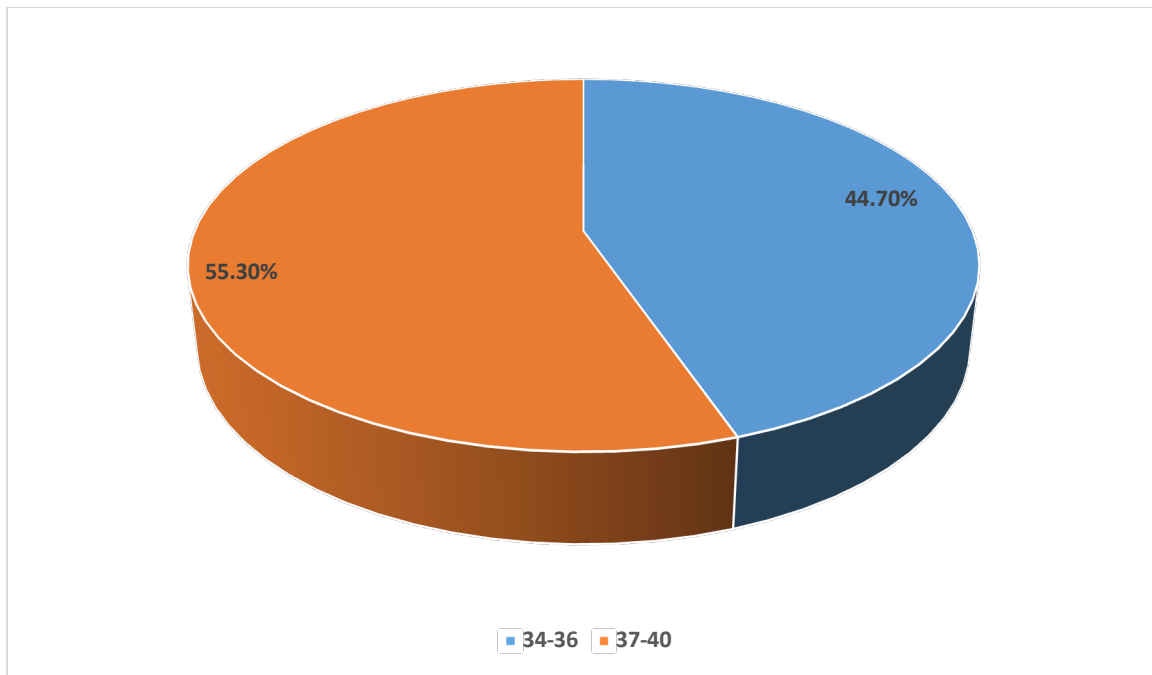
**Table 2: Distribution of patients according to gestational age**

Gestational age	Frequency	Percentage
34-36	55	44.7%
37-40	68	55.3%
<b>Total</b>	<b>123</b>	<b>100%</b>

Table 2 and graph 2 illustrates the gestational age distribution, with 44.7% of pregnancies at 34-36 weeks and a slightly higher percentage (55.3%) at 37-40 weeks, indicating a good balance between late preterm and term pregnancies.

**Graph 2: Distribution of patients according to gestational age**



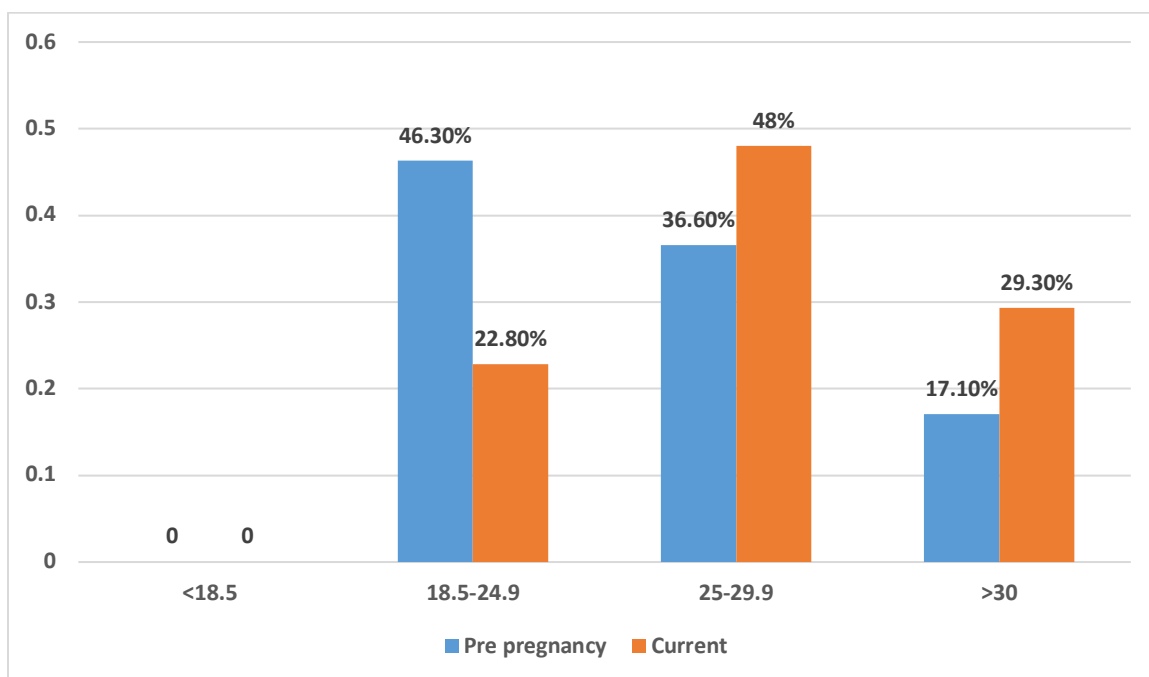


**Table 3: Distribution of patients according to BMI**

BMI	Pre pregnancy	Current
<18.5	0	0
18.5-24.9	57 (46.3%)	28 (22.8%)
25-29.9	45 (36.6%)	59 (48%)
>30	21 (17.1%)	36 (29.3%)

Table 3 and graph 3 demonstrates a significant shift in BMI from pre-pregnancy to current measurements, with notable increases in the overweight (25-29.9) category from 36.6% to 48% and the obese (>30) category from 17.1% to 29.3%, suggesting considerable weight gain during pregnancy.

**Graph 3: Distribution of patients according to BMI**

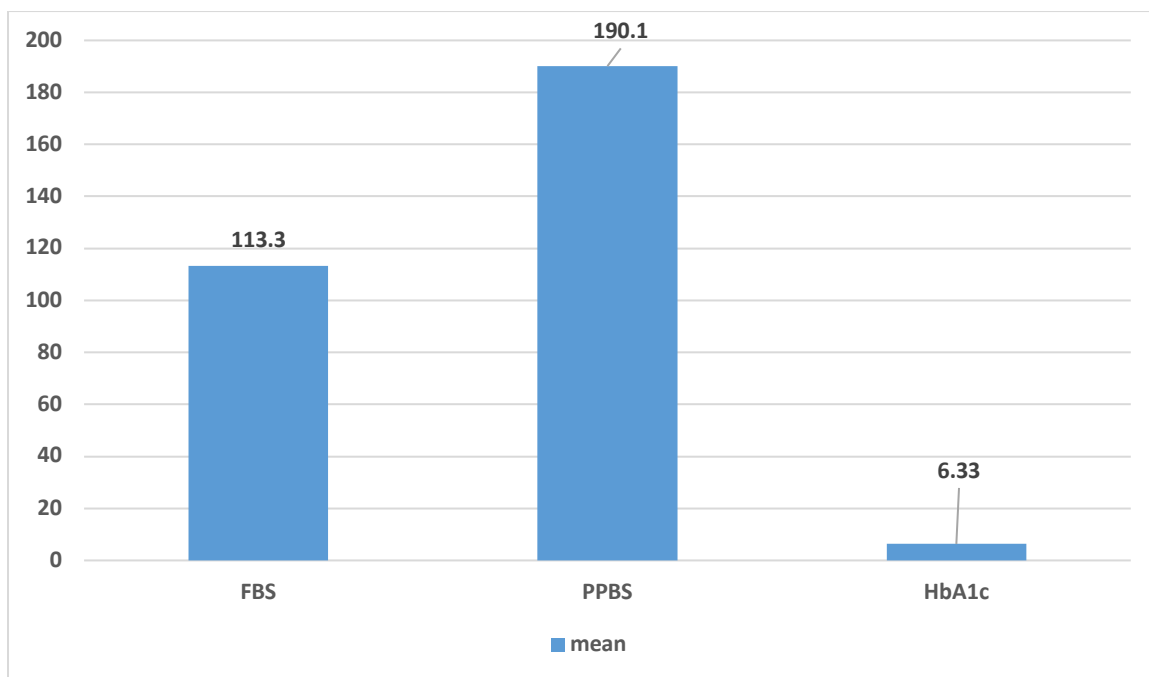


**Table 4: Distribution of patients according to blood sugar levels**

blood sugar levels	FBS	PPBS	HbA1c
Mean	113.3	190.1	6.33
SD	18.1	35.8	0.72

Table 4 and graph 4 presents the blood sugar parameters of the study population, showing mean fasting blood sugar of 113.3 mg/dL, post-prandial blood sugar of 190.1 mg/dL, and HbA1c of 6.33%, all of which indicate elevated glycemic values consistent with gestational diabetes.

**Graph 4: Distribution of patients according to blood sugar levels**

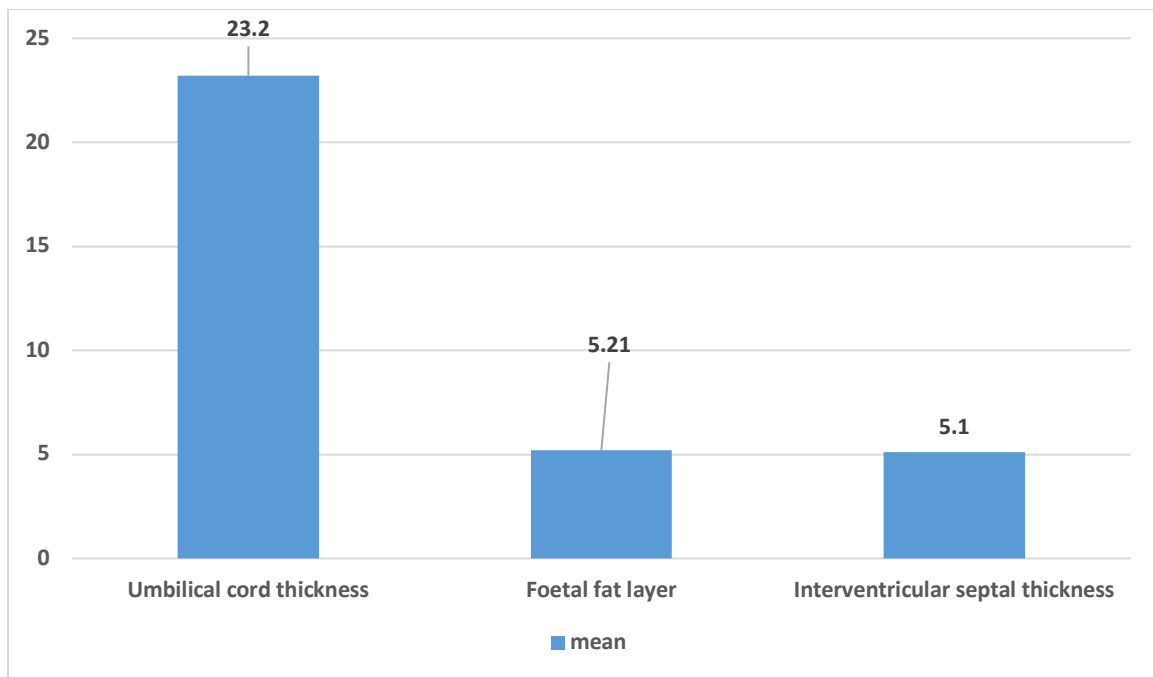


**Table 5: Distribution of patients according to sonographic measurements**

Measurements (mm)	Umbilical cord thickness	Foetal fat layer	Interventricular septal thickness
Mean	23.2	5.21	5.1
SD	6.9	1.2	1.33

Table 5 and graph 5 displays the mean sonographic measurements with umbilical cord thickness averaging 23.2 mm (SD 6.9), fetal fat layer averaging 5.21 mm (SD 1.2), and interventricular septal thickness averaging 5.1 mm (SD 1.33), providing baseline data for these parameters.

