



Acute Respiratory Failure During Pregnancy

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Abstract

Acute respiratory failure during pregnancy, although relatively uncommon, continues to be a prominent cause of maternal mortality, accounting for 30% of maternal deaths. Major respiratory changes in pregnancy which increase maternal susceptibility to acute respiratory failure can result from a variety of conditions, most of which are not pulmonary in origin. Early diagnosis of underlying disease is critical, as it will guide the management approach. Treatment goals respiratory failure in the pregnant woman are similar to those outside of pregnancy-to maintain adequate ventilation and to provide hemodynamic and nutritional support.

Introduction

Acute respiratory failure during pregnancy, although relatively uncommon, continues to be a prominent cause of maternal mortality, accounting for 30% of maternal deaths ^[1-5], and is the most common reason for admission to the intensive care unit (ICU) of critically ill obstetric patients. Pregnancy results in many changes to the female body, which make it more susceptible to respiratory complications ^[6-13]. Although the incidence of infections is not increased during pregnancy, complications from some infections, such as certain types of pneumonia, are increased. Hypercoagulability associated with pregnancy results in a marked increase in the incidence of thromboembolic disease. Although rare, pregnancy may be associated with other embolic phenomena, including amniotic fluid embolism, air embolism and trophoblastic embolism. Since there is an increase in intravascular volume and cardiac output during pregnancy, females with underlying cardiac disease may present with acute pulmonary oedema. The course of certain pulmonary diseases, such as bronchial asthma, may also worsen during pregnancy, causing respiratory failure. The outcome of pregnancies complicated by acute respiratory failure is not predicted by the aetiology of respiratory failure; however, patients presenting with a low arterial blood pH, initial loss of consciousness, disseminated intravascular coagulation and sepsis may be at higher risk of mortality, based on the results of one study ^[14] complicated by acute respiratory failure. Pregnancy of itself does not seem to increase mortality beyond that expected from the severity of the presenting illness ^[15]. Implications for the pregnant patient. Am J Respir Crit Care Med 2001; 163: 1051-1054. In general, pregnant patients with acute respiratory failure are treated

similarly to the no pregnant patient with the same illness, except for changes to accommodate the altered maternal physiology. This review focuses on the clinical manifestations and management issues of the most common causes of respiratory failure during pregnancy can occur due to many disorders. It can result in significant maternal and fetal morbidity and mortality.

RESPIRATORY PHYSIOLOGY IN PREGNANCY

Major respiratory changes in pregnancy involve three factors: 1. Mechanical- Effects of the enlarging uterus with diaphragm displacement 2. Consumption- Increased need for oxygen 3. Stimulation- Progesterone acts as a respiratory stimulant

Respiratory mechanics, in pregnancy Diaphragm rises 4 cm • Less negative intrathoracic pressure • Decreased Functional Residual Capacity • Decreased Expiratory Reserve Volume • Decreased Residual Volume No impairments in diaphragmatic or thoracic muscle motion • Lung compliance remains unaffected

Consumption • Oxygen consumption increases by 15-20% • 50% of this increase is required by uterus • Despite increase in oxygen requirements with the increase in CO₂ and increase in alveolar ventilation oxygen consumption exceeds the requirements, arterial PCO₂ falls

Stimulation • Progesterone directly stimulates ventilation • It also increases the sensitivity of the respiratory centers to CO₂

Additional respiratory points Minute ventilation=Respiratory rate x Tidal Volume • Tidal volume increases and RR stays same, so therefore minute ventilation increases • Vital capacity- remains unchanged

Dyspnea of pregnancy • Common symptom in pregnancy , Up to 60-70% of women will experience dyspnea at some point in their pregnancy • Mechanism not established but may involve increased sensitivity and lower threshold to PCO₂. (6TH, ED. text book of Obstetrics, DC DUTTA, CH1-5) .

