"MATERNAL AND FETAL OUTCOME OF PLACENTA PREVIA IN TERTIARY CARE CENTRE"

By

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Under the guidance of
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LIST OF ABBREVIATIONS USED

APH	-	Antepartum haemorrhage
NICU	-	Neonatal intensive care unit
FFP	-	Fresh frozen plasma
PRBC	-	Packed red blood cells
PNM	-	Perinatal mortality
MMR	-	Maternal mortality rate
IVF	-	Invitro fertilization
TAS	-	Trans-abdominal ultrasound
TVS	-	Trans-vaginal ultrasound
MRI	-	Magnetic Resonance Imaging
LSCS	-	Low segment caesarean section
PP	-	Placenta previa
PIH	-	Pregnancy induced hypertension
PPH	-	Postpartum haemorrhage
IUGR	-	Intra uterine growth retardation

ABSTRACT

Introduction: placenta previa is one of the major causes of antepartum haemorrhage and is an important cause of maternal and fetal morbidity and mortality in India

Aim: To determine the incidence, risk factors, maternal and fetal outcome of placenta previa

Material and method: A cross sectional observational study, conducted in the Department of Obstetrics and Gynaecology at R.L.Jalappa Hospital and Research Centre, Tamaka Kolar attached to Devaraj Urs Medical College from June 2017 to May 2019. All antenatal cases (booked, un booked or referred) with gestational age >/= 28 diagnosed with placenta previa were later observed for risk factors, outcome of pregnancy, maternal morbidity, mortality and fetal outcome.

Results: the incidence of placenta previa is 1.57%, more commonly present among multiparous women and most common in the age group between 20-29years (88%). Most common risk factors were previous LSCS (25%), multiparity (25%) and abortions in 11.8%. Out of 76 cases studied 24 cases had atonic PPH and 2 cases underwent peripartum hysterectomy due to placenta accrete. Most of cause for perinatal mortality was prematurity (46.1%) and respiratory distress (19.7%). preterm deliveries in the present study was between 28-32(18.4%) and 32-36weeks (25%) weeks. there was no maternal mortality in this study.

Conclusion.

Advancing maternal age, multiparity, prior caesarean section, and prior abortions are independent risk factors for placenta previa. An increase in the incidence of placenta previa due to presence of these risk factors. Placenta previa is associated with adverse maternal and foetal outcome, hence early recognition of risk factors and if possible, prevention of these risk factors is therefore important. Early diagnosis, early transfer to tertiary care centre with better management including, early resuscitation and blood transfusion and timely delivery would help to reduce maternal and fetal complications with placenta previa

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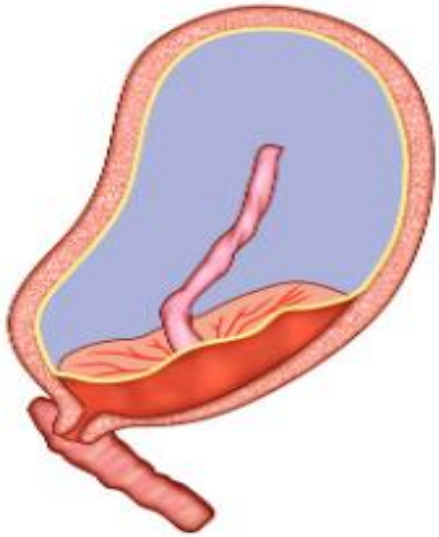

Figure no 1: PLACENTA PREVIA

(Photography courtesy of Dutta text book of obstetrics)

INTRODUCTION

The Latin word previa means going before and in this sense, the placenta goes before the foetus into the birth canal.[1]Placenta previa (PP) is a form of abnormal placentation in which placenta is inserted low in the lower uterine segment, resulting in complete or partial overlaying of the internal cervical ostium. The overall reported estimated incidence of placenta praevia at term is 1 in 200 births.[1]

It is one of the major causes of maternal and neonatal morbidity and mortality[2] because of the associated risk of massive antepartum haemorrhage and intra-partum haemorrhage.[3] The prevalence of placenta previa has been on rise and recently estimated to be approximately 0.5% of all pregnancies. This increase correlates to the elevated caesarean section rate.[3]Caesarean delivery increases risk of placenta previa in subsequent pregnancies. This risk rises as the number of prior caesarean sections

increases. Most of the women with PP required caesarean delivery and commonest cause of maternal morbidity being anaemia. [4]

Risk factors for PP are old age, multiparity, previous caesarean delivery, abortions, Artificial reproductive techniques, maternal smoking, cocaine abuse and male foetus.[3]

Painless, causeless, unprovoked, recurrent bleeding is the hallmark of placenta PP. It is the common cause of 2nd and 3rd trimester bleeding. Bleeding from a previa usually begins without warning and without pain or contractions in a woman who has had an uneventful prenatal course.[1] Initial bleeding in placenta previa is called warning haemorrhage.

Haemorrhage in obstetrics is almost life-threatening emergency especially in the last trimester. Obstetrical haemorrhage along with hypertension and sepsis is one of the infamous "triad" causing maternal deaths in both underdeveloped and developed countries.

It is a leading reason in pregnant women requiring intensive care units' admissions. [5, 6] Any bleeding from or into the genital tract after 28th week of pregnancy but before the birth of the baby is called antepartum haemorrhage (APH). When placenta is implanted partially or wholly situated in the lower uterine segment (LUS) (over and adjacent to internal cervical os) it is called as Placenta previa .In placenta previa, the bleeding occurs from the placental site, which is situated in the LUS, which stretches during the latter half of pregnancy. Postpartum haemorrhage (PPH) is the most common cause of maternal mortality worldwide. More than half of all maternal deaths occur within 24 hours of delivery, mostly from excessive bleeding. [7]

There has been substantial decrease in the maternal mortality in PP throughout the globe because of early diagnosis even prior to the bleeding, avoiding internal examination outside the hospital, availability of blood transfusion facilities, wider use of caesarean section with expert anaesthesiology, skill and judgement. All these factors reduced maternal mortality from placenta previa to <1% or even to zero in some centres.

Because of wide gap of the extension of medical facilities in developing countries and also the difference in the patient's profile between urban and rural population maternal mortality from placenta previa in hospital statistics range from <1% to as high as 5%.

Patients are rushed to referral hospital after repeated bouts of haemorrhage often with history of vaginal examination, inadequate antenatal care, delay in referral, transport difficulties. So, diagnosing placenta previa early and transport them to major referral hospital is very important.

Placenta previa cases are to be managed only in centres with blood transfusion facilities, 24 hours caesarean delivery and neonatal intensive care units (NICU) to attend the preterm babies.

Universal institutional antenatal care of all women to improve their general health and to correct anaemia. Family planning and limitation of births, reducing caesarean delivery rate and thorough screening of the patient with second trimester scan, better referral system, transport and more hospitals with 24 hours blood bank facility are the need of the hour. All these measures can probably decrease the maternal and perinatal mortality and morbidity rate and achieve the standards of developed countries.

Hence early diagnosis of placenta previa and referral to major hospital is very important. Hence this study is undertaken to know the maternal and foetal outcome there by decreasing the maternal and foetal morbidity and mortality.

Shock, sepsis, antepartum, intrapartum and postpartum haemorrhage, placenta previa accrete and peripartum hysterectomy, caesarean hysterectomy are the complications which can increase the morbidity and mortality in pregnant woman.[5,]

Preterm labour, low birth weight, increased rate NICU admission, intra uterine death are foetal complications.[2]

Perinatal mortality and morbidity are high due to preterm labour and its related complications like birth asphyxia, low birth weight and neonatal sepsis.

The maternal and perinatal outcome can be definitely improved in PP as it can be diagnosed in the antenatal period even before the warning haemorrhage. Determining location of placenta is one of the first aims of routine mid pregnancy (18+6to 21+6weeks of gestation) transabdominal obstetric ultrasound examination.

OBJECTIVES

OBJECTIVES OF THE STUDY

1. To estimate the occurrence of placenta previa
2. To analyse the risk factors and clinical presentation of placenta previa
3. To evaluate the maternal and perinatal outcome in placenta previa

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

In a study conducted at Mandya Institute of Medical Sciences, the prevalence of PP was 0.2% and was most commonly present among multiparous women (75.8%). Out of 62 cases in that study,10 had atonic PPH and 2 women underwent peripartum hysterectomy.[3]

The risk of placenta previa increases with advancing maternal age, an association has also been observed with parity.[8]

A study was conducted at a tertiary care hospital which showed the incidence of placenta previa was highest in the maternal age group of 20-29 years (72.9%).[5]Infertility treatment, prior caesarean delivery and advancing maternal age are the independent risk factors for previa. Rise in the incidence of the above factors probably contributes to multiparity complicated with PP and its association with adverse maternal and perinatal outcome. [8]

A hospital-based study conducted at tertiary centre concludes the risk factors for PP were short inter pregnancy interval, scarred uterus, instrumentation and patients with addictions. Most of the women with PP required caesarean delivery and commonest cause of maternal morbidity being anaemia. [4]

Prior dilatation and curettage history, previous caesarean delivery are risk factors for PP. Neonates born to them are at higher risk of premature birth low APGAR score and increased admission to NICU.[2]

According to study conducted in the department of obstetrics and gynaecology of King George hospital, Visakhapatnam the PP cases were highest in the age group 20-29 years 82% and in multiparous group being 74%. Most common risk factor was

previous caesarean section 44% followed by abortions in 24%. Placenta previa major constitutes 74%, adherent PP is seen in 8%, and 14% required hysterectomy[9]

A study was conducted in 2016 according to which artificial reproductive technique, singleton pregnancies reported a RR of 3.71 for placenta praevia .[10] That was confirmed by a meta-analysis conducted in 2017.[11]

Furthermore, a 2017 meta-analysis of the impact of maternal smoking on placental position[6] has found an increased risk of placenta praevia.

Systematic review and meta-analysis of 22 studies including over two million deliveries indicated that the placenta previa incidence increases from ten in one thousand deliveries with 1 previous caesarean delivery to twenty eight in one thousand deliveries with three or more caesarean deliveries.[68] A 2014 meta-analysis confirmed these findings and reported an overall odds ratio of 1.47 for PP after caesarean section.[12]

Cohort studies conducted have also reported that risk of placenta praevia in caesarean delivery increased where the second pregnancy is within 1 year[13].Compared with vaginal birth, a previous pre labour caesarean delivery is associated with an increased risk of placenta previa in the 2nd delivery.

A study by Dr Kavitha B in 2018 concluded the incidence of PP in uterus with previous scar was 2.75% which was much higher than in unscarred uterus which was 1.4%. Anterior placenta previa was seen in 63.8% in scarred uterus and 47.7% in unscarred uterus. Placenta was adherent in in 13.9% in scarred uterus and 4.55 in unscarred uterus. [14]

Primary aim in the form of reduction in rate of primi caesarean section must be done in to prevent risk of placenta previa in scarred uterus.[15]

Women with PP are more likely to have abnormal presentation, preterm delivery, postpartum haemorrhage, obstetrical hysterectomy and postpartum transfusion. New-borns of mothers with PP are more likely to have low birth weight (LBW), APGAR score <7 and increased NICU admission.[16]

Globally APH is an important cause of maternal and perinatal mortality in pregnant women with placenta previa accounting for 51.6%. [17]

Patients with pregnancy complicated by PP had significantly different obstetrical characteristics including bad obstetric history (8% vs.4%), recurrent abortions(11% vs.5%), antepartum haemorrhage in the 2nd half of pregnancy (3% vs 0%),gestational diabetes (8% vs 5.5%), placental abruption (10% vs.1%).Adherent placenta (4% vs0.5%)preterm delivery(52% vs 8%),with a median gestational age of 36 vs 39 weeks <0.001. The composite outcome was significantly more prevalent in the PP group (21% vs 13%).[18]

The data collected from women diagnosed with PP in 10 Austrian hospitals in the province of Styria between 1993 and 2012 the incidence of PP was 0.15%.Maternal morbidity was high due to, antepartum bleeding(42.3%), post-partum haemorrhage (7.1%) , maternal anaemia(30%), comorbid adherent placentation (4%), hysterectomy (5.2%) and neonatal complications were frequent preterm birth 54.9%, low birth weight <2500g 35.6% APGAR score after five minutes <7in (5.8%) , and foetal mortality 1.5% .Women with major PP had a higher incidence of preterm delivery, birth weight less than 2500g and Apgar –score after five minutes <7. [19]

Retrospective cohort of 67 895 singleton and twin pregnancies found that monochorionic (RR 3.29, 95% CI 1.32–8.21) and dichorionic (OR 1.54, 95% CI 1.15–2.06) twin gestation have a higher risk of placenta praevia compared with singletons.[20]

The Cochrane systematic review by Nielson on the impact of an intervention in women diagnosed as having, or likely to have a PP, which has not been updated since October 2002, includes only one small RCT (n=53) comparing hospital versus home care for symptomatic placenta praevia.[21]This trial found little evidence of any clear advantage or disadvantage to a policy of home v/s hospital care, and the only significant difference was a reduction in duration of hospital stay.[22]

Two large retrospective studies of women presenting with praevia at the routine foetal anomaly scan have proposed scores to predict the risk of emergency caesarean delivery. The first study (n=250) found that the risk is increased if the 1st (sentinel) vaginal bleeding episode occurs before 29 weeks of gestation, and with the occurrence of 3 or more episodes of APH .[23]

The 2nd study (n=214) found that independent predictors for emergency delivery are a history of caesarean section; antepartum haemorrhage on one, two, three or more episodes and need for blood transfusion.[24]

The results of these studies suggest that predictors for emergency delivery in women with PP can be used for individualised antenatal care regarding need for hospital admission, corticosteroid administration and timing of delivery.

A large case–control study found that perinatal morbidities in placenta praevia include risk of low 5-minute APGAR scores, NICU admissions, anaemia, respiratory distress syndrome, mechanical ventilation and intraventricular haemorrhage.[25]

There is no evidence, that neonates born after pregnancies with PP are more likely to be small for gestational age (SGA) when compared to non-praevia controls.[26]

Compared with no treatment or placebo with antenatal corticosteroids (betamethasone, dexamethasone or hydrocortisone), there was have decreased adverse outcomes related to prematurity, including perinatal death, respiratory distress syndrome, intraventricular haemorrhage and necrotising enterocolitis antenatal corticosteroids .[27]

The 2016 randomised control study has found that the administration of betamethasone in singleton pregnancy who at risk for late preterm delivery (34+0 to 36+5 weeks of gestation) significantly reduced the rate of neonatal respiratory complications.[28]

A decision analytic model designed to compare total maternal and neonatal quality-adjusted life years for delivery of women at 34+0 to 36+6 weeks of gestation indicated that corticosteroids administration at 35+5 weeks of gestation followed by planned delivery at 36 weeks of gestation optimises maternal and neonatal outcomes.[29]

A US population-based cohort study using the Centres for Disease Control and Prevention's Linked Birth and Infant Death data files has evaluated the effects of delivering placenta previa at 35,36 and 37 weeks of gestation on the risk of several neonatal outcomes.[30] Compared with neonates born at 38 weeks of gestation, those

delivered at gestational age of 35, 36 and 37 weeks have no greater odds of meconium passage, foetal distress, foetal anaemia, neonatal seizures, increased ventilator needs or infant death at 1 year. However, a 5-minute Apgar scores of <7 are greater at thirty five and thirty six weeks of gestation (a OR 3.33, 95% CI 1.71–6.47; and a OR 2.17, 1.11–4.22,respectively) as are odds of NICU admission rates(a OR 2.25, 95% CI 2.01–2.50; and a OR 1.57, 1.38–1.76, respectively) [30]

Women undergoing caesarean section for PP are at high risk of blood loss of more than1000 ml compared with, women undergoing caesarean section for other indications (RR 3.97, 95% CI 3.24–4.85).[31]

Women with anterior placenta are at high risk of blood loss. PP covering the internal cervical os and anterior placentation are independent risk factors (OR 4.1 and OR 3.5, respectively) for massive haemorrhage during caesarean section.[32]

A US case–control study from the National Institute of Child Health and Human Development (NICHD) Maternal-Foetal Medicine Units (MFMU) Network Caesarean Section Registry has shown that morbidity due to maternal haemorrhage is more common in praevia case (19% versus 7%, adjusted RR 2.6, 95% CI 1.9–3.5) and the main associated risk factors with maternal haemorrhage include pre-delivery anaemia, thrombocytopenia, diabetes and magnesium use.[33]

During caesarean delivery maternal complications increases when the primary surgeon is a trainee rather than an experienced surgeon.[27] PP is often associated with additional complications, including fetal malpresentation (transverse or breech presentation) requiring complex intraoperative manoeuvres to deliver the baby.[34]

An RCT of regional versus general anaesthesia for placenta praevia, including women with placenta accreta, has indicated that blood transfusion requirements (although not estimated blood loss) are greater in the general anaesthetic group.[35]

A 4-year observational study at 19 US academic centres of women undergoing caesarean delivery, found that the risk factors for haemorrhage-related morbidity are more in those undergoing general anaesthesia.[35]

A study conducted in the dept of OBG, Shri M P shah govt medical college Gujarat , about the study of associated complications in placenta previa cases , malpresentation contributed to 30%, 1st and 2nd trimester bleeding in 26.66%, severe anemia 6.66%, and pre-eclampsia was found in 3.33% of cases. In the same study 86% of cases required blood transfusion, shock and hypotension was noticed in 43%, PPH in 3.33%.[36]

In a trial of thirteen patients with PP randomly assigned for insertion of a balloon tamponade or haemostatic square sutures for control of intractable postpartum bleeding, operative time and amount of intraoperative bleeding were less in the balloon group (time: 63 versus 78 minutes; intraoperative bleeding: 1520 versus 1946 mL) .[37]

ANTEPARTUM HEMORRHAGE (APH)

APH is defined as any bleeding from or into the genital tract after 28th week of gestation, but before the delivery of the baby (the 1st and 2nd stage of labour is thus included)[38]

Gestational age of 28 weeks is taken as the lower limit of foetal period of viability. The 2 most serious causes are placenta praevia and abruptio placenta.

INCIDENCE

About one-third cases of APH belong to placenta previa. Incidences for placenta previa average 0.3 percent or 1 case per 300 to 400 deliveries.

PLACENTA PREVIA

HISTORICAL ASPECTS

Parisian physician was the first to give description of placenta praevia in 1885, Paul Portal (1630–1703) was the first to describe the placenta attachment to the lower uterine segment.

The elegant, yet poignant, drawings of this disease from partially dissected dead women by the Scot, William Hunter (1718 − 1783), living in London are a vivid reminder of the danger of placenta praevia, which still exists in many countries today.[39]

Mr Edward Rigby in 1775 was the first who differentiated painless third trimester antepartum haemorrhage due to placenta previa, from painful 3rd trimester bleeding due to abruption of placenta. Notelovitz et all 1979 described women with painless abruption of the placenta and Kalstone 1969 described a case with total PP who developed Couvelaire uterus.[40]

Normal physiology

The blastocyst implantation occurs into upper posterior portion uterine wall, which has rich vascular supply. After which there is growth of villi into the decidua. At 1st these chorionic villi surround the blastocyst, soon after the portion villi comes in contact with decidua basalis and proliferates into placenta, remaining atrophies.

The chorionic villi are two types, one opens into intervillous space and other type acts as anchoring villi to stabilize the embryo and placenta and is normally confined to endometrium.

Functional villi have 2 invasions.

Primary invasion occurs into endometrium.

Secondary invasion occurs by invading into 1/3rd of myometrium.

The Nitabuch's membrane limits the invasion of anchoring villi.

Incidence [41,42]

The overall reported incidence of placenta previa at delivery is 1 in 200 births. In the second trimester, placenta previa may occur in up to 6% of pregnancies. The term placental migration has been used to explain this resolution of placenta previa that is noted near term. Three theories have been suggested to account for this phenomenon.

The first hypothesis proposes that as the pregnancy advances, the stationary lower placental edge relocates away from the cervical os with the development of lower uterine segment. Indeed, the LUS has been noted to increase from 0.5cm at 20 weeks to more than 5cm at term.

Secondly, the placenta-free uterine wall has been proposed to grow at a faster rate than the uterine wall covered by the placenta.

A final hypothesis suggests that trophotropism, the growth of trophoblastic tissue away from the cervical os towards the fundus, results in resolution of the placenta previa.

ETIOLOGY[43]

It results due to aberration in local uterine blood supply. The distinction between the areas of chorionic laevae and chorionic frondusum doesn't occur and the developing ovum derive its nourishment directly from the LUS. The blastocyst which usually implants in the thicker more receptive endometrium of the upper uterine segment gets implanted in the endometrium of the isthmus or over previous lower segment uterine scar. Invasion by the trophoblast secures the embryo and when the uterus grows to form a lower segment later in pregnancy, the placenta remains in the lower segment.in the last week of pregnancy ,or in labour the lower segment stretches and thus the inelastic placenta is sheared off the uterine wall with bleeding from the placental bed.

The decidua basalis is less developed in LUS. The fibrinoid layer of Nitabuch which stops further invasion of chorionic villi may be absent. Therefore placenta tissue may come into direct contact with the myometrial tissue leading to placenta accreta, Increta and percreta.

1 Dropping down theory[44]

The fertilized ovum drops down and its implantation in the lower segment. This could be the result of poor decidual reaction in the upper segment. It explains central PP. Failure of zona pellucida to disappear in time can be a hypothetical possibility. This explains the formation of central placenta previa.

2 Persistence of chorionic activity [44]in the decidua capsularis and its subsequent development into capsular placenta which comes in contact with decidua vera of the lower segment can explain the formation of lesser degrees of PP.

Defective decidua results in spreading of the chorionic villi over a wide area in the uterine wall to get nourishment.

During this process, not only the placenta becomes membranous but encroaches onto the lower segment. Such a PP may invade the underlying decidua or myometrium to cause placenta accreta, increta or percreta

Hyperplacentosis Bigger surface area of the placenta as in twins, Rh iso-immunization placenta may encroach onto the lower segment.

3 Placental Migration theory[45]

Placental migration is called trophotropism ,up to 5% women have ultrasound evidence of low lying placenta at 16-18 weeks, but only 0.5% have placenta previa at term. It is called placental migration and is due to differential growth of lower and upper segment as pregnancy progresses. During third trimester there is tenfold growth of LUS as compared with the placenta. This apparent movement may be due to

- Development of LUS in later part of pregnancy
- Placenta grows preferentially towards better vascularised fundus and the placenta over less vascularised cervix atrophies

Placental migration is less likely[43]

- Placenta is posterior, thick
- Lower edge is less than 2cm from the os
- History of previous caesarean section

Placental migration has been quantified in several studies-

Sanderson and Milton (1991) [46]studied 4300 women at mid pregnancy and found that 12 percent had a low-lying placenta. Of those not covering the internal os, previa did not persist, and none subsequently had placental hemorrhage. Conversely, approximately 40 percent of placentas that covered the os at mid pregnancy continued to do so until delivery. Thus, placentas that lie close to but not over the internal os up to the early third trimester are unlikely to persist as a previa by term.

(Dashe, 2002; Laughon, 2005; Robinson, 2012)[47,48,49] Bohrer and associates (2012)[50] reported that a 2nd trimester low lying placenta was associated with antepartum admission for haemorrhage and increased blood loss at delivery.

