International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Shivangi et al. Int J Reprod Contracept Obstet Gynecol. 2026 Mar;15(3):970-975
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20260556

Original Research Article

Association of disorders of coagulation profile with feto-maternal outcome in abruptio placentae

Shivangi, Harsha S. Gaikwad, Kavita Agarwal*

Department of Obstetrics and Gynecology, Vardhman Mahavir Medical College and Safdarjung Hospital, Delhi, India

Received: 03 January 2026
Accepted: 06 February 2026

***Correspondence:**
Dr. Kavita Agarwal,
E-mail: drku93@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Placental abruption is a significant cause of maternal morbidity and perinatal mortality. There is a paucity of literature on evaluation of coagulation profile in patients with placental abruption and their feto-maternal outcome. Therefore, the present study was designed to evaluate disorders of coagulation system and feto-maternal outcome in patients with placental abruption.
Methods: It was a prospective, observational study on 254 pregnant women diagnosed as abruption at >28 weeks gestation. Women were tested for prothrombin time, activated Partial Thromboplastin Time (aPTT), Internalised Normalised Ratio (INR), plasma fibrinogen, factor VIII, Von Willebrand Factor, factor IX, protein C, protein S and antiphospholipid antibodies. They were followed till discharge for feto-maternal outcome. Variables were compared using Unpaired t-test/Mann-Whitney Test/Chi-Square test/Fisher's exact test.
Results: Prolonged prothrombin time was found in 251/254 (98.82%) women, factor VIII and factor IX deficiency was found in 248/254 (97.64%) and 247/254 (97.24%) women respectively. Of 254 women, 143 (56.30%) had prolonged aPTT, 134 (52.76%) had raised INR decreased serum fibrinogen was found in 111/ 254 (43.70%) women. Cases of abruption with any one coagulation defect had adverse maternal and fetal outcome. It was interesting to find that a proportion of women who had no coagulation defect were not associated with hypertensive disorders of pregnancy, delivered vaginally, and had no NICU admission or neonatal death.
Conclusions: Most of the abruption cases had factor VIII, factor IX deficiency and prolonged prothrombin time and had adverse feto-maternal outcome. However, women without any coagulation disorders had a favourable outcome.

Keywords: Abruption, Coagulation disorders, Factor VIII deficiency, Factor IX deficiency, Feto-maternal outcome

INTRODUCTION

Placental abruption refers to separation of placenta partially or totally from its implantation site before delivery. It can be either revealed, with bleeding typically insinuating between membranes and uterus ultimately escaping through the cervix to cause external haemorrhage, or it can be concealed, with blood retained between detached placenta and uterus.[1] Incidence of placental abruption averages between 0.5 to 1 in 200 deliveries.[2] Various risk factors for abruption can be maternal age >35years, parity more than three, smoking, cocaine use, hyperhomocysteinemia, thrombophilia, anemia, uterine anomaly, pre- eclampsia, chorioamnionitis and premature rupture of membranes.[3]

Coagulation disorder as thrombophilia whether inherited or acquired leads to venous thromboembolism and bleeding. There is increased risk of vascular damage, infarcts and fibrinoid necrosis in the placenta of such women. These changes may lead to an array of adverse pregnancy outcomes as severe pre-eclampsia, placental abruption, fetal growth restriction (FGR), and stillbirth. The varied defects being factor V leidin mutation, protein C deficiency, protein S deficiency, antithrombin deficiency, increased prothrombin levels,

hyperhomocysteinemia (inhibits activation of protein C). Deficiency of protein S, activated protein C, and antithrombin either alone or in combination leads to hypercoagulable state, thromboembolism and bleeding which manifests as placental abruption during pregnancy. Acquired thrombophilia like antiphospholipid syndrome is associated with obstetric complications in about 15-20% like fetal loss after nine weeks, placental abruption, severe preeclampsia and FGR.[4] Placental abruption is a significant cause of maternal morbidity and perinatal mortality. Maternal adverse outcomes include haemorrhage, need for blood transfusions, peripartum hysterectomy, disseminated intravascular coagulopathy (DIC), renal failure and death. Fetal adverse outcomes include FGR, low birthweight, preterm delivery, asphyxia, stillbirth and perinatal death.[5]

There is a paucity of Indian literature on evaluation of coagulation profile in patients with placental abruption and their feto-maternal outcome. Therefore, the present study ained to evaluate disorders of coagulation system and feto-maternal outcome in patients with placental abruption.

