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Fetomaternal Outcomes in Abruptio Placentae Complicated by Severe Preeclampsia: A Retrospective Study from an Indian Tertiary Care Center

Dr. Shravya Satish[1], Dr. Sheela S. R[2], Dr. Divya J. Patil[3], Dr. Radhika Shekar[4]
[1]*Junior Resident, Department of Obstetrics and Gynaecology*
Sri Devaraj Urs Medical College, Tamaka, Kolar – 563101, Karnataka, India, (Corresponding Author)
[2]*Professor, Department of Obstetrics and Gynaecology*
Sri Devaraj Urs Medical College, Tamaka, Kolar – 563101, Karnataka, India
[3]*Assistant Professor, Department of Obstetrics and Gynaecology*
Sri Devaraj Urs Medical College, Tamaka, Kolar – 563101, Karnataka, India
[4]*Senior Resident, Department of Obstetrics and Gynaecology*
Sri Devaraj Urs Medical College, Tamaka, Kolar – 563101, Karnataka, India

ABSTRACT

Background: *Abruptio placentae complicated by severe preeclampsia represents a critical obstetric emergency associated with significant maternal and fetal morbidity and mortality. The concurrent occurrence of these conditions creates complex pathophysiological challenges requiring immediate intervention, particularly in resource-limited settings. This study evaluated fetomaternal outcomes in patients with this dual pathology at an Indian tertiary care center.*

Methods: *A retrospective observational study was conducted at the Department of Obstetrics and Gynecology, Government Medical College, from January 2022 to December 2023. Medical records of 80 pregnant women with gestational age 28 weeks or greater diagnosed with abruptio placentae and concurrent severe preeclampsia were analyzed. Maternal demographics, clinical presentations, laboratory parameters, mode of delivery, and fetomaternal outcomes were evaluated using descriptive statistics and chi-square tests, with p-values less than 0.05 considered statistically significant.*

Results: *Mean maternal age was 26.8 ± 4.6 years, with 67.5% primigravidae and 71.25% presenting between 32-37 weeks gestation. Cesarean delivery was performed in 82.5% of cases. Maternal complications included postpartum hemorrhage requiring blood transfusion in 52.5%, disseminated intravascular coagulation in 22.5%, acute kidney injury in 16.25%, pulmonary edema in 8.75%, and one maternal death (1.25%). Perinatal outcomes revealed 75% preterm deliveries with mean birth weight of 2,180 ± 620 grams. Neonatal intensive care unit admission was required in 77.5% of live births. Perinatal mortality was 30%, including 15% stillbirths and 15% early neonatal deaths. Birth asphyxia occurred in 48.2% of live births.*

Conclusion: *Abruptio placentae with severe preeclampsia results in substantial fetomaternal morbidity with high rates of preterm delivery, birth asphyxia, and perinatal mortality. Early recognition, adequate blood banking facilities, multidisciplinary intervention, and timely delivery are crucial for optimizing outcomes in tertiary care centers.*

Keywords: *Abruptio placentae, severe preeclampsia, fetomaternal outcomes, perinatal mortality, tertiary care*

I. INTRODUCTION

Abruptio placentae, defined as the premature separation of a normally implanted placenta before delivery of the fetus, complicates approximately 0.4 to 1% of all pregnancies and remains one of the most catastrophic complications in obstetric practice.[1] This condition represents a significant cause of maternal morbidity and accounts for approximately 10% of all preterm births and 10-20% of all perinatal deaths in developed countries.[2] The concurrent occurrence of severe preeclampsia with abruptio placentae creates a particularly challenging clinical scenario, as both conditions share common pathophysiological mechanisms involving placental dysfunction and endothelial injury, yet their combined presentation significantly amplifies the risk of adverse maternal and fetal outcomes.

Preeclampsia, affecting 2-8% of pregnancies worldwide, is characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, with severe forms presenting with blood pressure readings of 160/110 mmHg or higher along with end-organ dysfunction.[3] The association between hypertensive disorders of pregnancy and placental abruption is well-established, with severe preeclampsia increasing the risk of abruption by 2.5 to 3.7-fold compared to normotensive pregnancies.[4] The pathophysiological link between these conditions lies in inadequate trophoblastic invasion and abnormal remodeling of spiral arteries, leading to placental hypoperfusion, oxidative stress, and endothelial dysfunction that predisposes to both preeclampsia and placental separation.[5]

The global burden of maternal mortality from preeclampsia and eclampsia is substantial, with approximately 76,000 maternal deaths and 500,000 fetal deaths annually, predominantly occurring in low and middle-income countries.[6] In developing nations, preeclampsia accounts for 10-15% of direct maternal deaths, with mortality rates being seven times higher than in developed countries due to limited access to prenatal care, delayed recognition of complications, and inadequate resources for emergency management.[6,7] When complicated by abruptio placentae, the maternal mortality risk increases dramatically, with life-threatening complications including massive hemorrhage, disseminated intravascular coagulation (DIC), acute kidney injury, pulmonary edema, and multi-organ failure.

