# Clinical Critical Care

E-ISSN 2774-0048

VOLUME 33 NUMBER 1

JANUARY-DECEMBER 2025

Clinical Critical Care
eISSN 2774-0048

Clinical Critical Care
Volume 33, Article ID e250009, 8 pages
https://doi.org/10.54205/ccc.v33.273471

REVIEW

# Management of critically ill obstetric patients

Touchapong Taksinwarajarn[1], Tananchai Petnak[2]

[1]Division of Critical Care Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400;
[2]Division of Pulmonary and Pulmonary Critical Care Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400

OPEN ACCESS
**Citation:**
Taksinwarajarn T, Petnak T. Management of critically ill obstetric patients. Clin Crit Care 2025; 33: e250009.

**Received:** February 3, 2025
**Revised:** March 13, 2025
**Accepted:** March 19, 2025



**Data Availability Statement:**
The data and code were available upon reasonable request (Tananchai Petnak, email address: tananchai.pet@gmail.com)

**Funding:**
No funding support.

**Competing interests:**
No potential conflict of interest relevant to this article was reported.

**Corresponding author:**
Tananchai Petnak
Division of Pulmonary and Pulmonary Critical Care Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand, 10400
Tel: (+66) 2-201-1629
E-mail: tananchai.pet@gmail.com

## ABSTRACT:

Pregnant patients requiring intensive care management present unique challenges due to physiological adaptations and pregnancy-specific conditions. While many aspects of critical care remain similar to non-pregnant patients, modifications in fluid resuscitation, medication selection, ventilatory support, and anticoagulation strategies are important to balance maternal stabilization with fetal safety. A multidisciplinary team approach, including obstetricians, intensivists, anesthesiologists, and neonatologists, is necessary for the effective management of critically ill pregnant patients. This review outlines key considerations in the critical care of pregnant patients, including hemodynamic, respiratory, and airway management, as well as sedation, thromboprophylaxis, and nutritional support. Additionally, pregnancy-related complications such as preeclampsia, pulmonary embolism, amniotic fluid embolism, and air embolism require specialized diagnostic and therapeutic approaches to ensure optimal maternal and fetal outcomes.

**Keywords:** Critical care in pregnancy; ICU; Pregnancy complications

## INTRODUCTION

During pregnancy, physiological changes occur to support maternal adaptation, fetal development, and preparation for the stresses of fetal growth and parturition. However, these adaptations can affect multiple organ systems and exacerbate underlying conditions, increasing the risk of critical illness. While most pregnancies progress without complications, a subset of patients develop pregnancy-specific conditions such as preeclampsia, postpartum hemorrhage, and peripartum cardiomyopathy, many of which may require intensive care unit (ICU) management and contribute significantly to maternal and fetal morbidity and mortality. Additionally, pregnant patients are also vulnerable to general critical care conditions, such as sepsis, acute respiratory distress syndrome, and trauma, which often require tailored approaches due to the physiological changes of pregnancy. Effective management of critically ill pregnant patients requires a multidisciplinary approach to optimize outcomes for both mother and fetus.

This review aims to provide an overview of the physiological changes during normal pregnancy, outline general principles of critical care management in pregnant patients, and discuss common obstetric and non-obstetric critical care conditions.

## PHYSIOLOGICAL CHANGES DURING PREGNANCY

### Alternation of the respiratory system

During pregnancy, the upper respiratory tract undergoes significant mucosal changes, including hyperemia, edema, plasma leakage, glandular hypersecretion, increased phagocytic activity, and elevated mucopolysaccharide content [1]. These changes contribute to upper airway congestion and increase the risk of epistaxis, particularly following procedures such as nasal intubation. Additional factors, such as reduced lung volumes due to decreased caudal traction on the upper airway and fat infiltration of the airway tissues, predispose pregnant patients to upper airway collapse, which is associated with a higher incidence of maternal-fetal complications, including sleep-disordered breathing, hypertension, preeclampsia, and fetal growth restriction (FGR).

