



## Emergency Medical Services Management of Placental Abruption: Current Clinical Practices and Prehospital Care

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### Abstract

**Background:** Placental abruption is a rare but life-threatening obstetric emergency characterized by the premature separation of the placenta from the uterine wall before delivery. It is a major cause of antepartum hemorrhage and is associated with significant maternal and fetal morbidity and mortality, requiring rapid recognition and coordinated multidisciplinary management.

**Aim:** This review aimed to summarize the current evidence regarding the epidemiology, etiology, pathophysiology, clinical presentation, evaluation, and management of placental abruption, with particular emphasis on the role of emergency medical services and prehospital care.

**Methods:** A comprehensive narrative review of contemporary evidence-based literature was conducted to examine current knowledge related to placental abruption. The review integrates information on risk factors, disease mechanisms, diagnostic strategies, emergency assessment, prehospital stabilization, definitive hospital management, and interprofessional collaboration to provide practical guidance for healthcare professionals.

**Results:** Placental abruption remains an unpredictable obstetric emergency despite advances in prenatal care. Early recognition of maternal symptoms, rapid assessment of fetal well-being, prompt hemodynamic stabilization, and expedited transport to specialized obstetric centers are critical determinants of favorable outcomes. Diagnosis relies primarily on clinical assessment supported by laboratory investigations, fetal monitoring, and ultrasonography. Management varies according to disease severity, gestational age, and maternal-fetal status, ranging from close observation in stable patients to emergency delivery and massive transfusion in severe cases. Successful outcomes depend on coordinated multidisciplinary care involving emergency medical services, obstetricians, anesthesiologists, neonatologists, and critical care teams.

**Conclusion:** Early recognition, evidence-based emergency management, and seamless multidisciplinary collaboration remain fundamental for reducing maternal and neonatal morbidity and mortality associated with placental abruption.

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## Introduction

Placental abruption, also referred to as abruptio placentae, is a significant obstetric emergency characterized by the premature detachment of a normally implanted placenta from the uterine wall before the completion of the second stage of labor. This pathological separation disrupts the normal maternal-fetal interface, compromising placental perfusion and impairing the exchange of oxygen and essential nutrients required for fetal development. As a consequence, placental abruption represents a critical clinical condition that demands prompt recognition, comprehensive assessment, and immediate medical intervention to minimize maternal and fetal morbidity and mortality. Despite advances in obstetric care, this disorder continues to pose substantial diagnostic and therapeutic challenges due to its variable clinical presentation and the rapid progression of maternal and fetal compromise that may occur in severe cases [1][2]. Although placental abruption is considered an uncommon complication of pregnancy, its clinical significance extends far beyond its relatively low incidence because of the potentially devastating consequences associated with the condition. It is recognized as one of the principal causes of antepartum hemorrhage during the second half of pregnancy and remains a major contributor to adverse obstetric outcomes worldwide. The premature separation of the placenta initiates a cascade of pathophysiological events that may result in extensive maternal hemorrhage, hypovolemic shock, disseminated intravascular coagulation, fetal hypoxia, preterm birth, and, in severe circumstances, fetal or maternal death. The degree of placental separation largely determines the severity of clinical manifestations, with larger areas of detachment producing more profound maternal blood loss and greater impairment of fetal oxygenation [1][2].

From a clinical perspective, placental abruption constitutes a time-sensitive obstetric emergency that requires coordinated multidisciplinary management involving emergency medical services, obstetricians, anesthesiologists, neonatologists, and critical care specialists. Early identification of characteristic symptoms, including vaginal bleeding, abdominal pain, uterine tenderness, increased uterine tone, and evidence of fetal distress, is essential for facilitating rapid diagnosis and timely intervention. Immediate stabilization of maternal hemodynamic status, continuous fetal monitoring, and appropriate planning for delivery are fundamental components of effective management strategies aimed at preserving maternal health while optimizing fetal survival. Consequently, placental abruption remains one of the most critical complications encountered in emergency obstetric practice, emphasizing the importance of maintaining a high index of suspicion and implementing evidence-based management protocols to improve clinical outcomes for both mother and fetus [1][2].

## Etiology

The precise etiology of placental abruption has not been fully elucidated, and its development is considered multifactorial, involving a complex interaction of maternal, placental, fetal, and environmental influences. Although no single mechanism has been identified as the definitive cause, numerous predisposing factors have been consistently associated with an increased likelihood of premature placental separation. These risk factors are generally classified into three principal categories, including maternal health history and behavioral characteristics, conditions arising during the current pregnancy, and external physical trauma. Recognition of these contributing factors is essential for identifying women at elevated risk, implementing preventive strategies when possible, and facilitating early diagnosis in patients presenting with compatible clinical features [3][4]. Maternal characteristics and previous obstetric history constitute important determinants of placental abruption risk. Advanced maternal age, particularly among women older than 35 years, has been associated with a greater incidence of placental vascular abnormalities that may predispose to placental separation. Chronic hypertension and pregnancy-related hypertensive disorders contribute significantly by promoting vascular endothelial injury, impaired uteroplacental circulation, and pathological remodeling of maternal blood vessels supplying the placenta. Lifestyle-related factors, including cigarette smoking and cocaine use during pregnancy, further increase susceptibility by inducing vasoconstriction, endothelial dysfunction, and ischemic injury within the placental circulation. In addition, a previous history of placental abruption represents one of the strongest predictors of recurrence in subsequent pregnancies, suggesting persistent maternal vascular abnormalities or underlying predispositions that continue across gestations [3][4].