METHODS

This study was a prospective, observational study conducted in the department of Obstetrics and Gynaecology, done over a period of 18 months from August 2018 to February 2020. Institutional ethics committee approval was obtained before starting the study.

A total of 254 pregnant women diagnosed as abruptio placentae either clinically or sonographically at >28 weeks period of gestation or after delivery were included in the study. A written and informed consent was taken from all the enrolled women in a language well understood by them. Women with diagnosed placenta previa, premature rupture of membranes, multiple pregnancy, history of thromboembolism, uterine leiomyomas, genital tract lesions, medical co-morbid condition or on any anti coagulants were excluded from the study.

A detailed history including demographic profile, menstrual history, obstetric history, history of uptake of any anticoagulant or coagulation defect was taken. A thorough general, systemic and obstetric examination was performed to look bleeding per vaginum, blood-stained liquor, uterine tenderness and fetal distress**.**

Blood group, Rh typing, complete blood count, liver function test, renal function test, blood sugar, thyroid function test and investigations to rule out coagulation defect were performed. For coagulation defect, peripheral blood samples of patients were collected in 3.2% sodium citrate vacutainers for Prothrombin time (PT), Activated partial thromboplastin time (APTT), Internalised Normalised Ratio (INR), plasma fibrinogen levels, factor VIII, Von Willebrand Factor (VWF), factor IX, protein C, protein S and Antiphospholipid Antibodies (APLA) including anti cardiolipin antibodies, beta2 glycoprotein levels and Lupus anticoagulant antibodies. All these coagulation investigations were performed on compact ceveron Alpha Automated Coagulation Analyzer (Compact bio-sciences Ltd), based on clotting, chromogenic and turbidimetric analysis. Normal values of these tests of coagulation are mentioned in Table 1. The patients were followed till the discharge of both mother and baby. The feto-maternal outcome was recorded in a proforma.

Table 1: Normal values for various factors.

Parameters	Normal values in third trimester
PT (sec)	9.6-12.9
INR	0.80-1.09
APTT (sec)	22.6-35.0
Fibrinogen(mg/dl)	301-696
Protein C functional (%)	67-135
Protein S, total (%)	33-101
Factor VIII (%)	143-353
Factor IX (%)	164-235
Von Willebrand factor antigen (%)	84-422

The women were classified into having grade 0, grade 1, grade 2 and grade 3 placental abruption. Grade 0 as asymptomatic, a small retroplacental clot detected on a routine sonography scan or at the time of delivery. Grade 1: vaginal bleeding, uterine tenderness present, but no signs of maternal or fetal distress. Grade 2: vaginal bleeding, uterine contractions, no signs of maternal shock, signs of fetal distress present. Grade 3: severe bleeding present or concealed, uterine hypertonus, maternal shock and fetal distress or intrauterine death.

The primary outcome was proportion of women with abruptio placentae having coagulation defects. The secondary outcomes were maternal and fetal. Maternal outcome in terms of mode of delivery, haemodynamic instability/shock, need for blood and/or blood products, disseminated intravascular coagulation (DIC) and maternal death. Foetal outcome was assessed as prematurity, FGR, Intra Uterine Death (IUD), neonatal intensive care (NICU) admissions and neonatal deaths.

Categorical variables were presented in number and percentage (%) and continuous variables as mean ± SD and median. Quantitative variables were compared using Unpaired t-test/Mann-Whitney Test. Qualitative variables were compared using Chi-Square test /Fisher's exact test. A p value of <0.05 was considered statistically significant. Data analysis was done using SPSS version 21.0.

RESULTS

A total of 254 pregnant women diagnosed as abruptio placentae were included in the study. Most of the women in our study were between 21-30 years of age (212/254;

83.46%) with mean age 25.4±3.7. Majority were from rural background (171/254; 67.32%), were housewives (216/254; 85.04%) and belonged to lower socioeconomic strata (170/254; 66.93%). Most of the women were primigravida (155/254; 61.02%) with mean period of gestation 36.39±3.12.