Pregnancy also induces significant physiological and anatomical changes in the chest wall, including widening of the subcostal angle and elevation of the diaphragm. These changes are primarily affected by the hormone relaxin, which relaxes the lower rib-cage ligaments, as well as by the effects of an enlarged uterus and increased maternal weight [2]. These changes reduce chest wall compliance and decrease functional residual capacity (FRC) by approximately 10–25% (300–500 mL) [3]. The reduction in FRC, which is the largest oxygen reservoir in the lungs, increases the risk of hypoxemia during anesthesia or intubation [4]. The risk is further exacerbated by the increased oxygen consumption associated with pregnancy.

Despite these changes, most pulmonary parameters remain unchanged or minimally altered. Airway resistance is either unchanged or slightly decreased due to the bronchodilatory effects of progesterone and relaxin [2]. Vital capacity, total lung capacity, and diffusing capacity are generally unaffected. However, increased oxygen consumption, driven by the metabolic demands of the fetus, leads to an elevation in tidal volume and minute ventilation. This is driven by an enhanced central respiratory drive, mediated by a threefold sensitization of central chemoreceptors and a twofold increase in the hypoxic ventilatory response. These adaptations result in a state of primary respiratory alkalosis with compensatory metabolic acidosis, which is normal in pregnancy. Typical arterial blood gas values in pregnancy reflect these changes, with pH ranging from 7.40 to 7.47, $PaCO_2$ levels between 27 and 34 mmHg, slightly elevated $PaO_2$ (>100 mmHg), and serum $HCO_3$ levels of 18 to 21 mEq/L [5]. A summary of these respiratory physiological changes is provided in Table 1.

### KEY MESSAGES:

- Physiological changes during pregnancy significantly affect the functioning of multiple organ systems.
- Early diagnosis and a multidisciplinary team approach are essential for effectively managing critically ill pregnant patients and reducing maternal and fetal morbidity and mortality.

### Alternation of the cardiovascular system

Maternal cardiovascular adaptations during pregnancy are essential for maintaining placental blood flow and ensuring adequate fetal oxygen delivery. Circulatory failure leading to reduced placental blood flow is associated with FGR and fetal asphyxia. To support both maternal and fetal circulations, maternal blood volume increases gradually, reaching approximately 2 liters or 40% above pre-pregnancy levels. Consequently, cardiac output increases by 30-45% compared to baseline [6]. This rise in cardiac output is primarily driven by an increase in

**Table 1**. Respiratory physiological changes in pregnancy.

| Parameter | Change | Time course |
|---|---|---|
| Oxygen consumption | Increases 20-35% | Peak at 3rd trimester |
| Tidal volume | Increases 30-35% | Peak at 3rd trimester |
| Respiratory rate | Unchanged | |
| Minute ventilation | Increases 20-40% | Peak at 3rd trimester |
| Total lung capacity | Unchanged | |
| Chest wall compliance | Decreased | Peak at 3rd trimester |
| Lung compliance | Unchanged | |
| Functional residual capacity | Decreased 10-25% | Peak at 3rd trimester |
| Forced vital capacity | Unchanged | |
| FEV1 | Unchanged | |
| Diffusion capacity | Unchanged | |
| Expiratory reserve volume | Decreased | Peak at 3rd trimester |
| Residual volume | Decreased | Peak at 3rd trimester |

stroke volume, resulting from increased preload due to expanded blood volume and decreased systemic vascular resistance (SVR) [7].

Hormonal influences, particularly elevated estrogen and activation of the renin-angiotensin system, promote salt and water retention. Additionally, decreased plasma osmotic pressure due to lower serum albumin concentrations increases the risk of pulmonary edema. In the third trimester, cardiac output becomes position-dependent as the gravid uterus compresses the aorta and inferior vena cava in the supine position, impairing venous return and potentially leading to maternal hypotension [8].

