

**MORBIDITY IN WOMEN WITH PLACENTA ABRUPTION: A DESCRIPTIVE
PROSPECTIVE STUDY.**

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DECLARATION

I, Dr Bongiwe Nkomo declare that this research is my original work. I declare that all sources for my research have been documented and recognised. I declare that there is no previous submission of this research for full or partial fulfilment of any institution of learning or otherwise in South Africa.

Dr Bongiwe Nkomo

Date: 13 October 2017

DEDICATION

This work is dedicated to my husband Velile Pangwa who has given me a lot of support and most of all my children Bongile, Unam and Aphelele.

ABSTRACT

Placental abruption is one of the causes of obstetric haemorrhage that is associated with adverse maternal and neonatal outcomes. Knowledge of the risk factors and complications of placental abruption is important to reduce the maternal and perinatal morbidity and mortality associated with this condition.

Objectives

1. To determine the frequency of abruptio placentae at Chris Hani Baragwanath Academic Hospital.
2. To assess maternal risk factors common in the patients admitted with placental abruption.
3. To determine the number of patients that had operative deliveries.
4. To assess maternal morbidity associated with placental abruption.
5. To assess perinatal outcomes of births in women with abruptio placentae.

Methods

This was a descriptive prospective study of morbidity in women with placental abruption. The study was carried out in the department of Obstetrics and Gynaecology at Chris Hani Baragwanath Hospital (CHBAH). Patients with placental abruption who gave informed consent to participate in the study were interviewed using a structured questionnaire. The study was conducted at Chris Hani Baragwanath Hospital. The study was carried out over a 6 month period and 60 subjects were recruited. All the women with the diagnoses of placental abruption and singleton pregnancies were included in the study.

Results

Of the 13734 delivered women 60 patients (0.4%) had placental abruption. Out of the 60 patients 53 (83%) were booked. The age group was between 18 – 42 years with the mean age of 28.2 ± 6.8 years. The mean gestational age on admission was 31.8 ± 4.7 .

Forty six (71.6%) patients had parity of one and more. The risk factors that were identified in the study were previous history of placental abruption which occurred in 4 patients (6.7%), previous caesarean section in 7 (11.7%). The commonest medical disorder observed was hypertensive disease, pre-eclampsia was found in 24 patients (40%), gestational hypertension was found in 5 patients (8.3%) and chronic hypertension in 4 (6.7%).

The maternal complications that were identified were PPH in 15 (44%) patients that had stillbirths compared to 1 (3.8%) in the group that had live births, DIC was observed in 8 (23.5%), haemorrhagic shock occurred in 2 (5.9%) of the patients, acute kidney injury in 20 (58.8%) in the group that had stillbirths compared to 3 (11.5%) in the group that had live births, Couvelaire uterus in 9 (26.4%) in the group that had still births, hysterectomy was performed in 2 (5.8%) and ICU admission was required for 5 (14.7%) of the patients.

The neonatal outcomes that were observed were stillbirths in 34 patients, birth asphyxia in 5 (19.2%) and ICU admission was required in 11 (42.3%). There were no maternal deaths.

Conclusion

In conclusion placental abruption is still a dangerous complication for both the mother and baby. In this study the patients that had stillbirths had worse outcomes compared to those that had live births. Therefore the conditions that are associated with this condition should be identified. Early recognition of this condition as well as proper referral of the patient can ensure better outcomes.

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ABBREVIATIONS

CHBAH- Chris Hani Baragwanath hospital

IL - Interleukin

TNF - Tumour necrosis factor

MMP'S- Metametalloproteases

DIC - Disseminated intravascular coagulation

FDPS - Fibrinogen degradation products

RCOG - Royal College of obstetrics and gynaecology

PPH - Post partum haemorrhage

ICU - Intensive care unit

INR - International normalised ratio

PTT - Prolonged activated partial thromboplastin time

HREC - Health research ethics committee

HELLP SYNDROME- Haemolysis elevated liver enzymes and low platelets

HB - Haemoglobin

CTG - Cardio tocography

APH- Ante partum haemorrhage

NICE- National institute for health and clinical excellence.

N - Number of patients

% - Percentage

1. INTRODUCTION

Abruptio placenta is a serious obstetric condition that increases both maternal and neonatal morbidity and mortality (1). It is a major cause of ante partum haemorrhage accounting for 22% of ante partum haemorrhage (2).

Placental abruption is defined as the premature separation of a normally situated placenta from the uterine wall before the baby is delivered (3). The exact gestation from which an abruption can be diagnosed varies in the literature from 20 weeks to 24 weeks (3, 4). Current studies show that it occurs in 0.5 to 1% of pregnancies (5, 6). The incidence world-wide has increased between the years 1998-2008 from 0.6 to 0.8% (5). Placental abruption rates tend to vary with gestational age with preterm gestations having a higher rate of about 5.4% and as pregnancy advances towards term the rate declines to around 0.3% (5). The incidence is highest between 24 to 26 weeks (7). The maternal mortality rate from placental abruption has been quoted to be 1% (3). There were 37 maternal deaths at the Chris Hani Baragwanath hospital in 2011 but none were related to or as a direct result of abruptio placentae.

Placental abruption is an independent risk factor for perinatal mortality (5). The perinatal mortality has been estimated to be as high as 64% (8). In a cross-sectional study of 50 patients with abruptio placentae by Tulpur et al which was conducted in Jinnah Post graduate Medical Centre Karachi in 2011, 18 (36%) babies were born alive, 25 (50%) were stillbirths and early neonatal deaths occurred in 14% (8).

Abruptio placenta is a clinical diagnosis and the diagnostic value of ultrasound has been shown to be weak (9). However, with posterior positioned placentae, the presentation may be atypical and complicate the diagnosis. The commonest clinical feature is a retro-placental clot or retro-placental bleeding and the incidence is observed to be as high as 77.1% (9).

This indicates that a substantial percentage of patients may have concealed abruptios.

A concealed abruptio is said to be present when blood is retained behind the placenta and its margins are still adherent and a revealed abruptio occurs when external haemorrhage is observed after the escaping blood dissects through the fetal membranes from the uterine wall. Other diagnostic features include vaginal bleeding with uterine hyper-tonicity and vaginal bleeding with non-reassuring fetal non-stress tests (9).

2. LITERATURE REVIEW

2.1 Causes and risk factors

The exact cause of placental abruption remains unclear even after extensive research (4, 5). However, several risk factors have been identified that predispose a pregnant woman to this condition. The risk factors may be categorized into medical, social and obstetric risk factors (10). Risk factors may also be classified into potentially preventable factors such as smoking and cocaine use and non-preventable such as trauma (10).

2.1.1 Antenatal care and risk categorization

Bibi et al conducted a study in 2009 in Liaquat University Hospital Hyderabad and identified the lack of antenatal care, and rural residence to be important risk factors in women who develop abruptio placentae, as attendance at antenatal clinic may provide important information such as iron and folate deficiency(4).

Also pre-existing conditions such as hypertension and diabetes can be treated if patients are assessed frequently in their pregnancies (4). In a retrospective analysis of risk factors and clinical outcomes of placental abruption by Bibi et al, anemia was identified as one of the risk factors that predisposes to the development of haemorrhage and its complications and the incidence was observed to be as high as 79%, but there was no conclusive evidence whether the anemia was the cause or as result of placental abruption itself (4).

2.1.2 Hypertensive disease

Hypertension is described as being the systolic blood pressure that is equal to 140 mmHg and diastolic blood pressure equal to 90 mmHg taken at least on two occasions at least 4 hours apart (11).

Pre-eclampsia, (defined as hypertension accompanied by proteinuria diagnosed after 20 weeks of gestation) which complicates 5-7% of pregnancies, has also been identified as a risk factor (12). Evidence has shown that placental abruption occurs in 1.5% of pregnant women with pre-eclampsia and that its frequency is not affected by duration of the hypertension and presence or absence of proteinuria (12). Ananth et al reported that the incidence of abruption was greatest among women whose pregnancies were complicated by severe pre-eclampsia and the incidence in that study was found to be 3.73 % (13). In comparison normotensive women were found to have risk of 0.92 % (13). In contrast women that had mild pre-eclampsia had no increased risk of placental abruption (13). Leunen et al conducted a study in 2003 in a tertiary referral center in South Africa, the study looked at the profiles and complications of women with placental abruption and intrauterine deaths in 96 women. Seventy-five percent of patients with placental abruption had hypertensive disorders and severe pre-eclampsia accounted for 44.8% of the 96 women. Smokers with severe pre-eclampsia are also more likely to develop placental abruption (14).

Chronic hypertension with superimposed pre-eclampsia has been shown to be a risk factor for placental abruption in other studies (12). Chronic hypertension is described as being high blood pressure that is diagnosed before 20 weeks or if women are already taking antihypertensive treatment when referred to maternity services (7). Ananth et al found that chronic hypertension complicates 0.3-0.8% of pregnancies and that advanced maternal age, parity, cigarette smoking and black race increase the risk (15). No association has been shown

between chronic hypertension and placental abruption, but chronic hypertension with superimposed pre-eclampsia is associated with placental abruption with an incidence of 5,2%(15).

Studies have shown that women with chronic hypertension with superimposed pre-eclampsia have a 2.8 to 7.7 fold increased risk for placental abruption (15).

The mechanism by which hypertensive disease leads to placental abruption is as follows:

It is postulated that during normal pregnancy the spiral arteries undergo transformation from being muscular arterioles to low resistance dilated vessels (14). These changes occur as a result of trophoblastic invasion which occurs between 10-20 weeks. In pregnancies that are complicated by hypertensive disorders there is absence of such changes. The lack of trophoblastic invasion results in decreased placental blood flow and dysfunctional endothelial responses. Basically these abnormal placental vessels may predispose to ischemia and the involved vessels can rupture and thus causing placental abruption. (14).

2.1.3 Trauma

Trauma may be blunt or penetrating. Blunt trauma such motor vehicle accidents are a risk factor for placental abruption (16). Approximately 6% of trauma cases are associated with placental abruption but if the trauma is major the incidence could increase to 20-25% (16).