Several pregnancy-specific conditions also contribute substantially to the development of placental abruption through mechanisms that alter uterine anatomy, placental attachment, or intrauterine pressure. Multiple gestations increase uterine distention and mechanical stress on placental attachment sites, while polyhydramnios produces excessive expansion of the uterine cavity that may place abnormal tension on the uteroplacental interface. Disorders such as preeclampsia further exacerbate placental vascular insufficiency through widespread endothelial dysfunction and abnormal placental perfusion. Sudden uterine decompression, which may occur following rapid drainage of excessive amniotic fluid or the delivery of the first fetus in multiple pregnancies, creates abrupt mechanical changes that can precipitate placental separation. Likewise, the presence of a relatively short umbilical cord may generate excessive traction on the placenta during fetal movement or labor, increasing the likelihood of premature detachment

from the uterine wall [3][4]. Physical trauma remains another well-recognized precipitating factor, particularly when direct blunt force is applied to the maternal abdomen. Motor vehicle collisions, accidental falls, interpersonal violence, and other traumatic injuries may produce sufficient mechanical disruption to compromise placental attachment and damage the uteroplacental circulation. Because placental injury may occur even in the absence of obvious external maternal injuries, pregnant patients experiencing abdominal trauma require careful clinical assessment and prolonged fetal monitoring to exclude occult placental abruption. The severity of placental separation following trauma depends on the magnitude of the force, gestational age, and the extent of vascular injury sustained at the uteroplacental interface [3][4]. The underlying pathophysiological mechanism of placental abruption involves disruption of the vascular structures responsible for anchoring the placenta to the uterine wall and maintaining adequate maternal-fetal circulation. Normally, an extensive network of maternal blood vessels supplies oxygen and nutrients to the intervillous space, supporting fetal growth and development. When these vascular connections are compromised, bleeding occurs between the placenta and the decidua, resulting in the formation of a retroplacental hematoma. Progressive accumulation of blood further separates the placenta from the uterine lining, creating a self-perpetuating cycle of vascular disruption, hemorrhage, and placental detachment. This process significantly reduces uteroplacental blood flow, impairing fetal oxygenation while simultaneously increasing the risk of severe maternal hemorrhage and associated complications [3][4].

Anatomical differences between the uterus and placenta further explain the susceptibility to placental separation under conditions of excessive mechanical stress. The uterine wall consists predominantly of smooth muscle tissue that possesses considerable elasticity and the capacity to accommodate progressive enlargement throughout pregnancy. In contrast, the placenta is composed primarily of epithelial, connective, and vascular tissues that exhibit substantially less elasticity. Consequently, when the uterus undergoes sudden stretching or rapid alterations in size, the relatively rigid placental attachment cannot adapt to these changes. The resulting shearing forces disrupt the delicate vascular connections between the placenta and the uterine wall, leading to tearing of maternal blood vessels, hemorrhage, and premature placental separation. This combination of vascular compromise and mechanical stress represents the fundamental pathological basis underlying the development of placental abruption and its potentially life-threatening maternal and fetal consequences [3][4].