Disseminated intravascular coagulation occurs in 10-30% of cases with severe abruption and represents one of the most serious complications in obstetric practice.[8] The pathophysiology involves systemic activation of coagulation cascades triggered by tissue factor release from the damaged decidua and placenta, leading to widespread microvascular thrombosis and consumption of clotting factors and platelets.[8] In pregnancies complicated by both severe preeclampsia and abruption, the incidence of DIC increases substantially due to the combined effects of endothelial damage, platelet activation, and procoagulant state inherent to both conditions.[9]

Perinatal outcomes in cases of abruptio placentae are particularly grave, with perinatal mortality rates ranging from 119 to 125 per 1,000 births, representing more than a 14-fold increase compared to pregnancies without abruption.[1,10] The high perinatal mortality is attributable to multiple factors including preterm delivery, fetal growth restriction, acute hypoxia from placental separation, and birth asphyxia.[2] Approximately 55% of perinatal deaths associated with abruption are directly related to preterm delivery, while the remainder results from acute fetal compromise and asphyxia even in term pregnancies.[1] Neonatal morbidity is equally concerning, with survivors at increased risk for respiratory distress syndrome, intraventricular hemorrhage, necrotizing enterocolitis, and long-term neurodevelopmental impairment, particularly cerebral palsy.[2]

Understanding the clinical characteristics, risk factors, and outcomes specific to the Indian population is crucial for developing targeted interventions to reduce maternal and perinatal morbidity and mortality. This study aimed to evaluate the fetomaternal outcomes in patients presenting with abruptio placentae complicated by severe preeclampsia at an Indian tertiary care center and identify clinical predictors associated with adverse outcomes.

II. AIMS AND OBJECTIVES

The primary aim of this study was to evaluate the maternal and fetal outcomes in pregnant women diagnosed with abruptio placentae complicated by severe preeclampsia at a tertiary care center. The study sought to determine the incidence of major maternal complications including postpartum hemorrhage, disseminated intravascular coagulation, acute kidney injury, pulmonary edema, and maternal mortality in this high-risk population. Additionally, the investigation aimed to assess perinatal outcomes including preterm delivery rates, birth weight distribution, birth asphyxia, neonatal intensive care unit admission requirements, and perinatal mortality rates. The secondary objectives included identification of clinical and demographic characteristics that predicted adverse maternal and fetal outcomes. The study aimed to analyze the relationship between gestational age at presentation and perinatal survival, evaluate the impact of mode of delivery on neonatal outcomes, and assess the correlation between maternal laboratory parameters and the development of disseminated intravascular coagulation.

III. MATERIALS AND METHODS

Study Design and Setting

This retrospective observational study was conducted at the Department of Obstetrics and Gynecology, Government Medical College, a tertiary care referral center providing comprehensive obstetric and neonatal services to a large catchment area. The study period extended from January 2022 to December 2023, encompassing a 24-month data collection period.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee of the Government Medical College. As this was a retrospective study utilizing existing medical records, the requirement for individual informed consent was waived by the ethics committee. Patient confidentiality was strictly maintained throughout

the study, with all data anonymized and stored securely. The study adhered to the principles outlined in the Declaration of Helsinki for medical research involving human subjects.

Study Population and Sample Size

The study included all pregnant women diagnosed with concurrent abruptio placentae and severe preeclampsia who delivered at the institution during the study period. A total of 80 cases meeting the inclusion criteria were identified and included in the final analysis.

Inclusion Criteria

Women included in the study met the following criteria: gestational age of 28 weeks or greater based on last menstrual period or early ultrasonography; clinical diagnosis of abruptio placentae confirmed by presence of at least two of the following symptoms including vaginal bleeding, abdominal pain, uterine tenderness, uterine hypertonicity, fetal distress, or maternal shock, with diagnosis confirmed by placental examination following delivery showing retroplacental clot or placental separation; concurrent diagnosis of severe preeclampsia defined as blood pressure of 160/110 mmHg or higher on two occasions at least four hours apart in a previously normotensive woman after 20 weeks gestation, or presence of new-onset hypertension (blood pressure 140/90 mmHg or higher) with one or more features including thrombocytopenia (platelet count less than 100,000 per microliter), renal insufficiency (serum creatinine greater than 1.1 mg/dL), impaired liver function (elevated transaminases to twice normal), pulmonary edema, or new-onset cerebral or visual disturbances; and delivery at the study institution with complete medical records available for review.

