

**A CROSS SECTIONAL STUDY TO EVALUATE
THE IMPACT OF PLACENTAL LOCATION ON
MATERNAL AND FETAL OUTCOME**

By

Dr. NAVEENA ALAKONDA

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Under the guidance of

Dr.NEELAMMA.PATIL

PROFESSOR

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY

BLDE (Deemed to be University)

SHRI B.M.PATIL MEDICAL COLLEGE

HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

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**“A CROSS SECTIONAL STUDY TO EVALUATE THE IMPACT OF PLACENTAL
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ABBREVIATIONS

PE	Pre-Eclampsia
PROM	Pre Mature Rupture of Membranes
HPR	Histo-Pathological Report
Rh	Rhesus
USG	Ultrasonogram
IUGR	Intra Uterine Growth Restriction
IUD	Intra Uterine Death
TVS	Trans Vaginal sonogram
Dpf	Days post fertilization
TE	Trophoectoderm
SCT	Syncytiotrophoblast
VCT	Villous Cytotrophoblast
HLA	Human Leukocyte Antigen
FcRn	Fc Receptor
IgG	Immunoglobulin G
NK	Natural Killer
ATP	Adenosine Triphosphate
Hcg	Human Chorionic Gonadotropin
GLUT	Glucose transporter
HCS	Human Chorionic somatomammotropin
HPL	Human Placental Lactogen
HGH	Placental Growth Hormone
EVT	Extra Villous Trophoblast
UNK	Uterine Natural killer
GOS	Great obstetric Syndrome
FGR	Foetal Growth Restriction
APGAR	Appearance Pulse Grimace Activity Respiration
NICU	Neonatal Intensive Care Unit
YRS	Years

-VE	Negative
+VE	Positive
GHTN	Gestational Hypertension
HELLP	Hemolysis Elevated liver Enzymes Low Platelet count
LSCS	Lower Segment Caeserian Section

TABLE OF CONTENTS

S.No.	TOPIC	PAGE NO.
1	INTRODUCTION	19
2.	OBJECTIVES	21
3.	BACKGROUD	20
4.	REVIEW OF LITERATURE	23
5.	MATERIALS AND METHODS	73
6.	RESULTS	76
7.	DISCUSSION	158
8.	LIMITATIONS	198
9.	CONCLUSIONS	199
10.	SUMMARY	201
11.	RECOMMENDATIONS	204
12.	BIBILOGRAPHY	205
13.	ANNEXURES	
	Ethical Clearance	224
	Consent	225
	Proforma	227
	Masterchart	229

LIST OF FIGURES

S. No.	DESCRIPTION	P. No.
Figure 1	EARLY STAGES OF HUMAN PLACENTAL DEVELOPMENT	29
Figure 2	MATERNAL -FOETAL INTERFACE AND TROPHOBLAST SUBTYPES	33
Figure 3	BAR CHART OF DISTRIBUTION OF AGE	79
Figure 4	BAR CHART OF DISTRIBUTION OF MEAN AGE	79
Figure 5	BAR CHART OF DISTRIBUTION OF OBSTETRIC HISTORY	80
Figure 6	PIE CHART OF DISTRIBUTION OF PLACENTAL LOCATION	80
Figure 7	BAR CHART OF DISTRIBUTION OF MODE OF DELIVERY	81
Figure 8	BAR CHART OF DISTRIBUTION OF HPR OF MATERNAL SURFACE OF PLACENTA	81
Figure 9	BAR CHART OF DISTRIBUTION OF HPR OF DECIDUAL ARTERIES OF PLACENTA	82
Figure 10	BAR CHART OF DISTRIBUTION OF HPR OF FOETAL SURFACE OF PLACENTA	83

LIST OF TABLES

S.No.	Description	P. No.
Table 1	Association Between Fundal Placenta and Mild PE	85
Table 2	Association Between Right Lateral Placenta and Mild PE	85
Table 3	Association Between Left Lateral Placenta and Mild PE	86
Table 4	Association Between Posterior Placenta and Mild PE	86
Table 5	Association Between Anterior Placenta and Mild PE	87
Table 6	Association Between Placenta Previa and Mild PE	87
Table 7	Association Between Fundal Placenta and Severe PE	88
Table 8	Association Between Right Lateral Placenta and Severe PE	88
Table 9	Association Between Left Lateral Placenta and Severe PE	89
Table 10	Association Between Posterior Placenta and Severe PE	89
Table 11	Association Between Anterior Placenta and Severe PE	90
Table 12	Association Between Placenta Previa and Severe PE	90
Table 13	Association Between Fundal Placenta and Eclampsia	91
Table 14	Association Between Right Lateral Placenta and Eclampsia	91
Table 15	Association Between Left Lateral Placenta and Eclampsia	92
Table 16	Association Between Posterior Placenta and Eclampsia	92
Table 17	Association Between Anterior Placenta and Eclampsia	93
Table 18	Association Between Placenta Previa and Eclampsia	93
Table 19	Association Between Fundal Placenta and Abruptio	94
Table 20	Association Between Right Lateral Placenta and Abruptio	94
Table 21	Association Between Left Lateral Placenta and Abruptio	95
Table 22	Association Between Posterior Placenta and Abruptio	95
Table 23	Association Between Anterior Placenta and Abruptio	96
Table 24	Association Between Placenta Previa and Abruptio	96
Table 25	Association Between Fundal Placenta and FGR	97
Table 26	Association Between Right Lateral Placenta and FGR	98
Table 27	Association Between Left Lateral Placenta and FGR	98
Table 28	Association Between Posterior Placenta and FGR	99

Table 29	Association Between Anterior Placenta and FGR	99
Table 30	Association Between Placenta Previa and FGR	100
Table 31	Association Between Fundal Placenta and Oligohydramnios	100
Table 32	Association Between Right Lateral Placenta and Oligohydramnios	101
Table 33	Association Between Left Lateral Placenta and Oligohydramnios	101
Table 34	Association Between Posterior Placenta and Oligohydramnios	102
Table 35	Association Between Anterior Placenta and Oligohydramnios	102
Table 36	Association Between Placenta Previa and Oligohydramnios	103
Table 37	Association Between Fundal Placenta and Polyhydramnios	103
Table 38	Association Between Right Lateral Placenta and Polyhydramnios	104
Table 39	Association Between Left Lateral Placenta and Polyhydramnios	104
Table 40	Association Between Posterior Placenta and Polyhydramnios	105
Table 41	Association Between Anterior Placenta and Polyhydramnios	105
Table 42	Association Between Placenta Previa and Polyhydramnios	106
Table 43	Association Between Fundal Placenta and Preterm	106
Table 44	Association Between Right Lateral Placenta and Preterm	107
Table 45	Association Between Left Lateral Placenta and Preterm	107
Table 46	Association Between Posterior Placenta and Preterm	108
Table 47	Association Between Anterior Placenta and Preterm	108
Table 48	Association Between Placenta Previa and Preterm	109
Table 49	Association Between Fundal Placenta and PROM	109
Table 50	Association Between Right Lateral Placenta and PROM	110
Table 51	Association Between Left Lateral Placenta and PROM	110
Table 52	Association Between Posterior Placenta and PROM	111
Table 53	Association Between Anterior Placenta and PROM	111
Table 54	Association Between Placenta Previa and PROM	112
Table 55	Association Between Fundal Placenta and Vaginal Delivery	112
Table 56	Association Between Right Lateral Placenta and Vaginal Delivery	113
Table 57	Association Between Left Lateral Placenta and Vaginal Delivery	113
Table 58	Association Between Posterior Placenta and Vaginal Delivery	114
Table 59	Association Between Anterior Placenta and Vaginal Delivery	114

Table 60	Association Between Placenta Previa and Vaginal Delivery	115
Table 61	Association Between Fundal Placenta and LSCS	115
Table 62	Association Between Right Lateral Placenta and LSCS	116
Table 63	Association Between Left Lateral Placenta and LSCS	116
Table 64	Association Between Posterior Placenta and LSCS	117
Table 65	Association Between Anterior Placenta and LSCS	117
Table 66	Association Between Placenta Previa and LSCS	118
Table 67	Association Between Fundal Placenta and Instrumental Delivery	118
Table 68	Association Between Right Lateral Placenta and Instrumental Delivery	119
Table 69	Association Between Left Lateral Placenta and Instrumental Delivery	119
Table 70	Association Between Posterior Placenta and Instrumental Delivery	120
Table 71	Association Between Anterior Placenta and Instrumental Delivery	120
Table 72	Association Between Placenta Previa and Instrumental Delivery	121
Table 73	Association Between Fundal Placenta and NICU	121
Table 74	Association Between Right Lateral Placenta and NICU	122
Table 75	Association Between Left Lateral Placenta and NICU	122
Table 76	Association Between Posterior Placenta and NICU	123
Table 77	Association Between Anterior Placenta and NICU	123
Table 78	Association Between Placenta Previa and NICU	124
Table 79	Association Between Fundal Placenta and Mother-side	124
Table 80	Association Between Right Lateral Placenta and Mother-side	125
Table 81	Association Between Left Lateral Placenta and Mother-side	125
Table 82	Association Between Posterior Placenta and Mother-side	126
Table 83	Association Between Anterior Placenta and Mother-side	126
Table 84	Association Between Placenta Previa and Mother-side	127
Table 85	Association Between Fundal Placenta and Perinatal Death	127
Table 86	Association Between Right Lateral Placenta and Perinatal Death	128
Table 87	Association Between Left Lateral Placenta and Perinatal Death	128
Table 88	Association Between Posterior Placenta and Perinatal Death	129

Table 89	Association Between Anterior Placenta and Perinatal Death	129
Table 90	Association Between Placenta Previa and Perinatal Death	130
Table 91	Association Between Fundal Placenta and Fresh Still Birth	130
Table 92	Association Between Right Lateral Placenta and Fresh Still Birth	131
Table 93	Association Between Left Lateral Placenta and Fresh Still Birth	131
Table 94	Association Between Posterior Placenta and Fresh Still Birth	132
Table 95	Association Between Anterior Placenta and Fresh Still Birth	132
Table 96	Association Between Placenta Previa and Fresh Still Birth	133
Table 97	Association Between Fundal Placenta and Macerated Still Birth	133
Table 98	Association Between Right Lateral Placenta and Macerated Still Birth	134
Table 99	Association Between Left Lateral Placenta and Macerated Still Birth	134
Table 100	Association Between Posterior Placenta and Macerated Still Birth	135
Table 101	Association Between Anterior Placenta and Macerated Still Birth	135
Table 102	Association Between Placenta Previa and Macerated Still Birth	136
Table 103	Association Between Fundal Placenta and HPR of Maternal Surface of Placenta	135
Table 104	Association Between Right Lateral Placenta and HPR of Maternal Surface of Placenta	137
Table 105	Association Between Left Lateral Placenta and HPR of Maternal Surface of Placenta	138
Table 106	Association Between Posterior Placenta and HPR of Maternal Surface of Placenta	139
Table 107	Association Between Anterior Placenta and HPR of Maternal Surface of Placenta	140
Table 108	Association Between Placenta Previa and HPR of Maternal Surface of Placenta	142
Table 109	Association Between Fundal Placenta and HPR of Decidual Arteries of Placenta	143
Table 110	Association Between Right Lateral Placenta and HPR of Decidual Arteries of Placenta	144
Table 111	Association Between Left Lateral Placenta and HPR of Decidual Arteries of Placenta	145
Table 112	Association Between Posterior Placenta and HPR of Decidual	146

	Arteries of Placenta	
Table 113	Association Between Anterior Placenta and HPR of Decidual Arteries of Placenta	147
Table 114	Association Between Placenta Previa and HPR of Decidual Arteries of Placenta	148
Table 115	Association Between Fundal Placenta and HPR of Foetal Surface of Placenta	149
Table 116	Association Between Right Lateral Placenta and HPR of Foetal Surface of Placenta	150
Table 117	Association Between Left Lateral Placenta and HPR of Foetal Surface of Placenta	151
Table 118	Association Between Posterior Placenta and HPR of Foetal Surface of Placenta	152
Table 119	Association Between Anterior Placenta and HPR of Foetal Surface of Placenta	153
Table 120	Association Between Placenta Previa and HPR of Foetal Surface of Placenta	154

ABSTRACT

BACKGROUND

The human placenta is the critical organ responsible for the facilitation of nutrient uptake, waste elimination, and gas exchange between mother and foetus. The placenta is also a vital source of hormone production such as progesterone and human chorionic gonadotropin that maintain the pregnancy.

AIMS AND OBJECTIVES

To examine the relation between placental location and foetal outcomes. Also, to correlate with histopathology of the placenta.

MATERIALS AND METHODS

The present study is a Cross Sectional study. This Study was conducted from January 2021-April 2022 at Department of OBSTETRICS AND GYNAECOLOGY in B.L.D.E (DEEMED TO BE UNIVERSITY) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura. Total 1301 patients were included in this study.

RESULT

We have seen that there was a positive and significant association between Fundal implantation and Severe PE and PROM. There was positive and significant association between left lateral Implantation and Severe PE. The Histopathological changes seen in HPR of foetal surface of placenta also had positive and significant association with Fundal location of placenta.

CONCLUSION

This study proves that Fundal, Left Lateral placentation has abnormal results, such patients can be considered as high risk and can be given meticulous antenatal care, depending on placental location at 18-20 weeks USG.

KEY WORDS

Location of Placenta, Blood group and Rh Type, HPR of Placenta

INTRODUCTION

Placenta, which is a vital link between mother and the foetus for metabolic exchange, endocrine, and other body functions, is critical for maternal, neonatal wellbeing. The site of implantation that decides the location of placenta is likely to be the essential determinant of placental blood flow and therefore, pregnancy outcome ⁽¹⁾.

The placenta is the vital organ which connects the foetus to the uterine wall ⁽²⁾. In placenta, there are two circulations- maternal and foetal. Hence abnormalities in the placental implantation may affect the blood supply of foetus leading to adverse maternal and foetal outcomes such as gestational hypertension, pre-eclampsia, gestational diabetes, malpresentation, malposition, preterm birth, small for gestational age, IUGR, low birth weight, IUD and stillbirth ⁽³⁾.

Anterior placental implantation is associated with an increased risk of pregnancy-induced hypertension, gestational diabetes mellitus, placental abruption, IUGR and IUD. ⁽⁴⁾

Posterior placental location is attributed to foetal distress, increased caesarian rates, the incidence of meconium-stained liquor and increase in foetal heart rate deceleration. ⁽⁵⁾

Uterus receives blood supply from a uterine-arteries- branches of the iliac artery, supplying the corresponding side of the uterus ⁽⁶⁾. Although anastomoses between two arteries exist, there is no proof for the same.

In centrally located placenta, both uterine arteries demonstrated similar resistance and the uteroplacental blood flow needs are met by equal distribution of both uterine arteries. But when the placenta is laterally located, the uteroplacental blood flow needs are met with primarily by one uterine artery via collateral circulation ⁽⁷⁾. This degree of collateral circulation is not the same in all patients and deficient in this may lead to the development of placental insufficiency.

Non-invasive abnormal Doppler waveforms of uterine arteries in the second trimester would suggest defective uterine perfusion due to placental implantation when one uterine artery is the dominant supply of intervillous flow. ⁽⁸⁾

USG of the placenta is primarily directed toward determining the location of the placenta and identifying its abnormalities in the latter weeks of pregnancy. However, the advent of high-resolution transvaginal ultrasound (TVS) has revolutionized the understanding of placental studies, and it is believed that placental evaluation in early pregnancy could be useful in identifying the risks for subsequent disorders. ⁽⁹⁾

So, in this study, we want to study the co-relation between placental location and pregnancy outcomes, depending on placental location at 18-20 weeks USG.

OBJECTIVE FOR THE STUDY

OBJECTIVE FOR THE STUDY:

1. To examine the relation between placental location with fetal and maternal outcomes.
2. Also, correlate with histopathology of the placenta.

OUTCOME:

Primary: To see the co-relation between placental location and pregnancy outcomes

Secondary:

- 1.Incidence of different placental location.
- 2.To correlate placental location and HPR.

BACKGROUND

The human placenta is a vital organ that facilitates the absorption of nutrients, the removal of waste, and the exchange of gases between the mother and foetus ⁽¹⁰⁾. Progesterone plus human chorionic gonadotropin, among others, are produced by the placenta and are essential for maintaining pregnancy ⁽¹⁰⁾. Accordingly, a variety of negative foetal outcomes can result from placental malfunction. Moreover, because the placenta reflects the metabolic milieu of both mother and fetus, it serves as a valuable tool for studying the metabolic perturbations that may take place during pregnancy, such as diabetes mellitus.

Ultrasound imaging has become an integral component of routine prenatal medical care for most pregnant women. During an obstetrical ultrasound, evaluation of the fetus is chief priority but often, the other components (placenta, umbilical cord, and amniotic fluid) which represent an integral part of gestation, are arguably not given the attention they deserve ⁽¹¹⁾.

Both the ACOG and the American Institute of Ultrasound in Medicine recommended that the standard obstetric sonogram in the second and/or third trimester should include the evaluation of placental position and morphology ^(12,13).

While abnormalities in amniotic fluid volume and umbilical cord Doppler velocimetry immediately alert the sonographer (possible implications on the continuation of physiological pregnancy), sonographic assessment of placental location, after exclusion of previa or marginal insertion (necessary to assess the option of vaginal delivery), is often limited to a mere notional description without any link to possible implications on pregnancy and childbirth ^(14,15)

REVIEW OF LITERATURE

DEVELOPMENT OF PLACENTA

Each pregnancy begins with the formation of the zygote, and as a result, the placenta and accompanying extraembryonic membranes share the same genetic makeup as the foetus ⁽¹⁷⁾. The trophoctoderm that makes up the blastocyst's wall and the underneath extra - embryonic mesoderm are the two main tissue origins ⁽¹⁷⁾. The placenta's epithelium is formed by the differentiation of the trophoctoderm into trophoblast, which also give rise to a subset of penetrating extra-villous trophoblast cells. The stromal core of the placenta is made up of extraembryonic mesoderm, which gives rise to the fibroblasts, vascular system, and resident macrophage population ⁽¹⁷⁾.

The mature placenta, is a fairly discoid organ with a diameter of approximately of 22 cm, with central thickness of 2.5 cm, weighing approximately 500 grams ⁽¹⁸⁻¹⁹⁾. It has two surfaces: the basal plate, which encroaches the mother's endometrium, and the chorionic plate to which the umbilical cord is connected, faces the foetus.

The placenta is the largest foetal organ made up of its parenchyma, chorion, amnion, and umbilical cord. The foetal structures form from the zygote and therefore separate the foetus from the endometrium. The foetal tissues form from the chorionic sac - which includes the amnion, chorion, yolk sac, and allantois. These tissues get delivered after birth. The maternal part comes from the endometrium and is called the decidua. There are three parts to the decidua - the decidua basalis (deep at the implantation site), the decidua capsularis (covers the implantation site), and the decidua parietalis (everything else) ⁽²⁰⁾.

PRELACUNAR PHASE

After fertilization of the sperm and ovum, four cell division leads to a morula (16 cells). Around the fourth day after fertilization, the morula enters the uterus as a blastocyst. The blastocyst divides into trophoblast and embryoblast. The trophoblast (TE), the pre-implantation embryo's outer layer, originates at around five days after fertilisation and is the precursor of the placenta (dpf). The blastocyst, or pre-implantation embryo, is now divided into the inner cell mass (ICM) and the TE lineages. The trophoblast (TE) adheres to the surface epithelium of the uterine mucosa as the polar TE (the part of the TE that is continuous with the underlying ICM) at 6-7dpf (Fig. 1A). Following which, TE fuses to form a primary syncytium.

LACUNAR PHASE

After implantation, the primary syncytium quickly invades via surface epithelium into the underlying endometrium, which is transformed during pregnancy into a specialised tissue known as decidua ⁽²¹⁾ (Fig. 1B). By the time of the first missed menstrual period (~14 dpf), the blastocyst is completely embedded in the decidua and is covered by the surface epithelium ⁽²²⁾ (Fig. 1C). Fluid-filled spaces (lacunae) then appear within the syncytial mass that enlarge and merge, partitioning it into a system of trabeculae. The syncytium also erodes into decidual glands, allowing secretions to bathe the syncytial mass ⁽²²⁾.

VILLOUS PHASE

As the pregnancy progresses, the villous cytotrophoblast slowly disappears from the chorionic villi. Additionally, the villi structure develops to reduce the distance between the maternal blood and foetal vessels — this change benefits maternal-foetal-exchanges. By the end of a pregnancy, five types of villi comprise the placenta: mesenchymal villi, immature intermediate villi, stem villi, mature intermediate villi, and terminal villi ⁽²³⁾.

- Mesenchymal villi play a significant role early in the first trimester as the most primitive type of villi. The villi are mostly filled with mesenchymal cells and with poorly developed villi. Mesenchymal cells will later differentiate into a variety of other cells including the following: endothelial cells, blood cells, macrophages, myofibroblasts, smooth muscle cells, and fibroblasts.
- Immature intermediate villi are prevalent in the mid-first trimester. These reticular structures contain fluid as well as macrophages called Hofbauer cells. Between the stroma, small arterioles and venules start to develop.
- Stem villi appear condensed with collagen fibers during the mid-first trimester. Additionally, there are few fibroblasts and macrophages as well as muscularized arteries and veins in the villi.
- Mature intermediate villi, in mid-gestation, are bundles of connective tissue with numerous peripheral capillaries as well as some small terminal arterioles and collecting venules.
- Terminal villi mainly function during the late second trimester through the early third trimester. These villi have no stroma and predominantly contain sinusoidal capillaries

⁽²⁴⁾⁽²⁵⁾⁽²⁶⁾⁽²⁷⁾.

These villi help communication between the chorionic plate and decidua. Most of the villi float freely in the intervillous space while other villi attach to the decidua as structural stability for the placenta.

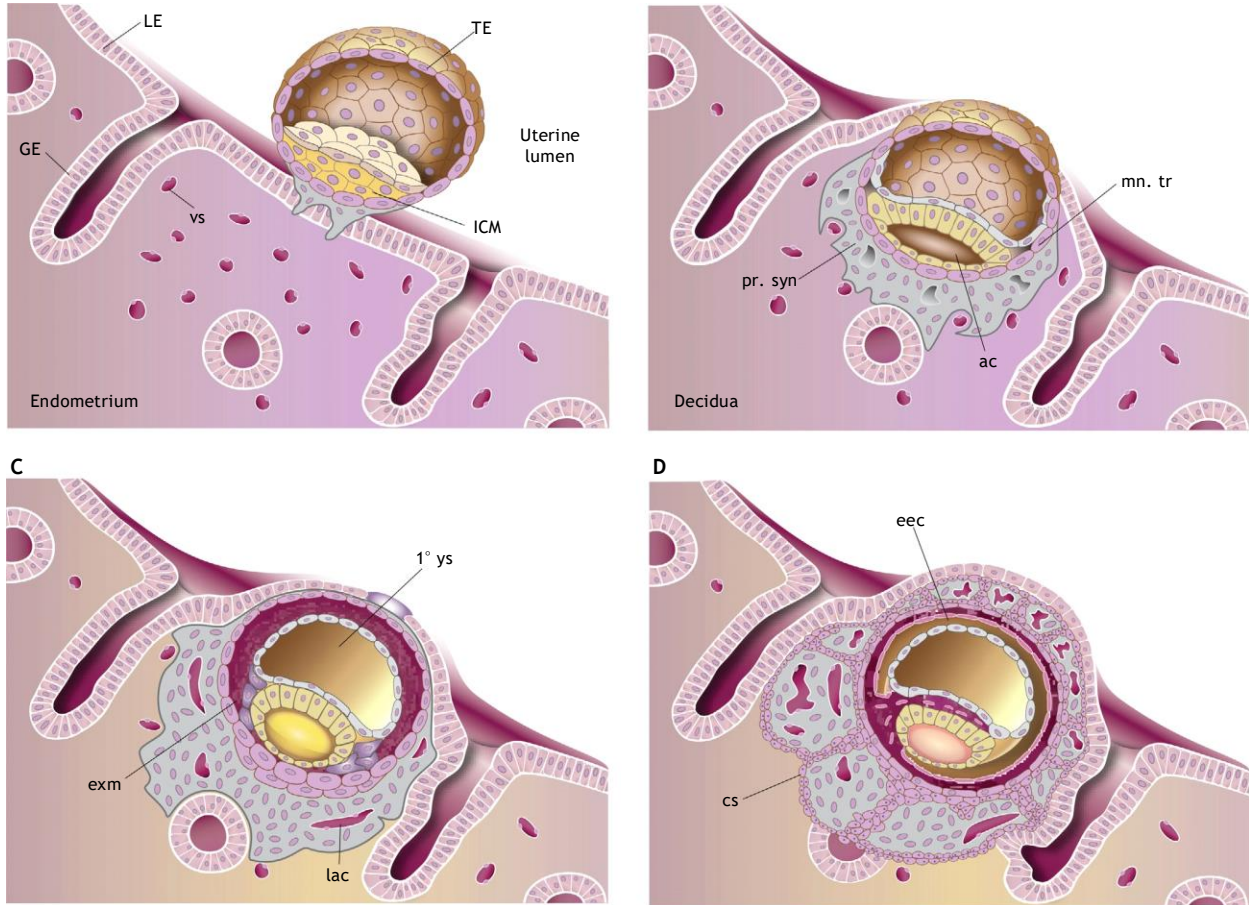


Fig. 1. The early stages of human placental development. Diagram depicting the early steps in placenta formation following blastocyst implantation. (A, B) The pre-lacunar stages. (C) The lacunar stage. (D) The primary villous stage. 1° ys, primary yolk sac; ac, amniotic cavity; cs, cytotrophoblastic shell; eec, extra-embryonic coelom; exm, extra-embryonic mesoderm; GE, glandular epithelium; ICM, inner cell mass; lac, lacunae; LE, luminal epithelium; mn. tr, mononuclear trophoblast; pr. syn, primary syncytium; TE, trophoctoderm; vs, blood vessels.

