International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Clinical Profile and Maternal–Perinatal Outcomes of Antepartum Haemorrhage in Primigravida

Srilakshmi Kundena[1], Sushma G[2], Sirisha Reddy Ch[3]

[1]Associate Professor, Department of Obstetrics and Gynaecology, Government Maternity Hospital, Sultan Bazar, Koti, Osmania Medical College, Hyderabad, Telangana, India
[2]Associate Professor, Department of Obstetrics and Gynaecology, Government Medical College, Nirmal, Telangana, India
[3]Postgraduate Student, Department of Obstetrics and Gynaecology, Government Maternity Hospital, Sultan Bazar, Koti, Osmania Medical College, Hyderabad, Telangana, India

Corresponding Author:

Dr. Kundena Srilakshmi
Associate Professor, Department of Obstetrics and Gynaecology, Government Maternity Hospital, Sultan Bazar, Koti, Osmania Medical College, Hyderabad, Telangana, India

Received: 01-04-2025
Accepted: 28-05-2025
Available online: 02-06-2025

ABSTRACT

Background: Antepartum haemorrhage is an obstetric emergency that may rapidly compromise maternal circulation and placental oxygen transfer. Placental abruption and placenta previa account for most clinically important cases, but their patterns of morbidity differ. Evidence focused specifically on primigravidae remains limited in many Indian tertiary-care settings. The study is designed to describe the clinical profile, associated risk factors, mode of delivery, and maternal and fetal outcomes among primigravidae presenting with antepartum haemorrhage.

Materials and Methods: This prospective observational study included 100 consenting primigravidae with singleton pregnancies and antepartum haemorrhage between 28 and 42 weeks of gestation. The study was conducted over 24 months. Clinical assessment, laboratory investigations, ultrasonography and maternal-fetal surveillance were performed. Outcomes were analysed using IBM SPSS Statistics for Windows, version 28.0. Categorical variables were compared using the Pearson chi-square test or Fisher exact test, as appropriate, with $p<0.05$ considered statistically significant.

Results: The mean maternal age was 23.74 +/- 2.88 years and the mean gestational age at presentation was 35.11 +/- 2.31 weeks. Abruptio placentae accounted for 65% of cases and placenta previa for 35%. Anaemia, pregnancy-induced hypertension and severe preeclampsia were significantly concentrated among women with abruption. Seventy-five percent underwent caesarean delivery, including 50% emergency procedures. Postpartum haemorrhage occurred in 60%, blood transfusion was required in 60%, and 30% required intensive care. There were 80 live births, 15 intrauterine fetal deaths and five stillbirths. Adverse fetal outcome was significantly associated with abruption (exact $p<0.001$). NICU admission and an APGAR score of 5 or less were each more frequent with abruption than with previa (49.2% versus 11.4%; chi-square=14.11, $p<0.001$). Mean birth weight was 2.16 +/- 0.55 kg.

Conclusion: Antepartum haemorrhage in primigravidae was associated with substantial maternal and perinatal morbidity. Placental abruption carried a markedly greater burden of hypertensive risk, postpartum haemorrhage, intensive care use, fetal loss and compromised neonatal condition. Early risk recognition, correction of anaemia, surveillance for hypertensive disorders, rapid referral, ready access to blood components and coordinated obstetric-neonatal care are central to improving outcomes.

Keywords: *antepartum haemorrhage; abruptio placentae; placenta previa; primigravida; postpartum haemorrhage; perinatal outcome; neonatal intensive care.*

INTRODUCTION

Antepartum haemorrhage refers to bleeding from or into the genital tract after the threshold of fetal viability and before birth. The operational definition varies across clinical guidelines and research settings. In the present study, bleeding occurring from 28 completed weeks to delivery was considered antepartum haemorrhage. Although the event may begin with a limited external blood loss, maternal and fetal status can deteriorate quickly, particularly when bleeding is concealed or accompanied by placental separation.

Placenta previa and placental abruption are the two principal placental causes. Placenta previa results from implantation close to or over the internal cervical os and most often presents with painless bleeding. Placental abruption is premature separation of a normally implanted placenta and commonly presents with pain, uterine tenderness, hypertonicity or fetal compromise. Global estimates indicate that placenta previa is uncommon at the population level, yet it contributes disproportionately to emergency caesarean delivery, transfusion and preterm birth [1,2]. Placental abruption is similarly infrequent but carries a high risk of fetal hypoxia, stillbirth, maternal coagulopathy and severe haemorrhage [3,4].

The consequences of antepartum haemorrhage reflect both blood loss and loss of functional placental exchange. Maternal complications include hypovolaemia, postpartum haemorrhage, disseminated intravascular coagulation, acute kidney injury, intensive care admission and, in severe cases, death. Fetal consequences include acute asphyxia, indicated preterm delivery, low birth weight, neonatal intensive care admission and perinatal death [5]. The risk profile is influenced by the underlying cause. Hypertensive disorders are strongly linked to abruption, whereas previous uterine surgery and assisted reproduction are prominent risk factors for placenta previa [6,7].

Primigravidae warrant focused evaluation because previous caesarean delivery and multiparity are absent, allowing the contribution of first-pregnancy factors, particularly anaemia and hypertensive disease, to be examined more clearly. In public tertiary hospitals, many women arrive after referral from peripheral facilities, sometimes with established fetal compromise or significant maternal blood loss. Local data are therefore important for planning blood-bank readiness, theatre access, critical-care support and neonatal services.