Despite increased stroke volume and cardiac output, systemic blood pressure generally decreases during pregnancy. This is primarily due to a reduction in SVR caused by the low-resistance uteroplacental circulation and hormone-mediated vasodilation [9]. Similarly, pulmonary vascular resistance decreases, ensuring that mean pulmonary arterial pressure remains stable despite the elevated cardiac output [5]. A summary of these physiological changes is provided in Table 2.

### Alternation of the renal and gastrointestinal system

During pregnancy, renal blood flow gradually increases, reaching 60% to 80% above pre-pregnancy levels, causing an increase in glomerular filtration rate (GFR) throughout pregnancy. As a result, serum creatinine levels that are considered normal in non-pregnant patients may indicate renal dysfunction in pregnant patients. Additionally, fluid retention leads to a mild reduction in serum sodium levels [10].

Pregnancy also increases the risk of gastroesophageal reflux disease (GERD) and aspiration. This is primarily attributed to decreased lower esophageal sphincter tone caused by the effects of progesterone, as well as mechanical displacement of the stomach by the enlargement of the uterus and delayed gastric emptying [11]. In terms of hepatic function, serum alkaline phosphatase levels increase due to placental production, while serum aminotransferases and bilirubin remain unchanged. Additionally, serum albumin levels decrease mildly as a result of the hemodilution effect caused by plasma volume expansion.

### Alternation of the hematologic system

Maternal blood volume increases significantly in pregnancy, leading to hemodilution anemia due to a greater expansion of plasma volume compared to red blood cell mass [6]. The physiological stress during pregnancy also induces mild leukocytosis, primarily driven by an increase in neutrophils. Additionally, pregnancy is also associated with a hypercoagulable state characterized by elevation of coagulation factor levels, including fibrinogen, factors II, VII, VIII, X, XII, and von Willebrand factor [12]. While this change helps minimize hemorrhage during delivery, it also increases the risk of venous thromboembolism (VTE) [13].

## GENERAL CRITICAL CARE MANAGEMENT IN PREGNANCY

### General principles of critical care in pregnancy

Management for critically ill pregnant patients requires a balance between optimized maternal outcomes and minimizing fetal risks. Any intervention must be assessed for its potential impact on both, as treatments beneficial to the mother may pose risks to the fetus and vice versa. A thorough risk-benefit assessment is essential to guide clinical decisions and optimize outcomes. Managing these patients effectively requires a multidisciplinary team that includes intensivists, anesthesiologists, obstetricians, neonatologists, pharmacists, and specialized nurses [14]. Clear communication and collaboration among team members are key to ensuring timely interventions and comprehensive care. A patient-centered approach, guided by evidence-based practices, helps minimize complications and improve both maternal and fetal outcomes.

**Table 2**. Cardiovascular physiological changes in pregnancy.

| Parameter | Change | Time course |
|---|---|---|
| Maternal blood volume | Increases 40% | Peak at 3rd trimester |
| Hemoglobin | Increases 20-40% | Peak at 3rd trimester |
| Hematocrit | Decreased 12% | Nadir at 3rd trimester |
| Heart rate | Increases 10-30% | Peak at 3rd trimester |
| Stroke volume | Increases | Peak at 3rd trimester |
| Cardiac output | Increases 30-50% | Peak at 3rd trimester |
| Mean arterial pressure | Decreased 10-20% | Nadir at 2rd trimester |
| Mean pulmonary arterial pressure | Unchanged | |
| Central venous pressure | Unchanged | |
| Pulmonary capillary wedge pressure | Unchanged | |
| Systemic vascular resistance | Decreased 20-30% | |
| Pulmonary vascular resistance | Decreased 20-30% | |

## Hemodynamic management

The physiological changes of pregnancy significantly affect cardiac function and systemic vascular resistance, as previously mentioned. In cases requiring ICU care, comprehensive assessment and monitoring of hemodynamic parameters are essential for guiding treatment decisions. Cardiac output can be evaluated using both invasive methods and non-invasive methods. Invasive techniques, such as thermodilution and pulmonary artery catheters, provide detailed hemodynamic data but carry procedural risks. In contrast, non-invasive approaches, including echocardiography and thoracic bioimpedance, have become widely used due to their accessibility and ability to assess hemodynamic status without the risks associated with invasive monitoring. Among these, echocardiography provides real-time evaluation of left and right ventricular function, fluid responsiveness, and fluid intolerance. Additionally, echocardiography is a valuable diagnostic tool for pregnancy-related conditions such as peripartum cardiomyopathy, pulmonary embolism, and amniotic fluid embolism [14].

