"STUDY OF OBSTETRIC OUTCOME IN ABRUPTIO PLACENTA IN A RURAL TEACHING HOSPITAL"

By

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IN

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Under the Guidance of

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Dr. Seema B.R.

LIST OF ABBREVIATIONS USED

SL.NO	Abbreviation	Full form
1.	DIC	Disseminated intravascular coagulation
2.	PROM	Prelabour rupture of membranes
3.	IUGR	Intrauterine growth restriction
4.	PIH	Pregnancy induced hypertension
5	HLA	Human Leukocyte antigen
6.	NK	Natural Killer
7.	IL	Interleukin
8.	TNF	Tumor necrosis factor
9.	MMP	Matrix metalloproteinase
10	PlGF	Placental growth factor
11	VEGF	Vascular endothelial growth factor
13	sFlt1	Soluble fms like tyrosine kinase 1
14	sEng	Soluble endoglin
15.	MSAFP	Maternal serum alpha feto protein
16.	CTG	Cardiotocograph
17.	FDP	Fibrin degradation products
18.	PPH	Post partum hemorrhage
19.	HELLP	Hemolysis, elevated liver enzymes & low platelet
20.	NICU	Neonatal intensive care unit
21.	LSCS	Lower segment cesarean section
22.	FHS	Fetal heart sounds

ABSTRACT

BACKGROUND:

Abruptio placenta is one of the obstetrical emergencies. The maternal effect of abruptio placenta depends primarily on its severity, whereas the fetal effects are determined by both severity and gestational age. The poor perinatal outcome is due to low birth weight, prematurity and still birth.

OBJECTIVES:

- To study the incidence and clinical profile of patients presenting with abruptio placenta
- To study the outcome of pregnancy in terms of maternal and perinatal morbidity and mortality in those patients

METHODS:

A total 100 cases were studied during the period January 2012 to August 2013 in department of Obstetrics & Gynecology. Detailed clinical history and investigations were noted. Patients were initially stabilized hemodynamically and delivered early either by vaginally or by Caesarean section depending on maternal and fetal status. Details of mode of delivery and admission to delivery interval with maternal, fetal mortality and morbidity were noted.

RESULTS

In the present study the incidence of abruptio placenta was 2.52%. Majority of our cases were primigravida (42%). The common associated condition was pre-eclampsia/eclampsia (29.05%). Clinical diagnosis was made in 77% of cases and sonographic diagnosis in 23% of cases. The incidence of live births in Grade 1 and 2 by vaginal route and cesarean delivery in the present study was 85.2% and 88.2%,

respectively. Maternal complications found were PPH (7%), Couvelaire uterus (7%), disseminated intravascular coagulation (3%) and acute renal failure (3%). Perinatal mortality rate was 58%.

CONCLUSION:

- Diagnosis of abruptio placenta is essentially clinical and sonography has got limited role.
- Vaginal delivery is quite effective in severe abruptio placenta with dead fetus.
- Short admission to delivery interval did not increase the fetal survival.
- Earlier presentation to hospital and early diagnosis improve the prognosis of abruptio placenta.

KEYWORDS: Abruptio placenta, mode of delivery, fetal outcome

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INTRODUCTION

Abruptio placenta or accidental hemorrhage is one of the obstetrical emergencies and is truly accidental with few warning signs. In developed countries the incidence is 1% of deliveries, whereas in developing countries it is around 2-8%.[1,2,3] In many countries the rate of placental abruption has been increasing, perhaps due to advancing maternal age and increasing cesarean section rates.[4,5,6,7] It is one of the significant causes of maternal and perinatal mortality and morbidity. The incidence of maternal mortality is around 1%, whereas perinatal mortality is much higher accounting for 66% of deliveries.[4,8] The maternal effect of abruptio placenta depends primarily on its severity, whereas the fetal effects are determined by both severity and gestational age at which it occurs. The etiology of abruptio placenta is obscure. However, there are many risk factors associated with the condition like hypertensive disorders of pregnancy, polyhydramnios, intrauterine growth restriction, advanced maternal age, maternal trauma, cigarette smoking, alcohol consumption, cocaine abuse, short umbilical cord, premature rupture of membranes, grand multipartiy, etc.[9,10,11,12] The major maternal complications of abruptio placenta are hemorrhagic shock, disseminated intravascular coagulation, acute renal failure, post partum hemorrhage and maternal death.[11,13] The poor perinatal outcome is due to low birth weight, prematurity and still birth.[14,15] With the better availability of blood and blood products and coagulation factors, the management of shock and DIC has decreased the maternal and perinatal morbidity and mortality over last few decades. There has been increase in the use of Cesarean delivery over recent years in abruptio placentae which have resulted in a better obstetric outcome. Present study is planned to study the obstetric outcome in patients of abruptio placenta in a tertiary care referral hospital in a rural set up and this is helpful to plan management strategies to decrease mortality and morbidity due to abruptio placenta

OBJECTIVE

- To study the incidence and clinical profile of patients presenting with abruptio placenta

- To study the outcome of pregnancy in terms of maternal and perinatal morbidity and mortality in those patients

REVIEW OF LITERATURE

OVERVIEW

General aspects

The placenta is a unique organ which links mother and fetus. It provides oxygen, nourishment and protection to the fetus and at the same time it has excretory and endocrine functions also. A blastocyst is formed after several mitotic divisions of the zygote. The blastocyst divides in to an outer shell of cells, the trophoblast, and an inner cell mass, the embryoblast. The blastocyst gets attached to the endometrium via the trophoblastic cells, which rapidly proliferate and differentiate into an outer layer of syncytiotrophoblast and an inner layer of cytotrophoblasts.[16] The syncytiotrophoblasts form primary, secondary and finally tertiary villi and cytotrophoblasts form intervillous space. The placenta is fixed to the uterine wall by anchoring villi. The placenta achieves its full functioning form by the end of the fourth month of gestation. Further growth, villous branching and formation of new villi continue til term.[17] Placenta separates naturally after the delivery of the baby during the third stage of the labor which is brought about by the contraction and retraction of the uterus.[18]

DEFINITION

Abruptio placenta is defined as the premature separation of the normally situated placenta after the 20th week of gestation and before the birth of the fetus.[4] It is also been variously called as abruptio placentae, placental abruption or accidental hemorrhage.

HISTORICAL ASPECTS

Louis Bourgeos recognized premature separation of placenta in 1609. De Lee and Coole, in 1848, used the term "Abruptio placentae" to mean sudden forcible separation of placenta from its normal site. In 1901, Holmes of Chicago proposed the term 'abruptio placenta'. In 1912, Couvelaire gave the term 'uteroplacental apoplexy' to severe forms of accidental hemorrhage.

INCIDENCE

The incidence of abruptio placenta varies widely as reported from various studies. This variation is due to different criteria adopted by different authors for the diagnosis and also because it is mainly a clinical diagnosis. The overall incidence of placental abruption varies from 0.5 to 1.0% deliveries in developed countries.[19,20,21&22] In a study conducted by Saftlas and colleagues the incidence of abruption placenta was 11.5/1000 deliveries.[23] In a case-control study done by Dafallah, Saad E.Babikir, Hayder during 1997-2002 the incidence of abruption was 6.5% when the total number of cases studied was 15,620.[24] The highest incidence is found at 24-26 weeks of gestation, and gradually drops with advancing gestational age.[12,25] However, the incidence after the 36th week of gestation is about 50% of cases.[19] Some, but not all, studies have reported increasing overall rates.[10,12,23,26]

PATHOGENESIS AND PATHOLOGY OF ABRUPTIO PLACENTAE

Placental abruption is a multifactorial disease. Its etiology is not fully understood but impaired placentation, placental insufficiency, intrauterine hypoxia, and uteroplacental underperfusion are the mechanisms involved in abruption.[27-30] Abruption results from a rupture of maternal decidual artery. The blood collected causes a dissection at the decidual-placental interface, either around placental margin or behind the membranes.[16] During the normal

separation at delivery, the myometrium of empty uterus contracts around the open maternal sinus causing hemostasis. This mechanism is absent in abruptio placenta because the uterus is distended with products of conception.[31] The decidual placenta eventually leads to separation, compression and destruction of the placenta.[32] The various mechanisms involved in placental separation are acute vasospasm of small vessels, thrombosis of the decidual vessels with associated decidual necrosis and venous hemorrhage, and in some cases, blunt trauma or rapid decompression of the over distended uterus can cause abruption. It may also result from a longstanding process perhaps dating back to the first trimester.[25] In early stages the condition is discovered only upon examination of freshly delivered placenta, showing depression of few millimeters on maternal side covered by dark clotted blood. Whereas recently separated placenta may appear no different from the normal one.[32] The age of retroplacental clots cannot be determined exactly.

After the decidual spiral artery ruptures, it leads to collection of retroplacental hematoma. As the size of the hematoma increases it disrupts more vessels to separate more placenta reaching the margins The blood escaping under the deciduas basalis can then pursue one of the four courses

- Dissect under the membranes eventually leading to vaginal bleeding which is seen externally
- Breakthrough the membranes into the amniotic cavity which is seen when the membranes rupture
- The blood can collect behind the placenta resulting in concealed hemorrhage leading to separation of placenta from the maternal surface

- Seepage of blood through the myometrium where the uterus takes on a purplish colour referred as Couvelaire uterus.[33]

The diapedesis of blood from the deciduas into the myometrium acts like ecbolic agent and is associated with a contraction that may be well localized or diffuse and tetanic. Tetanic contraction of the uterus interferes with the uteroplacental circulation leading to fetal hypoxemia, acidosis and possible fetal death. In addition the increased intra-amniotic fluid pressure secondary to the tetanic contraction may further jeopardize the uteroplacental circulation and fetal health.[31]

Normally the uterine wall is more elastic than the placenta. In case of traumatic etiology, during an impact, the unrestrained body decelerates against some object and the uterus flattens against the part of the abdomen which decelerates first. The vertical waves cause rapid elongation and shortening of the long axis of the uterus shearing placental attachment.[34]

Immunological rejection

Immunological defects may play a role in the origin of placental abruption.[36,37] In normal pregnancy cell mediated immunity is suppressed and humoral immune response is upregulated.[37,38] This does not occur in placental abruption which can lead to exaggerated maternal immune rejection of the fetus, activation of fetal monocytes and release of inflammatory agents.[36,39] Similarly, human leukocyte antigen (HLA) G which is the decesive factor for avoidance of rejection of fetus is reduced in placental abruption, hence, switching the cytokine profile towards Th2 response. Normally there is an interaction between trophoblastic cells & Natural killer cells (NK) which expresses receptors recognizing HLA combinations. When this interaction is defective it causes poor trophoblastic invasion, defective spiral artery

remodeling, placental infarction and thrombosis.[36,40] Hence, this suggests that placental failure is due to flawed maternal immune response to paternal antigens.[41]

Inflammation

Placental abruption may be a manifestation of acute or chronic inflammatory process.[28] Infections cause a rapid release of various inflammatory mediators at the Maternal-fetal interface.[28,42] The placentas of women with abruption show increase in neutrophils and macrophages.[25] Oxidative stress and products of vascular activation and coagulation such as thrombin has similar effects.[28] In abruption there is association of thrombin enhanced expression of interleukin (IL) 8 which is a potent neutrophil chemoattractant.[43] There is also increased production of proinflammatory cytokines such as tumor necrosis factor (TNF) α and IL β1 which stimulates the production of matrix metalloproteinases (MMP) by trophoblasts.[28] Increased production of MMP results in the destruction of the extracellular matrix that lead to premature detachment of placenta.[28]

Vascular disease

Normal placentation requires trophoblastic cells invasion of maternal spiral arteries, and development of a highflow, low resistance uteroplacental circulation.[44] In the presence of various proangiogenic and antiangiogenic factor vascular remodeling occurs.[40,45,46,47,48] The proangiogenic factors like placental growth factor (PlGF) and vascular endothelial growth factor (VEGF), promote the formation of placental blood vessels and also the invasion of trophoblasts in the spiral arteries.[40,45,47] Antiangiogenic factors like soluble fmslike tyrosine kinase 1 (sFlt1) binds to the biologically active forms of PlGF and VEGF, and soluble endoglin (sEng) which blocks the binding of transforming growth factor isoforms to endothelial receptors.[48,49] It appears

that PlGF deficiency and sFlt1 excess may result from placental hypoxia associated with incomplete remodeling of maternal spiral arteries.