The present prospective observational study was undertaken at a high-volume tertiary maternity hospital in Hyderabad to examine maternal and fetal outcomes among primigravidae with antepartum haemorrhage. The study assessed demographic and gestational characteristics, clinical type of haemorrhage, associated maternal risk factors, mode of delivery, maternal complications and immediate fetal and neonatal outcomes. The study was undertaken to assess maternal and fetal outcomes among primigravidae presenting with antepartum haemorrhage at a tertiary-care hospital. It also aimed to describe the clinical presentation and underlying type of haemorrhage, identify maternal risk factors associated with placental abruption and placenta previa, and evaluate the mode of delivery together with major maternal complications. Fetal outcomes were assessed in terms of survival, birth weight, APGAR score category, and requirement for neonatal intensive care. In addition, maternal and neonatal outcomes were compared according to the type of antepartum haemorrhage.

MATERIALS AND METHODS

Study design and setting

A prospective observational study was conducted in the Department of Obstetrics and Gynaecology at Government Maternity Hospital, Sultan Bazar, Koti, Hyderabad, a tertiary referral hospital affiliated with Osmania Medical College, Hyderabad, Telangana, India. The hospital provides round-the-clock emergency obstetric services, operative delivery, blood transfusion support, maternal intensive care and neonatal intensive care. The observational design was selected to document the presentation, management and outcomes of consecutive eligible women without assigning an intervention.

Study duration and sampling

The study was carried out over 24 months during 2023-2025. The thesis did not specify the exact calendar months. All eligible women presenting during the study period were considered for inclusion, which represents a consecutive, hospital-based sampling approach. A total of 100 participants were enrolled. No separate a priori sample-size calculation was documented in the source thesis.

Study participants

The study population comprised primigravidae with a singleton pregnancy who presented with antepartum haemorrhage between 28 and 42 weeks of gestation and provided written informed consent. Women were excluded when they had a multiple pregnancy, bleeding before 28 weeks, a known bleeding disorder, a local cervical or vaginal cause of bleeding, or declined participation. Gestational age was determined from the last menstrual period and available antenatal ultrasonography.

Clinical assessment and data collection
At admission, demographic and obstetric details were recorded using a structured case-record form. Information included maternal age, socioeconomic and booking status, gestational age, referral status, characteristics of bleeding and relevant medical or obstetric risk factors. Initial assessment included pulse, blood pressure, respiratory rate, oxygen saturation, pallor and signs of haemodynamic compromise. Abdominal examination assessed uterine tone, tenderness, contractions, fetal lie, presentation and fetal heart activity. A sterile speculum examination was performed when clinically indicated. Digital vaginal examination was avoided until placenta previa had been excluded.

Laboratory and imaging evaluation
Investigations included complete blood count, blood group and Rh typing, cross-match, coagulation profile, renal function tests, liver function tests and serum electrolytes. Antenatal ultrasonography was used to locate the placenta, assess fetal growth, estimate amniotic fluid and identify findings supportive of placental abruption. Uterine or fetal Doppler studies were performed when indicated by the maternal or fetal condition. Placenta previa was diagnosed primarily by sonographic localization, while abruption was diagnosed using the combined clinical picture, fetal status and imaging findings, recognizing the limited sensitivity of ultrasonography for acute abruption.

Clinical management
Management began with stabilization of airway, breathing and circulation, establishment of intravenous access, collection of blood samples and arrangement of compatible blood components. Crystalloids and blood products were administered according to clinical need. Continuous or repeated maternal and fetal monitoring was undertaken. The decision for expectant management or delivery was individualized according to gestational age, amount and recurrence of bleeding, maternal haemodynamic status, labour, fetal viability and evidence of fetal compromise. Delivery was by normal vaginal delivery, elective lower-segment caesarean section or emergency lower-segment caesarean section, as clinically indicated.

Outcome measures
Maternal outcomes included mode of delivery, postpartum haemorrhage, disseminated intravascular coagulation, intensive care admission, acute kidney injury, puerperal sepsis and requirement for blood transfusion. Fetal and neonatal outcomes included live birth, intrauterine fetal death, stillbirth, birth weight, APGAR category and neonatal intensive care admission. The source thesis reported intrauterine fetal death and stillbirth as separate outcome categories; these classifications were retained for consistency with the primary dataset.

Statistical analysis
Data were organised in Microsoft Excel and analysed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, New York, USA). Continuous variables were summarised as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The Pearson chi-square test was used to compare categorical variables when expected cell counts were adequate. Fisher exact test was used for sparse 2 x 2 tables, and an exact test was used for the 3 x 2 fetal-outcome distribution. All tests were two-sided and $p<0.05$ was considered statistically significant. For preparation of this manuscript, test statistics and p values were recalculated from the tabulated frequencies in the thesis. This was necessary because several values in the narrative and association tables were internally inconsistent. The tabulated NICU frequency of 36 was used, rather than the 30% stated in the thesis summary.

Ethical considerations
The study was undertaken after approval from the Institutional Ethics Committee of Osmania Medical College, Hyderabad. Written informed consent was obtained before enrolment, and clinical care was not altered by participation. Participant information was handled confidentially. The ethics approval number and approval date were not stated in the thesis text and should be added from the official committee letter before submission to a journal.

RESULTS

Participant profile and gestational age
A total of 100 primigravidae with antepartum haemorrhage were evaluated. The mean maternal age was 23.74 +/- 2.88 years. Most participants were 19-25 years old (71%), followed by 26-30 years (26%); three women were aged 18 years. The mean gestational age at presentation was 35.11 +/- 2.31 weeks. Half of the women presented at 34-36 weeks, 30% at 37 weeks or later and 20% at 28-33 weeks (Table 1). This distribution indicates that late-preterm presentation accounted for the largest proportion of admissions.