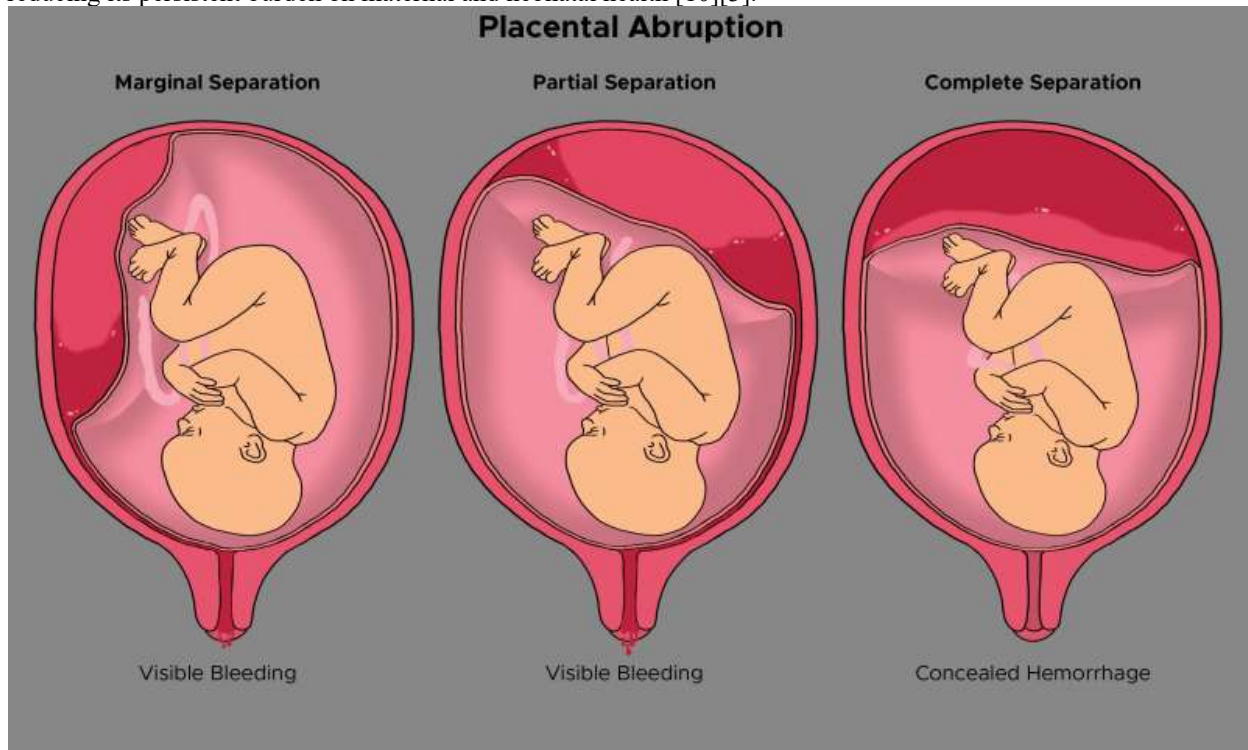
### **Epidemiology**

Placental abruption is an infrequent yet highly significant obstetric complication that continues to represent a major cause of maternal and perinatal morbidity and mortality worldwide. The condition affects approximately 0.4% to 1.0% of all pregnancies, making it relatively uncommon in comparison with other obstetric disorders. Despite its low incidence, placental abruption is regarded as a true obstetric emergency because of its rapid clinical progression and the substantial risks it poses to both maternal and fetal health. The unpredictable nature of the disorder, combined with its potential to cause catastrophic hemorrhage and fetal compromise within a short period, necessitates prompt recognition and immediate multidisciplinary intervention to optimize maternal and neonatal outcomes [5]. The majority of placental abruption cases occur before 37 weeks of gestation, making the condition an important contributor to medically indicated preterm delivery and adverse pregnancy outcomes. Premature placental separation during the late second or third trimester significantly compromises uteroplacental blood flow, resulting in impaired fetal oxygenation and nutrient delivery at a critical stage of fetal development. Consequently, placental abruption accounts for a considerable proportion of maternal complications while remaining one of the leading causes of perinatal mortality across diverse healthcare settings. The severity of maternal and fetal outcomes generally correlates with the extent of placental detachment, the volume of maternal blood loss, gestational age at presentation, and the timeliness of clinical intervention. Early diagnosis and rapid access to advanced obstetric and neonatal care remain essential determinants of survival and long-term prognosis [6][7].

Maternal complications associated with placental abruption are often severe and may rapidly become life-threatening. Extensive hemorrhage is the most common immediate consequence, frequently necessitating aggressive fluid resuscitation, blood transfusion, and emergency surgical intervention. In cases of uncontrolled bleeding, hysterectomy may become necessary to preserve maternal life, resulting in permanent loss of reproductive capacity. Furthermore, massive hemorrhage can precipitate serious hematological complications, including disseminated intravascular coagulation, which is characterized by widespread activation of the coagulation cascade followed by depletion of clotting factors and uncontrolled bleeding. Additional complications include acute kidney injury resulting from hypoperfusion, multiorgan dysfunction, and, in rare circumstances, maternal death despite advances in critical care and transfusion medicine. Severe postpartum hemorrhage may also lead to ischemic necrosis of the anterior pituitary gland, a condition known as Sheehan syndrome, which can result in permanent endocrine dysfunction requiring lifelong hormonal replacement therapy [8]. An important epidemiological characteristic of placental abruption is its limited predictability. Although numerous maternal, obstetric, and behavioral risk factors have been identified, approximately 70% of affected patients are considered to have a low-risk pregnancy before the onset of symptoms. This observation highlights the limitations of current risk stratification models and emphasizes that placental abruption may develop unexpectedly in women without recognized predisposing conditions. Consequently, healthcare professionals must maintain a high degree of clinical vigilance when evaluating pregnant patients

presenting with abdominal pain, vaginal bleeding, uterine tenderness, or fetal distress, regardless of their apparent baseline risk profile. The inability to accurately predict many cases underscores the ongoing need for improved screening methods and a deeper understanding of the underlying biological mechanisms responsible for disease development [9].

Advances in modern obstetric practice, including improved maternal monitoring, widespread availability of blood products, enhanced surgical techniques, and comprehensive neonatal intensive care, have substantially reduced the incidence of maternal mortality directly attributable to placental abruption. Nevertheless, the condition continues to contribute significantly to overall maternal mortality because of its association with severe hemorrhagic and multisystem complications. From the fetal perspective, placental abruption remains a leading cause of adverse neonatal outcomes, including preterm birth, low birth weight, perinatal asphyxia, intrauterine fetal demise, stillbirth, and neonatal death. Survivors, particularly those born prematurely or following prolonged fetal hypoxia, may also experience long-term neurodevelopmental impairment and chronic health complications [10][3]. Interestingly, epidemiological data from numerous countries indicate that the incidence of placental abruption has gradually increased despite continuous improvements in prenatal surveillance, obstetric management, and maternal healthcare services. This apparent paradox suggests that advances in clinical care alone are insufficient to prevent the disorder and reinforces the concept that placental abruption arises from a complex interaction of genetic susceptibility, maternal vascular disease, environmental exposures, and pregnancy-specific physiological changes. Continued epidemiological research is therefore essential to clarify the multifactorial pathogenesis of placental abruption, improve early identification of high-risk pregnancies, and develop more effective preventive and therapeutic strategies aimed at reducing its persistent burden on maternal and neonatal health [10][3].



**Fig. 1:** Placental Abruption.

### Pathophysiology

Placental abruption develops as a consequence of premature disruption of the vascular connections between the maternal uterine circulation and the normally implanted placenta. The pathological process begins when maternal decidual blood vessels rupture, resulting in hemorrhage within the potential space between the uterine wall and the maternal surface of the placenta. The accumulating blood forms a retroplacental hematoma that progressively enlarges, exerting mechanical pressure that further separates the placenta from its implantation site. This expanding hematoma perpetuates additional vascular disruption, leading to continued bleeding and increasing placental detachment. The resulting cycle of hemorrhage and separation compromises the structural and functional integrity of the uteroplacental interface, ultimately impairing placental perfusion and threatening both maternal and fetal survival [11][12]. The placenta serves as the essential organ responsible for maintaining fetal life throughout pregnancy by facilitating the continuous exchange of oxygen, nutrients, metabolic substrates, hormones, and waste products between the maternal and fetal circulatory systems. Efficient diffusion across the placental membrane depends on an intact maternal vascular supply and adequate uteroplacental blood flow. Once placental separation occurs, maternal blood can no longer effectively perfuse the intervillous space, leading to a rapid decline in oxygen and nutrient transfer to the fetus.

Simultaneously, fetal metabolic waste products accumulate because their elimination through the placental circulation becomes progressively impaired. The degree of physiological disruption is directly related to the proportion of placental tissue that has separated, with extensive detachment producing severe fetal hypoxia, metabolic acidosis, and, if not promptly corrected, intrauterine fetal demise or stillbirth [11][12].

