# "A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"

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**MADRAS MEDICAL COLLEGE,**

**CHENNAI.**

**MAY 2022**

## BONAFIDE CERTIFICATE

This is to certify that the dissertation **"A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"** is a bonafide record of original work done by **DR. K. VIDHYA LAKSHMI** under the guidance of **Dr. S.VIJAYA, M.D., D.G.O.,** Professor & Director of Obstetrics and Gynaecology in Institute of Obstetrics and Gynaecology, Egmore, Chennai in partial fulfillment of the requirements for MS Degree in Obstetrics and Gynaecology branch II examination of the Tamil Nadu Dr.MGR Medical university to be held in May 2022. The period of post graduate study and training from May 2019 to April 2022.

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# DECLARATION

I, **DR. K. VIDHYA LAKSHMI** Post Graduate, Department of Obstetrics and Gynaecology, Madras Medical College solemnly declare that this dissertation **"A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"** was prepared by me at Department of Obstetrics and Gynaecology, Madras Medical College, Chennai, under the guidance and supervision of Professor & Director **Dr. S.VIJAYA, M.D., D.G.O.,** Institute of Obstetrics and Gynaecology, Egmore, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University**, Chennai in partial fulfillment of the University regulations for the award of the degree of **M.S. (Obstetrics and Gynaecology)**.

Place : Chennai
Date :

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**DR. K. VIDHYA LAKSHMI**

# CONTENTS

# INTRODUCTION

Multiple pregnancies, an object of fascination from before the stories of Cain and Abel still present a special challenge for the clinician today. Multiple pregnancy is a well-recognized risk factor that increases maternal and perinatal morbidity and mortality

Maternal mortality is 2.5 times higher in multiple pregnancy when compared to singleton pregnancy and in that line the neonatal morbidity and mortality is 6 times higher for a multiple pregnancy when compared to a singleton.

The major part of the contribution stands with preterm labour and fetal growth restriction. WHO Multicountry Survey on Maternal and Newborn Health (WHOMCS, 2010–2011) has established the association of adverse maternal outcomes associated with multiple pregnancy using the MNM criteria ( Maternal near-miss) , and the data strongly suggest that multiple pregnancy is associated with 3 times higher risk of maternal near-miss and 4 times higher risk of maternal death. The incidence of twin rate has increased upto 70% since 1980 and the rate of triplets , quadruples and higher-order multiple has

increased even more. increased maternal age at conception and the advent of in vitro fertilization techniques has paved the way for the recent increase in the incidence. There is wide international variation in twinning rates, largely driven by differences in the incidence of dizygotic (DZ) twin and multiple pregnancies. While the incidence of monozygotic (MZ) twin pregnancies is relatively constant at 3/1000, dizygotic twinning is increased by a number of factors including maternal age, parity, ethnicity, maternal family history of twins, and smoking. The highest rates of twinning are seen in Sub-Saharan Africa and the lowest frequency is reported in South East Asia and Latin America

Incidence of twins – 1 in 80 pregnancy

Incidence of triplets is 1 in $80^2$ pregnancy

Incidence of quardruples is 1 in $80^3$

Multiple pregnancy is not as simple as two for the price of one – despite the fact that they constitute fewer than 2% of births, In this context, it is clear that all obstetricians need to appreciate the unique development of multiple pregnancies and the importance of specialized antenatal care in their management. The current study aims to asses

sociodemographic, obstetric characteristics and the occurrence of maternal complications in multiple pregnancy and its associated adverse perinatal outcome.

## AIMS AND OBJECTIVES OF THE STUDY

- TO STUDY THE MATERNAL OUTCOME IN MULTIPLE PREGNANCY.

- TO STUDY THE PERINATAL MORBIDITY AND MORTALITY ASSOCIATED WITH MULTIPLE PREGNANCY.

# REVIEW OF LITERATURE

Multiple pregnancy as the name says is the pregnancy carrying more than one fetus. The incidence of which has been in the increasing trend in recent years with the advent of ART .

Among multiple pregnancy, twins is more common.

Binovular twin and uniovular twin are the two varieties of twin

Binovular otherwise known as dizygotic or non identical twins , They develop from 2 different ova need not be from the same ovary and are fertilised separately by two different spermatozoa. The sex can be of the same or different . They have 4 membranes interlacing between them two chorion and two amnion. This type of twin is 4 times common when compared to uniovular twin.

Uniovular or monozygotic twin is pregnancy that has developed from one formed embryo that after fertilisation has divided to form two or more embryo.

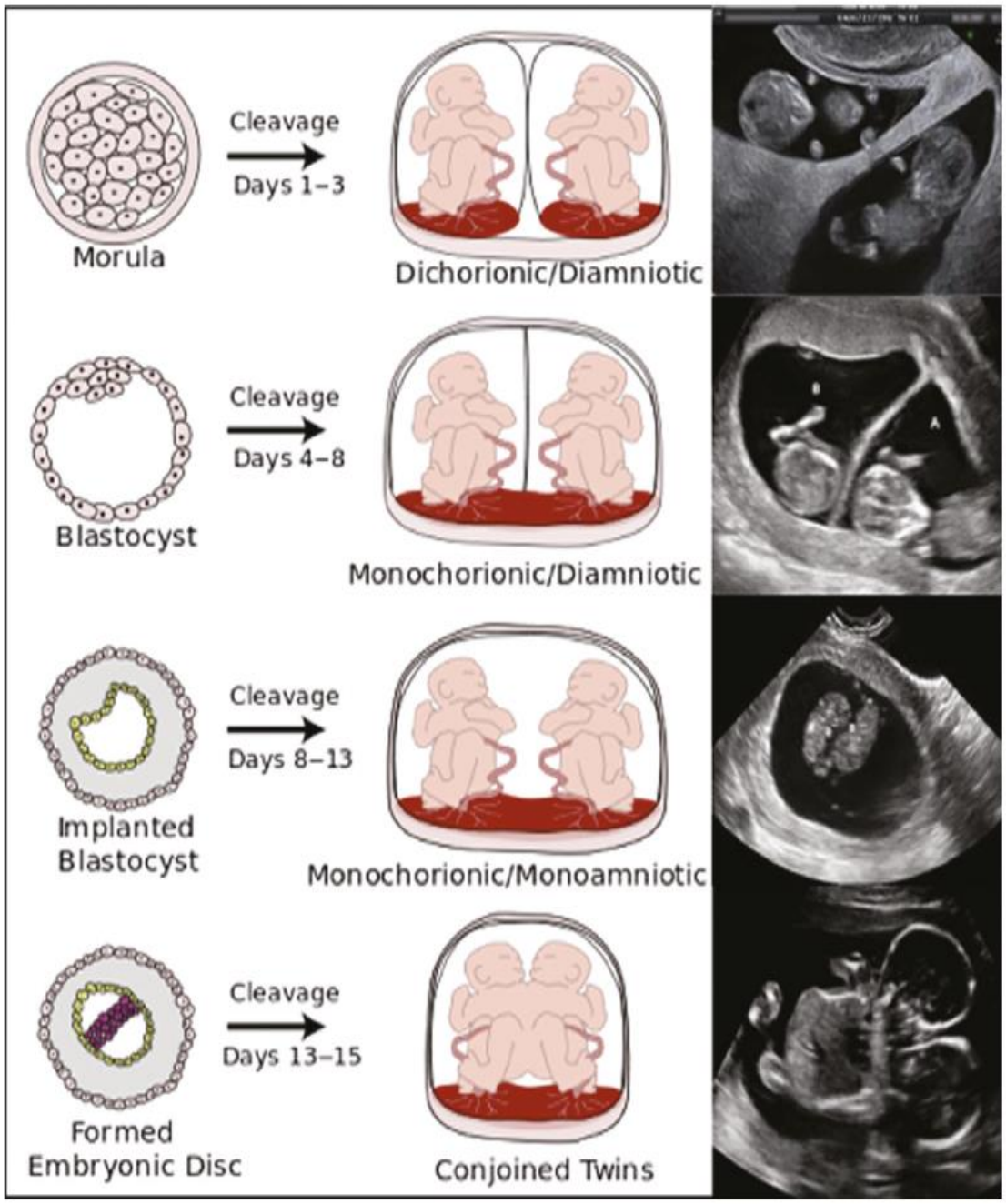
They will be of the same sex, and have the same physical features and blood group

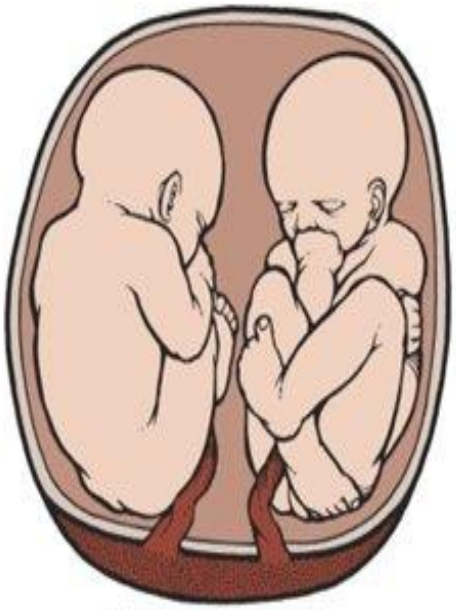
The placentation and development in uniovular twins depends on the day of division .Early division, prior to development of the blastocyst 1-3 days occurs in around 25% of MZ pregnancies and will lead to separate placentas and membranes (dichorionic diamniotic (DCDA) in twins or trichorionic triamniotic (TCTA) in triplets).

Division after the development of a single inner cell mass (day 4–8) will lead to a shared chorion, but separate membrane sacs (monochorionic diamniotic). It is important to recognize that although all monochorionic pregnancies should be monozygous, not all dichorionic pregnancies are dizygous Later division of the embryonic plate is rare, but division between day 8 and 13 will lead to a shared placenta and membrane sac (monochorionic monoamniotic) which is observed in only 1% of all twin pregnancies. Division after 14 days leads to conjoined twins, with an estimated incidence of 1.5/100,000 births.

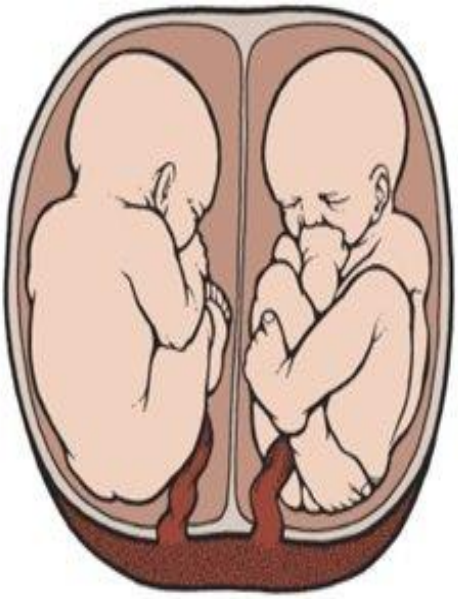
Zygosity has an important role in the clinical outcome of multiple pregnancy Monozygous twins are at higher risk of congenital anomalies, but most of the increased perinatal risks of MZ pregnancies

are related to the fact that most monozygous pregnancies are monochorionic.

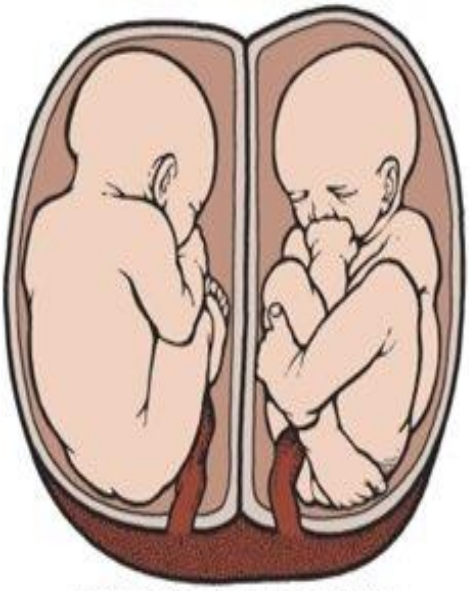




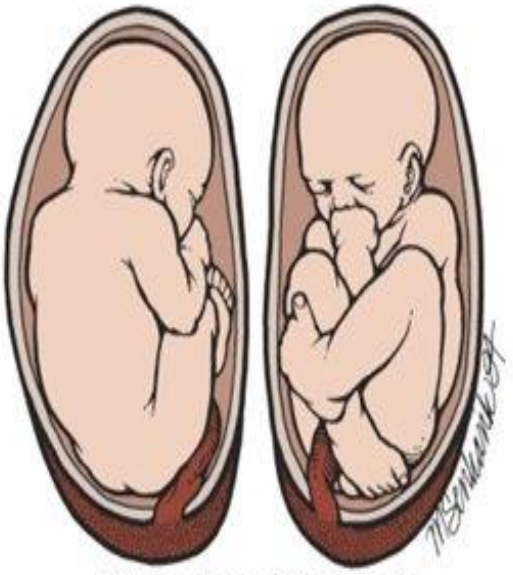
Monochorionic
Monoamniotic



Monochorionic
Diamniotic



Dichorionic Diamniotic
(fused placentae)



Dichorionic Diamniotic
(separate placentae)

# MATERNAL ADAPTATION AND MORBIDITY IN MULTIPLE PREGNANCY

The maternal physiological changes of pregnancy are unsurprisingly greater with multiple pregnancy as the mother adapts to accommodate the greater metabolic demands and physical size of multiple fetuses and placentas.

Higher levels of placentally derived hormones can contribute to a higher frequency of hormonally mediated maternal morbidity. For example, human placental lactogen (hPL) is associated with gestational diabetes and is increased in multiple pregnancy, which correlates with the observed clinical increase in gestational diabetes in multiple pregnancy. Equally, increased human chorionic gonadotropin (hCG) is observed in multiple pregnancies and has been linked to an increased frequency of hyperemesis gravidarum.

**Cardiovascular adaptation** occurs as described in singleton pregnancy and is exaggerated in multiple pregnancy. Plasma volume expansion occurs to a greater degree, leading to reduced oncotic pressure with a higher frequency of both peripheral and pulmonary

edema, and also a greater degree of dilutional anemia. By the time the pregnancy is full term, the weight of the gravid uterus in a twin pregnancy may exceed 8 kg and occupy 10 liters of space. The physical effects of this weight on the pelvis, bladder and back exacerbate the common complaints in pregnancy of pelvic girdle pain and urinary frequency.

Alteration in other body systems has also been reported, such as changes in renal, respiratory and liver function. While this exaggerated maternal response to pregnancy is important for fetal growth of twins, it is also of relevance in the clinical management of the women expecting twins. Physiological changes can raise suspicion and aid in diagnosis of multiple pregnancy.

Different standards of normality have to be set for multiple pregnancies compared with singleton pregnancies, both in the diagnosis of complications, for example anaemia, and in their management, for example preeclampsia when altered renal and liver function is important in monitoring the condition.

**Renal System:**

Glomerular filtration rate in a singleton pregnancy increases by around 50% by the time of 12 weeks. It reaches a peak of approximately around 180 ml/min from the normal value of 120 ml/min. No significant difference in GFR is noted between twin and singleton gestation with regard to GFR.

**Hematologic system:**

Pregnancy is a potential thrombogenic state. Fibrinogen, factors XII, X, IX, VII, VIII and von Willebrand factor levels increase in the blood. Factor XI falls and prothrombin and factor V remain the same. Anticogulants like protein C and antithrombin III levels also falls or remain the same and protein S falls15.

As a result of these changes there is an increased susceptibility towards thrombosis in antenatal period and till 6 weeks postpartum. These changes mainly help in bringing down the loss of blood during delivery.

Dietary intake in twin pregnancy is similar to that of Singleton pregnancy although total fetal weight is greater . The adaptation of the

mother to multiple Pregnancy is thus sufficient to enhance absorption of Nutrients from the diet to optimum levels for fetal growth of the twins. Nutrient handling and metabolism is influenced

By the mother's hormone changes. While broadly similar to those of singleton pregnancies these are enhanced .To favour optimal placental transfer of nutrients to the Developing fetuses with adequate supply of essential factors and energy. For example, there is a slower rate of glucose dispersal after a glucose load in twin pregnancies compared to singletons

DIAGNOSIS OF MULTIPLE PREGNANCY

The tool for determination of chorionicity is first trimester ultrasound by identifying lamda sign and twin peak sign. Dating of multiple pregnancy is another most important point in the management of multiple pregnancy which helps us to avoid postmaturity.

The current ISUOG recommendation is that multiple pregnancy are best dated when the crown-rump length is between 44 and 84 mm (between 11+0 to 13+6 weeks of gestation). Another most important

role of first-trimester ultrasound is the determination of no of a gestational sac, this is even more important in higher-order multiples where selective fetal reduction can be planned.

Assign nomenclature to babies (for example, upper and lower, or left and right) in twin and triplet pregnancies and document this clearly in the woman's notes to ensure consistency throughout pregnancy.

If the woman comes after 14 weeks of her pregnancy then, both CRL is measured and the largest is taken for dating purposes.

Other diagnostic tools include abdominopelvic radiograph, this is particularly useful when the number of an embryo is uncertain in the case of higher-order multifetal pregnancy. However, this tool is inefficient if used before 18 weeks as the skeleton of the fetus is less radio-opaque and cannot be seen accurately

Sometimes blood group determination of the fetuses also help in determining chorionicity. When the two fetuses have different blood group, it confirms the chorionicity as dizygotic whereas presence of same blood group does not confirm monozyosity. St. Clair and

associates suggested that other tests like finger printing may be used for the diagnosis. Study by Ingruise Louse et al showed that dizygotic twin pairs share two HLA haplotypes more commonly than ordinary siblings born out of separate pregnancies and are thus genetically more alike.

Ultrasound indicator of chorionicity

| Gestation visible | Dichorionic diamniotic (DCDA) | Monochorionic diamniotic (MCDA) | Monochorionic monoamniotic (MCMA) |
|---|---|---|---|
| <10 weeks | Two yolk sacs | Two yolk sacs | Single yolk sac |
| <10 weeks | Two amniotic sacs | Two amniotic sacs | Single amniotic sac |
| <10 weeks | Two gestational sacs | Two gestational sacs | Single gestational sac |
| Up to 16–20 weeks | Chorionic peak 'lambda sign' | 'T sign' | No inter-twin membrane visible |
| From 14–16 weeks | Discordant fetal sex | Concordant fetal sex is expected | Concordant fetal sex is expected |
| Throughout pregnancy | Distinct placental masses | Single placental mass | Single placental mass |

In the second trimester, in addition to the detection of the twin condition, advantages of ultrasound examination include reliable estimation of gestational age; exclusion of placenta previa; detection of major structural anomalies, such as spina bifida, acrania, or conjoined twinning; and an opportunity for early bonding between a mother and her children. It is important to remember that later ultrasound is more productive for the detection of structural and/or body stalk

abnormalities and progress of growth than for the optimal detection of chorionicity. For this reason, some experts believe that the improved outcomes that accrue from early detection of any deviation from normal more than compensate for the extra cost of multiple ultrasound examinations.

## FETAL GROWTH AND ANTENATAL MONITORING

Multiples do not simply grow as if they were entirely independent single fetuses, even when they have individual placentation. Although multiples and singletons seem to have similar growth patterns in the second trimester,[24] by the third trimester growth velocity in multiples is consistently found to be less than in singletons.

Since it can be observed that multiple pregnancies are genuinely more at risk of stillbirth and perinatal loss than singletons, the finding that fetuses in a multiple pregnancy are smaller than singletons plausibly reflects an increased prevalence of true growth restriction in multiple pregnancies. There is, however, evidence to suggest that the use of twin-specific charts leads to fewer babies being diagnosed with FGR without failing to identify those small babies that go on to suffer

IUFD or neonatal death.[26,27,28,29] It seems that there is an element of physiological adaptation to a shared intrauterine environment, and growth parameters alone are insufficient to determine those babies most at risk of perinatal complications.

**Frequency of growth assessment**

Most international bodies recommend scanning dichorionic (DC) twins every 4 weeks and monochorionic (MC) pregnancies every 2 weeks, on the basis that MC pregnancies are at greater risk of all adverse perinatal outcomes than DC twins. Longer scan intervals are likely to be associated with a more severe presentation at diagnosis of complications and consequently with poorer outcomes Although additional tests are likely to identify problems earlier, there are significant resource implications for increased screening, particularly in DC pregnancies which represent the majority of twin clinic attendees.

Clinicians must also weigh carefully the risk of missing a struggling fetus against the fact that additional screening, particularly with imperfect diagnostic tools, will inevitably lead to additional iatrogenic deliveries of babies suspected to be compromised. In

multiple pregnancies these iatrogenic deliveries affect not only the mother but also any healthy but potentially immature siblings.