	Anatomical changes mucosal friability, rhinitis	Edema,	Physiological alterations increased
Thorax including Lung parenchyma	Widened diameters, subcostal angle, elevated diaphragm	widened	respiratory drive
			Hyperventilation
			Reduced functional residual capacity
			increased tidal volume
			preserved vital capacity
Abdomen	Enlarged uterus		respiratory alkalosis
			Normal oxygenation
			Reduced chest wall compliance
Cardiovascular System	Increased left ventricular (LV) mass		Increased cardiac output
			Increased blood volume
Arterial blood gas			7.40-7.45 pH
			28-32 mmHg PaCO ₂
			106-110 mmHg PaO ₂

Causes of Respiratory failure in pregnancy

The various causes of acute respiratory failure are summarized in Table 1.

A. ARDS

- The criteria for diagnosis of ARDS are similar to nonpregnant women.

B. Asthma in pregnancy

- Rule of thirds- one-third of patients with asthma in pregnancy improve, and one-third shows no change. One-third worsens and can present in acute severe asthma.
- This explains the unpredictable effect pregnancy on asthma.

Table 1: Differential diagnosis of acute respiratory failure during pregnancy.

Conditions unique to pregnancy	Conditions can be affected by pregnancy	Conditions unaffected by pregnancy
Peripartum cardiomyopathy	Acute pulmonary edema	ARDS-direct/pulmonary
Amniotic fluid embolism	Aspiration of gastric contents	Bacterial pneumonia
Tocolytic therapy-induced acute pulmonary edema	Asthma	Fat embolism
Severe preeclampsia	Venous thromboembolism	Inhalational injury
Chorioamnionitis, endometritis	Bacterial and viral pneumonia	Indirect
Ovarian hyperstimulation syndrome (OHSS)	Malaria, fungal infection	Sepsis, trauma, burns
		Acute pancreatitis, transfusion-related acute lung injury (TRALI)

C. Pulmonary embolism in pregnancy

- Pregnancy itself is a hypercoagulable state and an independent risk factor for pulmonary embolism (PE).
- Clinical prediction models that are used to predict pretest probability of PE have not been validated in pregnant patient.
- D-dimers are likely produce differently in pregnant population as D-dimers may be falsely high in pregnant patients.
- Radiographic imaging remains the primary testing modality for diagnosing PE, and it should not be delayed because of concerns about radiation exposure.
- Multidetector compound tomography (MDCT) pulmonary angiography is currently the most preferred mode for confirming diagnosing PE in pregnant patients. The main concern with (MDCT) are radiation exposure to the fetus in suspected PE. It has been seen that exposure to radiation is less to the fetus.
- Compression ultrasonography and transesophageal echocardiography (TEE) are the initial test to deep venous thrombosis.
- chest radiograph involve minimal radiation.
- The accuracy of ventilation –perfusion scan in pregnancy is not available and outcome studies are limited.

D. Ovarian hyperstimulation syndrome (OHSS)

Gestation of 3-8 weeks.

Increased vascular permeability-fluid shifts from intravascular to extravascular space-causing pleural or pericardial effusion, ascites, electrolyte imbalance, dyspnea, oliguria, severely enlarged polycystic ovaries, hemoconcentration and hypercoagulabilities, electrolyte imbalance are the common presentation.

Table 1 Criteria that define the severe and life threatening stages of OHSS

Severe OHSS	Life threatening OHSS
Variable enlarged ovary	Vaiable enlarged ovary
Massive ascites with or without hydrothorax	Tense ascites with or without hydrothorax
Hematocrit >45%	Hematocrit>55%
WBC Count > 1'5000	WBC > 25'000
Oliguria	Oliguria
Creatinine level 1.0-1.5mg/dl	Creatinine level>1.6mg/dl
Creatinine clearance > 50ml/min	Creatinine clearance<50ml/min
Liver dysfunction	Renal failure
Anasarca	Thromboembolic phenomena
	ARDS

E.Peripartumcardiomyopathy (PPCM)

- Risk factor include hypertension, preeclampsia, multiparity, multiplegestations, and older maternal age.
- Signs and symptoms are paroxysmal nocturnal dyspnea, pulmonarycrackles, increased jugular venous pressure, and hepatomegaly.
- Identify other cardiac and noncardiac condition such as coronary, rheumatic, and valvular heart disease; and family history of cardiomyopathy.