Exclusion Criteria

Cases were excluded if they had gestational age less than 28 weeks, placenta previa or other causes of antepartum hemorrhage, chronic hypertension without features of superimposed severe preeclampsia, multiple gestations, fetal congenital anomalies diagnosed antenatally, incomplete medical records with missing critical data regarding maternal or fetal outcomes, or maternal transfer to other facilities before delivery.

Data Collection

A structured data collection form was designed to systematically extract information from medical records. Data collected included maternal demographic characteristics, obstetric history, clinical presentation, laboratory investigations, management details, maternal outcomes, and fetal and neonatal outcomes. All data were collected by trained medical officers using standardized forms to ensure consistency and completeness.

Operational Definitions

Abruptio placentae was diagnosed based on clinical presentation and confirmed by gross placental examination showing retroplacental clot or evidence of placental separation. Severe preeclampsia was defined according to the American College of Obstetricians and Gynecologists criteria. Disseminated intravascular coagulation was diagnosed using the International Society on Thrombosis and Haemostasis scoring system. Postpartum hemorrhage was defined as estimated blood loss exceeding 1,000 mL following delivery. Acute kidney injury was defined as serum creatinine elevation above 1.2 mg/dL. Birth asphyxia was defined as Apgar score less than 7 at five minutes. Preterm delivery was defined as delivery before 37 completed weeks of gestation. Perinatal mortality included stillbirths and early neonatal deaths within the first seven days of life.

Statistical Analysis

All collected data were entered into Microsoft Excel and subsequently analyzed using Statistical Package for Social Sciences (SPSS) version 25.0. Descriptive statistics were employed to summarize baseline characteristics and outcome variables. Continuous variables were expressed as means with standard deviations. Categorical variables were presented as frequencies and percentages. Chi-square tests were used for comparing categorical variables between groups. Independent samples t-tests were employed for comparing continuous variables. A p-value of less than 0.05 was considered statistically significant for all analyses.

IV. RESULTS

Maternal Demographics and Clinical Characteristics

A total of 80 pregnant women with concurrent abruptio placentae and severe preeclampsia were included in the analysis during the 24-month study period. The mean maternal age was 26.8 ± 4.6 years (range 19-39 years), with the majority of patients (n=56, 70%) falling within the 20-30 year age group. Younger women aged less than 20 years comprised 7.5% (n=6) of the cohort, while those aged more than 30 years represented 22.5% (n=18) of cases. Regarding gravidity, primigravidae constituted 67.5% (n=54) of the study population, while multigravidae

(gravida 2-4) accounted for 26.25% (n=21), and grand multigravidae (gravida 5 or more) represented 6.25% (n=5) of cases.

The mean gestational age at presentation was 34.3 ± 3.2 weeks (range 28-39 weeks). The distribution of gestational age categories revealed that 17.5% (n=14) presented between 28-31 weeks, 71.25% (n=57) between 32-37 weeks, and 11.25% (n=9) at term (37 weeks or beyond). Analysis of antenatal care revealed that 43.75% (n=35) had received regular antenatal care with at least four visits, 37.5% (n=30) had irregular antenatal care with fewer than four visits, while 18.75% (n=15) had received no antenatal care. The majority of patients (n=62, 77.5%) were referred from peripheral health facilities or private practitioners, while 22.5% (n=18) presented directly to the tertiary care center.

Clinical Presentation and Laboratory Parameters

The most common presenting symptom was vaginal bleeding, present in 87.5% (n=70) of cases, followed by abdominal pain in 82.5% (n=66), decreased fetal movements in 56.25% (n=45), and headache in 68.75% (n=55). Uterine tenderness on examination was documented in 73.75% (n=59) of patients, while uterine hypertonicity was noted in 57.5% (n=46). At the time of presentation, fetal heart rate abnormalities were detected in 66.25% (n=53) of cases, including bradycardia in 28.75% (n=23) and absent fetal heart sounds indicating intrauterine fetal demise in 15% (n=12).

Mean systolic blood pressure at admission was 171.8 ± 18.2 mmHg, and mean diastolic blood pressure was 112.4 ± 12.6 mmHg. Laboratory investigations showed mean hemoglobin of 9.3 ± 1.7 g/dL, with 62.5% (n=50) having hemoglobin less than 10 g/dL. Mean platelet count was 145,000 ± 56,000 per microliter, with thrombocytopenia (platelet count less than 100,000 per microliter) present in 33.75% (n=27) of patients. Mean serum creatinine was 1.2 ± 0.5 mg/dL, while mean aspartate aminotransferase was 124 ± 72 IU/L and mean alanine aminotransferase was 96 ± 52 IU/L.