Volume status assessment is important to ensure the benefit of fluid administration in pregnant patients, as they are at high risk for pulmonary edema. However, traditional techniques using heart-lung interaction, such as pulse pressure variation, stroke volume variation, and respiratory variation of inferior vena cava diameter, may be less reliable in late pregnancy due to increased intra-abdominal pressure from the enlarged uterus [15]. A small prospective study by Brun et al. demonstrated that a >12% increase in the velocity time integral (VTI) of subaortic blood flow during passive leg raising is predictive of fluid responsiveness in patients with severe preeclampsia and oliguria (AUC = 0.93, 95% CI: 0.83–1.00). Furthermore, a <12% increase in VTI during passive leg raising, combined with a baseline VTI <21 cm, also predicts fluid responsiveness [16]. However, due to the small sample size and its limitations in late pregnancy, these findings should be interpreted with caution.

Organ perfusion assessment in pregnant patients with hemodynamic instability requires evaluating both macrocirculation and microcirculation. Beyond the common signs of end-organ dysfunction, such as level of consciousness, capillary refill time, urine output, serum lactate levels, and central venous oxygen saturation ($ScvO_2$), fetal heart rate (FHR) monitoring becomes an important part of perfusion assessment, particularly in patients with >24 weeks of gestation. The abnormality of FHR indicates fetal hypoxia, enabling the early detection of inadequate maternal resuscitation [17]. Integrating FHR monitoring into hemodynamic management helps guide treatment strategies aimed at optimizing outcomes for both the mother and fetus.

Resuscitation in pregnant patients follows the same fundamental principles as in general ICU care, focusing on restoring intravascular volume, maintaining adequate perfusion, and supporting cardiac function. Positioning adjustment by a 15-degree left lateral tilt in the supine position reduces aortocaval compression and consequently improves venous return and cardiac output. Adequate fluid resuscitation guided by fluid responsiveness is able to restore intravascular volume, enhance stroke volume, and avoid fluid overload. Finally, the use of vasopressors, inotropic agents, and mechanical circulatory support generally follows the same principles as in non-pregnant ICU patients [14].

## Airway management

Anatomical and physiological changes during pregnancy increase the risk of aspiration, difficult intubation, and peri-intubation hypoxemia. Pre-oxygenation with high-flow nasal cannula (HFNC) or non-invasive ventilation (NIV) is recommended to prolong safe apnea time and reduce the risk of peri-intubation hypoxemia. Nasal intubation should be avoided due to increased airway edema, which raises the risk of mucosal bleeding. Rapid-sequence induction (RSI) with endotracheal intubation is preferred, ideally performed by an experienced provider using a video laryngoscope to optimize first-pass success and minimize the risk of gastric aspiration and failed intubation [14].

## Ventilation management

Ventilation support is an important management tool for acute respiratory failure. In pregnancy, the efficacy and safety of HFNC and NIV have not been established through randomized controlled trials (RCTs). However, studies in non-pregnant patients suggest that HFNC and NIV may reduce the work of breathing, improve gas exchange, and potentially prevent the need for intubation. NIV should be used with caution, as it carries risks of pneumonia, barotrauma, aspiration, and hemodynamic compromise. Close monitoring is necessary to avoid delayed intubation, which may worsen maternal and fetal outcomes [18].

For pregnant patients with acute respiratory distress syndrome (ARDS), no RCTs have established the optimal management strategy. A lung-protective approach with low tidal volume ventilation and a plateau pressure target of <30 $cmH_2O$ is generally recommended [19]. Increased intra-abdominal pressure from the gravid uterus can decrease the chest wall compliance, resulting in increased airway pressures. Transpulmonary pressure monitoring is helpful to ensure optimal ventilatory support and minimize the risk of ventilator-induced lung injury [20].