Clinical criteria for the diagnosis of PPCM

- Development of cardiac failure in the last month of pregnancy or within 5 months of post partum.
- Absence of another identifiable cause for the cardiac failure.
- Absence of other recognizable heart disease before the last month of pregnancy.
- LV systolic dysfunction shown by echocardiographic data such as depressed shortning fraction

Or sudden death and other risk factors of cardiac disease such as hypertension, diabetes, dyslipidemia, thyroiddisease, anaemia, prior chemotherapy or mediastinalradiation, sleepdisorder,c ollagen vascular disease.

- The diagnosis of PPCM is a diagnosis of exclusion and shoud be made when other possible causes of acute/subacute heart failure have been ruled out

Treatment of the specific cause

The general management of respiratory failure in pregnancy is similar to the management in non pregnant woman, although one should be careful about normal physiological alterations that occur in parturient state and effect of ventilator strategies.

A. Management of ARDS and mechanical ventilation in pregnant woman

- Lung protective strategy to avoid volutrauma, barotrauma, atelectrauma leading to less ventilator induced lung injury has been found to reduce mortality and improve outcome in patients with ARDS
- Lung-protective strategy causes hypoventilation, which is tolerated to maintain (permissive hypercapnia) the PH between 7.25 and 7.35.
- Permissive hypercapnia can cause fetal acidosis, an increase in intracranial pressure, and a right shift in hemoglobin dissociation curve and in first 72 hours may lead to retinopathy of prematurity, so lung protective ventilator strategy in pregnant patients should be used in close monitoring with the fetal status with the biophysical profile.
- Oxygen levels should be closely monitored in pregnancy and should be kept higher than in non pregnant women (preferably SpO₂>95%)

B. Management of asthma in pregnancy

- Management of asthma in pregnancy similar to non pregnant woman.
- Beta-agonists, bronchodilator and corticosteroids are the mainstay of the treatment.

C. Pulmonary embolism during pregnancy

- Acute treatment of pulmonary embolism can be done with low molecular weight heparin or unfractionated heparin and should be started when pulmonary embolism is suspected or confirmed.
- LMWH is first line therapy for PE in the pregnant population and in the pregnancy as the risk of bleeding in pregnant woman is not different from non pregnant woman.
- Thrombolysis increases the risk of obstetric and neonatal complication, such as pregnancy loss, abruption, and preterm labour. Thus thrombolysis in pregnancy should be reserved for woman with PE who are hemodynamically unstable or with refractory hypoxemia.
- The American college of chest physicians guidelines recommends the use of anticoagulants for 6 months at least in the postpartum period.
- Always give injectable heparins during the entire period of pregnancy. start oral anticoagulants only after delivery.

D.OHSS

- Syndrome is self-limiting, and resolution parallels the decline in serum HCG levels:7 days in non pregnant patients and 10-20 days in pregnant patients.
- Monitor frequently for deterioration with physical examination, daily weights, and periodic laboratory measurements of complete blood counts, electrolytes, hepatic and renal functions.
- Severe disease-placement of two large bored peripheral intravenous catheters or a central venous catheters (preferred)for fluid management may be required.
- Use of foleys catheter for close monitoring of urine output.
- Normal saline with or without glucose is the fluid of choice and potassium containing solution should be avoided because patients with OHSS develop hyperkalemia.
- In more severe cases with significant hypovolemia,hemoconcentration(hematocrit<45%),hyposalbuminemia(serum albumin<3.0g/dl),or severe ascites, albumin can be given as plasma expander along with diuretics once hematocrit is 36-38%
- If ARDS develops the mechanical ventilation is required,lung protective strategy must be used.