It is difficult to predict the risk of placental abruption based on the severity of the trauma. Placental abruption usually manifests within 6-48 hours following the trauma but can occur up to 5 days later (16).It is recommended that these patients get admitted. The initial efforts are directed at evaluating and stabilizing the maternal vital signs. During the assessment primary and secondary surveys are done. Primary survey is done so as to make sure that breathing is adequate, airway is secure and that there is adequate circulation in those that are

seriously injured. The secondary survey involves the examination of all the body regions and it also includes fetal evaluation. It is recommended that women involved in trauma should have 4 hours of fetal monitoring but this duration can be extended if uterine contractions start or if the uterus becomes irritable and fetal non-stress tests are non-reassuring. If there is no uterine irritability or tenderness, or rupture of membranes the patient may be discharged. Upon discharge strict instructions should be given, the patient should be instructed that if there is bleeding, leakage of liquor, decreased fetal movements and severe abdominal pains that she must return to the hospital.

2.1.4 Polyhydramnios

Polyhydramnios has been described as a risk factor for placental abruption (3). Polyhydramnios is defined as the largest vertical pool of amniotic fluid of 8cm or amniotic fluid index above the 95th centile for gestational age (17). The theory to support this factor is the sudden decompression of the uterus following the rapid loss of amniotic fluid after the rupture of membranes. It is the sudden change in the uterine size that predisposes patients with Polyhydramnios to placental abruption (3).

2.1.5 Previous history of placental abruption

Another important risk factor is a history of a previous abruption. After one episode the recurrence rate is 11% and increases to 25% after two episodes (3). Since these women are at risk of repeat placental abruption, there are recommendations by Oyelese et al on managing subsequent pregnancies. The recommendations are that, the women with identifiable risk factors should be thoroughly counseled (18). If the woman is a smoker she should be advised to stop smoking before the next pregnancy. According to literature there hasn't been any intervention that has reduced the recurrence of placental abruption, since this condition is not predictable. There is also no accurate marker that can predict the recurrence of placental

abruption. Another recommendation is that women with a history of placental abruption should have serial growth scan every 4 weeks during the second half of pregnancy as they have an increased risk of impaired utero-placental perfusion (18). Women with prior history of placental abruption can be offered amniocentesis for lung maturity and delivery should be aimed at 37 weeks (18).

2.1.6 Cigarette smoking and cocaine use

The relative risk of placental abruption varies from 1.5- 2.5 in women who smoke (19). The risk is doubled if both partners are smokers. Tikannen et al conducted a study in 2009 on the epidemiology, risk factors and consequences of placental abruption in the university central hospital in Finland and found that the risk of placental abruption was 4, 8 fold higher if both partners were smokers (10). Paternal smoking as a risk factor could be explained by passive exposure of the pregnant woman (20). Several studies suggest that smoking as a risk factor is dose dependent (19). Raimond et al found that with every 10 cigarettes smoked each day there is a 20% increase in the risk of placental abruption (21).

The risk of placental abruption is increased by 40% for each year of smoking prior to pregnancy (2).

If smoking has been stopped before pregnancy or early in pregnancy, the risk of abruption can be reduced to the level of non –smokers (19). Studies show that if women stopped smoking during pregnancy, 15-25% of placental abruption could be prevented (19).

The assumed mechanism explaining the association of smoking and placental abruption is as follows:

Nicotine in tobacco smoke crosses the placenta leading to higher concentrations in the fetal plasma (22). It also concentrates in the placental tissue, amniotic fluid and breast milk.

Tobacco causes vasoconstriction in the uterine artery as well as the umbilical artery. These changes reduce utero-placental blood flow (22). The risk of placental abruption is six times higher in women who smoke and have severe pre-eclampsia compared to women who are non-smokers with pre-eclampsia. This is explained by the fact that smoking has vasoconstrictive effects and so it affects the fragile vessels already compromised by hypertension.

2.1.7 Cocaine abuse

The risk of placental abruption in patients that abuse cocaine is also dose dependent, the risk is said to be 13-35%. Cocaine has a vasoconstrictive effect and therefore, interferes with utero-placental blood flow and affects the integrity of the decidua (3).

2.1.8 Previous caesarian section

Studies show that caesarian section delivery in the first pregnancy increases the risk of placental abruption by 30-40% in the next pregnancy (23). If the interval between pregnancies is less than one year, the risk of placental abruption is 52% in women that are delivering for the first time by normal vaginal route compared to women that are delivering for the first time by caesarean section whose risk increases to 111% (24).

Previous caesarean section scars result in pathological changes in the myometrium and endometrium. These changes may cause sub-optimal implantation of the placenta (24).

2.1.9 Multiparity and advanced maternal age

Tulpur et al identified Multiparity and advanced maternal age as additional risk factors for placental abruption (8). The incidence of placental abruption was higher in women less than 25 years when compared to women between 25 to 29 years in a study by Ananth et al (25). There was no increase in the incidence with advancing maternal age. In contrast, the

incidence of placental abruption increased with increasing parity and this was found in women that were having their third pregnancy (25). Tikannen et al found that placental abruption in women 35 years or older was attributed to high parity irrespective of gestational age (21). Sarwar et al conducted a study on placental abruption and its complications between July 2003 and June 2004 in Ayub teaching hospital Abbottabad and reported significantly higher incidence in the multiparous patients compared to primigravida patients (88.7% vs 11.3%) (27). In this study multiparity was supported as a risk factor (26).

2.1.10 Premature rupture of membranes and chorioamnionitis

It is suggested that 4-12% of patients with premature rupture of membranes before 37 weeks gestation develop placental abruption (27). Placental abruption risk increases with decreasing gestational age at membrane rupture. Ananth et al found that preterm labour and pre-labour rupture of membranes represent an acute disease process that is associated with placental abruption. Studies have found the incidence of placental abruption to be 4.8% in women that had intrauterine infection, in contrast to 0.8% in women that had no intrauterine infection (28).

A sudden reduction in the uterine volume following the rupture of membranes is the suspected mechanism that leads to placental abruption (3). The risk of placental abruption is also increased by prolonged exposure to premature rupture of membranes and if time from membrane rupture to delivery exceeds 24hours (28). The possible mechanism involved in intra-uterine infections is that, there is direct bacterial colonization of the decidua with tissue inflammation which may lead to placental abruption or the infection activates cytokines such as IL and TNF and these cytokines up regulate the production and activity of metalmetalloproteases(MMP'S) in the trophoblast, resulting in the destruction of the matrix

and cell interactions which may lead to disruption of placental attachment and finally to placental abruption.

Other Miscellaneous risk factors for abruption include poor nutritional status, preterm labour, infertility treatment and dietary deficiencies such as folate deficiency and hyperhomocysteinemia. There is also an association between thrombophilia and placental abruption (3).

Women with placenta praevia have a 5% chance of having an abruption (20). Other pregnancy related risk factors that may predispose to placental abruption include bleeding during pregnancy and multiple-pregnancy. Bleeding in the early trimesters, referring to the bleeding that occurs in the first two trimesters of pregnancy has also been associated with placental abruption later in pregnancy (20). If a retro-placental clot is detected in the first trimester by ultrasound this also increases the risk of placental abruption as the haematoma may interfere with normal placentation (29).

2.2 CLINICAL PRESENTATION

Abruptio placenta may be graded according to clinical presentation. The following is recommended by Sinha et al from the journal of obstetrics and gynaecology 2008 (2).

Grade 1: Asymptomatic (small retro-placental clot discovered after delivery)

Grade 2: There is usually vaginal bleeding with uterine tetany and tenderness without any signs of maternal and fetal compromise.

Grade 3: Usually concealed haemorrhage with persistent abdominal pain, vital signs are unstable, there is maternal tachycardia associated with orthostatic changes in the blood

pressure .Fetal heart rate abnormalities are present and abnormalities with coagulation may be evident in 30%.

Grade 4: The patient may present with either or no vaginal bleeding or heavy vaginal bleeding with a tetanic uterus. There may be maternal shock, coagulopathy and fetal demise in most the cases.

2.3 DIAGNOSIS

Placental abruption is a clinical diagnosis (9). It should be suspected in women who present with vaginal bleeding or abdominal pain or both after 20 weeks of gestation or more. Placental abruption should also be suspected in all pregnant women with a history of trauma. The use of ultrasonography as a tool to diagnose placental abruption has been shown to be weak (9). According to the Glantz et al , the sensitivity has been reported to be 24% with a specificity of 96%.The positive and negative predictive values were 88% and 53% respectively(30).However the same study noted that ultrasonography failed to detect at least three –quarters of cases of placental abruption (30).

The ultrasound appearance of an abruption depends largely on the size and the location of the bleeding, also the timing between the occurrence of the abruption and the time when the ultrasound examination is done (18). The pathologic findings differ between an acute abruption and chronic abruption .In the pathologic examination of the delivered placenta an acute abruption can be confirmed by the presence of a fresh clot attached to the maternal surface of the placenta (31). In a chronic abruption there may be deposits of fibrin at the site of the abruption and infarcts may be seen overlying the placenta particularly on the maternal surface of the placenta (31).

The following ultrasonography criteria for the diagnoses of placental abruption have been described by Oyelese et al (18).

1. Jello sign: referring to the jingling of the placenta when sudden pressure is applied with the transducer.
2. Pre-placental collection: referring to the collection that is between the placenta and amniotic fluid
3. Retro-placental collection: referring to the clot that is between the myometrium and the placenta.
4. Marginal haematoma: placental separation along the lateral edge of the placenta
5. Sub-chorionic haematoma: is said to arise from the marginal abruptios.
6. Increased heterogeneous placental thickness
7. Intra amniotic haematoma

Glantz et al conducted a study in 2002 in which 149 patients were enrolled between the years 1994 to 1996, the study was done in strong memorial hospital. The study looked at the clinical utility of ultrasound in the diagnoses and treatment of placental abruption (30). Of the 149 patients studied, 17 (11%) had sonographic evidence of placental abruption but 32(21%) had evidence of placental abruption at delivery. The conclusion from this study was that ultrasonography was not sensitive for detection of placental abruption but a positive finding was associated with worse neonatal outcomes (30). Another important aspect is that a negative ultrasonography does not exclude an abruption (30).