Prone positioning has been reported as a safe and effective intervention in small case series and case reports. In late pregnancy, modifications, such as strategic padding above and below the gravid uterus, may be considered to prevent aortocaval compression [21]. Extracorporeal membrane oxygenation (ECMO) has been reported in case studies as a salvage therapy for severe ARDS with refractory hypoxemia [22].

The primary management goal in pregnant ARDS patients is to prevent maternal and fetal hypoxemia by maintaining $PaO_2$ >90 mmHg. Additionally, excessive hypercapnia and hypocapnia should be avoided to prevent uteroplacental compromise, fetal hypoxemia, and acidosis. The target of $PaCO_2$ levels ranges between 27–34 mmHg [14].

### Sedation in ICU

Sedation in pregnant patients is an important issue since physiological changes during pregnancy, including an increased volume of distribution, enhanced renal clearance, and upregulation of the cytochrome P450 (CYP450) enzyme, affect drug pharmacokinetics. Additionally, certain sedatives and neuromuscular blocking agents can cross the placental barrier, which may potentially cause fetal distress, particularly during the delivery period.

Propofol is considered a safer option (category B), while dexmedetomidine, morphine, and fentanyl (category C) may be used when benefits outweigh risks, though fetal effects should be monitored [23]. Benzodiazepines should be avoided, as they have been associated with congenital anomalies, including cleft palate [24].

Neuromuscular blocking agents must also be used with caution during pregnancy. Careful titration and nerve stimulation monitoring are recommended to limit fetal exposure. Cisatracurium and rocuronium are preferred (category B), whereas vecuronium, succinylcholine, and atracurium require careful dosing and monitoring (category C) [25]. Importantly, these agents should be administered at the lowest effective dose for the shortest duration to reduce fetal exposure [14].

### Other management

Critically ill pregnant patients require comprehensive management beyond hemodynamic and respiratory support. Additional considerations should also be considered. Maternal acid-base balance should be closely monitored and maintained, and thromboprophylaxis should be initiated early in critically ill pregnant patients to reduce the risk of thromboembolic complications [26]. There are no specific nutritional guidelines for critically ill pregnant patients. However, nutritional requirements can be assessed using general ICU nutritional guidelines. Energy expenditure is ideally determined using indirect calorimetry (IC). If IC is unavailable, the caloric requirement can be estimated using weight-based equations (20–25 kcal/kg/day). Protein intake should be approximately 1.3 g/kg of actual body weight or adjusted body weight per day [27]. Additionally, delivery should be considered when there are clear maternal or fetal indications, particularly if the gestational age is ≥25 weeks [28].

## COMMON ICU DISORDERS IN OBSTETRICS

In addition to general critical care considerations, pregnant patients are at risk for severe pregnancy-specific conditions that require specialized management. These conditions can lead to significant maternal and fetal morbidity if not promptly recognized and treated.

### Preeclampsia

Preeclampsia is a multisystem disorder of vascular dysfunction in pregnancy, characterized by new-onset hypertension and proteinuria after 20 weeks of gestation. It affects 2–8% of pregnancies and may persist postpartum. The etiology remains unclear, but it is believed to result from abnormal placental vascular development, leading to placental ischemia, oxidative stress, and altered angiogenic factor production. These changes disrupt maternal endothelial function, increasing vascular permeability, vasopressor sensitivity, and activation of the coagulation cascade [29].

Risk factors for preeclampsia include nulliparity, multifetal gestation, previous preeclampsia, a family history of preeclampsia, hydatidiform mole, chronic hypertension, chronic renal disease, diabetes mellitus, maternal age ≥35 years, autoimmune disease, antiphospholipid syndrome, obesity (body mass index >30), and obstructive sleep apnea [30].