**Graph 5: Distribution of patients according to sonographic measurements**

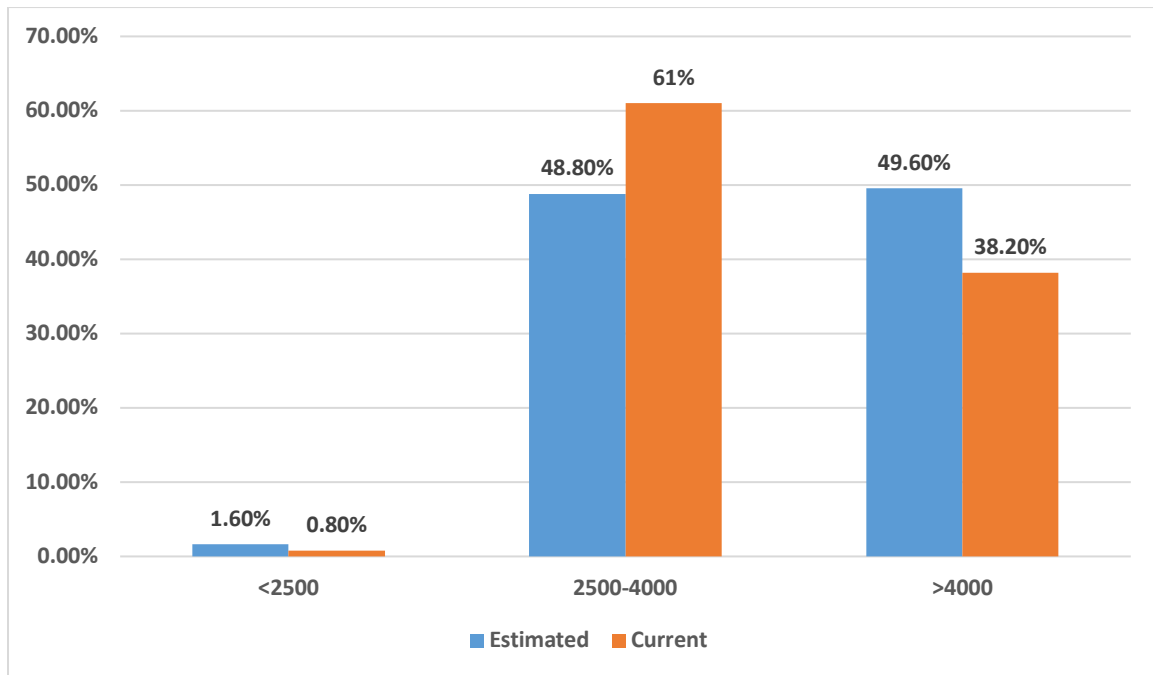


**Table 6: Distribution of patients according to birth weight**

Birth weight (grams)	Estimated	Current
<2500	2 (1.6%)	1 (0.8%)
2500-4000	60 (48.8%)	75 (61%)
>4000	61 (49.6%)	47 (38.2%)

Table 6 and graph 6 compares estimated versus actual birth weights, showing a shift toward more normal weight babies (2500-4000g) in actual birth weights (61%) compared to estimated (48.8%), and fewer macrosomic babies (>4000g) than estimated (38.2% actual vs 49.6% estimated).

**Graph 6: Distribution of patients according to birth weight**

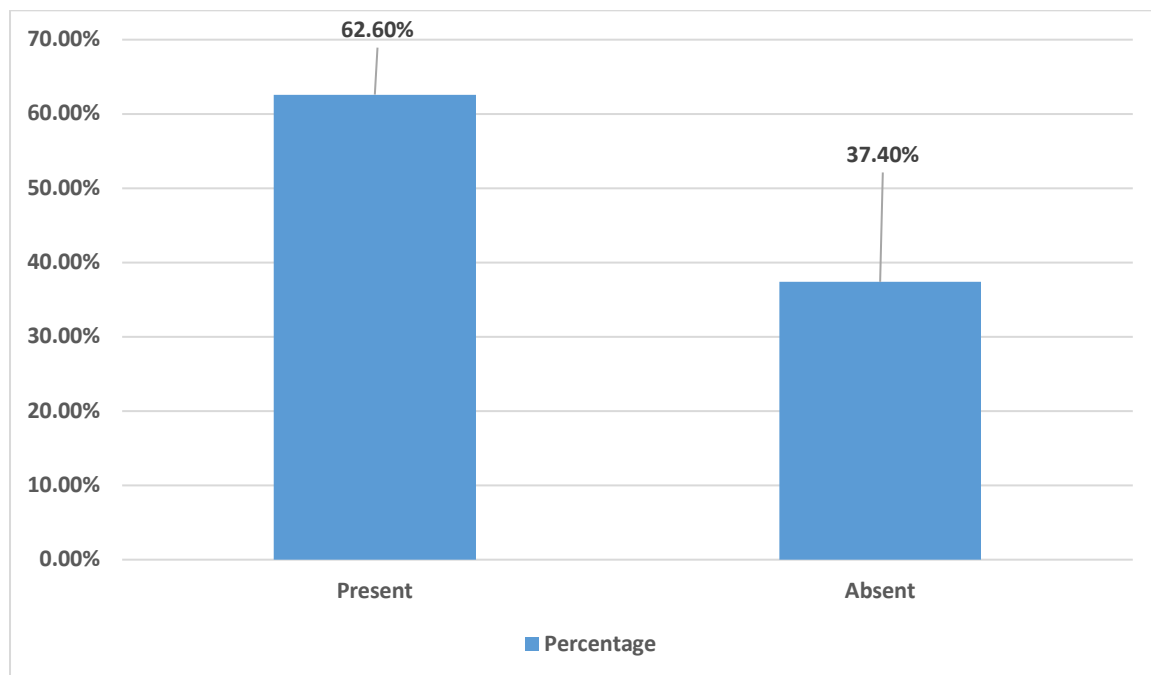


**Table 7: Distribution of patients according to macrosomia**

Macrosomia	Frequency	Percentage
Present	77	62.6%
Absent	46	37.4%
Total	123	100%

Table 7 and graph 7 reveals that a significant proportion (62.6%) of the fetuses in this study exhibited macrosomia, while 37.4% did not, highlighting the high prevalence of macrosomia in pregnancies complicated by gestational diabetes.

**Graph 7: Distribution of patients according to macrosomia**

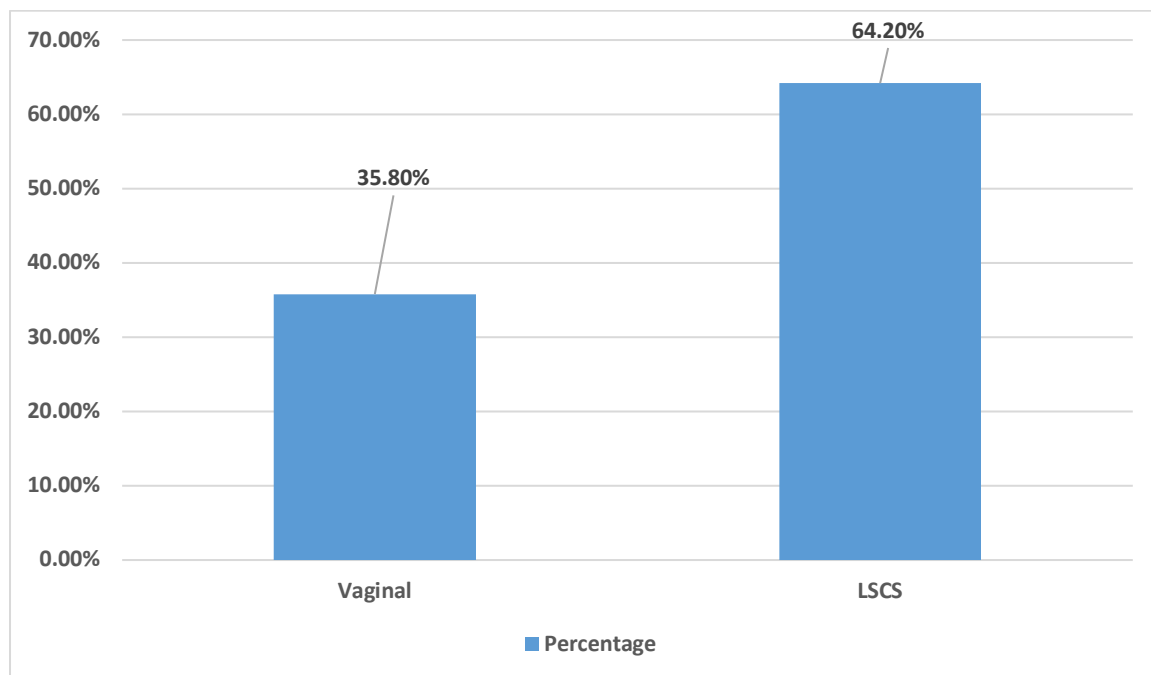


**Table 8: Distribution of patients according to mode of delivery**

Mode of delivery	Frequency	Percentage
Vaginal	44	35.8%
LSCS	79	64.2%
Total	123	100%

Table 8 and graph 8 shows that cesarean section (LSCS) was the predominant mode of delivery at 64.2%, while vaginal deliveries accounted for 35.8%, likely reflecting concerns about delivery complications in this high-risk population.