No case of protein S deficiency was found. Protein C deficiency was found in one case. APLA was found to be negative in all cases. Out of 254 women, 248 (97.64%) had factor VIII level below normal, 247 (97.24%) had Factor IX level lower than normal and 12 cases (4.72%) had VWF deficiency. The values of PT, aPTT, and INR, were prolonged in 251/254 (98.82%), 143/254 (56.30%) and 134/254 (52.76%) respectively. Serum fibrinogen was below normal range in 111/254 (43.70%) of cases (Table 2). Out of 254 women, 50 (19.69%) had grade 0 abruption, 96 (37.80%) had grade 1 abruption, 80 (31.50%) had grade 2 abruption and 28 (11.02%) had grade 3 abruption (Table 3).

Table 2: Distribution of PT/APTT/INR and serum fibrinogen of study subjects.

PT/APTT/INR	Frequency	Percentage
PT		
Normal	3	1.18
Abnormal (>12.9 sec.)	251	98.82
Mean ± SD	14.51±1.25	
APTT (seconds)		
Normal	111	43.70
Abnormal (>35 seconds)	143	56.30
(35-38.1seconds)	132	51.97
(>38.1 seconds)	11	4.33
Mean ± SD	36.03±1.50	
INR		
Normal	120	47.24
Abnormal (>1.09)	134	52.76
Mean ± SD	1.14±0.12	
Serum fibrinogen		
Normal	143	56.30
Abnormal (<301mg/dL)	111	43.70
Mean ± SD	309.86±46.63	

Hypertensive disorder of pregnancy like gestational hypertension, preeclampsia was seen in 50 women out of 254 (19.69%) women with abruption. All these 50 women (100%) had prolonged PT and factor IX deficiency, 49/50 (98.0%) also had factor VIII deficiency. Of these 50 women, prolonged INR, prolonged aPTT and decreased serum fibrinogen levels were also seen in 27 women (54.0%), 26 women (52.0%) and 16 women (32.0%) respectively.

In our study, at the time of admission, 224 (88.19%) women were anemic and 154 (60.63%) women had thrombocytopenia. Postpartum hemorrhage was seen in 57 (22.44%) women out of a total of 254 cases in the study. Hundred women (39.37%) required multiple blood transfusions. Out of these hundred women, 99 had prolonged PT and 98 also had factor VIII and IX deficiency along with prolonged PT. Prolonged aPTT, prolonged INR, and low serum fibrinogen levels were also seen in 56 women, 52 women and 37 women respectively.

A total of 82 women had cesarean section. All women requiring cesarean section had prolonged PT, 81 women also had factor VIII and IX deficiency. Prolonged INR, prolonged aPTT and low serum fibrinogen levels were also seen in 48 women, 46 women and 32 women respectively requiring cesarean section.

Out of 254 cases, 163 (64.17%) had normal fetal heart rate, 82 (32.28%) had fetal bradycardia, and 9 (3.54%) had absent fetal cardiac activity at the time of admission. A total of 104 women (40.94%) had SGA babies, 70 women (27.65%) had NICU admissions, 17 women (6.69%) had stillbirths, 7 women (2.76%) had neonatal deaths. Almost all women with adverse fetal outcome had prolonged PT and decreased levels of factors VIII and factor IX. Nearly 50% of these women also had prolonged aPTT, prolonged INR and low levels of serum fibrinogen along with prolonged PT and factor VIII and IX deficiency. Table 4 and 5 shows the association of feto-maternal outcome with Factor VIII, factor IX, prolonged PT, INR, aPTT and decreased serum fibrinogen levels.

The study found three abruption cases with normal coagulation profile and no coagulation defect. These women didn't have any hypertensive disorder. Also, they delivered vaginally, had no NICU admission or neonatal death.

Table 3: Grades of abruption and coagulation defects.