Stafford and coworkers(2010)[51] but not Trudell and colleagues (2013)[46] found that a previa and a 3rd trimester cervical length less than thirty mm increased the risk for hemorrhage, uterine activity, and preterm labour.

Friszer and associates (2013)[52] showed that women admitted for bleeding had a greater chance of delivery by 7 days with the cervix < 25 mm, although Trudell [53] and colleagues (2013) did not confirm this.

PLACENTA PREVIA CLASSIFICATION

Placenta praevia was originally defined using transabdominal scan (TAS) as placenta developing within the LUS and classified according to the relationship and the distance between the lower placental edge and the internal cervical os . In 1980s the introduction of transvaginal scanning (TVS) in obstetrics has allowed for a more precise evaluation of the distance between the placental edge and internal os.

JAUNIX AND CAMPBELL in 1921 classified placenta previa into 4 types[54]

- Type 1(Low lying) placenta previa: The placenta encroaches on LUS but does not reach as far as the internal os.
- Type 2(Marginal) placenta previa: The placenta edge reaches the margin of internal os but does not cover it.
- Type 3(Partial central or incomplete) placenta previa: Placenta partially covers the internal os.
- Type 4(Total or central) placenta previa: When the placenta completely covers the internal os.

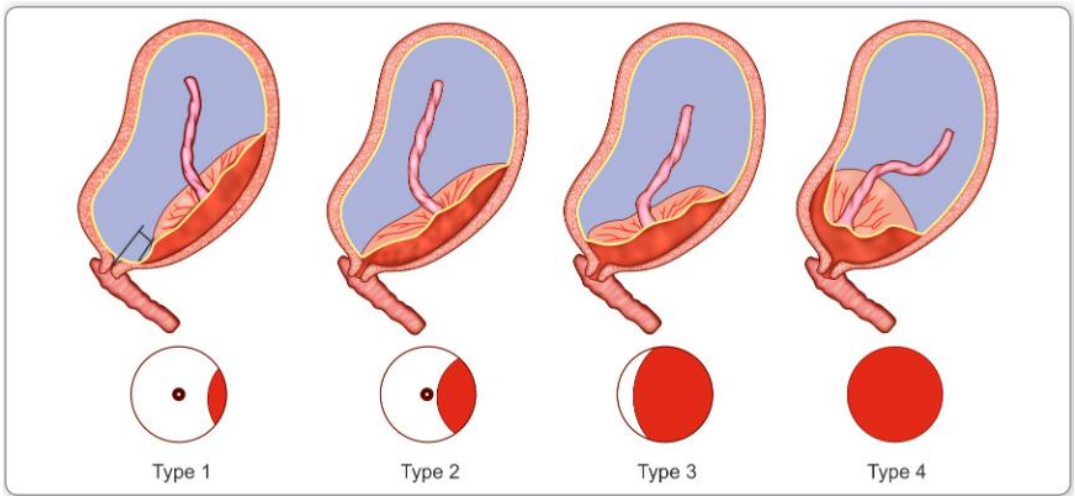

Fig no. 2 classification of placenta previa (Photography courtesy of Dutta text book of obstetrics)

According to American Institute Of Ultrasound in medicine [55]

A recent multidisciplinary workshop of the American Institute of Ultrasound in Medicine (AIUM) 8 has recommended to avoid the terms 'partial' and 'marginal'

placenta previa suggesting that the term 'placenta praevia' where the placenta lies directly over the internal cervical os.

For pregnancies >16 weeks of gestation, the placenta should be reported as 'low lying' when the placental edge is < 2 cm from the internal cervical os, and as normal when the placental edge is 2 cm or more from the internal os on TAS or TVS.

Oppenheimer A classification of PP [AMJ Obstetricgynecal 2009][56]

To describe the distance on TVS which is performed within 28 days of term in the following way. Oppenheimer et al. (1991) performed transvaginal sonography and measured distance between the placental lower edge and internal os

>20 mm away from internal os	Caesarean section not indicated
With in 11-20 mm from internal os	Caesarean section indicated
With in 0-10 mm from internal os	Caesarean section indicated
Overlap of internal os	Caesarean section indicated

RCOG Classification 2018 [27]

However clinically placenta previa is commonly classified into Major or Minor placenta previa depending upon whether it covers internal os or not respectively

Grades I and II are also often defined as '**Minor'** placenta praevia

Grades III and IV are referred to as '**Major'** placenta praevia

The classification of PP is very important as the maternal and fetal morbidity and mortality increases as the grade of PP increases, regardless of type of classification used, also classification assists in management decision.

RISK FACTORS

1. PARITY

Multiparity reported to have Five percent risk for PP compared with 0.2 percent among nulliparous women [57]The risk for PP increases with parity. The obvious effects of old age and parity are confounding. Still, Babinszki and colleagues (1999) reported that the 2.2-percent significant increase in incidence of PP in women with multiparity was compared with that of women with lower parity.[58]

Incidence of PP is higher with increasing parity .[59]

1.2 times following 1 delivery

1.5 times following two deliveries

0.2 percent in nulliparous and in grand multiparas it is 5%

Incidence of PP was 2.2% in multipara ang it is 0.3% in general population

LUS formation occur in weeks leading up to labour in nulliparous women. This development in less pronounced in multi parous women and occur as part of the labour process. This explains the large observed difference in incidence of PP between nulliparous and multiparous.

2. Advanced maternal age [1]

"The frequency of previa increases with maternal age (Biro, 2012).[60]

At Parkland Hospital, this incidence increased from a low rate of approximately one in 1660 for women nineteen years or younger to almost one in 100 for women older than 35.

Coincidental with increasing maternal age in the United States and Australia, the overall PP incidence has increased substantively (Frederiksen, 1999; Roberts,2012).

The FASTER Trial, which included more than 36,000 women, cited the frequency of previa to be 0.5 percent for women <35 years compared with 1.1% among those women > 35 years (Cleary-Goldman, 2005) [61]

With advancing maternal age, collagen slowly replaces normal smooth muscles of myometrial arteries. These lesions may restrict the luminal expansion of the arteries and restricting the blood supply to placenta.

The In older women atrophic changes may also lead to defective decidual vascularization. Both under vascularization and under perfusion have been postulated in development of placenta previa.

3. Maternal race[57]

A large population-based cohort, the rate of PP among white, black and other races was 3.3, 3 and 4.5 per one thousand live births respectively. Asian women appeared to have the highest rates of placenta previa.

4. **Ethnic origin and socio-economic status**

In one study conducted to know the relation between ethnic origin on the incidence of PP they found higher incidence in Asian women when compared to white women.

5. Cigarette smoking: [62]

Smoking has a three-fold increased risk for PP formation, probably due to defective decidual vascularization and hypoxaemia which leads to compensatory hypertrophy

of the placenta. Women smoking >20 cigarettes/day had a 2.6 to 4.4 times higher risk of PP when compared with non-smokers.

6. Cocaine abuse-[43]

A positive correlation between maternal cocaine and opiate use has been demonstrated with placenta previa. study conducted in 1991 have shown a 2-fold high risk of PP and is confirmed by other studies of Ananth (2003).

7. Assisted reproductive technique [55]

PP is more common after ART including in vitro fertilisation and Intracytoplasmic sperm injection. The prevalence is 6-fold higher in in vitro fertilisation pregnancies compared to spontaneous conception, 4-fold higher in Intracytoplasmic sperm injection pregnancies.

8. Multifetal gestation [63]

The rate of PP is 40% higher among twin births in comparison to singleton pregnancies because of the bigger size of placenta, which may encroach on LUS.

9. Previous PP (recurrence of 2.4%)[64]

The recurrence rate of placenta previa is 2.4%, which is an 8-fold increase compared to the normal incidence.

10. Previous caesarean delivery [65,66]

In a study conducted by DR Kavitha B in 2018 the incidence of PP in a woman with previous uterine scar was 2.75% which was much higher than in unscarred uterus which was 1.4%. Anterior placenta previa was seen in 63.8% in scarred uterus and

47.7% in unscarred uterus. Placenta was adherent in in 13.9% in scarred uterus and 4.55 in unscarred uterus.[67]

LSCS, abortions and intrauterine surgery and previous history of placenta removed manually have shown to raise the risk of PP, and is highest in the pregnancy immediately following LSCS and rises with number of prior LSCS. A 2.6-fold rise in the risk of placenta previa is seen in subsequent pregnancy. The incidence of placenta praevia increases from 10 in 1000 deliveries with 1 previous caesarean delivery to 28 in one thousand deliveries with three or more caesarean deliveries.[27] A 2014 meta-analysis confirmed the findings and reported an overall odds ratio of 1.47 for PP after caesarean section.

In an unscarred uterus with placenta previa, the risk of placenta accreta is 5%.This rises to 24% with PP and one previous LSCS and rises to 67% with PP and 4 or more Caesarean Section (Clark et al 1985), prior LSCS with PP increase incidence of Caesarean hysterectomy by 25%, Friederiksen[68]and co-workers 1999 reported 25 percent hysterectomy rate in women with repeat section with previa with only six percent compared in primary caesarean for placenta previa.

11. <u>Endometrial damage associated with previous abortions</u> [69]

Damage to endometrium or myometrium has been shown to be a risk factor for low placental implantation. There is significant connection between previous history of dilation and curettage with placenta previa.

12. Preterm delivery [70]

Spontaneous delivery at gestational age <37 weeks is more commonly complicated by bleeding from a low-lying placenta than delivery at term pregnancy. In a study they examined 198 placentae from preterm labour at period of gestation between 28-37 week and reported an incidence of 2.91 percent for PP and low implantation. This may be due to delivery before the LUS is well formed. Hence chances of migration of placenta is less.

Another possibility is that the uterine contractions are caused by stress activation of the foetal hypothalamic-pituitary-adrenal axis secondary to foetal growth restriction that is a common finding in women with abnormal placentation.

13. Uterine Scar and Pathology

Uterine scars from surgical procedures such as myomectomy, endometritis, submucous fibroids, adenomyosis and uterine adhesions may all be predisposing factors to placenta previa due to endometrial damage and subsequent endometrial scars under perfusion of placenta leads to enlargement of placenta because of scar formation in LUS. Migration of placenta is hindered and defective decidual vascularization results the possible end result of inflammatory and atrophic changes. A second pregnancy within a year after caesarean delivery has 1.7 times higher incidence of placenta previa.[71]

14. Placental pathology

Placental membranacea is an extremely rare cause of PP, marginal or velamentous cord insertions, Succenturate lobes, bipartite placenta and fenestrated placenta are all commonly seen in PP.

15. Elevated Prenatal Screening MSAFP Levels [72]

Women who have otherwise unexplained abnormally elevated prenatal screening levels of maternal serum alpha-fetoprotein (MSAFP) are at an increased risk for PP and a host of other abnormalities.

Women with a PP who also had a MSAFP level ≥ 2.0 MoM at 16 weeks of gestation were at increased risk for late-pregnancy bleeding and preterm labour.

CLINICAL PRESENTATION

The most common clinical presentation of placenta previa is painless vaginal bleeding. And undoubtedly, some late abortions are caused by an abnormally located placenta. Bleeding from a previa usually begins without warning and without pain or contractions in a woman who has had an uneventful prenatal course.

This so-called sentinel bleed is rarely so profuse as to proved fatal. Usually it ceases, only to recur. In perhaps 10 % of women, particularly those with a placenta implanted near but not over the internal cervical os, there will be no bleeding until labour onset. Bleeding at this time varies from slight to profuse, and it may clinically mimic placental abruption. A specific sequence of events leads to bleeding in cases where the placenta is overlying on the internal cervical os.

Uterine body under goes remodels first to form the LUS. With this, the cervical os dilates, and some of the implanted placenta inevitably separates. Bleeding that ensues is augmented by the inherent inability of myometrium in the lower uterine segment to contract and there by constrict avulsed vessels. Similarly, bleeding from the lower segment site of implantation also frequently continues after placental delivery.

Last, there may be lacerations in the friable cervix. This may be especially problematic following manual removal of a somewhat adhered placenta.

2. Malpresentation[-50]

Malpresentations like transverse lies or breech presentation were observed in 30% of cases. In either of the above case the effect claimed was reduced available length of the uterus, hence predispose to foetal position other than the longitudinal lie.

Women with PP have a higher risk of foetal malpresentation such as breech or transverse lie when compared to women with normal placental sites.

It has been suggested that the combination of marginal PP and breech presentation increases the Caesarean Section rate associated with previa.

Malpresentation in PP is assumed to be due to the placental bulk in the lower uterine segment there by preventing engagement of foetal head.

3. Abnormal placentation

"Placenta accreta spectrum risk rises with the number of previous caesarean sections.

Systematic review reported that the incidence of placenta accreta increase from 3.3–4.0% in women with PP and number of previous caesarean delivery, to 50–67% in women with 3 or more caesarean deliveries. [35]

When stratified for the number of previous caesarean delivery, the OR for placenta accreta spectrum in a subsequent pregnancy ranges between 8.6 (95% CI 3.536–21.078) and 17.4 (95% CI 9.0–31.4) for 2 previous caesarean sections, and 55.9 (95% CI 25.0–110.3) for 3 or more caesarean sections.[35]

4. Congenital abnormalities

A study by Kancherla V et al. reported that women with PP more frequently delivered foetuses with serious congenital malformations.[73]

5. Small for gestational age (SGA)

There is conflicting evidence regarding the connection between PP and SGA babies i,e birth weight of less than 10^{th} percentile for gestation, rates as high as sixteen percent have been noted.

6. Vasa previa

On rare occasions blood loss in APH is foetal and not maternal. A child can be born with bleeding from rupture of umbilical vessel, where the cord had a velamentous insertion. Curl and Johnson (1968) demonstrated the pulsating vessels within the cervix with intact membranes, whose constant compression caused foetal bradycardia.

RCOG 2011[74]-Vasa previa occurs when the feta vessels run through the free placental membranes. Unprotected by placental tissue or wharton's jelly of the umbilical cord, a vasa previa is likely to rupture in active labour, or when amniotomy is performed to induce or augment labour, in particular when near or over the cervix, under the foetal presenting part.

This can be due to a velamentous cord insertion in a single or bilobed placenta (vasa praevia type1) or from foetal vessels when it connects the placenta with a succenturiate or accessory lobes (vasa previa type2).

The reported prevalence ranging between 1 in 1200 and 1 in 5000 pregnancies. Unlike placenta previa, vasa previa carries no major maternal risk, but is associated

with significant risk to foetus. When the foetal membrane is ruptured either spontaneously or artificially, the unprotected foetal blood vessels are at risk of disruption with consequent foetal haemorrhage. Vasa previa therefore often presents with fresh vaginal bleeding at the time of membrane rupture and abnormal foetal heart rate patterns such as decelerations, bradycardia, a sinusoidal trace.

7. Posterior placenta previa

"Placenta which is situated posteriorly hinders easy engagement of the head and pressure applied to the foetal head in cephalic presentation, will compress the placenta and there by causes a decrease in foetal heart rate, the Stallworthys sign.

It lies over the sacral promontory and because of placental bulk it decreases the available space at the brim for the head to pass through the cervix hence the presenting part remains high and it will be found difficult to push the head through it. It is associated with the battledore placenta in which cord is attached to margin of placenta hence cord compression and abnormal FHR pattern result and there by causes foetal death. Ultra sound scan of a posterior placenta previa is likely if the distance between foetal skull bone and adjacent sacral margin is greater than 15 mm (Anderson Wellin, 1992) with accuracy rate of 95%, false negative of 0.7%.

EXAMINATION

Patients general condition depends on the amount of bleeding at the time of admission. On examination the patient may be tachycardic, anaemic and may also have hypotension. General condition and anaemia are proportionate to the visible blood loss.

On abdominal palpation reveals soft nontender uterus that relaxes in between the contractions unless the women are in labour. Malpresentations is common, if it is cephalic presentation, head is usually mobile (cannot be pushed down into pelvis).

DIAGNOSIS:

Various methods have been used to diagnose placenta previa.

Double set up technique

Digital vaginal examination may cause haemorrhage. Because local causes are likely to be benign, speculum examination is probably best deferred until ultrasonography has excluded the diagnosis. It is performed using double set up technique. [1]

Previa should not be excluded until sonographic evaluation has clearly proved its absence. If sonography is not readily available, diagnosis by clinical examination is done using the double set up technique because it requires that a finger be passed through the cervix and the placenta palpated. A digital examination should not be performed unless delivery is planned. A cervical digital examination is done with the woman in an operating room and with preparations for immediate caesarean delivery. Even the gentlest examination can cause torrential haemorrhage. Fortunately, double set up examination is rarely necessary because placental location can almost always be ascertained sonographically. [1]Hence prior to any digital vaginal examination, it is of importance in determining placental location to rule out placenta previa.

Placental localization

The diagnostic techniques include

- Sonographic placental localisation – Transabdominal scan, TVS, Trans perineal colour Doppler
- Soft tissue radiography
- Radio isotope placentography
- Air cystography
- Infrared thermographic placentography
- MRI

Transabdominal sonography [27]

"Diagnostic ultrasound scanning is simple, safest, non-invasive and most accurate method of diagnosis of PP. As part of the routine anomaly scan at 20 to 22 weeks' gestation placental localization is considered in many centres, there by identifying women at risk of persisting placenta praevia or a low-lying placenta Placental site location at 20-week of gestation by transabdominal ultrasound has a low positive predictive value for placenta previa later in pregnancy.

The term placenta praevia is used when the placenta lies directly over the internal cervical os. For pregnancies at more than 16 weeks the term low-lying placenta should be used when the placental edge < 2 cm from the internal os on TAS or TVS.

Conversely a false-negative scan for a low-lying placenta is seen in as many as 7% of patients at twenty weeks of gestation. These results are common when the maternal

bladder is full, posteriorly situated placenta, the foetal head obscures the margin of the placenta, or the operator does not scan the lateral uterine wall.

Low-lying placenta is more common in early pregnancy because the LUs does not exist. The apparent migration of placenta is caused by enlargement of the upper segment and LUS formation. Many apparently low placentas are found above the lower segment. Comeau and associates, Ruparelia and Chapman showed that the more advanced the pregnancy, the more accurate the diagnosis of PP based on scanning findings.[27]Migration of placental is less likely to occur in cases with a previous caesarean delivery.

In twin gestation, the likelihood of persistence of PP is also dependent on the period of gestation at ultrasonography detection. Among those with PP diagnosed in the 2nd trimester the majority of cases resolve by 32 weeks of gestation.[75]

Apparent placental 'migration' following the development of the lower segment during the 3rd trimester of pregnancy results in the resolution of the low-lying placenta in 90% of the cases before term.[76,28]

Fifty percent of the remaining PP will resolve after 32 weeks of gestation around, with no further changes after 36 weeks of gestation.

After 2nd trimester scanning ninety five percent migration is seen, extension of LUS from 0.5 cm at gestational age of 20 weeks to five cm at term is the cause for this seemingly upward movement of the placenta for distance of 3-9 cm this is consistent with observation of degree of elongation of the LUS so a repeat scan advised at 30-32 weeks.

The timing of a confirmatory ultrasound examination in the 3rd trimester has varied between gestational age 32 weeks and 36 weeks depending on the extent of the placenta praevia over the internal cervical os.[27]

Although placental localization can be made with 100% accuracy by ultrasound, the determination of whether the placenta crosses the cervix is less accurate due to difficulty in precisely localizing the internal cervical os .

When the lower portion of the placenta is detected inferior to the presenting parts and covers the region of the internal os then diagnosed as anterior PP, whereas posterior PP will displace the presenting foetal part anteriorly and cover the os. To diagnose marginal or partial placenta previa, it is important to record the echoes from the cervical canal or posterior vagina. This is best accomplished with maternal bladder distended to the patient's tolerance.

Although transabdominal Sonography has become the method of choice for diagnosing placenta previa, there are several technical problems when using transabdominal Sonography for outlining the placenta location and its relation with internal os

Most of the false positive results are due to myometrial contractions and/ or an over distended bladder. Myometrial contractions may simulate placenta or displace the placental edge low down, whereas, in distended bladder anterior and posterior uterine walls are compressed together giving false impression of PP. In late pregnancy false negative rate of Transabdominal Sonography is 2%.

In women with a persistent low-lying placenta or PP at gestational age of 32 weeks who remain asymptomatic, an additional trans vaginal ultrasonography is

recommended at around 36 weeks of gestation to plan and discuss about place of delivery and mode of delivery.

In asymptomatic women with placenta praevia, measurement of cervical length may help facilitate management decisions. A short cervical length on trans vaginal ultrasonography before gestational age of 34 weeks increases the risk of emergency preterm delivery and massive haemorrhage at caesarean section.[27]

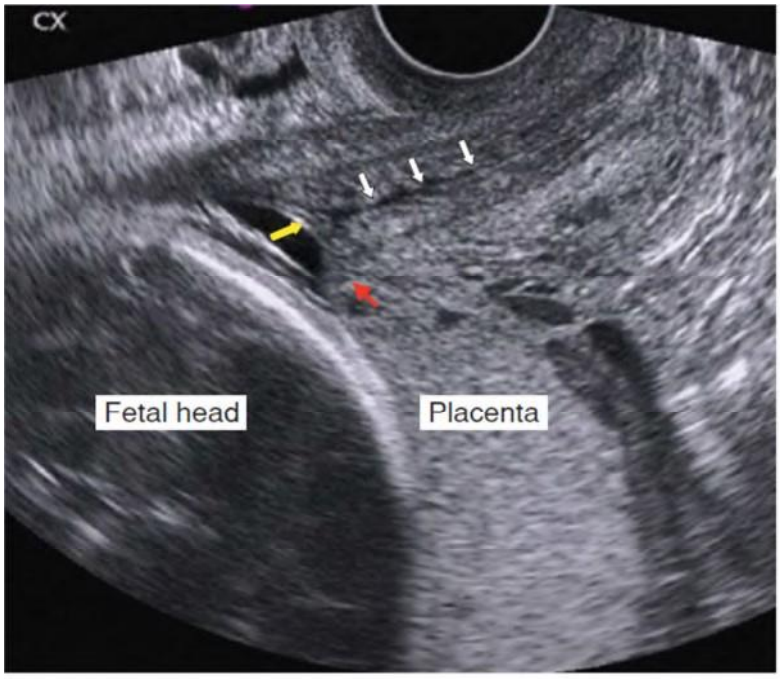

Fig no.3 Transvaginal sonogram of an anterior placenta previa. (Photography courtesy of Williams text book of obstetrics 24th edition)

The placental margin (red arrow) extends downwards towards the cervix. The internal os (yellow arrow) and cervical canal (short white arrows) are marked to show their relationship to the leading edge of the placenta.

Transvaginal sonography (TVS)[27]

TVS is safe and accurate method of diagnosis especially in posterior PP and provides better resolution with visualization of placental edge and the internal os . It is not only accurate but has the benefit of reduced scanning time with sensitivity of 87.5% and specificity of 98.8%. (Leerentred et al.,1990)[53].