The chorion forms the placenta and consists of the syncytiotrophoblast, cytotrophoblast, and extraembryonic mesoderm. The cytotrophoblast grows into the syncytiotrophoblast as finger-like projections, which are called the primary chorionic villi. The extraembryonic mesoderm splits into somatic and splanchnic mesoderm, and the somatic mesoderm grows into the primary villi

creating the secondary villi. The mesenchyme gives rise to blood cells and vessels, which designates tertiary villi when formed. Capillary beds grow from the villi, which connect to the embryo heart. Maternal blood flowing through the embryonic capillaries provide oxygen and nutrients to the foetus. The villi continue to grow and branch into the villus chorion, which is the foetal placenta ⁽²⁷⁾.

As development continues, cells from the cytotrophoblast continue to extend through the syncytiotrophoblast to eventually form a cytotrophoblastic shell. As progesterone increases, the decidua connective tissue develops into “decidua cells,” which help protect the uterus from an invasion of the syncytiotrophoblast. As the sac continues to grow, the decidua capsularis villi degenerate and eventually disappear as they fuse with the decidua parietalis.

The amniotic sac enlarges faster than the chorionic sac, which causes them eventually to come into contact and fuse into the amniochorionic membrane. The amniochorionic membrane then fuses to the decidua capsularis and, ultimately, the decidua parietalis for stability. The amniochorionic membrane ruptures during labor. The amniochorionic membrane with the fetal vessels makes up the chorionic plate. Parts of the decidua basalis grow into the chorionic plate dividing it into separated septa called cotyledons, in which each contains stem villi ⁽²⁸⁾.

The foeto-maternal junction provides stability for the chorion. The chorionic villi that attach to the decidua basalis are an anchor for the foetal chorionic sac to the endometrium. Endometrial vessels, called spiral arteries, make their way through openings in the cytotrophoblastic shell and reside inside the villi where they release maternal blood to bath the chorionic villi in each cotyledon; this allows for maternal blood to provide oxygen and nutrients to the feotus across the placental membrane. Endometrial veins then drain the blood. Although the foetal vessels are

bathed in maternal blood, there is normally no mixing between maternal and foetal red blood cells ⁽²⁹⁾.

The placental membrane is where the mother and foetus exchange gases, nutrients, etc. The membrane forms by the syncytiotrophoblast, cytotrophoblast, embryonic connective tissue (Wharton's jelly), and the endothelium of foetal blood vessels.

The umbilical cord serves to attach the foetus to the placenta and consists of two umbilical arteries and one umbilical vein.

CELL TYPES OF THE HUMAN PLACENTA

The trophoblast cells, also referred to as cytotrophoblast cells, are initially not in direct contact with maternal tissue underneath the syncytium, but they soon proliferate to create projections that push the primary syncytium to become primary villi (a cytotrophoblast core with an outer layer of syncytiotrophoblast, SCT) (Fig. 1D). Further proliferation and branching result in the formation of the villous trees, and the lacunae develop into the intervillous zone. A continuous cytotrophoblast shell that surrounds the conceptus between the villi and the decidua is formed when cytotrophoblast cells ultimately pass through the primary syncytium and fuse laterally. (Fig. 1D). The inner chorionic plate, which is in touch with the original cavity, the villi, which are separated by the intervillous gap, and the cytotrophoblast shell, which is in contact with the decidua, are the three layers that now cover the blastocyst. ⁽²³⁾.

Trophoblast Cells

Trophoblast cells carry out the placenta's primary activities. Ambrosius Arnold Willem Hubrecht, a Dutch embryologist, coined the word "trophoblast" in 1889 to refer to cells that carry

nutrients and provide a barrier of protection between the mother and baby ⁽³⁰⁾. The trophoblast depends on decidua to maintain its growth and is intrinsically very invasive ⁽³¹⁾. There are several distinct human trophoblast subtypes. These include extra villous trophoblasts (EVT), syncytiotrophoblasts (SCT), and villous cytotrophoblasts (VCT).

The SCT is the placental villi's outer lining, which comes into contact with the mother's glandular secretions and eventually, the blood that flows into the intervillous gap. (Fig. 2). It is the primary location for the transport of gases plus nutrients between the mother and foetus that are important for the development of the foeto-placental complex. The SCT's surface area is increased by 5-7fold by a highly polarised epithelium layer that is densely coated in microvilli. ⁽³²⁾.

To promote diffusion across its whole structure and safeguard the foetus from infections, the SCT is a multinucleated tissue with no cell boundaries ⁽³³⁾, contains many Growth factor and hormone receptors in its microvilli ⁽³⁴⁾. Transporter proteins that efflux xenobiotics as well as amino acids and glucose are abundant in the SCT's apical and basal membranes. The SCT is a significant endocrine organ that secretes hormones and proteins into the bloodstream of the mother to support the metabolic and physiological changes brought on by pregnancy.

Furthermore, SCT serves as a protective immunological barrier since it does not express any human leukocyte antigen (HLA) molecules, which means that circulating immune cells will not recognise the SCT as "non-self" despite the presence of the allogeneic foetus ⁽³⁵⁾. The neonatal Fc receptor (FcRn), which enables the transfer of maternal IgG antibodies to the foetal blood, is also expressed by the SCT ⁽³⁶⁾. Digalactosylated IgG1 molecules are the antibodies that preferentially attach to FcRn on the SCT, and they work well to activate foetal NK cells to defend the neonate before birth ⁽³⁷⁾.

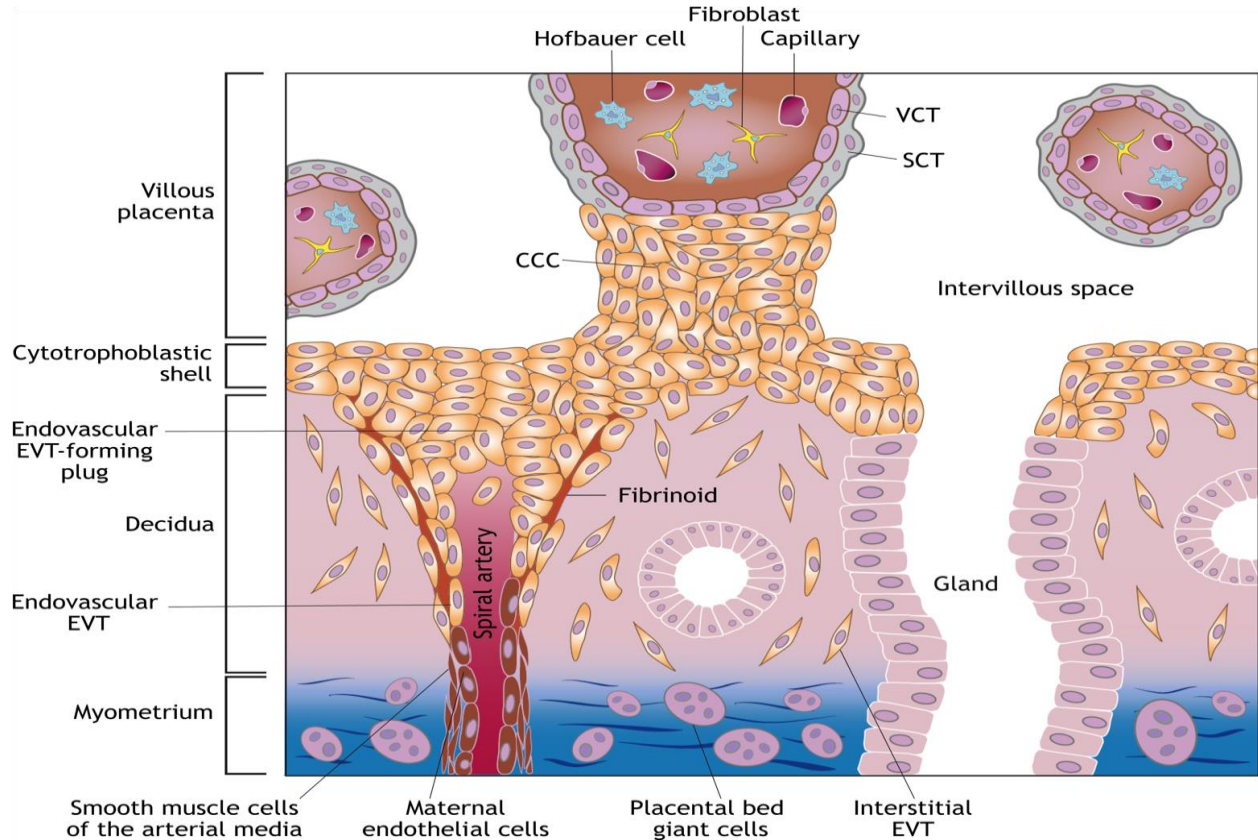


Fig. 2. The maternal-fetal interface and trophoblast subtypes. Cells contained within the villi of the early first trimester placenta and the major trophoblast subtypes in relation to the decidua are represented. The decidual region has been illustrated to include the myometrium. Syncytiotrophoblast (SCT, grey), villous cytotrophoblast (VCT, pink), the cytotrophoblast cell column (CCC) and extravillous trophoblast (EVT) populations (endovascular and interstitial EVT, orange) are indicated. The endpoint of EVT differentiation, placental bed giant cells, are also indicated.

On a basement membrane, the mononuclear VCT is located underneath the SCT (Fig. 2).

Because the VCT are mitotic and show proliferative markers, they have historically been regarded as the 'germinative' layer of trophoblast⁽³⁸⁾. The VCT are cuboidal cells with a high nucleus-to-cytoplasm ratio that first appear in early pregnancy and thereafter coalesce into a continuous layer. Only a thin syncytial layer protects the majority of the villous core from maternal blood at term, when the villous trees have expanded and the VCT layer has become discontinuous, covering only 25% of the villous surface⁽³⁹⁾.

FETAL PLACENTAL VASCULAR TREE DEVELOPMENT

One of the main sources of angiogenesis and vasculogenesis is the placenta, which produces a vascular network more than 500 kilometres long in just nine months. During the third week following fertilisation, haemangioblastic clusters that are already present inside the mesenchymal core of early villi begin to differentiate for vasculogenesis ⁽⁴⁰⁾. The clusters, which often lie just below the trophoblastic basement membrane, create cords of cells. In fact, it is believed that the cytotrophoblast cells' secretion of angiogenic growth factors causes them to differentiate ⁽⁴¹⁾. With increasing gestational age, the cords gradually enlarge to create a network of endothelial cells connected by tight junctions, whose molecular organisation matures ⁽⁴²⁾. Haematopoietic stem cells delaminate from the inner surface of the clusters once a lumen has formed, and after further differentiation, they produce a distinctive clump of closely packed nucleated erythrocytes. These don't move until the foetal placental circulation starts, which happens at the end of the first trimester. Throughout gestation, the villous capillary network continues to grow and change ⁽⁴³⁾, in response to variations in oxygen tension and mechanical stimuli including shear stress and cyclic strain, most likely through angiogenic factors ⁽⁴⁰⁾.

The local release of factors regulates the vasomotor regulation of the foetal placental circulation in the absence of an autonomic nerve supply to the placenta. Nitric oxide and carbon monoxide have been linked to altering the vasomotor tone of the muscular arteries found inside the stem villi, which are regarded to be the primary resistance vessels within the placenta ^(44,45). Recently, it was shown that hydrogen sulphide is an effective vasodilator ⁽⁴⁶⁾. This would provide maximum placental efficiency by matching foetal blood flow inside a lobule to maternal perfusion, albeit there are currently no experimental data to support this theory.

METABOLISM OF PLACENTA

Changes in the mother's blood flow, hunger, and metabolism brought on by the placenta guarantee the placenta a bountiful supply of nutrients. However, because the placenta represents a barrier in the passage of nutrients from the mother to the foetus, there is a risk that it will preferentially use up the supply before it reaches the foetus. The placenta has its own metabolic requirements. According to estimates, the placenta uses 40% of the oxygen given to the foetal-placental unit to produce proteins, with the remaining 30% going to active transport and ionic pumping ⁽⁴⁷⁾. The placenta's structural and metabolic features are likely to restrict this potentially harmful effect.

First off, the development of vasculosyncytial membranes guarantees that the quantity of syncytioplasm interposed between the maternal and foetal circulations is kept to a minimum. These locations often lack mitochondria and other oxygen-consuming organelles like the endoplasmic reticulum; instead, they are concentrated in thicker syncytioplasm regions far from the foetal capillaries. Contrary to what would happen if the syncytiotrophoblast layer were uniformly thick throughout the villous surface, the creation of vasculo-syncytial membranes arranges the metabolic needs of the placenta and foetus in parallel rather than in series.

Second, even when the maternal circulation is formed towards the end of the first trimester, placental metabolism is predominantly glycolytic ⁽⁴⁸⁾. With a pH of 7.18 compared to 7.38 in the maternal serum, a lactate concentration of 0.6 mmol l⁻¹ compared to 0.3 mmol l⁻¹, and a base excess of 27.8 mmol l⁻¹ compared to 22.6 mmol l⁻¹, analysis of the coelomic fluid that is in

communication with the placental tissues at 7 to 11 weeks of pregnancy revealed evidence of anaerobic glycolysis ⁽⁴⁹⁾. Even under conditions of high oxygenation, estimates based on placental tissues delivered at term and perfused in vitro imply that 22% of the glucose eaten is converted to lactate ⁽⁵⁰⁾. The foetus may benefit from part of the eaten glucose being converted to lactate rather than carbon dioxide via the citric acid cycle since it can use lactate as a substrate whereas the placenta cannot. The placental metabolism may be reserving resources for the foetus in this manner.

However, besides fermentation to lactate, there are other ways to regenerate the NAD⁺ required for sustaining glycolysis in early placental tissues. The phylogenetically old polyol pathways are very active in the early human placenta, and the coelomic fluid contains significant amounts of sorbitol, inositol, erythritol, mannitol, and ribitol ⁽⁵¹⁾. The pentose phosphate route, which is crucial for the synthesis of nucleotides to sustain fast cell proliferation, is intimately interwoven with many of the polyol processes. NADPH, which is necessary for the renewal of reduced glutathione and the efficient operation of antioxidant defences, is also produced through the pentose phosphate pathway.

Therefore, the placenta will expand quickly and be protected from harm caused by free radicals if there is a ready supply of glycolytic intermediates that can be sent along these routes.

Because it depends on less complex intracellular machinery, glycolysis may be advantageous in circumstances when resources are not limited, while producing just a small portion of the ATP per glucose molecule that may be obtained by oxidative phosphorylation ⁽⁵²⁾. Given that the placenta is a temporary structure and that mitochondria are energy-intensive to create and maintain, it could be more effective to rely on glycolysis for the majority of energy generation.

Given that the endometrial secretions are carbohydrate-rich and glycogen builds up in the syncytioplasm throughout the first trimester, there is undoubtedly no scarcity of glucose for the placental tissues. ^(53,54).

The trophoblast will use less oxygen thanks to its high dependence on aerobic glycolysis, also known as Warburg metabolism, than it would if oxidative phosphorylation predominated. As a result, the foetus has access to more oxygen as well as protected resources like lactate.

PLACENTAL FUNCTIONS

The placenta plays a vital role in maternal-foetal physiology. The placenta has numerous responsibilities:

Implantation

The syncytiotrophoblast, which later grows as part of the placenta, facilitates implantation by directly invading the wall of the endometrium in the uterus ⁽¹⁷⁾.

Maternal recognition of pregnancy

Human chorionic gonadotropin (hCG) is synthesized and released from the syncytiotrophoblast to stimulate luteal progesterone production to maintain the pregnancy. Without hCG production, the absence of progesterone would trigger menses and, therefore, the sloughing of the endometrium with the implanted zygote ⁽²³⁾.

Transport Function

Although there are two potential exceptions, the lack of intercellular connections in the syncytiotrophoblast layer implies that exchange must occur through the apical and basal plasma membranes. First, it has been suggested that transtrophoblastic routes exist. Second, it is widely acknowledged that all human placentas have minor, dispersed abnormalities that result in the development of fibrin plaques ^(55,56). In this context, the plaques constitute a potential pathway for hydrophilic molecule diffusion; but, in a broader sense, they may also be potential entry points for maternal immune cells and a vertical pathogen transmission.

Diffusion, transporter-mediated mechanisms, and endocytosis/exocytosis are the three primary processes that allow exchange to take place across an intact placental membrane.

According to Fick's law of diffusion, the rate of diffusion of an uncharged molecule is proportional to the surface area for exchange, the molecule's diffusivity, and its concentration gradient, and inversely related to the diffusion distance between the circulations. It is plausible to believe that the needs for diffusional exchange, and in particular oxygen exchange, are the primary drivers of placental design given the significance of these structural features. Therefore, as the pregnancy progresses, the terminal villi and vasculosyncytial membranes will become more elaborate, increasing the organ's ability to diffuse. This theory is reinforced by the fact that the placenta's specific theoretical diffusing capacity for oxygen, measured stereologically (ml per min per kPa per kg foetus), remains constant with gestational age ⁽⁵⁷⁾.

Small, comparatively hydrophobic molecules quickly diffuse over the plasma membrane, including breathing gases. As a result, rather than the villous membrane's surface area or thickness, their flow is more dependent on the concentration gradient across it. The rate of blood flow through the membrane, however, has a greater impact on the concentration gradient than do maternal and ambient influences. As a result, the interchange of these molecules is known as

being "flow-limited." Therefore, uterine or umbilical circulation problems can significantly affect how quickly a foetus grows. For lipid insoluble (hydrophilic) substances, such as glucose, that do not diffuse through plasma membranes as quickly, the concentration gradient is frequently more sustained. In this instance, the villous membrane's structural characteristics are more important, and exchange is described as "membrane- or diffusion-limited."

Transporter proteins may be introduced into the plasma membrane to facilitate the exchange of hydrophilic or charged molecules. Although they are a broad and varied family, transporter proteins have several characteristics in common, including substrate selectivity, saturation kinetics, and the capacity for competitive inhibition ⁽⁵⁸⁾. The process of exchange along a concentration gradient may be expedited by transporter proteins more quickly than by simple diffusion alone. The GLUT family of transporters, which moves glucose, is the prime example in the placenta. As an alternative, they can facilitate active transport, an energy-dependent mechanism that allows molecules like amino acids to exchange against a concentration gradient. Leptin upregulates glucose and amino acid transporters, boosting the transfer of nutrients by regulating the expression of the genes encoding transporter proteins ⁽⁵⁹⁾. One of the main advantages of transporter-mediated exchange is that, in challenging circumstances, the rate may be adjusted by changing the quantity of proteins introduced into the plasma membrane ⁽⁶⁰⁾. Thus, placental expression of several amino acid transporters increases, boosting the flow, when the surface area for exchange is reduced experimentally in mice or the mother is malnourished ^(61,62). Invaginations develop at the apical cell surface, pinch off, and then proceed deeper into the cytoplasm by the process of endocytosis. There, they could join forces with lysosomal vesicles or move within the cell and join forces with the basal surface during exocytosis. Both are present in the human placenta's syncytiotrophoblast ^(63,64). Several proteins of maternal origin build up in the

coelomic and amniotic fluids during the first trimester of pregnancy⁽⁶⁵⁾, whereas immunoglobulin G (IgG) penetrates the placenta via this method later in pregnancy⁽⁵⁸⁾. The presence of IgG receptors in the microvillous membrane invaginations and vesicles may give specificity and the capacity to evade lysosomal degradation during the endocytosis phase.

Endocrine modulation of maternal metabolism

Many hormones are released from the placenta to uphold a pregnancy. The placental growth factor is released from the placenta to prepare the mother's body for pregnancy in terms of cardiovascular adaption. Additionally, the placental growth factor promotes foetal development and maturity. Human chorionic somatomammotropin (HCS), also known as human placental lactogen (HPL) promotes breast development and alters the metabolism of the mother. It decreases maternal insulin sensitivity so that more glucose is available for the foetus ^(17,59).

The placenta is a major endocrine organ, and placental hormones have diverse profound effects on maternal physiology and behaviour^(66,67). They promote an increase in food intake and energy storage during the early stages of pregnancy, but as the pregnancy progresses, they utilise these reserves to support foetal development and nursing ^(68,69). The family of closely related placental lactogens (hPL) and placental growth hormone (hGH), which share 96% of their amino acid sequences, are the two most significant hormones in this regard.

The hormones progesterone and hPL both stimulate the appetite, and by the end of the first trimester, when the conceptus' metabolic needs are still relatively modest, the mother's food consumption has increased. Increased fat deposition is the outcome, and the usual homeostatic

systems that control energy balance are lost. Adipose tissue produces leptin, which typically feeds back to the brain to reduce appetite, but pregnancy causes central leptin resistance. Leptin secretion by the syncytiotrophoblast is greatly increased during pregnancy and is partially controlled by human chorionic gonadotropin and 17 β -oestradiol ⁽⁵⁹⁾. Expression levels peak towards the conclusion of the second and in the beginning of the third trimester and are strongly correlated with maternal serum concentrations. The hormone affects hunger both centrally and locally, including the expression of placental transporters. The central insensitivity appears to be mediated by placental lactogen and prolactin, which are released by the trophoblast and decidua, respectively⁽⁷⁰⁾. During the first trimester of pregnancy⁽⁷⁰⁾, these hormones also promote the growth of beta cells in the mother's pancreas, raising insulin levels and promoting the synthesis of fat once more ⁽⁶⁹⁾.

The mother experiences insulin resistance later in the pregnancy, which is accompanied by an increase in lipolysis, circulating triglycerides, and free fatty acids. Previously, these alterations were thought to be caused by placental lactogen and/or prolactin, but more recent research questions this theory ⁽⁶⁸⁾. It seems that placental growth hormone may have a more significant impact. The syncytiotrophoblast secretes placental growth hormone tonically, as opposed to the pituitary, which secretes it in a pulsatile manner. Only 13 amino acids out of a total of 191 amino acids are different between the two forms, and this similarity is enough for the placenta to limit the synthesis of maternal pituitary growth hormone by the middle of pregnancy. As implied by its name, it possesses potent growth-promoting properties that work through GH receptors.

Another crucial regulator of insulin-like growth factor 1 is placental growth hormone. ^(63,52)

Despite not entering the foetal circulation, this protein has a significant impact on foetal development. The activities of maternal concentrations are assumed to be mediated by

modifications in maternal metabolism and nutritional partitioning, stimulation of placental morphogenesis, and an increase in maternal blood flow to the placenta. Maternal concentrations are correlated with birthweight ^(69,71).

Foetal Protection from Any Immunologic Attack

The placenta holds the ability to metabolize numerous substances and protect against microbial infection. Macrophage in the stroma of the chorionic villi and syncytiotrophoblast play a critical role in the protection of the foetus. Additionally, many leukocytes reside in the decidua of the endometrium to support a successful pregnancy ^(54,55).

A Selective Barrier: The Placenta

To ensure the development of its neuroendocrine and gonadal systems, the foetus needs a separate microenvironment that is free from ambient contaminants, stress hormones, and maternal sex. In order to ensure the detoxification and efflux of xenobiotics, the syncytiotrophoblast is therefore equipped with a range of enzymes and transporters, acting similarly to the hepatic cells in an adult. The enzyme 11-b-hydroxysteroid dehydrogenase 2 (11-bHSD2), which oxidises maternal cortisol to the inactive metabolite cortisone, is one of the better studied instances. In this way, the placenta protects the foetus from the potentially damaging effects of maternal stress hormones, which when given directly to the foetus inhibit development and induce lower cell proliferation. There have been reports of sex-specific changes in placental 11-bHSD2 activity ^[72], which may help to explain why boys are more likely than

females to suffer illnesses like autism as a result of developmental programming after having negative prenatal experiences.

At term, P-glycoprotein and members of the multidrug resistance protein family have been localised to the endothelium of the villous capillaries and the apical surface of the syncytiotrophoblast ⁽⁷³⁾. The ATP dependent efflux of a wide variety of anionic organic substances is mediated by these transporters, protecting the embryo from exposure to potentially harmful xenobiotics.

Only at a rate equal to that of the placenta's distribution of nutrients and oxygen does foetal development occur. There is now abundant proof that the placenta is not only a passive conduit connecting the mother and foetus, but also has the capacity to react to the mother's supply signals and the baby's demand signals ^(47,74). A complicated interplay between placental development, transporter protein expression, placental blood flow rates, transmembrane concentration gradients, and the metabolic needs of the placental tissues controls how well placental exchange occurs. Under ideal circumstances, this interplay between maternal, placental, and foetal hormones delivers an appropriate supply to the foetus without excessively depleting maternal reserves. One advantage is that hydrophilic solutes, which are expected to travel via water-filled transtrophoblastic channels, can pass through it more easily.