## Schedule of specialist antenatal appointments

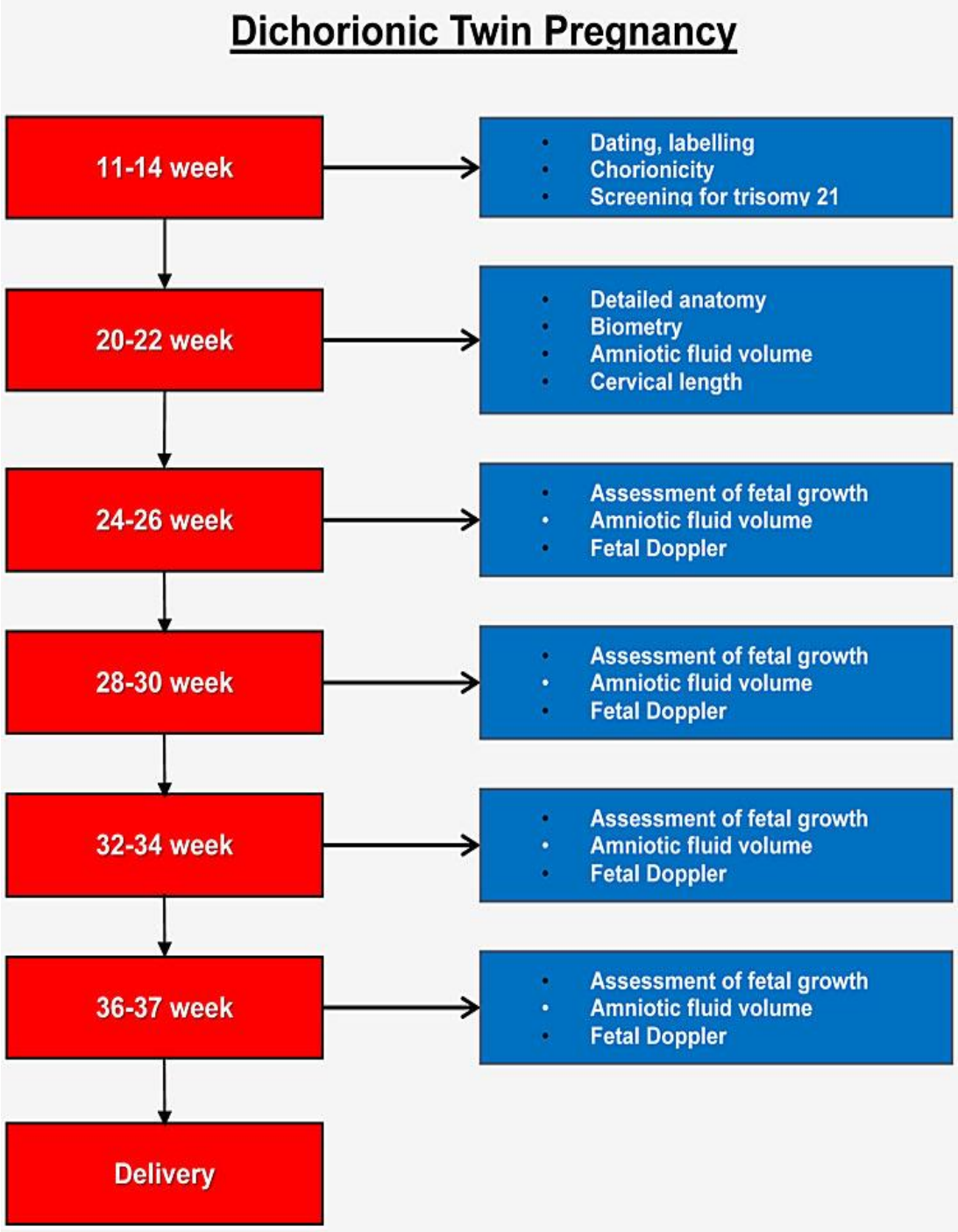
| Type of pregnancy | Weeks 6 to 19 | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
| All pregnancy types below | | | | | | | | | | | | | Anomaly scan ($18^{+0}$ to $20^{+6}$ weeks)** | |
| Monochorionic diamniotic twins | Booking appt by 10 weeks* | | | | | Appt + early scan (approximately $11^{+0}$ to $13^{+6}$ weeks) | | | | | Appt/scan FFTS | | Appt/scan FFTS | |
| Dichorionic twins | | | | | | | | | | | Appt only (no scan) | | | |
| Monochorionic & dichorionic triplets (triamniotic) | | | | | | | | | | | Appt/scan FFTS | | Appt/scan FFTS | |
| Trichorionic triamniotic triplets | | | | | | | | | | | Appt only (no scan) | | | |

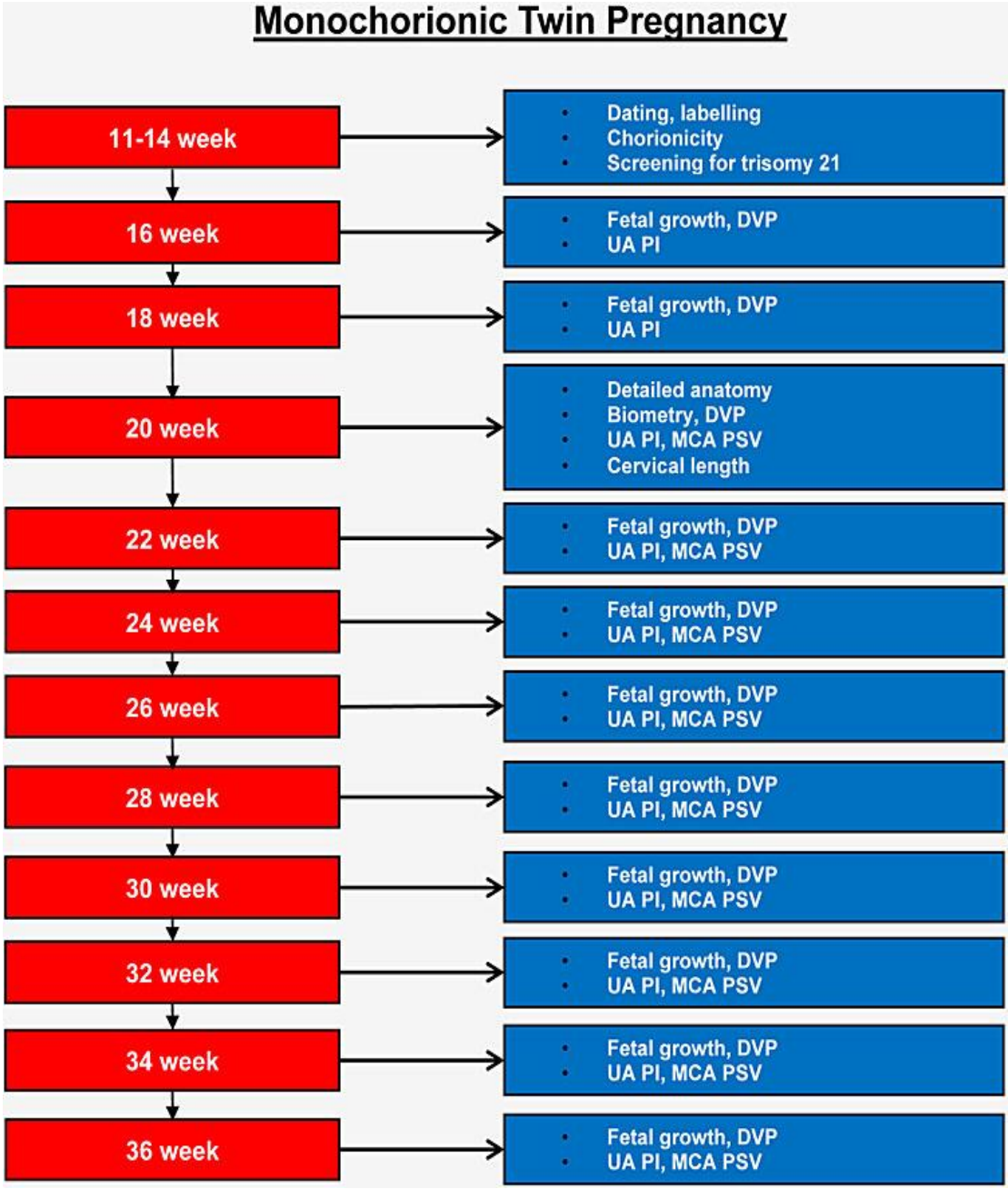
| Type of pregnancy | Weeks 20 to 29 | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 |
| All pregnancy types below | Anomaly scan ($18^{+0}$ to $20^{+6}$ weeks) | | | | | | | | | |
| | Monitor for IUGR at each scan from 20 weeks | | | | | | | | | |
| Monochorionic diamniotic twins | Appt/scan FFTS | | Appt/scan FFTS | | Appt/scan FFTS | | | | Appt/scan | |
| Dichorionic twins | Appt/scan | | | | Appt/scan | | | | Appt/scan | |
| Monochorionic triamniotic & dichorionic triamniotic triplets | Appt/scan FFTS | | Appt/scan FFTS | | Appt/scan FFTS | | Appt/scan | | Appt/scan | |
| Trichorionic triamniotic triplets | Appt/scan | | | | Appt/scan | | | | Appt/scan | |

| Type of pregnancy | Weeks 30 to 37 | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 30 | 31 | 32 | 33 | 34 | 35 | 36 | 37 |
| All pregnancy types below | Monitor for IUGR at each scan from 20 weeks | | | | | | | |
| Monochorionic diamniotic twins | | | Appt/scan | | Appt/scan | | Offer birth<br>If declined: weekly appts + scans | |
| Dichorionic twins | | | Appt/scan | | Appt only (no scan) | | Appt/scan | Offer birth<br>If declined: weekly appts + scans |
| Monochorionic triamniotic & dichorionic triamniotic triplets | Appt/scan | | Appt/scan | | Appt/scan | Offer birth<br>If declined: weekly appts + scans | | |
| Trichorionic triamniotic triplets | | | Appt/scan | | Appt/scan | Offer birth<br>If declined: weekly appts + scans | | |

**Key**

Appt/scan: Appointment plus scan (note that all women should have at least two of their appointments with the specialist obstetrician);
FFTS: Monitor for feto-fetal transfusion syndrome; IUGR: Intrauterine growth restriction


Dichorionic Twin Pregnancy
11-14 week
Dating, labelling
Chorionicity
Screening for trisomy 21
20-22 week
Detailed anatomy
Biometry
Amniotic fluid volume
Cervical length
24-26 week
Assessment of fetal growth
Amniotic fluid volume
Fetal Doppler
28-30 week
Assessment of fetal growth
Amniotic fluid volume
Fetal Doppler
32-34 week
Assessment of fetal growth
Amniotic fluid volume
Fetal Doppler
36-37 week
Assessment of fetal growth
Amniotic fluid volume
Fetal Doppler
Delivery


Monochorionic Twin Pregnancy
11-14 week
Dating, labelling
Chorionicity
Screening for trisomy 21
16 week
Fetal growth, DVP
UA PI
18 week
Fetal growth, DVP
UA PI
20 week
Detailed anatomy
Biometry, DVP
UA PI, MCA PSV
Cervical length
22 week
Fetal growth, DVP
UA PI, MCA PSV
24 week
Fetal growth, DVP
UA PI, MCA PSV
26 week
Fetal growth, DVP
UA PI, MCA PSV
28 week
Fetal growth, DVP
UA PI, MCA PSV
30 week
Fetal growth, DVP
UA PI, MCA PSV
32 week
Fetal growth, DVP
UA PI, MCA PSV
34 week
Fetal growth, DVP
UA PI, MCA PSV
36 week
Fetal growth, DVP
UA PI, MCA PSV

ANTENATAL CARE IN MULTIPLE PREGNANCY

(1) To enhance early diagnosis of multiple pregnancy, specifically the diagnosis of chorionicity;

(2) To institute intensive prenatal, antepartum, and intrapartum care; and

(3) To reduce the risk of adverse maternal, fetal, and neonatal outcomes.

A rational plan of prenatal and intrapartum care can be developed and implemented, either in the setting of a special twin clinic or in the office of an individual practitioner. This plan can and should be based on an awareness of the frequency and the timing of potential adverse events and the need for special educational preparation of mothers. Special written materials about twin pregnancy can be prepared and given to parents, along with recommendations for books to read. A similar suggestion has been successful for singleton pregnancies.

Recently the National Academy of Sciences (NAS) recommended an ideal total weight gain of 35 to 45 lb for twin pregnancy,[28] but provided no definition of excessive weight gain. The

NAS recommendation is useful, however, because it can be the basis of early intervention after initial diagnosis. Indeed, Luke[29] and others[15] have refined this recommendation to include the necessity of gaining 24 lb by the 24th week. Before recommending a comprehensive plan of weight gain, care givers should take into account the maternal prepregnancy body mass index and stature as well as the fact that about one half of twin pregnancies deliver at the 37th week of gestation or earlier. This latter point mandates an early gain, as opposed to waiting until after 32 weeks to try to augment weight.

## NUTRITIONAL CONSIDERATIONS

Two independent studies paralleled the NAS publication and provided an indication of the future direction of subsequent clinical investigations. In one, based on birth/death certificates from the Office of Vital Statistics, Kansas Department of Health and Environment, from the years 1980 to 1996, a total of 922 twin gestations delivered at term were described. It was determined that the proportion of infants born with LBW declined as maternal prepregnancy weight status increased. It was further determined that infant birth weights increased linearly with prenatal weight gain for

women who entered their pregnancy either underweight or at normal weight, but not for those who were overweight or obese at the start of their pregnancy. In another study, optimal pregnancy outcome (greater than 37 weeks' gestation; both infants greater than 2500 g each, with Apgar scores greater than 7) was associated with gestational weight gains of 44 lb (20 kg) compared to 37 lb (16.8 kg) for women with less-than-optimal outcomes.

A companion investigation was conducted by Luke and associates at the Twins Day Festival in Twinburg, Ohio, in the years 1989, 1990, 1991, and 1993. A total of 924 mothers of twins were interviewed, and data were obtained on their 1848 twin children. Study variables were compared by “ideal outcomes” (2500 to 2800 g birth weight and 35 to 38 weeks' gestational age) versus “nonideal outcomes” (birth weight above or below 2500 to 2800 g and/or gestational duration less than 35 weeks).

Ideal twin outcomes had significantly fewer birth weights below the 10th percentile (11% vs 28%; $p < 0.0001$).[40] Further, a significantly greater number of mothers with ideal outcomes did not smoke during their pregnancies (95.5% vs 17.3%; $p = 0.01$); they gained significantly

more weight than mothers with nonideal outcomes (44.8 vs 41.1 lb [$p$ = 0.005] and 1.23 lb/week vs 1.14 lb/week [$p$ = 0.02]). Finally, it was determined that both body mass index and weight gain were positive factors affecting outcome. There was a progressive increase in the odds ratio of an ideal twin outcome with increasing weight gain.

## MATERNAL COMPLICATION

Twin pregnancy remains a common occurrence with an increased risk of adverse outcomes for mother and baby. Excess maternal risks include anemia, urinary tract infection, hypertension, gestational diabetes, hemorrhage and maternal mortality and as such require greater surveillance compared to singleton pregnancies

## MINOR COMPLICATIONS

In the second half of twin pregnancy many might consider the minor complications of pregnancy increased. As a result of the enlarged uterus and increased hormone output, backache and lower abdominal pain become more frequent. As intra-abdominal pressure rises the frequency of micturition, constipation, varicose veins and oedema are all noticed. Sometimes, in late pregnancy it becomes difficult for the woman expecting twins to be comfortable in any

position at rest, and walking is also difficult partly due to locomotor difficulties and partly to breathlessness, as the diaphragm is pushed upwards and splinted. Heartburn can also be a major problem particularly at night, disturbing sleep patterns even further. Although these problems may not be of major concern to obstetric staff, they can be a considerable burden on the woman expecting twins. The obstetrician should adopt a sympathetic approach, reassuring women that these are very common in multiple pregnancy and that they are based in the exaggerated response to multiple pregnancy and will not be a problem following delivery.

**Major Complications**

Antenatal complications of pregnancy, which pose a life threatening risk to the mother to be considered, are hypertensive disorders of pregnancy, antepartum haemorrhage and anaemia. Preterm labour, a very common occurrence in multiple pregnancy, does not usually pose a threat to the mother unless infection intervenes or it arises as a side effect of management with the use of tocolytic agents combined with steroids carrying an increased risk of pulmonary oedema. Other problems in labour namely operative delivery and postpartum haemorrhage will be reviewed.

**Hypertensive Disorders — Preeclampsia**

The incidence of gestational hypertension, preeclampsia and eclampsia has recently been confirmed again to be greater in twin pregnancies when compared to singleton pregnancies, both in primiparae and multiparae (Campbell & MacGillivray, 1999; Coonrod et al., 1995). This was not influenced by the sex of the offspring or zygosity but preeclampsia was more commonly associated with monochorionic placentation. Because of the increased frequency of severe disease and its association with growth retardation, this is a very serious condition for both mother and babies. Special vigilance over a mother expecting twins is needed with more frequent blood pressure checking and urine testing, especially after 30 weeks when weekly checks are indicated. Any single sign of developing preeclampsia, for example, proteinuria alone or a mild rise in blood pressure, should be considered as a reason for hospital admission as the progression of the disease may be very rapid in multiple pregnancy. Additionally proteinuria may present at lower diastolic blood pressures than in singleton pregnancy (Campbell, 1995) and women with proteinuria only in a multiple pregnancy should be treated as having preeclampsia unless it is proven otherwise. Management following admission to hospital is along the lines of management in singleton pregnancy with

frequent monitoring of both maternal and fetal well being to enable optimal timing of delivery. The recent study from Aberdeen (Campbell & MacGillivray, 1999) indicates that for the twins fetal outcome, with respect to growth and mortality, was not significantly poorer in preeclamptic women than normotensive women when gestation at delivery was taken into consideration.

**Antepartum Haemorrhage**

Antepartum haemorrhage is believed to occur more frequently in twin than in singleton pregnancies on account of the greater incidence of preeclampsia with the possibility of placental abruption and the larger area of placental tissue with the likelihood of placental separation. However, the expected increase in incidence of antepartum haemorrhage is not confirmed in all studies (MacGillivray & Campbell, 1988; Patel et al., 1984). A modest increase in antepartum haemorrhage of unknown origin, possibly secondary to better reporting in twin than in singleton pregnancy, has also been shown. No difference in the rates of any type of antepartum haemorrhage by zygosity or placentation has been noted (MacGillivray & Campbell, 1988). Placental abruption and placental praevia should be managed in twin pregnancy in a similar fashion to singleton pregnancy.

Vaginal bleeding in early pregnancy, that is, threatened miscarriage, is not usually life threatening but is nevertheless more common in women who delivered twins than in singletons (MacGillivray & Campbell, 1988; Patel et al., 1984).

**Anaemia**

Bearing in mind the physiological changes in plasma volume and red cell volume the definition of anaemia in a multiple pregnancy requires consideration. Haemoglobin concentration and packed cell volume are unreliable indicators of anaemia. Perhaps more reliance should be placed on mean corpuscular haemoglobin concentration which does not change. The 1983 Scottish Twins Study (Patel et al., 1984) found no difference in the incidence of anaemia in twin and singleton pregnancies when anaemia was defined as haemoglobin concentration less than 9.5 g/dl. Studying 123 twin pregnancies in Grampian over a defined time period (Hall et al., 1979), it was concluded that the incidence of clinically significant anaemia was low after extensive studies including repeat peripheral blood examination and sternal marrow examination. Although both iron and folic acid stores may be reduced transiently during twin pregnancy, the authors

concluded at the time of their writing that prophylactic iron and folic acid should not be recommended for the prevention of anaemia in multiple pregnancy but specific treatment should be given when there was evidence of significant anaemia. However, there is still debate among obstetricians about the need for supplementation. Women in developing countries who often have inadequate or absent iron stores may be susceptible to iron deficiency during a multiple pregnancy and supplementation of such women is required when severe anaemia can be a risk to life.

**Operative Delivery**

Caesarean section rates have been rising steadily over the years in both singleton and multiple pregnancies with rates of well over 50% reported in twins (Cetrulo, 1986). A rise in both elective and emergency caesarean sections in multiple pregnancy has been reported (Campbell & MacGillivray, 1988a). Very high rates of such operative deliveries may well lead to increased maternal mortality and morbidity post delivery, but little has been reported with respect to postnatal complications such as venous thrombosis and embolism.

**Postpartum Haemorrhage**

Because of the increased placental size, uterine over-distension and a greater tendency to uterine atony, it is generally agreed that postpartum haemorrhage is a significant problem for the mother in a multiple pregnancy. Many years ago active management of the third stage with controlled cord traction and use of oxytocic agents was claimed to minimise this risk in twin delivery (Wood & Pinkerton, 1966). Recent reviews of intrapartum management in multiple pregnancy do not comment on the incidence of primary postpartum haemorrhage. Manual removal of the placenta and secondary postpartum haemorrhage were both noted in the Aberdeen series to be more common following twin deliveries (Campbell & MacGillivray, 1988b).

**Gestational diabetes:**

The incidence of GDM increases in twins compared to singletons. 22 to 39% of triplets have gestational diabetes as compared to twins where the rate is 3 to 6%.

## PRETERM BIRTH

Multiple pregnancies are at a greater risk of preterm birth than singletons – in fact, 57% of twin pregnancies are delivered before 37 weeks and 11% before 32 weeks through a combination of increased rates of spontaneous preterm labor and a higher risk of antenatal maternal and fetal complications mandating scheduled preterm delivery. For triplet pregnancies, over 75% will be spontaneously delivered before 35 weeks and the majority of deliveries will be indicated by labor or maternal or fetal compromise. Scheduled delivery is the exception, not the rule, but where triplet pregnancies continue beyond 35 weeks, continuing the pregnancy further is associated with increasing risks of perinatal mortality and morbidity.[3]

The majority of the neonatal morbidity and mortality observed in multiples is attributable to prematurity,[12] and effective prevention of spontaneous preterm delivery in multiples is a key target for reducing the perinatal burden associated with multiple pregnancy.

## PRETERM PREMATURE RUPTURE OF MEMBRANES:

The incidence of PPROM in multiple pregnancy (7.4%) is twice than that in singleton pregnancy (3.7%)44. Rupture takes place in the sac of presentation in majority of cases. The risk of infection, abruption, cord accident, overall perinatal mortality is the same in PPROM in twin as well as singletons.