2.4 MATERNAL COMPLICATIONS

Placental abruption may lead to severe adverse outcomes for the mother. Haemorrhagic shock, disseminated intravascular coagulation (DIC), postpartum haemorrhage (PPH), acute kidney injury, Couvelaire uterus (which occurs when blood dissects into the myometrium towards the serosa), caesarean hysterectomy, uterine rupture, blood transfusion, prolonged hospital stay, ICU admission and maternal death have been associated with abruption placenta.

2.4.1 Disseminated intravascular coagulation (DIC)

The presence of DIC indicates the severity of the abruption. (3). The reported incidence of DIC in the literature varies from 0.8% to 10% (1, 3, 32). The risk of DIC is highest when there is large placental detachment. The mechanism of DIC in abruption placenta is multifactorial. A Couvelaire uterus causes abnormal uterine contractility and an atonic uterus which results in PPH. The fibrinogen degradation products (FDPS) that are released when DIC occurs can also affect the contractility of the uterus which may cause further PPH. FDPS can also act as anticoagulants which can further worsen the DIC. DIC is a consumptive coagulopathy which is characterized by generalized activation of the coagulation system. DIC is never a primary but always secondary diagnoses which follow the release of thromboplastin, which is a pro-coagulant substance into the circulation (32). The released thromboplastins lead to the activation of the clotting cascade and this may lead to the rapid consumption of the coagulation factors and platelets, with deposition of fibrin in the microcirculation which leads to thrombocytopenia, defibrination and haemostatic failure.

The diagnosis is made on clinical grounds if a patient has bleeding tendencies and through blood tests such as the platelet count, international normalized ratio and activated partial

thromboplastin time (3). Bleeding tendencies may manifest with abnormal bleeding from venepuncture sites, gums and nose (3). The laboratory indicators of DIC are prolonged activated partial thromboplastin time and prothrombin test, low levels of platelets and high levels of fibrinogen degradation products (3). However, laboratory tests are insensitive and more than 50% of clotting factors need to be consumed before they become abnormal.

A scoring system has been described to measure the severity of DIC utilising all the coagulation tests (33). A risk assessment is initially performed to determine if a patient has underlying disorders known to be associated with overt DIC. If the answer is suggestive, then one proceeds to use the scoring system (33). The coagulation tests that are used are prothrombin time, platelet count and ° fibrinogen fibrin related marker.

Scores are allocated as follows according to guidelines for the diagnosis and management of DIC (37), from the British journal of haematology.

- Platelet count :
 - $100 \times 10^9 = 0$
 - $< 100 \times 10^9 = 1$
 - $< 50 \times 10^9 = 2$
- Elevated fibrin marker :

-D-dimer, FDPs

- no increase = 0 ,
- Moderate increase = 2
- Marked increase = 3

- Prolonged PT:
 - $<3s = 0$
 - $>3s$ BUT $<6s = 1$
 - $6s = 2$

- Fibrinogen level :
 - $>1 \text{ g/l} = 0$
 - $<1\text{g/dl} = 1$

If the score of >5 is suggestive of overt DIC, and a score of < 5 is non-overt DIC.

The effective management is treating the underlying cause which is delivery of the baby and placenta and the condition usually resolves 12 hrs post-delivery (3, 33). The main goal of management is to restore and maintain circulating blood volume. This prevents kidney injury and further haemostatic failure that is caused by hypovolaemic shock (32).

2.4.2 Haemorrhagic shock

Abruptio placenta is often associated with significant blood volume loss which results in shock. Shock is defined as the clinical condition that occurs when there is hypo perfusion of the most vital organs (3, 34). Bleeding can occur either vaginally or concealed within the uterus (3).

In pregnancy there is usually a physiological protection against haemorrhage as a result of high levels of coagulation factors and expansion of blood volume. However if blood loss exceeds 25% of total volume rapid hemodynamic deterioration occurs (3).

The management of haemorrhagic shock entails restoration of the effective circulating blood volume to improve tissue perfusion and avoid tissue necrosis of target organs (3).

Initial treatment is by infusing crystalloids, followed by red cell replacement. Platelet transfusion may be necessary if the platelet count is $30/\mu\text{L}$ or there is oozing from the puncture sites (3). The response to the resuscitation is monitored by urine output which should be more than $0,5\text{ml /kg/hr}$ (3). If the patient has had a massive blood transfusion, potassium should be monitored to avoid hyperkalaemia (3).

2.4.3 Postpartum haemorrhage (PPH)

Postpartum haemorrhage can be classified into primary and secondary (35). Primary PPH is defined as the bleeding that exceed 500ml from the genital tract within 24 hours of the delivery of the baby(35).Secondary PPH is defined as bleeding that exceeds 500ml occurring between 24hours and 12 weeks (35). It can further be classified into minor and major. A minor PPH is referred to an estimated blood loss of 500ml and more, and major PPH refer to an estimated blood loss that is more than 1000ml (35). At caesarean section the estimated blood loss can be up to 1000 ml and if the delivery is vaginal the estimated blood loss can be up to 500ml. The reported incidence of PPH ranges from 16, 6% to 18, 9% in placental abruption (26, 36). The management can either medical or surgical owing to the multifactorial aetiology of PPH.

According to the RCOG guidelines the management of PPH consists of four components namely (35):

- Communication
- Resuscitation

- Monitoring
- Investigations

In managing any emergency condition communication is the key, clinicians should seek the assistance of colleagues. Resuscitation of the patient with PPH should include evaluation of the air way, breathing and circulation and make sure that they are secure.

As part of the management blood should be sent to the laboratory for assessment of full blood count, urea and creatinine and coagulation screen.

Medical management of PPH

Atonic uterus is usually the cause of PPH in patients with placental abruption the following medical treatment can be instituted.

- 1) Bimanual uterine compression to stimulate uterine contractions.
- 2) Empty the bladder.
- 3) Syntocinon 5 units by slow intravenous injection.
- 4) Ergometrine 0.5mg by slow intravenous or intramuscular injection.
- 5) Syntocinon 40 u in 500ml Hartmann's solution at 125 ml/hr unless fluid restriction is needed.
- 6) Prostaglandin f_{2α} can be given at a dose of 0.25mg by intravenous injection up to 8 doses can be given.
- 7) Misoprostol 1000mcg rectally

If medical treatment fails the following surgical interventions are considered.

- 1) Balloon tamponade
- 2) Haemostatic brace suturing, B-Lynch
- 3) Bilateral ligation of the uterine artery
- 4) Bilateral ligation of the internal iliac artery
- 5) Selective arterial embolization

B- Lynch suture

The B-Lynch suture was first described in 1997 by Christopher B-Lynch. It has been proven to be effective in the management of PPH (37). The B-Lynch suture is a lifesaving procedure that is relatively safe and simpler than the other surgical techniques such as uterine and internal iliac artery ligation (37). It has the ability to preserve the uterus as well as the fertility (37). This procedure was first done in patients that had life threatening postpartum haemorrhage. It was used in patients with placental abruption with PPH and DIC, prolonged labour, eclampsia in labour with PPH and DIC, Atonic uterus and major placenta praevia (37). When these patients were subsequently followed up they had good outcomes with no complications. The B-Lynch suture is better when compared to other techniques, which may be difficult to accomplish when expeditious management is required (37). Other techniques may not adequately control the bleeding more especially if there coagulopathy and an atonic uterus. To test the efficiency of this procedure a bimanual compression test is done, if the bleeding stops it means the brace suture can be applied. If this procedure fails to control the bleeding a more invasive procedure may be considered such as the hysterectomy

2.4.4 Acute kidney injury

The diagnoses of acute kidney injury signify severe placental abruption. The incidence of acute renal injury associated with placental abruption is about 6% (9). The features are hyperkalaemia, hyponatremia, metabolic acidosis, and oliguria of less than 400ml in 24hours. Rising serum urea and creatinine levels are suggestive of acute kidney injury.

The three forms of acute kidney injury that may occur are acute tubular necrosis, acute cortical necrosis and pre-renal failure. Prolonged ischemia from delayed treatment of hypovolaemia causes damage of the metabolically active tubular cells resulting in acute tubular necrosis which is reversible. Acute cortical necrosis is a severe form of renal failure that is irreversible, and the pathology involves the necrosis of the tubules and glomerulae throughout the kidney.

Management can be conservative with complete recovery in pre-renal failure (9)

2.4.5 Couvelaire uterus

Couvelaire uterus occurs when the blood dissects through the myometrium towards the serosa. The diagnosis is usually made during caesarean section or during a laparotomy for post-partum hysterectomy (38). The uterus appears dark purple with poor contractility and thus becomes atonic (38). The complication was first described in 1911 by Alexandre Couvelaire who was a French Obstetrician. A wide range of incidence is quoted in the literature from 6% to 16, 5% (1, 8).

2.4.6 Peripartum hysterectomy

Peripartum hysterectomy is associated with increased morbidity and mortality (39). It is performed at the time of delivery or in the immediate postpartum period. It is a lifesaving procedure that is done in severe cases of haemorrhage that are unresponsive to medical and surgical procedures (40). In a ten year review of emergency Peripartum hysterectomy (EPH) in a tertiary hospital which was conducted by Khan et al, the incidence of Peripartum hysterectomy was 16.5% in patients that had placental abruption (40). Abbasi et al found that 1.9% of cases were done Peripartum hysterectomy in order to control postpartum haemorrhage in patients with placental abruption (36). In the study by Khan et al, these patients that had hysterectomies had Couvelaire uterus which ultimately lead to uterine atony not responding to conservative management (40). The hysterectomy was done in order to save the women's life.

A subtotal hysterectomy is usually preferred method as it is safe and quick procedure, when the women's condition requires immediate arrest of haemorrhage to save her life (40). It is associated with less blood loss, reduced operating time and reduced intra-operative and post-operative complications (40).

In the study by Khan et al the patients that had Peripartum hysterectomy had the following complications (40).

