COMPARATIVE STUDY OF MATERNAL AND PERINATAL OUTCOME OF ABRUPTIO PLACENTA IN NORMOTENSIVE AND HYPERTENSIVE PATIENTS

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CERTIFICATE

This is to certify that this dissertation entitled " **COMPARATIVE STUDY OF MATERNAL AND PERINATAL OUTCOME OF ABRUPTIO PLACENTA IN NORMOTENSIVE AND HYPERTENSIVE PATIENTS** " is a bonafide record work done by **Dr.A.NITHIYA** submitted as partial fulfillment for the requirements of M.D Degree Examination- Obstetrics and Gynaecology to be held in April 2012.

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Contents

Introduction

Antepartum hemorrhage is defined as bleeding from genital tract after 20 weeks of gestation. There is no identifiable cause in almost half of the patients. Bleeding from the placental bed is the most common identifiable cause. [14] The major causes of antepartum hemorrhage includes placenta previa (20%) and placental abruption (30%) .[7]

Uterine bleeding during the second and third trimesters is a relatively common complication of pregnancy occurring in approximately 4-5% [14,28,]of pregnancies and from whatever the cause, is associated with an increase in premature births and perinatal deaths.

Placental abruption is a serious obstetric condition that increases maternal and neonatal morbidity and mortality [39].

Abruptio placenta is defined as separation of the normally located placenta after the 20th week of gestation and before the birth of fetus.[14,27] Placental abruption is due to bleeding into decidua basalis leading to separation of the placenta from the uterine wall resulting in compromise of blood supply to the fetus[26] .

Other names of abruptio placenta are - Abalatio placenta, premature separation of the placenta & accidental hemorrhage.

Edward Rigby (1774) identified accidental hemorrhage as separate entity. Lec(1848) and coole(1848) used the term placental apoplexy .De lee(1982) used abruption which denotes sudden accident.

Rudolf Holmer(1901) interpreted ablation of placenta as carrying away of placenta. This term deeply routed in British literature as accidental hemorrhage. In Latin, abruption means rending asunder of placenta.[49]

Two Major pathways [9,46] have been proposed to account for placental abruption.

1. **Inflammation associated process**

Histologic chorioamnionitis is associated with placental abruption[8], [11].

2. **Ischemia associated process**

The causes of abruptio placenta are unknown however many maternal risk factors have been identified including maternal age, parity, socioeconomic status, tobacco & cocaine use, previous history of abruptio placenta, multifetal gestation, premature rupture of membranes & hypertension.[34]

Maternal complications of abruption includes hemorrhagic shock, disseminated intravascular coagulation(DIC), renal failure & death. Neonatal complications include prematurity, birth asphyxia, still birth [3, 23].

Hypertensive disorders during pregnancy have accounted for a relatively high incidence of all cases of abruptio placenta that occur. However there is controversy in the outcome of women with hypertensive disorders and abruptio placenta compared with normotensive women with abruptio placenta. The objective of this study was to compare the perinatal and maternal outcome in normotensive and hypertensive women with abruptio placenta.

AIM OF THE STUDY

1. To conduct an in depth analysis of abruption in normotensive and hypertensive patients in order to find out the influence of various parameters.
2. To find out the incidence of abruption in our institution.
3. To find out the relationship of incidence to age and parity.
4. To find out various maternal morbidity & mortality.
5. To find out perinatal morbidity &mortality rate.
6. To find out the factors contributing to maternal and perinatal morbidity and mortality.
7. To find out the influence of hypertension in the incidence, maternal and perinatal outcome of abruption.

MATERIALS AND METHODS

This is a prospective study conducted in Raja Mirasudhar Hosptial, Thanjavur. during the period of January 2010 to June 2011.

Inclusion Criteria

Group A (Normotensive patients with abruptio placenta)

1. Only those women who delivered singletons beyond 20 weeks of gestation were included in this study.
2. Abruption was diagnosed mainly on clinical signs and symptoms and ultra sound.

Group B (Hypertensive patients with abruptio placenta)

1. Hypertension was diagnosed based on either two measurements of BP $\geq$ 140/90 mmHg on two or more consecutive occasions and taken at least 4 hours apart.
2. If patient presented with hypovolemic shock, hypertension was apparent once the depleted intravascular compartment is adequately refilled. (Pritchard and co-workers 1991) [49]

Exclusion criteria:

1. All patients came with history of antepaetum hemorrhage but where diagnosed to have placenta previa and other local causes.
2. All patients with abruption delivered in other hospitals and referred to our hospital for the management of post partum complications.

Methods:

1. Detailed history was collected regarding maternal characteristics including maternal age, parity, gestational age at admission, socio economic status, antenatal visits, past obstetric history including a history of previous pregnancy with placental abruption, any current obstetric complications like Chronic hypertension, pre eclampsia, prelabour rupture of membrane, polyhydramnios.
2. All study subjects underwent a complete obstetrical clinical workup including general physical examination, abdominal and pelvic examination.

3. USG done for placental localization and for retroplacental clots, fetal presentation, fetal maturity, fetal viability, liquor.
4. Venous blood sample taken for blood grouping & Rh typing, cross matching, Hb%, blood urea, serum creatinine, clotting time,clot retraction time, prothrombin time, platelet count
5. Bladder catheterisation done for monitoring urine output.
6. Blood pressure and pulse rate monitored every 15 min. urine output and clotting time monitored hourly.
7. ARM and inj. oxytocin for acceleration of labour if there is no contraindication for vaginal delivery.
8. Lower segment caesarean section done wherever needed.
9. Hypovolemic shock managed with intravenous infusion of normal saline, ringer lactate, whole blood transfusion.
10. Coagulation failure managed with fresh frozen plasma, platelet transfusion.
11. Appropriate management of renal failure if occurs.
12. Neonatal outcome data were recorded including gestational age at devliery, birth weight , sex of the baby, apgar score at 1 minute and 5minute.

In this study, the diagnosis of placental abruption is made primarily by clinical examination, and the diagnosis is confirmed by the presence of retro placental clots after delivery.

Review of literature

Definition:

Placental abruption is defined as separation of the placenta from its implantation site before the delivery of the fetus [49,21] The cumbersome term premature separation of normally implanted placenta is most descriptive. It differentiates the placenta that separate prematurely but is implanted some distance beyond the internal os that is placenta previa.[49]

Incidence

There is wide variation in the incidence of abruption mainly due to variation in the diagnosis. [14]

The incidence varies from 0.49 to 1.8 %. [25] The incidence of abruption is 0.6 to 1% of all births. [21,25,47] it is about 1 in 200 deliveries [49]

The incidence of abruption increases with gestational age. Abruption usually occurs in the last 4 weeks of gestation[21] & most commonly around 34 weeks [25]. More than 90% fetus involved weighed more than 1500 grams. [21]

Abruption severe enough to cause fetal death occurs in approximately 1 in 100 deliveries with a range of 0.52 to 1.29% [21].

Incidence of abruption differ in different socio economic groups [21].

Types of abruption:

1.Revealed hemorrhage.

2.Concealed hemorrhage.

3.Chronic placental abruption.

Revealed hemorrhage

The incidence of revealed hemorrhage is 65- 80 % [7,14]. Abruption may be"revealed," in which case blood tracks between the membranes and the decidua, and escapes through the cervix into the vagina [49].

Concealed hemorrhage

The incidence of concealed hemorrhage is 20-35% of [14] .

The blood does not escape externally but it retained between detached placenta and the uterus.Concealed hemorrhage has much greater maternal and fetal hazards because of possible consumptive coagulopathy and the extent of hemorrhage is not readily appreciated & the diagnosis typically is delayed.[49]

concealed hemorrhage occurs because of following reasons [49] :

1. There is an effusion of blood behind the placenta, but its margins still remain adhered.

2. The placenta is completely separated yet the membranes retain their attachment to the uterine wall.

3. Blood gains access to the amniotic cavity after breaking through the membranes.

4. The fetal head is so closely applied to the lower uterine segment that blood cannot make its way past.

But Most commonly mixed variety is seen. [25] The extent to which bleeding is concealed and revealed may be determined by the tone and contractility of uterine musculature.[25]

Depending upon the extent of placental separation ,abruption may be total, involving the entire placenta, in which case it typically leads to fetal death [10], or partial, with only a portion of the placenta detached from the uterine wall.[49]

One more variety is chronic placental abruption.

Chronic placental Abruption:

In some women hemorrhage with retro placental hematoma formation is somehow arrested completely without delivery. We can document this phenomenon by labeling maternal red cells with 5% chromium . In one case red blood cells well concealed as a 400 ml clot, which was found within the uterus at delivery 3 weeks later. In other case the clot contained no radio chromium whereas peripheral blood at that time did. The blood in the clot therefore had accumulated before the erythrocytes were labeled. [49]

Aetiology

Primary cause of placental abruption is unknown but several associated risk factors for placental abruption were [14,21]

Risk factor	relative risk
1.Increased age and parity	1.3 – 1.5
2.Preeclampsia	2.1 – 4.0
3 .Chronic hypertension	1.8 – 3.0
4. Preterm ruptured membrane	2.4 – 4.9
5. Multifetal gestation	2.1
6. Low birth weight	14.0
7 .Polyhydromnios	2.0
8. Cigarette smoking	1.4 – 1.9
9. Thrombophilias	3- 7
10 .Cocaine use	NA
11. Prior abruption	10 – 25
12. Uterine leiomyoma	NA
13. External trauma	

14. Intrauterine infection

15. Oligohydromnios

16. Diabetes mellitus

17. Short umbilical cord

18. Alcohol (> 14 drink / week)

19. Collagen vascular disease

Nearly > 50% cases with placental abruption has no identifiable risk factor. [47]

Age

Incidence of abruption increases with maternal age. In the FASTER trial (first and second Trimester evaluation of Risk Trial) women older than 40 years were 2.3 times more likely to experience abruption compared with those 35 yrs (or) younger. (Cleary- Goldman and co- workers 2005).[49]

Advanced maternal age does not appear to be significant etiological factor.[14,21,25]

Parity

Pritchard and colleagues (1991) - reported the incidence to be higher in woman of greater parity [14,21,49]. The incidence is four times higher and the risk increases after fifth gravida if pregnancy occurs at shorter interval. [25]

Whereas Toohey & associates (1995) - did not find this**.** [15,49]

Race

Pritchard & co workers (1991) from Park land hospital reported that abruption was more common in African & American & Caucasian women (1 in 200) than Asian(1 in 300) or Latin- American woman(1 in 450).[49]

Familial factors :-

Raimussen & Irgens (2009) - If a woman had severe abruption, then the risk for her sister was doubled and the heritability risk was estimated to be 16 %. [49]

Hypertension

The most common condition associated with placental abruption is some type of hypertension –gestational hypertention, preeclampsia, chronic hypertention or combination of these.[7,49] In cases of abruptions that are severe enough to cause fetal death, 50% are due to hypertension [47]of which 25% are from chronic hypertension and 25% are from preeclampsia .[7] The relative risk of abruption is 3.8 for severe preeclampsia, 2.8 for chronic hypertention with superimposed preeclampsia**.** [25]Abruption was caused by increased blood pressure in 50% of cases. [27]

According to Prichard & co workers (1991) hypertension was apparent in approximately half of the women once the depleted intravascular compartment was adequately refilled. Sibai & co workers (1998) reported for the maternal fetal medicine units net work that 1.5% of pregnant women with chronic hypertension suffered placental abruption. [49]

Ananth & associates (2007) reported a 2.4 fold increased incidence of abruption with chronic hypertension. This was further increased if there was superimposed preeclampsia and fetal growth restriction. Zetterstrom & colleagues (2005) reported 2 fold increased incidence of abruption with chronic hypertension compared with normotensive women - an incidence of 1.1 vs 0.5%. [49]

Abdella and colleagues evaluated 265 cases of abruption and estimated an incidence of 1% in total obstetric population of which a quarter of case were complicated by hypertensive disorder**.**[14,22,49] According toHertzberg et al 1983 - preeclampsia , chronic

hypertensive disorder, eclampsia were found to have a 2%, 10% and 24% incidence of abruption respectively.[22] Pritch and et al (1970) found that 45% of their patients with abruption severe enough to kill the fetus had elevated Blood pressure. [49] According to Within & colleagues (1999) and Zefterstrom & co workers (2005)- the severity of hypertension does not necessarily correlate with the incidence of abruption. Observations from Magpie trial collaborative group2002 suggest that women with pre - eclampsia may have a reduced risk of abruption when treated with Mgso4. [49]

The pathophysiology of placental abruption in preeclamptic patients has been proposed to result from thrombotic lesion in the placental vasculature, leading to decidual necrosis, separation & hemorrhage. A vicious cycle then continues as the decidual hemorrhage results in further separation. This cycle may be aggravated by co existing hemostatic compromise.[22]

Patient with PIH had a 4 fold increased risk of still birth, neonatal death compared that those without PIH.[3,34] According to Munim and chadhury et al there was no difference in the grades of abruption , maternal & perinatal outcome between the hypertensive and normotensive women.[37]

Prematurely ruptured membrane & preterm delivery

There is increased incidence of abruption when the membrane ruptures before term. Major & colleagues (1995) reported that 5% of 756 women with ruptured membrane between 20 to 36 weeks developed an abruption. Kramer and co workers (1997) found an incidence of 3.1% in all women if membrane ruptured for longer than 24 hrs. [49] Ananth associates (2004) reported a 3 fold increased risk of abruption with prematurely ruptured membranes. This risk was further increased with infection.[49] Inflammation & infection may be the primary cause of placental abruption. Neutrophil infiltration of the fetal membranes and cervix as seen with

PROM and chorioamnionitis is associated with placental abruption.[47] Preterm premature rupture of membranes are 3 times more likely to have abruption especially those associated with oligohydromnios.[25]

Smoking

Studies from collaborative perinatal project linked cigarette smoking with increased risk of abruption (mira Ananth 1999, Naeye 1980). Ananth & colleagues (1999) found a two fold increased risk for abruption in smokers. This was up to 5 to 8 fold if smokers had chronic hypertention, severe preeclampsia or both.[25, 49] Similar findings have been reported by mortensen 2001, Hogberg (2007), kaminsky 2007 & all their associates[49] maternal smoking is a significant contributory factor to the development of placental infarcts and abruption [23,49]. Naeye reported an incidence of placental abruption is 1.69% in non smoker and 2.46% in smokers.[14] In smokers evidence of decidual necrosis at the edge of placenta was found. These findings represents the effect of smoking on uteroplacental blood flow and decidual integrity. [14]

smoking is an independent risk factor associated with a 90% increase in the risk of placental abruption. The risk increases with number of cigarettes smoked per day**.** [17]Mortality rate increases by 40% with each cigarette pack per day. [27]

Cocaine

Cocaine use increases abruption rate by 10%.[27] In the report from Bingol & colleagues (1987) of 50 women who abused cocaine during pregnancy, there were 8 still births caused by placental abruption. Addis & associates (2001) reported that placental abruption was more common in female who used cocaine than in those who did not. [25,49]

Thrombophilias:

The combination of thrombophilic factors like hyper homocystinemia, deficiency of protein c , protein s and antithrombin , genetic mutation of factor v leiden increases the risk of abruption by 3- 7 fold. [14,25,47,,29,51]

Factor V Leiden or prothrombin gene mutation are associated with placental abruption and infarction as well as preeclampsia (Kenny & colleagues 2009**).**[49] Inherited thrombophilias are associated with placental abruption (facchinetti et al 2003, prochazca et al 2003**).** [21] The strongest association are with the factor V leiden mutation, prothrombin promoter mutation, and hyperhomocystenemia. [21]Hyper homocystenemia results from a homozygous mutation in the gene coding for the enzyme Methyl tetrahydrofolate reductase.The elevated level of homocysteine can be normalized with the ingestion of folic acid. The risk is higher in women with more than one thrombophilias. [21]

The determination of the relationship between thrombophilia and abruptio placentae (decidual hemorrhage) is difficult because of the limited number of studies and confounding variables, including chronic hypertension, and cigarette smoking and cocaine use.

De Vries found that 9 out of 31 (29%) patients with abruption had a protein S deficiency, compared with their general population prevalence of 0.2–2%. The prevalence of Factor V Leiden , prothrombin gene mutation and protein S deficiency was in the ranges 22–30%, 18–20%, and 0–29%, respectively .[46]

Multiple gestation

1.2% risk of abruption in twins, 1.5% in triplets . [13,44,.]

Traumatic Abruption

Following external trauma, usually with motor vehicle accidents or physical violence placental separation may occur.[25,49] Traumatic abruption evolves within 24 hours**.**[27]

Studies from Parkland hospital 2% of placental abruption causing fetal death were due to trauma. Kettel (1988), stafford (1988), appropriately stressed that abruption can be caused by relatively minor trauma. Placental separation from trauma is likely to be caused by deformation of the elastic myometirum around the relatively inelastic placenta (Crosby and associates 1968). This may result from a deceleration injury. Reis & colleagues 2000 reported that abruption is more likely if the vehicle speed is 30 mph.Traumatic abruption is more likely to be concealed and generate higher intrauterine pressures associated coagulopathy. Pearlman & associates 1990 found that if contractions were fewer than every 10 min during 4 hours of electronic monitoring then the abruption is unlikely, female with frequent contraction had 20% abruption.[49] procedure like external cephalic version can cause abruption. The risk of abruption is 1-5% in minor injuries and 40 – 50% in major injuries**.** [25]

Leiomyomas

Fibroid uterus especially if located behind the placental implantation site, predispose to abruption. [25,49] Rice and associates 1989 reported that 8 out of 14 female with retroplacental myoma had abruption whereas abruption developed in only 2/79 female whose leiomyona were not retroplacental . submucous fibroid volume > 200 cm3 cause abruption.[49]

Uterine anomalies

The risk of placental abruption increased 8 times with uterine malformation like mullerian duct anomalies. [25,49]

Genetic influence

Recently there has been some evidence suggestive of a genetic influence in the pathogenesis of placental abruption. A review and meta analysis by zdoukopoulos and co workers found a positive association for the Arg506Gln and F2G20210A polymorphisms.[14]

Previous history of abruption

The risk of recurrence is approximately 17% for patients with one abruption and as high as 25% for patients who have had more than one episode**.** [14,21] Abruption is 10 to 15 times more common in subsequent pregnancies. [25]

Prichard & co workers (1970) identified 12% recurrence in subsequent pregnancy. Tikkanen & colleagues (2006) found that of 114 parous women who experienced an abruption 9% had a prior abruption. According to Furuhashi & colleagues (2002) the recurrence rate of abruption was 22% and 4 to 6 recurrence were at a gestational age 1 to 3 weeks earlier than the previous abruption. Rasmussen & Iregns (2009) reported an odds ratio of 6.5 for recurrent mild abruption & 11.5 for recurrent severe abruption. Women who had 2 severe placental abruption, the risk become 50 fold. Teivonn & colleagues (2002) reported that antipartum fetal testing is usually not predictive of future recurrent abruption[49].