Grade of abruption	Factor VIII deficiency (N=248) (%)	Factor IX deficiency (N=247) (%)	VWF deficiency (N=12) (%)	Abnormal PT (N=251) (%)	Abnormal INR (N=134) (%)	Abnormal aPTT (N=143) (%)	Low serum fibrinogen (N= 111) (%)
0	49 (19.76)	47 (19.03)	2 (16.67)	50 (19.92)	31 (23.13)	34 (23.78)	21 (18.92)
1	92 (37.10)	94 (38.06)	7 (58.33)	95 (37.85)	44 (32.84)	53 (37.06)	49 (44.14)
2	79 (31.85)	79 (31.98)	2 (16.67)	80 (31.87)	45 (33.58)	45 (31.47)	28 (25.23)
3	28 (11.29)	27 (10.93)	1 (08.33)	26 (10.36)	14 (10.45)	11 (7.69)	13 (11.71)

Table 4: Association of feto-maternal outcome with factor VIII, factor IX and VWF.

Feto-maternal outcome	Factor VIII deficiency			Factor IX deficiency		
	Abnormal (n=248) (%)	Normal (n=6) (%)	P value	Abnormal (n=247) (%)	Normal (n=7) (%)	P value
NICU admission	69 (29.87)	1 (16.67)	0.673	69 (29.87)	1 (16.67)	0.673
Neonatal death	7 (2.82)	0 (0)	1	7 (2.83)	0 (0)	1
Intrauterine death	17 (6.85)	0 (0)	1	16 (6.48)	1 (14.29)	0.388
SGA	101 (40.73)	3 (50)	0.691	100 (40.49)	4 (57.14)	0.449
Hypertensive disorder of pregnancy	49 (19.79)	1 (16.67)	0.736	50 (20.24)	0 (0)	1
Blood transfusion	98 (39.52)	2 (33.33)	1	98 (39.68)	2 (28.57)	0.707
Cesarean section	81 (32.66)	1 (16.67)	0.667	81 (32.79)	1 (14.29)	0.434

Table 5: Association of feto-maternal outcome with PT, INR, aPTT, S. fibrinogen.

Feto-maternal outcome	PT			INR			aPTT			S. fibrinogen		
	Abnormal (n=251)	Normal (n=3)	P value	Abnormal (n=134)	Normal (n=120)	P value	Abnormal (n=143)	Normal (n=111)	P value	Abnormal (n=111)	Normal (n=143)	P value
NICU admission, N (%)	70 (29.66)	0 (0)	1	39 (30.95)	31 (27.93)	0.611	40 (29.20)	30 (30)	0.894	34 (32.69)	36 (27.07)	0.346
Neonatal death, N (%)	7 (2.79)	0 (0)	1	5 (3.73)	2 (1.67)	0.452	4 (2.80)	3 (2.70)	1	3 (2.70)	4 (2.80)	1
Intrauterine death, N (%)	15 (5.98)	2 (66.67)	0.012	8 (5.97)	9 (7.50)	0.626	6 (4.20)	11 (9.91)	0.071	7 (6.31)	10 (6.99)	0.828
SGA, N (%)	102 (40.64)	2 (66.67)	0.569	52 (38.81)	52 (43.33)	0.464	49 (34.27)	55 (49.55)	0.014	46 (41.44)	58 (40.56)	0.887
Hypertensive disorder of pregnancy, N (%)	50 (19.92)	0 (0)	1	27 (20.15)	23 (19.17)	0.972	26 (18.19)	24 (21.62)	0.774	16 (14.41)	34 (23.78)	0.276
Blood transfusion, N (%)	99 (39.44)	1 (33.33)	1	52 (38.81)	48 (40)	0.846	56 (39.16)	44 (39.64)	0.938	37 (33.33)	63 (44.06)	0.634
Caesarean section, N (%)	82 (32.67)	0 (0)	0.553	48 (35.82)	34 (28.33)	0.203	46 (32.17)	36 (32.43)	0.964	32 (28.83)	50 (34.97)	0.3

DISCUSSION

The present study was done on 254 patients with abruption. Around 70% women with coagulation defects had grade 1 and grade 2 abruption. 97-99% women had factor VIII, factor IX deficiency and prolonged prothrombin time. Prolonged aPTT, INR and low serum fibrinogen levels were seen in 45-50% cases. vWF deficiency was seen in 4.7% women. Only one patient had Protein C deficiency and none of them was found to have Protein S deficiency or APLA.