Table 1: Baseline demographic and gestational profile of participants (n=100)

Variable	Category/statistic	n (%) or mean +/- SD	Inferential p value
Maternal age	18 years	3 (3.0)	Not applicable
	19-25 years	71 (71.0)	
	26-30 years	26 (26.0)	

	Mean age	23.74 +/- 2.88 years	
Gestational age	28-33 weeks	20 (20.0)	Not applicable
	34-36 weeks	50 (50.0)	
	>=37 weeks	30 (30.0)	
	Mean gestational age	35.11 +/- 2.31 weeks	

SD, standard deviation. This table is descriptive; no between-group hypothesis test was prespecified for age or gestational-age distribution.

Clinical type of haemorrhage and mode of delivery

Placental abruption was identified in 65 women and placenta previa in 35. The clinical pattern of bleeding mirrored the etiological distribution: 65 women had painful bleeding and 35 had painless bleeding (Table 2 and Figure 1). Three-quarters of the cohort underwent caesarean delivery. Emergency caesarean section was the most frequent mode of delivery (50%), while elective caesarean section and normal vaginal delivery each accounted for 25% (Table 2).

Table 2: Clinical presentation and mode of delivery (n=100)

Domain	Category	n	%	p/Chi-square
Type of APH	Abruptio placentae	65	65.0	Descriptive
	Placenta previa	35	35.0	
Nature of bleeding	Painful	65	65.0	Descriptive
	Painless	35	35.0	
Mode of delivery	Elective LSCS	25	25.0	Descriptive
	Emergency LSCS	50	50.0	
	Normal vaginal delivery	25	25.0	

APH, antepartum haemorrhage; LSCS, lower-segment caesarean section. Percentages use the total cohort as denominator. No comparative p value was required for this descriptive distribution.

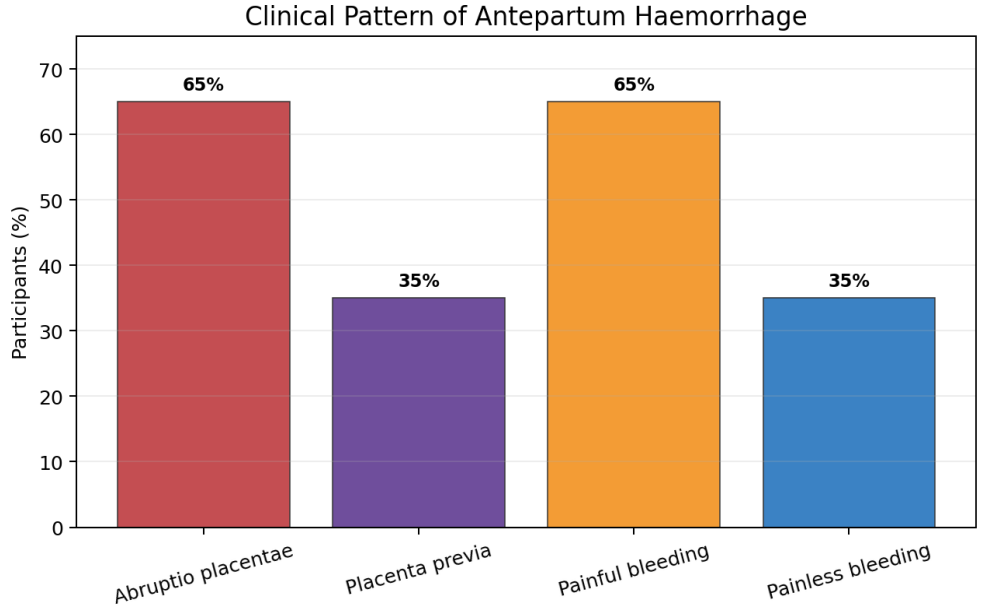

Figure 1: Distribution of the type of antepartum haemorrhage and nature of bleeding among the 100 study participants.

Maternal risk factors according to type of antepartum haemorrhage

Pregnancy-induced hypertension was recorded in 55 women, all of whom were in the abruption group. Anaemia was present in 45 women, including 38 of 65 women with abruption and seven of 35 with placenta previa. Severe preeclampsia was documented in 15 women, all with abruption. Anaemia (Chi-square=13.60, p<0.001), pregnancy-induced hypertension (Fisher exact p<0.001) and severe preeclampsia (Fisher exact p=0.001) were significantly associated with abruption. Hypothyroidism, other recorded risk factors and absence of an identifiable risk factor did not differ significantly between the two APH groups (Table 3 and Figure 2).

Table 3: Maternal risk factors according to type of antepartum haemorrhage

Risk factor	Abruptio (n=65)	Previa (n=35)	Test	p value
Anaemia	38 (58.4%)	7 (20.0%)	Chi-square=13.60	<0.001

Pregnancy-induced hypertension	55 (84.6%)	0 (0.0%)	Fisher exact	<0.001
Severe preeclampsia	15 (23.1%)	0 (0.0%)	Fisher exact	0.001
Hypothyroidism	1 (1.5%)	3 (8.6%)	Fisher exact	0.122
Other recorded risks	10 (15.4%)	5 (14.3%)	Fisher exact	1.000
No recorded risk factor	5 (7.7%)	5 (14.3%)	Fisher exact	0.313

Percentages are calculated within each APH group. Participants could have more than one risk factor, so column totals may exceed 100%. Other risks in the thesis included gestational diabetes, polyhydramnios and Rh-negative status. Two-sided p<0.05 was considered significant.