Dug off & colleagues (2004) observed an association between abnormally elevated maternal serum markers like serum alpha fetoprotein in the 1st trimester & subsequent abruption. Ananth (2006) and weiss(2004) and their associates have correlated first and second trimester bleeding with placental abruption in third trimester.[29,49]

Pathology

Abruption is initiated by hemorrhage into the deciduas basalis,[21,27,49] The decidua then splits leaving thin layer adhered to the myometrium. This process consist of decidual hematoma that leads to separation compression and ultimate destruction of the placenta adjacent to it and separates the placenta from the maternal vascular system , causing impairment in fetal oxygenation and nutrition.[16] Nath and colleagues 2007 found histological evidence of inflammation more commonly in cases of placental abruption. They suggest the inflammation and infection may be a contributor to causual pathway. [49]

In its early stage, there may be no clinical symptom but circumscribed depression on the placental maternal surface was discovered upon examination of the freshly delivered placenta. Circumscribed depression usually measures a few cm in diameter & is covered by dark, clotted blood.[25,49]According to Benirechke & kaudmann (2000) The age of retro placental clot cannot be determined exactly. [49]

In some instances, a decidual spiral artery ruptures to cause a retro placental hematoma. [47] When it expands it disrupts more placenta to separate. The area of separation rapidly becomes more extensive & reaches the margin of the placenta. Because uterus is still distended by the product of conception, it is unable to contract sufficiently to compress the torn vessels that supply the placental site. The escaping blood may dissect the membranes from uterine wall & eventually appear externally or may be completely retained within the uterus.[49]

In the full blown case of concealed hemorrhage, the uterus size is greater than the period of gestation. The blood may dissect into the myometrium towards the serosa resulting in couvelaire uterus (uteroplacental apoplexy). It is characteristically shows ecchymoses on its serous surface. The muscle bundles of uterine wall are heavily infiltrated with extravasated blood and edema fluid resulting in an atonic uterus. [25]

The laceration in the decidual layers allows free communication between intradecidual space and the maternal circulation of the placenta, in which tissue substances including thromboplastin from the decidua enters directly into the maternal circulation resulting in coagulopathy. [25]

Fetomaternal hemorrhage

Bleeding with placental abruption is almost always maternal, because the separation is within the maternal decidua. In non traumatic placental abruption, Fetal to maternal

hemorrhage is 20% and the volume of fetal blood was less than 10ml (stettler and colleagues 1992). Significant fetal bleeding is much more likely with traumatic abruption . In this circumstance, fetal bleeding results from a tear or fracture in the placenta rather than from the placental separation itself. Stettler and colleagues (1992) reported that there was fetal to maternal hemorrhage of 80 to 100ml in 3out of 8 cases of traumatic placental abruption.[49]

Pearlman and associates (1990) documented fetal bleeding that averaged 12ml in a third of women with traumatic abruption. Good win and Breen (1990) 90% of traumatic abruption the volume of fetal bleeding is less than 15ml. Muench and colleagues (2004) reported a 20 fold increased risk of associated uterine contraction & preterm labor if there is evidence of fetomaternal bleed**.** [49]

Laboratory tests that identify the fetal cells in the maternal circulation includes Ogita, Londersloot, Apt, or Kleihauer—Bentke tests .[6, 17]

In Rh negative mother, placental abruption may have had massive fetal- maternal transfusion, requiring larger than usual inj.anti D dosage in order to avoid alloimmunisation. [49] But according to Keith & Craig et al, the incidence of fetal-to-maternal hemorrhage does not appear to be increased in pregnancies that are complicated by third-trimester bleeding when compared to noncomplicated control subjects or to other obstetrically complicated pregnancies. This information would suggest that the routine administration of additional anti-D immune globulin (beyond the current recommended protocol) to women who are Rh D-negative whose pregnancies are complicated by third-trimester bleeding is not indicated. (Am J Obstet Gynecol 2003;188:1615-21.)

Clinical diagnosis

The classical clinical presentation of abruption includes vaginal bleeding , abdominal pain, uterine tenderness,hypertonic uterus,fetal demise which does not occur frequently. The

signs and symptoms of placental abruption can vary considerably. Majority of patients have atleast one of these signs, but occasionally none of them will present**.** [7.21] Common presentation of abruption is mild vaginal bleeding, no uterine tenderness, no coagulopathy.[7,21] Nearly 50% of patients with placental abruption are in established labour.[14] some patients may have nausea, anxiety,thirst,restlessness,and feeling of faintness.[14] However, about 30% of placental separations are small with few or no symptoms and are identified only after inspection of the placenta at delivery. [18]

Symptom	**Occurrence (%)** [14,49]
Vaginal bleeding	80
Abdominal or back pain	67
Uterine hypertonus	17
Fetal distress	60
Fetal demise	15

Uterine hypertonus is defined as more than 5 contraction in10 min.[14] Indeed no vaginal bleeding observed in 25-35% patients [49]this vaginal bleeding is characteristically dark and non clotting.[14]

Sometimes external bleeding can be profuse placental separation may not be so extensive as to compromise the fetus. Rarely there may be no external bleeding but the placenta may be completely sheared off and the fetus is dead as a direct consequence.[49]

The size of the uterus ,the height of fundus and the abdominal girth all should be recorded as it may be disproportionately larger than that expected for the period of gestation. [25]

Once the placenta previa is ruled out,vaginal examination was performed to confirm the fetal presentation and to assess the prospect of expeditious vaginal delivery**.**[25]

Classification:

Sher 1978 proposed a clinical grading system for placenta abruption**.** [21,27]

Grade I – the diagnosis is made retrospectively, retroplacental clot volume of approximately 150 ml and none had more than 500ml. Fetuses are usually not at risk and favorable perinatal outcome occurs.

Grade II – The classic features of abruptio placenta present and the fetus is alive. 92% of patients have abnormal fetal heart patterns and perinatal mortality is high.

Grade III - Incorporates the features of grade II but fetal demise is confirmed. It is further subdivided based on the presence (B) or absence (A) of coagulopathy. Virtually all maternal mortalities in association with abruptio placenta occur in grade III patients.

USG in placental abruption

Ultrasound is not a sensitive method of diagnosing placental abruption. [14]

The sensitivity and specificity of ultrasound in the diagnosis of placental abruption were24% and 96%, respectively So, while ultrasound is very helpful in ruling out other causes of third trimester bleeding, it lacks the sensitivity needed to reliably detect placental abruption. [47]

Sonography infrequently confirms the diagnosis of placental abruption atleast acutely, because the placenta and fresh clot have similar sonographic appearances. Sholl 1987 used sonography and confirmed the clinical diagnosis in only 25% of women. Glantz and purnell 2002 reported 24% sensitivity.[49]The diagnosis missed on ultrasound in 50 % women with clinical signs suggestive of abruption. [25]Importantly negative findings with sonogrpahic examination do not exclude placental abruption.[49]

The most important USG finding in abruption is a globular placenta with a diameter of at least 6cm [21]USG is also useful to asses fetal presentation, estimated weight& fetal well being.[21]

Initially the hemorrhage may be seen as hyperechoic or isoechoic in comparison to the placenta but as the hematoma starts resolving it becomes hypoecohic after a week and sonolucent after two weeks. [14,47]

Differential diagnosis

Other causes of third trimester bleeding includes placenta previa, cervicitis,cervical erosion, endocervical polyp, cancer cervix, vaginal/ vulval and cervical varicocities,vaginal infection, foreign bodies, genital laceration,bloody show, vasa previa &marginal placental separation **.** [21,49]

Milder are more common forms of abruption may be difficult to recognize with certainty and the diagnosis is often made by exclusion. Unfortunately, neither laboratory test nor diagnostic methods are available to detect lesser degrees of placental separation accurately. With severe placental abruption the diagnosis is generally obvious. A woman presented with vaginal bleeding with live fetus. It often becomes necessary to exclude placenta previa and other causes of bleeding by clinical and sonographic evaluation. [15]In about 10% of cases placenta previa was associated with placental abruption.[17]

Clinically, it has long been taught that painful uterine bleeding signifies placental abruption, where as painless uterine bleeding is indicative of placenta previa. Labour accompanying previa may cause pain suggestive of placental abruption. Pain from abruption may mimic labour or it may be painless especially with a posterior placenta. At times, the cause of the vaginal bleeding remains obscure even after delivery.[49]

MATERNAL COMPLICATIONS

The most serious maternal complications in abruptio placenta are hypovolemic shock, acute renal failure, disseminated intravascular coagulation, couvelaire uterus, postpartum uterine atony, amniotic fluid embolism, massive fetomaternal hemorrhage & rarely sheehan syndrome.[49]

Hypovolemic shock

Hypovolemic shock is directly due to maternal blood loss. Tritchard and Brekken 1967 has proved that in 141 women with abruption so severe as to kill the fetus, the blood loss is often amounted to at least half of the pregnant blood volume conversely neither hypotension nor anemia is obligatory even with extreme concealed hemorrhage. It was once held that the shock sometimes seen with placental abruption was disproportionate to the amount of hemorrhage. [49]

Consumptive coagulopathy:

DIC has many etiologies including sepsis, giant hemangiomas& malignancies. This syndrome occurs frequently in obsetetric conditions such as abruptio placenta, amniotic fluid embolism & prolonged fetal death inutero.[49]

Abruption is one of the most common cause of clinically significant consumptive coagulopathy in obstetrics.[49] DIC was first reported to occur in association with placental abruption by De Lee in 1901[47]

The incidence of DIC is 35% to 38% and it occurs mainly in the severe form of abruption (grade 3). [14,47] DIC occurs in approximately 13% of patient with concealed bleeding' [21] because in concealed hemorrhage intrauterine pressure is high therefore forcing more thromboplasin into the maternal venous system.[49] It is usually limited to patients with abruption severe enough to cause fetal demise.[21] In approximately a third of women with an

abruption severe enough to kill the fetus, there are measurable changes in coagulation factors[49]. DIC usually persists for 12hours. [27]

Placental thromboplastin enters the maternal circulation and incites intravascular coagulation [21,49] and other features of the amniotic fluid embolism syndrome. The major mechanism is activations of intravascular coagulation with varying degree of defibrination. procoagulants are also consumed in the retroplacental clots, although the amount recovered are insufficient to account for all the missing fibrinogen (Pritchard and brekken 1967).

Bonnar and coworkers 1968 have observed that the levels of fibrin degradation products are higher in peripheral blood than blood contained in the uterine cavity, the reverse would be anticipated in the absence of significant intravascular coagulation.[21]

An important consequence of intravascular coagulation is the activation of plasminogen to plasmin which lyses fibrin microemboli to maintain microcirculatory patency. Overt thrombocytopenia may or may not accompany severe hypofibrinogenemia initially but become common after repeated blood transfusion.[49,21]

In DIC there is clinically significant hypofibrinogenemia(plasma levels of fibrin less than150mg/dl) along with elevated levels of fibrin degradation products and D-dimers & decreased coagulation factors. [21]

DIC may cause severe hemorrhage , renal failure, and death.[14]

Renal failure:

Acute renal failure may be seen with severe placental abruption. It is more common if treatment of hypovolemia is delayed (or) incomplete. [14,21] Kuklina & Coworker (2009) reported that abruption contributes significantly to the increasing incidence of obstetric related acute kidney injury. Drakeley and Colleagues(2002) described out of 72 pregnant women with acute renal failure a third had suffered an abruption.[21] According to Lind

Heimer and associates (2007) acute cortical necrosis,when it occurs in pregnancy is usually caused by placental abruption. In older reports by Grunfeld and pertuieset(1987), a third of women with this lesion had suffered an abruption.[21]

Most cases of acute kidney injury are reversible. Oliguria from inadequate renal perfusion that is observed in these circumstances is responsive to vigorous intraveneous fluid and blood infusion. Seriously impaired renal perfusion is the consequence of massive haemorrhage. Even when abruption is complicated by severe intravascular coagulation, prompt and vigorous treatment of hemorrhage with blood and crystalloid solution prevents clinically significant renal dysfunction.[21]

Because preeclampsia frequently coexists with placental abruption, renal vasospasm and hypoperfusion are likely intensified (hauth and Cunningham 1999). Even without preeclampsia, proteinuria is initially common especially with more severe forms of placental abruption. It usually clears soon after delivery.

Post partum hemorrhage

PPH can result from coagulation failure or from couvelaire uterus in which severe bleeding occur into myometrium and impairs the ability to contract.[14] Postpartum hemorrhage secondary to uterine atony is associated with abruption. [47]

Couvelaire uterus:

There is widespread extravasation of blood into the uterine musculature and beneath the utrine Serosa first described by couvelaire in the early 1900s as uteroplacental apoplexy. It is now termed couvelaire uterus. Such effusion of blood are also seen beneath the tubal serosa, between the leaves of the broad ligament, in the substance of the ovaries and free in the peritoneal cavity. These myometrial hemorrhages, seldom interfere with myometrial contraction to cause atony and they are not an indication for hysterectomy.[18,49] But some authors say that it interferes with uterine contraction so it cause post partum hemorrhage.[14, 24]

Sheehan Syndrome:

Rarely severe intrapartum or early postpartum hemorrhage is followed by pituitary failure. It is characterized by failure of lactation, amenorrhoea, breast atrophy, loss of pubic and axillary hair, hypothyroid ,adrenal cortical insufficiency[15,17]**.**The exact pathogenesis is not well understood, such endocrine abnormality develop infrequently even in women who hemorrhage severely. There may be varying degree of anterior pituitary necrosis and impaired secretion of one or more tropic hormone. it is diagnosed by MRI. [15]

Maternal mortality

Maternal mortality rate is approximately 1%[14]. It varies from 0.5% to 5.0%. [7]

The causes of maternal mortality includes

DIC,

Hemorrhagic shock ,

Postpartum hemorrhage ,

Acute renal failure,

Puerperal sepsis. [7,25]

Severe hemorrhage is usually the major cause leading to maternal mortality [7,14,25]

Perinatal morbidity and mortality

Perinatal mortality associated with placental abruption was 119 per1000 live births [14,49] compared with 8.2 per 1000 among all other birth.

Fetal mortality occurs in about 35% of all clinically relevant antepartum placental abruption and it can be as high as 50% to 80% in cases of severe placental abruption.[7] In the presence of hypertension fetal mortality rate increases by 3 fold.[14] Extent of placental separation has a profound effect on still birth, although evident only among those with at least 50% separation.[25]

Prematurity accounts for 50% death in placental abruption.[12,14,47]The remaining perinatal mortality is associated with fetal hypoxia, and fetal growth restriction **.**[10,40,47] Risk

factors for perinatal death includes smoking, severe preeclampsia & small for gestational age fetus.[25]

Abdella and associates reported that 16% of mortality rate were within 4 weeks with most infants weighing less than 2500 grams.For babies weighing more than 2500 grams the reported survival rate was 98%.

Fetal complications includes prematurity, fetal growth restriction, respiratory distress syndrome, anaemia, hyperbilirubinemia.[17] Fetal growth restriction was reported in upto 80% of infants born before 36 weeks.[14,47]

The long term complication of abruption, according to Abdella and associates (1984) , 15% of infants had significant neurological deficit within first year of life. Matsuda and coworkers reported that 20% of infant had cerebral palsy. [49]In surviving infants after severe placental abruption , long term neurological sequele like cerebral palsy may be four times higher.[25,45]

Management :

Treatment for placental abruption depends upon the severity of abruption, maternal condition, gestational age, fetal condition. [14,49]

Expectant management in preterm pregnancy

If the diagnosis is uncertain and the fetus is alive without evidence of compromise, then close observation can be practiced in facilities capable of immediate intervention.[49]Expectant management is considered in cases of mild abruption occurring before 37 weeks.[6,14] The goal of expectant management is to prolong the pregnancy for improving fetal maturity and survival.[25,14] when the initial episode is small and self limiting and there is no acute (abnormal CTG finding or biophysical profile) or chronic (growth restriction, oligohydromnios,or abnormal umbilical artery Doppler recording) fetal compromise , no evidence supports the induction of labour. Induction of labour at term is

often advocated in such patients.[14] No evidence supports routine admission when there is no maternal or fetal compromise and no uterine contraction. The retroplacental clot was monitored with serial ultrasound. If the fetal condition deteriorates , delivery is expedited.[14]

When the fetus is preterm, prolonging pregnancy may prove beneficial. Bond and associates (1989) expectantly managed 43 women with placental abruption before 35 weeks, 31 of them were given tocolytic therapy. The mean time to delivery in all 43 was approximately 12 days, there were no stillbirths. Ceasarean section was performed in 75% of cases.[49]Towers and associates reported that mean increase in time from bleeding until delivery was 18.9 days.[14] The diagnosis to delivery interval has been reported to range between 7 to 34 +/- 24 days.[25]

Women with evidence of very early abruption frequently develop oligohydramnios with or without premature rupture of membrane. Elliott and associates (1998) reported that women with an abruption at a mean of 20 weeks also developed oligohydramnios. They were delivered at an average gestational age of 28 weeks. [49]

These fetus can be monitored with CTG but lack of ominous decelarations does not guarantee the safety of the intrauterine environments. The major disadvantage of expectant management is the placenta may further separate at any instant and seriously compromise or kill the fetus unless delivery is performed immediately. [49]

Tocolysis

If preterm pregnancy complicated by suspected abruption without fetal compromise tocolytic therapy can be beneficial for prolong the pregnancy, but clinically evident placental abruption should be considered as a contraindication to tocolytic therapy.[14,49]

Sholl (1987) and combs and coworkers (1992) provided data showing that tocolysis improved outcome in a highly selected group of preterm pregnancies complicated by partial abruption. According to Towers and coworkers (1999) perinatal mortality did not differ from

treated and non treated group. The major disadvantage of tocolysis reported by Hurd and Associates (1983) that abruption went unrecognized for dangerously long periods if tocolysis was initiated**.** [49]

Timing of delivery after severe placental abruption

When the fetus is dead (or) previable, There is no evidence that establishing an arbitrary time limit for delivery is necessary [21,49.] In the past there was a dictum that these patients should be delivered within 4-6 hours but with appropriate maintenance of the maternal status, the time period for vaginal delivery may be safely extended upto 24 hours.[21] Maternal outcome depends on the diligence with which adequate fluid and blood replacement therapy is pursued, rather than on the interval between abruption delivery[49.]According to Brame and Associates (1968), at the university of Virginia hospital, women with severe placental abruption who were transfused for 18 hours (or) more before delivery, experienced complications that were neither more numerous nor greater in severity than did the group in which delivery was accomplished sooner, the same has been proved by Principal and Brekken (1967) from the Parkland hospital. [49]

The relationship between admission delivery interval and neonantal outcome with a clinically overt placental abruption and fetal bradycardia was studied by Kayani and Collegues 2003, of the 22 neurologically intact survivors, 15 were delivered Within 20 minutes of the decision time. This suggests that the speed of response is an important factor in neonatal outcome. [49]A decision to delivery interval of ≤ 20 minutes is associated with a substantial reduction in neonatal morbidity and mortality in cases of fetal bradycardia[7] .