Similar to our study, Zarka Alfuveric et al didn't find Protein S deficiency and Lupus anticoagulant to be associated with abruption but found placental abruption to be associated with anticardiolipin antibodies (OR 2.8).[6] Also, Ananth et al didn't find protein S deficiency and activated protein C resistance to be associated with abruption. Although, contrary to our study, they found abruption cases to be associated with decreased protein C (<5th percentile); OR 3.2.[7] The differences in the results can be because in the present study, antithrombin III and activated protein C resistance were not studied. Maslow et al also found abruption to be associated with abnormal coagulation profile in setting of IUD.[8] They found 19 patients had coagulation score (include PT, aPTT, platelet count and fibrinogen) greater than or equal to four and in these 19 patients, 12 had abruption. Changes in coagulation profile can be attributed to DIC occurring as a result of abruption.

Hypertensive disorders of pregnancy were found in 19.68% women, postpartum hemorrhage was seen in 22.44%, 39.37% required multiple blood transfusions and 32.28% had delivery by cesarean section. Nearly all women with adverse maternal outcome were found to have prolonged PT and factor VIII and IX deficiency. More than 50% of these women also had prolonged aPTT and INR. Also, 30-40% women had low levels of serum fibrinogen along with prolonged PT, aPTT, INR and Factor VIII and IX deficiency.

Mikuscheva et al in 2021 reported three cases of severe abruption as a first symptom of precelampsia.[9] Study by Heilmann et al also found thrombophilia in setting of preeclampsia patients.[10] Activated protein C resistance was found in 21.3% patients, protein S deficiency in 6.5% patients, increased lupus anticoagulant in 45.9%, anticardioloipin antibodies in 40.9% and factor V Leidin in 19.6% preeclampsia patients. Retrospective study by Wang et on 61 patients with placental abruption found that predelivery fibrinogen can predict adverse maternal and fetal outcomes.[11] They found 71.5% patients with serum fibrinogen levels <155 mg/dl to have moderate haemorrhage. Recent study found incidence of PPH to be 44% in women with Von Willebrands disease.[12]

The present study found fetal heat rate less than 110 beats per minute in 32.28% cases, NICU admissions in 27.65% women, 2.76% had neonatal deaths, 6.69% had still births and 40.94% had SGA babies. Nearly all women with adverse fetal outcome had prolonged PT and factor VIII and IX deficiency. Around 50% of these women had prolonged aPTT, INR and low levels of serum fibrinogen. Study by Wang et al on case of abruption also found umbilical pH less than seven in 77.1% women with fibrinogen levels ≤ 250mg/dl.[11] Monari et al found a statistically significant association of g20210A prothrombin mutation (factor II mutation) in mothers with still births compared to controls (p=0.0.1).[13] Also, Ananth et al found extent of placental separation had a profound effect on stillbirth (aRR for 75% separation, 31.5;95%CI).[1] Kinzler at al studied 135 women with placental abruption and carried out their thrombophilia screen.[14] They found that woman with at least one diagnosed thrombophilia had higher rates of meconium staining of membranes, old placental infarcts and decidual necrosis compared with those cases of abruption with no thrombophilia. The present study also found abruption cases with no coagulation defect didn't had hypertension, no NICU admission or neonatal death.

The strength of our study is that it is one of the studies in which thrombophilia and adverse feto-maternal outcome have been studied in patients with abruption. Also, we have additionally evaluated factor VIII, factor IX, VWF and APLA. The weaknesses of our study are that it is not a case control study, small sample size and patients were limited to one hospital only. Also, antithrombin III, activated protein C resistance, chromosomal analysis like factor V Leidin mutation, prothrombin mutation G20210A were not done.

CONCLUSION

To conclude, most of the women with abruption had factor VIII deficiency, factor IX deficiency and prolonged prothrombin time. Around 50% women also had prolonged aPTT, INR and decreased serum fibrinogen. Cases of abruption with any one coagulation defect had adverse maternal and fetal outcome. Whereas women with no coagulation defect didn't have hypertensive disorder, delivered vaginally, had no NICU admission or neonatal death.