**Graph 8: Distribution of patients according to mode of delivery**

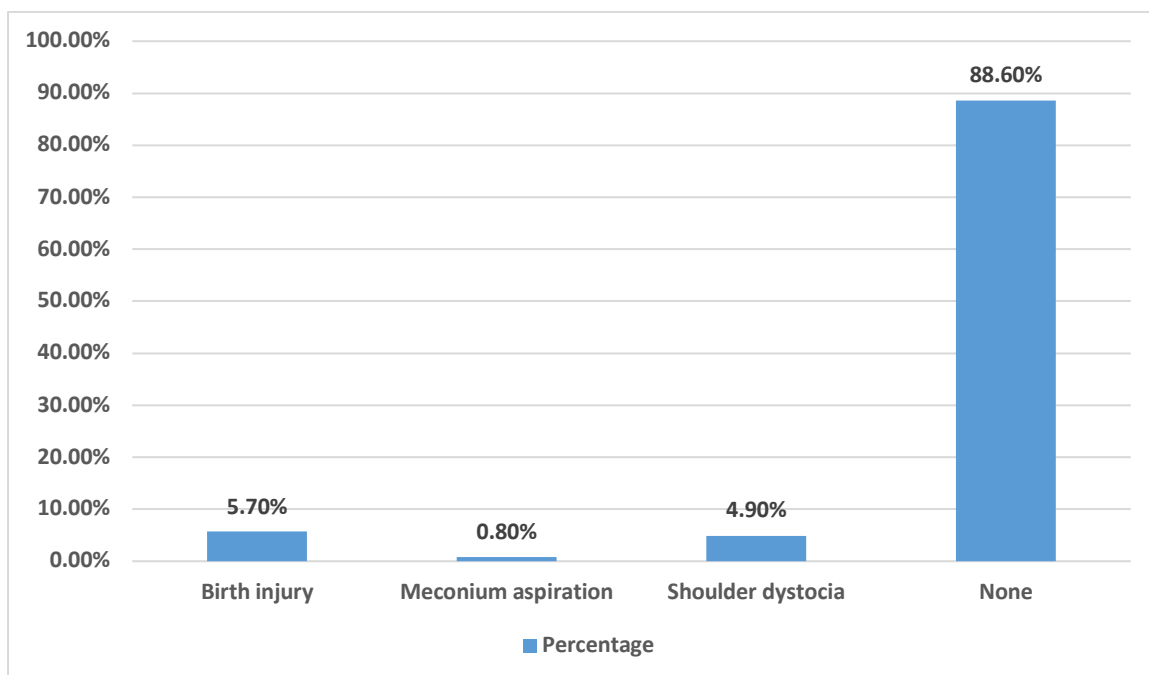


**Table 9: Distribution of patients according to complications**

Complications	Frequency	Percentage
Birth injury	7	5.7%
Meconium aspiration	1	0.8%
Shoulder dystocia	6	4.9%
None	109	88.6%
Total	123	100%

Table 9 and graph 9 summarizes delivery complications, with the majority (88.6%) having no complications, while birth injuries occurred in 5.7%, shoulder dystocia in 4.9%, and meconium aspiration in 0.8% of cases.

**Graph 9: Distribution of patients according to complications**



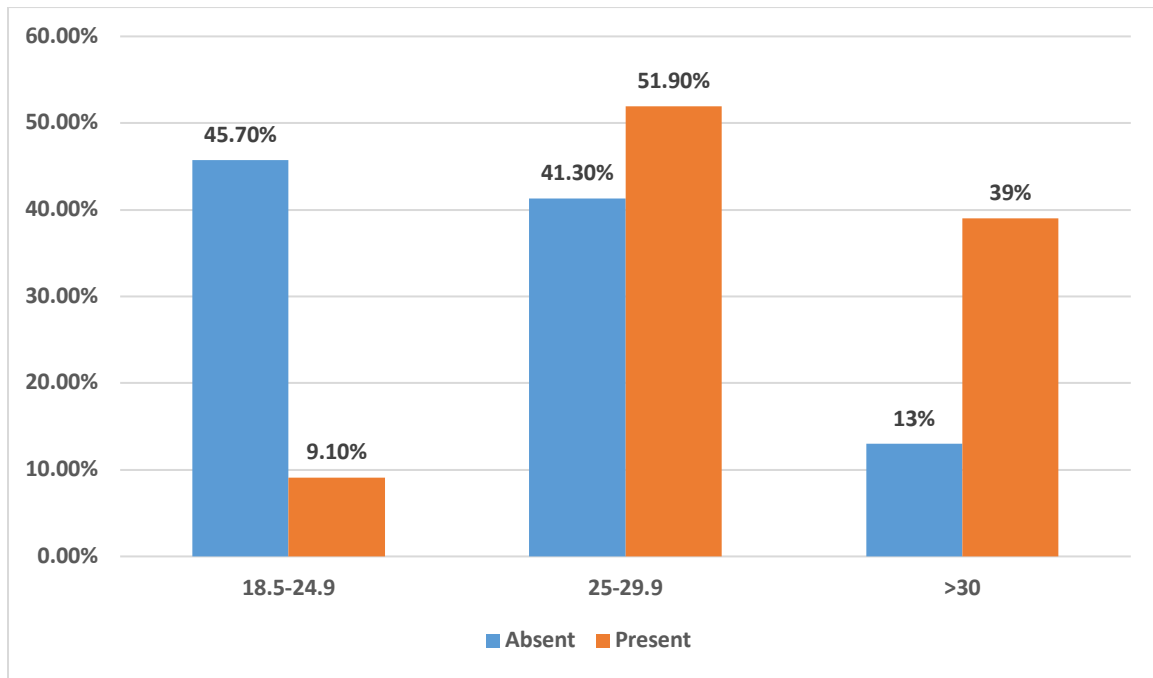
**Table 10: Association of Macrosomia with BMI**

BMI	Macrosomia		p-value
	Absent	Present	
18.5-24.9	21 (45.7%)	7 (9.1%)	<0.001
25-29.9	19 (41.3%)	40 (51.9%)	
>30	6 (13%)	30 (39%)	
<b>Total</b>	<b>46 (100%)</b>	<b>77 (100%)</b>	

Table 10 and graph 10 demonstrates a highly significant association ( $p < 0.001$ ) between increasing maternal BMI and fetal macrosomia, with 39% of macrosomic babies born to mothers with BMI  $>30$  compared to only 9.1% born to mothers with normal BMI.

**Graph 10: Association of Macrosomia with BMI**



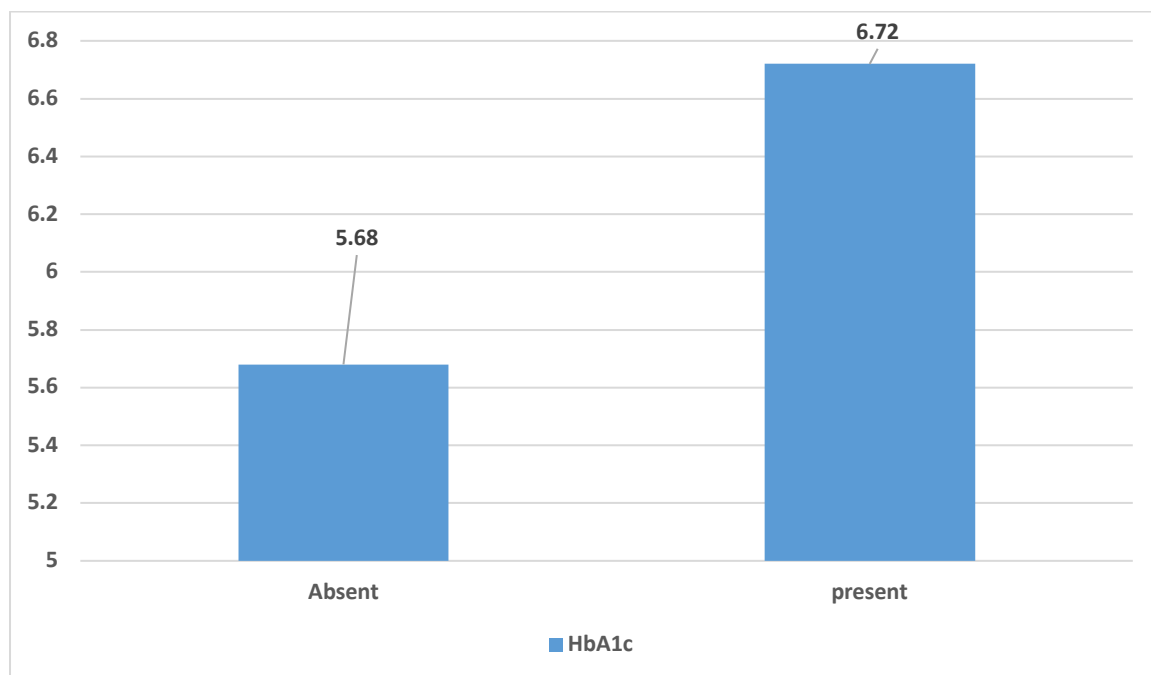


**Table 11: Association of Macrosomia with HbA1c**

HbA1c	Macrosomia		p-value
	Absent	Present	
mean±SD	5.68±0.63	6.72±0.44	<0.001

Table 11 and graph 11 shows a strong association between HbA1c levels and macrosomia ( $p < 0.001$ ), with mothers of macrosomic babies having significantly higher mean HbA1c ( $6.72 \pm 0.44$ ) compared to mothers without macrosomic babies ( $5.68 \pm 0.63$ ).

**Graph 11: Association of Macrosomia with HbA1c**

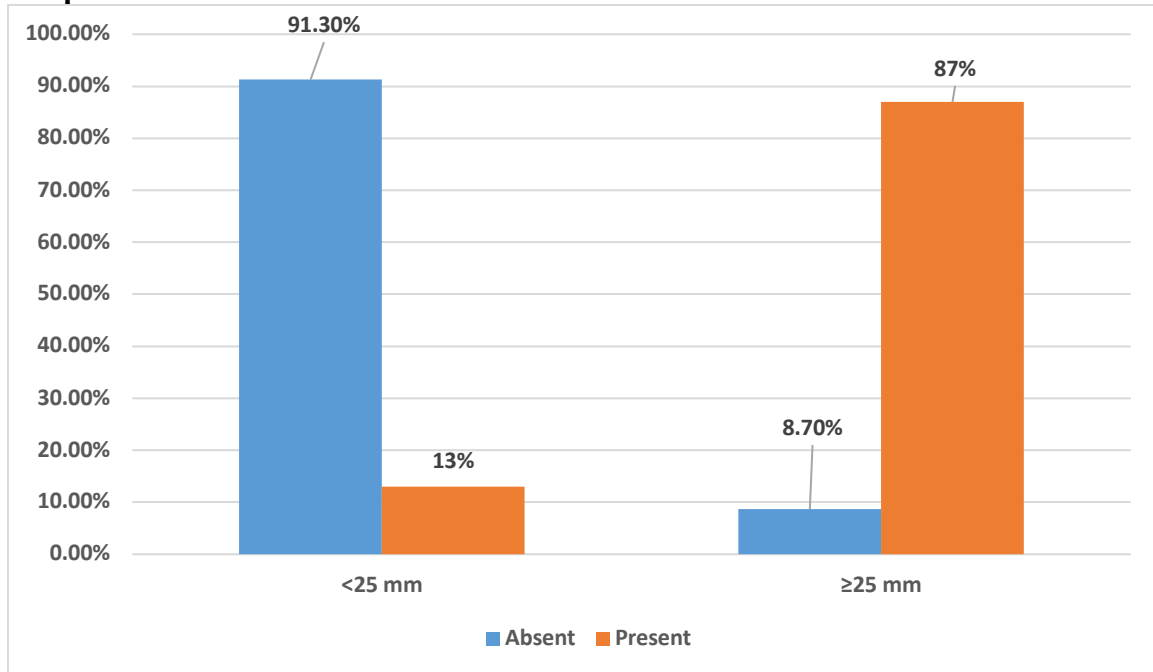


**Table 12: Association of Macrosomia with umbilical cord thickness**

Umbilical thickness	cord	Macrosomia		p-value
		Absent	Present	
<25 mm		42 (91.3%)	10 (13%)	<0.001
≥25 mm		4 (8.7%)	67 (87%)	
Total		46 (100%)	77 (100%)	

Table 12 and graph 12 reveals a strong correlation between umbilical cord thickness and macrosomia ( $p < 0.001$ ), with 87% of macrosomic fetuses having umbilical cord thickness  $\geq 25$  mm, compared to only 8.7% of non-macrosomic fetuses.