The diagnosis of preeclampsia is based on the diagnostic criteria, including new-onset hypertension, defined as systolic blood pressure (SBP) of ≥140 mmHg or diastolic blood pressure (DBP) of ≥90 mmHg on two occasions at least 4 hours apart, along with proteinuria (≥0.3 g in a 24-hour urine collection or a protein/creatinine ratio ≥0.3) occurring after 20 weeks of gestation. Preeclampsia with severe features is defined as preeclampsia with any of the following: severe hypertension (SBP ≥160 mmHg or DBP ≥110 mmHg), pulmonary edema, renal insufficiency, impaired liver function, thrombocytopenia, CNS impairment, or visual disturbances [31].

The most important complications of preeclampsia include HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets) and eclampsia. HELLP syndrome occurs in 10–20% of preeclampsia with severe features and is associated with increased maternal and fetal morbidity. Eclampsia refers to the development of seizures in preeclampsia without an alternative cause, such as epilepsy or intracranial hemorrhage. Eclampsia can occur unexpectedly, even in patients without prior symptoms of preeclampsia, and may develop up to one month postpartum [31,32].

The primary treatment for preeclampsia is delivery, which is indicated at ≥37 weeks of gestation or when expectant management is contraindicated. Magnesium sulfate is recommended for seizure prophylaxis and should be administered to all patients with preeclampsia with severe features or eclampsia. Therapy should continue for 24 hours postpartum, with clinical monitoring and serum magnesium level assessment to prevent toxicity. In cases of overdose, intravenous calcium gluconate (10%) is the antidote, and diuretics may be used to enhance magnesium excretion [29]. Blood pressure control is necessary for acute-onset severe hypertension to prevent end-organ damage. Labetalol and hydralazine are the preferred antihypertensive agents, while long-acting oral nifedipine is an alternative when first-line options are contraindicated. Nitroprusside and angiotensin-converting enzyme inhibitors should be avoided due to fetal toxicity [30,31].

### Venous thromboembolism

Venous thromboembolism (VTE) is a leading cause of maternal morbidity and mortality, with the highest incidence occurring postpartum, affecting approximately 0.5 to 1.2 per 1,000 pregnancies. The increased risk of VTE during pregnancy is due to a combination of venous

stasis from uterine compression and immobility, hypercoagulability caused by elevated coagulation factors, and vascular injury that may result from delivery-related trauma. Additional risk factors include a history of VTE, advanced maternal age, inherited or acquired thrombophilia such as antithrombin deficiency, protein S or C deficiency, factor V Leiden mutation, prothrombin G20210A mutation, or antiphospholipid syndrome. Additionally, obesity and underlying medical comorbidities also contribute to the elevated risk [33].

Diagnosing VTE in pregnancy is challenging because symptoms such as shortness of breath, tachycardia, and leg swelling can resemble normal physiological changes of pregnancy. A combination of clinical evaluation and imaging is necessary for accurate diagnosis. Diagnostic options include chest X-ray, Doppler ultrasound, ventilation-perfusion (V/Q), single-photon emission computed tomography (SPECT), computed tomography pulmonary angiography (CTPA), and magnetic resonance angiography (MRA). The pregnancy-adapted YEARS algorithm, combined with D-dimer testing, is commonly used to rule out VTE and reduce unnecessary imaging. However, its diagnostic accuracy is highest in the first trimester and declines in the third trimester [13].

Anticoagulation therapy is the cornerstone of treatment for both massive and non-massive PE during pregnancy. Low-molecular-weight heparin (LMWH) and unfractionated heparin (UFH) are the preferred options due to their safety profile for both the mother and fetus [34]. Anti-activated coagulation factor X (anti-Xa) monitoring is recommended for patients with recurrent VTE despite adequate treatment, renal impairment, or extremes of body weight [35]. Fondaparinux may be considered in patients with allergies or adverse reactions to LMWH. However, warfarin and novel oral anticoagulants (NOACs) should be avoided during pregnancy due to the risk of embryopathy in the first trimester and complications such as fetal hemorrhage and placental abruption in the third trimester [36]. Anticoagulant therapy should be continued for at least six weeks postpartum, with a minimum overall treatment duration of three months. In cases of life-threatening VTE or high-risk thromboembolism, thrombolytic therapy or surgical embolectomy may be required [13]. The role of inferior vena cava (IVC) filters in pregnancy remains uncertain.