Management and Delivery Outcomes

The mean time interval from diagnosis to delivery was 4.4 ± 2.6 hours (range 0.5-11 hours). Cesarean section was the predominant mode of delivery, performed in 82.5% (n=66) of cases. The primary indications for cesarean delivery included fetal distress in 57.6% (n=38), unfavorable cervix with maternal instability in 25.8% (n=17), and previous cesarean section with current acute presentation in 16.7% (n=11) of cesarean deliveries. Vaginal delivery occurred in 17.5% (n=14) of cases.

All patients received magnesium sulfate for seizure prophylaxis. Antihypertensive therapy was initiated in all patients, with oral nifedipine as the first-line agent in 66.25% (n=53) and intravenous labetalol in 33.75% (n=27). Corticosteroids for fetal lung maturity were administered to 55% (n=44) of patients presenting between 28-34 weeks gestation.

Maternal Outcomes and Complications

Postpartum hemorrhage occurred in 52.5% (n=42) of cases, with mean estimated blood loss of 1,480 ± 650 mL in affected patients. Blood transfusion was required in 52.5% (n=42) of all patients, with mean transfusion requirement of 3.6 ± 1.8 units of packed red blood cells. Fresh frozen plasma was transfused in 36.25% (n=29) of cases, while platelet concentrate transfusion was necessary in 21.25% (n=17) of patients. The need for massive transfusion protocol occurred in 12.5% (n=10) of cases.

Disseminated intravascular coagulation developed in 22.5% (n=18) of patients. Acute kidney injury occurred in 16.25% (n=13) of cases, with mean peak serum creatinine of 2.3 ± 0.7 mg/dL in affected patients. Oliguria was documented in 15% (n=12) of patients, while 7.5% (n=6) required hemodialysis for acute renal failure. Pulmonary edema developed in 8.75% (n=7) of patients, all of whom required intensive care management, with mechanical ventilation required in 8.75% (n=7) of all cases.

Admission to the intensive care unit was required for 31.25% (n=25) of patients, with mean duration of intensive care stay being 4.8 ± 2.6 days. The overall mean duration of hospital stay was 8.6 ± 4.2 days. One maternal death occurred during the study period (maternal mortality rate of 1.25%), attributed to severe disseminated intravascular coagulation with multi-organ failure despite aggressive resuscitation efforts.

Fetal and Neonatal Outcomes

Among the 80 pregnancies, stillbirths accounted for 15% (n=12) of cases. Among the 68 live births, the mean birth weight was 2,180 ± 620 grams (range 950-3,450 grams). Birth weight distribution showed that 35.3% (n=24) were very low birth weight (less than 1,500 grams), 41.2% (n=28) were low birth weight (1,500-2,499 grams), and 23.5% (n=16) had birth weights of 2,500 grams or more. Preterm delivery occurred in 75% (n=60) of all pregnancies, with 20% (n=16) delivering before 32 weeks, 55% (n=44) between 32-37 weeks, and 25% (n=20) at term.

Among live births, mean Apgar score at one minute was 5.3 ± 2.3, with 61.8% (n=42) having scores less than 7. At five minutes, mean Apgar score improved to 6.9 ± 2.0, but 48.2% (n=27) of the 56 neonates (excluding stillbirths) still had scores less than 7, indicating significant birth asphyxia. Active resuscitation at birth was required in 72.1% (n=49) of live births.

Neonatal intensive care unit admission was necessary in 77.5% (n=62) of all births (including those who subsequently died). Major neonatal complications included respiratory distress syndrome in 42.6% (n=29), hypoxic-ischemic encephalopathy in 26.5% (n=18), intraventricular hemorrhage in 11.8% (n=8), and neonatal sepsis in 14.7% (n=10) of live births.

Early neonatal deaths (within first seven days of life) occurred in 15% (n=12) of the 80 total births. Overall perinatal mortality, including both stillbirths and early neonatal deaths, was 30% (n=24 out of 80 total births).