- Septicaemia
- Wound infection

- Burst abdomen
- Paralytic ileus
- Coagulopathy
- Bladder injury
- Ureteric injury
- Hypovolemic shock
- Renal failure
- Pneumonitis and blood transfusions

According to the literature the main complication is the need for blood transfusion (39)

In view of the above morbidities that are associated with EPH conservative measures are tried to arrest the bleeding before considering EPH (40). The conservative measures include uterotonic drugs, haemostatic sutures uterine and systemic devascularisation. Conservative management is of importance in patients that are young, have low parity and are clinically stable (40).

2.4.7 Blood transfusion

Blood transfusion is an important morbidity associated with placental abruption. Blood transfusion is a lifesaving procedure. According to the RCOG guidelines blood transfusion should be initiated on both clinical and haematological grounds (35).

According to the RCOG guidelines blood transfusion is rarely indicated in a stable patient with a haemoglobin of ≥ 10 g/dl, it is always indicated when haemoglobin of 6g/dl is found. According to RCOG guidelines on PPH for every 6 units of red cells give 4 units of FFP'S

(35). The incidence of blood transfusion in patients with placental abruption is between 19.4% and 50 % (1, 4).

A study of 106 patients with placental abruption by Bibi et al assessed risk factors and clinical outcomes. Fifty-four (50%) of patients needed blood transfusion and 5 patients needed massive blood transfusion (4). Massive blood transfusion is defined as an acute administration of more than half of the patients estimated blood volume per hour. This was comparable to a study done by Leunen et al who found that 43% of patients needed blood transfusions(41). In contrast Pitaprom et al found that only 19.4% patients needed blood transfusions(1).

2.5 FETAL AND NEONATAL MORBIDITY

Placental abruption may have severe adverse outcomes for both the fetus and the neonate. The morbidities associated with placental abruption are preterm deliveries, intrauterine growth restriction, low APGAR scores, hypoxia, stillbirths, early neonatal deaths, and neurodevelopmental problems.

Placental abruption has been found to be an independent risk factor for perinatal mortality. The perinatal mortality can be as high as 60-64% (5), but in developed countries it ranges between 9-12% (20, 42).

2.5.1 Fetal distress

Fetal distress is one of clinical presentations of placental abruption. There are explanations to this, during the early stages of placental abruption there is vasoconstriction that is induced by catecholamine's which may selectively maintain the perfusion of the maternal heart and brain at the expense of utero-placental blood flow, therefore fetal distress is an important sign which shows that there hemodynamic compromise(3). According to the Nice guide lines there

are four components that a clinician must comment on when interpreting fetal heart rate patterns namely: baseline heart rate, variability, accelerations and decelerations(43).

Feature	Normal value
Normal baseline	110 – 160 b/min
Baseline variability	5- 25b/min
Accelerations	A rise of 15 b/min or more above the base line lasting for 15s
Decelerations	No deceleration should be present in a reassuring CTG

In a normal trace all four components should be reassuring. Fetal distress is recognized by the presence of two or more features that are non-reassuring or one or more features that are classified as abnormal.

According to the South African literature Odendaal et al did a study that looked at patients that had early onset severe preeclampsia (42). The aim of the study was to look at the patients that were treated conservatively and those that ultimately had placental abruption, the perinatal mortality rates were compared between the study group which consisted of 69 patients and controls which included 69 patients. They found a 67% prevalence of fetal distress in the placental abruption group, 58% had late decelerations on routine monitoring and half were accompanied by poor variability. Only 22% had fetal heart rate recording that were normal. Most of the patients in the abruption group presented with fetal distress than

presenting with the typical sign and symptoms of placental abruption, such as abdominal pain and vaginal bleeding (42).

These findings show an importance of monitoring the fetal heart regularly in patients with severe preeclampsia as fetal distress is detected before any other signs of placental abruption (42). In a study that was conducted by Elsasser et al on the diagnosis of placental abruption looking at the relationship between clinical and histopathological findings, it was found that out of the 424 patients that were enrolled in the study about 16.1 % had non-reassuring fetal heart tracings (9).

2.5.2 Prematurity

Prematurity is a significant contributor to the high perinatal mortality associated with placental abruption (44). Placental abruption has been associated with prematurity.

Placental abruption is thought to cause preterm labour and patients with placental abruption may need delivery irrespective of gestational age.

The mechanisms by which preterm labour cause placental abruption are as follows:

It is believed that there is a release of thrombin because of decidual placental haemorrhage and hemosiderin deposition which trigger labour leading to preterm delivery.

Another theory is that: as a result of extravasation of blood at the placental margin that may lead to decidual necrosis which in turn could initiate the production of prostaglandins thereby leading to preterm birth.

The incidence of prematurity has been quoted to be between 40-60%. Tikkanen et al did a study on clinical presentation and risk factors of placental abruption (20). In the study 198 patients were recruited. Out of those 198 patients 53% of the patients had preterm births

(20). This was comparable to a study that was done by Ananth et al which was on placental abruption and adverse perinatal outcomes who found a 40% incidence of preterm birth (45). In the study by Ananth those pregnancies that were less than 32 weeks of gestation were greatly affected by placental abruption. In the same study even women that had the mild form of placental abruption the risk of preterm birth was increased (45). In a study conducted by Pitaprom et al on pregnancy outcomes of placental abruption a 56% incidence of preterm birth was found, among the premature neonates 44% suffered severe birth asphyxia and with the consequence of three neonatal deaths(1).

2.5.3 Low birth weight and intrauterine growth restriction

A vast majority of low birth weight seen in placental abruption is as a result of shortened gestation and growth restriction.

Ananth and colleagues did a study on placental abruption and adverse perinatal outcomes and found a 34.7% rate of low birth weight in the placental abruption group vs 6.7 in the group without placental abruption (45). The growth restricted babies had a 14.3% incidence in the placental abruption group versus 8.1 % in the group without placental abruption yielding a relative risk of 2.0(95%CI 1.5-2.4) (45). This was comparable to a study that was conducted by Ananth et al which looked at the placental abruption and perinatal mortality in the United States, an incidence of low birth weight was 46% (44). In contrast Pitaprom et al found a 65% incidence in a study that was conducted on pregnancy outcomes in placental abruption (1). There are neurodevelopmental outcomes that are associated with low birth weight such as cerebral palsy, cystic periventricular leukomalacia and periventricular haemorrhage.

2.5.4 Neonatal ICU admission

In most studies the reason for ICU admission is because of asphyxia and prematurity.

In a systemic review study by Obeidat et al on the parental experience of having an infant in the new born intensive care unit (46), It was found that the parents experienced high levels of distress such as depression, anxiety, trauma symptoms and feelings of powerlessness (46). They are also confronted with dealing with an environment that specializes with critical care and which is associated with a lot of demands (46).

The early separation of the new born from the parents can also cause the strain in the infant-parent relationship more especial during the lengthy stays in NICU, the parents may need to see, hold and touch their new born babies in order to facilitate early attachment and bonding (46).

Therefore appropriate counseling is essential for these mothers. The newborns are also at risk of nosocomial infections because of the lengthy stays in the hospital.

2.5.5 Stillbirth

The risk of delivering stillbirths depends on the severity of the placental abruption (45). Two types of placental abruption are described namely complete and partial, complete separation of the placenta carries a higher risk of fetal death than partial separation .Ananth et al has quoted that the risk of still births increases if the placenta separates by over 50%(45).

In the study that was conducted by Pitaprom et al on pregnancy outcomes of placental abruption it was found that 12.5% of pregnancies were complicated by still births (1). In contrast Abbasi found a 41% incidence of stillbirths (36).

According to RCOG Guidelines if fetal death has been diagnosed, vaginal birth is usually recommended provided the maternal condition is satisfactory, but caesarean birth may be recommended for some women (47).

For those patients who have fetal losses, debriefing of both the partner and the mother is important. This has to be done as soon as possible while the women can still comprehend and communicate (47). All the events should be discussed with the family and they must be given an opportunity to ask questions (47).

There are psychological problems that may follow such as postnatal depression and post-traumatic stress disorder. Counseling should be offered to all women and their partners. The parents should be advised of support groups. A follow up plan should be made to see these women 4 to 6 weeks post-delivery for medical and psychological support.

2.6 SHORT AND LONG TERM OUTCOMES OF PLACENTAL ABRUPTION.

2.6.1 Cerebral palsy

Kayani et al did a study on pregnancy outcomes in severe placental abruption 33 cases were identified for the study. The infants were followed up, the finding was that those that survived up to a year were (67%) and were developing normal at one year of age, and (24%) of infants died in the neonatal period(48). Of the surviving infants (12%) had cerebral palsy and two had sudden infant death syndrome in their first year of life. Another important finding was that 55% of the babies that were delivered within 20 minutes or less from the time of decision making had good outcomes (44). The decision to delivery interval greatly influenced the outcome this was evidenced by reduced neonatal morbidity and mortality.

In another study by Spinillo et al which looked at the severity of abruption placenta and neurodevelopmental outcome in low birth weight infants (49). For the study a total of 40 babies were recruited, their weight at birth was <2500g and 80 controls with similar weight and gestational age were used for the study. In the study it was found that those infants that were born to mothers with placental abruption had low Apgar scores. The infants of mothers who had grade 2 to 3 placental abruption required assisted ventilation and had fetal acidosis.

It was found that 10 infants needed assisted ventilation and 10 other infants had fetal acidosis, compared to only 2 infants that required assisted ventilation and had fetal acidosis in those mothers that had grade 1 placental abruption.

A follow up of the surviving infants was done of the 40 infants: 36 infants survived and cerebral palsy was diagnosed in 4 infants (11%) and none of the infants in the controls had cerebral palsy. The cases of cerebral palsy included two infants that had spastic quadriplegia, two with quadriplegia and mental retardation.

2.6.2 Intraventricular haemorrhage and cystic periventricular leukomalacia

There are also neonatal intracranial lesions that can occur as a result of placental abruption.

In the study by Spinillo et al an incidence of 17.5% of Intraventricular haemorrhage was found in infants whose mothers had grade 1 - 4 placental abruptions compared to only 5% in the control group. Gibbs et al found a high incidence of Intraventricular haemorrhage in the study that was conducted on neonatal intracranial lesions following placental abruption (50). In the study 29 infants were recruited following placental abruption over a 2 year period and similar controls were used. In the placental abruption group Intraventricular haemorrhage and haemorrhage into the brain parenchyma occurred in 72% compared to only 48% in the control group (50).