It is seen that 26-60% of cases in second and 12.5% in third trimester diagnosed as Placenta Previa by transabdominal scan reclassified by TVS as normally placed placenta

In asymptomatic minor PP cases follow up scan can be left until 36 weeks whereas ,scan should be performed at 32 weeks in asymptomatic major placenta previa to plan management. (RCOG 2011)

Diagnosis of PP is made when the distance between placental edge and internal os is <2cm. The positive value of TVS in diagnosing PP is 71% when compared to TAS which is 31%. TVS is more accurate than TAS in diagnosing placenta previa. When TVUS is not available Transperineally or trans labial ultrasound may be a useful alternative, can also improve upon the diagnostic accuracy of transabdominal ultrasound. Trans vaginal sonography is superior when compared to transabdominal and trans perineal approaches, and is safe.

Placenta previa accreta spectrum are now the consequence of low placentation into a previous caesarean section scar, TVS plays crucial role in the early diagnosis, follow-up, differential diagnosis between adherent and invasive accreta placentation, and further management of placenta accreta spectrum.

Ultrasound criteria of placenta accrete 2D Grey scale

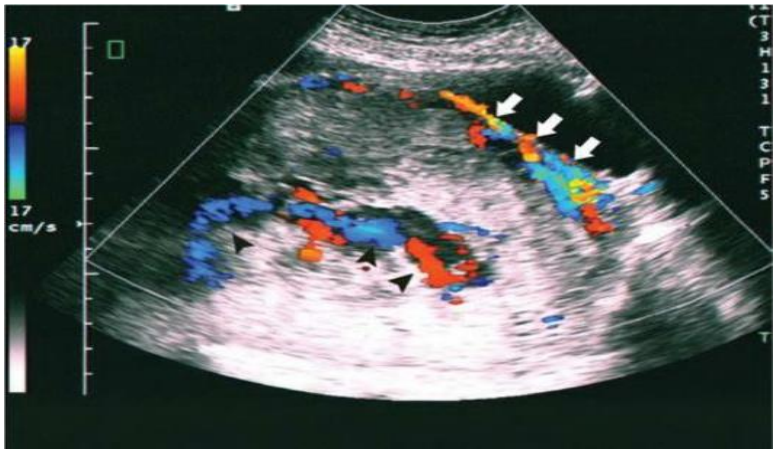

Fig no.4 Transvaginal sonogram of placental invasion with accrete syndrome.

(Photography courtesy of Williams text book of obstetrics 24th edition)

Retroplacental vessels (white arrows) invading the myometrium and obscure the bladder-serosal interface.

- Loss of the retroplacental hypoechoic clear zone
- Loss of bladder wall-uterine interface
- Presence of abnormal placental lacunae
- Myometrial thinning
- Placental bulge
- Focal exfoliating mass
- Bridging vessels
- Placental lacunae feeder vessels
- Presence of hypervascularity of the interface between the uterine serosa and the bladder wall on colour doppler

<u>Colour Doppler</u>

In patients with persistent and anterior PP colour doppler imaging improves the diagnostic accuracy in the prediction of placenta accrete . According to RCOG In patients with a previous cesarean section and an anterior PP should have colour-flow Doppler as the choice of investigation to rule out placenta accrete.

2D Doppler findings in accreta

Focal lacunar flow pattern

Loss of retroplacental hypoechoic clear zone

Vascular lakes with turbulent flow (peak systolic velocity over 15cm/s)

Hypervascularity of the interface between the uterine serosa-bladder wall

Markedly dilated vessels over peripheral sub placental zone.

Three dimensional power Doppler:

Intra placental hypervascularity

Complex, irregular arrangement of numerous placental vessels, exhibiting tortuous courses and varying calibres.

Numerous coherent vessels involving the whole of the uterine serosa- bladder junction (basal view)

Hyper vascularity (lateral view) **,**Inseparable cotyledonal and intervillous Inseparable circulations, chaotic branching, detour vessels (latera view).

2. Soft tissue radiography

Direct visualization of placenta or Soft tissue radiography was a reliable technique before sonar imaging was developed, this method still has place where sonar is not available.

3. Radioisotope placentography

Radioisotope placentography is an acceptable alternative when sonar is not available. In this method an Intravenous injection of $99^{m}Tc$ bound to red blood cell. The placenta is detected because of the greater blood pool in relation to the adjacent myometrium.

4. Air cytography

This method is mainly applicable to cephalic presentation and can detect previa in the anterior and anterolateral position of LUS. Only 0.8 to 1.5 cm of soft tissues lie between bladder and foetal head in normal cases. Displacement of head upwards occurs by placental tissue there by increasing the distance up to 3 or 4 cm

5. Magnetic resonance imaging (MRI)

MRI is a complimentary imaging modality to ultrasound for the evaluation of the gravid uterus. Its use is advocated when ultrasonographic findings are equivocal or when ultrasonography is non diagnostic secondary to technical limitations.

MRI similar to ultrasound involves no ionizing radiation, it is non-invasive and provides images in multiple planes.

In case of previa sonographic diagnosis is difficult when the foetal head obscures a posterior previa or when a low-lying placenta is laterally located. A sagittal MRI can

precisely delineate the relationships of the inferior aspects of the placenta to the internal cervical os.

"MRI findings placenta accrete [27]

The main MRI features of placenta accreta include

- Abnormal uterine bulging
- Disorganised vasculature of placenta and disruption of the uteroplacental zone
- Focal thinning or absence of the myometrium at the site of placental implantation
- Nodular interface between the placenta on the uterus causing outer bulge
- Loss of tissue plane between placenta and bladder wall
- Heterogeneous signals intensity within the placenta
- Dark intraplacental bands over T2- weighted imaging :

MANAGEMENT[38]

Major decision concerns the ideal institution for delivery.

Exigencies to be considered are appropriate surgical, anaesthesia, and blood banking facilities .

An obstetrical surgeon, urological, and interventional radiological consultant should be available (Eller,2011; Stafford, 2008).

PREVENTION:

- Placenta previa is not usually preventable as in most cases the aetiology is not known. The following guidelines are useful to minimise the risks.

- Adequate antenatal care to improve their general health status and to correct anaemia.
- Family planning and limitation of births
- Reducing rising rate of caesarean deliveries as previous caesarean delivery predispose to PP
- Universal target scan at 18 weeks of gestation can diagnose low lying placenta which can later become placenta previa

Asymptomatic patients

Despite the risk of APH, the most common outcome for placenta praevia is to remain asymptomatic until planned admission for caesarean section. As antenatal care evolves, the following clinical factors are important:

- Asymptomatic women remaining ambulant with major placenta praevia should be counselled specifically on the following: to be with other adults at all times, carry a cell-phone, consider not driving, stay in urban areas, avoid flying or other travel, avoid sexual intercourse and constipation. Such women should be certified off work on medical grounds in the third trimester, and earlier if any APH occurs. Hospital triage unit contacts should be reviewed.
- Explained about the risks of preterm labour and obstetric haemorrhage, and their care should be tailored according to their individual needs.
- Maintain haemoglobin of > 10g/dL or treat iron deficiency − if necessary, with IV iron.[77]
- Antenatal corticosteroid therapy is recommended for women with a low-lying placenta or PP between 34+0 and 35+6 weeks of gestation and is appropriate prior

to 34+0 weeks of gestation in women at higher risk of preterm birth. (RCOG 2018)

- Timing of delivery should be individualized according to antenatal symptoms. Women who present with uncomplicated placenta praevia, delivery should be considered between 36+0 weeks and 37+0 weeks of gestation. [27]
- Refine gestational age for caesarean delivery based on several factors, including abnormal lie, recurrent APH, co-morbidities (e.g. hypertension), fetal wellbeing, a prior history of preterm delivery and cervical length.[27]
- The risk of major haemorrhage increases rapidly after 36 weeks, expert opinions have highlighted that decisions regarding timing of delivery must be individualised and suggest that on the basis of the limited data available, women with uncomplicated praevia should undergo scheduled birth by caesarean section between 36 and 37 weeks of gestation.[78,79]
- Women with asymptomatic low-lying placenta in third trimester the mode of delivery should be based on the clinical background, the woman's preferences, and supplemented by ultrasonography findings, including the distance between the placental edge and the foetal head position relative to the leading edge of the placenta on TVS.[27]
- Prior to delivery, all women with PP and their partners should have a discussion regarding delivery. Need for blood transfusion and peripartum hysterectomy should be reviewed and any plans to decline blood or blood products should be discussed openly and documented.
- Anterior placenta previa and low-lying placenta carry a higher risk of massive obstetric haemorrhage and peripartum hysterectomy. Delivery should be planned in health care centre with blood transfusion services and access to critical care.

- In a woman with PP, planned for caesarean section the surgical procedure should be carried out by an experienced obstetrician and a senior obstetrician (usually a consultant) and senior anaesthetist (usually a consultant) should be present within the delivery or operation theatre where the surgery is occurring.[27]
- Careful assessment for invasive placentation in women with major placenta previa and previous caesarean section.
- When an emergency arises, the senior obstetrician and senior anaesthetist should be immediately alerted and attend urgently.
- Regional anaesthesia is safe and risks of haemorrhage is less when compared to general anaesthesia.[27]
- Close communication with the hospital transfusion laboratory facility is essential for women with PP or a low-lying placenta.[27]
- Fluid warming and rapid infusion devices should be immediately available.[27]
- Fresh frozen plasma, Red cells, and cryoprecipitate or fibrinogen concentrate are all kept by blood banks supplying obstetric units. If the haemoglobin is < 7 g/dl in the postoperative period, and no ongoing or threat of bleeding, the decision to transfuse should be made on an informed individual basis.[79]
- In an extreme situation and when the blood group is unknown, group O rhesus D-negative red cells should be given.[79]

Symptomatic patients:

Patient presenting with bleeding

Management at home

Once antepartum haemorrhage is visible

- The patient is immediately put to bed
- Assessment of the blood loss
- Inspection of the clothing's soaked with blood
- To note the pulse, blood pressure and degree of anaemia
- Quick but gentle abdominal examination to mark the fundal height, to auscultate the foetal heart sound and to note any tenderness on the uterus
- Vaginal examination must not be done.

Transfer to hospital Arrangement is made to shift the patient to a well-equipped hospital preferably a tertiary care centre having facilities of blood transfusion, emergency caesarean section and NICU. 'Flying Squad' service is ideal for transfer of such type of patients. An intravenous Ringer lactate or dextrose-saline drip should be started and is kept running during transport. Patient should be accompanied by two or three persons fit for donation of blood, if necessary.

Treatment at hospital:

All cases of antepartum haemorrhage, should be admitted.

The reasons are

All the cases of APH should be suspected as due to PP unless proved otherwise

The bleeding may recur sooner or later and none can predict when it recurs and how much she will bleed.

TREATMENT ON ADMISSION

- Immediate attention
- Formulation of the line of treatment

IMMEDIATE ATTENTION:

Overall assessment of the case is quickly made as regards :

- Quick assessment of general condition and amount of the blood loss— by noting the general condition, pallor, pulse rate and blood pressure, inspecting the sheets and clothes and the blood stains on the thighs and legs.
- Gentle abdominal palpation to exclude abruption and uterine contractions, and auscultation to note the foetal heart rate.
- Two large-bore IV cannulae (14G) are sited and a crystalloid or colloid drip is started after taking a blood sample for haemoglobin estimation, blood grouping and cross matching.
- Confirmation of diagnosis is made from the history, physical examination and with bed side sonographic examination to localise placenta, foetal presentation and foetal condition. An assessment of risk factors for venous thromboembolism in pregnancy should be performed.
- Further management depends on the period of gestation, maternal and foetal condition and, bleeding is present or absent

  Vaginal examination is absolutely contraindicated in PP.

FORMULATION OF THE LINE OF TREATMENT:

The definitive treatment depends upon period of gestation, foetal and maternal status and extent of the haemorrhage.

- Active (Definite) management
- Expectant management

ACUTE CARE OF BLEEDING PLACENTA PREVIA

PP with active bleeding is a potential obstetric emergency. These women should be admitted to the Labour and Delivery ward for foetal and maternal monitoring, and the anaesthesia team should be informed.

Goals -The major goals in managing a patient with bleeding PP are to:

- To maintain maternal hemodynamic stability
- emergency caesarean delivery is indicated

Maternal and foetal assessment — Maternal heart rate, blood pressure, respiratory rate, peripheral oxygen saturation, and urine output are monitored. tachycardia, hypotension, tachypnoea, low oxygen saturation, and air hunger are signs of hypovolemia.

Continuously FHR monitoring is done for patterns suggestive of hypoxemia or anaemia. Accurate vaginal blood loss estimation is difficult to determine visually, particularly when blood is partially saturating or soaking maternity pads, towels, or gauze sponges, or dripping onto the floor [126,127]

Techniques to quantify blood loss[80]

Collect blood in a graduated volumetric container.

Use visual aids that correlate the size and appearance of blood on specific surfaces (eg, maternity pad, emesis basin, bed sheet, lap sponge) with the volume of blood absorbed by that surface.

Measuring the total weight of bloody materials and then subtract the known weight of the same materials when dry. The difference in weight between dry and wet in grams approximates the volume of blood in milliliters.

Grading shock -estimated blood loss based on patients clinical signs at presentation

	Class I	Class II	Class III	Class IV
Blood loss (ml) Blood loss (%of blood volume	Up to 750 ml Up to 15%	750-1500 15-30%	1500-2000 30-40%	>2000 >40%
Pulse rate (beats/min)	<100	100-120	120-140	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (per min)	14-20	20-30	30-40	>40
Urine output (ml/hr)	>30	20-30	5-15	Negligible
Mental status	Sligtly anxious	Mildly Anxious	Anxious, Confused	Confused, Lethargic
Fluid replacement	Crystalloids	Crystalloids	Crystalloid and blood	Crystalloid and blood

Attempt to account for fluids other than blood (eg, amniotic fluid, irrigation fluid, urine) that are collected or absorbed.

Laboratory investigations —Blood sample for complete blood count, blood grouping and cross matching, Cross-match 2-4 units of PRBC when bleeding is heavy or increasing, delivery is likely for any reason, or we anticipate difficulty in procuring compatible blood.

Evaluation for coagulopathy (fibrinogen level, activated partial thromboplastin time, prothrombin time) is indicated in patients with suspected coexistent abruption or with heavy blooding resulting in hemodynamic instability Prolonged oozing from needle puncture sites also suggests coagulopathy.

Stabilization

Intravenous access and crystalloid -1 or 2 large bore intravenous lines are secured and crystalloid (Ringers lactate or normal saline) infused to achieve and maintain hemodynamic stability and adequate urine output (at least 30 mL/hour).

Transfusion — Transfusion of blood products in a woman with an actively bleeding previa should be guided by the amount blood loss over time and changes in hemodynamic parameters, as well as the haemoglobin level

In an otherwise healthy young woman acute haemorrhage may not be associated with an immediate reduction in either haematocrit or blood pressure. Thus, a low threshold for ordering a transfusion should be maintained in patients with APH once the diagnosis of PP is made. A failure to correct tachycardia or hypotension with a normal saline bolus, or documentation of a haemoglobin value less than10 g/dL should prompt immediate transfusion.

Initially transfuse 2-4 units of typed and crossed PRBC, without fresh frozen plasma or platelets as long as the fibrinogen level is more than 250 mg/dL and the platelet count is more than 100,000/microL. The goal of transfusion is to achieve a final haemoglobin level of > 10 g/dL.

If the patient fails to stabilize, a massive transfusion protocol should be initiated.

If the patient continues to bleed, then the same blood product transfusion ratios used for patients with severe haemorrhage of other aetiologies: a 1:1:1 ratio of PRBC: fresh frozen plasma: platelets.

If delivery is not imminent, continue transfusion until the bleeding is decreased, patient has stabilized and haemoglobin is at least 10 g/dL. This haemoglobin is chosen to provide a margin of safety since the patient is at increased risk for another, more severe bleeding event. However, if delivery is imminent, a preoperative or intraoperative target haemoglobin of 8 g/dL is reasonable.

Outcome — Most women who present initially with symptomatic PP respond to supportive therapy, as described above, and do not require immediate delivery.

Caesarean delivery is indicated for:

- Active labour.
- A non-reassuring foetal heart rate tracing unresponsive to resuscitative measures.
- Severe and persistent vaginal bleeding such that maternal hemodynamic stability cannot be achieved or maintained.
- Significant bleeding per vagina after 34 weeks –

Because the neonatal benefits from avoiding preterm delivery decrease with advancing gestational age, whereas maternal risks from recurrent or persistent bleeding probably increase, to balance the maternal risks versus foetal benefit, favours delivery in women with significant vaginal bleeding after 34 weeks of gestation. Delivery should not be delayed to administer antenatal corticosteroids [124.]

Admission with minor APH : Typically in this situation the vaginal bleeding settles during triage assessment of a haemodynamically stable woman. In the absence of any uterine contractility or concerns with the non-stress test, such women can be admitted to the antenatal unit. Where no formal diagnosis of placenta praevia has been made, elective high-quality transabdominal and transvaginal ultrasound should be arranged to establish this diagnosis.

Women transferred to the labour and delivery area with intermediate level APH and/or uterine contractions < 32 weeks are at greater risk of delivery in the subsequent 48 hours, and therefore co-care with anaesthesia is important. Some women merit blood transfusion, to stay ahead of blood loss. Tocolysis may be useful in this setting.[81] All women admitted in the window 24 − 32 weeks with APH should be given an intramuscular course of antenatal steroids to promote fetal lung maturation.[82] For viable deliveries < 32 weeks women should also be started on a 12-hour IV regimen of magnesium sulphate for fetal neuro-protection. [83] These two evidence-based interventions make sense in high-resource settings but may be understandably omitted where resources must be focused on survival of the mother and term new-borns.

Expectant treatment: **Macafee and Johnson regimen)**

Subsequent expectant management : If the patient is < 36 weeks' gestation, has no contractions, the bleeding has settled for 48 hours, the fetus is objectively healthy and there are no maternal co-morbidities that direct the need for delivery (e.g. preeclampsia), a period of expectant treatment is reasonable in order to gain time for fetal maturation

The aim is to continue the pregnancy for foetal maturity and if possible, till term without compromising the maternal health. Expectant treatment is meant only for achieving fetal maturity and benefits the fetus without increasing undue maternal hazards.

Vital prerequisites:

Availability of blood for transfusion whenever required

Caesarean section facilities should be available round the clock.

Selection of cases:

Suitable cases for expectant management are:

- Mother is in good health status
- Gestational age is less than 37 weeks
- Absence of active vaginal bleeding is
- Foetal wellbeing is assured by ultrasound and NST.

Conduct of expectant treatment:

- Bed rest with bathroom privileges. Should be accompanied by an attendant 24 hours.
- Investigations—like haemoglobin estimation, blood grouping and urine for protein are done.
- Inspection of the vulval pads and foetal monitoring with USG at an interval of 2–3 weeks

- Supplementary haematinics and the blood transfusion are given for blood loss. Keep blood and blood products arranged.
- After 2-3 days of stoppage of bleeding, a gentle speculum (Cusco's) examination is made to exclude local cervical and vaginal lesions for bleeding. However, their presence does not negate placenta previa
- Use of tocolytic agents like terbutaline, ritodrine, magnesium sulphate and calcium channel blockers have been tried in an attempt to arrest bleeding if associated with uterine contractions.
- Use of cervical cerclage to reduce bleeding and to prolong pregnancy is not helpful [27]
- Anti D given to all Rh negative (unsensitized) women.
- Antenatal corticosteroid therapy is indicated if the duration of pregnancy is less than 34 weeks to prevent respiratory distress syndrome in new-born.

Discharge criteria – if bleeding has stopped for a minimum of 24 hours and those who have no other pregnancy complications can be discharged. [84]

Expectant management at Hospital or at Home?

- Hospital setting is ideal. But considering the cost of prolonged hospitalization and psychological morbidity, home care may be allowed in after proper counselling.

Selected cases are —

- Patient lives close to hospital
- 24-hour transportation is available
- Bed rest assured

- Patient is well motivated to understand the risks.
- Reactive non stress test on discharge.
- Availability of 24 hr transport services between home and hospital.
- 4 Weekly clinical follow up until delivery including serial Hb% and repeat ultrasound.
- Be able to come to the hospital within 20 minutes [84].
- Be reliable (ie, will follow the instructions about sexual activity, etc).
- Understands the risks of outpatient management.
- Available adult companion 24 hours/day who can immediately transport the woman to the hospital if there is light bleeding or call an ambulance in case of severe bleeding.

Any woman being treated at home must report to the hospital immediately if she experiences any bleeding, including spotting, contractions or pain (including vague suprapubic period-like aches).

Termination of the expectant treatment:

The expectant treatment is carried up to 37 weeks of pregnancy. By this time, the baby becomes sufficiently mature.

However, preterm labour may have to be done in conditions, such as:

- Recurrence of brisk haemorrhage and which is continuing
- The foetus is dead
- The foetus is found congenitally malformed on investigation.

- Repeated small bouts of haemorrhage is not an indication for termination of expectant treatment. Replacement of the blood loss can be made by blood transfusion. However, there is the risk of IUGR.

Timing of delivery —In stable (no bleeding or minimal bleeding) placenta previa patients' caesarean delivery is planned at 36+0 to 37. Late preterm (34+0 to 36+6 weeks of gestation) delivery should be done for women presenting with PP or a low-lying placenta and a history of vaginal bleeding or other associated risk factors for preterm delivery.Delivery timing should be tailored according to antenatal symptoms.

MODE OF DELIVERY IN WOMEN WITH A LOW-LYING PLACENTA

Women presenting with a placental edge < 20 mm from the internal os in the 3rd trimester are more likely to need delivery by caesarean section when the placental edge is thicker (over 10 mm) and/or contains a sponge-like echo or marginal 'sinus'. These additional ultrasound features are poorly evidence defined, not routinely assessed in UK practice and the success rates of vaginal delivery when the placental edge is between 10 and 20 mm from the internal os vary widely (56% and 93%, respectively).

"CAESAREAN DELIVERY

Caesarean delivery without vaginal examination is treatment of choice for major degree placenta previa as the exact location of the placenta can now be determined in almost all cases with the use of ultrasound.

Indications of caesarean delivery

Major degree of PP

Minor degree of PP with excessive bleeding

Preparation — elective caesarean section for PP must be done during day time so that there are senior doctors of obstetrics available for either directly performing or supervising the procedures.

Keeping adequate blood in hand is lifesaving especially in complicated cases like placenta accrete, percreta etc, or cases of prior caesarean delivery.

Appropriate surgical instruments for performing of hysterectomy should also be available since these patients who are high risk of placenta accreta, even in the absence of a prior caesarean delivery.

"TYPE OF OPERATION

Lower segment caesarean section

Advantages

- Familial technique
- Bleeding sinuses at the placental site can be better managed under direct vision.
- Placenta adherent morbidly if found can also managed effectively"

The disadvantages are

- Dilated vessels on the anterior LUS especially in anterior PP may bleed excessively when cut
- In anterior PP, the placenta either has to be cut or separated to deliver the baby.

- Risk of severe fetal anemia due to fetal exsanguination.
- The edges of the cut margins become friable and vascular that the tissues may cut through during suturing.

"Classical caesarean section

The advantages of classical caesarean section are

operation can be done more quickly and fetus can be quickly extracted without disturbing the placenta and without exsanguination

Disadvantages

The disadvantage is that the it is difficult to control bleeding because placenta which is implanted in the LUS cannot be visualized.