The maternal physiological response to placental abruption is equally significant. Ongoing hemorrhage may be concealed behind the placenta or become clinically apparent through vaginal bleeding, although the visible blood loss often underestimates the actual volume of hemorrhage. Progressive blood loss results in intravascular volume depletion, reduced tissue perfusion, and activation of compensatory cardiovascular mechanisms, including tachycardia and peripheral vasoconstriction. In severe cases, persistent hemorrhage overwhelms these compensatory responses, leading to hypovolemic shock and multiorgan dysfunction. Furthermore, the release of thromboplastin from damaged placental tissue into the maternal circulation may trigger disseminated intravascular coagulation, resulting in widespread consumption of clotting factors and platelets, thereby increasing the risk of uncontrolled hemorrhage and life-threatening coagulopathy. This combination of massive blood loss and coagulation abnormalities substantially contributes to maternal morbidity and mortality associated with severe placental abruption [11][12]. The clinical manifestations and severity of placental abruption depend primarily on both the extent of placental separation and the anatomical location of the detachment. The separation may be partial or complete, while the hemorrhage may occur at the placental margin or centrally beneath the placenta. These anatomical variations influence the amount of visible vaginal bleeding, the degree of uterine irritation, maternal hemodynamic stability, and the extent of fetal compromise. Consequently, placental abruption demonstrates a broad clinical spectrum, ranging from asymptomatic cases identified only after delivery to catastrophic obstetric emergencies characterized by profound maternal hemorrhage and fetal death.

Class 0 placental abruption represents the mildest form of the condition and remains clinically silent throughout pregnancy and labor. In these cases, no symptoms are evident before delivery, and diagnosis is established retrospectively when a blood clot is identified on the maternal surface of the placenta following childbirth. This finding indicates that a limited placental separation had occurred without producing clinically significant maternal or fetal consequences. Class 1 placental abruption is considered mild and is generally associated with partial or marginal placental separation. Patients may experience no vaginal bleeding or only minimal bleeding accompanied by mild uterine tenderness. Maternal blood pressure and heart rate typically remain within normal physiological limits, reflecting preserved hemodynamic stability. Importantly, fetal oxygenation is usually maintained, and no evidence of fetal distress is detected during clinical assessment. Class 2 placental abruption represents a moderate degree of placental separation and is associated with more pronounced maternal and fetal compromise. Vaginal bleeding may be absent or moderate in volume because a significant proportion of hemorrhage may remain concealed behind the placenta. Patients frequently develop marked uterine tenderness accompanied by sustained tetanic uterine contractions resulting from irritation of the myometrium by accumulated blood. Maternal vital signs often demonstrate tachycardia and orthostatic hypotension, reflecting ongoing blood loss and reduced circulating blood volume. Laboratory evaluation may reveal hypofibrinogenemia due to consumption of clotting factors, while fetal monitoring commonly demonstrates evidence of fetal distress resulting from impaired placental oxygen exchange. Class 3 placental abruption constitutes the most severe clinical presentation and is typically associated with complete or central placental separation. Vaginal bleeding may range from absent to profuse despite extensive internal hemorrhage. The uterus becomes rigid, tense, and characteristically board-like on palpation because of extensive myometrial infiltration with blood. Maternal shock develops as a consequence of severe hemorrhage, while profound coagulation abnormalities, including hypofibrinogenemia and disseminated intravascular coagulation, significantly increase the risk of uncontrollable bleeding. Complete interruption of placental function frequently results in fetal death due to prolonged and severe deprivation of oxygen and nutrients. In general, Class 0 and Class 1 abruptions correspond to partial or marginal placental separation with relatively limited physiological disruption, whereas Class 2 and Class 3 abruptions reflect complete or central placental separation associated with extensive maternal hemorrhage, severe fetal compromise, and substantially poorer clinical outcomes [11][12].

### **History and Physical**

Placental abruption is one of the most important causes of vaginal bleeding during the second half of pregnancy and represents a true obstetric emergency that requires immediate clinical assessment. Because its presentation can overlap with other causes of antepartum hemorrhage, including placenta previa, vasa previa, uterine rupture, and local cervical or vaginal pathology, a comprehensive history and meticulous physical examination are fundamental components of the diagnostic process. Although the definitive diagnosis of placental abruption is established only after delivery through direct examination of the placenta, timely recognition of the condition during pregnancy depends largely on clinical findings. Therefore, a systematic evaluation is essential for guiding immediate management decisions, determining the severity of maternal and fetal compromise, and minimizing the risk of adverse outcomes for both members of the maternal-fetal dyad [12]. The clinical history should begin with a detailed review of the current pregnancy and previous obstetric experiences. Particular attention should be directed toward antenatal ultrasound findings, especially documentation of placental location, as this information assists in differentiating placental

abruption from other causes of antepartum bleeding. A previous history of placental abruption should also be carefully explored because recurrence remains one of the strongest established risk factors. Additional obstetric information, including gestational age, previous pregnancy complications, hypertensive disorders, multiple gestations, and other maternal medical conditions, contributes to a comprehensive risk assessment and assists in determining the probability of placental abruption as the underlying diagnosis.

A thorough symptom analysis is equally important. The clinician should establish the exact time of symptom onset, the progression of clinical manifestations, and the estimated amount of vaginal bleeding. Patients should be questioned regarding the presence of abdominal pain, persistent uterine tenderness, lower back pain, uterine contractions, or cramping, all of which may indicate placental separation. It is important to recognize that the apparent quantity of vaginal bleeding does not necessarily reflect the severity of the condition because a substantial proportion of hemorrhage may remain concealed behind the placenta. Consequently, severe placental abruption may occur with minimal or even absent external bleeding, making reliance on vaginal blood loss alone potentially misleading during clinical assessment [12]. Assessment of maternal history should also include careful evaluation of behavioral and environmental risk factors. Cigarette smoking, cocaine use, and other forms of substance abuse should be addressed through sensitive, nonjudgmental questioning because these exposures are well-recognized contributors to placental vascular injury. Likewise, the clinician should inquire about recent abdominal trauma, including motor vehicle collisions, accidental falls, or direct blunt injury. Screening for intimate partner violence is particularly important, as some pregnant individuals may be reluctant to disclose episodes of physical abuse because of fear, emotional distress, or concerns regarding personal safety. Establishing a supportive and confidential environment encourages accurate disclosure and facilitates appropriate medical and psychosocial intervention when necessary.