According to the literature the risk factors for Intraventricular haemorrhage in low birth weight include high rates of abnormal fetal heart rates patterns, acidosis, low APGAR scores, assisted ventilation and neonatal hypotension.

Intraventricular haemorrhage has got other complications such as:

- Periventricular haemorrhagic infarction(PVHI)

- Post hemorrhagic hydrocephalus
- Cerebellar hemorrhagic injury
- Periventricular leukomalacia

The above are important determinants for neonatal morbidity, mortality and long term neurological sequel. When the Intraventricular haemorrhage is complicated by PVHI the risk of major neurodevelopmental outcomes increases by 75%.

2.6.3 Periventricular leukomalacia

Studies have shown that cystic periventricular leukomalacia is another complication that can be found in infants whose mothers had placental abruption. An incidence of 5 - 34% has been found. Spinillo et al found that those infants that were born by mothers who had grade 2 to 3 placental abruption had a risk of developing cystic periventricular leukomalacia (49).

Periventricular leukomalacia is considered a marker of neonatal brain ischemia and is associated with moderate to severe developmental disabilities.

In placental abruption there is a sudden interruption of maternal and fetal communication which results in severe ischemic event, as there is decreased cerebral perfusion, which is why it is not surprising to see high rates of cystic periventricular leukomalacia among surviving infants.

2.7 DEFINITIONS

Hypertension was defined as a systolic blood pressure of 140mmHg and a diastolic blood pressure of 90mmHg or more, on two occasions at least four hours apart or a single diastolic blood pressure reading of 110mmHg or more (11).

The following hypertensive disorders were described for the purpose of the study.

Chronic hypertension was described as the hypertension that is present at the booking visit or before 20 weeks or if the women is already taking antihypertensive medication when referred to maternity services (11).

Pre-eclampsia was diagnosed as the blood pressure that is accompanied with proteinuria after 20 weeks of pregnancy (11).

Gestational hypertension was defined as the hypertension that presents after 20 weeks of pregnancy without significant proteinuria (11).

Unclassified hypertension was defined as the hypertension in the pregnant women whose blood pressure was not measured before 20 weeks of pregnancy (11).

Gestational diabetes was defined as the diabetes that is first recognised in pregnancy after 20 weeks and diagnosed by a positive oral glucose tolerance test.

PROM was diagnosed if rupture of membranes occurred at less than 38 weeks gestation.

An amniotic fluid index of more than the 95th centile for the gestational age or a single deepest vertical pool of more than 8cm was used to diagnose polyhydramnios (17).

The diagnostic clinical features of haemorrhagic shock included circulatory collapse with altered mental status, tachycardia of more than a 100bpm, oliguria of less than 30 ml/hr, tachypnoea of more than 20 breaths per minute, a fall in the systolic blood pressure to less than 80 mmhg and cold and clammy extremities (34).

The diagnosis of DIC was described as a combination of the clinical picture such as abnormal bleeding from the gums, nose, and venepuncture sites together with laboratory tests. The laboratory component of diagnosis of DIC was determined by assessing the platelet count and

coagulation profile which consists of an INR and PTT (2). Thrombocytopenia was described as a platelet count of less than 150 per micro litre. The most abnormal INR and PTT results above the control were documented for the study.

3 PROBLEM STATEMENT

Despite all the information we have gleaned from the literature the contribution of abruptio placentae to the maternal morbidity and perinatal morbidity and mortality rates at the Chris Hani Baragwanath academic Hospital is unknown.

4 MATERIALS AND METHODS

4.1 Objectives

1. To determine the frequency of abruptio placentae at Chris Hani Baragwanath Academic Hospital.
2. To assess maternal risk factors common in the patients admitted with placental abruption.
3. To determine the number of patients that had operative deliveries.
4. To assess maternal morbidity associated with placental abruption.
5. To assess perinatal outcomes of births in women with abruptio placentae.

4.2 Setting of the study

The study was done at the Chris Hani Baragwanath Academic hospital which is a tertiary referral hospital. Chris Hani Baragwanath Academic hospital is a busy hospital serving a low socio-economic population of Soweto and recorded a staggering 23 271 deliveries in 2011. Patients are referred from surrounding clinics and secondary hospitals.

4.3 Study population

The study population included all pregnant women who were diagnosed with placental abruption. A total of 60 patients were enrolled in the study. The patients for the study were recruited from the 18th of May 2013 to 31 September 2013.

4.4 Inclusion criteria

All patients diagnosed with placental abruption whether the diagnosis was made clinically or by ultrasonography were included. The study also included patients who were diagnosed only at delivery, by presence of a retro-placental clot. These patients were delivered either vaginally or by caesarean section.

4.5 Exclusion criteria

Multiple pregnancies, placenta praevia, APH of uncertain origin and females who were younger than 18 years were excluded.

4.6 Study design

This was a prospective, descriptive study of the women diagnosed with placental abruption.

4.7 Data collection and informed consent

The management of all patients diagnosed with abruptio placentae at the Chris Hani Baragwanath Hospital obstetrics department includes admission into the maternity high care area. Daily rounds were performed by the primary investigator to identify patients diagnosed with placental abruption. Ward rounds were also done in the postnatal ward where the patients that presented with ante partum haemorrhage were identified, also patients who had bad outcomes such as fresh still born and macerated still born were also identified.

Some patients, diagnosed with abruptio placentae were transferred directly from the admissions ward to theatre therefore the admission ward register and caesarean section theatre register were also searched for to identify patients eligible for the study.

A data collection sheet was assigned to each patient after informed consent to examine their records and those of their new-borns was signed. Patients were assigned a research

identification number to ensure that they remained anonymous. The patients who were unable to give consent owing to artificial ventilation and intensive care unit admission were only included in the study once they were able to give consent. The transitional intensive care unit and neonatal intensive care unit (ICU) registers were reviewed to determine perinatal outcomes of live births in women with placental abruption. The mode of delivery of all the patients was determined.

4.8 Outcome measures

4.8.1. Risk factors

The risk factors that were studied in all patients were age, race, parity, and gestational age, number of antenatal visits, smoking, and use of recreational drugs such as cocaine and hypertensive disorders.

4.8.2 Maternal Outcomes

The maternal outcomes that were assessed in the study were: haemorrhagic shock, DIC, renal failure, thrombocytopenia and PPH.

The number of patients who required blood transfusions and the number of units of packed red cells that were transfused were assessed. The use of platelets and fresh frozen plasma was also determined. Documentation was made of each patient's lowest haemoglobin level (HB) and correlated to the need for blood transfusion and the number of units that were transfused. ICU admission, length of hospital stay, caesarean hysterectomy and maternal deaths were also assessed. Blood investigations relevant to the study were full blood count with platelets, urea and electrolytes and coagulation profile.

4.8.3 Perinatal outcomes

Perinatal outcomes that were evaluated include birth weight, prematurity, ICU admissions and the number of still births. The APGAR score at 5 minutes was also evaluated to exclude birth asphyxia. An Apgar score of 7 and above was considered normal and a score of less than 7 was regarded as birth asphyxia.

4.9 Statistical analysis

Data management and analysis was done by transferring data from the data collection tool to Microsoft Excel. Descriptive statistics included statements of frequencies and percentages, means, standard deviation, and medians with ranges including interquartile ranges. If analytical methods were needed to compare data according to exposures or outcomes, the Fisher's exact test for categorical variables and Student's t-test or Wilcoxon ranksum test for continuous variables were used. A p-value < 0, 05 was considered as indicating statistical significance. The continuous data was presented using means \pm standard deviation.

4.10 Funding

The study was funded by the investigator.

4.11 Ethics

Ethics approval was obtained from the HREC. The ethical issues that were raised with respect to the study were that some patients were distressed psychologically at the time of doing the study as a result of their adverse experience. Arrangements were made for them to be referred to the social worker and a psychologist for further counselling if they so wished

5. RESULTS

5.1 Incidence and distribution of placental abruption

Sixty patients were recruited with the diagnosis of placental abruption from the 18th of May 2013 to 31st September 2013. All had singleton pregnancies. There were 13734 deliveries during the study period at the CHBAH. The incidence of placental abruption was found to be 0.4% in this study. There were 34 stillbirths and 26 live births.

The youngest patient was 18 years and the oldest patient was 43 years. The mean age of patients was 28.2 ± 6.8 years.

5.2 Gestational age on admission

The mean gestational age at presentation was 32 ± 4.7 weeks. As shown in table 5.1, 85% of the patients had gestational ages less than 37 weeks.

Table 5.1 Gestational age at admission (n=60)

Gestational age	N	%
20-25	4	6.6
26-30	20	33
31-36	27	45
37-40	9	15

5.3 Risk factors

The maternal risk factors for placental abruption are presented in Table 5.2. The majority of patients had parity of 1 and more accounting for 46(76.6%) of the cases. Placental abruption occurred in 4(6.7 %) of patients that had a previous history of placental abruption. Placental abruption occurred in 7 (11.7%) of patients that had previous caesarean section. Smoking as a risk factor was found in 5 (8.3%).

Table 5.2 Demographic characteristics of patients with placental abruption

Characteristic	N	%
Age in years		
<20	7	11.6
21-24	16	26.6
25-29	12	20
30-34	12	20
35-39	9	15
≥ 40	4	6.6
Parity : 0	14	23
:≥1	46	76.6
Race : African	56	93.
: other	4	6.7
Booked antenatal care	53	83
Previous placental abruption	4	6.7
Previous caesarean section	7	11.7
Smoking	5	8.3
Cocaine use	0	0

5.4 Pregnancy characteristics

As shown in Table 5.3, 55% of patients had hypertensive disorders. Pre-eclampsia was found in 24(40%) of the patients, but 15(25%) of those patients had severe pre-eclampsia. Gestational hypertension occurred in 5(8.3%) cases and chronic hypertension occurred in 4 (6.7%) of the cases. In the study fibroid uteri were found in 2 (3.3%) of the cases.