RISK FACTORS

The etiology of abruption remains unknown in majority of cases. In few cases, apparent association is seen with sudden uterine decompression and trauma. There is a controversial relationship between abruption, maternal age, parity, folate deficiency and socio economic status. In addition to above there may be relationship between abruption, cigarette smoking, uterine anomalies, previous preterm labour and unexplained elevation of second trimester maternal serum alfa feto protein. Various studies have reported risk factors for abruption placentae. According to them, following are the associated risk factors for abruption.[32]

- Age
- Parity
- Race
- Sex of the offspring
- Nutritional state
- Socio-economic status
- Hypertensive disorders of pregnancy
- Diabetes
- Premature rupture of membranes
- Chorioamnionitis
- Preterm labour
- Small for gestational age

- Folic acid deficiency
- Cigarette smoking
- Drugs like cocaine, marijuana, alcohol
- Trauma
- Hydramnios
- External cephalic version
- Multifetal gestation
- Uterine anomalies and tumours
- Previous history of abruptio placentae
- Miscellaneous:

  - Snake bite

  - Increased MSAFP

  - Inherited or acquired thrombophilia

  - Hyperhomocysteinaemia

  - Ascorbic acid deficiency

1. Age

The evidence regarding relationship between maternal age and abruptio placenta is conflicting. In white women, the rate is increased for all age groups but 20 - 24 year old women. In non-white women, the rate is increased for all age groups. According to Bryan M. Hibbard, the incidence increases with maternal age, and patients over 35 years are twice as prone to

abruption as patients under 25 years.[50] Karegard M et al found a higher incidence of abruption placentae in primigravidae less than 20 years of age.[51]

2. Parity

The relationship between abruptio placenta and parity has been controversial. Some authors have found association between high parity and abruption while others did not. They found that the association was due to concomitant advancing age. Hibbard B.M and Jeffcoate T.N.A found an association of abruption with high parity.[50]

3. Race

Pritchard J.A and collegues found that the incidence was least common among Latin Americans (1 in 1473) and highest among African Americans (1 in 595) while in Whites the incidence was 1 in 876 deliveries.[52] According to Morgan M.A et al there was increased incidence among Black people with hypertension.[53]

4. Sex of the offspring

Karegard M and Krohn M found an increased incidence of abruption placenta in pregnancies with male offspring.[51,54] While Mortensen found no difference in incidence of abruptio placenta for male and female off springs.[55]

5. Nutrition

Naeye R.L. found that abruption was common in IUGR babies due to under nutrition during pregnancy.[33] Williams M.A found a positive correlation with low pre pregnancy body mass.[56] Kramer M.S found no relation between gestational weight gain, pre -pregnancy weight and abruptio placentae.[29]

6. Socio economic status

According to Krohn the incidence is high among unmarried women and those not living with husband.[54] Raymond found that the incidence was high among those with education less than 12 years.[57] But Kramer M.S found no such relationship.[29] Probably the increased risk may be related to increased parity and poor nutrition.

7. Hypertensive disorders in pregnancy

Hypertensive disorders in pregnancy i.e. chronic hypertension, chronic hypertension with superimposed preeclampsia and preeclampsia have all been found to be risk factors for placental abruption in many but not all studies.[19,20,26,27,29] In one study the rate of abruption among women with or without chronic hypertension was 1.56% and 0.6% in singleton pregnancies, respectively. After adjusting confounders women with chronic hypertension were at 2.4 fold increased risk for abruption.[26] In another study women with chronic hypertension had no increased risk for abruption (RR 1.4; 95% CI 0.53.6).[27]

Although chronic hypertension alone has not been a risk factor for placental abruption in all studies, chronic hypertension with superimposed preeclampsia has increased the risk for placental abruption 2.8 to 7.7 fold in several studies.[26,27] Severe preeclampsia is a strong risk factor for placental abruption.[20,27] The risk for abruption is further increased among women who have hypertensive disorder and who smoke.[20] In two previous Finnish studies chronic hypertension or PIH showed borderline association with placental abruption.[58,59] One of the two studies found strong association between preeclampsia and placental abruption.[59]

8. Maternal disease

a. Diabetes

Rasmussen S and Krohn M held the view that diabetes was responsible for placental dysfunction leading to various complications like preterm labour, IUGR, and abruptio placenta.[30,54] Morgan M A found that those with abruptio placenta and hypertension were more likely to have had diabetes mellitus compared to normotensive with abruption.[53]

b. Maternal Essential Hypertension and Nephritis

Maternal essential hypertension and nephritis were associated with abruptio placenta and the mechanism was similar to that of hypertensive disorders of pregnancy. Pritchard J.A et al found high incidence of abruption among those with hypertension.[52] Naeye R.L found no such relationship.[33]

c. Maternal genetic disease/Immune disorders

Inherited or acquired thrombophilias were found to be associated with abruption and infarction. According to Ananth CV et al several thrombophilic mutations have been identified in women with serious complications of pregnancy, including abruption placentae and fetal growth restriction as well as severe pre-eclampsia and still birth.[20] Several thrombophilias such as activated protein C resistance and the associated Factor V Leiden mutation, Prothrombin gene mutation, Methyltetrahydrofolate reductase have a high prevalence in women with placental abruption.

9. Premature rupture of membranes

Vintzelios A M found that there was increased incidence, 6.3%, in expectant management of preterm premature rupture of membranes (PROM), whereas with intact membranes it was only 2%.[60] Also those with oligohydramnios had a high risk among Preterm premature rupture of membranes. Gonen R et al found an incidence of 5.6% in 143 pregnancies less than 34 weeks when membranes were ruptured for more than 24 weeks.[61] Holmgren found that there was inverse correlation between gestational age at which PROM occurred and risk of abruptio placentae.[62]

10. Chorioamnionitis

Kramer M.S and Saftlas found an increased risk of abruption in chorioamnionitis.[23,29] Darby et al found that there was histological chorioamnionitis associated with preterm premature placental abruption.[63] But Wood D.L found no difference in incidence of abruption in those who had amniotic fluid infection and those who didn't have.[64]

11. Preterm labour

Abu Hejja and Graf Von Balles Trom found positive correlation between preterm delivery and abruption placenta.[65,66]

12. Small for gestational age

Kramer M.S and Krohn found an increased risk of abruption in cases of small for gestational age; probably this was related to placental dysfunction responsible for spectrum of complications like IUGR, preterm delivery and abruptio placenta.[29,54] Voigt L F did not find correlation between smoking and abruption placenta with small for gestational age. And small

for gestational age in abruption was due to placental dysfunction from premature placental separation.[67]

13. Folic acid deficiency

Hibbard B.M and Hibbard E.D found that folate deficiency was important in placental abruption but Pritchard J.A, Naeye R.L found no such correlation.[33,52,68] Maternal folic acid deficiency and megaloblastic anemia were found to be risk factors for abruptio placenta, but it was not proved whether they had common etiologic factor. Hibbard B.M and Jeffcoate T.N.A found abnormal morphology in the bone marrow in 63% and folic acid deficiency in 97.5% and concluded that folate deficiency was important etiologic factor.[50]

14. Smoking

In collaborative perinatal project, cigarette smoking was linked to risk of abruption according to Ananth C V, Charles Eagley and Robert Cefalo. They found that cigarette smoking to be an important etiological factor and two common lesions were noted in the placenta where the risk factor existed.[20,69] They were

1. Necrosis of deciduas basalis at the margin of the placenta.

2. Large retroplacental infarcts

Williams M A found dose response relationship between number of cigarettes and abruption.[56] According to Goujard et al smoking had vasoconstrictive effect on uteroplacental circulation and release of this spasm caused arterial rupture and bleeding.[70] Naeye R.L. found that the decidua at the edge of the placenta appears to be predisposed to subsequent necrosis. The effect of smoking

is thought to be due to nicotine or it may be related to relative deficiency of folic acid or vitamins and also found that maternal serum alfafetoprotein is elevated in mothers who smoke.[33]

15. Drugs

a. Cocaine

Gene Burkett showed an incidence of 14.3% and an increased risk of 2-3 folds if cocaine test is positive at delivery.[71] Roe D A found that placenta metabolized cocaine by choline esterase activity and those with abnormal enzyme activity had high risk of abruptio placenta and IUGR.[72]

b. Alcohol

In a study conducted it was found that ethanol consumption was related to abruption.[65]

16. Trauma

The most common traumatic events have been motor accidents with or without the use of seat belts.[73] But in developing countries, more cases are due to assault or due to fall. Kettel et al found that relatively minor trauma may cause fetal jeopardy which is not always associated with immediate evidence of placental separation. They advised at least 4 hours of monitoring to exclude subclinical abruption.[74]

17. Hydramnios

In normal pregnancy surface area of the uterus is reduced by 10% after rupture of membranes, while in hydramnios it may be as high as 30%. In those with small fetus this may be about 40%. The placenta which is inelastic remains as it is unlike the uterine wall to which it is

attached. This peels off the placenta from its attachment. This doesn't happen in normal cases where the reduction in surface area is only 10%.[65]

18. External cephalic version

Hibbard B.M and Hibbard E.D found a correlation between external cephalic version and abruptio placenta.[68]

19. Supine hypotension syndrome

Inferior venacaval compression was thought to cause abruption according to Mengert seen in those prior to caesarian delivery because of pressure of gravid uterus over inferior venacava, in supine position.[75] According to Pritchard J.A, supine hypotension syndrome is not a common cause of abruption as several women who had inferior venacava ligation for pulmonary embolism had no abruption.[52]

20. Short cord

While an unusually short cord has been held responsible for occasional cases of abruption, large studies have found no such relationship according to Pritchard J.A and Naeye R.L.[33,52]

21. Multiple gestation

Multiple gestation was found to be associated with abruptio placentae in various studies. Coyle et al reported that there were 10 cases of abruption among 443 cases of multiple gestation.[76] Ashar et al found one case of multiple gestation among 422 cases of abruption.[77]

22. Uterine tumours

Uterine leiomyomata if located behind placental implantation predisposes to abruption.[52] The incidence of leiomyoma was 1.4% in pregnancy according to Rice et al. Also he found that 8 of 14 women with retroplacental myomas developed abruption and by contrast abruption developed in only 2 of 79 women whose myomas were not retroplacental.[78]

23. Variation in placental anatomy

In circumvallete placenta, there is more or less a complete ring on the fetal surface of the placenta at some distance, 4cm from its margin. The ring divides the placenta into inner central and outer peripheral zone. There may be intermittent bleeding, due to separation from margin which may lead to abruption of placenta. Scott studied cases of placenta extrachorialis (Placenta marginata and placenta circumvallete). He found an incidence of 18% with mostly revealed type of hemorrhage.[79]

25. Previous history of abruption

Karegard M et al reported that recurrent abruption rate was increased to 10-fold.[51] James D.K et al found that after two episodes of abruption, the recurrence rate rise to 25%.[80] Hibbard B.M and Jeffcoate T.N.A found that some degree of abruption recurred in 17% of women with previous history of abruption.[50] Pritchard J.A found that about 7% of women with abruption severe enough to kill fetus will have same outcome in pregnancy.[52]

26. Miscellaneous

a. Short Labour

Mahon T R reported a case of abruption with precipitate labour.[81]

b. Snake Bite

Zergaid et al reported a case of abruption following snake bite.[82]

c. Increased MSAFP

Unexplained increased levels of MSAFP in 2nd trimester above 2 multiples of median in the absence of structural defects or amniotic fluid abnormality is associated with increased risk of abruption. This was thought to be due to placentation and leakage of fetal protein into maternal circulation. In these patients, the risk was aggravated by cigarette and hypertension. All these speculate that pathogenesis for abruption may be in place much earlier in pregnancy than previously thought.[69]

d. Hyperhomocysteinemia

In a study conducted by Steeger it was found that hyperhomocysteinemia was a risk factor.[83]

e. Ascorbic acid and Histamine

Ascorbic acid deficiency and subsequent increase in histamine was found to cause abruption according to Clemetson.[84]

CLASSIFICATION

Currently Sher's Classification is used to classify abruptio placenta.[85]

- Grade 0: Patient is asymptomatic and diagnosis is based on finding retroplacental clot on examination of placenta.
- Grade 1: Mild external bleeding; mild uterine tetany and tenderness may be present; but no evidence of maternal shock or fetal distress.
- Grade 2: Moderate to severe external bleeding; uterine tetany and tenderness present; fetal distress is seen but no evidence of maternal shock.
- Grade 3: External bleeding; marked uterine tetany and persistent abdominal pain; Maternal shock or coagulation defect and intrauterine demise.