Mode of delivery:

The mode of delivery is dependent primarily on the condition of the mother and fetus.

Cesarean delivery

With a fetus of viable age, and if vaginal delivery is not imminent, then emergency caesarean delivery is done by most of clinicians [25,49].In grade 2 abruption with evidence of fetal nonreassuring testing – rapid delivery typically by cesarean is indicated[47]. If the infant is alive and the uterus is rigid the abruption is probably large but less than 50% and the chances of fetal distress during labour are more than 90% so this baby should be delivered by immediate cesarean section unless there are no maternal shock or previable fetus.[21]

Other indications for caesarean delivery includes

- hemorrhage that is so brisk that it cannot be successfully managed even by vigorous blood replacement.
- Presence of other obstetrical complications that prevent vaginal delivery,
  failure of labour to progress. [25,49]
- If the uterus becomes hypertonic during labour or if the FHR monitoring becomes nonreassuring it must be assumed that abruption has extended and a ceasarean section should be done**.**[21]

The presence of a long, hard cervix is not an indication for cesarean section, in most patients the cervix will efface and dilate rapidly after oxytocin induction or vaginal prostaglandin administration[21].

Caesarean delivery at the time of severe consumptive coagulopathy have proved dangerous for the mother because the abdominal and uterine incisions are prone to bleed excessively when coagulation is impaired [49]**.**DIC by itself is not an indication for cesarean section but rather a strong contraindication. [21]

Evaluation of the patients hemostatic profile and preparation for transfusion is a must before preparing for caesarean section. Overt coagulopathy when abruption is not associated

with fetal demise is extremely rare. However coagulopathy and bleeding may develop during or immediately after the surgical intervention. [21]

Vaginal delivery

If placental separation is so severe, that the fetus had died then vaginal delivery is usually preferred [14,21,25,49]. In severe abruption (grade 3, fetal demise, often withDIC) – vaginal delivery is indicated. [47]

In most cases, for mild abruption (grade 1, no evidence of maternal or fetal compromise) – vaginal delivery is indicated and studies of women with mild (or grade 1) abruptions, mothers who delivered vaginally had a similar perinatal mortality rate to mothers who had a cesarean delivery. [47]If the uterus is not tense /tender the pregnancy should be interrupted by induction of labour. In these cases the abruption will probably not be greater than 25% and the chances of significant coagulopathy are extremely low and the prospects for a vaginal delivery with a favourable outcome are excellent. In the absence of uterine rigidity, fetal distress, and obstetric contraindication for vaginal delivery. The large majority of the patients should have a vaginal delivery. [21]

Labour

With extensive placental Abruption, the uterus is persistently hypertonic. The baseline intra amniotic pressure may be 50 mm Hg or higher with rhythmic increases up to 75 to 100 mm Hg [21,49] **.** Because of persistent hypertonus, it may be difficult at times to determine by palpation whether the uterus is contracting and relaxing to any degree.

Amniotomy

Rupture of the membranes as early as possible has long been championed in the management of placental abruption. The benefits of amniotomy includes

1. Diminished amnionic fluid volume might allow better spiral artery compression and decreases the bleeding from the implantation site. [49]

2. Reduces the entry of thromboplastin into the maternal circulation. [49]

3. If the fetus is reasonably mature, rupture of membrane may hasten the delivery. [14]

If the fetus is immature the intact sac may be more efficient in promoting cervical dilation than a small fetal parts poorly applied to the cervix [49] **.** Blood stained liquor on artificial rupture of membranes can be seen in some patients **.**[14,25]

Oxytocin

The rigidity of uterus or the presence of high intrauterine resting pressure should not deter the use of oxytocin [21], although baseline hypertonus characterizes myometrial function in most cases of severe placental abruption. If no rthythmic uterine contractions are superimposed, and there has been no previous uterine surgery then oxytocin is given in standard doses. Uterine stimulation to effect vaginal delivery usually provides benefits that override the risks**.**[25,49]

The use of oxytocin has been challenged on the basis that it might enhance the escape of thromboplastin into the maternal circulation and thereby initiate (or) enhance consumptive coagulopathy or amniotic fluid embolism syndrome. There is no evidence to support this fear. (clark and colleagues 1995; pritched and Brekken 1967).[49]

If there is no malpresentation, start on intravenous infusion of oxytocin; high doses of oxytocin may be required & monitoring of uterine acitivity is unreliable and the best index of progress of labour is cervical changes. [21]

Evaluation and replacement of blood loss

Pritchard and Brekken (1967) demonstrated that when abruptio placenta is severe enough to kill the fetus, the average intrapartum blood loss, mostly retroplacental is about 2500 ml[21]. All patients with severe abruptio placentae have significant blood loss and require aggressive measures to avoid progressive impairment in organ perfusion. Therefore transfusion of at least 2 units of Packed red blood cell should be instituted regardless of the initial vital signs and the initial hemoglobin and hematocrit value [21].

Immediately after admission while the initial assessment is performed, the intravascular volume should be expanded using RL solution. Expansion of volume with crystalloids is inefficient since only 250 ml of each 1000ml injected intravenously will remain in the intravascular compartment. Also in cases of severe bleeding in addition to volume expansion it is critical to improve oxygen – carrying capacity therefore transfusion of packed red blood cell should be started as soon as possible[21].

Inspite of significant blood loss if the patient was previously hypertensive the blood pressure may be normal, and the pulse may be normal until appropriate dehydration produces tachycardia. In patient with concealed hemorrhage, a vast under estimation of blood loss frequently occur. Therefore patients with abruptio placenta severe enough to cause fetal demise should be transfused despite normal hematocrit / hemoglobin values and norml vital signs.[21]

The aim of administration of red blood cells and intravenous fluids to women with severe abruption are to keep a hemotocrit of at least 30% and a urinary output of at least 30 ml / hour. By keeping the hematocrit at 30% or more. The patients oxygen carrying capacity is sustained. By maintaining the urinary output at 30 ml / hour or more, We can be relatively confident that the effective intravascular volume is being preserved and that acute tubular necrosis or bilateral cortical necrosis will be avoided. [21]

Management of coagulopathy

For the evaluation of the hemostatic system in patients with abruptio placenta, most - laboratories use a DIC profile which includes the following

Normal values for DIC profile [21]

Fibrinogen	150 - 600 mg/dl
Prothrombin time	11- 16 seconds
Partial thromboplastin time	22-37 seconds

Platelet count	12000-350000/ mm^3
D-dimer	0.5 mg/L
Fibrin degradation products	< 10 mg/dl (A)

Close to 40% of patients with severe abruptio placetae have plasma fibrinogen concentrations below 150 mg/dL, of which 28% has the fibrinogen level less than 100 mg/dL as a consequence of acute DIC. Patients with DIC also show prolonged partial thromboplastin time, prothrombin time, increased D-dimer concentration & Low platelet count. [21]

To minimize excessive blood loss at the time of delivery. It is safer to replace critically depleted coagulation factors, particularly platelets and fibrinogen. Patients with fibrinogen concentration of less than 100 mg/dl benefit from the administration of 10-20 units cryoprecipitate immediately before and during cesarean delivery.[21]

The coagulopathy will resolve within hours in the postpartum period with appropriate blood replacement and preservation of the intravascular volume. However, the uterus is occasionally a source of excessive bleeding because high levels of FDP(Fibrin degradation products) inhibit myometrial contractility[21].

Vaginal delivery can be managed in the presence of extremely depleted clotting components if episiotomy and unusual trauma are avoided, but in the presence of DIC cesarean section is contraindicated.[21]

Prevention of abruption;

There are no trials to assess any intervention for prevention of abruption or its complications.[47]

Smoking cessation counselling, avoidance of cocaine, and, if possible, avoidance of other risk factors can prevent abruptio placenta. [47]

Results and Observations

This study deals with maternal and perinatal outcome of abruption in normotensive and hypertensive patients .

Period of study:

From January 2010 to June 2011.

1 year and 6 months.

Total deliveries during this period.

Total no of deliveries in RMH – 18,685

Total no of normotensive deliveries – 16,774

Total no of hypertensive deliveries – 1.911

Total no abruption - 226

Normotensive patients with abruption – 126

Hypertensive patients with abruption -100

Incidence of abruption in RMH - 1.2% , 1 in 82 deliveries

Incidence of abruption in normotensive pts - 0.75 %

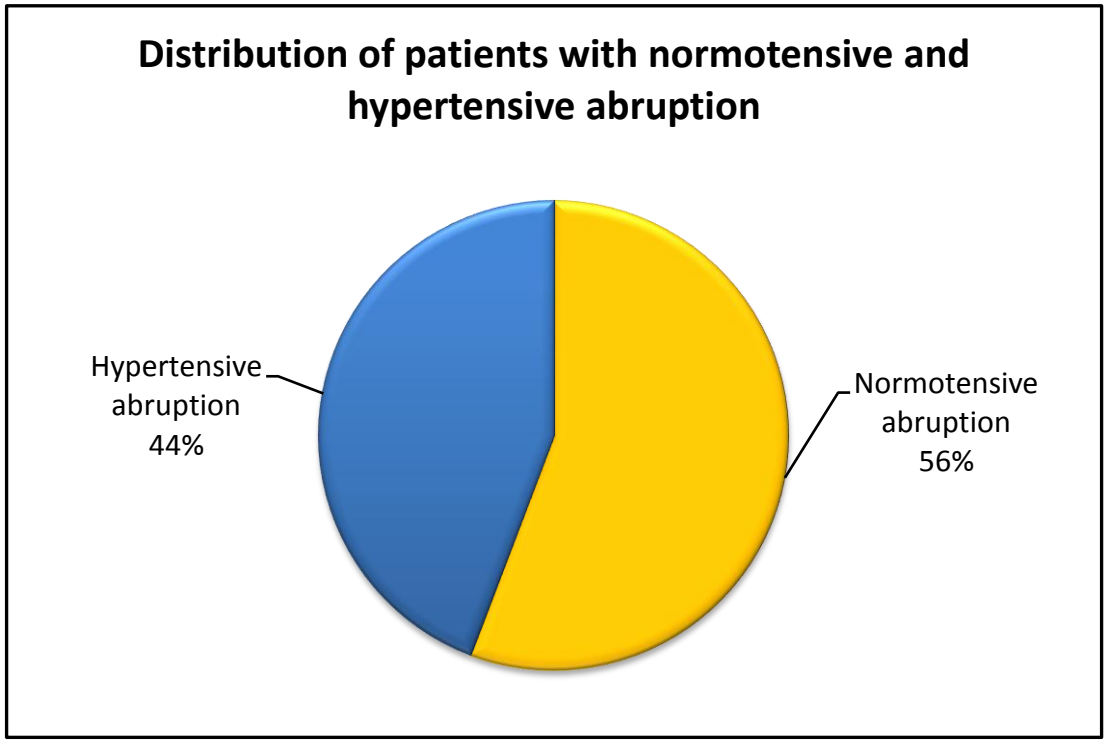
Incidence of abruption in hypertensive pts - 5.23 %

	Total no of deliveries	Toal no of abruption	incidence
total 226 cases	18,685	226	1.2%
normotensive	16,774	126	0.75%
hypertensive	1,911	100	5.23%

Total number of patients taken for the study = 226

Normotensive patients with abruption = 126

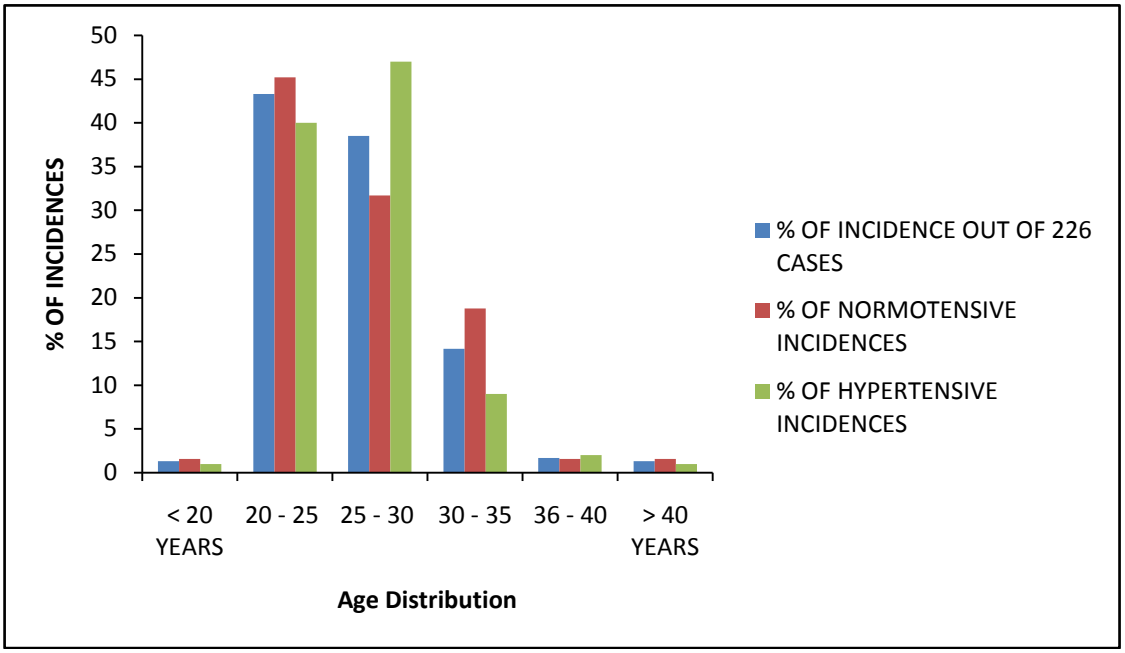
Hypertensive patients with abruption = 100



Distribution of abruption in relation to age group

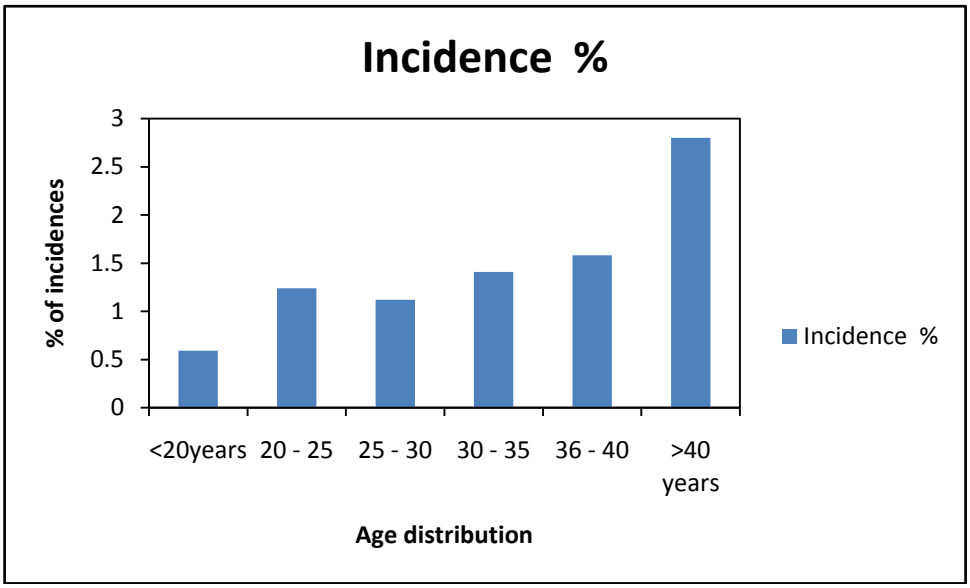
Age distribution	Total out of 226		Normotensive		Hypertensive	
	no	%	No	%	no	%
<20years	3	1.3	2	1.58	1	1
20 - 25	97	43.29	57	45.2	40	40
25 - 30	87	38.49	40	31.7	47	47
30 - 35	32	14.15	23	18.77	9	9
36 - 40	4	1.7	2	1.58	2	2
>40 years	3	1.3	2	1.58	1	1
	226		126		100	

Incidence of abruption is high in 20 – 30 years of age
in both normotensive and hypertensive patients.