The physical examination focuses primarily on assessing maternal hemodynamic stability while simultaneously evaluating for signs of placental abruption. Measurement of vital signs is an essential initial step because maternal tachycardia, hypotension, tachypnea, or altered mental status may indicate significant blood loss and evolving hypovolemic shock. Importantly, maternal physiological compensation during pregnancy may initially preserve blood pressure despite substantial hemorrhage; therefore, subtle changes in pulse rate or orthostatic vital signs should not be overlooked. Continuous reassessment of maternal status is necessary because clinical deterioration may occur rapidly in severe cases [12]. Abdominal examination plays a central role in the diagnosis of placental abruption. Palpation of the gravid uterus should assess uterine tenderness, muscle tone, consistency, and the presence, frequency, and duration of uterine contractions. Patients with significant placental abruption often demonstrate localized or diffuse uterine tenderness accompanied by increased uterine tone or persistent tetanic contractions. In severe cases, the uterus may become rigid and board-like because of extensive retroplacental bleeding infiltrating the myometrium. These findings provide important clinical evidence supporting the diagnosis, particularly when interpreted alongside maternal symptoms and fetal assessment. A careful pelvic examination is also required to evaluate the source and characteristics of vaginal bleeding. A sterile speculum examination permits visualization of the cervix while identifying active bleeding, cervical lesions, rupture of membranes, or other local causes of hemorrhage. The quantity, color, and presence of blood clots should be documented because these observations may provide additional diagnostic information. However, if placental location has not been confirmed by prior ultrasonography, digital cervical examination should be postponed until placenta previa has been excluded, as digital examination in the presence of placenta previa may provoke catastrophic hemorrhage. Importantly, the absence of visible vaginal bleeding does not exclude placental abruption because concealed hemorrhage remains a common clinical presentation [12].

Assessment of fetal well-being constitutes an equally critical component of the evaluation because fetal compromise may develop before significant maternal instability becomes apparent. Initial assessment includes auscultation of fetal heart sounds and detailed questioning regarding recent fetal movements, with particular attention to reductions in fetal activity. For pregnancies that have reached fetal viability, continuous electronic fetal monitoring is recommended to detect abnormalities such as prolonged fetal bradycardia, decreased baseline variability, recurrent late decelerations, or other signs of fetal hypoxia. In pregnancies below the threshold of viability, confirmation of fetal cardiac activity using Doppler ultrasonography is generally sufficient. Integration of maternal clinical findings with continuous fetal surveillance enables clinicians to determine disease severity, prioritize emergency interventions, and make timely decisions regarding delivery when maternal or fetal status deteriorates [12].

## **Evaluation**

The evaluation of suspected placental abruption is primarily based on careful clinical assessment because no single laboratory investigation or diagnostic imaging modality can definitively confirm or exclude the diagnosis before delivery. Instead, the diagnostic approach relies on integrating the patient's history, physical examination findings, maternal hemodynamic status, fetal assessment, laboratory investigations, and imaging studies to determine the likelihood and severity of placental abruption while simultaneously excluding other causes of antepartum hemorrhage. Prompt evaluation is particularly important because maternal and fetal conditions may deteriorate rapidly, necessitating urgent intervention even in the absence of definitive diagnostic confirmation [13][14][15]. Ultrasonography is commonly performed as an initial imaging study in pregnant patients presenting with vaginal bleeding or abdominal pain because of its value in identifying placental location and excluding alternative obstetric

conditions, particularly placenta previa. Determining whether the placenta overlies the cervical os is critical before performing any digital cervical examination, thereby reducing the risk of provoking severe hemorrhage in cases of placenta previa. Despite its widespread availability and importance in obstetric practice, ultrasonography has limited sensitivity for detecting placental abruption. During the acute stage of hemorrhage, the retroplacental blood clot frequently appears isoechoic, exhibiting an echogenicity similar to that of the surrounding placental tissue. This similarity makes visualization of concealed retroplacental hemorrhage challenging, particularly during the early stages of separation. Consequently, a normal ultrasound examination does not exclude placental abruption, and the absence of ultrasonographic abnormalities should never delay appropriate clinical management when the patient's presentation strongly suggests the diagnosis. Likewise, ultrasound findings alone are insufficient to establish a definitive diagnosis of placental abruption [13][14][15].

Continuous evaluation of fetal status represents one of the most valuable components of the diagnostic process. Electronic fetal heart rate monitoring provides indirect evidence regarding the adequacy of uteroplacental perfusion and fetal oxygenation, allowing clinicians to identify early signs of fetal compromise. Although fetal heart rate monitoring cannot independently confirm or exclude placental abruption, it serves as a critical indicator of disease severity and guides subsequent management decisions. A Category I fetal heart tracing is considered reassuring and reflects normal fetal acid-base status, although it does not eliminate the possibility of placental abruption. Category II tracings are indeterminate and require continued surveillance, additional diagnostic evaluation, and repeated clinical reassessment because fetal status may evolve rapidly. In contrast, a persistent Category III fetal heart tracing strongly suggests significant fetal hypoxia and acidemia, indicating the need for immediate intrauterine resuscitative measures and, if no improvement occurs, prompt delivery to prevent irreversible fetal injury. The presence of a sinusoidal fetal heart rate pattern warrants particular concern because it may indicate severe fetal anemia secondary to substantial fetomaternal hemorrhage associated with placental abruption [16]. For patients with relatively stable maternal and fetal conditions in whom conservative management is considered appropriate, additional methods of fetal surveillance may be employed. The biophysical profile provides a comprehensive assessment of fetal well-being by combining ultrasonographic evaluation with nonstress testing. This examination assesses fetal breathing movements, gross body movements, fetal muscle tone, amniotic fluid volume, and fetal heart rate reactivity. Serial biophysical profile assessments may assist clinicians in monitoring fetal condition over time and identifying deterioration that necessitates expedited delivery.