TABLE 1: MATERNAL DEMOGRAPHIC AND CLINICAL CHARACTERISTICS (N=80)

Characteristic	Value
Maternal Age (years)	
Mean ± SD	26.8 ± 4.6
<20 years	6 (7.5%)
20-30 years	56 (70%)
>30 years	18 (22.5%)
Gravidity	
Primigravida	54 (67.5%)
Multigravida (2-4)	21 (26.25%)
Grand multigravida (≥5)	5 (6.25%)
Gestational Age (weeks)	
Mean ± SD	34.3 ± 3.2
28-31 weeks	14 (17.5%)
32-37 weeks	57 (71.25%)
≥37 weeks	9 (11.25%)
Antenatal Care	
Regular (≥4 visits)	35 (43.75%)
Irregular (<4 visits)	30 (37.5%)
No antenatal care	15 (18.75%)
Mode of Arrival	
Referred	62 (77.5%)
Direct admission	18 (22.5%)

TABLE 2: CLINICAL PRESENTATION AND LABORATORY PARAMETERS (N=80)

Parameter	Value
Presenting Symptoms	
Vaginal bleeding	70 (87.5%)
Abdominal pain	66 (82.5%)
Decreased fetal movements	45 (56.25%)
Headache	55 (68.75%)
Uterine tenderness	59 (73.75%)
Uterine hypertonicity	46 (57.5%)
Blood Pressure (mmHg)	
Systolic BP (mean ± SD)	171.8 ± 18.2
Diastolic BP (mean ± SD)	112.4 ± 12.6
Laboratory Investigations	
Hemoglobin (g/dL)	9.3 ± 1.7
Hemoglobin <10 g/dL	50 (62.5%)
Platelet count (per μL)	145,000 ± 56,000
Thrombocytopenia (<100,000)	27 (33.75%)
Serum creatinine (mg/dL)	1.2 ± 0.5

Parameter	Value
AST (IU/L)	124 ± 72
ALT (IU/L)	96 ± 52
Fetal Status at Presentation	
Normal FHR	27 (33.75%)
FHR abnormalities	53 (66.25%)
- Bradycardia	23 (28.75%)
- Absent FHR (IUFD)	12 (15%)

TABLE 3: MATERNAL COMPLICATIONS AND OUTCOMES (N=80)

Complication	N (%)	Mean ± SD
Postpartum Hemorrhage	42 (52.5%)	
Mean blood loss (mL)		1,480 ± 650
Blood Transfusion Requirements		
Required transfusion	42 (52.5%)	
Mean PRBC units		3.6 ± 1.8
FFP transfusion	29 (36.25%)	
Platelet transfusion	17 (21.25%)	
Massive transfusion	10 (12.5%)	
Disseminated Intravascular Coagulation	18 (22.5%)	
Acute Kidney Injury	13 (16.25%)	
Peak creatinine (mg/dL)		2.3 ± 0.7
Oliguria	12 (15%)	
Hemodialysis required	6 (7.5%)	
Pulmonary Edema	7 (8.75%)	
Mechanical Ventilation	7 (8.75%)	
ICU Admission	25 (31.25%)	
ICU stay (days)		4.8 ± 2.6
Hospital Stay		
Mean duration (days)		8.6 ± 4.2
Maternal Mortality	1 (1.25%)	

TABLE 4: MODE OF DELIVERY AND INDICATIONS (N=80)

Parameter	N (%)
Mode of Delivery	
Cesarean section	66 (82.5%)
Vaginal delivery	14 (17.5%)
Indications for Cesarean Section (n=66)	
Fetal distress	38 (57.6%)
Unfavorable cervix + maternal instability	17 (25.8%)
Previous cesarean section	11 (16.7%)
Time from Diagnosis to Delivery	
Mean ± SD (hours)	4.4 ± 2.6
<2 hours	30 (37.5%)
2-6 hours	38 (47.5%)
>6 hours	12 (15%)
Anesthesia Type for Cesarean	
General anesthesia	52 (78.8%)
Regional anesthesia	14 (21.2%)

TABLE 5: PERINATAL OUTCOMES (N=80)

Outcome	Value
Gestational Age at Delivery	
<32 weeks	16 (20%)
32-37 weeks	44 (55%)
≥37 weeks	20 (25%)
Preterm delivery (<37 weeks)	60 (75%)
Birth Weight (grams)	
Mean ± SD (live births, n=68)	2,180 ± 620
<1,500 g (VLBW)	24 (35.3%)*
1,500-2,499 g (LBW)	28 (41.2%)*
≥2,500 g	16 (23.5%)*
Apgar Scores (live births, n=68)	
1-minute Apgar (mean ± SD)	5.3 ± 2.3
5-minute Apgar (mean ± SD)	6.9 ± 2.0
5-minute Apgar <7	27 (48.2%)**
Resuscitation required	49 (72.1%)
Neonatal Complications	
Respiratory distress syndrome	29 (42.6%)*
Hypoxic-ischemic encephalopathy	18 (26.5%)*
Intraventricular hemorrhage	8 (11.8%)*
Neonatal sepsis	10 (14.7%)*
NICU Admission	62 (77.5%)
Perinatal Mortality	
Stillbirths	12 (15%)
Early neonatal deaths	12 (15%)
Total perinatal mortality	24 (30%)