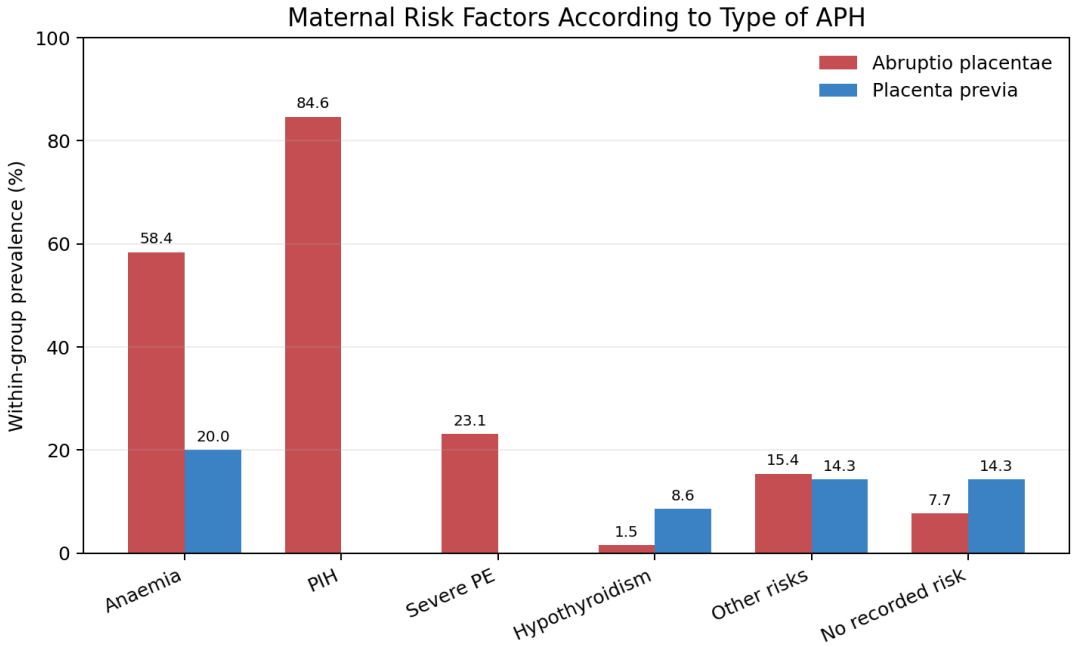

Figure 2: Maternal risk-factor profile in women with abruptio placentae and placenta previa. Bars show percentages within each APH group

Maternal morbidity

Postpartum haemorrhage and blood transfusion were each recorded in 60% of participants. Thirty women required intensive care, nine developed acute kidney injury, four developed disseminated intravascular coagulation and four developed puerperal sepsis. Maternal morbidity was concentrated in the abruption group. Postpartum haemorrhage occurred in 55 of 65 women with abruption compared with five of 35 women with previa (Chi-square=46.89, p<0.001). Intensive care admission was also substantially more frequent with abruption (44.6% versus 2.9%; Chi-square=18.89, p<0.001). Acute kidney injury was associated with abruption (Fisher exact p=0.025). In contrast, the small observed differences for disseminated intravascular coagulation and puerperal sepsis did not reach statistical significance after exact testing (Table 4 and Figure 3).

Table 4: Maternal complications overall and according to type of antepartum haemorrhage

Complication	Overall n (%)	Abruptio (n=65)	Previa (n=35)	Test and p value
Postpartum haemorrhage	60 (60.0)	55 (84.6%)	5 (14.3%)	Chi-square=46.89; p<0.001
Disseminated intravascular coagulation	4 (4.0)	4 (6.2%)	0 (0.0%)	Fisher exact; p=0.295
ICU admission	30 (30.0)	29 (44.6%)	1 (2.9%)	Chi-square=18.89; p<0.001
Acute kidney injury	9 (9.0)	9 (13.8%)	0 (0.0%)	Fisher exact; p=0.025
Puerperal sepsis	4 (4.0)	4 (6.2%)	0 (0.0%)	Fisher exact; p=0.295
Blood transfusion	60 (60.0)	Not reported	Not reported	Comparison unavailable

ICU, intensive care unit. Percentages in the APH columns are within-group percentages. The thesis provided the transfusion total but not its distribution by APH type. Exact p values were recalculated from the reported counts.

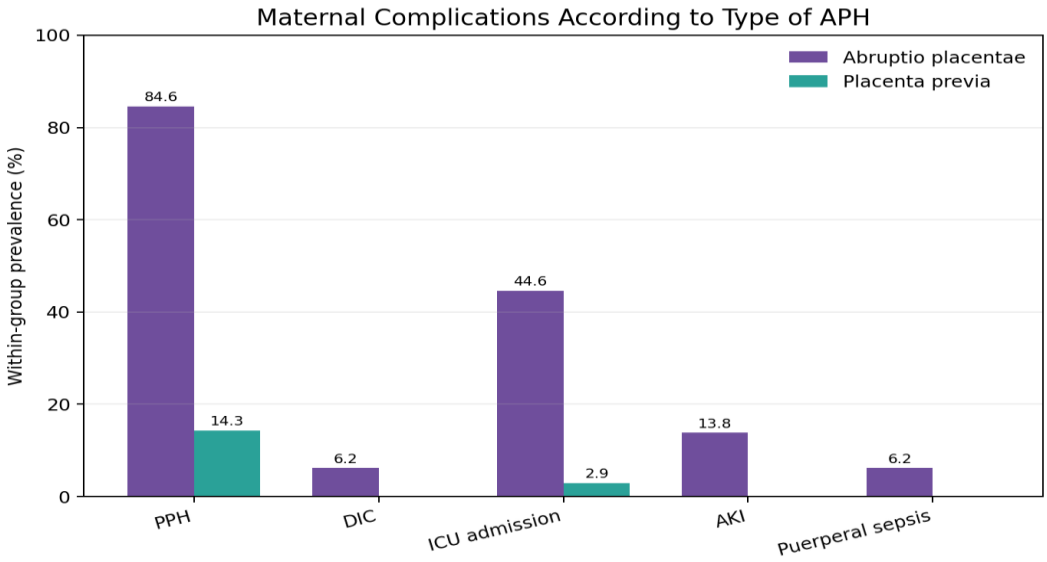

Figure 3: Maternal complications according to the type of antepartum haemorrhage. The transfusion variable is not shown because type-specific counts were unavailable.