CLINICAL FEATURES

Although the symptoms of placental abruption are typical and have been well described, they can vary considerably from one patient to another.[86] The onset of symptoms occurs before labour in more than half the cases, after the onset of labour in 35% and simultaneously with the onset of labour in 7% of cases. Clinically, patients with severe abruption, the symptoms are vaginal bleeding, pain abdomen, back ache (posteriorly located placenta), loss of fetal movements and episodes of collapse or giddiness. Examination reveals a tonically contracted uterus, uterine tenderness, non-reassuring fetal heart patterns or absent fetal heart sounds. Even if the amount of vaginal bleeding is minimal the patient may present in shock with a rapid and weak pulse, hypotension, cold and moist skin and stupor due to concealed hemorrhage which is not externally visible.[69] The majority of patients are asymptomatic until they experience sudden

onset of abdominal pain, vaginal bleeding or both, but 11% of patients have experienced vaginal bleeding 1 to 49 days earlier.[87]

Vaginal bleeding is present in 70-80% of cases. It is characteristically dark and non-clotting and sometimes intermixed with amniotic fluid when the membranes are ruptured. But there is little relationship between the amount of visible bleeding prior to delivery and the amount of placental separation, the amount of maternal hemorrhage or the degree of hypofibrinogenemia.[86,88] The amount of visible vaginal bleeding may be minimal even though the fetus is dead, the placenta is completely separated from the uterus, the mother is in shock and severe DIC exists.[52]

Uterine pain may be due to extravasation of blood into the myometrium, overdistension of the uterus due to retroplacental bleeding, or the frequent contractions associated with the release of prostaglandins. Severe pain abdomen is usually a manifest of massive concealed hemorrhage. This may be associated with a dead fetus. There is a correlation between the extent of placental separation and the risk of stillbirth. More than 50% of placenta should be involved to cause still birth. In case of a posteriorly located placenta, the woman complains of severe back pain.[89] Decreased fetal movements or absent fetal movements could be a sign of fetal distress which has to be taken seriously, especially in high risk women.

Clinical signs include shock, tachycardia with or without hypotension due to cardiac compensation or associated hypertension. The clinical signs of blood loss are out of proportion to the amount of vaginal bleeding. Uterine tenderness is present in 66% and tonic uterine contractions in 34% of case of abruption.[17] In case of a tonically contracted uterus the uterus is woody-hard to feel and tense without any intermittent relaxation as in normal labor. Typically,

there is uterine hypertonus with associated high-frequency (>5 in 10 mins), low-amplitude uterine contractions. In a case of advancing abruption, the abdominal girth or the uterine height increases progressively. Because labor is the most common factor precipitating abruption, nearly 50% of patients with placental abruption are in established labor. Several cases of silent abruption severe enough to be associated with fetal demise has been reported in cases where the placental site was the posterior wall of uterus and persistent low back pain was the only feature.[89] It is thus imperative that a sonogram be performed before a diagnosis of fetal death is made, because many of these fetuses with inaudible heart sounds are alive.

In severe abruption, disseminated intravascular coagulation (DIC) and shock are common features. In addition, intense systemic vasospasm commonly occurs and this may in turn cause the patient to become hypertensive or normotensive in spite of marked hypovolemia.[69]

DIAGNOSIS

The diagnosis of accidental hemorrhage is mainly clinical. The history and the clinical examination are the chief parameters in a pregnant woman with history of sudden onset of vaginal bleeding, pain, tenderness and a tetanically contracted uterus with or without fetal death, with or without shock. If pain, tenderness and tetanically contracted uterus are present, abruption is easily distinguished from placenta previa or local causes of vaginal bleeding. If vaginal bleeding is the only symptom, then a careful speculum examination should be performed to rule out local causes of vaginal bleeding and a careful ultrasound examination should be performed to rule out placenta previa. If the cause of bleeding is still has not been found after the above procedures, then there will be diagnostic and management dilemmas. The presence of albuminuria, hypertension, thrombocytopenia, or hypofibrinogenemia certainly should heighten

clinical suspicion of abruption as should hypotension out of proportion to observed blood loss. Even in the absence of any of these, the diagnosis of abruption placenta cannot be excluded. But because a diagnosis cannot be made, there is often a delay in aggressive treatment, especially if the gestation is less than 36 weeks and symptoms are so minimal that prolongation of pregnancy in hopes of improving the chance of neonatal survival is important.[90] It is in these cases that the visualization of a retroplacental hematoma by ultrasound may be helpful.

Placental abruption is often confirmed by gross examination of delivered placenta. In recent abruption the inspection of placenta demonstrates a craterlike depression on the maternal surface of the placenta covered by dark clotted blood, so called "delle".[44] In older abruptions fibrin deposits appear on the site of abruption.[25] A totally abrupted placenta may not differ on the maternal surface from a normal placenta at delivery.[44] Bleeding may occur into the uterine myometrium, leading to a purple colored uterus, so called Couvelaire uterus.[25] Such an uterus contracts poorly which can result in postpartum hemorrhage.[88]

Ultrasound

If placental abruption is suspected based on clinical symptoms, ultrasound examination is often performed in an attempt to visualize the extent of subchorionic or retroplacental hematoma. In some cases, placental abruption may be detected based on ultrasonographic findings even in asymptomatic patients.[37] The ultrasonographic appearance of abruption depends on the size and location as well as the age of the hematoma. The appearance of hematoma in the acute phase of abruption is from hyperechoic to isoechoic when compared with the placenta. When the hematoma resolves it becomes more hypoechoid within 1 week and sonolucent within 2 weeks.[91] Small abruptions or acute revealed abruptions are often not detectable by ultrasound.[25]

Concealed hemorrhage may be more easily seen by ultrasound. In one study ultrasound correctly diagnosed abruption only in 25% of cases. When a clot was visualized by ultrasound, the positive predictive value for abruption was 88%. Also, when a subchorionic or retroplacental hematoma was identified by ultrasound the management was more aggressive and perinatal outcome was worse. Although ultrasound is not accurate in the diagnosis of abruption it is useful in monitoring cases managed expectantly and in excluding coincident placenta previa.[88] Despite improvement in sonographic equipments the sensitivity of the diagnosis of abruption has not improved. A retroplacental hematoma is visualized sonographically as an anechoic collection between the placenta and uterine wall. As the hematoma becomes organized, its echogenicity may increase presenting internal echoes that may be difficult to distinguish from a degenerating leiomyoma. Nyberg D A reported different location of hematoma of which 81% were subchorionic, 60% retroplacental, 4% preplacental, also he found that acute hemorrhage was hyperechoic to isoechoic within one week and sonolucent within two weeks. So he said because of wide spectrum they could be overlooked or misdiagnosed.[91]

Cardiotocographic changes

In severe cases of placental abruption the fetus presents with heart rate abnormalities. A variety of fetal cardiotocographic (CTG) patterns have been described in association with placental abruption and fetal distress, and may include repetitive late or variable decelerations, decreased beattobeat variability, bradycardia, or sinusoidal fetal heart rate pattern.[25] Abnormal CTG in association with placental abruption predicts poor fetal outcome, even death. On the other hand, conservative expectant management seems to be safe in preterm pregnancies with placental abruption and normal CTG.[92]

Placental histopathology

Histopathology of abrupted placentas often shows evidence of acute and chronic lesions. Acute lesions include neutrophil infiltration of the chorionic plate and chronic lesions include placental infarcts in the decidua.[25] Chronic lesions develop due to a lack of adequate trophoblastic invasion.[93] Histological signs of chorioamnionitis and deciduitis with neutrophil infiltration are associated with placental abruption in one third of the cases.[14,94] Acute atherosis in spiral arteries leads to distinctive necrotizing decidual lesions ultimately leading to vascular thrombosis, placental infarcts and fibrin deposits.[25,44,63,94] Intervillous thrombosis results from intraplacental hemorrhage from villous capillaries and is associated with chorionic villous hemorrhage. Intervillous thrombosis is more common in smoking women with placental abruption. This may further reduce uteroplacental and fetal blood flow leading to chronic underperfusion. Chronic hypoxia is manifested by increased villous fibrosis and trophoblast knotting.[94] One study found that necrosis in the decidua basalis at the margin of the placenta was most frequent in smoking women suggesting that such necrosis could initiate placental abruption.[33]

DIFFERENTIAL DIAGNOSIS

Conditions other than placenta previa which mimics abruption placenta include

1. Acute hydramnios

2. Tonic contraction of uterus

3. Rupture of uterus especially incomplete

4. Other surgical acute abdominal conditions

5. Anterior abdominal wall hematoma and rectus sheath hematoma

6. Degenerating fibroid

In acute hydramnios, sudden distension of abdomen may give rise to increase in pulse rate and a mild degree of shock. Unless the patient is already anemic, usually there is no pallor as in accidental hemorrhage and in both, neither fetal parts are easily palpable nor are the fetal heart sounds heard. If os is open vaginal examination will reveal a very tense bag of membranes in hydramnios. It is difficult at times to distinguish accidental hemorrhage, especially if it occurs in labour from an incomplete rupture of uterus. In the latter there may be history of prolonged obstructed labour, history of uterine scar. Uterus is tonically contracted, tender, lower segment stretched out, fetal heart sound present or absent. Vaginal examination may reveal jammed presenting part and caput in cases of obstructed labour causing rupture. Sometimes rupture of abdominal viscus, torsion of ovary may give rise to signs and symptoms of hemorrhage and shock similar to those in abruption. Careful examination of the patient and detailed history help in arriving at diagnosis. Occasionally laparotomy may be the safest when the diagnosis is uncertain.