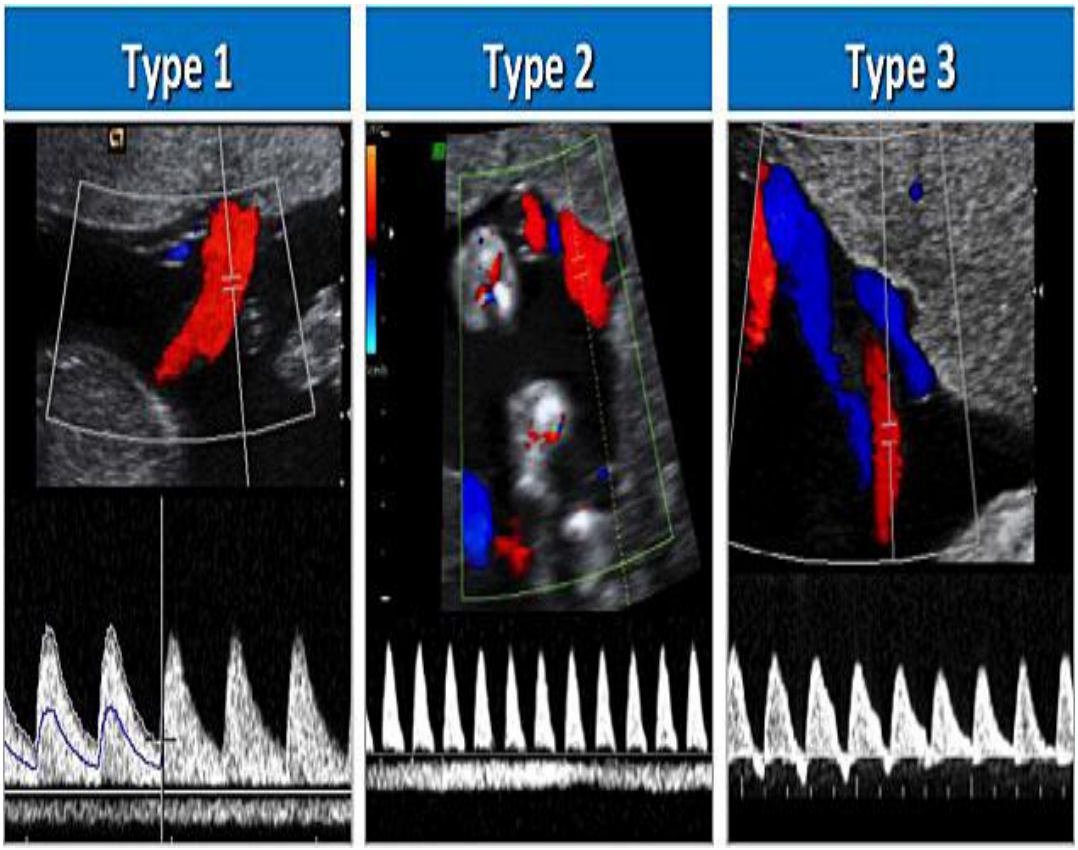
## NEONATAL COMPLICATIONS

GROWTH RESTRICTION

As with most pregnancy complications, the risk of FGR is increased in twin pregnancies, and more so in monochorionic (MC) twin pregnancies. Where one fetus is identified as small and the other is not, this is termed selective fetal growth restriction (sFGR). sFGR occurs in both DC and MC pregnancies but the shared placental circulation in MC pregnancies significantly affects presentation, progression and outcome. An increase in perinatal adverse outcomes can be observed when the fetal growth discrepancy is only 18%, even after the exclusion of TTTS pregnancies.[58] Most national bodies recommend a discrepancy of 20–25% as a trigger for referral to fetal medicine experts or additional monitoring

**Table 2** The classification, placental anatomy and pregnancy outcomes of selective fetal growth restriction in monochorionic twin pregnancy.

| | Umbilical artery Doppler findings in the smaller twin[62] | Corresponding placental vascular findings | Perinatal mortality[65] |
|---|---|---|---|
| **Type I** | Normal end-diastolic flow (EDF) | Similar to uncomplicated cases | 4.1% (95% CI 1.2–8.7) |
| **Type II** | Absent or reversed EDF (AREDF) | Few large AA anastomoses | 16.1% (95%CI 4.6–32.7) |
| **Type III** | Intermittently absent or reversed EDF (iAREDF) | Many large diameter AA anastamoses | 11.5% (95%CI 7.7–16.0) |



## COMPLICATIONS SPECIFIC TO MONOCHORIONIC PREGNANCIES

### TTTS

TTTS occurs in 10%–15% of all MCDA twins. TTTS mainly occurs because at some point in pregnancy the bidirectional inter-twin flow becomes unbalanced. In most cases, TTTS is associated with the number and/or diameter of AV anastomoses from the donor to the recipient fetus. While the presence of AV anastomoses is the prerequisite for TTTS, other factors, including fetal weight discordance, relative placental growth, cord insertions or fetal cardiac defects may help triggering the disease in individual cases. TTTS is a severe hemodynamic disorder characterized by hypovolemia, oliguria and oligohydramnios in the donor, and hypervolemia, polyuria, and polyhydramnios in the recipient.

Additionally, vasoactive molecules and sustained oliguria lead to hypertension and renal tubular damage in the donor, while transfer of these vasoactive molecules to the recipient is thought to produce hypertension and contribute further to hypertrophic cardiomyopathy. Irrespective of its complex pathophysiology, TTTS is invariably associated with remarkable changes in fetal diuresis that

lead to very obvious differences in the amniotic fluid (AF) deepest pocket and the bladder size of each fetus.

**Diagnostic criteria and staging of severity in TTTS.[29,30]**

| Diagnostic criteria | Severity staging |
|---|---|
| (1) Confirmed monochorionic pregnancy | I The bladder is still visible in the donor twin |
| (2) Polyhydramnios in the recipient with a deepest vertical pocket of 8 cm or more* | II The bladder is no longer visible in the donor |
| (3) Oligohydramnios in the donor with a deepest vertical pocket <2 cm | III Critically abnormal Doppler in either twin: absent-reverse diastolic flow in the umbilical artery of the donor or recipient and/or absent/reverse flow in the DV or pulsatile flow in the UV of the recipient |
| (4) Discordant fetal bladders with markedly enlarged bladder in the recipient and very small or non-visible bladder in the donor during most of the examination | IV Hydrops in either fetus |
| | V Demise of one or both twins |

DV: Ductus venosus; TTTS: Twin-to-twin transfusion syndrome; UV: Umbilical vein.

*The cut-off above 20 weeks is still subject of debate. A cut-off of ≥10 cm beyond 20 weeks has been used in randomized trials and is commonly used by European groups, while a unique cut-off of 8 cm is more commonly used in the United States. Both cut-offs are considered to be acceptable for the diagnosis.

**Twin anemia polycythemia sequence (TAPS)**

TAPS presents in two clinical forms, spontaneously or after laser therapy for TTTS. Spontaneous TAPS occurs in 3%–5% of MCDA twins, normally during the third trimester.[42] TAPS is a form of inter-twin unbalanced transfusion, however, occurring in a placenta where interfetal anastomoses are very small. Thus, there is discordant AV interfetal flow, but the difference with TTTS is that in TAPS the magnitude is much smaller.[43] Chronic subtle transfusion in TAPS leads

to a hematological disorder, that is, anemia-polycythemia, but the severe hemodynamic fetal imbalance of TTTS does not occur. When occurring after laser, TAPS is the result of an incomplete coagulation leaving one or two very small placental vessels. This may occur in 0.5%–6% of cases, depending on definitions, laser technique, and center experience.

**Diagnostic criteria for TAPS.[52]**

| Period | Diagnostic criteria |
|---|---|
| Prenatal | MCA-PSV ≥1.50 MoM in the anemic<br>***and***<br>MCA-PSV ≤0.8 MoM in the fetus with polycythemia<br>***OR***<br>Delta MCA-PSV ≥1.0 MoM |
| Postnatal | Inter-twin hemoglobin difference ≥8.0 g/dL<br>***and***<br>Inter-twin reticulocyte count ratio (anemic/fetus with polycythemia) ≥1.7 |

MCA-PSV: Middle cerebral artery peak systolic velocity as measured with spectral Doppler; MoM: Multiple of the Median; TAPS: Twin anemia polycythemia sequence.

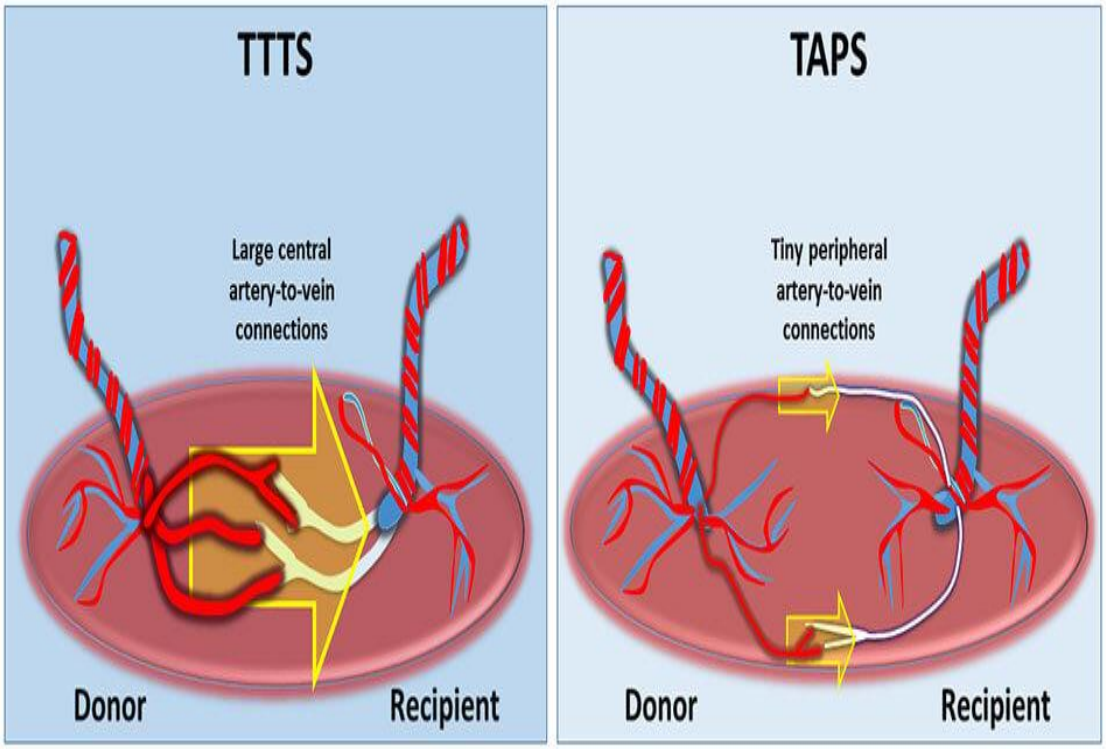
The prognosis in spontaneous cases of TAPS is normally good and most cases can be managed expectantly. However, anecdotal cases of death of the co-twin have been reported. TAPS after laser for TTTS is usually more aggressive and requires therapy. Therapy for TAPS is

indicated if middle cerebral artery Doppler discordance progresses rapidly or pre-hydropic signs are observed in the donor. The only causative treatment is laser therapy. However, in post-laser cases, this option can be difficult sometimes, and repeat transfusions to the donor often achieve good outcomes.

**Acute feto-fetal transfusion by single intrauterine fetal death (sIUFD)**

Single fetal death may occur unexpectedly in approximately 1%–1.5% of uncomplicated MCDA twins, and this is the main reason to recommend elective delivery between 36 and 37 weeks. However, the majority of cases of sIUFD occur in pregnancies complicated with TTTS and sFGR. The risk of brain injury is lower if sIUFD occurs in the first half of gestation or before 28 weeks.[59] Irrespective of the gestational age, the management is based on evaluating the presence of neurological damage in the survivor, preferably by dedicated neurosonography combined with fetal brain MRI around 30 weeks and ideally not before 3 weeks post-sIUFD.

Acute feto-fetal transfusion occurs during pregnancy the consequences can be devastating. The characteristic clinical scenario facilitating this complication is sIUFD. The surviving twin may suffer a massive exsanguination into the circulation of the dying fetus, which is associated with a reported 18%–34% of brain injury, especially if the death occurs beyond 28 weeks, and about 15% of spontaneous co-twin death, with greater risk before 28 weeks. In addition, there is an increased risk of preterm birth with higher incidence in pregnancies complicated by TTTS. The risk of these complications after sIUFD depends on the size of vascular anastomoses and feto-placental mass of the demised twin, and, therefore, it is largely unpredictable.



**LOW BIRTH WEIGHT :**

Low birth weight is also more frequent among twin pregnancies. A previous study found that this risk was 8.3 times higher than in singletons, with a mean birth weight of 2300 g . This risk is associated with the increase in Apgar score at 5th minute < 7 and death during the first year of life .Adequacy of weight for gestational age better assesses the size of the fetus for a given gestational age (compared to birth weight alone). This is particularly useful in populations where preterm birth rates are high. A fetus that is small for gestational age is more likely to experience perinatal morbidity and mortality and adverse effects in adult life. Few studies have evaluated this outcome among twin deliveries, but associations between twin pregnancies and higher rates of small-for-gestational-age have been reported .For these estimations, we used the curves of Fenton et al. because we believed that it was more appropriate to be used when the prevalence of preterm birth is very high, as is the case among twin pregnancies in this population. However, due to the number of cases to have such estimates, it was not feasible to have such assessment performed using different nomograms for comparison.

The risk for low 5th minute Apgar score was three times higher for twin pregnancy (either for the first or second twin) than for singletons. Additionally, it was 1.3 times higher for the second when both twins were compared.

This significantly lower Apgar score for the second twin is always taken into consideration in discussions about the best mode of delivery for twin pregnancies and the time interval between first and second twin, although not justifying an indication for a systematic Cesarean section for twin pregnancies . The higher rates of admission to a neonatal intensive care unit we found have also been reported by previous studies on the topic. Prevalence of fetal death of one of the twins varies from 0,5-6,8% with the worst result for monochorionic pregnancy presenting a high prevalence for this condition (50–70%) and risk for the surviving fetus including the fetal death of this co-twin, neurological morbidity and iatrogenic preterm delivery. In the current study, we have not data on chorionicity, however fetal death (death after 28 weeks) occurred over 1.5 times (3.6%) for the first twin and almost 3 times (5.7%) for the second twin when compared to singletons (2.0%).

Perinatal death has been described as up to four times higher in twin pregnancies than in singletons, mainly due to preterm birth, fetal growth restriction, low Apgar scores and extremely low birth weight. In our study, it was found to be 2.5 times higher for the first twin and 3.5 for the second one. This difference between both twins has already been described . In the current study, we also observed a higher risk for fetal and early neonatal death, supporting previous findings from other studies.

# MATERIALS AND METHODS

## STUDY SETTING:

Institute of Obstetrics and Gynaecology,

Madras Medical College,

Egmore ,Chennai.

**STUDY PERIOD**: 12 Months (February 2021 - January 2022)

**SAMPLE SIZE: 200**

## SAMPLING TECHNIQUE:

Prospective study.

## INCLUSION CRITERIA:

All the women with multiple pregnancy completing 28 weeks of gestation and had delivered in our hospital.

## EXCLUSION CRITERIA:

Those multiple pregnancies who were admitted for observation

- Multiple pregnancy with gestational age <28 weeks

- Women with pre-existing medical disorders like chronic hypertension, overt diabetes mellitus, cardiac disease, renal disease .
- Patient refusal

**MATERIALS AND METHOD:**

- It is a prospective observational study on 100 cases of twins admitted at our hospital
- Detailed history will be taken, general and systemic examination will be done, all routine and specific investigations will be done.
- Data will be collected by proforma containing demographic details, present ,past, family history, antepartum, intrapartum, postpartum complications, neonatal outcome and complications and perinatal mortality.
- All these patients were delivered in our hospital Under close observation
- All stages of labour were carefully managed in the presence of team of obstetricians.
- All babies were examined by neonatologist and NICU care was given as and when required

- Necessary information will be collected in a pre designed data sheet and finally the findings will be compiled and analysed

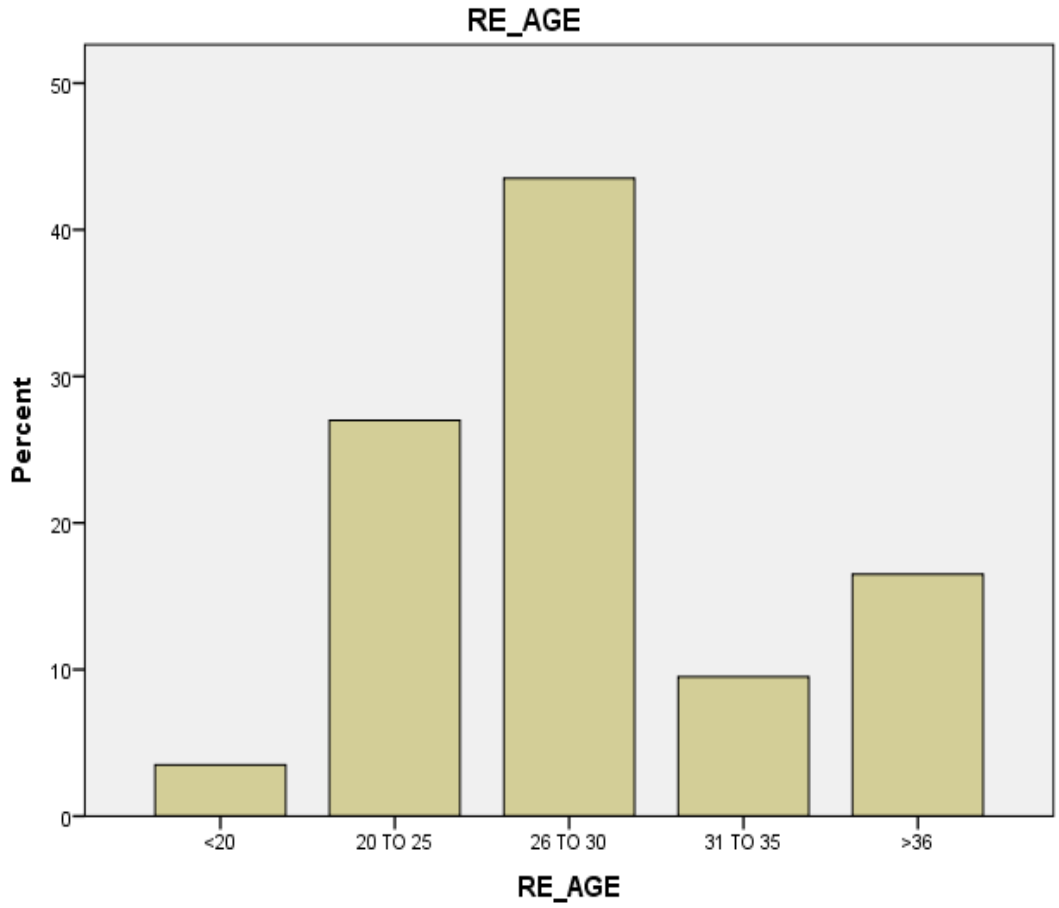
# RESULTS

Table 1 showing distribution of 200 mother with multiple pregnancy with respect to age

TABLE 1:AGE DISTRIBUTION

| **Age distribution** | | | | | |
|---|---|---|---|---|---|
| | | Frequency | Percent | Valid Percent | Cumulative Percent |
| | <20 | 7 | 3.5 | 3.5 | 3.5 |
| | 20 TO 25 | 54 | 27.0 | 27.0 | 30.5 |
| | 26 TO 30 | 87 | 43.5 | 43.5 | 74.0 |
| | 31 TO 35 | 19 | 9.5 | 9.5 | 83.5 |
| | >36 | 33 | 16.5 | 16.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

Figure 1 showing age distribution of 200 cases of multiple pregnancy



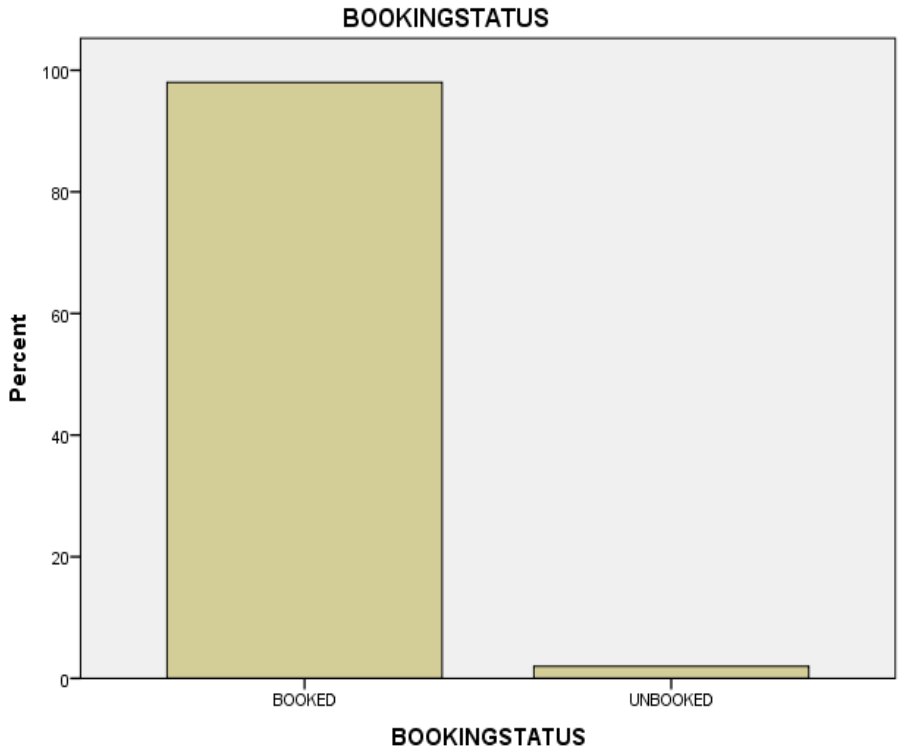

From the above figure, 43% of cases are between 26-30 years of age group, next maximum lies in the range of 20-25. Out of 200 cases,33 cases fall under the age cut off of >36 years which correlates with long period of infertility and conceived after artificial reproductive techniques.