## Amniotic embolism

Amniotic fluid embolism (AFE) is a rare but life-threatening obstetric emergency that occurs during labor, delivery, or immediately postpartum [37]. The mortality rate varies across studies, ranging from approximately 11% to 43%.

The pathophysiology of AFE remains unclear, but it is hypothesized that the entry of fetal squamous cells and other amniotic debris into the maternal circulation leads to pulmonary vascular obstruction. This triggers a sudden increase in pulmonary vascular resistance, causing acute right ventricular failure followed by left ventricular failure. Additionally, amniotic fluid components activate a systemic inflammatory response through C1-esterase inhibitor depletion, complement activation, and the release of leukotrienes and arachidonic acid metabolites, such as prostaglandin F2α [38]. These mechanisms contribute to hemodynamic collapse, disseminated intravascular coagulation (DIC), and multi-organ failure.

AFE typically presents with sudden respiratory failure, cardiovascular collapse, seizures, and coagulopathy during labor, delivery, or uterine manipulation [39]. In rare cases, it has been reported in first- or second-trimester abortions. Chest imaging may show bilateral parenchymal opacities, consistent with pulmonary edema and increased permeability-related lung injury, which can progress to ARDS.

There is no definitive treatment for AFE, and management is supportive. This includes cardiopulmonary resuscitation to ensure adequate oxygenation and circulatory stabilization, as well as effective bleeding control through correction of coagulopathy. For severe respiratory failure, invasive mechanical ventilation using a lung-protective strategy is recommended while monitoring for respiratory acidosis. In cases of persistent hypotension due to systemic vasoplegia, vasoactive medications should be administered to maintain perfusion. Advanced therapies such as inhaled nitric oxide, ECMO, and intra-aortic balloon pump support have been reported in refractory shock [40]. If maternal demise occurs, an emergency peri-resuscitative cesarean section may be necessary to improve fetal survival.

## Air embolism

Air embolism is a rare but potentially fatal complication that occurs when air enters the venous circulation, leading to obstruction of the pulmonary arterioles. Although the overall incidence is low, air embolism accounts for approximately 1% of maternal deaths.

Air embolism is primarily an iatrogenic complication associated with procedures involving venous circulation. Obstetric risk factors include placenta previa, criminal abortions involving air injection, and vaginal insufflation during gynecological procedures. Clinical presentation varies based on the volume of air entrained and may include cough, chest pain, dyspnea, respiratory distress, lightheadedness, arrhythmia, right heart failure, and hypotension. In patients with a right-to-left shunt, air can bypass the pulmonary circulation and emboli to the cerebral circulation, resulting in cerebral infarction [41].

Diagnosis of air embolism is based on clinical suspicion, history, and imaging studies. Echocardiography may detect air bubbles in the heart, right ventricular (RV) and IVC dilation, and RV dysfunction. CT imaging may reveal air densities localized within the heart chambers and pulmonary arteries, particularly when the patient is supine [42].

For hemodynamically stable patients, management includes close monitoring and administration of 100% oxygen to enhance nitrogen resorption. In unstable patients, hemodynamic support with intravenous fluids, inotropic agents, and vasopressors may be required [43]. Patient positioning in the left lateral decubitus and Trendelenburg position (Durant's position) is sometimes recommended to prevent further air embolization into the pulmonary circulation, though its effectiveness remains uncertain. Hyperbaric oxygen therapy, if available, can

facilitate air resorption and reduce the risk of complications. In cases of large air embolism, aspiration of air using an intracardiac catheter may be considered as an additional intervention [43].