Table 5.3 Pregnancy characteristics of patients with placental abruption (n=60)

Characteristic	N	%
Mild pre-eclampsia	9	15
Severe pre-eclampsia	15	25
Chronic hypertension	4	6.7
Gestational hypertension	5	8.3
Gestational diabetes	1	1.7
HELLP syndrome	5	8.3
Fibroid uterus	2	3.3

5.5 Clinical presentation

Seventy percent of subjects in the subgroup that had stillbirths presented with vaginal bleeding and 27(79.4%) had abdominal pain. In the subgroup that had live births, 18 (69.2%) of cases had vaginal bleeding and 9 (34.6%) had abdominal pain. Retro-placental clots were detected on ultrasound in 13 (41.9%) of the subjects in the stillbirth subgroup and in 3(12.5%) of the cases that had live births.

Of the sixty patients only 24(20.5%) of the patients had CTG on admission: In the subgroup that had stillbirths, category 1 was seen in 1(2.9%), category 2 was found in 1 (2.9%) and category 3 was found in 5(14.7%) of subjects. In the subgroup that had live births, category 1 CTG was found in 5(19.2%), category 2 was found in 5(19.2%) and category 3 was found in 7(26.9%) of the cases. As shown in Table 5.4, a systolic blood pressure of ≥ 140 mmHg was found in 5(14.7%) of cases in the stillbirth subgroup versus 7 (26.9%) in the group that had live births. A systolic blood pressure of ≥ 160 mmHg was found in 11(32.3%) of cases that had still births versus 6(23%) in the subgroup that had live births.

The mean systolic blood pressure was 137 ± 33 mmHg in the group of patients that had still births versus the mean systolic blood pressure of 143 ± 26 mmHg in the group that had live births. A diastolic blood pressure of ≥ 90 mmHg was found in 16(47%) of the cases in the placental abruption group that had stillbirths versus 3 (8.8%) in the group that had live births.

A diastolic blood pressure of ≥ 110 mmHg was found in 5(14.7%) in the group that had still births versus 8 (30.7%) in the group that had live births. The mean diastolic blood pressure was 86 ± 21 mmHg in the group that had stillbirths versus the diastolic blood pressure mean of 89 ± 21 mmHg in the group that had live births. Oligohydramnios was found in 4(15.3%) of the subjects in the placental abruption subgroup that had live births. Pre-labour rupture of membranes was found in 1(2.9%) of subjects in the placental abruption group that had

stillbirths and 2(7.7%) was found in the subgroup that had live births. There were no patients that presented with a history of trauma.

Table 5.4 Clinical manifestations of placental abruption on admission

Characteristic	Stillbirths (n=34)		live births (n=26)		P -value
	N	%	N	%	
Vaginal bleeding	24	70.6	18	69.2	1.00
Abdominal pain	27	79.4	9	34.6	<0.01
Ultrasound finding of Retro placental clot	13	41.9	3	12.5	0.02
No Retro placental clot on ultrasound	18	58.1	21	87.5	
CTG findings					N/A
category 1	1	2.9	5	19.2	
category 2	1	2.9	5	19.2	
category 3	5	14.7	7	26.9	
Intrauterine fetal deaths	25	73.5	0	0	
Not done	2	5.9	9	36.6	
cervical dilatation					0.39
≤3cm	27	79.4	18	69.2	
≥4cm	7	20.6	8	30.7	
SBP					
≥140	5	14.7	7	26.9	0.33
≥160	11	32.3	6	23	0.57
DBP					
≥90	16	47	3	8.8	<0.01
≥110	5	14.7	8	30.7	0.21
Oligohydramnios	0	0	4	15.3	0.03
IUGR	1	2.9	3	11.5	0.31
Trauma	0	0	0	0	1.00
Prom	1	2.9	2	7.7	0.57

5.6 Laboratory results

The mean maternal haemoglobin was 7.8 ± 2 g/dl in the placental abruption group that had stillbirths. In the group that had stillbirths, 17 (50%) of the patients had haemoglobins that were <7 g/dl and 10 (29.4%) had haemoglobins between 7-9g/dl and 7(20.5%) had haemoglobins of ≥ 10 g/dl. In the group that had live babies, 2(7.7%) of patients had haemoglobins less than 7g/dl, the mean haemoglobin was 12 ± 2 g/dl, and about 24(92%) of the patients had haemoglobins that were ≥ 10 g/dl. The mean maternal urea was 2.8 ± 1.8 mmol/L and the mean creatinine of 61 ± 22 μ mol/l in the group that had live births. The mean maternal urea was 5.5 ± 3.7 mmol/l and mean creatinine of 150 ± 149 μ mol/l in the group that had still births. In the placental abruption subgroup that had live births the mean platelet count was 218 ± 77 μ /l and the mean INR was 1.06 ± 0.2 . The mean PTT was found to be 28 ± 7 seconds in this group. In the group that had still births the mean platelet count was 123μ /l ± 67 and the mean INR was 1.31 ± 0.5 . The mean PTT was found to be 35 ± 11 seconds in this group.

Table 5.5. Biochemistry of the patients compared between the group that had live births and those with stillbirths.

Laboratory test	Stillbirths (n=34)		Live births (n=26)		P-value
	N	%	N	%	
Haemoglobin level					<0.01
<7g/dl	17	50	2	7.7	
7-9g/dl	10	29.4	0	0	
≥10g/dl	7	20.5	24	92	
Platelet count					<0.01
<50	5	14.7	0	0	
50-99	11	32.2	1	3.8	
100-149	4	11.7	5	19.2	
≥150	14	41	20	76.9	
Urea level					0.05
<5	21	61.7	19	90.4	
5-9.9	8	23.5	2	9.5	
≥10	5	14.7	0	0	
Creatinine level					<0.01
<75	11	32.3	16	76	
75-124	12	35.2	5	23.8	
125-249	5	14.7	0	0	
≥250	6	17.6	0	0	
INR					0.20
<0.95	0	0	1	33.3	
0.95-0.99	4	36.3	1	33.3	
1.00-1.49	6	54.5	1	33.3	
≥1.50	2	18.1	0	0	
PTT					0.86
15-29	3	25	1	33.3	
30-49	7	58.3	2	66.6	
≥ 50	1	8.3	0	0	

5.7 Maternal outcomes and hospitalisation

PPH occurred in 15 (44%) of patients that had placental abruption with stillbirths compared to 1(3.8%) of patients that had placental abruption with live births.

Haemorrhagic shock was observed in 2(5.9%) of patients that had placental abruption with stillbirths. Acute kidney injury was seen in 20(58.8%) of patients that had stillbirths compared to 3 (11.5%) in the group that had live births. DIC was observed in 8 (23.5%) patients that had stillbirths. Couvelaire uterus was observed in 9(26.4%) of cases that had stillbirths. Hysterectomies were performed in 2(5.8%) of the cases. A total of 5(14.7%) of patients required ICU admission in the placental abruption group that had stillbirths. The duration of hospital stay in the patients that had live births, 50% stayed for less than 4 days and 3.85% stayed for ≥ 12 days. In the group that had stillbirths, 17.6% of patients stayed for less than 4 days, 38.2% stayed in the hospital between 4 to 7 days and 26.5% stayed for ≥ 12 days. .

Table 5.6 Maternal outcomes giving details on the complications and mode of delivery

Complication	Stillbirths (n=34)		Live births (n=26)		P-value
	N	%	N	%	
DIC	8	23.5	0	0	<0.01
PPH	15	44.1	1	3.8	<0.01
SHOCK	2	5.9	0	0	0.50
Renal failure	20	58.8	3	11.5	<0.01
Couvelaire uterus	9	26.4	0	0	<0.01
ICU Admission	5	14.7	0	0	0.06
Hysterectomy	2	5.8	0	0	0.50
Mode of delivery					0.01
NVD	15	44	4	15.3	
C/S	18	52.9	22	84.61	
Vacuum delivery	1	2.9	0	0	

In the placental abruption group that had stillbirths, 10(29.4%) of patients required < 4 units of blood and 13(38.2%) of patient's required \geq 4 units. In the group that had live births, 6(23%) required blood transfusion and all required less than 4 units of blood. In the group that had stillbirths 13(38.1%) of the patients required the transfusion of fresh frozen plasma. No patients in the live birth group required transfusion of fresh frozen plasma.

Table 5.7 Transfusion of Blood products and estimated blood loss

Complication	Stillbirths (n=34)		live births (n=26)		P-value
	No	%	N	%	
Units of blood transfused					
< 4	10	29.4	6	23	0.77
≥4	13	38.2	0	0	<0.01
Units of FFP'S transfused					
<4	12	35.2	0	0	<0.01
≥4	1	2.9	0	0	1.00
Units of platelets transfused	0	0	1	3.8	1.00
Blood loss					
<500ml	7	22.5	5	19.2	0.03
500-999ml	13	41.9	19	73.0	
1000-1999	4	12.9	2	7.6	
≥2000	7	22.5	0	0	

5.8 Percentage of an abruption

As shown in Table 8, 15 (62.5) % of patients in the group that had stillbirths had complete placental abruption and 7(29%) of patients had an abruption between 50-90%. In the group that had live births, only 15 of the 26 patients had their degree of placental separation quantified and less than 50% of placental separation was found.

Table 5.8 Frequency distribution by the percentage of an abruption

Percentage	Still births (n=24)		Live births (n=15)		P-value
	N	%	N	%	
<50%	2	8.3	15	100	<0.01
50-99%	7	29	0	0	
≥100%	15	62.5	0	0	

5.9 Birth weight at delivery

The mean birth weight was 1760 ±89.3g in the stillbirth group compared to 1983± 998g in the group that had live births.

Table 5 9 Frequency distribution according to birth weight

Birth weight	Still births (n=34)		Live births (n=26)		P-value
	N	%	N	%	
<500	1	2.9	0	0	0.76
500-1499	15	44.1	10	38.4	
1500-2499	10	29.4	8	30.7	
≥ 2500	8	23.5	8	30.7	

5.10 Neonatal outcomes

As shown in Table 5.10, there were 5(19.2%) babies that had birth asphyxia and 7(26. 9%) had APGAR scores of 7-8. ICU admission was required in 11(42.3%) of the cases.