Difficulties encountered during caesarean section in PP

- The LUS is not well formed before term.
- May require lower segment vertical incision
- If there is placenta accrete, hysterectomy or internal iliac artery ligation may be required
- If the placental bed bleeds excessively, apply hot packs, mattress suture or gel foam.

Precautions to be taken — While entering the uterus the operating surgeon should avoid placental disruption. If the placenta is incised, haemorrhage from foetal vessels can result in significant neonatal anaemia.

Use of preoperative and /or intraoperative ultrasonography is considered to precisely determine placental location and the optimal place for uterine incision.[27]

If placental is located anterolaterally, vertical incision is made on LUS in the opposite side from the placenta. In cases where placenta wraps around the cervix extending from anterior to posterior LUS, a transverse or vertical incision may be possible above it [101,102], although this often results in extension into the upper uterine segment. Vertical skin and/or uterine incisions is considered when it is transverse lie to avoid the placenta, particularly before 28 weeks of gestation. 85

"During incision on the uterus if the placenta is transacted, immediately clamp the umbilical cord after foetal delivery to avoid excessive foetal blood loss.

"In cases of anterior placenta praevia, cutting through the placenta is often associated with increased maternal haemorrhage. Hence avoiding incision of the anterior PP after gestational age of 24 weeks reduced the need for maternal blood transfusion during or after caesarean delivery[85]

"A 'J'-shaped uterine incision has been evaluated in women presenting with placenta praevia in a small retrospective study and shown to reduce intraoperative blood loss[86]

Routine oxytocin administration reduces the risk of postpartum haemorrhage"

Placenta previa accreta if found accidentally can also be tackled effectively by, after trimming the umbilical cord leave the placenta and close hysterotomy incision

In few cases, placenta resorbs spontaneously, few cases require hysterectomy.

For those cases in whom the placenta is in situ further advised for serial estimation serum β-hCG and USG.

Management of postpartum bleeding — After placental delivery, severe haemorrhage may occur from placenta implantation site, due to ineffective contraction and retraction of LUS.

Postpartum haemorrhage is managed in the following ways.

Medical Method

- Administration of uterotonic drugs and tranexamic acid — The dose of oxytocin can be increased as needed to control heavy bleeding.
- If oxytocin alone does not control haemorrhage, then administer tranexamic acid along with other uterotonic drugs (carboprost [prostaglandin F2 alpha] or methylergometrine

Surgical methods

"1. Uterine Artery Ligation [45]

Several surgical procedures may be helpful to arrest of obstetrical hemorrhage. Of these, the technique for unilateral or bilateral uterine artery ligation is used primarily for lacerations at the lateral part of a hysterotomy incision Uterine artery ligation- The suture goes through the lateral uterine wall anteriorly, curves around posteriorly, then re-enters anteriorly. When tied, it encompasses the uterine artery

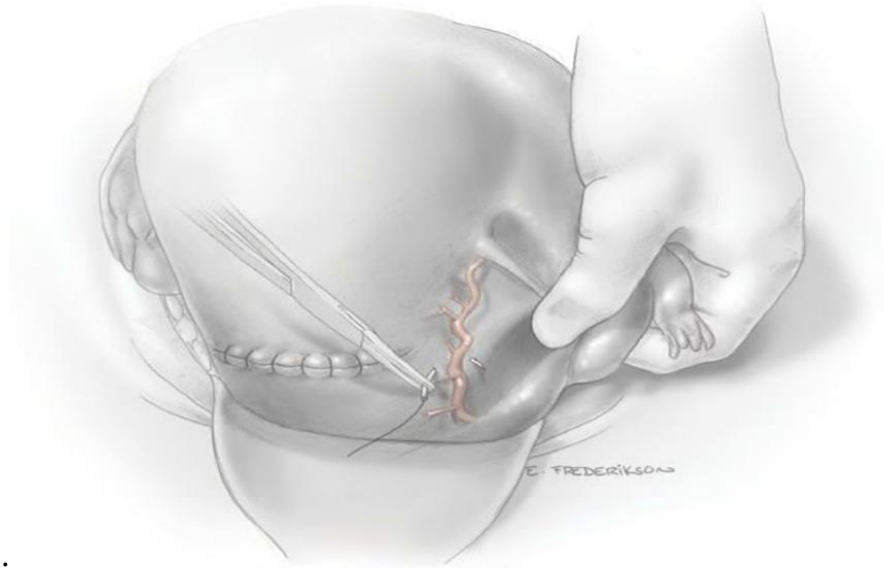

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Fig no. 5 Uterine artery ligation (photography courtesy of Williams text book of obstetrics 25th edition

Intrauterine balloon tamponade — Intrauterine balloon tamponade is a fast and often effective procedure for control of haemorrhage in patients who have not responded to drug therapy or focal placental site suture ligation. [87,50]Intrauterine balloon tamponade and/or uterine compression sutures — If bleeding persists, the next step is either intrauterine balloon tamponade or placement of uterine compression sutures.

Uterine compression sutures are more effective for uterine atonicity and fundal bleeding, whereas the balloon may be more effective for controlling lower segment bleeding. However, an advantage of placing the balloon first is that it is easy and quick procedure and if it doesn't work, the balloon is deflated, compression sutures can be placed, and if needed the balloon can be inflated. If the compression sutures are placed first, then they will have to be removed in order to place a balloon.

<u>Uterine compression sutures</u> — If balloon tamponade is ineffective in controlling bleeding, the balloon can be deflated and a B-Lynch uterine compression suture is applied.

Almost 20 years ago a surgical technique to arrest hemorrhage for severe postpartum atony was introduced by B-Lynch and coworkers (1997).[88]The procedure involves placement of a No. 2-chromic suture to compress the anterior and posterior uterine walls together. Because they give the appearance of suspenders, they are also called braces.Several modifications of the B-Lynch technique have been described (Cho, 2000(47) ,Hayman, 2002[89]; Matsubara, 2013b[90] Nelson, 2007[91]). Indications vary for its application, and this will affect the success rate. B-Lynch (2005) cited 948 cases with only seven failures. Conversely, Kayem and associates (2011) [92] described 211 women in whom compression sutures were employed. The overall failure rate of 25 percent did not differ between B-Lynch sutures and their modifications.

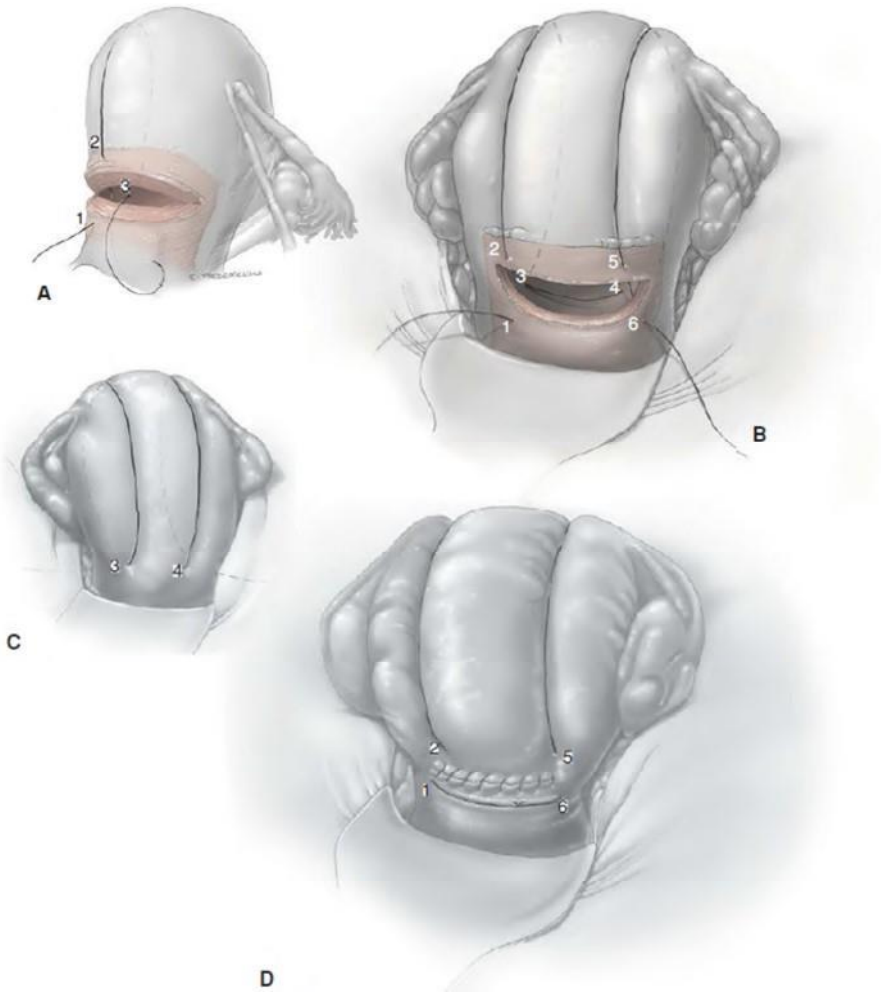


Fig no. 6 Uterine compression suture or "brace." (Photography courtesy of Williams text book of obstetrics 25th edition)

The B-Lynch suture technique is illustrated from an anterior view of the uterus in Figures A, B, and D and a posterior view in Figure C.

The numbers denote the sequential path of the suture and are shown in more than one figure.

Step 1. Beginning below the incision, the needle pierces the lower uterine segment to enter the uterine cavity.

Step 2. The needle exits the cavity above the incision. The suture then loops up and around the fundus to the posterior uterine surface.

Step 3. The needle pierces the posterior uterine wall to reenter the uterine cavity. The suture then traverses from left to right within the cavity.

Step 4. The needle exits the uterine cavity through the posterior uterine wall. From the back of the uterus, the suture loops up and around the fundus to the front of the uterus.

Step 5. The needle pierces the myometrium above the incision to re-enter the uterine cavity.

Step 6. The needle exits below the incision and the sutures at points 1 and 6 are tied below the incision. The hysterotomy incision is then closed in the usual fashion

Internal Iliac Artery Ligation

Ligation of one or both internal iliac arteries has been used for many years to reduce haemorrhage from pelvic vessels (Allahbadia, 1993[92]; Joshi, 2007[93]).

Drawbacks are that the procedure may be technically difficult and is only successful half of the time (American College of Obstetricians and Gynecologists2012b[94]).

step1. Adequate exposure is obtained by opening the peritoneum over the common iliac artery and dissecting down to the bifurcation of the external and internal iliac arteries.

step 2. Branches distal to the external iliac arteries are palpated to verify pulsations at or below the inguinal area.

Step 3. Ligation of the internal iliac artery 5 cm distal to the common iliac bifurcation will usually avoid the posterior division branches (Bleich, 2007[95])

Step 4. The areolar sheath of the artery is incised longitudinally, and a right-angle clamp is carefully passed just beneath the artery from lateral to medial. Care must be taken not to perforate contiguous large veins, especially the internal iliac vein.

Step 5. Suture—usually nonabsorbable—is passed under the artery with a clamp, and the vessel is then securely ligated.

Mechanism: Following ligation, pulsations in and distal to the external iliac artery are again confirmed. The mechanism of action with internal iliac artery ligation is an 85-percent reduction in pulse pressure in those arteries distal to the ligation (Burchell, 1968[86]). This converts an arterial pressure system into one with pressures approaching those in the venous circulation. This creates vessels more amenable to haemostasis via pressure and clot formation.

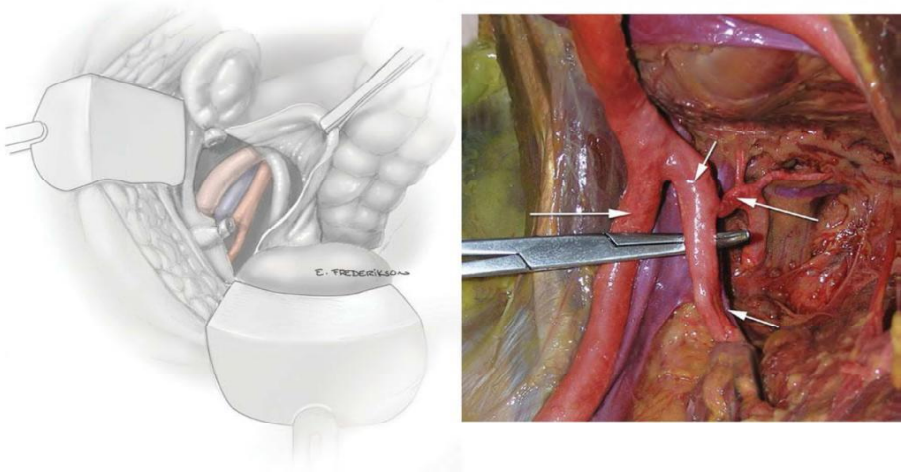


Fig no.7 Ligation of the right internal iliac artery (Photography courtesy of Williams text book of obstetrics 25th edition)

"A .The peritoneum covering the right iliac vessels is opened and reflected.

B .Unembalmed cadaveric dissection shows the right-angle clamp passing underneath the anterior division of the intenal iliac artery just distal to its posterior division

In refractory cases

Pelvic vessel embolization

Consider arterial embolization — When the above measures have failed, uterine or hypogastric artery embolization in an operation theatre with the full surgical team in attendance is an option if the facility has a hybrid operating room, or an operating room that allows simultaneous surgery and embolization (an appropriately sensitive portable C-arm and carbon fibre table).

Hysterectomy for placenta previa, placenta accrete

Hysterectomy is a definitive treatment of uterine bleeding when fertility preserving procedures have not reduced the bleeding to a manageable level. Ideally, it should be performed before severe hypovolemia, tissue hypoxia, hypothermia, electrolyte abnormalities, and acidosis have developed, which further compromise the patient's status.

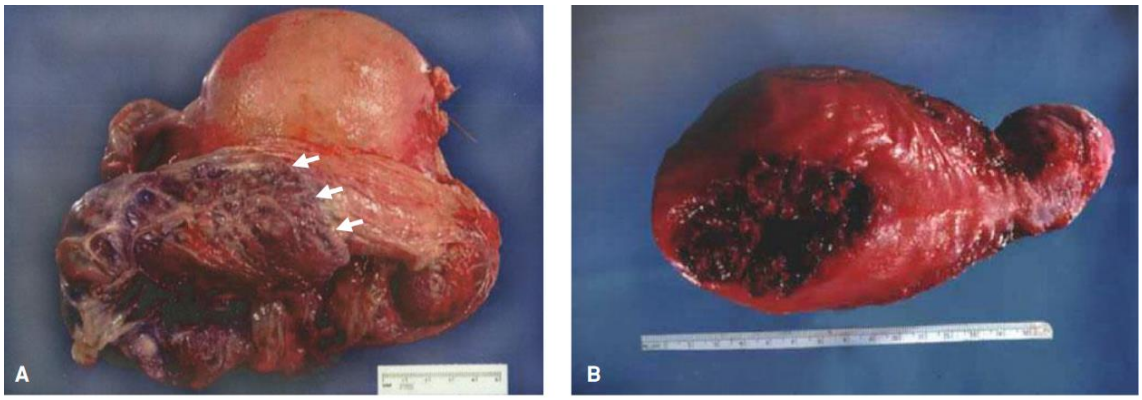


Fig no.8 Photographs of accrete syndrome hysterectomy specimen Caesarean hysterectomy specimen containing a total placenta previa with percreta involving the lower uterine segment and cervical canal white arrows show the invading line of the placenta through the myometrium. (Photography courtesy of Williams text book of obstetrics 24th edition) Hysterectomy specimen containing a partial placenta previa with placenta percreta that invaded the lateral fundal region to cause hemoperitoneum.

MATERNAL OUTCOME

MATERNAL MORBIDITY AND MORTALITY

Obstetric haemorrhage is the most common cause of maternal deaths In developing countries like India. Even though APH is less common when compared to PPH, it contributes to a significant number of cases. Maternal mortality rates have improved with regard to PP because of

- Improvement in the health status of the pregnant women by antenatal care.
- Early diagnosis of PP by Ultra Sound Scan.
- Availability of blood and blood products transfusion facilities.

- Avoiding digital vaginal examination in case of accidental placental haemorrhage.
- Increasing number of caesarean deliveries.

In spite of above measures mortality and morbidity in PP is high because of the following factors.

1. Shock

Shock can be in antepartum, intrapartum and postpartum period.

Antepartum: In majority of cases the first bout of bleeding is severe but torrential haemorrhage can be easily provoked following internal examination. in such situation patients who are anaemic is placed at an even greater risk.

Intrapartum: In minor degree placenta previa cases allowed for trial of vaginal delivery all measures to be taken to control the bleeding during delivery.

Postpartum haemorrhage:

Post-partum haemorrhage is particularly likely to occur in these cases for several reasons.

- Placental site is in the lower segment of the uterus which does not retract well
- Placenta is usually larger and thinner and, as a result, part of the placenta may be retained
- Morbid adherence too, is not uncommon
- Placental site is generally larger, there is a greater area for which bleeding can occur hence fast and accurate uterine incision should be undertaken.

2. Puerperium

Attention to asepsis and antisepsis must be meticulous, because the patient in her exsanguinated state and a large placental surface is more vulnerable for puerperal sepsis.

Maternal risk

Maternal deaths due to bleeding has reduced due to introduction of conservation management which includes hemodynamic support and also expectant management, but still there are associated risks such as

- Complications related to anaesthetic and surgery especially in major previa cases undergoing emergency Caesarean delivered due to suboptimal preparation for surgery.
- PPH and postpartum sepsis.
- Due Placenta previa accreta.
- Battledore insertion and velamentous distribution of vessels are common features.
- Air embolism risk due to open placenta sinuses.

Foetal risk

- In placenta previa cases perinatal mortality and morbidity is high because of increase in preterm labour.
- Placenta previa is associated with 2.5% risk of perinatal mortality compared to 0.75% with normal singleton pregnancy and this was explained by gestational age at the time of delivery, associated congenital anomalies and maternal age.

- Respiratory distress syndrome was frequently associated with an odds ratio of 4.9%.
- Incidence of serious congenital malformation is increased in placental previa cases almost 1.6-fold.
- Malpresentation, Umbilical cord prolapse, foetal anaemia, unexplained intra uterine death due to rupture of vasa previa or from severe maternal hypovolemic shock.

Hence early detection of placental previa and careful evaluation with timely delivery and intervention is needed to decreases maternal end perinatal complication.

FOETAL OUTCOME

PERINATAL MORTALITY AND MORBIDITY

Perinatal mortality ranges from 7-25% and is three times higher than general population n placenta previa cases neonatal and perinatal morbidity and mortality is due to.

The causes of death are as follows

- Prematurity
- Birth asphyxia
- Congenital malformations
- Cord accidents
- Maternal hypovolemia and shock.

• Intrauterine asphyxia

Asphyxia can result from placental separation irrespective of the cause. Maternal hypotension due to the haemorrhage or shock can also lead to foetal hypoxia/anoxia".

• Respiratory distress syndrome (RDS)

RDS associated with prematurity resulting from previa.

• Congenital malformations

Congenital malformations like spina bifida, hydrocephalus and anencephaly, respiratory, gastrointestinal, Cardiovascular abnormalities seen commonly.

• Prematurity

Due to spontaneous or iatrogenic deliveries before 37 completed weeks of gestation or a new-born weighing <2500 gms at birth."

METHODOLOGY

METHODOLOGY

Materials and Methods-

1 Source of data: Pregnant women diagnosed with placenta previa who visited outpatient department and admitted in labour ward in R L Jalappa hospital attached to Devaraj Urs Medical College and Research Centre Tamaka, Kolar during the period of study.

2 Study design: A cross sectional observational study

3 Study period: JUNE 2017 - MAY 2019

4 Method of collection of data

A cross sectional observational study, conducted in the Department of Obstetrics and Gynaecology at R.L .Jalappa Hospital and Research Centre, Tamaka Kolar attached to Devarj urs medical college from June 2017 to May 2019. All antenatal cases (booked, un booked or referred) with gestational age >/= 28 diagnosed with placenta previa were later observed for risk factors, outcome of pregnancy, maternal morbidity, mortality and foetal outcome.

Inclusion Criteria:

- All pregnant women diagnosed with placenta previa during the study period.
- Asymptomatic women diagnosed during caesarean section
- Gestational age >28 weeks.
- Patients who had given written and informed consent for the study.

Exclusion Criteria

1 Other causes of antepartum haemorrhage. (Abruptio placenta/ local lesions of cervix, vagina, external genitalia).

2 Patients who are not willing to participate in the study.

Sample size

Sample size estimated based on proportion of PPH i,e 16.1% reported in placenta previa cases in a study' Fetomaternal outcome in placenta previa a retrospective study in teaching hospital, with 95% confidence interval with 10% Absolute error, the sample size is 51.

$$n = \frac{Z^2_{1-\alpha} P(1-P)}{d^2}$$

P-Expected proportion

d-absolute error

$Z_{1-\alpha}$ -- Standard normal deviate at 95%=1.96

Methodology for data collection:

After obtaining clearance and approval from the Institutional Ethical committee, all pregnant women who visited outpatient department & labour ward with painless vaginal bleeding to RLJH hospital from June 2017 to May 2019 was admitted. Detailed history was taken, general physical examination and obstetric examination

was done. Diagnosis was confirmed by ultrasonography. At the time of enrolment, a written informed consent was obtained from the pregnant women.

Blood samples was collected for complete blood count, Blood group and cross matching, VDRL, HIV, HbsAg

Each pregnant woman was followed up until delivery and the maternal and foetal outcome was recorded, after applying inclusion and exclusion criteria.

Data collected include-

Data was collected on patient age ,Parity ,Gestational age ,Clinical features at presentation .Detailed history of current pregnancy and previous pregnancies, history of warning bleeding. Past history of abortions, curettage, any surgeries involving uterus. Patient was subjected to detailed clinical examination. Duration of hospitalization, period of gestation at delivery, types of placenta previa, mode of delivery noted. Need for extra interventions during operative delivery to control bleeding, need for ICU admissions noted, need for blood transfusions, mortality was noted. B

Birth weight, respiratory distress, prematurity, IUGR, need for NICU admission, congenital anomalies, septicaemia death were noted.

Methodology for data analysis:

STATISTICAL METHODS:

Post op complications, Intra op complications, Respiratory distress were considered as primary outcome variables. Type of placenta previa (Minor degree Vs. Major

degree) was considered as Primary explanatory variable. Age, socio economic status etc., were considered as Secondary explanatory variables.

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Non normally distributed quantitative variables were summarized by median and interquartile range (IQR). Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

All Quantitative variables were checked for normal distribution within each category of explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as normal distribution.

Categorical outcomes were compared between study groups using Chi square test.

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.(1)

RESULTS

RESULTS.