**Graph 12: Association of Macrosomia with umbilical cord thickness**

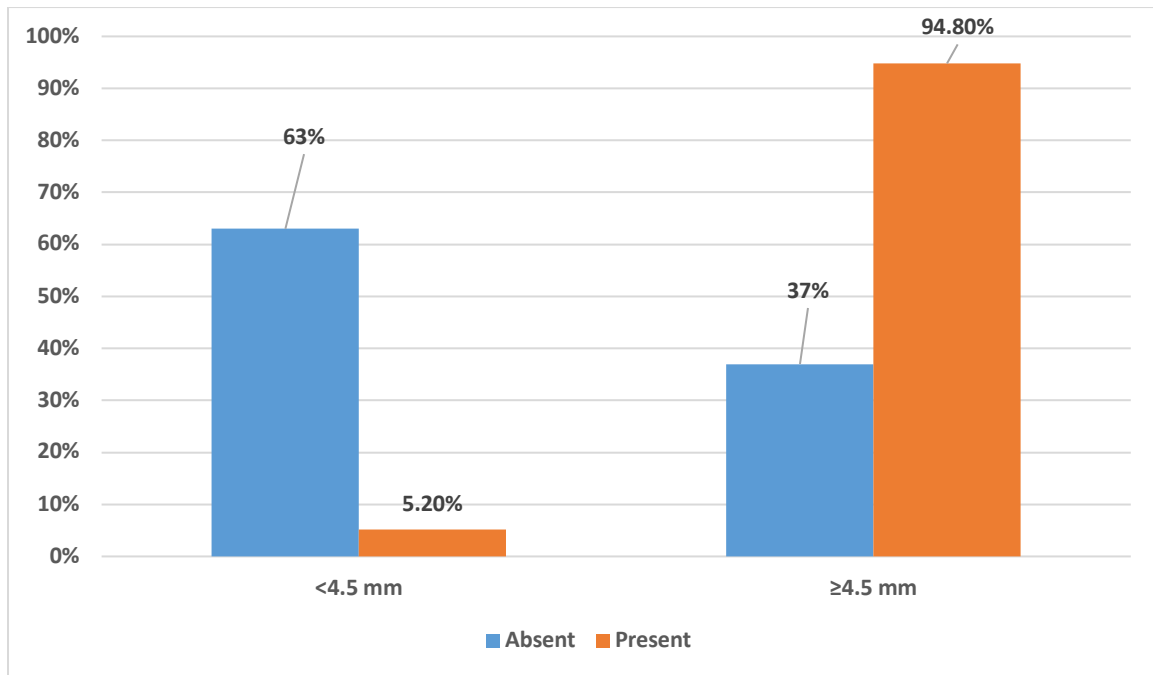


**Table 13: Association of Macrosomia with foetal fat layer**

Foetal fat layer	Macrosomia		p-value
	Absent	Present	
<4.5 mm	29 (63%)	4 (5.2%)	<0.001
≥4.5 mm	17 (37%)	73 (94.8%)	
Total	46 (100%)	77 (100%)	

Table 13 and graph 13 demonstrates a significant association between fetal fat layer thickness and macrosomia ( $p < 0.001$ ), with 94.8% of macrosomic fetuses having fat layer  $\geq 4.5$  mm, compared to only 37% of non-macrosomic fetuses.

**Graph 13: Association of Macrosomia with foetal fat layer**

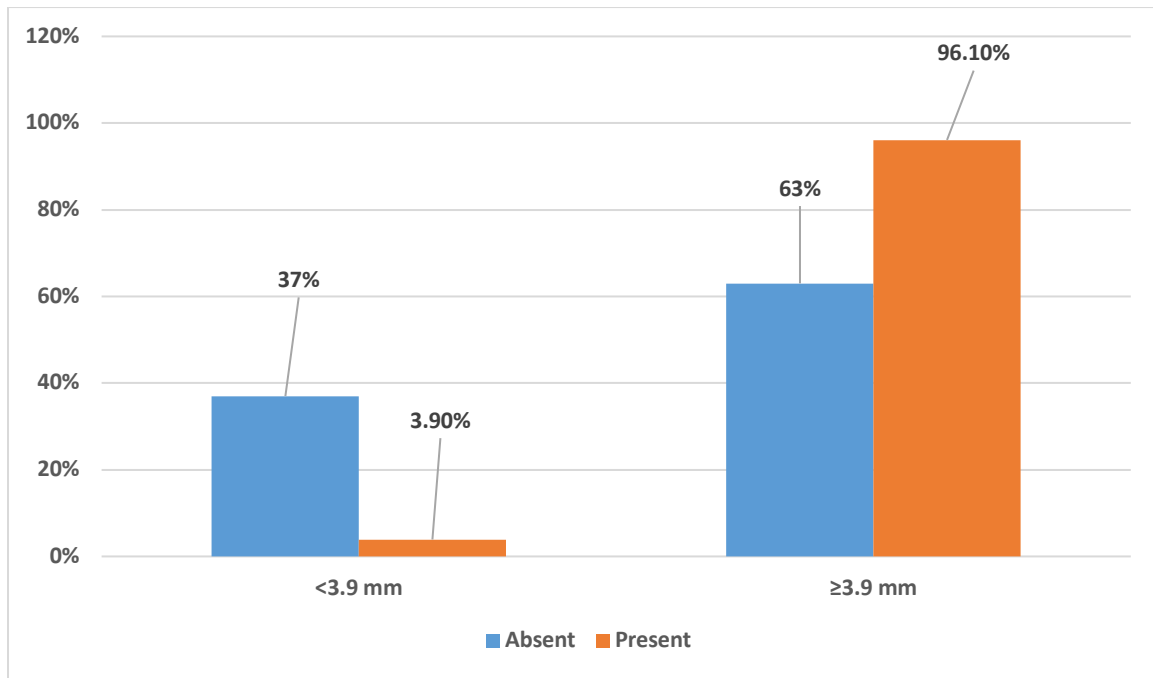


**Table 14: Association of Macrosomia with inter ventricular septal thickness**

Inter ventricular septal thickness	Macrosomia		p-value
	Absent	Present	
<3.9 mm	17 (37%)	3 (3.9%)	<0.001
≥3.9 mm	29 (63%)	74 (96.1%)	
<b>Total</b>	<b>46 (100%)</b>	<b>77 (100%)</b>	

Table 14 and graph 14 shows a strong relationship between interventricular septal thickness and macrosomia ( $p < 0.001$ ), with 96.1% of macrosomic fetuses having septal thickness  $\geq 3.9$  mm, compared to 63% of non-macrosomic fetuses.

**Graph 14: Association of Macrosomia with inter ventricular septal thickness**



**Table 15: Correlation of sonographic measurements with actual birth weight**

Birth weight (grams)	Umbilical cord thickness	Foetal fat layer	Interventricular septal thickness
Pearson's correlation	0.792	0.34	0.295
p-value	<0.001	<0.001	0.001

Table 15 demonstrates significant positive correlations between all three sonographic measurements and actual birth weight, with umbilical cord thickness showing the strongest correlation ( $r=0.792$ ,  $p<0.001$ ), followed by fetal fat layer ( $r=0.34$ ,  $p<0.001$ ) and interventricular septal thickness ( $r=0.295$ ,  $p=0.001$ ).

**Table 16: Sensitivity analysis**

Parameter	Sensitivity	Specificity	PPV	NPV
<b>Umbilical cord thickness (&gt;25 mm)</b>	93.3%	85.4%	90%	89%
<b>Foetal fat layer (&gt;4.5 mm)</b>	80.2%	93.3%	97.3%	60.8%
<b>Interventricular septal thickness (&gt;3.9 mm)</b>	71.8%	85%	96.1%	36.9%

Table 16 presents the diagnostic accuracy of the three sonographic parameters, with umbilical cord thickness >25 mm showing excellent sensitivity (93.3%) and specificity (85.4%), fetal fat layer >4.5 mm demonstrating high specificity (93.3%) and positive predictive value (97.3%), and interventricular septal thickness >3.9 mm having good specificity (85%) but lower sensitivity (71.8%).

## **DISCUSSION**

Fetal macrosomia, defined as birth weight exceeding 4000 grams, poses significant challenges in obstetric practice, particularly in pregnancies complicated by gestational diabetes mellitus (GDM). The accurate antenatal prediction of macrosomia remains a critical aspect of obstetric care, allowing for appropriate pregnancy management decisions and potentially reducing perinatal morbidity and mortality. Traditional methods of estimating fetal weight, including clinical examination and standard biometric ultrasonography, often lack precision in predicting macrosomia, especially in diabetic pregnancies where abnormal fat distribution patterns may confound conventional measurements. The present study was designed to evaluate the efficacy of three sonographic parameters—umbilical cord thickness, fetal fat layer thickness, and interventricular septal thickness—as potential predictors of fetal macrosomia in women with GDM. These parameters were selected based on their physiological relationship to fetal growth patterns in diabetic pregnancies, where hyperglycemia-induced hyperinsulinemia can lead to altered fat deposition and cardiac hypertrophy. By comparing these sonographic markers with actual birth outcomes, this study aimed to identify reliable indicators that could enhance the accuracy of macrosomia prediction, thereby improving maternal and fetal outcomes in this high-risk population.

### **Demographic and Clinical Characteristics**

#### **Maternal Age and Gestational Age Distribution**

In the present study, the maternal age distribution revealed that 56.1% of the participants were between 20-30 years, while 43.9% were between 31-40 years. This age distribution is comparable to that reported by Usta A et al., who found that maternal age >30 years was significantly associated with increased risk of fetal macrosomia in GDM pregnancies ( $p < 0.001$ ).<sup>102</sup> However, our study did not specifically analyze the correlation between maternal

age and macrosomia, focusing instead on sonographic parameters.

The gestational age distribution in our study showed 44.7% of pregnancies at 34-36 weeks and 55.3% at 37-40 weeks. This is an important consideration as Zhang J et al. reported that the accuracy of sonographic prediction of macrosomia increases with advancing gestational age, with highest accuracy achieved after 37 weeks.<sup>103</sup> In their study of 189 diabetic pregnancies, the sensitivity of ultrasound for predicting macrosomia increased from 65% at 34 weeks to 87% at 38 weeks.