Laboratory investigations play an essential supportive role in the evaluation of placental abruption by establishing baseline physiological parameters, assessing the severity of maternal hemorrhage, and identifying complications related to coagulation abnormalities. Blood typing and antibody screening are routinely performed to facilitate timely blood transfusion should significant hemorrhage occur. A complete blood count provides valuable information regarding hemoglobin concentration, hematocrit, and platelet count, enabling assessment of acute blood loss and ongoing hematological changes. Coagulation studies, including fibrinogen concentration, prothrombin time, and activated partial thromboplastin time, are particularly important because severe placental abruption may precipitate disseminated intravascular coagulation through widespread activation of the coagulation cascade. Serial monitoring of these parameters allows early detection of coagulopathy and guides replacement therapy with blood products when necessary. Measurement of blood urea nitrogen and other indicators of renal function further assists in evaluating end-organ perfusion and identifying complications resulting from hypovolemia and shock [13][14][15]. The Kleihauer-Betke test may also be incorporated into the diagnostic evaluation to quantify the degree of fetomaternal hemorrhage by identifying fetal erythrocytes within the maternal circulation. Although this test does not establish the diagnosis of placental abruption, it provides clinically important information regarding the extent of fetal blood transfer into the maternal bloodstream. Its greatest clinical value is observed in Rh-negative pregnant patients, in whom fetomaternal hemorrhage may result in maternal sensitization and subsequent Rh isoimmunization. Quantification of fetal red blood cells enables clinicians to calculate the appropriate dose of Rh(D) immune globulin required to prevent maternal alloimmunization and reduce the risk of hemolytic disease in future pregnancies. Therefore, while no individual laboratory or imaging study can definitively diagnose placental abruption before delivery, the combined use of clinical assessment, fetal surveillance, laboratory evaluation, and targeted imaging provides a comprehensive framework for timely diagnosis, risk stratification, and evidence-based management of this potentially life-threatening obstetric emergency [13][14][15].

### **Treatment / Management**

Placental abruption is an obstetric emergency characterized by its abrupt onset, unpredictable progression, and potential to rapidly compromise both maternal and fetal health. Because the condition may deteriorate within minutes, immediate recognition and timely intervention are essential to reduce maternal morbidity, fetal hypoxia, and perinatal mortality. The primary objectives of management include stabilization of the mother's physiological condition, continuous evaluation of fetal well-being, prevention of hemorrhagic complications, and determination of the safest timing and mode of delivery. Successful management requires close collaboration among emergency medical services personnel, obstetricians, anesthesiologists, neonatologists, hematologists, and critical care specialists to ensure coordinated, evidence-based care throughout the patient's clinical course [17][18]. Management begins in the

prehospital setting, where early recognition of placental abruption is critical. Pregnant patients presenting with abdominal pain, vaginal bleeding, uterine tenderness, or signs of maternal shock should be treated as having a potential obstetric emergency until proven otherwise. Advanced life support measures should be initiated promptly, including airway assessment, administration of supplemental oxygen when indicated, establishment of intravenous access, and continuous monitoring of maternal vital signs. Rapid transport to a healthcare facility equipped with comprehensive obstetric services, emergency operative capability, blood bank support, and a neonatal intensive care unit is essential because definitive management may require immediate surgical intervention and advanced neonatal resuscitation [17][18].

Upon hospital arrival, maternal stabilization remains the initial priority. Intravenous fluid resuscitation is initiated to maintain adequate circulating blood volume and preserve tissue perfusion, while supplemental oxygen is administered to optimize maternal and fetal oxygen delivery. Continuous monitoring of maternal cardiovascular status and fetal heart rate, when gestational age permits fetal viability, provides ongoing assessment of disease progression. Simultaneously, a comprehensive clinical evaluation, including detailed history, physical examination, laboratory investigations, and appropriate imaging studies, is performed to determine the severity of placental separation, identify associated complications, and guide subsequent management decisions. The therapeutic approach is individualized according to maternal hemodynamic stability, gestational age, fetal condition, extent of placental separation, and the presence or absence of active hemorrhage [17][18]. Patients diagnosed with mild, or Class 1, placental abruption who demonstrate no evidence of maternal instability or fetal compromise and whose pregnancies remain preterm, generally before 37 weeks of gestation, may be candidates for conservative management. These patients are typically admitted to a specialized obstetric unit for close maternal and fetal surveillance. Continuous observation allows early detection of any progression in hemorrhage, uterine activity, or fetal compromise that would necessitate immediate delivery. Intravenous access is maintained throughout hospitalization, and blood samples are obtained for blood typing, cross-matching, complete blood count, and coagulation studies in anticipation of possible transfusion requirements. In selected clinically stable patients, outpatient management with strict follow-up may be considered after careful risk assessment. Such management includes frequent prenatal evaluations, serial fetal surveillance using non-stress testing and biophysical profiles, and repeated ultrasonographic assessment of fetal growth because placental abruption is associated with an increased risk of fetal growth restriction and placental insufficiency [17][18].

The decision to proceed with delivery depends on continuous reassessment of both maternal and fetal status rather than gestational age alone. Important considerations include maternal cardiovascular stability, severity of hemorrhage, laboratory evidence of coagulopathy, fetal viability, and evidence of fetal distress. In cases of confirmed intrauterine fetal demise, prompt delivery is generally recommended to minimize maternal complications, including disseminated intravascular coagulation and severe hemorrhage. Conversely, when the fetus remains viable and placental abruption is classified as moderate or severe, expedited delivery is usually indicated to preserve fetal survival while preventing further maternal deterioration. Continuous communication among obstetric, anesthesia, and neonatal teams is essential to coordinate the timing and preparation for delivery [17][18]. The route of delivery is determined according to the evolving clinical situation. Vaginal delivery may be achieved rapidly in many patients because uterine hypertonicity and strong contractions frequently accelerate labor. When maternal and fetal conditions permit, vaginal birth is often preferred because it carries a lower risk of surgical blood loss and avoids operative complications, particularly in patients with existing coagulation abnormalities. However, emergency cesarean delivery becomes necessary when persistent fetal distress, worsening maternal hemorrhage, progressive hemodynamic instability, or failure to achieve timely vaginal delivery threatens maternal or fetal survival. During operative management, meticulous attention must be directed toward maintaining adequate intravascular volume through appropriate crystalloid administration and blood component replacement to correct anemia and coagulation abnormalities while preventing circulatory collapse [17][18].