Fetal outcome and perinatal loss

Eighty pregnancies resulted in a live birth. Fifteen were classified as intrauterine fetal deaths and five as stillbirths, giving a reported fetal loss of 20%. All fetal deaths occurred in the abruption group. Among women with abruption, 45 (69.2%) had a live birth, 15 (23.1%) had an intrauterine fetal death and five (7.7%) had a stillbirth. All 35 women with placenta previa had a live birth. The distribution of fetal outcome differed significantly by APH type (exact p<0.001; Table 5 and Figure 4).

Table 5: Fetal and immediate neonatal outcomes according to type of antepartum haemorrhage

Outcome	Abruptio (n=65)	Previa (n=35)	Test	p value
Live birth	45 (69.2%)	35 (100.0%)	Exact test for 3 x 2 distribution	<0.001
Intrauterine fetal death	15 (23.1%)	0 (0.0%)		
Stillbirth	5 (7.7%)	0 (0.0%)		
NICU admission: yes	32 (49.2%)	4 (11.4%)	Chi-square=14.11	<0.001
NICU admission: no	33 (50.8%)	31 (88.6%)		
APGAR >5	33 (50.8%)	31 (88.6%)	Chi-square=14.11	<0.001
APGAR <=5	32 (49.2%)	4 (11.4%)		

NICU, neonatal intensive care unit; APGAR, Appearance, Pulse, Grimace, Activity and Respiration. The fetal-outcome exact test compares the complete three-category distribution

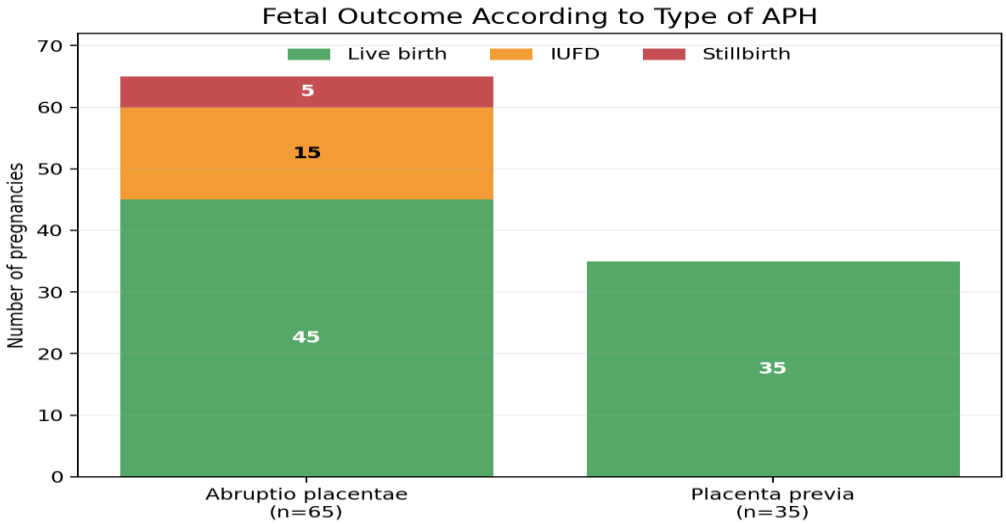

Figure 4: Fetal outcome according to the type of antepartum haemorrhage. All reported fetal deaths occurred in the abruptio placentae group

Neonatal intensive care, APGAR category and birth weight

NICU admission was recorded for 36 neonates in the primary results table. Admission was required for 32 of 65 pregnancies in the abruption group and four of 35 in the previa group. This difference was statistically significant (Chi-square=14.11, p<0.001). An APGAR score of 5 or less was reported in the same number and distribution, 32 in the abruption group and four in the previa group, and was also significantly associated with abruption (Chi-square=14.11, p<0.001; Table 5 and Figure 5).

The mean neonatal birth weight was 2.16 +/- 0.55 kg. Eighteen neonates weighed less than 1.5 kg, 52 weighed 1.5-2.5 kg and 30 weighed more than 2.5 kg. Thus, 70% had a birth weight of 2.5 kg or less (Table 6 and Figure 6). The source data did not provide birth-weight categories separately for abruption and previa, so no comparative test was possible.

Table 6: Distribution of neonatal birth weight (n=100)

Birth-weight category	n	%	Summary/test
<1.5 kg	18	18.0	
1.5-2.5 kg	52	52.0	
>2.5 kg	30	30.0	
Mean birth weight			2.16 +/- 0.55 kg

This was a descriptive distribution. Birth-weight data by APH type were not reported, so a between-group p value could not be calculated.