Relationship between age group and incidence of abruption

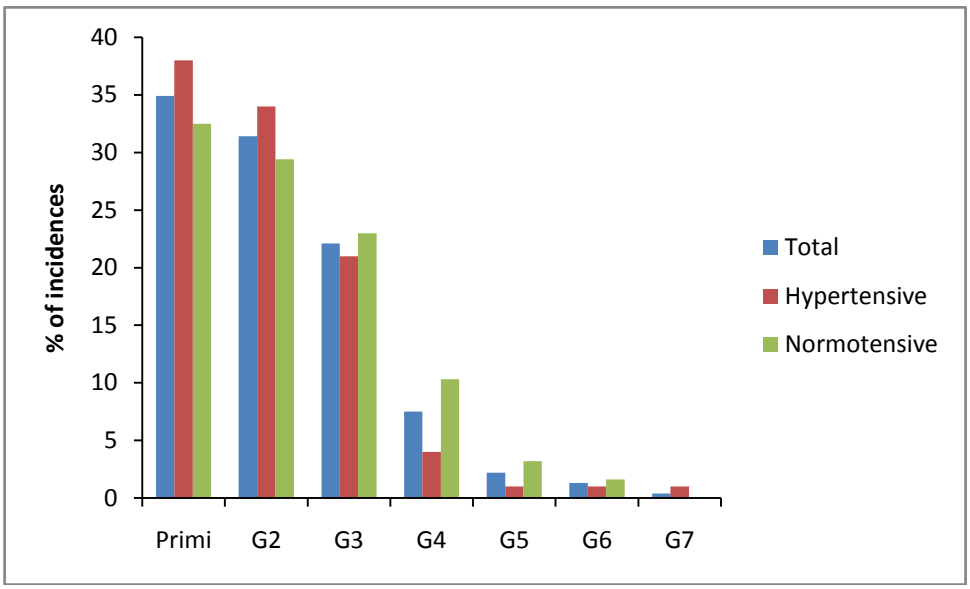
Age distribution	Total no of patients	Total no of cases with abruption	Incidence %
<20years	508	3	0.59
20 - 25	7820	97	1.24
25 - 30	7726	87	1.12
30 - 35	2269	32	1.41
36 - 40	253	4	1.58
>40 years	107	3	2.8



Incidence of abruption was increased after 40 years of age.

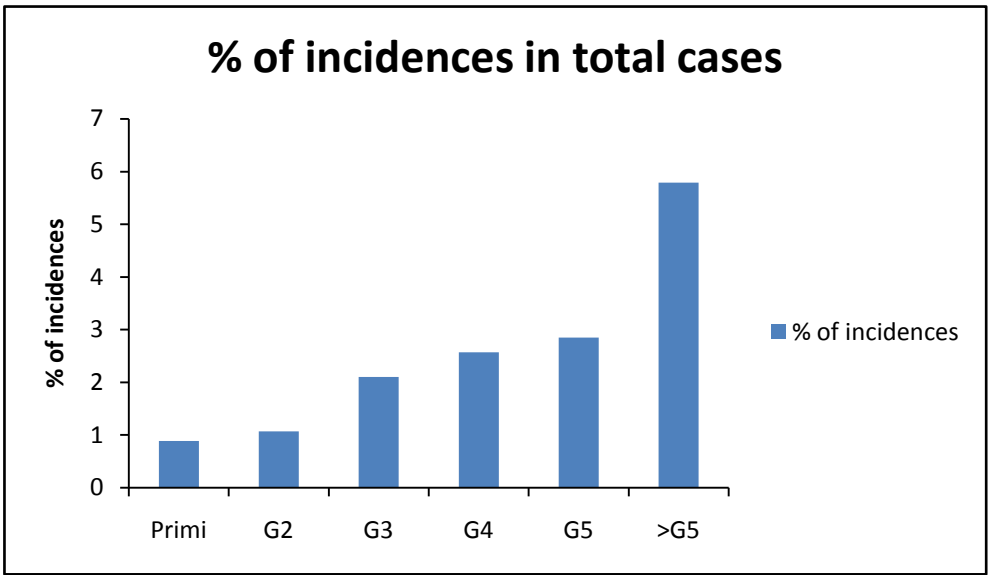
Distribution of patients according to parity and incidence

Parity	Total cases		Normotensive patients		Hypertensive patients	
	No	%	No	%	No	%
Primi	79	34.9	41	32.5	38	38
G2	71	31.4	37	29.4	34	34
G3	50	22.1	29	23.0	21	21
G4	17	7.5	13	10.3	4	4
G5	5	2.2	4	3.2	1	1
G6	3	1.3	2	1.6	1	1
G7	1	0.4	0	0	1	1



Distribution of patients according to parity and incidence

parity	No of patients	Abruption	
		No	Incidence
primi	8813	79	0.89 %
G2	6597	71	1.07 %
G3	2370	50	2.10 %
G4	661	17	2.57 %
G5	175	5	2.85 %
>G5	69	4	5.79 %



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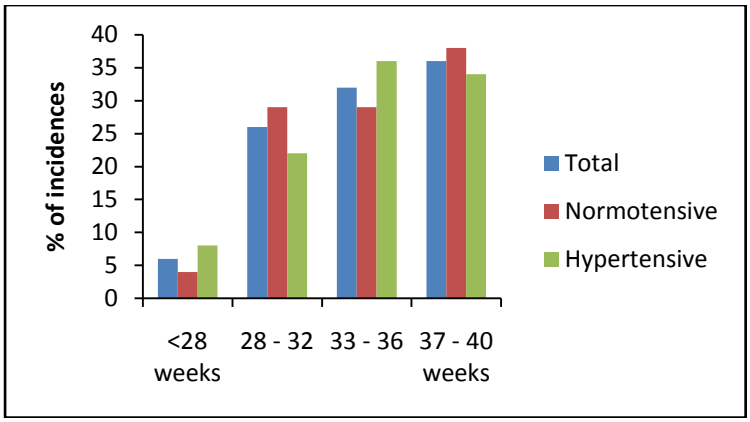
Incidence of abruption increases after 5th gravid.

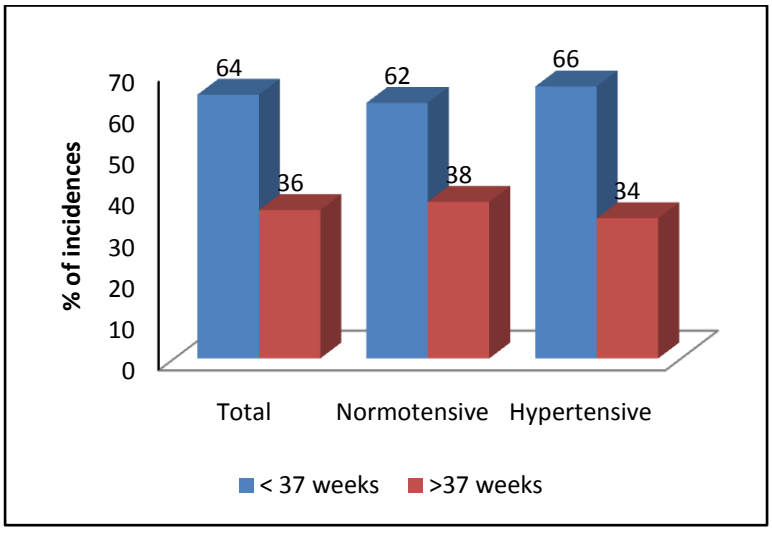
Gestational age distribution in relation to abruption

GA	Total		Normotensive		Hypertensive	
	no	%	no	%	no	%
<28	13	6	5	4	8	8
28 - 32	58	26	36	29	22	22
33 - 36	73	32	37	29	36	36
37 - 40	82	36	48	38	34	34



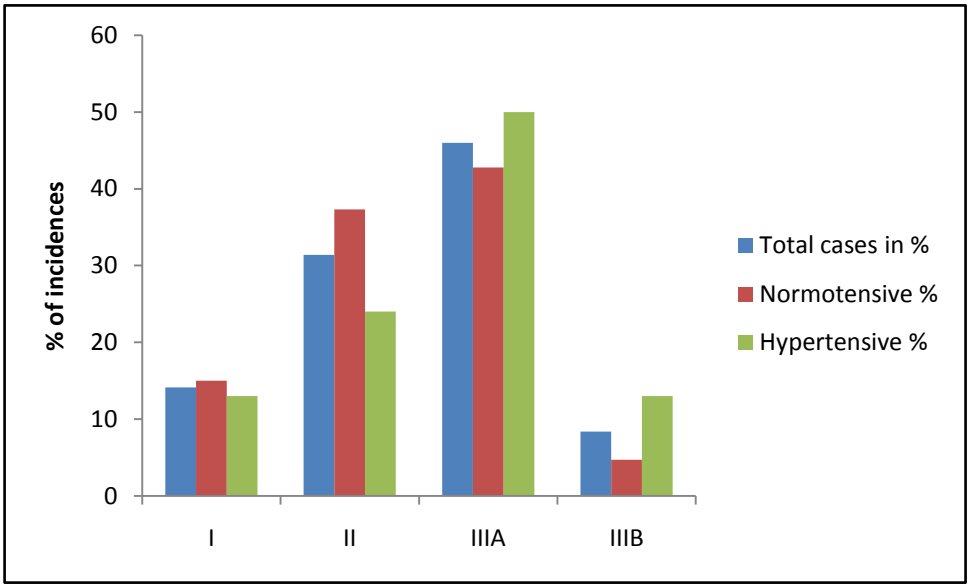
GA	Total	Normotensive	Hypertensive
< 37 weeks	64%	62%	66%
>37 weeks	36%	38%	34%

The incidence of preterm delivery is 64%. It is 62% in normotensive patients & 66% in hypertensive patients.



Abruption Grade Distribution

Abruption Grade	Total cases		Normotensive		Hypertensive	
	no	%	no	%	No	%
I	32	14.15	19	15	13	13
II	71	31.4	47	37.3	24	24
IIIA	104	46	54	42.8	50	50
IIIB	19	8.4	6	4.7	13	13



Antenatal care and incidence of abruption

Status	No of cases	Incidence %
Booked	73	32.3
unbooked	153	67.69

Abruption Grade	TOTAL	Booked	Unbooked
I	32	8	24
II	71	24	47
IIIA	104	35	69
IIIB	19	6	13

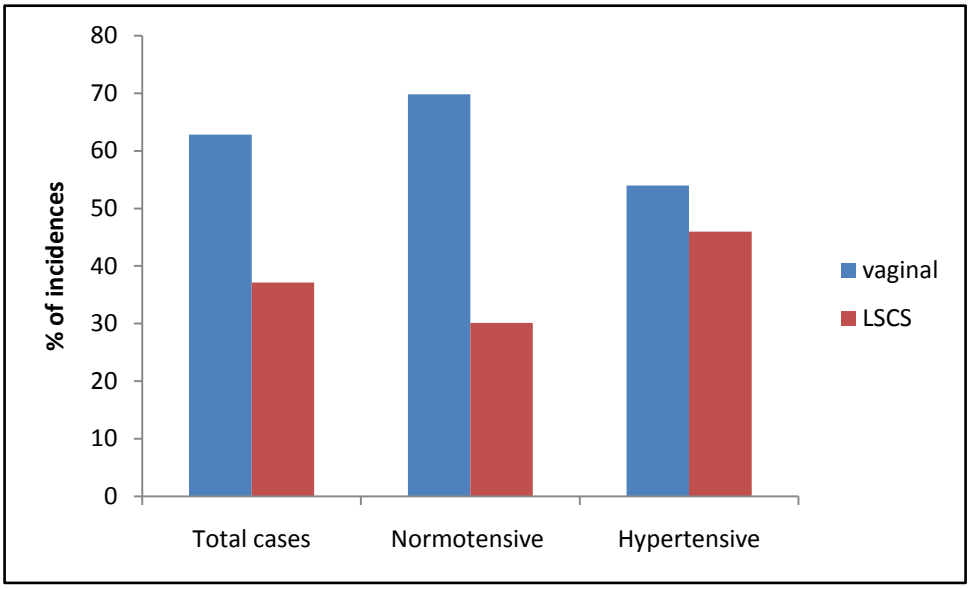
Incidence of abruption is high in unbooked cases.

Mode of delivery

Delivery	Total cases	Normotensive	Hypertensive
Labour Natural	136	84	52
LSCS	84	38	46
Asst.Breech	1	0	1
forceps	5	4	1

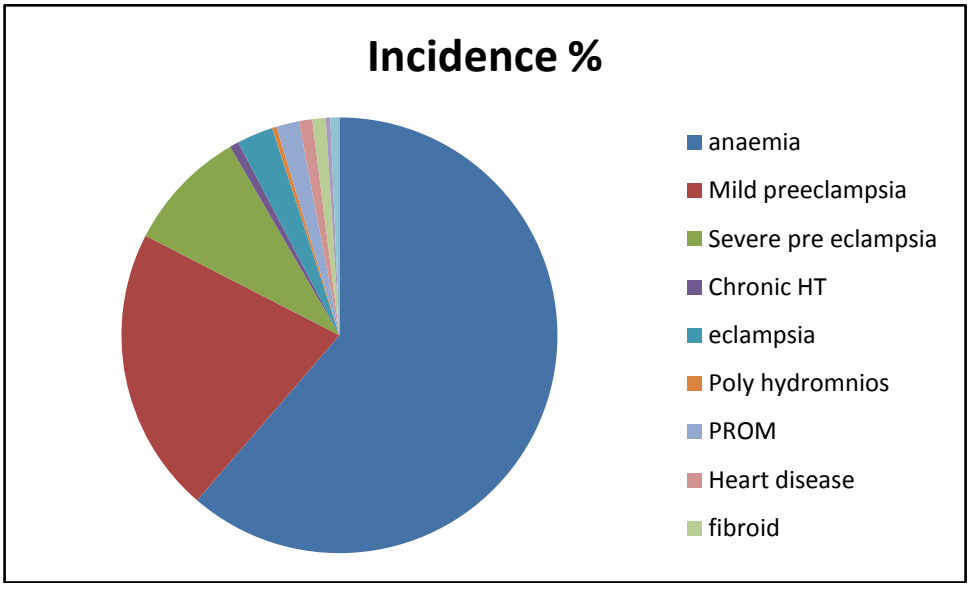
Delivery	Total cases %	Normotensive %	Hypertensive %
vaginal	62.83	69.84	54
LSCS	37.16	30.15	46

Incidence of vaginal delivery is high because of higher incidence of grade 3 abruption.



Maternal risk factors associated with abruption

complications	No of cases	Incidence %
Anaemia	182	80.53
Mild preeclampsia	63	27.87
Severe pre eclampsia	27	11.94
Chronic HT	2	0.88
Eclampsia	8	3.53
Poly hydromnios	1	0.44
PROM	5	2.21
Heart disease	3	1.3
Fibroid	3	1.3
Hypothyroid	1	0.44
GDM	2	0.88

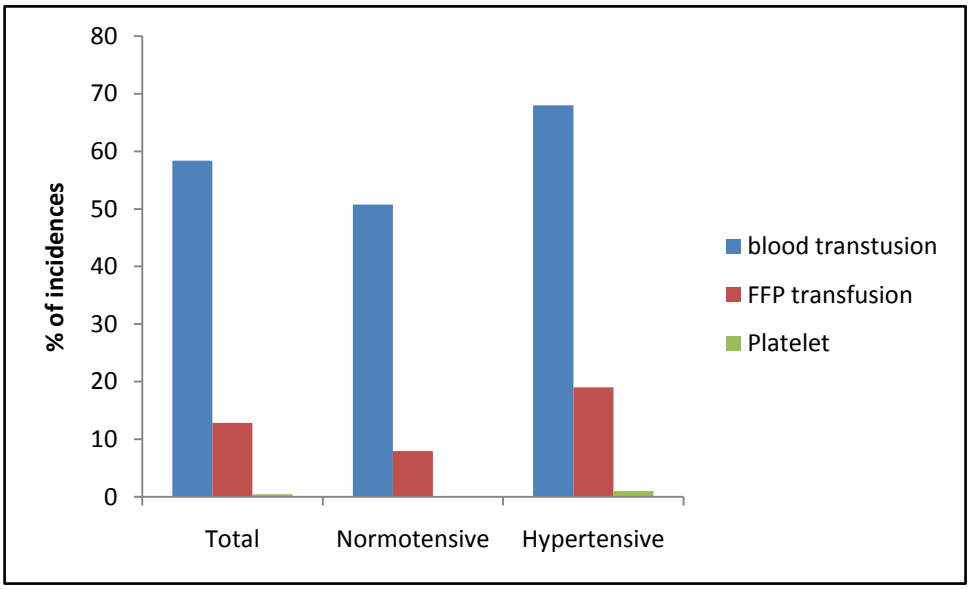


Maternal morbidity

	Total		Normotensive		Hypertensive	
	no	%	no	%	No	%
Hemorrhagic shock	51	22.56	28	22.22	25	25
DIC	19	8.4	6	4.76	13	13
ARF	3	1.3	1	0.79	2	2
Couvelaire uterus	16	7.07	7	5.55	9	9
Atonic PPH	2	0.88	2	1.58	0	0
Blood transfusion	162	71.68	74	58.73	88	88
Wound infection	6	2.6	2	1.58	4	4
HELLP Syndrome	3	1.3	-	-	3	3
Subtotal hysterectomy	2	0.88	2	1.58	-	-

Blood component transfusion

	Total 226 cases		Normotensive		Hypertensive	
	no	%	no	%	no	%
Blood transfusion	132	58.40	64	50.73	68	68
FFP transfusion	29	12.83	10	7.93	19	19
Platelet	1	0.44	0	-	1	1