COMPLICATIONS

1. Maternal complications includes:
    - Hypovolemic shock
    - Acute renal failure
    - Disseminated Intravascular Coagulation
    - Postpartum Hemorrhage
    - Sheehan’s syndrome

- Maternal mortality
- Fetomaternal hemorrhage
- Recurrence in the subsequent pregnancy

2. Fetal complications includes:
    - Fetal growth restriction
    - Still births
    - Neonatal death

1. Hypovolemic Shock

Hypovolemic shock is mainly due to hemorrhage either revealed/ concealed. In severe abruption, there may be intense systemic vasospasm and the patient may become hypertensive or normotensive in spite of marked hypovolemia. This may lead to under treatment.[69] Prevoiusly hypertension patients may be close to shock in spite of having normal BP and pulse rate.[95] Normal pulse rate and BP may be found with blood loss of 35% of maternal blood volume. Tachycardia and pallor indicate 40-50% of maternal blood volume lost. External bleeding may be minimal while retroplacental bleeding may be extensive.[96] Pritchard J A demonstrated that if abruptio placentae was severe enough to kill a fetus, the average intrapartum blood loss, mostly retroplacental is about 2500 ml.[52] Supposedly, thromboplastin from deciduas and placenta enters maternal circulation and incited intravascular coagulation and other features of amniotic fluid embolism syndrome, including hypotension. This sequence is rare, and the intensity of shock is seldom out of proportion to blood loss.[32]

2. Acute renal failure

The renal failure is mainly due to hypovolemia.[52] It was found that more than half of the women who died due to abruption at autopsy were found to have bilateral cortical necrosis according to Nilsen, while others had lower nephron necrosis.[97] The renal failure is related to prolonged maternal hypovolemia and is largely preventable by appropriate fluid replacement and central venous pressure monitoring. Other postulated mechanism is due to severe and prolonged spasm of the glomerular vessels which results in severe anoxia and death of glomeruli. Spasm may be due to toxin liberated by the placenta. Acute tubular necrosis is the renal lesion more commonly encountered. The important cause is impaired renal function due to both decreased cardiac output and intrarenal spasm. And sometimes associated with acute and chronic hypertensive disorders which themselves produce vasospasm independent of hypovolemia.

3. Dissiminated Intravascular Coagulation

Abruption is one of the most common causes of disseminated intravascular coagulation in obstetrics. Thromboplastin enters the maternal circulation from the deciduas and causes DIC. The greater the degree of placental separation, more likely the development of a serious coagulation defect. Hypofibrinogenemia below 100mg/dl occurs in 4-10% of abruption cases but it occurs in about 30% of cases of abruption severe enough to cause fetal death.[72] Evidence of DIC should be sought in any patient with abruption placenta. Prolonged bleeding from venopuncture sites indicates serious hypofibrinogenemia. A serum fibrinogen level below 100mg/dl or fibrin degradation products above 10mg/ml confirms the diagnosis. A prolonged thrombin time indicates either hypofibrinogenemia or increased levels of FDP. While awaiting the results of these tests, a quick method of evaluating the patient for DIC is to observe her

freshly drawn blood in a test tube. Failure of the blood to clot in 8 min indicates decreased fibrinogen. By the end of 1 hour the clot should retract from the sides of the tube. Furthermore, if the clot dissolves within one hour it is likely that the patient has excessive fibrinolysis.[69] Of all obstetric complications, the combination of DIC and abruption tends to be the most lethal with nearly uniform perinatal mortality and very high maternal mortality.[98] When DIC does develop, it usually develops quite rapidly, nearly always within 8 hours of the onset of clinical symptoms of abruption. Conversely, when the fibrionogen concentration is above 150mg/dl six to eight hours after the onset of clinical symptoms of abruption, serious hypofibrinogenemia usually does not develop even if delivery is delayed for several more hours.[52] It is believed that the DIC is initiated by release of thromboplastin from the deciduas at the separation site. This decidua is extremely rich in thromboplastin. At the same time, a fibrinolytic mechanism lyses much of the fibrin generated in the maternal circulation. It has become clear that the fibrinolysis is a secondary event to intravascular coagulation in that all patients showing increased fibrinolytic activity have hypofibrinogenemia while pure fibrinolysis (increased fibrin degradation products) occurs only in a small number of cases.[52] Cause of hypofibrinogenemia in abruption may be due to any of the following causes:

- Consumption of fibrinogen by deposition in the retroplacental region.
- Primary fibrinolysis
- DIC consuming fibrinogen
- Dysfibrinogenemia
- Thromboplastinemia and intravascular coagulation.

4. Post partum hemorrhage

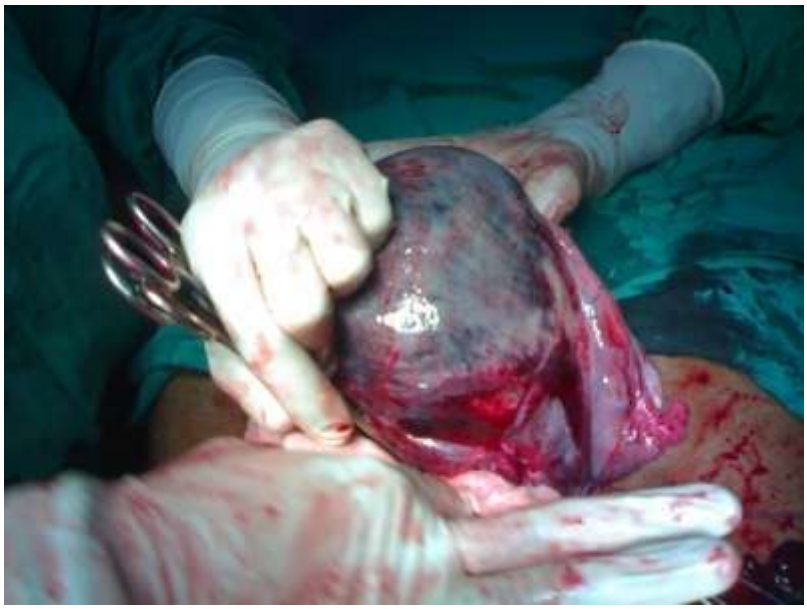
After delivery the contracted myometrium normally compresses the maternal vascular sinuses, but approximately 27% of patients with the combination of couvelaire uterus and DIC may develop PPH. The occurrence of post partum uterine bleeding is not related to the degree of hypofibrinogenemia, but is associated with an increase in fibrin degradation products (fibrinolysis). The presence of early products of fibrinogen proteolysis (fragments X or Y) is almost invariably associated with post partum hemorrhage, whereas the presence of late products (fragments D or E) alone is not strongly associated with post partum hemorrhage. In vitro contractility of myometrial strips removed from patients in labour is completely inhibited when exposed to fibrin degradation products. Basu H.K also noted increase fibrinolytic activity in the uterus which had high fibrin degradation product. So in effect, FDP were produced by local uterine fibrinolysis.[99] Initial resuscitative measures are done to control PPH. The management of severe intractable PPH is cesarean hysterectomy.

5. Feto maternal hemorrhage

The bleeding with placental abruption is almost always maternal. In non traumatic placental abruption, evidence of fetomaternal hemorrhage was seen in 20%. However, in all instances it was less than 10ml. Fetomaternal hemorrhage was more in traumatic abruption and fetal bleeding that averaged 12 ml in non catastrophic trauma.[100] Cardwell M.S found 75% of fetomaternal hemorrhage in non catastrophic abruptio placenta consistent with ultrasound findings.[101]

6. Couvelaire uterus/Uteroplacental apoplexy

Couvelaire uterus is a life threatening condition, wherein, the retroplacenta blood penetrates through the myometrium forcing its way in to the peritoneal cavity. The uterus is bluish/purplish in colour with mottled appearance. Effusion is occasionally seen beneath tubal serosa, the connective tissue of the broad ligament and in the substance of ovaries as well as peritoneal cavity.[32] The patient will have uterine tetany. The myometrium either ruptures due to increased intrauterine pressure associated with uterine contractions or it may get exhausted. Up on delivery of the fetus the uterus may not contract and retract adequately due to the interfering blood in the myometrium which may end up in atonic post partum hemorrhage. This so called uteroplacental apoplexy which was first described by Couvelaire in 1911 is now called couvelaire uterus. The treatment of couvelaire uterus is immediate delivery of the fetus and stimulation of uterine contractions with oxytocics. In worst cases, where the uterus is not responding to any of the medical or surgical treatment, hysterectomy is performed.



Photograph 1: Picture showing Couvelaire uterus

7. Sheehan's syndrome

Few cases of abruption who survived will later manifest with signs of panhypopituitarism and may present with failure of lactation, amenorrhoea, and intolerance to cold, hypogonadism. It has been related to prolonged periods of shock.

8. Maternal mortality and morbidity

The cause of maternal death is mainly due to hypovolemic shock, renal failure, DIC, post partum hemorrhage, etc. The maternal mortality has fallen from nearly 10% early in this century to well under 1% today.[102] The reduced maternal mortality is due to early diagnosi, vigourous correction of hypovolemia with transfusion of blood and blood products, prevention of complications like DIC, renal failure and early delivery within 8-10 hours of admission.

9. Perinatal mortality and morbidity

Placental abruption causes increased perinatal mortality (20-30%), preterm delivery and intrauterine growth restriction. Most perinatal loses are due to intrauterine death before admission, whereas, neonatal deaths are mainly due to prematurity. Fetuses die in utero due to acute anoxia caused by detachment of placenta and due to hypertonicity/hypotension associated with abruption both of which cause uteroplacental insufficiency. Abdella T.N et al found 14% of infants which survived had neurological deficits within first year of life. [103]

MANAGEMENT

Management depends on status of mother and fetus, associated conditions and gestational age and stage of labor. In cases with moderate or severe abruptio placentae and where the diagnosis is clear, the principles of management are stabilization of the mother and early delivery of the baby. When the diagnosis is uncertain (e.g., antepartum hemorrhage of uncertain origin) or when the retroplacental clot is small, self-limited, and asymptomatic, treatment may be individualized taking the gestational age into account.

Principles in management includes

1. Maternal resuscitation
2. Monitoring of mother and fetus
3. Early diagnosis and treatment of complications
4. Early delivery

Maternal resuscitation

A rapid evaluation of the maternal condition should be done. The vital signs must be monitored frequently with particular attention to the maternal heart rate, as underlying hypertension can mask concealed blood loss. Fetal death indicates a larger blood loss with a high chance of associated complications, such as shock, DIC, and renal failure. A retrospective study of 96 cases of abruption with fetal death found that 53% developed major complications, but that with careful tertiary management, the impact could be reduced.[100]

In severe abruption, it is necessary to establish a clear airway and administer oxygen. Restoration of maternal circulating volume should be the next priority. The first step is to setup

two intravenous lines of large bore. Blood should be taken for complete blood count, coagulation studies and type and cross-match, and the blood bank should be informed of the potential for coagulopathy. A Foley catheter should be placed and the hourly urine output should be monitored closely.

Initial resuscitation is with crystalloid solution 1 to 2 L if there are any signs of hypovolemia, after which blood components (usually packed red cells and fresh frozen plasma) are used as required. Two to three ml of crystalloid solution should be given for each ml of blood loss to maintain normovolemia. A Central venous pressure line must be used to gauge the optimum transfusion to prevent undertransfusion leading to renal shutdown or overtransfusion causing pulmonary edema. Blood should then be transfused to keep the hemotocrit above 30% and the urinary output above 30 ml per hour. A useful rule is that an abruption severe enough to cause fetal death merits at least 2 units of blood replacement or trebling the volume of blood clot gives rough estimate.

The key to prevention of complications of DIC or renal failure is vigorous blood and fluid replacement to combat hypovalemic shock. If DIC occurs the management should be done in consultation with the hematologist. Coagulation studies at regular intervals until the patient is stable. The main stay of treatment is delivery of the baby followed by replacement of blood and cloting factors. Delivery can only halt the process of DIC. Clear evidence of spontaneous resolution after delivery has been presented.[52] Fresh blood is the ideal replacement as it contains red cells, clotting factors and fibrinogen. Packed cells are usually transfused along with fresh frozen plasma or cryoprecipitate to provide clotting factors.