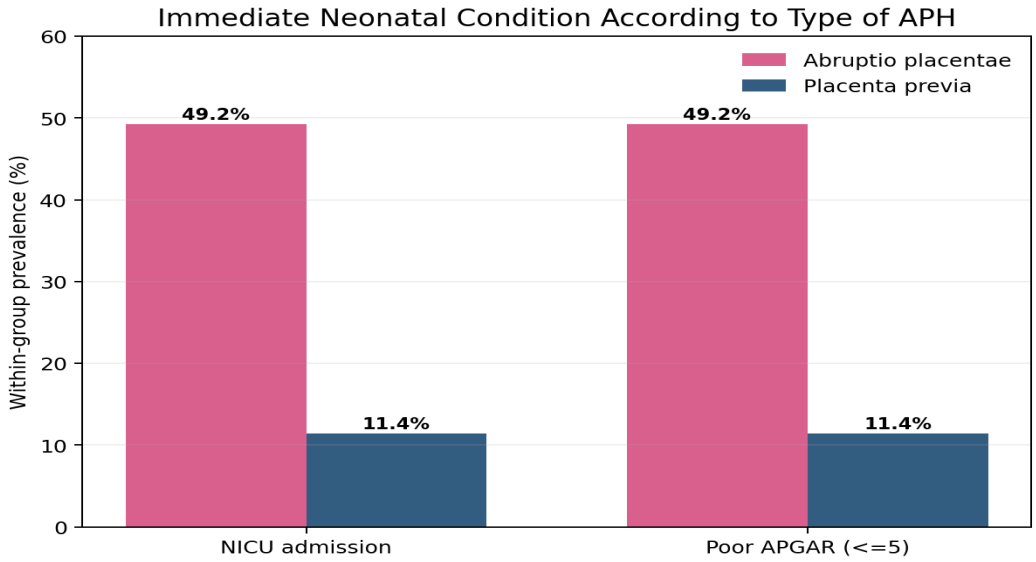

Figure 5: NICU admission and poor APGAR category according to type of antepartum haemorrhage. Percentages are calculated within each APH group

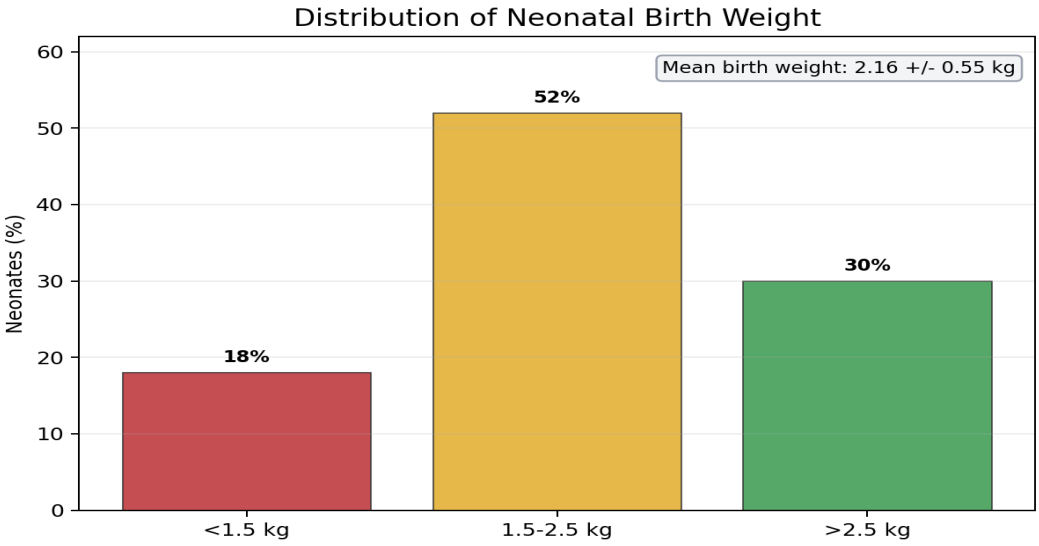

Figure 6: Neonatal birth-weight distribution in the complete study cohort

DISCUSSION

Principal findings

This study documents a high burden of maternal and fetal morbidity among primigravidae admitted with antepartum haemorrhage. Placental abruption was almost twice as frequent as placenta previa. The abruption group also carried the greater clinical burden, including a strong concentration of hypertensive disorders, more postpartum haemorrhage, greater intensive-care use, all reported fetal deaths, more NICU admissions and poorer APGAR categories. These findings support the clinical distinction between the two principal causes of APH: placenta previa commonly produces visible bleeding that prompts intervention, whereas abruption may combine blood loss with sudden loss of placental exchange, uterine hypertonicity, concealed haemorrhage and coagulopathy.

Clinical profile and type of antepartum haemorrhage

The mean gestational age at presentation was approximately 35 weeks, and half of the cohort presented between 34 and 36 weeks. This pattern is clinically relevant because management during the late-preterm period requires a balance between continuing pregnancy for fetal maturation and avoiding recurrent or catastrophic bleeding. The 65% frequency of abruption in this selected hospital cohort should not be interpreted as a population incidence. Tertiary referral centres tend to receive a disproportionate number of severe or complicated cases. Population reviews show that placenta previa and abruption are individually uncommon, but both contribute substantially to preterm delivery and emergency intervention [1,3,9].

Painful bleeding occurred in the same proportion as abruption, while painless bleeding paralleled the prevalence of placenta previa. This concordance is clinically plausible and reflects classic presentations. Nevertheless, symptoms alone cannot reliably establish the diagnosis. Placenta previa requires sonographic localization, preferably using transvaginal imaging when needed, while abruption remains predominantly a clinical diagnosis because ultrasonography may not detect an acute retroplacental clot [7,8].

Maternal risk factors

Pregnancy-induced hypertension was the dominant recorded risk factor and was confined to women with abruption. Severe preeclampsia showed a similar pattern. Hypertensive disease can promote decidual arteriolar injury, placental ischaemia and rupture of maternal vessels, providing a biologically coherent explanation for the association. Earlier meta-analytic and cohort evidence has consistently linked chronic hypertension and pregnancy-associated hypertension with placental abruption [4,16]. In a primigravid cohort, recognition of this relationship is particularly important because prior caesarean delivery and high parity, which often influence placenta previa, are absent.

Anaemia was present in 45% of the cohort and was more frequent among women with abruption. Anaemia may not directly cause every episode of APH, but it reduces the physiological reserve available to tolerate acute blood loss. It is also associated with low birth weight and preterm delivery, which may compound the fetal effects of placental disease [17]. Antenatal screening, iron replacement and timely treatment of moderate or severe anaemia therefore remain practical components of risk reduction even when the initiating placental event cannot be prevented.

Hypothyroidism and the heterogeneous category of other risks were not associated with APH type. These comparisons involved small numbers and were underpowered. The absence of statistical significance should not be interpreted as proof of no relationship, particularly because the study did not adjust for coexisting factors or disease severity.