Table no 1: Incidence of placenta previa

Total number of deliveries from June 2017-May 2019	**4820**
Total number of Placenta previa cases	**76**
Incidence	**1.57%**

In the study period total number of deliveries were 4820, among them diagnosed cases of placenta previa were 76. Accordingly, the incidence of placenta previa in the study period was 1.57%. (Table no 1)

Table no 2: Descriptive analysis of age in the study subjects (n=76)

Age (Yrs)	**Number=76**	**Percentage**
20-25	46	60.5
26-30	20	26.3
31-35	7	9.2
>35	3	3.9

Among the 76 study subjects studied, 46 (60.5%) were aged between 20 to 25 years, 20 (26.3%) were aged between 26 to 30 years, 7 (9.2%) were aged between 30 to 35 years and 3 (3.9%) were aged more than 35 years. (Table 2 & Figure no 9)

Figure no 9: Bar chart of age in the study subjects (n=76)

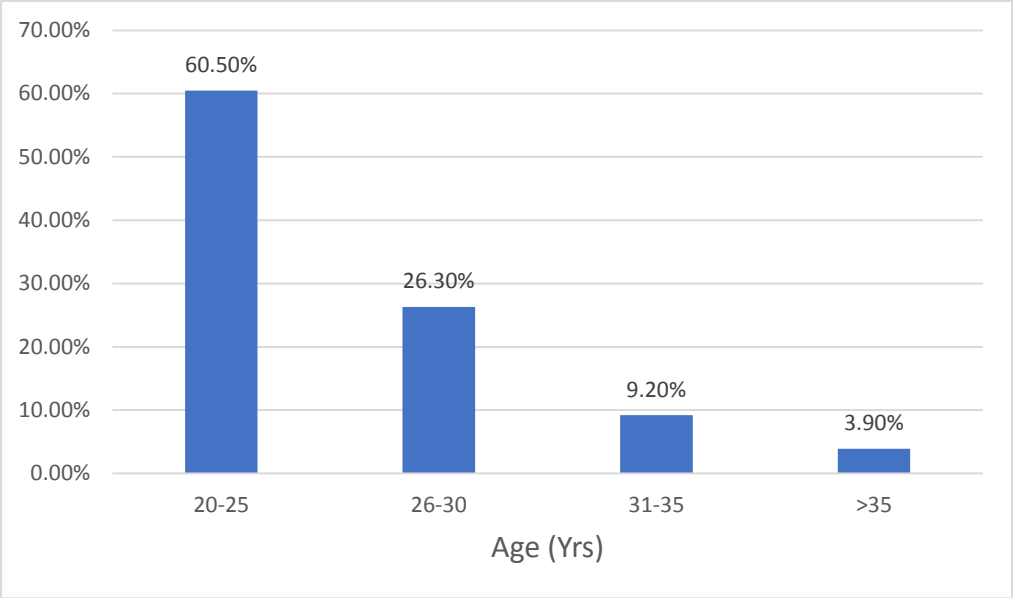


Table no 3: Study of socio-economic status in the study subjects (n=76)

Socio economic status	**Number=76**	**Percentage**
Upper income class	20	26.3
Upper Middle class	13	17.1
Middle class	30	39.5
Lower middle class	13	17.1

Among the 76 study subjects studied, the socio-economic status was, 20 (26.3%) had upper income class,13 (17.1%) had upper middle class, 30 (39.5%) had middle class and 13(17.1%) had lower middle class. (Table no 3 & Figure no 10)

Figure no 10: Pie chart of socio-economic status in the study population (n=76)

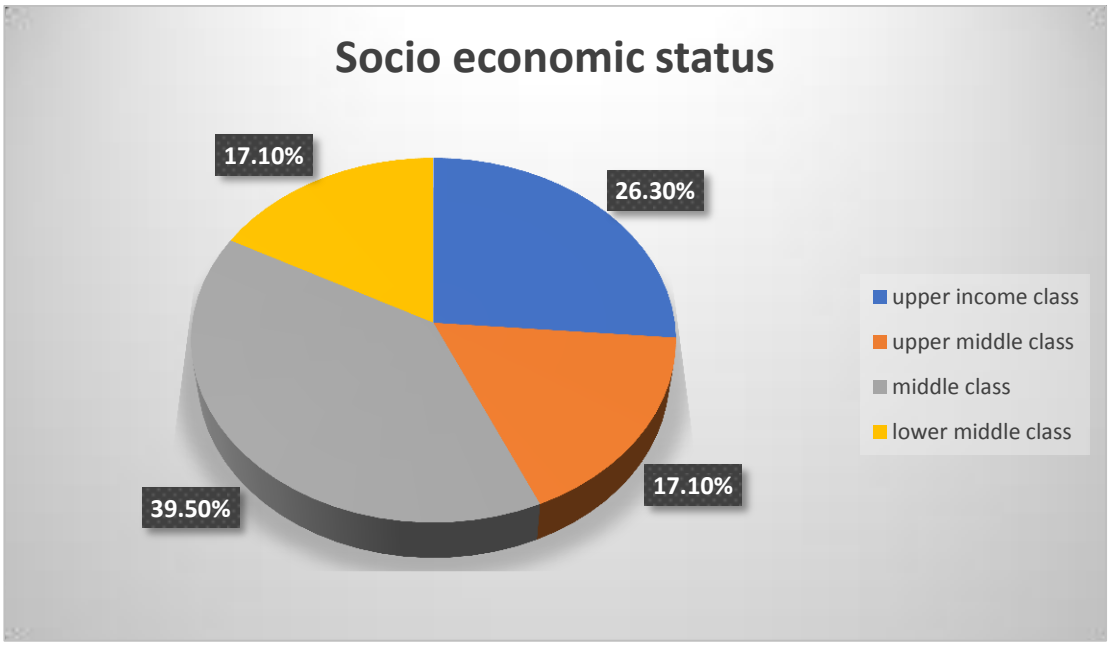


Table no 4: Study of parity distribution in the study subjects (n=76)

Parity	**Number=76**	**Percentage**
Primigravida	18	23.7
Multi Gravida	58	76.3

Among the 76 study subjects studied, 18 (23.7%) were with primigravida and 58 (76.3%) were with multi gravida. (Table 4 & Figure no 11)

Figure no 11: Pie chart of parity in the study subjects (n=76)

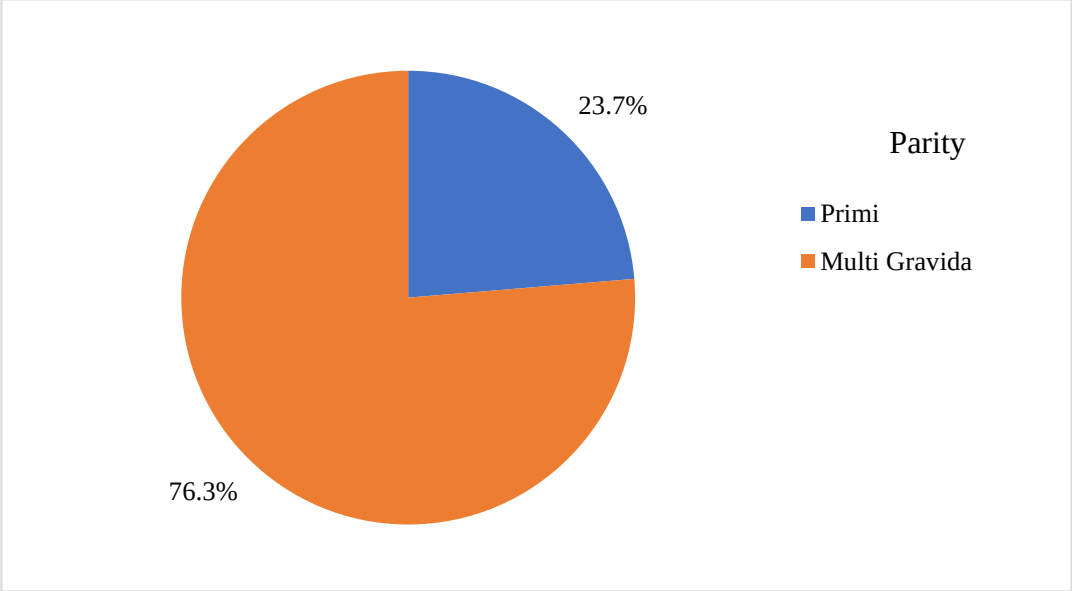


Table no 5: Study of gestational age at diagnosis (n=76)

Gestational age at diagnosis (weeks)	**Number=76**	**Percentage**
28-32	19	25
32+1-36	47	61.9
36+1-40	7	9.2
>40	3	3.9

Among the 76 study subjects, 19 (25%) subjects period of gestation at diagnosis ranged between 28 to 32 weeks, 47 (61.9%) subjects period of gestation at diagnosis was between 32+1 to 36 weeks, 7(9.2%) subjects period of gestation at diagnosis was between 36+1-40 weeks and 3 (3.9%) subjects period of gestation at diagnosis was more than 40 weeks. (Table 5 & Figure no 12)

Figure no 12: Bar chart of period of gestation at diagnosis in the study subjects (n=76)

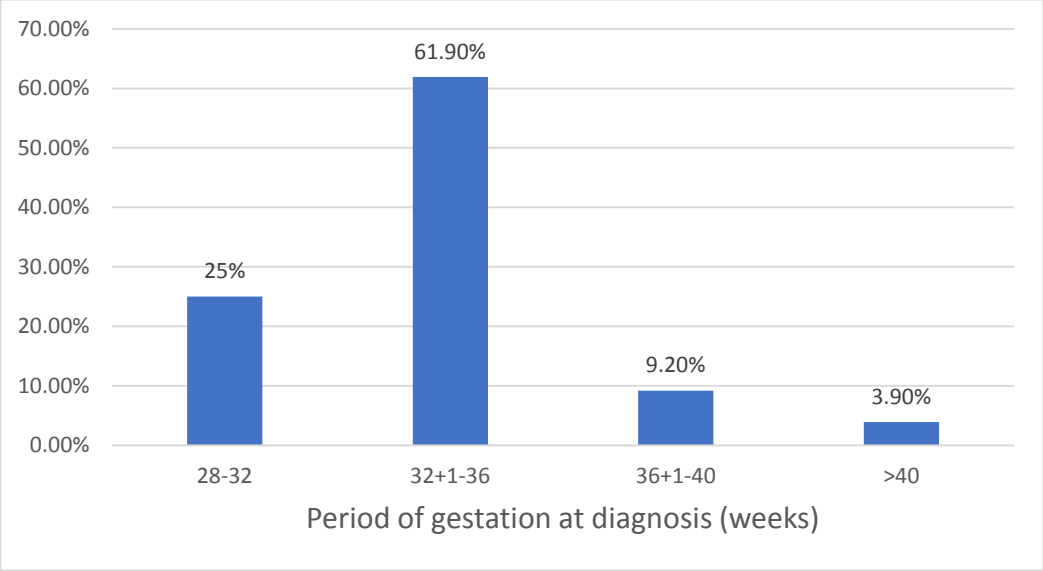


Table no 6: Study of booking status in the study subjects (n=76)

Booking status	**Number=76**	**Percentage**
Booked	50	65.8
Un booked	26	34.2

Among the study population, 50 (65.8%) were booked and 26 (34.2%) were un booked. (Table 6 & Figure no 13)

Figure no 13: Bar chart of booking status in the study population (n=76)

Table no 7: Study of risk factor in the study subjects (n=76)

Risk Factor	**Number =76**	**Percentage**
Abortion	9	11.8
Dilatation and curettage	6	7.8
Previous caesarean section	19	25
Multi parity	19	25
Previous history of placenta previa	1	1.3
Nil	33	43.2

Among the 76 study subjects, 9(11.8%) had history of abortions, 6(7.8%) had history of D and C, 19 (25%)had history of previous caesarean section among them 3 subjects had history of previous 2 LSCS,19(25%) subjects were multiparous and 1(1.32%) case had previous history of placenta previa and 43.2% did not have any risk factors.(Table no 7)

Table no 8: Study of clinical presentation in study subjects (n=76)

Symptoms	Number=76	Percentage
Bleeding per vagina	41	53.9
Pain abdomen	10	13.1
Intra uterine foetal demise	2	2.6
Leaking per vagina	1	1.3
Asymptomatic	26	34.2

In the present study 41(53.9 %) subjects, presented with bleeding per vagina followed by pain abdomen in 10 (13.1%) among them 3 cases had abruptio placenta and other 7 came in labour, intra uterine foetal demise seen in 2 (2.6%) subjects and leaking per vagina 1.3%. Most of the study subjects were asymptomatic accounting for 34.2%. Among 41 cases who presented with bleeding 6 cases had severe bleeding per vagina. (Table 8 & Figure no 14).

Figure no 14. Pie chart showing presenting symptoms among study subjects

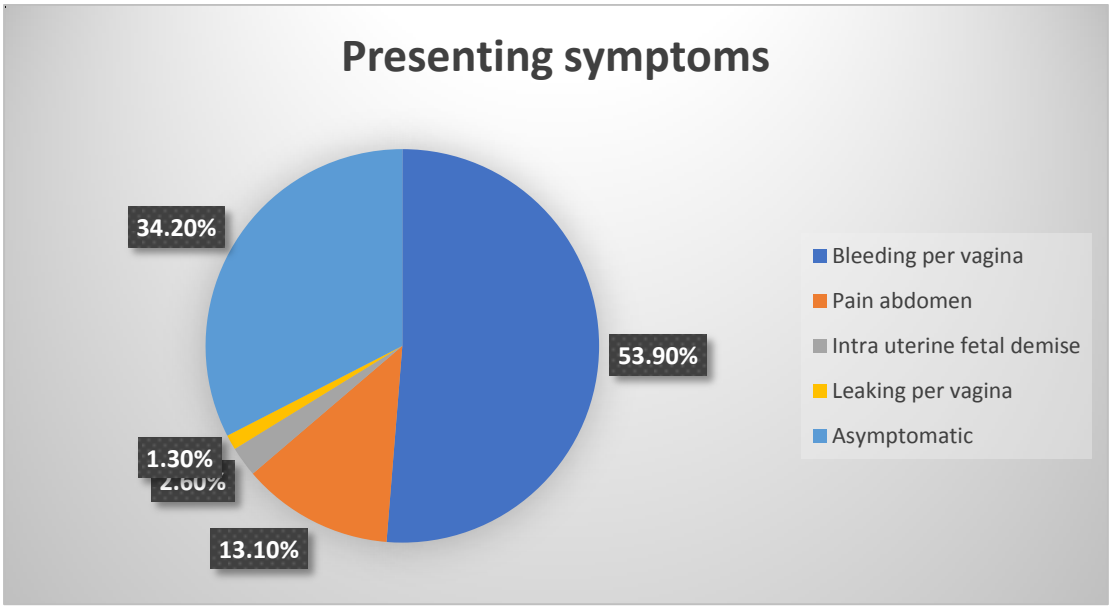


Table no 9: Study of foetal presentation in the study subjects (n=76)

Presentation	Number=76	Percentage
Cephalic	60	78.9
Breech	10	13.2
Transverse lie	6	7.9

Among the 76 study subjects, 60 (78.9%) were with cephalic, 10 (13.2%) were with breech and 6 (7.9%) were with transverse lie. (Table 9 & Figure no 15)

Figure no 15: **Bar chart of foetal presentation**

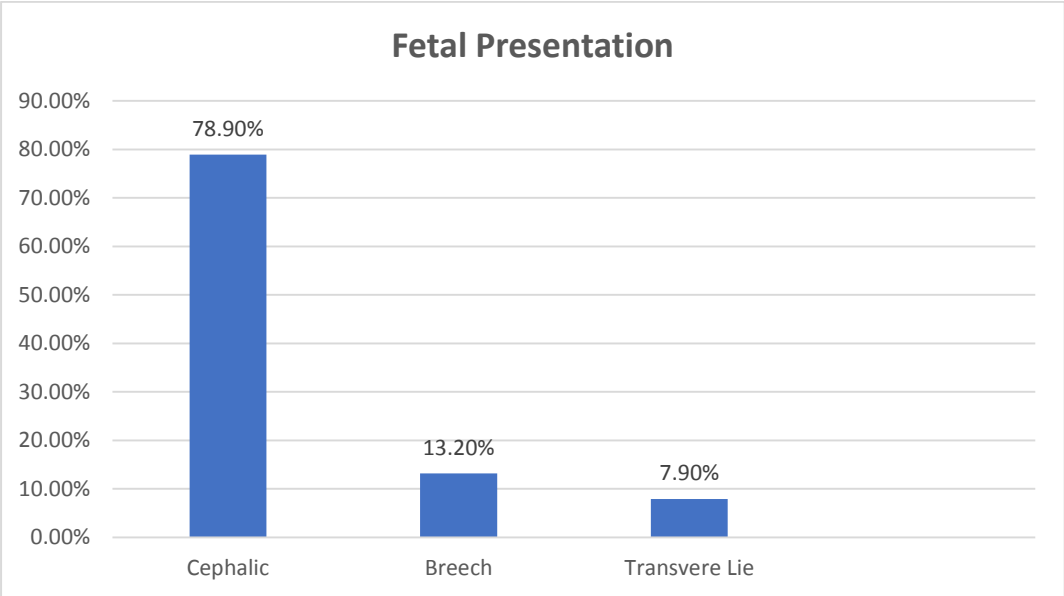


Table no 10: Study of associated complications with placenta previa

Complications	Number=76	Percentage
2nd trimester bleeding	41	53.9
Severe anaemia	11	14.4
Pre-eclampsia	8	10.5
Abruption with placenta previa	3	3.9
Nil	26	34.2

In the present study the associated complications with placenta previa were 53.9% presented with 2nd trimester bleeding, 14.4% had severe anaemia, 10.5% had Pre-eclampsia, 3.9% had abruption with placenta previa and 34.2% didn't have any complications. (Table 10 & Figure no 16)

Figure no 16: Bar chart showing associated complications in the present study

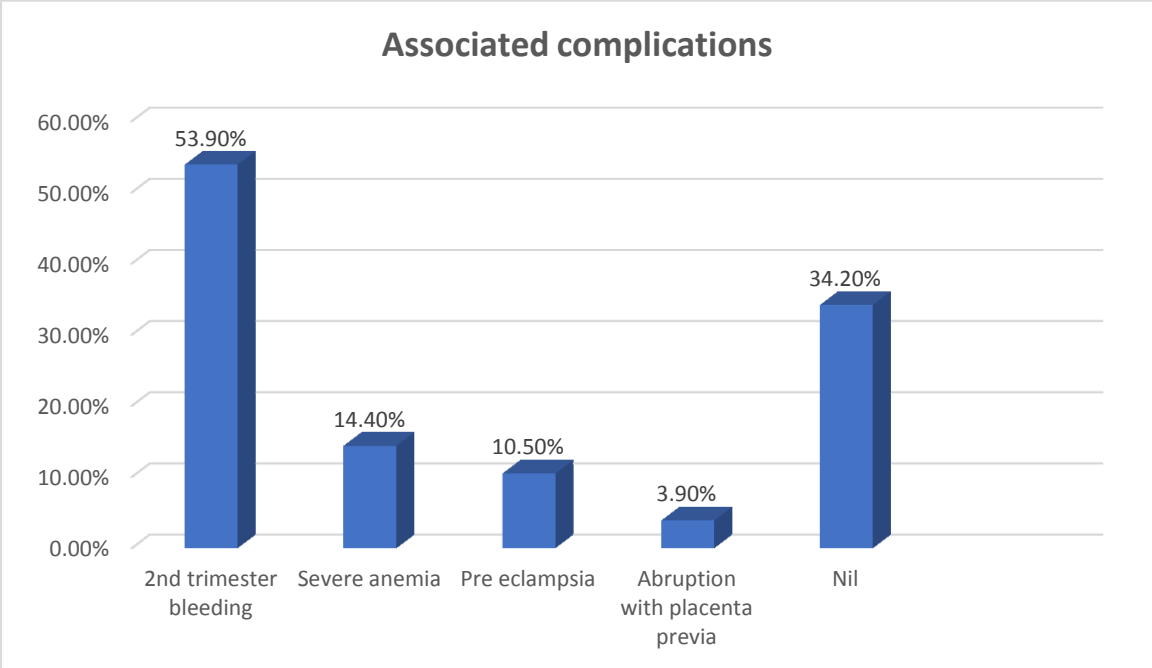


Table no 11: Ultrasound diagnosis (n=76)

USG diagnosis	Number=76	Percentage
Type I	15	19.7
Type II	17	22.4
Type III	18	23.7
Type IV	26	34.2

Among the 76 study subjects, 15 (19.7%) were with type I, 17 (22.4%) were with type II, 18 (23.7%) were with type III and 26 (34.2%) were with type IV placenta previa. (Table 11 & Figure no 17)

In the present study based on the ultra sound findings the classification of placenta previa was done as follows: Minor degree- includes Type 1 and Type 2 anterior placenta previa = 32 cases

Major degree- includes Type 2 posterior, Type 3, Type 4 Placenta previa = 44 cases

Figure no 17: Pie chart of ultrasonography diagnosis (n=76)

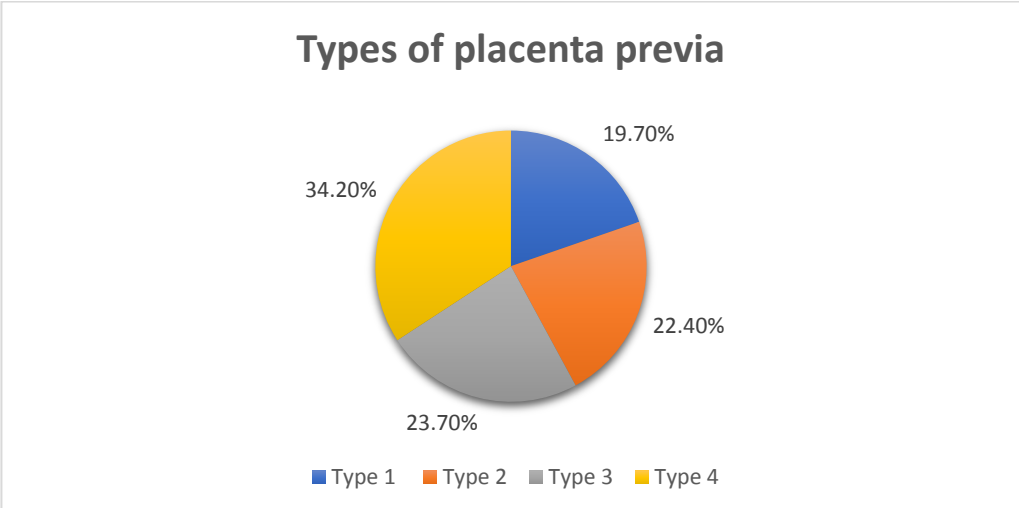


Table no 12: Association of placenta accreta in the study subjects (n=76)

MRI diagnosis	Frequency	Percentages
Placenta accreta	2	2.6%
Not needed	74	97.4%

In the present study, out of 76 cases MRI was done for 2 cases and diagnosed with placenta accrete. (Table no 12)

Table no 13: Study of management protocol (N=76)

Management protocol	Number=76	Percentage
Expectant	16	21.1
Active	60	78.9

In the present study 61(78.9%) of cases managed with active management and 16 (21.1%) were treated conservatively according to Macafee and Johnson's regimen. In the present study according Macafee and Johnson,s regimen pregnancy was prolonged to maximum of 7 weeks and minimum of 4 weeks. (Table No 13 & Figure no 18)

Figure no 18 : Pie chart of management protocol in the study subjects (n=76)