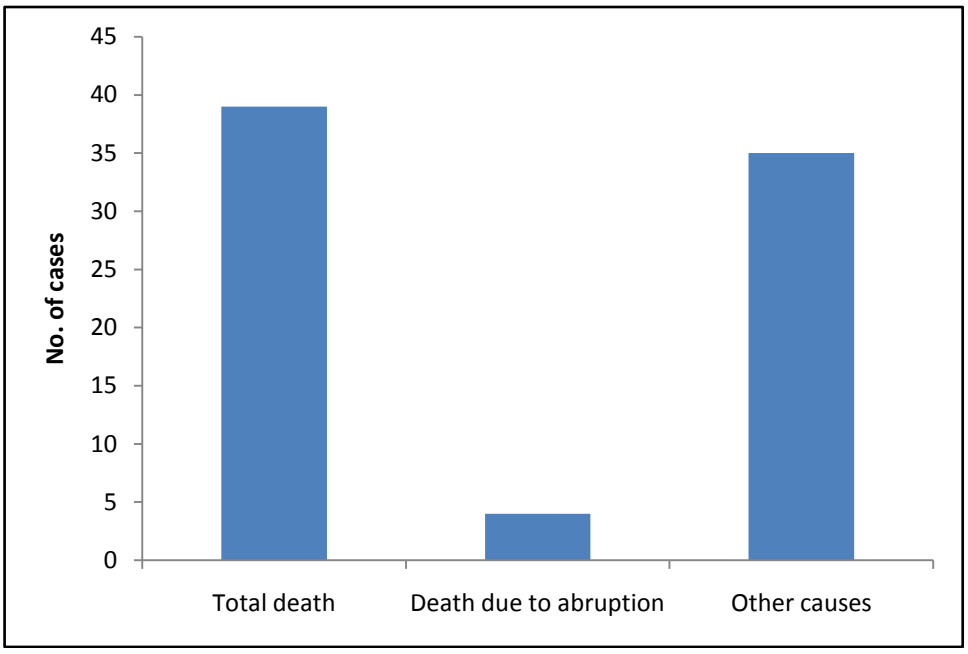
Maternal mortality due to abruption

Total no of maternal death during 2009 Jan to 2010 June -- 39

Maternal death due to abruption - 4

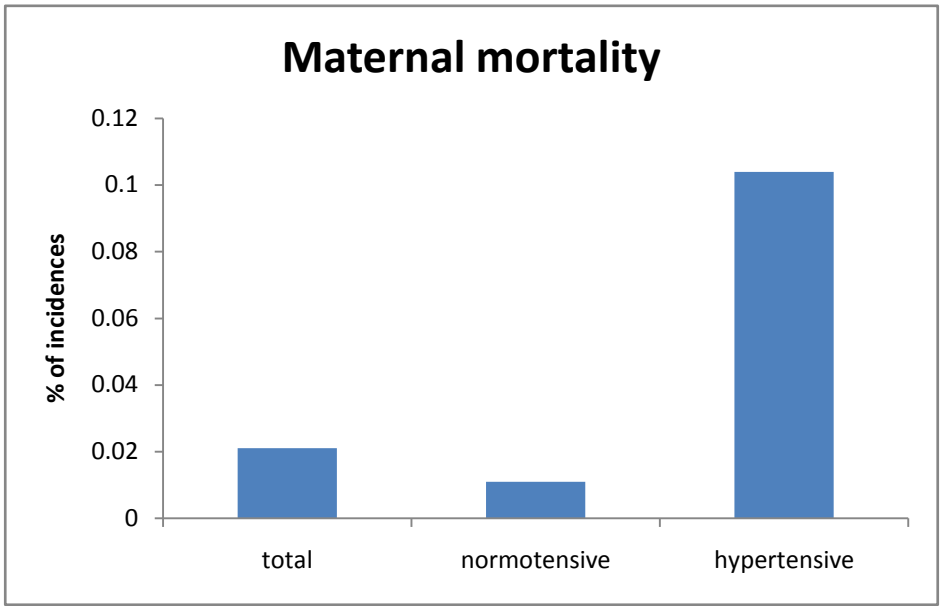
Abruption contributes to 10.25% of incidence.

Total death	Death due to abruption	Incidence
39	4	10.25 %



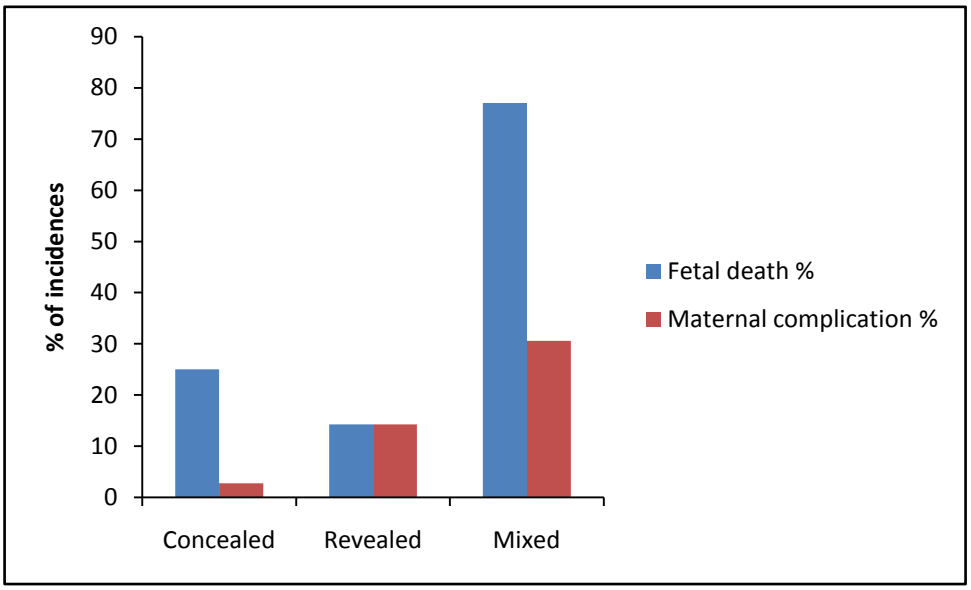
Maternal mortality due to abruption

	Total no of deliveries	No of death due to abruption	Incidence %
Total	18685	4	0.021
Normotensive	16774	2	0.011
Hypertensive	1911	2	0.104



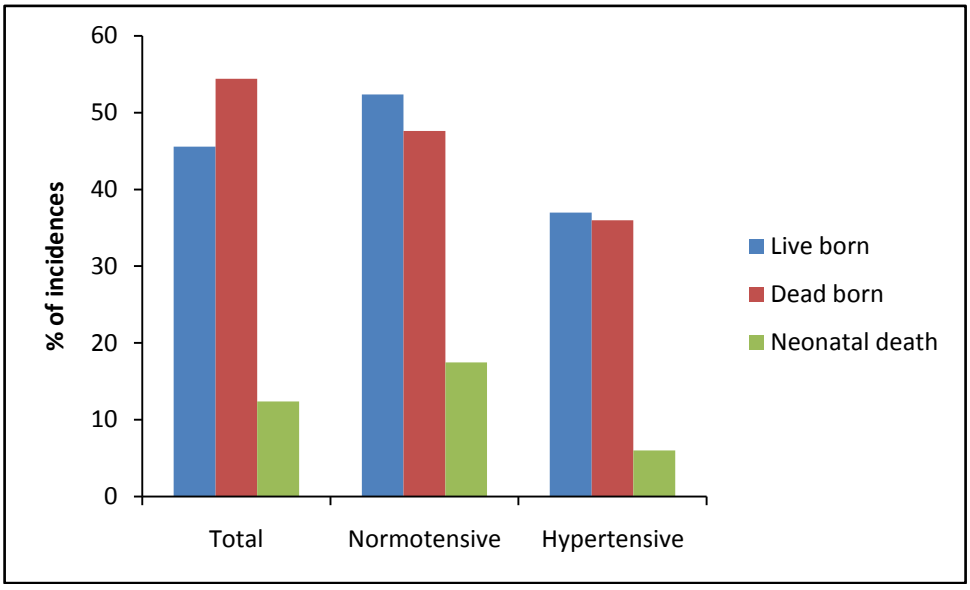
Type of abruption & maternal , perinatal outcome

	Total cases	Fetal death		Maternal complications	
		no	%	no	%
Concealed	36	9	25	1	2.78
Revealed	7	1	14.29	1	14.29
Mixed	183	141	77.05	56	30.60



Birth Distribution

	Total		Normotensive		Hypertensive	
	No	%	No	%	no	%
Live born	103	45.57	66	52.38	37	37
Dead born	123	54.42	60	47.61	63	63
Neonatal death	28	12.38	22	17.46	6	6



Perinatal mortality rate – 66.8

Perinatal mortality rate in normotensive pts – 65.07%

Perinatal mortality rate in hypertensive pts - 70%

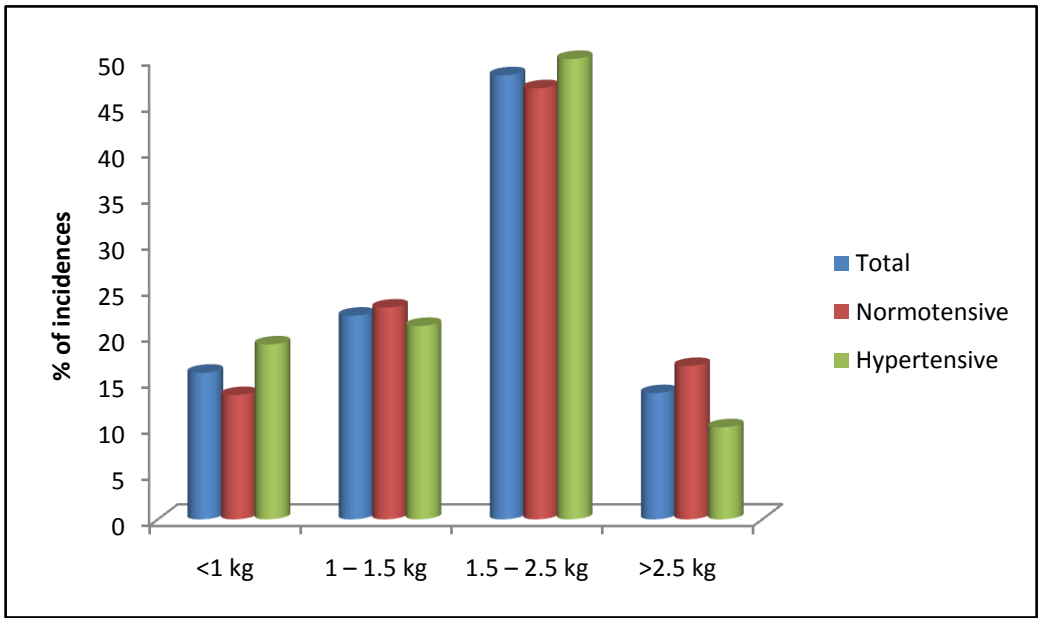
Birth Weight Distribution

Birth weight	Total		Normotensive		hypertensive	
	No	%	No	%	no	%
<1 kg	36	15.92	17	13.5	19	19
1 – 1.5 kg	50	22.12	29	23.0	21	21
1.5 – 2.5 kg	109	48.23	59	46.82	50	50
>2.5 kg	31	13.71	21	16.66	10	10

Percentage of low birth weight babies

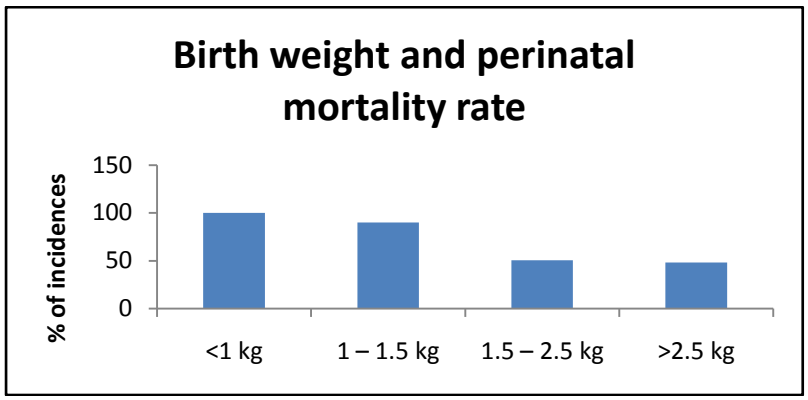
Birth weight	Total		Normotensive		Hypertensive	
	no	%	No	%	No	%
<2500 gms	195	86.28%	105	83.33%	90	90%
>2500 gms	31	13.71%	21	16.66%	10	10%

Incidence of low birth weight babies is 86.28%



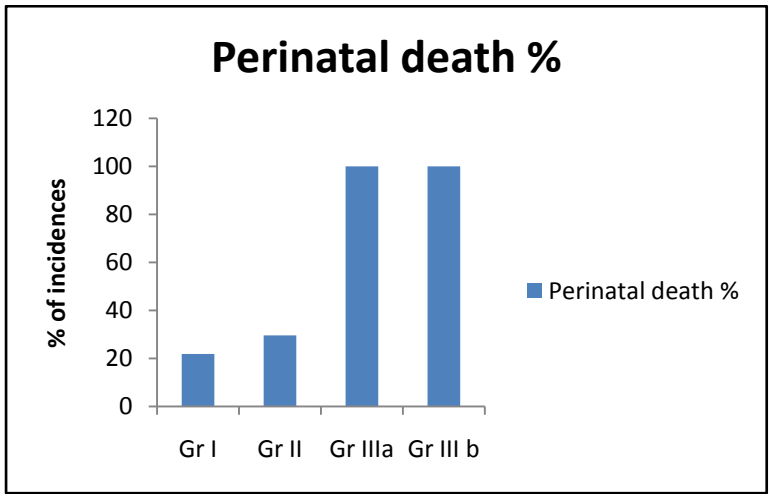
Birth weight and perinatal mortality rate

Birth weight	No of babies	No of perinatal death	incidence
<1 kg	36	36	**100%**
1 – 1.5 kg	50	45	**90%**
1.5 – 2.5 kg	109	55	**50.45%**
>2.5 kg	31	15	**48.38%**



Grade of abruption and perinatal mortality rate

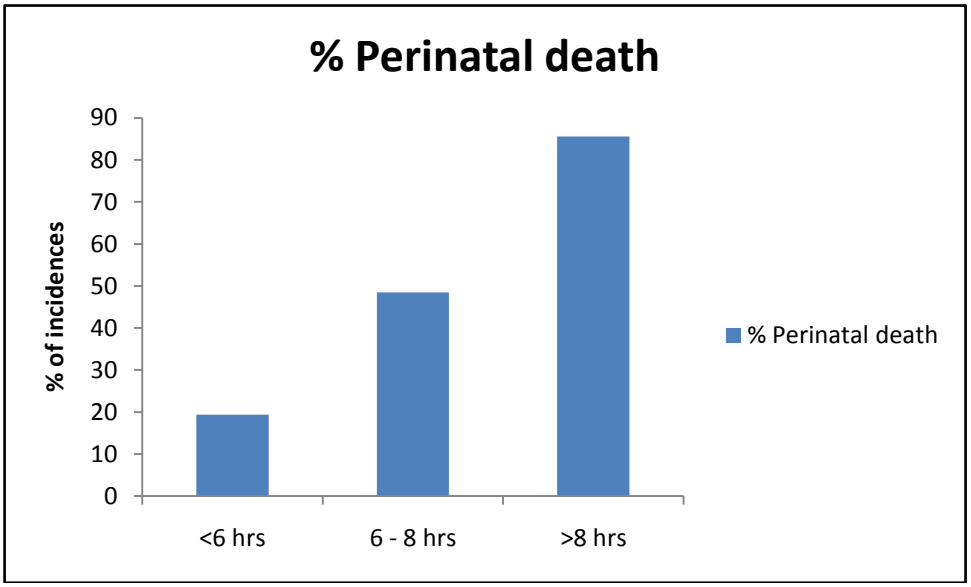
Grade	ABRUPTION		Perinatal death	
	No	percentage	no	percentage
Gr I	32	14.15 %	7	21.87%
Gr II	71	31.4 %	21	29.57%
Gr IIIa	104	46 %	104	100%
Gr III b	19	8.4 %	19	100%



Abruption delivery interval and perinatal mortality rate

Duration	% Perinatal death
<6 hrs	19
6 - 8 hrs	48
>8 hrs	86

The risk of perinatal death increases if the abruption delivery interval increase more than 8 hours.



Discussion

The incidence of abruption in present study is 1.2%, about 1 in 82deliveries.

The incidence of abruption in various studies

authour	Year	incidence
Morgan	1988-1992	0.7%
Sharief & manther	1988	2%
Dafallah & babikir	1997-2002	6.5%
Amornrath & sukcharoen	1995-2004	0.092%
Saadia	2003	2%
Iram sarwar	2003-2004	4.4%
Razia Mustafa abbasi	2007	1.87%
Nazli hossain	2008	3.75%

The incidence of abruption in our study is closely correlate with the incidence of the following studies Morgan 1988-1992, Razia Mustafa abbasi2007, sharief manthur 1998, saadia 2003.

The incidence of abruption in hypertensive patient in our study is 5.23% , that is 1 in 19 deliveries. In this study 44% of patients with abruption had elevated blood pressure.pritch et al 1970 found that 45% of their patients with abruption had high blood pressure.[21] According to leunen et al at university of leunen , Belgium 2003, hypertensive disorder were found in 75% of women with abruption [41] . The incidence of abruption in hypertensive patients ranged from 11.3% to 64% in the university of California.[34]

Advanced maternal age was not significantly associated with an increased incidence of abruption as observed by Dafallash& babikir et al [15,33]**.** The incidence of abruption is high in women with parity more than 5 but it is not statistically significant. There is no difference

in the incidence of abruption in relation to age & parity in normotensive and hypertensive women.