Mode of delivery and maternal outcomes

Seventy-five percent of women underwent caesarean delivery, with emergency caesarean section accounting for half of the entire cohort. This high operative rate is expected in a referral population in which active bleeding, placenta previa, fetal compromise or maternal instability may limit the safety of labour. Caesarean delivery is often required in major placenta previa and in abruption with a viable compromised fetus, although vaginal delivery may be appropriate when fetal death has occurred, labour is advanced or maternal circumstances favour rapid vaginal birth.

Postpartum haemorrhage affected 60% of participants and was strongly associated with abruption. The magnitude is high and likely reflects the severity and referral profile of the cohort, as well as potential overlap between antepartum blood loss, uterine atony and coagulation disturbance. A systematic review has shown that placenta previa also increases the risk of postpartum haemorrhage, especially when the placenta overlies the lower uterine segment, where contractile haemostasis is less efficient [12]. In this study, however, the absolute burden was much greater in abruption.

Blood transfusion was required in 60% of women, underscoring the need for immediate access to cross-matched blood and components. The thesis did not provide transfusion quantities or type-specific transfusion counts, preventing assessment of massive transfusion or dose-response relationships. A clinically useful future dataset should record packed red cells, plasma, platelets, cryoprecipitate, estimated blood loss, lowest haemoglobin and activation of a massive-transfusion protocol.

ICU admission occurred in 30% and was strongly associated with abruption. Acute kidney injury was limited to the abruption group and reached statistical significance. Renal injury in severe haemorrhage may result from hypoperfusion, shock, microvascular thrombosis or delayed resuscitation. Disseminated intravascular coagulation and puerperal sepsis were observed only in abruption, but their low frequencies produced non-significant exact tests. This illustrates why statistical interpretation must consider both effect pattern and sample size. The recalculated p value for DIC was not significant, in contrast to the value originally printed in the thesis association table.

Fetal and neonatal outcomes

The reported fetal loss was 20%, and every loss occurred in women with abruption. This finding is consistent with the pathophysiology of placental separation, where the effective exchange area may fall abruptly and lead to fetal hypoxia or death. Placental abruption has been associated with stillbirth, preterm birth and neonatal mortality across large observational studies and systematic reviews [5,10,14]. The complete absence of fetal death among the 35 previa cases may reflect timely recognition and operative delivery, although the sample was too small to exclude uncommon adverse outcomes.

Nearly half of neonates in the abruption group required NICU admission compared with approximately one in nine in the previa group. Poor APGAR categories followed the same distribution. These results suggest acute fetal compromise in abruption, but interpretation requires caution. The thesis did not state whether the APGAR value was measured at one or five minutes, and the table used all pregnancies, including fetal deaths, as the denominator. A journal submission should verify these fields against the master chart and report APGAR only among liveborn neonates at a specified time point.

The mean birth weight was 2.16 kg, and 70% of neonates weighed 2.5 kg or less. Low birth weight may reflect prematurity, growth restriction or both. Chronic placental dysfunction can restrict growth, while active bleeding may lead to medically indicated preterm delivery. The study did not report gestational-age-specific birth-weight centiles, small-for-gestational-age status or birth weight by APH type, so the relative contribution of prematurity and fetal growth restriction cannot be separated.

Clinical implications

The results have immediate service implications. Women with antepartum bleeding should undergo rapid triage with simultaneous maternal resuscitation and fetal assessment. Digital vaginal examination must be deferred until placenta previa has been excluded. Facilities receiving obstetric referrals should maintain an escalation pathway that links the labour ward, operating theatre, anaesthesia, blood bank, laboratory, intensive care and neonatology. In women with hypertension or severe preeclampsia, new abdominal pain, uterine tenderness, altered fetal movements or abnormal fetal heart tracing should raise urgent suspicion of abruption even when external bleeding is limited.

Antenatal measures cannot eliminate APH, but they may reduce preventable morbidity. These include early registration, repeated blood-pressure assessment, prompt management of hypertensive disorders, correction of anaemia, counselling about warning symptoms and referral before haemodynamic instability develops. For placenta previa, accurate sonographic localization and planned delivery in a facility with blood products and surgical expertise can reduce emergency presentations. Current guidance also emphasizes careful evaluation for placenta accreta spectrum when previa coexists with uterine scarring [8].

Strengths and limitations

A strength of the study is its prospective collection of clinical and outcome data in a clearly defined primigravid population. The inclusion of maternal complications, fetal survival, birth weight, APGAR category and NICU admission provides a broad view of the immediate burden of APH. The use of type-specific comparisons also demonstrates the substantially different risk carried by abruption and previa.

Several limitations should be considered. The study was conducted at one tertiary referral centre, so referral and severity bias are likely, and the findings should not be used to estimate community prevalence. The sample size was modest and no a priori calculation was documented. The analysis was unadjusted, making it impossible to determine whether APH type independently predicted outcomes after accounting for gestational age, anaemia, hypertension or disease severity. Risk factors could overlap, and definitions of postpartum haemorrhage, acute kidney injury, DIC and severe preeclampsia were not fully specified in the thesis text. The source reported IUFD and stillbirth as separate categories without operational definitions. Exact study months, ethics approval identifiers, transfusion quantities, maternal mortality, duration of ICU stay and detailed neonatal morbidity were unavailable. Finally, the thesis summary stated a NICU admission rate of 30%, whereas the primary results table contained 36 admissions; the table-based value was used in this manuscript. These data points should be verified against the original master chart before publication.