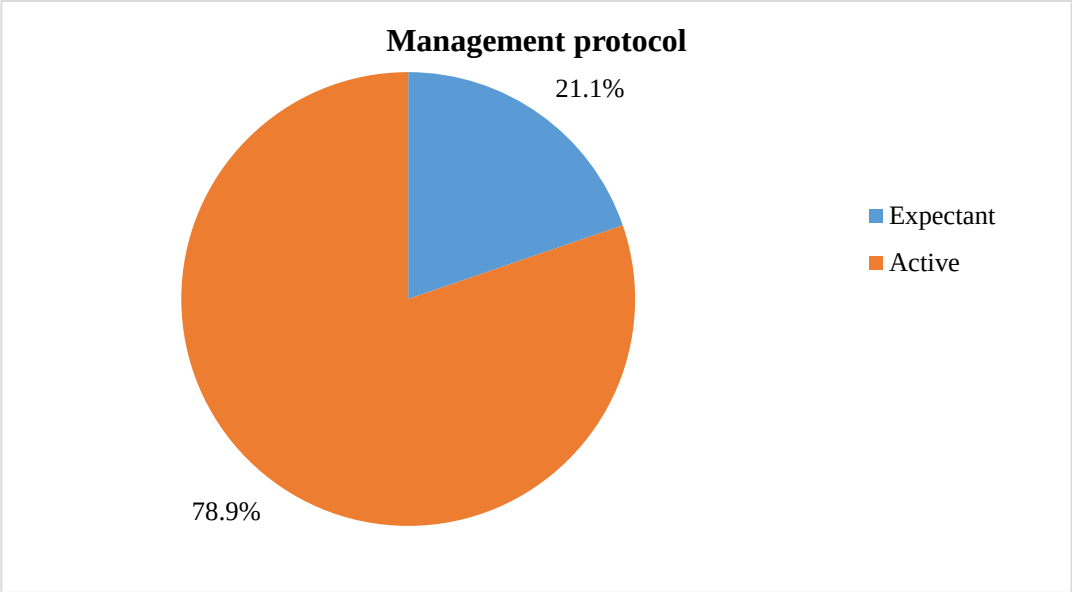


Table no 14: Period of gestation at delivery (n=76)

Period of gestation at delivery (weeks)	**Number=76**	**Percentage**
28-32	14	18.4
32+1-36	19	25.0
36+1-40	39	51.3
>40	4	5.3

In the present study 51.3% delivered at 36+1-40 weeks, 25% delivered at 32+1-36 weeks 18.4% delivered at 28-32weeks 5.3% delivered at more than 40weeks. (Table No 14, Figure no 19)

Figure no 19: Bar chart of period of gestation at delivery in the study subjects (n= 76)

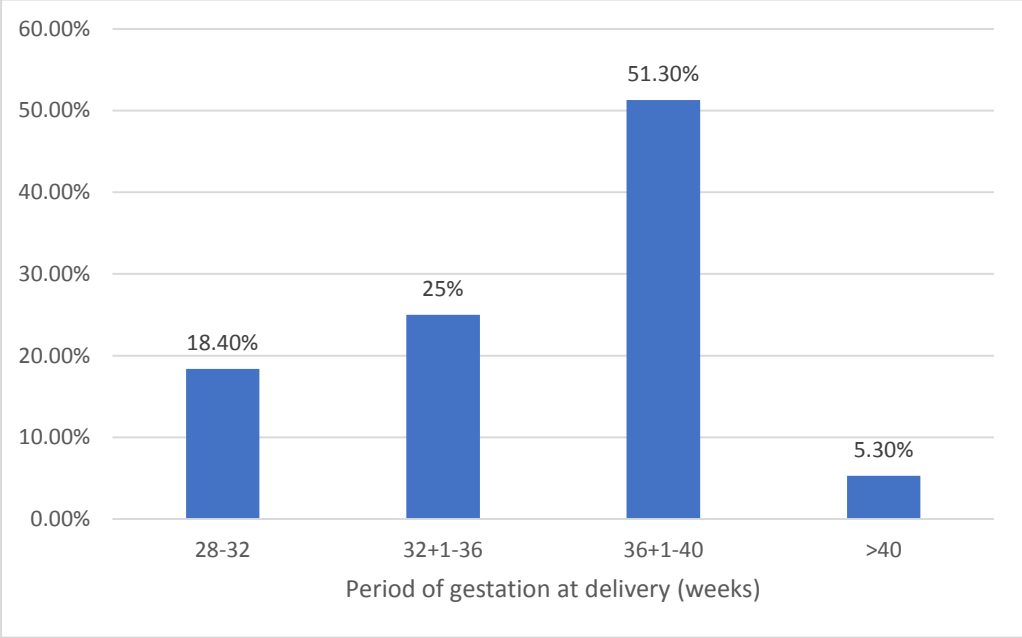


Table no 15: Study of mode of delivery (N=76)

Mode of delivery	Number=76	Percentage
Caesarean	**72**	**94.7**
Vaginal delivery	**4**	**5.3**

In the present study out of 76 cases studied, 4(5.3%) cases had vaginal delivery among them 3 cases were type 1 and 1 case was type 2A Among 72 cases who underwent caesarean delivery, 41 cases had Elective LSCS,31 cases underwent emergency LSCS. (Table No 15, Figure no 20)

Figure no 20: Pie chart showing mode of delivery N=76

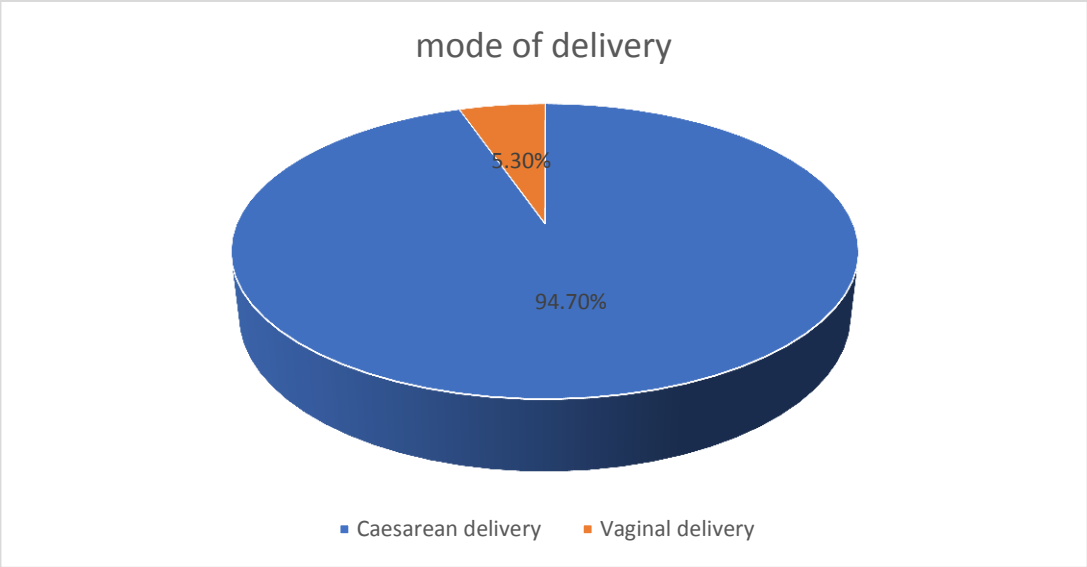


Table no 16: Study of intra operative complications in the study subjects (n=76)

Intra operative Complications	**Number=76**	**Percentage**
Haemorrhagic Shock	6	7.89
Atonic PPH	24	31.5
Abruptio placenta	3	3.9
Placenta accrete	2	2.6
Nil	44	57.9

In the present study out of 76 study subjects 57.9% had no intra- operative complications, 31.5% had PPH, 7.89% had haemorrhagic shock ,3.9% had abruptio placenta and placenta accreta was seen in 2.6%. (Table No 16)

Table no 17: Comparison of intra operative complications between types of placenta previa (N=76)

Intra operative Complications	Types Of Placenta Previa (N=76)	
	Minor Degree (N=32)	**Major Degree (N=44)**
PPH	13(40.6%)	11(25%)
Abruptio placenta	3(9.3%)	0 (0%)
Placenta Accreta	0 (0%)	2 (4.55%)
Hemorrhagic Shock	3 (9.3%)	3(6.8%)
Nil	15 (46.88%)	29 (65.91%)

No statistical test was applied- due to 0 subjects in the cells

In minor degree of placenta previa, 40.6% had PPH, 9.3% had abruption placenta, shock being 9.3% and 46.8% didn't have any complications. Minor degree placenta previa had more complications compared to major placenta previa due to associated abruptio placenta.

In major degree of placenta previa, 11 cases (25%) had post-partum haemorrhage, 3 case (6.8%) had shock and 2 cases (4.55%) had placenta accrete (Table No 17)

Table no 18: Surgical interventions to control PPH in the study subjects (N=24)

Surgical Interventions	Number=24	Percentage
Internal iliac artery ligation	**1**	**4.1**
Bilateral uterine artery ligation + internal iliac artery ligation	**1**	**4.1**
Hysterectomy	**2**	**8.3**

In the present study among the76 subjects studied, 24 cases had intra-operative complications like PPH. The above techniques were followed after failure of medical management. 1(4.1%) case required bilateral uterine artery ligation followed by internal iliac artery ligation and 1(4.1%) case required only internal iliac artery ligation. 2(8.3%) cases of placenta accrete underwent peripartum hysterectomy (2.63%). (Table no 18)

Table no 19: Comparison of Types of placenta previa requiring surgical intervention to control PPH

Surgical Interventions	Types of Placenta Previa(N=24)	
	Minor Degree (N=13)	Major Degree (N=11)
Internal Iliac Artery Ligation	0 (0%)	1 (9.1%)
Bilateral uterine artery ligation+ Internal Iliac Artery Ligation	0 (0%)	1 (9.1%)
Hysterectomy	0(0%)	2 (18.2%)
Not Required	13(100%)	7(63.6%)

No statistical test was applied- due to 0 subjects in the cells

In the present study among 32 minor degree of placenta previa 13 cases had PPH , all cases managed medically.In the major degree of placenta previa out of 44 cases , 11 case had PPH 1(9.1%) case required internal iliac artery ligation, 1(9.1%) case required bilateral uterine artery ligation followed by internal iliac artery ligation and 2(18.2%) cases underwent Peripartum hysterectomy for placenta accrete. (Table No 19)

Table no 20: Study of post-operative complications in the study subjects (N=76)

Post-operative complications	Number=76	Percentage
Haemorrhagic shock	4	5.2
DIC	1	1.3
Nil	72	93.5

In the present study out of 76 cases, 71 cases had uneventful post-operative period (93.4%). 4 cases (5.2%) had haemorrhagic shock, 1 case had Disseminated intravascular coagulation accounting for 1.3%. (Table No 20, Figure no 21)

Figure no 21: Bar chart of Post-operative complications in the study population (n=76)

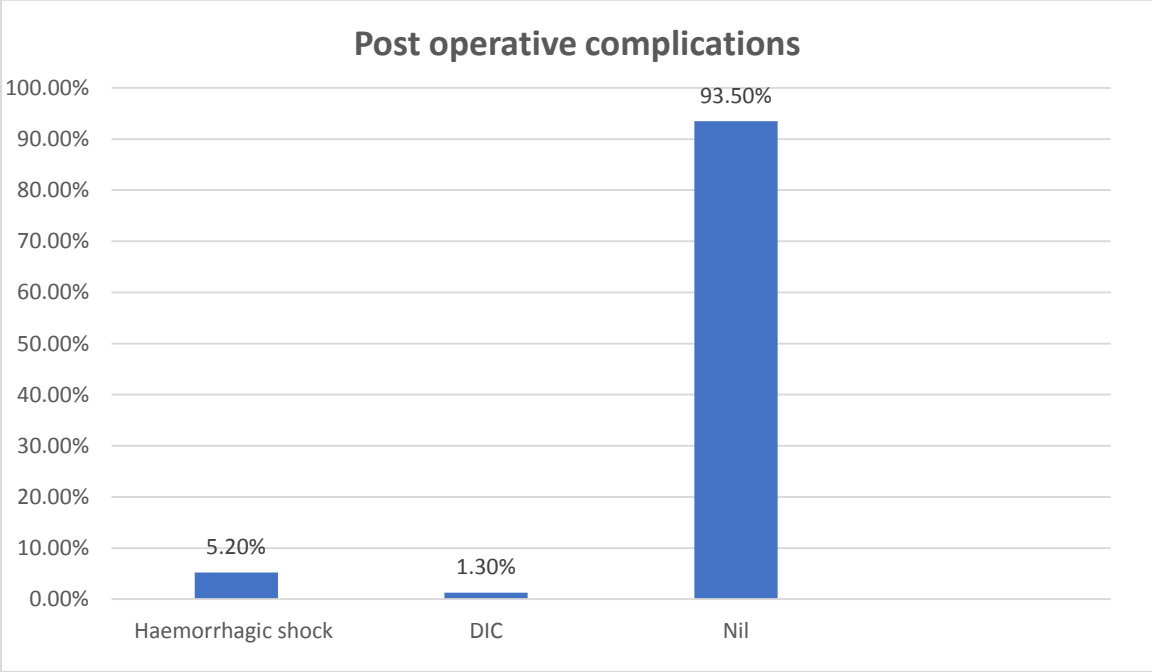


Table no 21: Comparison of Types of Placenta Previa and postoperative complications.

Post-operative Complications	Types of Placenta Previa (n=76)	
	Minor Degree (N=32)	Major Degree (N=44)
Hemorrhagic shock	0 (0%)	4 (9.1%)
DIC	0 (0%)	1 (2.3%)
Nil	32(100%)	39(88.6%)

*No statistical test was applied- due to 0 subjects in the cells

In the present study among 44 cases of major degree placenta previa ,9.1% had haemorrhagic shock in the post-operative period, 1 (2.3%) case had Disseminated intravascular coagulation due to PPH and other 39 cases didn't have any complications (88.6%). Among 32 cases of minor degree placenta previa post operative complications were nil. (Table no 21)

Table no 22: Study of blood transfusion requirement in the study subjects (n=76)

Blood Transfusion requirement	Number=76	Percentage
PRBC	40	52.6
FFP	10	13.1

Among 76 study subjects studied, 40 (52.6%) cases required packed red blood cell transfusion, 10(13.1%) cases required packed cells with fresh frozen plasma transfusion. (Table No 22)

Table no 23: Study of birth weight in the study subjects (n=76)

Birth weight (kg)	**Number=76**	**Percentage**
<1	**4**	**5.3**
1-1.49	8	10.5
1.5-2.49	30	39.5
2.5-3	27	35.5
>3	7	9.2

In the present study out of 76 new-born ,7 babies birth weight was >3 kg accounting for 9.2%, 27 babies (35.5%) had birth weight of 2.5kg-3kg, 30 babies (39.5%) had birth weight in between 1.5-2.49 kg, 8 cases had weight in between 1-1.49 kg which accounts for 10.5%, 4(5.3%) case had birth weight <1kg. (Table No 20, Figure no 22)

Figure no 22: Bar chart of Birth weight in the study subjects (n=76)

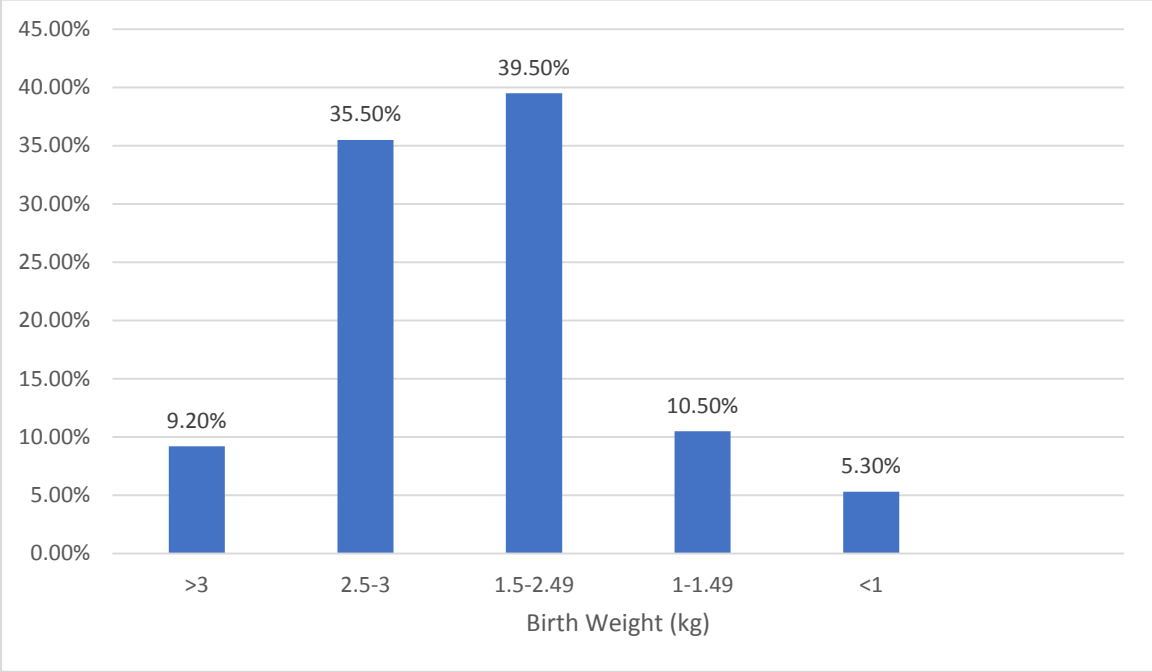


Table no 24: Study of perinatal mortality in the study subjects (n=76)

Perinatal mortality	Number=76	Percentage
Yes	15	19.7
No	61	80.3

In our present study out of 76 cases, 15 cases had perinatal mortality accounting for 19.7%, cause being Respiratory distress, Extreme prematurity and very low birth weight. (Table No 24, Figure no 22)

Figure no 23: Bar chart of perinatal death in the study subjects

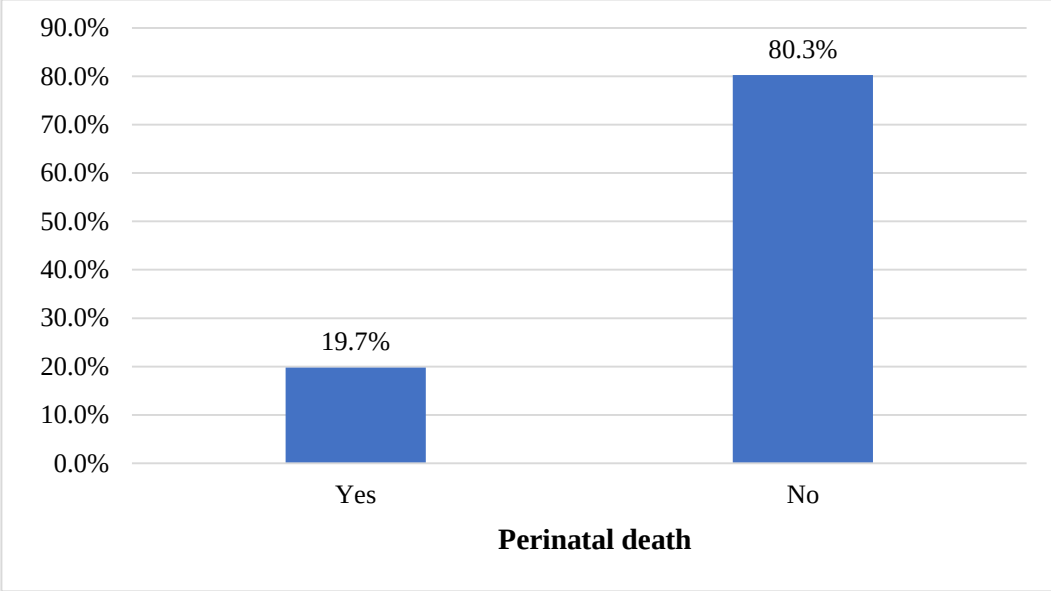


Table no 25. Study of NICU admission in the live born foetuses (n=66)

NICU Admission	Number=66	Percentage
Yes	**32**	**48.5**
No	**34**	**51.5**

*Not applicable for 10 patients

Among the study population, 32 (48.5%) were admitted in NICU. (Table 25)

Table no 26: Comparison of NICU admission between types of placenta previa (n=76)

NICU Admission	Types of Placenta Previa		Chi square	P value
	Minor Degree (Live-born N=27)	Major Degree (Live-born N=39)		
Yes	12 (44.44%)	20 (51.28%)	0.299	0.585
No	15 (55.56%)	19 (48.72%)		

*Not applicable for 10 patients

Among the 32 study subjects with minor degree placenta previa, there was 27 live-born out of which 12 (44.44%) babies were admitted in NICU. Among the major placenta previa, there were 39 live-born out of them, 20 (51.28%) were admitted in NICU. The difference in the proportion of NICU admission between type of placenta previa was statistically not significant (P value 0.585). (Table 26 & Figure no 24)

Figure no 24: Cluster bar chart of comparison of NICU admission between types of placenta previa (n=66)

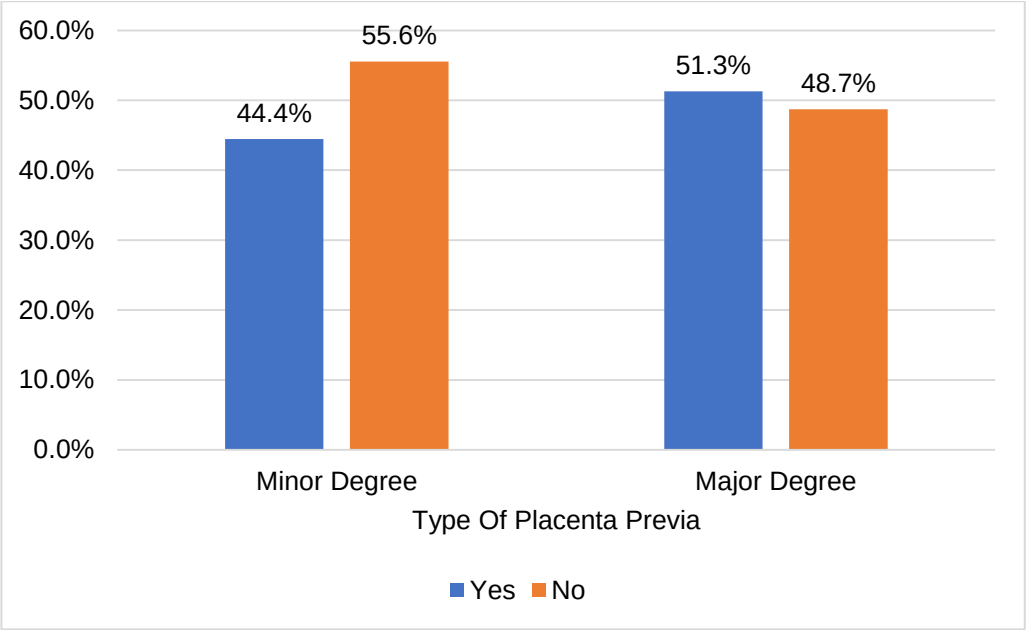


Table No 27: Study of respiratory distress in the live born foetuses (N=66)

Respiratory Distress	Number=66	Percentage
Yes	13	19.7
No	53	80.3

*Not applicable for 10 patients

Among the study population, 13 (19.7%) had respiratory distress. (Table No 27, Figure no 25)

Figure no 25: Pie chart of respiratory distress in the live born foetuses (n=66)

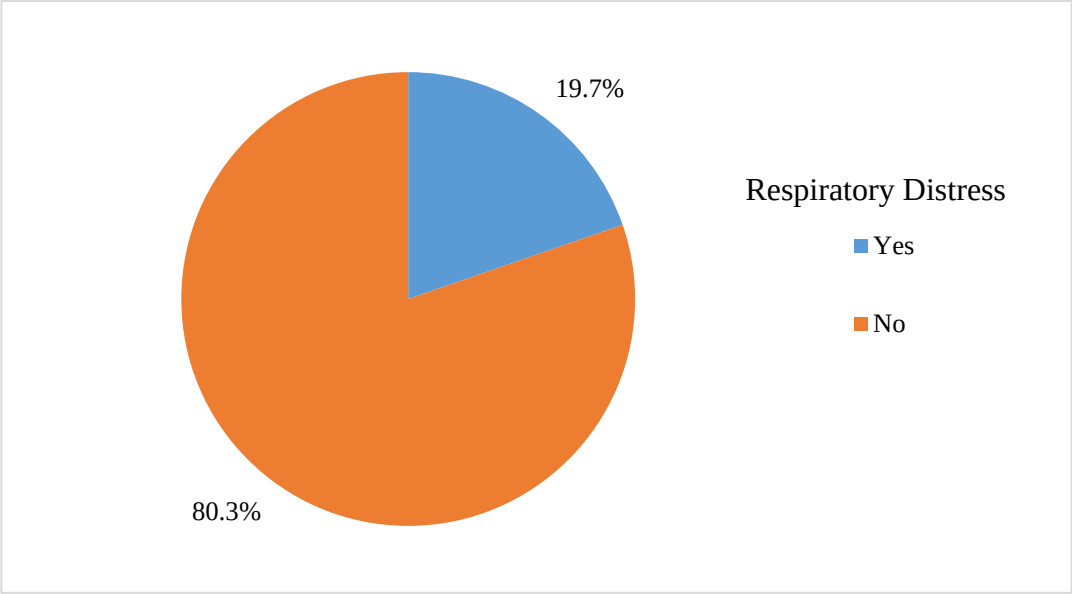


Table no 28: Study of prematurity in the live born foetuses (N=66)

Prematurity	**Number=66**	**Percentage**
Yes	36	54.55
No	30	45.45

*Not applicable for 10 patients

Among the study population, 36 (54.55%) were with prematurity.