Table 2 shows the booking status of 200 cases.

**BOOKINGSTATUS**

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| BOOKED | 196 | 98.0 | 98.0 | 98.0 |
| UNBOOKED | 4 | 2.0 | 2.0 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

Figure 2 depicts booking status of 200 cases

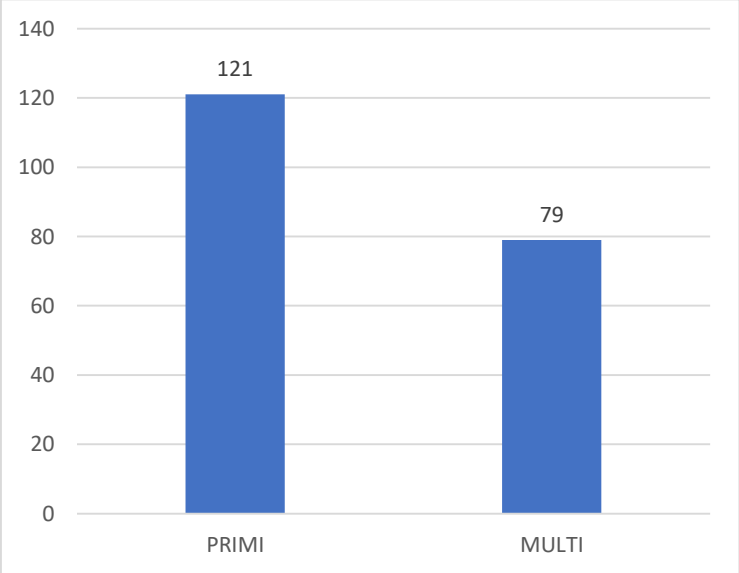



Out of 200 cases, 196 cases are booked and 4 cases were unbooked

TABLE 3 shows the distribution of 200 multiple pregnancy cases with respect to gravida.

| -GRAVIDA | Total | PERCENTAGE |
|---|---|---|
| PRIMI | 121 | 60.5% |
| MULTI | 79 | 39.5% |

Figure 3 depicts the distribution of gravida among 200 cases.




Among 200 cases, 121 cases were primi and 79 were multigravida.

## TABLE 4: GESTATIONAL AGE AT DELIVERY

| GA AT DELIVERY | FREQUENCY | PERCENTAGE |
|---|---|---|
| 28 | 3 | 1.50% |
| 29 | 6 | 3% |
| 30 | 5 | 2.50% |
| 31 | 9 | 5% |
| 32 | 18 | 9.00% |
| 33 | 25 | 13% |
| 34 | 16 | 8.00% |
| 35 | 28 | 14% |
| 36 | 45 | 22.50% |
| 37 | 20 | 10% |
| 38 | 20 | 10.00% |
| 39 | 5 | 9% |

FIGURE 4: BAR DIAGRAM SHOWING GESTATIONAL AGE AT DELIVERY.

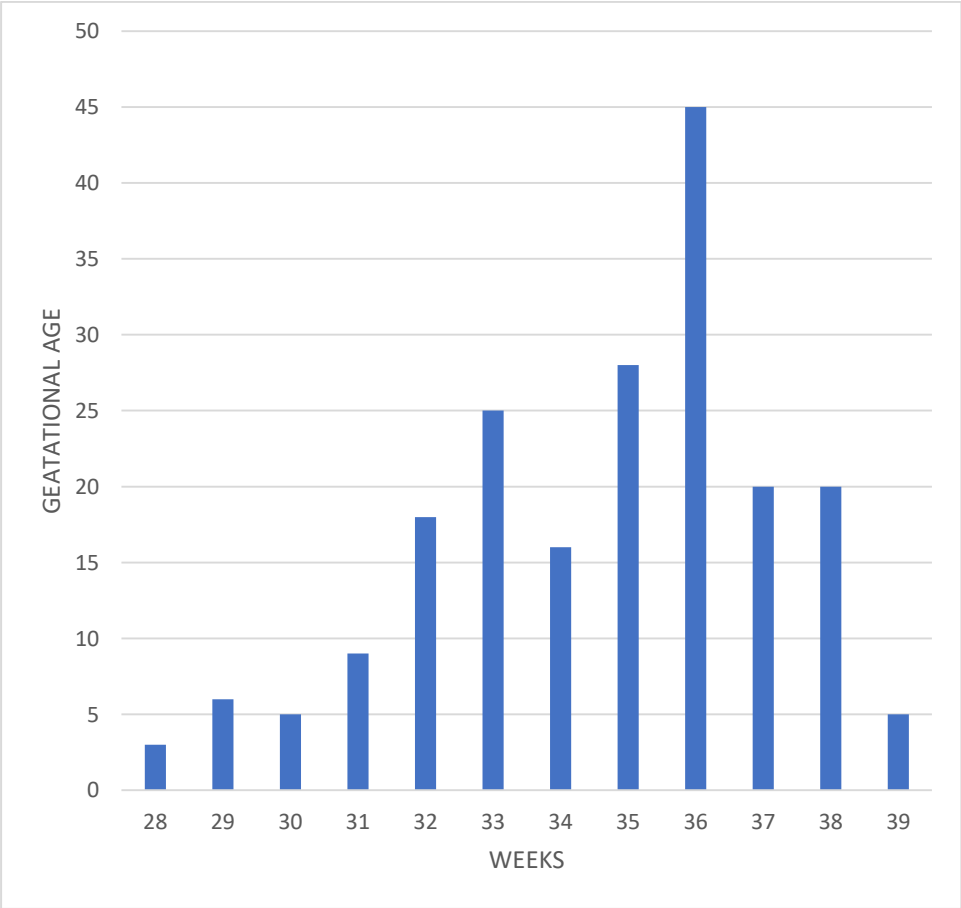



FIGURE 4 depicts the gestational age at delivery of the 200 cases,the mean gestational age being 36 weeks.

**Table 5: MODE OF CONCEPTION**

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| IUI | 12 | 6 | 6 | 6 |
| IVF | 27 | 13.5 | 13.5 | 19.5 |
| OI | 18 | 9.0 | 9.0 | 28.5 |
| S | 143 | 71.5 | 71.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

**FIGURE 5 MODE OF CONCEPTION**

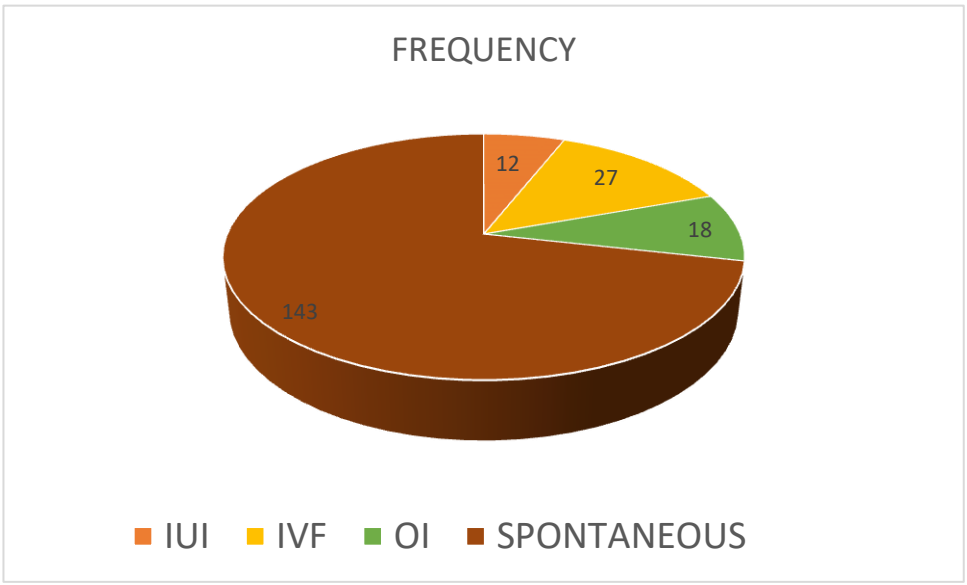


**TABLE 6:CHORIONICITY**

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| DCDA | 126 | 63.0 | 63.0 | 63.0 |
| MCDA | 59 | 29.5 | 29.5 | 92.5 |
| MCMA | 8 | 4.0 | 4.0 | 96.5 |
| TCTA | 7 | 3.5 | 3.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

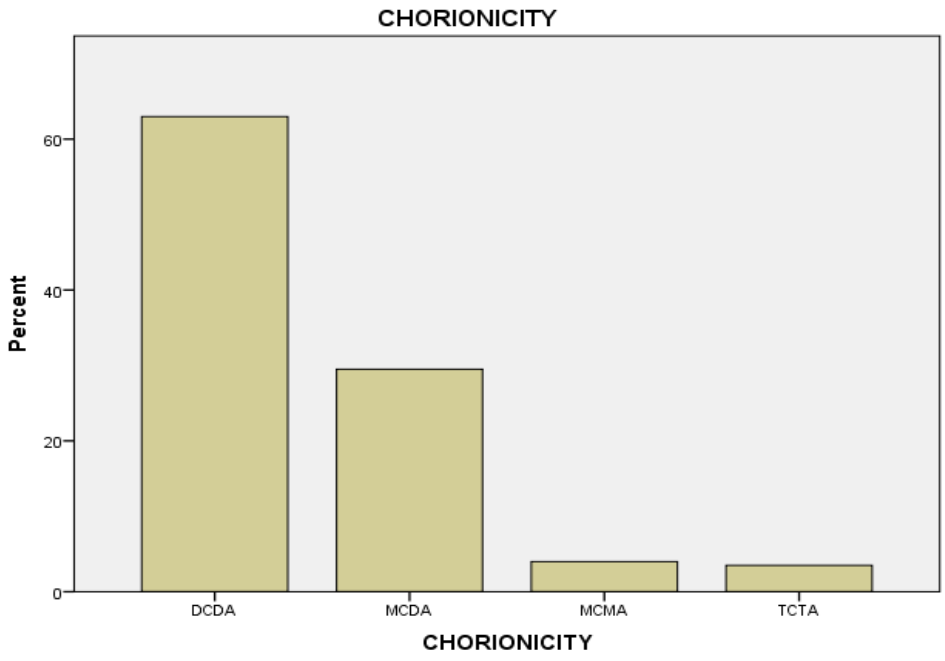



FIGURE 6: bar diagram depicts chorionicity distribution among 200 multiple pregnancy out of which , 63% where dichorionic and diamniotic, 29% were monochorionic and diamniotic and 4% were monochorionic and monoamniotic and 3% were trichorionic and triamniotic pregnancy

Table 7: maternal complications associated with multiple pregnancy.

| MATERNAL COMPLICATION | n | % |
|---|---|---|
| GDM | 14 | 7.0% |
| OVERT DM | 6 | 3.0% |
| GHTN | 24 | 12.0% |
| PRE ECLAMPSIA | 12 | 6.0% |
| AP ECLAMPSIA | 7 | 3.5% |
| PPROM | 17 | 8.5% |
| PROM | 4 | 2.0% |
| LOWHB | 40 | 20.0% |
| FGR | 11 | 5.5% |
| SFD | 9 | 4.5% |
| PLACENTAPREVIA | 3 | 1.5% |
| ABRUPTION | 4 | 2.0% |
| PRETERMLABOUR | 124 | 62.0% |
| CORDACCIDENTS | 4 | 2.0% |
| PPH | 45 | 22.5% |
| RETAINEDPLACENTA | 8 | 4.0% |
| CESAREANHYSTERECTOMY | 3 | 1.5% |

Table 7 depicts various maternal complications and its percentage associated with multiple pregnancy among them , preterm delivery is the most common complication about 62%, and the second one is postpartum hemorrhage 22.5%.

Followed by anemia 20%, other rare complications include single fetal demise, fetal growth restriction weighs about 4.5% and 5.5% respectively, though it is rare it is specific for multiple pregnancy

FIGURE 8: BAR DIAGRAM DEPICTS VARIOUS MATERNAL COMPLICATION.

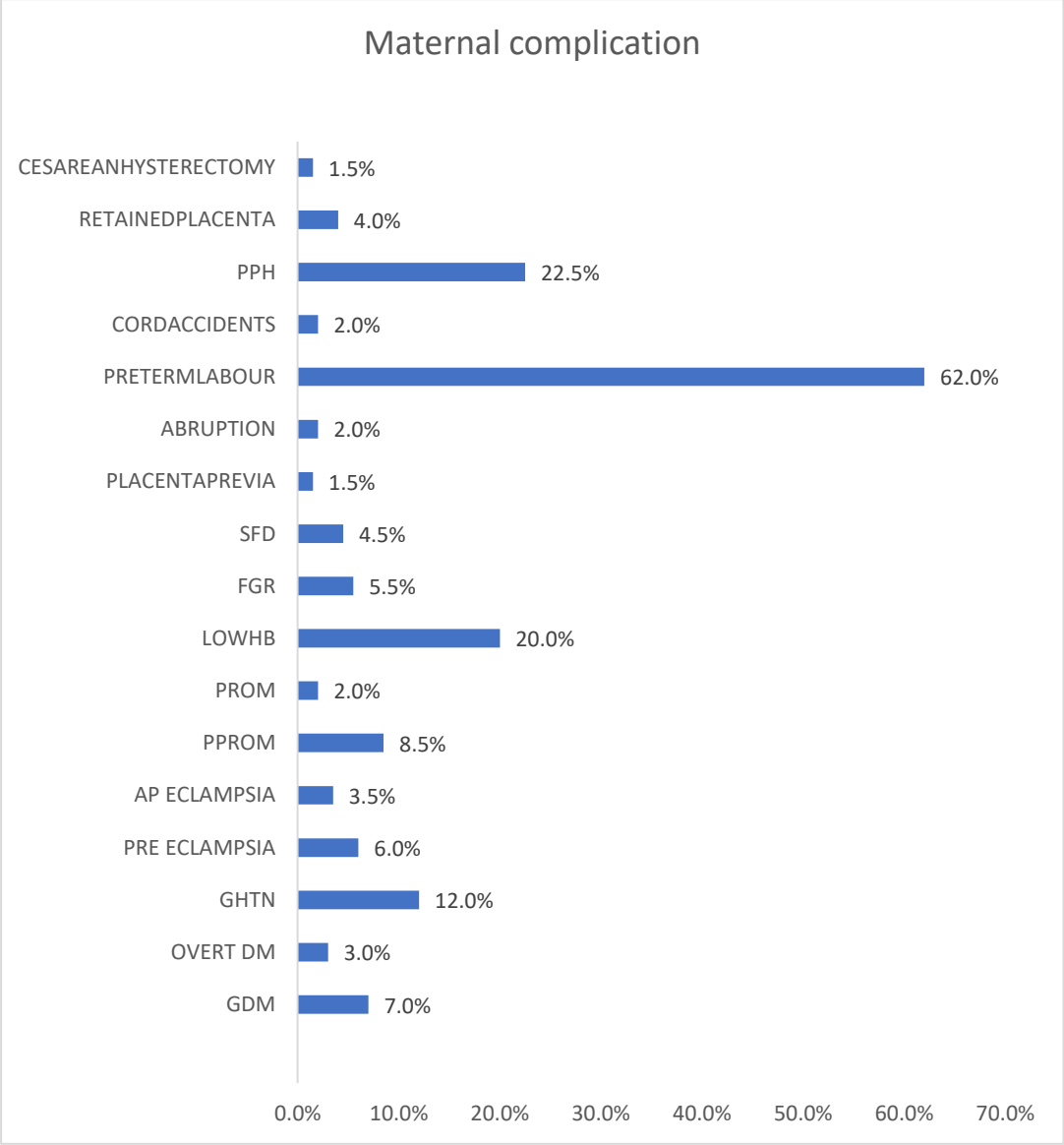


TABLE 8: MODE OF DELIVERY.

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| LN | 61 | 30.5 | 30.5 | 30.5 |
| LSCS | 127 | 63.5 | 63.5 | 94.0 |
| OUTLET | 3 | 1.5 | 1.5 | 95.5 |
| VACUUM | 9 | 4.5 | 4.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

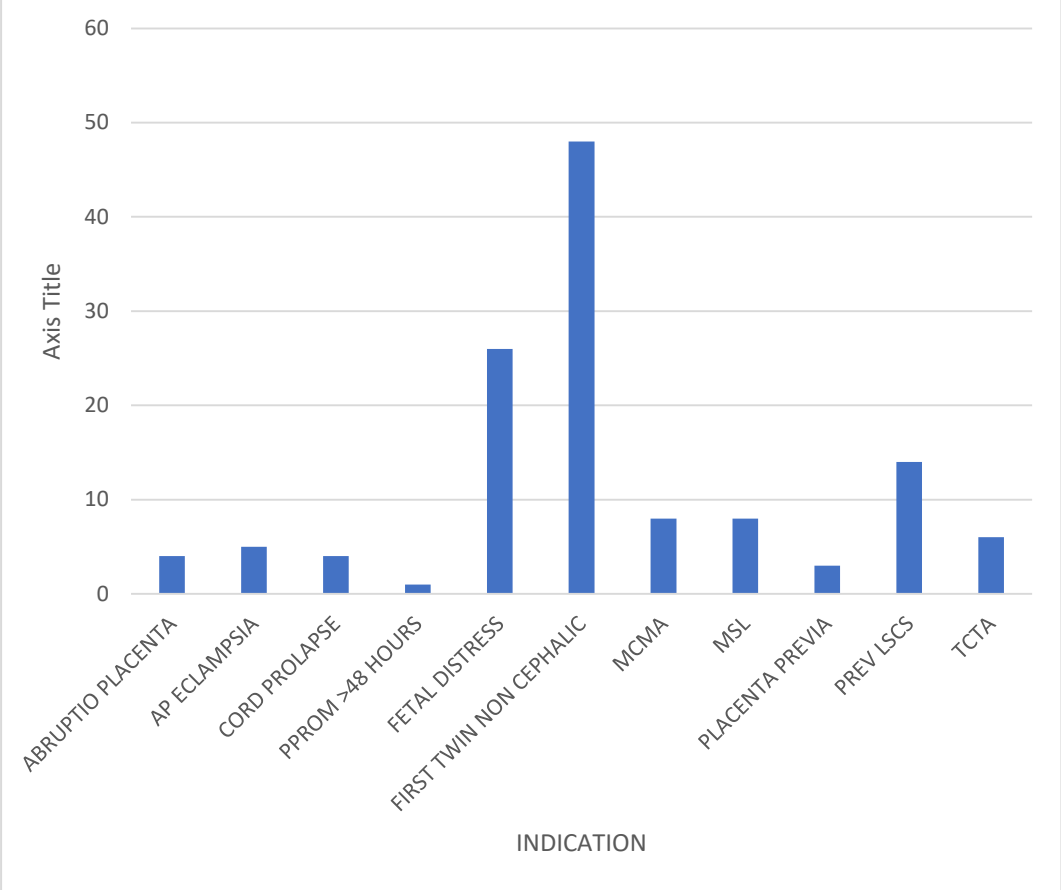
FIGURE 8: BAR DIAGRAM SHOWING MODE OF DELIVERY .


From the above diagram it is shown that cesarean mode of delivery is the most common mode of delivery when it comes to multiple pregnancy.and the most common indication being discussed in the subsequent table.