CONCLUSION

Antepartum haemorrhage among primigravidae at this tertiary referral hospital was associated with considerable maternal and perinatal morbidity. Placental abruption was the predominant diagnosis and was strongly linked to hypertensive disorders, postpartum haemorrhage, intensive-care admission, acute kidney injury, fetal loss, NICU admission and poor immediate neonatal condition. The findings reinforce the need for early detection of anaemia and hypertensive disease, rapid assessment of any third-trimester bleeding, timely referral, availability of blood components and coordinated obstetric, anaesthetic, critical-care and neonatal support. Verification of the original master chart and standardized outcome definitions are recommended before journal submission.

Editorial verification note: Before submission, confirm author order, corresponding-author details, exact study months, ethics approval number/date, APGAR timing and denominator, and NICU frequency against the original institutional records and master chart.

REFERANCES

1. Cresswell JA, Ronsmans C, Calvert C, Filippi V. Prevalence of placenta praevia by world region: a systematic review and meta-analysis. Trop Med Int Health. 2013;18(6):712-724. doi:10.1111/tmi.12100.
2. Faiz AS, Ananth CV. Etiology and risk factors for placenta previa: an overview and meta-analysis of observational studies. J Matern Fetal Neonatal Med. 2003;13(3):175-190. doi:10.1080/jmf.13.3.175.190.
3. Oyelese Y, Ananth CV. Placental abruption. Obstet Gynecol. 2006;108(4):1005-1016. doi:10.1097/01.AOG.0000239439.04364.9a.
4. Tikkanen M. Placental abruption: epidemiology, risk factors and consequences. Acta Obstet Gynecol Scand. 2011;90(2):140-149. doi:10.1111/j.1600-0412.2010.01030.x.
5. Ananth CV, Berkowitz GS, Savitz DA, Lapinski RH. Placental abruption and adverse perinatal outcomes. JAMA. 1999;282(17):1646-1651. doi:10.1001/jama.282.17.1646.
6. Getahun D, Oyelese Y, Salihu HM, Ananth CV. Previous cesarean delivery and risks of placenta previa and placental abruption. Obstet Gynecol. 2006;107(4):771-778. doi:10.1097/01.AOG.0000206182.63788.80.
7. Oyelese Y, Smulian JC. Placenta previa, placenta accreta, and vasa previa. Obstet Gynecol. 2006;107(4):927-941. doi:10.1097/01.AOG.0000207559.15715.98.
8. Jauniaux E, Alfirevic Z, Bhide AG, Belfort MA, Burton GJ, Collins SL, et al. Placenta praevia and placenta accreta: diagnosis and management: Green-top Guideline No. 27a. BJOG. 2019;126(1):e1-e48. doi:10.1111/1471-0528.15306.
9. Brandt JS, Ananth CV. Placental abruption at near-term and term gestations: pathophysiology, epidemiology, diagnosis, and management. Am J Obstet Gynecol. 2023;228(5 Suppl):S1313-S1329. doi:10.1016/j.ajog.2022.06.059.
10. Downes KL, Grantz KL, Shenassa ED. Maternal, labor, delivery, and perinatal outcomes associated with placental abruption: a systematic review. Am J Perinatol. 2017;34(10):935-957. doi:10.1055/s-0037-1599149.
11. Fan D, Wu S, Liu L, Xia Q, Wang W, Guo X, et al. Prevalence of antepartum hemorrhage in women with placenta previa: a systematic review and meta-analysis. Sci Rep. 2017;7:40320. doi:10.1038/srep40320.
12. Fan D, Xia Q, Liu L, Wu S, Tian G, Wang W, et al. The incidence of postpartum hemorrhage in pregnant women with placenta previa: a systematic review and meta-analysis. PLoS One. 2017;12(1):e0170194. doi:10.1371/journal.pone.0170194.
13. Baczkowska M, Kosinska-Kaczynska K, Zgliczynska M, Brawura-Biskupski-Samaha R, Rebizant B, Ciebiera M. Epidemiology, risk factors, and perinatal outcomes of placental abruption: detailed annual data and clinical perspectives from a Polish tertiary center. Int J Environ Res Public Health. 2022;19(9):5148. doi:10.3390/ijerph19095148.
14. Ananth CV, VanderWeele TJ. Placental abruption and perinatal mortality with preterm delivery as a mediator: disentangling direct and indirect effects. Am J Epidemiol. 2011;174(1):99-108. doi:10.1093/aje/kwr045.
15. Han CS, Schatz F, Lockwood CJ. Abruption-associated prematurity. Clin Perinatol. 2011;38(3):407-421. doi:10.1016/j.clp.2011.06.001.
16. Ananth CV, Savitz DA, Williams MA. Placental abruption and its association with hypertension and prolonged rupture of membranes: a methodologic review and meta-analysis. Obstet Gynecol. 1996;88(2):309-318. doi:10.1016/0029-7844(96)00088-9.
17. Levy A, Fraser D, Katz M, Mazor M, Sheiner E. Maternal anemia during pregnancy is an independent risk factor for low birthweight and preterm delivery. Eur J Obstet Gynecol Reprod Biol. 2005;122(2):182-186. doi:10.1016/j.ejogrb.2005.02.015.
18. Nankabirwa V, Tumwine JK, Tylleskar T, Ndeezi G. Antepartum hemorrhage: etiology, risk factors and adverse outcomes in a tertiary hospital in Uganda. PLOS Glob Public Health. 2023;3(1):e0001476. doi:10.1371/journal.pgph.0001476.
19. Elsharkawy AM, Osman MM, Elzeneiny W. Risk factors for placental abruption in a tertiary care center: a case-control study. J Obstet Gynaecol India. 2022;72(4):347-353. doi:10.1007/s13224-021-01609-7.

20. Chhabra S, Sinha P. Maternal mortality with antepartum hemorrhage in a rural tertiary hospital of India. J South Asian Feder Obst Gynae. 2021;13(3):140-145. doi:10.5005/jp-journals-10006-1914.