Following delivery, intensive postpartum monitoring remains essential because maternal complications may continue despite successful birth. Patients should be observed closely for postpartum hemorrhage, persistent uterine atony, ongoing concealed bleeding, disseminated intravascular coagulation, and evidence of multiorgan dysfunction. Serial laboratory evaluation of hemoglobin concentration, platelet count, fibrinogen levels, and coagulation parameters guides ongoing transfusion therapy and correction of hemostatic abnormalities. Simultaneously, the neonatal team should be present at delivery and informed of the suspected placental abruption because fetomaternal hemorrhage and prolonged fetal hypoxia increase the likelihood of neonatal anemia, respiratory compromise, metabolic acidosis, and the need for advanced neonatal resuscitation and intensive care. Comprehensive multidisciplinary management extending from prehospital recognition through postpartum and neonatal care remains the cornerstone for improving outcomes in this life-threatening obstetric emergency [17][18].

### **Enhancing Healthcare Team Outcomes**

Placental abruption is a life-threatening obstetric emergency that requires coordinated management by an experienced interprofessional healthcare team to optimize maternal and neonatal outcomes. Effective care depends on seamless collaboration among obstetricians, emergency physicians, obstetric nurses, obstetric anesthesiologists, neonatologists, critical care specialists, blood bank personnel, and emergency medical services providers. Because the clinical

presentation ranges from mild vaginal bleeding with stable maternal and fetal conditions to catastrophic hemorrhage accompanied by maternal shock and fetal compromise, rapid clinical assessment and early recognition are essential for determining the appropriate level of intervention. Immediate evaluation of maternal hemodynamic status, continuous fetal monitoring when appropriate, prompt initiation of resuscitative measures, and timely notification of the obstetric team form the foundation of successful management. Patients with significant hemorrhage or evidence of coagulopathy require activation of massive transfusion protocols, preparation of blood products, early involvement of intensive care specialists, and readiness of the operating room should emergency delivery become necessary. Simultaneously, the neonatal intensive care team must be informed to prepare for advanced neonatal resuscitation and management of complications related to prematurity, fetal hypoxia, or fetal anemia. This coordinated multidisciplinary approach significantly reduces maternal and neonatal morbidity and mortality while facilitating rapid, evidence-based clinical decision-making. Although placental abruption cannot always be prevented, reducing modifiable risk factors remains an important component of comprehensive obstetric care. Smoking cessation, avoidance of cocaine and other vasoactive substances, and referral for substance use counseling or rehabilitation when indicated contribute to lowering the risk of placental vascular injury. Careful management of chronic hypertension before and throughout pregnancy has also been shown to reduce the incidence of placental abruption by improving maternal vascular health and uteroplacental perfusion [19][20][21]. Despite advances in obstetric care, placental abruption continues to carry substantial maternal and fetal risks. Extensive placental separation may rapidly result in fetal death unless immediate delivery is achieved, with reported fetal mortality ranging from 1% to 40% depending on gestational age and disease severity. Maternal complications include severe hemorrhage, blood transfusion, disseminated intravascular coagulation, hysterectomy, cesarean delivery, and increased risk of recurrent placental abruption in subsequent pregnancies, with recurrence rates estimated between 3% and 10%. In the United States, placental abruption remains responsible for approximately 1% to 5% of maternal deaths each year, emphasizing the continued importance of vigilant surveillance, timely intervention, and multidisciplinary management throughout pregnancy and delivery [2][22].

## Conclusion

Placental abruption remains one of the most critical obstetric emergencies encountered in clinical and prehospital practice because of its rapid onset, unpredictable progression, and potentially devastating consequences for both mother and fetus. Although relatively uncommon, it contributes substantially to maternal hemorrhage, fetal hypoxia, preterm birth, perinatal mortality, and maternal morbidity. Prompt recognition of characteristic clinical manifestations, comprehensive maternal and fetal assessment, and rapid stabilization are essential to improve outcomes. Since no single diagnostic test can definitively confirm placental abruption before delivery, clinical judgment supported by fetal monitoring, laboratory investigations, and targeted imaging remains the cornerstone of diagnosis. Effective management requires individualized treatment based on maternal hemodynamic status, gestational age, fetal condition, and disease severity, with timely delivery when maternal or fetal compromise is evident. Emergency medical services play a pivotal role by initiating early resuscitation, ensuring safe transport, and facilitating rapid access to definitive obstetric care. Close collaboration among emergency clinicians, obstetricians, anesthesiologists, neonatologists, intensive care specialists, and transfusion services is fundamental for reducing complications and improving survival. Continued emphasis on risk factor modification, evidence-based protocols, multidisciplinary training, and coordinated emergency response will further enhance the quality of care and optimize maternal and neonatal outcomes in patients affected by placental abruption.