TABLE 9: INDICATIONS OF CESAREAN DELIVERY

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| ABRUPTIO PLACENTA | 4 | 3.1 | 3.1 | 38.5 |
| AP ECLAMPSIA | 5 | 3.9 | 3.9 | 41.0 |
| CORD PROLAPSE | 4 | 3.1 | 3.1 | 43.0 |
| PPROM >48 HOURS | 1 | 0.7 | 0.7 | 43.5 |
| FETAL DISTRESS | 26 | 20.4 | 20.4 | 59.5 |
| FIRST TWIN NON CEPHALIC | 48 | 37.7 | 37.7 | 80.0 |
| MCMA | 8 | 6.2 | 6.2 | 84.5 |
| MSL | 8 | 6.2 | 6.2 | 88.5 |
| PLACENTA PREVIA | 3 | 2.3 | 2.3 | 90.0 |
| PREV LSCS | 14 | 11 | 11 | 97.0 |
| TCTA | 6 | 4.7 | 4.7 | 100.0 |
| Total | 127 | 100.0 | 100.0 | |

FIGURE 9: BAR DIAGRAM SHOWING INDICATION SPLIT FOR CESAREAN DELIVERY

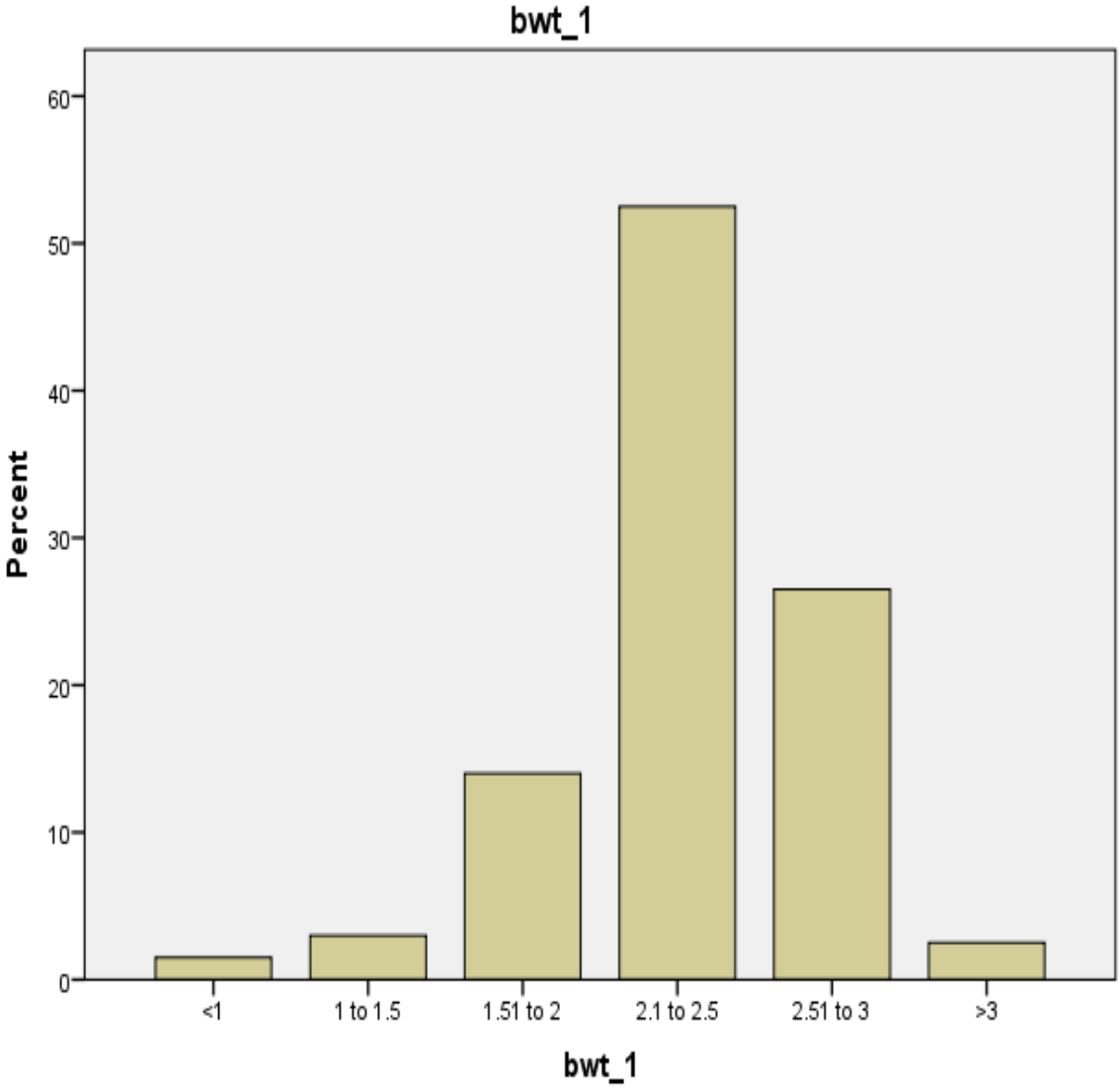



From the above graph , non cephalic presentation of first twin pose the major threat to increase the primary cesarean section rate, followed by fetal distress , all the 6 TCTA triplets were delivered by cesarean mode of delivery keeping in mind the prematurity of the fetus and most of them had growth restriction.

## TABLE 10.1: BIRTH WEIGHT DISTRIBUTION OF TWIN 1

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| <1 | 3 | 1.5 | 1.5 | 1.5 |
| 1 to 1.5 | 6 | 3.0 | 3.0 | 4.5 |
| 1.51 to 2 | 28 | 14.0 | 14.0 | 18.5 |
| 2.1 to 2.5 | 105 | 52.5 | 52.5 | 71.0 |
| 2.51 to 3 | 53 | 26.5 | 26.5 | 97.5 |
| >3 | 5 | 2.5 | 2.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

FIGURE 10.1: BAR DIAGRAM DEPICTING BIRTH WEIGHT DISTRIBUTION OF TWIN 1



## TABLE 10.2: BIRTH WEIGHT DISTRIBUTION OF TWIN 2

| BIRTH WEIGHT | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| <1 | 5 | 2.5 | 2.0 | 2.0 |
| 1 to 1.5 | 8 | 4.0 | 4.0 | 6.0 |
| 1.51 to 2 | 40 | 20.0 | 20.0 | 26.0 |
| 2.1 to 2.5 | 96 | 48.0 | 48.0 | 74.0 |
| 2.51 to 3 | 45 | 22.5 | 22.5 | 96.5 |
| >3 | 6 | 3.5 | 3.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

FIGURE 10.2: BAR DIAGRAM SHOWING BIRTH WEIGHT DISTRIBUTION OF TWIN 2

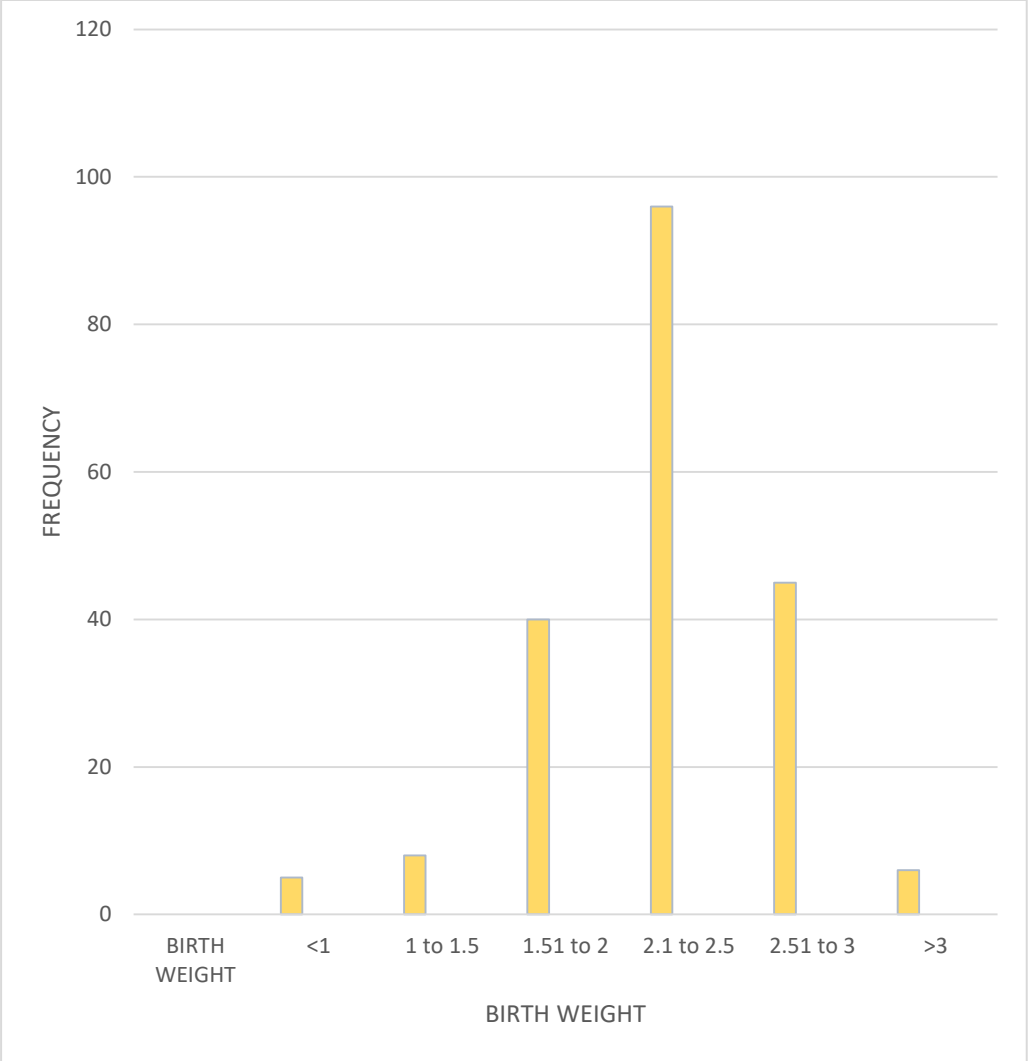
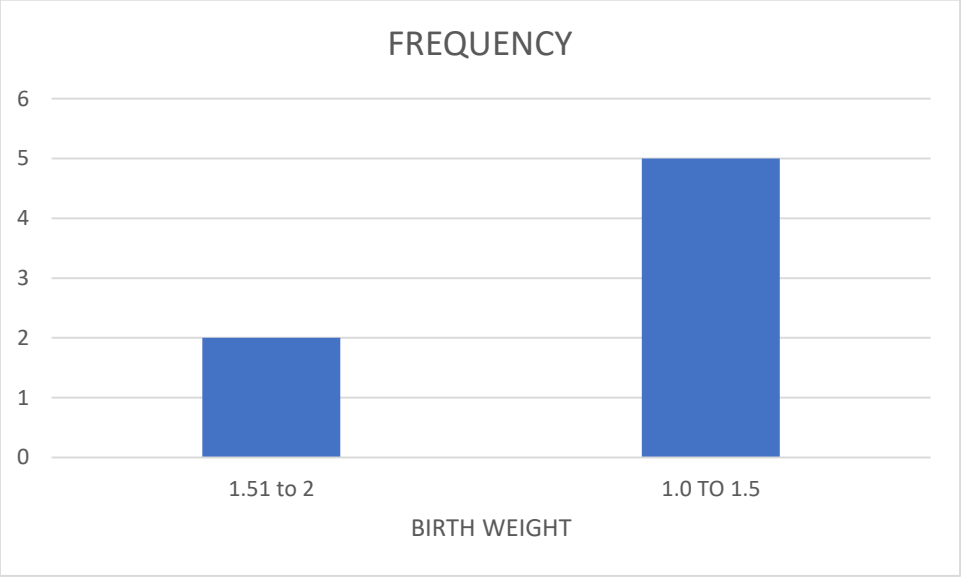


TABLE 10.3 : BIRTH WEIGHT DISTRIBUTION OF TRIPLET 3.

| | Frequency | Percent |
|---|---|---|
| 1.51 to 2 | 2 | 28.5 |
| 1.0 TO 1.5 | 5 | 71.5 |
| Total | 7 | 100 |

FIGURE 10.3: BAR DIAGRAM DEPICTING BIRTH WEIGHT OF TRIPLET 3




From the above 3 table it is evident that birth weight of twin babies mostly fall in the range of 2.1 to 2.5 this can be extrapolated to the preterm nature of the babies of multiple pregnancy.

TABLE 11.1: APGAR OF TWIN 1

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| <5,5 | 18 | 9.0 | 9.0 | 9.0 |
| 5,5 to 8,8 | 161 | 80.5 | 80.5 | 89.5 |
| >8,8 | 21 | 10.5 | 10.5 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

**TABLE 11.2: APGAR OF TWIN 2**

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| <5,5 | 20 | 10.0 | 10.0 | 10.0 |
| 5,5 to 8,8 | 162 | 81.0 | 81.0 | 91.0 |
| >8,8 | 18 | 9.0 | 9.0 | 100.0 |
| Total | 200 | 100.0 | 100.0 | |

TABLE 11.3: APGAR OF TRIPLET 3

| | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|
| **<5,5** | 3 | 42.8 | 42.8 | 42.8 |
| 5,5 to 8,8 | 4 | 57.1 | 57.1 | 57.1 |
| >8,8 | 0 | | | |
| Total | 7 | 100.0 | 100.0 | |

Figure 11.1: APGAR OF TWIN 1

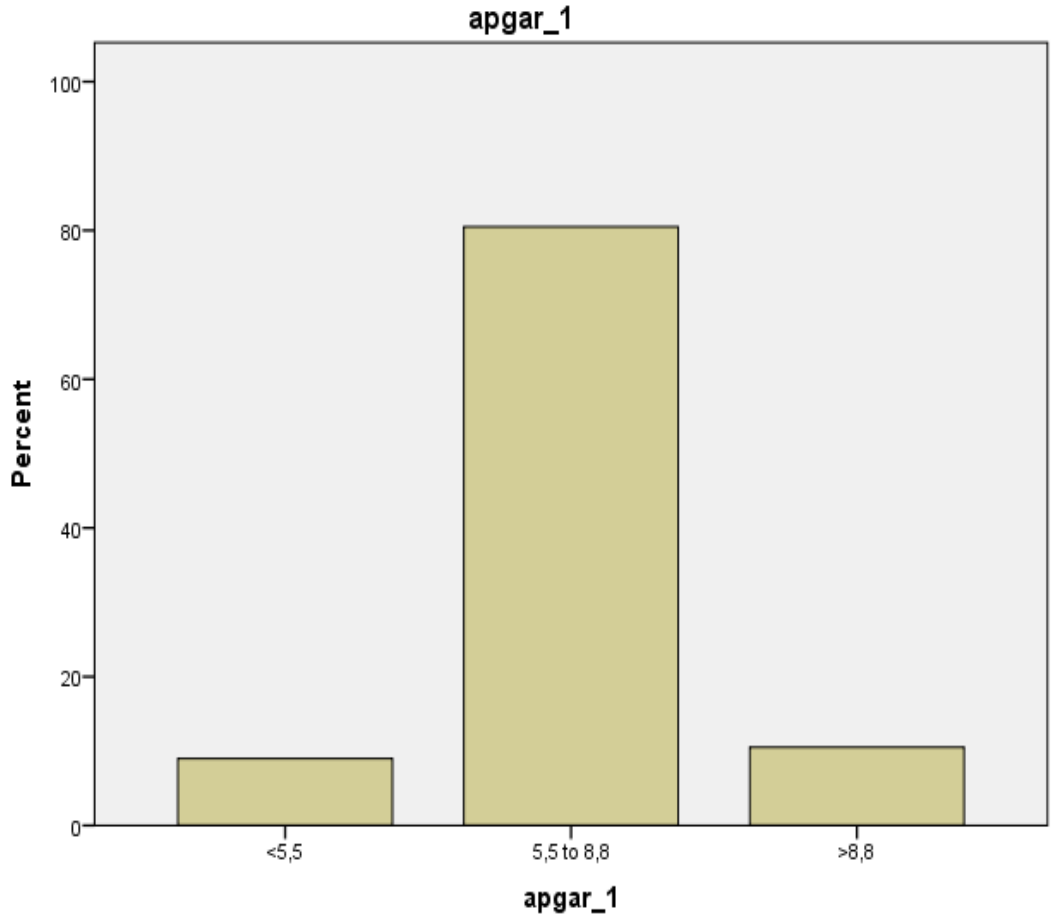


FIGURE 11.2: APGAR OF TWIN 2

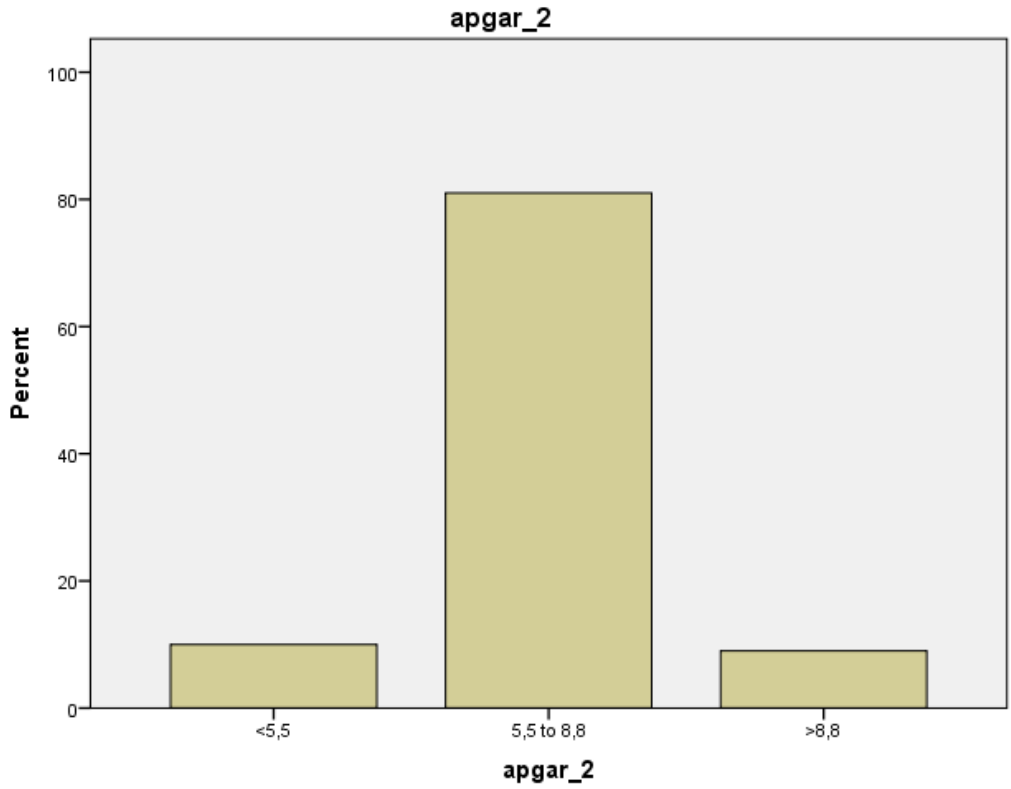
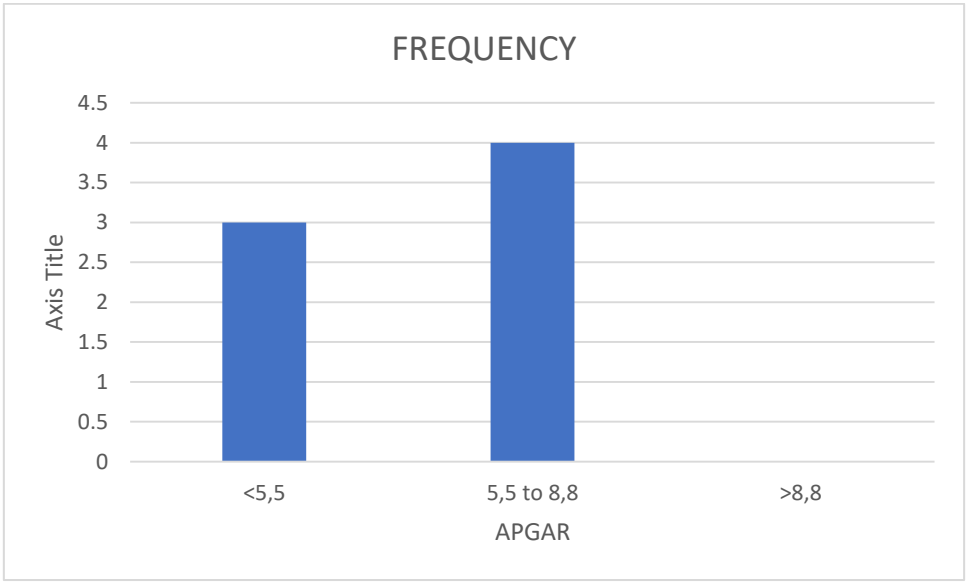



FIGURE 11.3: APGAR OF TRIPLET 3.

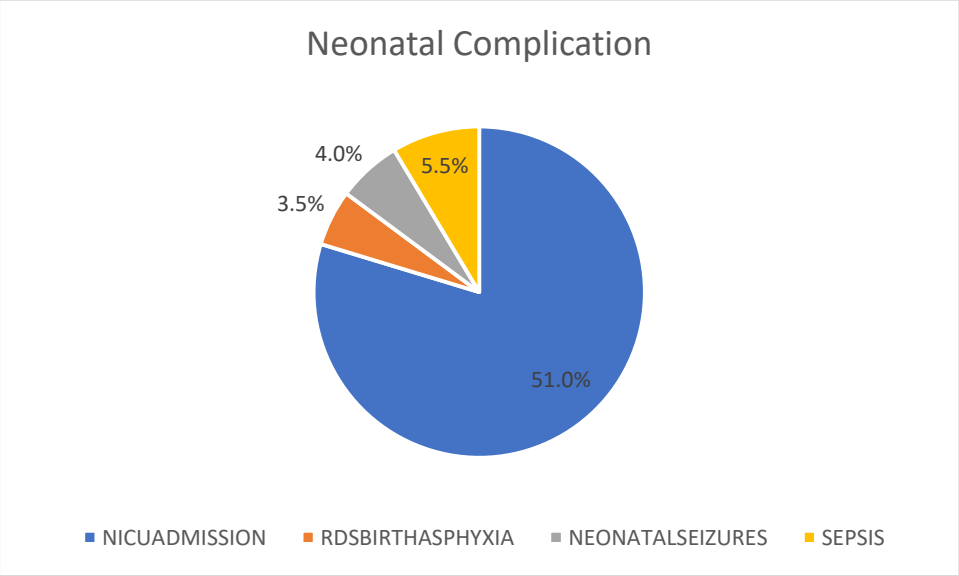



From the above graph it is evident that 5 minute apgar less than 8 was found in 89% of twin 1 and 91% of twin 2 and 100% of triplet 3.

TABLE 12: NEONATAL COMPLICATION

| NEONATAL COMPLICATION | N | %\ |
|---|---|---|
| NICUADMISSION | 102 | 51.0% |
| RDSBIRTHASPHYXIA | 7 | 3.5% |
| NEONATALSEIZURES | 8 | 4.0% |
| SEPSIS | 11 | 5.5% |

FIGURE 12: PIE CHART DEPICTING NEONATAL COMPLICATION




From the above graph it is evident that 51% of babies had NICU admissions the number were quite large when compared to singleton pregnancy and 5.5 % babies had birth asphyxia and 4% of the admitted babies developed neonatal seizures and 3.5% babies developed sepsis

# DISCUSSION

We conducted a study on 200 multiple pregnancy parturients admitted at IOG egmore. The aim of this study was to determine the maternal and neonatal complications associated with multiple pregnancies.

Among the 200 cases, based on age distribution, 43 % of the cases were between 26-30 years of age group, Although it was thought that prevalence of multiple births is more in the advanced age group due to the use of fertility treatment and rise in FSH concentration with age, A study by **Tomar SP et al** also revealed 81% of multiple births in the age group of 20-29 years. The high prevalence of multiple pregnancies at a young age may be due to early age at marriage and childbirth in the study population. The next maximum lies in the range of >36 years which correlates with the fact of the long period of infertility and use of assisted reproductive techniques.