Monitoring

Clinical monitoring and investigations

Cardiovascular system: Pulse rate every 15 minutes, blood pressure every 15 minutes (continuous non-invasive recording if possible), central venus pressure every half hourly.

Respiratory System: Auscultation of lung bases, respiratory rate every half hourly

Uterine contraction every half hourly and fetal heart rate every 15 minutes. It is better to use a continuous electronic fetal monitoring to note the fetal heart rate pattern.

Acid - base status (pH, blood gases analysis)

Renal Function: Urine output and testing for protein - hourly, urine specific gravity - hourly, urine microscopy for casts, blood urea and electrolytes (initially) and every four hourly. Further renal function tests are necessary in patients with renal failure.

Hemotological function: Hemoglobin concentration and hematrocrit four hourly, coagulation profile (crude clotting time, fibrinolysin test, prothrombin time, partial thromboplastin time, thrombin time, fibronogen, D-dimer, FDP, platelet count, four hourly. Weiners clot observation test – 5 ml of venous is kept in a 15 ml of dry test tube at room temperature. If blood clot forms within 6 minutes it suggests that blood fibrinogen levels is above 150 mg/dl and if the clot is not formed even after 30 min, the fibrinogen level is less than 100 mg/dl).

Early delivery

Early delivery by quickest route is vital. The management of placental abruption depends on the severity of presentation, the gestational age, severity, presence of complications, state of the fetus, whether live or dead.

When the fetus is alive and viable, and the diagnosis of moderate or severe abruptio placentae is clear, delivery should be expedited. Any sign of fetal distress delivery by cesarean section is required. However, if the heart rate tracing is normal and the uterus relaxes between contractions, vaginal delivery may be attempted. No specific time limit for delivery need be applied as long as continuous fetal and intensive maternal surveillance reveals no deterioration and labor progresses normally. In case the placental abruption is progressive it is better to deliver by cesarean section unless labor is far advanced.[104,105,106,107,108,109] In case the woman is in DIC blood product replacement and delay in delivery until hematologic parameters have improved are generally associated with good maternal outcomes.

If the fetus is alive on admission, there is a close association between diagnosis to delivery time and perinatal mortality.[52,110] Knab noted that most of the post admission fetal deaths occurred in fetuses delivered more than two hours after admission. A short interval from admission to delivery within 3 hours of separation of placenta could reduce fetal mortality by 50%.[111] It is important to remember that both the maternal and fetal conditions can deteriorate rapidly and indecision may lead to fetal death. Therefore cesarean section is recommended for live fetuses with abruption when vaginal delivery is not imminent and it should be performed soon after maternal resuscitation before the developement of fetal distress.

When managing patients with severe placental abruptions and fetal demise, maintenance of maternal volume status and replacement of blood products is essential. In cases of severe abruption with fetal death, regardless of gestational age, as long as the mother is stable, it is reasonable, in the absence of other contraindications, to allow the patient to have a vaginal delivery and vaginal delivery is preferable unless there is persistent hemorrhage with slow progress of labor in which case, caesarean delivery should be performed. The fetal membranes should be ruptured as soon as possible to decrease the intrauterine pressure, which causes dissemination of thromboplastins into the maternal circulation, and to hasten labor. Oxytocin should only be used after careful consideration due to the risk of overstimulation and uterine rupture. In the event of the cervix being unfavourable vaginal prostaglandins may be used.

After delivery, the patient should be monitored closely, with particular attention paid to vital signs, amount of blood loss, and urine output. In addition, the uterus should be observed closely to ensure that it remains contracted and is not increasing in size, and blood loss should be monitored closely. An oxytocin infusion will help keep the uterus well contracted, thereby avoiding postpartum hemorrhage.

Expectant management

In selected cases prolongation of pregnancy is helpful to achieve fetal lung maturity. Sholl managed 72 women with pregnancy between 26 to 37 weeks who were clinically diagnosed with placental abruption, 50% delivered within 3 days, either because of significant hemorrhage or fetal distress or both. Interestingly, caesarean rate was 50% for those who delivered soon after admission and in those who were postponed for at least 3 days.[112] Coombs concluded that mild abruption is self limited, so expectant management could be done with

regular fetal heart monitoring and serial ultrasound and coagulation profile.[113] Lack of ominous decelerations does not guarantee the safety of intrauterine environment for any period of time. The placenta may further separate at any instant and seriously compromise or kill the fetus, unless delivered immediately.[50] In such cases caesarean delivery was advocated by many authors. Hurd et al found abruption if unrecognized for longer periods, if tocolysis was done, may prove dangerous, but Sholl and Coombs advocated tocolysis in selected groups with preterm abruption.[91,112,113] Towers used magnesium sulphate and terbutaline in those under 36 weeks and perinatal mortality was 5% and was equal to non treated group and concluded that randomized trial could be conducted safely using tocolytics.[114] Cunningham et al also contradicts tocolysis in case of abruption and advocates further trials.[50] However, in current day obstetrics there is very little role for expectant management of abruptio placenta. Expectant management is considered in mild placental abruption occurring before 37 weeks of gestation, slight vaginal bleeding, mild abdominal pain and the patient is hemodynamically stable.[80]

MATERIALS AND METHODS

Study group consisted of all pregnant women diagnosed with abruptio placenta with gestational age more than or equal to 28 weeks admitted at R. L. Jalappa Hospital and Research Center attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, were taken prospectively during the period January 2012 to August 2013.

On admission a detailed history of all the patients including age, parity, period of gestation, vaginal bleeding, pain abdomen, fetal movements, history of trauma, history suggestive of hypertensive disorder, previous medical disorders and history of outcome of previous pregnancy was taken.

Clinical examination was done and vitals were recorded. Abdominal examination was done documenting the fundal height, state of the uterus whether it is tense and tender and uterine contractions, lie and presentation of the fetus and fetal heart sounds were recorded.

Diagnosis of abruptio placenta was done based on clinical signs and symptoms and also supplemented by ultrasonography.

After ruling out placenta previa by ultrasound examination, vaginal examination was done to note the amount of vaginal bleeding, cervical effacement, dilatation, membrane status, presence of any blood stained liquor, presenting part, position, station and adequacy of pelvis. Grading of abruption was done on the basis of Sher's classification (1985).

In all cases complete blood count, urine for protein, blood grouping and typing, bleeding time, clotting time, coagulation profile and renal function test were done.

Patients were initially stabilized hemodynamically and delivered early either by vaginally or by Caesarean section depending on maternal and fetal status.

Details of mode of delivery and admission to delivery interval were noted down.

Maternal complications include post partum hemorrhage, couvelaire uterus, acute renal failure, disseminated intravascular coagulation were recorded. Fetal outcome in terms of Apgar score and perinatal death were documented.

Inclusion criteria

All mothers with gestational age ≥28 weeks with abruptio placenta

Exclusion criteria

- Multiple pregnancy
- Previous caesarean section
- Congenital malformations of fetus
- Cardiac disease with pregnancy
- Diabetes mellitus in pregnancy

STATISTICAL ANALYSIS

Data was coded and entered in to excel data sheet and analysed using EPI INFO 7 software. Frequencies and proportions were computed for qualitative data. Chi-square is the test of significance for categorical data. $p < 0.05$ is considered as statistically significant.

RESULTS

During the study period (Jan 2012– March 2013) there were a total of 4,209 deliveries. Of these there were 117 cases of abruptio placenta with incidence of 2.52%. Out of 117 cases, 100 cases fulfilled the inclusion criteria and were included in the study.

Table 1: Age distribution

Age	n= 100	Percentage
≤ 20 years	17	17%
21- 25 years	50	50%
26 -30 years	28	28%
> 30 years	5	5%

Table 1 shows the prevalence of abruptio placenta in different age groups. Out of 100 cases of abruptio placenta 50 (50%) were in the age group 21-25 years, 28 (28%) were in the age group of 26-30 years, 17 (17%) in the age group ≤ 20 years and > 30 years were 5 (5%).

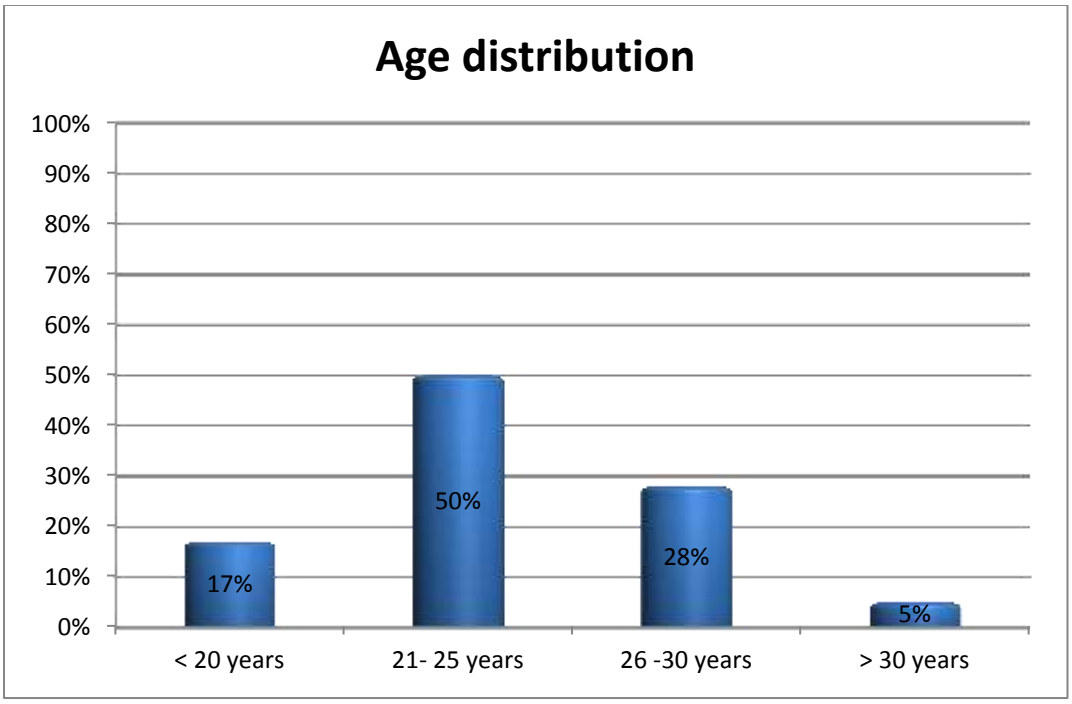


Figure 1: Age distribution

Table 2: Parity distribution

Gravida	n=100	Percentage
1	42	42%
2	26	26%
3	19	19%
≥ 4	13	13%

Table 2 shows the parity distribution of women with abruptio placenta. The highest prevalence is seen in primigravida 42 (42%) and in gravida 2: 26 (26%), gravid 3: 19 (19%) and gravid ≥4: 13 (13%).