In present study out of 76 study subjects 10 were still born. Out of 66 live born, 32(48.5%) cases required NICU admission. Cause for NICU admission being respiratory distress (19.7%), prematurity (53.03%) and out of 32 case admitted to NICU 5 babies died 3 babies due to prematurity and 3 babies due to both respiratory distress and prematurity. (Table No 27,28, Figure no 25)

Table 29: Comparison of prematurity between types of placenta previa (N=76)

Prematurity	Type of Placenta Previa		Chi square	P value
	Minor Degree (No of live born=27)	Major Degree (No of live born=39)		
Yes	15 (55.56%)	21 (53.85%)	0.019	0.891
No	12 (44.44%)	18 (46.15%)		

*Not applicable for 10 patients

Among the minor placenta previa, 15 (55.56%) were premature. Among the major placenta previa, 21 (53.85%) were premature babies. The difference in the proportion of prematurity between type of placenta previa was statistically not significant (P value 0.891). (Table 29 & Figure no 26)

Figure no 26: Cluster bar chart of comparison of prematurity between types of placenta previa (N=66)

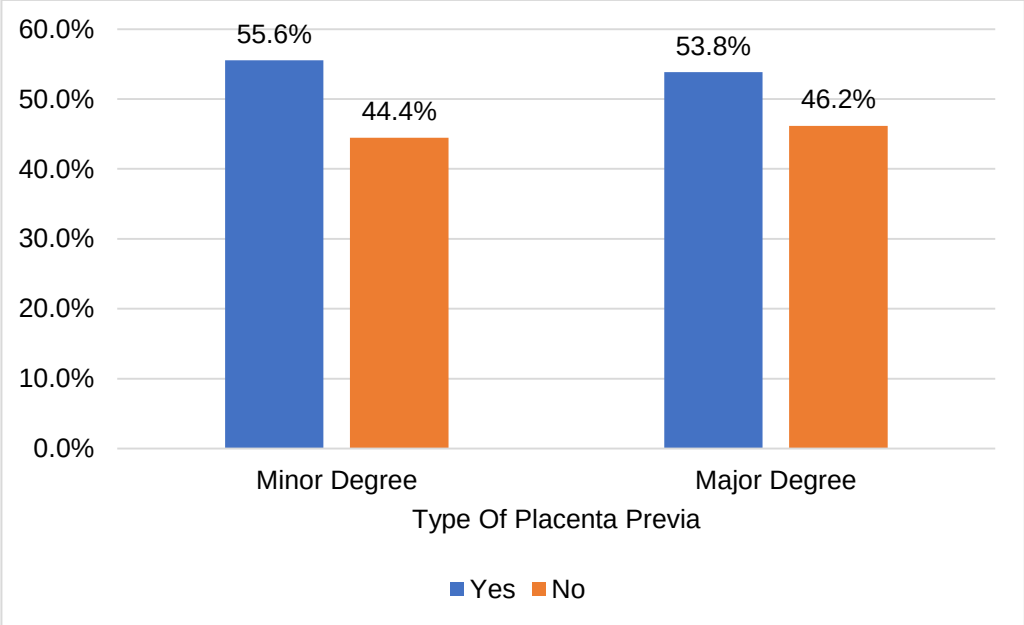


Table no 30: Descriptive analysis of congenital defects in the study subjects

Foetal Anomalies	Number(N=76)	Percentage
Present	3	3.9
Absent	73	96.1

In the present study out of 32 placenta previa minor ,3 babies had congenital anomalies (Table 30 Figure no 27,) (Dolichocephaly, CTEV , congenital pulmonary airway malformations type 3) and there were no congenital anomalies in placenta previa major

Figure no 27: Bar chart of anomalies in the study population (N=76)

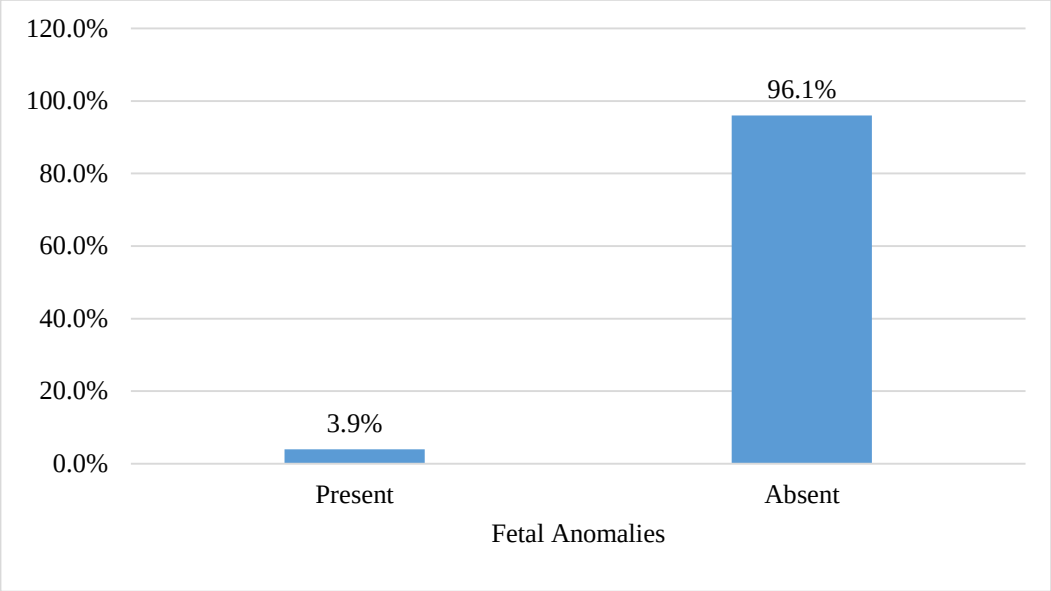


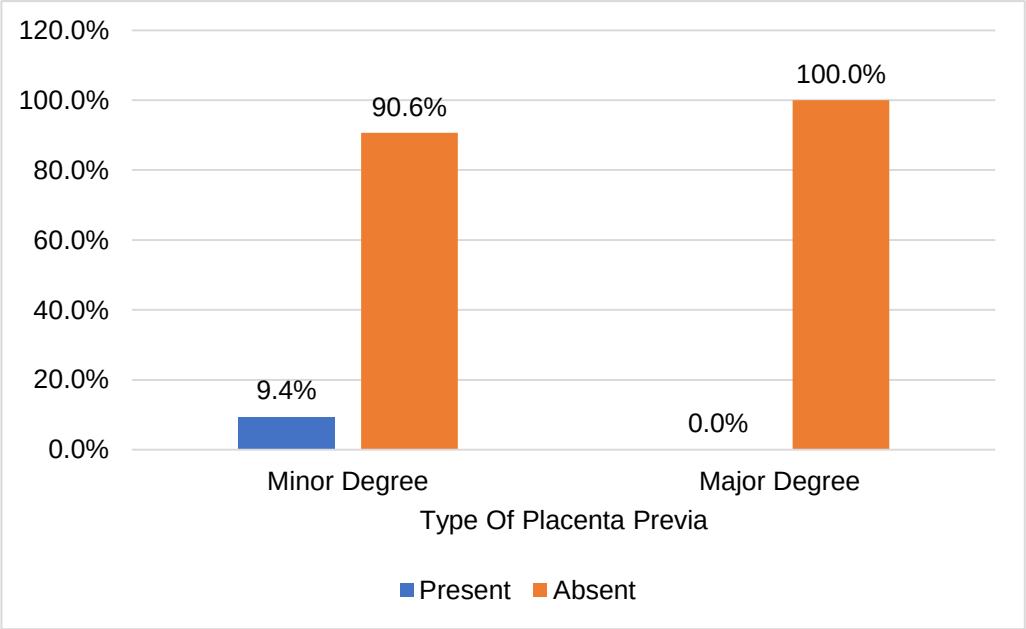
Table 31: Comparison of anomalies between types of placenta previa (n=76)

Anomalies	Types of Placenta Previa (N=76)	
	Minor Degree (N=32)	Major Degree (N=44)
Present	3 (9.38%)	0 (0%)
Absent	29 (90.63%)	44 (100%)

*No statistical test was applied- due to 0 subjects in the cells

Among the placenta previa minor cases, 3 (9.38%) had anomalies. (Table 31 & Figure no 28)

Figure no 28: Cluster bar chart of comparison of anomalies between types of placenta previa (n=76)



DISCUSSION

DISCUSSION

In the present study, analysis of maternal and perinatal outcomes in cases of placenta previa occurring over a period of 2 years from June 2017 to May 2019 at R L Jalappa hospital attached to Devaraj Urs Medical college Tamaka Kolar.

We studied 76 eligible cases of placenta previa, after detail analysis of the cases, the present study was compared with other studies. Our study shows that among 76 placenta previa cases 65.8% were booked while 34.2% remained un booked

From the above study our incidence of placenta previa (1.57%) is almost similar to study conducted by Bhavneet Kaur which was 1.9%.[2] The higher incidence rate observed in our study has been attributed to increasing tendency nowadays to perform caesarean sections even without medical indication on maternal request.

The association between maternal age and incidence of placenta previa was studied in the present study and the incidence of placenta previa was highest in the age group of 20-30 years i.e. 88%. Data correlates with the incidence seen in the same age group reported by Vandana G R[9]in Vishakapatnum(2019) the placenta previa cases were highest in the age group 20-29 years(82%) .Also the data correlates with highest incidence of placenta previa in the same age group I,e 72.9% reported by Prashanth S et al (2016)[97]

In the present study the 58 (76.3%) multiparous women had placenta previa, while 23.6% women were primigravidae. These studies correlate with the statistics of study done by Vandana G R in Vishakapatnum (2019) (78.4%). [9]Also the data correlates with highest incidence of placenta previa (73.55%) in multiparous group reported by Prashanth S et al (2016)[97]and in the study by Sarojini et al. the incidence was more than 80.2% in multiparous.[98]

In the present study the risk factors studied were abortions (11.8%), D and C (7.8%), multiparity (25%) and previous LSCS (25%). Among the subjects with prior caesarean section, 3 subjects had history of previous 2 LSCS, 2 subjects with previous LSCS had Placenta accreta and underwent peripartum hysterectomy. These data correlate with the study by Reddy M et al. where the risk factors were previous LSCS (29.8%), previous abortions 18.3%.[99]

In the present study 78.9% had cephalic presentation, 13.1% had breech presentation, 7.8% had transverse lie, the data correlates with the study by Reddi P Rani in the year 1999 the incidence of malpresentation was 20%, and similar study by McShane (1985) reported that incidence of malpresentation was 27% .[100,101]

In present study in ultrasonography, 34.2% had type 4 placenta previa, 23.6% had type 3 placenta previa followed by type 2 and type 1 being 22.3%, 19.7% respectively, where type 4 placenta previa being more common.

When compared with study by Dwevedi seema et al. (2017) Low lying placenta was present in 75(56.4%) of all Previa cases followed by marginal placenta previa in 34 cases (25.6%) and 11 (8.27 %) had central placenta previa in sonography[102]. In a study of Rangaswamy et al .in 2015, type 1 was the most common type of placenta previa seen in 23 (37.2%) cases[3].

In the present study, out of 76 cases MRI was done for only 2 cases, among them both cases had placenta accrete. Both the cases of placenta accreta underwent peripartum hysterectomy

Placenta praevia is another important risk factor for placenta accreta spectrum. A large multicentre US cohort study noted that for women presenting with placenta

praevia and prior caesarean section the risk of accreta placentation is 3%, 11%, 40%, 61% and 67% for 1, 2, 3, 4, and 5 or more caesarean deliveries, respectively.[103]

The national case–control study using the UK Obstetric Surveillance System found that the incidence of placenta accreta spectrum increases from 1.7 per 10 000 women overall to 577 per 10 000 in women with both a previous caesarean section and placenta praevia.[104]

In the present study among 76 cases, expectant management done for 16 cases according to Macafee and Johnson's protocol, which includes bed rest, periodic blood investigations and cross matched blood ready, frequent foetal surveillance with USG, steroid prophylaxis if gestational age < 34 weeks. Among those cases in whom expectant management was done there was 4 preterm deliveries, 4 babies were admitted to NICU and there was 1 intra uterine foetal demise. Study subjects in this protocol were admitted for steroid prophylaxis and feto-maternal monitoring, later they were discharged. In this protocol gestational age prolonged was maximum 7 weeks and minimum 4 weeks. The data correlates with Sarojini et al. in that study 26.4% of women were managed by Macafee and Johnson's protocol.[98]

In the present study out of 76 cases, 4(5.3%) cases had vaginal delivery, all case delivered by oxytocin augmentation (5.3%). 72(94.7%) cases underwent caesarean section, among them 41 cases had Elective LSCS and 31 cases underwent emergency LSCS. Among 41 cases who underwent elective caesarean section 15 cases had history of the previous LSCS, 24 cases major placenta previa and 2 caesarean section was done for maternal desire. This correlates with data from Sorakayalapeta et al. regarding rate of LSCS (96.1%).

In the present study out of 76 cases 31.5% had atonic PPH of which 13 (40.6%) being minor degree placenta previa. In placenta previa major 11(25%) had PPH and 2 cases (4.5%) had placenta accreta.

Abruption placenta was 3(3.9%) in placenta previa minor of which all three had haemorrhagic shock. Compared to study by Sarojini et al (2012) , 13 (12.7%) cases had postpartum haemorrhage[98] and 5 (4.7%) had adherent placenta and in the study done by Manohar Rangaswamy et al (2016) , Out of 62 cases 10 (16.1%) cases had Atonic PPH, 4 (6.4%) cases were minor degree PPH and 6 (9.6%) cases were of major degree of PPH, of which 4 cases went for haemorrhagic shock. [3]

In this study Minor degree placenta previa had more complications compared to major placenta previa due to associated abruptio placenta.

In the present study out of 76 cases ,24 had intra-operative complications like atonic PPH. The following techniques were followed after failure of medical management. 1 case required bilateral uterine artery ligation (4.1%) followed by internal iliac artery ligation. 2 cases of placenta accrete underwent peripartum hysterectomy (8.3%) and 1(4.1%) case required internal iliac artery ligation to control PPH.

Compared to study done by Sarojini et al (2012), surgical procedures / manoeuvres carried out to control bleeding was 5(4.7%) cases required cervico-isthmic sutures , B-lynch sutures was needed for 3 cases (2.8%), 1.9% required uterine artery ligation, Emergency peripartum hysterectomy was done for 5 cases (4.7%), Emergency peripartum hysterectomy followed by internal artery ligation performed in 01 case (0.9%).[98]

Regarding maternal complications there is increased rate of post-partum haemorrhage, multiple blood and blood products transfusion, ICU admissions which

are attributable to placenta previa. In our study out of 76 cases, 71 (93.4%) cases had uneventful post-operative period. 4cases (5.2%) had haemorrhagic shock, 1 (1.3%) case had Disseminated intravascular coagulation resulted following atonic PPH. All 5 cases required ICU for monitoring. All the 5 cases were stabilised and shifted to ward. And there was no maternal mortality in the present study due to placenta previa. Compared to study done by Sarojini et al (2012) out of 106 cases there were 92 (86.8%) ICU admissions, 4 (3.8%) cases of acute kidney injury, 1 (0.9%) case of septicaemia and 1(0.9%) maternal death.[98]

In the present study out of 76 cases, 40 cases required packed red blood cell transfusion, 10 cases required fresh frozen plasma transfusion, Compared to data from Sarojini et al. where 83% required blood transfusion.[98]

Regarding the perinatal outcome in the present study out of 76 new-born, 7 babies weighed >3kg (9.2%), 27 (35.5%) babies had birth weight of 2.5-3kg, 20 babies (26.3%) had birth weight in between 2-2.49 kg, 10 cases (13.2%) had Birth weight in between 1.5-1.99 kg, 8 cases had weight in between 1-1.49 kg accounts for 10.5%, 4 babies had birth weight <1kg(5.3%).

In our study out of 76 study subjects there was 10 still born babies and 66 live born. Out of which 34 (51.5%) babies didn't require NICU admission, 32 (48.5%) babies required NICU admission. Cause for NICU admission being respiratory distress (19.7%), prematurity (46.1%) and follow up was done. Preterm deliveries in the present study was between 28-32 weeks 18.4%, between 32-36 weeks 25%.

The perinatal mortality was more in the 28-32 weeks gestation group i.e. 14.5% (11 babies), whereas in the 32+1-36 weeks and 36+ weeks gestation group it was (3 babies) 3.9% and 1 baby (1.3%) respectively. Infants with birth weights above 2500

grams had a good survival rate and infants with birth weight <1000 grams were 4 babies out of which only 1 baby survived

Compared to study done by Zlatnik MG et al: (2007), concluded that placenta previa is associated with preterm delivery prior to 28 weeks 3.5%, 32 weeks 11.7% and 34 weeks 16.1%.[70]

CONCLUSION

CONCLUSION

Advancing maternal age, multiparity, prior caesarean section, and prior abortions are independent risk factors for placenta previa. An increase in the incidence of placenta previa due to presence of these risk factors. Placenta previa is associated with adverse maternal and foetal outcome, hence early recognition of risk factors and if possible, prevention of these risk factors is therefore important. Early diagnosis, early transfer to tertiary care centre with adequate management including blood transfusion and timely delivery would likely to reduce maternal and fetal complications with placenta previa

SUMMARY

SUMMARY

The present study was undertaken to evaluate the risk factors, clinical presentation and feto-maternal outcome in placenta previa. In the present study 76 cases of placenta previa were studied regarding studied regarding the risk factors, type of clinical presentation, the clinical course in the hospital, the perinatal and maternal outcome.

The information obtained was analysed statistically. In the present study patients who are unbooked and admitted to hospital as an emergency admission had maximum incidence of maternal morbidity and perinatal mortality.

In the present study, the cases of placenta previa were highest in the maternal age group of 20-30 years, i.e. 86.8%. It was 9.2% in the age group of 31-35 years, 3.9% in the age group >35 years.

In the present study, incidence of placenta previa was highest (76.3%) in the multiparous group, and 23.7% in the primigravida group. In the present study, the risk factors were caesarean section, abortion and multiparity.

In the present study the incidence of placenta previa in prior caesarean section is 25%, abortions (11.8%), D and C (7.8%), multiparity (25%). Of the associated complications studied, in the present study severe anaemia (<7 gm%) contributed to 14.4%, malpresentation contributed to 21% and PIH was found in 10.5% of cases.

The increasing rate of caesarean section will result in increase in the incidence of placenta previa and placenta accreta. Expectant antenatal management will reduce but not totally avoid perinatal mortality. Anticipation of the complications at caesarean section is an important factor in reducing maternal morbidity.

In the present study, 39.5% of cases required blood transfusion and shock/hypotension was noticed in 5.2% of cases, PPH was noticed in 31.5% of cases. In one case internal iliac artery ligation was done, in the other case bilateral uterine artery ligation was done followed by internal iliac artery ligation and 2 cases of placenta accrete underwent hysterectomy.

In the present study, perinatal morbidity was studied as the percentage of babies requiring NICU admission and it was 48.5%.

In the present study, the percentage of perinatal deaths was 19.7%. Prematurity was the major contributor to perinatal deaths, i.e. 54.5% followed by RDS 19.7%.

The perinatal mortality was the same in both the minor and major types of placenta previa I,e 5 babies in each group .

The perinatal mortality was more in the 28-32 weeks gestation group i.e. 14.5% (11 babies), whereas in the 32+1-36 weeks and 36+ weeks gestation group it was (3 babies) 3.9% and (1 baby) 1.3%. Infants with birth weights above 2500 grams had a good survival rate and infants with birth weight <1000 grams had a very poor survival rate. Out of were 4 babies of birth weight <1000 grams only 1 baby survived.

Limitations of this study:

This study is done for two years duration. If the same study is done for longer duration then the data (in form of incidence, etiological factors, associated maternal and foetal morbidity and mortality) will be more accurate. In this study only the cases of placenta previa who were admitted in labour room and who were delivered are included. Thorough follow-up of placenta previa cases during antenatal period was not done. Exact staging of all cases of placenta previa was not done as some patients came directly as emergency cases in labour room and their general physical condition were not that good. So, they were directly taken to operation theatre.

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ANNEXURES

PATIENT CONSENT FORM

Case no:

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I have understood that I have the right to refuse consent or withdraw it at any time during the study and this will not affect my treatment in any way. I consent voluntarily to participate in this study

".MATERNAL AND FETAL OUTCOME IN PLACENTA PREVIA AT TERTIARY CARE CENTRE"

Name of Participant_____

Signature/ thumb print of Participant _____

Date _____

Statement by the researcher/person taking consent:

I have accurately read out the information sheet to the potential participant and to the best of my ability made sure that the participant understands that the following will be done:

I confirm that the participant was given an opportunity to ask questions about the study and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

Name of Researcher/person taking the consent_____

Signature of Researcher /person taking the consent_____

Date _____

Name and Address of Principal Investigator: Dr Rashmi S Jayaraj .