THE DECIDUA REGULATES PLACENTAL DEVELOPMENT.

The endometrium, which becomes decidua during pregnancy under the influence of progesterone released by the corpus luteum, is where the human placenta develops. The ovarian-pituitary axis hormones regulate the endometrium's extremely dynamic cyclical regeneration, differentiation, and shedding during the menstrual cycle. In humans, characteristics of decidualisation (pre-decidual alteration) start to appear around the spiral arteries following the mid-secretory period of the menstrual cycle. After implantation, the endometrium must properly decidualize in order for the placenta to grow. This process is expected to involve all of the major biological components of the endometrium, including the glands, vessels, stromal cells, and immune cells.

Stromal cells and endometrial glands

When an embryo implants and the placenta grows, the endometrial glands are crucial. Early in pregnancy, when endovascular plugs merely let seepage of maternal blood into the intervillous space, the conceptus relies on glandular secretions as the source of histotrophic sustenance.

The endometrium's stromal cells also generate a variety of growth factors that activate the glands. The basement membrane proteins fibronectin and laminin are secreted by the stromal cells as they decidualize, creating a scaffold for the EVT to pass through. Uncertainty still exists on what exactly constitutes a receptive decidualized endometrium that can sustain the growing placenta. This is a significant problem since there is mounting evidence that imperfect decidualization precedes pregnancy issues. Although it is unknown how these factors impact decidualization and embryo receptivity, maternal diet, extremes of reproductive life, low or high BMI, endocrine abnormalities (such as thyroid illness), and diabetes can all alter the cycling of a healthy endometrium.

Other immune cells and uterine leukocytes

Uterine natural killer (uNK) cells are an innate lymphoid cell subtype that predominate during the first trimester of pregnancy. In the uterine environment, these cells constitute about 70% of the immune cells, compared to 20% of macrophages and 10% of T cells. Neutrophils are few, while B cells, mast cells, and neutrophils are almost non-existent. Innate immune system cells rather than adaptive immune system cells (T and B cells) are consequently more prevalent in this atypical immunological milieu.

Due to their invasive characteristics, EVT cells have been likened to tumour cells. The behaviour of trophoblast cells inside the decidual milieu is managed, unlike that of tumour cells. As the EVT penetrates deeper into the tissue, necrosis of the decidua is thus not observed, with the exception of the mysterious Nitabuch's layer, a thin ring of fibrinoid tissue next to the shell at the maternal-foetal border.

EVT cells, which develop from the tips of anchoring villi that connect the villous trees to the endometrium, are most prevalent during the first trimester of pregnancy. The cells multiply before migrating away from the placenta, either through the endometrial stroma or along the spiral artery lumens. They interact with maternal immune cells, namely the uterine natural killer (uNK) cells of the innate immune system, through the latter route. In the late secretory phase of the non-pregnant cycle, uNK cells build up in the endometrium and are particularly prevalent at the early implantation site. Despite their moniker, uNK cells don't really destroy the trophoblast cells that are migrating. Instead, it is believed that in response to the right stimulus, they produce proteases and cytokines that control trophoblast migration and affect artery remodelling ⁽⁷⁵⁻⁷⁷⁾.

With the help of killer-cell immunoglobulin-like receptors (KIRs) on the uNK cells and polymorphic HLA-C ligands on the trophoblast, there is a carefully crafted conversation between

the two cell types. Pregnancy issues including miscarriage, pre-eclampsia, and growth restriction are more likely when specific ligand and receptor combinations are present ⁽⁷⁸⁾.

The "major obstetrical syndromes" have been linked to inadequate remodelling of the spiral arteries ⁽⁷⁹⁾. The greatest mechanistic connection is shown in pre-eclampsia, when the placenta's subsequent mal-perfusion is hypothesised to result in oxidative stress ⁽⁸⁰⁾.

The syncytiotrophoblast can produce pro-inflammatory cytokines and angiogenic regulators in response to oxidative stress, which activates the maternal endothelium and causes preeclampsia ^(81,82). Recently, normotensive foetal growth restriction and closely associated endoplasmic reticulum stress have been found in placentas from instances of early-onset pre-eclampsia ^(83,84). Suppression of protein translation, which in vitro lowers the rate of cell proliferation, is one of the effects of endoplasmic reticulum stress. As a result, we hypothesise that placental endoplasmic reticulum stress is primarily responsible for growth restriction ⁽⁸⁵⁾, even if similar pathways can also play a role in the activation of pro-inflammatory responses when present in high concentrations ⁽⁸⁶⁾.

Adopting bipedalism may make these stressors in humans worse because while standing up straight, a growing uterus forces the inferior vena cava against the lordosis of the lumbar spinal column ⁽⁸⁷⁾. By reducing venous return to the heart, such compression will jeopardise cardiac output. Additionally, it will engorge the veins in the intervillous region, limiting blood flow and perhaps causing variations in placental oxygenation. Fluctuations in oxygenation are a strong stimulation for the development of placental oxidative stress ⁽⁸⁹⁾, and the impact is more noticeable when the mother is lying on her back. ⁽⁸⁸⁾

PREGNANCY COMPLICATIONS AND ABNORMAL PLACENTAL DEVELOPMENT

Pregnancy issues often start as aberrant placenta development in the first trimester ⁽⁹⁰⁾. The major obstetric syndromes (GOSs) are a group of issues that include pre-eclampsia, foetal growth restriction (FGR), unexplained stillbirth, placental abruption, and premature labour ⁽⁹¹⁾. A significant fraction of maternal and neonatal morbidity and death observed across all populations is caused by these diseases ⁽⁹²⁾.

The root cause of the GOSs is defective trophoblast invasion. For proper foetal growth and development, trophoblast cells must successfully enter the decidua to acquire access to the mother's blood supply and alter around 30 to 40 spiral arteries deep inside the myometrium ^(93,94). Blood flow into the intervillous area is disordered if the arteries are not properly converted and maintain their contractile medium. This, together with a lack of nutrients and oxygen, prevents the villous tree from spreading out as the pregnancy progresses, decreasing the surface area available for exchange and raising the possibility of FGR and stillbirth. Additionally, the chorionic membranes may prematurely split, leading to placental abruption or preterm labour, if the process of chorion frondiosum regression to produce the chorion-laeve does not proceed appropriately. Pre-eclampsia is caused by the leakage of products from the stressed and inadequately perfused placenta into the mother's bloodstream, which sets off a systemic endothelial dysfunction ^(95,96). Therefore, the degree of arterial invasion and the number of invaded arteries determine the precise clinical consequence of faulty trophoblast invasion. Defective artery transformation has been difficult to characterise and, consequently, diagnose early in pregnancy since profound trophoblast invasion into the uterus is a trait seen only in humans and big apes. To address this, several clinical tests are being developed, such as uterine

artery Doppler velocimetry, which evaluates blood flow resistance and serves as an illustrative indicator of the extent of spiral arterial remodelling ⁽⁹⁷⁾. Measurement of pregnancy-associated plasma protein-A (PAPPA-A) in maternal serum in the first trimester is a helpful indicator of a GOS because it is expressed more often as the EVT progresses deeper ⁽⁹⁸⁾.

It is crucial to comprehend how EVT invasion into the uterus is controlled since faulty trophoblast invasion is the root cause of the GOS. Clinical studies in which the placenta implants on a spot where decidua is missing or defective make it obvious that decidua plays a function in preventing placental cells from migrating too far ⁽⁹⁹⁾. This can happen over a scar from a prior caesarean section or in the bottom part of the uterus near the cervix. When this happens, the EVT enters the myometrium and kills the smooth muscle cells, giving off a "fibrinoid" look similar to that of the spiral arterial media that the trophoblast converts. Additionally, there is a marked reduction in the fusing to placental bed giant cells that is often seen at the conclusion of EVT migration (100). Together, these findings show how the decidua mediates the delicate balancing act required to maintain the territorial barrier between the placenta and the mother.

Another factor could be adopting an upright position, which presents special haemodynamic difficulties to the placental circulations (101). Bipedalism and human reproduction therefore interact in ways that go beyond the problem of pelvic limitation.

The placenta's blood supply is not distributed equally. As a result, placental blood flow and subsequent pregnancy success are probably significantly influenced by the site of implantation and the placenta's subsequent placement inside the uterus. In humans, both uterine arteries contain a large number of branches, and each one nourishes the uterus' corresponding side. There is no evidence that the anastomoses between the two uterine arteries are functional. Both uterine arteries in individuals with a centrally placed placenta showed comparable resistance, and the

uteroplacental blood flow requirements are satisfied by equal distribution from both uterine arteries.

However, when the placenta is positioned laterally, one uterine artery typically provides the bulk of the uteroplacental blood flow requirements, with some assistance from the other uterine artery through collateral circulation. Different subjects may have different levels of collateral circulation, and a lack of contribution may encourage the onset of pre-eclampsia, intrauterine growth retardation, or both. The need of a healthy placenta for this cytotrophoblastic invasion is significant, and pre-eclampsia causes the cytotrophoblasts to fail to develop a vascular adhesions phenotype.

Pre-eclampsia, intra uterine growth restriction (IUGR), or both have a substantial correlation with placental position, uterine artery resistance, and undesirable consequences. Thus, pre-eclampsia may be predicted non-invasively, cost-effectively, and safely by using ultrasonography (USG) to assess the placental position between 18 and 24 weeks. In order to determine if the Location of the placenta between weeks 18 and 24 may be utilised as a predictor for adverse foeto-maternal outcomes., this study was done.

STUDIES THAT SHOWED CORRELATION BETWEEN PLACENTAL LOCATION AND FOETO-MATERNAL OUTCOMES:

Devarajan K et al⁶³(2012) found that previous studies suggest that placental location may affect foetal growth and the risks of preterm birth and preeclampsia. They studied the association between placental location and new-born weight. They conducted a retrospective cohort study of 796 consecutive singleton births in women who delivered at ≥ 37 weeks' gestation between July and October 2009. They evaluated placental location at the time of the second trimester prenatal ultrasound at 16 to 24 weeks' gestation. Placental location was classified as lateral or central/fundal. They assessed the difference in new-born weight according to placental location and the incidence of small for gestational age birth weight < 10 th percentile and pregnancy-induced hypertension. Using logistic regression analysis, odds ratios were adjusted for maternal age, world region of birth, gravidity, parity, maternal weight, history of hypertension or diabetes, current smoking or illicit drug use, and infant sex. Among women with lateral versus central/fundal placentas, the respective mean (SD) birth weights were 3298 (550) g and 3352 (579) g (mean difference 54 g, 95% CI 53 to 161; $P = 0.32$). Relative to central/fundal location, laterally located placenta as had an adjusted OR of 0.81 (95% CI 0.42 to 1.54) for SGA and 0.62 (95% CI 0.18 to 2.10) for preeclampsia/gestational hypertension. Placental location was not associated with differences in new-born weight or other perinatal outcomes.

Zia S et al⁶⁵(2013) showed that the purpose of this study was to determine if placental location is associated with adverse pregnancy outcome and to assess whether any association exists between different blood groups and location of the placenta. Medical records of women were reviewed retrospectively and placental position as documented in the case notes at routine antenatal (20-38 weeks) ultrasonography was identified. Placental position was categorised as anterior, posterior and fundal. Association of placental location with foeto-maternal outcome and different blood groups was noted. A total 474 case notes of women were analysed for placental

location, fetomaternal outcome and blood groups. Anterior placenta was found to have a relation with a greater risk of pregnancy-induced hypertension, gestational diabetes mellitus and placental abruption ($p < 0.001$), while posterior placenta had a significant association with preterm labour ($p < 0.001$). Regarding foetal outcome, an anterior placenta was significantly associated with intrauterine growth retardation and intrauterine foetal death ($p < 0.001$). The majority (54%) of women with an anterior placenta were O-positive blood group, while 46% of women in the posterior placenta group were A-positive blood group ($p < 0.001$). Anterior placental implantation is associated with an increased risk of pregnancy-induced hypertension, gestational diabetes mellitus, placental abruption, intrauterine growth retardation and intrauterine foetal death. Posterior placenta has a significant association with preterm labour and A-positive blood group. Anterior placenta is common in women with O-positive blood group. Placental location may be an important determinant of pregnancy outcome.

Gizzo S et al ⁶⁷(2015) found that during a standard obstetrical sonogram, the assessment of placental location (PL) is often limited to a mere notional description without formulating any association to possible implications on pregnancy and childbirth. The aim of the study was to speculate if different sites of PL may have a role in influencing fetal presentation-(FP) at birth and if certain pregnancy-complications may be more closely associated with one rather than with another PL. They conducted an observational-prospective-cohort study on pregnant women referred to the Ob/Gyn Unit of Padua University for routine third-trimester ultrasound scan. For all eligible patients They evaluated the correlation between sites of PL and perinatal maternal/fetal outcomes. Non-cephalic presentation was found in 1.4% of anterior, 8.9% of posterior, 6.2% of fundal and 7.2% of lateral insertions. FP at the beginning of the third trimester as opposed to presentation at birth was concordant in 90.3% of anterior, 63.3% of posterior and

76.5% of lateral insertions. Considering only non-cephalic fetuses we observed a decreasing probability for spontaneous rotation in the following lies: 88% anterior-PL, 80% posterior-PL, 77% lateral-PL, and 70% fundal-PL. Patients with posterior-PL (significantly associated with previous-CS) had a significantly higher CS-rate (due to previous-CS and breech-presentation). Significant differences were found in terms of gestational-hypertension and fresh-placental-weight between different sites of PL. In conclusion their data showed that an understanding of the role that PL plays in influencing the incidence of certain maternal-fetal conditions may assist Clinicians in improving perinatal maternal/fetal outcomes.

Jing L et al⁶⁹(2018) aimed to evaluate the site of placentation on the pregnancy outcomes of patients with placenta previa. This retrospective study included 678 cases of placenta previa. Basic information and pregnancy outcome data were collected. Differences between the different placenta previa positions and pregnancy outcomes were compared using the chi-square and independent *t* tests. Logistic and multiple regression analyses were used to calculate the odds ratios (ORs) to determine the risk factors for PAS disorders and postpartum hemorrhage and evaluate the effect of placental attachment site on pregnancy outcomes. There was no significant difference between the PAS disorders rate and the incidence of complete placenta previa depending on the type of placentation; however, placental attachment site influenced the pregnancy outcome. Placental attachment to the anterior wall was associated with shorter gestational age, low birth weight, lower Apgar score, higher prenatal bleeding rate, increased postpartum hemorrhage, longer duration of hospitalization, and higher blood transfusion and hysterectomy rates compared to cases with lateral/posterior wall placenta. Placental attachment at the incision site of a previous cesarean section significantly increased the incidence of complete placenta previa and PAS disorders compared with placental attachment at a site

without incision, but did not significantly influence pregnancy outcomes. Placental attachment to the anterior wall was an independent risk factor for postpartum hemorrhage in patients with placenta previa. Placental attachment to a previous incision site was an independent risk factor for PAS disorders. The site of placental attachment in patients with placenta previa has an important influence on the pregnancy outcome. When the placenta is located on the anterior wall, clinicians should pay attention to the adverse pregnancy outcomes and the possibility of massive postpartum hemorrhage. In cases of placental attachment to the uterine incision site, physicians should be highly vigilant regarding the occurrence of PAS disorders.

Harper LM et al⁶¹(2010) showed that to estimate the association between placenta previa and abnormal fetal growth. Retrospective cohort study of consecutive women undergoing ultrasound between 15–22 weeks. Groups were defined by the presence or absence of complete or partial placenta previa. The primary outcome was intrauterine growth restriction (IUGR), defined as a birth weight <10th percentile by the Alexander growth standard. Univariable, stratified and multivariable analyses were used to estimate the effect of placenta previa on fetal growth restriction. Of 59,149 women, 724 (1.2%) were diagnosed with a complete or partial previa. After adjusting for significant confounding factors (black race, gestational diabetes, preeclampsia, and single umbilical artery,), the risk of IUGR remained similar (adjusted odds ratio 1.1, 95% CI 0.9–1.5). The presence of bleeding did not impact the risk of growth restriction. Placenta previa is not associated with fetal growth restriction. Serial growth ultrasounds are not indicated in patients with placenta previa.

Jang DG et al⁶²(2011) observed that the purpose of this retrospective cohort study was to elucidate whether the location of placenta below uterine incision in cesarean section is important

in the development of maternal complications in placenta previa patients. The study was conducted on 409 patients 414 parturition at 3 hospitals in affiliation with the Catholic Medical Center, Seoul, Korea from May 1999 to December 2009. The subjects were divided to two groups: the group whose placenta was located in the anterior portion of the uterus (anterior group) and the group whose placenta was located in the posterior portion of the uterus (posterior group). And then they are compared to each other. Logistic regression was used to control for confounding factors. In the anterior group, regardless of confounding factors, the incidence of excessive blood loss (OR 2.97; 95% CI: 1.64-5.37), massive transfusion (OR 3.31; 95% CI: 1.33-8.26), placental accreta (OR 2.60, 95% CI: 1.40-4.83), and hysterectomy (OR 3.47, 95% CI: 1.39-8.68) was higher. Sonographic determination of the placental position where its location beneath the uterine incision is very important to predict maternal outcomes in placenta previa patients, and such cases, close attention should be paid for massive hemorrhage.

and clinical factors, such as infant sex, weight and race, on placental structure and function.

Nagpal K et al ⁷⁰(2018) showed that placenta is the connecting organ between the mother and the fetus. It supplies oxygen and all the necessary elements for the growth and development of the fetus. In normal pregnancy, the growth of the placenta remains concordant with the growth of the fetus. The sonographic assessment of placenta can give information about the nutritional status of the fetus. It is known that normal placental thickness approximately equals gestational age. It is historically documented that placental weight is one-fifth of the fetal weight and abnormally thin or thick placenta is associated with increased incidence of perinatal morbidity and mortality. However, there are very few studies correlating placental thickness with Neonatal outcome. To correlate ultrasonographic placental thickness at 32 and 36 weeks pregnancy with neonatal outcome. To propose placental thickness as a simple test for prediction of neonatal

outcome. Placental thickness at 32 and 36 weeks was measured by ultrasound, in 130 pregnant mothers with confirmed dates and uncomplicated singleton pregnancy. Placental thickness was categorized as normal (10th–95th percentile), thin (<10th percentile) and thick (>95th percentile) at each stage and was correlated with birth weight and neonatal outcome. Neonatal outcome was good in women with normal placental thickness (10th–95th percentile) at 32 and 36 weeks and was compromised in women with thin (<10th percentile) and thick (>95th percentile) placentae. Placental thickness at 32 and 36 weeks corresponds well with gestational age and is a good prognostic factor in assessing neonatal outcome. Therefore, placental thickness should be measured in addition to biometric parameters in antenatal women undergoing ultrasound.

Granfors M et al ⁷²(2019) found that the impact of placenta previa on pregnancy, delivery and infant outcomes has been extensively studied. However, less is known about the possible association of placental location other than previa with pregnancy outcomes. The aim of this study was to investigate if placental location other than previa is associated with adverse pregnancy, delivery and infant outcomes. This is a population-based cohort study, with data from the regional population-based Stockholm-Gotland Obstetric Cohort, Sweden, from 2008 to 2014. The study population included 74 087 nulliparous women with singleton pregnancies resulting in live-born infants, with information about placental location from the second-trimester ultrasound screening. The association between placental location (fundal, lateral, anterior or posterior) and pregnancy outcomes was estimated using logistic regression analysis. Odds ratios (OR) with 95% confidence intervals (95% CI) were calculated, and adjustments were made for maternal age, height, country of birth, smoking in early pregnancy, sex of the infant and in vitro fertilization. Main outcome measures were pregnancy, delivery and infant outcomes. Compared with posterior placental location, fundal and lateral placental locations were

associated with a number of adverse pregnancy outcomes, the most important being: very preterm birth (<32 weeks of gestation) (adjusted OR [aOR] 1.78, 95% CI 1.18-2.63 and aOR 2.12, 95% CI 1.39-2.25, respectively), moderate preterm birth (32-36 weeks of gestation) (aOR 1.23, 95% CI 1.001-1.51 and aOR 1.62, 95% CI 1.32-2.00, respectively), small-for-gestational-age birth (aOR 1.67, 95% CI 1.34-2.07 and aOR 1.77, 95% CI 1.39-2.25, respectively) and manual removal of the placenta in vaginal births (aOR 3.27, 95% CI 2.68-3.99 and aOR 3.27, 95% CI 2.60-4.10, respectively). Additionally, lateral placental location was associated with preeclampsia (aOR 1.30, 95% CI 1.03-1.65) and severe postpartum hemorrhage (aOR 1.42, 95% CI 1.27-1.82). Compared with posterior placental location, fundal and lateral placental locations are associated with a number of adverse pregnancy, delivery and infant outcomes.

Wasim T et al⁷⁴(2020) showed that to assess maternal and fetal morbidity associated with placenta previa and morbidly adherent placenta (MAP). All patients with placenta previa who delivered in services hospital from April 1, 2017 to March 31, 2019 were included. Maternal and fetal outcomes were compared amongst patients with placenta previa and MAP. Total of 8002 patients delivered with 152 (1.9%) diagnosed as placenta previa and 56 (36.8%) amongst them had MAP. One hundred thirty-one out of One hundred fifty-two (86.1%) of Their patients were booked. Increased number of caesarean section, multi parity and anterior placenta had significant association with MAP ($p<0.0001$). Maternal morbidity in terms of postpartum hemorrhage >2000ml, caesarean hysterectomy, number of blood transfusions, bladder injury, need for ICU admission was significantly more in patients with MAP ($p<0.0001$). Case fatality was 3% with two maternal deaths in MAP and none in placenta previa. Fetal outcome was good in both groups as gestational age at delivery was 36 weeks or more, birth weight was ≥ 2.5 kg and >6 APGAR score ($p<0.05$). Two neonatal deaths occurred in MAP and one in placenta previa owing

to prematurity. MAP is a dreadful complication of placenta previa with increased maternal morbidity. Regular antenatal care with adequate arrangement of blood transfusion and multidisciplinary approach can reduce maternal mortality.

Sun X et al⁷⁸(2021) exclaimed that the placenta is a transitory organ indispensable for normal fetal maturation and growth. Recognition of abnormal placental variants is important in clinical practice, and a broader understanding of the significance of placental variants would help clinicians better manage affected pregnancies. Increased thickness of the placenta is reported to be a nonspecific finding but it is associated with many maternal and fetal abnormalities, including preeclampsia and abnormal fetal growth. In this review, They address the questions regarding the characteristics of placenta thickness and the relationship between thickened placenta and poor pregnancy outcomes.

Shinde GR et al⁸⁰(2021) observed that variations in placental thickness are associated with increased perinatal morbidity and mortality. However, only very few studies have been established on the correlation between placental thickness with birth outcomes. This study correlated placental thickness in 2nd and 3rd trimesters with neonatal outcome, maternal weight gain, and body mass index (BMI). A total of 116 patients aged between 20 to 50 years with singleton pregnancy and regular menstrual history (and sure about their last menstrual period) were included. Placental thickness was measured at 24 and 36 weeks by ultrasound and was divided into three groups: Group A (normal placenta), Group B (thin placenta), and Group C (thick placenta); and correlated with neonatal outcome, maternal weight gain, and BMI. Out of the 116 pregnant women, 55 (47.4%) were primigravida and 61 (52.6%) were multigravida. Six patients (3.6%) delivered pre-term before 36 weeks. In the 2nd and 3rd trimesters, most cases had

normal placental thickness (Group A; 93.1% and 92.7%), followed by thin placenta (Group B; 5.2% and 7.3%) and thick placenta (Group C; 1.7% and 0), respectively. Two patients with thin placenta had neonatal death. A significant positive correlation was found between birth weight and placental thickness (at 24 weeks; 0.516^r , $P<0.00001$ and at 36 weeks; 0.669^r , $P<0.00001$) and maternal weight gain and birth weight (0.563^r , $P<0.00001$). Placental thickness on ultrasonography demonstrated well the correlation between birth weight in 2nd and 3rd trimesters and increased incidence of antenatal and postpartum complications resulting from thin placenta.

Alsammani Jr MA et al ⁸²(2021) exclaimed that placenta previa is a major obstetric problem with high rates of fetomaternal mortality and morbidity. This study aimed to determine the prevalence and fetal and maternal outcomes of major degree placenta previa among Sudanese women. This is a prospective descriptive study conducted in the period from January 1 to June 30, 2109, at Omdurman Maternity Hospital, Khartoum, Sudan. Fetal and maternal complications associated with major degree placenta were analyzed using descriptive statistics. The total number of deliveries was 22,000, of which 87 cases were of major degree placenta previa, giving a prevalence rate of 0.4%, the hysterectomies rate was 23% (n= 20), and the total maternal deaths were 6.9% (n= 6). Intraoperative interventions used to control the bleeding were multiple hemostatic sutures in 34.5% (n=30) of cases, followed by uterine backing (20.7%; n= 18), and uterine artery ligation (12.6%; n=11). The common reported maternal complications were bladder injuries (28.7%; n= 25) followed by bowel injuries (4.6%; n=5). Of all mothers, 48.27% (n=42) were admitted to the intensive care unit (ICU). Of all deliveries, 26.4% (n=23) were preterm, and 38% (n=33) of neonates were admitted to the newborn intensive care unit (NICU), and 9.2% (n=8) were fresh stillbirth (FSB). Neonatal complications were comparable to

other studies but maternal deaths were relatively high. The study indicated the need for effective management protocols and more training of the medical staff in order to overcome the problem.