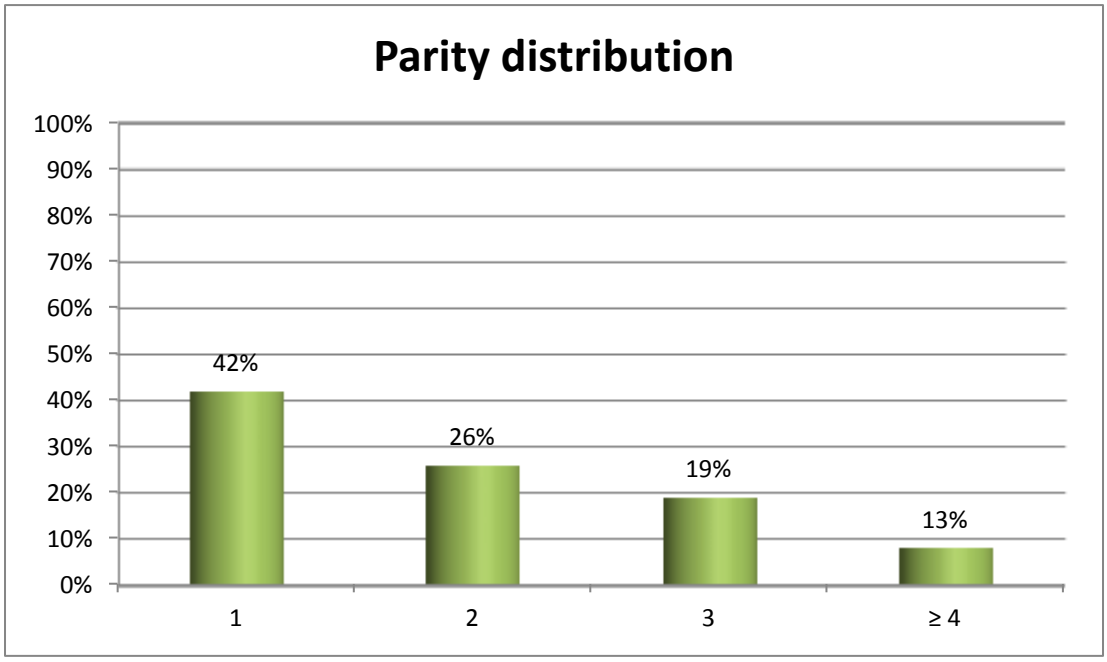


Figure 2: Parity distribution

Table 3: Gestational age at presentation

Gestational age	n=100	Percentage
28-33 weeks	37	37%
34-36 weeks	27	27%
≥ 37 weeks	36	36%

Table 3 shows the distribution of women based on the gestational age at presentation. Out of 100 cases 37 (37%) presented at gestational age between 28-33 weeks. Women with gestational age ≥ 37 weeks included 36 (36%) and 27 (27%) of women in the gestational age group of 34-36 weeks.

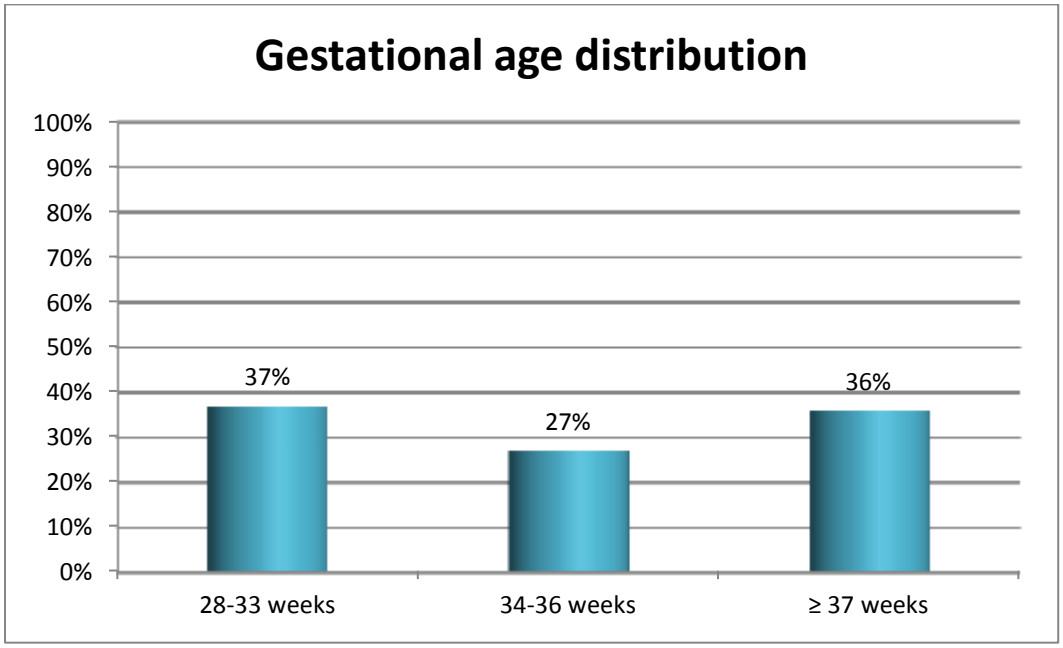


Figure 3: Gestational age at presentation

Table 4: Associated conditions with abruptio placenta

Associated condition	n=117	Percentage
Pre-eclampsia/ eclampsia	34	29.05%
Anemia	24	20.5%
Previous LSCS	11	9.4%
PROM	5	4.27%
Oligohydramnios	3	2.56%
Multiple pregnancy	2	1.7%

Table 4 shows the associated conditions with abruptio placenta and include Pre-eclampsia/eclampsia 34 (29.05%), anemia 24 (20.5%), previous LSCS 11 (9.4%), PROM 5 (4.27%), oligohydramnios 3 (2.56%) and multiple pregnancy 2 (1.7%) of cases.

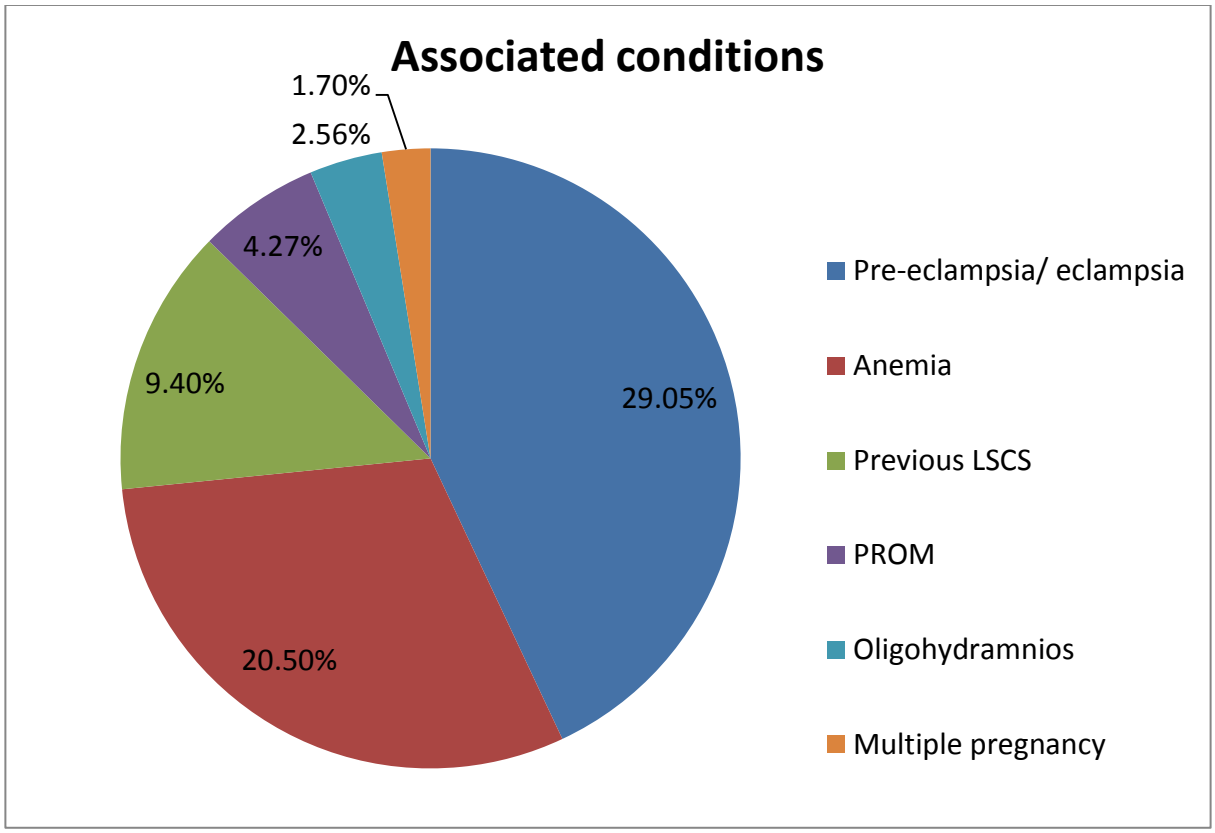


Figure 4: Associated conditions

Table 5: Type of abruptio placenta

Type of abruption	n=100	Percentage (%)
Revealed	9	9%
Mixed	68	68%
Concealed	23	23%

Table 5 shows the distribution of cases based on type of abruptio placenta. Majority of the cases were mixed type 68 (68%), concealed was 23 (23%) and revealed types 9 (9%).

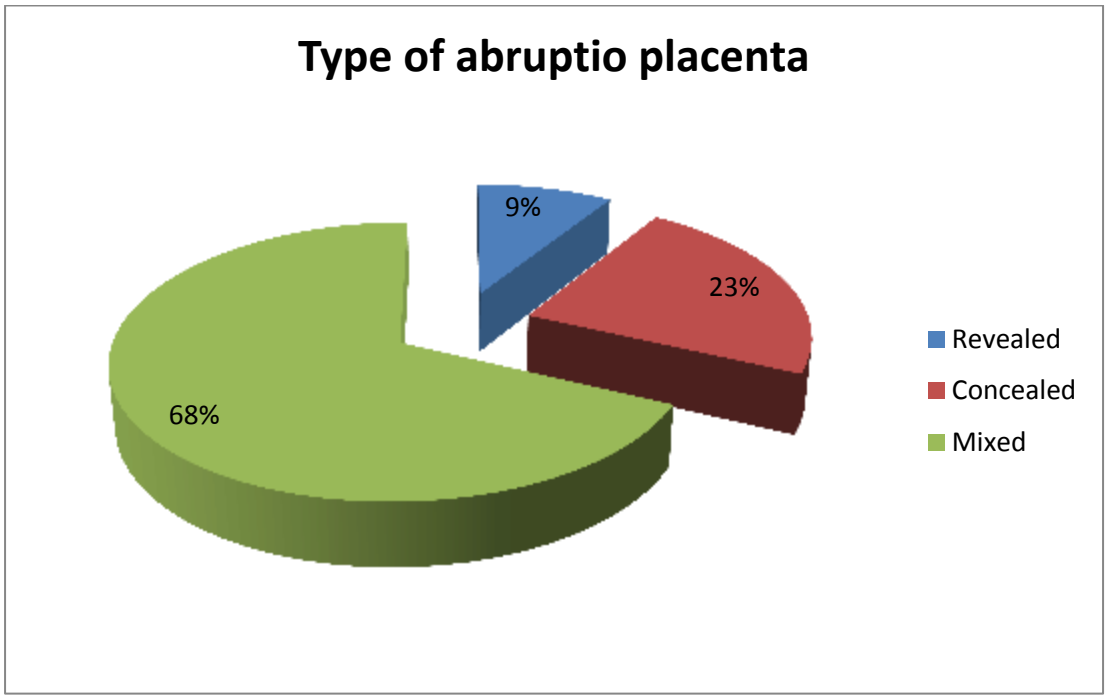


Figure 5: Type of abruptio placenta

Table 6: Grades of abruptio placenta

Grade of abruption	n=100	Percentage
Grade 0	3	3%
Grade 1	17	17%
Grade 2	27	27%
Grade 3	53	53%

Table 6 shows the distribution of cases based on the Grade of abruptio placenta. Grade 0 3 (3%), Grade 1 17 (17%), Grade 2 27 (27%) and Grade 3 53 (53%) of cases.