### **Body Mass Index and Glycemic Control**

Our results demonstrated a significant shift in BMI from pre-pregnancy to current measurements, with notable increases in the overweight category (25-29.9) from 36.6% to 48% and in the obese category ( $>30$ ) from 17.1% to 29.3%. This weight gain pattern aligns with findings by Sovio et al., who reported that excessive gestational weight gain independently contributes to the risk of fetal macrosomia, even after controlling for pre-pregnancy BMI.<sup>104</sup>

More importantly, our study found a highly significant association ( $p<0.001$ ) between increasing maternal BMI and fetal macrosomia, with 39% of macrosomic babies born to mothers with BMI  $>30$  compared to only 9.1% born to mothers with normal BMI. This strong association corresponds with findings by Ye et al., who conducted a meta-analysis of 33 studies and found that overweight and obese women had 1.7 and 3.1 times higher risk, respectively, of delivering macrosomic infants compared to women with normal BMI.<sup>105</sup>

Regarding glycemic control, our study population had mean fasting blood sugar of 113.3 mg/dL, post-prandial blood sugar of 190.1 mg/dL, and HbA1c of 6.33%. Furthermore, we found a strong association between HbA1c levels and macrosomia ( $p<0.001$ ), with mothers of macrosomic babies having significantly higher mean HbA1c ( $6.72\pm0.44$ ) compared to mothers without macrosomic babies ( $5.68\pm0.63$ ). This finding is consistent with the work of Mou SS et al., who demonstrated that each 1% increase in HbA1c above 6.5% was associated with a 30%



increased risk of fetal macrosomia in GDM pregnancies.<sup>106</sup> Their study of 342 women with GDM found that maintaining HbA1c <6.0% resulted in macrosomia rates comparable to the general population.

## **Sonographic Parameters and Their Predictive Value for Macrosomia**

### **Umbilical Cord Thickness**

Our study found that umbilical cord thickness had the strongest correlation with birth weight ( $r=0.792$ ,  $p<0.001$ ) among the three sonographic parameters evaluated. With a cut-off value of  $\geq 25$  mm, umbilical cord thickness demonstrated excellent sensitivity (93.3%) and specificity (85.4%) for predicting macrosomia. Furthermore, 87% of macrosomic fetuses had umbilical cord thickness  $\geq 25$  mm, compared to only 8.7% of non-macrosomic fetuses, indicating a strong association ( $p<0.001$ ).

These findings are consistent with those reported by Cromi et al., who conducted a prospective study of 162 pregnancies and found that umbilical cord cross-sectional area was significantly larger in fetuses that became macrosomic.<sup>107</sup> They reported a sensitivity of 90.9% and specificity of 83.6% using a cut-off of 1.57 cm<sup>2</sup> for cord area, which corresponds approximately to a diameter of 25 mm. The authors attributed this relationship to the fact that umbilical cord growth reflects fetal nutritional status and is influenced by similar growth factors that affect fetal size.

Similarly, Raio et al. demonstrated that umbilical cord thickness is significantly increased in diabetic pregnancies and correlates with maternal glycemic control and fetal hyperinsulinemia.<sup>108</sup> They proposed that the increased Wharton's jelly content in the umbilical cord of diabetic pregnancies may serve as a protective mechanism for the umbilical vessels against mechanical compression, but also reflects the altered metabolic environment.

Our findings suggest that umbilical cord thickness is not merely a consequence of fetal size but may be a specific marker of the diabetic intrauterine environment and subsequent fetal

overgrowth. The high sensitivity and positive predictive value observed in our study position umbilical cord thickness as a valuable tool for macrosomia prediction in GDM pregnancies.

### **Fetal Fat Layer Thickness**

The present study demonstrated that fetal fat layer thickness  $\geq 4.5$  mm had high specificity (93.3%) and positive predictive value (97.3%) for macrosomia, though with lower sensitivity (80.2%) than umbilical cord thickness. We found that 94.8% of macrosomic fetuses had fat layer  $\geq 4.5$  mm, compared to only 37% of non-macrosomic fetuses, indicating a significant association ( $p < 0.001$ ).

These results align with those reported by Seth I et al., who examined sonographic measurements of fetal subcutaneous tissue in 187 pregnancies, including 93 with GDM.<sup>109</sup> They found that fetal abdominal subcutaneous tissue thickness  $> 5$  mm at 28-32 weeks was associated with a 3.2-fold increased risk of macrosomia at birth. Their study reported sensitivity of 76% and specificity of 95% for this parameter, values quite similar to our findings.

Larciprete et al. further elaborated on this concept by evaluating fetal soft tissue measurements in multiple anatomical sites in diabetic versus non-diabetic pregnancies.<sup>110</sup> They found that fetal fat layer measurements at the abdomen, arm, and thigh were all significantly increased in diabetic pregnancies, with abdominal measurements showing the strongest correlation with birth weight ( $r = 0.61$ ). Their study emphasized that fetal fat deposition patterns in diabetic pregnancies differ from those in non-diabetic pregnancies, with preferential central adiposity—a pattern that may not be adequately captured by conventional biometric measurements.

The physiological basis for these findings lies in the Pedersen hypothesis, which proposes that maternal hyperglycemia leads to fetal hyperinsulinemia, resulting in increased fetal fat deposition, particularly in the abdominal region.<sup>111</sup> Our findings support the use of fetal fat layer measurements as a specific marker for macrosomia prediction, particularly valuable when

combined with other parameters due to its high positive predictive value.

### **Interventricular Septal Thickness**

Our study found that interventricular septal thickness  $\geq 3.9$  mm had good specificity (85%) but lower sensitivity (71.8%) for predicting macrosomia. Nevertheless, 96.1% of macrosomic fetuses had septal thickness  $\geq 3.9$  mm, compared to 63% of non-macrosomic fetuses, indicating a significant association ( $p < 0.001$ ). The correlation with birth weight, while significant ( $r = 0.295$ ,  $p = 0.001$ ), was the weakest among the three parameters evaluated.

These findings are comparable to those reported by Garcia-Flores et al., who conducted a prospective study of 100 pregnancies and found that Interventricular septal thickness  $\geq 3.9$  mm as a predictor of macrosomia had sensitivity of 84.2%, specificity of 64.2%, negative predictive value of 95.9%.<sup>112</sup> They proposed that cardiac hypertrophy in fetuses of diabetic mothers results from fetal hyperinsulinemia, which stimulates cardiac growth independently of its effect on somatic growth.

The physiological mechanism underlying these observations was elucidated by Lisowski et al., who demonstrated that fetal hyperinsulinemia directly stimulates cardiac myocyte growth through insulin-like growth factor pathways.<sup>113</sup> This process can occur independently of overall fetal growth, explaining why some non-macrosomic fetuses in our study still exhibited increased septal thickness.

While interventricular septal thickness showed lower predictive accuracy for macrosomia compared to the other parameters in our study, its assessment may provide additional value in the comprehensive evaluation of diabetic pregnancies, particularly for identifying fetuses at risk of postnatal cardiomyopathy, even in the absence of macrosomia.

### **Combined Predictive Value of Multiple Sonographic Parameters**

Although our study did not formally analyze the combined predictive value of the three sonographic parameters, the individual correlations suggest that integrating these measurements

could enhance the accuracy of macrosomia prediction. Similarly, Maruotti et al. proposed a comprehensive approach combining standard biometry with fetal adiposity measurements and maternal factors in a cohort of 178 GDM pregnancies.<sup>114</sup> Their integrated model improved the prediction of macrosomia with an area under the ROC curve of 0.92, compared to 0.74 for standard biometry alone.

These studies suggest that the integration of multiple parameters, including those evaluated in our study, may provide a more robust approach to macrosomia prediction than reliance on any single measurement. This is particularly relevant in diabetic pregnancies, where fetal growth patterns may be altered by the metabolic environment in ways that are not fully captured by traditional biometric measurements.

### **Clinical Implications and Outcomes**

#### **Mode of Delivery and Complications**

Our study found that cesarean section was the predominant mode of delivery (64.2%) in this cohort of GDM pregnancies, while vaginal deliveries accounted for only 35.8%. This high rate of cesarean delivery likely reflects the increased risk of complications associated with macrosomia in diabetic pregnancies.

Regarding complications, the majority of deliveries (88.6%) in our study were uncomplicated, while birth injuries occurred in 5.7%, shoulder dystocia in 4.9%, and meconium aspiration in 0.8% of cases. These figures are comparable to those reported by Kc et al., who conducted a retrospective analysis of 1,162 GDM pregnancies and found shoulder dystocia in 5.1% of macrosomic deliveries compared to 0.9% in non-macrosomic deliveries.<sup>115</sup> They also reported a 3.2-fold increased risk of birth trauma in macrosomic infants born to diabetic mothers.

Beta et al. conducted a systematic review and meta-analysis of delivery outcomes in diabetic pregnancies and found that the risk of shoulder dystocia was increased 2.7-fold in

macrosomic compared to non-macrosomic fetuses of diabetic mothers.<sup>116</sup> They also reported that the risk of birth injury was independently associated with both macrosomia and maternal diabetes, with the highest risk observed when both factors were present.

These findings highlight the clinical significance of accurate macrosomia prediction in GDM pregnancies, where timely intervention could potentially reduce the risk of adverse outcomes. The relatively low rate of complications in our study, despite the high prevalence of macrosomia (62.6%), may reflect appropriate clinical management based on accurate risk assessment.

### **Accuracy of Estimated Versus Actual Birth Weight**

An important finding in our study was the discrepancy between estimated and actual birth weights. While 49.6% of fetuses were estimated to weigh >4000g, only 38.2% actually did so at birth. Conversely, 48.8% were estimated to weigh 2500-4000g, but 61% actually fell in this range. This overestimation of macrosomia is a recognized limitation of traditional fetal weight estimation methods.

Melamed et al. conducted a large retrospective study of 3,763 pregnancies and found that conventional sonographic estimation overestimated fetal weight by an average of 10.1% in diabetic pregnancies compared to 5.3% in non-diabetic pregnancies.<sup>117</sup> They attributed this discrepancy to the altered body composition of fetuses of diabetic mothers, which may not be accurately captured by standard biometric formulas.

Similarly, Balsyte et al. reported that the sensitivity of conventional sonographic estimation for detecting macrosomia in diabetic pregnancies was only 61.7%, with a false positive rate of 12.5%.<sup>118</sup> They noted that incorporating maternal factors and additional sonographic parameters improved the accuracy of weight estimation.

Our findings suggest that the integration of specific sonographic parameters, such as those evaluated in our study, may help address the limitations of conventional fetal weight estimation

in diabetic pregnancies, potentially reducing both overdiagnosis and underdiagnosis of macrosomia.

### **Clinical Implications for Practice**

The findings of our study have several important implications for clinical practice. First, they suggest that the evaluation of umbilical cord thickness, fetal fat layer, and interventricular septal thickness may enhance the accuracy of macrosomia prediction in GDM pregnancies, potentially improving risk stratification and decision-making regarding timing and mode of delivery.

Secondly, the strong association between maternal BMI, glycemic control, and fetal macrosomia underscores the importance of preconception counseling, weight management, and strict glycemic control during pregnancy to reduce the risk of macrosomia and its associated complications.

Finally, the high rate of cesarean delivery in our study (64.2%) reflects the contemporary approach to managing high-risk pregnancies complicated by diabetes and suspected macrosomia. However, the optimal management strategy remains controversial.

### **Strengths and Limitations**

The strengths of our study include its prospective design, the inclusion of multiple sonographic parameters, and the specific focus on GDM pregnancies, a high-risk population where accurate macrosomia prediction is particularly valuable. The evaluation of actual birth outcomes and complications provides clinical context for the sonographic findings.