### Sepsis

Sepsis is a significant cause of maternal and fetal mortality due to immune system changes during pregnancy (pregnancy-induced immunosuppression) and challenges in early recognition. Physiological changes in pregnancy, such as decreased SVR, increased heart rate, and mild leukocytosis, can mimic a systemic inflammatory response syndrome (SIRS). Sepsis should be promptly suspected when there are rapid changes in hemodynamics or abnormal laboratory findings. Sepsis in pregnancy can arise from both obstetric and non-obstetric causes. Obstetric causes include postpartum endometritis, chorioamnionitis, and septic abortion. Non-obstetric causes in pregnant patients are similar to those in non-pregnant individuals [44].

Pneumonia is a common non-obstetric cause of sepsis in both pregnant and non-pregnant patients. Although the incidence of pneumonia is not higher in pregnancy compared to the general population, pregnant individuals are at greater risk of severe complications, such as preterm labor and respiratory failure. The bacterial pathogens causing community-acquired pneumonia in pregnant patients are similar to those in the general population. However, pregnancy increases susceptibility to viral and fungal infections due to downregulation of cell-mediated immunity [45].

The main treatment for sepsis involves prompt administration of appropriate antimicrobial therapy and effective control of the infection source. If sepsis progresses to septic shock, it should be managed urgently following established septic shock protocols, including fluid resuscitation, vasopressors, inotropic agents, and corticosteroids in cases of refractory shock [46]. Teratogenic and fetotoxic agents, such as aminoglycosides, tetracyclines, fluoroquinolones, and tigecycline, should be avoided [14].

### Obstetric hemorrhage

Obstetric hemorrhage can occur during both the antepartum and postpartum periods. Postpartum hemorrhage (PPH) is defined as blood loss > 500 mL within the first 24 hours following vaginal delivery or >1000 mL after a cesarean section. The etiology of hemorrhage during pregnancy includes ectopic pregnancy, abortion, placenta previa, placental abruption, uterine rupture, uterine atony, uterine inversion, retained placenta, trauma, and coagulopathy. Among these, uterine atony is the most common cause of PPH. Risk factors for uterine atony include uterine overdistension, placental abruption, retained intrauterine contents, rapid labor and delivery, prolonged labor, oxytocin use, cesarean delivery, and chorioamnionitis [47].

The main treatments for obstetric hemorrhage include blood transfusion, hemodynamic stabilization, oxygen supplementation, and ventilation support as needed. In cases of massive hemorrhage, management should follow the massive transfusion protocol to prevent complications such as dilutional coagulopathy and thrombocytopenia. There are various methods for controlling bleeding, depending on its cause. Uterotonics (e.g., oxytocin, ergot alkaloids, and prostaglandins) are effective in managing uterine atony. Additional interventions to control bleeding include selective arterial embolization, uterine packing, balloon tamponade, and uterine suturing. In cases of uncontrollable, life-threatening postpartum hemorrhage, an urgent hysterectomy may be required [48].

Recombinant activated factor VIIa has shown potential utility in severe refractory obstetric hemorrhage, as documented in some case reports [49]. Tranexamic acid is another significant drug that plays a role in the prevention and treatment of obstetric hemorrhage. The WOMAN trial demonstrated that early administration of tranexamic acid following vaginal delivery or cesarean section significantly reduces death due to bleeding, particularly in patients with uterine atony, when given within 1–3 hours of delivery [50]. Furthermore, the TRAAP study found that prophylactic use of tranexamic acid in women undergoing vaginal delivery, alongside oxytocin, reduced the incidence of clinically significant postpartum hemorrhage and decreased the need for additional uterotonic agents [51].

## CONCLUSION

The physiological changes during pregnancy are complex and affect multiple organ systems. The primary goal of treatment in critically ill pregnant patients is to minimize complications and reduce maternal and fetal mortality. Effective critical care management requires early diagnosis, careful maternal-fetal monitoring, and appropriate management. A multidisciplinary team approach is essential to ensure pregnant ICU patients receive comprehensive care from specialists in relevant fields.

## ACKNOWLEDGEMENT

None

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