Among 200 cases of multiple pregnancies, 121 were primi and 79 were multigravida. N Rezavand proved no difference in parity among cases of multiple pregnancies.

With regards to chorionicity, 63% were dichorionic and 29% were monochorionic and diamniotic and 4% were monochorionic and monoamniotic and 3.5% were trichorionic and triamniotic. Studies by Assuncao et al45 conducted in 289 twin pregnancies between 2003 to 2006 it was found that 60% were DCDA, 30.8% were MCDA and 6.6% were MCMA. Our study results were similar to this study.

Out of 200 cases, 154 conceived spontaneously and the remaining 46 come under the category of assisted reproductive technique use. And the number was quite larger when compared with singleton pregnancy

Out of 46 cases, 1- IUI and 27 – IVF conception, 18 were out of ovulation induction. With respect to triplets , 7 of them were TCTA 4 conceived after IVF

Discussing about maternal complications, the most common maternal complication observed in this study was preterm labour which was around 62%.(124/200).

Deepthi et al and Nandmer G et al reported preterm delivery in 60% and 67% of twin pregnancies respectively. The inherited risk of preterm delivery in multiple pregnancies is supported by higher incidence of preterm delivery in this study

The next most common complication observed in this study is postpartum hemorrhage which is 22.5%, a study conducted by chowdry et al showed similar values of 18% of PPH in a study conducted by them, still a vigilant monitoring and active management of third stage of labour is needed , and prevention and treatment of anemia in adolescent girls and young adults will pave way for preventionof APH and PPH.

The third most common complication encountered in this study is anemia which is about 20 %.This high prevalence of anaemia in this study may be due to more number of referred patients who had taken inadequate treatment in antenatal period.

Followed by hypertensive disorder of pregnancy , which is 12% . Deepti et al, Sheela SR et al and Chowdhury S et al reported hypertensive disorders of pregnancy in 11.66%, 14.5%, 22.6% of

multiple pregnancies respectively. Other complications of ghtn spectrum like Pregnancy complicated by pre eclampsia accounts to 6% and by ANTEPARTUM ECLAMPSIA accounts to 3.5%.

GDM as such totally accounts for 10% of which overt DM was 3% and GDM was 7%. Chowdhury S et al have reported GDM in 5.7% of patients. Buhling KJ et al reported GDM in 3.4% of patients and did not found any association with twin pregnancy.

Other minor complications includes cord accidents like cord prolapse and occult cord prolapse occurred in 2% of cases, with regards to APH abruptio placentae accounts to 2% and placenta previa accounts to 1.5%.

Twin specific complications like single fetal demise accounts to 4.5 % of cases and selective fetal growth restriction accounts to 5.5% of cases

Out of 200 cases 8.5% of cases presented with PPPROM and 2.0% presented with PROM.

Next analysing the mode of delivery among 200 cases of multiple pregnancy,63% of cases ie 127 cases delivered by LSCS The rate of cesarean section in study supported by Shetty MB et al, Chowdhury et al and Deepthi et al who have reported cesarean section in 68%, 49% and 45% of twin pregnancies respectively.

A study by Assuncao et al has reported cesarean section in 84.8% of patients that is explained by higher incidence of (42.8%) iatrogenic preterm delivery in that study.

The most common indication for LSCS being first twin non cephalic 37% followed by feal distress 26% and prev LSCS 14%. All the 7 TCTA triplets were delivered by caesarean mode of delivery keeping in mind the fact of prematurity and malpresentation and a component of growth restriction . And All MCMA twin were delivered by caesarean mode of delivery in order to avoid complication while allowing for normal vaginal delivery

Among birth weight of the twins, 52.5 % fall under 2.1 to 2.5 %. Cumulative percentage of 71% of twin 1 , 74% of twin 2 and all 100% of triplet 3 were of low birth weight , ie <2.5 kg posing a major threat

to neonatal morbidity and mortality. Low birth weight is mostly because of preterm delivery and iatrogenic preterm termination of MCMA and TCTA pregnancy.

# SUMMARY

200 multiple pregnancy cases were studied in our institute, out of which 98% of the cases were booked, 61% of them are primi gravida and 39% of them were multigravida.78% of them delivered at an gestational age less than 36 weeks, preterm delivery.71% of them conceived spontaneously and 29 % conceived after assisted reproductive techniques out of which , 19% were out of IVF conception.63% of them were DCDA ,29% were of MCDA ,4% were MCMA ,3.5% of them were TCTA.

Various maternal complications and its percentage associated with multiple pregnancy among them , preterm delivery is the most common complication about 62%, and the second one is postpartum hemorrhage 22.5%.

Followed by anemia 20%, other rare complications include single fetal demise,fetal growth restriction weighs about 4.5%

and 5.5% respectively,though it is rare it is specific for multiple pregnancy.

Cesarean mode of delivery is the most common mode of delivery when it comes to multiple pregnancy,which accounts for 63%.

Among indications for cesarean deliveries , non cephalic presentation of first twin pose the major threat to increase the primary cesarean section rate, followed by fetal distress , all the 6 TCTA triplets were delivered by cesarean mode of delivery keeping in mind the prematurity of the fetuses and most of them had growth restriction.

From our study it is evident that birth weight of twin babies mostly fall in the range of 2.1 to 2.5 this can be extrapolated to the preterm nature of the babies of multiple pregnancy.

With regards to APGAR , 5 minute apgar less than 8 was found in 89% of twin 1 and 91% of twin 2 and 100% of triplet 3.

Among neonatal complications, 5 minute apgar less than 8 was found in 89% of twin 1 and 91% of twin 2 and 100% of triplet 3. 51% of babies had NICU admissions the number were quite large when compared to singleton pregnancy and 5.5 % babies had birth asphyxia and 4% of the admitted babies developed neonatal seizures and 3.5% babies developed sepsis

# CONCLUSION

A dramatic increase in the numbers of multiple births in developed countries has stimulated interest in the progress and outcome of such pregnancies. As well as considerable health risks, the social, psychological and financial impact of multiple births, especially higher order multiple births, has been highlighted

This study revealed higher percentage preterm labour, anemia, gestational hypertension in twin pregnancy as compared to other studies. Anaemia, hypertensive disorder of pregnancy along with postpartum haemorrhage are the well-known causes of maternal mortality, thus frequent antenatal visits should be aimed to detects maternal complications earlier. Aggressive treatment of anemia with intravenous iron should be considered whenever there are adverse effects or noncompliance to oral iron. Atonic postpartum haemorrhage should be anticipated in all multiple pregnancies, therefore prophylactic treatment should be prearranged for its prevention.

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# PROFORMA

**"A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"**

**DATE:**

**NAME:**

**AGE:**

**IPNO:**

**ADDRESS:**

**PHONE NUMBER:**

**SOCIOECONOMIC STATUS:**

**OBSTETRIC CODE:**

**GESTATIONAL AGE:**

**MENSTRUAL HISTORY:**

| | |
|---|---|
| **REGULAR** | **IRREGULAR** |
| **LMP** | **EDD** |

**MARITAL HISTORY:**

| | |
|---|---|
| **MARRIED SINCE: in years** | **CONSANGUINITY:** |
| | **YES** / **NO** |

**OBSTETRIC HISTORY:**

**BOOKING STATUS:**

**ANTENATAL VISITS**

**MODE OF CONCEPTION:**

**-SPONTANEOUS**

**-INDUCED**

**HISTORY OF PRIMARY OR SECONDARY INFERTILITY:**

**HISTORY OF TREATMENT TAKEN FOR INFERTILITY:**

**-OVULATION INDUCTION:**

**-IUI:**

**-INVITRO FERTILISATION:**

**1ST TRIMESTER HISTORY:**

**-HYPEREMESIS**

**-BLEEDING PV**

**-H/O UTI**

**2ND TRIMESTER HISTORY:**

**-ABDOMINAL PAIN**

**-BLEEDING PV**

**-EXCESSIVE WEIGHT GAIN**

**-IMMINENT SIGNS(HEADACHE,BLURRING OF VISION,UPPER ABDOMINAL PAIN,REDUCED URINE OUTPUT)**

**-H/O RAISED OGCT**

**-LEAKING PV**

**3RD TRIMESTER HISTORY:**

**ABDOMINAL PAIN**

**-BLEEDING PV**

**-EXCESSIVE WEIGHT GAIN**

**-IMMINENT SIGNS(HEADACHE,BLURRING OF VISION,UPPER ABDOMINAL PAIN,REDUCED URINE OUTPUT)**

**-H/O RAISED OGCT**

**-LEAKING PV**

**PAST OBSTETRIC HISTORY:**

**PAST HISTORY:**

**H/O DM/HTN/PTB/BA/EPILEPSY/THYROID/DRUG ALLERGY/BLOOD TRANSFUSION.**

**FAMILY HISTORY OF MULTIPLE PREGNANCY:**

**H/O PREVIOUS SURGERIES.**

**EXAMINATIONS:**

**GENERAL EXAMINATION:**

**BUILT-**

**NOURISHMENT-**

**VITALS-**

**PR- BP-**

**PALLOR- PEDAL EDEMA- ICTERUS-**

**BREAST- THYROID- SPINE-**

**SYSTEMIC EXAMINATION:**

**CVS-**

**RS-**

**CNS-**

**OBSTETRICS EXAMINATION:**

**PER ABDOMEN-**

**PER SPECULUM-**

**PERVAGINAL EXAMINATION:**

**INVESTIGATION:**

**HB- TC- DC- RBC- PLT- PCV-**

**RBS- UREA- CREAT-**

**T.BILIRUBIN- D.BILIRUBIN-**

**SGOT- SGPT-**

**T.PROTEIN-**

**S.ALBUMIN-**

**FBS-**

**PPBS-**

**HBA1C**

**BLOOD GROUPING AND TYPING**

**VIRAL MARKERS:**

**HIV-**

**HBsAG-**

**VDRL-**

**OGCT-**

**TSH-**

**ECHO-**

**BLEEDING TIME-**

**CLOTTING TIME-**

**S.URIC ACID-**

**URINE SPOT PCR-**

**APTT-**

**PT-**

**INR-**

**S.FIBRINOGEN-**

**LDH-**

**URINE ANALYSIS-**

**URINE ROUTINE-**

**URINE C/S**

**ANTENATAL USG:**

**DELIVERY OUTCOME:**

**INDUCTION OF LABOUR-**

**DETAILS-**

**MODE OF DELIVERY**

**LABOUR NATURAL-**

**INSTRUMENTAL-**

**LSCS-**

**PRIMARY LSCS-**

**INDICATION FOR LSCS**

**BABY DETAILS:**

**TWIN A**

**BIRTH WEIGHT**

**APGAR 1MIN 5 MIN 7MIN**

**D.NO-**

**TWIN B**

**BIRTH WEIGHT**

**APGAR 1MIN 5 MIN 7MIN**

**D.NO-**

**TWIN C**

**BIRTH WEIGHT**

**APGAR 1MIN 5 MIN 7MIN**

**D.NO-**

**OUTCOME**

**ANTEPARTUM**

**ANEMIA**

**HYPEREMESIS**

**GESTATIONAL DIABETES MELLITUS**

**GESTATIONAL HYPERTENSION AND RELATED**

**GESTATIONAL THROMBOCYTOPENIA**

**ANTEPARTUM HEMORRHAGE**

**PPROM**

**PROM**

**PRETERM LABOUR-**

**<u>INTRAPARTUM COMPLICATION:</u>**

**PROM-**

**CORD PROLAPSE-**

**ABRUPTION-**

**INTERLOCKING-**

**PPH-**

**RETAINED PLACENTA-**

**CONVERSION OF SECOND TWIN TO TRANSVERSE LIE-**

**TIME TAKEN FOR DELIVERY OF SECOND TWIN:**

**<u>POSTPARTUM COMPLICATION:</u>**

**PPH-**

**NEED FOR BLOOD TRANSFUSION-**

**SUBINVOLUTION**

**LACTIONAL FAILURE**

**SEPSIS**

**FEVER**

**POSTPARTUM ECLAMPSIA**

**PERIPARTUM HYSTERECTOMY-**

**MATERNAL MORTALITY-**

**<u>NEONATAL COMPLICATION:</u>**

**PREMATURITY**

**LOW BIRTH WEIGHT**

**PERINATAL ASPHYSIA**

**HIE**

**PATIENT CONSENT FORM**

Patient may check ( ) these boxes:

( ) I confirm that I Have understood the purpose of procedure for the above study. I have the opportunity to ask questions and all my questions and doubts have been answered to my complete satisfaction.

( ) I Understand that my participation in the study is voluntary and that i am free to withdraw at anytime without giving reason , without my legal rights being affected.

( ) I Understand that sponsor of the clinical study , others working on the sponsor's behalf ,the ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and my further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access.

( ) However , I understand that my identify will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

**TITLE: "A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"**

**Study Centre: Institute of Obstetrics and Gynaecology, Egmore.**

**Patient name: Age: IP No:**

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately in form the form study staff if I suffer from any

deterioration in my health or well being or any unexpected or unusual symptoms.

I hereby consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic tests including haematological , biochemical, radiological tests and to undergo treatment.

( Signature/left thumb impression)

Name of the participant:

Complete postal address:

This is to certify that the above consent has been obtained in my presence;

Signature of the principal investigator:

Date:

Place:

# INFORMATION SHEET

**TITLE: "A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"**

| | | |
|---|---|---|
| Name of investigator | : | Dr. Vidhya Lakshmi |
| Name of participant | : | |
| Purpose of research | : | To find out the outcome of maternal and neonate in multiple pregnancy |
| Study design | : | Analytical Study |
| Study population | : | Multiple pregnancy mothers more than 28 weeks and delivered in IOG. |
| Possible risks | : | No risk to the patient |

Confidentiality of information obtained from you:

Privacy of the patients in the research will be maintained throughout the study.in the event of any publication or any presentation resulting from the research no personally identifiable information will be shared.

Can you decide to stop participating in the study?

Taking part in the study is voluntary. You are free to decide whether to participate in the study or to withdraw from the study at any time

How will your decision in not participating in the study affect you?

Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date:

Place:

**அனுமதியுடனான ஒப்புதல் படிவம்:**

-இந்த ஆய்விற்கான செயல்முறையின் நோக்கத்தை நான் புரிந்துள்ளேன் என்பதை உறுதிப்படுத்துகிறேன். எனக்கு கேள்விகளை கேட்க வாய்ப்பு உள்ளது. என்னுடைய எல்லாகேள்விகளும் சந்தேகங்களும் என் முழுதிருப்திக்கு பதில் அளித்துள்ளன.

-ஆய்வில் எனது பங்கேற்பு தன்னார்வமாக இருப்பதையும், என் சட்டஉரிமைகள் பாதிக்கப்படாமல், காரணத்தைத் தெரிவிக்காமல் எப்போது வேண்டுமானாலும் விலக்கிக்கொள்ளலாம் என்பதையும் நான் புரிந்துகொள்கிறேன்.

-ஆய்வில் இருந்து நான் விலகி வந்தாலும் கூட, ஆராய்ச்சிக்கு பொருந்தக்கூடிய என் உடல்நல ஆவணங்களைப் பார்க்க என் நெறிமுறைக்குழு மற்றும் ஒழுங்குமுறை அதிகாரிகளுக்கு எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்து கொள்கிறேன். இந்தஅணுகலை நான் ஏற்கிறேன்.

-இருப்பினும், சட்டத்தின்கீழ் தேவைப்பட்டாலன்றி, மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிட்ட எந்த தகவலிலும் என் அடையாளத்தை வெளிப்படுத்தமுடியாது என்பதை நான் புரிந்து கொள்கிறேன். இந்த ஆய்விலிருந்து எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டைக் கட்டுப்படுத்துவதை நான் ஏற்றுக் கொள்கிறேன்.

-மேலே உள்ள படிப்பில்கலந்துகொள்ளவும், ஆய்வின் போதுகொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும், ஆய்வுக் குழுவோடு ஒத்துழைக்கவும், என் உடல்நலம் அல்லது நலம் அல்லது எந்தவொரு எதிர்பாராத அல்லது அசாதாரண அறிகுறிகளிலும் நான் பாதிக்கப்படுகையில் உடனடியாக ஆய்வு ஊழியர்களுக்கு தெரிவிக்கவும், இந்த ஆய்வில் பங்கேற்க ஒப்புக் கொள்கிறேன்.
நான் இதனுடன் முழுமையான மருத்துவ பரிசோதனை மற்றும் நோயறிதல் சோதனைகள் இரத்தம், உயிர்வேதியியல், கதிரியக்க சோதனைகள் உட்பட சிகிச்சைக்கு உட்படுத்த அனுமதிக்கிறேன்.

ஆய்வுதலைப்பு:
ஆய்வுமையம்: எம்.எம்.சி, சென்னை
பங்கேற்பாளரின்பெயர்:
பங்கேற்பாளரின்வயது:
நோயாளிஎண்:

நோயாளியின்கையொப்பம்
நோயாளியின்பெயர்மற்றும்முகவரி:
ஆராய்ச்சியாளரின்கையொப்பம்:

**<u>நோயாளியின் ஒப்புதல் படிவம்</u>**

**<u>ஆய்வு தலைப்பு :</u>** ஒன்றுக்கும் மேற்பட்ட கற்பதில் தாய்க்கு ஏற்பட கூடிய சிக்கல்கள் மற்றும் சிசுவிற்கு ஏற்பட கூடிய சிக்கல்கள் - ஒரு ஆய்வு

| | | |
|---|---|---|
| முக்கிய ஆய்வாளரின் பெயர் | : | டாக்டர். வித்யாலட்சுமி. க |
| நிறுவன முகவரி | : | அரசு மகளிர் மகப்பேறு மருத்துவமனை, எழும்பூர், சென்னை – 600 008 |

நீங்கள் இந்த ஆய்வில் பங்கு பெற வரவேற்கப்படுகிறீர்கள், இந்த தாளில் அளிக்கப்பட்டுள்ள விவரங்கள் நீங்கள் ஆய்வில் பங்கு பெறுவது குறித்து தீர்மானிக்க உதவும். சந்தேகங்கள் மற்றும் கேள்விகள் தயக்கமின்றி வரவேற்கப்படுகின்றன.

நாங்கள் இந்த ஆய்விற்காக தலைமை நெறிமுறை குழுவின் (Institutional Ethics Committee) அனுமதி பெற்றுள்ளோம்.

**<u>பகுதி - I</u>**

**<u>நோயாளியின் தகவல் படிவம்</u>**:

**உங்கள் தகவல் குறித்த நம்பிக்கை**

உங்களை பற்றிய தகவல் (பரிசோதனைவிவரங்கள்) எவருக்கும் தெரிவிக்கப்படமாட்டாது. இந்த ஆய்விலிருந்து அறியப்படும் விவரங்கள்

கூட்டங்களில், பத்திரிக்கைகளில் இடப்படும் போது உங்களைப் பற்றிய தனிப்பட்ட தகவல்கள் இரகசியம் காக்கப்படும்.

நீங்கள் இந்த ஆய்வில் பங்கு கொள்ளாவிட்டாலும் உங்களுடைய மருத்துவ சிகிச்சையோ அல்லது ஆய்வாளருடன், மருத்துவமனையுடன் உங்களது உறவு பாதிக்கப்படாது. இதனால் உங்களுக்கு கிடைக்கப்பெற இருக்கும் எந்த ஒரு சிகிச்சை முறையிலும் மாறுதல் ஏற்படாது. நீங்கள் இந்த ஆய்வில் பங்கு பெறுவது உங்களுடைய விருப்பம். எந்த நேரத்திலும், எந்த விளக்கமும் அளிக்காமல் நீங்கள் விலகிக் கொள்ள உரிமை உண்டு.

ஆய்வாளரின் கையொப்பம்

பங்கேற்பவரின் பெயர் :

பங்கேற்பவரின் கையொப்பம்

நாள்

இடம்:

**<u>சுய ஆய்வு ஒப்புதல் படிவம்</u>**

**ஆய்வு செய்யப்படும் தலைப்பு :** **ஒன்றுக்கும் மேற்பட்ட கற்பதில் தாய்க்கு ஏற்பட கூடிய சிக்கல்கள் மற்றும் சிசுவிற்கு ஏற்பட கூடிய சிக்கல்கள் - ஒரு ஆய்வு**

**ஆய்விடம் :** **மகப்பேறு மகளிர் நோயியல் மற்றும் அரசு தாய்சேய் நல மருத்துவமனை, எழும்பூர், சென்னை.**

**பங்கு பெறுபவரின் பெயர் :**

**பங்கு பெறுபவரின் எண்:**

**பங்கு பெறுபவரின் வயது:**

**மருத்துவமனை எண்:**

1. எனக்கு தரப்பட்ட ஆராய்ச்சியில் பங்கு பெறுவோர்க்கான தகவல் படிவத்தை முழுமையாக படித்து புரிந்து கொண்டேன்.
2. ஆராய்ச்சியின் தன்மை முழுமையாகவும் விரிவாகவும் எடுத்து உரைக்கப்பட்டது.
3. எனது எல்லா கேள்விகளுக்கும் விடையளிக்கப்பட்டது.
4. ஆய்வாளர் என் உரிமைகளையும், பொறுப்புகளையும் நன்கு விளக்கினார்.
5. நான் ஆய்வாளருக்கு முழு ஒத்துழைப்பு கொடுக்கவும், பரிசோதனை செய்து கொள்ளவும் அனுமதிக்கிறேன்.
6. எனக்கு இரத்த பரிசோதனை, ஸ்கேன் மற்றும் ஆய்விற்கு தேவையான அனைத்து பரிசோதனைகளும் செய்து கொள்ள சம்மதம்.