R.L Jalappa Hospital

Tamaka, Kolar.

PROFORMA

Name

MR NO :

Age

IP NO :

Address

Occupation

Husband's name

Socioeconomic status

Education

HISTORY

CHIEF COMPLAINTS

HISTORY OF PRESENTING ILLNESS duration

H/O amenorrhea yes/no

H/o bleeding /PV spotting yes/no

H/o pain abdomen yes/no

HISTORY OF PRESENT PREGNANCY

BOOKED/UNBOOKED/REFERRED

TRIMESTER1

TRIMESTER2

TRIMESTER3

OBSTERTRIC HISTORY

G P L D A Age at marriage

Married life

MENSTRUAL HISTORY

Age of menarche

Past menstrual cycles: regular/irregular duration of flow

Duration of cycle heavy/mod/less

Dysmenorrhea:

Last menstrual period:

Expected date of delivery:

Gestational age

PAST HISTORY

Previous history of placenta previa yes/no

H/o Hypertension: yes/no

H/o Diabetes: yes/no

Past history of medical illness/blood transfusion/surgeries

Habits: smoking: alcohol: others:

PHYSICAL EXAMINATION

Concious or not :

Height:

Temperature: Weight:

Blood pressure:

BMI:

Pallor +/-

Icterus +/-

Edema +/-

B/L Breast, spine, thyroid:

R/S: CVS:

P/A:

Uterine height:

Uterine contraction:

Presentation:

Head= Floating / engaged

Position:

P/S:

P/V- (In Operation theatre)

PROVISIONAL DIAGNOSIS

INVESTIGATIONS

Hb: PCV:

Urine routine:

HIV HBsAG VDRL

Obstetric Ultrasonography:

Non Stress Test:

TREATMENT:

Mode of delivery: Vaginal/Caesarean section

Maternal outcome: Postpartum haemorrhage:

Need for blood or blood components transfusion: Yes /no

Puerpural complications: yes/no

Death : yes/no

Fetal outcome: Preterm/term:

Live born/still born/macerated

Sex:

Congenital anomalies

Birth weight:

Apgar score

If live born ,mother side or NICU care

Condition on discharge:

ANNEXURE-I

CONSENT FORM

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಅಭ್ಯಾಸದ ಹೆಸರಿಕೆ:

ಅಭ್ಯಾಸದ ಕ್ರಮ ಸಂಖ್ಯೆ:

ವ್ಯಕ್ತಿಯ/ರೋಗಿಯ ಹೆಸರು:

ಜನ್ಮ ದಿನಾಂಕ/ವಯಸ್ಸು:

- ಈ ಮೇಲೆ ಹೇಳಿದ ಎಲ್ಲಾ ವಿವರಣೆಗಳನ್ನು ಹಾಗೂ ಈ ಅಭ್ಯಾಸದ/ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ನಾನು ಓದಿ ಅರ್ಥ ಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಎಂದು ಖಚಿತಪಡಿಸುತ್ತೇನೆ. ಈ ಅಭ್ಯಾಸದ/ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ಮಾಡಿಕೊಟ್ಟಿದ್ದಾರೆ.

- ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಸ್ವಇಚ್ಛೆಯಿಂದ ಭಾಗವಹಿಸುತ್ತಿದ್ದೇನೆ ಎಂದು ನಾನು ತಿಳಿದಿದ್ದೇನೆ.

- ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲೂ ಕೂಡ ಈ ಅಧ್ಯಯನದಿಂದ ಮುಕ್ತಗೊಳಿಸಲು/ಹೊರಬರಲು ನನಗೆ ಸ್ವಾತಂತ್ರವಿದೆ. ಹಾಗೂ ಈ ಅಧ್ಯಯನದಿಂದ ಹೊರಬರಲು ನಾನು ಯಾವುದೇ ರೀತಿಯ ಕಾರಣ, ವೈದ್ಯಕೀಯ ಕಾಳಜಿ ಹಾಗೂ ಕಾನೂನು ರೀತಿಯಿಂದ ತೊಂದರೆಯಾಗದಂತೆ ನೋಡಿಕೊಳ್ಳುತ್ತೇನೆ.

- ಈ ಅಭ್ಯಾಸಕ್ಕೆ / ಅಧ್ಯಯನಕ್ಕೆ ಪ್ರಾಯೋಜಿಸಿದವರು, ಪ್ರಾಯೋಜಕರ ಪರವಾಗಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಕೆಲಸ ಮಾಡುವವರು, ಅಧ್ಯಯನದ ನೈತಿಕ ಮೌಲ್ಯವನ್ನು ನಿರ್ಧರಿಸುವ ಮಂಡಳಿ ಮತ್ತು ಕಾರ್ಯಕಾರಿ ಮಂಡಳಿಯವರು ನನ್ನ ಆರೋಗ್ಯದ ಬಗ್ಗೆ ಸಂಶೋಧನೆ ಮಾಡುವಾಗ ಯಾವುದೇ ರೀತಿಯ ಮಾಹಿತಿ/ದಾಖಲೆಗಳನ್ನು ನೋಡುವಲ್ಲಿ ನಷ್ಟ ಪರವಾನಿಗೆಯನ್ನು, ತೆಗೆದುಕೊಳ್ಳಬೇಕಾಗಿಲ್ಲ. ನಾನು ಈ ಅಧ್ಯಯನದಿಂದ ಮುಕ್ತಗೊಂಡರೂ ಕೂಡ ಅವರಿಗೆ ಮಾಹಿತಿ ಬೇಕಾದಲ್ಲಿ ತೆಗೆದುಕೊಳ್ಳಬಹುದು.

- ಯಾವುದೇ ಸಂದರ್ಭದಲ್ಲೂ ನನ್ನ ಗುರುತಿನ ಬಗ್ಗೆ, ನನ್ನ ಮಾಹಿತಿಯ ಬಗ್ಗೆ ಯಾವುದೇ ಮೂರನೇ ವ್ಯಕ್ತಿಗೆ ತಿಳಿಯದಂತೆ ಅಧ್ಯಯನ ಮಾಡುತ್ತಾರೆ ಎಂದು ನಾನು ತಿಳಿದುಕೊಂಡಿರುತ್ತೇನೆ.

ವೈದ್ಯರ ಸಹಿ/ಹೆಸರು: ವ್ಯಕ್ತಿಯ/ರೋಗಿಯ ಸಹಿ:

ಮಾರ್ಗದರ್ಶಿಯ ಹೆಸರು/ಸಹಿ: (ವ್ಯಕ್ತಿಯ/ರೋಗಿಯ ಪೂರ್ಣ ಹೆಸರು)

PATIENT INFORMATION SHEET

MATERNAL AND FETAL OUTCOME IN PLACENTA PREVIA AT TERTIARY CARE HOSPITAL

Principal investigators: Dr. Rashmi s. Jayaraj / Dr. Munikrishna M I, Dr. Rashmi S Jayaraj, a post graduate student at the Sri Devaraj Urs Medical College, will be conducting a study titled MATERNAL AND FETAL OUTCOME IN PLACENTA PREVIA AT TERTIARY CARE HOSPITAL

for my dissertation under the guidance of Dr. Munikrishna M, Professor, Department of Obstetrics and Gynecology. Participants of this study i,e pregnant women diagnosed with placenta previa will be included in a observational study where in all the pregnant women who visit outpatient department and labour ward with painless bleeding to RLJH hospital will be admitted and examined thoroughly by clinical examination and diagnosis is confirmed by ultrasography , routine antenatal investigations done. Maternal and Perinatal outcome is documented. patients participating in this study wont have any financial compensation .

All the data will be kept confidential and will be used only for research purpose by this institution. You are free to provide consent for participation in this study.

Name and signature of the Chief Inspector

Date

KEY MASTER CHART

AGE (yrs)

<20-1

20-25-2

25-30-3

30-35-4

>35-5

SOCIOECONOMICAL STATUS

UPPER INCOME >6346 -1

UPPER MIDDLE 3173-6345-2

MIDDLE CLASS-1904-3172 -3

LOWER MIDDLE 952-1903 -4

LOWER CLASS <-951 -5

ANC

BOOKED 1

UNBOOKED 2

PARITY

PRIMI -1

multigravida 2

POG AT DELIVERY

28-32 Weeks-1

32+1 -36 weeks - 2

36+1-40weeks-3

>40 Weeks -4

SYMPTOMS

ASYMP-0

PAIN ABDOMEN-1

BLEEDING PV-2

LEAK P/V -3

INCIDENTAL FINDING-4

RISK FACTOR

NO RISK -0

ABORTION -1

LSCS -2

MUTIPARITY -3

previous history of placenta previa-4

ANTENATAL COMPLICATIONS

ANTENATAL COMPLICATIONS

NIL -0

2ND TRIMESTER BLEEDING -1

SEVERE ANEMIA (<7)-2

PIH -3

IUD-4

GDM-5

ABRUPTION WITHPLACENTA PREVIA -6

CHORIOAMNIONITS -7

PRESENTATION

VERTEX -1

BREECH-2

TRANSVERSE -3

USG FINDINGS

TYPE I -1

TYPE II-2

TYPE III -3

TYPE Iv-4

MRI FINDING

MRI FINDING

NORMAL -0

ACCRETA -1

INCRETA-2

PERCREATA-3

NOT DONE-4

MANAGEMENT

EXPECTANT -1

ACTIVE -2

ROUTE OF DELIVERY

ABDOMINAL -A VAGINAL -B

EMERGENCY -1 SPONTENOUS-1

ELECTIVE -2 OXYTOCIN AUG-2

INTRA OP COMPLICATIONS

INTRA OP COMPLICATIONS

NIL-0

SHOCK-1

PPH-2

ABRUPTION-3

AKI-4

ACRETA-5

INCRETA-6

PERCRETA-7

RUPTURE UTERUS-8

EXTRA SURGICAL INTERVENTIONS

SURGICAL INTERVENTIONS

B/L UAL-1

B-LYNCH-2

HAYMANS-3

INTERNAL ILIAC ARTERY LIGATION -4

HYSTERCTOMY-5

HYSTERCTOMY+ BLADDER REPAIR-6

NOT REQUIRED-0

POST OP COMPLICATIONS

haemorrhagic shock-1

SEPSIS-2

AKI-3

DIC-4

DEATH-5

NIL-6

BLOOD TRANSFUSION DETAILS

NOT REQUIERD -0

PRBC -1

PRBC+FFP-2

PERINATAL OUTCOME

BIRTH WEIGHT

2.5-3kg=1

2-2.49=2

1.5-1.99=3

1-1.49=4

<1 KG=5

NICU ADMISSION

YES-1

NO-2

RESP DISTRESS

YES-1

NO-2

PREMATURITY

YES-1

NO-2

SEPSIS

YES-1

NO-2

DEATH

YES-1

NO-2

POG at diagnosis

<30 weeks-1

31-36 weeks -2

36+1-40weeks-3

>40weeks-4

congenital anomolies in neonate

present-1

absent-2

		AGE (	SOCIOECONON	ANC	PARITY	POG at d	POG AT A	SYMPT(	RISK FA(	ANTENA	PRESENTA	USG FIND	MRI FIN	POG at	POG AT DELIVERY		MANAGE(	ROUTE OF D	ROUTE OF D	INTRA O	EXTRA SURG	POST OP COM	BLOOD TRA	PERINATAL O	NICU ADMIS	RESP DIS	PREMATURITY		DEATH	ANOMAL
UHID	**name**	**ge (yrs)**																**abdominal A**	**vaginal B**										
507784	vijay laks	3	3	1	2	2	3	0	2	0	1	1	0	1	3		1	A-2		0	0	6	1	2	1	2	1	2	2
519187	Sowmya	2	1	1	2	1	1	0	0	1,2	1	4	0	1	1		2	A-2		0	0	6	2	4	still born	2	1	1	2
525041	Aruna	2	1	2	2	1	1	2	2	1	1	4	0	1	1		2	A-2		0	0	6	0	4	1	2	1	1	2
523728	yashoda	3	1	1	2	2	3	2	1,2	0	2	4	0	1	3		1	A-1		0	0	6	0	1	2	2	2	2	2
512707	veena	2	2	2	2	1	1	2	3	1	1	2	0	1	1		2	A-1		2	0	1	1	4	1	1	1	2	2
525755	sunitha	5	3	1	2	2	3	1,2	1	1,3	1	4	0	1	3		1	A-1		0	0	6	1	2	1	1	2	2	2
527914	renuka	3	3	1	1	2	3	0	0	0	1	4	0	2	3		1	A-2		0	0	6	0	1	2	2	2	2	2
542590	Indrani	2	3	1	1	2	3	2	0	1	1	3	0	2	3		1	A-1		0	0	6	0	1	2	2	2	2	2
531807	Swathi	2	3	1	1	2	4	0	0	0	1	1	0	2	4		2	A-2		0	0	6	0	1	2	2	2	2	2
546647	anitha	2	1	1	1	2	3	1	0	2	1	1	0	1	3		2	A-1		2	0	6	2	0	1	1	2	2	2
548205	Nagarata	4	1	1	2	2	3	0	2	0	1	1	0	2	3		1	A-2		0	0	6	0	1	2	2	2	2	2
548482	Misba	2	1	1	2	2	2	0	1,2,3	0	1	1	0	2	2		1	A-2		2	0	6	0	2	1	2	1	2	2
554653	shahisth	2	3	1	2	2	3	2	0	1	1	3	0	1	3		2	A-1		0	0	6	0	1	2	2	2	2	2
555925	ruhi	3	4	1	2	2	3	2	0	1	1	3	0	1	3		1	A-1		0	0	6	0	2	2	2	2	2	2
561552	savitha	2	3	2	2	2	2	2	0	1	1	2	0	2	2		2	A-1		0	0	6	0	2	1	2	1	2	2
554540	Farzana	3	1	1	2	2	3	0	3	0	1	2	0	1	3		1	A-2		0	0	6	0	1	2	2	2	2	2
505077	Asma	3	1	1	1	1	3	2	0	1	1	1	0	1	3		1	A-1		0	0	6	0	1	2	2	2	2	2
573640	Sushma	2	3	2	2	2	2	2	1	1,2	2	2	0	2	2		2	A-1		3	0	6	1	3	1	1	1	2	1
580390	saritha	2	3	2	2	4	4	2	0	1	1	2	0	2	4		2	A-1		2	0	6	1	1	2	2	2	2	2
581582	Muniratl	4	3	1	2	1	1	2	0	0	1,2	1	0	1	1		2	A-1		3	0	6	1	5	1	1	1	1	2
582687	Muni rat	5	3	2	2	1	1	2	3	1	2	4	0	1	1		2	A-1		2	0	6	1	5	1	2	1	1	2
583317	Priyanka	2	3	1	2	2	2	2,3		0	1	1	3	0	2	2	2	A-1		0	0	6	1	1	1	1	1	2	2
584693	Bhavya	3	1	1	1	2	3	2	0	1	1	3	0	1	3		2	A-1		0	0	6	1	1	2	2	2	2	2
587287	Tasina ta	2	2	2	1	2	2	0	0	0	1	3	0	1	2		2	A-1		0	0	6	1	2	1	2	1	2	2
581714	shahira	2	1	1	2	3	3	1	2	0	1	4	0	2	3		1	A-1		2	4	1	1	2	2	2	2	2	2
591584	mala	2	3	1	2	2	3	0	1	0	1	4	0	2	3		1	A-1		0	0	6	1	0	2	2	2	2	2
445444	Laxmide	2	3	2	2	2	2	2	3	1	1	4	0	1	2		2	A-2		1	0	6	0	0	1	1	2	2	2
592564	lavanya	2	3	1	1	1	1	0	0	3,4	1	4	0	1	1		2	A-2		2	0	6	1	4	1	2	1	1	2
446587	sunnada	2	1	1	1	2	3	0	0	0	1	2	0	1	3		1	A-2		0	0	6	0	1	2	2	2	2	2
598929	srimathi	2	1	1	2	2	3	2	4	1,2,4	3	2	0	2	3		2	A-2		1,3	0	6	2	3	2	2	2	2	1
461818	mubin ta	2	1	1	2	2	3	2	3	1,3	2	4	0	1	3		2	A-2		2	0	6	1	2	still born	2	1	1	2
6509	shameer	2	4	1	2	2	3	2	2	1	1	2	0	2	3		2	A-2		2	0	6	1	2	2	2	1	2	2
611976	Lakshma	2	3	2	1	4	4	2	0	1	2	1	0	4	4		2	A-2		2	0	6	1	1	2	2	2	2	2
615933	shahida	2	3	2	1	3	3	2	0	1	1	4	0	3	3		2	A-2		0	0	6	0	0	2	2	2	2	2
616341	rani	2	3	1	2	2	3	1	2	2	1	3	0	3	3		2	A-2		0	0	6	1	1	2	2	2	2	2
582612	mamtha	2	3	1	1	2	3	2	0	1	1	3	0	2	3		1	A-2		0	0	6	0	1	2	2	2	2	2
569951	kalavath	2	4	1	2	2	3	0	0	0	2	1	0	3	3		1	A-1		2	0	6	1	2	1	1	2	2	2
618317	supriya	2	1	1	2	2	3	0	2	0	1	2	0	3	3		2	A-1		0	0	6	0	1	2	2	2	2	2
704382	mouseer	2	1	2	1	1	1	2	0	1,2	1	1	0	1	1		2		B-2	0	0	6	1	3	still born	2	1	1	2
692057	hajira	2	1	1	2	2	2	2	2	1	1	3	0	2	2		2	A-2		0	0	6	0	4	still born	2	1	1	2
670122	shilpa	2	2	2	2	1	1	2	3	1	1	1	0	1	1		2		B-2	2	0	6	1	3	still born	2	1	1	1
688545	roopa	2	3	2	2	3	3	1	2	2	3	4	0	3	3		2	A-2		2	0	6	1	2	1	1	2	2	2
674002	anusha	2	3	1	2	2	2	2	0	1	1	3	0	1	2		2	A-2		2	0	6	1	3	1	2	1	2	2
678677	ameena	2	2	1	2	2	3	0	1,3	3	1	2	0	2	3		2	A-2		2	0	6	1	3	1	2	2	2	2
698891	sharadha	3	1	1	2	1	3	0	1,3	0	2	3	0	1	3		2	A-1		0	0	6	0	1	2	2	2	2	2
676832	suma	3	4	1	2	2	2	2	2	1	2	1	0	1	2		2	A-2		1,2	0	6	1	1	1	2	1	2	2
677649	nusrath	3	1	2	2	1	1	2	1,2	1,2,4	1	2	0	1	1		2		B-2	1	0	6	2	4	IUD	2	1	1	2
699810	leelavath	3	2	2	2	3	3	2	3	1,2	3	3	0	2	3		2	A-1		2	0	6	2	0	1	2	2	2	2
622532	sabithri	3	3	2	2	2	2	2	0	1	1	1	0	1	2		2	A-1		2	0	6	2	2	2	2	1	2	2
622838	nikhath	2	3	2	1	4	4	0	0	0	1	2	0	2	4		2	A-1		2	0	6	2	1	2	2	2	2	2
630886	manjula	2	3	2	2	1	1	2	3	1,2	1	4	0	1	1		2	A-1		0	0	6	0	3	1	1	1	2	2
692143	saraswat	2	1	1	1	1	3	0	0	0	3	4	0	1	3		2	A-1		2	0	6	0	1	2	2	2	2	2
640621	hemalat	5	1	1	2	1	3	0	1,2	0	3	3	0	2	3		2	A-1		2	0	6	0	1	2	2	2	2	2
645589	shabana	3	4	2	2	3	3	0	3	0	1	4	0	2	3		2	A-1		0	0	6	0	0	1	1	2	2	2
618372	padma	2	4	1	2	2	3	0	3	0	3	4	0	1	3		2	A-2		0	0	6	0	1	2	2	2	2	2
570007	mouna	2	3	1	2	2	2	2	0	1	1	3	0	1	2		2	A-1		0	0	6	0	2	2	2	1	2	2
548779	madhavi	2	4	1	2	2	3	0	0	0	1	3	0	2	3		2	A-2		0	0	6	0	0	2	2	2	2	2
652140	chaithan	3	4	1	2	2	3	0	1,2	0	1	2	0	2	3		2	A-1		2	0	6	1	1	2	2	2	2	2
654816	saniredd	3	4	1	1	2	3	2	0	1,3	1	3	0	2	3		2	A-2		0	0	6	0	1	1	1	2	2	2
642387	ashwini	4	2	1	2	2	2	0	0	4	1	4	0	1	2		1	A-2		0	0	6	0	2	IUD	2	1	1	2
654899	nagalaks	3	3	2	2	1	1	2	3	1	1	4	0	1	1		2	A-2		0	0	6	1	3	1	2	1	2	2
666154	deepa	2	4	1	1	2	2	1	0	3,6	2	2	0	1	2		2	A-2		0	0	6	0	4	STILL BORN	2	1	1	2
682977	gayathri	2	4	1	2	1	3	1	1	0	1	2	0	2	3		2	A-2		0	0	6	1	2	1	2	2	2	2
683024	kavitha	3	4	2	2	3	3	1	2	0	1	2	0	2	3		2	A-2		0	0	6	0	1	2	2	2	2	2
548779	monika	3	4	2	2	2	2	2	1	1,6	1	2	0	1	2		2	A-2		0	0	6	1	2	1	2	1	2	2
704750	gultaz	3	3	2	2	3	3	2	3	1	1	4	0	2	3		2	A-2		0	0	6	0	1	2	2	2	2	2
724360	apsari sa	4	3	1	2	2	2	1	2,3	1,,5	1	4	1	2	2		2	A-2		5	5	1	2	2	1	2	1	2	2
714053	usha	4	2	2	2	1	1	2	1,3	1	1	1	0	1	1		2		B-2	0	0	6	0	5	still born	2	1	1	2

723441	jayashre	2	2	1	2	1	2	2	2	1,3	1	4	0	2	2		2	A-2		2	0	6	1	3	1	2	1		2	2
709851	felena	4	2	2	1	2	2	1	0	1	1	3	0	2	2		2	A-2		0	0	6	0	5	1	2	1		2	2
721860	vasantha	2	2	1	2	2	3	0	1,3	0	1	1	0	2	3		2	A-1		0	0	6	0	2	2	2	2		2	2
738925	vani	2	2	1	2	2	1	2	0	0	1	4	0	1	1		2	A-2		0	0	6	0	4	still born	2	1		1	2
707779	shwetha	3	2	1	2	2	2	0	2	2	1	3	1	2	2		2	A-2		5	5	1	0	2	1	2	1		2	2
760993	shanthi s	2	2	2	2	1	1	2	0	1,6	2	4	0	1	1		2	A-2		1,3	0	6	2	3	1	1	1		1	2
741274	shireen t	2	3	1	2	2	3	0	3	3	1	4	0	2	3		2	A-2		0	0	6	1	1	2	2	2		2	2
717935	arshiya b	4	3	1	2	2	2	2	1	1	1	4	0	2	2		2	A-2		1,2	1,4	4	2	2	1	2	1		2	2