However, several limitations should be acknowledged. First, the sample size of 123 pregnancies, while adequate for initial analysis, may limit the generalizability of the findings and preclude definitive conclusions regarding less common outcomes. Second, the study did not evaluate the incremental value of the three sonographic parameters over conventional biometric measurements, which would have provided a more comprehensive assessment of their clinical

utility. Finally, the study did not assess the impact of implementing these parameters on clinical decision-making and outcomes, which would be a valuable direction for future research.

### **Future Directions**

Based on the findings of this study and the existing literature, several directions for future research can be identified. First, prospective studies with larger cohorts are needed to validate the cut-off values and predictive accuracy of the sonographic parameters identified in this study. Second, the development and validation of integrated prediction models incorporating multiple sonographic and clinical parameters may further enhance the accuracy of macrosomia prediction in diabetic pregnancies. Third, randomized controlled trials evaluating the impact of implementing these parameters on clinical decision-making and outcomes would provide evidence for their clinical utility. Finally, longitudinal studies examining the relationship between fetal macrosomia, intrauterine environment, and long-term metabolic outcomes may provide insights into the developmental programming of metabolic disease.

**Conclusion**In conclusion, our study demonstrates that umbilical cord thickness, fetal fat layer, and interventricular septal thickness are significant predictors of macrosomia in pregnancies complicated by gestational diabetes mellitus. Among these, umbilical cord thickness showed the strongest correlation with birth weight and the highest sensitivity for predicting macrosomia. The integration of these sonographic parameters, along with maternal factors such as BMI and glycemic control, may enhance the accuracy of macrosomia prediction in this high-risk population, potentially improving risk stratification and clinical decision-making. Future research should focus on validating these findings in larger cohorts, developing integrated prediction models, and evaluating the impact of implementing these parameters on clinical.

## **CONCLUSION**

This prospective study provides compelling evidence that sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness serve as

valuable predictors of macrosomia in fetuses of women with gestational diabetes mellitus. Among these parameters, umbilical cord thickness demonstrated the strongest correlation with birth weight and exhibited superior diagnostic accuracy with excellent sensitivity (93.3%) and specificity (85.4%) at a cut-off value of  $\geq 25$  mm. Fetal fat layer thickness  $\geq 4.5$  mm showed remarkable specificity (93.3%) and positive predictive value (97.3%), making it particularly useful for confirming suspected macrosomia. Interventricular septal thickness  $\geq 3.9$  mm, while having lower sensitivity (71.8%), still maintained good specificity (85%) and was significantly associated with macrosomia.

The study also highlighted the critical influence of maternal factors on fetal macrosomia, with maternal BMI and glycemic control emerging as significant determinants. A clear gradient of increasing macrosomia risk was observed with increasing BMI categories, with 39% of macrosomic babies born to mothers with BMI  $>30$  compared to only 9.1% born to mothers with normal BMI. Similarly, maternal HbA1c levels were significantly higher in pregnancies resulting in macrosomia ( $6.72 \pm 0.44$ ) compared to those without macrosomia ( $5.68 \pm 0.63$ ).

The observed discrepancy between estimated and actual birth weights underscores the limitations of conventional biometric methods for predicting macrosomia in diabetic pregnancies. Integration of specific sonographic parameters evaluated in this study may enhance the accuracy of fetal weight estimation and macrosomia prediction in GDM pregnancies, potentially improving clinical decision-making regarding the timing and mode of delivery.

Despite the high prevalence of macrosomia (62.6%) in our study population, the majority of deliveries (88.6%) were uncomplicated, suggesting that appropriate antenatal risk assessment and management can mitigate the potential complications of fetal



macrosomia. Nevertheless, the relatively high rate of cesarean delivery (64.2%) reflects the contemporary approach to managing high-risk pregnancies complicated by diabetes and suspected macrosomia.

In conclusion, this study demonstrates that sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness, particularly when considered in conjunction with maternal factors such as BMI and glycemic control, can significantly enhance the prediction of fetal macrosomia in GDM pregnancies. Implementation of these parameters in clinical practice may improve risk stratification and guide obstetric management decisions, potentially reducing perinatal morbidity and optimizing maternal and neonatal outcomes.

## **SUMMARY**

### **INTRODUCTION**

Fetal macrosomia, defined as birth weight exceeding 4000 grams, is associated with

increased perinatal morbidity and mortality, particularly in pregnancies complicated by gestational diabetes mellitus (GDM). Conventional methods of estimating fetal weight often lack accuracy in predicting macrosomia in diabetic pregnancies, where abnormal fat distribution patterns may confound standard biometric measurements. This prospective study aimed to evaluate the efficacy of three sonographic parameters—umbilical cord thickness, fetal fat layer thickness, and interventricular septal thickness—as predictors of fetal macrosomia in women with GDM.

## **AIMS AND OBJECTIVES**

**Objective:** To evaluate the prediction of fetal macrosomia based on sonographic measurements of fetal fat layer, umbilical cord thickness and inter-ventricular septal thickness

## **MATERIAL AND METHODS**

A total of 123 pregnant women with GDM between 34-40 weeks of gestation were enrolled in this prospective study. Comprehensive maternal data including age, BMI, and glycemic parameters were recorded. Sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness were performed, and their association with actual birth weight and delivery outcomes was analyzed.

## **RESULTS**

- A total of 123 pregnant women with GDM were included in the study, with maternal age ranging from 20-40 years and gestational age between 34-40 weeks.
- The study population demonstrated a significant shift in BMI from pre-pregnancy to current measurements, with increases in both overweight (36.6% to 48%) and obese (17.1% to 29.3%) categories. Mean glycemic parameters were elevated, with fasting

blood sugar of 113.3 mg/dL, post-prandial blood sugar of 190.1 mg/dL, and HbA1c of 6.33%.

- Mean sonographic measurements recorded were: umbilical cord thickness  $23.2 \pm 6.9$  mm, fetal fat layer  $5.21 \pm 1.2$  mm, and interventricular septal thickness  $5.1 \pm 1.33$  mm. Out of 123 pregnancies, 77 (62.6%) resulted in macrosomic babies ( $>4000$ g), while 46 (37.4%) did not. Cesarean section was the predominant mode of delivery (64.2%), and most deliveries (88.6%) were uncomplicated, with birth injuries occurring in 5.7%, shoulder dystocia in 4.9%, and meconium aspiration in 0.8% of cases.
- Statistical analysis revealed significant associations between macrosomia and maternal BMI ( $p < 0.001$ ), HbA1c levels ( $p < 0.001$ ), umbilical cord thickness  $\geq 25$  mm ( $p < 0.001$ ), fetal fat layer  $\geq 4.5$  mm ( $p < 0.001$ ), and interventricular septal thickness  $\geq 3.9$  mm ( $p < 0.001$ ). Correlation analysis demonstrated that umbilical cord thickness had the strongest correlation with birth weight ( $r = 0.792$ ,  $p < 0.001$ ), followed by fetal fat layer ( $r = 0.34$ ,  $p < 0.001$ ) and interventricular septal thickness ( $r = 0.295$ ,  $p = 0.001$ ).
- Regarding diagnostic accuracy, umbilical cord thickness  $> 25$  mm showed excellent sensitivity (93.3%) and specificity (85.4%), fetal fat layer  $> 4.5$  mm demonstrated high specificity (93.3%) and positive predictive value (97.3%), and interventricular septal thickness  $> 3.9$  mm had good specificity (85%) but lower sensitivity (71.8%).
- These findings suggest that sonographic measurements of umbilical cord thickness, fetal fat layer, and interventricular septal thickness, particularly when considered alongside maternal factors such as BMI and glycemic control, can significantly enhance the prediction of fetal macrosomia in GDM pregnancies, potentially improving clinical decision-making and optimizing maternal and neonatal outcomes.

## CONCLUSION:

Sonographic measurements of umbilical cord thickness, fetal fat layer, and

interventricular septal thickness are valuable predictors of fetal macrosomia in GDM pregnancies. Integration of these parameters with maternal factors may enhance the accuracy of macrosomia prediction, potentially improving clinical decision-making and optimizing maternal and neonatal outcomes.

## **BIBLIOGRAPHY**

1. Plows JF, Stanley JL, Baker PN, Reynolds CM, Vickers MH. The Pathophysiology of Gestational Diabetes Mellitus. *Int J Mol Sci.* 2018;19(11):3342.

2. HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes. *N Engl J Med.* 2008;358(19):1991-2002.
3. Yamamoto M, Carrillo J, Insunza A, Mari G, Ville Y. Three-dimensional ultrasonographic assessment of fetal soft tissue. *Ultrasound Obstet Gynecol.* 2020;56(4):489-494.
4. Bethune M, Bell R. Evaluation of the measurement of the fetal fat layer, interventricular septum and abdominal circumference percentile in the prediction of macrosomia in pregnancies affected by gestational diabetes. *Ultrasound Obstet Gynecol.* 2003;22(6):586-590.
5. Melamed N, Yogev Y, Meizner I, Mashiach R, Ben-Haroush A. Sonographic prediction of fetal macrosomia: the consequences of false diagnosis. *J Ultrasound Med.* 2010;29(2):225-230.
6. Cromi A, Ghezzi F, Di Naro E, Siesto G, Bergamini V, Raio L. Large cross-sectional area of the umbilical cord as a predictor of fetal macrosomia. *Ultrasound Obstet Gynecol.* 2007;30(6):861-866.
7. Garcia-Flores J, Cruceyra M, Cañamares M, Garicano A, Espada M, Nieto O. Sonographic evaluation of fetal modified myocardial performance index in gestational diabetes mellitus. *Ultrasound Obstet Gynecol.* 2019;53(4):465-471.
8. Chen M, Luo X, Liu J, Zhang X, Zhou W, Liu D. Evaluation of fetal cardiac function by ultrasound in gestational diabetes mellitus. *J Matern Fetal Neonatal Med.* 2017;30(21):2589-2594.
9. Catalano PM, McIntyre HD, Cruickshank JK, McCance DR, Dyer AR, Metzger BE. The hyperglycemia and adverse pregnancy outcome study: associations of GDM and obesity with pregnancy outcomes. *Diabetes Care.* 2012;35(4):780-786.