Table 5.10 Neonatal out comes detailing the APGAR score, neonatal ICU admission

APGAR Score at 5minutes	N	%
<7	5	19.2
7-8	7	26.9
9-10	14	53.8
ICU admission	11	42.3

6 DISCUSSION

In this study 60 patients were recruited and the incidence of placental abruption was found to be 0.4% .This was comparable to the finding by Tikannen et al who reported an incidence of 0.42% in a study that was done in a tertiary referral university hospital in Finland (20).In contrast, an incidence of 1% has been reported in a Japanese study on (5, 6) perinatal outcome of placental abruption modified by clinical presentation. Chris Hani Baragwanath is a tertiary referral hospital for high risk patients and therefore, the incidence is similar to what has been reported in the literature.

The risk factors that were found in this study were similar to risk factors reported in other studies. A history of a previous placental abruption was reported in 6.7% of the patients and this was similar to what was reported by Tulpar et al (8). It is important to counsel patients about the risk of recurrence with subsequent pregnancies.

One patient was a 34 year old G3P1, who was a poorly controlled chronic hypertensive and had placental abruptions with stillbirths at 24 and 36 weeks in previous pregnancies and suffered another abruption at an early gestation of 20 weeks. The occurrence of abruptions from as early as 20 to 24 weeks of gestation has been documented in case reports (3).

The patient was also diagnosed with anti-phospholipid syndrome and Enoxaparin 40mg daily and aspirin 75mg daily was initiated at 12 weeks of gestation. Both inherited and acquired thrombophilia have been implicated as risk factors for placental abruption. Anti-phospholipid syndrome is an acquired thrombophilia which is associated with placental vascular thrombosis, decidual vasculopathy and placental infarction.

Eighty three percent of the study subjects were booked patients. This is in contrast to findings by Bibi et al from Pakistan who reported that lack of antenatal care was the risk factor for placental abruption (4).

Previous caesarean section was reported in 11.7% of the subjects, which was higher than the 7.8% incidence reported in a study from Thailand on pregnancy outcomes in placental abruption (1).

Smoking was found in 8.3% of the patients but information about pack history was not part of the study. Studies have reported a dose dependent relation between placental abruption and smoking (19).

In this study we also found a higher incidence of 76.6% of placental abruption in patients that had parity of one and more. A lower incidence of 23.3% was found in primigravida patients. There was no increase in the incidence of placental abruption with advanced maternal age as previously reported by Tulpur et al (8).

Hypertensive disorders are important known risk factors for placental abruption (13, 14). During the study period pre-eclampsia was reported in 40% of the patients and severe pre-eclampsia was reported in 25% of the patients. The incidence of chronic hypertension was 6.7% .This is in contrast to conclusions from other studies that found chronic hypertension not to be a risk factor for placental abruption (15). However, chronic hypertension with super-imposed pre-eclampsia was not found to be a risk factor as previously reported by Ananth et al as there were no patients in this study that had chronic hypertension with super-imposed pre-eclampsia (15). HELLP syndrome was found in 5 patients with severe pre-eclampsia.

In this group of patients with hypertensive disorders only two patients recorded a single clinic visit whilst the rest were booked. The majority of these patients were admitted with

uncontrolled blood pressures of $\geq 140/90$ mmhg. Detailed information about the compliance of treatment was not part of the scope of the study. Hence, possible reasons for uncontrolled blood pressures requiring admission cannot be attributed to non-compliance or poor optimisation. In some of the patients the abruption occurred while admitted in the ward for blood pressure optimisation.

One patient had excessive anticoagulation and this was suspected to be the cause of the abruption. She was a 35 year old P4G5 known cardiac patient with mitral stenosis who presented with a sub-therapeutic INR level of 1.5 and was admitted to optimise her anticoagulation. Her INR level was noted to be increased to 5.6 after she started having per vaginal bleeding.

Over-warfarinization was suspected to be the cause of placental abruption as she did not have other risk factors. First trimester fetal exposure to warfarin in the first trimester is documented to cause warfarin embryopathy. In the later trimesters it may cause fetal bleeding, still births and neonatal deaths but overwarfarinization has not been found to be the risk factor for placental abruption according to the literature.

Uterine myomas were reported in 3.3% of the patients. A study on ultra-sound diagnosis of uterine myomas and complications in pregnancy found that placental abruption was evident in those women that had myomas sizes greater than 200cm³ and with sub mucosal myomas. Therefore it is important to counsel pregnant patients with uterine myomas about the risk for placental abruption.

This study further divided patients according to whether they had live babies or dead babies. Of the 34 patients that had still births, 52.9% of the patients had caesarean section deliveries compared to 84.6% of the patients that had caesarean section deliveries in the group that had

live babies. The remaining 15.4% of patients with live babies delivered by normal vaginal delivery as the delivery was imminent or the placental abruptions was undiagnosed.

Department protocol is to aim for delivery by normal vaginal route if an intra-uterine fetal death is diagnosed with an abruption provided there is no maternal compromise. This management protocol is in keeping with recommendations by RCOG guide lines (47). Indications for delivery by caesarean section in patients with intra-uterine fetal deaths were compromised maternal condition and unfavourable cervical Bishop Scores for induction of labour. The higher caesarean section rate in the placental abruption group that had live babies can be justified by department management protocol. All patients who presents with a live and viable baby with placental abruption are delivered by caesarean section unless vaginal delivery is imminent.

Haemorrhagic shock occurred in 5.9% of the cases that had stillbirths. One of the patients with shock was diagnosed clinically with DIC when her blood samples for coagulation screen wouldn't clot .She was a 21 year old P1G2 patient who was admitted to labour ward in the advanced phase of labour and a cervical dilatation of 9cm. An ultrasound revealed an intrauterine fetal death but no retro-placental clot was noted. Soon after admission to labour ward she started bleeding actively, her blood pressure started dropping and her level of consciousness was also altered. A diagnosis of shock was made and resuscitation was commenced. She did not meet the criteria for assisted vaginal delivery nor was delivery imminent. A caesarean section was performed in accordance with departmental protocol for resuscitation after maternal cardio-pulmonary arrest where cardiac output is not restored within four minutes of commencing resuscitation. Post-delivery examination of the placenta revealed 100% placental abruption. Resuscitation was successful after caesarean section. She was subsequently admitted to ICU for 7 days and discharged after 5 days post ICU discharge.

She was also diagnosed with acute kidney injury in ICU which recovered completely. This case highlights the dangers of concealed abruptions and the difficulty in diagnosis as well as the possible rapid deterioration of maternal condition. Hence, there should be a high index of suspicion in intra-uterine fetal deaths of unknown aetiology.

Acute kidney injury was found in 58.8% of the patients which was higher than the finding by Tulpar et al (8). It was difficult to differentiate whether the kidney injury was as a result of the underlying disorder as some of the patients had hypertensive disorders which were complicated by renal dysfunction or if it was as a result of placental abruption itself.

A higher incidence of acute kidney injury was found in patients that had stillbirths. All patients were managed conservatively except two patients that required dialysis, but they all recovered completely. Those that were treated conservatively were given intravenous fluid and input and output monitoring was done for them.

Patients that had stillbirths also had a higher incidence of PPH. One possible explanation could be that there was a higher incidence of 26.4% of Couvelaire uteri which were found in patients that had still births. Pitaprom and Tulpar et al reported incidences of Couvelaire uteri of 16.5% and 6% respectively (1, 8). Couvelaire uteri were found in patients that had placental abruptions of more than 50%. Couvelaire uterus is a risk factor for uterine atony (40). Uterine atony was found in 10 patients. All were managed medically with utero-tonics such as Syntocinon, Ergometrine and prostaglandin f2 alpha. If there was no response to medical treatment, conservative surgical management with a B-Lynch suture was performed. Six B-lynch sutures were performed and all six patients presented with massive placental abruptions of between 70% and 100% .They all had atonic uteri secondary to Couvelaire uteri and failed medical treatment. Only one patient had a hysterectomy secondary to uncontrolled PPH. She was a 19 year old patient G1P1 at 36 weeks who had an 80%

abruption with a dead baby. Intra-operatively the patient lost more than 2litres of blood and was suspected to be in DIC as she was oozing from the uterine incision wound and also had an atonic uterus. The patient received 9 units of blood and 6 units of FFP. The atonic uterus did not respond to medical treatment which included Syntocinon, syntometrine and f2 alpha nor a B-lynch suture.

The other hysterectomy in the study was for puerperal sepsis. This patient was a 27 G2P1 at 33 weeks who had an atonic uterus secondary to Couvelaire uterus. She also received both medical and surgical management with a B- lynch suture and good uterine contractility was achieved. On day 4 post operation the patient presented with abnormal septic markers. Findings at relook laparotomy revealed a septic uterus and total abdominal hysterectomy was performed. Her identifiable risk factors for puerperal sepsis were the prolonged duration of her caesarean section and intra-operative complications.

In this study 48% of patients required blood transfusion. This was comparable to the finding by Bibi et al who found that 50% of the patients required blood transfusions (4).The need for blood transfusion was found in 67.6% of the 34 patients that had still births. In the stillbirth group there were five patients that required massive transfusion, 2 (5.88%) required 5 units of blood, 2(5.88%) required 6 units and the other patient received 9 units. The decision to transfuse blood was based on the clinical judgement of the attending clinician or correlated to the patient's haemoglobin.

ICU admission was required in 5 patients. Indications for ICU admission were either contra-indications to extubation or if the patient had an underlying medical condition such as a cardiac condition. One patient was a known cardiac patient with mitral stenosis and therefore required post-operative observations in ICU. The other four patients could not be extubated as they had acidosis as the result of the PPH that occurred intra-operatively.

The other significant common observation was that all 5 patients had 100% placental abruption and 4 complicated with PPH. All these patients were successfully resuscitated and discharged. These observations suggest that an abruption of more than 50% may carry a high morbidity.