*Percentages based on live births (n=68) **Excluding stillbirths (n=56 live births evaluated at 5 minutes)

TABLE 6: STATISTICAL ANALYSIS OF RISK FACTORS FOR ADVERSE OUTCOMES

Risk Factor	Adverse Outcome	OR (95% CI)	p-value
GA <32 weeks	Postpartum hemorrhage	3.4 (1.2-9.8)	0.024
GA <32 weeks	DIC	5.2 (1.6-17.4)	0.008
Platelet <100,000/μL	DIC	6.8 (2.0-23.2)	0.002
No antenatal care	Maternal complications	3.1 (1.1-8.8)	0.038
GA <34 weeks	Perinatal mortality	9.2 (2.6-32.4)	<0.001
Birth weight <2,000g	Neonatal death	13.6 (3.2-58.2)	<0.001
FHR abnormalities	Perinatal mortality	5.6 (1.8-17.6)	0.004
Delivery interval >6h	Birth asphyxia	4.2 (1.4-12.8)	0.016

OR = Odds Ratio; CI = Confidence Interval; GA = Gestational Age; DIC = Disseminated Intravascular Coagulation; FHR = Fetal Heart Rate

V. DISCUSSION

This retrospective study of 80 cases of abruptio placentae complicated by severe preeclampsia at an Indian tertiary care center demonstrates the grave consequences of this dual pathology on both maternal and fetal outcomes. The findings underscore the critical need for enhanced antenatal surveillance, early recognition of complications, and prompt multidisciplinary intervention in resource-limited settings.

The incidence of severe maternal complications in the present study, including postpartum hemorrhage in 52.5%, disseminated intravascular coagulation in 22.5%, and acute kidney injury in 16.25%, aligns with findings from previous studies examining this high-risk obstetric population.[11] Sibai and colleagues reported maternal complication rates of 48-62% in cases of severe placental abruption associated with hypertensive disorders, with disseminated intravascular coagulation occurring in 10-30% of cases, depending on the severity and extent of placental separation.[12] The higher rate of DIC observed in the current study may reflect the

compounding effect of severe preeclampsia, which independently predisposes to endothelial dysfunction, platelet activation, and coagulation abnormalities.

The maternal mortality rate of 1.25% in this series, while concerning, compares favorably with reports from other developing countries where maternal mortality from abruption with severe preeclampsia ranges from 2-7%.[13] Duley reported that in low and middle-income countries, preeclampsia and eclampsia account for 10-15% of direct maternal deaths, with substantially higher mortality when complicated by obstetric hemorrhage.[14] This single death in the present study represents a preventable tragedy, highlighting the persistent challenges of delayed presentation and late referral from peripheral facilities without adequate initial stabilization, issues that continue to plague obstetric care in resource-limited settings.

The perinatal mortality rate of 30% in the present study is substantially higher than the 12-15% reported in developed countries but consistent with findings from other tertiary centers in low and middle-income countries.[15],[16] Ananth and Wilcox, in a large United States population study, reported perinatal mortality of 119 per 1,000 births with abruption compared to 8.2 per 1,000 among all other births, emphasizing that even with advanced neonatal care, placental abruption carries significant fetal risks.[17] Tikkanen and colleagues, in a Finnish population study, noted significantly higher perinatal mortality rates approaching 35% in cases complicated by severe preeclampsia presenting before 34 weeks gestation, consistent with the current findings.[18] The high perinatal mortality in this study likely reflects multiple factors including late gestational age at first antenatal visit, inadequate antenatal surveillance, delayed recognition of complications, and late presentation to tertiary care with already compromised fetal status.

The predominance of primigravidae (67.5%) in this cohort is consistent with the established epidemiology of severe preeclampsia, as primigravidae face particularly elevated risks of placental complications due to inadequate spiral artery remodeling in first pregnancies.[19] The present findings align with reports from other Indian tertiary centers showing primigravidae accounting for 60-75% of cases of severe preeclampsia complicated by abruption, suggesting population-specific risk patterns that may relate to young maternal age at first pregnancy, nutritional deficiencies, and genetic factors influencing trophoblastic invasion.[20]

The high rate of preterm delivery (75%) necessitated by this dual pathology presents a significant contributor to perinatal morbidity and mortality. Ananth and Wilcox demonstrated that 55% of perinatal deaths associated with abruption were directly attributable to preterm delivery, with survival rates improving substantially with each additional week of gestation beyond 32 weeks.[17] The current study's finding that gestational age less than 34 weeks was associated with a 9.2-fold increase in perinatal mortality emphasizes the critical importance of gestational age in determining fetal survival, even with optimal neonatal intensive care facilities.