Racher ML et al⁸³(2021) showed that the purpose to the study was to determine the relationship, if any, between the placental location site and antepartum complications of pregnancy. A University research librarian conducted a comprehensive literature search using the search engines PubMed and Web of Science. The search terms were "placental location" AND "pregnancy complications" OR "perinatal complications. There were no limits put on the years of the search. The search identified 110 articles. After reviewing all the abstracts, relevant full articles, and references of full articles, there were 22 articles identified specific to antepartum complications. Central + fundal locations compared to all lateral were associated with a lower risk of hypertension during pregnancy [RR = 0.47, 95% CI: 0.31-0.71]. Central location compared to all lateral was also associated with lower risk of hypertension during pregnancy [RR = 0.39, 95% CI: 0.26-0.59]. Placenta locations in the lower uterine segment were associated with greater risk of antepartum hemorrhage (APH) [RR = 2.99, 95% CI: 1.16-7.75] compared to above the lower uterine segment. No differences were observed in placental locations and gestational diabetes (GDM), preterm prelabor rupture of membranes (PPROM), preterm delivery (PTD) or on a placental abruption. Central and fundal location sites and central location alone decreased the risk of hypertension during pregnancy. Low uterine segment location sites increased the risk for APH. There were no effects of placenta location sites on the development of GDM, PPRM, PTD or abruption.

Tairy D et al⁸⁴(2021) observed that the uterine location of placenta previa (PP), anterior vs. posterior has an impact on pregnancy outcome. They aimed to study maternal and neonatal

outcome and placental histopathology lesions in anterior vs. posterior PP. The medical records and histopathology reports of all singleton cesarean deliveries (CD) performed due to PP, from 24 to 41 weeks, between 12.2008 and 10.2018, were reviewed. Placental lesions were classified into maternal and fetal vascular malperfusion lesions (MVM, FVM), maternal and fetal inflammatory responses (MIR, FIR). Gestational age (GA) at delivery was similar between the anterior PP (n = 67) and posterior PP (n = 105) groups. As compared to the posterior PP group, the anterior PP group had higher rate of previous CD ($p < 0.001$), placental accreta spectrum ($p = 0.04$), lower neonatal Hb at birth ($p = 0.03$), higher rate of neonatal blood transfusion ($p = 0.007$) and prolonged maternal hospitalization ($p = 0.02$). Placentas from the anterior PP group had lower weights ($p = 0.035$), with increased rate of MVM lesions ($p = 0.017$). The anterior PP location is associated with increased adverse maternal and neonatal outcome, lower placental weights and increased rate of malperfusion lesions. Abnormal placentation in the scarred uterine wall probably has an impact on placental function.

Doctory N et al⁸⁵(2022) found that to explore the potential association of lateral placentation with pregnancy outcome. The database of a tertiary medical center was searched for women who gave birth to a singleton infant in 2012-2020 for whom placental location was documented during antepartum sonographic examination. Clinical data were compared between patients with a central (anterior/posterior/fundal) or lateral placenta using standard statistics. The primary outcome measure was neonatal birthweight; secondary outcome measures were pregnancy complications and mode of delivery. The cohort included 12,306 women: 11,608 (94%) with a central placenta and 698 (5.6%) with a lateral placenta. The lateral placenta group had higher rates ($P < 0.05$) of prior and current cesarean delivery, assisted delivery, and preterm birth. On multivariate regression analyses, placental location (aOR 1.36, 95% CI 1.11-1.66) and maternal

age (aOR 1.02, 95% CI 1.01-1.03) were associated with risk of preterm birth; lateral placenta (aOR 1.22, 95% CI 1.02-1.47), maternal age (aOR 1.07, 95% CI 1.06-1.08), parity (aOR 0.32, 95% CI 0.28-0.35), and prior cesarean delivery (aOR 12.00, 95% CI 10.60-13.60) were associated with risk of current cesarean delivery. The findings suggest that lateral placentation may pose a risk of preterm birth and cesarean delivery compared to central placentation.

Lillegard JB et al⁸⁶(2022) exclaimed that uterine incision based on the placental location in open maternal-fetal surgery (OMFS) has never been evaluated in regard to maternal or fetal outcomes. The aim of this study was to investigate whether an anterior placenta was associated with increased rates of intraoperative, perioperative, antepartum, obstetric, or neonatal complications in mothers and babies who underwent OMFS for fetal myelomeningocele (fMMC) closure. Data from the international multicenter prospective registry of patients who underwent OMFS for fMMC closure (fMMC Consortium Registry, December 15, 2010-June 31, 2019) was used to compare fetal and maternal outcomes between anterior and posterior placental locations. The placental location for 623 patients was evenly distributed between anterior (51%) and posterior (49%) locations. Intraoperative fetal bradycardia (8.3% vs. 3.0%, $p = 0.005$) and performance of fetal resuscitation (3.6% vs. 1.0%, $p = 0.034$) occurred more frequently in cases with an anterior placenta when compared to those with a posterior placenta. Obstetric outcomes including membrane separation, placental abruption, and spontaneous rupture of membranes were not different among the 2 groups. However, thinning of the hysterotomy site (27.7% vs. 17.7%, $p = 0.008$) occurred more frequently in cases of an anterior placenta. Gestational age (GA) at delivery ($p = 0.583$) and length of stay in the neonatal intensive care unit ($p = 0.655$) were similar between the 2 groups. Fetal incision dehiscence and wound revision were not significantly different between groups. Critical clinical outcomes including fetal demise,

perinatal death, and neonatal death were all infrequent occurrences and not associated with the placental location. An anterior placental location is associated with increased risk of intraoperative fetal resuscitation and increased thinning at the hysterotomy closure site. Individual institutional experiences may have varied, but the aggregate data from the fMMC Consortium did not show a significant impact on the GA at delivery or maternal or fetal clinical outcomes.

Arroyo J et al ⁸⁸(2022) observed that ultrasound imaging is a vital component of high-quality Obstetric care. In rural and under-resourced communities, the scarcity of ultrasound imaging results in a considerable gap in the healthcare of pregnant mothers. To increase access to ultrasound in these communities, They developed a new automated diagnostic framework operated without an experienced sonographer or interpreting provider for assessment of fetal biometric measurements, fetal presentation, and placental position. This approach involves the use of a standardized volume sweep imaging (VSI) protocol based solely on external body landmarks to obtain imaging without an experienced sonographer and application of a deep learning algorithm (U-Net) for diagnostic assessment without a radiologist. Obstetric VSI ultrasound examinations were performed in Peru by an ultrasound operator with no previous ultrasound experience who underwent 8 hours of training on a standard protocol. The U-Net was trained to automatically segment the fetal head and placental location from the VSI ultrasound acquisitions to subsequently evaluate fetal biometry, fetal presentation, and placental position. In comparison to diagnostic interpretation of VSI acquisitions by a specialist, the U-Net model showed 100% agreement for fetal presentation (Cohen's κ 1 ($p < 0.0001$)) and 76.7% agreement for placental location (Cohen's κ 0.59 ($p < 0.0001$)). This corresponded to 100% sensitivity and specificity for fetal presentation and 87.5% sensitivity and 85.7% specificity for anterior

placental location. The method also achieved a low relative error of 5.6% for biparietal diameter and 7.9% for head circumference. Biometry measurements corresponded to estimated gestational age within 2 weeks of those assigned by standard of care examination with up to 89% accuracy. This system could be deployed in rural and underserved areas to provide vital information about a pregnancy without a trained sonographer or interpreting provider. The resulting increased access to ultrasound imaging and diagnosis could improve disparities in healthcare delivery in under-resourced areas.

MATERIAL AND METHODS

SOURCE OF DATA

Patients admitted in Department of OBSTETRICS AND GYNAECOLOGY in B.L.D.E (DEEMED TO BE UNIVERSITY) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura.

TYPE OF STUDY

Cross sectional study.

PERIOD OF STUDY

January 2021- April 2022.

INCLUSION CRITERIA

1. All patients with Singleton pregnancy of Gestational age >28 weeks.
2. Patients who gave informed and written consent in accordance with declaration of Helsinki.

EXCLUSION CRITERIA

1. Gestational age <28 weeks
2. Multiple pregnancies
3. Uterine anomalies
4. Patient who did not giving informed and written consent

SAMPLE SIZE CALCULATION

With anticipated Proportion of placental insufficiency in abnormally located placenta 2.5% ⁽¹⁰⁾, the study would require a sample size of 1301 patients with a 99% level of confidence and 1% absolute precision. Formula used

$$n = \frac{z^2 p q}{d^2}$$

$$d^2 =$$

Where Z= Z statistic at α level of significance $d^2 =$

Absolute error

P= Proportion rate

$$q = 100 - p$$

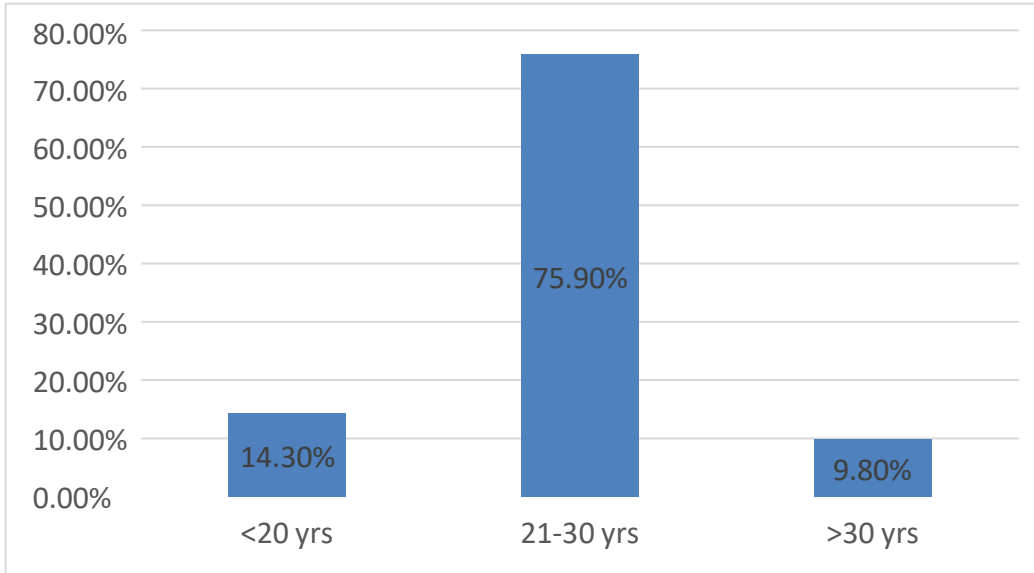
- **Statistical Analysis**
- The data obtained was entered in a Microsoft Excel sheet, and statistical analysis was performed using statistical package for the social sciences (Version 20).
- Results was presented as Mean (Median) \pm SD, counts and percentages and diagrams.
- For normally distributed continuous variables was compared using independent t- test. For not normally distributed variables Mann Whitney U test was used.
- Categorical variables were compared using the Chi-square test.
- $P < 0.05$ was considered statistically significant. All statistical tests will perform two-tailed.

METHODOLOGY

1. All patients who delivered in B.L.D.E (DEEMED TO BE UNIVERSITY) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura were included in the study.
2. Consent was taken in accordance with declaration of Helsinki.
3. For patients who were enrolled in our study, USG was done to look for localization of placenta on admission.
4. Maternal outcomes like mode of delivery, Gravid status when patient got admitted for delivery, complications like Pregnancy Induced Hypertension, Abruption Placenta. Foetal Growth Restriction, Oligohydramnios, Polyhydramnios, Premature Rupture of Membranes, Preterm Delivery and Perinatal outcome like birth weight, APGAR score, respiratory distress syndrome, hypoxic ischemic encephalopathy, NICU admission, intrauterine death neonatal death was noted.
5. Placenta was sent for histopathology.

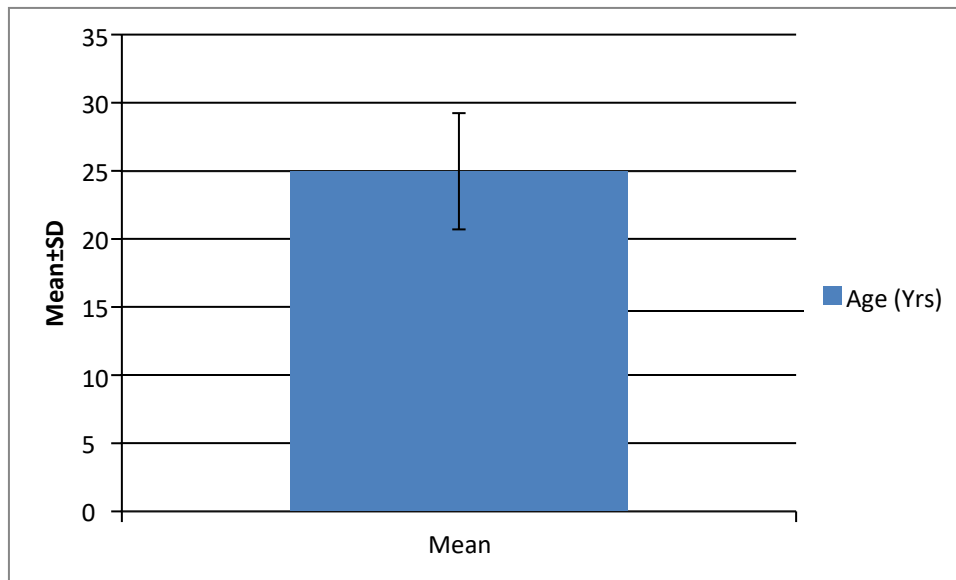
RESULTS

Diagram 1: Distribution of Age in Group



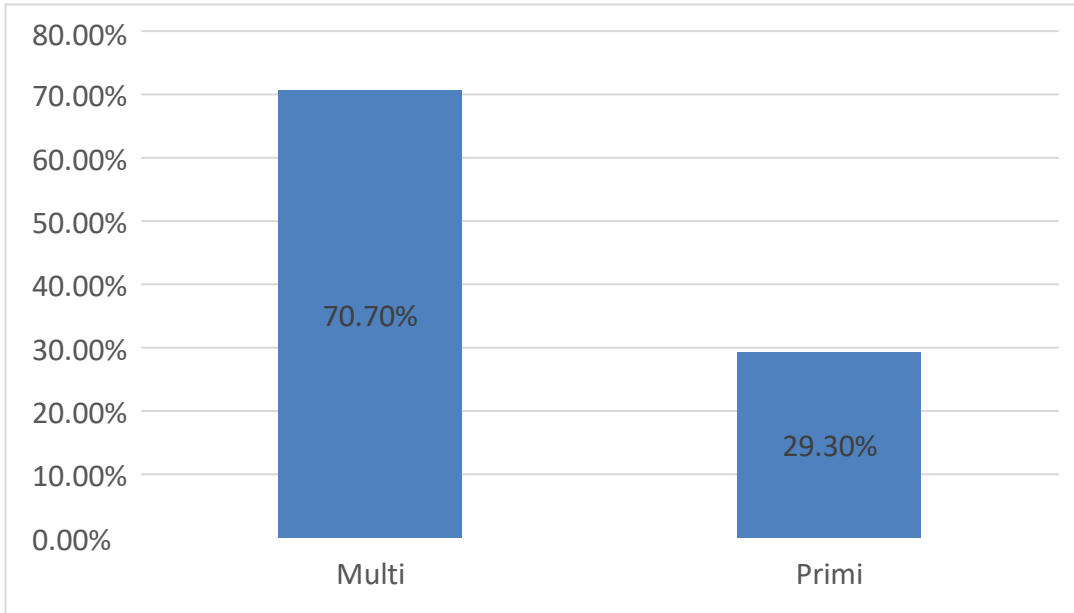
In our study, 186 (14.3%) patients were <20years of age, 988 (75.9%) patients were 21-30years of age and 127 (9.8%) patients were ≥ 31 years of age.

Diagram 2: Distribution of mean Age (Yrs)



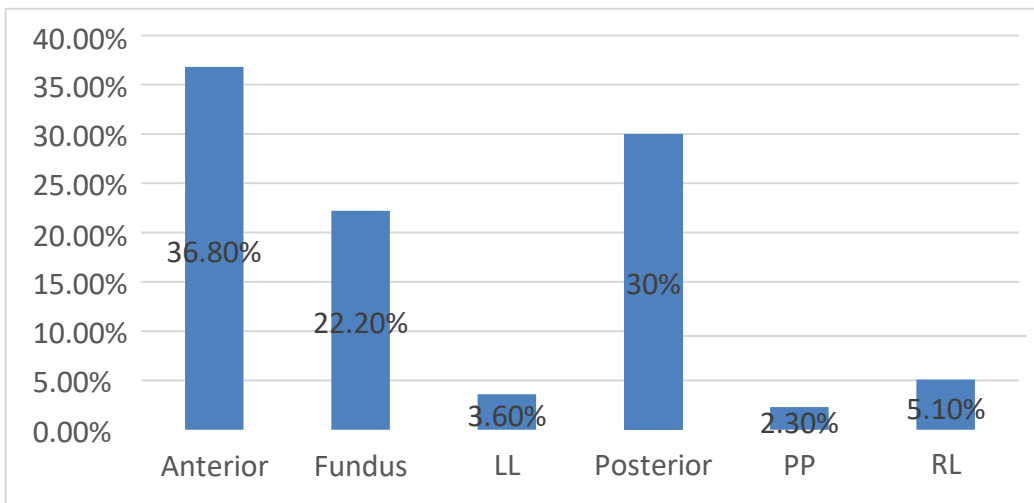
In above table showed that the mean Age (Years) (mean \pm S.D) of patients was 24.9570 ± 4.2701

Diagram 4: Distribution of Obstetric History



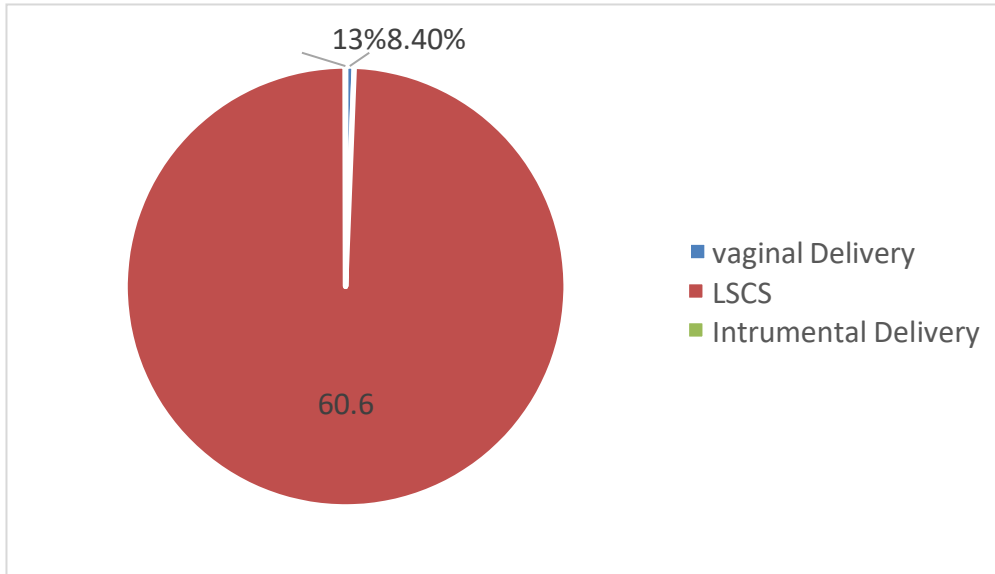
In our study, 920 (70.7%) patients had Multi and 381 (29.3%) patients had Primi in Obstetric History.

Diagram 5: Distribution of Placental Location



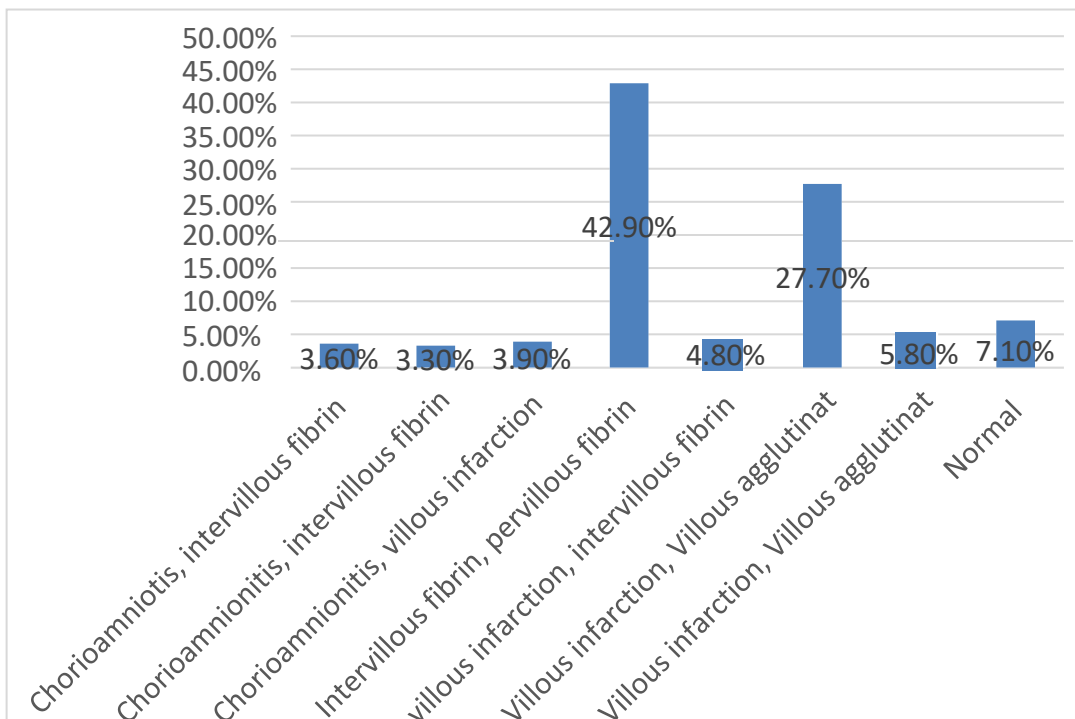
In our study, 479 (36.8%) patients had Anterior, 289 (22.2%) patients had Fundus, 47 (3.6%) patients had LL, 390 (30.0%) patients had Posterior, 30 (2.3%) patients had PP and 66 (5.1%) patients had RL.

Diagram 14: Distribution of MODE OF DELIVERY



In our study, 778 (60.6%) patients had LSCS and 499 (38.4%) patients had Vaginal Delivery and 1% had Instrumental Delivery.

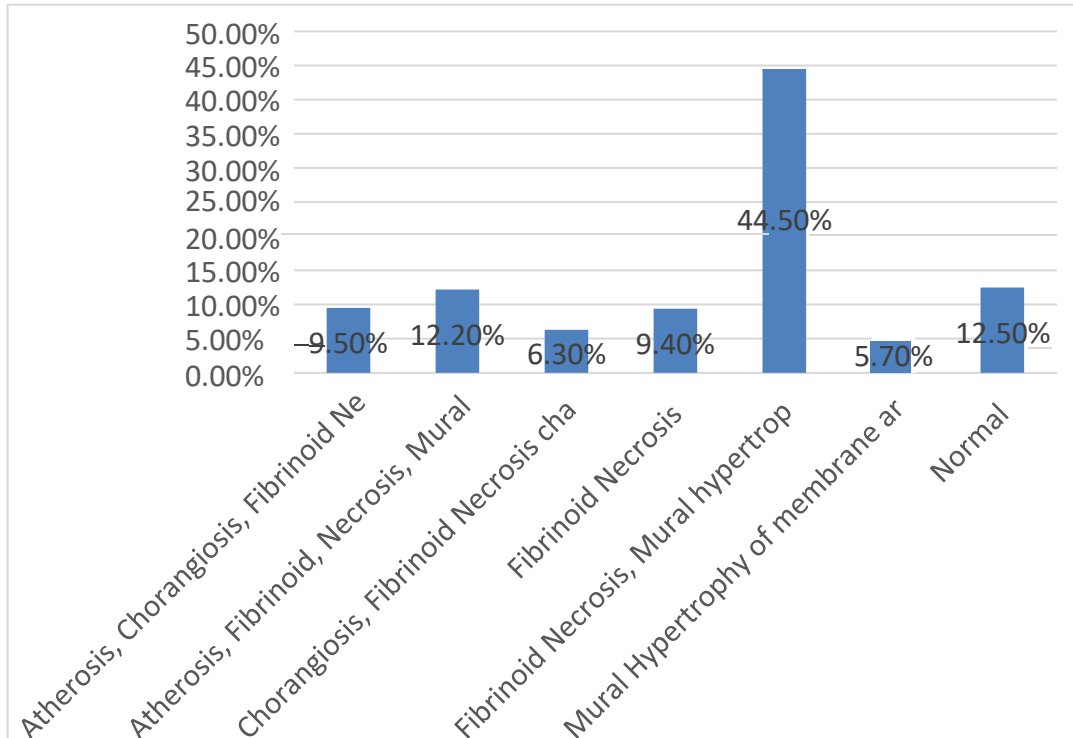
Diagram 18: Distribution of HPR of Maternal Surface



Among all the placentas sent for HPR 47(3.6%) showed Chorioamnionitis with intervillous fibrin, 42(3.3%) had chorioamnionitis, Intervillous Fibrin and Peri-villous Fibrin, 51(3.9%)

placentas had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin, 559(42.9%) Placents had Intervillous Fibrin and Peri-villous Fibrin, 63(4.8%) placentas had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin, 360(27.7%) placentas had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Peri-villous Fibrin, 75(5.8%) placentas had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus and 92(7.1%) patients had Normal Maternal Surface of placenta.

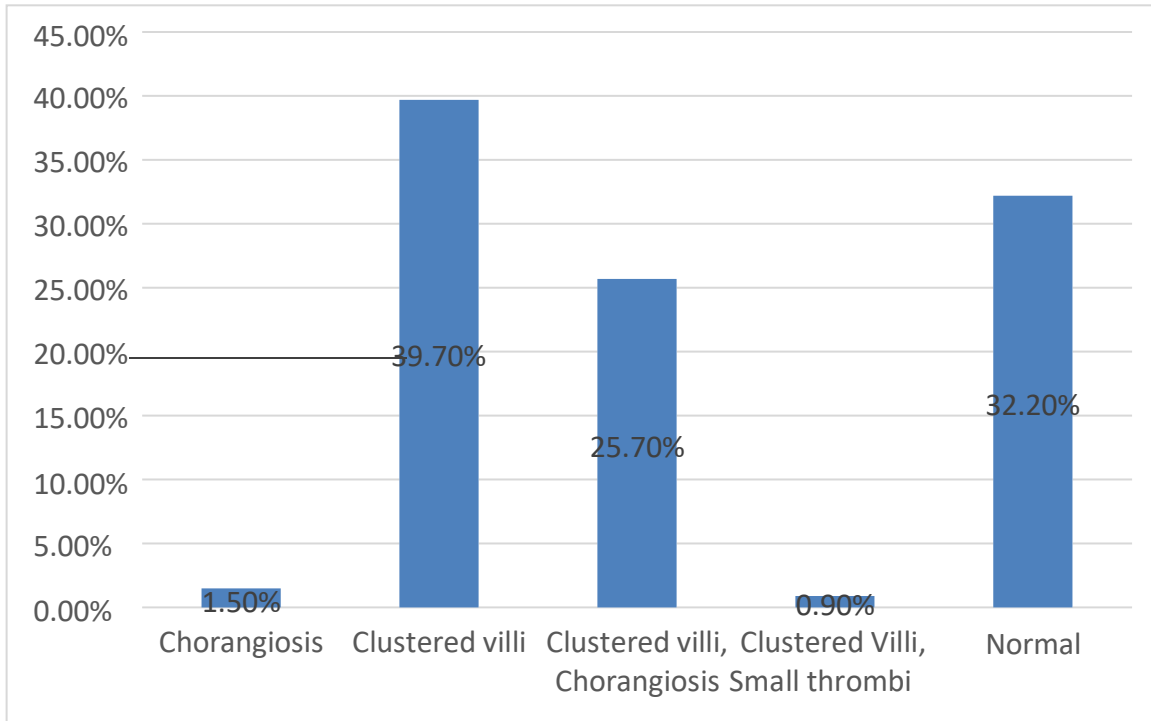
Diagram 19: Distribution of Decidual Arteriopathy



Among all the placentas sent for HPR Atherosclerosis, Chorangiomas, Fibrinoid Necrosis changes are seen in 123(9.5%) placentas, 158(12.2%) had Atherosclerosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane Arterioles, 82(6.3%) patient’s placentas showed Chorangiomas, Fibrinoid Necrosis, 122(9.4%) placentas showed fibrinoid Necrosis, 579(44.5%) placentas had Fibrinoid Necrosis,

Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 74(5.7%) placentas had Mural Hypertrophy of Membrane Arterioles and 162(12.5%) patients had normal decidual arteries in Placenta.

Table: Distribution of HPR of Foetal Surface



Out of 1301 placentas which were sent for HPR 19(1.5%) placentas showed Chorangiomas, 516(39.7%) placentas showed Clustered Villi, 335(25.7%) placentas showed Clustered Villi and Chorangiomas, 12(0.9%) placentas showed Clustered Villi, Small Thrombi in Blood vessels and 419(32.2%) Placentas showed Normal Fetal Surface.

Table 1: Association Between Fundal placenta and Mild PE

			Fundus		Total	Chi-square value	p-value
			NO	YES			
MILD	NO	Count	992	282	1274	0.857	0.354
		% within Fundus	98.1%	97.2%	97.9%		
	YES	Count	19	8	27		
		% within Fundus	1.9%	2.8%	2.1%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal placenta and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 2: Association Between Right Lateral placenta and Mild PE

			Right Lateral		Total	Chi- square value	p-value
			NO	YES			
MILD	NO	Count	1209	65	1274	0.107	0.743
		% within RL	97.9%	98.5%	97.9%		
	YES	Count	26	1	27		
		% within RL	2.1%	1.5%	2.1%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral placenta and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 3: Association Between Left Lateral placenta and Mild PE

			Left Lateral		Total	Chi – square value	p-value
			NO	YES			
MILD	NO	Count	1225	49	1274	1.079	0.299
		% LL	97.8%	100.0%	97.9%		
	YES	Count	27	0	27		
		% LL	2.2%	0.0%	2.1%		
Total		Count	1252	49	1301		
		% LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral placenta and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 4: Association Between Posterior placenta and Mild PE

			Posterior		Total	Chi- square value	p- value
			NO	YES			
MILD	NO	Count	800	474	1274	0.000	0.986
		% within Posterior	97.9%	97.9%	97.9%		
	YES	Count	17	10	27		
		% within Posterior	2.1%	2.1%	2.1%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior placenta and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 5: Association Between Anterior placenta and Mild PE

			Anterior		Total	Chi- square value	p- value
			NO	YES			
MILD	NO	Count	720	554	1274	0.081	0.771
		% within Anterior	97.8%	98.1%	97.9%		
	YES	Count	16	11	27		
		% within Anterior	2.2%	1.9%	2.1%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior placenta and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 6: Association Between Placenta Previa and Mild PE

			Placenta Previa		Total	Chi- square value	p- value
			NO	YES			
MILD	NO	Count	1244	30	1274	0.651	0.420
		% within PP	97.9%	100.0%	97.9%		
	YES	Count	27	0	27		
		% within PP	2.1%	0.0%	2.1%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Mild PE is shown in table, was not significant statistically ($P>0.05$).

Table 7: Association Between Fundal placenta and Severe PE

			Fundus		Total	Chi- square value	p-value
			NO	YES			
SEVERE	NO	Count	914	277	1191	7.608	0.006*
		% within Fundus	90.4%	95.5%	91.5%		
	YES	Count	97	13	110		
		% within Fundus	9.6%	4.5%	8.5%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Fundal placenta and Severe PE is shown in table, and there was positive and was significant statistically ($P < 0.05$).

Table 8: Association Between Right Lateral placenta and Severe PE

			Right Lateral		Total	Chi- Square value	p-value
			NO	YES			
SEVERE	NO	Count	1131	60	1191	0.036	0.849
		% within RL	91.6%	90.9%	91.5%		
	YES	Count	104	6	110		
		% within RL	8.4%	9.1%	8.5%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral placenta and Severe PE is shown in table, was not significant statistically ($P > 0.05$).

Table 9: Association Between Left Lateral placenta and Severe PE

			Left Lateral		Total	Chi-Square value	p- value
			NO	YES			
SEVERE	NO	Count	1150	41	1191	4.076	0.043*
		% within LL	91.9%	83.7%	91.5%		
	YES	Count	102	8	110		
		% within LL	8.1%	16.3%	8.5%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Left Lateral placenta and Severe PE is shown in table, and there was positive and was significant statistically ($P < 0.05$).

Table 10: Association Between Posterior placenta and Severe PE

			Posterior		Total	Chi – Square value	p-value
			NO	YES			
SEVERE	NO	Count	746	445	1191	0.157	0.692
		% within Posterior	91.3%	91.9%	91.5%		
	YES	Count	71	39	110		
		% within Posterior	8.7%	8.1%	8.5%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior placenta and Severe PE is shown in table, was not significant statistically ($P > 0.05$).

Table 11: Association Between Anterior placenta and Severe PE

			Anterior		Total	Chi-Square value	p-value
			NO	YES			
SEVERE	NO	Count	676	515	1191	0.201	0.654
		% within Anterior	91.8%	91.2%	91.5%		
	YES	Count	60	50	110		
		% within Anterior	8.2%	8.8%	8.5%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior placenta and Severe PE is shown in table, was not significant statistically ($P>0.05$).

Table 12: Association Between Placenta Previa and Severe PE

			Placenta Previa		Total	Chi-square value	p-value
			NO	YES			
SEVERE	NO	Count	1164	27	1191	0.095	0.758
		% within PP	91.6%	90.0%	91.5%		
	YES	Count	107	3	110		
		% within PP	8.4%	10.0%	8.5%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Severe PE is shown in table, was not significant statistically ($P>0.05$).

Table 13: Association Between Fundal placenta and Eclampsia

			Fundus		Total	Chi-Square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	954	278	1232	1.010	0.315
		% within Fundus	94.4%	95.9%	94.7%		
	YES	Count	57	12	69		
		% within Fundus	5.6%	4.1%	5.3%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal placenta and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 14: Association Between Right Lateral placenta and Eclampsia

			Right Lateral		Total	Chi-Square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	1172	60	1232	1.986	0.159
		% within RL	94.9%	90.9%	94.7%		
	YES	Count	63	6	69		
		% within RL	5.1%	9.1%	5.3%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral placenta and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 15: Association Between Left Lateral placenta and Eclampsia

			Left Lateral		Total	Chi- square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	1183	49	1232	2.852	0.091
		% within LL	94.5%	100.0%	94.7%		
	YES	Count	69	0	69		
		% within LL	5.5%	0.0%	5.3%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral placenta and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 16: Association Between Posterior placenta and Eclampsia

			Posterior		Total	Chi- square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	775	457	1232	0.116	0.733
		% within Posterior	94.9%	94.4%	94.7%		
	YES	Count	42	27	69		
		% within Posterior	5.1%	5.6%	5.3%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior placenta and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 17: Association Between Anterior placenta and Eclampsia

			Anterior		Total	Chi-square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	694	538	1232	0.548	0.459
		% within Anterior	94.3%	95.2%	94.7%		
	YES	Count	42	27	69		
		% within Anterior	5.7%	4.8%	5.3%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistical Not Significant							

The association between Anterior placenta and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 18: Association Between Placenta Previa and Eclampsia

			Placenta Previa		Total	Chi-Square value	p-value
			NO	YES			
ECLAMPSIA	NO	Count	1204	28	1232	0.114	0.736
		% within PP	94.7%	93.3%	94.7%		
	YES	Count	67	2	69		
		% within PP	5.3%	6.7%	5.3%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Eclampsia is shown in table, was not significant statistically ($P>0.05$).

Table 19: Association Between Fundal location of placenta and Abruption

			Fundus		Total	Chi-Square value	p- value
			NO	YES			
ABRUPTION	NO	Count	977	285	1262	2.082	0.149
		% within Fundus	96.6%	98.3%	97.0%		
	YES	Count	34	5	39		
		% within Fundus	3.4%	1.7%	3.0%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Location of placenta and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 20: Association Between Right Lateral location of placenta and Abruption

			Right Lateral		Total	Chi-square value	p-value
			NO	YES			
ABRUPTION	NO	Count	1198	64	1262	0.000	0.987
		% within RL	97.0%	97.0%	97.0%		
	YES	Count	37	2	39		
		% within RL	3.0%	3.0%	3.0%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Location of placenta and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 21: Association Between Left Lateral location of placenta and Abruption

			Left Lateral		Total	Chi-square value	p- value
			NO	YES			
ABRUPTION	NO	Count	1215	47	1262	0.206	0.650
		% within LL	97.0%	95.9%	97.0%		
	YES	Count	37	2	39		
		% within LL	3.0%	4.1%	3.0%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Location of placenta and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 22: Association Between Posterior placenta and Abruption

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
ABRUPTION	NO	Count	794	468	1262	0.252	0.616
		% within Posterior	97.2%	96.7%	97.0%		
	YES	Count	23	16	39		
		% within Posterior	2.8%	3.3%	3.0%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior placenta and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 23: Association Between Anterior placenta and Abruption

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
ABRUPTION	NO	Count	713	549	1262	0.094	0.759
		% within Anterior	96.9%	97.2%	97.0%		
	YES	Count	23	16	39		
		% within Anterior	3.1%	2.8%	3.0%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior placenta and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 24: Association Between Placenta Previa and Abruption

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
ABRUPTION	NO	Count	1232	30	1262	0.949	0.330
		% within PP	96.9%	100.0%	97.0%		
	YES	Count	39	0	39		
		% within PP	3.1%	0.0%	3.0%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Abruption is shown in table, was not significant statistically ($P>0.05$).

Table 25: Association Between Fundal placenta and FGR

			Fundus		Total	Chi-square value	P- value
			NO	YES			
FGR	NO	Count	980	282	1262	0.073	0.787
		% within Fundus	96.9%	97.2%	97.0%		
	YES	Count	31	8	39		
		% within Fundus	3.1%	2.8%	3.0%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal placenta and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 26: Association Between Right Lateral placenta and FGR

			Right Lateral		Total	Chi-square value	P- value
			NO	YES			
FGR	NO	Count	1196	66	1262	2.149	0.143
		% within RL	96.8%	100.0%	97.0%		
	YES	Count	39	0	39		
		% within RL	3.2%	0.0%	3.0%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral placenta and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 27: Association Between Left Lateral placenta and FGR

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
FGR	NO	Count	1216	46	1262	1.710	0.191
		% within LL	97.1%	93.9%	97.0%		
	YES	Count	36	3	39		
		% within LL	2.9%	6.1%	3.0%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral placenta and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 28: Association Between Posterior placenta and FGR

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
FGR	NO	Count	792	470	1262	0.029	0.864
		% within Posterior	96.9%	97.1%	97.0%		
	YES	Count	25	14	39		
		% within Posterior	3.1%	2.9%	3.0%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior placenta and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 29: Association Between Anterior placenta and FGR

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
FGR	NO	Count	716	546	1262	0.458	0.499
		% within Anterior	97.3%	96.6%	97.0%		
	YES	Count	20	19	39		
		% within Anterior	2.7%	3.4%	3.0%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statically Not Significant							

The association between Anterior placenta and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 30: Association Between Placenta Previa and FGR

			Placenta Previa		Total	Chi – Square value	P – value
			NO	YES			
FGR	NO	Count	1232	30	1262	0.949	0.330
		% within PP	96.9%	100.0%	97.0%		
	YES	Count	39	0	39		
		% within PP	3.1%	0.0%	3.0%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statically Not Significant							

The association between Placenta Previa and FGR is shown in table, was not significant statistically ($P>0.05$).

Table 31: Association Between Fundal Placenta and Oligohydramnios

			Fundus		Total	Chi – Square value	P - value
			NO	YES			
OLIGOHYDRAMNIOS	NO	Count	826	242	1068	0.468	0.494
		% within Fundus	81.7%	83.4%	82.1%		
	YES	Count	185	48	233		
		% within Fundus	18.3%	16.6%	17.9%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Oligohydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 32: Association Between Right Lateral Placenta and Oligohydramnios

			Right Lateral		Total	Chi – Square value	P – value
			NO	YES			
OLIGO	NO	Count	1015	53	1068	0.151	0.697
		% within RL	82.2%	80.3%	82.1%		
	YES	Count	220	13	233		
		% within RL	17.8%	19.7%	17.9%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Oligohydramnios is shown in table, was statistically not significant ($P>0.05$).

Table 33: Association Between Left Lateral Placenta and Oligohydramnios

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
OLIGO	NO	Count	1027	41	1068	0.087	0.768
		% within LL	82.0%	83.7%	82.1%		
	YES	Count	225	8	233		
		% within LL	18.0%	16.3%	17.9%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Oligohydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 34: Association Between Posterior Placenta and Oligohydramnios

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
OLIGO	NO	Count	664	404	1068	0.999	0.318
		% within Posterior	81.3%	83.5%	82.1%		
	YES	Count	153	80	233		
		% within Posterior	18.7%	16.5%	17.9%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Oligohydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 35: Association Between Anterior Placenta and Oligohydramnios

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
OLIGO	NO	Count	609	459	1068	0.493	0.483
		% within Anterior	82.7%	81.2%	82.1%		
	YES	Count	127	106	233		
		% within Anterior	17.3%	18.8%	17.9%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Oligohydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 36: Association Between Placenta Previa and Oligohydramnios

			Placenta Previa		Total	Chi – Square value	P- value
			NO	YES			
OLIGO	NO	Count	1041	27	1068	1.307	0.253
		% within PP	81.9%	90.0%	82.1%		
	YES	Count	230	3	233		
		% within PP	18.1%	10.0%	17.9%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Oligohydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 37: Association Between Fundal Placenta and Polyhydramnios

			Fundus		Total	Chi – Square value	P- value
			NO	YES			
POLY	NO	Count	1003	289	1292	0.654	0.419
		% with in Fundus	99.2%	99.7%	99.3%		
	YES	Count	8	1	9		
		% with in Fundus	0.8%	0.3%	0.7%		
Total		Count	1011	290	1301		
		% with in Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Polyhydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 38: Association Between Right Lateral Placenta and Polyhydramnios

			Right Lateral		Total	Chi – Square value	P - value
			NO	YES			
POLY	NO	Count	1227	65	1292	0.686	0.407
		% within RL	99.4%	98.5%	99.3%		
	YES	Count	8	1	9		
		% within RL	0.6%	1.5%	0.7%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Polyhydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 39: Association Between Left Lateral Placenta and Polyhydramnios

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
POLY	NO	Count	1244	48	1292	1.349	0.245
		% with in LL	99.4%	98.0%	99.3%		
	YES	Count	8	1	9		
		% within LL	0.6%	2.0%	0.7%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Polyhydramnios is shown in table, was not significant statistically ($P > 0.05$).

Table 40: Association Between Posterior Placenta and Polyhydramnios

			Posterior		Total	Chi- square value	P - value
			NO	YES			
POLY	NO	Count	812	480	1292	0.203	0.652
		% within Posterior	99.4%	99.2%	99.3%		
	YES	Count	5	4	9		
		% within Posterior	0.6%	0.8%	0.7%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Polyhydramnios is shown in table, was not significant statistically ($P > 0.05$).

Table 41: Association Between Anterior Placenta and Polyhydramnios

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
POLY	NO	Count	729	563	1292	1.659	0.198
		% within Anterior	99.0%	99.6%	99.3%		
	YES	Count	7	2	9		
		% within Anterior	1.0%	0.4%	0.7%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Polyhydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 42: Association Between Placenta Previa and Polyhydramnios

			Placenta Previa		Total	Chi – Square value	P - value
			NO	YES			
POLY	NO	Count	1262	30	1292	0.214	0.644
		% within PP	99.3%	100.0%	99.3%		
	YES	Count	9	0	9		
		% within PP	0.7%	0.0%	0.7%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Polyhydramnios is shown in table, was not significant statistically ($P>0.05$).

Table 43: Association Between Fundal Placenta and Preterm

			Fundus		Total	Chi – square value	P - value
			NO	YES			
PRETERM	NO	Count	777	218	995	0.355	0.552
		% within Fundus	76.9%	75.2%	76.5%		
	YES	Count	234	72	306		
		% within Fundus	23.1%	24.8%	23.5%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 44: Association Between Right Lateral Placenta and Preterm

			Right Lateral		Total	Chi – Square value	P - value
			NO	YES			
PRETERM	NO	Count	940	55	995	1.816	0.178
		% within RL	76.1%	83.3%	76.5%		
	YES	Count	295	11	306		
		% within RL	23.9%	16.7%	23.5%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 45: Association Between Left Lateral Placenta and Preterm

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
PRETERM	NO	Count	961	34	995	1.424	0.233
		% within LL	76.8%	69.4%	76.5%		
	YES	Count	291	15	306		
		% within LL	23.2%	30.6%	23.5%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 46: Association Between Posterior Placenta and Preterm

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
PRETERM	NO	Count	632	363	995	0.938	0.333
		% within Posterior	77.4%	75.0%	76.5%		
	YES	Count	185	121	306		
		% within Posterior	22.6%	25.0%	23.5%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 47: Association Between Anterior Placenta and Preterm

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
PRETERM	NO	Count	550	445	995	2.890	0.089
		% within Anterior	74.7%	78.8%	76.5%		
	YES	Count	186	120	306		
		% within Anterior	25.3%	21.2%	23.5%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 48: Association Between Placenta Previa and Preterm

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
PRETERM	NO	Count	976	19	995	2.950	0.086
		% within PP	76.8%	63.3%	76.5%		
	YES	Count	295	11	306		
		% within PP	23.2%	36.7%	23.5%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Preterm is shown in table, was not significant statistically ($P>0.05$).

Table 49: Association Between Fundal Placenta and PROM

			Fundus		Total	Chi – Square value	P - value
			NO	YES			
PROM	NO	Count	946	261	1207	4.287	0.038*
		% within Fundus	93.6%	90.0%	92.8%		
	YES	Count	65	29	94		
		% within Fundus	6.4%	10.0%	7.2%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Fundal Placenta and PROM is shown in table, and was positive and was significant statistically ($P < 0.05$).

Table 50: Association Between Right Lateral Placenta and PROM

			Right Lateral		Total	Chi – Square value	P - value
			NO	YES			
PROM	NO	Count	1147	60	1207	0.361	0.548
		% within RL	92.9%	90.9%	92.8%		
	YES	Count	88	6	94		
		% within RL	7.1%	9.1%	7.2%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral and PROM is shown in table, and was not significant statistically ($P > 0.05$).

Table 51: Association Between Left Lateral Placenta and PROM

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
PROM	NO	Count	1161	46	1207	0.092	0.761
		% within LL	92.7%	93.9%	92.8%		
	YES	Count	91	3	94		
		% within LL	7.3%	6.1%	7.2%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and PROM is shown in table, was not significant statistically ($P>0.05$).

Table 52: Association Between Posterior Placenta and PROM

			Posterior		Total	Chi – square value	P – value
			NO	YES			
PROM	NO	Count	755	452	1207	0.433	0.511
		% within Posterior	92.4%	93.4%	92.8%		
	YES	Count	62	32	94		
		% within Posterior	7.6%	6.6%	7.2%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and PROM is shown in table, was not significant statistically ($P>0.05$).

Table 53: Association Between Anterior Placenta and PROM

			Anterior		Total	Chi – square value	P – value
			NO	YES			
PROM	NO	Count	687	520	1207	0.815	0.367
		% within Anterior	93.3%	92.0%	92.8%		
	YES	Count	49	45	94		
		% within Anterior	6.7%	8.0%	7.2%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and PROM is shown in table, was not significant statistically ($P>0.05$).

Table 54: Association Between Placenta Previa and PROM

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
PROM	NO	Count	1178	29	1207	0.694	0.405
		% within PP	92.7%	96.7%	92.8%		
	YES	Count	93	1	94		
		% within PP	7.3%	3.3%	7.2%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and PROM is shown in table, was not significant statistically ($P>0.05$).

Table 55: Association Between Fundal Placenta and Vaginal Delivery

			Fundus		Total	Chi – Square value	P - value
			NO	YES			
VAGINAL	NO	Count	631	170	801	1.370	0.242
		% within Fundus	62.4%	58.6%	61.6%		
	YES	Count	380	120	500		
		% within Fundus	37.6%	41.4%	38.4%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Vaginal Delivery is shown in table, was not significant statistically ($P > 0.05$).

Table 56: Association Between Right Lateral Placenta and Vaginal Delivery

			Right Lateral		Total	Chi – square value	P – value
			NO	YES			
VAGINAL	NO	Count	772	29	801	9.132	0.003*
		% within RL	62.5%	43.9%	61.6%		
	YES	Count	463	37	500		
		% within RL	37.5%	56.1%	38.4%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Right Lateral Placenta and Vaginal Delivery is shown in table, and was Positive and significant statistically ($P < 0.05$).