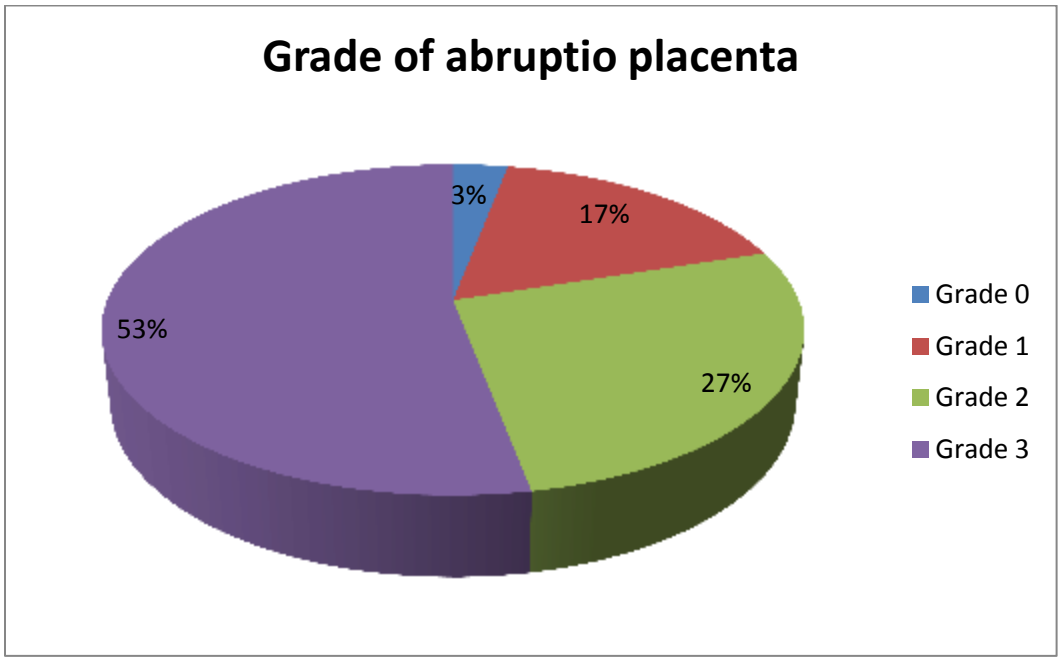


Figure 6: Grades of abruptio placenta

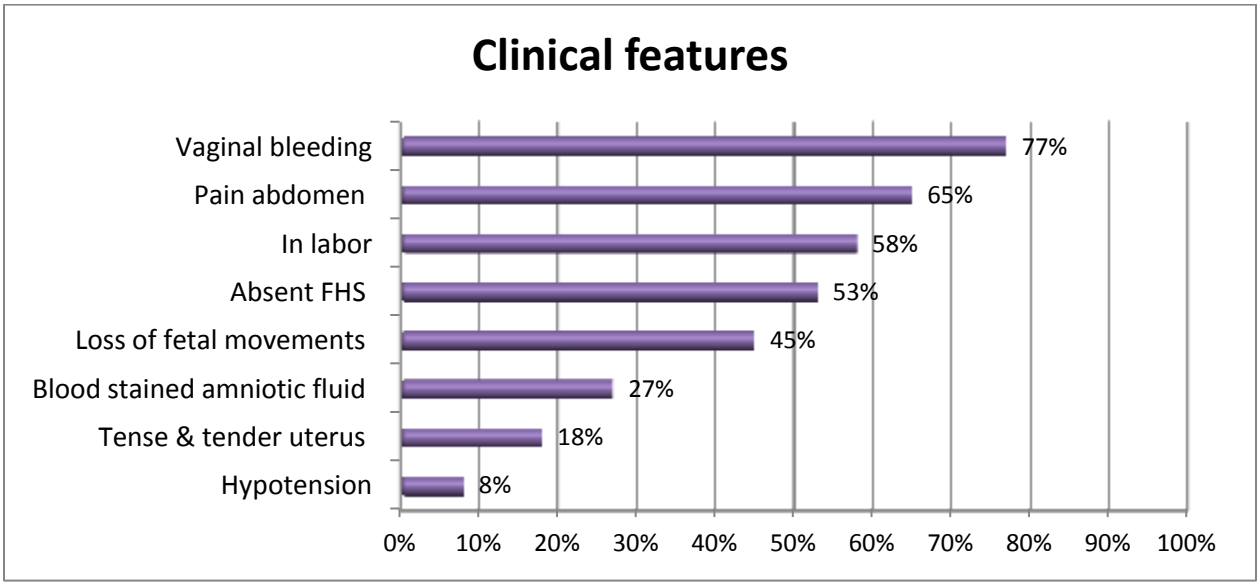


Figure 7: Clinical features

Figure 7 shows the clinical features in women with abruptio placenta. Out of 100 cases, 77 (77%) cases presented with vaginal bleeding, pain abdomen 65 (65%), loss of fetal movements 45 (45%), tense and tender abdomen 18 (18%), dead fetus 53 (53%), blood stained amniotic fluid 27 (27%), hypotension 8 (8%) and 58 (58%) already in labor.

Clinical diagnosis was made in 77 (77%) of cases and sonographic diagnosis supported in 23 (23%) of cases.

Table 7: Mode of delivery and fetal outcome in Grade 1 and 2 of abruptio placenta (n=44)

Mode of delivery	Vaginal delivery (n=27)	Cesarean section (n=17)	p value
Live borns	23 (85.2%)	15 (88.2%)	χ^2=1.032 df=1 p=0.596
Still borns	4 (14.8%)	2 (11.7%)	
Apgar score < 7 at 1 min	6 (22.2%)	7 (41.2%)	

Table 7 shows the mode of delivery and the fetal outcome in Grade 1 and 2 of abruptio placenta. Twenty seven women had vaginal delivery and 17 women underwent cesarean section. In those women delivered vaginally, live borns were 23 (85.2%), still borns were 4 (14.8%) and 1 minute Apgar score < 7 in 6 (22.2%) compared with 15 (88.2%), 2 (11.7%) and 7 (41.2%), respectively with cesarean section group. There was no statistical significance between the two groups.

Out of 53 (53%) cases Grade 3 abruptio placenta, 50 (94.33%) delivered vaginally and 3 (5.66%) underwent cesarean section. The indication for cesarean section in the first case was primigravida with footling presentation with contracted pelvis. In the second case the indication was for fetal distress and in the third case it was for severe abruptio placenta with intrauterine fetal demise with non-progression of labor.

Table 8: Admission to delivery interval and fetal outcome with vaginal delivery in Grade 1 and 2 abruption (n=27)

Admission to delivery interval	Live borns (n=23)	Still borns (n=4)	p value
≤ 8 hours	16 (59.6%)	2 (7.4%)	χ^2=0.587 df=1 p=0.4436
‘> 8 hours	7 (25.9%)	2 (7.4%)	

Table 8 shows the fetal outcome with vaginal delivery based on the admission to delivery interval in Grade 1 & 2 abruptio placenta. In the babies delivered before 8 hours of admission there were 16 (59.6%) live borns and 2 (7.4%) still borns and in the babies delivered after 8 hours there were 7 (25.9%) live borns and 2 (7.4%) still borns. Though the live borns delivered before 8 hours were more it was not statistically significant.

Table 9: Admission to delivery interval and fetal outcome with cesarean section in Grade 1 and 2 abruption (n=17)

Admission to delivery interval	Live borns (n=15)	Still borns (n=2)	p value
≤ 8 hours	14 (82.3%)	2 (11.7%)	χ^2=0.1417 df=1 p=0.7066
‘> 8 hours	1 (5.8%)	-	

Table 9 shows the fetal outcome with cesarean section based on the admission to delivery interval in Grade 1 & 2 abruptio placenta. In the babies delivered before 8 hours of admission there were 14 (82.3%) live borns and 2 (11.7%) still borns and in the babies delivered after 8 hours there were 1 (5.8%) live born and no still borns. Though the live borns delivered before 8 hours were more it was not statistically significant.

Table 10: Maternal complications

Complication	n = 100	Percentage
PPH	7	7%
Couvelaire uterus	7	7%
DIC	3	3%
Acute renal failure	3	3%

Table 10 shows the maternal complications of abruptio placenta. Post-partum hemorrhage was found in 7 (7%), couvelaire uterus 7 (7%), DIC 3 (3%) and acute renal failure 3 (3%) of cases. None of the patients required cesarean hysterectomy.

In Grade 3 abruptio placenta 11 (11%) women delivered beyond 12 hours. Out of these 11 women, 1 developed HELLP syndrome due to pre-eclampsia and 1 had hypovolemic shock. Four cases delivered more than 24 hours later and the maximum duration of delivery from admission in Grade 3 abruption was 34 hours without any maternal complication.

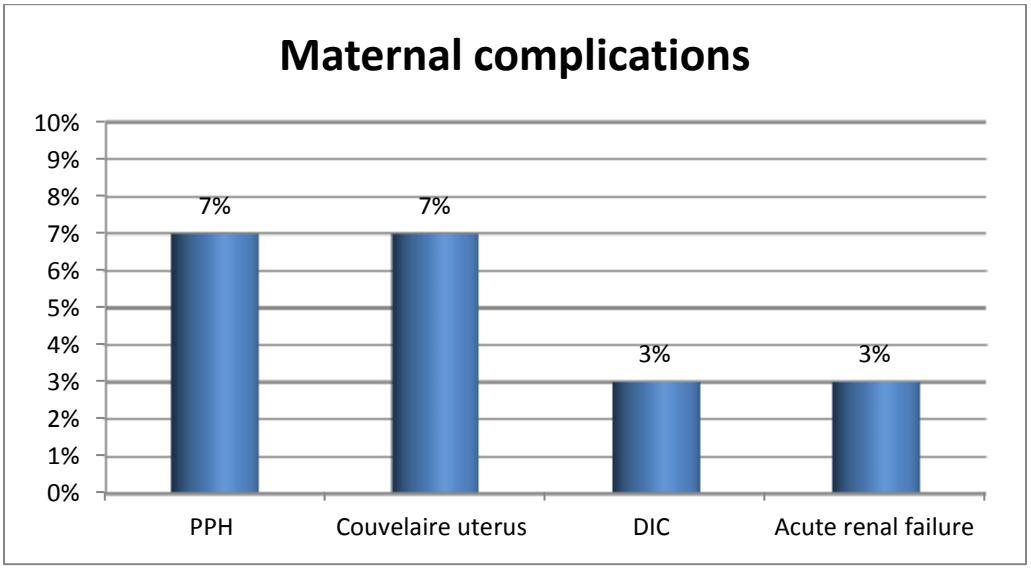


Figure 8: Maternal complications

Table 11: Fetal outcome

Fetal outcome	n=100	Percentage
Live births	42	42%
Still births	58	58%

Table 11 shows the perinatal outcome of the babies. Out of 100 cases, there were 42 (42%) live born and 58 (58%) were still borns. There were 64 (64%) premature babies.

DISCUSSION

In the present study there were a total of 4,209 deliveries. Of these there were 117 cases of abruptio placenta, incidence being 2.52%. Similar incidence was found by Purandare et al, Krishna Menon et al, Pariente et al and Hossain et al in their studies as shown in the table below.[115,116,117,8] In studies by Siddiqui and Bibi the incidence was found to be a little high, 6.2% and 4.7%, respectively.[118,119] Trends on the rates of abruptio placenta over a period of time found that the rate has increased from 0.81% to 1.0% from 1981 to 2001.[4]

Table 12: Comparison of incidence of abruptio placenta with different studies

Study	Incidence
Purandare et al[115]	0.63%
Krishna Menon et al[116]	1.8%
Pariente et al[117]	0.7%
Hossain et al[8]	3.75%
Siddiqui et al[118]	6.2%
Bibi et al[119]	4.7%
Present study	**2.52%**

As seen in table 12, the highest incidence of abruption placenta was in the age group 21-25 years accounting for 50%. The incidence in the age group > 30 years is 5%. This could be because the younger age group formed the larger subset of women who deliver in this age group. Similar results were found in studies done by Mondal et al, Siddiqui et al & Sarwar et al, whereas, study conducted by Ananth et al showed that abruption placenta was more common in

age more than 35 years.[120,118,121,13] As this study was conducted in the west, advanced maternal age at marriage could be one possible explanation.