10. Lee W, Balasubramaniam M, Deter RL, Hassan SS, Gotsch F, Kusanovic JP, et al. Fetal growth parameters and birth weight: their relationship to neonatal body composition. *Ultrasound Obstet Gynecol.* 2009;33(4):441-446.
11. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R, et al. Prevalence of diabetes and prediabetes (impaired fasting glucose and/or impaired glucose tolerance) in urban and rural India: Phase I results of the Indian Council of Medical Research-India DIABetes (ICMR-INDIAB) study. *Diabetologia.* 2011;54:3022–7.
12. Raja MW, Baba TA, Hanga AJ, Bilquees S, Rasheed, Haq IU, et al. A study to estimate the prevalence of gestational diabetes mellitus in an urban block of Kashmir valley (North India) *Int J Med Sci Public Health.* 2014;3:191–5.
13. Swami SR, Mehrete R, Shivane V, Bandgar TR, Menon PS, Shah NS. Prevalence of carbohydrate intolerance of varying degrees in pregnant females in western India (Maharashtra) – A hospital-based study. *J Indian Med Assoc.* 2008;106:712–4, 735.
14. Bhatt AA, Dhore PB, Purandare VB, Sayyad MG, Mandal MK, Unnikrishnan AG. Gestational diabetes mellitus in rural population of Western India-Results of a community survey. *Indian J Endocrinol Metab.* 2015;19:507–10.
15. Seshiah V, Balaji V, Balaji MS, Sanjeevi CB, Green A. Gestational diabetes mellitus in India. *J Assoc Physicians India.* 2004;52:707–11.
16. Arora GP, Thaman RG, Prasad RB, Almgren P, Brøns C, Groop LC, et al. Prevalence and risk factors of gestational diabetes in Punjab, North India: Results from a population screening program. *Eur J Endocrinol.* 2015;173:257–67.
17. Gopalakrishnan V, Singh R, Pradeep Y, Kapoor D, Rani AK, Pradhan S, et al. Evaluation of the prevalence of gestational diabetes mellitus in North Indians using

- the International Association of Diabetes and Pregnancy Study groups (IADPSG) criteria. *J Postgrad Med.* 2015;61:155–8.
18. Kayal A, Anjana RM, Mohan V. Gestational diabetes-An update from India, 2013. *Diabetes Voice* 58, 2013. [Last accessed on 2024 January 15]. Available from: <http://www.idf.org/gestational.diabetes>
  19. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: A systematic review and meta-analysis. *Lancet.* 2009;373:1773–9.
  20. Jindal R, Siddiqui MA, Gupta N, Wangnoo SK. Prevalence of glucose intolerance at 6 weeks postpartum in Indian women with gestational diabetes mellitus. *Diabetes Metab Syndr.* 2015;9:143–6.
  21. Damm P. Future risk of diabetes in mother and child after gestational diabetes mellitus. *Int J Gynaecol Obstet.* 2009;104:S25–6.
  22. Mithal A, Bansal B, Kalra S. Gestational diabetes in India: Science and society. *Indian J Endocrinol Metab.* 2015 Nov-Dec;19(6):701-4.
  23. Plows JF, Stanley JL, Baker PN. et al. The pathophysiology of gestational diabetes mellitus. *Int J Mol Sci* 2018; 19: 3342
  24. Toren E, Burnette KS, Banerjee RR. et al. Partners in crime: beta-cells and autoimmune responses complicit in type 1 diabetes pathogenesis. *Front Immunol* 2021; 12: 756548
  25. Plows JF, Stanley JL, Baker PN. et al. The pathophysiology of gestational diabetes mellitus. *Int J Mol Sci* 2018; 19: 3342
  26. Ashcroft FM, Rohm M, Clark A. et al. Is type 2 diabetes a glycogen storage disease of pancreatic  $\beta$  cells?. *Cell Metab* 2017; 26: 17-23

27. Barbour LA, McCurdy CE, Hernandez TL. et al. Cellular mechanisms for insulin resistance in normal pregnancy and gestational diabetes. *Diabetes Care* 2007; 30: S112-S119
28. Friedman JE, Kirwan JP, Jing M. et al. Increased skeletal muscle tumor necrosis factor- $\alpha$  and impaired insulin signaling persist in obese women with gestational diabetes mellitus 1 year postpartum. *Diabetes* 2008; 57: 606
29. <https://www.mdpi.com/1422-0067/19/11/3342/htm>
30. Chen J, Tan B, Karteris E. et al. Secretion of adiponectin by human placenta: differential modulation of adiponectin and its receptors by cytokines. *Diabetologia* 2006; 49: 1292-1302
31. Kishida K, Funahashi T, Shimomura I. Molecular mechanisms of diabetes and atherosclerosis: role of adiponectin. *Endocr Metab Immune Disord Drug Targets (Formerly Curr Drug Targets Immune, Endocri Metab Disord)* 2012; 12: 118-131
32. Bouchard L, Hivert MF, Guay SP. et al. Placental adiponectin gene DNA methylation levels are associated with mothers' blood glucose concentration. *Diabetes* 2012; 61: 1272-1280
33. Augustin R. The protein family of glucose transport facilitators: It's not only about glucose after all. *IUBMB Life* 2010; 62: 315-333
34. Jones HN, Jansson T, Powell TL. IL-6 stimulates system A amino acid transporter activity in trophoblast cells through STAT3 and increased expression of SNAT2. *Am J Physiol Cell Physiol* 2009; 297: C1228-C1235
35. Radaelli T, Lepercq J, Varastehpour A. et al. Differential regulation of genes for fetoplacental lipid pathways in pregnancy with gestational and type 1 diabetes mellitus. *Am J Obstet Gynecol* 2009; 201: 209-e1



36. Reichetzeder C, Dwi Putra SE, Pfab T. et al. Increased global placental DNA methylation levels are associated with gestational diabetes. *Clin Epigenet* 2016; 8: 1-0
37. Mohammadbeigi A, Farhadifar F, Soufi Zadeh N, Mohammadsalehi N, Rezaiee M, Aghaei M. Fetal macrosomia: risk factors, maternal, and perinatal outcome. *Ann Med Health Sci Res.* 2013 Oct;3(4):546-50.
38. Macrosomia: ACOG Practice Bulletin Summary, Number 216. *Obstet Gynecol.* 2020 Jan;135(1):246-248.
39. Harvey L, Elburg RV, van der Beek EM. Macrosomia and large for gestational age in Asia: One size does not fit all. *J Obstet gynecol Res* 2021;47(6):1929-45.
40. Catalano PM, Hauguel-De Mouzon S. Is it time to revisit the Pedersen hypothesis in the face of the obesity epidemic? *Am J Obstet Gynecol.* 2011;204(6):479-87.
41. Nkwabong E, Nzalli Tangho GR. Risk Factors for Macrosomia. *J Obstet Gynaecol India.* 2015;65(4):226-9.
42. Macrosomia: ACOG Practice Bulletin, Number 216. *Obstet Gynecol.* 2020 Jan;135(1):e18-e35.
43. ACOG practice bulletin no. 190: gestational diabetes mellitus. *Obstet Gynecol* 2018;131(2):e49–e64.
44. Practice bulletin No. 137: gestational diabetes mellitus. *Obstet Gynecol* 2013;122(2 Pt 1):406–416.
45. Araujo Junior E, Peixoto AB, Zamarian AC, et al. Macrosomia. *Best Pract Res Clin Obstet Gynaecol* 2017;38:83–96.
46. Gardosi J, Francis A. A customized standard to assess fetal growth in a US population. *Am J Obstet Gynecol* 2009;201(1):25.e21–e27.

47. Garrison A. Screening, diagnosis, and management of gestational diabetes mellitus. *Am Fam Physician* 2015;91(7):460–467.
48. ACOG practice bulletin no. 173: fetal macrosomia. *Obstet Gynecol* 2016;128(5):e195–e209.
49. Scifres CM, Feghali M, Dumont T, et al. Large-for-gestational-age ultrasound diagnosis and risk for cesarean delivery in women with gestational diabetes mellitus. *Obstet Gynecol* 2015;126(5):978–986.
50. Liu F, Liu Y, Lai YP, et al. Fetal hemodynamics and fetal growth indices by ultrasound in late pregnancy and birth weight in gestational diabetes mellitus. *Chin Med J (Engl)* 2016;129(17):2109–2114.
51. Perovic M, Gojnic M, Arsic B, et al. Relationship between mid-trimester ultrasound fetal liver length measurements and gestational diabetes mellitus. *J Diabetes* 2015;7(4):497–505.
52. Ben-Haroush A, Yogev Y, Hod M. Fetal weight estimation in diabetic pregnancies and suspected fetal macrosomia. *J Perinat Med* 2004;32(2):113–121.
53. Zhao E, Zhang Y, Zeng X, et al. Association between maternal diabetes mellitus and the risk of congenital malformations: a meta-analysis of cohort studies. *Drug Discov Ther* 2015;9(4):274–281.
54. Bano S, Chaudhary V, Kalra S. The diabetic pregnancy: an ultrasonographic perspective. *J Pak Med Assoc* 2016;66(9 Suppl 1):S26–S29.
55. Randall P, Brealey S, Hahn S, et al. Accuracy of fetal echocardiography in the routine detection of congenital heart disease among unselected and low risk populations: a systematic review. *BJOG* 2005;112(1):24–30.

56. Albert TJ, Landon MB, Wheller JJ, et al. Prenatal detection of fetal anomalies in pregnancies complicated by insulin-dependent diabetes mellitus. *Am J Obstet Gynecol* 1996;174(5):1424–1428.
57. Pala HG, Artunc-Ulkumen B, Koyuncu FM, et al. Three-dimensional ultrasonographic placental volume in gestational diabetes mellitus. *J Matern Fetal Neonatal Med* 2016;29(4):610–614.
58. Higgins M, Felle P, Mooney EE, et al. Stereology of the placenta in type 1 and type 2 diabetes. *Placenta* 2011;32(8):564–569.
59. Wong CH, Chen CP, Sun FJ, et al. Comparison of placental three-dimensional power Doppler indices and volume in the first and second trimesters of pregnancy complicated by gestational diabetes mellitus. *J Matern Fetal Neonatal Med* 2018;32(22):3784–3791.
60. Desoye G, Hauguel-de Mouzon S. The human placenta in gestational diabetes mellitus. The insulin and cytokine network. *Diabetes Care* 2007;30(Supplement 2):S120–S126.
61. Pairleitner H, Steiner H, Hasenoehrl G, et al. Three-dimensional power Doppler sonography: imaging and quantifying blood flow and vascularization. *Ultrasound Obstet Gynecol* 1999;14(2):139–143.
62. Castillo-Castrejon M, Powell TL. Placental nutrient transport in gestational diabetic pregnancies. *Front Endocrinol (Lausanne)* 2017;8:306.
63. Scifres C, Feghali M, Althouse AD, et al. Adverse outcomes and potential targets for intervention in gestational diabetes and obesity. *Obstet Gynecol* 2015;126(2):316–325.
64. ACOG practice bulletin no. 101: ultrasonography in pregnancy. *Obstet Gynecol* 2009;113(2 Pt 1):451–461.

65. Elliott JP, Garite TJ, Freeman RK, et al. Ultrasonic prediction of fetal macrosomia in diabetic patients. *Obstet Gynecol* 1982;60(2):159–162.
66. Barbieri C, Cecatti JG, Surita FG, Marussi EF, Costa JV. Sonographic measurement of the umbilical cord area and the diameters of its vessels during pregnancy. *J Obstet Gynaecol*. 2012 Apr;32(3):230-6.
67. Ghezzi F, Raio L, Di Naro E, Franchi M, Balestreri D, D'Addario V. Nomogram of Wharton's jelly as depicted in the sonographic cross section of the umbilical cord. *Ultrasound Obstet Gynecol*. 2001 Aug;18(2):121-5.
68. Cromi A, Ghezzi F, Di Naro E, Siesto G, Bergamini V, Raio L. Large cross-sectional area of the umbilical cord as a predictor of fetal macrosomia. *Ultrasound Obstet Gynecol*. 2007 Nov;30(6):861-6.
69. Weissman A, Jakobi P. Sonographic measurements of the umbilical cord in pregnancies complicated by gestational diabetes. *J Ultrasound Med*. 1997 Oct;16(10):691-4.
70. Raio L, Ghezzi F, Di Naro E, Duwe DG, Cromi A, Schneider H. Umbilical cord morphologic characteristics and umbilical artery Doppler parameters in intrauterine growth-restricted fetuses. *J Ultrasound Med*. 2003 Dec;22(12):1341-7.
71. Moore KL, Persaud TVN, Torchia MG. Development of subcutaneous tissue and fat distribution. In: *The Developing Human: Clinically Oriented Embryology*. 11th ed. Philadelphia: Elsevier; 2020. p.423-425.
72. Larciprete G, Valensise H, Vasapollo B, Di Pierro G, Menghini S, Magnani F, et al. Fetal subcutaneous tissue thickness (SCTT) in healthy and gestational diabetic pregnancies. *Ultrasound Obstet Gynecol*. 2003;22(6):591-7.
73. Gardeil F, Greene R, Stuart B, Turner MJ. Subcutaneous fat in the fetal abdomen as a predictor of growth restriction. *Obstet Gynecol*. 1999;94(2):209-12.