## References

1. Workalemahu T, Enquobahrie DA, Gelaye B, Thornton TA, Tekola-Ayele F, Sanchez SE, Garcia PJ, Palomino HG, Hajat A, Romero R, Ananth CV, Williams MA. Abruptio placentae risk and genetic variations in mitochondrial biogenesis and oxidative phosphorylation: replication of a candidate gene association study. *Am J Obstet Gynecol*. 2018 Dec;219(6):617.e1-617.e17.
2. Martinelli KG, Garcia EM, Santos Neto ETD, Gama SGND. Advanced maternal age and its association with placenta praevia and placental abruption: a meta-analysis. *Cad Saude Publica*. 2018 Feb 19;34(2):e00206116.
3. Workalemahu T, Enquobahrie DA, Gelaye B, Sanchez SE, Garcia PJ, Tekola-Ayele F, Hajat A, Thornton TA, Ananth CV, Williams MA. Genetic variations and risk of placental abruption: A genome-wide association study and meta-analysis of genome-wide association studies. *Placenta*. 2018 Jun;66:8-16.
4. Sylvester HC, Stringer M. Placental abruption leading to hysterectomy. *BMJ Case Rep*. 2017 Dec 11;2017
5. Schneider E, Kinzler WL. Placental Abruption: Pathophysiology, Diagnosis, and Management. *Clin Obstet Gynecol*. 2025 Mar 01;68(1):98-104.
6. Oyelese Y, Ananth CV. Placental abruption. *Obstet Gynecol*. 2006 Oct;108(4):1005-16.
7. Tikkanen M. Placental abruption: epidemiology, risk factors and consequences. *Acta Obstet Gynecol Scand*. 2011 Feb;90(2):140-9.
8. Brandt JS, Ananth CV. Placental abruption at near-term and term gestations: pathophysiology, epidemiology, diagnosis, and management. *Am J Obstet Gynecol*. 2023 May;228(5S):S1313-S1329.

9. Smith CD, Lee A. Placental abruption. *BJA Educ.* 2024 Sep;24(9):305-308.
10. Miller C, Grynspan D, Gaudet L, Ferretti E, Lawrence S, Moretti F, Lafreniere A, McGee A, Lattuca S, Black A. Maternal and neonatal characteristics of a Canadian urban cohort receiving treatment for opioid use disorder during pregnancy. *J Dev Orig Health Dis.* 2019 Feb;10(1):132-137.
11. Plowman RS, Javidan-Nejad C, Raptis CA, Katz DS, Mellnick VM, Bhalla S, Cornejo P, Menias CO. Imaging of Pregnancy-related Vascular Complications. *Radiographics.* 2017 Jul-Aug;37(4):1270-1289.
12. DeRoo L, Skjærven R, Wilcox A, Klungsøyr K, Wikström AK, Morken NH, Cnattingius S. Placental abruption and long-term maternal cardiovascular disease mortality: a population-based registry study in Norway and Sweden. *Eur J Epidemiol.* 2016 May;31(5):501-11.
13. Ananth CV, Wapner RJ, Ananth S, D'Alton ME, Vintzileos AM. First-Trimester and Second-Trimester Maternal Serum Biomarkers as Predictors of Placental Abruption. *Obstet Gynecol.* 2017 Mar;129(3):465-472.
14. Arlier S, Adiguzel C, Yilmaz ES, Seyfettinoglu S, Helvacioğlu C, Ekin GU, Nazik H, Yucel O. The role of mean platelet volume and platelet distribution width in the prediction of placental abruption. *J Obstet Gynaecol.* 2016 Oct;36(7):950-953.
15. Saphier NB, Kopelman TR. Traumatic Abruptio Placenta Scale (TAPS): a proposed grading system of computed tomography evaluation of placental abruption in the trauma patient. *Emerg Radiol.* 2014 Feb;21(1):17-22.
16. Tweddell SM, Bahr TM, Henry E, Page JM, Ilstrup SJ, Ohls RK, Christensen RD. Placental abruption and neonatal anemia. *J Perinatol.* 2023 Jun;43(6):782-786.
17. Society for Maternal-Fetal Medicine (SMFM). Electronic address: [pubs@smfm.org](mailto:pubs@smfm.org). Gyamfi-Bannerman C. Society for Maternal-Fetal Medicine (SMFM) Consult Series #44: Management of bleeding in the late preterm period. *Am J Obstet Gynecol.* 2018 Jan;218(1):B2-B8.
18. Downes KL, Grantz KL, Shenassa ED. Maternal, Labor, Delivery, and Perinatal Outcomes Associated with Placental Abruption: A Systematic Review. *Am J Perinatol.* 2017 Aug;34(10):935-957.
19. Boisramé T, Sananès N, Fritz G, Boudier E, Aissi G, Favre R, Langer B. Placental abruption: risk factors, management and maternal-fetal prognosis. Cohort study over 10 years. *Eur J Obstet Gynecol Reprod Biol.* 2014 Aug;179:100-4.
20. Abe M, Arima H, Yoshida Y, Fukami A, Sakima A, Metoki H, Tada K, Mito A, Morimoto S, Shibata H, Mukoyama M. Optimal blood pressure target to prevent severe hypertension in pregnancy: A systematic review and meta-analysis. *Hypertens Res.* 2022 May;45(5):887-899.
21. Tita AT, Szychowski JM, Boggess K, Dugoff L, Sibai B, Lawrence K, Hughes BL, Bell J, Aagaard K, Edwards RK, Gibson K, Haas DM, Plante L, Metz T, Casey B, Esplin S, Longo S, Hoffman M, Saade GR, Hoppe KK, Foroutan J, Tuuli M, Owens MY, Simhan HN, Frey H, Rosen T, Palatnik A, Baker S, August P, Reddy UM, Kinzler W, Su E, Krishna I, Nguyen N, Norton ME, Skupski D, El-Sayed YY, Ogunyemi D, Galis ZS, Harper L, Ambalavanan N, Geller NL, Oparil S, Cutter GR, Andrews WW., Chronic Hypertension and Pregnancy (CHAP) Trial Consortium. Treatment for Mild Chronic Hypertension during Pregnancy. *N Engl J Med.* 2022 May 12;386(19):1781-1792.
22. Downes KL, Shenassa ED, Grantz KL. Neonatal Outcomes Associated With Placental Abruption. *Am J Epidemiol.* 2017 Dec 15;186(12):1319-1328.