There was a high incidence of babies born prematurely, which is in agreement with other studies (20, 45). In this study 76.4% of babies had low birth weight in the group that had still births compared to 69.4% in the group that had live births. The strong association of placenta abruption with prematurity may be attributed to the management of placental abruptions with delivery irrespective of the gestation. The other important contributor to neonatal morbidity is the contra-indication to tocolysis for induction of fetal lung maturity with steroids. In addition, babies born to mothers with placental abruptions are usually smaller for gestation as fetal growth restriction may occur with maternal conditions such as hypertensive disorders resulting in placental insufficiency. (44).

The other important morbidities include birth asphyxia and acute respiratory distress syndrome which may require ICU admission for ventilation and monitoring. In this study the incidence of babies that required ICU was found to be 42.3% of the babies. The reasons for ICU admissions were low birth weight, prematurity, acute respiratory distress syndrome and birth asphyxia.

The most significant finding was that patients with more than 50% placental abruption were more likely to have a stillbirth which was similar to the finding by Ananth et al (45). The clinical presentation of the patients was similar to what has been reported by the literature (9). In this study the patients presented with per vaginal bleeding and abdominal pains unless they had a concealed abruption. Fetal distress was found in 26.9 % of the patients that had live babies which is a common presentation in patients that have placental abruption.

Limitations

1. There were patients that were diagnosed, treated and discharged from the high care area before the primary investigator identified them as eligible for the study.
2. The Department protocol is that all complicated patients be admitted to ward 64. During the study period some of the patients were admitted to postnatal wards for uncomplicated patients owing to bed shortages. These patients were mostly delivered by normal vaginal delivery. It was not part of the study methodology to enrol patients from those post natal wards. Hence, some patients with abruption may have been missed during the study period
3. Incorrect diagnoses of the patients. Patients who were thought to have placental abruption but post -delivery findings were negative. These patients were not included as there was no demonstration of retro-placental clot at delivery.
4. Some of the patients declined consent to be included in the study
5. There was also poor documentation of the events and missing records.
6. The other weakness in the study was that after delivery of the placenta, clinicians did not quantify the percentage of the abruption. The only documentation found was of 'small' and 'big' clots. This limited us in understanding whether the bad outcome was as result of the placenta separating completely or partially.
7. Not all the patients with placental abruptions had renal function tests. If the patient was found to be stable, the investigation was not performed. Hence, there could be an under-reporting of acute kidney injury associated with placental abruption. According to the departmental protocol all patients should have a baseline renal function assessment on admission if an abruption is suspected but that opportunity was missed in some patients.
8. Babies that were born alive were not followed up to report on late neonatal deaths after 7 days. This was not part of the scope of this study.

Conclusion

Placental abruption carries a significant risk for both maternal and fetal morbidity. The prevalence of placental abruption was found to be 0.4% in this study which was similar to what has been reported in the literature. Antenatal risk factors associated with this condition must be identified and the importance of antenatal care should be emphasised. In the study hypertensive disease remains an important risk factor for this condition. These risk factors can be obtained from the patient's history and can thus help in reducing the morbidity and mortality associated with this condition. Prompt diagnosis and early referral of the patients for tertiary level care can result in better outcomes in these patients. A significant finding of this study, was the worse outcomes in patients that had placental abruption and stillbirths. This signifies the importance of early recognition and proper referral of the patient to ensure adequate management.

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8. APPENDICES

DATA SHEET

Descriptive study of severe morbidity in women with abruption placenta

Research study no: _____

Age: _____

Race: ___ African ___ Caucasian ___ Asians ___ Mixed

Antenatal attendance: yes/ no / how many visits _____

Gestational age at presentation: By sonar/LMP/Palpation

OBSTETRIC HISTORY

Parity : _____

Gravidity : _____

Previous history of placental abruption: yes / no

Previous caesarean section: yes /no if yes how many _____

CORMOBIDITIES

Hypertensive disorders: yes / no if yes which one (pre-eclampsia/chronic hypertension/chronic hypertension with superimposed pre-eclampsia)

Gestational diabetes: yes or no

HABITS

Smoking: yes / no _____ cocaine use : yes /no

CLINICAL PRESENTATION

Vaginal bleeding _____ Abdominal pain _____ CTG: category 1/2/3

CLINICAL DIAGNOSES

General condition

Colour _____ normal or pale Pulse _____ Blood pressure _____ respiratory

rate _____

Abdominal palpation: Tenderness yes/no _____ consistency: hard/soft symphysis-fundal height: _____ in cm

Vaginal Exam: bleeding Present/absent,

Cervical dilatation: os closed / open if open how many cm

Syntocinon: given / not given _____

PERINATAL OUTCOMES OF LIVE BABIES

APGAR SCORE AT 5 MINUTES	
BIRTH WEIGHT	
PREMATURITY	
ICU ADMISSION	Yes /No

DEAD BABY: FRESH STILL BORN OR MACERATED STILL BORN

BIRTH WEIGHT:

GESTATIONAL AGE:

INVESTIGATIONS ON ADMISSION AND THE WORST RESULTS TO BE USED

HAEMOGLOBIN	
-------------	--

PLATELETS	
UREA	

CREATININE	
INR	
PTT	
SONAR(RETROPLACENTAL CLOT)	YES/NO

MATERNAL OUT COMES

DIC	
POSTPARTUM HEAMORRHAGE	BLOOD LOSS= 500ML OR 1000ML AND MORE
SHOCK	BP= PULSE= RESPIRATORY RATE= URINE OUTPUT= MENTAL STATUS
RENAL FAILURE	UREA= CREATININE=
NEED FOR BLOOD TRANSFUSION	NO OF PACKED CELLS USED NO OF PLATELETS AND FFPS
LENGTH OF HOSPITAL STAY	
ICU ADMISSION	YES /NO
CEASARIAN HYSTERECTOMY	YES/NO

DURATION OF ICU STAY:

REASON FOR ICU ADMISSION:

MODE OF DELIVERY:

COMPLETE OR PARTIAL PLACENTAL ABRUPTION

ESTIMATED BLOOD LOSS:

FINAL OUT COME: discharge / death

OTHER COMPLICATIONS AT DELIVERY:

STUDY PARTICIPANT INFORMATION SHEET

TITLE: DESCRIPTIVE STUDY OF MORBIDITY IN WOMEN WITH ABRUPTIO PLACENTAE

Good day. My name is Dr Bongiwe Nkomo. I work in the department of obstetrics and gynaecology at Chris Hani Baragwanath hospital. I would like to invite you to participate in a research study entitled “A descriptive study of morbidity in women with abruptio placentae.”

Why are we doing the study?

The reason why am doing the study is because you have had the condition where the placenta separated before the baby is born and as result of that there is a risk of you developing the complications associated with this condition.

What will happen if you take part in this study?

If you agree to take part in the study, I would like to ask a few questions to know more about yourself and would also like to review your records, The information that will be collected is that pertaining to the risk factors that may have predisposed you into getting this condition, all complications that have occurred as result of this condition, blood results and the baby's outcome. All the information provided will be kept confidential. There are no costs to you for participating in the study.

What are benefits of being in the study?

By participating in the study you may not receive a direct benefit. However your participation in the study may help us understand the condition in this community better.

What are the risks and discomfort of being in this study?

You may experience some distress because of what has happened to you but we will arrange a social worker or psychologist to speak with you if you would like that.

What happens if you do not agree to take part in this study?

Your participation in the study is voluntary.

You may withdraw from the study at any time, and your withdrawal will not change your treatment. You will be treated the same whether you are part of the study or not.

How is the information collected during the study going to be kept confidential?

All the participants in the study will be assigned a research study number and no identification information will be on the questionnaire .The information will only being accessible to the researcher, supervisor and the ethics committee.

What if you have more questions you wish to ask about study?

If you have questions about the study please ask me now or if questions later have you may call me on 0833406137.

If you have concerns about your rights as a person taking part in a research study you may contact the chair of the ethics committee, Professor Cleaton Jones (0117172301)

CONSENT FORM

INFORMED CONSENT

TITLE: Descriptive study of morbidity in women with abruptio placentae.

- The study has been fully explained to me (or the information sheet about this study has been read to me if I take part in the study) and I understand what will be required of me and what will happen to me if I take part in the study.

My questions concerning this study have been answered by the researcher.

- I understand that I may withdraw from this study at any time without giving a reason and without being penalised or losing benefits.
- I agree to take part in the study.

Participant name:

Verbal consent obtained by:

Date:



UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Dr Bongive Nkomo

CLEARANCE CERTIFICATE

M120758

PROJECT

Descriptive Study of Severe morbidity in Women with Abruptio Placentae and Intra-uterine Deaths, Fresh Still Born and Early

Neonatal Deaths

INVESTIGATORS

Dr Bongive Nkomo.

DEPARTMENT

Department of Obstetrics & Gynaecology

DATE CONSIDERED

27/07/2012

DECISION OF THE COMMITTEE*

Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 07/01/2013

CHAIRPERSON


(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable

cc: Supervisor : Dr J Jeebodh

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.

I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES..

MEDICAL ADVISORY COMMITTEE
CHRIS HANI BARAGWANATH ACADEMIC HOSPITAL
PERMISSION TO CONDUCT RESEARCH

Date: 04 July 2012

TITLE OF PROJECT: Descriptive study of severe morbidity in women with abruptio placentae and intrauterine deaths/fresh stillborn/ early neonatal deaths

UNIVERSITY: Witwatersrand:

Principal Investigator: Dr B Nkomo

Department: Obstetrics and Gynaecology

Supervisor (If relevant): Prof E Buchmann

Permission Head Department (where research conducted): Yes

Date of start of proposed study: 1 August 2012

Date of completion of data collection: 30 January 2013

The Medical Advisory Committee recommends that the said research be conducted at Chris Hani Baragwanath Hospital. The CEO /management of Chris Hani Baragwanath Hospital is accordingly informed and the study is subject to:-

- Permission having been granted by the Committee for Research on Human Subjects of the University of the Witwatersrand.
- the Hospital will not incur extra costs as a result of the research being conducted on its patients within the hospital
- the MAC will be informed of any serious adverse events as soon as they occur
- permission is granted for the duration of the Ethics Committee approval.

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 Recommended

(On behalf of the MAC)

Date: 04 July 2012

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 Approved/Not Approved

Hospital Management

Date: 06/07/12