Table 57: Association Between Left Lateral Placenta and Vaginal Delivery

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
VAGINAL	NO	Count	776	25	801	2.394	0.122
		% within LL	62.0%	51.0%	61.6%		
	YES	Count	476	24	500		
		% within LL	38.0%	49.0%	38.4%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Vaginal Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 58: Association Between Posterior Placenta and Vaginal Delivery

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
VAGINAL	NO	Count	493	308	801	1.393	0.238
		% within Posterior	60.3%	63.6%	61.6%		
	YES	Count	324	176	500		
		% within Posterior	39.7%	36.4%	38.4%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Vaginal Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 59: Association Between Anterior Placenta and Vaginal Delivery

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
VAGINAL	NO	Count	446	355	801	0.674	0.412
		% within Anterior	60.6%	62.8%	61.6%		
	YES	Count	290	210	500		
		% within Anterior	39.4%	37.2%	38.4%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Vaginal Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 60: Association Between Placenta Previa and Vaginal Delivery

			Placenta Previa		Total	Chi – Square value	P - value
			NO	YES			
VAGINAL	NO	Count	780	21	801	0.923	0.337
		% within PP	61.4%	70.0%	61.6%		
	YES	Count	491	9	500		
		% within PP	38.6%	30.0%	38.4%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Vaginal Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 61: Association Between Fundal Placenta and LSCS

			Fundus		Total	Chi – square value	P - value
			NO	YES			
LSCS	NO	Count	391	122	513	1.087	0.297
		% within Fundus	38.7%	42.1%	39.4%		
	YES	Count	620	168	788		
		% within Fundus	61.3%	57.9%	60.6%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and LSCS is shown in table, was not significant statistically ($P>0.05$).

Table 62: Association Between Right Lateral Placenta and LSCS

			Right Lateral		Total	Chi- Square value	P - value
			NO	YES			
LSCS	NO	Count	476	37	513	8.050	0.005*
		% within RL	38.5%	56.1%	39.4%		
	YES	Count	759	29	788		
		% within RL	61.5%	43.9%	60.6%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Right Lateral Placenta and LSCS is shown in table, and was positive and was significant statistically ($P<0.05$).

Table 63: Association Between Left Lateral Placenta and LSCS

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
LSCS	NO	Count	488	25	513	2.863	0.091
		% within LL	39.0%	51.0%	39.4%		
	YES	Count	764	24	788		
		% within LL	61.0%	49.0%	60.6%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and LSCS is shown in table, was not significant statistically ($P>0.05$).

Table 64: Association Between Posterior Placenta and LSCS

			Posterior		Total	Chi – square value	P - value
			NO	YES			
LSCS	NO	Count	333	180	513	1.621	0.203
		% within Posterior	40.8%	37.2%	39.4%		
	YES	Count	484	304	788		
		% within Posterior	59.2%	62.8%	60.6%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and LSCS is shown in table, was not significant statistically ($P>0.05$).

Table 65: Association Between Anterior Placenta and LSCS

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
LSCS	NO	Count	296	217	513	0.439	0.508
		% within Anterior	40.2%	38.4%	39.4%		
	YES	Count	440	348	788		
		% within Anterior	59.8%	61.6%	60.6%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and LSCS is shown in table, was not significant statistically ($P>0.05$).

Table 66: Association Between Placenta Previa and LSCS

			Placenta Previa		Total	Chi – Square value	P - value
			NO	YES			
LSCS	NO	Count	504	9	513	1.144	0.285
		% within PP	39.7%	30.0%	39.4%		
	YES	Count	767	21	788		
		% within PP	60.3%	70.0%	60.6%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and LSCS is shown in table, was not significant statistically ($P>0.05$).

Table 67: Association Between Fundal Placenta and Instrumental Delivery

			Fundus		Total	Chi – Square value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	1000	288	1288	0.362	0.548
		% within Fundus	98.9%	99.3%	99.0%		
	YES	Count	11	2	13		
		% within Fundus	1.1%	0.7%	1.0%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 68: Association Between Right Lateral Placenta and Instrumental Delivery

			Right Lateral		Total	Chi – Square Value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	1222	66	1288	0.702	0.402
		% within RL	98.9%	100.0%	99.0%		
	YES	Count	13	0	13		
		% within RL	1.1%	0.0%	1.0%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 69: Association Between Left Lateral Placenta and Instrumental Delivery

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	1240	48	1288	0.558	0.455
		% within LL	99.0%	98.0%	99.0%		
	YES	Count	12	1	13		
		% within LL	1.0%	2.0%	1.0%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 70: Association Between Posterior Placenta and Instrumental Delivery

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	808	480	1288	0.233	0.630
		% within Posterior	98.9%	99.2%	99.0%		
	YES	Count	9	4	13		
		% within Posterior	1.1%	0.8%	1.0%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 71: Association Between Anterior Placenta and Instrumental Delivery

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	730	558	1288	0.580	0.446
		% within Anterior	99.2%	98.8%	99.0%		
	YES	Count	6	7	13		
		% within Anterior	0.8%	1.2%	1.0%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 72: Association Between Placenta Previa and Instrumental Delivery

			Placenta Previa		Total	Chi – Square value	P - value
			NO	YES			
INSTRUMENTAL	NO	Count	1258	30	1288	0.310	0.578
		% within PP	99.0%	100.0%	99.0%		
	YES	Count	13	0	13		
		% within PP	1.0%	0.0%	1.0%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Instrumental Delivery is shown in table, was not significant statistically ($P>0.05$).

Table 73: Association Between Fundus Placenta and NICU admission

			Fundus		Total	Chi – square value	P - value
			NO	YES			
NICU	NO	Count	469	148	617	1.950	0.163
		% within Fundus	46.4%	51.0%	47.4%		
	YES	Count	542	142	684		
		% within Fundus	53.6%	49.0%	52.6%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundus Placenta and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 74: Association Between Right Lateral Placenta and NICU admission

			Right Lateral		Total	Chi – square value	P - value
			NO	YES			
NICU	NO	Count	586	31	617	0.006	0.939
		% within RL	47.4%	47.0%	47.4%		
	YES	Count	649	35	684		
		% within RL	52.6%	53.0%	52.6%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 75: Association Between Left Lateral Placenta and NICU admission

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
NICU	NO	Count	589	28	617	1.928	0.165
		% within LL	47.0%	57.1%	47.4%		
	YES	Count	663	21	684		
		% within LL	53.0%	42.9%	52.6%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 76: Association Between Posterior Placenta and NICU admission

			Posterior		Total	Chi – square value	P - value
			NO	YES			
NICU	NO	Count	386	231	617	0.028	0.867
		% within Posterior	47.2%	47.7%	47.4%		
	YES	Count	431	253	684		
		% within Posterior	52.8%	52.3%	52.6%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 77: Association Between Anterior Placenta and NICU admission

			Anterior		Total	Chi – square value	P - value
			NO	YES			
NICU	NO	Count	354	263	617	0.308	0.579
		% within Anterior	48.1%	46.5%	47.4%		
	YES	Count	382	302	684		
		% within Anterior	51.9%	53.5%	52.6%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 78: Association Between Placenta Previa and NICU admission

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
NICU	NO	Count	608	9	617	3.740	0.053
		% within PP	47.8%	30.0%	47.4%		
	YES	Count	663	21	684		
		% within PP	52.2%	70.0%	52.6%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and NICU admission is shown in table, was not significant statistically ($P>0.05$).

Table 79: Association Between Fundal Placenta and Mother-side

			Fundus		Total	Chi – square value	P -value
			NO	YES			
MOTHERSIDE	NO	Count	582	155	737	1.557	0.212
		% within Fundus	57.6%	53.4%	56.6%		
	YES	Count	429	135	564		
		% within Fundus	42.4%	46.6%	43.4%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 80: Association Between Right Lateral Placenta and Mother-side

			Right Lateral		Total	Chi – square value	P - value
			NO	YES			
MOTHERSIDE	NO	Count	699	38	737	0.024	0.876
		% within RL	56.6%	57.6%	56.6%		
	YES	Count	536	28	564		
		% within RL	43.4%	42.4%	43.4%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 81: Association Between Left Lateral Placenta and Mother-side

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
MOTHERSIDE	NO	Count	713	24	737	1.219	0.269
		% LL	56.9%	49.0%	56.6%		
	YES	Count	539	25	564		
		% LL	43.1%	51.0%	43.4%		
Total		Count	1252	49	1301		
		% LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 82: Association Between Posterior Placenta and Mother-side

			Posterior		Total	Chi – square value	P - value
			NO	YES			
MOTHERSIDE	NO	Count	464	273	737	0.019	0.891
		% within Posterior	56.8%	56.4%	56.6%		
	YES	Count	353	211	564		
		% within Posterior	43.2%	43.6%	43.4%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 83: Association Between Anterior Placenta and Mother-side

			Anterior		Total	Chi – square value	P - value
			NO	YES			
MOTHERSIDE	NO	Count	414	323	737	0.110	0.740
		% within Anterior	56.2%	57.2%	56.6%		
	YES	Count	322	242	564		
		% within Anterior	43.8%	42.8%	43.4%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 84: Association Between Placenta Previa and Mother-side

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
MOTHERSIDE	NO	Count	716	21	737	2.229	0.135
		% within PP	56.3%	70.0%	56.6%		
	YES	Count	555	9	564		
		% within PP	43.7%	30.0%	43.4%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Mother-side is shown in table, was not significant statistically ($P>0.05$).

Table 85: Association Between Fundal Placenta and Perinatal Death

			Fundus		Total	Chi – square value	P - value
			NO	YES			
DEATH	NO	Count	1010	289	1299	0.888	0.346
		% within Fundus	99.9%	99.7%	99.8%		
	YES	Count	1	1	2		
		% within Fundus	0.1%	0.3%	0.2%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 86: Association Between Right Lateral Placenta and Perinatal Death

			Right Lateral		Total	Chi – square value	P - value
			NO	YES			
DEATH	NO	Count	1233	66	1299	0.107	0.744
		% within RL	99.8%	100.0%	99.8%		
	YES	Count	2	0	2		
		% within RL	0.2%	0.0%	0.2%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 87: Association Between Left Lateral Placenta and Perinatal Death

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
DEATH	NO	Count	1250	49	1299	0.078	0.779
		% within LL	99.8%	100.0%	99.8%		
	YES	Count	2	0	2		
		% within LL	0.2%	0.0%	0.2%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 88: Association Between Posterior Placenta and Perinatal Death

			Posterior		Total	Chi – square value	P - value
			NO	YES			
DEATH	NO	Count	816	483	1299	0.140	0.708
		% within Posterior	99.9%	99.8%	99.8%		
	YES	Count	1	1	2		
		% within Posterior	0.1%	0.2%	0.2%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 89: Association Between Anterior Placenta and Perinatal Death

			Anterior		Total	Chi – Square value	P - value
			NO	YES			
DEATH	NO	Count	735	564	1299	0.035	0.851
		% within Anterior	99.9%	99.8%	99.8%		
	YES	Count	1	1	2		
		% within Anterior	0.1%	0.2%	0.2%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 90: Association Between Placenta Previa and Perinatal Death

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
DEATH	NO	Count	1269	30	1299	0.047	0.828
		% within PP	99.8%	100.0%	99.8%		
	YES	Count	2	0	2		
		% within PP	0.2%	0.0%	0.2%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Perinatal Death is shown in table, was not significant statistically ($P>0.05$).

Table 91: Association Between Fundal Placenta and Fresh Still Birth

			Fundus		Total	Chi – square value	P - value
			NO	YES			
FSB	NO	Count	979	282	1261	0.125	0.724
		% within Fundus	96.8%	97.2%	96.9%		
	YES	Count	32	8	40		
		% within Fundus	3.2%	2.8%	3.1%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal location of Placenta and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 92: Association Between Right Lateral Placenta and Fresh Still Birth

			Right Lateral		Total	Chi – square value	P - value
			NO	YES			
FSB	NO	Count	1198	63	1261	0.505	0.477
		% within RL	97.0%	95.5%	96.9%		
	YES	Count	37	3	40		
		% within RL	3.0%	4.5%	3.1%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 93: Association Between Left Lateral Placenta and Fresh Still Birth

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
FSB	NO	Count	1213	48	1261	0.183	0.669
		% within LL	96.9%	98.0%	96.9%		
	YES	Count	39	1	40		
		% within LL	3.1%	2.0%	3.1%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Left Lateral Placenta and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 94: Association Between Posterior Placenta and Fresh Still Birth

			Posterior		Total	Chi – Square value	P - value
			NO	YES			
FSB	NO	Count	793	468	1261	0.138	0.710
		% within Posterior	97.1%	96.7%	96.9%		
	YES	Count	24	16	40		
		% within Posterior	2.9%	3.3%	3.1%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior location of Placenta and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 95: Association Between Anterior Placenta and Fresh Still Birth

			Anterior		Total	Chi – square value	P - value
			NO	YES			
FSB	NO	Count	712	549	1261	0.197	0.657
		% within Anterior	96.7%	97.2%	96.9%		
	YES	Count	24	16	40		
		% within Anterior	3.3%	2.8%	3.1%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 96: Association Between Placenta Previa and Fresh Still Birth

			Placenta Previa		Total	Chi – square value	P – value
			NO	YES			
FSB	NO	Count	1231	30	1261	0.974	0.324
		% within PP	96.9%	100.0%	96.9%		
	YES	Count	40	0	40		
		% within PP	3.1%	0.0%	3.1%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Fresh Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 97: Association Between Fundal Placenta and Macerated Still Birth

			Fundus		Total	Chi- square value	P - value
			NO	YES			
MSB	NO	Count	1007	288	1295	0.424	0.515
		% within Fundus	99.6%	99.3%	99.5%		
	YES	Count	4	2	6		
		% within Fundus	0.4%	0.7%	0.5%		
Total		Count	1011	290	1301		
		% within Fundus	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Fundal Placenta and Macerated Still Birth is shown in table, was not significant statistically ($P > 0.05$).

Table 98: Association Between Right Lateral Placenta and Macerated Still Birth

			Right Lateral		Total	Chi – Square value	P – value
			NO	YES			
MSB	NO	Count	1229	66	1295	0.322	0.570
		% within RL	99.5%	100.0%	99.5%		
	YES	Count	6	0	6		
		% within RL	0.5%	0.0%	0.5%		
Total		Count	1235	66	1301		
		% within RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and Macerated Still Birth is shown in table, was not significant statistically ($P > 0.05$).

Table 99: Association Between Left Lateral Placenta and Macerated Still Birth

			Left Lateral		Total	Chi – Square value	P - value
			NO	YES			
MSB	NO	Count	1247	48	1295	2.768	0.096
		% within LL	99.6%	98.0%	99.5%		
	YES	Count	5	1	6		
		% within LL	0.4%	2.0%	0.5%		
Total		Count	1252	49	1301		
		% within LL	100.0%	100.0%	100.0%		

The association between Left Lateral Placenta and Macerated Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 100: Association Between Posterior Placenta and Macerated Still Birth

			Posterior		Total	Chi – square value	P - value
			NO	YES			
MSB	NO	Count	814	481	1295	0.423	0.516
		% within Posterior	99.6%	99.4%	99.5%		
	YES	Count	3	3	6		
		% within Posterior	0.4%	0.6%	0.5%		
Total		Count	817	484	1301		
		% within Posterior	100.0%	100.0%	100.0%		

Statistically Not Significant

The association between Posterior Placenta and Macerated Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 101: Association Between Anterior Placenta and Macerated Still Birth

			Anterior		Total	Chi – square value	P - value
			NO	YES			
MSB	NO	Count	732	563	1295	0.250	0.617
		% within Anterior	99.5%	99.6%	99.5%		
	YES	Count	4	2	6		
		% within Anterior	0.5%	0.4%	0.5%		
Total		Count	736	565	1301		
		% within Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and Macerated Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 102: Association Between Placenta Previa and Macerated Still Birth

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
MSB	NO	Count	1265	30	1295	0.142	0.706
		% within PP	99.5%	100.0%	99.5%		
	YES	Count	6	0	6		
		% within PP	0.5%	0.0%	0.5%		
Total		Count	1271	30	1301		
		% within PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and Macerated Still Birth is shown in table, was not significant statistically ($P>0.05$).

Table 103: Association Between Fundal Placenta and HPR of Maternal Surface of Placenta

		Fundus		Total	Chi – Square value	P – value	
		NO	YES				
MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	35	12	47	14.441	0.071
		% Fundus	3.5%	4.1%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Perivillous fibrin	Count	36	6	42		
		% Fundus	3.6%	2.1%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Perivillous fibrin	Count	45	6	51		
		% Fundus	4.5%	2.1%	3.9%		
	Intervillous fibrin	Count	10	2	12		
		% Fundus	1.0%	0.7%	0.9%		
	Intervillous fibrin, Perivillous fibrin	Count	413	146	559		
		% Fundus	40.9%	50.3%	43.0%		
	Normal	Count	75	17	92		
		% Fundus	7.4%	5.9%	7.1%		
	Villous infarction, Intervillous fibrin, Perivillous fibrin	Count	46	17	63		
		% Fundus	4.5%	5.9%	4.8%		
	Villous infarction, Villous agglutination, Intervillous fibrin, Perivillous fibrin	Count	293	67	360		
		% Fundus	29.0%	23.1%	27.7%		
Villous infarction, Villous agglutination, Intervillous fibrin, Perivillous fibrin, Intervillous thrombus	Count	58	17	75			
	% Fundus	5.7%	5.9%	5.8%			

Total	Count	1011	290	1301	
	% Fundus	100.0%	100.0%	100.0%	
Statistically Not Significant					

The association between Fundal Placenta and HPR of Maternal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 104: Association Between Right Lateral Placenta and HPR of Maternal Surface of Placenta

			Right Lateral		Total	Chi – square value	P – value
			NO	YES			
MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	45	2	47	12.393	0.134
		% RL	3.6%	3.0%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Peri- villous fibrin	Count	41	1	42		
		% RL	3.3%	1.5%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	47	4	51		
		% RL	3.8%	6.1%	3.9%		
	Intervillous fibrin	Count	11	1	12		
		% RL	0.9%	1.5%	0.9%		
	Intervillous fibrin, Peri- villous fibrin	Count	522	37	559		
		% RL	42.3%	56.1%	43.0%		
	Normal	Count	88	4	92		
		% RL	7.1%	6.1%	7.1%		
	Villous infarction, Intervillous fibrin, Peri- villous fibrin	Count	63	0	63		
		% RL	5.1%	0.0%	4.8%		
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	349	11	360		
		% RL	28.3%	16.7%	27.7%		

	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin, Intervillous thrombus	Count	69	6	75
		% RL	5.6%	9.1%	5.8%
Total		Count	1235	66	1301
		% RL	100.0%	100.0%	100.0%
Statistically Not Significant					

The association between Right Lateral Placenta and HPR of Maternal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 105: Association Between Left Lateral Placenta and HPR of Maternal Surface of Placenta

		Left Lateral		Total	Chi – square value	P – value	
		NO	YES				
MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	45	2	47	6.564	0.584
		% LL	3.6%	4.1%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Peri-villous fibrin	Count	39	3	42		
		% LL	3.1%	6.1%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	50	1	51		
		% LL	4.0%	2.0%	3.9%		
	Intervillous fibrin	Count	12	0	12		
		% LL	1.0%	0.0%	0.9%		
	Intervillous fibrin, Peri-villous fibrin	Count	533	26	559		
		% LL	42.6%	53.1%	43.0%		
	Normal	Count	88	4	92		
		% LL	7.0%	8.2%	7.1%		
	Villous infarction, Intervillous fibrin, Peri-villous fibrin	Count	60	3	63		
		% LL	4.8%	6.1%	4.8%		
	Villous infarction, Villous	Count	351	9	360		

	agglutination, Intervillous fibrin, Peri-villous fibrin	% LL	28.0%	18.4%	27.7%
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin, Intervillous thrombus	Count	74	1	75
		% LL	5.9%	2.0%	5.8%
Total		Count	1252	49	1301
		% LL	100.0%	100.0%	100.0%
Statistically Not Significant					

The association between Left Lateral Location of Placenta and HPR of Maternal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 106: Association Between Posterior Placenta and HPR of Maternal Surface of Placenta

			Posterior		Total	Chi – Square value	P – value
			NO	YES			
MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	45	2	47	6.564	0.584
		% Posterior	3.6%	4.1%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Peri-villous fibrin	Count	39	3	42		
		% Posterior	3.1%	6.1%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	50	1	51		
		% Posterior	4.0%	2.0%	3.9%		
	Intervillous fibrin	Count	12	0	12		
		% Posterior	1.0%	0.0%	0.9%		
	Intervillous fibrin, Peri-villous fibrin	Count	533	26	559		
		% Posterior	42.6%	53.1%	43.0%		
	Normal	Count	88	4	92		
		% Posterior	7.0%	8.2%	7.1%		
	Villous infarction, Intervillous	Count	60	3	63		

	fibrin, Peri-villous fibrin	% Posterior	4.8%	6.1%	4.8%
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	351	9	360
		% Posterior	30.0%	23.8%	27.7%
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin, Intervillous thrombus	Count	49	26	75
		% Posterior	6.0%	5.4%	5.8%
Total	Count		817	484	1301
	% Posterior		100.0%	100.0%	100.0%
Statistically Not Significant					

The association between Posterior Placenta and Maternal Surface of Placenta is shown in table, was not significant statistically ($P > 0.05$).

Table 107: Association Between Anterior Placenta and HPR of Maternal Surface of Placenta

			Anterior		Total	Chi – square value	P – value
			NO	YES			
MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	23	24	47		
		% Anterior	3.1%	4.2%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Peri-villous fibrin	Count	27	15	42		
		% Anterior	3.7%	2.7%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	29	22	51		
		% Anterior	3.9%	3.9%	3.9%		
	Intervillous fibrin	Count	7	5	12		
		% Anterior	1.0%	0.9%	0.9%		

	Intervillous fibrin, Peri-villous fibrin	Count	347	212	559	18.119	0.080		
		% Anterior	47.1%	37.5%	43.0%				
Normal	Count	49	43	92					
	% Anterior	6.7%	7.6%	7.1%					
Villous infarction, Intervillous fibrin, Peri-villous fibrin	Count	36	27	63					
	% Anterior	4.9%	4.8%	4.8%					
Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	176	184	360					
	% Anterior	23.9%	32.6%	27.7%					
Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin, Intervillous thrombus	Count	42	33	75					
	% Anterior	5.7%	5.8%	5.8%					
Total	Count	736	565	1301					
	% Anterior	100.0%	100.0%	100.0%					
Statistically Not Significant									

The association between Anterior Placenta and HPR of Maternal Surface of Placenta is shown in table, was not significant statistically ($P > 0.05$).

Table 108: Association Between Placenta Previa and Maternal Surface of Placenta

	PP		Total	Chi – square value	P – value
	NO	YES			

MATERNAL SURFCAE	Chorioamnionitis, Intervillous fibrin	Count	46	1	47	7.893	0.888
		% PP	3.6%	3.3%	3.6%		
	Chorioamnionitis, Intervillous fibrin, Peri-villous fibrin	Count	41	1	42		
		% PP	3.2%	3.3%	3.2%		
	Chorioamnionitis, Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	50	1	51		
		% PP	3.9%	3.3%	3.9%		
	Intervillous fibrin	Count	11	1	12		
		% PP	0.9%	3.3%	0.9%		
	Intervillous fibrin, Peri-villous fibrin	Count	551	8	559		
		% PP	43.4%	26.7%	43.0%		
	Normal	Count	91	1	92		
		% PP	7.2%	3.3%	7.1%		
	Villous infarction, Intervillous fibrin, Peri-villous fibrin	Count	60	3	63		
		% PP	4.7%	10.0%	4.8%		
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin	Count	348	12	360		
		% PP	27.4%	40.0%	27.7%		
	Villous infarction, Villous agglutination, Intervillous fibrin, Peri-villous fibrin, Intervillous thrombus	Count	73	2	75		
		% PP	5.7%	6.7%	5.8%		
Total	Count	1271	30	1301			
	% PP	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Placenta Previa and Maternal Surface of Placenta is shown in table, was statistically not significant ($P > 0.05$).

