www.jmscr.igmpublication.org

Impact Factor 3.79
Index Copernicus Value: 5.88
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: http://dx.doi.org/10.18535/jmscr/v4i02.43

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

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 incritically ill obstetric patients and is the most common reason for admission to the intensive care unit (ICU).Altered maternal respiratory physiology affects the assessment and management of these patients. Respiratory failure may result from pregnancy-specific conditions such as preeclampsia, amniotic fluid embolism or peripartum cardiomyopathy. Pregnancy may increase the risk or severity of other conditions, including thromboembolism, asthma, viral pneumonitis, and gastric acid aspiration. Management during pregnancy is similar to the nonpregnant patient. Endotracheal intubation in pregnancy carries an increased risk, due to airway edema and rapid oxygen desaturation following apnea. Few data are available to direct prolonged mechanical ventilation in pregnancy. Chest wall compliance is reduced, perhaps permitting slightly higher airway pressures. Optimizing oxygenation is important, but data on the use of permissive hypercapniaare limited. Delivery of the fetus does not always improve maternal respiratory function, but should be considered if benefit to the fetus is anticipated.

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. Hypercoagul ability 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 trophoblasticembolism. 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 Med2001; 163: 1051–1054. Ingeneral, 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

1. The diaphragm is displaced upwards by up to 4 cm, but the potential loss of lung volume is offset by widening of the anteroposterior and transverse thoracic diameters. Functional residual capacity (FRC) decreases by 10% to 25% by term.
2. The vital capacity remains unchanged, and total lung capacity decreases only minimally. Measurements of airflow (FEV1) and lung compliance are not altered during pregnancy, but chest wall and total respiratory compliance are reduced in the third trimester.
3. Minute ventilation increases progressively during pregnancy, beginning in the first trimester and reaching 20% to 40% above baseline by term. An increase in respiratory drive is caused by elevated serum progesterone levels, producing an increase in tidal volume with very little change in respiratory rate.
4. A respiratory alkalosis develops with compensatory renal excretion of bicarbonate, with PaCO2 falling to 28 to 32 mmHg (3.8–4.3 kPa) and plasma bicarbonate falling to 18 to 21 mEq/L.
5. Alveolar-to-arterial oxygen tension differences (PAO2-PaO2) are usually unchanged by pregnancy, although mild hypoxemia may develop in the supine position as FRC diminishes near term. Oxygen consumption is increased, beginning in the first trimester and reaching 20% to 33% above baseline by the third trimester. The combination of a reduced FRC and increased oxygen consumption cause the pregnant patient to the rapidly develop hypoxia in response to hypoventilation or apnea.
6. Alkalosis may worsen fetal oxygenation by reducing uterine blood flow.
7. This can occur during hyperventilation related to labor as well as due to a metabolic alkalosis that can be produced by volume depletion and vomiting. Adequate pain relief blunts this ventilatory response, and can correct the hyperventilation associated with active labor.

	Anatomical changes Edema, mucosal friability, rhinitis	**Physiological alterations increased respiratory drive**
		Hyperventilation
Thorax including Lung parenchyma	Widened diameters, widened subcostal angle, elevated diaphragm	Reduced functional residual capacity
		increased tidal volume
		preserved vital capacity
		respiratory alkalosis
		Normal oxygenation
Abdomen	Enlarged uterus	Reduced chest wall compliance
Cardiovascular	Increased left ventricular (LV) mass	Increased cardiac output

System	
	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 nonpregnantwomen.

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
		Bacterial pneumonia
Amniotic fluid embolism	Aspiration of gastric contents	Fat embolism
Tocolytic therapy-induced acute pulmonary edema	Asthma	Inhalational injury
Severe preeclampsia	Venous thromboembolism	Indirect
Chorioamnionitis, endometritis	Bacterial and viral pneumonia	Sepsis, trauma, burns
Ovarian hyperstimulation syndrome (OHSS)	Malaria, fungal infection	Acute pancreatitis, transfusion-related acute lung injury (TRALI)

C.Pulmonary embolism in pregnancy

- Pregnancy itself is a hypercoaguiable state and an independent risk factor for pulmonary embolism (PE).
- Clinical prediction models that are used to be 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.
- Mulidetector compound tomography (MDTC) 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.
- Compresion ultrasonography and transesophagialechocardiography (TEE) are the initial test to deep venous thrombosis.
- chest radiograph involve minimal radiation.

- The accuracy of ventilation –perfusion can in pregnancy is not available and outcome studies are limited.

D.Ovarian hyper stimulation syndrome(OHSS)

Gestation of 3-8 weeks.

Increased vascular permeability-fluid shifts from intravascular to extravascular space-causing pleural or pericardial effusionicardialeffusion, ascites, electrolyteimbalance, dyspnea, oliguria, severlyenlarged poly cystic ovaries, hemoconcentration and hypercoagubilities, 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, atelactrauma 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 unfractioned heparin and shoud 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 obstretic 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 detoriation with physical examination, daily weights, and periodic laboratory measurements of complete blood counts, electolytes, 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%),hypoalbuminemia(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 (sustaiened 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.