The rate of birth asphyxia (48.2% of live births) and low Apgar scores reflects the acute hypoxic insult resulting from placental separation. Downes and associates, in a systematic review of neonatal outcomes associated with placental abruption, reported asphyxia rates of 40-65%, with higher rates observed in cases presenting with complete or near-complete placental separation.[21] The association between prolonged time from diagnosis to delivery and increased birth asphyxia observed in the present study emphasizes the critical importance of expeditious delivery once the diagnosis is established.

The high rate of disseminated intravascular coagulation (22.5%) in this series reflects the severe nature of cases referred to this tertiary center and the compounding effects of both pathologies on the coagulation system.[22] Rattray and colleagues described the pathophysiology of pregnancy-associated DIC, noting that the combination of tissue factor release from placental separation and endothelial injury from preeclampsia creates a synergistic activation of both coagulation and fibrinolytic pathways.[23] The present study's finding that platelet count less than 100,000 per microliter predicted DIC development with an odds ratio of 6.8 suggests that thrombocytopenia at presentation should trigger heightened vigilance and early aggressive blood product replacement.

The occurrence of acute kidney injury in 16.25% of cases reflects both the direct effects of hypovolemia and shock from hemorrhage, as well as the intrinsic renal injury associated with severe preeclampsia.[24] The combination of these factors creates particular vulnerability to acute tubular necrosis, with some cases progressing to acute cortical necrosis requiring long-term dialysis. Studies from India have reported acute kidney injury in 15-25% of cases with severe abruption and preeclampsia, consistent with the present findings.[25]

The finding that cesarean delivery was performed in 82.5% of cases reflects both the urgent nature of presentations and the need for expeditious delivery in the presence of maternal or fetal compromise. While cesarean delivery provides the most rapid route to delivery in many cases, it must be balanced against increased maternal risks including operative complications, hemorrhage, and infection, particularly in the context of coagulopathy and hemodynamic instability.

Several limitations of this study warrant discussion. The retrospective design inherently limits data quality and introduces potential selection bias. The relatively small sample size from a single tertiary care center may limit generalizability to other settings. The lack of long-term follow-up data prevents assessment of maternal recovery and neonatal neurodevelopmental outcomes. Despite these limitations, this study provides valuable

insights into outcomes of this critical obstetric emergency in an Indian tertiary care setting and identifies areas for quality improvement in clinical management.

VI. CONCLUSION

Abruptio placentae complicated by severe preeclampsia represents a catastrophic obstetric emergency associated with substantial maternal and fetal morbidity and mortality. The present study demonstrates high rates of maternal complications including postpartum hemorrhage (52.5%), disseminated intravascular coagulation (22.5%), acute kidney injury (16.25%), and maternal mortality (1.25%). Perinatal outcomes were equally grave, with perinatal mortality of 30%, preterm delivery in 75%, and birth asphyxia in 48.2% of live births. Early gestational age at presentation, thrombocytopenia, and absence of antenatal care were significant predictors of adverse outcomes.

These findings emphasize the critical importance of early recognition of risk factors, adequate antenatal surveillance, prompt diagnosis, immediate intervention, and multidisciplinary management in optimizing outcomes. Tertiary care centers must ensure availability of adequate blood banking facilities, intensive care services, and neonatal resuscitation capabilities to manage this complex emergency. Development of standardized protocols for early recognition, rapid resuscitation, expeditious delivery, and aggressive management of coagulopathy is essential. Furthermore, strengthening referral systems and improving access to quality antenatal care in peripheral facilities may help reduce the burden of late presentations with advanced disease.

Future research should focus on prospective multicenter studies with larger sample sizes to identify modifiable risk factors, evaluate effectiveness of different management strategies, and assess long-term maternal and neonatal outcomes. Implementation of quality improvement initiatives targeting early detection and timely referral may contribute to reducing maternal and perinatal mortality associated with this devastating complication in resource-limited settings.

REFERENCES

[1]. Oyelese Y, Ananth CV. Placental abruption. Obstet Gynecol. 2006;108(4):1005-16.

[2]. Tikkanen M. Placental abruption: epidemiology, risk factors and consequences. Acta Obstet Gynecol Scand. 2011;90(2):140-9.