Table 109: Association Between Fundal Placenta and HPR of Decidual Arteriopathy of Placenta

	Fundus	Total	Chi –	P-Value
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			NO	YES		square value	
DECIDUAL ARTERIPATHY	Atherosis, Chorioangiosis, Fibrinoid Necrosis	Count	92	31	123	9.573	0.144
		% Fundus	9.1%	10.7%	9.5%		
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	124	34	158		
		% Fundus	12.3%	11.7%	12.1%		
	Chorioangiosis, Fibrinoid Necrosis	Count	59	23	82		
		% Fundus	5.8%	7.9%	6.3%		
	Fibrinoid Necrosis	Count	97	26	123		
		% Fundus	9.6%	9.0%	9.5%		
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	462	117	579		
		% Fundus	45.7%	40.3%	44.5%		
	Mural Hypertrophy of Membrane arterioles	Count	49	25	74		
		% Fundus	4.8%	8.6%	5.7%		
	Normal	Count	128	34	162		
		% Fundus	12.7%	11.7%	12.5%		
Total	Count	1011	290	1301			
	% Fundus	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Fundal Placenta and HPR of Decidual Arteriopathy of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 110: Association Between Right Lateral Placenta and HPR of Decidual Arteriopathy of Placenta

		Right Lateral		Total			
		NO	YES				
DECIDUAL ARTERIPATHY	Atherosis, Chorangiopsis, Fibrinoid Necrosis	Count	118	5	123	2.861	0.826
		% RL	9.6%	7.6%	9.5%		
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	147	11	158		
		% RL	11.9%	16.7%	12.1%		
	Chorangiopsis, Fibrinoid Necrosis	Count	79	3	82		
		% RL	6.4%	4.5%	6.3%		
	Fibrinoid Necrosis	Count	117	6	123		
		% RL	9.5%	9.1%	9.5%		
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	549	30	579		
		% RL	44.5%	45.5%	44.5%		
	Mural Hypertrophy of Membrane arterioles	Count	69	5	74		
		% RL	5.6%	7.6%	5.7%		
	Normal	Count	156	6	162		
		% RL	12.6%	9.1%	12.5%		
Total	Count	1235	66	1301			
	% RL	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Right Lateral Placenta and HPR of Decidual Arteriopathy of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 111: Association Between Left Lateral Placenta and HPR of Decidual Arteriopathy of Placenta

			LL		Total	Chi – square value	P – value
			NO	YES			
DECIDUAL ARTERIPATHY	Atherosis, Chorangiostis, Fibrinoid Necrosis	Count	119	4	123	1.613	0.952
		% LL	9.5%	8.2%	9.5%		
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	154	4	158		
		% LL	12.3%	8.2%	12.1%		
	Chorangiostis, Fibrinoid Necrosis	Count	79	3	82		
		% LL	6.3%	6.1%	6.3%		
	Fibrinoid Necrosis	Count	117	6	123		
		% LL	9.3%	12.2%	9.5%		
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	556	23	579		
		% LL	44.4%	46.9%	44.5%		
	Mural Hypertrophy of Membrane arterioles	Count	72	2	74		
		% LL	5.8%	4.1%	5.7%		
	Normal	Count	155	7	162		
		% LL	12.4%	14.3%	12.5%		
Total	Count	1252	49	1301			
	% LL	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Left Lateral Placenta and HPR of Decidual Arteriopathy of Placenta is shown in table, was not significant statistically (P>0.05).

Table 112: Association Between Posterior Placenta and HPR of Decidual Arteriopathy of Placenta

			Posterior		Total	Chi – square valur	P – value			
			NO	YES						
DECIDUAL ARTERIPATH Y	Atherosis, Chorangiosis, Fibrinoid Necrosis	Count	69	54	123	6.311	0.389			
		% Posterior	8.4%	11.2%	9.5%					
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	96	62	158					
		% Posterior	11.8%	12.8%	12.1%					
	Chorangiosis, Fibrinoid Necrosis	Count	49	33	82					
		% Posterior	6.0%	6.8%	6.3%					
	Fibrinoid Necrosis	Count	75	48	123					
		% Posterior	9.2%	9.9%	9.5%					
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	383	196	579					
		% Posterior	46.9%	40.5%	44.5%					
	Mural Hypertrophy of Membrane arterioles	Count	47	27	74					
		% Posterior	5.8%	5.6%	5.7%					
	Normal	Count	98	64	162					
		% Posterior	12.0%	13.2%	12.5%					
	Total	Count	817	484	1301					
		% Posterior	100.0%	100.0%	100.0%					
	Statistically Not Significant									

The association between Posterior Placenta and HPR of Decidual Arteriopathy of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 113: Association Between Anterior Placenta and HPR of Decidual Arteriopathy of Placenta

			Anterior		Total	Chi – square value	P – value
			NO	YES			
DECIDUAL ARTERIPATHY	Atherosis, Chorangi- osis, Fibrinoid Necrosis	Count	76	47	123	5.965	0.427
		% Anterior	10.3%	8.3%	9.5%		
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	94	64	158		
		% Anterior	12.8%	11.3%	12.1%		
	Chorangi- osis, Fibrinoid Necrosis	Count	52	30	82		
		% Anterior	7.1%	5.3%	6.3%		
	Fibrinoid Necrosis	Count	70	53	123		
		% Anterior	9.5%	9.4%	9.5%		
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	310	269	579		
		% Anterior	42.1%	47.6%	44.5%		
	Mural Hypertrophy of Membrane arterioles	Count	44	30	74		
		% Anterior	6.0%	5.3%	5.7%		
	Normal	Count	90	72	162		
		% Anterior	12.2%	12.7%	12.5%		
Total	Count	736	565	1301			
	% Anterior	100.0%	100.0%	100.0%			

Statistically Not Significant

The association between Anterior Placenta and Decidual Arteriopathy of Placenta is shown in table, was statistically not significant ($P > 0.05$).

Table 114: Association Between Placenta Previa and HPR of Decidual Arteriopathy of Placenta

			PP		Total	Chi – square value	P – value
			NO	YES			
DECIDUAL ARTERIPATHY	Atherosis, Chorangioidis, Fibrinoid Necrosis	Count	121	2	123	0.960	0.987
		% PP	9.5%	6.7%	9.5%		
	Atherosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	154	4	158		
		% PP	12.1%	13.3%	12.1%		
	Chorangioidis, Fibrinoid Necrosis	Count	80	2	82		
		% PP	6.3%	6.7%	6.3%		
	Fibrinoid Necrosis	Count	120	3	123		
		% PP	9.4%	10.0%	9.5%		
	Fibrinoid Necrosis, Mural Hypertrophy of Membrane arterioles	Count	564	15	579		
		% PP	44.4%	50.0%	44.5%		
	Mural Hypertrophy of Membrane arterioles	Count	73	1	74		
		% PP	5.7%	3.3%	5.7%		
	Normal	Count	159	3	162		
		% PP	12.5%	10.0%	12.5%		
Total	Count	1271	30	1301			
	% PP	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Placenta Previa and HPR of Decidual Arteriopathy of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 115: Association Between Fundal Placenta and HPR of Foetal Surface of Placenta

			Fundus		Total	Chi – square value	P – value
			NO	YES			
FETAL SURFACE	Chorangiomas	Count	16	3	19	10.701	0.030*
		% Fundus	1.6%	1.0%	1.5%		
	Clustered Villi	Count	391	125	516		
		% Fundus	38.7%	43.1%	39.7%		
	Clustered Villi, Chorangiomas	Count	275	60	335		
		% Fundus	27.2%	20.7%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	6	6	12		
		% Fundus	0.6%	2.1%	0.9%		
	Normal	Count	323	96	419		
		% Fundus	31.9%	33.1%	32.2%		
	Total	Count	1011	290	1301		
		% Fundus	100.0%	100.0%	100.0%		
*Statistically Significant							

The association between Fundal Placenta and HPR of Foetal Surface of Placenta is shown in table, and was positive and was significant statistically ($P < 0.05$).

Table 116: Association Between Right Lateral Placenta and HPR of Foetal Surface of Placenta

			Right Lateral		Total	Chi – square value	P - value
			NO	YES			
FETAL SURFACE	Chorangiosis	Count	18	1	19	1.552	0.817
		% RL	1.5%	1.5%	1.5%		
	Clustered Villi	Count	487	29	516		
		% RL	39.4%	43.9%	39.7%		
	Clustered Villi, Chorangiomas	Count	317	18	335		
		% RL	25.7%	27.3%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	12	0	12		
		% RL	1.0%	0.0%	0.9%		
	Normal	Count	401	18	419		
		% RL	32.5%	27.3%	32.2%		
	Total	Count	1235	66	1301		
		% RL	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Right Lateral Placenta and HPR of Foetal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 117: Association Between Left Lateral Placenta and HPR of Foetal Surface of Placenta

			Left Lateral		Total	Chi – square value	P - value
			NO	YES			
FETAL SURFACE	Chorangiosis	Count	17	2	19	5.854	0.210
		% LL	1.4%	4.1%	1.5%		
	Clustered Villi	Count	500	16	516		
		% LL	39.9%	32.7%	39.7%		
	Clustered Villi, Chorangiomas	Count	325	10	335		
		% LL	26.0%	20.4%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	12	0	12		
		% LL	1.0%	0.0%	0.9%		
	Normal	Count	398	21	419		
		% LL	31.8%	42.9%	32.2%		
Total	Count	1252	49	1301			
	% LL	100.0%	100.0%	100.0%			
Statistically Not Significant							

The association between Left Lateral Placenta and HPR of Foetal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 119: Association Between Posterior Placenta and HPR of Foetal Surface of Placenta

			Posterior		Total	Chi – square value	P - value
			NO	YES			
FETAL SURFACE	Chorangiosis	Count	13	6	19	7.264	0.123
		% Posterior	1.6%	1.2%	1.5%		
	Clustered Villi	Count	317	199	516		
		% Posterior	38.8%	41.1%	39.7%		
	Clustered Villi, Chorangiomas	Count	223	112	335		
		% Posterior	27.3%	23.1%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	4	8	12		
		% Posterior	0.5%	1.7%	0.9%		
	Normal	Count	260	159	419		
		% Posterior	31.8%	32.9%	32.2%		
	Total	Count	817	484	1301		
		% Posterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Posterior Placenta and HPR of Foetal Surface of Placenta is shown in table, was not significant statistically ($P > 0.05$).

Table 119: Association Between Anterior Placenta and HPR of Foetal Surface of Placenta

			Anterior		Total	Chi – square value	P - value
			NO	YES			
FETAL SURFACE	Chorangiomas	Count	10	9	19	6.439	0.169
		% Anterior	1.4%	1.6%	1.5%		
	Clustered Villi	Count	300	216	516		
		% Anterior	40.8%	38.2%	39.7%		
	Clustered Villi, Chorangiomas	Count	176	159	335		
		% Anterior	23.9%	28.1%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	10	2	12		
		% Anterior	1.4%	0.4%	0.9%		
	Normal	Count	240	179	419		
		% Anterior	32.6%	31.7%	32.2%		
	Total	Count	736	565	1301		
		% Anterior	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Anterior Placenta and HPR of Foetal Surface of Placenta is shown in table, was not significant statistically ($P>0.05$).

Table 120: Association Between Placenta Previa and HPR of Foetal Surface of Placenta

			Placenta Previa		Total	Chi – square value	P - value
			NO	YES			
FETAL SURFACE	Chorangiosis	Count	19	0	19	2.905	0.574
		% PP	1.5%	0.0%	1.5%		
	Clustered Villi	Count	504	12	516		
		% PP	39.7%	40.0%	39.7%		
	Clustered Villi, Chorangiogenesis	Count	324	11	335		
		% PP	25.5%	36.7%	25.7%		
	Clustered Villi, Small Thrombi in Blood vessels	Count	12	0	12		
		% PP	0.9%	0.0%	0.9%		
	Normal	Count	412	7	419		
		% PP	32.4%	23.3%	32.2%		
	Total	Count	1271	30	1301		
		% PP	100.0%	100.0%	100.0%		
Statistically Not Significant							

The association between Placenta Previa and HPR of Foetal Surface of Placenta is shown in table, was not significant statistically ($P > 0.05$).

DISCUSSION

The blood supply of uterus is not uniformly distributed and placental location is an important determinant of placental blood flow, as measured by uterine artery Doppler velocimetry. There are limited data on association between placental location, maternal complications and foetal outcome.

In our study 1861 cases were screened ,250 cases were excluded as they did not meet inclusion criteria, 88 cases did not give consent for study and 222 cases were excluded as their placentas could not be sent for HPR.A total of 1301 cases were included in the study.

Age Group

Out of 1301 cases studied 186 cases (14.3%) were <20 years of age, 988 cases (75.9%) belonged to 21-30 years of age and 127 cases (9.8%) belonged to ≥ 31 years of age.

The mean age (years) of patients was 24.9570 ± 4.2701 . Most of the patients were [988 (75.9%)] 21-30 years of age. Age was statistically significant with Placental Location ($p=0.0116$).

Obstetric History

Among 1301 patients, 920(70.7%) patients are multigravida and 381(29.3%) belong to primigravida. Most of the patients had Multi Obstetric History [335 (69.9%)] in Anterior compared to Posterior [270 (69.2%)], Fundus [213 (73.7%)], RL [50 (75.8%)], PP [21 (70.0%)] and LL [31 (66.0%)] it was not statistically significant ($p=0.6797$).

Association of Obstetric Score with Placental Location was not statistically significant ($p=0.7448$).

Past History

In our study past history of patients seen are failed IUI, allergic to oral iron preparations, Ante partum Eclampsia in previous pregnancy, Cervical Encirclage, Hypothyroidism, Corrected ASD, Drug allergy, Disc Prolapse since 2 Yrs, GDM since 7 MOA, HbsAg positive status, Infertility treatment, Pre-Eclampsia in previous history, Varicose veins etc.

Association of Past History with Placental Location was not statistically significant ($p=0.9947$).

Placental Location

In our study 479(36.8%) patients had Anterior location of placenta, 289(22.2%) patients had fundal location of placenta, 47 (3.6%) patients had left Lateral location of placenta, 390(30%) patients had posterior location of placenta, 30(2.3%) patients had Placenta previa and 66(5.1%) patients had Right Lateral location of placenta. There-fore the most common location of placenta according to our study is Anterior Location of Placenta.

Maternal Complication

Among 1301 cases we studied 27(2.1%) patients had mild pre-eclampsia, 110(8.5%) patients had Severe Pre-eclampsia, 69(5.3%) patients suffered from Eclampsia, 39(3%) patients had Abruptio, 39(3%) patients presented with FGR, 233(17.9%) patients had Oligohydramnios, 9(0.7%) patients had Polyhydramnios, 306(23.5%) patients delivered by Preterm delivery, and 94(7.2%) patients had Premature Rupture of Membranes. From our study the incidence of Preterm delivery is higher among all the other maternal complications. Others are Abnormal Doppler Changes, GDM, Ante Partum Eclampsia, Candidiasis, Covid positive, Drug allergy, GHTN, HELLP, Gestational Thrombocytopenia, Hyperthyroidism, Hypothyroidism, Obstructed Breech Labour, PPH, Syphilis, Uterine Rupture, Vaginitis etc.

Association of Other complications with Placental Location was not statistically significant (p=1.000).

Mode of Delivery

From 1301 patients studied in our study 778(60.6%) patients underwent LSCS, while 499(38.4%) patients delivered vaginally and 13(1%) patients had Instrumental Delivery. From our study we can say that most of the patients underwent LSCS.

There was positive and was significant statistic association between Right Lateral location of Placenta and Vaginal Delivery and LSCS.

Fetal Outcome

Out of 1301 patients 684(52.6%) patients had NICU admission of their new-borns, 564(43.4%) new-borns were given mother-side, 2(0.2%) patients had perinatal deaths,40(3.1%) patients had fresh still births, 6(0.5%) patients had Macerated still births. Most of the patients had NICU admissions of their neonates.

There was no statistically significant association seen between any of the neonatal outcomes and placental Location.

Histopathological Report Of Placenta

Maternal Surface:

47(3.6%)showed Chorioamnionitis with intervillous fibrin, 42(3.3%)had chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 51(3.9%) placentas had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 559(42.9%) Placenta's had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 63(4.8%) placentas had

Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 360(27.7%) placentas had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 75(5.8%) placentas had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface and 92(7.1%) patients had Normal Maternal Surface of placenta.

There was no statistically significant association seen between HPR of Maternal Surface of Placenta.

Decidual Arteriopathy:

Atherosclerosis, Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, 158(12.2%) placentas had Atherosclerosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 82(6.3%) patient's placentas showed Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries, 122(9.4%) placentas showed fibrinoid Necrosis change in Decidual arteries, 579(44.5%) placentas had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 74(5.7%) placentas had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries and 162(12.5%) patients had normal decidual arteries in Placenta.

There was no statistically significant association seen between HPR of Decidual Arteriopathy of Placenta.

Foetal Surface:

19(1.5%) placentas showed Chorangiomas of fetal surface vessels, 516(39.7%) placentas showed Clustered Villi at the Foetal Surface, 335(25.7%) placentas showed Clustered Villi and Chorangiomas changes in foetal Surface, 12(0.9%) placentas showed Clustered Villi, Small

Thrombi in Blood vessels of foetal Surface and 419(32.2%) Placentas showed Normal Foetal Surface.

There was a positive and statistically significant association seen between HPR of Foetal Surface of Placenta and Fundal Implantation of Placenta

ASSOCIATION BETWEEN LOCATION OF PLACENTA AND MATERNAL OUTCOME

(1) MILD PRE-ECLAMPSIA

Fundal Location

Out of 27(2.1%) patients who had Mild Pre-Eclampsia, 8(2.8%) patients had Fundal implantation of Placenta. The Association between Mild Pre-Eclampsia and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 27(2.1%) patients who suffered from Mild Pre-Eclampsia, 1(1.5%) patient had Right Lateral Location of Placenta. The Association between Mild Pre-Eclampsia and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 27(2.1%) patients who had Mild Pre-Eclampsia, 0 patients had Left Lateral implantation of Placenta. The Association between Mild Pre-Eclampsia and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 27(2.1%) patients who had Mild Pre-Eclampsia, 10(2.1%) patients had Posterior implantation of Placenta. The Association between Mild Pre-Eclampsia and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 27(2.1%) patients who had Mild Pre-Eclampsia, 11(1.9%) patients had Anterior implantation of Placenta. The Association between Mild Pre-Eclampsia and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 27(2.1%) patients who had Mild Pre-Eclampsia, 0 patients had Placenta Previa. The Association between Mild Pre-Eclampsia and Placenta Previa was not statistically significant ($P>0.05$).

(2) SEVERE PRE-ECLAMPSIA

Fundal Location

Out of 110(8.5%) patients who had Severe Pre-Eclampsia, 13(4.5%) patients had Fundal implantation of Placenta. The Association between Severe Pre-Eclampsia and Fundal Location of Placenta was Positive and statistically significant ($P<0.05$).

Right Lateral Location

Among 110(8.5%) patients who suffered from Severe Pre-Eclampsia, 6(9.1%) patients had Right Lateral Location of Placenta. The Association between Severe Pre-Eclampsia and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 110(8.5%) patients who had Severe Pre-Eclampsia, 8(16.3%) patients had Left Lateral implantation of Placenta. The Association between Severe Pre-Eclampsia and Left Lateral Location of Placenta was Positive and statistically significant ($P<0.05$).

Posterior Location

Out of 110(8.5%) patients who had Severe Pre-Eclampsia, 39(8.1%) patients had Posterior implantation of Placenta. The Association between Severe Pre-Eclampsia and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 110(8.5%) patients who had Severe Pre-Eclampsia, 50(8.8%) patients had Anterior implantation of Placenta. The Association between Severe Pre-Eclampsia and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 110(8.5%) patients who had Severe Pre-Eclampsia, 3(10%) patients had Placenta Previa. The Association between Severe Pre-Eclampsia and Placenta Previa was not statistically significant ($p->0.05$).

(3) ECLAMPSIA

Fundal Location

Out of 69(5.3%) patients who had Eclampsia, 12(4.1%) patients had Fundal implantation of Placenta. The Association between Eclampsia and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 69(5.3%) patients who suffered from Eclampsia, 6(9.1%) patients had Right Lateral Location of Placenta. The Association between Eclampsia and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 69(5.3%) patients who had Eclampsia, 0 patients had Left Lateral implantation of Placenta. The Association between Eclampsia and Left Lateral Location of Placenta was not significant statistically ($P>0.05$).

Posterior Location

Out of 69(5.3%) patients who had Eclampsia, 27(5.6%) patients had Posterior implantation of Placenta. The Association between Eclampsia and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 69(5.3%) patients who had Eclampsia, 27(4.8%) patients had Anterior implantation of Placenta. The Association between Eclampsia and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 69(5.3%) patients who had Eclampsia, 2(6.7%) patients had Placenta Previa. The Association between Eclampsia and Placenta Previa was not statistically significant ($P>0.05$).

(4) Abruption

Fundal Location

Out of 39(3%) patients who had Abruption, 5(1.7%) patients had Fundal implantation of Placenta. The Association between Abruption and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 39(3%) patients who suffered from Abruption, 2(3%) patients had Right Lateral Location of Placenta. The Association between Abruption and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 39(3%) patients who had Abruption, 02(4.1%) patients had Left Lateral implantation of Placenta. The Association between Abruption and Left Lateral Location of Placenta was not significant statistically ($P>0.05$).

Posterior Location

Out of 39(3%) patients who had Abruption, 16(3.3%) patients had Posterior implantation of Placenta. The Association between Abruption and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 39(3%) patients who had Abruption, 16(2.8%) patients had Anterior implantation of Placenta. The Association between Abruption and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 39(3%) patients who had Abruption, 0 patients had Placenta Previa. The Association between Abruption and Placenta Previa was not statistically significant ($P>0.05$).

(5) FETAL GROWTH RESTRICTION

Fundal Location

Out of 39(3%) patients who had Foetal Growth Restriction, 8(2.8%) patients had Fundal implantation of Placenta. The Association between Foetal Growth Restriction and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 39(3%) patients who suffered from Foetal Growth Restriction, 0 patients had Right Lateral Location of Placenta. The Association between Foetal Growth Restriction and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 39(3%) patients who had Foetal Growth Restriction, 03(6.1%) patients had Left Lateral implantation of Placenta. The Association between Foetal Growth Restriction and Left Lateral Location of Placenta was Positive and statistically significant ($P<0.05$).

Posterior Location

Out of 39(3%) patients who had Foetal Growth Restriction, 14(2.9%) patients had Posterior implantation of Placenta. The Association between Foetal Growth Restriction and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 39(3%) patients who had Foetal Growth Restriction, 19(3.4%) patients had Anterior implantation of Placenta. The Association between Foetal Growth Restriction and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 39(3%) patients who had Foetal Growth Restriction, 0 patients had Placenta Previa. The Association between Foetal Growth Restriction and Placenta Previa was not statistically significant ($P>0.05$).

(6) OLIGOHYDRAMNIOS

Fundal Location

Out of 233(17.9%) patients who had Oligohydramnios, 48(16.6%) patients had Fundal implantation of Placenta. The Association between Oligohydramnios and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 233(17.9%) patients who suffered from Oligohydramnios, 13(19.7%) patients had Right Lateral Location of Placenta. The Association between Oligohydramnios and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 233(17.9%) patients who had Oligohydramnios, 08(16.3%) patients had Left Lateral implantation of Placenta. The Association between Oligohydramnios and Left Lateral Location of Placenta was not significant statistically ($P>0.05$).

Posterior Location

Out of 233(17.9%) patients who had Oligohydramnios, 80(16.5%) patients had Posterior implantation of Placenta. The Association between Oligohydramnios and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 233(17.9%) patients who had Oligohydramnios, 106(18.8%) patients had Anterior implantation of Placenta. The Association between Oligohydramnios and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 233(17.9%) patients who had Oligohydramnios, 3(10%) patients had Placenta Previa. The Association between Oligohydramnios and Placenta Previa was not statistically significant ($P>0.05$).

(7) POLYHYDRAMNIOS

Fundal Location

Out of 9(0.7%) patients who had Polyhydramnios, 1(0.3%) patient's had Fundal implantation of Placenta. The Association between Polyhydramnios and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 9(0.7%) patients who suffered from Polyhydramnios, 1(1.5%) patient had Right Lateral Location of Placenta. The Association between Polyhydramnios and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 9(0.7%) patients who had Polyhydramnios, 1(2%) patient had Left Lateral implantation of Placenta. The Association between Polyhydramnios and Left Lateral Location of Placenta was not significant statistically ($P>0.05$).

Posterior Location

Out of 9(0.7%) patients who had Polyhydramnios, 4(0.8%) patients had Posterior implantation of Placenta. The Association between Polyhydramnios and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 9(0.7%) patients who had Polyhydramnios, 2(0.4%) patients had Anterior implantation of Placenta. The Association between Polyhydramnios and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 9(0.7%) patients who had Polyhydramnios, 0 patients had Placenta Previa. The Association between Polyhydramnios and Placenta Previa was not statistically significant ($P>0.05$).

(8) PRETERM DELIVERY

Fundal Location

Out of 306(23.5%) patients who had Preterm Delivery, 72(24.8%) patients had Fundal implantation of Placenta. The Association between Preterm Delivery and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 306(23.5%) patients who suffered from Preterm Delivery, 11(16.7%) patients had Right Lateral Location of Placenta. The Association between Preterm Delivery and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 306(23.5%) patients who had Preterm Delivery, 1(2%) patients who had Left Lateral implantation of Placenta. The Association between Preterm Delivery and Left Lateral Location of Placenta was not statistically significant (>0.05).

Posterior Location

Out of 306(23.5%) patients who had Preterm Delivery, 121(25%) patients had Posterior implantation of Placenta. The Association between Preterm Delivery and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 306(23.5%) patients who had Preterm Delivery, 120(21.2%) patients had Anterior implantation of Placenta. The Association between Preterm Delivery and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 306(23.5%) patients who had Preterm Delivery, 11(36.7%) patients had Placenta Previa. The Association between Preterm Delivery and Placenta Previa was not statistically significant ($P>0.05$).

(8) PREMATURE RUPTURE OF MEMBRANES

Fundal Location

Out of 94(7.2%) patients who had Premature Rupture of Membranes, 29(10%) patients had Fundal implantation of Placenta. The Association between Premature Rupture of Membranes and Fundal Location of Placenta was Positive and statistically significant ($P<0.05$).