Grandmultiparity has been shown to be associated with abruptio placenta in the study conducted by Ananth et al.[13] Also, Sarwar et al and Hossain et al found that multiparous women had more abruptio placenta.[8,121] But in the present study the incidence was high (42%) among the primigravida.

Studies conducted by Parikh et al and Siddiqui et al showed that most of the women with abruptio placenta presented with gestational age above 37 weeks and the study conducted by Bibi et al showed that more than 50% were less than 32 weeks of gestation at the time of presentation.[122,118,119] Our study showed similar incidence in both these groups, but it is 27% in the gestational age group of 34-36 weeks. This was inconsistent with the study conducted by Menon et al who found maximum incidence in the gestational age of 33-36 weeks.[116]

The common associated conditions with abruptio placenta in the current study were pre-eclampsia/eclampsia (29.05%), anemia (20.5%), previous cesarean section (9.4%), premature rupture of membranes (4.27%), oligohydramnios (2.56%) and multiple pregnancy (1.7%). Similarly in studies conducted by Pitaphrom et al and Matsuda et al the incidence of pre-eclampsia/eclampsia was found to be 30.2% and 37.2%, respectively.[123,124] According to Sibai et al, the incidence of hypertensive disease in pregnancy associated with abruption placenta recorded a wide range of incidence of 4.1-22.9%.[125]

The incidence of PROM in abruptio placenta was 20.8% in study conducted by Sarwar et al, whereas, the incidence of PROM was 8.7% in study done by Pitaphrom et al which is similar to the present study.[121,123] The incidence of previous LSCS with abruptio placenta was 8.7% in study done by Wandbwa et al and 7.8% by Pitaphrom et al comparable to the present study.[126,123] The incidence of multiple pregnancy in studies conducted by Jabeen et al and Abbasi et al were 4.63% and 2.08%, respectively, which is similar to the present study.[127,128]

In the current study the type of abruptio placenta is concealed type in 23%, revealed in 9% and in mixed in 68%. This is consistent with the study conducted by Ashar et al who found the incidences being 25%, 7% and 66%, respectively.[77]

We found the incidence of Grade 0 abruption is 3%, Grade 1 is 17%, Grade 2 is 27%, and Grade 3 is 53%. In a study conducted by Siddiqui et al similar incidences were found showing Grade 1 as 18%, Grade 2 as 36% and Grade 3 as 46%.[118]

Table 13: Comparison of clinical features with different studies

	Ashar et al[77]	Haynes et al[129]	Hossain et al[8]	**Present study**
Vaginal bleeding	74%	87%	84%	**77%**
Pain abdomen	54%	57%	NA	**65%**
Tense/tender abdomen	NA	26%	NA	**18%**
Absent FHS	NA	31%	NA	**53%**
Blood stained amniotic fluid	NA	NA	45%	**27%**

Table 13 shows the magnitude of symptoms and signs of abruption placenta in different studies. From our study we found incidence of signs and symptoms similar to studies by Ashar et al, Haynes et al and Hossain et al except high rate of absent FHS (53%).[77,129,8]

In the present study the diagnosis of abruption was made ultrasonographically in 23% of cases which was similar to study done by Glanz et al (24%).[130] However, in studies done by Matsuda et al and Jaffe et al the incidence was found 68.8% and 50%, respectively.[124,131]

The Cesarean section rate in the present study is 21% and similar results were found in studies conducted by Mudaliar et al (16%), Sarwar et al (30.2%) and Bibi et al (27%).[132,121,119] However, in studies conducted by Tikanen et al (91%) and Hossain et al (45%) the cesarean rates were high (Table 13).[133,8] The lower cesarean rates in our study could be due to more women in Grade 3 abruption and also because 37% of women were in the gestational age between 28 and 33 weeks with very low birth weight babies.

Table 14: Comparison of the rates of cesarean section with different studies

Study	Incidence
Mudaliar et al[132]	16%
Sarwar et al[121]	30.2%
Bibi et al[119]	27%
Tikanen et al[133]	91%
Hossain et al[8]	45%
Present study	**21%**

In the present study live births in Grade 1 and 2 abruptio placenta with vaginal delivery group is 85.2% and in the cesarean section group is 88.2%. However, study conducted by Haynes et al found the incidence of live births in the vaginal delivery was 55.95% and in cesarean section group was 39.39%.[129]

There were no of maternal deaths in the present study probably due to availability of blood and blood products and good intensive obstetric care. In studies conducted by Palaniyappan et al, Purandare et al, Ashar et al, Menon et al, Mondal et al, Parikh et al, Siddiqui et al and Bibi et al, the maternal mortality rates were 1.5%, 0.57%, 1.6%, 4.4%, 6.4%, 2.1% and 5%, respectively.[134,115,77,116,120,122,118,119]

Table 15: Maternal mortality compared with different studies

Study	Incidence
Palaniyappan et al[134]	1.5%
Purandare et al[115]	0.57%
Ashar et al[77]	1.6%
Menon et al[116]	4.4%
Parikh et al[120]	6.4%
Siddiqui et al[118]	2.1%
Bibi et al[119]	5%

Maternal complication of PPH in the present study is 7%, which is less than the study conducted by Sarwar et al (18.9%) and Memon et al (33.33%).[121,135] But study conducted by Siddiqui et al found the incidence of PPH was 9.4% which is similar to our study.[118]

Couvelaire uterus is found in 7% of cases in present study which was similar to the study conducted by Talpur et al (6%), whereas, the incidence was 16.5% in a study done by Pitaphrom et al.[136,123] Disseminated intravascular coagulation is 3% in our study which is less compared to studies by Pitaphrom et al (5.8%) and Bibi et al (8%).[123,119] Acute renal failure is complicated in 3% of cases similar to studies done by Bibi et al, Siddiqui et al and Memon et al which was 2%, 3% and 5.5%, respectively.[119,118,135] None of the women in the present study required hysterectomy for management of PPH.

The perinatal mortality in the present study is 58%. Similar results were noted in studies by Palaniyappan et al (60%), Purandare et al (79%), Mondal et al (60%), Siddiqui et al (52.97%), Bibi et al (53.4%) and Hossain et al (65%).[134,115,120,118,119,8] However, few studies the perinatal mortality rate was too high like in Ashar et al (87%) and Parikh (82%).[77,122] Prematurity and low birth weight babies were the main causes for perinatal mortality with abruptio placenta. The high perinatal mortality in the present study is probably due to high rate of dead fetus at presentation, more number of premature babies and late presentation to hospital.

Table 16: Perinatal mortality compared with other studies

Study	Incidence
Palaniyappan et al[134]	60%
Purandare et al[115]	79%
Mondal et al[120]	60%
Siddiqui et al[118]	52.97%
Bibi et al[119]	53.4%
Hossain et al[8]	65%
Ashar et al[77]	87%
Parikh et al[122]	82%
Present study	**58%**

SUMMARY

- In the present study the incidence of abruptio placenta among 4,209 deliveries was 2.52%.
- Highest incidence was among the maternal age group of 21-25 years (50%).
- Majority of the cases were primigravida (42%).
- Maximum incidence was among the gestational age groups 28-33 weeks (37%) and more than 37 weeks (36%).
- Common associated conditions were pre-eclampsia/eclampsia (29.05%) and anemia (20.5%).
- Mixed type of abruptio placenta was the most common type accounting for 68%, followed by concealed (23%) and revealed (9%).
- Most women (53%) at presentation were in Grade 3 abruptio placenta.
- Vaginal bleeding was the commonest symptom (77%). The other clinical features were pain abdomen (65%), absent fetal heart sounds (53%), blood stained amniotic fluid (27%) and tense/tender abdomen (18%).
- Clinical diagnosis was made in 77% of cases and sonographic diagnosis in 23% of cases.
- Cesarean section rate was 21% in the present study.
- The incidence of live births in Grade 1 and 2 by vaginal route and cesarean delivery in the present study was 85.2% and 88.2%, respectively. And the still birth incidence by vaginal delivery and cesarean section was 14.8% and 11.7%, respectively.
- In Grade 3 abruptio placenta 94.33% delivered vaginally and 5.66% underwent cesarean section.

- In Grade 1 and 2 abruptio placenta delivered vaginally, live births were 59.6% in women delivered ≤ 8 hours and 25.9% delivered more than 8 hours and still birth was 7.4% in both ≤ 8 hours and more than 8 hours duration. Whereas, in cesarean section group the live births were 82.3% delivered ≤ 8 hours and 5.8% delivered more than 8 hours and still birth was 11.7% in ≤ 8 hours.
- There was no maternal mortality in the present study.
- Maternal complications found were PPH (7%), Couvelaire uterus (7%), disseminated intravascular coagulation (3%) and acute renal failure (3%).
- Prematurity found in 64% of cases.
- Perinatal mortality rate was 58%.

CONCLUSION

- The incidence of abruptio placenta in the current study was 2.52%.
- The maximum incidence of abruptio placenta was found in primigravida (42%) and in the age group 21-25 years (50%).
- Common associated condition with abruptio placenta was pre-eclampsia/eclampsia (29.05%).
- Most women presented with Grade 3 (53%) abruptio placenta.
- Vaginal bleeding was the commonest presentation (77%).
- Diagnosis of abruptio placenta is essentially clinical and sonography has got minor role.
- Vaginal delivery was quite effective in severe abruptio placenta with dead fetus.
- Short admission to delivery interval did not increase the fetal survival.
- Earlier presentation to hospital and early diagnosis by the clinician with good management will improve the prognosis of abruptio placenta.

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ANNEXURES

NAME: **AGE:** **IP NO:**

DOA: **Time of admission:** **DOD:**

OCCUPATION:

ADDRESS:

H/O presenting complaints:

Obstetric history: Married life: Consanguineous/Non consanguineous:

Gravid: Para: Abortions: Living: Dead:

Previous pregnancy details:

Present pregnancy details:

Menstrual history: Age of menarche:

Previous menstrual cycles:

LMP: EDD: POG:

Past history:

Family history:

Personal history: Diet: Appetite:

Sleep: Bowel/Bladder habits:

Addiction:

General physical examination:

Built :

Nourishment :

Height :

Weight :

BMI :

Pallor Icterus Clubbing Cyanosis Lymphadenopathy Edema

Breast :

Thyroid :

Spine :

Vital signs: Pulse rate: BP:

Temperature: Respiratory rate:

Systemic examination:

Respiratory system:

Cardiovascular system:

Per abdomen:

Uterus size:

Relaxed /Acting:

Presentation:

FHS:

Per speculum:

Per vagina:

DIAGNOSIS:

GRADE OF ABRUPTION:

COMPLICATIONS:

TREATMENT:

DETAILS OF DELIVERY:

Mode of delivery: Vaginal delivery/ Caesarean section

Admission delivery interval:

DETAILS OF NEONATE:

Sex :

Birth wt :

APGAR :

Admission to NICU:

INVESTIGATIONS:

Hemoglobin: PCV: RBC:

Blood group: Platelet count:

BT: CT:

Coagulation profile:

Renal function test:

BU: SC:

RBS:

Urine analysis:

Albumin: Sugar:

WBC: RBC:

Epi cells:

Liver function test:

Total bilirubin:	Total protein:
Direct bilirubin:	Albumin:
SGOT:	Globulin:
SGPT:	A/G ratio:
Alkaline phosphatase:	Gamma GT:

Uric acid:

LDH:

Obstetric ultrasound: