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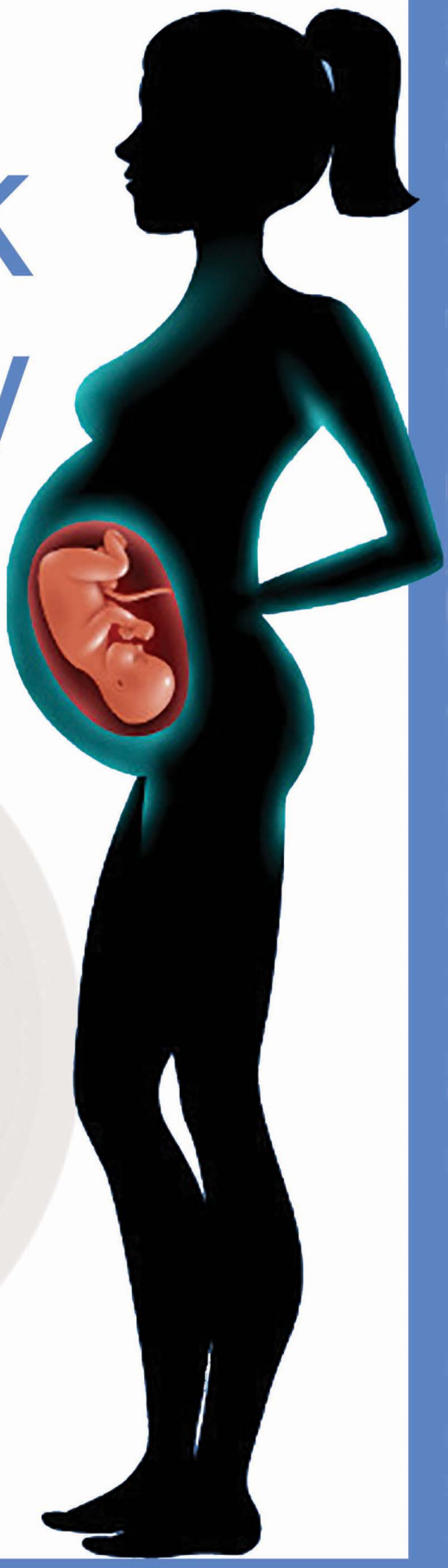
Textbook on High-Risk Pregnancy for Postgraduates



Professor Firoza Begum



Foreword
TA Chowdhury

JAYPEE

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CHAPTER

4

HELLP Syndrome

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INTRODUCTION

Hemolysis, elevated liver enzymes, and low platelets (HELLP) is an acronym that refers to a syndrome in pregnant and postpartum women characterized by hemolysis with a microangiopathic blood smear, elevated liver enzymes, and a low platelet count. It probably represents a severe form of preeclampsia, but the relationship between the two disorders remains controversial because as many as 15–20% of patients with HELLP syndrome do not have antecedent hypertension or proteinuria. Although different diagnostic benchmarks have been proposed, many clinicians use the following criteria to make the diagnosis: lactate dehydrogenase (LDH) elevated to 600 IU/L or more, aspartate aminotransferase (AST), and alanine aminotransferase (ALT) elevated more than twice the upper limit of normal, and the platelets count <100,000mm^3.

Hemolysis, elevated liver enzymes, and low platelets syndrome may have an insidious and atypical onset, with up to 15% of the patients lacking either hypertension or proteinuria. The main presenting symptoms are right upper quadrant (RUQ) pain and generalized malaise in up to 90% of cases and nausea and vomiting in 50% of cases. Some patients may present without these signs/symptoms when first evaluated for unexplained thrombocytopenia. The diagnosis of HELLP syndrome should be considered in any pregnant patient presenting in the second half of gestation or immediate postpartum with significant new-onset epigastric/RUQ pain until proven otherwise.

INCIDENCE

Hemolysis, elevated liver enzymes, and low platelets syndrome occurs in approximately 0.2–0.6% of all pregnancies. In pregnancies complicated by preeclampsia, laboratory findings of HELLP are present in 2–20% of cases; in those complicated by eclampsia, HELLP syndrome is present in 10–30% of cases. About 70% of cases occur in the third trimester of pregnancy, and the remainder occurs within 48 hours of delivery.

RISK FACTORS

A variety of genetic variants associated with an increased risk for HELLP syndrome has been reported, but they have no role in clinical management. A prior pregnancy with preeclampsia with or without HELLP syndrome is a risk factor for HELLP syndrome. In contrast to preeclampsia, nulliparity is not a risk factor for HELLP syndrome. Key risk factors include white ethnicity, advanced maternal age >35 years, multiparity, obesity, chronic hypertension, diabetes mellitus, autoimmune disorders, and abnormal placentation (e.g., molar pregnancy).

PATHOGENESIS

The pathogenesis of HELLP syndrome is not well understood. The findings of this multisystem disease are attributed to abnormal vascular tone, vasospasm, and coagulation defects. To date, no common precipitating factor has been found. The syndrome seems to be the final manifestation of some insult that leads to microvascular endothelial damage and intravascular platelet activation. With platelet activation, thromboxane A and serotonin are released, causing vasospasm, platelet agglutination and aggregation, and further endothelial damage. Dysfunction in the complement system via excessive activation or defective regulation for a given amount of endothelial injury has been proposed to cause damage to hepatic vessels in HELLP. In <2% of patients with HELLP syndrome, the underlying etiology appears to be related to fetal long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency.

PATHOPHYSIOLOGICAL CHANGES

The hemolysis in HELLP syndrome is a microangiopathic hemolytic anemia. Red blood cells become fragmented as they pass through small blood vessels with endothelial damage and fibrin deposits. The peripheral smear may reveal spherocytes, schistocytes, triangular cells, and burr cells. The elevated liver enzyme levels in the syndrome are thought to be secondary to obstruction of hepatic blood flow by fibrin deposits in the sinusoids. This obstruction

leads to periportal necrosis and, in severe cases, intrahepatic hemorrhage, subcapsular hematoma formation, or hepatic rupture. The thrombocytopenia has been attributed to increased consumption and/or destruction of platelets.

Although some investigators speculate that disseminated intravascular coagulopathy (DIC) is the primary process in HELLP syndrome, most patients show no abnormalities on coagulation studies except those with well-developed HELLP syndrome who shows features of coagulopathy.

DIAGNOSIS

Clinical Presentation

The syndrome generally presents in the third trimester of pregnancy, although it occurs in <27 weeks of gestation in an estimated 11% of patients. HELLP syndrome occurs in 69% of patients in the antepartum and in 31% of patients in the postpartum period. With postpartum presentation, the onset is typically within the first 48 hours after delivery; however, signs and symptoms may not become apparent until as long as 7 days after delivery.

The vague nature of the presenting complaints can make the diagnosis difficult. Approximately 90% of patients present with generalized malaise, 65% with epigastric pain, 30% with nausea and vomiting, and 31% with headache. Because early diagnosis of this syndrome is critical, any pregnant woman who presents with malaise or a viral-type illness in the third trimester should be evaluated with a complete blood cell count and liver function tests (LFTs).

The physical examination may be normal. On physical examination, two-thirds patients manifest edema and hypertension; RUQ tenderness is present in as many as 90% of affected women as are brisk reflexes. Jaundice and bleeding manifestations, such as hematuria may rarely be present.

The differential diagnosis of HELLP syndrome includes acute fatty liver (AFP) of pregnancy, thrombotic thrombocytopenic purpura (TTP), and hemolytic uremic syndrome. Because of the variable nature of the clinical presentation, the diagnosis of HELLP syndrome is generally delayed for an average of 8 days. Many woman with this syndrome are initially misdiagnosed with other disorders, such as cholecystitis, esophagitis, gastritis, hepatitis, or idiopathic thrombocytopenia.

Laboratory Criteria for Diagnosis

The following laboratory criteria should be met for diagnosis of HELLP syndrome:

- *Hemolysis:* One of the features of HELLP syndrome is microangiopathic hemolytic anemia. Schistocytes or helmet cells, burr cells, and polychromasia present on a peripheral blood smear are diagnostic of microangiopathic hemolytic anemia, making peripheral smears useful in the workup for HELLP syndrome **(Fig. 1)**.
- Elevated LDH (>600 IU/L or twice the upper limit of normal concentration)

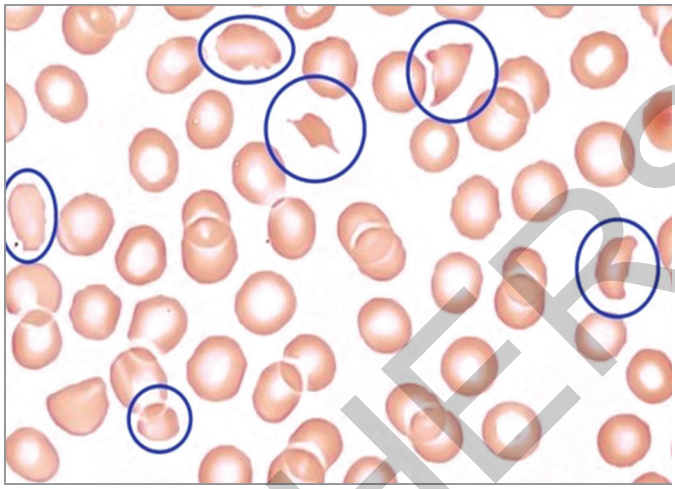

Fig. 1: Schistocytes.
Source: GP online (Photograph: D Roberts and J Burthem)

- Elevated total and indirect (unconjugated) bilirubin (total bilirubin 1.2 mg/dL or more)
- *Elevated liver transaminases:* AST and/or ALT >70 IU/L, or twice the upper limit of normal concentration
- *Thrombocytopenia:* Platelets <100,000/mm^3.
- Low serum haptoglobin (≤25 mg/dL)

In HELLP syndrome, an elevated LDH level is a nonspecific marker that can be associated with severe hemolysis, acute hepatocellular injury, or both. The total bilirubin level is increased as a result of an increase in the indirect (unconjugated) fraction from hemolysis. Haptoglobin level is a specific marker of hemolysis: 25 mg/dL provides the best cutoff between hemolytic and nonhemolytic disorders.

CLASSIFICATION

Two classification systems are used for HELLP syndrome. "The Tennessee classification" is based on the number of abnormalities that are present. Patients are classified as having partial HELLP syndrome when one or two abnormalities are present or full HELLP syndrome when all three abnormalities are present. Women with full HELLP syndrome are at higher risk for complications, including DIC, than women with the partial syndrome.

"Mississippi classification" measures the severity of the syndrome using the lowest observed platelet count along with the other two main clinical criteria (LDH and AST). Class I is the more severe, with a relatively high risk of morbidity and mortality, compared to the other two classes.

- *Class 1, severe thrombocytopenia:* Platelet count ≤50,000/mm^3, LDH ≥600 IU/L, AST, and/or ALT ≥70 IU/L (major maternal morbidity 40–60%)
- *Class 2, moderate thrombocytopenia:* Platelet >50,000 to ≤100,000/mm^3, LDH ≥600 IU/L, and AST and/or ALT ≥70 IU/L (major maternal morbidity 20–40%)
- *Class 3, mild thrombocytopenia:* Platelet >100,000 to ≤150,000/mm^3, LDH ≥600 IU/L, and AST ≥40 IU/L (major maternal morbidity 20%).

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LABORATORY STUDIES

Initial laboratory studies in all patients with clinical features suspicious for severe preeclampsia or HELLP syndrome should include:

- Complete blood count (CBC)
- Liver function tests (AST, bilirubin, and LDH)
- Peripheral blood smear
- Uric acid, fibrinogen, and prothrombin time/partial thromboplastin time. These are undertaken in the patient with severe disease who might have evidence of advanced renal compromise, placental abruption, or the suspicion of a similar disorder.
- Optional studies include serum electrolytes, serum creatinine, and serum glucose.
- Patients with HELLP syndrome who complain of severe RUQ pain, neck pain, or shoulder pain should be considered for hepatic imaging, [computed tomography/magnetic resonance imaging (CT/MRI)] of liver in addition to ultrasound (USG) regardless of the severity of the laboratory abnormalities, to assess for subcapsular hematoma or rupture.

While hemolytic anemia is part of the pathology, patients with HELLP syndrome are frequently hemoconcentrated at baseline, a situation that may mask the hemolysis, at least until later stages of the disease. A urinalysis should be performed in all patients, and proteinuria, caused by increased renal tubular permeability, is found in 66–100% of patients. Uric acid level should be performed in patients with severe disease who might have evidence of advanced renal compromise, placental abruption, or the suspicion of an imitator disorder. Prolonged prothrombin time/partial thromboplastin time or reduced fibrinogen levels (<100 mg/dL) in addition to thrombocytopenia is indicative of progression to DIC. A positive D-dimer test in the setting of preeclampsia has recently been reported to be predictive of patients who will develop HELLP syndrome. The *D-dimer* is a more sensitive indicator of subclinical coagulopathy and may be positive before coagulation studies are abnormal.

HISTOPATHOLOGY

In the liver, intravascular fibrin deposits give rise to sinusoidal obstruction, intrahepatic vascular congestion, and increased hepatic pressures leading to hepatic necrosis. This may eventually result in intraparenchymal or subcapsular hemorrhage and capsular rupture.

DIFFERENTIAL DIAGNOSIS

Hemolysis, elevated liver enzymes, and low platelets syndrome should be differentiated from other disorders of pregnancy with similar features **(Table 1)**:

- *Preeclampsia:* Usually normal/slightly high liver enzymes, with normal platelet count. Schistocytes (fragmented red cells) are also absent.
- *Acute fatty liver of pregnancy:* Hypoglycemia is present in AFL but absent in HELLP syndrome.
- *Thrombotic thrombocytopenic purpura:* Usually manifests in second or third trimester, and liver abnormalities are not as elevated as in HELLP syndrome. Patients are typically normotensive and have undetectable ADAMTS 13 activity.
- *Hemolytic-uremic syndrome (HUS):* HUS has the same findings as TTP except that its incidence is higher in the postpartum period, and patients have signs of renal failure.
- *Lupus flare:* Liver pathology is absent in lupus.
- *Antiphospholipid syndrome (APS):* Dominant features of APS are arterial/venous thrombosis and repeated pregnancy loss. Lupus anticoagulant, cardiolipin

TABLE 1: Differentiating features.

Findings	*HELLP syndrome*	*AFLP*	*TTP*	*HUS*
Jaundice (%)	5–10	40–90	Rare	Rare
Urine findings	Proteinuria and evidence of hemolysis	Occasional proteinuria with conjugated bilirubin	Proteinuria with blood	Proteinuria
Thrombocytopenia	Present	Present	Present	Present
Hemolysis (%)	50–100	15–20	100	100
Anemia	Sometimes	No	Yes	Yes
DIC (%)	<20	50–100	Uncommon	Uncommon
Hypoglycemia	No	Common	No	No
Elevated ammonia	Rare	Sometimes	No	No
Elevated transaminases	High	High	Usually mild	Usually mild
Elevated bilirubin	Sometimes	Always	Always	Always
Impaired renal function (%)	50	90–100	30	100

Source: Sibai BM. Imitators of severe preeclampsia. Obstet Gynecol. 2007;109(4):956-66.
(AFLP: acute fatty liver of pregnancy; DIC: disseminated intravascular coagulopathy; HELLP: hemolysis, elevated liver enzymes, and low platelets; HUS: hemolytic-uremic syndrome; TTP: thrombotic thrombocytopenic purpura)

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antibodies, beta-glycoprotein antibodies, prothrombin time, and an activated partial thromboplastin time (aPTT) should be checked to confirm the diagnosis.

- *Other:* Viral hepatitis, cholecystitis, cholangitis, gastritis, gastric ulcer, acute pancreatitis, and upper urinary tract infection (UTI).

MANAGEMENT

Although the disorder is considered to be a severe form of preeclampsia, not all patients affected by HELLP syndrome meet the presenting diagnostic criteria for severe preeclampsia. In independent studies, severe hypertension was initially absent in 12–18% of patients and in another 15% blood pressure was normal, and 4–14% of patients had no proteinuria. So, all women suspected of preeclampsia, including those with nonspecific symptoms such as nausea, vomiting, or malaise, should be evaluated for possible HELLP syndrome.

Hemolysis, elevated liver enzymes, and low platelets syndrome is a serious condition characterized by progressive and sometimes rapid maternal and fetal deterioration. Prompt recognition of HELLP syndrome and timely initiation of therapy are vital to ensure the best outcome for mother and the fetus. Recent research suggests that morbidity and mortality do not increase when patients with HELLP are treated conservatively. The treatment approach should be based on the estimated gestational age and the condition of the mother and fetus. Once, the diagnosis of HELLP syndrome has been established, the best markers to follow are the LDH level and the platelet count.

Antenatal administration of dexamethasone in a high dosage of 10 mg intravenously every 12 hours has been shown to markedly improve the laboratory abnormalities associated with HELLP syndrome. Other expected outcomes include less progression to class 1 HELLP syndrome, infrequent need for antihypertensive therapy, less transfusion, a lower incidence of new major maternal morbidity after starting therapy, increased latency between diagnosis and delivery to enable vaginal delivery, facilitating maternal transfer to a tertiary care center, and postnatal maturity of fetal lungs. In these studies, dexamethasone is administered intravenously until delivery for any patient with class 1 or class 2 HELLP syndrome regardless of gestational age or any patient with class 3 HELLP syndrome regardless of gestational age who also has eclampsia, severe epigastric pain, severe hypertension, or any major organ system morbidity shown improvement in clinical and laboratory features. Dexamethasone was initiated for antepartum and/or first detected postpartum disease, following delivery and with evidence of resolving laboratory and clinical parameters of disease the dose of dexamethasone was reduced for at least 2 doses and then stopped.

The two largest, randomized, double-blind, placebo-controlled trials evaluating the use of dexamethasone to improve maternal outcome in patients with HELLP syndrome did not establish a benefit in contrast to initial observational studies and small randomized trials that suggested more rapid improvement in maternal laboratory and clinical parameters.

Patients with HELLP syndrome should be treated prophylactically with magnesium sulfate to prevent seizures, whether hypertension is present or not. A bolus of 4–6 g of magnesium sulfate as a 20% solution is given initially. This dose is followed by a maintenance infusion of 2 g/hour. The infusion should be titrated to urine output and serum magnesium level.

Antihypertensive therapy should be initiated if blood pressure is consistently >160/110 mm Hg to reduce the risk of maternal cerebral hemorrhage, placental abruption, and seizure. The goal is to maintain diastolic blood pressure between 90 and 100 mm Hg. The most commonly used antihypertensive agent is hydralazine (apresolin) given intravenously in small incremental doses of 2.5–5 mg (with 5 mg as the initial dose) every 15–20 minutes until the desired blood pressure is achieved. Labetalol and nifedipine are also used. A hypertensive crisis may be treated with a continuous infusion of nitroglycerin or sodium nitroprusside (nipride).

Between 38 and 93% of patients with HELLP syndrome receive some form of blood product. Patients with a platelet count >40,000/mm^3 (40×10^9/L) are unlikely to bleed. These patients do not require transfusion unless the platelet count drops to <20,000/mm^3 (20×10^9/L). Actively bleeding patients with thrombocytopenia should be transfused with platelets. Patients who undergo cesarean section should be transfused if their platelet count is <50,000/mm^3 (50×10^9/L). Effect of platelet transfusion is only transient as consumption occurs rapidly. One unit of platelets is expected to increase the platelet count by 5,000. Prophylactic transfusion of platelets at delivery does not reduce the incidence of postpartum hemorrhage or hasten normalization of the platelet count and therefore use is controversial.

Fibrinogen replacement is necessary at levels <100 mg/dL. To increase the serum level of fibrinogen by 25 mg, 1 g of exogenous fibrinogen has to be administered. This amount is provided by 1 unit of fresh frozen plasma or 6 units of cryoprecipitate. Those having DIC should be given fresh frozen plasma and packed red blood cells.

The laboratory abnormalities in HELLP syndrome typically worsen after delivery and then begin to resolve by 3–4 days postpartum. Plasmapheresis has been successful in patients with severe laboratory abnormalities (i.e., a platelet count of <30,000/mm^3 (30×10^9/L) and continued elevation of liver function values], who require repeated transfusions to maintain their hematocrit at 72 hours postpartum. In these patients, plasmapheresis has resulted in an increase in the platelet count and a decrease in the LDH level.

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Candidates for prompt delivery: The cornerstone of therapy for HELLP occurring during pregnancy is delivery, which is the only effective treatment. There is consensus among experts that prompt delivery is indicated after maternal stabilization for any of the following conditions:

- Pregnancies ≥34 weeks of gestation.
- Pregnancies that have not reached a stage of fetal maturity that ensures a reasonable chance of extrauterine survival.
- Fetal demise.
- Abruptio placentae.

In the absence of any of these scenarios *DIC, pulmonary edema, or acute kidney injury* or urgent clinical scenarios patient should be stabilized and delivered, delivery may be delayed until a course of corticosteroids has been administered. For this group of patients, recommendation is to give corticosteroids (betamethasone 12 mg intramuscularly every 12 hours for 2 doses or dexamethasone 12 mg intravenously every 12 hours for 4 doses) then deliver 24 hours after the last dose. Steroid administration is not only beneficial to the fetus for lung maturity but also for improvement of laboratory values in patients, particularly in elevating platelet counts. Maternal-fetal monitoring should be performed throughout each step of management.

In the past, delivery in patients with HELLP syndrome was routinely accomplished by cesarean section. Patients with severe HELLP syndrome, superimposed DIC, or a gestation of <32 weeks should be delivered by cesarean section. A trial of labor is appropriate in patients with mild to moderate HELLP syndrome who are stable, have a favorable cervix, and are at 32 weeks of gestation or greater.

ANESTHESIA

Pain relief with intravenous narcotics and local anesthesia is acceptable. Thrombocytopenia and coagulation abnormalities may preclude use of neuraxial anesthesia for labor and delivery. The minimum platelet count that is necessary to safely perform neuraxial anesthesia is unknown, and practice varies. Insertion of an epidural catheter is generally safe in patients with a platelet count >80,000/mm^3 (80 × 10^9/L), normal coagulation studies, and a normal bleeding time. General anesthesia can be used when regional anesthesia is considered unsafe.

PROGNOSIS

Hemolysis, elevated liver enzymes, and low platelets syndrome is a life-threatening condition. The mortality rate of women with HELLP syndrome is 0–24%, with a perinatal death rate of up to 37%. Maternal death most commonly occurs due to DIC, placental abruption, postpartum hemorrhage, acute renal failure, adult respiratory distress syndrome, subcapsular hematoma, and hepatic rupture. DIC occurs in 15–62.5% of the cases. Placental abruption occurs in 11–25% of the cases, postpartum hemorrhage occurs in 12.5–40%, and acute renal failure in 36–50% of the cases. Poor perinatal prognosis is because of placental abruption, intrauterine hypoxia and asphyxia, prematurity, and low birth weight.

Patients with HELLP syndrome have a 19–27% risk of developing HELLP syndrome in subsequent pregnancies. Class 1 HELLP syndrome has the highest recurrence rate. Recurrent cases occur in the latter part of the gestation period and are less severe after two episodes.

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High-Risk Pregnancy
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- This book provides a comprehensive overview of high-risk pregnancy management, based on all available literature, guidelines, and recent advances in medical technology
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- The chapters in this book cover the pathophysiology, formulation of diagnosis, systematic investigations and appropriate management of maternal and fetal disorders
- The content in this book will enable readers to acquire knowledge on different factors that affect both the mother and the fetus and identify conditions which can make a pregnancy high-risk.



Professor Firoza Begum is a reputed Obstetrician and Gynecologist of Bangladesh. Her credentials include extensive clinical experience and a distinguished academic career over many decades. She graduated from Sir Salimullah Medical College, Dhaka University, in 1979 and obtained fellowship (FCPS) in Obstetrics and Gynecology in July 1987. She became Professor of Feto-Maternal Medicine, Bangabandhu Sheikh Mujib Medical University (BSMMU) in 2006. Under her direct guidance and leadership, the Feto-Maternal Medicine Department was established in BSMMU and she became the Founding Chairman of the Department in 2018. As an academic, she keeps herself engaged in teaching postgraduate students, conducting research, and supervising research work of postgraduate students. Her field of expertise includes birth defects, recurrent pregnancy loss and assisted reproductive technology.

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