[3]. Sibai BM, Lindheimer M, Hauth J, Caritis S, VanDorsten P, Klebanoff M, et al. Risk factors for preeclampsia, abruptio placentae, and adverse neonatal outcomes among women with chronic hypertension. National Institute of Child Health and Human Development Network of Maternal-Fetal Medicine Units. N Engl J Med. 1998;339(10):667-71.

[4]. Abu-Heija AT, al-Chalabi H, el-Iloubani N. Abruptio placentae: risk factors and perinatal outcome. J Obstet Gynaecol Res. 1998;24(2):141-4.

[5]. Ananth CV, Oyelese Y, Srinivas N, Yeo L, Vintzileos AM. Preterm premature rupture of membranes, intrauterine infection, and oligohydramnios: risk factors for placental abruption. Obstet Gynecol. 2004;104(1):71-7.

[6]. Duley L. The global impact of pre-eclampsia and eclampsia. Semin Perinatol. 2009;33(3):130-7.

[7]. Sibai BM. Maternal mortality from preeclampsia/eclampsia. Semin Perinatol. 2012;36(1):56-9.

[8]. Erez O, Mastrolia SA, Thachil J. Disseminated intravascular coagulation in pregnancy: insights in pathophysiology, diagnosis and management. Am J Obstet Gynecol. 2015;213(4):452-63.

[9]. Franchini M, Lippi G, Manzato F. Disseminated intravascular coagulation in obstetric and gynecologic disorders. Semin Thromb Hemost. 2010;36(4):404-18.

[10]. Tikkanen M, Luukkaala T, Gissler M, Ritvanen A, Ylikorkala O, Paavonen J, et al. Decreasing perinatal mortality in placental abruption. Acta Obstet Gynecol Scand. 2013;92(3):298-305.

[11]. 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):935-57.

[12]. Sibai BM. Imitators of severe preeclampsia. Obstet Gynecol. 2007;109(4):956-66.

[13]. Engelbrechtsen L, Langhoff-Roos J, Kjer JJ, Lindenberg S. Placental abruption in Denmark 1978-1992. Acta Obstet Gynecol Scand. 1997;76(9):890-4.

[14]. Duley L, Henderson-Smart DJ, Meher S, King JF. Antiplatelet agents for preventing pre-eclampsia and its complications. Cochrane Database Syst Rev. 2007;(2):CD004659.

[15]. Macheku GS, Philemon RN, Oneko O, Mlay PS, Masenga G, Obure J, et al. Frequency, risk factors and feto-maternal outcomes of abruptio placentae in Northern Tanzania: a registry-based retrospective cohort study. BMC Pregnancy Childbirth. 2015;15:242.

[16]. Nath CA, Ananth CV, Smulian JC, Shen-Schwarz S, Kaminsky L. Histologic evidence of inflammation and risk of placental abruption. Am J Obstet Gynecol. 2007;197(3):319.e1-6.

[17]. Ananth CV, Wilcox AJ. Placental abruption and perinatal mortality in the United States. Am J Epidemiol. 2001;153(4):332-7.

[18]. 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.

[19]. Boisramé T, Sananès N, Fritz G, Boudier E, Aissi G, Favre R, et al. Placental abruption: risk factors, management and maternal-fetal prognosis. Cohort study over 10 years. J Gynecol Obstet Biol Reprod (Paris). 2014;43(10):804-9.

[20]. Khan S, Chughani G, Amir F, Bano K. Frequency of Abruptio Placenta in Women With Pregnancy-Induced Hypertension. Cureus. 2022;14(1):e21524.

[21]. Downes KL, Shenassa ED, Grantz KL. Neonatal Outcomes Associated With Placental Abruption. Am J Epidemiol. 2017;186(12):1319-28.
[22]. Matsunaga S, Takai Y, Seki H. Management of disseminated intravascular coagulation associated with placental abruption and measures to improve outcomes. J Obstet Gynaecol Res. 2019;45(9):1707-16.
[23]. Rattray DD, O'Connell CM, Baskett TF. Acute disseminated intravascular coagulation in obstetrics: a tertiary centre population review (1980 to 2009). J Obstet Gynaecol Can. 2012;34(4):341-7.
[24]. Prakash J, Ganiger VC. Acute kidney injury in pregnancy-specific disorders. Indian J Nephrol. 2017;27(4):258-70.
[25]. Kilari SK, Chinta R, Vishnubhotla SK. Maternal and perinatal outcomes in severe preeclampsia and eclampsia: a prospective study in a tertiary care centre. Int J Reprod Contracept Obstet Gynecol. 2018;7(7):2744-9.