78% patient presented with complaints of vaginal bleeding and 65% of patient had abdominal pain ,54% of had fetal demise. 64 % of women in our study delivered before 37 weeks ,this correlates the study of Lubna amin, [14,33] . The mean gestational age is 34.25 weeks which is same in both normotensive and hypertensive women . The mean gestational age in other studies iram sarwaret al 33.81 $_{+}$ 3.64 weeks. [5] , amornrath et al 35.3+_3.4 weeks .[3]

The incidence of vaginal delivery is 62.83% and LSCS is 37.16%. As most of our patients had intrauterine fetal death or severe fetal bradycardia at the time of admission , these patients are delivered vaginally and we had less LSCS rate as observed by Iram sarwar [26]. The mode of delivery in other studies

study	**LSCS**	**Vaginal delivery**
Amornrath	84.5%	15.5%
Iram sarwar	30.2%	69.8%
Lubna amin	52.38%	47.61%
Nazli Hossain,	45%	50%
Tikkan et al	91%	9%

Hypertensive women were no more likely to be delivered before 37 weeks and to be delivered by LSCS as observed by morgan et al ,where as sharief et al reported that hypertensive women were more likely to be delivered before 37 weeks and more often by LSCS.[34]

In our study 71.68% patients require blood and blood component transfusion. Because all of our patients are belongs to low socio economic group, most of our patients are anaemic

and they cannot tolerate even minimal amount of blood loss these patients has to be transfused atleast one unit of blood.

Maternal mortality:

Out of 18685 deliveries we had 39 maternal deaths during the year January 2009 June 2010. Among 39 total maternal death, 4 were due to abruption and its complications. Abruption contributes to 10% of maternal deaths. Among 226 cases of abruption we had 4 maternal death . Incidence of maternal mortality is 1.76% in this study.Mortality in these cases was duo to DIC/irreversiblehemorrhagic shock, in one case due to associated antepartum eclampsia & pulmonary edema.

MMR in abruption according to various authours

Author	Year	Incidence
Nazli hossain	2008	2.46%
Razia Mustafa abbasi	2007	8.33%

Regarding the maternal morbidity in abruption , hemorrhagic shock accounts for 22.56% of cases. This is slightly higher than that reported by Amornrath and Sukcharoen (1995 to 2004)19.4%. 3 patient (1.3%) had acute renal failure which is less than 6.5% of incidence seen in Razia Mustafa Abbasi study during 2007. In our study the incidence of DIC is 8.4%. According to Razia Mustafa Abbasi (2007) and amornrath (2004) the incidence of DIC is 4.16% and 5.8% respectively. Couvelaire uterus is present in 7.07% of cases. The exact incidence of couvelaire uterus in abruption cannot be identified because laparotomy was not done in all abruption cases. Amornrath (2004) found that couvelaire uterus present in 16.5%. Subtotal hysterectomy was done in 2 patients for atonic PPH not responding to conservative management,among the two, one patient had torsion uterus about 180^0 with polyhydramnios (AFI 24).

Perinatal morbidity and mortality:

The perinatal mortality in our study is 66.81%,this is similar to PNMR of 67% reported by Nazili hossain 2008 [38] & PNMR of 67.9% reported by Iram sarwar 2004. [26] The perinatal mortality rate in hypertensive patient with abruption is 70% and normotensive patient with abruption is 65.07%, sharief and mather stated that PNMR of 38-72% in hypertensive patients and 32-58% in normotensive patients [34,39] . The early neonatal mortality in our study is 12.39 %.

Perinatal mortality rate is high in very low birth weight and extremely low birth weight babies, PNMR is 90% and 100% respectively. PNMR is only 48.38% in normal birth weight babies. PNMR is greatly influenced by the severity of abruption. PNMR is 100% in grade III abruption and it is 29.57%, 21.87% in grade II and grade I abruption respectively.the major cause of perinatal mortality in our study is prematurity.

The mean birth weight of neonates in our study is 1.8 kg +/_ 760 grams. it is 1.8kg in normotensive women and 1.7 kg for hypertensive women. According to amornrath & sukcharoen the mean birth weight is 2269+/- 737gms, [3] Nazli hossain the mean birth weight is 2400+/- 710 gms. [38] Regarding birth weight there was no great difference between the two groups and most of babies weighing 1 to 2.5 kg, as already reported by morgan et al 1992 , [36] & sharief and manther 1998.[34]

In this study perinatal outcome was not significantly different between hypertensive women and normotensive women experiencing abruption as already proved by the study conducted in Agakhan university , Pakistan [37].and university of California, Irvine.[36],amornrath et al , chulalongkorn university [3].

Placental abruption has various presentations and severity, therefore the maternal and perinatal outcomes are different. Because placental abruption is not only related to preterm birth but also to perinatal death and sequel from perinatal asphyxia, the study of the factors

contributed to these poor outcomes should be investigated for prevention of the serious sequel.

Summary

- During 18 months study period ,there was 18685 deliveries, of which we had 226 cases of abruption. 16774 normotensive patients , 1911 hypertensive patients.
- Incidence of abruption in our study is 1.2%. Incidence of abruption in normotensive patient is 0.75% and hypertensive patient is 5.23%. There is 7 fold increased incidence of abruption in hypertensive patients.
- There is no increased incidence of abruption in relation to maternal age and parity.
- 64% of patients delivered before 37 weeks. 62% of normoensive patients and 66% of hypertensive women delivered before 37 weeks. The P value is 0.604.The mean gestational age is 34+_2.5 weeeks.
- The incidence of caesarean section in our study is 37.16%.It is about 30.15% in normotensive patients and 46% in hypertensive patients. The P value is 0.729.
- Blood and blood component transfusion required in 71.68% of patients. 58.66%

  Of normotensive patients and 82% hypertensive patients required blood transfusion.
- Incidence of grade III abruption in normotensive patients is 47.5% , in hypertensive patient is 63%. The P value is 0.647.
- Maternal mortality in our study is 1.76% . In normotensive patients 1.58% and in hypertensive patients 2%.
- Among total 39 maternal deaths during the study period abruption contributes to 10.29% of cases.
- There is no difference in the abruption delivery interval (p value 0.999) and admission delivery interval (0.943) in normotensive and hypertensive patients with abruption.
- Perinatal mortality rate was 66.81%.

  Perinatal mortality in normotensive patient is 65.07%.

Perinatal mortality in hypertensive patient is 70%. The P value is 0.547.

- The most common reason for perinatal mortality is prematurity.
- Incidence of low birth weight babies is 86.28% .

  Incidence of low birth weight babies in normotensive cases 83.33%.

  Incidence of low birth weight babies in hypertensive cases 90%.

  The P value is 0.432.

Conclusion

In this present study hypertensive women experiences 7 fold increased incidence of abruption but the overall maternal and perinatal outcome was not significantly different from that of normotensive women experiencing abruptio placenta.. Hypertensive disorders of pregnancy can be identified and treated early in antenatal period so that abruption can be prevented and associated mortality& morbidity can be prevented. Perinatal mortality can be lowered by identification of risk factors, intensive fetomaternal monitoring , early detection and diagnosis of abruption and readiness to deliver by LSCS.

To conclude there is no doubt that abruptio placenta represents a potentially serious obstetric problem that tends to threaten fetal viability ,neonatal mortality and morbidity and maternal health and well being so that abruptio placenta should be managed in centres where there is advanced maternal and neonatal facilities are available.

BIBLIOGRAPHY

1. Abdella TN et al ,Relationship of hypertensive disorder to abruption placenta. Obstetrics and gynaecology 1984, 63: 365-70
2. Abu- Heija A, al-chalabi,el-lloubani N , Analysis of risk factors and perinatal outcome of abruption. Jordan university of science and technology,irbid,Jordan.
3. Amornrath pitaphram , Nares sukcharoen, pregnancy outcome in placental abruption. Journal of med assoc Thai 2006; 89(10): 1572.
4. Ananth CV, savitz DA, Williams MA, Placental abruption and its association with hypertension and prolonged rupture of membranes ; a methodologic review and meta analysis . Obstetrics and gynaecology, 1996: 88 ; 309-18.
5. Andrea G witlin, George r saade, risk factor for abruptio placenta& eclampsia analysis of 445 consecutively managed women with severe preeclampsia and eclampsia. American journal of obstetrics and gynaecology vol 24,issue6.june 2011
6. Arulkumaran management of labour. 3 rd edition
7. Blueprints Obstetrics and Gynecology, Fifth Edition
8. C.A.Nath, C.V.Anandh, ' Histological evidence of inflammation and risk for risk for Placental abruption' American journal of obstetrics and gynaecology. Vol.197 No.3, pp. 319 e1- 319 e6. 2007
9. C.V.Ananth , D.Getahun, M.R.peltier Placental abruption in term and preterm gestation : evidence for heterogenesity in clinical pathway, Obstetrics and gynaecology ,vol 107: no.4 ,pp785-792,2006.
10. C.V.Ananth, G.S. Berkowitz, D.A.Savitz, Placental abruption and adverse perinatal outcome ,Journal of American medical association, vol 282, no 17, pp 1646 – 1651, 1999.
11. C.V.Ananth,Y.oyelese,N.srinivas, Preterm premature rupture of membrane ,

intrauterine infection, oligohydromnios, risk factor for placental abruption Obstetrics and gynaecology , vol. 104. No.1, pp71-77, 2004.

12. Cande V. Ananth and Allen J. Wilcox , Placental abruption and perinatal mortality in the United States. American Journal of Epidemiology Vol. 153, No. 4, pp 332- 337, 2001.
13. Cande V. Ananth, John C. Smulian, Kitaw Demissie, Anthony M. Vintzileos, and Robert A. Knuppel2 . Placental abruption among Singleton and twin Births in the United States: Risk Factor Profiles ,American Journal of Epidemiology Vol. 153, No. 8.
14. D.K.James. High risk pregnancy management options, 3 rd edition.
15. Dafallah SE,Babikir HE. Risk factors predisposing to abruptio placenta maternal and fetal outcome. Saudi med journal 2004 september; 25(9):123
16. Danforth's Obstetrics and Gynecology, 9th Edition. Chapter 20.
17. Deja review obstetrics and gynaecology, page no 309-31
18. Dutta text book of obstetrics 6 th edition.
19. E.C.Nwosu, B.Kumar, S.Hollis, Is fetal gender significant in the perinatal outcome of pregnancies complicated by placental abruption, Journal of obstetrics and Gynaecocogy ,vol.19, no.6, pp 612 – 614, 1999.
20. E.J.Roccella, Report of the National High Blood pressure education program workin Group on high blood pressure in pregnancy, American journal of obstetrics and Gynaecology , vol.183, no.1,s1-s22, 2000.
21. Fernando arias. Practical guide to high risk pregnancy and delivery. 3 rd edition
22. Fiona lyalland Michal Belfort , Preeclampsia –etiology & clinical practise ,. 1 st Edition . page no 406.
23. Hladky K, yankowitz, Hansen WF, placental abruption . Obstetrics and Gynaecology surv 2002: 57, 299-305.
24. Hubbard JL, Hosmer SB, Couvelaire uterus, Journal of American osteopath

association 1997; 97 (9),536-7.

25. Ian Donald's practical obstetrics problem.6 th edition. Page no 310. 318-325.
26. Iram sarwar,Aziz un Nisa, Ansa Islam, Abruptio placenta and its complications at Ayub teaching hospital. Journal of Ayub Med coll abbottabad 2006;18(1)
27. John E Turrentine MD DMin. Clinical protocols in obstetrics and gynaecology. 3 rd edition. Page no 6-8.
28. John T. Queenan , Management of High-Risk Pregnancy an evidence-based approach 5 th edition.
29. John T. Queenan MD,John C. Hobbins MD,Catherine Y. Spong MD Protocols For High-Risk Pregnancies,An Evidence-Based approac,5 th edition.
30. karegard M, gennser G. Incidence and recurrence rate of abruptio placenta in Sweden. Obstetrics and gynaecology, 1986, 67: 523-8.
31. Keith D.Balderston,Craig V Towers, Pamela J Rumney, is the incidence of fetal to maternal maternal hemorrhage in patients with third trimester bleeding, American journal of obstetrics and gynaecology 2003, 188,1615-21.
32. Kramer MS , usher PH, Pollack R , Etiological determinants of abruptio Placenta .Obstetrics and gynaecology, 1997 : 89: 221-6.
33. Lubna amin & shaheena asif, Risk factors and placental abruption.
34. M.Sharief and A.A.Manther. abruption placenta : perinatal outcome in normotensive and hypertensive patients in Basra,Iraq. Vol.4,issue 2. 1998, page 319-323.
35. Michelle . Williams ,Ellice Lieberman ,Robert Mittendorf , and Risk factors for abruptio placenta. American journal of epidemiology 1991; 134 (9), 965-72.

36. Morgan MA, Berkowitz KM, Thomas Sj. Abruption placenta : perinatal outcome in normotensive and hypertensive patients American journal of obstetrics and gynaecology,1994 june ;170(6): 1595-9
37. Munim S, Chaudhury,Jamal K. Outcome of abruption in normotensive and hypertensive patients in Aga Khan university, Pakistan. J obstetrics and gynaecological res.1999 Feb; 25(1): 69-70
38. Nazli Hossain, Nusrat Khan, Syeda Seema Sultana, Nazeer Khan Abruptio placenta and adverse pregnancy outcome . JPMA 60;443; 2010
39. Neilson JP , Interventions for treating placental abruption .journal of Obstetrics aand Gynaecology, 2001; 21 (5); 443-447.
40. Rasmussen , irgens,dalaka,The Occurrence of placental abruption in Norway 1967-1991.acta obstet gynaecol scand 1996,75 (3), 222-8.
41. Razia Mustafa Abbasi, Naushaba Rizwan, Firdous Mumtaz and Shaista Farooq ,Feto Maternal outcome among abruptio placentae Cases at a university Hospital of Sindh .JLUMHS ,may – august 2008.
42. saftlas AF dt al, National trends in the incidence of abruptio placenta. 1979-1987 Obstetrics and gynaecology 1991, 78: 1081-6.
43. Sakikawa M, Adachi T. Clinical management and outcome of pregnancies Complicated by previous abruption. Maternal and child health centre,Tokyo, Japan .
44. Salihu HM , Bekan B, aliyu MH, perinatal mortality associated with singleton& Multiples. American journal of obstetrics and gynaecology. 1977, 128: 740-6.
45. Salma IK, Stephen AW, carrol P, pregnancy outcome in severe placental abruption. British journal of obstetrics and gynaecology. 2003;110:679-83
46. Seishi Furukawa, Hiroshi sameshima,Tsuyomu Ikenoue. Is the Perinatal outcome of placental abruption modified by clinical Presentations.Journal of pregnancy. Volume 2011.

47. Vincenzo Berghella MD FACOG ,Obstetric Evidence Based Guideline ,

Page no 195-201.

48. Wang YN, Yang Z . Multivariate analysis of risk factors with placental abruption in preeclampsia. Peking university third hospital, Beijing, china.

49. Williams obstetrics 23 rd edition. Page no761-769.

50. Witlin AG,sibai BM, Perinatal and maternal outcome following abruptio placenta Division of maternal-fetal medicine, university of texas medical branch,Galveston.

51. zoftnat weiner megnagi, Izhar ben-shlomi, Yael Goldberg, Resistance to activated protein C and the Leiden mutation: High prevalence in patients with abruptio placentae**.** American Journal of Obstetrics & Gynecology, may 1998.

PROFORMA

Name

Age

IP No

Husband name & address

Socioeconomic class

Obstetric formula

Complaints-

h/o lower abdominal pain- onset , duration

h/o bleeding per vagina- onset, duration, amount of bleeding, associated with pain

h/o amenorrhea

h/o draining PV

h/o diminished / loss of fetal movements

h/o pre eclampsia – when diagnosed, duration, drugs-dosage,

High risk factors

PROM ,polyhydromnios, GDM,fibroid,thrombophilias

Menstrual history-

Menstrual cycle regular or irregular

LMP, EDD

Marital history-

Married since

Consanguinity

Obstetric history-

Gravid, para, live children,abortion

LMP

EDD

Gestational age

Booked/unbooked

Previous mode of delivery- vaginal delivery/ LSCS

LCB

Previous history of abruption

Past history-

h/o hypertension,DM, TB , asthma, heart disease, epilepsy,thyroid disorder.

h/o previous uterine surgery / abdominal surgery.

h/o chronic drug intake, drug allergy

Family history-

H/ O hyper tension, DM, TB , congenital anomalies, twins.

Personal history-

Smoker, alcohol.

General examination-

Consciousness

Orientation

Pallor

Jaundice

Pedal edema

Breast

Thyroid

Vitals signs

Pulse rate

Blood pressure

Respiratory rate

Temperature

Systemic examination

Cardiovascular system

Respiratory system

Obstetric examination

Fundal height

Tense , tender

Acting

Presenting part

Fetal heart sound

Examination of external genitalia

Speculam examination

Local causes

Per vaginal examination

Bishop score

Pelvic assessment

CPD assessment

Colour of liquor.

Diagnosis

Investigations

Urine albumin , sugar

Hemoglobin

Blood grouping typing

Bleeding time, clotting time

Clot retraction time

Blood sugar

Renal function test

Blood urea, serum creatinine

Liver function test in preeclampsia patients

Platelet count

Prothrombin time

Ultra sound

Placental position

Retroplacental clot

Fetal presentation

Fetal heart sound

Liquor

Geatational age

Treatment

Rest

Sedation

Artificial rupture of membrane

Oxytocin augmentation

Antibiotics

IV fluids

Blood transfusion

FFP transfusion

Platelet transfusion

Maternal outcome

- Gestational age at delivery
- Mode of delivery
  - Vaginal delivery
  - Forceps
  - LSCS
- Retroplacental clot
- Couvelaire uterus

Details of abruption

- Abruption delivery interval
- Admission delivery interval
- Abruption grade
- Abruption type

Maternal complications

- Shock
- Renal failure
- DIC
- PPH
- Death

Baby

- Live / dead born
- Term / preterm
- Weight
- Apgar score
- Sex of the baby.