Right Lateral Location

Among 94(7.2%) patients who suffered from Premature Rupture of Membranes, 6(9.1%) patients had Right Lateral Location of Placenta. The Association between Premature Rupture of Membranes and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 94(7.2%) patients who had Premature Rupture of Membranes, 3(6.1%) patients had Left Lateral implantation of Placenta. The Association between Premature Rupture of Membranes and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 94(7.2%) patients who had Premature Rupture of Membranes, 32(6.6%) patients had Posterior implantation of Placenta. The Association between Premature Rupture of Membranes and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 94(7.2%) patients who had Premature Rupture of Membranes, 45(8%) patients had Anterior implantation of Placenta. The Association between Premature Rupture of Membranes and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 94(7.2%) patients who had Premature Rupture of Membranes, 1(3.3%) patient's had Placenta Previa. The Association between Premature Rupture of Membranes and Placenta Previa was not statistically significant ($P>0.05$).

ASSOCIATION BETWEEN PLACENTAL LOCATION AND FETAL OUTCOME

1. NICU ADMISSION

Fundal Location

Out of 684(52.6%) patients whose neonates needed NICU Admission, 142(49%) patients had Fundal implantation of Placenta. The Association between NICU Admission and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 684(52.6%%) patients whose neonates needed NICU Admission, 35(53%) patients had Right Lateral Location of Placenta. The Association between NICU Admission and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 684(52.6%%) patients whose neonates needed NICU Admission, 21(42.9%) patients had Left Lateral implantation of Placenta. The Association between NICU Admission and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 684(52.6%%) patients whose neonates needed NICU Admission, 253(52.3%) patients had Posterior implantation of Placenta. The Association between NICU Admission es and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 684(52.6%%) patients whose neonates needed NICU Admission, 302(53.5%) patients had Anterior implantation of Placenta. The Association between NICU Admission and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 684(52.6%%) patients whose neonates needed NICU Admission, 21(70%) patients had Placenta Previa. The Association between NICU Admission and Placenta Previa was not statistically significant ($P>0.05$).

2. MOTHERSIDE

Fundal Location

Out of 564(43.4%) patients whose neonates were given Mother-side, 135(46.6%) patients had Fundal implantation of Placenta. The Association between Mother-side and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 564(43.4%) patients whose neonates were given Mother-side, 28(42.4%) patients had Right Lateral Location of Placenta. The Association between Mother-side and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 564(43.4%) patients whose neonates were given Mother-side, 25(51%) patients had Left Lateral implantation of Placenta. The Association between Mother-side and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 564(43.4%) patients whose neonates were given Mother-side, 211(43.6%) patients had Posterior implantation of Placenta. The Association between Mother-side and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 564(43.4%) patients whose neonates were given Mother-side, 242(42.8%) patients had Anterior implantation of Placenta. The Association between Mother-side and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 564(43.4%) patients whose neonates were given Mother-side, 9(30%) patients had Placenta Previa. The Association between Mother-side and Placenta Previa was not statistically significant ($P>0.05$).

(3) PERINATAL DEATH

Fundal Location

Out of 2(0.2%) Perinatal Death, 1(0.3%) patient's had Fundal implantation of Placenta. The Association between Perinatal Death and Fundal Location of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Among 2(0.2%) Perinatal Death, 0 patients had Right Lateral Location of Placenta. The Association between Perinatal Death and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 2(0.2%) Perinatal Death, 0 patients had Left Lateral implantation of Placenta. The Association between Perinatal Death and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 2(0.2%) Perinatal Death, 1(0.2%) patient's had Posterior implantation of Placenta. The Association between Perinatal Death and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 2(0.2%) Perinatal Death, 1(0.2%) patient's had Anterior implantation of Placenta. The Association between Perinatal Death and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 2(0.2%) Perinatal Death, 0 patients had Placenta Previa. The Association between Perinatal Death and Placenta Previa was not statistically significant ($P>0.05$).

(4) FRESH STILLBIRTH

Fundal Location

Out of 40(3.1%) Fresh Still Births, 8(2.8%) patients had Fundal implantation of Placenta. The Association between Fresh Still Births and Fundal Location of Placenta was statistically not significant ($P>0.05$).

Right Lateral Location

Among 40(3.1%) Fresh Still Births, 3(4.5%) patients had Right Lateral Location of Placenta.

The Association between Fresh Still Births and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 40(3.1%) Fresh Still Births, 1(2%) patient who had Left Lateral implantation of Placenta.

The Association between Fresh Still Births and Left Lateral Location of Placenta was not statistically significant ($P>0.05$).

Posterior Location

Out of 40(3.1%) Fresh Still Births, 16(3.3%) patients had Posterior implantation of Placenta. The Association between Fresh Still Births and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 40(3.1%) Fresh Still Births, 16(2.8%) patients had Anterior implantation of Placenta. The Association between Fresh Still Births and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 40(3.1%) Fresh Still Births, 0 patients had Placenta Previa. The Association between Fresh Still Births and Placenta Previa was not statistically significant ($P>0.05$).

(5) MACERATED STILL BIRTH

Fundal Location

Out of 6(0.5%) Macerated Still Births, 2(0.7%) patients had Fundal implantation of Placenta.

The Association between Macerated Still Births and Fundal Location of Placenta was statistically not significant ($P>0.05$).

Right Lateral Location

Among 6(0.5%) Macerated Still Births, 0 patients had Right Lateral Location of Placenta. The Association between Macerated Still Births and Right Lateral Location of Placenta was not statistically significant ($P>0.05$).

Left Lateral Location

Out of 6(0.5%) Macerated Still Births, 1(2%) patient had Left Lateral implantation of Placenta. The Association between Macerated Still Births and Left Lateral Location of Placenta Placenta was statistically not significant ($P>0.05$).

Posterior Location

Out of 6(0.5%) Macerated Still Births, 3(0.6%) patients had Posterior implantation of Placenta. The Association between Macerated Still Births and Posterior Location of Placenta was not statistically significant ($P>0.05$).

Anterior Location

Out of 6(0.5%) Macerated Still Births, 2(0.4%) patients had Anterior implantation of Placenta. The Association between Macerated Still Births and Anterior Location of Placenta was not statistically significant ($P>0.05$).

Placenta Previa

Out of 6(0.5%) Macerated Still Births, 0 patients had Placenta Previa. The Association between Macerated Still Births and Placenta Previa was not statistically significant ($P>0.05$).

ASSOCIATION BETWEEN PLACENTAL LOCATION AND HPR OF PLACENTAMATERNAL SURFACEFundal location

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 12(4.1%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 6(2.1%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 6(2.1%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 146(50.3%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 17(5.9%) had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 67(23.1%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 17(5.9%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 17(5.9%) Placentas had Fundal implantation. The Association between Fundal location of placenta and HPR of Maternal Surface of Placenta was not statistically significant ($P>0.05$).

Right Lateral Location

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 2(3%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 1(1.5%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 4(6.1%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 37(56.1%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 0 had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 11(16.7%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 6(9.1%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 4(6.1%) Placentas had Right Lateral implantation. The Association between Right Lateral location of placenta and HPR of Maternal Surface of Placenta was not statistically significant($P>0.05$).

Left Lateral Location

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 2(4.1%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 3(6.1%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 1(2%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 26(53.1%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and

Peri-villous Fibrin changes on Maternal Surface, 3(6.1%) had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 9(18.4%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 1(2%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 4(8.2%) Placentas had Left Lateral implantation. The Association between Left Lateral location of placenta and HPR of Maternal Surface of Placenta was not statistically significant($P>0.05$).

Posterior Location

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 13(2.7%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 20(4.1%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 20(4.1%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 224(46.3%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 26(5.4%) had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 115(23.8%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 26(5.4%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 36(7.4%) Placentas had Posterior implantation. The

Association between Posterior location of placenta and HPR of Maternal Surface of Placenta was not statistically significant($P>0.05$).

Anterior Location

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 24(4.2%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 15(2.7%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface of Placenta, 22(3.9%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 212(37.5%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 27(4.8%) had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 184(32.6%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 26(5.4%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 33(5.8%) Placentas had Anterior implantation. The Association between Anterior location of placenta and HPR of Maternal Surface of Placenta was not statistically significant($P>0.05$).

Placenta Previa

Out of 47(3.6%)Placentas which showed Chorioamnionitis with intervillous fibrin, 1(3.3%) had ; Out of 42(3.3%) Placentas which showed chorioamnionitis, Intervillous Fibrin and peri-villous Fibrin changes on Maternal Surface of Placenta, 1(3.3%) had ; Out of 51(3.9%) placentas which had chorioamnionitis, Villous Infarction, Villous agglutination, Intervillous Fibrin and Peri-

villous Fibrin changes on Maternal Surface of Placenta, 1(3.3%) had ; Out of 559(42.9%) Placentas which had Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 8(26.7%) had ; Out of 63(4.8%) placentas which had Villous Infarction, Intervillous Fibrin and Peri-villous Fibrin changes on Maternal Surface, 3(10%) had, Out of 360(27.7%) placentas which had Villous Infarction, Villous Agglutination, Intervillous Fibrin, and Per-villous Fibrin changes in Maternal Surface, 12(40%) had, Out of 75(5.8%) placentas which had Villous Infarction, Villous agglutination, Intervillous Fibrin, Per-villous Fibrin and Intervillous Thrombus changes in Maternal Surface, 2(6.7%) had, Out of 92(7.1%) placentas which had Normal Maternal Surface of placenta 1(3.3%) Placentas had Placenta Previa. The Association between Placenta Previa and HPR of Maternal Surface of Placenta was not statistically significant($P>0.05$).

2. DECIDUAL ARTERIOPATHY

Fundal Location

Atherosclerosis, Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 31(10.7%) had, Out of 158(12.2%) placentas which had Atherosclerosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 34(11.7%) had, Out of 82(6.3%) placentas which showed Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries, 23(7.9%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries, 26(9%) had, Out of 579(44.5%) placentas which had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 117(40.3%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 25(8.6%) had, Out of 162(12.5%) patients which had normal decidual arteries

in Placenta, 34(11.7%) had Fundal Implantation of Placenta. The association between Fundal Location of Placenta and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

Right Lateral Location

Atherosis, Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 5(7.6%) had, Out of 158(12.2%) placentas which had Atherosis, Fibrinoid Necrois, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 11(16.7%) had, Out of 82(6.3%) placentas which showed Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries, 3(4.5%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries,6(9.1%) had, Out of 579(44.5%) placentas which had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 30(45.5%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 5(7.6%) had, Out of 162(12.5%) patients which had normal decidual arteries in Placenta, 6(9.1%) had Right Lateral Implantation of Placenta. The association between Right Lateral Location of Placenta and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

Left Lateral Location

Atherosis, Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 4(8.2%) had, Out of 158(12.2%) placentas which had Atherosis, Fibrinoid Necrois, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 4(8.2%) had, Out of 82(6.3%) placentas which showed Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries, 3(6.1%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries, 6(12.2%) had, Out of 579(44.5%) placentas which had Fibrinoid

Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 23(46.9%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 2(4.1%) had, Out of 162(12.5%) patients which had normal decidual arteries in Placenta, 7(14.3%) had Left Lateral Implantation of Placenta. The association between Left Lateral Location of Placenta and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

Posterior Location

Atherosis, Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 54(11.2%) had, Out of 158(12.2%) placentas which had Atherosis, Fibrinoid Necrois, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 62(12.8%) had, Out of 82(6.3%) placentas which showed Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries, 33(6.8%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries, 48(9.9%) had, Out of 579(44.5%) placentas which had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 196(40.5%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 27(5.6%) had, Out of 162(12.5%) patients which had normal decidual arteries in Placenta, 64(13.2%) had Posterior Implantation of Placenta. The association between Posterior Location of Placenta and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

Anterior Location

Atherosis, Chorangiostis, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 47(8.3%) had, Out of 158(12.2%) placentas which had Atherosis, Fibrinoid Necrois, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 64(11.3%)

had, Out of 82(6.3%) placentas which showed Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries, 30(5.3%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries, 53(9.4%) had, Out of 579(44.5%) placentas which had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 269(47.6%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 30(5.3%) had, Out of 162(12.5%) patients which had normal decidual arteries in Placenta, 72(12.7%) had Anterior Implantation of Placenta. The association between Anterior Location of Placenta and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

Placenta Previa

Atherosclerosis, Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries are seen in 123(9.5%) placentas, out of which 2(6.7%) had, Out of 158(12.2%) placentas which had Atherosclerosis, Fibrinoid Necrosis, Mural Hypertrophy of Membrane Arterioles of Decidual arteries, 4(13.3%) had, Out of 82(6.3%) placentas which showed Chorangiomas, Fibrinoid Necrosis changes in Decidual arteries, 2(6.7%) had, Out of 122(9.4%) placentas which showed fibrinoid Necrosis change in Decidual arteries, 3(10%) had, Out of 579(44.5%) placentas which had Fibrinoid Necrosis, Mural Hypertrophy Of Membranes arterioles of Decidual Arteries, 15(50%) had, Out of 74(5.7%) placentas which had Mural Hypertrophy of Membrane Arterioles changes in decidual arteries, 1(3.3%) had, Out of 162(12.5%) patients which had normal decidual arteries in Placenta, 3(10%) had Placenta Previa. The association between Placenta Previa and HPR of Decidual Arteriopathy was statistically not significant ($P>0.05$).

FETAL SURFACE

Fundal Location

Out of 19(1.5%) placentas which showed Chorangiomas of fetal surface vessels, 3(1%) had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 125(43.1%) had ; Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiomas changes in fetal Surface, 60(20.7%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 6(2.1%) had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 96(33.1%) had Fundal Implantation of Placenta. The association between Fundal Location of Placenta and HPR of Foetal Surface of Placenta was positive and Statistically significant ($p=0.030$).

Right Lateral Location

Out of 19(1.5%) placentas which showed Chorangiomas of fetal surface vessels, 1(1.5%) had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 29(43.9%) had ; Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiomas changes in fetal Surface, 18(27.3%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 0 had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 18(27.3%) had Right Lateral Implantation of Placenta. The association between Right Lateral Location of Placenta and HPR of Foetal Surface Placenta was Statistically not significant ($P>0.05$).

Left Lateral Location

Out of 19(1.5%) placentas which showed Chorangiomas of fetal surface vessels, 2(4.1%) had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 16(32.7%) had ;

Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiogenesis changes in fetal Surface, 10(20.4%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 0 had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 21(42.9%) had Left Lateral Implantation of Placenta. The association between Left Lateral Location of Placenta and HPR of Foetal Surface of Placenta was Statistically not significant ($P>0.05$).

Posterior Location

Out of 19(1.5%) placentas which showed Chorangiogenesis of fetal surface vessels, 6(1.2%) had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 199(41.1%) had ; Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiogenesis changes in fetal Surface, 112(23.1%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 8(1.7%) had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 159(32.9%) had Posterior Implantation of Placenta. The association between Posterior Location of Placenta and HPR of Foetal Surface of Placenta was Statistically not significant ($P>0.05$).

Anterior Location

Out of 19(1.5%) placentas which showed Chorangiogenesis of fetal surface vessels, 9(1.6%) had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 216(38.2%) had ; Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiogenesis changes in fetal Surface, 159(28.1%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 2(0.4%) had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 179(31.7%) had Anterior Implantation of Placenta. The

association between Anterior Location of Placenta and HPR of Foetal Surface of Placenta was Statistically not significant ($P>0.05$).

Placenta Previa

Out of 19(1.5%) placentas which showed Chorangiomas of fetal surface vessels, 0 had ; Out of 516(39.7%) placentas which showed Clustered Villi at the Fetal Surface, 12(40%) had ; Out of 335(25.7%) placentas which showed Clustered Villi and Chorangiomas changes in fetal Surface, 11(36.7%) had ; Out of 12(0.9%) placentas which showed Clustered Villi, Small Thrombi in Blood vessels of fetal Surface, 0 had ; Out of 419(32.2%) Placentas which showed Normal Fetal Surface, 7(23.3%) had Placenta Previa. The association between Placenta Previa and HPR of Foetal Surface of Placenta was Statistically not significant ($P>0.05$).

LIMITATIONS

In spite of every sincere effort my study has lacunae.

The notable short comings of this study are:

1. The sample size was small. Only 1301 cases are not sufficient for this kind of study.
2. The study has been done in a single Teritiary Health Care Centre.
3. The study was carried out in a tertiary care hospital, so hospital bias cannot be ruled out.
4. The Cross-sectional nature of this study and different sonographers locating the placental Position with variable reporting styles and experience resulting in “observer variation”.

CONCLUSION

In our study, out of 1301 patients, most of the patients were 21-30 years of age. The majority of patients included in the study were multigravida. Maximum number of patients underwent LSCS.

The results of the study showed the association between Placental Implantation detected after 28 weeks of gestation and their Maternal-Foetal Outcomes, Blood grouping and Rh typing, and HPR of placentas. Over-all the most common placental location was Anterior, while the most common Blood group was O +VE. Most of the patients had Preterm Delivery, while most of the Neonates were admitted in NICU.

We have seen that there was a positive and significant association between Fundal implantation of Placenta and Severe Pre-Eclampsia and Premature Rupture of Membranes. The possible basis is Fundal implantation of placenta places the weakest point of membrane over the cervical OS and thus increases the risk of developing Premature Rupture of Membranes. There was positive and significant association between left lateral position and Severe Pre-Eclampsia.

There was a positive and statistically significant association seen between HPR of Foetal Surface of Placenta.

In conclusion of our study, the implantation of placenta at 18-22 weeks can be used as a tool to evaluate pregnancies and categorize them into high-risk groups for maternal complications and foetal outcomes. Severe Pre-Eclampsia, Premature Rupture of membranes have significant association with Fundal location of Placenta. Left Lateral Location of Placenta has significant association with Severe Pre-Eclampsia. Therefore this study is complimentary to the hypothesis

that Placental Location and Pregnancy Outcomes are interlinked. However this observation needs additional research to confirm the observations.

SUMMARY

In our study 1301 patients attending in-patient department of Obstetrics and Gynecology, BLDE (Deemed to be University), Shri B. M. Patil Medical College, Hospital and Research Centre. The study was conducted to study, the impact of Placental Location on Maternal and Foetal Outcome, association between Placental Location and Blood group and Rh type, association between Placental Location and HPR of placenta.

1. The most common age group was 21-30 years with 75.9%, followed by 14.3% were <20 years and 9.8% were ≥ 31 years.
2. The most common Placental location was Anterior, followed by Posterior, Fundus, Right Lateral, Left Lateral and Placenta Previa.
3. Out of all the blood groups and Rh types O+VE is more common
4. Most of the patients were Multigravidas when compared to Primigravida.
5. The most common maternal complication we observed in our study was preterm delivery, followed by Oligohydramnios, Severe Pre-Eclampsia, Premature Rupture of Membranes, Eclampsia, Abruptio, Foetal Growth Restriction, and Mild Pre-Eclampsia.
6. Among the Neonates Delivered in our study highest number of them had NICU admissions. while there were no IUD's observed.
7. Majority of the Patients underwent LSCS when compared to Vaginal and Instrumental Delivery.
8. Among the Histopathological changes seen in Maternal Surface of Placenta, Decidual Arteriopathy most of them were in Anterior location compared to Fundal, Right Lateral, Left Lateral, Posterior, Placenta Previa, it was not statistically Significant.

9. We found that, most of the patients had O +VE Blood Group and Rh Type in Anterior location compared to Posterior, Fundus, Left Lateral, Right Lateral and Placenta Previa but this was not statistically significant. Most of the patients had Multi Obstetric History in Anterior compared to Posterior, Fundus, Right Lateral, Placenta Previa and Left Lateral it was not statistically significant
10. Our study showed that, majority of patients had Mild Preeclampsia in Anterior compared to Fundus, Right Lateral, Left Lateral, Posterior, Placenta Previa though it was not statistically significant.
11. More number of patients had Eclampsia in Anterior and Posterior locations compared to Fundus, Right Lateral, Left Lateral and Placenta Previa locations but this was not statistically significant.
12. More number of patients had Abruptio in Anterior and Posterior locations compared to Fundus, Right Lateral, Left Lateral and Placenta Previa locations but this was not statistically significant.
13. More number of patients had Foetal Growth Restriction in Anterior location compared to Fundus, Right Lateral, Left Lateral, Posterior and Placenta Previa locations but this was not statistically significant.
14. More number of patients had Polyhydramnios in Posterior location compared to Fundus, Right Lateral, Left Lateral, Anterior and Placenta Previa locations but this was not statistically significant.
15. More number of patients had Oligohydramnios in Anterior location compared to Fundus, Right Lateral, Left Lateral, Posterior and Placenta Previa locations but this was not statistically significant.

16. More number of patients had Preterm Delivery in Posterior location compared to Fundus, Right Lateral, Left Lateral, Anterior and Placenta Previa locations but this was not statistically significant.
17. In our study Majority of patient's neonates had NICU admissions in Anterior Location compared to Posterior, Fundal, Right Lateral, Left Lateral and Placenta Previa Locations but this was not Statistically significant.
18. In our study Majority of patient's neonates who were given mother-side in Anterior Location compared to Posterior, Fundal, Right Lateral, Left Lateral and Placenta Previa Locations but this was not Statistically significant.
19. In our study Majority of patient's neonates who were given Perinatal Death in Anterior and Posterior locations, but this was not Statistically significant.
20. In our study Majority of patients who delivered Fresh still Birth in Anterior and Posterior Locations compared to Posterior, Fundal, Right Lateral, Left Lateral Locations but this was not Statistically significant.
21. In our study Majority of patients who delivered Macerated still Birth in Posterior Locations compared to Anterior, Fundal, Left Lateral Locations but this was not Statistically significant.

RECOMMENDATIONS

1. More Studies to be done on this hypothesis, so that we can include placental location as one of the parameters to categorise pregnant women into high-risk groups and monitor them regularly for better pregnancy outcomes.
2. A multicentric study from various Hospitals would help us to study more number of patients, maternal complications and foetal complications. In association with placental location.
3. Adding Doppler Velocimetry to the study would help us to confirm and analyse the actual pathology involved in the association between placental location and maternal complications and foetal outcomes.

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ANNEXURE 1 -ETHICAL CLEARANCE



B.L.D.E. (DEEMED TO BE UNIVERSITY)

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

IEC/20-09/2021
Date-22/01/2021


INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11-00 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A cross sectional study to evaluate the impact of placental location on maternal and fetal outcome.

Name of PG student: Dr Naveena Alakonda.
Department of Obst/Gynaec

Name of Guide/Co-investigator: Dr Neelamma Patil, Professor of
Obst/Gynaec


DR. S.V. PATIL
CHAIRMAN, IEC

Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

ANNEXURE 2 CONSENT FORM

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/RESEARCH

I, the undersigned, _____, D/O W/O _____, aged _____ years, ordinarily resident of _____ do hereby state/declare that Dr. ALAKONDA NAVEENA of Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on _____ at _____ (place) and it has been explained to me in my own language that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases. Further Dr. ALAKONDA NAVEENA informed me that he/she is conducting dissertation/research titled “A CROSS SECTIONAL STUDY TO EVALUATE THE IMPACT OF PLACENTAL LOCATION ON MATERNAL AND FETAL OUTCOME.” under the guidance of Dr. NEELAMMA PATIL requesting my participation in the study. Further Doctor has informed me that my participation in this study would help in evaluation of the results of the study which is useful reference to treatment of other similar cases in near future, and I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made photographs video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes. The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis. At the same time, I have been

informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged. After understanding the nature of dissertation or research, diagnosis made, mode of treatment, I the undersigned Smt _____ under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Date:

Place:

ANNEXURE 3 CASE PROFORMA

NAME: IP No:
Age: Case no:
Address: Occupation:
DOA: Contact no.:
DO study: Blood Grouping and Typing:

1. Obstetric History:

Obstetric score:

Gestational age:

2. Past History:

3. Family history:

4. USG findings: Placental location: (a). fundus
(b). right lateral
(c). left lateral
(d). posterior
(e). anterior
(f). placenta previa

5. MATERNAL COMPLICATIONS:

1. Pre-eclampsia:

Mild- YES NO

Severe- YES NO

2. Eclampsia: YES NO

3. Abruption: YES NO

4. Foetal Growth Restriction: YES NO

5. Oligohydramnios: YES NO
6. Preterm: YES NO
7. PROM: YES NO
8. Any other complication: YES NO
9. Mode of delivery: (a). Vaginal
- (b). LSCS
- (c). Instrumental

FETAL OUTCOME:

- (1). NICU: YES NO
- (2). Mother side: YES NO
- (3). Death: YES NO
- (4). IUD:
- (a). Fresh still birth:
- (b). Macerated still birth:
- (5). Neonatal death:
- (a). Early neonatal death (<24hrs):
- (b). Late neonatal death(>24hrs-1week):

HPR OF PLACENTA:

A CROSS SECTIONAL STUDY TO EVALUATE THE PLACENTAL LOCATION ON MATERNAL AND F

ORIGINALITY REPORT

16%
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PRIMARY SOURCES

1 repository-tnmgrmu.ac.in
Internet Source

2 www.ncbi.nlm.nih.gov
Internet Source

3 ijrcog.org
Internet Source

4 dev.biologists.org
Internet Source

5 www.repository.cam.ac.uk
Internet Source

6 royalsocietypublishing.org
Internet Source

7 Elham Keshavarz, Afarin Sadeghian, A Ganjalikhan Hakemi, Fatemeh Talei Khatami
"Prediction of Pre-Eclampsia Development by Placenta Location: A Simple Predictor"
Journal of Obstetrics, Gynecology and Reproductive Health, 2017
Publication