7. எனக்கு இந்த ஆய்வின் போது அறுவை சிகிச்சை மேற்கொள்ளும் போது தேவைப்பட்டால் என் வயிற்று பகுதியின் பாதிப்புகளை புகைப்படம் எடுக்கவும், அதனை மருத்துவரின் தேவைக்கேற்பு உபயோகிக்கவும் அனுமதிக்கிறேன்.
8. நான் இந்த ஆராய்ச்சியில் பங்கேற்பதால் ஏற்படும் சாதகபாதகங்களை ஆய்வாளர் விளக்கிக் கூற அறிந்து கொண்டேன்.
9. எப்பொழுது வேண்டுமானாலும் நான் இந்த ஆய்வில் இருந்து விலகி கொள்ளலாம் என்பதை அறிவேன். அவ்வாறு விலகிக் கொள்வதால் எனக்கு கொடுக்கப்படும் சிகிச்சையில் எந்த மாற்றமும் இருக்காது என அறிந்து கொண்டேன்.
10. இந்த ஆய்வுக்காக பெறப்படும் தகவல்களை ஆய்விதழ்களிலோ, கருத்தரங்கிலோ வெளியிட எனக்கு எந்தவித மறுப்போ, ஆட்சேபணையோ இல்லை.
11. எனது அடையாளங்கள் மற்றும் தனிப்பட்ட விவரங்கள் ஆய்விதழ்களிலோ, கருத்தரங்கிலோ வெளியிடப்படமாட்டாது என்று எனக்கு உறுதியளிக்கப்பட்டது.
12. எனக்கு இந்த ஆராய்ச்சி குறித்த சந்தேகம் இருந்தால் உடனே ஆய்வாளரை கேட்டு தெளிவுபடுத்தி கொள்ளலாம் என உறுதியளிக்கப்பட்டது.
13. இந்த ஒப்புதல் படிவத்தில் கையொப்பமிடுவதின் மூலம் இந்த படிவத்தில் உள்ளவையாவும் எனக்கு தெளிவாக எடுத்துரைக்கப்பட்டது. அதை நான் நன்கு புரிந்து கொண்டேன் என தெரிவித்துக் கொள்கிறேன்.

நோயாளியின் பெயர்

கையொப்பம் / பெருவிரல்சுவடு தேதி

ஆராய்ச்சியாளர் பெயர்

கையொப்பம் / பெருவிரல்சுவடு தேதி

சாட்சி 1

பெயர் கையொப்பம் / பெருவிரல்சுவடு தேதி

சாட்சி 2

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## PLAGIARSM CERTIFICATE

This is to certify that this dissertation work titled "**A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"** of the candidate **DR K. VIDHYA LAKSHMI REG. NO. 221916905 ,** for the award of M.S in the branch of **OBSTETRICS AND GYNAECOLOGY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows EIGHTEEN percentage of plagiarism in the dissertation (D126486391)

Signature and Seal of the Guide

**Prof. Dr. S.VIJAYA, M.D.,D.G.O**
**Director and Professor ,**
Institute of Obstetrics &Gynaecology,
Madras Medical College, Chennai.

Curiginal

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## Sources included in the report

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| SA | **FMO IN MULTIPLE PREGNANCY article.docx**<br>Document FMO IN MULTIPLE PREGNANCY article.docx (D110296489) | 1 |
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# INSTITUTIONAL ETHICS COMMITTEE
# MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013/RR-16
Telephone No.044 25305301
Fax: 011 25363970

## CERTIFICATE OF APPROVAL

To
**Dr.VIDHYA LAKSHMI K,**
MS Post Graduate (2019-2022),
Department of Obstetrics and Gynaecology,
Madras Medical College,
Chennai-600003.

Dear Dr. VIDHYA LAKSHMI K,

The Institutional Ethics Committee has considered your request and approved your study titled **"A PROSPECTIVE STUDY ON MATERNAL AND PERINATAL OUTCOME IN MULTIPLE PREGNANCY"- NO.22022021.** The following members of Ethics Committee were present in the meeting held on **17.02.2021** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst. of Nephrology, MMC, Ch : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai : Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

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

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

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

MASTER CHART

| | | | | | | | | | | | | ANTEPARTUM | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SNO | AGE | BOOKING STATUS | OBS CODE | GA AT DELIVERY | SES | MODE OF CONCEPTION | IF ART | | | NO OF ANTENATAL VISITS | CHORIONICITY | 1ST TRIMESTER BLEEDIN PV | GDM/ OVERTDM | GHTN | | | PPROM /PROM | LOW HB | FGR | SFD | AF |
| | | | | | | | OI | IUI | IVF | | | | | GHTN | PRE ECLAMPSIA | ECLAMPSIA | | | | | PLACENTA PREVIA |
| 1 | 40 | BOOKED | G2A1 | 32 | 3 | S | | | YES | 9 | DCDA | NO | GDM | | PRE ECLAMPSIA | | | YES | | | |
| 2 | 27 | BOOKED | PRIMI | 33 | 4 | S | | | | 6 | MCDA | NO | | | | | PPROM | | | | |
| 3 | 27 | BOOKED | G2P1L1 | 34 | 3 | S | | | | 8 | DCDA | NO | | GHTN | | | | | | | |
| 4 | 23 | BOOKED | PRIMI | 32 | 4 | S | | | | 8 | MCDA | NO | | | | | | | YES | | |
| 5 | 24 | BOOKED | PRIMI | 35 | 4 | S | | | | 6 | DCDA | NO | | | | | | YES | | | |
| 6 | 25 | BOOKED | G2P1L1 | 37 | 5 | S | | | | 7 | DCDA | NO | | | | AP ECLAMPSIA | | | | | |
| 7 | 25 | BOOKED | G2P1L1 | 39 | 3 | S | | | | 8 | DCDA | NO | | | PRE ECLAMPSIA | | | | | | |
| 8 | 32 | BOOKED | PRIMI | 36 | 3 | S | | | | 8 | DCDA | NO | | | | | | | | | |
| 9 | 44 | BOOKED | PRIMI | 33 | 4 | S | | | | 8 | MCMA | NO | | | | | | | | | |
| 10 | 24 | BOOKED | G2A1 | 28 | 2 | S | | | | 6 | MCDA | YES | | | | | PPROM | | | | |
| 11 | 24 | BOOKED | PRIMI | 37 | 3 | S | | | | 6 | DCDA | NO | | | PRE ECLAMSIA | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12 | 27 | BOOKED | PRIMI | 31 | 3 | S | | 6 | MCDA | NO | | | YES | | | | | | | |
| 13 | 26 | BOOKED | PRIMI | 37 | 4 | S | | 7 | DCDA | NO | | GHTN | | YES | | | | | | |
| 14 | 26 | BOOKED | PRIMI | 31 | 3 | S | | 7 | MCDA | NO | | | | | | | | | | |
| 15 | 27 | BOOKED | G2P1L1 | 37 | 4 | S | | 6 | DCDA | NO | | | YES | | | | | | | |
| 16 | 26 | BOOKED | PRIMI | 29 | 3 | S | | 7 | MCDA | NO | | | | | | | | | | |
| 17 | 29 | BOOKED | PRIMI | 28 | 4 | S | | 6 | MCDA | NO | | | | | | | | | | |
| 18 | 37 | BOOKED | G2P1L1 | 28 | 4 | S | | 6 | DCDA | NO | | GHTN | | PPROM | | YES | | | | |
| 19 | 38 | BOOKED | PRIMI | 35 | 4 | S | YES | 9 | MCDA | NO | GDM | | | | | | | | | |
| 20 | 30 | BOOKED | G3P2L2 | 36 | 3 | S | | 7 | DCDA | NO | | | YES | | | | | | | |
| 21 | 27 | BOOKED | PRIMI | 32 | 4 | S | | 7 | MCDA | NO | | GHTN | PRE ECLAMPSIA | | PPROM | | | | | |
| 22 | 23 | BOOKED | G2P1L1 | 37 | 5 | S | | 6 | MCDA | YES | | | | YES | | | | | | |
| 23 | 24 | BOOKED | G2P1L1 | 33 | 4 | S | | 6 | DCDA | NO | | | | | PPROM | | | | | |
| 24 | 30 | BOOKED | G2P1L1 | 35 | 3 | S | | 5 | DCDA | NO | GDM | GHTN | | | | | | | | |
| 25 | 24 | BOOKED | PRIMI | 37 | 3 | S | | | DCDA | | | | | | | YES | | | | |
| 26 | 25 | BOOKED | PRIMI | 36 | 3 | S | | | MCDA | | | | | | | | YES | | | |
| 27 | 24 | BOOKED | PRIMI | 36 | 3 | S | | | DCDA | | | GHTN | | | | YES | | | | |
| 28 | 28 | BOOKED | PRIMI | 38 | 4 | S | | | DCDA | | | | | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 29 | 29 | BOOKED | PRIMI | 29 | 2 | S | | | | | DCDA | | OVERT | GHTN | | | | | | | |
| 30 | 32 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | GHTN | | | | | | YES | |
| 31 | 36 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 32 | 25 | BOOKED | G2P1L1 | 32 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 33 | 29 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 34 | 25 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 35 | 30 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | GDM | | | | | | | | |
| 36 | 21 | BOOKED | PRIMI | 35 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 37 | 25 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | GHTN | | | PROM | | | | |
| 38 | 27 | BOOKED | G2P1L1 | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 39 | 34 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 40 | 33 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 41 | 26 | BOOKED | G2A1 | 36 | 3 | S | | | | | DCDA | | | | | | | | YES | | |
| 42 | 25 | BOOKED | PRIMI | 33 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 43 | 23 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 44 | 29 | BOOKED | PRIMI | 31 | 3 | S | | | | | MCDA | | | | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 45 | 21 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | GDM | | | | | | | | |
| 46 | 23 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 47 | 25 | BOOKED | G2P1L1 | 36 | 3 | S | | | | | MCDA | | | | | AP ECLAMPSIA | PPROM | | | | |
| 48 | 27 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 49 | 29 | BOOKED | G3A2 | 38 | 3 | S | | | | | DCDA | | | GHTN | | | | | | | |
| 50 | 30 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 51 | 33 | BOOKED | PRIMI | 35 | 3 | OI | | | | | DCDA | | | | | | | | | | |
| 52 | 35 | BOOKED | PRIMI | 33 | 3 | IVF | | | | | DCDA | | | | | | | | | YES | |
| 53 | 36 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | DCDA | | GDM | | PRE ECLAMSIA | | | | YES | | |
| 54 | 22 | BOOKED | PRIMI | 34 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 55 | 23 | BOOKED | PRIMI | 34 | 2 | S | | | | | MCDA | | | | | | PPROM | YES | | | |
| 56 | 26 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 57 | 25 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 58 | 23 | UNBOOKED | G2P1L1 | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 59 | 28 | BOOKED | PRIMI | 35 | 2 | S | | | | | MCDA | | | GHTN | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 60 | 29 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 61 | 23 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 62 | 30 | BOOKED | G2A1 | 37 | 2 | S | | | | | DCDA | | | | | | | | | | |
| 63 | 25 | BOOKED | PRIMI | 32 | 3 | S | | | | | MCDA | | | | | | | YES | | | |
| 64 | 27 | BOOKED | PRIMI | 36 | 2 | S | | | | | MCDA | | | | | | | | | | |
| 65 | 24 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 66 | 26 | BOOKED | G2P1L1 | 36 | 3 | S | | | | | MCDA | | | | | | PPROM | | | | |
| 67 | 28 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | GHTN | | | | | | | |
| 68 | 27 | BOOKED | PRIMI | 36 | 3 | OI | | | | | DCDA | | | | | | | | | | |
| 69 | 29 | BOOKED | G3P1L1 A1 | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 70 | 33 | BOOKED | PRIMI | 34 | 2 | S | | | | | DCDA | | | | | | | YES | | | |
| 71 | 22 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | YES | | yes |
| 72 | 21 | BOOKED | G2A1 | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 73 | 27 | BOOKED | PRIMI | 32 | 3 | IUI | | | | | TCTA | | | | | | | | | YES | |
| 74 | 33 | BOOKED | G4PILIA 2 | 37 | 4 | S | | | | | DCDA | | OVERT DM | | | | PROM | | | | |
| 75 | 28 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 76 | 29 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCMA | | | | | | | | | | |

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| 77 | 20 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 78 | 25 | BOOKED | G2A1 | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 79 | 27 | BOOKED | PRIMI | 37 | 2 | S | | | | | DCDA | | | | | | | YES | | | |
| 80 | 25 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | GHTN | | | | | | | |
| 81 | 29 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 82 | 27 | BOOKED | G3P1L1 A1 | 36 | 3 | S | | | | | DCDA | | | GHTN | | | PPROM | | | | |
| 83 | 21 | BOOKED | PRIMI | 29 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 84 | 22 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 85 | 28 | BOOKED | G3P2L2 | 39 | 4 | S | | | | | DCDA | | | | | | | YES | | | |
| 86 | 29 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 87 | 26 | BOOKED | G2P1L1 | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 88 | 25 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 89 | 28 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 90 | 20 | BOOKED | PRIMI | 34 | 2 | IVF | | | | | TCTA | | | | | | | | YES | | |
| 91 | 27 | BOOKED | PRIMI | 35 | 3 | S | | | | | MCDA | | | GHTN | | | PPROM | | | | |
| 92 | 27 | UNBOOKED | G2P1L1 | 39 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 93 | 29 | BOOKED | PRIMI | 35 | 3 | OI | | | | | DCDA | | | | | | | | | | |

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| 94 | 27 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | AP ECLAMPSIA | | | | | |
| 95 | 26 | BOOKED | PRIMI | 34 | 2 | IVF | | | | | TCTA | | | | | | | YES | | | |
| 96 | 20 | BOOKED | PRIMI | 35 | 3 | S | | | | | MCDA | | | | | | | | | YES | |
| 97 | 24 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCDA | | | GHTN | | | | | | | |
| 98 | 27 | BOOKED | G2A1 | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 99 | 20 | BOOKED | PRIMI | 32 | 4 | S | | | | | MCDA | | | | | | | | | | |
| 100 | 30 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 101 | 38 | BOOKED | G4A3 | 32 | 3 | IVF | | | | | DCDA | | | | | | | | | | |
| 102 | 37 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 103 | 32 | BOOKED | PRIMI | 39 | 2 | S | | | | | DCDA | | | | | | | YES | | | |
| 104 | 37 | BOOKED | PRIMI | 34 | 3 | S | | | | | MCDA | | GDM | | PRE ECLAMPSIA | | | | | | |
| 105 | 45 | BOOKED | G2P1L1 | 35 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 106 | 28 | BOOKED | PRIMI | 36 | 4 | S | | | | | MCDA | | | | | | | YES | | | |
| 107 | 38 | BOOKED | PRIMI | 32 | 3 | IVF | | | | | DCDA | | | | | | | | | | |
| 108 | 31 | BOOKED | G2P1LO | 32 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 109 | 33 | BOOKED | PRIMI | 33 | 3 | S | | | | | TCTA | | | | | | | | YES | | |
| 110 | 28 | BOOKED | PRIMI | 38 | 2 | S | | | | | DCDA | | | | | | | | | | |
| 111 | 29 | BOOKED | G2P1L1 | 29 | 3 | S | | | | | MCMA | | | | | | | | | | |

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| 112 | 33 | BOOKED | PRIMI | 36 | 3 | IVF | | | | | DCDA | | | | | | | | | | |
| 113 | 29 | BOOKED | PRIMI | 37 | 4 | S | | | | | MCDA | | | GHTN | | | | | | | |
| 114 | 42 | BOOKED | G3P1L1 A1 | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 115 | 44 | BOOKED | PRIMI | 36 | 3 | IVF | | | | | DCDA | | | | | | | | | | |
| 116 | 29 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | PPROM | YES | | | |
| 117 | 33 | BOOKED | G2P1L1 | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 118 | 32 | BOOKED | PRIMI | 36 | 2 | S | | | | | DCDA | | | | | | | | | | |
| 119 | 36 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCDA | | | | | | | YES | | | yes |
| 120 | 45 | BOOKED | G2P1L1 | 33 | 3 | IVF | | | | | DCDA | | | | | | | | | | |
| 121 | 22 | BOOKED | G2P1L1 | 33 | 3 | S | | | | | DCDA | | | GHTN | | | | | | | |
| 122 | 28 | BOOKED | PRIMI | 38 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 123 | 20 | BOOKED | PRIMI | 37 | 2 | S | | | | | DCDA | | | | | | | YES | | | |
| 124 | 36 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 125 | 37 | BOOKED | PRIMI | 29 | 3 | S | | | | | MCMA | | | | PRE ECLAMPSIA | | | | | | |
| 126 | 25 | BOOKED | PRIMI | 33 | 3 | IVF | | | | | TCTA | | | | | | | | | | |

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| 127 | 26 | BOOKED | PRIMI | 38 | 2 | S | | | | | DCDA | | | | | | | | | | |
| 128 | 27 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | AP ECLAMPSIA | | | | | |
| 129 | 29 | BOOKED | PRIMI | 33 | 2 | S | | | | | MCDA | | | GHTN | | | | | | | |
| 130 | 26 | BOOKED | PRIMI | 32 | 3 | IVF | | | | | TCTA | | | | | | PPROM | YES | | | |
| 131 | 34 | BOOKED | G2A1 | 33 | 3 | S | | | | | MCDA | | | | | | | YES | YES | | |
| 132 | 27 | BOOKED | PRIMI | 38 | 2 | S | | | | | DCDA | | | | | | | | | | |
| 133 | 29 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | GDM | | | | | | | | |
| 134 | 21 | BOOKED | PRIMI | 35 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 135 | 25 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 136 | 27 | BOOKED | PRIMI | 33 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 137 | 29 | BOOKED | G2P1L1 | 38 | 4 | OI | | | | | DCDA | | | | | | PROM | | | | |
| 138 | 26 | BOOKED | PRIMI | 37 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 139 | 24 | BOOKED | PRIMI | 38 | 4 | S | | | | | DCDA | | | | | | | | | | |
| 140 | 26 | BOOKED | PRIMI | 39 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 141 | 28 | BOOKED | PRIMI | 32 | 4 | S | | | | | MCMA | | | | | | | | | | |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 142 | 29 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | AP ECLAMPSIA | | | | YES | |
| 143 | 26 | BOOKED | PRIMI | 38 | 4 | S | | | | | DCDA | | | | | | PROM | | | | |
| 144 | 20 | BOOKED | PRIMI | 30 | 3 | S | | | | | MCDA | | | GHTN | | | | YES | | | |
| 145 | 26 | BOOKED | PRIMI | 35 | 3 | S | | | | | MCDA | | | | | | PPROM | | | | yes |
| 146 | 22 | UNBOOKED | PRIMI | 29 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 147 | 27 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | GDM | | | | | | | | |
| 148 | 26 | BOOKED | G2P1L1 | 30 | 2 | S | | | | | MCDA | | | | | AP ECLAMPSIA | | | | | |
| 149 | 29 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCMA | | | | | | | | | | |
| 150 | 38 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 151 | 39 | BOOKED | PRIMI | 33 | 4 | S | | | | | MCDA | | | | | | | | | | |
| 152 | 42 | BOOKED | PRIMI | 32 | 3 | IVF | | | | | DCDA | | OVERT DM | | PRE ECLAMPSIA | | | | YES | | |
| 153 | 37 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 154 | 33 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 155 | 32 | BOOKED | PRIMI | 38 | 2 | S | | | | | DCDA | | | GHTN | | | | | | | |
| 156 | 36 | BOOKED | G2A1 | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 157 | 27 | BOOKED | PRIMI | 32 | 4 | S | | | | | MCDA | | | | | | | | | | |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 158 | 21 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 159 | 29 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 160 | 33 | BOOKED | PRIMI | 34 | 3 | S | | | | | MCDA | | | | | | PPROM | | | | |
| 161 | 37 | BOOKED | PRIMI | 33 | 2 | S | | | | | MCDA | | OVERT DM | GHTN | | | | YES | | | |
| 162 | 26 | BOOKED | PRIMI | 33 | 3 | OI | | | | | TCTA | | | | | | | | YES | | |
| 163 | 37 | BOOKED | G3A2 | 39 | 4 | S | | | | | DCDA | | | | | | | | | | |
| 164 | 24 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 165 | 28 | BOOKED | G2A1 | 32 | 3 | S | | | | | DCDA | | | | | | PPROM | | | | |
| 166 | 26 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 167 | 22 | BOOKED | PRIMI | 37 | 2 | S | | | | | DCDA | | GDM | | | | | | | | |
| 168 | 26 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 169 | 28 | BOOKED | PRIMI | 38 | 4 | S | | | | | DCDA | | | | | | | | | | |
| 170 | 26 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | GDM | | | | PPROM | | | | |
| 171 | 23 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCMA | | | | | | | YES | | | |
| 172 | 25 | UNBOOKED | PRIMI | 37 | 3 | OI | | | | | DCDA | | | | PRE ECLAMPSIA | | | | | YES | |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 173 | 27 | BOOKED | PRIMI | 34 | 4 | S | | | | | MCMA | | | | | | | | | | |
| 174 | 28 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | MCDA | | | | | | | YES | | | |
| 175 | 22 | BOOKED | G2P1L1 | 38 | 2 | S | | | | | DCDA | | GDM | | | | | | | | |
| 176 | 20 | BOOKED | PRIMI | 37 | 2 | S | | | | | MCDA | | | | | | | | | | |
| 177 | 42 | BOOKED | G2A1 | 36 | 4 | IVF | | | | | DCDA | | OVERT DM | | ORE ECLAMPSIA | | | | | | |
| 178 | 27 | BOOKED | PRIMI | 33 | 2 | S | | | | | DCDA | | | | | | | | | YES | |
| 179 | 36 | BOOKED | PRIMI | 38 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 180 | 41 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 181 | 38 | BOOKED | PRIMI | 36 | 2 | OI | | | | | DCDA | | OVERT DM | | | | | | | | |
| 182 | 29 | BOOKED | G3P1L1A1 | 37 | 3 | S | | | | | MCDA | | | | | | | | | | |
| 183 | 21 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 184 | 27 | BOOKED | PRIMI | 36 | 3 | S | | | | | DCDA | | GDM | | PRE ECLAMPSIA | | | | | | |
| 185 | 33 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 186 | 40 | BOOKED | G2P1L1 | 35 | 4 | IVF | | | | | DCDA | | | | | | | | | | |
| 187 | 29 | BOOKED | PRIMI | 36 | 3 | S | | | | | MCDA | | | | | | PPROM | YES | | | |

| | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 188 | 37 | BOOKED | PRIMI | 36 | 2 | S | | | | | MCDA | | | | | | | | YES | | |
| 189 | 27 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 190 | 21 | BOOKED | PRIMI | 39 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 191 | 26 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | PRE ECLAMPSIA | | | | | | |
| 192 | 37 | BOOKED | G2A1 | 36 | 4 | S | | | | | DCDA | | | | | | | YES | | | |
| 193 | 22 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 194 | 36 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | YES | |
| 195 | 38 | BOOKED | PRIMI | 33 | 3 | S | | | | | MCDA | | GDM | | | | | | | | |
| 196 | 21 | BOOKED | PRIMI | 32 | 2 | S | | | | | MCDA | | | GHTN | | | | | | | |
| 197 | 24 | BOOKED | PRIMI | 37 | 3 | S | | | | | DCDA | | | | | | | | | | |
| 198 | 27 | BOOKED | G2P1L1 | 37 | 4 | S | | | | | DCDA | | | | | | | | | | |
| 199 | 25 | BOOKED | PRIMI | 35 | 3 | S | | | | | DCDA | | | | | | | YES | | | |
| 200 | 23 | BOOKED | G2P1L1 | 38 | 3 | S | | | | | DCDA | | | GHTN | | | | YES | | | |

| | | INTRAPARTUM | | | | POSTPARTUM | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PH | PRETERM LABOUR | CORD ACCIDENTS | PPH | RETAINED PLACENTA | TIME TAKEN FOR DELIVERY OF SECOND TWIN | SEPSIS | CESAREAN HYSTERECTOMY | MORTALITY | PRESENTATION | MODE OF DELIVERY | | INDICATION IF LSCS | TWIN A/TRIPLET A | | | | TWIN B/ TRIPLET B | | | |
| ABRUPTION | | | | | | | | | CEPHALIC, CEPHALIC | | CESAREAN | | SEX | B.WT | APGAR | TIME | SEX | B,WT | APGAR | TIME |
| | YES | | | YES | 1MIN | | | | B,VX | | LSCS | DCDA ,1ST TWIN NON CEPHALIC | BOY | 2.2 | 8,9 | 11;29 | BOY | 1.9 | 7,8 | 11:30 |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 1.9 | 7,8 | 5.22 | GIRL | 1.78 | 7,8 | 5.35 |
| | YES | | YES | | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 2.34 | 8,9 | 7.18 | BOY | 2.21 | 7,8 | 7.36 |
| | YES | | YES | | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 2.7 | 8,9 | 3.56 | GIRL | 2 | 8,9 | 3.58 |
| | YES | | YES | | | | | | CEPHALIC, CEPHALIC | LN | | | BOY | 2 | 7,8 | 3.12 | BOY | 2.15 | 7,8 | 3.13 |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 2.3 | 7,8 | 8.42 | BOY | 2.24 | 6,8 | 8.43 |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 2.42 | 7,8 | 6.34 | GIRL | 2.3 | 7,8 | 6.55 |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | BOY | 2.2 | 8,9 | 7.22 | BOY | 2.4 | 8,9 | 7.46 |
| | YES | | | | | | | | | | LSCS | MCMA | GIRL | 2.5 | 8,9 | 10.56 | GIRL | 2.65 | 7,8 | 10.58 |
| | YES | | | YES | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 1.9 | 6,8 | 2.01 | GIRL | 1.7 | 6,8 | 2.14 |
| | | | YES | | | | | | | | LSCS | MSL | BOY | 2.2 | 7,8 | 9.45 | BOY | 2.33 | 8,9 | 9.46 |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | YES | | YES | | CEPHALIC, CEPHALIC | LN | | | BOY | 2 | 8,9 | 12.20 | GIRL | 2.07 | 8,9 | 12.21 |
| | | | YES | | CEPHALIC, CEPHALIC | LN | | | BOY | 2.22 | 8,9 | 3.56 | GIRL | 2.33 | 7,8 | 4.11 |
| | YES | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 2.67 | 8,9 | 5.22 | GIRL | 2.43 | 8,9 | 5.3 |
| | | | | | CEPHALIC, CEPHALIC | LN | | | GIRL | 1.9 | 7,8 | 3.33 | BOY | 2.11 | 7,8 | 3.46 |
| | YES | YES | | | | | LSCS | CORD PROLAPSE | GIRL | 2.14 | 5,7 | 3.55 | GIRL | 2.24 | 8,9 | 3.57 |
| yes | YES | | | | | | LSCS | ABRUPTIO PLACENTA | GIRL | 960 | 7,8 | 2.4 | BOY | 1.07 | 7,8 | 2.55 |
| | YES | | YES | | CEPHALIC, CEPHALIC | LN | | | BOY | 750 GM | 0,0 | 8.43 | BOY | 1.130 | 7,8 | 8.55 |
| | YES | | | | | | LSCS | MCDA FETAL DISTRESS | BOY | 2.08 | 7,8 | 1.4 | BOY | 2.12 | 6,8 | 1.41 |
| | YES | | | | | | LSCS | FETAL DISTRESS | GIRL | 2.3 | 7,8 | 2.33 | BOY | 2.21 | 6,8 | 2.35 |
| | YES | | | | CEPHALIC, CEPHALIC | OUTLET | | | GIRL | 1.29 | 8,9 | 11.34 | GIRL | 1.7 | 7,8 | 11.50 |
| | YES | | YES | | | | LSCS | AP ECLAMPSIA | BOY | 2.6 | 7,8 | 3.56 | BOY | 2.33 | 8,9 | 3.58 |
| | YES | | YES | | CEPHALIC, CEPHALIC | LN | | | BOY | 1.34 | 7,8 | 3.45 | BOY | 2.44 | 8,9 | 3.5 |
| | YES | | | | | | LSCS | PREV LSCS | BOY | 2.3 | 8,9 | 2.21 | BOY | 2.5 | 7,8 | 2.22 |
| | | | | | | | | | | | | | | | | |
| | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.5 | 6,7 | | | 2.6 | 7,8 | |
| | YES | | YES | | | | LSCS | FETAL DISTRESS | | 2.4 | 6,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | LSCS | FETAL DISTRESS | | 2.4 | 6,8 | | | 2.5 | 7,8 | |
| | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.6 | 4,6 | | | 2.6 | 3,7 | |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 1.7 | 5,7 | | | 1.9 | 5,9 | |
| | | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.1 | 5,6 | | | 250 GM | 0,0 | |
| | | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 5,7 | | | 2.8 | 3.8 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.2 | 7,7 | | | 2.5 | 7,8 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.2 | 7,8 | | | 2.5 | 6,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.6 | 5,8 | | | 2.6 | 5,8 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.5 | 6,8 | | | 2.8 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.2 | 4,8 | | | 2.4 | 5,8 | |
| | | | YES | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.5 | 6,8 | | | 2.5 | 6,8 | |
| | | | YES | | | | | | | | LSCS | PREV LSCS | | 2.4 | 5,6 | | | 2.6 | 5,8 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.5 | 5,7 | | | 2.5 | 6,7 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.8 | 6,8 | | | 2.6 | 6,8 | |
| | YES | | | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 4,8 | | | 1.8 | 4,8 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 1.9 | 7,8 | | | 2.1 | 7,8 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.7 | 6,7 | | | 2.6 | 6,7 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 1.8 | 5,6 | | | 1.9 | 5,5 | |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.4 | 7,7 | | | 2.5 | 7,8 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.5 | 6,7 | | | 2.6 | 6,7 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.2 | 6,7 | | | 2.5 | 6,8 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.9 | 5,8 | | | 3.2 | 6,8 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 3.3 | 6,7 | | | 2.9 | 6,8 | |
| yes | YES | | YES | | | | | | | | LSCS | ABRUPTIO PLACENTA | | 3.1 | 6,8 | | | 2.9 | 6,8 | |
| | YES | | | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 5,7 | | | 2.4 | 5,7 | |
| | YES | | YES | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.2 | 7,8 | | | 560 GM | 0,0 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.3 | 7,8 | | | 1.7 | 7,8 | |
| | YES | YES | YES | | | | YES | | | | LSCS | CORD PROLAPSE | | 2.4 | 6,7 | | | 2.3 | 7,8 | |
| | YES | | | YES | | | | | CEPHALIC, BREECH | LN | | | | 2.5 | 7,8 | | | 2.6 | 7,8 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 3.1 | 7,8 | | | 2.9 | 7.8 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 3.1 | 6,7 | | | 2.7 | 6,7 | |
| | YES | | | | | | | | | | LSCS | PREV LSCS | | 2.6 | 7,8 | | | 2.8 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.3 | 4,7 | | | 2.4 | 5,7 | |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | YES | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 6,7 | | | 2.5 | 6,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.9 | 8,9 | | | 3 | 8,9 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 3 | 6,7 | | | 2.6 | 6,7 | |
| | YES | | | | | | | | | | LSCS | MSL | | 2.2 | 7,8 | | | 2.4 | 6,7 | |
| | YES | | YES | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 6,7 | | | 2.6 | 6,7 | |
| | | | | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.7 | 7,8 | | | 2.9 | 7,8 | |
| | YES | | | | | | | | | | LSCS | PREV LSCS | | 2.5 | 7,8 | | | 2.5 | 7,8 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.7 | 6,8 | | | 2.9 | 7,8 | |
| | YES | | | | | | | | | | LSCS | MSL | | 2.7 | 6,7 | | | 2.7 | 6,7 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.9 | 7,8 | | | 2.7 | 6,8 | |
| | YES | | YES | | | | | | CEPHALIC, BREECH | LN | | | | 2.7 | 7,8 | | | 2.8 | 7,8 | |
| | YES | | YES | | | | | | | | LSCS | PLACENTA PREVIA | | 1.7 | 6,7 | | | 2.5 | 6,7 | |
| | YES | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.4 | 5,7 | | | 2.6 | 5,7 | |
| | YES | | | | | | | | | | LSCS | TCTA | | 1.6 | 0,0 | | | 1.7 | 7,8 | |
| | | YES | | YES | | | | | | | LSCS | CORD PROLAPSE | | 2.1 | 6,8 | | | 2.2 | 6,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.6 | 5,7 | | | 2.8 | 5,8 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 2.1 | 7,8 | | | 2 | 7,8 | |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.6 | 6,7 | | | 2.7 | 6,7 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 4,7 | | | 2.6 | 4,7 | |
| | | | YES | | | | YES | | | | LSCS | FETAL DISTRESS | | 2.8 | 7,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 6,8 | | | 2.5 | 4,6 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.5 | 4,7 | | | 2.6 | 5,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.7 | 7,8 | | | 2.6 | 7,8 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 1.7 | 8,9 | | | 1.9 | 8,9 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.5 | 5,8 | | | 2.6 | 6,8 | |
| | | | YES | | | | | | | | LSCS | PREV LSCS | | 2.7 | 7,8 | | | 2.9 | 7,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.8 | 7,8 | | | 2.7 | 8,9 | |
| | YES | | YES | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.7 | 7,8 | | | 2.9 | 7,8 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.7 | 6,7 | | | 2.9 | 6,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 3.1 | 5,7 | | | 2.7 | 5,8 | |
| | YES | | YES | | | | | | | | LSCS | TCTA | | 1.7 | 6,7 | | | 1.4 | 6,7 | |
| | YES | | | YES | | | | | CEPHALIC, BREECH | LN | | | | 2.5 | 3,7 | | | 2.3 | 4,7 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.9 | 7,8 | | | 3 | 7,9 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 1.8 | 7,8 | | | 2.1 | 7,8 | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | LSCS | AP ECLAMPSIA | | 2.6 | 8,9 | | | 2.7 | 8,9 | |
| | YES | | | | | | | | | | LSCS | TCTA | | 1.5 | 7,8 | | | 1.7 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 1.2 | 0,0 | | | 2.4 | 5,7 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.2 | 6,7 | | | 2.1 | 6,7 | |
| | | | | | | | | | | | LSCS | MSL | | 3 | 6,8 | | | 2.7 | 6,9 | |
| | YES | | | | | | | | | | LSCS | MSL | | 2.4 | 6,7 | | | 2.1 | 6,7 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.8 | 7,9 | | | 2.6 | 7,9 | |
| | YES | | YES | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 7,7 | | | 1.9 | 7,7 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.9 | 7,8 | | | 2.7 | 7,8 | |
| | | | YES | | | | | | CEPHALIC, BREECH | LN | | | | 2.8 | 8,9 | | | 2.7 | 8,9 | |
| yes | YES | | | | | | | | | | LSCS | ABRUPTIO PLACENTA | | 2.2 | 6,7 | | | 1.8 | 5,7 | |
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.4 | 8,9 | | | 2.2 | 8,9 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.1 | 6,7 | | | 2.4 | 6,7 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.2 | 7,8 | | | 2.5 | 7,9 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.1 | 7,9 | | | 2 | 7,9 | |
| | YES | | | | | | | | | | LSCS | TCTA | | 1.3 | 6,7 | | | 1.6 | 6,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.5 | 7,9 | | | 2.7 | 7,9 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 1.7 | 6,7 | | | 1.9 | 6,7 | |

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| | YES | | | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 6,8 | | | 2.7 | 6,9 | |
| | | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.4 | 7,8 | | | 2.3 | 7,8 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.4 | 6,7 | | | 2.5 | 6,7 | |
| | YES | YES | | | | | | | | | LSCS | CORD PROLAPSE | | 2.2 | 4,6 | | | 2.1 | 6,7 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 7,9 | | | 2.7 | 7,9 | |
| | | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.6 | 7,9 | | | 2.7 | 7,9 | |
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.2 | 6,9 | | | 2.4 | 6,9 | |
| | YES | | YES | | | | | | | | LSCS | PLACENTA PREVIA | | 2.1 | 7,8 | | | 2.2 | 7,8 | |
| | YES | | | | | | | | | | LSCS | MSL | | 2.2 | 7,9 | | | 2.5 | 7,8 | |
| | YES | | | | | | | | | | LSCS | PREV LSCS | | 2.1 | 6,8 | | | 2 | 6,9 | |
| | | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.5 | 5,7 | | | 2.3 | 5,7 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 7,9 | | | 2.7 | 7,9 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 7,8 | | | 2.3 | 7,8 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 1.8 | 6,7 | | | 1.9 | 7,8 | |
| | YES | | | | | | | | | | LSCS | TCTA | | 1.6 | 7,8 | | | 1.8 | 7,8 | |

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| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 8,9 | | | 2.8 | 8,9 | |
| | YES | | | | | | | | | | LSCS | AP ECLAMPSIA | | 2.2 | 7,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.1 | 5,7 | | | 2.1 | 5,7 | |
| | YES | | YES | YES | | | | | CEPHALIC, CEPHALIC | LN | | | | 1.4 | 6,7 | | | 1.5 | 6,7 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 1.8 | 7,8 | | | 2.4 | 7,8 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.7 | 6,7 | | | 2.9 | 6,8 | |
| | | | YES | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.9 | 7,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.2 | 5,7 | | | 2.3 | 5,7 | |
| | | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.7 | 8,9 | | | 2.1 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.1 | 7,8 | | | 2.4 | 7,8 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.1 | 6,7 | | | 2.5 | 6,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.5 | 4,7 | | | 2.6 | 4,6 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.6 | 7,8 | | | 2.5 | 7,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 6,7 | | | 2.8 | 6,7 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 1.9 | 7,8 | | | 2.1 | 7,8 | |

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| | | | | | | | | | | | LSCS | AP ECLAMPSIA | | 450GM | 0,0 | | | 2.1 | | |
| | | | YES | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 6,7 | | | 2.6 | 4,7 | |
| | YES | | YES | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 1.8 | 6,7 | | | 1.7 | 7,8 | |
| | YES | | | | | | | | | | LSCS | PLACENTA PREVIA | | 2.3 | 6,7 | | | 2.4 | 6,8 | |
| yes | YES | | | | | | | | | | LSCS | ABRUPTIO PLACENTA | | 2.2 | 6,7 | | | 2.4 | 6,7 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.6 | 3,6 | | | 2.5 | 3,8 | |
| | YES | | | | | | | | | | LSCS | AP ECLAMPSIA | | 1.9 | 5,7 | | | 1.8 | 5,7 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 2.2 | 6,7 | | | 2.1 | 7,8 | |
| | | | YES | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.6 | 6,7 | | | 2.7 | 6,8 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.1 | 6,7 | | | 2.2 | 6,7 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 1.9 | 5,8 | | | 2 | 5,7 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.6 | 6,7 | | | 2.8 | 6,7 | |
| | YES | | YES | | | | | | CEPHALIC, CEPHALIC | LN | | | | 2.9 | 3,6 | | | 2.8 | 3,6 | |
| | | | | | | | | | | | LSCS | MSL | | 2.9 | 5,7 | | | 2.9 | 5,7 | |
| | | | | | | | | | | | LSCS | MSL | | 2.7 | 7,8 | | | 2.9 | 7,8 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 1.9 | 6,7 | | | 1.7 | 4,7 | |

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| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.7 | 7,8 | | | 2.8 | 7,9 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.5 | 8,9 | | | 2.7 | 8,9 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.2 | 6,7 | | | 2.5 | 6,7 | |
| | YES | | YES | | | | | | CEPHALIC, BREECH | LN | | | | 2.2 | 5,7 | | | 2.1 | 5,8 | |
| | YES | | | | | | | | | | LSCS | TCTA | | 1.2 | 7,8 | | | 1.1 | 7,8 | |
| | | | YES | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2.5 | 7,8 | | | 2.8 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.6 | 6,7 | | | 2.9 | 6,7 | |
| | YES | | YES | YES | | | | | CEPHALIC, BREECH | LN | | | | 2.2 | 8,9 | | | 2 | 8,7 | |
| | YES | | | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.4 | 6,8 | | | 2.2 | 6,9 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 6,9 | | | 2.8 | 6,8 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 6,8 | | | 2.5 | 6,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 7,8 | | | 2.8 | 7,8 | |
| | YES | | | | | | | | CEPHALIC, CEPHALIC | OUTLET | | | | 2.1 | 8,9 | | | 2.7 | 8,9 | |
| | YES | | | | | | | | | | LSCS | MCMA | | 1.9 | 7,8 | | | 1.8 | 7,7 | |
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.2 | 6,7 | | | 560GM | 0,0 | |

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| | YES | | | | | | | | | | LSCS | MCMA | | 2.1 | 6,7 | | | 2.1 | 6,8 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.1 | 4,7 | | | 2.2 | 4,6 | |
| | | | | | | | | | | | LSCS | PREV LSCS | | 2.6 | 7,8 | | | 2.9 | 7,8 | |
| | | | | | | | | | BREECH,TRANSVERSE LIE | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 6,7 | | | 2.7 | 6,7 | |
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.6 | 8,9 | | | 2.8 | 8,9 | |
| | YES | | YES | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 7,8 | | | 750 GM | 0,0 | |
| | | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.5 | 7,8 | | | 2.5 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.2 | 6,7 | | | 2.5 | 6,7 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.6 | 7,8 | | | 2.6 | 7,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.6 | 7,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.2 | 6,8 | | | 2.1 | 6,8 | |
| | YES | | | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 7,8 | | | 2.7 | 7,8 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.1 | 6,8 | | | 2 | 6,8 | |
| | YES | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2 | 6,8 | | | 2.4 | 6,7 | |
| | YES | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.5 | 6,7 | | | 2.7 | 6,7 | |

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| | | | | | | | | | CEPHALIC, CEPHALIC | VACUUM | | | | 2 | 4,6 | | | 2.6 | 5,7 | |
| | | | YES | | | | | | | | LSCS | FETAL DISTRESS | | 2.5 | 6,8 | | | 2.9 | 6,8 | |
| | | | | | | | | | BREECH,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.4 | 7,8 | | | 2.7 | 7,8 | |
| | | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.4 | 6,8 | | | 2.6 | 6,7 | |
| | YES | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.4 | 7,8 | | | 2.7 | 6,8 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 5,7 | | | 1.8 | 5,7 | |
| | | | | | | | | | CEPHALIC, BREECH | LN | | | | 2.1 | 0,0 | | | 2.1 | 6,7 | |
| | YES | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.1 | 5,7 | | | 2.2 | 5,7 | |
| | YES | | | | | | | | BREECH,CEPHALIC | | LSCS | FIRST TWIN NON CEPHALIC | | 2.1 | 6,7 | | | 2 | 4,6 | |
| | | | | | | | | | | | LSCS | FETAL DISTRESS | | 2.3 | 6,7 | | | 2.1 | 6,7 | |
| | | | YES | | | | YES | | | | LSCS | PREV LSCS | | 2.2 | 7,8 | | | 2.4 | 7,8 | |
| | YES | | | | | | | | TRANSVERSE LIE,BREECH | | LSCS | FIRST TWIN NON CEPHALIC | | 2.2 | 6,7 | | | 2.6 | 4,7 | |
| | | | | | | | | | CEPHALIC, CEPHALIC | OUTLET | | | | 2.6 | 6,8 | | | 2.8 | 6,8 | |
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| **TRIPLET C** | | | | **NICU ADMISSION** | RDS/BIRTH ASPHYXIA | **NEONATAL SEIZURES** | **SEPSIS** |
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| | 1.2 | 7,8 | | YES | | | | |
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