Recommendations

Hence, we recommend that Factor VIII, Factor IX, PT, aPTT, INR and serum fibrinogen should be assessed routinely in cases with abruption. Women with coagulation defect should have transfusion of blood and blood products, maternal vitals charting and continuous fetal heart rate monitoring. Early referral of abruption cases to a tertiary care hospital with facilities of testing of coagulation defects, blood transfusion and continuous fetal heart rate monitoring can improve their feto-maternal outcome.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee (IEC/VMMC/SJH/Thesis/Oct/2018-204)

REFERENCES

1. Ananth CV, Lavery JA, Vintzileos AM, Skupski DW, Varner M, Saade G, et al. Severe placental abruption: Clinical definition and associations with maternal complications. Am J Obstet Gynecol. 2016;214(2):272.e1-272 e9.
2. Tikkanen M, Nuutila M, Hiilesmaa V, Paavonen J, Ylikorkala O. Clinical presentation and risk factors of placental abruption. Acta Obstet Gynecol Scand. 2006;85(6):700-5.
3. Ananth CV, Wilcox AJ. Placental abruption and perinatal mortality in the United States. Am J Epidemiol. 2001;153(4):332-7.
4. Bodelon C, Bernabe-Ortiz A, Schiff MA, Reed SD. Factors associated with peripartum hysterectomy. Obstet Gynecol. 2009;114(1):115-23.
5. Brosens I, Pijnenborg R, Vercruysse L, Romero R. The great obstetrical syndromes are associated with disorders of deep placentation. Am J Obstet Gynecol. 2011;204(3):193-201.
6. Alfirevic Z, Roberts D, Martlew V. How strong is the association between maternal thrombophilia and adverse pregnancy outcome? A systematic review. Eur J Obstet Gynecol Reprod Biol. 2002;101(1):6-14.

7. Ananth CV, Nath CA, Philipp C. The normal anticoagulant system and risk of placental abruption: Protein C, Protein S and resistance to activated protein C. J Matern Fetal Neonatal Med. 2010;23(12):1377-83.
8. Maslow AD, Breen TW, Sarna MC, Soni AK, Watkins J, Oriol NE. Prevalance of coagulation abnormalities associated with intrauterine fetal death. Can J Anaesth. 1996;43(12):1237-43.
9. Mikuscheva A, Strassding F, Mackenzie E. Three cases of severe placental abruption as a first symptom of preeclampsia. Case Rep Obstet Gynecol. 2021;2021:3863607.
10. Heilmann L, von Tempelhoff GF, Pollow K. ProC Global assay in the evaluation of women with history of severe preeclampsia or HELLP syndrome. Clin Appl Thromb Hemost. 2002;8(4):319-24.
11. Wang L, Matsunaga S, Mikami Y, Takai Y, Terui K, Seki H, et al. Pre delivery fibrinogen predicts adverse maternal or neonatal outcomes in patients with placental abruption. J Obstet Gynaecol Res. 2016;42(7):796-802.
12. Govorov I, Löfgren S, Chaireti R, Holmström M, Bremme K, Mints M. Postpartum hemorrhage in women with von Willebrand disease- a retrospective observational study. PLoS One. 2017;12(2):e0172185.
13. Monari F, Alberico S, Avagliano L, Cetin I, Cozzolino S, Gargano G, et al. Relation between maternal thrombophilia and stillbirth according to causes/associated conditions of death. Early Hum Dev. 2012;88(4):251-4.
14. Kinzler WL, Prasad V, Ananth CV, New Jersey-Placental Abruption Study Investigators. The effect of maternal thrombophilia on placental abruption: Histologic correlates. J Matern Fetal Neonatal Med. 2009;22(3):243-8.

Cite this article as: Shivangi, Gaikwad HS, Agarwal K. Association of disorders of coagulation profile with feto-maternal outcome in abruptio placentae. Int J Reprod Contracept Obstet Gynecol 2026;15:970-5.