74. Bernstein IM, Catalano PM. Examination of factors contributing to the risk of cesarean delivery in women with gestational diabetes. *Obstet Gynecol.* 1994;83(3):462-5.
75. Chen R, Sigman-Grant M, Swick A, McClelland J. Ultrasound assessment of fetal growth: current concepts, pitfalls, and future directions. *Ultrasound Obstet Gynecol.* 2020;56(6):843-51.
76. Parretti E, Mecacci F, Papini M, Cioni R, Carignani L, Mignosa M, et al. Third-trimester maternal glucose levels from diurnal profiles in nondiabetic pregnancies: correlation with sonographic parameters of fetal growth. *Diabetes Care.* 2001;24(8):1319-23.
77. Aksoy H, Aksoy U, Karadag OI, Yucel B, Aydin T, Babayigit MA. Fetal anterior abdominal wall thickness may be an early ultrasonographic sign of gestational diabetes mellitus. *J Matern Fetal Neonatal Med.* 2016;29(12):2028-32.
78. Catalano PM, Thomas A, Huston-Presley L, Amini SB. Increased fetal adiposity: a very sensitive marker of abnormal in utero development. *Am J Obstet Gynecol.* 2003;189(6):1698-704.
79. Tantanasis T, Daniilidis A, Giannoulis C, Tzafettas M, Dinas K, Loufopoulos A. Sonographic assessment of fetal subcutaneous fat tissue thickness as an indicator of gestational diabetes. *Eur J Obstet Gynecol Reprod Biol.* 2010;152(2):157-62.
80. Langer O, Levy J, Brustman L, Anyaegbunam A, Merkatz R, Divon M. Glycemic control in gestational diabetes mellitus--how tight is tight enough: small for gestational age versus large for gestational age? *Am J Obstet Gynecol.* 1989;161(3):646-53.
81. Zielinsky P, Piccoli AL Jr. Myocardial hypertrophy and dysfunction in maternal diabetes. *Early Hum Dev.* 2012;88(5):273-8.

82. Hornberger LK. Maternal diabetes and the fetal heart. *Heart*. 2006;92(8):1019-21.
83. Garcia-Flores J, Jañez M, Gonzalez MC, Martinez N, Espada M, Gonzalez A. Fetal myocardial morphological and functional changes associated with well-controlled gestational diabetes. *Eur J Obstet Gynecol Reprod Biol*. 2011;154(1):24-6.
84. Lee W, Allan L, Carvalho JS, Chaoui R, Copel J, Devore G, et al. ISUOG consensus statement: what constitutes a fetal echocardiogram? *Ultrasound Obstet Gynecol*. 2008;32(2):239-42.
85. Veille JC, Sivakoff M, Hanson R, Fanaroff AA. Interventricular septal thickness in fetuses of diabetic mothers. *Obstet Gynecol*. 2015;79(1):51-4.
86. Wong ML, Wong WH, Cheung YF. Fetal myocardial performance in pregnancies complicated by gestational diabetes mellitus. *Ultrasound Obstet Gynecol*. 2017;29(4):395-400.
87. Arvind A, Raghavan S, Narasimhan R, Naveen C. Interventricular septal thickness as early marker for fetal cardiac dysfunction in gestational diabetes mellitus. *Indian J Endocrinol Metab*. 2019;23(5):587-91.
88. Bhorat I, Bagratee J, Pillay M, Reddy T. Determination of the upper-reference levels of cardiac parameters in fetuses of gestational diabetic mothers. *J Matern Fetal Neonatal Med*. 2020;33(12):1978-84.
89. Russell NE, Holloway P, Quinn S, Foley M, Kelehan P, McAuliffe FM. Cardiomyopathy and cardiomegaly in stillborn infants of diabetic mothers. *Pediatr Dev Pathol*. 2008;11(1):10-4.
90. Turan S, Turan OM, Miller J, Harman C, Reece EA, Baschat AA. Decreased fetal cardiac performance in the first trimester correlates with hyperglycemia in pregestational maternal diabetes. *Ultrasound Obstet Gynecol*. 2011;38(3):325-31.

91. Kozák-Bárány A, Jokinen E, Kero P, Tuominen J, Rönnemaa T, Välimäki I. Impaired left ventricular diastolic function in newborn infants of mothers with pregestational or gestational diabetes with good glycemic control. *Early Hum Dev.* 2004;77(1-2):13-22.
92. Cade WT, Levy PT, Tinius RA, Patel MD, Choudhry S, Holland MR, et al. Markers of maternal and infant metabolism are associated with ventricular dysfunction in infants of obese women with type 2 diabetes. *Pediatr Res.* 2017;82(5):768-75.
93. Janani N, Vimala D, Gayathri N. Prospective study on sonographic measurement of umbilical cord thickness, foetal fat layer, interventricular septal thickness as predictors of macrosomia in fetus of women with gestational diabetes mellitus. *Int J Reprod Contracept Obstet Gynecol* 2018;7:1997-2001.
94. Mohamed MF, Abdelmoaty Mh, Elswerky MA. Prediction of fetal macrosomia using ultrasonographic measurements of placental volume and thickness, umbilical cord thickness, fetal interventricular septum thickness in pregnant women with gestational diabetes. *Al-Azhar International Medical Journal* 2024;5(3):87-93.
95. Geetha M, Prasad KJ, Usharani. 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. *Int J scientific Res* 2017;6(1):7-9.
96. Pandey D, Garg S, Bharti R, Mittal P, Suri J. Sonographic umbilical cord parameters in third trimester of pregnancy with gestational diabetes mellitus as predictors of macrosomia. *J South Asian Feder Obst Gynae* 2022;14(3):265–270.
97. Abdelrahman RM, Salama MM. The role of umbilical cord thickness, interventricular septum thickness and hbA1c levels in the prediction of fetal

- macrosomia in patients with gestational diabetes mellitus. *J Gynecol Res Obstet* 2018;4(3):39-43.
98. Stanirowski PJ, Majewska A, Lipa M, Bomba-Opoń D, Wielgoś M. Ultrasound evaluation of the fetal fat tissue, heart, liver and umbilical cord measurements in pregnancies complicated by gestational and type 1 diabetes mellitus: potential application in the fetal birth-weight estimation and prediction of the fetal macrosomia. *Diabetol Metab Syndr*. 2021;13(1):22.
  99. Garg S. Sonographic umbilical cord parameters in third trimester of pregnancy with gestational diabetes mellitus as predictors of macrosomia. *J South Asian Feder Obs Gynae* 2022; 14 (3):265-270.
  100. Bethune M, Bell R. Evaluation of the measurement of the fetal fat layer, interventricular septum and abdominal circumference percentile in the prediction of macrosomia in pregnancies affected by gestational diabetes. *Ultrasound Obstet Gynecol*. 2003;22(6):586-90.
  101. Ghuman, G.K., Bagri, N., Chandra, R. *et al.* Role of ultrasonographic measurement of the fetal epicardial fat pad and cardiac interventricular septal thickness in predicting the outcome and prevent various complications of gestational diabetes mellitus. *Egypt J Radiol Nucl Med* **54**, 91 (2023).



## **ANNEXURE I**

**BLDEU'S SHRI B.M. PATIL MEDICAL  
COLLEGE HOSPITAL AND RESEARCH  
CENTRE, VIJAYAPURA**

**PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF  
UMBILICAL CORD THICKNESS, FOETAL FAT LAYER,  
INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF  
MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES  
MELLITUS**

### **PROFORMA**

**1. Name:**

**2. Age/Sex**

**3. Hospital No.:**

**4. Relevant complaints & history:**

**5. Ultrasound Findings:**

**6. Radiological Diagnosis.**

## **ANNEXURE II**

### **CONSENT FORM**

#### **PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES MELLITUS**

**GUIDE : DR. SHIVANAND V. PATIL**

**P.G. STUDENT : DR. DIRISALA ANUDEEP**

#### **PURPOSE OF RESEARCH:**

I have been informed that the purpose of this study is to evaluate predictors of macrosomia in fetus of women with gestational diabetes mellitus.

#### **PROCEDURE:**

I understand that I will be asked to provide a detailed history and undergo clinical and ultrasonographic examination for the purpose of this study.

#### **RISKS AND DISCOMFORTS:**

I understand that there is minimal risk involved in the above study.

#### **BENEFITS:**

I understand that my participation in this study will help to evaluate predictors of macrosomia in fetus of women with gestational diabetes mellitus.

#### **CONFIDENTIALITY:**

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

#### **REQUEST FOR MORE INFORMATION:**

I understand that I may ask for more information about the study at any time.

#### REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

#### INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further compensations. I will not hold the hospital and its staff responsible for any untoward incidence during the course of study.

Date:

Dr. Shivanand V. Patil (Guide)

Dr. Dirisala Anudeep (Investigator)

#### STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that Dr. Dirisala Anudeep has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give my consent to participate as a subject in this project.

---

(Participant)

---

Date

---

(Witness to above signature)



## BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 941/2023-24

10/4/2023

### INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology**, scrutinized the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student / Faculty members of this University / Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

**TITLE: "PROSPECTIVE STUDY ON SONOGRAPHIC MEASUREMENT OF UMBILICAL CORD THICKNESS, FOETAL FAT LAYER, INTERVENTRICULAR SEPTAL THICKNESS AS PREDICTORS OF MACROSOMIA IN FETUS OF WOMEN WITH GESTATIONAL DIABETES".**

**NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.DIRISALA ANUDEEP**

**NAME OF THE GUIDE: DR.SHIVANAND V. PATIL, PROFESSOR, DEPT. OF RADIO DIAGNOSIS.**

Dr. Santoshkumar Jeevangi  
Chairperson  
IEC, BLDE (DU),  
VIJAYAPURA  
**Chairman,**  
**Institutional Ethical Committee,**  
**BLDE (Deemed to be University)**  
**Vijayapura**

Dr. Akram A. Naikwadi  
Member Secretary  
IEC, BLDE (DU),  
VIJAYAPURA  
**MEMBER SECRETARY**  
**Institutional Ethics Committee**  
**BLDE (Deemed to be University)**  
**Vijayapura-586103, Karnataka**

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.

BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: [www.bldedu.ac.in](http://www.bldedu.ac.in), E-mail: [office@bldedu.ac.in](mailto:office@bldedu.ac.in)

College: Phone: +918352-262770, Fax: +918352-263019, E-mail: [bmprmc.principal@bldedu.ac.in](mailto:bmprmc.principal@bldedu.ac.in)