Normotensive patients with abruptio placenta

Case no	Name	Age	Ipno	parity	GA	**B/UB**	grade	type	Mode of delivery	Rpclot	Admis deliv	Abr del	blood	MC	Live/dead	ND	wt	apgar	
																		1min	5 min
1	SANTHYA MARY	20	103408	primi	32	UB	III A	M	ISCS	75	6 Hrs	8 Hrs	1 U	No	D		750 gm		
2	ELANJIUM	32	947610	Primi	40	UB	I	C	LSCS	50	3 Hrs 45 min				L		2.6	8/10	9/10
3	SANGEETHA	24	109389	Primi	40	UB	II	M	LN	100	30 min	6 Hrs	1 U		L		2.6	7/10	8/10
4	AMUDHA	25	109303	G2P1L1	39	UB	I	C	LN	50	20 min				L		3.3	8/10	8/10
5	VALARMATHI	35	109339	G3P2L2	39	UB	II	M	LN	100	3 Hr	6 Hr			L		3.2	6/10	7/10
6	VIMALA	23	109174	Primi	34	UB	III A	M	LN	200	50 min	7 Hr 50 min	2 U	Shock	D		1.01		
7	KAMALAM	23	108777	G3P2L2	32	UB	II	M	LN	100	40 min	3 hr 40 min	1 U		L		2	6/10	7/10
8	SOUNDARAVALLI	30	109797	G3P2L2	35	UB	III A	M	LN	400	1 Hr 20 min	9 hrs 20 min	3 u	Shock	D		2		
9	SARITHA	23	159709	G2P1L1	38	B	I	C	LN	50	6 Hrs			No	L		2.7	8/10	9/10
10	KANAGA	21	159202	Primi	38	UB	I	C	LN	50	12 Hrs				L		2.2	8/10	9/10
11	BHUVANESWARI	28	159204	Primi	36	B	II	R	LN	0	6 Hr	9 Hr			L		2.7	8/10	9/10
12	GANDHI	28	158618	G3A2	32	B	II	M	LN	75	15 min	7 Hr 15 min			L	+	1.8	4/10	6/10
13	THARAGESWARI	36	165603	G3P2L2	38	UB	IIIA	M	LN	150	9 Hr	18 Hr			D		2.3		

Normotensive patients with abruptio placenta

14	KANIMOZHI	24	165961	G2A1	32	UB	III A	M	LN	200	1 Hr	8 Hr	1U	shock	D		1.5		
15	BADHAMPRIYA	32	163900	G4P2L2A1	32	UB	III A	M	LN	200	10 min	6 Hr 10 min			D		1.4		
16	RAJATHI	22	162830	G2A1	32	UB	II	M	LN	100	7 Hr	9 Hrs			L		2.3	8/10	8/10
17	MALARKODI	18	164577	Primi	38	UB	III A	M	LN	100	2 Hr	13 Hr			D		1.5		
18	BAKIYALAKSHMI	22	162810	Primi	38	UB	I	C	LSCS	75	45 min				L		1.9	8/10	9/10
19	KALAISELVI	30	168619	G4P3L2	34	UB	III A	M	LN	150	3 Hr 30 min	10 Hr 30 min	1 PRC	Shock	D		1.8		
20	VALLI	28	170130	G4P3L3	28	UB	III A	M	LN	75	2 Hr 75 min	11 Hr 15 min	1 Unit	Shock	D		1.5		
21	POONKODI	28	159291	G4P3L1	38	UB	II	M	LN	75	30 min	4 Hr 30 min			L	+	1.6	3/10	5/10
22	MANGAIYARKARASI	27	111832	G3P2L2	34	UB	II	M	LSCS	100	30 min	2 Hr 30 min	1 Unit		L		2.2	7/10	9/10
23	VANITHA	22	159473	G2P1L1	32	UB	III A	M	LN	100	3 Hr	10 Hr			D		710 gm		
24	GEETHA	31	112409	Primi	38	B	III A	M	LN	100	5 Hr	10 Hr	1 U		D		2.26		
25	ELANJIUM	45	112268	G5P4L4	34	UB	IIIA	M	LSCS	150	1 Hr 3 min	13 Hr 30 min	3 u	Shock	D		1.1 kg		

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26	RANJITHAM	27	113056	G2P1L1	36	UB	IIIB	M	LSCS	700	30 min	18 Hr	4U 3U FFP	DIC shock MM	D		2 Kg		
27	PRIYA	23	160579	Primi	34	UB	III A	M	LN	150	6 Hr 20 min	13 hrs 20 min			D		2 kg		
28	SAVITHA	19	161077	Primi	32	UB	I	C	LN	120	40 min				L	+	1.5	6/10	7/10
29	RANI	30	161386	G3P2L0	38	UB	III A	M	LSCS	150	4 Hr	10 hr			D		2.2		
30	SUDHA	24	161551	Primi	32	UB	III A	M	LN	100	1.5 hr	8 hr			D		900 gm		
31	NAJEEMA BANU	20	162100	Primi	30	UB	II	M	LN	75	10 Hr	13 Hr			L	+	1.3	3/10	5/10
32	RADHIKA	25	160358	Primi	34	B	II	M	LSCS	75	7 hr	16hr			L		2.6	7/10	9/10
33	RAJESWARI	22	128872	G3P1L1A1	33	B	I	C	LN	50	30 min				L	+	1.2	7/10	9/10
34	KAVITHA	23	128109	Primi	39	B	II	M	LN	100	5 hr	7 hr	1 U		L		2.4	8/10	9/10
35	SHANTHI	23	159622	G2P1L0	34	UB	II	R	LN		40 MIN	1hr 40 min	1U		L		2.4	9/10	9/10
36	INDHIRA GANDHI	25	154611	Primi	35	UB	II	M	LN	50	4 hr	6 hr			L		2	8/10	9/10
37	SAROJINI	26	129062	G2P1L1	40	UB	II	M	LN	50	1/2 hr	3 1/2 hr			L		3	8/10	9/10
38	MUMTAJ	23	130318	G3P1L0A1	31	UB	II	M	LN	75	6 hr	9 hr	1 u		L	+	1.3	6/10	7/10
39	BENITA AROKIYAMARY	24	129938	Primi	36	UB	I	C	LSCS	50					L		2.2	8/10	9/10

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40	ANANTHA VALLI	25	130432	G3P2L2	32	UB	III A	M	LSCS	200	3 hr	9 HR	2U	shock	D		700 gm		
41	FATHIMA	22	126206	G2P1L1	23	UB	II	M	LSCS	100	3 hr	5 hr	1U		L		1.8	7/10	9/10
42	SARITHA	24	157136	G4P3L1	30	UB	II	M	LN	75	4.45	8.45			L	+	1.6	6/10	7/10
43	SHANMUG AREENA	21	129493	Primi	39	UB	II	M	LSCS	50	9 hr	1 hr			L		1.75	8/10	9/10
44	MAHESWARI	25	158653	G2P1L1	28	UB	II	M	LN	100	20 min	6 hr 20 min			L	+	1.1	5/10	6/10
45	SAVITHA	26	153799	G3P2L2	34	UB	II	M	LN	200	1 hr 15 min	6hr			L	+	1.3	4/10	6/10
46	SEETHA	26	111355	G2P1L1	39	B	III A	M	LN	250	3 hr	9 hr	1u	shock	D		3.3		
47	MADHAVI	26	111384	G2P1L1	39	UB	III A	M	LN	150	4 hr	9 hr	Iu	shock atonic pph	D		1.9		
48	THENMOZHI	25	112641	G2P1L1	38	B	II	M	LSCS	130	3hr20 min	6hr	2u	shock	L		1.8	7/10	9/10
49	SELVI	28	112742	G3P2L2	40	UB	II	M	LSCS	150	40MIIN	2HR 40 MIN	1u	-	L		2.2	8/10	9/10
50	MANGAIYARKARASI	27	111832	G2P1L1	40	B	II	M	LSCS	100	3HR	4HR	1u	-	L		2.2	8/10	9/10
51	RAJINA BEGAM	35	116003	G3P2L2	39	UB	II	M	LN	100	8hr	10hr	-	-	L		2.4	9/10	9/10
52	MENAGA	28	153148	Primi	40	UB	II	R	LSCS	-	1HR	3HR	-	-	L		3	8/10	9/10
53	BAGAVATHI	26	126631	Primi	36	B	II	M	LN	150	20min	2hr20min	1u	-	L		2.5	9/10	9/10

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54	MANJULA	23	120642	G2P1L1	30+3	B	II	M	LN	300	30min	6HR30MI	1u	shock	L	+	1.1	4/10	7/10
55	VIJAYALAKSHMI	30	120355	G4P3L3	40	UB	IIIA	M	LN	150	3hr	10hr	3u4ffp	shock	D		2.4	-	-
56	MANGALA KAMATCHI	26	118539	Primi	35	B	II	M	LN	50	2hr	6HR30MIN	1u	-	L	+	1.4	3/10	5/10
57	SINDHANAI SELVI	21	114607	Primi	33	B	IIIB	M	LSCS	200	3hr	10hr	3u60ffp	Shock, DIC	D		950 gm	-	-
58	DEVI	20	116913	G6P1L1A4	20	UB	IIIA	M	LN	200	1hr30min	9hr	1u	-	D		2.2	-	-
59	RADHIKA	22	116446	Primi	35+5	B	II	M	LN	200	4hr	5hr	2u	Shock	L		1.8	7/10	8/10
60	BABY	35	114410	G4P1L1A2	30+5	B	IIIA	C	LSCS	750	5hr	8hr	5u	Shock	D		2kg	-	-
61	VALLI	22	117827	Primi	36	B	IIIB	M	LN	750	3hrs	18hrs	10u10ffp	,SHOCK DIC ,ARFMM	D		2.8		
62	RENUKA	24	118790	G2P1L1	35	B	IIIB	M	LN	500	30min	15hr30min	3u4ffp	DIC SHOCK	D		2.3		
63	SUMATHI	28	119682	Primi	39	B	I	C	LN	50	11/2hr	-	-	-	L		1.8	8/10	9/10
64	MEENATCHI	30	118514	Primi	33	UB	II	M	LSCS	300	2hr	5hr	1u2uffp	Shock	L		2	7/10	8/10
65	SUMATHY	30	122387	G2P1L1	30	UB	II	M	LN	50	30min	3hr30min	-	-	L	+	1	7/10	8/10
66	ANITHA	26	119974	G2A1	33	UB	IIIA	M	LSCS	50	1hr	9hr	1U4FFP		D		1.5		

Normotensive patients with abruptio placenta

67	FATHIMA	25	152963	Primi	30	UB	IIIA	M	LN	100	4hr	10hr	-	-	D		1.5		
68	KAVITHA	23	153030	Primi	34	B	IIIA	M	LN	100	2hr	14hr	-	-	D		1.7		
69	KANAGA	22	153124	G3P2L2	36	UB	IIIA	M	LN	200	20 MIN	10hr	1U	-	D		3		
70	VANITHA	32	150652	G5P4L1	30	UB	I	C	LN	75	15min				L	+	1.3	5/10	5/10
71	REVATHY	23	150411	G2P1L1	35	B	IIIA	M	LSCS	200	4hr	12hr	1u	-	D		3.2		
72	SANGEETHA	22	150530	G3P1L1A1	32	UB	IIIA	M	LSCS	300	4hr	16hr	2u	Shock	D		1.8		
73	BANUMATHY	25	151231	G3P2L2	28	UB	IIIA	M	LN	260	9hr	18hr	2u	shock	D		750 gm		
74	PANDESWARI	24	14996	G4P3L1	32-4	UB	IIIA	M	LN	200	2hrs	12hr	3u	Shock	D		900 gm		
75	SINDHU	20	146380	G2P1L1	23	B	IIIB	M	LN	600	1hr15 min	19hr 15min	4U, 4U FFP	SHOCK, DIC	D		610 gm	-	-
76	JEEVAMARY	22	149531	G3P2L2	38	UB	II	M	LSCS	200	7hr	10hr	2u		L		2.75	7/10	8/10
77	MATHAVI	22	149128	Primi	30	UB	IIIA	R	LN	-	4hrs	10hr	-	-	D		900 gm		
78	VIJAYARANI	29	146013	Primi	39	UB	II	M	LSCS	25	1hr	6hrs			L		2.6	8/10	9/10
79	NAGAMMAL	32	146280	G4P3L2	32	UB	IIIA	M	LN	150	1.5 HR	8.5 HR	2u3 uffp	Shock	D		2.7		
80	VIJAYALAKSHMI	24	146255	G2P1L1	39	UB	I	C	LN	125	30 MIN	-	1u	-	L		2.7	8/10	9/10
81	LAKSHMI	28	146275	Primi	36	UB	II	M	LSCS	100	4hr	8hr	2u	-	L		2.5	7/10	9/10
82	VEDHANAYAGI	25	142456	G2P1L1	32	B	IIIA	M	LSCS	100	1hr10 min	6hr	1u		D		2.25		
83	VASANTHI	30	142468	G3P2L1	36	UB	II	M	LSCS	150	50min	1hr50mi	1u		L		2kg	8/10	9/10

Normotensive patients with abruptio placenta

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84	RANI	30	142380	G4P3L1	37	B	IIIA	M	VBAC LN	150	30min	16hr 30min	1U		D		2.8		
85	ELIYARANI	23	110635	G4P3L2	37	UB	II	M	LN	50	1hr15 min	4hr	1U		L		1.95	8/10	9/10
86	INDHIRA GANDHI	28	152709	G3P2L1	34+3	UB	II	M	LN	100	4hrs	6hrs			L	+	1.9	4/10	5/10
87	REVATHY	24	111119	Primi	32	UB	IIIA	M	LN	150	30min	71/2 hrs	1u		D		1.4		
88	GEETHA LAKSHMI	26	110986	G5P2L2A2	38	UB	I	C	LN	100	1hr15 min		1u		L	+	900	5/10	6/10
89	USHA	23	113039	Primi	39	B	IIIA	M	LN	100	21/2he	81/2 hr	1u		D		2.2		
90	CHELLAM	36	112517	G4P3L2	38	UB	IIIA	M	LN	100	15 MIN	10 HR			D		3.26		
91	KAVITHA	24	111501	Primi	34+2	UB	II	M	LSCS	200	1hr	4hr	-	-	L		2.5	9/10	9/10
92	LAKSHMI	25	111923	G2P1L1	35+3	B	IIIA	M	outlet	50 gm	20min	7hr	1u		D		1.3		
93	ELANJIUM	45	112268	G5P4L4	33+4	UB	IIIA	M	LSCS	150gn	1.5hr	61.5	3u	shock	D		1.1		
94	CHANDRA	26	114294	G2P1L1	30	UB	II	M	LN	75	5hr	8hr	1u	-	L	+	1.4	5/10	6/10
95	MARUTHAMBAL	25	114779	G3P2L2	32	UB	II	M	LN	100	30 MIN	7.5 HR	2u	stock	L	+	1.2	4/10	6/10
96	SHANTHI	24	116126	G2P1L1	29	UB	IIIA	M	VBAC	300	2HR40MIN	10HR40MIN	IU		D		1.35		
97	SAGAYA MARY	23	116834	G2P1L1	25	B	IIIA	M	LSCS	50	4hr	8hr	1u		D		600 gm		
98	MAHESWARI	27	117322	G2P1L1	27	B	IIIA	M	LN	50	30min	81/2 hr	-	-	D		3.1		

Normotensive patients with abruptio placenta

99	KALA	35	115869	Primi	35	B	I	C	LN	50	3 HR	-			L		1.9	8/10	9/10
100	MERCILILN HORATHI	24	147042	G3P2L 1	36	UB	IIIA	M	LN	100	25 MIN	10 HR	1u		D		2.3		
101	MANIMEG ALAI	24	147013	G3P2L 2	36	UB	IIIA	M	LN	100	4hr	11H R	1u		D		2		
102	JEYANTHI	31	132181	G4P3L 3	38	UB	IIIB	M	LN	800	11/2hr	16hr	3u2 UPP	SHOC K DIC	D		2.1		
103	VIYAGULA MARY	35	132317	G3P1L 1	28	B	IIIA	M	LN	50	3hr	9hr	-	-	D		900 gm		
104	LAKSHMIP RIYA	21	132448	Primi	32	B	I	C	LN	50	50min	-	-	-	L	+	1kg	4/10	6/10
105	SAMANAS AMARY	34	133510	G3A2	37	B	IIIA	M	LSCS	200	1hr	6hr	1U	-	D		1.8		
106	CHITRA	23	133901	G3P2L O	34	UB	IIIA	M	LSCS	200	1hr	4hr	1U	-	D		1.2		
107	CHINNATH A	20	136403	G2P1L 1	38	UB	I	C	LN	50	2hr	-	-	-	L		2.4	7/10	9/10
108	ANANDHAJ OTHI	25	138526	G3P2L 1	38	B	IIIA	M	LN	150	3hr	6hr	-	-	D		1.7		
109	NEELAVEN I	31	139506	G2P1L 1	37	B	IIIA	M	LSCS	100	1hr	10hr	-	-	D		2		
110	RAJALAKS HMI	24	139539	G2A1	38	UB	IIIA	M	LSCS	300	1hr	9hr	1U	-	D		1.8		
111	SUNDHARI	25	139682	G3P2L 1	30	UB	IIIA	M	VBAC LN	130	30min	71/2 hrs	-	-	D		900 gm		
112	SUMATHI	25	139696	G2P1L 1	28	UB	IIIA	M	LN	300	2hr	8hr	2U	shock	D		850 gm		
113	TAMILARA SI	27	139725	G3P2L 1	38	B	II	M	LN	75	2hr	6hr	-	-	L	+	1.4	4/10	6/10
114	SUGINA BEGAM	23	139715	Primi	30	B	II	R	LSCS		12hr	1hr			L		1.8	9/10	9/10
115	RAGMATH NISHA	25	139866	G2P1L 1	38	UB	II	M	LSCS	150	1hr	4hr			L		2	8/10	9/10
116	SHANTHI	22	140772	G2P1L 1	38	UB	I	C	LSCS	100	1hr				L		K	7/10	8/10