E.PPCM

- Diuretics are indicated in most patients because they cause symptomatic relief of pulmonary and peripheral edema and used as an adjuvant to other definitive therapies. Furosemide are most commonly used.
- Aldosterone antagonist are used to increase survival of selected heart failure patients, these agents are still not used in pregnancy
- Hydralazine and nitrates are vasodilator of choice for pregnant women
- B-Blockers (sustained release metoprololsuccinate, carvedilol and bisoprolol) have been used to reduce mortality with current or prior heart failure and with reduced ejection fraction and therefore constitute the firstline therapy for all stable patients unless contraindicated.
- Digoxin improves symptoms, quality of life,and improve exercise tolerance in mild to moderate heart failure.

MECHANICAL VENTILATION IN PREGNANCY

Critical illness requiring admission to an intensive care unit (ICU) is a relatively uncommon complication of pregnancy, accounting for less than 1% of ICU admissions ⁽¹⁶⁾ In a case control study El-Solh and Grant, retrospectively compared 93 critically ill pregnant patients with a variety of diagnoses to a similar number of nonpregnant females of the same age admitted to intensive care units. Respiratory illness (31%) and the need for mechanical ventilation (38%) were slightly greater in obstetric patients than in the nonobstetric patients (24 and 24%, respectively) The extrapolation of existing studies and principles in the context of a particular clinical situation is necessary when data are not available to reach an evidence-based conclusion. The need for this extrapolation is intensified when the medical decision involves a critically ill pregnant patient in whom altered maternal and fetal physiology must be considered (A. CAMPBELL LUCY and A. KLOCKE ROBERT "Implications for the Pregnant Patient", American Journal of Respiratory and Critical Care Medicine, Vol. 163, No. 5 (2001), pp. 1051-1054)

NONINVASIVE MECHANICAL VENTILATION

The pregnant female requiring endotracheal intubation has a four-fold higher risk of having a difficult airway and an eight-fold higher risk of a failed intubation(. Successful use of noninvasive ventilation in pregnancy Charles C. Allred, Antonio Matíassquinas, Jonathan Caronia, Ramyar Mahdavi, Bushra A. Mina). NIPPV has had limited application in pregnant patients due to the perceived risk of aspiration. The current case series has its own limitations in providing definitive recommendations for the indications for and use of NIPPV in the pregnant patient.

Obstetric patients with preexisting medical problems are more likely to require intensive-care support than those without preexisting medical conditions.^[17] Sickle cell disease is the most common major underlying chronic medical condition in obstetric patients admitted to our ICU. ACS is believed to be a specific form of acute lung injury that can progress to acute respiratory distress syndrome causing ARF.^[18]

Young adults have a lower incidence of ACS, but it tends to be more severe and is often fatal.^[19]

A significant incidence of right ventricular dysfunction and pulmonary hypertension in asymptomatic patients with sickle cell anemia has been reported⁽²⁰⁾

Currently there is not enough evidence to support safe use of NIPPV in ARF in a pregnant patient. The current case series provides the best available evidence to support the use of NIPPV in ARF during pregnancy. In closely monitored pregnant patients with ARF, NIPPV seems to have the potential to shorten ICU and hospital stay. A well-conducted randomized controlled clinical trial is required to confirm this finding.

Conclusion

Pregnant patients with ARF admitted to ICU need aggressive and timely intervention from a multidisciplinary team of physicians to minimize the morbidity and mortality associated with this devastating complication. Moreover, acute respiratory failure contributes substantially to maternal morbidity and mortality; it can also harm the fetus by compromising fetal oxygen delivery. Critical illness in pregnant women poses special challenges. Physiologic changes that affect cardiovascular and respiratory function are normal during pregnancy. Through interaction with preexisting or new co-morbidities, those changes can give rise to life-threatening complications. Always present, too, is the need to consider the effects of both disease and its treatment on fetal development and outcome.

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