Ventilatory management of pregnant patients:

Ventilatory management in the pregnant patient Intubation Endotracheal intubation in the pregnant patient carries considerable risk. Failed intubation is 8 times more common in the obstetric population than in other anesthetic intubations [16]. The reduced FRC and increased oxygen consumption in pregnancy cause rapid oxygen desaturation during apnea or hypoventilation. Upper airway mucosal edema and friability can adversely affect visualisation and increase the risk of bleeding. Nasal intubation should be avoided and a smaller size endotracheal tube may be required. Preoxygenation is important, but over ventilation and respiratory alkalosis must be avoided. The risk of aspiration should always be considered.

Noninvasive ventilation

Noninvasive ventilation is well suited to short-term ventilatory support, and avoids the potential complications of endotracheal intubation and the associated sedation. This modality has a role in obstetric respiratory complications which reverse rapidly [17]. The major concern with this form of ventilator support is the risk of aspiration. Noninvasive ventilation should therefore only be used in the pregnant patient who is alert, protecting her airway and where there is an expectation of a relatively brief need for mechanical ventilation.

Invasive mechanical ventilation

Prolonged mechanical ventilation of pregnant patients in the ICU is relatively uncommon, and few data are available to guide management. Hyperventilation and alkalosis should be avoided to prevent uterine vasoconstriction [18]. Lung protective ventilation, sometimes producing permissive hypercapnia, has not been assessed in pregnancy. Chest wall compliance is reduced by the enlarging uterus, and the usual pressure limits (e.g. plateau pressure of 35 cmH2O) may not be appropriate. Slightly higher airway pressures (without increased transpulmonary pressure) may be needed to achieve appropriate tidal volumes in pregnant women near term. Blood gas abnormalities may adversely affect the fetus. [19]. Oxygenation should be optimized to ensure adequate fetal oxygenation. A maternal oxygenation goal of pO2 greater than 70 mmHg has been suggested. However, a short-term study of controlled maternal hypoxemia (585%) using inhalation of 10% oxygen demonstrated no adverse effects on fetal monitoring [20]. Maternal oxygen saturation is only one factor contributing to fetal oxygenation, placental perfusion usually playing a more significant role. While excessive hypocapnia may cause fetal harm by reducing placental perfusion, the effects of hypercapnia on the fetus are less clear. Maternal CO2 levels are normally reduced to about 27 to 34 mmHg, producing a gradient to facilitate placental excretion of fetal CO2. Permissive hypercapnia has not been evaluated in pregnancy, and maternal hypercapnia could produce fetal respiratory acidosis. This acidosis likely does not have the same ominous implications for the fetus as the lactic acidosis produced by

hypoxemia, which implies significant tissue hypoxemia. Small clinical studies have evaluated the short-term effect of mild hypercapnia in pregnancy. Women undergoing cesarean delivery were subjected to mild hypocapnia (mean 23 mmHg) or mild hypercapnia (39.3 mmHg) [21] Hypocapnia produced a lower Apgar score and delayed neonatal breathing. Another small study compared ventilated women delivered with mild hypercapnia (mean CO2 57.6 mmHg) with ventilated women with mild hypocapnia (mean CO2 26.4 mmHg) and women managed with a local anesthesia block (mean CO2 30.1 mmHg). The hypercapnic group had a statistically significantly higher Apgar score at delivery. If necessary, mild hypercapnia with PaCO2 maintained less than 60 mmHg, has been recommended for pregnancy. It should be noted that the right shift of the haemoglobin oxygen dissociation curve caused by acidosis may negate the beneficial oxygen-carrying characteristics of fetal haemoglobin.

Nonconventional support

Few data are available to support the use of interventions such as inhaled nitric oxide, prone positioning and high-frequency oscillation in pregnancy, although these modalities have been used successfully and described in case reports and small series. The 2009 H1N1 influenza epidemic resulted in a marked increased utilization of extracorporeal life-support for ARDS, including pregnant patients with reasonably good outcome.

Delivery of the fetus

It has been suggested that delivery of the pregnant patient with respiratory failure will result in improvement in the mother's condition. However, a significant benefit to the mother has not been consistently demonstrated [23,24]. If the fetus is at a viable gestation and is at risk due to intractable maternal hypoxia, there may well be a benefit to the fetus in delivery. The mode of delivery should be determined by standard obstetrical principles. Although cesarean section may allow more rapid delivery in the critically ill patient, there is significantly increased physiological stress, and operative delivery has been associated with higher mortality in these patients. Delivery should not be performed solely in an attempt to improve maternal oxygenation or ventilation. It is essential that the ICU have prearranged plans for urgent delivery and neonatal resuscitation in the event of spontaneous labor or sudden maternal or fetal deterioration. This should include immediate availability of all necessary equipment, drugs and staff contact details.

Conclusion

Respiratory failure complicates a relatively small number of pregnancies, but carries significant potential risks for both mother and fetus. Causes of respiratory failure may be related to pregnancy-specific conditions or other respiratory diseases, and management requires a multidisciplinary team approach, involving obstetrics, maternal-fetal medicine, neonatology, obstetric medicine, pulmonology, and critical care. A major decision facing this multidisciplinary team is the potential benefit to the mother of delivery—this cannot always be predicted, and the decision should be based on the overall risk balance to both mother and fetus.

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