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117	VAIJEYANTHIMALA	24	138143	G2P1L1	38	UB	IIIA	M	LN	150	30min	71/2 hrs			D		2.2		
118	ANNAKILLI	35	141024	G3P2L1	37	B	IIIA	M	LN	75	15min	10hr			D		1.45		
119	INDHIRA	21	135350	G2A1	38	B	II	C	LN	50	45min	7hr			L	+	1.3	4/10	5/10
120	AJINA BEGAM	23	139715	G2P1L1	37	UB	II	M	LSCS	100	1hr	4hr			L		1.8	7/10	9/10
121	DEVI	24	132335	Primi	36	UB	II	M	LN	200	45min	6hr			L	+	1.2	5/10	6/10
122	ILAVARASI	30	134325	Primi	37	UB	II	M	LN	200	1hr	7hr			D		2		
123	PARVATHI	21	121345	Primi	38	B	I	C	LN	50	30min				L		2.7	8/10	9/10
124	ANGAIYARKANI	22	132178	Primi	32	UB	I	C	LN	50	20min				L	+	680 gms	4/10	5/10
125	DEVI	20	123543	G6P1L1	34	B	IIIA	M	LN	200	1hr15 min	8hr			D		2.2		
126	GEETHA	26	132111	G2P1L1	38	UB	II	M	LN	250	3hr				L		3.3	9/10	9/10

Hypertensive patients with abruption

No	Name	Age	Ipno	Parity	GA	B/UB	grade	type	delivery	rpclot	Admideli very	abruptiondeli	BT	Maternc ompli	Live/dead	Baby wt	ND	apgar	PIH type
1	Latha	27	112890	G2P1L1	37	B	III A	M	LN	100	50 min	7 hr	1U	_	D	1.9	_	_	Mild
2	Kalapriya	27	112450	primi	39	B	II	M	LSCS	100	1hr	3 hr	1U	_	L	2.7	_	8/10 9/10	Mild
3	Mariyammal	28	113104	G3P2L2	25	UB	III B	M	LN	350	1.5 hr	13.5 HR	3U 14ffp	D/C Shock ARF	D	900 gm	_	_	ppeclam
4	Punithavalli	27	130324	primi	35	B	I	C	LSCS	75	6 hr	_	_	_	L	1.6	_	7/10 9/10	Mild
5	Ramani	32	167439	G5P4L4	34	UB	III A	M	LN	50	2.5 hr	14.5 hr	1U	_	D	2.5	_	_	Mild
6	Shanthi	23	159622	G2P1L1	35	B	II	R	LN	_	25 min	4.5 hr	1U	_	L	2.4	_	_	Mild
7	Savitha	25	159709	G2P1L1	34	UB	II	M	ASSIS	200	1 hr	8 hr	2U	Shock	L	1.7	+	4/5 4/5	Mild
8	Durga	23	154665	G3P2L1	33+5	UB	III A	M	LN	200	40 min	10 hr	1U	_	D	1.6	_	_	Severe
9	Madhiyalagi	28	154986	G3P2L2	26	UB	II	M	LN	100	1 hr 40min	7hr 40min	1U	_	L	1.5	+	5/10 6/10	Severe
10	Parimala	25	154934	G2P1L1	38	B	III A	M	LSCS	100	1 hr	9hr	1U	_	D	1.6	_	_	Mild
11	Eswari	22	156225	G2P1L1	32	UB	III A	M	LN	100	2 hr	10 hr	_	_	D	1.7	_	_	Mild
12	Tamilarasi	27	155600	G3P1L1A1	30	UB	III A	M	LSCS	200	40 min	5.4	1U	_	D	700	_	_	Mild
13	Ramya	19	167211	primi	29	B	III B	M	LN	200	20 min	8 hr	2U 4UF FP	Shock DIC	D	1.4	_	_	Apecla
14	Mayilarasi	27	157059	primi	40	B	III A	M	LN	75	6 hr	10 hr	2U	_	D	1.69	_	_	Severe
15	Kavitha	21	126970	G2P1L1	34	UB	I	C	LN	75	1 hr	_	_	_	L	2	_	7/10 8/10	Apecla

] Hypertensive patients with abruption

16	Meena	28	135182	G3A2	38	UB	II	C	LSCS	150	7 hr	2 hr	_	_	L	3	_	8/10 9/10	Mild
17	Savitha	22	130319	primi	30+5	B	III A	M	LSCS	500	2 hr	8hr	2U	shock	D	2	_	_	Mild
18	Padmavathi	25	130497	G2A1	37	UB	I	C	LSCS	100	1hr	_	_	_	L	1.8	_	8/10 9/10	Mild
19	Valliyammal	34	130180	G3P2L2	36+1	UB	III B	M	LSCS	100	30 min	12hr	2U,4 uffp	shock DIC	D	2	_	_	Apecla
20	Revathi	26	128304	G3P2L2	37+6	UB	II	M	LSCS	300	1 hr	6 hr	1U	_	L	2.3	_	8/10 9/10	Mild
21	Chandrakala	26	128195	primi	34	UB	II	M	LSCS	100	1 hr	4 hr	1U	_	L	1.5	+	5/10 6/10	Mild
22	Nithiya	22	166600	primi	37	B	II	M	LSCS	300	1 hr	9 hr	1U	Shock	L	2.1	_	7/10 8/10	Mild
23	Rajathi	27	160809	G3P2L2	35	UB	III A	M	LN	250	30 min	10 hr	1U	_	D	900	_	_	Severe
24	Priya	21	166787	primi	34	B	III A	M	LN	200	2 .5 hr	5 hr	1U	_	D	1.2	_	_	Mild
25	Mariyammal	28	113704	G3P2L2	29+3	UB	III A	M	LN	300	1 hr	8 hr	3U 2Uffp	Shock	D	900	_	_	Mild
26	Thenmozhi	25	112641	G2P1L1	37	UB	II	M	LN	300	3.5 hr	7 hr	2U 3Uffp	Shock	L	1.8	+	3/10 3/10	Severe
27	Sarasvathi	24	112689	G3A2	24	B	III B	M	LSCS	750	1 hr	11hr	4U 6Uffp	Shock DIC	D	1.5	_	_	Apecla
28	Sathyavani	27	159732	primi	34	B	I	C	LSCS	75	7 hr	_	_	_	L	1.4	_	7/10 8/10	Mild
29	Parimala	21	158609	primi	36	UB	II	M	LN	75	2 hr	6 hr	_	_	L	1.4	_	8/10 9/10	Mild
30	Kasthuri	29	170198	primi	37	B	III A	M	LN	200	2 hr	9 hr	2U	Shock	D	2.6	_	_	Mild
31	Rameswari	37	150154	G3P2L2	26	UB	III A	M	LN	200	4.5 hr	8 hr	2U	_	D	550 gm	_	_	Severe

Hypertensive patients with abruption

32	Chellammal	20	151165	primi	33	B	I	C	LN	50	_		2U 10U	HELLP	L	1.1	+	5/10 6/10	Severe
33	Meena	21	150301	primi	40	UB	II	M	LSCS	150	3 hr	6 hr	_	_	L	2.7	_	8/10 9/10	Mild
34	Maheswari	27	150299	G2P1L1	41	B	II	M	LSCS	100	1.5 hr	7 hr	_	_	L	2.25	_	7/10 8/10	Mild
35	Vembu	23	151672	primi	38 +4	UB	I	C	LSCS	100	0.5 hr	_	1U	_	L	3	_	8/10 9/10	Severe
36	Usha	23	151909	primi	32+4	B	III A	M	LN	200	4 hr	14	1U	_	D	1.3	_	_	Mild
37	Sumathi	25	151944	primi	30+5	UB	III A	M	LN	300	6 hr	12	1U	_	D	850	_	_	Mild
38	Mahalakshmi	26	173210	primi	28	B	III A	M	LN	100	3 hr	7 hr	1U	_	D	900	_	_	Severe
39	Nageshri Anu	22	149141	G2P14	30	UB	III A	M	LN	100	10 hr	16	1U	_	D	1kg	_	_	Mild
40	Kalaivani	21	146081	primi	30	UB	III A	C	LN	50	6 hr	12	1U	_	D	1.2	_	_	Apecla
41	Silambarasi	22	146035	primi	37	B	I	C	LSCS	100	3 hr	6 hr	1U	_	L	2.1	_	8/10 9/10	Mild
42	Annalakshmi	25	147121	G4P2L2 A1	39	UB	III A	M	LSCS	600	5 hr	10 hr	3U	shock	D	2.8	_	_	Mild
43	Kavipriya	22	145859	primi	31	UB	II	M	LSCS	250	2 hr	8 hr	1U	_	L	1.6	_	8/10 9/10	Mild
44	Sangeetha	22	146710	primi	39	B	II	M	LSCS	200	15 min	6 hr	1U	_	L	2.3	_	7/10 8/10	Mild
45	Fathima	25	152963	primi	34	UB	III A	M	LN	100	4hr	8hr	2U	shock	D	1.5	_	_	Mild
46	Muniyammal	25	153173	G2P1L1	30	B	III B	M	LSCS	200	1.5 hr	12 hr	3 U 6U	Shock DIC	D	800	_	_	Apecla
47	Mutheswari	27	116284	G3P2L2	34+5	UB	I	C	LN	100	12 hr	_	1U	_	L	2.1	_	8/10 9/10	Mild
48	Sasikala	30	117287	G2P1L1	29+4	UB	III A	M	VBAC LN	50	30 min	8 hr	1U	_	D	780	_	_	Mild
49	Rajeswari	21	116351	G2A1	35+3	B	III	M	LSCS	100	11/2	111/	IV	-	D	3.5	-	-	Mild

Hypertensive patients with abruption

							A				hr	2hr						
50	Jeyamala	27	115072	G2P1L1	38+2	UB	III B	M	LSCS	700	21/2 hr	18hr	4U10 U	DIC Shock ARF	D	2.5	-	-	Mild
51	Mariyappa	26	115765	G3P1L1	28+4	UB	IIIa	M	LN	100	40MIN	10hr	-	-	D	690gn	-	-	Severe
52	Anandhi	24	153398	primi	33+1	UB	III B	M	LSCS	150	11/2 hr	12hr	3U 4U FFP	DIC SHOCK	D	2.2	-	-	Mild
53	Uma	28	114788	G2P1L1	35	UB	III A	M	LSCS	300	6hr	10hr	2U	SHOCK	D	1.6	-	-	Mild
54	Kalaiselvi	27	114057	G4P3L3	30	UB	III B	M	LN	150	3hr	10hr	2U , 4U FFP	DIC	D	700	-	-	Mild
55	Revathi	23	114650	primi	39	UB	III A	M	LN	500	6hr	12hr	4U , 1U FFP	SHOCK	D	1.8	-	-	Mild
56	Anitha	22	152920	G2P14	23	UB	III B	M	LSCS	350	4hr	9hr	,3U ,4U FFP	DIC	D	850	-	-	Mild
57	Praveena	23	112323	G2P1L1	26+4	UB	III A	M	LN	100	2hr	8hr	-	-	D	570	-	-	SEVEN
58	Anjammal	20	111183	G3P2L0	28+2	B	III A	M	LN	250	20 MIN	7hr	1U	-	D	720g	-	-	SEVEN
59	Analakshmi	24	111568	G3P1L1A1	36	UB	III A	M	LN	200	25 MIN	6hr	1U	-	D	1.2	-	-	SEVEN
60	Nalini	24	110255	primi	36	B	II	M	LSCS	100	1hr	7hr	1U	-	L	2.1	-	8/10/9/10	SEVEN
61	Jeebaithabevi	21	136512	primi	39	UB	III A	M	LSCS	100	1hr	6hr	-	-	D	1.2	-	-	SEVERE
62	Sami Nayagi	30	112694	G4P3L3	35	UB	III A	M	LN	400	6hr	16hr	3U, 3U FFP	SHOCK	D	2.1	-	-	MILD
63	Manimozhi	24	112408	G2A1	38	UB	II	M	LSCS	100	20mi	4hr	IV	-	L	2.4	-	-	SEVEN

Hypertensive patients with abruption

64	Usha	21	111076	primi	38	UB	III A	M	LN	100	21/2 hr	8hr	-	-	D	2	-	-	Mild
65	Shanthi	32	110886	G2P1L1	33+5	B	III A	M	LN	400	4hr	10hr	2U	SHOCK	D	1.6	-	-	Mild
66	Maheswari	27	110667	PRIMI	34	UB	III A	M	LN	100	6hr	12HR	1U	-	D	1.4	-	-	Mild
67	usha	25	110244	G2P14	31	UB	III A	M	LSCS	150	5hr	10hr	-	-	D	1.8	-	-	Mild
68	Malathy	25	107608	G4P2L1A1	36	UB	I	C	LSCS	50	21/2 hr	-	1u	-	L	2		8/109/10	Mild
69	Rajakumani	35	121770	primi	32	UB	III A	M	LN	500	25mini	10hr	2u	SHOCK	D	1.28	-	-	SEVERE
70	Jeevitha	23	119243	primi	33	B	III B	M	LSCS	150	3 hr 20 min	18hr	4u100FFP	shock ,DIC, ARF , MM	D	2.69			Apecla
71	Geetha	24	117177	primi	36	B	III A	M	LSCS	200	3hr	11hr	1u	-	D	1.6	-	-	SEVERE
72	sivasankani	30	121477	primi	36	UB	I	C	LSCS	50	11hr	-	-	-	L	1.8	-	8/109/10	SEVERE
73	Nadhiya	28	121044	G3P2L2	35+5	UB	II	M	LSCS	150	40m	12hr	2u	SHOCK	L	2.7	-	7/10/8/10	Mild
74	Sangeetha	24	153848	G3P1L1A1	29	UB	II	M	LN	100	1hr40m	9hr	-	-	L	1.4	-	8/109/10	SEVERE
75	pauvathi	37	118452	G6P5L4	28	UB	III A	M	LN	50	6hr	10hr	-	-	D	900	-	-	Severe
76	Sugnathy	25	117412	G2P1L1	32	UB	III A	M	LN	250	1/2hr	15hr	2u	SHOCK	D	1.4	-	-	Mild
77	Latha	29	115791	G3P2L1	35+4	B	III B	M	LSCS	200	3hr	18hr	2u4uffp	DIC	D	2	-	-	severe
78	Bhunwaneswari	27	118633	G2A1	37	UB	II	M	LN	50	31/2 hr	7hr	-	-	L	2	-	8/109/10	Mild
79	sumathy	29	114684	G3P1L1	37+2	B	III A	M	LN	300	1/2hr	91/2	3u	SHOCK	D	2.4	-	-	Recurrent
80	Megala	25	107786	G3P1L1	26	UB	III	M	LN	50	50mi	8hr	1u	-	D	1.4			Mild

Hypertensive patients with abruption

							A				ni							
81	Jeyapriya	22	107607	G2A1	25+4	UB	II	M	LN	125	2hr	10	-	-	L	700	+	4/10 6/10	Mild
82	Maheswari	28	97678	G2P1L1	32+5	UB	III A	M	LN	250	3hr	8hr	1u	-	D	1.3	-	-	Mild
83	Surya	23	109268	G2P1L1	35+2	UB	I	C	LN	130	30MI	-	-	-	L	2.07	-	8/10 9/10	Mild
84	Gowsalya	26	138063	G2P1L1	38	B	II	M	LSCS	100	1 HR	6HR	-	-	L	2	-	7/10 9/10	SEVERE
85	Maheswari	25	140740	primi	34+3	UB	III B	M	LSCS	750	30MIN	20HR	4U,4UFFP,1PLT	SHOCK,DIC,	D	900	-	-	Mild
86	anthoniyammal	28	145183	primi	35	UB	II	C	LSCS	50	2HR	5HR	1U	-	L	2	-	-	Mild
87	Christipriya	32	142459	G3P212	38	UB	III A	M	LN	200	40MIN	10HR	1U	-	D	1.4	-	-	Mild
88	pushpam	26	145131	G2P1L1	32+6	B	I	C	LSCS	100	1HR		IU	-	L	2.5	-	-	Mild
89	vedhavalli	28	145563	primi	39	B	III B	R	LN	-	4HR	10HR	2U,4UFFP	DIC	D	2.6	-	-	Mild
90	Ilavarai	22	131684	primi	38	UB	III A	M	LN	150	10MI	11HR	-	-	D	1.6	-	-	Severe
91	Vasanthi	30	141960	PRIMI	34	UB	II	M	LSCS	100	2HR	7HR	-		L	1.7	-	-	Severe
92	Mala	25	164510	G2A1	30	UB	III A	M	LN		20 MIN			MM	D	2.4	-	-	severe
93	Nagavalli	40	140216	G7P6L6	38	UB	III A	M	LN	400	1.5HR	16.5 HR	3U, 2U FFP	SHOCK	D	2.9	-	-	SEVERE
94	viji	24	136391	G2P1L1	37	UB	III A	M	LSCS	300	1HR	6 HR	1U	-	D	4.5	-	-	Mild
95	Kanimozhi	22	140305	G2P1L1	30	B	I	C	LSCS	125	6 HR		-	-	L	1.5	-	8/10 9/10	Mild
96	Latha	23	140348	G2P1L1	38	UB	III A	M	LSCS	100	3 HR	7 HR	-	-	D	2.3	-	-	Mild

Hypertensive patients with abruption

97	Pasirakani	29	131527	G2P1L1	28	B	II	M	LSCS	100	25 MIN		-	-	L	900G	+	5/10 6/10	Mild
98	Usha	21	117687	primi	38	UB	III A	M	LN	100	2 HR	8 HR	-	-	D	2	-	-	Mild
99	uma	23	143275	G2P1L1	34	UB	III A	M	LSCS	600	5hr	10hr	-	-	D	1.6	-	-	Mild
100	MADHAVI	26	113204	G2P1L1	36	UB	III A	M	LN	150	5HR	9 HR	-	-	D	1.9	-	-	MILD

Abbreviations

UB - Unbooked

B - Booked

ARF - Acute renal failure

DIC - dissiminated intravascular coagulation

LN - labour natural

LSCS - Lower segment casarean section

MM - Maternal mortality

PPH - post partum hemorrhage

STH - subtotal hysterectomy

C - concealed hemorrhage

R - reavealed hemorrhage

M - mixed hemorrhage